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ADHD: a scientific fact or a factual opinion? A critique of the veracity of Attention Deficit Hyperactivity Disorder

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This paper is a contribution to the debate on the phenomenon of Attention Deficit Hyperactivity Disorder (ADHD). It explores how and why the discourses surrounding the aetiology and interventions are dominated by a bio-medical understanding of ADHD. Competing discourses are examined, particularly those marginalised because they do not support the prevailing neurological paradigm of ADHD. The reasons for the controversy over psychostimulant medications are explored by examining the reasoning and evidence which contribute to the medicalisation of behaviour. Given the uncertainties, possible contradictions and ambiguities within the bio-medical model of ADHD, educational professionals would do well to look beyond the label to the child's needs rather than assume that such a condition 'exists' and is the provenance of those in the medical profession.

Keywords: Attention Deficit Hyperactivity Disorder; biomedical discourse; truth

Introduction

> Science … is viewed to be the lynchpin of psychiatric practices. It is science that permits the boundary to be drawn between the 'normal' and the 'pathological'; it is science that creates possibilities of accurate identification of mentally ill; it is science that provides effective methods of cure. (Busfield 1986, 17)

Contemporary popular literature based on the aetiology of and intervention for Attention Deficit Hyperactivity Disorder (ADHD) tends to reify ADHD into an uncomplicated, biomedically based phenomenon which is identified and framed within a biological discourse. This is where the aetiology of ADHD is perceived to be a disease caused by biomedical factors, for which psychostimulant medication is an effective and safe intervention. This perspective has apparent exclusive rights to the epistemological understanding of ADHD, as empirical credence verifies that ADHD is a true biomedical 'illness', thus polarising the dominance and 'truth' the biological paradigm holds over the current debate surrounding ADHD. However, if this is the case, then why is the notion of ADHD controversial? Why is there an epistemological uncertainty regarding the biological aetiology of ADHD? Why is psychostimulant medication intervention controversial? Why are other epistemologies that seek to challenge the biomedical 'truth' and existence of ADHD marginalised?

The purpose of this paper is to question the veracity of ADHD as a biological category which reifies the existence of ADHD into an object of knowledge seen through

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the modalities of neurology. The paper will explore how and why the biomedical discourses regarding the aetiology and intervention of ADHD have attained a status of dominance and truth, despite being challenged by other epistemologies. In doing so, this paper will describe the competing discourses that exist; particularly those which are marginalised as they potentially pose a threat to the dominant biomedical paradigm.

Epidemiology of ADHD

ADHD is the contemporary label for one of the most prevalent and intensively studied disorders in child psychiatry, and possibly the most controversial (Schachar and Tannock 1997; Breggin 1999; Baldwin 2000a; Barkley 2005; Graham 2008). The current epistemology of ADHD, as a broad category, is the diagnostic label which represents characteristics such as:

> the inability to marshal and sustain attention, modulate activity level, and moderate impulsive actions. (Rappley 2005, 165)

From existing epidemiological data, it can be seen that America is the 'epicentre of the ADHD diagnosis in children' (Lloyd, Stead, and Cohen 2006, 5). It is estimated that *'five million school-aged children'* (Rafalovich 2005, 307) meet the Diagnostic Statistical Manual IV (DSM IV) criteria for ADHD (Breggin 2000). The ADHD diagnosis rate in the UK is relatively low compared with the USA, but is rising steeply. It is currently estimated that 1 in 100 UK children between the ages of 5 and 16 manifest the most severe symptoms and acute difficulties associated with ADHD, and about 5 in 100 children manifest less severe symptoms (NICE 2008). Nonetheless, it is reported that the diagnosis rate for children in the UK is rapidly spiralling, as there has been a '700% increase rate in the diagnosis of ADHD in children during the last ten years … and that only includes England' (Lloyd, Stead, and Cohen 2006, 3).

Parallel with this dramatic rise in ADHD diagnosis has been the prescribing of psychostimulant medication, such as methylphenidate (Baldwin 2000a, 2000b; Breggin 2003; Timimi 2004; Graham 2008). Both of these factors have fostered major concerns, because the exact 'truth' regarding the aetiology of ADHD is elusive, as is the mechanism for the treatment (Baldwin 2000b; Rafalovich 2005; Tait 2005; Timimi 2008). For this reason, the conceptualisation of ADHD is a contentious area which often arouses virulent debate.

