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ABSTRACT

Response Inhibition, Motivation, and Anxiety in Adult ADHD

By

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Attention deficit hyperactivity disorder (ADHD) is a behavioral syndrome characterized by impaired attention, hyperactivity, and impulsivity. A substantial subgroup of children with ADHD continue to have significant problems in adulthood. These adults continue to exhibit many symptoms of ADHD and are at risk for multiple comorbid disorders (such as anxiety disorders) and other negative outcomes. Although interest in adults with ADHD has recently increased, they remain less well studied than children with the disorder. A handful of studies based on Gray's (1987) theory of response regulation have examined whether a motivational inhibition deficit mediates childhood ADHD. The results are mixed. Although Gray's theory addresses anxiety in depth, few studies based on his theory have looked at the interplay of anxiety and an ADHD inhibitory deficit. The current study was designed to evaluate models of ADHD in adults based on Gray's model and to investigate possible moderator effects of motivation and anxiety, using Newman's modified go/no-go task. The results did not support motivational models of ADHD, or find an ADHD inhibitory deficit on the go/no-go task. The results did support Newman's theory of anxious impulsivity but, given null results for group differences, the interplay of anxiety and an ADHD inhibitory deficit was not clear.

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INTRODUCTION

The symptomatology of attention deficit hyperactivity disorder (ADHD) is complex, and children with the disorder are a heterogeneous group (Tannock, 1998). In particular, several dysfunctions are often "comorbid" with ADHD, such as conduct and anxiety disorders (Jensen, Martin, & Cantwell, 1997). It is now recognized that a substantial subgroup of children with ADHD continues to have significant problems in adulthood (Weiss & Hechtman, 1995). These adults continue to exhibit considerable symptoms of ADHD and are at risk for multiple comorbid disorders (Mannuzza, Klein, Besslar, Malloy, & La Padua, 1993) and other negative outcomes including accidents, employment problems and interpersonal problems (Weiss & Hechtman, 1995). Understanding why the disorder sometimes persists requires study of the underlying deficits in ADHD that may persist into adulthood in these cases. Unfortunately, although interest in adult ADHD has recently increased, it remains less well studied than the childhood form of the disorder.

Current neuropsychological theories of ADHD emphasize a deficit in behavioral inhibition (Barkley, 1997; Quay, 1997). Much research on these theories therefore has focused on executive control (Pliszka, Borcherding, Spratley, Leon, & Irick, 1997; Oosterlaan & Sergeant, 1996, 1998). Other research has drawn on Gray's (1987) theory of motivational cues and response inhibition (Iaboni, Douglas, & Baker, 1995; Milich, Hartung, Martin, & Haigler, 1994; Quay, 1997). Although Gray's theory addresses anxiety and although

Quay applied Gray's theory to ADHD, few ADHD studies of Quay's model have examined comorbid anxiety as well.

Attention-Deficit Hyperactivity Disorder in Children and Adults: An Overview

According to the Diagnostic and Statistical Manual of Mental Disorders (4th Ed.) (American Psychiatric Association, 1994), behavioral symptoms of ADHD in children are organized into two domains. The inattention-disorganization domain includes a lack of attention paid to detail in work or schoolwork, a failure to listen, a failure to follow through on instructions, and avoidance of tasks that require sustained mental effort. The hyperactivity-impulsivity domain includes fidgeting, behaving as though one is "driven by a motor," running around excessively, and interruption of others. Research on the 2 dimensions has yielded 3 subtypes: predominantly inattentive type, predominantly hyperactive-impulsive type, or combined type.

As children with ADHD approach adolescence and adulthood, they are at risk for developing conduct disorder and oppositional defiant disorder, alcoholism and drug abuse, antisocial behavior, depression, and anxiety (Spencer, Biederman, Willens, et al., 1996; Wender, 1995). Recent prevalence data indicate that ADHD occurs in 3% to 6% of the child population with boys outnumbering girls 3:1 (Anderson, Williams, Mc Gree, & Silva, 1987; Szatmari, Offord, & Boyle, 1989; Baumgartel, Wolraich & Dietrich, 1995).

ADHD in Adulthood

In the past, it was thought that ADHD symptoms remit as children approach adolescence and adulthood. However, recent studies suggest that

symptoms often continue into adulthood (Biederman, Faraone, et al., 1993; Mannuzza, Gittelman-Klein, et al., 1991; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993). Cognitive and behavioral correlates of ADHD are emphasized in many models of the adult syndrome. Such correlates include affective lability and moodiness, temper outbursts, immaturity and poor peer relations, hostility, acting out and other delinquent behaviors, and dyslexia (Wender, 1995). Estimates of the proportion of ADHD children who continue to have the disorder in adulthood range from 10% to 60%. One reason for the wide range of estimates is that diagnosis of ADHD in adulthood is complicated. Researchers disagree on the symptoms that comprise the adult syndrome. Wender (1995) criticizes an aspect of diagnostic criteria in the Diagnostic and Statistical Manual of Mental Disorders (3rd Ed., 1980), which also applies to DSM-IV (1994). Because criteria are based on symptoms of children between the ages of 6 and 10, the ages most often referred for ADHD, many criteria for diagnosis are appropriate for children but not for adolescents or adults. Such criteria included "difficulty sticking to play activities," "difficulty awaiting one's turn in games or in group situations," and "runs about and climbs on things excessively." DSM-IV, 1994) criteria also require characterization of adulthood ADHD as "In Partial Remission" if adults do not meet the same number of criteria that children are required to meet. This is problematic because even if an adult meets fewer criteria than a child, the adult's behavior may be just as age-inappropriate and impairing as the child's behavior. Thus, fewer symptoms could lead to the same relative degree of difficulty in an adult. Although DSM-IV (1994) suggests

applying age-appropriate examples of inattention, impulsivity, and hyperactivity to adults, it does not give adult-specific examples. Thus, Wender (1995) contends that clinicians faced with making a diagnosis of adult ADHD need adult-specific criteria, which are still lacking in the field.

Wender and colleagues proposed adult criteria but the field has not agreed on these either (see Wender, 1995, for a review). The Wender Utah Rating Scale (WURS) assesses a range of associated problems (i.e. emotional outbursts, general mood instability, disorganization, etc.) thought by Wender to comprise the adult syndrome. More recently, others have also developed adult scales. While these often include criteria very similar to the DSM-IV (1994) criteria, many have added additional criteria and/or adult-relevant examples (Brown, 1996; Achenbach, 1997; Conners, 1999). These vary in psychometric strength and in factorial dimensions. As a result, there is no single agreed upon means of assessing ADHD in adulthood. The DSM-IV (1994) remains the most conservative approach and was relied on primarily in the current study. However, it was also thought to be important to supplement DSM-IV criteria with these related measures and/or models of ADHD in adults.

Anxiety and ADHD

The tendency for ADHD to co-occur with anxiety or anxiety disorders is of particular interest in the current research. Studies indicate that significant anxiety occurs in about 25% of children with ADHD (Biederman, Newcorn, & Sprich, 1991). Lifetime diagnosis of anxiety disorders in adults with ADHD may be even higher, ranging from 43% to 52% (Biederman, Newcorn, et al., 1993). The co-

occurrence of anxiety may be important because it could indicate different cognitive deficits (Jensen et al., 1997) and may predict poorer response to pharmacological treatment (Pliszka, 1989). This problem of "comorbid" anxiety with ADHD creates an opportunity in the present study because, as already noted, Gray's (1987) theory addresses anxiety. Surprisingly, studies using Gray's model have not explicitly targeted anxiety in ADHD samples, a point discussed later.

Behavioral inhibition and motivation in ADHD

Several current theories of the causal mechanisms in ADHD highlight a deficit in behavioral inhibition (Barkley, 1990, 1997; Quay, 1997; Schachar, Tannock, & Logan, 1993; Sonuga-Barke, Holberg, & Hall, 1994). There are at least two schools of thought about the nature of the inhibitory deficit. Some have theorized on what will be termed "*executive*" inhibition problems -- that a primary deficit in behavioral inhibition affects secondary functions such as working memory, self-regulation, and affect/motivation (Barkley, 1997; Douglas, 1988 cited in Barkley, 1997). In this case, people with ADHD are expected to display an inhibitory deficit under a number of conditions requiring such inhibition. Others have focused on a primary *motivational* deficit, which causes behavioral disinhibition (Milich et al., 1994; Newman & Wallace, 1993; Quay, 1998; Sonuga-Barke, 1994). In this case, people with ADHD are expected to show an inhibitory deficit *only in specific motivational contexts*, such as in the presence of reward or punishment cues.

Executive behavioral inhibition

Barkley (1997) defines behavioral inhibition as involving 3 related processes: (1) stopping an initial prepotent response to a stimulus (i.e., a response associated with immediate reinforcement); (2) ceasing an ongoing response; and (3) protecting the delay this cessation creates, which allows self-directed actions to take place in the interim. Much ADHD research has focused on a deficit in behavioral inhibition similar to Barkley's definition. Although these models all refer to difficulty inhibiting a prepotent response, they define "prepotent response" very differently.

