America's Children and the Environment, Third Edition

DRAFT Indicators

Health: Neurodevelopmental Disorders

EPA is preparing the third edition of *America's Children and the Environment* (ACE3), following the previous editions published in December 2000 and February 2003. ACE is EPA's compilation of children's environmental health indicators and related information, drawing on the best national data sources available for characterizing important aspects of the relationship between environmental contaminants and children's health. ACE includes four sections: Environments and Contaminants, Biomonitoring, Health, and Special Features.

EPA has prepared draft indicator documents for ACE3 representing 23 children's environmental health topics and presenting a total of 42 proposed children's environmental health indicators. This document presents the draft text, indicators, and documentation for the neurodevelopmental disorders topic in the Health section.

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Neurodevelopmental Disorders in Children

Neurodevelopmental disorders are disabilities associated primarily with the functioning of the neurological system and brain. Examples of neurodevelopmental disorders in children include intellectual disability (also known as mental retardation), attention-deficit/hyperactivity disorder (ADHD), autism, and learning disabilities. Children with neurodevelopmental disorders experience difficulties with language and speech, motor skills, behavior, memory, learning, or other neurological functions. While the symptoms and behaviors of neurodevelopmental disabilities often changes as a person ages, some individuals with neurodevelopmental disabilities in childhood do have permanent disabilities. Diagnosis and treatment of these disorders can be difficult; treatment often involves a combination of professional therapy, pharmaceuticals, and home- and school-based programs.

Based on parental response to survey questions, approximately 12% of children in the United States ages 3 to 17 years are affected by neurodevelopmental disorders such as ADHD, learning disorders, or intellectual disability.[1] Many of these children have more than one of these conditions. Some researchers have stated that the prevalence of certain neurodevelopmental disorders, specifically autism and ADHD, has been increasing over the last four decades.[2-7] Long-term trends in these conditions are difficult to detect with certainty due to a lack of data to track prevalence over many years as well as changes in awareness and diagnostic criteria. However, some detailed reviews of historical data have concluded that the actual prevalence seems to be rising for at least some neurodevelopmental disorders, such as autism.[5,8,9] Surveys of educators and pediatricians have reported a rise in the number of children seen in classrooms and exam rooms with behavioral and learning disorders.[10-12]

Genetics can play an important role in many neurodevelopmental disorders, and some cases of certain conditions such as intellectual disability are associated with specific genes. However, most neurodevelopmental disorders have complex and multiple contributors rather than any one clear cause. These disorders likely result from a combination of genetic, biological, psychosocial and environmental risk factors, as well as behavioral risk factors such as alcohol, tobacco, or illicit drug use.

Studies have found that several widespread environmental contaminants can damage a child's developing brain and nervous system. For example, childhood exposure to lead may contribute to learning problems, such as reduced cognitive development, that define or are common in developmental disorders.[13-19] Studies have also found associations between childhood exposure to lead and ADHD,[20-23] hyperactivity and distractibility;[24-31] increased likelihood of dropping out of high school, reading disability, lower vocabulary, and lower class standing in high school;[32] and increased risk for antisocial, delinquent, or criminal behavior.[27,33-39] Lead exposure has also been found to cause deficits in memory and planning[16,31,40,41] and cause impulsiveness in children.[42]

Methylmercury also can have negative impacts on children's neurological development. Prenatal exposure to particularly high levels of mercury from poisoning incidents in Japan and Iraq has

been found to cause intellectual disability, as well as impaired motor and sensory function.[43,44] Studies of mercury's more subtle effects have focused on island populations where frequent fish consumption leads to mercury exposure in pregnant women. Results from such studies in New Zealand and the Faroe Islands suggest that increased prenatal mercury exposure due to maternal fish consumption was associated with impacts on intelligence[45,46] and decreased functioning in the areas of language, attention, and memory.[47-50] These associations were not seen in a similar study in the Seychelles Islands.[51] However, further studies in the Seychelles did find associations between prenatal mercury exposure and neurodevelopmental problems, after researchers had accounted for the developmental benefits of fish consumption.[52,53] Other more recent studies have found associations between neurodevelopmental effects and mercury body burdens in U.S. women that are within the range of typical U.S. exposures.[54,55]

Several studies of children who were prenatally exposed to elevated levels of polychlorinated biphenyls (PCBs) have suggested linkages between these contaminants and neurodevelopmental effects, including lowered intelligence and behavioral deficits such as inattention and impulsive behavior.[56-61] Problems with learning and memory have also been linked to PCB exposure.[56,62] Most of these studies found that the effects are associated with exposure in the womb resulting from the mother having eaten food contaminated with PCBs,[63-68] although some other studies have detected relationships between adverse effects and PCB exposure during infancy and childhood.[62,68-70] Although there is some inconsistency in the epidemiological literature, the overall evidence supports a concern for effects of PCBs on children's neurological development.[69,71-74] In addition, adverse effects on intelligence and behavior have been found in children of women who were highly exposed to mixtures of PCBs, chlorinated dibenzofurans, and other pollutants prior to conception.[75-77]

Additional human studies suggest that exposures to metals such as cadmium, arsenic, and manganese may have adverse effects on neurological development.[24,78-88] Other types of pollutants have also been associated with neurodevelopmental effects in animal studies. Numerous studies link both prenatal and postnatal exposure to organophosphate and organochlorine pesticides to neurodevelopmental effects.[89-99] Studies of certain polybrominated diphenyl ethers (PBDEs) found adverse effects on behavior, learning, and memory in animals.[100-102] Two recent epidemiological studies in New York City and the Netherlands found significant associations between children's prenatal exposure to PBDEs and reduced performance on IQ tests and other tests of neurological development in 5- and 6-year-old children.[103,104] Perchlorate, a naturally occurring and man-made chemical that is used to manufacture fireworks, explosives, and rocket propellant, is known to disrupt thyroid hormone levels in pregnant women, which can be a risk factor for neurodevelopmental impairment.[105-107]

A child's brain and nervous system are vulnerable to adverse impacts from pollutants because they go through a long developmental process beginning shortly after conception and continuing through adolescence.[108,109] This complex developmental process requires the precise coordination of cell growth and movement, and may be disrupted by even short-term exposures to environmental contaminants if they occur at critical stages of development. This disruption can lead to neurodevelopmental deficits that may have an effect on the child's achievements and behavior even when they do not result in a diagnosable disorder.

Attention Deficit/Hyperactivity Disorder (ADHD)

Attention-deficit/hyperactivity disorder (ADHD) is a disruptive behavior disorder characterized by ongoing inattention and/or hyperactivity-impulsivity, occurring in several settings and more frequently and severely than is typical for other individuals in the same stage of development.[110] ADHD can make family and peer relationships difficult, diminish academic performance, and reduce vocational achievement.

As the medical profession has developed a greater understanding of ADHD through the years, the name of this condition has changed. The American Psychiatric Association adopted the name "attention deficit disorder" in the early 1980s and revised it to "attention-deficit/hyperactivity disorder" in 1987.[111] A diagnosis of ADHD is based on observation of multiple symptoms of inattention or hyperactivity/impulsivity. Some children may display primarily hyperactive behavior traits, while others primarily display inattentive traits. It is possible for an individual's primary symptoms of ADHD to change over time, and older children are more likely to display inattentive behaviors.[112] Children with ADHD frequently have other disorders, such as learning disabilities and conduct disorders.[112,113]

The body of research on ADHD is expanding rapidly. While uncertainties remain, many scientists believe that this disorder is caused by a combination of factors, rather than by any one factor. Research indicates that individual genetic features influence the incidence of ADHD, but often in combination with environmental factors.[112,114-117] The role of environmental contaminants in contributing to ADHD, either alone or in conjunction with certain genetic susceptibilities, is becoming better understood, as a growing number of studies look explicitly at the relationship between ADHD and exposures to environmental contaminants. Recent epidemiological studies (most published since 2006) have linked increased levels of lead in hair and blood, mercury in blood, phthalate metabolites in urine, and the pesticide chlorpyrifos in cord blood (indicative of prenatal exposure) with increased likelihood of ADHD.[20-23,94,118-122]

Additionally, many of the behaviors that are observed in children with ADHD have been associated with elevated exposures to certain environmental contaminants. Several studies have found a relationship between attention problems, hyperactivity, and impulsivity, which are common behaviors of ADHD, and exposures to lead,[24-26,28-32,36,42,123-131] PCBs,[56-60,62,64-67] mercury,[47,48,50] and certain pesticides.[57,93,120] Animal studies that examine the link between environmental exposures and animal models of ADHD, or common ADHD behaviors, provide supporting evidence that exposures to lead, PCBs, mercury, and pesticides may contribute to ADHD.[108,129,132-142]

Additional studies on links between exposure to environmental contaminants and brain alterations augment the findings of associations between environmental contaminants and ADHD. For example, researchers have found that children diagnosed with ADHD and/or displaying characteristic behaviors of ADHD have altered levels and activity of the chemical messenger dopamine.[117,143-147] These same types of alterations have been found in children and animals exposed to lead, mercury, PCBs, and pesticides, thus highlighting a potential causal pathway of the disorder.[148-159]

Learning Disability

Learning disability (or learning disorder) is a general term for a neurological disorder that affects the way that a child's brain can receive, process, retain, and respond to information. A child with a learning disability may have trouble learning and using certain skills, including reading, writing, listening, speaking, reasoning, and doing math, although learning disabilities vary from child to child. Children with learning disabilities are not unintelligent or unmotivated; instead, they usually have average or above-average intelligence, but there are differences in the way their brains process information.[160]

As with many other neurodevelopmental disorders, the causes of learning disabilities are not completely understood. Often learning disabilities run in the family, suggesting that heredity may play a role in their development. Problems during pregnancy and birth, such as drug or alcohol use during pregnancy, low birth weight, lack of oxygen, or premature or prolonged labor, may also lead to learning disabilities.[161] The potential role of environmental contaminants in the development of learning disabilities is supported by a developing body of research on a number of metals and other contaminants.

Elevated levels of lead in teeth and hair, cadmium in hair, magnesium in hair, and dioxins and furans in blood have all been shown to be associated with diagnosed learning disabilities in children.[162-166] Exposures to lead have been associated with impaired memory, rule learning, difficulty following directions, planning, verbal abilities, and speech processing in children.[16,18,31,32,40,41,128,167,168] Exposures to mercury have been linked to dysfunctions in children's language abilities and memory.[48,50] Studies have found that children exposed prenatally to PCBs had poorer concentration and memory deficits compared with unexposed children.[56,62]

Autism Spectrum Disorders

Autism spectrum disorders (ASDs) are a group of developmental disabilities that cause significant social, communication, and behavioral challenges. The term "spectrum disorders" refers to the fact that although people with ASDs share some common symptoms, ASDs affect different people in different ways, with some experiencing very mild symptoms and others experiencing severe symptoms. ASDs encompasses autistic disorder, and the generally less severe forms, Asperger's syndrome and pervasive developmental disorder-not otherwise specified (PDD-NOS). Children with ASDs very commonly have social difficulties. They may lack interest in other people, have trouble showing or talking about feelings, and avoid or resist physical contact. A range of communication problems are seen in children with ASDs: some speak very well, while about 40% of children with an ASD do not speak at all. A third hallmark characteristic of ASDs is the demonstration of restrictive or repetitive interests or behaviors, such as lining up toys, flapping hands, rocking his or her body, or spinning in circles.[169]

To date, no single risk factor sufficient to cause ASD has been identified; rather each case is likely to be caused by the combination of multiple risk factors. Several ASD research findings

and hypotheses may imply an important role for environmental contaminants. First, there has been a sharp upward trend in prevalence that cannot be fully explained by younger ages at diagnosis, migration patterns, changes in diagnostic criteria, and inclusion of milder cases.[8] Also, the neurological signaling systems that are impaired in children with ASDs can be affected by certain environmental chemicals. For example, several pesticides are known to interfere with acetylcholine (Ach) and γ-aminobutyric acid (GABA) neurotransmission, which are chemical messenger systems altered in certain subsets of autistic individuals.[170] Furthermore, many of the identified genetic risk factors for autism are *de novo* mutations, meaning that the genetic defect is not present in either of the parents' genes, yet can be found in the genes of the child when a new genetic mutation forms in a parent's germ cells (egg or sperm), potentially from exposure to contaminants.[171,172] These *de novo* mutations in autistic children have been found in the genes that are involved in the structure or function of nervous system synapses.[173-175] Many environmental contaminants have been identified as agents capable of causing mutations in DNA, by leading to oxidative DNA damage and by inhibiting the body's normal ability to repair DNA damage.[173] Children with autism have been shown to display markers of increased oxidative stress, which may strengthen this line of reasoning.[176-178] Many studies have linked increasing paternal and maternal age with increased risk of ASDs.[179-182] The role of parental age in increased autism risk may be explained by evidence that shows advanced parental age to contribute significantly to the frequency of *de novo* mutations in a parent's germ cells.[173,183,184] Advanced parental age signifies a longer period of time when environmental exposures may act on germ cells and cause DNA damage and *de novo* mutations.

Studies, limited in number and often limited in research design, have examined the possible role that mercury may play in the development of ASDs. Earlier studies showed that higher levels of mercury have been found in the blood, baby teeth, and urine of children with ASD compared with control children;[185-187] however, another more recent study found no difference in the blood mercury levels of children with autism and typically developing children.[188] Proximity to industrial and power plant sources of environmental mercury has been linked to increased autism prevalence in a study conducted in Texas.[189] A study conducted in the San Francisco Bay Area found an association between the amount of airborne pollutants at a child's place of birth (mercury, cadmium, nickel, trichloroethylene, and vinyl chloride) and the risk for autism, but a similar study in North Carolina and West Virginia did not find such a relationship.[190,191] Thimerosal is a mercury-containing preservative that is used in some vaccines to prevent contamination and growth of harmful bacteria in vaccine vials. Since 2001, thimerosal has not been used in routinely administered childhood vaccines, with the exception of some influenza vaccines.[192] The Institute of Medicine has rejected the hypothesis of a causal relationship between thimerosal-containing vaccines and autism.[193] Finally, a study of indoor environments found an increased risk of ASDs in children born to families with PVC flooring, which contains phthalates.[194]

Intellectual Disability (Mental Retardation)

The most commonly used definitions of intellectual disability (also referred to as mental retardation) emphasize subaverage intellectual functioning before the age of 18, usually defined as an intelligence quotient (IQ) less than 70 and impairments in life skills such as

communication, self-care, home living, and social or interpersonal skills. Different severity categories, ranging from mild to severe retardation, are defined on the basis of IQ scores.[195,196]

"Intellectual disability" is used as the preferred term for this condition in the disabilities sector, but the term "mental retardation" continues to be used in the contexts of law and public policy when designating eligibility for state and federal programs.[195]

Researchers have identified some causes of intellectual disability, including genetic disorders, traumatic injuries, and prenatal events such as maternal infection or exposure to alcohol.[196,197] However, the causes of intellectual disability are unknown in 30–50% of all cases.[197] The causes are more frequently identified for cases of severe retardation (IQ less than 50), whereas the cause of mild retardation (IQ between 50 and 70) is unknown in more than 75% of cases.[198,199] Exposures to environmental contaminants could be a contributing factor to the cases of mild retardation where the cause is unknown. Exposure to high levels of lead and exposure to particularly high levels of mercury have been shown to be associated with intellectual disability. Furthermore, lead, mercury, and PCBs all have been found to have adverse effects on intelligence and cognitive functioning in children.[14-18,45,46,60] Exposure to these environmental contaminants therefore has the potential to increase the proportion of the population with IQ less than 70, thus increasing the incidence of intellectual disability in an exposed population.[200]

Indicators in this Section

This section presents indicators of the number of children ages 5 to 17 years reported to have ever been diagnosed with ADHD (Indicator ND1), learning disabilities (Indicator ND2), autism (Indicator ND3), and intellectual disability (Indicator ND4). These four conditions are examples of neurodevelopmental disorders that may be influenced by exposures to environmental contaminants. Intellectual disability and learning disabilities are disorders in which a child's cognitive or intellectual development is affected, and ADHD is a disorder in which a child's behavioral development is affected. Autism spectrum disorders are disorders in which a child's behavior, communication and social skills are affected.

Indicator ND1: Percentage of children ages 5 to 17 years reported to have attention-deficit/hyperactivity disorder, by sex, 1997–2008

Indicator ND2: Percentage of children ages 5 to 17 years reported to have a learning disability, by sex, 1997–2008

Indicator ND3: Percentage of children ages 5 to 17 years reported to have autism, 1997–2008

Indicator ND4: Percentage of children ages 5 to 17 years reported to have intellectual disability (mental retardation), 1997–2008

Overview

Indicators ND1, ND2, ND3, and ND4 present information about the number of children who are reported to have ever been diagnosed with four different neurodevelopmental disorders: attention-deficit/hyperactivity disorder (ADHD), learning disabilities, autism, and intellectual disability. The data come from a national survey that collects health information from a representative sample of the population. The four indicators show how the rates of children's neurodevelopmental disorders have changed over time, and, when possible, how the rates differ between boys and girls.

National Health Interview Survey

The indicators use data obtained from the National Health Interview Survey (NHIS). NHIS is a large-scale household interview survey of a representative sample of the civilian noninstitutionalized U.S. population, conducted by the Centers for Disease Control and Prevention. From 1997–2005, interviews were conducted for approximately 12,000–14,000 children annually. Since 2006, interviews have been conducted for approximately 9,000–10,000 children per year. The parents of children in the survey were asked "Has a doctor or health professional ever told you that <child's name> had Attention Deficit/Hyperactivity Disorder (ADHD) or Attention Deficit Disorder (ADD)? Autism? Mental Retardation?" Another question on the NHIS survey asked "Has a representative from a school or a health professional ever told you that <child's name> had a learning disability?"

Data Presented in the Indicators

The following indicators display the prevalence of ADHD, learning disabilities, autism, and intellectual disability among U.S. children, for the years 1997–2008. Diagnosing neurodevelopmental disorders in young children can be difficult: many affected children may not

receive a diagnosis until they enter preschool or kindergarten. For this reason, the indicators here show children ages 5 to 17 years.

Although the NHIS provides the best national-level data available, NHIS data likely underestimate the prevalence of neurodevelopmental disorders. Reasons for this underestimate may include late identification of affected children and the exclusion of institutionalized children from the NHIS survey population. A diagnosis of a neurodevelopmental disorder depends not only on the presence of particular symptoms and behaviors in a child, but on concerns being raised by a parent or teacher about the child's behavior and on the child's access to a doctor to make the diagnosis. Further, the NHIS relies on parents reporting that their child has been diagnosed with the neurodevelopmental disorder, and accuracy of parental responses could be affected by cultural and other factors. Long-term trends in these conditions are difficult to detect with certainty due to a lack of data to track prevalence over many years, as well as changes in awareness and diagnostic criteria, which could explain at least part of the observed increasing trends.[201-203]

There may be potential complications with the diagnosis itself. For example, a diagnosis of ADHD relies on recognition of various types of behaviors in different combinations, and therefore requires a certain amount of judgment on the part of a doctor, similar to diagnosis of other mental disorders. Many other problems, including anxiety disorders, depression, and learning disabilities, can be expressed with signs and symptoms that resemble those of ADHD. As many as half of those with ADHD also have other mental disorders, which can make it harder to diagnose and treat ADHD.[204] Despite these facts, ADHD has good clinical validity, meaning that impaired children share similarities, exhibit symptoms, respond to treatment, and are recognized with general consistency across clinicians.[112]

Because autism is the only autism spectrum disorder (ASD) referred to in the survey, it is not clear how parents of children with other ASDs, i.e., Asperger's syndrome and pervasive developmental disorder-not otherwise specified (PDD-NOS), may have responded. The estimates shown by Indicator ND3 could represent underestimates of ASD prevalence if parents of children with Asperger's syndrome and PDD-NOS did not answer yes to the NHIS questions about autism.

