### QUICK FACTS

Age, genetics, medical health, socioeconomic status, behaviors, health care, and environmental exposures all affect the ability to conceive, carry, and deliver a healthy full-term baby.<sup>1,2,3</sup>

The role of environmental exposures in reproductive and infant health is complex and not largely understood.

No national database to track all birth defects exists. Currently, over 40 states have populationbased birth defects surveillance systems and participate in the National Birth Defects Prevention Network, a nonprofit organization that collects, analyzes, and disseminates birth defect surveillance data.<sup>4</sup> Of the 6 million pregnancies in the United States each year, approximately 2.2 million end with miscarriage or stillbirth or are voluntarily terminated; about a half million babies are born prematurely; and about 120,000 babies have birth defects.

### INTRODUCTION

More than three million healthy babies are born annually in the United States. The parents' age, genetics, medical health, socioeconomic status, behaviors, access to health care, and environmental exposures all affect their ability to conceive, carry, and deliver a healthy full-term baby. Unfortunately, not every woman can become pregnant; not every woman who becomes pregnant can carry her baby to full term; and not every baby is born healthy. Identification of the specific risk factors involved is needed to develop prevention and intervention strategies to prevent adverse reproductive outcomes.

Our understanding of what causes adverse reproductive outcomes has increased greatly over the past decades. However, there is still much we do not know. In particular, the role of environmental exposures in reproductive and infant health is complex and largely not understood. This chapter summarizes our current understanding of environmental risk factors for infertility, adverse birth outcomes, birth defects, and developmental disabilities. We look specifically at the following:

- Infertility
- Premature (or preterm) births
- Low birth weight
- Fetal and infant death and miscarriage
- Birth defects
- Low IQ and attention deficit/ hyperactivity disorder (ADHD) diagnoses

Preterm birth and low birthweight are associated with compromised health and developmental disabilities, and ADHD diagnoses are associated with learning disabilities.

### INFERTILITY

Not all couples can bear children, because of infertility or impaired fecundity. Infertility is the inability of a couple to conceive a child. Impaired fecundity encompasses problems conceiving and also problems carrying a pregnancy to term. Each of these conditions contributes to lower birth rates and a lower total fertility rate. For the purposes of this discussion, infertility is used more broadly to refer to all fertility impairments.

The fertility of an individual male or female is affected by many factors, including the following:

- Age
- Genetics
- Nutrition
- Behavior
- Infections of the reproductive tract
- Stress
- Some medications

Fertility may also be affected by environmental exposures, although the roles they play in altering fertility are complex, relatively unexplored, and mostly not understood. Even when we know the immediate reason for infertility (low sperm count, endometriosis, etc.), the underlying causes are often unknown. Both nonenvironmental and environmental factors are implicated.



### PICTURE OF AMERICA REPORT

# WHAT ARE THE RISK FACTORS FOR INFERTILITY?

NON-ENVIRONMENTAL RISK FACTORS Cancer of the reproductive organs; endometriosis; sexually transmitted diseases, such as gonorrhea and chlamydia; and other diseases involving the reproductive organs or hormones are known to increase the risk of infertility.6,7 Physical anomalies of the reproductive organs may also interfere with fertility. Although these conditions are generally considered nonenvironmental risk factors, environmental exposures could be responsible for causing some of the diseases and anatomical variations. Maternal or paternal alcohol consumption and smoking increase the risk of infertility<sup>8,9</sup> as do some medicines and drugs, poor diet, athletic training, and being overweight or underweight.<sup>1,10</sup> Many comorbidities are also associated with infertility, such as obesity<sup>11</sup>, epilepsy<sup>12</sup>, injury<sup>13,14</sup>, eating disorders<sup>15</sup>, and cancer.<sup>16</sup>

