Review article

Prevalence and correlates of adult attention-deficit hyperactivity disorder: meta-analysis

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Background
In spite of the growing literature about adult attention-deficit hyperactivity disorder (ADHD), relatively little is known about the prevalence and correlates of this disorder.

Aims
To estimate the prevalence of adult ADHD and to identify its demographic correlates using meta-regression analysis.

Method
We used the MEDLINE, PsycLit and EMBASE databases as well as hand-searching to find relevant publications.

Results
The pooled prevalence of adult ADHD was 2.5% (95% CI 2.1–3.1). Gender and mean age, interacting with each other,

were significantly related to prevalence of ADHD. Meta-regression analysis indicated that the proportion of participants with ADHD decreased with age when men and women were equally represented in the sample.

Conclusions
Prevalence of ADHD in adults declines with age in the general population. We think, however, that the unclear validity of DSM–IV diagnostic criteria for this condition can lead to reduced prevalence rates by underestimation of the prevalence of adult ADHD.

Declaration of interest
None.

Although attention-deficit hyperactivity disorder (ADHD) has long been thought to be a disabling and common disorder that occurs only in childhood, more recent research, including prospective longitudinal follow-up studies, suggests that ADHD persists into adulthood in a high proportion of cases.[1–8] Adult ADHD studies indicate a high degree of genetic predisposition,[9,10] and reveal structural and functional brain abnormalities[11–14] congruent with neuropsychological data.[14–16] Attention-deficit hyperactivity disorder is a serious risk factor for comorbid psychiatric disorders (antisocial personality disorder, substance misuse and affective disorders),[17,18] and also shows significant correlation with poor socio-economic outcome and functional impairment (lower level of education, higher level of unemployment, and higher rates of unsuccessful marriages, criminality and road traffic accidents).[1,7,8,18–23]

In spite of the growing literature dealing with adult ADHD, relatively little is known about the prevalence of the disorder among adults and its correlates. To our knowledge no meta-analysis of the epidemiological data on adult ADHD has been published. The aim of our study was to estimate the prevalence of ADHD in adulthood using a meta-regression approach and to identify demographic factors that might influence the prevalence of ADHD in a given population.

Method

Study selection

We searched MEDLINE, PsycLit and EMBASE for publications dealing with the epidemiology of adult ADHD. Only publications in English were considered. As a first step, we created four databases with the keywords ADULT, ADHD, EPIDEMIOLOGY and PREVALENCE respectively. Second, we connected the ADULT and ADHD databases with a logical 'and' operation, generating a new database containing only those publications that were part of both ADULT and ADHD databases in the first step. The other two databases (EPIDEMIOLOGY and PREVALENCE) were

connected with the 'or' operation, creating a new database including all publications that were originally in the EPIDEMIOLOGY and PREVALENCE databases. During the final step, the two new databases were connected with the 'and' operation. In addition to this search procedure, we used the reference lists of the identified publications to find further relevant articles. After excluding follow-up and family studies – which do not provide prevalence data for adult ADHD – and studies that dealt with the prevalence of ADHD in special groups (people with panic or bipolar disorder, drug addiction or obesity, or people in prison), 12 population-based studies remained:

(a) one study estimated the cumulative incidence of ADHD at the age of 19 years based on retrospective analysis;[24]

(b) one study estimated the prevalence of adult ADHD among licensed drivers;[25]

(c) three studies estimated the prevalence of ADHD among university students;[26–28]

(d) one study estimated the prevalence of ADHD among a non-clinical sample from an out-patient psychiatric service;[29]

(e) six studies provided a community-based estimate: oppositional defiant disorder only and ADHD only v. oppositional defiant disorder + ADHD in clinic and community adult samples;[30] a cross-national survey;[31] the National Comorbidity Survey Replication;[32] the Mexican National Comorbidity Survey;[33] a telephone survey;[34] and the Nijmegen Health Area Study 2.[19]

For our meta-regression analyses six studies were omitted. Three of these studies (Kessler et al,[32] Medina-Mora et al[33] and Fayyad et al[31]) were not included because they did not provide raw data for the prevalence and demographic variables necessary for the computations. The study by Barbaresi et al[24] was not included because it dealt only with the cumulative incidence of ADHD between the ages of 5 and 19 years and accordingly provided information about ADHD in adolescents rather than

in adults. The study by Weyandt et al[26] was not included because it measured only the prevalence of attention-deficit symptoms and not the prevalence of adult ADHD. Finally, we omitted the study by Gadow et al[30] because these authors did not use DSM–IV[35] criteria for the diagnosis of adult ADHD. The modified diagnostic criteria used by Gadow et al did not include age at onset or functional impairment criteria, and applied a threshold of five rather than six symptoms.[30] Lowering the diagnostic threshold concerning symptom counts has a dramatic effect on prevalence estimates; inclusion of data based on a lower symptoms threshold would therefore have introduced substantial heterogeneity in the meta-analysis.

Variables

For the purpose of the meta-analysis we extracted the following domains or variables from the articles that were finally included:

(a) data describing the study – date of publication, country, number of arms;

(b) data describing the target population – sample size, mean age, age range, standard deviation for age range, gender composition (proportion of males in the sample);

(c) diagnostic tools for adult ADHD – self-report, structured interview;

(d) results – prevalence rate according to DSM–IV criteria (total and subtypes if provided), prevalence rate according to alternative criteria, if available (total and subtypes, if given).

