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Axis I and II Comorbidity in Adults With ADHD

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Ongoing debate over the validity of the attention-deficit/hyperactivity disorder (ADHD) construct in adulthood is fueled in part by uncertainty regarding implications of potentially extensive yet incompletely described comorbid Axis I and II psychopathology. Three hundred sixty-three adults ages 18 to 37 completed semistructured clinical interviews; informants were also interviewed, and best estimate diagnoses were obtained. Results were as follows: First, ADHD combined type (ADHD–C) had an excess of externalizing and internalizing Axis I disorders, suggesting a gradient-of-severity relationship between it and ADHD inattentive type (ADHD–I). Second, ADHD–C and ADHD–I did not differ in frequency of Axis II disorders. Third, however, ADHD overall was associated with increased rates of Axis II disorders, compared with rates in non-ADHD control participants, including both Cluster B (primarily borderline personality disorder) and Cluster C disorders. Fourth, ADHD incrementally accounted for clinician-rated global assessment of functioning scores above and beyond comorbid conditions or symptoms on either Axis I or Axis II. Results further inform nosology of ADHD in adults.

Keywords: attention-deficit/hyperactivity disorder (ADHD), comorbidity, *DSM–IV* subtypes, personality disorders, functional impairment

Attention-deficit/hyperactivity disorder (ADHD) is characterized by a persistent pattern of inattentive, hyperactive, and impulsive behaviors that begin in early childhood, often persist throughout development, and interfere with adaptive functioning (American Psychiatric Association, 2000). Population surveys estimate the prevalence of ADHD in adulthood to be about 5% (Faraone & Biederman, 2005; Kessler et al., 2006), and neurobiologic and genetic findings from adults with ADHD are similar to results seen in children (Faraone, 2004). Although interest in ADHD in adults has been long-standing (Wender, 1974) and has intensified in recent years (Faraone, 2000), adult ADHD remains relatively underinvestigated. Historically, ADHD was primarily conceptualized as a disorder of childhood. Subsequently, long-term follow-up studies of children with ADHD established that ADHD persists into adulthood in a substantial proportion of cases (Barkley, Fischer, Smallish, & Fletcher, 2004; Mannuzza, Klein, & Moulton, 2003; Weiss & Hechtman, 1993). A meta-analysis of follow-up studies found that, although estimates of persistence vary with how the diagnosis is defined, 65% of children with ADHD will show impairing symptoms of ADHD in adulthood (Faraone, Biederman, & Mick, 2006).

Patterns of clinical comorbidity, both Axis I and Axis II, are critical to evaluating the clinical validity of ADHD in adults for multiple reasons. First, comorbidity is likely very common. This is the case in children with ADHD (Biederman et al., 1993) and likely to also be true in adults. Although description of Axis I comorbidity has begun in adult ADHD (Barkley, 2002; Barkley, Fischer, Fletcher, & Smallish, 2002; Barkley et al., 2004; Marks, Newcorn, & Halperin, 2001; Murphy & Barkley, 1996; Young, Toone, & Tyson, 2003), Axis II comorbidity is inadequately mapped as we describe later. Even for Axis I psychopathology, findings in adults with ADHD have been somewhat inconsistent, perhaps because of relatively small sample sizes in many studies; predominantly male samples; and in some studies, reliance on rating scales or self-report to assess ADHD. With regard to gender, results of a prospective follow-up study of girls with ADHD showed continued impairment into adolescence but found only limited differences between ADHD subtypes (Hinshaw, Owens, Sami, & Fargeon, 2006).

Second, developmental change was not addressed in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) criteria, which are the same regardless of age. Yet substantial developmental changes occur in attention and activity level from childhood to adulthood (Faraone et al., 2006). For example, hyperactive behaviors apparently decline with development relative to inattentive symptoms (Hart, Lahey, Loeber, & Hanson, 1994). Adults with ADHD consequently may experience relatively more impairment from inattention, disorganization, and subjective restlessness rather than hyperactivity. Consequently, the validity and appropriateness of the *DSM–IV* ADHD subtype structure (combined, ADHD–C; primarily inattentive, ADHD–I; primarily hyperactive–impulsive, ADHD–H) for clinical characterization of adults is unclear. As a result, clinical comorbidity, which tends to differ in ADHD subtypes in children, may not show similar patterns in adults, at least when *DSM–IV* criteria are used.

In children, clinical data support the validity of distinguishing ADHD–I from ADHD–C (Carlson & Mann, 2000). ADHD–C is associated with more externalizing problems (Eiraldi, Power, & Nezu, 1997; Gaub & Carlson, 1997), whereas at least some studies

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suggest either that ADHD–I is associated with relatively more internalizing disorders or problems (Lahey et al., 1994; Weiss, Worling, & Wasdell, 2003) or that there are equivalent levels of internalizing in the two subtypes (Eiraldi, Power, Karustis, & Goldstein, 2000). Therefore, a strong prediction from the hypothesis that ADHD–C and ADHD–I are distinct disorders (Milich, Balentine, & Lynam, 2001) would be that in adults, ADHD–C would be associated with an excess of externalizing disorders, whereas ADHD–I would be associated with an excess of internalizing disorders. However, if ADHD–I is hypothesized to be a mild form of the more severe ADHD–C (Faraone, Biederman, Weber, & Russell, 1998), then it would be predicted that by adulthood, ADHD–C would show excess comorbidity across all domains relative to ADHD–I.