In much cognitive research, and in Barkley's conceptualization, a response is called prepotent when it has been repeated so many times previously that it has become a rote response to a given cue. In this type of research, experimenters first present a cue for the motor response and then, after several "go" trials, introduce a cue for inhibiting this response – a stopping cue (Logan & Cowan, 1984; Schachar & Logan, 1990). The repeated nature of this first "go" response creates a prepotency to that response. Many studies using one such paradigm, the stop-signal paradigm, have found that children with ADHD have a longer stop-signal reaction time (SSRT) than control children (Oosterlaan & Sergeant, 1996; Pliszka et al., 1997; Schachar & Logan, 1990). Many of the same studies, however, also find that the SSRT *standard deviation* is greater in the ADHD groups than in the control groups (see Oosterlaan & Sergeant, 1996; Pliszka et al., 1997; Schachar & Logan, 1990). High variability in response time appears common among children with ADHD (Chee, Logan,

Schachar, Lindsay, & Wachsmuth, 1989; Zahn, Krusei, & Rapoport, 1991). Although these studies have found that children with ADHD often cannot inhibit an ongoing motor response as well as controls (a deficit in behavioral inhibition), the high response time variability could also mean that ADHD children are not focusing on the task as well as control children (a deficit in vigilance). It is possible that this inability to focus on the task at hand, result from a lack of motivation.

Motivational Inhibition

In studies of the influence of motivation on ADHD symptoms (Newman & Wallace, 1993; Quay, 1997), prepotency is created by a participant's tendency to respond to reinforcement cues (i.e., rewards and response costs) prior to experimental manipulation. For instance, Newman and Wallace (1993) propose that children with ADHD display approach behavior that is more resistant to being stopped by punishment cues when reward cues are also present. Further understanding of these motivational inhibition models of ADHD requires an overview of Gray's model, on which they were based.

Gray's Theory of Response Regulation

Gray's model (1987, 1991,1994) of response regulation is based on an amalgamation of neural research and instrumental learning paradigms in animals (Gray, 1987) as well as on research on emotion and personality in humans (Eysenck, 1981). The model is speculative and has been the subject of several volume-length works. Briefly, it model involves three subsystems: the behavioral activation system (BAS), the behavioral inhibitory system (BIS), and the

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fight/flight system (FFS). Each system contributes to behavioral regulation and together, function to modulate response sets (Newman & Wallace, 1993). The BAS responds to conditioned stimuli of reward and non-punishment. In response to these stimuli, one feels a hopeful or relieved if he or she detects a signal of non-punishment in a noxious field. Activation of the BAS causes approach behavior or active avoidance of stimuli. The BAS also serves to inhibit the BIS. The BIS, in turn, is thought to inhibit the BAS and to respond to conditioned signals that predict punishment and non-reward, novel stimuli, and stimuli that are innately fear-related. BIS activation causes immediate stopping of ongoing motor behavior, increased cortical arousal, and orientation of attention toward relevant signals that enhance the analysis of potential threat. The FFS is thought to respond to unconditioned aversive stimuli with escape or defensive aggression. Gray (1987) also posits that a fourth system, the nonspecific arousal system (NAS), which receives excitatory input from both the BIS and the BAS. Activation of the NAS affects responses quantitatively more than qualitatively, increasing the rapidity and forcefulness of one's dominant response set whether it is approach or withdrawal.

A Model and Implicated Structures of the BAS and BIS

Emotional states are thought to be closely tied to actions (Frijda, 1986). Gray (1994) posits that the way one detects or interprets stimuli (information processing) is also influenced by emotional state and that the three systems (BAS, BIS, FFS) respond to different reinforcement contexts by enhancing different types of behavior. Each system involves different but interrelated brain

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structures, which process discrete types of information. Gray describes these systems on three levels: (1) a behavioral level whereby inputs and outputs of the system are described, (2) a speculated neural level whereby the proposed substrates of the system are delineated, and (3) a cognitive level whereby the information processing functions of the systems are elaborated. Gray calls his model a " preliminary sketch map" (Gray, 1994, pg. 29) of how behavioral, neural, and cognitive levels might be integrated in a neuropsychological model, rather than a complete theory. Despite some of its more postulatory elements (especially some of its intricacies at the neural level), the model has stimulated much motivational inhibition research on ADHD. Therefore, some background on each level is described below. Also, although the three (four, including the NAS) systems are interdependent and share several additional subsidiary components, the BAS and BIS are most integral to theoretical constructs of ADHD.

The Behavioral Inhibition System: Anxiety

On the behavioral level, the BIS responds to signals for punishment, signals of non-reward, novel stimuli, and innate fear stimuli. The system's output includes behavioral inhibition, increased arousal, and increased attention to input-stimuli. Gray (1987) defends the existence of this system on the basis that multiple physiological interventions affect all of the system's outputs to any one of the inputs. For instance, specific lesions or drugs may impair one's ability to respond to threats of punishment with behavioral inhibition but may not affect one's ability to respond with behavioral inhibition in order to obtain a reward.

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Thus, such lesions do not produce a failure of inhibitory control generally, but only in response to threats of punishment. Drugs that reduce anxiety in humans (e.g., benzodiazepines, barbiturates, and alcohol) appear to cut off the input-output relationships of the BIS (Gray, 1987).

Gray (1987) argues that the BIS performs its inhibitory function by a series of steps. It combines information about the present state of the external environment, a person's (or animal's) current motor program, information about past (stored) regularities that relates stimulus events to each other, and responses to subsequent stimulus events, and uses this information to predict the next state of the external environment. Then it compares the actual state of the environment to its predicted state of the environment. If they are congruent, it runs through these steps again. If they are not congruent, it stops the current motor program and directs attention to taking in more information so as to resolve the discrepancy.

According to Gray (1994), It is the septohippocampal system that executes the functions of the BIS. The system includes the septal area, entorhinal cortex, dentate gyrus, hippocampus, and subicular area. Gray believes that the subicular area receives "descriptions" of the external world via efferents from the entorhinal cortex, which receives input from cortical association areas. The subicular area receives "predictions" from other structures of the Papez circuit: the mammilary bodies, anterioventral thalamus, and cingulate cortex. The subicular area also receives information about the current motor program from the prefrontal cortex. Finally, the septohippocampal

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system would be able to halt the current motor program through projections to motor programming systems. Neurotransmitter substrates of the BIS are noradrenergic pathways from the locus coeruleus and serotonergic pathways from the raphe nucleus. These pathways have a general alerting function in response to threats.

The Behavioral Activation System: Approach

The BAS subserves approach, bringing one closer to conditioned stimuli for reward and non-punishment. Gray (1994) depicts the system as a simple positive feedback loop. Input to the system includes signals for reward and non-punishment. The resulting output is approach behavior. This, in turn, should bring someone more cues (or a stronger cue) for reward, as it is postulated that an appetitive stimulus' cues for reward are stronger the closer the one is to their source.

At the neural level, two subsystems are thought to function together in the BAS. The caudate and putamen could bring together motor and sensory-motor cortices, dorsal striatum, globus pallidus, ventral anterior and ventral lateral nuclei of the thalamus, and the dopaminergic pathway from the substantia nigra to the caudate-putamen. This subsystem, according to Gray (1994), deals with detailed sensory-motor content of motor programs. Another subsystem involves prefrontal cortex and the cingulate gyrus, ventral striatum, ventral globus pallidus, dorsomedial nucleus of the thalamus, and dopaminergic projections to the nucleus accumbens from nucleus A 10. Gray postulates that this subsystem pertains to goal direction and incentive motivation (Gray, 1994).

Gray, Feldon, Rawlins, Hemsley, and Smith (1991) lay out a simplified outline by which the BAS might function. The specific steps of a motor program first are encoded by the caudate system via connections with sensory motor and motor cortices. The nucleus accumbens may work with the caudate system to allow switching between motor programs. Projections from the amygdala to the nucleus accumbens are thought to convey information about cue reinforcement contingencies and these may mediate sequencing of steps and orderly running of a motor program. Projections from the subiculum to the nucleus accumbens may be a means by which the septohippocampal system checks whether the actual outcome of a motor step in the program matches the expected outcome. The prefrontal cortex may coordinate the activity of the BAS and the BIS via connections with cortical components of the caudate system, the nucleus accumbens, dorsomedial thalamus, amygdala, entorhinal cortex and cingulate cortices.

