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Assessing Symptoms of Attention Deficit Hyperactivity Disorder in Children and Adults: Which Is More Valid?

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The assessment of attention deficit hyperactivity disorder (ADHD) in adults has been a source of controversy. The authors tested competing ideas by evaluating familial transmission among adult and nonadult relatives of ADHD children. They analyzed ADHD symptom data collected by structured interviews from the members of 280 ADHD and 242 non-ADHD families. For both past and current symptoms, both the boys' and girls' families showed significantly more familial aggregation for adult relatives than for nonadult relatives. The results were similar for inattentive and hyperactive–impulsive symptoms and for relatives with and without psychiatric comorbidity. The results provide further evidence for the validity of adult ADHD and support the intriguing idea that, from a familial perspective, the assessment of ADHD may be more valid in adults than in children. They do not support the idea that parents of ADHD children are biased to report ADHD symptoms in themselves because of their exposure to an ADHD child.

Despite media attention and the success of popular books, investigators continue to debate the validity of attention deficit hyperactivity disorder (ADHD) in adults. Some assert that most cases of ADHD remit in adulthood; they conclude that adult ADHD is very rare (Hill & Schoener, 1996; Shaffer, 1994). If so, most referred cases must be due to referral artifacts or missed differential diagnoses. In contrast, others claim that many cases of ADHD persist into adulthood and that ADHD in adults should be considered a valid disorder (Barkley, 1997; Murphy & Barkley, 1996a; Spencer, Biederman, Wilens, & Faraone, 1994).

Given this skepticism about the validity of adult ADHD, researchers have sought to assess the validity of the disorder using the criteria of Robins and Guze (1970). In their view, the validity of any psychiatric disorder derives not from a single study, but from a pattern of consistent data. For psychiatric disorders, standard validation criteria include clinical correlates, treatment response, laboratory studies, follow-up studies, and family history. Our prior reviews of these validity criteria suggest ADHD may be a valid disorder (Faraone, 2000; Spencer et al., 1994; Spencer, Biederman, Wilens, & Faraone, 1998).

If adult ADHD is valid, we would expect the children of ADHD adults to have an elevated prevalence of ADHD (Faraone, Tsuang, & Tsuang, 1999; Faraone & Tsuang, 1995). This has been found by the two extant family studies of adult ADHD (Biederman, et al., 1995; Manshadi, Lippmann, O'Daniel, & Blackman, 1983). These studies both produced the same intriguing result: The risk of ADHD among children of ADHD adults was much higher than the risk for ADHD among relatives of children with ADHD. For example, we found a 57% prevalence of ADHD among children of ADHD adults, which was much higher than the 15% prevalence of ADHD among siblings of ADHD children (Biederman et al., 1995). These results are somewhat counterintuitive. If adult ADHD is an uncertain diagnosis, fraught with the difficulties of retrospective recall and self-referral biases, there should be more false positive cases of ADHD among adults than among children. If that were the case, then evidence for familial transmission should be lower in families sampled through adults compared with those sampled through children. Yet the opposite is true.

The high familial loading of adult ADHD suggests that genes (or other familial risk factors) may play a smaller role in the etiology of remitting ADHD than they do for persistent ADHD. We tested this persistence hypothesis in two ways. In a prospective study, we examined 140 ADHD boys and 120 non-ADHD boys at a baseline assessment and completed a 4-year follow-up study. By

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mid-adolescence, 85% of the ADHD boys continued to have the disorder; 15% remitted. The prevalence of ADHD was significantly higher among the relatives of persistent ADHD probands compared with relatives of remitted ADHD probands (Biederman, et al., 1996). Parents of persistent ADHD probands were 20 times more likely to have ADHD than parents of controls whereas parents of nonpersistent ADHD probands showed only a fivefold increased risk. Similarly, siblings of persistent ADHD probands were 17 times more likely to have ADHD than siblings of controls, whereas siblings of nonpersistent ADHD probands showed only a four-fold increased risk (Faraone, Biederman, & Monuteaux, 2000). In a retrospective study, we compared ADHD adolescents having retrospectively reported childhood onset ADHD with ADHD children. The relatives of adolescent probands had higher rates of ADHD compared with the relatives of child probands (Biederman et al., 1998). Thus, a prospective study of children and a retrospective study of adolescents suggest that, when ADHD persists into adolescence and adulthood, it is highly familial.

Taken together, these data suggest that, from a familial perspective, not only is the adult ADHD diagnosis valid, but it might actually be more valid than the childhood diagnosis. By more valid we mean that a validity coefficient indexing the familial transmission of ADHD would be greater for adults than children. This idea makes a straightforward prediction: When selecting families through ADHD children, the evidence for familial transmission should be greater when examining the risk to adult relatives than it is when examining the risk to non-adult relatives. Although this prediction is straightforward, testing it is complicated by uncertainties about the correct symptoms threshold for defining ADHD in adults.

Although extensive psychometric studies provided empirical support for the symptom thresholds used to diagnose ADHD in children (Lahey et al., 1994), little is known about the validity of these symptom thresholds for adult ADHD. Barkley (1998) suggested that applying current ADHD criteria to adults is not developmentally sensitive. The fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–IV*; American Psychiatric Association, 1994) recognizes developmental changes in the expression of ADHD in several ways. It cautions diagnosticians that, with maturation, symptoms become less conspicuous. Older children may be restless and fidgety but not overly hyperactive. With age, inattention may predominate as school tasks require increasing levels of attention. The *DSM* also specifies that symptoms are considered present only if they are maladaptive and inconsistent with developmental level. The *DSM–IV* also includes the category of ADHD in partial remission for individuals (especially adolescents and adults) who currently have symptoms but no longer meet full criteria.

The net effect of developmental changes is to make it more difficult for ADHD children to meet criteria for the disorder as they get older. This situation was shown empirically by Murphy and Barkley (1996b), who used self-report rating scales to diagnose ADHD in 720 adults applying for or renewing their drivers licenses in the state of Massachusetts. In this population sample of adults, they found a systematic decrease in the prevalence of ADHD symptoms with age. Although this decrease may indicate true remission of symptoms with age, it may also indicate a measurement problem: reduced sensitivity of ADHD symptoms with age. If the latter is true, then using the same symptom threshold to define deviance at each age will reduce the number of diagnosable cases among older individuals.

This issue was addressed by Heiligenstein, Conyers, Berns, Miller, and Smith (1998). They examined ADHD diagnoses and symptom scores among 448 college students not selected for any psychiatric diagnosis. Four percent of the students met *DSM–IV* criteria for ADHD. They then defined ADHD as deviance from the norm: Students were defined as having ADHD if their total symptom score exceeded the 93rd percentile of the sample. The subjects who met this criterion showed clinically significant symptoms (Heiligenstein, Guenther, Levy, Savino, & Fulwiler, 1999; Heiligenstein & Keeling, 1995). Thus, the authors argued that the *DSM–IV* criteria were too stringent because they did not identify many subjects who were both deviant from the norm and showed symptoms warranting clinical attention.

The choice of a symptom threshold also affects estimates of the persistence of ADHD into adulthood. For example, Fischer (1997) followed 148 hyperactive children for 15 years. At a mean age of 21, the self-report of these participants indicated that only 3% met criteria for ADHD as defined by the revised 3rd edition of the *DSM* (*DSM–III-R*; American Psychiatric Association, 1987). But when they used a psychometric criterion to define ADHD (symptoms exceeding the 93rd percentile of severity of the control group), 25% met criteria for ADHD. We reported similar findings from our longitudinal study of ADHD boys (Biederman, Mick, & Faraone, 2000). We found that by the age of 19, 38% of children had the full ADHD diagnosis, 72% showed persistence of at least one-third of the symptoms required for the diagnosis, and 90% showed evidence of clinically significant impairment.

These considerations have led to the idea that ADHD be recast as a norm-referenced rather than a criterion-referenced diagnosis (Barkley, 1998; Faraone, 2000). This idea suggests that one deal with the diagnosis of ADHD as one does the constructs of adaptive functioning or intelligence. For example, one would not have adults complete the Wechsler Intelligence Scales for Children and then conclude that intelligence increases with age. Instead, one uses different test batteries for different age groups; within a single battery, a score is considered high or low in reference to people of the same age.