In addition to the data shown in the indicator graphs, supplemental tables provide information regarding the prevalence of neurodevelopmental disorders for different age groups and prevalence by race/ethnicity, sex, and family income. These comparisons use the most current four years of data available. The data from four years are combined to increase the statistical reliability of the estimates for each race/ethnicity, sex, and family income group.

Other Estimates of ADHD and Autism Prevalence

In addition to NHIS, other CDC studies provide data on prevalence of ADHD and ASDs among children. The National Survey of Children's Health (NSCH), conducted in 2003 by the Centers for Disease Control and Prevention, found that 7.8% of children ages 4 to 17 years had ever been diagnosed with ADHD. The same survey, when conducted again in 2007, found that 9.5% of children ages 4 to 17 years had ever been diagnosed with ADHD.[2] Both estimates are somewhat

higher than the ADHD prevalence estimates from the NHIS for those years. The 2007 NSCH also estimates that 7.2% of children ages 4 to 17 years currently have ADHD. The 2007 NSCH also provides information at the state level: North Carolina had the highest rate, with 15.6% of children ages 4 to 17 years having ever been diagnosed with ADHD; the rate was lowest in Nevada, at 5.6%.[2]

In 2002 and 2006, CDC performed thorough data gathering in selected areas to examine the prevalence of ASDs in eight-year-old children. The ASD prevalence estimate for 2002 was 0.66%, or 1 in 152 eight-year-old children, and the estimate for 2006 was 0.9%, or 1 in 110 eight-year-old children.[205,206] The 2007 NSCH also provides an estimate of 1.1% of children ages 3 to 17 years reported to have ASDs, or about 1 in 90.[207]

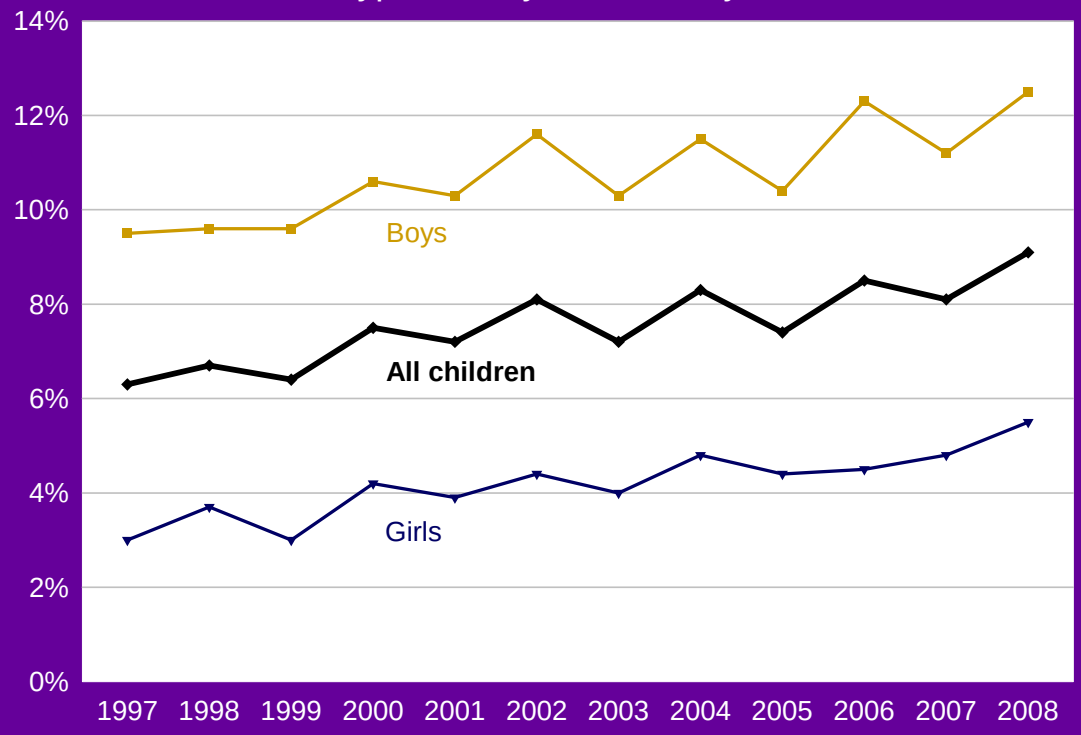
Statistical Testing

Statistical analysis has been applied to the indicators to determine whether any changes in prevalence over time, or any differences in prevalence between demographic groups, are statistically significant. These analyses use a 5% significance level ($p \leq 0.05$), meaning that a conclusion of statistical significance is made only when there is no more than a 5% chance that the observed change over time or difference between demographic groups occurred randomly. It should be noted that when statistical testing is conducted for differences among multiple demographic groups (e.g., considering both race/ethnicity and income level), the large number of comparisons involved increases the probability that some differences identified as statistically significant may actually have occurred randomly.

A finding of statistical significance for a health indicator depends not only on the numerical difference in the value of a reported statistic between two groups, but also on the number of observations in the survey and various aspects of the survey design. For example, if the prevalence of a health effect is different between two groups, the statistical test is more likely to detect a difference when data have been obtained from a larger number of people in those groups. A finding that there is or is not a statistically significant difference in prevalence between two groups or in prevalence over time is not the only information that should be considered when determining the public health implications of those differences.

Indicator ND1

Percentage of children ages 5 to 17 years reported to have attention-deficit/hyperactivity disorder, by sex, 1997-2008



SOURCE: U.S. EPA. America's Children and the Environment. www.epa.gov/envirohealth/children

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey

- In 2008, 9.1% of children ages 5 to 17 years were reported to have ever been diagnosed with attention-deficit/hyperactivity disorder (ADHD), compared with 6.3% in 1997.
    - Statistical note: The increase was statistically significant for children overall, for both boys and girls considered separately, as well as for children below poverty level and those at or above poverty level. There was also a statistically significant increasing trend for children ages 11 to 17 years, but not for children ages 5 to 10 years. (See Table ND1a and Table ND1b.)

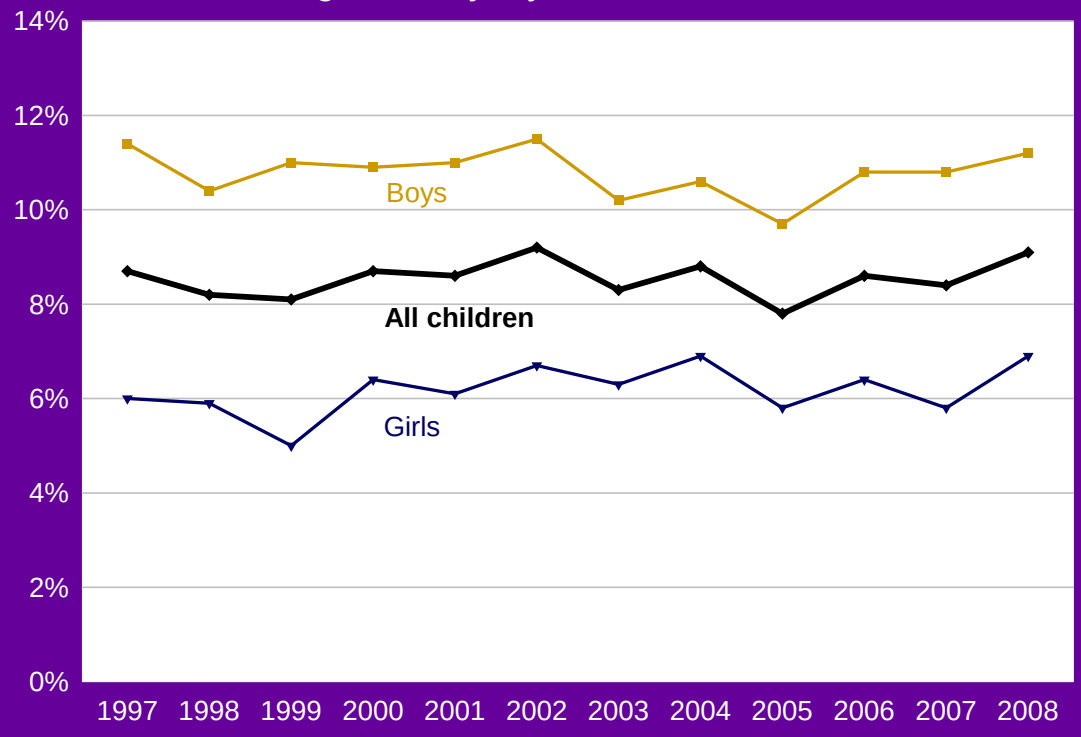
- For the years 2005–2008, the percentage of boys reported to have ADHD (11.6%) was higher than the rate for girls (4.8%). This difference was statistically significant. (See Table ND1a.)

- In 2005–2008, 9.6% of White non-Hispanic children, 8.5% of Black non-Hispanic children, 5.0% of Hispanic children, and 2.0% of Asian non-Hispanic children were reported to have ADHD. (See Table ND1b.)
    - Statistical note: These differences were statistically significant, with the exception of the difference between White and Black non-Hispanic children.

- In 2005–2008, 9.6% of children from families living below the poverty level were reported to have ADHD compared with 8.0% of children from families living at or above the poverty level. (See Table ND1b.)
    - Statistical note: The difference between income groups was statistically significant.

Indicator ND2

Percentage of children ages 5 to 17 years reported to have a learning disability, by sex, 1997-2008



SOURCE: U.S. EPA. America's Children and the Environment. www.epa.gov/envirohealth/children

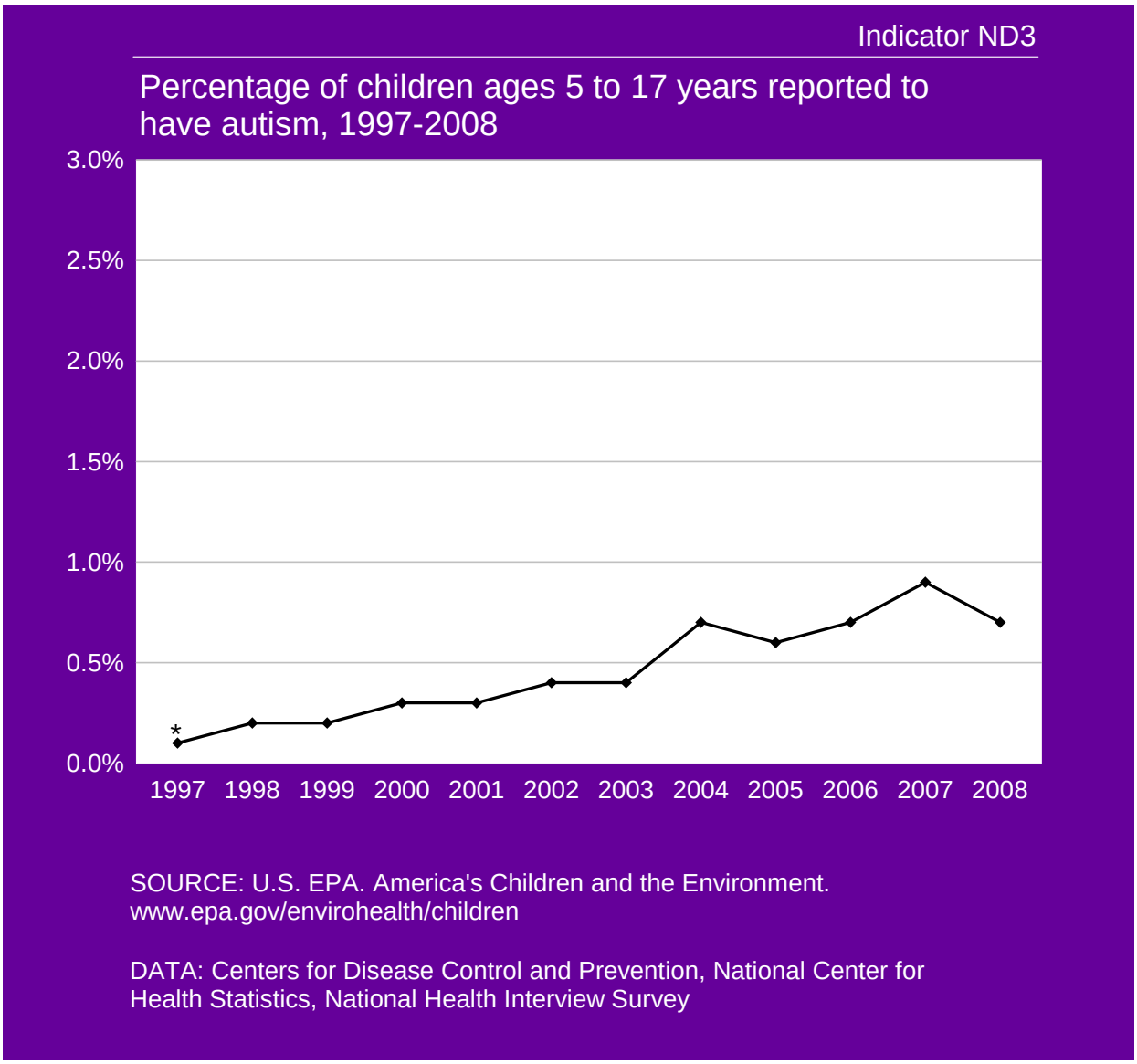
DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey

- The percentage of children reported to have ever been diagnosed with a learning disability stayed relatively constant between 1997 and 2008.
    - Statistical note: There was not a statistically significant change in this indicator among children overall, but there was a statistically significant decrease in the percentage of boys reported to have a learning disability, after accounting for the influence of other demographic differences (i.e., differences in race/ethnicity, age, and family income).

- For the years 2005–2008, the percentage of boys reported to have a learning disability (10.6%) was higher than the rate for girls (6.2%). This difference was statistically significant.

- For the years 2005–2008, the percentage of children reported to have a learning disability was higher for children living below the poverty level (11.8%) compared with those living at or above the poverty level (7.8%). (See Table ND2a.)
    - Statistical note: The difference between income groups was statistically significant.

- Rates of learning disability vary by race and ethnicity. The highest rates of learning disability are reported for Black non-Hispanic children (9.2%) and for White non-Hispanic children (9.1%). Asian children have the lowest rate of learning disability, at 2.1%. (See Table ND2b.)
    - Statistical note: The differences between the rate of learning disability reported for Asian children and the rates for Black non-Hispanic and White non-Hispanic children were statistically significant.



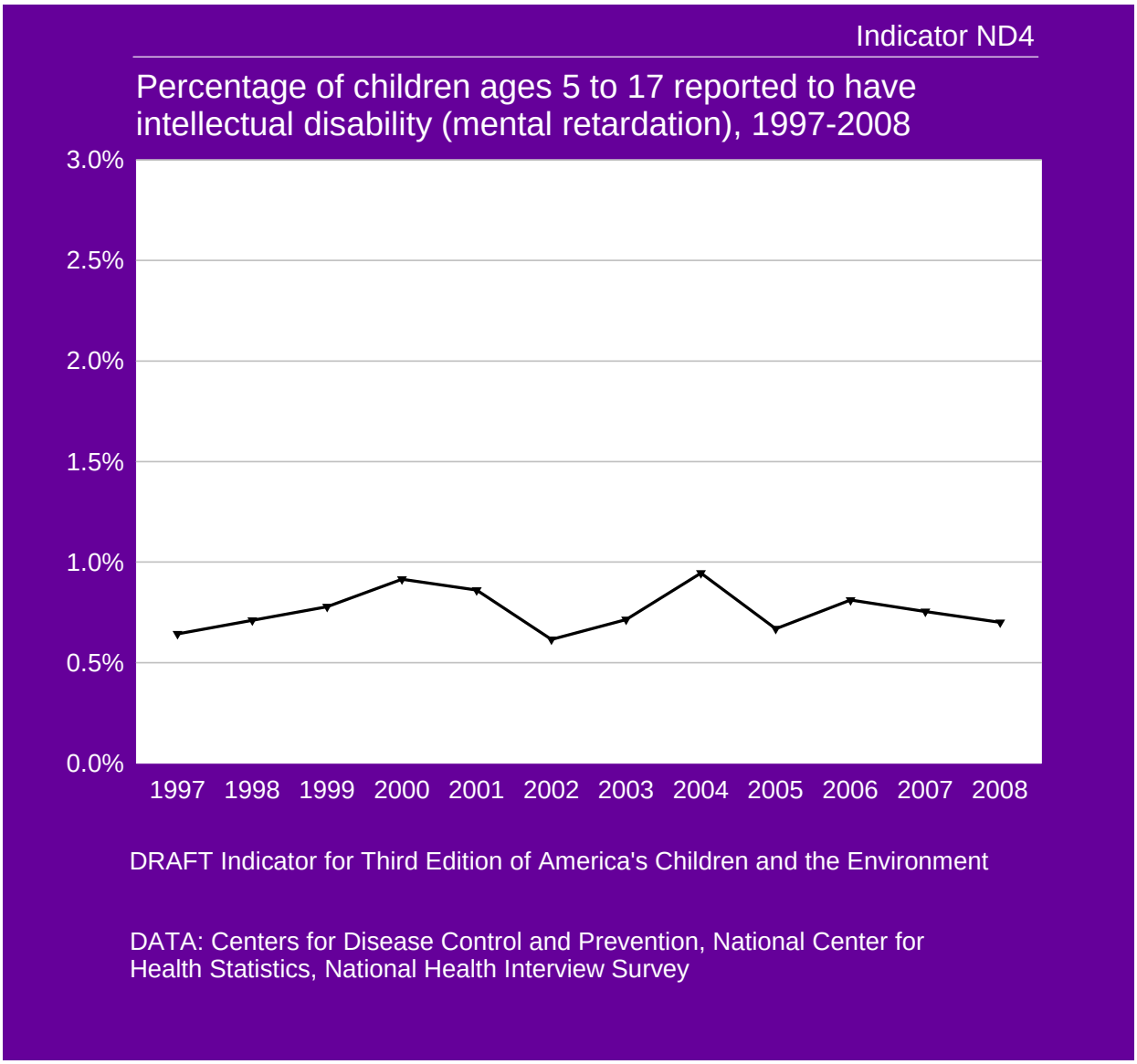
* The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate).

- The percentage of children reported to have ever been diagnosed with autism rose from 0.1% in 1997 to 0.7% in 2008. More than nine times as many children were reported to have been diagnosed with autism in 2008 as in 1997. This increase was statistically significant.

- For the years 2005–2008, the rate of reported autism was more than three times higher in boys than in girls, 1.1% and 0.3%, respectively. (See Table ND3a.) This difference was statistically significant.

- For the years 2005–2008, the rates of autism reported were similar for children living below the poverty level and those living at or above the poverty level. (See Table ND3b.)

- Rates of reported cases of autism vary by race and ethnicity. The highest rates of autism are for White non-Hispanic children (0.9%). Autism rates were lower for Black non-Hispanic children (0.5%) and Hispanic children (0.4%). (See table ND3b.)
    - Statistical note: The differences between the rate of autism for White non-Hispanic children and the rates for Black non-Hispanic and Hispanic children were statistically significant.



- In 2008, about 0.7% of children ages 5 to 17 years were reported to have ever been diagnosed with intellectual disability (mental retardation). There has been no statistically significant change in the prevalence of intellectual disability between 1997 and 2008.

- In 2005–2008, about 0.8% of boys were reported to have intellectual disability, compared with about 0.7% of girls, but this difference was not statistically significant. (See Table ND4a.)