### ENVIRONMENTAL RISK FACTORS

Numerous chemicals found in the environment are suspected to be associated with health conditions known to, or which have the potential to, cause infertility (Table 1). Studies conducted mostly on animals have shown that exposure to high levels of pesticides, phthalates, bisphenol A, dioxins, PCBs, heavy metals, or various organochlorine or polybrominated chemicals is associated with health problems causing, or sometimes co-occurring with, infertility. These problems may include cancer or physical anomalies of the reproductive organs, reduced sperm counts and quality, and alterations in the female menstrual cycle, among other problems.<sup>17-46</sup>

### HOW ARE WE TRACKING INFERTILITY?

No national surveillance system exists to track infertility, although the National Survey of Family Growth collects information on the prevalence of infertility from a representative sample of the U.S. population. This survey has historically addressed infertility only in women, but in 2002, the survey began gathering data on infertility in men.<sup>48</sup>

Table 1. Chemical exposures during adulthood and fertility-related effects (Adapted from Luoma, 2005<sup>36</sup>)

	Bisphenol A	Chlorinated hydrocarbons	Dioxins/ furans	Heavy Metals*	Organic Solvents**	PCBs	Pesticides	Phthalates
Abnormal sperm or decreased semen quality								
Endometriosis								
Menstrual, estrous, and/or ovulatory irregularities								
Miscarriage or fetal loss								
Reduced fertility								
Chromosomal abnormalities/ changes								
Hormonal changes								

\*Mercury, manganese, and cadmium

\*\* Benzene, toluene, xylene, perchloroethylene, and others

## STATUS AND TRENDS FOR INFERTILITY

The National Center for Health Statistics reports the prevalence of impaired fecundity has been increasing. In 2002, nearly 12% (7.3 million) of women aged 15–44 years reported impaired fecundity, representing a 2% increase from 1988 and 1995 levels.<sup>49,50</sup> However, some of the increase may be attributable to aging of the population, coupled with intentionally delayed childbearing (aging reduces fertility). The number of visits to doctors for infertility concerns is also rising, although these statistics may be influenced by greater availability of infertility services, new treatment technologies, demographics of the U.S. population, and couples delaying childbearing.<sup>43,45,51,52</sup>

## ADDITIONAL RESOURCES

 MedlinePlus (from the U.S. National Library of Medicine) at www.nlm.nih.gov/medlineplus/ infertility.html

- Womenshealth.gov (from the U.S. Department of Health & Human Services) at www.womenshealth.gov
- Report titled "Challenged Conceptions: Environmental Chemicals and Fertility" at www.prhe.ucsf.edu/prhe/events/ Challenged\_Conceptions.pdf
- The Collaborative on Health and the Environment's toxicant and disease database for reduced fertility Web site at www.database.healthandenvironment. org/index.cfm?id=758

## WHAT ARE ADVERSE BIRTH OUTCOMES?

Most newborns weigh 7 to 10 pounds and are born after a pregnancy of approximately 40 weeks. However, about 1 in 12 babies is underweight (less than 5.5 pounds) at birth, and approximately 1 in 8 newborns is delivered prematurely (less than 37 weeks).<sup>53</sup> A small but significant number of babies die before delivery or are born live but die before their first birthday. Although fewer pregnancies now end in fetal or infant death compared with the death rate 20 years ago, a higher percentage of births are premature, and more newborns are below the optimum survival weight.

The causes for most of these adverse birth outcomes are not well understood but most likely involve the co-occurrence of multiple factors from many areas of a woman's life. Infant mortality is closely associated with preterm (premature) birth and low birthweight. Preterm birth is the most frequent cause of infant mortality, accounting for over one third of infant deaths. The declining trends in infant death have generally been related to advances in medical care, which increase survival of preterm, low birthweight infants. Although improvements in medicine have reduced death and disability among infants born too early and too small, preterm infants remain at increased risk of illness, developmental disabilities, neurological disorders, and other chronic health conditions requiring increased levels of long-term medical care, parental care, and special education services.<sup>1</sup>

