

**Suffer the Restless Children:
The Evolution of ADHD and Pediatric Stimulant Use, 1900-1980**

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Abstract

This article traces the historical evolution of Attention Deficit/Hyperactivity Disorder (ADHD) and the controversial use of stimulants as a treatment for children diagnosed with the disorder in North America. While the children in question have exhibited similar behavior over the last century, the diagnostic labels used to identify them have changed due largely to cultural, medical and scientific changes and discoveries. For decades, children's use of psychotropic drugs was sufficiently controversial that pharmaceutical companies would not finance research in the area. The only substantial source of research funding for pediatric psychopharmacology in the U.S. from the 1950s to the 1970s was the National Institute of Mental Health (NIMH). In 1970, the first in a long-running series of controversies erupted over children's use of stimulants.

Key words: ADHD, amphetamines, history, pediatric psychopharmacology, Ritalin

In the 1950's, educators learned about [the] . . . psychopharmacological aspect of behavior modification, and began to encourage parents to seek such help from the child's physician. Soon it became evident that these drugs were being used indiscriminately—prescription would depend mostly upon a description of behavior by a teacher or parent. There was little awareness or use of the supporting information required to differentiate the hyperkinetic impulse disorder from other types of behavior disorders in which overactivity was also a predominant feature.

Eric Denhoff

Journal of Learning Disabilities (1971)

To those who have seen the results of such treatment [stimulants] in minimal brain dysfunction children, many of whom had failed to improve or had worsened with traditional therapies, the present limited use of drug therapy is as upsetting as it is unbelievable. . . It would not be hard to argue that in many instances psychotherapy of children with this syndrome virtually constitutes malpractice—a harmful withholding of useful treatment from a child.

Paul Wender

Minimal Brain Dysfunction in Children (1971)

This article examines the conceptual history of ADHD by highlighting how the array of behavioral problems that today constitute ADHD—academic difficulties, extreme restlessness, hyperactivity, inattention, and so on—came within the realm of medicine. If the modern observer finds the debate over children's use of stimulants to treat ADHD contentious and convoluted, they need only look at the history of the diagnosis and the drugs to understand why. Very hyperactive, restless, and inattentive children have been identified by clinicians and medical researchers dating back to at least 1902. Since then, upwards of 20 different diagnostic labels have been used to categorize children who exhibit these problematic behaviors.

What is striking about the numerous terms used to describe these children is the fact that they have outlined essentially the same behavioral symptoms that were first outlined back in 1902. And what is perhaps equally striking is that while these children have remained similar in terms of their description decade after decade, under different diagnostic labels, the explanations offered for their condition have varied dramatically. From the 1900s to the 1970s, explanations for the disorder's underlying cause changed frequently based on shifting cultural attitudes, medical developments, and scientific progress. Even when researchers from different eras reached similar conclusions that the disorder was biological in origin and the result of some damage to (or a deficiency in) a child's central nervous system, they often arrived at very different explanations for both the source of this damage or deficiency and how the disorder's biological basis operated on a child.

Another aspect of the controversy, and the one that principally drives it, has to do with children using psychotropic drugs. The discovery of the effectiveness of stimulants on hyperactive children was—like most psychiatric drug discoveries—accidental. It came in 1937, more than three decades after the first clinical description of inordinately hyperactive and inattentive children. Another three decades would elapse before the federal government made the first ever research grant to study the therapeutic effects of stimulants on children. During this period, most of the very few children in the U.S. who used psychiatric drugs were those with severe mental disorders and hospitalized for either short or long durations.

So controversial was the issue of children using psychotropic drugs that pharmaceutical companies would not finance research in the area, nor would they publicly recommend that children use these drugs. The only substantial source of research funding for pediatric psychopharmacology in the 1960s and 1970s was the National Institute of Mental Health

(NIMH) (Werry; 1978: 113). Three years after the NIMH awarded the first grant for studying children's use of stimulants in 1967, the first in a long-running series of controversies erupted over the ethics of children using these drugs. Not only has it never been fully resolved to anyone's satisfaction, the debate among clinicians, researchers and within the general public has grown in intensity while always revolving around the same basic issue: the appropriateness of millions of children using stimulant drugs.

This study stops its analysis in the year 1980, for this is the year in which the APA officially codified "Attention Deficit Disorder" (ADD), thereby beginning the contemporary nomenclature of this disorder. The publication of *DSM-III* and the official "birth" of ADD inserted a degree of psychiatric legitimacy into the discussion of childhood hyperactivity and impulsivity. Though controversies regarding ADHD—the "H" for hyperactivity was added by the APA as a possible subtype in 1987 by the APA with the publication of *DSM III-R*—and stimulant treatment currently persist, the year 1980 represented a turning point in which ADD became institutionalized in North American psychiatric practice. It is important to note that the APA's nomenclature varies significantly from its European counterparts who use a much more strict definition for childhood hyperactivity, adopting not *DSM-IV* criteria but the International Classification of Diseases-10 (ICD-10) system of diagnosis. Though influenced in part by European medical discussions, the conceptual history of ADHD (not its European equivalent) speaks to North American psychiatric practice. This practice is particularly visible in North America, not just because of the high incidence of children taking stimulant medication, but also, due to increased diagnoses of so-called "Adult ADHD" in the United States and Canada.

A “Defect of Moral Control,” Social Darwinism, and Sir George Fredrick Still

Placing childhood moral problems within the province of medicine began in 1902 with the clinical description of twenty “behaviorally disturbed” children by English pediatrician Sir George Frederick Still, who practiced medicine at King’s College Hospital in London. In a lecture series he gave before the Royal College of Physicians in March 1902, Still observed that the children he studied were of normal intellect, but ‘exhibited violent outbursts, wanton mischievousness, destructiveness and a lack of responsiveness to punishment’ (Still; 1902: 1009). They were often restless and fidgety with a ‘quite abnormal incapacity for sustained attention, causing school failure even in the absence of intellectual retardation’ (Still; 1902: 1009).

