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VIEWPOINT ARTICLE

Exploring five common assumptions on Attention Deficit Hyperactivity Disorder

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ABSTRACT

The number of children diagnosed with attention deficit hyperactivity disorder (ADHD) and treated with medication is steadily increasing. The aim of this paper was to critically discuss five debatable assumptions on ADHD that may explain these trends to some extent. These are that ADHD (i) causes deviant behaviour, (ii) is a disease, (iii) is chronic and (iv) is best treated by medication and (v) that classification should precede treatment.

Conclusion: We argue that ADHD is not a disease, not the cause of deviant behaviour and in most cases not chronic. Treatment for attention and hyperactivity problems could start with psychosocial interventions and without a diagnostic label. A stepped diagnosis approach may reduce overdiagnosis without risking undertreatment.

INTRODUCTION

The worldwide prevalence of attention deficit hyperactivity disorder (ADHD) in school-aged children is estimated to be 5–7% (1), which makes ADHD the most prevalent childhood psychiatric disorder. In many countries, the rise in ADHD diagnosis and the associated use of medication are of public concern (2).

ADHD is usually considered to be a chronic disease that causes deviant behaviour and should be treated with medication after elaborate diagnostics (3). Actually, this view is based on several assumptions that may be disputed. Each one of these assumptions may explain to some degree the increase in ADHD diagnosis and medication use and we aim to critically discuss them. In addition, a stepped care and stepped diagnosis approach is suggested, which may reduce overdiagnosis, without risking undertreatment.

ASSUMPTION 1: ADHD CAUSES DEVIANT BEHAVIOUR

ADHD is diagnosed when a child exhibits attention problems, impulsivity, hyperactivity and associated impairments. ADHD is one of the disorders described in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5). Although most DSM disorders, such as ADHD, are defined as descriptive syndromes, they are often reified and taken to be discrete biomedical entities. Reification refers to treating more or less abstract concepts as things,

existing out there in the world. This may result in confusing label with cause, naming with explaining. As Taylor and Rutter (4) warn us, 'It needs to be kept in mind that psychiatric diagnoses are usually descriptive, not explanatory. ADHD is a description of the behaviour of a child who is inattentive and impulsive, not a disease that explains why the child behaves that way'.

But even though some prominent psychiatrists recognise reification as a major problem (5,6), it is still rather widespread. It is not just patients, parents and teachers who are prone to this fallacy, but healthcare professionals and scientists as well. For example, Biederman and Faraone (3) state in a frequently cited review that 'ADHD affects 8–12% of children worldwide, and results in inattention, impulsivity, and hyperactivity'. However, just as the category bachelor does not *result* in a man being single, the category ADHD does not *result* in hyperactivity and inattention.

Reification is likely to play a significant role in the increased rates of psychiatric disorders, because it tends to exaggerate the power of the concepts: we seem to understand the problem and we have to look no further. Besides, seeing ADHD as the cause of the child's problems may bring relief to patients and their families, freeing all parties from feelings of guilt (Table 1).

ASSUMPTION 2: ADHD IS A DISEASE

Reification of diagnostic categories may facilitate the tendency to explore their biological background. Studies comparing groups of children with and without ADHD show small group differences in terms of genes and brain anatomy and function (7–10). However, interpretation of these differences is complicated. First, biological or genetic differences do not automatically imply abnormalities or pathology, for example, blue eyes and height are hereditary characteristics. Second, associations do not necessarily imply causality: associated factors also may represent epiphenomena of ADHD or result from comorbid conditions. Third, selection bias – the more rigorous screening for diagnosis in studies at academic centres – may limit how the findings can be generalised to children diagnosed in the community. Fourth, the biological differences demonstrated so far are nonspecific for ADHD, as similar differences have been reported for autism, schizophrenia, reading disability and other disorders (10,11). Fifth, the differences do not apply to many individuals diagnosed with ADHD: although within-group variations are large, between-group differences are small and can only be demonstrated at group level.

ADHD is a man-made classification with no definite clinical test. The diagnosis is based on decisions made by clinicians, with parents and teachers as primary sources of information. This means that factors such as parents' and teachers' tolerance, skills and expectations and the clinicians', and even society's, view on normal and deviant child behaviour may affect criterion assessment. Assessment of impairments may be even more subjective. For example, being among the youngest children in a class doubles the chance of being diagnosed and treated for ADHD (12,13). Apparently, teachers sometimes see relative immaturity as an indication of ADHD.

Despite these problems, ADHD is frequently presented as a disease or compared with a disease and even by professionals (7, p330). For example, referring to her recent well-publicised study (10), the researcher stated in the media (McFadden, 2010): 'Now we can say with confidence that ADHD is a genetic *disease* and that the brains of children with this condition develop differently to those of other children' (14). This conclusion was based on the study's finding that 15.6% of the children diagnosed with ADHD had chromosomal abnormalities called copy number variants (CNVs), compared with 7.5% of the controls. In other words, most children diagnosed with ADHD did not have CNVs and most children with CNVs did not have a diagnosis of ADHD. Hence, the general conclusion that the study proves ADHD to be primarily a genetic disease seems hardly justified.

