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Motor Vehicle Traffic-Related Pedestrian Deaths — United States, 2001–2010

Motor vehicle traffic crashes are the leading cause of unintentional injury-related death in the United States, resulting in 33,687 deaths in 2010 (*1*). Pedestrian travel makes up 10.5% of all trips (i.e., any travel from one address to another) taken in the United States, and pedestrians represent 13% of all motor vehicle traffic-related deaths (*1*,*2*). To determine traffic-related pedestrian death rates by sex, age group, race/ethnicity, and urbanization level, CDC analyzed 2001–2010 data from the National Vital Statistics System (NVSS). The results of that analysis indicated that the overall, annualized, age-adjusted traffic-related pedestrian death rate was 1.58 deaths per 100,000 population. Persons aged ≥75 years and those categorized as American Indian/Alaska Native (AI/AN) had the highest death rates, and age group differences varied by race/ethnicity. The results suggest that the overall pedestrian death rate could increase with the aging and growing racial/ethnic diversity of the U.S. population. The U.S. Census Bureau projects that the number of persons aged ≥75 years will more than double, from approximately 18 million in 2011 (6% of the U.S. population) to 44 million in 2040 (12% of the population); minority racial/ethnic populations are projected to increase from 116 million in 2010 (37% of the population) to 186 million in 2040 (49% of the population).* Strategies to prevent pedestrian deaths should include consideration of the needs of older adults and cultural differences among racial/ethnic populations.

NVSS data were accessed through CDC WONDER, which provides customized reports of mortality data, and information on other health outcomes and risk factors (e.g., birth data and sexually transmitted disease morbidity).† NVSS collects death certificate data from vital statistics offices in all 50 states and the District of Columbia.§ Motor vehicle traffic-related pedestrian deaths were defined as any deaths for which the underlying cause recorded on death certificates was one of the following *International Classification of Diseases, 10th Revision* codes: V02–V04 (.1,.9) or V09.2.¶ Pedestrian deaths and annualized death rates per 100,000 population for the years 2001–2010 were examined by sex, age group, race/ethnicity, and urbanization level. Annualized death rates for sex, race/ethnicity, and urbanization level were age-adjusted to the 2000 standard U.S. population. Traffic-related pedestrian death counts less than 20 (and the associated rates) were not reported for racial/ethnic populations because of concerns regarding statistical reliability and data confidentiality. However, such counts were included in the statistics for all pedestrians combined.

Race/ethnicity was coded into five mutually exclusive categories: white, black, AI/AN, Asian/Pacific Islander (A/PI), and Hispanic. All persons categorized in the first four groups were non-Hispanic. Persons categorized as Hispanic might be of any race. Urbanization was categorized into six levels of area: large central metro, large fringe metro, medium metro, small metro, micropolitan (nonmetro), and noncore (nonmetro).**

* Population estimates and projections available at http://www.census.gov.
† Available at http://wonder.cdc.gov.
§ Additional information available at http://www.cdc.gov/nchs/deaths.htm.
¶ Includes pedestrians injured in traffic collisions with cars, pick-up trucks, vans, two- or three-wheeled motor vehicles, heavy transport vehicles, buses, and other motor vehicles.

** Additional information available at http://wonder.cdc.gov/wonder/help/cmf/urbanization-methodology.html.

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During 2001–2010, a total of 47,392 pedestrians (32,873 males and 14,519 females) died from traffic crashes (Table 1). The overall, annualized age-adjusted traffic-related pedestrian death rate was 1.58 deaths per 100,000 population. The age-adjusted death rate for males (2.29) was 2.5 times the rate for females (0.92). Pedestrian death rates increased with age. For males, death rates were highest among those aged ≥85 years (6.35), followed by those aged 75–84 years (4.53); rates were lowest among those aged 0–14 years (0.83), followed by those aged 15–24 years (1.98). For females, death rates were highest among those aged 75–84 years (2.43), followed by those aged ≥85 years (2.16); rates were lowest among those aged 0–14 years (0.43), followed by those aged 25–34 years (0.72) and 15–24 years (0.78).

AI/ANs, among both males (7.73) and females (2.22), had the highest annualized, age-adjusted traffic-related pedestrian death rates of all races/ethnicities (Table 1). For males, Hispanics and blacks had the next highest death rates (3.93 and 3.73, respectively), followed by A/PIs (1.96). For females, A/PIs had the second highest death rate (1.46), followed by blacks (1.31) and Hispanics (1.27). Among both males (1.78) and females (0.79), whites had the lowest pedestrian death rates. By urbanization level, among both males (2.90) and females (1.23), those living in large central metro areas had the highest pedestrian death rates.

For males in the 15–24, 25–34, 35–44, and 45–54 year age groups, racial/ethnic disparity patterns generally were similar (Figure). In each of these age groups, the highest death rates were among AI/ANs (range: 8.13–11.72), followed by blacks (2.29–5.97) and Hispanics (2.61-4.60). Whites (range: 1.66–2.28) and A/PIs (0.70–1.36) had the lowest death rates. For males aged 75–84 and ≥85 years, Hispanic (11.05 and 14.70, respectively) and A/PI (12.30 and 20.53, respectively) death rates were statistically greater than the rates for whites (3.61 and 5.41, respectively) and blacks (6.78 and 6.95, respectively).

Among females, AI/ANs also had the highest death rates for each of the age groups 15–24, 25–34, 35–44, and 45–54 years (Figure). Across those age groups, the death rate for AI/ANs ranged from 2.29 to 4.17, and was followed by the rate for blacks (range: 0.96–1.88). Hispanics (range: 0.62–1.15), whites (0.68–0.86), and A/PIs (0.55–0.97) had similar death rates. For females aged 75–84 and ≥85 years, Hispanic (5.33 and 4.03, respectively) and A/PI (8.82 and 6.87, respectively) death rates were statistically greater than the rates for whites (2.06 and 2.02, respectively) and blacks (1.94 and 1.36, respectively).

Pedestrian death rates generally increased with age across all six urbanization levels (Table 2). In large central metro areas, those aged 35–44 years (2.08), 45–54 years (2.60), 55–64 years (2.60), 65–74 years (3.36), 75–84 years (5.19), and ≥85 years (5.24) had statistically higher death rates than those in the same age groups at other urbanization levels. By race/ethnicity, in large central metro areas, death rates for whites (1.57) and Hispanics (2.74) were statistically greater than in nonmetro areas (whites, micropolitan: 1.18 and whites, noncore: 1.13; Hispanics, micropolitan: 2.26 and Hispanics, noncore: 1.89). However, the rates for other races/ethnicities, notably AI/ANs,

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TABLE 1. Number of motor vehicle traffic-related pedestrian deaths and annualized death rates,* by sex and selected characteristics — National Vital Statistics System, United States, 2001–2010

Characteristic	Males			Females		
	No.	Annualized death rate	(95% CI)	No.	Annualized death rate	(95% CI)
Overall	**32,873**	**2.29**	**(2.26–2.31)**	**14,519**	**0.92**	**(0.90–0.93)**
Age group (yrs)						
0–14	2,496	0.83	(0.80–0.86)	1,353	0.43	(0.41–0.46)
15–24	4,308	1.98	(1.92–2.04)	1,616	0.78	(0.74–0.82)
25–34	4,349	2.18	(2.12–2.24)	1,428	0.72	(0.68–0.76)
35–44	5,399	2.51	(2.44–2.58)	2,122	0.98	(0.94–1.02)
45–54	6,278	3.00	(2.93–3.07)	2,250	1.04	(1.00–1.08)
55–64	3,958	2.64	(2.56–2.72)	1,641	1.02	(0.97–1.07)
65–74	2,658	2.96	(2.85–3.07)	1,507	1.43	(1.36–1.50)
75–84	2,399	4.53	(4.35–4.71)	1,866	2.43	(2.32–2.54)
≥85	953	6.35	(5.95–6.75)	719	2.16	(2.00–2.32)
Race/Ethnicity						
White	17,839	1.78	(1.76–1.81)	8,659	0.79	(0.78–0.81)
Black	6,063	3.73	(3.63–3.83)	2,484	1.31	(1.26–1.36)
Hispanic	6,809	3.93	(3.82–4.04)	2,120	1.27	(1.22–1.33)
A/PI	966	1.96	(1.83–2.10)	914	1.46	(1.36–1.56)
AI/AN	891	7.73	(7.20–8.26)	272	2.22	(1.95–2.49)
Urbanization level of area						
Large central metro	11,843	2.90	(2.85–2.96)	5,558	1.23	(1.19–1.26)
Large fringe metro	6,564	1.90	(1.85–1.94)	3,031	0.81	(0.78–0.84)
Medium metro	6,460	2.26	(2.21–2.32)	2,719	0.88	(0.84–0.91)
Small metro	2,796	2.02	(1.95–2.10)	1,190	0.82	(0.77–0.86)
Micropolitan (nonmetro)	3,138	2.08	(2.00–2.15)	1,272	0.82	(0.77–0.86)
Noncore (nonmetro)	2,072	2.16	(2.07–2.26)	749	0.75	(0.70–0.81)

Abbreviations: CI = confidence interval; A/PI = Asian/Pacific Islander; AI/AN = American Indian/Alaska Native.
* Per 100,000 population. Death rates overall and by race/ethnicity and urbanization level are age-adjusted.

did not follow this pattern. For example, the pedestrian death rate for AI/ANs living in noncore (nonmetro) areas (7.04) was approximately twice that for AI/ANs living in large central metro areas (3.58).

