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Pertussis Epidemic — Washington, 2012

Since mid-2011, a substantial rise in pertussis cases has been reported in the state of Washington. In response to this increase, the Washington State Secretary of Health declared a pertussis epidemic on April 3, 2012. By June 16, the reported number of cases in Washington in 2012 had reached 2,520 (37.5 cases per 100,000 residents), a 1,300% increase compared with the same period in 2011 and the highest number of cases reported in any year since 1942. To assess clinical, epidemiologic, and laboratory factors associated with this increase, all pertussis cases reported during January 1–June 16, 2012, were reviewed. Consistent with national trends, high rates of pertussis were observed among infants aged <1 year and children aged 10 years. However, the incidence in adolescents aged 13–14 years also was increased, despite high rates of vaccination with tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis (Tdap) vaccine, suggesting early waning of immunity. The focus of prevention and control efforts is the protection of infants and others at greatest risk for severe disease and improving vaccination coverage in adolescents and adults, especially those who are pregnant. Pertussis vaccination remains the single most effective strategy for prevention of infection.

Case Classification and Clinical Characteristics

For this analysis, all cases of pertussis reported to the Washington State Department of Health during January 1–June 16, 2012, were reviewed. Cases were classified according to the Council of State and Territorial Epidemiologists case definition (*1*). Cumulative incidence was calculated as the number of confirmed and probable cases reported per 100,000 residents using age, race, and county-specific population figures from the U.S. Census Bureau as of June 16, 2012. Confirmed and probable cases reported in Washington were compared with U.S. data in the National Notifiable Diseases Surveillance System from January 1 through June 14, 2012.

During January 1–June 16, 2012, a total of 2,520 pertussis cases were reported in Washington, of which 2,069 were confirmed (83.4% laboratory-confirmed and 16.6% epidemiologically linked) and 451 were probable. In comparison, 180 of 966 total cases for the year had been reported during the same period in 2011 (Figure 1). Cases were reported in 32 of the 39 counties (median: 24 cases; range: 1–485 cases). Statewide incidence was 37.5 cases per 100,000 population, ranging from 4.9 to 414.9 by county. Incidence was highest in infants aged <1 year and children aged 10, 13, and 14 years (Figure 2). Among the 1,867 cases with known race and ethnicity, statewide cumulative incidence was higher in Hispanics than non-Hispanics (53.1 versus 24.6 cases per 100,000 population). Of the 155 reported pertussis cases in infants aged <1 year, 34 (21.9%) were managed in a hospital. Among these hospitalized infants, 14 (41.2%) were aged <2 months. Of the 2,360 cases involving children aged ≥1 year with known outcome, 14 of the children (0.6%) were hospitalized. No fatalities were reported.

Compared with the incidence in Washington, the national incidence for the same period in 2012 was lower overall (4.2 cases per 100,000 population). However, the national

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What is already known on this topic?

The incidence of reported pertussis has increased in the United States after reaching historic lows in the 1970s. Since 2007, children aged 7–10 years have accounted for a substantial proportion of pertussis cases in the United States, a finding attributed to waning immunity in persons fully vaccinated with acellular vaccines in childhood.

What is added by this report?

During January 1–June 16, 2012, the number of reported cases of pertussis in Washington reached 2,520 (37.5 cases per 100,000 residents), a 1,300% increase compared with the same period in 2011. In this epidemic, high rates of disease were observed in adolescents aged 13–14 years, despite high vaccination coverage and recent tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis (Tdap) vaccine administration. Preliminary national incidence data are consistent with the Washington findings.

What are the implications for public health practice?

Increased rates of pertussis among adolescents aged 13–14 years who were fully vaccinated with acellular vaccines in childhood suggests early waning of immunity after vaccination with Tdap vaccine. Studies are ongoing to evaluate Tdap duration of protection in adolescents. The focus of prevention and control efforts is the protection of infants and others at greatest risk for severe disease and improving vaccination coverage in adolescents and adults, especially those who are pregnant. Pertussis vaccination remains the single most effective strategy for prevention of infection.

incidence was increased among infants and children aged 10, 13, and 14 years, consistent with observations in Washington (Figure 3). Throught June 14, 2012, eight deaths have been reported in the United States, with a provisional case-fatality rate of 0.62 per 1,000 for reported cases. In comparison, 0.79 to 2.3 deaths per 1,000 reported cases occurred annually during 2000–2011.

Laboratory Testing

Laboratory confirmation of pertussis cases in Washington was performed by clinical and state health laboratories. Pertussis was laboratory-confirmed in 83.4% of cases: 94.7% by polymerase chain reaction (PCR) alone, 2.4% by culture alone, and 2.9% by both PCR and culture. To further confirm *Bordetella pertussis* as the etiology and evaluate the contribution of other *Bordetella* species, multitarget PCR assays were performed on all specimens submitted to the clinical microbiology laboratory at Seattle Children's Hospital and on a subset of specimens submitted to CDC by the Washington State Public Health Laboratories and a commercial laboratory during January 1–June 16, 2012. Among 5,086 specimens tested at Seattle Children's Hospital, 193 had *Bordetella* DNA detected by PCR. Of these, 175 (90.7%) were positive for *B. pertussis,* 11 (5.7%) for *Bordetella parapertussis*, two (1.0%) for *Bordetella holmesii*, and five (2.6%) were indeterminate. Culture was performed on all 193 PCR-confirmed specimens. *Bordetella* spp.

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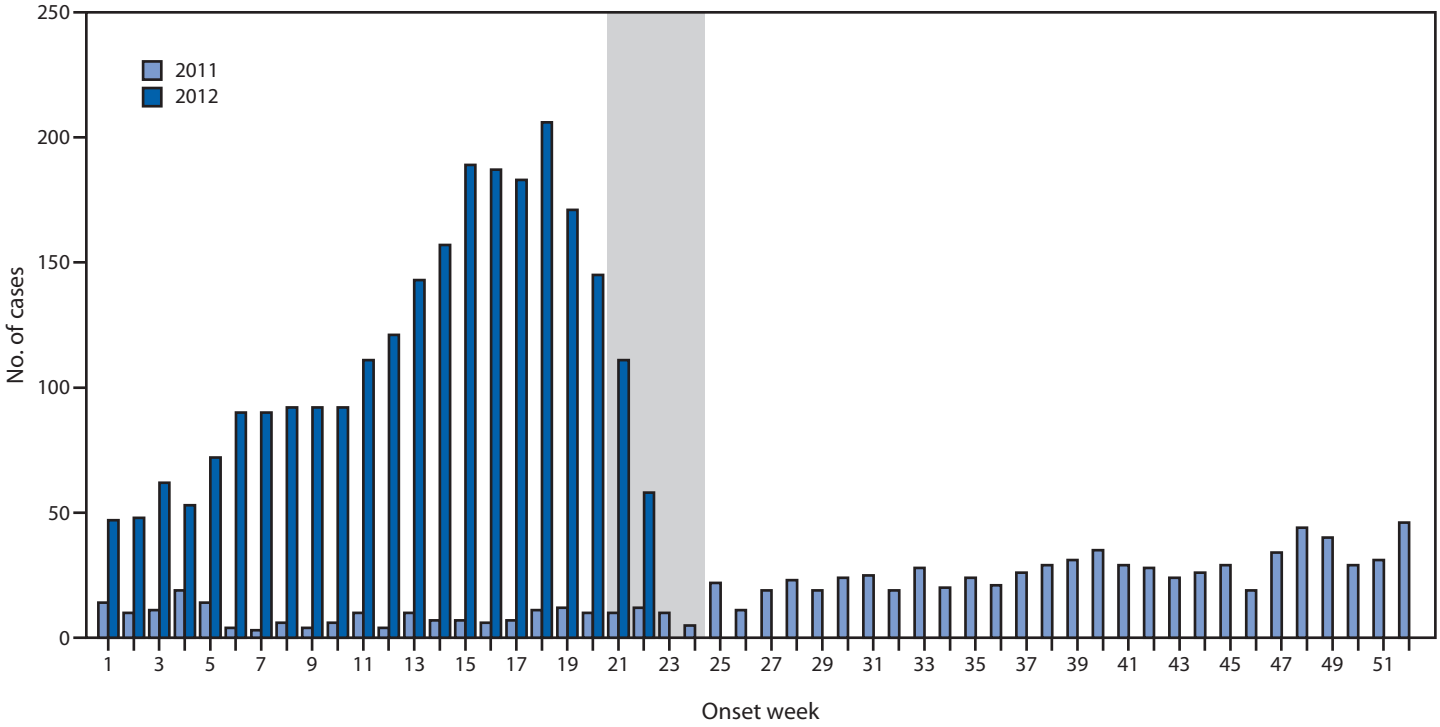
FIGURE 1. Number of confirmed and probable pertussis cases reported, by week of onset — Washington, January 1, 2011–June 16, 2012*



* Reports for 2012 as of June 16, 2012. The shaded area represents a lag during which additional cases likely occurred during 2012, but had not yet been reported to the Washington State Department of Health.

were isolated from 92 (47.7%) specimens, among which *B. pertussis* was identified in 85 (92.4%), *B. parapertussis* in six (6.5%), and *B. holmesii* in one (1.1%). No discrepancies were detected between culture identification and PCR. At CDC, of the 69 specimens tested by multitarget PCR, 59 (85.5%) were positive for *B. pertussis,* one (1.5%) was positive for *B. holmesii*, and nine (13.0%) were indeterminate.

CDC also performed pulsed-field gel electrophoresis (PFGE) testing on 55 isolates and compared those results with a national database of more than 5,000 *B. pertussis* PFGE profiles compiled by CDC during 1990–2011. Among the 55 isolates, 14 PFGE profiles were identified; 30 (54.5%) of the isolates represented the four most commonly identified profiles in the national database. Of the remaining isolates, 20 demonstrated seven of the less common PFGE profiles, and five had three PFGE profiles not previously seen in the national database.

Vaccination Status

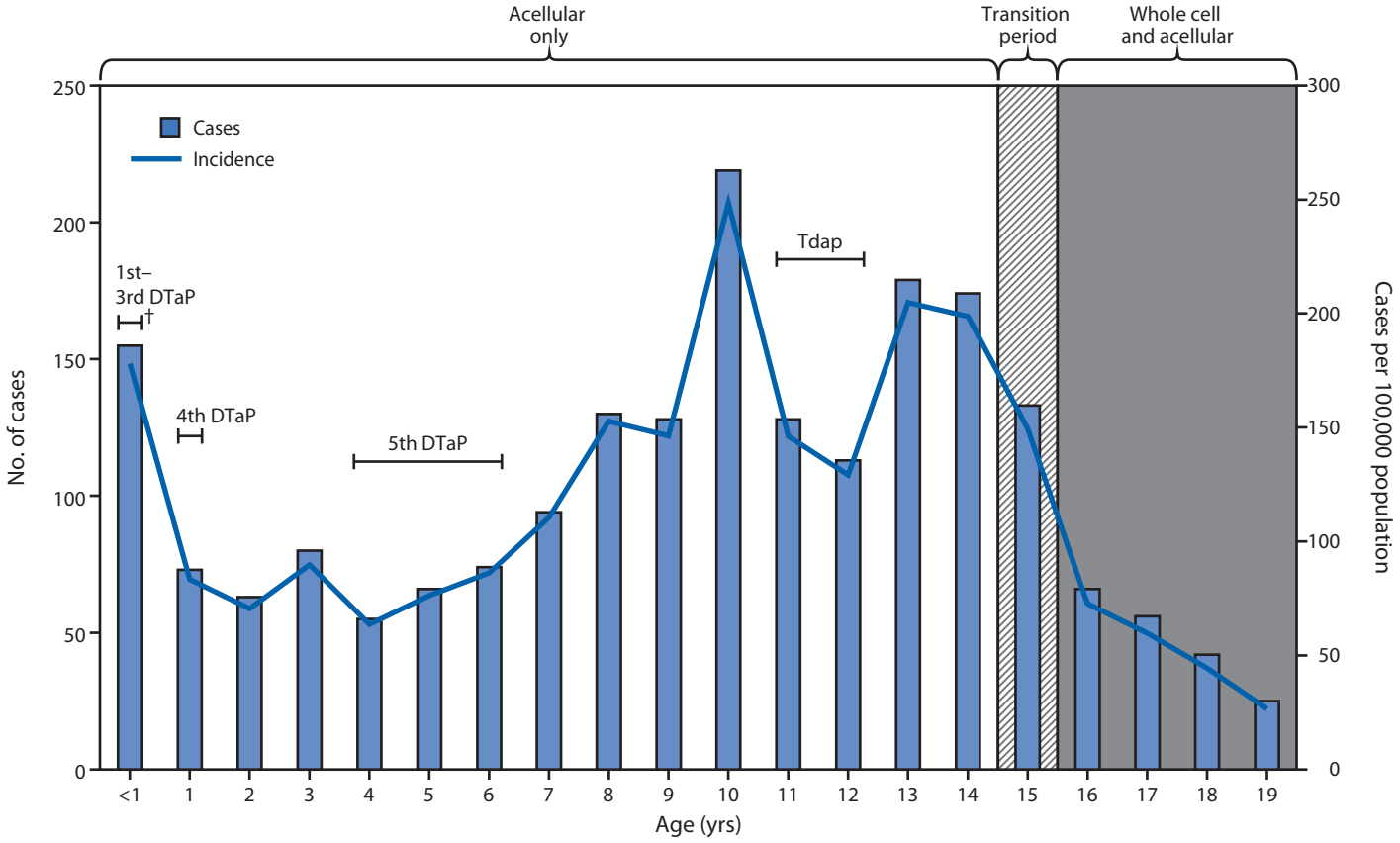
The vaccination status of patients was determined by review of medical records and by patient or parent report. Vaccination was considered up-to-date if the minimum number of doses by age had been received, as recommended by the Advisory Committee on Immunization Practices (*2*). Patients with invalid dose dates (e.g., date of dose preceding date of birth) were excluded from the vaccination status analysis. Individual doses were excluded if administered <14 days before symptom onset.