These uncertainties about the developmental course of ADHD symptoms raise a key question about the decline of ADHD symptoms over time: Does this decline reflect remission of the disorder, decreases in severity with age, or both? Or is this decline an artifact of measurement because the symptoms used to define ADHD in children are not sensitive[1] measures of ADHD in adulthood? Although the answers to these questions must await future research, the issues they raise suggest that studies of adult ADHD examine the disorder in a manner that attends to the potential reduced sensitivity of the diagnosis in adulthood.

In summary, family studies suggest not only that the adult ADHD diagnosis is valid, but also that it might actually be more valid than the childhood diagnosis (i.e., ADHD that persists into adulthood is more familial than ADHD that remits in childhood). Our prior work has shown (a) children of clinically referred ADHD adults have very high rates of ADHD (Biederman et al.,

[1] In this context, sensitivity is the probability that the diagnosis of ADHD will correctly identify an ADHD adult as having ADHD.

1995), and (b) in both retrospective (Biederman et al., 1998) and prospective (Biederman, Faraone, Milberger, et al., 1996; Faraone, 2000) studies, when ADHD persists into adolescence and young adulthood, it is highly familial.

Our prior studies have addressed the implications of persistence by looking at the relatives of persistent and nonpersistent ADHD probands. They have not, however, examined persistence in the relatives. If our hypothesis about persistence is correct, then evidence for familial transmission should be greater when examining the risk to adult relatives (parents and adult siblings) than it is when examining the risk to nonadult relatives (other siblings). Thus, this report sought to extend our prior work by testing this prediction. Moreover, given the uncertainties about how to diagnose ADHD in adults, the present article sought to address this issue in a manner that would deal with the effects of varying symptom thresholds. We did so by using receiver operating characteristic (ROC) analyses of ADHD symptoms among relatives ascertained in our two previously reported family studies of ADHD (Biederman et al., 1999; Biederman et al., 1992).

Method

Participants

We analyzed data from two family–genetic studies of ADHD that we have presented in previous publications (Biederman et al., 1999, 1992). Our family study of ADHD boys selected two groups of index male participants: 140 ADHD probands and 120 boys without ADHD comparisons. These groups had 454 and 368 first-degree biological relatives, respectively. Our family study of ADHD girls studied two groups of index female participants: 140 ADHD probands and 122 girls who did not have ADHD. These groups had 417 and 369 first-degree biological relatives, respectively, who provided data.

All probands were between the ages of 6 and 17. Potential probands were excluded if they had been adopted or if their nuclear family was not available for study. We excluded children if they had major sensorimotor handicaps (e.g., paralysis, deafness, blindness), psychosis, autism, or an estimated Full-Scale IQ score less than 80. Also, to minimize the potential confounds of extreme social adversity, we excluded participants from the lowest Hollingshead–Redlich socioeconomic class. Each of the ADHD probands met diagnostic criteria for current ADHD at the time of the clinical referral; at the time of recruitment, each had active symptoms of the disorder.

Two independent sources provided the proband children. We selected psychiatrically referred ADHD probands from consecutive referrals to the Pediatric Psychopharmacology Unit at the Massachusetts General Hospital (MGH). At MGH, we recruited normal controls from the Pediatric Ambulatory Service. The Harvard Community Health Plan provided both pediatrically referred ADHD and control probands. We screened controls only for the presence of ADHD.

Procedure

Psychiatric assessments of participants and siblings relied on the Schedule for Affective Disorders and Schizophrenia for School-Age Children: Epidemiologic Version (Kiddie SADS-E; Orvaschel & Puig-Antich, 1987). For the probands nonadult siblings, and adult siblings, we collected maternal- and self-reports of symptoms, except for children younger than 12 years of age, who were not directly interviewed. The assessment personnel were blind to participant diagnosis and ascertainment site. For parents, we collected self-reports of symptoms using the Structured Clinical Interview for *DSM–III–R* (SCID; Spitzer, Williams, Gibbon, & First, 1990), supplemented with modules from the Kiddie SADS-E covering childhood diagnoses. We collected data on nearly all the parents: 99% for control boys, 100% for boys with ADHD, 98% for control girls, and 98% for girls with ADHD. Assessments of parents were masked with regard to proband diagnosis. All parents signed a written consent form prior to participation in the study.

To be given a diagnosis of adult ADHD, the participant must have met the following criteria: (a) On the basis of their retrospective report, participants must have met *DSM–III–R* criteria for a diagnosis of ADHD by the age of 7; (b) they must have had at least five *DSM–III–R* symptoms of ADHD at the time of assessment; and (c) they must have described a chronic course of ADHD symptoms from childhood to adulthood. To elicit ADHD symptoms, we used the ADHD module from the Kiddie- SADS-E, wording questions in the past tense. Thus, the symptoms used to assess childhood ADHD and current adult ADHD were identical. We assessed chronicity of symptoms by inquiring about the presence of symptoms during childhood, adolescence, and adulthood.

The five interviewers had undergraduate degrees in psychology; they were trained to high levels of interrater reliability. The principal investigator, Joseph Biederman supervised the interviewers throughout the study. We computed kappa coefficients of agreement by having three experienced, board-certified child and adult psychiatrists diagnose participants from audio-taped interviews made by the assessment staff. On the basis of 61 interviews, all disorders achieved kappas higher than 0.82, with the exception of alcohol abuse, which was 0.75. The mean kappa was 0.90. Kappas of 1.0 were obtained for ADHD (95% confidence interval: 0.8–1.0), substance abuse (0.7–1.0), and substance dependence (0.7–1.0). Although these kappa coefficients showed high interrater agreement, they did not demonstrate the validity of the diagnoses. Full information regarding kappa coefficients is available on request.

A committee of four board-certified child and adult psychiatrists resolved all diagnostic uncertainties. The committee members were blind to the participants' ascertainment group, ascertainment site, and all data collected from other family members. For children older than 12 years, the diagnosticians combined data from interviews of children with data from interviews of mothers about their children by considering a diagnostic criterion positive if endorsed in either interview.

Statistical Analysis

Our analyses sought to test one main hypothesis: When selecting families through ADHD children, the evidence for familial transmission should be greater for the risk to adult relatives than for the risk to nonadult relatives. We used a combination of logistic regression and ROC analysis to provide a full examination of how varying symptom thresholds might affect evidence for familial transmission. Before testing our study hypothesis, we examined demographic and clinical features of the sample and determined if these were associated with either ADHD or age of relative. These analyses used logistic regression (for binary outcomes) or linear regression (for continuous outcomes).

To test our hypothesis, we estimated several sets of logistic regression models separately for boy and girl proband families. Each model used proband status (ADHD and controls) as the dependent variable and number of ADHD symptoms in relatives as the independent variable. We let p denote the probability that the relative comes from an ADHD family (i.e., a family ascertained through a participant with ADHD). Then, $\log[p/(1-p)]$ is the logit of p. Logistic regression models the logit as a linear function of the number of ADHD symptoms in the adult and nonadult relatives. For each model, the magnitude and significance of familial transmission was assessed by the estimated coefficient for the symptom variable as a predictor of group status. This regression coefficient (β_i) indicates the change in the logit associated with an increase of one in the relative's ADHD symptom count. $\text{Exp}(\beta_i)$ is the odds ratio, that is, the ratio of the odds that a relative with N symptoms comes from an ADHD family compared with a relative who has $N-1$ symptoms. Thus, the odds ratio is an index of the

degree of familial transmission. An odds ratio of 1.0 signifies no familial transmission. As the odds ratio increases above 1.0, so does evidence for the familial transmission of ADHD. We assessed the significance of β_i (and hence of the odds ratio) by a z score ($\beta_i/SE[\beta_1]$).