Gray's Model Applied to ADHD

Early work by Newman on Psychopathy. Application of Gray's model to ADHD developed subsequent to Newman's application of Gray's model to adult psychopaths. According to Cleckley (1976), characteristics of a psychopath include untruthfulness, lack of remorse and shame, antisocial behavior, poverty of major affective reactions, poor judgement, inadequate motivation, and failure to learn from experience. It is well established in clinical research that psychopaths show less arousal (as measured by skin conductance) in response to anticipation of an electric shock than the normal population (Hare, 1965; Hare,

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1970). Hare (1965, 1970) interpreted these results to mean that psychopaths were relatively insensitive to punishment cues. Newman disagrees and suggests that psychopaths are insensitive to punishment cues only when reward cues are simultaneously presented (Newman 1987; Newman & Kosson, 1986; Newman, Widom, & Nathan, 1985). Newman (1987) found partial support for his view from studying adult psychopaths' from responses on a card-playing task. When reward and punishment cues were presented simultaneously, psychopaths showed greater response perseveration than controls. To separate the cue contingencies, Newman modified the go/no-go task (Newman, Widom, & Nathan 1985; Newman & Kosson, 1986) by attaching reward and response cost (punishment) contingencies, creating 3 conditions: a reward only condition, a response cost only condition, and a reward-response const condition (e.g. both rewards and response costs were presented in the same condition). The Go/no-go stimuli consisted of 8 sets of 8 index cards. On each card in a set, a unique, two-digit number was printed. Four numbers were arbitrarily designated as winning numbers, 4 as losing numbers. Participants were required to learn, by trial and error, to respond to positive stimuli and not to respond to negative stimuli. Responding to a negative stimulus was considered an error of commission; not responding to a positive stimulus was considered an error of omission. In a reward only condition, participants were given 25¢ for picking each winning card. In a reward-response cost condition, they were given 25¢ for each winning card and lost 25¢ for each losing card chosen.

In the reward- response cost condition, psychopaths made more commission errors than normals (Newman et al., 1985). However, normal and psychopathic groups did not differ in the reward-only or punishment-only conditions. This was interpreted as refuting the possibility that errors were due to overactive approach (BAS hyperactivation) or underactive passive avoidance (BIS hypoactivation) in isolation (Newman et al., 1985). Newman concluded that the evidence supported his theory whereby, in psychopathy, punishment cues are relatively ignored while approach responding is engaged.

Quay's application of Gray's model to ADHD. Like Hare's theory on psychopathy, Quay postulated that individuals with ADHD might be insensitive to cues for punishment (Quay, 1988) due to weak BIS functioning (Quay, 1997). He also extended Newman's theory of psychopathy to children by stating that undersocialized conduct disorder in childhood (a syndrome involving extreme antisocial behavior and a possible precursor to psychopathy) was caused by a BAS that dominated over the BIS, whereby children focus on rewards at the expense of punishments (Daugherty & Quay, 1991). To test his hypothesis that the BAS dominated over the BIS in conduct disorder, Quay postulated that children with ADHD "comorbid" with conduct disorder would have a higher degree of disinhibition than those with ADHD or conduct disorder. Thus, he assumed that the deficits involved in each might be "additive" in nature (Quay, 1988). The differences, although not significant, were in the predicted direction (Daugherty, Quay, & Ramos, 1993). A later study did find that children with ADHD + conduct disorder had higher commission error rates on a "door opening"

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task featuring a steadily increasing rate of punishment cues over reward cues (Matthys, van Goozen, de Vries, Cohen-Kettenis, & van Engeland, 1998). Also in support of Quay's theory, Abramowitz, O'Leary, and Rosen (1987) found that punishments effectively decreased off-task behavior in the classroom, whereas rewards did not. It could be that this punishment increases the activity of an otherwise hypoactive BIS.

Despite some initial support for Quay's ideas, closer inspection of experimental methods reveals some problems in interpretation of results and some questions that are left unanswered. Specifically, Quay cites a number of studies that used the stopping-task to support his theory of hypoactivation of the BIS in ADHD (Quay, 1997). However, the stopping-task (Logan & Cowan, 1984) contains no explicit cues for reward or punishment. The door-opening task used by Matthys et al. (1998) did include explicit cues for reward and punishment, but cues were not controlled such that one could tell which specific cues affected participants with ADHD, conduct disorder, and ADHD+conduct disorder. Presumably, one would have to present punishments and rewards alone in order to tell whether ADHD children were responding with increased disinhibition to one reinforcement cue but not the other. Furthermore, a study by Abramowitz et al. (1987) has been cited as evidence for punishment reducing off-task behavior in ADHD children specifically (Milich et al., 1994), even though the diagnostic status of participants in the study is not clear. Participants in the Abramowitz et al. (1987) study were included if they scored 2 standard deviations above the mean on the Hyperactivity *or* Daydreaming/Inattention subscales of the Conners

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Teachers Rating Scale (Conners, 1969, as cited in Abramowitz et al., 1987) *or* displayed significant reading or mathematics deficits. In fact, out of sixteen participants in the experimental group, four scored two standard deviations above the mean on the Hyperactivity subscale; five participants, two of whom were also above Hyperactivity thresholds, scored two standard deviations above the mean of the Daydreaming/ Inattentive subscale.

Another problem with Quay's theory concerns comorbid anxiety. If as Gray (1987) contends, BIS activity is synonymous with anxiety and if, as Quay (1988) argues, ADHD is caused by an inactive BIS, then ADHD and anxiety, theoretically, could not coexist. Thus, according to Quay, those with ADHD "comorbid" with anxiety could not exhibit the same BIS deficit as those with ADHD alone or ADHD with low levels of anxiety.

Newman's alternative model of ADHD. Contrary to Quay, but also following Gray's theory, Newman and Wallace (1993) argue that problems of behavioral disinhibition in ADHD, rather than being caused by "too much" approach (BAS hyperactivity) or "too little" passive avoidance (BIS hyperactivity) alone, but instead may be caused by their deficient interaction when reward and punishment cues are presented simultaneously (Newman & Wallace, 1993). This is reminiscent of the theory Newman applied to psychopathy (Newman 1987; Newman & Kosson, 1986; Newman et al., 1985) and that Quay applied to conduct disorder (Daugherty, Quay, and Ramos, 1993). Newman points out that psychopathy and ADHD may be similar, not in the types of behavior exhibited but

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Newman and Wallace (1993) proposed that their theory might apply to ADHD because a number of researchers have linked situational factors to deficits in ADHD, including rewards and punishments (see Barkley, 1990; Schachar, Rutter, & Smith, 1981). Furthermore, cognitive and behavioral treatment improves behavior of impulsive children (Baer & Nietzel, 1991). and ADHD children (Pelham, Carlson, et al., 1993).

Milich et al. (1994) used Newman's go/no-go task (Newman & Wallace, 1993) with reward-response cost and reward only conditions to test Quay's dysfunctional BIS theory and Newman's disregulation theory. Milich et al. (1994) studied adolescent participants with a history of psychiatric or behavioral disorders. They measured ADHD symptoms as a continuous variable, rather than selecting participants with and without an ADHD diagnosis. ADHD symptoms were significantly correlated with commission errors when both rewards and response costs were present but not when response costs were presented alone, similar to Newman's psychopathic population. The results supported Newman's disregulation theory of ADHD.

Iaboni et al. (1995) used a task similar to Milich et al. (1994) but added a response-cost condition to the go/no-go task. The study also differed from Milich et al., (1994) in that it included group of children diagnosed with ADHD. ADHD participants made more commission errors than controls across *all* conditions, whereas differences *between* conditions and group-by-condition interactions

were non-significant. Thus, neither Quay's (1988) nor Newman's (Newman & Wallace, 1993) motivational models were supported. Instead, it appeared that the ADHD children had a generalized inhibitory deficit regardless of motivational cues presented. This supported Douglas' (1988) own theory as well as Barkley's (1997) theory of a non-motivational, generalized inhibitory deficit in ADHD. The participants in Iaboni et al.'s (1995) study, however, were young children (ages 8-13) who may have found the go/no-go task more frustrating than Milich et al.'s (1994) adolescents. Although the task was modified to accommodate the young age of the participants (only 3 go and 3 no-go cards per trial versus 4 go and 4 no-go in the original task), the ADHD children, perhaps more susceptible to frustration on the task, may have given up more readily than controls. Furthermore, in the repeated measures design, children underwent all 4 reward-response cost contingencies. Children could have been confused about what responses lead to rewards or punishments becasue these rules changed between the conditions.

The latter point brings up a noteworthy problem with the go/no-go task, given the task's centrality in evaluating Gray's theory in humans. Although theories generated by these studies are applied to inhibitory functioning, success on the go/no-go task requires learning and working memory as well. In each condition, participants must recall which of 6-8 numbers were "winners" and which numbers were "losers" (i.e., which numbers they would be rewarded for responding to and which numbers they would be punished for responding to). Furthermore, participants are not told to remember specific numbers (vs.

searching for a pattern) in the task instructions. Prior studies did not record whether participants ever understood this. Thus, some participants may become frustrated early, thinking that rewards and punishments are doled out more or less randomly.

One solution might be to modify the stop-signal task by adding motivational cues. Oosterlaan and Sergeant (1998) worked rewards and punishments into the stop-signal task, which minimized the involvement of working memory. Like Iaboni et al. (1995), they found that ADHD children committed more commission errors than controls across both reward and punishment conditions. However, Oosterlaan and Sergeant (1998) tested the effects of rewards and response costs only in isolation. This did not adequately test Newman's (Newman &Wallace, 1993) theory of their deficient interaction in ADHD. Another potential pitfall of that study was the use of the stop- signal task, despite its advantages in relation to low working memory load. In the stop-signal paradigm, participants are instructed not to wait for the inhibition cue. However, if punishment is given when participants do not withhold action in response to the inhibition cue, it may be difficult to keep participants from waiting for this cue to occur, which, in turn, may confound results.