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Editorial Note

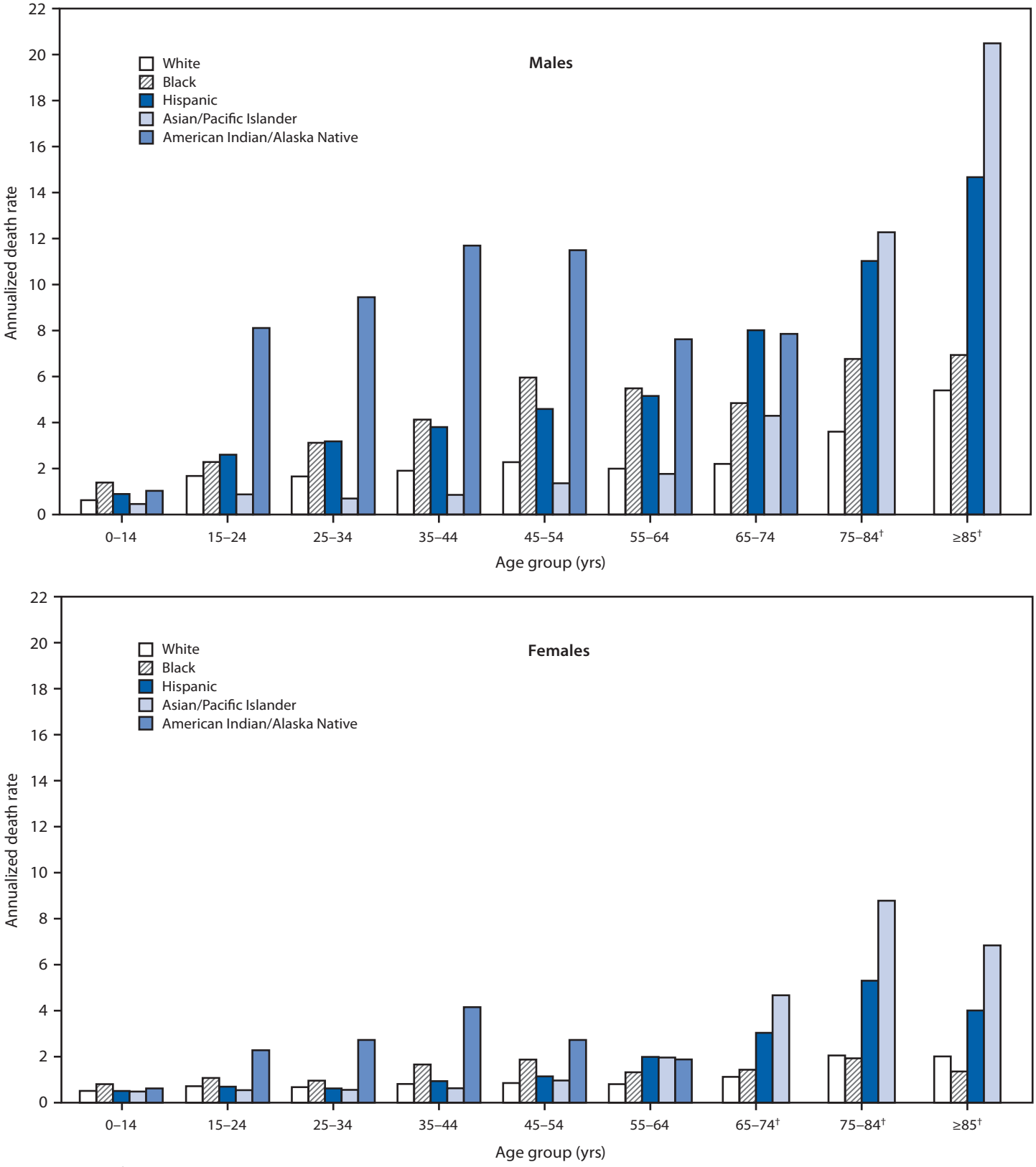
This report examines annualized motor vehicle traffic-related pedestrian death rates by key sociodemographic variables. The results indicated that, among racial/ethnic populations, AI/ANs had the highest traffic-related pedestrian death rates, and by age group, persons aged ≥75 years had the highest rates. Age-related patterns in death rates varied by race/ethnicity.

These results support those from previous research showing that males consistently have higher traffic-related pedestrian death rates than females (*3*). Recent research has shown that, on average, males and females walk similar distances, and although males have a slightly higher risk for being involved in a collision as a pedestrian, the observed differences have been found largely driven by a higher case-fatality rate among males than females (*4*). Some researchers have speculated that males exhibit riskier pedestrian behaviors or walk in more dangerous settings, but little research has explored the differences by sex in pedestrian death rates.

Among both males and females, pedestrian death rates generally increased with age. The highest death rates for both sexes were observed among those aged 75–84 and ≥85 years. Studies of travel behavior have found that older adults take fewer walking trips and walk, on average, fewer miles per year than younger persons (*2*); however, when struck, older adult pedestrians are more likely than younger adults to die from their injuries (*5*). Higher prevalence of chronic disease, disability, and frailty among older adults might contribute to these higher case-fatality rates. In addition, age-related declines in cognitive functioning, vision, and physical functioning might place older adult pedestrians at greater risk for being struck by a vehicle. For example, older adults take longer than younger adults to cross roadways (*6*).

Previous research also has found that certain racial/ethnic populations are disproportionally affected by pedestrian crashes (*7*). The current study supports these differences, including the fact that among all ages combined, AI/ANs had higher death rates than persons in other racial/ethnic populations. The

FIGURE. Annualized motor vehicle traffic-related pedestrian death rates* for males and females, by age group and race/ethnicity — National Vital Statistics System, United States, 2001–2010



* Per 100,000 population.
† Data not shown for American Indian/Alaska Natives in these age groups because of small numbers.

TABLE 2. Number of motor vehicle traffic-related pedestrian deaths and annualized death rates,* by urbanization level of area, age group, and race/ethnicity — National Vital Statistics System, United States, 2001–2010

Characteristic	Large central metro		Large fringe metro		Medium metro		Small metro		Micropolitan (nonmetro)		Noncore (nonmetro)	
	No.	Annualized death rate (95% CI)	No.	Annualized death rate (95% CI)	No.	Annualized death rate (95% CI)	No.	Annualized death rate (95% CI)	No.	Annualized death rate (95% CI)	No.	Annualized death rate (95% CI)
Overall	**17,401**	**2.01 (1.98–2.04)**	**9,595**	**1.34 (1.31–1.37)**	**9,179**	**1.52 (1.49–1.55)**	**3,986**	**1.38 (1.34–1.43)**	**4,410**	**1.42 (1.38–1.46)**	**2,821**	**1.47 (1.41–1.52)**
Age group (yrs)												
0–14	1,296	0.72 (0.68–0.76)	724	0.50 (0.46–0.54)	810	0.65 (0.60–0.69)	366	0.64 (0.57–0.70)	405	0.67 (0.61–0.74)	248	0.69 (0.60–0.78)
15–24	1,821	1.44 (1.37–1.51)	1,292	1.38 (1.30–1.46)	1,155	1.32 (1.24–1.40)	561	1.22 (1.12–1.32)	631	1.42 (1.31–1.53)	464	1.86 (1.69–2.03)
25–34	2,044	1.51 (1.44–1.58)	1,197	1.30 (1.23–1.37)	1,104	1.44 (1.36–1.52)	499	1.39 (1.27–1.51)	552	1.54 (1.41–1.67)	381	1.78 (1.60–1.96)
35–44	2,722	2.08 (2.00–2.16)	1,476	1.31 (1.24–1.38)	1,496	1.79 (1.70–1.88)	629	1.64 (1.51–1.77)	737	1.80 (1.67–1.93)	461	1.78 (1.62–1.94)
45–54	3,123	2.60 (2.51–2.69)	1,726	1.59 (1.51–1.67)	1,715	2.03 (1.93–2.13)	772	1.94 (1.80–2.08)	752	1.71 (1.59–1.83)	440	1.52 (1.38–1.66)
55–64	2,176	2.60 (2.49–2.71)	1,177	1.56 (1.47–1.65)	1,036	1.66 (1.56–1.76)	446	1.48 (1.34–1.62)	470	1.34 (1.22–1.46)	294	1.22 (1.08–1.36)
65–74	1,702	3.36 (3.20–3.52)	819	1.85 (1.72–1.98)	769	1.94 (1.80–2.08)	303	1.55 (1.38–1.72)	355	1.49 (1.34–1.64)	217	1.27 (1.10–1.44)
75–84	1,794	5.19 (4.95–5.43)	841	2.87 (2.68–3.06)	778	2.91 (2.71–3.11)	286	2.23 (1.97–2.49)	353	2.29 (2.05–2.53)	213	1.97 (1.71–2.23)
≥85	681	5.24 (4.85–5.63)	331	3.01 (2.69–3.33)	295	3.01 (2.67–3.35)	119	2.52 (2.07–2.97)	145	2.54 (2.13–2.95)	101	2.46 (1.98–2.94)
Race/Ethnicity												
White	7,335	1.57 (1.53–1.61)	6,151	1.14 (1.11–1.17)	5,489	1.25 (1.21–1.28)	2,676	1.20 (1.15–1.24)	2,998	1.18 (1.13–1.22)	1,849	1.13 (1.08–1.18)
Black	3,892	2.57 (2.49–2.65)	1,548	2.01 (1.90–2.11)	1,460	2.38 (2.26–2.51)	579	2.19 (2.00–2.37)	652	2.60 (2.39–2.80)	416	2.62 (2.37–2.87)
Hispanic	4,715	2.74 (2.65–2.83)	1,430	2.27 (2.13–2.41)	1,703	2.64 (2.50–2.78)	470	2.10 (1.89–2.31)	432	2.26 (2.02–2.50)	179	1.89 (1.59–2.19)
A/PI	1,095	1.83 (1.72–1.94)	344	1.34 (1.18–1.49)	298	1.54 (1.36–1.71)	55	1.47 (1.04–2.01)	77	1.72 (1.36–2.16)	—†	—
AI/AN	142	3.58 (2.97–4.18)	71	2.55 (1.96–3.25)	180	4.18 (3.53–4.82)	186	6.24 (5.31–7.16)	226	5.09 (4.41–5.77)	358	7.04 (6.29–7.79)

Abbreviations: CI = confidence interval; A/PI = Asian/Pacific Islander; AI/AN = American Indian/Alaska Native.
* Per 100,000 population. Death rates overall and by race/ethnicity are age-adjusted.
† Data not shown because of small numbers.

