Attention Deficit/Hyperactivity Disorder: A Review of Research

An Honors Thesis (HONRS 499)

by

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Abstract

Attention deficit hyperactivity disorder (ADHD) is a relevantly new term for an old disorder. This is a review of research on the disorder of ADHD. History, prevalence statistics, diagnosis trends, medication information, and effects of exercise on the disorder are addressed. The review of research is not comprehensive, but includes the main points throughout the history of the disorder. The disorder is continually being researched and revised, so it is essential to review all current information concerning the disorder.
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Table Of Contents

Title Page ................................................................................................................... 1

Abstract ..................................................................................................................... 2

Acknowledgements................................................................................................... 3

Table of Contents ...................................................................................................... 4

I. Introduction ............................................................................................................ 5

II. History ................................................................................................................... 7

III. Prevalence ............................................................................................................ 15

IV. Diagnosis ............................................................................................................. 17

V. Treatment Options ............................................................................................... 20

VI. Exercise Effects ................................................................................................... 26

VII. Conclusion ......................................................................................................... 34

Appendix A ............................................................................................................... 36

References ................................................................................................................. 39
I. INTRODUCTION

What is a behavioral disorder? Aren’t most of us a little quirky anyway? Many researchers and psychological professionals have attempted to define behavioral disorders over time, mostly in the last two centuries. Attention deficit hyperactivity disorder (ADHD) is a behavioral disorder that has been examined in the past 150 years. ADHD is defined as “an early onset, biological disorder, classically characterized by a triad of symptoms: hyperactivity, inattention, and impulsivity. These three symptoms are persistent and at developmentally inappropriate levels” (www.ADHD.org.nz).

ADHD is considered to be a neurobiological disorder affecting certain areas of the brain that control behavior, such as the frontal lobe. It is suggested that there is impairment in the brain with dopamine response; however, the specific underlying neurochemical deficiency is not fully understood. Dopamine is believed to be a chemical neurotransmitter in the brain that controls behavior, specifically improving attention and inhibiting impulsive and hyperactive behavior. Dopamine response in the brain is being researched and the latest information suggests that this impairment is due to a reduced sensitivity of dopamine receptors, not necessarily inadequate levels of dopamine (Biederman et al. 1999). Thus, ADHD is considered a behavioral disability that can be explained by an underlying neurochemical dysfunction. However, the signs and symptoms of ADHD manifest themselves in different ways in each individual. As the knowledge of the disorder improves so too will the understanding of the biological basis for the behavior as well as the methods of treatment.

Diagnosis, treatment, and prevention are largely difficult to describe because each one of these topics closely relate with many other psychological or behavioral disorders.
These similarities and differences are significant because the disorder is constantly being redefined. The significance of research is highly crucial to defining the signs and symptoms of this particular disorder.

ADHD is a debilitating disorder because it hinders most children from responding properly to parents, teachers, or any other condition where behavior needs to be optimized. Inattention, impulsivity, and hyperactivity vary across individuals, but each component has an impact on the societal, psychological, and physiological well being of that individual. If a child is in school and cannot sit still for more than a few minutes at a time, this becomes a problem because it disrupts class and the child does not engage in the learning activity. This disorder not only affects the learning environment, but can also affect the home environment, or even playing with other children. In each case, there may be consequences for specific actions. For example, a child might be hyperactive and get placed in a special class because of not being able to be “normal.” In most cases, some behavioral problems are easier to notice than others. For example, inattention may be more difficult to notice than hyperactivity or impulsivity since it appears to be more of a problem within the child as opposed to hyperactivity or impulsivity which can lead to problems between children or elders. As a result of the child’s behavior problems associated with ADHD, social, emotional, and cognitive development may be impaired.

The purpose of this literature review is to present the history of ADHD, describe the prevalence rates, outline current theories on the diagnosis of this disorder, examine the various treatment options (medication and alternative techniques), and identify the effects of exercise on behavior and physiological responses. Despite the wealth of
research that has been conducted on a variety of areas related to the disorder, it is still not fully understood.

II. HISTORY OF ADHD

Is ADHD a relatively new disorder or has it existed for thousands of years? Many people believe ADHD to be a new disorder, but in fact, it has been documented for at least 150 years, and likely around for many more. ADHD has been known formerly as many different names, ranging from “Fidgety Phil” to “ADD.” Different names have been ascribed to this disorder, which appears to stem from different views and characterizations on the disorder, have been postulated over time. Part of the variation in the name of the disorder is related to beliefs regarding the cause or causes of ADHD including brain damage from birth, encephalitis, and genetic inheritance. A brief history of naming trends is outlined here below (Matson 1993).

- 1854 - “Fidgety Phil”
- 1902 - Brain damage syndrome (Still’s disease)
- 1934 - Organic drivenness
- 1941 - Organic behavior syndrome
- 1955 - Minimal brain damage syndrome
- 1957 - Hyperkinetic impulse disorder
- 1962 – Minimal brain dysfunction
- 1968 – Hyperactivity
- 1970 – Hyperkinetic syndrome
- 1975 – Hyperactive child syndrome
- 1980 – Attention deficit disorder (ADD)
- 1987 – Attention deficit hyperactivity disorder (ADHD)