Thus, a handful of studies examined motivational inhibition in ADHD, but results have not been consistent, and there are no studies of motivational inhibition on adults with the disorder. One reason for these inconsistent results in studies of children may have been a failure to assess anxiety levels

Anxiety, Motivational Contingencies, and ADHD Comorbid with Anxiety

Gray (1987) contends that high trait anxiety predicts improved learning when participants are presented with conditioned punishment stimuli (Corr, Pickering, & Gray, 1997). However, Newman's group has found that when anxious individuals are required to respond to signals for punishment, they tend to respond impulsively (Wallace, Newman, & Bochorowski, 1991). Newman theorizes that when anxious individuals are presented with cues for punishment but are not allowed to make their preferred passive avoidance response, their approach responses will be more disorganized and vigorous (Newman & Wallace, 1993). A test that exemplifies this "anxious impulsivity" is the circle drawing task (Bochorowski & Newman, 1990; Wallace & Newman, 1990). In this task, participants are required to trace a circle as slowly as possible while they are simultaneously presented with a stimulus designed to activate the BAS (such as having a chance to win $3.00) or a stimulus designed to activate the BIS (such as having a chance of losing $3.00). Neurotic introverts generally displayed the fastest tracings under conditions designed to activate the BIS, while neurotic extroverts displayed the fastest tracings under conditions designed to activate the BAS. Thus, anxious individuals could look behaviorally as though they have ADHD under conditions in which the BIS is activated and passive avoidance is not an option. An example might be when an anxious individual is forced to speak in front of a large group of people. The individual may prefer not to speak at all, but when forced to speak, may do so with poorly regulated speed and volume. However, following Gray's theory, ADHD individuals who are also

anxious should exhibit *less* impulsivity than those who are not anxious, when passive avoidance *is* allowed. In fact, children with ADHD and high anxiety do act less impulsively in general than those with ADHD alone (Pliszka, 1989).

Admittedly, caution is needed in extrapolating from findings with normal range neurotic introverts to individuals with clinical disorders. Although studies on ADHD and anxiety have investigated clinical levels of anxiety through diagnostic procedures (Biederman et al, 1991; Jensen et al., 1997), tests of Gray's theory of response modulation have investigated subclinical levels of anxiety (Corr et al., 1997; Wallace et al., 1991) implementing personality inventories and the State-Trait Anxiety Inventory (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). Nevertheless, if subclinical levels of anxiety could cause disinhibition in select situations, one might reason that clinical levels of anxiety should be at least as powerful in exerting the same effects.

Thus, high levels of anxiety are likely to affect ADHD performance on the go/no-go task in one of two ways that could obscure ADHD results on the task. Either high anxiety will make ADHD participants *less* impulsive when response-costs are presented, or it will make ADHD participants *more* impulsive when response-costs are presented and passive avoidance is blocked. Therefore, one goal of the present study was to test the effects of anxiety on go/no-go inhibition, and what role these effects may play in ADHD performance on the go/no-go task.

Summary. Adult ADHD is not well understood, but it has become clear that a large number of children with the disorder continue to exhibit symptoms

into adulthood (Beiderman, et al., 1993; Mannuzza et al., 1991; Mannuzza et al., 1993) as well as significant comorbid diagnoses (Spencer et al, 1996; Wender, 1995). While researchers have found that an inhibitory deficit may be a key element of the disorder in children (Barkley, 1990, 1997; Quay, 1997; Schachar et al., 1993; Sonuga-Barke, Holberg, & Hall, 1994 there is no agreement on the nature of this inhibitory deficit. Theories of motivational inhibition have been applied to childhood ADHD with mixed results, and it is unknown whether they may apply better or worse to the disorder in adulthood. Anxiety is an important confound in this research as (a) it is possible that higher levels of anxiety can be associated with more impulsivity or less impulsivity under certain conditions and (b) ADHD tends to be associated with high anxiety or with the presence of anxiety disorders.

General Predictions

I. On a go/no-go task, motivational cues will either:

(1) have no effect on the performance of ADHD individuals on a go/no-go task -- young adults with ADHD will show a general inhibitory deficit across conditions, regardless of what specific motivational cues are given within each condition (Iaboni et al.,1995), or

(2) motivational cues will affect ADHD individuals in one of three ways, based on different interpretations of the application of Gray's (1987) theory to ADHD. ADHD individuals will demonstrate a greater inhibitory deficit than controls in conditions designed to stimulate either (a) the BIS (Quay, 1997), (b) the BAS

(Gorenstein & Newman, 1980), or (c) the BAS and the BIS simultaneously (Newman & Wallace,1993).

II. ADHD inhibitory responding will be related to anxiety in one of three ways: either (1) an ADHD inhibition deficit could be hidden (fewer errors) by high anxiety (thus, the ADHD deficit would become larger once anxiety is covaried out), (2) ADHD inhibition problems could be made even worse by high anxiety (thus, the ADHD deficit would be reduced once anxiety is covaried) or, (3) the ADHD group's inhibition problems may not be affected by high levels of anxiety (the ADHD main effect would not change when anxiety is covaried).

The additional analysis that comprises Part II relies on the assumption that the STAI is measuring the same construct in the ADHD and control groups. One could imagine, for instance that a person with ADHD may respond to questions on the STAI such as "I feel calm" or "I feel jittery" in the same way as a person with anxiety, but for very different underlying reasons. A preliminary check was done to insure this cross-group validity assumption bears out before the Part II analysis was conducted.

METHOD

Participants

Participants were 23 adults with ADHD and 24 controls ages 18-39. They were recruited from Michigan State University and Lansing Community College. Participants were included in the ADHD group if they (1) had been diagnosed previously with ADHD, (2) displayed at least 6 inattention and 6 hyperactive-impulsive symptoms before age seven on the NIMH Diagnostic Interview Schedule for the DSM-IV (DSM-VI, 1994) and a self-report DSM-IV (1994) check list, the self SNAP, a modified version of the SNAP-IV parent report (Pelham, Gnagy, Greenslade, & Milich, 1992) and (3) symptoms persisted into adulthood as measured by a T-score greater than 65 on the Conners Adult ADHD Rating Scale (1999) or the Young Adult Self Report (Achenbach, 1991) (YASR). The Brown Attention-Deficit Disorder Scale for Adults (ADDSA) (Brown, 1996) and the Wender Utah Rating Scale (WURS) (Wender, 1995) were also given but were not required for diagnosis. Participants were included in the control group if they had no history of ADHD, if they had T-scores below 64 on the Conners and YASR, did not meet diagnostic thresholds on any ADHD instrument, and endorsed 4 or fewer child symptoms of either inattention or hyperactivity-impulsivity. Participants were also excluded if the DIS (1995) indicated current major depression, or mania, or if their Full Scale IQ was below 70 on the Wechsler Adult Intelligence Scale – Revised Edition (WAIS-R) (Wechsler, 1981). Participants with generalized anxiety disorder or other anxiety related conditions and learning disorders were retained in the study. Because of the small number

of ADHD participants with current or past generalized anxiety disorder (n=7), and to attain greater power, anxiety was measured as a continuous variable with the State Trait Anxiety Inventory (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). Details on all instruments are provided later.

Procedure

Participants completed the go/no-go task and other measures as part of a diagnostic interview and test battery given at Michigan State University. The entire battery was completed over the course of four visits lasting one to two hours each. The go/no-go task took approximately 15 minutes to complete while the WAIS-R, questionnaires, and diagnostic interview took approximately three hours to complete. All tests and measures were completed in a standard order. Those participants taking stimulant medication for ADHD symptoms went through a 24-hour washout period before each testing session. Tests were administered by trained graduate and undergraduate psychology students who were blind to participants' final diagnostic status. Participants were compensated for their time with course credit and/or payment, depending on the circumstances of their enrollment in the study.

Measures

The Go/no-go task (Newman, 1985): The go/no-go task was used for two reasons. First, rewards and response costs could be added to the task without changing its fundamental nature, unlike the stop-signal task. Second, neuroimaging data have indicated that a go/no-go task (without motivational cues) engaged areas of the brain, including the prefrontal cortex, globus pallidus,

and the caudate, that (a) are implicated in Gray's theoretical work on motivational contingencies in inhibitory functioning (Gray, 1987, 1991, 1994), (b) seem to be necessary for response inhibition (Casey, Orendi, et al., 1997), and (c) are thought to be abnormal in individuals with ADHD (Casey, Castellanos, et al., 1997).

The go/no-go task was the same task used by Iaboni et al.(1995). It was presented on a PC computer running in DOS. The program was written in Q-Basic. Each participant was tested under the 3 counterbalanced go/no-go task conditions described below. Stimuli consisted of 3 sets of 6 two-digit numbers presented on a computer screen. Each set was presented 12 times, for a total of 216 trials, each condition lasting approximately 5 minutes. Three of the numbers were arbitrarily designated as winning numbers (S+), and 3 as losing numbers (S-). Participants were required to learn by trial and error to respond to S+ numbers and not to respond to S- numbers. Responding to a S- number was considered an error of commission; not responding to an S+ number was considered an error of omission. Participants were given 2 seconds to respond (by pressing a key) or to withhold response. Rewards and/or punishments were presented during the trial. Reward was defined by giving the participant 10 cents and punishment was defined by taking away 10 cents. After each trial, a screen appeared, telling the participant (1) the amount of money won or lost in that trial, and (2) the total amount of money acquired so far. The 3 conditions are elaborated in Table 1.