Valid vaccination history was available for 1,829 of 2,006 (91.2%) patients aged 3 months–19 years. Overall, 758 of 1,000 (75.8%) patients aged 3 months–10 years were up-to-date with the childhood diphtheria and tetanus toxoids and acellular pertussis (DTaP) doses. Receipt of Tdap was documented in 97 of 225 (43.1%) patients aged 11–12 years and in 466 of 604 (77.2%) patients aged 13–19 years. Estimated DTaP coverage in Washington among children aged 19–35 months was 93.2% for ≥3 doses and 81.9% for ≥4 doses in 2010; Tdap coverage in adolescents aged 13–17 years was estimated at 70.6% (*3*).

Epidemic Response

In response to this ongoing epidemic, the state health department established an incident command structure to coordinate epidemic response and surveillance activities. State guidance to local health jurisdictions and American Indian tribes for case investigations was modified to prioritize identification of persons at high risk (i.e., infants and pregnant women). Health-care provider education has focused on clinical presentation, appropriate diagnostic testing, and treatment and prevention recommendations, with specific emphasis on preventing transmission to persons at high risk through vaccination

FIGURE 2. Number and incidence of confirmed and probable pertussis cases among persons aged ≤19 years, by patient age and vaccines received* — Washington, January 1–June 16, 2012



Abbreviations: DTaP = diphtheria and tetanus toxoids and acellular pertussis; DTwP = diphtheria and tetanus toxoids and whole-cell pertussis; Tdap = tetanus and reduced diphtheria toxoids and acellular pertussis.

* Acellular vaccines (DTaP) replaced whole-cell vaccines (DTwP) for the 4th and 5th doses in 1992 and all 5 doses of the childhood series in 1997. Tdap was recommended for adolescents aged 11–12 years in 2006. Thus, all children aged ≤14 years are likely to have received acellular vaccines for the complete childhood series. Adolescents aged 15 years were born during a transition year from whole-cell to acellular vaccines for the childhood series. Adolescents aged ≥16 years received whole-cell vaccines for the first 3 doses, and acellular vaccines for the 4th and 5th doses.

† Ages during which the Advisory Committee on Immunization Practices recommends that specified vaccine doses be administered.

and targeted antibiotic chemoprophylaxis. Public awareness efforts have focused on informing residents about the signs and symptoms of pertussis and vaccination recommendations. Recommended vaccines for children aged ≤18 years are provided by Washington's Universal Childhood Vaccine Program. Tdap receipt among adults increased substantially; from March 25 to May 26, 2012, the state immunization registry recorded 82,453 doses of Tdap in adults aged ≥19 years, compared with 34,171 recorded during the same period in 2011, a 140% increase. An additional 27,000 doses of Tdap were allocated for uninsured or underinsured adults.

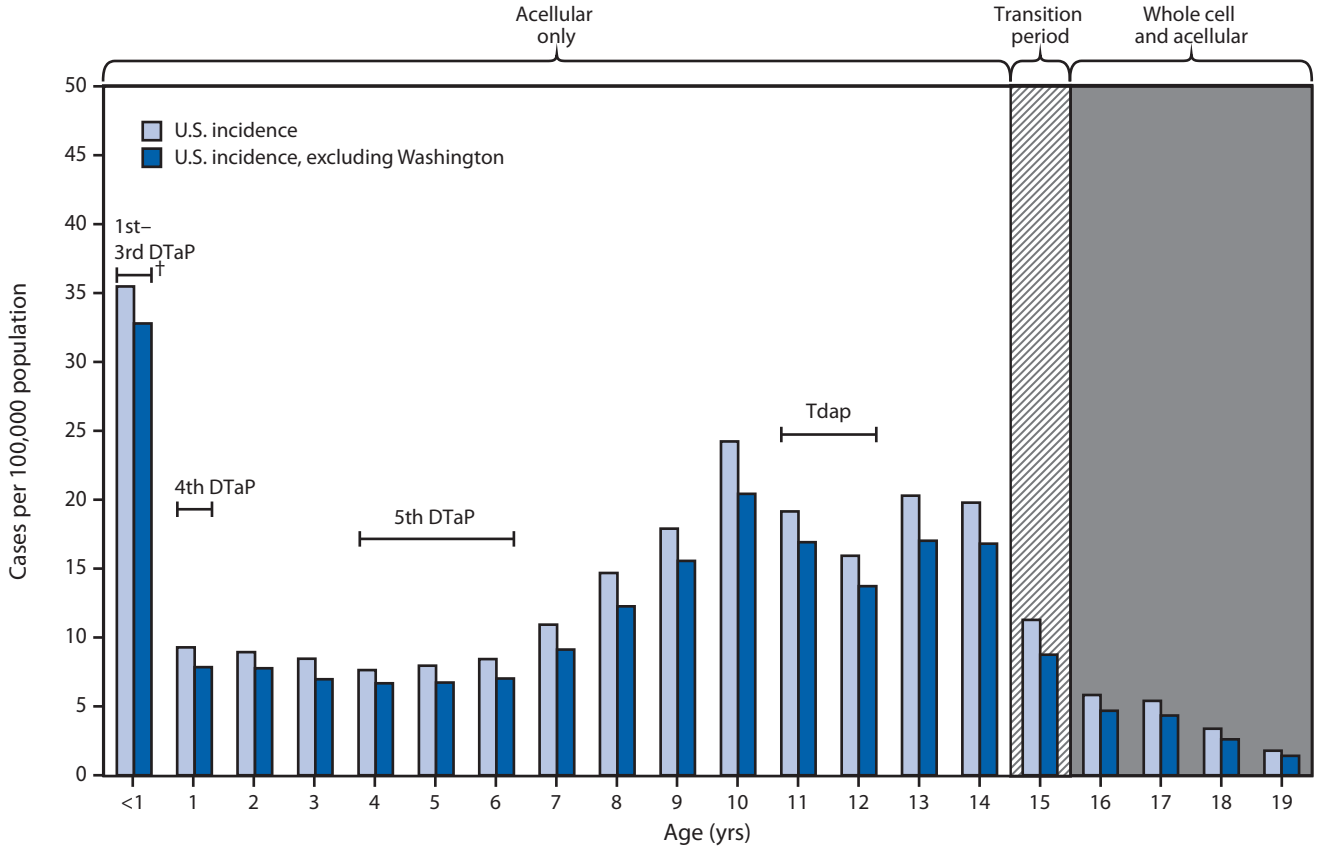
Reported by

Chas DeBolt, MPH, Azadeh Tasslimi, MPH, Janna Bardi, MPH, Brandon Troy Leader, PhD, Brian Hiatt, Washington State Dept of Health. Xuan Quin, PhD, Microbiology Laboratory, Seattle Children's Hospital. Manisha Patel, MD, Stacey Martin, MSc, Maria Lucia Tondella, PhD, Pam Cassiday, MS, Amanda Faulkner, MPH, Nancy E. Messonnier, MD, Thomas A. Clark, MD, Div of Bacterial Diseases, National Center for Immunization and Respiratory Diseases; Sarah Meyer, EIS Officer, CDC. ***Corresponding contributor:*** *Sarah Meyer, smeyer@cdc.gov, 404-639-2761.*

Editorial Note

Pertussis is endemic in the United States. Although cyclical in nature, a gradual and sustained increase has been observed in the United States after reaching historic lows in the 1970s. In 2010, 27,550 pertussis cases were reported. Year-to-date case counts from 2012 have surpassed those from the previous 5 years for the same period. The high rates of pertussis among adolescents aged 13–14 years in Washington reflect national trends and provide observational data suggesting early waning of immunity from acellular vaccines.

FIGURE 3. Incidence of confirmed and probable pertussis among persons aged ≤19 years, by patient age and vaccines received* — National Notifiable Diseases Surveillance System, United States, January 1–June 14, 2012



Abbreviations: DTaP = diphtheria and tetanus toxoids and acellular pertussis; DTwP = diphtheria and tetanus toxoids and whole-cell pertussis; Tdap = tetanus and reduced diphtheria toxoids and acellular pertussis.

* Acellular vaccines (DTaP) replaced whole-cell vaccines (DTwP) for the 4th and 5th doses in 1992 and all 5 doses of the childhood series in 1997. Tdap was recommended for adolescents aged 11–12 years in 2006. Thus, all children aged ≤14 years are likely to have received acellular vaccines for the complete childhood series. Adolescents aged 15 years were born during a transition year from whole-cell to acellular vaccines for the childhood series. Adolescents aged ≥16 years received whole-cell vaccines for the first 3 doses, and acellular vaccines for the 4th and 5th doses.

† Ages during which the Advisory Committee on Immunization Practices recommends that specified vaccine doses be administered.

Acellular and whole-cell vaccines both have high efficacy during the first 2 years after vaccination, but recent changes in the epidemiology of pertussis in the United States strongly suggest diminished duration of protection afforded by childhood acellular vaccine (DTaP) compared with that of diphtheria and tetanus toxoids and whole-cell pertussis (DTwP) vaccine (*4*). In contrast with acellular vaccines, which contain several specific antigens, whole-cell vaccines are suspensions of entire killed *B. pertussis* organisms. The additional antigenic components in DTwP vaccines might induce immune responses with greater durability. Concerns about adverse events associated with DTwP led to replacement with DTaP for the complete childhood series in 1997. Since the mid-2000s, the incidence of pertussis among children aged 7–10 years has increased. Moreover, the observed increase in risk by year of life from age 7–10 years suggests a cohort effect of increasing susceptibility as those children who exclusively received acellular vaccines continue to age.

In 2006, Tdap was recommended for adults and adolescents, with routine vaccination recommended at age 11–12 years. Although the relative reduction in incidence of pertussis among adolescents aged 11–12 years demonstrates immediate vaccine effectiveness, the increasing number of cases in adolescents aged 13–14 years in both Washington and the United States suggests immunity wanes after Tdap vaccination in those adolescents fully vaccinated with acellular vaccines during childhood (*5*). In observational studies, Tdap effectiveness was 66%–72% among adolescents who largely received DTwP for some of the childhood doses (*5,6*). Studies evaluating Tdap effectiveness and duration of protection in adolescents fully vaccinated with DTaP are being conducted in Washington and California.

Investigation of the Washington epidemic demonstrates multiple *B. pertussis* strains causing infection, primarily in vaccinated persons. Given the high transmissibility of *B. pertussis*, a proportion of vaccinated persons remains susceptible and can

become infected during a pertussis outbreak. Unvaccinated children have at least an eightfold greater risk for pertussis than children fully vaccinated with DTaP (*7*). However, because in most of the cases the patients were vaccinated, the 4.5% of Washington school children who were exempted from vaccination during 2011–2012 represented only a small proportion of those at risk for pertussis in the state. Although vaccinated children can develop pertussis, they are less infectious, have milder symptoms and shorter illness duration, and are at reduced risk for severe outcomes, including hospitalization (*8–10*).

