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Attention Deficit Hyperactivity Disorder Linked to Lead: A Preventable Exposure with Life Time Consequences.

Marchee Madonna Briant

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Master of Public Health Thesis

ATTENTION DEFICIT HYPERACTIVITY DISORDER LINKED TO LEAD:
A PREVENTABLE EXPOSURE WITH LIFE TIME CONSEQUENCES

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2005
Lead linked to Attention Deficit Hyperactivity Disorder:
A Preventable Exposure with Life Time Consequences

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B.S., University of Maryland, University College, 2000

A Thesis
Submitted in Partial Fulfillment of the
Requirements for the Degree of
Master of Public Health
at the
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2005
Acknowledgments

Much gratitude is owed to the people who helped me during the different phases of this endeavor. Special thanks to Dr. Timothy Morse, Dr. John Meyer and Professor Paul Schur, whose input, consideration, and flexibility allowed this project to be a valuable learning experience.

True appreciation is owed to my family and friends for their patience and constant support for without their encouragement the completion of this degree would not have been possible.

A particular thank you to my phenomenal husband Bill – your love and support combined with your uncanny ability to recover my lost documents kept me sane on a daily basis.

Ultimately, I am most grateful to my parents for instilling in me a love of learning and the stubbornness to persist through trying times. It seems unnecessary to add that this work is dedicated to my father, Eddie Mitchell, Sr. (1932-1996).
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Introduction

This thesis addresses the link between lead exposure and Attention Deficit Hyperactivity Disorder (ADHD). Interest in this subject stemmed from the author’s exploratory research project on the causes of increased diagnosis of ADHD and environmental lead exposure. It will first provide a description of the problem: then a definition of ADHD, the etiology, the association of lead poisoning and cognitive impairment, the public health impact of ADHD, and societal cost/political implications.

Methodology

A systematic review of observational studies of blood lead concentrations, retrospective studies of lead in cord blood samples and prospective studies of birth cohorts was conducted to obtain the data herein utilizing PubMed and FastSTATs.

Definition of ADHD

ADHD, also known as Attention Deficit Disorder (ADD), is the most commonly diagnosed mental health condition in American children today and may be underestimated by school and public health officials (National Center on Birth Defects and Developmental Disabilities, 2002). The Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV) defines the essential feature of ADHD as 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 (Appendix V).

ADHD may be accompanied by learning disabilities, depressions, anxiety, conduct disorder, and oppositional defiant disorder. The etiology of ADHD is unknown, and the disorder may have several different causes (Agency for Health Care Policy and Research, 1999).
In the past decade, there has been a tremendous upsurge of scientific and public interest in ADHD and most recently, a potential link suggested between ADHD and lead exposure (NIH, 2003, Safe Child net, 2003). The National Center for Health Statistics estimates that almost 5 million children 3-17 years of age have been diagnosed with a learning disability (National Center for Health Statistics, 2002)

The History of Lead

Lead is a naturally occurring, malleable bluish-gray metal that is found in small quantities in the earth’s crust and has been used almost since the beginning of civilization. Lead is a versatile metal and its documented use dates back to prehistoric times. Because of the many industrial applications that have brought about its wide distribution, lead is ubiquitous in the environment and as a result, all humans have some level of lead in their bodies. There is a long history of public exposure to lead in food and drink. Lead poisoning resulting from the use of lead in water pipes and earthenware containers used in wine storage is documented back to Roman times (Millstone, 1997).

The oldest known lead object was a statue excavated in Turkey and dated somewhere around 6500 B.C. Lead objects have been found in Egyptian tombs and in ancient Syria. Lead was used in rods and in pieces as a means of currency.

World production of lead 4,000 years ago has been estimated at 160 tons per year; 2,700 years ago, it was 10,000 tons per year; and during the Roman Empire, lead production increased to 80,000 tons per year (Lin-Fu, 1985). After its discovery around 3500 B.C., vast amounts of lead began to be mined and smelted in both southwest Asia and Europe, mainly for its utility in building.
However, despite its wide usage, this dull metal remained in relative obscurity until the Romans began large-scale use of it in their elaborate aqueduct systems. It is believed that 12,000 tons of lead was used in the construction of a single siphon unit in the greatest aqueduct at Lyons. Both the Romans and the Greeks found many applications for this useful metal (Lin-Fu, 1985).

The use of lead in the aqueducts carried over into the Middle Ages when the metal found wide use as a building material. Today, lead remains an important metal in construction. The problem with lead is that once it is mined, processed, and introduced into the environment, it is a potential problem without end. No known or foreseeable technology will destroy it or render it harmless. Nearly all of the lead in the environment is due to human activities (Chambers, 2002).

The magnitude of the problem of lead toxicity from its use and misuse in certain cultures has caused historians to speculate that the decline of both the Roman Empire and the Shan Dynasty in China may be linked to lead poisoning. Since both civilizations found lead to be such a valuable metal in building their vast empires, it was used for constructing aqueducts, bridges and buildings. Lead was ever present in the environment (Chambers, 2002).

Centuries of mining, smelting, and use have released millions of tons of lead into the environment. Since its discovery about 5,000 years ago, and particularly since the Industrial Revolution of the mid-18th century, there has been a steady increase in lead production and the dispersion of this toxic metal into the environment, thus increasing the potential for occupational and residential exposures.
Lead’s versatility, as well as the favorable physical and chemical properties, account for its extensive use. Much of its usefulness is due to its plasticity and softness. Lead can be rolled into sheets, which can be made into rods and pipes. It can be molded into containers and mixed with other metallic elements.

Lead has been used in building construction, especially roofing, cornices, electrical conduits, and water and sewer pipes (Chambers, 2002). Studies of the lead concentration of snow sheets in the Greenland ice cap dated by carbon 14 determination, extending from 800 B.C. to recent decades, have demonstrated a sharp increase in the lead content of sheets since 1750, the beginning of industrialization. The lead concentrations of the snow layers tripled in the second half of the 18th century and again tripled between 1935 and 1965. In 1965 the lead concentration of Greenland snow sheets was about 400 times that found in 800 B.C. (Chambers, 2002).

Lead Exposure

The use of lead in our most recent history is also well documented. Human exposure to lead represents a serious environmental health problem in many urban areas. Studies have found a statistically significant connection between the exposure of people to lead and reductions in their intellectual performance measured by IQ (The International Bank for Reconstruction and Development, 1999).