- In 2005–2008, reported rates of intellectual disability were 1.1% for Black non-Hispanic children, 0.9% for Hispanic children, and 0.6% for White non-Hispanic children. (See Table ND4b.)
    - Statistical note: Only the difference between Black non-Hispanic and White non-Hispanic children was statistically significant. This difference was not statistically

significant after accounting for the influence of other demographic differences (i.e., differences in age, sex, and family income).

- In 2005–2008, 1.3% of children from families with incomes below the poverty level were reported to have intellectual disability, compared with 0.6% of children from families at or above the poverty level. (See Table ND4b.)
    - Statistical note: The difference between income groups was statistically significant, even after accounting for other demographic differences (i.e., differences in race/ethnicity or age profile).

Data Tables

Table ND1: Percentage of children ages 5 to 17 years reported to have attention-deficit/hyperactivity disorder, by sex, 1997-2008

	1997	1998	1999	2000
All children	6.3%	6.7%	6.4%	7.5%
Boys	9.5%	9.6%	9.6%	10.6%
Girls	3.0%	3.7%	3.0%	4.2%
	2001	**2002**	**2003**	**2004**
All children	7.2%	8.1%	7.2%	8.3%
Boys	10.3%	11.6%	10.3%	11.5%
Girls	3.9%	4.4%	4.0%	4.8%
	2005	**2006**	**2007**	**2008**
All children	7.4%	8.5%	8.1%	9.1%
Boys	10.4%	12.3%	11.2%	12.5%
Girls	4.4%	4.5%	4.8%	5.5%

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey.

NOTE: Data represent parents' responses to the survey question: “Has a doctor or health professional ever told you that <child’s name> had Attention Deficit/Hyperactivity Disorder (ADHD) or Attention Deficit Disorder (ADD)?”

Table ND1a: Percentage of children reported to have attention-deficit/hyperactivity disorder, by age and sex, 2005-2008

	Ages 5-17	Ages 5-10	Ages 11-17
All children	8.3%	6.3%	9.8%
Boys	11.6%	8.5%	14.1%
Girls	4.8%	4.1%	5.4%

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey.

NOTE: Data represent parents' responses to the survey question: “Has a doctor or health professional ever told you that <child’s name> had Attention Deficit/Hyperactivity Disorder (ADHD) or Attention Deficit Disorder (ADD)?”

Table ND1b: Percentage of children ages 5 to 17 years reported to have attention-deficit/hyperactivity disorder, by race/ethnicity and family income, 2005-2008

	All Incomes	< Poverty Level	≥ Poverty Level	≥ Poverty Level Detail	
				100-200% of Poverty Level	≥ 200% of Poverty Level
All races/ethnicities	8.3%	9.6%	8.0%	9.2%	7.5%
White non-Hispanic	9.6%	13.2%	9.2%	12.1%	8.5%
Black or African-American non-Hispanic	8.5%	10.4%	7.5%	9.1%	6.3%
Asian non-Hispanic	2.0%	NA**	2.1%	NA**	2.4%
Hispanic	5.0%	5.5%	4.7%	4.8%	4.7%
Mexican	4.4%	4.4%	4.4%	4.1%	4.8%
Puerto Rican	9.4%	12.0%	7.9%	11.5%	5.5%
Other †	10.4%	15.3%	9.1%	13.6%	7.1%
American Indian or Alaska Native non-Hispanic	8.4%*	NA**	NA**	NA**	NA**

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey.

NOTE: Data represent parents' responses to the survey question: “Has a doctor or health professional ever told you that <child’s name> had Attention Deficit/Hyperactivity Disorder (ADHD) or Attention Deficit Disorder (ADD)?”

† “Other” includes non-Hispanic respondents whose race is neither White, Black, or Asian, or who report multiple races.

* The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, exceeds 30% (RSE = standard error divided by the estimate).

** The estimate is not reported because it has large uncertainty: the relative standard error, RSE, exceeds 40% (RSE = standard error divided by the estimate).

Table ND2: Percentage of children ages 5 to 17 years reported to have a learning disability, by sex, 1997-2008

	1997	1998	1999	2000
All children	8.7%	8.2%	8.1%	8.7%
Boys	11.4%	10.4%	11.0%	10.9%
Girls	6.0%	5.9%	5.0%	6.4%
	2001	**2002**	**2003**	**2004**
All children	8.6%	9.2%	8.3%	8.8%
Boys	11.0%	11.5%	10.2%	10.6%
Girls	6.1%	6.7%	6.3%	6.9%
	2005	**2006**	**2007**	**2008**
All children	7.8%	8.6%	8.4%	9.1%
Boys	9.7%	10.8%	10.8%	11.2%
Girls	5.8%	6.4%	5.8%	6.9%

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey.

NOTE: Data represent parents' responses to the survey question: "Has a representative from a school or a health professional ever told you that <child's name> had a learning disability?"

Table ND2a: Percentage of children reported to have a learning disability, by age and sex, 2005-2008

	Ages 5-17	Ages 5-10	Ages 11-17
All children	8.5%	6.9%	9.7%
Boys	10.6%	8.6%	12.3%
Girls	6.2%	5.2%	7.1%

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey.

NOTE: Data represent parents' responses to the survey question: "Has a representative from a school or a health professional ever told you that <child's name> had a learning disability?"

Table ND2b: Percentage of children ages 5 to 17 years reported to have a learning disability, by race/ethnicity and family income, 2005-2008

	All	< Poverty	≥ Poverty	≥ Poverty Level Detail

				100-200% of Poverty Level	≥ 200% of Poverty Level
All races/ethnicities	8.5%	11.8%	7.8%	9.3%	7.2%
White non-Hispanic	9.1%	15.8%	8.4%	10.5%	7.9%
Black or African-American non-Hispanic	9.2%	11.8%	7.9%	10.6%	6.0%
Asian non-Hispanic	2.1%	NA**	1.9%	NA**	2.0%
Hispanic	7.2%	8.5%	6.6%	7.1%	6.2%
Mexican	6.9%	7.3%	6.8%	6.9%	6.6%
Puerto Rican	11.2%	14.7%	9.1%	11.5%	7.5%
Other †	9.2%	14.8%	7.7%	10.7%	6.4%
American Indian or Alaska Native non-Hispanic	12.1%*	NA**	9.7%*	NA**	NA**

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey.

NOTE: Data represent parents' responses to the survey question: "Has a representative from a school or a health professional ever told you that <child's name> had a learning disability?"

† "Other" includes non-Hispanic respondents whose race is neither White, Black, or Asian, or who report multiple races.

* The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, exceeds 30% (RSE = standard error divided by the estimate).

** The estimate is not reported because it has large uncertainty: the relative standard error, RSE, exceeds 40% (RSE = standard error divided by the estimate).

Table ND3: Percentage of children ages 5 to 17 years reported to have autism, 1997-2008

	1997	1998	1999	2000
All children	0.1%*	0.2%	0.2%	0.3%
	2001	**2002**	**2003**	**2004**
All children	0.3%	0.4%	0.4%	0.7%
	2005	**2006**	**2007**	**2008**
All children	0.6%	0.7%	0.9%	0.7%

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey.

NOTE: Data represent parents' responses to the survey question: "Has a doctor or health professional ever told you that <child's name> had Autism?"

* The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, exceeds 30% (RSE = standard error divided by the estimate).

Table ND3a: Percentage of children reported to have autism, by age and sex, 2005-2008

	Ages 5-17	Ages 5-10	Ages 11-17
All children	0.7%	0.9%	0.5%
Boys	1.1%	1.4%	0.9%
Girls	0.3%	0.5%	0.2%*

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey.

NOTE: Data represent parents' responses to the survey question: "Has a doctor or health professional ever told you that <child's name> had Autism?"

* The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, exceeds 30% (RSE = standard error divided by the estimate).

Table ND3b: Percentage of children ages 5 to 17 years reported to have autism, by race/ethnicity and family income, 2005-2008

	All Incomes	< Poverty Level	≥ Poverty Level	≥ Poverty Level Detail	
				100-200% of Poverty Level	≥ 200% of Poverty Level
All races/ethnicities	0.7%	0.7%	0.7%	0.6%	0.8%
White non-Hispanic	0.9%	1.2%	0.8%	0.7%	0.9%
Black or African-American non-Hispanic	0.5%	NA**	0.5%	NA**	NA**
Asian non-Hispanic	NA**	NA**	NA**	NA**	NA**
Hispanic	0.4%	NA**	0.4%	NA**	0.5%*
Mexican	0.4%*	NA**	0.4%*	NA**	NA**
Puerto Rican	NA**	NA**	NA**	NA**	NA**
Other †	NA**	NA**	NA**	NA**	NA**
American Indian or Alaska Native non-Hispanic	NA**	NA**	NA**	NA**	NA**

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey.

NOTE: Data represent parents' responses to the survey question: "Has a doctor or health professional ever told you that <child's name> had Autism?"

† "Other" includes non-Hispanic respondents whose race is neither White, Black, or Asian, or who report multiple races.

* The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, exceeds 30% (RSE = standard error divided by the estimate).

** The estimate is not reported because it has large uncertainty: the relative standard error, RSE, exceeds 40% (RSE = standard error divided by the estimate).

Table ND4: Percentage of children ages 5 to 17 years reported to have intellectual disability (mental retardation), by sex, 1997-2008

	1997	1998	1999	2000
All children	0.6%	0.7%	0.8%	0.9%
Boys	0.6%	0.8%	0.8%	1.2%
Girls	0.7%	0.6%	0.7%	0.6%
	2001	**2002**	**2003**	**2004**
All children	0.9%	0.6%	0.7%	0.9%
Boys	1.0%	0.6%	0.8%	1.0%
Girls	0.7%	0.6%	0.6%	0.9%
	2005	**2006**	**2007**	**2008**
All children	0.7%	0.8%	0.8%	0.7%
Boys	0.7%	0.9%	0.9%	0.7%
Girls	0.6%	0.8%	0.6%*	0.6%

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey.

NOTE: Data represent parents' responses to the survey question: "Has a doctor or health professional ever told you that <child's name> had Mental Retardation?"

* The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, exceeds 30% (RSE = standard error divided by the estimate).

Table ND4a: Percentage of children reported to have intellectual disability (mental retardation), by age and sex, 2005-2008

	Ages 5-17	5-10	11-17
All children	0.7%	0.8%	0.7%
Boys	0.8%	0.9%	0.7%
Girls	0.7%	0.6%	0.7%

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey.

NOTE: Data represent parents' responses to the survey question: "Has a doctor or health professional ever told you that <child's name> had Mental Retardation?"

Table ND4b: Percentage of children ages 5 to 17 years reported to have intellectual disability (mental retardation), by race/ethnicity and family income, 2005-2008

	All Incomes	< Poverty Level	≥ Poverty Level	≥ Poverty Level Detail	
				100-200% of Poverty Level	≥ 200% of Poverty Level
All races/ethnicities	0.7%	1.3%	0.6%	0.9%	0.5%
White non-Hispanic	0.6%	1.2%*	0.5%	0.8%	0.5%
Black or African-American non-Hispanic	1.1%	1.4%*	0.9%	1.5%	0.5%*
Asian non-Hispanic	0.6%*	NA**	NA**	NA**	NA**
Hispanic	0.9%	1.3%*	0.7%	0.7%	0.7%
Mexican	0.7%	NA**	0.5%	0.7%*	NA**
Puerto Rican	1.2%	1.3%*	NA**	NA**	NA**
Other †	NA**	NA**	NA**	NA**	NA**
American Indian or Alaska Native non-Hispanic	NA**	NA**	NA**	NA**	NA**

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey.

NOTE: Data represent parents' responses to the survey question: “Has a doctor or health professional ever told you that <child’s name> had Mental Retardation?”

† “Other” includes non-Hispanic respondents whose race is neither White, Black, or Asian, or who report multiple races.

* The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, exceeds 30% (RSE = standard error divided by the estimate).

** The estimate is not reported because it has large uncertainty: the relative standard error, RSE, exceeds 40% (RSE = standard error divided by the estimate).

References

1. P. Pastor, and C. Reuben. National Center for Health Statistics, email to Daniel Axelrad, U.S. EPA, January 27, 2009. Calculations from the National Health Interview Survey, 2001-2006, considering responses for the following conditions: Mental retardation, other developmental delay, Down syndrome, autism, learning disability, and attention-deficit/hyperactivity disorder., January 27, 2009.

2. Visser, S.N., R.H. Bitsko, M.L. Danielson, R. Perou, and S.J. Blumberg. 2010. Increasing prevalence of parent-reported attention-deficit/hyperactivity disorder among children --- United States, 2003 and 2007. *Morbidity and Mortality Weekly Report* 59 (44):1439-43.

3. Froehlich, T.E., B.P. Lanphear, J.N. Epstein, W.J. Barbaresi, S.K. Katusic, and R.S. Kahn. 2007. Prevalence, recognition, and treatment of attention-deficit/hyperactivity disorder in a national sample of US children. *Archives of Pediatrics & Adolescent Medicine* 161 (9):857-64.

4. Grandjean, P., and P.J. Landrigan. 2006. Developmental neurotoxicity of industrial chemicals. *Lancet* 368 (9553):2167-78.

5. Newschaffer, C.J., M.D. Falb, and J.G. Gurney. 2005. National autism prevalence trends from United States special education data. *Pediatrics* 115 (3):e277-82.

6. Prior, M. 2003. Is there an increase in the prevalence of autism spectrum disorders? *Journal of Paediatrics and Child Health* 39 (2):81-2.

7. Rutter, M. 2005. Incidence of autism spectrum disorders: changes over time and their meaning. *Acta Paediatrica* 94 (1):2-15.

8. Hertz-Picciotto, I., and L. Delwiche. 2009. The rise in autism and the role of age at diagnosis. *Epidemiology* 20 (1):84-90.

9. Newschaffer, C.J. 2006. Investigating diagnostic substitution and autism prevalence trends. *Pediatrics* 117 (4):1436-7.

10. Grupp-Phelan, J., J.S. Harman, and K.J. Kelleher. 2007. Trends in mental health and chronic condition visits by children presenting for care at U.S. emergency departments. *Public Health Reports* 122 (1):55-61.

11. Kelleher, K.J., T.K. McInerny, W.P. Gardner, G.E. Childs, and R.C. Wasserman. 2000. Increasing identification of psychosocial problems: 1979-1996. *Pediatrics* 105 (6):1313-21.

12. U.S. Department of Education. 2007. *27th Annual (2005) Report to Congress on the Implementation of the Individuals with Disabilities Education Act, Vol. 1*. Washington, DC.

13. Bellinger, D., J. Sloman, A. Leviton, M. Rabinowitz, H.L. Needleman, and C. Waternaux. 1991. Low-level lead exposure and children's cognitive function in the preschool years. *Pediatrics* 87 (2):219-27.

14. Bellinger, D.C., A. Leviton, and C. Waternaux. 1987. Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. *New England Journal of Medicine* 316 (17):1037-43.

15. Jusko, T.A., C.R. Henderson, B.P. Lanphear, D.A. Cory-Slechta, P.J. Parsons, and R.L. Canfield. 2008. Blood lead concentrations < 10 microg/dL and child intelligence at 6 years of age. *Environmental Health Perspectives* 116 (2):243-8.

16. Lanphear, B.P., K. Dietrich, P. Auinger, and C. Cox. 2000. Cognitive deficits associated with blood lead concentrations <10 micrograms/dL in U.S. children and adolescents. *Public Health Reports* 115 (6):521-9.

17. Lanphear, B.P., R. Hornung, J. Khoury, K. Yolton, P. Baghurst, D.C. Bellinger, R.L. Canfield, K.N. Dietrich, R. Bornschein, T. Greene, S.J. Rothenberg, H.L. Needleman, L. Schnaas, G. Wasserman, J. Graziano, and R. Roberts. 2005. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environmental Health Perspectives* 113 (7):894-9.

18. McMichael, A.J., P.A. Baghurst, N.R. Wigg, G.V. Vimpani, E.F. Robertson, and R.J. Roberts. 1988. Port Pirie cohort study: environmental exposure to lead and children's abilities at the age of four years. *New England Journal of Medicine* 319 (8):468-75.

19. Schnaas, L., S.J. Rothenberg, M.F. Flores, S. Martinez, C. Hernandez, E. Osorio, S.R. Velasco, and E. Perroni. 2006. Reduced intellectual development in children with prenatal lead exposure. *Environmental Health Perspectives* 114 (5):791-7.

20. Braun, J.M., R.S. Kahn, T. Froehlich, P. Auinger, and B.P. Lanphear. 2006. Exposures to environmental toxicants and attention deficit hyperactivity disorder in U.S. children. *Environmental Health Perspectives* 114 (12):1904-9.

21. Nigg, J.T., G.M. Knottnerus, M.M. Martel, M. Nikolas, K. Cavanagh, W. Karmaus, and M.D. Rappley. 2008. Low blood lead levels associated with clinically diagnosed attention-deficit/hyperactivity disorder and mediated by weak cognitive control. *Biological Psychiatry* 63 (3):325-31.

22. Tuthill, R.W. 1996. Hair lead levels related to children's classroom attention-deficit behavior. *Archives of Environmental Health* 51 (3):214-20.

23. Wang, H.-L., X.-T. Chen, B. Yang, F.-L. Ma, S. Wang, M.-L. Tang, N.-G. Hao, and D.-Y. Ruan. 2008. Case-control study of blood lead levels and attention-deficit hyperactivity disorder in Chinese children *Environmental Health Perspectives* 116 (10):1401-1406.

24. Calderon, J., M.E. Navarro, M.E. Jimenez-Capdeville, M.A. Santos-Diaz, A. Golden, I. Rodriguez-Leyva, V. Borja-Aburto, and F. Diaz-Barriga. 2001. Exposure to arsenic and lead and neuropsychological development in Mexican children. *Environmental Research* 85 (2):69-76.

25. Canfield, R.L., D.A. Kreher, C. Cornwell, and C.R. Henderson, Jr. 2003. Low-level lead exposure, executive functioning, and learning in early childhood. *Child Neuropsychology* 9 (1):35-53.

26. Chiodo, L.M., C. Covington, R.J. Sokol, J.H. Hannigan, J. Jannise, J. Ager, M. Greenwald, and V. Delaney-Black. 2007. Blood lead levels and specific attention effects in young children. *Neurotoxicology and Teratology* 29 (5):538-46.

27. Chiodo, L.M., S.W. Jacobson, and J.L. Jacobson. 2004. Neurodevelopmental effects of postnatal lead exposure at very low levels. *Neurotoxicology and Teratology* 26 (3):359-71.

28. Mendelsohn, A.L., B.P. Dreyer, A.H. Fierman, C.M. Rosen, L.A. Legano, H.A. Kruger, S.W. Lim, and C.D. Courtlandt. 1998. Low-level lead exposure and behavior in early childhood. *Pediatrics* 101 (3):E10.

29. Minder, B., E.A. Das-Smaal, E.F. Brand, and J.F. Orlebeke. 1994. Exposure to lead and specific attentional problems in schoolchildren. *Journal of Learning Disabilities* 27 (6):393-9.

30. Needleman, H.L. 1993. The current status of childhood low-level lead toxicity. *Neurotoxicology* 14 (2-3):161-6.

31. Surkan, P.J., A. Zhang, F. Trachtenberg, D.B. Daniel, S. McKinlay, and D.C. Bellinger. 2007. Neuropsychological function in children with blood lead levels <10 microg/dL. *Neurotoxicology* 28 (6):1170-7.

32. Needleman, H.L., A. Schell, D.C. Bellinger, A. Leviton, and E.N. Allred. 1990. The long term effects of exposure to low doses of lead in childhood, an 11-year follow-up report. *New England Journal of Medicine* 322 (2):83-8.