What is already known on this topic?

Motor vehicle traffic crashes are the leading cause of unintentional injury-related death in the United States. Pedestrians are particularly vulnerable road users and disproportionally represented in motor vehicle traffic deaths.

What is added by this report?

Adults aged ≥75 years and American Indians/Alaska Natives had the highest traffic-related pedestrian death rates in the United States during 2001–2010. Age-related patterns in traffic-related pedestrian death rates differed by race/ethnicity.

What are the implications for public health practice?

The overall pedestrian death rate could increase given the aging and growing racial/ethnic diversity of the U.S. population. Strategies to prevent traffic-related pedestrian deaths should consider the needs of older adults as well as persons of different races and ethnicities.

study further found that racial/ethnic patterns in pedestrian death rates differed across age groups. Research findings are mixed regarding why certain racial/ethnic populations have higher death rates. A report on 2006 U.S. traffic fatality data showed that higher percentages of AI/AN pedestrians and pedalcyclists who died in motor vehicle traffic crashes had some level of alcohol or a blood alcohol concentration of ≥0.8 g/dL, compared with pedestrians and pedalcyclists of other races/ethnicities (*7*). Other research has shown that increased risks remain for certain minority populations, even after controlling for lower socioeconomic status, increased exposure to traffic, and increased use of alcohol (*8*). Additional research is needed to understand the factors that place certain racial/ethnic populations at increased risk for pedestrian death, and the patterns in racial/ethnic differences by age group.

Approximately three fourths of all pedestrian deaths in 2010 occurred in urban areas (*3*). Higher pedestrian death rates in urban areas are, at least in part, a result of more concentrated vehicle and pedestrian activity in these areas. The current study found that for many age groups and racial/ethnic populations, patterns in pedestrian death rates by level of urbanization were similar to those for overall pedestrian death rates and further found that the differences in pedestrian deaths when comparing large central metro and noncore (nonmetro) areas were most pronounced among adults ≥65 years. In contrast, death rates among AI/ANs were higher in nonmetro areas than in large central metro areas, which is consistent with previous research (*9*).

The findings in this report are subject to at least five limitations. First, vehicle, driver, and roadway characteristics (e.g., vehicle speed, driver alcohol use, and traffic density) that are known risk factors for crash-related deaths were not available from NVSS. Second, the small numbers of pedestrian deaths among certain groups (i.e., AI/AN females aged ≥65 years, AI/AN males aged ≥75 years) prevented estimation of death rates among these groups. Third, the urbanization level variable is defined as the county of the person's legal residence, not the county where the crash occurred. Fourth, for some motor vehicle traffic-related deaths, the road user type (e.g., occupant, pedestrian, pedalcyclist, or motorcyclist) was unknown; therefore, pedestrian death rates might be underestimated. Finally, because NVSS data are extracted from death certificates, some racial misclassification is likely. This can result in underestimated death rates for some minority populations, particularly AI/ANs (*10*).

Approximately 4,000 pedestrians die from crash-related injuries each year in the United States, and certain populations are disproportionately affected (*1*). Addressing the risks that pedestrians of different ages, sexes, and races/ethnicities face in various settings requires a multifaceted approach. *Pedestrian Safety: a Road Safety Manual for Decision-Makers and Practitioners* will be released by the World Health Organization to coincide with Global Road Safety Week (May 6–12, 2013), which this year focuses on pedestrian safety. The manual will include effective strategies for reducing pedestrian deaths such as roadway engineering improvements (e.g., installing and/or upgrading crosswalks, sidewalks, and raised medians); slowing vehicle speeds by implementing traffic calming measures (e.g., speed humps); enforcing speeding, distracted driving, and pedestrian-right-of-way laws; creating pedestrian safety zones and streets designated for walking; and improving mass transit route design and access. Many of these strategies can help prevent pedestrian deaths among high-risk older adults, but additional approaches that are specific to older adults also might be needed (e.g., longer pedestrian walk signals). Research on how to effectively tailor strategies (e.g., community outreach and media campaigns) to minority populations also is needed.

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Incidence and Trends of Infection with Pathogens Transmitted Commonly Through Food — Foodborne Diseases Active Surveillance Network, 10 U.S. Sites, 1996–2012

Foodborne diseases are an important public health problem in the United States. The Foodborne Diseases Active Surveillance Network* (FoodNet) conducts surveillance in 10 U.S. sites for all laboratory-confirmed infections caused by selected pathogens transmitted commonly through food to quantify them and monitor their incidence. This report summarizes 2012 preliminary surveillance data and describes trends since 1996. A total of 19,531 infections, 4,563 hospitalizations, and 68 deaths associated with foodborne diseases were reported in 2012. For most infections, incidence was highest among children aged <5 years; the percentage of persons hospitalized and the percentage who died were highest among persons aged ≥65 years. In 2012, compared with the 2006–2008 period, the overall incidence of infection† was unchanged, and the estimated incidence of infections caused by *Campylobacter* and *Vibrio* increased. These findings highlight the need for targeted action to address food safety gaps.

FoodNet conducts active, population-based surveillance for laboratory-confirmed infections caused by *Campylobacter*, *Cryptosporidium*, *Cyclospora*, *Listeria*, *Salmonella*, Shiga toxin–producing *Escherichia coli* (STEC) O157 and non-O157, *Shigella*, *Vibrio*, and *Yersinia* in 10 sites covering 15% of the U.S. population (48 million persons in 2011).§ FoodNet is a collaboration among CDC, 10 state health departments, the U.S. Department of Agriculture's Food Safety and Inspection Service (USDA-FSIS), and the Food and Drug Administration (FDA). Hospitalizations occurring within 7 days of specimen collection date are recorded, as is the patient's vital status at hospital discharge, or at 7 days after the specimen collection date if the patient was not hospitalized. All hospitalizations and deaths that occurred within a 7-day window are attributed to the infection. Surveillance for physician-diagnosed postdiarrheal hemolytic uremic syndrome (HUS), a complication of STEC infection characterized by renal failure, is conducted through a network of nephrologists and infection preventionists and by hospital discharge data review. This report includes 2011 HUS data for persons aged <18 years.

Incidence was calculated by dividing the number of laboratory-confirmed infections in 2012 by U.S. Census estimates of the surveillance population area for 2011.¶ A negative binomial model with 95% confidence intervals (CIs) was used to estimate changes in incidence from 2006–2008 to 2012 and from 1996–1998 to 2012 (*1*). The overall incidence of infection with six key pathogens for which >50% of illnesses are estimated to be foodborne (*Campylobacter*, *Listeria*, *Salmonella*, STEC O157, *Vibrio*, and *Yersinia*) was calculated (*2*). Trends were not assessed for *Cyclospora* because data were sparse, or for STEC non-O157 because of changes in diagnostic practices. For HUS, changes in incidence from 2006–2008 to 2011 were estimated.

Incidence and Trends

In 2012, FoodNet identified 19,531 laboratory-confirmed cases of infection (Table 1). The number of infections and incidence per 100,000 population, by pathogen, were as follows: *Salmonella* (7,800; 16.42), *Campylobacter* (6,793; 14.30), *Shigella* (2,138; 4.50), *Cryptosporidium* (1,234; 2.60), STEC non-O157 (551; 1.16), STEC O157 (531; 1.12), *Vibrio* (193; 0.41), *Yersinia* (155; 0.33), *Listeria* (121; 0.25), and *Cyclospora* (15; 0.03). As usual, the highest reported incidence was among children aged <5 years for *Cryptosporidium* and the bacterial pathogens other than *Listeria* and *Vibrio*, for which the highest incidence was among persons aged ≥65 years (Table 2).

Among 6,984 (90%) serotyped *Salmonella* isolates, the top three serotypes were Enteritidis, 1,238 (18%); Typhimurium, 914 (13%); and Newport, 901 (13%). Among 183 (95%) *Vibrio* isolates with species information, 112 were *V. parahaemolyticus* (61%), 25 were *V. vulnificus* (14%), and 20 were *V. alginolyticus* (11%). Among 496 (90%) serogrouped STEC non-O157 isolates, the most common serogroups were O26 (27%), O103 (23%), and O111 (15%). Among 2,318 (34%) *Campylobacter* isolates with species information, 2,082 (90%) were *C. jejuni*, and 180 (8%) were *C. coli*.