We tested two logistic regression models for current ADHD symptoms. Model 1 was as follows: number of current ADHD symptoms for adult relatives (age greater than 18) as a predictor of proband diagnosis. Model 2 was as follows: number of current ADHD symptoms for nonadult relatives as a predictor of proband diagnosis. Our hypothesis predicted that the odds ratios that index familial transmission should be greater for Model 1 compared with Model 2. We tested this hypothesis using a likelihood ratio chi-square test. We also tested a parallel set of models for the relative's past history of ADHD symptoms. Then, to explore the effects of ascertainment site and psychiatric comorbidity, we repeated these analyses after stratifying the participants on these variables. We also separately examined past and current symptoms for the two main clusters of ADHD symptoms: hyperactive–impulsive and inattentive.

For analyses of current symptoms, we excluded child or adult relatives who were being treated for ADHD at the time of assessment. The numbers (and percentages of total relatives) excluded were as follows: 33 child relatives of girl probands (15%), 16 adult relatives of girl probands (3%), 10 child relatives of boy probands (4%), and 5 adult relatives of boy probands (1%).

Because relatives from the same family are not statistically independent from one another, standard logistic regression methods will produce inaccurate p values. To address this intrafamily clustering, we adjusted variance estimates using Huber's (1967) formula as implemented in STATA (Stata Corporation, 1997). This formula is a theoretical bootstrap that produces robust statistical tests. The method works by entering the cluster scores (i.e., sum of scores within families) into the formula for the estimate of variance. The resulting p values are valid even when observations are not statistically independent of one another.

We used the logistic regression models to generate ROC curves as follows. Each model allows us to compute two statistics that assess the degree to which each possible number of ADHD symptoms in relatives discriminates the relatives of ADHD probands from the relatives of control probands. These statistics are sensitivity and the false positive rate. In this context, *sensitivity* refers to the proportion of ADHD probands that have a relative with N or more symptoms. The *false positive rate* is the proportion of control probands that have a relative with N or more symptoms. We can then compute a pair of sensitivity and false positive rate statistics for each number of ADHD symptoms. To plot the ROC curve, all possible pairs of sensitivity and false positive rate values are joined into one curve by plotting sensitivity on the vertical axis and the false positive rate on the horizontal axis (McNeil & Hanley, 1984).

If ADHD symptoms are familial, then the ROC curve should rise above the diagonal line connecting the [0, 0] and [1, 1] points of the ROC graph. If, as our hypothesis predicts, symptoms in adult relatives are more familial than symptoms in child relatives, then the ROC curve for adults should rise above the curve for children. That is, the area under the adult relative's curve should be greater than the area under the child relative's curve. We tested this difference using the method of Hanley and McNeil (1983).

Results

Clinical and Demographic Data

Table 1 presents the clinical and demographic features of the adult and child relatives of ADHD boys. It shows that, compared with control probands the relatives of ADHD probands came from lower social classes and less intact families. Adult relatives did not differ from child relatives on these demographic features. Table 1 also shows that the relatives of ADHD probands were at higher risk for ADHD, conduct disorder, major depression, and anxiety disorders. Major depression and anxiety disorders were more prevalent in the adult sample, and ADHD was more prevalent in the child sample. This latter effect was seen for both the hyperactive–impulsive and inattentive total symptom counts, whether measured currently or in the past.

Table 2 presents the same information for relatives of ADHD girls. Relatives of ADHD probands were slightly younger than relatives of controls, and the adult relatives came from a somewhat lower social class than the child relatives. Relatives of ADHD girl probands were at higher risk for ADHD, major depression, and anxiety disorders but not conduct disorder. ADHD was more prevalent in the child compared with the adult sample of relatives. These lower rates of ADHD were seen for both the hyperactive–impulsive and inattentive total symptom counts whether measured currently or in the past.

Analyses of Current Symptoms

Table 3 provides a summary of the logistic regression analyses. For the analyses of current symptoms, the results were identical for the relatives of boy and girl probands. Both showed statistically significant familial aggregation for adult relatives but not for nonadult relatives. Moreover, for both samples, the differences between the adult and nonadult samples were statistically significant: boys, $\chi^2_1(1, N = 776) = 10.9$, $p = .001$; girls, $\chi^2_1(1, N = 735) = 11.6$, $p < .001$. The pattern of results can be seen in the odds ratios. These ratios indicate the increased odds of being a relative of an ADHD proband afforded by an increment of one in the level of the relative's ADHD symptoms count.

The implications of the logistic regression analyses can be seen in Figure 1. Panel A shows ROC curves separately for adult and nonadult relatives of boy probands. If ADHD were not familial, the points on the ROC curve would fall on the diagonal line connecting the [0, 0] and [1, 1] points. Points lying above the diagonal provide evidence for familial transmission.

Each point on the graph indicates, for a different diagnostic threshold, N, the proportion of ADHD probands that have a relative with N or more symptoms (the vertical axis) and the proportion of non-ADHD probands that have a relative with N or more symptoms (the horizontal axis). For example, the point labeled "A" in Panel A of Figure 1 shows that 30% of ADHD probands have an adult relative with two or more current symptoms. In contrast, only 10% of control probands have an adult relative with two or more current symptoms. If we move down the ROC curve toward the [0, 0] point, the next point we encounter tells us that 21% of ADHD probands, but only 6% of controls, have an adult relative with three or more current symptoms. The point labeled "B" shows that 30% of ADHD and 25% of control probands have a nonadult relative with two or more current symptoms. Moving down the ROC curve toward the [0, 0] point, the next point we encounter tells us that 23% of ADHD probands and 19% of controls have a nonadult relative with three or more current symptoms.

Consistent with the logistic regression analyses, the area under the adult ROC curve is greater than the area under the nonadult curve, indicating that ADHD is more familial in the adult compared with the nonadult relatives ($z = 3.0$, $p = .001$). The data from the sample of families selected through girl probands show

Table 1
Demographic and Clinical Features of Adult and Child Relatives of Male Probands, Stratified by ADHD Status

Variable	Control families						ADHD families						Statistical analyses	
	Child relatives (n = 100)			Adult relatives (n = 268)			Child relatives (n = 139)			Adult relatives (n = 315)			Effect of ADHD status	Effect of relative age category
Demographic characteristics														
Age (years)	11.2	2.9		39.8	8.3		11.0	3.1		38.7	8.3		$t(260) = -1.6$, $p = .103$	$t(260) = 78.1$, $p < .001$
SES[a]	1.5	0.8		1.5	0.7		1.9	1.0		1.9	1.0		$z = 2.8$, $p = .005$	$z = -0.4$, $p = .687$
Intactness (N%)	89	89		223	83		105	76		227	72		$z = -2.3$, $p = .022$	$z = -1.7$, $p = .091$
Psychiatric diagnoses (N%)														
Conduct disorder	2	2		15	6		11	8		33	11		$z = 2.6$, $p = .011$	$z = 1.6$, $p = .100$
Major depression[b]	5	5		18	7		13	9		50	16		$z = 3.3$, $p = .001$	$z = 2.1$, $p = .036$
Multiple anxiety disorder[c]	9	9		34	13		19	14		75	24		$z = 3.5$, $p = .001$	$z = 2.7$, $p = .007$
ADHD	6	6		6	2		27	19		45	15		$z = 4.2$, $p < .001$	$z = 2.1$, $p = .040$
ADHD symptoms														
Total symptoms, past	2.0	2.9	0–14	1.1	1.9	0–11	3.4	4.1	0–14	3.0	3.7	0–14	$t(260) = 6.6$, $p < .001$	$t(260) = -2.5$, $p = .015$
Total symptoms, current	1.3	2.4	0–14	0.5	1.1	0–8	2.4	3.5	0–13	1.7	2.7	0–14	$t(260) = 5.2$, $p < .001$	$t(260) = -2.9$, $p = .004$
Inattentive symptoms, past	1.0	1.6	0–6	0.6	1.2	0–6	1.6	2.1	0–6	1.5	1.9	0–6	$t(260) = 6.0$, $p < .001$	$t(260) = -1.9$, $p = .059$
Inattentive symptoms, current	0.7	1.3	0–6	0.3	0.8	0–6	1.2	1.8	0–6	0.8	1.5	0–6	$t(260) = 4.7$, $p < .001$	$t(260) = -2.6$, $p = .009$
Hyperactive–impulsive symptoms, past	1.0	1.7	0–8	0.6	1.1	0–6	1.8	2.3	0–8	1.5	2.1	0–8	$t(260) = 6.2$, $p < .001$	$t(260) = -2.5$, $p = .014$
Hyperactive–impulsive symptoms, current	0.7	1.4	0–8	0.2	0.6	0–4	1.2	1.9	0–8	0.8	1.5	0–8	$t(260) = 4.8$, $p < .001$	$t(260) = -2.6$, $p = .010$

Note. Entries in the table represent means and standard deviations for age and socioeconomic status (SES); frequency (%) for intactness and psychiatric diagnoses; and means, standard deviations, and minimum–maximum value for ADHD symptoms. ADHD = attention deficit hyperactivity disorder, as determined by the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.); Min.–max. values = range of minimum to maximum values.
[a] SES, higher values correspond to lower social class levels. [b] Major depression (MD) = MD with severe impairment. [c] Multiple anxiety disorder represents ≥2 anxiety disorders.