Lead has been used in paints to ensure that paint adheres better, dries more quickly, and resists chipping and peeling better. It has been used in the solder of pipes as a bonding agent. That lead can then leach from the pipes into drinking water (Kitman, 2000). Houses that contain plumbing with lead or lead solder can pose a risk for lead poisoning, as lead is tasteless, odorless and cannot be seen.
The largest source of lead in the atmosphere has been from leaded gasoline combustion, but this problem has decreased considerably with the phase out of leaded gasoline in the United States. However, children in developing countries may be particularly vulnerable to exposure because they spend a significant part of their time on the streets, and often lack proper nutrition that increases their susceptibility to lead poisoning. Decision-makers in an increasing number of countries have recognized that eliminating the use of lead additives in gasoline is a cost-effective way of reducing this threat (The International Bank for Reconstruction and Development, 1999). Lead additives in gasoline have been used since the 1930’s. Lead was originally added to gasoline as a lubricant for motor vehicles by protecting exhaust valves from excessive wear and to boost octane levels. Lead has also provided lubrication of the engine valve seats, allowing automobile manufacturers in the past to use low-grade soft metals. Due to the advancement of automobile technology, and the introduction of catalytic converters, however, most major manufacturers started to produce automobiles with hardened valves during the 1970’s and 1980’s. Lead was outlawed as an automotive gasoline additive in the United States in 1986 – more than 60 years after its introduction – to enable the use of emissions-reducing catalytic converters in cars.

Thousands of tons of lead have been removed from the environment as a result of its removal from automotive gasoline. Lead is not naturally occurring in gasoline; it is only present in gasoline if it is placed there (Kitman, 2000). Other airborne sources include the combustion of solid waste, coal, and emissions from stationary or point sources such as iron and steel production, and lead smelters.
Food, water and soil can also contain lead. Prior to regulatory changes in gasoline, leaded combustion from the roadway was deposited in the soil and retained by some crops, particularly leafy vegetables.

Lead doesn’t break down in soil, and it remains for long periods, clinging to organic matter. Soil-lead concentrations in large cities are 10 to 100 times greater than samples taken from smaller cities (World Health Organization, 2003). Lead dust moves through the environment and will eventually rest on the soil. Therefore, drinking water and foods grown in a residential garden may contain lead (Kitman, 2000).

Leaded gasoline was banned in the United States in 1978. In the United States, with the ban of lead in paint and gasoline, the average blood lead level for persons 1 to 74 years dropped from 128 µg/dL in 1980 to 2.9 µg/dL in 1991 (Needham & Cloutman, 1999). However, soil contaminated from automobile exhaust is still a possible source for exposure. During the 1960’s, the United States began reducing the use of lead in paint, the use of lead has not been completely banned, although the percentage of lead in paint has been significantly reduced (Consumer Product Safety Commission, 1996). However, the interiors of houses built prior to 1950 were painted with lead-based paint. Flaking paint, paint chips and weathered paint powder are a major source of exposure for children. Although many cases of lead poisoning were reported in the United States in the early 20th century, it was not until the 1950’s that health officials began to link the cause of lead poisoning to housing with deteriorated lead-based paint.
Among the first cities in the United States to ban the use of lead-based paint in the interiors of homes were Baltimore, Chicago, Cincinnati, Jersey City, New Haven, Philadelphia, St. Louis, Washington D.C., and New York. In mid 1955, the paint industry adopted a voluntary standard limiting the use of lead in interior paints to no more than 1% by weight of nonvolatile solids (Connecticut Department of Public Health, 1999). Lead-based paint in good condition is not usually hazardous but peeling, chipping, chalking or cracking lead-based paint is a hazard and needs immediate attention. Plastic window blinds, candlewicks, costume jewelry and children’s knapsacks have been found to contain high levels of lead (Sanborn, 2002). The introduction of lead into the human environment has recently decreased in part because of public health campaigns to educate the public about the dangers of environmental lead exposure and the de-leading of gasoline (Jensen, 2000; Sanborn, 2002).

**Lead Poisoning**

In 1971, the federal government enacted the Lead-Based Paint Poisoning Prevention Act (LBPPPA), which, most importantly, required the Secretary of Health and Human Services to prohibit the use of lead-based paint in residential structures constructed or rehabilitated by the federal government or with federal assistance in any form (Connecticut Department of Public Health, 1999). Lead-based paint is defined as paint containing more than 1% lead by weight. The LBPPPA also authorized a national program to encourage and assist states and cities to conduct mass screening programs to identify children with elevated lead levels.

The symptoms of lead poisoning often go unrecognized and some effects of lead exposure appear to be long-lasting and irreversible.
Lead is a poison that affects nearly every system in the body. Historians have shown that knowledge of the dangers of lead poisoning to workers and children can be traced back to the 19th century and in the early part of the 20th century a broad scientific literature on the subject accumulated in Australia, England, and the United States (Sanborn, 2002).

American workers in the pigment manufacturing, battery, painting, plumbing, ceramics, pottery, and other industries were routinely exposed to high concentrations of lead. Children from all social and economic levels can be affected by lead poisoning, although those living at or below the poverty level are at highest risk. Children living at or below the poverty level often live in older housing where the interior of the dwelling may be painted with lead based paint, the houses may have chipping or flaking paint, further some inner city playground equipment may be painted with lead based paint. The diet of lower SES families don’t include fruits, vegetables and dairy as a result of poor dietary habits the aforementioned children are at higher risk for lead exposure and lead poisoning (Sanborn, 2002).

Young children absorb approximately 50 percent of the lead to which they are exposed, whereas, adults absorb approximately 10 percent. Children’s bodies are much more receptive to lead, thus creating a more efficient and thorough process of absorption (Needham & Cloutman, 1999). A child does not need to eat loose paint chips to be exposed to the toxin; normal hand-to-mouth behavior, coupled with the presence of lead dust in the environment, is the usual method of poisoning (Needham & Cloutman, 1999). Children’s smaller body size and developing systems place them at greater risk than adults for complications resulting from exposure to lead.
Because they are smaller, children receive higher doses of toxins per pound of body weight. Pound for pound, children drink more water, eat more food, and breathe more air than adults. Many organ systems in young children, such as the nervous system and the lungs, undergo rapid growth and development in the first years of life. It is during these years of development, that children’s organ systems are especially vulnerable to injury (Agency for Toxic Substances and Disease Registry, 2003).

