Published in final edited form as:
Biol Psychiatry. 2009 January 1; 65(1): 46–54. doi:10.1016/j.biopsych.2008.10.005.

Childhood predictors of adult ADHD: Results from the WHO World Mental Health (WMH) Survey Initiative

Carmen Lara, MD, PhD[1], **John Fayyad, MD**[2], **Ron de Graaf, PhD, MSc**[3], **Ronald C. Kessler, PhD**[4], **Sergio Aguilar-Gaxiola, MD, PhD**[5], **Matthias Angermeyer, MD**[6], **Koen Demytteneare, MD, PhD**[7], **Giovanni de Girolamo, MD**[8], **Josep Maria Haro, MD, MPH, PhD**[9], **Robert Jin, MA**[4], **Elie G. Karam, MD**[2], **Jean-Pierre Lépine, MD**[10], **Maria Elena Medina Mora, PhD**[11], **Johan Ormel, PhD**[12], **José Posada-Villa, MD**[13], and **Nancy Sampson, BA**[4]

[1]*Autonomous University of Puebla, Puebla, Mexico* [2]*Institute for Development, Research, Advocacy, and Applied Care (IDRAAC), St. George Hospital University Medical Center, Beirut, Lebanon* [3]*Netherlands Institute of Mental Health and Addiction, Utrecht, Netherlands* [4]*Health Care Policy, Harvard Medical School, Boston, Massachusetts, United States* [5]*Center for reducing Health Disparities, University of California, Sacramento, California, United States* [6]*Department of Psychiatry, Leipzig University, Germany* [7]*University Hospital Gasthuisburg, Leuven, Belgium* [8]*Health Care Research Agency, Emile-Romagna Region, Bologna, Italy* [9]*Sant Joan de DEU-SSM, Barcelona, Spain* [10]*Hospital Fernand Widal, Paris, France* [11]*Division of Epidemiological and Social Research, Mexican Institute of Psychiatry, Mexico City, Mexico* [12]*Department of Psychiatry & Department of Epidemiology and Bioinformatics, University Medical Center Groningen; Graduate School of Behavioural and Cognitive Neurosciences & Graduate School for Experimental Psychopathology, University of Groningen, the Netherlands* [13]*Ministry of Social Protection, Colegio Mayor de Cundinamarca University, Bogota, Colombia*

Abstract

Background—Although it is known that childhood ADHD often persists into adulthood, the childhood predictors of this persistence have not been widely studied.

Methods—Childhood history of DSM-IV ADHD and adult ADHD were assessed in ten countries in the WHO World Mental Health (WMH) 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. The risk factors included age, gender, childhood ADHD symptom profiles and 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-

Corresponding author: Dr. Ronald C. Kessler, Harvard Medical School, Department of Health Care Policy, 180 Longwood Ave., Boston, MA 02115. Phone: 617-432-3587, Fax: 617-432-3588, e-mail: Kessler@hcp.med.harvard.edu.

Financial Disclosure Professor Kessler has been a consultant for GlaxoSmithKline Inc., Pfizer Inc., Wyeth-Ayerst, Sanofi-Aventis, Kaiser Permanente, and Shire Pharmaceuticals; has served on advisory boards for Eli Lilly & Company and Wyeth-Ayerst; and has had research support for his epidemiological studies from Eli Lilly and Company, Pfizer Inc., Ortho-McNeil Pharmaceuticals Inc., and Bristol-Myers Squibb.

The remaining authors report no biomedical financial interests or potential conflicts of interest

hyperactive type, OR = 12.4, compared to the lowest associated with the impulsive-hyperactive type), symptom severity (OR = 2.0), comorbid major depressive disorder (OR = 2.2), high comorbidity (three or more 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 ROC Curve = .76).

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

Keywords

Attention-deficit/hyperactvity disorder (ADHD); adult ADHD; epidemiology; course of illness; risk factors for disorder persistence

---

Adult 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, though, because the low treatment rate of ADHD at the time these studies started means 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 ADHS 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-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 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 US 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, gender, 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, though, to clinical follow-up studies. The current report presents additional data on the childhood predictors of adult ADHD using the same retrospective design from ten general population surveys carried out as part of the WHO World Mental Health (WMH) Survey Initiative (12). Our aim is to see if the results in the earlier US study hold up cross-nationally. The sample is very 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

Samples

WMH is a WHO project designed to facilitate community epidemiological surveys of mental disorders (13). WMH surveys, so far administered in over two dozen countries (www.hcp.med.harvard.edu/wmh), are based on household probability samples that use the same procedures to train (a seven-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 post-processing.