Unable to sit still, the children proved easily distractible, inattentive and, according to Still, unable to focus for long on any one thing (Still; 1902: 1166). He went on to state that “this pattern occurred more often in boys than in girls, became frequently apparent by early school years, was sometimes accompanied by peculiarities of physical appearance, generally showed little relationship to the child’s training and home environment, and commonly shared a poor prognosis (Sandberg and Barton; 1996: 5). The resemblance of these children to modern day boys and girls diagnosed with ADHD has been used both as a reference point for discussing the medical history of the disorder and as fodder for proponents on either side of the ADHD debate.

Still’s work contributed to a new track of medical discourse, which argued that a lack of morality was not necessarily an individual shortcoming but a matter of biology. As Still claimed, these children demonstrated a ‘defect of moral control’ (Still; 1902: 1008) that superseded the individual will. Hence, these children “suffered” from immorality. Still argued that these children were a new medical discovery who were too intelligent to be considered “idiots” (defined medically as “extreme stupidity”) and too young to be viewed as “criminal

minds” (Rafalovich; 2001: 105-6). Such children’s neurological defects were shown through ‘a severe lack of reserve signaled by persistent self-gratification, shamelessness, immodesty, and passionateness’ (Lakoff; 2000: 149-50). Still’s central hypothesis was that this moral deficit represented ‘the manifestation of some morbid *physical* condition’ (Still; 1902: 1165). Reflecting Darwinian sentiments, he hypothesized that moral ineptitude showed ‘a special liability to loss or failure in development [that is] quite in accordance with the phenomenon of evolution’ (Still; 1902: 1165).

This marks the beginning of the child with ADHD as object of science, or the point at which the discourse of medicine began to compete with the conventional perspectives that separated morality from any type of medical concern. The interplay of ideas within medicine with regard to these children was in its infancy. Despite Still’s arguments that childhood immorality could be explained through physiological processes, there was no empirical evidence produced to this effect. The work of Alfred Tredgold began a more sophisticated neurological discussion of childhood immorality and demonstrates some of the earliest attempts at making the ADHD child empirically visible.

Alfred Tredgold, “Feeble-Mindedness,” and Influenza 1918

Contributing further to the discourse that behavior patterns found in morally defective children stemmed from physiology, not from character flaws or lack of discipline, Alfred F. Tredgold suggested that some form of mild brain damage had occurred—probably during birth—that went undetected until the formal demands of early education exposed it (Tredgold; 1922). Widely considered Britain’s leading expert on mental impairment, Tredgold was a senior

member of the English Royal Commission on Mental Deficiency. ‘After the passing of the Education Act of 1876, making attendance at public elementary or other schools compulsory,’ he wrote, ‘it gradually became apparent that a group of children existed who were so far mentally defective that they could not be satisfactorily taught in the ordinary public schools, but who were not sufficiently defective to be certified as imbeciles or idiots under the Idiots Act of 1886’ (Tredgold; 1922: 174). Extending Still’s analysis, Tredgold’s arguments for the existence of a physical mental deficiency described failure within the institution of education—a social environment that is the most common realm where today’s cases of ADHD are suspected—as a “symptom” rather than a character flaw. Similar to Still, Tredgold observed that these children were not overtly ‘mentally defective,’ but exhibited markedly anti-school behavior. Tredgold continued Still’s argument that childhood immorality was a medical problem by suggesting that these moral shortcomings were most visible when children were subjected to the demands of school—an institution that was increasingly seen as crucial to a child’s moral development.

These medicalizing discourses were bolstered by the Mental Deficiency Act of 1913—the outcome of the Royal Commission’s work that ‘intended to embrace all grades of defect’ (Tredgold; 1922: 176). Heavily influenced by Tredgold’s work, the act applied the term ‘feeble-mindedness’ to the ‘mildest grade’ of mental deficiency, and placed the feeble-minded into two crude classes: adults and children (Tredgold; 1922: 176). Feeble-mindedness was not an especially precise medical terminology. As Tredgold wrote: ‘Mentally defective or feeble-minded children differ greatly in the degree of their deficiency. The lower members of the class closely approximate to, and cannot be distinctly separated from, the imbeciles. The higher members, on the other hand, are but little removed from the merely dull and backward of the normal population’ (Tredgold; 1922: 181). Supplementing his notion that mild brain damage

might be the culprit for childhood feeble-mindedness, Tredgold also noted that a number of such children exhibited a variety of slight physical ‘anomalies,’ or ‘the so-called stigmata of degeneracy,’ which included abnormal head shape and size (about half an inch less than that of a normal child), poor coordination, and ‘abnormalities of the palate’ (Tredgold; 1922: 181-2).

Like Still, Tredgold’s celebrity is largely based on the fact that the children he described in the early twentieth century bore a striking resemblance to modern day children diagnosed with ADHD. He concurred with Still that immorality was essentially a form of mental deficiency caused by some ‘organic abnormality on the higher levels of the brain,’ and argued that the areas of the brain where the sense of morality was located were also the product of the more recent development in the course of human evolution and, thus, were more susceptible to damage (Sandberg and Barton; 1996: 7). Further implicating physiology, Tredgold viewed environmental circumstances as not the cause, but the product of the condition’s ‘pronounced morbid inheritance’ (Tredgold; 1922: 184).

For Tredgold, such ‘pronounced morbid inheritance’ was visible through a profound inattentiveness:

Attention.—The most trifling thing serves to distract these children from their occupation, so that even where the attention is readily gained, it is with difficulty held. Many of them become capable of pursuing a congenial task with a certain amount of patience, but the majority have neither sufficient power of concentration or will to be capable of sustained mental effort against inclination or interposed obstacles. . . School-teachers often complain of the lack of *memory* of these children. . .

Control is very feebly developed in these children, and action is always along the line of least resistance. Volition is by no means absent, but their behaviour is more often the result of sudden desires and impulses than of deliberate purpose (Tredgold; 1922: 184).

It is important to note that Tredgold separated himself from social Darwinism, as he did not consider feeble-minded children to be disproportionately concentrated among the lower classes. Instead, they existed throughout society: ‘With regard to the social status of these children there is little to be said. The labouring classes have no monopoly of mental defect, and, although I am unable to give any actual figures, my general impression is that it is just as prevalent amongst the upper as the lower classes of this country’ (Tredgold; 1922: 181). Given that Tredgold firmly believed in a link between some form of brain damage and ‘feeble-mindedness,’ anything that could cause brain damage on a wide scale would result in a vastly increased incidence of mental defectiveness in all social classes and geographic environments (Tredgold; 1922: 180). The later discussion of the pandemic of *encephalitis lethargica* (e.g. “sleepy sickness”) was one of the key ways in which would-be ADHD symptoms could occur despite one’s lifestyle or class position.

