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Differential Effects of Personality Disorders on Treatment Outcome for Adult Patients with ADHD

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THE DIFFERENTIAL EFFECTS OF PERSONALITY DISORDERS ON

TREATMENT OUTCOME FOR ADULT PATIENTS WITH ADHD

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Dissertation Approval

This is to certify that the thesis presented to us by Emily M. Tomer on the __24__ day of

__May__, 20_10_, in partial fulfillment of the requirements for the degree of

Doctor of Psychology, has been examined and is acceptable in both scholarship and

literary quality.

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Abstract

Approximately seven million adults meet the diagnostic criteria for attention deficit/hyperactivity disorder (ADHD) and the prevalence rate is between 3.3 and 5.3%; however, these are likely underestimates because the diagnostic criteria are based on those originally devised for children, aged four to 17 years. Not only does this underdiagnosis cause a problem in making an accurate diagnosis, but also the high rate (65-89%) of psychiatric comorbidity complicates the situation, resulting in poorer treatment outcome for individuals with comorbid diagnoses, when compared with those who have no comorbid diagnoses. Therefore, the current study was designed to examine whether or not the presence of personality disorder symptoms will hinder treatment and result in poorer treatment outcomes in individuals with ADHD. This is an archival study, based on Rostain and Ramsay (2006). The hypotheses were as follows: 1) There will be a significant reduction in symptoms of ADHD and mood disorders after a combined treatment including medication and cognitive behavioral therapy; 2) There will be a positive association between symptoms of ADHD and maladaptive beliefs, both pre- and post-treatment; 3) The presence of maladaptive thinking, attendant to personality disorders, will interfere with reductions in ADHD symptoms; and 4) The presence of a personality disorder will interfere with reductions in symptoms of mood disorders. Forty-three participants took part in the study; however, only 35 reports were retained for this study. The first hypothesis was supported, and the second hypothesis was partially supported. The third hypothesis was not supported. Interestingly, in terms of the fourth hypothesis, those with symptoms of dependent, antisocial, and avoidant personality disorder appeared to have a greater improvement of mood symptoms, post-treatment.

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Chapter One: Introduction

Statement of the Problem

Attention deficit/hyperactivity disorder (ADHD) was originally conceptualized as a psychiatric disorder, diagnosed in childhood, which could persist into adolescence (American Psychiatric Association [APA], 2000). In the past few decades, it has been accepted that ADHD symptoms can continue into adulthood in up to 70% of individuals diagnosed with ADHD (Barkley, 2006; Klein & Mannuzza, 1991; Mannuzza & Klein, 1999; Wilens, Biederman, & Spencer, 2002).

More specifically, it is estimated that approximately seven million adults are currently diagnosed with ADHD (Kirley & Fitzgerald, 2002). In the United States, Barkley (2006) found that the prevalence rates for adult ADHD ranged between 3.3 to 5.3%. Similarly, the international prevalence rate of ADHD in adults was found to be 3.4%, indicating that ADHD is a worldwide concern and a problem not only in the United States (Fayyad et al., 2007). Fayyad et al. (2007) found that higher-income countries have prevalence rates of approximately 4.2%. Also, the diagnosis of adult ADHD is more common in urban settings and in northeastern and north central states (Kessler, 2006). Because the normative data of the diagnostic criteria of the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR) (APA, 2000) are based on children aged 4 to 17 years, it is likely that these previously cited prevalence rate percentages are underestimated because there are no developmentally appropriate criteria to diagnose adults with ADHD (Finkel, 1997; Rosenfield, Ramsay, & Rostain, 2008; Wender, Wolf, & Wasserstein, 2001).

Problems Experienced by Adults Diagnosed with ADHD

In early childhood, children growing up with ADHD often display aggression, temper tantrums, disorderly behavior, and increased irritability, especially when the disorder is not treated properly (Woodard, 2006). Then in later childhood, individuals diagnosed with ADHD may experience attitude problems, decreased self-esteem, poor peer interactions, increased lying, problems within the school setting, as well as problems with concentration, impulse control, awareness, organization, frustration tolerance, risky behaviors, and poor decision-making skills (Anckarsäter et al., 2006; Kirley & Fitzgerald, 2002; Woodard, 2006).

As adolescents, these individuals may have engaged in defiant behaviors, dropped out of school, engaged in disrespectful behaviors towards adults, experienced increased problems in the school setting, experienced feelings of worthlessness, had driving violations and accidents, had increased propensity to use and abuse drugs and alcohol, and engaged in risky sexual activity (Woodard, 2006). With these symptoms, it is not surprising that they experienced a higher prevalence rate of oppositional defiant disorder (ODD), conduct disorder, and major depressive disorder (Barkley, 2006; Ramsey & Rostain, 2008). Thus as adults, individuals with ADHD are at a higher risk for developing multiple psychiatric disorders and symptoms than is the general population (Biederman et al., 2006).

As adults, individuals diagnosed with ADHD experience significant impairments in social and occupational functioning, as well as in a variety of learning environments (Hansen, Weiss, & Last, 1999). More specifically, they are significantly more likely to have such difficulties as problems in completing school, in earning lower salaries, in

frequent job changes, increased divorce rates, pessimism, decreased life satisfaction, and

legal problems (Conners et al., 1999; Ramsay & Rostain, 2008; Rosenfield, Ramsay, &

Rostain, 2008; Woodard, 2006). Adults diagnosed with ADHD also engage in higher

rates of healthcare utilization and prescriptive drug use, resulting in greater overall

medical costs when compared with other individuals seeking medical treatment (Secnik,

Swenson, & Lage, 2003). These individuals have also been found to have significantly

more traffic citations, traffic accidents, and motor vehicle violations, as well as increased

risks of injury, suspension of their drivers' licenses, and being caught drinking and

driving (Barkley, Murphy, DuPaul, & Bush, 2002; Murphy & Barkley, 1996; Woodward,

Fergusson, & Horwood, 2000).

Comorbid Psychological Problems and Adult ADHD

There is a high rate of psychiatric comorbidity in individuals diagnosed with

ADHD. For instance, prevalence rates of psychiatric comorbidity for adults diagnosed

with ADHD have been found to range from 65-89% (Sobanski, 2006; Wender et al.,

2001). These high rates of comorbid disorders are problematic, because they significantly

obscure accurate clinical diagnoses and appropriate treatment planning, which can, in

turn, affect treatment outcome (Sobanski, 2006; Wender et al., 2001). Overall, adults

diagnosed with ADHD are more likely to experience increased levels of psychological

distress, which negatively impacts their life satisfaction, life options, identities, outlook

on life, and self-acceptance (Ramsay & Rostain, 2008).

More specifically, comorbid disorders for adults diagnosed with ADHD include

substance use disorders (i.e. there is a significantly elevated lifetime prevalence of

substance abuse and dependence, especially with alcohol, marijuana, cocaine, stimulants,

opiates, and nicotine), learning disorders, developmental disabilities or mental

retardation, obsessive-compulsive disorders, autism spectrum disorders, conduct

disorders, oppositional defiant disorders, major depressive disorders, dysthymia, bipolar

disorders, generalized anxiety disorders, social phobia, posttraumatic stress disorders,

somatization disorders, panic attacks, bulimia nervosa, sleeping difficulties, and

personality disorders (Barkley, 2006; Barkley, Murphy, & Fischer, 2008; Biederman et

al., 2006; Gillig, Gentile, & Atiq, 2005; Jacob et al., 2007; Sobanski, 2006). Jacob et al.

(2007) reported that the most prevalent comorbid disorders for adults diagnosed with

ADHD are depression and anxiety (i.e., 30-50% experience a lifetime prevalence of at

least one depressive episode and 40-60% suffer from an anxiety disorder).

Because the focus of this paper is on personality disorders, it is important to note

that ADHD has also been found to be associated with extremes of personality traits.

(Nigg et al., 2002). Miller, Nigg, and Faraone (2007) reported that the ADHD behaviors

of impulsivity and behavioral dysregulation "suggest a theoretical connection between

ADHD symptoms and personality traits and, by extension, personality disorders, which

are defined as chronic, maladaptive personality traits" (p. 520). It is also possible that

ADHD may alter personality, increasing the risk for the development of personality

disorders.

Moreover, these personality traits can amplify the effects of ADHD (Miller et al.,

2007). Problematic personality characteristics have been found to hinder treatment and

prolong the suffering of these individuals because of their distorted cognitions, affective

dysregulation, and poor interpersonal functioning (APA, 2000; Rostain & Ramsay,

2008). It may also be that the relationship between ADHD and personality disorders

might be phenomenological due to the overlapping diagnostic criteria (Miller et al.,

2008). It may also be that these disorders co-occur because of common neurobiological

and/or risk factors (e.g. hypothalamic-pituitary-adrenal axis deysregulation and/or

adverse early experiences with fear or anger). For example, research has found a

relationship between borderline personality disorder and ADHD, possibly due to shared

similar clinical features, such as emotional dysregulation and impulsivity (Philipsen et al.,

2008).

Purpose of the Study

Because many adults diagnosed with ADHD seek psychological treatment to help

to reduce suffering and improve their day-to-day functioning, it is logical to determine

the effectiveness of treatment outcome. Complicating treatment is the fact that 65-89% of

adults diagnosed with ADHD meet symptom criteria for one or more additional

psychological disorders, resulting in diagnostic problems, inappropriate treatment

planning, and poorer treatment outcome (Sobanski, 2006; Wender et al., 2001).

As the existing research suggests, those who suffer from more severe ADHD

symptoms, along with one or more comorbid disorders, may have poorer treatment

outcome than those with a less complicated ADHD diagnosis (APA, 2000; Rostain &

Ramsay, 2008). In fact, the combination of ADHD and one or more personality disorders

is increasingly likely to hinder treatment, prolonging the suffering of these individuals

because of their distorted cognitions, affective dysregulation, and poor interpersonal

functioning, which negatively affect their life functioning (APA, 2000; Rostain &

Ramsay, 2008). Therefore, the current study is designed to further examine whether or

not the presence of personality disorder symptoms will hinder treatment in adults

diagnosed with ADHD and thus result in poorer treatment outcome than that experienced

by those with ADHD alone.

Overview of the Literature Review

Researchers make it clear that a widely-accepted diagnostic criteria for adult

ADHD is not available and those that have been proposed are not adequate to

conceptualize this diagnosis (Barkley, 2006; Magnússon et al., 2006; Murphy &

Schachar, 2000; Ramsay & Rostain, 2008; Trott, 2006). For instance, the criteria for

inattention and hyperactivity-impulsivity do not take into consideration the fact that when

ADHD children become adults, many symptoms may decrease with maturation (Barkley

et al., 2008; Conners et al., 1999; Finkel, 1997). Specifically, when one ages, the

symptoms of hyperactivity and impulsivity decrease and there are fewer objective signs

of physical restlessness; however, there are increases in a subjective sense of restlessness,

mental activity, and fidgetiness (Ramsay & Rostain, 2008). It is possible that the lack of

literature on the subject of adult ADHD results from this conceptualization difficulty.

Another complication is that adult ADHD has been found to have many comorbid

diagnoses, including personality disorders. The lack of diagnostic criteria and the

accompanying comorbid diagnoses further complicate the clinician's ability to plan

appropriate treatment, to make accurate diagnoses, and to provide positive treatment

outcomes (Barkley, 2006; Ramsay & Rostain, 2008).

Relevance to Cognitive Behavior Therapy

Beck (1976) was the first to conceptualize cognitive therapy as a coherent

treatment modality designed to treat a specific disorder. The overarching theory of this

model is that one's mood and behavior are affected by one's thoughts. Cognitive therapy

"alleviates[s] psychological distress through the medium of correcting faulty conceptions and self-signals"; access to one's emotions, which is usually the source of distress, occurs through one's cognitions (Beck, 1976, p. 214).

More specifically, cognitive distortions are irrational thoughts or ideas that maintain negative thinking and help to maintain negative emotions (e.g. all-or-nothing thinking, jumping to conclusions, making "should" statements) (Beck, 1976). Cognitive behavioral therapy (CBT) posits the idea that one's distorted thinking can be identified and corrected, which can reduce or eliminate problematic symptoms. In order to help in this process, this model focuses on identifying and correcting distorted cognitions, underlying dysfunctional beliefs, and disconfirming maladaptive beliefs, as well as on improving reality testing (Beck, 1967; Beck, 1976; Beck, Rush, Shaw, & Emery, 1979).

Because individuals diagnosed with ADHD tend to undergo many negative experiences, their belief systems may be altered in terms of how they view themselves, the world, and the future (Beck et al., 1979). In CBT, these maladaptive cognitions are identified and alternative thoughts and beliefs are explored (Ramsay & Rostain, 2008). In fact, the greatest impact of CBT in the treatment of ADHD may result from the fact that patients learn to use appropriate self-talk and coping skills; that is, more accurate cognition and more adaptive behavior (Ramsay & Rostain, 2007; Rosenfield et al., 2008).

Chapter Two: Review of the Literature

ADHD: History of a Diagnosis

The first description of what is currently known as Attention Deficit/Hyperactivity Disorder (ADHD) occurred in Heinrich Hoffman's 1844 poem about a hyperactive child named "fidgety Phil" (Barkley, 2006; Hoffman, 1844; Trott, 2006).

> Let me see if Philip can/ Be a little gentleman;/Let me see if he is able To sit still for once at table:/Thus Papa bade Phil behave;/And Mamma looked very grave./But fidgety Phil,/He won't sit still;/He wriggles, And giggles,/And then, I declare,/Swings backwards and forwards, And tilts up his chair,/Just like any rocking-horse-/"Philip! I am getting cross!"

> See the Naughty, restless child/Growing more rude and wild,/Till his chair falls over quite./Philip screams with all his might,/Catches at the cloth, but then/That makes matters worse again./Down upon the ground they fall,/Glasses, plates, knives, forks, and all./How Mamma did fret and frown,/When she saw them tumbling down!/And Papa made such a face!/Philip is in sad disgrace.

> Where is Philip, Where is he?/Fairly covered up you see!/Cloth and all are lying on him;/He has pulled down all upon him./What a terrible to-do!/Dishes, glasses, snapped in two!/Here a knife, and there a fork!/Philip, this is cruel work./Table all so bare, and ah!/Poor Papa, and poor Mamma/Look quite cross, and wonder how/They shall have dinner now.

In 1902, George Still and Alfred Tredgold were the first individuals to focus on this problem as a clinical entity (Barkley, 2006; Bradley, 1950). Still believed that inattention and hyperactivity were due to a deficit in moral control. Such moral deficiency was evidenced by aggression and rebelliousness, which in turn, resulted from three factors: a) a discrepancy between the child's cognitions and his/her environment; b) a deficit in the child's moral consciousness; and c) a deficient ability of the child to inhibit his/her behaviors (Barkley, 2006; Wender et al., 2001). Still's hypothesis was that

these three deficits were related to each other, as well as to the same underlying neurological deficit (Barkley, 2006). Still found that this lack of "moral control" had a tendency to be a chronic condition, indicating for the first time that ADHD could persist into adulthood (Barkley, Murphy, & Fischer, 2008).

After the 1918 influenza epidemic, symptoms similar to the current conceptualization of ADHD were noticed in children who had suffered and recovered from encephalitis lethargica (Cheyette & Cummings, 1995). The symptoms that occurred during this illness included severe lethargy and stupor; however, upon recovery, related pathology appeared in the central nervous system, including severe damage in the substantia nigra with less extensive damage in the hypothalamus and the lenticular nucleus, the latter comprising the putamen and the globus pallidus within the basal ganglia. Damage to the lenticular nucleus and hypothalamus was found to lead to problems in attention, mood lability, anger, and conduct.