After seeing the progression in names, one may understand that the disorder itself, definition of it, and causes for it have evolved over the past 150 years. The earliest report of any classification or naming of ADHD appears in 1854 as “Fidgety Phil.” A German physician named Hoffinan coined the term “Fidgety Phil” in 1854. He had a patient named Philip whom he could not quite describe, so he nicknamed him “Fidgety Phil” (Conners and Wells 1986). In Hoffman’s story, there was no official diagnosis for Phil, but he was significantly different than the other children. Phil had a difficult time sitting still, paying attention, and refraining from interrupting others. In 1902, George Still noticed other symptoms in patients and called this behavioral condition “brain damage syndrome” or “morbid defect in moral control.” At this time, the condition was more popularly called “Still’s Disease” rather than “brain damage syndrome.” It was Still’s belief that the syndrome stemmed from a “defect in moral control” (Still 1902). The behavior in children with defects of moral control included a broad impairment of intellect and behavioral excess. Furthermore, he believed that these children acted this way due to self-gratification. Some of the characteristics used to describe these children were passionateness, spitefulness, jealousy, dishonesty, mischievousness, lawlessness, shamelessness, destructiveness, sexual immorality, and viciousness (Still 1902). Still did not notice any damage to any specific area of the brain, but postulated that brain damage may exist in this syndrome. Therefore, he made two distinctions. The first group is described by symptoms linked with a previously known disease such as epilepsy, encephalitis, head injury, brain lesion, or mental retardation. The second group was
different in that there were no signs of disorder other than behavioral symptoms. He also noted that there was a possible relation to parental disorder in the second grouping (Still 1902), and he may have been the first researcher to relate a possible genetic component of this disorder, but had inconclusive research. Thus, the pertinence of familial transmission existed at the turn of the century. Still did have great motivation behind his studies, but the underlying cause of the disorder remained unknown.

In 1934, Kahn and Cohen called this disorder “organic drivenness.” They believed that the U.S. encephalitis epidemic in 1917 and 1918 brought about certain characteristics in survivors of the epidemic. The researchers observed that the primary characteristics of this disorder were general hyperkinesis, clumsiness of motor movements, and behavioral disinhibition, which refers to the inability to behave in a socially acceptable manner. They suspected that these characteristics came about because of damage to the brain stem, such as seen in encephalitis survivors. They also suspected that possible birth or prenatal injury could have caused this damage, but no conclusive evidence was found (Kahn and Cohen 1934).

In 1947, Strauss and Lehtinen coined the term “minimal brain dysfunction” to describe the disorder. In researching and studying this disorder, they came up with many descriptions of symptoms and disabilities inherent in these children. Their effort was to define handicaps in these children, rather than looking at the broad spectrum of symptoms. They also attempted to link the dysfunction to the encephalitis epidemic. The symptoms they noted were instability, impulsivity, lack of inhibition, and hyperactivity (Strauss and Kephart 1947). Excessive motor activity and distractibility with brain damage were noticed as symptoms also, but convinced the researchers of brain damage to
specific areas of the brain, such as the brain stem. In 1955, Strauss and Kephart revised the name of the disorder to “minimal brain damage syndrome” (Strauss and Kephart 1955). They called it a syndrome rather than a dysfunction. A dysfunction is defined as “abnormal or impaired functioning, especially of a bodily system or social group” (www.dictionary.com). A syndrome is defined as “a group of symptoms that collectively indicate or characterize a disease, psychological disorder, or other abnormal condition” (www.dictionary.com). They thought that a dysfunction was based on specific areas of the brain being damaged, versus multiple symptoms being grouped together to describe the disorder as a syndrome. They were the first to suggest a reduction in external stimulation, or environmental management to aid the troubled individual. Environmental management is an idea of controlling the variables surrounding an individual. One solution might be to place an individual in atmospheres conducive for good behavior or freedom to move around. Another solution might also contain reducing distractions. They found that environmental management was successful in helping children with this syndrome. This suggestion still remains in the current guidelines for controlling hyperkinetic behavior.

In 1957, Maurice Laufer and Eric Denhoff coined the term “hyperactivity syndrome.” These two individuals are recognized for this and followed the footsteps of Charles Bradley’s work at the Emma Pendleton Bradley Home, which was a place for research on this disorder and other behavioral disorders. Charles Bradley was a physician who had conducted previous research in this area. In 1948, these three researchers worked together and identified some of the criteria and characteristics that seemed feasible and reasonable including short attention span, mood lability, hyperactivity,
impulsiveness, and poor memory (Rosenfeld and Bradley 1948). Bradley, Laufer, and Denhoff also believed that the disorder arose from specific central nervous system damage. They attributed the damage to illness in infancy or hypoxia at birth. In 1957, after being inspired by Bradley’s early papers and research, Laufer and Denhoff renamed the syndrome “hyperkinetic impulse disorder.” They added higher than normal levels of motor activity, poor concentration, inability to delay gratification, irritability, fits of anger, low frustration tolerance, poor school performance, and explosiveness to this growing list of characteristics describing this behavioral problem. They found that the disorder was seen in infancy or early childhood and dissipated between ages 8 to 18. They also believed that this was, in part, a result of a damaged area in the diencephalon of the brain due to pre- or perinatal complications (Laufer and Denhoff 1957). Laufer subsequently published more works in 1970’s furthering the notions of brain dysfunction and procedures in diagnosis.

Research on this disorder continued into the 1960’s and indicated that brain damage and behaviorally disordered children did not go hand in hand. This led to renaming the disorder to “minimal brain dysfunction” by Clements and Peters in 1962. This term stayed around for a short period and it was accepted that children could have behavioral problems without any brain damage at all, but a specific dysfunction was too difficult to pinpoint. They stressed more importance on neurological dysfunction (Clements and Peters 1962). Further along in the 1960’s, Werry also tried to relate behavioral disabilities to physiological function. In 1968, he attempted to develop norms for evaluating excessive motor activity. He produced a quantitative scale, which was widely used, for assessing motor activity and monitoring treatment effects. Some of the
factors included in the scale included the response to medication, abnormalities in motor activity, and signs of behavioral disability (Werry 1968).

