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Individual Differences in Attention Deficit Hyperactivity Disorder Symptoms and Associated Executive Dysfunction and Traits: Sex, Ethnicity, and Family Income

Michelle M. Martel

University of New Orleans

Department of Psychology, University of Kentucky

Abstract

The goal of the present investigation was to investigate sex, ethnic, and socioeconomic status (SES) influences on attention deficit hyperactivity disorder (ADHD) symptoms and risk markers, including executive dysfunction and temperament traits. Participants were 109 children who were 3 to 6 years old (64% male; 36% ethnic minority) and their primary caregivers and teachers who completed a multistage, multi-informant screening, and diagnostic procedure. Parents completed a diagnostic interview and diagnostic and temperament questionnaires, teachers completed questionnaires, and children completed cognitive control tasks. Because of targeted overrecruitment of clinical cases, 56% of children in the sample were diagnosed with ADHD. Results suggested minimal sex differences, but prominent ethnic differences, in ADHD symptoms and temperament and executive function risk markers. Further, low family income was associated with increased ADHD symptoms and more temperament and executive function risk markers, and low family income explained many ethnic differences in ADHD symptoms and these risk markers. There were prominent interactions among child sex, ethnicity, and family income. Thus, study results suggest that children with multiple individual difference demographic risk factors (e.g., such as being male and ethnic minority) are at highly increased risk of ADHD symptoms and associated risk markers in the temperament and executive function domains.

Keywords

ethnic minority children; attention deficit hyperactivity disorder; ethnicity; socioeconomic status; family income; negative affect; surgency; effortful control; executive function; additive risk

Attention deficit hyperactivity disorder (ADHD) is a highly heterogeneous and impairing behavioral condition characterized by inattention, hyperactivity, and impulsivity that is often first diagnosed early in childhood (American Psychiatric Association [APA], 2000, 2013; Barkley, 2006; Pelham, Foster, & Robb, 2007; Wilens et al., 2002). Individuals with ADHD exhibit substantial individual differences in the behavioral symptoms of ADHD (APA, 2000; Lahey & Willcutt, 2010). They also exhibit substantial variability in risk markers of

executive dysfunction (i.e., problems with problem solving to attain a future goal; Pennington & Ozonoff, 1996) and extreme temperament traits (i.e., constitutionally based, individual differences in reactivity [high negative affect and positive affect] and self-regulation [low effortful control]; Martel, Goth-Owens, Martinez-Torteya, & Nigg, 2010; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Rothbart & Posner, 2006) that are believed to increase risk of ADHD (Barkley, 1997; Martel, 2009; Sonuga-Barke, 2005). Sex, ethnic, and socioeconomic status (SES) differences are potentially important categories of individual differences that remain underexplored in relation to ADHD and its associated risk markers (Gaub & Carlson, 1997; Gershon, 2002; Gingerich, Turnock, Litfin, & Rosen, 1998; Miller, Nigg, & Miller, 2009; Samuel et al., 1997). However, individual differences in ADHD and risk markers based on sex, ethnicity, and SES are likely to be important for providing a complete picture of interindividual heterogeneity within ADHD and for helping to explain heterogeneity in pathways to the disorder.

Work on individual differences in ADHD based on sex, ethnicity, and SES remains limited, and these three classes of individual differences are seldom examined together, the goal of the present investigation. It is well established that ADHD is diagnosed approximately 3 times as often in boys as in girls (APA, 2000; Egger & Angold, 2006; Hartung & Widiger, 1998; Pineda et al., 1999). Further, there has been some suggestion that symptoms of ADHD may vary somewhat between boys and girls, with boys exhibiting higher levels of hyperactivity-impulsivity than girls, although findings for inattention have been mixed (Gaub & Carlson, 1997; Gershon, 2002; Hartung et al., 2002). Further, there are well-established sex differences in the temperament traits of negative affect (i.e., fear, anger, and sadness), surgency (i.e., approach, sociability), and effortful control (i.e., thoughtful, deliberate regulation) in the general population. Negative affect is higher in girls, and positive affect is higher in boys than girls, beginning as early as infancy (Campbell & Eaton, 1999; Garstein & Rothbart, 2003; Zahn-Waxler, Cole, & Barrett, 1991), while effortful control is lower in boys than girls beginning from early childhood (Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006; Kochanska, Murray, & Coy, 1997). It has been suggested that these normative sex differences in traits may help to explain sex differences in ADHD prevalence rates (De Pauw & Mervielde, 2011; Martel, 2009; Tackett, 2006). Notably, no work to date has evaluated whether boys and girls diagnosed with ADHD exhibit these normative sex differences in traits. In contrast, sex differences in executive function in the general population and in children with ADHD seem to be modest to nonexistent (Brocki & Bohlin, 2004; Seidman et al., 2005).

Recent work suggests that there are also important ethnic differences in ADHD symptoms (Gingerich et al., 1998; Samuel et al., 1997). ADHD symptoms seem to be higher in African Americans compared with Caucasians (Cuffe, Moore, & McKeown, 2005; Epstein, March, Conners, & Jackson, 1998; Lee, Oakland, Jackson, & Glutting, 2008; Reid et al., 1998; Reid, Casat, Horton, Anastopoulos, & Temple, 2001; Samuel et al., 1998; reviewed by Miller et al., 2009), as are levels of aggression (McLaughlin, Hilt, & Nolen-Hoeksema, 2007). Although the structure of temperament (and personality) has been well validated and fairly invariant across cultures and ethnic groups in general population samples (Costa, Terracciano, & McCrae, 2001; McCrae et al., 2002), little work has examined the possibility that there might be ethnic differences in temperament traits in children with ADHD. Further,

no work to date has examined ethnic differences in executive function in the general population or in children with ADHD.

