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Title: The overdiagnosis of Attention-Deficit/Hyperactivity Disorder: An ideological perspective or cause for concern?

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Abstract

Increased prescription rates for ADHD are viewed from two perspectives. Proponents of the disorder consider it to be due to better identification and improved medication. Critics consider it to be overdiagnosed and overmedicated. An International Consensus Statement was released in 2002 strongly promoting a proponent view. The statement was responded to by a group of critics claiming it was an attempt to stifle debate. In a reply to the critique it was specified that ADHD had been verified via Wakefield's Harmful Dysfunction (HD) analysis. This implies that a failed biological and evolutionary mechanism relative to ADHD had been identified; specifically a deficit in behavioural inhibition. In an attempt to verify this claim the theory of the first signatory Russell Barkley is examined. Barkley attributes his model of dysfunction to a "profound" work by the mathematician and documentary commentator the late Jacob Bronowski. Bronowski's theory is an articulation of differences between animal and human language. It is not listed on any scholarly database and by Bronowski's own admission is the view of an amateur. Barkley's linking of everyday behaviours to Bronowski's work is not supported, nor is his claim that Bronowski attributed these behaviours to the prefrontal cortex. As a number of the signatories of the consensus statement have competing interests via their links to pharmaceutical companies questions are raised about the real purpose of the statement.

The overdiagnosis of Attention-Deficit/Hyperactivity Disorder: An ideological perspective or cause for concern?

Attention-Deficit/Hyperactivity Disorder (ADHD) is a diagnostic label that initially appeared in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III), (American Psychiatric Association, 1980) as Attention Deficit Disorder (ADD) with or without hyperactivity, before becoming ADHD in a revised version (DSM-III-R; American Psychiatric Association, 1987). It is now one of the most widely diagnosed childhood disorders with treatment often involving the use of psychotropic medication (Rosenman, 2006). A review of Australian government databases of dispensed stimulant medications between 2002 and 2009 found there was a 300% increase in methylphenidate prescriptions with boys receiving four times the number of prescriptions compared to girls (Hollingworth et al., 2011). Hollingworth et al. called for further research to determine whether such prescribing rates were appropriate.

Whilst many factors are likely to influence prescribing rates, the appropriateness of such an increase may in part, depend on an individual's perspective (Whitley, 2011). Proponents claim that ADHD is a neurobiological condition that is partially due to genetic/biological causes. They argue that treatments with medications are safe and effective; that increased prescribing rates are due to improvements in diagnostic criteria, as well as there being greater access to appropriate pharmaceutical treatments. Critics are more likely to take the view that a 300% increase in stimulant prescriptions is indicative of overdiagnoses and over medication.

A much-cited handbook for the diagnosis and treatment of ADHD (Barkley, 2006) clearly endorses the former view. Included in the text is a reprint of a self-described International Consensus Statement (Barkley et al., 2002) written by a consortium of experts. They express concern about what they consider to be the inaccurate portrayal of ADHD by the media. They say that ADHD is a non-benign medical disorder, but many with the disorder are not receiving treatment. It is their belief that this is in part due to the irresponsibility of the media; hence their reason for writing the statement. They urge the media, in the strongest of terms to depict ADHD in the manner it is depicted in science, which is as a valid disorder

The statement is critiqued by another group of concerned professionals (Timimi et al., 2004) refuting claims that there is evidence to identify ADHD as a specific neurological disorder. They assert that Barkley et al.'s (2002) insistence that untreated ADHD leads to impairment and harm is not empirically supported and cite evidence that suggests drug treatment may at best be inconsequential. Concern is also expressed about the ongoing increase in the prescription of psychotropic drugs particularly to preschoolers. Timimi et al. noted that many of the signatories of the statement are well known advocates of drug treatment for children, and should have declared their financial interests and links with the pharmaceutical companies. A cultural perspective of ADHD is then given and the statement concludes that the debate is not yet over.

Barkley and a smaller group of co-endorsers respond to the critique. Whilst taking exception to the suggestion of pharmaceutical involvement, Barkley et al. (2004) address points raised by Timimi et al (2004). The tone of the response might reasonably be seen as patronising, even at times scathing, with an accompanying tone of having the scientific "high-ground". Aside from using the word "science" in some

form 25 times, they insist that any “debate” over the validity of ADHD as a genuine disorder exists only in the popular media. They claim the alternative explanations of ADHD-type behaviours put forward by Timimi et al. are “historical dead-ends in understanding child psychopathology” (p.69). Barkley et al. state that Timimi et al. provide no standards by which the merits of their argument can be judged, whereas their standards are “obvious” most notably that: “valid disorders are failures or severe deficiencies in psychological adaptations (functional mental mechanisms) that are universal to humans” (p. 66). This specific criterion is attributed to Wakefield (1997, 1999).

