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An Examination of Proactive Interference and Negative Priming in Individuals wIth and without Attention-Deficit/Hyperactive Disorder

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An Examination of Proactive Interference and Negative Priming in Individuals with and without Attention-Deficit/Hyperactive Disorder

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Abstract

Attention-deficit/hyperactivity-disorder (ADHD) is a disorder classified by behaviors of inattention, hyperactivity, and impulsivity. When put in a task that requires selective attention, many neurotypical individuals are slower to respond to previously ignored information–a phenomenon called negative priming–wherein that pattern is not always apparent in individuals with ADHD. One explanation for negative priming is that it is rooted in memory retrieval, which proposes that individuals with ADHD are less likely to retrieve information about items that they have previously ignored. If individuals with ADHD exhibit less negative priming because they are less likely to retrieve prior information, then they may also exhibit reduced proactive interference. Proactive interference is observed in recall tasks when old information interferes with current recall. It was predicted that those with ADHD would be less affected by old information and therefore exhibit atypical proactive interference, as compared to neurotypical individuals. In the current study, ADHD was assessed using the CAARS S:S (in Bates College participants) and ASRS (in Amazon Mechanical Turk participants) scales. Proactive interference, measured using a Brown-Peterson task, was intact among those with ADHD. In a second experiment, negative priming, assessed using a flanker task, suggested that negative priming may be absent in individuals with ADHD, though the findings remain inscrutable. This may be indicative of intact memory retrieval in those with ADHD, and further attribute negative priming effects to underlying mechanisms unassociated with memory retrieval.

An Examination of Proactive Interference and Negative Priming in Individuals wIth and without Attention-Deficit/Hyperactive Disorder

Attention-deficit/hyperactivity disorder (ADHD) has become increasingly prevalent in the media in recent years. Much of the information online, especially in pop psychychology, relay inaccurate or misleading information–wherein it continues to be considered by many as an insignificant and unimportant deficiency despite leading to glaring difficulties and impactful issues in the lives of those diagnosed with ADHD (Low, 2021). The current study examines ADHD over the course of three experiments. I begin by discussing attention-deficit/hyperactivity disorder and its clinical and physiological manifestations. Next, I review negative priming and the prominent theories behind it. Correspondingly, I mention several associated studies examining negative priming effects on those diagnosed with ADHD compared to neurotypical individuals. Of the mentioned proposed theories, I focus my review on the implicit-retrieval memory-based theories and inhibitory theories. Finally, I discuss another memory-based effect: proactive interference. Subsequently, I discuss the features of proactive interference as well as prominent past studies associated with it.

Attention-Deficit/Hyperactivity Disorder

Attention-deficit/hyperactivity disorder, or ADHD, is a disorder that can be classified by behaviors of inattention, hyperactivity, and impulsivity, hindering an individual's ability to function as well as their development. Individuals with ADHD usually have issues with both hyperactivity-impulsivity and inattention, though some individuals primarily display one or the other (Parekh, 2017). Most people experience these behaviors to some degree; however, it becomes a problem when such behaviors interfere with and reduce the quality of an individual's life. According to the National Institute of Mental Health (2019), individuals who struggle

primarily with symptoms of inattention, also struggle with actions and feelings such as overlooking details, maintaining attention for tasks and conversations, procrastinating tasks that require mental attention and effort, staying organized, meeting deadlines, distractions, forgetfulness, etc. Individuals who struggle primarily with hyperactivity-impulsivity have issues with actions and tasks such as staying in one place, excessive talking, and interrupting others. These symptoms must be consistent over time and chronic for a physician to diagnose an individual with ADHD, though symptoms are subject to change over time (Meyer & Lasky, 2021).

Physiology of ADHD

Physiologically, the dysfunction in the dopaminergic and noradrenergic neurotransmitters are implicated in adversities presented by ADHD; both of these neurotransmitters are additionally fundamental parts of the frontostriatal and frontoparietal circuits (Arnsten et al., 1996). As such, individuals with ADHD have been seen to display irregular activity in the prefrontal cortex, frontoparietal and frontostriatal circuits (Booth et al., 1995; Castellanos, 1997; Durston et al., 2003; Giedd et al., 2001; Lou et al., 1989; Seidman, 2005). The anterior cingulate cortex (ACC), which is seen to be implicated in cognitive control and attention direction, is therefore of particular interest regarding its dysfunction in individuals with ADHD. This performance discrepancy becomes apparent through the implementation of a Stroop (1935) task to assess inhibitory control in conjunction with magnetic resonance imaging (MRI). A Stroop task is where participants have to identify the color in which the word is written when presented with a color word while ignoring the written word. For instance, the word “red” could be written in the color green, in which case the participants would have to respond with green. Though no significant disparities were able to be identified based on Stroop

task performance, the data showed that those with ADHD presented with significant thinning in the right rostral ACC, which also accounted for a significant amount of the variance found in the symptoms reported by the parents and teachers of the participants; based on this correlation, it was concluded that the ACC plays a role in behaviors of inattention, impulsivity, and hyperactivity (Bledsoe et al., 2013).

Other anatomical regions have been associated with the neurological deficits of ADHD, particularly in the frontostriatal network, involving the lateral prefrontal cortex, dorsal anterior cingulate cortex, and caudal nucleus. Additionally, a substantial decay was present in the total cerebral volume in the prefrontal cortex, basal ganglia (striatum), dorsal anterior cingulate, cortex, corpus callosum, and cerebellum (Emond et al., 2009). Developmental studies conducted on individuals with and without ADHD showed impediments in cortical maturation over the course of three years compared to the neurotypical controls. MRI data has additionally correlated ADHD symptoms with diminished white matter connectivity and volume within the midsagittal corpus callosum areas and further corroborated the findings regarding reduced cortical thickness (Makris et al., 2008). These irregularities in white matter are ubiquitous throughout the brain primarily affecting the inferior parietal, occipitoparietal, inferior frontal, and inferior temporal cortex, regions which constitute a portion of the white matter pathways connecting the prefrontal and parietooccipital regions with the striatum and cerebellum. White matter pathways affected are also seen in prefrontal regions and in pathways proximal to the basal ganglia and cerebellum. This suggests decreased myelin sheathing of axons may lead to less insulation of the neuronal impulses, and ergo elicit inefficient action potentials (Curatolo et al., 2010; D'Agati et al., 2010).

Clinical Diagnosis of ADHD

Clinically, ADHD is diagnosed on a behavioral rather than biological basis based on the prevalence of symptoms of inattention in comparison to hyperactivity. According to DSM-V criteria, at least five symptoms of either hyperactivity, impulsivity, or both, must persist for six months or more to be recognized as an adult ADHD diagnosis (American Psychiatric Association, 2013). There are three different subtypes of ADHD: inattentive, impulsive-hyperactive, and combined. Though there is no apparent cause for ADHD, there are several contributing factors including genes, alcohol or drug use during pregnancy, smoking during pregnancy, exposure to high amounts of toxins as an infant, being underweight at birth, and brain injuries. Currently, there is no cure for this condition, however, symptoms can be managed using medications, psychotherapy, education, or a combination of any of these (Oh et al., 2018).

ADHD is prevalent in 4.4% of the adult population, though it is chronically underdiagnosed (National Institute of Mental Health); fewer than 20% of adults with ADHD receive a diagnosis or treatment (Hallowell, 2021). It has been shown to be associated with other psychiatric disorders such as mood disorder, anxiety disorder, substance abuse, as well as antisocial and illegal behaviors. As such, ADHD has been shown to incite numerous long-term health and economic consequences–including costs to an individual's mental health, education, social service, and criminal justice. Adults with ADHD were also seen to display significant functional impairments. Despite these ramifications, ADHD continues to be clinically underrepresented (Oh et al., 2018).

ADHD and Anxiety

Individuals with ADHD have been shown in the literature to have a particularly high comorbidity rate with anxiety disorder, being prevalent in 34% to 40% of individuals with an

ADHD diagnosis (Sobanski 2007; Uchida et al., 2018). It is considered to be among the highest inherited psychiatric disorders with a 76% chance of an individual inheriting symptoms of anxiety (Johnson et al., 2009). Experiencing anxiety is also seen to increase the liability for other psychiatric disorders–such as depression, somatoform disorder, conduct disorder, and substance abuse–and is often a pertinent element in the prognosis and treatment of various psychiatric disorders, including ADHD (Gaynes et al., 1999; Hensley & Varela, 2008; Schmidt et al., 2007). Anxiety can have significant repercussions on one's self-esteem, social relationships, academic performance, and is shown to lead to considerable functional impairments (Nima et al., 2013; Richardson et al., 2012). Further, anxiety has been seen to be a mediator between the severity of ADHD symptoms and functional impairment (Kroenke et al., 2013). High sensitivity to anxiety can provide foresight regarding the severity of an individual's panic disorder, and can increase the risk of a mood disorder, psychosomatic disorder, and post-traumatic stress disorder. Additionally, it can increase the probability of suicidal ideation and poor impulse control, which in combination can be detrimental and even lead to suicidal ideations and attempts (Oh et al., 2018). With ADHD, anxiety has been shown to foreshadow poor behavioral regulation and externalizing issues in ADHD children (Brooker et al., 2020). Furthermore, both conditions have been recently found to share a common genetic pathophysiology. Greater levels of anxiety have been significantly positively correlated with the severity of ADHD symptoms; trait anxiety predominantly moderates elements of impulsivity and emotional liability associated with anxiety (Oh et al., 2018).

Theories of ADHD

There have been a wide array of theories proposed attempting to describe the differences seen in those with ADHD. One major category of ADHD theories include cognitive dysfunction

theories which attribute ADHD discrepancies to deficits in sustained attention and response inhibition (Barkley, 1997), working memory (Baddeley, 1992), and executive function (i.e., the cognitive mechanisms necessary to sustain the appropriate response) (Willcutt et al., 2005). For example, the Inhibition Model (Barkley et al., 1992; Barkley, 1997) suggests that ADHD core deficits are a consequence of poor behavioral inhibition. Similarly, the Executive Dysfunction Theory postulates ADHD arises from diminished executive control as a consequence of deviations in physiology, function, and chemical processes in frontoparietal and frontostriatal neuronal circuitries (Aman et al., 1998; Barkley et al., 1992; Barnett et al., 2001; Breen, 1989; Cairney et al., 2001; Carter et al., 1995; Willcutt et al., 2005). In contrast, state regulation models of ADHD suggest that such individuals have inadequate allotted supplemental effort causing them to struggle with persistent maintenance of the optimal arousal state. ADHD symptoms may be exacerbated or impeded depending on the current cognitive state of the individual; for instance, inattentive symptoms may be more evident in less cognitively demanding or slower tasks (Johnson et al., 2009). Alternatively, several theories have emphasized the importance of reinforcement processes (Shiels & Hawk, 2010). For instance, one theory posits that inhibition, or executive function, and delay aversion performance demonstrate two independent pathways (Dalen et al., 2004; Sonuga-Barke et al., 2003). In this consolidated paradigm, inattention and hyperactivity in ADHD are credited to efforts in minimizing the delay of experienced gratification in appropriate circumstances where such delay is inevitable. Another major reinforcement theory behind ADHD is the Dynamic Developmental Theory (DDT), which ascribes ADHD deficiencies to inappropriate reinforcement and inadequate extinction of incorrect behavior (Sagvolden et al., 2005) which must occur within a critical time period for neurotypical individuals, and similarly fails to be

impeded due to lower baseline levels of dopamine–as phasic reduction of said dopamine is typically indicative of extinction processes. This consequently suggests impaired anterior cingulate, dorsolateral prefrontal, and motor circuit activity (Johnson et al., 2009; Shiels & Hawk, 2010).

Negative Priming

There have been many studies done over the years regarding the cognitive differences among individuals with ADHD versus neurotypical individuals (Christiansen & Oades, 2010; Curatolo et al., 2010; Gaultney et al., 1999; Gilden, 2002; Keppel & Underwood, 1962; Marriott, 1998; Mclaughlin, 2003; Ossman & Mulligan, 2003; Pritchard et al., 2006; Pritchard et al., 2008; Shin, 2005; Shin, 2006; Storm & White 2010; White, 2007). One important discovery that has been recurrently deliberated is potential disparities in levels of negative priming presented in those diagnosed with ADHD. Negative priming is a phenomenon in which individuals are slower to respond to previously ignored items, known as the ignored repetition, than items that were not previously ignored, the control (Tipper, 1985). For example, if an individual is asked to say aloud the letter in the middle of "ABA" in the first round and then the letter in the middle of "CAC" in the second round, reaction times in the second round will be slower than situations where they had not just ignored the target letter (A).

An Overview of the Proposed Theories Regarding Negative Priming

A reduction in negative priming in individuals with ADHD indicates that whatever cognitive mechanism is responsible for negative priming may be disrupted with ADHD. There are several theories behind the reasoning for why negative priming occurs, most notably characterized as inhibition theories or retrieval-based accounts. In generally comparing the two major frameworks, inhibition theories involve either activation of the prime target and inhibition

of the prime distractor (Frings et al., 2015); however, where inhibition theory involves inhibiting distractors which must subsequently be responded to, retrieval theories attribute slower response times to complications in memory retrieval.

Inhibition Theory

The original explanation for negative priming is the inhibition theory–which, as the name suggests, attributes the phenomenon to inhibitory causes—and claims that negative priming reflects a person's ability to inhibit distracting information. In 1985, Tipper did a study evaluating the processing of ignored objects during the selection of an attended object; he evaluated their stage of internal representation and the fate of said representation. A three-part study was conducted to ascertain the presence of negative priming, the effect on negative priming of a shortened interstimulus interval, and the priming effects experienced by both the selected and ignored objects. Results showed that there was, in fact, an opposite priming effect (i.e., negative priming) on objects that had previously been ignored eliciting longer response times. This is suggestive of the fact that during selection on the probe trial, the internal representation of the ignored item may be affiliated with inhibition. Response times when the selected and ignored objects were semantically related increased by 31 milliseconds, suggesting that ignored objects may be processed at the categorical level, and ergo, inhibition may also be at this level (Tipper, 1985). Rather than progressively diminishing, mental representations of ignored objects are further processed such that their next representations are hindered (Tipper, 1987).

These findings are further reinforced by the finding that if the probe and prime were presented immediately without an interstimulus interval, negative priming did not have time to form and was thereby diminished; that is, negative priming needed time to be developed

(Tipper, 1985). This theory later extrapolated into the Selective Inhibition Account, which additionally proposed that such inhibition may only be implemented if the activated mental representations and the behaviors are consistent; if not, the organism is inclined to inhibit the desired response (Fox, 1995; Tipper et al., 1994). This account differs from the inhibition account as it proposes that an additional step involving the facilitation of relevant information (i.e. amplification) in addition to inhibition of irrelevant information. According to this view, people with ADHD may have more difficulty inhibiting distraction than individuals without ADHD. This view would attribute negative priming to inhibition in attention rather than retrieval in memory.

Implicit-Retrieval Based/Selective Attention Theories behind Negative Priming

Feature Mismatch Account

Implicit retrieval-based theories posit that negative priming is due to trace activation in memory of a perceived target mirroring the specific stimulus from a previously processed episode. In the previous trials, the memory trace of the indicated target stimulus was previously appraised to be "ignored" or as "do not respond," thereby interfering with one's ability to rapidly and accurately respond to said item. One such theory is the feature mismatch account, which speculates that negative priming may be attributed to a discrepancy among the features of an ignored item in the prime conditions and subsequent probe targets (Fox, 1995).

This is corroborated by a study done by Lowe (1979) with a Stroop color-naming task whereupon a color-word was presented in the prime stimulus followed by an unanticipated presentation of another color-word or color-patch. This study found significant negative priming was present when the color word was congruent with the probe stimulus, however, when the probe was a color patch, responses were facilitated. He argued that if the color name presented

in the probe-target is dually displayed as an ignored distractor and the selected target–that is, if participants were presented with the word “blue” written in red, followed by the word “yellow” written in blue–this causes the color name to be highly inscrutable. This incompatibility–or mismatch–among the stimuli elicits further processing of the stimuli to “check,” resulting in slower response times, producing negative priming due to the mismatch of features between the ignored distractor and subsequent target (Fox, 1995).

Alternatively, the selective inhibition account would not attribute this finding to the same mechanisms as it ascribes negative priming to the inhibition of an ignored item that then becomes the target. The feature mismatch theory describes negative priming as a feedforward effect from mismatching messages from successional trials rather than the distractor inhibition from the prime trial lingering during the probe display (Fox, 1995).