Beginning in 1922, the same year of the fourth edition of his influential book, *Mental Deficiency (Amentia)*, Tredgold and a growing number of medical researchers detailed the negative neurological and behavioral effects found in children who had survived a bout with *encephalitis lethargica* (Ebaugh; 1923: 89-97; Hohman; 1922: 372-5; Strecker and Ebaugh; 1924: 443-53). The World War I epidemic killed upwards of 30 million people worldwide and infected roughly half of the earth’s human population (Barry; 2004).¹ An often fatal illness, encephalitis was ‘characterized by tremendous sluggishness, hallucinations, and fever, sometimes bringing with it periods of remission—something doctors viewed as a hopeful sign’ (Rafalovich; 2001: 107). After the influenza epidemic subsided, increasing numbers of clinicians began encountering children who had survived their infection but were exhibiting characteristics that would later be considered similar to ADHD. Clinicians described symptoms

of antisocial behavior, irritability, impulsiveness, severe emotional swings, and hyperactivity, but without significant cognitive damage (Strother; 1973: 8-9).

Although only a few of the child survivors of the epidemic would fit modern descriptions of children with ADHD, “postencephalitic behavior disorder” (as it became known) seemed to buttress the notion that there was a link between both severe brain damage and severe behavioral disturbances and, by extension, mild brain damage and mild behavioral disturbances. The latter were the “feeble-minded” and mentally defective (Sandberg and Barton; 1996: 8; Schachar; 1986: 24). Thus, the discussion of *encephalitis lethargica* was significant, not simply because it drew suspicion to the causal connection between behavior and neurological impulse, but because it medicalized unconventional behavior specific to children. Many of these symptoms would later be highlighted by neurologists and placed under the rubric of ADHD (Rafalovich; 2001).

“Organic Drivenness,” Amphetamines (Benzedrine), and Charles Bradley

Tredgold’s and others’ work in the area of *encephalitis lethargica* further crystallized the would-be ADHD child as medical object by linking so-called “minimal” brain damage to a disease process. Given the esteem of Tredgold and his colleagues, it is a matter of course that the medical community largely accepted the legitimacy of “postencephalitic behavior disorder” as the cause of a perceived rash of childhood misbehavior after World War I. Tredgold, and others’ discussion of discussion of idiocy, imbecility, and encephalitis, operated in a conceptually medicalizing capacity; that is, they raised the salience of childhood misbehavior in the medical community, but did not offer any specific strategy for treating it. This changed forever, though, with the work of Charles Bradley in 1937.

Three years before, in 1934, researchers Eugene Kahn and Louis Cohen identified a number of patients whose clinical condition was marked by an inability to remain quiet, abruptness, clumsiness and explosiveness of voluntary activity (Kahn and Cohen; 1934). In their *New England Journal of Medicine* article, they noted that all their patients' symptoms were secondary to a primary behavioral abnormality (hyperactivity), which they argued was a result of 'organic drivenness' or 'a surplus of inner impulsion' (Sandberg and Barton; 1996: 10; Schachar; 1986: 25). Unlike the post-encephalitic children or those described by Still and Tredgold, however, most of these new children described by Kahn & Cohen did not have a specific history of neurological trauma. To account for this, they suggested that a congenital defect in the brainstem organization that controlled activity level could be responsible for this 'organic drivenness' (Sandberg and Barton; 1996: 10; Schachar; 1986: 25). They also revived hints of social Darwinism as a potential causative factor, given 'that the over- as well as the under-development of certain brain areas serves as a sort of background of certain plus and minus members of the species' (Kahn and Cohen; 1934: 750). Essentially, Kahn & Cohen were pointing to the existence of superior and inferior human genetic types to buttress their belief in a biological—rather than an environmental—basis for hyperactivity.

Kahn & Cohen's work did not garner widespread attention when it was first published (it did later). But as fate would have it, "frequent reference to 'organic drivenness'" and Kahn & Cohen's article was made in 'a unique and curious setting known as the Bradley Home, which devoted itself to the emotional problems of children,' according to Maurice Laufer, a medical researcher who spent part of his residency there and who later went on to provide the first specific name—'hyperkinetic impulse disorder'—for the cluster of behavioral symptoms that characterized this 'organic drivenness' (Laufer; 1975: 105-6). The Home's wealthy donors, Mr.

and Mrs. George L. Bradley, had established it as a memorial to their only child who had suffered from the severe 1918 strain of influenza in her early childhood. It left her mentally retarded, cerebral-palsied, and epileptic (Laufer; 1975: 105-6). Her parents, with all their wealth, found no resources available for her. In their wills they expressed the hope that their home in Rhode Island and their estate might be used so that from their suffering ‘might come comfort and hope for many’ (Laufer; 1975: 106). Dr. Charles Bradley, a pediatrician, headed the Home’s staff of pediatricians and psychiatrists, most of whom were imported from local adult private mental hospitals and Yale University’s School of Medicine in New Haven, Connecticut (Laufer; 1975: 106).

In the early days of the Bradley Home, notes Laufer, it was widely believed that abnormalities of the structure of the central nervous system were probably responsible for the children’s difficult behavior and that neurosurgical remedies might be an effective treatment. Therefore, a procedure after the admission of some children to the Home was to perform a pneumoencephalogram (a painful spinal tap) upon them, which regularly resulted in complaints of severe headaches (Laufer; 1975: 108). In response, Bradley began treating these children with a new amphetamine, Benzedrine, which he hoped would stimulate the choroid plexus—located in the ventricular system of the brain—to produce spinal fluid and, thereby, reduce pressure on the children’s sinuses (American Journal of Psychiatry; 1998: 968).