During the 1920s, Hohman treated this "postencephalitic behavior disorder" in children who had difficulty with behavioral problems and headaches (Finkel, 1997). Although his treatment was originally intended to target headache symptoms, Hohman may have been the first to use stimulant medication for this syndrome, with the notion that stimulation of the central nervous system increased the rate of cerebral spinal fluid production, reducing post lumbar puncture headaches (Finkel, 1997). Even though he found that stimulants were not helpful in reducing headaches, they were serendipitously effective in improving the children's behavioral problems (Finkel, 1997). Similarly, Bradley (1937) found that Benzedrine significantly improved inattention and restlessness in these children. Despite these early findings, it was not until the 1960s that the first

papers on adult ADHD were written, with recommendations for the use of stimulant

medication and therapy (Barkley, 2006; Barkley et al., 2008).

Also during this time period, the terms "organic driveness," "brain-injured child,"

"minimal brain damage," "minimal brain dysfunction," and "restlessness syndrome"

were used to label symptoms of ADHD (Barkley, 2006; Clements, 1966). Any milder

form of hyperactivity was perceived as being a result of "psychological causes, such as

'spoiled' childrearing practices or delinquent family environments" (Barkley, 2006, p. 6).

When, in the 1960s, these children were found to have vague signs of

neurological impairment, the conceptualization of "minimal brain damage" began to fade

(Barkley, 2006). This finding improved perceptions of this disorder. For example,

because the focus was taken away from neurological problems, researchers were able to

collect data on a child's increased activity level (Barkley et al., 2008). Also, the belief

that parents were to blame for their child's behavior, which occurred when the focus was

on neurological problems, was significantly reduced (Barkley et al., 2008). Also during

this time period, clinicians began to codify other neurologically-related phenomena (i.e.

"dyslexia," "language disorders," "learning disabilities," and "hyperactivity") (Barkley,

2006; Trott, 2006).

It was not until 1968 that the first diagnostic classification of symptoms similar to

the current conceptualization of ADHD was reported in the DSM-II (APA, 1968). In fact,

the DSM-II was the first diagnostic manual to identify and label children with

predominately excessive hyperactivity as "Hyperkinetic Reaction of Childhood"

(Barkley, 2006). The disorder was characterized by "overactivity, restlessness,

distractibility, and short attention span, especially in young children; the behavior usually

diminishes in adolescence" (DSM-II, 1968, p. 50).

In 1967, Menkes, Rowe, and Menkes were the first to report that ADHD may

persist from childhood into adulthood (De Quiros & Kinsbourne, 2001). However, it was

not until 1972, that Arnold, Strobl, and Weisenberg made the first adult diagnosis of

ADHD (De Quiros & Kinsbourne, 2001). This adult diagnosis may have also been aided

by the fact that in 1971, two important books were written by Wender (*Minimal Brain

Dysfunction in Children)* and Anderson (*America Pays the High Price of Minimal Brain

Dysfunction in America).* They reported that "minimal brain dysfunction" was found to

continue into adulthood, had genetic components, and was a precursor for other

psychiatric disorders (Anderson, 1972; Wender, 1971).

In the 1970s, the diagnostic symptoms of hyperkinesis changed to include the

symptoms of impulsivity, low frustration tolerance, short attention span, distractibility,

and aggressiveness (Barkley, 2006). Because of the belief that the disorder was outgrown

by puberty, the prognosis for this disorder was no longer considered poor, but fair

(Barkley, 2006). Also in the 1970s, the notion that poor or disrupted parenting caused

ADHD reemerged within some circles (Barkley, 2006).

However, it was not until 1980 that the American Psychiatric Association initially

used the diagnostic label of Attention Deficit Disorder in the DSM-III (APA, 1980). At

that time, this disorder included two subtypes: with or without hyperactivity (Barkley,

2006; Mehringer et al., 2002; Trott, 2006; Weinstein, Staffelbach, & Biaggio, 2000;

Wender et al., 2001). In 1987, the DSM-III-R changed the diagnosis to Attention

Deficit/Hyperactivity Disorder (ADHD) (APA, 1987; Barkley, 2006; Mehringer et al.,

2002; Weinstein et al., 2000; Wender et al., 2001). The DSM-IV (1994) added subtypes

to the ADHD diagnosis: a) Combined Type with symptoms of hyperactivity, inattention,

and impulsivity; b) Predominantly Inattentive Type with symptoms of inattention, and c)

Predominantly Hyperactive-Impulsive Type with symptoms of hyperactivity and

impulsivity but not inattention problems (Wender et al., 2001). To summarize, ADHD

has historically had the following names: minimal brain damage, minimal brain

dysfunction, hyperactive child syndrome, hyperkinesis, and minimal cerebral dysfunction

(Weinstein et al., 2000; Wender et al., 2001). Each of these terms had similar cognitive

and behavioral components, potentially including affective lability and poor attention

span, leading to poor interpersonal relationships, hostility, behavioral acting out,

defiance, learning difficulties, as well as central nervous system (CNS) dysfunction (i.e.

hyperactivity and impulsivity).

DSM-IV- TR Diagnostic Criteria for ADHD

The DSM-IV-TR's (APA, 2000) ADHD diagnosis is both empirically based and

highly tested (Barkley et al., 2008). The diagnostic criteria must be met prior to or at the

age of seven. Significant impairment must occur in at least two settings, with "Clear

evidence of clinically significant impairment in school, academic, or occupational

functioning" is also required (APA, 2000, p. 93).

In order to be diagnosed with ADHD, six of the nine symptoms listed must be met

for one or both subtypes, inattention and/or hyperactivity/impulsivity (APA, 2000). A

minimum of 12 symptoms is required for a diagnosis of the combined type. These

symptoms persist for at least six months, are maladaptive, and are developmentally

inappropriate. The diagnosis cannot be made if the symptoms occur only during the

course of pervasive developmental disorders, schizophrenia, or other psychotic disorders. ADHD also cannot be diagnosed if the symptoms are better described by another mental disorder such as a mood disorder, anxiety disorder, dissociative disorder, or a personality disorder. Adults and adolescents who no longer meet the full criteria are to be specified as "In Partial Remission". The diagnosis of ADHD Not Otherwise Specified is made when there are current symptoms requiring clinical attention, but ones that do not fulfill full criteria for any of the subtypes listed above.

The symptoms for the inattentive subtype include: failing to pay attention to details; making careless mistakes; appearing not to listen when spoken to; failing to follow through with directions and failing to finish work; organizing difficulties; avoiding tasks that require sustained attention; losing necessary items for activities; distractibility, and forgetting about daily activities (APA, 2000). The symptoms for the hyperactive-impulsive subtype include, being fidgety; leaving one's seat when being seated is required; feelings of restlessness that are both objective and subjective that are developmentally inappropriate for adolescents and adults; engaging in leisure activities loudly; moving excessively, and talking excessively.

When diagnosing ADHD, one must be cognizant that there are other psychiatric syndromes that can mimic ADHD. These other disorders include oppositional defiant disorder (ODD), conduct disorder (CD), posttraumatic stress disorder, mental retardation (MR), stereotypic movement disorder, personality change due to a general medical condition, sleep disorders, and any substance-related disorder (APA, 2000; Woodard, 2006). When considering whether or not ADHD symptoms are results of a general medical condition, one must rule out the following biological etiologies: chronic

migraines, thyroid disorders, seizures, asthma, Fragile X Syndrome, and anemia

(Woodard, 2006). The following psychosocial factors that can also mimic ADHD;

symptoms include: parental psychopathology, child abuse/neglect, normal developmental

variations, reactions to the death of a close family member or pet, parental divorce,

parental or child substance abuse, and family violence (Woodard, 2006).

Limitations of the DSM-IV-TR Diagnostic System

Although the DSM-IV-TR (2000) diagnostic criteria for ADHD is both

empirically based and highly tested, its reliability is limited because of a lack of

physiological, microbiological, and pathological markers, as well as a polythetic

diagnostic criterion (Barkley et al., 2008; Wender et al., 2001). This problem has resulted

in the use of criteria that can be either too rigid or too loose for making this diagnosis

(Wender et al., 2001). As a result, the lack of reliability, variability, and inconsistent

prevalence rates leave many individuals with ADHD undiagnosed and untreated (Wender

et al., 2001).

Using the DSM-IV-TR diagnostic criteria for adults is even more problematic.

For instance, the criteria for inattention and hyperactivity-impulsivity do not take into

consideration the possibility that when ADHD children become adults, many symptoms

may decrease with maturation (Barkley et al., 2008; Conners et al., 1999; Finkel, 1997).

Specifically, when one ages, the symptoms of hyperactivity and impulsivity decrease and

there are fewer objective signs of physical restlessness; however, there are increases in a

subjective sense of restlessness, mental activity, and fidgetiness (Ramsay & Rostain,

2008).

Also, the normative data for the DSM-IV (1994) was based on males, aged 4-17

and has no validation for the subtypes or residual category for adults (Barkley et al.,

2008; Conners et al., 1999). Furthermore, according to Barkley et al. (2008), this age

requirement has no empirical basis or rationale. There is evidence that symptoms

appearing prior to the age of 16 may constitute a sufficient age of onset to diagnose adults

with ADHD (Barkley et al., 2006). It has been found that there is no difference in

symptoms and impairment for individuals who experience symptoms prior to age seven

or later (Faraone, 2005).

These previously identified problems with the DSM-IV (1994) diagnostic criteria

for adult ADHD led Wender (1995) to develop the first adult ADHD criteria known as

the Utah Criteria. He proposed the following seven symptoms: hyperactivity, mood

liability, inattentiveness, irritability and hot temper, disorganization, impaired stress

tolerance, and impulsivity. The Utah Criteria were the first to utilize a retrospective

childhood diagnosis and third-party informants when available (Barkley et al., 2008). The

Utah Criteria also provided clarification of current adult symptoms, which are the

standards of today's practice (Barkley, Murphy, & Fischer, 2008). However, there are

some criticisms of these diagnostic criteria even though they have become widely

accepted (Conners et al., 1999). For instance, there is lack of empirical support for the

criteria; the criteria are outdated because they are based on the DSM-III (APA, 1980);

there is an absence of normative data or field tests to verify the cutoff scores specified,

and the symptoms described may overlap with ODD, personality disorders, or other

diagnoses (Conners et al., 1999).

Prevalence Rates of Adult ADHD

The difficulty in determining prevalence rates for adults is due to a scarcity of research in this area to date (Ramsay & Rostain, 2008). As mentioned previously, finding diagnostic criteria that are consistent in effectively defining the developmental deficits experienced by adults diagnosed with ADHD has been problematic. For example, longitudinal studies beginning in childhood differ in their use of the DSM criteria and the measures used. Some studies have found that 3.2 to 4.5% of adults meet the full diagnostic criteria, and 6.6% meet the criteria for a partial diagnosis (Barkley, 2006; Faraone, 2005; Mannuzza, Klein, Beister, Malloy, & Lapadulam, M., 1993; Murphy & Adler, 2004; Weiss, Hechtman, Milroy, & Perlman, 1985). This variability in the percentages may also be the result of diagnosing according to varying versions of the DSMs, the population studied, and the criteria utilized to make the diagnosis (e.g. loose or rigid). ADHD has recently been considered a chronic condition with symptoms continuing from childhood through adulthood in some individuals (Biederman et al., 2006; Conners et al., 1999; Ramsay & Rostain, 2008).

Biopsychosocial Correlates of ADHD

In general, multiple biopsychosocial factors including environmental, genetic, and other biological etiologies account for manifestations of ADHD. As mentioned previously, biological factors can lead to the development of syndromes similar to ADHD. For example, the diagnosis of ADHD in adulthood when there is no history of childhood or adolescent ADHD can occur as a result of a brain injury or from other organic causes (Gillig et al., 2005).

Genetic familial studies have found strong evidence that there is genetic heritability in the diagnosis of ADHD. Sprich et al. (2000) found that 6% of adoptive parents of ADHD children had ADHD, compared with 18% of biological parents of ADHD children and Levy, Hay, McStephen, Wood, and Waldman (1997) found that there was 0.75 to 0.91 heritability across familial relationships. There is also evidence that the D4 dopamine receptor gene (DRD4 7) and the amount of norepinephrine and epinephrine at this site are associated with the development of ADHD (Comings, 2001; Faraone et al., 1999). Sobanski (2006) stated that a more severe form of ADHD genetic loading occurred with the diagnoses of antisocial personality disorder in adulthood and conduct disorder in childhood.

Assessment of ADHD Symptoms

In order to make an appropriate diagnosis of ADHD, whether for children or adults, there are many factors that need to be taken into consideration. Consequently, it is often necessary to obtain the following: collateral information (if available) from teachers, parents, or significant others; structured interview assessing childhood development; past and current functioning at school (e.g. academic history, type of classes taken (i.e. regular education or special education); difficulties with reading or spelling; poor grades, below those normally expected by intelligence level; flunking or quitting school; past and current functioning at work (e.g. job performance, forced terminations, job changes, and promotions); socialization history (i.e. ability to maintain relationships); activities of daily living; and use of various measurements assessing one's level of functioning, symptoms and impairments (Adler & Cohen, 2004; Wender, 1998).

ADHD and Comorbid Psychiatric Disorders

Jacob et al. (2007) found that 83.7% of the individuals who were studied suffered from at least one comorbid Axis I disorder and approximately 60% had two or more Axis I disorders. Those diagnosed with the ADHD inattentive type were five times more likely to suffer from at least two or more externalizing disorders, and those diagnosed with ADHD combined type were more likely to suffer from an internalizing disorder (Miller et al., 2007).

Biederman et al. (2006) found that adults who had been followed from age 11 to 21 were significantly at higher risk for elevated lifetime prevalence of substance abuse and dependence, especially with alcohol, marijuana, cocaine, stimulants, opiates, and nicotine. Similarly, Miller et al. (2007) found that individuals diagnosed with ADHD were more likely to have a substance use disorder, anxiety disorder, mood disorder, and antisocial personality disorder than individuals without ADHD.

A more severe course of substance abuse and dependence tends to occur for individuals diagnosed with ADHD, because the substance use begins with an earlier onset, has a shorter period of time between the onset of drug abuse and dependence, and has a lower remission rate (Sobanski, 2006). During their lifetimes, 12-53% of adults diagnosed with ADHD will meet criteria for alcohol dependence or abuse and 8-35% may meet criteria for other substance abuse (e.g. 17.8% experience cannabis dependence and an additional 15.2% experience cannabis abuse) (Barkley et al., 2008; Jacob et al., 2007). Sobanski (2006) found that for individuals diagnosed with ADHD, 67% abuse marijuana, 23% abuse cocaine, and 18% abuse stimulants. Stimulants, cocaine, and nicotine may improve cognitive performance in the short-term and alcohol, marijuana,

and opiates may reduce emotional dysregulation and inner restlessness (Sobanski, 2006).

The risk for substance abuse and dependence are significantly higher for individuals

diagnosed with the comorbid ADHD and one or more of the following disorders: conduct

disorder, bipolar disorder, and antisocial personality disorder (Barkley et al., 2008). Other

risk factors include being involved in deviant peer groups; receiving medication

treatment later in the course of ADHD; using these substances to self-medicate, and

having a higher genetic risk both for ADHD and for substance use disorders (Barkley et

al., 2008).

Thus, there is a two-fold lifetime risk for the development of a substance use

disorder in adults having been diagnosed with ADHD since childhood (Biederman,

Wilens, Mick, Milberger, Spencer, & Faraone, 1995; Sobanski, 2006). In fact,

Biederman, Wilens, Mick, Faraone, and Spencer (1998) found that the association

between alcohol use disorders in ADHD individuals with subsequent drug abuse or

dependence was accounted for by ADHD alone, suggesting that ADHD may influence a

developmental course of polysubstance abuse chronicity from substance abuse to

substance dependence.