Thus it is reasonable to assume that the merits of Barkley et al.’s (2002, 2004) argument supporting the validity of ADHD can be resolved by examining the references made to Wakefield (1997, 1999). Wakefield (1992) believed the DSM-III’s definition of disorder to be insufficient. Thus, he proposed the “HD concept of analysis” as an alternative. By 1997, Wakefield claimed that the analysis had been well scrutinised, critiqued, and defended, and that its explanatory power was superior to its rivals, a view endorsed by Spitzer (1999), the chairman of the DSM-III (1980) and DSM-III-R (1987) Task Forces.

The central concept of Wakefield’s (1992, 1997, 1999) theory is that a medical disorder can be said to exist when a biological mechanism is unable to perform the function that it has evolved to perform. This inability must be caused by a failure within the mechanism and not be due to an expected response to stressful or problematic social environments. Identification of a failed biological mechanism is reliant on a scientific judgment. Here, Wakefield (1997) specifies that harm alone is not enough for disorder. A condition can only be considered disordered if it fulfills the criteria of dysfunction. Thus, with respect to ADHD it is important to ascertain the

mechanism that has failed in people diagnosed with ADHD. The consensus statement claims that ADHD is due to a deficit in behavioral inhibition. Barkley (2006) cites his own theory as being relative to such a view (see p.94).

Barkley (1997) defines behavioural inhibition as the ability to control an initial response for a period of time so as to allow relevant cognitive activities to occur that facilitate alternative decision-making. At the same time, the individual must be able to ignore any competing events or responses. A framework Barkley (1994, 1997) drew from linking ADHD to poor behavioral inhibition is an essay written by Bronowski (1977) described by Barkley as “profound” (p.19, 1994). A search of all databases using Thomson Reuters ‘Web of Knowledge’ did not list the book suggesting it is not catalogued as a scholarly work. This is problematic in light of Barkley’s claims.

Bronowski’s (1977) theory, according to Barkley (1994, 1997) matched his view that the principal feature of ADHD is impairment in the ability to delay responding, that is, a deficiency in behavioural inhibition. Whilst acknowledging that Bronowski’s theory is one of “delayed responding within the context of distinctions between human and animal languages” (1994, p.20), Barkley (1994) sees no reason why it cannot be extended to ADHD. This is presumably because both theories relate to an initial delay in response to external stimuli.

In Bronowski’s (1997) theory the delay is an essential difference in humans relative to other species. This difference in response, according to Bronowski enabled humans to separate the emotional charge surrounding a message, something he called the *separation of affect*. He believed that this led to the development of memory, foresight, hindsight and imagination something he termed *prolongation*. The next stage was the ability to internalise speech, which he called *internalization*. Finally he

proposed *reconstitution*, this is the ability to analysis and synthesis one's thoughts. In Bronowski's view it is this that gives language its current depth and complexity.

From this Barkley formulated his theory by naming the initial delay to external stimuli *behavioral inhibition*. The other stages from Bronowski's theory are lumped together and termed *executive functions*. Their functioning is dependent on the correct functioning of behavioural inhibition this in turn leads to normal behaviour, or not in the case of ADHD. Thus it is reasonable to assume a biological mechanism relative to an initial delay in response has been identified. This would support the linking of the two theories, as well as give credibility to claims that ADHD has been validated via HD analysis.

What then is the specific mechanism that enables a delay to a response? Barkley (1997) states that: "The capacity to delay responses as well as the four consequent mental functions flowing from it are attributed to the brain's prefrontal cortex" (p.69). He cites another work by Bronowski (also a book for the general public) as being relative to this explanation. Bronowski (1976) does mention the prefrontal lobes but only in very general terms and not in any manner related to the theory used by Barkley. The biological component relative to a delayed response in Bronowski's (1977) theory is the enzyme uricase. Bronowski hypothesized that the higher primates have lost this enzyme; as such they cannot remove uric acid giving them a surplus especially in the brain cells. Bronowski believes that as uric acid is purine it has the effect of slowing down responses. So strictly speaking the extension of Bronowski's theory to ADHD would mean that sufferers are unable to inhibit their initial responses due to a lack of uric acid at the brain cell level. If this deficit were then judged to be harmful to the individual, ADHD would be validated via HD Analysis (Wakefield, 1992, 1997, 1999). As Barkley's theory does not make any such

connection it is not possible to validate ADHD as claimed (Barkley et al., 2002. Barkley et al., 2004).

Whether Wakefield's analysis of dysfunction is a reliable means of distinguishing disorder may well be open to debate. What is really concerning is in attempting to identify a specific deficit relative to ADHD it has been found that a highly influential theory has no scientific basis. Even more concerning is how this theory has been used to implicate intervention with stimulant medication. The original version of Barkley's theory (Barkley, 1994) retained the terms used by Bronowski, claiming they had implications for the treatment of ADHD. Barkley states that the normalising effect of stimulant medication on the processes of *separation of affect*, *prolongation*, and *reconstitution*, remains speculative. However, he expects that further research will lead to similar outcomes as those found by Berk and Potts (1991). Their study found private speech (*internalization*) to be normalised by stimulant medication, on this basis Barkley declared that:

ADHD children should start on medication earlier than is now the case, and treatment should be maintained over many years, perhaps allowing for the normal development of proficiency in these four downstream cognitive processes (p.46).

