BEDFORD GROUP FOR LIFECOURSE & STATISTICAL STUDIES
Institute of Education, University of London

BG occasional paper: no. 1

Attention-Deficit/Hyperactivity Disorder

An overview and review of the literature relating to the correlates and lifecourse outcomes for males and females

Angela Brassett-Grundy and Neville Butler

INSTITUTE OF EDUCATION
UNIVERSITY OF LONDON

Attention-Deficit/Hyperactivity Disorder

An overview and review of the literature relating to the correlates and lifecourse outcomes for males and females

Angela Brassett-Grundy and Neville Butler

August 2004

First published in 2004 by the
Bedford Group for Lifecourse and Statistical Studies
Institute of Education, University of London
20 Bedford Way
London
WC1H 0AL
UK

tel: +44 (0)20 7612 6900
fax: +44 (0)20 7612 6880
email: publications@bg.ioe.ac.uk
web: www.ioe.ac.uk/bedfordgroup

The Bedford Group for Lifecourse and Statistical Studies (BG) is based in the Institute of Education, University of London. BG members share interests in the creation and analysis of longitudinal and multilevel data, which are applied to a wide range of topics in the education, social and health sciences. Further information on BG is available at www.ioe.ac.uk/bedfordgroup.

Contents

The authors

Angela Brassett-Grundy is a consultant research psychologist with ten years' experience of carrying out research into mental health, child development and education. She is also a trained psychotherapist and works part time at the Eating Disorder Unit at Guy's Hospital in London.

Neville Butler is a Visiting Professor at the Bedford Group for Lifecourse and Statistical Studies. He was the originator of two major British cohort studies, the 1958 National Child Development Study (NCDS) and the 1970 British Cohort Study (BCS70). He is currently working on the Millennium Cohort Study (MCS). Professor Butler is also Director of the International Centre for Child Studies (ICCS), an independent non-profit making charity set up in 1982.

Acknowledgements

We would like to thank the International Centre for Child Studies, the Dulverton Trust and the Teresa Rosenbaum Golden Charitable Trust for providing funding for this research. We are extremely grateful to Professor Shirley Dex, Director of the Bedford Group for Lifecourse and Statistical Studies at the Institute of Education, University of London, for her comments on this report.

1. Introduction

Attention-deficit/hyperactivity disorder (AD/HD) is a common disorder of childhood onset, characterised by problems with concentration, impulse control and overactivity, and reported to be associated with a variety of adverse adolescent and adult outcomes. However, the definition, measurement and causes of AD/HD are considered to be debatable issues, which could raise questions for whether and how meaningful research into its lifecourse and outcomes can be conducted. In spite of these reservations it is the most extensively studied childhood disorder, with several thousands of peer-reviewed papers in the scientific literature.

In this report we aim to provide an overview of what AD/HD is, take a brief look at some of the current debates surrounding the conceptual and measurement issues associated with AD/HD, and then review some of the recent literature with a specific focus on the lifecourse outcomes for individuals diagnosed with AD/HD.

It should be noted that this review is a brief one given that the literature on this disorder is now extensive (see, for example, the bibliography composed by the United States National Library of Medicine, 1998), and has been annotated more than 350 times already (for example, see Swanson et al, 1993). The main aim of our review is to provide the necessary background to a larger project. The intention here is to identify important gaps in the literature and knowledge base, which secondary analysis of prospective large-scale longitudinal cohorts, tracking individuals from birth to adulthood, could help to fill. This review highlights the fact that such datasets are rare and that their analysis offers important opportunities for advancing knowledge, particularly of the adult outcomes of childhood AD/HD.

In section 2, we ask ‘What is AD/HD?’ We then present a discussion of the aetiology, identification, prevalence and treatment of AD/HD in sections 3-6. In section 7 we discuss the correlates of childhood AD/HD. In section 8, the adolescent and adult outcomes reported in children with AD/HD are reviewed, with a focus on recent literature. In section 9 we offer some conclusions based on the debates and evidence presented. Finally we highlight areas that are worthy of more investigation and explain this in respect of the potential of the 1970 British Cohort Study (BCS70), in which information on AD/HD is contained in an ongoing national survey of a cohort of British children born in 1970 and followed up over a 30-year timespan.

2. What is AD/HD?

Something like AD/HD was described by the ancient Greeks (Hussain and Cantwell, 1991) and widely observed by physicians many decades ago, such as Still (1902), who supplied the first formal description. Current opinion amongst psychiatrists and researchers is that AD/HD is a relative inability to regulate and organise behaviour, which is equated to an impairment of the executive functions that are largely controlled by the frontal lobes of the brain (Barkley et al, 1992b; Barkley, 1997; Brown, 2000). Brown (2000) produced a model of executive function focusing on six areas, alongside which he listed the expected impairments. These are illustrated in Table 1.

Table 1: Brown's model of executive functions impaired in AD/HD (2000)

EXECUTIVE FUNCTION	EXAMPLE OF RESULTING IMPAIRMENT
1. Organise, prioritise and activate	• trouble getting started • difficulty organising work • misunderstand directions
2. Focus, shift and sustain attention	• lose focus when trying to listen • forget what has been read and need to re-read • easily distracted
3. Regulate alertness, effort and processing speed	• excessive daytime drowsiness • quickly lose interest in task • effort fades quickly • difficult to complete a task on time • slow processing speed • inconsistent productivity
4. Manage frustration and modulate emotion	• very easily irritated • often sad, worried, unhappy • fussy about getting things perfect • feelings hurt easily • overly sensitive to criticism
5. Working memory and accessing recall	• forget to do a planned task • forget intended words or actions • difficulty recalling learned material • difficulty following sequential directions • lose track of belongings • quickly lose thoughts put on hold
6. Monitor and regulate action	• find it hard to sit still or be quiet • rush things – slap-dash • often interrupt, blurt things out

Brown emphasised that AD/HD is not simply a problem of hyperactivity and impulsivity but of the ability to monitor activity in appropriate settings. As can be seen from Brown's model, the executive functions implicate abilities in goal-orientated processes such as initiation and maintenance of efficient strategies, the programming and planning of motor behaviour skills, learning and applying contingency rules, abstract reasoning, problem solving, and of sustaining attention and concentration. They require effort and active 'on-line' monitoring resources. Van der Meere (1996) stated that AD/HD children are unable to allocate mental effort in an adaptive way to meet the needs of different situations.

Barkley (1997, 1998) offered an alternative conceptualisation, suggesting that AD/HD children have a poor ability to relate to time and to chronology. The human ability to plan is linked to the ability to look back in time and appraise past experiences. In Barkley's framework AD/HD is essentially an impaired capacity for marking time and sensing its duration in order to anticipate the setting of future motor responses and more complex behavioural chains.

However it is conceptualised, AD/HD is clearly characterised by symptoms that are both cognitive (e.g. working memory, speed processing deficits) and behavioural (inattention, hyperactivity and increased impulsivity). Swanson et al (1998a) described AD/HD as a '…polytypic syndrome with multiple biological bases' that has '…different clinical manifestations associated with multiple underlying cognitive processes and with multiple neural networks of attention' (p. 456). This alerts us to the fact that AD/HD can manifest itself in a variety of different ways and has a variety of causes, making it a very complex condition to understand. It is believed by some that AD/HD is located at the extreme end of a continuum with symptoms of inattention, hyperactivity and impulsivity being distributed continuously in the general population (e.g. Murphy et al, 2000; Solanto et al, 2001). What marks AD/HD apart is that the levels of activity, distractibility and impulsivity are considered developmentally inappropriate, cause impairment to normal functioning, and are evident in multiple settings, including home, school and in social relationships.

AD/HD was known variously in the 1970s as hyperkinesis (Latin for 'superactive') or hyperactivity, and in the 1980s as Attention Deficit Disorder (ADD), which could be diagnosed with or without hyperactivity. The condition is now referred to as AD/HD, which has various subtypes that can be identified in recognition that in different people there may be different emphasis between inattention and hyperactivity. Thus, the change in nosology reflects the developments in research; AD/HD better describes the clinical phenomena. Also, the consistent findings in the factor-analysis literature demonstrate that only two dimensions are needed to explain the covariation of AD/HD symptoms, i.e. inattention and hyperactivity-impulsivity. As a result, the diagnostic criteria listed in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*, (DSM-IV) published by the American Psychiatric Association (1994) (see Box 1 in section 4), refer to three major subtypes:

(1) inattentive type;
(2) hyperactive-impulsive type; and
(3) combined type (inattention and hyperactivity-impulsivity).

These subtypes were considered by Faraone et al (1998) to represent developmental phases of AD/HD, with hyperactivity and impulsivity appearing first, followed by the development of inattentive symptoms. 'Hyperkinetic Disorder' (HKD) is seen as a condition linked to AD/HD, as described by The World Health Organisation's (1993) *International Classification of Diseases 10th Edition* (ICD-10). It has the same criteria as for combined-type AD/HD, and as such its definition is narrower and diagnostic criteria are more stringent[1]. The diagnostic systems of the ICD are traditionally used in Britain and Europe rather than the DSM. This may go some way to explaining why AD/HD is a less well-known condition outside the USA, and why prevalence rates appear lower than in the USA.

As highlighted in the introduction, the definition of AD/HD is controversial. Some still argue that AD/HD does not really exist but is an excuse for bad behaviour in normal children who, if disciplined properly by their parents, would not have problem behaviour. Others have claimed that AD/HD does not exist because children diagnosed with AD/HD can concentrate for hours on things such as computer/video games. The persistence of the debate surrounding the existence of AD/HD has become primarily a public one conducted in the media, although a few mental

[1] See Swanson et al (1998b) for a discussion of the diagnostic, epidemiological, treatment and prognostic differences between AD/HD and HKD.

health professionals have joined the dissenters. Duplantier (2003) quotes Tom DeWeese, of the American Policy Center, who argued that psychologists have developed a habit of making up diseases and disorders in order to make money, AD/HD being one example. DeWeese claimed wrongly that there have been no peer-reviewed scientific papers showing that AD/HD exists, and that it does not meet the medical definition of a disease or syndrome, but instead that it does meet a financial definition, pointing to healthcare and educational profiteering. Dr Bob Jacobs, a US psychologist, echoed this point, stating that doctors and pharmaceutical companies have turned a childhood behavioural problem into a disorder (Goodenough, 2003). Goodenough (2003) also quoted Dr Fred Baughman Jr, a US neurologist, who wrote a letter in 1998 to the US Attorney General referring to the representation of AD/HD as a disease and the drugging of millions of normal children as the 'single, biggest health care fraud in US history'.

Other critics believe that AD/HD has more to do with social change than illness, such as Prof. Priscilla Anderson (Young Minds, 2003). She attributed AD/HD in the UK to the great changes over the last twenty years that have restricted children's activity, such as the rigid curriculum, increased formal testing, and reduced playtime and creative activities in schools, and the closure of some playgroups, adventure centres, parks and youth clubs. The many poverty-stricken families that cannot afford holidays or day trips on which a child's excess energy could be expended are cited, and it is suggested that children of the twenty-first century in the UK are more contained inside schools, homes, cars and childcare settings than ever before, unlike children in other European countries. She stated, somewhat cynically, that diagnosing AD/HD and prescribing stimulants costs less than trying to change and improve services for children.

What is the answer to these criticisms? A suspected child developmental disorder should surely be the subject of rigorous scientific research to determine its existence rather than be aired in the media often by non-specialists and sometimes in a sensational manner. Indeed, much research has been conducted over the last century, and more recently using a great deal of sophisticated methodology, such as advanced brain-imaging techniques and/or randomised double-blind controlled cross-over designs. Research has shown consistently that a group of young people can be identified exhibiting distinct features that cluster together. Whilst most people may have one or two AD/HD symptoms, it is much less common for people to have a whole cluster of these symptoms. Many individuals do exist who possess this cluster and it is agreed that these represent the extreme end of a continuum of normally occurring symptoms. AD/HD is now recognised by all the major professional, medical, psychiatric, psychological and educational associations, and the Office for Civil Rights, the US Congress and the National Institutes of Health, all of which are informed by the scientific research. These are points that have been well articulated by Barkley (2002a).

Like other children, those with AD/HD have the ability to concentrate in situations that are very stimulating and interest them. But unlike others, those with AD/HD find it much more difficult to concentrate for sustained periods of time on everyday things that are more mundane, e.g. what the teacher is saying throughout a class at school. In fact, it can be argued that computer games are perfect for children with AD/HD because they allow them to be impulsive and to seek immediate gratification. One of the problems of AD/HD is an inability to plan ahead and appreciate the consequences of an action before it is executed. Computer games encourage impulsive responses without any real consequences – there is always another life to be used or the restart button can be pressed. In games where there is a pay-off for being more devious or less impulsive, AD/HD children tend not to learn the lesson and continue to press the fire button. Even though their space ship is exterminated or their super-hero killed, they happily play the game over and over again, repeating the same mistakes. Real life is not like this and it is here where AD/HD children have a problem, for example, often making enemies of their peers for lashing out or interrupting and shouting out in class.

In response to the allegation that a lack of discipline was at the root of the problems experienced in AD/HD, Barkley and colleagues endeavoured to test the effectiveness of providing AD/HD

children with more discipline, in the absence of any other intervention. They found that this actually worsened rather than improved the children's behaviour (quoted by Booth et al, 2003). In addition, if one accepts that the cause of AD/HD has a genetic and biological background, then how parents treat their child cannot cause AD/HD. Parents could, however, have an effect on how AD/HD manifests itself, for example, by providing positive reinforcement for good behaviour. This is one strategy that has been shown to be effective in helping AD/HD children cope with their symptoms. If one observes any one of the parents who have a child diagnosed with AD/HD, it becomes apparent that bad parenting is not the cause, and authoritarian parenting is not the answer. Many of these parents have other children whom they are parenting in a similar way but who do not exhibit the extreme cluster of symptoms seen in AD/HD. Parents are also often aware that from an early age there was a difference between their AD/HD child and other children, which cannot be explained simply by the degree of discipline they deliver.

Nowadays the controversy surrounding AD/HD exists largely in the media, whilst the overwhelming majority of child psychiatrists, psychologists and those employed in affiliated professions pay little heed to such arguments, preferring instead to get on with the business of producing a solid evidence-base to inform assessment, diagnosis, treatment and expected outcomes of this condition. AD/HD is described the world over and has been recognised for over one hundred years. As such, its existence cannot be attributed to the social changes in Britain over the last twenty years. Rather, it is our ability to recognise and diagnose AD/HD that has increased over time and, just like any other disability, it would benefit from being perceived as a challenge rather than an excuse.

3. Aetiology of AD/HD

Research into the aetiology of AD/HD has revealed genetic, biochemical, environmental and social factors. Hill and Taylor (2001) maintained that AD/HD is a syndrome caused by several different pathologies, among which a genetic susceptibility seemed to be the most common. Although environmental factors should not be discounted, evidence from family, twin and adoption studies point to a substantial genetic component in the aetiology of AD/HD. Heritability estimates range from 0.50 to 0.98; in monozygotic twins, AD/HD concordance rates are in the higher range of 0.80-0.98 (Faraone and Doyle, 2000; Faraone et al, 2000b; Faraone et al, 2000d; Gillis et al, 1992; Gjone et al, 1996; Levy et al, 1997; Pennington and Ozonoff, 1996). It has been advanced that hyperactive-impulsive type AD/HD tends to 'breed true' but inattentive and combined subtypes do not (Faraone et al, 2000a).