Interestingly, all longitudinal studies have found that the diagnosis of ADHD

precedes a diagnosis of a substance use disorder (Wilens & Biederman, 2006). As a

result, it makes sense that the development and severity of a substance use disorder may

be lessened when given appropriate and timely treatment (Bukstein, 2008). In fact, when

comparing children who had been treated with stimulant medication, Wilens, Faraone,

Biederman, & Gunawardene (2003) found that those who had been treated had fewer

occurrences of a substance use disorder in adolescence when compared with those who

had not received pharmacotherapy.

Many theories have been proposed to explain the relationship between ADHD and substance use disorders. Researchers have found that the diagnoses of ADHD, antisocial personality disorder, and alcohol abuse co-exist in families, suggesting a genetic link (Wender, Wolf, & Wasserstein, 2001). Another genetic link may be personality traits such as novelty seeking and impulsivity, which are found within these disorders (Chambers, Taylor, & Ptenza, 2003; Wilens & Biederman, 2006).

The high incidence rates of ADHD and co-morbid alcohol use may cause difficulty in appropriately treating individuals with these disorders. More specifically, the symptoms of these disorders tend to mirror one another. For instance, late onset ADHD may actually mirror the onset of a substance use disorder rather than ADHD (Faraone et al., 2007). Similarly, subthreshold symptoms may mirror non-specific risk factors of substance use disorders instead of ADHD. With such complications, it should not be surprising that the available literature supports the use of multimodal therapy (Sullivan & Rudnik-Levin, 2001).

In terms of mood disorders, 16-31% of individuals diagnosed with ADHD meet the diagnostic criteria for major depressive disorder; 19-37% meet the criteria for dysthymic disorder, and 9.5% meet the diagnostic criteria for bipolar disorder (14.7% males and 5.7% females) (Barkley et al., 2008; Sobanski, 2006). Over the course of their lifetimes, 30-50% adults diagnosed with ADHD experience at least one depressive episode; 40-60% meet criteria for an anxiety disorder, specifically; 10-45% suffer from generalized anxiety disorder; 6% suffer from posttraumatic stress disorder; 9-34% have an increased risk for social phobia, and less than 5% have difficulties with obsessive

compulsive disorder, somatization disorder, or panic attacks (Jacob et al., 2007; Matos, Saboya, Ayrao, & Segenreich, 2003; Sobanski, 2006). Three to 9% of adults diagnosed with ADHD also meet the diagnostic criteria for bulimia nervosa during their lifetimes (Jacob et al., 2007; Sobanski, 2006).

Chronic sleep difficulties for individuals diagnosed with ADHD include onset insomnia, sleep maintenance, poor sleep quality, and waking up earlier than desired (Sobanski, 2006). It is common for individuals with ADHD to become so engrossed in nighttime activities, such as watching television, surfing the internet, or more social pursuits, that they have difficulty "shutting down" their hyperarousal to go to sleep. Of course, ceasing stimulating activity to climb into bed in a dark room in such a state of arousal could be very aversive and, thus, could be avoided (Rosenfield, personal communication). As a result of the subsequent sleep deficit, these individuals have difficulty remaining alert and aroused throughout the day, which can lead to a circadian rhythm sleep disorder. Without medication, problems with increased nocturnal movements may also occur. As a result of their poor sleep quality, these individual are at heightened risk for intensified cognitive and behavioral symptoms of ADHD.

Axis II disorders personality disorders, can become apparent in childhood and adolescence and can extend into adult life; however, some of these disorders may decrease in intensity as one ages (APA, 2000). In general, one's personality influences how one makes sense of and adapts to one's environment (Ramsay & Rostain, 2008). When personality traits, which are "enduring patterns of perceiving, relating to, and thinking about the environment and oneself that are exhibited in a wide range of social and personal contexts," become inflexible, maladaptive, cause significant functional

impairment, and create subjective distress, the criteria for a personality disorder is met

(APA, 2000, p. 686). More specifically, the general diagnostic criteria for personality

disorders are "an enduring pattern of inner experience and behavior that deviates

markedly from the expectations of the individual's culture" as manifested by cognitions,

affect, interpersonal functioning, and impulse control that leads to clinically significant

distress or impairment in social, occupational, and other areas of functioning (APA, 2000,

p. 689). Given the fact that personality is complex and stable over time, personality

disorders require much more effort to change than other psychological disorders (Ramsay

& Rostain, 2008).

According to Nigg et al. (2002), ADHD may be associated with extremes of

personality traits. It is also possible that ADHD may alter personality, increasing the risk

for the development of personality disorders (Miller et al., 2007). The effects of long-

standing personality difficulties are that other psychological disorders become harder to

treat, intensifying the effects of these other disorders, including ADHD.

DSM-IV-TR Diagnostic Criteria for Personality Disorders

The DSM-IV-TR (2000) divides personality disorders into three clusters. Cluster

A disorders include schizoid personality disorder, schizotypal personality disorder, and

paranoid personality disorder. Cluster A personality disorders are characterized by odd or

eccentric behavior. Cluster B disorders include antisocial personality disorder, borderline

personality disorder, narcissistic personality disorder, and histrionic personality disorder.

Cluster B personality disorders are characterized by dramatic, erratic, or emotional

behaviors. Cluster C disorders include obsessive-compulsive personality disorder,

dependent personality disorder, and avoidant personality disorder. Cluster C personality

disorders are characterized by anxious or fearful behavior. The DSM-IV-TR lists the

general diagnostic criteria for the diagnosis of personality disorders. The diagnosis must

include at least two of the following: 1) cognition, 2) affect, 3) interpersonal functioning,

and 4) impulse control (p. 287). This pattern must be inflexible and pervasive across

settings, lead to clinically significant distress and impairment, be stable and long standing

(with the onset to at least early adulthood or adolescence), and not be better accounted for

by another mental disorder or be a result of substance use or a general medical condition.

The personality disorders that will be a focus of this paper are the ones typically

comorbid with the diagnosis of ADHD. These include antisocial personality disorder,

borderline personality disorder, narcissistic personality disorder, histrionic personality

disorder, avoidant personality disorder, obsessive-compulsive personality disorder,

depressive personality disorder, and passive-aggressive personality disorder (Burket et

al., 2005; Fischer, Barkley, Smallish, & Fletcher, 2002; May & Bos, 2000). Also, the

inclusion of passive-aggressive personality disorder and depressed personality disorder in

the hypotheses are due to the use of the SCID-II.

Cluster B personality disorders associated with the diagnosis of ADHD.

Individuals diagnosed with antisocial personality disorder have been found to lack

empathy, violate the rights of others, be irresponsible, and show little remorse for the

consequences of their actions (APA, 2000). In order to meet the DSM-IV-TR criteria for

antisocial personality disorder, at least three of the following criteria must be met: a)

disregard for social norms and laws that can result in being arrested, b) dishonesty (e.g.

using aliases, conning others purposefully), c) impulsivity, d) physical aggression and

irritability, e) lack of respect for the safety of others and oneself, f) irresponsibility, and

g) experiencing no guilt or regret for what one has done to others (APA, 2000). In order

to eligible for this diagnosis, the individual must have also met criteria for conduct

disorder prior to the age of 15 and must be at least 18 years of age.

Individuals diagnosed with borderline personality disorder have difficulty with

relationship stability and intensity, self-image, and affect (APA, 2000). In order to meet

DSM-IV-TR criteria, at least five of the following criteria must be met: a) frenzied efforts

to avoid abandonment, whether real or imagined, b) difficulties with interpersonal

relationships due to the individual's wavering from extremes of devaluation and

idealization, c) unstable self-image due to identity disturbance, d) self-damaging

impulsivity, e) consistent suicidal behavior or self-mutilation, f) reactive mood, causing

affect instability, g) persistent feelings of emptiness, h) extreme anger and/or problems

controlling anger, i) brief, stress-related dissociative symptoms or paranoia.

An accurate diagnosis of histrionic personality disorder requires at least five of

the following criteria: a) must be the center of attention, b) inappropriate sexually

seductive behavior when interacting with others, c) sudden changing of shallow

emotions, d) frequent attempts to gain attention through use of physical appearance, e)

vague and impressionistic speech, f) exaggerated emotional expression, g) engagement in

flirtatious behavior, and h) misperceives relationships to be more personal than they truly

are (APA, 2000).

Another cluster B personality disorder is narcissistic personality disorder, which

requires at least five of the following criteria: a) self-grandiosity without commensurate

accomplishments, b) preoccupation with fantasies of power, success, idealized love, and

beauty, c) belief in personal uniqueness, which can be understood only by high-power

individuals or institutions, d) sense of entitlement, e) exploitation of others, f) lack of

empathy, g) envies others or believes others are envious of him/her, and h) arrogance

(APA, 2000).

Cluster C personality disorders associated with the diagnosis of ADHD.

Individuals diagnosed with avoidant personality disorder must meet at least four

of the following criteria: a) avoidance of activities involving others due to fears of

negative evaluation, b) must perceive self as liked before socializing, c) avoidance of

intimate relationships due to fears of being negatively judged, d) preoccupation with

being criticized or rejected by others, e) hesitancy in developing new relationships due to

feelings of inadequacy, f) perceives self as incompetent, unappealing, or mediocre, and g)

avoidance of taking risks or engaging in new activities due to fear of embarrassment

(APA, 2000).

In order to meet criteria for dependent personality disorder, at least five of the

following criteria must be met: a) needs excessive advice and encouragement from others

when making daily decisions, b) needs others to direct one's life, c) fears disagreements

due to fears of losing support or approval, d) lacks self-confidence in judgment and

abilities, resulting in problems initiating tasks alone, e) seeks excessive nurturance,

resulting in performing jobs that one feels unpleasant about, f) fears being alone because

one believes that one cannot take care of oneself, g) urgently seeks another relationships

immediately when another one ends for care and support, and h) fears that one will have

to care for oneself (APA, 2000).

The DSM-IV-TR (2000) criteria for obsessive-compulsive personality disorder

requires at least four of the following: a) preoccupation with details, rules, and

organization, losing the point of the overall activity, b) perfectionism that interferes with

task completion, c) extreme devotion to work, resulting in loss of personal time and time

with family and friends, d) inflexibility with matters of morality, values, or ethics, e)

difficulty with discarding objects that no longer serve a purpose, f) tasks that have to be

delegated, which these individuals dislike, the delegate must submit his/her ideas to the

person suffering from OCD, and g) inflexibility and stubbornness (APA, 2000).

Prior DSM personality disorder labels associated with the diagnosis of ADHD.

Depressive personality disorder was described in the DSM-IV (1994) (Kelly,

2008a). It is currently in the appendix of the DSM-IV-TR and is mainly there for research

purposes (APA, 2000). This diagnosis requires a pervasive pattern of depressive

cognitions and behaviors that began in early adulthood and appears in a variety of

contexts (Kelly, 2008a). At least five of the following symptoms must have been met to

acquire this diagnosis: a) consistent mood of misery, unhappiness, and gloominess, b)

central beliefs about the self as inadequate, useless, with feelings of low self-esteem, c)

responds to self as critical, derogatory, and fault finding, d) prone to brooding and

worrying, e) negative, critical, and judgmental towards others, f) pessimistic, and g)

frequent feelings of guilt or remorse (http://www.ptypes.com/depressivepd.html, 2008).

This disorder does not occur only during a major depressive episode and must not be

better accounted for by dsythymic disorder (Kelly, 2008a).

Self-defeating personality disorder or masochistic personality disorder was

discussed in the appendix of the DSM-III-R for research purposes (APA, 1987). This

disorder is described as a pervasive pattern of self-defeating behavior that begins by early

adulthood and appears across a variety of contexts. "The person may often avoid or

undermine pleasurable experiences, be drawn to situations or relationships in which he or she will suffer, and prevent others from helping him". At least five of the following symptoms must be met to warrant the diagnosis: a) instead of choosing better options that are available, he/she chooses people and situations that lead to dissatisfaction, disappointment, or mistreatment, b) rejects or renders ineffective help from others, c) after a positive personal event, he/she responds with depression, guilt, or a behavior that produces pain, d) after provoking anger or rejection from others, he/she feels hurt, defeated, or humiliated, e) does not seek opportunities for happiness or is not open to stating he/she is enjoying him/herself, f) fails to accomplish tasks crucial to his/her personal objectives despite demonstrated ability to do so, g) is uninterested in or rejects people who consistently treatment him/her well, and h) engages in excessive self-sacrifice that is unsolicited by the intended recipients of the sacrifice. These behaviors do not manifest themselves only in response to or in anticipation of being physically, sexually, or psychologically abused, or when the person is depressed.

Passive-aggressive personality disorder was first described in the DSM-IV (1994) for research purposes (Kelly, 2008b). To receive this diagnosis, there must be a pervasive pattern of negativistic attitudes and passive resistance to demands for adequate performance, beginning by early adulthood and appearing in a variety of contexts. At least four of the following symptoms must be met to warrant this diagnosis: a) passively resists fulfilling routine social and occupational tasks, b) complains of being misunderstood and unappreciated by others, c) is brooding and confrontational, d) unfairly disparages and disrespects authority, e) is envious and resentful towards those that appear more fortunate, f) exaggerates and continually complains of his/her own

misfortune, and g) alternates between hostile defiance and remorse. The disorder does not

occur only during a major depressive episode and is not better accounted for by

dysthymic disorder.

ADHD and Comorbid Personality Disorders

There are studies that have found small to large relationships between adult

ADHD and personality disorders. These discrepancies may result from the standard for

the DSM diagnostic criteria used for adult ADHD (whether loose or rigid), the population

studied, and the measures used. As mentioned previously, personality disorders can begin

in adolescence, whether or not one is diagnosed with ADHD or another Axis I disorder.

For example, when studying hospitalized adolescent females with ADHD and without

ADHD, Burket et al. (2005) found that both groups had comorbid Axis II personality

disorders in the following order: passive-aggressive personality disorder, histrionic

personality disorder, and borderline personality disorder. However, the ADHD group was

more likely to have comorbidity with paranoid, borderline, histrionic, passive-aggressive,

and dependent personality disorders. In fact, all subjects in the ADHD group met criteria

for at least one personality disorder.

For adults diagnosed with ADHD, Jacob et al. (2007) found that the lifetime

comorbidity for having at least one personality disorder was 78.5%, and 45% were found

to suffer from multiple personality disorders. More specifically, they found that the

frequencies of Axis II diagnoses were: paranoid (12%, cluster A), histrionic (35.2%,

cluster B), with only 5.7% receiving the diagnosis of antisocial personality disorder, and

avoidant personality disorder (18.3%, cluster C). However, Barkley et al. (2008) found

that 7-44% of their subjects met the diagnostic criteria for antisocial personality disorder and many of those who did not meet full criteria had antisocial personality traits.

In another study, May and Bos (2000) placed adult participants in one of four categories: ADHD; ADHD and oppositional defiant disorder; ADHD and a comorbid disorder; and ADHD, oppositional defiant disorder, and a comorbid disorder. They found that each of these groups differed significantly on seven of thirteen personality scales on the Millon Clinical Multiaxial Inventory-II (MCMI-II). The ADHD only group had a significant elevation on the histrionic scale; the ADHD and comorbid group had significant elevations on the avoidant and dependent personality scales; the ADHD and oppositional defiant disorder group had significant elevations on the histrionic, narcissistic, aggressive-sadistic, and negativistic personality scales; the ADHD, oppositional defiant disorder, and comorbid disorder group had significant elevations on the avoidant, histrionic, narcissistic, aggressive-sadistic, negativistic, and self-defeating personality scales. The ADHD, oppositional defiant disorder, and comorbid group was found to be associated with the diagnosis of antisocial personality disorder. Overall, the Axis II disorders associated with adult ADHD were histrionic, avoidant, dependent, narcissistic, aggressive-sadistic, negativistic, and self-defeating.