Only a handful of studies have examined this question in ADHD subtypes in adults, with mixed results. Millstein, Wilens, Biederman, and Spencer (1997) found more bipolar, oppositional, and substance use disorders in ADHD–C than ADHD–I or ADHD–H. Murphy, Barkley, and Bush (2002) found that both ADHD–C and ADHD–I had excess dysthymia, alcohol and drug dependence–abuse, learning disorders, and psychological distress, but ADHD–C was further associated with oppositional defiant disorder (ODD), suicide attempts, arrests, and interpersonal hostility and paranoia. In a study of children and adolescents, Volk, Henderson, Neuman, and Todd (2006) found that impairment among ADHD subtypes was not increased by comorbidity with conduct disorder (CD), ODD, or major depressive disorder.

Third, aside from antisocial personality disorder, which is often associated with ADHD in adulthood (Biederman et al., 1993; Downey, Stelson, Pomerleau, & Giordani, 1997; Faraone et al., 2000; Levin, Evans, & Kleber, 1998; Loeber, Burke, & Lahey, 2002), surprisingly little research has considered Axis II comorbidity. None, to our knowledge, have considered whether Axis II comorbidity can account for ADHD-related impairment. Data on normal personality traits suggest that ADHD may be associated with extreme standing on personality (Nigg et al., 2002). Such findings, as well as the aforementioned stability and chronicity of the impulsive and dysregulated behaviors that compose ADHD, suggest a theoretical connection between ADHD symptoms and personality traits and, by extension, *personality disorders*—which are defined as chronic, maladaptive personality traits (American Psychiatric Association, 2000). This connection emerges in various ways. One possibility is that ADHD alters personality and thus increases risk for personality disorder later in development. Another possibility is that both ADHD and certain personality disorders are related to the same extreme personality diathesis. In either case, the question arises whether both ADHD and personality disorder diagnoses add incremental validity in predicting impairment.

Cluster B personality disorders are conceptually the most similar to ADHD (Rey, Morris-Yates, Singh, Andrews, & Stewart, 1995) and therefore are the group we expected to be most likely to co-occur with adult ADHD. They are characterized by inability to control or regulate behavior, affect, and cognition and by social and interpersonal problems that are at least superficially similar to those seen in ADHD (Akiskal et al., 1985; Tzelepis, Schubiner, & Warbasse, 1995; Weiss, Hechtman, & Weiss, 1999). Indeed, the idea that ADHD may be a precursor to borderline personality disorder (BPD) has been long-standing (Akiskal et al., 1985), although research on this overlap is scarce. Yet a small number of studies suggest that ADHD is associated with BPD (Dowson et al., 2004; Rey et al., 1995), may be superimposed on the personality difficulties of those patients with BPD (Weiss et al., 1999), and appears more often in the childhood history of patients with adult BPD (Fossati, Novella, Donati, Donini, & Maffei, 2002). However, these studies were relatively small and/or did not use *DSM–IV* criteria or structured diagnostic interviews, leaving their conclusions in some doubt. Other personality traits and disorders that may be related to adult ADHD include histrionic and narcissistic traits (Fischer, Barkley, Smallish, & Fletcher, 2002; May & Bos, 2000), obsessive–compulsive personality disorder (OCPD), and histrionic personality disorder (Modestin, Matutat, & Wuermle, 2001), as well as personality dimensions such as novelty or sensation seeking (Downey et al., 1997).

Fourth, the extensive comorbidity associated with ADHD underscores divergent conceptualizations of how ADHD is taxonomically related to comorbid conditions (Faraone, 2000). Within a *hierarchical* framework, a patient meeting criteria for two or more disorders would only be diagnosed with the higher ranking disorder; *DSM–IV* follows this approach to some degree for ADHD by requiring that the ADHD symptoms not be better accounted for by some other mental disorder. The *comorbidity* framework allows for the assessment of all disorders; diagnostic overlap is viewed more as the rule than the exception. From a hierarchical perspective, it remains unclear whether there is additional clinical utility in diagnosing ADHD in the presence of co-occurring disorders or their symptoms. One way to assess this is to examine whether ADHD adds to the statistical prediction of impairment after comorbid disorders have been covaried. Lahey et al. (2004) found this relationship in children: Children with ADHD continued to display significant impairment relative to control participants when comorbid psychopathology was statistically controlled. This critical question of ADHD's incremental validity relative to clinical impairment remains essentially untested in adults, although some data show that ADHD is a risk factor for substance use disorders in adults, even after the researchers controlled for a history of conduct or bipolar disorders (Biederman et al., 1995). In a controlled study of community adults, ADHD (without regard to comorbid conditions) was associated with impairment in terms of lower degree attainment, less employment, more job changes, more arrests and divorce, and lower personal satisfaction (Biederman et al., 2006), but comorbid conditions were not controlled.

Aims of the Present Study

The present study sought to clarify clinical validity of the ADHD construct in adults by examining independent predictors of adaptive impairment and subtype differences in relation to Axis I and Axis II comorbidity. Key questions were (a) whether ADHD–C accrues more comorbid externalizing disorders than ADHD–I and controls and whether the comorbid profile differs across ADHD subtypes (interaction of Comorbid Domain × Subtype), (b) which domains of Axis II personality disorders are associated with ADHD regardless of subtype, and (c) whether ADHD or its symptoms have an influence on impairment above and beyond that which is accounted for by Axis I and Axis II psychopathology.

