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Childhood Predictors of Adult Attention-Deficit/Hyperactivity Disorder: Results from the World Health Organization World Mental Health Survey Initiative

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Background: Although it is known that childhood attention-deficit/hyperactivity disorder (ADHD) often persists into adulthood, childhood predictors of this persistence have not been widely studied.

Methods: Childhood history of ADHD and adult ADHD were assessed in 10 countries in the World Health Organization World Mental Health Surveys. Logistic regression analysis was used to study associations of retrospectively reported childhood risk factors with adult persistence among the 629 adult respondents with childhood ADHD. Risk factors included age; sex; childhood ADHD symptom profiles, severity, and treatment; comorbid child/adolescent DSM-IV disorders; childhood family adversities; and child/adolescent exposure to traumatic events.

Results: An average of 50% of children with ADHD (range: 32.8%-84.1% across countries) continued to meet DSM-IV criteria for ADHD as adults. Persistence was strongly related to childhood ADHD symptom profile (highest persistence associated with the attentional plus impulsive-hyperactive type, odds ratio [OR] = 12.4, compared with the lowest associated with the impulsive-hyperactive type), symptom severity (OR = 2.0), comorbid major depressive disorder (MDD; OR = 2.2), high comorbidity (\geq 3 child/adolescent disorders in addition to ADHD; OR = 1.7), paternal (but not maternal) anxiety mood disorder (OR = 2.4), and parental antisocial personality disorder (OR = 2.2). A multivariate risk profile of these variables significantly predicts persistence of ADHD into adulthood (area under the receiving operator characteristic curve = .76).

Conclusions: A substantial proportion of children with ADHD continue to meet full criteria for ADHD as adults. A multivariate risk index comprising variables that can be assessed in adolescence predicts persistence with good accuracy.

Key Words: Adult ADHD, Attention-deficit/hyperactivity disorder (ADHD), course of illness, epidemiology, risk factors for disorder persistence

dult follow-up studies show that many children treated for attention-deficit/hyperactivity disorder (ADHD) continue to have ADHD as adults (1–3). This finding has been challenged, however, because the low treatment rate of ADHD at the time these studies started means that the children studied might have been especially severe cases with atypically high persistence (4). The fact that ADHD diagnostic criteria differed from current criteria raises further questions. Another limitation is that baseline cases lost to follow-up are known to be healthier than those who participate (5), presumably biasing estimates of persistence. These limitations have been addressed in recent community epidemiological surveys that assessed prevalence of adult ADHD (6,7). Adult ADHD was shown to be a relatively common disorder (3%–6% prevalence) in these studies. Consistent with clinical follow-ups, respondents with adult ADHD represent between 30% and 80% of those who retrospectively reported childhood ADHD. Given this high persistence, predictors of adult ADHD become of interest. Although such predictors have been examined in several clinical follow-up studies, these studies focused mainly on associated features of childhood ADHD (8,9). Number and severity of childhood symptoms were the strongest predictors of persistence. Only two prospective studies examined a broader set of predictors (10,11), but these studies were limited to follow-ups into adolescence. History of ADHD in relatives, presence of comorbid childhood disorders (especially

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conduct disorder), and childhood psychosocial adversity were the strongest predictors of persistence in these studies.

The same limitations of clinical follow-up studies in estimating prevalence of adult ADHD (i.e., sample selection bias) limit analysis of predictors. We are aware of only one general population study that addressed these limitations by examining predictors of adult persistence of ADHD (5). That study, based on a nationally representative U.S. survey, used retrospective adult reports to assess childhood predictors of adult ADHD persistence. As in clinical follow-up studies, childhood symptom profiles and severity were significant predictors of persistence, but no other predictors (including age, sex, comorbid childhood disorders, and child adversities) were significant.

Retrospective case–control studies such as this one could be biased by recall error. Studies of this type nonetheless provide a useful counterpoint, however, to clinical follow-up studies. This report presents additional data on the childhood predictors of adult ADHD using the same retrospective design from 10 general population surveys carried out as part of the World Health Organization (WHO) World Mental Health (WMH) Survey Initiative (12). Our aim is to determine whether the results in the earlier U.S. study hold up cross-nationally. The sample is heterogeneous, and the number of respondents retrospectively classified as having had childhood ADHD (n = 629) is also quite large, providing good statistical power to detect predictors.

Methods and Materials

Samples

The WMH is a WHO project designed to facilitate community epidemiological surveys of mental disorders (13). So far the WMH surveys have been administered in more than two dozen countries (http://www.hcp.med.harvard.edu/wmh) and are based on household probability samples that use the same procedures to train (a 7-day training program discussed in more detail elsewhere) (14) and monitor interviewer performance. Interviews are administered face-to-face in the homes of respondents using an interview translated using standard WHO procedures (15). Informed consent is obtained before beginning interviews. The Human Subjects Committee of responsible institutions in every country approves and monitors the WMH recruitment, consent, and field procedures. Centralized data cleaning, coding, and data analysis are used to maintain uniformity of postprocessing.

ADHD was an optional WMH diagnosis assessed in 10 countries (Table 1). Seven of these 10 are classified by the World Bank (16) as developed and three as developing. Eight of the 10 surveys were based on nationally representative samples, the other two on representative samples of urbanized areas. Sample sizes ranged from 2372 to 9282, with a combined 43,772 respondents. Response rates ranged from 45.9% to 94.3%, with a weighted (by sample size) average of 67.9%.

The WMH interview was administered in two parts. All respondents completed Part I, which assessed core disorders. Part I respondents who met criteria for any of these disorders plus a probability subsample of other Part I respondents then received Part II, which assessed additional disorders and correlates. Adult ADHD was assessed in Part II. Part II respondents who did not have a Part I disorder were weighted by the inverse of their probability of selection to make the Part II sample representative of the entire population. As shown elsewhere (17), the weighted Part II sample distributions match the Census population distributions on numerous sociodemographic variables. Because one requirement for a diagnosis of ADHD is

childhood onset, ADHD assessment was limited to respondents aged 18–44 to reduce retrospective recall bias. The combined number of Part II respondents in this age range was 11,422.

