

## **NEURODEVELOPMENTAL DISORDERS: ADHD AND AUTISM**

Iain McClure and Matthew Smith

### **LEARNING OBJECTIVES**

In this chapter, students will develop an understanding of attention deficit hyperactivity disorder (ADHD) and autism by examining:

- The primary symptoms and hallmarks associated with ADHD and autism
- Why ADHD and autism became popular childhood diagnoses during the second half of the twentieth century
- The many theories proposed to explain ADHD and autism
- Debates about the various treatment alternatives for ADHD and autism

### **INTRODUCTION**

How do we know when a child is developing normally? In terms of physical development, we have numerous indicators, ranging from height and weight to dexterity and strength. But what about the development of intellect, emotional stability, or the ability to develop social relationships? How do we know when a child is developing normally in these areas? How do we know what is normal in the first place? And what should we do when a child's development in one of these areas appears to be impaired? Is it the child that should be adjusted? Or should we focus more on adjusting the environment in which the child lives?

During the last fifty years or so, focus on intellectual, emotional, and social development has increasingly centred on the brain or central nervous system. As such, impairments in such developmental areas have tended to be described as neurodevelopmental disorders. In this category is included a large and diverse array of conditions, including intellectual disabilities, foetal alcohol

spectrum disorder (FASD), motor disorders (including Tourette's syndrome), communication disorders, genetically determined disorders (including Down syndrome and Williams syndrome), and various conditions associated with traumatic and congenital brain injuries (such as cerebral palsy). Two of the most common neurodevelopmental disorders are the ones that are addressed in this chapter: ADHD and autism.

One look at this list reveals just how broad and varied the category of neurodevelopmental disorders is. Not only are such disorders characterized by a wide variety of symptoms, but these symptoms can also vary considerably in severity. While we tend to think of neurodevelopmental disorders in terms of psychological symptoms or intellectual disability, they are also associated with a range of physical symptoms, ranging from the motor difficulties caused by cerebral palsy to the cardiac defects that often accompany Down and Williams syndromes. Many neurodevelopmental disorders also come with distinct physiognomic characteristics which not only make them recognizable, but also hint at their distinctiveness. The causes of such disorders are also bewilderingly diverse. While genetic factors, perinatal trauma or injury, or brain damage are the primary cause of some such disorders, the precise causes of others remain obscure or a subject for debate.

So what do such disorders actually have in common, then? Ultimately, neurodevelopmental disorders as commonly understood all share two features: 1) the idea that at the root of such disorders is a neurological dysfunction that affects the individual's capacity for intellectual, emotional, social and – in some cases - physical development; and 2) the fact that individuals diagnosed with such disorders often struggle to fit into society. As this chapter will suggest with respect to ADHD and (to a lesser extent) autism, too much focus on addressing and trying to “fix” the presumed neurological problem believed to be causing such disorders has meant that we have spent too little time and effort addressing alternative explanations for such disorders *and* trying to “fix” society so that it is more accommodating to those with neurodevelopmental disorders. While this is truer for ADHD and autism than it is for other neurodevelopmental disorders (especially disorders, such as FASD, which have a specific and

preventable cause), it nevertheless is a point worth remembering with respect to all neurodevelopmental disorders and mental illness more generally.

We have chosen to focus on ADHD and autism in our discussion of neurodevelopmental disorders for a number of reasons. First, ADHD and autism are by far the most common of the neurodevelopmental disorders, with ADHD being the most common childhood psychiatric disorder in most countries. From being unheard of before the Second World War, ADHD and autism are now diagnosed with regularity across the globe, with rates increasing rapidly since the 1980s. Second, these two disorders are arguably the most complex, controversial, yet also compelling of the neurodevelopmental disorders with respect to what they represent about children, society, and our understanding and tolerance of behaviours that diverge from the norm. The symptoms that characterize ADHD and autism are bewilderingly diverse and can be plotted on a continuum stretching from those that are relatively normal, especially for children, to those that can be profoundly disabling, especially in certain environments or in particular social situations. Both disorders have also been subject to almost continuous debate since they emerged during the post-war period about their causes, treatments, and even whether they exist or not. Third, both disorders became popular at roughly the same period of time, raising questions about the historical context in which they emerged. Finally, while ADHD and autism share some important characteristics that we will explore, they also have many differences, thus highlighting how generalizations about neurodevelopmental disorders are not always helpful in trying to understand them.

In addition to examining the questions listed above, this chapter discusses two primary themes with regard to ADHD and autism:

First, students will examine the framing of ADHD and autism as twentieth-century epidemics. How can we best explain the dramatic rise in rates of these disorders over the last sixty years? Students will learn about the range of explanations, tied largely to broader social changes: trends in parenting styles,

growing intolerance of difference, alterations in food production and diet, technological and scientific developments, and even the broader geopolitical context in which the disorders emerged.

Secondly, this chapter asks students to think about how individual identity may be affected by mental health-related diagnoses. Both of these disorders are primarily diagnosed in childhood, a key period of identity formation. How does a diagnosis (and often a prescription) alter a person's identity? And what happens to an individual's identity when diagnostic practices change over time? When *DSM-5* was released, for example, many people who had developed identities as "aspies" (those diagnosed with Aspergers syndrome – something they saw as quite distinct from autism) were shocked to discover that their diagnosis was rolled into autism spectrum disorder or ASD. Students will also examine the emergence of the neurodiversity movement, which seeks to reposition autism- and ADHD-related disorders as part of regular human variation rather than as a deviation from a single standard of "normality."

## **CLINICAL PERSPECTIVES**

### **ADHD**

When we think of a child with ADHD, a few different images come to mind. We tend to think of a boy (boys are at least three times more likely to be diagnosed with the disorder in most countries) who struggles to sit still, who fidgets, who lacks the ability to organize himself effectively, who has difficulty concentrating or staying on task, and who has trouble resisting impulses, including, at times, the urge to act out angrily or aggressively. Indeed, ADHD, as the term suggests, is characterized by inattentive, hyperactive, and impulsive behaviour. But it is also worth considering the contexts in which these ADHD symptoms are most problematic. Although the symptoms of ADHD might cause problems in a variety of settings, it is usually at school where they are first identified, and when they were first identified historically, specifically during the late 1950s in the United States. Schoolchildren are expected to stay at their seat, attend to their work, and repel the urge to interrupt their teacher or to disrupt the work of their fellow classmates; children diagnosed with ADHD often struggle with such

expectations. ADHD behaviours are seen to interfere with learning - or at least learning in traditional, subject-centred and teacher-centred settings. In other settings, such as in the playground, on the basketball court, or even in certain types of employment, the symptoms associated with ADHD may be less problematic, and might even be productive. As such, children diagnosed with ADHD may find it easier to succeed at schools that engage in active, flexible, and hands-on learning.

It is clear that most children exhibit hyperactive, impulsive, and inattentive behaviours at different times and places, and – as we discuss below – for different reasons. But it is important to remember that these behaviours are not particularly unusual, especially for younger, less mature children. Most clinicians would argue that it is the frequency and persistence of such behaviours, as well as their effect upon learning that generally lead to a diagnosis of ADHD. Although this may well be true, it is nevertheless worth unpacking how this process tends to take place.