ADHD was an optional WMH diagnosis assessed in ten countries. (Table 1) Seven of these ten are classified by the World Bank (16) as developed and three as developing. Eight of the ten surveys were based on nationally representative samples, the other two on representative samples of urbanized areas. Sample sizes range from 2,372 to 9,282, with a combined 43,772 respondents. Response rates range 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 sub-sample 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 socio-demographic variables. As one requirement for a diagnosis of ADHD is childhood onset, ADHD assessment was limited to respondents ages 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 for DSM-IV (SCID) (19) found acceptable-good concordance between DSM-IV/CIDI diagnoses and DSM-IV/SCID diagnoses in the four WMH countries where 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 sub-sample of 154 respondents in the US 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. Based on 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 US 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 US study because not all countries included all predictors used in the US imputation equation.

Predictors of adult persistence

We examined six classes of predictors: age and gender; childhood ADHD symptoms-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: (i) inattentive type (6-9 inattentive symptoms, no impulsivity-hyperactivity symptoms; (ii) impulsive-hyperactive type (6-9 hyperactive-impulsive symptoms, no inattentive symptoms); (iii) inattentive and sub-threshold (1-5 symptoms) impulsive-hyperactive type; (iv) impulsive-hyperactive and sub-threshold (1-5 symptoms) inattentive type; and (v) Combined type (6-9 inattentive symptoms and 6-9 impulsive-hyperactive symptoms).

Childhood ADHD-related severity was assessed with four yes-no questions that asked if ADHD interfered a lot 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) prior to 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 prior to 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-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-mood disorders included major depression, 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 NCS (28).

Questions were also included about exposure to over two dozen traumatic life events that occurred prior to the age of 16 assessed in the CIDI PTSD 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 above, MI was used to assign predicted diagnoses of clinician-assessed adult DSM-IV ADHD to respondents who did not participate in the US ADHD clinical reappraisal study. As detailed elsewhere (23), ten individual-level imputations of adult ADHD were generated for each respondent based on the coefficients in the MI prediction equation in ten 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 ten imputed datasets. Parameter estimates reported here are averages of the coefficients in these ten 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. As the number of respondents with ADHD was small in individual surveys, regression coefficients were estimated across all ten surveys combined using nine dummy control variables to distinguish countries. The first equations examined predictive effects of age and gender, 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 due to significant inter-correlations among predictors. As predictors were both correlated with childhood ADHD and with 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 (95% 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 χ^2 tests. Statistically significant predictors were combined into a risk index treated as a count variable to predict persistence.

RESULTS

Persistence

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

Age and gender differences in persistence

Persistence does not differ significantly by respondent age ($\chi^2_2 = 1.6$, p = .45) or gender ($\chi^2_1 = 0.0$, 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 sub-threshold impulsivity-hyperactivity (26.5%). (Table 3) Smaller numbers reported either the impulsive-hyperactive type with sub-threshold inattention (6.4%) or the Combined type (8.8%). Persistence is 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, gender, and country, childhood ADHD severity of role impairment is significantly associated with adult persistence (OR = 2.0). Childhood ADHD treatment, in comparison, is not associated with adult persistence (OR= 0.9). Childhood treatment was uncommon, though, with only 79 of 629 respondents receiving treatment prior to age 16.

Childhood adversities

Childhood adversities were highly prevalent among respondents with childhood ADHD, with 71.6% of such respondents experiencing at least one such adversity and 45.5% at least two. Ten of 12 adversities are significantly associated with childhood ADHD. (Table 4) Only one of these ten, though, significantly predicts 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 gender. Paternal anxiety-mood disorder has a significant OR predicting ADHD persistence (2.4), while maternal anxiety-mood disorder does not (1.2). The OR for parental ASPD predicting persistence, though, is very similar for fathers (2.1) and mothers (2.3). Excluding these coefficients, other childhood adversities have 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 prior to 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, exists between number of traumas and childhood ADHD ($\chi^2_3 = 251.8$, p < .001). No significant association exists, though, between number of childhood traumas and persistence of ADHD ($\chi^2_3 = 1.3$, p = .74).).