Similar to the discovery of many new psychiatric drugs in the history of mental health, this one came entirely by accident (Elliott; 2004: 251-2). The children’s headaches were not particularly affected or relieved by the drug (Bradley; 1950: 25). But ‘possibly the most striking change in behavior during the week of Benzedrine therapy occurred in the school activities of many of these patients,’ reported Bradley in 1937 (Bradley; 1937: 577-8). The behavior and

school performance of many of the thirty children who received the drug underwent a dramatic change characterized by increased interest in school work, better work habits, and a significant reduction in disruptive behavior. The drug calmed many of the children without dulling their attention span (Bradley; 1937: 578-80). The effect was not limited to children with any particular behavioral disorder, but ranged from the child who had ‘specific educational disabilities, with secondary disturbed school behaviour, to the retiring schizoid child on the one hand and the aggressive, egocentric epileptic child on the other’ (Schachar; 1986: 26).

Bradley prescribed the drug not only to children suffering from severe post-pneumoencephalogram headaches but also—as a control—to children who had not recently undergone the spinal tap procedure. Thus, although Bradley’s experiment was not fully randomized, double-blind, and controlled, it was single-blind and controlled. As it turned out, Benzedrine had the same basic effect on both groups of children, who began to refer to the medication as their ‘arithmetic pills’ and would often spontaneously remark, ‘I have joy in my stomach,’ ‘I feel fine and can’t seem to do things fast enough today,’ and ‘I start to make my bed and before I know, it is done’ (Bradley; 1937: 579). One month before Bradley’s original 1937 article on his patients appeared, two other researchers similarly found ‘improvement in the performance on IQ tests of subjects who were given amphetamines [Benzedrine]’ (Motlitch and Sullivan; 1937: 519-22).

Bradley acknowledged that ‘it appears paradoxical that a drug known to be a stimulant should produce subdued behavior in half of the children.’ He noted, however, ‘that portions of the higher levels of the central nervous system have inhibition as their function, and that stimulation of these portions might indeed produce the clinical picture of reduced activity through increased voluntary control’ (Bradley; 1937: 582). Moreover, on the first day that

Benzedrine was discontinued, the effects of the drug disappeared and the children's behavior problems returned. 'To see a single daily dose of Benzedrine produce a greater improvement in school performance than the combined efforts of a capable staff working in a most favorable setting, would have been all but demoralizing to the teachers, had not the improvement been so gratifying from a practical viewpoint,' observed Bradley. Nevertheless, he concluded his article by cautioning that 'any indiscriminate use of Benzedrine to produce symptomatic relief might well mask reactions of etiological significance [other physical, psychological and/or educational factors that could be the underlying cause of the children's behavioral problems] which should in every case receive adequate attention' (Bradley; 1937: 582, 584).

What ultimately proved critical from Bradley's accidental findings was that the first experimentation with amphetamines opened two avenues of research: (1) the calming effect on children's behavior and activity, and (2) the stimulating effect on their academic performance (Grinspoon and Singer; 1973: 521).

Minimal Brain Damage, "Hyperkinetic Impulse Disorder," and the Advent of Ritalin

In the 1950s, the disorder and an effective treatment for it began to be paired up in a more formal and medically meaningful way. What later became known as ADHD was first officially named "hyperkinetic impulse disorder," by Maurice Laufer, Eric Denhoff and Gerald Solomons in 1957 (Laufer et al.; 1957: 38-49). The three researchers worked at the same Bradley Home where Benzedrine was first found to be helpful to children with severe behavioral disorders twenty years before. In their article, they 'acknowledged their indebtedness to Charles Bradley, M.D., whose pioneer observations provided the inspiration for these studies' (Laufer et al.; 1957:

38). As a new and specific diagnostic category, noted Laufer and his colleagues, ‘hyperkinetic impulse disorder’ is a behavior pattern that ‘may be noted from early infancy on or not become prominent until five or six years of age. . . Hyperactivity is the most striking item. . . There are also a short attention span and poor powers of concentration, which are particularly noticeable under school conditions. . . The child is impulsive . . . irritable and explosive, with a low frustration tolerance. Poor school work is frequently quite prominent’ (Laufer et al.; 1957: 38). The disorder was observed as ‘more frequent in males than in females’ and, peculiarly, more frequent ‘in first-born than in subsequent children’ (Laufer and Denhoff; 1957: 464).

Laufer and his colleagues suggested that ‘hyperkinetic impulse disorder’ was the result of an ‘injury to or dysfunction of the diencephalon in early life’ (Laufer et al.; 1957: 45-6). The diencephalon, they explained, ‘is a small part of the brain that acts to sort, route and pattern impulses coming from sensory receptors before they become amplified at higher levels of the brain. In this capacity it functions as an inhibitor of irrelevant stimuli, keeping them from ‘flooding’ the cortex. If the diencephalon is not functioning properly, the cortex can become overwhelmed by more stimuli than it can adequately deal with’ (Grinspoon and Singer; 1973: 534). The result, noted Laufer and Denhoff in a separate article, ‘is an undue sensitivity of the central nervous system to stimuli constantly pouring in from peripheral receptors. . . The resulting components of sensitivity and forced responsiveness to stimuli, inability to inhibit and delay responses, and visual-motor difficulties—all combine to produce the characteristics of the hyperkinetic syndrome’ (Laufer and Denhoff; 1957: 467).

Yet Laufer acknowledged decades later that he and his colleagues had ‘a nagging concern over the fact that so many children who presented the picture that we were coming to recognize and that we later characterized as ‘hyperkinetic impulse disorder’ presented no clear diagnostic

evidence of involvement of the central nervous system and had nothing in their history that would provide an acceptable etiological statement' (Laufer; 1975: 113). In short, many of the children they diagnosed with 'hyperkinetic impulse disorder' did not have any evidence of brain injury, nor was there an obvious biological or physical explanation for their behavior (e.g., retardation).

