Attention-deficit hyperactivity disorder: a conceptual debate

Irish Journal of Psychological Medicine (2018), **35**, 251–265.
doi:10.1017/ipm.2018.8

DEBATE

Attention-deficit hyperactivity disorder (ADHD): progress and controversy in diagnosis and treatment

D. M. Foreman*

Department of Child and Adolescent Psychiatry, PO85, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK

Knowledge about attention-deficit hyperactivity disorder (ADHD) is rapidly accumulating. Recent advances in diagnosis, genetics, neuroimaging, drug and non-drug treatments are considered, and the results are related to the critical attack on the ADHD diagnosis, which argues it a medicalising social construct, unhelpfully sustaining power relationships. The advances reviewed suggest that, while this attack can be conclusively dismissed as wrong and misleading, the phenomenological definition of ADHD is no longer sufficient for construct validity, though continues to be valuable as a guide for clinicians. The humanising and individualising concerns underlying the attack on the diagnosis could usefully be redirected to improving effective measurement of patient outcomes.

Received 24 August 2016; Revised 14 January 2018; Accepted 15 January 2018; First published online 19 June 2018

Key words: ADHD, anti-psychiatry, critical psychiatry, narrative review.

Attention-deficit hyperactivity disorder (ADHD) is a syndrome comprising inattention, impulsivity and over-activity in variable proportions, sufficient to cause impairment in functioning. It was initially understood as a disorder of childhood, but it is now recognised as affecting adults also (Kooij *et al.* 2010), and is well established within conventional psychiatry. However, psychiatry itself is currently changing rapidly. A new approach to diagnosis, Research Domain Criteria (RDoC) for research purposes has been proposed (National Institute for Mental Health, 2016), separate from the traditional Diagnostic and Statistical Manual (DSM) and International Classification of Diseases (ICD) systems, which also continue to evolve; DSM version 5 (DSM-5) having been published in 2013, and ICD-11 due in 2018. Attempts to automate diagnosis are gathering pace. Molecular genetics, epigenetics and neuroimaging are converging to enable accurate accounts of brain function in a range of mental states. The drive towards evidence-based practice has led to a much more systematic consideration of published research about treatment effectiveness. Meanwhile, the critical psychiatry movement continues to argue that the construct of ADHD exemplifies why the diagnoses employed by psychiatry are wrong and harmful. While some attacks on ADHD have simply been mendacious (Barrett, 2015), legitimate criticism has come from two directions. Some argue that ADHD is a medicalising social construct, originating in the United States but being globalised (Conrad & Bergey, 2014). Others claim, using participant observation, that the evidence on which the diagnosis of ADHD is justified is viewed through a distorting cultural lens, where the knowledge that is privileged is chosen to support a dominant power structure, 'the biomedical framework' (Moncrieff & Timimi, 2013). This leads to concerns that a diagnosis-led approach to the care of ADHD is, in important respects, dehumanising (Gambrill, 2014) and delivered for the benefit of vested interests, for example, the pharmaceutical industry (Phillips, 2006). This paper therefore considers how these new advances affect our understanding, diagnosis and treatment of ADHD.

* Address for correspondence: D. M. Foreman, Department of Child and Adolescent Psychiatry, PO85, Institute of Psychiatry, Psychology and Neuroscience, King's College London, 16 De Crespigny Park, London, SE5 8AF, UK.

(Email: David_Foreman@doctors.net.uk)

Diagnosis

RDoC

Though not currently intended to replace the clinical systems of ICD and DSM, RDoC greatly expands the traditional approach of symptoms and signs, to capture the changes in our understanding of psychiatric disorder arising from the insights achieved through genomics and imaging. As a newly developed research framework for general use, a detailed discussion of it is beyond the scope of this paper. For ADHD, it should

help clarify the set of gene–behaviour associations that remain currently unmapped, despite overall heritability of 0.7–0.8, including those which also affect disorders other than ADHD. For example, genetic abnormalities common to multiple disorders suggest a general vulnerability to psychopathology, irrespective of diagnostic type (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013). However, many of the RDoC domains have not been validated in children. While validation research is ongoing (Doyle, 2015) clinicians working with ADHD in children should be cautious about extrapolating RDoC-based research that they read.