The estimated incidence of infection was higher in 2012 compared with 2006–2008 for *Campylobacter* (14% increase; confidence interval [CI]: 7%–21%) and *Vibrio* (43% increase; CI: 16%–76%) and unchanged for other pathogens (Figure 1). In comparison with 1996–1998, incidence of infection was

* Additional information available at http://www.cdc.gov/foodnet.

† The overall incidence of infection combines data for *Campylobacter*, *Listeria*, *Salmonella*, STEC O157, *Vibrio*, and *Yersinia*, six key bacterial pathogens for which >50% of illnesses are estimated to be transmitted by food.

§ FoodNet personnel regularly contact clinical laboratories to ascertain all laboratory-confirmed infections in residents of the surveillance areas.

¶ Final incidence rates will be reported when population estimates for 2012 are available.

TABLE 1. Number of cases of bacterial and parasitic infection, hospitalizations, and deaths, by pathogen — Foodborne Diseases Active Surveillance Network, United States, 2012*

Pathogen	Cases			Hospitalizations		Deaths	
	No.	Incidence†	Objective§	No.	(%)	No.	(%)
Bacteria							
Campylobacter	6,793	14.30	8.5	1,044	(15)	6	(0.09)
Listeria	121	0.25	0.2	116	(96)	13	(10.74)
Salmonella	7,800	16.42	11.4	2,284	(29)	33	(0.42)
Shigella	2,138	4.50	N/A¶	491	(23)	2	(0.09)
STEC O157	531	1.12	0.6	187	(35)	1	(0.19)
STEC non-O157	551	1.16	N/A	88	(16)	1	(0.18)
Vibrio	193	0.41	0.2	55	(29)	6	(3.11)
Yersinia	155	0.33	0.3	59	(38)	0	(0.00)
Parasites							
Cryptosporidium	1,234	2.60	N/A	236	(19)	6	(0.49)
Cyclospora	15	0.03	N/A	3	(20)	0	(0.00)
Total	**19,531**			**4,563**		**68**	

Abbreviations: N/A = not available; STEC = Shiga toxin–producing *Escherichia coli*.
* Data for 2012 are preliminary.
† Per 100,000 population.
§ *Healthy People 2020* objective targets for incidence of *Campylobacter*, *Listeria*, *Salmonella*, STEC O157, *Vibrio*, and *Yersinia* infections per 100,000 population.
¶ No national health objective exists for these pathogens.

significantly lower for *Campylobacter*, *Listeria*, *Shigella*, STEC O157, and *Yersinia*, whereas the incidence of *Vibrio* infection was higher (Figure 2). The overall incidence of infection with six key pathogens** transmitted commonly through food was lower in 2012 (22% decrease; CI: 11%–32%) compared with 1996–1998 and unchanged compared with 2006–2008.

The incidence of infections with specific *Salmonella* serotypes in 2012, compared with 2006–2008, was lower for Typhimurium (19% decrease; CI: 10%–28%), higher for Newport (23% increase; CI: 1%–50%), and unchanged for Enteritidis. Compared with 1996–1998, the incidence of infection was significantly higher for Enteritidis and Newport, and lower for Typhimurium.

Among 63 cases of postdiarrheal HUS in children aged <18 years (0.57 cases per 100,000 children) in 2011, 33 (52%) occurred in children aged <5 years (1.09 cases per 100,000). Compared with 2006–2008, the incidence was significantly lower for children aged <5 years (44% decrease; CI: 18%–62%) and for children aged <18 years (29% decrease; CI: 4%–47%).

Hospitalizations and Deaths

In 2012, FoodNet identified 4,563 hospitalizations and 68 deaths among cases of infection with pathogens transmitted commonly through food (Table 1). The percentage of patients hospitalized ranged from 15% for *Campylobacter* to 96% for *Listeria* infections. The percentage hospitalized was greatest among those aged ≥65 years for STEC O157 (67%), *Vibrio* (58%), *Salmonella* (55%), *Cyclospora* (50%), *Shigella* (41%), STEC non-O157 (34%), *Cryptosporidium* (33%), and *Campylobacter* (31%). At least 95% of patients with *Listeria* infection in each age group†† with cases were hospitalized. The percentage of patients who died ranged from 0% for *Yersinia* and *Cyclospora* to 11% for *Listeria* infections. The percentage that died was highest among persons aged ≥65 years for *Vibrio* (6%), *Salmonella* (2%), STEC O157 (2%), *Cryptosporidium* (1%), *Shigella* (1%), and *Campylobacter* (0.2%).

TABLE 2. Incidence* of laboratory-confirmed bacterial and parasitic infections in 2012,† by pathogen and age group — Foodborne Diseases Active Surveillance Network, United States

Pathogen	Age group (yrs)				
	<5	5–9	10–19	20–64	≥65
Bacteria					
Campylobacter	24.08	10.54	9.42	14.54	15.26
Listeria	0.17	0.00	0.03	0.17	1.05
Salmonella	63.49	19.33	11.26	12.15	17.22
Shigella	16.92	14.77	2.96	3.10	1.42
STEC§ O157	4.71	2.31	1.65	0.58	0.74
STEC non-O157	4.81	1.33	1.65	0.70	0.92
Vibrio	0.07	0.26	0.14	0.43	0.78
Yersinia	1.33	0.29	0.16	0.23	0.49
Parasites					
Cryptosporidium	3.68	3.09	1.70	2.54	3.01
Cyclospora	0.00	0.00	0.00	0.04	0.03

* Per 100,000 population.
† Data for 2012 are preliminary.
§ Shiga toxin–producing *Escherichia coli*.

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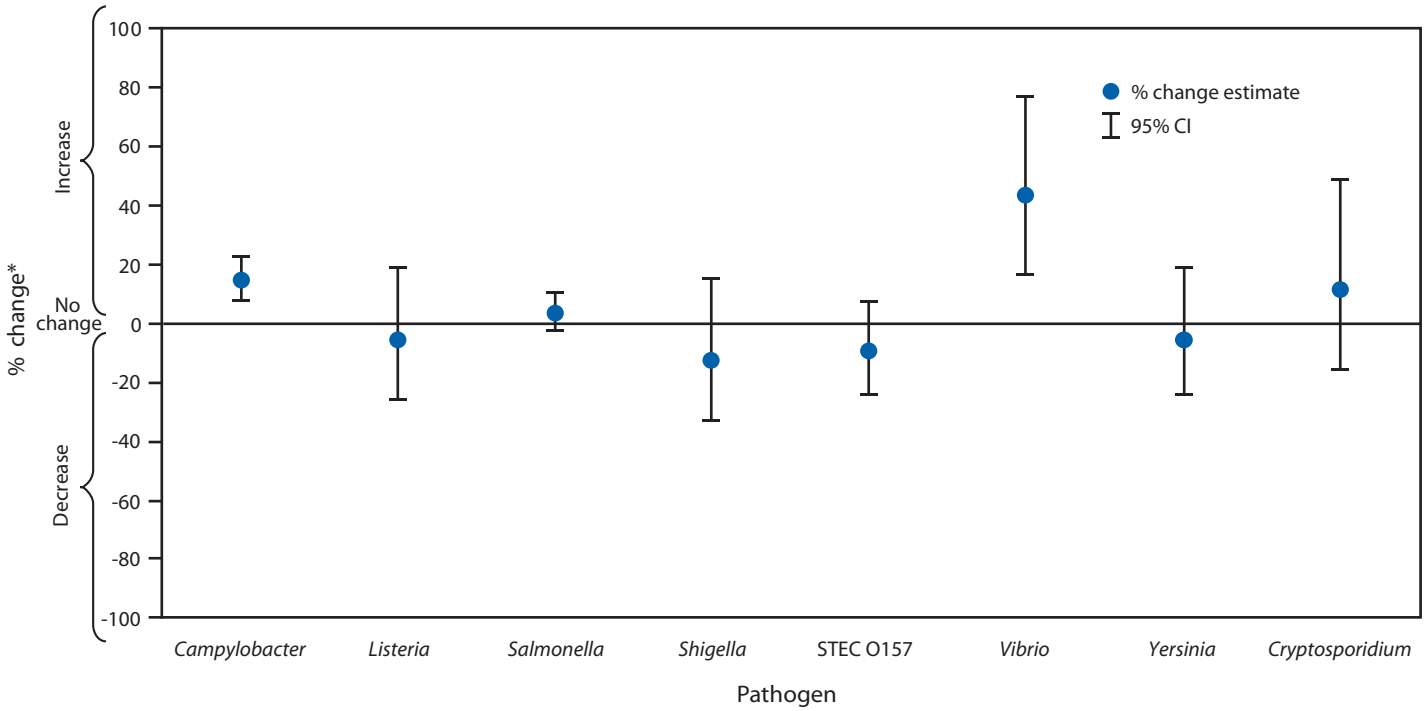
** *Campylobacter*, *Listeria*, *Salmonella*, STEC O157, *Vibrio*, and *Yersinia*.

†† Age groups defined as <5 years, 5–9 years, 10–19 years, 20–64 years, and ≥65 years.