The notion of ADHD

The controversy surrounding the notion of ADHD has its roots in a lack of definitive and unified explanations regarding its aetiology and intervention strategies, thereby questioning the ontological validity of the disorder. This is because ADHD is contextualised within various disciplines, such as the biomedical, psychological and sociological paradigms, each of which provide an array of conflicting discourses that elucidate the aetiology and interventions of ADHD. For example, the biological discourse asserts that ADHD is the result of innate biomedical impairment which is effectively relieved by chemical solutions such as psychostimulant medication. However, the sociological discourse contends that ADHD has been reified into a biomedical concept, whereby the 'qualities of reality [have been] ascribed to that which has no real or independent existence' (Carson 2003, 1134). Thus, from this perspective ADHD does not exist as a true objective disorder. Instead, the sociological discourse argues that the concept of ADHD is a social and

cultural construct whereby 'disorders in society [have created] disorders in children' (Graham 2008, 66). Within the recent past despite these specialised discourses, it is widely asserted that, the notion of ADHD is wholly positioned within the realm of the scientific discourse. This is where the primary causality of ADHD is seen exclusively through a biomedical lens and therefore the primary intervention is also purely biomedically based. As a result, other discourses which question the biological basis of ADHD have been marginalised (Graham 2008). This therefore means that the biomedical discourse is currently dominating the debate around the nature of ADHD to the extent that ADHD is considered to be psychiatry's 'number one biologically based disease' (Baughman 1999, 34).

The medical aetiology of ADHD

The biomedical discourse advocates that ADHD is the result of a 'developmental failure in the brain's circuitry that underlies inhibition and self control' (Barkley 1998, 67). This perspective has gained credence as a result of the empirical justifications within the scientific position. For example, within the biomedical paradigm, the majority of the research has focused around the field of molecular genetics, which has suggested that the underlying cause of ADHD is dysfunctional genes (Tannock 1998; Todd 2000; Faraone and Doyle 2001; Faraone 2005; Stevenson et al. 2005; Hay et al. 2007). Research studies established in the field of molecular genetics are based on neuroimaging tools, namely PET and MRI scans, to enable researchers to compare the brain activities of those children diagnosed as having ADHD with those of other members of the family who are not diagnosed with ADHD (Waldman et al. 1998; Daly et al. 1999). Moreover, familial research based on monozygotic (MZ) and dizygotic (DZ) twins, and adoption studies (Faraone et al. 2005; Hay et al. 2007) have documented a 'strong genetic basis for ADHD which have been strengthened by molecular genetic studies which search for allelic variations of specific genes which are functionally associated with ADHD' (Swanson et al. 2000, 21). For example, Faraone et al. (2005) conducted genome scan studies and identified eight genes which showed a statistically significant correlation with the causality of ADHD. This finding argued that the causality of ADHD can be accredited to genetic factors, specifically 'genes [that are] involved in dopaminergic transmission' (Daley et al. 2008), and 'serotonin transporter genes' (Manor et al. 2001).

Developing from the molecular genetic research, there has been a preponderance of empirical data which buttress the role of dopamine dysfunction in the aetiology and treatment of ADHD. Such pharmacogenomic research focuses on the identification of specific dopamine receptors that are believed to be concerned with the causality of ADHD. This evidence is obtained by exploring the different levels of dopamine transmission in children diagnosed with ADHD compared with children who do not show ADHD symptoms (Hudziak 2001). This form of research is usually based on MRI scans to pinpoint the concentration of activity within specific regions in the brain (Tannock 1998). A study conducted by McCracken et al. (2000) found evidence to substantiate the 'contribution of genetic variation at the DRD4 locus to the etiopathogenesis'. They deduced from this that the dopamine receptor DRD*4* 120-bp polymorphism, which is responsible for encoding one of the receptors that mediates postsynaptic dopamine action, is aberrant, thereby resulting in ADHD symptomology, specifically inattention (McCracken et al. 2000). This research finding was repeated by Todd and O'Malley (2001), who found evidence for a linkage between 'a 120-bp duplication polymorphism in the DRD4 gene and ADHD'. Thus, the pharmacogenomic research into the genetic

association of the dopamine receptor DRD4 polymorphism strengthened the possibility of a correlation between the role of dopamine concentrations and the causation of ADHD.

