RESEARCH TRAINING PROPOSAL

(I) Specific Aims

Specific Aim #1: To test for ethnic differences in the internal validity of DSM-IV ADHD.

In order to better understand ethnic differences in the manifestation of the disorder, the internal validity of symptoms dimensions and DSM-IV subtypes will be compared in two community samples of African American (AA) and Caucasian American (CA) subjects.

Specific Aim #2: To test for ethnic differences in the external correlates of DSM-IV ADHD.

2a: The current study will assess functional impairment, academic achievement, and comorbidities with other psychiatric disorders across ethnic groups. If there are prevalence differences between DSM-IV subtypes across groups, the current study will determine whether those differences are meaningful in terms of functional impairment.

2b: This study will also test for ethnic differences in the neuropsychological profiles of children with ADHD. We will compare the performance of participants by group on measures of behavioral inhibition, sustained attention and vigilance, working memory, processing speed, and delay aversion to determine whether the same deficits exist, in the same proportions, in an AA sample as in a CA sample.

(II) Background/Significance

Attention deficit/hyperactivity disorder (ADHD) is one of the most common disorders of childhood, affecting about 3-5% of school-age children. Although it is a common focus of research studies, with a recent search on PsychInfo yielding over 6,400 published articles on the disorder, the vast majority of the work has been conducted with Caucasian American (CA) children. Many of these studies include children of other ethnicities in proportion to their representation in the general population, yet these numbers are frequently not large enough to examine ethnic differences. Thus, relatively little is known about the manifestation of ADHD in other ethnic populations. The purpose of this study is to examine the internal and external validity of ADHD in an African American (AA) sample, including examining whether neuropsychological profiles vary by group.

The following sections will provide an overview of the internal and external validity of ADHD in the current research literature, as well as what is known about its cognitive profile. This review includes studies which predominantly use CA samples. Next, the literature on ADHD in AA samples will be discussed, with important issues highlighted. Finally the goals of the current study will be explained in greater detail.

ADHD in Typically-Defined Samples:

ADHD has been extensively researched in samples composed mostly of CA children, resulting in several well replicated results. There is a stable and reliable two-factor structure that defines ADHD (DSM-IV-TR, American Psychiatric Association, 2000). The first component is defined by symptoms of inattention and the second component is defined by symptoms of hyperactivity and impulsivity. There are three subtypes, defined by the number of symptoms in each of these two factors: predominantly inattentive, predominantly hyperactive/impulsive, and combined. Six of the nine symptoms endorsed on each factor leads to the combined subtype, and someone who meets those criteria on only one factor qualifies for that subtype. Current research suggests that there is more internal and external validity for the predominantly inattentive and combined subtypes than the predominantly hyperactive/impulsive subtype (Willcutt, Pennington, & DeFries, 2000).

Research on the etiology of ADHD suggests that it is a highly heritable disorder ($h^2=.76$; Faraone et al. 2005), and there are several replicated candidate genes associated with ADHD, including DAT1 (Cook, Stein, Ellison, & Unis, 1995) and DRD4 (Faraone, Doyle, Mick, & Biederman, 2001). Research on the environmental influences on ADHD has primarily focused on the effect of prenatal exposure to tobacco and alcohol (Linnett et al., 2003; Barkley, 1996; Milberger, Biederman, Faraone, Chen, et al., 1996; Mick, Biederman, Faraone, Sayer, & Kleinman, 2002) as well as sequellae from perinatal and birth complications (Mick, Biederman, Prince, Fischer, Faraone, 2002; Breslau, Chilcoat, & DelDotto, 1996; Hartsough & Lambert, 1985). However, when thinking about these as “environmental” events, it is important to remember that exposure to these substances and events is not randomly assigned and could be correlated with ADHD in the parents, thus confounding these environmental risk factors with genetic ones. There is no evidence that the social environment, including parenting practices, can directly cause ADHD (Faraone, 2005), although it may play a role in its expression.
Gender ratios in referred samples of children with ADHD have been reported to be as high as 9:1 (males:females), and epidemiological studies report gender ratios of 3:1 (Szatmari, Offord, & Boyle, 1989). Over half the children who meet diagnostic criteria for ADHD also qualify for a comorbid disorder (Biederman, Faraone, Keenan, & Benjamin, 1992), which include conduct disorder and oppositional defiant disorder, depression, anxiety, Tourette Syndrome, dyslexia, and bipolar disorder. ADHD is a disorder that is most frequently first observed in toddlerhood and preschool aged children (Palfrey, Levine, Walker, & Sullivan, 1985) and is a chronic disorder across the lifespan (Gittelman, Mannuzza, Shenker, & Gonagura, 1985, Faraone, 2005).

