Introduction

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Something strange has been happening to children in many Western societies in the past couple of decades. The diagnosis of attention deficit hyperactivity disorder (ADHD) has reached epidemic proportions, particularly amongst boys in North America. The diagnosis is usually made by a child psychiatrist or paediatrician with advocates of the diagnosis claiming that children who present with what the diagnoser considers to be overactivity, poor concentration and impulsivity are suffering from a medical condition which needs treatment with medication, mainly in the form of stimulants such as Ritalin™, whose chemical properties are similar to the street drugs speed and cocaine. ADHD has become firmly established in many local cultures, particularly in North America, Australasia, and Northern Europe, with economically and politically powerful groups (such as drug companies, doctors, psychologists and teachers) having had a major, but often unacknowledged impact on local communities' conceptions about the nature of childhood. A new category of childhood has emerged – that of the ADHD child.

A brief history of ADHD

Overactivity, poor concentration and impulsivity in children were first conceptualized as medical phenomena in the early years of the last century. The first recorded medical interest in children with poor attention and hyperactivity dates back to when a paediatrician, Frederick Still, described a group of children who showed what he felt was an abnormal incapacity for sustained attention, restlessness and fidgetiness, and went on to argue that these children had deficiencies in volitional inhibition, but he offered no treatment other than good discipline (Still, 1902).

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Hyperactivity and poor attention in children then came to be viewed as linked when the diagnosis of minimal brain damage (MBD) was coined. The idea of MBD had originally gained favour following epidemics of encephalitis in the first decades of the twentieth century. Post-encephalitic children often presented with restlessness, personality changes and learning difficulties. Then, in the 1930s, came a chance discovery that psycho-stimulant medication could reduce the restlessness, hyperactivity and behavioural problems that some of these children presented with (Bradley, 1937). Bradley believed that this calming effect he observed was likely to apply to anyone who took low-dose stimulants, not just the hyperactive kids he was treating.

During the next few decades there were few doctors who showed much interest in this or saw such childhood behaviours as areas of legitimate concern for medicine. A few speculated that children who presented as hyperactive might have organic lesions in the brain that were the cause of their hyperactivity. Strauss's writing in the 1940s (e.g. Strauss and Lehtinen, 1947) is one such example. His suggestion was that hyperactivity, in the absence of a family history of sub-normality, should be considered as sufficient evidence for a diagnosis of brain damage, believing that the damage was too minimal to be easily found.

By the 1960s, however, the term MBD was losing favour as evidence for underlying organic lesions in children who displayed poor attention and overactivity was not being found. Instead, with the growing interest in behaviourally defined syndromes, the goal posts were about to be moved and a behaviourally defined syndrome articulated. Despite the abandonment of the minimal brain damage hypothesis, the assumption that this syndrome does indeed have a specific and discoverable physical cause, related to some sort of brain dysfunction, survived in the new definition. Yet, studies have shown that demonstrable minimal brain damage due to a variety of causes predisposes a child to the development of a wide range of psychiatric diagnoses as opposed to a particular type, such as ADHD (e.g. Schmidt *et al.*, 1987). Rutter (1982) concluded that the available evidence shows that overactivity is not a sign of brain damage and that brain damage does not usually lead to overactivity. We are not aware of any subsequent data that contradicts this conclusion.

In the mid 1960s the North American-based Diagnostic Statistical Manual (DSM), second edition (DSM-II) coined the label 'Hyperkinetic reaction of childhood', to replace the diagnosis of MBD

(American Psychiatric Association, 1966). Over the following three decades this new behaviourally defined condition rose from a matter of peripheral interest in child psychiatric practice and research in North America to a place of central prominence.