Diagnostic Assessment

Lifetime and current DSM-IV disorders were assessed using the WHO Composite International Diagnostic Interview (CIDI) Version 3.0 (12), a fully structured, lay-administered interview. Organic exclusion rules and hierarchy rules were used in making all diagnoses. No informants were interviewed. As detailed elsewhere (18), blinded clinical reappraisal interviews with the Structured Clinical Interview (SCID) for DSM-IV (19) found acceptable to good concordance between DSM-IV/CIDI diagnoses and DSM-IV/SCID diagnoses in the four WMH countries in which clinical reappraisal studies were administered.

The CIDI retrospective assessment of childhood ADHD was based on the Diagnostic Interview Schedule (DIS) (20). Respondents classified as having had childhood ADHD were asked whether they still had problems with inattention or impulsivity/ hyperactivity and, if so, were asked about impairments due to these symptoms. A probability subsample of 154 respondents in the U.S. sample with a history of childhood ADHD was administered blinded clinical follow-up interviews to assess DSM-IV adult ADHD using the validated form of the Adult ADHD Clinical Diagnostic Scale (ACDS) Version 1.2 (21,22) with probes. This clinical reappraisal survey is described in more detail elsewhere (5).

Logistic regression analysis was used in the clinical reappraisal sample to predict DSM-IV/ACDS diagnoses of adult ADHD from CIDI symptom questions. Diagnostic classification accuracy was good, with area under the receiver operating characteristic curve (AUC) of .86. On the basis of this result, the method of multiple imputation (MI) (23) was used to assign imputed clinical diagnoses of adult ADHD to respondents in all WMH surveys using the prediction equation in the U.S. clinical reappraisal sample. This approach implicitly assumes that the association between CIDI responses and clinical diagnoses is constant across countries. If this assumption is incorrect, the results will be biased. It would have been preferable to implement clinical reappraisal studies in other countries, but this was not possible.

The statistical details of the MI method are discussed elsewhere (5). The important points to emphasize here is that MI generates unbiased prevalence estimates under the model; that individual-level estimates have good accuracy when, as in this case, AUC is high; and that the method adjusts estimates of standard errors for the effects of classification error due to imperfect imputation. The imputation equation used here was somewhat less refined than in the earlier U.S. study because not all countries included all predictors used in the U.S. imputation equation.

Predictors of Adult Persistence

We examined six classes of predictors: age and sex, childhood ADHD symptom severity, childhood ADHD treatment, comorbid child/adolescent DSM-IV disorders, childhood adversities, and childhood traumatic events.

Childhood ADHD symptom profiles were divided into five categories: 1) inattentive type (six to nine inattentive symptoms, no impulsivity/hyperactivity symptoms; 2) impulsive/hyperactive type (six to nine hyperactive/impulsive symptoms, no inattentive symptoms); 3) inattentive and subthreshold (one to five symptoms) impulsive/hyperactive type; 4) impulsive/hyperactive and

Table 1. World Mental Health Survey Sample Characteristics

					Sample Size			
	Survey	Sample Characteristics ^a	Field Dates	Age Range	Part I	Part II	Part II and Age 18–44	Response Rate ^b
I. WHO Region:	The Americas	s (AMRO)						
Colombia	NSMH	Stratified multistage clustered area probability sample of household residents in all urban areas of the country (approximately 73% of the total national population)	2003	18–65	4426	2381	1731	87.7
Mexico	M-NCS	Stratified multistage clustered area probability sample of household residents in all urban areas of the country (approximately 75% of the total national population)	2001–2	18–65	5782	2362	1736	76.6
United States	NCS-R	Stratified multistage clustered area probability sample of household residents; NR	2002–3	18+	9282	5692	3197	70.9
II. WHO Region		Mediterranean (EMRO)						
Lebanon	LEBANON	Stratified multistage clustered area probability sample of household residents; NR	2002–3	18+	2857	1031	595	70.0
III. WHO Regior	n: Europe (EUF	RO)						
Belgium	ESEMeD	Stratified multistage clustered probability sample of individuals residing in households from the national register of Belgium residents; NR	2001–2	18+	2419	1043	486	50.6
France	ESEMeD	Stratified multistage clustered sample of working telephone numbers merged with a reverse directory (for listed numbers); initial recruitment was by telephone, with supplemental in-person recruitment in households with listed numbers; NR	2001–2	18+	2894	1436	727	45.9
Germany	ESEMeD	Stratified multistage clustered probability sample of individuals from community resident registries; NR	2002–3	18+	3555	1323	621	57.8
Italy	ESEMeD	Stratified multistage clustered probability sample of individuals from municipality resident registries; NR	2001–2	18+	4712	1779	853	71.3
Netherlands	ESEMeD	Stratified multistage clustered probability sample of individuals residing in households that are listed in municipal postal registries; NR	2002–3	18+	2372	1094	516	56.4
Spain	ESEMeD	Stratified multistage clustered area probability sample of household residents; NR	2001–2	18+	5473	2121	960	78.6

ESEMeD, the European Study of the Epidemiology of Mental Disorders; NSMH, the Colombian National Study of Mental Health; LEBANON, Lebanese Evaluation of the Burden of Ailments and Needs of the Nation; M-NCS, the Mexico National Comorbidity Survey; NCS-R, the U.S. National Comorbidity Survey Replication; NR, nationally representative; WHO, World Health Organization.

^aMost World Mental Health (WMH) surveys are based on stratified multistage clustered area probability household samples in which samples of areas equivalent to counties or municipalities in the United States were selected in the first stage followed by one or more subsequent stages of geographic sampling (e.g., towns within counties, blocks within towns, households within blocks) to arrive at a sample of households, in each of which a listing of household members was created and one or two people were selected from this listing to be interviewed. No substitution was allowed when the originally sampled household resident could not be interviewed. These households amples were selected from census area data in all countries other than France (where telephone directories were used to select households) and the Netherlands (where postal registries were used to select households). Several WMH surveys (Belgium, Germany, Italy) used municipal resident registries to select respondents without listing households. Eight of the 10 surveys are based on NR household samples, while two others are based on nationally representative household samples in urbanized areas (Colombia, Mexico).

^bThe response rate is calculated as the ratio of the number of households in which an interview was completed to the number of households originally sampled, excluding from the denominator households known not to be eligible either because of being vacant at the time of initial contact or because the residents were unable to speak the designated languages of the survey.

subthreshold (one to five symptoms) inattentive type; and 5) combined type (six to nine inattentive symptoms and six to nine impulsive/hyperactive symptoms).