FIGURE 1. Estimated percentage change in incidence of laboratory-confirmed bacterial and parasitic infections in 2012 compared with average annual incidence during 2006–2008, by pathogen — Foodborne Diseases Active Surveillance Network, United States



Abbreviations: CI = confidence interval; STEC = Shiga toxin–producing *Escherichia coli*.
* *No significant change* = 95% CI is both above and below the no change line; *significant increase* = estimate and entire CI are above the no change line; *significant decrease* = estimate and entire CI are below the no change line.

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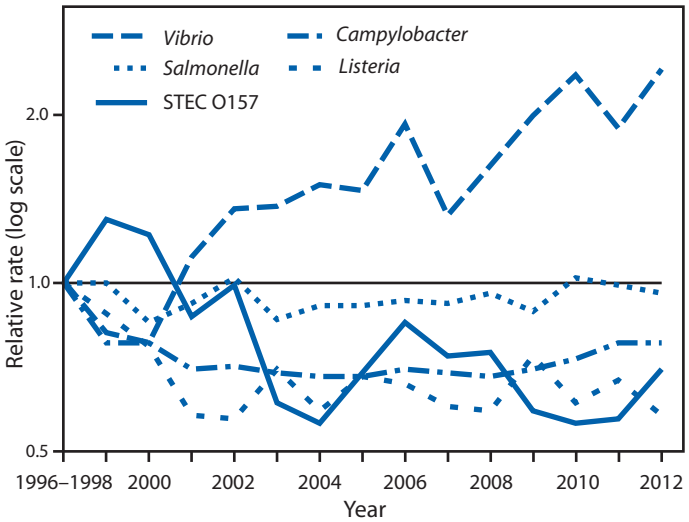
Editorial Note

In 2012, the incidence of infections caused by *Campylobacter* and *Vibrio* increased from the 2006–2008 period, whereas the incidence of infections caused by *Cryptosporidium*, *Listeria*, *Salmonella*, *Shigella*, STEC O157, and *Yersinia* was unchanged. These findings highlight the need to continue to identify and address food safety gaps that can be targeted for action by the food industry and regulatory authorities.

FIGURE 2. Relative rates of laboratory-confirmed infections with *Campylobacter*, STEC* O157, *Listeria*, *Salmonella*, and *Vibrio* compared with 1996–1998 rates, by year — Foodborne Diseases Active Surveillance Network, United States, 1996–2012†



* Shiga toxin–producing *Escherichia coli*.
† The position of each line indicates the relative change in the incidence of that pathogen compared with 1996–1998. The actual incidences of these infections cannot be determined from this figure.

After substantial declines in the early years of FoodNet surveillance, the incidence of *Campylobacter* infection has increased to its highest level since 2000. *Campylobacter* infections are more common in the western U.S. states and among children aged <5 years (*3*). Although most infections are self-limited, sequelae include reactive arthritis and Guillain-Barré syndrome.[§§] Associated exposures include consumption of poultry, raw milk, produce, and untreated water, and animal contact (*4,5*).

Declines in U.S. campylobacteriosis during 1996–2001 might have been related to measures meat and poultry processors implemented to comply with the Pathogen Reduction and Hazard Analysis and Critical Control Points (HACCP) systems regulations issued by USDA-FSIS in the late 1990s.[¶¶] In 2011, USDA-FSIS issued new *Campylobacter* performance standards for U.S. chicken and turkey processors.[***] Continued FoodNet surveillance can help to assess the public health impact of these standards and other changes. Detailed patient exposure information coupled with information on strain subtypes could help in assessing the relative contribution of various sources of infection and the effectiveness of control measures.

Although a significant increase was observed in reported *Vibrio* infections, the number of such infections remains low (*6*). *Vibrios* live naturally in marine and estuarine waters, and many infections are acquired by eating raw oysters (*7*). These infections are most common during warmer months, when waters contain more *Vibrio* organisms. Infections can be prevented by postharvest treatment of oysters with heat, freezing, or high pressure (*8*), or by thorough cooking. Persons who are immunocompromised or have impaired liver function should be informed that consuming raw seafood carries a risk for severe *Vibrio* infection. *Vibrios* also cause wound and soft-tissue infections among persons who have contact with water; for example, *Vibrio alginolyticus* typically causes ear infection (*9*).

The decrease in incidence of HUS in 2011 compared with 2006–2008 mirrors the decrease in the incidence of STEC O157 infection observed in 2011. The incidence of STEC O157 infection, which had declined since 2006, was no longer decreasing in 2012, and now exceeds the previously met *Healthy People 2010* target of one case per 100,000 persons. The continued increase in STEC non-O157 infections likely reflects increasing use by clinical laboratories of tests that detect these infections.

[§§] Additional information available at http://www.who.int/mediacentre/factsheets/fs255/en/index.html.

[¶¶] Additional information available at http://www.fsis.usda.gov/oppde/rdad/frpubs/93-016f.pdf.

[***] Additional information is available at http://www.fsis.usda.gov/science/haccp_verification_campylobacter_results_2011/index.asp.

What is already known on this topic?

The incidence of infections transmitted commonly by food that are tracked by the Foodborne Diseases Active Surveillance Network (FoodNet) has changed little in recent years. Foodborne illness continues to be an important public health problem.

What is added by this report?

Preliminary surveillance data show that the incidence of infections caused by *Campylobacter* and *Vibrio* increased in 2012, whereas incidence of other foodborne infections tracked by FoodNet was unchanged (i.e., *Cryptosporidium*, *Listeria*, *Salmonella*, *Shigella*, Shiga toxin–producing *Escherichia coli* O157, and *Yersinia*).

What are the implications for public health practice?

Reducing the incidence of foodborne infections will require commitment and action to implement measures known to reduce contamination of food and to develop new measures. Farmers, the food industry, regulatory agencies, the food service industry, consumers, and public health authorities all have a role.

FoodNet surveillance relies on isolation of bacterial pathogens by culture of clinical specimens; therefore, the increasing use of culture-independent tests for *Campylobacter* and STEC might affect the reported incidence of infection (*10*). Data on persons with only culture-independent evidence of infection suggests that in 2012, the number of laboratory-identified *Campylobacter* cases could have been 9% greater and the number of STEC (O157 and non-O157) cases 7%–19% greater than that reported (CDC, unpublished data, 2013). The lack of recent decline in STEC O157 incidence is of concern; continued monitoring of trends in the incidence of HUS and use of culture-independent testing might aid in interpreting future data on STEC O157 incidence.

The findings in this report are subject to at least four limitations. First, health-care–seeking behaviors and other characteristics of the population in the surveillance area might affect the generalizability of the findings. Second, many infections transmitted commonly through food (e.g., norovirus infection) are not monitored by FoodNet because these pathogens are not identified routinely in clinical laboratories. Third, the proportion of illnesses transmitted by nonfood routes differs by pathogen, and the route cannot be determined for individual, nonoutbreak-associated illnesses and, therefore, the data provided in this report do not exclusively relate to infections from foodborne sources. Finally, in some cases counted as fatal, the infection with the enteric pathogen might not have been the primary cause of death.

Most foodborne illnesses can be prevented. Progress has been made in decreasing contamination of some foods and

reducing illness caused by some pathogens, as evidenced by decreases in earlier years. In 2010, FDA passed the Egg Safety Rule,††† designed to decrease contamination of shell eggs with *Salmonella* serotype Enteritidis. In 2011, USDA-FSIS tightened its performance standard for *Salmonella* contamination to a 7.5% positive rate for whole broiler chickens.§§§ Finally, the Food Safety Modernization Act of 2011 gives FDA additional authority to improve food safety and requires CDC to strengthen surveillance and outbreak response.¶¶¶ Collection of comprehensive surveillance information further supports reductions in foodborne infections by helping to determine where to target prevention efforts, supporting efforts to attribute infections to sources, guiding implementation of measures known to reduce food contamination, and informing development of new measures. Because consumers can bring an added measure of safety during food storage, handling, and preparation, they are advised to seek out food safety information, which is available online.****

††† Additional information available at http://www.fda.gov/food/guidanceregulation/guidancedocumentsregulatoryinformation/eggs/ucm170615.htm.

§§§ Additional information available at http://www.gpo.gov/fdsys/pkg/FR-2011-03-21/pdf/2011-6585.pdf.

¶¶¶ Additional information available at http://www.fda.gov/food/guidanceregulation/fsma/ucm242500.htm.

**** Additional food safety information is available at http://www.cdc.gov/winnablebattles/foodsafety/index.html, http://www.foodsafety.gov and http://www.fightbac.org.

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Assessment of Current Practices and Feasibility of Routine Screening for Critical Congenital Heart Defects — Georgia, 2012

In September 2011, the U.S. Secretary of Health and Human Services recommended that critical congenital heart defects (CCHD) be added to the Recommended Uniform Screening Panel (RUSP) for newborns. Anecdotal reports in early 2012 suggested that some Georgia hospitals had begun screening for CCHD using pulse oximetry. To better understand the prevalence of routine CCHD screening, specific practices among screening hospitals, and barriers to screening among all birthing hospitals in the state, CDC and the Georgia Department of Public Health (DPH) conducted two surveys of Georgia hospitals in June 2012. Eleven pulse oximetry screenings at five hospitals also were observed to estimate screening time. The initial survey was sent to 89 birthing hospitals, among which 71 (80%) responded; 22 (31%) reported currently screening for CCHD and 20 (28%) planned to start in 2012. Barriers to screening included lack of a clear follow-up protocol for positive screening tests, uncertainty about reporting screening results to public health organizations, and cost concerns. Sixteen (73%) currently screening hospitals responded to the second survey. Only one third of screening hospitals followed the CCHD screening protocol endorsed by the American Academy of Pediatrics; the remaining hospitals screened at different times or had different criteria for a positive screen. Screening time averaged 10 minutes per newborn. In the absence of a state mandate, routine screening has begun in many Georgia hospitals. Use of a standardized screening protocol for CCHD could reduce current variation in screening practices among Georgia hospitals. Working agreements between hospitals also are needed to ensure access to echocardiography and follow-up of newborns with possible CCHD.

