faith SEEKING UNDERSTANDING

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ADHD: an Imbalance of Fire over Water or a Case of the Fidgets?

In 493 BC Hippocrates identified a condition that seems compatible with what is now called ADHD. He described individuals who had "quickened responses to sensory experience, but also were less tenaciousness because the soul moves on quickly to the next impression." Hippocrates attributed this condition to an "overbalance of fire over water" within the blood, one of the four humors with which the human body was composed. Hippocratic cures for illness and protection from disease resulted from maintaining balance and avoiding imbalance within the body's constitution.

The condition we know today as ADHD dates to the mid-twentieth century, when physicians developed a diagnosis for a set of conditions variously referred to as "minimal brain damage", "learning/behavioral disabilities" or "hyperactivity": "ADHD is defined as a persistent syndrome characterized by inattention, excessive motor activity, and impulsivity of a given developmental stage." Estimates suggest that ADHD affects 8 to 12 percent of children worldwide and approximately 4 percent of adults. The three main symptoms of ADHD are inattention, hyperactivity, and impulsivity. People with ADHD may have educational difficulties, low income, underemployment, and impaired social relationships. (Bernston and Cacioppo 2009, 1020-21).

What is ADHD?

Hippocrates described ADHD simply as an imbalance of fire over water within the blood. Sir Alexander Crichton described a condition of "mental restlessness," " a disease of attention" and "fidgets" in his 1798 book, *An Inquiry Into the Nature of Mental Derangement*.

> The incapacity of attending with a necessary degree of constancy to any one object, which arises casually, like other diseases, accompanies every nervous disorder. . . . In this disease of attention, if it can with propriety be called so, every imperfection seems to agitate the person, and gives him or her an unnatural degree of mental restlessness. . . . When people are affected in this manner, which they very frequently are, they have a particular name for the state of their nerves, which is expressive enough of their feelings. They say they have the *fidgets.*

> Should this state of the nerves continue for a great length of time, or often recur, a habit of inattention is the consequence, which is afterwards with difficulty removed. (Crichton 1798, 271-273)

The cause of ADHD is not known, but a number of factors including genetics, diet as well as social and physical environments seem to contribute or exacerbate ADHD. Twin studies indicate that genetics are a factor in about 75% of all cases of ADHD. In 2007 a study published in *The Lancet*, a British medical journal, noted an association between many common artificial food colors, the preservative sodium benzoate and hyperactivity. The World Health Organization (WHO) stated that the diagnosis of ADHD may suggest family dysfunction or inadequacies in the educational system rather than individual psychopathology. Adherents to neurodiversity theory believe that atypical (neurodivergent) neurological development is a normal human difference that should be tolerated and respected like other human differences. Social construction theory states that societies determine where the line between normal and abnormal is drawn. So society members such as physicians, parents, teachers and others determine what diagnostic criteria are applied, and as a result how many people are affected. One example of this is when the DSM IV (commonly used in North America) was used to diagnose ADHD, it arrived at levels that were three to four times higher than those obtained with the ICD-10 criteria (commonly used in European countries). (Attention Deficit Hyperactivity Disorder, Wikipedia)

According to neurologist Fred Baughman and others, ADHD is *not* a disease. "For a disease to exist, pathology, a word denoting an abnormality, must be present." He suggested that a simple, straightforward definition of disease was that disease=abnormality. No disease meant the individual had no abnormality; in other words, they were normal. "Medical diagnoses cannot be made by opinion, suspicion, or committee; there must be proven facts, a demonstrated, demonstrable abnormality. If there are no abnormalities the result is exactly what we have now, a perversion of language and ethics so twisted that any normal person can be labeled as suffering from a disease." (Baughman 2006, 88; 91)

In a National Institute of Mental Health (NIMH) article, Thomas Insel (2011) said: It has become an NIMH mantra to describe mental disorders [including ADHD] as brain disorders." In contrast to classical neurological disorders, "mental disorders appear to be disorders of brain circuits." This distinction is now made through the use of brain imaging techniques (PET, fMRI, MEG, and high resolution EEG). "For the first time, we can study the mind via the brain." The hope is that a brain disorder approach will transform the way we diagnose mental disorders. Yet we need to recognize that a range of unknowns still exist in understanding brain circuit functions. "While the neuroscience discoveries are coming fast and furious, one thing we can say already is that earlier notions of mental disorders as chemical imbalances or as social constructs are beginning to look antiquated." Much of what is being learned about the neural basis of mental illness is not yet ready for clinical use, "but there can be little doubt that clinical neuroscience will soon be helping people with mental disorders recover."

Peter Breggin, a psychiatrist, noted that at this point in neuroscience, "how the brain functions is still beyond our comprehension." Despite the advances in neuroscience, we have very little understanding of how a single neuron "manages to respond to and transmit messages between itself and tens of thousands of other cells at the very same moment in time." On a larger scale, we have no idea what the organizational or operating principles are that result in what we call *mind* or *consciousness*. "We have little understanding of how the billions of cells and their trillions of connections actually create a functioning brain or mind."

> Given our current state of knowledge, we have no real capacity to make scientific connections between brain function and the kind of complex mental phenomena and behavior such as the ADHD-like problems we deal with in everyday life and in psychiatry. The idea that "ADHD" is caused by a biochemical imbalance is sheer nonsense made up to justify the role of medically oriented experts and the use of drugs. (Breggin 2002, 122-124).

The American Psychiatric Association says that ADHD is: "a pattern of behavior that is present in multiple settings where it give rise to social, educational, or work performance difficulties" manifested within symptoms of inattention and/or hyperactivity and impulsivity. The soon-to-be published DSM V has made some minimal changes to the diagnostic criteria found in the currently used DSM IV. However, there is a significant change with regard to making an ADHD diagnosis. In the DSM IV, several of the symptoms had to be present by the age of 7. Now that threshold age has been raised to 12—seemingly leading to a potential increase in ADHD diagnoses. See Table 1 below for the pending DSM V diagnostic criteria for ADHD.

Both Baughman and Breggin are highly critical of the seeming medical legitimacy given to ADHD via the DSM. Peter Breggin said that the very nature of the DSM diagnosis for ADHD meant that it was absurd to hope that a common biological or genetic basis can be found. ADHD, as defined by the DSM, was not a disease that could be transmitted genetically; it is a description of behaviors that "annoy adults and demand attention. . . . The list was developed over several decades by a core of drug-oriented professionals in order to justify the medicating of children who step out of line and don't conform to teacher expectations." ADHD is largely in the eye of the beholder. "A list of behaviors with such diverse causes will never be found to be rooted in the mythical disorder called ADHD. Nor will behaviors like these ever be genuinely treated with drugs. These children do not have something wrong in their brain, but rather in their lives." (Breggin 2002, 126; 128)

Table 1: Diagnostic Criteria for Attention Deficit/Hyperactivity Disorder[1]

[1] The following are the proposed revisions for Attention Deficit/Hyperactivity Disorder for the DSM V, which was scheduled for publication in May of 2013. None of the diagnostic criteria were changed

Six (or more) of the following symptoms must have persisted for at least six months for either subtype—Inattention of Hyperactivity and Impulsivity—to a degree that is inconsistent with developmental level and that impacts directly on social and academic/occupational activities. Most children have the combined type—six or more of both inattention and hyperactivity-impulsivity.