Statistical analysis

A mixed-effect (with fixed and random effects) meta-regression – a meta-analytic technique of multivariate linear regression across studies – was applied to estimate the prevalence of ADHD across various study samples and in order to evaluate the impact of potential demographic variables of interest including age and gender on the prevalence estimates. The meta-regression analysis that we adopted in this investigation was based on van Houwelingen et al's general linear mixed-model technique based on the approximate likelihood approach.[36] In particular, the log-odds of the observed prevalence in each study were regressed using intercept and basic study-level demographic covariates that included average age and gender composition from each of the individual studies. Interaction between the two covariates (age, gender composition) was also included in the model. In addition, a random-effect intercept term representing systematic between-study variation (heterogeneity) was also incorporated in the meta-regression model. A common weighted prevalence estimate for ADHD was calculated as a DerSimonian & Laird estimator, based on the random effects component of the mixed model that incorporated both fixed and random effects.[37]

Results

Study design

In all the articles included in the analysis we found that although the sample sizes were large (typically several hundreds of participants), the authors collected samples of convenience, which do not assure representativeness. Accordingly, the raw estimates of prevalence from these studies cannot be extended to the general population. We note that in the study by Faraone & Biederman,[34] raw prevalence estimates were weighted by US census data (based on age, ethnicity, education, geographic region and number of telephone lines within the household) in order to derive prevalence estimates generalisable for the population; however, the final derived prevalence estimates remain questionable in light of the high refusal rate (approximately 80%) in the target population that was used to derive the prevalence estimates in the sampling phase of the study. In the study by DuPaul et al, in addition to the problem with representativeness, there were remarkable differences across the three subsamples in terms of the number, gender and age range of the participants (Tables 1 and 2).[27]

Age

In most of the studies, the sample's mean age was low compared with the mean age of a typical adult population. Specifically, although the mean ages were 19.4–44.9 years for all samples in the analysis (the mean age, weighted by the number of participants in each study, was 34 years), for the majority of samples the mean age ranged between 19.4 and 28.5 years. Only one study had a mean age of 44.9 years,[19] whereas two studies had a mean age of around 35 years.[25,34] (Of these two studies, Faraone & Biederman provided estimates for mean age based on weighting using the US census data;[34] Table 1).

Gender

With the exception of one study sample (the USA arm of the study by DuPaul et al),[27] the gender proportions were neither balanced nor representative of the target population. There were extreme differences in the male:female ratio across the groups in the study by DuPaul et al,[27] with a substantial departure from the

Table 1 Demographic data of samples included in the meta-analysis

Study	Sample size, n	Age, years Mean (s.d.)	Age, years Range	Gender proportions Male/female, %
Murphy & Barkley[25]	720	35 (13.2)	17–84	60/40
Heiligenstein et al[28]	448	20.6 (4)	17–46	56/44
Du Paul et al[27,a]	1209			
Italy	197	21.6 (3)[b]	18–35	14/86[b]
New Zealand	213	19.4 (4)	17–51	17/83
USA	799	21.3 (4.9)	17–49	51/49
Kooij et al[19]	1815	44.9[c]	18–75	45/55
Faraone & Biederman[34]	966	35.9[d]		48/52
Almeida Montes et al[29]	149	28.5		32.9/67.1

a. Data available separately by subsample (country) in the original publication.
b. Our calculation from the given data (number of females and males in all groups).
c. Our calculation from the given proportion of age range groups and numbers of participants.
d. Derived from US census data referred to in the original article.

Table 2 Descriptive data for studies included in the meta-analysis

	Design	Diagnostic procedure
Murphy & Barkley[25]	One-stage sampling Community-based study Non-representative, sample of convenience	DSM–IV symptom list Self-report
Heiligenstein et al[28]	One-stage sampling College students Non-representative, sample of convenience	DSM–IV symptom list Self-report No data from childhood
DuPaul et al[27]	One-stage sampling Three study arms University students Non-representative, sample of convenience	DSM–IV symptom list Self-report No data from childhood
Kooij et al[19]	One-stage sampling Community-based study in general practices Non-representative, probability sample	DSM–IV Modified Dutch version of DSM–IV ADHD Rating Scale Self-report and structured interview
Faraone & Biederman[34]	One-stage sampling Community-based telephone survey Non-representative, probability sample	DSM–IV symptom list Self-report
Almeida Montes et al[29]	One-stage sampling Non-clinical population from a psychiatric out-patient service Non-representative, sample of convenience	DSM–IV MINI Structured interview

ADHD, attention-deficit hyperactivity disorder; MINI, Mini International Neuropsychiatry Interview.

population gender distribution in two arms of this study, possibly as a result of the above-mentioned convenience sampling (Table 1).

Diagnosis

The studies included in our meta-analysis applied different methodology and design with regard to sampling and diagnosing adults with ADHD (Table 2). All studies employed DSM–IV diagnostic criteria, even though all – except for Faraone & Biederman[34] and Almeida Montes et al[29] – questioned the validity of DSM–IV criteria for ADHD when applied to adults.[35] In terms of association between symptoms that underlie the DSM–IV diagnosis of adult ADHD and functional impairment (used as an external validator of the disorder), Kooij et al found the strongest association from four symptoms being present (as opposed to the threshold of six symptoms according to the DSM–IV diagnostic system).[19] DuPaul et al[27] and Heiligenstein et al[28] applied alternative diagnostic criteria with a lower threshold, besides the original DSM–IV criteria. Although Murphy & Barkley used only DSM–IV diagnostic criteria in their study, they suggested the possibility of modifying these criteria for adult ADHD in future.[25] Faraone & Biederman considered two types of diagnoses for adult ADHD: a 'broad' diagnosis for screening purposes, which followed the DSM–IV criteria but was more inclusive concerning symptom severity; and a 'narrow' diagnosis based solely on DSM–IV criteria.[34]

Estimated prevalence and correlates of adult ADHD

Mixed-effect meta-regression analysis was applied to estimate the prevalence across samples and to investigate prevalence as a function of gender composition and mean age in the respective samples. Results of the meta-regression analysis indicated that the pooled prevalence of ADHD across samples was 2.5% (95% CI 2.1–3.1; $t=42.3$, $P<0.0001$) (Fig. 1).

