ADHD is best understood as a cultural construct

SAMI TIMIMI / ERIC TAYLOR

Edited and introduced by Mary Cannon, Kwame McKenzie and Andrew Sims.

INTRODUCTION

Attention-deficit hyperactivity disorder (ADHD) has received significant research attention and is a problem that is rarely out of the news – whether it is concerns about treating children with amphetamines, its over- or underdiagnosis, or the long-term outcomes.

Despite all the research it has been difficult to gain and maintain professional agreement on what ADHD is or what should be done about it. In 2002 an eminent group of psychiatrists and psychologists published the first consensus statement on the science, diagnosis and treatment of ADHD (Barkley et al, 2002). However, the statement could probably best be described as a position statement because the diverse views of what ADHD is and what should be done about it were not reflected or represented. Fundamental to the discussion are questions about whether the diagnosis of ADHD actually holds water and what it is that psychiatrists are trying to treat. Are differences in the rate of ADHD a reflection of changes in its incidence or in society's tolerance for behaviour that does not conform? We asked Dr Sami Timimi, a child and adolescent psychiatrist and author of Pathological Child Psychiatry and the Medicalization of Childhood, and Professor Eric Taylor, a child psychiatrist from the Institute of Psychiatry and researcher into the aetiology, outcome and treatment of ADHD, to discuss the proposition that ADHD is best understood as a cultural construct.

FOR

There are no specific cognitive, metabolic or neurological markers and no medical tests for ADHD. Because of uncertainty about definition, epidemiological studies produce hugely differing prevalence rates: from 0.5% to 26% of children. Despite attempts at standardising criteria, in crosscultural studies major and significant differences between raters from different countries in the way they rate symptoms of ADHD, as well as major differences in the way children from different cultures are rated for symptoms of ADHD, are apparent. More than 30 neuroimaging studies have been published; however, researchers have yet to compare unmedicated children diagnosed with ADHD with an agematched control group. Sample sizes in these studies have been small and have produced a variety of inconsistent results. In no study were the brains considered clinically abnormal, nor is it possible to work out whether any differences seen are caused by (rather than being the causes of) different styles of thinking, or are the result of the medication the children had taken. What we end up with is a modern version of the long-discredited 'science' of phrenology. Genetic studies show that ADHD is linked with being male (boys are four to ten times more likely to get this diagnosis in practice) and is associated with the normal genetic variation found with traits such as height. Comorbidity is extremely high, throwing doubt on the specificity of the diagnosis. There are no specific treatments for ADHD, with the most widely debated treatment (methylphenidate) being known to have similar effects on otherwise normal children. There is no established prognosis, and association and cause frequently are confused in the literature. ADHD has generated huge profits for the pharmaceutical industry against a background of poor-quality research, publication bias and payments to some of the top

academics in this field. Thus, the mainstream dogma on ADHD is contaminated and misleading (Timimi, 2002).

To explain the recent rise, to epidemic proportions, of rates of diagnosis of ADHD, a cultural perspective is necessary. The immaturity of children is a biological fact, but the ways in which this immaturity is understood and made meaningful is a fact of culture. In modern Western culture many factors adversely affect the mental health of children and their families. These include loss of extended family support, mother blame (mothers are usually the ones who shoulder responsibility for their children), pressure on schools, a breakdown in the moral authority of adults, parents being put in a double bind on the question of discipline, family life being busy and 'hyperactive', and a market economy value system that emphasises individuality, competitiveness and independence (Prout & James, 1997). Throw in the profit-dependent pharmaceutical industry and a highstatus profession looking for new roles and we have the ideal cultural preconditions for the birth and propagation of the ADHD construct.

Is a medical model of ADHD therapeutically helpful? Quite the opposite; it offers a decontextualised and simplistic idea that leads to all of us - parents, teachers and doctors - disengaging from our social responsibility to raise well-behaved children. We thus become a symptom of the cultural disease we purport to cure. It supports the profit motive of the pharmaceutical industry, which has been accused of helping to create and propagate the notion of ADHD in order to expand its own markets. By acting as agents of social control and stifling diversity in children, we are victimising millions of children and their families by putting children on highly addictive drugs that have no proven long-term benefit (Timimi, 2002) and have been shown in animal studies to have brain-disabling effects (Moll et al, 2001; Sproson et al, 2001; Breggin, 2002). By conceptualising problems as medically caused we end up offering interventions (drug and behavioural) that teach ADHD-type behaviour to the child. ADHD causes 'tunnel vision' in the system, making it more difficult to think about context, leading to interpersonal issues being marginalised. ADHD scripts a potentially life-long story of disability and deficit, resulting in an attitude of a 'pill for life's problems'. We create unnecessary dependence on doctors, discouraging children and their families from engaging their own abilities to solve problems. ADHD is de-skilling for us as a profession as there is minimal skill involved in ticking off a checklist of symptoms and reaching for the prescription pad.

Declaration of interest

None.

S. Timimi Ash Villa Child & Adolescent Unit, Willoughby Road, South Rauceby, Sleaford, Lincolnshire NG34 8QA, UK. E-mail: stimimi@talk2l.com

AGAINST

Hyperactivity is neither a social construct nor a genetic disease. The professional task is to understand how genetic and social influences interact, not to simplify into a polemic.

Individual differences in hyperactivity have known physical counterparts: in brain structure and function and DNA composition (Schachar & Tannock, 2002). Genetic influences are strong and some molecular genetic variations (especially of genes affecting dopamine systems) have been robustly replicated. They work in interaction with the environment, and the psychological environment helps to set the course into adjustment or disorder. Consistent associations with changes in brain structure have been found, even in unmedicated children, with neuroimaging.