Regardless of what specifically caused 'hyperkinetic impulse disorder,' Laufer and his colleagues confirmed that amphetamines were effective in counteracting its symptoms (Laufer et al.; 1957: 47; Laufer and Denhoff; 1957: 465-6). They suggested 'that amphetamine may in some way, perhaps by raising the level of synaptic resistance, alter the functions of the diencephalon in such a way that it once more can keep the cortex from being flooded by streams of unmodulated impulses coming in through sensory receptors' (Laufer et al.; 1957: 47; Laufer and Denhoff; 1957: 465-6). What is often overlooked in other historical accounts of Laufer and his colleagues' work is how they subordinated the importance of stimulant medications to psychotherapy and the Freudian views that dominated psychiatry at the time. Their articles are littered with references to 'associated ego disturbances' and 'ego weaknesses,' mothering issues, Freud, neurosis and neurotic, and 'the present permissive era of child management' (Laufer et al.; 1957: 45-8; Laufer and Denhoff; 1957: 470). In the first article they published in 1957, Laufer and his colleagues wrote: 'In other words the amphetamine did not interfere with the operation of the psychotherapeutic process and *the fostering of a basic inner change*' (Laufer et al.; 1957: 44, italics added). They concluded their second article with the observation that 'it would be unfortunate if, as a result of these observations, amphetamine were used indiscriminately for the treatment of behavior disturbance in children or if the need for specific

psychotherapy were overlooked. Amphetamine has a specific role, *but is no substitute for psychotherapy*' (Laufer and Denhoff; 1957: 474).

Laufer and his colleagues' work on 'hyperkinetic impulse disorder' represented one of the very few research initiatives in the U.S. at the time on children's use of psychiatric drugs. Less than a dozen clinical research papers were published between 1937 and the early 1950s on the use of stimulant drugs by children (Safer; 1971: 491). The general consensus among clinicians and medical researchers at the time, noted the NIMH's first Director Robert Felix, was that the use of psychopharmacological drugs in children may be 'tools of tremendous value but also may contain elements of danger' (Fisher; 1958: vii).

At the same time, in the mid-1950s new psychiatric drugs became available for treating adults with severe mental illness and disorders. The whole field of psychopharmacology entered a phase of rapid acceleration with experiments that showed the effectiveness of tranquilizers for adult psychiatric patients and antidepressants for severe mood problems (Ross and Ross; 1976: 18-9). These developments led to a resurgence of interest in the use of drugs for hyperactive and emotionally disturbed children. A number of articles appeared in 1955 and 1956 that pointed to the effectiveness of chlorpromazine—a powerful and revolutionary psychiatric medication also known by its trade name Thorazine (or, Largactil, as it was known in Europe)—in treating hyperactive children (Freed and Peifer; 1956: 22-6; Freedman et al.; 1955: 479-86). "At this point the question may justly be asked, 'Why use such a potent drug on these children instead of psychotherapy?'" wrote Herbert Freed and Charles Peifer in the *American Journal of Psychiatry* in 1956. 'Fundamentally, the chief reason was the need to improve a situation, such as individual misbehavior in a school room where the authorities could use only limited controls in dealing with the student' (Freed and Peifer; 1956: 22). Besides, the authors noted, 'Twenty of

these children (80%) were either illegitimate or came from broken homes. Psychotherapy with the remaining parent was therefore, for economic or other reasons, impossible' (Freed and Peifer; 1956: 22).

Yet chlorpromazine was a powerful psychiatric drug with some especially nasty side effects (Lehman; 1989: 263-5; Lehman and Hanrahan; 1954: 227-37; Swazey; 1974).² Its primary and most effective use was on adults in mental asylums suffering from psychosis (Bower; 1954: 689-92; Shorter; 1997: 246-55). Thus, it did not become a serious competitor against amphetamines, such as Benzedrine and Dexedrine (which had their own unpleasant side effects), for use by inordinately hyperactive and restless children. A Swiss pharmaceutical firm, J. R. Geigy, had been working since the 1940s to develop a drug that would reproduce the same stimulant effects as amphetamines, but with fewer side effects and with less potential for abuse. In 1955, just as chlorpromazine was transforming the treatment of severely psychotic mental patients in many state mental asylums, Geigy synthesized a drug called methylphenidate (Ferguson et al.; 1956: 1303-4; Ferguson; 1957: 1479-80; Wax; 1997: 203-9). Given the brand name Ritalin, it was first mentioned as a possible treatment for children with 'hyperkinetic behavior syndrome' in Laufer and Denhoff's 1957 article in the *Journal of Pediatrics* (Laufer and Denhoff; 1957: 473). The authors noted, however, that their experience with Ritalin and a handful of other drugs was 'too limited for any valid statement as to their usefulness' (Laufer and Denhoff; 1957: 473).

As part of an effort to build on the success of chlorpromazine and to aid in the development of new psychiatric drugs, such as imipramine, the NIMH created the Psychopharmacological Research Branch (PRB) in 1956 (Lipmann; 1974: 2-3,13). Two years later the PRB sponsored the first ever conference 'on the use of drugs in children with psychiatric problems'

(Fisher; 1958: vii-viii). One of the leading voices at the conference was a young psychiatrist at Johns Hopkins University's School of Medicine by the name of Leon Eisenberg. The PRB's conference on children's use of psychiatric drugs coincided with the federal government's first ever research grant on child psychopharmacology in 1958, which went to Eisenberg. The grant was for studying the use of tranquilizers on children, however, and did not include plans for the investigation of stimulants (Swanson et al.; 1995: 286). Eisenberg recruited a young instructor in pediatrics and medical psychology by the name of Keith Conners, who together initiated a series of ground-breaking studies in pediatric psychopharmacology (Fisher; 1958: xi). Conners, a former Rhodes Scholar who earned his doctoral degree with highest honors at Harvard University, and Eisenberg, who earned his M.D. at the University of Pennsylvania, interned at Mount Sinai Hospital in New York City, and served as a Captain in the Army Medical Corps, brought a new level of scientific rigor and biomedical respectability to the study of using psychiatric medications on children.