The ongoing pertussis epidemic in Washington reflects the evolving epidemiology of pertussis in the United States. Although acellular pertussis vaccines provide excellent short-term protection, early waning of immunity might be contributing to increasing population-level susceptibility. Nevertheless, vaccination continues to be the single most effective strategy to reduce morbidity and mortality caused by pertussis. Vaccination of pregnant women and contacts of infants is recommended to protect infants too young to be vaccinated. In light of the increased incidence of pertussis in Washington and elsewhere, efforts should focus on full implementation of DTaP and Tdap recommendations to prevent infection and protect infants.

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Motor Vehicle Crash Deaths in Metropolitan Areas — United States, 2009

Although rates have declined in recent years, motor vehicle crashes (MVCs) remain a leading cause of injury death in the United States (*1*). In 2009, a total of 34,485 MVC deaths were reported among U.S. residents, and 22% of those who died were aged 15–24 years. MVCs were the leading cause of death for that age group, which represents approximately 14% of the total U.S. population (*1*). To assess patterns in MVC death rates for persons of all ages and for those aged 15–24 years, in recognition of the elevated risk for this age group, CDC used data from the National Vital Statistics System (NVSS) and the U.S. Census Bureau for 2009 representing the 50 most populous U.S. metropolitan statistical areas (MSAs). The overall MVC death rate (age-adjusted) for all 50 MSAs combined was 8.2 per 100,000 residents, compared with a national rate of 11.1; among MSAs, rates ranged from 4.4 to 17.8. For persons aged 15–24 years, the MVC death rate was 13.0 per 100,000 residents for all MSAs combined (range: 7.3–25.8), compared with a national rate of 17.3. Although rates for the MSAs generally were lower than the rate for the nation as a whole, higher rates for persons aged 15–24 years were observed both in the MSAs and nationally. The wide variation in rates among MSAs suggests a need to better understand how urban development patterns might relate to MVC deaths and to identify and implement effective strategies to reduce the number of such deaths.

NVSS data for the year 2009 were used to identify MVC deaths among U.S. residents (*2*). Geographic codes indicating county and city of residence were used to tabulate MVC death counts for the 50 largest MSAs (by population rank as of midyear 2009) and for 63 major cities within these MSAs.* These counts were combined with U.S. Census Bureau population estimates for MSAs and cities to calculate population-based rates for persons of all ages and for those aged 15–24 years. Overall rates (all ages) were age-adjusted to the year 2000 U.S. standard population. MVC death counts <20 (and associated rates) were not reported for MSAs and cities because of concerns regarding statistical reliability and data confidentiality. However, such counts were included in the statistics for all MSAs and all cities combined.

The overall MVC death rate for all 50 MSAs combined was 8.2 per 100,000 residents, compared with a national rate of 11.1 (Table). Among the 50 MSAs, rates ranged from 4.4 to 17.8, and 37 (74%) had rates lower than the overall national rate. Rates for MSAs generally were higher in southern states, with the highest rates concentrated in the southeastern United States (Figure). Within the 50 MSAs, the overall MVC death rate for the 63 major cities combined was 7.9 per 100,000 residents. The overall rate for the 53 cities with individually reported data ranged from 3.9 to 19.4. Although the combined rate for the cities was slightly lower than the combined rate for MSAs, 24 (45%) of the 53 cities with individually reported data had higher overall rates than their MSAs.

Among persons aged 15–24 years, the MVC death rate for all MSAs combined was 13.0 per 100,000 residents, compared with a national rate of 17.3. Data for this age group were reported individually for 47 of the 50 MSAs; rates ranged from 7.3 to 25.8 and were uniformly higher than the overall (all ages) rates within the respective MSAs. The combined rate for persons aged 15–24 years residing in major cities within these MSAs was 10.9. Rates for this age group were reported individually for only 10 of the 63 major cities, limiting further assessment.

Reported by

Scott R. Kegler, PhD, Office of Statistics and Programming, Laurie F. Beck, MPH, Erin K. Sauber-Schatz, PhD, Div of Unintentional Injury Prevention, National Center for Injury Prevention and Control, CDC. ***Corresponding contributor:*** *Laurie F. Beck, lbeck@cdc.gov, 770-488-4327.*

Editorial Note

The combined overall MVC death rate for the 50 most populous MSAs in the United States was lower than the overall national rate; residents of the MSAs represented 54% of the U.S. population in 2009, while accounting for only 40% of all MVC deaths. In addition, the combined overall MVC death rate for 63 major cities within the MSAs was lower than the combined overall rate for the MSAs. These findings are consistent with statistics showing that MVC death rates are lower in urban areas than in rural areas (*3*).

Although overall MVC death rates in the MSAs generally were lower than the national rate, rates varied widely among MSAs. A similar pattern was observed for MVC death rates among persons aged 15–24 years. In general, rates were higher in MSAs in the southern United States. Urban sprawl, which is a function of residential density, land use mix, presence (or absence) of town centers, and street accessibility (a measure of street density and interconnectedness), might, in part, explain these findings (*4*). Previous research has shown that sprawl is more common in the southern United States, and that MVC death rates are higher in sprawling metropolitan areas than in compact metropolitan areas (*5,6*). A growing body of literature

* An MSA is defined by the U.S. Office of Management and Budget as "a core area containing a substantial population nucleus, together with adjacent communities."

TABLE. Number and annual rate* of motor vehicle crash deaths for the 50 most populous metropolitan statistical areas and 63 of their major cities — National Vital Statistics System and U.S. Census Bureau, 2009

	Overall†		Ages 15–24 yrs	
Metropolitan statistical area/Major cities	No.	Rate§	No.	Rate
U.S. total	**34,485**	**11.1**	**7,451**	**17.3**
Metropolitan statistical area total (n = 50)	**13,696**	**8.2**	**2,903**	**13.0**
Major city total (n = 63)	**3,985**	**7.9**	**769**	**10.9**
Atlanta – Sandy Springs – Marietta, Georgia	564	10.7	94	12.9
Atlanta	64	12.0	—¶	—
Austin – Round Rock, Texas	174	10.3	37	15.5
Austin	64	8.3	—	—
Baltimore – Towson, Maryland	253	9.1	46	12.4
Baltimore	44	6.6	—	—
Birmingham – Hoover, Alabama	176	15.3	37	25.8
Birmingham	37	15.4	—	—
Boston – Cambridge – Quincy, Massachusetts – New Hampshire	244	5.0	63	10.0
Boston	—	—	—	—
Buffalo – Niagara Falls, New York	81	6.9	22	14.1
Buffalo	—	—	—	—
Charlotte – Gastonia – Concord, North Carolina – South Carolina	171	10.0	34	15.2
Charlotte	66	9.8	—	—
Chicago – Naperville – Joliet, Illinois – Indiana – Wisconsin	565	5.9	116	8.8
Chicago	174	6.0	33	8.1
Cincinnati – Middletown, Ohio – Kentucky – Indiana	171	7.8	37	12.8
Cincinnati	30	9.0	—	—
Cleveland – Elyria – Mentor, Ohio	96	4.4	—	—
Cleveland	—	—	—	—
Columbus, Ohio	150	8.3	30	12.7
Columbus	45	6.3	—	—
Dallas – Fort Worth – Arlington, Texas	611	9.8	135	15.6
Dallas	99	8.0	21	12.2
Fort Worth	62	8.8	—	—
Arlington	36	10.4	—	—
Plano	—	—	—	—
Denver – Aurora – Broomfield, Colorado	186	7.5	34	10.9
Denver	45	7.8	—	—
Aurora	28	8.6	—	—
Detroit – Warren – Livonia, Michigan	336	7.5	76	13.4
Detroit	113	12.6	24	17.3
Hartford – West Hartford – East Hartford, Connecticut	78	6.4	—	—
Hartford	—	—	—	—
Houston – Sugar Land – Baytown, Texas	722	12.6	174	21.5
Houston	292	12.9	67	21.3
Indianapolis – Carmel, Indiana	141	8.2	43	19.2
Indianapolis (balance)	75	9.2	21	19.6
Jacksonville, Florida	179	13.3	45	25.4
Jacksonville	101	12.4	31	27.2
Kansas City, Missouri – Kansas	216	10.6	45	17.3
Kansas City	61	12.6	—	—
Las Vegas – Paradise, Nevada	158	8.6	33	13.7
Las Vegas	85	15.7	—	—
Henderson	21	8.1	—	—
Los Angeles – Long Beach – Santa Ana, California	848	6.6	173	9.5
Los Angeles	293	7.7	54	9.8
Long Beach	27	6.1	—	—
Santa Ana	—	—	—	—
Anaheim	25	7.8	—	—
Louisville – Jefferson County, Kentucky – Indiana	146	11.8	23	14.8
Louisville (metro)	72	10.0	—	—

See table footnotes on page 526.

TABLE. (*Continued*) Number and annual rate* of motor vehicle crash deaths for the 50 most populous metropolitan statistical areas and 63 of their major cities — National Vital Statistics System and U.S. Census Bureau, 2009

	Overall†		Ages 15–24 yrs	
Metropolitan statistical area/Major cities	**No.**	**Rate§**	**No.**	**Rate**
Memphis, Tennessee – Mississippi – Arkansas	225	17.8	40	22.0
Memphis	114	17.4	—¶	—
Miami – Fort Lauderdale – Pompano Beach, Florida	634	11.1	124	18.0
Miami	75	16.1	—	—
Milwaukee – Waukesha – West Allis, Wisconsin	102	6.4	21	10.0
Milwaukee	43	7.0	—	—
Minneapolis – St. Paul – Bloomington, Minnesota – Wisconsin	206	6.3	40	9.2
Minneapolis	28	7.3	—	—
St. Paul	—	—	—	—
Nashville-Davidson – Murfreesboro – Franklin, Tennessee	203	13.0	47	22.7
Nashville-Davidson (balance)	60	9.9	—	—
New Orleans – Metairie – Kenner, Louisiana	142	12.2	26	16.5
New Orleans	35	9.9	—	—
New York – Northern New Jersey – Long Island, New York – New Jersey – Pennsylvania	986	5.1	179	7.3
New York City (five boroughs)	330	3.9	46	4.3
Newark	—	—	—	—
Oklahoma City, Oklahoma	142	11.6	36	19.7
Oklahoma City	54	9.7	—	—
Orlando – Kissimmee, Florida	239	11.3	49	17.6
Orlando	47	19.4	—	—
Philadelphia – Camden – Wilmington, Pennsylvania – New Jersey – Delaware – Maryland	444	7.3	92	11.2
Philadelphia	111	7.1	—	—
Phoenix – Mesa – Scottsdale, Arizona	392	9.1	74	12.9
Phoenix	137	9.1	27	12.5
Mesa	39	8.5	—	—
Glendale	42	16.7	—	—
Pittsburgh, Pennsylvania	228	9.2	49	16.5
Pittsburgh	22	7.2	—	—
Portland – Vancouver – Beaverton, Oregon – Washington	148	6.8	41	14.8
Portland	36	6.2	—	—
Providence – New Bedford – Fall River, Rhode Island – Massachusetts	119	7.1	22	9.6
Providence	—	—	—	—
Raleigh – Cary, North Carolina	108	9.6	28	19.4
Raleigh	28	6.5	—	—
Richmond, Virginia	144	11.5	29	17.0
Richmond	21	9.6	—	—
Riverside – San Bernardino – Ontario, California	434	10.6	110	16.8
Riverside	35	11.9	—	—
Sacramento – Arden-Arcade – Roseville, California	178	8.3	34	11.0
Sacramento	69	14.4	—	—
St. Louis, Missouri – Illinois	302	10.6	73	19.4
St. Louis	31	8.6	—	—
Salt Lake City, Utah	87	8.4	—	—
Salt Lake City	—	—	—	—
San Antonio, Texas	271	13.4	59	19.6
San Antonio	164	12.3	37	17.8
San Diego – Carlsbad – San Marcos, California	247	7.8	61	13.3
San Diego	77	5.6	—	—
San Francisco – Oakland – Fremont, California	244	5.6	51	10.1
San Francisco	37	4.0	—	—
Oakland	20	5.0	—	—
San Jose – Sunnyvale – Santa Clara, California	105	6.0	23	10.0
San Jose	71	8.1	—	—