Adopting the likelihood approach as recommended by Hardy & Thompson[36] and van Houwelingen,[38] heterogeneity among studies included in the meta-analysis was tested by the likelihood ratio statistic, by comparing the maximum log-likelihood (LL) of the random-effect model with that of the fixed-effect model. Our results showed that the random and fixed-effects models yielded maximum LL values of -9.9 and -42.5 respectively. This indicates a statistically significant heterogeneity across studies ($\chi^2=65.2$, d.f.=1, $P<0.0001$), which (as shown by subsequent analyses) was due, at least in part, to the principal demographic variables that we examined in our study. In particular, our results showed that the prevalence of ADHD was significantly related to the gender composition in the sample ($t=4.34$, $P=0.012$, standardised beta for log-odds of observed prevalence 15.19×10^{-2}) and to the mean age ($t=3.03$, $P=0.039$, standardised beta for log-odds of observed prevalence 20.98×10^{-2}). Furthermore, the interaction between the two covariates also reached statistical significance ($t=-3.42$, $P=0.027$, standardised beta for log-odds of observed prevalence 0.50×10^{-2}). The association between the proportion of participants with ADHD and gender composition and mean age is shown in Fig. 2. Owing to the statistically significant interaction reported above, for illustrative purposes the association of prevalence with gender composition is displayed at various ages (20, 30 and 40 years; Fig. 2(a)); for younger age groups the prevalence increases, whereas for the older age group prevalence decreases with higher proportion of males in the sample. Analogously, for illustrative purposes the association of prevalence with mean age was broken down by male percentage of the sample (a third, a half, two-thirds; Fig. 2(b)); the prevalence decreases with age when men are represented at 50% or more in the sample, but increases with age when women are predominantly represented in the sample (male proportion, 33.3%).

We note that the above results are based on prevalence data that relied on DSM–IV diagnostic criteria. Individual studies included in our meta-analysis used other diagnostic criteria as well, but these alternative criteria varied between studies, precluding a meaningful pooling of the results. Indeed, as Table 3 shows, these alternative thresholds lead to substantial variation in the results (prevalence between 2.5% and 42.3%), reflecting the heterogeneity of the alternative diagnostic approaches in the individual studies.

Discussion

In general, epidemiological data about adult ADHD have been collected from three different sources: family studies, follow-up
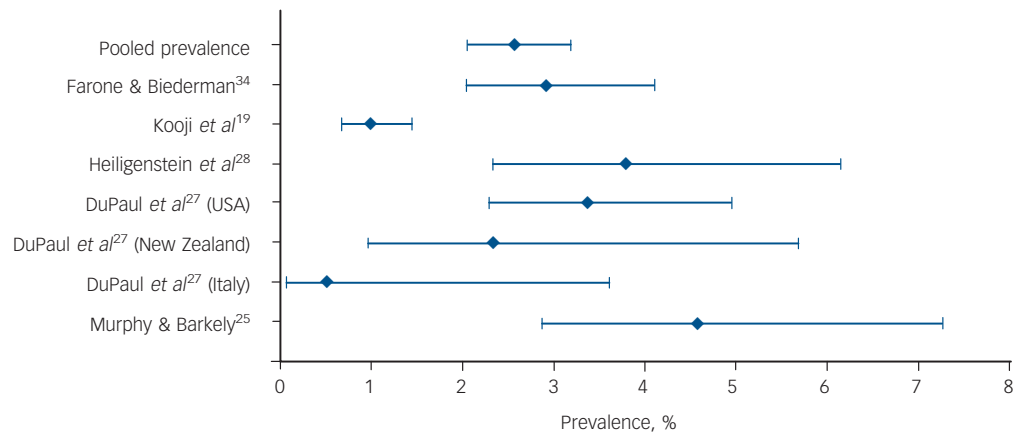
Fig. 1 Prevalence estimates and 95% confidence intervals of adult attention-deficit hyperactivity disorder in individual investigations and pooled prevalence estimated across studies using meta-regression analysis.

studies and population-based studies. In family studies, parents of children who did not have ADHD – who had taken part in case–control ADHD studies as the control group – were examined for adult ADHD. The results of these studies cannot be generalised since they used a strongly selected sample, excluding a genetically predisposed group – parents of children with ADHD.[34]

Follow-up studies are long-term prospective studies designed to determine the persistence of ADHD among adolescents and adults by following an index ADHD group of school-aged children and a matched control group. Follow-up studies show that ADHD persists in 4–66% of the cases into adulthood.[1–8] Such variability in the persistence of the disorder into adulthood can be explained – at least in part – by methodological differences such as small sample sizes; non-representative, predominantly clinical samples; different diagnostic criteria among and across studies; and changing the source of information during the follow-up from parent report to self-report only. These methodological differences imply that follow-up studies are difficult to compare and the results of those studies can neither be generalised nor used for estimating prevalence of ADHD in adulthood.

Population-based studies estimated prevalence rates of adult ADHD at 1–7.3% applying DSM–IV criteria.[19,24,25,27–29,31–35] Most of these studies were designed for direct estimation of the prevalence of adult ADHD in a target population such as a community, university students, prisoners or a special population of patients. These studies typically used a large sample and therefore were usually appropriate for estimating prevalence with sufficient precision. However, they did not assure representativeness, since they were based on a sample of convenience. In general, the mean age of the participants was low compared with a typical adult population, and there were several studies in which the gender proportion of the sample was significantly unbalanced. In addition, the diagnostic tools and the approach for the identification of cases usually varied from study to study.

Gadow *et al* provided estimates of the prevalence of adult ADHD using a large, representative sample of the general population.[30] Nevertheless, because these authors applied only modified diagnostic criteria, their prevalence data are difficult to compare with the prevalence estimates from other studies that relied on the original DSM–IV classification. Two studies, being parts of large-scale epidemiological surveys – the National Comorbidity Survey[39] and the World Health Organization (WHO) World Mental Health Surveys[40] – did not provide crude

(a)