Fischer, Barkley, Smallish, and Fletcher (2002) assessed adults diagnosed with ADHD for current personality disorders using the *Structured Clinical Interview for the DSM-IV-TR Part II* (SCID-II). The research groups included those who were hyperactive with current ADHD (H+ADHD); hyperactive without current ADHD (H-ADHD) and a community control group. It was found that adults in the H+ADHD cohort were twice as likely to be diagnosed with a personality disorder, especially antisocial personality

disorder, passive-aggressive personality disorder, borderline personality disorder, obsessive-compulsive personality disorder, paranoid personality disorder, dependent personality disorder, and avoidant personality disorder. Comorbid personality disorders occurred in 66.7% of the H+ADHD adults, which was almost twice the frequency of that of the H-ADHD adults and 1.5 times more frequently than those in the community sample. More than 84% of the H+ADHD group had at least one other psychiatric disorder, which was twice as many as those in the H-ADHD group and four times as many as those in the community control group.

However, Miller et al. (2007) did not find that the two subtypes of ADHD, inattentive type and combined type, differed on Cluster A, Cluster B, or Cluster C disorders. Instead, ADHD in general was associated with an increased likelihood of having a Cluster B personality disorders (i.e. borderline personality disorder, antisocial personality disorder, histrionic personality disorder, and narcissistic personality disorder). In fact, both genders were found to be more likely to have two or more cluster B disorders. Miller et al. (2007) concluded "ADHD influenced impairment above and beyond that accounted for by antisocial personality disorder, borderline personality disorder, generalized anxiety disorder, or major depressive disorder" (p. 525).

Sobanski (2006) asserted that there is evidence that when there is diagnosis of ADHD in childhood, there is a higher risk factor for developing borderline personality disorder in adulthood. This relationship may be phenomenological due to the overlapping diagnostic criteria (Miller, Miller, Newcorn, & Halperin, 2008; Philipsen et al., 2008). It may also be that these disorders co-occur because of common neurobiological and/or risk

factors (e.g. hypothalamic-pituitary-adrenal axis deysregulation and/or adverse early experiences with fear or anger).

Accuracy of Clinical Measures

Murphy and Schachar (2000) reported that the subjective and informant rating measures for behaviors and symptoms of ADHD have been found to have good correlations, suggesting that individuals' ratings of their own ADHD symptoms are valid and legitimate. However, differences have been found between self-report rating scale scores and clinician rating scale scores (Demyttenaere & De Fruyt, 2003; Lambert et al., 1986). For example, the accuracy of ADHD subjects' recall can be affected by the phrasing of questions (i.e. specific questions provide more accurate responses than those that are more general) (Murphy & Schachar, 2000). Also, individuals completing self-report measures may have better awareness of their subjective feelings than do clinicians measuring similar symptoms; however, when subjects have no awareness (limited insight/poor reality testing) of their symptoms, clinicians may have greater expertise and clinical training to identify them (Demyttenaere & De Fruyt, 2003; Lambert et al., 1986). Thus the tandem use both of self-rating and of observer-rating scales can provide more valid and reliable data, leading to a clearer and more complete clinical picture.

ADHD Measure

After 25 years of clinical interviews with children, adolescents, and adults diagnosed with Attention Deficit Disorder (ADD)/ADHD, Brown (1996) developed the examiner administered Brown Attention Deficit Disorder Scale – Adult (BADDS – Adult) based on the DSM-IV (1994) criteria (Harrison, 2004; http://www.drthomasebrown.com/brown_model/index.html, n.d.). The symptoms

reported were compared with non-ADHD controls, allowing the BADDS to identify

adults suffering from inattentive symptoms of ADHD (Harrison, 2004;

http://www.drthomasebrown.com/brown_model/index.html, n.d.). Cutoff scores above 50

indicate probable, but not certain ADHD, and cutoff scores above 55 indicate that there is

a clinically significant risk for ADHD (Harrison, 2004).

Brown's model is based on the idea that as the brain matures, particularly in the

frontal cortex, cognitive functions, especially the executive functions, become more

complex (http://www.drthomasebrown.com/brown_model/index.html, n.d.). Brown

posits that executive functions are delineated along six clusters: Activation, Focus, Effort,

Emotion, Memory, and Action. It is believed that these clusters work together and are

sometimes used quickly and without conscious effort. Based on a cognitive deficit model,

the BADDS was designed to assess specific cognitive deficits believed to be associated

with ADHD. Because individuals diagnosed with ADD/ADHD have been found to have

excessive difficulty with procrastination, the Activation cluster focuses on organizing,

prioritizing, estimating time, and getting started on tasks. Individuals diagnosed with

ADD/ADHD have been found to have significant difficulty with distraction from external

and internal stimuli. Thus the Focus cluster measures the ability to focus, to maintain

focus, and to change focus from one task to another. Because individuals diagnosed with

ADD/ADHD have been found to have significant difficulty processing information, the

Effort cluster was created to assess the extent to which the individual is regulating

alertness, is maintaining effort, and is paying attention to his or her processing speed.

Individuals with ADHD also have difficulty keeping their emotions in perspective and

thus the Emotion cluster focuses on managing frustration and adjusting emotions as

necessary, because individuals with ADD/ADHD may have excessive difficulty with regulating emotions (e.g. frustration, disappointment, worry, anger, desire, etc.). The Memory cluster focuses on accessing and using memory because individuals diagnosed with ADHD have difficulty accessing learned information from their memories when it is needed. The Action cluster focuses on monitoring and modifying needed actions because individuals with ADD/ADHD may have significant difficulty in this area even without hyperactive behavior.

Limitations: The BADDS subscales were not developed using empirical psychometric standards, because the symptom clusters were developed, based only on face validity and not from empirical methods or research-based literature (Harrison, 2004; Mehringer et al., 2002). Additionally, the instrument was not cross-validated and was standardized using small samples (Harrison, 2004; Mehringer et al., 2002). Also, the BADDS manual does not state how the clusters differentiate ADHD from other disorders (Harrison, 2004).

Depression Measures

According to Demyttenaere & De Fruyt (2003), depression scales were initially developed to assess the treatment effects of antidepressant medications in the 1950s. The scales that have been developed are classified either as self-report measures or as observer rating scales. Two of the most widely used measures are the Beck Depression Inventory – II (BDI-II), a self-report measure, and the Hamilton Depression Measure (HAM-D), structured clinical interview (Beck, Steer, & Brown, 1996; Groth-Marnat, 1997; Hamilton, 1967). In 1961, the original BDI was developed, based on Beck's observations of individuals engaged in psychoanalytic psychotherapy (Beck, Ward,

Mendelson, Mock, & Erbaugh, 1961). Dr. Beck's observations of depressed patients'

attitudes and symptoms were also used in developing the scale (Beck et al., 1961).

The current Beck Depression Inventory – II (BDI-II) (Beck, Steer, & Brown,

1996) is a self-report measure used to assess cognitions associated with depression. The

instrument is currently used for both psychiatric and nonclinical populations. Because the

BDI-II is related to the DSM-IV, it can differentiate responses to treatment via its ability

to distinguish between cognitive-affective and somatic-vegetative symptomatology

(Dozois, Dobson, & Ahnberg, 1998). For example, the BDI-II assesses irritability,

sleeping problems, feelings of guilt, loss of appetite, and feelings of failure, all of which

can lead to suicidal thoughts (Groth-Marnat, 1997).

Limitations: The BDI-II has been found to overestimate the severity of depressive

symptoms in patients diagnosed with somatic problems (Brown-DeGange, McGlone, &

Santor, 1998). Another limitation relates to the instrument's test-retest reliability, which

will be discussed later (Richter et al., 1997). When compared with the HAM-D scores,

the BDI symptoms were less likely to be endorsed by individuals with lower levels of

education (Sayer et al., 1993).

The HAM-D (Hamilton, 1967) is a clinician-administered scale. It assesses the

severity of current depressive symptoms such as feelings of guilt, helplessness, and

somatic symptoms (Hamilton, 1967). The severity of the depression is indicated by

increases in the score. A score of less than 8 indicates remission of symptoms and scores

ranging from 8-14 indicate partial remission (Frank et al., 1991; Tedlow et al., 1998).

Limitations: It is reported that the HAM-D is biased toward somatic and

behavioral symptoms because approximately 50% of the total score is accounted for by

these symptoms (Möller, 2000). The total scores also do not differentiate between the specific types of depressive symptoms (e.g. cognitive vs. somatic symptoms; agitated vs. psychoretardation symptoms) (Demyttenaere & De Fruyt, 2003). Moreover, it has been found that only eight items are correlated with the diagnosis of major depression; the other items are related to generalized anxiety (Riskind, Beck, Brown, & Steer, 1987). As a result, those experiencing more generalized anxiety symptoms will score higher on this measure than those experiencing more depressive symptoms (Riskind et al., 1987). The items are also rated either on a 3-point Likert scale or on a 5-point Likert scale, which causes those items rated on a 5-point scale to be more heavily weighted (Demyttenaere & De Fruyt, 2003). Overall, these limitations affect the reliability of the scores of the diagnostic categories and the treatment outcome (Demyttenaere & De Fruyt, 2003).

There are significant differences between the BDI-II and the HAM-D scales. They differ on the symptoms that are measured, the number of questions, and the criteria that were used to develop the scoring (Demyttenaere & De Fruyt, 2003). The HAM-D and the BDI-II are supposed to measure a range of symptoms for depression; however, they tap different areas of depression. The HAM-D does not measure atypical symptoms (i.e. hypersomnia, weight or appetite increase), although it focuses on somatic, vegetative, and anxiety-irritability symptoms, as well as on the behavioral manifestations of depression (Demyttenaere & De Fruyt, 2003; Enns, Larsen, & Cox, 2000). The BDI-II does not measure psychomotor retardation or anxiety symptoms, but rather focuses on the psychological and subjective experiences of depression including pessimism, cognitive symptoms, irritability somatic preoccupations, and punitive beliefs (Demyttenaere & De Fruyt, 2003; Enns et al., 2000; Lambert et al., 1986), as well as behavioral manifestations

of depression. Additionally, each of these measures assesses symptoms that are not included in the DSM criteria for major depressive disorder (Demyttenaere & De Fruyt, 2003).

It is possible that when depression starts to dissipate, the HAM-D may reflect changes in vegetative symptoms, which can be the first sign of treatment improvement (Lambert et al., 1986). After those changes occur, the BDI-II can reflect further treatment changes in cognition and affect (Lambert et al., 1986).Therefore, the tandem use of both the BDI-II and the HAM-D can provide different, relevant information that can be used when performing an assessment because there may be "important differences in the detection of change in therapeutic studies when scales such as the HAM-D and the BDI (which are not highly correlated) are used" (Enns et al., 2000, p. 39).

Hopelessness Measure

The Beck Hopelessness Scale (BHS) (Beck & Steer, 1989) was developed during an era when it was recognized that negative thoughts and emotions could contribute to the development and maintenance of both psychological and of physical illness (Steed, 2001). The purpose of this measure is to tap the construct of hopelessness (Beck & Steer, 1989; Steed, 2001). It is a self-report measure that assesses one's level of pessimism or optimism towards the future (Bhar, Brown, & Beck, 2008).

The study of hopelessness is important because it is a precursor to and predictor of suicide (Beck, Brown, Berchick, Stewart, & Steer, 1990). The items of the BHS were selected from pessimistic statements made by psychiatric patients that appeared hopeless (Steed, 2001). Dowd (1992) reported that the BHS was well-constructed and was a valid instrument, with adequate reliability.

Limitations: The BDI predicts suicidal ideation and behavior better than the BHS (Aiken, 2002). Moreover, Young, Halper, Clark, Scheftner, and Fawcett (1992) reported that the BHS was not as effective in determining lower levels of hopelessness.

Anxiety Measures

The Beck Anxiety Inventory (BAI) (Beck & Steer, 1990) is a self-report measure that assesses physiological (somatic), behavioral, and cognitive symptoms of anxiety. It was developed to differentiate anxiety from depression. The BAI items represent subjective, neurophysiological, autonomic, and panic symptoms of self-reported anxiety (Beck & Steer, 1991). Symptoms identified must occur during the previous week, including the day of the test (Beck & Steer, 1990). This measure is not designed to make diagnoses.

Limitations: As with other self-report questionnaires, the BAI is vulnerable to each person's differing subjective interpretations of the items (Beck, Butler, Brown, Dahlsgaard et al., 2001). The overall score can also be affected not only by one's affective state while completing the measure, but also by social desirability. Leyfer, Ruberg, and Woodruff-Borden (2006) found that the BAI had acceptable sensitivity; however, individuals without an anxiety disorder scored in the presence range of pathological anxiety.

Cox, Cohen, Direnfield, & Swinson (1996) also consider the BAI to be a measure of symptoms of panic rather than a measure of anxiety. They reported that the normative sample used to develop the BAI consisted of 40% of individuals with panic disorder or agoraphobia with panic attacks, and 19% of individuals with mood or adjustment disorders, meaning that there was an over-representation of panic disorder patients,

making the measure more highly panic symptom-oriented. They note that the majority of

individuals presenting with anxiety symptoms have panic attacks.

Another anxiety inventory, the Hamilton Anxiety Measure (HAM-A) (Hamilton,

1959) is a clinician-administered scale that assesses the current level of severity of

anxiety symptoms. The items that are measured include anxious and depressed mood,

fears, insomnia, cognitive difficulties, autonomic symptoms, somatic muscular and

sensory symptoms, cardiovascular symptoms, gastrointestinal symptoms, genitourinary

symptoms, and tension.

Limitations: Rehm & O'Hara, (1984) claimed that HAM-A items reflecting

agitation, loss of weight, and gastrointestinal symptoms detract from the measure's

internal consistency and concurrent validity. In terms of assessing the effectiveness of

treatment outcomes, this measure cannot distinguish between the effects of anxiolytics

and antidepressants (Maier, Buller, Philipp, & Heuser, 1988). Also, the somatic side

effects of the medications are strongly related to the somatic anxiety subscale (Maier et

al., 1988). Thus, the ability of the HAM-A to distinguish the effectiveness of anxiolytic

treatment is limited.

Personality Measures

Cognitive theory postulates that dysfunctional beliefs characterize and perpetuate

personality disorder symptoms (Atrnz, Dreessen, Schouten, & Weertman, 2004; Beck,

Butler, Brown, & Dahlsgaard, 2000; Trull, Goodwin, Schopp, Hillenbrand, & Schuster,

1993). This theory proposes that each personality disorder contains a specific set of

beliefs or schemas, with the possible exception of borderline personality disorder and

schizoid personality disorder because individuals with these disorders may not display a

typical characteristic set of beliefs and behaviors (Atrnz et al., 2004; Beck, Butler, Brown, & Dahlsgaard, 2000; Nelson-Gray, Huprich, Kissling, & Ketchum, 2004; Trull et al., 1993).

The Personality Beliefs Questionnaire (PBQ) (Beck & Beck, 1991) is a self-report measure that assesses potentially maladaptive beliefs that are often associated with specific Axis II personality disorders. Initially, the items of the PBQ were published as a list of schemas for each of the personality disorders (Beck, Freeman, & Associates, 1990). Items of the PBQ were developed from theory and clinical work (Butler, Beck, & Cohen, 2007). More specifically, the conceptualizations of the cognitions of each personality disorder's behaviors, dysfunctional assumptions, and beliefs were created (Butler, Beck, & Cohen, 2007).

The items are categorized into nine scales that are consistent with the DSM-IV personality disorders (Bhar et al., 2008; Trull et al., 1993). Clinical interviews have found that the more highly a belief set is endorsed, the more likely the individual may meet the criteria for the corresponding disorder (Beck et al., 2001). This measure can be used to complement the clinical interview by assessing the client's dysfunctional beliefs (Beck & Beck, 1991).