Children diagnosed with ADHD are typically first identified as potentially having the disorder by someone at their school, following either misbehaviour, poor academic achievement, or a combination of these factors. The student may then be referred to a psychologist, psychiatrist, pediatrician, or general practitioner for diagnosis and treatment. If a clinician uses the fifth edition of the *Diagnostic and Statistical Classification of Mental Disorders (DSM-5)* checklist in order to make the diagnosis, the child must have presented six out of eighteen symptoms consistently over a period of six months. Among these symptoms include “often have difficulty waiting their turn,” “often talks excessively,” “often fails to give close attention to detail or makes mistakes,” or “often does not follow through on instructions and fails to finish schoolwork or workplace duties.” While these sorts of “symptoms” may certainly be problematic, it is easy to see how they can be interpreted rather subjectively, leading to over-diagnosis of the disorder. A recent estimate of the global prevalence of ADHD suggested that over 5% of children had the disorder: does this mean that millions of children have a psychiatric disorder or that we are simply branding relatively normal behaviours as pathological? (Polanczck et al, 2007).

Moreover, many of these sorts of behaviours may also be associated with immaturity, rather than an inherent neurodevelopmental disorder. A series of recent studies focussing on American, Canadian, Taiwanese, Swedish, and Icelandic children have all indicated that the youngest children in a school cohort are the most likely to get an ADHD diagnosis and even more likely to be prescribed medication. One of the American studies found that children born one month before the cut-off point for kindergarten entry were 122% more likely to be diagnosed with ADHD and 137% more likely to be medicated than their eldest classmates (Elder, 2010). Although another American study found that children born 1-3 months before the cut-off were 27% more likely to be diagnosed and 24% more likely to be medicated than those born 10-12 months before (a wider scale than many of the other studies), the authors estimated that these differences meant that “approximately 1.1 million children received an inappropriate diagnosis and over 800,000 received stimulant medication due only to relative maturity” (Evans, Morrill, and Parente, 2010, 672). That such a simple factor has been overlooked for so long in the incessant rise in ADHD diagnoses highlights the need to scrutinize carefully the full array of factors that contribute to childhood misbehaviour.

*Box: A rose by any other name...*

Although the term ADHD is widely used today, it was not the first term to describe hyperactive, impulsive, and inattentive children. As stated below, hyperkinetic impulse disorder was the term coined by Eric Denhoff and Maurice Laufer in 1957, but despite its applicability, it was not widely used during the 1950s and 1960s. Indeed, in another 1957 article, they called the condition hyperkinetic behavior syndrome. Many physicians opted for the term minimal brain damage, which indicated the belief that such symptoms were normally caused by brain trauma. When it became apparent that some children had no history of such injury, the term minimal brain dysfunction was used instead. Other physicians preferred the term hyperkinesis or simply hyperactivity, and in *DSM-II* (1968), the term hyperkinetic reaction of childhood (or adolescence) was used.

Along with indicating the dominance of biological psychiatry in the US, *DSM-III* (1980) also introduced the term Attention Deficit Disorder with or without Hyperactivity or ADD/ ADD-H into popular usage. The focus on attention signified a shift from focussing on hyperactivity or impulsivity as the predominant feature of the disorder, to focussing on inattention. This transition meant that quieter, less disruptive, but nonetheless inattentive children (including girls) and later adults could now be diagnosed with the disorder. When *DSM-III* was revised in 1987, however, the term ADHD finally came into being, though ADD and some other terms were still used (the World Health Organization's International Classification of Disease still uses the term hyperkinetic disorder as well as ADHD). It was only after the term ADHD was introduced that the disorder began to reach epidemic proportions, first in the US, and then globally. With each of these changes in terminology came subtle - and sometimes not so subtle – changes in what was meant by the disorder, who could be diagnosed with it, and what it said about the children who had it. The explosion in ADHD diagnoses has not simply been down to a name, but it is important to recognize the power labels have and what they convey about a disorder.

## **Autism**

As with ADHD, there is a popular image of autism that does not always reflect reality. Dustin Hoffman's Oscar-winning portrayal of Raymond Babbitt in *Rain Man* (1988), for instance, was one of the first portrayals of a person diagnosed with autism that reached a wide audience. Raymond exhibits many of the social, communication, and compulsive behaviours associated with most people with autism, but he also savant skills, such as extraordinary recall, that are much rarer. While Raymond Babbitt may be one of the first images that comes to mind when autism is mentioned, he is not particularly representative. More recent portrayals of people with autism, such as the Danish detective Saga Norén in the Danish/Swedish crime drama *The Bridge* focus somewhat less on savant skills, but nevertheless depict people with autism as highly capable of achieving success (at least in some respects). Such positive portrayals are good in that they demonstrate the capabilities of people with autism, but do not convey the full range of people who may be placed on the autism spectrum (along with the less

characteristic, but still common symptoms associated with the disorder), nor do they always indicate the array of challenges faced by them and their families.

Autism is a curious condition in many ways. Essentially, the problem is that the person, to a greater or lesser extent, has difficulty understanding the need to be social. Being “social” is the instinctive thing that mammals do, again, to a greater or lesser extent, to maintain the survival of the pack (whatever form that might take). In human terms, the pack is the family and one can speculate that, in prehistoric society (which occupies the vast majority of human history) having autism may not have been helpful when it came to survival in early years. This suggests that autism must convey some other benefits to being human which have counterbalanced this risk of a- or dys-sociality in toddlers and teenagers. For example, one could speculate that if a human with autism did make it to adulthood (albeit that lifespans were probably much shorter in early human history, with people probably dying soon after their teeth fell out, for obvious reasons) the need to be social might have been less important for survival in some of the harsher climates and isolated environments that humans had to face. Having autism might have been positively advantageous in certain situations.

The problem now is that, with the shift to urban lifestyles during the last 200 years, especially in the industrial world, people have been living together in more concentrated social environments. The person who was the (at least tolerated) village eccentric up until even the early part of the twentieth century has experienced, and posed, more intense social challenges in the ensuing period. It is probably no coincidence, therefore, that the autism concept (Evans, 2017) did not begin to evolve until the 1940s and the prevalence has been steadily increasing since then, in the context of social change discussed earlier in this chapter in relation to ADHD. One can also reasonably speculate that one of the causes of increasing rates of psychiatric disorder will be that of humans of all ages with unrecognised autism struggling to cope with the pressure of social life. This is certainly the experience of the clinician. The main clinical task in autism management, in many ways, is to help everyone decide, including the



person themselves (depending on their age) if they have autism or not. It is quite likely that there are several ‘autisms’ leading to a ‘final common pathway’ of clinical presentation.