In contrast, substantial work supports an association between SES and executive function in the general population. Lower SES is associated with poorer executive function (Noble, McCandliss, & Farah, 2007; Noble, Norman, & Farah, 2005; Sarsour et al., 2011; reviewed in Hackman & Farah, 2009; Hackman, Farah, & Meaney, 2010). However, it remains unclear whether SES may increase ADHD symptoms or lead to more extreme child temperament traits (e.g., lower effortful control, higher negative emotionality).

Importantly, few, if any, studies have examined individual differences in the three domains of sex, ethnicity, and SES together in the same study, the goal of the present investigation. Therefore, the associations among these three types of individual differences remain poorly understood. For example, individual differences such as child sex, ethnicity, and SES may all be important for understanding psychopathology and its risk markers via a cumulative, or additive, risk model, wherein having more risk factors increases risk in an additive, linear fashion (Appleyard, Egeland, van Dulmen, & Sroufe, 2005; Biederman et al., 1995; Rutter, 1979; Sameroff, 2000). In contrast, individual differences such as ethnicity and SES might be conflated, as ethnic minorities in the United States are more likely to grow up in poverty (Barbarin & Soler, 1993; Garcia Coll et al., 1996). Thus, ethnic differences in symptoms or risk markers might be better explained by SES differences. Finally, individual differences in sex, ethnicity, and SES might interact with one another such that males who are ethnic minority who are raised in low SES environments may exhibit differentially worse outcomes than male Caucasians, female African Americans, or those brought up in higher SES environments (Barbarin, 1993; Garcia Coll et al., 1996; Lengua, 2002).

It is critically important to tease apart these three possibilities, as they have very different applied implications. The current study examined sex differences, ethnic differences, and SES-related differences in ADHD symptoms and its associated risk markers to evaluate additive, conflation, and interactive models of risk. First, sex, ethnic, and SES differences in ADHD symptoms and risk markers were evaluated. Study hypotheses were that sex, ethnic, and SES differences would be apparent in child ADHD symptoms and risk markers such that males, ethnic minorities, and children from low SES families would exhibit increased ADHD symptoms, more impaired executive function, and more extreme temperament traits (i.e., lower effortful control and higher negative emotionality and surgency). Second, additive, conflation, and interactive models of sex, ethnic, and SES effects on ADHD symptoms and its mechanisms were examined. It was hypothesized that sex, ethnicity, and SES would interactively relate to ADHD symptoms and its risk markers such that African American males growing up in poverty would exhibit a multiplicative increase in ADHD symptoms, executive dysfunction, and extreme levels of traits.

Method

Participants

Overview—Participants were 109 preschoolers between the ages of 3 and 6 ($M = 4.34$ years, $SD = 1.08$) and their primary caregivers (hereafter termed parents for simplicity; 67%

mothers with the remaining 33% fathers + mothers together, fathers only, foster parents, or grandmothers with guardianship). As shown in Table 1, 64% of the sample was male and 36% of the sample was ethnic minority (28% African American and 8% other, including Latino, American Indian, and mixed race children). Family income ranged from below \$20,000 to above \$100,000 annually (coded on a scale: 0 = annual income less than \$20,000, 1 = between \$20,000 and \$40,000, 2 = between \$40,000 and \$60,000, 3 = between \$60,000 and \$80,000, 4 = between \$80,000 and \$100,000, and 5 = over \$100,000 annually; sample mode = 3). Parental highest educational level ranged from grade school to doctorate (coded on a scale: 0 for grade school, 1 for some high school, 2 for high school equivalent, 3 for high school degree, 4 for some college, 5 for associate's degree, 6 for bachelor's degree, 7 for master's or equivalent degree, and 8 for doctorate; sample mode = 4), and family employment ranged from unemployed to full-time weekly (coded on a scale: 0 for unemployed, 1 for 1–19 hr part-time weekly, 2 for 20–39 hr part-time weekly, and 3 for full-time weekly; sample mode = 3). Based on current recommendations (APA, 2007), in the current study, family SES was conceptualized as multidimensional and included the aforementioned indices of family income, parental education, and parental occupation. Family income was emphasized in analyses because it was viewed as encompassing the other two domains, but follow-up analyses examined the influence of parental education and occupation. Further, for models evaluating additive or cumulative risk, risk was summed across the dichotomous variables of child sex (risk = male), child ethnicity (risk = African American), and family income (risk = household income less than \$40,000 per year).

Based on multistage and comprehensive diagnostic screening procedures (detailed later), preschoolers were recruited into two groups: disruptive behavior disordered children (DBD; $n = 79$), subdivided into ADHD only ($n = 18$), oppositional defiant disorder (ODD) only ($n = 18$), and ADHD + ODD ($n = 43$), and non-DBD children ($n = 30$). The non-DBD group included preschoolers with subthreshold symptoms to provide a more continuous measure of ADHD symptoms, consistent with research suggesting that ADHD and ODD may be better captured by continuous dimensions than categorical diagnosis (Haslam et al., 2006; Levy, Hay, McStephen, Wood, & Waldman, 1997). No siblings were included.