High levels of exposure to lead can cause damage to almost all organs and organ systems, most importantly the central nervous system. Blood, brain and kidney damage have been reported at blood levels of 100 µg/dL blood in adults and 80 µg/dL blood in children (Lewendon, 2001). Gastrointestinal symptoms, such as colic, have been noted in acute exposures at blood lead levels of approximately 60 µg/dL blood in adults and children. Death from lead poisoning may occur in children who have blood lead levels greater than 125 µg/dL. Children up to three years of age absorb lead 5 -10 times more effectively than adults, thereby making children more vulnerable to lead related health problems (Sanborn, 2002).

Ancient Greeks were the first to write about lead poisoning. They identified a link between the ingestion of lead and delays in neurological development in children. However, they didn’t discontinue the use of lead. For the majority of its long history, lead had not been suspected as a hazard (Lin-Fu, 1985). In Australia, lead poisoning in children was first reported in 1892 (Gibson, 1904). Over the years, doctors have utilized lead to treat various diseases.
A medical dictionary dated 1945 suggested that lead dissolved in a mild acid such as vinegar might be used to cure sores or skin diseases (Lin-Fu, 1985). Others claimed that lead therapy could cure consumption, diabetes, dysentery, and epilepsy.

Whatever the source, lead is readily inhaled or ingested into the body and can be found in the blood and soft tissue and eventually in long-term storage depots in bone, where it can remain for decades. When lead is ingested the body mistakes it for calcium (Child & Family Resources, Inc. 2003). Diagnosing children with elevated lead levels poses a complex clinical problem. Early signs of lead toxicity can go unnoticed and often manifest as subtle neurobehavioral changes that affect social interaction.

This has prompted physicians to consider testing for lead exposure in children who present with growth failure, behavioral disorders, hearing loss, speech, language and attention deficits, developmental delays, microcytic anemia or sleep problems (Sanborn, 2002). Obtaining a blood level is the only valid and reliable measure of current lead exposure and during the summer, when lead exposure tends to peak, is the optimal season to test a child’s lead level.

The Centers for Disease Control and Prevention (CDC) tracks children’s blood lead levels (BLLs) in the United States using three methods: (1) nationally representative surveys (2) state and local surveillance and (3) local prevalence surveys (Centers for Disease Control and Prevention, 1997).

Because lead exposure in children varies among populations and communities, all three approaches are needed to understand the burden of elevated BLLs among young children across the United States.
Utilizing the three data sources enables the CDC to describe children at risk and to measure trends in BLLs among young children. Special attention is given to BLLs $\geq 10$ μg/dL.

Many studies point to a link between BLLs $\geq 10$ μg/dL and harmful health effects, in particular learning disabilities and behavior problems. The Department of Health and Human Services’ Healthy People 2010 initiative has set a national goal of eliminating BLLs $\geq 10$ μg/dL among children aged 1-5 years by 2010 (Centers for Disease Control and Prevention, 2003).

The National Health and Nutrition Examination Surveys (NHANES) conducted by CDC’s National Center for Health Statistics have been tracking BLLs since the 1970s. These national surveys are designed to estimate BLLs only at the national level and not at the state or local levels. NHANES has documented a substantial decrease in BLLs among young children (National Center for Environmental Health, 2004). The NHANES II 1976-1980 reported a geometric mean BLL of 15μg/dL among children aged 1-5 years during the four year time period.

The most current NHANES (1999-2000) data show that geometric mean BLLs continue to decrease in young children. BLLs $\geq 10$ μg/dL were estimated for 2.2% of children aged 1-5 years according to NHANES 1999-2000, dramatically lower than the 88.2% for the 1976-1980 (Table 1). The 2.2% estimate translates to 434,000 children with a 95% confidence interval (CI) from 189,000 to 846,000 children (Table 1). Future NHANES should help confirm the downward trend shown in Table 1. Despite the overall decline in average BLLs, the risk for elevated BLLs in children tested remains high in some counties and varies greatly among and within states.
### Table 2 - Race, Ethnicity, and Poverty Status of Connecticut Children <6 Years of Age (2000 Census); % Children <6 Years of Age

<table>
<thead>
<tr>
<th>Location</th>
<th>% Black</th>
<th>% Hispanic</th>
<th>% Children &lt;6 Below Poverty Level</th>
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<tr>
<td>Connecticut</td>
<td>20.6</td>
<td>25</td>
<td>10.9</td>
</tr>
<tr>
<td>Hartford</td>
<td>71</td>
<td>88.2</td>
<td>40</td>
</tr>
<tr>
<td>Bridgeport</td>
<td>63</td>
<td>68.2</td>
<td>24.5</td>
</tr>
<tr>
<td>New Haven</td>
<td>83.2</td>
<td>54.7</td>
<td>21.4</td>
</tr>
<tr>
<td>Waterbury</td>
<td>37.9</td>
<td>59.3</td>
<td>26.3</td>
</tr>
<tr>
<td>Stamford</td>
<td>30</td>
<td>33.3</td>
<td>9.4</td>
</tr>
</tbody>
</table>

This variation most likely reflects geographic variation in the prevalence of risk factors for elevated BLLs such as residence in older housing and poverty.


<table>
<thead>
<tr>
<th>Survey</th>
<th>Prevalence of BLLs ≥10 µg/dL (%)</th>
<th>Children with BLLs ≥10 µg/dL</th>
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<tr>
<td></td>
<td>(95% CI)</td>
<td>No. (95% CI)</td>
</tr>
<tr>
<td>1976–1980</td>
<td>88.2 (83.8–92.6)</td>
<td>13,500,000 (12,800,000–14,100,000)</td>
</tr>
<tr>
<td>1988–1991</td>
<td>8.6† (4.8–12.4)</td>
<td>1,700,000 (960,000–2,477,000)</td>
</tr>
<tr>
<td>1991–1994</td>
<td>4.4 (2.9–6.6)</td>
<td>890,000 (590,000–1,330,000)</td>
</tr>
<tr>
<td>1999–2000</td>
<td>2.2** (1.0–4.3)</td>
<td>434,000** (189,000–846,000)</td>
</tr>
</tbody>
</table>

Source: National Health and Nutrition Examination Surveys (NHANES).

Note: In 1991, NHANES III Phase 1 was completed and Phase 2 was begun.

* Estimated number of children aged 1–5 years with BLLs ≥10 µg/dL divided by estimated population of children aged 1–5 years.
† CDC has determined BLL ≥10 µg/dL is a level of concern.
§ Confidence interval.
‖ Estimate differs slightly from that published previously because of updates in coding and weighting of survey data.
** Data for 1999–2000 are highly variable (relative standard error >30%).