Limitations: The PBQ may be vulnerable to response bias because all items are scored in the same direction and, hence, it is possible that higher scores may result from a person's tendency to agree or disagree with any item regardless of content (Trull et al., 1993). This measure also does not contain subscales for schizotypal personality disorder and borderline personality disorder, which limits its use for research and clinical purposes (Trull et al., 1993).

The Structured Clinical Interview for DSM-IV Part II (SCID-II) (First, Gibbon, Spitzer, Williams, & Benjamin, 1997) is administered in two stages. Initially, it is administered as a self-report measure. If the patient meets criteria for PDs, the clinician can then follow up the self-report responses with a semi-structured diagnostic interview. The SCID-II is based on the 10 DSM-IV diagnostic criteria for Axis II psychiatric disorders. It also assesses depressive personality disorder and passive-aggressive personality disorder. This measure can be used to make Axis II diagnoses, either categorically (present or absent) or dimensionally (by noting the number of personality disorder criteria for each diagnosis coded "3") (First et al., 1997). Because the symptoms for each Axis II disorder are grouped together, it allows the interviewer to explore any area in which the patient answers "yes" to a minimum number of questions in any category (Jain, 2003). The presence of each personality disorder is determined as the interview progresses (First et al., 1997).

Limitations: Due to the problems of assessment and/or conceptualization found within the SCID-II, many of the diagnostic criteria detract from the overall internal consistency and diagnostic efficiency of the measure (Rief, Nanke, Emmerich, Bender, & Zech, 2004). The SCID-II also has problems with discriminating individuals with specific personality disorders from those without any disorders (Rief, Nanke, Emmerich, Bender, & Zech, 2004). A person's responses to the questions contained within this measure may be significantly affected by his/her insight and/or motivation, limiting an accurate diagnosis being made (Westen, 1997).

Chapter Three: Hypotheses e

This study included 4 research hypotheses. The following are summaries of the hypotheses, the rationale for each individual hypothesis, and the respective operational definitions.

Hypothesis 1

There will be a significant reduction in symptoms of ADHD and mood disorders post-treatment.

Rationale.

CBT has been found to be associated with improvements on measures of ADHD symptoms, mood, and anxiety symptoms, as well as on overall levels of functioning in various types of research studies (e.g. open clinical, nonrandomized control, and randomized control). According to Ramsay (2007), the combination of CBT and pharmacotherapy is currently the treatment of choice for adult ADHD.

Formula.

There will be a significant difference between pre- and post-treatment ADHD scores as measured by the BADDS subscales. There will be a significant difference between pre- and post-treatment mood scores as measured by the BDI-II, BAI, BHS, HAM-A, and HAM-D.

Hypothesis 2

There will be a positive association between symptoms of ADHD pre- and post-treatment and disordered thinking.

Rationale.

There is a lack of research in this area (i.e. the relationship between ADHD symptoms and PBQ subscales). It seems that as the number of maladaptive beliefs increase, the more likely it is that the ADHD severity will increase.

Formula.

The total score on the BADDS pre- and post-treatment will positively correlate with pre-treatment PBQ and SCID-II scores.

Hypothesis 3

The presence of personality disordered thinking will interfere with changes in ADHD symptoms.

Rationale.

There is also a lack of research in this area (i.e. the relationship between personality disorder symptoms and ADHD symptoms). However, due to the durability of beliefs of individuals diagnosed with personality disorders, individuals with personality disorders will experience poorer treatment outcome on measures of maladaptive beliefs.

Formula.

Severity of PDs as measured by high scores on the pre-treatment SCID-II and the PBQ will predict poorer outcome on the BADDS subscales.

Hypothesis 4

The presence of a personality disorder will interfere with reductions in symptoms of mood disorders.

Rationale.

Due to the difficulty in changing the cognitions of individuals with personality disorders, the depression, anxiety, and hopelessness scores will remain higher than those without personality disorders after treatment. Newton-Howes, Tyrer, and Johnson (2006) reported that a presence of a personality disorder doubles the probability of a poor outcome in depression. Kuyken et al. (2001) found that for depressed outpatients, avoidant and paranoid beliefs were associated with treatment response, which "is consistent with some recent findings that circumscribed maladaptive beliefs affect change over the course of cognitive therapy" (p. 5). Tang and DeRubeis (1999) stated that significant changes in one's maladaptive beliefs lead to changes in one's depression level.

Formula.

Severity of PDs as measured by high scores on the SCID-II and the PBQ will negatively correlate with changes in scores on the BDI-II, BAI, BHS, HAM-A, and HAM-D.

Chapter Four: Methods

Subjects

  This project is based on a prior study performed by Rostain and Ramsay (2006). The original participants sought outpatient treatment at the Adult ADHD Treatment and Research Program of the University Of Pennsylvania Department Of Psychiatry, located in Philadelphia, Pennsylvania. The initial participant pool consisted of 108 consecutive outpatients calling for appointments. Of these individuals, 96 were assessed and 86 met the criteria for the diagnosis of ADHD. Of these 86 individuals, 74 participated in the study; however, 10 subjects dropped out before treatment completion. 45 subjects participated in the combined treatment of CBT and medication; however, 2 subjects dropped out. Thus, the total retention rate was 86%, with a total subject pool of 43 individuals.

  Upon entering the program, each new participant underwent a standard diagnostic evaluation, which was administered by a psychiatrist, a psychologist, and a research assistant. Included in this evaluation were a brief neuropsychological battery, and an extensive psychological assessment, including the following measures: the Beck Depression Inventory – II, the Beck Anxiety Inventory, the Beck Hopelessness Scale, the Hamilton Anxiety Measure, the Hamilton Depression Measure, the Personality Beliefs Questionnaire, the Clinical Global Impression for ADHD (CGI-A), the Clinical Global Impression (CGI), the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV-Axis I disorders (SCID-I), and the Structured Clinical Interview for DSM-IV Part II (SCID-II). Upon completing the evaluation, the assessment data were compiled into a diagnostic formulation.

Inclusion Criteria

To be eligible for the study, participants were required to be at least 18 years of age and meet the DSM-IV diagnostic criteria for ADHD. Subjects were also required to fulfill the DSM-IV diagnostic criteria for ADHD. A total raw score of at least 40 (i.e. "ADD probable but not certain" range) was required on the BADDS. Corroborative clinical data included a clinical interview confirming childhood symptoms and a positive finding on the childhood symptom checklists by the participant and corroborating individuals (if possible). Minimum educational requirements included a high school degree or a GED. Additionally, participants needed either to be currently enrolled in college or graduate school, or be employed, or be a stay at home parent.

Exclusion Criteria

Subjects were excluded if they met the diagnosis of substance dependence, as indicated by the SCID-I, requiring specialized treatment. Other exclusion criteria included actively suicidal, homicidal, or psychotic symptoms. Participants requiring specialized or emergent clinical attention due to severe impairment from one or more psychiatric diagnoses were also excluded from the study. However, comorbid diagnoses were acceptable if the symptoms were amenable to the combined treatment of medication and CBT provided in the study.

Research Design

The current study is an archival study utilizing data gathered by Rostain and Ramsay (2006).

Clinical Measures

  Brown Attention Deficit Disorder Scale – Adult Version

  The BADDS (Brown, 1996) is an examiner-administered instrument that

measures various ADHD symptoms in adults, based on the nine symptoms of the DSM-

IV (1994) criteria for inattention. The BADDS assesses one's ability to sustain attention,

get started on tasks, initiate and sustain attention, maintain effort necessary to complete

tasks, regulate moods, and recall information encountered in daily life (Ramsay &

Rostain, 2008). Brown (1996) developed five subscales on components of executive

functions: Activation, Attention, Effort, Affect, and Memory. This measure can identify

changes in symptoms as a result of treatment interventions.

  This measure contains 40 items rating the frequency of the subjects' symptoms

during the week on a 4-point Likert scale: 0 "none of the time," 1 "a few times during the

week," 2 "sometimes during the week," and 3 "frequently during the week" (Collett,

Ohan, & Myers, 2003). Example questions include: a) "difficulty getting started" and

"feel[ing] overwhelmed" on the Organizing and Activating to Work subscale; b) "'spaces

out' when reading" and "becomes distracted easily" on the Sustaining Attention and

Concentration subscale; c) "needs extra time" and "is criticized as lazy" on the Sustaining

Energy and Effort subscale; d) "is excessively impatient" and "is sensitive to criticism"

on the Managing Affective Interference subscale; and e) "I am excessively forgetful" and

"has difficulty memorizing" on the Utilizing Working Memory and Accessing Recall

subscale (Brown, 1996). A score of 50 is the recommended cut off, suggesting further

screening is needed (Brown, 1996); however, Rostain and Ramsay (2006) used a cut off

of 40 so that individuals who may be underreporting their symptoms, despite clear

clinical evidence that symptoms may be present, would be included in the study (Rostain
& Ramsay, 2006). This cut off score was found to result in false negatives 4% of the time
and false positives 6% of the time across all age groups (Brown, 1996).

BADDS reliability.

Brown (1996) found that 35 items have a high level of internal consistency ($r \leq$
0.50), with correlations ranging from 0.70 to 0.80 on 14 of the items. The Chronbach
Coefficient Alpha equaled 0.96. Also, 32 items on the subscales correlated 0.50 or better
with the total score for their cluster and 22 items on the subscales correlated 0.60 or
better. The cluster alphas were found to range from 0.79 to 0.92.

Beck Depression Inventory-II

The Beck Depression Inventory-II (BDI-II) (Beck, Steer, & Brown, 1996) is a 21-
item questionnaire used to assess cognitions associated with depression for both
psychiatric patients and for non-psychiatric individuals and has the ability to differentiate
these two populations. It takes about 5-10 minutes to complete (Groth-Marnat, 1997); a
fifth to sixth grade reading level is required.

The BDI-II assesses one's sense of failure, irritability, sleeping problems, feelings
of guilt, and appetite loss (Groth-Marnat, 1997). The content of the questions were
derived from a clinician consensus of depressed patients' symptoms, as well as from six
of the nine DSM-III (1980) categories for the diagnosis of depression. The individual is
asked to answer each of the items with a time frame of the previous two weeks. The items
are scored on a 4-point Likert scale. Examples of questions include a rating of 0 for "I do
not feel sad"; 1 for "I feel sad"; 2 for "I am sad all the time and I can't snap out of it"; 3
for "I am so sad or unhappy that I can't stand it", and a range of 0 for "I don't have any

thoughts of killing myself"; 1 for "I have thoughts of killing myself, but I would not carry

them out"; 2 for "I would like to kill myself", and 3 for "I would kill myself if I had the

chance."

The total range of scores is 0-63 with minimal depression scores ranging from 5-

9; mild to moderate depression scores ranging from 10-18; moderate to severe depression

scores ranging from 19-29, and severe depression scores ranging from 30-63 (Groth-

Marnat, 1997). Scores below four indicate possible denial of depression, faking good and

unusual scores endorsed by non-depressed individuals. Scores above 40 are significantly

higher than the scores for severely depressed persons, indicating possible exaggeration of

depression or possibly histrionic or borderline personality disorder, or may actually

indicate significant levels of depression and nothing else.

BDI-II reliability.

Beck et al. (1996) reported that the BDI-II has a high internal consistency for

college students (0.93) and for outpatients (0.92). Doszois, Dobson, and Ahnberg (1998)

found an internal consistency of 0.91 for the BDI-II, which is similar to the score on the

original BDI. For adolescents, the internal consistency was found to be 0.92 with an

average inter-item correlation of 0.35 (Osman, Barrios, Gutierrez, Williams, & Bailey,

2008). Thus, the internal consistency scores range from 0.91 to 0.93.

BDI-II validity.

The convergent validity between the BDI and the BDI-II was found to be 0.93

(Dozois et al., 1998). Palmer and Binks (2008) found that the for incarcerated males aged

18-21, convergent validity was established due to the higher BDI-II scores for individuals

with histories of suicidal behavior. They also found that convergent validity was further

supported by the significant correlation of the BDI-II with the Beck Hopelessness Scale

($r = 0.55$).

Beck Anxiety Inventory

The Beck Anxiety Inventory (BAI) (Beck & Steer, 1990) is a 21-item self-report

measure that assesses physiological and cognitive symptoms of anxiety. It was developed

to differentiate anxiety from depression. The items assess somatic, behavioral, and

cognitive manifestations of anxiety. The items endorsed must refer to the previous week,

including the day one completes the measure. The measure takes approximately 5-10

minutes to complete.

The symptoms of anxiety are measured on a 4-point Likert scale from 0 "not at

all" to 3 "severely – it bothered me a lot". Examples of some of the symptoms that are

assessed include: numbness or tingling, feeling hot, wobbliness in legs, unsteadiness, fear

of losing control, nervousness, and indigestion. Each column is then summed for a total

score: scores of 0-7 indicate minimal anxiety; scores of 8-15 indicate mild anxiety; scores

of 16-25 indicate moderate anxiety, and scores of 26-63 indicate severe anxiety.

BAI reliability.

The one-week test-retest reliability for the BAI was found to be 0.75 (Beck,

Epstein, et al., 1988). The internal consistency was found to be 0.92. Beck and Steer

(1991) found that the items for the subjective, neurophysiological, autonomic, and panic

subscales were 0.87, 0.86, 0.74, and 0.72, respectively.

BAI validity.

Beck, Epstein, et al. (1988) found that the HAM-A was modestly correlated with

the BAI with a correlation of 0.51. For younger adults, the BAI was found to discriminate

panic and generalized anxiety disorders from major depression and dysthymia (Beck, Epstein, et al., 1988; Beck & Steer, 1991). Individuals who suffered from panic disorders scored significantly higher on the subjective, panic, and neurophysiological subscales (Beck, Epstein, et al., 1988; Beck & Steer, 1991). The BAI discriminated anxious diagnostic groups from non-anxious diagnostic groups (Beck, Epstein, et al., 1988). Beck and Steer (1991) found that there was discriminant validity because the mean BAI subjective, neurophysiological, and panic subscales scores of patients diagnosed with panic disorder were significantly higher than those diagnosed with generalized anxiety disorder (Beck, Epstein, et al., 1988). BAI items are also able to be distinguished from the BDI-II items (Hewitt & Norton, 1993). The BAI was mildly correlated (0.25) with the revised Hamilton Depression Rating Scale (Beck, Epstein, et al., 1988).

For older adults, Morin et al. (1999) found the following correlations for the BAI: low with the Brief Symptom Inventory global severity index; moderate with the BDI, the HARSD, the Hamilton Anxiety Rating Scale (HARS), and the Brief Symptom Inventory depression subscale; and high with the Brief Symptom Inventory anxiety subscale. Stuart et al. (1998) utilized the BAI at 14 weeks and 30 weeks for postpartum females. They found the following correlations at 14 weeks for the BAI with the BDI as 0.55, State Anxiety as 0.64, Trait Anxiety of 0.59, and Edinburgh Postnatal Depression Scale (EPDS) as 0.67. At 30 weeks, the correlations were found to be 0.28 for the BDI, 0.30 for State Anxiety, 0.42 for the Trait Anxiety, and 0.29 for the EPDS. Thus, the scores at 14 weeks was a fair predictor of later anxiety.

According to Hamilton (1959), the BAI subscales display both concurrent and discriminant validities for psychiatric outpatients diagnosed with anxiety disorders. He

found that there were positive correlations between the BAI subscales with the previously

established HARS Somatic and Psychic subscales. Hamilton (1959) believed that this

supported the concurrent validity of the BAI subscales.