Recruitment and identification—Participants were recruited from the community primarily through direct mailings to families with children between the ages of 3 and 6 from the greater New Orleans area and Internet postings, as well as through advertisements in newspapers and flyers posted at doctors' offices, community centers, daycares, and on campus bulletin boards. Two sets of advertisements were utilized; one set of advertisements targeted children between ages 3 and 6 with disruptive behavior problems or attention problems and a second set of advertisements targeted children between ages 3 and 6 without these types of problems. After recruitment, families passed through a multistaged screening process. An initial telephone screening was conducted to rule out children prescribed psychotropic medication or children with neurological impairments, mental retardation, psychosis, autism spectrum disorders, seizure history, head injury with loss of consciousness, or other major medical conditions. Only 10 families were screened out at this phase. All families screened into the study at this point completed written and verbal

informed consent procedures consistent with the Institutional Review Board, the National Institute of Mental Health, and American Psychological Association guidelines.

During the second stage, parents and preschoolers made a campus laboratory visit. Parents of children taking psychostimulant medication were asked to consult with a physician about discontinuing children's medication for 24 to 48 hr prior to the visit depending on their dosage and type of medication to ensure a more accurate measure of cognitive performance (less than 5% of children in the study were prescribed medication for attention problems). Before and during the laboratory visit, diagnostic information was collected via parent and teacher or caregiver ratings. Parents completed the Kiddie Disruptive Behavior Disorders Schedule (K-DBDS; Leblanc et al., 2008), a semistructured diagnostic interview modeled after the Schedule for Affective Disorders and Schizophrenia for School-Age Children (Orvaschel & Puig-Antich, 1995) administered by a trained graduate student clinician. Questions about endorsed DBD symptoms were followed by questions that determine symptom severity, duration, onset, and cross-situational pervasiveness. For endorsed symptoms to count toward diagnosis, the symptom must have been present in more than one setting (i.e., school, home, or public) and must have occurred frequently compared with same-aged peers. The K-DBDS demonstrates high test-retest reliability and high interrater reliability in the preschool population (Leblanc et al., 2008). In the current study, fidelity to interview procedure was determined by calculation of reliability of blind interviewer ratings of DBD symptoms on a randomly chosen 10% of families. Interrater clinician agreement was adequate for ODD and ADHD symptoms ($r = .99, p < .001, r = 1.00, p < .001$, respectively).

Families were mailed teacher or caregiver questionnaires 1 week prior to the laboratory visit and instructed to provide the questionnaires to children's teacher, day-care provider, or babysitters who then mailed the completed questionnaires back to the university. When available (i.e., available on 50% of participating families), teacher or caregiver report on DBD symptoms was obtained via report on the Disruptive Behavior Rating Scale (DBRS; Barkley & Murphy, 2006). In the current study, approximately 67% of completed teacher or caregiver reports were provided by teachers, with most of the remaining questionnaires completed by day-care providers or babysitters. Some families did not have teacher or caregiver reports available because they could not identify a second reporter; however, in most cases of missing data, teachers or caregivers did not return the questionnaire measures. The response rate did not differ based on child DBD diagnostic group, $\chi^2(3) = .59, p = .9$.

Ultimately, clinical diagnoses were determined by the principal investigator, a licensed clinical psychologist, after a review of parent ratings on the K-DBDS and (when available) teacher or caregiver ratings on the DBRS, consistent with current best practice guidelines for current diagnosis (Pelham, Fabiano, & Massetti, 2005).

Measures

Symptom counts—Parent and teacher or caregiver reports on symptoms were available via the DBRS (Barkley & Murphy, 2006), which assesses symptoms using a 0 to 3 scale for a more continuous dimension. Endorsed symptoms are summed within each diagnostic subdomain (i.e., ODD symptoms, ADHD total symptoms [inattentive + hyperactive–

impulsive], inattentive ADHD symptoms, hyperactive–impulsive ADHD symptoms) to determine symptom counts within these categories. The DBRS has high internal consistency, ranging from .78 to .96 in the preschool age range (Pelletier, Collett, Gimple, & Cowley, 2006). All scales for parent and teacher or caregiver report on the DBRS had high internal reliability (all $\alpha > .92$) in the current sample. Primary analyses were conducted using parent report on the DBRS with secondary checks conducted on teacher report on the DBRS.

Temperament traits—To measure temperament traits, parents completed the very short form of the Child Behavior Questionnaire (CBQ; Putnam & Rothbart, 2006; Rothbart, Ahadi, Hershey, & Fisher, 2001). The very short form of the CBQ contains 36 descriptive statements that parents rate on a 1 (*extremely untrue of your child*) to 7 (*extremely true of your child*) scale. Negative affect (i.e., fear, anger, sadness), surgency (i.e., approach, sociability), and effortful control (i.e., deliberate, thoughtful regulation) were measured using the scales suggested by Rothbart et al. (2001). Composite scores were generated by reverse-scoring selected items and computing the average. The scales had acceptable internal reliability coefficients of .70 or above in the current sample.