33. Braun, J.M., T.E. Froehlich, J.L. Daniels, K.N. Dietrich, R. Hornung, P. Auinger, and B.P. Lanphear. 2008. Association of environmental toxicants and conduct disorder in U.S. children: NHANES 2001-2004. *Environmental Health Perspectives* 116 (7):956-62.

34. Dietrich, K.N., M.D. Ris, P.A. Succop, O.G. Berger, and R.L. Bornschein. 2001. Early exposure to lead and juvenile delinquency. *Neurotoxicology and Teratology* 23 (6):511-8.

35. Needleman, H.L., C. McFarland, R.B. Ness, S.E. Fienberg, and M.J. Tobin. 2002. Bone lead levels in adjudicated delinquents. A case control study. *Neurotoxicology and Teratology* 24 (6):711-7.

36. Needleman, H.L., J.A. Riess, M.J. Tobin, G.E. Biesecker, and J.B. Greenhouse. 1996. Bone lead levels and delinquent behavior. *Journal of the American Medical Association* 275 (5):363-9.

37. Nevin, R. 2007. Understanding international crime trends: the legacy of preschool lead exposure. *Environmental Research* 104 (3):315-36.

38. Ris, M.D., K.N. Dietrich, P.A. Succop, O.G. Berger, and R.L. Bornschein. 2004. Early exposure to lead and neuropsychological outcome in adolescence. *Journal of the International Neuropsychological Society* 10 (2):261-70.

39. Wright, J.P., K.N. Dietrich, M.D. Ris, R.W. Hornung, S.D. Wessel, B.P. Lanphear, M. Ho, and M.N. Rae. 2008. Association of prenatal and childhood blood lead concentrations with criminal arrests in early adulthood. *Public Library of Science Medicine* 5 (5):e101.

40. Canfield, R.L., M.H. Gendle, and D.A. Cory-Slechta. 2004. Impaired neuropsychological functioning in lead-exposed children. *Developmental Neuropsychology* 26 (1):513-40.

41. Froehlich, T.E., B.P. Lanphear, K.N. Dietrich, D.A. Cory-Slechta, N. Wang, and R.S. Kahn. 2007. Interactive effects of a DRD4 polymorphism, lead, and sex on executive functions in children. *Biological Psychiatry* 62 (3):243-9.

42. Stewart, P.W., D.M. Sargent, J. Reihman, B.B. Gump, E. Lonky, T. Darvill, H. Hicks, and J. Pagano. 2006. Response inhibition during Differential Reinforcement of Low Rates (DRL) schedules may be sensitive to low-level polychlorinated biphenyl, methylmercury, and lead exposure in children. *Environmental Health Perspectives* 114 (12):1923-9.

43. Bakir, F., H. Rustam, S. Tikriti, S.F. Al-Damluji, and H. Shihristani. 1980. Clinical and epidemiological aspects of methylmercury poisoning. *Postgraduate Medical Journal* 56 (651):1-10.

44. Harada, M., H. Akagi, T. Tsuda, T. Kizaki, and H. Ohno. 1999. Methylmercury level in umbilical cords from patients with congenital Minamata disease. *Science of the Total Environment* 234 (1-3):59-62.

45. Crump, K.S., T. Kjellstrom, A.M. Shipp, A. Silvers, and A. Stewart. 1998. Influence of prenatal mercury exposure upon scholastic and psychological test performance: benchmark analysis of a New Zealand cohort. *Risk Analysis* 18 (6):701-13.

46. Kjellstrom, T., P. Kennedy, P. Wallis, and C. Mantell. 1989. *Physical and Mental Development of Children with Prenatal Exposure to Mercury From Fish. Stage 2: Interviews and Psychological Tests at Age 6*. Solna, Sweden: National Swedish Environmental Protection Board. 3642.

47. Budtz-Jorgensen, E., P. Grandjean, and P. Weihe. 2007. Separation of risks and benefits of seafood intake. *Environmental Health Perspectives* 115 (3):323-7.

48. Debes, F., E. Budtz-Jorgensen, P. Weihe, R.F. White, and P. Grandjean. 2006. Impact of prenatal methylmercury exposure on neurobehavioral function at age 14 years. *Neurotoxicology and Teratology* 28 (5):536-47.

49. Grandjean, P., E. Budtz-Jorgensen, R.F. White, P.J. Jorgensen, P. Weihe, F. Debes, and N. Keiding. 1999. Methylmercury exposure biomarkers as indicators of neurotoxicity in children aged 7 years. *American Journal of Epidemiology* 150 (3):301-5.

50. Grandjean, P., P. Weihe, R.F. White, F. Debes, S. Araki, K. Yokoyama, K. Murata, N. Sorensen, R. Dahl, and P.J. Jorgensen. 1997. Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. *Neurotoxicology and Teratology* 19 (6):417-28.

51. Myers, G.J., P.W. Davidson, C.F. Shamlaye, C.D. Axtell, E. Cernichiari, O. Choisy, A. Choi, C. Cox, and T.W. Clarkson. 1997. Effects of prenatal methylmercury exposure from a high fish diet on developmental milestones in the Seychelles Child Development Study. *Neurotoxicology* 18 (3):819-29.

52. Davidson, P.W., J.J. Strain, G.J. Myers, S.W. Thurston, M.P. Bonham, C.F. Shamlaye, A. Stokes-Riner, J.M. Wallace, P.J. Robson, E.M. Duffy, L.A. Georger, J. Sloane-Reeves, E. Cernichiari, R.L. Canfield, C. Cox, L.S. Huang, J. Janciuras, and T.W. Clarkson. 2008. Neurodevelopmental effects of maternal nutritional status and exposure to methylmercury from eating fish during pregnancy. *Neurotoxicology* 29 (5):767-75.

53. Strain, J.J., P.W. Davidson, M.P. Bonham, E.M. Duffy, A. Stokes-Riner, S.W. Thurston, J.M. Wallace, P.J. Robson, C.F. Shamlaye, L.A. Georger, J. Sloane-Reeves, E. Cernichiari, R.L. Canfield, C. Cox, L.S. Huang, J. Janciuras, G.J. Myers, and T.W. Clarkson. 2008. Associations of maternal long-chain polyunsaturated fatty acids, methyl mercury, and infant development in the Seychelles Child Development Nutrition Study. *Neurotoxicology* 29 (5):776-782.

54. Lederman, S.A., R.L. Jones, K.L. Caldwell, V. Rauh, S.E. Sheets, D. Tang, S. Viswanathan, M. Becker, J.L. Stein, R.Y. Wang, and F.P. Perera. 2008. Relation between cord blood mercury levels and early child development in a World Trade Center cohort. *Environmental Health Perspectives* 116 (8):1085-91.

55. Oken, E., J.S. Radesky, R.O. Wright, D.C. Bellinger, C.J. Amarasiriwardena, K.P. Kleinman, H. Hu, and M.W. Gillman. 2008. Maternal fish intake during pregnancy, blood mercury levels, and child cognition at age 3 years in a US cohort. *American Journal of Epidemiology* 167 (10):1171-81.

56. Jacobson, J.L., and S.W. Jacobson. 2003. Prenatal exposure to polychlorinated biphenyls and attention at school age. *Journal of Pediatrics* 143 (6):780-8.

57. Sagiv, S.K., J.K. Nugent, T.B. Brazelton, A.L. Choi, P.E. Tolbert, L.M. Altshul, and S.A. Korrick. 2008. Prenatal organochlorine exposure and measures of behavior in infancy using the Neonatal Behavioral Assessment Scale (NBAS). *Environmental Health Perspectives* 116 (5):666-73.

58. Stewart, P., S. Fitzgerald, J. Reihman, B. Gump, E. Lonky, T. Darvill, J. Pagano, and P. Hauser. 2003. Prenatal PCB exposure, the corpus callosum, and response inhibition. *Environmental Health Perspectives* 111 (13):1670-7.

59. Stewart, P., J. Reihman, B. Gump, E. Lonky, T. Darvill, and J. Pagano. 2005. Response inhibition at 8 and 9 1/2 years of age in children prenatally exposed to PCBs. *Neurotoxicology and Teratology* 27 (6):771-80.

60. Stewart, P.W., E. Lonky, J. Reihman, J. Pagano, B.B. Gump, and T. Darvill. 2008. The relationship between prenatal PCB exposure and intelligence (IQ) in 9-year-old children. *Environmental Health Perspectives* 116 (10):1416-22.

61. Lai, T.J., X. Liu, Y.L. Guo, N.W. Guo, M.L. Yu, C.C. Hsu, and W.J. Rogan. 2002. A cohort study of behavioral problems and intelligence in children with high prenatal polychlorinated biphenyl exposure. *Archives of General Psychiatry* 59 (11):1061-6.

62. Vreugdenhil, H.J., P.G. Mulder, H.H. Emmen, and N. Weisglas-Kuperus. 2004. Effects of perinatal exposure to PCBs on neuropsychological functions in the Rotterdam cohort at 9 years of age. *Neuropsychology* 18 (1):185-93.

63. Darvill, T., E. Lonky, J. Reihman, P. Stewart, and J. Pagano. 2000. Prenatal exposure to PCBs and infant performance on the Fagan test of infant intelligence. *Neurotoxicology* 21 (6):1029-38.

64. Jacobson, J.L., and S.W. Jacobson. 1996. Intellectual impairment in children exposed to polychlorinated biphenyls in utero. *New England Journal of Medicine* 335 (11):783-9.

65. Jacobson, J.L., and S.W. Jacobson. 1997. Teratogen update: polychlorinated biphenyls. *Teratology* 55 (5):338-347.

66. Patandin, S., C.I. Lanting, P.G. Mulder, E.R. Boersma, P.J. Sauer, and N. Weisglas-Kuperus. 1999. Effects of environmental exposure to polychlorinated biphenyls and dioxins on cognitive abilities in Dutch children at 42 months of age. *Journal of Pediatrics* 134 (1):33-41.

67. Stewart, P., J. Reihman, E. Lonky, T. Darvill, and J. Pagano. 2000. Prenatal PCB exposure and neonatal behavioral assessment scale (NBAS) performance. *Neurotoxicology and Teratology* 22 (1):21-9.

68. Walkowiak, J., J.A. Wiener, A. Fastabend, B. Heinzow, U. Kramer, E. Schmidt, H.J. Steingruber, S. Wundram, and G. Winneke. 2001. Environmental exposure to polychlorinated biphenyls and quality of the home environment: effects on psychodevelopment in early childhood. *Lancet* 358 (9293):1602-7.

69. Schantz, S.L., J.J. Widholm, and D.C. Rice. 2003. Effects of PCB exposure on neuropsychological function in children. *Environmental Health Perspectives* 111 (3):357-576.

70. Jacobson, J.L., S.W. Jacobson, and H.E. Humphrey. 1990. Effects of exposure to PCBs and related compounds on growth and activity in children. *Neurotoxicology and Teratology* 12 (4):319-26.

71. Boucher, O., G. Muckle, and C.H. Bastien. 2009. Prenatal exposure to polychlorinated biphenyls: a neuropsychologic analysis. *Environmental Health Perspectives* 117 (1):7-16.

72. Ribas-Fito, N., M. Sala, M. Kogevinas, and J. Sunyer. 2001. Polychlorinated biphenyls (PCBs) and neurological development in children: a systematic review. *Journal of Epidemiology and Community Health* 55 (8):537-46.

73. Schantz, S.L., J.C. Gardiner, D.M. Gasior, R.J. McCaffrey, A.M. Sweeney, and H.E.B. Humphrey. 2004. Much Ado About Something: The Weight of Evidence for PCB Effects on Neuropsychological Function. *Psychology in the Schools* 41 (6):669-679.

74. Wigle, D.T., T.E. Arbuckle, M.C. Turner, A. Berube, Q. Yang, S. Liu, and D. Krewski. 2008. Epidemiologic evidence of relationships between reproductive and child health outcomes and environmental chemical contaminants. *Journal of Toxicology and Environmental Health Part B Critical Reviews* 11 (5-6):373-517.

75. Chen, Y.C., Y.L. Guo, C.C. Hsu, and W.J. Rogan. 1992. Cognitive development of Yu-Cheng ("oil disease") children prenatally exposed to heat-degraded PCBs. *Journal of the American Medical Association* 268 (22):3213-8.

76. Chen, Y.C., M.L. Yu, W.J. Rogan, B.C. Gladen, and C.C. Hsu. 1994. A 6-year follow-up of behavior and activity disorders in the Taiwan Yu-cheng children. *American Journal of Public Health* 84 (3):415-21.

77. Rogan, W.J., B.C. Gladen, K.L. Hung, S.L. Koong, L.Y. Shih, J.S. Taylor, Y.C. Wu, D. Yang, N.B. Ragan, and C.C. Hsu. 1988. Congenital poisoning by polychlorinated biphenyls and their contaminants in Taiwan. *Science* 241 (4863):334-6.

78. Bouchard, M., F. Laforest, L. Vandelac, D. Bellinger, and D. Mergler. 2007. Hair manganese and hyperactive behaviors: pilot study of school-age children exposed through tap water. *Environmental Health Perspectives* 115 (1):122-7.

79. Grandjean, P., and K. Murata. 2007. Developmental arsenic neurotoxicity in retrospect. *Epidemiology* 18 (1):25-6.

80. Ljung, K., and M. Vahter. 2007. Time to re-evaluate the guideline value for manganese in drinking water? *Environmental Health Perspectives* 115 (11):1533-8.

81. Marlowe, M., A. Cossairt, C. Moon, J. Errera, A. MacNeel, R. Peak, J. Ray, and C. Schroeder. 1985. Main and interaction effects of metallic toxins on classroom behavior. *Journal of Abnormal Child Psychology* 13 (2):185-98.

82. Rosado, J.L., D. Ronquillo, K. Kordas, O. Rojas, J. Alatorre, P. Lopez, G. Garcia-Vargas, M. Del Carmen Caamano, M.E. Cebrian, and R.J. Stoltzfus. 2007. Arsenic exposure and cognitive performance in Mexican schoolchildren. *Environmental Health Perspectives* 115 (9):1371-5.

83. Stewart-Pinkham, S.M. 1989. The effect of ambient cadmium air pollution on the hair mineral content of children. *Science of the Total Environment* 78:289-96.

84. Thatcher, R.W., M.L. Lester, R. McAlaster, and R. Horst. 1982. Effects of low levels of cadmium and lead on cognitive functioning in children. *Archives of Environmental Health* 37 (3):159-66.

85. Wang, S.-x., Z.-h. Wang, X.-t. Cheng, J. Li, Z.-p. Sang, X.-d. Zhang, L.-l. Han, X.-y. Qiao, Z.-m. Wu, and Z.-q. Wang. 2007. Water arsenic and fluoride exposure and children's intelligence quotient and growth in Shanyin County, Shanxi, China. *Environmental Health Perspectives* 115 (4):643-7.

86. Wasserman, G.A., X. Liu, F. Parvez, H. Ahsan, P. Factor-Litvak, J. Kline, A. van Geen, V. Slavkovich, N.J. Loiacono, D. Levy, Z. Cheng, and J.H. Graziano. 2007. Water arsenic exposure and intellectual function in 6-year-old children in Araihazar, Bangladesh. *Environmental Health Perspectives* 115 (2):285-9.

87. Wright, R.O., C. Amarasiriwardena, A.D. Woolf, R. Jim, and D.C. Bellinger. 2006. Neuropsychological correlates of hair arsenic, manganese, and cadmium levels in school-age children residing near a hazardous waste site. *Neurotoxicology* 27 (2):210-6.

88. Claus Henn, B., A.S. Ettinger, J. Schwartz, M.M. Téllez-Rojo, H. Lamadrid-Figueroa, M. Hernández-Avila, L. Schnaas, C. Amarasiriwardena, D.C. Bellinger, H. Hu, and R.O. Wrighta. 2010. Early postnatal blood manganese levels and children's neurodevelopment. *Epidemiology*.

89. Eskenazi, B., A. Bradman, and R. Castorina. 1999. Exposures of children to organophosphate pesticides and their potential adverse health effects. *Environmental Health Perspectives* 107 (Suppl. 3):409-19.

90. Lovasi, G.S., J.W. Quinn, V.A. Rauh, F.P. Perera, H.F. Andrews, R. Garfinkel, L. Hoepner, R. Whyatt, and A. Rundle. 2010. Chlorpyrifos exposure and urban residential environment characteristics as determinants of early childhood neurodevelopment. *American Journal of Public Health* Epub 3/18/2010.

91. Morales, E., J. Sunyer, F. Castro-Giner, X. Estivill, J. Julvez, N. Ribas-Fitó, M. Torrent, J.O. Grimalt, and R.d. Cid. 2008. Influence of glutathione s-transferase polymorphisms on cognitive functioning effects induced by p,p´-DDT among preschoolers *Environmental Health Perspectives* 116 (11):1581-1585.

92. Puertas, R., M.J. Lopez-Espinosa, F. Cruz, R. Ramos, C. Freire, M. Perez-Garcia, A. Abril, J. Julvez, M. Salvatierra, C. Campoy, and N. Olea. 2010. Prenatal exposure to mirex impairs neurodevelopment at age of 4 years. *Neurotoxicology* 31 (1):154-60.

93. Ribas-Fito, N., M. Torrent, D. Carrizo, L. Munoz-Ortiz, J. Julvez, J.O. Grimalt, and J. Sunyer. 2006. In utero exposure to background concentrations of DDT and cognitive functioning among preschoolers. *American Journal of Epidemiology* 164 (10):955-62.

94. Sagiv, S.K., S.W. Thurston, D.C. Bellinger, P.E. Tolbert, L.M. Altshul, and S.A. Korrick. 2010. Prenatal organochlorine exposure and behaviors associated with attention deficit hyperactivity disorder in school-aged children. *American Journal of Epidemiology* 171 (5):593-601.

95. Slotkin, T.A., B.E. Bodwell, I.T. Ryde, E.D. Levin, and F.J. Seidler. 2008. Exposure of neonatal rats to parathion elicits sex-selective impairment of acetylcholine systems in brain regions during adolescence and adulthood. *Environmental Health Perspectives* 116 (10):1308-14.

96. Slotkin, T.A., M.M. Cousins, C.A. Tate, and F.J. Seidler. 2001. Persistent cholinergic presynaptic deficits after neonatal chlorpyrifos exposure. *Brain Research* 902 (2):229-43.

97. Slotkin, T.A., E.D. Levin, and F.J. Seidler. 2006. Comparative developmental neurotoxicity of organophosphate insecticides: effects on brain development are separable from systemic toxicity. *Environmental Health Perspectives* 114 (5):746-51.

98. Slotkin, T.A., I.T. Ryde, E.D. Levin, and F.J. Seidler. 2008. Developmental neurotoxicity of low dose diazinon exposure of neonatal rats: effects on serotonin systems in adolescence and adulthood. *Brain Research Bulletin* 75 (5):640-7.

99. Torres-Sanchez, L., L. Schnaas, M.E. Cebrian, C. Hernandez Mdel, E.O. Valencia, R.M. Garcia Hernandez, and L. Lopez-Carrillo. 2009. Prenatal dichlorodiphenyldichloroethylene (DDE) exposure and neurodevelopment: a follow-up from 12 to 30 months of age. *Neurotoxicology* 30 (6):1162-5.

100. Eriksson, P., E. Jakobsson, and A. Fredriksson. 2001. Brominated flame retardants: a novel class of developmental neurotoxicants in our environment? *Environmental Health Perspectives* 109 (9):903-908.