(b)
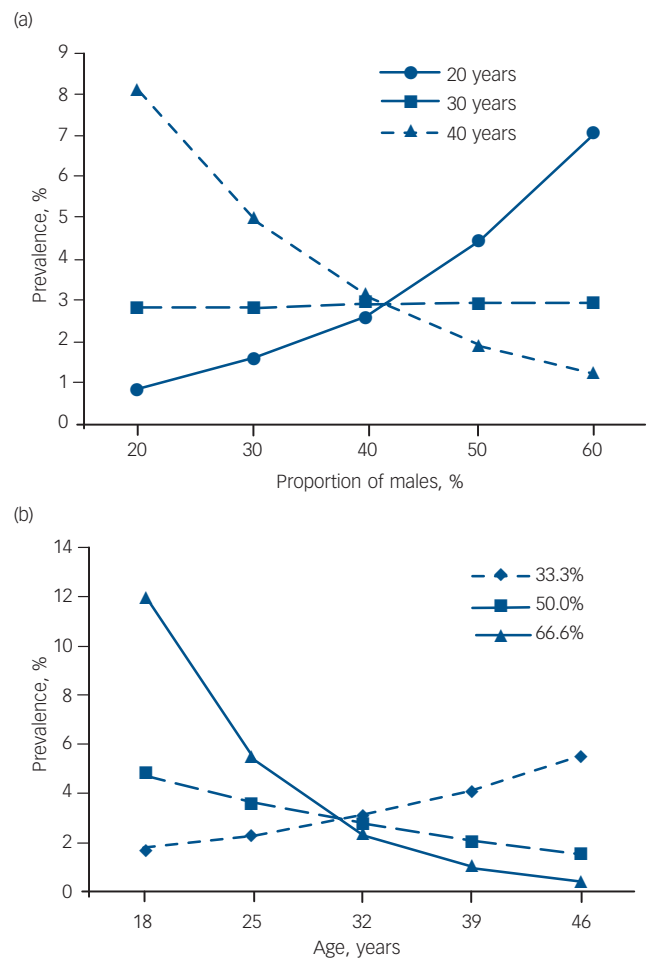
Fig. 2 Relationship between gender composition (% male) and prevalence (%) of adult attention-deficit hyperactivity disorder (ADHD). Meta-regression analysis indicated that gender and mean age, interacting with each other, were statistically significantly related to the prevalence of ADHD in the sample. (a) Relationship between gender composition and prevalence at ages 20, 30 and 40 years. (b) Relationship between age and prevalence as a function of gender composition (a third, a half, two-thirds males).

Table 3 Results of the studies included in the meta-analysis

	Prevalence defined by DSM–IV criteria, %				Prevalence defined by criteria other than DSM–IV, %			
	Total[a]	I[a]	HI	Comb.	Total[a]	I[a]	HI	Comb.
Murphy & Barkley[25]	4.7	1.3	2.5	0.9	No data available			
Heiligenstein et al[28]	4	2.24[b]	0.88	0.88	11	3.74[b]	3.96	3.30
					Reduced number of symptoms required (4)			
DuPaul et al[27]								
Italy	1.01	0.51[b]	0.51	0	42.31	8.82[b]	23.42	98.68
New Zealand	2.81	0.47[b]	2.34	0	36.06	9.50[b]	17.08	9.48
USA	3.39	0.75[b]	2.15	0.5	26.91	4.97[b]	13.90	7.15
					Reduced number of symptoms required (3)			
Kooij et al[19]	1	0.2	0.5	0.3	2.5	0.3	1.2	1.0
					Reduced number of symptoms required (4)			
Faraone & Biederman[34]	2.9	0.7	1.1	1.1	16.4	5.8	3.7	6.9
					'Broad' ADHD[c]			
Almeida Montes et al[29]	5.37				No data available			

ADHD, attention-deficit hyperactivity disorder; Comb., combined subtype; HI, hyperactive–impulsive subtype; I, inattentive subtype.
a. Total: all subtypes of ADHD pooled (inattentive subtype, hyperactive–impulsive subtype, combined subtype).
b. Our calculation from data given in the original article.
c. Referred as screening diagnosis in the original article.

estimates for the prevalence of adult ADHD in their sample; they used indirect estimation in order to assess the prevalence of adult ADHD in the general population. The first of these studies (Kessler et al[32]) examined an US sample, whereas the second (Fayyad et al[31]) estimated cross-national prevalence in ten countries. We note that despite these two studies applying the same general approach, the first estimated prevalence at 4.4%,[32] whereas the second estimated the prevalence in the US sample at 5.2%.[31] Based on the authors' comments, this discrepancy is attributable to the fact that certain predictors for the prevalence estimation that were used in the first (USA only) study were not available in the second (multinational) study. In the second study, the prevalence estimates of adult ADHD across samples showed a substantial variation: they were between 1.2% and 7.3%, with an estimated general cross-national prevalence of 3.4%.[31] In both studies, prevalence estimates were based on multiple imputation using a combination of directly interviewed cases and multiply imputed cases from the remainder of the sample. In all cases (directly interviewed and multiply imputed) in both samples the individuals were aged 18–44 years; prevalence estimates for higher age ranges were based on weighting data.[31,32] The aforementioned indirect estimations (applied in both studies) of the prevalence of adult ADHD in the general population hinge on prediction equations that were obtained in a relatively small sample (n=154). It is not clear how reliably these equations can predict the occurrence of ADHD, and what the exact predictors are. With regard to the multinational study, it must be noted that the prediction equation of the US sample was extrapolated to other countries, a potential limitation pointed out by the authors. A third study, conducted as part of the WHO survey, estimated the 12-month prevalence of ADHD in Mexico;[33] however, like the parent study it did not provide a crude prevalence estimate for the targeted sample and therefore was not included in our meta-analysis.

In summary, published estimates of the prevalence of adult ADHD vary greatly.[19,25,27–29,31–34] After reviewing the pertinent publications, we attributed this variability to methodological and diagnostic differences between the studies. In addition, only self-reports were used as a source of information and in some studies there was a lack of information about the relevant childhood symptoms that would be necessary for the proper diagnosis of adult ADHD.[26–28]

Correlation of prevalence with gender and age

Our finding of a pooled prevalence rate for adult ADHD of 2.5% (95% CI 2.1–3.1) seems to be conservative in the context of the research discussed above. Our pooled prevalence estimates were derived from studies that provided data for crude prevalence based on strict DSM–IV criteria for diagnosing ADHD. In two of these studies, however, indirect estimates were derived by assessing ADHD symptoms in childhood and asking only a single question about the persistence of problems with ADHD into adulthood.[31,32]

Polanczyk et al recently estimated the worldwide prevalence of ADHD in a meta-regression analysis of 102 articles regarding child and adolescent ADHD.[41] Although the pooled prevalence of ADHD in children and adolescents according to these authors was 5.29%, they also reported that the prevalence in adolescents was around 3%.[41] This estimate is consistent with our pooled prevalence data, especially in light of the finding about the relationship between age and prevalence of ADHD.

