

Attention-deficit hyperactivity disorder in adults: validity unknown¹

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Abstract Attention-deficit hyperactivity disorder (ADHD) is a commonly diagnosed childhood psychiatric disorder. Debate over its diagnostic validity, aetiology, presentation and treatment has extended from the clinical to the public domain. As children with ADHD diagnoses reach adulthood there is increasing interest in 'adult ADHD'. Cohorts followed up show poorer outcomes as adults than do controls. Self-referred adults, sometimes relatives of children with ADHD, are also of interest regarding adult ADHD. Innovative work is being done examining issues of aetiology, treatment, outcomes and comorbidity in these groups, but heterogeneity among those diagnosed with ADHD and changes in classification systems and diagnostic criteria over time complicate comparison of research findings. The diagnostic validity of adult ADHD remains uncertain and needs further study.

This article is the first in a news series entitled 'life-span psychiatry', which considers the developmental psychiatry of adulthood. Subsequent articles will discuss autistic-spectrum disorders, early developmental aspects of psychopathy and psychosis and training implications.

The diagnosis of attention-deficit hyperactivity disorder (ADHD) in children has attracted much argument and controversy. Polarised opinion ranges from those who believe it is a myth and does not exist to those who argue that there is both genetic and physiological evidence of its existence (Jadad, 1999).

Similar controversy is expected for the concept of adult ADHD, as the debate will certainly grow with the increasing research, public awareness and interest surrounding the disorder.

Public fascination with ADHD has grown since the late 1980s (Weiss *et al*, 2002) and the internet has further increased the availability of information, often unevaluated, and led to the establishment of many ADHD support groups (e.g. the Attention Deficit Disorder Information and Support Service in the UK; website <http://www.addiss.co.uk/>). In March 2003 an internet search using the term ADHD and the Google search engine revealed 80 pages of links.

1. For an invited commentary on this article see Asherson (2004, this issue: pp. 257–259). For a related editorial see Green (2004, this issue: pp. 241–242).

As children with a diagnosis of ADHD grow into adulthood, adult mental health services will increasingly be faced with responsibility for their care. This demand has already been identified in the UK and has led to the establishment of a specialised service (Toone *et al*, 1997; Young & Toone, 2000).

We both work with children and adolescents diagnosed with ADHD and form impressions of them and their families. This in turn influences our ideas and beliefs. We have read and appraised a wide range of work, but in preparing this article we did not use a systematic, transparent methodology such as that outlined in the Cochrane Reviewer's Handbook (Clarke & Oxman, 2003). We offer a narrative rather than a systematic review, biased through our interest in the issues. Our aims are to highlight these and to encourage debate.

Evolution of the concept

The concept of ADHD has developed over time, and diagnostic criteria continue to evolve. In 1902, Still described children with hyperactivity and poor attention skills as having a 'defect of moral control' (Still, 1902). As early as 1937, Bradley wrote about the use of amphetamines in children to reduce hyperactivity (Bradley, 1937), and by the 1940s and 1950s the condition was attributed to 'minimal brain

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damage'. Later, it was renamed 'minimal brain dysfunction' because of the absence of evidence for actual brain lesions, and then as 'hyperkinetic impulse disorder' (Barkley, 1990).

In the 1960s, 'minimal brain dysfunction' was replaced by a variety of more differential concepts such as dyslexia, learning disability and hyperactivity, and by 1970 'hyperkinetic child syndrome' was used to describe impulsive children with short attention spans, distractibility and aggression (Barkley, 1990). DSM-II (American Psychiatric Association, 1968) described criteria for 'hyperkinetic reaction of childhood', and by 1980 the DSM-III 'attention-deficit disorder' was defined with two subtypes, with and without hyperactivity (American Psychiatric Association, 1980).

DSM-III was the first classification system to raise the possibility that symptoms might continue into adulthood as 'attention-deficit disorder (hyperactivity), residual state'. It also defined two subtypes: one focused on clinically significant problems with attention and the other on both inattention and hyperactivity-impulsivity.

The term 'attention-deficit hyperactivity disorder' first appeared in DSM-III-R (American Psychiatric Association, 1987) and its use has continued in the trend towards extension of the disorder to adults, for whom 'impairment in the workplace' is specifically mentioned. Despite this trend away from child-oriented symptom criteria, field trials for the current DSM-IV diagnostic criteria did not involve adults (Weiss *et al.*, 2002). DSM-IV defines three subtypes: predominantly inattentive, predominantly hyperactive-impulsive and combined (American Psychiatric Association, 1994).