Childhood ADHD–related severity was assessed with four yes–no questions that asked whether ADHD interfered significantly with functioning during childhood at home, school, in social life, and in personal relationships. High impairment was defined as endorsing all four questions. Childhood ADHD treatment was assessed in questions that asked about receiving medication and psychotherapy for ADHD in childhood. Treatment of ADHD of any type (i.e., either general medical treatment or specialty mental health treatment; either medication or psychotherapy) before age 16 was defined as having received treatment.

Comorbid DSM-IV child/adolescent disorders retrospectively

assessed in the CIDI were also considered as predictors of ADHD persistence. All CIDI disorders with onsets before age 16 were included as individual predictors and in various composite measures.

Twelve childhood adversities included as predictors included three types of child maltreatment (physical abuse, sexual abuse, neglect), three types of loss (death of parent, parental divorce, other major loss), three types of parental psychopathology (anxiety or mood disorder, substance disorder, antisocial personality disorder), family violence, family economic adversity, and respondent severe childhood physical illness. The child maltreatment measures were standard measures used in child welfare research (24). The measures of parental psychopathology were based on the Family History Research Diagnostic Criteria interview (25) and its expansion (26). The parental anxiety or mood disorders included MDD, panic disorder, and generalized anxiety disorder. The measure of family violence was based on the Revised Conflict Tactics Scale (27). The measures of family financial adversity and child physical illness were developed for the baseline National Comorbidity Survey (28).

Questions were also included about exposure to more than two dozen traumatic life events that occurred before age 16 assessed in the CIDI posttraumatic stress disorder trauma checklist. Included were traumas involving violence (e.g., physical assault, sexual assault), other personal traumas (e.g., natural disasters, automobile accidents), witnessing (e.g., observing acts of violence, seeing someone die in an accident), and traumas to a loved one (e.g., suicide or murder of a family member).

Data Analysis

As noted earlier, MI was used to assign predicted diagnoses of clinician-assessed adult DSM-IV ADHD to respondents who did not participate in the U.S. ADHD clinical reappraisal study. As detailed elsewhere (23), 10 individual-level imputations of adult ADHD were generated for each respondent based on the coefficients in the MI prediction equation in 10 samples the same size as the original clinical reappraisal sample drawn with replacement from that sample. Each equation assigned a predicted probability of adult ADHD to each respondent. An independent random draw from the binomial distribution for each of these 6290 predicted probabilities (10 for each of the 629 respondents with childhood ADHD) was used to assign a categorical (yes-no) adult ADHD diagnosis. Substantive analyses were replicated for each of the 10 imputed data sets. Parameter estimates reported here are averages of the coefficients in these 10 replications. Standard errors of parameter estimates are square roots of the sum of the average within-replicate coefficient variances and the variance of the coefficients across replicates. These standard errors take into consideration prediction error in the imputation equations.

Predictors of ADHD persistence were estimated using MI logistic regression analysis. Because the number of respondents with ADHD was small in individual surveys, regression coefficients were estimated across all 10 surveys combined using nine dummy control variables to distinguish countries. The first equations examined predictive effects of age and sex, which were controlled in later equations. The next equations examined separate predictive effects of childhood symptom profile and severity, which were controlled in later equations. Later equations examined one predictor at a time along with controls because coefficients were more stable in bivariate than multivariate models because of significant intercorrelations among predictors. Because predictors were correlated with both childhood ADHD and ADHD persistence, parallel results are reported for the associations of the predictors with each of these outcomes.

The MI logistic regression coefficients and their standard errors were exponentiated to create odds ratios (ORs) and 95% confidence intervals (CIs) for ease of interpretation. Regression equations were estimated using Taylor series linearization (29) implemented in SUDAAN (30) to adjust for design effects. Statistical significance was evaluated at the .05 level using two-sided tests. Simultaneous significance (e.g., a single test for significance of a series of predictors) was evaluated using Wald Chi-Square tests. Statistically significant predictors were combined into a risk index treated as a count variable to predict persistence. **Table 2.** Cross-National Variation in the Conditional Prevalence of CurrentAdult DSM-IV ADHD Among Respondents Who Met Criteria for ADHD inChildhood by Country ($c_9^2 = 20.3, p = .051$)

	%	(SE) ^a	(n) ^b
I. WHO Region: The Americas			
Colombia	75.8	(10.1)	(33)
Mexico	32.8	(7.3)	(88)
United States ^c	46.0	(4.9)	(346)
II. WHO Region: The Eastern Mediterranean			
Lebanon	52.4	(15.0)	(20)
III. WHO Region: Europe			
Belgium	71.9	(16.5)	(15)
France	58.8	(14.0)	(38)
Germany	67.9	(16.0)	(20)
Italy	84.1	(11.1)	(17)
Netherlands	82.3	(14.4)	(22)
Spain	33.6	(20.6)	(30)
IV. Weighted total	50.0	(4.8)	(629)

ADHD, attention-deficit/hyperactivity disorder; WHO, World Health Organization.

^aSE: Standard error of the prevalence estimate.

^bThe reported sample sizes are the numbers of respondents who are estimated to have met DSM-IV criteria for ADHD in childhood. The percentages are the proportions of these childhood cases that continued to meet DSM-IV criteria for ADHD at the time of interview.

^cThe proportion reported here differs somewhat from the estimate in a previous report (Kessler *et al.*, [44]) because it is based on a somewhat less refined imputation equation than the one used in the previous report. This is because some of the predictors used in the earlier imputation equation were not available in all the surveys.

Results

Persistence

As reported previously (6), estimated prevalence of adult ADHD in the pooled WMH countries was 3.4%, ranging from 7.3% in France to 1.2% in Spain. Adult persistence was estimated to be 50.0% in the total sample (Table 2), ranging from 84.1% in Italy to 32.8% in Mexico ($\chi^2_9 = 20.3$, p = .05).

Age and Sex Differences in Persistence

Persistence did not differ significantly by respondent age ($\chi^2_2 = 1.6$, p = .45) or sex ($\chi^2_1 = .0$, p = .88), even though men had a significantly higher prevalence of childhood ADHD than women.

Childhood ADHD Symptom Profiles, Severity, and Treatment

The majority of respondents with retrospectively assessed childhood ADHD reported having the inattentive type (35.3%), the impulsive/hyperactive type (23.0%), or the inattentive type with subthreshold impulsivity/hyperactivity (26.5%; Table 3). Smaller numbers reported either the impulsive/hyperactive type with subthreshold inattention (6.4%) or the combined type (8.8%). Persistence was highest for the combined type (84.5%), lowest for the impulsive/hyperactive type (29.0%), and intermediate for others (48.7%–58.3%; $\chi^2_4 = 27.7$, p < .001).