A growing number of studies indicate that biased samples might underlie extreme gender effects on the prevalence of ADHD in clinically referred paediatric study samples. Specifically, some of these studies suggest that a weaker association with conduct disorder and disruptive behaviour in girls compared with boys might result in lower numbers of female referrals.[42–44] In contrast to the clinical samples, in which male:female ratios as high as 10:1 have been observed,[45,46] community samples showed a less extreme gender ratio (male:female risk 3:1) in the prevalence of ADHD in childhood.[43,44] Compared with paediatric and adolescent studies, adult ADHD studies have generally shown a more balanced distribution of prevalence in men and women. This may be attributable to the fact that whereas childhood referrals are usually initiated by parents or teachers, in adulthood self-referrals are common. The observation that women with ADHD have more internalising problems than men, which leads to a higher rate of self-referrals in adulthood,[47] may underlie the more balanced gender ratio in adult samples.

In the studies that were included in our analysis, samples were community-based and the authors found heterogeneous gender ratios but no significant gender effect on prevalence in their samples when applying DSM–IV diagnostic criteria.[19,25,27–29,34] In two studies that were not included in our meta-analysis owing

to the lack of crude prevalence data,[31,32] the authors found modest gender effects on prevalence, with a significantly higher proportion of men in their ADHD group. In spite of the findings of the studied articles that supported no significant gender effect on prevalence, using the raw data of the individual studies we identified gender as another factor that has an impact on the prevalence of adult ADHD. In this case – as in the case of effect of age – we presume that methodological differences and questions concerning sample selection and case identification underlie the absence of or modest appearance of gender effects in community-based samples.

Our findings indicate that the prevalence of adult ADHD has a significant negative association with age, although this association is moderated by the gender composition of the sample. The explanation and the potential practical use of this finding are complex. Specifically, available literature and clinical experience indicate a modulation of the presentation of symptoms of ADHD by adulthood.[48–50] Conceptualisation of ADHD as a developmental disorder entails that, although the disabling feature of the disorder remains, both the quality and the severity of symptoms may change over time. Thus, applying the diagnostic criteria created for children may not be appropriate in adulthood. The developmental nature of the disorder also means that although new cases do not emerge in adulthood, there might be a certain number of children who 'outgrow' the disorder. This concept predicts reduced prevalence in adulthood because of the nature of the disorder. In view of our finding of a significant age–gender interaction, this concept might be mainly true for male ADHD cases with more hyperactive symptoms and linked disruptive behavioural problems than female ADHD cases in general.

Several studies reported that symptoms of ADHD declined with age.[25,28,31,32,34,51] At the same time, functional impairment and low socio-economic outcome can be detected even with a reduced number of symptoms[19,23,31,41,48] These observations lead us to another possible conclusion, that some children with ADHD do not outgrow the disorder but 'outgrow the diagnostic criteria',[1] meaning that reduced prevalence among adults results from an underestimation of the true prevalence of adult ADHD. Our finding that prevalence increases with age when women are predominantly represented in the sample might relate to the previously mentioned possibility of 'pseudo-new' cases of ADHD, when women with this disorder who were not referred for treatment in childhood owing to the absence of disruptive behavioural problems refer themselves in adulthood because of emerging comorbid psychiatric disease.

Two other factors concerning the diagnosis of adult ADHD should be mentioned, since either of them may result in underestimation of the prevalence of the disorder. First, based on the finding of the Milwaukee study,[1] – relevant also to clinical experience – it seems that the source of information might have a great impact on diagnosing ADHD: the persistence of ADHD was five to nine times higher when based on parent's report than when based on self-report, and parent's reports also showed higher potential to predict functional impairment than did self-report.[1] The second factor is the problem of symptom recall. Several authors pointed out that collecting data with retrospective self-report would underestimate the prevalence of adult ADHD, since adults do not remember their childhood symptoms properly. Empirical findings are inconsistent concerning this issue.[1,52–60] In the Milwaukee follow-up study, at the adult follow-up only 47% of the participants recalled having ADHD in childhood from the original ADHD index group.[1] Their self-report showed only 20% concordance with their parents' report concerning their childhood symptoms.[1] Manuzza et al on the other hand, in the results of the New York follow-up study, reported good symptom

recall (the sensitivity of retrospective diagnosis of ADHD was 0.78 and the specificity was 0.89) based on self-reports in the index group at the adult follow-up.[52] These authors noted that this might result from the fact that participants in the index group were from a clinically referred sample. Moreover, they suggested that adults who were not hospitalised in their childhood might have had poorer symptom recall.[52] The fact that in the New York study there was a high rate of false positive cases in the control group, according to Manuzza et al,[52] raises the possibility of problematic symptom recall among people who do not have ADHD.

In summary, we think that our finding is consistent with the suggestion that the prevalence of ADHD declines with age; however, the background of this phenomenon remains unclear and a caveat is needed in this regard. Specifically, the validity of DSM–IV diagnostic criteria for diagnosing adult ADHD is an important issue, emerging both from the interpretation of our findings and also from the relevant literature. It seems that diagnosing adult ADHD on the basis of strict DSM–IV criteria – as well as the above-mentioned methodological difficulties – may lead to underestimation of the prevalence of the disorder in this age group. Thus, further investigations are necessary to find out in what proportion methodological questions or natural developmental features are responsible for the observed decline in the prevalence of ADHD with age. Future well-designed, community-based epidemiological studies critically depend on an improved understanding of the aetiology and pathophysiology of the disorder, which in turn would help to improve the current diagnostic criteria and would thereby facilitate more reliable identification of people with ADHD. We must note that the small number of studies included in the meta-regression analysis and the above-detailed methodological difficulties of the reviewed and analysed studies are also potential limitations of our findings.

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References

1 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**: 279–89.

2 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**: 1424–31.