In the UK and the rest of Europe, researchers have used additional diagnostic frameworks. The ICD-10 (World Health Organization, 1992) classification 'hyperkinetic disorder' is similar to DSM-IV's ADHD (combined type), but its criteria are even more restricted: 'a persistent and severe impairment of psychological development resulting from a high level of inattentive, restless and impulsive behaviour'. By definition, the onset of hyperkinetic disorder is before the age of 7 years, but it is frequently recognised in children less than 2 years old (Taylor *et al.*, 1998). All three problems (attention deficit, hyperactivity and impulsiveness) must be present, more stringent criteria exist for the 'pervasiveness' of the condition across situations, and symptoms of other disorders are absent, apart from conduct disorder, which may be present as part of the subtype 'hyperkinetic conduct disorder'.

In a series of papers on a community-based child population from 1983 to 2000, Swedish researchers have described a disorder called 'deficits in attention, motor control and perception' (Rasmussen & Gillberg, 2000). This is similar to ADHD combined with developmental coordination disorder.

Diagnostic validity

The core symptoms of ADHD – inattention, hyperactivity and impulsivity – are normal behavioural traits present in children without the disorder. The extent to which each causes disability varies and should be seen within the context of a child's developmental level. An active 3-year-old, impulsive and frequently interrupting of others, for example, differs from a disruptive, unfocused 8-year-old unable to cope educationally. Yet both are displaying core symptoms of ADHD (Zwi *et al.*, 2000).

Clinical judgements are therefore made as to whether these traits are present to a greater extent than 'normal'. Judgements are also made as to whether they impair the function of the individual. Not surprisingly, therefore, children with ADHD are heterogeneous in respect of their problem presentation and clinical needs (Klassen *et al.*, 1999). The absence of a validated diagnostic test to confirm a clinical diagnosis means that the diagnosis is related to the perceived degree of impairment due to the core symptoms and their pervasiveness in a range of situations. Field trials are therefore undertaken to validate diagnostic criteria; for DSM-IV ADHD these include the trials reported by Frick *et al.* (1994), Lahey *et al.* (1994) and Applegate *et al.* (1997).

The high interrater reliability between expert assessors in such trials increases the internal validity of the concept but may have little effect on external validity. For example, the subjective nature of judgements on the presence or absence of diagnostic criteria such as 'is often forgetful in daily activities', judgements that may be applied differently by researchers in different centres, could result in thresholds of diagnosis that differ considerably.

Questions of diagnostic validity inevitably are greater for disorders in which no validated biological marker exists. This is true not only of ADHD or other childhood disorders, but of health care in general. Many conditions have signs or symptoms (e.g. height, blood pressure, hyperactive-impulsive behaviour) represented by measures on a continuum. These signs and symptoms, be they physical, emotional or psychopathological, require us to consider dimensional and categorical approaches to classification together with issues linked to development from childhood to adulthood. Taylor & Rutter (2002) have explored these issues in depth.

Given the heterogeneity of clinical signs, symptoms, comorbidities and presentations, as well as the progression of evolving diagnostic systems, ADHD is possibly better conceptualised as a heterogeneous, complex neurodevelopmental constellation of problems rather than a single disorder (Zwi *et al.*, 2000).

Does ADHD exist?

Not surprisingly, critics have challenged fundamental issues relating to ADHD, including the validity of the 'disorder' construct itself. Timimi (2001a) argues that the cut-off between normal behaviour and ADHD is arbitrary, and questions who defines it and why. He also asks whether ADHD is a research-generated concept with little relation to the complexity found in clinical practice. He subsequently criticises authors who suggest that the use of psychostimulants is 'nothing less than a call to doctors to medicate children for social control purposes' (Timimi, 2001b).

Concern about 'periodic inaccurate portrayal' of ADHD in the media led a consortium of 74 internationally acclaimed ADHD researchers to publish a consensus statement in which they argue in favour of the validity of the diagnosis (Barkley *et al.*, 2002b). The intention of the statement was to demonstrate consensus among a large body of established clinical researchers regarding the current state of the evidence about ADHD as a genuine disorder (Barkley, 2002b) and the signatories cite support from the US Surgeon General, the American Medical Association, the American Academy of Child and Adolescent Psychiatry, the American Psychological Association and the American Academy of Pediatrics.