Cognitive control: Response inhibition, set-shifting, and working memory—Selected cognitive control tasks assessing response inhibition, set-shifting, and working memory were utilized, based on their validity and sensitivity in the preschool population (Garon, Bryson, & Smith, 2008; Wiebe, Espy, & Charak, 2008). To measure response inhibition, the Shape School was used. The Shape School uses a bright, large storybook format with stimulus figures (i.e., colored circles and squares with happy and sad facial expressions, some of which wear hats; Espy, Bull, Martin, & Stroup, 2006). The second condition (of four conditions) is of most interest. It provides the most direct measure of response inhibition because it entails having the child name the colors of happy-faced figures while being asked to inhibit naming of the colors of sad-faced figures. Number of correctly identified stimuli divided by time to complete the second trial serves as a measure of response inhibition. This task has adequate reliability, with test–retest reliability ranging from .65 to .78 and alpha coefficients exceeding .71 for the second condition within this age range (Isquith, Crawford, Espy, & Gioia, 2005).

To assess set-shifting, an adaptation of the Trail-Making Task (TRAILS-P; Espy & Cwik, 2004) was administered. Condition B provides a measure of set-shifting. During condition B, the switching condition, children are asked to stamp dogs and bones in order of size, alternating between the two. Time to complete condition B served as a measure of set-shifting. This task has good test–retest reliability across a 2-week period, with mean correlations of .64 between conditions within this age range (Isquith et al., 2005). Backward Digit Span served as a measure of simple and complex working memory. In Digit Span backward condition, the child is instructed to repeat a series of numbers in the reverse order heard. The total of correctly completed items for this condition provided a measure of working memory (Garon et al., 2008).

Data Analysis

Univariate and multivariate analysis of variance (ANOVA, MANOVA) in SPSS were utilized to evaluate diagnostic, sex, and ethnic group differences in parent- and teacher-rated symptoms, parent-rated temperament traits, and laboratory performance on executive function tasks. Nonparametric correlations were conducted between family income and child symptoms, traits, and executive function with secondary analyses examining correlations between parental education and parental occupation and child characteristics. Family income was emphasized in the current study based on recommendations against use of a merged SES composite, which might obscure differences between various SES characteristics (APA, 2007) and to cut down on the number of conducted statistical tests and corresponding Type I error.

Finally, additive risk models were evaluated via nonparametric correlations among the child risk composite and parent- and teacher-rated symptoms, parent-rated temperament traits, and laboratory performance on executive function tasks, as recommended (Appleyard et al., 2005; Rutter, 1979). Interactive risk models were evaluated via MANOVA with the examination of interaction terms child sex \times child ethnicity, child ethnicity \times family income, and child sex \times family income.

Results

As shown in Table 1, child age and sex did not significantly differ between diagnostic groups (all $p > .1$). However, child ethnicity and family income significantly differed between the four diagnostic groups ($p < .05$). Specifically, there were more ethnic minorities within the ADHD and ADHD + ODD diagnostic groups compared with the ODD and control groups. Because the largest category of ethnic minority in the present sample was African American (28% vs. 8% Latino/American Indian and mixed), all subsequent analyses examining ethnic differences will focus on comparisons between Caucasians and African Americans. Family income was lower in the ADHD and ODD + ADHD groups.

DBD symptoms differed significantly in the expected direction between diagnostic groups. For example, preschoolers in the DBD groups exhibited higher DBD symptoms compared with preschoolers without DBD based on parent- and teacher-reported symptoms, and preschoolers in the ADHD, ODD, and comorbid ODD + ADHD groups exhibited higher symptoms in the relevant domains compared with other groups (e.g., children with ODD and ODD + ADHD exhibited higher ODD symptoms compared with the ADHD or non-DBD groups [all $p < .05$; see Table 1 for mean differences]). Bivariate correlations were also conducted to examine associations between parent- and teacher-reported child DBD symptoms (not shown). Bivariate correlations of parent and teacher DBD symptom ratings within domain were all significant and at least in the moderate range (r range from .52 to .59, all $p < .01$).

As shown in Table 1, temperament traits, but not executive function, also significantly differed between the diagnostic groups. Negative affect and surgency were significantly higher in the ODD + ADHD groups compared with the other groups, and effortful control was lower in the ODD + ADHD group than the other groups (all $p < .05$).

Sex and Ethnic Differences in Child Symptoms and Risk Markers

As shown in Table 2, based on a series of MANOVAs, there were no significant sex differences in child symptoms, temperament traits, or executive function (all $p > .1$). However, as shown in Table 3, there were significant ethnic differences in parent- and teacher-rated inattention and hyperactivity–impulsivity, $F(1) = 7.73, p = .007$; $F(1) = 5.07, p = .03$, for parent ratings; $F(1) = 9.02, p = .004$; $F(1) = 4.32, p = .04$ for teacher ratings, such that African Americans were rated as having higher inattention and hyperactivity–impulsivity than Caucasians. Further, negative affect and set-shifting significantly differed between African Americans and Caucasians, such that negative affect was higher and set-shifting worse in African Americans, compared with Caucasians, $F(1) = 3.76, p = .05$; $F(1) = 5.1, p = .03$, respectively.

Associations Between Family Income and Child Symptoms and Risk Markers

As shown in Table 4, on the basis of nonparametric correlations, lower family income was significantly correlated with increased parent-rated child ADHD symptoms (both inattention and hyperactivity–impulsivity; $r = -.2, -.27$, respectively; both $p < .05$), but not teacher-rated symptoms (all $p > .1$). Further, lower family income was significantly associated with greater child negative affect ($r = -.29, p < .05$) and worse working memory ($r = .25, p < .05$). Secondary examination of maternal and paternal education and employment was similar to findings for family income, with maternal education and paternal employment seeming to exhibit particularly powerful associations with ADHD symptoms and risk markers (also shown in Table 4).