# WHAT ARE THE RISK FACTORS FOR ADVERSE BIRTH OUTCOMES?

### NON-ENVIRONMENTAL RISK FACTORS

There are many well-known nonenvironmental risk factors for adverse birth outcomes, such as preterm birth and low birthweight.<sup>1,2,3</sup> Known risk factors include some maternal and paternal demographic factors as well as biological factors, such as genetics, prepregnancy obesity, or infections during pregnancy. Women who had a previous pregnancy with a poor

birth outcome have an increased risk for a subsequent poor birth outcome. Maternal smoking and substance abuse increase the risk of low birthweight and preterm birth. Social, economic, and neighborhood factors are also associated with adverse birth outcomes.

### ENVIRONMENTAL RISK FACTORS

Exposure of nonsmoking pregnant women to environmental tobacco smoke has been documented as a risk factor for preterm birth, low birthweight, and possibly miscarriage.<sup>55–64</sup> Evidence also supports a link between maternal exposure to components of air pollution (carbon monoxide, particulate matter, nitrogen dioxide) with both low birthweight and preterm birth, even at levels below the Environmental Protection Agency's (EPA) National Ambient Air Quality Standards (see Outdoor Air Quality module).<sup>56–74</sup>

Maternal exposure to lead is associated with preterm birth. Other adverse birth outcomes that may be linked with maternal or paternal lead exposure include low birthweight and spontaneous fetal loss.<sup>2,56,75-80</sup> High dose exposure levels to pesticides are also implicated for a range of adverse birth outcomes, including fetal death, spontaneous fetal loss, and slowed fetal development (small for gestational age). Although the evidence is limited at this time, it suggests limiting or completely avoiding maternal exposure to pesticides during pregnancy. (*see Poisonings module*).<sup>23,56,57,58,72,81-85</sup>

Measures of pregnancy and birth outcomes	Common abbreviation	Definition	
Preterm birth	РТВ	Birth at less than 37 completed weeks of gestation	
Very preterm birth	VPTB	Birth at less than 32 completed weeks of gestation	
Low birth weight	LBW	Weighing less than 2,500 grams (~5.5 pounds) at delivery	
Very low birth weight	VLBW	Weighing less than 1,500 grams (~3.3 pounds) at delivery	
Infant mortality	IM	Death of a live-born infant before the first birthday	
Fetal death (stillbirth)	FD	Death of a fetus after the 20 <sup>th</sup> week of gestation	
Spontaneous fetal losses	SFL	Recognized pregnancies that do not result in induced abortions or live births; includes miscarriages, ectopic (tubal) pregnancies, and FD	

#### Table 3. Measures of birth and pregnancy outcomes

Definitions for PTB, VPTB, LBW, VLBW from Martin JA, et al. 2009.75



**Figure 1**. Trends in infant mortality rates<sup>a,b</sup> in the United States, by race and sex<sup>79</sup>

<sup>a</sup> Race was reported based in the race of the child (1940-1979) or the race of the mother (1980-2006)

<sup>b</sup> Annual infant mortality rates are not available prior to 1975 in published sources. Trends presented form 1940-1974 are based on data published for 1940, 1950, 1960, and 1970.

## HOW ARE WE TRACKING ADVERSE BIRTH OUTCOMES?

All 50 states record vital statistics on state birth and death certificates, which include birth weight, gestational age, the age at death, and reports of fetal death, plus additional information. This data is gathered by CDC's National Center for Health Statistics to track the outcomes in Table 3 as well as numerous other measures.