The statement describes ADHD as a syndrome characterised by deficiencies in a set of psychological abilities that pose serious harm to most of those who have it. At the core of the disorder are deficits in behavioural inhibition and sustained attention, leading to impaired social, educational and occupational functioning as well as difficulties with social rules, norms and laws, and increased risk of physical injury and accidental poisoning (Barkley *et al.*, 2002).

The document, however, is a statement of opinion rather than a scientific argument, and it quickly generated criticism (Jureidini, 2002). Regrettably, it falls short of the requirements of evidence-based practice, which strives to evaluate the strengths and weaknesses of any question through the use of transparent methodologies that reduce known and unknown bias. It argues that, as 'a matter of science, the notion that ADHD does not exist is simply wrong'. This may be correct, and the statement's thesis may be supported by the authors' original work. However, by presenting a narrative statement rather than a scientific argument with a transparent methodology, critical appraisal of the literature and detailed referencing of each point made, the scientific validity of this document is weakened and it belongs not to the domain of scientific argument but to that of expert opinion. This is essentially a presentational point – there is considerable evidence

supporting the existence of ADHD, even if it is not cited.

Prevalence and aetiology

DSM-IV gives prevalence estimates for ADHD of 3–5% in school-aged children, but studies in different centres and countries using earlier versions of the DSM give rates that vary from 1.7% to 16.1% (Jadam *et al.*, 1999).

Comorbid psychiatric disorders are common in children with ADHD. These include oppositional defiant disorder (35%), conduct disorder (28%), anxiety disorder (26%), depressive disorder (18%) and learning difficulties (12%) (Green *et al.*, 1999).

The aetiology of ADHD is unclear (Green, 1999). There is evidence that it has a genetic component, and environmental factors may also be implicated. Studies have shown that ADHD in childhood is highly heritable (0.8) (Taylor *et al.*, 1998), but inheritance is complex and likely to be the result of several genes acting together.

New genetic research techniques are being used that may well improve our understanding. Traditional molecular genetic techniques have been supplemented by quantitative trait loci (QTL) mapping, an approach that is useful when traits (such as hyperactivity) are continuously distributed in the population (Plomin, 1999).

Molecular genetic studies have shown associations between ADHD and dopamine receptor genes and the dopamine transporter DAT₁ (Asherson & Curran, 2001; Barr, 2001). An international study is currently under way in the IMAGE project (Asherson *et al.*, 2003). Hypotheses have also been presented about the roles of other neurotransmitters, including noradrenaline (Arnsten, 2000; Biederman & Spencer, 2000) and serotonin (Quist & Kennedy, 2001). Some evidence exists regarding cognitive deficits in ADHD that affect executive functioning and inhibitory control (Taylor *et al.*, 1998) and lead to altered perception of time and an extreme dislike of waiting (Sonuga-Barke *et al.*, 1994).

It is not clear how environmental and genetic factors interact in the aetiology of ADHD in childhood. The disorder may result in behaviour that is particularly challenging for parents and consequent aggressive parenting might contribute to a worsening of hyperactivity (Woodward *et al.*, 1998).

Treatment

Concern has been raised about possible over-prescription of stimulants to children (Zito *et al.*, 2000) and initially this may have deterred professionals, particularly in the UK and the rest of

Europe, from developing specialist services for children diagnosed with ADHD and similar disorders (Taylor *et al.*, 1998). In 1999, it was estimated that the use of stimulants was 30 times higher in the USA than in the UK (Taylor, 1999), but we believe that increasing information about ADHD is resulting in a rise in their prescription in the UK (Zwi *et al.*, 2000).

Systematic reviews of the use of stimulants in ADHD concluded that, on balance, there is evidence of their benefit, at least in the short term (Miller *et al.*, 1998; Jadad *et al.*, 1999; Lord & Paisley, 2000). However, Lord & Paisley thought the overall methodological quality of trials in the ADHD literature to be poor, with a consequent high probability of bias. In their meta-analysis of 62 methylphenidate trials, Schachter *et al.* (2001) were even more cautious, reporting a modest benefit from methylphenidate that was balanced against adverse effects and publication bias that might have skewed results.