Table 1

Go/no-go task design.

Condition	When winning stimulus appears	When losing stimulus appears
1) reward only (72 trials)	response = reward no response = no effect	response = no effect* no response = reward
2) response cost only (72 trials)	response = no effect no response = punishment	response = punishment* no response = no effect
3) reward-response cost (72 trials)	response = reward no response = no effect	response = punishment* no response = no effect

* = a commission error (error in passive avoidance)

Participants began with no money at the beginning of the reward only task, $1.00 at the beginning of the reward-response cost task, and $2.50 at the beginning of the response cost only task. Before starting, they were told that the amount they had won or lost would be presented to them on the computer screen between trials and that they would be given the equivalent total amount of money when the testing was complete. The rest of the instructions were as follows:

> Now we are going to play 3 card games – it will be a bit like gambling because sometimes you will win money and, at other times, you will lose money, depending on how you respond to each number presented on the screen, and depending on the rules of the game you are playing. Some of the numbers will be "winners" and some numbers will be "losers." You want to press the space bar for all the winning numbers but not for any losing numbers, but at the beginning of each game, you won't know which numbers are which. For each of the 3 games, we are going to go through the numbers 12 times, so your job is to remember which numbers are "winners" and which numbers are "losers." This way, you can try to earn as much money as possible and lose as little as possible.

There was a practice trial for each condition, during which participants found out what would happen after each response. Eight participants (5 ADHD and 3 controls) had a net loss of money in the response cost condition. When this happened, they were presented with a negative amount of money on the screen, but they did not "pay" this amount when their final earnings were totaled. Rather, they received no money for the response cost condition. Commission errors and cash won were recorded in all conditions. If participants responded correctly on all trials, they could have earned a maximum of $14.00.

Possible Constraints of Memorization. Full testing of the effects of learning and working memory on results would have required a duplication of all conditions without the memorization element (such as having only 1 S+ and 1 S- stimulus per condition), which would have doubled the number of conditions. It was decided that the increase in testing time did not justify the addition, given that this was not a central focus of the study. Instead, after all stimulus presentations in each condition, the examiner (1) asked participants to recall which numbers were "winners" and which numbers were "losers," and (2) asked participants to identify the S+ stimuli and S- stimuli on a sheet of paper. Participants were not rewarded for this part of the task. This tested their ability to understand the task and whether they had learned which numbers were S+ and which numbers were S-. The examiner recorded their "number of correct" responses to this quiz. The Digit Span subtest of the WAIS-R served as an additional check on working memory for numbers.

Diagnostic Measures

Diagnostic Interview schedule for the DSM-IV (DIS-IV, Robins, Cottler, Bucholz, & Compton, 1995). The DIS-IV is a structured diagnostic interview that assesses severity of problem behaviors, remission, and age of onset for psychological disorders in adults according to DSM-IV criteria. Diagnoses are based on the overall problem score, age of onset, duration, and level of impairment for each module. The Residual ADHD model was used in case selection.

DSM-IV Symptom Ratings in Childhood. To assess symptoms in childhood, participants completed a modified version of the Swanson, Nolan, and Pelham, DSM-IV (1994) checklist (SNAP-IV) (Pelham et al., 1992). This is a DSM-IV (1994) symptom checklist, which is designed to assess participants' ADHD symptoms in childhood. The revision includes a change in instructions (from the original scale, which used parent's ratings of their child, to the revision, which uses self-ratings), but individual items are otherwise the same as the original SNAP-IV. Each item was rated on a 4-point scale (0-not at all, 1-just a little, 2-quite a bit, 3-very much). Items were scored by summing endorsed symptoms. Participants who did not meet at least 6 symptoms for inattention and 6 symptoms for hyperactivity-impulsivity were excluded from the ADHD group.

Young Adult Self Report (YASR, Achenbach, 1997) is intended to assess a variety of behavior and emotional problems in young adults, ages 18-27. the questionnaire consists of 115 items. The scale is grouped into 8 syndrome constructs: Anxious/Depressed, Withdrawn, Somatic Complaints, Thought

Problems, Attention Problems, Intrusive, Aggressive Behavior, and Delinquent Behavior. The endorsement of items is made on a 3-point scale of how "true" an item is of a participant's feelings or behavior (0-not true, 1-somewhat or sometimes true, 3-very or often true). Scores are added together within syndrome constructs and raw score are converted to T-scores indicating where the participant stands in relation to a normative sample of participants of the same sex. The YASR is able to accurately predict referred from non-referred individuals 73% and 81% of the time for females and males, respectively, and has a one-week test-retest reliability of 0.84 for the Attention Problems subscale. (Achenbach, 1997).

Conners Adults ADHD Rating Scale (CAARS, Conners, Erhardt, & Sparrow, 1999) is a 66-item scale with 9 subscales. These assess inattention/memory problems, hyperactivity/ restlessness, and impulsivity/emotional lability. Items are answered on a 4-point scale of frequency of occurrence (0-never, 1-once in a while, 2-often, 3-very frequently). Scores are added together within the scales and raw score are converted to T-scores indicating where the participant stands in relation to a normative sample. With a sample of 39 ADHD adults and 39 controls, sensitivity of the scale was found to be 87%, specificity 85%, false positive rate was 15%, false negative rate was 13% and kappa was .72 (Conners, Erhardt, & Sparrow, 1999).

The Wender Utah Rating Scale (WURS; Wender, 1985) was administered to assess participants' childhood ADHD symptoms retrospectively. The entire scale contains 61 items on which adults are asked to rate themselves as

children, but the present study used the total score for the 25 items that best identify ADHD retrospectively (Ward et al., 1993). The rating system consists of a 5-point Likert-type scale from 0 (not at all or very slightly) to 4 (very much).

Brown Attention-Deficit Disorder Scale for Adults (ADDSA, Brown, 1996) is intended to assess symptoms of ADHD and symptoms often associated with ADHD in adults according to Brown's model, which is similar to the Utah criteria. The ADDSA is a 40-item self-report scale. The scale is grouped into 5 clusters: organizing and activating to work, sustaining attention and concentration, sustaining energy and effort, managing affective interference, and using "working memory" and access to recall. The endorsement of each item is made on a 4-point scale of how often something occurs (0-never, 1-once a week, 2-twice a week, 3-daily). Scores are added within clusters and then the clusters are added together to yield a total score. Cronbach coefficient alphas for the scale have been measured at .93 for a nonclinical population (n=143) and .86 for a clinical population (n=142), indicating good internal consistency (Brown, 1996). Test-retest reliability of the scale (two-week delay) was measured at .87 (Brown, 1996).

State Trait Anxiety Inventory form Y (STAI, Spielberger et al., 1983) is intended to assess both current and general anxiety levels. The STAI consists of 40 items (twenty items assess state and 20 assess trait). Alpha coefficients for the trait scale were .90 for males (n=324) and .91 for females (n=531) in a college population (Spielberger et al., 1983). The state scale alpha coefficients were .91 for males and .93 for females in the same population (Spielberger et al.,

1983). Test-retest reliability of the trait scale, in the same college population, ranged from .84 for 1 hour to .73 for 104 days for men, and from .76 for 1 hour to .77 for 104 days for women (Spielberger et al., 1983).

Validity of the Trait scale was obtained by comparing normals' scores with psychiatric patients' scores for whom anxiety was a major symptom. Spielberger et al. (1983) report that scores on the STAI discriminate well between the two groups. The STAI T-scale also correlated well with other measures of anxiety such as the IPAT Anxiety Scale (Cattell & Scheier, 1963, cited in Spielberger, 1983) and the Taylor Manifest Anxiety Scale (TMAS; Taylor, 1953), with correlations between the three ranging from .73 to .85.

Hypotheses and specific predictions for ADHD.

The present study was designed to test four possible outcomes for individuals with ADHD versus normals, based on four competing hhpotheses. Three of these predictions were of group (ADHD vs. normal) by condition (type of motivational cues) interactions with different parameters. The fourth prediction was for a main effect of group but with no interaction with motivational cues.

Hypothesis 1: Overactive BAS. According to the overactive BAS hypothesis, adults with ADHD could exhibit an over-responsiveness to reward cues, or over-responsive BAS. If so, participants with ADHD would commit more commission errors when reward cues are present than when absent, and would respond to rewards more strongly than controls. Therefore, there would be a group by condition interaction in which the ADHD group would show more

commission errors than controls only in the reward only and reward-response cost conditions.

Hypothesis 2: Under-active BIS. Alternatively, according to the underactive BIS hypothesis, ADHD participants could exhibit under-sensitivity to punishment cues, or under-responsive BIS (Quay, 1997). If so, participants with ADHD alone would commit more commission errors than normals only when response costs were presented. Therefore, there would be a group-by-condition interaction in which the ADHD group would make more commission errors in the response cost only and reward-response cost conditions only.