Episodic Retrieval Theory

Episodic retrieval theory is another theory behind negative priming which is implicitly based in information retrieval (Neill et al., 1992). It is premised on the idea that information retrieved from the prime display contradicts the current and the correct response; that is, people tag in memory what should and should not be responded to on each trial. This theory highlights the role of the stimulus in the probe display as a memory-retrieval cue. It essentially postulates that in an ignored repetition condition, the probe-target is processed causing the automatic retrieval of the prime display in the preceding processing episode–which may contain information regarding the status of the item (i.e., as relevant/respond or irrelevant/do not respond). That is, in ignored repetition trials, an episodic memory trace encoding a non-response status conflicts with the appropriate response (Fox, 1995). To give an example, if participants were shown ABA and asked to report the middle letter, they will store in memory

"respond B" and "ignore A". If people are later shown CAC and if they recall the prior information, then they will have slow responses because they will recall that the letter A should be ignored (Neill et al., 1992).

Though this is quite similar to the feature mismatch theory, the episodic retrieval theory requires the probe-target to be previously classified as a distractor for negative priming to occur, while the feature mismatch theory stipulates that the current target must simply be present in preceding trials, as either the distractor or the target, for the development of negative priming. In fact, the episodic retrieval theory has been argued to be more closely related to the selective inhibition theory of negative priming, as both presume that the target's previous identity as an ignored distractor is the crucial element for negative priming.

Additional Variants of Retrieval-Based Negative Priming Theories

There have also been multiple derivatives of the retrieval-based theory of negative priming. For instance, Neill and Mathis (1998) proposed a new modified version of the episodic retrieval theory, applicable to numerous cognitive effects: the transfer-(in)appropriate processing (TIP/TAP) account. Though quite similar to episodic retrieval accounts of negative priming, this model is an extended account of said theory, and thereby able to be implemented to a variety of other cognitive effects beyond negative priming. This essentially states that a stimulus reintroduces processing activities that were previously applied to itself or a corresponding stimulus–observed in both implicit and explicit processing. In other words, the cognitive representation of said distractor may be facilitated in an ignored repetition trial; however, responses encoded for such items are now deemed inappropriate and thus cause interference in evaluating the target response (Fringes et al., 2015; Neill & Mathis, 1998).

Another perspective regarding negative priming is the temporal discrimination theory (Milliken et al., 1998; Milliken & Joordens, 1996). This postulates that the time required for the attentional system to determine whether the display is old (i.e., known) or new (i.e., yet unknown) is a function of the similarities between the prime and probe displays. If the prime distractor is subsequently implemented as the target in the probe, the display then holds both new and old information, slowing response times. This ambiguity may be the source of negative priming in ignored-repetition conditions. As this theory is quite similar to others, Frings et al. (2015) suggest that this account be integrated with others to construct a preferential retrieval account.

The stimulus-response retrieval theory may be seen as another variant of the episodic retrieval theory (Hommel, 1998; Hommel, 2004). This theory states that the entirety of the prime episode is consolidated into an event file in memory–including the prime target, distractors, and reaction to said target, which is then involuntarily retrieved if a formerly seen stimulus reappears. This activates a previously executed response, facilitating performance if the current and encoded responses coincide, and inhibiting it otherwise (i.e., slowing response times) (Frings et al., 2015). Though very similar, the episodic retrieval theory suggests an implicit cognitive selection of the distractor encoded with a do-not-respond tag and the target encoded with a tag indicating a response. Meanwhile, the stimulus retrieval theory makes no indication of any selection occurring in the prime display, but rather, that the entirety of the episode is encoded; this theory is distinct from those earlier theories as it does not assume selective encoding or storing to any degree (Frings et al., 2015).

Lastly, an expansion of the stimulus retrieval theory, named the binding and retrieval in action control (BRAC), is a memory-based theory that can be applied to effects beyond negative

priming. It is based on two core processes: feature binding and retrieval, and stipulates that current behaviors are molded by events in the immediate past (Frings et al., 2020). In terms of negative priming, the BRAC account claims that the prime stimuli and affiliated response are stored in an event file, which then upon the reappearance of some distractor, is retrieved along with an incompatible prime response in response to the probe display–thus causing interference (Frings et al., 2020).

Discussion of the Theories of Negative Priming

Essentially the differences in negative priming theories represent distinct predictions regarding the required aspects and processing of the prime display for negative priming to occur. For instance, selection against the distractor is necessary for priming in the distractor inhibition and episodic retrieval accounts but not in feature mismatch or temporal discriminability (Mayr & Buchner, 2007).

The feature mismatch and temporal discriminability theories lack substantial empirical evidence to be considered adequate models for negative priming (Mayr & Buchner, 2007). In terms of distractor inhibition as opposed to the episodic retrieval model, the evidence seems to lean towards the latter (Neill et al., 1992). Though Tipper's (1985) finding of semantic negative priming is better illustrated by the inhibition account, as they typically assume the capacity for neural spread expected to be present in such cognitive effects, the inhibition model fails to justify the presence of increased negative priming as a result of contextual resemblance among the prime and the probe (Fox & de Fockert, 1998; Neill, 1997; Stolz & Neely, 2001; Wong, 2000). By contrast, the episodic retrieval model suggests the representation of certain episodes, or memories, being stored, unassociated with any neural network. Episodic retrieval better

explicates the consequence of temporal discriminability in the probe trial as a result of trials prior (Neill et al., 1992).

The assumption made by inhibition accounts suggesting that probe interference is necessary for negative priming has been disproven. The formal connectionist model of inhibition proposed by Tipper et al. (1998) proposed that mental representations of stimuli compete for dominance over the response network according to their activation advantages; the probe shows reduced activation from inhibition in trials prior causing it to take longer to attain the threshold activation necessary for negative priming. This is seen in single probe conditions, where negative priming is absent as no competition is present between the cognitive representations of stimuli (Frings & Wentura, 2007). These findings can rather be rationalized as a consequence of learning strategies. Learning of prime and probe associations is likely facilitated by the absence of interference. This serves as evidence against the response blocking component of distractor inhibition accounts (Mayr & Buchner, 2007).

Inhibition theory also falls short in its rationale with the findings showing positive priming in single probe effects; this facilitation is rather explained by the blocking of the distractor stimuli being forsaken when there is no need for selection (Milliken & Joordens, 1996). Probe stimuli that are presented without interference fail to impose positive priming, disputing the credibility of distractor inhibition theory. Predominantly, the findings of the inhibition model are outdated and need to be reassessed with consideration of more recent developments in the literature (Mayr & Buchner, 2007). Further, studies have shown congruence among features does not play a significant role in the magnitude of observed negative priming, concluding the feature mismatch theory is not seen as a good fit (Mayr & Buchner, 2007).

Negative Priming and Attention-Deficit/Hyperactivity Disorder

There have been many studies looking at the diminished behavioral negative priming in individuals with ADHD (Christiansen & Oades, 2010; Curatolo et al., 2010; Gaultney et al., 1999; Gilden, 2001; Keppel & Underwood, 1962; Marriott, 1998; Mclaughlin, 2003; Ossman & Mulligan, 2003; Pritchard et al., 2006; Pritchard et al., 2008; Shin, 2005; Shin, 2006; Storm & White 2010; White, 2007). Christiansen and Oades (2010) compared response times of 35 children who were diagnosed with ADHD, 24 of their siblings who were unaffected by ADHD, and 37 other individuals between the ages of six to 17 without ADHD who were the independent controls when completing a Stroop task. They found that with the congruent condition, in children without diagnosed ADHD, there was apparent negative priming upon onset of the test. In comparison, the children with diagnosed ADHD initially displayed reduced levels of negative priming, seen to be independent of age or symptom severity, however, they performed in parallel to the control otherwise. Christiansen and Oades concluded that negative priming was initially reduced in cases of children with ADHD. Further, the negative priming effects were seen to be masked in the incongruent condition. As trials progressed, both groups showed increased facilitation from congruence among subsequent trials as opposed to response suppression from the previous trial (Christiansen & Oades, 2010). Additionally, the overall pattern of negative priming in both groups of participants across trials allowed for the conclusion that priming from the past trials increased accuracy, and that individuals with ADHD presented with abnormalities with respect to negative priming effects (Christiansen & Oades, 2010).

This reasoning is neurologically reinforced as a further indication of impaired negative priming among the experimental group as individuals with ADHD have been seen to display

impaired frontal lobe activity in regions implicated in attentional processing (Biederman, 2005; Bush, et al., 2005) This is supported by the finding that individuals with fronted lobe damage display similar inhibition of negative priming (i.e., positive priming) (Stuss et al., 1999).

A similar study assessed negative priming in individuals with ADHD over two different conceptual tasks: the Flanker test and the Stroop test. Responses were compared as a function of comorbid versus not-comorbid and type of ADHD as combined ADHD versus inattentive ADHD. Response time regarding identifying a color in an unrelated priming and ignored repetition condition were recorded. In the Flanker task, uniquely shaped color blobs were presented in 11 sets of three composed of a central target blob peripherally accompanied by two distractor blobs; there were 11 different colors for blob presentation (the same colors used in their Stroop task) with the distractor and target never appearing congruently in terms of color. The results of this study indicated that comorbidity in regards to ADHD does seem to have an effect on negative priming in specific tasks. This study showed that the not-comorbid and comorbid ADHD groups had comparable and intact negative priming in the Stroop task. Further, similar and sufficient negative priming was observed in the combined ADHD and inattentive ADHD subtypes. On the other hand, in the Flanker tasks, negative priming was only present in the not-comorbid group and absent in the comorbid ADHD group. In this task, negative priming was equally absent for both subtypes of combined ADHD and inattentive ADHD. This indicated that comorbidity in regards to ADHD does seem to have an effect on negative priming in specific tasks. This may suggest that comorbidity produced a confounding effect on the results of prior studies, potentially clarifying the reason behind the contradictory results presented in the past literature, as a majority of past studies did not control for comorbidity (Pritchard et al., 2008).

The negative priming and ADHD are seen in the literature to be quite ambiguous due to contradictory findings. The findings of Pritchard et al. (2008) may suggest that comorbidity produced a confounding effect on the results of prior studies, potentially clarifying the reason behind the contradictory results presented in the past literature, as a majority of past studies did not control for comorbidity (Pritchard et al., 2008). Previous studies showing no difference in negative priming between the ADHD and non-ADHD groups were performed using the Stroop task (Gaultney et al., 1999; Pritchard et al., 2006), in contrast to the studies which did find reduced negative priming that employed far more simplistic tasks, more like the Flanker task employed in my study (Marriott, 1998; Ossman & Mulligan, 2003). It is possible that tasks involving attentional focus culminate in selective deployment of attention in individuals with ADHD and an additional comorbid diagnosis. Further, it may be the case that the added dimensional congruences among stimuli and the semantically related (rather than based on identity) stimuli in the Stroop task may result in producing a sufficient negative priming effect in those with ADHD (Pritchard et al., 2008). Another possible factor resulting in the inconsistencies in the literature is the variability in conceptualized ADHD. As aforementioned, there are numerous frameworks for ADHD, including Barkley's (1997) Theory of ADHD, which posited that combined ADHD is primarily distinguished by behavioral deficits rather than cognitive. Opposingly, inattentive ADHD is primarily attributed to selective attention independent of behavioral inhibition. As the different subtypes of ADHD have different attributes, it is possible that the inconsistent results present in the literature are a consequence of failing to consider the ADHD subtype (Barkley, 1997).

Shin (2006) further explored this discrepancy in the literature through the use of a letter matching task (two target items were placed between distractors and participants had to identify

whether the targets were the same) and a localization task (where participants had to identify the location of a larger target placed among smaller distractors). Individuals with inattentive-type ADHD presented with higher levels of negative priming relative to the combined ADHD and control groups, in whom inhibition was seen to be additive. Additionally, those with inattentive ADHD showed a sharp release of negative priming around 1000 milliseconds which was not paralleled in the combined or control groups. Specifically, in the letter-matching task, both types of ADHD presented with a stark drop in facilitation between 500 and 1000 milliseconds whereas it remained stable for the control group as a function of the time delay between the response and stimuli interval. Contrarily, the two ADHD groups showed an abrupt build-up of facilitation between the same time interval, and the control again remained plateaued. In terms of inhibition, the data showed inattentive ADHD displayed greater negative priming than the control implying distractor information may be inhibited beyond what is required. Those with combined ADHD evidenced intact negative priming, on par with that seen in the control group. This, along with those with inattentive ADHD being unable to inhibit distractor information persistently as long as the control, can potentially be attributed to insufficient processes of inhibition. Further, equal facilitation was seen between the two ADHD subtypes, which was significantly different than that seen in the control. This may suggest that facilitation is more volatile among individuals with inattentive or combined ADHD relative to neurotypical individuals. The discerning patterns of inhibition and facilitation are congruent with Barkley's claims regarding the framework of inattentive ADHD being based in selective attention (Shin, 2006).

Those findings were established upon a previous study by Shin (2005) where a Stroop task was used to evaluate the mechanism behind cognitive inhibition in individuals with ADHD

(Shin, 2005). The stimuli presentation times were manipulated between 500 milliseconds and 1000 milliseconds. Those in the ADHD group had slower reaction times and lower accuracy than the control group. Additionally, individuals with ADHD showed reduced negative priming compared to the neurotypical participants under the 500 milliseconds time presentation condition (Shin, 2005), again consistent with Barkley's (1997) Model of ADHD. Furthermore, the ADHD group showed greater reaction times in the 500 milliseconds condition relative to the control group. Adversely, the opposite was observed within the 1000 milliseconds condition, wherein the ADHD group displayed a greater, though the difference was very small, level of negative priming compared to the control (Shin, 2005). Overall, the reaction times in the 1000 milliseconds condition were longer and thus greater than the conventional duration of inhibition in neurotypical individuals (Gilden, 2001). It was thus postulated that if children with ADHD are considered to have deficient processing, it can be concluded that they conjointly can be considered to have deficiencies in selective attention (Shin, 2005).

Fundamentally, the aforementioned studies indicate the speculation that the inhibitory theories regarding negative priming may not be a comprehensive enough explanation of the phenomena and its effects. It is possible that the mechanism fundamental to negative priming may also have roots in memory retrieval, favoring the involvement of implicit-retrieval based/selective attention theories.

Proactive Interference

The suggestion that negative priming may be a consequence of inadequate retrieval from memory in those with ADHD proposes the possibility that said individuals are also abnormally impacted by the effects of proactive interference. Essentially, in the framework of the memory-based theories of negative priming, reduced levels of negative priming for individuals

with ADHD may indicate that this group of individuals is less likely to retrieve the "do not respond" tag ascribed to distractors from past trials. Following that reasoning, individuals with ADHD may also exhibit different levels of proactive interference, defined as when older information interferes with an individual's memory for current information. Brown (1958) and Peterson and Peterson (1959) were instrumental in developing a task involving the presentation of target stimuli in triads along with some distractor task (such as counting backward), which significantly affects recall within a 20-second time interval. This task has proven to be fundamental in studying cognitive effects such as proactive interference to evaluate the immediate and delayed recall within short-term memory, as well as assess the direction of attention and information processing in working memory and executive function (Brown, 1958; Mertens et al., 2006; Peterson &Peterson, 1959).

Keppel and Underwood (1962) studied proactive interference by looking at performance as a function of how long participants had been in the experiment. They had three trials; in the first trial, participants were given a list of three consonants and then asked to count backward for three, nine, or 18 seconds. In the first trial, participants had perfect recall, there were no mistakes, and the amount of time counting backward had no effect. However, as the trials progressed, performance got worse. This can be attributed to a build-up of proactive interference. Here, the old trials are affecting the ability to remember the material introduced in the recent trial. The similarities between the presented stimuli, in this case, due to all the stimuli being letters, cause information from new and old trials to be muddled and thus cause interference. Keppel and Underwood suggested one possible explanation for this is that people have difficulty discriminating the new from the old in short-term memory, therefore in the first trial, there was no interference from trials before, resulting in the perfect recall. In the later

trials, when individuals tried to recall letters from the recent trial, they remember letters from previous trials instead, and all the old letters are interfering with remembering the new letters.