## STATUS AND TRENDS FOR BIRTH OUTCOMES

Measures of mortality (IM, FD, and SFL) have Measures of mortality (IM (Figure 1), FD, and SFL) have shown a decreasing trend over the past few decades.<sup>89</sup> Rates of preterm birth fell for the fifth straight year in 2011 and are 8% lower than the peak in 2006. Low birthweight has also decreased but has declined more slowly, at a rate of 2% from 2006 to 2011(Figure 2). Being born preterm increases the risk of infant mortality and the likelihood of poor health outcomes.<sup>2</sup> Low birthweight is also a risk factor for infant survival, and the birthweight distribution in the United States over the last 15 years has shifted markedly toward lower weights. The shift toward lower birthweights might be related in part to increased rates of multiple births (twins, triplets, etc.); older

maternal age at childbearing; obstetric interventions, such as cesarean delivery and induction of labor; maternal chronic disease; and increased use of fertility therapies.<sup>86</sup>

Disparities among races are particularly noteworthy. Although the preterm birth rate declined by 8-9% for non-Hispanic black infants, the lowest reported in three decades, Non-Hispanic black women have the highest rates of poor birth outcomes, substantially higher than the rates for non-Hispanic white or Hispanic women.<sup>86</sup> The black and white disparity in very short gestation infants has been linked to the black and white infant mortality gap.<sup>90</sup> According to the Institute of Medicine, racial disparities in preterm birth rates cannot be fully explained by socioeconomic differences or by differences in maternal behaviors, such as smoking or drug use.<sup>2</sup>

## ADDITIONAL RESOURCES

- CDC's Web site titled "Maternal and Infant Health Research" at www.cdc.gov/reproductivehealth/ MaternalInfantHealth/index.htm
- The National Institutes of Health offers a Web site on preterm labor and birth at www.nichd.nih.gov/ health/topics/Preterm\_Labor\_and\_Birth.cfm
- The March of Dimes "Pregnancy " Web site at www.marchofdimes.com/pnhec/pnhec.asp



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Figure 2. Preterm birth and low birth weight trends in the United States (Data from Martin JA, et al<sup>75</sup>)

### WHAT ARE BIRTH DEFECTS?

A birth defect is a structural, functional, or developmental abnormality that originates during pregnancy, primarily during the first three months when fetal organs are forming. Some birth defects can be detected during pregnancy, but others are not apparent until birth. For example, Down syndrome is often diagnosed during pregnancy, but cleft palate is normally first noticed at birth. Some birth defects do not show up until the person reaches reproductive age or unless specialized testing identifies the anomaly. For example, women whose mothers were given diethylstilbestrol (DES), a synthetic form of estrogen, during pregnancy may be born with abnormalities to their reproductive tract. These abnormalities are often not discovered until the women attempt to become pregnant themselves.<sup>91</sup> DES is a drug once prescribed during pregnancy to prevent miscarriages or premature deliveries. In the United States, an estimated 5 to 10 million persons were exposed to DES from 1938 to 1971, including pregnant women prescribed DES and their children. In 1971, the Food and Drug Administration (FDA) advised physicians to stop prescribing DES because it was linked to a rare vaginal cancer.<sup>91</sup> In addition, some birth defects, such as heart defects, are only discovered through specialized tests, such as echocardiograms, CT scans or X-rays.<sup>92</sup>

Birth defects are a large public health problem and are estimated to affect over 120,000 children in the United States every year.93 Approximately one of every 33 infants is born with a birth defect, which may range from mild to severe.<sup>94</sup> Birth defects are a leading cause of infant death; they account for more than 20% of all infant deaths.<sup>95</sup> A birth defect can affect almost any organ system of the body and might be lethal. Some common birth defects are not life-threatening but require medical attention. Others, such as undescended testicles, usually resolve within the first year of life without medical intervention. Infants born with multiple birth defects affecting different organ systems generally require extensive and frequent medical care, and their morbidity and mortality are higher than for infants with a single birth defect.<sup>96</sup>

Most birth defects are thought to be caused by the complex interaction of genetics, environmental exposures, and behavior, although how these factors interact is unknown. The amount of the exposure and its timing during the development of the embryo are important variables governing the risk of birth defects from environmental and behavioral exposures.<sup>97,98</sup>