Method

Participants

Overview. The sample included 363 adults (185 men and 178 women) ages 18 to 37 years. Participants' race closely mirrored the surrounding community from which they were obtained, with 86% Caucasian. Following procedures described later, participants were grouped into ADHD (any subtype; $n = 152$) and non-ADHD control participants ($n = 211$). For some analyses, participants with ADHD were further divided into ADHD–I ($n = 69$) and ADHD–C ($n = 64$). We had only 19 participants with ADHD–H; this group was judged too small for reliable analysis and so was omitted from subtype analyses.

Multistage recruitment process. Participants were recruited by a broad net of public advertisements, including radio, newspaper, movie theaters, and mailings to local clinics, in an effort to obtain as broadly representative a volunteer sample as possible. We advertised for ADHD with separate ads targeted at "individuals that have been diagnosed or suspect they have an attention deficit disorder (ADHD or ADD), or other attention problems." We advertised for the non-ADHD comparison group with ads seeking "volunteers in good health who do not have attention problems." These efforts resulted in 623 applicants contacting the project office. These prospective participants underwent a phone screening to check rule outs (inclusionary criteria were ages 18–40, no sensorimotor disability, no neurological illness, native English speaking, and currently prescribed antidepressant, antipsychotic, or anticonvulsant medications). At this stage, 533 participants were coded as eligible and went on to Stage 2. At Stage 2, eligible participants completed semistructured clinical interviews and normative rating scales to assess ADHD and comorbid Axis I and Axis II disorders as detailed next. Those data were then reviewed by the best estimate clinical team to determine final diagnostic assignment and study eligibility. One hundred seventy participants were excluded at this stage (because of lack of cross-informant convergence enabling clear classification as ADHD or non-ADHD, current major depression, current severe substance use, psychosis, or brain–head injury), yielding the final $N = 363$.

Diagnostic instruments. A retrospective Kiddie Schedule for Affective Disorders and Schizophrenia (K–SADS; Puig-Antich & Ryan, 1986) was administered to assess ADHD. This well-established procedure (Biederman et al., 1992; Biederman, Faraone, Keenan, Knee, & Tsuang, 1990) included the diagnoses of childhood *DSM–IV* ADHD, CD, and ODD. Current symptoms were assessed by structured interview and included K–SADS ADHD questions worded appropriately for current adult symptoms (Biederman et al., 1992). Respondents also completed the Barkley and Murphy (1998) Current ADHD Symptoms rating scale. We obtained normative, standardized dimensional ratings of attention problems as well as other current symptoms by having participants complete the Conners, Erhardt, and Sparrow (1999) Adult ADHD Rating Scale, the Achenbach (1991) Young Adult Self Report scale, and the Brown (1996) Adult ADHD rating scale.

To address potential reporting bias in self-report interviews of ADHD (Barkley et al., 2002), two informants who knew the participant well were interviewed. One informant, who knew the participant as a child (usually a parent) reported on the target participant's childhood behaviors via an ADHD Rating Scale and a retrospective K–SADS ADHD module. The second informant, who knew the participant currently (usually a spouse or friend), completed the Conners peer rating, the Barkley and Murphy peer ratings on adult symptoms, a brief screen of antisocial behavior and drug and alcohol use, and a structured interview about the participant's current ADHD symptoms, using the modified K–SADS for current symptoms.

Establishment of best estimate diagnosis for ADHD. A diagnostic team that included a licensed clinical social worker, a licensed clinical psychologist, and a board certified psychiatrist then arrived at a "best estimate" diagnosis (Faraone, 2000). Each team member reviewed all available information from the semistructured interviews, and rating scales to arrive at a judgment about ADHD present or absent, ADHD subtype, and comorbid disorders. In the case of disagreement, consensus was reached by discussion. Interrater agreement on presence or absence of ADHD (any type) was satisfactory ($k = .80$), and agreement on ADHD subtype (combined, inattentive, or hyperactive) in childhood and adulthood was also adequate (ranging from $k = .74$ to .85). In view of disagreement about whether the subtype classification should be represented by childhood or adult symptom profiles (Faraone, 2000), we relied on the adult subtype here.

Assessment of comorbid Axis I disorders. The Structured Clinical Interview for *DSM–IV* Axis I Disorders (SCID–I; First, Spitzer, Gibbon, & Williams, 1997) was administered by a trained master's level clinician after extensive training in the interview and checkout of taped interviews for validity by First and colleagues at Columbia. Diagnoses examined in the current study included major depressive episode, dysthymic disorder, bipolar disorder, substance abuse and dependence, psychotic disorders, obsessive–compulsive, panic disorder, agoraphobia, simple phobia, social phobia, and eating disorders. Autistic disorder was screened by added symptom questions and was a rule out. Twenty-five SCID interviews were videotaped and coded by two qualified interviewers to assess reliability of the interview procedures. Reliability for comorbid disorders was acceptable (e.g., substance use disorder, $k = .83$; mood disorder, $k = .80$; anxiety disorder, $k = .71$; antisocial personality disorder, $k = .84$).

Assessment of Axis II disorders. Participants completed the SCID–II prescreening form. Any disorder for which at least one symptom was endorsed was followed up with the SCID–II interview module for that disorder. This procedure results in a very low rate of false negatives (participants who endorse zero symptoms on the questionnaire virtually never have a disorder on administration of the full SCID–II interview) while capturing potential personality disorders and assessing them after the screen (First, Gibbon, Spitzer, Williams, & Benjamin, 1997).