Conflation of Ethnicity and Family Income

When family income, was entered as a covariate in the series of MANOVAs evaluating ethnic differences in ADHD symptoms and risk factors, all previously identified significant ethnic differences in child symptoms or risk markers become nonsignificant (dropped to $p > .1$), except for teacher-rated inattentive symptoms ($p = .04$). Thus, these results are not consistent with an additive model of risk, but rather suggest that ethnic differences in ADHD symptoms and risk markers appear better explained by low family income with the exception of teacher-rated inattentive symptoms.

Additive Effects of Sex, Ethnicity, and Family Income

Next, an additive risk model was evaluated using nonparametric correlations between the composite risk variables (i.e., a summation of risk across child sex [male], ethnicity [African American], and family income [less than \$40,000 per year]). As shown in Table 5, the cumulative risk composite was significantly associated with parent-rated hyperactivity–impulsivity and negative affect.

Interactive Effects of Child Sex, Child Ethnicity, and Family Income

Next, interactions among child sex, child ethnicity, and family income in relation to ADHD symptoms and risk factors were tested using MANOVA. There were no significant three-way interactions (all $p > .1$). However, there were several significant two-way interactions. As shown in Table 3, there was a significant interaction between child sex and child

ethnicity in relation to teacher-rated inattentive symptoms, $F(1) = 3.32, p = .046$. Although there was little evidence of sex differences in inattentive symptoms in Caucasians, there was a substantial sex difference in inattentive symptoms in African Americans with African American males exhibiting more inattentive symptoms compared with females.

As shown in Table 4, there was a significant interaction between family income and sex for parent-rated child inattentive symptoms, $F(5) = 3.16, p = .01$; the association between lower family income and increased inattention was stronger for females ($r = -.37, p < .05$ for females compared to $r = -.08, p > .1$ for males; significant difference test $z = -2.24, p = .01$). In addition, there was a significant interaction between family income and ethnicity in relation to child negative affect, $F(5) = 2.26, p = .05$. The association between lower family income and increased negative affect was stronger for African Americans than Caucasians ($r = -.54, p < .01$ for African Americans compared to $r = -.09, p > .1$ for Caucasians; significant difference test $z = -3.74, p = .0001$).

Thus, an interactive model of risk seems to best explain associations among child sex, ethnicity, and family income in relation to ADHD inattention, whereas an additive risk model seemed to best explain hyperactivity–impulsivity. Further, there were both additive and interactive effects on negative affect.

Discussion

This study largely replicates prior work regarding sex, ethnic, and SES differences in ADHD symptoms while extending prior work by simultaneous examination of the three domains in relation to ADHD symptoms and risk markers. Although sex differences in this study were minimal, ethnic differences were striking. African Americans exhibited increased parent- and teacher-rated ADHD symptoms in the domains of inattention and hyperactivity–impulsivity as well as increased negative affect and decreased set-shifting. Family income explained, or accounted for, many of the ethnic differences seen (with the exception of ethnic differences in teacher-rated inattention). Lower family income was associated with worse executive function, higher negative affect, and increased inattentive and hyperactive–impulsive ADHD symptoms. Finally, interactions among child sex, ethnicity, and family income were notable in relation to inattention, whereas additive effects seemed to operate in relation to hyperactivity–impulsivity. In contrast, both additive and interactive effects were important for negative affect. Each of these findings will be discussed in turn.

In contrast to prior work finding sex differences in ADHD symptoms (i.e., Gaub & Carlson, 1997; Gershon, 2002), there were no significant sex differences in ADHD symptoms in the present study. Temperamental effortful control was marginally lower in boys, compared with girls, in line with prior work (Else-Quest et al., 2006; Kochanska et al., 1997). No sex differences were apparent in executive function, also in line with prior work (Brocki & Bohlin, 2004; Seidman et al., 2005). Differences between the current study's findings and prior study findings might be because of a relative lack of power resulting from a relatively modest sample size (although power analyses suggested adequate power [.80] to detect medium size effects) or, perhaps more likely, because of the nature of the sample recruited. Overrecruitment of clinical cases may decrease the power to detect sex differences by

truncating the range of symptoms, executive function, and temperament traits. Further, if executive function and temperament traits do, in fact, increase risk of ADHD, one might not expect to see sex differences in these risk factors in a sample of children with ADHD.

However, ethnic differences in ADHD symptoms and the risk markers of executive dysfunction and temperament traits were notable. African Americans exhibited increased inattentive and hyperactive-impulsive ADHD symptoms compared with Caucasians (Cuffe et al., 2005; Epstein et al., 1998; Lee et al., 2008; Reid et al., 1998, 2001; Samuel et al., 1998; reviewed by Miller et al., 2009). African Americans were rated as exhibiting increased ADHD symptomatology whether rated by parents or teachers. They further exhibited increased negative affect and decreased set-shifting compared with Caucasians, suggesting possible ethnic differences in within-child risk markers that might account for ethnic differences in symptoms. Thus, African Americans may be at increased risk of ADHD because of their predisposing set-shifting problems and high negative affect (De Pauw & Mervielde, 2011; Martel, 2009; Tackett, 2006). These problems may also explain African Americans' increased risk of aggression and academic problems (Barbarin & Soler, 1993) because negative affect seems to increase risk of reactive aggression (Fite, Stoppelbein, & Greening, 2009; Shields & Cicchetti, 1998), and poor executive function seems to increase risk of academic problems (Bull, Espy, & Wiebe, 2008; Diamantopoulou, Rydell, Thorell, & Bohlin, 2007; Espy et al., 2004).

