Attention-deficit hyperactivity disorder in adults: validity unknown

Morris Zwi & Ann York

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.

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.

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

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 a set of psychological abilities linked to attention, planning, organisation, working memory, and the capacity to perform daily tasks. These core deficits result in social, educational and occupational functioning to impairments that affect executive functioning and control (Taylor et al, 1998) and lead to altered perception of time and an extreme dislike of waiting (Sonuga-Barke et al, 1994).

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 DAT, (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).

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% (Jadad 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.

Prevalence and aetiology

Support for the existence of ADHD is widely held, even if it is not cited.

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 et al., 1995; MTA Cooperative Group, 1999), it is a trial that was balanced against adverse effects and publication bias that might have skewed results.

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 inattentiveness, 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.

**References**


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 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.

<table>
<thead>
<tr>
<th>MCQ answers</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>a F</td>
<td>a F</td>
<td>a T</td>
<td>a F</td>
<td>a T</td>
<td></td>
</tr>
<tr>
<td>b T</td>
<td>b T</td>
<td>b T</td>
<td>b F</td>
<td>b F</td>
<td></td>
</tr>
<tr>
<td>c T</td>
<td>c T</td>
<td>c F</td>
<td>c F</td>
<td>c T</td>
<td></td>
</tr>
<tr>
<td>d T</td>
<td>d T</td>
<td>d F</td>
<td>d T</td>
<td>d F</td>
<td></td>
</tr>
</tbody>
</table>
Bridging the service divide

Invited commentary on... Attention-deficit hyperactivity disorder in adults

Philip P. Asherson

Most child and adolescent mental health services recognise the existence of, and need for treatment in, attention-deficit hyperactivity disorder (ADHD). Many specialist multidisciplinary ADHD clinics have been developed in recent years, and many paediatricians have included the treatment of ADHD as an important part of their clinical activity. A good deal of the justification for this increase in therapeutic activity has been the demonstration that ADHD is indeed a predictor of adult mental health problems. General adult psychiatry, however, has not followed suit in identifying and treating substantial numbers of affected people. It is likely none the less that an increasing load in adult psychiatry will develop. A rising number of young people will enter adult life still receiving stimulant medication or other treatment for ADHD, and adult psychiatrists are likely to be consulted. Furthermore, an increasing number of adults are likely to recognise themselves as having been disabled by ADHD and therefore to seek assistance. In many cases, individuals with adult ADHD who require specific treatment for the condition will have been treated unsuccessfully for disorders with overlapping symptom profiles such as anxiety, depression, bipolar disorder and antisocial personality disorder.

In support of adult ADHD?

Zwi & York (2004, this issue) present some of the available evidence supporting adult ADHD as a valid diagnostic entity. Their article shows that there remain many unanswered questions on the suitability of current definitions for adult ADHD, the size of the potential problem and long-term outcome for patients with the disorder. While further research is indicated for many common psychiatric conditions, the current state of the literature on adult ADHD is particularly weak, reflecting the general lack of recognition, in both clinical and research practice, of the disorder in the adult population. For this reason most research has been generated by child psychiatrists and psychologists following up patient groups they have been evaluating from childhood. Although the literature base is not large, the research is entirely consistent in demonstrating the continuity of ADHD into adult life (Faraone et al, 2000a). This is in contrast to the popular view that ADHD is essentially a benign condition that children grow out of by the time they are adults, as suggested but not demonstrated (Barkley, 2000) by the influential paper of Hill & Schoener (1996).

Different perceptions of ADHD

In fact, the existence of clinically significant ADHD in adult life is no longer viewed as controversial among large sections of the child psychiatric, paediatric and research communities. Child and adolescent psychiatrists are well aware that many of the children they treat successfully with stimulant medication continue to require treatment beyond late adolescence, when psychiatric care is passed over to family doctors and adult psychiatric services. In addition, they see ADHD in the parents of the children they are treating, owing to the high familial rate of the condition: around 20% of parents of children with ADHD are expected to have ADHD themselves (Faraone et al, 2000b). In contrast, the reluctance of adult psychiatrists to recognise ADHD as a valid disorder beyond the childhood years is surprising, given the considerable psychiatric morbidity that is well documented to be associated with the disorder in adult life and the existence of effective treatments in the form of stimulant medication.

There are several reasons why this difference in perception between child and adult psychiatrists has come about, the most obvious being that child psychiatrists frequently come into direct contact with individuals with ADHD and see for themselves the often dramatic changes in behaviour and mental state that result from the use of stimulants. More fundamental are differences in training, with child psychiatry focusing on developmental approaches in which the onset and progression of behaviours are documented throughout the life span. In contrast, adult psychiatrists are trained to focus on the development of symptoms and behavioural change from a premorbid baseline. For this reason,
Box 1 Persistent symptoms of adult ADHD

- Internal restlessness, agitation
- Uncontrolled and ceaseless thought processes
- Poorly controlled mood, which is typically volatile and fluctuating
- Low self-esteem
- Sleep problems, particularly initial insomnia
- Inability to focus on tasks that are not immediately rewarding or stimulating
- Forgetfulness
- Disorganisation, problems with time-keeping, procrastination

Symptoms and behaviours associated with ADHD that start in early childhood and are persistent and non-fluctuating are viewed as behavioural traits, rather than symptoms of a treatable disorder. Adults face similar problems of diagnostic recognition with other childhood-onset developmental disorders such as autistic-spectrum disorders, although the treatment implications are less dramatic owing to the absence of targeted drug treatments.