Biomedical intervention to reduce ADHD

Alongside the evidence that correlates the origins of ADHD to an innate biomedical dysfunction, pharmacogenomic research has also claimed to have established justifications for the use of pharmacological interventions to treat ADHD. The primary pharmacological treatment for prescribed children with ADHD is psychostimulants, which commonly include methylphenidate (*Ritalin* and *Concerta*) and amphetamines (Li et al. 2006). The efficacy of this type of medication for ADHD has been significantly demonstrated in clinical trials based on neuroimaging research which demonstrates how the psychotropic medications affect the brain and alter its function and structure in order to eradicate ADHD symptomatologies. Extensive research has been conducted by Volkow and Swanson (2003), who investigated the effects of psychostimulant medication, in particular methylphenidate and amphetamine. Their research was based on neuroimaging which enabled them to establish that:

> methylphenidate and amphetamines increases extracellular dopamine in the brain … Methylphenidate increases dopamine by blocking dopamine transporter and amphetamines (like methamphetamines) increases dopamine by releasing dopamine from the terminal. (Volkow and Swanson 2003, 1909)

Therefore, the chemicals in the psychostimulant medication can 'replicate the function of the neurotransmitter dopamine by arousing the nervous system' (Miller and Leger 2003, 22). This is where methylphenidate 'induces large volume changes in dopamine in the frontal regions of the brain within one hour of ingestion' (Volkow and Swanson 2003, 1907) in order to restore the dysfunction of dopamine in the brain and diminish the core symptomology of ADHD.

The efficacy of psychostimulant medication, specifically methylphenidate, has also been verified in a multimodal treatment study of ADHD (MTA 1999a). This 14-month randomised clinical trial has been the largest most comprehensive study conducted in order to identify the effectiveness[1] of various intervention treatments in the long term (Swanson 2001; Data Trends 2002). The results obtained from the study overall indicated that the medication condition was viewed to be a 'consistent dominant variable' (MTA Cooperative Group 1999b, 1088) in three of the experimental conditions, which were the Medication Management group, Community Care group, and Combined Treatment group (MTA 2004). This indicated that medical intervention is likely to be an effective and, perhaps, a sufficient treatment for children with ADHD, as it can yield optimal results when the correct medication dosage is administered to each child (Abikoff, Hechtman, and Klien 2004; MTA, 2004). These conclusions were repeated in the 10- and 24-month follow-up study, which intensified the longstanding view that:

> stimulant medication provides symptomatic relief for as long as it is administered … [which means that] this component of treatment has a strong empirical basis of effectiveness. (MTA 2004, 767).

The studies above argue that there is a significant amount of quantitative evidence that supports the notion of ADHD as the result of an innate biomedical impairment which can be verified by 'objectified, reliable, and valid' methodological tools (Fineman 2001, 45). As a result, it is advocated that the biomedical underpinnings of ADHD are ostensibly

based on an 'impartial means of absolute truth' (MacCoun 1998, 259), which objectively justifies the dominance of the biomedical perspective within the ADHD debate.

The 'truths' of ADHD as a biomedical entity

Although the biological discourse has gained dominance within the ADHD debate, an important question that often sparks virulent debate is that if the biological discourse provides such an incontrovertible explanation, why is ADHD a contentious area and why is the existence of ADHD as a medical category questionable?

The apparent empirical truth behind the biomedical discourse that positions the aetiology and intervention of ADHD within the realm of the biological paradigm is accompanied by a considerable amount of scepticism, which has sought to challenge the apparent firm ground of the biomedical conceptualisation of ADHD (Baughman 1999; Szasz 2000; Rafalovich 2005; Breggin 2002; Conrad 2006; Graham 2006; Shah 2008; Timimi 2008). The biomedical perspective questions the argument that ADHD can be exclusively framed within the biomedical discourse, by claiming that the objective empirical evidence that verifies the aberrant neurobiomedical mechanism that is correlated with the underlying causality of ADHD is 'oversimplified, often exaggerated and, thus, inaccurate' (Pellgrini and Horvat 1995, 13). The biomedical reification of ADHD is theoretically challenged on the basis that no 'biological abnormality has ever been specifically or unambiguously linked to the aetiology of ADHD through the mechanism of conventional techniques' (Baumeister and Hawkins 2001, 3), therefore bringing into question the existence of this biomedical 'disorder'. This can be demonstrated clearly in the study conducted by Baumeister and Hawkins, who reported discrepancies among neuroimaging studies, claiming that:

> the complexity of many of these [neuroimaging] studies and the methodological variation among them make it difficult to discern whether these inconsistencies are apparent or real. (Baumeister and Hawkins 2001, 2)

One of the most prominent ambiguities that Baumeister and Hawkins (2001) identified was that different biomedical discourses exemplified different causalities regarding the aetiology of ADHD. Although converging evidence has implicated abnormalities of dopamine neurotransmission to the pathology of ADHD (McCracken 1998; Volkow and Swanson 2003; Faraone 2005), this evidence is challengeable as there are no specific unified genetic variants that have been unequivocally demonstrated as contributing to the aetiology of ADHD. Instead, there are various empirical neuroimaging studies that pinpoint different genetic variables that are statistically correlated with the aetiology of ADHD. This can be illustrated in the research conducted by Faraone et al. (2005), McCracken et al. (2000) and Volkow and Swanson (2003), who, between them, specified various genetic markers associated with ADHD. For example, Faraone et al. (2005) identified eight genes in which the same variant was studied in three or more studies, seven of which showed statistically significant evidence of association with ADHD; whereas McCracken et al.'s (2000) study pointed only to the significance of the DRD4 polymorphism receptor in relation to the aetiology of ADHD. These inconsistent findings demonstrate that the scientific research conducted in relation to the aetiology of ADHD is questionable in terms of epistemological validity, as there is:

> a lack of consensus on which brain region or networks that are critically abnormal to pinpoint a unified biological marker to determine the aetiology of ADHD. (Bullmore and Fletcher 2003, 381)

This empirical contradiction has led to epistemological uncertainty regarding the aetiology of ADHD, which inevitably brings to question which discourse from the apparently objective biomedical paradigm is empirically exact and attains a degree of truth, if any at all.

Furthermore, it can be argued that the evidence obtained from the neuroimaging studies based on findings from research tools such as MRI, PET and other functional imaging modalities (see, for example, McCracken et al. 2000; Volkow and Swanson 2003; Farone et al. 2005) can be questioned in terms of its ontological validity and empirical reliability. This is because, according to Orden and Paap (1997), these tools simply measure transient changes in blood flow and deduce brain activity from that flow, thereby reducing brain activity to a mechanistic process which reduces behaviour to mere uncontrolled reactions to stimuli. The findings from such research tools, therefore, illustrate only how an individual's blood flow reacts to certain activity in a controlled environment (Breggin 2002). Consequently, it could be postulated that such 'objective' research cannot empirically identify the exact causality of ADHD and as a result of this it can be argued that the biomedical reification of ADHD is beset by uncertainty (Rafalovich 2001; Oak 2004). However, despite this, it would seem that the biological discourse appears to be immune from the charge of empirical misinterpretation on the basis that such discourse arises from 'prestigious' biomedical research tools which make it difficult to refute.

The 'truths' of treating children with pharmacological intervention

Not only has the empirical justification regarding the aetiology of ADHD been questioned, but the apparent efficacy of the spiralling use of psychostimulant medication in children has also been extensively embroiled in contention. This controversy derives from the basis that psychostimulant medications are very frequently prescribed as a first line of treatment (Travell and Visser 2006) despite the lack of an exact epistemological foundation to explicate the aetiological root of ADHD. At the heart of this controversy is the grave concern about the medicalisation of undesirable childhood behaviour and pathologising the failure of schools to control behaviours (Slee 1996). This is because there is converging evidence to illustrate that the use of pharmacological treatment has a 'paradoxical effect upon children' (Graham 2006, 7), resulting in potentially adverse effects both physically and psychologically (Baldwin 2000b; Breggin 1999; Cohen and Leo 2002; Baughman 1999; Cohen 2004; Fone and Nutt 2005; Graham 2006; Panorama 2007; Timimi 2008). It is widely acknowledged that stimulant medication such as methylphenidate (*Ritalin* and *Concerta*) shares many 'toxic properties with pure amphetamines, including rapid uptake, high addictive qualities and occasionally a proclivity to reaction' (Baldwin 2000a). This means that methylphenidate (specifically *Ritalin*) can be beneficial only within a short-term period, usually between four to five hours when administered orally (Breggin 2002; Volkow and Swanson 2003) and for about four to five weeks, before the stimulant starts to lessen its effect unless dosage is increased (Swanson 1993; Breggin 1999). Having said this, it has been argued that *Concerta* does have a more prolonged efficacy than *Ritalin*. However, Shah (2008) reports that within four weeks of consistent administration of *Concerta*, the efficacy of the medication decreases. Therefore, it could be argued that the short-term benefits of psychostimulant drug use are often exclusively limited to behavioural control such as reducing 'classroom disturbances' and ameliorating 'compliance and sustained attention' (MTA 1999a, 1077).