Socioeconomic status factors such as family income explained many ethnic differences in ADHD symptoms and risk markers. In the United States, ethnic minorities such as African Americans tend to have a lower family income compared with the ethnic majority (Barbarin & Soler, 1993; Garcia Coll et al., 1996). Thus, SES, which is highly conflated with ethnic minority status in the United States, may explain many ethnic differences in symptomatology and risk markers, perhaps directly and indirectly via economic stress (Hudson, 2005). This is in line with ADHD treatment outcome work that found that SES moderated treatment effects, but ethnicity did not (Arnold et al., 2003; Rieppi et al., 2002). However, it should be noted that SES did not explain ethnic differences in teacher-rated attention, possibly suggesting that there are risk factors other than low SES that are increasing inattention in African Americans.

Importantly, ethnic minority status interacted with child sex and family income to particularly magnify risk of ADHD symptoms of inattention. Child ethnicity and sex interacted in relation to teacher-rated ADHD symptoms; the expected sex differences in ADHD symptoms (boys > girls) were strikingly larger for African Americans than for Caucasians. That is, African Americans, particularly male African Americans, appeared particularly at risk for high ADHD symptoms.

In addition, family income interacted with child sex in relation to inattention; lower family income was associated with increased inattention, particularly for girls. That is, girls from families with low income appeared to exhibit increased risk of inattention. Girls in stressed environments may be particularly vulnerable to psychopathology via sensitivity to environment contexts influenced by family income. In line with this idea, some work has suggested that girls are more sensitive to interpersonal stressors such as parental sensitivity

and responsivity and conflict that may themselves be highly associated with family income (Davies & Lindsay, 2004; Shih, Eberhart, Hammen, & Brennan, 2006; Zahn-Waxler, Usher, Suomi, & Cole, 2005).

Finally, interactive and additive effects seemed to operate in relation to negative affect. Family income interacted with ethnicity in relation to negative affect such that lower family income was associated with increased negative affect, particularly for African Americans. Thus, African Americans from families with lower family income were at increased risk of high negative affect. African Americans may have increased risk of ADHD symptoms via trait risk markers like negative affect. Negative affect may make them differentially susceptible to negative environmental conditions, including dangerous neighborhoods, poor parental monitoring, and deviant peer influences (Belsky, Hsieh, & Crnic, 1998; Belsky & Pluess, 2009). Further, more risk factors seemed to increase risk for negative affect. Likewise, additive influences also influenced hyperactivity–impulsivity. Those children with more risk factors exhibited increased hyperactivity–impulsivity. Interactive influences on inattention and additive influences on hyperactivity–impulsivity suggest that there may be different pathways to the ADHD symptom domains, a finding in line with recent dual- and multiple-pathway models to ADHD (Nigg, Goldsmith, & Sachek, 2004; Sonuga-Barke, 2005; Sonuga-Barke, Bitsakou, & Thompson, 2010). Whereas risk factors for inattention may rapidly multiply in their effects, risk of hyperactivity–impulsivity may increase in a slower, more gradual fashion.

Thus, overall, the current study suggests that child sex, ethnicity, and family income have important influences on ADHD symptoms and associated risk markers in both additive and interactive fashions, with family income appearing to play a particularly important role in explaining individual differences in ADHD and its risk markers. Thus, interventions might be able to be personalized toward select groups of children. For example, ethnic minority boys, particularly those from low income families, might merit special intervention targeted at decreasing negative affect or increasing emotion regulation. Likewise, girls from low SES families might particularly benefit from interventions targeting inattention. Of course, more work addressing mechanisms of these effects (e.g., the parental environment, peer influences) is needed.

The current study is not without its limitations. For example, the present study cannot rule out rating bias. Limited work has addressed the possibility that raters might differentially endorse ADHD symptoms in children based on their ethnicity, and the work that has addressed this possibility has been mixed (for positive findings on bias: Hillemeier, Foster, Heinrichs, Heier, & the Conduct Problems Prevention Group, 2007; Mann et al., 1992; Reid et al., 2000; for negative findings: Epstein et al., 2005; Hosterman, DuPaul, & Jitendra, 2008). This is an important direction for future work. There are many other domains that sex, ethnicity, and family income might impact that the current study did not examine (e.g., language, academic outcomes), and there are a number of mechanisms by which sex, ethnicity, and family income might exert effects (e.g., family strain, social support, neighborhood environment, parenting, peer deviancy), but that are likely important. Pathways to childhood disorders like ADHD may vary, at least somewhat, based on child sex, ethnicity, or SES, and this will be important to examine in future work. Finally, the

current results need to be replicated in other kinds of samples to evaluate generalizability, in particular as samples recruited via advertisements are likely subject to a self-selection bias.

Overall, child sex, ethnicity, and family income appear important for explaining variability in ADHD symptoms and associated executive dysfunction and temperament traits. SES may be particularly important for explaining variability in early developmental outcomes like ADHD. However, interactions among child and family characteristics such as child sex, ethnicity, and family income are important to consider for explaining why some youth are at particularly increased risk of developmental psychopathology like ADHD.