In the 1970’s, Cantwell established a genetic link to the disorder. His study included the parents of 50 hyperactive children and 50 control children. He concluded that there were increased rates of alcoholism, sociopathy, and hysteria in the parents of hyperactive children. He also reported that 10% of those parents were hyperactive children as well, showing a possible genetic transmission (Cantwell 1972). He noted no direct evidence to brain injury and concluded that the presence of this disorder in adults and children suggested familial transmission. In a study of 39 more children in 1975, he compared biological and adoptive parents and found a higher rate of behavioral issues within the biological transmission. He also reported that biological parents, who had behavioral issues such as hyperactivity, alcoholism, and other issues, were likely to transmit these problems to their offspring. With adoptive parents, he found no abnormalities in behavior that was transmitted to the children. This also showed that there was a likelihood of genetic transmission of this disorder, but was not fully conclusive (Cantwell 1975). Further in the decade of 1970, hyperactivity and delayed physical and mental maturation seemed to be apparent. Barkley found this through neurological and electroencephalographic examinations in normal and hyperactive children in 1981, but the results were still not conclusive. Research showed a delay in development both physically and mentally. There were difficulties because of diverse findings in research, but nonetheless, the theory remained that genetic transmission was a large reason for this disorder (Barkley 1981).
By this time, many researchers were beginning to understand that many factors were separate and could even be situation-specific. There were also discrepancies between measuring devices, suggesting factors other than hyperactivity being measured. A researcher named DeFilippis noticed that even though a single factor did not include all symptoms, many characteristics did cluster together, showing that there is correlation between certain symptoms. In 1979, he outlined inclusion and exclusion criteria for the disorder. The exclusion criteria were “unequivocal signs” of brain damage, soft neurological signs, and abnormal electroencephalograms. The inclusion criteria were based around inattention (DeFilippis 1979). The observed signs of inattention were mostly situation-specific, but included difficulty paying attention when necessary and difficulty listening to instruction.

In the 1980’s, research on ADHD continued, but there was a shift in view. More focus was placed on attention deficits, rather than hyperactivity, genetic transmission, or delayed maturation. In 1980, the American Psychiatric Association published a revised edition of the *Diagnostic and Statistical Manual of Mental Disorders III* (APA 1980). The American Psychiatric Association (APA) was a pioneer in the terminology of “attention deficit disorder.” Attention deficit disorder (ADD) began to be a more common diagnosis for this disorder. ADD focused on inattention and impulsivity and hyperactivity were secondary in this terminology. In this edition of the manual, hyperactivity was listed as a symptom of ADD, but it did not have to be present for a positive diagnosis. One of the criteria for ADD was an onset before the age of seven. Also, there were several exclusionary criteria identified, including such handicapping conditions as psychosis, sensory defects, neurological disease, and mental retardation.
This is a key point because these criteria showed that the disruptive behaviors existed aside from these conditions. This was the first major hinge upon the theory of no underlying mental condition determining causality in the disorder. The distinction between behavior not relating to brain damage and other issues such as the ones described above is critical because the focus is taken off of brain damage and placed on less physical issues. The behavioral problems are still apparent in the absence of any underlying conditions, as previously thought (APA 1980).

In 1987, the APA as part of the revised edition of the *DSM III* coined the current term of attention deficit hyperactivity disorder (ADHD) (APA 1987). Throughout this time research was conducted on many levels, but one researcher still tried to link brain damage to the disorder. Rutter was able to show that there is a link between birth complications and defects and ADHD. In 1989, he emphasized that behavioral defects and brain dysfunction were linked together and some of those effects came from birth complications. However, this research was inconclusive and instead helped to prove that this was still factor analytic and not predominant among all individuals (Rutter 1989).

ADHD has a long history and many changes are being made as research continues. A more narrow definition might be helpful, as some of these researchers thought, but the history of this disorder continues to be written and studied. A few diagnoses have described the behavioral issues as part of the disorder and contains a cluster of symptoms. Similarly inattentive, impulsive, and overactive children have been variously diagnosed as a result of these clusters of symptoms. There is an agreement that exists stating that there are clusters of behaviors and/or symptoms that coexist. The effort
in understanding the disorder still continues, but the current state of affairs, prevalence, and treatment options are still at hand.

III. PREVALENCE

The prevalence of ADHD is estimated at three to five percent of school-age children across the United States (APA 1994). There are other estimates as high as 10-20 percent of children being affected, but this is not a widely known or used statistic (Shaywitz and Shaywitz 1992). Variations in estimates could be based on differences in measurement techniques and tools. Although ADHD is seen across the globe, it is predominantly seen more in Western countries such as the United States, which may be due to diagnostic issues rather than clinical presentation. Countries with affluence like the United States may have more concerned parents or availability to visit a physician. In poorer countries, children may have ADHD, but are not diagnosed due to insufficient funds for physician visits or medication. Variability between countries across the world could be due to amount of research, amount of funding for physician visits or medication, or ways of diagnosis. Not much data has been presented for adults or adolescents, but a slight decrease is expected, although it is becoming more apparent that adults can have ADHD, which is usually the result of not being diagnosed during childhood (NIMH 2000). The specific age of onset may be different for each individual, but difficulties occur in diagnosis when a child is younger then five years of age. It is common to see hyperactivity or inattention in a toddler. As a result, not many diagnoses are made prior to age 5. Also, young children do not participate in many activities that require sustained attention. The average 2-year-old child is noted as being able to have an attention span
long enough to look through picture books. If a child is difficult to contain or moves excessively, specific attention may be given to the child to monitor the behavior further.

"As children mature, symptoms become less conspicuous" (APA 1994). Even with possible symptoms during the toddler years, a child may grow out of these symptoms or as referred to above, become easier to observe. In school-age children, these symptoms can cause difficulty in completing schoolwork and affect academic performance overall.

