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Patterns and predictors of ADHD persistence into adulthood: Results from the National Comorbidity Survey Replication

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Abstract

BACKGROUND—Despite growing interest in adult ADHD, little is known about predictors of persistence of childhood cases into adulthood.

METHODS—A retrospective assessment of childhood ADHD, childhood risk factors, and a screen for adult ADHD were included in a sample of 3197 18–44 year old respondents in the National Comorbidity Survey Replication (NCS-R). Blinded adult ADHD clinical reappraisal interviews were administered to a sub-sample of respondents. Multiple imputation (MI) was used to estimate adult persistence of childhood ADHD. Logistic regression was used to study retrospectively reported childhood predictors of persistence. Potential predictors included socio-demographics, childhood ADHD severity, childhood adversity, traumatic life experiences, and comorbid DSM-IV child-adolescent disorders (anxiety, mood, impulse-control, and substance disorders).

RESULTS—36.3% of respondents with retrospectively assessed childhood ADHD were classified by blinded clinical interviews as meeting DSM-IV criteria for current ADHD. Childhood ADHD severity and childhood treatment significantly predicted persistence. Controlling for severity and excluding treatment, none of the other variables significantly predicted persistence even though they were significantly associated with childhood ADHD.

CONCLUSIONS—No modifiable risk factors were found for adult persistence of ADHD. Further research, ideally based on prospective general population samples, is needed to search for modifiable determinants of adult persistence of ADHD.

Keywords

Attention Deficit Hyperactivity Disorder; Diagnosis And Classification, Epidemiology

A number of prospective studies have followed children with ADHD into late adolescence or early adulthood to estimate prevalence of persistence and effects of ADHD on other adult outcomes (Barkley et al 2004; Biederman et al 1996b; Fischer et al 2002; Mannuzza et al 1993; Mannuzza et al 1998; Rasmussen and Gillberg 2000; Satterfield and Schell 1997; Weiss and Hechtman 1993). Controversy has surrounded the resulting persistence estimates (Barkley 1997; Hill and Schoener 1996) due to the estimated persistence varying substantially depending on the criteria used to select the original sample, the edition of DSM used to make diagnoses of adult cases, and whether adult symptom assessment was based on self-reports or informant-reports (Barkley et al 2002; Biederman et al 2000; Faraone et al 2000b). It is much more clear, though, that many childhood cases, as high as 60% in some studies, continue to have clinically significant symptoms of ADHD as adults whether or not they are defined as meeting full criteria for adult ADHD (Biederman et al 2000; Rasmussen and Gillberg 2000; Weiss and Hechtman 1993). It is also clear that childhood ADHD is a risk factor for numerous adverse adult outcomes, especially in interaction with comorbid disorders (Barkley et al 2004; Biederman et al 1996b; Crowley et al 1998; Fischer et al 2002).

Much less is known about why some cases of ADHD remit while others persist into adulthood. Only a small number of studies have considered the predictors of ADHD persistence. Most of these focused on persistence into late adolescence (Biederman et al 1996a; Fischer et al 1993; Greene et al 1997; Lahey et al 1994) rather than adulthood (Barkley et al 2004). None considered a comprehensive set of risk factors. Several consistent predictors of persistence have nonetheless been found: severity of childhood ADHD, comorbidity with other child and adolescent disorders, and various aspects of childhood adversity. It would be useful to replicate and extend these results in larger and more representative samples to pinpoint modifiable determinants of persistence. Respondents in the first generation of large community psychiatric epidemiological surveys of children that assessed ADHD (Conners et al 2003; Jensen et al 1999) will enter adulthood in the next decade and should be followed to study childhood predictors of adult ADHD. Prior to that time, though, analysis of such predictors can be carried out using retrospective designs. The current report presents such an analysis from the National Comorbidity Survey Replication (NCS-R; Kessler and Merikangas 2004), a nationally representative household survey of adult DSM-IV mental disorders. Prevalence of adult ADHD was assessed along with retrospective assessments of prevalence and correlates of childhood ADHD. A previous study by Murphy and Barkley (1996) used a similar design in a sample of adults from central Massachusetts and found an adult ADHD prevalence of 4.7% when both current and retrospectively recalled childhood symptoms had to meet DSM-IV criteria. However, Murphy and Barkley did not examine predictors of ADHD persistence. Although retrospective analysis of this sort is limited by recall bias, it can provide useful preliminary data in situations where prospective studies do not exist and would take a significant amount of time to collect (Schlesselman 1982).

METHODS

Sample

The NCS-R is a nationally representative survey of English-speaking household residents ages 18 and older carried out between February 2001 and April 2003 (Kessler and Merikangas 2004). Face-to-face interviews were completed with 9282 respondents. The response rate was 70.9%. Consent was verbal rather than written to parallel procedures in the baseline NCS (Kessler et al 1994) for purposes of trend comparison. The Human Subjects Committees of

Harvard Medical School and the University of Michigan both approved these recruitment and consent procedures. The interview was administered in two parts. Part I was administered to all respondents and included a core diagnostic assessment. Part II was administered to a probability sub-sample of 5692 Part I respondents (100% of those with any Part I disorder and a probability sub-sample of others) and included questions about correlates and additional disorders. ADHD was assessed in Part II and further restricted to respondents in the age range 18–44 ($n = 3197$) based on concern that older respondents would be less reliable in reporting childhood experiences. The ADHD sub-sample was weighted to be representative of the US population. A more detailed description of NCS-R weighting is presented elsewhere (Kessler et al 2004).