101. Kuriyama, S.N., C.E. Talsness, K. Grote, and I. Chahoud. 2005. Developmental exposure to low dose PBDE 99: effects on male fertility and neurobehavior in rat offspring. *Environmental Health Perspectives* 113 (2):149-54.

102. Rice, D.C., E.A. Reeve, A. Herlihy, R.T. Zoeller, W.D. Thompson, and V.P. Markowski. 2007. Developmental delays and locomotor activity in the C57BL6/J mouse following neonatal exposure to the fully-brominated PBDE, decabromodiphenyl ether. *Neurotoxicology and Teratology* 29 (4):511-20.

103. Herbstman, J.B., A. Sjodin, M. Kurzon, S.A. Lederman, R.S. Jones, V. Rauh, L.L. Needham, D. Tang, M. Niedzwiecki, R.Y. Wang, and F. Perera. 2010. Prenatal exposure to PBDEs and neurodevelopment. *Environmental Health Perspectives* 118 (5):712-9.

104. Roze, E., L. Meijer, A. Bakker, K.N. Van Braeckel, P.J. Sauer, and A.F. Bos. 2009. Prenatal exposure to organohalogens, including brominated flame retardants, influences motor, cognitive, and behavioral performance at school age. *Environmental Health Perspectives* 117 (12):1953-8.

105. Greer, M.A., G. Goodman, R.C. Pleus, and S.E. Greer. 2002. Health effects assessment for environmental perchlorate contamination: the dose response for inhibition of thyroidal radioiodine uptake in humans. *Environmental Health Perspectives* 110 (9):927-37.

106. Haddow, J.E., G.E. Palomaki, W.C. Allan, J.R. Williams, G.J. Knight, J. Gagnon, C.E. O'Heir, M.L. Mitchell, R.J. Hermos, S.E. Waisbren, J.D. Faix, and R.Z. Klein. 1999. Maternal thyroid deficiency during pregnancy and subsequent neuropsychological development of the child. *New England Journal of Medicine* 341 (8):549-55.

107. Miller, M.D., K.M. Crofton, D.C. Rice, and R.T. Zoeller. 2009. Thyroid-disrupting chemicals: interpreting upstream biomarkers of adverse outcomes. *Environmental Health Perspectives* 117 (7):1033-41.

108. Rice, D.C. 2000. Parallels between attention deficit hyperactivity disorder and behavioral deficits produced by neurotoxic exposure in monkeys. *Environmental Health Perspectives* 108 (Suppl. 3):405-408.

109. Rodier, P.M. 1995. Developing brain as a target of toxicity. *Environmental Health Perspectives* 103 Suppl. 6:73-6.

110. American Psychiatric Association. 2000. *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition Text Revision*. Washington D.C.: American Psychiatric Association.

111. American Psychiatric Association. 1987. *Diagnostic and Statistical Manual of Mental Disorders, Third Edition Text Revision (DSM-III-R).* Washington, D.C.

112. Nigg, J.T. 2006. *What Causes ADHD? Understanding What Goes Wrong and Why*. New York: The Guilford Press.

113. Pastor, P.N., and C.A. Reuben. 2008. Diagnosed attention deficit hyperactivity disorder and learning disability: United States, 2004-2006. *Vital and Health Statistics* 10 (237).

114. Ding, Y.C., H.C. Chi, D.L. Grady, A. Morishima, J.R. Kidd, K.K. Kidd, P. Flodman, M.A. Spence, S. Schuck, J.M. Swanson, Y.P. Zhang, and R.K. Moyzis. 2002. Evidence of positive selection acting at the human dopamine receptor D4 gene locus. *Proceedings of the National Academy of Sciences* 99 (1):309-14.

115. Kieling, C., R.R. Goncalves, R. Tannock, and F.X. Castellanos. 2008. Neurobiology of attention deficit hyperactivity disorder. *Child and Adolescent Psychiatric Clinics of North America* 17 (2):285-307, viii.

116. Kirley, A., Z. Hawi, G. Daly, M. McCarron, C. Mullins, N. Millar, I. Waldman, M. Fitzgerald, and M. Gill. 2002. Dopaminergic system genes in ADHD: toward a biological hypothesis. *Neuropsychopharmacology* 27 (4):607-19.

117. Thapar, A., J. Holmes, K. Poulton, and R. Harrington. 1999. Genetic basis of attention deficit and hyperactivity. *British Journal of Psychiatry* 174:105-11.

118. Cheuk, D.K., and V. Wong. 2006. Attention-deficit hyperactivity disorder and blood mercury level: a case-control study in chinese children. *Neuropediatrics* 37 (4):234-40.

119. Rauh, V.A., R. Garfinkel, F.P. Perera, H.F. Andrews, L. Hoepner, D.B. Barr, R. Whitehead, D. Tang, and R.W. Whyatt. 2006. Impact of prenatal chlorpyrifos exposure on neurodevelopment in the first 3 years of life among inner-city children. *Pediatrics* 118 (6):e1845-59.

120. Ribas-Fito, N., M. Torrent, D. Carrizo, J. Julvez, J.O. Grimalt, and J. Sunyer. 2007. Exposure to hexachlorobenzene during pregnancy and children's social behavior at 4 years of age. *Environmental Health Perspectives* 115 (3):447-50.

121. Kim, B.N., S.C. Cho, Y. Kim, M.S. Shin, H.J. Yoo, J.W. Kim, Y.H. Yang, H.W. Kim, S.Y. Bhang, and Y.C. Hong. 2009. Phthalates exposure and attention-deficit/hyperactivity disorder in school-age children. *Biological Psychiatry* 66 (10):958-63.

122. Froehlich, T.E., B.P. Lanphear, P. Auinger, R. Hornung, J.N. Epstein, J. Braun, and R.S. Kahn. 2009. Association of tobacco and lead exposures with attention-deficit/hyperactivity disorder. *Pediatrics* 124 (6):e1054-63.

123. Bellinger, D., H. Hu, L. Titlebaum, and H.L. Needleman. 1994. Attentional correlates of dentin and bone lead levels in adolescents. *Archives of Environmental Health* 49 (2):98-105.

124. David, O., J. Clark, and K. Voeller. 1972. Lead and hyperactivity. *Lancet* 2 (7783):900-3.

125. Fergusson, D.M., L.J. Horwood, and M.T. Lynskey. 1993. Early dentine lead levels and subsequent cognitive and behavioural development. *Journal of Child Psychology and Psychiatry* 34 (2):215-27.

126. Gittelman, R., and B. Eskenazi. 1983. Lead and hyperactivity revisited. An investigation of nondisadvantaged children. *Archives of General Psychiatry* 40 (8):827-33.

127. Hunter, J., M.A. Urbanowicz, W. Yule, and R. Lansdown. 1985. Automated testing of reaction time and its association with lead in children. *International Archives of Occupational and Environmental Health* 57 (1):27-34.

128. Needleman, H.L., C. Gunnoe, A. Leviton, R. Reed, H. Peresie, C. Maher, and P. Barrett. 1979. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *New England Journal of Medicine* 300 (13):689-95.

129. Rice, D.C. 1996. Behavioral effects of lead: commonalities between experimental and epidemiologic data. *Environmental Health Perspectives* 104 (Suppl. 2):337-51.

130. Silva, P.A., P. Hughes, S. Williams, and J.M. Faed. 1988. Blood lead, intelligence, reading attainment, and behaviour in eleven year old children in Dunedin, New Zealand. *Journal of Child Psychology and Psychiatry* 29 (1):43-52.

131. Thomson, G.O., G.M. Raab, W.S. Hepburn, R. Hunter, M. Fulton, and D.P. Laxen. 1989. Blood-lead levels and children's behaviour--results from the Edinburgh Lead Study. *Journal of Child Psychology and Psychiatry* 30 (4):515-28.

132. Ahlbom, J., A. Fredriksson, and P. Eriksson. 1995. Exposure to an organophosphate (DFP) during a defined period in neonatal life induces permanent changes in brain muscarinic receptors and behaviour in adult mice. *Brain Research* 677 (1):13-9.

133. Brockel, B.J., and D.A. Cory-Slechta. 1998. Lead, attention, and impulsive behavior: changes in a fixed-ratio waiting-for-reward paradigm. *Pharmacology Biochemistry and Behavior* 60 (2):545-52.

134. Costa, L.G., M. Aschner, A. Vitalone, T. Syversen, and O.P. Soldin. 2004. Developmental neuropathology of environmental agents. *Annual Review of Pharmacology and Toxicology* 44:87-110.

135. Danielsson, B.R., A. Fredriksson, L. Dahlgren, A.T. Gardlund, L. Olsson, L. Dencker, and T. Archer. 1993. Behavioural effects of prenatal metallic mercury inhalation exposure in rats. *Neurotoxicology and Teratology* 15 (6):391-6.

136. Eriksson, P., and A. Fredriksson. 1991. Neurotoxic effects of two different pyrethroids, bioallethrin and deltamethrin, on immature and adult mice: changes in behavioral and muscarinic receptor variables. *Toxicology and Applied Pharmacology* 108 (1):78-85.

137. Gong, Z., and H.L. Evans. 1997. Effect of chelation with meso-dimercaptosuccinic acid (DMSA) before and after the appearance of lead-induced neurotoxicity in the rat. *Toxicology and Applied Pharmacology* 144 (2):205-14.

138. Holene, E., I. Nafstad, J.U. Skaare, A. Bernhoft, P. engen, and T. Sagvolden. 1995. Behavioral effects of pre- and postnatal exposure to individual polychlorinated biphenyl congeners in rats. *Environmental Toxicology and Chemistry* 14 (6):967-76.

139. Holene, E., I. Nafstad, J.U. Skaare, and T. Sagvolden. 1998. Behavioural hyperactivity in rats following postnatal exposure to sub-toxic doses of polychlorinated biphenyl congeners 153 and 126. *Behavioral Brain Research* 94 (1):213-24.

140. Icenogle, L.M., N.C. Christopher, W.P. Blackwelder, D.P. Caldwell, D. Qiao, F.J. Seidler, T.A. Slotkin, and E.D. Levin. 2004. Behavioral alterations in adolescent and adult rats caused by a brief subtoxic exposure to chlorpyrifos during neurulation. *Neurotoxicology and Teratology* 26 (1):95-101.

141. Rice, D.C. 1997. Effect of postnatal exposure to a PCB mixture in monkeys on multiple fixed interval-fixed ratio performance. *Neurotoxicology and Teratology* 19 (6):429-34.

142. Winrow, C.J., M.L. Hemming, D.M. Allen, G.B. Quistad, J.E. Casida, and C. Barlow. 2003. Loss of neuropathy target esterase in mice links organophosphate exposure to hyperactivity. *Nature Genetics* 33 (4):477-85.

143. DiMaio, S., N. Grizenko, and R. Joober. 2003. Dopamine genes and attention-deficit hyperactivity disorder: a review. *Journal of Psychiatry and Neuroscience* 28 (1):27-38.

144. Faraone, S.V., and J. Biederman. 1998. Neurobiology of attention-deficit hyperactivity disorder. *Biological Psychiatry* 44 (10):951-8.

145. Faraone, S.V., A.E. Doyle, E. Mick, and J. Biederman. 2001. Meta-analysis of the association between the 7-repeat allele of the dopamine D(4) receptor gene and attention deficit hyperactivity disorder. *American Journal of Psychiatry* 158 (7):1052-7.

146. Jucaite, A., E. Fernell, C. Halldin, H. Forssberg, and L. Farde. 2005. Reduced midbrain dopamine transporter binding in male adolescents with attention-deficit/hyperactivity disorder: association between striatal dopamine markers and motor hyperactivity. *Biological Psychiatry* 57 (3):229-38.

147. Sagvolden, T., E.B. Johansen, H. Aase, and V.A. Russell. 2005. A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behavioral and Brain Sciences* 28 (3):397-419; discussion 419-68.

148. Agrawal, A.K., H.A. Tilson, and S.C. Bondy. 1981. 3,4,3',4'-Tetrachlorobiphenyl given to mice prenatally produces long-term decreases in striatal dopamine and receptor binding sites in the caudate nucleus. *Toxicology Letters* 7 (6):417-24.

149. Bourdineaud, J.P., M. Fujimura, M. Laclau, M. Sawada, and A. Yasutake. 2010. Deleterious effects in mice of fish-associated methylmercury contained in a diet mimicking the Western populations' average fish consumption. *Environment International* Epub 11/03/2010.

150. Cory-Slechta, D.A. 1995. Relationships between lead-induced learning impairments and changes in dopaminergic, cholinergic, and glutamatergic neurotransmitter system functions. *Annual Review of Pharmacology and Toxicology* 35:391-415.

151. Cory-Slechta, D.A., D.J. O'Mara, and B.J. Brockel. 1998. Nucleus accumbens dopaminergic medication of fixed interval schedule-controlled behavior and its modulation by low-level lead exposure. *Journal of Pharmacology and Experimental Therapeutics* 286 (2):794-805.

152. Cory-Slechta, D.A., M.J. Pokora, and R.A. Preston. 1996. The effects of dopamine agonists on fixed interval schedule-controlled behavior are selectively altered by low-level lead exposure. *Neurotoxicology and Teratology* 18 (5):565-75.

153. Dam, K., S.J. Garcia, F.J. Seidler, and T.A. Slotkin. 1999. Neonatal chlorpyrifos exposure alters synaptic development and neuronal activity in cholinergic and catecholaminergic pathways. *Brain Research, Developmental Brain Research* 116 (1):9-20.

154. Kala, S.V., and A.L. Jadhav. 1995. Low level lead exposure decreases in vivo release of dopamine in the rat nucleus accumbens: a microdialysis study. *Journal of Neurochemistry* 65 (4):1631-5.

155. Khan, M.A., C.A. Lichtensteiger, O. Faroon, M. Mumtaz, D.J. Schaeffer, and L.G. Hansen. 2002. The hypothalamo-pituitary-thyroid (HPT) axis: a target of nonpersistent ortho-substituted PCB congeners. *Toxicological Sciences* 65 (1):52-61.

156. Seegal, R.F., R.J. Okoniewski, K.O. Brosch, and J.C. Bemis. 2002. Polychlorinated biphenyls alter extraneuronal but not tissue dopamine concentrations in adult rat striatum: an in vivo microdialysis study. *Environmental Health Perspectives* 110 (11):1113-7.

157. Seegal, R.F., B.A. Pappas, and G.A. Park. 1998. Neurochemical effects of consumption of Great Lakes salmon by rats. *Regulatory Toxicology and Pharmacology* 27 (1 Pt 2):S68-75.

158. Shain, W., B. Bush, and R. Seegal. 1991. Neurotoxicity of polychlorinated biphenyls: structure-activity relationship of individual congeners. *Toxicology and Applied Pharmacology* 111 (1):33-42.

159. Zuch, C.L., D.J. O'Mara, and D.A. Cory-Slechta. 1998. Low-level lead exposure selectively enhances dopamine overflow in nucleus accumbens: an in vivo electrochemistry time course assessment. *Toxicology and Applied Pharmacology* 150 (1):174-85.

160. National Dissemination Center for Children with Disabilities. 2010. *Disability Fact Sheet-No. 7: Learning Disabilities*. Retrieved April 6, 2010 from http://www.nichcy.org/InformationResources/Documents/NICHCY%20PUBS/fs7.pdf.

161. National Center for Learning Disabilities. 2010. *LD at a Glance*. Retrieved April 6, 2010 from http://www.ncld.org/ld-basics/ld-explained/basic-facts/learning-disabilities-at-a-glance.

162. Lyngbye, T., O.N. Hansen, A. Trillingsgaard, I. Beese, and P. Grandjean. 1990. Learning disabilities in children: significance of low-level lead-exposure and confounding factors. *Acta Paediatrica Scandinavica* 79 (3):352-60.

163. Marlowe, M., A. Cossairt, K. Welch, and J. Errera. 1984. Hair mineral content as a predictor of learning disabilities. *Journal of Learning Disabilities* 17 (7):418-21.

164. Pihl, R.O., and M. Parkes. 1977. Hair element content in learning disabled children. *Science* 198 (4313):204-6.

165. Lee, D.H., D.R. Jacobs, and M. Porta. 2007. Association of serum concentrations of persistent organic pollutants with the prevalence of learning disability and attention deficit disorder. *Journal of Epidemiology & Community Health* 61 (7):591-6.

166. Capel, I.D., M.H. Pinnock, H.M. Dorrell, D.C. Williams, and E.C. Grant. 1981. Comparison of concentrations of some trace, bulk, and toxic metals in the hair of normal and dyslexic children. *Clinical Chemistry* 27 (6):879-81.

167. Counter, S.A., L.H. Buchanan, and F. Ortega. 2008. Zinc protoporphyrin levels, blood lead levels and neurocognitive deficits in Andean children with chronic lead exposure. *Clinical Biochemistry* 41 (1-2):41-7.

168. Leviton, A., D. Bellinger, E.N. Allred, M. Rabinowitz, H. Needleman, and S. Schoenbaum. 1993. Pre- and postnatal low-level lead exposure and children's dysfunction in school. *Environmental Research* 60 (1):30-43.

169. Centers for Disease Control and Prevention. 2010. *Autism Spectrum Disorders: Signs & Symptoms*. Retrieved March 25, 2010 from http://www.cdc.gov/ncbddd/autism/signs.html.

170. Pessah, I.N., R.F. Seegal, P.J. Lein, J. LaSalle, B.K. Yee, J. Van De Water, and R.F. Berman. 2008. Immunologic and neurodevelopmental susceptibilities of autism. *Neurotoxicology* 29 (3):532-45.

171. Beaudet, A.L. 2007. Autism: highly heritable but not inherited. *Nature Medicine* 13 (5):534-6.

172. Sebat, J., B. Lakshmi, D. Malhotra, J. Troge, C. Lese-Martin, T. Walsh, B. Yamrom, S. Yoon, A. Krasnitz, J. Kendall, A. Leotta, D. Pai, R. Zhang, Y.H. Lee, J. Hicks, S.J. Spence, A.T. Lee, K. Puura, T. Lehtimaki, D. Ledbetter, P.K. Gregersen, J. Bregman, J.S. Sutcliffe, V. Jobanputra, W. Chung, D. Warburton, M.C. King, D. Skuse, D.H. Geschwind, T.C. Gilliam, K. Ye, and M. Wigler. 2007. Strong association of de novo copy number mutations with autism. *Science* 316 (5823):445-9.

173. Kinney, D.K., D.H. Barch, B. Chayka, S. Napoleon, and K.M. Munir. 2010. Environmental risk factors for autism: do they help cause de novo genetic mutations that contribute to the disorder? *Medical Hypotheses* 74 (1):102-6.

174. Gauthier, J., D. Spiegelman, A. Piton, R.G. Lafreniere, S. Laurent, J. St-Onge, L. Lapointe, F.F. Hamdan, P. Cossette, L. Mottron, E. Fombonne, R. Joober, C. Marineau, P. Drapeau, and G.A. Rouleau. 2009. Novel de novo SHANK3 mutation in autistic patients. *American Journal of Medical Genetics B: Neuropsychiatric Genetics* 150B (3):421-4.

175. Daoud, H., F. Bonnet-Brilhault, S. Vedrine, M.V. Demattei, P. Vourc'h, N. Bayou, C.R. Andres, C. Barthelemy, F. Laumonnier, and S. Briault. 2009. Autism and nonsyndromic mental retardation associated with a de novo mutation in the NLGN4X gene promoter causing an increased expression level. *Biological Psychiatry* 66 (10):906-10.

176. James, S.J., P. Cutler, S. Melnyk, S. Jernigan, L. Janak, D.W. Gaylor, and J.A. Neubrander. 2004. Metabolic biomarkers of increased oxidative stress and impaired methylation capacity in children with autism. *American Journal of Clinical Nutrition* 80 (6):1611-7.