Wickens et al. (1963) proposed that triplets of words sharing homologous physical or semantic characteristics are grouped collectively in short-term memory as a single item and even further, a unique psychological class. When successive items are no longer comparative in class, the interference effect dissipates, suggesting the involvement of cognitive organization in the effects of proactive interference. They conducted a study in which there were two classes of words: those that were obviously related, and those with more complicated associations. The words were presented in triplets over sets of three to four trials in which the class of words was varied between each set. Participants showed a decrease in performance as the trials progressed. This provoked the assumption that there is a common thread involving cognitive encoding of stimuli in a certain class which is seen in a steep increase in performance between class switching. This is because there is no longer interference from old stored stimuli similarly stored due to trait congruences causing proactive interference (Wickens et al., 1963). This data indicates that greater similarity among stimuli will lead to greater interference in memory and more difficulty with retrieval from it; this finding is evidenced in the literature as well (Freidman & Reynolds, 1967; McGeoch & McDonald, 1931; Postman, 1965).

In essence, changing the class of words presented leads to a release from the proactive interference effect, indicating that inhibitory effects are a result of semantically similar items being confused in memory (Craig et al., 2013). This was also seen by Wickens and Clark (1968) in their study examining words with acoustic similarities and synonymous words in characteristically similar triplets. They found increased proactive interference (decrease in

performance) over categorically related trials and a striking increase in performance between-group shifts (Craig et al., 2013).

This leads to one plausible theory for why release from proactive interference occurs–the retrieval hypothesis. Evidence for this theory was found in a classic study done by Gardiner et al. (1972). In the study, people were told they needed to remember the information which they would be given. They were given a list of three items to remember and then given ten unrelated backwardly spelled distractor words at a rate of 1.5 seconds per word and asked to read them out loud before recalling the initial list of three words. Then, participants had a subtle category shift in their list of items on their last trial. For example, if the first three trials were cultivated flowers then the last trial would be wildflowers. They had different categories like this where participants had difficulty identifying what had changed unless they were notified of it. In their study, there were three groups of subjects. Group one was not informed of the category shift; they were the control group. In group one, the conductors of the study saw a buildup of proactive interference. Performance was seen to decrease on every single trial. Group two was informed of the change before the start of the experiment. This group showed a release in proactive interference in trial four where their performance increased dramatically. One possible explanation for their responses may be that these individuals encoded the information better, which means they were storing it into memory better as they knew about the change when they got the list of items and therefore it stood out to them. Another possibility is that they were better at the time of retrieval, meaning that the participants had all these flower names floating around in memory, and at the time of retrieval they were able to pick out the wildflowers more easily. Participants in the third group were informed of the category shift after the fourth trial but before the recall. They processed this information in the same manner as

group two, demonstrating a release from proactive interference. These results suggest the release from proactive interference is due to a benefit from the time of retrieval since this group had no encoding benefit but they performed equivalently to group two. From their data, Gardner et al. (1972) saw a buildup of proactive interference as performance decreased in over trials, resulting from the semantic similarities of the presented stimuli. It is not that the information is stored better, but rather it is just easier to retrieve.

Proactive Interference and Attention-Deficit/Hyperactivity Disorder

An analysis of the existing literature revealed no conclusive studies comparing proactive interference in neurotypical individuals and those with ADHD. In a study done by Egeland et al. (2010) individuals with different kinds of ADHD were seen to deploy significantly less efficient learning strategies compared to the neurotypical control group which was positively correlated with greater inconsistencies in memory. Participants were presented with two lists of words consisting of four sets of four semantically related words presented so that there were no categorically related items shown in sequence. List A contained four words from four semantically related categories of well-known words presented such that no two words from the same category were sequentially presented, and list B, the distractor list, contained a list of four distractor words. List A was shown over five learning trials, followed by the presentation and oral recall of the distractor list. Then, participants were asked to recall the items from the initially presented list A, once after recalling the distractor list, then again half an hour later. Specifically, proactive interference was assessed using the magnitude of the difference between response accuracy between lists A and B. Individuals with ADHD were shown to have diminished activity in the four assessed paradigms: diminished semantic clustering, retroactive interference, proactive interference, and performance for words presented in the middle of the

list. The results of this study showed decreased proactive interference in the inattentive ADHD group compared to the control, however, facilitation in performance was observed in the combined ADHD group, essentially presenting with no proactive interference. The authors hypothesize that this may be a result of either lower levels of semantic processing or possibly impaired inhibition, though it may potentially also be related to deficiencies in information retrieval (Egeland et al., 2010).

In a study done assessing learning in children with ADHD and the effect of sensory distractors, those with ADHD were found to less accurately recall information for all sections apart from the beginning in short-term memory tasks (Higginbotham & Bartling, 1993). This study employed a mixed factorial design to assess the effects of distractors on short-term memory in children with ADHD. Participants were individually tested on 30 statements of various difficulties and then asked to recite the statements to the proctor. In each trial, auditory, visual, or a combination of auditory and visual distractors were either presented or not presented at variable intervals and were randomly presented over intervals of 10 statements. The number and type of distractors were modulated to assess the sustainment of attention and distractibility. This study found that children with ADHD performed more poorly in terms of short-term recall compared to the control group in the final two blocks of 10, however, distractor presentation seemed to have a minimal effect on response accuracy. That is, those with ADHD only did slightly more poorly than those without. Essentially, as the number of blocks increased, performance in the ADHD group declined proportionally. These results may be attributable to an increase in proactive interference. However, it should be noted that this study takes a novel approach employing a methodology involving measuring the effect of unrelated distractors (such as noise from the hallway) on recollection of learned statements rather than the

Brown-Peterson task typically used in assessing proactive interference. Thus, the unique approach to the proactive interference framework in this study renders a lack of illustration of the effects of semantically similar items on impending recall. The author postulates that depreciating performance accredited to the specific sensory distractors was insignificant compared to the effect of accumulated proactive interference or item difficulty. The number and the growing complexity of the items may prioritize long-term rather than short-term memory processes (Higginbotham & Bartling, 1993).

The two main frameworks in which ADHD is considered are based on abnormalities in either working memory or behavioral inhibition. Further, one of the most prominent discrepancies between individuals with and without ADHD is seen regarding working memory (Martinussen et al., 2005; Willcutt et al., 2005). The dual-component model of working memory posits two independent components of primary (initial/implicit) memory maintenance and secondary (retrieved) memory recall. Free visual and spatial free recall tasks revealed that in individuals with ADHD, primary memory was intact while secondary memory seemed to be deficient, though both types of memory were present for visual and auditory information (Gibson et al., 2009). In replicating these findings, Raiker et al (2012) concluded that while implicit memory is intact in individuals with ADHD, retrieval from memory processes may be inadequate. It is therefore possible that the differences seen in those with ADHD may have attributes in memory, consequently affecting proactive interference and performance (Raiker et al., 2012).

In a study by White (2007), college students with and without ADHD (who had been unmedicated for at least two weeks prior) were recruited to complete spatial and semantic inhibition of return tasks in order to assess the effects of spatial and verbal proactive

interference in individuals with ADHD. In the spatial inhibition of the return task, participants were asked to indicate the target location from three possible locations as quickly and accurately as possible, where one of the locations was primed just before that trial. The semantic inhibition of return task was similarly set up, however, in this task, the prime location was marked by a flashed word, followed by a distractor word that was semantically unrelated to the prime or target. Subsequently, the target, semantically related or unrelated to the prime, was presented in a location either congruent or incongruent to the prime as either a word or a nonword. Participants were asked to indicate whether the stimulus was a word as quickly and accurately as possible. The results showed a reduction in spatial proactive inhibition (i.e. inhibition of return) but an increase in semantic proactive inhibition in those with ADHD (White, 2007). Prior researchers have suggested that locational negative priming and spatial inhibition of return may be one and the same. Both of these effects involve the emergence of inhibition when attention is redirected to an identical stimulus that has previously been attended to (Chao & Yeh, 2006; Neill & Mathis, 1998; Posner et al., 1985). This conclusion indicates that ignoring a distractor is not critical for negative priming to take place–in fact, some studies on negative priming have shown that you can get negative priming when you attend to both targets and distractors (MacDonald et al., 1999). The finding that individuals with ADHD may present reduced proactive interference (i.e. inhibition of return) could potentially suggest that these individuals may, by the same logic, display abnormal negative priming effects compared to neurotypical individuals, however, the literature remains inconclusive. Prior researchers have proposed that, though these paradigms are similar, inhibition of return relies on working memory resources while location negative priming does not (Kahan et al., 2013). Such inscrutability among the literature requires further studies to be done in support of such claims.

The Current Study

The current study examines how memory impacts those with ADHD through assessing the effects of proactive interference on individuals who have been previously diagnosed with attention-deficit/hyperactivity disorder. It is possible that if the reason that individuals with ADHD get less negative priming is due to the fact that they are less likely to retrieve prior episodes (i.e., they do not as readily retrieve the fact that they had ignored the item prior), then it is possible that these individuals may also display differing levels of proactive interference since this too is a memory-based effect that results from recently encountered stimuli. This is because if people have difficulty retrieving items that were previously ignored, ergo past episodes do not interfere as much with the current episodes, this may result in lower levels of proactive interference. To test this hypothesis, participants in the first experiment both with and without ADHD were assessed using a Brown-Peterson Task (Brown, 1958; Peterson & Peterson, 1959) with words from semantically related categories. If those with ADHD have difficulties with retrieving items from memory, it is thus possible that such individuals also display abnormal levels of proactive interference (as that is a cognitive phenomenon based on memory retrieval). Additionally, in the second experiment, a Flanker task was administered to both groups of participants to assess negative priming effects (Eriksen & Eriksen, 1974); in this experiment, negative priming is expected to find deficient negative priming in those with ADHD, and subsequently mirror the conclusions of past studies.

Experiment 1a

Method

Participants

Fifty participants were recruited from Bates College, of all genders—female (54.17%), male (41.67%), and nonbinary (4.17%)—between the ages of 18 and 22 with a mean age of 19.52 years old. This study was conducted with two groups of participants. Group one consisted of those with an ADHD diagnosis or who show significant symptoms aligned with ADHD, and group two included those without an ADHD diagnosis and those who do not show significant symptoms. Of the participants, three self-reported that they had been previously diagnosed with ADHD and the remaining 18 individuals were undiagnosed. In terms of CAARS scores, 10 individuals were categorized into the neurotypical group and 11 were assigned to the ADHD group.

Materials

Necessary instruments for this study included a computer with an attached keyboard to take the test and a quiet space without distractions.

The experimental tasks for all experiments were created and coded using the Open Sesame interface (Mathôt et al., 2012) and distributed on the corresponding OS Web desktop platform (Mathôt et al., 2012), using PsychoPy as the backend (Peirce, 2009). The studies were exported by being embedded into a Qualtrics survey containing the initial consent form and the concluding scales and remarks. The studies were hosted on and data were collected and stored in a Jatos open-source server (Lange et al., 2015), and subsequently analyzed using SPSS statistical analysis software as well as Microsoft Excel.

A list of semantically related words, which participants were asked to remember and later respond with when completing the experimental task, was obtained from a list of the commonly known English words from 56 common categories of nouns from the literature (Battig & Montague, 1969). The top 12 most common words were chosen from each of the 20 most common categories (see Appendix A).

Additionally, this investigation employed the Generalized Anxiety Disorder Assessment (GAD-7; Omani-Samani et al., 2018; Spitzer et al., 2006) (see Appendix B). Anxiety is seen to be positively correlated with the severity of ADHD symptoms; it is seen to exacerbate aspects of ADHD pathology such as impulsivity and deficits in control (Brooker et al., 2020). The GAD-7 is a seven-item measure of the severity of generalized anxiety disorder in an individual in accordance with DSM-IV criteria. This scale asks participants to rate how often they have been bothered by anxiety-related feelings or symptoms in the past two weeks on a Likert-based scale indicating the frequency of symptoms from 0 *"not at all"* to 3 *"nearly every day."* Scores can range from 0 to 21 and may be interpreted such that scores from 0 to 4 indicate no or minimal anxiety, 5 to 9 indicate mild anxiety, 10 to 14 indicate moderate anxiety, and 15 to 21 indicate severe anxiety.

Individuals were assessed on the presentation of symptoms indicative of ADHD using the Conners' Adult ADHD Rating Scales–Self-Report: Short Version (CAARS S:S; Adams et al., 2020; Conners et al., 1999; see Appendix C). Participants were asked how much or how frequently the following statements regarding behaviors or problems adults sometimes experience described them recently. This measure is a Likert-based 26-item questionnaire with responses ranging from 0 *"not at all, never"* to 3 *"very much, very frequently."* The CAARS S:S scale includes the four subscales of CAARS S:S A-Inattention/Memory Problems, CAARS

S:S B-Hyperactivity/Restlessness, CAARS S:S C-Impulsivity/Emotional Lability, and CAARS S:S D-Problems with Self-Concept, each containing 5 items, as well as a 12-item overall CAARS S:S E ADHD Index.

Procedure

This study measured the accuracy of recall, as a function of the serial position of the trial, in a list of given semantically related words. Prior to launching the study, participants were presented with a consent form wherein they were informed that individuals with a diagnosis of ADHD along with those who do not have this diagnosis are needed. They were informed that those without an ADHD diagnosis or those who are diagnosed with ADHD but not medicated should participate in the study in the morning before doing other tasks that day to control for the variability of attention and mental fatigue throughout the day as well as to equate the time of testing. Those who are taking medications for ADHD, primarily done in the morning, were asked to participate in the study one hour before the time that they normally take their medication, and then take their medication after. This was to control for the effect of treatment medication which may skew results.

Participants were asked to have a computer with an attached keyboard and to find a quiet space to complete this study. After signing a consent form and watching a brief instructional video, they were then directed to the study which was presented digitally via OSWeb. The experiment began by sequentially displaying three semantically related words at a speed of one word per second. After viewing the words, participants were asked to count backward by threes from a randomly chosen three-digit number four times to prevent rehearsal of the items prior to the recall. Participants typed in the four values they calculated into the displayed text box, separated by commas, and pressed the enter key to continue to the next trial.

Then, the participants were asked to type in the words from the previously presented triplet into a textbox, with commas between each word, and press the enter key when finished. They were directed to spell the word correctly to the best of their ability. The semantically similar triplets of words were presented in a series of four sets each from the same category (i.e., 12 words total from the category). Each participant completed 20 of these series, and both groups of participants were presented with the stimuli in the same order. The percent of words that were accurately recalled were assessed as a function of the set number and participant grouping–ADHD versus neurotypical.

After completing the OSWeb portion of the study, participants were redirected to a Qualtrics survey in which they were given the GAD-7 to assess anxiety levels. Subsequently, as many people may not have been diagnosed with ADHD but still present with manifestations of the condition (Gray et al., 2014), the CAARS S:S was administered and responses were recorded for the 26 items on the scale.

Participants were then asked to report whether they have ever received an ADHD diagnosis. Those with ADHD were also asked to report their prescription and dose, as well as their most recent intake of the medication. This information was not linked with participant names or identificatory information so all information may remain entirely anonymous, in concordance with HIPPA. Those without ADHD were also asked to report the same information regarding whether they ever utilized medications typically used for ADHD recreationally. Demographic information regarding gender, age, their highest level of education, and languages in which they were fluent were also collected.

Results

Word responses from the proactive interference study were first spell-checked and corrected according to the suggested spelling in Microsoft Excel as well as Google Sheets. They were then graded based on accuracy regardless of the order of the words presented. The percent of correct words of the three words presented in each trial were averaged for each of the four trials in a set for each participant.

CAARS responses from participants from Bates College were first analyzed by calculating the sum of scores of CAARS items corresponding to the five different subscales present within the CAARS ADHD scale. Due to technical difficulties, and the unfortunate recent passing of the author, the scoring information customarily required to grade CAARS responses proved to be quite difficult to obtain. Therefore, responses were assessed based on the five CAARS subscales, which were used to categorize participants into their respective ADHD or neurotypical groups. Of the subscales, the CAARS E ADHD Index was used to assign participants to the ADHD or neurotypical group. A median split (Mdn = 51.82) was used to separate the CAARS E scores into two groups where the higher scores were taken to indicate ADHD and the lower scores were grouped as more likely neurotypical. The Cronbach α for scale was .91, indicating high internal consistency.

GAD 7 responses were collected and responses from each item were summed. As mentioned, individuals with scores between 0 and 4 were considered to have minimal anxiety, scores from 5 to 9 indicated mild anxiety, 10 to 14 indicated moderate anxiety, and scores over 15 were seen to indicate severe anxiety. This had a Cronbach α of .93, indicating high internal consistency.