Physician visits concerning ADHD have increased dramatically since 1990. In a survey which involved children ages 5 to 18, the results showed an increase in physician visits. In 1990, 947,208 physician visits were recorded for ADHD diagnoses. In 1995, the number of visits increased to 2,357,833 (Robison et al 1999). It is quite possible that the number of visits to the doctor have increased even more dramatically since 1995. The mean patient age rose from 9.7 in 1990 to 10.8 in 1995, which is over one full year difference. In 1990, the percentage of office visits resulting in diagnosis was 1.1% and in 1995 it rose to 2.8% (Robison et al 1999), which represents an approximate 2.5 fold increase.

The disorder is seen less in females than in males. The male to female ratio ranges anywhere from 4:1 to 9:1 (APA 1994). However, there is evidence the frequency of female children being diagnosed with ADHD is increasing as indicated by a four-fold increase in the diagnosis of ADHD in girls between 1990 and 1995 (Robison et al 1999). One factor that may account for the disorder being diagnosed less frequently in girls versus boys is that girls may present themselves as predominantly inattentive. In fact, Silver (1999) reported that "Despite having attentional problems similar to those of boys with ADHD, girls with ADHD are less intrusive, and the show fewer aggressive
symptoms." Males are typically more hyperactive or impulsive and this reflects in their behavior, such as being overly aggressive or antisocial (Silver 1999). Males might disrupt class or other settings, so the behavior would be much more easily noticed. The ratio of male to female also varies depending on setting, such as general public or private clinics (Silver 1999). Other settings are school, work, home, and even extracurricular activities. Because of all of these variable situations, a range of ratios may exist. These different settings may affect the ratio due to the natural environment surrounding a child. Some children could be aggressive or inattentive at school, but that same child might not display these behaviors at home. The setting does not really affect the clinical diagnosis, but the child’s behavior can vary in different situations.

IV. DIAGNOSIS

The process used to diagnose ADHD has been is a topic of debate for a long time. Some individuals in the general public believe that ADHD may be over diagnosed, while others believe that it is under diagnosed. Part of the controversy stems from the fact that there is no objective test such as blood testing or biopsy analysis to verify the existence of the disorder. For this reason, the Diagnostic and Statistical Manual of Mental Disorders IV (DSM IV) has created very specific guidelines for diagnoses. The DSM has been published by the American Psychiatric Association (APA), which been revised on four different occasions due to new research and terminology. The APA consists of teams of many different researchers with a wide variety of educational backgrounds. The information used is considered to be the most reliable and accurate information possible. The primary criterion of ADHD diagnosis is a “persistent pattern of inattention and/or
hyperactivity-impulsivity that is more frequent and severe than is typically observed in individuals at a comparable level of development” (APA 1994).

There are five main criteria in a typical diagnosis, which are outlined in Appendix A. The five main criteria are symptoms of inattention or symptoms of hyperactivity/impulsivity, symptoms causing impairment before the age of 7, impairment obvious in at least two different settings (school, home work), clear evidence of clinically significant impairment in social, academic, or occupational functioning, and the symptoms do not occur in conjunction with another mental disorder. These criteria are included for the diagnosis of each individual.

To be diagnosed for ADHD, a person must possess six or more of symptoms related to inattention for at least six months, such as making careless mistakes, difficulty sustaining attention in tasks, does not listen when spoken to, does not follow through on instructions, difficulty organizing tasks, avoids activities that require sustained mental effort, loses things easily, easily distracted by extraneous stimuli, and is often forgetful. Also, a person must possess six or more hyperactive-impulsive symptoms for at least six months, such as fidgeting with hands or feet, leaves seat when expected to remain seated, excessive motor activity when inappropriate, difficulty engaging in activities quietly, is often “on the go,” talks excessively, blurts out answer prior to the question being finished, difficulty waiting, and interrupts or intrudes on others.

To be diagnosed, a person must be observed with these inattention or hyperactive-impulsive symptoms above, but also must be observed for more than six months at a time in at least two different settings, such as school, home, or work. There are three subtypes of ADHD based on the symptoms listed above to categorize a diagnosis: predominantly
inattentive – having six or more of the inattention symptoms for more than six months, but not more than six of the hyperactive-impulsive symptoms; predominantly hyperactive-impulsive – having six or more of the hyperactive-impulsive symptoms for at least six months, but not more than six of the inattention symptoms; and combined type – having six or more symptoms of both inattention and hyperactive-impulsive behavior for at least six months in two different settings (APA 1994). The diagnostic procedures require that children complete standard tests and for parents, and sometimes teachers, fill out standard behavioral surveys. Clinicians are strongly urged to use the criteria and the procedures outlined in the DSM IV to more accurately assess the child for this disorder as well as ensure that the appropriate course of action is taken to manage and treat ADHD.

Despite the effort to standardize the diagnostic procedures, concern is still expressed over the objectivity and validity of this disorder. As referred to above, there are multiple settings that a child may entertain behavioral problems. A physician may not be able to determine behavioral problems in a typical visit. Parents may not be able to accurately describe the child’s behavior on standardized surveys. Most of the questionnaires for parents are very specific. Each individual child may exhibit different behaviors in different settings like home or school. This creates a problem because there is no standardization for each individual behavior. A parent may not want to obtain an official diagnosis, so they may not be completely honest in a survey. A child may be frightened of a doctor. There are a number of other variables of the like contributing to a diagnosis. This is why a diagnosis for ADHD is subjective and should be handled very carefully and only after extensive evaluation.
V. TREATMENT OPTIONS

The most common method of treatment is medication. There are other forms of treatment such as behavioral modification, environmental management, exercise, and other alternative therapies, but medication is by far the most prevalent. One must remember that ADHD is not a disease with a known cure, but it is a disorder with specific ways to combat the symptoms. Historically ADHD has been treated with one of four classes of medications: stimulants, antidepressants, adrenergics, and antipsychotics.