Examination of the Berk and Potts (1991) study found it to be a naturalistic design with a total of 19 ADHD boys with matched controls. Out of this a subsample of 8 of the boys were tracked on and off medication. Aside from the very small sample size being problematic, the authors also conceded that the naturalistic design did not permit counterbalancing the medication; or permit for the variables to be examined under placebo conditions.

Just how effective has stimulant medication been in developing proficiency in the so-called "downstream cognitive processes" (Barkley, 1994, p.46)? Recent

research outcomes would suggest hardly at all. According to researchers from Princeton, Cornell and the University of Toronto (Currie et al., 2013), the research on ADHD drug treatment lacks compelling evidence of long-term efficacy (e.g., Molina et al., 2009). Currie et al. (2013) report on short- and long-term outcomes associated with what the authors describe as a policy experiment that increased insurance coverage for ADHD medications. This experiment led to what the authors describe as a “sharp increase” in the use of Ritalin. In the face of this increased usage, the research addressed both short- and longer-term emotional and academic outcomes. They found an increase in problems over time associated with medication usage: for girls, in emotional problems; boys, reductions in academic attainment.

A longitudinal study into general health and wellbeing carried out in Perth, Australia (Government of Western Australia, 2010) was able to identify 131 adolescents who had received a diagnosis of ADHD. Analysis of this data found at age five none had taken stimulants, by the time the group reached 14 three comparison groups of ‘never medicated’, ‘previously medicated’, and ‘currently medicated’ were identified. Significant findings showed that the long-term use of medication was associated with higher diastolic blood pressure. The effect on school performance was negative, as any child having “ever receiving stimulant medication was found to increase the odds of being identified as performing below age-level by a classroom teacher by a factor of 10.5 times” (p.6).

Finally a large-scale systematic review that compared the benefits and harms of different pharmacological treatments for ADHD found a lack of good-quality evidence on the use of drugs relating to global academic performance or social achievements (McDonagh, Christensen, Peterson and Thakurta, 2009). Taken together, these findings are replications of an extensive review conducted by Barkley

nearly forty years ago when disrupted children were labeled as being hyperactive (Barkley, 1977). Barkley reviewed 110 studies that recorded the effects of stimulant drugs on 4200 children and concluded that while “the drugs seem to facilitate the short-term management of hyperactive children; they have little impact on the long-term social academic, or psychological adjustment of these children” (p.158).

So has anything changed since 1977? Or are we still dealing with the same problems, same treatment, same results, just a different label? And who is benefitting from the current paradigm, those receiving treatment or those dispensing the treatment? Despite Barkley et al.’s (2004) claims to the contrary pharmaceutical company involvement in regard to ADHD is beyond doubt. The American Academy of Child and Adolescent Psychiatry has a web page with practice parameters to aid mental health professionals (www.aacap.org). Although the ones for ADHD are now listed as out of date, a list of declared conflicts of interest is available. Out of 17 experts who contributed to the parameters only 4 declared no conflicts of interest. Barkley’s interests are listed thus:

Russell Barkley, Ph.D.: receives or has received research support, acted as a consultant and/or served on a speaker's bureau for Eli Lilly and Company, Shire Pharmaceuticals Group plc, and McNeil Pediatrics. Dr. Barkley also has or has had books/intellectual property with Guilford Publications.

How then should the International Consensus Statement be viewed? Is it as declared a statement of fact, or is it an attempt to silent anyone prepared to challenge the current paradigm and prescribing practices? Its impact is hard to quantify, it has 250 (Google Scholar) citations but remains on Barkley’s web page as a ‘Fact Sheet’. Barkley’s web site is dedicated to the education of mental health professional and has been endorsed by the following organisations: The American Psychological

Association (APA), the Association of Social Work Boards (AWSB), the National Board for Certified Counselors (NBCC), the California Board of Behavioural Science (CA-BBS), Ohio Counselor, Social Worker, & Marriage and Family Therapist Board (OH-CSWMFT). The influence of Barkley's (1997) theory is more quantifiable as according to Google scholar it now has 5,402 citations. The Handbook for the Treatment of ADHD (Barkley, 2006) is listed as having 6545 citations. The view that ADHD is due to a neurological abnormality is reflected in DSM-5 where it appears under the heading "Neurodevelopment Disorders".

The signatories of the consensus statement called for the media to portray ADHD accurately, specifically in relation to its validity as an undertreated harmful medical disorder. This analysis would suggest there are several reasons to be concerned. The theory underpinning the claims of a specific deficit is without scientific merit, and it has no connection to everyday behaviours or a specific brain location. As such it cannot be validated in the manner claimed. The efficacy of stimulant medications on long-term social, academic and psychological outcomes is as doubtful today as it was 38 years ago. Finally it seems that a disproportionate number of ADHD advisors and experts have extensive connections to various pharmaceutical companies. Taking these points together we now ask just how should ADHD be portrayed? Not just by the media, but by anyone involved in the mental well being of children and adolescents.

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