DSM-5

The introduction of DSM-5 involved considerable controversy (Strakowski & Frances, 2012) some of it involving ADHD. There was a general concern that the boundaries of psychopathology were being extended too far: for ADHD, there were also specific concerns that modifying the age of onset criterion would impair the specificity of the diagnosis. Data are now accumulating to address these concerns. There appears to be a bimodal distribution for child and adult ADHD: very few child onset cases have symptoms by 38 years, and most adult cases developed symptoms after 12 years (Moffitt *et al.* 2015): the childhood group also had more marked cognitive and behavioural difficulties, though ADHD symptomatology met similar criteria. This has subsequently been replicated with another cohort (Agnew-Blais *et al.* 2016). Unlike age of onset, the 18 symptomatic criteria for ADHD were carried through largely unchanged. All were found to have discriminatory power and predict impairment, though not all to the same degree (Rosales *et al.* 2015). The domain model of RDoC thus offers a framework to research these commonalities and differences.

Diagnostic technologies

Traditional methods of diagnosing ADHD are resource-intensive, leading to significant delays in treatment, even when streamlined by guidelines (Foreman, 2010). The oldest technology available is rating scales, which typically are used for screening in ADHD, as they tend to over-identify (Foreman *et al.* 2008). Structured clinical interviewing provides reliable assessments (Goodman *et al.* 2000). Advances in information processing have enabled computer-supported methods to be developed, which offer potential advantages in time, either through not requiring the diagnostician to be present for data collection (Foreman *et al*. 2009), or using combinations of tests with computerised algorithms and cognitive testing to provide diagnoses (Hall *et al.* 2014), though these last are still in development. This range of approaches offers an opportunity to test one prediction arising from ADHD diagnostic criticisms. Were the diagnosis arising from a cultural lens, rates would vary significantly across different cultures and times, given their different 'distances' from the dominant viewpoint. Also, combining culture-independent cognitive tests with the conventional diagnosis would reduce diagnostic reliability, as the cultural bias would be diluted. A recent systematic review and meta-regression analysis were able to distinguish between the methodological and geographical or study year variance components of prevalences (Polanczyk *et al.* 2014). Contrary to the cultural lens hypothesis, it found significant differences between studies could only be explained by differences in their methodologies. The cognitive test used most frequently in relation to ADHD diagnosis is the Continuous Performance Test (CPT) for attention, while activity has been measured using actigraphy. Unfortunately, most studies have used these as independent measures to predict diagnosis, using quite small sample sizes. The results found were variable (Hall *et al.* 2015) consistent with the variable discriminatory power of different individual symptoms of ADHD identified by Rosales *et al.* (2015). The cultural lens hypothesis predicts that diagnostic reliability should be reduced when such tests are *added* to existing clinical assessments, as clinical support. Little research on this has been undertaken, but a combination of CPT and actigraphy measurement appeared to improve ADHD repeat reliability (Vogt & Shameli, 2011) and also improve discriminant validity between ADHD and Autism (Groom *et al.* 2016). These results are not consistent with the claim that the diagnostic category of ADHD results from cultural bias.

Genetics

Though the high heritability of ADHD has been known for some time, unequivocal evidence, unconfounded with potential environmental effects was identified in 2010, when an international population with ADHD was shown to have a greater proportion of copy number variants than controls (Williams *et al.* 2010). It also identified the genetic overlap between ADHD and other disorders that have since been replicated (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013) and so contributed to the development of the RDoC project discussed above. Conceptually, it has meant that the difficulties associated with ADHD diagnosis are no longer grounded in questions of social construction (Quinn & Lynch, 2016) but of measurement, as it now seems clear that a purely phenomenological account of ADHD (as provided

https://doi.org/10.1017/ipm.2018.8 Published online by Cambridge University Press

by DSM or ICD) is inadequate for determination of aetiology. However, a recent meta-analysis (Middeldorp *et al.* 2016) has suggested that either dichotomous or dimensional measures of ADHD capture the genetically common phenotype of the disorder, while differing symptom profiles reflect different proportions of single nucleotide polymorphisms, which is consistent with the phenomenological observations of Rosales *et al.* (2015). So, the diagnostic approach adopted by ICD and DSM, while inadequate for achieving full construct validity, is sufficient for predictive validity.