Hypothesis 3: Dysregulation of BAS over BIS. Still a third possiblity possibility is that adults with ADHD exhibit dysregulation of the BAS and BIS such that of they are insensitive to punishment cues when cues for reward are presented simultaneously (Newman & Wallace, 1993). If so, ADHD participants would make more commission errors only when both rewards and response costs were presented. Therefore, there would be a group by condition interaction in which the ADHD group would show higher commission error rates than controls in the reward-response cost condition only.

Hypothesis 4: General inhibitory deficit. Finally, adults with ADHD could have a generalized inhibitory deficit irrespective of reward or punishment cues (Barkley, 1997; Douglas 1988 cited in Barkley, 1997; Iaboni et al., 1995). If so, ADHD participants would make more commission errors than controls across all motivational conditions, but there would be no significant difference between

ADHD participants' performance between conditions. This would appear as a main effect for group with no group by task interaction.

Hypotheses and specific predictions for ADHD and anxiety

This study was also designed to test three competing hypothesis regarding the relatioship between anxiety and ADHD. as stated earlier, it is assumed that the STAI is measuring the same construct (anxiety) in the ADHD and control groups. This cross-group validity assumption will be checked before the Part II analysis is conducted. To check cross-group validity, correlations between the STAI and commission errors in the response cost only condition will be run for each group. Then correlations will be converted to Z-scores using the Fisher r-to-z transformation (Cohen, 1982) and compared with a Z-test.[1] If correlations between the two groups are not significantly different, and assuming the between groups results and direction of the anxiety-commission errors correlation makes the following analyses informative, hypotheses 5-7 will be tested.

Hypothesis 5: If high anxiety improves response inhibition (Gray, 1987), an ADHD inhibition deficit could be hidden (fewer errors) by high anxiety under certain response cost contingencies. If so, and assuming an ADHD inhibition deficit is found, after anxiety is covaried out, then the ADHD deficit would become larger under the response cost only condition.

[1] Cohen (1982) suggests using the Fisher r-to-z transformation when making comparisons between correlations as r is not normally distributed.

Hypothesis 6: If high anxiety yields poorer response inhibition (Newman & Wallace, 1993) an ADHD inhibition deficit could be made even worse by high anxiety. If this was the case, and again, assuming an ADHD inhibition deficit was found, then once anxiety level is covaried, the ADHD deficit would shrink (improve) under the response cost only condition.

Hypothesis 7: High anxiety may not affect the ADHD group's inhibition deficit. If the ADHD group demonstrates a response inhibition deficit under the response cost only condition, the effect of anxiety is not necessarily additive or subtractive in terms of this deficit. Therefore, once anxiety level is covaried, the ADHD group effect on commission errors may neither increase nor decrease under the response cost only condition.

Power and Sample Size

Effects in the literature for comparisons between ADHD and control groups (Pennington & Ozonoff, 1996) and effects sizes for studies using the go/no-go task (Milich et al, 1994; Newman et al., 1985) suggest an effect size of d = .50 or larger. Cohen (1992) suggest that for the design used here, a sample size of 23 participants per group would achieve a power of .76 with alpha set at .05, with a "medium" effect size (d=.50).

Data Analysis Plan

Part I. Test for effects of motivational contingencies on ADHD alone. To evaluate the first 4 hypotheses, interaction and main effects were examined in a 2 (group) x 3 (condition) mixed factorial ANOVA. If there is a non-significant interaction, but if the ADHD group made more commission errors than controls

across all conditions, hypothesis 4 would be supported. If an omnibus interaction is detected, then it would mean that motivational contingencies have different effects on ADHD and control participants. Planned comparisons would be carried out to isolate the source of the interaction, testing hypotheses 1-3.

Part II. Test for effects of anxiety. To make sure the STAI was measuring the same construct in the ADHD and control groups the correlations between the STAI and commission errors in the response cost only condition were compared across groups using the Fisher r-to-z transformation and then compared with a Z-test. It was planned that if correlations between the two groups were not significantly different, and if between-group effects and direction of the anxiety-commission errors correlation made the following analysis informative, the following analysis would be performed.

To evaluate hypotheses 5-7 the interaction and main effects would be examined in a 2 (group) x 3 (condition) mixed factorial ANCOVA with state and trait anxiety covaried. If commission error rate is greater for ADHD participants when anxiety is covaried out, hypothesis 5 will be supported, i.e., response inhibition is improved by anxiety. If commission error rate is lower for ADHD participants when anxiety is covaried, then hypothesis 6 will be supported: response inhibition is made even worse by anxiety. If commission error rate does not change for ADHD participants, then hypothesis 7 will be supported: anxiety does not affect response inhibition in ADHD.

Results

Preliminary Description.

As data are presented in the text, F values and p values are listed as appropriate, along with eta-squared (η^2). η^2 is an estimate of effect size, describing the proportion of total variance attributable to a factor (Cohen 1992). It is interpreted in a similar manner as r^2 is interpreted.

Although the measures used to assess ADHD (DSM-IV, CAARS, YASR, WURS, ADDSA) represent slightly different means of conceptualizing the construct, the ADHD group reported more symptoms of ADHD than the control group on all diagnostic measures ($p < .001$ in all cases, see Table 2). Thus, the diagnostic groupings appear to be valid.

Table 2 suggests that diagnostic groups were well matched in terms of age, sex and IQ. It also reveals that the ADHD group had higher scores than controls on the YASR aggression scale and the YASR delinquency scale. These results agree with studies suggesting that antisocial behavior is a frequent co-occurring problem in childhood and adult ADHD (Jensen, 1994; Mannuzza et al., 1991; 1993). The ADHD group also reported higher trait anxiety than controls but only marginally higher state anxiety. It should be noted that means for state or trait anxiety on the STAI did not reach the clinical range for either group (In the ADHD group n=4 reported clinically significant trait or state anxiety = T>65). In the control group, only1 reported clinically significant state anxiety, and only 3 reported clinically significant trait anxiety. Overall, the behavioral questionnaire

Table 2

Descriptive data for ADHD and control participants.

Questionnaire	ADHD Mean (SD)	Control Mean (SD)	P Value
Age (years)	21.95 (4.50)	20.91 (4.61)	.43
IQ	110 (12.04)	112 (12.45)	.58
Proportion males to females (raw number)	10/13	12/12	--
Childhood DSM-IV Attention (number of symptoms endorsed)	5.34 (3.17)	.87 (2.01)	< .001
Childhood DSM-IV Hyperactivity (number of symptoms endorsed)	5.96 (3.20)	1.16 (2.51)	< .001
ADDSA Attention (T-score)	77.04 (15.01)	59.13 (12.13)	< .001
ADDSA Activity (T-score)	75.35 (17.27)	55.45 (8.92)	< .001
CAARS Attention (T-score)	77.23 (11.00)	46.46 (7.93)	< .001
CAARS Hyperactivity (T-score)	71.78 (11.21)	41.58 (8.41)	< .001
YASR Attention (T-score)	65.91 (8.06)	56.30 (6.40)	< .001
YASR delinquency (T-score)	60.68 (9.38)	55.09 (5.25)	.02
YASR aggression (T-score)	56.78 (6.39)	54.41 (5.75)	.004
WURS (raw score)	41.17 (11.16)	10.38 (7.48)	< .001
STAI state anxiety (raw score)	41.09 (11.55)	35.54 (9.99)	.09
STAI trait anxiety (raw score)	45.96 (11.72)	35.25 (6.79)	< .001
YASR anxiety/depression (T score)	56.48 (10.97)	52.91 (4.42)	.16

Abbreviations represent the following. ADDSA: Brown Attention Deficit Disorder Scale for Adults; CAARS: Conners Adult ADHD Rating Scale; YASR: Young Adult Self Report Scale; WURS: Wender Utah Rating Scale; STAI: State-Trait Anxiety Inventory

data support diagnostic groupings and reflect expected differences between groups.

The Go/no-go Task Preliminary Data.

On the go/no-go task, commission error rates were highest in the reward only condition, slightly lower in the response cost only condition, and lowest in the reward-response cost condition, though the differences between these conditions did not reach significance F (2,44) = 2.66, p = .08, η^2 = .056. The marginal difference between commission errors on the three conditions suggests that motivational conditions did not have the predicted effect on responding whereby commission errors would be most frequent in the reward only condition and least frequent in the response-cost only condition.

Women made marginally more errors than men F (1,44) = 3.11, p = .09, η^2 = .06. overall, but no group by sex interaction was found F (1,43) = 1.28, p = .26, η^2 = .03. Older age correlated with more errors in the reward only condition (r = .31, p = .04), but not in the response cost only (r = -.05, p = .72) or reward/response cost conditions (r = -.02, p = .88). Higher IQ was associated with fewer commission errors across the reward (r = -.35, p = .01), response cost (r = -.36, p = .01), and reward/response cost (r = -.50, p <.001) conditions.

