Neurotoxic Chemicals in the Environment

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Abstract

Recent studies indicate that there is a possible link between neurological disorders and neurotoxins in the environment. The objective of this project was to quantify normalized disorder prevalence in relation to the level of toxins present in the United States between the years of 1990 to 2007. The disorders investigated were autism, attention deficit hyperactivity disorder, cerebral palsy, and mental retardation. The neurotoxins studied were polyvinyl chloride, lead, mercury, and polychlorinated biphenyls. Analysis of the trends indicated that an increase in autism rates was associated with an increase in certain neurotoxins. Further research would be beneficial in elucidating the exact cause of the rising trends in these neurological disorders.

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Introduction

Over the past several decades, it has been hypothesized that the rates of neurological diseases such as Autism, Attention Deficit Hyperactivity Disorder (ADHD), Cerebral Palsy, and Mental Retardation have been increasing in prevalence. Why have these rates been increasing? There have been numerous hypotheses for the answer to this, including changes in the way the diseases have been diagnosed, vaccines, and even neurotoxic chemicals in the environment, the major focus of this project. Research has shown that many neurological disorders may be caused by neurotoxic chemicals such as Lead, Mercury, Polyvinyl chloride, and Polychlorinated biphenyls. The first researchers to study this possible link were Grandjean and Landrigan in their paper "Developmental Neurotoxicity of Industrial Chemicals, Lancet, 2007." Using this paper as a basic backbone for this project, the team set out to gather, study, and analyze emissions and prevalence of the four industrial chemicals mentioned above, as well as gather information on the neurological diseases stated earlier.

Throughout the twenty weeks working on this project, several tools were utilized to gather information including the Environmental Protection Agency Toxic Release Inventory, the Center for Disease Control, the National Institutes of Health and numerous papers and articles. Data found was tabulated and placed in graphs which are presented later in this document. Some data, especially that on cerebral palsy and mental retardation was not publicly available, so the increasing rate hypothesis could not be confirmed for those disorders.

For the data that was available, it was found that Autism and ADHD both increase as time goes on. From 1997-2003 the rates of ADHD for the entire United States increased. Autism data increased across the United States from 1992-2003. The increase of these disorders is confirmed, but the question asked is why.

The answer to why could lie in the data from the neurotoxins. There are strong correlations between PCB and Mercury emissions and Autism. Every time the Mercury or PCB emissions spike, the autism rate seems to increase. This idea is explored further in the paper.

Major challenges associated with this project include the elusiveness of the data as well as there being no direct cause. The data presented here is only a hypothesis to a contributing factor. These disorders could be caused by other factors and would need many more years of study to find a direct correlation.

Although the data does not link neurological disorders to neurotoxic chemicals in a major way, it is useful to know the amount of industrial chemicals still present in the environment today. Most of these chemicals are known carcinogens as well, so due caution is necessary. One must be careful of bioaccumulation in the body from fish or food that has been contaminated with these chemicals. As time and studies continue, if a decrease in neurotoxic chemicals is found, it would be interesting to see if a decrease in neurological disorders is seen as well.

Neurological Disorders

A neurodevelopmental disorder is defined as a disorder that affects the proper functioning of the nervous system. Usually this disorder begins during development while the brain is forming in the womb. The nervous system may be impeded by a chemical imbalance, a misfire of synapses, or may not be known, as is the case for many of the disorders studied. Major neurological disorders that have been linked to neurotoxins are Autism, ADHD, Cerebral Palsy, and Mental Retardation.

Autism is a disorder that affects the social abilities of a child. Three major abnormalities are recognized as a part of this disease: impaired social interaction, impaired communication ability, and increased incidence of repetitive and/or obsessive behaviors. Of these abnormalities, each can occur in varying severity, resulting in a wide variety of symptoms and a challenging diagnosis (Health, 2008). The broad range of autism disorders is called "autism spectrum disorders". It is estimated that "3.4 [of] every 1,000 children ages 3-10" has an autism spectrum disorder (Health, 2008). Each of the neurotoxins being studied has been linked to autism in one way or another.

Cerebral palsy is another neurological disorder that could be caused by neurotoxins in the environment. The National Institute of Neurological Disorders and Stroke estimates that 800,000 children and adults are living with the disease, while 10,000 newborns each year are diagnosed with cerebral palsy (Stroke, 2008). This disease affects body movement and muscle coordination in varying degrees of severity. Many children can walk and talk with the disease while others are wheelchair bound. The disease is non-progressive, so once diagnosed, it does not get worse. (Stroke, 2008) Cerebral palsy is linked to neurotoxins because the disease is normally caused by

a developmental problem of the motor cortex (Stroke, 2008). Each neurotoxin has been proven to cause brain damage in or around that area of the brain.

Neurological and developmental factors can also trigger Attention Deficit Hyperactivity Disorder, more commonly referred to as ADHD. This condition is usually diagnosed in children during preschool and early elementary. This provides a structured environment, which allows the symptoms to become apparent. In the U.S., 3-5% of children have been identified as having ADHD. There are three different types of ADHD based on the predominate signs; inattentiveness, hyperactive-impulsive, or a combination of both. It has been observed that the structure and function of parts of the brain differ in children with ADHD. Although there has been no exact cause identified, problems during pregnancy or toxins from the environment could have had an affect on the development of the children's brain (National Institute of Mental Health, 2008).

Another disorder that is reflected through neurological and developmental behaviors is mental retardation. People diagnosed with mental retardation are characterized as having limitations in mental functioning. These delays result in having a lower intellectual capacity. Mental retardation is thought to be triggered by genetic defects, diseases, problems during pregnancy, and environmental toxins (Sebastian, 2006).

Autism

Autism was first discovered in the mid 1900's by Dr. Leo Kanner. He initially called this disease early infantile autism. As Kanner was doing his research, the German scientist, Dr. Hans Asperger diagnosed a milder form of the same condition and called it Asperger's syndrome. Asperger's is now known as one of the Autism Spectrum Disorders (ASD). Later, in 1964, Dr.

Bernard Rimland found that autism was a biological disorder (Research, 2002). As the century continued, more breakthroughs on what autism and autism spectrum disorders are were made. The most recent autism event was a finding that certain genes may be responsible for the disease. (Research, 2002) ASD encompasses a wide range of varying symptoms, ranging from mild to severe (Health, 2008).

The symptoms of Autism are different in every child, but can be detected as early as 18 months. Most cases are reported by the time a child is 3 years old (Health, 2008). Symptoms of autism and autism spectrum disorders are that the child: does not babble, point, or make meaningful gestures by 1 year of age, does not speak one word by 16 months, does not combine two words by 2 years, does not respond to name, loses language or social skills, has poor eye contact, doesn't seem to know how to play with toys, excessively lines up toys or other objects, is attached to one particular toy or object, doesn't smile, or at times seems to be hearing impaired (Health, 2008).

The more severe ASD is known as autistic disorder while the less severe is known as Asperger syndrome. Children that do not exhibit all of the symptoms of either ASD may be diagnosed with pervasive developmental disorder not otherwise specified (PDD-NOS). Effective diagnosis of ASDs consists of developmental screening and a comprehensive evaluation. There is a general consensus that early detection and treatment of an ASD can result in substantial improvement. There is no single panacea treatment for an ASD due to the large amount of variation in symptoms and severity. It is not uncommon however, for treatment to involve educational/behavioral interventions or medications (Health, 2008).

In addition to the normal behavioral disorders associated with ASDs, a number of other problems may accompany affected individuals. These additional problems include, but are not

limited to: sensory problems, mental retardation, seizures, Fragile X syndrome, and tuberous sclerosis (Health, 2008).

The cause of autism is currently unknown, although it is generally accepted that abnormalities in the brain structure and function are associated with the neurological disorder. Most scientists believe that the deformities stem from a combination of genetic and environmental sources. Other theories that have been investigated include the link between vaccines, problems during pregnancy, and hereditary (Autism Society of America, 2008).

Parents initially became worried when the signs of autism appeared around the same time their children received the measles-mumps-rubella vaccine, also known as the MMR vaccine. The MMR vaccine protects children against three dangerous and potentially deadly diseases; however it consists of thimerosal, which is a preservative that contains very small amounts of mercury (Mayo Clinic Staff, 2006). The first vaccine is given to children between 12-15 months old with the second dose administered between four to six years of age (Centers of Disease Control and Prevention, 2007).

A study that was performed in 1998 suggested that MMR vaccines caused bowel problems which then led to autism. However, the results were inaccurate due to the small number of cases observed. The study consisted of only 12 children. The other problem with the generalization that this study proposed was that the symptoms of autism appeared before the symptoms of the bowl disorder in several children. In 2004, ten out of the thirteen authors of the study retracted the interpretations that they had concluded from the 1998 study (Centers of Disease Control and Prevention, 2007).

In larger studies that have been carried out, there has been no relationship shown between autism and thimerosal-containing vaccines. The United Kingdom studied the link through 498

children with autism born between 1979 and 1998. The results concluded that the percentage of children with autism was the same as the percentage of children without autism who received the same MMR vaccine. In the cases that autism was diagnosed, the onset of the regressive symptoms did not occur within 2, 4, or 6 months of receiving the MMR vaccine. It was also indicated that there was no difference in the age of diagnosis of autism in vaccinated and unvaccinated children. It was concluded in 2005 by the Institute of Medicine that there is no association between autism and vaccines that contain thimerosal, including the MMR vaccine (Centers of Disease Control and Prevention, 2007).

A commonly accepted theory for the cause of autism is genetic defects. Even though a single gene has not been identified as the root of the problem, there are speculations that autistic children inherited irregular segments of genetic code. The irregular segments could result in the children being born more susceptible to autism, although the stimuli have not yet been narrowed down to an exact "trigger" (Autism Society of America, 2008).

There have been studies that link autism to birth complications. As a group, children who have developed autism tend to have had more birth problems than normal children. This includes near miscarriages, induced labor, fetal distress, and cesarean section. Studies have shown that autistic children tend to be first-borns, and that the mothers had conceived at an older age. The complications during birth did not likely result in autism. It is more likely that the irregular fragments in the genetic code, which may have been inherited by autistic children, resulted in difficulties at birth as well (WebMD, 2004).

Along with the genetic predisposition, many environmental factors are considered major contributors in the development of Autism. These include viral infections, metabolic imbalances, and exposure to environmental chemicals. Fetal exposure to the rubella virus can result in

impaired brain development leading to blindness, deafness, birth defects, mental retardation, and/or autism (Schettler).

There have been studies that try to discover a link between autism and certain neurotoxins such as lead, PCBs, mercury, and PVCs. In one study, three inner city children diagnosed with autism and lead poisoning were studied. It was found that the poisoning may have contributed to the acceleration of autism symptoms but was not an actual cause for the autism diagnosis (Accardo, 1988). Another study tested 40 autistic boys and 40 normal boys for metal concentration in the hair. It was found that the autistic children had a higher concentration of lead, mercury, and uranium (Fido, 2005). Although a solid link between autism and lead is yet to be found, the speculation still remains.

Polychlorinated Biphenyls (PCB's) have also been linked to autism. Again, there is no solid link of PCB's to autism but the speculation is very real. In one study the brains of rats exposed to PCB's has holes in it and neurons that should have fired did not. This affected the rat's hearing (Kenet, 2007). The fact that holes were found contributes to the notion that PCBs are neurotoxic chemicals. In another study, children exposed to PCB's while still in the womb had lower IQ's and trouble learning and memorizing (Jacobson, 1996). These are some symptoms of autism and other disorders such as ADHD. This paper will try to analyze the link between PCBs and autism, if one does exist.

A study done by Dr. Amy Holmes revealed a possible connection between mercury and autism. Baby hair cuttings were observed from children around 18 months of age; 94 from autistic and 45 from other children. The average level of mercury was analyzed to be 0.47 parts per million in children who were later diagnosed as autistic and 3.63 parts per million in the other children. The lower levels of mercury are a result of the children with autism not being able

to properly use and dispose of the harmful toxin. Therefore, the mercury is retained in the cells, and does not get excreted. The interpretation that was concluded was that the lower the levels of mercury in the hair, the more severe the child's autism is (BBC News, 2003).

Polyvinyl Chloride has been an issue of concern for a while due to its harmful affects in humans and the environment. Toxins are released during production, usage, and break down of PVC. The major component that is linked to health concerns is chlorine, which is highly reactive and readily combines with the building block of life, carbon. The toxins from PVC that may have a possible connection with autism include phthalates, dioxins, and biphenol A. Action has been taken to limit the exposure of PVC to children, especially during early years when their brains are in the process of developing, by banning the plasticizers from toys and bottles (Autism Society of America, 2008).

There are many theories on the causative agent of autism. Exposure to environmental toxins is a major interest in the development of children's brains. Although many studies have supported this hypothesis, more research is needed to confirm the toxicity's affect on the development of autism.

ADHD

Attention Deficit Hyperactivity Disorder, known as ADHD, is a common neurobehavioral disorder that affects children and can continue on through adolescence and adulthood (Centers for Disease Control and Prevention, 2005). The condition causes a person to have difficulty in paying attention and focusing on tasks. Without treatment, ADHD can cause further problems at school, work, home, and with relationships (Healthwise, Inc., 2006).

General symptoms include having a continual level of inattention, impulsivity, and hyperactivity. These signs must appear early in life and last for at least six months. In most children, symptoms and signs are present before the age of seven. In order to be diagnosed with ADHD, the symptoms must considerably affect at least two areas of life, typically at home and at school (Mayo Clinic Staff, 2007).

There are different types of ADHD. A person is categorized based on their strongest symptoms. The three types are Predominantly Inattentive Type, Predominantly Hyperactive-Impulsive Type, and a combination of the two called the Combined Type (Centers for Disease Control and Prevention, 2005).

Characteristics of the Predominantly Inattentive Type include being easily distracted or forgetful with details of daily routines. Other related symptoms are failing to pay close attention to details, difficulty maintaining attention, and struggling to follow instructions. People under this type have trouble with organization and often makes careless mistakes (CHADD).

Predominately Hyperactive-Impulsive Type exhibit symptoms of hyperactivity and impulsivity but not inattention. These include having difficulty remaining seated, running around or climbing excessively, difficulty engaging in activities quietly, talking excessively, and difficulty waiting or taking turns. People labeled under this type usually act as if they are being powered by a motor. They tend to blurt out answers before the question has even been finished, and interrupts others. Another sign is fidgeting with hands or feet and squirming in chairs (CHADD).

The Combined Type consists of people who show signs from both of the other two types. The two types described above are equally predominant (Centers for Disease Control and

Prevention, 2005). The symptoms are labeled under both inattentive and hyperactive/impulsive criteria.

The precise causes of ADHD are still unidentified. However, it is known that there is a very strong neurobiological basis. It is believed that the structural changes in the brain may be the primary cause of ADHD. Brain scans have discovered that children with ADHD have 4% less brain volume than children without ADHD. There are also differences in the function of areas in the brain. The brain's chemical messengers, called the neurotransmitters, don't perform properly, and there is less activity in the regions of the brain that are in charge of activity and attention in people with ADHD (Mayo Clinic Staff, 2007).

Another possibility for the source of ADHD is problems surrounding pregnancy and toxins in the environment. From conception throughout the first year of life, the brain is continuously developing. Alcohol and cigarettes distort growing nerve cells. Children with Fetal Alcohol Syndrome share the same inattention, hyperactivity, and impulsivity symptoms as children with ADHD. Other drugs, such as cocaine, affect the normal development of brain receptors which may lead to ADHD. Finally, toxins in the environment and daily activities may be associated with disruption in brain development and brain processes (Jaffe, Benedictis, & Segal, 2007).

Cerebral Palsy

In 1861, Dr. William John Little described a peculiar new disease in which children's muscles were stiff, twitched often, and were incredibly weak. At that time, the disease was dubbed "Little's Disease" (For My Child). This was the first time this disease had been studied and documented, although it has been hypothesized that the disease existed ever since children were being born. (National Institute of Neurological Disorders and Stroke, 2008) It was

presumed that the disease was caused by a lack of oxygen during birth since most the infants with the disease went through complicated births. In 1897, Sigmund Freud disagreed with Little's theory and said that complicated birth was only one of the ways a child could be afflicted with the disease. It was not until the 1980's that some light was shed on what this disease was and how it could be treated. (National Institute of Neurological Disorders and Stroke, 2008)

Cerebral palsy is an umbrella term used to describe a myriad of symptoms that affect muscle movement and control. The disease is broken into three groups: spastic, athetoid, and ataxic. Each of these groups then encompasses several other groups characterized by more detailed muscle movement. Table 1 shows these types of cerebral palsy based on muscle movement.

Table 1: Types of Cerebral Palsy, Adapted from (National Institute of Neurological Disorders and Stroke, 2008)

Type of Cerebral Palsy	Affliction
Spasticity	
Spastic Hemiplegia	Affects arm and hand on one side of the body. Children walk on tip toes due to tightened muscles, and may develop scoliosis. Intelligence is not normally affected.
Spastic Diplegia	Affects legs for the most part. Severe tightness in the leg muscles cause scissor like walking. Intelligence is not normally affected. Many children with this type may need leg braces.
Spastic Quadriplegia	Most severe form of cerebral palsy. There is usually a lot of brain damage associated with this type, and speaking audibly is almost impossible. Children with this type have major spasms in the legs and arms, and may have uncontrollable seizures. Intelligence is usually greatly affected.

Athetoid or Dyskinetic	This type of cerebral palsy is characterized by slow and uncontrollable writhing movements in the hands, feet, arms, or legs. The face may be affected, causes grimaces or drooling. Intelligence is not normally affected.
Ataxia	Ataxic forms of cerebral palsy are rare and affect balance and depth perception. Poor coordination as well as having a hard time with basic tasks such as writing and buttoning shirts are characteristic. The hands may tremble quite a bit. Intelligence is not normally affected.