The NHANES reported a decrease in the number of children reported with confirmed elevated BLLs from 130,512 in 1999 to 74,887 in 2001. However, the Healthy People 2000 national goal to eliminate BLLs >25 µg/dL among young children by 2000 was not achieved, and tens of thousands of children remain exposed to lead (MMWR-Center for Disease Control, 2003).

Utilizing urine to measure lead levels is not recommended (Sanborn, 2002). However, recently hair and bones have been used as reliable sources to measure long term lead
exposure. The half-life of lead in the blood is thirty days, so a blood level is useful if the exposure occurred within three months. If an exposure occurred prior to that time, a zinc protoporphyrin test (half-life 68 days) may be helpful but sometimes yields false positive test results in a child with iron deficiency anemia.

It is estimated that the prevalence of elevated lead levels above the 10 μg/dL blood lead level, children in the United States is 4%-5%, which is comparable to Canada which has neither universal nor targeted screening for childhood lead exposure (Sandborn, 2002).

**Lead Burden**

The greatest problem remains the presence of many older homes with deteriorating lead-based paint. Homes built between 1900 and 1950 are most likely to contain lead-based paint in the interiors. Those built from 1950 through 1978 may contain lead-based paint, especially on exterior surfaces. The older the home, the more likely it is that lead-based paint was used. A few homes built subsequent to the 1978 ban on leaded paint may even contain this paint, because products remaining on store shelves were sold and used. Chipping, flaking, and chalking lead-based paint contribute to the environmental dust found in houses. Although new layers of paint may cover old lead-based ones, all layers are involved when deterioration occurs (Needham & Cloutman, 1999).
Figure 1. Number of pre-1950 housing units and children with confirmed elevated blood lead levels (EBLLs) greater than or equal to 10 μg/dL.

*Arkansas, Arizona, Delaware, District of Columbia, Hawaii, Montana, New Mexico, New York City, Utah, Vermont, and Wyoming.*
The Center for Disease Control and Prevention (CDC) has repeatedly lowered the acceptable blood lead level, now set at 10 µg per deciliter blood. The median blood lead level in US children has fallen from 15 µg per deciliter blood (0.724 µmol per liter) in 1978 to 2 µg per deciliter blood (0.097 µmol per liter), since the removal of lead from gasoline. Various other regulations such as; the reduction of allowable percentage of lead in paint, stringent regulations pertaining to pre 1950 housing, lead removal abatement, and oversight by the US Consumer Product Safety Commission have resulted in a triumph for public health – a reduction in BLLs in children.

Canadian Lead Prevention Campaign

In 1991, American public health authorities launched a population-wide prevention campaign aimed at eliminating childhood lead poisoning by the year 2000. The extent of the prevention campaign elicited various reactions in Canada, where health authorities had not considered that poisoning constituted a public health problem for Canadian children. As a result Health Canada has promoted awareness of issues concerning lead and health by educating the public, health professionals, and industry. Health Canada, in partnership with various groups, has released many publications on topics such as lead and home renovations and lead risks associated with arts and crafts.

Canada’s Lead Risk Reduction Strategy for Consumer Products was implemented to protect children from exposure to lead through consumer products. Under the Canadian Hazardous Products Act and Regulations specific products are regulated for lead content: paints, enamels and other liquid coating materials, decorative coatings on pencils and artists’ brushes, liquid coatings on children’s furniture and other articles intended for
children’s, toys, equipment and other products for use by a child in learning or play, glazed ceramics and glassware, and kettles.

In 1990, the Gasoline Regulation Act resulted in the phasing-out of leaded gasoline in Canada. Other provisions under the Act have reduced air emissions of lead from major industrial sources (Health Canada, 2005)

**Lead and the Environment**

Sustained use of large quantities of lead over many years has resulted in extensive environmental contamination. Lead is still a major risk factor and its effects are mainly concentrated in developing countries (Lovi, 1998). Due to insufficient evidence it is hard to ascertain the burden of disease due to environmental lead exposure. It is likely that the exact burden is underestimated because of a lack of data; the exclusion geographical “hotspots”; the adoption of conservative assumptions; and because a number of health outcomes and social consequences of are difficult to ascertain (Walsh, 2001). Because lead does not decompose or break down into smaller particles, lead deposited in the past is still present in the soil. Coupled with deteriorating lead-based paint falling from the exterior of houses, soil has become a significant pathway by which lead poisoning can occur (Needham & Cloutman, 1999).

Playground soil could possible contribute to the lead exposure of young children. Lead-contaminated dust from old playground equipment painted with lead based paints and soil previously contaminated by automobile exhaust can be tracked into the home. Although lead occurs naturally in small quantities in the earth’s crust, by far the greatest risk of exposure to lead comes from man-made processes and products.
Today, the principal industrial use of lead is in the manufacture of electrical storage batteries. Other current uses include the production of ammunition, various chemicals, and sinkers for fishing lures. Family members engaged in the aforementioned occupations provide another route of exposure by transporting lead dust home on their work clothes and shoes or inside work vehicles.

The use of lead in paint additives, gasoline additives, solders, and pipes has been reduced substantially or eliminated (Connecticut Department of Public Health, 1999). Nevertheless, old installed products or residuals from their use remain in the environment. There are three major sources of lead exposure: (1) lead-based paint, (2) urban soil and dust (deposits from paint, gasoline, and industrial sources), and, (3) drinking water, primarily from lead solder, brass fittings and fixtures, and service lines. Other sources can result in high exposures in individual cases. Contributions from other sources add to the problem and are, therefore, cause for concern (Connecticut Department of Public Health, 1999).

**ADHD Burden**

ADHD is a neurobiological disorder which according to the Surgeon General’s report on Mental Health afflicts between 3% and 5% of school aged children. ADHD was previously believed to be more common in boys than girls. However, a recent study that reviewed a national data source reported a 2.7-fold increase in a diagnosis of ADHD among girls between 1991-1992 and 1997-1998 (Robinson, 2002). ADHD often develops before age seven, but it is most often diagnosed between the ages of eight and ten.
According to a report issued by the Agency for Health Care Policy and Research, one-third of children diagnosed with ADHD also qualify for a diagnosis of oppositional defiant disorder. One-fourth of children diagnosed with ADHD also qualify for a diagnosis of conduct disorder.