Assessment of Impairment

Global assessment of functioning (GAF; American Psychiatric Association, 2000) scores were assigned by the interviewing clinician at the end of the structured clinical interviews. This score of overall functional adjustment was used as an index of impairment; high scores indicate better functioning and low scores indicate more impairment. To evaluate reliability of the impairment scores, 20 SCID–KSAD interviews were taped and reviewed by a second clinical rater, blind to the GAF rating or diagnoses of the first rater. GAF scores were assigned by the second rater and were compared

with those assigned by the first rater. The interclass correlation (absolute agreement) of .714 reflected adequate interrater reliability.

Data Analytic Plan

We tested a series of questions regarding subtype effects and Axis I disorders with multinomial and binomial logistic regression, with sex effects covaried. We tested a series of questions concerning impairment using linear multiple regression and multiple logistic regression models. Statistical power for all analyses exceeded .90 to detect Cohen's (1992) medium-sized effects (r = .15), with the exception of some pairwise post hoc tests.

Results

Sample Description and Review of Potential Demographic Confounds

Demographic information for the sample is provided in Table 1. Ratings data all showed marked clinical elevations in the ADHD sample, indicating validity of ADHD assignments regardless of instrument or model used. Parental household incomes were similar in the two groups ($p > .4$), indicating that they came from similar socioeconomic backgrounds. Despite this, and consistent with prior reports (Murphy & Barkley, 1996), individuals with ADHD were less likely to complete high school than control participants (see Table 1). The ADHD group was less likely to attend college than the control group (53% vs. 62%, $p < .05$). Those who attended a 4-year college were more likely to be control participants than participants with ADHD (36% vs. 20%, $p < .01$), whereas those who attended a 2-year community college were more likely to have ADHD than be control participants (18% vs. 10%, $p < .01$). Thus, the ADHD group had lower educational attainment overall. Personal incomes were qualitatively lower for the nonstudent ADHD (M = $32,000) than non-ADHD group (M = 40,200) though this effect was not significant. The gender difference, with a greater proportion of male participants with ADHD (see Table 1), occurred despite our efforts to overselect female participants with ADHD; it is common in studies of ADHD and in part may reflect the male preponderance of ADHD in the population. Because some of the comorbid disorders vary by gender, we controlled statistically for gender. Groups did not differ significantly in percentage of minority participants, although there were qualitatively more minorities in the ADHD group ($p < .07$). We therefore checked all results with ethnicity covaried; results were unchanged from those reported in this article. Consistent with other studies of ADHD, participants with any ADHD were more likely to have substance use disorder, $\chi^2(1, N = 363) = 9.22$, $p < .01$; mood disorder, $\chi^2(1, N = 363) = 23.70$, $p < .001$; anxiety disorder, $\chi^2(1, N = 363) = 8.81$, $p < .01$; and antisocial personality disorder, $\chi^2(1, N = 363) = 7.32$, $p < .01$, than the non-ADHD comparison group.

Hypothesis 1: Subtype Comorbid Profiles for Axis I Disorders

To test this question, we summed total number of lifetime (a) externalizing disorders (lifetime ODD, CD, substance use disorders, and antisocial personality disorder), and (b) internalizing disorders (all mood and anxiety disorders). Because these were ordinal count variables, we analyzed group effects with multinomial logistic regression. To obtain adequate cell sizes, we categorized number of externalizing and internalizing disorders as follows: 0 (none), 1, and 2 or more. Table 2 shows the resultant frequencies. We checked all models for Sex × Group interactions and they were not significant, so we simply covaried sex.

For externalizing disorders, the three-group multinomial logistic regression model (with sex covaried) indicated a significant model overall, $\chi^2(6, N = 344) = 27.1$, $p = .001$, –2LL = 54.3; the main effect of group was significant as well, $\chi^2(4, N = 344) = 20.8$, $p = .001$, –2LL = 75.0. Using the control group as the reference, presence of one externalizing disorder was more likely for participants with ADHD–C (odds ratio [OR] = 2.05, $p = .050$), but not for ADHD–I ($p = .766$). Presence of two or more externalizing disorders was more likely for ADHD–C (OR = 5.12, $p = .001$) and ADHD–I (OR = 2.12, $p = .038$). Thus, ADHD–C conferred a fivefold increase in risk for two or more externalizing disorders whereas ADHD–I conferred a doubling of such risk versus the control group. The OR was significantly higher for ADHD–C than ADHD–I for two or more disorders ($p = .044$), indicating that ADHD–C conferred more risk of externalizing disorder than ADHD–I.

For internalizing disorders, the three-group multinomial logistic regression model (with sex covaried) indicated a significant model overall, $\chi^2(6, N = 344) = 39.7$, $p = .001$, –2LL = 49.6; the main effect of group was again significant, $\chi^2(4, N = 344) = 33.3$, $p = .001$, –2LL = 82.9. Using the control group as the reference, we found that presence of one internalizing disorder was more likely for ADHD–C (OR = 4.59, $p = .001$) and ADHD–I (OR = 3.22, $p = .001$). Presence of two or more internalizing disorders was significant for ADHD–C (OR = 3.76, $p = .001$) and for ADHD–I (OR = 4.02, $p = .001$). Although the OR was slightly higher for ADHD–I than ADHD–C for two or more disorders, the ADHD–C versus ADHD–I effect was nonsignificant ($p = .854$). As can be seen in Table 2, although the ADHD–I group had slightly more likelihood of two or more internalizing disorders, the ADHD–C group had more likelihood of internalizing disorder overall, although this difference also was nonsignificant.