Analyzing Groups by Self-Reported Clinical Diagnosis

The data were initially analyzed by basic group membership on clinical diagnosis of ADHD versus those who identified as neurotypical. A 4 (set number: 1, 2, 3, or 4) x 2 (clinical diagnosis: diagnosed with ADHD or neurotypical) only found a significant main effect of the set on response accuracy [$F(3,57) = 6.81$, $p < .001$]. Follow-up Fisher's LSD tests revealed performance in set 1 ($M = 78.10\%$, $SD = 2.19$) was significantly better than in set 2 ($M = 72.50\%$, $SD = 2.10$) or 4 ($M = 71.20\%$, $SD = 2.44$), $p = .004$ and $p < .001$.

Analyzing Groups by Assessment on ASRS Survey

The data were analyzed by basic group membership on CAARS score indicating ADHD versus those who identified as neurotypical. A 4 (set number: 1, 2, 3, or 4) x 2 (CAARS categorization: ADHD or neurotypical) analysis of variance (ANOVA) regarding performance over the four trials showed a main effect of trial number within each set of categorically related four trials [$F(3,57) = 7.70$, $p < .001$]. Follow-up Fisher's LSD tests revealed performance in set 1 ($M = 77.80\%$, $SD = 4.70$) was significantly different than in set 2 ($M = 72.40\%$, $SD = 4.60$) or 4 ($M = 70.90$, $SD = 5.30$), $p = .001$ for both, and set 3 ($M = 75.00\%$, $SD = 4.60$) was also significantly different from set 4 ($M = 70.90\%$, $SD = 5.30$), $p = .010$. Set 1 and 2 were not significantly different, neither were set 2 and 3 or set 1 and 3. No significant main effect of clinical diagnosis [$F(1,19) = 1.01$, $p = .329$], or interaction between clinical diagnosis and trial number [$F(3,57) = 1.79$, $p = .159$] was found.

Discussion

There were several limitations of Experiment 1a which make the interpretation of the findings questionable. After data collection, the CAARS scoring manual was unfortunately unattainable, due to the recent passing of the author and limited time and resources–that is, the official purchasing of the test materials were quite expensive and needed additional resources

for scoring which were not readily available for use or easily obtained. Furthermore, the publication services were not understanding of undergraduate use of said measure and the lack of qualification. Additionally, the order of which words appeared in which set was not random, so perhaps easier words to remember just happened to be shown in the third set, which was shown to have accuracy similar to sets 1 and 2 (i.e., proactive interference did not increase linearly across sets). Finally, the example words in the instructional video were part of the experimental task stimuli, and thus participants saw those items twice during the study. Experiment 1b was conducted to fix the problems of Experiment 1a. Specifically, a more conclusive ADHD assessment was used–the Adult ADHD Self-Report Scale (ASRS) v1.1 (Kessler et al., 2005). This assessment was chosen as it is a comparably efficient assessment of ADHD yet much more simple to analyze than the CAARS. Also, the item sequence within each category, as well as the order of the categories themselves, was randomized across sets, and the words and categories which appeared in the instructional video were replaced with equal common replacements obtained from the same source as before (Battig & Montague, 1969). In other words, the categories of words were randomized in the order in which they were presented, and the words themselves within each category were additionally randomized. Lastly, because it was difficult to obtain enough participants who self-reported an ADHD diagnosis at Bates College, a new sample was obtained from an online crowdsourcing website.

Experiment 1b

Methods

Participants

In this experiment, 52 participants were recruited from Amazon Mechanical Turk of ages ranging from 21 to 66 (M = 34.94, SD = 1.32) including 30 women (57.7%), 20 men

(38.5%), and two individuals identifying as non-binary (3.8%). These participants were evenly distributed with 26 participants who self-reported as having been previously diagnosed with ADHD and 26 participants who had never received an ADHD diagnosis. ADHD and neurotypical identifying participants were recruited separately and simultaneously. In addition to reported ADHD diagnoses received, presentation of ADHD symptoms were assessed using the ASRS v1.1. A majority of the participants had completed some level of higher education (56%), with some who completed some college (16%), and most others completing high school or some GED-equivalent (28%). All participants were proficient in English.

Materials

The necessary materials required for this study are the same as that for Experiment 1a, however, the Adult ADHD Self-Report Scale (ASRS) v1.1 (see Appendix D), was used rather than the CAARS ADHD scale to assess the extent of ADHD symptoms displayed by participants.

The ASRS v1.1 is an 18-item scale composed of DSM-IV-TR criteria, six of which are seen to be predictive of symptoms synonymous with ADHD (Kessler et al., 2005). Individuals with four or more shaded boxes (as indicated on the scale) within those six items were considered to have symptoms consistent with ADHD with 68.7% sensitivity and 99.5% accuracy. Additionally, the items are categorized into two subscales indicating a preeminence of hyperactivity and inattention symptoms within an individual which are scored out of a total score of 36 for each respective subscale (Georgiopoulos et al., 2018). The Cronbach α for scale was .91, indicating high internal consistency.

The experimental task, presented using Open Sesame and Jatos as before, was nearly identical to Experiment 1a. The task was the same except in this portion of the experiment, the

order of the words within each category as well as the categories themselves were randomized. This was to ensure that there was no residual effect of word complexity on participants' responses. Additionally, the first category of words, which was briefly shown during an instructional video that the participants were required to watch prior to beginning the experiment, was exchanged for a different category of words obtained from the same list (see Appendix E) which was not in the experimental trials. This was to ensure there was no effect of the word repetition on the collected responses.

Procedure

The procedure was repeated in the same manner as done in Experiment 1a, except this time the ASRS v1.1 was used to assess ADHD rather than the CAARS.

Results

Word responses and GAD-7 scores were assessed identically to Experiment 1a. The Cronbach α for scale was .96, indicating high internal consistency. ASRS responses collected from each participant were first quantified for the number of shaded boxes, as indicated on the scale, and were marked in the first six items on the ADHD self-assessment (with a Cronbach α of .96, indicating high internal consistency). Additionally, the scores from all the items were summed in aggregate and individually based on the two independent ADHD subscales present in the ASRS (attention and hyperactivity).

Analyzing Groups by Self-Reported Clinical Diagnosis

The data were first analyzed by basic group membership on self-reported clinical diagnosis of ADHD versus those who identified as neurotypical. A 4 (serial trial number in a set: 1, 2, 3, or 4) x 2 (clinical diagnosis: diagnosed with ADHD or neurotypical) ANOVA on response accuracy across the four trials in each set revealed no significant interaction between

trial number and clinical diagnosis [$F(3, 147), = .403$, $p = .751$] and no significant main effect of clinical diagnosis [$F(3, 147) = .31$, $p = .580$]. A significant main effect of trial number within a set was found on response accuracy, $F(3, 147) = 4.41$, $p = .005$. Fisher's LSD post hoc tests revealed performance across trial 1 ($M = 92.96\%$, $SD = 1.60$) was significantly higher than in trial 3 ($M = 91.26\%$, $SD = 1.60$; $p = .045$) and 4 ($M = 90.18\%$, $SD = 1.59$; $p < .001$), , and accuracy on trials 2 ($M = 91.92\%$, $SD = 1.37$) was significantly higher than trials 3 ($M = 91.26\%$, $SD = 1.60$; $p = .022$) . No significant difference was found between trials 2 and trials 1 or 3.

A Bayesian analysis was calculated to quantify the evidence in favor of the null hypothesis compared to other alternative hypotheses (Jarosz & Wiley, 2014; Masson, 2011; Wagenmakers, 2007). This was conducted using the Jeffreys's Amazing Statistics Program (JASP) software (JASP Team, 2018), according to Wagenmakers et al. (2007) (see Table 1). The BF_{10} column indicates that the data best supports the model that includes only a main effect of trial number; the data are 5.73 times more likely to result under this model compared to the null hypothesis ($BF_{10} = 5.73$). In addition, a model with two main effects and an interaction is 20.46 times less likely than the model with a main effect of trial number (5.73/.28=20.46).

Analyzing Groups by Assessment on ASRS Survey

The data were then analyzed by basic group membership on ASRS scores indicating ADHD versus those who identified as neurotypical. There was a high overlap between self-reported diagnosis and assessment based on ASRS scores. However, the correspondence was not perfect: 6 of the 26 participants identifying as neurotypical were assigned to the ADHD group by their responses on the ASRS, and 5 of the 26 participants self-reporting being diagnosed with ADHD were categorized as neurotypical based on their ASRS responses. Thus,

in my analysis, there were 27 participants in the ADHD group and 25 participants in the neurotypical group, as per the ASRS. The analyses were recalculated based on the ASRS assessment, which has been shown to be an equivalently valid measure of ADHD (Georgiopoulos et al., 2018; Kessler et al., 2005).

A 4 (serial trial number in a set: 1, 2, 3, or 4) x 2 (ASRS categorization: ADHD, neurotypical) ANOVA showed a significant main effect of set on response accuracy across the four trials, $F(3,147) = 4.41$, $p = 0.005$ (Figure 1). Pairwise comparisons using Fisher' LSD showed accuracy in both groups in trial 1 ($M = 93.04\%$, $SD = 1.52$) was significantly higher than that in trials 3 ($M = 91.32\%$, $SD = 1.50$, $p = .045$) or 4 ($M = 90.24\%$, $SD = 1.51$, $p < .001$, and accuracy in trial 2 ($M = 91.96\%$, $SD = 1.31$) was significantly higher than in trial 4 ($M = 90.24\%$, $SD = 1.51$, $p = .023$). There was also a main effect of diagnosis, $F(1,49) = 6.49$, $p < .001$. Those categorized by their ASRS scores showing symptoms of ADHD tended to generally score lower ($M = 88.13\%$, $SD = 1.93$) compared to those categorized as neurotypical ($M = 95.15\%$, $SD = 1.97$). Parallel to the effects seen with the clinical neurological diagnoses, no interaction was found between set and group, $F(3,147) = .37$, $p = .776$.

A Bayesian analysis was again calculated to quantify the evidence in favor of the null hypothesis compared to other alternative hypotheses (Jarosz & Wiley, 2014; Masson, 2011; Wagenmakers, 2007). The BF_{10} column indicates that the data best supports the model that includes a main effect of trial number and ASRS categorization; the data are 18.59 times more likely to result under this model compared to the null hypothesis ($BF_{10} = 18.59$).

Given this, however, a major determinant of the ambiguity in past studies is proposed to be due to failure to consider the different subtypes of ADHD and the effects of stimulant medications (Shin, 2005). The subtypes of ADHD may entail different mechanisms behind the

cognitive processing deficiencies present in such individuals, therefore, the participant ASRS responses were used to sort participants into four different groups: combined-ADHD, inattentive-ADHD, hyperactive-ADHD, and neurotypical. Additionally, this data were covaried with whether or not the participant had taken stimulant medications as they have been seen to reduce the reaction time variability in those with ADHD (Shahar et al, 2016). Furthermore, comorbidity of ADHD with anxiety symptoms has also been shown to have a significant impact on the performance of individuals with ADHD in such tasks, and thus was additionally assessed as a covariate (Pritchard et al., 2008).

No participants had more than five of the critical boxes marked in the ASRS questions indicating hyperactivity dominant ADHD, however, 21 individuals marked over five of the critical boxes for the ASRS items indicating inattentive ADHD, and 9 individuals showed both indicating combined-ADHD. A 4 (serial trial number in a set: 1, 2, 3, or 4) x 3 (ASRS categorization: neurotypical, inattentive ADHD, combined ADHD) showed main effects of set [$F(3,144), = 5.82, p = <.001$] and of ASRS categorization [$F(2,48), = 3.33, p = .044$]. Post hoc tests showed accuracy in both groups in trial 1 ($M = 93.04\%$, $SD = 1.52$) was significantly higher than that in trials 3 ($M = 91.32\%$, $SD = 1.50$, $p = .045$) or 4 ($M = 90.24\%$, $SD = 1.51$; further, neurotypical individuals were seen to perform the best ($M = 94.37\%$, $SD = 7.17$, $p = .297$), followed by those with inattentive ADHD ($M = 89.27\%$, $SD = 11.12$, $p = .051$), with the combined ADHD group doing the worst ($M = 85.28\%$, $SD = 17.23$, $p = .635$). However, there was no interaction found between the set number and ASRS categorization [$F(3,144), = 1.36, p = .233$]. This was maintained when covaried with medication [set: $F(3,141), = 4.57, p = .004$; group: $F(2,47), = 4.52, p = .016$; $F(6,141), = 1.76, p = .112$]. However, when covaried with

anxiety, all significant main effects disappeared [set: $F(3,141), = 2.05$, $p = .110$; group: $F(2,47), = 1.05$, $p = .356$; interaction: $F(6,141), = 1.14$, $p = .345$].

A Pearson's *r* showed that there was a significant negative correlation between average performance over the four trials within a set and GAD-7 scores, indicating anxiety level, $r(49) = -.36$, $p = .01$ (two-tailed), as well as with the number of shaded boxes marked in the critical first six items of the ASRS scale $r(49) = -.34$, $p = .02$ (two-tailed), the number of shaded boxes on ASRS items indicating inattention, $r(49) = -.37$, $p = .01$ (two-tailed), hyperactivity, $r(49) = -.35$, $p = .01$ (two-tailed), and aggregate ASRS scores, $r(49) = -.38$, $p = .01$ (two-tailed).

Discussion

Past research exploring differences in proactive interference and ADHD has presented rather inscrutable findings. This study aimed to investigate the differences in cognitive processing between individuals with and without ADHD specifically regarding differences in negative priming and proactive interference. Supposing that individuals with ADHD exceed neurotypical individuals in retrieving previously ignored items from memory, in this study I evaluated whether those with ADHD may also display abnormal levels of proactive interference–as that may also be attributed to a memory phenomenon. To evaluate this, proactive interference was measured with a Brown-Peterson task, in which I expected to see an interaction where proactive interference differed for the two groups, similarly to findings seen in other studies (Egeland et al., 2010; White, 2007). Additionally, performance was expected to decrease across the trials in each set to indicate effective proactive interference as the initial trials in the set interfere with the recent ones.

In both Experiments 1a and b performance accuracy on the first set of each category was significantly better than that on the last set of each category. The drop in accuracy over

subsequent trials suggests an apparent effect of proactive interference, as expected. The semantically related information from the former trials was being confused in memory with more recent information from the later trials, causing decreased recall accuracy as the trials progressed. This ensured that performance in the Brown-Peterson task arrangement in this study effectively reflected proactive interference levels across all individuals. Similarly, performance was also affected by group categorization where those with ADHD generally performed worse than those without, when group categorization was based on the ASRS. In terms of ADHD symptoms, greater indications of ADHD and anxiety were associated with worse performance in terms of response accuracy. This can suggest the deficiencies associated with ADHD and anxiety disorders are related in some capacity to proactive interference or its mechanisms. Contrary to my hypothesis, however, those with ADHD, regardless of where participants were recruited from, did not display a significantly different increase in proactive interference than controls. This finding contradicts the conclusion from past studies, which have found differences in proactive interference (Egeland et al., 2020; White, 2007), though those studies did not measure proactive interference in a traditional Brown-Peterson task.

Conducting a Bayesian analysis showed that a model including a main effect of both set and ADHD category is indeed the best fit for the data collected (being nearly 20 times more likely than the null hypothesis and higher than the likelihood of other potential models). This indicates that performance was seen to be significantly different as a factor of the trial number in each set and the ADHD group of the participant. Contrary to my predictions, participants did not show a difference in proactive interference between the ADHD and control groups

Further, accounting for ADHD again according to the ASRS subscale scores reflected the same pattern of significance. There was no change in significant effects with the additional

covariate of ADHD medication. However, when anxiety was added as a covariate, all significance was lost. This indicates that the difference observed between the groups was an effect of anxiety symptoms rather than reflecting differences due to ADHD symptoms. This further emphasized that the performance differences between the groups may not be due to ADHD deficits but might be due to anxiety symptoms of those who had the additional comorbidity.