Comorbid DSM-IV disorders

The vast majority of the other DSM-IV CIDI disorders with onsets before age 16significantly predicted childhood ADHD. (Table 2 in Supplement 1) Only one of these, though, significantly predicts adult persistence of ADHD. This one, major depressive disorder, has an OR of 2.2 with persistence. The ORs of bipolar disorder, oppositional-defiant disorder, and conduct disorder with persistence are all very 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 exists, though, 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 count of comorbid disorders might predict persistence even though most of the individual conditions are 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 in order 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 above 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, while 4 points were assigned for having the inattentive type with sub-threshold impulsivity-hyperactivity and 1 point for either the pure inattentive type or the impulsive-hyperactive type with sub-threshold inattentive symptoms. (The omitted category of having had the pure impulsive-hyperactive type was given no points.) Pervasive childhood ADHD-related role impairment, child-adolescent MDD/dysthymia, 3+ child-adolescent anxiety or substance use disorders, paternal anxiety-mood disorder, and parental ASPD were each assigned 1 point. A strong dose-response relationship exists between scores on this index and odds of adult persistence, with the highest category of risk having an odds-ratio of 23.8 compared to the lowest category. (Table 5) The index has an area under the ROC curve (AUC) of .76 in predicting ADHD persistence among respondents with childhood ADHD

DISCUSSION

Several limitations 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 US. Nonetheless, caution is needed in interpreting results due to the fact that 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 in the introduction, 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 requires 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

are needed that consider the joint predictive effects of all these variables 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 major depressive disorder, high comorbidity, paternal (but not maternal) anxiety-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 as adults 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 ten WMH countries, but this variation is unrelated either to 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 persistent 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 due to the earlier studies focusing on clinical samples and following 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-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 four-year follow-up period. This difference might be due to Wozniak et al. excluding respondents whose nuclear family was not available for study and assessing only 8 traumatic life events compared to the more than two dozen in the WMH surveys. As 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 non-bipolar 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 under-detection 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 known to occur quite often 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 needed to investigate this possibility and, if positive, to determine if successful treatment of secondary comorbid adolescent disorders can help reduce risk of adult persistence of ADHD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

The surveys discussed in this article were carried out in conjunction with the World Health Organization World Mental Health (WMH) Survey Initiative. We thank the WMH staff for assistance with instrumentation, fieldwork, and data analysis. These activities were supported by the United States National Institute of Mental Health (R01MH070884), the John D. and Catherine T. MacArthur Foundation, the Pfizer Foundation, the US Public Health Service (R13-MH066849, R01-MH069864, and R01 DA016558), the Fogarty International Center (FIRCA R01-TW006481), the Pan American Health Organization, the Eli Lilly & Company Foundation, Ortho-McNeil Pharmaceutical, Inc., GlaxoSmithKline, and Bristol-Myers Squibb. The authors appreciate the helpful comments of Lenard Adler, Thomas Spencer, and David Van Brunt on an earlier version of the paper. A complete list of WMH publications can be found at http://www.hcp.med.harvard.edu/wmh/. The Colombian National Study of Mental Health (NSMH) is supported by the Ministry of Social Protection. The European Study of the Epidemiology of Mental Disorders (ESEMeD) was funded by the European Commission (Contract QLG5-1999-01042, SANCO 2004123), the Piedmont Region (Italy), Fondo de Investigación Sanitaria, Instituto de Salud Carlos III, Spain (FIS 00/0028), Ministerio de Ciencia y Tecnología, Spain (SAF 2000-158-CE), Red de Centros de Investigación en Epidemiología y Salud Pública, Spain (C03/09 and PI052302), Departament de Salut, Generalitat de Catalunya, Spain, and other local agencies and by an unrestricted educational grant from GlaxoSmithKline. The Lebanese National Mental Health Survey (LEBANON) is supported by the Lebanese Ministry of Public Health, the WHO (Lebanon), Fogarty International, Act for Lebanon, anonymous private donations to IDRAAC, Lebanon, and unrestricted grants from Janssen Cilag, Eli Lilly, GlaxoSmithKline, Roche, and Novartis. The Mexican National Comorbidity Survey (MNCS) is supported by The National Institute of Psychiatry Ramon de la Fuente (INPRFMDIES 4280) and by the National Council on Science and Technology (CONACy-TG30544- H), with supplemental support from the PanAmerican Health Organization (PAHO). The US National Comorbidity Survey Replication (NCS-R) is supported by the National Institute of Mental Health (NIMH; U01-MH60220) with supplemental support from the National Institute of Drug Abuse (NIDA), the Substance Abuse and Mental Health Services Administration (SAMHSA), the Robert Wood Johnson Foundation (RWJF; Grant 044708), and the John W. Alden Trust.