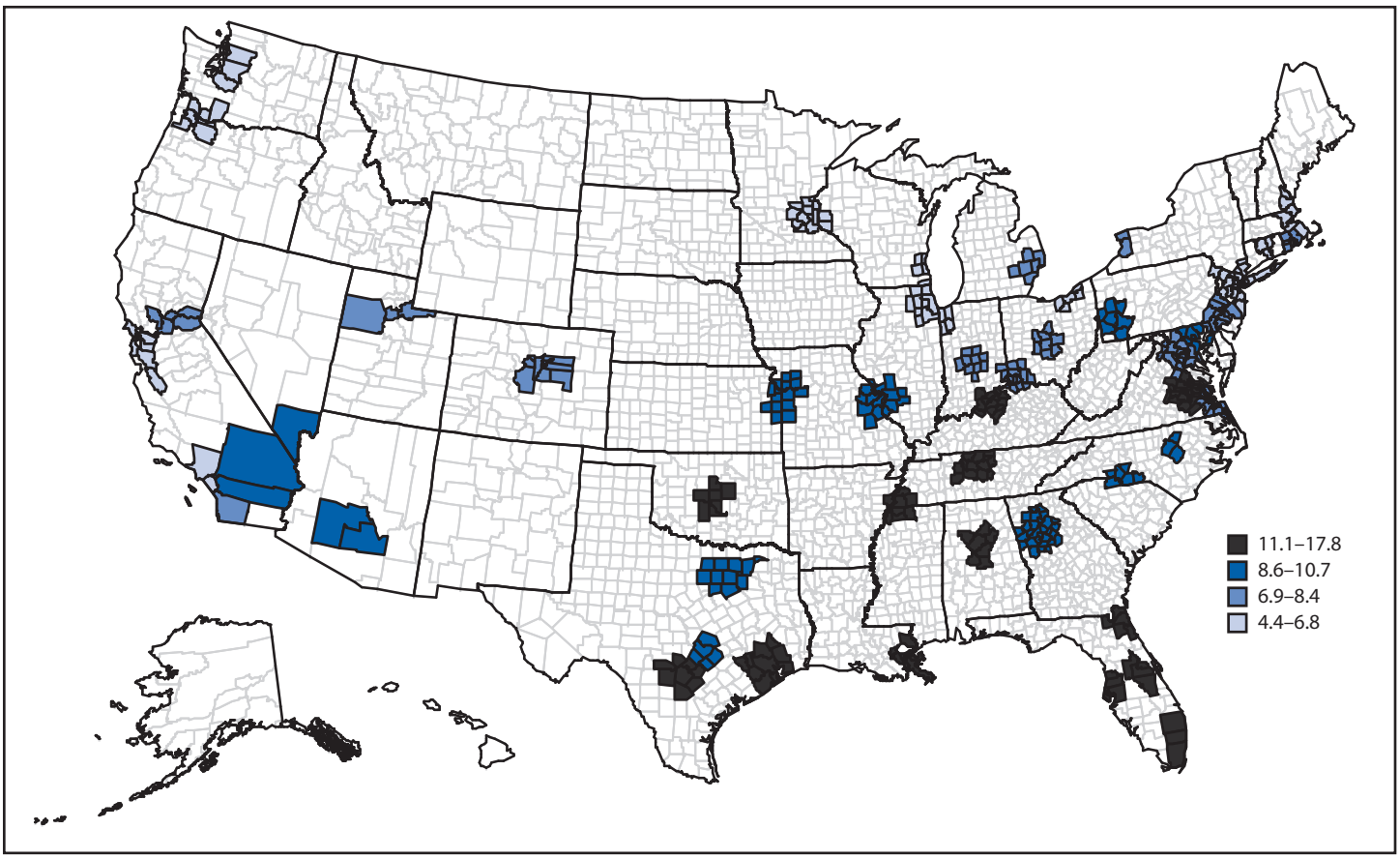
See table footnotes on page 526.

TABLE. (*Continued*) Number and annual rate* of motor vehicle crash deaths for the 50 most populous metropolitan statistical areas and 63 of their major cities — National Vital Statistics System and U.S. Census Bureau, 2009

Metropolitan statistical area/Major cities	Overall†		Ages 15–24 yrs	
	No.	Rate§	No.	Rate
Seattle – Tacoma – Bellevue, Washington	206	5.9	48	10.9
Seattle	34	5.2	—¶	—
Tampa – St. Petersburg – Clearwater, Florida	358	12.6	64	19.5
Tampa	49	13.9	—	—
Virginia Beach – Norfolk – Newport News, Virginia – North Carolina	128	7.3	34	12.6
Virginia Beach	39	8.6	—	—
Washington – Arlington – Alexandria, DC – Virginia – Maryland – West Virginia	408	7.5	105	14.4
Washington, DC	37	5.9	—	—

Abbreviation: DC = District of Columbia.
* Per 100,000 population. Numbers and rates reflect decedent place of residence, not place of occurrence.
† Five deaths were excluded from metropolitan statistical area and major city tabulations because of undocumented decedent age.
§ Age-adjusted to the year 2000 United States standard population.
¶ Data not shown because of statistical instability or data confidentiality concerns, both associated with small numbers.

FIGURE. Motor vehicle crash death rates* for the 50 most populous metropolitan statistical areas — National Vital Statistics System and U.S. Census Bureau, 2009



* Per 100,000 population, age-adjusted.

examines urban development and its association with MVCs. For example, urban sprawl has been associated with greater driving exposure (i.e., number of miles driven) (*4,6*).

MVCs among teens and young adults (i.e., persons aged 15–24 years) are of particular concern because they represent the leading cause of death in this age group (*1*). Although MVC death rates for this age group generally were lower in MSAs than for the nation, they nonetheless were routinely higher than overall rates within individual MSAs. For all MSAs combined, the MVC death rate among persons aged 15–24 years was 13.0, which is 59% higher than the combined overall rate for MSAs of 8.2. Well-known risk factors (e.g., inexperience, lack of seat belt use, driving with teen passengers, and alcohol-impaired driving) for teens and young adults likely contributed to the higher MVC death rates observed for this age group.

Graduated driver licensing (GDL) programs initially limit teens' independent driving and gradually introduce them to more complex and higher-risk conditions as they gain more experience and move through successive stages (i.e., permit, intermediate/provisional license, and fully unrestricted license) (*7*). GDL is one evidence-based intervention that can reduce crashes among young drivers, with stronger programs exhibiting greater effect (*8*). For example, fatal crash rates are lower where GDL programs restrict young drivers from having any passengers than where programs permit one or more passengers (*7*).

Effective interventions to reduce alcohol-impaired driving (e.g., sobriety checkpoints and ignition interlock programs for drivers convicted of alcohol-impaired driving) benefit drivers of all ages, including young drivers who have disproportionately high rates of impaired driving and involvement in alcohol-related fatal MVCs (*9,10*).

The findings in this report are subject to at least four limitations. First, death rates were based on decedent place of residence rather than on place of MVC occurrence; some deaths actually might have resulted from incidents that occurred far away from the city or MSA for which they were reported. Second, MVC death statistics for certain MSAs and cities were not reported because of reliability and confidentiality concerns associated with small counts; for persons aged 15–24 years, data were reported for only 10 of the 63 cities considered. Third, information on risk factors associated with MVC deaths (e.g., nonuse of seat belts, blood alcohol concentration at the time of crash, or vehicle speed) are not included in the NVSS data and therefore, assessing the effects of such factors was not possible. Finally, NVSS data for Ohio, New Jersey, West Virginia, and the District of Columbia showed notable increases for the year 2009 in the number of records with the underlying cause of death coded as "other ill-defined and unspecified causes of mortality," whereas the data for Georgia showed a notable decrease in such records. Although the impact of these changes on MVC statistics is not known, the percentage of such records for each individual state has remained consistently low (≤5%).

Motor vehicle injury prevention is an important public health priority. The findings in this report indicate that MVC death rates in 2009 varied widely among MSAs, both for residents of all ages and for those aged 15–24 years. Better identification of risk factors related to higher MVC death rates in certain MSAs is needed, followed by implementation of effective strategies for minimizing risks and associated deaths. Proven population-based interventions such as strong graduated driver licensing and alcohol-impaired driving prevention policies offer the potential to further reduce MVC deaths among teens and young adults, the population most at risk for MVCs.

What is already known on this topic?

Although rates have been declining in recent years, motor vehicle crashes (MVCs) remain a leading cause of injury death in the United States and represent the leading cause of death among teens and young adults nationally.

What is added by this report?

In 2009, MVC death rates for the 50 most populous U.S. metropolitan statistical areas (MSAs) combined were lower than for the nation overall, for persons of all ages and for those aged 15–24 years. However, MVC death rates varied widely among MSAs, ranging from 4.4 to 17.8 per 100,000 residents of all ages (age-adjusted) and from 7.3 to 25.8 per 100,000 residents aged 15–24 years. In general, rates were higher among MSAs in the southern states. Higher MVC death rates for persons aged 15–24 years were observed both for the MSAs and nationally, compared with rates for persons of all ages.

What are the implications for public health practice?

The variation in MVC death rates among MSAs suggests a need to better understand the influence of urban development patterns and other factors so that effective strategies can be implemented to reduce MVC deaths. Proven population-based interventions such as strong graduated driver licensing and alcohol-impaired driving prevention policies offer the potential to reduce MVC deaths among teens and young adults.

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Tornado-Related Fatalities — Five States, Southeastern United States, April 25–28, 2011

During April 25–28, 2011, a massive storm system generated 351 tornadoes (including 15 registering 4 or 5 on the Enhanced Fujita [EF] scale*), killing 338 persons in Alabama, Arkansas, Georgia, Mississippi, and Tennessee (*1*). This was the third-deadliest tornado event in the United States, surpassing an April 1974 event that resulted in 315 fatalities (*1,2*). This event also was historic because of the record number of fatalities that occurred despite modern advances in tornado forecasting, advanced warning times, and media coverage (*1–3*). Risk factors for death and injury from tornadoes are sheltering in mobile homes, proximity to an EF-4 or EF-5 tornado, being an older adult (aged ≥65 years), lack of accessibility to safe rooms (e.g., basements or reinforced shelters), and a night-time tornado impact (*4–6*). To describe the fatalities by demographic characteristics, type of shelter used, cause of death, and tornado severity and location, CDC reviewed data from the American Red Cross (Red Cross), death certificates, and the National Weather Service (NWS). This report summarizes the results of that review. Among the 338 decedents, median age was 55.0 years (range: 4 days–97 years); approximately one third were older adults. On tornado impact, 46.7% of decedents were in single-family homes, and 26.6% were in mobile homes. The leading cause of death was traumatic injury, including 21.9% with head injuries. Half of the deadly tornadoes were rated EF-4 or EF-5 and were responsible for 89.5% of the deaths. To prevent tornado-related deaths, health messaging should encourage the public (especially older adults and residents of mobile/manufactured homes) to pre-identify an accessible safe room, prepare the room with personal protection items (e.g., blankets and helmets), and monitor local weather (*7,8*).

NWS forecast the storm system 5 days in advance and issued numerous tornado watches (mean lead time: 2.4 hours) and warnings (mean lead time: 22 minutes) (*1,3*). Through extensive local media coverage, residents were encouraged to prepare for tornado impact and to seek immediate cover in a safe place (*1,3*). Because of the limitations of tornado meteorology, the exact geographic impact and strength of the tornadoes were not determined by NWS (per protocol) until after the storm (*1,9*).