In essence, this showed proactive interference was intact among those with and without ADHD deficits–regardless of how the analysis was conducted. As the trials progressed within a set and more semantically similar items were added, performance in both groups declined. These findings, however, contradict past research where those with inattentive ADHD performed worse than the controls, and the combined-ADHD groups showed proactive interference to be nonexistent (Egeland et al., 2020). In my study, those with combined ADHD had the worst performance (i.e., greatest proactive interference). It should be considered that in the study conducted by England et al. (2020) the target recall items were encoded at a far deeper level. The target list of words was rehearsed multiple times prior to the presentation of the distractor and recall task and ergo processed into long-term memory. By contrast, my study only required participants to hold the items in short-term memory prior to recall and were thus cognitively processed at a much lower level. Proactive interference has been considered by some to be the consequence of deep-level encoding (Egeland et al., 2020), and therefore it is possible that the level of encoding evoked in my study was not adequate enough to be sensitive to proactive interference differences in those with and without ADHD. As such, the reasoning behind these oppositional findings continues to remain uncertain, nevertheless, it is possible that

the level of processing plays a role in the distinction and will require further investigation before any conclusions may be drawn.

It is also possible, however, that the factors of sensory modality and/or distractor-target congruence are responsible for these contradictions. Studies that concluded negative priming discrepancies among the ADHD and non-ADHD groups also involved other presentation or response modalities which may justify the similarity in proactive interference observed in my study (Egeland et al., 2020; Higginbotham & Bartling, 1993; White, 2007). The study by Egeland et al. (2020) involved oral presentation and rehearsal of the words; Higginbotham and Bartling (1993) presented sentences orally for participants to verbally recall; White (2007) saw a significant decrease in proactive interference in only the spatial domain, and a significant increase in the semantic domain, indicative of the fact that the mechanisms behind proactive interference across the two domains are related but independent of one another. Hence, it is possible that though semantic proactive interference is the same among the two groups, there are significant differences in proactive interference lie in other domains; however, further studies are necessary to examine whether modality affects proactive interference levels in those with ADHD. It should be further noted that these studies employed very different methodologies from my own and therefore are targeting different underlying cognitive mechanisms and producing different results.

It had been predicted that if the reason prior studies found that individuals with ADHD have shown less negative priming is caused by episodic retrieval issues, then these individuals might exhibit less proactive interference since this too is a memory-based effect that results from retrieving prior information. However, the results of Experiment 1 showed that proactive interference, as assessed using a Brown-Peterson task, was not diminished in the ADHD group.

Perhaps the reason for this discrepancy is that the reduced negative priming seen in those with ADHD may be dependent on ignoring distracting information (Frings, 2015), whereas distracting information is not part of a Brown-Peterson task. If distraction is important, then participants with ADHD should still exhibit reduced negative priming compared to controls, even if they show the same amount of PI. Because the negative priming findings with a Stroop task have not always shown group differences (Gaultney et al., 1999; Pritchard et al., 2006), Experiment 2 used a flanker (Eriksen & Eriksen, 1974) task modeled after Ossmann and Mulligan's (2003) study with ADHD and control groups, though the task was changed from pronunciation to keypress to facilitate online data collection.

Experiment 2

Methods

Participants

Participants (N = 52) were once again recruited from Amazon Mechanical Turk in a congruent manner to Experiment 1b. Participants included 30 men (57.69%), 21 women (40.39%) and 1 nonbinary (1.92%). In terms of clinical diagnosis, 27 participants reported being diagnosed with ADHD, and 25 reported being neurotypical.

Materials

The required materials for this portion of the experiment were nearly identical to that needed in Experiment 1b. Participants from MTurk were administered the ASRS v1.1 (with a Cronbach α of .96, indicating high internal consistency) to assess ADHD symptoms and GAD-7 (with a Cronbach α of .96, indicating high internal consistency) to indicate anxiety levels. The study was presented in Qualtrics with an embedded Open Sesame study, just as before, however, in this case, a flanker task was administered rather than a Brown-Peterson task.

Procedure

A flanker task (Eriksen & Eriksen, 1974) was administered in which the words *stone*, *clock*, *match*, and *label* were randomly displayed in 40-point Arial bold font and all capital letters in pairs. One of the words in the pair was presented in red pixels on the display screen and the other was presented in green pixels, centered horizontally with one word placed 20 pixels above the center and the other 20 pixels below. Participants were randomly assigned to one of two conditions where either the green pixels signified a target word and the red pixelated word in the pair signified the distractor word or vice versa (where the red pixel word was the target and the green pixel word was the distractor). Each word was paired equally often with every other word, in each location (top or bottom), and in each color. Participants were asked to respond to the target word by indicating its identity using the S, C, M, or L keyboard keys for *stone*, *clock*, *match*, and *label*, respectively, as quickly and accurately as possible. Participants completed 6 practice trials in addition to 288 experimental trials. After completing the flanker task, participants were directed back to the Qualtrics survey, where they responded to the same questionnaire items as Experiment 1b.

Results

Responses were graded based on accuracy in the control and ignored repetition conditions. Accuracy rates and response times across the ADHD and neurotypical groups were assessed in the two conditions. ADHD and neurotypical categorization according to the ASRS and assessment of anxiety symptoms with the GAD-7 were assessed as done before in part 1 of this study. Geometric means were used to reduce the effects of outliers on the reaction time data.

Analyzing Groups by Self-Reported Clinical Diagnosis

The data were initially analyzed by the participant's reported diagnosis indicating ADHD versus those who identified as neurotypical. A 2 (clinical diagnosis: ADHD or neurotypical) x 2 (trial condition: control or ignored repetition) mixed ANOVA conducted on response accuracy showed no significant effect of negative priming [$F(1,50) = .63$, $p = .432$], diagnosis [$F(1,50) = 1.75$, $p = .169$], or interaction between the two [$F(1,50) = .09$, $p = .771$].

For reaction times, a 2 (clinical diagnosis: ADHD, neurotypical) x 2 (trial condition: control, ignored repetition) mixed ANOVA again showed no significant effect of negative priming [$F(1,50) = 2.94$, $p = .093$], diagnosis [$F(1,50) = 3.00$, $p = .089$], or interaction [$F(1,50) = .03$, $p = .858$].

Analyzing Groups by Assessment on ASRS Survey

The data were next analyzed by basic group membership on ASRS scores indicating ADHD versus those who identified as neurotypical. Again, there was an imperfect overlap between self-reported diagnosis and assessment based on ASRS scores: 6 of the 26 participants identifying as neurotypical were also assigned to the ADHD group based on ASRS responses, and 5 of 26 participants identifying as having ADHD were assigned to the neurotypical group based on ASRS responses. As such, in my analysis, there were 18 participants in the ADHD group and 34 participants in the neurotypical group. A 2 (ASRS categorization: ADHD or neurotypical) x 2 (trial condition: control or ignored repetition) mixed ANOVA conducted on response accuracy showed no significant interaction between negative priming displayed and ASRS categorization [$F(1,50) = .62$, $p = .435$], and no significant main effect of negative priming [$F(1,50) = 1.02$, $p = .317$]. However, there was a significant main effect of ASRS categorization [$F(1,50) = 12.15$, $p = .001$]. The means are displayed in Figure 2. When covaried with GAD-7 anxiety scores no change in significance was seen. There was still no significant

interaction, [$F(1,50) = 1.86$, $p = .178$], or main effect of negative priming, [$F(1,50) = 2.79$, $p = .101$] and there was a significant main effect of ASRS categorization, [$F(1,50) = 11.04$, $p = .002$].

A 2 (ASRS categorization: ADHD or neurotypical) x 2 (trial condition: control or ignored repetition) mixed ANOVA conducted on reaction times similarly showed no significant interaction between ASRS categorization and negative priming [$F(1,50) = .17$, $p = .683$], and no significant main effect of negative priming [$F(1,50) = 2.29$, $p = .136$], though there was a marginally significant main effect of ASRS categorization, [$F(1,50) = 3.76$, $p = .058$]. The means are displayed in Figure 3. When covaried with GAD-7 scores, all significance was eradicated; that is, there was no significant main effect of negative priming [$F(1,49) = .08$, $p = .778$], or ASRS categorization [$F(1,49) = .13$, $p = .725$], and there was no significant interaction between the two, [$F(1,49) = .53$, $p = .470$].

As my predictions for this study specifically related to negative priming being apparent in the neurotypical group but not the ADHD group, even though no significant interaction was seen, separate *t*-tests were conducted on each respective group to independently observe the effects of negative priming. A paired samples *t*-test revealed that reaction times for neurotypical individuals was significantly lower in the control condition ($M = 718$, $SD = 143.08$) as compared to the ignored repetition condition ($M = 729$, $SD = 154.29$), $t(33) = -2.10$, $p = .043$ (two-tailed). However, a paired samples *t*-test revealed for individuals with ADHD revealed reaction times for the controlled condition ($M = 816$, $SD = 214.46$) were not significantly different from the ignored repetition condition ($M = 823$, $SD = 199.02$), $t(17) = -.51$, $p = .307$ (two-tailed).

Discussion

Past studies have shown individuals with ADHD may display reduced negative priming (Christiansen & Oades, 2010; Curatolo et al., 2010; Gaultney et al., 1999; Gilden, 2001; Keppel & Underwood, 1962; Marriott, 1998; Mclaughlin, 2003; Ossman & Mulligan, 2003; Pritchard et al., 2006; Pritchard et al., 2008; Shin, 2005; Shin, 2006; Storm & White 2010; White, 2007). As such, in my study, individuals with ADHD were expected to present lower levels of negative priming relative to the neurotypical controls.

Results from the negative priming task in terms of response accuracy showed there was no effect of the negative priming condition and there was a significant difference in performance based on participant group categorized by ASRS scores. This means that though I did not see a significant negative priming effect in the ANOVA, neurotypical individuals still had significantly more accurate responses overall compared to those with ADHD. This effect maintained significance even when anxiety was added as a covariate, supporting the conclusion that these results were indeed a result of ADHD symptoms rather than a confounding effect from anxiety. Opposingly, when reaction times were assessed, again only ASRS group had a significant effect, however, this significance was eradicated when anxiety was added as a covariate. This suggests that the disparity in reaction times between the two groups may be attributable to anxiety rather than being an effect of ADHD.

Even though no significant interaction was observed between ASRS categorization and negative priming, further investigation into this relationship showed that when the two groups are analyzed independently, neurotypical individuals do display a significant negative priming effect while those with ADHD do not. It is possible that this juxtaposition in findings is a power issue within the statistical analysis itself. Essentially this shows that negative priming differences between those with and without ADHD remain elusive.

General Discussion

Results from Experiment 1 clearly demonstrate that the ASRS was effective in identifying individuals who differ on ADHD symptomatology, as evidenced by the fact that the two groups differed on overall accuracy levels. In addition, the methodological design used here allowed me to replicate proactive interference as both groups performed significantly worse on later trials within a semantic category compared to earlier trials. However, the data also clearly indicate that the amount of proactive interference does not differ significantly across these two groups of participants. Equivalence in proactive interference was supported by a Bayesian analysis. As such, if there is a difference in the amount of negative priming exhibited by these two groups, as some prior studies have found, then the mechanism responsible for that is not the same mechanism that causes proactive interference. This possibility is consistent with the idea that negative priming is caused by inhibition rather than memory retrieval, or if it is caused by memory retrieval, it is caused by a different memory mechanism. However, another possibility is that prior reports of differences in negative priming are spurious and not replicable. Data from Experiment 2 are inconclusive on this point. On the one hand, the interaction between negative priming and group failed to reach significance. On the other hand, the pattern of data is going in the right direction, and the individual *t*-tests support the idea that the reduction in negative priming might replicate. Future research should continue to examine negative priming in ADHD. I would recommend using a flanker task with a pronunciation task rather than keypress, and a larger sample size. Overall, what has been learned is that these two groups do not differ on proactive interference.

One interesting finding regarding the classification of ADHD across all three studies was the reasoning behind the differing effects among the analyses conducted with the ASRS

categories vs. those with a self-identified diagnosis is uncertain. It is possible that those who have been diagnosed with ADHD may no longer exhibit symptoms as they might have prior to receiving any treatment (such as therapy or medication), ergo leading them to no longer be characterized as having ADHD by the ASRS assessment. Similarly, it is likely that some of those who identified as neurotypical yet are characterized as having ADHD by the ASRS have simply not been officially diagnosed. Therefore, perhaps the ASRS categorization more aptly captures those who currently have symptoms of ADHD and those who do not, though further investigation into this difference is needed before any conclusions may be reached.

This statistical pattern of effects is mirrored in multiple studies in the past regarding negative priming and ADHD (Hasher et al., 1991; Ossmann & Mulligan, 2003). The current study tenuously found a reduction in negative priming for individuals with ADHD using a flanker task. Given that prior experiments have not always found this reduction in negative priming, and the marginal nature of the results found here, the current study further adds to the elusive nature of this group difference. Future research should examine whether these inconsistent results are affected by the interstimulus interval during the negative priming task (see Shin, 2006; Tipper 1987) or ADHD subtypes (see Shin, 2005).

These findings may be considered as support towards Barkley's (1997) Inhibition Theory of ADHD. In the proactive interference study, though I saw no difference in proactive interference effects, those with ADHD showed worse performance on the short-term recall task overall. According to the Inhibition Theory, those with ADHD display insufficient inhibitory processes, which would suggest perhaps that those with ADHD are worse at inhibiting other distractors unrelated to the task (such as environmental distractors or wandering thoughts). This in turn may cause a decrease in accuracy overall compared to a neurotypical group. Similarly, in

the negative priming study, if those with ADHD are insufficient at inhibiting, they may have an easier time responding to the ignored repetition trials. The tenuous lack of negative priming in those with ADHD may be because the mental representation of the previously ignored distractor is not being adequately inhibited in the current trial which means that those with ADHD can respond to them quicker, as opposed to neurotypical individuals who are inhibiting the distractor interfering with them executing the appropriate response as efficiently as the ADHD group. If inhibition is the problem, the findings of the current study suggest that ADHD deficits may be a consequence of inadequate inhibitory processes, supporting Barkley's Theory of ADHD. Of course there's still the possibility that the marginal reduction in negative priming for the ADHD group is due to memory retrieval issues that are operating differently when effort is put into retrieving something, like in the proactive interference study, as opposed to the negative priming study where the aim is not to retrieve the distractor identity. This explanation doesn't seem related to Barkley's theory.

The current study adds to the growing literature regarding the underlying mechanism of negative priming and illustrates the specificities involved with ADHD. Going forward, this area of study will of course require more research as psychologists continue to learn more about the field and how memory works. Further research may involve investigating things like cognitive differences in individuals with ADHD on and off stimulant medications, the effects of task selection on negative priming levels (such as using the Stroop task versus the flanker task), as well as the suggestions mentioned earlier in this thesis. Additionally, further studies involving the addition of physiological assessments (such as EEG or fMRI data) can help provide a more comprehensive picture of the differences in those with ADHD, especially as many of the theories behind ADHD as well as the cognitive effects examined (i.e., proactive interference

and negative priming) are associated with brain regions. It would also be of interest to study whether there are any parallel effects seen regarding proactive interference levels in other populations implicated to have reduced negative priming, namely: schizophrenics (Park et al., 1996), schizotypals (Albertella et al., 2016), individuals with high cognitive failures (Tipper & Baylis, 1987), individuals with low Tellegen absorption (Campbell & Raz, 2012), individuals with low working memory (Ortells et al., 2016), children (Viarouge et al., 2019), elderly not tested in the morning (Hsu et al., 1997), and regular marijuana users (Albertella et al., 2016).

Limitations

One limitation of this study is the method of data collection. As the data were all collected online, participants were required to respond to the task via keypress. This meant that participants were not able to pronounce task items; a method that has been employed in past studies (Egeland et al., 2020; Higginbotham & Bartling, 1993). It is possible that this leads to greater variability within the results than would be in vocal response times in a lab. This may lead to the unreliability within the data and thus inaccuracies in the results.

Another potential limitation of this study was the lack of assessment of working memory. Though working memory deficits are not intrinsic to ADHD impairments, it has been postulated that those with ADHD have many deficits in working memory, particularly in terms of the central executive function (Kofler et al., 2020). Working memory also seems to be responsible for many of the cognitive abnormalities of individuals with ADHD (Bunge et al., 2001; Kane & Engle, 2000; Kasper et al., 2012; Postle & Brush, 2004). Further, those with greater working memory capacities are seen to be more susceptible to the effects of proactive interference (Rosen & Engle, 1997); individual differences in working memory load and capacity are seen to be modulators of negative priming effects (Pritchard et al., 2008). As such,

it is possible that the decreased accuracy of the ADHD group observed in the current study may be an effect of individual differences in working memory among these individuals rather than fully being a retrieval or inhibition issue. Given this, future research regarding differences in proactive interference and negative priming in those with ADHD should be conducted with the added consideration of working memory capacity.