Respondents in the ADHD sub-sample were divided into four sampling strata based on their responses to the main NCS-R interview: those who denied ever having symptoms of childhood ADHD; those who reported too few childhood symptoms to meet criteria for childhood ADHD; those who reported enough symptoms to be retrospectively classified childhood cases, but who denied adult symptoms; and childhood cases who reported adult symptoms. An attempt was made to contact by telephone and administer a semi-structured adult ADHD clinical interview to 30 respondents in each of the first three strata and 60 in the fourth. The final quota sample of 154 respondents was slightly larger than the target because more pre-designated respondents kept their interview appointments than expected. Clinical reappraisal sample respondents were weighted to be representative of the US population in the age range of the sample. More details about the ADHD clinical reappraisal sample design are reported elsewhere (Kessler et al, 2005).

ADHD

The NCS-R assessment of childhood ADHD was based on the Diagnostic Interview Schedule for DSM-IV (Robins and Helzer 1985). Respondents were asked retrospective questions about frequent childhood occurrence of the nine DSM-IV Criterion A symptoms of inattention and nine symptoms of hyperactivity-impulsivity. Respondents who reported 6+ symptoms of either type were then administered follow-up questions about the other DSM-IV criteria. Respondents who were classified as having had ADHD in childhood were then asked a single question about whether they continued to have current problems with attention or hyperactivity-impulsivity. The clinical reappraisal interviews, in comparison, included comprehensive evaluations of ADHD based on the Adult ADHD Clinical Diagnostic Scale (ACDS) V 1.2 (Adler and Cohen 2004; Adler and Spencer 2004), a semi-structured interview that includes the ADHD Rating Scale (ADHD-RS; DuPaul et al 1998) to assess childhood ADHD and an adaptation of the ADHD-RS to assess current adult ADHD. The ACDS has been used in several clinical trials of adult ADHD (Michelson et al 2003; Spencer et al 2001). Four experienced clinical interviewers (all Ph.D. clinical psychologists) carried out the ACHS clinical reappraisal interviews. Clinical interviewers received 40 hours of training from two board certified psychiatrists who specialize in adult ADHD (LA, TS) and successfully completed five practice interviews. All clinical interviews were tape recorded and reviewed by a clinical supervisor. Weekly meetings were used to prevent drift. A clinical diagnosis of adult ADHD required six symptoms of either inattention or hyperactivity-impulsivity during the six months before the interview (DSM-IV Criterion A), at least two Criterion A symptoms before age seven (Criterion B), some impairment in at least two areas of living during the past six months (Criterion C), and clinically significant impairment in at least one of these areas (Criterion D). No attempt was made to operationalize DSM-IV diagnostic hierarchy rules (Criterion E). Inter-rater reliability for these diagnoses, as assessed by the intra-class correlation, was 0.78.

Predictors of ADHD persistence into adulthood

We examined five classes of predictors: socio-demographics; childhood ADHD severity; childhood ADHD treatment; comorbid child-adolescent DSM-IV disorders; and childhood adversities. Socio-demographics included respondent age, sex, and race-ethnicity. Childhood ADHD severity was indicated by two measures: symptom profiles and role impairments. Symptom profiles were divided into five categories: pure attention-concentration symptoms (AD); pure impulsive-hyperactive symptoms (HD); threshold AD symptoms (i.e., 6 or more AD symptoms) in conjunction with sub-threshold HD symptoms (i.e., 1–5 HD symptoms); sub-threshold AD symptoms in conjunction with threshold HD symptoms; and threshold symptoms of both AD and HD. Role impairment was assessed with four yes-no questions about whether ADHD interfered a lot with functioning at home, school, in their social life, and in personal relationships. High impairment was defined as endorsing all four questions. Respondents were asked whether they ever received professional treatment for their ADHD and, if so, the age when they first received treatment. We required treatment to begin as of age 15 to be included as a predictor. No information was obtained about type or persistence of treatment.

Comorbid DSM-IV disorders were retrospectively assessed using Version 3.0 of the WHO Composite International Diagnostic Interview (Kessler and Üstün 2004), a fully structured lay-administered diagnostic interview that assesses the lifetime prevalence and age-of-onset of anxiety disorders (panic disorder, generalized anxiety disorder, specific phobia, social phobia, agoraphobia, obsessive-compulsive disorder, post-traumatic stress disorder, separation anxiety disorder), mood disorders (major depressive disorder, dysthymia, bipolar I and II disorders), impulse-control disorders (oppositional-defiant disorder, conduct disorder, intermittent explosive disorder), and substance use disorders (alcohol and drug abuse and dependence). OCD was assessed only in a random one-third of the Part II sample, while other comorbid disorders were assessed among all people who were assessed for ADHD. Organic exclusion rules and diagnostic hierarchy rules were used in making diagnoses. Only disorders with onsets as of age 15 were used as predictors. As detailed elsewhere (Kessler et al 2004), good concordance was found between CIDI diagnoses and diagnoses based on the Structured Clinical Interview for DSMIV (SCID; First et al 2002) in a probability sub-sample of NCS-R respondents. Area under the receiver operator characteristic curve (AUC) was in the range .65–.81 for anxiety disorders, .75 for major depressive episode, .62–.88 for substance disorders, and .76 for any anxiety, mood, or substance disorder. No validation was made of the impulse-control disorders, as these are not assessed in the SCID.

The NCS-R retrospectively assessed 23 childhood adversities that included three types of child maltreatment (physical abuse, sexual abuse, neglect), six of maternal and paternal psychopathology (major depression, panic, generalized anxiety disorder, antisocial personality disorder, substance dependence, suicide attempt), one of financial adversity (public assistance in childhood), and seven of family instability (childhood death of a parent, parental divorce, otherwise living without a childhood male or female head of household, foster care, living away from home for at least one year for other reasons, living with a large number of different adult caregivers). Questions were also included about exposure to 27 traumatic life events that occurred as of age 15. The child maltreatment measures were based on standard measures used in child welfare research (Dubowitz et al 2004). The measures of parental psychopathology were based on the Family History RDC interview (Andreasen et al 1977) and its expansion (Kendler et al 1997). The measures of family financial adversity and instability were based on measures developed for the baseline NCS (Kessler et al 1997). The measures of traumatic life events were taken from the CIDI assessment of PTSD.