Congenital heart defects are associated with approximately eight births per 1,000 (*1*); approximately 25% of these defects are CCHD and require surgery or cardiac catheterization at age <1 year (*2*). Many CCHDs are detected prenatally or during physical examination after birth, but some infants with CCHD are discharged home without a diagnosis, putting them at risk for severe disability or death (*3*). In 2010, the Secretary's Advisory Committee on Heritable Disorders in Newborns and Children recommended that CCHD be added to the RUSP, and in September 2011, the Secretary accepted the committee's recommendation.* Currently, screening for CCHD is accomplished through pulse oximetry, a noninvasive test used to detect hypoxemia, which typically is present for the seven CCHD that are the primary targets of pulse oximetry screening (*3*). The predictive values and sensitivity of pulse oximetry screening varies based on the screening protocol that is used (e.g., timing of screening after birth or number of extremities measured) (*4*). Despite this federal recommendation to include CCHD on the RUSP, implementation is a state decision. Although universal CCHD screening currently is not mandated in Georgia, anecdotal reports in early 2012 indicated the practice had begun in some birthing hospitals.

DPH requested assistance from CDC to assess the current practices and feasibility of routine screening for CCHD in Georgia. In June 2012, CDC and DPH distributed a survey about CCHD screening practices using pulse oximetry to nurse managers at all the 89 Georgia birthing hospitals. Hospitals could complete the survey online, via fax, or by telephone. The 71 hospitals that completed the initial survey represented 80% of all birthing hospitals in Georgia and accounted for 87% of all live births in the state in 2011 (*5*). CDC and DPH distributed a follow-up online survey about specific screening procedures to the 22 hospitals that reported in the initial survey that they were currently screening for CCHD using pulse oximetry; 16 (73%) responded. From the 22 hospitals currently screening, a convenience sample of five were selected, at which CDC and DPH staff members observed five screening demonstrations and six actual screenings. Assessment of five screenings included quantification of transport time to and from the nursery, and six did not because other procedures (e.g., metabolic screening) were conducted during these same nursery visits. Two-sided Fisher's exact tests (significance level of 0.05) were used to assess the statistical significance of differences in the prevalence of hospital characteristics by screening status.

Of the 71 hospitals that responded to the initial survey, 22 (31%) reported currently screening for CCHD using pulse oximetry in their well-baby nursery (11 began in 2010 or 2011, nine in 2012, and two did not indicate when they started); 34 (48%) had plans to start (20 by the end of 2012 and 14 at other times); 14 (20%) had no plans to start; and one did not know of plans to start. No differences by hospital screening status were noted in the number of live births in 2011, availability of echocardiography onsite for infants, or in the availability of pediatric cardiologists for follow-up of babies with CCHD (Table). Several barriers to CCHD screening were reported more frequently among nonscreening hospitals (Table). Overall, 46 (65%) hospitals reported that they could perform echocardiography on-site. For follow-up of patients

* Available at http://www.hrsa.gov/advisorycommittees/mchbadvisory/heritabledisorders/recommendations/correspondence/cyanoticheartsecre09212011.pdf.

TABLE. Characteristics of 71 Georgia birthing hospitals currently screening for CCHD, planning to start soon, or with no plans to start screening,* as of June 2012

Characteristic	Currently screening (n = 22)		Planned to start screening in 2012 (n = 20)		Plan to start screening at other times (n = 14)		No plans to start screening or unknown* (n = 15)		p-value†
	No.	(%)§	No.	(%)§	No.	(%)§	No.	(%)§	
No. of births (2011)									
Mean	1,837		2,062		999		1,424		
(Range)	(175–5,500)		(111–1,752)		(200–3,300)		(165–3,238)		
Median	1,475		800		812.5		1,164		0. 427
Total	**40,411**		**39,185**		**13,992**		**21,353**		
How hospital records or plans to record pulse oximetry screening results									0.093
EMR only	15	(68)	11	(55)	9	(64)	12	(80)	
Paper only	4	(18)	6	(30)	5	(36)	0	(0)	
Both EMR and paper	2	(9)	3	(15)	0	(0)	0	(0)	
Missing	1	(5)	0	(0)	0	(0)	3	(20)	
Hospital has facilities to perform diagnostic echocardiography on-site for infants									0.844
Yes	15	(68)	14	(70)	8	(57)	9	(60)	
No one available	6	(27)	6	(30)	6	(43)	5	(33)	
Don't know if available	1	(5)	0	(0)	0	(0)	1	(7)	
Availability of pediatric cardiologists for follow-up and diagnosis of babies born with CCHD									0.067
Specialists are on-site at hospital	0	(0)	0	(0)	1	(7)	2	(13)	
Consultants with a specialty group see patients on-site at hospital	10	(45)	8	(40)	2	(14)	4	(27)	
Echocardiography are reviewed remotely; hospital transfer patients if further cardiac care is needed	3	(14)	2	(10)	0	(0)	4	(27)	
Hospital transfers patients out to another facility	9	(41)	10	(50)	9	(64)	4	(27)	
Hospital does not have pediatric cardiologist available at all	0	(0)	0	(0)	1	(7)	1	(7)	
Other	0	(0)	0	(0)	1	(7)	0	(0)	
Barriers to screening									
No clear plan for follow-up of positive results	5	(23)	6	(30)	8	(57)	5	(33)	0.211
Unsure of how to report results	4	(18)	5	(25)	6	(43)	7	(47)	0.200
Concerned about reimbursement for cost of screening (but no need for new staff or equipment)	7	(32)	8	(40)	4	(29)	2	(13)	0.402
Need to purchase new equipment to carry out the screening	2	(9)	7	(35)	7	(50)	5	(33)	**0.043**
No state mandate for screening	1	(5)	1	(5)	2	(14)	7	(47)	**0.003**
Waiting to hear about experiences of other hospitals	0	(0)	3	(15)	6	(43)	2	(13)	**0.004**
Believe number of false positives will be too high	1	(5)	1	(5)	2	(14)	4	(27)	0.168
Believe CCHD infants will be picked up through other mechanisms	0	(0)	0	(0)	0	(0)	3	(20)	**0.014**
Need to hire new staff to carry out the screening	0	(0)	0	(0)	1	(7)	1	(7)	0.240
Other									
Developing screening policies and guidance and educating staff about them	3	(14)	7	(35)	2	(14)	2	(13)	0.302
Physician support	2	(9)	4	(20)	2	(14)	3	(20)	0.727
Staff time	1	(5)	3	(15)	0	(0)	0	(0)	0.263
More evidence about pulse oximetry screening needed	0	(0)	1	(5)	0	(0)	2	(13)	0.214
Documentation of results	2	(9)	0	(0)	0	(0)	0	(0)	0.333
No barriers	9	(41)	2	(10)	1	(7)	2	(13)	**0.040**

Abbreviations: EMR = electronic medical record; CCHD = critical congenital heart defects.
* Includes responses from the one hospital that did not know its CCHD screening status.
† Fisher's exact test, comparison of all nonmissing responses or Kruskal-Wallis test for difference in median number of live births. Significant p-values (<0.05) are in bold.
§ Percentages might not sum to 100% because of rounding.

with suspected CCHD, 32 (45%) had to transfer patients. The median driving distance to a transfer hospital was 54 miles (range: 0–211 miles).

Among 16 (73%) of the 22 screening hospitals that responded to the follow-up survey, five (31%) reported following the CCHD screening protocol[†] endorsed by the American Academy of Pediatrics, the American College of Cardiology, and the American Heart Association (*6*). The remaining hospitals either screened at different times or used different criteria for a positive screen. No hospital reported providing written documentation to parents about the screening. Among the 16 hospitals, 12 did not know how often to send screening data to DPH and 11 did not know what types of screening data, such as true and false positives and negatives, could be sent to DPH. Four of the 16 screening hospitals had identified one or more infants with a CCHD through screening. Thirteen of the hospitals neither hired extra staff nor added extra staff hours to accommodate CCHD screening, and three did not respond to the question. The average time to conduct and document the 11 observed screens was 10 minutes per screen (range: 3–15 minutes).

CDC recommended that 1) guidance be provided to hospitals on the type of data to report to DPH, and the frequency of reporting; 2) an educational webinar be developed for hospitals on signs and symptoms of CCHD and the pulse oximetry screening protocol endorsed by the American Academy of Pediatrics; 3) educational materials that hospitals can provide to parents about CCHD screening be developed and disseminated; and 4) working agreements between hospitals be established to ensure access to echocardiography and follow-up for all newborns with possible CCHD.