After controlling for age, sex, and country, childhood ADHD severity of role impairment was significantly associated with adult persistence (OR = 2.0). Childhood ADHD treatment, in comparison, was not associated with adult persistence (OR= .9). Childhood treatment was uncommon, however, with only 79 of 629 respondents receiving treatment before age 16.

Childhood Adversities

Childhood adversities were highly prevalent among respondents with childhood ADHD, with 71.5% of such respondents **Table 3.** Distributions and Associations (Odds Ratios) of Childhood ADHD Symptom Profiles and Severity withCurrent DSM-IV Adult ADHD Among Respondents Who Met Criteria for ADHD in Childhood Pooled Across the 10Surveys (n = 629)

			Current ADHD Among Childhood Cases					
	Distribution ^a		Preva	alence ^b				
	%	(SE)	%	(SE)	OR	(95% CI)		
I. Childhood Symptom Profiles ^c								
Inattentive (IN)	35.3	(2.6)	48.7	(6.1)	2.7 ^d	(1.3–5.6)		
Impulsive-hyperactive (IH)	23.0	(2.1)	29.0	(5.7)	1.0	_		
Inattentive + Sub IH	26.5	(2.7)	58.3	(10.5)	5.1 ^d	(1.8–14.5)		
Impulsive-hyperactive + Sub IN	6.4	(1.3)	50.7	(10.3)	1.7	(.6–4.6)		
IN + IH	8.8	(1.6)	84.5	(5.3)	12.4 ^d	(4.5–34.5)		
$\chi^{2}_{4}^{e}$						27.7 ^d		
II. Childhood symptom severity ^f								
High	18.7	(1.9)	62.8	(7.4)	2.0 ^d	(1.1–3.5)		
Low	81.3	(1.9)	47.0	(4.9)	1.0	_		
χ^2_1						5.7 ^d		
III. Childhood treatment of ADHD ^g								
Yes	10.4	(1.8)	47.2	(8.1)	.9	(.4–2.0)		
No	89.6	(1.8)	50.3	(5.1)	1.0			
χ^2_1						.0		

ADHD, attention-deficit/hyperactivity disorder; CI, confidence interval; OR, odds ratio; SE, standard error of the prevalence estimate.

^aDistribution: The conditional prevalence of the childhood ADHD symptom profile or severity category described in the row among respondents with current adult ADHD. For example, 35.3% of respondents with current adult ADHD had a purely inattentive type of ADHD as children.

^bPrevalence: The conditional prevalence of current adult ADHD among respondents with a history of childhood ADHD in the sub-sample defined by the childhood symptom profile or severity category in the row. For example, 48.7% of the childhood cases with an inattentive type of ADHD continued to have adult ADHD at the time of interview.

^cInattentive (IN): respondents who had 6–9 childhood symptoms of inattentiveness, but no symptoms of impulsivity/hyperactivity; impulsive/hyperactive (IH): respondents who had 6–9 childhood symptoms of impulsivity/hyperactivity, but no symptoms of inattentiveness; inattentive + sub (subthreshold) IH: respondents who had 6–9 childhood symptoms of inattentiveness and 1–5 symptoms of impulsivity/hyperactivity; impulsive-hyperactive + sub (subthreshold) IN: respondents who had 6–9 childhood symptoms of inattentiveness; IN + IH: respondents who had 6–9 childhood symptoms of inattentiveness and 6–9 sym

^dSignificant at the .05 level, two-sided test.

^eThe 4 degree of freedom c² evaluates the joint significance of the different childhood ADHD symptom profiles in predicting adult persistence.

^fHigh severity is defined as reportedly having childhood impairment in all four of the domains assessed in the survey (school, home, work, and relationships). The OR is based on a pooled within-country logistic regression equation that controlled for country, age, and sex.

^gThe OR is based on a pooled within-country logistic regression equation that controlled for childhood symptom profile and severity as well as for country, age, and sex.

experiencing at least one such adversity and 45.4% at least two. Ten of 12 adversities were significantly associated with childhood ADHD (Table 4). Only one of these 10, however, significantly predicted adult persistence: parental antisocial personality disorder (ASPD; OR = 2.2). Based on a suggestion in previous research that paternal psychopathology might be more important than maternal psychopathology in predicting adult persistence of ADHD (8), we looked at parental psychopathology by sex. Paternal anxiety or mood disorder had a significant OR predicting ADHD persistence (2.4), whereas maternal anxiety or mood disorder did not (1.2). The OR for parental ASPD predicting persistence, however, was very similar for fathers (2.1) and mothers (2.3). Excluding these coefficients, other childhood adversities had no significant predictive effects.

Traumatic Stress Exposure

The majority (76.1%) of respondents with childhood ADHD was exposed to at least one traumatic life event before age 16 (Table 1 in Supplement 1). A strong dose–response relationship,

with ORs ranging from 10.4 for exposure to three or more traumas to 2.9 for exposure to a single trauma, existed between number of traumas and childhood ADHD (χ^2_3 = 251.8, *p* < .001). No significant association existed, however, between number of childhood traumas and persistence of ADHD (χ^2_3 = 1.3, *p* = .74).

Comorbid DSM-IV Disorders

The vast majority of the other DSM-IV CIDI disorders with onsets before age 16 significantly predicted childhood ADHD (Table 2 in Supplement 1) Only one—MDD—however, significantly predicted adult persistence of ADHD, with an OR of 2.2 with persistence. The ORs of bipolar disorder, oppositionaldefiant disorder, and conduct disorder with persistence were all close to random levels (1.0–1.3) despite these disorders having strong associations with childhood ADHD (ORs of 7.6–11.9). A clear sign pattern existed, however, in the ORs of comorbid child/adolescent disorders predicting persistence, with all 16 of the ORs greater than 1.0. This raises the possibility that a summary

Table 4. Associations (Odds Ratios) of Childhood Adversities with Childhood ADHD in the Total Sample ($N = 11,422$) and with Current DSM-IV Adult
ADHD Among Respondents Who Met Criteria for ADHD in Childhood ($n=629$) Pooled Across the 10 Surveys