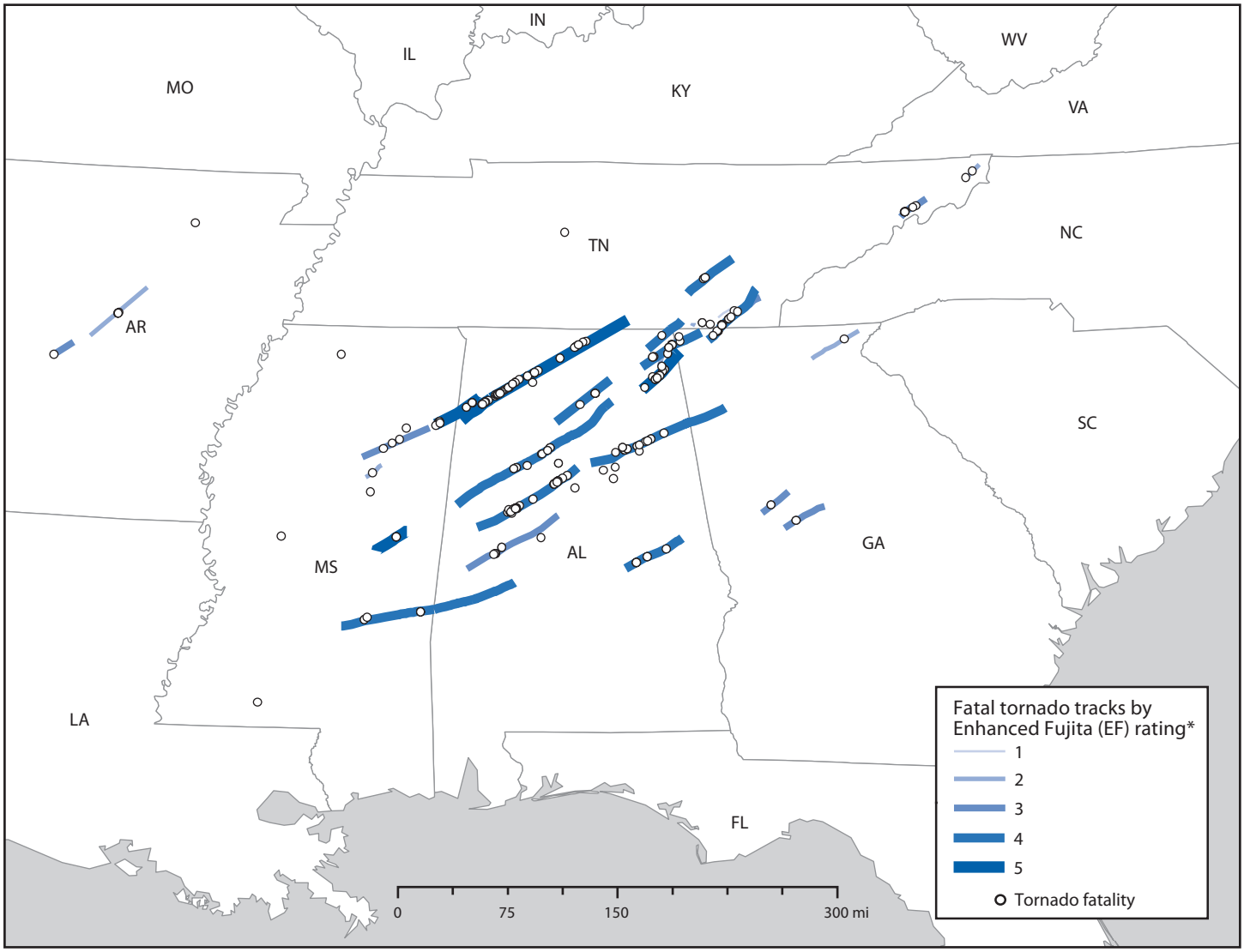
The Red Cross identified tornado-related fatalities from various sources, such as media reports, coroners, funeral homes, and emergency managers. Volunteers conducted semi-structured interviews with available next-of-kin to identify any disaster-related needs. For each fatality, the Red Cross completed a standardized mortality surveillance form that captured demographics of the deceased, the location of the deceased at the time of injury, the location of the recovery of the body, the cause of the injury, and the circumstance surrounding the death. A case was defined as any fatality attributed directly (e.g., house collapse) or indirectly (e.g., stress-induced myocardial infarction) to the April 25–28 tornado disaster that affected the southeastern states. The mortality form data, including the immediate and underlying cause(s) of death, were verified with official death certificates from the state health departments of the five affected states. A list of the decedents' names was provided to the five state vital statistics offices by CDC to obtain the death certificates. The five vital statistics offices conducted additional searches using key word searches (e.g., tornado) and *International Classification of Diseases, 10th Revision* code X37 ("cataclysmic storm") to identify additional tornado-related deaths occurring through December 31, 2011. Frequencies of the variables were calculated.

Spatial information on the tornado tracks, including the lengths and widths and maximum strength (on the EF scale) were available via NWS post-storm damage assessments (*1,2*). Using these data, a composite of the multistate tornado tracks was created, and physical address at the time of injury or illness for each fatality was geocoded using mapping software. Addresses were then spatially assigned to specific tornadoes by nearest proximity using a 5-mile (8-km) radius. A death within the radius was linked to that particular tornado.

A total of 338 fatalities caused by 27 tornadoes occurred in the five states; 15 of the 27 deadly tornadoes reached EF-4 or EF-5 strengths (Figure). These powerful EF-4 and EF-5 tornadoes were especially dangerous because of their rapid forward velocity of >50 mph (>81 km/h) and prolonged duration on the ground, averaging 66 miles (106 km) and crossing multiple counties and states (*1,2,9*). The majority of the fatalities occurred during the late afternoon and evening hours on April 27, when numerous long-track tornadoes (paths >15 miles [>24 km]) moved from central and northern Mississippi to the major metropolitan areas of Tuscaloosa and Birmingham, Alabama, as well as across parts of eastern Tennessee and Georgia (*1,9*; Gregory Carbin, NWS, personal

* EF tornado rating scale estimates the strongest wind gusts that occur 10 meters above the ground: EF-0 (65–85 mph [105–137 km/h]), EF-1 (86–110 mph [138–177 km/h]), EF-2 (111–135 mph [178–217 km/h]), EF-3 (136–165 mph [218–266 km/h]), EF-4 (166–200 mph [267–322 km/h]), and EF-5 (>200 mph [>322 km/h]). Additional information available at http://www.depts.ttu.edu/weweb/pubs/fscale/efscale.pdf.

FIGURE. All direct and indirect tornado-related fatalities and associated tornado tracks — southeastern United States, April 25–28, 2011



* EF tornado rating scale estimates the strongest wind gusts that occur 10 meters above the ground: EF-0 (65–85 mph [105–137 km/h]), EF-1 (86–110 mph [138–177 km/h]), EF-2 (111–135 mph [178–217 km/h]), EF-3 (136–165 mph [218–266 km/h]), EF-4 (166–200 mph [267–322 km/h]), and EF-5 (>200 mph [>322 km/h]). Additional information available at http://www.depts.ttu.edu/weweb/pubs/fscale/efscale.pdf.

communication, 2012). A total of 306 (90.5%) addresses of decedents were within a 5-mile (8-km) radius of a tornado; of these, 274 (89.5%) were attributed to an EF-4 or EF-5 tornado (Figure).

Overall, 57.7% of the decedents' were female, and 32.5% were aged ≥65 years (median: 55.0 years [range: 4 days–97 years]). The majority of the deceased were white (83.7%) and non-Hispanic (92.9%). The most common locations of injury were single-family homes (46.7%) and mobile homes (26.6%) (Table 1). Based on the narratives documented on the death certificates and from interviews with next-of-kin, 306 (90.5%) of the injuries occurred indoors, yet 125 (37.0%) of the bodies were recovered outside (Table 1). The majority of the deaths were on the date of the tornado exposure (319 [94.0%]), directly related to the tornado (318 [94.1%]), and from multisystem trauma (324 [95.6%]), including 74 (21.9%) that indicated a head injury (Table 2). A total of 20 deaths were indirectly related to the tornados: seven from smoke and carbon monoxide asphyxiation, four from cardiovascular events, three from medical equipment failure during a power failure, two from medical complications secondary to tornado injuries, two from motor vehicle crashes, and two from falls or injuries during cleanup (Table 2).

TABLE 1. Number and percentage of tornado fatalities, by date of death, location of injury, and location of body recovery or death — five states, southeastern United States, April 25–28, 2011

	Total*		Alabama		Arkansas		Georgia		Mississippi		Tennessee	
Characteristic	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**
No. of fatalities	**338**	**(100.0)**	**247**	**(73.1)**	**7**	**(2.1)**	**15**	**(4.4)**	**36**	**(10.6)**	**33**	**(9.8)**
Date of death												
April 25	**5**	**(1.5)**	0	—	5	(71.4)	0	—	0	—	0	—
April 26	**2**	**(0.6)**	0	—	1	(14.3)	0	—	1	(2.8)	0	—
April 27	**271**	**(80.2)**	212	(85.8)	1	(14.3)	7	(46.7)	32	(88.9)	19	(57.6)
April 28	**40**	**(11.8)**	20	(8.1)	0	—	8	(53.3)	1	(2.8)	11	(33.3)
After April 28	**18**	**(5.3)**	15	(6.1)	0	—	0	—	0	—	3	(9.1)
Location of injury or illness												
Single-family home	**158**	**(46.7)**	133	(53.8)	0	—	7	(46.7)	12	(33.3)	6	(18.2)
Mobile home	**90**	**(26.6)**	51	(20.6)	2	(28.6)	3	(20.0)	12	(33.3)	22	(66.7)
Motor vehicle	**15**	**(4.4)**	11	(4.5)	1	(14.3)	0	—	2	(5.6)	1	(3.0)
Outside	**11**	**(3.3)**	4	(1.6)	1	(14.3)	1	(6.7)	4	(11.1)	1	(3.0)
Other or home type unspecified†	**58**	**(17.2)**	44	(17.8)	3	(42.9)	4	(26.7)	6	(16.7)	1	(3.0)
Location of body recovery or death												
Private home§	**148**	**(43.8)**	97	(39.3)	1	(14.3)	6	(40.0)	20	(55.6)	24	(72.7)
Outside	**125**	**(37.0)**	95	(38.5)	2	(28.6)	7	(46.7)	13	(36.1)	8	(24.2)
Hospital	**31**	**(9.2)**	27	(10.9)	1	(14.3)	0	—	2	(5.6)	1	(3.0)
Other	**12**	**(3.6)**	10	(4.0)	2	(28.6)	0	—	0	—	0	—
Motor vehicle	**8**	**(2.4)**	6	(2.4)	1	(14.3)	1	(6.7)	0	—	0	—

* Missing data: Alabama (location of injury or illness = four instances; location of body recovery or death = 12), Georgia (location of body recovery or death = one), Mississippi (date of body recovery or death = two; location of body recovery or death = one), and Tennessee (location of injury or illness = two).
† Includes an apartment or business.
§ Includes a home belonging to the decedent, family, or friends, or home type unspecified.

TABLE 2. Number and percentage of fatalities, by tornado-relatedness and cause of death — five states, southeastern United States, April 25–28, 2011

	Total		Alabama		Arkansas		Georgia		Mississippi		Tennessee	
Characteristic	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**
No. of fatalities	**338**	**(100.0)**	**247**	**(73.1)**	**7**	**(2.1)**	**15**	**(4.4)**	**36**	**(10.6)**	**33**	**(9.8)**
Tornado-relatedness*												
Direct	**318**	**(94.1)**	235	(95.1)	6	(85.7)	15	(100.0)	33	(91.7)	29	(87.9)
Indirect	**20**	**(5.9)**	12	(4.9)	1	(14.3)	0	—	3	(8.3)	4	(12.1)
Cause of death												
Trauma	**324**	**(95.6)**	239	(96.8)	6	(85.7)	15	(100.0)	35	(97.2)	29	(87.9)
Asphyxiation†	**7**	**(2.1)**	4	(1.6)	0	—	0	—	1	(2.8)	2	(6.1)
Cardiovascular	**4**	**(1.2)**	2	(0.8)	1	(14.3)	0	—	0	—	1	(3.0)
Other	**3**	**(0.9)**	2	(0.8)	0	—	0	—	0	—	1	(3.0)

* Relatedness was direct when a death was caused by the environmental force of the disaster (e.g., wind or tornado) or by the direct consequences of these forces (e.g., structural collapse or flying debris). Indirect were those deaths attributed to unsafe or unhealthy conditions, or conditions that cause a loss or disruption of usual services that contributed to the death. Unsafe or unhealthy conditions include hazardous road conditions, stressful environment resulting in myocardial infarction, and falls while escaping tornado. Disruptions of usual services include interruption of utilities or medical supplies or services (e.g., oxygen machine failure during power outage).
† Because of smoke or carbon monoxide.