Research on the neuropsychology of ADHD has established that executive deficits are a cognitive correlate of ADHD. Specifically, measures of behavioral inhibition, vigilance, verbal working memory, and processing speed all differentiate individuals with and without ADHD (Willcutt et al. 2005). However, executive deficits do not account for all individuals with ADHD (Doyle, Biederman, Seidman, Weber, & Faraone, 2000). Research on other explanations for ADHD besides an executive deficit has explored the role of motivational and temperamental factors on the expression of ADHD. One of the main alternative theories of ADHD is the Delay Aversion theory (Sonuga-Barke, Taylor, Sembali, & Smith, 1992). This theory posits that individuals with ADHD dislike waiting for a rewarding stimulus and choose shorter delays with a lesser reward rather than waiting longer for a larger reward. In addition, Delay Aversion problems and behavioral inhibition as measured by the Stopping Task both account for independent variance in ADHD symptoms (Solanto et al., 2001; Sonuga Barke, Auerbach, Campbell, Daley, & Thompson, 2005; Sonuga-Barke, 2002; Sonuga-Barke, Dalen, & Remington, 2003). Current research is beginning to explore multiple deficit accounts of ADHD, which allow for multiple genes, brain regions, cognitive, and motivational or temperamental deficits to play a role in the expression of the disorder (Pennington, in press).

**ADHD in African Americans:**

Although the number of ethnic minorities in the United States is steadily rising and ADHD is one of the most common disorders of childhood, there have been very few studies specifically examining ADHD in ethnic minority samples. In fact, a 1997 paper by Samuel and her colleagues attempted to identify all the existing research papers published on ADHD in African American samples and only found 16 articles on the topic. Within those 16, only a few had ethnicity as a primary focus of the study. Since that time, only a handful of additional studies have addressed the issue. The majority of the research has attempted to explore internal validity issues in ADHD rating scales, yet there remains a variety of unexplored domains in both the internal and external validity of ADHD in African American samples. In spite of the fact that little is known about whether the construct of ADHD operates similarly across ethnic groups, it is commonly used to diagnose individuals in minority populations.

**Internal Validity:** One of the most important questions is whether ADHD as it is typically defined and normed is a valid diagnosis for AA individuals, and there is some evidence that it is. Specifically, several studies have found the general two factor structure of ADHD exists in AA samples (Reid, 1995; DuPaul, Power, Anastopoulos, Reid, McGoey, Ikeda, 1997; Epstein, March, Conners, & Jackson, 1998; Reid, Casat, Norton, Anastopoulos, & Temple, 2001). In addition, one pilot study found that ADHD is familial in a small AA sample (Samuel et al., 1999), although that result needs to be replicated in larger samples. Finally, the male preponderance is also found in AA samples (Reid et al., 2000).

However, there is also some evidence that suggests that the construct of ADHD does not work identically in AA and CA samples. Several studies that have examined rates of ADHD across ethnic groups have found that AA children are consistently rated more highly by teachers on ADHD rating scales than their CA counterparts (Reid, 1995; Reid et al., 1998; Epstein et al., 1998; DuPaul et al. 1997; Reid et al. 2000; Samuel et al., 1997; Reid et al., 2001; Arnold et al., 2003; Nolan, Gadow, & Sprafkin, 2001). There is conflicting evidence about diagnostic rates as evidenced by parent reports, with some studies finding increased rates of ADHD in AA compared to CA samples (DuPaul et al. 1998), and some finding lower rates by parental report (Stevens, Harman, & Kelleher, 2005). Any difference in prevalence rate or systematic difference between teacher and parent ratings needs to be understood before establishing the validity of this construct in AA samples. In addition, there are ethnic differences in prevalence rates of symptoms which vary by symptom dimension, such that AA children are rated higher on HL symptoms than CA children when rated by a teacher (DuPaul et al., 1997). There is also some evidence that gender interacts with ethnicity in ADHD ratings (Epstein et al. 1998; Reid et al., 2000). Reid and his colleagues (2000) found that AA males were seen as most severe, the AA females and CA males were indistinguishable and separable from AA males, and the CA females were seen as the least severe. The
greatest difference in ratings was found for CA children, on which males and females were rated quite differently. Symptoms of ADHD in AA males and females were rated much more similarly.