Evolutionary genetics and ADHD

Darwin's principles of natural selection imply that either the ADHD phenotype itself conveys a reproductive advantage in our environment or the genetic variability associated with it does. A recent study of very low levels of ADHD symptomatology attempted to explore this (Greven *et al.* 2016), finding, for this group, low heritability with high non-shared and shared environmental influences, with significantly associated advantage. While evolutionary theories about the value of the ADHD phenotype have been proposed, none have been validated (Thagaard *et al.* 2016). This evidence points towards the associated genetic variability being associated with advantage, rather than the phenotype itself. So, taking a strongly biological and empirical stance in relation to ADHD leads to a conservative position on either eugenic or genetically engineered attempts to eradicate the disorder, as we are currently ignorant of the negative impact of reducing our genetic variability in this way. Advances in the genetics of ADHD thus serve to prevent, rather than promote, potentially dehumanising programmes of care.

Neuroimaging

By analogy with the Human Genome Project, the Human Connectome Project (HCP) (National Institutes for Health, 2016) seeks to provide an atlas of the human 'connectome', that is, a comprehensive map of neural connections in the brain. It employs a twin/sibling design, thus facilitating genetic studies, and can achieve unparalleled levels of resolution. Its measures have been specifically chosen to map onto RDoC. Unfortunately, at present no paediatric HCP exists, but clearly will be needed if the advances this project offers are to be directed towards ADHD (Baroni & Castellanos, 2015).

At present, imaging offers a persuasive, but not conclusive, an account of the neurological basis for ADHD phenomenology. This is probably due to methodological problems, in particular, the small sample sizes used in studies employing this very expensive technology. This underpins the future value of HCP-based studies. In the meantime, meta-analyses can increase effective sample size. They may also propagate error, which can be greater in smaller studies, so their own results tend to be over-inflated, but only modestly (Button *et al.* 2013). So, carefully designed meta-analyses can provide more accurate assessments of imaging findings, with an acceptable margin of error.

Functional neuroanatomy of ADHD

From meta-analytic studies, there is reliable evidence that the right-lateralised ventral attention network (Corbetta Lab, 2016), and the fronto-parietal central executive networks (Menon, 2015) are hypoactive in individuals with ADHD (Hart *et al.* 2013), which correspond to the attentional and impulsive phenomenology of ADHD, respectively. The co-heritability of both ADHD traits and lower levels of measured executive function (Crosbie *et al.* 2013) provides convergent validity. Imaging also confirms fronto-striatal dysfunction (Castellanos & Proal, 2012) and some differences between children and adults, though interpreted developmentally at the time (Frodl & Skokauskas, 2012) converge with the findings of adult–child differences discussed above. Both developmental and cross-sectional differences in the ventral striatum have also been identified, consistent with the abnormal reward sensitivity typical of ADHD (Baroni & Castellanos, 2015).

Lack of evidence for a biological substrate to ADHD has been a key component in arguing that the diagnosis is inappropriate medicalisation of a social phenomenon (Lindstrøm, 2012). The last critical review of neuroimaging in ADHD was published 7 years ago (Leo & Cohen, 2009) and, while correctly identifying the problems of individual neuroimaging studies, did not consider meta-analytic methodology. The convergence of these meta-analyses with genetic, neurocognitive and phenomenological descriptions of ADHD confirm the existence of a biological substrate to the phenomenological syndrome, which has not yet been characterised fully.

Drug treatment of ADHD

Following studies such as the multimodal treatment for ADHD (Jensen *et al.* 1999) it became generally accepted that there was sufficient evident to be confident in prescribing stimulant medication, most frequently methylphenidate, to children with ADHD. However, this has become contested, following a recent Cochrane Systematic Review (Storebø *et al.* 2015), which has been challenged by other expert groups (Banaschewski *et al*. 2016). The disagreement is a technical one between expert meta-analysts, and beyond

https://doi.org/10.1017/ipm.2018.8 Published online by Cambridge University Press

the scope of this article. However, these two groups have provided competing estimates of effect size, and it is instructive to compare the difference between them. For teacher-rated reduction in ADHD symptoms, the effect sizes are estimated as 0.77 (Storebø *et al.* 2015) and 0.89 (Banaschewski *et al.* 2016), giving a difference of 0.12. This corresponds to a 4% difference in treatment efficacy, estimated as a group-level improvement, where the lower estimate of efficacy would be just below 79% (Coe, 2002). So, while the debate is of importance regarding appropriate standards for meta-analysis and future research, the findings themselves do not greatly modify prior conclusions regarding the efficacy of stimulant medication for ADHD.

