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Outbreaks Associated with Untreated Recreational Water — United States, 2000–2014

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Outbreaks associated with untreated recreational water can be caused by pathogens, toxins, or chemicals in fresh water (e.g., lakes, rivers) or marine water (e.g., ocean). During 2000–2014, public health officials from 35 states and Guam voluntarily reported 140 untreated recreational water–associated outbreaks to CDC. These outbreaks resulted in at least 4,958 cases of disease and two deaths. Among the 95 outbreaks with a confirmed infectious etiology, enteric pathogens caused 80 (84%); 21 (22%) were caused by norovirus, 19 (20%) by *Escherichia coli*, 14 (15%) by *Shigella*, and 12 (13%) by *Cryptosporidium*. Investigations of these 95 outbreaks identified 3,125 cases; 2,704 (87%) were caused by enteric pathogens, including 1,459 (47%) by norovirus, 362 (12%) by *Shigella*, 314 (10%) by *Cryptosporidium*, and 155 (5%) by *E. coli*. Avian schistosomes were identified as the cause in 345 (11%) of the 3,125 cases. The two deaths were in persons affected by a single outbreak (two cases) caused by *Naegleria fowleri*. Public parks (50 [36%]) and beaches (45 [32%]) were the leading settings associated with the 140 outbreaks. Overall, the majority of outbreaks started during June–August (113 [81%]); 65 (58%) started in July. Swimmers and parents of young swimmers can take steps to minimize the risk for exposure to pathogens, toxins, and chemicals in untreated recreational water by heeding posted advisories closing the beach to swimming; not swimming in discolored, smelly, foamy, or scummy water; not swimming while sick with diarrhea; and limiting water entering the nose when swimming in warm freshwater.

An outbreak associated with untreated recreational water* is the occurrence of similar illnesses in two or more persons, epidemiologically linked by location and time of exposure to recreational water or to pathogens, toxins, or chemicals aerosolized or volatilized from recreational water into the surrounding air. Public health officials in the 50 states, the District of Columbia, U.S. territories, and Freely Associated States† can voluntarily report recreational water–associated outbreaks to CDC. This report focuses on data on two groups of untreated recreational water–associated outbreaks: 1) those that began during 2000–2012 and were previously reported (*1*), and

* Untreated recreational water is water that has not undergone a disinfection or treatment process to maintain good microbiological quality for recreation.

† Includes Federated States of Micronesia, Marshall Islands, and Palau.

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2) those that began during 2013–2014 and were electronically reported to the Waterborne Disease and Outbreak Surveillance System (WBDOSS)[§] by December 31, 2015. Data on each outbreak include case count,[¶] number of deaths, etiology, setting (e.g., park), and venue (e.g., lake/reservoir/pond) where the exposure occurred, and earliest illness onset date. Poisson regression analysis was conducted to assess the trend in the annual counts of outbreaks.

During 2000–2014, public health officials from 35 states and Guam voluntarily reported 140 untreated recreational water–associated outbreaks that resulted in at least 4,958 cases[**] (Table) and two deaths. Etiology was confirmed for 103 (74%) outbreaks. Among these, 95 (92%) were caused by pathogens, including five outbreaks with multiple etiologies,[††] and resulted in at least 3,125 cases; enteric pathogens caused 80 (84%) of the 95 outbreaks and 2,704 (87%) of the 3,125 cases. Among the 95 outbreaks with a confirmed infectious etiology, 21 (22%) were caused by norovirus, 19 (20%) by *E. coli*, 14 (15%) by *Shigella*, and 12 (13%) by *Cryptosporidium*. Investigations of the 95 outbreaks identified 1,459 (47%) cases caused by norovirus, 362 (12%) by *Shigella*, 345 (11%) by avian schistosomes, 314 (10%) by *Cryptosporidium*, and 155 (5%) by *E. coli*. The two deaths occurred within a single outbreak caused by *Naegleria fowleri*.[§§] Of the 103 outbreaks with confirmed etiology, eight (8%) were caused by toxins or chemicals and resulted in at least 78 cases. Of the eight outbreaks caused by toxins or chemicals, seven (88%) were caused by algal toxins from harmful algal blooms.

Public parks (50 [36%]) and beaches (45 [32%]) were the leading settings associated with the 140 outbreaks. Most outbreaks were associated with a lake/reservoir/pond venue (117 [84%]). Among the 140 outbreaks, the majority started during June–August (113 [81%]), with 65 (58%) staring in July (Figure). None of the outbreaks started during December–February. Poisson regression analyses indicated the annual outbreak count did not change significantly over the 15 years (p = 0.477).

Discussion

A total of 140 untreated recreational water–associated outbreaks were reported to CDC during 2000–2014. The

[§] 2013–2014 are the last years for which finalized data were available. For more information on WBDOSS, visit https://www.cdc.gov/healthywater/surveillance/index.html; outbreaks resulting from recreational water exposures on cruise ships are not reported to WBDOSS.

[¶] Based on the estimated number of primary cases. For outbreaks that started before 2009, if both the actual and estimated case counts were reported, the estimated case count was used if the population was sampled randomly or the estimated count was calculated by applying the attack rate to a standardized population.

[**] https://www.cdc.gov/healthywater/surveillance/rec-water-tables-figures.html.

[††] The five outbreaks categorized as multiple included outbreaks of 1) *Shigella* and *Plesiomonas shigelloides*; 2) *Shigella*, norovirus, and *Yersinia enterolytica*; 3) *Shigella*, *Campylobacter*, and norovirus; 4) *Shigella*, *Escherichia coli*, and *Plesiomonas shigelloides*; and 5) *Giardia duodenalis* and norovirus.

[§§] *Naegleria fowleri* typically causes isolated cases of primary amebic meningoencephalitis. For these two cases, despite an investigation by local public health authorities, the location of common exposure was not definitively identified.

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TABLE. Number of untreated recreational water–associated outbreaks, cases, and median number of cases, by etiology — United States, 2000–2014

Etiology	Outbreaks no. (%)	Cases no. (%)	Cases per outbreak median no. (range)
Total	**140 (100)***	**4,958 (100)**	**9 (2–1,341)**
Bacterium	**43 (31)**	**604 (12)**	**5 (2–141)**
Campylobacter	1 (1)	6 (0)	6 (—)†
Escherichia coli	19 (14)	155 (3)	5 (3–45)
Leptospira	6 (4)	74 (2)	3 (2–43)
Plesiomonas shigelloides	3 (2)	7 (0)	2 (2–3)
Shigella	14 (10)	362 (7)	14 (2–141)
Parasite	**25 (18)**	**685 (14)**	**7 (2–220)**
Avian schistosomes	8 (6)	345 (7)	17.5 (4–200)
Cryptosporidium	12 (9)	314 (6)	6.5 (3–220)
Giardia	4 (3)	24 (0)	6 (2–10)
Naegleria fowleri	1 (1)	2 (0)	2 (—)†
Virus	**22 (16)**	**1,491 (30)**	**27.5 (8–597)**
Adenovirus	1 (1)	32 (1)	32 (—)†
Norovirus	21 (15)	1,459 (29)	26 (8–597)
Multiple§	**5 (4)**	**345 (7)**	**56 (45–125)**
Chemical/Toxin	**8 (6)**	**78 (2)**	**8.5 (2–20)**
Algal toxin	7 (5)	75 (2)	9 (2–20)
Copper sulfate	1 (1)	3 (0)	3 (—)†
Unidentified¶	**37 (26)**	**1,755 (35)****	**8 (2–1,341)**

* Outbreak etiology proportion by group sums to >100% because of rounding.
† Not applicable because only one outbreak was nationally reported for that etiology.
§ The five outbreaks categorized as having multiple etiologies included outbreaks of 1) *Shigella* and *Plesiomonas shigelloides*; 2) *Shigella*, norovirus, and *Yersinia enterolytica*; 3) *Shigella*, *Campylobacter*, and norovirus; 4) *Shigella*, *Escherichia coli*, and *Plesiomonas shigelloides*; and 5) *Giardia* and norovirus.
¶ Approximately 1,341 cases were associated with an outbreak with predominantly skin illness caused by an etiology that was unidentified but suspected to be poison ivy when dirt was mixed with water to create an obstacle in an endurance race.
** All outbreaks without a confirmed etiology (e.g., outbreaks with a suspected or unknown etiology) were classified as having an unidentified etiology for this analysis. Unidentified etiology indicates lack of laboratory confirmation but not necessarily absence of traditional epidemiologic and environmental health data indicative of a particular etiology.

outbreaks of known infectious etiology were caused by a diverse array of chlorine-susceptible pathogens, including enteric bacteria, parasites, and viruses. Many of the pathogens that cause outbreaks in untreated recreational water venues rarely cause outbreaks in treated recreational water (e.g., pools) (*2*). Well-operated, treated recreational water venues in which water disinfectant (chlorine or bromine) concentrations are properly maintained are at decreased risk for pathogen transmission. The diversity among the etiologies of untreated recreational water–associated outbreaks also requires different sets of steps swimmers and parents of young swimmers can take to protect themselves and others from illness.

The untreated recreational water–associated outbreaks were predominantly caused by enteric pathogens. Norovirus, *E. coli*, *Shigella*, *Cryptosporidium*, and other enteric pathogens can be transmitted via untreated recreational water when fecally contaminated water is ingested. Swimmers can be a source of fecal contamination if they have a fecal incident in the water or fecal material washes off their bodies. Other sources of fecal contamination include storm water runoff, flooding, sewage overflows, sewage treatment plant discharges, septic systems, boating waste, and animal waste on or near a beach. *E. coli* and *Cryptosporidium* contamination can be introduced by human or animal feces; norovirus and *Shigella* are indicative of human fecal contamination. Swimming in untreated recreational water that is shallow, poorly circulating, or overcrowded; frequented by children aged <5 years with no or limited toileting skills; without adequate, easily accessible, and well-stocked hygiene facilities (e.g., toilets or diaper-changing stations); or swimming soon after heavy rain can increase risk for exposure to enteric pathogens.