While there is some evidence that the general construct of ADHD is valid across CA and AA groups, these mixed findings imply that the diagnosis may not work identically in the two groups. There are a variety of reasons why ratings of ADHD symptoms might differ across ethnic groups. First, there may be genuine differences in symptom levels between AA and CA children such that AA children are more likely to have ADHD (e.g., Eme, 1992). This may stem higher levels of environmental risk factors in AA communities or even from genetic differences. There has been no research on the behavioral or molecular genetics of ADHD in AA samples, so little is known about the possibility of genetic differences in etiology. In contrast, research on bioenvironmental risk factors has suggested that AA babies are more likely to be born prematurely, be smaller at birth, and die during the first year of life (Ketterlinus, Henderson, & Lamb, 1990). In addition, AA mothers are less likely to have had prenatal care (Abel, 1996) and are more likely to abuse substances during pregnancy (Finch, Vega, Kolody, 2001). Even though some of these environmental risk factors may be confounded with parental ADHD, there are likely to be some ethnic group differences in exposure to environmental risk factors that are truly environmental in nature.

There may also be some cultural influences on behavior across ethnic groups that influence actual or perceived behavior. Prevalence differences may be explained by variation in parenting styles across cultures in which AA children are encouraged to be more assertive (Garcia Coll, 1990), by differing standards of normalcy across CA and AA samples (Reid, 1995), by the stress caused by living in a chronically stressful environment (Barbarin & Soler, 1993), or by rater biases, particularly between CA teachers and AA students (DuPaul et al., 1997; Epstein, March, Conners, & Jackson, 1998; Reid et al., 1998; Reid et al., 2001). Realistically, there are important cultural differences in the demands of the environment in the homes, schools, and communities of people from different ethnic and cultural groups. These may play a role in what is defined as ADHD in different cultures (Livingston, 1999) and thus affect the way different individuals are diagnosed with the disorder. More specifically, there may be some aspect of AA culture that causes AA individuals to be rated more highly on ADHD rating scales that is not specifically related to the disorder, thus affecting the validity of the diagnosis in AA samples.

Diagnosing ADHD by using rating scales is potentially more problematic with ethnic minority samples for a variety of reasons. First, culturally diverse individuals are frequently not represented in normative groups for rating scales, and thus using these scales with diverse populations must be done with extreme caution (Reid, 1995; DuPaul et al. 1997). In addition, as discussed above, there may be aspects of culture that are confounded with ADHD symptoms and may be misunderstood by raters from different cultures. Reid (1995) outlines requirements for users of diagnostic rating scales: (a) raters should share a common understanding of the attribute to be rated, (b) raters should share a common understanding of the behaviors that are representative of the attribute to be rated, (c) raters must share a common metric in order to accurately scale behaviors relevant to the attribute being rated, and (d) raters should be capable of accurately determined the occurrence and nonoccurrence of behaviors relevant to the attribute being rated. Since ADHD and the rating scales used to rate it stem from white, middle class, and Western values of normality, there are a number of ways that these scales may not be culturally equivalent for people who are culturally and ethnically different (Bauermeister, Berrios, Jimenez, Acevedos, & Gordon, 1990; Reid, 1995).

A halo effect is found when raters rate a child highly on all maladaptive or problematic behaviors even if they have genuinely high ratings on only one domain of problem behavior. Halo effects have been demonstrated with rating scales of ADHD, such that if raters perceive students as demonstrating high rates of oppositional behaviors, they will also rate them highly on symptoms of ADHD (Abikoff, Courtney, Pelham, & Koplewicz, 1993). Thus, if CA teachers tend to perceive AA students as more oppositional or threatening than CA students, halo effects might account for higher ratings of ADHD symptoms in that group. Several researchers have found potential halo effects in AA groups on ADHD rating scales (Reid et al., 1998; Epstein et al. 1998; Sonuga-Barke et al. 1993; Reid et al. 2000; Reid et al., 2001). Although examples of teacher bias against AA children are troubling, it must be remembered that these biases are most frequently not examples of overt racism. Modern social psychological research on interpersonal biases suggests that the majority of ethnicity-related negative judgments or biases are unconscious (e.g., Dovidio & Gaertner, 1998). However, they are real phenomena with real impact on individuals in minority groups, and in the case of ADHD, on diagnostic decision making and treatment planning. Because of halo
effects and other rater bias issues as well as cultural differences, Reid and his colleagues (2000) argue for the use of different norms across ethnicities in order to avoid a “one size fits all” definition of ADHD.