Analysis methods

The multiple imputation (MI) method (Rubin 1987) was used to assign predicted diagnoses of clinician-assessed adult ADHD to respondents who did not participate in the ADHD clinical reappraisal interviews based on the four ADHD sampling strata described above. As reported elsewhere (Kessler et al in press), a strong monotonic relationship was found between these strata and blinded clinical diagnoses of adult ADHD, with area under the receiver operator characteristic curve (AUC) of .86. As detailed elsewhere (Kessler et al in press), simulation was used to implement the MI method using standard procedures (Rubin 1987). The increase in variance due to between-replication variance adjusted for the additional variance in estimates introduced by using imputation rather than direct clinical evaluation of all respondents who were screened for adult ADHD.

The proportion of childhood ADHD cases estimated to meet full criteria for clinical-assessed adult ADHD was estimated as a mean of the individual-level MI case estimates. Predictors of persistence were estimated using logistic regression analysis. We report parallel associations of the same predictors with retrospectively assessed childhood ADHD. Because the sample design used weighting and clustering, all parameters were estimated using the Taylor series linearization method (Wolter 1985), a design-based method implemented in the SUDAAN software system (Research Triangle Institute 2002). Significance tests of sets of coefficients in the logistic regression equations were made using Wald χ^2 tests based on design-corrected MI coefficient variance-covariance matrices. Statistical significance was evaluated using two-sided design-based .05 level tests.

RESULTS

Socio-demographic correlates of persistence

Adult persistence of ADHD, defined as the conditional prevalence of clinician-assessed adult ADHD among the 8.1% (0.6) of NCS-R respondents classified as having had childhood ADHD, was estimated to be 36.3% (4.6) in the total sample. (Table 1) Persistence does not differ significantly by respondent sex ($\chi^2_1 = 0.8$, $p = .376$), age ($\chi^2_2 = 0.7$, $p = .705$), or race-ethnicity ($\chi^2_3 = 1.3$, $p = .726$). This contrasts with two of these three socio-demographic variables being meaningfully related to retrospectively reported childhood ADHD – a significantly elevated odds-ratio (OR) of childhood ADHD among males compared to females (1.6) and a lower OR of childhood ADHD among Non-Hispanic Blacks and Hispanics than Non-Hispanic Whites (0.6–0.8).

Childhood ADHD severity

The majority of respondents with retrospectively assessed childhood ADHD reported either pure AD (35.2%) or pure HD (21.3%) symptoms in childhood. The remaining 43.5%, those with AD and HD, have significantly elevated odds of adult persistence (OR = 2.4; $\chi^2_1 = 9.3$, $p = .002$). (Table 2) Adult persistence is equivalent among respondents whose childhood symptoms were pure AD versus pure HD (28.0% vs. 28.2%, $z = 0.2$), higher among those whose childhood symptoms included both sub-threshold AD and threshold HD (35.7%), and highest among those whose childhood symptoms included threshold AD with either sub-threshold HD (49.3%) or threshold HD (41.7%). In addition, after controlling for these childhood symptom profiles, retrospectively assessed pervasiveness of childhood role impairment is significantly associated with adult persistence (OR = 3.4, $\chi^2_1 = 8.7$, $p = .003$).

Treatment

Childhood treatment of ADHD is associated with significantly elevated odds of adult persistence (OR = 4.5 (1.7–11.8), $\chi^2_1 = 9.9$, $p = .002$) after controlling for socio-demographics and childhood ADHD severity.

Comorbid DSM-IV disorders

The 17 other DSM-IV CIDI child- and adolescent-onset disorders assessed in the CIDI are significantly and positively associated with childhood ADHD ($\chi^2_{17} = 430.9$, $p < .001$) (Table 3) and have elevated bivariate ORs (i.e. ORs greater than 1.0) in predicting childhood ADHD (15 statistically significant). These early-onset disorders, however, do not significantly predict adult persistence either individually or overall ($\chi^2_{17} = 13.9$, $p = .675$). The one large OR (5.7) is associated with the most rare disorder in the analysis, obsessive-compulsive disorder, and the estimate is very unstable. Replication in an independent dataset would consequently be needed before we concluded that OCD is an important predictor of ADHD persistence.

Childhood adversities

The childhood adversities assessed in the NCS-R are significantly and positively associated overall with childhood ADHD ($\chi^2_{23} = 258.5$, $p < .001$). (Table 4) Twenty-one of the 23 adversities have elevated bivariate ORs in predicting childhood ADHD (18 statistically significant). These adversities, however, do not significantly predict adult persistence of ADHD either individually or overall ($\chi^2_{22} = 14.2$, $p = .895$).

Although the number of respondents exposed to individual traumatic events as of age 15 was too small to allow analysis to be carried out for the 27 separate traumatic events assessed in the NCS-R, aggregate analysis was carried out that compared the 51.4% of NCS-R respondents who reported being exposed to none of these events as of age 15 with those having one (26.1%), two (11.0%), or more (11.5%) than two such experiences. This early exposure to trauma is a significant correlate of childhood ADHD ($\chi^2_3 = 88.8$, $p < .001$), with monotonic ORs in the range 1.5–5.4 for exposure to between one and three or more traumas. (Table 5) However, childhood trauma exposure does not significantly predict adult persistence of ADHD ($\chi^2_3 = 1.4$, $p = .701$).