Furthermore, the long-term efficacy and safety of such psychostimulant medications have not yet been sufficiently researched (Cozza, Crowford, and Dulcan 2003). This is because it is advocated that the potential adverse side effects of stimulant medication may

be dangerous, permanent and irreversible as a result of the toxic properties of the medication (Baldwin and Anderson 2000; Breggin 2000; Ghodse 2007). For this reason, it could be postulated that the short-term benefits of the psychostimulant medication may outweigh the potentially long-term adverse side effects (Travell and Visser 2006).

A further issue that stems from this controversy is the question of who actually benefits from the short-term efficacy of the stimulant medication. Diller (2006), in discussing the controversy surrounding ADHD, points to the pressure that psychiatrists and general practitioners come under from parents who want a medical diagnosis for the behaviour difficulties they perceive in their child. Concomitant with this is the medical solution of medication. It is claimed that the short-term behavioural improvements in children who are prescribed methylphenidate are more likely to be advantageous to parents and teachers rather than to the children themselves (Cantwell 1999; Miller and Leger 2003). An example of this can be clearly illustrated by the findings obtained in the MTA study (1999a), which concluded that psychostimulant medication, particularly methylphenidate, is a safe and effective treatment for children with ADHD. However, the data collected in this study can be critiqued on the basis that the findings relied entirely on information provided by parents and teachers. The children within the study were not asked for their feelings or thoughts regarding the intervention to which they were assigned (Breggin 2000). As a result, some of the adverse drug effects of methylphenidate, such as 'depression, worrying, irritability and loss of spontaneity' were not surfaced, as such information could only be unmasked by asking the children about their feelings whilst taking the psychostimulant medication.

Thus, in essence, the researchers and indeed psychiatrists and physicians failed to collect data on the most important factor, which is how the stimulant medication impacts the child who is administered the potentially dangerous psychostimulant drug (Breggin 2002; Diller 2006; Timimi and Taylor 2004; Graham 2006; Williams and Taylor 2006). For this reason, opponents of the biological discourse criticise the use of psychostimulant medication for children when treating ADHD. They argue that the use of the ADHD label and the administration of methylphenidate are utilised as a means of social control, whereby unruly behaviours that are socially undesirable are recognised by an invalid diagnosis and thus 'cured' by dubious clinical interventions (Baldwin 2000a; Jacobson 2006). Such intervention is said to be an 'iatrogenic drug epidemic' (Breggin 1999a, 303), whereby dangerous medical intervention generates a 'mindless obedience that suppresses emotions and ideas, diminishes self esteem, and takes away the sense of self' (Miller and Leger 2003, 46). As a result, when the biomedical intervention is implemented, the ADHD symptomology is seen wholly through the biological lens, thus robbing the practitioner of the opportunity to appraise the impact of social factors.

The biomedical reification of ADHD and the notion of truth

It has been argued so far in this paper that the biological discourses regarding ADHD might be based on inaccurate interpretations of data. Tait sees ambiguities and contradictions in:

> almost every aspect of the disorder, its prevalence, its symptoms, its aetiology, its consequences, its treatment, its longevity, and its constituency. (Tait 2006, 94)

If this is the case, then why has the biological paradigm gained such dominance within the ADHD debate, perceived to embody a status of established truth?