Reported by

Mary Casey-Lockyer, MHS, American Red Cross. Catherine Molchan Donald, MBA, Center for Health Statistics, Alabama Dept of Health. Judy Moulder, MHS, Office of Health Statistics, Mississippi State Dept of Health. Deborah Aderhold, State Office of Vital Records, Georgia Dept of Public Health. Doreen Harris, Div of Health, Arkansas Center for Health Statistics. Gary Woodall, MS, Douglas Young, Brenton MacAloney, Gregory Carbin, National Weather Svc. Melissa Morrison, MPH, Thomas Chester, MD, Jay Roth, MPH, Career Epidemiology Field Officer Program, Office of Public Health Research; Rebecca S. Noe, MN, MPH, Amy Schnall, MPH, Amy Wolkin, MSPH, Sara Vagi, PhD, Div of Environmental Hazards and Health Effects, National Center for Environmental Health; Caitlin E. Mertzlufft, MPH, Agency for Toxic Substances and Disease Registry; David Sugerman, MD, National Center for Injury Control and Prevention; Cindy Chiu, PhD, EIS Officer, CDC. ***Corresponding contributor:*** *Rebecca S. Noe, rnoe@cdc.gov, 770-488-3437.*

Editorial Note

The southeastern United States is considered particularly vulnerable to tornado fatalities because of the high concentration of mobile homes and tornados with EF-4 and EF-5 ratings (*6*). In the tornado storms of April 25–28, 2011, nearly half of the 338 fatalities occurred in single-family homes, and approximately 40% of bodies were recovered outdoors near the impact area. Similar to other tornadoes, the leading cause of death was trauma (96%), with nearly one fifth of trauma deaths involving head injuries (*4–6,10*). Multisystem trauma and recovery of bodies outdoors was not unexpected because 90% of the deaths were associated with EF-4 or EF-5 tornadoes. Tornados reaching this strength are capable of removing a house off its foundation and debarking trees, and historically these tornadoes have been responsible for 70% of tornado-related deaths (*1,5,6*). Nearly half of the deadly tornadoes in this disaster reached EF-4 or EF-5. The precise strength and geographic extent of a tornado track are not determined until after an event; therefore, persons should prepare for the worst-case scenario when they receive a tornado warning (*1*).

Federal and state assessments conducted after this disaster found a general inadequacy of available storm shelters in the impacted areas (*1,3,9*). The overall magnitude of tornado-related deaths observed in the wake of the disaster and the high proportion of deaths occurring in single-family homes support current CDC recommendations to shelter in a safe room, take personal protective actions, and develop a disaster-preparedness plan ahead of time (*7*). In addition, emergency planning for vulnerable older adults is important; guidance on developing plans, building registries, sheltering, and caregiver preparedness are available from CDC online at http://www.cdc.gov/aging/emergency. A safe room is either an underground shelter, such as the interior part of a basement, or a specific tornado-safe room that is a hardened (e.g., concrete) aboveground structure specifically designed to meet Federal Emergency Management Administration (FEMA) criteria for providing "near-absolute protection" in extremely powerful weather events (*1,8,9*). Personal protective actions include preparing a safe room with items to provide protection of the body, including the head, and remaining informed of storm watches and warnings by using a weather band radio or other means (*1,7*). Currently, no data are available regarding the effectiveness of helmet use to prevent head injuries during a tornado. If persons choose to use helmets to protect their heads, they should know where the helmets are and have them readily accessible, because time to react might be short. Choosing to use helmets to protect the head should not be considered an alternative to seeking appropriate shelter. Tornado preparedness also should include preparation for power outages (e.g., flashlights) and plans to prevent potentially fatal hazards such as house fires, falls, and carbon monoxide exposure (*4,7,10*)

What is already known on this topic?

Known risk factors for death and injury from tornadoes are sheltering in mobile homes, proximity to the path of a tornado registering 4 or 5 on the Enhanced Fujita (EF) scale, being an older adult (aged ≥65 years), lack of accessibility to a safe room (e.g., a basement or reinforced shelter), and night-time tornado impacts. The southeastern United States is considered particularly vulnerable to tornado fatalities because of the high concentration of mobile homes and frequency of EF-4 and EF-5 tornadoes. Traumatic injury, including head injury, is the leading cause of death during tornadoes.

What is added by this report?

Although extensive public health warnings were broadcast before the tornadoes touched down, this was the third-deadliest tornado disaster in U.S. history, with 338 confirmed fatalities. Head injury was the cause of death in nearly 22% of fatalities. Approximately 47% of the fatalities were in single-family homes. A composite of the multistate deadly tornado tracks was created from the National Weather Service's tornado survey points in a geographic information system (GIS); more than 90% of the geocoded addresses were spatially linked to a deadly tornado.

What are the implications for public health practice?

To prevent tornado-related fatalities, public health messaging needs to specify what constitutes a safe room and to increase awareness that these should be used during all tornadoes. Spatial analysis of health impacts of tornadoes using GIS provides a better understanding of risk factors and the underlying characteristics of the affected population for public health preparedness and response.

The findings in this report are subject to at least two limitations. First, spatial analysis might not accurately reflect the exact location of death or tornado-relatedness because of geocoding limitations, tornado track overlap, and path estimates based on few NWS survey points (*1,6*). For this reason, the exposed population and rates were not estimated or calculated. Second, data on warnings heard, protective actions taken, and housing damage incurred are not described in this report because the Red Cross did not collect these data in all five affected states.

Given the number of fatalities and current limitations in determining a tornado's characteristics, increased awareness of the need to prepare for the worst-case scenario by pre-identifying and sheltering in an adequate tornado-safe room during a tornado remain critical to saving lives (*5,6*).

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Alcohol Use and Binge Drinking Among Women of Childbearing Age — United States, 2006–2010

Alcohol use during pregnancy is a leading preventable cause of birth defects and developmental disabilities. Alcohol-exposed pregnancies (AEPs) can lead to fetal alcohol syndrome and other fetal alcohol spectrum disorders (FASDs), which result in neurodevelopmental deficits and lifelong disability (*1*). In 2005, the Surgeon General issued an advisory urging women who are pregnant or who might become pregnant to abstain from alcohol use (*2*). *Healthy People 2020* set specific targets for abstinence from alcohol use (MICH-11.1) and binge drinking (MICH-11.2) for pregnant women (*3*). To estimate the prevalence of any alcohol use and binge drinking in the past 30 days among women aged 18–44 years, CDC analyzed 2006–2010 Behavioral Risk Factor Surveillance System (BRFSS) data. Based on their self-reports, an estimated 51.5% of nonpregnant women used alcohol, as did 7.6% of pregnant women. The prevalence of binge drinking was 15.0% among nonpregnant women and 1.4% among pregnant women. Among pregnant women, the highest prevalence estimates of reported alcohol use were among those who were aged 35–44 years (14.3%), white (8.3%), college graduates (10.0%), or employed (9.6%). Among binge drinkers, the average frequency and intensity of binge episodes were similar, approximately three times per month and six drinks on an occasion, among those who were pregnant and those who were not. Clinical practices that advise women about the dangers associated with drinking while pregnant, coupled with community-level interventions that reduce alcohol-related harms, are necessary to mitigate AEP risk among women of childbearing age and to achieve the *Healthy People 2020* objectives.

BRFSS is a state-based, random-digit–dialed telephone survey that collects information on health-related behaviors from a representative sample of civilian, noninstitutionalized adults aged ≥18 years. CDC aggregated and analyzed BRFSS data for 2006–2010 from all 50 states and the District of Columbia for 345,076 women aged 18–44 years. The median response rate among states, based on Council of American Survey and Research Organizations guidelines, ranged from 50.6% to 54.6%, and the median cooperation rate ranged from 72.1% to 76.9%.* The prevalence of any alcohol use, defined as having at least one drink of any alcoholic beverage in the past 30 days, and binge drinking, defined for women as four or more drinks on an occasion in the past 30 days, among pregnant and nonpregnant women, were estimated.† Logistic regression was used to examine, separately for pregnant and nonpregnant women, the association of selected demographic characteristics with any alcohol use and with binge drinking. The regression model adjusted for age, race/ethnicity, education, employment, and marital status. The average number of binge episodes in the past 30 days (frequency) and the average maximum number of drinks consumed on an occasion in the past 30 days (intensity) among binge drinkers were estimated with 95% confidence intervals. Because of small sample sizes, binge drinking frequency and intensity estimates for demographic subgroups among pregnant women were unreliable and are not reported. Data were weighted to state population estimates and combined to represent a nationwide estimate. Analyses were conducted using statistical software to account for the complex sampling method used in BRFSS.

The study population of 345,076 women aged 18–44 years included 13,880 (4.0%) pregnant women and 331,196 (96.0%) women who were not pregnant. Prevalence estimates for any alcohol use in the past 30 days during 2006–2010 were 7.6% among pregnant women and 51.5% among nonpregnant women (Table 1). The 2006–2010 prevalence estimates for binge drinking in the past 30 days were 1.4% among pregnant women and 15.0% among nonpregnant women.

Among pregnant women, those aged 35–44 years reported the highest prevalence of any alcohol use (14.3%) (adjusted odds ratio [AOR] = 3.3), compared with women aged 18–24 years (4.5%). Among pregnant women, the odds of reporting binge drinking were nearly two and a half times greater among those who were employed (AOR = 2.4), compared with those who were not employed, and even greater for those who were unmarried (AOR = 3.1), compared with those who were married.

Among nonpregnant women, white women reported the highest prevalence of any alcohol use (58.3%) and binge drinking (17.7%) in the past 30 days, compared with nonpregnant women in any of the other race/ethnicity groups. Compared with their Hispanic counterparts, nonpregnant white women reported higher prevalences of alcohol use (AOR = 1.9) and binge drinking (AOR = 1.8). The prevalence of reported binge drinking among nonpregnant women aged 18–24 years (20.5%) was nearly double that of nonpregnant women aged 35–44 years (11.8%).

* The response rate reflects telephone sampling efficiency and the degree of participation among eligible respondents contacted. The cooperation rate reflects the proportion of persons who completed an interview among eligible persons contacted.

† Pregnancy status was assessed by asking the woman if, to her knowledge, she was currently pregnant. BRFSS questionnaires are available at http://www.cdc.gov/brfss/questionnaires/questionnaires.htm.

TABLE 1. Estimated percentages* and adjusted odds ratios of women aged 18–44 years who reported any alcohol use or binge drinking,† by pregnancy status and selected characteristics — Behavioral Risk Factor Surveillance System, United States, 2006–2010

	Pregnant (n = 13,880)								Nonpregnant (n = 331,196)							
	Any use				Binge drinking				Any use				Binge drinking			
Characteristic	%	(95% CI)	AOR§	(95% CI)	%	(95% CI)	AOR	(95% CI)	%	(95% CI)	AOR	(95% CI)	%	(95% CI)	AOR	(95% CI)
Total	**7.6**	**(6.9–8.4)**	—	—	**1.4**	**(1.1–1.7)**	—	—	**51.5**	**(51.2–51.8)**	—	—	**15.0**	**(14.8–15.3)**	—	—
Age group (yrs)																
18–24	4.5	(3.5–5.8)	1.0	Referent	1.3	(0.7–2.3)	1.0	Referent	48.5	(47.6–49.4)	1.0	Referent	20.5	(19.8–21.3)	1.0	Referent
25–29	6.9	(5.5–8.7)	1.5	(1.0–2.2)	1.3	(0.9–2.1)	1.3	(0.7–2.4)	53.8	(53.0–54.6)	1.1	(1.0–1.1)	18.5	(17.9–19.1)	1.0	(0.9–1.0)
30–34	7.9	(6.7–9.3)	1.7	(1.2–2.5)	1.4	(0.9–2.1)	1.4	(0.7–2.8)	51.6	(50.9–52.3)	0.9	(0.9–1.0)	13.3	(12.9–13.8)	0.7	(0.6– 0.7)
35–44	14.3	(12.0–16.8)	3.3	(2.3–4.8)	1.5	(0.9–2.3)	1.5	(0.8–3.0)	52.2	(51.8–52.7)	0.9	(0.9–0.9)	11.8	(11.5–12.0)	0.6	(0.5–0.6)
Race/Ethnicity																
White, non-Hispanic	8.3	(7.4–9.3)	1.2	(0.8–1.8)	1.5	(1.1–2.0)	1.2	(0.6–2.5)	58.3	(58.0–58.7)	1.9	(1.9–2.0)	17.7	(17.4–18.0)	1.8	(1.7–1.9)
Black, non-Hispanic	7.3	(5.4–9.7)	1.0	(0.6–1.6)	0.7	(0.4–1.3)	0.4	(0.2–0.9)	43.9	(43.0–44.9)	1.1	(1.0–1.2)	9.8	(9.3–10.5)	0.8	(0.7–0.9)
Hispanic	5.7	(4.2–7.6)	1.0	Referent	1.4	(0.9–2.2)	1.0	Referent	35.7	(34.8–36.6)	1.0	Referent	10.3	(9.7–10.9)	1.0	Referent
Other	8.1	(5.5–11.6)	1.2	(0.7–2.0)	1.3	(0.5–2.9)	1.0	(0.4–2.8)	43.4	(42.0–44.7)	1.0	(0.9–1.1)	12.1	(11.2–13.1)	1.1	(1.0–1.2)
Education																
High school diploma or less	5.0	(4.0–6.2)	1.0	Referent	1.4	(0.9–2.2)	1.0	Referent	37.0	(36.4–37.6)	1.0	Referent	12.5	(12.1–12.9)	1.0	Referent
Some college	7.7	(6.3–9.4)	1.4	(1.0–2.0)	1.3	(0.9–2.1)	1.0	(0.5–1.8)	53.4	(52.8–54.0)	1.7	(1.7–1.8)	16.6	(16.1–17.0)	1.3	(1.2–1.3)
College degree	10.0	(8.7–11.3)	1.6	(1.1–2.4)	1.4	(1.0–1.9)	1.0	(0.5–1.9)	64.6	(64.1–65.1)	2.7	(2.6–2.7)	16.3	(15.9–16.7)	1.4	(1.3–1.5)
Employed																
Yes	9.6	(8.5–10.8)	1.6	(1.2–2.0)	1.8	(1.4–2.3)	2.4	(1.3–4.5)	57.9	(57.5–58.3)	1.7	(1.6–1.7)	16.7	(16.4–17.0)	1.5	(1.4–1.5)
No	5.2	(4.3–6.3)	1.0	Referent	0.8	(0.5–1.5)	1.0	Referent	41.3	(40.8–41.9)	1.0	Referent	12.4	(12.0–12.8)	1.0	Referent
Married																
Yes	7.6	(6.7–8.5)	1.0	Referent	1.0	(0.8–1.4)	1.0	Referent	52.3	(51.9–52.7)	1.0	Referent	12.0	(11.8–12.3)	1.0	Referent
No	7.6	(6.3–9.1)	1.8	(1.4–2.5)	2.1	(1.4–3.1)	3.1	(1.8–5.6)	50.6	(50.1–51.2)	1.2	(1.1–1.2)	18.9	(18.5–19.3)	1.7	(1.6–1.7)