Reported by

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Editorial Note

In Georgia, a state without mandated CCHD screening, at least 42 birthing hospitals, accounting for 60% of births in the state (*5*), are conducting routine CCHD screening in their well-baby nursery (20) or planned to start (22) by the end of 2012. Frequently cited barriers to CCHD screening include the lack of a clear protocol for follow-up for positive screening results, uncertainty about how to report results to Georgia DPH, and cost concerns. In addition, many hospitals are unable to perform echocardiography on-site or have to transfer patients for follow-up of suspected CCHD. Even among hospitals already screening, screening protocols and practices varied.

Published reports from other states are limited. A survey of Wisconsin hospitals found similar results to this assessment; approximately 25% of Wisconsin hospitals had voluntarily begun screening. Barriers to screening included lack of access to echocardiography, long transfer hospital distances, and variation in screening procedures and protocols (*7*). The average screening time of 10 minutes per newborn from this assessment is greater than previous estimates of 2–3.5 minutes (*8,9*). Despite the added potential burden of approximately 274 hours per year devoted to CCHD screening for the typical Georgia birthing hospital (based on a mean of 1,642 births among hospitals currently screening or planning to begin screening by the end of 2012), none of the hospitals that responded to the survey added staff or hours to accommodate screening.

The findings in this report are subject to at least three limitations. First, the survey response rates were 80% to the initial survey and 73% to the second survey. Nonresponders

What is already known on this topic?

In September 2011, the U.S. Secretary of Health and Human Services recommended that critical congenital heart defects (CCHD) be added to the Recommended Uniform Screening Panel for newborns. Universal screening for CCHD using pulse oximetry is not mandated in Georgia, but anecdotal reports in early 2012 suggested screening had begun in some birthing hospitals.

What is added by this report?

Among 71 of 89 Georgia birthing hospitals that responded to the initial survey, 42 (59%) reported currently (22) or planning to start (20) screening for CCHD using pulse oximetry by the end of 2012. Barriers to screening in some hospitals and variation in screening practices remain. Nearly one third of hospitals are unable to perform echocardiography for infants on-site in their facility and almost half need to transfer newborns with possible CCHD to another facility for follow-up and diagnosis.

What are the implications for public health practice?

Implementation of routine screening for CCHD in the absence of a state mandate has led to variation in screening protocols. Use of a standard screening protocol and educational programs might alleviate these differences. Hospitals need recommendations as to what screening data to collect and report. Working agreements between hospitals are needed to ensure access to echocardiography and follow-up of newborns with suspected CCHD.

[†] Available at http://www.cdc.gov/ncbddd/pediatricgenetics/pulse.html.

might have had different CCHD screening experiences from responders; if so, these results might not be applicable to all birthing hospitals in Georgia. Second, screening practices were reported by the nurse manager who filled out the survey and might not reflect those of all nurses in a given facility. Finally, the numbers of hospitals conducting CCHD screening and the specific screening procedures used are likely to change over time, so the results of this assessment might not reflect Georgia hospitals' current screening practices.

The findings from this assessment of CCHD screening practices in Georgia might be useful to other states. Routine screening has voluntarily begun in many Georgia hospitals, although screening practices vary and not all hospitals are able to provide appropriate follow-up for infants with possible CCHD. Georgia hospitals need guidance on a standardized screening protocol for CCHD. Working agreements also need to be created between hospitals to ensure access to echocardiography and follow-up of newborns with possible CCHD in Georgia hospitals.

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Rapid Implementation of Pulse Oximetry Newborn Screening to Detect Critical Congenital Heart Defects — New Jersey, 2011

In August 2011, New Jersey implemented a statewide newborn screening protocol for critical congenital heart defects (CCHD) using pulse oximetry. In January 2012, CDC responded to a request from the New Jersey Department of Health (NJDOH) to assist with an assessment of the implementation. Out of the 52 birthing facilities in New Jersey, a sample of 11 was selected. Staff interviews were conducted to assess screening and data collection processes, data flow and tracking procedures, electronic medical record (EMR) capabilities, and capacity to report data to NJDOH. Feedback also was obtained about the questionnaire being used to follow-up on positive screening results. All 11 facilities were screening for CCHD. Among the 11 facilities, three were electronically entering and maintaining data into an EMR, five were manually entering and maintaining data into paper charts and logs, and three were both electronically and manually entering and maintaining data. Facilities reported that implementation of newly mandated CCHD screening posed a low burden to hospital staff members. NJDOH receives aggregate pulse oximetry screening data from all New Jersey birthing facilities. During the first 3 months of screening, preliminary data indicated that 98.2% of 25,214 newborns were screened. Hospitals reported data on 12 newborns with positive screening results; two newborns were newly diagnosed with CCHD as a result of pulse oximetry screening. Because of state-specific factors, such as out-of-state referral patterns, these findings might underestimate the anticipated number of positive screens in states with varying referral patterns and use of prenatal diagnosis. Rapid implementation of universal CCHD screening posed a relatively low burden to hospitals in New Jersey.

The system assessment began in January 2012 (5 months after hospitals commenced routine screening). The objectives were to assess EMR capabilities, assess the capacity to report screening data to NJDOH, and evaluate the data flow and tracking at a sample of birthing facilities. As part of this investigation, 11 of 52 New Jersey birthing hospitals were visited. Four of these birthing facilities were included because they had identified newborns with positive screening results during the first 3 months of implementation. The other seven hospitals were selected as a random sample of all other birthing facilities in the state, stratified by geographic location, hospital birth census, and hospital level of care.

NJDOH's mechanism for pulse oximetry surveillance includes collection of aggregate data reports from each licensed birthing facility and reports of positive screening results to the confidential New Jersey Birth Defects Registry (NJBDR). The aggregate data reports submitted to NJDOH contain the number of live births, number of newborns screened, an explanation of any discrepancies between those numbers, and the number of positive screens. During the investigation, NJDOH staff members shared results of these preliminary aggregate screening data from the first 3 months of system operation (August 31–November 30, 2011).

A structured questionnaire with open-ended questions was developed by the investigation team and distributed to hospitals before the field investigation. Face-to-face interviews were conducted by CDC and NJDOH personnel to assess pulse oximetry screening procedures, data collection and maintenance procedures, reporting practices, and burden (i.e., increased workload or additional duties) of screening and reporting by hospital staff. Staff members were asked to rate the level of burden on a scale ranging from 1 = no burden to 10 = very burdensome. Key personnel, such as well baby nursery and neonatal intensive care unit managers and staff nurses, clinical educators, and representatives from the hospital's biomedical services department, participated in each interview. Medical charts were reviewed with hospital staff members at the four facilities that previously had reported positive screens and the process of reporting data to NJDOH was discussed. Feedback on the questionnaire being used for follow-up of positive screens was obtained from facility staff members. Members of the investigation team observed pulse oximetry screening and documentation practices in each hospital's well baby nursery and neonatal intensive care unit.

All 11 hospitals had incorporated screening for CCHD into routine nursing care in their well baby nurseries and neonatal intensive care units. Hospital nurses reported that the addition of the newly mandated screening processes posed minimal burden (average score 2.1). Nurses indicated that pulse oximetry was a familiar skill and screening all newborns was easily incorporated into their routine tasks. Three of the 11 hospitals were electronically entering and maintaining data in an EMR, five of the 11 were manually entering and maintaining data into paper charts or logs, and three of the 11 were both electronically and manually entering and maintaining data. All facilities had mechanisms for collecting and reporting aggregate screening data to NJDOH and positive screening results to NJBDR. All facilities reported that aggregate screening data would be submitted to NJDOH as requested. Hospitals reported the process of submitting aggregate screening data to NJDOH

posed a moderate burden (average score 4.2) to staff members. Hospitals requested a form with detailed instructions to report discrepancies between the number of live-births and number of newborns screened for future aggregate screening data requests. All facilities reported that individual-level screening and clinical data would be reported to NJBDR for positive screening results. The NJBDR follow-up questionnaire was modified based on feedback from nurses.

In the first 3 months following implementation of the mandate to screen all newborns for CCHD using pulse oximetry, preliminary data indicated that 98.2% of 25,214 newborns born in licensed birthing facilities were screened, with 12 positive screens. Two positive screens were confirmed CCHD cases initially detected by pulse oximetry screening (no prior diagnosis), which otherwise might have resulted in death or disability.

Reported by

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Editorial Note

Universal newborn screening is the process by which newborns are screened shortly after birth for conditions that can cause severe illness, disability, or death. Through early identification and treatment, newborn screening provides an opportunity to reduce morbidity and mortality (*1*). The Recommended Uniform Newborn Screening Panel is a list of conditions for which all newborns should be screened, as recommended by the U.S. Secretary of Health and Human Services.* In September 2011, the Secretary approved the addition of screening for critical congenital heart disease using pulse oximetry to the panel.† Congenital heart disease occurs in nearly 1% of live births; approximately one quarter of cases will be CCHD, defined as those defects requiring cardiac surgery or catheterization before age 1 year (*2,3*).