What we call ADHD might best be seen as behaviours resulting from a very mixed bag of genetic and temperamental variations, immaturities or nonoptimal conditions of the central nervous system, cognitive, motor and sensory regulatory problems and motivational deficiencies, interacting with many environmental and societal influences. Seeing ADHD as an extreme of normal temperament has the added advantage of bringing in an evolutionary perspective. ADHD behaviours may once have had survival value, but do not match very well with expectations of functioning in modern society. If societal values determine that hyperactive and distractible behaviour is abnormal, we

Table 1 Examples of reification in informational sources for patients, parents and teachers

ADHD causes hyperactivity and restlessness, so use up that excess energy	http://www.ehow.com/way_5475224_focus-adhd-remedies.html (accessed on January 7 2014)
ADHD causes inattention because the child's brain is unable to focus for any period of time	http://www.attentiondeficit-add-adhd.com/bipolar-and-adhd.htm (accessed on January 7 2014)
ADD/ADHD causes inattention It's just hard to stay focused on what someone says to you, and how they are saying it	http://www.choosehelp.com/topics/adult-add-adhd/adult-add-adhd-how-to-gain-social-skills-by-improving-non-verbal-communication (accessed on January 7 2014)
If clinicians can help patients understand the disorder, offer a plausible rationale for how it causes their symptoms …	R.A. Barkley (7) Handbook ADHD, pp. 694

view possible associated biological differences as abnormalities. By representing ADHD as a disease, a neurobiological disorder, hyperactive and inattentive behaviours are attributed to an intrinsic condition of the child that needs medical treatment. This may facilitate overdiagnosis and pharmacological overtreatment (Table 2).

ASSUMPTION 3: ADHD IS PERSISTENT

In the literature, ADHD is often defined as a chronic disorder, an impairing lifelong condition (7, 15). However, the issue of ADHD persisting into adulthood is not as clear cut as is often suggested. Estimates of ADHD persistence depend heavily on disorder criteria, length of follow-up, attrition rate and reporting source. Biederman et al. (16) report inconsistent persistence rates in the ADHD literature, varying from 4% to 66%. Recently, important evidence for delayed brain maturation, as opposed to deviant development, in ADHD has emerged, especially in remitting cases (17). According to some experts, 'a majority of diagnosed young people no longer meet criteria for ADHD in adult life' (18), whereas others state that 'in the majority of cases ADHD persists into adult life' (19). To sum up, there is more evidence for speaking in terms of persisters and remitters than for representing ADHD as a persisting disorder period.

Biederman et al. (16) argue that subsyndromal forms of ADHD are associated with significant impairment and may therefore need clinical attention. The follow-up of the much-cited Multimodal Treatment Study of Children with ADHD (MTA) could be interpreted in the same vein. Although at the 8-year follow-up, only 30% of the rigorously diagnosed participants still met the DSM-IV criteria for ADHD, the ADHD group functioned significantly less well than a non-ADHD comparison group (20). Maybe the associated impairments are more chronic than core symptoms. Hence, it might be worthy to focus treatments more on childhood social, family and school problems and less on the pharmacological reduction in ADHD core symptoms.

ASSUMPTION 4: THE BEST TREATMENT FOR ADHD IS MEDICATION

Mitchell and Read (21) showed that information websites about ADHD are strongly biased towards bio-genetic aetiological explanations and that, in particular, drug company funded websites recommend medication rather than psycho-social treatments. Pharmaceutical companies

Table 2 Examples of presenting group differences as individual differences

		Preferred way
Neuroimaging has demonstrated 'Several structural differences to the brains of adults with ADHD compared with unaffected individuals, further supporting a diagnosis of ADHD in adults'.	Rösler, Casas, Konofal & Buitelaar *The World Journal of Biological Psychiatry*, 2010; 11: 684–698 (Conclusions)	Neuroimaging has demonstrated 'Several structural differences to the brains of *groups* of* adults with ADHD compared with *groups of* unaffected individuals, further supporting a diagnosis of ADHD in adults'.
'Functional MRI data show differences in brain functioning between ADHD and controls including some studies of drug naïve patients'.	Kooij et al. BMC Psychiatry 2010, 10:67 http://www.biomedcentral.com/1471-244X/10/67	'Functional MRI data show differences in brain functioning between groups of persons with ADHD and controls ….'
'Adults with ADHD have subtle volume reductions in the caudate and possibly other brain regions involved in attention and executive control supporting frontostriatal models of ADHD'	In: Seidman LJ, Biederman J, Liang L, Valera EM, Monuteaux MC, Brown A, Kaiser J, Spencer T, Faraone SV, Makris N. Biol Psychiatry. 2010 Epub ahead of print. Conclusion.	'*Groups of* adults with ADHD have subtle volume reductions in the caudate and possibly other brain regions ….'
'In subjects with ADHD, there is a thinning of the cortical surface in the right frontal lobe, which is present in the children, adolescents and in adults'.	In: Almeida LG, Ricardo-Garcell J, Prado H, Barajas L, Fernández-Bouzas A, Avila D, Martínez RB. J Psychiatr Res. 2010;44:1214–23. Conclusion.	'*In groups of subjects diagnosed* with ADHD, there is a thinning of the cortical surface in the right frontal lobe, which is present in *groups of* children, adolescents and in adults'.
'Children with ADHD show specific abnormalities on neuroimaging'.	Sharkey & Fitzgerald, in Handbook of ADHD, ed. Fitzgerald, Bellgrove & Gill, 2007	'*As a group*, children with ADHD show specific *differences* on neuroimaging'.
The neuroimaging literature shows evidence of structural brain abnormalities in individuals with ADHD, including smaller volumes in the frontal cortex, cerebellum, and subcortical structures.	Wilens, p.7, in editorial, pp. 6-8, JAACAP, January 2011	The neuroimaging literature shows evidence of structural brain *differences* in *groups of individuals diagnosed* with ADHD, including smaller volumes in the frontal cortex, cerebellum, and subcortical structures.