DSM-II was replaced in the early 1980s by the third edition (DSM-III, American Psychiatric Association, 1980). The disorder was now termed Attention Deficit Disorder (ADD). This could be diagnosed with or without hyperactivity and was defined using three dimensions (three separate lists of symptoms), one for attention deficits, one for impulsivity and one for hyperactivity. The three-dimensional approach was abandoned in the late 1980s when DSM-III was revised (and became DSM-III-R, American Psychiatric Association, 1987), in favour of combining all the symptoms into one list (one dimension). The new term for the disorder was Attention Deficit Hyperactivity Disorder (ADHD), with attention, hyperactivity and impulsiveness now assumed to be part of one disorder with no distinctions. When the fourth edition of DSM (DSM-IV, American Psychiatric Association, 1994), reconsidered the diagnosis the criteria were again changed, this time in favour of a two-dimensional model with attention deficit being one sub-category and hyperactivity-impulsivity the other. According to DSM-IV, the diagnosis 'ADHD not otherwise specified' should be made if there are prominent symptoms of inattention or hyperactivity-impulsivity that do not meet the full ADHD criteria. If we were to interpret this concretely (as doctors often do) it suggests that, as of DSM-IV, nearly all children (particularly boys) at some time in their lives could meet one of the definitions and warrant a diagnosis of ADHD.

The modern champion of the ADHD diagnosis and one of the strongest advocates for a brain dysfunction model and the use of drugs to 'treat' these children is Professor Russell Barkley. Barkley's (1981) book *Hyperactive Children: A Handbook for Diagnosis and Treatment* received widespread attention from both the public and professional communities. From there Barkley's campaign quickly caught the interest of the pharmaceutical industry and soon an avalanche of research to find more support for the disease theory and drug treatment ensued. This new partnership between the commercial interests of the pharmaceutical industry, the personal interests of individual researchers, and the professional interests of medical sub-specialities such as child psychiatry and paediatrics, has subsequently shaped the academic debate and clinical practice, leading it, in our opinion, away

from scientific accuracy and towards an ideological position that has led the mass use of stimulants on children (primarily boys) to control their behaviour and improve their school grades.

ADHD today

The formal technical definition for ADHD can be found in the *The Diagnostic Statistical Manual (DSM)* now in its 4th edition (American Psychiatric Association, 1994). This defines ADHD as:

(A) Either (1) or (2):
(1) Six (or more) of the following symptoms of ***inattention*** have persisted for at least six months to a degree that is maladaptive and inconsistent with developmental level:

Inattention
- often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities
- often has difficulty sustaining attention in tasks or play activities
- often does not seem to listen when spoken to directly
- often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behaviour or failure to understand instructions)
- often has difficulty organizing tasks and activities
- often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)
- often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools)
- is often easily distracted by extraneous stimuli
- is often forgetful in daily activities.

(2) Six (or more) of the following symptoms of ***hyperactivity-impulsivity*** have persisted for at least six months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity
- often fidgets with hands or feet or squirms in seat
- often leaves seat in classroom or in other situations in which remaining seated is expected
- often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)

- often has difficulty playing or engaging in leisure activities quietly
- is often 'on the go' or often acts as if 'driven by a motor'
- often talks excessively.

Impulsivity

- often blurts out answers before questions have been completed
- often has difficulty awaiting turn
- often interrupts or intrudes on others (e.g., butts into conversations or games).

(B) Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age 7 years.
(C) Some impairment from the symptoms is present in two or more settings (e.g., at school [or work] and at home).
(D) There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.
(E) The symptoms do not occur exclusively during the course of a pervasive developmental disorder, schizophrenia, or other psychotic disorder and are not better accounted for by another mental disorder (e.g., mood disorder, anxiety disorder, dissociative disorder, or personality disorder).

Those with a critical eye would have spotted that words such as 'often', 'seems', 'difficulties', 'reluctant', 'easily', 'quietly', and 'excessively' that are used to 'define' ADHD symptoms are hard to define. For example the word 'often' appears in every one of the above 'symptoms', but what does it mean? Does it mean that the child does those behaviours at least once a day or at least once a minute?