Given the impulsive and driven motor-like behaviour characteristic of AD/HD it is perhaps not surprising to find that research has implicated dopamine, a neurotransmitter used by the brain with a central role in psychomotor activity and reward-seeking behaviour. Decreased dopaminergic functioning has been found in the brains of those with AD/HD (Bowden et al, 1988; Rapoport et al, 1974; Thapar et al, 1999), and a positive correlation between the expression of the dopamine transporter gene 'DAT1' and hyperactivity-impulsivity has also been reported (Cook et al, 1995). Elevated levels of dopamine transporter genes have also been found in the brains of AD/HD adults, by Dougherty et al (1999). Research into the dopamine D4 receptor gene (DRD4) has demonstrated a link with AD/HD, where a 7-fold repeat form of DRD4 has been found to occur more frequently in AD/HD individuals (La Hoste et al, 1996; Malone et al, 1994; Swanson et al, 1998c). Other neurotransmitters have been implicated such as noradrenaline (referred to as 'norepinephrine' in the USA), the role of which has been linked to vigilance, alertness and the 'turning on' of dopamine pathways from the cerebellum. Reduced levels of noradrenaline metabolites have been reported in those with AD/HD (Shekim et al, 1979; Shen and Wang, 1984; Zametkin et al, 1984), which is believed by some to account for inattentive symptoms (Heilman et al, 1991). Other genes have been implicated, such as the 4-repeat allele[2] which was associated with enhanced risk of AD/HD, and the interleukin-1 receptor antagonist 2-repeat allele (related to the differentiation of dopaminergic neurons) which was associated with decreased risk for AD/HD (Segman et al, 2002). But molecular genetic studies have produced conflicting results and Faraone and Doyle (2000) pointed out, importantly, that the mode of familial transmission is still unclear and more work needs to be done on delineating genetically homogenous subtypes. They also stated that larger sample sizes must be studied and/or families selected in which genes are exerting the largest risk, e.g. those where conduct disorder is comorbid, or those where AD/HD persists into adolescence (Faraone et al, 2000d).

Looking beyond genes and brain chemistry, but staying with physical and biological factors, a number of studies using a variety of sophisticated brain imaging techniques have claimed differences in the morphology of the brains of those with AD/HD, which may themselves be a result of genetic susceptibility. These have revealed a bewildering array of findings including: a dysfunction in the prefrontal and subcortical striatal areas (Castellanos et al, 1996); decreased volume in the anterior corpus callosum, right anterior white matter and cerebellar, decreased blood flow and energy utilisation in the prefrontal cortex and corpus striatum, less brain asymmetry and dysregulation of catecholamine systems (Paule et al, 2000); reversed asymmetry of the head of the caudate nucleus, smaller volume of the head of the left caudate, and smaller volume of the white matter in the right frontal lobe (Semrud-Clikeman et al, 2000); functional abnormalities in the putamen (which is mainly involved in the regulation of motor behaviour) (Teicher et al, 2000); involvement of the dorsolateral regions of the prefrontal cortex, dysfunctional circuitry to/from/in the basal ganglia, cerebellum, and locus coeruleus (Solanto et

[2] An 'allele' is any of two or more genes that have the same relative position on structurally dietetically similar chromosomes.

al, 2001); reduced regional brain size localised mainly to the inferior portions of the dorsal prefrontal cortices bilaterally, reduced brain size in the anterior temporal cortices bilaterally, and increased grey matter in the large portions of the posterior temporal and inferior parietal cortices bilaterally (Sowell et al, 2003; Swanson et al, 1998a); and, finally, differences have been found in resting brain activity and in evoked (i.e. event-related) potentials (see the review by Barry et al, 2003). Barkley (2002a) provided the following summary of the complex research that has revealed brain differences in those with AD/HD:

> *The central psychological deficits in those with ADHD have now been linked through numerous studies using various scientific methods to several specific brain regions (the frontal lobe, its connections to the basal ganglia, and their relationship to the central aspects of the cerebellum). Most neurological studies find that as a group those with ADHD have less brain electrical activity and show less reactivity to stimulation in one or more of these regions. And neuro-imaging studies of groups of those with ADHD also demonstrate relatively smaller areas of brain matter and less metabolic activity of this brain matter than is the case in control groups used in these studies.*
>
> (Barkley, 2002a: 90)

Words of caution are necessary when evaluating the genetic and neurological evidence for the cause of AD/HD. First, different diagnostic criteria have been used over the years to define samples, which will give rise to different results. Second, sample sizes have tended to be small which can present problems for interpretation and generalisation. Third, AD/HD is a complex group of behaviours so one can assume that it will not fit a discrete unitary category, rendering the search for a single neurological cause too simplistic. Finally, most of the studies in this area have been cross-sectional suggesting in some instances correlation rather than causation. For example, it could be argued that brain differences may be an effect of AD/HD, or of its treatment, as opposed to the cause. Regarding medical treatment, a study by Castellanos et al (2002) suggests that stimulant drugs, at least, do not contribute to group differences in brain morphology in people with AD/HD. Although the methodology of brain imaging research is becoming increasingly more sophisticated, there is still a long way to go in this area. This will no doubt benefit from a prospective longitudinal approach.

Over the last twenty years some studies have claimed a link between complications of pregnancy, delivery and infancy, and the later development of AD/HD. It is possible that foetal distress may selectively damage striatal neurons and affect the developing frontal lobe-basal ganglia networks, i.e. the brain regions indicated in the aetiology of this disorder. Gillberg et al (1983) and Taylor et al (1991) found such an association but only for those with severe hyperkinetic syndrome in childhood as opposed to AD/HD. Sprich-Buckminster et al (1993), however, found an association with AD/HD that was strongest in those with comorbidity and non-familial AD/HD. Milberger et al (1997) found that it was specifically maternal bleeding, maternal smoking and illicit drug use in pregnancy that might account for these findings. Kortimaa et al (2003) found in a birth cohort population study an association between maternal smoking during pregnancy and AD/HD which held true even after controlling for gender, family structure, socio-economic status (SES), mother's age and mother's alcohol intake. Using a population-based sample of over 1,400 pairs of twins Thapar et al (2003) confirmed that maternal smoking was associated with AD/HD in offspring, independent of potential confounds and in addition to genetic effects. Evidence has also been presented for a link between AD/HD and low birthweight (Breslau et al, 1996; Whitaker et al, 1997) and preterm birth (Bhutta et al, 2002) (both of which may be caused by maternal smoking), while severe and chronic sleep problems in infancy were another association reported (Thunstrom, 2002).

Aside from the focus on the environment experienced in the womb, the search for environmental causes of AD/HD has looked outside, to the family in which AD/HD children are raised. Some have found a link between AD/HD and low SES (Peterson et al, 2001; Schachar et al, 1981),

whilst others have not (Szatmari et al, 1989; Taylor et al, 1991). Peterson et al (2001) suggested that AD/HD was associated with a lowered IQ, which in itself was related to a low SES background. Children with AD/HD have been found more often to have parents with mental health problems, such as anxiety disorders and substance abuse (Egeland et al, 1990; Russo and Beidel, 1994). Research in the same area suggested a link between AD/HD and family conflict, marital discord (Edwards et al, 1995; Gillberg et al, 1983; Milberger et al, 1997) and familial psychosocial problems, especially critical expressed emotion (Peris et al, 2003; Taylor et al, 1991; Thunstrom, 2002). But parenting practices do not appear to be ultimate causes of AD/HD (Barkley, 1998; Hinshaw, 1994; Johnston and Mash, 2001). Indeed, it is likely that patterns of family interaction and family influence may be a consequence of AD/HD (Barkley, 1989; Marshall et al, 1990; Woodward et al, 1998). These parenting attributes may also shape and exacerbate AD/HD symptoms (Campbell, 1990). Many of the above associations will need teasing out further. As Wolf and Wasserstein (2001) point out, psychosocial context and non-genetic familial influences are critical variables that have been undervalued in research to date.

Another cause of AD/HD sometimes cited is 'poor nutrition'. The possible link between nutrition and AD/HD began in the 1970s when Feingold (1975) suggested that food dyes and preservatives caused genetic changes, the effect of which was to make children become intolerant to these substances and to suffer behavioural and learning difficulties. However, double-blind trials of the effects of food colourings did not offer any support to Feingold's hypothesis (Mattes and Gittelman, 1981). More recently people have considered intolerances to natural foodstuffs, as opposed to added chemicals, colourings and preservatives, and they have recommended exclusion diets that identify foods that trigger AD/HD symptoms. Single case studies have shown that some children can be helped with diets that exclude the things of which they are intolerant (Kaplan et al, 1989; National Institute of Mental Health, 2003; Weiss et al, 1980). However, the fact that a few children can be helped with a change to their diet is clearly not proof that poor nutrition is the cause of their AD/HD. Although research has suggested that limiting food additives and sugar does not prevent AD/HD, it seems common sense to pay attention to the overall general health of any child, including exercise, rest and nutrition, which may help a child with AD/HD to manage their symptoms.

Thus, it is apparent from the review of AD/HD aetiology provided here, as Sowell et al (2003) recognise, that AD/HD is a disorder of heterogeneous causes that has correspondingly heterogeneous neuroanatomical underpinnings. The weight of the evidence of recent research indicates that a genetic susceptibility seems to be the most common factor, although biochemical, psychosocial and environmental factors also play a part and merit further investigation.

4. Recognising AD/HD

Allied to the problems of defining AD/HD are the clinical problems of identification and measurement of AD/HD. AD/HD is a real disorder, characterised by impairment to the executive functions, but how do we identify accurately those with this cluster of impairments? How valid, sensitive and selective are our tests, and to what degree do these reflect different tolerance levels between families, schools, societies and cultures?

One could argue that AD/HD cannot be accurately diagnosed since there is no unique clinical, psychological or biological test for AD/HD. Also, given that AD/HD can represent the extreme end of a continuum of normally occurring symptoms, difficulties might be faced with drawing a cut-off point for a clinical case of AD/HD. That there is no unique test for AD/HD is true of most psychiatric disorders, including other disabling pathology such as schizophrenia and autism. In spite of this criticism, clear-cut clinical diagnostic criteria have been developed, based upon years of research, and these are being refined constantly. The most widely accepted diagnostic criteria in use for identifying AD/HD are those in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*, (DSM-IV) (American Psychiatric Association, 1994), which are shown in Box 1. These criteria represent the dominant conceptualisation of AD/HD for clinical diagnosis and research purposes, and go some way to defining a threshold for a clinical case, with conditions of chronicity, disability and pervasiveness, and recognition of sub-threshold symptomatology.

Box 1: DSM-IV Criteria for AD/HD

A. Either 1 or 2:

1. Six or more of the following symptoms of **inattention** have persisted for at least six months to a degree that is maladaptive and inconsistent with developmental level:

- **a** Often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities.
- **b** Often has difficulty sustaining attention in tasks or play activities.
- **c** Often does not seem to listen when spoken to directly.
- **d** Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions).
- **e** Often has difficulty organizing tasks and activities.
- **f** Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as homework).
- **g** Often loses things necessary for tasks or activities (toys, school assignments, pencils, books, or tools).
- **h** Is often easily distracted by extraneous stimuli.
- **i** Is often forgetful in daily activities.

2. Six or more of the following symptoms of **hyperactivity-impulsivity** have persisted for at least six months to a degree that is maladaptive and inconsistent with developmental level:

   Hyperactivity

   a. Often fidgets with hands or feet or squirms in seat.
   b. Often leaves seat in classroom or in other situations in which remaining seated is expected.
   c. Often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness).
   d. Often has difficulty playing or engaging in leisure activities quietly.
   e. Is often 'on the go' or often acts as if 'driven by a motor'.
   f. Often talks excessively.

   Impulsivity

   g. Often blurts out answers before questions have been completed.
   h. Often has difficulty awaiting turn.
   i. Often interrupts or intrudes on others (such as butting into conversations or games).

B. Some hyperactive, impulsive, or inattentive symptoms that caused impairment were present before age 7 years.

C. Some impairment from the symptoms is present in two or more settings (such as in school or work and at home).

D. There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.

E. The symptoms do not occur exclusively during the course of a pervasive developmental disorder, schizophrenia, or another psychotic disorder and are not better accounted for by another mental disorder such as a mood, anxiety, dissociative, or personality disorder.

AD/HD Types Using DSM-IV criteria

AD/HD, predominantly inattentive type
Meets inattention criteria (section A1) for the past 6 months.

AD/HD, predominantly hyperactive-impulsive type
Meets hyperactive-impulsive criteria (section A2) for the past 6 months.

AD/HD, combined type
Meets criteria for section A1 and section A2 for the past 6 months.

AD/HD, not otherwise specified
Prominent symptoms of inattention or hyperactivity-impulsivity that do not meet the criteria for AD/HD.
In partial remission.

The criteria in A, B, C, D and E in Box 1 have to be met in order for a diagnosis of AD/HD to be made. These criteria show that a minimum number of symptoms must be present in at least two settings, typically at home and at school. The value of assessment of AD/HD appears to be better appreciated by a wider variety of health professionals in the USA compared to the UK, where

some have called for a more clearly defined role for general practitioners in screening for AD/HD (e.g. Levy, 1997). Researchers such as Mannuzza et al (2002) warned against relying on adult retrospective self-report of the symptoms of childhood AD/HD, arguing instead for the importance of obtaining contemporaneous information on childhood symptoms in establishing a childhood history of AD/HD.

Mota and Schachar (2000) criticised the DSM-IV diagnostic algorithm for AD/HD due to its low inter-rater agreement. They expressed a preference for an algorithm based on a 'receiver operating characteristic' (ROC) procedure[3], which identified symptoms that predicted impairment. They found that this was most effective for identifying AD/HD when parents and teachers both agreed, which again highlights the importance of detecting symptoms in more than one life domain. However, most doctors and psychiatrists accept that a clinically valid diagnosis of AD/HD can be reached through a comprehensive and thorough evaluation using well-tested diagnostic interview methods, alongside the DSM-IV criteria. These comprise: obtaining a thorough history covering the presenting symptoms; ruling out other physical or mental conditions that may have similar symptoms; detailing possible comorbid conditions; and obtaining medical, developmental, educational, psychosocial and family histories. Criteria normally specify that AD/HD symptoms should have persisted for a length of time, typically six months, to a degree that is maladaptive and inconsistent with the child's developmental level. A thorough assessment will ascertain whether symptoms are occurring exclusively during the course of a pervasive developmental disorder, schizophrenia, or other psychotic disorder, in which case AD/HD may be ruled out, and also whether the symptoms are not better accounted for by another mental disorder (e.g. mood disorder, anxiety disorder, dissociative disorder, or a personality disorder).

It is widely agreed that a multi-modal approach to assessment is optimal for an accurate diagnosis of AD/HD (Barkley, 1990; Doyle et al, 2000; DuPaul et al, 1992; Schaughency and Rothlind, 1991) and a wealth of diagnostic schedules and tools have been developed to achieve this. These specify that data are gathered where possible from parents, doctors, teachers, psychologists, peers and the individuals themselves. It seems the problem with diagnosing AD/HD effectively is not that a method of measurement does not exist, but rather that multi-modal, multi-agency assessments take time. Moreover, there are few specialists available to carry them out, especially in Britain. In addition, there are pitfalls in distinguishing accurately between those who will not pay attention (with intentional problems) from those who cannot pay attention (AD/HD); the former may exhibit disruptive behavioural symptoms that lead to false diagnosis of AD/HD. However, according to Nakamura (2002), there is little scientific evidence of overdiagnosis of AD/HD. Rather, it is probably the case that fewer children are being identified with AD/HD than suffer from it.

[3] ROC is part of a statistical technique in which one plots the sensitivity of a diagnostic test as a function of its non-specificity (one minus the specificity). The resulting ROC curve indicates the intrinsic properties of a test's diagnostic performance and can be used to compare the relative merits of competing procedures.

5. Prevalence of AD/HD

We turn now to the questions, 'Who has AD/HD and how big a problem is it?'

Many believe that AD/HD is the single most important specific problem complicating school life, the seriousness of which is further highlighted by the fact that the cost to the national exchequer in the UK was estimated at £1 billion per annum (Knapp, 1997). Prevalence figures for childhood AD/HD, based on clinical populations, range from 3-10 per cent, and the maximum age risk lies somewhere between 5 and 10 years (American Psychiatric Association, 1994; Costello, 1989; Parr et al, 2003; Szatmari et al, 1989; Taylor, 1994). Prevalence figures of course vary according to: the definition or diagnostic criteria used; the nationality, size and selection of the sample; and the subtype under study, e.g. the inattentive subtype seems to be more prevalent than the combined type which is slightly more prevalent than the hyperactive-impulsive type (Baumgaertal et al, 1995; Gomez et al, 1999; Pineda et al, 1999; Wolraich et al, 1996). Some epidemiological studies have claimed higher prevalence rates for AD/HD, ranging from 9-19 per cent (Paule et al, 2000; Shekim et al, 1985; Taylor et al, 1991), again suggesting that under-diagnosis may be common.