The MTA

The Multimodal Treatment Study of Children with Attention-Deficit/Hyperactivity Disorder (MTA) was the largest, most rigorous randomised controlled trial to date, involving 579 children aged 7–9.9 years (MTA Cooperative Group, 1999). Despite methodological issues raised by some authors (Boyle & Jadad, 1999; Taylor, 1999), it is a trial that confirmed the effectiveness of medication management in children and adolescents.

The MTA also found, controversially, that intensive behavioural therapy involving the child, family and teachers added little to well-supervised medication management. Klassen *et al.* (1999) have suggested that this might be an artefact related to trial methodology. Behavioural interventions, for example, may be better at reducing ‘associated features’ of ADHD such as conflictual relationships or academic achievement, outcomes that may be difficult to measure. In contrast, stimulants may be most effective at reducing the core symptoms of ADHD (inattention, hyperactivity and impulsivity) and both the intervention and the outcome lend themselves well to highly controlled experimental studies.

The landmark cohort studies

Three cohort studies of children with diagnoses of ADHD (or earlier diagnostic terms used to describe what we now call ADHD) have followed individuals from childhood into adulthood (Weiss *et al.*, 1985; Mannuzza *et al.*, 1993; Rasmussen & Gillberg, 2000). They all report relatively low rates of ADHD

in the adult years compared with childhood and adolescence, but they also report that those who had had childhood ADHD (compared with controls) showed higher rates in adulthood of impaired educational and occupational outcomes, antisocial personality disorder, substance misuse and persistent social impairment.

It is difficult to compare these three cohort studies directly with one another because their selection criteria, drop-out rates and reported outcomes differ. This is further complicated by heterogeneity regarding clinical signs, symptoms, comorbidity and presentations, not to mention changes in diagnostic classification systems over time. However, we give a brief outline of each study below.

Weiss *et al.*'s Montreal study

The Montreal study (Weiss *et al.*, 1985) followed 63 hyperactive children and 41 controls for 15 years, until they were in their mid-20s. Diagnostic interviews were not blinded and the loss to follow-up over 15 years was 33.6%. DSM-III was not in use at the start of the study, so the authors attributed retrospective DSM-III diagnoses at publication. All of the children received diagnoses of ‘ADD(H)’ (attention-deficit disorder (hyperactivity)), and ‘the majority’ had an associated conduct disorder.

They reported on symptoms of ‘the hyperactive syndrome’ and found that 66% of hyperactive individuals (compared with 7% of the control group) complained of at least one disabling symptom. They did not report the rates of those with the full DSM-III diagnosis at the end of the 15-year period, which is surprising. Interestingly, the only DSM-III diagnosis that differed between the two groups was antisocial personality disorder, present in 23% of the hyperactive individuals and 2.4% of the controls.

Of the hyperactive individuals, 90% had received 10–20 therapeutic interviews and 10% had received family therapy during childhood and adolescence. None had been prescribed methylphenidate but four had taken dexamphetamine for 6 months or ‘on and off’ for 2 years; 20 had been treated with chlorpromazine for between 6 months and 2 years.

Mannuzza *et al.*'s New York study

Mannuzza *et al.*'s (1993) study in New York followed two cohorts of a total of 207 predominantly middle-class White boys of average IQ referred to a child psychiatric clinic, from childhood to their mid-20s. Conduct disorder was virtually absent from this group because those with aggression or antisocial behaviour were excluded. Assessors were blind to the person's diagnostic status. Loss to follow-up was 12% for the first cohort and 18% for the second

over a period of 15–21 years. About 85–90% of those with hyperactivity had been prescribed stimulant drugs in childhood. Some of these received stimulants only during childhood, whereas others continued to receive them for years (S. Mannuzza, personal communication, 2003).

In late adolescence, ADHD was present in 40% of the index cases and 3% of controls (Gittelman *et al.*, 1985), and 27% of the index group, v. 8% of the controls, had antisocial personality disorder. By the time the subjects had reached a mean age of 25, 'clinically impairing ADHD symptoms and syndromes' were present in 11% of the index group and in only 1% of the controls; in their second cohort only 4% of the index group and none of the controls had ADHD (Mannuzza *et al.*, 1993, 1998).