DISCUSSION

The most important limitation of the above analysis is that childhood ADHD and predictors of persistence were assessed with retrospective self-reports. Retrospective reports have the problem of recall bias, while self-reports have the additional problems of some respondents lacking insight into their condition and others consciously failing to admit symptoms (Barkley et al 2002). In the case of children with ADHD, self-report problems are addressed by basing diagnoses largely on informants (Jensen et al 1999). Informant reports are much more difficult to obtain for adults, though, making it necessary to base diagnoses on self-reports. Only one methodological study compared self-reports with informant-reports of ADHD in an adult non-clinical sample (Murphy and Schachar 2000). Fairly strong associations were found between the two reports with no significant difference in estimated symptom severity. The methodological literature on retrospective reports about childhood experiences is much larger. Several systematic reviews (Brewin et al 1993; Hardt and Rutter 2004; Maughan and Rutter 1997) concluded that despite general under-reporting, childhood experiences are recalled with sufficient accuracy to provide useful information in retrospective studies.

The assessment of adult ADHD in the NCS-R is also limited in four ways. First, DSM-IV criteria for ADHD were developed with children in mind and offer only limited guidance regarding adult ADHD. This is of concern because clinical studies show that symptoms are

more heterogeneous and subtle in adults than children (De Quiros and Kinsbourne 2001; Wender et al 2001), leading some clinical researchers to suggest an increase in the variety of symptoms assessed (Barkley 1995), a reduction in the severity threshold (Ratey et al 1992), or a reduction in the DSM-IV six-of-nine symptom requirement (McBurnett 1997) for ADHD among adults. To the extent that these changes would lead to a more valid assessment, our estimate of 38.7% adult persistence is conservative. Second, only the clinical reappraisal subsample received a direct clinical evaluation of adult ADHD. Diagnoses were imputed in other cases. Although the imputation equation was strong ($AUC = .86$), the need to impute made it impossible to carry out symptom-level investigations. Third, even though the clinical interview has been used in previous studies, no standard clinical validation of adult ADHD exists, limiting the interpretability of results. Fourth, sample bias could exist, either because cases might be over-represented among non-respondents or among population segments excluded from the sampling frame (most notably homeless people and people living in institutional settings).

Within the context of these limitations, the results are consistent with previous studies in finding that a substantial minority of children with DSM-IV ADHD continues to meet criteria in early and middle adulthood. No attempt was made to assess persistence of clinically significant sub-threshold symptoms, as the sparseness of the NCS-R data prohibited this type of specification search. The results of other studies would lead us to believe, though, that this proportion might be as large as the proportion that meets full criteria (Barkley et al 2002; Biederman et al 2000; Fergusson and Horwood 1995).

The finding of no age differences in persistence raises the possibility that ADHD cases that persist into adulthood no longer have the age-related pattern of remission seen in childhood and adolescence. Another possibility is that genuine age differences are suppressed by age-related recall bias. The finding that childhood ADHD is significantly more common among men than women is consistent with previous research (Scahill and Schwab-Stone 2000). The finding that sex is not related to persistence contrasts with the high proportion of women in clinical samples of adult ADHD. One plausible interpretation of this discrepancy is that women are more likely than men to seek treatment for ADHD (Biederman et al 2004), a possibility that needs to be investigated in future general population screening studies. It is also possible that, due to the greater prevalence of antisocial behavior among males than females with ADHD, more males enter the corrections system and are not ascertainable in either clinical or epidemiologic studies. The finding of no race-ethnic difference in persistence contrasts with the retrospective finding of higher childhood prevalence among Non-Hispanic Whites than either Non-Hispanic Blacks or Hispanics. The latter was an unexpected finding due to previous research not finding race-ethnic differences in childhood ADHD (Lahey et al 1994) and might be due to a race-ethnic difference in reporting accuracy.

The finding that childhood ADHD severity significantly predicts persistence is consistent with prospective studies (Biederman et al 1996a; Hart et al 1995; Fischer et al under review). This might reflect nothing more than the fact that symptoms that decrease in severity in adulthood are more likely to remain clinically significant if they started at a high level of severity. If so, though, we would expect an association between age and persistence or an interaction between age and childhood severity in predicting persistence, neither of which was found.

We are not aware of any previous study that distinguished childhood symptom profiles of AD-only, HD-only, and combined AD-HD in predicting adult persistence. However, our finding that childhood cases with threshold AD symptoms and at least some HD symptoms are more likely than other cases to persist into adulthood is consistent with the clinical observation, confirmed in at least two prospective studies (Biederman et al 2000; Hart et al 1995), that AD symptoms are more likely than HD symptoms to persist into adulthood. This greater importance of childhood AD than HD is also of clinical significance due to the fact that HD continues to

be a greater focus of treatment than AD among children with ADHD. It might be that persistence of symptoms into adulthood would be reduced if AD became a greater focus of childhood treatment. A methodological interpretation is also possible: that adult symptoms, in which AD predominates over HD, are associated with recall of childhood symptoms, leading to a greater recollection of childhood AD than childhood HD among adult cases.

Our finding that pervasiveness of childhood ADHD impairment predicts persistence is consistent with the finding in one prospective study that persistent cases had higher childhood ADHD role impairment than remitted cases (Biederman et al 1996a). In some sense, this finding is not surprising, as we would expect that proportional decrease in symptoms with age would be expected to result in residual symptoms being more likely to continue to meet criteria for disorder when the base level was higher in childhood. Another possible explanation is that current adult symptoms are associated with recall bias about the severity of childhood symptoms.

The finding that childhood treatment of ADHD is associated with elevated risk of adult persistence might be interpreted as due to unmeasured aspects of severity. Treatment, in this view, stands as a proxy for severity, which is imperfectly measured by the two rough severity indicators in the NCS-R. The possibility exists, of course, that childhood treatment promotes adult persistence. Indeed, a suggestion along these lines has been made that stimulant treatment promotes subsequent drug use disorders among children with ADHD (Goldman et al 1998). However, review of long-term adult follow-up studies of patients who, as children, were included in treatment studies shows clearly that childhood stimulant treatment is actually associated with a reduction in adult substance use disorders (Faraone and Wilens 2003). Another possibility is that history of treatment sensitizes respondents to the existence of their current residual symptoms, which might actually be substantially higher than in childhood if treatment was discontinued, leading to more accurate reports about those symptoms among respondents with a history of treatment than among those who were not treated in childhood.