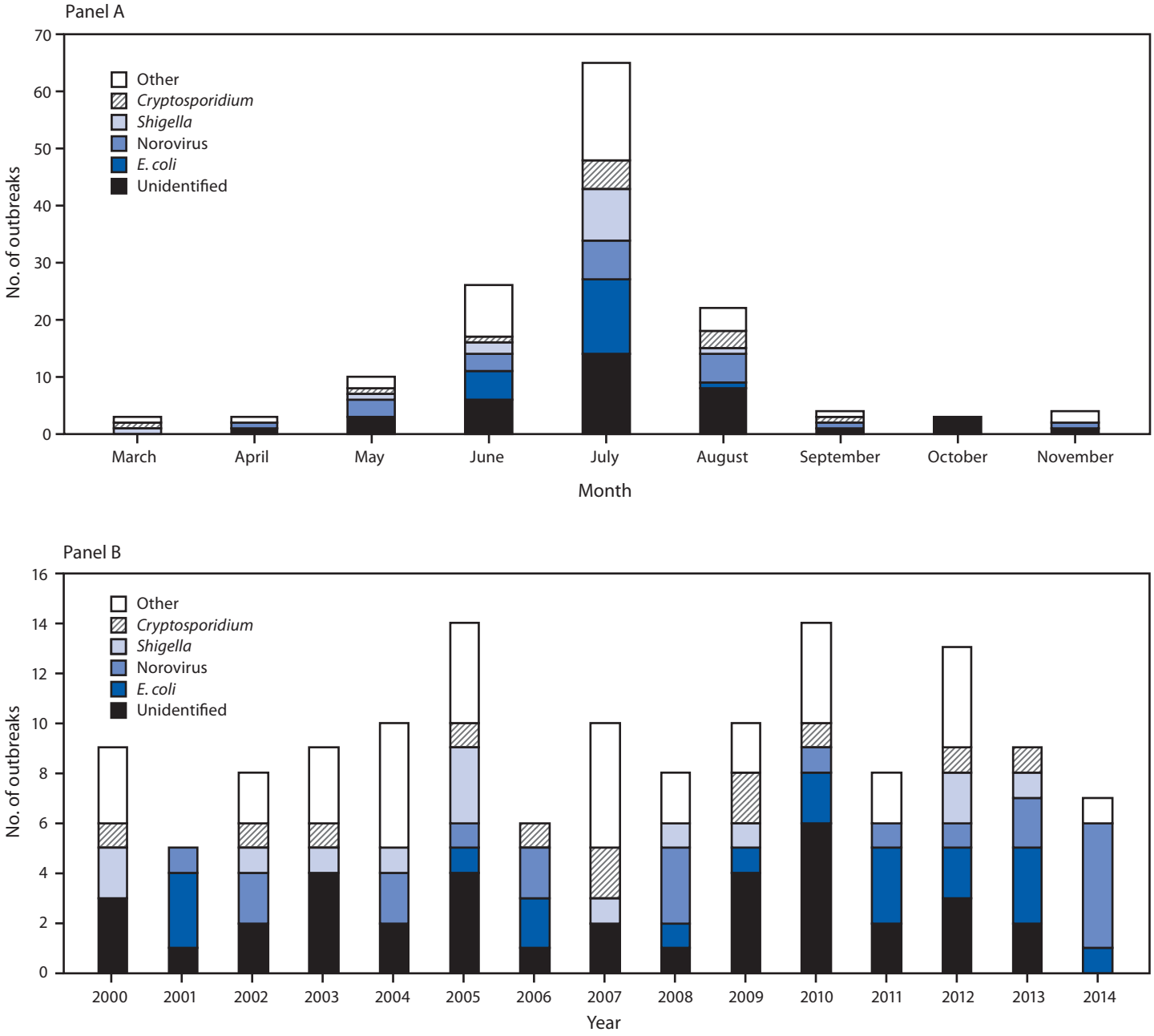
Other etiologies identified in this summary are unique to untreated recreational water. Avian schistosomes can cause cercarial dermatitis (swimmer's itch) in persons exposed to either freshwater or brackish water in which infected birds contaminate the water and where the intermediate host snails are found. Cercarial dermatitis appears as a skin rash and is caused by an allergic reaction when cercariae in the water penetrate the skin. However, the cercariae do not mature into adult worms in humans, who are accidental hosts.

Algal toxins produced by harmful algal blooms in freshwater or marine water can cause a range of illnesses, from skin or eye irritation to respiratory, gastrointestinal, or neurologic symptoms depending on type of toxin and the route of exposure. In recent years, harmful algal blooms have been observed with increasing frequency and in more locations in the United States, possibly because of increasing nutrient pollution and warming water or improved surveillance (*3*). In 2016, CDC launched the One Health Harmful Algal Bloom System¶¶ an electronic system that allows state and territorial public health agencies and their partners to report cases of human or animal illness or environmental data on harmful algal blooms. A better understanding of harmful algal blooms is needed to optimize prevention of associated illness and harmful algal blooms.

Naegleria fowleri causes primary amebic meningoencephalitis after water containing the ameba enters the body through the nose and the ameba travels to the brain via the olfactory nerve. Infection, which is usually fatal, typically occurs when persons swim or dive in warm, untreated freshwater. The recent survival of two U.S. patients with primary amebic meningoencephalitis suggests that early diagnosis and treatment might improve outcomes (*4*). Steps can be taken by swimmers and parents of young swimmers to minimize exposure to enteric pathogens, avian schistosomes, algal toxins, and *Naegleria fowleri* in untreated recreational water (Box).

¶¶ https://www.cdc.gov/habs/ohhabs.html.

FIGURE. Number* of untreated recreational water–associated outbreaks by etiology and month (panel A) and year (panel B) — United States, 2000–2014†



Abbreviation: *E. coli* = *Escherichia coli*.
* N = 140.
† Other includes all outbreaks of confirmed etiology other than *Cryptosporidium*, *E. coli*, *Shigella*, or norovirus.

The findings in this report are subject to at least three limitations. First, the outbreak counts presented likely underestimate actual disease incidence, in part because of variation in public health capacity and reporting requirements across jurisdictions. In addition, untreated recreational water–associated outbreaks might be difficult to detect given that persons who travel long distances to untreated recreational water venues might become ill after returning to geographically dispersed homes in multiple public health jurisdictions, so that the illnesses are never linked to a common exposure (*5*). Entering freshwater and marine water has been associated with a wide range of illnesses despite an absence of reported outbreaks (*5*). Second, for this analysis,

BOX. Preventing exposure to germs and harmful algal bloom toxins in untreated recreational water

Stay out of the water if

- Beach is closed or an advisory is posted for high bacterial levels or other conditions, such as sewage spills or harmful algal blooms.
- A recent heavy rain has occurred.
- A discharge pipe can be seen on the beach.
- Fish or other animals in or near the water are dead.
- Water is discolored, smelly, foamy, or scummy.

Diarrhea-causing germs

- Don't swim or let children swim if sick with diarrhea.
  - If diarrhea is caused by *Cryptosporidium*, wait until 2 weeks after diarrhea has stopped to go swimming.
- Don't swallow recreational swimming water.

https://www.cdc.gov/healthywater/swimming/swimmers/steps-healthy-swimming.html.

Avian schistosomes

- Don't swim near or wade in marshy areas where snails are commonly found.
- Towel dry or shower immediately after exiting the water.

https://www.cdc.gov/parasites/swimmersitch/.

Harmful algal blooms

- Avoid water that contains harmful algal blooms (when in doubt stay out).
- Keep children and pets from drinking discolored, smelly, foamy, or scummy water.
- Get out and rinse off with clean water as soon as possible after swimming in water that might contain a harmful algal bloom.
- Rinse off pets, especially dogs, immediately if they swim in discolored, smelly, foamy, or scummy water. Do not let them lick the algae off their fur.

https://www.cdc.gov/habs/prevention-control.html.

Naegleria fowleri

The only certain way to prevent a *Naegleria fowleri* infection caused by swimming is to refrain from water-related activities in warm freshwater. To reduce exposure risk

- Use nose clips, hold your nose shut, or keep head above water when taking part in water-related activities in bodies of warm freshwater.
- Avoid putting your head under the water in hot springs and other untreated thermal waters.
- Avoid water-related activities in warm freshwater during periods of high water temperature.

https://www.cdc.gov/parasites/naegleria/prevention.html.

Summary

What is already known about this topic?

Untreated recreational water–associated outbreaks can be caused by pathogens, toxins, or chemicals in freshwater (e.g., lakes) or marine water (e.g., ocean).

What is added by this report?

During 2000–2014, 140 untreated recreational water–associated outbreaks that caused at least 4,958 illnesses and two deaths were reported; 80 outbreaks were caused by enteric pathogens.

What are the implications for public health practice?

Swimmers should heed posted advisories closing the beach to swimming; not swim in discolored, smelly, foamy, or scummy water; not swim while sick with diarrhea; and limit water entering the nose when swimming in warm freshwater.

all outbreaks without a laboratory-confirmed etiology (e.g., outbreaks with a suspected or unknown etiology) were classified as having an unidentified etiology. Unidentified etiology therefore does not necessarily indicate absence of traditional epidemiologic and environmental health data indicative of a particular etiology. Finally, reporting and review procedures changed over time, which affects the ability to compare data across years.

Given the connections among swimmer health, animal health, and the environment, preventing untreated recreational water–associated outbreaks requires a One Health*** approach. Collaboration among those with expertise across multiple disciplines (including epidemiologists, environmental health practitioners, veterinarians, and ecologists) and multiple sectors working at the human-animal-environment interface should focus on taking steps to prevent and remediate fecal contamination of the water (e.g., prevent sewage overflows and increase water circulation through engineering), manage wildlife (e.g., encourage birds to leave the beach area) and other animals, properly monitor water quality for bacterial concentration and nutrient pollution (which promotes harmful algal blooms), and encourage a robust monitoring and notification program for untreated recreational waters (*6*). Sections of the BEACH Act of 2000††† allow the Environmental Protection Agency to provide grants to coastal and Great Lakes authorities to monitor their beaches and notify the public of potentially unsafe water quality conditions. The related Beach Advisory

*** https://www.cdc.gov/onehealth.

††† Coastal Recreation Water Quality Monitoring, 33 U.S.C. Sect 1346 (2006). https://www.gpo.gov/fdsys/pkg/USCODE-2011-title33/pdf/USCODE-2011-title33-chap26-subchapIV-sec1346.pdf; Report on Coastal Recreation Waters, 33 U.S.C Section 1375a (2000). https://www.gpo.gov/fdsys/pkg/USCODE-2010-title33/pdf/USCODE-2010-title33-chap26-subchapV-sec1375a.pdf.

and Closing Online Notification[§§§] database provides a resource for swimmers to obtain information on water conditions. However, these are limited to coastal/marine and Great Lakes beaches, whereas most reported outbreaks are associated with smaller, inland lakes, reservoirs, and ponds. This requires swimmers and parents of young swimmers to check for local beach advisories and water conditions in addition to following the steps of healthy swimming. The prevention of untreated recreational water outbreaks includes actions such as engaging and educating the public about healthy swimming, and disseminating healthy swimming messages, particularly before and during June–August. These include heeding posted advisories closing the beach to swimming; not swimming in discolored, smelly, foamy, or scummy water; not swimming while sick with diarrhea; and limiting water entering the nose when swimming in warm freshwater.

[§§§] https://watersgeo.epa.gov/beacon2/.

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State, territorial, local, and Freely Associated State waterborne disease coordinators, epidemiologists, and environmental health practitioners.

Conflict of Interest

CDC receives funding from the Great Lakes Restoration Initiative (a program administered by the Environmental Protection Agency) to support public health initiatives focused on the Great Lakes region. The Great Lakes Restoration Initiative had no involvement in the data collection, analysis, drafting, or review of this manuscript. No other conflicts of interest were reported.

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Geographic Variation in Pediatric Cancer Incidence — United States, 2003–2014

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Approximately 15,000 persons aged <20 years receive a cancer diagnosis each year in the United States (*1*). National surveillance data could provide understanding of geographic variation in occurrence of new cases to guide public health planning and investigation (*2*,*3*). Past research on pediatric cancer incidence described differences by U.S. Census region but did not provide state-level estimates (*4*). To adequately describe geographic variation in cancer incidence among persons aged <20 years in the United States, CDC analyzed data from United States Cancer Statistics (USCS) during 2003–2014 and identified 171,432 cases of pediatric cancer during this period (incidence = 173.7 cases per 1 million persons). The cancer types with the highest incidence rates were leukemias (45.7), brain tumors (30.9), and lymphomas (26.2). By U.S. Census region, pediatric cancer incidence was highest in the Northeast (188.0) and lowest in the South (168.0), whereas by state (including the District of Columbia [DC]), rates were highest in New Hampshire, DC, and New Jersey. Among non-Hispanic whites (whites) and non-Hispanic blacks (blacks), pediatric cancer incidence was highest in the Northeast, and the highest rates among Hispanics were in the South. The highest rates of leukemia were in the West, and the highest rates of lymphoma and brain tumors were in the Northeast. State-based differences in pediatric cancer incidence could guide interventions related to accessing care (e.g., in states with large distances to pediatric oncology centers), clinical trial enrollment, and state or regional studies designed to further explore variations in cancer incidence.

USCS includes incidence data from CDC's National Program of Cancer Registries (NPCR) and the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program (*1*). Data on new cases of cancer diagnosed during 2003–2014 were obtained from population-based cancer registries affiliated with NPCR and SEER programs in all U.S. states and DC. This study included incidence data for all registries that met USCS publication criteria* during 2003–2014, which represented >99% of the U.S. population, excluding data only from Nevada, which did not meet criteria in 2011. This report includes all cases of malignant† cancer diagnosed among persons aged <20 years; it includes first primary cases only and excludes recurrent cases. Diagnosis histology and primary site were grouped according to the *International Classification of Childhood Cancer* (ICCC).§

Pediatric cancer rates were expressed per 1 million persons and were age-adjusted to the 2000 U.S. standard population.¶ Rates were estimated by sex, age group, race/ethnicity, state, U.S. Census region,** county-level economic status, county-level rural/urban classification, and ICCC group.