Beck Hopelessness Scale

The Beck Hopelessness Scale (BHS) (Beck & Steer, 1989) is a 20-item self-report

measure that assesses one's level of pessimism or optimism towards the future (Bhar et

al., 2008). Eleven items are negatively phrased and nine are positively phrased to reduce

response bias. The items are rated either as true or as false and these scores are then

summed with total scores ranging from 0-20 (Bhar et al., 2008). Examples of questions

include: "I might as well give up because I can't make things better for myself"; "My

future seems dark to me"; "I am helpless when left on my own"; "I don't expect to get

what I really want"; "I look forward to the future with hope and enthusiasm"; "I have

great faith in the future", and "In the future, I expect to succeed in what concerns me the

most".

BHS reliability.

Beck, Steer, and Carbin (1988) found that the internal consistencies of the BHS

ranged from 0.87 to 0.93. The BHS has a one-week test-retest reliability of 0.69 and a

three-week test-retest reliability of 0.85 (Holden & Fekken, 1988). Durham (1982) found

that the reliability of the measure for psychiatric samples ranged from 0.86 to 0.83;

however, the reliability for college samples was 0.65. This indicates that the BHS may

not be a suitable measure for non-clinical populations. Bouvard, Charles, Guérin,

Aimard, and Cottraux (1992) found a test-retest reliability of 0.81 and an internal

consistency of 0.97.

BHS validity.

The concurrent validity for the BHS was found to range from 0.62 to 0.74 (Beck, Weissman, Lester, & Trexler, 1974). Another study found good concurrent validity with other scales measuring depressive cognitions, the automatic thoughts questionnaire, and a scale assessing suicide risk; however, no concurrent validity was found with scales measuring the intensity of depression, such as the BDI, and the HAM-D (Bouvard et al., 1992). The correlations between the BHS and the BDI pessimism items were found to range from 0.42 to 0.64 (Beck & Steer, 1989). In terms of predictive validity, Beck and Steer (1989) found that individuals with scores of at least nine were approximately 11 times more likely to commit suicide than those with scores of eight or below.

Hamilton Depression Measure

The Hamilton Depression Measure (HAM-D) (Hamilton, 1967) is a 17-item, clinician-administered structured interview scale. It assesses the severity of current depressive symptoms, including feelings of guilt, helplessness, and somatic problems. Sample items include: a) agitation – 0 "none", 1 "fidgetiness", 2 "playing with hands, hair, etc.", 3 "moving about, can't sit still", and 4 "hand wringing, nail biting, hair-pulling, biting of lips", and b) anxiety (psychological) – 0 "no difficulty"; 1 "subjective tension and irritability"; 2 "worrying about minor matters"; 3 "apprehensive attitude apparent in face and or speech", and 4 "fears expressed without questioning".

As scores increase, there is more of an indication of severe depression. Scores ranging from 0-7 indicate "no depression"; 8-12 indicate "mild depression"; 13-17 indicates "less than major depression" 18-29 indicate "major depression", and 30+ indicate "more than major depression" (Bech, 1993; Hamilton, 1967). When the HAM-D

scores decrease by 50%, therapeutic treatment outcome can be assessed. Also it can identify remission of symptoms with scores of less than eight and partial remission of symptoms with scores ranging from 8-14 (Frank, Prien, Jarrett, et al., 1991; Tedlow et al., 1998).

HAM-D reliability.

Bagby, Ryder, Schuller, and Marshall (2004) performed a literature review and found that the internal consistency of the HAM-D ranges from 0.46 to 0.97. Inter-rater reliabilities ranged from 0.82 to 0.98. Test-retest reliability ranged from 0.81 to 0.98.

HAM-D validity.

The convergent validity was found to be adequate overall except for two scales, one of which included the major depression section of the SCID-I. Thus there is evidence that there is a non-correspondence between the HAM-D and the DSM-IV (1994). Riskind et al. (1987) found that only eight items (depressed mood, suicide, feelings of guilt, work and activities, late insomnia, loss of weight, and psychomotor retardation) were significantly correlated with the diagnosis of major depression and the others were related to generalized anxiety. More specifically, Riskind et al. (1987) found that 38% ($r = 0.62$) of the content of the HAM-A and the HAM-D overlapped.

Hamilton Anxiety Measure

The Hamilton Anxiety Measure (HAM-A) (Hamilton, 1959) is a 14-item clinician administered scale that assesses the current level of severity of anxiety symptoms. The items that are measured include anxious and depressed mood, fears, insomnia, cognitive difficulties, autonomic symptoms, somatic muscular and sensory symptoms, cardiovascular symptoms, gastrointestinal symptoms, genitourinary symptoms, and

tension. These items are rated on a 5-point Likert scale ranging from 0 "not present"; 1 "mild"; 2 "moderate"; 3 "severe", and 4 "very severe". As the total score increases, the indications of anxiety become more severe.

HAM-A reliability.

The correlations for the HAM-A somatic and psychic subscales were 0.79 and 0.73, respectively (Beck & Steer, 1991). The internal consistency for this measure was found to be 0.78 (Riskind et al., 1987). Rehm and O'Hara (1984) found that the items reflecting agitation, gastrointestinal symptoms, and loss of weight detract from the measure's internal consistency and concurrent validity.

HAM-A validity.

Beck, Epstein, et al. (1988) found that the HAM-A was modestly correlated with the BAI with a correlation of 0.51. As mentioned above, the items that reflect agitation, gastrointestinal symptoms, and loss of weight detract from the measure's concurrent validity (Rehm & O'Hara, 1984). Also mentioned previously, Riskind et al. (1987) found that 38% (r = 0.62) of the content of the HAM-A and the HAM-D overlapped.

Structured Clinical Interview for DSM-IV Part II

The Structured Clinical Interview for DSM-IV Part II (SCID-II) is a semi-structured diagnostic instrument based on the 10 DSM-IV-TR (2000) diagnostic criteria for Axis II psychiatric disorders, including two non-DSM-IV diagnoses (depressive personality disorder and passive-aggressive personality disorder) (First et al., 1997). This measure can be used to make Axis II diagnoses, either categorically (present or absent) or dimensionally (by noting the number of personality disorder criteria for each diagnosis that are coded "3") (First et al., 1997). There are two parts to this instrument: a 120 item

self-report questionnaire, which takes about 15 minutes to complete, and the structured

interview (Jain, 2003).

The subject answers "yes" or "no" to items such as, "Are you usually quiet when

you meet new people?"; "Are you afraid of new things?", and "Do you usually feel

uncomfortable when you are by yourself?" (First et al., 1997). Then the interviewer

follows up the "yes" answers with greater in-depth questioning, allowing each personality

disorder criterion to be rated either as "?"; "1"; "2", or "3" (Jain, 2003). The rating of "?"

means that there is inadequate information to code the criterion as 1, 2, or 3; "1" means

the symptom described is absent or false; "2" means the threshold for the criterion is

almost, but not quite met; and "3" means that the threshold for the criterion is met or true.

The presence of each personality disorder is determined as the interview progresses.

When the interview is finished, the Summary Scoresheet is completed; this consists of the

dimensional score for each personality disorder as a result of summing the number of

items rated as positive.

SCID-II reliability.

Kuyken, Kurzer, DeRubeis, Beck, and Brown (2001) reported that the SCID-II

has fair to good reliability and good test-retest reliability. Maffei et al. (1997) found that

each scale had the following correlations: 0.97 for avoidant; 0.86 dependent; 0.83 for

obsessive-compulsive personality disorder; 0.91 for passive-aggressive; 0.65 for

depressive; 0.93 for paranoid; 0.91 for schizotypal; 0.91 for schizoid; 0.92 for histrionic;

0.98 for narcissistic; 0.91 for borderline, and 0.95 for antisocial.

SCID-II validity.

The convergent validity for the SCID-II and the DSM-IV (1994) Personality

Disorders were found to be 0.97 for avoidant personality disorder; 0.86 for dependent

personality disorder; 0.83 for obsessive-compulsive personality disorder; 0.91 for

passive-aggressive personality disorder; 0.65 for depressive personality disorder; 0.93 for

paranoid personality disorder; 0.91 for schizotypal personality disorder; 0.91 for Schizoid

personality disorder; 0.92 for histrionic personality disorder; 0.98 for narcissistic

personality disorder; 0.91 for borderline personality disorder, and 0.95 for antisocial

personality disorder (Maffei et al., 1997).

In terms of divergent validity, Ryder, Costa, and Bagby (2007) found that 53.2%

of the SCID-II symptoms correlated better with their own personality disorders,

compared with the other personality disorders. It was also found that 57.2% of the SCID-

II symptoms correlated with the 30 facets of the NEO-PI-R. Only 21.8% of the SCID-II

symptoms were correlated with Global Assessment of Functioning (GAF) ratings. In

conclusion, they found that most of the personality disorder traits were related to their

parent disorders; however, these relationships to their parent disorders were also highly

associated with other personality disorders.

Personality Beliefs Questionnaire

The Personality Beliefs Questionnaire (PBQ) is a 126-item self-report measure

that assesses beliefs that are associated with specific personality disorders based on the

DSM-IV (1994) personality disorders (Bhar et al., 2008). There are nine scales that

contain 14 items and each item is rated on a 4-point Likert scale with 0 as "I don't believe

it at all" and 4 as "I believe it totally" (Bhar et al., 2008; Nelson-Gray et al., 2004; Trull

et al., 1993). Example items include, "I cannot tolerate unpleasant feelings" (avoidant);

"If I am not loved, I will always be unhappy" (dependent), and "The only way I can

preserve my self-respect is by asserting myself indirectly" (passive-aggressive) (Jones,

Burrell-Hodgson, & Tate, 2007). All items are scored in the same direction with higher

scores representing higher levels of dysfunction (Trull et al., 1993). Each of the 14 items

on each of the nine scales is summed, resulting in a total score for each subscale (Trull et

al., 1993).

PBQ reliability.

Trull et al. (1993) found satisfactory reliability for the avoidant, dependent,

obsessive-compulsive, narcissistic, paranoid and the composite borderline personality

disorder subscales. Beck and Beck (2001) also found that each scale has adequate

reliabilities. The internal consistency of the PBQ was found to be good, because it ranged

from 0.77 to 0.93 (Trull et al., 1993). Similarly, Nelson-Gray et al. (2004) found that the

PBQ scales are highly, positively inter-correlated. The internal reliability of the PBQ has

been found to be adequate with correlations of 0.80 (Beck & Steer, 1993). The test-retest

correlations for the individual scales were found to be 0.57 for avoidant; 0.63 for

dependent; 0.74 for obsessive-compulsive; 0.81 for narcissistic; 0.71 for paranoid; 0.60

for histrionic; 0.80 for passive-aggressive; 0.78 for schizoid, and 0.93 for antisocial (Beck

et al., 2001).

Beck et al. (2001) found that each of the five scales (avoidant, dependent,

obsessive-compulsive, narcissistic, and paranoid) was related to the corresponding SCID-

II diagnosed personality disorder. Similarly, the PBQ subscales were found to correlate

from poor to good (i.e. 0.09 for antisocial to 0.57 for dependent) with corresponding

SCID-II trait scores (Arntz et al., 2004; Fydrich, Schmitz, Hennch, & Bodem, 1996).

PBQ validity.

Trull et al. (1993) found satisfactory validity for the avoidant, dependent,

obsessive-compulsive, narcissistic, and paranoid subscales, as well as for the composite

borderline personality disorder subscale. Nelson-Gray et al. (2004) found modest

concurrent validity between the PBQ and the MMPI-Personality Disorder Scales and the

Personality Diagnostic Questionnaire-Revised. The correlations with this measure and

clinical ratings of hopelessness are in the 0.70s (Beck & Steer, 1993).

Jones et al. (2007) found that the avoidant, dependent, passive-aggressive and

schizoid subscales correlated modestly but significantly with each of the Millon Clinical

Multiaxial Personality Inventory-III (MCMI-III) groupings, except for the schizoid and

dependent personality disorder subscales. These authors concluded the PBQ had good

discriminant validity and concurrent validity for passive-aggressive personality disorder,

avoidant personality disorder, schizoid personality disorder, dependent personality

disorder, and borderline personality disorder. Each of these personality disorders was

predicted only by the matched PBQ subscale score.

Procedure

All prospective participants were given the agency's standard three hour initial

diagnostic evaluation. During this time, each participant met with a psychiatrist and a

psychologist, as well as with a research assistant. A comprehensive history for each

participant was developed. At this time, all patients were given a brief

neuropsychological battery and various clinical assessments (i.e. the Brown Attention

Deficit Disorder Scale – Adult Version, the Beck Depression Inventory-II, the Beck

Anxiety Inventory, the Beck Hopelessness Scale, the Hamilton Depression Measure, the

Hamilton Anxiety Measure, the Personality Beliefs Questionnaire, the Clinical Global

Impression, the Clinical Global Impression for ADHD, the Structured Clinical Interview

for Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV Axis I disorders,

and the Structured Clinical Interview for DSM-IV Part II, the Structured Clinical

Interview for DSM-IV. Feedback was given to each participant regarding his/her

diagnostic impressions and for those that met the inclusion criteria, informed consent was

obtained.

Pharmacological intervention.

The medication used in the study was Adderall. Forty-five participants received

this medication; however, two participants were not included in the analysis because it

was found that they did not meet the inclusionary criteria of the study. The schedule was

as follows: 10 mg twice a day for one week; 15 mg twice a day for one week, and 20 mg

twice a day for one week. After an appropriate dose was found, based on the participant's

response, that dose was maintained throughout the study. Any changes to the medication

were discussed with the participant and changes were made, based on the participant's

approval.

Psychosocial intervention.

Forty-five participants received the cognitive-behavioral treatment; however, two

participants were not included in the analysis because it was found that they did not meet

the inclusionary criteria of the study. The CBT component consisted of sixteen, fifty

minute individual psychotherapy sessions, lasting six months. The treatment was adapted

for adult individuals diagnosed with ADHD. Initial sessions focused on skill development

and as therapy progressed, sessions were less frequent, allowing participants to rely on

their coping skills. The specific interventions included psychoeducation on ADHD;

individual case conceptualizations for each participant based on his or her difficulties;

reviews of coping strategies; cognitive and behavioral modification of patterns that limit

the use of appropriate coping skills, and the utilization of each of the skills.

Chapter Five: Results

Participants

Of the 43 original participants who sought outpatient treatment at the Adult

ADHD Treatment and Research Program of the University Of Pennsylvania Department

Of Psychiatry, located in Philadelphia, Pennsylvania, 35 SCID-II and 32 PBQ records

were used in this study. The mean age of participants was 30.8 with a standard deviation

of 9.4. Thirty-two males (74.4%) and 11 females (25.6%) participated in this study, with

a total sample of 43 participants. No gender differences between the groups were found.

The distribution of race was as follows: 88.4% were Caucasian; 0% were African

American; 2.3% were Asian; 2.3% were Hispanic, and 7.0% were listed as Other. The

mean years of the participants' education were 15.9 with a standard deviation of 2.8. The

distribution of ADHD subtypes were as follows: 69.8% the subjects met the criteria for

ADHD combined type, and 30.2% met the criteria for ADHD inattentive type. No

participant met the criteria for ADHD hyperactive-impulsive type. 16.3% of the subjects

had no comorbid psychiatric diagnoses, except for adjustment disorders. For those who

did have comorbid diagnoses, 62.8% met DSM-IV-TR criteria for a mood disorder;

53.5% met DSM-IV-TR criteria for an anxiety disorder, and 11.6% abused substances.

Table 1 provides the number of personality disorder symptoms endorsed by

individual subjects on the SCID-II. As can be seen in the table, participants most

frequently endorsed obsessive-compulsive personality disorder and borderline personality

disorder symptoms.