Spasticity often refers to tightened muscles with frequent muscle spasms. The three types of spastic cerebral palsy, as mentioned above, affect different areas of the body. Hemiplegia and diplegia affect the arms and legs respectively, with quadriplegia affecting all four limbs. Athetoid cerebral palsy is characterized by slow writhing movements, while ataxia affects balance. It is important to note that these categories are not all inclusive, as some children may have symptoms that fit into many of the characterizations. (National Institute of Neurological Disorders and Stroke, 2008)

The causes of cerebral palsy are uncertain. It has been noted that a small number of cases are due to lack of oxygen during birth, as Little hypothesized. This only accounts for 5-10% of the cerebral palsy cases however. An even smaller percentage of children are diagnosed with cerebral palsy after birth due to brain damage from infections or head injury. The majority of cases, between 90 and 95% have the disease at birth, with complications beginning during fetal development. Every case of CP however has one or more of: white matter brain damage,

abnormal development of the brain, bleeding in the brain, and brain damage caused by asphyxia. (National Institute of Neurological Disorders and Stroke, 2008)

The most important causes to this report are the first two, white matter damage and abnormal development of the brain. The neurotoxins mentioned earlier have all been noted to cause brain damage, and affect neurodevelopment. Data to analyze the affect of the neurotoxins and cerebral palsy was not available.

Mental Retardation

Mental retardation limits the amount of mental functioning an individual is capable of and is the most common developmental disorder. Limitations in motor skills, learning skills, language skills, and social skills are some of the many things that mental retardation causes. Retardation affects a person throughout his or her life starting when the child is very young. "Learning milestones", such as when a child begins to crawl, walk, and speak, along with several possible physical developments, are persistently delayed and dragged out or do not occur at all. These delays cause the child to grow into an adult with a significantly low intellectual capacity. Retardation can be measured by the intelligence quotient, specifically if the person has an IQ below 70. The Diagnostic and Statistical Manual of Mental Disorder considers three different criteria before a person is diagnosed with a mental disorder; an IQ below 70, limitations in the person's ability to function at age level in a normal environment, and confirmation that these limitations became apparent during their childhood. (National Center on Birth Defects and Developmental Disabilities, 2005)

Mental retardation is a matter of evaluating intelligence and adaptive behavior. Two or more areas of adaptive behavior must be limited in order for a person to be considered disabled. Adaptive behaviors are the skills that develop in children over time that allow them to eventually live independently. In order to evaluate whether these skills are developing, professionals compare behaviors of the subject to children of the same age. Daily living skills, communication skills, and social skills are assessed and measured in relation to others. (National Dissemination Center for Children with Disabilities, 2004)

Signs of mental retardation include trouble speaking, lack of memory, missing social cues, having trouble solving social problems, logical thinking, and constant childish behavior. Mild disability, IQ's between 60 and 70, is hard to distinguish in children before they enter school. Poor performance in academics is easily blamed on simple learning disorders, but then found to be mental disability. These people can function in society and are rarely referred to as "retarded". Moderate disability, IQ's between 50 and 60, is more noticeable early in a child's life. Children with a moderate disability usually have social and academic troubles and may need to be placed in special classes. They can function in society, but with more trouble, and cannot live on their own as adults. Most times they will have to live with parents or in a group home. Severe disability occurs when a person scores less than a 50 on an IQ test. Only about one in eight people score lower than 50, and will need more supervision and support throughout their lives. The table below summarizes the level of retardation and at which IQ level they occur. (Sebastian, 2006)

Table 2: Characteristic IQ levels for Mental Retardation

Class	IQ
Profound mental retardation	Below 20
Severe mental retardation	20-34
Moderate mental retardation	35-49
Mild mental retardation	50-69
Borderline mental retardation	70-79

(Sebastian, 2006)

Mental retardation has several causes, one being genetics. When one or both parents have abnormal genes, it is possible that the offspring may inherit them. Error can also occur when genes combine with one another. Genetic errors are the cause of diseases like fragile X syndrome, Mowat-Wilson syndrome, Down syndrome, Phelan-McDermid syndrome, and phenylketonuria. Problems during pregnancy can also result in complications. The fetus may suffer complications in the womb, such as when cell division does not occur properly. Also, if a mother consumes alcohol, the baby is susceptible to infections such as rubella and may be born with mental retardation. Complications during birth have been known to result in mental retardation as well. During birth, if the baby does not receive enough oxygen, the brain will not develop properly. Children can suffer mental complications as a result of diseases caught after birth or exposure to toxins. Measles, whooping cough, and meningitis can cause disability as well as exposure to lead or mercury. Extreme malnutrition or lack of medical care is another cause. If a child is raised in an area with severe environmental restrictions or is sensory deprived, he or she can develop a medical condition over time. Sensory deprivation includes being left in an isolated area for long periods of time repeatedly or lack of parent- child interactions. Lastly

and most notable, iodine deficiency is the leading preventable cause of mental disorder. (Sebastian, 2006)

Neurotoxins

A neurotoxin is any compound coming from an exogenous source (i.e. the environment) that affects neurological development. After examining lists of the most abundant types of toxins in the atmosphere and which have the most affect on neurological development, the group narrowed the focus down to four main toxins: lead, mercury, PVC (polyvinyl chloride), and PCB's (Polychlorinated Biphenyls). These toxins can affect a person throughout their life if they are exposed to too much overtime and can affect their mental state, their reproductive abilities, and may even cause death (Miller, 2007). Also, these neurotoxins may affect a child from birth if the mother is exposed to excessive levels of any of these toxins. Humans may come into contact with lead, mercury, PVC, and PCB's through inhalation, ingestion, or simply through the skin. Although these toxins can be harmful if one is exposed to too much, these toxins are used in the production of many everyday products.

Mercury can exist in many forms from elemental, inorganic, and organic. Primarily, humans are exposed to elemental, otherwise known as metallic mercury, by inhaling mercury vapors in the atmosphere. When inhaled, it moves from the lungs through the bloodstream and in some cases to the brain. Metal mercury is used in products such as thermometers, dental fillings, and several electronic devices. Inorganic mercury, or mercury salts, are formed when mercury is combined with sulfur, oxygen, or chlorine. These salts cause harmful affects when ingested through the stomach or intestines, or in most cases, when it is absorbed through the skin. Organic mercury is formed when mercury is combined with carbon. The most common form of organic mercury is Methylmercury, which had been used to protect against fungal infections for many years before the adverse health affects caused by these compounds was discovered. Exposure to mercury regardless of what form it is in causes mostly nervous system damage and in all of its

organs. Symptoms include retarding of the development of visual and motor skills, attention, language, and memory. Impaired speech and hearing are also negative symptoms caused by this toxin. (Agency for Toxic Substances and Disease Registry, 1999)

Lead compounds are prevalent in the environment due to their presence in industrial waste. Lead was previously used in the production of leaded gasoline and in lead paint before it was banned for use in both products because it can lead to the retardation of neurological development in children all over the world if they are exposed too heavily to lead. Even though production of lead paint is no longer occurring since it's banning, 25% of homes in the United States still contain some form of deteriorating lead paint, which is the main route of exposure to lead and a very serious concern for children's health. The use of lead in gasoline was ended by the Clean Air Act in 1996, but as a result of many years of leaded fuel usage, soil alongside roads in high traffic areas has become contaminated. Lead contaminated waters can also cause adverse health affects. Water becomes contaminated by passing through lead- based pipes. Water used in the production of any food or beverage also contains traces of lead which can be harmful to the consumer. It has been reported that over 400,000 children under 6 years old have dangerously high levels of lead in their blood. (Agency for Toxic Substances and Disease Registry , 2007)

PCBs, or Polychlorinated Biphenyls, are a class of about 209 chemicals containing two benzene rings and any number of chlorine atoms attached. PCBs have been used for many years as coolants, lubricants, inks, pesticides, and hydraulic fluids until their usage in the production of these products was banned in the U.S in 1970. However, PCBs are still a major environmental issue today. PCBs are extremely durable and take many years to break down naturally in the environment. Because of their extreme stability, PCBs can build up in certain areas, becoming

harmful to the living things around it. These toxins find homes in the fatty tissue of humans and animals. PCBs bind to sediment easier than any other substance. They many bind to sediment in the ocean floor, which are consumed by organisms, which are then consumed by fish, which are consumed by birds etc. This process, bioaccumulation, is an example of how sturdy and easy it is for PCBs to spread throughout the environment. The first reported instance of PCB poisoning was in Asia where many people had eaten contaminated rice oil and became sick. As a result, pregnant women with high levels of PCB in their blood gave birth to children with several neurological disorders, lower IQ's, and lower birth weights. The United States banned PCBs in 1976, which unfortunately came after 3.4 million pounds of PCBs had been produced worldwide. Not only are PCBs carcinogens, but they are proven neurotoxins that may cause several neurological disorders (Polychlorinated Biphenyls, 2007).

PVC, or polyvinyl chloride, was originally designed by Waldo Semon in 1926 when he was assigned to develop a manmade adhesive (The Standring Brothers). Because of its rubbery and flexible nature, PVC was used in the manufacturing of items like shoe heels and raincoats. Today, TVC is the second most used plastic in the world, used in the making of anti-oxidants, flame retardants, pigments, plasticizers, impact modifiers, and fillers. Although it is a very useful substance in the consumer product world, it can be harmful to humans. Every time PVC is burned, buried, used and disposed of, toxins are released into the atmosphere. Production of PVC involves usage of chlorine which becomes dangerous when put into contact with carbon, an essential element for all living things. Vinyl chloride, a dangerous carcinogen, and ethylene dichloride, harmful to the kidneys, lungs, cardiovascular, immune, and nervous system, are two damaging substances that can be formed during the production of PVC. Phthalates and Bisphenol A can be added to plastics to make them soft and can be broken down over time, causing a

release of these chemicals into the environment and potentially causing impairment to humans. PVC may cause carcinoma, liver damage, reproductive and neurobehavioral effects (Department of Health and Human Services, 2006).

Mercury

Mercury, existing in metallic (or elemental), inorganic, and organic form, can have adverse health affects on people of all ages. However, children under the age of six, along with fetuses, are at the greatest level of risk. Factors that may affect the severity of exposure include the chemical form mercury is in during the time of exposure, the amount the subject is exposed to, the age of the person exposed, how long the person was exposed, route of exposure, and the current health of the person exposed.

Elemental mercury is a silver- white liquid at room temperature. Metallic mercury is the pure form of mercury, meaning it is not mixed with any other chemical. Primarily, exposure to pure mercury occurs in vapor form when it is inhaled through the lungs when spilled in areas where not much air circulation occurs and via consumption, most commonly of fish containing mercury. When the elemental form of mercury is inhaled, it is absorbed through the lungs and then by the blood stream. Red blood cells convert the element to divalent mercury, while some of the mercury travels to the brain, where is crosses the blood-brain barrier and is oxidized. The vapor itself is colorless and odorless, but can have a slight taste when breathed in. (Agency for Toxic Substances and Disease Registry, 1999)

When mined, mercury is in a cinnabar ore state and can be developed into metallic form when heated above 1,000 degrees Fahrenheit. This process turns the cinnabar into a vapor which is then cooled to form metal mercury. The metallic mercury end product can then be used in

several every day projects like thermometers, barometers, dental fillings, and electrical mechanisms. Because the mercury used in these products is contained, it doesn't pose any health risk. Elemental mercury can also be used to produce caustic soda and chlorine gas, and can be used to extract gold from ore as well. Aside from its practical uses, elemental mercury is still used in several countries as herbal remedies. Unfortunately, these remedies can be harmful to the user and to others who are exposed to the vapors released into the air. (Agency for Toxic Substances and Disease Registry, 1999)

Elemental mercury can have an affect on several different areas of the brain, causing a variety of symptoms. Examples of the symptoms brought on by elemental mercury include tremors, moodiness, insomnia, headaches, neuromuscular changes, changes in nerve response, and decrease in cognitive function. At higher levels of exposure, one may experience kidney effects, respiratory failure, slurred speech, loss of memory, fatigue, and even death. (U.S. Environmental Protection Agency, 2007)

The inorganic forms of mercury, or mercury salts, can also be harmful to humans. Mercury is said to be ionic when combined with elements like sulfur, oxygen, or chlorine. Inorganic mercury is commonly used in fungicides and in several skin products. Mercurous chloride, in the past, has been used in various medicinal products such as laxatives, but has since been replaced with less hazardous agents. However, there are still several mercury containing chemicals, such as mercurochrome and thimerosal, used in antibacterials today. They are mainly used as preservatives in medications. Mercuric oxide can be used to color paints along with mercuric sulfide, is one of the red coloring agents in tattoo dyes. Exposure to a hazardous amount of inorganic mercury may cause damage to the kidneys, nervous system, and damage to the route of exposure, the gastrointestinal tract. Symptoms of exposure to the inorganic form of

mercury include skin rash, dermatitis, mood swings, memory loss, and muscle weakness. Majority of inorganic compounds of mercury enter the body through ingestion or through the skin. When ingested, only about 10% of the compound is absorbed directly through the lungs. Forty percent, however, can be exposed through the stomach and intestines, and then moves to several different tissues. When the compound comes into contact with the skin, only a small amount is actually ale to penetrate the skin. The mercury will slowly exit the body through urine and feces, or through a nursing mother's breast milk. (Agency for Toxic Substances and Disease Registry, 1999)

Organic mercury occurs when mercury is combined with carbon. The most common form of these organomecurials is methylmercury. Mercury occurs in nature in its several forms. The most common, naturally found forms are metallic mercury, cinnabar, methylmercury, and mercuric chloride. Microorganisms like bacteria and fungi can change mercury from one form to the next. Methylmercury is the most common example of these transformations. Before the 1970's, methylmercury and ethylmercury were used to protect against fungal infections. However, once the negative health affects of these compounds was discovered, their use in fungicides was quickly banned. (Agency for Toxic Substances and Disease Registry, 1999)

Methylmercury is known for its adverse neurological affects on adults, children, infants, and fetuses. A fetus's exposure to methylmercury occurs in the womb, which can occur as result of a mother consuming fish or shellfish that contain the compound. Here, the damage is done to the developing brain and nervous system. Children exposed to methylmercury while in the womb show negative symptoms through cognitive thinking, memory, attention, language, and motor and visual skills. Mercury has a very large affect on the nervous system more than anything. Exposure to the compound can result in permanent brain and kidney damage. During periods of

methylmercury outbreak, mothers with no symptoms of nervous system damage gave birth to infants with severe disabilities, proving that fetal nervous systems may be more vulnerable to the compound than the adult nervous system. This has been shown to occur mostly in areas of regularly high fish consumption. (U.S. Environmental Protection Agency, 2007)

Aside from harmful levels of mercury in humans, the Center for Disease Control and Prevention claims that most people have blood mercury levels below a harmful level. Symptoms include impaired vision, lack of coordination, impaired speech and hearing, and general muscle weakness. Methylmercury is mostly absorbed through the gastrointestinal tract when food contaminated with it is consumed. From here, it is absorbed into the blood stream and is carried throughout the body.

Other common organic forms of mercury include phenylmercury which is still used in many commercial products, and dimethylmercury, which is used as a reference standard in many chemical tests. Similar to methylmercury and ethylmercury, phenylmercury was previously used as a fungicide, but in paints. These were also banned because of the mercury vapors the paints release into the atmosphere. Dimethylmercury is the only form of organic mercury that has been found in sites containing hazardous waste, and is very dangerous to both people and animals. In contrast to phenylmercury and methylmercury which occur naturally as solid white crystals, dimethylmercury occurs as a colorless liquid. Unlike methylmercury, dimethylmercury is easily absorbed through the skin. In general, organic compounds enter the blood stream after being inhaled and move to tissues and the brain. Also, the blood of a pregnant woman can be passed onto her growing fetus. Similar to nature, the form of mercury can be changed by the human body. The mercury will then begin to leave the body over a long period of time through feces. (Agency for Toxic Substances and Disease Registry, 1999)

Mercury occurs in the environment as a result of the break down of minerals in rocks and soil. The amount of naturally occurring mercury has been relatively constant in recent years, causing it to rise steadily. Eighty percent of human- caused releases of mercury are a result of the burning of fossil fuels, smelting, solid waste incineration, and mining. Fifteen percent of these releases are absorbed by the soil, and another five percent is released from industrial waste water to other planetary bodies of water. Human activity generates one to two- thirds of the total amount of mercury released. Prior to the industrial era, the amount of mercury in the atmosphere was very low. Although the amount of mercury in the air we breathe is still not enough to pose a health risk, it is six times higher than it was before the amount of human activity increased. (Agency for Toxic Substances and Disease Registry, 1999)

The most common forms of mercury found in the environment are metallic mercury and inorganic mercury compounds, however the amount of mercury found in one place will never be very high. Methylmercury is famous for its abundance in the food chain. Small fish will eat food that has been contaminated by methylmercury in water and is then absorbed into their tissues. Bigger fish will then eat smaller fish or other foods containing methylmercury. Logically, the larger the fish, the higher the level of mercury in its body is. Plants, even if grown in soil with a higher level of mercury, don't tend to have a high mercury concentration. (Agency for Toxic Substances and Disease Registry, 1999)

Because mercury occurs naturally, everyone is exposed to a small amount every day. This may sound menacing, but the levels of mercury in the atmosphere are very far below the amount that is safe to breathe. The concentration of mercury in the air is lower in more rural areas. Also, the amount of mercury present in drinking water is approximately 1,000 times lower than the standard for harmless drinking water. A very common source of mercury exposure to

people is through dental amalgam fillings. These silver fillings contain about 50% elemental mercury, amongst several other metals. Mercury is released from these fillings as a result of corrosion through usage and then ingested. A very small amount of mercury can also be released through vapors as well. Although there is a small amount of mercury exposure involved with these fillings, no health risk is posed. A more sensitive population of people includes groups like pregnant women, people with damaged kidneys, and children under the age of 6. These people should be overly cautious about their exposure to mercury. (Agency for Toxic Substances and Disease Registry, 1999)

Metallic mercury used in herbal and religious remedies can also be harmful. Religions like Voodoo, Palo Mayombe, and Espiritismo, use metallic mercury sold under the name "azogue" in capsules. It is then used in various ways; burned in candles, used in baths, worn around the neck, or may be dusted in various locations. One may be affected by the mercury vapors in a negative way. Also, the vapors may contaminate the area the mercury was used in for years. Although the amount of mercury present in the atmosphere is generally very low, just a few drops of elemental mercury can raise air concentrations to a harmful level. The longer one breathes in contaminated air, the more harmful. Air around hazardous waste sites may also have traces of mercury vapors. (University of Minnesota; Environmental and Occupational Health)

It is also harmful to handle soil in hazardous areas, drinking well- water, eating fish living in contaminated waters, fungicides, and using dated medical products (before the 1970's). Employees in plants manufacturing electrical or automotive equipment containing mercury are at a higher risk of mercury exposure. Furthermore, people close to the employees of these factories may also be at slight risk because of the amount of mercury that stays in ones clothes. Basically, the three conventional ways mercury can enter ones body is through the air, skin, and ingestion.