A1 **Inattention**

- a Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities
- b Often has difficulty sustaining attention in tasks or play activities
- c Often does not seem to listen when spoken to directly
- d Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace
- e Often had difficulty organizing tasks and activities
- f Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort
- g Often loses things necessary for tasks or activities
- h Is often easily distracted by extraneous stimuli
- i Is often forgetful in daily activities

A2 **Hyperactivity and Impulsivity**

- a Often fidgets with or taps hands or feet or squirms in seat
- b Often leaves seat in situations when remaining seated is expected
- c Often runs or climbs in situations where it is inappropriate
- d Often unable to play or engage in leisure activities quietly
- e Is often "on the go" acting as if "driven by a motor"
- f Often talks excessively
- g Often blurts out an answer before a question has been completed
- h Often has difficulty waiting his or her turn
- i Often interrupts or intrudes on others

B Several of the symptoms must be present before the age of 12.

C Criteria for the disorder must be met in two or more settings (e.g., at home, school or work, with friends or relatives or in other activities).

D There must be clear evidence that the symptoms interfere with or reduce the quality of social, academic, or occupational functioning.

E The symptoms do not occur exclusively during the course of schizophrenia or another psychotic disorder and are not better accounted for by another mental disorder (e.g., mood disorder, anxiety disorder, dissociative disorder, or a personality disorder). See Table 1 below for the DSM V ADHD diagnostic criteria for inattention and hyperactivity and impulsivity.

Baughman said that since the criteria for ADHD in the DSM IV had no scientific basis, the diagnosis was open to be misused and abused. "The most striking thing about the DSM IV diagnostic criteria, second only to its complete lack of anything

from what was given in the DSM IV. In "B" the age was raised from 7 to 12. The wording of "E" has been revised.

resembling science, is how dependent it is on how children function in school, which is why there is no surprise that the bulk of initial identifications of ADHD candidates come from schools." Baughman went on to say that when schools use unscientific criteria to get children diagnosed and drugged, "they cheat children out of the opportunity to draw upon the resources they possess to change, adapt, and cope with their environments." Instead of recognizing the problems that occur within the normal developmental process for some students and helping them navigate through these difficulties, "educators label this normal process as a disease" when it conflicts with what they expect out of the children.

> Instead of building a child's confidence in her ability to grow into a successful adult, they undermine it by insisting a neurological abnormality exists whenever the child annoys them, teaching the child they need a pill to function like a normal person. (Baughman 2006, 112; 120)

Let's look at how the DSM diagnostic criteria for ADHD are open for misuse and abuse. First notice that the word "often" is used in every single symptom, without any clarification as to what "often" means. While there is a time frame specified for the symptoms (symptoms have to persist for six months), there is still a significant amount of judgment in determining whether or not the listed behavior constitutes a symptom of ADHD: "to a *degree that is inconsistent with developmental level* and that *impacts directly* on social and academic/occupational activities." Further evidence of the subjectivity of making an ADHD diagnosis is in the vagueness of needing "several" (Three? Five? Nine? More?) of the symptoms to be present before the age of 12. Oh, and does this mean that "several" of the symptoms must have "often" been present for six months or more before the age of twelve?

Second, note that a preponderance of the "symptoms" address behaviors that create difficulties for teachers and other adults trying to manage groups of children. Fred Baughman said the DSM's criteria for ADHD look like they were created by someone "who observed a group of normal children in school and tried to figure out a mechanism for making a disease out of the more irritating, yet normal, behaviors children exhibit, with an eye toward creating as many patients as possible." (Baughman 2006, 121)

Third, regardless of whether a behavior/symptom was classified as hyperactive, impulsive, or inattentive, they can result from a number of different causes that lie *outside* of the children themselves. There could be any number of school problems—such as teacher frustration, student boredom, overly large and disorderly classes; harassment or abuse by others students. There could be unrealistic expectations for academic performance or conformity in classroom behavior. Family problems such as parental conflict or divorce, a lack of parental attention, and inadequate parenting techniques could be behind the ADHD "symptoms." The DSM's diagnostic criteria do not consider potential environmental influences on these behaviors/symptoms.

> [T]he behaviors that we characterize as ADHD have an infinite variety of causes—from boring classrooms to poor discipline at home. Or they can be the normal response of an energetic child to situations that make unrealistic demands for conformity. Often children with ADHD-like behaviors have the capacity to become exceptionally creative and productive human beings. If they escape being diagnosed and drugged, and instead receive needed adult guidance, they can live unusually wonderful lives as children and adults. (Breggin 2002, 136)

One final issue with a DSM diagnosis of ADHD is the caveat of "ADHD Not Elsewhere Classified." This diagnostic code can be used "in cases in which the individuals are below threshold for ADHD or for whom there is insufficient opportunity to verify all criteria. However, ADHD-related symptoms should be associated with impairment, and they are not better explained by any other mental disorder." (American Psychiatric Association. "Proposed Revisions for 'Neurodevelopmental Disorders: Attention Deficit/Hyperactivity Disorder'") In other words, if there wasn't enough time in the evaluation to verify the symptoms, if there weren't the needed six or more of the symptoms persisting for at least six months, if the criteria weren't met in two or more settings, etc., a diagnosis could still be made as "Attention Deficit/Hyperactivity Disorder Not Elsewhere Classified."

Concerns with regard to ADHD diagnosis cannot be dismissed as the ravings of fringe critics of psychiatric practice. Allen Frances, a psychiatrist and professor emeritus at Duke who served as the chair of the DSM IV Task Force, commented on March 3, 2012 that ADHD rates in children have tripled in the past 15 years. While ADD has become a public health problem, "It is much less clear the degree to which the increased rates are real vs. being a manufactured fad." He pointed out that it is no coincidence that rates of ADHD began to skyrocket immediately after two unrelated events in the late 1990s. First, new ADHD drugs were brought to market. These drugs were no better than the old drugs, "but they were lots more expensive and provided a rich profit incentive for aggressive marketing." Second, FDA deregulation freed the drug companies to engage in "unrestrained direct-to-consumer multimedia advertising. The companies quickly determined that peddling the ADD ill was the royal road to expanding the market for their new expensive pills."

Frances said the ADHD epidemic started exactly when aggressive drug company marketing began to "educate" and sensitize doctors, parents, and teachers to spot ADHD in children previously considered to be on the normal side of the spectrum's boundary. Medication is often prescribed carelessly and without clear indication, "causing side effects for the individual and troubling social costs." There is now a thriving secondary market for recreational use and/or performance enhancement; one-third of college student now use stimulants. With regard to the DSM V, he said:

> The ADD proposals being considered for DSM 5 are perversely wrong-headed. Rather than attempting to contain a runaway ADD epidemic, DSM 5 would further fan its flames. Diagnostic thresholds would be

> dropped precipitously. Dr. Bastra [a Dutch psychologist] worries that: "DSM 5 will further inflate the already too broadly defined category of ADD, especially for adults. This will lead to even more misdiagnosis—with consequent misuse and illegal use of stimulant drugs, stigma, and resource misallocation." (Frances, 2012).