## WHAT ARE THE RISK FACTORS FOR BIRTH DEFECTS?

NON-ENVIRONMENTAL RISK FACTORS Birth defects are associated with maternal characteristics and exposures including

- Alcohol use
- Smoking
- Diabetes
- Prepregnancy obesity
- Poor nutrition
- Infections
- Drugs and medications
- Radiation

Fetal alcohol syndrome, which results from maternal intake of alcohol during pregnancy, is 100% preventable if a woman does not drink alcohol while she is pregnant.<sup>99</sup> Maternal smoking during pregnancy has been linked to a range of birth defects, including cleft lip and palate,<sup>100</sup> clubfoot,<sup>101</sup> and some types of heart defects.<sup>102,103</sup> Limited evidence also suggests that paternal smoking may be associated with birth defects.<sup>104</sup>

**Figure 3.** Estimated prevalence of selected birth defects in the United States, 1999-2001 and 2004-2006, per 10,000 live births.<sup>138,140</sup>



# REPRODUCTIVE OUTCOMES

Maternal diabetes is a well-established risk factor for birth defects, causing malformations in most organs. Maternal prepregnancy obesity has been associated with increased risk for neural tube defects and congenital heart defects.<sup>105-107</sup>

In addition, maternal deficiency in folic acid (a B vitamin) is a recognized risk factor for neural tube birth defects. Maternal infections, such as rubella (German measles), cytomegalovirus (CMV), syphilis, and toxoplasmosis<sup>96,108</sup> are known risk factors for a broad range of birth defects, specifically heart defects.<sup>96,107,109–112</sup> Maternal exposure to certain medications, such as thalidomide, isotretinoin, and valproic acid, are known to cause certain birth defects.<sup>113,114</sup>

Mothers who experience prolonged fever early in pregnancy have a higher risk of having a pregnancy affected by spina bifida or anencephaly.<sup>115–118</sup> The higher risk may be attributable to the underlying cause of the fever or illness. Some research suggests that maternal hyperthermia from use of saunas, hot tubs, and tanning beds may be associated with neural tube defects.<sup>114,119</sup>

### ENVIRONMENTAL RISK FACTORS

Living near a hazardous waste site has been associated with a range of birth defects, including neural tube defects (e.g., spina bifida), cleft lip or palate, gastroschisis, hypospadias, chromosomal congenital anomalies (e.g., Down syndrome), and some heart and vasculature defects.<sup>120-126</sup> Numerous studies also document links between various endocrine disruptors (e.g., PCBs, dioxins, and pesticides) and birth defects. Maternal or paternal exposure to pesticides in populations occupationally exposed to pesticides (e.g., agricultural operations, pesticides applicators) has been associated with an increased risk of birth defects, such as cleft lip or palate, heart defects, nervous system defects, and eye anomalies (see Poisonings module).<sup>23,35,84</sup> There are critical periods during fetal development when exposure to pesticides may be more harmful. There is ever-increasing scientific evidence showing that conceptions in the spring are associated with more birth defects than any other season.<sup>35,84</sup> Exposure to water disinfection by-products in drinking water may increase the risk of some types of birth defects, especially neural tube defects (see drinking Water *Quality module*).<sup>127,128</sup> Maternal exposure to some air pollutants may also increase the risk of some types of birth defects, though evidence has been inconsistent.<sup>129–134</sup> For example, maternal exposure to benzene

may increase the risk for spina bifida, and exposure to high levels of particulate matter may increase the risk for certain heart defects.<sup>130,133</sup>

## HOW ARE WE TRACKING BIRTH DEFECTS?

There is no national database for tracking all birth defects. Birth defects are tracked through statebased surveillance systems, but not all states collect birth defects data. In the most recent report from the National Birth Defects Prevention Network (NBDPN), a nonprofit organization of state and other population-based birth defects surveillance programs, data from 41 birth defects surveillance programs were published.<sup>4</sup> The NBDPN requests prevalence data annually on up to 45 major birth defects. Data collection methods differ among the states that do track birth defects, making state-tostate comparisons and national estimates difficult. Specifically, birth defect definitions, data sources, and calculation methods vary significantly from state to state.<sup>135–138</sup> The Environmental Public Health Tracking Program collects birth defects data on 12 major birth defects from 15 states.<sup>139</sup>