In summary, ADHD–C was associated with more comorbid externalizing disorders than ADHD–I or non-ADHD status. However, contrary to a "distinct disorder" hypothesis, as displayed in Table 2 ADHD–I was associated with qualitatively lower, not higher, rates of total internalizing disorders than ADHD–C, although ADHD–I was associated with a slightly but not significantly greater chance of having two or more internalizing disorders versus ADHD–C.

Hypothesis 2: Axis II Comorbidity in Relation to ADHD

For these analyses, we created three personality disorder composite variables: Cluster A disorders present or absent (presence of one or more of paranoid, schizoid, and/or schizotypal personality disorder), Cluster B disorders present or absent (presence of one or more of borderline, antisocial, histrionic, and/or narcissistic personality disorder), and Cluster C disorders present or absent (presence of one or more of avoidant, dependent, and/or OCPD). Table 3 shows the frequencies of the Axis II disorders by cluster for each group. The ADHD subtypes did not differ on any of these clusters:

Table 1
Description of Sample

Variable	Control (n = 211)				Any ADHD (n = 152)					ADHD–I (n = 69)				ADHD–C (n = 64)				
	n	%	*M*	*SD*	*n*	%	*M*	*SD*	*p*	*n*	%	*M*	*SD*	*n*	%	*M*	*SD*	*p*
Male	89	42			96	63			<.001	47	68			41	64			.504
White	174	83			136	90			.062	60	87			58	91			.625
Age (in years)			23.7	4.5			23.7	4.6	1.00			23.31	4.7			23.78	4.8	.828
Conners ADHD T score			47.2	11.1			62.1	10.1	<.001			58.51	9.7			66.10	9.5	.526
No. of current *DSM* inattentive symptoms			1.81	2.2			7.0	1.7	<.001			7.26	1.5			7.29	1.4	.922
No. of current *DSM* hyperactivity–impulsivity symptoms			1.7	2.0			5.68	2.5	<.001			3.74	2.1			7.10	1.5	<.001
No. of childhood *DSM* inattentive symptoms			1.93	2.1			6.78	1.6	<.001			6.99	1.3			7.06	1.4	.188
No. of childhood *DSM* hyperactivity–impulsivity symptoms			1.64	1.7			5.13	2.2	<.001			3.63	1.9			6.23	1.7	<.001
Parent annual combined income[a]			3.73	1.1			3.64	1.2	.484			3.83	1.1			3.43	1.2	<.05
Lifetime substance use disorder	84	40			88	57			<.001	35	50			44	67			.112
Lifetime mood disorder	58	28			82	53			<.001	36	51			38	58			.370
Lifetime any anxiety disorder	36	17			47	30			<.05	24	34			18	27			.888
Lifetime antisocial personality disorder	8	4			15	11			<.05	6	10			8	15			.356
Completed high school	137	94			91	84			<.05	41	87			38	79			.298
Marital status									.865									.500
Married	26	14			16	13				6	10			7	14			
Single	155	81			103	84				50	86			43	84			
Divorced	9	5			4	3				2	4			1	2			

Note. The probability value reflects the two-group comparison (control vs. any ADHD) and is based on an independent-samples t test for continuous variables or a chi-square test for dichotomous variables. Sample sizes varied slightly for some measures because of missing data for some disorders. ADHD = attention-deficit/hyperactivity disorder; ADHD–I = ADHD inattentive type; ADHD–C = ADHD combined type.

[a] Parent annual combined income categories were as follows: 1 = $25,000–$50,000; 2 = $50,000–$75,000; 3 = $75,000–$100,000; 4 = greater than $100,000.

Table 2
Hypothesis 1: Frequencies of Externalizing and Internalizing Disorders for Control Participants and for Participants With Attention-Deficit/Hyperactivity Disorder Combined (ADHD–C) and Inattentive (ADHD–I) Subtypes

	Control		ADHD–C		ADHD–I	
Frequency	*n*	%	*n*	%	*n*	%
Externalizing disorders						
0	120	56.9	18	28.1	31	44.9
1	60	28.4	20	31.3	19	27.5
2 or more	31	14.7	26	40.6	19	27.5
Internalizing disorders						
0	137	64.9	22	33.4	27	39.1
1	39	18.5	26	40.6	22	31.9
2 or more	35	16.6	16	25.0	20	29.0

Note. Externalizing disorders include lifetime oppositional defiant disorder, conduct disorder, substance use disorders, and antisocial personality disorder. Internalizing disorders include mood and anxiety disorders.

Table 4
Hypothesis 2: Relationship Between Attention-Deficit/Hyperactivity Disorder (ADHD) and Personality Disorder Cluster—Summary of Binomial Logistic Regression With Sex Covaried

Variable	*B*	*SE B*	OR
Cluster A			
ADHD	0.476	0.541	1.610
Sex	0.004	0.542	1.004
Constant	−3.369	0.447	0.034***
Cluster B			
ADHD	1.132	0.309	3.102***
Sex	−0.069	0.302	0.934
Constant	−2.223	0.265	0.108***
Cluster C			
ADHD	1.869	0.404	6.482***
Sex	−0.371	0.353	0.690
Constant	−2.966	0.361	0.052***

Note. OR = odds ratio.
*** $p < .001$.

Cluster A, $\chi^2(2, N = 363) = 4.57$, $p = .10$; Cluster B, $\chi^2(2, N = 363) = 5.58$, $p = .061$; Cluster C, $\chi^2(2, N = 363) = 0.57$, $p = .75$. We therefore collapsed across ADHD subtypes and proceeded to our primary analysis of ADHD versus non-ADHD using binomial logistic regression analysis, with sex covaried in all models. To control Type I error, for each cluster, we conducted an initial omnibus logistic regression for excess of total disorders in that cluster. If that omnibus test was significant, we proceeded to examine effects of individual Axis II disorders.