Less than one-fifth of children with ADHD also have a depressive disorder. More than one-fourth of children with ADHD qualify for a diagnosis of anxiety disorder and almost one-third of children with ADHD also have more than one comorbid condition. Overall, the prevalence rates of comorbid ADHD are high. Estimates of the prevalence rates of various comorbid conditions in children with ADHD range from 12.36 percent (learning disorders) to 35.15 percent (conduct disorder) (Agency for Health Care Policy and Research, 1999).

Nearly eight million children in the U.S. suffer from mental disorders, and ADHD is one of the more prevalent (Safe Child Network, 2003). A 2002 report issued by the Department of Health and Human Service (DHHS), reported that in 1997-98 over 2.6 million children 6-11 years of age were reported to have either a diagnosis of ADHD or a Learning Disability (Centers for Disease Control, 2003). According to a report issued by National Center for Health Statistics, almost five million children 3-17 years of age (8%) had a learning disability; 10% of boys had a learning disability compared with 6% girls. Approximately, four million children 3-17 years of age (7%) had ADHD. Boys were more than twice as likely as girls to have ADHD (10% versus 4%), although the prevalence ADHD among girls is on the rise.
Critics of ADHD believe the disorder is misnamed and that no fundamental defect in general attention has been satisfactorily demonstrated. According to an article in the Official Journal of the Canadian Pediatric Society the majority of children and adults diagnosed with ADHD simply have no tolerance for boredom and attend well to what interests him or her (Fox, 2002).

**Lead and Neurodevelopment**

Lead is the most-well-studied example of an environmental contaminant that interferes with learning, the result of complex interactions among genetic, environmental and social factors that impact children during vulnerable periods of development. The epidemiologic evidence of the link between low-level lead exposure early in life and later deficits in intellectual and school performance is strong (Center for Children’s Health, 2003). More recently, scientific evidence has left little doubt that lead can cause changes in both cognitive and behavioral function.

An observational study conducted by Canfield, et al of the blood lead concentrations in 172 children concluded that blood lead concentrations, even those below 10 μg/dL blood, were inversely associated with children’s IQ scores at three and five years of age, and associated declines in IQ are greater at these concentrations. Studies have also shown that exposure to lead has been linked to disruptive classroom behavior and reduced ability to pay attention. Lead, an undisputed neurotoxin, may affect numerous cell functions, including the release of neurotransmitters such as dopamine and serotonin (Rogan, 2003).

ADHD is a challenging disorder for children, families, schools and clinicians. ADHD has been linked with heavy metal toxicity, most notably lead.
In a public health study of more than 200 school children, teachers were asked to administer a questionnaire rating their students’ classroom behavior. Hair samples were also taken and analyzed for lead content. Researchers found a striking dose-response relationship between hair lead levels and a negative rating from the teachers with an even stronger relationship between physician-diagnosed ADHD (Rogan, 2003). Studies utilizing hair lead in the same children concluded that there is no safe threshold for lead in a child’s body, and that scalp hair should be considered a useful epidemiological tool for assessing a child’s chronic low-level lead exposure (Lead Environmental Awareness and Detection, 2003).

**ADHD Diagnosis**

ADHD is the most common behavioral disorder of childhood and is generally thought to account for the majority of referrals for mental health treatment (Richters et al., 1995). There is no screening test available to test for ADHD prior to the onset of symptoms. The DSM criteria for ADHD changed in 1980 (DSM-III), 1987 (DSM-III-R) and 1994 (DSM-IV), and they differ from the ICD-10 (1992) criteria.

The different diagnostic criteria classify different groups and proportions of children. In addition to these diagnostic criteria, there are numerous rating scales and other procedures for assessing ADHD. Because there is no single gold standard, data from multiple procedures must be aggregated for both research and clinical purposes. As such, ADHD is clinical diagnosis is based on the family’s description of the child’s behavior, the school report, the clinician’s observation, and at times comprehensive testing.
There are no specific tests for ADHD. However, Conner’s Parent Questionnaire and Teacher Rating Scale can help to elicit a quantitative assessment of the specific ways the behaviors may present. There are also various types of continuous performance tests used to assess the child’s ability to pay attention to routine, uninteresting stimuli, and components of many psychological tests—such as trail making and mazes, to assess attentional abilities (Centers for Disease Control- Epidemiology of ADHD Abstracts, 2002).

Evidence indicates that the symptoms associated with ADHD have a central nervous system basis. Symptoms related to ADHD tend to worsen in situations that require sustained attention, mental effort, or situations that lack appeal or novelty. Activities such as listening to teachers or working on monotonous, repetitive tasks are often difficult if not impossible for children with ADHD. Diagnosis is based on a collaborative process that involves children and adolescent psychiatrists or other physicians, the child, and school-based or other professionals as appropriate. Psychosocial stressors, such as parental divorce, child abuse, death of a loved one, environmental disruption (change in residence or school), or disasters can result in temporary symptoms of inattention, impulsivity, or over-activity. Under the aforementioned circumstances the symptoms will arise suddenly and have no long-term history (Center for Disease Control, 2003) (Nass, 1995).

There has been concern in some circles that ADHD has been over-diagnosed among those with regular access to health care and there is equal concern that the problem is under-diagnosed among those with limited or no access to care (National Center for Health Statistics (2002).
Individual treatment plans require a careful diagnostic assessment after a comprehensive evaluation of psychiatric, social, cognitive, educational, family and medical neurological factors (Safe Child Network, 2003).

Types of ADHD

There are three types of ADHD: Predominantly Inattentive Type (PIT), Predominantly Hyperactive-Impulsive Type (PHIT) and Combined Type (CT) (Appendix V). Children with PIT find it hard to organize or finish a task, pay attention to details or follow instructions or conversations. Children with PHIT are impulsive, fidget and talk a lot. They find it hard to sit still for long periods of time and often interrupt or speak at inappropriate times. Children with PHIT also find it hard to wait their turn or listen to directions and often have more accidents and injuries than other children. In CT the symptoms of PIT and PHIT that are equally prevalent (National Center for Birth Defects, 2002).