Different forms of mercury have different affects on the nervous system, partly due to the way they enter the body. (University of Minnesota; Environmental and Occupational Health)

Lead

Lead is heavy metal element that has been used throughout history in many different applications. However, lead is also a potent neurotoxin with several adverse health effects. The lead used today comes from three different sources: mined ore, recycled scrap metal, and recycled lead-acid car batteries. The third largest lead producer, the United States, extracts most of its lead from ore mined in Alaska and Missouri. The lead from these sources was and still is used in several products whose use and consumption result in human exposure and intake of lead and/or lead compounds. Of these products, the ones least hazardous to developing children are lead-acid car batteries, ammunition, and fishing sinkers to name a few. Lead paint, leaded gasoline, and lead-contaminated water on the other hand, are serious hazards to developing children. (Agency for Toxic Substances and Disease Registry , 2007)

Lead paint was used in homes in the US before it was banned in 1978. The US Department of Housing and Urban Development estimates that about 24 million homes in the US (25%) pose some sort of lead paint hazard. This kind of hazard is characterized by deteriorating lead paint. Ingestion of the deteriorated paint in the form of lead-contaminated dust is the main vector of contamination. Intact lead paint is not an immediate concern. Lead compounds such as tetraethyl lead and tetramethyl lead were originally used in gasoline to increase its octane rating. The sale of leaded gasoline for use in automobiles was banned in the US in 1996 in accordance with the Clean Air Act. The use of this leaded gasoline in automobiles has resulted in lead-contaminated soil near roads and highways with high traffic volumes. The subsequent ingestion

of this lead-contaminated soil is another vector of contamination. The third hazard to developing children, although not as prominent as the previous two, involves water contaminated with lead through the use of lead pipes and solder. Any foods or beverages processed with lead-contaminated water also pose a hazard. (Agency for Toxic Substances and Disease Registry , 2007)

Blood lead levels are measured in micrograms/deciliter (µg/dL). The US Center for Disease Control and Prevention (CDC) states the the "level of concern" for children with lead in their blood is 10 µg/dL. At this level adverse health effects can begin to set in. The New England Journal of Medicine in April 2003 stated that levels as low as 5 µg/dL can also cause adverse effects. Presently there is no effective treatment for blood levels under 45 µg/dL. Exposure to lead hazards happens mainly due to the behavior of young children, who tend to put things in their mouth and play outside often. (National Safety Council, 2004)

It is estimated that over 400,000 children under 6 years of age have too much lead in their blood. Younger children and fetuses are at the highest level of risk because they are still developing. Low levels of exposure to children under 6 years of age can result in reduced IQ, learning disabilities, attention deficit disorders, behavioral problems, stunted growth, and impaired hearing. Higher levels of exposure can cause mental retardation, coma, and even death. In addition to interfering with the brain and neurological development, lead also affects blood forming organs and the kidneys. The specific effects of exposure vary as well from case to case. During pregnancy, lead can be transferred from the mother to the developing fetus through blood exchange. Sometimes the stress of pregnancy can lead to the release of lead from the mother's bones into her blood and eventually into the fetus's blood. (National Safety Council, 2004)

Polychlorinated Biphenyls

Polychlorinated biphenyls are a class of approximately 209 chemicals, each of which have any number of chlorine atoms attached to two benzene rings, or a biphenyl (Figure 1).

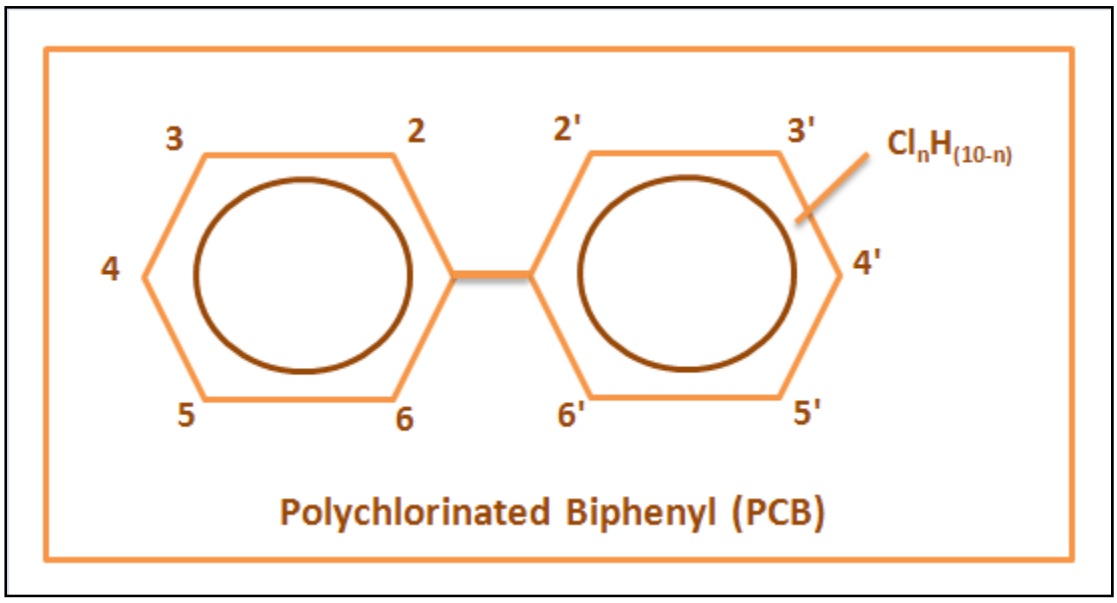


Figure 1: Structure of Polychlorinated Biphenyl. The structure can have up to 10 chlorine atoms attached to it. Adapted from (Polychlorinated Biphenyls, 2007)

The 209 compounds are named and categorized according to how many chlorine atoms exist on the biphenyls, and how the chlorine atoms are counted. Each different combination has a different name, and is said to be a congener (Polychlorinated Biphenyls, 2007). When first manufactured, the congeners were mixed together, making a chemical with a certain percentage of chlorine by weight. This was needed for different coolants, but has also made it hard to determine the exact composition of the compound found in the environment.

Since their production in 1929, PCB's have made it to almost every corner of the Earth. The structures of these compounds make them incredibly stable and they do not dissolve in water. They are resistant all forms of natural decay and last in the environment for many years

(Colborn, Dumanoski, & Myers, 1997). This stability allows the molecules to be carried by ocean currents, winds, and through groundwater. Throughout time, the amount of PCB's in a given location builds up, and the animals around that area, including humans, are affected. The molecules make there way into the body, and are stored in fatty tissues. Polychlorinated Biphenyls have even been found in the fat of polar bears in the Arctic (Colborn, Dumanoski, & Myers, 1997).

Polychlorinated Biphenyls were first made in 1929 by the Swann (later Monsanto) Chemical Company. The market for these molecules was great. For the first time, the government began to require non-flammable coolants in transformers. Polychlorinated biphenyls fit that description. Eventually PCBs were used as lubricants, hydraulic fluids, cutting oils, liquid seals, rubber, paints, varnishes, inks and pesticides (Colborn, Dumanoski, & Myers, 1997).

Early testing of PCB's showed no known health or environmental effects. In 1964 however, the chemist Soren Jensen began testing human blood for DDT. Although he did find DDT, he also found a chemical component that was not known at the time. He soon found that this chemical was a type of PCB. He found it everywhere. It was in wildlife specimens, in the environment, and in hair samples from Jensen's own family (Colborn, Dumanoski, & Myers, 1997). When other scientist began looking, they too found PCB's in the soil, water, mud of lakes, rivers, estuaries, in the ocean, and in the wildlife that live in these habitats (Colborn, Dumanoski, & Myers, 1997). Polychlorinated Biphenyls love sediment, and bind to it stronger than to other particles (such as water). As PCB's find their way to rivers, they settle to the bottom with the sediment, and then the particles are eaten by organisms. The PCB's are then transferred organism to organism, reaching fish, birds, and humans that eat the fish. This is a process called bioaccumulation (Resources, 2006).

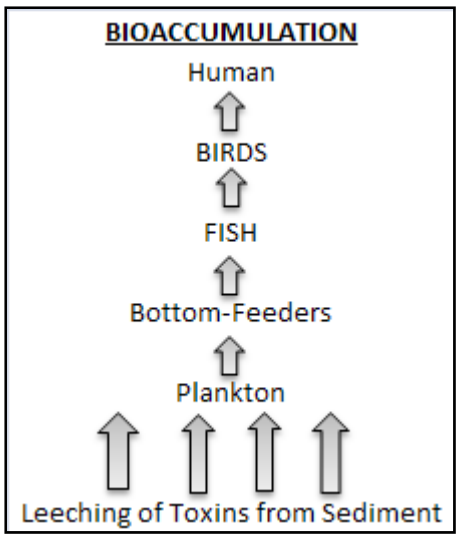


Figure 2: Bioaccumulation proceeds from the sediments in rivers, up through the food chain, ending in humans. Adapted from (Resources, 2006).

The above diagram shows how easy it is for an animal that does not live near PCB contaminated land to pick up the molecule in their blood. For example, many Polar Bears have been tested and determined to have very high levels of PCBs in their blood. They live the Arctic, far from any manufacturing plants. As noted above however, birds that live near plants during the south then fly north and are preyed up by the polar bear. Once eaten, the polar bear ingests the PCBs that were in the fat tissues of the birds. The same is true for humans. If a fish or bird has been contaminated with PCB's and then eaten, those molecules are transferred to humans (Colborn, Dumanoski, & Myers, 1997).

Due to the versatility of polychlorinated biphenyls, they are found throughout the world. The first reported contaminations of PCBs were in Asia. The people ate contaminated rice oil, and most people became sick. Of the women exposed at the time, their infants showed slower developmental growth, low birth weight, and low IQ's. It has been suggested that women who have been exposed to PCBs and breast feed with also have developmentally inept children. The suggested mechanism for the developmental delay is that of maternal thyroid function in

children (Grandjean, 2006). Another place where PCB contamination is bad has been in the Great Lakes region of the United States. Many fish have been found to be contaminated in those waters (Colborn, Dumanoski, & Myers, 1997).

The United States and other developed countries banned the use of PCBs in 1976. However, the damage to the environment had already been done as 3.4 million pounds of PCBs had been produced worldwide. Most of this was disposed of improperly, and thus the cleanup continues. The only known ways to take care of PCBs are incineration or dechlorinating the compounds so that they can decay (Colborn, Dumanoski, & Myers, 1997).

Polychlorinated Biphenyls are a proven neurotoxin. When exposed, the development of infant and child brains is severely impaired. Due to this evidence, there may also be a correlation between PCBs in the environment and the rise in neurodegenerative diseases such as autism, ADD, cerebral palsy, and muscular dystrophies.

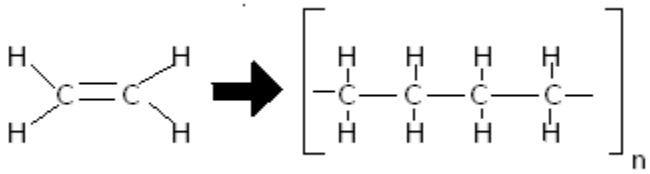
Polyvinyl Chloride

The origin of polyvinyl chloride can be interpreted in many ways. Polyvinyl chloride, also known as PVC, was first formed in 1872 but a German chemist named Eugen Baumann (The Standring Brothers), but it wasn't until 1926 that it became useful. Baumann never patented his invention of PVC, so Waldo Semon was given the credit.

Waldo Semon was an assistant chemistry teacher at the University of Washington. He also did research for B.F. Goodrich Company. In 1926, Semon was assigned to develop a new synthetic adhesive (Jezek, 2006).

While Semon was experimenting on forming a new manmade adhesive, he started to run out of bromide, which is an essential element in adhesive. As a substitute, Semon tried chloride.

The chemicals, heat, and chloride formed polyvinyl chloride in powder form. Once mixed with hot solvent, the powder turned into a rubbery gel substance that has become known as polyvinyl chloride material (Jezek, 2006). Figure 1 shows the structures of the monomer vinyl chloride and of the polymer PVC.



Monomer
Vinyl Chloride

Polymer
Polyvinyl Chloride

Figure 3: Structures of vinyl chloride and PVC (Jezek, 2006)

The PVC that Baumann discovered was nothing like the material Semon developed. Semon made PVC elastic as well as resilient (Massachusetts Institute of Technology, 1999). It could be molded into any shape and was resistant to moisture and humidity (Lenntech Water Treatment & Air Purification Holding B.V., 2008).

PVC manufacturing began with making heels for shoes and coatings for various products, such as the shower curtain, raincoats, and umbrellas. In the 1940s, PVC became a vital material for the production of war products (Jezek, 2006).

The properties of PVC make the material very versatile. PVC is a thermoplastic material. "*Thermoplastic materials are those that can be melted again and again. These materials can be heated to a certain temperature and will harden again as they cool"* (Lenntech Water Treatment & Air Purification Holding B.V., 2008). PVC can be clear or colored and rigid or flexible. Nowadays, PVC is found in construction (window frames, shutters, pipe cabling and coating),

records (aka vinyl records), industrial sectors, healthcare, automotive, food packaging, and toy production. Figure 2 displays the different uses and quantities of PVC found in construction, while Figure 3 illustrates where PVC can be found in all the various areas in the U.S. and Canada.

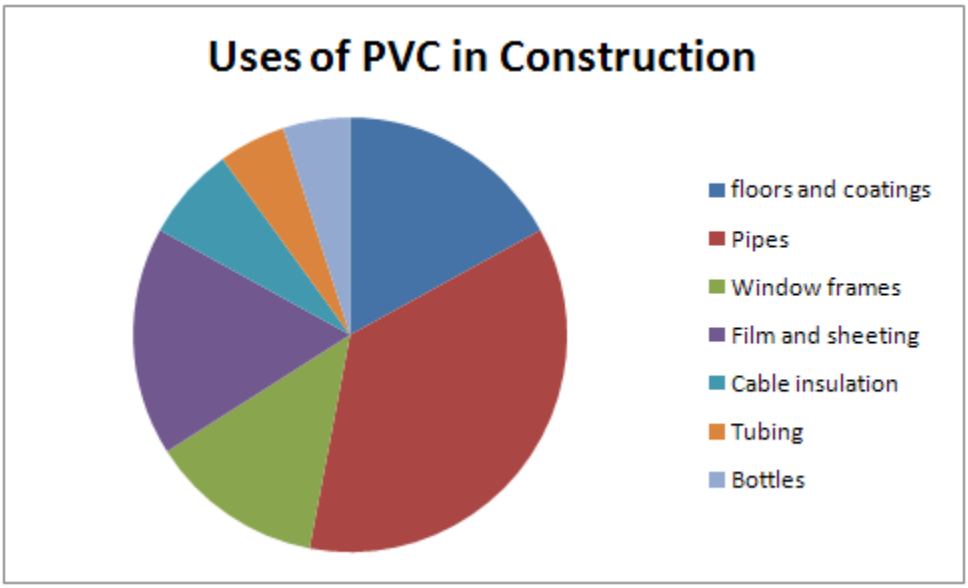


Figure 4: Where PVC is found in construction (Lenntech Water Treatment & Air Purification Holding B.V., 2008)

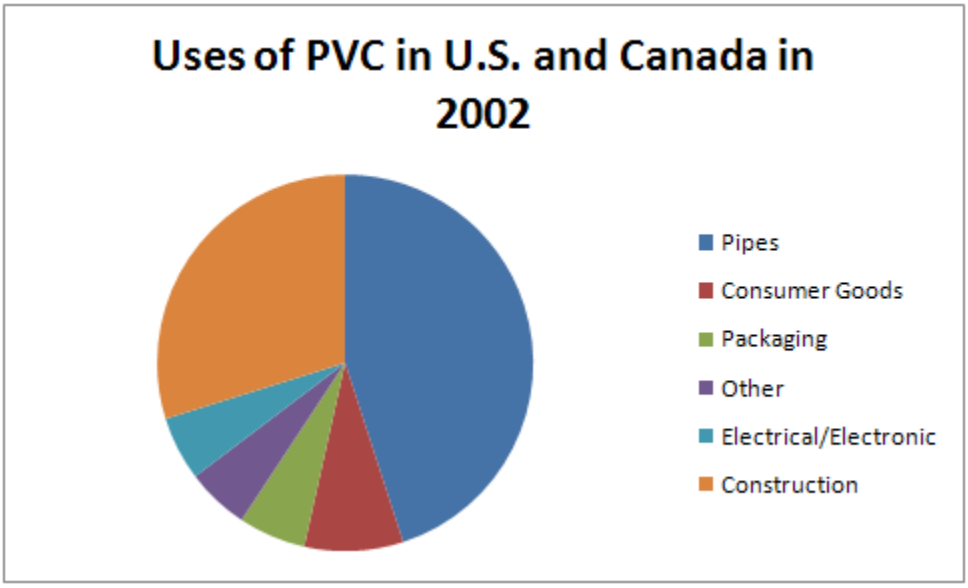


Figure 5: Uses of PVC in U.S. and Canada in 2002 (Belliveau&Lester, December 2004)

PVC is the second most used plastic in the world due to its ideal properties (Polyvinyl Chloride, 2006). By adding additives to the plastic, properties such as flexibility, color, durability, and flam resistance. Table 1 lists the additives and the properties that they aid in achieving.