	Childhood ADHD in the Total Sample					Current ADHD Among Childhood Cases						
	Distribution ^a		Prevalence ^b				Distribution ^d		Prevalence ^e			
	%	(SE)	%	(SE)	OR ^c	(95% CI)	%	(SE)	%	(SE)	OR ^f	(95% CI)
I. Neglect and Abuse												
Neglect	17.7	(2.1)	12.9	(1.4)	4.5 ^g	(3.2–6.3)	17.6	(3.1)	45.8	(7.8)	1.0	(.5–2.1)
Physical abuse	27.3	(2.1)	9.8	(.9)	4.6 ^g	(3.6–5.9)	30.6	(3.4)	56.0	(5.8)	1.6	(.9–2.7)
Sexual abuse	9.5	(1.2)	11.7	(1.5)	3.0 ^g	(2.2-4.1)	8.3	(1.9)	43.2	(7.6)	.7	(.3–1.5)
II. Loss												
Parental death	9.6	(1.4)	3.6	(.5)	1.1	(.8–1.5)	11.2	(2.2)	58.4	(10.6)	1.2	(.5–3.0)
Parental divorce	20.9	(2.4)	6.4	(.8)	1.1	(.8–1.5)	20.9	(3.3)	46.0	(6.8)	1.1	(.6-2.0)
Other major loss	10.8	(1.4)	7.8	(1.0)	2.2 ^g	(1.6-3.0)	9.6	(1.8)	44.1	(6.7)	.8	(.5-1.5)
III. Paternal Psychopathology												
Anxiety or mood disorder ^h	8.6	(1.3)	14.1	(2.1)	4.4 ^g	(3.1–6.5)	11.7	(2.3)	68.1	(8.3)	2.4 ^{<i>g</i>}	(1.1-5.5)
Substance use disorder	14.6	(2.1)	11.3	(1.5)	3.3 ^g	(2.3–4.8)	13.7	(2.5)	46.7	(7.5)	.9	(.5–1.7)
ASPD	12.4	(1.3)	12.4	(1.3)	2.9 ^g	(2.2–3.9)	14.9	(2.4)	60.0	(8.4)	2.1	(1.0-4.3)
IV. Maternal Psychopathology												(
Anxiety or mood disorder ^h	25.9	(2.5)	13.0	(1.2)	4.5 ^g	(3.4–5.9)	27.6	(3.4)	53.3	(6.7)	1.2	(.7–2.1)
Substance use disorder	4.4	(.9)	16.3	(3.5)	3.1 ^g	(1.8–5.1)	4.1	(1.1)	46.4	(10.6)	.8	(.3–2.3)
ASPD	3.4	(.7)	14.9	(2.9)	2.5 ^g	(1.5-4.1)	4.1	(1.3)	60.4	(14.3)	2.3	(.8–6.9)
V. Parental (Either Father or Mother)												
Psychopathology												
Mental disorder	29.0	(2.8)	12.2	(1.2)	4.4 ⁹	(3.3–5.8)	32.0	(3.9)	55.2	(6.4)	1.4	(.8–2.5)
Substance use disorder	17.8	(2.2)	11.9	(1.4)	3.4 ^g	(2.4–4.7)	16.2	(2.6)	45.3	(6.5)	.8	(.5–1.4)
ASPD	14.7	(1.4)	12.6	(1.3)	2.9 ^g	(2.2–4.0)	17.8	(2.7)	60.5	(7.7)	2.2 ^g	(1.2-4.2)
VI. Other Adversities												
Family violence	27.8	(2.4)	9.4	(.8)	3.3 ^g	(2.6–4.2)	27.9	(3.2)	50.1	(5.9)	1.2	(.7–2.0)
Economic adversity	13.8	(1.5)	8.4	(1.0)	1.7 ^g	(1.3–2.3)	15.5	(2.5)	56.0	(6.4)	1.0	(.6–2.0)
Severe childhood illness	7.9	(1.5)	7.5	(1.5)	1.9 ^g	(1.2–3.0)	6.8	(1.9)	42.5	(9.9)	1.0	(.4–2.6)
VII. Number of Adversities												(
None	28.4	(2.7)	1.8	(.2)	1.0	_	25.4	(3.7)	44.7	(7.4)	1.0	
Exactly 1	26.1	(2.4)	4.2	(.5)	1.1	(.8–1.4)	28.3	(3.3)	54.0	(7.0)	1.1	(.6–1.8)
Exactly 2	14.8	(2.0)	5.5	(.8)	1.5 ^g	(1.1–2.0)	14.5	(2.5)	48.9	(7.2)	1.1	(.6–2.1)
Exactly 3	9.4	(1.3)	7.2	(1.0)	1.9 ^g	(1.3–2.7)	9.8	(1.7)	52.1	(8.2)	1.0	(.5–2.0)
4 or more	21.2	(2.0)	15.9	(1.5)	4.9 ^g	(3.6–6.7)	22.0	(2.8)	51.9	(5.8)	1.4	(.8–2.3)
$\chi^{2}_{4}^{i}$,,		151.8 ^g	-	···· · · · · · · · · · · · · · · · · ·		(/		3.3		(/
(n)				11,422)						(629)		

ADHD, attention-deficit/hyperactivity disorder; ASPD, Antisocial personality disorder; CI, confidence interval; OR, odds ratio; SE, standard error of the prevalence estimate.

^aDistribution: The conditional prevalence of the childhood adversity indicated in the row among respondents with a history of childhood ADHD.

^bPrevalence: The conditional prevalence of childhood ADHD among respondents with the childhood adversity in the row.

^cEach OR in Sections I–VI is based on a separate pooled within-country logistic regression equation that controlled for country, age, and sex in the total sample to predict childhood ADHD. All the ORs in Section VII, in comparison, are based on a single equation.

^dDistribution: The conditional prevalence of the childhood adversity indicated in the row among respondents with current adult ADHD.

^ePrevalence: The conditional prevalence of current adult ADHD among respondents with a history of childhood ADHD among respondents with the childhood adversity in the row.

⁷Each OR in Sections I–VI is based on a separate pooled within-country logistic regression equation that controlled for childhood symptom profile and severity as well as for country, age, and sex in the subsample of respondents with a history of childhood ADHD to predict current adult ADHD. All the ORs in Section VII, in comparison, are based on a single equation.

^gSignificant at the .05 level, two-sided test.

^hMajor depression, panic disorder, or generalized anxiety disorder.