In sum, there is some evidence that the construct of ADHD is the same in AA and CA samples on a broad level, yet it is worrisome that there are higher rates of AA children rated as ADHD compared to CA samples and that the construct may not work the same way across genders within each ethnic group. On the one hand, differences in prevalence based on rating scales may be accounted for by higher levels of bioenvironmental risk and other cultural differences, yet on the other hand, there is also some evidence for rater bias. Examining external correlates should help address these validity concerns.

**External Validity:** Less is known about the external validity of ADHD in AA samples, as most studies have focused on assessment of ADHD as a construct and the use of rating scales. Most of this research on external validity has been conducted in the area of comorbidity. Samuel and her colleagues (1998) found narrower patterns of comorbidity and dysfunction in the AA group with ADHD compared to what is typically found in CA samples. For example, although the findings for mood disorders mirrored typical findings, the rates of other disruptive behavior disorders and anxiety disorders were less frequent than is found in typically defined ADHD samples. In a later family study from their lab, they also found that relatives of AA children with ADHD had fewer instances of bipolar disorder and a much narrower spectrum of anxiety disorders than CA samples typically do (Samuel et al. 1999). In contrast, Epstein and colleagues (1998) found higher rates of Antisocial items in AA boys endorsed by teachers on the Conner’s Teacher Rating Scale. Across both boys and girls, teachers tended to rate AA children higher than CA children on factors relating to externalizing behaviors.

Response to treatment is another important aspect of establishing external validity. Winsberg and Comings (1999) found that only 53% of the AA children in their sample responded to methylphenidate, in comparison to the 70-80% typically found in treatment studies with predominantly CA samples. In contrast, Arnold and colleagues (2003) found that response to treatment between CA and AA samples did not differ after controlling for confounds. In her earlier review of studies on ADHD in AA, Samuel and her colleagues (1997) found that among the published studies at that time, there is evidence that AA children with ADHD who have been treated with methylphenidate may be at higher risk for hypertension.

In sum, the existing research on topics pertaining to external validity of ADHD in AA samples is also mixed. There is some evidence of differences in the range of comorbid disorders found in these samples. In addition, there are mixed findings on differential treatment responses to stimulant medications. Differences in functional impairment have not been a focus of research, and there are no published studies on differential rates of neuropsychological impairments and different profiles in AA compared to CA samples.

**The Current Study:**

Because at this point in time we know a significant amount about ADHD in typically defined samples in comparison to ethnic minority samples, and the scant existing research on ADHD in AA samples is mixed, it is particularly timely to investigate ADHD in an ethnic minority population. In this next section, a brief overview of the goals and hypotheses of the current study will be presented.

**Internal Validity:** One of the first goals is to test for ethnic differences in the internal validity of DSM-IV ADHD. This will be tested by examining overall means on ADHD rating scales, by examining factor structure and subtypes across groups, by examining the diagnostic predictive power of symptoms, and by examining gender differences by group. Overall, if the construct of ADHD works the same in both AA and CA groups, there should be no ethnicity by diagnostic group interactions on any of the variables. In contrast, finding an ethnicity by group interaction would provide evidence that the construct of ADHD differs across ethnic groups.

First, in order to better understand ethnic differences in the manifestation of the disorder, the mean, range, and variance of ADHD symptoms will be compared in both AA and CA samples. Based on previous research, we expect the means to be higher in the AA samples than the CA samples. To understand this prevalence difference further, mean ratings of ADHD will be examined in both teacher and parent reports. If an ethnicity by group interaction is found such that teachers consistently rate AA children higher than CA children in comparison to parents, this will replicate previous findings and be taken as evidence of rater bias. Since ethnicity information is also available for the majority of teachers of these children, variations in ratings of AA children by teacher ethnicity will also be examined. Rater bias hypotheses would predict that teachers from a different ethnic background would be more likely to perceive behavior as threatening than teachers from the same ethnic background.
Second, the internal validity of symptoms dimensions and DSM-IV subtypes will be compared between AA and CA subjects. Exploratory and then confirmatory factor analyses will be conducted in order to examine whether the two factor structure of ADHD fits an AA sample. Because previous research has found that the two factor structure also best explains the data in an AA sample, we expect to replicate that finding in this sample.