Table 3: list of additives and the properties that can be achieved for PVCs

Additives	Properties
Anti-oxidants and other stabilizers	slow down rate of degradation of polmer by oxygen, heat, visible light, or UV radiation
Compatibilizers	enables mixture of other plastics and helps in recycling
Flame retardants	reduce flammability
Pigments	adds color
Plasticisers	produces flexibility
Impact modifiers	absorbs shock without damage
Fillers	add bulk

With such high quantities of PVC being produced, it is a tragedy that with each production, usage, burning, burying, and disposal of PVC, toxins are released. This creates an environmental and health concern. The toxins that are associated with PVCs are a direct result of the life cycle of PVC plastic. They include chlorine, vinyl chloride monomer, ethylene dichloride, phthalates, lead, cadmium, tin, bisphenol A, and dioxins (Belliveau & Lester, December 2004). The life cycle is depicted in Figure 4.

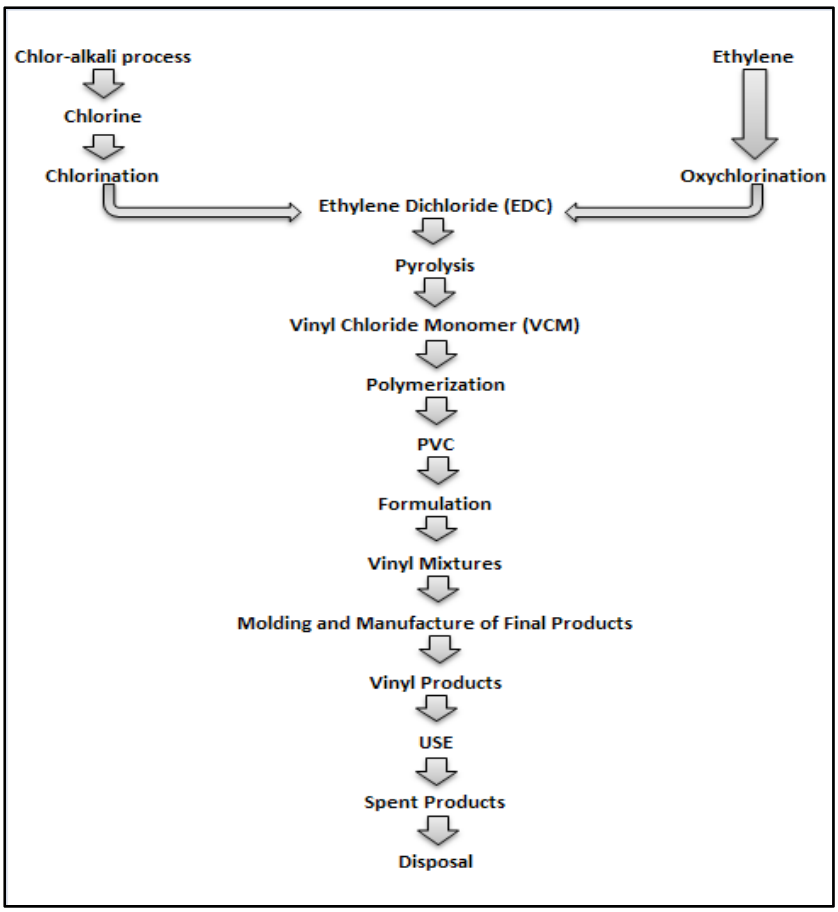


Figure 6: Life cycle of PVC plastic, in which releases of toxins can be viewed (Belliveau & Lester, December 2004)

The production of PVC involves the use of highly polluting chlorine. Chlorine is hazardous because it reacts with carbon. Carbon is an essential element for living organisms. It combines with hydrogen, oxygen, and nitrogen to produce molecules vital for life, such as DNA, proteins, hormones, sugars, starches, and fats. When chlorine reacts with the carbon, the original molecules that are vital for life become altered and no longer function the way they should (Belliveau & Lester, December 2004).

Chlorine is also involved in the process of making vinyl chloride monomer. Figure 5 demonstrates the process of how vinyl chloride monomer, also known as VCM, is produced.

Although there are no studies that directly link VCMs to defects in children, there have been cases on animals that show a link between VCM and affects on growth and development (Department of Health and Human Services, 2006).

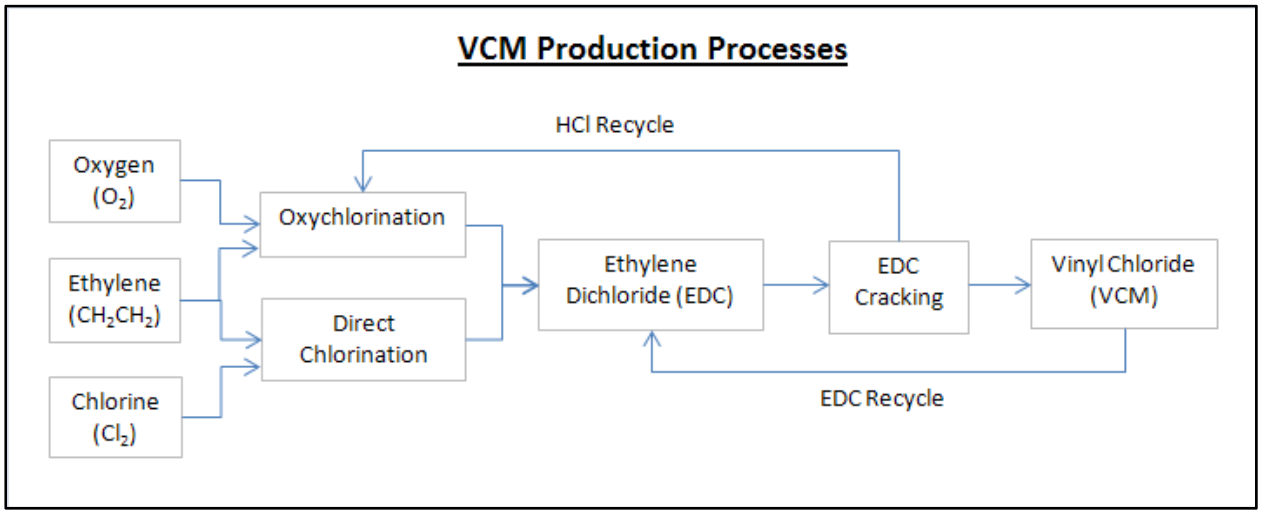


Figure 7: The process of VCM Production (Department of Health and Human Services, 2006)

The U.S. Department of Health and Human Services has labeled vinyl chloride monomer as a carcinogen (Department of Health and Human Services, 2006). A carcinogen is any agent, such as a chemical, radiation, and virus, that cause or initiate cancer (Encyclopaedia Britannica Online , 2008clopaedia Britannica Online). There are studies showing that workers who breathe in vinyl chloride have an increased risk for cancer, particularly brain, lung, and certain cancers of the blood (Department of Health and Human Services, 2006).

Along with vinyl chloride, ethylene dichloride is also a toxic formed during the production of PVC. Ethylene dichloride is a clear, pleasant smelling, liquid that is not found naturally in the environment. Fortunately, ethylene dichloride is not considered a carcinogen, and studies have found no connection to birth defects. However, ingestion and inhalations of

ethylene dichloride is believed to be harmful to the lungs, kidneys, and neurological, cardiovascular, and immune systems (The Dow Chemical Company, 2007).

Of all the quantities of ethylene dichloride produced, 95% is used to make vinyl chloride monomer, which in return, 99.5% is to produce PVC. Figure 6 illustrates the connection between ethylene dichloride, vinyl chloride monomer, and PVC.

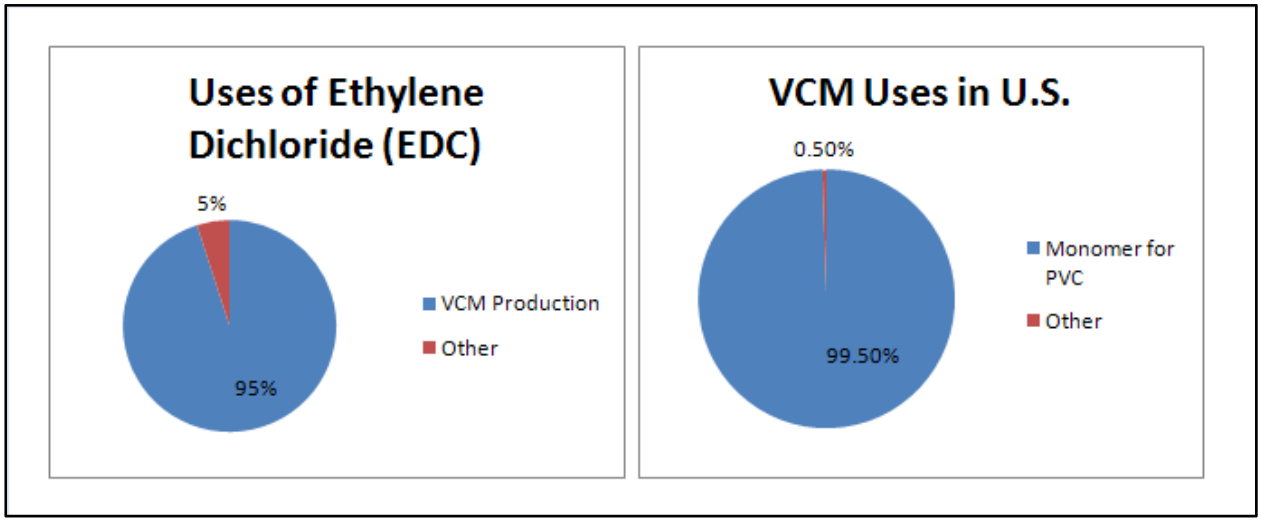


Figure 8: Quantities of toxins used in the production of polyvinyl chloride

Once PVC is manufactured, toxins are released during usage. Phthalates are examples of these chemicals that are destructive to the environment and health. Phthalates are added to plastic, or PVC, to make the products soft. They can be found in a variety of items including plastic bags used to hold blood, plasma, and intravenous fluids, catheters, exam gloves, children's toys, shower curtains, and cosmetics (Conis, 2007). The two main types of phthalates are di(2-ethylhexyl) phthalate, also known as DEHP, and diisononyl phthalate, known as DINP.

DEHP and DINP are colorless, oily liquids used to plasticize PVC. Figure 7 illustrates the structure of DEHP. Figure 8 depicts the structure of the DINP, which is the primary plasticizer in children's products (Babich, 1998).

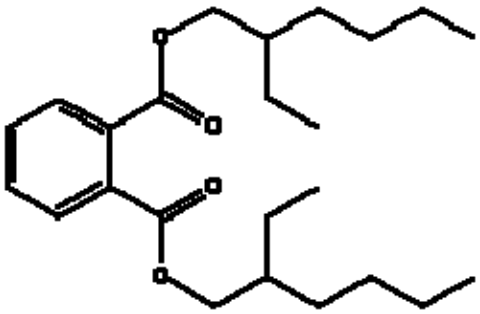


Figure 9: **DEHP structure**

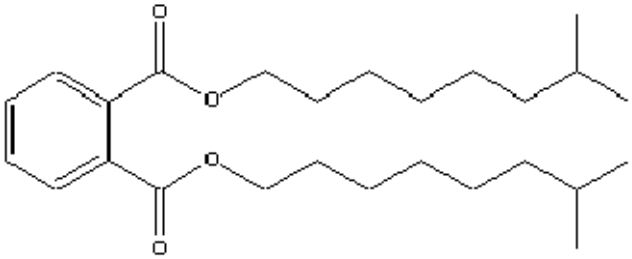


Figure 10: **DINP structure**

The main routes for human exposure to DEHP and DINP are inhalation, ingestion, dermal contact, and through medical procedures (National Toxicology Program; Department of Health and Human Services). In small children, exposure is due to sucking on or skin contact with plastic toys (Agency for Toxic Substances & Disease Registry, 2002).

Patients are introduced to the additives through dialysis treatments and blood transfusions. Many of the tubes and containers that hold the intravenous fluids are coated with DEHP or DINP. Workers in plants are exposed to phthalates through inhalation during the

manufacturing, formulations, and processing of plastics. (National Toxicology Program; Department of Health and Human Services).

The health risks of DEHP and DINP exposures have been researched through tests with rats and mice. Since the absorption and breakdown of the phthalates are not the same, it is unsure these health effects occur in humans (Agency for Toxic Substances & Disease Registry, 2002). High levels of both of the phthalates resulted in damage in liver and increased incidences in hepatocellular adenoma and carcinoma. Only under high amounts of DEHP in rodents were birth defects, fetal death, and reproductive impairment experienced (Babich, 1998).

Bisphenol A is another chemical used to produce plastics (EFSA, 2008). Figure 9 displays the structure. To make polycarbonate plastic, bisphenol A molecules bind together through ester bonds, which are unstable, to form a polymer. Polycarbonate plastic are transparent, rigid plastic that is used to make returnable beverage bottles, infant bottles, tableware, and storage containers (EFSA, 2008). Changes in temperatures and pH levels cause the bond to break, releasing the bisphenol A into food and beverages in contact (Environment California).

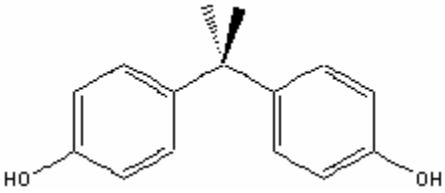


Figure 11: **Bisphenol A structure**

Bisphenol A is a health concern because it is an endocrine disrupter; it interacts with the hormone systems of the body. The toxin can imitate the female hormone estrogen (EFSA, 2008). A number of animal studies have found Bisphenol A linked to diabetes, obesity, hyperactivity, and infertility. It is also associated with "reduced sperm count, impaired immune system

functioning, increases in prostate tumor proliferation, altered prostate and uterus development, insulin resistance, alteration of brain chemistry, early puberty, and behavioral changes" (Oregon Environmental Council).

When PVC is burned in fires, incinerators, and stoves, the byproduct, dioxin, is released (Greenpeace International). Figure 10 shows the structure of the toxin.

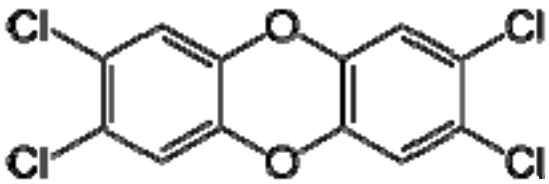


Figure 12: Structure of Dioxin

Through animal research, dioxin is shown to be a complete carcinogen. The toxin can also cause peripheral nerve damage, liver damage, and a skin disorder called chloracne. The suspected health effects include respiratory and prostate cancer, multiple myeloma, porphyria cutanea tarda, diabetes, reduced sperm count, and neurobehavioral development effects in infants (Agency for Toxic Substances and Disease Registry; Division of Toxicology and Environmental Medicine, 2006).

From production to disposal, PVC releases toxins that have the potential to cause serious health effects to children and adults. Much research has been done on animals, but it is hard to link the effects shown with those in humans. Possible health concerns that are suspected to be linked with PVC products include carcinoma, liver damage, and reproductive and neurobehavioral effects.

Results

In order to view if there were any correlations between the environmental toxins and the neurological disorders, data was collected during the years 1990 through 2007. The toxins that were analyzed included lead, mercury, PCBs, and PVCs. The only neurobehavioral disorders that had information available on prevalence throughout the years were autism and ADHD.

The amount of lead exposed to children was directly related to the level of lead present in their blood. It has been found that blood lead levels at levels of 10μg/dL or more have profound effects on the neurological and behavior development of children (Centers for Disease Control and Prevention, 1997). The ages that were examined were six years and younger. This is because the brain is more likely to be influenced by environmental toxins at an earlier age. The data was collected from various states from 1990 to 2007. It was recorded as the percentage of children less than six years of age with blood lead levels greater than 10μg/dL.

PCBs are another form of dangerous environmental toxins. They are of concern because they are can remain in the environment for many years. They are resistant to all forms of natural decay (Colborn, Dumanoski, & Myers, 1997). The collected emissions were recorded in millions of pounds from 1990 to 2007. All data was obtained and adapted from Environmental Protection Agency's Toxic Release Inventory. The data was based on on- and off- site disposal of or released for all the facilities within that specific state. On-site disposal or releases included emissions to the air, discharges to bodies of water, disposal at the facility to land, and disposal in underground injection wells. Off-site disposal or releases were characterized as discharges of toxic chemicals to the environment as a result of a facility's transfer of toxic waste to an off-site location for disposal or other release (Environmental Protection Agency, 2008).