Abbreviations: CI = confidence interval; AOR = adjusted odds ratio.
* Percentages weighted to represent the U.S. population.
† Defined as having consumed four or more drinks on an occasion at least one time in the past 30 days.
§ Model includes age, race/ethnicity, education, employment, and marital status.

Among pregnant and nonpregnant women who reported binge drinking, the estimated average frequency and intensity of binge drinking were similar, approximately three times per month and six drinks on an occasion (Figure). Among age groups of nonpregnant women, average frequency and intensity of binge episodes were highest among women aged 18–24 years (3.3 times per month and 6.7 drinks on an occasion) (Table 2). The frequency and intensity of binge drinking episodes decreased with increasing education. On average, women with a high school diploma or less reported binge drinking 3.4 times per month and 6.4 drinks on an occasion, compared with 2.5 times per month and 5.4 drinks on an occasion among college graduates. Frequency and intensity of binge drinking episodes also were greater among unmarried women (3.3 times per month and 6.4 drinks on an occasion), compared with married women (2.6 times per month and 5.4 drinks on an occasion).

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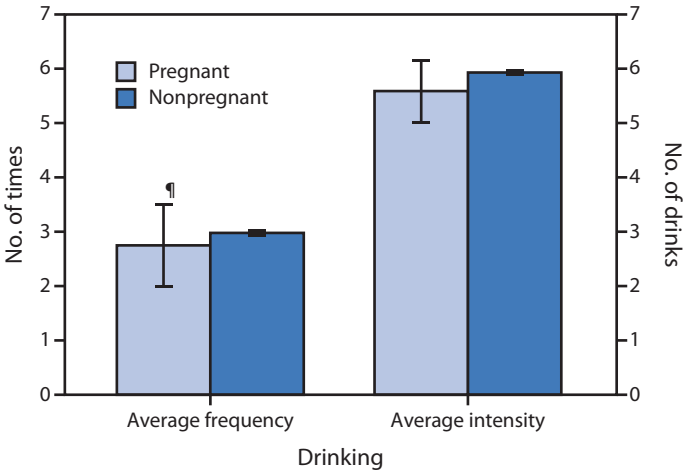
Claire M. Marchetta, MPH, Clark H. Denny, PhD, R. Louise Floyd, DSN, Nancy E. Cheal, PhD, Joseph E. Sniezek, MD. Div of Birth Defects and Developmental Disabilities, National Center on Birth Defects and Developmental Disabilities; Lela R. McKnight-Eily, PhD, Div of Population Health, National Center for Chronic Disease Prevention and Health Promotion, CDC. ***Corresponding contributor:*** *Claire M. Marchetta, vtj6@cdc.gov, 404-498-6788.*

Editorial Note

FASDs, which include fetal alcohol syndrome, alcohol-related birth defects, and alcohol-related neurodevelopmental disorder, are estimated to affect at least 1% of all births in the United States (*4*). FASDs have been associated with alcohol consumption patterns that produce high blood alcohol concentrations, such as binge drinking (*5*). Animal studies have found that binge-like drinking patterns are particularly dangerous, especially to fetal brain development, even if the total amount

FIGURE. Estimated average frequency* and intensity† of binge drinking§ among women of childbearing age who reported binge drinking in the past 30 days — Behavioral Risk Factor Surveillance System, United States, 2006–2010



* Number of times respondent reported consuming four or more drinks on an occasion in the past 30 days.
† Largest number of drinks consumed on an occasion in the past 30 days.
§ Defined as having consumed four or more drinks on an occasion at least one time in the past 30 days.
¶ 95% confidence interval.

of alcohol consumed is less than that consumed in a more continuous drinking pattern (*5*). Although the prevalence of binge drinking is much lower among pregnant women than among nonpregnant women (1.4% versus 15.0%), those who did report binge drinking in the past 30 days did so with similar frequency (average of approximately three times a month) and similar intensity (average of approximately six drinks on an occasion) to nonpregnant women. These frequency and intensity estimates for pregnant and nonpregnant women of childbearing age are similar to the findings previously reported for all adult women (*6*).

Women who binge drink in the preconception period are more likely than non–binge drinkers to continue drinking, even after becoming pregnant (*1*). Among nonpregnant binge drinkers, binge drinking prevalence, frequency, and intensity were highest among those aged 18–24 years. Alcohol screening and brief interventions (SBI) among nonpregnant women, which include short counseling sessions, feedback, advice, and goal-setting conducted by health-care providers, might be helpful for reducing alcohol misuse§ among women at risk for an AEP (*7*).

§ According to the U.S. Preventive Services Task Force, alcohol misuse includes consumption of more than seven drinks per week or more than three drinks per occasion for women and any alcohol consumption during pregnancy.

TABLE 2. Estimated average frequency* and intensity† of binge drinking§ among nonpregnant women of childbearing age who reported binge drinking in the past 30 days, by selected characteristics — Behavioral Risk Factor Surveillance System, United States, 2006–2010

	Frequency			Intensity		
Characteristic	**No.¶**	**Mean****	**(95% CI)**	**No.**	**Mean**	**(95% CI)**
Total	**47,900**	**3.0**	**(2.9–3.0)**	**45,352**	**5.9**	**(5.9–6.0)**
Age group (yrs)						
18–24	6,982	3.3	(3.1–3.4)	6,481	6.7	(6.5–6.8)
25–29	8,657	2.9	(2.7–3.1)	8,205	6.1	(5.9–6.2)
30–34	9,478	2.7	(2.6–2.8)	9,005	5.7	(5.6–5.8)
35–44	22,783	2.9	(2.8–3.0)	21,661	5.3	(5.3–5.4)
Race/Ethnicity						
White, non-Hispanic	37,653	3.0	(2.9–3.1)	35,839	6.0	(5.9–6.0)
Black, non-hispanic	3,217	3.4	(3.0–3.7)	2,964	5.3	(5.1–5.6)
Hispanic	3,700	2.7	(2.5–2.9)	3,457	5.7	(5.5–5.9)
Other	3,136	3.1	(2.8–3.4)	2,917	6.4	(6.0–6.7)
Education						
High school diploma or less	14,052	3.4	(3.3–3.5)	13,001	6.4	(6.3–6.6)
Some college	14,771	3.2	(3.0–3.3)	13,958	6.1	(6.0–6.2)
College degree	19,057	2.5	(2.4–2.6)	18,380	5.4	(5.3–5.5)
Employed						
Yes	35,660	2.9	(2.8–3.0)	33,892	5.8	(5.8–5.9)
No	12,182	3.2	(3.0–3.3)	11,408	6.1	(6.0–6.3)
Married						
Yes	23,559	2.6	(2.5–2.6)	22,553	5.4	(5.3–5.4)
No	24,265	3.3	(3.2–3.4)	22,734	6.4	(6.3–6.5)

Abbreviation: CI = confidence interval.
* Number of times respondent reported consuming four or more drinks on an occasion in the past 30 days.
† Largest number of drinks consumed on an occasion in the past 30 days.
§ Defined as having consumed four or more drinks on an occasion at least one time in the past 30 days.
¶ Unweighted sample size for nonpregnant binge drinkers.
** Weighted mean.

What is already known on this topic?

Alcohol misuse is associated with fetal alcohol spectrum disorders (FASDs), miscarriage, motor vehicle crashes, intimate partner violence, and other adverse outcomes. Alcohol use during pregnancy is a leading preventable cause of birth defects and developmental disabilities. FASDs are estimated to affect at least 1% of all births in the United States. There is no known safe level of alcohol consumption during pregnancy and binge drinking is a high-risk pattern of alcohol consumption.

What is added by this report?

Based on 2006–2010 data from the Behavioral Risk Factor Surveillance System, among nonpregnant women aged 18–44 years, 51.5% used alcohol in the past 30 days, as did 7.6% of pregnant women of the same age. The prevalence of binge drinking in the past 30 days was estimated to be 15.0% among nonpregnant women and 1.4% among pregnant women. Among binge drinkers, pregnant and nonpregnant women drank with similar frequency and intensity.

What are the implications for public health practice?

Alcohol consumption (any use and binge drinking) among pregnant women is a public health concern. Public health interventions, such as alcohol screening and brief interventions and community-level policy interventions (e.g., increasing alcohol excise taxes and limiting alcohol outlet density) can help reduce alcohol misuse by pregnant and nonpregnant women of childbearing age.

For 2001–2005, CDC previously estimated binge drinking at 1.8% among pregnant women and 12.6% among nonpregnant women (*8*). For the 2006–2010 period, estimated binge drinking among pregnant women was lower (1.4%), but higher among nonpregnant women (15.0%). Until 2004, binge drinking was defined for men and women as five or more drinks on an occasion. In 2004, the National Institute on Alcohol Abuse and Alcoholism changed the definition of binge drinking for women to four or more drinks on an occasion to account for physiologic differences between men and women that affect the absorption of alcohol. BRFSS adopted the new sex-specific definition in 2006 (*9*). This definition change sets a lower threshold for binge drinking among women, and therefore has the effect of increasing the prevalence estimate (*9*). A possible reason this increase is not observed in the pregnant population for the 2006–2010 data might be a change in the BRFSS questionnaire. Beginning in 2006, pregnancy status was asked before the alcohol consumption questions; the order was reversed in earlier questionnaires. Women who already have disclosed that they are pregnant might be less likely to report alcohol use in the past 30 days. Regardless of the binge drinking definition change and questionnaire change, these results indicate that binge drinking during pregnancy continues to be a concern.

The findings in this report are subject to at least three limitations. First, BRFSS data are self-reported and subject to misclassification, recall, and social desirability biases, which can lead to underestimates of alcohol consumption. Second, the prevalence of households without landline telephones and only cellular telephones is increasing, which excludes persons from landline-only surveys such as BRFSS who only use cellular telephones and might be more likely to consume alcohol and binge drink. BRFSS will include data for respondents with cellular telephones beginning with the 2011 data set. Finally, BRFSS also does not collect information from persons living in institutional settings (e.g., on college campuses), and so data might not be representative of those populations.

Pregnant and nonpregnant women of childbearing age who misuse alcohol might benefit from public health interventions. SBI and community level policy interventions, such as increased alcohol excise taxes and limiting alcohol outlet density[¶] might be effective in reducing alcohol misuse among women and help to achieve the *Healthy People* 2020 goals of 98.3% abstinence from any alcohol use and 100% abstinence from binge drinking among pregnant females aged 15–44 years. Alcohol SBI is an evidence-based approach to address alcohol misuse in adults, including pregnant women, that has been recommended by the U.S. Preventive Services Task Force (*7*). CDC currently supports FASD Regional Training Centers to provide training to medical and allied health students, residents, and practitioners regarding prevention, identification, and management of FASDs. This includes teaching how to screen and intervene with women at risk for an AEP. CDC also is developing a guide for implementing SBI in primary-care settings and promoting public health efforts based on adaptations of Project CHOICES (Changing High-Risk Alcohol Use and Improving Contraception Effectiveness Study), an effective intervention that uses motivational interviewing to aid women of reproductive age in reducing their risk for an AEP (*10*). Widespread adoption of SBI in primary care settings, including obstetricians' offices, and community interventions might help reduce FASDs and other adverse pregnancy outcomes.