William Carey (1998), a respected pediatrician, said the following at the NIH Consensus Conference on ADHD: "What is now most often described as ADHD in the United States appears to be a set of normal behavioral variations that sometimes lead to dysfunction through dissonant environmental interactions. This discrepancy leaves the validity of the construct in doubt."

Is ADHD a Neurochemical Imbalance?

WebMD, a popular medical website, stated that the exact cause of ADHD is not known, but that researchers continue to study the brain for clues. Heredity, chemical imbalance and brain changes (areas of the brain controlling attention are less active in children without ADHD) were hypothesized as factors contributing to the condition. Additional factors that could contribute were: poor nutrition, infection, substance abuse during pregnancy, exposure to toxins, and brain injury or disorder. Implicit here is the assumption that ADHD is primarily caused by a combination of biological factors.

At the 1998 NIH Consensus Conference on ADHD, James Swanson and F. Xavier Castellanos presented a review of ADHD research that "provided considerable evidence of multiple biological bases of ADHD/HKD [Hyperkinetic Disorder]." Converging evidence found that there was a size reduction in "specific neuroanatomical regions of the frontal lobes and basal ganglia," suggesting abnormalities in "neural networks that affect input-output processing and attention (alerting and executive function)." Dopamine was believed to be the principal neurotransmitter involved.

Fred Baughman indicated that the 14 studies reviewed by Swanson and Castellanos at the Consensus Conference all scanned ADHD-treated subjects; there was not one ADHD untreated group. During a question and answer period following the presentation of the paper by James Swanson, Baughman asked him why he failed to mention that virtually all the ADHD subjects in the neuroimaging studies had been on chronic stimulant therapy; and that this was the likely cause of their brain atrophy. Swanson's reported response was: "This is a critical issue and in fact I am planning a study to investigate that." In other words, Swanson admitted that the studies reviewed and reported on as evidence for the biological basis of ADHD had been carried out on ADHD "treated" Ritalin/amphetamine-exposed subjects alone (Baughman 2006, 182).

In 2001, Alan Baumeister and Mike Hawkins did a review of neuroimaging studies of ADHD and said:

> Neuroimaging studies have been conducted with increasing frequency in recent years in attempts to identify structural and functional abnormalities in the brains of persons with attention deficit/hyperactivity disorder. Although the results of these studies are frequently cited in support of a biologic etiology for this disorder, inconsistencies among studies raise questions about the reliability of the findings. The present review shows that no specific abnormality in brain structure or function has been convincingly demonstrated by neuroimaging studies. (Baumeister and Hawkins 2001, abstract; Baughman 2006, 183-84).

F. Xavier Castellanos et al. commented in their 2002 study: "This is the first neuroimaging study to our knowledge to include a substantial number (n=49) of previously unmedicated children and adolescents with ADHD." Castellanos et al. 2002, 1746; Baughman 2006, 184) The unmedicated ADHD patients in the study were significantly younger than the medicated ADHD patients, 8.3 years to 10.9 respectively. Unmedicated patients had a strikingly smaller volume of white matter brain tissue. White matter increases with increasing age (11 year olds would be expected to have a greater volume of white brain matter than 8 year olds), so the authors performed secondary statistical analyses to correct for the age difference and reported that all the measures remained the same. They concluded "that decreased brain volumes in ADHD in both white and gray matter compartments are not due to drug treatment." ADHD was said to be associated with about a 3% decrease in volume throughout the brain. They "did not find evidence of a primarily frontal abnormality in ADHD;" concluding that: we have no evidence that stimulant drugs cause abnormal brain development." (Castellanos et al. 2002, 1743; 1746-47)

Baughman disputed the findings of the Castellanos et al. study, stating the two groups were not suitable for comparison. He rejected the authors' claim that age differences between the two groups were controlled for by their secondary analyses. "A truly matched control group could have been constituted, but was not." Therefore the conclusions reached by Castellanos et al. were not justified, "given that none of the comparisons were valid." (Baughman 2006, 184-85)

> In this study and in all such studies in "biological psychiatry" the only abnormalities found—and they are real—are those induced by the brain-damaging drugs they are invariably put on. In fact, the entire ADHD-MRI literature, all of it showing atrophy of the brain and brain parts, all of it performed on stimulant-treated subjects is proof, replicated time and again, that these medications … are the cause of the brain atrophy. (Baughman 2006, 185)

Jonathan Leo and David Cohen (2003) were also critical of the Castellanos et al. study. What at first seemed to be a rather straightforward comparison of two groups of children became "a tangle of unnecessary complications, including secondary statistical analyses of subgroups, discussions about differences in amounts of white matter in 8-year olds versus 10-year olds, or concerns about

height or weight as variables." A simple comparison of unmedicated ADHD children and controls would have been an important experiment, "but in this study peripheral questions did hopelessly confuse the essential comparison." (Leo and Cohen 2003, 46)

Leo and Cohen (2003) noted that in their 2001 study, Giedd, Blumenthal, Malloy and Castellanos concluded there was clear evidence of brain pathology or dysfunction and a diagnosis of ADHD.[2] They commented that like most researchers in the field, Giedd et al. did not report on a confounding variable, whether or not subjects had been treated previously with stimulants or other psychotropic drugs: "when striving to establish whether cerebral pathology or dysfunction is associated with a given psychiatric diagnosis … it is critical to be able to rule out the probable impact on the brain of prior psychotropic drug use." (Leo and Cohen 2003, 33)

They re-examined the relevant data from the more than thirty neuroimaging studies on children diagnosed with ADHD originally reviewed by Giedd et al. and found that "most subjects diagnosed with ADD or ADHD had prior medication use, often for several months or years." This "substantial confound" invalidated any suggestion of there being evidence for an ADHD-specific neuropathology in these reported studies, as suggested by Giedd et al. "Because of this confound any suggestions about differences between the brains of 'ADHD' children and the brains of 'normal' children must await future studies." (Leo and Cohen 2003, 29, 47; Baughman 2006, 186)

A recent study by Fusar-Poli et al. (2012) did a meta-analysis of PET (positron emission tomography) and SPECT (single-photon emission computed tomography) studies to examine the evidence for a consistent alteration of striatal dopamine transporter density levels across studies.[3] What they found was that the alteration of striatal dopamine transporter levels in ADHD depended upon psychostimulant treatment. Patients with low striatal dopamine transporter levels were medication-naïve, whereas patients receiving long-term medication had high levels. This suggests that a high dopamine transporter level is not part of the ADHD pathophysiology, "but is secondary to years of psychostimulant treatment and reflects an adaptive brain response to the long-term blockade of dopamine transporters by psychostimulants." (Fusar-Poli et al. 2012, 268) This is consistent with findings that methylphenidate (Ritalin, Metadate, Methylin, Concerta) is effective in the short term, while long-term effectiveness is limited. Larger doses are

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[2] Giedd et al. (2001) concluded that research evidence supported the involvement of right frontal-striatal circuitry (neural pathways that connect frontal lobe regions with the basal ganglia that mediate motor, cognitive and behavioral programs in the brain) with cerebellar modulation in ADHD.