Stimulant medications are the most commonly prescribed because they treat a large range of symptoms. Common stimulant medications are methylphenidate (Ritalin, Concerta), amphetamine (Adderall), dextro-amphetamine (Dexedrine) and pemoline (Cylert). Antidepressants are used to treat a few different symptoms and include imipramine (Tofranil), desipramine (Petrofrane), fluoxetine (Prozac), and buspirone (BuSpar). Adrenergics that are commonly used are clonidine (Catapres) and guanfacine (Tenex). The last class of medications is antipsychotics. Thioridazine (Mellaril), haloperidol (Haldol), and chlorpromazine (Thorazine) are the typical antipsychotics in use (www.uams.edu). More recently a new classification of non-stimulant drugs has become available. One of the newest non-stimulant medications is a non-addictive medication made by Eli Lilly called Strattera. This is also a medication that is supposed to last up to twelve hours.

For the purpose of this discussion, the focus of this section will be on stimulant medications - how they work, the benefits, and the side effects. Stimulants have been the most prescribed and researched drugs for this disorder. An abundance of research has been accumulated on the effects of stimulant medication on behaviors associated with
ADHD, particularly methylphenidate. This is likely due to the fact that stimulants have been used longer than the other classes of drugs. With stimulant medications, 70% of children studied show mild to moderate improvement in behavioral and parent ratings (Wilens et al 1992). The pharmacological treatment of stimulant medications works by decreasing the uptake of dopamine, which concentrates dopamine, and will then affect the sensitivity to dopamine in the brain. The efficacy of stimulant medication seems to be apparent, which is why they are so frequently used. Many different researchers have documented the short-term effects. The short-term effects are improvement in inattention, impulsivity, and hyperactivity. Stimulants are also shown to improve academic productivity, behavior, physical aggression, and social interaction (Swanson et al 1993).

However, despite the effectiveness of stimulant medication there are several untoward side effects. Most side effects are mild, but some serious side effects have been seen. One of the mild side effects is a decreased appetite. This usually is seen after taking the medication. It is recommended that medications be taken with or after meals (Barkley 1990). There is a mild weight loss that has been documented within the first few months of using medication. This is partially due to a decreased appetite (Reeve 1991). Insomnia or difficulty sleeping is also common due to the nature of stimulant medications. Various aches and pains, whining, moodiness, irritability, and crying are generally seen as well. Most of these symptoms go away after a few weeks, but if these symptoms persist, the physician should be consulted. About one third of children have increased hyperactivity or behavioral rebound anywhere from one to three hours after ingesting the medication. At this time, it could be the right time to take a small dose if effects are necessary throughout a day (Johnston 1988).
The serious side effects are still being researched, but these have all been seen on multiple occasions. A tic develops in nine percent of children on stimulant medication. A tic is an involuntary, non-purposeful, repetitive, muscle movement or vocalization such as eye blinking, utterances, facial grimacing, sniffing, snorting, coughing, or throat clearing. Most of the children with multiple tics see this discontinue in time, but in approximately one percent of subjects tics remain permanent (Lipkin 1994). There are other rare serious side effects such as hallucinations, slowed growth, increased heart rate, and increased blood pressure. To monitor these serious side effects, physician visits are recommended every three to four months. It is also recommended to be off of medication once per year to test the progress of the medication and see if it is needed any longer.

Stimulant medications usually absorb into the bloodstream within thirty minutes and the effects last anywhere from one to three hours (Pelham and Milich 1991). It is believed that a few of the new drugs being marketed are supposed to last up to 12 hours, requiring only one dose per day, but these drugs are very new and little research has displayed their effectiveness. Approximately two to six percent of all children are treated with some sort of stimulant medication and approximately sixty to eighty percent of children diagnosed with ADHD are prescribed stimulant medication (NIMH 2000). Treatment is always indefinite. A common belief used to be that children grow out of this disorder, but ADHD is still common among adolescents and is growing among adults (www.uams.edu).

Stimulant medications are used so frequently because the positive effects generally outweigh the side effects.

One research study examined the cardiovascular response to stimulant medication, specifically methylphenidate. Heart rate, blood pressure, and oxygen
consumption were measured in hyperactive children in this study. The hypothesis of this study was that methylphenidate would increase cardiovascular response.

Methylphenidate and a placebo drug were used among 27 hyperactive subjects and 23 control subjects. An increase in heart rate, systolic blood pressure, and mean blood pressure were found during rest, exercise, and recovery when using methylphenidate. These increases were not observed with placebo medication. Oxygen consumption was not altered from stimulant medication to placebo medication. The children that had been on medication prior to testing showed just as much exercise induced change as the control subjects did. This study showed an obvious increase in heart rate, systolic blood pressure, and mean blood pressure. These results lasted for four to five hours after ingesting the medication (Ballard et al 1976). This research was relatively new in 1976 and has shaped much research containing exercise since then.

Another study used subjects without ADHD. The study examined the effects of methylphenidate on heart rate, blood pressure, and plasma epinephrine. The study used four adult males and eight adult females. Methylphenidate was intravenously injected at 0.3 milligrams per kilogram of body weight. The research showed an increase in heart rate, systolic blood pressure, diastolic blood pressure, and plasma epinephrine. The research did not show an increase in plasma norepinephrine. This research was compared with research on caffeine and D-amphetamine cardiovascular effects. The results of methylphenidate versus the other two stimulants were somewhat different. D-amphetamine elevates plasma norepinephrine, systolic and diastolic blood pressure, but does not elevate heart rate or plasma epinephrine. Caffeine elevates plasma norepinephrine and epinephrine, systolic and diastolic blood pressure, and heart rate.
With caffeine, the heart rate rose only after an initial decline. Each one of these stimulants had somewhat of a different effect on cardiovascular response, showing that all stimulants do not have the same effects, especially on cognitive or behavioral functioning (Joyce et al 1984). This research examined normal individuals and supported evidence for methylphenidate to be used as an effective medication for hyperactive or impulsive behavior, but the problem is that the study examined normal individuals, which leaves the question of combating the problem in individuals with the disorder. Stimulant medications have obviously been proved to produce desired benefits that outweigh the risks previously mentioned. “Nine out of 10 children improve on one of the three stimulant drugs (Ritalin, Dexedrine, Cylert).” (NIMH 2000) Stimulant medications are effective, but what if these medications do not work? Other medications such as the antidepressants, andrenergics, or antipsychotics are being prescribed with caution. So many parents are afraid of the serious side effects of the stimulant medications, but there are also side effects of these other medications. Because of the effectiveness of stimulant medications, these other medications are secondarily used. Also, other alternative therapies are being used and researched in place of medication.