Ritalin was eventually approved by the Food and Drug Administration in 1961 for use in children with behavioral problems (Swanson et al.; 1995: 270-1). In 1963, Conners & Eisenberg published the first article in a medical journal explicitly advocating the use of Ritalin for treatment of 'disturbed' children (Conners and Eisenberg; 1963: 458-64). In their study, they included 81 children who came from 'two residential care institutions,' one of which was a foster home for children 'unsuitable for care in individual foster homes,' and the other was a psychiatric treatment center (Conners and Eisenberg; 1963: 458). Children who took Ritalin showed demonstrably and statistically significant improvement following their use of the drug (Conners and Eisenberg; 1963: 458). Conners noted many years later that he was 'struck by the size of the improvement effect in the children who received stimulants,' because his 'earlier

experience treating similarly disturbed children with psychotherapy yielded virtually no improvement even after a year's worth of psychotherapy.³ Key to Conners & Eisenberg's research was the fact that parents and child care workers—rather than physicians—rated the children's symptoms before and after treatment—and, secondly, that the study was both double-blind (nobody knew which children received what treatment) and controlled (half of the children received a placebo) (Conners and Eisenberg; 1963: 462). 'The only adverse side effect,' noted Conners & Eisenberg, 'was a high report (70%) of appetite loss in the drug group' (Conners and Eisenberg; 1963: 462).

With today's strict and demanding scientific requirements for "informed consent" by human research participants and other safety procedures, it is interesting to note how relaxed the research environment was back in the 1960s. "I would walk down the street in Baltimore to School 102, which served children with conduct disorders," recalls Conners, "and I'd tell the principal about the research I was interested in doing: using psychopharmacology to try to help the kinds of students he had. The principal responded, 'If you could help us, that would be great!' The parents of the children were even more enthusiastic than the principal," he adds. "I was able to conduct a safe, rigorous, double-blind, cross-over study within ten days!"⁴

The composite picture of hyperactivity that emerged in the early 1960s, then, was that 'of a brain damage syndrome to be treated with stimulant drugs, minimal stimulation classrooms, and possibly psychotherapy, and having a favorable prognosis for the adolescent years' (Ross and Ross; 1976: 19).

Minimal Brain Dysfunction, Stimulant Politics, and the Critical Role of the NIMH

By the mid 1960s, however, growing numbers of researchers were openly questioning the assumption that there was a definitive link between some degree of brain damage and hyperactivity (Bax and MacKeith; 1963; Birch; 1964; Denhoff et al.; 1959; Herbert; 1964; Rapin; 1964). Many children who were diagnosed with ‘hyperkinetic impulse disorder’ showed no evidence of any brain damage (Chess; 1960).⁵ The term ‘became recognized as vague, overinclusive, of little or no prescriptive value, and without much neurological evidence,’ notes Russell Barkley (Barkley; 1990: 10). Consequently, the term ‘minimal brain damage’ died a slow death and morphed into ‘minimal brain *dysfunction*,’ which still stressed some deficiency in the child’s central nervous system but left vague what the underlying cause for this deficiency might be (Clements and Peters; 1962). The new term’s description of extremely hyperactive and inattentive children closely resembled many previous descriptions dating back to Still’s famous 1902 lecture:

The term “minimal brain dysfunction” [MBD] refers . . . to children of near average, average, or above average general intelligence with certain learning or behavioral disabilities ranging from mild to severe, which are associated with deviations of function of the central nervous system. These deviations may manifest themselves by various combinations of impairment in perception, conceptualization, language, memory, and control of attention, impulse, or motor functions. . . These aberrations may arise from genetic variations, biochemical irregularities, perinatal brain insults or other illness or injuries sustained during the years which are critical for the development and maturation of the central nervous system, *or from unknown causes* (Clements; 1966: 9-10, italics added).

The new term's hedging on what caused MBD partly reflected the reality that psychodynamic and Freudian-inspired psychotherapy still held sway within the field of psychiatry in the 1960s. Until Eisenberg replaced him in 1961, the Chief of Child Psychiatry at Johns Hopkins was Leo Kanner, one of the first academic child psychiatrists in America. While critical of psychoanalysis, Kanner espoused a "dynamic" approach to human personality that he learned from his profoundly influential mentor, Adolph Meyer. According to Kanner, origins of present troubles were reactions to experiences of the past; mental illness was essentially the behavioral product of personal difficulties and sufferings stretching back to early childhood (Freedman; 1992: 858-66; Wortis; 1986: 677-81; Shorter; 1997: 91-3,109-12). Thus, when the second edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM-II) appeared in 1968, all childhood disorders were described as 'reactions,' and 'hyperkinetic impulse disorder' or 'hyperactive child syndrome' became 'hyperkinetic reaction of childhood' (American Psychiatric Association; 1968: 50). Parents and teachers were urged, among other things, to try to reduce the amount of stimuli around children diagnosed with the disorder.⁶

As clinicians and researchers continued to debate the best diagnostic term for children with significant behavior disorders, the late 1960s marked the beginning of a turning point for research in pediatric psychopharmacology (Lipman; 1974: 204). It was initiated almost exclusively by financial support from the U.S. federal government's NIMH. The pharmaceutical industry would not finance research in this area, largely because administering psychoactive drugs to children—except in the most extreme and extraordinary situations—was considered unethical and potentially harmful (Ross and Ross; 1976: 116). Moreover, at the time, the drug companies did not envision that the pediatric market for psychiatric drugs would ever become

big enough to be worth investing in.⁷ They were focusing their efforts almost exclusively on the adult market.⁸

In 1967, the NIMH made the first federal government research grant to study the effectiveness of stimulants on behaviorally disturbed children to Keith Conners, who had left Johns Hopkins for Harvard Medical School and Massachusetts General Hospital (Swanson et al.; 1995: 286). The NIMH concluded that it ‘needed more quality studies comparing, in concurrent designs, the relative efficacy of drugs of different classes; stimulants, phenothiazines, antidepressants, and minor tranquilizers.’ It also wanted more long-term efficacy studies (e.g., longer than eight weeks and hopefully as long as a few years) (Lipman; 1974: 208-9). Consequently, the NIMH made three grants in 1968 to researchers at the University of California, Davis, Hillside Hospital in New York, and the District of Columbia’s Department of Mental Health to study the ‘comparative drug effects in hyperkinetic children’ and the ‘pharmacotherapy of hyperactive children’ (Lipman; 1974: 205).