The retrospective finding in the NCS-R that comorbid disorders as of age 15 are significantly related to childhood ADHD is consistent with previous studies of children in both clinical and epidemiological samples (Hazell 1997; Pliszka 1998; Pliszka 2000). A noteworthy methodological implication of this consistency is the demonstration that adults are able to provide retrospective reports about childhood disorders that yield results consistent with those obtained in direct studies of children. This makes it all the more striking that we failed to find significant associations of comorbidity as of age 15 and the persistence of adult ADHD. It is important to be clear that adult ADHD is comorbid with many other adult DSM disorders in the NCS-R. However, comorbidity in early adolescence (i.e., as of age 15), at least as retrospectively assessed, among respondents who had ADHD in childhood, did not predict whether the ADHD continued into adulthood. This failure to find a predictive association of comorbid conditions is inconsistent with a prospective epidemiological survey that documented an effect of adolescent OCD in predicting adult persistence of ADHD symptoms (Peterson et al 2001) as well as with a prospective clinical study that documented effects of child and adolescent anxiety, mood, and impulse-control disorders in predicting ADHD persistence (Biederman et al 1996a). This inconsistency suggests, at least indirectly, that respondents with adult ADHD might be downwardly biased in their retrospective recall of child and adolescent disorder. Another possibility is that the prospective studies were in error because they did not control for severity of childhood ADHD, raising the possibility that the gross associations of comorbidity with persistence were mediated by childhood ADHD symptom severity. However, further analysis of the NCS-R data (results not reported, but available on request) failed to find evidence for this possibility.

The retrospective NCS-R finding that childhood adversity is significantly related to childhood ADHD is consistent with previous studies that were carried out among children (Biederman et al 1995b; Chronis et al 2003), while we failed to find significant associations of childhood adversity with adult ADHD persistence. This failure is inconsistent with a prospective clinical study that documented considerably higher childhood adversity among cases of ADHD that persisted into late adolescence versus remitted (Biederman et al 1996a). However, this study reported only gross associations without adjusting for the presumed associations of adversity with childhood ADHD symptom severity. Although it is consequently possible that the gross associations of childhood adversity with ADHD persistence were mediated by childhood symptom severity, further analysis of the NCS-R data (results not reported, but available on request) failed to find evidence of this. The implication is that ADHD, once it occurs, has a course that is impervious to the major stressors assessed in the NCS-R, which means that aggressive treatment in childhood probably holds out more promise than interventions aimed at reducing environmental stress in reducing adult persistence of the disorder.

One potentially important predictor of persistence that we failed to include in the NCS-R is family history of ADHD. Family genetic studies show that ADHD is highly heritable (Faraone et al in press; Galili-Weisstub and Segman 2003). In addition, family studies document high familial aggregation of adult ADHD (Biederman et al 1995a; Manshadi et al 1983), that persistence into mid-adolescence is strongly related to family history of the disorder (Biederman et al 1996a; Faraone et al 2000c), and that family aggregation of ADHD is stronger in the families of adolescents with ADHD than children with ADHD (Biederman et al 1998a; Biederman et al 1998b; Faraone et al 2000a). This effect of familiarity on persistence is clearly not mediated by the kinds of childhood adversity studied here, as the latter do not significantly predict persistence. However, genetic or family environmental factors could help explain or specify the effects we found of childhood symptom severity on persistence. More work is needed to elucidate such connections.

Given the high adult persistence of ADHD and the substantial impairment associated with this disorder in adulthood, it is striking that symptom severity was the only modifiable risk factor found to predict persistence significantly. It should be noted that the failure to find effects of childhood adversity is consistent with the results of previous analyses of the baseline NCS in which childhood adversities were found to have powerful effects in predicting first onset of numerous early-onset mental disorders, but generally did not predict adult persistence of these disorders (Kessler et al 1997). The failure to find effects of childhood and adolescent comorbidity is somewhat more surprising, as we would expect the existence of comorbid disorders to be an indirect indicator of disorder severity. Methodological interpretations are difficult to envision, as plausible types of bias (e.g., lower recall of less serious childhood comorbidities) would be expected to induce an upward bias in the estimated effects of early comorbid disorders on ADHD persistence.

As noted in the introduction, prospective study of the childhood predictors of adult persistence takes many years to complete. A great many such studies exist in other areas of medicine, most notably in the clinical epidemiology of cardiovascular disorders, where very large samples are followed for a decade or more in search of baseline predictors of disease progression (Jensen et al 2004; Wannamethee et al 2004). Given the substantial societal cost of adult ADHD, a long-term multi-center prospective study of this sort would be justified for ADHD as well. Before carrying out such a study, though, more work is needed to refine our understanding of predictors in small short-term prospective and retrospective studies and in secondary analyses of existing prospective studies. The absence of published reports on these predictors is striking in light of the fact that quite a few small prospective studies now exist that have followed childhood cases of ADHD into adulthood. It would be useful to bring these datasets together to carry out parallel analyses of childhood predictors of adult persistence, making sure to focus

initially on the effects of childhood disorder severity and to control for these effects in examining other predictors. A somewhat more ambitious undertaking, but one that might well be worth the effort given the enormous societal burden of adult ADHD, would be to carry out a coordinated follow-up survey of respondents in all the major existing prospective studies of ADHD children in order to collect retrospective data about a wide range of potential childhood predictors. Consistent documentation of significant associations in this blended retrospective-prospective design would create a firm foundation for launching a new generation of prospective studies and subsequently of secondary preventive interventions aimed at studying and manipulating modifiable predictors of persistence of ADHD into adulthood.