Environmental management, psychotherapy, cognitive-behavioral therapy, social skills training and support groups are some of the alternative strategies that can be employed to deal with children with ADHD (NIMH 2000). Environmental management works to reduce extraneous stimuli. This could be to remove a television, computer, or any other distracting object in an environment to help maintain the attention of an individual. The purpose of environmental management is to control every part of the individual’s environment to best captivate their attention.
Psychotherapy primarily works towards an individual accepting who they are and the disorder itself. Things like upsetting thoughts or bothersome occurrences are discussed in therapy sessions. Feelings are the primary symptom treated with psychotherapy. A therapist will discuss how to better handle emotions and manage the environment surrounding the individual (NIMH 2000).

Cognitive-behavioral therapy is also used to tackle immediate issues. This therapy is designed to change behavior in an immediate context, rather than understand feelings. An example might be thinking through specific tasks or organizing work. Another effort may be a reward system. The basic premise for this approach is a concept of mind over matter. If an individual can believe a specific way, the body and mind might not react with undesired symptoms (NIMH 2000).

Social skill training is another beneficial way for children to cope with ADHD. A therapist might model appropriate behavior techniques in specific situations and then discuss that behavior with the children. Sharing toys, waiting for a turn, asking for help, or responding to teasing might be some of the examples modeled. A child might learn to interpret a tone of voice or facial expression in order to modify their behavior. The primary goal of this approach is to develop tact and appropriate behavior responses in specific situations (NIMH 2000).

Support groups can also be formed for many reasons, but can be helpful in coping with this disorder. Many adults with ADHD and parents find support groups useful to deal with issues of the disorder. Support groups are geared towards sharing experiences in a group and learning from others' experiences. Parents can be trained for specific skills as well. Therapists give special classes to give parents special tools or techniques for
managing the child’s behavior. Environmental management is the key in this approach. A parent must remove a child from an agitating atmosphere or situation. A parent can also offer rewards and this is used in conjunction with the cognitive-behavioral approach. In a very basic sense, parents are taught to notice specific behaviors in children and combat them with positive techniques to sculpt the thinking pattern in children (NIMH 2000).

VI. EXERCISE EFFECTS

This section will include research on the effects of exercise on measures of behavior and the physiological effects of medication and exercise. Along with behavioral therapy, exercise is considered by some individuals to be a possible treatment for ADHD. The rationale underlying the therapeutic effect of exercise is that physical activity may alter the brain neurochemistry in a manner that is similar to the effect of stimulant medication. In a similar manner, the interaction of medication and exercise is of interest. Stimulants are known to increase heart rate and blood pressure, which is also an effect of exercise. Thus, the potential for a synergistic effect exists. There are studies that have examined the effects of exercise in conjunction with medication and some of the results will be discussed here. Typically exercise studies can be divided into the assessment of the acute effect of exercise or the chronic adaptation resulting form regularly performed exercise.

In a study by Tantillo et al. (2001) the rate of spontaneous eye blinks, the acoustic startle eye blink response (ASER), and motor impersistence among 8 to 12 year-olds were examined before and after an acute bout of exercise. This study included ten boys and eight girls. The children met the criteria for diagnosis of ADHD from the revised
edition of the DSM III and all were reported to be positive responders to stimulant medication. Subjects without ADHD were used for comparison purposes. The children underwent two different exercise session and one quiet rest session, which were all standardized to the extent possible. Exercise intensity was designed to evoke a brain dopaminergic response. The first bout of exercise measured a maximum oxygen uptake on the treadmill, the second bout was performed at a submaximal intensity of 60 – 75% of maximum oxygen uptake to test for if exercise responses were dependent upon medication dosage, and the third was a quiet rest session. Intensities were based upon the physical ability of each individual child. The results of the study showed that boys with ADHD had a faster blink rate after maximal exercise than at rest or submaximal levels. Girls and control subjects varied very little. The boys with ADHD had a decreased latency of the ASER after maximal and submaximal exercise. Girls with ADHD showed a decreased latency of the ASER, but an increase in the amplitude of the ASER after submaximal exercise. Improved performance on motor impersistence was seen among boys and girls with ADHD. Neither the ASER nor spontaneous blink rate in control children was affected by exercise. With all of that said, the study revealed a few differences between genders that were somewhat unexplainable. The study hoped to reveal somewhat of a dopaminergic effect from exercise on the ASER and spontaneous blink rate, showing an increased response in the brain. It is believed that the ASER and spontaneous blink rate are affected by dopaminergic neural activity. The study yielded very little conclusive data other than the rates mentioned above, which did not reveal much about the overall condition of the body post-exercise in ADHD children. The conclusion would be premature in stating that exercise definitely has a dopaminergic
effect and would increase dopamine receptor sensitivity, through studying spontaneous
blink rates and ASER. However, this study did uncover some gender differences and
opened a pathway to curiosity on gender differences and dopaminergic responses to be
studied with a different approach (Tantillo 2001).