It is important however, that the student realises that ‘autism’ (and all neurodevelopmental conditions for that matter) is not a medical condition in the same way that a physical disease is. We call this concept ‘reification’. In physical disorder, for example tuberculosis, a bacterium causes caseating lesions in the lungs which lead to clinical symptoms and signs, such as coughing up blood. Once the patient presents, they can have an x-ray which shows up the lung problem and sputum samples will reveal the nasty TB bugs under the microscope. There is no shadow of a doubt (pun intended!) that TB is a real ‘thing’ that someone has got and, if it is not treated, it may, ultimately, kill them. No such concept or pathological process exists for any psychiatric condition - autism, ADHD, schizophrenia, depression or anorexia nervosa. Dementia (presenting at any age) is the one exception and is therefore the only psychiatric disorder which is unarguably “real” or “reifiable” in medical terms. Whatever is causing the non-dementia psychiatric problems in the brain, CT or MRI scans will show absolutely no difference from normal brains (with minimal exceptions), as with genetic screens. If there was a cause for autism we would have heard it shouted loud from the rooftops, because autism is extremely expensive to society (Buescher, Cidav, Knapp and Mandell, 2014) ).

Instead, all we can do currently is *describe* the condition, as it presents, much like physicians of the early 19th century had to describe TB before they discovered the tubercle bacillus and its pathological process. In order to do this, psychiatrists have evolved categorical classification systems. *DSM-5* and *ICD-11* (immanent) being the latest versions of such systems. They are all limited in many ways and, inevitably, with the passage of time, these limitations fail to match up with evolving information and they therefore require revision.

## Emergence of ADHD and Autism

### ADHD

Since the symptoms of ADHD are somewhat synonymous with childhood more generally, it has been easy for physicians and some historians and social scientists to pore through medical journals and other literature of times past and find examples of what appears to children with ADHD. Perhaps the most famous example of this is Fidgety Philip, the creation of German physician Heinrich Hoffmann (1809-1894), who depicted Philip along with many other troublesome children in his 1844 book of nursery rhymes *Struwwelpeter or Merry Stories and Funny Pictures* (Zipes, 2002). Others have claimed that the clinical depictions of misbehaving children by Sir Alexander Crichton (1763-1856), Sir Thomas Clouston (1840-1915), and Sir George Still (1868-1941) are also examples of ADHD (Palmer & Finger, 2001). Finally, many historical figures, ranging from Oliver Cromwell and Winston Churchill to Wolfgang Amadeus Mozart and Lord Byron, have been retrospectively diagnosed as having ADHD, often in an attempt to prove that ADHD is a “universal” and “essential” condition that has always existed in the human population (Fitzgerald, 2000; Fitzgerald, 2001; Siddiqui & Fitzgerald, 2003; Doyle, 2004; Timimi & Maitra, 2009, 203-4).

There are a number of problems with this approach to tracing ADHD’s apparent history. The first is that, when one examines such examples in close detail, it becomes apparent that the children depicted retrospectively as having ADHD do not always bear that much similarity to children diagnosed with the disorder today. For example, Still’s 1902 Goulstonian lectures on defect of moral control in children, which are often stated as the first description of hyperactive children, actually depicted children who were seriously disturbed, capable of extreme violence, and likely to end up in an institution. Unlike the millions of children diagnosed with ADHD today, Still stated that it was very difficult to locate such children, indicating that they were rare, not ubiquitous. Historical figures, such as Mozart and Churchill, may have presented behaviours that seem like the symptoms of ADHD, but often it was these very characteristics that made them successful, raising questions about how we perceive and understand

exceptional children today. Perhaps most tellingly, during the first half of the twentieth century, the child most of concern to pediatricians and psychiatrists was not the one who was hyperactive and impulsive, but the one who was shy, withdrawn, and neurotic, as the title of the mid-twentieth century journal *Nervous Child* attests. Finally, when the symptoms of ADHD were recognized in children prior to the 1950s, they tended to be associated with either brain damage (either caused by infection, perinatal damage, or physical injury) or food allergy (Clarke, 1950; Smith, 2015). Again, in these cases, the symptoms presented by brain damaged or allergic children tended to be more severe than those seen in most children diagnosed with ADHD today and, in the case of food allergy, were effectively treated once the problem foods were identified.

*BOX: Dennis the Menace*

During the spring of 1951, one of the strangest coincidences in the history of comic books occurred. Within five days of one another both the British and the American Dennis the Menace made their first appearances on the public stage. While the dark-haired Dennis graced the pages of the *Beano*, the fair-haired American Dennis quickly became a fixture of American newspapers. Described as a precocious, enthusiastic, and energetic 5 1/2 year-old-boy, the American Dennis, like his British counterpart, is a habitual trouble maker whose adventures often end up irritating his long-suffering neighbour, Mr. Wilson, whom Dennis paradoxically regards as his best friend.

Much of Dennis' popularity arose from his recognisability. Kids like Dennis were on every street in the burgeoning American suburbs during the 1950s, tearing around corners on their tricycles or hitting baseballs into kitchen windows. They were thought to be normal boys, doing what boys did. Or were they? Within a decade of Dennis the Menace's first appearance, his exuberant, mercurial tendencies were beginning to be seen in a very different light. Boys like Dennis were increasingly described as impulsive, hyperactive, and inattentive, and being referred by school counsellors to physicians for medical treatment. Instead of being seen as part of the fabric of American society, like Tom Sawyer

and Huckleberry Finn, boys like Dennis were being diagnosed with what we now call ADHD, and prescribed medication to treat their pathological behaviour.

As this chapter explains, the emergence of ADHD can be linked closely to the space race and the demand for a highly-educated workforce. It is ironic, then, that Dennis' father, Mr. Mitchell, is an aerospace engineer. And, just like Americans sought to win the space race – and the Cold War – through their technological superiority to the Soviets, technology, this time emanating from pharmaceutical laboratories, would also be employed to deal with boys like Dennis. By the mid-1960s, drugs such as methylphenidate (Ritalin) dominated the treatment of hyperactive, impulsive, and inattentive children.

But although it is true that millions of “real life” Dennis the Menaces have been prescribed medication to treat their supposedly pathological symptoms, it is also telling that the figure of Dennis the Menace – and boys and girls like him – has endured, undiagnosed and un-medicated. It is the boisterous, super-charged, impetuous, and imaginative nature of figures like Bart Simpson, Calvin (from the *Calvin and Hobbes* comic strip), Ramona Quimby (from Beverley Cleary's *Beezus and Ramona*), and, of course, both versions of Dennis the Menace that endears us to them.

Rather than trying to uncover unconvincing examples of ADHD in times past, it is much more useful to determine when hyperactive, impulsive, and inattentive children were first identified as particularly problematic and why this was the case. When one does this, the year 1957 becomes important. This is because 1) the first description of a disorder that resembles what we now call ADHD is introduced in 1957; and 2) the Soviet launch of *Sputnik* (Smith, 2012).