[¶] These community interventions are recommended by the Task Force for Community Preventive Services. Additional information available at http://www.thecommunityguide.org/alcohol/index.html.

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Notes from the Field

Tuberculosis Cluster Associated with Homelessness — Duval County, Florida, 2004–2012

Despite a decrease in incidence of tuberculosis (TB) in Duval County, Florida, from 102 cases (11.2 per 100,000 population) in 2008 to 71 cases (8.2 per 100,000) in 2011,* analysis of *Mycobacterium tuberculosis* genotyping data revealed a substantial increase in the percentage of TB cases with the same genotype.† That percentage increased from 27% (10 of 37) of genotyped cases in 2008 to 51% (30 of 59) of genotyped cases in 2011 (Florida Department of Health, unpublished data, 2012). During this period, the percentage of patients with this genotype who were homeless or who abused substances also increased. Because of concern over potential ongoing TB transmission involving these hard-to-reach populations, the Duval County Health Department, Florida Department of Health, and CDC conducted an investigation during February 15–March 13, 2012. As of March 13, review of medical records and interviews with TB patients had identified 99 cases related to the cluster based on matching genotype results and epidemiologic links (48 cases), matching genotype only (22), epidemiologic links only (22), or common social risk factors for TB (e.g., homelessness, incarceration, or substance abuse within 1 year of TB diagnosis) and suspected epidemiologic links (seven). The first known case with a matching genotype occurred in 2004.

Among the 99 TB cases during 2004–2012, a total of 96 (97%) patients were U.S.-born; 78 (79%) were male; 76 (77%) were black; 78 (79%) had a history of homelessness, incarceration, or substance abuse (i.e., alcohol or illicit substances); and 43 (43%) had been homeless within 1 year of TB diagnosis. Three patients were children aged <5 years. Twenty patients had known human immunodeficiency virus infection; 13 patients, all with comorbidities, had died. Site visits and review of electronic databases that track use of Duval County homeless services and incarceration found that the TB patients had stayed in several different homeless shelters and in a local jail. In addition, social network analysis identified one particular shelter and an outpatient mental health facility that serves the homeless community as the sites of concern for TB transmission during 2010–2012.

Duval County Health Department organized the screening of approximately 2,300 persons; approximately 2,100 additional persons are considered a high priority for TB screening because of recent exposure in a congregate setting to a patient with sputum smear-positive TB (*1*). To control ongoing TB transmission and detect and treat additional cases of active TB disease or latent TB infection, Duval County public health workers are finding and evaluating high-priority contacts and conducting TB evaluations at sites with evidence of recent TB transmission. Long-term control measures at homeless shelters will include enhanced infection control programs involving TB education, respiratory hygiene, periodic systematic TB screening of clients and workers, and environmental controls.

Genotyping data, combined with epidemiologic investigation, enabled recognition of this cluster and subsequent understanding of chains of TB transmission. Newly available electronic data systems in Duval County that document use of homeless services, stays at homeless shelters, and incarceration at a local jail also were critical in identifying likely transmission sites. Although TB incidence continues to decline in Florida and nationwide, outbreaks still occur among homeless persons, requiring sustained and aggressive control measures (*2,3*). Prompt identification of TB patients through symptom screening, radiographic screening, and testing for TB infection, along with evaluation of contacts of TB patients, can be difficult in hard-to-reach populations but is crucial to achieving the national goal of TB elimination (*4*).

Reported by

Vincy Samuel, MPH, Cynthia Benjamin, Ozzie Renwick, Aaron Hilliard, PhD, Duval County Health Dept; Sherrie Arnwine, Debra Spike, Jose Zabala, MHSA, Kateesha McConnell, MPH, Max Salfinger, MD, Florida Dept of Health. Kiren Mitruka, MD, Tracie Gardner, PhD, Div of TB Elimination, National Center for HIV/AIDS, Viral Hepatitis, STD, and TB Prevention. Emma Johns, CDC Experience Fellow. Robert Luo, MD, Madsen Beau de Rochars, MD, Raymund Dantes, MD, EIS officers, CDC. ***Corresponding contributor:*** *Max Salfinger, max_salfinger@doh.state.fl.us, 850-245-4350.*

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Robert Harmon, MD, Duval County Health Dept; Richard Hopkins, MD, Florida Dept of Health. Gail Burns-Grant, Dan Ruggiero, MPS, Jimmy Keller, DHSc, Div of TB Elimination, National Centers for HIV/AIDS, Viral Hepatitis, STD, and TB Prevention, CDC.

* Additional information available at http://www.doh.state.fl.us/disease_ctrl/tb/trends-stats/trends.html.

† Spoligotype 777776777760601, and 12-locus mycobacterial interspersed repetitive units–variable number of tandem repeats (MIRU-VNTR) pattern 224325143323.

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Notes from the Field

Severe Varicella in an Immunocompromised Child Exposed to an Unvaccinated Sibling with Varicella — Minnesota, 2011

Varicella usually is a self-limited disease but can result in serious complications (e.g., encephalitis, pneumonia, sepsis, hemorrhagic varicella, and death), especially among immunocompromised persons. Implementation of the varicella vaccination program in the United States, beginning in 1995 ,has led to declines of >95% in varicella-related hospitalizations and deaths among populations routinely vaccinated (*1*).

On December 13, 2011, the Minnesota Department of Health was notified of varicella in a girl, aged 3 years, admitted to a hospital after a 2-day history of fever of 102.7°F (39.3°C) and an extensive maculopapulovesicular rash (>500 skin lesions) with vesicles in the mouth and throat. The child received weekly immunosuppressive therapy with methotrexate (12.5 mg) for juvenile rheumatoid arthritis diagnosed at age 18 months. Neither she nor her younger sibling, aged 21 months, had received a first dose of varicella vaccine (routinely recommended at age 12–15 months). Their parents refused vaccination because of personal beliefs. The parents reported varicella in the younger sibling 2 weeks before her older sister was admitted. The older sister had not received prophylactic varicella zoster immune globulin (VariZIG); however, her parents monitored her for varicella symptoms.

The patient was treated with intravenous acyclovir for 7 days. Her fever resolved, and no new skin lesions appeared after hospital day 2. Moderate thrombocytopenia (platelet count: 103,000/μL; normal: 150,000–450,000/μL) resolved by hospital day 6. No other substantial laboratory abnormalities or signs of organ dysfunction were reported. She was discharged in good condition on hospital day 8.

Varicella vaccination is not recommended for children with congenital or acquired T-lymphocyte immunodeficiency (except certain categories of human immunodeficiency virus–infected children), including children receiving long-term immunosuppressive therapy, because of risk for complications from live vaccine virus infection (*2*). However, these patients are at high risk for severe or fatal varicella and depend on indirect protection through high levels of varicella immunity among the general population, and especially among their close contacts, to prevent exposure. Varicella vaccination of household contacts of immunocompromised patients is recommended if contacts lack evidence of varicella immunity. If exposure to varicella zoster virus occurs, postexposure prophylaxis with VariZIG (available through an Investigational New Drug protocol*) is recommended for immunocompromised patients and other persons at high risk for severe disease who lack evidence of varicella immunity (*2*). In 2011, the period after exposure during which a contact may receive VariZIG was extended from 96 hours to 10 days; VariZIG should be administered as soon as possible after exposure (*3*).

Clinicians should remain vigilant for opportunities to prevent varicella through vaccination of household members of immunocompromised patients and administration of passive immunoprophylaxis (VariZIG) for up to 10 days after a susceptible, immunocompromised patient is exposed. Resources to help clinicians discuss vaccination with hesitant parents are available at http://www.cdc.gov/vaccines/spec-grps/hcp/conv-materials.htm.

Reported by

Vicki Buttery, MS, Lynn Bahta, Claudia Miller, MS, Minnesota Dept of Health. Mona Marin, MD, Div of Viral Diseases, National Center for Immunization and Respiratory Diseases; Sarah K. Kemble, MD, EIS Officer, CDC. ***Corresponding contributor:*** *Sarah K. Kemble, skemble@cdc.gov, 312-942-2061.*

* Additional information available at http://www.fffenterprises.com/products/varizig.aspx.

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Erratum

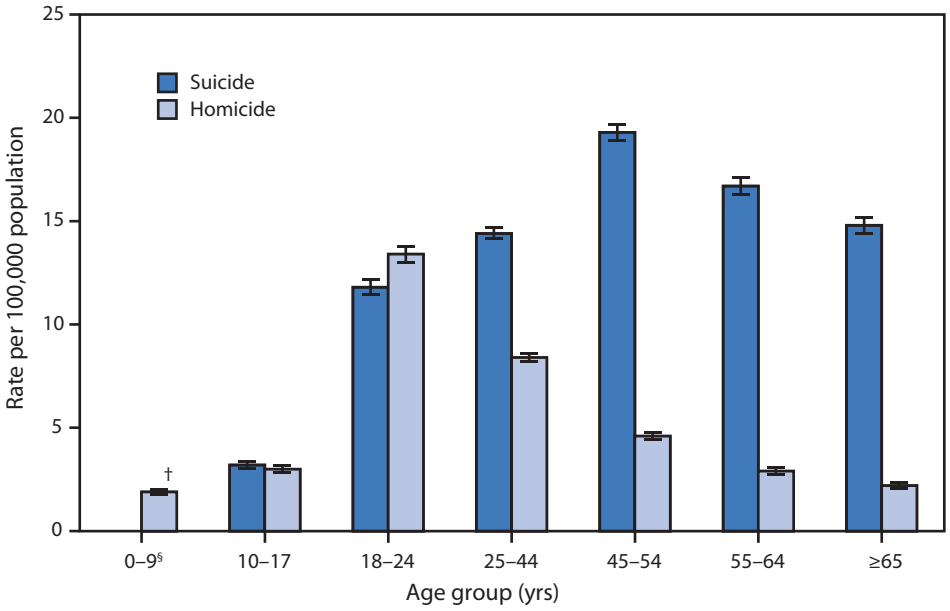
Vol. 61, No. RR-3

In the *MMWR Recommendations and Reports* "Updated CDC Recommendations for the Management of Hepatitis B Virus–Infected Health-Care Providers and Students," an error occurred on page 4 in the second sentence of the section headed "Treatments for Chronic Hepatitis B Infection." The sentence should read, "Currently, seven therapeutic agents are approved by the Food and Drug Administration for the treatment of chronic hepatitis B, including two formulations of interferon (interferon alpha and pegylated interferon) and five nucleoside or nucleotide analogs (lamuvidine, telbivudine, **adefovir,** entecavir, and tenofovir)."

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Suicide and Homicide Rates,* by Age Group — United States, 2009



* Per 100,000 population in age group. Suicides are coded as *U03, X60–X84, and Y87.0, and homicides are coded as *U01–*U02, X85–Y09, and Y87.1 according to the *International Classification of Diseases, 10th Revision*.
† 95% confidence interval.
§ Suicide data for persons aged 0–9 years are suppressed based on a child's inability to form and understand suicidal intent and consequences.

In 2009, the age-adjusted suicide rate for the total population (11.8 per 100,000 population) was approximately twice as high as the age-adjusted homicide rate (5.5). Persons aged 18–24 years had the highest rate of homicide in 2009, whereas persons aged 45–54 years had the highest rate of suicide. The suicide rate was higher than the homicide rate among those aged ≥25 years, and this difference increased with age. For persons aged 25–44 years, the rate of suicide was nearly twice the rate of homicide, whereas for those aged ≥65 years, the rate of suicide was nearly seven times the homicide rate.

Sources: National Vital Statistics System mortality data. Available at http://www.cdc.gov/nchs/deaths.htm.

US Department of Health and Human Services, Office of Disease Prevention and Health Promotion. Healthy people 2020. Washington, DC: US Department of Health and Human Services; 2012. Available at http://www.healthypeople.gov.

Reported by: Kimberly Hurvitz, MHS, ifo7@cdc.gov, 301-458-4756; Deepthi Kandi.

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