Whilst, as stated earlier, there is an argument which seeks to justify ADHD as an objective biomedical disorder, it is necessary to understand how ADHD has come to be

classified as an objective medical category in the absence of empirical identification of the aetiological root(s) of ADHD (Szasz, 2000).

First and foremost, it could be that the biomedical paradigm has achieved the degree of credence it has primarily because research into ADHD has been centred largely on an integration of subjective clinical knowledge with scientific investigation, thereby upgrading what might be regarded as a largely subjective observation into an objective empirical disorder and one that is perceived to embody a notion of truth. This scientific investigation has been amplified by the use of methodological mechanisms such as MRI and PET scans, which have played a pivotal role in transforming observations into a biomedical fact, and one based on an absolute status of 'objectified truth'. This is because these methodological tools have generated progressive empirical verifications to 'identify' a biological root cause of ADHD. As a result, the apparent unambiguous specialised biomedical discourses based on neurotransmitters and genetic processes are perceived to inspire confidence through an image of meticulousness and objectivity, thereby strengthening their clarity and utility and, consequently, making such a discourse difficult to refute. For this reason, the aetiology and treatment of ADHD is often perceived exclusively as an aberration of neurological function, uncovered by contemporary scientific methodological research tools, which means that the notion of ADHD as a biomedical entity is processed into an absolute objective truth, free from subjective elusiveness (Oak 2004; Rose and Rose 2000; Farah 2002; Biederman et al. 2004; Barkley 2005; Tait 2005). This discourse transpires into 'specialised professional discourses' (Lloyd and Norris 2000, 58), as it is conveyed to the public by the powerful people who are perceived to have the specialist knowledge within this discipline, such as doctors, psychiatrists and pharmaceutical companies, thereby quashing the stance that questions the ontological existence of ADHD.

Parallel to the empirical evidence derived from the methodological research tools, the biomedical 'truth' of ADHD is further strengthened by the apparent efficacy of methylphenidate in treating the ADHD symtomatology (Tait 2001). This is because proponents of the biomedical model argue that ADHD is reducible to brain structure abnormalities (Barkley 1997a; Fuster 1997; Volkow and Swanson 2003). This perspective maintains that psychostimulant medication, such as methylphenidate regulates the dopamine transmission in the brain and so normalises behaviour in children with ADHD. Therefore, because methylphenidate effectively works as a treatment by dispensing with unwanted behaviour as seen by adults, it can be argued that the biomedical aetiology of ADHD works best as an explanation (Tait 2006; Rafalovich 2007; Graham 2008), thereby intensifying the biomedical causality and treatment of ADHD.

In this paradigm ADHD is constructed through the use of an amalgam of various empirical, statistical, pharmacological, observational, behavioural and educational data which have all been accumulated to the point where their combined presence corresponds to the existence of an apparent objectified disorder (Tait 2006). Consequently, proponents of the biomedical paradigm advocate that:

> biologism now completely dominates the discourse on the causes and treatment of mental illness, especially ADHD. (Graham 2008, 34)

Why is ADHD often seen exclusively through the biomedical lens?

Although the biomedical conceptualisation of ADHD has gained dominance, there might be 'no epistemological foundation that may legitimise such biological conceptualisation'

(Foucault 1972, 205). As a result, it could be argued that the biomedical paradigm of ADHD fails to reach its own ontological and epistemological standards as an objective pathology (Tait 2006). However, this point accentuates a pivotal question: that is, if there is no definitive, unitary biological framework within which the biomedical causation and treatment can be reliably positioned, then why is the conceptualisation of ADHD robustly embedded within the biomedical paradigm? In order to address this question, it is prudent to discuss the association of scientific concepts with the notion of truth.