[3] The dopamine transporter (also dopamine active transporter, DAT) is a membrane-spanning protein that pumps dopamine out of the synapse. Dopamine reuptake via DAT is the primary mechanism through which dopamine is cleared from synapses. DAT is believed to be implicated in a number of disorders, including ADHD, bipolar disorder, clinical depression and alcoholism. See "Dopamine Transporter" in wikipedia.org.

required to maintain clinical effectiveness and clinical effectiveness seems to wane after years of medication. "Consequently, the previously reported high dopamine transporter density in ADHD patients may potentially represent up-regulation secondary to chronic administration of psychostimulants, rather than primary pathology of ADHD." (Fusar-Poli et al. 2012, 271)

> Despite regular pronouncements that its biologic roots have been discovered, no proof of a definite physical or chemical abnormality is ever found. All such research and all such claims … have been a sham, meant to create illusions of science and disease while proving nothing. (Baughman 2006, 208)

> The neurobiological basis for ADHD remains a cornerstone of the argument for diagnosing and drugging children, even as the search for scientific evidence continues to flounder. The search for a genetic and biological cause of ADHD can never succeed because the biopsychiatric researchers are looking in the wrong place. When a child lacks self-discipline or feels bored and frustrated by school tasks, the fault does not lie in the child's biology but in the adult world's failure to discipline and to engage the child. There are an infinite number of psychological and educational approaches to helping the kind of children who get falsely labeled with ADHD, but these better methods will never be fully implemented until the diagnosis of ADHD and the use of toxic chemical have been abandoned by the psychiatric and educational establishment. (Breggin 2008, 271-72)

To Use or Not to Use ADHD Medications

Peter Breggin reviewed the adverse drug reactions associated with several well-known ADHD medications: dextroamphetamine (Dexedrine and Adderall) methamphetamine (Desoxyn and Gradumet), and methylphenidate (Ritalin). He said: "The limited, questionable, and controversial benefit of stimulant drugs seems to pale beside their suppressive mental effects and many adverse reactions, including persistent brain dysfunction and potentially irreversible CNS damage." He concluded that one of the gravest risks of these psychostimulants was that they would have the intended effect upon children; that they would: "suppress autonomous, spontaneous, social, playful behavior and bring about compliance, docility, and overly-focused obsessive and rote behavior." The widespread use of stimulants enables adults to subdue and control children without improving their parenting or teaching; without addressing issues within the family structure and educational systems. (Breggin 1999, 28-29)

> The behavioral or clinical effects of stimulants may be understood as a continuum of CNS [central nervous system] toxicity. The drugs suppress spontaneous and social behaviors while promoting

> obsessive/compulsive or perseverative behaviors. These adverse drug effects make children more manageable in structured or controlled situations, especially those that lack sufficient adult supervision and attention. The effects are independent of any diagnosable disorder and occur in entirely normal animals and children. (Breggin 1999, 4)

In other words, stimulant medications work because they "disable" the brain.[4] And they have the same effects on the brain, whether or not the individual has been diagnosed with ADHD. According to Peter Breggin, all psychiatric drugs cause a generalized impairment of the brain that reduces overall mental and emotional function. This disabling effect occurs with all individuals who use psychotropic medication—whether or not they have the mental health diagnosis for which the medication is prescribed.

All stimulants work by putting a governor of the person's brain, "literally suppressing the brain's ability to generate spontaneous mental life and behavior." (Breggin 2002, 18) Within an hour of taking a single dose of a stimulant drug, "any child tends to become more obedient, more narrow in focus, more willing to concentrate on humdrum tasks and instructions." (Breggin and Breggin 1995; Breggin 2008, 303) From the first dose of a stimulant drug, blood flow is reduced in all areas of the brain, including the frontal lobes, where the highest human functions are generated. Reduced blood flow causes the brain to receive less oxygen and less nutrition, resulting in a loss of overall function. Researchers found in animal studies that Ritalin reduced the gross metabolic rate throughout many portions of the brain; many parts of the brain stopped functioning at their normal level of activity. These changes would occur in any animal or person, whether or not the person had ADHD-like behaviors/symptoms. "When your child seems to become quieter and even 'more content' after the first dose of any stimulant drug, your child is in effect suffering from a discernable degree of 'brain failure.' With less brain function, your child is less spontaneous and demanding." (Breggin 2002, 44-45)

> All the commonly used stimulants are highly addictive and subject to abuse. All are similar to cocaine in their properties. They can cause many physical problems, including cardiovascular dysfunction, growth suppression, and tics. They can also cause many serious psychiatric side effects such as agitation, aggression, psychosis, mania, depression, and obsessive-compulsive disorder. (Breggin 2002, xv)

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[4] The brain-disabling concept of biopsychiatric treatment states that: "all psychiatric treatments—drugs, electroshock, and lobotomy—work by disabling the functions of the brain and mind, creating effects that are then interpreted (or misinterpreted) as improvements." (Breggin 2008, 1)

Psychiatric Problems and ADHD Medications

All stimulant medications are required to carry a warning of potential psychiatric problems. What follows is the warning in the Medication Guidelines for Ritalin-SR®. Other ADHD medications have similarly worded warnings.

Mental (Psychiatric) problems:
All Patients

- new or worse behavior and thought problems
- new or worse bipolar illness
- new or worse aggressive behavior or hostility

Children and Teenagers

- New psychotoc symptoms (such as hearing voices, believing things that are not true, suspicious) or new manic symptoms

Tell your doctor about any mental problems you or your child have, or about a family history of suicide, bipolar illness, depression.
Call your doctor right away if you or your child have new or worsening mental symptoms or problems while taking Ritalin-SR®, especially seeing or hearing things that are not real, believing things that are not real, or are suspicious.[5]

Peter Breggin examined eight representative clinical trails for adverse drug reactions (ADRs). All of them were conducted by advocates of stimulants and aimed to prove that drugs were safe and effective. He estimated that the reported rate of serious adverse reactions in children was as high as 10 to 20 percent. The rate in clinical practice would likely be even higher. "If clinically observable, potentially significant ADRs are included, the rate is much higher, in the 20-50% (or more) range." Beggin 1999, 10)

One of four studies that evaluated a spectrum of ADRs reported that 26.1% of the children suffered from "irritability;" 18.8% suffered from lethargy; 5.8% had an increase of stereotypical behaviors (hand wringing, arm waving, teeth grinding, etc.); 7% had disturbing ADRs that included manic, incessant talking, aggressive behavior and being "wild" and "out of control." Allowing for overlapping reports of one or more ADR, he estimated that over 50% of the children suffered from lethargy and other adverse CNS reactions.

Three studies focused on obsessive/compulsive ADRs. One study, of children with one daily dose of MPH (Ritalin) over 3 days found that 42% became "overaroused" with "cognitive perseveration" [compulsive, persistent, meaningless activies]. Another study had a rate of 25% obsessive ADRs over three weeks of MPH treatment. One study, by Boreherding et al. was particularly disturbing. Tics, overfocusing, and other compulsive behaviors were observed in 76% of the participants. Perseverative ADRs were observed in 51% of the children. Of these, over half (31% of the original subjects) suffered one or more abnormal movements. At least three suffered sever drug-induced obsessive-compulsive symptoms. For

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[5] See the Medication Guidelines link below for Ritalin-SR®.

example, one child played with Legos for 36 hours without a break to eat or sleep. Another raked leaves for 7 consecutive hours; and still felt compelled to rake individual leaves as they fell. "Despite such high rates for serious, severe ADRs, the rates and severity of ADRs should be expected to be much higher under routine clinical conditions." These conditions would likely include much longer exposures to stimulants (months or years instead of the 1-3 weeks in most of the controlled studies); often higher doses; less adequate medical evaluations and supervision; "and parents and teachers who are not educated to identify ADRs and to terminate treatment before they worsen." Breggin 1999, 4-10)

By his research, Breggin compiled the following harmful stimulant effects, which are often mistakenly seen as improvements in children diagnosed with ADHD.