These lists of behaviours that are used to define ADHD appear in questionnaires that are then usually given to parents and teachers. These questionnaires are the closest we get to having a 'test' for ADHD. These questionnaires can only rate a particular adult's perception of a particular child at a particular moment in time and in a particular setting. In other words they are measures of the *subjective* perception of the adult filling in the rating scale. What they cannot be is an *objective* factual piece of 'hard data' that measures something intrinsic to the child.

Hyperactivity, impulsivity and poor concentration are behaviours that occur on a continuum. All children, particularly boys, will present with such behaviour in some settings at some point. They are not

behaviours that would be interpreted as abnormal whenever they occur. Without any medical tests to establish which individual has a physical problem causing these behaviour problems, defining the cut-off between normal and ADHD is arrived at by an arbitrary decision. Those who have argued that ADHD does not exist as a real disorder often start by pointing this out. Because of this uncertainty about definition it is hardly surprising that epidemiological studies have produced very different prevalence rates for ADHD ranging from about 0.5 per cent of school-age children to 26 per cent of school-age children (see Timimi, 2006).

Although this obviouse problem with definition confronts you before you even start examining the scientific literature, it has not stopped, perhaps even encouraged (due to the criteria being so open to interpretive variation), increasing popularity of the diagnosis with a concurrent increase in the use of stimulants in the young. National consumption of Ritalin™ in the United States more than doubled between 1981 and 1992. Prescriptions of Ritalin™ have continued to increase in the 1990s, with over 11 million prescriptions of Ritalin™ written in 1996 in the United States. The amount of psychiatric medication prescribed to children in the United States increased nearly three-fold between 1987 and 1996, with over 6 per cent of boys between the ages of 6 and 14 taking stimulants by 1996. One study in Virginia in 1999 found that in two school districts, 17 per cent of white boys at primary school were diagnosed with ADHD and taking stimulants. There has also been a large increase in prescriptions of stimulants to preschoolers (see Timimi, 2005). In the UK prescriptions for stimulants have increased from about 6,000 in 1994 to over 450,000 by 2004, a staggering 7,000+ per cent rise in one decade (Department of Health, 2005).

Possible reasons for this dramatic change in medical practice in such a short space of time are explored further in this book. However, an important contribution to this trend comes from the way ADHD is currently portrayed in mainstream academic and other public institutions. A good example comes from the ADHD advocacy group – Children and Adults with Attention Deficit Hyperactivity Disorder (CHADD) – a large American-based 'parent support group'. CHADD engages in lobbying and claims to provide science-based, evidence-based information about ADHD to parents and the public. Critics point out that CHADD's basic function appears to have become that of promoting stimulant medications manufactured by its corporate donors. For example, pharmaceutical companies donated a total of $674,000 in

the fiscal year 2002–2003 (Hearn, 2004). CHADD's website has this to say about ADHD:

> Attention-deficit/hyperactivity disorder (AD/HD) is a condition affecting children and adults that is characterized by problems with attention, impulsivity, and overactivity. It affects between 3 and 7 percent of schoolage children, and between 2 and 4 percent of adults . . . The body of scientific literature documenting the reality of this condition is immense. (see <http://www.help4adhd.org/en/about/what>)

And:

> Although precise causes have not yet been identified, there is little question that heredity makes the largest contribution to the expression of the disorder in the population.
> In instances where heredity does not seem to be a factor, difficulties during pregnancy, prenatal exposure to alcohol and tobacco, premature delivery, significantly low birth weight, excessively high body lead levels, and postnatal injury to the prefrontal regions of the brain have all been found to contribute to the risk for AD/HD to varying degrees.
> Research does not support the popularly held views that AD/HD arises from excessive sugar intake, food additives, excessive viewing of television, poor child management by parents, or social and environmental factors such as poverty or family chaos (see http://www.help4adhd.org/en/about/causes).