# STATUS AND TRENDS FOR BIRTH DEFECTS

In 2006, national prevalence estimates for 21 birth defects were published based on combined data from 1999 to 2001 from eleven states with active birth defects surveillance systems (Alabama, Arkansas, California, Georgia, Hawaii, Iowa, Massachusetts, North Carolina, Oklahoma, Texas and Utah) to calculate (Figure 3).<sup>140</sup> In 2010, eleven states with active birth defects surveillance systems (Arkansas, Arizona, California, Georgia, Iowa, Massachusetts, North Carolina, Oklahoma, Puerto Rico, Texas, and Utah) combined data from 2004 to 2006 to update the previous national prevalence estimates (Figure 3).<sup>138</sup> Prevalence estimates for the total U.S. population were obtained by adjusting the pooled state data for the racial and ethnic distribution of U.S. live births. Down syndrome and cleft lip with or without cleft palate occur more frequently than the other defects reported. Although changes in the prevalence of selected defects were noted by the authors (e.g., the prevalence of transposition of great arteries decreased

from 1999–2001 to 2004–2006 while the prevalence of gastroschisis increased slightly during the same time period), any deter mination of a trend with only two data points is neither accurate nor reliable.

## ADDITIONAL RESOURCES

- The CDC Birth Defects Web page at www.cdc. gov/ncbddd/birthdefects/index.html
- The Collaborative on Health and the Environment Web page on birth defects and the environment at www.healthandenvironment.org/birth\_ defects/peer\_reviewed
- The MotherToBaby Web site on various exposures of concern, including information about exposure during pregnancy and the possibility of birth defects at http://www.mothertobaby.org/ otis-fact-sheets-s13037

## INTELLECTUAL DISABILITIES AND ATTENTION-DEFICIT/HYPERACTIVITY DISORDER (ADHD)

Intellectual disability is characterized both by a significantly below average score on a test of mental ability or intelligence and by limitations in the ability to function in areas of daily life, such as communication, self-care, and getting along in social situations and school activities. Intellectual disability can start any time before a child reaches the age of 18 years and may range in severity from mild to profound. Although there are many recognized genetic and environmental causes of intellectual disability, they collectively account for less than half of all cases, and therefore, for a great proportion of children, the cause of their intellectual disability is unknown.<sup>141</sup>

Attention-deficit/hyperactivity disorder (ADHD) is a developmental disability characterized by pervasive inattention and hyperactivity and impulsivity that often results in functional impairment. It is currently the most frequently diagnosed cognitive and behavioral disorder among school children. Current research suggests that children with an intellectual disability are at increased risk for ADHD. Although genes are believed to play an important role in the etiology of ADHD, environmental exposures and gene-environment interactions are strongly implicated as well.

## WHAT ARE THE RISK FACTORS FOR INTELLECTUAL DISABILITIES AND ADHD?

### NON-ENVIRONMENTAL RISK FACTORS

### Intellectual Disabilities

Genetic conditions, such as Down syndrome, account for up to two thirds of all genetic causes of intellectual disability.<sup>145–149</sup> Other significant nongenetic risk factors include chronic maternal alcohol use during pregnancy, maternal infections, nutritional deficiencies, maternal use of certain medications, and various perinatal outcomes, such as low birthweight, preterm birth, and asphyxia. Postnatal factors associated with intellectual disability include acquired infections and injuries to the head.<sup>150,151</sup>