The concept 'truth' is a tool in which specialised discourse(s) are used to express agreement, to emphasise claims or to form certain types of generalisations which advocate a normalising discourse which either affirms or negates particular ways of being (Foucault 1972). For example, the biomedical discourse of ADHD finds a way of conveying ADHD through a specific repertoire of knowledge, which then limits its domain. This is done firstly by defining the concept to mark its existence, then ascribing the concept's describable qualities by reifying the concept into a biomedical entity and, finally, contextualising the concept to the public for the purpose of identification and clinical implementation (Graham 2006). However, in order for the biomedical knowledge of ADHD to gain ascendancy over other competing discourses and to establish the value of absolute truth, such biomedical knowledge is channelled to the public by those people who are in a position of power, such as doctors and psychiatrists, who are perceived to create, sustain and convey the knowledge which crystallises what is deemed to be 'right' and 'wrong', what is deemed to be 'normal' and what is 'deviant' (Graham 2005, 5). This tacit knowledge is then established as a particular 'regime of truth' (Foucault 1977):

> system of ordered procedures for the production, regulation, distribution, circulation and operation of statement. (Foucault 1980, 23)

Thus, it could be said that the biomedical discourse is constructed by relations of power, invested with ideologies (Fairclough 1992, 28), which means that the biomedical discourses create 'effects of truths which are neither true nor false' (Foucault 1980, 116) . However, because such truth about ADHD is produced as a result of 'legitimated knowledge' (Tait 2001, 22) which is globally recognised for its mantra of reliability, objectivity and truth, the biological discourse inevitably gains the status of trust. As a result, the biomedical discourse is processed as a 'normalising discourse' which becomes the standard against which childhood is judged. This can be illustrated in the realm of the ADHD debate as other discourses are marginalised to make way for the 'infallible', biomedical paradigm which bestows the notion of absolute truth regarding ADHD, and disregarding the fact that:

> Science is a social process, [which means that] the truths it produces are forged within specific social contexts. (Tait 2001, 27)

Furthermore, according to Neitzsche (1967, 40), 'there are no eternal facts, as there are no absolute truths…only interpretations'. This means that, although the biomedical paradigm is now the dominant perspective in explicating the aetiology and treatment of ADHD, possibilities of continuous shift in clinical 'regimes of truths' may result in the vulnerability of the biomedical explanation and give way to the dominance of another paradigm. The scientific battles and competition that arise from the competing discourses in order to define knowledge and truth are the result of a conflict in power, and not the result of an intellectual debate (Rafalovich 2007). Thus, the different forms of knowledge existing for

the notion of ADHD are merely 'institutive interpretations' (Papineau 1994, 47), and therefore can not represent a status of objective truth.

The bio-psychosocial perspective

The lack of true certainty in the biomedical aetiology and treatment of ADHD makes it difficult to contextualise the concept of ADHD within a single framework. As a result, Cooper (1997) is persuasive when he advocates a bio-psychosocial approach. This is where he asserts that the concept of ADHD should be seen through a more holistic, multimodal lens whereby ADHD comes to be understood through a bio-psychosocial framework which incorporates multiple perspectives on the aetiology and intervention of ADHD (Cooper 2002; Timimi 2004; Graham 2008). Such a perspective argues that the aetiology of ADHD originates from the theoretical underpinning of biological, psychological and sociological factors and, therefore, can only be treated effectively via a 'multi disciplinary approach'. Thus, the intervention would be based on the integration of pharmacological treatment alongside non-pharmacological treatment (Cooper 2000) in order to provide a more unified and balanced intervention strategy for children diagnosed with ADHD. However, as this paper has argued, this perspective is often marginalised in order to make way for the dominance of the reductive biomedical discourse which retains a strong link to the biological discourse and medicalises behaviour.

Conclusion

The notion of ADHD is currently positioned predominantly in the biomedical paradigm, with the aetiology and intervention too commonly perceived through a reductive biomedical lens that claims to produce objectified truth regarding that aetiology and intervention. This discourse cannot provide conclusive justification to correlate the causality of ADHD with a unified genetic or biological component. The biomedical discourse continues to contain uncertainties, possible contradictions and ambiguities. Therefore, ADHD should still remain an area of contention, with the efficacy and appropriateness of some medical interventions remaining open to challenge. Continuing uncertainties about the biological justification also bring into question the veracity of the conceptualisation of ADHD as a biologically based 'disorder'. With no end to the debate in sight, the biomedical 'truth' of ADHD remains a hypothetical disorder with uncertain foundations. Professionals working with those labelled as 'ADHD' would do well to step back and examine the behaviour in the totality of the context in which it occurs.

Note

1. In the MTA study, the term 'effective' denoted how the intervention treatments resulted in behaviour that reduced inattentiveness, impulsivity, and hyperactivity (MTA,1999a).

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