Results of binomial logistic regression analyses for Cluster A, B, and C personality disorders are presented in Table 4. For Cluster A, the binomial logistic regression model indicated a nonsignificant model overall, $\chi^2(2, N = 363) = 0.81$, $p = .666$, –2LL = 124.1; there was no significant effect of ADHD status on likelihood of having excess Cluster A (OR = 1.6, $p = .38$; ADHD = 5.3%, control = 3.3%) and no significant Group × Gender interaction ($p = .948$). We therefore did not analyze Cluster A disorders further.

Table 3
Hypothesis 2: Frequencies of Axis II Disorders for Control Participants and for Participants With Each Attention-Deficit/Hyperactivity Disorder (ADHD) Subtype

	Control		ADHD–C		ADHD–I		ADHD–H	
Frequency	*n*	%	*n*	%	*n*	%	*n*	%
Cluster A								
No PDs	204	96.7	58	90.6	68	98.6	18	94.7
One or more PDs	7	3.3	6	9.4	1	1.4	1	5.3
Cluster B								
No PDs	191	90.5	50	78.1	55	79.7	10	52.6
One or more PDs	20	9.5	14	21.9	14	20.3	9	47.4
Cluster C								
No PDs	202	95.7	49	76.6	55	79.7	16	84.2
One or more PDs	9	4.3	15	23.4	14	20.3	3	15.8

Note. ADHD–C = ADHD combined type; ADHD–I = ADHD inattentive type; ADHD–H = ADHD primarily hyperactive–impulsive type; PD = personality disorder.

For Cluster B, the omnibus binomial logistic regression model indicated a significant model overall, $\chi^2(2, N = 363) = 14.50$, $p = .001$, –2LL = 300.75. ADHD was associated with increased likelihood of having a Cluster B personality disorder (OR = 3.10, $p = .001$; ADHD = 24.4%, control = 9.5%). The Group × Gender interaction was nonsignificant, though marginal ($p = .074$). Post hoc individual likelihood ratio chi-squares indicated an excess in the ADHD versus control group of BPD (20.3% vs. 3.9%, $p < .001$), antisocial personality disorder (11.3% vs. 3.9%, $p < .01$), histrionic personality disorder (2.3% vs. 0%, $p = .017$), and narcissistic personality disorder (12% vs. 3%, $p < .01$). Because of overlap among the personality disorders, unique effects were examined. When all four Cluster B disorders (present–absent) were entered as categorical predictors in a simultaneous logistic regression model to predict ADHD (present–absent), only excess BPD was a significant predictor ($B = 1.6$, $p < .001$; all other Cluster B personality disorders, $p > .25$).

With regard to the marginal Sex × Group interaction, in light of lower power to detect interactions (Keppel & Wickens, 2004), we conducted a post hoc exploratory examination for descriptive purposes only; we do not interpret these effects because the decomposition was not justified by our Fisherian decomposition strategy. We found a smaller effect in men than in women overall (men with any Cluster B disorder, ADHD = 20.9%, control = 12.5%; women with any Cluster B disorder, ADHD = 30.3%, control = 7.3%). Women with ADHD were more likely than control women to have one Cluster B disorder (ADHD = 19.6%, control = 5.7%), as well more likely to have two or more Cluster B disorders (ADHD = 10.7%, control = 1.6%). Men, on the other hand, had no excess likelihood of having one disorder (ADHD = 9.4%, control = 11.4%) but had a greater incidence of two or more disorders (ADHD = 11.5%, control = 1.1%).

For Cluster C, the binomial logistic regression model was also significant, $\chi^2(2, N = 363) = 26.12$, $p = .001$, –2LL = 229.65.

The Group × Sex interaction was nonsignificant ($p = .11$). ADHD was associated with increased likelihood of having one or more Cluster C personality disorders (OR = 6.48, $p < .001$; ADHD = 21.0%, control = 4.3%). Although absolute rates of Cluster C disorders in the sample were relatively low, post hoc single-disorder effects were significant for each disorder, with ADHD more likely to have OCPD (14% vs. 4%, $p = .001$), avoidant personality disorder (13% vs. 1%, $p < .001$), and dependent personality disorder (5% vs. 0%, $p < .001$). When all three Cluster C disorders (present–absent) were entered as categorical predictors in a simultaneous logistic regression model to predict ADHD while controlling the overlap among the personality disorders, ADHD was predicted by excess OCPD ($B = 1.1$, $p < .05$) and avoidant personality disorder ($B = 2.3$, $p < .01$) but not dependent personality disorder ($p > .9$).

Hypothesis 3a: Specificity of Impairment to ADHD in Relation to Major Axis I and II Comorbidity

In our first analysis of specificity to impairment, we focused on common co-occurring disorders on Axis I and Axis II. A hierarchical multiple regression analysis was implemented wherein the outcome was the GAF impairment score, and the predictors (Block 1) were the following diagnoses: antisocial personality disorder (yes–no), BPD (yes–no), generalized anxiety disorder (yes–no), and major depressive disorder (yes–no). ADHD was added in Block 2 to examine whether it retained a significant unique association with impairment when the other disorders were controlled. Results are portrayed in Table 5. ADHD accounted for significant variance in impairment above and beyond the effects of the other disorders, $\Delta R^2 = .033$, $F(5, 333) = 28.60$, $p < .001$. As Table 5 shows, the other disorders were also significant predictors of impairment, even with ADHD in the model.