Intelligence has also been seen to have a wide range of effects on multiple aspects of cognition such as recall, inhibition, and working memory capacity (Egeland et al., 2010). Disparities in IQ and working memory, therefore, may lead to findings that do not independently reflect ADHD deficits. Learning disabilities were also unaccounted for in this study and may have consequences on the ability of participants to rapidly read and store the stimuli information. This can in turn skew response accuracy in tasks such as the Brown-Petterson task. And lastly, age was inadvertently not assessed among participants who were recruited from Amazon Mechanical Turk in experiment 2, which can also have an effect on baseline cognitive performance. Given this, it is recommended that future research controls for IQ, participant age, and learning disorders in order to more clearly understand the potential cognitive differences in memory in those with ADHD.

Conclusion

Though the specific mechanisms remain inconclusive, those with ADHD do indeed exhibit differences in memory in some capacity compared to neurotypical people. The current study proposes that the cognitive mechanisms behind proactive interference and negative priming are indeed distinct. This possibly suggests that negative priming may not be a complication with memory retrieval, or if it is caused by memory retrieval, it is caused by a different memory mechanism, though further evidence is necessary before any such claims can

be made. It is recommended that future research be conducted while controlling for individual differences in working memory, IQ, age, and other potential disabilities as well as assessing additional factors like presentation modalities and different interstimulus intervals. The current study and future research give psychologists a better understanding of the inner workings of memory and attention-deficit/hyperactivity disorders. This research will provide a deeper understanding of inhibition and recall processes in memory, and how they may differ among individuals.

References

Adams, Z., Adams, T., Stauffacher, K., Mandel, H., & Wang, Z. (2020). The effects of inattentiveness and hyperactivity on posttraumatic stress symptoms: Does a diagnosis of posttraumatic stress disorder matter? *Journal of Attention Disorders, 24*(9), 1246–1254. https://doi.org/10.1177/1087054715580846

Albertella, L., Le Pelley, M. E., & Copeland, J. (2016). Frequent cannabis use is associated with reduced negative priming among females. *Experimental and Clinical Psychopharmacology*, *24*(5), 313–319. https://doi.org/10.1037/pha0000085

Aman, C. J., Roberts Jr, R. J., & Pennington, B. F. (1998). A neuropsychological examination of the underlying deficit in attention deficit hyperactivity disorder: Frontal lobe versus right parietal lobe theories. *Developmental Psychology*, *34*(5), 956.

American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 5th edition. Arlington, VA., *American Psychiatric Association*, 2013.

Arnsten, A. F., Steere, J. C., & Hunt, R. D. (1996). The contribution of alpha 2-noradrenergic mechanisms of prefrontal cortical cognitive function. Potential significance for attention-deficit hyperactivity disorder. *Archives of General Psychiatry*, *53*(5), 448–455. https://doi.org/10.1001/archpsyc.1996.01830050084013

Baddeley, A. (1992). Working memory. *Science*, *255*(5044), 556-559.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121*(1), 65–94. https://doi.org/10.1037/0033-2909.121.1.65

Barkley, R. A., Grodzinsky, G., & DuPaul, G. J. (1992). Frontal lobe functions in attention deficit disorder with and without hyperactivity: A review and research report. *Journal of Abnormal Child Psychology*, *20*(2), 163-188.

Barnett, R., Maruff, P., Vance, A., Luk, E. S., Costin, J., Wood, C., & Pantelis, C. (2001). Abnormal executive function in attention deficit hyperactivity disorder: The effect of stimulant medication and age on spatial working memory. *Psychological Medicine*, *31*(6), 1107-1115.

Battig, W. F., & Montague, W. E. (1969). Category norms of verbal items in 56 categories a replication and extension of the Connecticut category norms. *Journal of Experimental Psychology*, *80*(3, Pt.2), 1–46. https://doi.org/10.1037/h0027577

Biederman, J. (2005). Attention-deficit/hyperactivity disorder: A selective overview. *Biological Psychiatry, 57*, 1215-1220.

Bledsoe, J. C., Semrud-Clikeman, M., & Pliszka, S. R. (2013). Anterior cingulate cortex and symptom severity in attention-deficit/hyperactivity disorder. *Journal of Abnormal Psychology*, *122*(2), 558–565. https://doi.org/10.1037/a0032390

Brooker, R. J., Moore, M. N., Van Hulle, C. A., Beekman, C. R., Begnoche, J. P., Lemery-Chalfant, K., & Goldsmith, H. H. (2020). Attentional control explains covariation between symptoms of attention-deficit/hyperactivity disorder and anxiety during adolescence. *Journal of Research on Adolescence: The Official Journal of the Society for Research on Adolescence*, *30*(1), 126–141. https://doi.org/10.1111/jora.12506

Booth, J. R., Burman, D. D., Meyer, J. R., Lei, Z., Trommer, B. L., Davenport, N. D., ... & Marsel Mesulam, M. (2005). Larger deficits in brain networks for response inhibition

than for visual selective attention in attention deficit hyperactivity disorder (ADHD). *Journal of Child Psychology and Psychiatry*, *46*(1), 94-111.

Breen, M. J. (1989). Cognitive and behavioral differences in ADHD boys and girls. *Journal of Child Psychology and Psychiatry*, *30*(5), 711-716.

Brown, J. (1958). Some tests of the decay theory of immediate memory. *Quarterly Journal of Experimental Psychology*, *10*(1), 12-21.

Bunge, S. A., Ochsner, K. N., Desmond, J. E., Glover, G. H., & Gabrieli, J. D. (2001). Prefrontal regions involved in keeping information in and out of mind. *Brain*, *124*(10), 2074-2086.

Bush, G., Valera, E. M., & Seidman, L. J. (2005). Functional neuroimaging of attention-deficit/hyperactivity disorder: A review and suggested future directions. *Biological Psychiatry, 57*, 1273-1284.

Cairney, S., Maruff, P., Vance, A., Barnett, R., Luk, E., & Currie, J. (2001). Contextual abnormalities of saccadic inhibition in children with attention deficit hyperactivity disorder. *Experimental Brain Research, 141(4)*, 507-518.

Campbell, N. K., & Raz, A. (2012). Subsidiary analysis of different Stroop-embedded negative priming trials. *Consciousness and Cognition*, *21*(3), 1586–1590. https://doi.org/10.1016/j.concog.2012.05.009

Carter, C. S., Krener, P., Chaderjian, M., Northcutt, C., & Wolfe, V. (1995). Abnormal processing of irrelevant information in attention deficit hyperactivity disorder. *Psychiatry Research*, *56*(1), 59-70.

Castellanos, F. X. (1997). Toward a pathophysiology of attention-deficit/hyperactivity disorder. *Clinical Pediatrics, 36*(7), 381-393.

Chao, H.-F., & Yeh, Y.-Y. (2006). Inhibition of return lasts longer at repeatedly stimulated locations than at novel locations. *Psychonomic Bulletin & Review*, *13*(5), 896–901. https://doi.org/10.3758/BF03194016

Christiansen, H., & Oades, R. D. (2010). Negative priming within a stroop task in children and adolescents with attention-deficit hyperactivity disorder, their siblings, and independent controls. *Journal of Attention Disorders, 13*(5), 497-504. doi:10.1177/1087054708325974

Conners, C. K., Erhardt, D., & Sparrow, E. (1999). *CAARS: Conners' Adult ADHD Rating Scale.* North Tonawanda, NY: Multi-Health Systems.

Craig, K. S., Berman, M. G., Jonides, J., & Lustig, C. (2013). Escaping the recent past: which stimulus dimensions influence proactive interference?. *Memory & Cognition, 41*(5), 650–670. https://doi.org/10.3758/s13421-012-0287-0

Curatolo, P., D'Agati, E., & Moavero, R. (2010). The neurobiological basis of ADHD. *Italian Journal of Pediatrics*, *36*(1), 79. https://doi.org/10.1186/1824-7288-36-79

D'Agati, E., Casarelli, L., Pitzianti, M. B., & Pasini, A. (2010). Overflow movements and white matter abnormalities in ADHD. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *34*(3), 441–445. https://doi.org/10.1016/j.pnpbp.2010.01.013

Dalen, L., Sonuga-Barke, E. J., Hall, M., & Remington, B. (2004). Inhibitory deficits, delay aversion and preschool AD/HD: Implications for the dual pathway model. *Neural Plasticity, 11*(1-2), 1-11.

Durston, S., Tottenham, N. T., Thomas, K. M., Davidson, M. C., Eigsti, I. M., Yang, Y., Ulug, A. M., & Casey, B. J. (2003). Differential patterns of striatal activation in young children

with and without ADHD. *Biological Psychiatry, 53(10)*, 871–878. https://doi.org/10.1016/s0006-3223(02)01904-2

Egeland, J., Nordby Johansen, S., & Ueland, T. (2010). Do low-effort learning strategies mediate impaired memory in ADHD? *Journal of Learning Disabilities*, *43*(5), 430–440. https://doi.org/10.1177/0022219409355473

Emond, V., Joyal, C., & Poissant, H. (2009). Neuroanatomie structurelle et fonctionnelle du trouble déficitaire d'attention avec ou sans hyperactivité (TDAH) [Structural and functional neuroanatomy of attention-deficit hyperactivity disorder (ADHD)]. *L'Encephale, 35*(2), 107–114. https://doi.org/10.1016/j.encep.2008.01.00

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & Psychophysics, 16*(1), 143–149. https://doi.org/10.3758/BF03203267

Fox, E. (1995). Negative priming from ignored distractors in visual selection: A review. *Psychonomic Bulletin & Review, 2*(2), 145–173. https://doi.org/10.3758/BF03210958

Fox, E., & de Fockert, J.W. (1998). Negative priming depends on prime-probe similarity: Evidence for episodic retrieval. *Psychonomic Bulletin and Review, 5*, 107–113.

Friedman, M. J., & Reynolds, J. H. (1967). Retroactive inhibition as a function of response-class similarity. *Journal of Experimental Psychology, 74*(3), 351–355. https://doi.org/10.1037/h0024714

Frings, C., Hommel, B., Koch, I., Rothermund, K., Dignath, D., Giesen, C., Kiesel, A., Kunde, W., Mayr, S., Moeller, B., Möller, M., Pfister, R., & Philipp, A. (2020). Binding and retrieval in action control (BRAC). *Trends in Cognitive Sciences, 24*(5), 375–387. https://doi.org/10.1016/j.tics.2020.02.004

Frings, C., Rothermund, K., & Wentura, D. (2007). Distractor repetitions retrieve previous responses to targets. *The Quarterly Journal of Experimental Psychology, 60*, 1367–1377.

Frings, C., Schneider, K.K. & Fox, E. (2015). The negative priming paradigm: An update and implications for selective attention. *Psychonomic Bulletin and Review, 22*, 1577–1597 . https://doi.org/10.3758/s13423-015-0841-4

Gardiner, J. M., Craik, F. I., & Birtwistle, J. (1972). Retrieval cues and release from proactive inhibition. *Journal of Verbal Learning & Verbal Behavior, 11*(6), 778–783. https://doi.org/10.1016/S0022-5371(72)80012-4

Gaultney, J. F., Kipp, K., Weinstein, J. A., & McNeill, J. (1999). Inhibition and mental effort in attention deficit hyperactive disorder. *Journal of Developmental and Physical Disabilities*, *11*(2), 105–114. https://doi.org/10.1023/a:1021890919601

Gaynes, B. N., Magruder, K. M., Burns, B. J., Wagner, H. R., Yarnall, K. S., & Broadhead, W. E. (1999). Does a coexisting anxiety disorder predict persistence of depressive illness in primary care patients with major depression?. *General Hospital Psychiatry, 21*(3), 158–167. https://doi.org/10.1016/s0163-8343(99)00005-5

Giedd, J. N., Blumenthal, J., Molloy, E., & Castellanos, F. X. (2001). Brain imaging of attention deficit/hyperactivity disorder. *Annals of the New York Academy of Sciences*, *931*, 33–49. https://doi.org/10.1111/j.1749-6632.2001.tb05772.x

Georgiopoulos, A. M., Friedman, D., Porter, E. A., Krasner, A., Kakarala, S. P., Glaeser, B. K., Napoleon, S. C., & Wozniak, J. (2018). Screening for ADHD in adults with cystic fibrosis: Prevalence, health-related quality of life, and adherence. *Journal of Cystic Fibrosis*, *17*(2), 276–280. https://doi.org/10.1016/j.jcf.2017.08.011

Gibson, B. S., Gondoli, D. M., Flies, A. C., Dobrzenski, B. A., & Unsworth, N. (2009). Application of the dual-component model of working memory to ADHD. *Child Neuropsychology*, *16*(1), 60–79. https://doi.org/10.1080/09297040903146958

Gilden, D. (2002) Cognitive emissions of 1/f noise. *Psychological Review, 108*, 33–56.

Gray, S., Woltering, S., Mawjee, K., & Tannock, R. (2014). The Adult ADHD Self-Report Scale (ASRS): Utility in college students with attention-deficit/hyperactivity disorder. *PeerJ, 2, e324*. https://doi.org/10.7717/peerj.324

Hallowell, E. (2021, January 12). *The downside of undiagnosed adult ADHD*. ADDitude. Retrieved February 27, 2022, from https://www.additudemag.com/undiagnosed-adult-adhd-diagnosis-symptoms/

Hasher, L., Stoltzfus, E. R., Zacks, R. T., & Rypma, B. (1991). Age and inhibition. *Journal of Experimental Psychology Learning, Memory, and Cognition*, *17*(1), 163–169. https://doi.org/10.1037//0278-7393.17.1.163

Hensley, L., & Varela, R. E. (2008). PTSD symptoms and somatic complaints following Hurricane Katrina: the roles of trait anxiety and anxiety sensitivity. *Journal of Clinical Child and Adolescent Psychology : The Official Journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53*, *37*(3), 542–552. https://doi.org/10.1080/15374410802148186

Higginbotham, P., & Bartling, C. (1993). The effects of sensory distractions on short-term recall of children with attention deficit-hyperactivity disorder versus normally achieving children. *Bulletin of the Psychonomic Society*, *31*(6), 507–510. https://doi.org/10.3758/bf03337337

Hommel, B. (1998). Event files: Evidence for automatic integration of stimulus–response episodes. *Visual Cognition, 5*(1-2), 183–216. https://doi.org/10.1080/713756773

Hommel B. (2004). Event files: feature binding in and across perception and action. *Trends in Cognitive sciences*, *8*(11), 494–500. https://doi.org/10.1016/j.tics.2004.08.007

Hsu, J. Y., Tzeng, O. J. L., & Hung, D. L. (1997). Repetition facilitation, but not suppression of ignored stimulus during selective attention: Evidence from negative priming task in old adults. *Chinese Journal of Psychology, 39*(2), 139–156.

Jarosz, A. F., Wiley, J. (2014). What are the odds? A practical guide to computing and reporting Bayes factors. *Journal of Problem Solving, 71*(1), 2–9. https://doi.org/10.7771/1932-6246.1167

JASP Team (2022). JASP (Version 0.16.1)[Computer software].

Johnson, K.A., Wiersema, J.R. & Kuntsi, J. (2009). What would Karl Popper say? Are current psychological theories of ADHD falsifiable? *Behavior and Brain Function, 5, 15* https://doi.org/10.1186/1744-9081-5-15

Kahan, T. A., Oldak, V. A., & Lichtman, A. S. (2013). Working memory loads affect location-based negative priming differently than inhibition of return. *Journal of Cognitive Psychology*, *25*(4), 473–492. https://doi.org/10.1080/20445911.2013.789855

Kane, M. J., & Engle, R. W. (2000). Working-memory capacity, proactive interference, and divided attention: limits on long-term memory retrieval. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *26*(2), 336.