The 4 degree of freedom χ^2 values evaluate the joint significance of the four number-of-adversity dummies predicting the outcomes.

count of comorbid disorders might predict persistence even though most of the individual conditions do not. Further analysis found that a measure of high comorbidity, defined as having any three or more child/adolescent disorders in addition to ADHD, has a significant OR predicting persistence (1.7 [1.1–2.6]).

A Composite Risk Index

We created a weighted composite risk index to determine how well a simple scoring scheme could classify young people with ADHD into those with higher or lower risk of adult persistence. The index summed information about the significant predictors described earlier and used simple weights based roughly on the sizes of the ORs for those predictors in the tables presented so far. Respondents having a childhood history of both the inattentive and impulsive/hyperactive types were assigned 11 points in this summary measure, whereas 4 points were assigned for having the inattentive type with subthreshold impulsivity/ hyperactivity and 1 point for either the pure inattentive type or the impulsive/hyperactive type with subthreshold inattentive symptoms. (The omitted category of having had the pure impul-

Table 5. Distributions and Associations (Odds Ratios) of Scores on the Composite Risk Index with Current DSM-IVAdult ADHD Among Respondents Who Met Criteria for ADHD in Childhood Pooled Across the 10 Surveys (n = 629)

				Current ADHD Among Childhood Cases						
	Distributon ^a		Prevalence ^b		Conditional Distribution ^c					
	%	(SE) ⁴	%	(SE) ⁴	%	(SE) ⁴	OR^d	(95% CI)		
I. Composite Risk Index										
0	16.2	(2.1)	21.2	(5.7)	6.9	(1.7)	1.0	_		
1–2	41.7	(2.8)	46.0	(5.3)	38.5	(4.2)	3.5 ^e	(1.6–7.6)		
3–6	32.1	(2.7)	58.6	(9.5)	37.6	(4.2)	8.3 ^e	(2.9–23.8)		
7+	9.9	(1.7)	85.7	(5.1)	17.1	(3.2)	23.8 ^e	(7.6–74.4)		
$\begin{array}{c} \chi^2{}_3{}^f\\ \text{AUC} = .76 \end{array}$								32.8 ^e		

ADHD, attention-deficit/hyperactivity disorder; CI, confidence interval; OR, odds ratio; SE, standard error of the prevalence estimate.

^aDistribution: The conditional prevalence of scores on the risk index among respondents with a history of childhood ADHD.

^bPrevalence: The conditional prevalence of current adult ADHD among respondents with a history of childhood ADHD in sub-samples defined by the level of the risk index in the row.

^cConditional distribution: The proportion of all respondents with adult ADHD who have scores at each level of the risk index.

^dThe ORs are based on a single logistic regression equation that controlled for country in the subsample of respondents with a history of childhood ADHD to predict current adult ADHD.

^eSignificant at the .05 level, two-sided test.

^{*f*}The 3 degree of freedom χ^2 evaluates the joint significance of the three coefficients for level on the composite risk index in predicting adult persistence of ADHD.

sive/hyperactive type was given no points.) Pervasive childhood ADHD-related role impairment, child/adolescent MDD/dysthymia, three or more child/adolescent anxiety or substance use disorders, paternal anxiety or mood disorder, and parental ASPD were each assigned 1 point. A strong dose–response relationship existed between scores on this index and odds of adult persistence, with the highest category of risk having an OR of 23.8 compared with the lowest category (Table 5). The index had an AUC of .76 in predicting ADHD persistence among respondents with childhood ADHD.

Discussion

Several limitations to this study are noteworthy. First, the good concordance (AUC = .86) between CIDI diagnoses and clinical diagnoses in the clinical reappraisal sample might be called into question because the clinical interviews, like the CIDI, were administered only to respondents and not informants. Arguing against this is the observation that methodological research has documented good concordance between community diagnoses of adult ADHD based on respondent and informant interviews (31), arguing for the validity of the WMH diagnosis of adult ADHD, at least in the United States. Nonetheless, caution is necessary in interpreting results because clinician interviews were not administered to all respondents. Future cross-national comparative studies should carry out clinical reappraisal interviews in each country studied to confirm validity of lay diagnoses and to obtain clinician-based diagnoses in as many cases as possible.

Retrospective diagnoses of childhood ADHD are probably less accurate than diagnoses of adult ADHD because of retrospective recall bias. Recall bias could also affect measures of childhood predictors. As noted earlier, the prospective cohort design used in clinical studies avoids this bias but introduces attrition bias. In light of the different biases in retrospective and prospective designs, predictors significant in both types of studies are likely to be of most value in expanding our understanding of determinants of ADHD persistence.

Another limitation related to diagnostic assessment is that DSM-IV ADHD criteria were developed for children. Clinical studies show that ADHD symptoms are more subtle and heterogeneous in adults (32), suggesting that accurate assessment might require an increase in the variety of symptoms assessed (33), a modification in the severity threshold (34), a change in the DSM-IV six-of-nine symptom requirement (35), and a change in the age-of-onset requirement (36). To the extent that such changes would lead to a more valid assessment, WMH estimates of persistence and correlates might be biased.

A final noteworthy limitation is that neither parental ADHD nor any of the biological variables shown to predict ADHD persistence (37–39) were included in our analysis. Prospective studies that consider the joint predictive effects of all these variables are needed to develop a more refined risk index than the preliminary index developed here.

Within the context of these limitations, we found that roughly 50% of childhood cases of ADHD continue to meet full criteria for ADHD as adults and that adult persistence is significantly associated with retrospectively reported childhood ADHD severity, childhood symptom profile (highest persistence associated with the attentional plus impulsive/hyperactive type, lowest with the impulsive/hyperactive type), comorbid MDD, high comorbidity, paternal (but not maternal) anxiety or mood disorder, and parental antisocial personality disorder. A multivariate risk index predicted adult persistence with AUC = .76.

Our estimate that roughly 50% of childhood cases continue to meet full DSM-IV criteria for ADHD in adulthood is consistent with reports from clinical samples (2,10,40) and with the only published longitudinal study of ADHD persistence in a community sample (40). It is unclear why persistence appears to vary across the 10 WMH countries, but this variation is unrelated to either level of economic development or childhood ADHD prevalence.

Our finding that persistence is lowest among childhood cases of the impulsive/hyperactive type is consistent with the observation in clinical follow-up studies that inattention symptoms persist into adulthood more than impulsivity/hyperactivity (41). Our finding that persistence is highest among childhood cases with the combined type is also consistent with some (10), but not all (11), clinical reports.