During 2003–2014, CDC identified 171,432 new cases of pediatric cancer (Table 1). Overall incidence was 173.7 cases per 1 million population. The cancer types with the highest incidence rates were leukemias (45.7 per 1 million), brain tumors (30.9), and lymphomas (26.2). Rates were higher in males (181.5) than in females (165.5) and in persons aged 0–4 years (228.9) and 15–19 years (213.3) than in persons aged 5–9 years (122.6) and 10–14 years (133.0). Among all racial/ethnic groups, the highest incidence rate was among whites (184.4), and the lowest was among blacks (133.3).

Rates were highest in the Northeast U.S. Census region, followed by the Midwest, the West, and the South. Rates were highest in the Northeast across all age groups and among whites and blacks. Among Hispanics, rates were highest in the South. Pediatric cancer incidence rates were highest in the 25% of counties with the highest economic status and were higher in metropolitan areas with populations ≥1 million than in nonmetropolitan areas.

By state, pediatric cancer incidence rates ranged from 145.2–205.5 per 1 million. Rates were highest in New Hampshire (205.5), DC (194.0), and New Jersey (192.3) and lowest in South Carolina (149.3) and Mississippi (145.2) (Table 2). Incidence among whites ranged from 157.0 in Montana to 255.2 in Hawaii; among blacks, from

* Cancer registries' incidence data met the following five USCS criteria: 1) ≤5% of cases ascertained solely on the basis of death certificate; 2) ≤3% of cases missing information on sex; 3) ≤3% of cases missing information on age; 4) ≤5% of cases missing information on race; and 5) ≥97% of registry's records passed a set of single-field and interfield computerized edits that test the validity and logic of data components. https://nccd.cdc.gov/uscs/.

† Used behavior code = 3. https://seer.cancer.gov/behavrecode/.

§ https://seer.cancer.gov/iccc/iccc-who2008.html and https://onlinelibrary.wiley.com/doi/full/10.1002/cncr.20910. The ICCC applies the rules and nomenclature of the *International Classification of Diseases for Oncology, Third Edition*: http://codes.iarc.fr/.

¶ Population estimates incorporate bridged single-race estimates derived from the original multiple race categories in the 2010 U.S. Census. https://seer.cancer.gov/popdata.

** https://www.census.gov/geo/reference/gtc/gtc_census_divreg.html.

TABLE 1. Age-adjusted incidence rate* of cancer† among persons aged <20 years, by U.S. Census region§ — United States,¶ 2003–2014

			U.S. Census region							
	Total		Northeast		Midwest		South		West	
Characteristic	No.	Rate (95% CI)	No.	Rate (95% CI)	No.	Rate (95% CI)	No.	Rate (95% CI)	No.	Rate (95% CI)
Overall	**171,432**	**173.7 (172.9–174.5)**	**31,893**	**188.0 (185.9–190.0)**	**37,702**	**172.9 (171.1–174.6)**	**61,998**	**168.0 (166.7–169.3)**	**39,839**	**172.9 (171.2–174.6)**
Sex										
Male	**91,667**	**181.5 (180.3–182.7)**	16,860	194.5 (191.6–197.5)	20,228	180.3 (178.8–182.8)	33,045	175.1 (173.3–177.0)	21,534	182.3 (179.9–184.8)
Female	**79,765**	**165.5 (164.3–166.6)**	15,033	181.1 (178.2–184.0)	17,474	164.3 (161.6–166.5)	28,953	160.6 (158.7–162.4)	18,305	163.0 (160.7–165.4)
Age group (yrs)										
0–4	**54,419**	**228.9 (227.0–230.8)**	9,467	242.7 (237.9–247.7)	12,001	227.0 (228.3–230.6)	20,161	222.7 (219.7–225.8)	12,790	226.1 (222.2–230.0)
5–9	**29,181**	**122.6 (121.2–124.1)**	5,161	128.7 (125.2–132.3)	6,323	121.2 (116.7–124.6)	10,862	121.4 (119.1–123.7)	6,835	123.2 (120.3–126.1)
10–14	**33,042**	**133.0 (131.5–134.4)**	6,256	145.1 (141.5–148.7)	7,128	131.5 (126.0–134.0)	12,042	130.4 (128.1–132.7)	7,616	131.9 (128.9–134.8)
15–19	**54,790**	**213.3 (211.5–215.1)**	11,009	238.5 (234.0–243.0)	12,250	211.5 (210.0–215.5)	18,933	200.5 (197.7–203.4)	12,598	213.5 (209.8–217.3)
Race/Ethnicity**										
White	**103,650**	**184.4 (183.3–185.5)**	21,580	200.8 (198.1–203.5)	28,309	183.3 (177.7–185.9)	34,798	178.9 (177.0–180.8)	18,963	184.9 (182.3–187.5)
Black	**20,188**	**133.3 (131.5–135.2)**	3,402	143.6 (138.8–148.5)	3,894	131.5 (125.4–135.6)	11,194	131.9 (129.5–134.4)	1,698	132.7 (126.4–139.1)
Hispanic	**36,197**	**168.9 (167.2–170.7)**	4,758	170.0 (165.2–175.0)	3,473	167.2 (153.5–170.2)	13,250	175.5 (172.5–178.5)	14,716	165.6 (162.9–168.3)
AI/AN	**1,507**	**147.6 (140.2–155.2)**	53	93.1 (69.7–121.9)	262	140.2 (118.9–155.2)	450	143.7 (130.7–157.6)	742	162.3 (150.8–174.5)
API	**7,089**	**144.6 (141.2–148.0)**	1,488	151.8 (144.2–159.8)	937	141.2 (133.6–148.0)	1,402	127.7 (121.1–134.6)	3,262	150.4 (145.3–155.7)
County-level economic status by percentile††										
≤25%	**19,536**	**165.7 (163.4–168.0)**	1,848	173.7 (165.9–181.9)	2,888	163.4 (162.3–168.7)	9,902	164.6 (161.3–167.8)	4,898	163.9 (159.3–168.5)
25–75%	**98,385**	**171.3 (170.2–172.4)**	15,032	182.2 (179.3–185.1)	21,073	170.2 (167.2–172.8)	38,515	167.8 (166.2–169.5)	23,765	172.1 (169.9–174.3)
≥75%	**48,268**	**181.8 (180.2–183.4)**	14,996	196.1 (193.0–199.3)	8,894	180.2 (175.8–183.3)	13,252	171.7 (168.8–174.7)	11,126	178.5 (175.2–181.9)
County-level rural/urban continuum††										
Metropolitan population ≥1 million	**93,181**	**177.1 (176.0–178.3)**	21,451	189.2 (186.6–191.7)	15,634	176.0 (171.5–178.0)	31,810	172.0 (170.2–173.9)	24,286	175.9 (173.6–178.1)
Metropolitan population 250,000 to <1 million	**35,919**	**171.1 (169.4–172.9)**	6,283	184.7 (180.2–189.4)	6,290	169.4 (169.1–172.7)	14,186	164.3 (161.6–167.0)	9,160	172.0 (168.5–175.6)
Metropolitan population <250,000	**14,349**	**165.7 (163.0–168.4)**	1,556	183.3 (174.2–192.7)	3,958	163.0 (161.0–168.4)	5,721	162.2 (158.0–166.5)	3,114	164.0 (158.3–169.8)
Nonmetropolitan counties	**22,962**	**167.2 (165.0–169.3)**	2,586	188.8 (181.5–196.3)	6,982	165.0 (165.3–169.3)	10,173	163.0 (159.9–166.2)	3,221	160.8 (155.3–166.4)

Sources: CDC's National Program of Cancer Registries; National Cancer Institute's Surveillance, Epidemiology, and End Results Program.
Abbreviations: AI/AN = American Indian/Alaska Native, API = Asian/Pacific Islander, CI = confidence interval.
* Rates are per 1 million persons and age-adjusted to the 2000 U.S. Standard population.
† Cases included all malignant cancers (with behavior code = 3) as grouped by the *International Classification of Childhood Cancer*.
§ *Northeast:* Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont. *Midwest:* Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, and Wisconsin. *South:* Alabama, Arkansas, Delaware, District of Columbia, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, and West Virginia. *West:* Alaska, Arizona, California, Colorado, Hawaii, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, and Wyoming.
¶ Incidence data are compiled from cancer registries that meet the data quality criteria for all years 2003–2014 (covering >99% of the U.S. population). Nevada is excluded. Registry-specific data quality information is available at https://www.cdc.gov/cancer/npcr/uscs/pdf/uscs-2014-technical-notes.pdf. Characteristic values with other, missing, or blank results are not included in this table.
** White, black, AI/AN, and API persons are non-Hispanic. Hispanic persons might be of any race. Counts exclude unspecified or unknown race/ethnicity.
†† Excludes Kansas, Minnesota, and Nevada.

TABLE 2. Age-adjusted incidence rate* of cancer† among persons aged <20 years, by state, overall and by race/ethnicity — United States,§ 2003–2014