In 1957, a team of child psychiatrists, based at Rhode Island's Emma Pendleton Bradley Home coined the terms hyperkinetic impulse disorder and hyperkinetic behavior syndrome (Laufer, Denhoff, & Solomons; Laufer and Denhoff). Even if the terms did not stick (see box below), the concept did. The

condition Laufer, Denhoff, and Solomons described was specific enough to delineate a specific type of child, yet broad enough to be applicable to millions of children. It included both children who had a history of brain damage, which amounted to about 10 percent of the total in their study, and those who had no such history. Finally, they acknowledged that the symptoms that made up hyperkinetic impulse disorder were not all that dissimilar to the behaviours exhibited by ordinary children. Children, “as compared with adults ... are hyperkinetic, have short attentions span and powers of concentration, and are impulsive ... In the course of their development, they outgrow this mode of behavior and actually, in the course of time, so do most of the children with the hyperkinetic syndrome” (Laufer, Denhoff, & Solomons, 1957, 45). As pediatrician Howard Fischer stated in an article commemorating the fiftieth anniversary of the term, there were only minor differences between the 1957 description and how ADHD is described today (2007).

The other development that made it possible for ADHD to become the most widespread child psychiatric disorder had to do with the broader geopolitical context: the Soviet launch of *Sputnik-I* on the 4th of October, 1957. *Sputnik* convinced American politicians, scientists, and educators that they were losing the brain race with the Soviets and that, if the education system was not radically transformed, the US was in danger of losing the Cold War altogether. For many commentators, *Sputnik* proved that progressive education, the child-centred, hands-on, and flexible approach to learning that had dominated American education since the 1930s, was not up to the job of producing the scientists and engineers who would win the space race (Ravitch, 1983). Amongst the slew of works published by the critics of progressive education in the aftermath of *Sputnik* were books such as Admiral Hyman Rickover’s *Education and Freedom* (1959), James Conant’s *The American High School Today* (1959), Arthur S. Trace’s *What Ivan Knows that Johnny Doesn’t* (1961) and Max Rafferty’s *Suffer, Little Children* (1962). As Rickover, better known as the “father of the nuclear navy” declared, “the schools are letting us down at a time when the nation is in great peril. To be undereducated in this trigger-happy world is to invite catastrophe” (1959, 32).

The most tangible example of such concern was the passage of the National Defense Education Act (NDEA) in 1958, which provided \$1 billion to improve performance in core subjects, including science, mathematics, English, and foreign languages, to prevent students from dropping out of school too early, and to hire guidance counsellors in order to ensure these aims were realized. The combination of these objectives, along with a number of other factors which will be discussed below, created a situation in which “hyperkinetic impulse disorder” could morph into a widespread, ubiquitous disorder amongst American children. The renewed focus on core subjects, along with the rejection of progressive education, meant that classrooms were transformed from being active spaces where learning could be done on the move, to static spaces where students were expected to sit and learn at their desks, which were now typically arranged in a grid pattern facing the teacher. Rather than learning “by doing,” (for instance learning about science, mathematics, and language by growing vegetables and selling them at a stand using advertising), students now had to learn “by the book.” In this setting, children who were naturally more active, inclined to move around the classroom, and adept at hands-on learning struggled to succeed.

NDEA also put a premium on students staying in school as long as possible, in the hope of developing a highly educated workforce. Students were not only expected to graduate from high school but go on to college and university. Dropping out of school for work as an “uneducated muscleman” was no longer thought to be an option; indeed, it was believed (as it is once again today) that automation would reduce the need for such workers altogether (Warren, 1964, 550-51). Moreover, as James Byran Contant’s *Slums and Suburbs* attested, it was not merely students from American suburbs who were supposed to stay in school, but also those from the slums of American cities (1963). Adding to these expectations was the legacy of the GI Bill (Servicemen’s Readjustment Act of 1944), helped 2.2 million veterans attend college or university by 1956, many of whom would have been the first in their family to achieve such levels of education. Although the legacy of the GI Bill may be mixed (Frydl, 2009), it certainly helped create the expectation that post-secondary education was a necessary condition for success. What all of this meant with respect to ADHD was that children who would have likely left

school in their early- or mid-teens for employment as manual labourers were now expected to complete high school or, ideally, go on to university. When children struggled to achieve such levels of academic success – especially if their average or high IQs suggested that they should be doing better - they were singled out as particularly problematic; the behaviours associated with such under-achievement often mapped onto what we now call ADHD.

In order to identify these children, NDEA provided funding for the hiring of thousands of guidance counsellors, at all levels of American education. Guidance counsellors served as the intermediary between the educational sphere, where such children were identified as problematic, and the medical sphere, where they were diagnosed as having a psychiatric condition. Conant believed that the student to guidance counsellor ratio should be around 250-1, and that counsellors should be particularly attentive to bright boys and girls who tended to underachieve (1959, 44-45). This image of the intelligent child who struggles to succeed at school quickly became associated with ADHD.

Although the impact of *Sputnik* on American education was vital in catalyzing interest in hyperactive, inattentive, and impulsive children, it was not the only factor that gave rise to ADHD. The post-war period also gave birth to the 75-million strong baby boom generation, the largest cohort in American history. Baby boomers were not only seen as vitally important in winning the Cold War, but also overcrowded a school system that was already overstretched from years of underinvestment. These children, who were increasingly raised in the burgeoning American suburbs, were the first to watch television, to eat highly processed foods, and to rely primarily on the automobile for transportation. They also, partly because of the renewed focus on education and the rise of the suburb, spent less and less time outdoors engaging with nature. Finally, they were a generation that saw the start of a gradual move against corporal punishment. As discussed below, all these factors have been cited as having a role in children's behaviour.

Factors within American psychiatry after the Second World War also fuelled interest in ADHD.

American psychiatry reached its zenith in terms of influence during these decades. During the Second World War, the importance of psychiatry increased, as the minds of soldiers were seen to be as vital to the fight as their bodies. Following the war, psychiatrists were able to capitalize on their newfound prestige, with the foundation of the National Institute of Mental Health in 1949 being but one example of the priority placed on mental health in the US.

While psychoanalysis might have dominated American psychiatry at war's end, social psychiatry and biological psychiatry were also on the rise (Smith, 2008). The development of new psychiatric drugs, such as the antipsychotic chlorpromazine and the antidepressant imipramine, in particular, suggested to biological psychiatrists that the answer to mental health problems was to be found in the brain. Along with Miltown and Valium, Ritalin became one of the wonder drugs that placed American psychiatry – and Americans more generally – on the path that led to widespread psychiatric medication for all manner of mental maladies.

Although ADHD clearly emerged as an American disorder, it has recently become a more global phenomenon. While the disorder spread to Canada, Australia, and New Zealand within a decade of it becoming widespread in the US, it took somewhat longer to emerge in the UK for a variety of reasons (Smith, 2017). In recent decades, ADHD has become common throughout the world, with rates of the disorder increasing in China and India, and Iceland now outpacing the US in terms of per capita consumption of Ritalin. Despite this story of medical globalization, however, it is also important to note the nuances in the global history of ADHD. Some countries, such as Finland (which ranks at the top of every education league table), have largely eschewed the disorder (M. Jahnukhainen, 2009). Others, including the UK, have been more willing to accept alternative explanations for the disorder. Perhaps most importantly, rates of diagnosis vary considerably within countries, perhaps most notably in the US. In order to understand why ADHD has emerged where and when it has – thus coming to



grips with the disorder in a more sophisticated and comprehensive fashion – it is crucial to examine each of these stories and learn from them.