Table Two: Harmful Stimulant Effects

Obsessive Compulsive Effects

- Compulsive persistence at meaningless activities (called stereotypical or perseverative behavior)
- Increased obsessive-compulsive behavior (e.g., repeating chores endlessly and ineffectively)
- Mental rigidity (called cognitive perseveration)
- Inflexible thinking
- Overly narrow or excessive focusing

Social Withdrawal Effects

- Socially withdrawn and isolated
- General dampened social behavior
- Reduced communicating or socializing
- Decreased responsiveness to parents and other children
- Increased solitary play and diminished overall play

Behaviorally Suppressive Effects

- Compliant in structured environments; socially inhibited, passive and submissive
- Somber, subdued, apathetic, lethargic, drowsy, dopey, dazed, and tired
- Bland emotionally flat; humorless; not smiling; depressed and sad, with frequent crying
- Lacking in initiative or spontaneity, curiosity, surprise, or pleasure

(Breggin 1999, 25; Breggin 2002, 24; Breggin 2008, 306)

The following central nervous system side effects were noted in *Psychotropic Drug Fast Facts,* a handbook frequently used by physicians.

Table 3: Central Nervous System Side Effects

- Confusion, a "dopey feeling"—especially arises with over 1 mg daily of methylphenidate;
- dizziness;
- dysphoria— occurs with all stimulants but especially with methylphenidate, which causes: mild dysphoria; subtle social withdrawal; dulled affect, emotional blunting; cognitive "overfocusing;" preseveration (compulsive persistence at meaningless tasks). Dysphoria may be related to withdrawal effects. Tics can occur, but usually with higher doses.
- Insomia—usually occurs at initiation of treatment; affects 30% of children on moderately high stimulant doses.
- Mania, psychosis—at high doses can induce symptoms of mania and psychosis. Symptoms generally resolve within 2 days after discontinuation of the stimulant. Symptoms lasting 6 days or longer have been reported.
- Restlessness

(Maxmen, Ward and McIntyre 2008, 204-05)

Stimulant drugs often cause obsessive-compulsive behavior in children. But teachers value these traits and almost never interpret them as negative drug effects. An imaginative child who used to become easily distracted by her own thoughts; but on stimulants, she is compulsively focused. The energetic boy who could not sit still becomes drained of spontaneity now sits in his chair for the duration of the school day. The social butterfly who wants to chat with her friends now sits through every lesson as if she has no friends in class. Parents, who were weary of their child's need for attention and resistance to doing homework or chores, get relief in their compulsive attention to homework or their preoccupation with computer games. "These quieter, preoccupied children provide a respite for their parents and even seem to be doing 'better' when in fact they are suffering from stimulant drug toxicity." (Breggin 2008, 306-07)

Many studies have compared stimulant-induced psychoses to the symptoms of schizophrenia. MPH (Ritalin) has even been used experimentally to produce or worsen psychotic symptoms in adults diagnosed with schizophrenia. "Stimulants are also known to cause a disorder that may remain chronic and become indistinguishable from schizophrenia." (Breggin 1999, 11)

Addiction and ADHD Medications

Shankar Vedantam of the *Washington Post* reported on a study published in the *Journal of Drug and Alcohol Dependence,* which estimated that 7 million Americans have misused ADHD medications; and substantial numbers of teenagers and young adults showed signs of addiction. "[A]bout 1.6 million teenagers and young adults had misused these stimulants during a 12-month period and that 75,000 showed signs of addiction." Nora Volkow, the director of the National Institute on Drug Abuse, was quoted as saying with regard to taking prescription stimulants to boost academic performance: "You are playing with roulette. . . . If you get addicted, you will not only not get into Harvard, you will not finish high school." (Vedantam 2006)

According to a 2010 national survey, an estimated 1.1 million persons aged 12 and older were currently using stimulant drugs illicitly. The average age for an initial use of cocaine and other stimulants was 21.2 years. It was estimated that 343,000 people sought treatment for their use of stimulants in the year prior to the survey. (2010 National Survey on Drug Use and Health)

The 2012 World Drug Report said that amphetamine-like stimulants were the second most widely used class of drugs worldwide, with between 14.3 (.3%) and 52.5 (1.2%) million users worldwide. Increased seizures of methamphetamine in South West Asia, Central Asia and Transcaucasia (a region comprising Georgia, Armenia, and Azerbajian between the Caucasus Mountains and the borders of Turkey and Iran) suggest that its use may be on the increase in those areas. (World Drug Report 2012)

The DEA website states: "Methylphenidate [Ritalin], a Schedule II substance, has a high potential for abuse and produces many of the same effects as cocaine or amphetamines. . . . The increased use of this substance for the treatment of ADHD has paralleled an increase in its abuse among adolescents and young adults."

Within a 1995 article in the *Archive of General Psychiatry,* Volkow et al. found that the distribution of cocaine and methylphenidate (Concerta, Ritalin) in the brain were identical, "but that the latter remained for a longer period of time." An editorial in the same issue stated: "Cocaine, one of the most reinforcing and addictive of abuse drugs, had pharmacological actions very similar to those of MPH [methylphenidate], one of the most commonly prescribed psychotropic medications for children in the United States." (Breggin 2008, 300).

Volkow and Swanson (2003) noted that when abused, methyphenidate is usually administered intranasally (snorted) or injected intravenously. It is rarely abused orally. Oral doses with a >50% dopamine transporter blockade did not reliably produce the subjective experience of being "high." A threshold of greater than 50% for dopamine transporter blockade was found to be necessary, but not sufficient to produce the high effects from methyphenidate. These effects are dependent upon the rapid increase of dopamine levels, meaning that snorting or intravenous use of methyphenidate is usually necessary experience a high. Volkow and Swanson speculate that methyphenidate's ability to induce rapid increases in dopamine is

associated with abuse, whereas slower steady-state increases in dopamine are associated with clinical use of methyphenidate. They concluded if you take methyphenidate as directed, you will rarely have a problem with abuse.

> Reinforcing effects occur when methyphenidate elicits large and fast dopamine increases … whereas therapeutic effects occur when methyphenidate elicits slow, steady-state dopamine increases. . . . Thus the characteristics of clinical use (low doses administered orally and titrated for therapeutic effects) constrain methyphenidate abuse. (Volkow and Swanson 2003, 1909)

Nadine Lambert (2005) conducted a 28-year longitudinal study of ADHD children and normal controls. The participants were followed through childhood and adolescence; and then interviewed 3 times as adults. When other variables were accounted for, the severity of ADHD increased the odds of dependence on the substances in the investigation: tobacco, cocaine, amphetamine and cocaine/amphetamine. Lambert also found that: "Stimulant treatment increased the odds of dependence on tobacco, cocaine, and cocaine/amphetamine. . . . ADHD and problem behavior did not increase the odds of either daily smoking or lifetime use of any of the substances." Breggin (2008, 303) commented that Lambert's findings indicated that: "It is not ADHD but the treatment for ADHD that puts children at risk for future drug abuse."