			Race/Ethnicity¶									
	Total		White		Black		Hispanic		AI/AN		API	
State**	No.	Rate (95% CI)	No.	Rate (95% CI)	No.	Rate (95% CI)	No.	Rate (95% CI)	No.	Rate (95% CI)	No.	Rate (95% CI)
Northeast												
Connecticut	**2,060**	**185.8 (177.8–194.0)**	1,399	194.8 (184.7–205.4)	199	144.6 (125.2–166.3)	361	176.8 (159.0–196.1)	—††	—††	63	133.1 (102.2–170.5)
Maine	**725**	**190.5 (176.9–205.0)**	685	194.8 (180.4–210.0)	—††	—††	—††	—††	—††	—††	—††	—††
Massachusetts	**3,584**	**181.5 (175.6–187.5)**	—§§	—§§	—§§	—§§	—§§	—§§	—§§	—§§	—§§	—§§
New Hampshire	**816**	**205.5 (191.6–220.2)**	746	207.6 (192.9–223.2)	—††	—††	31	177.8 (120.6–252.5)	—††	—††	18	157.1 (92.6–249.7)
New Jersey	**5,308**	**192.3 (187.1–197.5)**	3,168	211.8 (204.4–219.3)	633	148.6 (137.2–160.6)	1,043	175.2 (164.7–186.2)	—§§	—§§	345	145.7 (130.7–162.0)
New York	**11,378**	**190.0 (186.5–193.5)**	6,679	209.3 (204.3–214.4)	1,538	147.9 (140.6–155.5)	2,290	175.9 (168.7–183.2)	—§§	—§§	701	164.5 (152.5–177.1)
Pennsylvania	**7,167**	**186.6 (182.3–191.0)**	—§§	—§§	—§§	—§§	494	150.6 (137.6–164.6)	—§§	—§§	—§§	—§§
Rhode Island	**547**	**170.0 (156.0–185.0)**	429	196.3 (177.9–216.0)	28	105.8 (70.2–153.0)	59	96.8 (73.7–124.9)	—††	—††	—††	—††
Vermont	**308**	**164.2 (146.2–183.9)**	299	171.1 (152.0–191.9)	—††	—††	—††	—††	—††	—††	—††	—††
Midwest												
Illinois	**7,227**	**171.8 (167.9–175.8)**	4,320	183.9 (178.4–189.4)	934	124.4 (116.5–132.7)	1,548	171.2 (162.8–180.0)	—§§	—§§	273	146.7 (129.7–165.2)
Indiana	**3,691**	**171.5 (166.0–177.2)**	2,957	178.4 (172.0–185.0)	336	127.6 (114.4–142.1)	296	160.7 (142.7–180.4)	—††	—††	55	139.2 (104.7–181.3)
Iowa	**1,762**	**178.6 (170.4–187.2)**	1,508	181.2 (172.1–190.6)	60	115.7 (88.2–149.1)	130	166.2 (138.6–197.8)	—††	—††	30	140.0 (94.3–200.1)
Kansas	**1,713**	**177.0 (168.8–185.6)**	—§§	—§§	—§§	—§§	254	172.8 (152.0–195.7)	—§§	—§§	—§§	—§§
Michigan	**5,786**	**178.9 (174.3–183.6)**	4,339	188.1 (182.6–193.8)	826	140.5 (131.1–150.4)	296	135.8 (120.7–152.3)	34	127.1 (87.8–178.1)	116	122.3 (101.1–146.8)
Minnesota	**3,109**	**179.9 (173.6–186.3)**	2,420	181.4 (174.3–188.8)	177	122.8 (105.2–142.4)	203	162.6 (140.6–187.0)	46	159.1 (116.4–212.2)	159	162.2 (137.9–189.5)
Missouri	**3,120**	**163.1 (157.4–168.9)**	2,481	168.9 (162.3–175.6)	400	135.8 (122.8–149.8)	139	137.2 (115.0–162.3)	—††	—††	44	116.5 (84.6–156.5)
Nebraska	**1,133**	**183.2 (172.7–194.2)**	868	184.9 (172.8–197.7)	69	161.3 (125.3–204.2)	142	165.8 (139.2–196.0)	—††	—††	20	151.2 (92.2–233.7)
North Dakota	**341**	**158.7 (142.3–176.6)**	295	163.4 (145.2–183.2)	—††	—††	—††	—††	33	174.0 (119.6–244.7)	—††	—††
Ohio	**6,225**	**168.3 (164.1–172.5)**	4,999	175.6 (170.8–180.6)	751	124.5 (115.8–133.7)	206	122.2 (105.9–140.3)	—††	—††	106	147.5 (120.7–178.6)
South Dakota	**413**	**150.3 (136.1–165.5)**	347	162.4 (145.8–180.5)	—††	—††	—††	—††	49	126.9 (93.8–167.8)	—††	—††
Wisconsin	**3,182**	**175.6 (169.5–181.8)**	2,525	181.9 (174.8–189.1)	220	125.1 (109.1–142.7)	247	154.7 (135.7–175.4)	41	181.8 (130.3–246.7)	92	150.1 (120.9–184.1)
South												
Alabama	**2,377**	**157.0 (150.7–163.4)**	1,600	172.2 (163.8–180.8)	619	129.4 (119.4–140.1)	102	124.4 (100.7–152.0)	—††	—††	25	133.2 (86.1–196.8)
Arkansas	**1,523**	**161.7 (153.7–170.1)**	—§§	—§§	—§§	—§§	—§§	—§§	—§§	—§§	—§§	—§§
Delaware	**504**	**180.9 (165.5–197.5)**	—§§	—§§	—§§	—§§	—§§	—§§	—§§	—§§	—§§	—§§
District of Columbia	**306**	**194.0 (172.6–217.3)**	77	215.2 (165.9–274.7)	152	152.0 (128.7–178.2)	28	159.2 (104.6–231.4)	—††	—††	—††	—††
Florida	**9,160**	**169.9 (166.4–173.4)**	4,625	174.8 (169.8–179.9)	1,526	130.9 (124.4–137.6)	2,714	191.8 (184.7–199.2)	—††	—††	165	111.9 (95.5–130.4)
Georgia	**5,291**	**161.9 (157.6–166.3)**	2,884	177.1 (170.7–183.6)	1,556	136.2 (129.5–143.2)	634	166.9 (153.8–180.7)	—††	—††	159	144.2 (122.6–168.4)
Kentucky	**2,377**	**174.4 (167.4–181.5)**	—§§	—§§	—§§	—§§	—§§	—§§	—§§	—§§	—§§	—§§
Louisiana	**2,378**	**156.9 (150.7–163.4)**	1,453	177.7 (168.7–187.1)	753	127.1 (118.2–136.5)	113	164.2 (134.8–198.0)	—††	—††	42	173.9 (125.3–235.1)

See table footnotes on next page.

TABLE 2. (*Continued*) Age-adjusted incidence rate* of cancer† among persons aged <20 years, by state, overall and by race/ethnicity — United States,§ 2003–2014

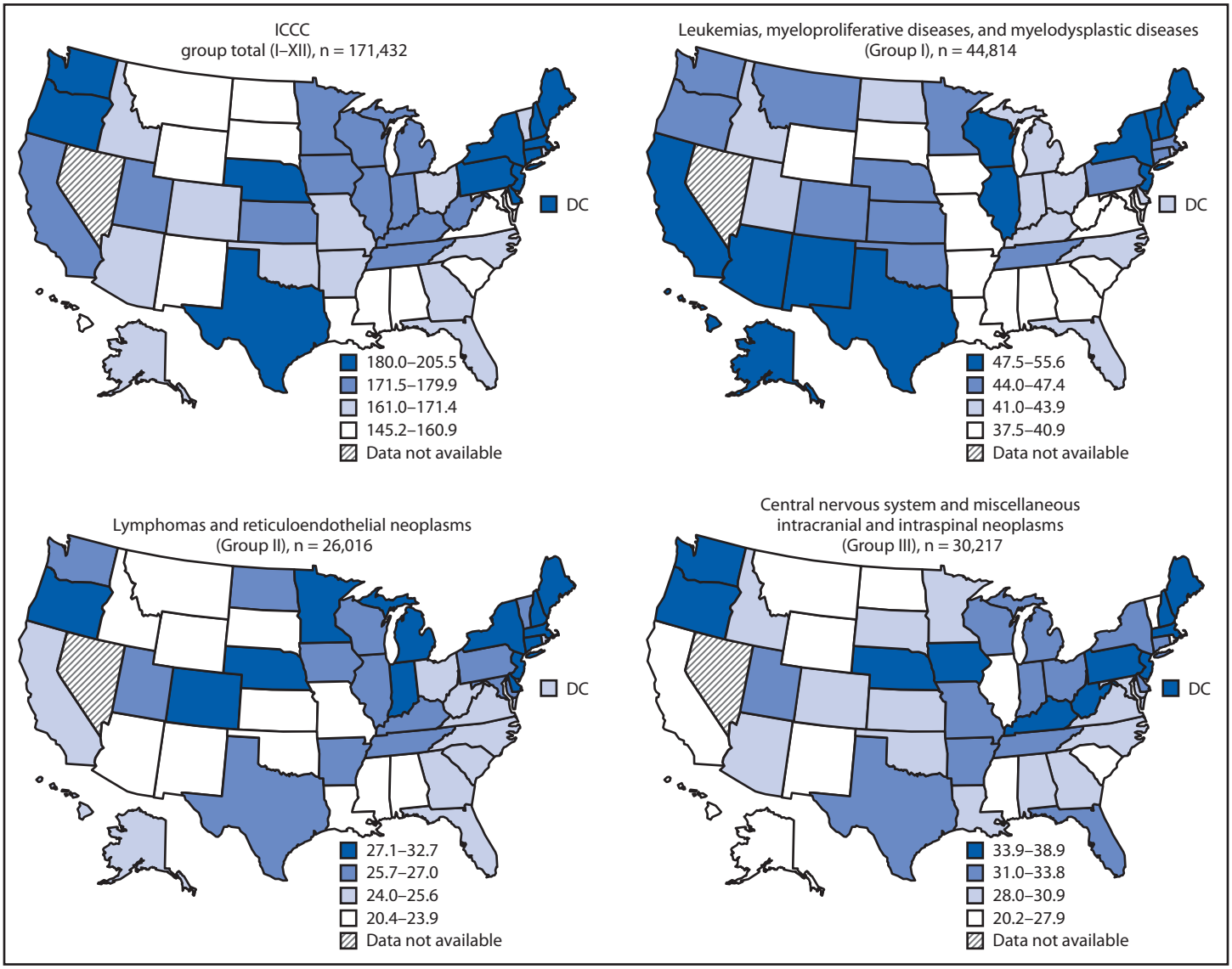
			Race/Ethnicity¶									
	Total		White		Black		Hispanic		AI/AN		API	
State**	No.	Rate (95% CI)	No.	Rate (95% CI)	No.	Rate (95% CI)	No.	Rate (95% CI)	No.	Rate (95% CI)	No.	Rate (95% CI)
Maryland	**2,942**	**160.0 (154.2–165.9)**	1,664	179.7 (171.2–188.6)	773	125.1 (116.4–134.3)	286	156.0 (138.1–175.4)	—††	—††	99	95.1 (77.2–115.8)
Mississippi	**1,476**	**145.2 (137.9–152.8)**	860	166.0 (155.1–177.5)	548	121.7 (111.7–132.4)	45	138.5 (100.2–186.3)	—††	—††	—††	—††
North Carolina	**4,834**	**161.6 (157.1–166.2)**	3,052	175.2 (169.0–181.5)	991	129.3 (121.4–137.7)	560	155.6 (142.6–169.4)	38	88.7 (62.8–121.8)	111	138.6 (113.9–167.1)
Oklahoma	**2,082**	**168.3 (161.1–175.6)**	1,273	166.1 (157.0–175.4)	170	131.0 (112.0–152.2)	276	168.9 (149.2–190.4)	296	194.1 (172.6–217.5)	36	142.5 (99.8–197.4)
South Carolina	**2,162**	**149.3 (143.1–155.8)**	1,370	164.7 (156.1–173.6)	600	122.2 (112.6–132.4)	149	154.4 (130.0–182.0)	—††	—††	24	114.2 (73.1–170.0)
Tennessee	**3,411**	**172.1 (166.4–178.0)**	2,500	180.4 (173.4–187.6)	614	144.5 (133.3–156.4)	211	160.4 (138.7–184.4)	—††	—††	48	142.2 (104.7–188.6)
Texas	**16,368**	**183.2 (180.4–186.0)**	6,598	200.7 (195.8–205.6)	1,571	140.0 (133.1–147.1)	7,503	179.7 (175.6–183.8)	47	162.0 (118.8–216.0)	431	134.0 (121.6–147.3)
Virginia	**3,899**	**156.4 (151.5–161.4)**	2,553	169.2 (162.7–175.9)	710	124.1 (115.1–133.6)	355	139.1 (124.8–154.5)	—††	—††	175	118.2 (101.3–137.1)
West Virginia	**908**	**172.0 (160.9–183.5)**	855	175.4 (163.8–187.5)	28	110.2 (73.1–159.3)	—††	—††	—††	—††	—††	—††
West												
Alaska	**424**	**169.4 (153.6–186.3)**	232	158.0 (138.3–179.7)	—††	—††	25	138.7 (89.5–204.3)	115	217.2 (179.3–260.7)	40	232.0 (165.7–316.0)
Arizona	**3,590**	**168.8 (163.3–174.4)**	1,683	176.1 (167.8–184.7)	130	122.4 (102.2–145.3)	1,454	164.4 (156.1–173.1)	199	164.2 (142.1–188.7)	79	132.7 (105.0–165.5)
California	**21,725**	**173.2 (170.9–175.6)**	7,505	189.9 (185.6–194.2)	1,184	137.9 (130.1–146.0)	10,525	170.1 (166.9–173.4)	101	138.7 (112.8–168.8)	2,187	148.3 (142.1–154.6)
Colorado	**2,767**	**171.3 (165.0–177.8)**	1,754	175.6 (167.4–184.0)	103	121.7 (99.3–147.6)	762	162.4 (151.1–174.5)	20	153.2 (93.2–237.6)	88	171.8
Hawaii	**652**	**160.1 (148.0–172.9)**	134	255.2 (213.7–302.4)	—††	—††	46	75.0 (54.3–101.0)	—††	—††	439	155.6
Idaho	**941**	**170.0 (159.3–181.3)**	789	178.3 (166.0–191.2)	—††	—††	121	136.5 (113.1–163.3)	—††	—††	—††	—††
Montana	**488**	**160.2 (146.2–175.0)**	398	157.0 (141.9–173.2)	—††	—††	24	162.8 (104.0–242.7)	56	182.4 (137.7–237.0)	—††	—††
New Mexico	**1,077**	**157.0 (147.7–166.6)**	393	198.7 (179.5–219.4)	20	126.9 (77.5–196.1)	539	139.7 (128.2–152.0)	101	131.0 (106.7–159.2)	16	186.7 (106.6–303.7)
Oregon	**2,114**	**182.6 (174.9–190.6)**	1,591	192.1 (182.7–201.8)	40	111.6 (79.7–152.0)	343	155.1 (139.0–172.6)	27	134.5 (88.5–196.4)	81	146.1 (116.0–181.6)
Utah	**1,984**	**178.3 (170.5–186.4)**	1,596	182.2 (173.3–191.3)	23	130.1 (82.1–195.9)	309	180.9 (161.1–202.5)	—††	—††	40	120.5 (86.0–164.0)
Washington	**3,797**	**180.7 (175.0–186.5)**	2,656	189.8 (182.6–197.2)	163	135.8 (115.8–158.4)	542	146.9 (134.6–159.9)	83	200.1 (159.3–248.2)	276	158.1 (140.0–177.9)
Wyoming	280	156.8 (139.0–176.3)	232	159.1 (139.3–181.0)	—††	—††	26	118.1 (76.8–173.4)	—††	—††	—††	—††