Those views are concordant with a vocal section of the medical community who appear to have appointed themselves as representative of 'mainstream' thinking. A good example of their beliefs and the style with which they prounounce their essentially ideological stance can be found in the article 'International consensus statement on ADHD' (Barkley *et al*., 2002):

> Numerous studies of twins demonstrate that family environment makes no significant separate contribution to these traits. This evidence, coupled with countless studies on the harm posed by the disorder and hundreds of studies on the effectiveness of medication, buttresses the need in many, though by no means all, cases for

> management of the disorder with multiple therapies . . . To publish stories that ADHD is a fictitious disorder or merely a conflict between today's Huckleberry Finns and their caregivers is tantamount to declaring the earth flat, the laws of gravity debatable, and the periodic table in chemistry a fraud. ADHD should be depicted in the media as realistically and accurately as it is depicted in science – as a valid disorder having varied and substantial adverse impact on those who may suffer from it through no fault of their own or their parents and teachers. (Barkley *et al*., 2002; 89–90)

More recently evidence is emerging that the diagnosis of ADHD has opened the door to stimulants being used for an age-old indication – that of perceived performance enhancement. For instance, 'Results of a survey of physicians suggest that parents often request a "behavioral drug," such as Ritalin™, with the goal of enhancing their child's academic performance rather than treating an illness' (Gale, 2006). Despite this headline's apparent surprise at this practice, the prescribing of stimulants to improve academic performance is fully sanctioned by a leading ADHD researcher. According to Joseph Biederman, 'If a child is brilliant but is doing OK in school, that child may need treatment, which would result in performing brilliantly in school' (Gale, 2006).

A case study in the journal *Pediatrics* provides an interesting example of the forces at work in the diagnosis of an individual child with ADHD in the current academic and clinical climate. In 1999, the editors elicited commentaries from several prominent physicians about the case of a teenage boy who had been taking Ritalin™ for several years. The editors saw the boy's scenario as an interesting case, worthy of commentary from a group of prominent child psychiatrists. Ironically they unintentionally provided a much more interesting case study. From a sociological point of view the subject of the case was not the boy, but, instead, was the doctors and the editors. The case provides an excellent example of: (1) how a major determination in the diagnosis of ADHD is adult satisfaction, (2) how the medical community fully supports the use of stimulant medication as a performance-enhancing drug, (3) how the same mindset that approves of using one psychotropic drug easily leads to the use of multiple medications, and (4) how the mainstream medical journals have given little attention to the ethical implications of controlling and altering children to meet the demands of our contemporary educational/cultural system:

> The 15-year-old boy announced to his parents and his paediatrician that he wanted to stop taking his medication: 'I don't need it . . . I'm fine . . . I don't see why I should take it.' He purposefully did not take the medication for a few weeks and he said he could not tell the difference . . . However, his parents observed that his test results, when off the medication, were below his standard scores . . . They also noted that he was more distractible and less attentive when doing his homework during that time. (Cohen *et al.*, 2002)

As stated by the physicians, the most important variable in determining whether this boy should keep taking his medication was the parental satisfaction with the medication, and the subsequent commentaries all focused on how to persuade the boy to continue taking his medication. The boy's wishes were not something to be listened to, but rather something to be managed. One of the commentators even suggested that the boy's reluctance to keep taking his Ritalin™ suggested this was a sign that he needed another medication. Thus the boy, who wants come off his one medication, would instead get two. None of the commentators in the *Pediatrics* article contemplated that the boy's wishes might be legitimate, but more importantly, as a sign of how one-sided the issue has become, the editors did not give space to a single commentator who questioned the ethics of giving a medication to improve grades.

What most of the current ADHD 'experts' are reluctant to acknowledge to the general public is that, no matter what area of their research one chooses, whether it is genetics, neuroimaging, or chemical imbalances, the more studies they publish, the further away the goal of finding a biological marker to help with diagnosing children with ADHD seems to become. To account for an increasing list of disparate results, their answer has been to develop ever more complicated theories about the biological basis of ADHD, but these theories can obscure only for so long a simpler possibility – that there may be no biological marker for ADHD.