### ADHD

Family studies have confirmed that genetic risk factors contribute to ADHD, but other risk factors also play a role. Low birthweight is a known risk factor for ADHD, and other pregnancy and delivery complications have been implicated as well. Numerous studies of fetal exposure to alcohol and maternal smoking suggest that these factors increase the risk of ADHD or ADHD-like neurobehavioral deficits<sup>143,152</sup> although some studies have found no increased risk.<sup>152-155</sup>

### ENVIRONMENTAL RISK FACTORS

REPRODUCTIVE

### Intellectual Disabilities

Childhood exposure to lead is a well-documented risk factor for intellectual disability. More recent studies have shown an association between maternal exposure during pregnancy to high levels of polychlorinated biphenyls (PCBs) and methylmercury and intellectual disability in infants and young children.<sup>156–164</sup> High blood lead levels are an irreversible yet preventable cause of intellectual disability. When pregnant women were exposed to high levels of PCBs, mainly through consumption of PCB-contaminated fish, intellectual disability occurred in their offspring.<sup>157,158,165-168</sup> Some studies have shown that prenatal environmental exposure to high levels of methylmercury has also been associated with intellectual disability.<sup>169–174</sup> In the U.S. population, mercury levels are well below those associated with the most subtle neurodevelopmental effects.

### ADHD

Exposures to lead and mercury may be risk factors for ADHD. Many studies have reported correlations between lead exposure and ADHD or behaviors common in ADHD (inattentiveness, hyperactivity, disorganization).<sup>175-178</sup> A 2006 study noted a significant association between ADHD and blood lead levels as low as  $2-5 \mu g/L$ .<sup>152</sup> Mercury is known to be a potent neurodevelopmental toxicant and has been linked with ADHD in children.<sup>179</sup>

## HOW ARE WE TRACKING INTELLECTUAL DISABILITIES AND ADHD?

### Intellectual Disabilities

Overall, surveillance for developmental disabilities is limited. In the United States, the two primary methods for ID surveillance have been nationally representative surveys based on parental report and multiple source administrative record review. The National Health Interview Survey (NHIS), conducted by CDC's National Center for Health Statistics, asks individuals from a representative sample of the U.S. population about various health conditions. The survey does include data on intellectual disability. However, these data have been shown to underestimate the prevalence of intellectual disability.<sup>180</sup>

One of the few programs in the world that tracks intellectual disability in a large, diverse population is CDC's Metropolitan Atlanta Developmental Disabilities Surveillance Program (MADDSP).<sup>181</sup> MADDSP is an active, population-based surveillance system which monitors the number of school-aged children living in a five-county metropolitan area with one or more of five conditions, including intellectual disability. The MADDSP methods have been replicated by a select number of participating sites in the Autism and Developmental Disabilities Monitoring (ADDM) Network. Currently for the 2010 surveillance year, 6 ADDM sites, including MADDSP, are conducting surveillance of intellectual disability. These data will help us determine a more accurate estimate of the prevalence of intellectual disability.

#### ADHD

ADHD is not tracked on a national scale, but is included in the NHIS. These data are comparable with other survey data on ADHD and are used to estimate the national prevalence of ADHD among children.<sup>180,182</sup>

### STATUS AND TRENDS FOR INTELLECTUAL DISABILITIES AND ADHD

#### Intellectual Disabilities

Prevalence estimates for intellectual disability generated from epidemiologic studies dating back to the early 1960s vary from 2 to 79 per 1,000.<sup>183</sup> Since the early 1980s, studies in developed countries have reported a slightly more narrow range, from 5 to 42 per 1,000. Much of the range across studies is attributable to differences in methods used across studies.

Intellectual disability prevalence varies by severity and social and demographic characteristics, most notably age, gender, race and ethnicity, and socioeconomic status.<sup>141</sup> For monitoring purposes, the prevalence of intellectual disability is commonly examined using two IQ severity levels, with an IQ 50–55 to 70–75 being considered a mild intellectual disability (MID) and an IQ<50–55 being considered a moderate to profound intellectual disability (MPID). Most studies have shown that the majority of individuals have MID (75%–80%). Since the 1980s, estimates of MID prevalence have shown great variability ranging from

3.0 to 37.4 per 1,000 in developed countries with much less variability for MPID prevalence ranging from 1.4 to 4.5 per 1,000 in developed countries.