Kasper, L. J., Alderson, R. M., & Hudec, K. L. (2012). Moderators of working memory deficits in children with attention-deficit/hyperactivity disorder (ADHD): A meta-analytic

review. *Clinical Psychology Review*, *32*(7), 605–617. https://doi.org/10.1016/j.cpr.2012.07.001

Keppel, G., & Underwood, B. (1962). Proactive inhibition in short-term retention of single items. *Journal of Verbal Learning and Verbal Behavior*, *1*(3), 153-161. https://doi.org/10.1016/S0022-5371(62)80023-1

Kessler, R. C., Adler, L., Ames, M., Demler, O., Faraone, S., Hiripi, E., Howes, M. J., Jin, R., Secnik, K., Spencer, T., Ustun, T. B., & Walters, E. E. (2005). The World Health Organization ADULT ADHD self-report scale (ASRS): A short screening scale for use in the general population. *Psychological Medicine*, *35*(2), 245–256. https://doi.org/10.1017/s0033291704002892

Kofler, M. J., Singh, L. J., Soto, E. F., Chan, E., Miller, C. E., Harmon, S. L., & Spiegel, J. A. (2020). Working memory and short-term memory deficits in ADHD: A bifactor modeling approach. *Neuropsychology*, *34*(6), 686–698. https://doi.org/10.1037/neu0000641

Kroenke, K., Outcalt, S., Krebs, E., Bair, M. J., Wu, J., Chumbler, N., & Yu, Z. (2013). Association between anxiety, health-related quality of life and functional impairment in primary care patients with chronic pain. *General Hospital Psychiatry*, *35*(4), 359–365. https://doi.org/10.1016/j.genhosppsych.2013.03.020

Lange, K., Kühn, S., & Filevich, E. (2015). "Just Another Tool for Online Studies" (JATOS): An easy solution for setup and management of web servers supporting online studies. *PLoS ONE 10*(6), e0130834. https://doi.org/10.1371/journal.pone.0130834

Lou, H. C., Henriksen, L., Bruhn, P., Børner, H., & Nielsen, J. B. (1989). Striatal dysfunction in attention deficit and hyperkinetic disorder. *Archives of Neurology, 46*(1), 48–52. https://doi.org/10.1001/archneur.1989.00520370050018

Low, K. (2021, May 30). *Why ADHD awareness is important*. Verywell Mind. Retrieved May 3, 2022, from https://www.verywellmind.com/the-importance-of-adhd-awareness-20474

Lowe, D. G. (1979). Strategies, context, and the mechanism of response inhibition. *Memory & Cognition*, *7*(5), 382–389. https://doi.org/10.3758/bf03196943

MacDonald, P.A., Joordens, S., & Seergobin, K.N. (1999). Negative priming effects that are bigger than a breadbox: Attention to distractors does not eliminate negative priming, it enhances it. *Memory & Cognition, 27*, 197-207.

Makris, N., Buka, S. L., Biederman, J., Papadimitriou, G. M., Hodge, S. M., Valera, E. M., Brown, A. B., Bush, G., Monuteaux, M. C., Caviness, V. S., Kennedy, D. N., & Seidman, L. J. (2008). Attention and executive systems abnormalities in adults with childhood ADHD: A DT-MRI study of connections. *Cerebral cortex (New York, N.Y. : 1991)*, *18*(5), 1210–1220. https://doi.org/10.1093/cercor/bhm156

Marriott, J. M. (1998). *Selective attention, negative priming, and hyperactivity: Investigating the 'AD' in ADHD* (Doctoral dissertation).

Martinussen, R., Hayden, J., Hogg-Johnson, S., & Tannock, R. (2005). A meta-analysis of working memory impairments in children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*(4), 377–384. https://doi.org/10.1097/01.chi.0000153228.72591.73

Masson, M. J. (2011). A tutorial on a practical Bayesian alternative to null-hypothesis significance testing. *Behavior Research Methods, 433*(3), 679–690. https://doi.org/10.3758/s13428-010-0049-5

Mathôt, S., Schreij, D., & Theeuwes, J. (2012). OpenSesame: An open-source, graphical experiment builder for the social sciences. *Behavior Research Methods, 44*(2), 314-324. doi:10.3758/s13428-011-0168-7

Mayr, S., & Buchner, A. (2007). Negative priming as a memory phenomenon: A review of 20 years of negative priming research. *Journal of Psychology, 215*(1), 35-51.

McGeoch, J. A., & McDonald, W. (1931). Meaningful relation and retroactive inhibition. *The American Journal of Psychology, 43*(4), 579–588. doi:10.2307/1415159

Mclaughlin, E. N. (2003). Cognitive inhibition in children with Attention Deficit Hyperactivity Disorder [ProQuest Information & Learning]. *In Dissertation Abstracts International: Section B: The Sciences and Engineering* (*Vol. 64*, Issue 1–B, p. 426).

Mertens, V., Gagnon, M., Coulombe, D., & Messier, C. (2006). Exploratory factor analysis of neuropsychological tests and their relationship to the brown–peterson task. *Archives of Clinical Neuropsychology*, *21*(7), 733–739. https://doi.org/10.1016/j.acn.2006.08.005

Meyer, H. R., & Lasky, S. K. (2021, February 27). *Harold Robert Meyer MBA SCAC and Susan Karyn Lasky Ma Scac*. ADD Resource Center. Retrieved February 26, 2022, from https://www.addrc.org/disorder-105-tips-for-teachers/

Milliken, B., & Joordens, S. (1996). Negative priming without overt prime selection. *Canadian Journal of Experimental Psychology, 50*, 333–346.

Milliken, B., Joordens, S., Merikle, P. M., & Seiffert, A. E. (1998). Selective attention: A reevaluation of the implications of negative priming. *Psychological Review, 105*, 203–229.

National Institute of Mental Health. (2019). *Attention-deficit/Hyperactivity disorder*. NIMH » Home. https://www.nimh.nih.gov/health/topics/attention-deficit-hyperactivity-disorder-adhd/index.shtml

Neill, W.T. (1997). Episodic retrieval in negative priming and repetition priming. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 23*, 1291–3105.

Neill, W. T., & Mathis, K. M. (1998). Transfer-inappropriate processing: Negative priming and related phenomena. In D. L. Medin (Ed.), *The psychology of learning and motivation: Advances in research and theory, Vol. 38*, pp. 1–44). Academic Press.

Neill, W. T., Valdes, L. A., Terry, K. M., & Gorfein, D. S. (1992). Persistence of negative priming: II Evidence for episodic trace retrieval. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 18*(5), 993–1000. https://doi.org/10.1037/0278-7393.18.5.993

Nima, A. A., Rosenberg, P., Archer, T., & Garcia, D. (2013). Anxiety, affect, self-esteem, and stress: mediation and moderation effects on depression. *PloS One*, *8*(9), e73265. https://doi.org/10.1371/journal.pone.0073265

Oh, Y., Yoon, H. J., Kim, J.-H., & Joung, Y. S. (2018). Trait anxiety as a mediator of the association between attention deficit hyperactivity disorder symptom severity and functional impairment. *Clinical Psychopharmacology and Neuroscience*, *16*(4), 407–414. https://doi.org/10.9758/cpn.2018.16.4.407

Omani-Samani, R., Maroufizadeh, S., Ghaheri, A., & Navid, B. (2018). Generalized anxiety disorder-7 (GAD-7) in people with infertility: A reliability and validity study. *Middle East Fertility Society Journal*, *23*(4), 446–449. https://doi.org/10.1016/j.mefs.2018.01.013

Ortells, J. J., Noguera, C., Álvarez, D., Carmona, E., & Houghton, G. (2016). Individual differences in working memory capacity modulates semantic negative priming from single prime words. *Frontiers in Psychology, 7*.

Ossmann, J. M., & Mulligan, N. W. (2003). Inhibition and attention deficit hyperactivity disorder in adults. *The American Journal of Psychology*, *116*(1), 35. https://doi.org/10.2307/1423334

Parekh, R. (2017, July). *What Is ADHD?* What is ADHD? Retrieved March 15, 2022, from https://www.psychiatry.org/patients-families/adhd/what-is-adhd

Park, S., Lenzenweger, M. F., Püschel, J., & Holzman, P. S. (1996). Attentional inhibition in schizophrenia and schizotypy: A spatial negative priming study. *Cognitive Neuropsychiatry, 1*(2), 125–149. https://doi-org.lprx.bates.edu/10.1080/135468096396596

Peirce, J. W. (2009). Generating stimuli for neuroscience using PsychoPy. *Frontiers in Neuroinformatics, 2* (10), 1-8. doi:10.3389/neuro.11.010.2008

Peterson, L., & Peterson, M. J. (1959). Short-term retention of individual verbal items. *Journal of experimental psychology*, *58*(3), 193.

Posner, M. I., Rafal, R. D., Choate, L. S., & Vaughan, J. (1985). Inhibition of return: Neural basis and function. *Cognitive Neuropsychology*, *2*(3), 211–228. https://doi.org/10.1080/02643298508252866

Postle, B. R., Brush, L. N., & Nick, A. M. (2004). Prefrontal cortex and the mediation of proactive interference in working memory. *Cognitive, Affective & Behavioral Neuroscience*, *4*(4), 600–608. https://doi.org/10.3758/cabn.4.4.600

Postman, L. (1965). Unlearning under conditions of successive interpolation. *Journal of Experimental Psychology*, *70*(3), 237.

Pritchard, V. E., Healey, D., & Neumann, E. (2006). Assessing selective attention in ADHD, highly creative, and normal young children via Stroop negative priming effects. *Cognition and language: Perspectives From New Zealand*, 207-22

Pritchard, V. E., Neumann, E., & Rucklidge, J. J. (2008). Selective attention and inhibitory deficits in ADHD: Does subtype or comorbidity modulate negative priming effects? *Brain and Cognition, 67*(3), 324–339. https://doi.org/10.1016/j.bandc.2008.02.002

Raiker, J. S., Rapport, M. D., Kofler, M. J., & Sarver, D. E. (2012). Objectively-measured impulsivity and attention-deficit/hyperactivity disorder (ADHD): Testing competing predictions from the working memory and behavioral inhibition models of ADHD. *Journal of Abnormal Child Psychology*, *40*(5), 699–713. https://doi.org/10.1007/s10802-011-9607-2

Richardson, M., Abraham, C., & Bond, R. (2012). Psychological correlates of university students' academic performance: A systematic review and meta-analysis. *Psychological Bulletin*, *138*(2), 353–387. https://doi.org/10.1037/a0026838

Rosen, V. M., & Engle, R. W. (1997). The role of working memory capacity in retrieval. *Journal of Experimental Psychology: General, 126*(3), 211–227. https://doi.org/10.1037/0096-3445.126.3.211

Sagvolden, T., Johansen, E. B., Aase, H., & Russell, V. A. (2005). A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *The Behavioral and Brain Sciences*, *28*(3), 397–468. https://doi.org/10.1017/S0140525X05000075

Schmidt, N. B., Buckner, J. D., & Keough, M. E. (2007). Anxiety sensitivity as a prospective predictor of alcohol use disorders. *Behavior modification*, *31*(2), 202–219. https://doi.org/10.1177/0145445506297019

Seidman, L. J., Valera, E. M., & Makris, N. (2005). Structural brain imaging of attention-deficit/hyperactivity disorder. *Biological psychiatry*, *57*(11), 1263–1272. https://doi.org/10.1016/j.biopsych.2004.11.019

Shahar, N., Teodorescu, A. R., Karmon-Presser, A., Anholt, G. E., & Meiran, N. (2016). Memory for action rules and reaction time variability in attention-deficit/hyperactivity disorder. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *1*(2), 132–140. https://doi.org/10.1016/j.bpsc.2016.01.003

Shiels, K., & Hawk Jr, L. W. (2010). Self-regulation in ADHD: The role of error processing. *Clinical psychology review*, *30*(8), 951-961.

Shin, M. (2005). Interference and cognitive inhibition in children with ADHD using Stroop task. *Psychologia: An International Journal of Psychology in the Orient, 48*(3), 171–181. https://doi.org/10.2117/psysoc.2005.171

Shin, M. (2006). Different time course of negative priming in the subtypes of ADHD [ProQuest Information & Learning]. In *Dissertation Abstracts International: Section B: The Sciences and Engineering* (Vol. 67, Issue 4–B, p. 2244).

Sobanski, E., Brüggemann, D., Alm, B., Kern, S., Deschner, M., Schubert, T., Philipsen, A., & Rietschel, M. (2007). Psychiatric comorbidity and functional impairment in a clinically referred sample of adults with attention-deficit/hyperactivity disorder (ADHD). *European archives of psychiatry and clinical neuroscience, 257*(7), 371–377. https://doi.org/10.1007/s00406-007-0712-8

Sonuga-Barke, E. J., Dalen, L., & Remington, B. (2003). Do executive deficits and delay aversion make independent contributions to preschool attention-deficit/hyperactivity disorder symptoms? *Journal of the American Academy of Child & Adolescent Psychiatry, 42*(11), 1335-1342.

Spitzer, R. L., Kroenke, K., Williams, J. B., & Löwe, B. (2006). A brief measure for assessing generalized anxiety disorder. *Archives of Internal Medicine*, *166*(10), 1092. https://doi.org/10.1001/archinte.166.10.1092

Stolz, J.A., & Neely, J.H. (2001). Taking a bright view of negative priming in the light of dim stimuli: Further evidence for memory confusion during episodic retrieval. *Canadian Journal of Experimental Psychology, 55*, 219–230.

Storm, B. C., & White, H. A. (2010). ADHD and retrieval-induced forgetting: Evidence for a deficit in the inhibitory control of memory. *Memory, 18*(3), 265–271. https://doi.org/10.1080/09658210903547884

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*(6), 643–662. https://doi.org/10.1037/h0054651

Stuss, D. T., Toth, J. P., Franchi, D., Alexander, M. P., Tipper, S., & Craik, F. I. M. (1999). Dissociation of attentional processes in patients with focal frontal and posterior lesions. *Neuropsychologia, 37*, 1005-1027.

Tipper, S. P. (1985). The negative priming effect: Inhibitory priming by ignored objects. *The Quarterly Journal of Experimental Psychology A: Human Experimental Psychology, 37A*(4), 571–590. https://doi.org/10.1080/14640748508400920

Tipper, S. P., & Baylis, G. C. (1987). Individual differences in selective attention: The relation of 18 priming and interference to cognitive failure. *Personality and Individual Differences, 8*(5), 667– 19 675.

Tipper, S. P., Weaver, B., & Houghton, G. (1994). Behavioural goals determine inhibitory mechanisms of selective attention. *The Quarterly Journal of Experimental Psychology A: Human Experimental Psychology, 47A*(4), 809–840. https://doi.org/10.1080/14640749408401098

Uchida, M., Spencer, T. J., Faraone, S. V., & Biederman, J. (2018). Adult Outcome of ADHD: An overview of results from the MGH longitudinal family studies of pediatrically and psychiatrically referred youth with and without ADHD of both sexes. *Journal of Attention Disorders*, *22*(6), 523–534. https://doi.org/10.1177/1087054715604360

van Hulst, B. M., de Zeeuw, P., Vlaskamp, C., Rijks, Y., Zandbelt, B. B., & Durston, S. (2018). Children with ADHD symptoms show deficits in reactive but not proactive inhibition, irrespective of their formal diagnosis. *Psychological Medicine*, *48*(15), 2515–2521. https://doi.org/10.1017/S0033291718000107

Viarouge, A., Houdé, O., & Borst, G. (2019). Evidence for the role of inhibition in numerical comparison: A negative priming study in 7- to 8-year-olds and adults. *Journal of Experimental Child Psychology*, *186*, 131–141. https://doi-org.lprx.bates.edu/10.1016/j.jecp.2019.05.011

Wagenmakers, E. (2007). A practical solution to the pervasive problems of *p* values. *Psychonomic Bulletin & Review, 145*(5), 779–804. https://doi.org/10.3758/BF03194105

White, H. A. (2007). Inhibitory control of proactive interference in adults with ADHD. *Journal of Attention Disorders, 11*(2), 141–149. https://doi.org/10.1177/1087054706295604

Wickens, D. D., Born, D. G., & Allen, C. K. (1963). Proactive inhibition and item similarity in short-term memory. *Journal of Verbal Learning and Verbal Behavior, 2*, 440 – 445. doi:10.1016/S0022-5371(63)80045-6 (1).

Wickens, D. D., & Clark, S. (1968). Osgood dimensions as an encoding class in short-term memory. *Journal of Experimental Psychology, 78*(4, Pt.1), 580–584. https://doi.org/10.1037/h0026643

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: A meta-analytic review. *Biological Psychiatry*, *57*(11), 1336–1346. https://doi.org/10.1016/j.biopsych.2005.02.006

Wong, K.F.E. (2000). Dissociative prime-probe contextual similarity effects on negative priming and repetition priming: A challenge to episodic retrieval as a unified account of negative priming. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 26*, 1411–1422.