Table 3

Commission errors across motivational conditions and groups.

Motivational Condition	ADHD Mean (SD)	Control Mean (SD)	Total Mean (SD)
Reward only	11.57 (8.04)	11.67 (8.64)	11.61 (8.26)
Response cost only	9.87 (7.16)	12.75 (7.74)	11.34 (7.52)

Reward/response cost	8.73 (5.17)	9.45 (5.49)	9.11 (5.28)
Total	10.06 (6.79)	11.29 (7.29)	10.69 (7.02)

The effect of motivational cues on ADHD alone.

A 2 (group) x 3 (motivational condition) mixed factorial ANOVA was performed to test hypotheses 1-4. There was no main effect of group F (1,45) = .61, p = .44, η^2 = .01, or group x motivational condition interaction F (2,90) = .75, p = .47 , η^2 = .02. In post hoc analyses, diagnosis was not related to commission errors in the reward only F (1,45) =.00, p = .96 , response cost only F (1,45) =1.75, p = .19 , or reward/response cost condition, F (1,45) = .21, p = .65. The group main effect remained non significant when age (p = .38), sex (p = .37), IQ (p = .21), and scores on the YASR aggression scale (p = .74) and the YASR delinquency scale (p = .66) were covaried. Inspection of the means in Table 3 reveals that the ADHD group actually performed slightly better (non-significant) than the control group, suggesting that null results were not due to limitations in power or sample size.

Thus, Hypotheses 1-4 were not supported. A 2 (group) x 3 (motivational condition) mixed factorial ANOVA was performed on the total cash won. This was done to determine whether the ADHD group may have been more motivated to do well *overall*, either by the motivational manipulations (in a way that was not necessarily consistent with Gray's model) or by some other factor (such as interest in the study). The ADHD group did not perform significantly better on the task than the control group F (1.45) = .11, p = .74, η^2 = .00, and no significant

interaction was found for diagnosis x money won between motivational conditions $\underline{F}$ (2.45) = .38, $\underline{p}$ = .68, η^2 = .01. To determine whether a difference in omission errors accounted for the fact that ADHD participants made fewer commission errors but did not win more money, a 2 (group) x 3 (motivational condition) mixed factorial ANOVA was performed on omission errors, with no difference between groups F (1,44) < .01. Thus the hypotheses that (1) the ADHD group would show worse overall performance on the task than the control group, or (2) that the motivational conditions would have different effects the ADHD and control groups, were not supported.

The effect of anxiety.

Before running an ANCOVA to determine the effects of anxiety, a check was made to determine that the STAI was measuring the same construct (anxiety) in both groups. First, a correlational analysis was performed on state anxiety, trait anxiety, and commission errors in the reward only, response cost only, and reward-response cost conditions for the ADHD and control groups, separately and together.

As seen in Table 4 (next page), the control group's state anxiety correlated with errors in the reward-response cost condition only, while in the ADHD group, state anxiety correlated in the reward only and response cost only conditions. Trait anxiety in the control group correlated with errors in the reward only condition while trait anxiety in the ADHD group correlated with errors in the response cost only condition.

Unexpectedly, significant correlations were not confined to the response cost only or reward-response cost conditions. In light of these results, correlations were then converted to Z-scores using Fisher's r-to-z computation and compared across conditions, rather than only making comparisons in the conditions containing response costs.

Table 4

Correlation between anxiety and commission errors in ADHD and control groups.

	STAI state anxiety			STAI trait anxiety		
Motivational Condition	**Control Group**	**ADHD Group**	**Across Groups**	**Control Group**	**ADHD Group**	**Across Groups**
Reward only	r =.17 p =.42	r =.41 p =.04*	r = .29 p =.04*	r = .61 p =.001*	r = .25 p =.25	r = .33 p = .02*
Response cost only	r =.25 p =.24	r =.55 p =.007*	r = .33 p = .02*	r =.34 p =.11	r = .44 p =.04*	r =.24 p = .10
Reward-response cost	r =.49 p =.02*	r =.23 p =.30	r = .32 p = .03*	r =.01 p =.95	r = .11 p =.60	r =.01 p =.91
Total across conditions	r =.39 p =.06	r =.50 p =.01*	r = .41 p = .005*	r = .56 p =.004*	r = .33 p =.12	r = .29 p = .05*

* indicates a significant finding at $p < .05$

Of the 8 between group comparisons (state and trait), two were significant and in opposite directions: ADHD adults had a larger anxiety-commission errors correlation than controls for state anxiety in the response cost only condition; controls had a larger correlation for trait anxiety in the reward condition. Overall, there was no clear directional trend and the average omnibus correlations did not differ. That two correlations were significantly different between groups could suggest that the STAI state scale did not measure the same construct between

groups. The fact that the two significant correlations were in the opposite direction makes this suggestion tentative

Because correlations were not confined to the expected conditions, the same simple correlation was performed (STAI state and strait scores by the three motivational conditions) across groups to see if increasing power would clarify results. As the Table 3 shows, trait anxiety correlated with errors in the reward only condition while state anxiety correlated with errors across all three conditions.

Although significant correlations were not confined to the expected conditions (reward-response cost and/or response cost only), note that all correlations were in the positive direction. These findings were inconsistent with a simpler view of BIS functioning, but were consistent with Newman's theory of anxious impulsivity (Newman & Wallace, 1993). As stated in the Introduction, anxious impulsivity can occur because anxiety levels are indicative not only of high BIS functioning, but also of high arousal, especially if passive avoidance responding is not possible (as was thought to occur in the response cost condition, wherein participants are punished for passive avoidance). It is possible that some uncontrolled cue for punishment existed across conditions (such as embarrassment when responses were incorrect), which may have induced this anxious impulsivity.

Because faster reaction time may also be an indicator of anxious impulsivity, a correlational analysis was run on reaction time, state anxiety scores, and trait anxiety scores. Yhese correlations, however, were non-

significant. Trait anxiety was not significantly correlated with the reaction time of correct responses (r = -.21, p = .15) or reaction time of incorrect responses (r = .16, p = .28). State anxiety was also not significantly correlated with reaction time of correct responses (r = -.21, p = .15) or reaction time of incorrect responses (r = .00, p = .98). Although correlations were in the predicted direction for correct responses, incorrect responses did not appear to be due to faster responding.

Results thus far have shown a non-significant difference in commission errors between the ADHD and control group on the go/no-go task and a positive correlation between anxiety and commission errors. Given this, the results of an ANCOVA did not promise to clarify the relationship between anxiety and ADHD. Because anxiety levels were higher in the ADHD group, if anxiety is covaried, it can serve only to further boost ADHD performance in relation to controls.[2] Although it appears as though this could support hypothesis 6 – that anxiety may contribute to response disinhibition in ADHD, the fact that an ADHD inhibition deficit was not found makes the hypothesis moot as far as the explanation of clinical problems is concerned.

The Constraints of Memory.

To verify that Go/no-go performance was not due to differences in learning and working memory abilities, Digit Span, free recall of stimuli, and cued recall of stimuli were measured. Groups did not differ on Digit Span F (1,44) = .13, p =

[2] In fact, after stat state anxiety was covaried in an ANCOVA the results showed that the ADHD group made fewer commission errors than controls on the go/no-go task F (1,21) = 7.08, p = .02, η^2 = .25.

.71, free recall of stimuli F (1,44) = .97, p = .33, or recognition memory of stimuli F (1,44) = 1.36, p = .25). Note that the mean differences in performance on these tasks, however, are in the opposite direction from those on the Go/no-go task, with the ADHD group performing slightly worse on the memory tasks (see Table 5, next page). This makes it unlikely that memory task performance would explain the error rate data between the groups. Differences between diagnostic groups on memory task performance remained non-significant when IQ, state anxiety, and trait anxiety were covaried. Also, effects of diagnosis on go/no-go task performance remained non-significant when Digit Span, free recall of stimuli, and cued recall of stimuli were covaried. Thus, it does not appear that working memory abilities explain the results of the ADHD and control groups on the go/no-go task.

Table 5

Measures of Memory across the ADHD and control group.

Task	ADHD Mean (SD)	Control Mean (SD)	P Value
Mean Digit Span raw score	17.50 (4.07)	17.95 (4.24)	.71
Free recall (number recalled)	14.42 (3.10)	15.28 (2.43)	.33
Recognition Memory (number recalled)	16.35 (1.81)	17 (1.80)	.25

Discussion

Motivational inhibition

The purpose of this study was to test competing theories of a motivational inhibition deficit in ADHD, using a version of Newman's (Newman et al., 1985) modified Go/no-go task. Although previous studies examined adult psychopaths, and other studies examined children with ADHD, this was the first study of a motivational inhibition deficit in adults with ADHD. Prior studies using the go/no-go task have found mixed results in children and adolescents, indicating that either (a) an inhibitory deficit in ADHD may be related to a response modulation problem, which is visible when rewards and response costs are simultaneously presented (Milich et al., 1994), or (b) a primary deficit in ADHD may well be inhibitory in nature but not because of an underlying deficit in motivational inhibition, per se (Iaboni, et al., 1995). The current study of adults did not support either of those prior child findings, or other current theories that posit a motivational inhibition deficit in ADHD (e.g., Quay, 1988; Newman & Wallace,1993). These results, together with the mixed outcomes of past studies of children, suggest that Quay's motivational model and the go/no-go paradigm may not be promising in determining the nature of inhibitory deficits in ADHD.