## **Autism**

Autism, as with ADHD, is a disorder that largely emerged in the twentieth century. It is possible to retrospectively diagnose historical figures, ranging from the wild boy of Aveyron to Isaac Newton, with autism, but it is more important to understand why physicians started to diagnose children and adults with autism in the twentieth century. Where and when to trace the origins of autism, however, have been a matter for debate. Most historians and physicians point to the pioneering articles of Austrian-American psychiatrists Leo Kanner and German paediatrician Hans Asperger, published in 1943 and 1944 respectively, as the first descriptions of autism. British autism researcher Lorna Wing would popularize the term Asperger's syndrome (named after Asperger) in 1981 as referring to a distinctive, yet related, condition to autism, though whether or not Asperger's syndrome should be part of the autism spectrum or a separate disorder remains a contentious issue (Wing, 1981). Others, including, historian Bonnie Evans, however, have argued that the origins of autism should be traced further back to the work of Eugen Bleuler (who coined the term autism in 1911), as well as French psychologist Jean Piaget, whose model of autism was introduced into Britain via his book, *The Language and Thought of the Child* (1923; 2016).

As with ADHD, however, it takes more than simply a term to create a widely diagnosed disorder. The twentieth century, once described as the “century of the child,” saw a pronounced increase in childhood as a focus for research and social intervention (Key, 1909). Child guidance clinics, for instance, emerged in the US during the early twentieth century and then spread to the UK and elsewhere (Stewart, 2013; Kritsotaki, 2014). With Freudian psychoanalysis in the ascendency for the first half of the twentieth century, childhood took centre stage as a developmental period that could explain later mental health problems. Child psychiatry also emerged as a specialty, with the 1937 edition of Kanner's own *Child Psychiatry* textbook serving as the first of its kind. The deinstitutionalization

witnessed in mental health after the war not only meant that many adults with autism were now returned to their communities, but also that children with mental health problems and learning disabilities would depart the mental deficiency institutions in which they had been placed, and now receive care in the community; a parallel rights-based movement saw such children taught in integrated classrooms with “normal” pupils (Carey, 2009; Waltz, 2013; Kritsotaki, Long, & Smith, 2016; Evans, 2017). Indeed, Evans has argued that the construction of the notion of child rights during the late 1950s caused “the metamorphosis of the autism concept” (Evans, 2017, 415). Finally, as seen with ADHD, the educational field after the Second World War saw an added focus on achievement and attainment, as well as the drive to label children who struggled to succeed.

The admixture of all of these factors – and more – was to create a scenario in which unusual childhood behaviour was readily identified as problematic and pathologized: transformed into a medical malady. Of course, as a spectrum disorder, autism could affect an individual in a profoundly disabling manner or more mildly. But, once the “narrative structure” of an autistic child emerged and became more widespread, it was easier to identify such children, test them, and diagnose them (Waltz, 2013, 63). When the purportedly milder Asperger’s syndrome entered the medical lexicon, even more children could be placed on the autism spectrum. Normality, apparently, narrowed.

### **Explanations for ADHD and Autism**

Today, most psychiatrists will describe ADHD as a genetic neurological deficit. Researchers have spent millions, attempting to provide evidence for this explanation, mapping the brains of children with ADHD and trying to identify an ADHD gene. Such explanations gloss over the fact that many other explanations have been cited for the symptoms of ADHD, some of which require re-examination.

As explained above, the sort of hyperactivity, impulsivity, and inattention associated with ADHD was not seen as particularly problematic by psychiatrists or paediatricians until the late 1950s. When it was highlighted, however, a number of different explanations were put forward. Brain injury, caused by

infection (such as in the case of post-encephalitic disorder during the 1920s) or some other form of trauma, was often associated with such behaviours. Such theories, which were largely developed during the 1930s and 1940s, gave rise to the term “minimal brain damage,” one of many terms previously used to describe ADHD (see box above). But when the medical literature of the first half of the twentieth century is scoured, the most common explanation for such behaviour is something not usually suggested today, namely, food allergy (Smith, 2015).

The term allergy was coined in 1906 by Austrian paediatrician Clemens von Pirquet to describe pathological reactions to pollen, insect stings, animals, and food. By the 1910s and 1920s, American physicians were describing how food could cause behavioural problems in children (Hoobler, 1916; Shannon, 1922; Piness and Miller, 1925; Duke, 1925). Unlike the descriptions of so-called hyperactive children by Still, Clouston, and others – as well as the depictions of brain-injured children also associated with ADHD – these food allergy reactions are very similar to how ADHD would be described today.

A compelling insight into the link between food allergy and ADHD symptoms can be found in a 1950 article by allergist T. Wood Clarke. After giving a presentation to the American College of Allergy about food allergy and behavioural problems in 1949, Clarke was asked to survey other allergists to see how widespread the phenomenon was. Out of the 171 Canadian and American allergists surveyed, 95 of them assured him that allergies could indeed cause behavioural problems in children (58 of 171 did not treat children; 9 believed there was no link; and 7 believed allergy was psychosomatic – a widespread belief at the time). The descriptions of many of the cases provided to Clarke by his respondents are striking in how much they resemble children diagnosed with ADHD today (Clarke, 1950). Seeking to explain the physiology behind such reactions, Clarke suggested that, just as with other allergies, “neuro-allergy” or “cerebral allergy” was caused by swelling or inflammation which could impair the normal functioning of the brain (Clarke, 1948).

By the time of *Sputnik* and the coining of hyperkinetic impulse disorder, more allergists were writing about the relationship between allergy and psychiatric symptoms, but internal divisions within the allergy community were pushing the advocates of such theories to the margins. One of the factors stoking disagreement in allergy was the belief of some allergists that the increasing numbers of chemicals in the environment (ranging from pollutants in soil, air, and water to food additives) were responsible for an increasing number of allergic reactions, including those affecting behaviour (Randolph, 1962). These debates became most fervent during the 1970s and early 1980s when San Francisco allergist Ben F. Feingold (1899-1982) made the connection between food chemicals and hyperactivity in children and advocated a food additive-free “Feingold diet” (which he called the Kaiser Permanente or K-P diet after his employer) as an alternative to stimulant medication (Smith, 2011). The popularity of the Feingold diet can be attributed to a number of factors, ranging from broader fears about chemicals in food to Feingold’s decision to take his theory directly to the public via his Random House paperback *Why Your Child is Hyperactive* (1974), following unsuccessful attempts to publish his theory in leading medical journals. But most important was the fact that, by the 1970s, thousands of parents were dissatisfied with conventional treatments for their children’s behavioural problems and Feingold’s diet appeared to work. Although the popularity of the Feingold diet waned following his death in 1982, the Feingold Association of the US continued to provide support and guidance to parents and, early in the twenty-first century, a series of new studies appeared to provide support for the theory, particularly with respect to artificial food colours (Bateman, et al, 2004; McCann et al, 2007). Although 2011 FDA hearings decided by a “controversial” 8-6 ruling not to recommend banning artificial food colours, many food companies (ranging from Kraft to Marks and Spencer) have voluntarily removed such chemicals from their food products and in 2012, the EU made it mandatory for foods and drinks containing artificial colours to contain a warning label stating that such substances “may have an adverse effect on activity and attention in children” (Arnold, Lofthouse, and Hurt, 2012, 599; Food Standard Agency, 2012; Harrington, 2015).