In another study prior to the one above, the effects of exercise were studied on
cognitive performance in hyperactive and normal young boys. The study examined
whether the maximum cognitive performance of hyperactive boys occurs after a shorter
duration of exercise than normal boys. The researchers believed that there is an optimum
level of arousal at which a particular task is performed most efficiently. Another part of
the hypothesis was that hyperactive children are overaroused. The study included 7 to 10
year old boys. There were 31 hyperactive boys and 31 control boys. The exercise
performed was on a Monarch stationary bicycle ergometer for one, five, or ten minutes at
a rate of 18 to 20 kph. The targeted heart rate was 170, which was a work rate above the
level to induce exercise-related activity in the brain. The boys were tested directly after
exercise with a few standardized tests. Normal subjects scored higher on the tests than the
hyperactive boys, which was expected. There were not any significant differences among
separate trials. In the study, the researchers found little evidence to support any difference
between normal boys and hyperactive boys. They found that cognitive performance did
not vary directly with duration of prior exercise in control subjects or ADHD subjects.
The researchers did find that hyperactive boys performed worse in academic tests, which
further supported other research. This study revealed that cognitive performance varied
very little between control subjects and ADHD subjects. The study did not support the
hypothesis that hyperactive boys are overaroused. The main point of the study is that
cognitive performance did not vary from exercise, but was based out of attentional
deficits (Craft 1983). However, this research served as a catalyst for other research
studying cognitive functioning through other methods, with the research containing
different intensities, durations, and other standardized variables.

In a case study involving a young child the effect of antecedent exercise,
methylphenidate, and placebo were examined in relation to reducing hyperactive
behavior. The meaning of antecedent exercise is to intentionally decrease later disruptive
behavior; therefore, antecedent exercise is believed to have a moderate positive effect on
behavior. This boy was pre-school age and the goal of the study was to see if a
combination of any of these strategies reduced his hyperactive behavior. This study lasted
82 days. A questionnaire was used as a variable describing the boy’s symptoms. The boy
was prescribed 10 mg of methylphenidate to be taken daily at 11 AM. A placebo
medication was created for the study to be used on specific days of research. The
guardian monitored intake of the medications so that the research was standardized and
valid. Jogging was used as the antecedent exercise of choice. The boy exercised for 20
minutes at 65% to 80% of his maximum heart rate. To encourage his participation, he
was rewarded every five minutes. On the days the placebo was administered, the exercise
protocol did not change. The researchers found that exercise resulted in more hyperactive
behavior than using placebo medication and significantly more hyperactive behavior than
using methylphenidate medication alone. The study failed to show that exercise and
medication combined produced any more benefit than stimulant medication alone. The
study also failed at showing that exercise reduces hyperactive behavior, especially in this
young boy. One difficulty in this study was using a pre-school age child. Very little
research has documented specific side effects of medication or exercise for pre-school aged children. The researchers suggest “involvement in play-type activities without the sustained intensity of vigorous exercise might increase disruptive behavior” (Silverstein et al 1994). Since this was a case study on one individual, the scope may need to be broader to show any significant findings in the future.

Collectively these studies fail to show a significant benefit of using exercise to improve behavior in children with ADHD. However, there are sufficient limitations in the research design with respect to exercise protocols and behavior assessments to mitigate conclusions. In addition, there continues to be a wealth of anecdotal evidence that supports the concept that exercise may have a positive effect on the behavior in children with ADHD.

A second area of study with regard to exercise and children with ADHD centers upon the interaction between stimulant medication and the physiological responses to exercise. In the study by Ballard and Boileau (1976), the researchers examined cardiovascular responses to methylphenidate in hyperactive children. Out of the 27 hyperactive children and 23 control children studied, significant results were found. Methylphenidate was found to increase heart rate, systolic blood pressure, and mean blood pressure during rest and exercise. There were no significant differences measured in oxygen consumption though. The study also concluded that these effects were found each time the medication was taken and no tolerance was developed over time (Ballard et al 1976).

More recently, Mahon et al. (2003) tested the effects of stimulant medication on submaximal exercise responses in boys with ADHD. The study examined physiological
and perceptual responses during exercise. The study included twelve boys with ADHD. Two different trials were taken: one after taking medication during the normal morning hour, the next trial without morning medication. The study found that heart rates are higher on medication than off medication. Oxygen consumption, respiratory exchange ratio, ventilatory equivalent for oxygen consumption, and ratings of perceived exertion were not changed with medication treatment. The study also documented that medication dosage did not affect heart rate differences at separate workloads (Mahon 2003).

In a follow-up study of the one above, peak aerobic exercise capacity was examined in boys with ADHD. Eleven boys were used in this study and were treated with stimulant medication. A similar protocol was used in that one day was with medication and the next day was without medication. The study found that stimulant medication might help these children reach their full aerobic exercise capacity potential. Medication may help the children concentrate during exercise, which may be a factor in reaching full potential, but being on medication was seen to help the children reach their full potential for aerobic exercise (Mahon et al. 2003).

In another study conducted at Ball State University, high intensity exercise responses in boys with ADHD were researched. The goal of the study was to see if heart rate and ratings of perceived exertion was affected by stimulant medication during high intensity intermittent exercise. A scale called the OMNI scale was used to rate exertion levels. One day, the boys would come in after having a normal dose of medication. Another, they would come in without having medication. The boys performed at 90% of their peak work rate for thirty seconds at a time followed by thirty seconds of rest. The study found that heart rate and ratings of perceived exertion increased over time in both
ADHD subjects and control subjects, which was expected, but there were no significant results found between the days on medication and the days off of medication. The study did find that heart rate increases with intermittent exercise in boys with ADHD on medication versus off of medication (Mahon et al 2004). However, it appears that the increased heart rate while on medication was comparable to the heart rate observed in children without ADHD suggesting that the cardiovascular response to exercise is depressed in the absence of medication. This is consistent with observations that children with ADHD have a depressed catecholamine response to exercise (described in the next paragraph).