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Table 1

Associations (odds-ratios) of socio-demographic variables with childhood ADHD in the total sample (n = 3197) and with current adult ADHD in the subsample of childhood cases (n = 346)

Childhood ADHD in the total sample					Current adult ADHD among childhood cases							
	Distribution ¹		Prevalence ²		OR	(95% CI)	Distribution ³		Prevalence ⁴		OR	(95% CI)
	%	(se)	%	(se)			%	(se)	%	(se)		
Total sample					—	—					--	--
Sex												
Male	58.9	(3.4)	9.8	(1.0)	1.6*	(1.1–2.1)	64.3	(5.5)	39.7	(6.4)	1.4	(0.7–2.7)
Female	41.1	(3.4)	6.4	(0.6)	1.0	--	35.7	(5.5)	31.5	(5.8)	1.0	--
					$\chi^2_1=8.2, p=.004$						$\chi^2_1=0.8, p=.376$	
Age												
18–24	27.6	(3.2)	7.6	(1.1)	0.9	(0.6–1.4)	29.7	(5.8)	39.1	(7.4)	1.1	(0.5–2.5)
25–34	31.5	(3.1)	8.2	(0.7)	1.0	(0.7–1.4)	27.6	(5.0)	31.9	(5.7)	0.8	(0.4–1.7)
35–44	40.9	(3.1)	8.3	(1.0)	1.0	--	42.6	(6.2)	37.8	(7.0)	1.0	--
					$\chi^2_2=0.1, p=.938$						$\chi^2_2=0.7, p=.705$	
Race-ethnicity												
Non-Hispanic white	74.6	(3.2)	9.2	(0.8)	1.0	--	77.6	(5.2)	37.8	(5.4)	1.0	--
Non-Hispanic black	9.9	(1.7)	5.6	(1.1)	0.6	(0.4–1.0)	8.1	(2.6)	29.6	(7.9)	0.7	(0.3–1.7)
Hispanic	11.3	(2.1)	5.9	(1.1)	0.6	(0.4–1.0)	8.7	(2.6)	28.0	(9.5)	0.7	(0.2–2.0)
Other	4.2	(1.4)	7.0	(2.5)	0.8	(0.4–1.8)	5.6	(3.0)	48.6 (	(18.0)	1.7	(0.4–7.2)
					$\chi^2_3=6.7, p=.083$						$\chi^2_3=1.3, p=.726$	
(n)				(3197)							(346) ⁵	

ADHD, attention deficit hyperactivity disorder; se, standard error; OR, odds-ratios; CI, confidence interval

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

¹ Distribution of socio-demographic variables among respondents with a history of childhood ADHD

² Prevalence of childhood ADHD among respondents in sub-samples defined by the socio-demographic variables

³ Distribution of socio-demographic variables among respondents with current adult ADHD

⁴Prevalence of current adult ADHD among respondents with a history of childhood ADHD in sub-samples defined by the socio-demographic variables

⁵The 346 cases with lifetime ADHD equals the unweighted number of respondents who met criteria for the disorder. This number is larger than the product of the conditional prevalence estimates in the sub-groups multiplied by the total sample size (which yields an estimate of approximately 268 weighted cases) because the prevalence estimates are based on weighted data and the weights are inversely related to probability of ADHD due to the over-sampling of respondents who had Part I disorders into the Part II sample in which ADHD was assessed.

Table 2

Associations (odds-ratios) of childhood ADHD symptom profiles and severity with current adult ADHD in the sub-sample of childhood cases (n=346)

	Distribution ¹		Current adult ADHD among childhood cases		
	%	(se)	%	Prevalence ² (se)	OR ³ (95% CI)
I. Detailed symptom profiles ⁴					
AD-only	35.2	(3.5)	28.0	(7.4)	1.0
HD-only	21.3	(2.1)	28.2	(7.8)	0.9 (0.3–3.0)
AD+subthreshold HD	34.3	(3.2)	49.3	(6.0)	2.5* (1.2–5.5)
HD+subthreshold AD	4.7	(1.2)	35.7	(12.5)	1.2 (0.3–4.7)
AD+HD	4.5	(1.0)	41.7	(14.2)	2.2 (0.6–8.0)
$\chi^2_4=10.6, p=.032$					
II. Collapsed symptom profiles ⁴					
AD-only or HD-only	56.5	(3.4)	28.1	(5.4)	1.0
All others	43.5	(3.4)	47.0	(5.7)	2.4* (1.3–4.2)
$\chi^2_1=9.3, p=.002$					
III. Severity					
High	19.6	(2.3)	57.3	(10.4)	3.3* (1.5–7.4)
Low	80.4	(2.3)	31.2	(4.1)	1.0
$\chi^2_1=8.7, p=.003$					

ADHD, attention deficit hyperactivity disorder; se, standard error; OR, odds-ratio; CI, confidence interval; AD, attention deficit; HD, hyperactivity disorder

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

¹ Distribution of childhood ADHD symptom profiles and severity among respondents with a history of childhood ADHD.

² Prevalence of current adult ADHD among respondents with a history of childhood ADHD in sub-samples defined by the childhood symptom profiles and severity

³ Controlling for age, sex, and race-ethnicity

⁴ AD-only = the respondent had 6+ childhood symptoms of inattentiveness, but no symptoms of hyperactivity-impulsivity; HD-only = the respondent had 6+ childhood symptoms of hyperactivity-impulsivity, but no symptoms of inattentiveness; AD+subthreshold HD = the respondent had 6+ childhood symptoms of inattentiveness and 1–5 symptoms of hyperactivity-impulsivity; HD+ subthreshold AD = the

respondent had 6+ childhood symptoms of hyperactivity-impulsivity and 1–5 symptoms of inattentiveness; AD + HD = the respondent had 6+ childhood symptoms of hyperactivity-impulsivity and 6+ symptoms of inattentiveness.