This book therefore is an attempt at providing an antidote to the one-sided mainstream literature referred to above. In the past couple of decades an increasing number of authors have written about ADHD from a more critical perspective. These critiques have ranged from questioning the existence of the disorder and the way it is currently conceptualized in mainstream medicine to the safety and efficacy

of popular drug treatment regimes for ADHD. Each of these critical authors has focused on their own particular area of interest be this culture, genetics, the influence of drug company marketing, the effects of medication, particular treatment regimes, and so on. This book brings something new and of great importance for the critical literature on ADHD. In this book we bring together a variety of critical perspectives, with each contribution dealing with a particular issue from culture to genetics and from drug companies to nutrition. The contributing authors are well known internationally and include senior and experienced clinicians, academics, and best-selling authors. Although many of the chapter themes overlap, we have divided the book into four sections to highlight the differing focus of different contributions.

Part One: ADHD and the Medical Model

In Chapter 1, Lydia Furman summarizes all the problems with efforts to pinpoint a biological deficit in children diagnosed with ADHD. The research, whether it is anatomical imaging, functional imaging, genetics, or neuropsychological research, reveals that there is no clear evidence for a discrete disease. As Furman documents, the current discourse coming from professional organizations overstates the evidence base for ADHD. In spite of this lack of evidence, the convergence of societal and financial pressures has given rise to the ADHD industry.

In Chapter 2, Jay Joseph challenges long-held beliefs about the role of genetics in a DSM-IV condition. For anyone who has been sceptical about the supposed genetic basis of ADHD or the oft-heard promises that discoveries of ADHD genes are right around the corner, Joseph's chapter is a must read. According to the genetic researchers, the hunt for ADHD genes is justified because of the twin and adoption studies but, as Joseph shows, these studies are fundamentally flawed and a careful examination of the twin and adoption studies shows why the search for genes has been unsuccessful. In 2000, Joseph made the bald statement that a gene or genes for ADHD will not be discovered because they do not exist. Seven years later he has yet to be proven wrong.

In Chapter 3, Jonathan Leo and David Cohen critically appraise neuroimaging studies in ADHD. Carefully analysing the published results from ADHD neuroimaging that are frequently used to support the notion cerebral pathology underlies the ADHD, they note

that the variable of prior medication exposure must be carefully considered in studies used to support this claim. Their analysis reveals, however, that investigators have been prone to treat the variable of prior psychotropic drug use with less objectivity than its importance requires and have thus failed to show a consistent and distinct difference between children diagnosed with ADHD and controls. As a result an ADHD neuroimaging 'paradox' is emerging: as brain imaging technology becomes more sophisticated, as more imaging studies are published and more regions of the brain added to an ever-expanding list of potential problem areas with little reproducibility between studies, and as theories of ADHD become more and more speculative, then the likelihood of using imaging as a practical diagnostic tool becomes smaller and smaller. While some would say that this is a sign that researchers are becoming more sophisticated, the other possibility is that they simply do not want to acknowledge the obvious – that there is no biological marker in the brain for ADHD.

Part Two: ADHD and Culture

In Chapter 4, Sami Timimi sets out to explore the question: 'Why has there been a dramatic increase in diagnosis of ADHD and prescription of stimulant medication for this in the past few decades in most Western countries?' Shedding light on this question is crucial given the absence of good evidence to support the contention that ADHD is a physical condition. Timimi first addresses the question of what environmental factors may have resulted in a real increase in ADHD-type behaviours in children and then discusses the contribution of our changing understanding of childhood, child rearing and education and thus the changes in the way we think about, classify, and deal with children's behaviour. Finally he discusses the related question of why these dynamics of medicalization are occurring in the way that they are, at this point in time.

In Chapter 5, Craig Newnes critiques the role his profession has played in supporting the medical model discourse of ADHD. He notes that the profession of clinical psychology has become adept at jumping on bandwagons of a variety of practices as they become fashionable and develop potential for paying salaries. From psychometric assessment and psychotherapy to cognitive therapy and consultancy, the profession has embraced different practices while claiming a scientific basis for its new positions of power. He cautions that the enthusiasm

with which clinical psychology has seen a potential role in the ADHD explosion gives cause for concern that, as a scientific discipline, clinical psychology has 'gone off the rails', but notes that 'islands' of constructive criticism and alternative conceptualizations still have solid support.