Sources: CDC's National Program of Cancer Registries; National Cancer Institute's Surveillance, Epidemiology, and End Results Program.
Abbreviations: AI/AN = American Indian/Alaska Native, API = Asian/Pacific Islander, CI = confidence interval.
* Rates are per 1 million persons and age-adjusted to the 2000 U.S. Standard population.
† Cases included all malignant cancers (with behavior code = 3) as grouped by the *International Classification of Childhood Cancer*.
§ Incidence data are compiled from cancer registries that meet the data quality criteria for all years 2003–2014 (covering >99% of the U.S. population). Nevada is excluded. Registry-specific data quality information is available at https://www.cdc.gov/cancer/npcr/uscs/pdf/uscs-2014-technical-notes.pdf.
¶ White, black, AI/AN, and API are non-Hispanic. Hispanic persons might be of any race. Counts exclude unspecified or unknown race/ethnicity; the counts in the total column may not equal the sum of the individual race/ethnicity columns.
** States are grouped by U.S. Census region.
†† Case counts <16 are suppressed.
§§ Race/ethnicity data was suppressed for states that elected to be excluded from race/ethnicity analysis.

105.8 in Rhode Island to 161.3 in Nebraska; and among Hispanics, from 75.0 in Hawaii to 191.8 in Florida.†† Although incidence rates were highest among children aged 0–4 years overall, in some states (e.g., New Jersey, New York, and Illinois), the highest rates were among persons aged 15–19 years (Supplementary Table 1, https://stacks.cdc.gov/view/cdc/53585).

†† State-specific rate ranges by race/ethnicity do not include data suppressed for states that elected to be excluded from race/ethnicity analysis.

FIGURE. Age-adjusted incidence* of cancer† among persons aged <20 years, by U.S. state and ICCC type§ — United States, 2003–2014¶



See figure footnotes on next page.

Pediatric cancer incidence rates varied by state within each cancer type (Figure). Incidence rates were highest in the West for leukemias, myeloproliferative diseases, and myelodysplastic diseases (ICCC group I) and in the Northeast for lymphomas and reticuloendothelial neoplasms (group II) and central nervous system cancers (group III). Rates were also highest in the Northeast for neuroblastoma, retinoblastoma, bone tumors, soft tissue sarcomas, and thyroid cancer (Supplementary Table 2, https://stacks.cdc.gov/view/cdc/53586). Renal cancer rates were highest in the Northeast and South; hepatic tumor rates were highest in the Northeast and West. Germ cell tumor rates were highest in the West (Supplementary Table 2, https://stacks.cdc.gov/view/cdc/53586).

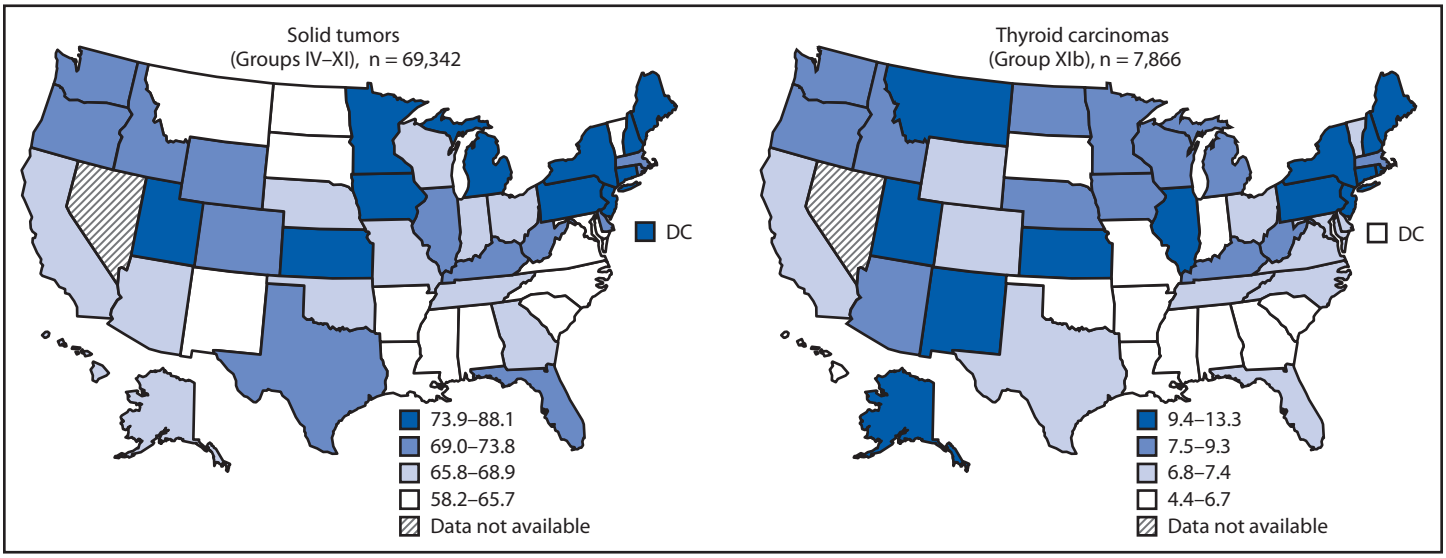
Discussion

This study used recent data with greater population coverage than past studies (*4*,*5*) to document geographic variation in pediatric cancer incidence rates by sex, age, type, and race/ethnicity. Consistent with past reports (*1*,*4*,*5*), pediatric cancer rates were highest in males, persons aged 0–4 years and 15–19 years, whites, and the Northeast U.S. Census region. Rates were highest in metropolitan areas with populations ≥1 million; state-based rates were highest in New Hampshire, DC, and New Jersey.

A strength of this report is the use of extensive population-based surveillance data (>99% coverage§§), which permits a

§§ https://www.cdc.gov/cancer/npcr/uscs/pdf/uscs-2014-technical-notes.pdf.

FIGURE. (*Continued*) Age-adjusted incidence* of cancer† among persons aged <20 years, by U.S. state and ICCC type§ — United States, 2003–2014¶



Sources: CDC's National Program of Cancer Registries; National Cancer Institute's Surveillance, Epidemiology, and End Results Program.
Abbreviation: ICCC = *International Classification of Childhood Cancer*.
* Rates are per 1 million persons and age-adjusted to the 2000 U.S. standard population.
† Cases included all malignant cancers (with behavior code = 3) as grouped by the ICCC.
§ Solid tumors (Groups IV–XI) include neuroblastoma and other peripheral nervous cell tumors, retinoblastoma, renal tumors, hepatic tumors, malignant bone tumors, soft tissue and other extraosseous sarcomas, germ cell and trophoblastic tumors and neoplasms of gonads, and other malignant epithelial neoplasms and melanomas. The ICCC group total map includes 258 cases not classified by ICCC.
¶ Incidence data are compiled from cancer registries that meet the data quality criteria for all years 2003–2014 (covering >99% of the U.S. population). Nevada is excluded. Registry-specific data quality information is available at https://www.cdc.gov/cancer/npcr/uscs/pdf/uscs-2014-technical-notes.pdf.

detailed description of state-based cancer incidence variation. Geographic variation in rates might account for differences in results from previous studies that were based on different populations such as state data (*2*,*3*), SEER registries (which cover 9%–28% of the U.S. population),¶¶ or other large data sets (*6*). A 2016 study specific to Delaware assessed pediatric cancer incidence by demographic group and ZIP Code; the study commented on local environmental exposures and possible incidence disparities based upon sex, age, race/ethnicity, geographic location, and economic status (*2*). USCS data provide states with a standardized way to gauge whether local pediatric cancer incidence rates differ relative to other states and might prompt states to conduct investigations similar to the one performed in Delaware.

Geographic variation in pediatric cancer incidence might be influenced by several factors.*** First, variation in childhood cancer incidence might be related to differences in exposures to carcinogenic chemicals (e.g., air pollution, secondhand smoke, food, or drinking water) or radiation (*7*). Second, genetic variation in certain populations (e.g., prevalence of cancer predisposition genes) (*2*,*4*,*5*) might contribute to geographic differences in cancer incidence. Third, the rates of certain cancer types might vary by race/ethnicity. For example, Hispanic children have the highest rate of the most common type of leukemia, pediatric acute lymphoblastic leukemia, and states with a higher proportion of Hispanics might have higher rates of acute lymphoblastic leukemia (*8*). Fourth, incidence of some types of cancer (e.g., thyroid carcinoma) might be

Summary

What is already known about this topic?

Past research on nationwide pediatric cancer incidence described differences by U.S. Census region but did not provide state-level estimates.

What is added by this report?

During 2003–2014, the pediatric cancer rate was highest in the Northeast, lowest in the South, and highest in metropolitan areas with populations ≥1 million and counties in the top 25% economic status. Incidence rates by state ranged from 145 to 206 per million and were highest in New Hampshire, the District of Columbia, and New Jersey. The highest rate of leukemia was in the West; the highest rates of lymphoma and brain cancer were in the Northeast.

What are the implications for public health practice?

Knowledge of these geographic differences in childhood cancer incidence can be used to enhance provider awareness, treatment capacity, survivorship care, and cancer surveillance.

¶¶ https://seer.cancer.gov/registries/data.html.
*** https://www.cdc.gov/cancer/npcr/uscs/data/00_guidance_include.htm.

related to enhanced detection and access to care, which can vary by geographic location (*5*,*9*).

In addition, geographic variation might be affected by age, economic status, or rural/urban classification (*4*,*8*,*10*). Similar to the findings from this report, recent data detailing adult cancers also indicate that the highest cancer incidence rates are in the Northeast (*10*). Rates of cancer types mostly affecting adults also varied by rural/urban status; some of these differences in adults might be related to factors such as obesity or smoking (*10*), which might or might not also explain rural/urban variation in pediatric cancer.

The findings in this report are subject to at least three limitations. First, Nevada was excluded because data for 2011 did not meet quality criteria, which limits the representativeness of the findings. Second, differences in diagnosis and cancer reporting among states might contribute to variation in cancer incidence rates (*8*). For example, states that were early adopters of electronic pathology reporting might report increased rates because of increased case ascertainment compared with other states. Finally, misrepresentation of race and ethnicity might exist; rate numerators might underestimate American Indians, Alaska Natives, and Hispanics, which could artificially lower rates among these groups; and U.S. Census populations used in rate denominators might undercount children and Hispanics, which could artificially increase rates in these populations (*8*).†††

Knowledge of pediatric cancer incidence variation by state and cancer type can prompt local and state cancer registries to evaluate reporting and diagnostic standards. Understanding geographic variation in incidence rates can help cancer control planners and clinicians address obstacles in access to care, which is especially relevant to states with large distances to pediatric oncology centers (*3*). Because 5-year pediatric cancer survival is >80%, and most cancer survivors require close monitoring by specialists throughout life (*5*), state-specific data by cancer type and patient age might help public health planners address ongoing chronic care needs. In addition, state-specific data by cancer type and patient age might help clinical trial organizers predict patient accrual. Finally, health care practitioners and researchers can use these data to guide investigations related to causes of pediatric cancer incidence variation (*2*,*3*). Continued surveillance will be needed to further validate findings and track geographic incidence patterns over time.