Most authors agree that males with AD/HD out-number females, but studies report varying degrees of male overrepresentation, from ratios of 1.5:1 to 12:1 (e.g. Gomez et al, 1999; Parr et al, 2003; Pineda et al, 1999; Swanson et al, 1998b; Wolraich et al, 1996). Some believe that these ratios represent a real gender difference relating to underlying biological factors (e.g. Anderson and Teicher, 2000) and may differ according to the subtype of AD/HD. Some studies have stated that this differential decreases with age (perhaps because boys exhibit more hyperactive-impulsive symptoms, which are reported to decrease with age). Others have argued that these ratios are biased by underdiagnosis of AD/HD in females and suggest that girls with AD/HD are a 'silent minority' since their symptoms of hyperactivity and aggression are not as florid as those of boys (Gaub and Carlson, 1997; Hynd and Hooper, 1995; Silverthorn et al, 1996). It is also likely that some of these differences represent identification and/or referral biases on the part of parents, teachers and health professionals (e.g. Glod et al, 1996; Swanson et al, 1998b; Taylor, 1994).

6. Treating AD/HD

Once AD/HD has been identified, what methods are available to treat the problems presented by their symptoms?

AD/HD has been treated in a variety of ways, and has focused on the individual him/herself and/or on the people with whom they interact, e.g. parents, teachers. One of the main treatments of choice has been pharmacological, specifically stimulants. These have gained popularity in the last twenty years although their beneficial effect on AD/HD-like childhood behaviour was first noted by Bradley (1937), a physician in the USA. A wealth of good quality research and clinical practice has demonstrated the efficacy of stimulants for treating AD/HD (e.g. see the reviews by Schachar and Tannock (1993), Swanson et al (1993) and Hechtman and Greenfield (2003)). Methylphenidate (commonly known by its trade name 'Ritalin') has been the most popular stimulant used in medicating those with AD/HD. It may seem counterintuitive to give someone who has problems with hyperactivity stimulant medication. However, such stimulants are classified as sympathomimetic compounds, and not as amphetamines, and do not act as amphetamines in the doses used for AD/HD. They act by releasing and inhibiting the reuptake of catecholamines, mainly dopamine, in the central nervous system. It has been suggested that this enhances the brain's executive functions such that the individual is capable of remaining focused for longer, thinking ahead, delaying gratification, and planning and appreciating the consequence of an action before it is carried out. Thus, by targeting dopaminergic sites in the brain, stimulants appear to augment regulation of effort over time. In recent years slow-release, longer-acting methylphenidate preparations have come on to the market, lasting up to twelve hours instead of up to four (e.g. 'Adderall' and 'Concerta'). Other stimulants used widely are dextroamphetamines (with trade names such as 'Dexedrine' and 'Dextrostat'). Stimulant drugs exert similar effects in people with and without AD/HD, supporting the notion that symptoms of inattention, hyperactivity and impulsivity are continuously distributed in the general population.

Antidepressants, such as nortriptyline, desipramine and bupropion, have also shown clinical efficacy but have been used mainly as a second line treatment in those who show a poor response to stimulants, or who have intolerable side-effects with stimulants. They can also benefit those who have comorbid conditions such as mood disorders, hypertension, anxiety or tics (e.g. Daviss et al, 2001; Spencer et al, 1993; Wilens et al, 1993; Wilens et al, 2001). Alpha-blockers such as clonidine and guanfacine have also been used with some success, although effect sizes may be smaller than that of stimulants (Connor et al, 1999; Scahill et al, 2001; Szymanski and Zolotor, 2001; Taylor and Russo, 2001). A new noradrenergic agent called atomoxetine ('Straterra') has recently been tested and has been shown to have longlasting effects on AD/HD symptoms. In the future it may prove useful as another non-stimulant treatment option (Simpson and Plosker, 2004).

Critics have claimed that the increase in stimulant prescriptions witnessed over the last 10 years is evidence that AD/HD is a fad disorder and we should not be medicating children to this extent. To investigate this claim Jensen et al (1999) examined epidemiological data from 1,285 children and their parents living in four US communities, which showed that only one in eight children meeting AD/HD criteria (5.1% of the pooled sample) had been treated with stimulants during the previous twelve months. They extrapolated from this to conclude that overtreatment with stimulants was not occurring across US communities in general. Swanson et al (1995) and Jensen (2000) have stated that there is a heightened professional and public awareness that has increased the level of identification and treatment of this disorder, leading to an increase in the number of stimulant prescriptions. It is probably the case that improved recognition of AD/HD has been catching up with all those people who have lived with AD/HD and yet who had not received any prior treatment, including adults. In addition, not all stimulant prescriptions represent medication for people with AD/HD: the figures also include exports to other countries and so do not necessarily reflect the number of people taking it in a particular country. Stimulants are also

prescribed for other conditions such as narcolepsy and some geriatric conditions (Safer et al, 1996), our awareness of which has also increased over the last decade.

Goodenough (2003) stated that Dr Bob Jacobs claimed that the misdiagnosis of AD/HD results in youngsters being prescribed powerful drugs such as Ritalin that may affect long-term mental and physical development, for example some have claimed it stunts growth. Others have argued that the medication quietens disruptive youngsters, making them more docile and dampens their spontaneity. It has also been suggested that treating children with stimulants will make them more likely to abuse illegal substances later in life. Others have pointed to the multi-million dollar profits earned by drug companies who manufacture medical treatments for AD/HD that would be wiped out quickly if the existence of AD/HD were disproved.

However, fifty years of research into medical treatments for AD/HD show that there is no evidence of negative long-term effects (Hechtman and Greenfield, 2003; Vitiello, 2001). Neither is there scientific evidence that it affects height (Hechtman and Greenfield, 2003; Klein and Mannuzza, 1988; Sund and Zeiner, 2002; Vincent et al, 1990). Jensen (2000) argued that sometimes it is the more toxic and dangerous drugs that have been used to treat AD/HD, such as antipsychotics (e.g. risperidone, thioridazine), often by those who do not understand how to medicate AD/HD properly, that give cause for concern; these do have permanent effects and should be used very carefully. In addition, Nakamura (2002) warned against the effects of not treating AD/HD in childhood: if the brain is not exposed to the proper cognitive and emotional stimuli, simply because a child cannot concentrate upon and process them, at a time when it is at a critical phase of development, there is a risk to brain integrity and function which will lead to long-lasting impairment. It seems that there is little scientific evidence of overprescription of stimulant medication, and that it is in fact the case that fewer children are being treated for AD/HD than suffer from it. Also, there is no evidence that taking stimulant medication causes later drug-abuse; in fact, stimulants appear to protect against drug abuse (see section 8.2). It is possible that those who go untreated in childhood begin to self-medicate by resorting to the use of illegal substances, as well as smoking cigarettes or ingesting excessive amounts of caffeine. It is true that not all those with AD/HD respond to medication with stimulants. To some extent this is explained by mismanagement of medication, e.g. failure to monitor progress and assess side-effects properly, failure to titrate doses properly, failure to investigate effects of alternative stimulants where methylphenidate is ineffective, and failure to use different medicines in combination.

But medication is not the only available treatment for AD/HD. There are many individuals who choose not to take drugs and would prefer a non-invasive, non-chemical approach. Indeed, a plethora of psychosocial interventions are available, a great deal of evidence for the effectiveness of which now exists (e.g. Barkley, 2002c; Barkley et al, 1992a; DuPaul and Eckert, 1998; Pelham et al, 1998; Pfiffner and McBurnett, 1997; Sonuga-Barke et al, 2001; Strayhorn and Weidman, 1989). The first, and perhaps most important, of these interventions is simply to educate and provide information about what AD/HD is to those who have it.

A major approach to the non-medical treatment of AD/HD is behavioural therapy, which is based on the principles of behaviour modification and uses positive and/or negative reinforcement to elicit or extinguish target behaviours. This utilises modelling and role-playing of target behaviour, and encourages the development of routines and habit. It teaches how to break tasks down into smaller units, how to prioritise and organise, and how to develop good systems for reminding and memory-jogging. It emphasises the use of praise and constructive feedback, with rewards for good achievement.

Time-management training may also be a component of behavioural therapy, including measures such as promoting the use of diary systems, and encouraging the planning of an event in advance, building in ‘getting ready’ time and aiming always to arrive early. It may also be helpful to encourage more creative thinking by matching skills to context, such as choosing school

subjects or a job that fit AD/HD symptoms, e.g. sports, carrying out two part time jobs, manual work. AD/HD individuals may be taught how to engage in 'acceptable' fidgeting like writing notes during class/meetings, and are encouraged to use break times to be active and let off hyperactive steam.

Cognitive therapy offers another psychosocial intervention for the treatment of AD/HD. This helps to restructure one's thinking, switching a focus on the negative (weaknesses) to a focus on the positive (strengths). This helps to counter demeaning self-perceptions and raise self-esteem by teaching those with AD/HD to think/say 'I can' instead of 'I can't'. Visualisation techniques may be used to imagine successful completion of tasks and appropriate thinking styles can be modelled by therapists.

Social skills training is another important component of the psychosocial interventions available, enabling an AD/HD individual to interact more successfully with those around them. This may include teaching good manners, how to greet someone, how to apologise, how to ask for and offer help, and how to pay and receive a compliment. This training can also teach how to respect boundaries and become more adept at noticing behavioural cues, for example, when it is appropriate to interrupt somebody. Again, therapists can teach responsibility, empathy and problem-solving by practising/modelling these skills.

All of these interventions may be tried with the AD/HD individual, their families, their educators and their peers, with each intervention individually tailored to suit the audience. For example, family therapy consisting of both behavioural and cognitive strategies may be used to educate parents about techniques of behaviour management, problem solving and more effective communication skills. It commonly encourages parents to avoid expressing criticism or hostility, and instead to express warmth and praise. Likewise, classroom interventions can be developed for use by teachers which may include: seating the AD/HD student at the front of the class to minimise distraction; surrounding them with good role models; making instructions clear, concise and simple; assigning one task at a time; delivering lessons briskly; varying voice tone and movements; using stimulating materials such as visual aids; encouraging the AD/HD student to write down homework assignments; allowing extended time for tests/exams; giving consistent and frequent rewards; allowing alternative test delivery, e.g. orally or on a computer; avoiding unpredictable transitions, i.e. giving clear warning of change; monitoring break times to spot bullying; reminding privately any AD/HD student on medication to take this, i.e. not in front of their peers; ignoring minor disruptions; planning the consequences for negative behaviour and administering them immediately when the behaviour is apparent; avoiding ridicule and criticism; educating other children about AD/HD; and, communicating with parents. Clearly, many of these strategies will be useful to parents too, as well as future educators and employers.

Another psychosocial approach to the treatment of AD/HD is coaching. This is an individualized and action-orientated service offered by a professional (often a trained counsellor, although not always), whose aim is to encourage their clients to be the best they can be personally and professionally. Coaches aim to assist AD/HD clients to improve performance of specific tasks and enhance the quality of their lives. This is achieved by identifying goals, by encouraging action and movement, and by providing structure and accountability. Coaches will guide clients into actions that (re)build self-esteem, self-awareness and self-regulation, and focus on clients' executive functioning skills to create customised systems that will improve consistency and effectiveness. For example, an AD/HD coach may look at a client's weekly timetable to help the client identify possible AD/HD pitfalls, and plan ways to circumvent those pitfalls. They also help clients to identify their positive AD/HD characteristics and to appreciate their strengths. They aim to nurture personal awareness and responsibility, and encourage clients to look for options that lead to progress and success. AD/HD coaches may offer a focus on school performance, relationships, family life, organisational skills, career choices, job satisfaction and financial stability.

Support groups are also available to AD/HD individuals and their families. These offer a forum in which people can gain emotional support from others in a similar situation. They are commonly run by parents of those with AD/HD, and may also provide practical help, information, guidance and feedback about AD/HD, as well as alternative methods of coping. Both the UK and the USA have large national charitable organisations that fulfil a similar role ('ADDISS' and 'CHADD'), as well as countless smaller local organisations run by teams of dedicated individuals who meet regularly to share their AD/HD-related thoughts, feelings and concerns.

As far as other types of psychosocial treatment for AD/HD are concerned, Hill and Taylor (2001) have reported that there is no scientific evidence for the effectiveness of homeopathy, psychoanalytic psychotherapy, naturopathy or cranial osteopathy.

Since it has been argued by some that nutrition plays a role in the cause of AD/HD, it follows that a nutritional treatment would be recommended, i.e. the exclusion of these things. Although there is some research that suggests some children benefit from this approach (Dykman and Dykman, 1998; Kaplan et al, 1989; National Institute of Mental Health, 2003; Weiss et al, 1980) there are no well-established nutritional interventions that have consistently demonstrated efficacy for assisting the majority of children with AD/HD. This form of dietary manipulation is a time-consuming process and more research is required to ascertain the characteristics of the small number of children who respond to this approach.

There is very good evidence for the effectiveness of using a combination of two or more of the other therapies already discussed, particularly from a large clinical trial carried out by Jensen et al (2001) for the US National Institute for Mental Health (NIMH). This involved over 150 research staff in multiple centres throughout the USA. Almost 600 AD/HD children and their families were randomised to one of four treatment conditions: medication alone; behavioural treatment alone involving the parents; medication and behavioural treatments combined; and community 'business as usual' treatment. The greatest improvements were seen in those who received a *combination* of medical and behavioural treatments, followed closely by the medical treatment alone. It also found that parents often wanted more than just medication for their children, favouring the combined treatment condition over the others, followed by the behavioural treatment alone, perhaps because they felt that they were actually doing something practical to help their child (see also previous reports on this research – MTA Co-operative Group, 1999a/1999b).

In recognition of these findings, it is likely that optimally a multi-modal, multi-agency approach should be followed, involving parents, teachers and health professionals in psychoeducation, counselling, supportive problem-directed therapy, behavioural interventions, coaching, cognitive remediation, couples and family therapy, and medication management (Hill and Taylor, 2001; Jensen et al, 2001; Szymanski and Zolotor, 2001; Wender et al, 2001). Multimodal and pharmacological treatments which lasted longer than one year were found to be better than those which lasted for less than one year (Paule et al, 2000). Such treatments should be monitored closely with a focus on the absence or presence of side-effects and changes to AD/HD symptoms. It is recognised that this type of approach to the treatment of AD/HD is costly and requires considerable collaboration of health professionals, educators and families all working together. It also requires some years of specialised training to become proficient at effective multimodal treatment. Unfortunately, in the UK Hill and Taylor (2001) noted that standards of good practice in treating AD/HD vary between practitioners. This was a view echoed by Baverstock and Finlay (2003) who found that those caring for young adults with AD/HD are still fairly unfamiliar with the condition. They also recommended that guidelines are required urgently to establish handover from paediatric to adult care.

7. Childhood correlates of AD/HD

A number of comorbid psychiatric conditions have been reported with childhood AD/HD. Indeed, it is much more common to find a child with AD/HD and a comorbidity, than it is to find a child with AD/HD alone. Kadesjo and Gillberg (2001) found that, in a population sample of Swedish children with AD/HD, the prevalence rate for another single comorbidity was 87 per cent, whilst the prevalence rate for two or more comorbidities was 67 per cent. The most common comorbid conditions reported have been antisocial disorders, especially conduct disorder (CD) and oppositional defiant disorder (ODD) (August et al, 1998; August et al, 1999; Biederman et al, 1991; Faraone et al, 2000c; Lavigne et al, 2001; Kadesjo and Gillberg, 2001). August et al (1999) found that the presence of comorbid antisocial disorders could often be linked to mothers' psychiatric disorders or to negative parenting practices.