Our finding that persistence is only weakly related to child/ adolescent externalizing disorders is inconsistent with two clinical follow-up studies that documented predictive effects of conduct disorder on ADHD persistence (10,11). This discrepancy might be because the earlier studies focused on clinical samples and followed respondents only into late adolescence. Although we could investigate this possibility in the WMH data by examining predictors of persistence separately for respondents who received childhood treatment, the number of such respondents is too small to carry out these analyses with adequate statistical power.

Our finding that childhood adversities are generally not related to ADHD persistence is consistent with the one clinical study that examined this association (10). That same study found, consistent with our results, that paternal but not maternal anxiety or mood disorders predicted ADHD persistence. Why this specification occurs is unclear, but our replication suggests that it is real. We are unaware of previous evidence regarding the predictive effect of parental ASPD, although this might be a proxy for the effect of parental ADHD, which was not assessed in the WMH surveys.

The high prevalence of child/adolescent traumatic events exposure in ADHD (76.1%) is striking and contrasts with Wozniak et al. (42), who found a relatively low proportion of ADHD probands reporting trauma exposure over a 4-year follow-up period. This difference might be because Wozniak et al. excluded respondents whose nuclear family was not available for study and assessed only eight traumatic life events compared with the more than two dozen in the WMH surveys. Because the vast majority of traumas reported by WMH respondents occurred after the age of onset of ADHD, they are best conceptualized as consequences of ADHD (or its determinants). Although extent of trauma exposure might consequently be seen as an indirect indicator of ADHD severity, we found no association between extent of trauma exposure and ADHD persistence. This is indirectly consistent with our finding that childhood adversities are unrelated to ADHD persistence.

We also found that child/adolescent nonbipolar depression and high comorbidity (i.e., 3+ child/adolescent disorders) significantly predict ADHD persistence. Although these patterns have not been found in previous clinical follow-up studies, this could reflect either sample selection bias in clinical studies or underdetection of other disorders in clinical evaluations of ADHD. Another possibility is that the comorbid conditions studied as predictors of ADHD persistence in clinical follow-up studies, which have been the early-onset disorders that occurred near the time ADHD treatment began, are less important in predicting persistence than subsequent child/adolescent disorders that occur secondary to ADHD and that we considered in the current report. It is noteworthy in this regard that secondary substance use disorders are quite often known to occur secondary to ADHD and to be more persistent than in the absence of ADHD (43). Although not statistically significant, the ORs of adolescent alcohol abuse and dependence were meaningfully elevated (1.9-2.7) in our data in predicting adult ADHD persistence. In light of this evidence, it might be useful for future prospective studies to evaluate the role of adolescent disorders secondary to ADHD in predicting adult ADHD persistence. It could be that the predictive effects of these disorders merely indicate aspects of child/adolescent ADHD symptom severity that were not assessed as accurately in the retrospective WMH reports as they could be in contemporaneous evaluations of child/adolescent cases in prospective studies. Another possibility is that high comorbidity somehow interferes with the processes that bring about recovery from ADHD in adolescence. Prospective studies that use information about adolescent severity and comorbidity to predict adult persistence will likely be necessary to investigate this possibility and, if positive, to determine whether successful treatment of secondary comorbid adolescent disorders can help reduce risk of adult persistence of ADHD.

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Supplementary material cited in this article is available online.

- Barkley RA, Fischer M, Edelbrock CS, Smallish L (1990): The adolescent outcome of hyperactive children diagnosed by research criteria: I. An 8-year prospective follow-up study. J Am Acad Child Adolesc Psychiatry 29:546–557.
- Barkley RA, Fischer M, Smallish L, Fletcher K (2002): The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. J Abnorm Psychol 111:279–289.
- Faraone SV, Biederman J, Mick E (2006): The age-dependent decline of attention deficit hyperactivity disorder: A meta-analysis of follow-up studies. *Psychol Med* 36:159–165.
- Mannuzza S, Klein RG, Moulton JL 3rd (2003): Persistence of attentiondeficit/hyperactivity disorder into adulthood: What have we learned from the prospective follow-up studies? J Atten Disord 7:93–100.
- Weiss G, Hechtman L, Milroy T, Perlman T (1985): Psychiatric status of hyperactives as adults: A controlled prospective 15-year follow-up of 63 hyperactive children. J Am Acad Child Psychiatry 24:211–220.
- Fayyad J, De Graaf R, Kessler R, Alonso J, Angermeyer M, Demyttenaere K, et al. (2007): Cross-national prevalence and correlates of adult attention-deficit hyperactivity disorder. Br J Psychiatry 190:402–409.
- Kessler RC, Adler L, Barkley R, Biederman J, Conners CK, Demler O, et al. (2006): The prevalence and correlates of adult ADHD in the United States: Results from the National Comorbidity Survey Replication. Am J Psychiatry 163:716–723.
- 8. Gittelman R, Mannuzza S, Shenker R, Bonagura N (1985): Hyperactive boys almost grown up. I. Psychiatric status. *Arch Gen Psychiatry* 42:937–947.
- Mannuzza S, Klein RG, Bessler A, Malloy P, LaPadula M (1993): Adult outcome of hyperactive boys. Educational achievement, occupational rank, and psychiatric status. Arch Gen Psychiatry 50:565–576.
- Biederman J, Faraone S, Milberger S, Curtis S, Chen L, Marrs A, et al. (1996): Predictors of persistence and remission of ADHD into adolescence: results from a four-year prospective follow-up study. J Am Acad Child Adolesc Psychiatry 35:343–351.
- Hart EL, Lahey BB, Loeber R, Applegate B, Frick PJ (1995): Developmental change in attention-deficit hyperactivity disorder in boys: A four-year longitudinal study. J Abnorm Child Psychol 23:729–749.
- Kessler RC, Üstün TB (2004): The World Mental Health (WMH) Survey Initiative Version of the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI). Int J Methods Psychiatr Res 13:93–121.
- Kessler RC, Üstün TB (2008): Overview and future directions for the WMH Survey Initiative In: Kessler RC, Üstün TB editors. *The WHO World Mental Health Surveys: Global Perspectives on the Epidemiology of Mental Disorders*. New York: Cambridge University Press, 557–567.
- Pennell B-E, Mneimneh Z, Bowers A, Chardoul S, Wells JE, Viana MC, et al. (2008): Implementation of the World Mental Health Surveys. In: Kessler RC, Üstün TB editors. The WHO World Mental Health Surveys: Global Perspectives on the Epidemiology of Mental Disorders. New York: Cambridge University Press, 33–57.
- 15. Harkness J, Pennell B-E, Villar A, Gebler N, Aguilar-Gaxiola S, Bilgen I (2008): Translation Procedures and Translation Assessment in the World Mental Health Survey Initiative. In: Kessler RC, Üstün TB editors. *The* WHO World Mental Health Surveys: Global Perspectives on the Epidemiology of Mental Disorders. New York: Cambridge University Press.
- World Bank (2003): World Development Report 2004: Making Services Work for Poor People. Washington, DC: The International Bank for Reconstruction and Development/The World Bank.
- Heeringa SG, Wells JE, Hubbard F, Mneimneh Z, Chiu WT, Sampson N, et al. (2008): Sample Designs and Sampling Procedures. In: Kessler RC, Üstün TB editors. The WHO World Mental Health Surveys: Global Perspectives on the Epidemiology of Mental Disorders. New York: Cambridge University Press, 14–32.
- Haro JM, Arbabzadeh-Bouchez S, Brugha TS, de Girolamo G, Guyer ME, Jin R, et al. (2006): Concordance of the Composite International Diagnostic Interview Version 3.0 (CIDI 3.0) with standardized clinical assessments in the WHO World Mental Health surveys. Int J Methods Psychiatr Res 15:167–180.