Mercury has been a chemical associated with autism since the studies on thermisol vaccines began. Data on the emissions of mercury throughout the U.S. was difficult to locate. Some states and regions provided no yearly information and therefore were left blank. This provided a segmented set of data. Mercury was analyzed as emissions in thousands of pounds. The data is a collection of on- and off- site disposal and other releases for each state per year, from 1990 to 2007 (Environmental Protection Agency, 2008).

The last environmental toxin that was examined was PVC. Data was scarcely provided for individual states per year. This could be because PVC is toxic throughout its whole life span. Toxins are released during production, usage, and disposal of PVC. This could make it difficult to record the prevalence. However, data was retrieved for the entire U.S. for certain years between 1990 and 2007. Unfortunately, this does not provide much validity to correlate with the neurological disorders of interest. The data was recorded as the amount of PVC consumed in billions of pounds (Center for Health, Environment, and Justice).

After graphing the environmental toxin emissions, it was necessary to obtain and produce similar graphic representation of the neurodevelopmental disorders that were being analyzed. Although several disorders were described previously, only two provided the number cases throughout the time period; autism and ADHD. The percentage of children between the ages of 3 and 17 who were diagnosed with ADHD was obtained for the entire U.S. between the years of 1990 and 2007. This set of data could be beneficial for making generalized assumptions. The percentages of children between the ages of 6 and 22 with autism were found for each state between the years 1990 and 2007.

The data collected for lead, mercury, PCBs, and autism was organized based on regions of the U.S. This allowed for a more precise analysis of the environmental toxins and disorders

and their correlation. The U.S. was divided into 10 regions, adapted from the U.S. Environmental Protection Agency website. Table 4 lists the states that are located in each of the regions of the U.S.

Table 4: List of the states within each region on the U.S.

EPA Regions of the United States									
Region 1	Region 2	Region 3	Region 4	Region 5	Region 6	Region 7	Region 8	Region 9	Region 10
CT	NJ	DC	AL	IL	AR	IA	CO	AZ	AK
MA	NY	DE	FL	IN	LA	KS	MT	CA	ID
ME		MD	GA	MI	NM	MO	ND	HI	OR
NH		PA	KY	MN	OK	NE	SD	NV	WA
RI		VA	MS	OH	TX		UT		
VT		WV	NC	WI			WY		
			SC						
			TN						

Figure 13 illustrates the results of region 1, which includes Connecticut, Massachusetts, Maine, New Hampshire, Rhode Island, and Vermont. The graphs include the prevalence of autism, percentage of children with high blood lead levels, pounds of mercury emissions, and pounds of PCB emissions.

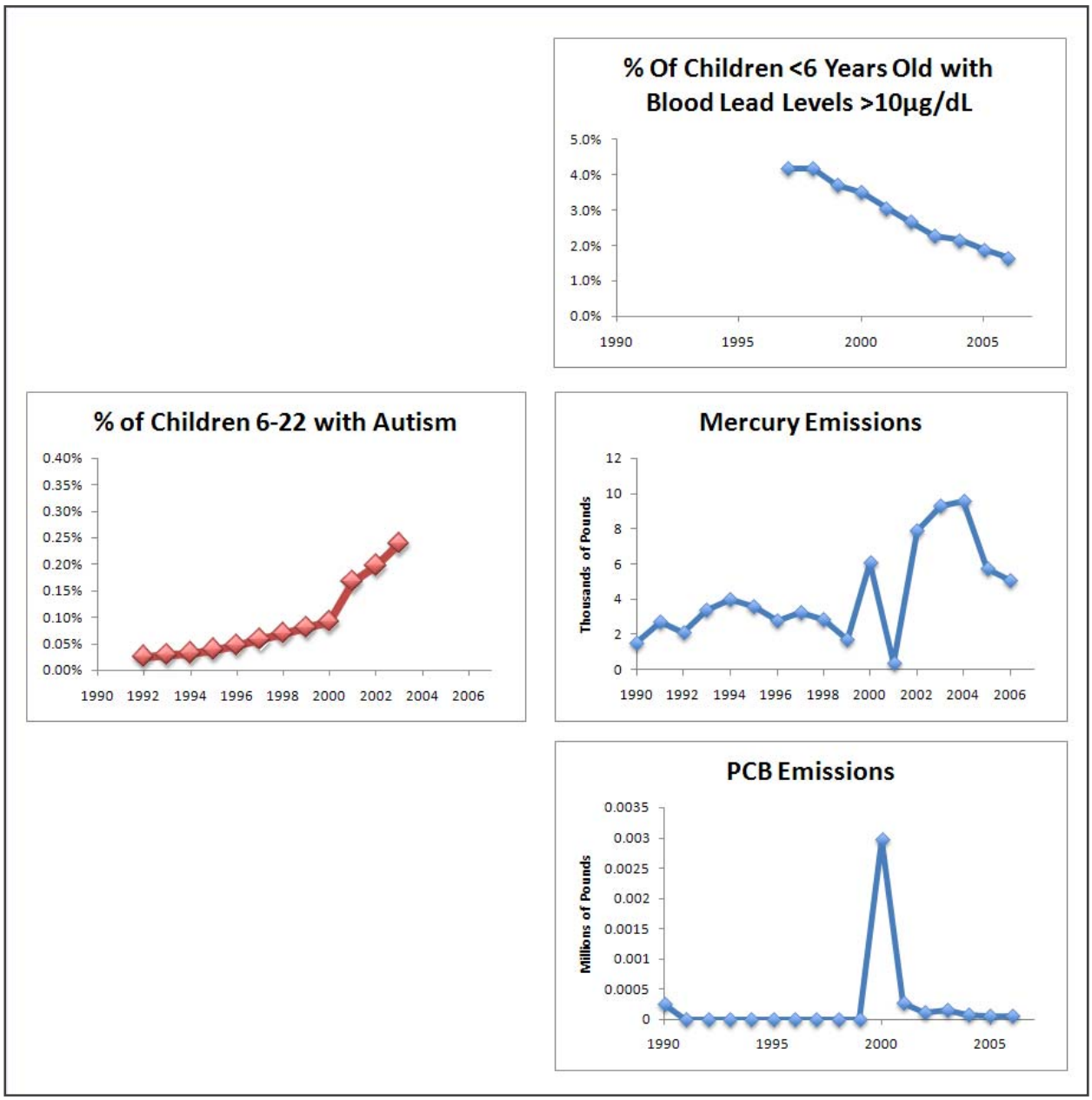


Figure 13: Region 1; prevalence of Autism against amount of neurotoxins

Region 2 includes New Jersey and New York. The data from autism and the neurotoxins available, which were PCB, mercury, and lead, were graphed in Figure 14.

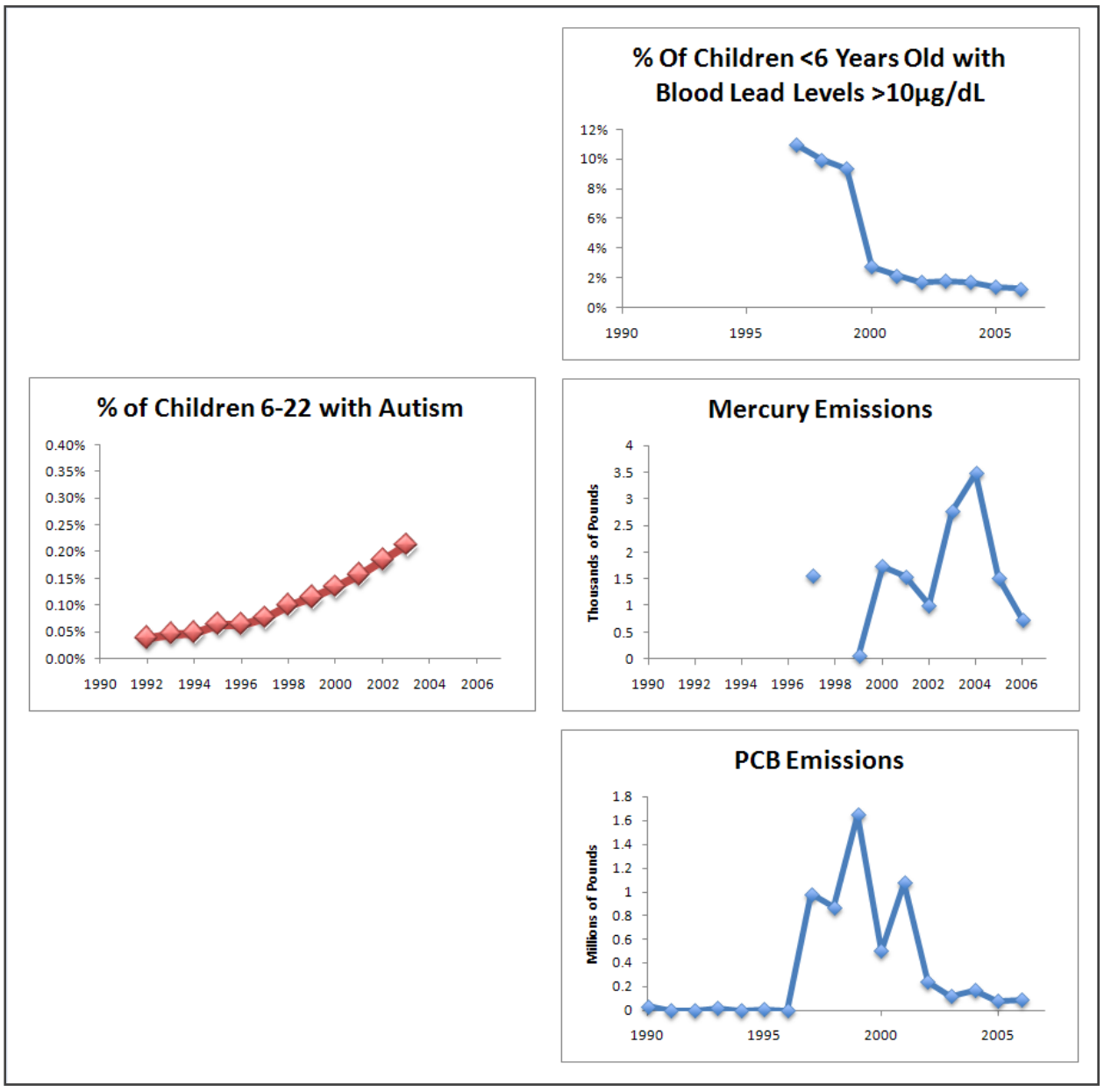


Figure 14: Region 2; prevalence of Autism against amount of neurotoxins

Figure 15 displays the trends of autism, lead, mercury, and PCB in graphs for region 3. In region 3, the states are Delaware, Maryland, Pennsylvania, Virginia, and West Virginia, as well as the District of Columbia.

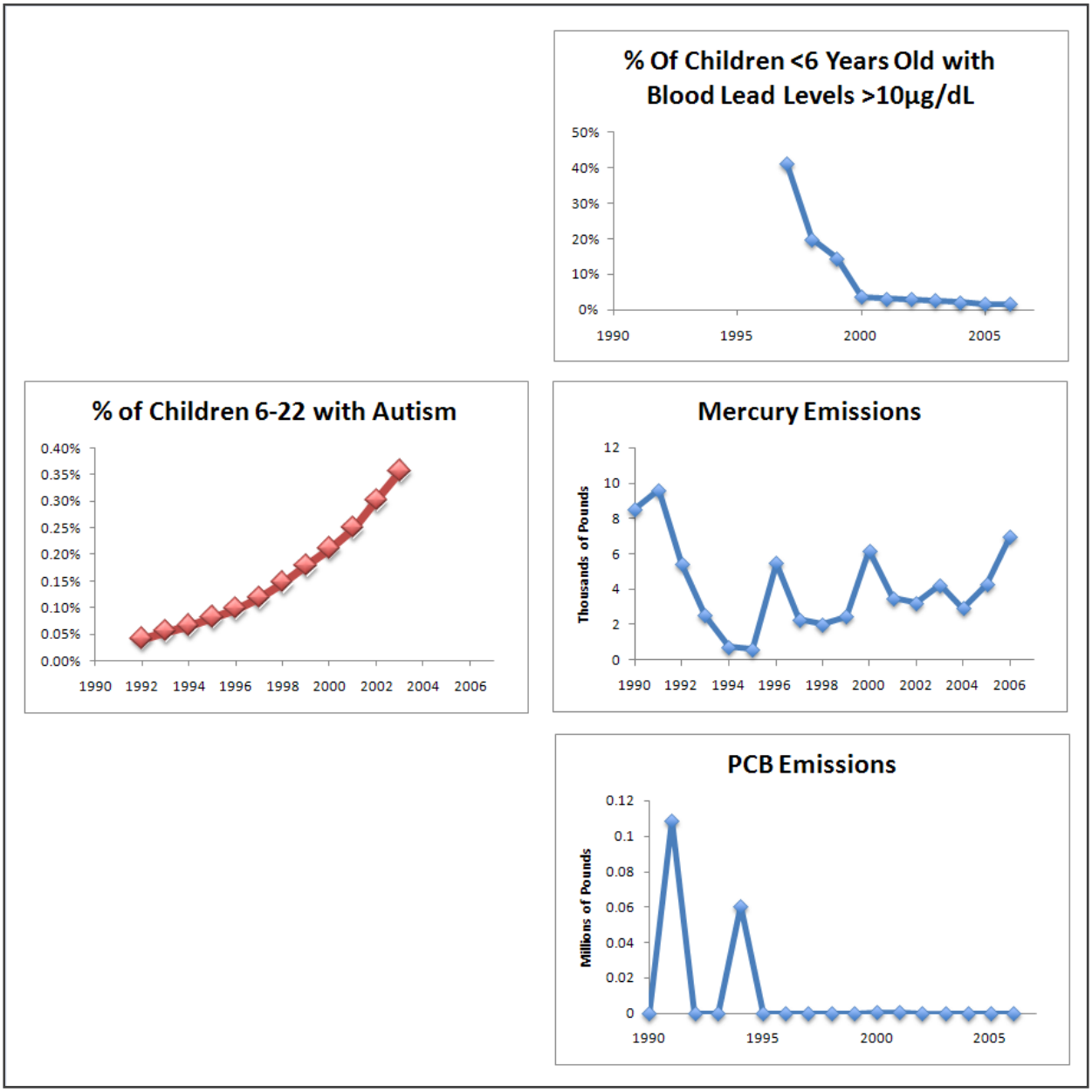


Figure 15: Region 3; prevalence of Autism against amount of neurotoxins

Alabama, Florida, Georgia, Kentucky, Mississippi, North Carolina, South Carolina, and Tennessee make up region 4. The association between autism and the neurotoxins are shown in Figure 16.

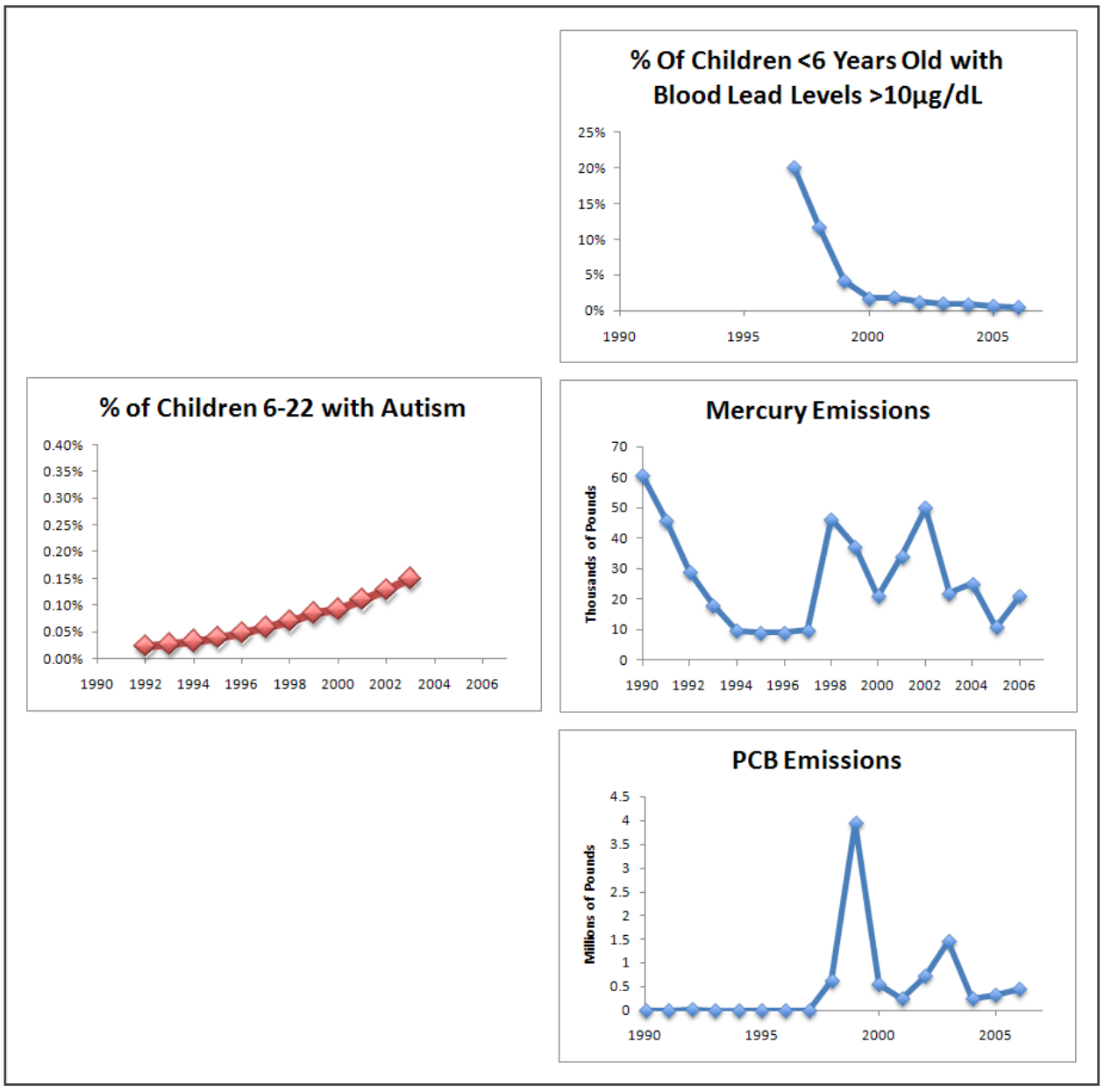


Figure 16: Region 4; prevalence of Autism against amount of neurotoxins

Region 5 comprises of Illinois, Indiana, Michigan, Minnesota, Ohio, and Wisconsin. Graphs of the data that was obtained for autism, lead, mercury, and PCB are displayed in Figure 17.