A spectrum of developmental and learning disorders has been found to be associated with children with AD/HD. These include: reading disabilities (such as dyslexia); dysgraphia; dyscalculia; dyspraxia; and Tourette's syndrome and tic disorders (Barkley, 1990; Biederman et al, 1991; Faraone et al, 1993; Kaplan et al, 2000; Peterson et al, 2001; Rabiner and Coie, 2000; Richards, 1995; Semrud-Clikeman et al, 1992; Shaywitz et al, 1994; Spencer et al, 1993). Until recently presence of pervasive developmental disorder, i.e. autism, was thought to rule out a diagnosis of AD/HD. However, research has demonstrated that the two can be diagnosed as separate conditions in the same individual (e.g. Blakemore-Brown, 2002; Santosh, 2004). Aside from this, it has been shown that other syndromes on the autistic spectrum such as Asperger's syndrome can co-occur with AD/HD (Ehlers and Gillberg, 1993; Ghaziuddin et al, 1998).

Other comorbid conditions reported to exist with AD/HD include: anxiety and/or mood disorders (i.e. depression) (Biederman et al, 1991; Lavigne et al, 2001; Russo and Beidel, 1995); borderline personality disorder (BPD) (Faraone et al, 1997; Faraone et al, 2001); obsessive-compulsive disorder (OCD) (Peterson et al, 2001); and bipolar disorder (BD) and/or manic-like symptoms (Biederman et al 1996; Kent and Craddock, 2003; Wilens et al, 2003a). Other childhood correlates reported with AD/HD have been social impairment, peer relationship difficulties and rejection, perhaps as a result of antisocial characteristics (Bagwell et al, 2001; Erhardt and Hinshaw, 1994; Greene et al, 2001), sleep difficulties (Ball and Koloian, 1995), enuresis (Biederman et al, 1995), and educational underachievement (Erhardt and Hinshaw, 1994).

Some have argued that instead of co-existing with AD/HD, conditions such as depression or learning problems may be responsible for apparent AD/HD symptomatology. The problem of correlates again raises questions about AD/HD as a pure disorder, and whether the cause(s) of AD/HD can be separated from the cause(s) of comorbid conditions. Current expert opinion is that it is uncommon to find 'pure' AD/HD, but that it is also rare to find 'pure' forms of other childhood disorders. It is argued we are all individuals with different potentialities exposed to different environments and life events, and as such we are bound to experience a multiplicity of problems at any one time. Just because a condition appears to be complex, this is no reason not to attempt to unravel and understand it. Although a full understanding of AD/HD is some way off, this is no reason to give up striving towards it; in fact, a great deal of headway is being made in this area and it is clear that certain conditions cluster with AD/HD, indicating that the aetiology of these conditions are perhaps related, and that the genes and brain pathways responsible may be similar. There is little doubt that comorbidity complicates research into the identification and treatment of AD/HD.

8. Outcomes: evidence from the literature

In this section we summarise the literature that describes the short-term outcomes of childhood AD/HD in adolescence, followed by the longer-term outcomes seen in adulthood. We cite studies with longitudinal designs that exemplify the main findings, and end the section with an evaluation of this research.

8.1 Adolescent outcomes of AD/HD

There are some who believe that children grow out of AD/HD, which they also claim is proof against the existence of a real disorder. If this is the case, there should be no differences in the function and performance of adolescents (and adults) who did and did not have childhood diagnoses of AD/HD. We would also expect little evidence of the persistence of AD/HD symptoms beyond childhood. However, given the aetiology, characteristics and correlates of childhood AD/HD, and the range of problems faced by children with AD/HD, it would be very surprising if it had no impact on adolescence (and adulthood), and if all symptoms did disappear.

A wide body of research has demonstrated that the problems of childhood AD/HD do in fact continue into adolescence. A number of follow-up studies have been conducted, lasting anything from four to eight years, confirming that AD/HD symptoms do persist beyond childhood (Barkley et al, 1990; Barkley et al, 1991; Biederman et al, 2000b; Faraone et al, 2002; Fischer et al, 1990; Fischer et al, 1993b; Mannuzza et al, 1991; Taylor et al, 1996). An early study by Barkley et al (1990) showed that 88 per cent of their childhood sample still met criteria for AD/HD eight years later. From a review of of the older prospective studies, Hill and Schoener (1996) concluded that children aged between 7- and 10-years-old had approximately a 40 per cent chance of still having AD/HD by age 13, which fell to a 15-30 per cent chance of AD/HD by age 18. However, recent studies have suggested that a larger proportion of child samples continued to experience AD/HD into adolescence, in the region of up to 70 per cent (August et al, 1998). Beiderman et al (2000b) argued that differences in remission rates reported in studies of the persistence of AD/HD into adolescence had more to do with the differing definitions used rather than with the disorder's natural course. However, it is widely observed that symptoms of hyperactivity decrease with age whilst symptoms of inattention persist (Hart et al, 1995; Levy et al, 1996). This may be a sign of normal maturation since this is a pattern common to all individuals. AD/HD does however persist into adolescence independently of the presence of other conditions, such as conduct disorder (e.g. Taylor et al, 1996).

Comorbid behavioural problems, and antisocial and conduct disorders have been shown to be continuing problems in adolescence in AD/HD children (Bagwell et al, 2001; Barkley et al, 1990; Biederman et al, 2001; Faraone et al, 2002; Fischer et al, 1993a; Fischer et al, 1993b; Molina and Pelham, 2003; Taylor et al, 1996). This may lead to contact with the criminal justice system (Mannuzza et al, 1989; Satterfield et al, 1982). Mood and anxiety disorders (Faraone et al, 2002; Mick et al, 2000; Rucklidge and Tannock, 2001), bipolar disorder (Biederman et al, 2001), OCD (Petereson et al, 2001) and general psychiatric problems are also found more commonly in adolescents diagnosed as AD/HD children (Goldstein and Brzostek, 2000; Taylor et al, 1996). Adolescent use of alcohol, cigarette and illegal substances is also encountered more commonly in those who had AD/HD as children (Barkley et al, 1990; Biederman et al, 2001; Daley, 2004; Molina and Pelham, 2003; Rohde et al, 2004; Tercyak et al, 2002), but these may be mediated by the presence of conduct disorder.

Aside from behavioural and psychiatric problems, children with AD/HD are also more likely to show poorer academic achievement or impaired cognitive skills when they reach adolescence (Barkley et al, 1990; Faraone et al, 1998; Faraone et al, 2002; Fergusson and Horwood, 1995; Fischer et al, 1990; Minde et al, 1972; Rucklidge and Tannock, 2001). Fischer et al (1990) argued that this may be the result of behavioural disinhibition since those without hyperactivity or

conduct problems may achieve better outcomes. Less information is available about females, although in adolescence those with childhood AD/HD studied by Rucklidge and Tannock (2001) were more likely to develop an adverse external locus of control and poorer attributional styles compared with those females without childhood AD/HD.

Research has also been conducted on the families of adolescents who had childhood AD/HD. The families were found to be less stable and characterised by more conflict (Barkley et al, 1990; Biederman et al, 2001). Mothers have been described as more negative and controlling (Barkley et al, 1991) and as having had more psychological problems (Barkley et al, 1991). Also, family discord, maternal depression, and hostile and inconsistent parenting were factors strongly associated with the persistence of AD/HD (e.g. Taylor et al, 1996).

8.2 Adult outcomes of AD/HD

Findings on adult outcomes of childhood AD/HD are available from some of the studies that followed children into adolescence and which continued to follow-up samples into young adulthood, typically over time spans ranging from eight to fifteen years. These studies, and the articles which review them, confirm that numerous adverse adult outcomes are evident in those who developed AD/HD in childhood, continuing the trend emerging in childhood and adolescence.

AD/HD symptoms of impulsivity, restlessness and inattention often persist into adulthood (Faraone et al, 2000e; Fischer et al, 2002; Mannuzza and Klein, 2000; Mannuzza et al, 1993; Mannuzza et al, 1998; Rasmussen and Gillberg, 2000; Weiss et al, 1985; Wender et al, 2001). The proportion affected in adulthood reported in these studies vary greatly (e.g. 4-60 per cent), the differences in prevalence rates are probably due to the different methods used for identifying adult AD/HD. Research shows that the symptoms of AD/HD decline with age, yet that a minority of individuals continue to have problems into early adulthood. Whilst this may be true, it may also be the case that the real instances of adult AD/HD are being missed because researchers are using diagnostic criteria originally developed for children, such as the DSM-IV. These might miss the subtler impairments in executive function experienced by AD/HD adults. Adults with AD/HD may have become adept in different degrees at masking their symptoms in order to cope with day-to-day life. Some may also seek out environments that suit their behaviour. Thus, more sophisticated methods of recognising adult AD/HD need to be developed. Indeed, there are some researchers who have developed, and are developing, diagnostic criteria and tools for assessing adult AD/HD (e.g. Brown, 1996). To date there is little good quality longitudinal research that has used such new adult scales.

A subgroup of those with childhood AD/HD are found in adulthood to exhibit antisocial behaviour, or have antisocial personality disorder (Faraone et al, 2000e; Fischer et al, 2002; Greenfield et al, 1988; Hechtman and Weiss, 1983; Hechtman and Weiss, 1986; Herrero et al, 1994; Mannuzza and Klein, 2000; Mannuzza et al, 1993; Mannuzza et al, 1998; Rasmussen and Gillberg, 2000; Weiss et al, 1985). The strongest associations with antisocial behaviour were found with combined type AD/HD (Faraone et al, 1998). However, Hechtman and Weiss (1986) did not find any significant association between childhood AD/HD and adult conduct disorder (CD) or oppositional defiant disorder (ODD) in one of their Canadian studies.

Childhood AD/HD has been reported as predictive of higher levels of contact with the police during adulthood (Hechtman et al, 1984a; Mannuzza and Klein, 2000; Mannuzza et al, 1993; Rasmussen and Gillberg, 2000). Babinski et al (1999) claimed that the association with adult crime was only true for males and not females, and that it was predominantly childhood symptoms of hyperactivity-impulsivity, rather than inattention, which contributed to the risk of criminal activity. They thought that this might have been a result of adult comorbid antisocial problems or early conduct problems but Farrington (1995) suggested that AD/HD itself was one of the single biggest risk factors for delinquency, reporting that a sizeable proportion of the prison population meets the diagnostic criteria for AD/HD. This contrasted with an earlier report by

Hechtman et al (1984a), who found that although the group with childhood AD/HD that they followed-up were more likely to be engaged in criminal activity than the control group, this was true only for the five years prior to follow-up but not for the preceding year. They opined that the outcome is thus more encouraging than they had expected.

Some research found that major depressive disorder was a common feature of adults who had childhood AD/HD (Faraone et al, 2000e; Fischer et al, 2002). This may be linked to inattentive type AD/HD (Faraone et al, 1998) or to the presence of childhood/adolescent conduct disorder (Fischer et al, 2002). In contrast, other research has reported no evidence of a link between childhood AD/HD and anxiety and mood disorders in adulthood (Mannuzza et al, 1993; Mannuzza et al, 1998). Other adult psychiatric outcomes predicted by AD/HD include bipolar disorder and tic disorders, both of which were reported after combined type AD/HD (Faraone et al, 1998), and OCD (Petereson et al, 2001). An association was also reported with personality disorders, although these were also linked to comorbid childhood conduct disorder (Fischer et al, 2002). Adults diagnosed with AD/HD in childhood were more likely to have smoked cigarettes (Lambert and Hartsough, 1998), abused alcohol (Ercan et al, 2003; Greenfield et al, 1988; Hechtman et al, 1984a; Rasmussen and Gillberg, 2000) and drugs (Hechtman et al, 1984a; Mannuzza et al, 1993; Mannuzza et al, 1998; Mannuzza and Klein, 2000). Mannuzza et al (1993) found that drug abuse in adulthood was more common in those whose AD/HD symptoms had persisted, a finding not shown earlier by Hechtman and Weiss (1986). Biederman et al (2000a) suggested that adult drug abuse is related more to parental substance abuse than to childhood AD/HD. It is worth repeating at this juncture that research has consistently shown that stimulant medication for childhood AD/HD is not associated significantly with adult drug use or dependence, and in fact may even protect against it (Barkley et al, 2003; Biederman, 2003; Wilens et al, 2003b).

Psychiatric problems such as generalised stress or anxiety have been reported in some studies as adult outcomes of childhood AD/HD (Barkley, 2002b; Fischer et al, 2002; Mannuzza et al, 1998). These may be linked to comorbid childhood conduct disorder and female gender (Dalsgaard et al, 2002). Problems have also been reported with social skills, emotional difficulties (Greenfield et al, 1988; Hechtman et al, 1980; Hechtman and Weiss, 1986; Hechtman and Weiss, 1983; Mannuzza and Klein, 2000) and low self-esteem (Hechtman et al, 1980; Mannuzza and Klein, 2000). These may have had an impact on the unsatisfactory relationships that were also often experienced by adult 'survivors' of childhood AD/HD (Barkley, 2002b), who become parents earlier, have smaller gaps between child-bearing, and suffer marital discord and frequent relationship breakdown.

When followed up in adulthood, individuals with childhood AD/HD have been reported to be more likely to experience academic underachievement and fewer years completed schooling (Barkley, 2002b; Hechtman and Weiss, 1983; Mannuzza and Klein, 2000; Mannuzza et al, 1993; Mannuzza et al, 1997; Rasmussen and Gillberg, 2000; Weiss et al, 1979; Weiss et al, 1985). Language and literacy problems have also been reported (Faraone et al, 1998; Rasmussen and Gillberg, 2000). This may in part explain why those with childhood AD/HD have been found to be more likely as adults to be in lower-ranking occupations and possess a lower social class than control groups (Barkley, 2002b; Faraone et al, 2000e; Mannuzza and Klein, 2000; Mannuzza et al, 1993; Mannuzza et al, 1997; Mannuzza et al, 1998).

Finally, some follow-ups have shown that adults who had childhood AD/HD were more likely to have problems driving or to have experienced more road traffic accidents (Barkley, 2002b; Barkley et al, 1993; Faraone et al, 2000e; Hechtman et al, 1984b). Possible explanations have been invoked such as symptoms of inattention, low tolerance of frustration, and impulsivity. Interestingly, one study of those who received medication during childhood for their AD/HD had fewer car accidents as adults than those who went untreated (Hechtman et al, 1984b).

Although this summary points to a comparatively gloomy future for those diagnosed with AD/HD in childhood, some research teams were keen to emphasise that there could be a more promising adult outcome than had previously been expected, such as Hechtman et al (1984a). Likewise, in a recent study by Mannuzza and Klein (2000) nearly all those followed-up were gainfully employed, and some had higher-level education and occupations. The authors concluded that although AD/HD children as a group fared poorly compared to those without AD/HD, two-thirds of the children showed no evidence of any mental disorder in adulthood. Many did not exhibit clinically significant emotional or behavioural problems once in their mid-twenties; an important point to bear in mind.

8.3 Caveats of the outcome literature

In spite of all the studies reviewed above, there is a dearth of objective longitudinal research into the outcomes of AD/HD, and a number of criticisms can be levelled at existing studies. For instance, none represented the results of a nationally representative cohort. The generalisability of these results are therefore questionable. Many studies suffer from a sample selection bias, having been based on clinic populations, who may be considered to be more severely ill (e.g. Barkley et al, 2003; Dalsgaard et al, 2002; Mannuzza et al, 1993; Mannuzza et al, 1998). Samples are also often gender biased, with an exclusive focus on males (e.g. Mannuzza et al, 1993; Taylor et al, 1991). Many of the outcome findings have also shown an ethnocentric and geographic bias, being largely based on North American samples. Sample sizes have rarely reached numbers beyond 200 participants, and when groups of control participants were included, these were often smaller in number (e.g. Barkley et al, 1990; Fischer et al, 1990; Molina and Pelham, 2003) and differed significantly from the clinical group at baseline in spite of occasional efforts to match them. Thus the control group used for comparison in the study reported by Mannuzza et al (1998) were significantly younger and had a significantly lower SES than the clinical group. Most of the studies that use control groups compare clinic-referred cases of AD/HD to individuals selected from the general population, so confounding the causes of AD/HD with the reason for referral (e.g. Fischer et al, 1993b). Small sample sizes and questionable control group characteristics have thus presented problems for statistical inference and generalisability.