3 Yan WW. An investigation of adult outcome of hyperactive children in Shanghai. *Psychiatry Clin Neurosci* 1998; **52 (**Suppl): s303–5.

4 Gittelman R, Mannuzza S. Hyperactive boys almost grown up. I. Psychiatric status. *Arch Gen Psychiatry* 1985; **42**: 937–47.

5 Mannuzza S, Klein RG, Bonagura N, Malloy P, Giampino TL, Addali KA. Hyperactive boys almost grown up. V. Replication of psychiatric status. *Arch Gen Psychiatry* 1991; **48**: 77–83.

6 Mannuzza S, Klein RG, Addali KA. Young adult mental status of hyperactive boys and their brothers: a prospective follow-up study. *J Am Acad Child Adolesc Psychiatry* 1991; **30**: 743–51.

7 Mannuzza S, Klein RG, Bessler A, Malloy P, LaPadula M. Adult psychiatric status of hyperactive boys almost grown up. *Am J Psychiatry* 1998; **155**: 494–8.

8 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 Child Psychol Psychiatry* 1985; **24**: 211–20.

9 Smalley S, McGough JJ, Del'Homme M, NewDelman J, Gordon E, Kim T. Familial clustering of symptoms and disruptive behaviors in multiplex families with attention-deficit/hyperactivity disorder. *J Am Acad Child Adolesc Psychiatry* 2000; **39**: 1135–43.

10 Faraone SV, Doyle AE. The nature and heritability of attention-deficit/hyperactivity disorder. *Child Adolesc Psychiatr Clin N Am* 2001; **10**: 299–316.

11 Castellanos FX, Lee PP, Sharp W, Jeffries NO, Greenstein DK, Clasen LS, Blumenthal JD, James RS, Ebens CL, Walter JM, Zijdenbos A, Evans AC, Giedd JN, Rapoport JL. Developmental trajectories of brain volume abnormalities in children and adolescents with attention-deficit/hyperactivity disorder. *JAMA* 2002; **288**: 1740–8.

12 Roth RM, Saykin AJ. Executive dysfunction in attention-deficit/hyperactivity disorder: cognitive and neuroimaging findings. *Psychiatr Clin North Am* 2004; **27**: 83–96.

13 Hesslinger B, Tebartz van Elst L, Thiel T, Haegele K, Henning J, Ebert D. Frontoorbital volume reductions in adult patients with attention deficit hyperactivity disorder. *Neurosci Lett* 2002; **328**: 319–21.

14 Schweitzer JB, Faber TL, Grafton ST, Tune LE, Hoffmann JM, Kilts CD. Alterations in the functional anatomy of working memory in adult attention deficit/hyperactivity disorder. *Am J Psychiatry* 2000; **157**: 278–80.

15 Barkley RA. Behavioral inhibition, sustained attention, and executive functions: constructive theory of ADHD. *Psychol Bull* 1997; **121**: 65–94.

16 Seidman LJ, Biederman J, Webwe W, Hatch M, Faraone SV. Neuropsychological function in adults with attention deficit hyperactivity disorder. *Biol Psychiatry* 1998; **44**: 260–8.

17 Wilens T, Faraone SV, Biederman J. Attention-deficit/hyperactivity disorder in adults. *JAMA* 2004; **292**: 619–23.

18 Biederman J, Faraone SV, Spencer T, Wilens T, Norman D, Lapey KA, Mick E, Lehman BK, Doyle A. Patterns of psychiatric comorbidity, cognition, psychosocial functioning in adults with attention deficit hyperactivity disorder. *Am J Psychiatry* 1993; **150**: 1792–8.

19 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**: 817–27.

20 Kessler RC, Adler L, Ames M, Barkley RA, Birnbaum H, Greenberg P, Johnston JA, Spencer T, Üstün TB. The prevalence and effects of adult attention deficit/hyperactivity disorder on work performance in a nationally representative sample of workers. *J Occup Environ Med* 2005; **47**: 565–72.

21 Babinski LM, Hartsough CS, Lambert NM. Childhood conduct problems, hyperactivity-impulsivity, and inattention as predictors of adult criminal activity. *J Child Psychol Psychiatry* 1999; **40**: 347–55.

22 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**: 565–76.

23 Biederman J, Faraone SV, Spencer TJ, Mick E, Monteaux MC, Aleardi M. Functional impairments in adults with self-reports of diagnosed ADHD: a controlled study of 1001 adults in the community. *J Clin Psychiatry* 2006; **67**: 524–40.

24 Barbaresi W, Katusic S, Colligan R, Weaver A, Pankratz V, Mrazek D, Jacobsen S. How common is attention-deficit/hyperactivity disorder? Towards resolution of the controversy: results from a population-based study. *Acta Paediatr Suppl* 2004; **93**: 55–9.

25 Murphy K, Barkley RA. Prevalence of DSM–IV symptoms of ADHD in adult licensed drivers: implication for clinical diagnosis. *J Atten Disord* 1996; **1**: 147–61.

26 Weyandt LL, Linterman I, Rice JA. Reported prevalence of attentional difficulties in a general sample of college students. *J Psychopathol Behav Assess* 1995; **17**: 293–304.

27 DuPaul GJ, Schaughency EA, Weyandt LL, Tripp G, Kiesner J, Ota K, Stanish H. Self-report of ADHD symptoms in university students: cross-gender and cross-national prevalence. *J Learn Disabil* 2001; **34**: 370–9.

28 Heiligenstein E, Conyers LM, Berns AR, Smith MA. Preliminary normative data on DSM–IV attention deficit hyperactivity disorder in college students. *J Am Coll Health* 1998; **46**: 185–8.

29 Almeida Montes LG, Hernandez Garcia AO, Ricardo-Garcell J. ADHD prevalence in adult outpatients with nonpsychotic psychiatric illnesses. *J Atten Disord* 2007; **11**: 150–6.

30 Gadow KD, Sprafkin J, Schneider J, Nolan EE, Schwartz J. ODD, ADHD, versus ODD+ADHD in clinic and community adults. *J Atten Disord* 2007; **11**: 374–83.