Volkow et al. (2007) found that subjects with ADHD reported more intense reinforcing effects (drug high) after intravenous methyphenidate (Concerta, Ritalin) than controls. "This association could contribute to the higher vulnerability for substance abuse comorbidity in adult subjects with ADHD." (938)

Physical Problems and ADHD Medications

At the 1998 NIH Consensus Conference on ADHD, James Swanson presented research on the then current genetic and brain scan studies allegedly showing the biological basis of ADHD. In the unpublished public discussion following Swanson's presentation, neurologist Fred Baughman asked if any of the studies in his review involved children without a history of drug treatment. "Swanson could not name a single study based on untreated patients." After hearing all the scientific presentations and discussions, the Consensus conference panel concluded that although evidence supporting the validity of the disorder can be found, "after years of clinical research and experience with ADHD, our knowledge about the cause or causes of ADHD remains speculative." (National Institutes of Health. 1998, November 16-18) Although the cause or causes of ADHD are not known, the damage done by ADHD medications is. Stimulant drugs cause serious malfunctions in the brain from the very first dose—the immediate sedation effect discussed above. "Exposure to repeated doses of stimulants in animals over a period of days or a few weeks can cause persistent damage to their brains." Multiple brain-scan studies of

children diagnosed with ADHD have shown abnormalities in a variety of areas of the brain (Breggin 1999, 16; Breggin 2008, 309).

Peter Breggin has pointed to a series of physical problems associated with the use of ADHD medications, in children which include: stunted growth, heart problems, stroke, tics, convulsions, gastrointestinal problems, headaches, and blurred vision. Additional physical problems with stimulants can include: seizures, decreased appetite, stomach pain, trouble sleeping, dizziness and weight loss. Here we will comment briefly on the first two problems, stunted growth, and heart problems. See Breggin 1999 (4-7) and Breggin 2002 (33, 43-66) for more detailed information.

Stimulants cause a marked dysfunction in the production of growth hormone, with an abnormal increase during the day and an abnormal suppression at night, "when it most significantly affects growth." The impact is so dramatic, that some researchers have observed that growth-hormone levels can be used to judge whether or not children are taking their ADHD medication. "The disruption of growth hormone should be viewed as an ominous finding. It means that all growth processes are being impaired, including the growth of the brain, heart, and lungs." (Breggin 2002, 54-56)

Stimulants produce a double assault on the heart by over stimulating heart rate and blood pressure, and then weakening the overstressed heart muscle. Below, the concern with heart problems is discussed as one of the reasons given for the need of a black box warning with ADHD medications. And the FDA is noted to have announced that they "did not find an association between the use of ADHD medications and cardiovascular events." But Peter Breggin commented that he has been consulted in several cases where stimulant drugs caused fatal cardiac arrhythmias in children. "In one case, the child's heart on autopsy showed a pattern of deterioration that the corner compared to changes he had seen in chronic cocaine addicts." All stimulants can cause strokes, probably as a result of hypertension caused by the ADHD drugs. "Overall, stimulants pose serious cardiovascular hazards. Individuals suffering from or at risk of experiencing hypertension, heart disease, or strokes should especially avoid stimulants." (Breggin 2002, 56-58)

Peter Breggin was one of the speakers at the 1998 NIH Consensus Conference on ADHD, where he presented the data supporting the warnings of psychiatric, physical and addictive problems discussed above. He commented that if he could identify the problems, the combined resources of the drug companies and the FDA should have been able to do so far quicker and easier. After he publicized the problem at the 1998 conference, the FDA and the drug companies no longer had an excuse for failing to conduct their own analyses to test and to confirm his observations. But they still did not act for nearly a decade.

During the 2006 FDA hearings on stimulant medication discussed below, Peter Breggin again presented his findings in the hope that the agency would take seriously the seemingly mutual concerns about adverse psychiatric effects such as

suicide and violence. “But the FDA was already withdrawing from its previous declarations about the risks associated with stimulants.” (Breggin 2008, 298)

Should ADHD Medications Carry a Black Box Warning?

On February 9, 2006 the FDA's Drug Safety and Risk Management advisory committee recommended by a vote of 8 to 7 (1 abstaining) that medications for ADHD carry a ‘black box’ warning (the Food and Drug Administration's strongest warning), because of the potential risks of heart damage and sudden death.[6] Since nearly 3.3 million Americans age 19 and younger used an ADHD drug in 2005 (according to Medco Health Solutions Inc., a prescription drug benefit program manager), the panel decided a black box warning should be added because of the health concerns. An additional recommendation by the committee (15 to 0, 1 abstaining) was to distribute Medication Guides warning of potential cardiovascular risks with stimulant drugs used for ADHD treatment.[7]

The unexpected vote came during a meeting when medical experts were being asked to determine “research approaches” that could be used to study whether ADHD drugs increase the risk of heart problems, according to an FDA description of the gathering. The potential heart-related concerns with ADHD medications include: stroke, hypertension, palpitations, arrhythmia and heart attacks. Of the 25 reported deaths, 19 were children. According to data presented to the advisory panel, a child on an ADHD drug increases his/her risk of sudden cardiac death by 1.5 to 2.5.

Members of the Drug Safety and Risk Management advisory committee said the recommendation was driven as much by worries that ADHD drugs are being overused in the United States as by the possible side effects. About 10 percent of 10-year-old American boys are taking such medications. “On the surface, it is hard to believe,” said Curt Furberg, professor of public health sciences at North Carolina's Wake Forest University Medical School, who voted for the black-box warning. “What is also interesting is this condition is not really recognized in other countries -- you wonder what we are treating. I am sure there are patients who need these drugs, but it is not 10 percent of all 10-year-old boys.”

While diagnosis is most common in children, especially boys, physicians have recently started writing more prescriptions for adults. Cardiologist Steven Nissen of the Cleveland Clinic, who was one of the committee members who pushed for the

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[6] “‘Black Box’ ADHD Drug Warning Rejected.” See the link below for the original article. A ‘black box’ warning is a type of warning that appears on prescription drugs that can cause serious adverse effects. It refers to the black border that usually surrounds the warning. A black box warning means that medical studies indicate that the drug carries a *significant* risk of serious or even life-threatening adverse effects. The U.S. Food and Drug Administration (FDA) can require a pharmaceutical company to place a black box warning on the drug's labeling, advertising and literature. It is the strongest alert the FDA can require.

[7] Results of the votes taken from the Summary minutes for the February 9 and 10 2006 meeting of the Drug Safety and Risk Management Committee. Found on fda.gov.

warning label, said the growing use of ADHD drugs in adults is a serious concern because the risk of heart attack rises among adults older than 50.

While the FDA isn't bound by its advisory panels' recommendations, it usually does what the advisory panel recommends. However, many health professionals were concerned that the move to a black box warning was too severe given that the risk of a cardiac event was said to be extremely low (supposedly less than one per million).