Associations (odds-ratios) or comorbid DSM-IV disorders as of age 15 with childhood ADHD in the total sample (n=3197) and with current adult ADHD in the sub-sample of childhood cases (n=346)

Childhood ADHD in the total sample				Current adult ADHD among childhood cases			
	Distribution ¹		Prevalence ²		OR ³	(95% CI)	
	%	(se)	%	(se)			
I. Mood disorders							
Major depressive disorder	14.0	(2.1)	20.3	(3.1)	3.3 [*]	(2.2–4.9)	
Dysthymia	4.9	(1.1)	27.6	(5.3)	4.8 [*]	(2.9–8.1)	
Bipolar I–II disorder	8.4	(1.5)	37.3	(5.1)	7.3 [*]	(4.6–11.7)	
II. Anxiety disorders							
Post-traumatic stress disorder	11.7	(1.8)	26.6	(4.7)	5.4 [*]	(3.4–8.6)	
Panic disorder	4.5	(1.1)	20.5	(4.9)	3.3 [*]	(1.8–6.1)	
Generalized anxiety disorder	7.6	(1.5)	31.2	(6.2)	5.8 [*]	(3.5–9.7)	
Specific phobia	25.2	(2.6)	15.6	(1.6)	2.7 [*]	(2.1–3.5)	
Social phobia	33.6	(3.1)	22.1	(2.4)	4.3 [*]	(3.1–6.0)	
Agoraphobia	2.6	(1.0)	28.0	(9.3)	4.7 [*]	(1.9–11.6)	
Obsessive-compulsive disorder ⁷	0.7	(0.5)	15.7	(15.4)	3.3	(0.4–27.6)	
III. Impulse-control disorders							
Oppositional-defiant disorder	36.1	(3.5)	36.3	(3.5)	9.8 [*]	(5.9–16.2)	
Conduct disorder	27.8	(2.9)	26.9	(2.6)	5.2 [*]	(4.0–6.9)	
Intermittent explosive disorder	22.6	(2.6)	23.3	(2.5)	4.0 [*]	(2.8–5.7)	
IV. Substance use disorder							
Alcohol abuse or dependence	2.6	(0.8)	18.3	(5.6)	2.6 [*]	(1.3–5.0)	
Alcohol dependence	3.6	(1.2)	31.0	(7.5)	5.1 [*]	(2.4–10.7)	
Drug abuse or dependence	2.7	(1.0)	19.4	(5.0)	2.5 [*]	(1.3–4.9)	
Drug dependence	1.1	(0.7)	12.8	(7.4)	1.7	(0.5–6.3)	

Childhood ADHD in the total sample				Current adult ADHD among childhood cases			
Distribution ¹		Prevalence ²		Distribution ⁴		Prevalence ⁵	
%	(se)	%	(se)	%	(se)	%	(se)
		OR ³ (95% CI)				OR ⁶ (95% CI)	
(3197)				(346)			
(n)							

ADHD, attention deficit hyperactivity disorder; se, standard error; OR, odds-ratio; CI, confidence interval; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition.

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

¹ Distribution of comorbid disorders among respondents with a history of childhood ADHD

² Prevalence of childhood ADHD among respondents in sub-samples defined by the comorbid disorders

³ Controlling for age, sex, and race-ethnicity

⁴ Distribution of comorbid disorders among respondents with current adult ADHD

⁵ Prevalence of current adult ADHD among respondents with a history of childhood ADHD in sub-samples defined by the comorbid disorders

⁶ Controlling for age, sex, race-ethnicity, and childhood ADHD symptom profiles and severity

⁷ Assessed only in a random one-third sub-sample of Part II respondents, resulting in higher standard errors and confidence intervals than other disorders

Table 4

Associations (odds-ratios) of childhood adversities with childhood ADHD in the total sample (n=3197) and with current ADHD among childhood cases (n=346)

	Childhood ADHD in the total sample				Current adult ADHD among childhood cases			
	Distribution ¹ %	(se)	Prevalence ² %	(se)	OR ³	(se)	95% CI	
I. Child maltreatment								
Physical abuse	19.6	(2.2)	23.8	(2.7)	4.3*	(2.7)	(3.1–5.9)	28.3 (5.1) 52.3 (9.2) 2.1 (0.9–4.9)
Sexual abuse	6.1	(1.4)	15.2	(3.3)	2.5*	(3.3)	(1.4–4.3)	5.8 (2.8) 34.2 (12.8) 0.9 (0.2–3.5)
Neglect	13.7	(2.3)	26.5	(3.7)	4.8*	(3.7)	(3.1–7.6)	14.7 (4.4) 39.0 (10.6) 1.2 (0.4–3.1)
II. Maternal psychopathology								
Major depression	5.3	(1.3)	33.5	(7.3)	8.7*	(7.3)	(4.2–18.1)	5.8 (2.4) 39.6 (14.0) 0.7 (0.3–1.5)
Panic disorder	18.6	(2.7)	22.0	(2.9)	3.7*	(2.9)	(2.5–5.5)	19.0 (4.8) 37.2 (9.4) 0.8 (0.5–1.4)
Generalized anxiety disorder	19.8	(2.7)	22.8	(2.7)	4.1*	(2.7)	(2.7–6.1)	20.2 (5.0) 37.2 (10.1) 0.8 (0.4–1.4)
Alcohol-drug dependence	2.4	(0.9)	14.1	(5.4)	1.9	(5.4)	(0.7–5.3)	3.0 (1.9) 45.8 (20.1) 0.8 (0.5–1.2)
Adult antisocial behavior	4.8	(1.2)	22.2	(5.3)	3.8*	(5.3)	(1.9–7.7)	6.3 (2.4) 47.2 (15.7) 0.8 (0.5–1.4)
Suicide attempt	9.8	(2.2)	25.2	(5.4)	4.6*	(5.4)	(2.5–8.7)	9.6 (4.1) 35.8 (13.6) 0.8 (0.4–1.4)
III. Paternal psychopathology								
Major depression	12.1	(2.1)	11.0	(2.0)	5.6*	(2.0)	(1.3–24.1)	13.7 (3.6) 41.2 (9.9) 1.1 (1.0–1.2)
Panic disorder	14.8	(2.5)	11.6	(2.0)	3.0*	(2.0)	(1.5–5.8)	18.7 (4.4) 46.0 (9.5) 1.1 (1.0–1.2)
Generalized anxiety disorder	19.3	(2.7)	4.0	(1.9)	4.4*	(1.9)	(2.7–7.1)	23.9 (5.6) 45.1 (10.5) 1.1 (1.0–1.2)
Alcohol-drug dependence	18.3	(2.9)	13.7	(2.0)	4.9*	(2.0)	(3.1–7.6)	20.2 (4.7) 40.0 (8.2) 1.1 (1.0–1.2)
Adult antisocial behavior	23.2	(3.2)	14.8	(1.7)	4.4*	(1.7)	(2.9–6.8)	22.9 (5.3) 35.8 (8.0) 1.1 (0.9–1.2)
Suicide attempt	13.8	(2.3)	11.6	(2.1)	3.6*	(2.1)	(1.5–8.6)	16.0 (3.9) 42.3 (9.3) 1.1 (1.0–1.2)
IV. Financial adversity								
Public assistance	15.8	(2.1)	10.5	(1.4)	1.6*	(1.4)	(1.1–2.4)	16.1 (4.1) 37.2 (9.4) 1.2 (0.5–3.1)
V. Family instability								
Death of a parent	1.0	(0.6)	5.6	(2.6)	0.7	(2.6)	(0.3–2.1)	0.6 (0.6) 19.5 (16.9) 0.3 (0.0–325.6)