References

1. Barkley RA, Fischer M, Edelbrock CS, Smallish L. The adolescent outcome of hyperactive children diagnosed by research criteria: I. An 8-year prospective follow-up study. J Am Acad Child Adolesc Psychiatry 1990;29:546–557. [PubMed: 2387789]

2. Barkley RA, Fischer M, Smallish L, Fletcher K. The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. J Abnorm Psychol 2002;111:279–289. [PubMed: 12003449]

3. Faraone SV, Biederman J, Mick E. The age-dependent decline of attention deficit hyperactivity disorder: A meta-analysis of follow-up studies. Psychol Med 2006;36:159–165. [PubMed: 16420712]

4. Mannuzza S, Klein RG, Moulton JL 3rd. Persistence of Attention-Deficit/Hyperactivity Disorder into adulthood: What have we learned from the prospective follow-up studies? J Atten Disord 2003;7:93–100. [PubMed: 15018358]

5. Weiss G, Hechtman L, Milroy T, Perlman T. Psychiatric status of hyperactives as adults: A controlled prospective 15-year follow-up of 63 hyperactive children. J Am Acad Child Psychiatry 1985;24:211–220. [PubMed: 3989165]

6. Fayyad J, De Graaf R, Kessler R, Alonso J, Angermeyer M, Demyttenaere K, et al. Cross-national prevalence and correlates of adult attention-deficit hyperactivity disorder. Br J Psychiatry 2007;190:402–409. [PubMed: 17470954]

7. Kessler RC, Adler L, Barkley R, Biederman J, Conners CK, Demler O, et al. The prevalence and correlates of adult ADHD in the United States: Results from the National Comorbidity Survey Replication. Am J Psychiatry 2006;163:716–723. [PubMed: 16585449]

8. Gittelman R, Mannuzza S, Shenker R, Bonagura N. Hyperactive boys almost grown up. I. Psychiatric status. Arch Gen Psychiatry 1985;42:937–947. [PubMed: 4037987]

9. Mannuzza S, Klein RG, Bessler A, Malloy P, LaPadula M. Adult outcome of hyperactive boys. Educational achievement, occupational rank, and psychiatric status. Arch Gen Psychiatry 1993;50:565–576. [PubMed: 8317950]

10. Biederman J, Faraone S, Milberger S, Curtis S, Chen L, Marrs A, et al. Predictors of persistence and remission of ADHD into adolescence: results from a four-year prospective follow-up study. J Am Acad Child Adolesc Psychiatry 1996;35:343–351. [PubMed: 8714323]

11. Hart EL, Lahey BB, Loeber R, Applegate B, Frick PJ. Developmental change in attention-deficit hyperactivity disorder in boys: A four-year longitudinal study. J Abnorm Child Psychol 1995;23:729–749. [PubMed: 8609310]

12. Kessler RC, Üstün TB. The World Mental Health (WMH) Survey Initiative Version of the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI). Int J Methods Psychiatr Res 2004;13:93–121. [PubMed: 15297906]

13. Kessler, RC.; Üstün, TB. 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, NY: Cambridge University Press; in press

14. Pennell, B-E.; Mneimneh, Z.; Bowers, A.; Chardoul, S.; Wells, JE.; Viana, MC., et al. 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, NY: Cambridge University Press; in press

15. Harkness, J.; Pennell, B-E.; Villar, A.; Gebler, N.; Aguilar-Gaxiola, S.; Bilgen, I. 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, NY: Cambridge University Press; 2008.

16. World Bank. World Development Report 2004: Making Services Work for Poor People. Washington, DC: The International Bank for Reconstruction and Development/The World Bank; 2003.