177. James, S.J., S. Melnyk, S. Jernigan, M.A. Cleves, C.H. Halsted, D.H. Wong, P. Cutler, K. Bock, M. Boris, J.J. Bradstreet, S.M. Baker, and D.W. Gaylor. 2006. Metabolic endophenotype and related genotypes are associated with oxidative stress in children with autism. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* 141B (8):947-56.

178. Deth, R., C. Muratore, J. Benzecry, V.A. Power-Charnitsky, and M. Waly. 2008. How environmental and genetic factors combine to cause autism: A redox/methylation hypothesis. *Neurotoxicology* 29 (1):190-201.

179. Grether, J.K., M.C. Anderson, L.A. Croen, D. Smith, and G.C. Windham. 2009. Risk of autism and increasing maternal and paternal age in a large North American population. *American Journal of Epidemiology* 170 (9):1118-26.

180. Shelton, J.F., D.J. Tancredi, and I. Hertz-Picciotto. 2010. Independent and dependent contributions of advanced maternal and paternal ages to autism risk. *Autism Research* 3 (1):30-9.

181. Croen, L.A., D.V. Najjar, B. Fireman, and J.K. Grether. 2007. Maternal and paternal age and risk of autism spectrum disorders. *Archives of Pediatric & Adolescent Medicine* 161 (4):334-40.

182. Lauritsen, M.B., C.B. Pedersen, and P.B. Mortensen. 2005. Effects of familial risk factors and place of birth on the risk of autism: a nationwide register-based study. *Journal of Child Psychology and Psychiatry* 46 (9):963-71.

183. Chandley, A.C. 1991. On the parental origin of de novo mutation in man. *Journal of Medical Genetics* 28 (4):217-23.

184. Crow, J.F. 2000. The origins, patterns and implications of human spontaneous mutation. *Nature Reviews Genetics* 1 (1):40-7.

185. Adams, J.B., J. Romdalvik, V.M. Ramanujam, and M.S. Legator. 2007. Mercury, lead, and zinc in baby teeth of children with autism versus controls. *Journal of Toxicology and Environmental Health A* 70 (12):1046-51.

186. Bradstreet, J., D.A. Geier, J.J. Kartzinel, J.B. Adams, and M.R. Feier. 2003. A case-control study of mercury burden in children with autistic spectrum disorders. *Journal of American Physicians and Surgeons* 8 (3).

187. Desoto, M.C., and R.T. Hitlan. 2007. Blood levels of mercury are related to diagnosis of autism: a reanalysis of an important data set. *Journal of Child Neurology* 22 (11):1308-11.

188. Hertz-Picciotto, I., P.G. Green, L. Delwiche, R. Hansen, C. Walker, and I.N. Pessah. 2010. Blood mercury concentrations in CHARGE Study children with and without autism. *Environmental Health Perspectives* 118 (1):161-6.

189. Palmer, R.F., S. Blanchard, and R. Wood. 2009. Proximity to point sources of environmental mercury release as a predictor of autism prevalence. *Health Place* 15 (1):18-24.

190. Kalkbrenner, A.E., J.L. Daniels, J.-C. Chen, C. Poole, M. Emch, and J. Morrissey. 2010. Perinatal Exposure to Hazardous Air Pollutants and Autism Spectrum Disorders at Age 8. *Epidemiology* 21 (5):631-41.

191. Windham, G.C., L. Zhang, R. Gunier, L.A. Croen, and J.K. Grether. 2006. Autism spectrum disorders in relation to distribution of hazardous air pollutants in the san francisco bay area. *Environmental Health Perspectives* 114 (9):1438-44.

192. Centers for Disease Control and Prevention. *Mercury and Thimerosal: Vaccine Safety*. CDC. Retrieved October 12, 2010 from http://www.cdc.gov/vaccinesafety/Concerns/thimerosal/index.html.

193. Institute of Medicine Immunization Safety Review Committee. 2004. *Immunization Safety Review: Vaccines and Autism*. Washington, DC: National Academies Press. http://www.nap.edu/catalog.php?record_id=10997.

194. Larsson, M., B. Weiss, S. Janson, J. Sundell, and C.G. Bornehag. 2009. Associations between indoor environmental factors and parental-reported autistic spectrum disorders in children 6-8 years of age. *Neurotoxicology* 30 (5):822-31.

195. American Association of Intellectual and Developmental Disabilities. 2009. *FAQ on Intellectual Disability*. Retrieved March 23, 2009 from http://www.aamr.org/content_104.cfm?navID=22.

196. Schroeder, S.R. 2000. Mental retardation and developmental disabilities influenced by environmental neurotoxic insults. *Environmental Health Perspectives* 108 (Suppl. 3):395-9.

197. Daily, D.K., H.H. Ardinger, and G.E. Holmes. 2000. Identification and evaluation of mental retardation. *American Family Physician* 61 (4):1059-67, 1070.

198. Flint, J., and A.O. Wilkie. 1996. The genetics of mental retardation. *British Medical Bulletin* 52 (3):453-64.

199. Murphy, C., C. Boyle, D. Schendel, P. Decouflé, and M. Yeargin-Allsopp. 1998. Epidemiology of mental retardation in children. *Mental Retardation and Developmental Disabilities Research Reviews* 4 (1):6-13.

200. Weiss, B. 2000. Vulnerability of children and the developing brain to neurotoxic hazards. *Environmental Health Perspectives* 108 (Suppl. 3):375-81.

201. De Los Reyes, A., and A.E. Kazdin. 2005. Informant discrepancies in the assessment of childhood psychopathology: a critical review, theoretical framework, and recommendations for further study. *Psychological Bulletin* 131 (4):483-509.

202. Owens, P.L., K. Hoagwood, S.M. Horwitz, P.J. Leaf, J.M. Poduska, S.G. Kellam, and N.S. Ialongo. 2002. Barriers to children's mental health services. *Journal of the American Academy of Child and Adolescent Psychiatry* 41 (6):731-8.

203. U.S. Department of Health and Human Services. 1999. *Mental Health: A Report of the Surgeon General—Executive Summary*. Rockville, MD: U.S. DHS, Substance Abuse and Mental Health Services Administration, Center for Mental Health Services, National Institutes of Health, National Institute of Mental Health. http://www.surgeongeneral.gov/library/mentalhealth/pdfs/ExSummary-Final.pdf.

204. National Center on Birth Defects and Developmental Disabilities. 2010. *Attention-Deficit-Hyperactivity Disorder: Other Concerns & Conditions*. Centers for Disease Control and Prevention. Retrieved December 7, 2010 from http://www.cdc.gov/ncbddd/adhd/conditions.html.

205. Centers for Disease Control and Prevention. 2007. Prevalence of autism spectrum disorders---autism and developmental disabilities monitoring network, 14 sites, United States, 2002. In: Surveillance Summaries. *Morbidity and Mortality Weekly Report* 56 (No. SS-1):12-28.

206. Centers for Disease Control and Prevention. 2009. Prevalence of autism spectrum disorders --- autism and developmental disabilities monitoring network, United States, 2006. *Morbidity and Mortality Weekly Report* 58 (SS 10):1-20.

207. Kogan, M.D., S.J. Blumberg, L.A. Schieve, C.A. Boyle, J.M. Perrin, R.M. Ghandour, G.K. Singh, B.B. Strickland, E. Trevathan, and P.C. van Dyck. 2009. Prevalence of parent-reported diagnosis of autism spectrum disorder among children in the US, 2007. *Pediatrics* 124 (5):1395-403.

Metadata

Metadata for	National Health Interview Survey (NHIS)
Brief description of the data set	The National Health Interview Survey (NHIS) collects data on a broad range of health topics through personal household interviews. The results of NHIS provide data to track health status, health care access, and progress toward achieving national health objectives.
Who provides the data set?	Centers for Disease Control and Prevention, National Center for Health Statistics.
How are the data gathered?	Data are obtained using a health questionnaire through a personal household interview. Interviewers obtain information on health history and demographic characteristics, including age, household income, and race and ethnicity from respondents, or from a knowledgeable household adult for children age 17 years and younger.
What documentation is available describing data collection procedures?	See http://www.cdc.gov/nchs/nhis.htm for detailed survey documentation by survey year.
What types of data relevant for children's environmental health indicators are available from this database?	Health history (e.g., asthma, mental health, childhood illnesses). Smoking in residences (for selected years). Demographic information. Health care use and access information.
What is the spatial representation of the database (national or other)?	NHIS sampling procedures provide nationally representative data, and may also be analyzed by four broad geographic regions: North, Midwest, South and West. Analysis of data for any other smaller geographic areas (state, etc.) is possible only by special arrangement with the NCHS Research Data Center.
Are raw data (individual measurements or survey responses) available?	Data for each year of the NHIS are available for download and analysis (http://www.cdc.gov/nchs/nhis/nhis_questionnaires.htm). Annual reports from the NHIS are also available (http://www.cdc.gov/nchs/nhis/nhis_products.htm) as are interactive data tables (http://www.cdc.gov/nchs/hdi.htm). The files available for download generally contain individual responses to the survey questions; however, for some questions the responses are categorized Some survey responses are not publicly released.
How are database files obtained?	Raw data: http://www.cdc.gov/nchs/nhis.htm.
Are there any known data quality or data analysis concerns?	Data are self-reported, or (for individuals age 17 years and younger) reported by a knowledgeable household adult, usually a parent. Responses to some demographic questions (race/ethnicity, income) are statistically imputed for survey participants lacking a reported response.
What documentation is available describing QA procedures?	http://www.cdc.gov/nchs/data/series/sr_02/sr02_130.pdf provides a summary of QA procedures.
For what years are data available?	Data from the NHIS are available from 1957–present. Availability of data addressing particular issues varies based on when questions were added to the NHIS. The survey is redesigned on a regular basis; many questions of interest for children's environmental health indicators were modified or first asked with the redesign that was implemented in 1997. For environmental tobacco smoke (regular smoking in the home), comparable data are available for 1994 and 2005.
What is the frequency of	Annually. Sampling and interviewing are continuous throughout each year.

Metadata for	**National Health Interview Survey (NHIS)**
data collection?	
What is the frequency of data release?	Annually.
Are the data comparable across time and space?	Survey design and administration are consistent across locations and from year to year. Many questions were revised or added in 1997, so data for prior years may not be comparable to data from 1997 to present.
Can the data be stratified by race/ethnicity, income, and location (region, state, county or other geographic unit)?	Race, ethnicity, income. Region (four regions only).

1

Methods

Indicator

ND1. Percentage of children ages 5 to 17 years reported to have attention deficit/hyperactivity disorder, by sex, 1997-2008.
ND2. Percentage of children ages 5 to 17 years reported to have a learning disability, by sex, 1997-2008.
ND3. Percentage of children ages 5 to 17 years reported to have autism, 1997-2008.
ND4. Percentage of children ages 5 to 17 years reported to have intellectual disability (mental retardation), 1997-2008.

Summary

Since 1957, the National Center for Health Statistics, a division of the Centers for Disease Control and Prevention, has conducted the National Health Interview Survey (NHIS), a series of annual U.S. national surveys of the health status of the noninstitutionalized civilian population. These indicators use responses to questions on neurodevelopmental disorders for children ages 5 to 17 from the NHIS 1997 to 2008 surveys. Indicator ND1 gives the trends in the percentages of children reported to have attention deficit/hyperactivity disorder, stratified by sex. Indicator ND2 gives the trends in the percentages of children reported to have a learning disability, stratified by sex. Indicator ND3 gives the trends in the percentages of children reported to have autism. Indicator ND4 gives the trends in the percentages of children reported to have intellectual disability (mental retardation), stratified by sex. For each indicator, the corresponding table ND1a, ND2a, ND3a, and ND4a gives the percentage of children reported to have the given neurodevelopmental disorder over the period 2005 to 2008, stratified both by age and sex. For each indicator, the corresponding table ND1b, ND2b, ND3b, and ND4b gives the percentage of children reported to have the given neurodevelopmental disorder over the period 2005 to 2008, stratified both by race/ethnicity (using NHIS information on race and Hispanic origin) and family income (using reported or imputed NHIS poverty-income ratio data for each respondent). Percentages are calculated by combining positive responses to the relevant questions with the survey weights for each respondent. The survey weights are the annual numbers of children in the noninstitutionalized civilian population represented by each respondent.

Data Summary

Indicator	ND1. Percentage of children ages 5 to 17 years reported to have attention deficit/hyperactivity disorder (ADHD), by sex, 1997-2008. ND2. Percentage of children ages 5 to 17 years reported to have a learning disability, 1997-2008. ND3. Percentage of children ages 5 to 17 years reported to have autism, 1997-2008. ND4. Percentage of children ages 5 to 17 years reported to have intellectual disability (mental retardation), 1997-2008.

Indicator	ND1. Percentage of children ages 5 to 17 years reported to have attention deficit/hyperactivity disorder (ADHD), by sex, 1997-2008. ND2. Percentage of children ages 5 to 17 years reported to have a learning disability, 1997-2008. ND3. Percentage of children ages 5 to 17 years reported to have autism, 1997-2008. ND4. Percentage of children ages 5 to 17 years reported to have intellectual disability (mental retardation), 1997-2008.					
Time Period	1997-2008					
Data	Neurodevelopmental disorder prevalence in children ages 5 to 17					
Years (1997-2002)	1997	1998	1999	2000	2001	2002
ADHD non-missing responses	9,971	9,536	9,155	9,481	9,617	8,845
ADHD missing responses	35	28	14	25	21	31
Learning disability non-missing responses	9,974	9,552	9,155	9,490	9,624	8,862
Learning disability missing responses	32	12	14	16	14	14
Autism non-missing responses	9,996	9,557	9,165	9,501	9,633	8,873
Autism missing responses	10	7	4	5	5	3
Intellectual disability non-missing responses	9,991	9,549	9,165	9,494	9,628	8,856
Intellectual disability missing responses	15	15	4	12	10	20
Years (2003-2008)	2003	2004	2005	2006	2007	2008
ADHD non-missing responses	8,722	8,813	8,952	7,003	6,595	6,311
ADHD missing	16	17	22	16	9	17

Indicator	ND1. Percentage of children ages 5 to 17 years reported to have attention deficit/hyperactivity disorder (ADHD), by sex, 1997-2008. ND2. Percentage of children ages 5 to 17 years reported to have a learning disability, 1997-2008. ND3. Percentage of children ages 5 to 17 years reported to have autism, 1997-2008. ND4. Percentage of children ages 5 to 17 years reported to have intellectual disability (mental retardation), 1997-2008.					
responses						
Learning disability non-missing responses	8,724	8,823	8,959	7,004	6,583	6,319
Learning disability missing responses	14	7	15	15	21	9
Autism non-missing responses	8,730	8,825	8,971	7,012	6,600	6,328
Autism missing responses	8	5	3	7	4	0
Intellectual disability non-missing responses	8,728	8,828	8,968	7,015	6,603	6,322
Intellectual disability missing responses	10	2	6	4	1	6

Overview of Data Files

The following files are needed to calculate this indicator. All these files together with the survey documentation and SAS programs for reading in the data are available at the NHIS website: http://www.cdc.gov/nchs/nhis.htm.

- NHIS 1997-2008: Sample child file samchild.dat. Person file personsx.dat, Family file familyxx.dat, Imputed income files 2005-2008: incmimp1.dat, incmimp2.dat, incmimp3.dat, incmimp4.dat, and incmimp5.dat. The Sample child file is an ASCII file containing interview data for children ages 17 years and under. Demographic data are obtained from the Person and Family files. The demographic variables needed for these indicators are the sample child survey weight (WTFA_SC), age (AGE_P), sex (SEX), the pseudo-stratum (STRATUM), the pseudo-PSU (PSU), the race (RACERPI2, using the

1997 OMB definitions), the Hispanic origin (ORIGIN_I), and the detailed Hispanic origin HISPAN_I. The pseudo-stratum and pseudo-PSU variables provide an approximation to the exact sample design variables, and were created by CDC by combining stratum information in a manner to protect the confidentiality of the publicly released data. From each of the imputed income files we need the imputed poverty income ratio (RAT_CATI), which gives the poverty income ratio category calculated from the reported exact family income, if available, or else gives the imputed category randomly generated by multiple imputation using regression models. The files are sorted and merged using the identifiers HHX, FMX, and FPX. The questionnaire variables needed for these analyses are the responses to the following questions: "Has a doctor or health professional ever told you that <child's name> had Attention Deficit/Hyperactivity Disorder (ADHD) or Attention Deficit Disorder (ADD)?" "Has a doctor or health professional ever told you that <child's name> had Autism?" "Has a doctor or health professional ever told you that <child's name> had Mental Retardation?" and "Has a representative from a school or a health professional ever told you that <child's name> had a learning disability?"

National Health Interview Survey (NHIS)

Since 1957, the National Center for Health Statistics, a division of the Centers for Disease Control and Prevention, has conducted the National Health Interview Survey (NHIS), a series of annual U.S. national surveys of the health status of the noninstitutionalized civilian population. This indicator uses responses to neurodevelopmental disorder prevalence questions in children ages 5 to 17 years for the surveys from 1997 to 2008. The NHIS data were obtained from the NHIS website: http://www.cdc.gov/nchs/nhis.htm.

For these indicators we used the responses to the following questions. Attention Deficit/Hyperactivity Disorder: "Has a doctor or health professional ever told you that <child's name> had Attention Deficit/Hyperactivity Disorder (ADHD) or Attention Deficit Disorder (ADD)?" Learning disability: "Has a representative from a school or a health professional ever told you that <child's name> had a learning disability?"Autism: "Has a doctor or health professional ever told you that <child's name> had Autism?" Intellectual disability (mental retardation): "Has a doctor or health professional ever told you that <child's name> had Mental Retardation?"

The NHIS uses a complex multi-stage, stratified, clustered sampling design. Certain demographic groups have been deliberately over-sampled. Oversampling is performed to increase the reliability and precision of estimates of health status indicators for these population subgroups. From 1997 to 2005, Blacks and Hispanics were over-sampled. From 2006, Blacks, Hispanics, and Asians were over-sampled. The publicly released data includes survey weights to adjust for the over-sampling, non-response, and non-coverage. The statistical analyses used the sample child survey weights (WTFA_SC, 1997 and later) to re-adjust the responses to represent the national population.

The sample design was changed in 2006. New strata were defined and PSUs were selected from these new strata. For example, pseudo-stratum 1 for 1997-2005 is unrelated to pseudo-stratum 1

for 2006-2008. To properly treat the 2006-2008 data as independent from the 2005 data, 1,000 was added to each of the year 2006, 2007, and 2008 pseudo-stratum numbers for these statistical analyses.[1]

Race/Ethnicity and Family Income

For Tables ND1b, ND2b, ND3b, and ND4b, the prevalence percentages were calculated for demographic strata defined by the combination of race/ethnicity and family income.

The family income was characterized based on the RAT_CATI variable, which gives the level of the ratio of the family income to the poverty level. The National Center for Health Statistics obtained the family income for the respondent's family during the family interview. The U.S. Census Bureau defines annual poverty level money thresholds varying by family size and composition. The poverty income ratio (PIR) is the family income divided by the poverty level for that family. The public release variable RAT_CATI gives the value of the PIR for various ranges, Under 0.5, 0.5-0.74, 0.75 to 0.99, …, 4.50-4.99, 5.00 and Over.