With respect to the question of anxiety, some analyses were rendered moot because the ADHD group did not make excess commission errors on the go/no-go task and because anxiety correlated with more, rather than fewer, commission errors. Nevertheless, the results support the possibility that anxiety may inflate ADHD symptom endorsement in the absence of an ADHD-specific

cognitive deficit. Given that high anxiety was correlated with more commission errors on the go/no-go task, a result that supported Newman's theory of anxious impulsivity (Newman & Wallace, 1993), one could speculate how high anxiety levels might exacerbate an inhibition deficit in ADHD. However, because ADHD participants did not display an inhibitory deficit on the go/no-go task, covarying anxiety with ADHD did not help to explain the relationship between anxiety and ADHD.

Findings: ADHD versus control group

ADHD adults reported more anxiety, aggression, and delinquency than the control group as is typical of such samples (Beiderman et al., 1993; Jensen et. al., 1997). These comorbid problems did not account for the results, however.

If an ADHD inhibitory deficit could be explained by a motivational inhibition theory one would have expected an interaction effect of diagnostic group and motivational condition on commission errors. No such interaction effect was found. Thus, while 2 prior studies of ADHD children found either marginal evidence of hypoactive BIS activation (Daugherty, Quay, and Ramos, 1993) or BAS/BIS dysregulation (Milich et al., 1994) in childhood, the present study supported neither theory in ADHD adults. The present finding agreed with Iaboni et al.'s study (1995), which found no group by condition interaction, which suggests no effect of motivational condition on ADHD performance. Unlike Iaboni et al. (1995), however, the present study failed to find an overall ADHD deficit in commission errors.

Possible Limitations

The failure to find an ADHD deficit was not likely to be due to insufficient power. Effect sizes for hypothesized outcomes in the present study were either very small or in the *opposite* direction as predicted and did not replicate the effect sizes of previous studies. No results were due to comorbid disorders.

There are a number of reasons why the present study may not have found an ADHD inhibitory deficit on the go/no-go task. One could argue that the present study's ADHD participants were not representative of those with ADHD on the whole. It is true that the present study used a high-functioning ADHD population (most participants were college students). Nevertheless, behavioral impairment was evident in this sample (see Table 2). Furthermore, a different study using the same ADHD sample found evidence of a behavioral inhibition deficit on an antisaccade task (Nigg et al., in review).

It is also possible that the ADHD group was more motivated to do well overall than the control group. It could be that they were generally more excited by the prospect of winning money, or they were more engaged in the study overall because they knew that the study was about ADHD. This might have cancelled out any effects of disinhibition that could have otherwise been apparent.

The fact that the ADHD group in the current study showed a deficit on an anti-saccade task, which is known to require inhibitory performance, suggests that the go/no-go task was not sensitive to an inhibitory deficit associated with ADHD. The go/no-go version used may have been too simple a task to elicit this

inhibitory deficit in adults with ADHD. It is possible that such a deficit would become apparent only beyond a certain threshold of task difficulty. But if this is so, one would not expect to find such a high degree of variability in the number of errors made between participants (i.e. there was no ceiling effect). Furthermore, many other tasks requiring forms of behavioral inhibition (such as Logan's stop task) have found deficits in ADHD, without explicit manipulation of motivational cues. This suggests that tasks requiring behavioral inhibition are likely to be necessary and sufficient to tap deficits in ADHD while, thus far, tasks requiring motivational inhibition have not demonstrated the same degree of reliable results.

If the go/no-go task were able to tap the proposed systems of motivation, one would have expected a main effect on errors rates across the groups. This effect was marginally significant (p=.08), suggesting that the reward and response cost cues may not have had a strong effect in activating the proposed systems of motivation. In particular, error rates were nearly identical in the reward and response cost conditions. Differences in commission errors may not have been evident between conditions because, as mentioned above, the motivational cues appear not to have had the desired effect on response modulation. There are a number of potential reasons for this, including the possibility that the monetary reward/punishment could have been too small to affect the proposed BAS or BIS. Furthermore, the response costs may not have been perceived as cues for punishment at all, since the money the participants were losing had been given to them at the beginning of the condition. Given that as anxiety increased *across* conditions commission errors also increased, some

other cue for punishment (such as embarrassment) may have existed in all conditions. One aspect of the design of the study also may not have been ideal: the within-subjects design wherein each participant was exposed to all 3 conditions, which means they may have failed to differentiate between the conditions.

Although the results are in line with other studies that found no motivational inhibition deficit in ADHD, they cannot specifically refute the existence of such a deficit in childhood, since the participants in the present study were adults. If such a motivational deficit does exist in childhood ADHD, and if the present data are accurate, then the absence of the deficit in ADHD adults needs to be explained. It is conceivable that if a motivational inhibition deficit does exist in children with ADHD (either in its own right or as a result of a primary behavioral inhibition deficit, as Barkley [1997] suggests), it could abate with development while a behavioral inhibition deficit remains. Those who successfully cope with ADHD (a likely characteristic of the ADHD college student group in this study) might have learned to exert more self control over their own motivation as adults, even while a behavioral inhibition deficit remains, such that they become better able to cope with such a deficit.

The role of anxiety

Between groups, high anxiety correlated with more commission errors under many conditions, but not in a predicted pattern. Across groups, higher state anxiety was related to more commission errors in *all* conditions. This further challenges the validity of the go/no-go design, suggesting, again, that the

motivational cues overall did not have the theorized differential effects on the BIS and BAS. If one supposes that participants were motivated not to make a mistake by a factor other than the monetary punishment cues (such as by the fact that they were being watched by a tester who would observe their mistake), then it is possible that cues for punishment existed more or less equally across all conditions. Given this possibility, Newman's theory of anxious impulsivity (Newman & Wallace, 1993) would explain the positive correlation between commission errors and anxiety level. Anxious impulsivity, manifest in the form of commission errors, may occur in the face of punishment cues if participants must respond to those cues.

Although this dysregulated responding was evident in error rate, it did not appear to significantly affect reaction time in the present study. In a previous study, such anxious impulsivity affected reaction time but did not lead to a significantly increased error rate (Wallace et al., 1991). This difference in results could be due to the very different experimental tasks. The go/no-go task required a key press in response to numbers that were tied either to rewards or response costs. Wallace and Newman's (1991) study involved tracing a circle template while participants had a chance of winning or losing money (which occurred while they were tracing but that had no direct connection to the quality of their tracing). Also, participants in Wallace and Newman's (1991) study were instructed to draw as slowly as possible. In the present study participants were given no instructions about the speed of their response. The two-second time

limit within trials of the go/no-go task also may have restricted the range of reaction time results, while the circle drawing task had no time limit.

When correlations between the self-report anxiety measures and commission errors were compared between groups, only 2 of the 8 differences were significant but in opposite directions of each other. This could suggest that the STAI is not measuring the same construct in ADHD participants and controls. However, because they were in opposite directions and omnibus correlations (collapsed across condition) for each group were not significantly different from each other even though no condition clearly tapped anxious impulsivity better than another condition, this interpretation is tentative.

Given the combined facts that (1) the ADHD group did not show an inhibitory deficit on the go/no-go task, (2) higher anxiety was generally correlated with more commission errors on the task, and (3) the ADHD group reported higher anxiety than the control group, the analysis of ADHD and anxiety was rendered moot. Even though the ADHD group did not demonstrate an inhibitory deficit on the go/no-go task, the covariance analysis could have been informative if high anxiety had been correlated with fewer commission errors on the task. This would have left open the possibility that anxiety was hiding an inhibitory deficit in the ADHD group. However, because anxiety correlated with *more* errors on the go/no-go task, covarying out its effects could only make the ADHD group appear even *less* disinhibited on the task, a largely uninformative result since it does not explain the inhibitory deficit in ADHD.

Conclusions

The present study found no evidence of a motivational inhibitory deficit in adults with ADHD. There was a secondary finding that high levels of anxiety could lead to anxious impulsivity, but this combination of findings made any analysis of the interplay of anxiety and ADHD uninformative.

Future studies using the go/no-go task should use an alternative method to ensure that rewards and response costs are having the desired effect on motivation. One possibility is to recruit more subjects so that a between groups design can be used without sacrificing power. This would eliminate the possibility that motivational cue contingencies could confound results between conditions. One could also add a Likert-type scale asking participants to what degree they were motivated to win or not to lose money, to perform well in front of the tester, or to do a good job for themselves. Given past findings that Gray's theory accounts well for disorders on the antisocial behavior spectrum (e.g., conduct disorder and psychopathy), it is also imperative that future studies control these comorbid disorders, which often occur with ADHD. It would also be beneficial for future research to focus on different effects of normal levels versus clinical levels of anxiety on response modulation. It is not clear whether the difference between the two is only one of degree or whether they will exert qualitatively different effects on response modulation.

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