17. Heeringa, SG.; Wells, JE.; Hubbard, F.; Mneimneh, Z.; Chiu, WT.; Sampson, N., et al. 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, NY: Cambridge University Press; in press

18. Haro JM, Arbabzadeh-Bouchez S, Brugha TS, de Girolamo G, Guyer ME, Jin R, et al. 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 2006;15:167–180. [PubMed: 17266013]

19. First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. Structured Clinical Interview for DSM-IV Axis I Disorders, Research Version, Non-patient Edition (SCID-I/NP). New York: Biometrics Research, New York State Psychiatric Institute; 2002.
20. Robins, LH.; Helzer, JE. Diagnostic Interview Schedule (DIS Version III-A). St Louis, MO: Washington University, Department of Psychiatry; 1985.
21. Adler L, Cohen J. Diagnosis and evaluation of adults with attention-deficit/hyperactivity disorder. Psychiatr Clin North Am 2004;27:187–201. [PubMed: 15063992]
22. Adler, L.; Spencer, T. The Adult ADHD Clinical Diagnostic Scale (ACDS), version 1.2. New York, NY: New York University School of Medicine; 2004.
23. Rubin, DB. Multiple Imputation for Nonresponse in Surveys. New York: John Wiley & Sons; 1987.
24. Dubowitz H, Pitts SC, Black MM. Measurement of three major subtypes of child neglect. Child Maltreat 2004;9:344–356. [PubMed: 15538034]
25. Andreasen NC, Endicott J, Spitzer RL, Winokur G. The family history method using diagnostic criteria. Reliability and validity. Arch Gen Psychiatry 1977;34:1229–1235. [PubMed: 911222]
26. Kendler KS, Davis CG, Kessler RC. The familial aggregation of common psychiatric and substance use disorders in the National Comorbidity Survey: A family history study. Br J Psychiatry 1997;170:541–548. [PubMed: 9330021]
27. Straus MA, Hamby SL, Boney-McCoy S, Sugarman DB. The revised Conflict Tactics Scales (CTS2): Development and preliminary psychometric data. J Family Issues 1996;17:283–316.
28. Kessler RC, Davis CG, Kendler KS. Childhood adversity and adult psychiatric disorder in the US National Comorbidity Survey. Psychol Med 1997;27:1101–1119. [PubMed: 9300515]
29. Wolter, KM. Introduction to Variance Estimation. New York, NY: Springer-Verlag; 1985.
30. Research Triangle Institute. SUDAAN: Professional Software for Survey Data Analysis. 8.0.1. Research Triangle Park, NC: Research Triangle Institute; 2002. computer program
31. Murphy P, Schachar R. Use of self-ratings in the assessment of symptoms of attention deficit hyperactivity disorder in adults. Am J Psychiatry 2000;157:1156–1159. [PubMed: 10873926]
32. De Quiros GB, Kinsbourne M. Adult ADHD. Analysis of self-ratings on a behavior questionnaire. Ann N Y Acad Sci 2001;931:140–147. [PubMed: 11462738]
33. Barkley RA. ADHD behavior checklist for adults. The ADHD Report 1995;3:16.
34. Ratey JJ, Greenberg MS, Bemporad JR, Lindem KJ. Unrecognized attention-deficit hyperactivity disorder in adults presenting for outpatient psychotherapy. J Child Adoles Psychopharmacol 1992;2:267–275.
35. Kooij JJ, Buitelaar JK, van den Oord EJ, Furer JW, Rijnders CA, Hodiamont PP. Internal and external validity of attention-deficit hyperactivity disorder in a population-based sample of adults. Psychol Med 2005;35:817–827. [PubMed: 15997602]
36. Barkley RA, Biederman J. Toward a broader definition of the age-of-onset criterion for attention-deficit hyperactivity disorder. J Am Acad Child Adolesc Psychiatry 1997;36:1204–1210. [PubMed: 9291721]
37. Li J, Kang C, Zhang H, Wang Y, Zhou R, Wang B, et al. Monoamine oxidase A gene polymorphism predicts adolescent outcome of attention-deficit/hyperactivity disorder. Am J Med Genet B Neuropsychiatr Genet 2007;144:430–433. [PubMed: 17427196]
38. Mackie S, Shaw P, Lenroot R, Pierson R, Greenstein DK, Nugent TF 3rd, et al. Cerebellar development and clinical outcome in attention deficit hyperactivity disorder. Am J Psychiatry 2007;164:647–655. [PubMed: 17403979]
39. Shaw P, Gornick M, Lerch J, Addington A, Seal J, Greenstein D, et al. Polymorphisms of the dopamine D4 receptor, clinical outcome, and cortical structure in attention-deficit/hyperactivity disorder. Arch Gen Psychiatry 2007;64:921–931. [PubMed: 17679637]
40. Rasmussen P, Gillberg C. Natural outcome of ADHD with developmental coordination disorder at age 22 years: A controlled, longitudinal, community-based study. J Am Acad Child Adolesc Psychiatry 2000;39:1424–1431. [PubMed: 11068898]
41. Polanczyk G, Rohde LA. Epidemiology of attention-deficit/hyperactivity disorder across the lifespan. Curr Opin Psychiatry 2007;20:386–392. [PubMed: 17551354]