31 Fayyad J, De Graaf R, Kessler RC, Alonso J, Angermeyer M, Demyttenaere K, De Girolamo G, Haro JM, Karam EG, Lara C, Lepine JP, Ormel J, Posada-Villa J, Zaslavsky AM, Jin R. Cross-national prevalence and correlates of adult attention-deficit hyperactivity disorder. *Br J Psychiatry* 2007; **190**: 402–9.

32 Kessler RC, Adler L, Barkley R, Biederman J, Conners CK, Demler O, Faraone SV, Greenhill LL, Howes MJ, Secnik K, Spencer T, Üstün TB, Walters EE, Zaslavsky AM. The prevalence and correlates of adult ADHD in the United States: results from the National Comorbidity Survey Replication. *Am J Psychiatry* 2006; **163**: 716–23.

33 Medina-Mora ME, Borges G, Lara C, Benjet C, Blanco J, Fleiz C, Villatoro J, Rojas E, Zambrano J. Prevalence, service use, and demographic correlates of 12-month DSM–IV psychiatric disorders in Mexico: results from the Mexican National Comorbidity Survey. *Psychol Med* 2005; **35**: 1773–83.

34 Faraone SV, Biederman J. What is the prevalence of adult ADHD? Results of a population screen of 966 adults. *J Atten Disord* 2005; **9**: 384–91.

35 American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (4th edn) (DSM–IV)*. American Psychiatric Association, 1994.

36 van Houwelingen HC, Arends LR, Stijnen T. Advanced methods in meta-analysis: multivariate approach and meta-regression. *Statist Med* 2002; **21**: 589–624.

37 DerSimonian R, Laird N. Meta-analysis in clinical trials. *Control Clin Trials* 1986; **7**: 177–88.

38 Hardy RJ, Thompson SG. A likelihood approach to meta-analysis with random effects. *Stat Med* 1996; **15**: 619–29.

39 Kessler RC, Merikangas KR. The National Comorbidity Survey Replication (NCS–R): design and field procedures. *Int J Methods Psychiatr Res* 1998; **13**: 60–8.

40 Demyttenaere K, Bruffaerts R, Posada-Villa J, Gasquet I, Kovess V, Lepine JP, Angermeyer MC, Bernert S, de Girolamo G, Morosini P, Polidori G, Kikkawa T, Kawakami N, Ono Y, Takeshima T, Uda H, Karam EG, Fayyad JA, Karam AN, Mneimneh ZN, Medina-Mora ME, Borges G, Lara C, de Graaf R, Ormel J, Gureye O, Shen Y, Huang Y, Zhang M, Alonso J, Haro JM, Vilagut G, Bromet EJ, Gluzman S, Webb C, Kessler RC, Merikangas KR, Anthony JC, Von Korff MR, Wang PS, Brugha TS, Aguilar-Gaxiola S, Lee S, Heeringa S, Pennel BE, Zaslavsky AM, Üstün TB, Chatterji S, WHO World Mental Health Survey Consortium. Prevalence, severity, and unmet need for treatment of mental disorders in the World Health Organization World Mental Health surveys. *JAMA* 2004; **291**: 2581–90.

41 Polanczyk G, De Lima MS, Horta BL, Biederman J, Rohde LA. The worldwide prevalence of ADHD: a systematic review and metaregression analysis. *Am J Psychiatry* 2007; **164**: 942–8.

42 Biederman J, Kwon A, Aleardi M, Chouinard VA, Marino T, Cole H, Mick E, Faraone SV. Absence of gender effects on attention deficit hyperactivity disorder: findings in nonreferred subjects. *Am J Psychiatry* 2005; **162**: 1083–9.

43 Biederman J, Faraone SV, Monteaux MC, Bober M, Cadogen E. Gender effects on attention-deficit/hyperactivity disorder in adults, revisited. *Biol Psychiatry* 2004; **55**: 692–700.

44 Gaub M, Carlson CL. Gender differences in ADHD: a meta-analysis and critical review. *J Am Acad Child Adolesc Psychiatry* 1997; **36**: 1036–45.

45 Biederman J, Mick E, Faraone SV, Braaten E, Doyle A, Wilens TE, Spencer T, Frazier E, Johnson MA. Influence of gender on attention deficit hyperactivity disorder in children referred to a psychiatry clinic. *Am J Psychiatry* 2002; **59**: 36–42.

46 Arnold L. Sex differences in ADHD: conference summary. *J Abnorm Child Psychol* 1999; **38**: 555–69.

47 Gershon J. A meta-analytic review of gender differences in ADHD. *J Atten Disord* 2002; **5**: 143–54.

48 Wender PH, Wolf LE, Wasserstein J. Adults with ADHD: an overview. *Ann N Y Acad Sci* 2001; **931**: 1–16.

49 Weiss M, Weiss J. A guide to the treatment of adults with ADHD. *J Clin Psychiatry* 2004; **65** (suppl 3): 27–37.

50 Biederman J, Mick E, Faraone SV. Age-dependent decline of symptoms of attention deficit hyperactivity disorder: impact of remission definition and symptom type. *Am J Psychiatry* 2000; **157**: 816–8.

51 Hill JC, Schroener EP. Age-dependent decline of attention deficit hyperactivity disorder. *Am J Psychiatry* 1996; **153**: 1143–6.

52 Mannuzza S, Klein RG, Klein DF, Bessler A, Shrout P. Accuracy of adult recall of childhood attention deficit hyperactivity disorder. *Am J Psychiatry* 2002; **159**: 1882–8.

53 Ericsson KA, Simon HA. Verbal reports as data. *Psychol Rev* 1980; **87**: 215–51.

54 Nisbett RE, Wilson TD. Telling more than we know. *Psychol Rev* 1977; **74**: 231–59.

55 Robins LN. Epidemiology: reflections on testing the validity of psychiatric interviews. *Arch Gen Psychiatry* 1985; **42**: 918–24.

56 Schaffer D. Attention deficit hyperactivity disorder in adults. *Am J Psychiatry* 1994; **151**: 633–8.

57 Wolkind S, Coleman EZ. Adult psychiatric disorder and childhood experiences: the validity of retrospective data. *Br J Psychiatry* 1983; **143**: 188–91.