- 20. Robins LH, Helzer JE (1985): *Diagnostic Interview Schedule (DIS Version III-A)*. St. Louis, MO: Department of Psychiatry, Washington University.
- Adler L, Cohen J (2004): Diagnosis and evaluation of adults with attention-deficit/hyperactivity disorder. *Psychiatr Clin North Am* 27:187–201.
- Adler L, Spencer T (2004): The Adult ADHD Clinical Diagnostic Scale (ACDS), version 1.2. New York: New York University School of Medicine.
- 23. Rubin DB (1987): Multiple Imputation for Nonresponse in Surveys. New York: Wiley.
- Dubowitz H, Pitts SC, Black MM (2004): Measurement of three major subtypes of child neglect. *Child Maltreat* 9:344–356.
- Andreasen NC, Endicott J, Spitzer RL, Winokur G (1977): The family history method using diagnostic criteria. Reliability and validity. Arch Gen Psychiatry 34:1229–1235.
- Kendler KS, Davis CG, Kessler RC (1997): The familial aggregation of common psychiatric and substance use disorders in the National Comorbidity Survey: A family history study. *Br J Psychiatry* 170:541–548.
- Straus MA, Hamby SL, Boney-McCoy S, Sugarman DB (1996): The revised Conflict Tactics Scales (CTS2): Development and preliminary psychometric data. J Family Issues 17:283–316.
- Kessler RC, Davis CG, Kendler KS (1997): Childhood adversity and adult psychiatric disorder in the US National Comorbidity Survey. *Psychol Med* 27:1101–1119.
- 29. Wolter KM (1985): Introduction to Variance Estimation. New York: Springer-Verlag.
- Research Triangle Institute (2002): SUDAAN: Professional Software for Survey Data Analysis [computer program]. 8.0.1. ed. Research Triangle Park, NC: Research Triangle Institute.
- Murphy P, Schachar R (2000): Use of self-ratings in the assessment of symptoms of attention deficit hyperactivity disorder in adults. *Am J Psychiatry* 157:1156–1159.
- De Quiros GB, Kinsbourne M (2001): Adult ADHD. Analysis of self-ratings on a behavior questionnaire. Ann NY Acad Sci 931:140–147.
- Barkley RA (1995): ADHD behavior checklist for adults. The ADHD Report 3:16.
- Ratey JJ, Greenberg MS, Bemporad JR, Lindem KJ (1992): Unrecognized attention-deficit hyperactivity disorder in adults presenting for outpatient psychotherapy. J Child Adoles Psychopharmacol 2:267–275.
- Kooij JJ, Buitelaar JK, van den Oord EJ, Furer JW, Rijnders CA, Hodiamont PP (2005): Internal and external validity of attention-deficit hyperactivity disorder in a population-based sample of adults. *Psychol Med* 35:817–827.
- Barkley RA, Biederman J (1997): Toward a broader definition of the age-of-onset criterion for attention-deficit hyperactivity disorder. J Am Acad Child Adolesc Psychiatry 36:1204–1210.
- Li J, Kang C, Zhang H, Wang Y, Zhou R, Wang B, et al. (2007): Monoamine oxidase A gene polymorphism predicts adolescent outcome of attention-deficit/hyperactivity disorder. Am J Med Genet B Neuropsychiatr Genet 144:430–433.
- Mackie S, Shaw P, Lenroot R, Pierson R, Greenstein DK, Nugent TF 3rd, et al. (2007): Cerebellar development and clinical outcome in attention deficit hyperactivity disorder. Am J Psychiatry 164:647–655.
- Shaw P, Gornick M, Lerch J, Addington A, Seal J, Greenstein D, et al. (2007): Polymorphisms of the dopamine D4 receptor, clinical outcome, and cortical structure in attention-deficit/hyperactivity disorder. Arch Gen Psychiatry 64:921–931.
- Rasmussen P, Gillberg C (2000): Natural outcome of ADHD with developmental coordination disorder at age 22 years: A controlled, longitudinal, community-based study. J Am Acad Child Adolesc Psychiatry 39:1424–1431.
- Polanczyk G, Rohde LA (2007): Epidemiology of attention-deficit/hyperactivity disorder across the lifespan. Curr Opin Psychiatry 20:386–392.
- Wozniak J, Crawford MH, Biederman J, Faraone SV, Spencer TJ, Taylor A, et al. (1999): Antecedents and complications of trauma in boys with ADHD: Findings from a longitudinal study. J Am Acad Child Adolesc Psychiatry 38:48–55.
- 43. Wilens TE, Upadhyaya HP (2007): Impact of substance use disorder on ADHD and its treatment. *J Clin Psychiatry* 68:e20.
- Kessler RC, Adler LA, Barkley R, Biederman J, Conners CK, Faraone SV, et al. (2005): Patterns and predictors of attention-deficit/hyperactivity disorder persistence into adulthood: results from the national comorbidity, survey replication. *Biol Psychiatry* 57:1442–1451.