The longer-term follow-up studies conducted have rarely reported on individuals beyond their mid-twenties, and some reported on groups of individuals with wide age ranges (e.g. Barkley et al, 2003; Dalsgaard et al, 2002; Mannuzza et al, 1997) that might mask important developmental differences. Attrition to samples is a problem in any long-term research and in some AD/HD-related research this has been high (e.g. at follow-up the sample studied by Weiss et al (1985) had suffered 40 per cent attrition). It is possible that those lost to follow-up represent those who have the most negative outcomes, and longitudinal research may therefore underestimate the outcomes of childhood AD/HD.

Problems may also be encountered in ascertaining the rate of persistence of AD/HD into adulthood, since it has not been possible to get good multi-method assessments of AD/HD that include ratings by employers. Not surprisingly, some individuals would prefer for their employers not to know about their possible AD/HD status. Faraone et al (2000e) stated that the ambiguity in follow-up of AD/HD children was probably due to how to define or diagnose AD/HD in adulthood. For example, Barkley (2002b) warned against follow-up studies that rely on adult self-report of AD/HD symptoms, which would underestimate the persistence of AD/HD into adulthood.

The longitudinal research that exists on the outcomes of AD/HD has also rarely included controls for confounding factors or the pre-existing and background characteristics of those with childhood AD/HD, to truly isolate the effects of AD/HD (e.g. Mannuzza et al (1998) controlled only for parental SES). It is always possible in these circumstances that the differences observed at follow-up may be due to some feature other than AD/HD, not measured at baseline.

It is difficult, therefore, to compare findings across the longitudinal studies which have been reported owing to the varying methodologies utilised. Often, varying definitions of AD/HD have been used, usually the product of the historical changes that have occurred to the diagnostic criteria and accepted nosology. Those with the longest follow-up are based mainly on samples of 'hyperactives' (e.g. Barkley et al, 2003; Fischer et al, 2002; Mannuzza et al, 1998; Weiss et al, 1985) and thus the outcome of those with symptoms primarily of inattention is unknown. This is something that Barkley (2002b) recognised, stating that future research should concentrate on outcomes for females and those with predominantly inattentive type AD/HD. Lilienfeld and Waldman (1990) also stated that most research up to that time in this area had been on those who were hyperactive and aggressive, and that child conduct problems may have been a major confounding factor in AD/HD research. Varying methods of data collection have also been used (e.g. trained undergraduate interviewers versus mental health professional interviewers) and in some cases, follow-up interviewers have not been blind of the research participants' baseline AD/HD status (e.g. Weiss et al, 1985). This could obviously have biased their perception of the adult functioning of clinical groups compared to control groups.

Even more of the criticisms levelled here at the longitudinal and experimental research may also be levelled at the cross-sectional studies. Much of these have been ethnocentric, geographically and gender biased, and carried out with small samples of individuals from clinic populations with combined or hyperactive type AD/HD. Cross-sectional research suffers the added drawback that it offers correlational rather than causal relationships.

It is clear that future research should aim to document the experience of AD/HD in females as well as males, include those with inattentive type AD/HD, and where possible be based on larger numbers of individuals from representative samples. Those with prospective designs will be better at teasing out causal pathways, and those that aim to isolate the long-term effects of AD/HD would benefit from the collection of a wealth of information at baseline and each follow-up enabling pre-existing characteristics to be controlled for. Such research will be possible using the data contained in at least two of the British national cohort studies, namely the 1958 National Child Development Study (see Shepherd, 1985) and the 1970 British Cohort Study (see Butler et al, 1986).

9. Summary and conclusions

The aim of this report has been not only to review the extensive and often confusing literature on AD/HD but also to consider ways in which this can be remedied by truly longitudinal research, particularly the potential for secondary analyses of large-scale longitudinal cohort data to advance knowledge about AD/HD. This review has: provided an overview of what AD/HD is; considered the current debates surrounding the conceptual and measurement issues associated with AD/HD; and reviewed the recent literature with a specific focus on the lifecourse outcomes for individuals diagnosed with AD/HD. In spite of great strides made by research focused on aetiology and outcomes, and the progress made in assessment, diagnosis and treatment, AD/HD remains a controversial disorder in some sectors. It appears that this is primarily due to frequently expressed opinions that are rarely based on sound scientific evidence. This can certainly cause confusion amongst families, teachers, policy-makers and even health care professionals. Available evidence, based on high quality research, including double-blind randomised controlled cross-over designs or sophisticated brain imaging techniques, has demonstrated that AD/HD is a very real and, if left untreated, disabling condition.

Our review shows AD/HD to be a disorder of childhood characterised by inattention, hyperactivity and impulsivity. It represents the extreme end of a continuum of symptoms distributed throughout the general population. It is a disorder of heterogeneous causes, including biological, environmental and psychosocial factors, although a genetic susceptibility seems to be the most common. Brain imaging studies have implicated dorsolateral regions of prefrontal cortex, as well as dysfunctional circuitry to/from/in the basal ganglia, cerebellum and locus coeruleus; in some instances the brains of those with AD/HD have different asymmetries, size, activity and response levels than the brains of those without AD/HD. Molecular genetic studies have implicated the dopamine pathways that modulate and integrate the neural activity of the networks connecting these brain regions. There is a high heritability for AD/HD (0.80-0.98 in monozygotic twins), and independent DNA studies have found moderate associations with dopamine and norepinephrine genes. But AD/HD is not a simple disorder of single cause, where an aberrant gene is all that needs to be identified. It is clear that multiple genes are working together in complex ways, which are still not understood fully. In addition, neurochemistry has a long way to go in understanding the role that hormones have to play in the expression of this condition. Research using larger samples is required to gain a better understanding of the exact nature of familial transmission.

A number of environmental and social factors have already been identified in the aetiology of AD/HD, including obstetric complications (perhaps caused by maternal smoking during pregnancy), low socio-economic status backgrounds and family conflict. It is unclear to what extent these factors have an impact, and psychosocial context and non-genetic familial influences need further investigation. It is possible that certain symptoms or subtypes of AD/HD have different causes, and that males and females are affected in different ways. Further research is required to explore these possibilities.

It is recommended that AD/HD is best identified using multiple assessment methods alongside well-researched and validated diagnostic criteria such as that supplied in the DSM-IV. These criteria require that a certain level of disability is evident from a certain age, for a minimum length of time, in multiple life domains. Such an assessment should make use of information supplied by a variety of people, in order to ensure that conditions which ‘look like’ AD/HD can be ruled out, or ruled in, as the case may be. Like any developmental disorder, there will be problems with diagnosis but the tools developed for AD/HD are as good as those for any other child behavioural problem. In addition, that we find something difficult to measure is no reason to claim it does not exist and we must strive for better methods of identification, as we do in any other area. Our diagnostic criteria are never set in stone and can only represent our understanding at the time; as our understanding of AD/HD changes, so our diagnostic criteria must change also. It would help if there was consistency in the use of our diagnostic criteria, in both research and clinical practice,

and the recent convergence in ICD-10 and DSM-IV criteria may go some way towards this. In addition, there are many researchers in the USA who have developed very good protocols for the diagnosis of AD/HD.

Another characteristic of AD/HD is that it is common to find a number of co-existing conditions, such as antisocial disorders (e.g. conduct disorder), developmental and learning disorders (e.g. dyslexia, Asperger's syndrome), depression and anxiety. These are all considered to be endogenous, stand-alone conditions, which may or may not have a similar underlying cause to AD/HD but which certainly merit treatment in their own right. However, there are some unanswered questions. Research has not yet managed to distinguish which conditions may have a similar underlying cause and whether there are further subtypes or expressions of AD/HD yet to be categorised, such as AD/HD with a tic disorder, or AD/HD with mania. These are areas in which further research is required.

Stimulants have been used successfully in the treatment of AD/HD, although not all those with AD/HD respond in a similar way and some prefer not to take medication. It is still not clear what the basic mechanisms are by which stimulants exert control, and this too is an area for further research. Given that AD/HD is not a uni-dimensional disorder, it does not lend itself to a universal treatment package, such as medication alone. Thus, recommendations are made for multimodal approaches to treatment, and those that have been shown to be the most effective combine medication with psychosocial interventions that are implemented over the long-term. But the problem here is the exact definition of a multimodal approach. It appears that it is a package tailored to suit the needs of each individual but this is, unfortunately, costly to deliver. There are still too few professionals involved in the treatment of AD/HD who are well-trained in the design and delivery of sophisticated psychosocial interventions working in conjunction with carefully monitored medication. Such treatment requires good channels of communication between different partners, flexibility of services, collaboration and careful planning, whilst avoiding fragmentation and duplication of service. This may be particularly difficult to achieve in the UK health profession where services are continually being restructured. There is also massive underfunding in Britain's mental health services, especially those specific to children and adolescents, yet the cost to society of not treating AD/HD, in terms of delinquency and crime, is high (Farrington, 1995; Taylor, 2001). However, sophisticated, multimodal treatment is not unattainable and there is clear evidence that there are services throughout North America that are delivering this. This is an area that requires future focus in Britain. We need to consider routine screening in primary care, better methods of handling the changeover from child/adolescent services to adult services, and raised awareness of AD/HD amongst the education, criminal justice, prison and probationary services. We need better systems in place to implement the guidelines for treating AD/HD, which are born of sound research, and better systems for encouraging collaboration across disciplines.

The weight of the longitudinal evidence summarised in this report suggests that a number of negative adolescent and adult outcomes await those with childhood AD/HD. These include the continuing problems of AD/HD symptoms and encompass problems in life domains such as mental health, education, economic status and relationships. However, the longitudinal research conducted to date has suffered from a number of problems, for example: male bias; ethnocentricism; small sample sizes; samples selected from clinic populations; bias towards those with hyperactive and combined subtypes; and few, if any, controls for background characteristics and confounding factors. Problems continue to exist in the identification of AD/HD symptoms in adults and few studies are employing the recently developed adult diagnostic scales. These are things to bear in mind for future research into adult functioning, which is necessary if we are to understand properly the longer-term impact of AD/HD on the lifecourse and isolate the effects of AD/HD from other characteristics.

Thus, we recommend that further research be conducted. Ideally, research on outcomes needs data containing characteristics of those with AD/HD in childhood and adulthood. Considerable

advancement and knowledge about outcomes would be gained from the availability and analysis of longitudinal nationally representative large-scale samples of individuals with and without AD/HD, containing sizeable numbers of males and females. The analyses should aim to isolate the long-term effects of AD/HD by controlling for the myriad background characteristics that may give rise to AD/HD, so that more reliable conclusions can be drawn. The untapped potential that exists in Britain's national birth cohort studies (e.g. the 1958 National Child Development Study – Shepherd, 1985; the 1970 British Cohort Study – Butler et al, 1986) may offer a step in the right direction towards a better understanding of the lifecourse and outcomes of AD/HD.

References

American Psychiatric Association (1994) *Diagnostic and statistical manual of mental disorder, 4th Edition*, Washington DC: American Psychiatric Association.

Anderson, S. L. and Teicher, M. H. (2000) 'Sex differences in dopamine receptors and their relevance to ADHD', *Neuroscience and Biobehavioral Reviews,* 24: 137-141.

Anon. (2000) *The AD/HD Debate*, 21 October.
Available URL: http://www.epinions.com/kifm-review-1B94-1DB82B5-39F1C928-prod1. Viewed 19 December 2003.

August, G. J., Braswell, L. and Thuras, P. (1998) 'Diagnostic stability of AD/HD in a community sample of school-aged children screened for disruptive behaviour', *Journal of Abnormal Child Psychology*, 25: 345-356.

August, G. J., Realmuto, G. M., Joyce, T. and Hektner, J. M. (1999) 'Persistence and desistance of oppositional defiant disorder in a community sample of children with AD/HD', *Journal of the American Academy of Child and Adolescent Psychiatry*, 38: 1262-1270.

Babinski, L. M., Hartsough, C. S. and Lambert, N. M. (1999) 'Childhood conduct problems, hyperactivity-impulsivity and inattention as predictors of adult criminal activity', *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 40: 347-355.

Bagwell, C. L., Molina, B. S., Pelham, W. E. J. and Hoza, B. (2001) 'Attention-deficit hyperactivity disorder and problems in peer relations: predictions from childhood to adolescence', *Journal of the American Academy of Child and Adolescent Psychiatry*, 40: 1285-1292.

Ball, J. D. and Koloian, B. (1995) 'Sleep patterns among ADHD children', *Clinical Psychology Review,* 15: 681-691.

Barkley, R. A. (1989) 'Hyperactive girls and boys: stimulant drug effects on mother-child interactions', *Journal of Child Psychology and Psychiatry*, 30: 379-390.

Barkley, R. A. (1990) *Attention Deficit Hyperactivity Disorder: a handbook for diagnosis and treatment*, New York: Guilford Press.

Barkley, R. A. (1997) 'Behavioral inhibition, sustained attention, and executive function: constructing a unifying theory of AD/HD', *Psychological Bulletin,* 121: 65-94.

Barkley, R. A. (1998) *Attention Deficit Hyperactivity Disorder: a handbook for diagnosis and treatment, 2nd Edition*, New York: Guilford Press.

Barkley, R. A. (2002a) International consensus statement on AD/HD, January 2002, signed by over 70 leading scientists, *Clinical Child Family Psychology Review*, 5: 89-111.

Barkley, R. A. (2002b) Major life activity and health outcomes associated with attention deficit/hyperactivity disorder, *Journal of Clinical Psychiatry*, 63: 10-15.

Barkley, R. A. (2002c) 'Psychosocial treatments for attention-deficit/hyperactivity disorder in children', *Journal of Clinical Psychiatry,* 63 (Suppl 12): 36-43.

Barkley, R. A., Fischer, M., Edelbrock, C. S. and Smallish, L. (1990) 'The adolescent outcome of hyperactive children diagnosed by research criteria: I. An 8-year prospective follow-up study', *Journal of the American Academy of Child and Adolescent Psychiatry*, 29: 546-557.

Barkley, R. A., Fischer, M., Edelbrock, C. S. and Smallish, L. (1991) 'The adolescent outcome of hyperactive children diagnosed by research criteria: III. Mother-child interaction, family conflict and maternal psychopathology', *Journal of Child Psychology and Psychiatry*, 32: 233-255.

Barkley, R. A., Fischer, M., Smallish, L. and Fletcher, K. E. (2002) 'The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder', *Journal of Abnormal Psychology*, 111: 279-289.

Barkley, R. A., Fischer, M., Smallish, L. and Fletcher, K. E. (2003) 'Does the treatment of attention-deficit/hyperactivity disorder with stimulants contribute to drug use/abuse? A 13 year prospective study', *Pediatrics*, 111: 97-109.

Barkley, R. A., Grodzinsky, G. and DuPaul, G. (1992b) 'Frontal lobe functions in attention deficit disorder with and without hyperactivity: a review and research report', *Journal of Abnormal Child Psychology,* 20: 163-188.

Barkley, R. A., Guevremont, D. C., Anastropoulos, A. D., DePaul, G. J. and Shelton, T. L. (1993) 'Driving-related risks and outcomes of attention deficit hyperactivity disorder in adolescents and young adults: a 3-5 year follow-up survey', *Pediatrics,* 92: 212-218.

Barkley, R. A., Guevremont, D. C., Anastopoulos, A. D. and Fletcher, K. E. (1992a) 'A comparison of three family therapy programs for treating family conflicts in adolescents with attention-deficit hyperactivity disorder', *Consulting Clinical Psychology,* 60: 450-462.

Barry, R. J., Johnstone, S. J. and Clarke, A. R. (2003) 'A review of electrophysiology in attention-deficit/hyperactivity disorder: II. Event-related potentials', *Clinical Neurophysiology*, 114: 184-98.

Baumgaertal, A., Wolraich, M. and Dietrich, M. (1995) 'Comparison of diagnostic criteria for AD/HD in a German elementary school sample', *Journal of the American Academy of Child and Adolescent Psychiatry,* 34: 423-431.