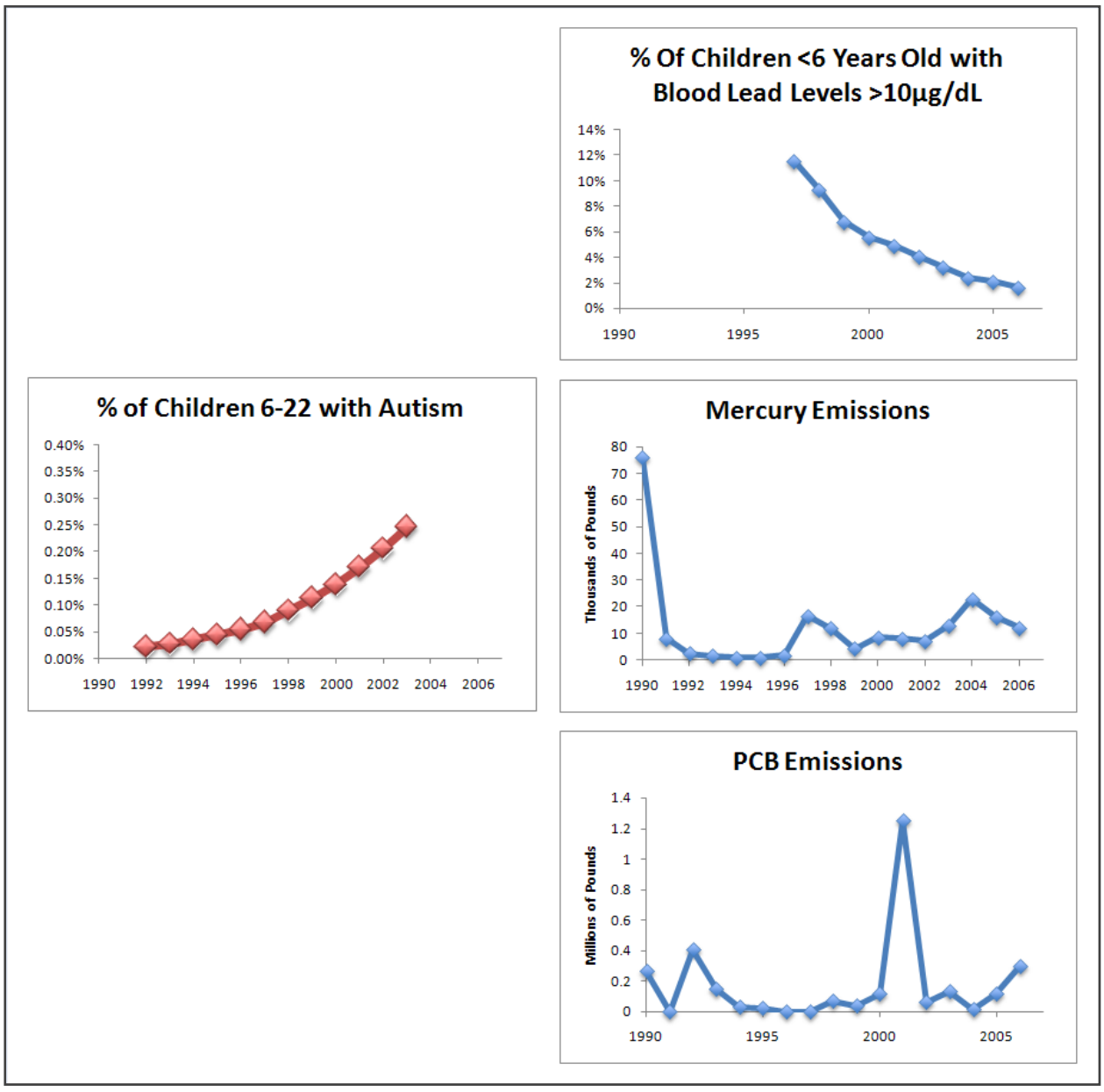


Figure 17: Region 5; prevalence of Autism against amount of neurotoxins

The states within Region 6 are Arkansas, Louisiana, New Mexico, Oklahoma, and Texas. The number of children who have high blood lead levels were used to calculated the percentage for that region. These results, along with the number of pounds of mercury and PCBs emitted, were graphed in Figure 18 next to the prevalence of autism to compare the correlations.

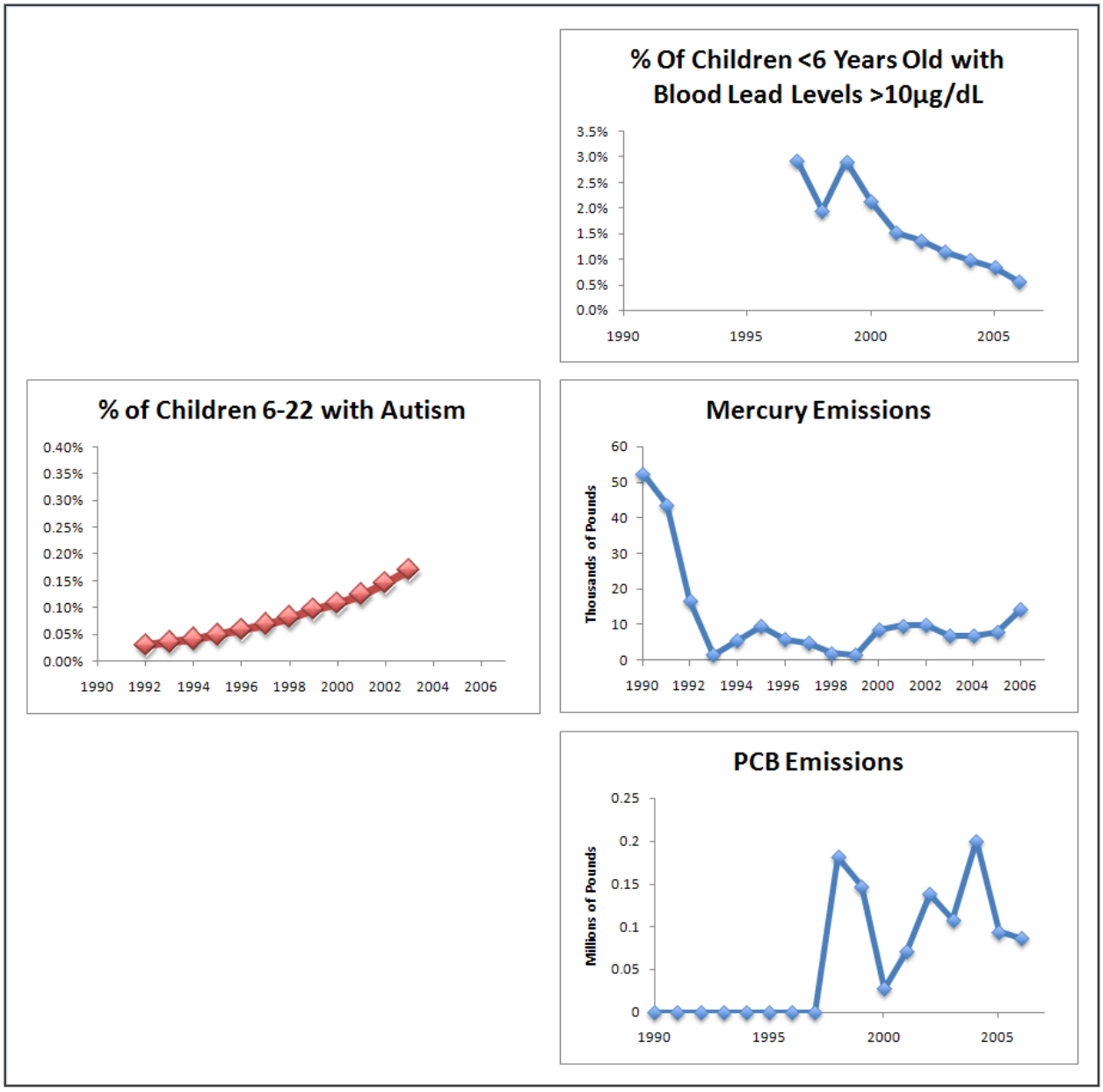


Figure 18: Region 6; prevalence of Autism against amount of neurotoxins

Figure 19 shows the trends in rising incidents of autism and emissions of mercury, the decreasing trends in number of children with high blood lead levels, and the sporadic incidences of PCB emissions. These curves are representative of region 7 includes Iowa, Kansas, Missouri, and Nebraska.

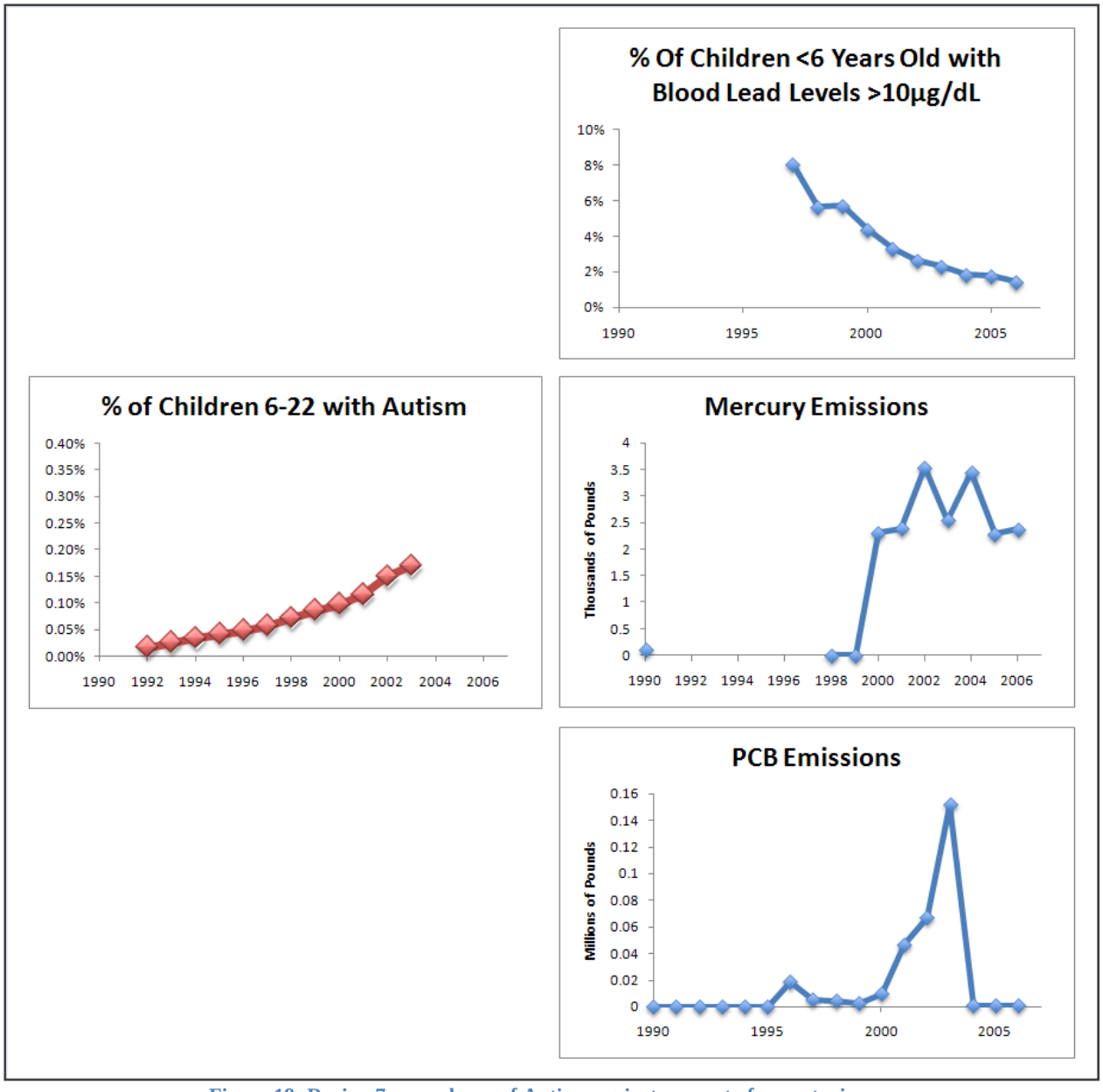


Figure 19: Region 7; prevalence of Autism against amount of neurotoxins

The trends were also graphed for region 8, and can be shown in Figure 20. Region 8 consists of Colorado, Montana, North Dakota, South Dakota, Utah, and Wyoming.

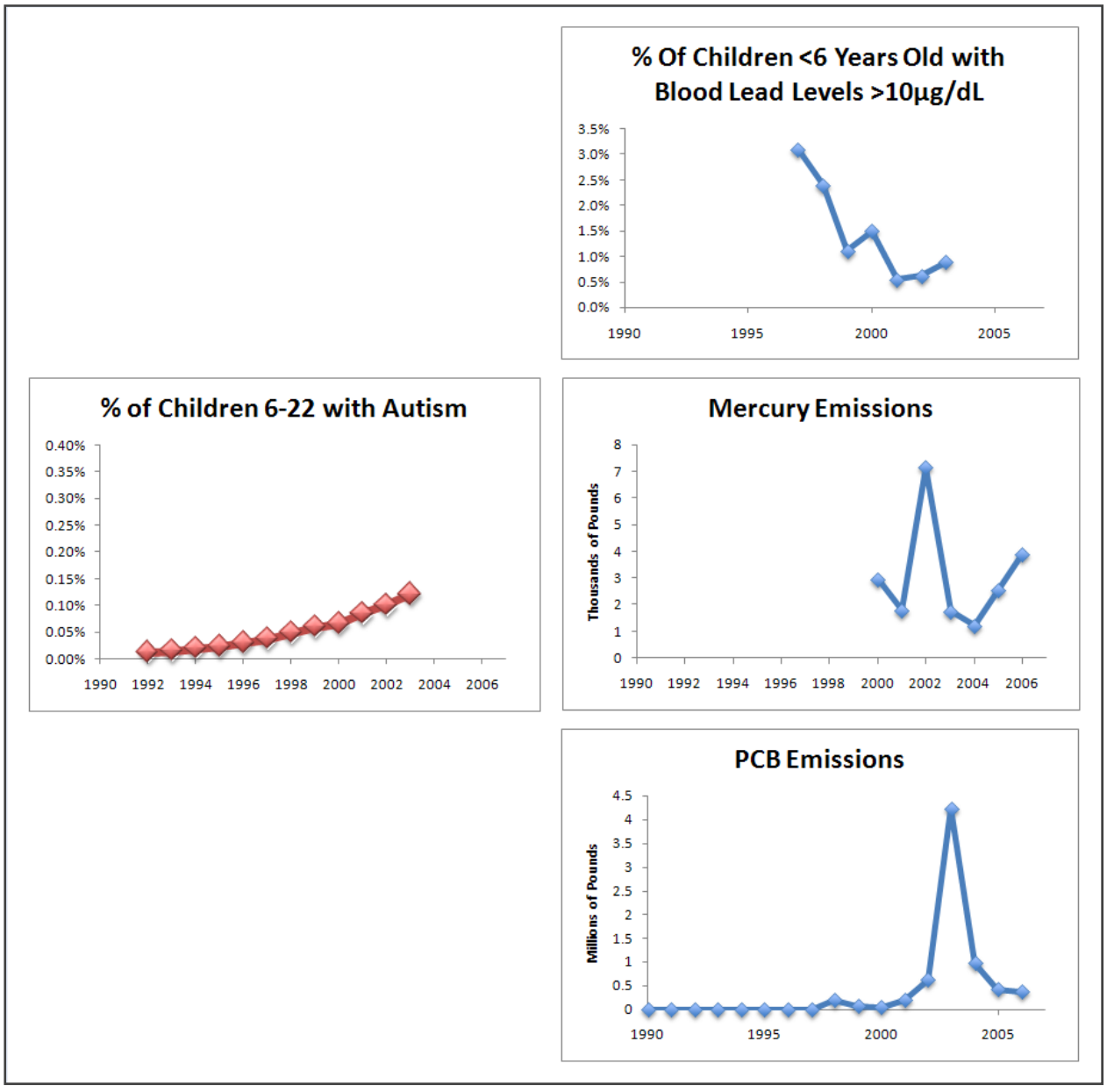


Figure 20: Region 8; prevalence of Autism against amount of neurotoxins

Region 9 consists of the states of Arizona, California, Hawaii, and Nevada. The incidents and toxic reports were also demonstrated through line graphs. This is shown in Figure 21.