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Table 1

Demographic and Descriptive Information on the Sample by Clinical Group

<i>M (SD)</i>	Non-DBD (c) <i>n</i> = 30	ODD (o) <i>n</i> = 18	ADHD (a) <i>n</i> = 18	ODD + ADHD (oa) <i>n</i> = 43
Age	3.9 (1.03)	4.56 (1.25)	4.56 (.92)	4.47 (1.05)
Sex (<i>N</i> [% male])	14 (46.7)	10 (55.6)	13 (72.2)	27 (62.8)
Ethnicity (<i>N</i> [% minority])	7 (23.3)	2 (11.2)	10 (55.6)	17 (39.6)*
Caucasian	23 (76.7)	16 (88.9)	8 (44.4)	26 (60.5)
African American	7 (23.3)	0 (0)	9 (50)	12 (27.9)
Latino	0 (0)	0 (0)	1 (5.6)	2 (4.7)
American Indian	0 (0)	1 (5.6)	0 (0)	0 (0)
Mixed	0 (0)	1 (5.6)	0 (0)	3 (7)
Family income (mode)	3	5	1	0*
Maternal education	4	6	4	4
Maternal occupation	0,3 [†]	0,3 [†]	0	3
Paternal education	4	4	4	4
Paternal occupation	3	3	3	3
ODD symptoms (P)	2.97 (3.08) ^{1,2}	10 (6.02) ^{1,3}	5.83 (3.28) ^{3,4}	11.6 (7.24) ^{2,4**} c<o<a<oa
ADHD symptoms (P)	8.6 (6.86) ^{1,2,3}	19.73 (12.98) ^{1,4}	26.72 (9.09) ^{2,5}	35.26 (13.54) ^{3,4,5**} c<o<a<oa
Inattention	3.77 (3.87) ^{1,2,3}	8.93 (6.77) ^{1,4}	11.39 (5.88) ^{2,5}	16 (7.29) ^{3,4,5**} c<o<a<oa
Hyper-Imp	4.83 (3.76) ^{1,2,3}	10.8 (6.62) ^{1,4,5}	15.33 (5.43) ^{2,4,6}	19.26 (7.04) ^{3,5,6**} c<o<a<oa
ODD symptoms (T)	2.77 (3.75) ¹	4 (3.84) ²	4.6 (4.16) ³	11.84 (6.68) ^{1,2,3**} others<oa
ADHD symptoms (T)	10.08 (9.11) ^{1,2}	7.78 (7.97) ^{3,4}	37.6 (10.16) ^{1,3}	36.32 (8.51) ^{2,4**} c,o<a,oa
Inattention	4.15 (4.26) ^{1,2}	3.89 (3.95) ^{3,4}	23.2 (2.95) ^{1,3,4}	17.95 (5.75) ^{2,4**} c,o<a, oa
Hyper-Imp	5.92 (5.59) ^{1,2}	3.89 (4.17) ^{3,4}	14.4 (8.91) ^{1,3}	18.39 (5.41) ^{2,4**} c,o<a,oa
Negative affect	3.45 (.76) ^{1,2,3}	4.18 (.72) ^{1,4}	4.6 (.81) ²	4.71 (.98) ^{3,4**} c<o<a<oa
Surgency	4.5 (.77) ¹	4.49 (.99) ²	4.53 (1.1) ³	5.14 (.94) ^{1,2,3*} oa> all others
Effortful control	5.24 (.76) ¹	4.73 (1)	4.76 (.69)	4.65 (.95) ^{1*} oa<c
Response inhibition	.77 (.31)	.93 (.28)	.69 (.3)	.75 (.4)
Set-shifting	31.48 (28.76)	27.67 (12.15)	38.73 (29.5)	36.55 (28.03)
Working memory	1.84 (2.29)	2.73 (2.76)	1.88 (2.16)	1.43 (2.21)

Note. Subgroup differences based on chi-square or ANOVA with follow-up LSD post hoc tests indicated with like superscripts. Family income modes: 0 = annual income less than \$20,000, 1 = between \$20,000 and \$40,000, 2 = between \$40,000 and \$60,000, 3 = between \$60,000 and \$80,000, 4 = between \$80,000 and \$100,000, and 5 = over \$100,000 annually. Parental education modes: 0 for grade school,

7 for master's or equivalent degree, and 8 for doctorate. Parental employment modes: 0 for unemployed, 1 for 1–19 hr part-time weekly, 2 for 20–39 hr part-time weekly, and 3 for full-time weekly; sample mode = 3. DBD = disruptive behavior disorder; ODD = oppositional defiant disorder; Hyper-Imp = hyperactive-impulsive. (P) = Parent report; (T) = Teacher report. Response inhibition = efficiency score. Set-shifting = Time to complete Trails B. Working memory = Digit Span backward raw score.

¹ for some high school,

² for high school equivalent,

³ for high school degree,

⁴ for some college,

⁵ for associate's degree,

⁶ for bachelor's degree,

* $p < .05$.

** $p < .01$.

† multiple modes.