This same model was then tested using dimensional psychopathology scores, using a linear multiple regression model in which the outcome was the GAF score and predictors now were the number of symptoms of antisocial personality disorder, BPD, anxiety (from the Young Adult Self Report), and depression (from the Young Adult Self Report), and total number of ADHD symptoms (by self-report). The bottom of Table 5 shows this result. Again, ADHD symptoms explained a significant amount of the variance in clinician-rated GAF score beyond that explained by comorbid symptoms, $\Delta R^2 = .040$, $F(5, 303) = 44.83$, $p < .001$. In summary, ADHD influenced impairment above and beyond that accounted for by antisocial personality disorder, BPD, generalized anxiety disorder, or major depressive disorder (both at the diagnosis level and at the symptom level).

Table 5
Hypothesis 3a: Summary of Hierarchical Multiple Regression of Variables Predicting Impairment (Global Assessment of Functioning Score)

Step and variable	*B*	*SE B*	β
Step 1			
ASPD (Y-N)	−9.60	2.14	−.219***
BPD (Y-N)	−9.74	1.81	−.269***
GAD (Y-N)	−7.05	1.90	−.182***
MDD (Y-N)	−3.74	1.17	−.159**
Step 2			
ASPD (Y-N)	−8.99	2.10	−.206***
BPD (Y-N)	−8.32	1.81	−.230***
GAD (Y-N)	−6.21	1.86	−.161**
MDD (Y-N)	−3.12	1.16	−.133**
ADHD (Y-N)	−4.40	1.11	−.194***

Note. ASPD = antisocial personality disorder; Y-N = disorder present (yes) or absent (no); BPD = borderline personality disorder; GAD = generalized anxiety disorder; MDD = major depressive disorder; ADHD = attention-deficit/hyperactivity disorder. $\Delta R^2 = .270$ for Step 1; $\Delta R^2 = .033$ for Step 2, $F(5, 333) = 28.60$, $p < .001$.
** $p < .01$. *** $p < .001$.

Hypothesis 3b: Specificity of ADHD to Functional Impairment in Relation to All Axis II Disorders

We next conducted a multiple regression with GAF impairment score as the outcome variable and all 10 Axis II disorders as predictors, with ADHD diagnosis as the last predictor entered. Results are displayed in Table 6. As it shows, ADHD was a significant and substantial incremental predictor of impairment ($p < .001$) after we controlled for all Axis II disorders. Impairment was also uniquely predicted by presence of borderline, antisocial, schizotypal, and paranoid personality disorders in this model. Similarly, symptoms of schizotypal, borderline, and antisocial personality disorders significantly predicted impairment (each with $p < .05$) after we controlled for ADHD symptoms.

Discussion

The purpose of this investigation was to further our understanding of validity of the ADHD construct for adults. In a well-characterized sample recruited in adulthood, we found that (a) ADHD–C and ADHD–I subtypes were differentiated by degree of severity, in that ADHD–C was associated with more externalizing disorders and the two subtypes did not differ for internalizing disorders; (b) ADHD was associated with an excess of Cluster B and Cluster C personality disorders (with no subtype effects), suggesting that Axis II comorbidity deserves closer scrutiny in future studies; and (c) ADHD diagnosis independently predicted functional impairment as rated by clinical interviewers, after we controlled for comorbid disorders, replicating the pattern seen in children (Lahey et al., 2004) and supporting the construct validity of ADHD in adulthood. We comment on each of these points in turn.

First, a primary goal of this investigation was to examine the discriminant validity of the ADHD subtypes in adulthood in relation to patterns of clinical comorbidity. We focused on ADHD–C and ADHD–I because of an inadequate sample size for ADHD–H. In children, some studies have reported a relatively greater association of ADHD–I with internalizing (Lahey et al., 1994) and of ADHD–C with externalizing disorders (Gaub & Carlson, 1997), but others have found that ADHD–C has similar rates of internalizing disorders to ADHD–I (Baumgaertel, Wolraich, & Dietrich, 1995; Eiraldi et al., 1997; Faraone, Biederman, Weber, & Russell, 1998; Morgan, Hynd, Riccio, & Hall, 1996; Paternite, Loney, & Roberts, 1995; Wolraich, Hannah, Pinnock, Baumgaetel, & Brown, 1996). Our result in adults was consistent with the latter set of studies in children. ADHD–C was associated with excess comorbid externalizing disorders, but ADHD–C and ADHD–I did

Table 6
Hypothesis 3b: Summary of Multiple Regression Analysis for Axis II Disorders and Attention-Deficit/Hyperactivity Disorder (ADHD) Predicting Global Assessment of Functioning Score

Variable	*B*	*SE B*	β	*p*
Paranoid PD	6.08	2.9	.10	.038*
Schizotypal PD	16.00	7.9	.14	.041*
Schizoid PD	7.18	12.8	.04	.581
Borderline PD	8.20	2.0	.23	<.001***
Antisocial PD	8.58	2.3	.19	<.001***
Histrionic PD	0.95	6.4	.01	.882
Narcissistic PD	2.95	2.4	.07	.217
Obsessive–compulsive PD	3.78	2.0	.09	.064†
Avoidant PD	1.70	2.5	.04	.497
Dependent PD	6.01	4.0	.08	.133
ADHD	4.93	1.14	.22	<.001***

Note. PD = personality disorder.
† $p < .10$. * $p < .05$. *** $p < .001$.

not differ in rates of internalizing disorders. Thus, in childhood there may be clinical support for the syndromal distinction of the ADHD–C and ADHD–I types (Milich et al., 2001), but these data suggest that by adulthood a gradient-of-severity model is occurring even for clinical comorbid profiles. It may be that the subtypes have less validity in adults than they do in children or that the subtype criteria are not optimal for adults.