ADHD Treatment

Treatment of ADHD has evolved over the last 50 years. Early interventions using medications began in the 1930's. The development of a treatment plan must encompass all major aspects of a child’s life. It may include the use of special education programs, psychotherapy and medication. There have been several controversial treatments: dietary replacements, exclusion, or supplementation; various vitamins, mineral or herbal regimens; biofeedback; perceptual stimulation; and a host of others (CHADD, 2003). Although these interventions have generated considerable interest and made dramatic claims in ADHD interventions, they have not been supported by research findings.
The supporters of the Feingold Diet suggested that an additive-free diet would improve most (if not all) of a child’s learning and attention problems. However numerous well-controlled studies published in peer-reviewed journals have consistently failed to find support for the Feingold Diet, although a few studies reported some limited success with this approach (CHADD, 2003).

Children properly treated for ADHD generally get along better with teachers, peers, and parents, which leads to a boost in self-esteem. Children who go untreated often have problems interacting with others, fall behind in school and are at increased risk for alcohol and drug abuse later in life. Medications can be extremely helpful for children with ADHD.

Research indicates that between 70% and 80% of children with ADHD respond to medication but medication alone is rarely an appropriate treatment for this disorder. Medication should only be used in concert with a comprehensive treatment plan. ADHD cannot be cured, but many successful treatments exist to help a child pay attention, to decrease activity levels in the classroom, and to help concentration with schoolwork (Safe Child Network, 2003). Ritalin is the most prescribed and most controversial medication for treatment of ADHD. There have been concerns in the media about the over-diagnosis and over-treatment of ADHD: however an article in the Current Psychiatry Report suggests a substantial under-diagnosis continues to occur (Jensen, 2000).
ADHD Etiology

Evidence implicates multiple factors in the etiology of ADHD, yet it is unclear how these factors interact at the level of the single case. The best studied factors for ADHD are family history and heritability. Depending on the study, heritability has been estimated to be between 50-80%. Yet these factors do not appear to be all or none. Instead, specific traits of ADHD appear to be heritable in a continuous function.

Prenatal and perinatal factors (hypoxia, maternal smoking, hyperbilirubinemia) and, more recently, exposure to environmental elements such as mercury and lead have been linked to ADHD (Center for Children’s Health and the Environment, 2003).

Association of Lead Poisoning and Cognitive Impairment

To some extent lead prevention is one of the small success stories of environmental health. The association of lead poisoning with cognitive impairment is well established and has resulted in the removal of lead from gasoline, paint and food cans. Despite these preventive measures, however, low-level lead exposure continues to present a problem for many communities and populations (Sanborn, 2002).

The adverse effect of acute and chronic exposure to neurotoxins has been extensively documented and numerous studies have shown that exposure to lead effects the cognition and development of children, and there appears to be no threshold below which effects do not occur. The evidence of an inverse association between low level lead exposure and IQ is unequivocal, although the evidence of a similar association between behavior and moderately raised lead concentrations is less clear and highlights the uncertainty as the real impact that lead makes on neurodevelopment (Lewendon, 2001).
The following table provides summary information on the housing stock in Connecticut’s children (Connecticut Department of Public Health, September 2001).

**Table 3 – Housing Stock Summary (2000 U.S. Census Data)**

<table>
<thead>
<tr>
<th>Location</th>
<th>% pre-1950 Units</th>
<th>% of Population Below Poverty Level in pre-1950 housing</th>
<th>% Occupied Rental Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Connecticut</td>
<td>31.4</td>
<td>9</td>
<td>33.2</td>
</tr>
<tr>
<td>Hartford</td>
<td>47.2</td>
<td>23</td>
<td>75.4</td>
</tr>
<tr>
<td>Bridgeport</td>
<td>46.9</td>
<td>14.7</td>
<td>56.8</td>
</tr>
<tr>
<td>New Haven</td>
<td>48.7</td>
<td>17.2</td>
<td>70.4</td>
</tr>
<tr>
<td>Waterbury</td>
<td>40.6</td>
<td>15</td>
<td>52.4</td>
</tr>
<tr>
<td>Stamford</td>
<td>26.6</td>
<td>7.8</td>
<td>43.3</td>
</tr>
</tbody>
</table>

NOTE: this summary does not reflect rural or semi-rural regions, which also have a significant proportion of pre-1950 housing.
Life Time Impact of ADHD

Current research suggests ADHD can extend beyond adolescence, affecting 2-4% of adults in the U.S. Attention and behavioral manifestations generally appear in several settings; home, school and social situations. The essential feature of ADHD is a persistent pattern of inattention and/or hyperactivity-impulsivity that is more frequently displayed and more severe than is typically observed in children at a comparable level of development (National Center on Birth Defects and Developmental Disabilities, 2004).

A 1987 study by Bellinger, published in the Journal Pediatrics linked the presence of lead in cord blood samples with later adverse developmental changes. One hundred and seventy middle and upper-middle class children from Boston were followed from birth to approximately 5 years of age. Lead exposure was assessed in these children as newborns using cord-blood lead levels (blood samples taken from the umbilical cord following birth) (Bellinger, 1987). The children’s performance was then measured using a scale called the Bayley Mental Developmental Index (MDI). This is a test widely used with young children which categorizes behavior into three groups: motor (coordination for example), cognition (ability to learn), and general behavior. Those applying the test to children identify and measure characteristics such as social orientation, how the child responds to the examiner, toys, or people; levels of cooperativeness in the child; fearfulness, attention, coordination and activity. After testing the Boston children, it was found that the group of two year olds who showed elevated cord blood lead levels at birth, levels between 10 and 25 µg/dL, displayed modest but persistent learning deficits in ability at age 24 months.
That is, the children exposed to higher levels of lead during their gestational period were lagging slightly behind their non-leaded peers (Bellinger, 1987).

In 1997, data from the U.S. National Health and Nutrition Examination Surveys showed that 4.4% of children in the United States had elevated blood lead levels. Physicians play an important role subsequent to identifying individuals with elevated blood lead levels, educating a patient to minimize family member’s exposure to lead has important economic benefits.

Minority children living in older housing, children living in metropolitan areas with populations of one million or more and poor children living in older housing are at highest risk of exposure (Sanborn, 2002).

Public Impact of ADHD and Lead

There is no evidence for a threshold below which lead has no adverse effects. The precautionary principle when applied to lead exposure means that even a slight reduction in exposure is beneficial to public health and the economy.

Children absorb three times more lead than adults (Lewendon, 2001) and are particularly at risk because they commonly put hands, toys and other items in their mouths, which may have come in contact with dust and dirt that contains lead.