Table 2
Demographic and Clinical Features of Adult and Child Relatives of Female Probands, Stratified by ADHD Status

Variable	Control families: Child relatives (n = 104)			Control families: Adult relatives (n = 265)			ADHD families: Child relatives (n = 110)			ADHD families: Adult relatives (n = 307)			Statistical analysis: Effect of ADHD status	Statistical analysis: Effect of relative age category
Demographic characteristics														
Age	11.5	3.2		41.2	9.0		11.2	3.5		39.5	8.7		$t(262) = -2.5$, $p = .012$	$t(262) = 71.3$, $p < .001$
SES[a]	1.8	0.9		1.7	0.8		2.0	0.9		1.9	0.9		$z = 1.5$, $p = .144$	$z = -1.9$, $p = .053$
Intactness (N%)	86	83		223	84		97	88		227	74		$z = -1.2$, $p = .233$	$z = -2.2$, $p = .027$
Psychiatric diagnoses (N%)														
Conduct disorder	3	3		13	5		8	7		21	7		$z = 1.5$, $p = .125$	$z = 0.4$, $p = .665$
Major depression[b]	4	4		20	8		14	13		38	12		$z = 2.5$, $p = .012$	$z = 0.4$, $p = .510$
Multiple anxiety disorder[c]	8	8		27	10		20	18		58	19		$z = 3.2$, $p = .001$	$z = 0.6$, $p = .575$
ADHD	15	14		8	3		32	29		70	23		$z = 6.1$, $p < .001$	$z = -2.2$, $p = .026$
ADHD symptoms														
Total symptoms, past	3.2	4.0	0–14	1.6	2.5	0–12	4.8	4.8	0–14	4.2	4.3	0–14	$t(262) = 7.7$, $p < .001$	$t(262) = -3.7$, $p < .001$
Total symptoms, current	2.6	3.6	0–13	1.0	1.9	0–10	3.8	4.2	0–14	2.7	3.5	0–13	$t(262) = 5.3$, $p < .001$	$t(262) = -2.4$, $p = .017$
Inattentive symptoms, past	1.7	2.1	0–6	0.8	1.4	0–6	2.3	2.3	0–6	2.2	2.3	0–6	$t(262) = 7.3$, $p < .001$	$t(262) = -3.3$, $p = .001$
Inattentive symptoms, current	1.5	2.0	0–6	0.5	1.2	0–6	1.9	2.2	0–6	1.5	2.0	0–6	$t(262) = 5.5$, $p < .001$	$t(262) = -2.3$, $p = .024$
Hyperactive–impulsive symptoms, past	1.5	2.3	0–8	0.7	1.3	0–7	2.5	2.8	0–8	2.1	2.4	0–8	$t(262) = 7.1$, $p < .001$	$t(262) = -3.6$, $p < .001$
Hyperactive–impulsive symptoms, current	1.1	2.0	0–8	0.5	1.0	0–6	1.9	2.3	0–8	1.2	1.8	0–7	$t(262) = 4.1$, $p < .001$	$t(262) = -1.9$, $p = .053$

Note. Entries in the table represent means and standard deviations for age and socioeconomic status (SES); frequency (%) for intactness and psychiatric diagnoses; and means, standard deviations, and minimum–maximum value for ADHD symptoms. ADHD = attention deficit hyperactivity disorder, as determined by the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.) Min.–max. values = range of minimum to maximum values.
[a] SES higher values correspond to lower social class levels. [b] Major depression (MD) = MD with severe impairment. [c] Multiple anxiety disorder represents ≥2 anxiety disorders.

Table 3
Results of Logistic Regression Models by Gender of Proband

Description of model			Statistical results		
Sample	Source of symptoms	Type of relative	Odds ratio	z	p
Boys	Current	Adult	1.4	5.2	<.001
	Current	Nonadult	1.1	1.6	.12
	Past	Adult	1.3	6.3	<.001
	Past	Nonadult	1.1	2.9	.003
Girls	Current	Adult	1.3	6.0	<.001
	Current	Nonadult	1.0	0.4	.68
	Past	Adult	1.3	7.7	<.001
	Past	Nonadult	1.1	2.7	.008

the same pattern of results (see Panel B of Figure 1; $z = 3.4$, $p < .001$).

Analyses of Past Symptoms

For the analyses of past symptoms, Table 3 shows that the results for the boys and girls samples were very similar. Both showed statistically significant familial aggregation for both adult and nonadult relatives. Notable was that the odds ratios show that the direction of effect was similar for boys and girls. Both showed greater odds ratios for adult compared with nonadult relatives. These differences between the adult and nonadult samples were statistically significant: boys, $\chi^2_1(1, N = 791) = 5.3$, $p = .021$; girls, $\chi^2_1(1, N = 784) = 11.4$, $p < .001$.

Figure 2 shows the ROC curves for past symptoms. As we saw for current symptoms, the results from the boys and girls samples are similar. The areas under both adult ROC curves are greater than the areas under the corresponding nonadult curves, indicating that ADHD is more familial in the adult compared with the nonadult relatives (boys: $z = 2.2$, $p = .014$; girls: $z = 3.1$, $p < .001$).

Analyses of Ascertainment Source, Symptom Clusters, and Psychiatric Comorbidity

Table 4 shows that the results were very similar when we stratified the samples by ascertainment source using analyses that combined relatives of boy and girl probands. With the exception of current symptoms for nonadult relatives, both clusters of symptoms showed statistically significant familial aggregation for both adult and nonadult relatives. The odds ratios show that the direction of effect was similar for both ascertainment sites. Both show greater odds ratios for adult compared with nonadult relatives. These differences between the adult and nonadult samples were statistically significant: MGH current symptoms, $\chi^2_1(1, N = 653) = 24.4$, $p < .001$; MGH past symptoms, $\chi^2_1(1, N = 700) = 14.1$, $p < .001$; health maintenance organization (HMO) current symptoms, $\chi^2_1(1, N = 858) = 6.2$, $p = .013$; HMO past symptoms, $\chi^2_1(1, N = 875) = 10.1$, $p = .002$.

Table 5 shows that the results were very similar when inattentive and hyperactive–impulsive symptoms were considered separately. The Appendix shows how these clusters were defined with *DSM–III–R* symptoms. These analyses combine relatives of boy and girl probands. With the exception of current symptoms for nonadult relatives, both clusters of symptoms showed statistically significant familial aggregation for both adult and nonadult relatives. The odds ratios show that, for both types of symptoms, the odds ratios were greater for adult compared with nonadult relatives. These differences between the adult and nonadult samples were statistically significant: current inattentive symptoms, $\chi^2_1(1, N = 1{,}510) = 21.5$, $p < .001$; past inattentive symptoms, $\chi^2_1(1, N = 1{,}575) = 18.7$, $p < .001$; current hyperactive–impulsive symptoms, $\chi^2_1(1, N = 1{,}511) = 22.9$, $p < .001$; past hyperactive–impulsive symptoms, $\chi^2_1(1, N = 1{,}575) = 18.1$, $p < .001$.