By the close of the 1960s, only an estimated 150,000 to 200,000 children in the U.S. were being treated with stimulants,⁹ which represented roughly 0.002 percent of the entire childhood population.¹⁰ Nevertheless, an event occurred in the summer of 1970 that permanently altered the field of pediatric psychopharmacology. It also served as a harbinger of the future debates and controversy over stimulant use by children. On 29 June 1970, in an article entitled “Omaha Pupils Given ‘Behavior Drugs’,” the *Washington Post* reported that 5 to 10 percent of school children in Omaha, Nebraska, were receiving behavior-controlling drugs (Ritalin), ‘that this was part of a directed program by the school system in which some parental coercion to submit to drug therapy was involved, and that drugs were being given without adequate medical supervision resulting in pill swapping in school’ (Maynard; 1970: 1). The article had several

inaccuracies: the 5-10 percent figure referred only to the percentage of special-education children using stimulants, not the entire student population. And parents were not being coerced into accepting drug therapy (Medical News; 1970). Yet the story generated considerable media attention (Ladd; 1970: 66-8, 81-3; Hentoff; 1970: 31-3; Hentoff; 1972: 20-1), and led to a congressional hearing,¹¹ a national conference on the subject ('The Use of Stimulant Drugs in the Treatment of Behaviorally Disturbed Young School Children') (Psychopharmacological Bulletin; 1971: 23),¹² and an official Report of the Conference in 1971.¹³

The committee's hearing came amidst growing public concern over the abuse of all drugs, but particularly stimulants (Graham; 1972: 14-22). In 1970, Congress revised the nation's existing federal drug regulations with the passage of the Comprehensive Drug Abuse Prevention and Control Act. The Act placed amphetamines and methylphenidate (Ritalin) in the category of Schedule III, which put limits on the number of refills a patient could obtain and how long an individual prescription could run. The inconsistency in recommending the use of stimulants for one purpose—treating children with minimal brain dysfunction—while trying to prevent their use for illicit purposes (e.g., appetite suppression, performance enhancement in athletics, recreational use, etc.), was politically awkward (Swanson et al.; 1995: 288).

With growing amphetamine abuse in the U.S. and a documented epidemic of Ritalin abuse in Sweden (New England Journal of Medicine; 1970: 760-2). Congress instructed the Drug Enforcement Administration (DEA) in 1971 to reclassify amphetamines and methylphenidate as Schedule II drugs (along with opiates such as Demerol and morphine), which tightened prescription regulations and created a production quota or limit for all stimulants (Smith and Wesson; 1973: 18). This new classification had several important consequences. Henceforth, pharmaceutical companies had to make requests to the DEA for how much total amphetamines

and other stimulants they were permitted to manufacture. The DEA reviewed these requests and then set an aggregate amount based on what it considered to be “appropriate.” The DEA also began monitoring the distribution of these drugs nationwide, which has provided annual data on the amount of the drugs produced and where they have been distributed dating back to the early 1970s (Drug Enforcement Administration; 1995).

The negative media blitz and increasing public and congressional interest in hyperactive children and stimulants culminated in 1975 with three major publications that found large audiences in both the academy and the general public. The first, *The Myth of the Hyperactive Child and Other Means of Child Control*, by journalists Peter Schrag and Diane Divoky, argued that school children were being labeled with a dubious diagnosis and treated with unnecessary and dangerous medications, particularly Ritalin, by a conspiracy of authoritarian physicians, school administrators, and teachers (Schrag and Divoky; 1975). The book was a classic screed. Stimulants were being used as ‘chemical straitjackets,’ the authors added, to control the natural exuberance and activity of children who came into conflict with teachers or other school personnel. The book was criticized for various inaccuracies and a general tone of hysterical exaggeration, but it was widely read. A second book, Benjamin Feingold’s *Why Your Child is Hyperactive*, made an argument that has persisted off and on to this day, despite a continuing lack of evidence for it (Conners; 1980). Hyperactivity in children, Feingold maintained, was the result of an allergic or toxic reaction to food additives (especially colorings and dyes). Remove these items from a child’s diet, he claimed, and the hyperactivity would disappear (Feingold; 1975). Feingold’s arguments became so widespread that Feingold Associates, part political-part public interest group comprised mainly of parents, developed in virtually every state in the country (Barkley; 1990: 16).

Finally, sociologist Peter Conrad asserted in a now classic article, ‘The Discovery of Hyperkinesis,’ that the diagnosis was simply a new example of an old societal tendency to ‘medicalize’ problematic but otherwise normal behaviors. ‘As one of the most effective means of social control,’ Conrad argued, diagnosing hyperactive children with a mental disorder and medicating them with stimulants was akin to silencing political nonconformists and religious heretics (Conrad; 1975: 12-21). A major factor in the disorder’s growing popularity with teachers and clinicians, he maintained, was the profit-driven marketing efforts of the pharmaceutical industry (particularly Ciba-Geigy, the maker of Ritalin) (Conrad; 1975: 12-21). Conrad also singled out the Association for Children with Learning Disabilities for playing an influential lobbying role in expanding this ‘medicalization of deviant behavior’ to include ‘hyperkinetic’ children (Conrad; 1975: 17-18). Conrad’s alternative suggestion, that the children’s school or home environment might be the root cause of their deviant behavior, found a receptive audience among the antiauthoritarian spirit of the time and has remained popular among many interested observers to this day.¹⁴

Finally, a discovery in 1978 further complicated the relationship between the diagnosis and the drugs, and added to the controversy over both. For decades, a hyperactive child’s positive response to stimulants was often considered strong supporting evidence for the child having the mental disorder. Clinicians and researchers would often work backwards from a post-stimulant improvement in a child’s behavior to a confirmation of the diagnosis. However, Judith Rapoport—a researcher at the NIMH’s Biological Psychiatry Branch and whose work was one of the first to receive government funding back in the early 1960s—found that stimulants had similar effects on normal children as on excessively hyperactive children or children with other related behavior problems (Rapoport et al.; 1978: 560-5; Rapoport et al.; 1980: 933-43). After

giving Dexedrine and Dextroamphetamine to hyperactive and normal children, she found that both groups experienced similar improvements in attention and on math tests (Rapoport et al.; 1978: 560-5; Rapoport et al.; 1980: 933-43). If the vast majority of children's behavior and performance benefited from taking stimulants, then how hyperactive did a child have to be to qualify as having a genuine mental impairment? Rapoport and her colleagues' findings demonstrated the need for psychiatrists to develop more accurate and reliable diagnostic methods for distinguishing children who had the disorder from the majority who did not. As it turns out, larger efforts along these lines were already well underway (Mayes and Horowitz; 2005).