The prevalence of intellectual disability appears to peak at ages 10 to 14 years, declines slightly among adolescents, and declines markedly thereafter.<sup>184</sup> The estimated prevalence of intellectual disability for children 4 years old or younger is 1 per 1,000, whereas the estimate for children 10 to14 years of age is 97 per 1,000.183,185,186 A factor contributing to the decline in prevalence into adulthood may be the reported higher mortality rates for persons with intellectual disability.<sup>187,188</sup> Most studies report a higher prevalence of intellectual disability among males compared with females, with the male to female ratio overall being approximately 1.5:1.<sup>189–191</sup> The higher proportion of intellectual disability among males may be driven, in part, by X-linked genetic conditions, such as Fragile X syndrome.<sup>192,193</sup>

Some studies have reported a higher prevalence of mild intellectual disability in individuals with a lower socioeconomic status. This higher prevalence may be due in part to poor living conditions, suboptimal obstetric care, and parental occupations with exposure to chemical agents.<sup>183,194</sup> In addition, MADDSP found that in 1996 and 2000 the prevalence of intellectual disability was higher in black children than in white children.<sup>181</sup>

The overall prevalence of ID appears to have been relatively stable over time which may be due to a balance between decreased numbers of pregnancies with chromosomal disorders associated with severe to profound ID (through elective termination from prenatal detection) and improved survival of preterm and medically compromised infants who otherwise would not have survived.<sup>195</sup> Ongoing population-based monitoring of ID is necessary to examine changes in underlying risk factors and prevalence among subgroups of the population.

#### ADHD

Analysis of NHIS data for 1997–2008 shows a steady increase in ADHD from a prevalence of 5.69 in 1997–1999 to 7.57 in 2006–2008 among

youth aged 3-17.<sup>180</sup> A previous analysis of NHIS data for 2004–2006 indicates that about 8.4% of youth 6 to17 years of age have been diagnosed with ADHD.<sup>196</sup> The percentage has increased gradually since 1997 (Figure 4). A CDC analysis of earlier data from the 2003 National Survey of Children's Health showed that the national prevalence of reported ADHD diagnoses among children 4 to17 years of age was 7.8%.<sup>142</sup> Other estimates of ADHD prevalence between 1998 to 2001 based on different populations, types of data, and analyses have suggested rates ranging from 2% to18%.<sup>197</sup> Invariably, the data show that ADHD is much more prevalent among boys than girls (Figure 5). Racial disparities are not pronounced, although CDC's analysis showed that the ADHD prevalence rate among non-Hispanics is about double the rate among Hispanics.

## ADDITIONAL RESOURCES

- National Dissemination Center for Children with Disabilities (NICHCY) Web site at www.nichcy. org/index.html
- MedlinePlus (from the National Library of Medicine) Web site on Developmental Disabilities at www.nlm.nih.gov/medlineplus/ developmentaldisabilities.html
- The CDC Web pages on Intellectual Disability and on ADHD at www.cdc.gov/ncbddd/dd/ddmr.htm and http://www.cdc.gov/ncbddd/adhd/



NOTES: ADHD is attention deficit hyperactivity disorder. "All diagnoses of ADHD" includes ADHD with and without LD. "All diagnoses of LD" includes LD with and without ADHD. SOURCE: COCNCHS, National Health Interview Survey, 1997-2006.

**Figure 4.** Estimated prevalence of selected birth defects in the United States, 1999–2001 (Adapted from Canfield, et al.<sup>120</sup>)



Figure 5. Trends in diagnosed ADHD among children 6–17 years of age, by sex: United States, 1997–2006<sup>182,198</sup>

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