Table 6 shows the results stratified by psychiatric comorbidity. For the analyses labeled "No Diagnosis," we included only relatives who did not have any of the following disorders: conduct disorder, major depressive disorder with severe impairment, multiple anxiety disorder, or antisocial personality disorder. Relatives with one or more of these diagnoses were included in the analyses labeled "At Least One Diagnosis." These analyses combined relatives of boy and girl probands. With the exception of current symptoms for nonadult relatives, both clusters of symptoms showed statistically significant familial aggregation for both adult and nonadult relatives. The odds ratios show that the direction of effect was similar for both the comorbid and non-comorbid groups. Both show greater odds ratios for adult compared with nonadult relatives. These differences between the adult and nonadult samples were statistically significant—no diagnoses, current symptoms, $\chi^2_1(1, N = 1{,}090) = 13.6$, $p < .001$; no diagnoses, past symptoms, $\chi^2_1(1, N = 1{,}120) = 15.6$, $p < .001$; and one or more diagnoses, current symptoms, $\chi^2_1(1, N = 386) = 5.9$, $p = .015$—with the exception of the case of past symptoms for relatives having one or more diagnosis, $\chi^2_1(1, N = 418) = 2.5$, $p = 0.114$.

Analyses of Reporter Bias Effects

Because adult relatives of ADHD children may be aware of the ADHD symptoms in their proband children, they may be biased to report ADHD symptoms in themselves. If so, our finding of greater familiality in adult relatives might be due to this reporter bias effect. This type of reporter bias makes a testable prediction: The adult relatives of ADHD children should have a greater number of symptoms than the nonadult relatives. Inspection of Tables 1 and 2 shows that this prediction does not hold. For both the boys and girls samples, the mean number of symptoms reported by adult relatives of ADHD probands was nonsignificantly

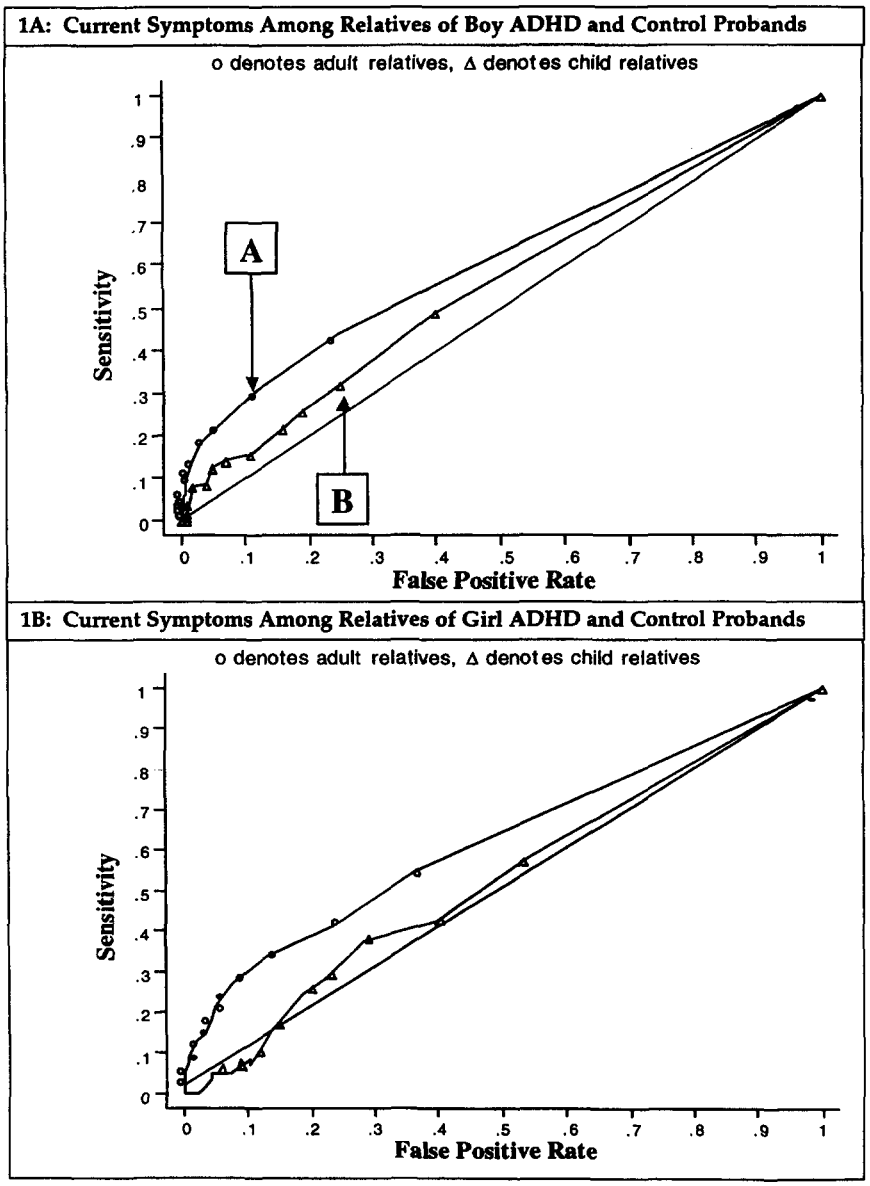

Figure 1. Receiver operating characteristic curves for current ADHD symptoms among relatives of boy and girl ADHD participants. ADHD = attention deficit hyperactivity disorder.

less than the mean number of symptoms reported by nonadult relatives. This was true for current and past symptoms and for inattentive and hyperactive–impulsive symptoms (all $ps > .05$).

Discussion

Our results support the study hypothesis: When selecting families through ADHD children, evidence for familial transmission is greater when examining ADHD symptoms in adult relatives than it is when examining symptoms in nonadult relatives. Thus, our results provide further evidence for the validity of adult ADHD and continue to support the intriguing idea that, from a familial perspective, the diagnosis of ADHD is more valid in adults than it is in children.

The pattern of results supporting this hypothesis was seen for both current and past assessments of total ADHD symptoms and for inattentive and hyperactive–impulsive symptoms considered separately. Moreover, our results cannot be accounted for by gender, psychiatric comorbidity, or ascertainment source because we found the same pattern of results for the subgroups defined by these variables.

We also considered the possibility that ADHD in children biases the self-reports of ADHD in their adult relatives. The adult relatives of ADHD children are usually aware of the ADHD symptoms in the child. That knowledge may bias them to report ADHD symptoms in themselves. If that occurs, then the rates of ADHD among adult relatives of ADHD children would be spuriously