Putting debates about food additives to one side, a growing number of researchers have begun to explore the possibility that malnutrition, ranging from vitamin deficiencies to simply not having anything for breakfast, could play a considerable role in causing ADHD symptoms. Indeed, Kellogg's recognized this possibility in a mid-1950s advertising campaign found in *Grade Teacher* magazine, which contended that all underachieving, troublesome children needed was a bowl of their Corn Flakes. Perhaps they were not so far off the mark.

Dietary theories, however, have not been the only controversial explanations for ADHD to emerge in the last sixty years. Psychoanalytic theories of childhood behaviour, for instance, often pointed to the role of parents, and usually mothers, in triggering such symptoms. The early issues of the *Journal of the Academy of Child Psychiatry* (founded in 1962), for instance, often posited psychoanalytical explanations that blamed faulty parenting. As was also the case with autism (see below), such "mother blame" contributed to the acceptability of neurological and genetic explanations which precluded the need to blame parents, despite the fact that parenting undoubtedly has a role in shaping child behaviour. Instead, given the influence of genetic theories of ADHD, any focus on parenting is now more likely to centre on the fact that the ADHD child's parent[s] are also likely to have the disorder and may struggle with aspects of parenting as a result. Such narrow and reductive thinking, represents an unfortunate over simplification, in terms of diagnostic formulation and is an interesting example of the current tendency for thinking about mental health to follow a "silo" mentality or, indeed, contain aspects of impulsive, even attention deficit evaluation of the clinical presentation.

Related to not only parenting, but also schooling, were changes in how children were disciplined. Although only one American state had banned corporal punishment in schools by 1957 (New Jersey in 1867), other states and municipalities began implementing bans in the 1970s, and physically punishing children either in the home or at school has become a criminal offense in many jurisdictions (Society for Adolescent Medicine, 2003; J. Durant and R. Ensom, 2012). One would hope that most people would support these developments, which have accelerated since the United Nation's General

Assembly ratified the Convention on the Rights of the Child in 1989. But eliminating corporal punishment does not negate the need to discipline children. It is ironic that, just as corporal punishment has become less tolerable, the practice of prescribing stimulant medication to children diagnosed with ADHD has become more acceptable. Has Ritalin, then, replaced the rod? (McClure, 2012)

A number of other explanations have emerged over the years to help explain ADHD, highlighting the many factors that may contribute to childhood behaviour. Paralleling Feingold's hypothesis about food additives was a quieter and less controversial theory about the effect lead could have on ADHD (Silbergeld and Goldberg, 1974). Others have argued that sleep deprivation, caused by anything from sleep apnoea to too much screen time at night, could be a causative factors (Bass, 2014). But perhaps the most controversial explanation for ADHD, however, is simply that it is a social construction; in other words, it does not exist. Given that it emerged in parallel with the anti-psychiatry movement of the 1950s and 1960s, when the work of RD Laing, Thomas Szasz, Erving Goffman, and Michel Foucault was raising serious questions about how mental illness was understood, it should not be too surprising that some were critical of a disorder that diagnosed misbehaviour as a medical disorder that required pharmacological treatment.

While "hyperkinetic syndrome" was already sparking debate amongst physicians, psychologists, teachers, and social workers by the mid-1960s (Schrager et al, 1966, 528), more sustained critiques of the disorder had emerged by the mid-1970s, including journalists' Peter Schrag and Diane Divoky's exposé *The Myth of the Hyperactive Child: And Other Means of Child Control* (1975). Schrag and Divoky's polemic claimed that pharmaceutical companies and school administrators colluded to make hyperactivity and the medication that went with it an acceptable, if not welcome, diagnosis. Aggressive pharmaceutical salesmen did not market drugs to beleaguered teachers and parents (who, along with physicians, were the targets of their campaigns), they sold the idea that hyperactivity, inattention, and impulsivity were not merely normal childhood behaviours, but the symptoms of a serious neurological

disorder that needed to be controlled. Similar critiques were also put forward contemporaneously by sociologist Peter Conrad, who rightly noted that the stimulant drugs typically prescribed for hyperactivity were available “long before the disorder was clearly conceptualized” (1975, 16).

In the decades that have elapsed since, claims that ADHD is a tool for social control that targets boys or children of certain races have continued to be proposed (Hopkinson, 2014). The global spread of ADHD has intensified such critiques (R. Whitaker, 2017), as have the broader concerns about psychiatric diagnosis that became prevalent with the publication of *DSM-5* in 2013 (R. Thomas, G. K. Mitchell, & L. Batstra, 2013). What the history of ADHD reveals, however, is that if there is an explanation for the disorder, it will be a complicated one that will undermine the simple genetic, neurological answers that have dominated mainstream medicine.

## **Autism**

If we can call the debates about the aetiology of ADHD controversial, it is difficult to find a strong enough word to describe those proposed about the causes of autism. There are a number of reasons for why this has been the case. Perhaps the most obvious one is that convincing explanations for autism have been slow to develop. As a neurodevelopmental disorder, autism is thought to be rooted in the brain, but whether genetic, environmental, or a combination or variety of factors are to blame has yet to be established. As Jennifer Singh has demonstrated, billions of dollars have been spent to find the genetic key to autism, but many questions remain (2016). In the vacuum of concrete answers, other explanations emerge.

Autism is also a disorder feared by parents. Although the emergent neurodiversity movement might assuage these fears to a certain extent (see below), the social, emotional, and behavioural challenges that come with autism are thought by many parents to be overwhelming. Autistic children are thought not only to be unmanageable, but it is also thought that they will never make the same emotional connection with their parents that “normal” children will. Will their child ever really love them? These

fears help to explain why parents would willingly forgo vaccination regimens, that will most certainly prevent the risk of their children contracting life-threatening infectious diseases, on the controversial and poorly supported theory that such vaccines could make their child autistic.

Finally – and partly due to its complex, confounding nature – autism has been a disorder that has fitted neatly into overarching medical theories, whether they have been authoritative or marginal in nature. Psychoanalytical, behavioural, genetic, dietary, and social theories have all been offered as an explanation for autism. In the midst of all these explanations, as Chloe Silverman has described, parents are often at a loss as to what to do (2012).