Table 1

SCID-II Results

D/Os	n	*M*	SD	R	DSM	Total	Percentage
APD	27	2.62	2.02	0-6	4	12	44%
DPD	31	2.53	1.81	0-7	5	12	19%
OCPD	33	4.52	1.94	0-8	4	24	72%
PAPD	28	3.12	2.32	0-8	4	13	46%
DSPD	30	3.5	2.29	0-7	5	13	43%
PPD	26	2.59	2.54	0-8	4	11	42%
STPD	13	0.94	1.41	0-4	5	0	0%
SPD	29	2.51	1.98	0-7	4	11	38%
HPD	28	1.86	1.42	0-5	5	2	7%
NPD	33	3.66	3.22	0-12	5	10	30%
BPD	31	4.77	3.54	0-13	5	18	58%
ASPD	26	2.14	2.44	0-11	3	12	46%

Note. Participants were more likely to endorse symptoms of OCPD and BPD, with 72% of the participants meeting the diagnosis of OCPD and 58% meeting the diagnosis of BPD.

Table 2 provides the results of the frequency with which individual subjects endorsed maladaptive beliefs on the PBQ. As can be seen in the table, participants most frequently endorsed passive-aggressive personality disorder and obsessive-compulsive personality disorder symptoms.

Table 2

PBQ Results

D/O	n	*M*	SD	R
APD	30	14.38	9.53	0-35
DPD	28	12.34	10.06	0-36
PAPD	31	18.60	10.99	0-48
OCPD	32	19.28	11.04	2-50
ASPD	32	9.62	6.57	2-34
NPD	32	10.91	7.41	1-26
HPD	32	14.34	8.26	2-35
SPD	32	15.22	8.93	3-41
PPD	28	11.63	12.54	0-48
Total	32	126.3	65.9	31-301

Note. Participants were more likely to endorse symptoms of PAPD and OCPD on the

PBQ.

Hypothesis 1

There will be a significant reduction in symptoms of ADHD and mood disorders

post-treatment.

A paired samples t-test was performed to determine if there was a difference

between pre- and post-treatment ADHD and mood disorder symptoms. The variables that

were analyzed included the BADDS Activation subscale, the BADDS Attention subscale,

the BADDS Effort subscale, the BADDS Affect subscale, the BADDS Memory subscale,

and the BADDS Total scores (See Table 3). Mood disorder symptoms were measured by

the BDI, BAI, BHS, HAM-D, and HAM-A (See Table 4).

Table 3

Changes in BADDS Subscale Scores from Pre- to Post-Treatment

BADDS Subscale	*df*	Pre-Tx Means	Post-Tx Means	*p*	*t*
Activation	26	73.70	64.56	.000**	4.281
Attention	26	75.48	62.89	.000**	5.287
Affect	26	64.67	57.67	.004*	3.126
Memory	26	68.22	57.93	.000**	5.284
Total	27	75.37	63.56	.002*	

Note. **$p <.001$, * $p <.05$, two-tailed test.

As seen in Table 3, there was a significant treatment effect on all of the BADDS

subscales. The t-test paired sample differences of each of the BADDS subscales from

initial scores to final scores were also significant, indicating a reduction in ADHD

symptoms from pre-treatment to post-treatment.

Table 4

Changes in Mood Scores from Pre- to Post-Treatment

Measures	*df*	Pre-Tx	Post-Tx	*p*	*t*
BDI-II	28	16.38	6.38	.000**	6.915
BAI	28	10.00	4.24	.003*	3.216
BHS	28	7.03	4.48	.001*	3.880
HAM-D	32	11.70	4.55	.000**	7.789
HAM-A	32	8.12	3.00	.000**	10.452

Note. BADDS = Brown Attention Deficit Disorder Scale; BDI-II = Beck Depression Inventory – II; BAI = Beck Anxiety Inventory; BHS = Beck Hopelessness Scale; HAM-D = Hamilton Depression Measure; HAM-A = Hamilton Anxiety Measure. **$p < .001$, *$p < .05$, two-tailed test.

As can be seen in Table 4, there was a significant effect for treatment on all of the mood measures from pre-treatment to post-treatment administration. Thus, all of the mood disorder symptoms showed a significant reduction in scores post-treatment.

Hypothesis 2

There will be a positive association between symptoms of ADHD pre- and post-treatment and disordered thinking.

A Pearson r correlation was performed to determine if there was a positive relationship between pre- and post-treatment BADDS scores and pre-treatment PBQ and SCID-II scores. More specifically, this was analyzed by correlating the initial BADDS score (total) with the scales of the PBQ and the SCID-II. At pre-treatment, four scales of the PBQ correlated significantly with pre-treatment scores BADDS score: Avoidant,

Dependent, Paranoid, and Total Score (See Table 5). Only the Histrionic scale of the

PBQ correlated with the post-treatment the BADDS score. Six scales on the SCID-II

were significantly correlated with the pre-treatment BADDS scores: Avoidant,

Dependent, Passive-Aggressive, Depressive, Paranoid, and Borderline (See Table 6).

Five scales of the SCID-II significantly correlated with the post-treatment BADDS

scores: Passive-Aggressive, Depressive, Schizotypal, Histrionic, and Borderline. A

limitation for the SCID-II correlations was that the *n* varied for each scale pre-treatment

because a small amount of the data was missing.

Table 5

Pearson r Correlations between the Total BADDS Pre- and Post-Treatment Scores and

the Pre-Treatment PBQ Scores

Scale	Pre-Tx BADDS	Post-Tx BADDS
	(*n* =32)	(*n* = 24)
Avoidant	.420*	.269
Dependent	.353*	.317
Passive-Aggressive	.342	.222
OCPD	.120	.309
Antisocial	.269	.054
Narcissistic	.234	.304
Histrionic	.283	.472*
Schizoid	.143	.241
Paranoid	.419*	.388
Total Score	.380*	.379

Note. *p <.05, two-tailed test.

Table 6

Pearson r Correlations between the Total BADDS Pre- and Post-Treatment Scores and

the Pre-Treatment SCID-II Scores

Scale	Pre-Tx BADDS	Post-Tx BADDS
Avoidant	.420*	.269
Dependent	.353*	.317
OCPD	.120	.309
Passive-Aggressive	.389*	.520**
Depressive	.392*	.474*
Paranoid	.419*	.388
Schizotypal	.252	.411*
Schizoid	.143	.241
Histrionic	.283	.472*
Narcissistic	.234	.304
Borderline	.435**	.438*
Antisocial	.269	.054

Note: **$p < .01$, *$p < .05$, two-tailed test.

Hypothesis 3

The presence of personality disordered thinking will interfere with changes in

ADHD symptoms.

A Pearson r correlational analysis was performed to determine if the presence of

personality disordered thinking correlates with changes in BADDS scores from pre- to

post-treatment. This was analyzed first by calculating change scores in symptoms of

ADHD on the BADDS from pre- to post-treatment. Next, these change scores were

correlated with PBQ scores and SCID-II scores. No scores reached significance,

suggesting that the presence of personality disordered thinking, as measured by the PBQ,

did not correlate with change in ADHD symptoms and consequently, did not interfere

with changes in symptoms of ADHD.

Table 7

Pearson r Correlations between the PBQ Pre-Treatment Scores and the Change in

BADDS Scores From Pre- to Post-Treatment

Scale	*BADDS* (n = 24)
Avoidant	.377
Dependent	.283
Passive-Aggressive	.332
OCPD	-.123
Antisocial	.330
Narcissistic	.137
Histrionic	.058
Schizoid	.186
Paranoid	.310
Total PBQ	.272

Note. Two-tailed test.

As can be seen in Table 7, no subscale scores on the PBQ were significantly

correlated with the Total BADDS post-treatment change scores, meaning that pre-

treatment personality disorder thinking did not interfere with the changes in BADDS

scores.

Table 8

Pearson r Correlations between the SCID-II Pre-Treatment Scores and the Change in

BADDS Scores From Pre- to Post-Treatment

Scale	BADDS
Avoidant	.377
Dependent	.283
OCPD	-.123
Passive-Aggressive	-.037
Depressive	.148
Paranoid	.310
Schizotypal	.035
Schizoid	.186
Histrionic	.058
Narcissistic	.137
BPD	.162
Antisocial	.330

Note. Two-tailed test.

As can be seen in Table 8, no scores on the SCID-II were significantly correlated

with BADDS post-treatment change scores, meaning that pre-treatment personality

disorder symptoms did not interfere with changes in BADDS scores. A limitation for the

SCID-II correlations was that the *n* values varied for each scale pre- and post-treatment.

Hypothesis 4

The presence of a personality disorder will interfere with reductions in symptoms of mood disorders.

A Pearson r correlational analysis was performed to determine if the presence of a personality disorder interferes with changes in mood scores. This was analyzed first by calculating change scores in symptoms of depression as indicated by the BDI-II, BAI, BHS, HAM-D, and HAM-A. Next, these change scores were correlated with the PBQ scores (See Table 9) and SCID-II scores (See Table 10). On the PBQ, none of the correlations between the PBQ and the HAM-D or HAM-A scores reached significance. However, a significant correlation was found between the PBQ Avoidant subscale and changes in the BDI-II scores, the BAI scores, and the BHS scores. A significant correlation was found between the PBQ Dependent subscale and changes in BAI scores. A significant correlation was also found between the PBQ Antisocial subscale and changes in BDI-II scores. On the SCID-II, the Dependent subscale was correlated significantly with changes in the BAI scores. The Antisocial subscale correlated significantly with changes in the BDI-II scores. The Avoidant subscale significantly correlated with changes in the BDI-II scores, the BAI scores, and the BHS scores. A limitation for the SCID-II correlations was that the *n* varied for each scale pre- and post-treatment.

Table 9

Pearson r Correlations between the Pre-Treatment PBQ Scores and the Change in Mood

Scores From Pre- to Post-Treatment

PBQ Scales	BDI	BAI	BHS	HAM-D	HAM-A
	(n = 26)	(n = 26)	(n = 26)	(n = 30)	(n = 30)
Avoidant	.474*	.435*	.391*	.161	.286
Dependent	.260	.395*	.322	.105	.301
Passive-Agg	.333	.058	.160	.323	.142
OCPD	-.229	-.061	-.259	-.104	-.089
Antisocial	.394*	.140	.214	.319	.117
Narcissistic	.149	.150	-.069	-.011	-.016
Histrionic	-.039	.052	-.127	.114	.014
Schizoid	.168	.023	-.005	.141	.035
Paranoid	.123	.217	.177	-.110	-.051
Total	.220	.204	.121	.118	.103

Note. *$p < .05$ level, two-tailed test.

Table 10

Pearson r Correlations between the Pre-Treatment SCID-II Scores and the Change in

Mood Scores From Pre- to Post-Treatment

PBQ Scales	BDI	BAI	BHS	HAM-D	HAM-A
Avoidant	.474*	.435*	.391*	.161	.286
Dependent	.260	.395*	.322	.105	.301
OCPD	-.229	-.061	-.259	-.104	-.089
Passive-Agg	.271	.149	-.040	-.009	.156
Depressive	.171	.095	.068	.022	.154
Paranoid	.123	.217	.177	-.110	-.051
Schizotypal	.117	.206	.020	.022	.249
Schizoid	.168	.023	-.005	.141	.035
Histrionic	-.039	.052	-.127	.114	.014
Narcissistic	.149	.150	-.069	-.011	-.016
BPD	.270	.176	.098	-.086	.030
Antisocial	.394*	.140	.214	.319	.117

Note. *p < .05 level, two-tailed test.

Chapter Six: Discussion

The prevalence of adult ADHD is between one and six percent of the general population and the disorder results in significant impairment for many of these individuals. There are a number of promising interventions for ADHD, both psychological and pharmacological. For example, a recent study by Rostain and Ramsay (2006) examined the effects of CBT, adapted to treat adult ADHD symptoms, in combinations with psychopharmacology (specifically Adderall). They found that this combination treatment resulted in improvements in self-reported symptoms, as well as in ratings by independent evaluators. These results are consistent with the other findings determining that the optimal treatment for adults ADHD is the use of a multi-modal approach that utilizes both behavioral and pharmacological interventions (e.g., Young, 2006).

Using the same data from the study performed by Rostain and Ramsay (2006), this study examined the potential adverse effects of personality disorder symptoms on the treatment efficacy for adult ADHD. Because there has been limited research focusing on the effects of personality disorder symptoms on the treatment of ADHD, this study adds to the literature pertaining to the interaction of personality disorder symptoms, maladaptive beliefs, and ADHD on treatment outcome. The importance of examining this interaction is that problematic personality characteristics have been hypothesized to hinder treatment and prolong the suffering of individuals because of distorted cognitions, affective dysregulation, maladaptive behavior, and poor interpersonal functioning (APA, 2000; Rostain & Ramsay, 2008).

Hypothesis 1

The first hypothesis predicted that there would be a significant reduction in symptoms of ADHD and mood disorders symptoms, post-treatment. This hypothesis was supported. All of the BADDS subscale scores from pre-treatment to post-treatment were found to be significant. All of the mood disorder and hopelessness scores (on the BDI-II, the BAI, the HAM-D, the HAM-A, and the BHS) also significantly changed from pre-treatment to post-treatment. Thus all of the ADHD and mood disorder symptoms showed a significant reduction in scores, post-treatment.

The following studies examined the effect of CBT on ADHD symptoms, but did not examine the role of personality disorder symptoms in treatment outcome. Solanto, Marks, Mitchell, Wasserstein, & Kofman (2008) examined a program utilizing CBT that focused on impairments in time management, planning, and organizational skills. They found significant improvement on measures of core ADHD symptoms of inattention, as well as on specific scales of activation, attention, memory, effort, and affect on the BADDS. Wilens et al. (1999) also found that CBT resulted in significant improvement in ADHD symptoms, anxiety, depression, and overall functioning when combined with pharacotherapy. Philipsen et al. (2007) found that group CBT combined with pharmacotherapy also resulted in improvements in ADHD severity and depressive symptoms, as measured by the BDI. Due to the high comorbidity rate of 78.5% (i.e. affective disorders, substance abuse/dependence disorders, and anxiety disorders), some of the participants may have met the criteria for an Axis II disorder.

This study focused on the treatment of ADHD with a combined treatment regimen of CBT and pharmacotherapy. The combination treatment of CBT and pharmacotherapy

has been found to be associated with improvements in symptoms of ADHD and overall

functioning and are considered to be the first-line treatment (Goodman, 2005; Ramsay,

2007; Safren et al., 2005; Torgersen, Gjervan, & Rasmussen, 2008; Wender, 1998;

Wilens, Biederman, & Spencer, 2002) because 20-50% of adults do not respond to

stimulant medication alone and of those who do, there is only a 50% or less reduction of

the core ADHD symptoms (Safren et al., 2005; Wilens, Spencer, & Biederman, 1998).

This may be, in part, due to CBT's use of teaching skill-building to help individuals

compensate for executive dysfunction; it may also be due, in part, to psychoeducation

and self-monitoring (Ramsay, 2007). Because Adderall helps increase one's attention, it

may reduce some of the executive dysfunction experienced by these individuals.

Resultant improvements in executive dysfunction may also reduce problems with

motivation, interpersonal interactions, affect, and impulse control that are often seen in

ADHD, anxiety, and mood disorders.

Post-treatment reductions in mood and hopelessness scores are consistent with the

notion that CBT reduces one's emotional intensity and dysfunctional thinking by helping

the individual challenge and replace his/her misperceptions and negative assumptions

with factual information (Goodman, 2005; Ramsay, 2007). In doing so, one is more able

to adjust his/her impulses, regulate his/her mood, and choose a more rational response to

various situations (Goodman, 2005). Based on this, CBT likely reduces the impulsive

symptoms found in ADHD, as well. By helping one handle possible future difficulties via

CBT, one is likely to develop a sense of resilience (Ramsay, 2007), helping to alleviate

mood symptoms.