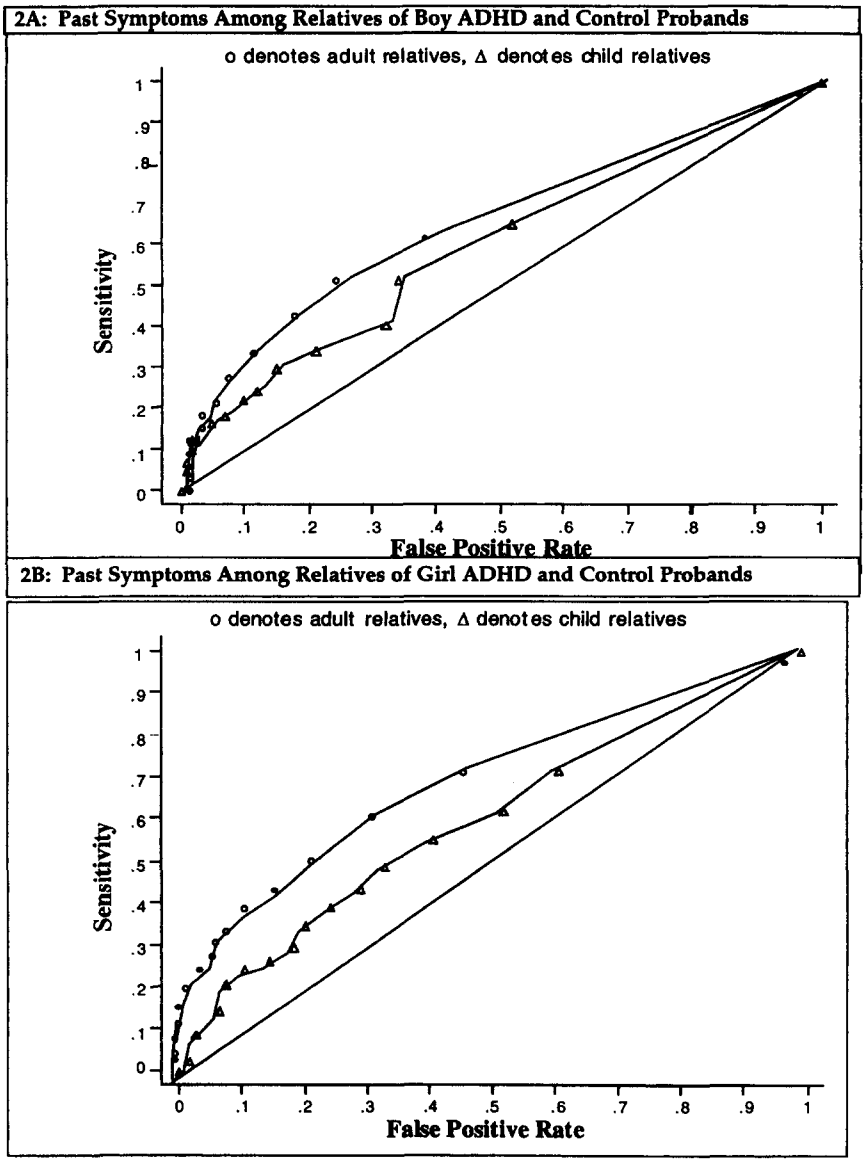

Figure 2. Receiver operating characteristic curves for past ADHD symptoms among relatives of boy and girl ADHD participants. ADHD = attention deficit hyperactivity disorder.

high, leading to the incorrect conclusion that adult ADHD is more familial than child ADHD. But if ADHD adults are biased to overreport symptoms, then the adult relatives of ADHD children should have had a greater number of symptoms than the child relatives. That was not the case. In fact, the adult relatives tended to report fewer symptoms, although the difference was not statistically significant.

We have made two statements that appear contradictory but are not: (a) Adult ADHD symptoms are more familial than child ADHD symptoms, and (b) the number of ADHD symptoms does not differ between adult and child relatives. The statements do not contradict one another because any index of familial transmission must measure differences between two quantities: the number of ADHD symptoms in ADHD relatives and the number of symptoms in control relatives (Faraone et al., 1999). Our finding that ADHD is more familial among adult relatives is due to symptom differences between child and adult relatives of controls. Because the adult relatives of controls had fewer symptoms than the child relatives of controls, the ADHD versus control comparison is greater for adult relatives.

This feature of our data can be seen by inspecting the ROC curves. Recall that each successive point on the ROC shows the sensitivity and false positive rate for successive ADHD symptom thresholds. For example, the point labeled "A" in Panel A of Figure 1 shows that 30% of ADHD probands and 10% of control probands have an adult relative with two or more current symptoms. Compare this point with the point labeled "B," which shows that 30% of ADHD and 25% of control probands have a nonadult

Table 4
Results of Logistic Regression Models by Ascertainment Source

Description of model			Statistical results		
Source	Source of symptoms	Type of relative	Odds ratio	*z*	*p*
HMO	Current	Adult	1.2	5.2	<.001
	Current	Nonadult	1.1	1.2	.231
	Past	Adult	1.2	6.7	<.001
	Past	Nonadult	1.1	1.4	.153
MGH	Current	Adult	1.4	5.6	<.001
	Current	Nonadult	1.0	0.3	.735
	Past	Adult	1.3	6.9	<.001
	Past	Nonadult	1.1	3.4	.001

Note. HMO = health maintenance organization; MGH = Massachusetts General Hospital.

relative with two or more current symptoms. These points tell us that, for the identical symptom threshold used to define ADHD (two or more), the sensitivity for adult relatives is identical to that for child relatives. In contrast, the false positive rate for child relatives is more than twofold that of adult relatives.

If we move down the ROC curve toward the [0, 0] point, the next point we encounter on the adult relative curve tells us that 21% of ADHD probands but only 6% of controls, have an adult relative with three or more current symptoms. Moving down the child ROC curve, we see that 23% of ADHD probands and 19% of controls have a nonadult relative with three or more current symptoms. Thus, at the threshold of three or more symptoms, child and adult relatives have a similar sensitivity. But, as we saw for points A and B, the child relatives have a greater false positive rate than the adult relatives.

The false positive rate for each symptom threshold is the proportion of control probands that have a relative whose symptoms exceed the threshold. Thus, from a familial perspective, we have shown that the diagnosis of ADHD yields more false positives in nonadult than adult relatives. This idea is counterintuitive given contemporary skepticism about the diagnosis of adult ADHD (Shaffer, 1994). If the self-report of adult ADHD is heavily influenced by recall biases and reports in the media, we should have found it to be less valid and more prone to false positive reports than the diagnosis of ADHD among youths.

Although our data cannot rule out the possibility of some biased reports of ADHD in adults, our evidence against reporter bias is consistent with our prior report from a different sample. In that study, we compared symptom rates between 26 clinically referred ADHD adults who had ADHD children and 49 clinically referred ADHD adults who did not have ADHD children (Faraone, Biederman, & Mick, 1997). We hypothesized the following: If having an ADHD child biased an adult to report ADHD symptoms, then ADHD adults having ADHD children should have reported more symptoms than ADHD adults who did not have ADHD children. We rejected that hypothesis by showing that the number of symptoms reported by ADHD adults did not differ between those who did and did not have ADHD children. Moreover, no individual symptom was more frequent among the ADHD adults who had ADHD children compared with those who did not have ADHD children. Thus, having an ADHD child did not bias ADHD adults to overreport ADHD symptoms.

Our results also address the idea that ADHD should be recast as a norm-referenced rather than a criterion-referenced diagnosis (Barkley, 1998; Faraone, 2000). As we reviewed in the introduction, some have argued that the symptom threshold for diagnosing adult ADHD should be lower than that for diagnosing ADHD in youths. The ROC curves addressed this issue because if a symptom threshold that defines a specific point also defines a familial form of ADHD, then that point should lie above the diagonal line connecting the [0, 0] and [1, 1] points. All of the four curves for adult relatives notably show the same finding: All of the points on the ROC lie above the diagonal. Thus, from a familial perspective, any of these points could be used as a symptom threshold to define

Table 5
Results of Logistic Regression Models for ADHD Symptom Clusters

Description of model			Statistical results		
ADHD symptom cluster	Source of symptoms	Type of relative	Odds ratio	*z*	*p*
Inattentive	Current	Adult	1.5	6.9	<.001
	Current	Nonadult	1.1	1.2	.241
	Past	Adult	1.4	9.3	<.001
	Past	Nonadult	1.1	2.9	.003
Hyperactive/Impulsive	Current	Adult	1.5	6.9	<.001
	Current	Nonadult	1.1	1.0	.318
	Past	Adult	1.5	9.2	<.001
	Past	Nonadult	1.2	3.7	<.001

Note. ADHD = attention deficit hyperactivity disorder.

Table 6
Results of Logistic Regression Models Stratified by Psychiatric Comorbidity

Description of model			Statistical results		
Diagnostic status[a]	Source of symptoms	Type of relative	Odds ratio	z	p
No diagnoses	Current	Adult	1.3	5.1	<.001
	Current	Nonadult	1.0	1.0	.332
	Past	Adult	1.3	7.2	<.001
	Past	Nonadult	1.1	2.1	.038
At least one diagnosis	Current	Adult	1.2	4.0	<.001
	Current	Nonadult	1.0	0.0	.974
	Past	Adult	1.2	5.2	<.001
	Past	Nonadult	1.1	2.0	.047

[a] Refers to the presence of at least one of the following: conduct disorder, major depressive disorder with severe impairment, multiple anxiety disorder, or antisocial personality disorder.

adult ADHD. We do not suggest that clinical symptom thresholds be chosen soley on familial data. Studies addressing the clinical significance of specific thresholds, in the context of appropriate normative data, are needed.