	Childhood ADHD in the total sample					Current adult ADHD among childhood cases						
	Distribution ¹		Prevalence ²		OR ³	(95% CI)	Distribution ⁴		Prevalence ⁵			
	%	(se)	%	(se)			%	(se)	%	(se)	OR ⁶	(95% CI)
Parental divorce	32.6	(3.3)	10.1	(1.1)	1.5*	(1.1–2.1)	31.5	(6.1)	35.1	(6.3)	0.9	(0.4–1.9)
Other absence of male head	10.7	(2.3)	9.2	(1.8)	1.5	(0.9–2.4)	9.8	(3.2)	33.3	(8.6)	1.2	(0.5–2.9)
Other absence of female head	0.6	(0.4)	15.2	(9.5)	2.3	(0.5–9.8)	0.0	(0.0)	0.0	(0.0)	0.0*	(0.0–0.0)
Foster care placement	3.1	(0.9)	19.8	(5.1)	2.9	(1.4–6.0)	4.6	(2.1)	54.9	(21.4)	3.2	(0.4–25.0)
Other living away from home	4.4	(1.3)	14.5	(3.8)	2.2*	(1.1–4.3)	2.8	(1.6)	23.6	(12.6)	0.9	(0.2–4.2)
Large number of different caregivers	26.2	(2.8)	11.7	(1.1)	1.8*	(1.4–2.5)	25.8	(5.2)	35.8	(7.5)	1.0	(0.4–2.2)
(n)	(3197)					(346)						

ADHD, attention deficit hyperactivity disorder; se, standard error; OR, odds-ratio; CI, confidence interval

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

¹ Distribution of childhood adversities among respondents with a history of childhood ADHD

² Prevalence of childhood ADHD among respondents in sub-samples defined by the childhood adversities

³ Controlling for age, sex, and race-ethnicity

⁴ Distribution of childhood adversities among respondents with current adult ADHD

⁵ Prevalence of current adult ADHD among respondents with a history of childhood ADHD in sub-samples defined by the childhood adversities

⁶ Controlling for age, sex, race-ethnicity, and childhood ADHD symptom profiles and severity

Table 5

Associations (odds-ratios) of childhood-adolescent traumatic life experiences with childhood ADHD in the total sample (n=3197) and with current adult ADHD among childhood cases (n=346)

	Childhood ADHD in the total sample					Current adult ADHD among childhood cases						
	Distribution ¹		Prevalence ²		OR ³	(95% CI)	Distribution ⁴		Prevalence ⁵		OR ⁶	(95% CI)
	%	(se)	%	(se)			%	(se)	%	(se)		
Traumatic experiences												
Three or more	28.2	(3.4)	19.9	(2.5)	1.5*	(1.0–2.3)	28.6	(5.8)	36.8	(6.8)	0.9	(0.4–2.2)
Two	18.5	(2.7)	13.6	(2.0)	3.2*	(2.0–5.1)	15.3	(3.8)	30.1	(7.5)	0.6	(0.2–1.6)
One	22.8	(2.6)	7.0	(0.8)	5.4*	(3.6–8.0)	23.0	(4.8)	36.8	(8.7)	0.8	(0.3–2.4)
None	30.5	(3.1)	4.8	(0.7)	1.0	--	33.1	(7.2)	9.2	(7.9)	1.0	--
(n)						$\chi^2_3=88.8, p=.000$						$\chi^2_3=1.4, p=.701$
					(3197)							(346)

ADHD, attention deficit hyperactivity disorder; se, standard error; OR, odds-ratios; CI, confidence interval

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

¹ Distribution of traumatic experiences among respondents with a history of childhood ADHD

² Prevalence of childhood ADHD among respondents in sub-samples defined by number of traumatic experiences

³ Controlling for age, sex, and race-ethnicity

⁴ Distribution of traumatic experiences among respondents with current adult ADHD

⁵ Prevalence of current adult ADHD among respondents with a history of childhood ADHD in sub-samples defined by number of traumatic experiences

⁶ Controlling for age, sex, race-ethnicity, and childhood ADHD symptom profiles and severity