According to 2000 Census Data, 78% of Connecticut houses were built prior to 1980 and 64% contain lead-based paint (U.S. Census Bureau, 2003). The direct costs of medical care for children and youth with ADHD are substantial and these costs represent a serious burden for many families because they frequently are not covered by health insurance.
Further, these individuals consume a disproportionate share of resources and attention from the health care system, criminal justice system, schools and other social service agencies.

The following table provides summary information on the basic prevalence of lead poisoning in Connecticut’s children (Connecticut Department of Public Health, September 2001).

**Table 4 – Prevalence of Lead Poisoning, 2000-2001 *  
% of Children with blood lead level greater than or equal to 10 µg/dL  
CLPPP Lead Surveillance System Data**

<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Connecticut</td>
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<td>4.6</td>
<td>3.4</td>
<td>3.5</td>
<td>3.5</td>
<td>3.8</td>
<td>2.8</td>
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</tr>
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<td>Hartford</td>
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<td>17.4</td>
<td>11.6</td>
<td>11.0</td>
<td>9.4</td>
<td>9.6</td>
<td>7.2</td>
<td>7.9</td>
</tr>
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<td>Bridgeport</td>
<td>6.4</td>
<td>7.7</td>
<td>5.3</td>
<td>6.3</td>
<td>5.5</td>
<td>7.1</td>
<td>4.3</td>
<td>5.5</td>
</tr>
<tr>
<td>New Haven</td>
<td>14.8</td>
<td>15.7</td>
<td>9.7</td>
<td>11.2</td>
<td>9.6</td>
<td>10.1</td>
<td>7.9</td>
<td>8.3</td>
</tr>
<tr>
<td>Waterbury</td>
<td>2.2</td>
<td>1.7</td>
<td>1.2</td>
<td>1.1</td>
<td>1.5</td>
<td>1.4</td>
<td>1.3</td>
<td>1.3</td>
</tr>
<tr>
<td>Stamford</td>
<td>5.5</td>
<td>5.1</td>
<td>4.7</td>
<td>3.7</td>
<td>5.7</td>
<td>5.2</td>
<td>4.5</td>
<td>3.6</td>
</tr>
</tbody>
</table>

* The preceding table on prevalence demonstrates that the cities in Connecticut with a high percentage of the risk factors associated with lead poisoning, do in fact have slightly declining lead levels, but still have a high prevalence of lead poisoning in children under six years of age with the highest rates in children one and two years old. It should also be noted that the percentage of children who were lead poisoned may be underestimated, as there were children who had initial screening tests and had elevated blood lead levels, but never received confirmatory testing. These statistics do not include that group of children.

**Lead Abatement**

Minority children living below the poverty level, living in old housing stock (pre-1950), in the Connecticut’s highest risk cities (Bridgeport, Hartford, New Haven, Waterbury, Stamford) are most at risk.
Treatment is difficult, government officials and physicians recommend the removal of chipping or flaking lead paint. However, sometimes it is necessary to encapsulate the lead paint or stripping it out completely.

A study conducted in 1991, by the CDC showed that cleaning up lead in pre-1950’s homes would save the nation about $62 billion in medical and special education costs over twenty years. However, abatement is very expensive as well. It may cost between $2500 to $10,000 or more per house depending on the size of the home and the lead involved (Spake & Couzin, 1999).

In 2000, the CDC estimated that approximately 434,000 U.S. children aged one to five exceed the 10 µg/dL blood lead levels established by the agency. There are approximately 24 million housing units in the United States that contain deteriorated leaded paint and elevated levels of lead-contaminated house dust and more than four million of these dwellings are home to one or more young children (Centers for Disease Control Childhood Lead Poisoning Fact Sheet, 2003).

In Connecticut, during 2000, there were 63,955 children screened for lead that resulted in 2,651 (4.1%) confirmed cases of elevated blood lead levels in children less than six years old (CT Department of Public Health, 2005). The cities of Bridgeport, New Haven and Hartford comprised approximately 59% (1558 cases) of the confirmed cases of elevated lead levels in the state. Although statewide less than 5% of the children screened were positive for elevated lead levels, the incidence of elevated levels in the aforementioned towns demonstrates there is still work to be done to ensure that all children live in a lead safe environment (CT Department of Public Health, 2005).
Treatment for Lead Poisoning

The CDC recommends that children with blood lead levels of 45 mcg/dL blood or greater be referred for chelation therapy immediately (Agency for Toxic Substances and Disease Registry, 2003). Chelation therapy historically involved a painful hospital procedure of injections that causes lead to be excreted in the urine. More recently oral chelation drugs have been developed that can be administered without hospitalization. Chelation procedures do not reverse damage already done to the body and it is thought that lead deposited in the brain tissue is not removed by this procedure.

In addition treatment requires careful clinical and laboratory surveillance to ensure that there is not continued exposure. Just as important in the treatment of lead poisoning is the removal of the source of lead (World Health Organization, 2003). Depending on the detected blood lead level there are different methods for treating lead poisoning ranging from diet adjustment to hospital stays. Lead toxicity can be magnified by certain deficiencies in iron, calcium or zinc which can facilitate the amount of lead absorbed by the body, a diet low in fat and high in calcium and iron can help.

What Remains to be Done

In the past three decades, studies have demonstrated inverse associations between blood lead levels, IQ and cognitive impairment at successively lower lead concentrations and it has been suggested that health care providers test blood lead levels as a normal course of treatment when children present with behavioral problems.

We have learned much about the sources of and the solutions to lead poisoning, but the taxpayer dollars available to ensure the effective use of these remedies fall short of the need, and for the most part the burden of addressing lead paint in housing has remained
with property owners and the taxpayers. Widespread health screening and surveillance is necessary to ensure the most at risk are identified and treated. Normally, the properties burdened with the worse lead hazards have access to the fewest resources. Additional resources are needed to effectively address the lead issue and public education is an essential element in reducing lead exposure/poisoning (Child & Family Resources, Inc., 2003).

**Lead Poison Prevention**

Lead poisoning is entirely preventable. The key is stopping children from coming into contact with lead and treating children who have been poisoned by lead. If someone suspects their home contains lead, sampling should be conducted. Prior to testing and/or lead removal, homeowners can take simple steps (interim controls) to reduce the risk of lead poisoning. These include repairing damaged painted surfaces; damp mopping floors; damp-wiping surfaces; frequently washing a child’s hands, pacifiers, and toys to reduce exposure to lead; avoid using home remedies such as arzacon, greta, pay-loo-ah; and avoid cosmetics that contain lead, kohl and alcohol (Child & Family Resources, Inc. 2003).