The critical account of ADHD does not deny the efficacy of the medication. Instead, it argues that, because the medication is being used as a means of social control, the adverse effects of this must outweigh any benefits, as there is no psychopathology to treat. Therefore, measures of symptomatic change do not address their argument. More appropriate are measures of quality of life (QoL). These broad measures of well-being are sensitive to many everyday aspects of life, not necessarily directly connected with symptomatology (Bai & Lazenby, 2015). The Cochrane review just discussed included three QoL studies: it reported the effect size as being 0.61, equivalent to an average group-level improvement of around 73%. However, only one, parent-rated measure was used, the Child Health Questionnaire (CHQ). Though the CHQ has been validated for ADHD (Rentz *et al.* 2004) the use of a parent-rating scale is open to challenge: a systematic review of QoL rating scales in ADHD has found variable self-reports in both ADHD-related QoL impairment, and in correlation with parent-rated scores (Danckaerts *et al.* 2009). One subsequent paper reported a self-rated QoL improvement associated with an improvement in ADHD control due to change of medication (Kordon *et al.* 2011) but the study was drug-company sponsored.

It seems that there is no more than suggestive evidence that self-rated QoL is improved by medication management of ADHD, the uncertainty arising from methodological difficulties in QoL measurement. However, the critical model of medication management implies that children's QoL would decrease, and no evidence for this was found.

Non-drug treatments for ADHD

In 2013, our understanding of non-drug treatments for ADHD changed. A systematic review (Sonuga-Barke *et al.* 2013) reported effect sizes which included only (probably) blinded studies, as well effect sizes that included unblinded ones. It focussed on core ADHD symptoms, that is, inattention, impulsivity and restlessness, but not behavioural problems, in relation to dietary and a variety of psychological interventions. It found that, when appropriate blinding was used, a small effect for free fatty acid supplementation (0.16) and modest effect size for artificial food colour exclusion (0.42) remained, but the effect of any psychological intervention upon core symptoms of ADHD was non-significant. This contrasted with effect sizes of 0.4–0.64 for psychological treatments when unblinded studies were included. An overlapping review of just cognitive training for ADHD, which also distinguished between blinded and unblinded ratings, obtained similar results (Rapport *et al.* 2013). To date, no study has appeared which has conclusively altered these findings: those with positive effects were unblinded for ADHD core symptoms; blinded studies found no effects. A possible exception was a sleep intervention study (Hiscock *et al.* 2015) which identified improvement in both unblinded ADHD assessment and blinded working memory assessment 6 months after the intervention. However, both the working memory and ADHD symptomatic improvement could simply be related to the cognitive benefits of better sleep. Consistent with this, the authors reported a *post-hoc* mediation analysis which ascribed 33% of the ADHD effect to better sleep. Exercise has also been used for ADHD treatment, and a recent meta-analysis has reported a moderate effect size for executive function (Vysniauske *et al.* 2016). Unfortunately, significant publication bias and poor study quality means this result is almost certainly exaggerated: only three additional negative studies would be required to turn the effect size non-significant. Interest in non-invasive brain stimulation for ADHD is increasing, but so far positive results have been at case study level (Rubio *et al.* 2016).

The definitive critical textbook on ADHD (Timimi & Leo, 2008), consistent with the critical formulation of ADHD as a social construct, recommends a range of psychosocial treatments, and these recommendations have not changed since. It can be seen that these are unlikely to be effective on core symptoms of ADHD, and reported benefits are likely to result from observer effects.

Conclusions

Recent advances have clarified the epidemiology, genetics and neurobiology of ADHD, so claims that ADHD is solely a social construct can be conclusively refuted. While the phenomenological approaches of DSM and ICD are no longer sufficient to provide construct validity for ADHD, they provide sufficient predictive validity to be valuable as guidance for clinicians, informing effective treatment plans. Furthermore, the alternative of formulating ADHD as a medicalisation of a social predicament (Taylor, 1979) leads naturally to a psychosocial intervention, which will not improve the

https://doi.org/10.1017/ipm.2018.8 Published online by Cambridge University Press