This finding is in line with the idea that we should view ADHD as a dimensional trait, not a discrete category. There are several lines of evidence that suggest strongly that a dimensional perspective on ADHD is valid. First, many studies have found an excellent correspondence between dimensional measures of ADHD (e.g., scales derived from the Child Behavior Checklist, the Conners Scales, and the Strengths and Difficulties Questionnaire) and the categorical diagnosis of ADHD (Biederman et al., 1993; Biederman, Faraone, Mick, Moore, & Lelon, 1996; Bird, et al., 1987; Boyle et al., 1997; Chen, Faraone, Biederman, & Tsuang, 1994; Edelbrock, 1986; Hudziak, 1997). These studies suggest that children with ADHD are at one extreme of a quantitative dimension, and that on this quantitative dimension, there is no obvious bimodality that separates ADHD children from non-ADHD children.

Further support for the validity of a quantitative approach to ADHD is the fact that quantitative measures of ADHD are highly heritable (about 70%) and as heritable as the ADHD diagnosis (Edelbrock, Rende, Plomin, & Thompson, 1995; Goodman & Stevenson, 1989a, 1989b; Judy et al., 1997; Sherman, Iacono, & McGue, 1997; Silberg et al., 1996; Stevenson, 1992; Thapar, Hervas, & McGuffin, 1995).

More important, twin studies have used mathematical modeling techniques to test directly the hypothesis that the clinical diagnosis of ADHD is the extreme of a quantitative trait. Gjone, Stevenson, and Sundet (1996) applied a mathematical model to determine if the heritability of attention problems increased with their severity. If ADHD accounted for the heritability of the broader dimension of attention problems, then heritability should increase with increasing severity. However, Gjone et al. found that heritability did not change with severity. They concluded that there was in the population a continuously distributed dimension of genetic liability to attention problems. A similar approach was applied by Levy et al. (1997). Like Gjone et al., they concluded that, with regard to the genetic components of its etiology, ADHD was best viewed as the extreme of a behavior that varies genetically throughout the entire population rather than as a categorical disorder.

Although available data support the idea that ADHD can be viewed as the extreme expression of a trait that varies quantitatively in the population, clinicians need ADHD symptom thresholds for clinical purposes as much as blood pressure thresholds are needed to define hypertension. Our data cannot provide the optimal symptom threshold for adult ADHD, but they do highlight the need for future research to examine this issue in more detail.

Our results must be considered in the context of some methodologic limitations. We do not know to what degree our findings will generalize to nonreferred ADHD children in the community, and although raters were blind to the diagnosis of the probands parents were not. Moreover, the retrospective assessments of childhood-onset ADHD and the assessments of current ADHD symptoms were made by the same interviewer. Thus, it is possible that the interviewer's knowledge of the adult's childhood symptoms might have contaminated the interviewer's ratings of the adult's current symptoms. This possibility cannot account for the differences we observed between child and adult relatives, but it might have led to an overestimate of current ADHD symptoms in adults who reported childhood symptoms.

Another limitation of our work is that we did not collect parental reports or school records to confirm our retrospective diagnoses of ADHD. It is possible that troubled adults who have been influenced by media reports of ADHD may recognize ADHD symptoms in themselves, and this might bias their recall of childhood symptoms. One safeguard against such bias was our use of a control group, because such biases should have equally affected the adults from ADHD and control families. Nevertheless, future studies of adult ADHD would further strengthen the knowledge base about adult ADHD by collecting informant reports or school records of childhood symptoms.

Despite these limitations, our two family studies of ADHD boys and girls provide further support for the hypothesis that ADHD is more familial in adult samples. This finding shores up the validity of retrospective diagnoses of adult ADHD and suggests that, compared with the childhood diagnosis, the adult diagnosis may be more valid and thus more informative for studies of the disorder.

References

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

Barkley, R. (1997). Age-dependent decline in ADHD: True recovery or statistical illusion? *The ADHD Report, 5,* 1–5.

Barkley, R. A. (1998). *Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment.* New York: Guilford Press.

Biederman, J., Faraone, S. V., Doyle, A., Krifcher Lehman, B., Kraus, I., Perrin, J., & Tsuang, M. T. (1993). Convergence of the Child Behavior Checklist with structured interview-based psychiatric diagnoses of ADHD children with and without hyperactivity. *Journal of Child Psychology and Psychiatry, 34,* 1241–1251.

Biederman, J., Faraone, S. V., Keenan, K., Benjamin, J., Krifcher, B., Moore, C., Sprich-Buckminster, S., Ugaglia, K., Jellinek, M. S., Steingard, R., Spencer, T., Norman, D., Kolodny, R., Kraus, I., Perrin, J., Keller, M. B., & Tsuang, M. T. (1992). Further evidence for family-genetic risk factors in attention deficit hyperactivity disorder: Patterns of comorbidity in probands and relatives psychiatrically and pediatrically referred samples. *Archives of General Psychiatry, 49,* 728–38.

Biederman, J., Faraone, S. V., Mick, E., Moore, P., & Lelon, E. (1996). Child behavior checklist findings further support comorbidity between ADHD and major depression in a referred sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 35,* 734–742.

Biederman, J., Faraone, S. V., Mick, E., Spencer, T., Wilens, T., Keily, K., Guite, J., Ablon, S., Reed, E. D., & Warburton, R. (1995). High risk for attention deficit hyperactivity disorder among children of parents with childhood onset of the disorder: A pilot study. *American Journal of Psychiatry, 152,* 431–435.

Biederman, J., Faraone, S., Mick, E., Williamson, S., Wilens, T., Spencer, T., Weber, W., Jetton, J., Kraus, I., Pert, J., & Zallen, B. (1999). Clinical correlates of ADHD in females: Findings from a large group of pediatrically and psychiatrically referred girls. *Journal of the American Academy of Child and Adolescent Psychiatry, 38,* 966–975.

Biederman, J., Faraone, S. V., Milberger, S., Curtis, S., Chen, L., Marrs, A., Ouellette, C., Moore, P., & Spencer, T. (1996). Predictors of persistence and remission of ADHD: Results from a four-year prospective follow-up study of ADHD children. *Journal of the American Academy of Child and Adolescent Psychiatry, 35,* 343–351.

Biederman, J., Faraone, S. V., Taylor, A., Sienna, M., Williamson, S., & Fine, C. (1998). Diagnostic continuity between child and adolescent ADHD: Findings from a longitudinal clinical sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 37,* 305–313.

Biederman, J., Mick, E., & Faraone, S. V. (2000). Age-dependent decline of attention deficit hyperactivity disorder: Impact of remission definition and symptom subtype. *American Journal of Psychiatry, 157,* 816–818.

Bird, H., Canino, G., Gould, M., Ribera, J., Rubio-Stipec, M., Woodbury, M., Huertas-Goldman, H., & Sesman, M. (1987). Use of the Child Behavior Checklist as a screening instrument for epidemiological research in child psychiatry: Results of a pilot study. *Journal of the American Academy of Child and Adolescent Psychiatry, 26,* 207–213.

Boyle, M., Offord, D., Racine, Y., Szatmari, P., Sanford, M., & Fleming, J. (1997). Adequacy of interviews vs checklists for classifying childhood psychiatric disorder based on parent reports. *Archives of General Psychiatry, 54,* 793–799.

Chen, W., Faraone, S., Biederman, J., & Tsuang, M. (1994). Diagnostic accuracy of the Child Behavior Checklist scales for attention deficit hyperactivity disorder: A receiver-operating characteristic analysis. *Journal of Consulting and Clinical Psychology, 62,* 1017–1025.

Edelbrock, C. (1986). Behavioral ratings of children diagnosed for attention deficit disorder. *Psychiatric Annals, 16,* 36–40.