*Each time 'group differences' are mentioned, the studies should also have underlined that the differences only could be demonstrated on group level, not at the level of the individual child or adult.

often refer to the findings of the above-mentioned MTA study and this may also have contributed to the increase in ADHD medication use. In 1999, the initial results of the MTA study (22) suggested that stimulant treatment had a better effect on core ADHD symptoms than behavioural therapy. However, the study design meant that it was more likely to demonstrate a positive outcome for pharmacological treatment, as it was measured four to six months after the intensive phase of behaviour treatment (23) and when the medication treatment was at its most intensive phase.

However, parents and teachers were more satisfied with the psychosocial treatments, despite the larger symptom reduction with intensive medication management. The reason could be that the behavioural interventions in the MTA study were not primarily aimed at symptoms, but rather at impairments such as problems in social, academic and family functioning. These impairments might very well be more important to parents and teachers than ADHD symptoms *per se*. As one of the MTA investigators states (24): 'The impact of medication is generally larger on symptom scales, while behavioural treatments have a larger impact on functional measures'.

Interestingly, analyses of the long-term effects three (25) and eight (20) years after the beginning of the study showed no differences in outcome between medicated and non-medicated children. Also, contrary to expectations, at the 3-year follow-up, there was no evidence that stimulant treatment in childhood protected against the emergence of delinquency and substance use. A recent meta-analysis confirmed that treatment of ADHD with stimulant medication neither protects nor increases the risk of later substance use disorders (26).

On the basis of these results, we suggest that it may be worthwhile to reconsider the prominent place of stimulants in the treatment of ADHD. There is evidence that behavioural treatments are effective for treating ADHD (27). We are not empty-handed without medication as our first-line treatment.

ASSUMPTION 5: ADHD DIAGNOSIS SHOULD PRECEDE TREATMENT

While establishing a DSM diagnosis is useful for communication and research, it is not a prerequisite for treatment in clinical practice. Many childhood behaviour problems can probably be dealt with adequately without making a DSM diagnosis first. Besides, even if a DSM diagnosis can be established, it is often of limited value in planning treatment for the individual child and family. 'Lack of treatment specificity is the rule rather than the exception' according to key DSM-IV and DSM-5 Task Force members (28). In addition, pure classifications are exceptions than the rule.

In many cases, explanatory hypotheses about the behaviour of the individual child in its family and school provide the key targets for treatment. Unfortunately, it seems that economy shapes practice. Nowadays, many reimbursements for mental health care are based on DSM categories; this may have driven the tendency to focus on the presence or the absence of diagnostic criteria, rather than understanding the behaviour of the individual child.

HOW TO REDUCE OVER DIAGNOSIS WITHOUT RISKING UNDER TREATMENT?

In conclusion, ADHD is often considered to be a chronic disease that causes deviant behaviour and should be treated pharmacologically after elaborate diagnostics. We argued that this view is supported by five debatable assumptions, which may play a role in the rise in ADHD diagnosis and treatment.

Establishing the diagnosis of ADHD may have some drawbacks, which we summarised in a previous paper (29). The diagnosis may lead to self-fulfilling prophecies, such as negative parental and teacher expectations, which may be perceived by the child who, in response, may underperform. The diagnosis may also have a stigmatising effect and the knowledge of being diagnosed may harm the child's self-image and self-efficacy. Also, a DSM diagnosis may give the false impression that we understand the problem and can stop asking questions. Therefore, it is important to avoid false positives wherever possible. The clinical challenge is to find the optimal balance between the risks of overdiagnosis and underdiagnosis and treatment.

We argued that the effects of behavioural strategies are well established and that diagnosis according to DSM-5 is not a prerequisite for behavioural intervention. In the light of this, we suggest an approach based on stepped diagnosis and stepped care (30). In many cases, management of child behaviour problems could start with behaviour modification techniques without a confirmed diagnosis. In cases where this approach proves insufficient, the child is referred for psychiatric assessment and medication treatment when appropriate. This approach offers the added advantage of limiting the flood of children referred to specialist child and adolescent mental health services for ADHD evaluation.

COMPETING INTEREST

None.

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