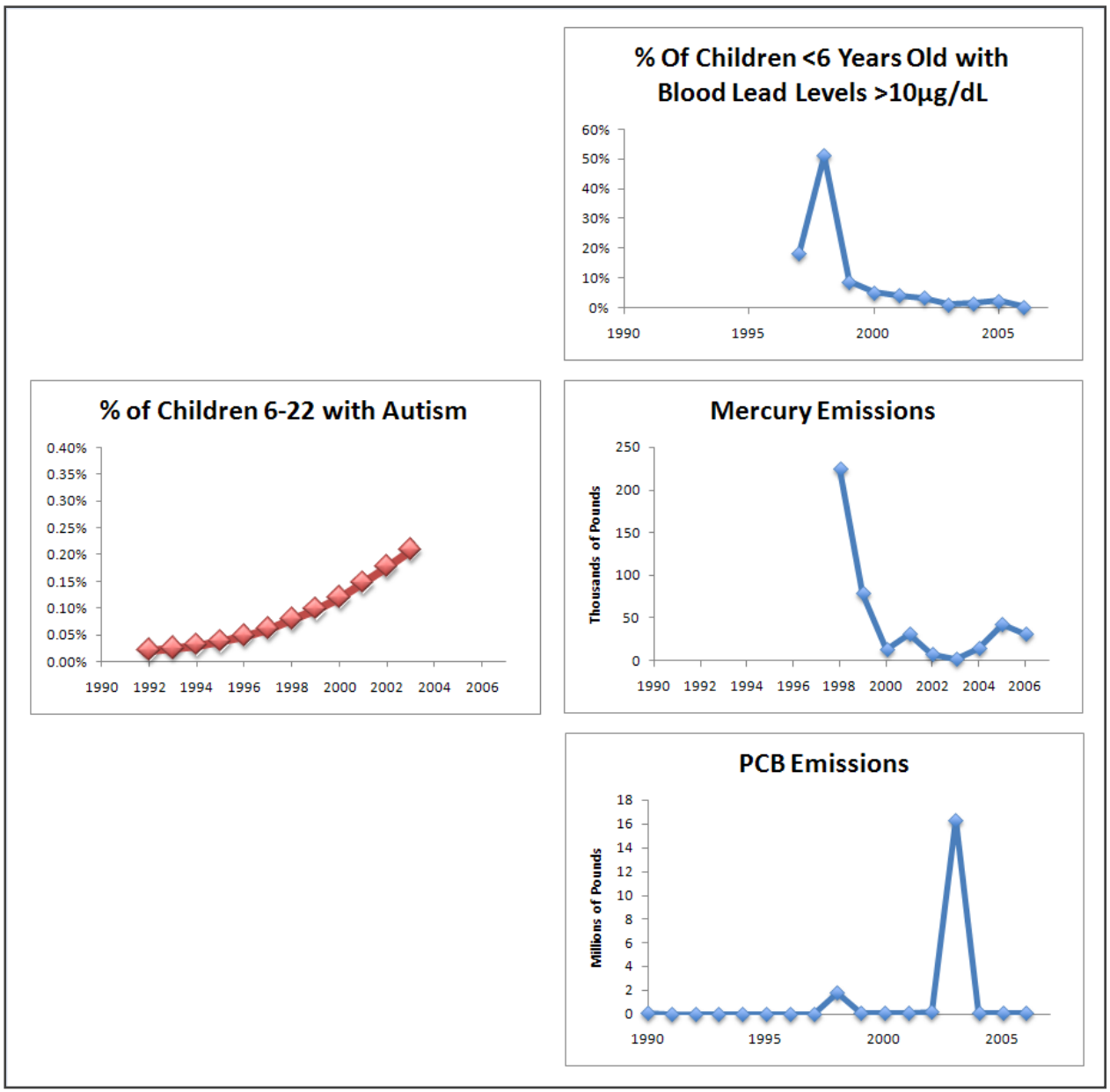


Figure 21: Region 9; prevalence of Autism against amount of neurotoxins

The last region that was analyzed was region 10, which include Alaska, Idaho, Oregon, and Washington. The graphs can be viewed in Figure 22.

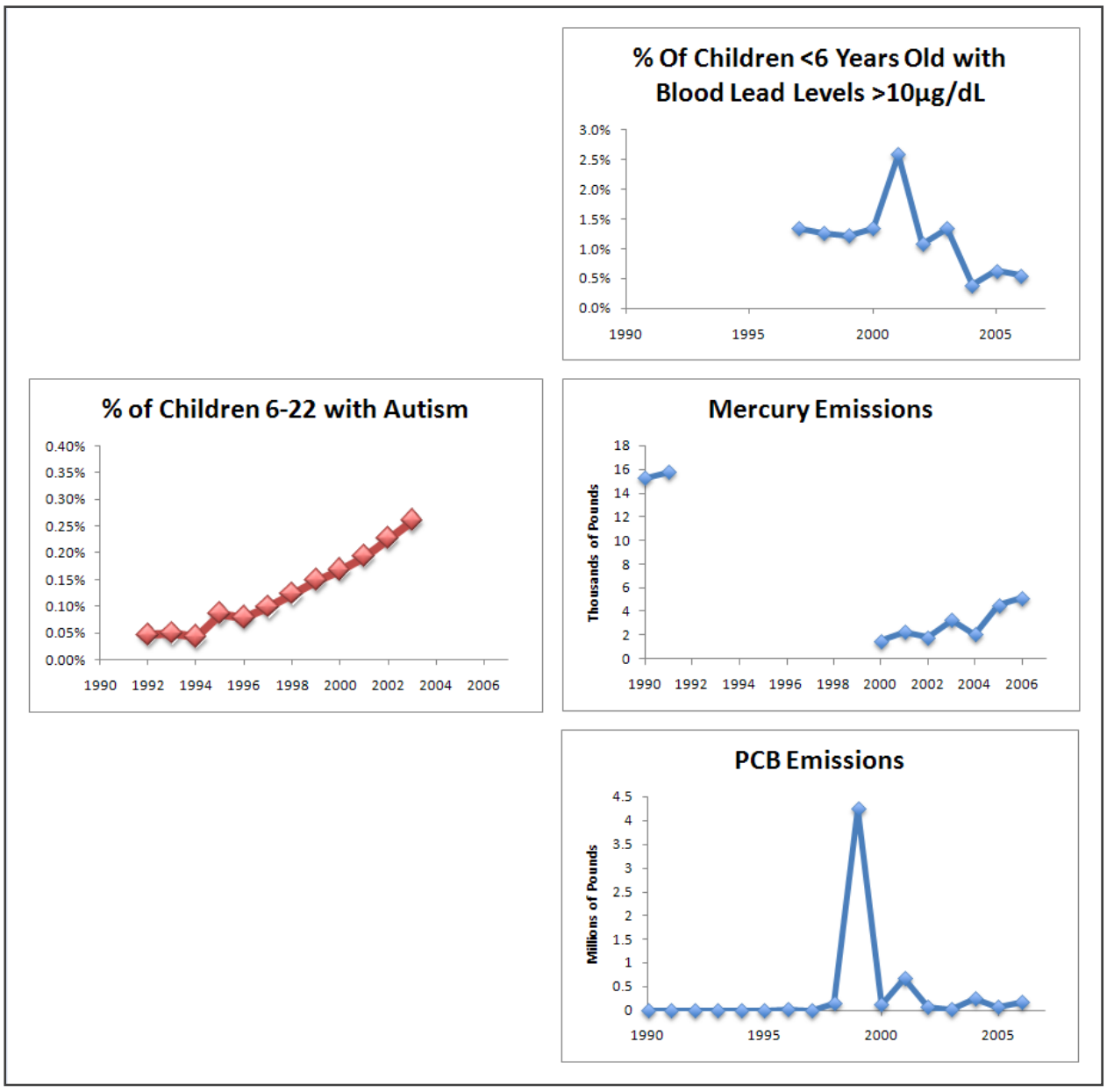


Figure 22: Region 10; prevalence of Autism against amount of neurotoxins

Lastly, the data that was collected from each region was combined to form an average for the entire U.S. Along with the percent of children with autism, the percentage of children diagnosed with ADHD was also found. These graphs were used to analyze the possible connection between the neurotoxins of interest. The pounds of PVC consumed were added into

the previous graphs of lead, mercury, and PCB. The reason that data on ADHD and PVC were not included in the individual regions is because only statistics were available for the entire U.S.

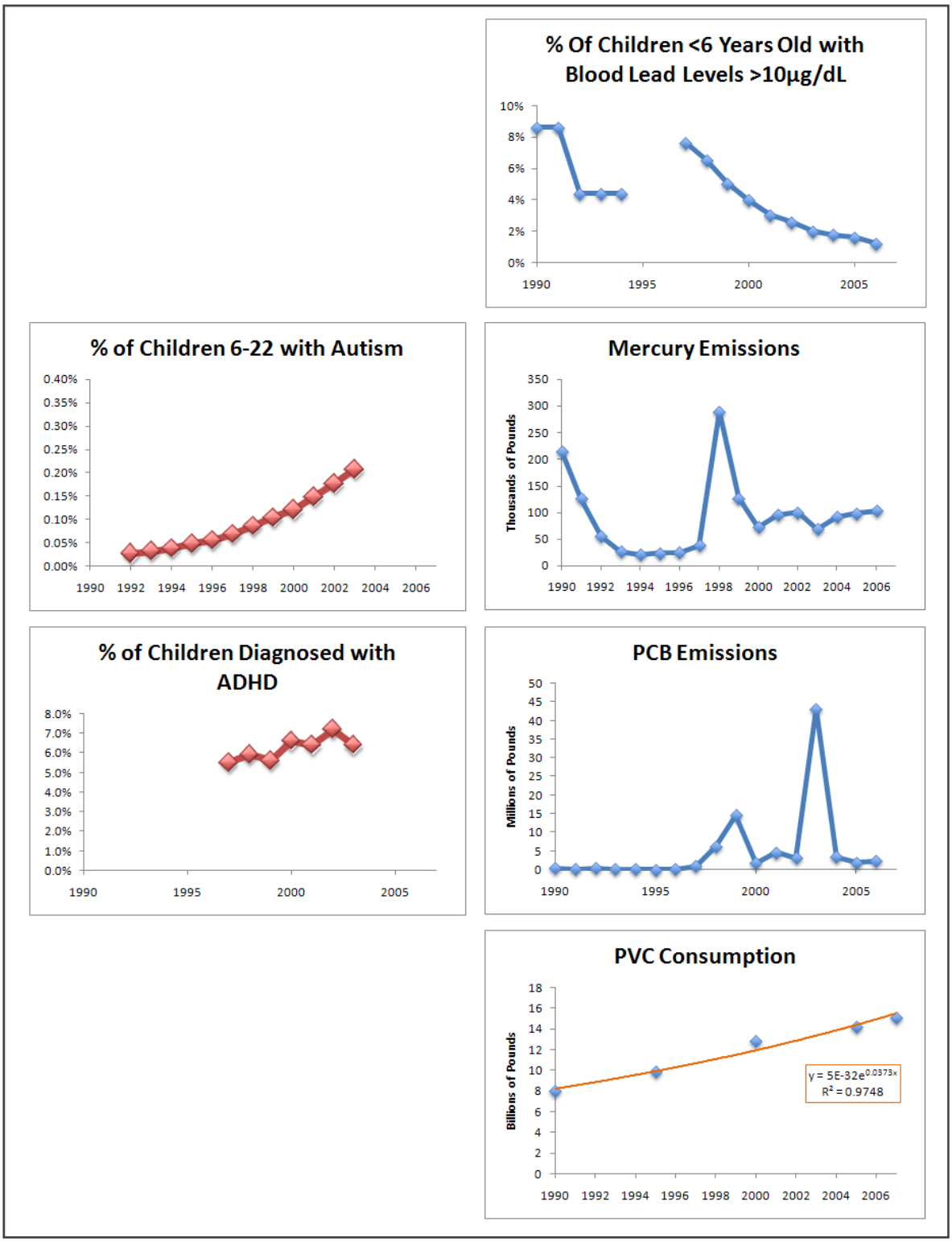


Figure 23: Whole U.S.; prevalence of autism and ADHD against amount of neurotoxins

Discussion

After analyzing neurotoxin emissions throughout the United States for the time period 1990 to 2007, several conclusions can be made based on their relation to the prevalence of neurological disorders (mainly autism). The possible existence of a cause and effect relationship was determined by comparing trends in neurological disorder rates and neurotoxin emissions. The time lapse between cause and effect can be attributed to the minimum age of the autism data (6 years). For example, if an unborn child was exposed to a neurotoxin via the mother in 1992, and this exposure resulted in the child developing autism, the child would not appear in the autism statistics until they reached 6 years of age in 1998. Each region was analyzed individually in order to look for trends dependent on geographical location and relative population.

In Region 1,the percentage of children diagnosed with autism increased by approximately 0.25% from 1990 to 2004. Over the given time period, the percentage of children with elevated BLLs decreased, thus lead levels in children isleast likely to be correlated with an increase in autism prevalence. Mercury emissions followed an upward trend, with many spikes high and low in between. The return to baseline for mercury emissions (about one thousand pounds in 1990) has not yet been achieved. The sharp increase in autism prevalence around 2001 may be attributed to the small peak in mercury emissions in 1994. The abrupt PCB emissions spike in 2000 may be attributed to states not properly reporting their emission rates. If the 2000 spike in PCB's were an indication of a link, the data for 1994 would show a sharp increase in autism cases. This appears to not have been the case.

Mercury emissions generally increased from 1999 to 2004 in Region 2. Similar to Region 1, the data is riddled with spikes in 2000 and 2004. A low is observed in 2002, with no return to baseline recorded in 2006. There is one lone data point in 1997, showing that data reporting was

inconsistent during previous years. The autism data for Region 2 is on a steady increase, which does not indicate a direct correlation between the observed autism rates and mercury emissions. The PCB emissions for Region 2 show a trend of high emissions from 1996 to 2002, reaching a peak high of 1.7 million pounds in 1999. Autism rates were not rising quite as fast during these years, thus a correlation is hard to confirm. It should be noted however that recent (in the year 2000 onward) mercury and PCB emissions mayhave yet to be translated into an increase in autism rates due to the time lapse between data sets.

The data for Region 3 shows a steady and prominent increase in autism rates relative to other regions. The percentage of children with elevated BLLs exhibits a dramatic drop from 1997 to 2000. PCB emissions in Region 3 drop down after peaks in emissions in 1991 and 1994. This is contradictory to the growing autism rates in the same area. Mercury emissions, however, seem to be fairly random and scattered within the time period of interest. Although these emissions are not steadily increasing they tend to peak, drop, and rise again. Mercury emissions may possibly be related to or at fault for the growing percentage of children with autism in these states.

The percentage of children with autism in Region 4, although not as prominent as Region 3, is still a growing concern. Again, lead seems to decrease dramatically after its peaking 1997. PCB emissions remain low until 1998 and then peak in 1999 and 2003. The percentage of children with autism appears to exhibit a linear increase. Mercury emissions rise prominently in 1998 and continue to fluctuate through 2007, showing that mercury may at least be a contributing factor to autism.

Region 5, similar to all other regions in the US, shows a steady, exponential increase in autism.Oddly, none of the three neurotoxins exhibit a corresponding increase. The percentage of children with elevated blood lead levelsconsistently decrease from 199 onward. Mercury emissions

peak in 1990 and drop dramatically the next year, staying relatively low in comparison to other regions. PCB emissions also stay low with the exception of one spike in 2001 where emissions reached approximately 1.3 million pounds, and then dropped again staying around 0.2 million pounds until 2006.

As far as mercury emissions and elevated blood lead levels in children are concerned in comparison to the increasing autism trend, the data for Region 6 are similar to Region 5 in that they appear to be contradictory. Elevated blood lead levels decrease from 1999 onward, and Mercury emissions remain at a relatively constant low after a decrease from 1991 to 1993. PCB emissions however, increase greatly in 1998 and maintain a sporadic pattern, increasing and decreasing dramatically from year to year. This large increase in PCB emissions could be related to the increase in autism in Region 6.

Region 7 exhibits a steep increase in both PCB emissions and Mercury emissions from the year 2000 onwards. Although the percent of children with autism in the region is not particularly high for the given time interval, this may be attributed to the lower population density relative to other regions. The smaller peak in PCB emissions around 1996 may correlate with the slightly larger jump in autism rates in the year 2002, foreboding a future increase in the rate of growth in the percentage of children with autism. PCB emissions recently seem to have dropped down. Although this is a good sign, the observed drop may unfortunately be due to lack of proper reporting of emissions.

The percentage of children with elevated BLLs in Region 8 exhibits a sharper decline than most other regions. Also, no data for elevated blood lead levels was reported in 2003. This was probably due to the extremely low percentage of children with elevated BLLs. It should be noted that this region has a particularly low maximum percentage of children with autism, maxing out

under 0.15% in 2003. Mercury emissions and the percentage of children with autism will most likely continue to increase in the future, possibly due to the recent spike in PCB emissions in 2003.