Similarly, Bramham, Young, Bickerdike, Spain, McCartan, and Xenitidis (2009) found that the use of brief CBT in a group format resulted in improvements on their participants' psychological symptoms (i.e. self-esteem, self-efficacy, anxiety, and depression), as well as their knowledge level. This study also measured ADHD symptoms, and the results support the notion that CBT can help reduce depression and anxiety, and improve self-image.

Hypothesis 2

The second hypothesis of this study predicted that there would be a positive association between symptoms of ADHD, pre- and post-treatment and pre-treatment maladaptive beliefs measured by the PBQ and SCID-II. More specifically, it was hypothesized that the more maladaptive beliefs one has, the more severe the total BADDS scores will be, both pre- and post-treatment. This hypothesis was partially supported. It was found that pre-treatment symptoms of avoidant, dependent, and paranoid personalities as measured by the PBQ were significantly correlated with pre-treatment BADDS scores. Conversely, only the beliefs associated with histrionic personality disorder were significantly correlated with post-treatment BADDS scores. However, on the SCID-II, symptoms of avoidant, dependent, passive-aggressive, depressive, paranoid, and borderline were significantly correlated with pre-treatment BADDS scores. At post-treatment, only the symptoms of passive-aggressive, depressive, schizotypal, histrionic, and borderline symptoms were significant with post-treatment BADDS scores. It appears that the differences between the two measures on pre- and post-treatment symptoms correlating with BADDS scores are due to the fact that the

SCID-II incorporates extra scales (i.e. passive-aggressive, borderline, schizotypal, and depressive personality disorders).

Hypothesis 3

The third hypothesis predicted that the presence of personality disordered thinking would interfere with reductions in ADHD symptoms. This hypothesis was not supported, suggesting that the presence of personality disordered thinking does not interfere with changes in ADHD symptoms. Similarly, Hardy et al. (1995) found that the diagnoses of cluster C personality disorders and the diagnosis of depression did not significantly predict cognitive therapy outcome.

Overall it appears that symptoms of ADHD improved from pre- to post-treatment despite the presence of personality disorder symptoms. This indicates that the combination of CBT and Adderall can help reduce the symptoms of both ADHD and personality disorders.

Hypothesis 4

The fourth hypothesis predicted that the presence of personality disorder symptoms would interfere with reductions in symptoms of mood disorders. This hypothesis was not supported. Instead, three scales of the PBQ were positively correlated with changes in mood symptoms. More specifically, the avoidant scale was significantly and directly correlated with the changes in the BDI-II scores, the BAI scores, and the BHS scores. The dependent personality scale was significantly correlated with the changes in the BAI scores. On the SCID-II, the Dependent subscale was correlated significantly with changes in the BAI scores. The Antisocial subscale significantly correlated with changes in the BDI-II scores. The Avoidant subscale correlated

significantly with changes in the BDI-II scores. The antisocial scale was significantly

correlated with the changes in BDI-II scores. These correlations suggest that the presence

of avoidant, dependent and antisocial personality symptoms actually aided in the

reduction of mood disorders in the treatment of ADHD. Thus it appears that these

symptoms actually increase the probability of therapeutic change.

Other researchers found similar results; that is, that personality disorder

symptoms did not impede treatment outcome for other Axis I disorders. CBT was

effective in treating major depression (Joyce et al., 2007), obsessive-compulsive disorder,

panic disorder with agoraphobia, and eating disorders, regardless of the presence or

absence of comorbid Axis II problems (van den Hout, Brouwers, & Oomen, 2006). These

authors found that there was no effect of personality disorders on treatment effectiveness

for the Axis I conditions and concluded "the presence or absence of comorbid Axis II

pathology seems irrelevant for the decision to treat Axis I patients with CBT" (p. 63).

Neuhaus et al. (2007) also found that a two-week CBT partial hospitalization program for

individuals diagnosed with mood, anxiety, and/or personality disorders had significant

improvements on psychiatric symptomatology, with decreased scores on the BDI-II and

the BHS, and increased skill acquisition.

Similarly Hesslinger, Tebartz van Elst, Nyberg, Dykierek, Richter, et al. (2002)

examined the effects of Dialectical Behavior Therapy and CBT on individuals diagnosed

both with ADHD and with borderline personality disorder and found significant

improvements on all measures. Joyce et al. (2007) found that patients with avoidant

symptoms responded better to CBT when compared with interpersonal psychotherapy;

however, this study did not specify whether or not any of their subjects were diagnosed

with ADHD. Similarly, Kuyken et al., (2001) found that maladaptive avoidant and paranoid beliefs improved the reduction of the severity of depression. However, they did not find that dependent, obsessive-compulsive, and narcissistic beliefs predicted response to cognitive therapy. This study also did not specify whether or not any of their subjects were diagnosed with ADHD.

Miller et al. (2008) reported that the relationship between ADHD and personality disorders might be phenomenological due to the overlapping diagnostic criteria. It may also be that these disorders co-occur because of common neurobiological and/or risk factors (e.g. hypothalamic-pituitary-adrenal axis dysregulation and/or adverse early experiences with fear or anger). For example, research has found a relationship between borderline personality disorder and ADHD, possibly due to shared clinical features and diagnostic criteria, such as emotional dysregulation and impulsivity (Philipsen et al., 2008). Ramsay, Rosenfield, and Harris (2011) hypothesized that the relationship between antisocial personality disorder and ADHD may be more common for individuals diagnosed with ADHD combined type, who previously had met the diagnostic criteria for conduct disorder.

Other researchers have concluded that individuals diagnosed either with certain personality disorders or with ADHD, as well as with depression, appear to have similar cognitive deficits, such as poor problem-solving abilities (Coolidge, Thede, & Jang, 2004; Harley et al., 2006). More specifically, Coolidge et al. (2004) reported that some individuals with personality disorders could appear to have many characteristics of executive function deficits (i.e. poor judgment, problems with decision making, difficulty with selective attention, impulsivity, and inflexibility), which are identical to the

symptoms found in ADHD. Coolidge et al. (2004) found that there were significant

genetic influences of executive function deficits and avoidant, borderline, dependent,

depressive, histrionic, passive-aggressive, and conduct personality disorder scales.

Consequently, one of the therapeutic benefits of CBT for depression and personality

disorder symptoms may be the targeting of executive function deficits, in addition to

schema modification.

Avoidant personality disorder

CBT for avoidant personality disorder symptoms in individuals with comorbid

ADHD nicely illustrates the heterogeneity of the ADHD population and the potential

synergistic benefits of CBT in such complicated cases. After repeated, perceived failures,

frustration, and rejection early in life, some patients with ADHD may develop avoidance

as a compensatory strategy because this maladaptive coping style allows them to escape

immediate, painful experiences (Beck, Freeman, et al., 1990; Beck, Freeman, Davis, et

al., 2004). For example, the reduction of avoidant symptoms may result from CBT's use

of cognitive and behavioral modification, which exposes individuals with ADHD to

situations and experiences that they would normally avoid, thus allowing them to develop

and reinforce newly learned coping skills (Ramsay, 2007). Specifically, one of the

aspects that Lovell, Marks, Noshirvani, Thrasher, and Livanou (2001) examined was the

effectiveness of exposure therapy on avoidant symptoms for individuals with

posttraumatic stress disorder. These authors found that the avoidant symptoms were

significantly reduced with the use of exposure therapy. More specifically, Franklin,

Ledley, and Foa (2008) report that the use of exposure therapy helps one confront

situations, thoughts, or objects that create anxiety or distress. This is achieved by

encouraging the individual to remain in the feared situation.

Coolidge et al. (2004) reported that individuals suffering from avoidant

personality disorder symptoms have feelings of inadequacy, low self-esteem, and doubts

about their social competence and hypothesized that these difficulties may be a result of

executive function deficits similar to those seen in ADHD. Thus it seems that if these

overlapping cognitive deficits are targeted in treatment, it makes sense that both

symptoms of ADHD and avoidant personality disorder symptoms would decrease.

Consequently, it appears that some of the symptoms of avoidant personality disorders are

similar to those found in some individuals diagnosed with ADHD. Therefore treatment of

both is similar and provides synergistic effects.

Dependent personality disorder

For those with dependent personality symptoms, one's underlying beliefs are

inadequate or one feels a sense of helplessness in a lonely and potentially dangerous

world. Compensatory strategies generally include dependence on others to support, guide

and protect these individuals. Dependent individuals happily relinquish responsibility to

others in a desperate attempt to avoid being abandoned (Beck, Freeman, et al., 1990;

Beck, Freeman, Davis, et al., 2004)). CBT challenges these individuals to be more

autonomous via making up their own agendas for each session, which can help them to

become more motivated for change. For instance, early in treatment, autonomy increases

via the individual collaboratively forming the agenda, rather than the therapist dictating

one. This allows these individuals to take more responsibility for their treatments, which

the therapist can then reinforce. As these individuals improve their autonomy,

generalizations can be made to their lives outside of the therapy session to further

challenge core beliefs of inadequacy and helplessness.

Antisocial personality disorder

Individuals diagnosed with antisocial personality disorder tend to behave

aggressively, believing that they can and must obtain power and control over others

(Nauth, 1995). Their underlying belief is that individuals in authority will control them if

they follow orders. Empowerment may occur through teaching basic emotions, and

through helping these individuals understand that they are in charge of their own

thoughts, feelings, and behaviors. Woody, McLellan, Luborsky, and O'Brien (1985)

found that comorbid depression aided individuals diagnosed with antisocial personality

via improving their willingness to work in therapy. More specifically, they found that

opiate-dependent individuals with these comorbid disorders improved significantly in

many areas and responded almost as well as those diagnosed only with depression. Black

(2007) also reported that treating comorbid disorders such as depression, bipolar disorder,

or substance use disorders might reduce antisocial symptoms. Bockian (2006) reported

that individuals with antisocial personality disorders are prone to depression and that

treatment can target the distorted beliefs, using cognitive techniques such as Socratic

dialogue and thought records. Behavioral contracting and cost-benefit analyses can also

be utilized (Bockian, 2006). CBT and the medication treatment may help these

individuals reduce their impulse control. Furthermore, role-playing with a consistent

therapist helps individuals with avoidant, dependent, and antisocial symptoms develop

improved social skills, which can disrupt the cycles of perceived rejection, disruptive

behavior, and actual rejection. Also, for these disorders, it appears that these individuals

developed improved attention and frustration tolerance, allowing them to make use of the

therapy to its full extent.

Significance

This study focused on determining personality factors that influence treatment

outcome for adult patients diagnosed with ADHD. According to Miller, Miller, Newcorn,

and Halperin (2008), "systematic assessment of personality in adolescents and adults

previously diagnosed with ADHD is likely to offer new insights into lifelong functioning

associated with ADHD, and may also provide information related to impairment" (p.

165). Similarly, Ramsay, Rosenfield, and Harris (2011) reported that determining

personality factors within the context of evaluating learning and attention problems helps

provide a comprehensive view of an individual's functioning. Nigg et al. (2002) also

reported that the symptoms of ADHD may be related to personality traits because of the

close connection between the executive and motivational systems of the brain, which

produce symptoms common to both disorders. Illuminating this relationship may help to

expand existing theories of the origins and outcomes for individuals with ADHD.

However, Nigg et al. (2002) noted "such theoretical considerations require more data

regarding the empirical association between ADHD symptoms and personality traits" (p.

452). This study provides further empirical clarification in this regard.

The four findings of this study have significant implications for the treatment of

adult ADHD and comorbid personality disorders. The use of Adderall and CBT appears

to help reduce both ADHD and mood disorder symptoms and improve symptoms of

ADHD, despite the presence of personality disorder symptoms. It is important for

therapists to recognize that even if an individual has maladaptive thoughts due to

personality disorder symptoms, improvement in ADHD symptoms can occur. Also, the

recognition that avoidant, antisocial, and dependent personality symptoms may enhance

treatment outcome is important. These results challenge the long-held belief within the

profession that personality disorders interfere with treatment change in the short- and

long-term. If these results can be duplicated, with the improvements listed later in this

document, the first line treatment for individuals with ADHD may be CBT and

pharmacotherapy. Of greatest importance, populations that do not have access to

specialty clinics will likely be able to be treated at local community mental health centers

that can adopt this approach. In the long-term, implementing these recommendations

should provide positive effects for patients and for society, by means of helping

individuals diagnosed with ADHD attain higher levels of education, improve work

performance, reduce healthcare costs and healthcare utilization, reduce motor vehicle

accidents, improve levels of self-esteem and life satisfaction, reduce legal problems, and

develop social skills, decision making skills, and impulse control skills.

Limitations

There are several limitations to this study. The sample size available for this study

was small (n = 35) and consisted of individuals not typical of many in the greater ADHD

population. More specifically, the subjects in the present study had access to a specialty

clinic research team that they were able to locate and for whom they were able to pay.

They were also probably more likely to adhere to treatment than individuals without this

access. The average education level was also atypically high. Additionally, subjects were

required to currently be enrolled in college or graduate school, or to be working, thus

excluding more functionally impaired individuals. This study also was also limited in the

cultural diversity of participants. Consequently, although these individuals may represent those seeking treatment at the coauthors' specialty clinic, they may not represent more severely impaired adults with ADHD who are more globally impaired.

Another limitation of this study was that no follow-up data were collected. In order to determine the durability of treatment response empirically, it is necessary to evaluate outcome at follow-up. Areas to examine at follow-up are functioning level, self-concept, and objective evidence of adaptive skills in daily life. In addition, post-treatment data were not collected on the PBQ and SCID-II subscales to detect whether or not CBT affects overall changes in personality disorder symptoms. These limitations can be addressed in future research.

Future Research

Future research should attempt to replicate the findings of this study. Areas to improve include increasing the sample size and using a randomized clinical control group. Future studies can examine whether or not the results of this study apply to individuals treated in various treatment programs that do not specialize in ADHD treatment, as well as to individuals that have lower levels of education and to those that are ethnically diverse. Adding another ADHD measure, such as the CAARS, can provide further validity of an ADHD diagnosis.

Collection of post-treatment data on PBQ and SCID-II scores would also be beneficial. Pre- and post-treatment data collection on personality disorders, interpersonal skills, and overall dysfunction would provide more meaningful clues about the reasons why this treatment was so effective.

Future research should include longitudinal studies examining the efficacy and effectiveness of this combination treatment on ADHD symptoms and comorbidities over a longer period of time to determine if treatment outcome is maintained. Furthermore, it would be valuable to assess the extent to which different personality disorder symptoms and beliefs comorbid with ADHD affect different areas of functioning (e.g. depression, anxiety, ADHD, and quality of life). A future study should examine whether or not improvements in one's level of self-esteem and self-efficacy are also contributing factors in the improvement in symptoms of comorbid personality disorders and ADHD.

Future research should also examine how CBT techniques affect the personality disorders and the reasons why they seemed actually to improve treatment outcome (i.e. dependent, antisocial, and avoidant symptoms). For example, what are the factors within dependent, antisocial, and avoidant symptoms that can lead to positive change? The fact that the presence of personality disorder symptoms did not impede successful outcome is an important finding and one that warrants future research, especially given the lack of research in this area.

Conclusion

Overall, the findings of this study indicate that the combination of CBT and Adderall can be efficacious in reducing the symptoms not only of ADHD, but also symptoms of comorbid personality disorders in adults. More specifically, it may be that this combination of treatment explicitly challenges and restructures the beliefs that develop as a result of maladaptive behavior patterns. This suggests that early treatment can alleviate the typical negative experiences of adults diagnosed with ADHD, allowing them to persevere through the difficulties they often endure. If these results are

replicated, it is likely that the treatment protocol in Rostain and Ramsay (2006) can be

applied as a first-line combination treatment for adults struggling both with ADHD and

with personality disorder symptoms.

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