Severe hyperactivity is a strong predictor of poor psychosocial adjustment (Taylor *et al*, 1996). The developmental risk is not trivial. Even those who are not diagnosed or medicated are more prone to accidents, conduct disorder, psychiatric problems in adolescence, educational and occupational failure, and a lack of constructive occupations or satisfactory relationships. This is why mental health services get involved.

Some authorities suggest that the institutions of society can actually cause the problem. For example, the decay of the family, or the school, or social cohesion, or leisure activity can be blamed for children going 'out of control'. (Different countries vary in the choice of scapegoat according to their perceived social problems.) Evidence for this is lacking. Indeed, twin studies indicate that individual differences are very little influenced by the shared environment. If there were a social determinant of hyperactivity, it would need to be

seen as making the whole population more hyperactive; that is, the prevalence of a diagnostic category such as ADHD would have to be increasing over time and be related to social structures. In the UK, this is probably not so. Two epidemiological studies 20 years apart have produced very similar prevalence rates for 'hyperkinetic disorder' (Taylor *et al*, 1991; Meltzer *et al*, 2000). Powerless groups such as immigrants do not have markedly increased rates, and 'ADHD' affects all classes.

Social factors can probably influence the degree of hyperactivity that is seen as a problem. Children do not usually refer themselves for help (although they often try); they are dependent upon others to determine their caseness. Families, schools and cultures vary somewhat in their tolerance. For example, epidemiological research in Hong Kong used the same measures as had been used in London, England, and found a higher rate of hyperactivity in Hong Kong when ratings were used, but a lower rate when more objective measures were employed. The likely interpretation is that hyperactive behaviour had a greater impact in the Hong Kong environment, which attaches particular importance to academic success.

This interaction between the child and the expectations of the adult world is important clinically. It is a reason to take more pains in making a diagnosis than just accepting a rating from a parent or teacher. Impairment and risk are as important as symptomatology. An assessment should be thorough enough to clarify the interaction in the individual case (as well, of course, as to be able to detect the emotional problems and the relationship difficulties that can both mimic hyperactivity and result from it).

Could these social influences lead to the condition being overdiagnosed? This carries particular weight because of the frequent use of stimulant medication. If there is overdiagnosis, then children treated will often be found to fall short of rigorous diagnostic criteria. In the USA, this may sometimes happen. There is some evidence there for a patchy mixture of undertreatment and overtreatment, and a high rate of medication in preschool children suggests that some practitioners are going beyond guidelines.

In the UK, however, the chief evidence is for undertreatment. Most children with markedly hyperactive behaviour are still not identified, referred or treated; yet they remain at risk. The obstacle probably lies largely in medical attitudes rather than public ones (Sayal *et al*, 2002). This is a pity, because there are several good ways of supporting children with severe hyperactivity.

In short, broad social influences probably contribute to the recognition of disorder rather than its presence. But these do not amount to a social construction of disorder – rather, in the UK at least, they work against recognition of a treatable risk.

Declaration of interest

E.T. has received menaces from an antipsychiatry organisation, which may have biased him against their views. He and his department have received fees for lecturing at educational meetings and scientific conferences that had sponsorship from pharmaceutical companies – including Eli Lilly and Janssen-Cilag, who manufacture drugs used in ADHD. He is a lead clinician in a National Health Service trust, so could have an interest in keeping costs of treatment down.

E. Taylor Institute of Psychiatry, De Crespigny Park, Denmark HIII, London SE5 8AF, UK.

REFERENCES

Barkley, R. A., Cook, E. H. Jr, Diamond, A., et al. (2002) International Consensus Statement on ADHD. Clinical Child and Family Psychology Review, 5, 89–111.

Breggin, P. (2002) The Ritalin Fact Book. Cambridge, MA: Perseus Publishing.

Meltzer, H., Gatward, R., Goodman, R., et al (2000) *Mental Health of Children and Adolescents in Great Britain.*London: Stationery Office.

Moll, G., Hause, S., Ruther, E., et al (2001) Early methylphenidate administration to young rats causes a persistent reduction in the density of striatal dopamine transporters. *Journal of Child and Adolescent Psychopharmacology*, 11, 15–24.

Prout, A. & James, A. (1997) Constructing and Reconstructing Childhood: Contemporary Issues in the Sociological Study of Childhood. London: Falmer Press.

Sayal, K., Taylor, E., Beecham, J., et al (2002) Pathways to care in children at risk of attention-deficit hyperactivity disorder. *British Journal of Psychiatry*, **181**, 43–48.

Schachar, R. & Tannock, R. (2002) Syndromes of hyperactivity and attention deficit. In *Child and Adolescent Psychiatry* (4th edn) (eds M. Rutter & E. Taylor), pp. 399–418. Oxford: Blackwell.

Sproson, E. J., Chantrey, J., Hollis, C., et al (2001) Effect of repeated methylphenidate administration on presynaptic dopamine and behavior in young adult rats. *Journal of Psychopharmacology,* **15,** 67–75.

Taylor, E., Sandberg, S., Thorley, G., et al (1991) The Epidemiology of Childhood Hyperactivity. Maudsley Monograph No. 33. Oxford: Oxford University Press.

Taylor, E., Chadwick, O., Heptinstall, E., et al (1996) Hyperactivity and conduct problems as risk factors for adolescentdevelopment. *Journal of the American Academy of Child and Adolescent Psychiatry*, **35**, 1213–1226.

Timimi, S. (2002) Pathological Child Psychiatry and the Medicalization of Childhood. Hove: Brunner-Routledge.