In Chapter 6 Brian Kean provides an overview of the history of ADHD in the United States, and an in-depth examination of its emergence in Australia, which was the first country outside of North America to make significant use of the diagnosis and subsequent drug treatment. As Kean points out, in the early 1990s the use of stimulant treatment in Australia was practically unheard of, but by 2003 there were reports of 7.5 per cent of 6–17-year-olds in Australia being diagnosed with ADHD. Kean looks at the forces and organizations responsible for this rise in ADHD diagnosis and medication.

In Chapter 7 Sami Timimi and Begum Maitra demonstrate the global to and fro movement of ideas about childhood behaviours, whether or not these are considered problematic, by whom, how responsibility is attributed, and the relationships between public and professional systems of attribution. They suggest that 'culture' is central to these systems of exchange. These global movements of people and ideas offer an opportunity to reconsider the basic premises of our professional beliefs about children and the construct of ADHD. To do this they examine some of the macro-dynamics of globalization, its relevance to psychiatry more generally, and then ADHD specifically. They argue that such an approach to the problematic concept of ADHD can help produce a greater diversity in our understanding and hopefully result in a more sophisticated approach to the way we deal with what is essentially a loose collection of qualitatively normal behaviours found in most children at some time in their lives.

In Chapter 8, Nick Hart and Louba Benassaya examine the different discourses of ADHD in Britain and the United States. ADHD research in the United States focuses on 'biology' and is confined to medical aspects. In contrast to the United States, in Britain there are much more data available to examine the ADHD phenomenon in a framework of social epidemiology. Instead of just biological explanations, these data show that the diagnosis of ADHD is dependent on environmental forces. Factors such as social class, health inequality, stressful life events, race, and parents' education all play a role in who gets diagnosed and is treated with medication. Unfortunately, there

is little chance of doing this type of analysis in America as the overwhelming emphasis, coming from institutions such as the National Institute of Mental Health, is on searching for deficits within children's brains.

Part Three: ADHD Drug Therapies

In Chapter 9, Grace Jackson reviews the evidence on the effects of stimulants on the growing body and brain and concludes with some passion that 40 years' evidence has shown that stimulants are a prevalent source of developmental toxicity: disrupting the formation of cartilage, myelin (white matter), and neurons (grey matter); altering endocrine functions; disturbing the sleep cycle; and destroying the brain's capacity to respond to future experiences with healthy rewiring and new growth. She also concludes that there is no evidence that these drugs reverse or normalize alleged delays in maturation, but may, in fact, preclude or postpone the development of self-control, abstract thought, and other forms of higher cognition.

In Chapter 10, Jonathan Leo and Jeffrey Lacasse document the unfounded claims made in consumer advertisements of ADHD medications. They illustrate how the advertising claims that are made are controversial from a scientific standpoint, and at best, most of them should be explained as tentative hypotheses, not as they are presented – as well-established facts. The degree to which this advertising is shaping the public's perceptions of the issues should not be underestimated. The full impact that the consumer advertisements have had on the diagnosis and treatment of ADHD requires further study, but the lack of well-balanced, scientifically based information on ADHD in these consumer advertisements is troubling. They hypothesize that they are very effective at having their intended effect: guiding patients to the doctor and even guiding the subsequent conversation with the doctor. The net effect on the public, and the children who end up taking ADHD medications, they conclude, is likely to be negative.

In Chapter 11 David Cohen, Shannon Hughes, and David Jacobs explore the extent to which new critical awareness, as well as parallel regulatory requirements, may have impacted on the initial clinical trials of Strattera™ (atomoxetine), a drug manufactured by Eli Lilly and Company and approved for marketing by the United States Food and Drug Administration (FDA) for the treatment of ADHD in children and adults in November 2002. They find extensive discrepancies

between published and non-published versions of clinical trial data, and problems with business-as-usual ways of evaluating psychoactive drugs for human consumption, resulting in likely preventible harm occurring to those prescribed this drug. They conclude that the clinical trials leave the clinician completely unprepared for the reality of the complex and unpredictable effects of psychoactive drugs such as Strattera™.