Third, subtypes will be analyzed to determine whether there are prevalence differences in DSM-IV subtypes as a function of ethnicity. If a halo effect has occurred and AA children are viewed as more oppositional by CA teachers, or if cultural factors such as parenting variations are encouraging more assertive behavior in AA children, we might expect to find higher rates of hyperactive/impulsive and combined subtypes in AA samples according. Examining teacher versus parent ratings can help address the question of bias. If we find that teachers are more likely to rate AA children highly on hyperactive/impulsive symptoms than parents, this could be evidence of bias. We would expect to find the same proportions of subtypes in both AA and CA samples if the construct operates the same across ethnicities, and there are no issues of bias or cultural influences in expression of behavior.

Fourth, diagnostic predictive validity will also be examined for each individual symptom in order to test for ethnic differences in the predictive power of that symptom. The positive predictive power of a symptom is the probability that the child has that disorder if the symptom is present, and the negative predictive power is the probability of the disorder being absent given that the symptom is not present. If constructs are similar across groups, we should also expect each individual symptom to work similarly across groups. If there are ethnic differences in the predictive power of the individual symptoms across groups, this suggests the construct does not work identically across groups.

Finally, we will test to see if there are gender differences as a function of ethnicity. If we find main effects of both gender and ethnicity, we could conclude that the construct is the same across groups. However, a gender by ethnicity interaction would suggest that the construct differs across groups.

**External Validity:** The second goal of the current study is to test for ethnic differences in the external validity of DSM-IV ADHD. Because there is very little work in this area, these findings are expected to help clarify the mixed results that have been found when looking at issues of internal validity. All of the following analyses will be conducted using a 2 (ethnicity) by 2 (diagnostic group) analysis of variance (ANOVA).

First, the current study will examine the rates of comorbid psychiatric disorders and determine whether these differ across ethnic groups. Based on previous research, it is expected that we will find similar levels of mood disorders, but fewer anxiety disorders in the AA group with ADHD. Previous research is mixed about comorbidity with other externalizing disorders, with some studies finding evidence of higher ratings on ODD/CD in AA samples and others finding lower ratings of disruptive behavior disorders. Higher rates of ODD/CD in this sample would replicate similar findings in predominantly CA samples. A disorder by ethnicity interaction on any of these comorbid symptom dimensions would suggest that the construct does not work similarly across ethnic groups.

Additionally, information about functional impairment, including academic achievement and neuropsychological deficits, will be collected. There are no published studies about functional impairment and neuropsychological profiles in AA, so we propose several hypotheses about these findings. If rater bias is operating and there are a greater percentage of Type 1 errors when diagnosing ADHD in AA individuals, we would expect to find less severe academic and neuropsychological impairments. If the severity of academic and neuropsychological deficits is the same across ethnic groups, it would indicate that ADHD is similar across ethnicities. Again, a deficit by ethnicity interaction will be taken as evidence that the construct is not operating the same across ethnic groups.

Groups will be carefully matched in age, gender, and SES in order to avoid any possible confounds. IQ is a consistent correlate of both ADHD and ethnicity, although the reason for differences in scores as a function of ethnicity is a subject of heated debate (Rushton & Jensen, 2005; Suzuki & Aronson, 2005). As a result, analyses will be conducted both with and without covarying IQ to avoid being overly conservative. Additionally, because there are two samples of participants included in this study, we can potentially replicate the findings in a second sample.

**(III) Research Design and Methods**
Participants: Analyses will be conducted on two separate samples of participants. Within each sample, there will be four groups of participants: AA with ADHD, AA without ADHD, CA with ADHD, and CA without ADHD. The groups will be matched on age, SES, and gender ratio within each sample.

Data for Sample 1 will be taken from a group of subjects who are being recruited as part of a project testing the validity of DSM-IV subtypes of ADHD in a community sample. In collaboration with the Denver Public Schools, parent and teacher ratings of academic and behavioral functioning are currently being obtained on approximately 11,000 children in the Denver metropolitan area. The gender ratio in this study is about 1:1, and the ethnicity of this sample is approximately 50% non-Hispanic CA children, 22% African American children, 24% Hispanic children, 3% Asian/Pacific Islander children, and 1% Native American children. For the current analyses, data from ### AA individuals and ### CA individuals will be included in the analyses. As the data collection is ongoing for this project, we are expected to obtain approximately ## more AA and ## more CA within two years.