As Leo Kanner observed, autism was a disorder “that seemed to straddle the divide between the psychological and the neurological, between affect and brain chemistry” (Silverman, 2012, 32). Because of this, it tended to divide biological psychiatrists and psychoanalysts. Since Kanner himself did not offer any firm theory about the causes of autism, subsequent researchers were able to pick and choose from his writing in order to support their own views (Silverman, 2012). Although he is thought to have favoured biological explanations for the disorder, he described how among the parents of the autistic children he observed, there were “very few warmhearted fathers or mothers” (Kanner, 1943, 250). Moreover, the parents tended to be “highly intelligent,” obsessive, “strongly preoccupied with abstractions of a scientific, literary, or artistic nature, and limited in genuine interest in people. Even some of the happiest marriages are cold and formal affairs. Three of the marriages are dismal failures” (Kanner, 1943, 250). Whereas a genetically-minded researcher might have suggested that such parental characteristics were indicative of hereditary factors (something that Kanner doubted), other investigators focussed on the possibility that parenting played a major role in the onset of autism. The most influential and contentious theory linking parenting and autism came from Austrian-American psychoanalyst Bruno Bettelheim and his 1967 bestseller *The Empty Fortress*. Extending Kanner’s observations about the “coldness” of the parents of autistic children, Bettelheim emphasised this especially in mothers, contributing to the notion of the “refrigerator mother.” Now discredited for



a variety of reasons ranging from questions about his credentials and accusations of plagiarism to claims of abuse and simply ignoring evidence that countered psychogenic theories of autism, Bettelheim's psychoanalytical interpretation of autism during the 1950s and 1960s reflected the broader influence of psychoanalysis in American psychiatry at the time and, in many ways, dominated the field for many years (Feinstein, 2010, 54). It also mirrored contemporary ideas about maternal deprivation theory, which were articulated by British psychiatrist John Bowlby following the experiences of the Second World War (1951). "Autism," as Silverman has explained, "served as a focal point for ideas about motherhood, childhood, and development in twentieth-century America" (2012, 63).

Rather than putting his research in context and trying to tease out any meaning from it, however, most accounts have dismissed any possible contribution from Bettelheim to understandings of autism (Feinstein, 2010). Instead, his dominance is thought to have prevented those who wanted to promote biological explanations, such as Bernard Rimland (whose son was diagnosed with autism) from having more influence, thus retarding neurological understandings of autism (1964). While there might not be much admirable in Bettelheim's approach, personality, or in his actual ideas about autism, such blanket rejection is not always helpful in trying to understand complex conditions such as autism. The concept of attachment, for instance, has gained renewed interest in recent years. A belief that attachment is an important, if not vital, factor in child development does not mean that one has to return to Bettelheim's ideas about refrigerator mothers, but it does suggest that those interested in child mental health should consider the role of parenting more carefully. One recent development, for instance, has been the attempt to differentiate autism from reactive attachment disorder, which occurs when children do not develop healthy attachments with their parents and caregivers (Davidson et al, 2015). As with mental illness more generally, more nuance and sophistication in our understanding tends to be the best approach.

One of the factors that Kanner noted in his 1943 paper was that children with autism tended to have dietary problems early on in life. As Silverman's work has demonstrated, many parents – and some

researchers – believe that diet plays a key role in autism, if not causing the disorder, then at least exacerbating its symptoms. Given the long association between food allergy and mental health problems (see above) this is not particularly surprising. It is also not surprising that, as with dietary explanations for ADHD, dietary explanations and treatments for autism have been controversial. Groups such as Defeat Autism Now!, founded by autism pioneer Bernard Rimland, have long suggested that parents pay attention not only to gluten, casein, and common food allergens but also to foods or food components thought to interfere with gut health, such as yeast, as well as sugar and other carbohydrates (Silverman, 2012). Such groups also advocate nutritional supplements and, most controversially, chelation therapy to detoxify the body. Although medical evidence for such measures is only now beginning to emerge and remains contentious, many parents were nonetheless convinced by the accretion of anecdotal evidence from others who found that they were helpful in reducing symptoms.

Most controversial by far, however, has been the MMR (measles, mumps and rubella) theory of autism, developed by (former) British gastroenterologist, Andrew Wakefield, during the 1990s. In 1998, Wakefield and his colleagues published a paper in the prestigious *Lancet* medical journal claiming that the MMR vaccine was responsible for the increase in rates of autism. Using the retrospectroscope, it is clear that there were startling and striking flaws in the original study by Wakefield and his co-authors. One significant concern is the tiny sample (just 12 children) used by Wakefield et al to make such extraordinary claims. The reputation of the *Lancet*, one of the world's leading medical journals, was undoubtedly damaged as a result, whilst the original 1998 paper was retracted in 2004 due to the fact that Wakefield et al's claims that the children had been "consecutively referred" and that their investigations were "approved" by the local ethics committee "have been proven to be false" (*Lancet*, 2010))

As mentioned earlier in connection with ADHD, researchers have attempted to discover genetic causation for autism or the autisms. The type of techniques used include "microarray comparative genomic hybridisation

(CGH), whole exome and whole genome sequencing.” (SIGN 145, 2016). Whilst no genetic causation for autism alone has been found there are several known genetic conditions which are associated with increased risk of ‘co-morbid’ autism such as Down’s Syndrome (REF SIGN 145, 2016). Thus genetic screening for cases of diagnosed autism is always something to consider in the light of a patient’s presentation. This will help parents, for example, in terms of genetic counselling re consideration of having further children. Researchers are often interested in genetics screening of diagnosed cases of autism as well as other non-autistic family members, especially siblings, identical and non-identical twins (to search for miniscule genetic variations).

### **Treatment Debates**

ADHD is a disorder that is almost synonymous with its most conventional treatment: stimulant drugs. And although many ADHD drugs have emerged over the years, the most famous (or infamous) remains the first one to be approved, namely Ritalin (methylphenidate), which was approved for use in children in 1962 by the FDA. It is important to note that, while Ritalin was made famous in its association with ADHD, it was originally marketed in the early 1950s to treat fatigue, lethargy, and depression originally in institutionalized patients and then in older patients. Having said this, it is possible to trace the history of prescribing stimulant drugs to treat childhood misbehaviour and learning problems back to 1937, when Charles Bradley, a psychiatrist at Emma Pendleton Bradley Home in Rhode Island (the same institution where hyperkinetic impulse disorder was coined in 1957) serendipitously discovered that stimulant drugs had an impact on the academic achievement of children under his care.

Bradley had prescribed Benzedrine, a stimulant, for patients who had undergone pneumoencephalography, to aid in their recovery from this painful neural imaging technique. Although the drug did not help his patients with the nausea and headaches that followed this procedure, Bradley found that it appeared to help improve academic performance and behaviour in such patients. But, while Bradley published his findings in leading journals in 1937 and 1950, they had little impact on the

medical community. It took the emergence of hyperactivity as a major childhood psychiatric disorder in the late 1950s for physicians to turn to stimulant drugs as a treatment option.

Ritalin, along with subsequent ADHD drugs, quickly became popular, but also controversial. While articles in *People* and *Life* magazines heralded the changes such drugs could have on hyperactive children, the drug did not work for approximately 20% of patients and, as recent research has suggested, the evidence supporting Ritalin's efficacy might not be particularly strong (Storebø et al, 2015). Others questioned the safety of such drugs, and whether it was ethical to prescribe powerful stimulants to young children that put them on a path to drug dependency. As an amphetamine, Ritalin and other ADHD drugs were readily associated with illicit amphetamines. As with earlier prescribed amphetamines, it did not take long before it was being sold illegally as a street drug, a practice that continues today in many schools and colleges (Iverson, 2008; Schwartz, 2016).