Rasmussen & Gillberg's Swedish study

Rasmussen & Gillberg's (2000) study in Sweden followed a community sample of 61 children with ADHD and comorbid developmental coordination disorder and 46 controls from childhood to adulthood. Diagnosis in childhood, as in the Montreal and New York studies, used a diagnostic system different from DSM-IV, so the authors scrutinised records and rediagnosed them according to DSM-IV. However, the index cases included children with motor and perceptual problems and the rediagnosed sample included 39 with ADHD and developmental coordination disorder, 11 with ADHD only and 5 with developmental coordination disorder only.

A unique aspect of this study is that none of the index cases had ever received stimulants. Follow-up extended over 15 years and loss to follow-up in both groups was 10%. Assessments of psychiatric status in adulthood were blinded.

In terms of 'current ADHD' they found that 49% of the index cases and 9% of the controls had 'marked symptoms of ADHD at age 22'. They also found that almost 60% of the children in the index group, compared with 13% in the control group, had 'a poor outcome', which included drug or alcohol misuse, living off a disability pension or welfare benefits, major personality disorder, chronic severe psychiatric disorder, autistic-spectrum disorder, and conviction for a criminal offence.

Extending the concept of ADHD to adulthood

It is not a simple matter to extend the concept of ADHD to adulthood. Should we think of it as a developmental disorder, showing continuity into adulthood (like, for example, autistic-spectrum disorders and conduct disorder, which in adulthood

is observed as antisocial behaviour and personality disorder), or as a psychiatric disorder (like depression)?

The diagnostic validity of ADHD poses more of a challenge in adults than it does in children, given the need for retrospective information, the extent of comorbidity with other disorders (Shaffer, 1994) and the fact that DSM-IV criteria have been validated only in children and adolescents, not in adults (Weiss *et al.*, 2002).

Wender, who described adult attention-deficit disorder as long ago as 1981, suggests that his Utah group has 'consistently found' that many adults with persistent ADHD symptoms do not report them (Wender, 2001).

Diagnostic interview tools that identify childhood symptoms may increase diagnostic validity in adult populations in which there is a high probability of childhood ADHD, as in groups referred to specialist ADHD clinics, but in the general population the picture may be different.

Mannuzza *et al.* (2002) report on long-term recall of childhood ADHD by adults who had been diagnosed in childhood and followed up. Interviewers blind to the childhood diagnosis used a semi-structured interview to ascertain whether adults reporting symptoms from childhood were able to provide sufficient information to confirm a retrospective ADHD diagnosis. In this clinic-referred population, the interviews achieved high sensitivity (0.78) and specificity (0.89) (Box 1) for adult recall of childhood ADHD symptoms. Thus, in a population in which the prevalence of true-positive cases should be much higher than in the general population anyway, the instrument used by Mannuzza *et al.* appears to be helpful. However,

Box 1 Sensitivity v. specificity

A diagnostic instrument with high sensitivity accurately identifies those *with* the disorder, who obtain a *positive* result using the instrument (the 'true positives').

The specificity is the proportion of people *without* the disorder that the instrument correctly identifies as *not* having it (i.e. who obtain a *negative* result). An instrument with low specificity gives a high number of 'false positives': people who do not have the disorder but whom the instrument identifies as having it.

These measures of an instrument's utility are interrelated and are also influenced by the disorder's prevalence, i.e. the total number of true-positive cases in the population.

when they recalculated the figures assuming a 5% prevalence of ADHD in the general population, the false-positive rate rose substantially, to 75%.

This highlights the danger of making retrospective diagnoses of childhood ADHD on the basis of self-reports in primary care settings and epidemiological surveys, where the prevalence of ADHD in the population is low.

It has also been argued that DSM-IV criteria are inappropriately worded for adults, again because they have been validated in children and adolescents (Murphy *et al.* 2002). Another issue is the natural history of ADHD. Symptoms and impairment may change as the individual develops, with the hyperactive-impulsive symptoms emerging initially in childhood (Loeber *et al.* 1992) before declining with age. Symptoms of inattention may emerge later (Applegate *et al.* 1997) and predominate with age, whereas hyperactivity may become less overt, appearing as restlessness and fidgetiness (Mannuzza *et al.* 1997; Biederman *et al.* 2000; Murphy *et al.* 2002; Weiss *et al.* 2002).