Data for Sample 2 will be taken from a larger dataset collected as part of the Colorado Learning Disabilities Research Center (CLDRC) twin study, and ongoing study of the etiology of learning disabilities, ADHD, and other related disorders (e.g., DeFries et al., 1997; Willcutt, DeFries, et al., 2003). In collaboration with 27 school districts in the Denver/Boulder metropolitan area, parents of all twins between the ages of 8 and 18 were contacted by letter and invited to participate in the study. After initial parental consent was obtained, two parallel recruitment procedures were conducted independently to identify twin pairs in which at least one of the twins met criteria for ADHD or at least one of the twins exhibited significant reading difficulties, as well as a comparison sample of twin pairs in which neither twin exhibited either ADHD or RD. For the current analyses, only one member of a twin pair will be chosen to participate. If only one child of the pair meets criteria for ADHD, that child will be chosen. If both children meet criteria for the disorder, the participant will be picked at random. There are currently 38 AA participants in the sample, who will be matched to the same number of CA participants. Of those, 29 have been diagnosed with ADHD.

Although direct comparisons between the two samples have not been made, the CLDRC dataset includes fewer ethnic minorities, involves twins, and likely includes children with somewhat higher SES than the community sample described above. By including two samples, we can examine which findings replicate across samples and which do not.

Measures:

**Assessment of ADHD:** The *Disruptive Behavior Rating Scale* (DBRS; Barkley & Murphy, 1998) was used in both samples to obtain parent and teacher ratings of the 18 symptoms of DSM-IV ADHD. Each symptom on the DBRS is rated on a four-point scale (*never or rarely, sometimes, often, and very often*). Items rated as *often or very often* were scored as positive symptoms and items rated as *never or rarely or sometimes* were scored as negative symptoms, consistent with the procedure used in previous studies of similar rating scales (e.g., Pelham, Gnagy, Greenslade, & Milich, 1992). Previous results from this sample and others indicate that parent and teacher ratings on the DBRS or other similar scales are internally consistent ($\alpha = .92 - .96$) and have adequate high test-retest reliability ($r = .59 - .89$; e.g., DuPaul, Power, Anastopoulos, & Reid, 1998).

The algorithm from the DSM-IV field trials for the disruptive behavior disorders will be used to combine parent and teacher ratings of ADHD symptoms (Lahey et al., 1994). This procedure codes each symptom as positive if it is endorsed by either the parent or the teacher. Consistent with DSM-IV criteria, children were categorized as ADHD only if symptoms were present prior to age seven and if these symptoms caused significant functional impairment. Individuals with six or more symptoms of inattention but fewer than six symptoms of hyperactivity-impulsivity were identified as predominantly inattentive type, participants with six or more symptoms of hyperactivity-impulsivity but fewer than six symptoms of inattention were categorized as predominantly hyperactive/impulsive type, and individuals with six or more symptoms on both dimensions were coded as combined type.

In addition to the DBRS, in Sample 1 the ADHD module from the DSM-IV version of the *Diagnostic Interview for Children and Adolescents* (DICA-IV; Reich, Welner, & Herjanić, 1997) was administered via computer to the mother or father of each participant, in order to provide confirmation of diagnosis and age of onset of each symptom.

Finally, the *Hillside Behavior Rating Scale* (Gittelman & Klein, 1985) was completed by the examiner after the testing session in both samples in order to provide information about the symptoms of ADHD during the session. A recent study showed that these ratings are reliable, are significantly
associated with parent and teacher ratings of ADHD, and provide a significant increment in the prediction of functional impairment over parent and teacher reports alone (Willcutt, Hartung, et al., 1999).

**Functional Impairment:** The DSM-IV criteria for ADHD specify that symptoms of ADHD must interfere with adaptive functioning in two or more domains. Thus, reliable and valid measures of impairment are essential in order to adequately assess ADHD in this population, for whom making diagnoses based solely on the symptom counts must be considered with caution. Several measures of functional impairment are administered as part of both the community and the twin study.

First, objective measures of academic functioning in math and reading have been administered to participants in both samples. In Sample 1, the Wechsler Individual Achievement Test (WIAT; The Psychological Corporation, 1992) was used to assess academic achievement. In Sample 2, the Peabody Individual Achievement Test (PIAT; Dunn & Markwardt, 1970) was administered to assess reading and mathematics achievement. Second, rating scales have been administered to parents and teachers in both samples to assess academic impairment. The DBRS includes several items that assess impairment by asking teachers to rate the extent to which the child’s ADHD behaviors interfere with peer relationships, interactions with adults, educational activities, and management of daily responsibilities. In addition, teachers complete the Teacher Assessment of Social Behavior (TASB; Cassidy & Asher, 1992), a rating scale which assesses the child’s current functioning four dimensions (prosocial, shy/withdrawn, disruptive, and aggressive). Parents also complete the DBRS Academic Impairment scale. In addition, parents provide a single rating of global impairment on the nonclinician version of the Child Global Assessment Scale (CGAS; Setterberg et al., 1992). Finally, participants complete a self-report instrument designed to assess their ability to make and keep friends and the extent they feel left out of peer activities (Cassidy & Asher, 1992).