core symptoms of the disorder. However, showing that the critical formulation is wrong and misguided does not address all the concerns that this response to ADHD raises. At present, the evidence we use is based on group-level data (Baroni & Castellanos, 2015). However, the treatments we deliver for diagnosed ADHD are to individuals; symptom profiles differ between them and are confounded with the individuals' other characteristics. The implications of this were illustrated by Oliver Sack's account of Witty Ticcy Ray (Sacks, 1992), who regulated the level of his medication according to his social setting. These considerations point towards a sensitive use of outcome measures, in particular, QoL. These measures appear less well developed than symptom scores, and when they are employed (e.g. as Patient-Rated Outcomes), clinicians appear less responsive to them than to symptom changes (Greenhalgh, 2009). This is an area where the critical psychiatry movement's concern with sensitivity to individual differences, and concern to protect patients' humanity, could gain useful traction.

Conflicts of Interest

None.

Financial Support

No funding body was involved in the preparation of this manuscript.

Ethical Standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committee on human experimentation with the Helsinki Declaration of 1975, as revised in 2008.

References

Agnew-Blais JC, Polanczyk GV, Danese A, Wertz J, Moffitt TE, Arseneault L (2016). Evaluation of the persistence, remission, and emergence of attention-deficit/hyperactivity disorder in young adulthood. *JAMA Psychiatry* **73**, 713–720.

Bai M, Lazenby M (2015). A systematic review of associations between spiritual well-being and quality of life at the scale and factor levels in studies among patients with cancer. *Journal of Palliative Medicine* **18**, 286–298.

Banaschewski T, Gerlach M, Becker K, Holtmann M, Döpfner M, Romanos M (2016). Trust, but verify. The errors and misinterpretations in the Cochrane analysis by O. J. Storebo and colleagues on the efficacy and safety of methylphenidate for the treatment of children and adolescents with ADHD. *Zeitschrift für Kinder- und Jugendpsychiatrie und Psychotherapie* **44**, 307–314.

Baroni A, Castellanos FX (2015). Neuroanatomic and cognitive abnormalities in attention-deficit/hyperactivity disorder in the era of 'high definition' neuroimaging. *Current Opinion in Neurobiology* **30**, 1–8.

Barrett S (2015). Some notes on ADHD and Peter R. Breggin's unfair attack on Ritalin (http://www.quackwatch.com/11Ind/breggin.html). Accessed 20 August 2016.

Button KS, Ioannidis JPA, Mokrysz C, Nosek BA, Flint J, Robinson ESJ, Munafò MR (2013). Power failure: why small sample size undermines the reliability of neuroscience. *Nature Reviews Neuroscience* **14**, 365–376.

Castellanos FX, Proal E (2012). Large-scale brain systems in ADHD: beyond the prefrontal-striatal model. *Trends in Cognitive Sciences* **16**, 17–26.

Coe RJ (2002). It's the effect size, stupid: what effect size is and why it is important (http://www.leeds.ac.uk/educol/documents/00002182.htm). Accessed 7 April 2013.

Conrad P, Bergey MR (2014). The impending globalization of ADHD: notes on the expansion and growth of a medicalized disorder. *Social Science & Medicine* **122**, 31–43.

Corbetta Lab (2016). Washington University in St. Louis - Medical School - Corbetta Lab (http://www.nil.wustl.edu/labs/corbetta/research_attention_networks.html). Accessed 21 August 2016.

Crosbie J, Arnold P, Paterson A, Swanson J, Dupuis A, Li X, Shan J, Goodale T, Tam C, Strug LJ, Schachar RJ (2013). Response inhibition and ADHD traits: correlates and heritability in a community sample. *Journal of Abnormal Child Psychology* **41**, 497–507.

Cross-Disorder Group of the Psychiatric Genomics Consortium (2013). Identification of risk loci with shared effects on five major psychiatric disorders: a genome-wide analysis. *The Lancet* **381**, 1371–1379.

Danckaerts M, Sonuga-Barke EJS, Banaschewski T, Buitelaar J, Döpfner M, Hollis C, Santosh P, Rothenberger A, Sergeant J, Steinhausen H-C, Taylor E, Zuddas A, Coghill D (2009). The quality of life of children with attention deficit/hyperactivity disorder: a systematic review. *European Child & Adolescent Psychiatry* **19**, 83–105.

Doyle AE (2015). Commentary: insights from across diagnostic boundaries: ADHD in the RDoC era – a commentary on Scerif and Baker (2015). *Journal of Child Psychology and Psychiatry* **56**, 274–277.