Family income was stratified into the following groups:

- Below Poverty Level: PIR < 1, i.e., RAT_CATI = 1, 2, or 3.
- Between 100% and 200% of Poverty Level: 1 ≤ PIR < 2, i.e., RAT_CATI = 4, 5, 6, or 7.
- Above 200% of Poverty level: PIR ≥ 2, i.e., RAT_CATI = 8, 9, 10, 11, 12, 13 or 14.
- Above Poverty Level: PIR ≥ 1 (combines the previous two groups).
- Unknown Income: PIR is missing ("undefinable"), i.e., RAT_CATI = 96.[2]

Approximately 30% of families did not report their exact family income. From 1997 to 2006, the majority of these families either reported their income by selecting from two categories (above or below $20,000) or from 44 categories. For 2007 and later, the income questions were revised, so that families not reporting an exact income were first asked to report their income as the two categories above or below $50,000, and were then asked appropriate additional questions to refine the income range as either 0-$34,999, $35,000-$49,999, $50,000-74,999, $75,000-$99,999, or $100,000 and above. In 2007 and 2008, 92% of families either gave the exact income or a categorical response.

NCHS reports[3] evidence that the non-response to the income question is related to person-level or family-level characteristics, including items pertaining to health. Therefore, treating the missing responses as being randomly missing would lead to biased estimates. To address this problem, NCHS applied a statistical method called "multiple imputation" to estimate or "impute"

[1] The addition of 1,000 was chosen to make the stratum numbers for 2005 and earlier distinct from the stratum numbers for 2006 and later. This follows the recommendations in Appendix III of the survey description document "2008 National Health Interview Survey (NHIS) Public Use Data Release NHIS Survey Description," CDC, June 2009, http://www.cdc.gov/nchs/nhis/quest_data_related_1997_forward.htm

[2] Although missing values of family income were statistically imputed for the vast majority of respondents, there were a few respondents that still had an unknown income after the income imputation.

[3] "Multiple imputation of family income and personal earnings in the National Health Interview Survey: methods and examples," http://www.cdc.gov/nchs/nhis/2008imputedincome.htm, August, 2009.

the family income based on the available family income and personal earnings information and on responses to other survey equations. A series of regression models were used to predict the exact family income from the available responses. Five sets of simulated family income values were generated for each family that did not report their exact family income. In this manner, NCHS generated five data sets, each containing a complete set of family income values (either the reported or the imputed values). The poverty income ratio categories were calculated from the income values and the family size and composition variables. An estimated prevalence percentage was computed for each of the five data sets. The overall estimated prevalence percentage is the arithmetic mean of the five estimates.

Race was characterized using the race variable for the 1997 OMB standards,[4] RACERPI2. The possible values of this variable are:

- 1. White only
- 2. Black /African American only
- 3. American Indian Alaska Native (AIAN) only
- 4. Asian only
- 5. Race group not releasable
- 6. Multiple race

The Native Hawaiian or Other Pacific Islander (NHOPI) race group is not specified in the public release version due to confidentiality concerns. Respondents with the single race NHOPI have RACERPI2 = 5 and respondents of multiple races including NHOPI have RACERPI2 = 6.

The ORIGIN_I variable indicates whether or not the ethnicity is Hispanic or Latino. ORIGIN_I = 1 if the respondent is Hispanic or Latino. ORIGIN_I = 2 if the respondent is not Hispanic or Latino.

The HISPAN_I variable indicates the specific Hispanic origin or ancestry.

- 00 Multiple Hispanic
- 01 Puerto Rico
- 02 Mexican
- 03 Mexican-American
- 04 Cuban/Cuban American
- 05 Dominican (Republic)
- 06 Central or South American
- 07 Other Latin American, type not specified
- 08 Other Spanish
- 09 Hispanic/Latino/Spanish, non-specific type

[4] Revised race standards were issued by the Office of Management and Budget in 1997 and were to be fully implemented across the federal statistical system by January 2003. Under the new standards, the minimum available race categories include: White, Black, AIAN, Asian, and Native Hawaiian or Other Pacific Islander (NHOPI). A very important change was that under the new standards, respondents may select more than one race category.

- 10 Hispanic/Latino/Spanish, type refused
- 11 Hispanic/Latino/Spanish, type not ascertained
- 12 Not Hispanic/Spanish origin

The race/ethnicity was defined based on RACERPI2, ORIGIN_I, and HISPAN_I:

Race/ethnicity:

- White non-Hispanic: RACERPI2 =1, ORIGIN_I = 2
- Black or African-American, Non Hispanic: RACERPI2 = 2, ORIGIN_I = 2
- Asian non-Hispanic: RACERPI2 = 4, ORIGIN_I = 2
- Hispanic: ORIGIN_I = 1
    - Mexican: ORIGIN_I = 1 and HISPAN_I = 02, 03
    - Puerto Rican: ORIGIN_I = 1 and HISPAN_I = 01
- Other: RACERPI2 = 3, 5 or 6, ORIGIN_I = 2
    - American Indian, Alaska Native, Non-Hispanic: RACERPI2 = 3, ORIGIN_I = 2

The "Other" category includes non-Hispanic respondents reporting multiple races, or reporting a single race that is neither White, Black, African-American, or Asian.

Some respondents gave missing or incomplete answers to the race/ethnicity questions. In those cases NCHS applied a statistical method called "hot-deck imputation" to estimate or "impute" the race or ethnicity based on the race/ethnicity responses for other household members, if available, or otherwise based on information from other households. The NHIS variables ORIGIN_I , HISPAN_I, and RACERPI2 use imputed responses if the original answer was missing or incomplete.

Calculation of Indicator

Indicator ND1 is the percentage of children reported to have attention deficit/hyperactivity disorder. Indicator ND2 is the percentage of children reported to have a learning disability. Indicator ND3 is the percentage of children reported to have autism. Indicator ND4 is the percentage of children reported to have intellectual disability (mental retardation). For each indicator, the corresponding table ND1a, ND2a, ND3a, and ND4a gives the percentage of children reported to have the given neurodevelopmental disorder over the period 2005 to 2008, stratified both by age and sex. For each indicator, the corresponding table ND1b, ND2b, ND3b, and ND4b gives the percentage of children reported to have the given neurodevelopmental disorder over the period 2005 to 2008, stratified both by race/ethnicity and family income.

To simply demonstrate the calculations, we will describe the calculations for the indicator ND1 for 2008, using the NHIS 2008 responses to the question : "Has a doctor or health professional ever told you that <child's name> had Attention Deficit/Hyperactivity Disorder (ADHD) or Attention Deficit Disorder (ADD)?" We shall refer to this question as the ADHD question. The calculations for the other indicators and supplementary tables use exactly the same method,

except for the stratification by family income, which uses the five sets of imputed income values as demonstrated below. We have rounded all the numbers to make the calculations easier.

We begin with all the non-missing responses to the ADHD question in the NHIS 2008 survey for children ages 5 to 17 years. Assume for the sake of simplicity that Yes or No responses were available for every sampled child. Each sampled child has an associated survey weight that estimates the total number of U.S. children in 2008 represented by that sampled child. For example, the first response for a child aged 5 to 17 years was No with a survey weight of 2,000, and so represents 2,000 children ages 5 to 17 years. A second child aged 5 to 17 years responded Yes with a survey weight of 6,000, and so represents 6,000 children ages 5 to 17 years. A third child aged 5 to 17 years responded No with a survey weight of 10,000, and so represents 10,000 children ages 5 to 17 years. The total of the survey weights for the sampled children equals 50 million, the total U.S. population of children ages 5 to 17 years for the year 2008.

To calculate the proportion of children ages 5 to 17 years with ADHD/ADD, we can use the survey weights to expand the data to the 2008 U.S. population of 50 million children ages 5 to 17 years. We have 2,000 No responses from the first child, 6,000 Yes responses from the second child, 10,000 Yes responses from the third child, and so on. Of these 50 million responses, a total of 4.5 million responses are Yes and the remaining 45.5 million are No. Thus 4.5 million of the 50 million children have ADHD/ADD, giving a proportion of about 9%.

In reality, the calculations need to take into account that Yes or No responses were not reported for every respondent, and to use exact rather than rounded numbers. There were non-missing responses for 6,311 of the 6,328 sampled children ages 5 to 17 years. (Don't know responses or refusals to answer are treated as missing). The survey weights for all 6,328 sampled children add up to 53.1 million, the total U.S. population of children ages 5 to 17 years. The survey weights for the 6,311 sampled children with non-missing responses add up to 52.9 million. Thus the available data represent 52.9 million children, which is more than 99 %, but not all, of the 2008 U.S. population of children ages 5 to 17 years. The survey weights for the Yes responses add up to 4.8 million, which is 9.1 % of the population with responses (4.8 million/52.9 million = 9.1 %). Thus we divide the sum of the weights for participants with Yes responses by the sum of the weights for participants with non-missing responses. These calculations assume that the sampled children with non-missing responses are representative of the children with missing responses.

For calculation of prevalence by income group in Tables ND1b, ND2b, ND3b, and ND4b, we use the five sets of imputed income values, which each give different results. For example, suppose we wish to estimate the proportion of White non-Hispanic children below the poverty level with ADHD/ADD in 2005-2008. Using the above calculation method applied for White non-Hispanic children below the poverty level for the combined set of years 2005 to 2008, the proportions for the five sets of imputed values are: 13.4 %, 13.0 %, 13.1 %, 13.4 %, and 13.1 %. The estimated proportion of White non-Hispanic children below the poverty level with ADHD/ADD in 2005-2008 is given by the average of the five estimates, (13.4 + 13.0 + 13.1 + 13.4 + 13.1) /5 = 13.2 %.

Equations

The following equations give the mathematical calculations for the example of White non-Hispanic children below the poverty level using the ADHD question. Let w(i) denote the survey weight for the i'th surveyed White non-Hispanic child of ages 5 to 17 years. Exclude any surveyed children with a response other than Yes or No. For the ADHD question, let the response indicator c(i) = 1 if the i'th surveyed White non-Hispanic child had a Yes response and let c(i) = 0 if the i'th surveyed White non-Hispanic child had a No response. Let the income indicator d(i, j) = 1 if the i'th surveyed White non-Hispanic child was below the poverty level according to the j'th set of imputed values and let d(i, j) = 0 if the i'th surveyed White non-Hispanic child was not below the poverty level according to the j'th set of imputed values.

1. Fix j = 1, 2, 3, 4 or 5. Sum (over i) all the survey weights multiplied by the income indicators to get the total weight W(j) for set j:

$$W(j) = \Sigma\, w(i) \times d(i, j)$$

2. Fix j = 1, 2, 3, 4 or 5. Sum (over i) all the survey weights multiplied by the response indicators and multiplied by the income indicators to get the total weight D(j) for set j for White non-Hispanic children below the poverty level with a Yes response:

$$D(j) = \Sigma\, w(i) \times c(i) \times d(i, j)$$

3. Divide D(j) by W(j) to get the percentage of children with ADHD/ADD in set j:

$$\text{Percentage (j)} = (D(j) / W(j)) \times 100\ \%$$

4. Average the percentages across the 5 sets to get the estimated percentage of children with ADHD/ADD:

$$\text{Percentage} = [\text{Percentage (1)} + \text{Percentage (2)} + \text{Percentage (3)} + \text{Percentage (4)} + \text{Percentage (5)}] / 5$$

If the demographic group of interest includes all incomes, then the percentages will be equal for all five sets of imputed values, so the calculation in steps 1 to 3 need only be done for j =1, and step 4 is not required.

Relative Standard Error

The uncertainties of the percentages were calculated using SUDAAN® (Research Triangle Institute, Research Triangle Park, NC 27709) statistical survey software. SUDAAN was used to calculate the estimated percentages and the standard errors of the estimated percentages. The standard error is the estimated standard deviation of the percentage, and this depends upon the survey design. The standard error calculation also incorporates the extra uncertainty due to the multiple imputations of the income variables (based on the variation between the estimated percentages from each of the five sets of imputations). For this purpose, the public release

version of NHIS includes the variables STRATUM and PSU, which are the Masked Variance Unit pseudo-stratum and pseudo-primary sampling unit (pseudo-PSU). For approximate variance estimation, the survey design can be approximated as being a stratified random sample with replacement of the pseudo-PSUs from each pseudo-stratum; the true stratum and PSU variables are not provided in the public release version to protect confidentiality.

The sample design was changed in 2006. New strata were defined and PSUs were selected from these new strata. For example, pseudo-stratum 1 for 2005 is unrelated to pseudo-stratum 1 for 2006-2008. To properly treat the 2006-2008 data as independent from the 2005 data, 1,000 was added to each of the year 2006, 2007, and 2008 pseudo-stratum numbers for these statistical analyses.

The relative standard error is the standard error divided by the estimated percentage:

$$\text{Relative Error (\%)} = [\text{Standard Error (Percentage)} / \text{Percentage}] \times 100\%$$

Percentages with a relative error less than 30% were treated as being reliable and were tabulated. Percentages with a relative error greater than or equal to 30% but less than 40% were treated as being unstable; these values were tabulated but were flagged to be interpreted with caution. Percentages with a relative standard error greater than or equal to 40%, or without an estimated relative standard error, were treated as being unreliable; these values were not tabulated and were flagged as having a large uncertainty.

Statistical Comparisons

Statistical analyses of the percentages of children with a positive response to the question of interest were used to determine whether the differences between percentages for different demographic groups were statistically significant. Using a logistic regression model, the logarithm of the odds that a given child has a positive response is assumed to be the sum of explanatory terms for the child's age group, sex, income group, and/or race/ethnicity. The odds of a positive response is the probability of a positive response divided by the probability of a negative response. Thus if two demographic groups have similar (or equal) probabilities of a positive response, then they will also have similar (or equal) values for the logarithm of the odds. Using this model, the difference in the percentage between different demographic groups is statistically significant if the difference between the corresponding sums of explanatory terms is statistically significantly different from zero. The uncertainties of the regression coefficients were calculated using SUDAAN® (Research Triangle Institute, Research Triangle Park, NC 27709) statistical survey software to account for the survey weighting and design. A p-value at or below 0.05 implies that the difference is statistically significant at the 5% significance level. No adjustment is made for multiple comparisons.

For these statistical analyses we used two income groups: below poverty level, and at or above poverty level. The small number of children with unknown (and unimputed) incomes were included in the at or above poverty level group. For the main analyses we also used five

race/ethnicity groups: White non-Hispanic, Black non-Hispanic, Asian non-Hispanic, Hispanic, Other. In addition, for specific comparisons between the Mexican and Puerto Rican subgroups, we applied a similar statistical analysis using three ethnicity groups: Mexican, Puerto Rican, Other Hispanic or Non-Hispanic. We also used two age groups: 5-10 and 11-17.

For each type of comparison, we present unadjusted and adjusted analyses. The unadjusted analyses directly compare a percentage between different demographic groups. The adjusted analyses add other demographic explanatory variables to the statistical model and use the statistical model to account for the possible confounding effects of these other demographic variables. For example, the unadjusted race/ethnicity comparisons use and compare the percentages between different race/ethnicity pairs. The adjusted analyses add age, sex, and income terms to the statistical model and compare the percentages between different race/ethnicity pairs after accounting for the effects of the other demographic variables. For example, if White non-Hispanics tend to have higher family incomes than Black non-Hispanics, and if the prevalence of a neurodevelopmental disorder strongly depends on family income only, then the unadjusted differences between these two race/ethnicity groups would be significant but the adjusted difference (taking into account income) would not be significant.

Comparisons of the prevalence of each neurodevelopmental disorder in children ages 5 to 17 years between pairs of race/ethnicity groups are shown in Table 1. For the unadjusted "All incomes" comparisons, the only explanatory variables are terms for each race/ethnicity group. For these unadjusted comparisons, the statistical tests compare the percentage for each pair of race/ethnicity groups. For the adjusted "All incomes (adjusted for age, sex, income)" comparisons, the explanatory variables are terms for each race/ethnicity group together with terms for each age, sex, and income group. For these adjusted comparisons, the statistical test compares the pair of race/ethnicity groups after accounting for any differences in the age, sex, and income distributions between the race/ethnicity groups.

In Table 1, for the unadjusted "Below Poverty Level" and "At or Above Poverty Level" comparisons, the only explanatory variables are terms for each of the 10 race/ethnicity/income combinations (combinations of five race/ethnicity groups and two income groups). For example, in row 1, the p-value for "Below Poverty Level" compares White non-Hispanics below the poverty level with Black non-Hispanics below the poverty level. The same set of explanatory variables are used in Table 2 for the unadjusted comparisons between one race/ethnicity group below the poverty level and the same or another race/ethnicity group at or above the poverty level. The corresponding adjusted analyses include extra explanatory variables for age and sex, so that race/ethnicity/income groups are compared after accounting for any differences due to age or sex.

Additional comparisons are shown in Table 3. The AGAINST = "age" unadjusted p-value compares the percentages for different age groups. The adjusted p-value includes adjustment terms for income, sex, and race/ethnicity in the model. The AGAINST = "sex" unadjusted p-value compares the percentages for boys and girls. The adjusted p-value includes adjustment terms for age, income, and race/ethnicity in the model. The AGAINST = "income" unadjusted p-value compares the percentages for those below poverty level with those at or above poverty level. The adjusted p-value includes adjustment terms for age, sex, and race/ethnicity in the

model. The AGAINST = "year" p-value examines whether the linear trend in the percentages is statistically significant; the adjusted model for trend adjusts for demographic changes in the populations from year to year by including terms for age, sex, income, and race/ethnicity. The SUBSET column specifies the demographic group of interest. For the AGAINST = "age," "sex," and "income" comparisons, the comparisons are for all children and so no SUBSET is defined. For the AGAINST = "year" trend analyses, results are given for the overall trend (SUBSET = missing) and for the trends in each sex, age, or income group, so that, for example, the SUBSET = "Males" examines whether there is a statistically significant trend for boys ages 5 to 17 years. The unadjusted p-values for the AGAINST="year" analyses are not shown.

For more details on these statistical analyses, see the memorandum by Cohen (2010).[5]

Table 1. Statistical significance tests comparing the percentages of children ages 5 to 17 years with neurodevelopmental disorders, between pairs of race/ethnicity groups, for 2005-2008.

			P-VALUES					
Variable	**RACE1**	**RACE2**	**All incomes**	**All incomes (adjusted for age, sex, income)**	**Below Poverty Level**	**Below Poverty Level (adjusted for age, sex)**	**At or Above Poverty Level**	**At or Above Poverty Level (adjusted for age, sex)**
ADHD/ADD	White non-Hispanic	Black non-Hispanic	0.060	0.002	0.090	0.044	0.010	0.010
ADHD/ADD	White non-Hispanic	Asian non-Hispanic	< 0.0005	< 0.0005	0.001	< 0.0005	< 0.0005	< 0.0005
ADHD/ADD	White non-Hispanic	Hispanic	< 0.0005	< 0.0005	< 0.0005	< 0.0005	< 0.0005	< 0.0005
ADHD/ADD	White non-Hispanic	Other	0.535	0.711	0.609	0.823	0.945	0.847
ADHD/ADD	Black non-Hispanic	Asian non-Hispanic	< 0.0005	< 0.0005	0.003	0.002	< 0.0005	< 0.0005
ADHD/ADD	Black non-Hispanic	Hispanic	< 0.0005	< 0.0005	< 0.0005	< 0.0005	< 0.0005	< 0.0005
ADHD/ADD	Black non-Hispanic	Other	0.145	0.065	0.177	0.236	0.196	0.130
ADHD/ADD	Asian non-Hispanic	Hispanic	< 0.0005	< 0.0005	0.033	0.028	< 0.0005	< 0.0005
ADHD/ADD	Asian non-Hispanic	Other	< 0.0005	< 0.0005	0.001	0.001	< 0.0005	< 0.0005
ADHD/ADD	Hispanic	Other	< 0.0005	< 0.0005	< 0.0005	0.001	< 0.0005	< 0.0005
ADHD/ADD	Mexican	Puerto Rican	< 0.0005	< 0.0005	< 0.0005	< 0.0005	0.005	0.005
Learning disability	White non-Hispanic	Black non-Hispanic	0.860	0.097	0.023	0.013	0.443	0.454
Learning disability	White non-Hispanic	Asian non-Hispanic	< 0.0005	< 0.0005	0.002	0.001	< 0.0005	< 0.0005
Learning disability	White non-Hispanic	Hispanic	< 0.0005	< 0.0005	< 0.0005	< 0.0005	0.001	0.002
Learning disability	White non-Hispanic	Other	0.926	0.741	0.789	0.637	0.633	0.739
Learning disability	Black non-Hispanic	Asian non-Hispanic	< 0.0005	< 0.0005	0.014	0.012	< 0.0005	< 0.0005
Learning disability	Black non-Hispanic	Hispanic	0.003	0.009	0.016	0.020	0.077	0.100

[5] Cohen, J. 2010. *Selected statistical methods for testing for trends and comparing years or demographic groups in ACE NHIS and NHANES indicators.* Memorandum submitted to Dan Axelrad, EPA, 21 March, 2010.