Table 2

Sex Differences in Child Symptoms and Risk Markers

	Male <i>n</i> = 64	Female <i>n</i> = 45
ODD symptoms (P)	8.02 (6.52)	7.78 (6.83)
ADHD symptoms (P)	24.2 (14.34)	23.64 (17.42)
Inattention (P)	10.23 (6.87)	11.33 (9.17)
Hyper-Imp (P)	13.97 (7.94)	12.31 (9.02)
ODD symptoms (T)	7.97 (7.34)	5.96 (5.7)
ADHD symptoms (T)	24.83 (17.2)	22.09 (15.54)
Inattention (T)	12.26 (8.71)	11.5 (9.51)
Hyper-Imp (T)	12.57 (9.19)	10.61 (7.74)
Negative affect	4.25 (.97)	4.26 (1.06)
Surgency	4.83 (.92)	4.67 (1.03)
Effortful control	4.72 (.81)	5.02 (.96) ⁺
Response inhibition	.81 (.38)	.74 (.28)
Set-shifting	30.11 (22.82)	39.54 (29.59)
Working memory	2.11 (2.42)	1.53 (2.21)

Note. Subgroup differences based on ANOVA/MANOVA. ODD = oppositional defiant disorder; Hyper-Imp = hyperactive-impulsive. (P) = Parent report; (T) = Teacher report. Response inhibition = efficiency score. Set-shifting = Time to complete Trails B. Working memory = Digit span backward raw score.

* $p < .05$. ** $p < .01$.

⁺ $p < .1$.

Table 3

Ethnic Differences in Child Symptoms and Risk Markers

	Caucasian <i>n</i> = 73	African American <i>n</i> = 28
ODD symptoms (P)	6.94 (6.01)	9.21 (8.01)
ADHD symptoms (P)	21.13 (14.62)	30 (16.23)**
Inattention (P)	9.14 (7.08)	13.82 (8.47)**
Hyper-Imp (P)	11.99 (8.29)	16.18 (8.38)*
ODD symptoms (T)	6.29 (6.14)	10.2 (7.63)
Male	6.89 (6.7)	14.75 (7.14)
Female	5.63 (5.58)	7.17 (6.82)
ADHD symptoms (T)	20.38 (14.98)	35.3 (15.39)**
Male	21.5 (14.75)	46 (8.12)
Female	19.13 (15.62)	28.17 (15.3)
Inattention (T)	9.88 (8.04)	18.7 (8.57)**
Male	10.33 (7.2)	24 (2.45)
Female	9.38 (9.11)	15.17 (9.56)
Hyper-Imp (T)	10.5 (8.23)	16.6 (7.86)*
Male	11.17 (8.41)	22 (6)
Female	9.75 (8.23)	13 (7.13)
Negative affect	4.1 (.96)	4.54 (1.14)*
Surgency	4.68 (1.02)	4.92 (.92)
Effortful control	4.79 (.9)	4.91 (.91)+
Response inhibition	.77 (.34)	.71 (.35)
Set-shifting	31.38 (24.44)	47.53 (31.29)*
Working memory	1.85 (2.35)	1.39 (2.06)

Note. Subgroup differences based on ANOVA/MANOVA. ODD = oppositional defiant disorder; Hyper-Imp = hyperactive-impulsive. (P) = Parent report; (T) = Teacher report. Response inhibition = efficiency score. Set-shifting = Time to complete Trails B. Working memory = Digit Span backward raw score.

+ $p < .1$.

* $p < .05$.

** $p < .01$.

Table 4

Correlations of Symptoms and Risk Markers With Family Income

	Family income	Mother education/employment	Father education/employment
ODD symptoms (P)	-.19 ⁺	-.1/.003	-.05/-.15
ADHD symptoms (P)	-.25*	-.18 ⁺ /.12	-.12/-.22*
Inattention (P)	-.2*	-.16 ⁺ /.1	-.09/-.22*
Male	-.08		
Female	-.37*		
Hyper-Imp (P)	-.27*	-.16/.14	-.14/-.18*
ODD symptoms (T)	-.16	-.21/.06	-.22/.01
ADHD symptoms (T)	-.09	-.28 ⁺ /-.09	-.14/-.04
Inattention (T)	-.1	-.37 [*] /-.09	-.2/-.1
Hyper-Imp (T)	-.11	-.17/-.04	-.1/-.01
Negative affect	-.29*	-.3 ^{**} /.03	-.2 [*] /-.17
Caucasian	-.09		
African American	-.54 ^{**}		
Surgency	-.06	-.12/.22*	-.07/-.02
Effortful control	.1	.14/-.13	.2/.03
Response inhibition	.17 ⁺	.08/-.2 ⁺	.09/.07
Set-shifting	-.18 ⁺	-.11/-.02	.01/-.15
Working memory	.25*	.1/-.07	.16/.2 ⁺

Note. ODD = oppositional defiant disorder; Hyper-Imp = hyperactive-impulsive. (P) = Parent report; (T) = Teacher report. Response inhibition = efficiency score. Set-shifting = Time to complete Trails B. Working memory = Digit Span backward raw score.

⁺ $p < .1$.

* $p < .05$.

** $p < .01$.

Table 5

Correlations of Symptoms and Risk Markers With Cumulative Risk Composite

	Cumulative risk
ODD symptoms (P)	.15
ADHD symptoms (P)	.23*
Inattention (P)	.15
Hyper-Imp (P)	.27**
ODD symptoms (T)	.2
ADHD symptoms (T)	.29
Inattention (T)	.27
Hyper-Imp (T)	.26
Negative affect	.22*
Surgency	.07
Effortful control	-.12
Response inhibition	-.09
Set-shifting	.16
Working memory	-.13

Note. ODD = oppositional defiant disorder; Hyper-Imp = hyperactive-impulsive. (P) = Parent report; (T) = Teacher report. Response inhibition = efficiency score. Set-shifting = Time to complete Trails B. Working memory = Digit Span backward raw score.

+ $p < .1$.

*
 $p < .05$.

**
 $p < .01$.