Foreman DM (2010). The impact of governmental guidance on the time taken to receive medication for ADHD in England. *Child and Adolescent Mental Health* **15**, 12–17.

Foreman DM, Dack S, Ford T (2008). Assessing the diagnostic accuracy of the identification of hyperkinetic disorders following the introduction of government guidelines in England. *Child and Adolescent Psychiatry and Mental Health* **2**, 1–7. https://doi.org/10.1186/1753-2000-2-32

Foreman DM, Morton S, Ford T (2009). Exploring the clinical utility of the Development And Well-Being Assessment (DAWBA) in the detection of hyperkinetic disorders and associated diagnoses in clinical practice. *Journal of Child Psychology and Psychiatry* **50**, 460–470.

Frodl T, Skokauskas N (2012). Meta-analysis of structural MRI studies in children and adults with attention deficit hyperactivity disorder indicates treatment effects. *Acta Psychiatrica Scandinavica* **125**, 114–126.

Gambrill E (2014). The Diagnostic and Statistical Manual of Mental Disorders as a major form of dehumanization in the modern world. *Research on Social Work Practice* **24**, 13–36.

Goodman R, Ford T, Richards H, Gatward R, Meltzer H (2000). The Development and Well-Being Assessment: description and initial validation of an integrated assessment of child and adolescent psychopathology. *Journal of Child Psychology & Psychiatry & Allied Disciplines* **41**, 645–655.

Greenhalgh J (2009). The applications of PROs in clinical practice: what are they, do they work, and why? *Quality of Life Research* **18**, 115–123.

Greven CU, Merwood A, Meer JM, Haworth C, Rommelse N, Buitelaar JK (2016). The opposite end of the attention deficit hyperactivity disorder continuum: genetic and environmental aetiologies of extremely low ADHD traits. *Journal of Child Psychology and Psychiatry* **57**, 523–531.

Groom MJ, Young Z, Hall CL, Gillott A, Hollis C (2016). The incremental validity of a computerised assessment added to clinical rating scales to differentiate adult ADHD from autism spectrum disorder. *Psychiatry Research* **243**, 168–173.

Hall CL, Valentine AZ, Groom MJ, Walker GM, Sayal K, Daley D, Hollis C (2015). The clinical utility of the continuous performance test and objective measures of activity for diagnosing and monitoring ADHD in children: a systematic review. *European Child & Adolescent Psychiatry* **25**, 677–699.

Hall CL, Walker GM, Valentine AZ, Guo B, Kaylor-Hughes C, James M, Daley D, Sayal K, Hollis C (2014). Protocol investigating the clinical utility of an objective measure of activity and attention (QbTest) on diagnostic and treatment decision-making in children and young people with ADHD —'Assessing QbTest Utility in ADHD'(AQUA): a randomised controlled trial. *BMJ Open* **4**, e006838.

Hart H, Radua J, Nakao T, Mataix-Cols D, Rubia K (2013). Meta-analysis of functional magnetic resonance imaging studies of inhibition and attention in attention-deficit/hyperactivity disorder: exploring task-specific, stimulant medication, and age effects. *JAMA Psychiatry* **70**, 185–198.

Hiscock H, Sciberras E, Mensah F, Gerner B, Efron D, Khano S, Oberklaid F (2015). Impact of a behavioural sleep intervention on symptoms and sleep in children with attention deficit hyperactivity disorder, and parental mental health: randomised controlled trial. *BMJ* **350**, h68.

Jensen PS, Arnold LE, Richters JE, Severe JB, Vereen D, Schiller E, Hinshaw SP, Elliou GR, Conners CK, Wells KC, Swanson JM, Wigal T, Cantwell DP, Abikoff HB, Hechtman L, Newcorn JH (1999). A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity 'strong' disorder. *Archives of General Psychiatry* **56**, 1073–1086.

Kooij SJJ, Bejerot S, Blackwell A, Caci H, Casas-Brugué M, Carpentier PJ, Edvinsson D, Fayyad J, Foeken K, Fitzgerald M (2010). European consensus statement on diagnosis and treatment of adult ADHD: the European Network Adult ADHD. *BMC Psychiatry* **10**, 67.