42. Wozniak J, Crawford MH, Biederman J, Faraone SV, Spencer TJ, Taylor A, et al. Antecedents and complications of trauma in boys with ADHD: Findings from a longitudinal study. J Am Acad Child Adolesc Psychiatry 1999;38:48–55. [PubMed: 9893416]

43. Wilens TE, Upadhyaya HP. Impact of substance use disorder on ADHD and its treatment. J Clin Psychiatry 2007;68:e20. [PubMed: 17876905]

Table 1

World Mental Health Survey sample characteristics

Survey[1]		Sample Characteristics[2]	Field Dates	Age Range	Sample Size			Response Rate[3]
					Part I	PartII	Part II and Age 18-44	
I. WHO region: the Americas (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: the Eastern Mediterranean (EMRO)								
Lebanon	LEBANON	Stratified multistage clustered area probability sample of household residents. NR	2002-3	18+	2857	1031	595	70.0
III. WHO region: Europe (EURO)								
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

[1] 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 US National Comorbidity Survey Replication).

[2] Most WMH surveys are based on stratified multistage clustered area probability household samples in which samples of areas equivalent to counties or municipalities in the US 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 household samples 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. 8 of the 10 surveys are based on nationally representative (NR) household samples, while two others are based on nationally representative household samples in urbanized areas (Colombia, Mexico).

[3]The 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.

Table 2

Cross-national variation in the conditional prevalence of current adult DSM-IV ADHD among respondents who met criteria for ADHD in childhood by country ($\chi^2_9 = 20.3$, p = .051)

	%	(SE)[1]	(n)[2]
I. WHO region: the Americas (AMRO)			
Colombia	75.8	(10.1)	(33)
Mexico	32.8	(7.3)	(88)
USA[2]	46.0	(4.9)	(346)
II. WHO region: the Eastern Mediterranean (EMRO)			
Lebanon	52.4	(15.0)	(20)
III. WHO region: Europe (EURO)			
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)

[1] SE: Standard error of the prevalence estimate

[2] The 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.

[2] The proportion reported here differs somewhat from the estimate in a previous report (Kessler et al., 2005) because it is based on a somewhat less refined imputation equation than the one used in the previous report. This is true due to the fact that some of the predictors used in the earlier imputation equation were not available in all the surveys.

Table 3

Distributions and associations (Odds-Ratios) of childhood ADHD symptom profiles and severity with current DSM-IV adult ADHD among respondents who met criteria for ADHD in childhood pooled across the ten surveys (n = 629)

			Current ADHD among childhood cases			
	Distribution[1] %	(SE)[3]	Prevalence[2] %	(SE)[3]	OR[4]	(95% CI)[4]
I. Childhood Symptom Profiles[5]						
Inattentive (IN)	35.3	(2.6)	48.7	(6.1)	2.7*	(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*	(1.8-14.5)
Impulsive-hyperactive + Sub IN	6.4	(1.3)	50.7	(10.3)	1.7	(0.6-4.6)
IN + IH	8.8	(1.6)	84.5	(5.3)	12.4*	(4.5-34.5)
χ^2_4[6]						27.7*
II. Childhood symptom severity[7]						
High	18.7	(1.9)	62.8	(7.4)	2.0*	(1.1-3.5)
Low	81.3	(1.9)	47.0	(4.9)	1.0	--
χ^2_1						5.7*
III. Childhood treatment of ADHD[8]						
Yes	10.4	(1.8)	47.2	(8.1)	0.9	(0.4 - 2.0)
No	89.6	(1.8)	50.3	(5.1)	1.0	--
χ^2_1						0.0

*Significant at the .05 level, two-sided test

[1]Distribution: 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.