††† https://www.cdc.gov/cancer/npcr/uscs/technical_notes/interpreting/race.htm.

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Conflict of Interest

No conflicts of interest were reported.

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Prevalence and Predictors of Provider-Initiated HIV Test Offers Among Heterosexual Persons at Increased Risk for Acquiring HIV Infection — Virginia, 2016

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Since 2006, CDC has recommended routine, provider-initiated human immunodeficiency virus (HIV) screening (i.e., HIV screening at least once in lifetime) for all patients aged 13–64 years in all health care settings (*1*). Whereas evidence related to the frequency of HIV testing is available, less is known about the prevalence and predictors of providers' HIV test offers to patients (*2*). National HIV Behavioral Surveillance (NHBS) data from Virginia were used to examine the prevalence and predictors of provider-initiated HIV test offers to heterosexual adults aged 18–60 years at increased risk for HIV acquisition. In a sample of 333 persons who visited a health care provider in the 12 months before their NHBS interview, 194 (58%) reported not receiving an HIV test offer during that time, approximately one third of whom (71, 37%) also reported never having had an HIV test in their lifetime. In multivariable analysis, the prevalence of HIV test offers was significantly lower among men than among women (adjusted prevalence ratio [aPR] = 0.72; 95% confidence interval [CI] = 0.53–0.97). Provider-initiated HIV test offers are an important strategy for increasing HIV testing among heterosexual populations; there is a need for increased provider-initiated HIV screening among heterosexual adults who are at risk for acquiring HIV, especially men, who were less likely than women to be offered HIV screening in this study.

NHBS collects HIV prevalence and risk behavior data via anonymous HIV testing and face-to-face interviews, and Virginia conducts NHBS data collection in the Norfolk-Newport News-Virginia Beach Metropolitan Statistical Area (Norfolk MSA) (*2*). In 2016, NHBS used respondent-driven sampling to recruit heterosexual, cis-gendered adults at increased risk for acquiring HIV attributed to heterosexual activity, defined as 1) no injection drug use (IDU) or male-to-male sexual contact in the past 12 months and 2) low socioeconomic status* (*3*). NHBS sampling methods are described in detail elsewhere (*2*,*3*). NHBS data in Virginia were collected during September–December 2016. The outcome of interest, an HIV test offer, was defined as a provider-initiated HIV test offer in the 12 months preceding the NHBS interview. Descriptive statistics of the analytic sample were conducted. Univariable log-binomial regression models were used to examine the association between HIV test offer and demographic (gender, age, race/ethnicity, current relationship status, and health insurance coverage) and behavioral characteristics (high-risk sexual activity,† noninjection drug use in the 12 months preceding the interview, and binge drinking [≥4 and ≥5 drinks in about 2 hours for women and men, respectively] in the past 30 days). All analysis variables, including HIV test offer, were self-reported. Variables associated with HIV test offer with a p-value <0.25 in univariable regression analyses were included in the multivariable, log-binomial regression model. In addition, aPRs for variables significant in the first multivariable regression model were recalculated with potential confounders selected a priori; significance in multivariable models was considered p<0.05. Unadjusted and adjusted prevalence ratios with 95% CIs are reported (*4*).

Face-to-face NHBS interviews were completed with 548 persons aged 18–60 years living in the Norfolk MSA (Figure). After excluding 215 (39%) respondents, including 74 who did not meet the high-risk heterosexual definition of low socioeconomic status and no recent IDU or male-to-male sexual contact, six who self-reported an HIV-positive status, 81 who had not visited a health care provider in the past 12 months, 49 who reported an HIV test >12 months before the interview with no recent high-risk sexual activity or STD diagnoses§ that might warrant retesting, and five who responded "Don't Know" to the HIV test offer question, a final analytic sample of 333 remained.

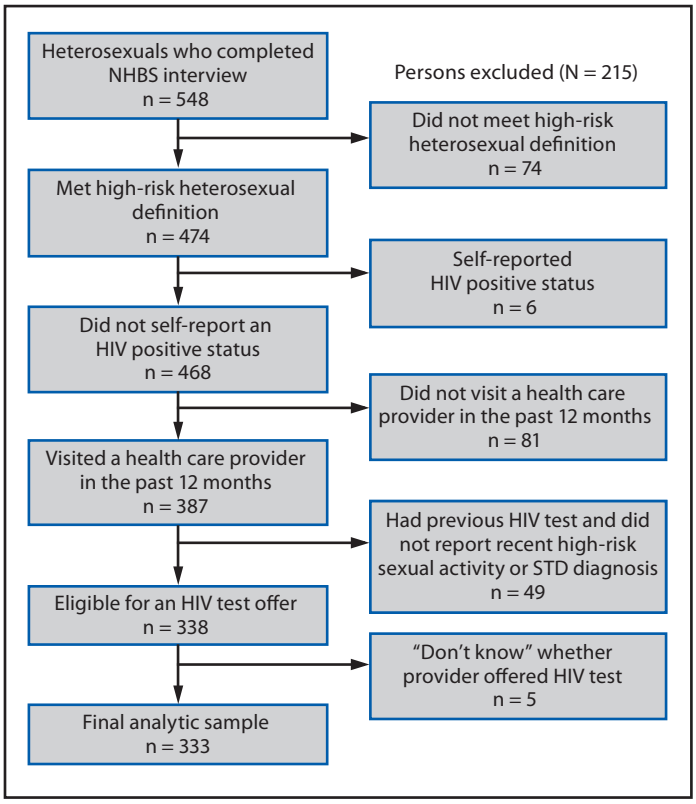
Overall, 139 (42%) persons reported receiving an HIV test offer from a health care provider. Among 194 (58%) persons who reported not receiving an HIV test offer, 156 (80%)

* No more than high school education or income at or below the U.S. Department of Health and Human Services poverty income guidelines A CDC pilot study (2006–2007) indicated socioeconomic status as a strong predictor of HIV prevalence, leading to the incorporation of socioeconomic status into the definition of heterosexuals at increased risk for HIV acquisition for NHBS data collection and analysis purposes. https://www.researchgate.net/publication/232226764.

† High-risk sexual activity was defined as any one of the following in the 12 months before the NHBS interview: any exchange sex, more than one sex partner, sex with a partner who "probably" or "definitely" had other sex partners concurrently, sex with a partner who has "probably" or "definitely" injected drugs, sex with a partner who has "probably" or "definitely" had male-to-male sexual contact, or sex with a partner who is HIV-positive. The high-risk sexual activity variable reflects the 2006 CDC recommendation for repeat HIV screening of all persons likely to be at high risk for HIV.

§ Self-reported chlamydia, gonorrhea, or syphilis diagnosis in the 12 months before the NHBS interview.

FIGURE. Exclusion criteria and selection of a sample of heterosexual adults aged 18–60 years at increased risk for acquiring human immunodeficiency virus (HIV) infection* — National HIV Behavioral Surveillance (NHBS), Virginia Beach-Norfolk-Newport News metropolitan statistical area, 2016



Abbreviation: STD = sexually transmitted disease.
* Persons who met the high-risk heterosexual definition had no injection drug use or male-to-male sexual contact in the past 12 months and either 1) no more than high school education or 2) income at or below the U.S. Department of Health and Human Services poverty income guidelines.

reported high-risk sexual activity, and 71 (37%) reported never having had an HIV test in their lifetime (Table 1). Among persons who received an HIV test offer, 71% reported HIV testing during the 12 months preceding the interview, whereas only 16% of persons not offered an HIV test reported HIV testing during that period (p<0.001). In univariable regression analyses, the following variables were predictive of HIV test offer (p<0.25): gender, age, health insurance coverage, and noninjection drug use. HIV test offer prevalence was lower among men than among women (prevalence ratio [PR] = 0.67; 95% CI = 0.50–0.89) and among persons without health insurance than among those with insurance (PR = 0.78; 95% CI = 0.59–1.03) (Table 2). Compared with persons aged 18–30 years, the prevalence of HIV test offers was higher among those aged 31–40 years (PR = 1.24; 95% CI = 0.89–1.72) and lower among those aged 51–60 years (PR = 0.71; 95% CI = 0.49–1.01). In the multivariable, log-binomial regression model including gender, age, health insurance, and noninjection drug use, only the relationship between gender and HIV test offer was significant (aPR = 0.72; 95% CI = 0.53–0.97). Furthermore, when this relationship was adjusted for potential confounders selected a priori (age, race/ethnicity, current relationship status, health insurance coverage, high-risk sexual activity, noninjection drug use, and binge drinking), men continued to have a significantly lower prevalence of HIV test offers than did women (aPR = 0.69; 95% CI = 0.51–0.93).

TABLE 1. Human immunodeficiency virus (HIV) testing and sexual risk characteristics among 333 heterosexual adults aged 18–60 years at increased risk for acquiring HIV infection, by provider-initiated HIV test offer — National HIV Behavioral Surveillance, Virginia Beach-Norfolk-Newport News metropolitan statistical area, 2016

Characteristic	No. (%)		P-value for chi-squared test statistic
	Received an HIV test offer (n = 139)	Did not receive an HIV test offer (n = 194)	
Ever had an HIV test			
Yes	133 (96)	121 (62)	<0.001
No	6 (4)	71 (37)	
Don't know	0 (0)	2 (1)	
Any HIV testing in past 12 months			
Yes	99 (71)	30 (16)	<0.001
No	40 (29)	164 (84)	
High-risk sexual activity in past 12 months			
Yes	105 (76)	156 (80)	0.287
No	34 (24)	38 (20)	

Summary

What is already known about this topic?

CDC recommends routine, provider-initiated HIV screening (i.e., HIV screening at least once in lifetime) for all patients aged 13–64 years in all health care settings.

What is added by this report?

In a sample of 333 health care–seeking, heterosexual adults at increased risk for acquiring HIV infection, 194 (58%) reported not receiving an HIV test offer at a recent medical visit(s), and men (versus women) had a significantly lower prevalence of provider-initiated HIV test offers (32% versus 48%). Recent HIV testing was higher among recipients of provider-initiated offers compared with nonrecipients (71% versus 16%).

What are the implications for public health practice?

Provider-initiated HIV test offers are an important strategy for increasing HIV testing among heterosexual populations. More provider-initiated HIV screening among heterosexual adults at increased risk for acquiring HIV infection, especially men, is needed.