In March of 2006 the FDA asked a different advisory committee, the Pediatric Advisory Panel, to examine the cardiac event issue as well as reports that psychosis or mania can occur in some juvenile patients at normal doses of any ADHD drug. FDA investigators had discovered that some children with no identifiable risk factors suffered potentially traumatizing visions, although they cannot point to a definitive link, "the predominance of hallucinations involving insects, snakes and worms is striking." They noted a "complete absence" of similar reports in children treated with dummy pills during dozens of clinical trials of the drugs. In many children, the events ceased once they stopped taking the drugs — and resumed in some once they restarted. "That's unlikely to be due to random chance, suggesting some effect of the drugs," said Dr. Andrew Mosholder, of the FDA's division of drug risk evaluation.

The Pediatric Advisory Panel did not support the Safety and Risk Management Committee's recommendation for a black box warning, but did suggest that labeling information for ADHD medications be updated to warn of possible side effects including aggression, cardiovascular events, and psychiatric symptoms such as psychosis and mania. The Pediatric Advisory Committee did not think that a black box warning was needed. Yet they did recommend that a strong warning be placed within the "Highlights" section of the labeling for patients with underlying structural cardiovascular defects or cardiomyopathies. (Pediatric Advisory Committee 2012).

As a result of the disagreement between the two FDA committees, it took the FDA several months to come to a decision about the conflicting labeling recommendations. In the meantime, within the April 2006 edition of the *American Journal of Psychiatry*, Zuvekas et al. (2006) concluded that a steep rate of increase in the use of stimulant medication among children between 1987 and 1996 had leveled off between 1997 and 2002. The reported prevalence rates for children under the age of 19 who used stimulants was 2.7% in 1997 and 2.9% in 2002. Peter Breggin noted this was a significantly lower estimate than that reported by the CDC in Vissler et al. (2005), where 11% of children had been diagnosed with ADHD at some time in their lives; and 4.3% overall were being treated with medication for ADHD.

Within a December 2006 editorial, "2006 in Review," Robert Freeman, one of the editors for *American Journal of Psychiatry* commented that the Zuvekas et al. study "showed that the prescription of stimulants to children has been remarkably stable over the past decade and that, if anything, too few children are treated." So it was

intentionally moved ahead several months from its original publication date so that it would appear "while the FDA hearings were ongoing, and the FDA decided to not issue a more severe warning about the safety and use of drugs that have a unique value in the treatment of childhood mental disorder."[8] Although the FDA decision against a black box warning on ADHD medications cannot be attributed to the influence of the Zuvekas et al. study, it does seem that the accelerated publication date was intended have an effect on that decision.

It wasn't until February 21, 2007, that the FDA officially directed the manufacturers of all drug approved for the treatment of ADHD "to develop Patient Medication Guidelines to alert patients to possible cardiovascular risks and risks of adverse psychiatric symptoms associated with the medicines." Patient Medication Guides are required handouts given when a medication is dispensed; and they contain FDA-approved patient information about the medication. The medicines that were the focus of the revised labeling and Patient Medication Guides included:

- Adderall (mixed salts of a single entity amphetamine product) Tablets
- Adderall XR (mixed salts of a single entity amphetamine product) Extended-Release Capsules
- Concerta (methylphenidate hydrochloride) Extended-Release Tablets
- Daytrana (methylphenidate) Transdermal System
- Desoxyn (methamphetamine HCl) Tablets
- Dexedrine (dextroamphetamine sulfate) Spansule Capsules and Tablets
- Focalin (dexmethylphenidate hydrochloride) Tablets
- Focalin XR (dexmethylphenidate hydrochloride) Extended-Release Capsules
- Metadate CD (methylphenidate hydrochloride) Extended-Release Capsules
- Methylin (methylphenidate hydrochloride) Oral Solution
- Methylin (methylphenidate hydrochloride) Chewable Tablets
- Ritalin (methylphenidate hydrochloride) Tablets
- Ritalin SR (methylphenidate hydrochloride) Sustained-Release Tablets
- Ritalin LA (methylphenidate hydrochloride) Extended-Release Capsules
- Strattera (atomoxetine HCl) Capsules[9]

The Medication Guides for the above ADHD medications warn of the following serious side effects when taking ADHD medications: 1) heart (cardiovascular)-related problems; 2) psychiatric problems; 3) growth suppression; 4) seizures; 5)

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[8] I'm indebted to Peter Breggin in *Brain Disabling Treatments in Psychiatry,* 2nd edition, for his original observations on the Zuvekas et al. (2006) study and the intent of the editors of the *American Journal of Psychiatry* in moving up its publication date.

[9] See the following link for the FDA February 21, 2007 press release: http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/2007/ucm108849.htm.

For the Medication Guides and Label information for these ADHD medications see: http://www.fda.gov/Drugs/DrugSafety/PostmarketDrugSafetyInformationforPatientsandProviders/ucm107918.htm.

blurred vision; 6) tics. All of the above except Strattera warn of all six serious side effects. Strattera lists only heart problems, psychiatric problems and growth suppression from the above; and adds suicidality and severe liver damage to the concerns. Additional so-called common side effects include: 1) headache; 2) decreased appetite; 3) stomach ache, pain, upset; 4) nervousness, anxiety, or mood swings; 5) trouble sleeping; 6) dizziness; 7) weight loss. Ritalin does not mention dizziness or weight loss. Strattera does not mention headache or weight loss.

On November 12 of 2010, the CDC published a report entitled "Increasing Prevalence of Parent-Reported Attention-Deficit/Hyperactivity Disorder Among Children --- United States, 2003 and 2007." Within that report, the percentage of children aged 4 to 17 with a parent-reported ADHD diagnosis increased from 7.8% to 9.5% during 2003-2007; an increase of 21.8% in 4 years. In total, 4.8% of all children aged 4 to 17 years (2.7 million) were taking medication for ADHD. Boys (at 13.2%) were more likely to be diagnosed with ADHD than girls (at 5.6%). The highest rates of parent-reported ADHD diagnosis were found with children covered by Medicaid and multiracial children. These prevalence rates also varied by state, from a low of 5.6% in Nevada to a high of 15.6% in North Carolina reported in 2007.[10]

Not only did the rate of ADHD diagnosis seem to increase in the CDC report, but the 2003 prevalence rate for parent-reported diagnosis was dramatically higher than the prevalence rate of 2.9% given in the Zuvekas et al. (2006) study. One of the limitations to their study noted by Zuvekas et al. (2006) was the potential for under reporting and underestimating drug use. Nevertheless, the authors believed their findings were consistent with those derived from a private national insurance claim database.

Within a 2011 update, the FDA announced that a recently completed study in children and young adults treated with ADHD medications "did not find an association between the use of ADHD medications and cardiovascular events." Nevertheless, "The possibility of a small to modest increase in risk cannot be ruled out because of the small number of serious cardiovascular events observed [7 in 1,200,438 children and young adults aged 2-24 years] in the patients studied."[11]

Ultimately, it seems that the FDA and the American Psychiatric Association believe that the benefits of using ADHD medication outweigh the risks. The FDA does not believe at this time that ADHD stimulant medications warrant a black box warning, while they do believe that such a warning should exist for children being prescribed SSRI, SNRI and NRI, antidepressants. The list of ADHD medications above (with the exception of Straterra) are classified as Schedule II Controlled Substances,

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[10] See the following link for the CDC 11/12/2010 report: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5944a3.htm?s_cid=mm5944a3_w. Also refer to http://www.cdc.gov/NCBDDD/adhd/data.html#med for additional CDC data and statistics on ADHD.