Kordon A, Stollhoff K, Niederkirchner K, Mattejat F, Rettig K, Schäuble B (2011). Exploring the impact of once-daily OROS® methylphenidate (MPH) on symptoms and quality of life in children and adolescents with ADHD transitioning from immediate-release MPH. *Postgraduate Medicine* **123**, 27–38.

Leo J, Cohen D (2009). A critical review of ADHD neuroimaging research. In *Rethinking ADHD: From Brain to Culture* (ed. S. Timimi and J. Leo), pp. 92–129. Palgrave Macmillan: England.

Lindstrøm JA (2012). Why attention-deficit/hyperactivity disorder is not a true medical syndrome. *Ethical Human Psychology and Psychiatry* **14**, 61–73.

Menon V (2015). Salience network. In *Brain Mapping: An Encyclopedic Reference*, vol. 2 (ed. A. W. Toga), pp. 597–611. Academic Press: Elsevier.

Middeldorp CM, Hammerschlag AR, Ouwens KG, Groen-Blokhuis MM St., Pourcain B, Greven CU, Pappa I, Tiesler CMT, Ang W, Nolte IM, Vilor-Tejedor N, Bacelis J, Ebejer JL, Zhao H, Davies GE, Ehli EA, Evans DM, Fedko IO, Guxens M, Hottenga J-J, Hudziak JJ, Jugessur A, Kemp JP, Krapohl E, Martin NG, Murcia M, Myhre R, Ormel J, Ring SM, Standl M, Stergiakouli E, Stoltenberg C, Thiering E, Timpson NJ, Trzaskowski M, van der Most PJ, Wang C, Nyholt DR, Medland SE, Neale B, Jacobsson B, Sunyer J, Hartman CA, Whitehouse AJO, Pennell CE, Heinrich J, Plomin R, Smith GD, Tiemeier H, Posthuma D, Boomsma DI (2016). A genome-wide association meta-analysis of attention-deficit/hyperactivity disorder symptoms in population-based paediatric cohorts. *Journal of the American Academy of Child & Adolescent Psychiatry* **55**, 896–905.e6.

Moffitt TE, Houts R, Asherson P, Belsky DW, Corcoran DL, Hammerle M, Harrington H, Hogan S, Meier MH, Polanczyk GV, Poulton R, Ramrakha S, Sugden K, Williams B, Rohde LA, Caspi A (2015). Is adult ADHD a childhood-onset neurodevelopmental disorder? Evidence from a four-decade longitudinal cohort study. *American Journal of Psychiatry* **172**, 967–977.

Moncrieff J, Timimi S (2013). The social and cultural construction of psychiatric knowledge: an analysis of NICE guidelines on depression and ADHD. *Anthropology & Medicine* **20**, 59–71.

National Institutes for Health (2016). Human Connectome Project | Mapping the human brain connectivity (http://www.humanconnectomeproject.org/). Accessed 21 August 2016.

National Institute for Mental Health (2016). Research Domain Criteria (RDoC) (http://www.nimh.nih.gov/research-priorities/rdoc/index.shtml). Accessed 20 August 2016.

Phillips CB (2006). Medicine goes to school: teachers as sickness brokers for ADHD. *PLOS Medicine* **3**, e182.

Polanczyk GV, Willcutt EG, Salum GA, Kieling C, Rohde LA (2014). ADHD prevalence estimates across three decades: an updated systematic review and meta-regression analysis. *International Journal of Epidemiology* **43**, 434–442.

Quinn M, Lynch A (2016). Is ADHD a 'real' disorder? *Support for Learning* **31**, 59–70.

Rapport MD, Orban SA, Kofler MJ, Friedman LM (2013). Do programs designed to train working memory, other executive functions, and attention benefit children with ADHD? A meta-analytic review of cognitive, academic, and behavioral outcomes. *Clinical Psychology Review* **33**, 1237–1252.

Rentz AM, Matza LS, Secnik K, Swensen A, Revicki DA (2004). Psychometric validation of the child health questionnaire (CHQ) in a sample of children and adolescents with attention-deficit/hyperactivity disorder. *Quality of Life Research* **14**, 719–734.

Rosales AG, Vitoratou S, Banaschewski T, Asherson P, Buitelaar J, Oades RD, Rothenberger A, Steinhausen H-C, Faraone SV, Chen W (2015). Are all the 18 DSM-IV and DSM-5 criteria equally useful for diagnosing ADHD and predicting comorbid conduct problems? *European Child & Adolescent Psychiatry* **24**, 1325–1337.