Conclusion

As the preceding analysis demonstrates, the historical evolution of the diagnoses (that we now refer to as ADHD) and the use of stimulants by children were profoundly affected by shifting social attitudes, political controversies, major episodes of disease and illness, and scientific developments and progress. From "social Darwinism," to eugenics, Influenza 1918, the rise of special education programs, Freud, and America's first "War on Drugs" in the 1970s, especially hyperactive and inattentive children have remained a population of enormous medical, parental and educational concern. There are many reasons for the convoluted development of both the disorder and stimulant drug treatment. There are also many reasons, mostly attitudinal, for the controversy that has grown up around them. But perhaps the key and irresolvable factor, argues Keith Conners, is that at the core of the disorder is a "black box": the child's brain.¹⁵ Only today are researchers beginning to be able to peer into it with new diagnostic imaging technology and incorporate genetic analyses. For virtually all of the twentieth century, however,

whatever was fundamentally amiss in a child's brain (if anything) had to be "taken on faith."¹⁶

And we all know how inherently controversial and divisive matters of faith can be, in the arena of mental health as much as in religion.

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Notes

¹ See also, *The American Experience, Influenza 1918*, PBS available at <http://www.pbs.org/wgbh/amex/influenza/> (last visited 16 July 2005).

² The most serious side effect associated with use of chlorpromazine was tardive dyskinesia, in which patients suffered from severe involuntary facial grimacing and embarrassing, uncontrollable bodily movements.

³ Conners telephone interview with the author.

⁴ Conners telephone interview with the author.

⁵ In Stella Chess' now famous report of 82 hyperactive children, only 14 were diagnosed as brain-damaged. For more on the lack of definitive evidence for a link between brain damage and hyperactivity, see M. Stewart, F. Pitts, A. Craig, W. Dieruf, "The Hyperactive Child Syndrome," *American Journal of Orthopsychiatry* 36 (October 1966):861-7.

⁶ Take, for example, Conners quoting Eisenberg in Conners, C. (1967). *The Syndrome of Minimal Brain Dysfunction: Psychological Aspects*. *Pediatric Clinics of North America* 14 (November), 761: "The child's parents, usually defeated, confused and angry by the time the physicians sees them, need to understand that his behavior is not malevolent and hostile but stems from a deficiency in neurophysiologic control mechanisms. If they can be helped to view his symptoms as a result of a treatable illness rather than as a personal attack on them, they will be the more able to apply the consistency of discipline, the firmness without anger and the anticipatory intervention that he requires. It is sound advice to suggest to parents that they avoid shopping trips, visits to restaurants, and attendance at large parties, for children are at their worst under such circumstances. . .

Similar guidance can be offered his teacher. For effective learning, class size should be small. The room should contain a minimum of distracting stimulation (i.e., materials not in use should be in cupboards out of sight). Class activities should be paced to his capacity for sustained participation,

with frequent periods of guided and supervised large muscle play. Indeed, with some children, freedom to leave the class and run about the playground has proved useful when motor tension mounts beyond control. (Clearly, this option can be made available only to the relatively mature and well motivated child.)”

⁷ Leon Eisenberg phone interview with the author, July 19, 2005.

⁸ Ibid.; Conners interview with the author.

⁹ Please consult the Hearing Before a Subcommittee of the Committee on Government Operations. (1970). Federal Involvement in the Use of Behavior Modification Drugs on Grammar School Children on the Right to Privacy Inquiry. House of Representatives, 91st Congress, 2nd Session (September 29), 16.

¹⁰ Please see, U.S. Bureau of the Census, Current Population Reports, Series P-25, No. 311, Estimates of the Population of the United States by Single Years of Age, Color, and Sex, 1900 to 1959, pages 22-23, 42-43. Series P-25 No. 519, Estimates of the Population of the United States, By Age, Sex, and Race: April 1, 1960 to July 1, 1973, Table 2. Series P-25, No. 917, Preliminary Estimates of the Population of the United States by Age, Sex, and Race: 1970 to 1981, Table 2.

¹¹ Hearing Before a Subcommittee of the Committee on Government Operations. (1970). Federal Involvement in the Use of Behavior Modification Drugs on Grammar School Children on the Right to Privacy Inquiry. House of Representatives, 91st Congress, 2nd Session (September 29), 16.

¹² Report on the Conference of the Use of Stimulant Drugs in the Treatment of Behaviorally Disturbed Young School Children. (1971). Psychopharmacological Bulletin 7, 23.

¹³ Report of the Conference on the Use of Stimulant Drugs in the Treatment of Behaviorally Disturbed Young Children, Sponsored by the Office of Child Development and the Office of the Assistant Secretary for Health and Scientific Affairs, Department of Health, Education, and Welfare,

Washington, D.C., January 11-12. (1971). Reprinted in *Journal of Learning Disabilities* 4 (November), 523-30.

¹⁴ The following quote from Conrad is very useful in this regard: “We tend to look for causes and solutions to complex social problems in the individual rather than in the social system. . . We then seek to change the “victim” rather than the society. . . Hyperkinesis serves as a good example. Both the school and the parents are concerned with the child’s behavior; the child is very difficult at home and disruptive in school. No punishments or rewards seem consistently to work in modifying the behavior; and both parents and school are at their wits’ end. A medical evaluation is suggested. The diagnosis of hyperkinetic behavior leads to prescribing stimulant medications. The child’s behavior seems to become more socially acceptable, reducing problems in school and at home. But there is an alternative perspective. By focusing on the symptoms and defining them as hyperkinesis we ignore the possibility that behavior is not an illness but an adaptation to a social situation. It diverts our attention from the family or school and from seriously entertaining the idea that the “problem” could be in the structure of the social system. And by giving medications we are essentially supporting the existing systems and do not allow this behavior to be a factor of change in the system” (Conrad; 1975: 19).

¹⁵ Connors interview with the author.

¹⁶ Ibid.