Pulse oximetry is a noninvasive technology that can be used to detect hypoxemia, a clinical sign of CCHD (*2*). In the absence of early detection, newborns with CCHD are at risk for death in the first few days or weeks of life. Current approaches for detection of CCHD include prenatal ultrasound or physical examination findings (e.g., cyanosis or tachypnea), although not all infants with CCHD are detected with prenatal ultrasound screening or by physical examination findings (*2,3*). Predictive values and sensitivity of pulse oximetry screening varies based on the screening protocol used (e.g., timing of screening after birth or number of extremities measured) (*4*). On June 2, 2011, New Jersey passed legislation requiring that all licensed birthing facilities screen newborns for CCHD at age ≥24 hours using pulse oximetry.§ This legislation became effective 90 days later, on August 31, 2011, making New Jersey the first state to implement mandatory statewide CCHD screening. This report represents the first systematic gathering of process data on legislatively mandated newborn screening for CCHD in the United States. Most states have not yet mandated universal newborn screening for CCHD; however, similar legislation was introduced or enacted in at least 18 other states during their 2011–2012 legislative sessions (American Academy of Pediatrics, Division of State Government Affairs, unpublished data, 2012).

What is already known on this topic?

Congenital heart disease occurs in approximately 1% of live births, and approximately one quarter of cases will be critical congenital heart defects (CCHD). Newborn screening using pulse oximetry can be used to detect hypoxemia, a clinical sign of CCHD. In August 2011, New Jersey implemented a statewide newborn screening protocol for critical congenital heart defects using pulse oximetry.

What is added by this report?

Five months after the CCHD screening program was implemented in New Jersey, all hospitals in a sample were screening and reporting data to the state health department. Hospitals reported that implementation of the newly mandated pulse oximetry screening posed minimal burden (i.e., increased workload or additional duties) to their nursing staff members. During the first 3 months of screening, two newborns were identified through screening as having previously unsuspected CCHD.

What are the implications for public health practice?

Rapid implementation of universal CCHD screening posed a relatively low burden to hospitals in New Jersey. Data collection and reporting are essential to evaluate the effect of this public health program.

* Available at http://www.hrsa.gov/advisorycommittees/mchbadvisory/heritabledisorders/recommendedpanel/index.html.

† Additional information available at http://www.hrsa.gov/advisorycommittees/mchbadvisory/heritabledisorders/recommendations/correspondence/cyanoticheartsecre09212011.pdf.

§ Birthing facilities required to perform pulse oximetry screening; rules, regulations, Pub. L. 2011 Chapter 74 (State of New Jersey, 2011). Available at http://www.njleg.state.nj.us/2010/bills/al11/74_.htm.

New Jersey birthing facilities were willing and capable of screening, collecting data, and reporting data to NJDOH. Reporting of aggregate birthing facility screening data to NJDOH is a short-term measure until pulse oximetry screening results can be incorporated into an electronic birth reporting system. For this short-term measure, CDC recommended that NJDOH provide a prescriptive form for future aggregate data requests. All facilities in the sample had incorporated screening and documentation of results. Some facilities had more sophisticated methods of data maintenance and tracking to ensure that all newborns are screened.

In the first 3 months of screening, two newborns were detected with CCHD using pulse oximetry that otherwise might have resulted in death or disability. Pulse oximetry screening also might identify other medical conditions, such as pulmonary conditions or sepsis, potentially improving newborn care and subsequent outcomes. New Jersey–specific characteristics, such as mothers at high risk choosing to deliver in birthing facilities in surrounding states, might have influenced the number of positive screens that were reported. These and other factors should be considered before using New Jersey data to estimate resource needs.

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Announcements

World Malaria Day — April 25, 2013

World Malaria Day is commemorated on April 25, the date in 2000 when 44 African leaders met in Abuja, Nigeria, and committed their countries to cutting malaria-related deaths. In the last decade, increased funding and political commitment have led to a scale-up of effective malaria prevention and control interventions, saving approximately 1.1 million lives globally and decreasing malaria mortality by nearly 25% globally and 33% in sub-Saharan Africa (*1*). Despite these successes, an estimated 660,000 malaria-related deaths occurred worldwide in 2010 (*1*). For 2013, the theme of World Malaria Day is "Invest in the Future: Defeat Malaria," which serves as a reminder of the ultimate goal.

CDC supports global malaria efforts through the President's Malaria Initiative, a U.S. government interagency initiative to reduce malaria incidence and mortality in 19 countries in sub-Saharan Africa and in the Greater Mekong Subregion in Asia. This effort has helped deliver millions of insecticide-treated mosquito nets, antimalarial drugs, and rapid diagnostic test kits to ensure that everyone at risk for malaria has access to life-saving prevention and treatment. In addition, CDC conducts multidisciplinary strategic and applied research globally to increase knowledge about malaria and develop safe, effective interventions that can lead to the elimination and eventual eradication of malaria.

Additional information about CDC's malaria activities is available at http://www.cdc.gov/malaria.

Reference

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National Infant Immunization Week — April 20–27, 2013

National Infant Immunization Week (NIIW) will be observed April 20–27, 2013. An annual event since 1994, NIIW brings together local and state health departments, national immunization partners, and health-care professionals across the country to hold community activities and events highlighting the importance of protecting infants from vaccine-preventable diseases through immunization.

Although immunization rates for vaccines routinely recommended for children remain at or near record highs, recent outbreaks of measles and pertussis in the United States underscore the importance of maintaining high immunization rates by successfully addressing parents' questions and concerns about childhood vaccines (*1*). This year, CDC developed educational and promotional materials to remind parents of the importance of vaccinating their children according to the recommended immunization schedule. For the second consecutive year, NIIW will be observed simultaneously with World Immunization Week, an initiative of the World Health Organization to promote immunization and advance equity in the use of vaccines and universal access to vaccination services. Additionally, the CDC Foundation and CDC will recognize recipients of the second annual CDC Childhood Immunization Champion Award, which recognizes individuals for their contributions to public health through their work in childhood immunizations.

Additional information about NIIW is available at http://www.cdc.gov/vaccines/events/niiw. Additional information about World Immunization Week is available at http://www.who.int/campaigns/immunization-week/2013/en/index.html.

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Erratum

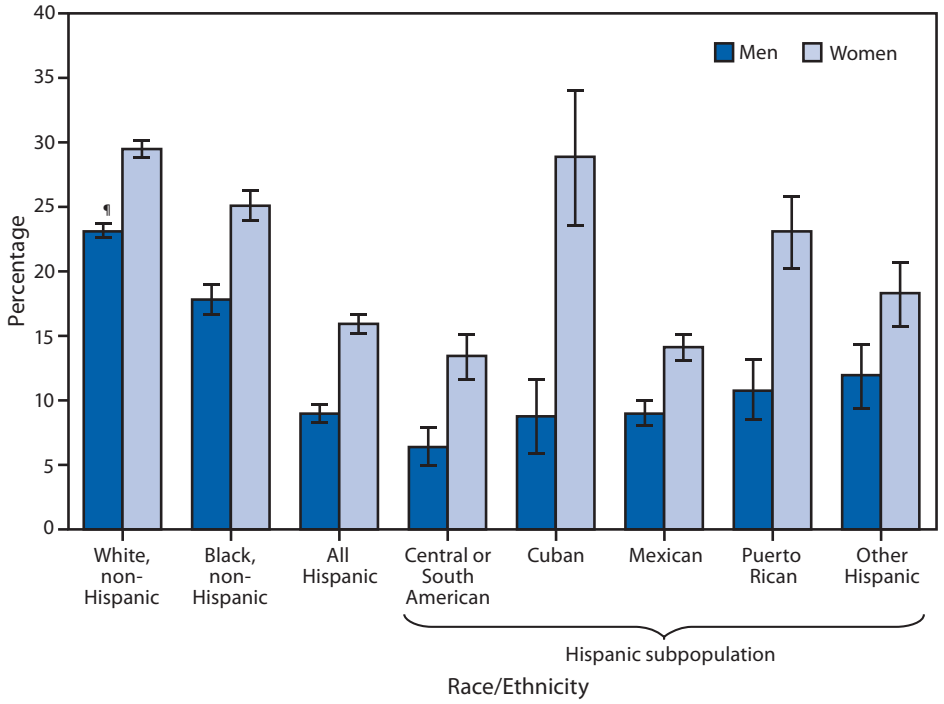
Vol. 62, No. 13

An error occurred in the first sentence of the announcement, "World Health Day — April 7, 2013," on page 237. The sentence should read as follows: "World Health Day and the **65th** anniversary of the World Health Organization (WHO) will be observed April 7."

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Percentage of Adults Ever Told They Have Some Form of Arthritis or a Related Condition,* by Sex, Race/Ethnicity, and Hispanic† Subpopulation — National Health Interview Survey, United States, 2011§



* Based on a survey question that asked respondents, "Have you ever been told by a doctor or other health professional that you have some form of arthritis, rheumatoid arthritis, gout, lupus, or fibromyalgia?" Those that answered yes were classified as having an arthritis diagnosis. Unknowns were not included in the denominators when calculating percentages.

† Persons of Hispanic ethnicity might be of any race or combination of races. Non-Hispanic persons are those who are not of Hispanic ethnicity, regardless of race.

§ Estimates are based on household interviews of a sample of the noninstitutionalized U.S. civilian population.

¶ 95% confidence interval.

During 2011, in each racial/ethnic group considered, women were more likely than men to have been told by a doctor or other health professional that they have arthritis or a related condition. Among men and women, Hispanic adults were less likely than non-Hispanic white and non-Hispanic black adults to have been told that they have arthritis. Among Hispanic subpopulations, considerable variation occurred, with notably higher rates for Cuban and Puerto Rican women.

Source: National Health Interview Survey, 2011 sample adult core component. Available at http://www.cdc.gov/nchs/nhis.htm.

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