Household water suspected to contain lead should be tested to avoid ingesting lead. Consume only cold water from household pipes and run water for several minutes prior to use boiling water is not an effective method to remove lead from water. Members of the household who have occupational exposures to lead should remove clothing and shoes before entering the house and clothes should be washed separately to reduce exposing other family members to lead.
To permanently remove lead hazards, a certified lead abatement contractor must be hired. Abatement methods include removing, sealing or enclosing lead-based paint with special materials and covering with paint designed for lead abatement. Covering lead-based paint with regular paint is not sufficient to remove the hazard (Centers for Disease Control and Prevention, 2003).

**Agencies Involved in Lead Prevention and ADHD**

The federal Environmental Protection Agency, CDC, Department of Housing and Urban Development, Food and Drug Administration and Consumer Product Safety Commission work together with state and local agencies; including the Connecticut Department of Public Health, Department of Environmental Protection, Department of Consumer Protection, and local Departments of Health to enforce lead standards and educate the public regarding lead exposure and the safe method to address household lead removal. The National Institute of Health supports research on developmental disorders. The National Institute of Child Health and Human Development and the National Institute of Mental Health seek to address unanswered questions about the causes of ADHD, the link between environmental toxins and ADHD, as well as methods to improve diagnosis and treatment.

**Conclusion**

A historical review of toxic chemicals reveals a disturbing pattern. As a rule, these chemicals are recognized as harmful long after their use has become routine and exposures widespread. In some cases, toxic chemicals have become entrenched global contaminants by the time their human health consequences are understood.
Because the fetus and developing child are most sensitive to the effects of these exposures, our children in particular bear the risks of regulatory polices that largely consider chemicals safe until proven harmful (Stein, 2002). Lead and other toxic chemicals are clearly linked to some childhood developmental disabilities. These disabilities are the result of complex interactions among genetic, environmental and social factors that impact children during vulnerable periods of development. Exposure to toxic chemicals deserves special scrutiny because they are preventable causes of harm.

The link between lead exposure and decreased IQ has been well studied. An observational study conducted by Canfield, et al of the blood lead concentrations in 172 children concluded that blood lead concentrations, even those below 10 μg/dL blood, were inversely associated with children’s IQ scores at three and five years of age, and associated declines in IQ are greater at these concentrations. Studies have also shown that exposure to lead has been linked to disruptive classroom behavior and reduced ability to pay attention. Lead, an undisputed neurotoxin, may affect numerous cell functions, including the release of neurotransmitters such as dopamine and serotonin. However, the link between lead and ADHD has not been clearly demonstrated. In a study by Bellinger, one hundred and seventy middle and upper-middle class children from Boston were followed from birth to approximately five years of age. The lead exposure in these children was measured using cord-blood lead levels as newborns. The children were administered the Bayley Mental Developmental index (MDI) to measure characteristics such as social orientation. After testing the Boston children, it was found that those children with elevated cord blood lead levels at birth, levels between 10 and 25 mcg/dL, displayed modest but persistent learning deficits in ability at age 24 months. However, this study would need to be replicated adjusting for confounders such as SES, dietary implications, gender and race. Lead, an undisputed neurotoxin, may affect numerous cell functions, including the release of neurotransmitters such as dopamine and serotonin.

In 2000, 6.6% of the population in Connecticut, were under the age of five, the primary vulnerable portion of our population that is at risk for lead poisoning.
Historically, efforts to control lead hazards in housing have relied on blood lead screening to identify already-poisoned children. But relying on children as lead detectors postpones action until after permanent damage has been done. Emerging evidence linking neurotoxins and childhood development is clearly documented as is the need to identify and control the environmental hazards that impacts a significant portion of our society. There is no cure for ADHD and children diagnosed with this disorder seldom outgrow it: however some individuals find adaptive ways to accommodate the disorder as they mature.

The benefits derived from eliminating childhood lead poisoning, reducing prenatal and postnatal exposure during formative years is two fold: the potential to reduce the prevalence of developmental disorders such as ADHD and to reduce the economic burden associated with lead remediation, decreased and lost productivity and increased health care costs (Stein, 2002). Historically, ADHD diagnosis has increased while lead exposure has gone down. Which raises an interesting question – how is that possible? Yes, BLL’s have gone down over the years as a result of the removal of lead from paint and automobile gasoline, however, exposure to lead environmentally and occupational has not. A large segment of our population is still routinely exposed to lead.

Recommendations for Future Research

There is no disagreement that lead can cause changes in both cognitive and behavioral function. Lead has been recognized as a dangerous substance for many years and the control and removal of lead from the environment is viewed by public health officials as public health issue of paramount importance. However, ADHD is a different matter, in truth we know little concerning the etiology of the disorder.
As a result, we know essentially nothing about primary or secondary prevention. More, however, is known about tertiary prevention, in that effective treatments have been demonstrated to improve long-term outcomes several years post treatment (Jensen, 1997). There is a lack of research in the area of prevention which suggests several avenues for future research such as: specific perinatal/prenatal influences in interaction with genetic susceptibility, the impact of home and environmental factors that might modulate the development of children’s attentional capacities, early learning experiences, exposure to television, video games or other factors that impact or shape a child’s attention styles, and early intervention strategies designed to remediate attentional and executive functional capacities in children at risk.

Any future study of ADHD and the risks associated with the disorder should combine assessments of some of the identified candidate genes with neuropsychologic and neurophysiologic studies, combined with direct observations. Research suggests that ADHD is a multifactoral syndrome. Further, given that cultural factors can and do have an effect on defining specific behavioral characteristics of the syndrome these factors should be examined along with environmental forces.
Appendix V

DSM-IV Diagnostic criteria for Attention-Deficit/Hyperactivity Disorder

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
Appendix V

DSM-IV Diagnostic criteria for Attention-Deficit/Hyperactivity Disorder

(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 hands 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).
Appendix V

DSM-IV Diagnostic criteria for Attention-Deficit/Hyperactivity Disorder

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 Criterion A1 is met but Criterion A2 is not met for the past 6 months

**314.01 Attention-Deficit/Hyperactivity Disorder, Predominantly Hyperactive-Impulsive Type:** if Criterion A2 is met but Criterion A1 is not met for the past 6 months

*Coding note:* For individuals (especially adolescents and adults) who currently have symptoms that no longer meet full criteria, "In Partial Remission" should be specified.
References