Edelbrock, C., Rende, R., Plomin, R., & Thompson, L. A. (1995). A twin study of competence and problem behavior in childhood and early adolescence. *Journal of Child Psychology and Psychiatry, 36,* 775–785.

Faraone, S. V. (2000). Attention deficit hyperactivity disorder in adults: Implications for theories of diagnosis. *Current Directions in Psychological Science, 9,* 33–36.

Faraone, S., Biederman, J., & Mick, E. (1997). Symptom reports by adults with attention deficit hyperactivity disorder: Are they influenced by attention deficit hyperactivity disorder in their children? *Journal of Nervous and Mental Diseases, 185,* 583–584.

Faraone, S. V., Biederman, J., & Monuteaux, M. C. (2000). Toward guidelines for pedigree selection in genetic studies of attention deficit hyperactivity disorder. *Genetic Epidemiology, 18,* 1–16.

Faraone, S. V., Tsuang, D., & Tsuang, M. T. (1999). *Genetics and mental disorders: A guide for students, clinicians, and researchers.* New York: Guilford Press.

Faraone, S. V., & Tsuang, M. T. (1995). Methods in psychiatric genetics. In M. Tohen, M. T. Tsuang, & G. E. P. Zahner (Eds.), *Textbook in psychiatric epidemiology* (pp. 81–134). New York: Wiley.

Fischer, M. (1997). The persistence of ADHD into adulthood: It depends on whom you ask. *The ADHD Report, 5,* 8–10.

Gjone, H., Stevenson, J., & Sundet, J. (1996). Genetic influence on parent-reported attention-related problems in a Norwegian general population twin sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 35,* 588–598.

Goodman, R., & Stevenson, J. (1989a). A twin study of hyperactivity: I. An examination of hyperactivity scores and categories derived from Rutter teacher and parent questionnaires. *Journal of Child Psychology and Psychiatry, 30,* 671–689.

Goodman, R., & Stevenson, J. (1989b). A twin study of hyperactivity: II. The aetiological role of genes, family relationships and perinatal adversity. *Journal of Child Psychology and Psychiatry, 30,* 691–709.

Hanley, J. A., & McNeil, B. J. (1983). A method of comparing the areas under receiver operating characteristic curves derived from the same cases. *Radiology, 148,* 839–843.

Heiligenstein, E., Conyers, L. M., Berns, A. R., Miller, M. A., & Smith, M. A. (1998). Preliminary normative data on *DSM-IV* attention deficit hyperactivity disorder in college students. *Journal of American College Health, 46,* 185–188. (Published erratum appears in *Journal of American College Health, 46,* 213)

Heiligenstein, E., Guenther, G., Levy, A., Savino, F., & Fulwiler, J. (1999). Psychological and academic functioning in college students with attention deficit hyperactivity disorder. *Journal of American College Health, 47,* 181–185.

Heiligenstein, E., & Keeling, R. P. (1995). Presentation of unrecognized attention deficit hyperactivity disorder in college students. *Journal of American College Health, 43,* 226–228.

Hill, J., & Schoener, E. (1996). Age-dependent decline of attention deficit hyperactivity disorder. *American Journal of Psychiatry, 153,* 1143–1146.

Huber, P. J. (1967). The behavior of maximum likelihood estimates under non-standard conditions. *Proceedings of the Fifth Berkeley Symposium on Mathematical Statistics and Probability, 1,* 221–233.

Hudziak, J. J. (1997). Identification of phenotypes for molecular genetic studies of common childhood psychopathology. In K. Blum & E. P. Noble (Eds.), *Handbook of psychiatric genetics* (pp. 201–217). Boca Raton, FL: CRC Press, Inc.

Judy, L., Silberg, J., Meyer, J., Maes, H., Simonoff, E., Pickles, A., Rutter, M., Reynolds, C., Heath, A., Truett, K., Meale, M., Erikson, M., Loeber, R., & Hewitt, J. (1997). Genetics and developmental psychopathology: II. The main effects of genes and environment of behavioral problems in the Virginia Twin study of Adolescent behavioral development. *Journal of Child Psychology & Psychiatry & Allied Discipline, 38,* 965–980.

Lahey, B., Applegate, B., McBurnett, K., Biederman, J., Greenhill, L., Hynd, G., Barkley, R., Newcorn, J., Jensen, P., Richters, J., Garfinkel, B., Kerdyk, L., Frick, P., Ollendick, T., Perez, D., Hart, E., Waldman, I., & Shaffer, D. (1994). *DSM-IV* field trials for attention deficit hyperac-

tivity disorder in children and adolescents. *American Journal of Psychiatry, 151,* 1673–1685.

Levy, F., Hay, D., McStephen, M., Wood, C., & Waldman, I. (1997). Attention-deficit hyperactivity disorder: A category or a continuum? Genetic analysis of a large-scale twin study. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 737–744.

Manshadi, M., Lippmann, S., O'Daniel, R., & Blackman, A. (1983). Alcohol abuse and attention deficit disorder. *Journal of Clinical Psychiatry, 44,* 379–380.

McNeil, B. J., & Hanley, J. A. (1984). Statistical approaches to the analysis of receiver operating characteristic (ROC) curves. *Medical Decision Making, 4,* 137–150.

Murphy, K., & Barkley, R. (1996a). Prevalence of *DSM-IV* symptoms of ADHD in adult licensed drivers: Implications for clinical diagnosis. *Journal of Attention Disorders, 1,* 147–161.

Murphy, K., & Barkley, R. A. (1996b). Attention deficit hyperactivity disorder adults: comorbidities and adaptive impairments. *Comprehensive Psychiatry, 37,* 393–401.

Orvaschel, H., & Puig-Antich, J. (1987). *Schedule for Affective Disorders and Schizophrenia for School-Age Children: Epidemiologic 4th Version.* Fort Lauderdale, FL: Nova University, Center for Psychological Study.

Robins, E., & Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry, 126,* 983–987.

Shaffer, D. (1994). Attention deficit hyperactivity disorder in adults. *American Journal of Psychiatry, 151,* 633–638.

Sherman, D., Iacono, W., & McGue, M. (1997). Attention deficit hyperactivity disorder dimensions: A twin study of inattention and impulsivity hyperactivity. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 745–753.

Silberg, J., Rutter, M., Meyer, J., Maes, H., Hewitt, J., Simonoff, E., Pickles, A., & Loeber, R. (1996). Genetic and environmental influences on the covariation between hyperactivity and conduct disturbance in juvenile twins. *Journal of Child Psychology and Psychiatry, 37,* 803–816.

Spencer, T., Biederman, J., Wilens, T., & Faraone, S. (1994). Is attention deficit hyperactivity disorder in adults a valid disorder? *Harvard Review of Psychiatry, 1,* 326–335.

Spencer, T., Biederman, J., Wilens, T. E., & Faraone, S. V. (1998). Adults with attention-deficit/hyperactivity disorder: a controversial diagnosis. *Journal of Clinical Psychiatry, 59,* 59–68.

Spitzer, R. L., Williams, J. B., Gibbon, M., & First, M. B. (1990). *Structured Clinical Interview for DSM-III-R: Non-Patient Edition (SCID-NP, Version 1.0).* Washington, DC: American Psychiatric Press.

Stata Corporation (1997). *Stata reference manual: Release 5.* College Station, TX: Author.

Stevenson, J. (1992). Evidence for a genetic etiology in hyperactivity in children. *Behavior Genetics, 22,* 337–344.

Thapar, A., Hervas, A., & McGuffin, P. (1995). Childhood hyperactivity scores are highly heritable and show sibling competition effects: Twin study evidence. *Behavior Genetics, 25,* 537–544.

Appendix

DSM–III–R ADHD Symptom Clusters

Inattentive symptoms	Hyperactive/impulsive symptoms
Shifts activities	Difficulty remaining seated
Difficulty sustaining attention	Is fidgety
Difficulty following instructions	Has difficulty playing quietly
Easily distracted	Talks excessively
Loses things	Interrupts
Does not listen	Blurts out
	Difficulty waiting turn
	Acts before thinking

Note. *DSM–III–R* = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.); ADHD = attention deficit and hyperactivity disorder.

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