As part of the preliminary data analyses, objective measure of academic achievement will be correlated with rating scales as an additional measure of rater bias. **Demographic Variables:** Ethnicity was assessed by parental self report in both samples. Parents were given a choice of non-Hispanic CA, African American, Hispanic, Asian/Pacific Islander, Native American, and “other.” In both samples, SES was assessed using parental report of their highest level of academic achievement and by using the Hollingshead 2 Factor Inventory (Hollingshead, 1975).

**Measures of Comorbid Psychopathology:** Since one primary goal of the current study is to compare and contrast the rates of comorbidity with other psychiatric disorders in AA as compared to CA samples, parent and teacher ratings of comorbid symptoms will be included in the analyses.

In Sample 1, parents and teachers rate symptoms of ODD and CD on the DBRS. In addition, each parent and teacher also completed a Behavioral Assessment System for Children (BASC; Reynolds & Kamphaus, 1992), a standardized measure of psychopathology which assesses a wide range of behavioral and emotional difficulties, such as Anxiety, Depression, Aggressive Behavior, and Delinquent Behavior. The DICA-IV modules for ODD, CD, generalized anxiety disorder, simple phobia, and dysthymia/major depressive disorder were administered to each parent. In addition, children completed a Child Depression Inventory (CDI; Kovacs, 1988) and a Multidimensional Anxiety Scale for Children (MASC; March, 1998) to supplement parent and teacher reports.

In Sample 2, parents and teachers also rate symptoms of ODD and CD on the DBRS. In contrast to Sample 1, however, each parent and teacher also completed a Child Behavior Checklist/Teacher Report Form (CBCL/TRF; Achenbach, ##), a standardized measure of psychopathology which assesses a wide range of behavioral and emotional difficulties, such as Anxiety, Depression, Aggressive Behavior, and Delinquent Behavior. The DICA-IV modules for generalized anxiety disorder, simple phobia, and dysthymia/major depressive disorder were administered to each parent in this sample. Finally, all children completed a Child Depression Inventory (CDI; Kovacs, 1988), and the DICA-IV Self Report modules for Anxiety and Depression to supplement parent and teacher reports.

**IQ:** In Sample 1, each child’s cognitive ability is estimated using the Block Design and Vocabulary subtests from the Wechsler Intelligence Scale for Children- Third Edition (WISC-III; Wechsler, 1991). In Sample 2, each child’s cognitive ability is obtained using the WISC-R (Wechsler, 19##). Unfortunately, since the CLDRC twin study has been ongoing for 20 years, the older version of the WISC has continued to be used in order to allow for comparisons to be made across the entire sample.

**Cognitive Measures:** In both Samples 1 and 2, the following were administered as tests of neurocognitive functioning.
Behavioral Inhibition: The Stopping Task (Logan, Schachar, & Tannock, 1997; Schachar, Mota, Logan, Tannock, & Klim, 2000) is a computerized measure of inhibitory control that is based on the dual-process model of inhibition proposed by Logan and colleagues (Logan, 1994). It yields a Stop Signal Reaction Time (SSRT), an estimate of inhibition speed which will be used as the primary dependent measure of behavioral inhibition. The Gordon Diagnostic System (GDS; Gordon, 1983) is a standardized continuous performance test (CPT) that assesses the ability to sustain attention and inhibit inappropriate responses during an extended visual task. Total commission errors will be used as the primary dependent measure of behavioral inhibition.

Sustained Attention and Vigilance: Sustained attention is measured by the GoRT and standard deviations of RT on the Stopping Task, as well as overall RT on the GDS. Number of omissions on the GDS and percent correct on the Stopping Task will be taken as measures of vigilance.

Processing Speed: The WISC-III Symbol Search task was administered to all participants in Sample 1 and a subset in Sample 2. In addition, all participants from Sample 2 were administered the WISC-R Coding subtest as a measure of processing speed. The Trailmaking test (Reitan & Wolfson, 1985) is included as a measure of processing speed because it assesses both direct processing speed (Trails A) and ability to rapidly shift cognitive set (Trails B). It also correlates highly with WISC-R Coding (Shanahan et al. under review).