In Chapter 12, Basant Puri first reviews the drawbacks associated with conventional pharmacotherapy for ADHD and then reviews the evidence for the safety and efficacy of various nutritional interventions. He concludes that while there are many adverse side-effects from conventional pharmacotherapy for ADHD, there appears to be good evidence in support of the alternative use of fatty acids and of removing artificial colourings from the diet. Based on the evidence detailed in his chapter, Puri believes that an ideal treatment would be to remove all artificial colourings and other additives from the diet, in combination with supplementation with ultra-pure eicosapentaenoic acid (EPA) and evening primrose oil with no docosahexaenoic acid (DHA) present.

Part Four: Alternative paradigms for ADHD

In Chapter 13, Jon Jureidini argues that although 'ADHD' may represent a conceptual advance on 'hyperactivity' and 'minimal brain dysfunction' by formulating behaviour problems in terms of deficits of attention and other executive functions, it still suffers from being an overly reductionist concept to the point of being a 'description masquerading as an explanation'. Brain and mind functioning is more complex than the current ADHD conceptualization would have us believe. In his chapter he shows how the concept of 'self-regulation' helps to make better sense of children's behaviour problems. He argues that the behaviourally disturbed child is compromised in their capacity to use imagination to perform what Jureidini calls 'mind magic' that is needed to deal with their predicament, so that she or he signals their need for outside help in self-regulation through displays of affect, and/or seemingly dysfunctional behaviours.

In the semi-autobiographical Chapter 14, Simon Sobo provides a quasi-anthropological study of childhood in a Jewish community of 1950s Queens in New York. Drawing on memories of his childhood

there, Sobo concludes that it has always been known that it is difficult to get children to do what you want them to do rather than what they want to do. He suggests that the symptoms of ADHD describe children when they cannot connect to imposed expectations. Sobo then argues that as culture changed from one in which moral concerns were at the centre of experience, to a more pleasure-oriented, stimulus-bound existence it becomes easier to get bored and distracted when work rather than fun is the agenda, and thus expression of ADHD behaviours has become more common.

In Chapter 15, Chris Mercogliano using the proverbial 'canary in the coal mine' analogy, compares childhood in modern America today with its quite recent past and suggests that labels such as ADHD are the new millennium's canaries in the coal mine highlighting that something has gone wrong with America's modern beliefs about and practices with children. He proposes that children attracting labels such as ADHD are not 'sick', as the medical establishment would have us believe; rather they are exhibiting signs of distress and unmet core needs. Mercogliano argues that what he calls their 'inner wildness' is being stifled. He believes that the right response to their 'signals' is not to classify and drug them, but to help them reclaim this 'inner wildness', and restore childhood to a place in which kids can grow slowly, and if need be, fitfully into a more 'authentic' self.

In Chapter 16, Thom Hartmann makes a passionate case for getting beyond seeing ADHD as 'pathology' and finding positives and strengths within the ADHD-labelled person. He argues that ADHD is best viewed as lying on a continuum of behaviour rather than as a discrete biological 'malfunction' of the brain. The focus on ADHD as a 'brain pathology' that needs correcting, else it causes all manner of poor outcomes, adversely affects the growing child's sense of themselves and their value to society. Hartmann believes that it is time for us to set aside these stories of sickness and villainy. It is time for us to look at the structure and nature of our schools. It is time for us to tell the rogue elements within the research community who seek to stigmatize our children with the 'no hope, no value' label of ADHD that we are not interested in our children being the villains in their dramas any more. Science does not support the absolute pathology model, common sense doesn't support it, and certainly any sincere hope for therapeutic outcomes and healthy children does not support it. For Hartmann it is time to look at ADHD in a new light, where strengths

and abilities are noticed and self-esteem is protected in a world where all children are valued for their unique gifts.

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