Because ADHD is related to a catacholaminergic deficiency in the central nervous system, it has been hypothesized that systemic measures of catecholamine activity also is deficient in children with ADHD. To test this hypothesis, Wigal et al. (2003) examined venous blood concentrations for epinephrine, norepinephrine and dopamine in children with and with ADHD. The goal of this study was to find any differences in catecholamine response between control subjects and ADHD-diagnosed subjects, possibly showing a blunted response accounting for adverse behaviors in ADHD subjects. The children diagnosed with ADHD were newly diagnosed and had not been treated with any medication prior to the study. The study included all boys of similar age and growth. Intensity and duration were based on the capacity of each individual. The study contained 10 ADHD subjects and 8 control subjects. The subjects exercised on two separate occasions within one week on an electronically braked cycle ergometer. The exercise was designed with interval bouts rather than prolonged exercise because of physical tolerance. Blood samples were taken via a venous catheter before exercise, during the last
tenth of exercise, 30 minutes after exercise, and 60 minutes after exercise. No significant differences between control subjects and ADHD subjects were found in maximum oxygen uptake, work rate, or lactate threshold. The study found a blunted lactate response in the ADHD children. The study also found a difference in catecholamine response between ADHD subjects and control subjects. Epinephrine and norepinephrine values did increase in both populations, but the response in ADHD subjects was significantly blunted although the work performed was no different. The amount of dopamine circulating increased in control subjects, but did not in ADHD subjects. ADHD subjects showed a lower lactate response than control subjects. These results give support to the theory of a blunted catecholamine response circulating in ADHD children, showing a dopamine deficiency of some sort, believed to be actual dopamine levels in the brain during this study. The study concludes that the catecholamine response is blunted in pharmacologic, physiologic, and cognitive challenges. This study does have implications for behavior because of the blunted catecholamine and dopamine response. These are two chemicals that deal with an individual’s behavior. The study obviously concludes that catecholamine response is blunted in children with ADHD, but the question remains as to how all of this data ties into combating ADHD. It is possible that part of the problem with ADHD is a blunted catecholamine and dopamine response and that having medication or other remedies to combat this could be very useful, but this study did not support exercise as being one of those ways to increase catecholamine response in ADHD children, which means there is no behavioral benefit from exercise. Research on the effects of exercise in ADHD children is still being performed and it is expected that many other findings will be published, but as for now, there is a lack of clarity and
supporting evidence to show that exercise alone is a sufficient remedy for ADHD behavior (Wigal et al 2003).

Knowing the results of the aforementioned research, many questions still exist as to what exactly is happening in the body with medication, exercise, or even on a general note. ADHD is a disorder that is being researched in many different ways. One of those ways is exercise and different hypotheses in what is really going on neurochemically and if exercise is an effective treatment in the disorder. The research would show that it is possible to help behavioral and cognitive functioning, but the evidence is not substantial enough to say that it is a significant treatment option. However, exercise can be very helpful for children physiologically, whether they have been diagnosed with ADHD or not.

VII. CONCLUSION

After reviewing the history, prevalence statistics, diagnosis trends, medication facts, and effects of exercise on ADHD, there seems to be an abundance of information on ADHD. The problem is that this information barely scrapes the surface of all the research that has been presented and published. There are many more articles, books, videos, and journals to be published as more research occurs. This disorder is one that is constantly being refined by definition. Many other unique characteristics of individuals that are diagnosed are being found through this research. There is a repetitive cycle of research on the horizon because as more studies are being conducted, more secrets are being uncovered, and more curiosity is arising in researchers. This review of research has
served as an educational tool and hopefully will educate any reader on the disorder of ADHD.
APPENDIX A

Criteria for Diagnosis of ADHD

A. Either (1) or (2):

(1) six (or more) of the following symptoms of inattention have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Inattention

(a) often fails to give close attention to details or makes careless mistakes in schoolwork, work, or 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 (not due to oppositional behavior or failure to understand instructions)
(e) often has difficulty organizing tasks and activities
(f) often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)
(g) often loses things necessary for tasks or activities (e.g. toys, school assignments, pencils, books, or tools)
(h) is often easily distracted by extraneous stimuli
(i) is often forgetful in daily activities

(2) six (or more) of the following symptoms of hyperactivity-impulsivity have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:
**Hyperactivity**

(a) often fidgets with hand or feet or squirms in seat

(b) often leaves seat in classroom or in other situations in which remaining seated is expected

(c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)

(d) often has difficulty playing or engaging in leisure activities quietly

(e) is often “on the go” or often acts as if “driven by a motor”

(f) often talks excessively

**Impulsivity**

(g) often blurts out answers before questions have been completed

(h) often has difficulty awaiting turn

(i) often interrupts or intrudes on others (e.g. butts into conversations or games)

B. Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age 7 years.

C. Some impairment from the symptoms is present in two or more settings (e.g. at school or work and at home).

D. There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.
E. The symptoms do not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder and are not better accounted for by another mental disorder (e.g. Mood Disorder, Anxiety Disorder, Dissociative Disorder, or a Personality Disorder).

Code based on type:

314.01 – Attention Deficit/Hyperactivity Disorder, Combined

Type: if both Criteria A1 and A2 are met for the past 6 months

314.00 – Attention Deficit/Hyperactivity Disorder,

Predominantly Inattentive Type: if Criteria A1 is met but Criteria A2 is not met for the past 6 months

314.01 – Attention Deficit/Hyperactivity Disorder,

Predominantly Hyperactive-Impulsive Type: If Criteria A2 is met but Criteria A1 is not met for the past 6 months.
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