Baverstock, A. C. and Finlay, F. (2003) 'Who manages the care of students with Attention Deficit Hyperactivity Disorder (AD/HD) in higher education?', *Child: Care, Health and Development*, 29: 163-166.

Bhutta, A. T., Cleves, M., Casey, P. H., Cradock, M. M. and Anand, K. J. (2002) 'Cognitive and behavioural outcomes of school-aged children who were born preterm: a meta-analysis', *JAMA: the Journal of the American Medical Association*, 288: 728-737.

Biederman, J. (2003) 'Pharmocotherapy for attention-deficit/hyperactivity disorder (AD/HD) decreases the risk for substance abuse: findings from a longitudinal follow-up of youths with and without AD/HD', *Journal of Clinical Psychiatry*, 64: 3-8.

Biederman, J., Faraone, S. V., Monuteaux, M. C. and Feighner, J. A. (2000a) 'Patterns of alcohol and drug use in adolescents can be predicted by parental substance use disorders', *Pediatrics*, 106: 792-797.

Biederman, J., Faraone, S. V., Mick, E., Wozniak, J., Chen, L., Ouellette, C., Marrs, A., Moore, P., Garcia, J., Mennin, D. and Lelon, E. (1996) 'Attention-deficit hyperactivity disorder and juvenile mania: an overlooked comorbidity?', *Journal of the American Academy of Child and Adolescent Psychiatry,* 35: 997-1008.

Biederman, J., Mick, E. and Faraone, S. V. (2000b) 'Age-dependent decline of symptoms of attention deficit hyperactivity disorder: impact of remission definition and symptom type', *American Journal of Psychiatry*, 157: 816-818.

Biederman, J., Mick, E., Faraone, S. V. and Burback, M. (2001) 'Patterns of remission and symptom decline in conduct disorder: a four-year prospective study of an AD/HD sample', *Journal of the American Academy of Child and Adolescent Psychiatry*, 40: 290-298.

Biederman, J., Newcorn, J. and Sprich, S. (1991) 'Cormorbidity of AD/HD with conduct, depressive, anxiety, and other disorders', *American Journal of Psychiatry,* 148: 546-577.

Biederman, J., Santangelo, S. L., Faraone, S. V. and Kiely, K. (1995) 'Clinical correlates of enuresis in ADHD and non-ADHD children', *Journal of Child Psychology and Psychiatry and Allied Disciplines,* 36: 865-877.

Blakemore-Brown, L. (2002) *Reweaving the autistic tapestry: autism, Asperger Syndrome and ADHD*, London: Jessica Kingsley.

Booth, B., Fellman, W., Greenbaum, J., Matlen, T., Markel, G., Morris, H., Robin, A. L. and Tzelepis, A. (2003) *Myths about ADD/ADHD*.
Available URL: http://www.add.org/content/abc/myths.htm. Viewed 19 December 2003.

Bowden, C. L., Deutsch, C. K. and Swanson, J. M. (1988) 'Plasma dopamine-B-hydroxylase and platelet monoamine oxidase in attention deficit disorder and conduct disorder', *Journal of the American Academy of Child and Adolescent Psychiatry,* 27: 171-174.

Bradley, C. (1937) 'The behavior of children receiving benzedrine', *American Journal of Psychiatry,* 94: 577-585.

Breslau, N., Brown, G. G., DelDotto, J. E., Kumar, S., Ezhuthachan, S., Adreski, P. and Hufnagle, K. G. (1996) 'Psychiatric sequelae of low birth weight at 6 years of age', *Journal of Abnormal Child Psychology*, 24: 385-400.

Brown, T. (1996) *Brown ADD scales*, San Antonio: The Psychological Corporation, Harcourt Bruce and Company.

Brown, T. (2000) *Attention deficit disorders and comorbidities in children, adolescents and adults*, Washington, DC: American Psychiatric Press.

Butler, N. R., Goulding, J. and Howlett, B. C. (eds) (1986) *From birth to five: a study of the health and behaviour of Britain's five-year-olds*, Oxford: Pergamon Press.

Campbell, S. B. (1990) *Behavior problems in preschool children: clinical and developmental issues*, New York: Guilford Press.

Castellanos, F. X., Giedd, J. N. and Marsh, W. L. (1996) 'Quantitative brain magnetic resonance imaging in attention deficit hyperactivity disorder', *Archives of General Psychiatry,* 53: 607-616.

Castellanos, F. X., Lee, P. P., Sharp, W., Jeffries, N. O., Greenstein, D. K., Clasen, L. S., Blumenthal, J. D., James, R. S., Ebens, C. L., Walter, J. M., Zijdenbos, A., Evans, A. C., Giedd, J. N. and Rapoport, J. L. (2002) 'Developmental trajectories of brain volume abnormalities in children and adolescents with attention-deficit/hyperactivity disorder', *JAMA: the Journal of the American Medical Association,* 288: 1740-1748.

Connor, D. F., Fletcher, K. E. and Swanson, J. M. (1999) 'A meta-analysis of clonidine for symptoms of attention-deficit hyperactivity disorder', *Journal of the American Academy of Child and Adolescent Psychiatry*, 38: 1551-1559.

Cook Jnr, E. H., Stein, M. A., Krasowski, M. D., Cox, N. J., Olkon, D. M., Kieffer, J. E. and Leventhal, B. L. (1995) 'Association of attention-deficit disorder and the dopamine transporter gene', *American Journal of Human Genetics,* 56: 993-998.

Costello, E. J. (1989) 'Developments in psychiatric epidemiology: introduction', *Journal of the American Academy of Child and Adolescent Psychiatry,* 28: 836-841.

Daley, K. C. (2004) 'Update on attention-deficit/hyperactivity disorder', *Current Opinion in Pediatrics,* 16: 217-226.

Dalsgaard, S., Mortensen, P. B., Frydenberg, M. and Thomsen, P. H. (2002) 'Conduct problems, gender and adult psychiatric outcome of children with attention-deficit hyperactivity disorder', *British Journal of Psychiatry*, 181: 416-421.

Daviss, W. B., Bentivoglio, P., Racusin, R., Brown, K. M., Bostic, J. Q. and Wiley, L. (2001) 'Bupropion sustained release in adolescents with comorbid attention deficit/hyperactivity disorder and depression', *Journal of the American Academy of Child and Adolescent Psychiatry,* 40: 307-314.

Dougherty, D. D., Bonab, A. A., Spencer, T. J., Rauch, S. L., Madras, B. K. and Fischman, A. J. (1999) 'Dopamine tranpsorter density in patients with attention deficit hyperactivity disorder. Research letter, 18-25 December', *The Lancet*, 354 (9196): 2132-2133.

Doyle, A. E., Biederman, J., Seidman, L. J., Weber, W. and Faraone, S. V. (2000) 'Diagnostic efficiency of neuropsychological test scores for discriminating boys with and without attention deficit-hyperactivity disorder', *Journal of Consulting and Clinical Psychology*, 68: 477-488.

DuPaul, G. J., Anastopoulos, A. D., Shelton, T. L., Guevremont, D. C. and Metevia, L. (1992) 'Multimethod assessment of attention-deficit hyperactivity disorder: the diagnostic utility of clinic-based tests', *Journal of Clinical Child Psychology*, 21: 394-402.

DuPaul, G. J. and Eckert, T. L. (1998) 'Academic interventions for students with attention-deficit/ hyperactivity disorder: a review of the literature', *Reading and Writing Quarterly: Overcoming Learning Difficulties,* 14: 59-82.

Duplantier, F. R. (2003) *Diagnosis: money-making opportunity. Is Attention Deficit Disorder a real disease, or is it just a psychologist's scam for making a quick buck?* Available URL: http://www.losthorizons.com/comments/archives/ADD_AD/HD.htm. Viewed 19 December 2003.

Dykman, K. D. and Dykman, R. A. (1998) 'Effect of nutritional supplements on attentional-deficit hyperactivity disorder', *Integrative Physiological and Behavioral Science,* 33: 49-60.

Edwards, M. C., Schultz, D. and Long, N. (1995) 'The role of the family in the assessment of attention deficit hyperactivity disorder', *Clinical Psychology Review,* 15: 375-394.

Egeland, B., Kalkoske, M., Gottesman, N. and Erickson, M. F. (1990) 'Preschool behavior problems: stability and factors accounting for change', *Journal of Child Psychology and Psychiatry*, 31: 891-909.

Ehlers, S. and Gillberg, C. (1993) 'The epidemiology of Asperger syndrome: a total population study', *Journal of Child Psychology and Psychiatry,* 34: 1327-1350.

Ercan, E. S., Coskunal, H., Varan, A. and Toksoz, K. (2003) 'Childhood attention deficit/hyperactivity disorder and alcohol dependence: a 1-year follow-up', *Alcohol Alcohol*, 38: 352-356.

Erhardt, D. and Hinshaw, S. P. (1994) 'Initial sociometric impressions of attention deficit hyperactivity disorder and comparison boys: predictions from social behaviours and from nonbehavioural variables', *Journal of Consulting and Clinical Psychology,* 62: 833-842.

Faraone, S. V., Biederman, J. and Friedman, D. (2000a) 'Validity of DSM-IV subtypes of attention-deficit/hyperactivity disorder: a family study perspective', *Journal of the American Academy of Child and Adolescent Psychiatry*, 39: 300-307.

Faraone, S. V., Biederman J, Lehman, B. K., Spencer, T., Norman, D., Seidman, L. J., Kraus, I., Perrin, J., Chen, W. J. and Tsuang, M. T. (1993) 'Intellectual performance and school failure in children with attention deficit hyperactivity disorder and in their siblings', *Journal of Abnormal Psychology,* 102: 616-623.

Faraone, S. V., Biederman, J., Mennin, D., Wozniak, J. and Spencer, T. (1997) 'Attention-deficit hyperactivity disorder with bipolar disorder: a familial subtype?', *Journal of the American Academy of Child and Adolescent Psychiatry*, 36: 1378-1387.

Faraone, S. V., Biederman, J., Mick, E., Williamson, S., Wilens, T., Spencer, T. J., Weber, W., Jetton, J., Kraus, I., Pert, J. and Zallen, B. (2000b) 'Family study of girls with attention deficit hyperactivity disorder', *American Journal of Psychiatry*, 157: 1077-1083.

Faraone, S. V., Biederman, J. and Monuteaux, M. C. (2000c) 'Attention-deficit disorder and conduct disorder in girls: evidence for a familial subtype', *Biological Psychiatry*, 48: 21-29.

Faraone, S. V., Biederman, J. and Monuteaux, M. C. (2000d) 'Toward guidelines for pedigree selection in genetic studies of attention deficit hyperactivity disorder', *Genetic Epidemiology*, 18: 1-16.

Faraone, S. V., Biederman, J. and Monuteaux, M. C. (2001) 'Attention deficit hyperactivity disorder with bipolar disorder in girls: further evidence for a familial subtype?', *Journal of Affective Disorders,* 64: 19-26.

Faraone, S. V., Biederman, J. and Monuteaux, M. C. (2002) 'Further evidence for the diagnostic continuity between child and adolescent AD/HD', *Journal of Attention Disorders*, 6: 5-13.

Faraone, S. V., Biederman, J., Spencer, T., Wilens, T., Seidman, L. J., Mick, E. and Doyle, A. E. (2000e) 'Attention-deficit/hyperactivity disorder in adults: an overview', *Biological Psychiatry*, 48: 9-20.

Faraone, S. V., Biederman, J., Weber, W. and Russell, R. L. (1998) 'Psychiatric, neuropsychological and psychological features of DSM-IV subtypes of attention-deficit/hyperactivity disorder: results from a clinically referred sample', *Journal of the American Academy of Child and Adolescent Psychiatry*, 37: 185-193.

Faraone, S. V. and Doyle, A. E. (2000) 'Genetic influences on attention deficit hyperactivity disorder', *Current Psychiatry Reports*, 2: 143-146.

Farrington, D. (1995) 'The twelfth Jack Tizard Memorial Lecture. The development of offending and antisocial behaviour from childhood: key findings of the Cambridge Study of Delinquent Development', *Journal of Child Psychology and Psychiatry,* 36: 929-964.

Feingold, B. F. (1975) 'Hyperkinesis and learning disabilities linked to artificial food flavors and colors', *The American Journal of Nursing*, 75: 797-803.

Fergusson, D. M. and Horwood, L. J. (1995) 'Early disruptive behavior, IQ, and later school achievement and delinquent behavior', *Journal of Abnormal Child Psychology,* 23: 183-199.

Fischer, M., Barkley, R. A., Edelbrock, C. S. and Smallish, L. (1990) 'The adolescent outcome of hyperactive children diagnosed by research criteria: II. Academic, attentional and nuerospsychological status', *Journal of Consulting and Clinical Psychology*, 58: 580-588.

Fischer, M., Barkley, R. A., Fletcher, K. E. and Smallish, L. (1993a) 'The adolescent outcome of hyperactive children: predictors of psychiatric, academic, social and emotional adjustment', *Journal of the American Academy of Child and Adolescent Psychiatry,* 32: 324-332.

Fischer, M., Barkley, R. A., Fletcher, K. E. and Smallish, L. (1993b) 'The stability of dimensions of behavior in AD/HD and normal children over an 8-year follow-up', *Journal of Abnormal Child Psychology*, 21: 315-337.

Fischer, M., Barkley, R. A., Smallish, L. and Fletcher, K. E. (2002) 'Young adult follow-up of hyperactive children: self-reported psychiatric disorders, comorbidity, and the role of childhood conduct problems and teen CD', *Journal of Abnormal Child Psychology*, 30: 463-475.

Gaub, M. and Carlson, C. L. (1997) 'Behavioral characteristics of DSM-IV ADHD subtypes in a school-based population', *Journal of Abnormal Child Psychology,* 25: 103-111.

Ghaziuddin, M., Weidmer-Mikhail, E. and Ghaziuddin, N. (1998) 'Comorbidity of Asperger syndrome: a preliminary report', *Journal of Intellectual Disability Research*, 42: 279-83.

Gillberg, C., Carlstrom, G. and Rasmussen, P. (1983) 'Hyperkinetic disorders in children with perceptual, motor and attention deficits', *Journal of Child Psychology and Psychiatry*, 24: 233-246.

Gillis, J. J., Gilger, J. W., Pennington, B. F. and DeFries, J. C. (1992) 'Attention deficit disorder in reading-disabled twins: evidence for a genetic etiology', *Journal of Abnormal Child Psychology*, 20: 303-15.

Gjone, H., Stevenson, J. and Sundet, J. M. (1996) 'Genetic influence on parent-reported attention-related problems in a Norwegian general population twin sample', *Journal of the American Academy of Child and Adolescent Psychiatry,* 35: 588-596.

Glod, C. A., Teicher, M. H., McGreenery, C. E., Ducsik, M., Anderson, C. M. and Polcari, A. M. (1996) 'Gender differences in hyperactivity in children', *American Psychiatric Association Abstract*, NR215: 126.

Goldstein, S. and Brzostek, J. S. (2000, October) 'Prevalence of AD/HD among high risk adolescents: preliminary findings', *The AD/HD Report, 8:* 10-11.

Gomez, R., Harvey, J., Quick, C., Scharer, I. and Harris, G. (1999) 'DSM-IV AD/HD: confirmatory factor models, prevalence, and gender and age differences based on parent and teacher ratings of Australian primary school children', *Journal of Child Psychology and Psychiatry,* 40: 265-274.

Goodenough, P. (2003) *Ritalin debate: some experts doubt existence of AD/HD*, CNS News. Available URL: http://www.cnsnews.com/Culture/Archive/200304/CUL20030418a.html. Viewed 19 December 2003.

Greene, R. W., Biederman, J., Faraone, S. V., Monuteaux, M. C., Mick, E., DuPre, E. P., Fine, C. S. and Goring, J. C. (2001) 'Social impairment in girls with ADHD: patterns, gender comparisons, and correlates', *Journal of the American Academy of Child and Adolescent Psychiatry,* 40: 704-710.

Greenfield, B., Hechtman, L. and Weiss, G. (1988) 'Two subgroups of hyperactives as adults: correlations of outcome', *Canadian Journal of Psychiatry*, 33: 505-508.