Verbal Working Memory: The Sentence Span test, adapted by Siegel and Ryan (1989) from a procedure developed by Daneman and Carpenter (1980), was administered to children in both Sample 1 and Sample 2. The subject is instructed to provide the last word for a set of simple sentences (e.g. "I throw the ball up and then it comes...") and then asked to reproduce the words they provided after all the sentences have been completed. The Sentence Span task (Case, Kurland, & Goldberg, 1982), was also administered to children in both samples. It requires participants to count aloud the number of yellow dots on a series of cards, and then asks them to recall in temporal order the number of dots on each card.

Delay Aversion: Delay Aversion is assessed with the Delay Aversion task (Sonuga-Barke, 1994). In this task, subjects are directed that they can choose either a short (5 second) delay in which they earn 25 cents or a long (30 second) delay in which they earn 50 cents. They have 20 trials irrespective of which delay they choose each time.

Planned Data Analyses:

Preliminary Analyses: Preliminary analyses will be conducted to ensure that all 4 groups match on SES, gender, and age within each sample. The two samples will also be compared on the same variables.

Main Analyses: The following analyses will be conducted with both samples 1 and 2.

Internal Validity: Descriptive statistics for each group will be computed, including mean, standard deviation, variance, and range, along with reliability coefficients (Chronbach’s alpha) for the two symptom domains and the total score. Analysis of variance (ANOVA) will be conducted in order to compare the mean score for each symptom, each dimension, and the total number of ADHD symptoms across AA and CA samples. To examine subtype differences, mean numbers of children in each group who meet criteria for each subtype will be compared with a 2 (ethnicity) by 3 (Subtype) ANOVA, and follow-up pairwise comparisons will be conducted to understand significant findings. To test for differences in teacher versus parent ratings, a 2 (rater) by 2 (ethnicity) ANOVA will be conducted on the descriptive statistics of the rating scales. If there is a significant difference between teacher and parent ratings, the results will be broken down further by teacher ethnicity to determine whether variable influences rating scores. To that end, a 2 (teacher ethnicity) by 2 (child ethnicity) ANOVA will be conducted. Differences in Chronbach’s alphas for each symptom dimension and the total score will be compared across ethnicities to determine whether the internal reliability is similar across ethnic groups. To assess the magnitude of differences across gender in each group, effect sizes for each item will be computed for both the CA and the AA group. In addition, a 2 (gender) by 2 (ethnicity) MANOVA will be conducted.

To further examine the internal validity of the symptom dimensions, the 18 symptoms of ADHD will be factor analyzed separately in the AA and CA groups. A principal axis factor (PAF) analysis will be conducted using an oblique rotation, which allows the factors to correlate. This analysis will allow for comparability with previous research on the factor structure of ADHD in AA samples (e.g. DuPaul et al. 1997). Following the exploratory factor analyses, structural equation modeling will be used to perform a confirmatory factor analysis (CFA) to test the results of the PAF solution. An invariance analysis will also be conducted to test that the factor weights are similar across both ethnic groups. A two factor model will be run including both CA and AA participants, and then an additional model will be run on each ethnic group to compare to the first model.
Finally, symptom utility estimates will be conducted across the two ethnic groups. Positive predictive power is the conditional probability that the disorder will be present given the presence of the symptom, and is determined by finding the proportion of individuals with the symptom who have the disorder. Negative predictive power refers to the conditional probability of the disorder being absent given the absence of the symptom, and is the proportion of individuals without the symptom who do not have the disorder. After applying several corrections to these proportions as outlined by Frick and colleagues (1994) the resulting symptom utility estimates provide scores that range from -1 to 1, with zero being the predictive power at chance. Each symptom is expected to predict diagnostic status similarly across groups. Significant variability in these proportions across groups suggests that the symptoms do not operate identically in each group and calls into question the internal validity of the diagnosis.

**External Validity:** The following analyses will be conducted with and without IQ as a covariate. A multivariate repeated measures ANOVA will be conducted using a two (ethnicity) by two (diagnostic group) design. The first will include the academic variables and other ratings of functional impairment as dependent variables, and the second will include the neuropsychological variables. This MANOVA will provide a profile analysis of the functional and neuropsychological variables to determine whether the profiles of the ethnic groups differ on these variables.

**Preliminary Studies:**
Basic stats... compare groups.

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