			P-VALUES					
Variable	**RACE1**	**RACE2**	**All incomes**	**All incomes (adjusted for age, sex, income)**	**Below Poverty Level**	**Below Poverty Level (adjusted for age, sex)**	**At or Above Poverty Level**	**At or Above Poverty Level (adjusted for age, sex)**
Learning disability	Black non-Hispanic	Other	0.996	0.610	0.390	0.451	0.926	0.970
Learning disability	Asian non-Hispanic	Hispanic	< 0.0005	< 0.0005	0.089	0.076	< 0.0005	< 0.0005
Learning disability	Asian non-Hispanic	Other	< 0.0005	< 0.0005	0.008	0.009	< 0.0005	< 0.0005
Learning disability	Hispanic	Other	0.111	0.053	0.041	0.059	0.379	0.338
Learning disability	Mexican	Puerto Rican	< 0.0005	0.001	0.002	0.002	0.073	0.074
Autism	White non-Hispanic	Black non-Hispanic	0.016	0.017	0.061	0.055	0.141	0.160
Autism	White non-Hispanic	Asian non-Hispanic	0.102	0.110	0.944	0.952	0.031	0.034
Autism	White non-Hispanic	Hispanic	0.005	0.003	0.109	0.097	0.015	0.013
Autism	White non-Hispanic	Other	0.483	0.569	0.872	0.963	0.541	0.560
Autism	Black non-Hispanic	Asian non-Hispanic	0.679	0.752	0.238	0.236	0.233	0.229
Autism	Black non-Hispanic	Hispanic	0.853	0.812	0.641	0.645	0.553	0.492
Autism	Black non-Hispanic	Other	0.069	0.080	0.207	0.238	0.194	0.216
Autism	Asian non-Hispanic	Hispanic	0.764	0.860	0.361	0.356	0.393	0.422
Autism	Asian non-Hispanic	Other	0.079	0.104	0.947	0.992	0.038	0.043
Autism	Hispanic	Other	0.052	0.055	0.314	0.355	0.090	0.090
Autism	Mexican	Puerto Rican	0.824	0.781	0.947	0.979	0.720	0.687
Intellectual disability	White non-Hispanic	Black non-Hispanic	0.010	0.127	0.810	0.820	0.048	0.046
Intellectual disability	White non-Hispanic	Asian non-Hispanic	0.877	0.843	0.488	0.495	0.409	0.416
Intellectual disability	White non-Hispanic	Hispanic	0.080	0.371	0.925	0.935	0.247	0.248
Intellectual disability	White non-Hispanic	Other	0.687	0.875	0.977	0.954	0.864	0.862
Intellectual disability	Black non-Hispanic	Asian non-Hispanic	0.146	0.315	0.561	0.563	0.077	0.078
Intellectual disability	Black non-Hispanic	Hispanic	0.477	0.527	0.875	0.876	0.430	0.422
Intellectual disability	Black non-Hispanic	Other	0.399	0.525	0.859	0.842	0.473	0.472
Intellectual disability	Asian non-Hispanic	Hispanic	0.286	0.502	0.511	0.513	0.177	0.181
Intellectual disability	Asian non-Hispanic	Other	0.671	0.791	0.560	0.551	0.475	0.479
Intellectual disability	Hispanic	Other	0.653	0.770	0.929	0.911	0.750	0.753
Intellectual disability	Mexican	Puerto Rican	0.160	0.189	0.631	0.627	0.148	0.146

Table 2. Statistical significance tests comparing the percentages of children ages 5 to 17 years with neurodevelopmental disorders, between pairs of race/ethnicity/income groups at different income levels, for 2005-2008.

1

			P-VALUES	
Variable	RACEINC1	RACEINC2	Unadjusted	Adjusted (for age, sex)
ADHD/ADD	White non-Hispanic, < PL	White non-Hispanic, >= PL	< 0.0005	< 0.0005
ADHD/ADD	White non-Hispanic, < PL	Black non-Hispanic, >= PL	< 0.0005	< 0.0005
ADHD/ADD	White non-Hispanic, < PL	Asian non-Hispanic, >= PL	< 0.0005	< 0.0005
ADHD/ADD	White non-Hispanic, < PL	Hispanic, >= PL	< 0.0005	< 0.0005
ADHD/ADD	White non-Hispanic, < PL	Other, >= PL	0.025	0.018
ADHD/ADD	Black non-Hispanic, < PL	White non-Hispanic, >= PL	0.250	0.184
ADHD/ADD	Black non-Hispanic, < PL	Black non-Hispanic, >= PL	0.011	0.008
ADHD/ADD	Black non-Hispanic, < PL	Asian non-Hispanic, >= PL	< 0.0005	< 0.0005
ADHD/ADD	Black non-Hispanic, < PL	Hispanic, >= PL	< 0.0005	< 0.0005
ADHD/ADD	Black non-Hispanic, < PL	Other, >= PL	0.434	0.494
ADHD/ADD	Asian non-Hispanic, < PL	White non-Hispanic, >= PL	0.003	0.003
ADHD/ADD	Asian non-Hispanic, < PL	Black non-Hispanic, >= PL	0.009	0.008
ADHD/ADD	Asian non-Hispanic, < PL	Asian non-Hispanic, >= PL	0.475	0.424
ADHD/ADD	Asian non-Hispanic, < PL	Hispanic, >= PL	0.052	0.045
ADHD/ADD	Asian non-Hispanic, < PL	Other, >= PL	0.004	0.003
ADHD/ADD	Hispanic, < PL	White non-Hispanic, >= PL	< 0.0005	< 0.0005
ADHD/ADD	Hispanic, < PL	Black non-Hispanic, >= PL	0.018	0.032
ADHD/ADD	Hispanic, < PL	Asian non-Hispanic, >= PL	< 0.0005	< 0.0005
ADHD/ADD	Hispanic, < PL	Hispanic, >= PL	0.283	0.243
ADHD/ADD	Hispanic, < PL	Other, >= PL	0.004	0.003
ADHD/ADD	Other, < PL	White non-Hispanic, >= PL	0.063	0.080
ADHD/ADD	Other, < PL	Black non-Hispanic, >= PL	0.011	0.015
ADHD/ADD	Other, < PL	Asian non-Hispanic, >= PL	< 0.0005	< 0.0005
ADHD/ADD	Other, < PL	Hispanic, >= PL	< 0.0005	< 0.0005
ADHD/ADD	Other, < PL	Other, >= PL	0.068	0.114
ADHD/ADD	Mexican, < PL	Mexican, >= PL	0.989	0.955
ADHD/ADD	Mexican, < PL	Puerto Rican, >= PL	0.015	0.018
ADHD/ADD	Puerto Rican, < PL	Mexican, >= PL	< 0.0005	< 0.0005
ADHD/ADD	Puerto Rican, < PL	Puerto Rican, >= PL	0.134	0.142
Learning disability	White non-Hispanic, < PL	White non-Hispanic, >= PL	< 0.0005	< 0.0005
Learning disability	White non-Hispanic, < PL	Black non-Hispanic, >= PL	< 0.0005	< 0.0005
Learning disability	White non-Hispanic, < PL	Asian non-Hispanic, >= PL	< 0.0005	< 0.0005
Learning disability	White non-Hispanic, < PL	Hispanic, >= PL	< 0.0005	< 0.0005
Learning disability	White non-Hispanic, < PL	Other, >= PL	< 0.0005	< 0.0005
Learning disability	Black non-Hispanic, < PL	White non-Hispanic, >= PL	0.001	0.001
Learning disability	Black non-Hispanic, < PL	Black non-Hispanic, >= PL	0.001	0.001
Learning disability	Black non-Hispanic, < PL	Asian non-Hispanic, >= PL	< 0.0005	< 0.0005
Learning disability	Black non-Hispanic, < PL	Hispanic, >= PL	< 0.0005	< 0.0005
Learning disability	Black non-Hispanic, < PL	Other, >= PL	0.030	0.033
Learning disability	Asian non-Hispanic, < PL	White non-Hispanic, >= PL	0.085	0.081
Learning disability	Asian non-Hispanic, < PL	Black non-Hispanic, >= PL	0.125	0.119
Learning disability	Asian non-Hispanic, < PL	Asian non-Hispanic, >= PL	0.175	0.209
Learning disability	Asian non-Hispanic, < PL	Hispanic, >= PL	0.238	0.216

			P-VALUES	
Variable	RACEINC1	RACEINC2	Unadjusted	Adjusted (for age, sex)
Learning disability	Asian non-Hispanic, < PL	Other, >= PL	0.160	0.138
Learning disability	Hispanic, < PL	White non-Hispanic, >= PL	0.906	0.722
Learning disability	Hispanic, < PL	Black non-Hispanic, >= PL	0.541	0.428
Learning disability	Hispanic, < PL	Asian non-Hispanic, >= PL	< 0.0005	< 0.0005
Learning disability	Hispanic, < PL	Hispanic, >= PL	0.029	0.023
Learning disability	Hispanic, < PL	Other, >= PL	0.637	0.637
Learning disability	Other, < PL	White non-Hispanic, >= PL	0.028	0.032
Learning disability	Other, < PL	Black non-Hispanic, >= PL	0.017	0.020
Learning disability	Other, < PL	Asian non-Hispanic, >= PL	< 0.0005	< 0.0005
Learning disability	Other, < PL	Hispanic, >= PL	0.002	0.003
Learning disability	Other, < PL	Other, >= PL	0.028	0.039
Learning disability	Mexican, < PL	Mexican, >= PL	0.591	0.539
Learning disability	Mexican, < PL	Puerto Rican, >= PL	0.263	0.293
Learning disability	Puerto Rican, < PL	Mexican, >= PL	< 0.0005	< 0.0005
Learning disability	Puerto Rican, < PL	Puerto Rican, >= PL	0.038	0.039
Autism	White non-Hispanic, < PL	White non-Hispanic, >= PL	0.307	0.303
Autism	White non-Hispanic, < PL	Black non-Hispanic, >= PL	0.049	0.056
Autism	White non-Hispanic, < PL	Asian non-Hispanic, >= PL	0.013	0.015
Autism	White non-Hispanic, < PL	Hispanic, >= PL	0.011	0.010
Autism	White non-Hispanic, < PL	Other, >= PL	0.996	0.973
Autism	Black non-Hispanic, < PL	White non-Hispanic, >= PL	0.128	0.119
Autism	Black non-Hispanic, < PL	Black non-Hispanic, >= PL	0.496	0.457
Autism	Black non-Hispanic, < PL	Asian non-Hispanic, >= PL	0.767	0.808
Autism	Black non-Hispanic, < PL	Hispanic, >= PL	0.688	0.675
Autism	Black non-Hispanic, < PL	Other, >= PL	0.117	0.114
Autism	Asian non-Hispanic, < PL	White non-Hispanic, >= PL	0.682	0.691
Autism	Asian non-Hispanic, < PL	Black non-Hispanic, >= PL	0.388	0.410
Autism	Asian non-Hispanic, < PL	Asian non-Hispanic, >= PL	0.154	0.165
Autism	Asian non-Hispanic, < PL	Hispanic, >= PL	0.284	0.288
Autism	Asian non-Hispanic, < PL	Other, >= PL	0.947	0.942
Autism	Hispanic, < PL	White non-Hispanic, >= PL	0.243	0.220
Autism	Hispanic, < PL	Black non-Hispanic, >= PL	0.814	0.750
Autism	Hispanic, < PL	Asian non-Hispanic, >= PL	0.425	0.462
Autism	Hispanic, < PL	Hispanic, >= PL	0.895	0.916
Autism	Hispanic, < PL	Other, >= PL	0.207	0.201
Autism	Other, < PL	White non-Hispanic, >= PL	0.610	0.695
Autism	Other, < PL	Black non-Hispanic, >= PL	0.347	0.423
Autism	Other, < PL	Asian non-Hispanic, >= PL	0.130	0.165
Autism	Other, < PL	Hispanic, >= PL	0.244	0.291
Autism	Other, < PL	Other, >= PL	0.881	0.952
Autism	Mexican, < PL	Mexican, >= PL	0.716	0.751
Autism	Mexican, < PL	Puerto Rican, >= PL	0.983	0.926
Autism	Puerto Rican, < PL	Mexican, >= PL	0.882	0.869
Autism	Puerto Rican, < PL	Puerto Rican, >= PL	0.936	0.928

			P-VALUES	
Variable	RACEINC1	RACEINC2	Unadjusted	Adjusted (for age, sex)
Intellectual disability	White non-Hispanic, < PL	White non-Hispanic, >= PL	0.036	0.035
Intellectual disability	White non-Hispanic, < PL	Black non-Hispanic, >= PL	0.443	0.439
Intellectual disability	White non-Hispanic, < PL	Asian non-Hispanic, >= PL	0.031	0.030
Intellectual disability	White non-Hispanic, < PL	Hispanic, >= PL	0.182	0.178
Intellectual disability	White non-Hispanic, < PL	Other, >= PL	0.247	0.245
Intellectual disability	Black non-Hispanic, < PL	White non-Hispanic, >= PL	0.005	0.006
Intellectual disability	Black non-Hispanic, < PL	Black non-Hispanic, >= PL	0.250	0.254
Intellectual disability	Black non-Hispanic, < PL	Asian non-Hispanic, >= PL	0.015	0.016
Intellectual disability	Black non-Hispanic, < PL	Hispanic, >= PL	0.055	0.055
Intellectual disability	Black non-Hispanic, < PL	Other, >= PL	0.156	0.157
Intellectual disability	Asian non-Hispanic, < PL	White non-Hispanic, >= PL	0.055	0.056
Intellectual disability	Asian non-Hispanic, < PL	Black non-Hispanic, >= PL	0.228	0.232
Intellectual disability	Asian non-Hispanic, < PL	Asian non-Hispanic, >= PL	0.036	0.038
Intellectual disability	Asian non-Hispanic, < PL	Hispanic, >= PL	0.112	0.114
Intellectual disability	Asian non-Hispanic, < PL	Other, >= PL	0.139	0.142
Intellectual disability	Hispanic, < PL	White non-Hispanic, >= PL	0.010	0.010
Intellectual disability	Hispanic, < PL	Black non-Hispanic, >= PL	0.312	0.315
Intellectual disability	Hispanic, < PL	Asian non-Hispanic, >= PL	0.022	0.022
Intellectual disability	Hispanic, < PL	Hispanic, >= PL	0.091	0.092
Intellectual disability	Hispanic, < PL	Other, >= PL	0.199	0.201
Intellectual disability	Other, < PL	White non-Hispanic, >= PL	0.271	0.286
Intellectual disability	Other, < PL	Black non-Hispanic, >= PL	0.676	0.698
Intellectual disability	Other, < PL	Asian non-Hispanic, >= PL	0.160	0.172
Intellectual disability	Other, < PL	Hispanic, >= PL	0.472	0.490
Intellectual disability	Other, < PL	Other, >= PL	0.423	0.437
Intellectual disability	Mexican, < PL	Mexican, >= PL	0.243	0.245
Intellectual disability	Mexican, < PL	Puerto Rican, >= PL	0.859	0.852
Intellectual disability	Puerto Rican, < PL	Mexican, >= PL	0.053	0.054
Intellectual disability	Puerto Rican, < PL	Puerto Rican, >= PL	0.758	0.762

Table 3. Other statistical significance tests comparing the percentages of children ages 5 to 17 years with neurodevelopmental disorders, for 2005-2008 (trends for 1997-2008).

					P-VALUES	
Variable	From	To	Against	Subset	Unadjusted	Adjusted*
ADHD/ADD	2005	2008	age		< 0.0005	< 0.0005
ADHD/ADD	2005	2008	sex		< 0.0005	< 0.0005
ADHD/ADD	2005	2008	income		0.005	< 0.0005
ADHD/ADD	1997	2008	year		< 0.0005	< 0.0005
ADHD/ADD	1997	2008	year	Males		< 0.0005
ADHD/ADD	1997	2008	year	Females		< 0.0005
ADHD/ADD	1997	2008	year	5-10 years		0.097
ADHD/ADD	1997	2008	year	11-17 years		< 0.0005
ADHD/ADD	1997	2008	year	Below Poverty Level		0.032

					P-VALUES	
Variable	**From**	**To**	**Against**	**Subset**	**Unadjusted**	**Adjusted***
ADHD/ADD	1997	2008	year	At or Above Poverty Level		< 0.0005
Learning disability	2005	2008	age		< 0.0005	< 0.0005
Learning disability	2005	2008	sex		< 0.0005	< 0.0005
Learning disability	2005	2008	income		< 0.0005	< 0.0005
Learning disability	1997	2008	year		0.652	0.048
Learning disability	1997	2008	year	Males		0.018
Learning disability	1997	2008	year	Females		0.902
Learning disability	1997	2008	year	5-10 years		0.445
Learning disability	1997	2008	year	11-17 years		0.051
Learning disability	1997	2008	year	Below Poverty Level		0.520
Learning disability	1997	2008	year	At or Above Poverty Level		0.060
Autism	2005	2008	age		0.002	0.002
Autism	2005	2008	sex		< 0.0005	< 0.0005
Autism	2005	2008	income		0.917	0.549
Autism	1997	2008	year		< 0.0005	< 0.0005
Autism	1997	2008	year	Males		< 0.0005
Autism	1997	2008	year	Females		< 0.0005
Autism	1997	2008	year	5-10 years		< 0.0005
Autism	1997	2008	year	11-17 years		< 0.0005
Autism	1997	2008	year	Below Poverty Level		0.102
Autism	1997	2008	year	At or Above Poverty Level		< 0.0005
Intellectual disability	2005	2008	age		0.770	0.883
Intellectual disability	2005	2008	sex		0.266	0.266
Intellectual disability	2005	2008	income		< 0.0005	0.002
Intellectual disability	1997	2008	year		0.898	0.152
Intellectual disability	1997	2008	year	Males		0.337
Intellectual disability	1997	2008	year	Females		0.357
Intellectual disability	1997	2008	year	5-10 years		0.394
Intellectual disability	1997	2008	year	11-17 years		0.007
Intellectual disability	1997	2008	year	Below Poverty Level		0.589
Intellectual disability	1997	2008	year	At or Above Poverty Level		0.184

*For AGAINST = ”age,” the p-values are adjusted for sex, race/ethnicity, and income.
For AGAINST = ”sex,” the p-values are adjusted for age, race/ethnicity, and income.
For AGAINST = ”income,” the p-values are adjusted for age, sex, and race/ethnicity.
For AGAINST = “year,” the p-values are adjusted for age, sex, race/ethnicity, and income.