In Region 9, and like other regions, the percentage of children diagnosed with autism steadily increased from 1992 through 2003. The percentage of children with elevated blood lead levels began with a jump from around 20% to 50%, and back down to 10%, where it then gradually continued to decrease. This leap could possibly be an error in census, or perhaps a few years with unregulated lead releases. However, the overall decrease in percentage of children with high blood lead levels does not correlate with the increase in percentage of children with autism. It wasn't until 1998 that data for mercury emissions were first available for this region. From 1998 to 2006, the data on mercury emissions did not show a consistent trend or pattern. These results from the mercury toxins show no relation with the steadily increasing percentage of children with autism in Region 9.The PCB emissions also did not appear to exhibit a connection with the rising percentage of children diagnosed with autism. The data pertaining to PCB emissions remained relatively low with an occasional dramatic increase, lasting only a couple years. Overall, the data for Region 9 was fairly inconclusive and difficult to interpret.

In Region 10, the percentage of children with autism remained constant at about 0.05%between 1992 and 1994.This was followed by a slight jump to 0.1%, where it then continued to gradually increase up to 2003. No consistent trend exists in the percentage of children with elevated blood lead levels for this region.The PCB emissions are also too scattered to warrant a relation to the trend in autism for Region 10. The available data for mercury emissions for Region 10 were limited. However, the overall trend for 2000 to 2006 is an increasingamount of mercury emissions. This could possibly correlate to a future increase in autism.

Even though each region displays unique trends, there are a few general patterns that can be seen throughout the US. The first of these is the overall decrease in the percentage of children with elevated blood lead levels. When considered with the ever-increasing percentage of children with autism, the most straightforward conclusion is that elevated blood lead levels in children do not affect whether or not children develop autism. However, there is another fact that needs to be considered here. It has been shown that during pregnancy, lead in the mother's bones can be released into the bloodstream and potentially have a negative impact on the developing fetus. Therefore,one way to rationalize the trends is that blood lead levels in adult women could be the statistic of concern rather than blood lead levels of young children.

Mercury emissions and PCB emissions for the entire US show trends that are difficult to characterize, yet similar to region-specific trends. If anything, these data should be considered to be underestimates of the actual emissions for a few reasons. First, consistent reporting of emissions for both neurotoxins was nonexistent. Not all states reported emissions for all years. This was inferred through observing massive differences between emissions of consecutive years. It seems unlikely that 40 million pounds of PCBs were emitted in 2003 while less that 5 million pounds were emitted in both 2002 and 2004. Second, these data represent the amount of neurotoxins introduced into the environment per year as opposed to the total amount present in the environment at a given time. It is entirely possible that exposure to previously existing neurotoxins in the environment could cause an increase in neurological disorders as opposed to exposure to neurotoxins just recently introduced into the environment.

Another set of general patterns can be obtained through data that was not available on a state-by-state basis, but rather on the scale of the US as a whole. These data include the percentage of children diagnosed with ADHD, earlier blood lead level investigations, and PVC consumption.

Although each may be limited by their respective time frames and non-specificity with regard to region, they do provide small clues as to what is happening in the big picture.The percentage of children diagnosed with ADHD in the US is increasing. This may be attributed to either exposure to neurotoxins or improved methods of diagnosis. PVC consumption almost doubles within the time period of interest, suggesting that it too could play a role in the increasing autism rates although more region-specific data would be helpful.

Overall, the data suggests that mercury, PCBs, and PVCs could account for the overall increase in autism rates although a direct correlation is still uncertain. This uncertainty stems from inconsistency and the intrinsic limitations of the data itself, as much of it is very circumstantial.

Conclusion

Following trends in the four most prominent neurotoxins present in our environment and relating them to the growth trends of autism and other neurological disorders, it was determined that there could be a possible link between nervous system damage and these toxins. However, not enough evidence is present to determine the exact cause neurological diseases.

Autism, ADHD, Cerebral Palsy, and mental retardation have been researched in terms of the symptoms a person with each disease my experience, the history of each disorder, and the possible causes of each including overexposure to the four neurotoxins. The neurotoxins were researched in four different areas: history, trends of use, production, forms, and the health effects.

The focus of the research was to determine what problems neurotoxins in the environment may cause in terms of development in humans. In order to go about deciding if there is in fact a connection between neurological development and neurotoxins, the growth trends of each toxin's emissions were mapped and compared to the trends in growth for each disease. Research included sorting information by the ten regions of the United States. Each toxin was considered separately, and it was necessary to obtain the amount of emissions in each state for the years 1990 through 2007.

After attaining these numbers, the amount of emissions each year in every state in each region was added together and the graphs showing trends in growth were formed. By comparing these, we found that there is no definite link between neurological disorder and neurotoxic chemicals in the environment, but these graphs still provide helpful information concerning the amount of each toxin in different regions of the country. For example, the regional graphs for lead show noticeable changes in the amount of the chemical in each part of the country and how its use in paints and

gasoline has been regulated by government policy in the late 1970's. These graphs also allow us to make predictions for each toxin's emissions in the future.

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Appendix

The following tables are the data that was retrieved on the neurological disorders and neurotoxins in the environment in the United States. These sets of data were used to graph the trends within each region as well as the entire U.S.

Autism % Ages 6-22

Year	Region 1	Region 2	Region 3	Region 4	Region 5	Region 6	Region 7	Region 8	Region 9	Region 10	US Totals
1992	0.025%	0.038%	0.041%	0.022%	0.021%	0.029%	0.016%	0.012%	0.021%	0.046%	0.026%
1993	0.029%	0.046%	0.056%	0.025%	0.026%	0.035%	0.025%	0.015%	0.025%	0.049%	0.031%
1994	0.032%	0.047%	0.065%	0.031%	0.035%	0.040%	0.033%	0.019%	0.031%	0.042%	0.037%
1995	0.038%	0.063%	0.081%	0.038%	0.043%	0.048%	0.041%	0.023%	0.038%	0.085%	0.047%
1996	0.047%	0.064%	0.098%	0.046%	0.054%	0.057%	0.048%	0.030%	0.048%	0.078%	0.055%
1997	0.057%	0.076%	0.118%	0.057%	0.067%	0.067%	0.057%	0.037%	0.061%	0.098%	0.067%
1998	0.069%	0.099%	0.147%	0.069%	0.089%	0.081%	0.071%	0.049%	0.079%	0.123%	0.085%
1999	0.080%	0.114%	0.178%	0.083%	0.112%	0.095%	0.086%	0.060%	0.097%	0.148%	0.103%
2000	0.091%	0.133%	0.211%	0.090%	0.138%	0.107%	0.097%	0.066%	0.119%	0.168%	0.120%
2001	0.167%	0.156%	0.250%	0.109%	0.170%	0.123%	0.115%	0.085%	0.147%	0.193%	0.147%
2002	0.198%	0.184%	0.301%	0.127%	0.205%	0.145%	0.149%	0.100%	0.177%	0.227%	0.175%
2003	0.239%	0.212%	0.356%	0.149%	0.246%	0.169%	0.170%	0.121%	0.208%	0.260%	0.206%

US Totals

Year	Lead	Mercury	PCB	PVC	Autism	ADHD	Autism % Ages 6-22
1990	8.60%	214354	417116	8.03			
1991	8.60%	125441	113092				
1992	4.40%	55998	427320		15292		0.0257%
1993	4.40%	26916	164470		18695		0.0311%
1994	4.40%	20952	94962		22329		0.0368%
1995		23912	34432	9.83	28720		0.0469%
1996		25042	97945		33999		0.0549%
1997	7.61%	38507	987640		42126	5.5%	0.0674%
1998	6.50%	289518	6127249		53644	5.9%	0.0850%
1999	5.03%	126792	14598073		65607	5.6%	0.1030%
2000	3.96%	71884	1722883	12.85	78928	6.6%	0.1197%
2001	3.03%	95690	4646321		97994	6.4%	0.1469%
2002	2.56%	100041	3155954		118071	7.2%	0.1752%
2003	2.00%	68231	43228331		140254	6.4%	0.2061%
2004	1.76%	91949	3333946				
2005	1.58%	98451	1833407	14.19			
2006	1.21%	103624	2245823				
2007				15.05			

Region 10							
Year	**Lead**	**Mercury**	**PCB**	**PVC**	**Autism**	**ADHD**	**Autism % Ages 6-22**
1990		15250	1113	8.03			
1991		15750	0				
1992			0		1098		0.0460%
1993			0		1191		0.0488%
1994			0		1050		0.0423%
1995			0	9.83	2158		0.0854%
1996			37449		2007		0.0782%
1997	1.35%		0		2551	0.055	0.0979%
1998	1.26%		151435		3243	0.059	0.1227%
1999	1.23%		4250781		3968	0.056	0.1484%
2000	1.35%	1534	118349	12.85	4622	0.066	0.1678%
2001	2.59%	2263	678997		5398	0.064	0.1934%
2002	1.08%	1802	82512		6422	0.072	0.2270%
2003	1.35%	3273	26150		7445	0.064	0.2602%
2004	0.39%	2061	240221				
2005	0.63%	4524	86179	14.19			
2006	0.55%	5136	182845				
2007				15.05			

Region 9							
Year	**Lead**	**Mercury**	**PCB**	**PVC**	**Autism**	**ADHD**	**Autism % Ages 6-22**
1990			58178	8.03			
1991			1				
1992			0		1872		0.0206%
1993			0		2267		0.0246%
1994			0		2840		0.0305%
1995			0	9.83	3558		0.0378%
1996			0		4564		0.0479%
1997	18.33%		0		5938	0.055	0.0613%
1998	51.16%	224400	1830940		7745	0.059	0.0787%
1999	8.86%	79407	93618		9744	0.056	0.0974%
2000	5.33%	13152	95074	12.85	12346	0.066	0.1191%
2001	4.21%	31809	105795		15503	0.064	0.1470%
2002	3.38%	8483	244965		18994	0.072	0.1772%
2003	1.06%	2516	16362814		22674	0.064	0.2083%
2004	1.63%	14526	129362				
2005	2.52%	42644	98135	14.19			
2006	0.42%	32012	98281				
2007				15.05			

Region 8							
Year	**Lead**	**Mercury**	**PCB**	**PVC**	**Autism**	**ADHD**	**Autism % Ages 6-22**
1990			0	8.03			
1991			250				
1992			0		234		0.0121%
1993			0		299		0.0151%
1994			0		391		0.0193%
1995			0	9.83	466		0.0226%
1996			0		624		0.0298%
1997	3.10%		0		789	0.055	0.0372%
1998	2.39%		204354		1044	0.059	0.0486%
1999	1.11%		64121		1298	0.056	0.0596%
2000	1.50%	2955	45606	12.85	1510	0.066	0.0660%
2001	0.55%	1802	192108		1968	0.064	0.0847%
2002	0.62%	7153	633800		2358	0.072	0.1002%
2003	0.89%	1755	4241969		2866	0.064	0.1207%
2004		1204	983562				
2005		2539	421764	14.19			
2006		3908	376358				
2007				15.05			

Region 7							
Year	**Lead**	**Mercury**	**PCB**	**PVC**	**Autism**	**ADHD**	**Autism % Ages 6-22**
1990		110	0	8.03			
1991			0				
1992			0		481		0.0162%
1993			0		745		0.0250%
1994			0		999		0.0332%
1995			0	9.83	1252		0.0414%
1996			18410		1451		0.0476%
1997	8.05%		5588		1744	0.055	0.0569%
1998	5.63%	5	4550		2183	0.059	0.0709%
1999	5.67%	5	2692		2664	0.056	0.0861%
2000	4.38%	2312	9767	12.85	3056	0.066	0.0966%
2001	3.27%	2386	47012		3662	0.064	0.1153%
2002	2.65%	3540	67022		4761	0.072	0.1492%
2003	2.28%	2539	152063		5438	0.064	0.1696%
2004	1.79%	3449	546				
2005	1.75%	2279	1280	14.19			
2006	1.42%	2378	473				
2007				15.05			

Region 6							
Year	**Lead**	**Mercury**	**PCB**	**PVC**	**Autism**	**ADHD**	**Autism % Ages 6-22**
1990		52430	0	8.03			
1991		43661	0				
1992		16851	0		2046		0.0288%
1993		1597	0		2493		0.0345%
1994		5608	0		2963		0.0404%
1995		9703	0	9.83	3556		0.0478%
1996		5994	0		4321		0.0573%
1997	2.94%	4926	0		5153	0.055	0.0674%
1998	1.95%	2070	181202		6280	0.059	0.0811%
1999	2.91%	1521	147525		7466	0.056	0.0953%
2000	2.12%	8567	27599	12.85	8729	0.066	0.1070%
2001	1.52%	9776	71712		10200	0.064	0.1234%
2002	1.37%	10012	138181		12121	0.072	0.1447%
2003	1.14%	6925	107743		14352	0.064	0.1691%
2004	0.98%	6866	200178				
2005	0.84%	7939	94414	14.19			
2006	0.56%	14302	86498				
2007				15.05			

Region 5							
Year	**Lead**	**Mercury**	**PCB**	**PVC**	**Autism**	**ADHD**	**Autism % Ages 6-22**
1990		75881	262658	8.03			
1991		7976	1182				
1992		2530	410120		2470		0.0214%
1993		1499	145259		3014		0.0259%
1994		1021	31441		4057		0.0347%
1995		963	26194	9.83	5077		0.0431%
1996		1676	255		6376		0.0538%
1997	11.58%	16644	0		7997	0.055	0.0671%
1998	9.32%	11867	72235		10628	0.059	0.0888%
1999	6.80%	4323	36714		13510	0.056	0.1123%
2000	5.55%	8372	118141	12.85	16865	0.066	0.1376%
2001	4.88%	8153	1250023		20980	0.064	0.1702%
2002	4.03%	7046	65805		25390	0.072	0.2051%
2003	3.20%	12908	133131		30582	0.064	0.2460%
2004	2.38%	22849	16110				
2005	2.10%	16244	118594	14.19			
2006	1.61%	12112	300584				
2007				15.05			

Region 4							
Year	**Lead**	**Mercury**	**PCB**	**PVC**	**Autism**	**ADHD**	**Autism % Ages 6-22**
1990		60613	255	8.03			
1991		45707	2600				
1992		29049	17200		2437		0.0216%
1993		17904	265		2886		0.0252%
1994		9587	0		3639		0.0313%
1995		9025	0	9.83	4456		0.0377%
1996		9093	0		5520		0.0461%
1997	20.06%	9833	0		6928	0.055	0.0570%
1998	11.72%	46306	624735		8510	0.059	0.0691%
1999	4.20%	37293	3942697		10385	0.056	0.0833%
2000	1.75%	20984	545225	12.85	11727	0.066	0.0898%
2001	1.83%	34105	249987		14389	0.064	0.1087%
2002	1.23%	49876	721037		17023	0.072	0.1269%
2003	1.01%	22013	1456049		20206	0.064	0.1487%
2004	0.95%	25009	242918				
2005	0.65%	10745	329905	14.19			
2006	0.45%	20983	453737				
2007				15.05			

Region 3							
Year	**Lead**	**Mercury**	**PCB**	**PVC**	**Autism**	**ADHD**	**Autism % Ages 6-22**
1990		8505	0	8.03			
1991		9606	108808				
1992		5449	0		1447		0.0412%
1993		2513	0		1971		0.0556%
1994		739	60622		2327		0.0652%
1995		601	0	9.83	2915		0.0811%
1996		5468	0		3545		0.0980%
1997	41.07%	2264	0		4307	0.055	0.1182%
1998	19.95%	2023	0		5381	0.059	0.1468%
1999	14.61%	2442	0		6597	0.056	0.1785%
2000	3.67%	6183	687	12.85	8037	0.066	0.2111%
2001	3.13%	3483	567		9606	0.064	0.2496%
2002	2.90%	3228	87		11717	0.072	0.3013%
2003	2.60%	4224	140		13976	0.064	0.3555%
2004	2.20%	2915	60				
2005	1.58%	4262	82	14.19			
2006	1.57%	6961	82				
2007				15.05			

Region 2							
Year	**Lead**	**Mercury**	**PCB**	**PVC**	**Autism**	**ADHD**	**Autism % Ages 6-22**
1990			35371	8.03			
1991			0				
1992			0		2413		0.0381%
1993			18946		2907		0.0457%
1994			2899		3020		0.0474%
1995			8238	9.83	4036		0.0632%
1996			4382		4071		0.0637%
1997	10.95%	1553	982052		4848	0.055	0.0757%
1998	9.96%		871069		6363	0.059	0.0992%
1999	9.35%	67	1651405		7329	0.056	0.1139%
2000	2.78%	1738	500436	12.85	8930	0.066	0.1332%
2001	2.16%	1550	1072942		10549	0.064	0.1564%
2002	1.72%	1007	241153		12454	0.072	0.1837%
2003	1.81%	2764	117180		14419	0.064	0.2117%
2004	1.73%	3485	167770				
2005	1.41%	1524	76926	14.19			
2006	1.31%	744	89428				
2007				15.05			

Region 1							
Year	**Lead**	**Mercury**	**PCB**	**PVC**	**Autism**	**ADHD**	**Autism % Ages 6-22**
1990		1565	250				
1991		2741	0				
1992		2119	0		794		0.0246%
1993		3403	0		922		0.0285%
1994		3997	0		1043		0.0322%
1995		3620	0		1246		0.0384%
1996		2811	0		1520		0.0467%
1997	4.17%	3287	0		1871		0.0572%
1998	4.17%	2847	0		2267		0.0691%
1999	3.71%	1734	0		2646		0.0802%
2000	3.51%	6087	2970		3106		0.0911%
2001	3.05%	363	278		5739		0.1671%
2002	2.67%	7894	115		6831		0.1976%
2003	2.27%	9314	159		8296		0.2389%
2004	2.15%	9585	74				
2005	1.88%	5751	50				
2006	1.66%	5088	53				
2007							