[2]Prevalence: 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.

[3]SE: Standard error of the prevalence estimate

[4]OR: Odds-ratio; CI: Confidence interval.

[5]Inattentive (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 (sub-threshold) IH: Respondents who had 6-9 childhood symptoms of inattentiveness and 1-5 symptoms of impulsivity-hyperactivity; Impulsive-hyperactive + Sub (sub-threshold) IN: Respondents who had 6-9 childhood symptoms of impulsivity-hyperactivity and 1-5 symptoms of inattentiveness; IN + IH: Respondents who had 6-9 childhood symptoms of inattentiveness and 6-9 symptoms of hyperactivity-impulsivity. The ORs are based on a pooled within-country logistic regression equation that controlled for country, age and gender.

[6]The 4 degree of freedom χ^2 evaluates the joint significance of the different childhood ADHD symptom profiles in predicting adult persistence.

[7]High 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 gender.

[8]The 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 gender.

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 ten surveys

	Childhood ADHD in the total sample						Current ADHD among childhood cases					
	Distribution[1]		Prevalence[2]				Distribution[5]		Prevalence[6]			
	%	(SE)[3]	%	(SE)[3]	OR[4]	(95% CI)[4]	%	(SE)[3]	%	(SE)[3]	OR[7]	(95% CI)[7]
I. Neglect and abuse												
Neglect	17.7	(2.1)	12.9	(1.4)	4.5*	(3.2-6.3)	17.6	(3.1)	45.8	(7.8)	1.0	(0.5-2.1)
Physical abuse	27.3	(2.1)	9.8	(0.9)	4.6*	(3.6-5.9)	30.6	(3.4)	56.0	(5.8)	1.6	(0.9-2.7)
Sexual abuse	9.5	(1.2)	11.7	(1.5)	3.0*	(2.2-4.1)	8.3	(1.9)	43.2	(7.6)	0.7	(0.3-1.5)
II. Loss												
Parental death	9.6	(1.4)	3.6	(0.5)	1.1	(0.8-1.5)	11.2	(2.2)	58.4	(10.6)	1.2	(0.5-3.0)
Parental divorce	20.9	(2.4)	6.4	(0.8)	1.1	(0.8-1.5)	20.9	(3.3)	46.0	(6.8)	1.1	(0.6-2.0)
Other major loss	10.8	(1.4)	7.8	(1.0)	2.2*	(1.6-3.0)	9.6	(1.8)	44.1	(6.7)	0.8	(0.5-1.5)
III. Paternal psychopathology												
Anxiety-mood disorder[8]	8.6	(1.3)	14.1	(2.1)	4.4*	(3.1-6.5)	11.7	(2.3)	68.1	(8.3)	2.4*	(1.1-5.5)
Substance use disorder	14.6	(2.1)	11.3	(1.5)	3.3*	(2.3-4.8)	13.7	(2.5)	46.7	(7.5)	0.9	(0.5-1.7)
ASPD[8]	12.4	(1.3)	12.4	(1.3)	2.9*	(2.2-3.9)	14.9	(2.4)	60.0	(8.4)	2.1	(1.0-4.3)
IV. Maternal psychopathology												
Anxiety-mood disorder[8]	25.9	(2.5)	13.0	(1.2)	4.5*	(3.4-5.9)	27.6	(3.4)	53.3	(6.7)	1.2	(0.7-2.1)
Substance use disorder	4.4	(0.9)	16.3	(3.5)	3.1*	(1.8-5.1)	4.1	(1.1)	46.4	(10.6)	0.8	(0.3-2.3)
ASPD[9]	3.4	(0.7)	14.9	(2.9)	2.5*	(1.5-4.1)	4.1	(1.3)	60.4	(14.3)	2.3	(0.8-6.9)
V. Parental (either father or mother) psychopathology												
Mental disorder	29.0	(2.8)	12.2	(1.2)	4.4*	(3.3-5.8)	32.0	(3.9)	55.2	(6.4)	1.4	(0.8-2.5)
Substance use disorder	17.8	(2.2)	11.9	(1.4)	3.4*	(2.4-4.7)	16.2	(2.6)	45.3	(6.5)	0.8	(0.5-1.4)
ASPD[9]	14.7	(1.4)	12.6	(1.3)	2.9*	(2.2-4.0)	17.8	(2.7)	60.5	(7.7)	2.2*	(1.2-4.2)
VI. Other adversities												
Family violence	27.8	(2.4)	9.4	(0.8)	3.3*	(2.6-4.2)	27.9	(3.2)	50.1	(5.9)	1.2	(0.7-2.0)
Economic adversity	13.8	(1.5)	8.4	(1.0)	1.7*	(1.3-2.3)	15.5	(2.5)	56.0	(6.4)	1.0	(0.6-2.0)
Severe childhood illness	7.9	(1.5)	7.5	(1.5)	1.9*	(1.2-3.0)	6.8	(1.9)	42.5	(9.9)	1.0	(0.4-2.6)
VII. Number of adversities												
None	28.4	(2.7)	1.8	(0.2)	1.0	--	25.4	(3.7)	44.7	(7.4)	1.0	--
Exactly one	26.1	(2.4)	4.2	(0.5)	1.1	(0.8-1.4)	28.3	(3.3)	54.0	(7.0)	1.1	(0.6-1.8)
Exactly two	14.8	(2.0)	5.5	(0.8)	1.5*	(1.1-2.0)	14.5	(2.5)	48.9	(7.2)	1.1	(0.6-2.1)
Exactly three	9.4	(1.3)	7.2	(1.0)	1.9*	(1.3-2.7)	9.8	(1.7)	52.1	(8.2)	1.0	(0.5-2.0)
Four or more	21.2	(2.0)	15.9	(1.5)	4.9*	(3.6-6.7)	22.0	(2.8)	51.9	(5.8)	1.4	(0.8-2.3)
χ^2_4[10]				151.8*						3.3		
(n)				(11,422)						(629)		