Some argue, therefore, that DSM-IV diagnostic criteria are too stringent when applied to adults, and point to the finding that in longitudinal studies as many as 66% continue to report the presence of at least one ADHD symptom severe enough to cause impairment (Murphy *et al.* 1996; Weiss *et al.* 2002). For example, the DSM-IV diagnostic criteria include the presence of symptoms before the age of 7 years, reflecting the developmental nature of the disorder. This early onset may be difficult to ascertain retrospectively in the absence of a clearly documented history of childhood ADHD, and Murphy *et al.* (2002) argue that it is unjustifiable to maintain this threshold, especially since DSM-IV field trials have shown that it significantly diminishes the reliability of the diagnosis (Applegate *et al.* 1997).

If the diagnostic criteria are too restrictive when applied to adults, it may lead to underdiagnosis (Millstein *et al.* 1997; Weiss *et al.* 2002; Murphy & Barkley, 2002). This could, of course, be argued the other way too and, in the absence of further validation studies, simply modifying the diagnostic criteria might lead to higher rates of false-positive diagnosis.

Another issue of importance is the difference between adults diagnosed with ADHD in childhood and adults who present with suspected ADHD with no confirmed childhood ADHD diagnosis. Children with ADHD are usually taken to clinics by parents, whereas adults who attend clinics are usually self-referred. Thus, factors associated with self-referral, such as educational and socio-economic status, may influence the composition of this population (Murphy & Barkley, 1996).

Patterns of comorbidity appear to be different in these two groups. Murphy & Barkley (1996) report that clinic-referred adults were more likely to have comorbid anxiety disorders (50% where affected), whereas hyperactive children followed to adulthood were more commonly found to have conduct, substance misuse and antisocial personality disorders.

Faraone (2000) suggests that we should view the diagnosis of ADHD as we do the construct of IQ, using 'different test batteries for different age groups and within a single battery [considering] a score high or low in reference to people of the same age'. He acknowledges, however, that age may be an imperfect proxy for development and suggests that the individual's developmental stage at the time of the diagnosis might be used instead.

He also highlights that changes in diagnostic criteria from DSM-III onwards have introduced a hierarchical approach to diagnosis rather than a 'comorbidity paradigm', thereby excluding some diagnoses in the presence of others. For example, where ADHD and depression are both present, the primary diagnosis would be that of ADHD. Depending on how these rules were applied, comorbidity and prevalence information might be lost. He asserts that a clash of theoretical paradigms is an important aspect of the debate over validity of adult ADHD. Researchers using hierarchical diagnoses would find fewer cases than those using a comorbidity paradigm, and clinicians using developmentally sensitive diagnoses would 'find adult ADHD where others do not'.

Treatment of adult ADHD

Although it might be argued that 'the weight of the available literature' shows that adult ADHD can be diagnosed reliably through historical self-reports of childhood symptoms (Spencer *et al.* 1995), it has also been shown that, to avoid false-positive diagnoses, it is vital to obtain contemporaneous data to substantiate retrospective diagnoses (Mannuzza *et al.* 2002).

As mentioned above, high interrater reliability between expert assessors may increase the internal validity of trials. Until questions regarding the validity of adult ADHD are settled through further validation studies, however, the external validity of these trials remains uncertain and the generalisation of results to others with 'adult ADHD' should be made with caution.

We will therefore limit our comments regarding treatment interventions to saying that (at the time of writing) there is limited evidence from a number of small trials (the largest involved only 41

participants) to suggest that methylphenidate (Spencer *et al*, 1995; Dorrego *et al*, 2002), mixed amphetamine salts (Spencer *et al*, 2001), bupropion (Wilens *et al*, 2001), desipramine (Wilens *et al*, 1996), lithium (Dorrego *et al*, 2002), modafinil (Taylor & Russo, 2000) and pemoline (Wilens *et al*, 1999) may be beneficial in adult ADHD. The selective inhibitor of the noradrenaline transporter, atomoxetine, showed promise in two larger trials (Michelson *et al*, 2003).

Future research

Much still needs to be understood about ADHD, particularly in adulthood. Its core symptoms of inattention, hyperactivity and impulsivity, as well as other ADHD-related behaviours, personality traits and disabilities, need thorough developmental observation and study across the life span. Further investigation is also needed on the role and interaction of genetic and environmental factors in the aetiology and course of ADHD throughout life.