[11] See the following link for the FDA November 1, 2011 update: http://www.fda.gov/Drugs/DrugSafety/ucm277770.htm#data

meaning they have a high potential for abuse, which can lead to severe psychological or physical dependence. Other Schedule II controlled substances include cocaine, hydrocodone (Vicodin), morphine, methadone and fentanyl. Strattera is not classified as a stimulant under the Controlled Substance Act. It is a selective norepinephrine reuptake inhibitor (NRI) approved for the treatment of ADHD in 2002. In 2004 the FDA directed that Straterra carry a black box warning, similar to SSRI and SNRI antidepressants because of the potential for serious mental health side effects including suicidal thoughts and psychosis in children and teenagers. So your current approved medication choices for treating ADHD in children either have a high potential for abuse, with a risk of cardiovascular problems or adverse psychiatric symptoms; or they can potentially cause suicidal thoughts and psychosis.

Madelyn S. Gould and others published a case-control study that looked at the association of sudden death and the use of stimulants. Although sudden unexplained death is a rare event, the study found that "the odds of using stimulants were six to seven times greater for the cases of sudden unexplained death than for matched motor vehicle accident deaths." (Gould et al. 2009, 997) There were several limitations to the study. The case-control studies could detect association, but not establish causality. And the authors were not able to systematically obtain information on the psychiatric status of the decedents. Nevertheless,

> This study reports a significant association or "signal" between sudden unexplained death and the use of stimulant medication, specifically methylphenidate. While the data have limitations that preclude a definitive conclusion, our findings draw attention to the potential risks of stimulant medications for children and adolescents. (Gould et al. 2009, 1000)

Long Term Effectiveness of ADHD Medication Questionable

There seems to be a growing consensus that a pure neurobiological explanation for ADHD is not adequate. Yet the neurobiological basis for ADHD continues to be used to justify the drugging of children.

> Growing scientific evidence suggests that ADHD cannot be explained by genetic or environmental factors alone. Research that integrates social and scientific perspectives is likely to achieve a more complete explanation. . . . The social, clinical and behavioural complexities of ADHD create enormous challenges for scientific research. Rather than looking for discrete causal factors in ADHD, investigations are increasingly focused on identifying complex genetic, biological and environmental risk factors. (Singh 2008, 958-59)

Despite the above discussion questioning its legitimacy, ADHD has become a globally recognized and diagnosed *neurobehavioral* childhood disorder, where "the evidence supporting medication-based interventions (such as methylphenidate) is

strong." A Global ADHD Working Group issued a consensus statement that aimed to "re-affirm ADHD as a valid disorder that exists across different cultures, has a significant global impact, and should be diagnosed and effectively treated wherever it occurs." (Remschmidt, H. & Global ADHD Working Group 2005, 127) Ilina Singh (2008) said that along with the global increase of ADHD diagnosis in children, there has been a concurrent growth in stimulant prescription. Indeed, the Global ADHD Working Group has even developed "consensus treatment algorithms" designed to guide the multimodal treatment of ADHD by qualified treatment professional worldwide. And yet, the long term effectiveness of ADHD medications has been acknowledged as ineffective even by some ADHD advocates.

The National Institute of Mental Health (NIMH) funded a nationwide, long-term study of the effectiveness of stimulants in treating ADHD by many of the long-time advocates of stimulant medication. It was almost a foregone conclusion that the resultant publications would be in favor of drug efficacy. The NIMH Multimodal Treatment Study of ADHD (MTA), identified several co-occurring mental health conditions with ADHD: 39.9% were diagnosed with oppositional defiant disorder; 38.7% with an anxiety disorder; 14.3% with a conduct disorder; 10.9% with a tic disorder; 3.8% with an affective (mood) disorder. Only 31.8% had pure ADHD; ADHD without the comorbid diagnoses. All the children met the criteria for the ADHD-combined type; 30% had been previously treated with stimulant medication. These co-occurring disorders were diagnosed through parental interviews with the Diagnostic Interview Schedule for Children (DISC).[12] (Jensen et al. 2001)

In 2007 the authors finally published their evaluation of long-term effectiveness. "By 36 months, the earlier advantage of having had 14 months of the medication algorithm was no longer apparent." All five subgroups showed an initial advantage of medication that disappeared by the 36-month assessment. (Jensen et al. 2007; Swanson et al 2007). In defense of their drugs, the MTA authors suggested that perhaps all the children simply got better over the 36 months; their ADHD just went away.

> First of all, this is contrary to the persistent argument made by drug advocates that ADHD is a real biological disease that does not go away and that requires long-term, even lifetime, treatment. Second, if it is true that so-called ADHD clears up on its own, that makes a good argument for never giving toxic drugs to children. (Breggin 2008, 284)
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> The limited, questionable, and controversial benefit of stimulant drugs seems to pale beside their suppressive mental effects and many adverse reactions, including persistent brain dysfunction and potentially irreversible CNS damage. Pharmacological interventions in

[12] The DISC is a highly structured diagnostic interview, designed to assess more than 30 psychiatric disorders occurring in children and adolescents. It can be administered by "lay" interviewers after a minimal training period.

> the brain to suppress spontaneous behavior and to promote obsessive ones is wrong in principle. Enough is already known about the lack of benefit and the negative impact of stimulants to stop prescribing them for "ADHD" or for the control of any symptoms or behaviors in children. (Breggin 1999, 29)

Even James Swanson has now admitted there seems to be no long-term benefit to ADHD medication. Within an article by Daniel Goleman (2014), Swanson was quoted as saying: "There are no long-term, lasting benefits from taking A.D.H.D. medications." Swanson and Goleman were referring to a 2013 follow-up study of the Preschool ADHD Treatment Study (PATS) that concluded: "Medication status during follow-up, on versus off, did not predict symptom severity change from year 3 to year 6 after adjustment for other variables." (Riddle et al. 2013)

Conclusion

Consistent with the neurodiversity theory noted above, Francis Fukuyama suggested that:

> ADHD isn't a disease at all but rather just the tail end of the bell curve describing the distribution of perfectly normal behavior. Young human beings, and particularly young boys, were not designed by evolution to sit around a desk for hours at a time paying attention to a teacher, but rather to run and play and do other physically active things. The fact that we increasingly demand that they sit still in classrooms, or that parents and teachers have less time to spend with them on interesting tasks, is what creates the impression that there is a growing disease." (Fukuyama 2002, 47)

ADHD may prove someday to be a legitimate disorder with a biological component. Yet the current diagnostic criteria used to diagnose it and justify the use of the array of harmful ADHD medications do not provide a scientific basis for these actions. Perhaps we'd be better off describing ADHD as an imbalance of fire over water like Hippocrates; or mental restlessness and a case of the fidgets as did Sir Alexander Crichton. Do some careful research before starting yourself or your child on any ADHD medication. Better yet, don't do it at all.

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