Discussion

Since 2006, CDC has recommended routine HIV screening for all persons aged 13–64 years (*1*), and from 2006 to 2009, the percentage of adults reporting ever receiving an HIV test

TABLE 2. Predictors of receiving a human immunodeficiency virus (HIV) test offer among heterosexual adults aged 18–60 years at increased risk for acquiring HIV infection — National HIV Behavioral Surveillance, Virginia Beach-Norfolk-Newport News metropolitan statistical area, 2016

Characteristic	No.	Offered HIV test, no. (%)	Received HIV test offer			
			PR (95% CI) (univariable analysis)	PR p-value	aPR (95% CI) (multivariable analysis)	aPR p-value
Sex						
Men	131	42 (32)	0.67 (0.50–0.89)	0.006	0.72 (0.53–0.97)	0.032
Women	202	97 (48)	Referent	—	Referent	—
Age group (yrs)						
18–30	105	47 (45)	Referent	—	Referent	—
31–40	47	26 (55)	1.24 (0.89–1.72)	0.213	1.17 (0.84–1.64)	0.344
41–50	83	35 (42)	0.94 (0.68–1.31)	0.723	0.97 (0.71–1.34)	0.872
51–60	98	31 (32)	0.71 (0.49–1.01)	0.059	0.77 (0.53–1.10)	0.149
Race/Ethnicity						
Black	299	124 (42)	0.94 (0.63–1.40)	0.763	—	—
Other	34	15 (44)	Referent	—	—	—
Current relationship status						
Married/Partnered	51	18 (35)	0.81 (0.54–1.20)	0.292	—	—
Separated/Divorced/Widowed	72	29 (40)	0.92 (0.67–1.27)	0.607	—	—
Never married	210	92 (44)	Referent	—	—	—
High-risk sexual activity in past 12 months						
Yes	261	105 (40)	0.85 (0.64–1.13)	0.271	—	—
No	72	34 (47)	Referent	—	—	—
Noninjection drug use in past 12 months						
Yes	183	82 (45)	1.18 (0.91–1.53)	0.214	1.21 (0.94–1.56)	0.146
No	150	57 (38)	Referent	—	Referent	
≥1 Binge drinking episode in past 30 days						
Yes	117	53 (45)	1.14 (0.88–1.47)	0.327	—	—
No	216	86 (40)	Referent	—	—	—
Health insurance coverage						
Yes	204	93 (46)	Referent	—	Referent	—
No	129	46 (36)	0.78 (0.59–1.03)	0.081	0.86 (0.65–1.13)	0.280
Total	**333**	**139 (42)**	—	—	—	—

Abbreviations: aPR = adjusted prevalence ratio; CI = confidence interval; PR = prevalence ratio.

increased from 40% to 45% (*5*). More recently, NHBS data indicate that among heterosexual adults at increased risk for HIV, the percentage who have ever been tested for HIV has increased (*2*,*6*,*7*). Nevertheless, an estimated 15% of HIV infections are undiagnosed, and missed opportunities for HIV testing remain (*7*). Provider-initiated offers for HIV testing are necessary to increase HIV testing and diagnosis of infection. In the current study, HIV testing during the 12 months preceding an NHBS interview was over three times higher among persons who received a provider-initiated HIV test offer than among those who did not. However, approximately half of heterosexuals at increased risk for HIV infection who sought health care in the 12 months before the interview were not offered an HIV test, and men were significantly less likely to receive a test offer than were women.

For this analysis, persons who reported that their most recent HIV test was >12 months before their interview and who had not experienced recent sexual risk or STD diagnoses were excluded from analysis to focus on heterosexual adults eligible for a provider-initiated HIV test offer. Among this high-risk group, nearly 60% were not offered an HIV test, and among those not offered screening, approximately one third had never received an HIV test in their lifetime. Sexual risk prevalence was high among those who did not report receiving an HIV test offer; thus, increased provider-initiated HIV screening, combined with discussion of preexposure prophylaxis and other HIV prevention strategies as appropriate, is needed (*8*).

Previous studies have reported that HIV testing prevalence is higher among women than among men (*7*,*9*). Similarly, this study found that the prevalence of HIV test offers was higher among female than among male heterosexuals. An ancillary analysis indicated that one quarter of women who received both an HIV test offer and HIV test in the past 12 months had recent testing at a family planning or obstetrics clinic, suggesting the higher prevalence of HIV test offers among women might be related to their participation in family planning services. Nevertheless, previous NHBS data suggest that heterosexual men report more sex partners than do women (*2*,*6*). In addition, men are less likely to seek health care and routine health screens than are women, making HIV screening among men who do seek care essential (*10*).

An important feature of the 2006 CDC guidance was the removal of the recommendation to conduct risk-based HIV screening to reduce barriers to and stigma around HIV screening (*1*). In light of this removal, it was not unexpected that in this analysis, high-risk sexual activity did not significantly predict HIV test offer, reflecting that risk behavior discussion and HIV screening need not be integrated. Nevertheless, repeat screening is recommended among persons considered to be at high risk for acquiring HIV. Although HIV screening and risk assessments need not coincide, exchange of sexual health information between providers and patients is necessary for identifying heterosexual persons in need of repeat screening for HIV.

The findings in this report are subject to at least three limitations. First, the data are cross-sectional, and causality should not be inferred from the results. Second, the data are self-reported during a face-to-face interview and subject to social desirability bias, though it is unlikely this would differ by HIV test offer status. Finally, the sample is composed of persons of low socioeconomic status living in the Eastern region of Virginia, with the majority identifying as black or African American; the results might not be generalizable to other sociodemographic groups. Future work should examine racial/ethnic, regional, and socioeconomic disparities in HIV test offers.

Provider-initiated HIV test offers are an important strategy for increasing HIV testing among heterosexual populations; there is a need for increased provider-initiated HIV screening among heterosexual adults at increased risk for acquiring HIV infection, especially men, who were less likely than were women to be offered HIV screening.

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Conflict of Interest

No conflicts of interest were reported.

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Fatal Sepsis Associated with Bacterial Contamination of Platelets — Utah and California, August 2017

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During August 2017, two separate clusters of platelet transfusion–associated bacterial sepsis were reported in Utah and California. In Utah, two patients died after platelet transfusions from the same donation. *Clostridium perfringens* isolates from one patient's blood, the other patient's platelet bag, and donor skin swabs were highly related by whole genome sequencing (WGS). In California, one patient died after platelet transfusion; *Klebsiella pneumoniae* isolates from the patient's blood and platelet bag residuals and a nontransfused platelet unit were matched using WGS. Investigation revealed no deviations in blood supplier or hospital procedures. Findings in this report highlight that even when following current procedures, the risk for transfusion-related infection and fatality persists, making additional interventions necessary. Clinicians need to be vigilant in monitoring for platelet-transmitted bacterial infections and report adverse reactions to blood suppliers and hemovigilance systems. Blood suppliers and hospitals could consider additional evidence-based bacterial contamination risk mitigation strategies, including pathogen inactivation, rapid detection devices, and modified screening of bacterial culture protocols

Investigation and Results

Utah cluster. In August 2017, two apheresis platelet units and one unit of plasma were manufactured from an apheresis blood donation in Utah. Both platelet units were distributed to hospital X (Figure), where a male (patient A) with acute myeloid leukemia and neutropenia received one of the platelet units. Thirty minutes after transfusion, he developed rigors; transfusion-transmitted bacterial infection was not considered then because of the patient's complex medical history. The patient died 4 days later. Anaerobic blood cultures, obtained shortly after transfusion, grew *C. perfringens* 5 days after collection.

Fourteen hours after patient A's transfusion, a female (patient B) with acute myeloid leukemia received the other platelet unit while on broad-spectrum antibiotics for neutropenia at hospital X. No immediate symptoms of sepsis followed transfusion. Later that day, routine laboratory testing revealed new intravascular hemolysis. Transfusion-transmitted bacterial infection was suspected, and Gram stain of platelet bag residuals was performed, revealing gram-positive bacilli; the platelet supplier was immediately notified. Patient B died 11 hours after transfusion. *C. perfringens* was isolated from an anaerobic culture of the residual platelets. Posttransfusion blood cultures from patient B were negative.

Platelet units transfused to patients A and B had been collected 4 days before transfusion (Figure). Routine inoculation for aerobic culture, performed 24 hours after donation, was negative for bacterial growth through 5 days.

The donor had previously donated platelets and whole blood with no recipient adverse reactions reported. The health department interviewed the donor, who reported no relevant infectious exposures or symptoms. The donor consented to skin swabs, collected from the axillae, antecubital fossae, and anus. Consent for environmental sampling was not provided by the donor.

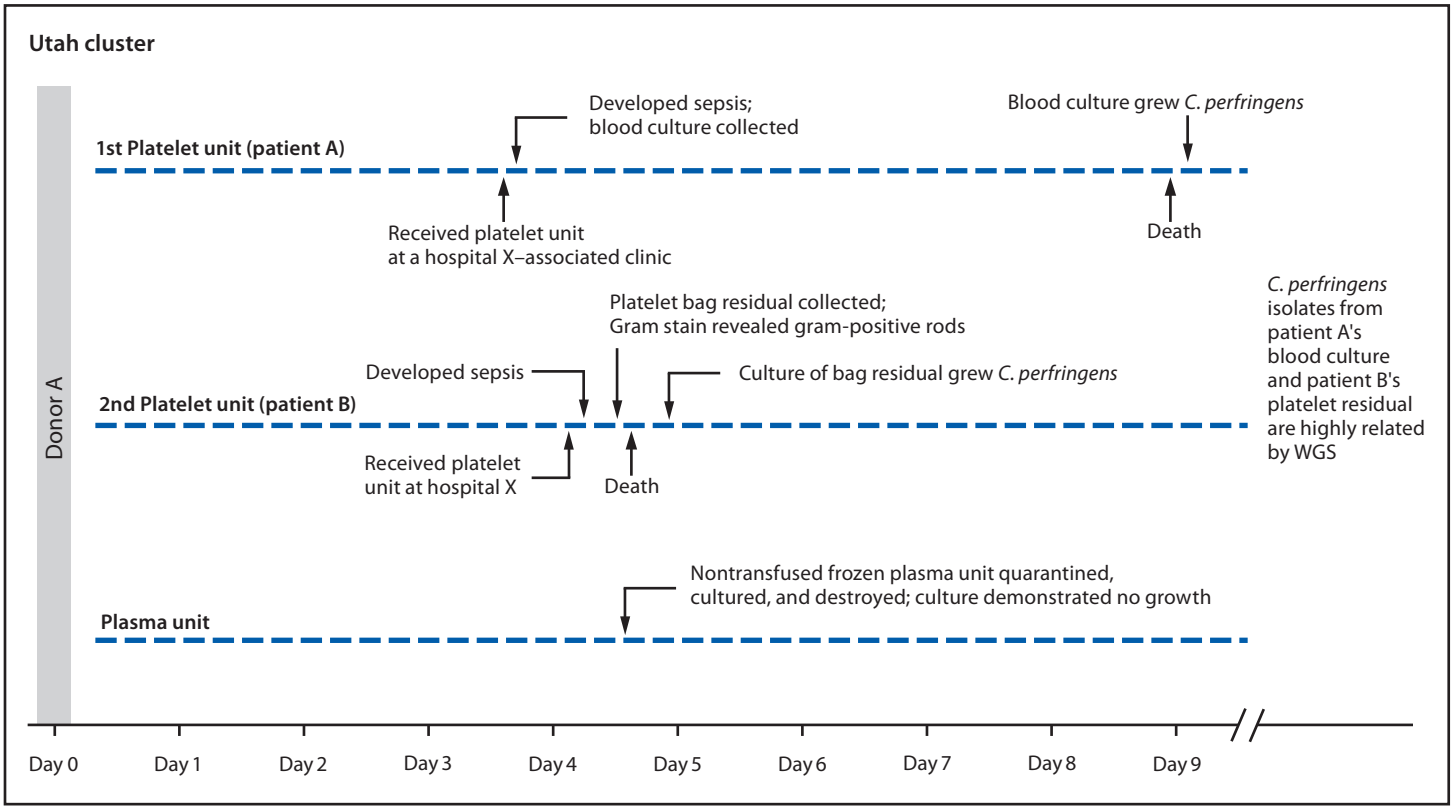
As part of the investigation, multiple samples from the donor, recipients, and platelet bags were cultured for *C. perfringens* under anaerobic conditions. DNA was isolated from cultures that had growth (donor axillae and both antecubital fossae swabs, patient A's blood, two isolates of patient B's platelet bag residual, and one control [an unrelated *C. perfringens* isolate]). WGS indicated all six epidemiologically linked isolates were highly related, with an average pairwise nucleotide difference of 3.35e-10 compared with an average pairwise nucleotide difference of 0.02 to the unrelated control isolate (*1*) (Supplementary Figure 1, https://stacks.cdc.gov/view/cdc/56097).