58 Yarrow MR, Campbell JD, Burton RV. Recollections of childhood. *Monogr Soc Res Child Dev* 1970; **35**: 1–83.

59 Henry B, Moffitt TE, Caspi A, Langley J, Silva PA. On the 'remembrance of things past'. *Psychol Assess* 1994; **6**: 92–101.

60 Holmshaw J, Simonoff E. Retrospective recall of childhood psychopathology. *Int J Methods Psychiatric Res* 1996; **6**: 79–88.

Psychiatrists in 19th-century fiction

Hard Cash (1863), Charles Reade

Fiona Subotsky

Hard Cash is a polemical novel about the injustice and poor treatment of the insane and allegedly insane. While it made the author, Charles Reade (1814–1884), quite wealthy, Dickens, as the overall series editor, disclaimed responsibility for its opinions, which he presumably thought were too forcibly expressed. Reade had legal, and perhaps some medical, training. He did copious research, and points out in his preface that: 'I have accumulated during the last few years a large collection of letters from persons deranged in various degrees, and studied them minutely, more minutely than most Psychologicals study anything but Pounds, Shillings, and Verbiage.' The plot is complex, and the hero Alfred is forced into several different asylums, with contrasting regimes and doctors. I shall concentrate here on Dr Wycherley, assumed for reasons which will become evident to be at least in part a portrait of Dr John Conolly.

Assessment

Alfred Hardie, the lovelorn hero, has accused his father of misappropriating £14 000, thus driving his intended future father-in-law to penury and madness. As he appears pale and miserable to his sister, the family doctor is called in; he diagnoses 'hyperaesthetic character' and then brings along a specialist, Dr Wycherley. The latter is described as 'so saturated . . . with circumlocution, that . . . he talked like an Article.' Indeed, before even seeing the young man the 'psycho-cerebral' announces that: 'from the diagnostics, I have no doubt whatever he is labouring under the first fore-shadowings of cerebro-psychical perturbation. To speak plainly, the symptoms are characteristic of the initiatory stage of the germination of a morbid state of the phenomena of intelligence.'

Pressed to be more specific, Dr Wycherley warns that 'it is the premonitory stage of the precursory condition of an organic affection of the brain,' although at this stage eminently curable. He outlines the characteristic symptoms of the 'Incubation of Insanity': first 'Kephalalgia' or headache; second a 'morbid affection of sleep'; third, low spirits, often with a 'latent delusion', carefully concealed. Excitability is also frequent. Alfred's caring sister thinks her brother displays all these symptoms, and though her father is more sceptical, he begins to spot an advantage to himself.

Wycherley sums up thus, in a clearly self-serving manner: 'The most advisable course is to give him the benefit of the personal superintendence of some skilful physician possessed of means and appliances of every sort for soothing and restraining the specific malady.'

He departs with the following peroration:

'it is not logical, reasoning *a priori*, to assume the possibility that the studious or other mental habits of a Kephalalgic, and gifted youth, can be reversed, and erotic monomania germinate, with all the morbid phenomena of isolation, dejection of the spirits, and abnormal exaltation of the powers of wit and ratiocination, without some considerable impairment, derangement, disturbance, or modification, of the psychical, motorial, and sensorial functions of the great cerebral ganglion. But it would be equally absurd to presuppose that these several functions can be disarranged for months, without more or less disorganisation of the medullary, or even of the cineritious, matter of the encephalon. Therefore – dissection of your talented son would doubtless reveal at this moment either steatonatous or atheromatous deposits in the cerebral blood-vessels, or an encysted abscess, probably of no very recent origin, or, at the least, considerable inspissation, and opacity, of the membranes of the encephalon, or more or less pulpy disorganisation of one or other of the hemispheres of the brain: good morning!!'

Subsequently Dr Wycherley and a compliant apothecary see Alfred and quiz him about his belief that his father has misappropriated money. Calling this a delusion, and his reaction 'excitement', they find grounds for his compulsory confinement to a lunatic asylum, which he is tricked into entering.

As to Conolly, his lectures were said by his son-in-law Henry Maudsley, in an intermittently unkind obituary, to have been 'diffuse and theoretical', 'vague and discursive', but to us both Reade and Maudsley seem at least as polysyllabic.

Moral treatment

Conolly is famous for popularising 'non-restraint' methods of caring for the mad – a system Alfred finds in place at the second asylum he is admitted to, run by Dr Wycherley. He is allowed to bathe and is examined by the assistant physician. His lesions from previous brutal treatment are noted, but no enquiries are made into his mind – 'indeed (the doctor) was little qualified for researches of the kind'. He has breakfast with 'a number of mad ladies and gentlemen, who by firmness, kindness, and routine, had been led into excellent habits; the linen was clean and the food good.' However, Alfred finds that this system is unfortunately accompanied by extra means to prevent escape, such as a higher number of attendants, and windows which do not open fully and have iron frames painted to look like wood.

Was Hamlet mad?

Alfred begins to realise that his attempts to get himself released might be long drawn-out and that he should perhaps both show less rage and also occupy himself by studying for his degree. Strategically, he becomes Wycherley's pet patient and receives useful tutelage for his Oxford exams, but they quarrel over whether Hamlet was mad or not. Alfred realises that, ironically, he must give in to Wycherley's 'monomania' on the subject in order to gain recognition of his own sanity.

' ''Doctor,'' said he, ''I have been thinking over your arguments, and I capitulate. If Hamlet ever existed, he was as mad as a March hare.'' And he blushed at this.' And so Dr Wycherley becomes convinced that Alfred is of sound mind.

A review of Conolly's 1863 *A Study of Hamlet* can be read in this *Journal*'s predecessor. Conolly did indeed hold that Hamlet had 'a temperament in which madness lies very near the surface, and which some violent shock . . . is certain to develop into disease', and that he was not feigning, but manifesting, madness. Maudsley remarks in his obituary that Conolly's essay, while elegant, reveals 'the extent of his insight and the depth of his philosophy'. However, Maudsley has just characteristically commented that 'the philosophical depths of mental phenomena he never cared to sound', thus clarifying his view.