Rubio B, Boes AD, Laganiere S, Rotenberg A, Jeurissen D, Pascual-Leone A (2016). Noninvasive brain stimulation in pediatric attention-deficit hyperactivity disorder (ADHD): a review. *Journal of Child Neurology* **31**, 784–796.

Sacks O (1992). Tourette's syndrome and creativity. *BMJ: British Medical Journal* **305**, 1515.

Sonuga-Barke EJS, Brandeis D, Cortese S, Daley D, Ferrin M, Holtmann M, Stevenson J, Danckaerts M, van der Oord S, Döpfner M, Dittmann RW, Simonoff E, Zuddas A, Banaschewski T, Buitelaar J, Coghill D, Hollis C, Konofal E, Lecendreux M, Wong ICK, Sergeant J (2013). Nonpharmacological interventions for ADHD: systematic review and meta-analyses of randomized controlled trials of dietary and psychological treatments. *American Journal of Psychiatry* **170**, 275–289.

Storebø OJ, Ramstad E, Krogh HB, Nilausen TD, Skoog M, Holmskov M, Rosendal S, Groth C, Magnusson FL, Moreira-Maia CR, Gillies D, Buch Rasmussen K, Gauci D, Zwi M, Kirubakaran R, Forsbøl B, Simonsen E, Gluud C (2015). Methylphenidate for children and adolescents with attention deficit hyperactivity disorder (ADHD). *Cochrane Database of Systematic Reviews* issue 11, CD009885.

Strakowski SM, Frances AJ (2012). What's wrong with DSM-5?, Medscape (http://www.medscape.com/viewarticle/763886). Accessed 20 August 2016.

Taylor D (1979). The components of sickness: diseases, illnesses, and predicaments. *The Lancet* **314**, 1008–1010.

Thagaard MS, Faraone SV, Sonuga-Barke EJ, Østergaard SD (2016). Empirical tests of natural selection-based evolutionary accounts of ADHD: a systematic review. *Acta Neuropsychiatrica* **28**, 249–256.

Timimi S, Leo J (2008). *Rethinking ADHD: From Brain to Culture*. Palgrave Macmillan: Basingstoke.

Vogt C, Shameli A (2011). Assessments for attention-deficit hyperactivity disorder: use of objective measurements. *The Psychiatrist Online* **35**, 380–383.

Vysniauske R, Verburgh L, Oosterlaan J, Molendijk ML (2016). The effects of physical exercise on functional outcomes in the treatment of ADHD a meta-analysis. *Journal of Attention Disorders*, https://doi.org/10.1177/1087054715627489

Williams NM, Zaharieva I, Martin A, Langley K, Mantripragada K, Fossdal R, Stefansson H, Stefansson K, Magnusson P, Gudmundsson OO, Gustafsson O, Holmans P, Owen MJ, O'Donovan M, Thapar A (2010). Rare chromosomal deletions and duplications in attention-deficit hyperactivity disorder: a genome-wide analysis. *The Lancet* **376**, 1401–1408.

doi:10.1017/ipm.2018.9

Attention-deficit hyperactivity disorder: a critique of the concept

S. Timimi*

Lincolnshire Partnership NHS Foundation Trust, Child and Family Services, Horizon Centre, Lincoln, UK

Attention-deficit hyperactivity disorder (ADHD) is a fact of culture rather than a fact of nature. For a diagnosis like ADHD to be scientifically useful you need to show that the concept leads to advancement of knowledge around causes. For it to be clinically useful, you need to show that use of the concept leads to improved clinical outcomes. As neither can be convincingly demonstrated, ADHD is unlikely to be either scientifically or clinically useful and the concept is well past its use-by date.

Received 28 October 2016; Revised 16 January 2018; Accepted 16 January 2018

Key words: ADHD, children, critical psychiatry, outcomes, stimulants, treatment, diagnosis.

* Address for correspondence: S. Timimi, Lincolnshire Partnership NHS Foundation Trust, Child and Family Services, Horizon Centre, Monson Street, Lincoln LN5 7RZ, UK.
(Email: stimimi@talk21.com)

Does the concept of attention-deficit hyperactivity disorder (ADHD) help advance scientific knowledge?

In psychiatry (apart from the dementias and a few other known organically based conditions), there is no