*Significant at the .05 level, two-sided test

[1]Distribution: The conditional prevalence of the childhood adversity indicated in the row among respondents with a history of childhood ADHD

[2]Prevalence: The conditional prevalence of childhood ADHD among respondents with the childhood adversity in the row

[3]SE: Standard error of the prevalence estimate

[4] OR: Odds-ratio; CI: Confidence interval. Each OR in Sections I-VI is based on a separate pooled within-country logistic regression equation that controlled for country, age and gender in the total sample to predict childhood ADHD. All the ORs in Section VII, in comparison, are based on a single equation.

[5] Distribution: The conditional prevalence of the childhood adversity indicated in the row among respondents with current adult ADHD

[6] Prevalence: The conditional prevalence of current adult ADHD among respondents with a history of childhood ADHD among respondents with the childhood adversity in the row.

[7] OR: Odds-ratio; CI: Confidence interval. Each 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 gender in the sub-sample 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.

[8] Major depression, panic disorder, or generalized anxiety disorder

[9] Anti-social personality disorder

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

Table 5

Distributions and associations (Odds-Ratios) of scores on the composite risk index with current DSM-IV adult ADHD among respondents who met criteria for ADHD in childhood pooled across the ten surveys (n = 629)

	Current ADHD among childhood cases							
	Distributon[1]		Prevalence[2]		Conditional distribution[3]			
	%	(SE)[4]	%	(SE)[4]	%	(SE)[4]	OR[5]	(95% CI)[5]
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*	(1.6-7.6)
3-6	32.1	(2.7)	58.6	(9.5)	37.6	(4.2)	8.3*	(2.9-23.8)
7+	9.9	(1.7)	85.7	(5.1)	17.1	(3.2)	23.8*	(7.6-74.4)
χ^2_3 [6]								32.8*
AUC = .76								

*Significant at the .05 level, two-sided test

[1]Distribution: The conditional prevalence of scores on the risk index among respondents with a history of childhood ADHD

[2]Prevalence: 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

[3]Conditional distribution: The proportion of all respondents with adult ADHD who have scores at each level of the risk index

[4]SE: Standard error of the prevalence estimate

[5]OR: Odds-ratio; CI: Confidence interval. The ORs are based on a single logistic regression equation that controlled for country in the sub-sample of respondents with a history of childhood ADHD to predict current adult ADHD.

[6]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.