A further difference is that child psychiatry has traditionally focused on objective descriptions of behaviours, whereas adult psychiatry focuses on subjective psychopathology and the mental state examination. For this reason, the signs of ADHD given in the DSM–IV criteria are essentially a list of behaviours that are maladaptive and inconsistent with the developmental level, whereas adults with ADHD have in most cases gained a degree of control over their external behaviours, which are therefore less overtly disruptive. Nevertheless, adults with ADHD describe in detail persistent symptoms that affect their daily lives (Box 1). As with other common psychiatric disorders such as anxiety and minor depression these symptoms are not qualitatively different from normal every day experiences, but they become clinically significant if they are more frequent and severe than usual, are uncontrolled and give rise to significant subjective distress in addition to psychosocial impairments at work and in personal relationships.

The fact that symptoms of adult ADHD overlap with those of personality disorder, anxiety and affective disorders and may therefore lead to difficulties in diagnosis and treatment does not make them any less valid. Importantly, where the symptoms overlap between ADHD and adolescent- or adult-onset disorders it is usually possible to make the distinction, since symptoms causing clinical impairment start early in life, persist over time and do not tend to fluctuate. There may of course be difficulties in obtaining accurate accounts of childhood behaviours from adult patients, although parents or older relatives can often provide such information. As in other areas of adult psychiatry, informant reports on current behaviour are useful to delineate the extent of psychosocial impairments in addition to the subjective complaints of individual patients. Finally, it should be recognised that many individuals currently presenting with adult ADHD will not have received the diagnosis as a child, often because the condition was less widely recognised when they were young.

Treatment with stimulants

Adult psychiatrists are also reluctant to use stimulant medications, a situation that is not helped by the current lack of licensing for their use in the adult population. This remains the case despite more than 200 controlled studies of stimulant efficacy and safety in children (Spencer et al, 1996; Schachter et al., 2000) and consistent evidence for similar high response rates among adults with ADHD (Faraone et al, 2004). Stories of adults selling stimulants on the black market remain anecdotal and have little relevance to individuals who are appropriately diagnosed and treated. There is no evidence that tolerance and addiction are a consequence of the appropriate therapeutic use of stimulants, and a number of studies have now demonstrated that the rate of drug misuse and addiction is in fact reduced by up to 50% among individuals treated for ADHD with stimulants (Huss & Lehmkuhl, 2002).

The European Consensus Statement

Experience of psychiatrists working with adult ADHD is consistent. The European Network for Adult ADHD has recently been established, in which clinicians from across Europe have come together and are currently working on a consensus statement for the diagnosis and treatment of ADHD in adults (http://www.parnassia-oud.nl/circ_volw2/zp_adhd/euro_netwerk/). What is most striking is the common perspective on the best way to diagnosis and treat adult ADHD, the impression being that direct clinical experience across diverse European countries is very similar. Overall, the evidence for the validity of adult ADHD is strong, and clinical experience suggests that it is a robust and stable concept with clear clinical implications. Nevertheless, this is an area where insufficient systematic research has been carried out on presentation, overlap with comorbid disorders, outcome and response to pharmaceutical and psychological interventions in this patient group.
References


Philip J. Asherson is a senior lecturer in molecular psychiatry at the Institute of Psychiatry (MRC Social Genetic Developmental Research Centre, SGDP Building, PO Box 80, Institute of Psychiatry, De Crespigny Park, Denmark Hill, London SE5 8AF, UK. E-mail: p.asherson@iop.kcl.ac.uk) and an honorary consultant psychiatrist at the Maudsley Hospital. His special interest is in the clinical management of adults with attention-deficit hyperactivity disorder. He has received sponsorship to attend meetings from Janssen-Cilag and Eli-Lilly.

New from the BOOKS BEYOND WORDS picture book series

Looking After My Balls

By Sheila Hollins and Justin Wilson, illustrated by Beth Webb

This book shows men with learning disabilities how to check their testicles, to look for anything that may be wrong and to seek help from their GP if they are worried. The story tells of Tom, who finds a lump while checking his balls in the shower. It contains details of physical examination and early investigation (e.g. ultrasound). It does not cover testicular cancer and its treatment. The book includes a summary section ‘How to Look After My Balls’, which can be copied for use as an A5 flyer.

June 2004, 88pp, ISBN 1 904671 05 5, Price £10.00

Many people understand pictures better than words.

Books Beyond Words is a series of picture books that has been developed to make communicating easier for people with learning or communication difficulties – and to enable discussion about difficult topics. Supporting text, suggested storylines and guidelines for use are provided for carers, supporters and professionals.

Available from: Book Sales, Royal College of Psychiatrists, 17 Belgrave Square, London SW1X 8PQ, UK.
Tel: +44 (0)20 7235 2351 x146, Fax: +44 (0)20 7245 1231.
Online ordering at: www.rcpsych.ac.uk/publications/bbw
Attention-deficit hyperactivity disorder in adults: validity unknown
Morris Zwi and Ann York
Access the most recent version at DOI: 10.1192/apt.10.4.248

References
This article cites 47 articles, 4 of which you can access for free at:
http://apt.rcpsych.org/content/10/4/248#BIBL

Reprints/permissions
To obtain reprints or permission to reproduce material from this paper, please write to permissions@rcpsych.ac.uk

You can respond to this article at
/letters/submit/aptrcpsych;10/4/248

Downloaded from
http://apt.rcpsych.org/ on September 6, 2017
Published by The Royal College of Psychiatrists