However, the *DSM–IV* criteria may result in bias toward a gradient-of-severity perspective, even in children (Faraone et al., 1998), because individuals who are one symptom shy of ADHD–C are diagnosed with ADHD–I. It should be noted that the argument that ADHD–I and ADHD–C are distinct (Milich et al., 2001) emphasizes a group of children who are sluggish and hypoactive (Carlson & Mann, 2002). Such alternative phenotypes for ADHD–I were beyond the scope of the present study of *DSM–IV* constructs. However, it will be of interest to examine comorbid profiles in a refined phenotype of ADHD–I.

Second, Axis II comorbidity remains insufficiently investigated in adults with ADHD (Akiskal et al., 1985; Dowson et al., 2004; Fischer et al., 2002; Rey et al., 1995). Axis II disorders may provide a stronger challenge than Axis I disorders to the validity of ADHD in adults, because although ADHD is an Axis I disorder, it involves chronic and maladaptive behavior patterns. This possibility carries important clinical implications for differential diagnosis and construct validity of both ADHD and the personality disorders.

Because of the large number of Axis II disorders and their overlap, we examined them in relation to their *DSM–IV* clusters to increase statistical power. High levels of comorbidity between ADHD and Cluster B disorders have been suggested because Cluster B disorders are characterized by inability to control or regulate behavior, affect, and cognition and by social and interpersonal problems that are conceptually similar to ADHD symptoms (Akiskal et al., 1985; Tzelepis et al., 1995; Weiss et al., 1999). Our data confirmed this supposition. ADHD was associated with more Cluster B disorders; this effect was carried uniquely by elevated BPD symptoms. Thus, it may be that ADHD predisposes one to Cluster B personality disorders in adulthood, perhaps by altering the trajectory of personality development. Alternatively, it may be that ADHD and Cluster B personality disorders share similar personality diatheses and thus tend to co-occur at above-chance levels. Further studies examining ADHD, Axis II, and personality can help to clarify these possibilities.

However, contrary to expectations, the ADHD group also had more Cluster C personality disorders than control participants. This could reflect the high overlap of fear and anxiety in inattentive symptoms in adults, but this issue is not well investigated, and we did not see subtype differences in our Cluster C effects. Modestin et al. (2001) found that patients with a history of ADHD developed OCPD more frequently than did control participants. Similar findings were reported by Geller et al. (2003, 2004). We replicated that result, with elevated OCPD in our ADHD sample, but also elevated avoidant personality disorder. It may be that an important etiological subgroup of ADHD is associated with anxious–obsessive features, perhaps in conjunction with an attentional overfocus; this warrants follow-up study.

Third, a crucial validity question is whether impairment is associated with ADHD after common comorbid conditions have been controlled. Although demonstrated in children (Lahey et al., 2004), this crucial issue has been underinvestigated in adulthood, where the validity of ADHD remains more disputed. The finding that ADHD incrementally accounted for deficits in impairment above and beyond the presence or absence of comorbid conditions or symptoms provides important new data suggesting that there is meaningful clinical validity to the ADHD construct in adults. Findings are also consistent with a comorbidity model, which allows for diagnostic overlap when disorders add unique contributions to the clinical profile. This is distinct from a hierarchical model that would suggest impairment is accounted for by the higher ranking disorders (Faraone, 2000).

ADHD is a heterogeneous condition (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005). Thus, ADHD may be a risk factor for personality pathology in more than one domain. One pathway, well recognized in the literature, may emanate from underregulated and erratic symptoms and be reflected in the overlap of ADHD and antisocial behavior problems (Biederman et al., 1993; Downey et al., 1997; Faraone et al., 2000) as well as histrionic personality disorder and BPD symptoms. A second pathway, less well recognized, may emanate from the overlap of ADHD with mood problems, leading into Cluster C disorders. This warrants further investigation from a personality perspective and could have significant implications for subtyping of adults with ADHD.

With regard to clinical implications, this study suggests that it is important to assess Axis I and Axis II comorbidity in adults presenting with possible ADHD. In addition, assessing ADHD in adults who come to treatment with other disorders has value in that it adds incremental validity to predicting impairment and thus treatment need. These findings also suggest that establishing valid diagnostic subtypes in adults with ADHD remains unclear. Most important, these findings confirm the importance of assessing Axis II comorbidity when assessing adults for ADHD, including not only Cluster B but also Cluster C disorders.

The main methodological limitation was that participants were recruited in adulthood. The overlap of this population with children followed forward into adulthood remains unclear (Weiss et al., 1999). Although one strength of this study was that we obtained informant reports of childhood symptoms, these assessments were nonetheless retrospective. In future research, it will be ideal to obtain objective records of childhood functioning in the

form of school and/or medical records or by contacting teachers who knew the probands as children.

In conclusion, results of this study support the clinical validity of the ADHD construct in adulthood, highlight the importance of Axis II comorbidity in this condition, and call into question the clinical difference between ADHD–C and ADHD–I as defined by *DSM–IV* in adults, other than severity. Further analysis of Axis II and personality may help in subtyping the ADHD condition as it presents clinically by adulthood.

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