Table 1

JASP Output for the Bayesian ANOVA for Proactive Interference Grouped by ASRS Scores for Experiment 1b.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.033	0.138	1.000	
Set Number + Grouping Based on ASRS	0.200	0.619	6.489	18.587	6.609
Set Number	0.200	0.188	0.927	5.654	0.755
Grouping Based on ASRS	0.200	0.115	0.520	3.455	6.833
Set Number + Grouping Based on ASRS + Set Number ✻ Grouping Based on ASRS	0.200	0.045	0.188	1.348	2.742

Note. All models include subject

Figure 1

Accuracy Rates Across the Four Sets within each Trial of Semantically Related Items for Individuals with ADHD and Neurotypical Individuals as Designated with the ASRS in Experiment 1a. Error Bars Represent 95% Confidence Intervals.

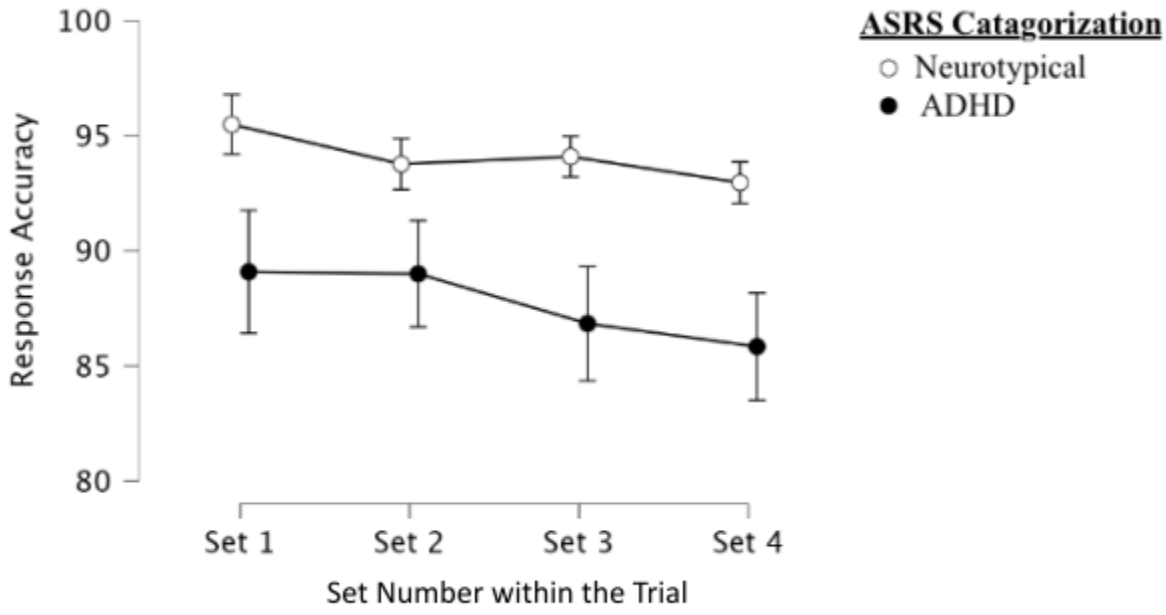


Figure 2

Accuracy Rates in the Control Condition and Ignored Repetition (IR) Condition for Individuals with ADHD and Neurotypical Individuals as Designated with the ASRS in Experiment 2. Error Bars Represent 95% Confidence Intervals.

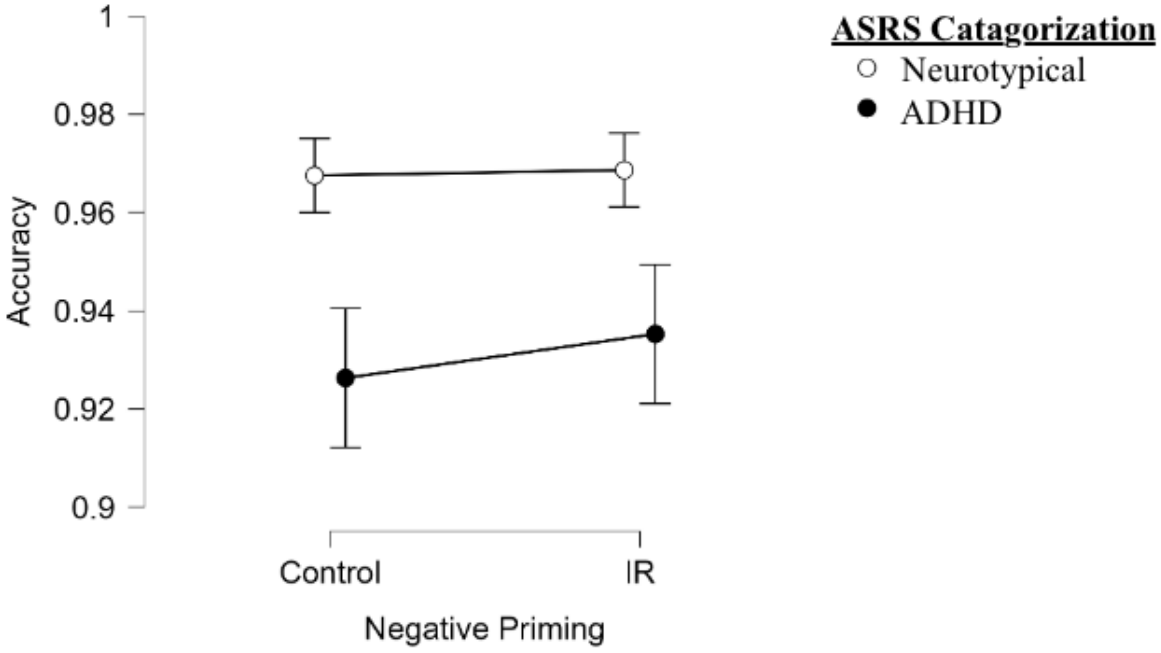
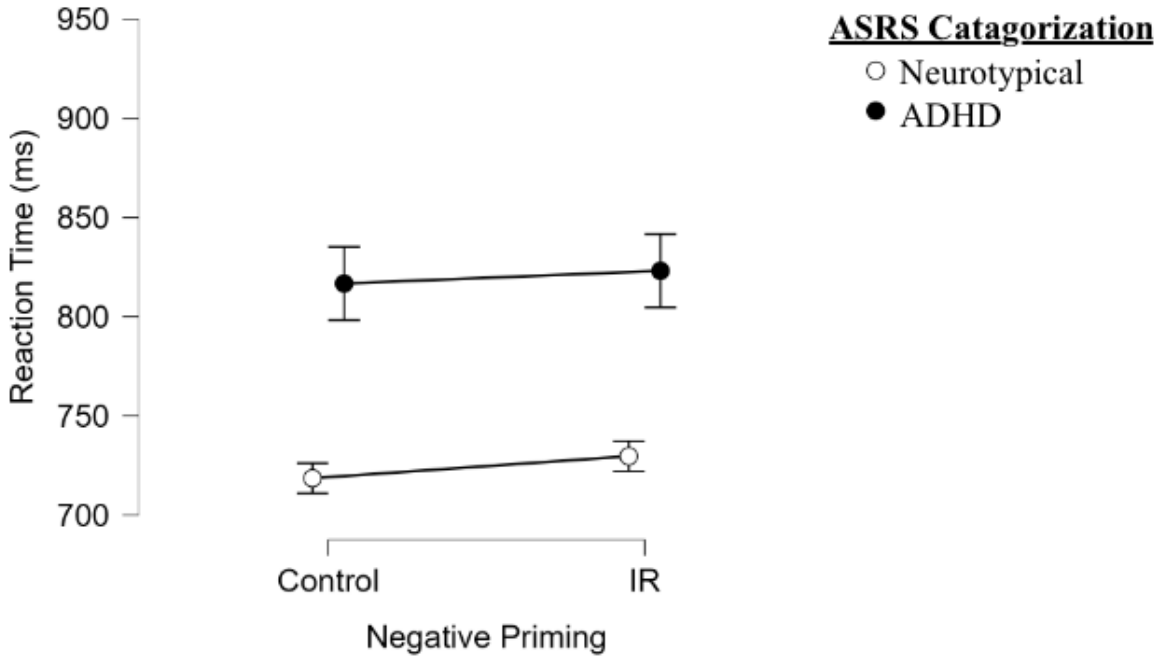


Figure 3

Response Times in the Control Condition and Ignored Repetition (IR) Condition for Individuals with ADHD and Neurotypical Individuals as Designated with the ASRS in Experiment 2. Error Bars Represent 95% Confidence Intervals.



Appendix A

Stimuli presented in Brown-Peterson Task in Experiment 1a

A precious stone: diamond, ruby, emerald, sapphire, pearl, opal, jade, topaz, amethyst, onyx, garnet, turquoise

A unit of time: hour, minute, second, year, day, century, month, decade, week, millisecond, eon, era

A relative: aunt, uncle, father, mother, brother, sister, cousin, grandmother, grandfather, nephew, niece, husband

A unit of distance: mile, foot, inch, yard, meter, centimeter, kilometer, millimeter, rod, light-year, decimeter, block

A metal: iron, copper, steel, gold, aluminum, silver, tin, zinc, brass, lead, bronze, magnesium

A type of reading material: magazine, book, newspaper, pamphlet, novel, textbook, paper, comic book, short story, journal, brochure, letter

A military title: lieutenant, general, sergeant, private, captain, colonel, major, corporal, admiral, commander, ensign, colonel

A four-footed animal: dog, cat, horse, cow, lion, tiger, elephant, pig, bear, mouse, rat, deer

A kind of cloth: cotton, wool, silk, rayon, nylon, dacron, linen, satin, orlon, madras, velvet, burlap

A color: blue, red, green, yellow, cyan, black, purple, white, pink, brown, violet, gray

A kitchen utensil: knife, spoon, fork, pan, pot, spatula, can-opener, stove, bowl, mixer, cup, dish

A building for religious services: church, synagogue, temple, chapel, cathedral, mosque, shrine, monastery, tabernacle, pagoda, sanctuary, basilica

A part of speech: noun, adjective, pronoun, verb, adverb, conjunction, preposition, interjection, participle, gerund, words, article

An article of furniture: chair, table, bed, sofa, desk, lamp, couch, dresser, television, stool, bureau, rug

A part of the human body: legs, arms, head, eye, chin, nose, finger, ear, hand, toe, mouth, stomach

A fruit: apple, orange, pear, banana, peach, grape, cherry, plum, grapefruit, lemon, tangerine, apricot

A weapon: bayonet, gun, rifle, bomb, club, sword, arrow, pistol, rope, cannon, spear, fists

An elective office: president, vice-president, senator, mayor, treasurer, secretary, governor, representative, congressman, councilman, chairman, sheriff

A type of human dwelling: house, apartment, tent, cave, hut, hotel, trailer, home, motel, shack, mansion, cottage

An alcoholic beverage: beer, whiskey, gin, wine, vodka, bourbon, scotch, rum, brandy, rye, champagne, vermouth

Appendix B

GAD-7 assessment items

Over the last two weeks, how often have you been bothered by the following problems? (0 - *not at all*, 1 - *several days*, 2 - *more than half the days*, 3 - *nearly every day*)

1. Feeling nervous, anxious, or on edge
2. Not being able to stop or control worrying
3. Worrying too much about different things
4. Trouble relaxing
5. Being so restless that it is hard to sit still
6. Becoming easily annoyed or irritable
7. Feeling afraid, as if something awful
8. might happen

If you checked any problems, how difficult have they made it for you to do your work, take care of things at home, or get along with other people?

- ☐ Not difficult at all
- ☐ Somewhat difficult
- ☐ Very difficult
- ☐ Extremely difficult

Appendix C

CAARS S:S assessment items

Listed below are items concerning behaviors or problems sometimes experienced by adults. Read each item carefully and decide how much or how frequently each item describes you recently. Indicate your response for each item by clicking the circle that corresponds to your choice. (0 - *not at all, never*; 1 - , 2 - *just a little, once in a while*; 3 - *pretty much, often*; 4 - *very much, very frequently*)

1. I interrupt others while talking.
2. I am always on the go as if driven by a motor.
3. I'm disorganized.
4. It's hard for me to stay in one place for very long.
5. It's hard for me to keep track of several things at once.
6. I'm bored easily.
7. I have a short fuse/ hot temper.
8. I still throw tantrums.
9. I avoid new challenges because I lack faith in my abilities.
10. I seek out fast paced, exciting activities.
11. I feel restless inside even if I am sitting still.
12. Things I see or hear distract me from what I am doing.
13. Many things set me off easily.
14. I am an underachiever.
15. I get down on myself.
16. I act okay on the outside, but inside I am unsure of myself.

17. I can't get things done unless there is an absolute deadline.
18. I have trouble getting started on a task.
19. I intrude on others' activities.
20. My moods are unpredictable.
21. I'm absent minded in daily activities.
22. Sometimes my attention narrows so much that I'm oblivious to everything else; other times it's so broad that everything distracts me.
23. I tend to squirm or fidget.
24. I can't keep my mind on something else unless it's really interesting.
25. I wish I had greater confidence in my abilities.
26. My past failures make it hard for me to believe in myself.

Appendix D

ASRS assessment items

Please answer the questions below. As you answer each question, click on the circle indicating the correct number that best describes how you have felt and conducted yourself over the past 6 months. (0 - *never*, 1 - *rarely*, 2 - *sometimes*, 3 - *often*, 4 - *always*)

1. How often do you have trouble wrapping up the final details of a project, once the challenging parts have been done?
2. How often do you have difficulty getting things in order when you have to do a task that requires organization?
3. How often do you have trouble wrapping up the final details of a project, once the challenging parts have been done?
4. When you have a task that requires a lot of thought, how often do you avoid or delay getting started?
5. How often do you fidget or squirm with your hands or feet when you have to sit down for a long time?
6. How often do you feel overly active and compelled to do things, like you were driven by a motor?
7. How often do you make careless mistakes when you have to work on a boring or difficult project?
8. How often do you have difficulty keeping your attention when you are doing boring or repetitive work?
9. How often do you have difficulty concentrating on what people say to you, even when they are speaking to you directly?

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

Appendix E

Stimuli presented in Brown-Peterson Task in Experiment 1b

A country: France, United States, Russia (USSR), England, Germany, Canada, Italy, Spain, Mexico, China, Japan, Sweden

A unit of time: hour, minute, second, year, day, century, month, decade, week, millisecond, eon, era

A relative: aunt, uncle, father, mother, brother, sister, cousin, grandmother, grandfather, nephew, niece, husband

A unit of distance: mile, foot, inch, yard, meter, centimeter, kilometer, millimeter, rod, light-year, decimeter, block

A metal: iron, copper, steel, gold, aluminum, silver, tin, zinc, brass, lead, bronze, magnesium

A type of reading material: magazine, book, newspaper, pamphlet, novel, text (book), paper, comic book, short story, journal, brochure, letter

A military title: lieutenant, general, sergeant, private, captain, colonel, major, corporal, admiral, commander, ensign, lieutenant colonel

A four-footed animal: dog, cat, horse, cow, lion, tiger, elephant, pig, bear, mouse, rat, deer

A kind of cloth: cotton, wool, silk, rayon, nylon, dacron, linen, satin, orlon, madras, velvet, burlap

A color: blue, red, green, yellow, cyan, black, purple, white, pink, brown, violet, gray

A kitchen utensil: knife, spoon, fork, pan, pot, spatula, can opener, stove, bowl, mixer, cup, dish

A building for religious service: church, synagogue, temple, chapel, cathedral, mosque, shrine, monastery, tabernacle, pagoda, sanctuary, basilica

A part of speech: noun, adjective, pronoun, verb, adverb, conjunction, preposition, interjection, participle, gerund, words, article

An article of furniture: chair, table, bed, sofa, desk, lamp, couch, dresser, television, stool, bureau, rug

A part of the human body: legs, arms, head, eye, chin, nose, finger, ear, hand, toe, mouth, stomach

A fruit: apple, orange, pear, banana, peach, grape, cherry, plum, grapefruit, lemon, tangerine, apricot

A weapon: bayonet, gun, rifle, bomb, club, sword, arrow, pistol, rope, cannon, spear, fists

An elective office: president, vice-president, senator, mayor, treasurer, secretary, governor, representative, congressman, councilman, chairman, sheriff

A type of human dwelling: house, apartment, tent, cave, hut, hotel, trailer, home, motel, shack, mansion, cottage

An alcoholic beverage: beer, whiskey, gin, wine, vodka, bourbon, scotch, rum, brandy, rye, champagne, vermouth