Hart, E. L., Lahey, B. B., Loeber, R., Applegate, B. and Frick, P. J. (1995) 'Developmental change in attention-deficit hyperactivity disorder in boys: a four-year longitudinal study', *Journal of Abnormal Child Psychology,* 23: 729-749.

Hechtman, L. and Greenfield, B. (2003) 'Long-term use of stimulants in children with attention deficit hyperactivity disorder: safety, efficacy, and long-term outcome', *Paediatric Drugs,* 5: 787-94.

Hechtman, L. and Weiss, G. (1983) 'Long-term outcome of hyperactive children', *American Journal of Orthopsychiatry*, 53: 532-541.

Hechtman, L. and Weiss, G. (1986) 'Controlled prospective fifteen year follow-up of hpyeractives as adults: non-medical drug and alcohol use and anti-social behaviour', *Canadian Journal of Psychiatry*, 31: 557-567.

Hechtman, L., Weiss, G. and Perlman, T. (1980) 'Hyperactives as young adults: self-esteem and social skills', *Canadian Journal of Psychiatry*, 25: 478-483.

Hechtman, L., Weiss, G. and Perlman, T. (1984a) 'Hyperactives as young adults: past and current substance abuse and antisocial behavior', *American Journal of Orthopsychiatry*, 54: 415-425.

Hechtman, L., Weiss, G. and Perlman, T. (1984b) 'Young adult outcome of hyperactive children who received long term stimulant treatment', *Journal of the American Academy of Child and Adolescent Psychiatry,* 23: 261-269.

Heilman, K. M., Voellar, K. S. and Nadeau, S. E. (1991) 'A possible pathophysiological substrate of AD/HD', *Journal of Child Neurology,* 6: S76-S81.

Herrero, M. E., Hechtman, L. and Weiss, G. (1994) 'Antisocial disorders in hyperactive subjects from childhood to adulthood: predictive factors and characterization of subgroups', *American Journal of Orthopsychiatry*, 64: 510-521.

Hill, J. C. and Schoener, E. P. (1996) 'Age-dependent decline of attention deficit hyperactivity disorder', *American Journal of Psychiatry,* 153: 1143-1146.

Hill, P. and Taylor, E. (2001) 'An auditable protocol for treating attention deficit/hyperactivity disorder', *Archives of Disease in Childhood*, 84: 404-409.

Hinshaw, S. P. (1994) *Attention deficits and hyperactivity in children*, Thousand Oaks, CA: Sage.

Hussain, S. A. and Cantwell, D. P. (1991) *Fundamentals of child and adolescent psychiatry*, Washington DC: American Psychiatric Press.

Hynd, G. W. and Hooper, S. R. (1995) *Neurological basis of childhood psychopathology*, London: Sage Publications.

Jensen, P. (2000) *Myths, mantras and miscommunications: the case of AD/HD*, NIMH Constituency Outreach and Education Program, 23 May.

Available URL: http://www.outreach.nimh.nih.gov/speech_myths/speech_myths.htm. Viewed 19 December 2003.

Jensen, P. S., Hinshaw, S. P., Swanson, J. M., Greenhill, L. L., Conners, C. K., Arnold, L. E., Abikoff, H. B., Elliott, G., Hechtman, L., Hoza, B., March, J. S., Newcorn, J. H., Severe, J. B., Vitiello, B., Wells, K. and Wigal, T. (2001) 'Findings from the NIMH multimodal treatment study of AD/HD (MTA): implications and applications for primary care providers', *Journal of Developmental and Behavioral Pediatrics*, 22: 60-73.

Jensen, P. S., Kettle, L., Roper, M., Sloan, M., Dulcan, M., Hoven, C., Bird, H., Bauermeister, J. and Payne, J. (1999) 'Are stimulants overprescribed? Treatment of ADHD in four US communities', *Journal of the American Academy of Child and Adolescent Psychiatry*, 38: 797-803.

Johnston, C. and Mash, E. J. (2001) 'Families of children with attention deficit/hyperactivity disorder: review and recommendations for future research', *Clinical Child and Family Psychology Review*, 4: 183-207.

Kadesjo, B. and Gillberg, C. (2001) 'The comorbidity of AD/HD in the general population of Swedish school-age children', *Journal of Child Psychology and Psychiatry*, 42: 487-492.

Kaplan, B. J., McNicol, J., Conte, R. A. and Moghadam, H. K. (1989) 'Dietary replacement in preschool-aged hyperactive boys', *Pediatrics*, 83: 7-17.

Kaplan, B. J., Crawford, S. G., Wilson, B. N. and Dewey, D. (2000) 'Does "pure" AD/HD exist?', *Journal of the International Neuropsychological Society*, 6: 153.

Kent, L. and Craddock, N. (2003) 'Is there a relationship between attention deficit hyperactivity disorder and bipolar disorder?', *Journal of Affective Disorders*, 73: 211-221.

Klein, R. G. and Mannuzza, S. (1988) 'Hyperactive boys almost grown up. III. Methylphenidate effects on ultimate height', *Archives of General Psychiatry,* 45: 1131-1134.

Knapp, M. (1997) 'Economic evaluations and interventions for children and adolescents with mental health problems', *Journal of Child Psychology and Psychiatry*, 38: 3-25.

Kotima, A. J., Moilanen, I., Taanila, A., Ebeling, H., Smalley, S. L., McGough, J. J., Hartikainen, A. L. and Jarvelin, M. R. (2003) 'Maternal smoking and hyperactivity in 8-year-old children', *Journal of the American Academy of Child and Adolescent Psychiatry*, 42: 826-833.

La Hoste, G. J., Swanson, J. M., Wigal, S. B., Globe, C., Wigal, T., King, N. and Kennedy, J. L. (1996) 'Dopamine D4 receptor gene is associated with attention deficit hyperactivity disorder', *Molecular Psychiatry,* 1: 121-124.

Lambert, N. M. and Hartsough, C. S. (1998) 'Prospective study of tobacco smoking and substance dependencies among samples of ADHD and non-ADHD participants', *Journal of Learning Disabilities,* 31: 533-544.

Lavigne, J. V., Cicchetti, C., Gibbons, R. D., Binns, H. J., Larsen, L. and DeVito, C. (2001) 'Oppositional defiant disorder with onset in preschool years: longitudinal stability and pathways to other disorders', *Journal of the American Academy of Child and Adolescent Psychiatry*, 40: 1393-1400.

Levy, F. (1997) 'Attention deficit hyperactivity disorder: editorial', *British Medical Journal,* 315: 894-895.

Levy, F., Hay, D. A., McStephen, M., Wood, C. and Waldman, I. (1997) 'Attention deficit-hyperactivity disorder: a category or a continuum?', *Journal of the American Academy of Child and Adolescent Psychiatry,* 36: 737-744.

Levy, F., Hay, D.A. and Rooney, R. (1996) 'Predictors of persistence of Attention Deficit Hyperactivity Disorder symptoms in a large-scale twin study', *ADHD Report*, 4: 12.

Lilienfeld, S. O. and Waldman, I. D. (1990) 'The relation between childhood attention deficit hyperactivity disorder and adult antisocial behaviour re-examined: the problem of heterogeneity', *Clinical Psychology Review,* 10: 699-725.

Malone, M. A., Kershner, J. R. and Swanson, J. M. (1994) 'Hemispheric processing and methylphenidate effects in AD/HD', *Journal of Child Neurology,* 9: 181-189.

Mannuzza, S. and Klein, R. G. (2000) 'Long-term prognosis in attention-deficit/hyperactivity disorder', *Child and Adolescent Psychiatric Clinics of North America*, 9: 711-726.

Mannuzza, S., Klein, R. G., Bessler, A., Malloy, P. and Hynes, M. E. (1997) 'Educational and occupational outcome of hyperactive boys grown up', *Journal of American Academy of Child and Adolescent Psychiatry*, 36: 1222-1227.

Mannuzza, S., Klein, R. G., Bessler, A., Malloy, P. and LaPadula, M. (1993) 'Adult outcome of hyperactive boys. Educational achievement, occupational rank and psychiatric status', *Archives of General Psychiatry*, 50: 565-576.

Mannuzza, S., Klein, R. G., Bessler, A., Malloy, P. and LaPadula, M. (1998) 'Adult psychiatric status of hyperactive boys grown up', *American Journal of Psychiatry*, 155: 493-498.

Mannuzza, S., Klein, R. G., Bonagura, N., Malloy, P., Giampino, T. L. and Addalli, K. A. (1991) 'Hyperactive boys almost grown up', *Archives of General Psychiatry,* 48: 77-83.

Mannuzza, S., Klein, R. G., Klein, D. F., Bessler, A. and Shrout, P. (2002) 'Accuracy of adult recall of childhood attention deficit hyperactivity disorder', *American Journal of Psychiatry*, 159: 1882-1888.

Mannuzza, S., Klein, R. G., Konig, P. H. and Giampino, T. L. (1989) 'Hyperactive boys almost grown up, IV: criminality and its relationship to psychiatric status', *Archives of General Psychiatry,* 46: 1073-1079.

Marshall, V. G., Longwell, L., Goldstein, M. J. and Swanson, J. M. (1990) 'Family factors associated with aggressive symptomatology in boys with attention deficit hyperactivity disorder: a research note', *Journal of Child Psychology and Psychiatry*, 31: 629-636.

Mattes, J. A. and Gittelman, R. (1981) 'Effects of artificial food colorings in children with hyperactive symptoms', *Archives of General Psychiatry*, 38: 714-718.

Mick, E., Santangelo, S. L., Wypij, D. and Biederman, J. (2000) 'Impact of maternal depression on ratings of comorbid depression in adolescents with attention-deficit/hyperactivity disorder', *Journal of the American Academy of Child and Adolescent Psychiatry*, 39: 314-319.

Milberger, S., Biederman, J., Faraone, S. V., Guite, J. and Tsuang, M. T. (1997) 'Pregnancy, delivery and infancy complications and attention deficit hyperactivity disorder: issues of gene-environment interaction', *Biological Psychiatry*, 41: 65-75.

Minde, K., Weiss, G. and Mendelson, N. (1972) 'A 5-year follow-up study of 91 hyperactive school children', *Journal of the American Academy of Child and Adolescent Psychiatry,* 11: 595-619.

Molina, B. S. and Pelham, W. E. J. (2003) 'Childhood predictors of adolescent substance use in a longitudinal study of children with AD/HD', *Journal of Abnormal Psychology*, 112: 497-507.

Mota, V. L. and Schachar, R. J. (2000) 'Reformulating attention-deficit/hyperactivity disorder according to signal detection theory', *Journal of the American Academy of Child and Adolescent Psychiatry*, 39: 1144-1151.

MTA Co-operative Group (1999a) 'A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder', *Archives of General Psychiatry,* 56: 1073-1086.

MTA Co-operative Group (1999b) 'Moderators and mediators of treatment response for children with attention-deficit/hyperactivity disorder: the multi-modal treatment study of children with attention-deficit/hyperactivity disorder', *Archives of General Psychiatry,* 56: 1088-1096.

Murphy, K., Gordon, M. and Barkley, R. (2000, June) 'To what extent are AD/HD symptoms common? A reanalysis of standardization data from a DSM-IV checklist', *The AD/HD Report,* 8*:* 1-5.

Nakamura, R. K. (2002) *Attention Deficit/Hyperactivity Disorders: are children being overmedicated?*, Testimony to the Committee on Government Reform, United States House of Representatives, 26 September.
Available URL: http://www.nimh.nih.gov/events/maAD/HD.cfm. Viewed 19 December 2003.

National Institute of Mental Health (2003) *Attention Deficit Hyperactivity Disorder (AD/HD) – questions and answers*.
Available URL: http://www.nimh.nih.gov/publicat.AD/HDqa.cfm. Viewed 19 December 2003.

Parr, J. R., Ward, A. and Inman, S. (2003) 'Current practice in the management of attention deficit with hyperactivity disorder (AD/HD)', *Child: Care, Health and Development*, 29: 215-218.

Paule, M. G., Rowland, A. S., Ferguson, S. A., Chelonis, J. J., Tannock, R., Swanson, J. M. and Castellanos, F. X. (2000) 'Attention deficit/hyperactivity disorder: characteristics, interventions and models', *Neurotoxicology and Teratology*, 22: 631-651.

Pelham, W., Wheeler, T. and Chronis, A. (1998) 'Empirically supported psychosocial treatments for attention deficit hyperactivity disorder', *Journal of Clinical Child Psychology*. 27, 190-205.

Pennington, B. F. and Ozonoff, S. (1996) 'Executive function and developmental psychopathology', *Journal of Child Psychology and Psychiatry,* 27: 307-319.

Peris, T. S. and Hinshaw, S. P. (2003) 'Family dynamics and preadolescent girls with AD/HD: the relationship between expressed emotion, AD/HD symptomatology, and comorbid disruptive behavior', *Journal of Child Psychology and Psychiatry*, 44: 1177-1190.

Peterson, B. S., Pine, D. S., Cohen, P. and Brook, J. S. (2001) 'Prospective, longitudinal study of tic, obsessive-compulsive, and attention-deficit/hyperactivity disorders in an epidemiological sample', *Journal of the American Academy of Child and Adolescent Psychiatry*, 40: 685-695.

Pfiffner, L. J. and McBurnett, K. (1997) 'Social skills training with parent generalization: treatment effects for children with attention deficit disorder', *Journal of Consulting and Clinical Psychology,* 65: 749-757.

Pineda, D., Ardila, A., Rosselli, M., Arias, B. E., Henao, G. C., Gomez, L. F., Mejia, S. E. and Miranda, M. L. (1999) 'Prevalence of attention-deficit/hyperactivity disorder symptoms in 4- to 17-year-old children in the general population', *Journal of Abnormal Child Psychology*, 27: 455-462.

Rabiner, D. and Coie, J. D. (2000) 'Early attention problems and children's reading achievement: a longitudinal investigation. The Conduct Problems Prevention Research Group', *Journal of the American Academy of Child and Adolescent Psychiatry*, 39: 859-867.

Rapoport, J. L., Quinn, P. O. and Lamprecht, F. (1974) 'Minor physical anomolies and plasma dopamine-beta-hydroxylase activity in hyperactive boys', *American Journal of Psychiatry,* 131: 386-389.

Rasmusson, P. and Gillberg, C. (2000) 'Natural outcome of AD/HD with developmental coordination disorder at age 22 years: a controlled, longitudinal, community-based study', *Journal of the American Academy of Child and Adolescent Psychiatry*, 39: 1424-1431.

Richards, I. (1995) 'ADHD, ADD, and dyslexia'. In P. Cooper and K. Ideus (eds) *Attention Deficit/Hyperactivity Disorder: Educational, Medical and Cultural Issue*s, Maidstone, Kent: The Association of Workers for Children with Emotional and Behavioural Difficulties: 63-73.

Rohde, P., Kahler, C. W., Lewinsohn, P. M. and Brown, R. A. (2004) 'Psychiatric disorders, familial factors, and cigarette smoking: II. Associations with progression to daily smoking', *Nicotine and Tobacco Research,* 6: 119-112.

Rucklidge, J. J. and Tannock, R. (2001) 'Psychiatric, psychosocial and cognitive functioning of female adolescents with AD/HD', *Journal of the American Academy of Child and Adolescent Psychiatry*, 40: 530-540.

Russo, M. F. and Beidel, D. C. (1994) 'Comorbidity of childhood anxiety and externalising disorders: Prevalence, associated characteristics, and validation issues', *Clinical Psychology Review,* 14: 199-221.

Safer, D. J., Zito, J. M. and Fine, E. M. (1996) 'Increased methylphenidate usage for attention deficit disorder in the 1990s', *Pediatrics*, 98: 1084-1088.

Santosh, P. (2004) *ADHD and Autism spectrum disorders: is there a comorbidity?* Paper presented at the Sixth International ADDISS Conference 'Paying Attention to ADHD?', Adelphi Hotel, Liverpool, UK. 16 March.