High-quality diagnostic validity studies are still necessary and large, high-quality randomised controlled trials are needed in adult ADHD. It is also vital that transparent methodologies are used and reported in research publications and that journal editorial boards adopt protocols such as the revised CONSORT statement on the reporting of randomised controlled trials (Moher *et al*, 2001).

Along with primary research, secondary research in the form of transparent systematic reviews and meta-analyses of the literature are vital to answer many contentious questions that arise in this field.

Conclusions

It is likely that professionals in adult mental health will increasingly receive referrals about adults who wish to be assessed and treated for ADHD. Some of these will be the young people who have been diagnosed and treated for ADHD in childhood and adolescence. Others may be parents of those with ADHD or people who wonder whether their problems might arise from 'undiagnosed ADHD'.

The imaginative work that went into the landmark cohort studies has been invaluable, despite their limitations. We now know that many children with hyperactivity go on to have long-term problems and poorer outcomes than those without it.

What is uncertain, however, is whether the same group of children entering cohort studies 25 years ago with diagnoses such as hyperactivity, minimal brain dysfunction or motor perception dysfunction would have been entered into these studies if current

criteria were applied to their selection. Although this may be likely, we cannot be sure that we are talking about the same condition when diagnostic criteria have changed. We cannot assume with certainty that the cohorts assembled then are the same as those we now identify as having ADHD. This is further complicated by the heterogeneity within ADHD in terms of the range of presentations and comorbid problems.

With so many more children being diagnosed and treated today, what we do not know is whether early intervention and treatment over a longer period will prevent the poor outcome seen in the landmark studies. The need for further, well-designed cohort studies is as important today as it was 30 years ago.

Finally, we would like to echo the words of Faraone regarding adult ADHD, that 'research should focus not only on the validity of the disorder, but also on the validity of the theories that buttress the diagnosis' (Faraone, 2000). There may be stronger grounds for the diagnostic validity of ADHD in children and adolescents, albeit with some reservations, than in adults. There should, however, still be considerable debate and research regarding the diagnostic validity of both.

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- a heterogeneity of clinical signs, symptoms, comorbidity and presentation, and issues of diagnostic validity
- c field trials validating some DSM–IV diagnostic criteria for ADHD involved only children and adolescents
- d retrospective evaluation of childhood symptoms poses significant methodological challenges to the diagnostic validity of adult ADHD.
- 2 Cohort trials of children with hyperactivity:**
- a have demonstrated that, in general, hyperactive children show better outcomes than controls in adulthood, which is explained by their entrepreneurial success
- b are said to show lower than expected rates of ADHD in adulthood by those who believe DSM–IV criteria to be insufficiently sensitive to the condition
- c are not easily compared because selection criteria, numbers lost to follow up and reported outcomes differ between studies
- d have shown poorer outcomes in children with hyperactivity than in controls.
- 3 The use of stimulants in children and adolescents with ADHD:**
- a has been shown to reduce hyperactivity–impulsivity and inattention in the short term
- b has been the subject of a number of high-quality systematic reviews
- c has shown a downward trend in prescriptions in recent times
- d has been shown to prevent negative outcomes in adulthood.
- 4 Randomised controlled trials in ADHD patients:**
- a have not been done using stimulants in children, because of ethical considerations
- b is not the appropriate study methodology for testing medication interventions
- c are no longer necessary since the D₄ gene has been shown to be pathognomonic of the disorder
- d have been evaluated in major systematic reviews by Miller *et al*, Jadad *et al* and Lord & Paisley.
- 5 Psychiatric comorbidity in ADHD:**
- a is high in children, with about 60% showing comorbid disorders
- b is the exception rather than the rule because it occurs infrequently
- c may be affected by assessors' use of hierarchical diagnostic systems
- d appears far less frequently than expected (at about 5%) in children with this complex neurodevelopmental disorder.

Multiple choice questions

1 Questions about the validity of adult ADHD are still relevant because:

- a field trials in 18- to 35-year-olds that demonstrate the validity of DSM–IV diagnostic criteria for ADHD require replication
- b results of existing small RCTs in adult ADHD cannot be generalised to other clinical populations, owing to

MCQ answers

1	2	3	4	5
a F	a F	a T	a F	a T
b T	b T	b T	b F	b F
c T	c T	c F	c F	c T
d T	d T	d F	d T	d F