Concern also emerged about the side effects associated with ADHD drugs. Among them included insomnia, depression, anorexia (and associated stunted growth), bed-wetting, irritability, stomach aches, facial tics, fatigue, cardiovascular problems, and hallucinations. Rather than considering alternative treatments, however, most physicians advised that children could go on "drug holidays," when children were away from school or prescribed additional drugs (ranging from antidepressants to growth hormones) to counteract the side effects (Tec, 1971, 1424). One psychiatrist also advised that caffeine be prescribed instead, since it had fewer side effects (Schnackenberg, 1973).

Questions that need to be asked about the side effect profile of stimulants used for ADHD often go un-investigated due to the fact that much of the research into this field is funded directly or indirectly by the pharmacological industry. Many researchers and clinicians involved in such research are remunerated by the same industry and ethical regulations in any self-respecting peer-reviewed medical journal require such potential conflicting interests to be declared (which sometimes are not). For

example, whilst there have been some studies looking at problems physical growth in children using stimulants, there has been no study which the authors can find looking at whether use of stimulants affects actual brain growth. This might seem an obvious area of enquiry and its absence is somewhat concerning.

Given the known side effects of ADHD drugs and the presence of alternative explanations, it is not surprising that numerous treatment alternatives have been proposed as well. Alongside elimination diets, such as the Feingold diet, diets that add nutrients that are thought to be lacking, such as omega-3 fatty acids and vitamin D have been prescribed for ADHD (Richardson, 2009; Kamal, Bener, & Ehlayel, 2014). Educational interventions, such as the one portrayed in the Danish film *Four Letters Apart: Children in the Age of ADHD* have also been explored, often to great effect. Cognitive behavioural therapy, neurofeedback, physical activity, and various forms of mind-body therapy have all been suggested as alternatives. Most of these treatments are more holistic in nature, in that they (for instance, physical activity or dietary therapy) tend to benefit the patient in ways that extend beyond improving ADHD symptoms. Unfortunately, however, the evidence base for these alternatives has yet to be developed sufficiently, especially in the face of the thousands of studies done on medication. Arguably, with ADHD, the treatment that is offered first (medication) is the alternative that should be provided only after all others have failed. As both historical evidence and clinical experience demonstrate, dozens of factors may cause, trigger, or exacerbate behavioural problems in children. If we really care about the children diagnosed with ADHD, we would reverse this situation, offering drugs only as a last resort. National organisations responsible for clinical prescribing practice and thereby, prescribing rates, have attempted to control rates of stimulant prescription in children (McClure, 2013). However, these rates continue to rise worldwide (Smith, 2017).

Also, ADHD is increasingly managed as a lifelong condition. It is not unusual practice for a clinician in the USA, UK or other industrialised nations, or indeed now in other countries such as Iceland or India, for a child to be

diagnosed with ADHD in his primary school years and told that he may have to continue taking stimulant medication for the rest of his life. This has significant implications for that child's sense of self, apart from anything else, and, in busy psychiatric out-patient clinics, reflective time is often sacrificed for patient throughput. The other kinds of medical conditions in which children are told they may need to take medication for life are epilepsy, diabetes, cancer and if someone has had an organ transplant. All of these other conditions are almost inevitably life-threatening if the medication is not taken. Are we really saying the same about ADHD? Surely not.

## **Autism**

Autism is very different to ADHD in terms of treatment debates. Fortunately, or not (depending on one's perspective) there is no 'magic bullet' drug which has been found for autism. It is undoubtedly the case that researchers, funded in part by the pharmaceutical industry, are hard at work trying to find a drug or drugs which will specifically reduce the core symptoms of autism - difficulties with reciprocal social interaction and communication, as well as repetitive, stereotypical, routines and behaviours and associated sensory symptoms. However, there is no evidence that any such drug has been found or will be found in the foreseeable future. Instead, other drugs, used for other conditions, are prescribed to try to reduce some of the more problematic symptoms and behaviours associated with autism, such as ADHD symptoms (stimulants), anxiety and depression (SSRI antidepressants), behaviour which challenges (antipsychotics) and sleep difficulty (melatonin). National guideline organisations such as SIGN in Scotland, and NICE in England and Wales, have produced detailed guidelines to advise clinicians of the evidence base for, and approach to prescription for, such medications (REF SIGN 145 and NICE ASD guidelines).

Beyond this, one of the purposes of such clinical guideline production has been to attempt to find evidence to support the myriad non-pharmacological interventions which have arisen for autism over the last 50 years. For example, Applied Behaviour Analysis, a psychological intervention, has gained enthusiastic following in some and equally hostile response from others. The same applies to many

other interventions and the debates about them continue. Clinical guidelines can help mainstream clinicians in terms of what to advise parents and people with autism, but, when the subjects of such interventions are complex human beings, who are all individually different, scientific evaluation of the efficacy of such behavioural approaches is extremely challenging, in terms of establishing adequate scientific validity and reliability.

It is perhaps easier to be confident about other interventions which have been shown to be just plain dangerous, such as chelation and secretin therapies (both of which can cause significant harm to very vulnerable patients) or highly suspect, such as facilitated communication (SIGN 145). But some other therapies are something of a more grey area e.g. dietary manipulation (such as gluten and casein free diets). What is certain is that, with increasing pressures of information overload, with the difficulties of establishing scientific validity and reliability in small research studies to demonstrate efficacy, interventions in autism are going to continue to be a controversial area for the foreseeable future.

But, even if an autism gene was discovered or a 'cure' developed, what would be the implications of 'eradicating' autism, as if it were polio or smallpox.? A recent BBC documentary by naturalist and broadcaster Chris Packham highlighted this conundrum. After meeting American researchers and clinicians who wanted to eliminate the condition, Packham, who was diagnosed with Asperger's Syndrome as an adult, expressed considerable concern at what such an approach said about him and others who see and experience the world differently. Who would he be without his autistic characteristics, both those that make his life difficult and those that have helped him to be a success? Reflecting such views, the neurodiversity movement has emerged in recent decades to argue that, rather than finding cures for autism, it is more humane (and likely more cost-effective) to work with people diagnosed with autism and other neurodevelopmental conditions to help them adapt to an often inflexible and unforgiving world, and – equally – to make society more accommodating to people with such conditions. Although this argument tends to be made with high-functioning and successful individuals, such as Packham, in mind, it is even more relevant to those who struggle even more to fit

into society and to be understood. Moreover, it is one that can be successfully made with respect to many psychiatric conditions. If we accept that all mental health conditions are mediated in at least some way by the societal context in which they exist, why are we reluctant to change society in ways that help people to adapt, particularly when ‘curing’ the individual increasingly seems to be an elusive and, possibly, inhumane hope?

## Conclusion

In this chapter we have highlighted the two conditions of autism and ADHD as examples of the wider phenomenon of what are termed neurodevelopmental conditions. Even considering these two conditions, it will be evident that, whilst overlaps between them exist, they are very different across all aspects, such as presentation, assessment, and management. They have commonalities as well, in the sense that they have galvanised debate about what is and what is not a mental disorder, what it means to be human in modern society and what rights does a person have to be who they are, not what society wants them to be? One thing is for certain, these debates are going to increase both in range and complexity, as more and more neurodevelopmental conditions in humans are ‘discovered’ and described (as they surely will be).

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