Satterfield, J. H., Hoppe, C. M. and Schell, A. M. (1982) 'A prospective study of delinquency in 110 adolescent boys with attention-deficit disorder and 88 normal adolescent boys', *American Journal of Psychiatry,* 139: 795-798.

Scahill, L., Chappell, P. B., Kim, Y. S., Schultz, R. T., Katsovich, L., Shepherd, E., Arnsten, A. F., Cohen, D. J. and Leckman, J. F. (2001) 'A placebo-controlled study of guanfacine in the treatment of children with tic disorders and attention deficit hyperactivity disorder', *American Journal of Psychiatry*, 158: 1067-1074.

Schachar, R. J., Rutter, M. and Smith, A. (1981) 'The characteristics of situationally and pervasively hyperactive children: implications for syndrome definition', *Journal of Child Psychology and Psychiatry*, 22: 375-392.

Schachar, R. J. and Tannock, R. (1993) 'Childhood hyperactivity and psychostimulants: a review of extended treatment studies', *Journal of Child and Adolescent Psychopharmacology,* 3: 81-97.

Schaughency, E. A. and Rothlind, J. (1991) 'Assessment and classification of attention deficit-hyperactive disorders', *School Psychology Review,* 20*:* 187-202

Segman, R. H., Meltzer, A., Gross-Tsur, V., Kosov, A., Frisch, A., Inbar, E., Darvasi, A., Levy, S., Goltser, T., Weizman, A. and Galili-Weisstub, E. (2002) 'Preferential transmission of interleukin-1 receptor antagonist alleles in attention deficit hyperactivity disorder', *Molecular Psychiatry*, 7: 72-74.

Semrud-Clikeman, M., Biederman, J., Sprich-Buckminster, S., Lehman, B., Faraone, S. and Norman, D. (1992) 'Comorbidity between ADDH and learning disability: a review and report in a clinically referred sample', *Journal of the American Academy of Child and Adolescent Psychiatry,* 31: 439-448.

Semrud-Clikeman, M., Steingard, R. J., Filipek, P., Biederman, J., Bekken, K. and Renshaw, P. F. (2000) Using MRI to examine brain-behavior relationships in males with attention deficit disorder with hyperactivity. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39: 477-484.

Shaywitz, B. A., Fletcher, J. M. and Shaywitz, S. E. (1994) 'Issues in the definition and classification of attention deficit disorder', *Topics in Language and Disability*, 14: 1-25.

Shekim, W. O., Dekirmenjian, H. and Chapel, J. L. (1979) 'Urinary MHPG excretion in minimal brain dysfunction and its modification by d-amphetamine', *American Journal of Psychiatry,* 136: 667-671.

Shekim, W. O., Kashani, J., Beck, N., Cantwell, D., Martin, J., Rosenberg, J. and Costello, A. (1985) 'The prevalence of attention deficit disorders in a rural midwestern community sample of nine-year-old children', *Journal of the American Academy of Child Psychiatry*, 24: 765-770.

Shen, Y. and Wang, Y. (1984) 'Urinary 3-methoxy-4-hydroxphenylglycol sulfate excretion in seventy-three school children with minimal brain dysfunction syndrome', *Biological Psychiatry,* 19: 861-877.

Shepherd, P. (1985) *The National Child Development Study: an introduction to the origins of the study and the methods of data collectionl,* National Child Development Study User Support Group Working Paper Series, No. 1, London: Social Sciences Research Unit, City University.

Silverthorn, P., Frick, P. J., Kuper, K. and Ott, J. (1996) 'Attention deficit hyperactivity disorder and sex: a test of two etiological models to explain the male predominance', *Journal of Clinical Child Psychology,* 25: 52-59.

Simpson, D. and Plosker, G. L. (2004) 'Spotlight on atomoxetine in adults with attention-deficit hyperactivity disorder', *Central Nervous System Drugs,* 18: 397-401.

Solanto, M. V., Arnsten, A. F. T. and Castellanos, F. X. E. (2001) *Stimulant drugs and AD/HD: basic and clinical neuroscience*, New York: Oxford University Press.

Sonuga-Barke, E. J., Daley, D., Thompson, M., Laver-Bradbury, C. and Weeks, A. (2001) 'Parent-based therapies for preschool attention-deficit/hyperactivity disorder: a randomized, controlled trial with a community sample', *Journal of the American Academy of Child and Adolescent Psychiatry*, 40: 402-408.

Sowell, E. R., Thompson, P. M., Welcome, S. E., Henkenius, A. L., Toga, A. W. and Peterson, B. S. (2003) 'Cortical abnormalities in children and adolescents with attention-deficit hyperactivity disorder', *The Lancet*, 362: 1699-1707.

Spencer, T., Biederman, J., Kerman, K., Steingard, R. and Wilens, T. (1993) 'Desipramine treatment of children with attention-deficit hyperactivity disorder and tic disorder or Tourette's syndrome', *Journal of the American Academy of Child and Adolescent Psychiatry*, 32: 354-360.

Sprich-Buckminster, S., Biederman, J., Milberger, S., Faraone, S. V. and Lehman, B. K. (1993) 'Are perinatal complications relevant to the manifestation of ADD? Issues of comorbidity and familiality', *Journal of the American Academy of Child and Adolescent Psychiatry*, 32: 1032-1037.

Still, G. F. (1902) 'Some abnormal psychical conditions in children', *The Lancet, i,* 1008-1012; 1077-1082; 1163-1168.

Strayhorn, J. M. and Weidman, C. S. (1989) 'Reduction of attention deficit and internalizing symptoms in preschoolers through parent-child interaction training', *Journal of the American Academy of Child and Adolescent Psychiatry,* 28: 888-896.

Sund, A. M and Zeiner, P. (2002) 'Does extended medication with amphetamine or methylphenidate reduce growth in hyperactive children?', *Nordic Journal of Psychiatry*, 56: 53-7.

Swanson, J. M., Lerner, M. and Williams, L. (1995) 'More frequent diagnosis of attention deficit-hyperactivity disorder', *The New England Journal of Medicine*, 333: 944.

Swanson, J. M., McBurnett, K., Wigal, T., Pfittner, L., Lerner, M. A., Williams, L., Christian, D. L., Tarm, L., Willaitt, E., Crowley, K., Clevenger, W., Khouzon, N., Woo, C., Crinella, F. M. and Fisher, T. D. (1993) 'Effect of stimulant medication on children with attention deficit disorder: a review of reviews', *Exceptional Children,* 60: 154-162.

Swanson, J. M., Posner, M. I., Cantwell, D., Wigal, S., Crinella, F., Filipek, P., Emerson, J., Tucker, D. and Nalcioglu, O. (1998a) 'Attention-deficit/hyperactivity disorder: symptom domains, cognitive processes, and neural networks'. In R. Parasuraman (ed.) *The attentive brain* Cambridge, Massachusetts: MIT Press: 445-460.

Swanson, J. M., Sergeant, J. A., Taylor, E., Sonuga-Barke, E. J., Jensen, P. S. and Cantwell, D. (1998b) 'Attention-deficit hyperactivity disorder and hyperkinetic disorder', *The Lancet*, 351: 429-433.

Swanson, J. M., Sunohara, G. A., Kennedy, J. L., Regino, R., Fineberg, E., Wigal, T., Lerner, M., Williams, L., La Hoste, G. J. and Wigal, S. (1998c) 'Association of the dopamine receptor D4 (DRD4) gene with a refined phenotype of attention deficit hyperactivity disorder (ADHD): a family-based approach', *Molecular Psychiatry,* 3: 38-41.

Szatmari, P., Offord, D. R. and Boyle, M. H. (1989) 'Ontario Child Health Study: prevalence of attention deficit disorder with hyperactivity', *Journal of Child Psychology and Psychiatry*, 30: 219-230.

Szymanski, M. L. and Zolotor, A. (2001) 'Attention-deficit/hyperactivity disorder: management', *American Family Physician*, 64: 1355-1362.

Taylor, E. (1994) 'Syndromes of attention deficit and overactivity'. In M. Rutter and E. Taylor and L. Hersov (eds), *Child and Adolescent Psychiatry: Modern Approaches*, Oxford: Blackwell Scientific Publications: 285-307.

Taylor, E. (2001) *From child to adult: the development of dysfunction*, paper presented at the ADDISS conference 'AD/HD in Adult Life', 9 November, Kensington Town Hall, London.

Taylor, E., Sandberg, S., Thorley, G. and Giles, S. (1991) *The epidemiology of childhood hyperactivity*, Maudsley Monographs No. 33. Oxford: Oxford University Press.

Taylor, E., Chadwick, O., Heptinstall, E. and Danckaerts, M. (1996) 'Hyperactivity and conduct problems as risk factors for adolescent development', *Journal of the American Academy of Child and Adolescent Psychiatry,* 35: 1213-1226.

Taylor, F. B. and Russo, J. (2001) 'Comparing guanfacine and dextroamphetamine for the treatment of adult attention-deficit/hyperactivity disorder', *Journal of Clinical Psychopharmacology*, 21: 223-228.

Teicher, M. H., Anderson, C. M., Polcari, A., Glod, C. A., Maas, L. C. and Renshaw, P. F. (2000) 'Functional deficits in basal ganglia of children with attention-deficit/hyperactivity disorder shown with functional magnetic resonance imaging relaxometry', *Nature Medicine*, 6: 470-473.

Tercyak, K. P., Lerman, C. and Audrain, J. (2002) 'Association of attention-deficit/hyperactivity disorder symptoms with levels of cigarette smoking in a community sample of adolescents', *Journal of the American Academy of Child and Adolescent Psychiatry,* 41: 799-805.

Thapar, A., Fowler, T., Rice, F., Scourfield, J., van den Bree, M., Thomas, H., Harold, G. and Hay, D. (2003) 'Maternal smoking during pregnancy and attention deficit hyperactivity disorder symptoms in offspring', *American Journal of Psychiatry,* 160: 1985-1989.

Thapar, A., Holmes, J., Poulton, K. and Harrington, R. (1999) 'Genetic basis of attention deficit and hyperactivity', *British Journal of Psychiatry,* 174: 105-111.

Thunstrom, M. (2002) 'Severe sleep problems in infancy associated with subsequent development of attention-deficit/hyperactivity disorder at 5.5 years of age', *Acta Paediatrica*, 91: 584-592.

United States National Library of Medicine. (1998) *Current bibliographies in medicine 98-2: diagnosis and treatment of Attention Deficit Hyperactivity Disorder*, National Institute of Health. Available URL: http://www.nlm.nih.gov/pubs/cbm/AD/HD.html#120. Viewed 18 November 2003.

Van der Meere, J. J. (1996) 'The role of attention'. In S. T. Sandberg (ed.) *monographs in child and adolescent psychiatry* (Chapter: Hyperactivity disorders of childhood), Cambridge: Cambridge University.

Vincent, J., Varley, C. K. and Leger, P. (1990) 'Effects of methylphenidate on early adolescent growth', *American Journal of Psychiatry,* 147: 501-502.

Vitiello, B. (2001) 'Long-term effects of stimulant medications on the brain: possible relevance to the treatment of attention deficit hyperactivity disorder', *Journal of Child and Adolescent Psychopharmacology*, 11: 25-34.

Weiss, G., Hechtman, L., Milroy, T. and Perlman, T. (1985) 'Psychiatric status of hyperactives as adults: a controlled prospective 15-year follow-up of 63 hyperactive children', *Journal of the American Academy of Child and Adolescent Psychiatry,* 24: 211-220.

Weiss, G., Hechtman, L., Perlman, T., Hopkins, J. and Wener, A. (1979) 'Hyperactives as young adults: a controlled prospective ten-year follow-up of 75 children', *Archives of General Psychiatry,* 36: 675-681.

Weiss, B., Williams, J. H., Margen, S., Abrams, B., Caan, B., Citron, L., Cox, C., McKibben, J., Ogar, D. and Schultz, S. (1980) 'Behavioral response to artificial food colors', *Science*, 207: 1487-1489.

Wender, P. H., Wolf, L. E. and Wasserstein, J. (2001) 'Adults with AD/HD. An overview', *Annals of the New York Academy of Sciences*, 931: 1-16.

Whitaker, A. H., Von Rossen, R., Feldman, J. F., Schonfeld, I. S., Pinto-Martin, J. A., Torre, C., Shaffer, D. and Paneth, N. (1997) 'Psychiatric outcomes of low-birth-weight children at age 6 years: relation to neonatal cranial ultrasound abnormalities', *Archives of General Psychiatry*, 54: 847-856.

Wilens, T. E., Biederman, J., Geist, D. E., Steingard, R. and Spencer, T. (1993) 'Nortriptyline in the treatment of AD/HD: a chart review of 58 cases', *Journal of the American Academy of Child and Adolescent Psychiatry*, 33: 142-3.

Wilens, T. E., Biederman, J., Wozniak, J., Gunawardene, S., Wong, J. and Monuteaux, M. C. (2003a) 'Can adults with attention-deficit/hyperactivity disorder be distinguished from those with comorbid bipolar disorder? Findings from a sample of clinically referred adults', *Biological Psychiatry,* 54: 1-8.

Wilens, T. E., Faraone, S. V., Biederman, J. and Gunawardene, S. (2003b) 'Does stimulant therapy of attention-deficit/hyperactivity disorder beget later substance abuse? A meta-analytic review of the literature', *Pediatrics*, 111: 179-185.

Wilens, T. E., Spencer, T. J., Biederman, J., Girard, K., Doyle, R., Prince, J., Polisner, D., Solhkhah, R., Comeau, S., Monuteaux, M. C. and Parekh, A. (2001) 'A controlled clinical trial of bupropion for attention deficit hyperactivity disorder in adults', *American Journal of Psychiatry*, 158: 282-288.

Wolf, L. E. and Wasserstein, J. (2001) 'Adult AD/HD. Concluding thoughts', *Annals of the New York Academy of Sciences*, 931: 396-408.

Wolraich, M. L., Hannah, J. N., Pinnock, T. Y., Baumgaertel, A. and Brown, J. (1996) 'Comparison of diagnostic criteria for attention-deficit hyperactivity disorder in a county-wide sample', *Journal of the American Academy of Child and Adolescent Psychiatry*, 35: 319-24.

Woodward, L., Taylor, E. and Dowdney, L. (1998) 'The parenting and family functioning of children with hyperactivity', *Journal of Child Psychology and Psychiatry*, 39: 161-169.

World Health Organisation (1993) *The ICD-10 Classification of mental and behavioural disorders: diagnostic criteria for research*, Geneva: World Health Organisation.

Young Minds. (2003) *A 21st century illness? The great AD/HD Debate*, Young Minds Magazine No. 66.
Available URL: http://www.youngminds.org.uk/magazine/66/AD/HD.php. Viewed 19 December 2003.

Zametkin, A. J., Karoum, F., Rapoport, J. L., Brown, G. L. and Wyatt, R. J. (1984) 'Phenylethylamine excretion in attention deficit disorder', *Journal of the American Academy of Child and Adolescent Psychiatry*, 23: 310-314.

Attention Deficit/Hyperactivity Disorder

An overview and review of the literature relating to the correlates and lifecourse outcomes for males and females

Attention-deficit/hyperactivity disorder (AD/HD) is a common disorder of childhood onset, characterised by problems with concentration, impulse control and overactivity, and reported to be associated with a variety of adverse adolescent and adult outcomes. However, the definition, measurement and causes of AD/HD are considered to be debatable issues, which could raise questions for whether and how meaningful research into its lifecourse and outcomes can be conducted. In spite of these reservations it is the most extensively studied childhood disorder, with several thousands of peer-reviewed papers in the scientific literature.

In this paper the authors aim to provide an overview of what AD/HD is, take a brief look at some of the current debates surrounding the conceptual and measurement issues associated with AD/HD, and then review some of the recent literature with a specific focus on the lifecourse outcomes for individuals diagnosed with AD/HD.

Bedford Group for Lifecourse and Statistical Studies
Institute of Education, University of London
20 Bedford Way
London WC1H 0AL
tel: +44 (0)20 7612 6900
fax: +44 (0)20 7612 6880
email: publications@bg.ioe.ac.uk
web: www.ioe.ac.uk/bedfordgroup