An investigation of the blood supplier and hospital X revealed no procedural deviations. The nontransfused plasma unit from the donor was quarantined. The donor was permanently deferred.

California Cluster. In August 2017, three apheresis platelet units and one unit of plasma were manufactured from an apheresis blood donation in California. One platelet unit was distributed to hospital Y, where it was divided into two aliquots, and two platelet units were distributed to hospital Z.

At hospital Y, one aliquot was transfused to a male who had received an autologous stem cell transplant (patient C); he developed vomiting, tachycardia, and hypotension approximately 15 minutes after transfusion initiation (Figure). Despite discontinuing transfusion, he died within 5 hours. Multiple posttransfusion blood cultures drawn after the transfusion reaction grew *K. pneumonia*e. Transfusion-transmitted bacterial infection was suspected, and Gram stain of platelet bag residuals was performed, revealing gram-negative rods. The

FIGURE. Timeline of two clusters of sepsis caused by bacterial contamination of platelets — Utah and California, August 2017



See figure footnotes on next page.

blood supplier was immediately notified. *K. pneumoniae* was isolated from the platelet bag residuals.

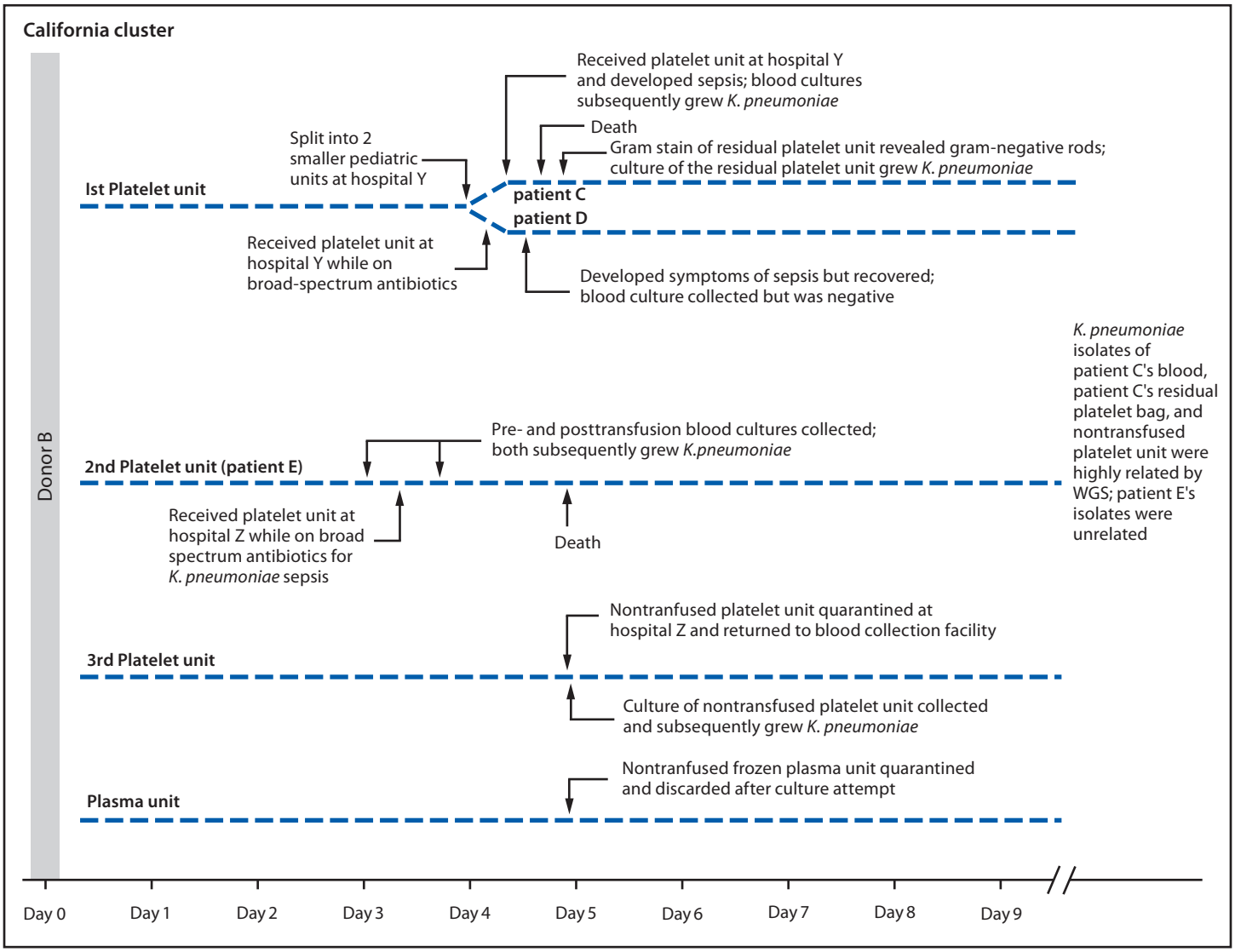
Five hours before patient C's transfusion, hospital Y's second platelet aliquot had been transfused to a male (patient D) with myelodysplastic syndrome, fever, and neutropenia, who was on multiple broad-spectrum antibiotics. Approximately 9 hours after transfusion, the patient developed septic shock but recovered. Multiple posttransfusion blood cultures were negative, presumably a result of the antibiotic regimen.

When the blood supplier notified hospital Z of gram-negative rods identified in the residual aliquot transfused into patient C, the hospital returned a nontransfused platelet unit from which *K. pneumoniae* was later isolated. Hospital Z's other platelet unit had been transfused 1 day before the notification. This platelet unit was transfused to a female (patient E) with disseminated intravascular coagulation and septic shock, for which she was receiving broad-spectrum antibiotics. She died the following day. Blood cultures obtained at the onset of sepsis (pretransfusion) and 8 hours after transfusion both grew multidrug-resistant *K. pneumoniae*.

The routine donor's platelet bacterial screening collection, inoculated 24 hours after donation, was negative for growth through 5 days. The frozen plasma unit was not cultured and was discarded. *K. pneumoniae* isolates from three patient C blood cultures, patient C's residual platelet product, and hospital Z's nontransfused platelets had similar antibiograms and were highly related by WGS, differing by only two single nucleotide polymorphisms (Supplementary Figure 2, https://stacks.cdc.gov/view/cdc/56098). However, pretransfusion and posttransfusion *K. pneumoniae* isolates from patient E demonstrated multidrug resistance and were unrelated from the other isolates using WGS. Patient E's possible source of sepsis was a pretransfusion urine infection with multidrug-resistant *K. pneumoniae*.

Investigation of the blood supplier and hospitals Y and Z indicated no procedural deviations. The donor met eligibility criteria and frequently donated platelets but had been deferred multiple times because of low hemoglobin. A platelet donation 9 months earlier was positive for *Enterobacter cloacae*. After the report of the *K. pneumoniae* cluster, medical history assessments did not identify donor bacterial infection risks. Nontransfused blood products from the implicated donation were quarantined, and the donor was permanently deferred.

FIGURE. (*Continued*) Timeline of two clusters of sepsis caused by bacterial contamination of platelets — Utah and California, August 2017



Abbreviations: *C. perfringens* = *Clostridium perfringens*; *K. pneumoniae* = *Klebsiella pneumoniae*; WGS = whole genome sequencing.

Discussion

Platelet-transmitted bacterial infections persist as a cause of transfusion-associated morbidity and mortality. Contamination of blood products most commonly occurs when skin microbiota are introduced during needle insertion but can also occur from asymptomatic donor bacteremia (*2*). Because the majority of platelets are stored at room temperature, bacteria can proliferate to clinically important levels by the time the unit is transfused (*3*). Approximately one in 5,000 platelet collections are contaminated with bacteria, and one in 100,000 platelet transfusions results in bacterial sepsis (*4*). Transfusion-transmitted bacterial infections are likely underdiagnosed (*2*) because recipients are often given broad spectrum antibiotics or have underlying medical conditions that increase sepsis risk, or the septic reaction might not be attributed to the transfusion.

Current practices to mitigate the risk for bacterial contamination of platelets include donor health screening, skin examination and disinfection, diversion of up to the first 40 mL of blood into a separate nontransfusable pouch to reduce the introduction of skin flora, visual inspection of platelet bags before transfusion, and aerobic bacterial culture screening (e.g., monitoring an aliquot for bacterial growth) at least 24 hours after platelet collection (*5*). Investigations confirmed that the Utah and California collection facilities followed current practices. This report highlights that, even when following current practices, the risk for fatalities persists, making additional, important interventions necessary.

Summary

What is already known about this topic?

Platelet-transmitted bacterial infections persist as a cause of transfusion-associated morbidity and mortality.

What is added by this report?

Whole genome sequencing was used to identify the source of fatal sepsis in three transfusion recipients resulting from bacterial contamination (*Clostridium perfringens* in Utah and *Klebsiella pneumoniae* in California) of platelet products.

What are the implications for public health practice?

Implementation of evidence-based strategies, including pathogen inactivation, rapid detection devices, and modified screening of bacterial culture protocols can mitigate the risk for bacterial contamination of platelets.

The Food and Drug Administration (FDA) has several recommendations related to platelet contamination and donation.* FDA recommends that blood suppliers control the risk for bacterial contamination either by using a pathogen reduction device or performing bacterial detection at least once. Additional requirements when a pathogen is identified include product quarantine, organism identification, determination whether the pathogen is endogenous to the donor blood stream, and, if so, donor deferral.

Additional evidence-based risk mitigation strategies, including pathogen inactivation, rapid detection at point-of-use, and modification of screening bacterial culture protocols, can reduce the risk for platelet-transmitted bacterial sepsis (*3*). Implementation of these modified and alternative strategies in the United States has been supported by advice from the FDA's Blood Products Advisory Committee but are not currently required (*3*). Pathogen inactivation technology was adopted in France, Belgium, and Switzerland, and although no confirmed septic transfusion reactions were reported from 2.3 million pathogen inactivation–treated platelet units, two possible cases have been reported after transfusion of pathogen inactivation–treated platelets (*6*). This same pathogen inactivation technology is approved by FDA for use with apheresis platelets and plasma in the United States.

Rapid bacterial detection devices, optimally used 72 hours after collection, can detect bacteria using <1 mL of platelet volume but only have detection limits of 10^3–10^6 organisms/mL. FDA has cleared one rapid device for extending platelet shelf life from 5 days to 7 days.†

Additional risk mitigation strategies modify existing bacterial culture screening protocols. Current methods differ by blood supplier, with most inoculating 8 mL into an aerobic blood culture microbial detection system sampled ≥24 hours after collection to allow for sufficient bacterial growth. If cultures are negative after 12–24 hours, platelet units are released and have a shelf life of up to 5 days, which can be extended up to 7 days with secondary testing (*3*). However, 8 mL of platelets sampled 24 hours after donation might not have sufficient bacterial loads to detect bacterial growth in the screening culture (*3*). Rather than using a fixed volume, one proposed strategy involves using a minimal proportional sample volume of 3.8% of the platelet total collection (*7*). In the United Kingdom, culture volumes of 16 mL are divided equally between aerobic and anaerobic culture bottles 36–48 hours after donation and have resulted in no recognized fatalities after approximately 1.8 million platelet units were transfused with shelf life extended to 7 days (*8*). However, on several reported occasions, platelet bags were suspected of contamination after visual inspection, and subsequent cultures confirmed contamination. In Ireland, repeat aerobic and anaerobic bacterial cultures are performed 4 days after collection to extend platelet shelf life to 7 days; no septic transfusion reactions have been reported after >100,000 apheresis collections (*3*). Although reporting by blood systems that have adopted modified culture screening methods is promising, demonstrating important clinical benefit is difficult because transfusion-associated bacterial sepsis is rare. However, when compared with current detection practices in the United States, methods based on larger volume culture, delayed sampling of platelets, and performing aerobic and anaerobic cultures after collection are likely to result in fewer cases of platelet-transmitted bacterial infections.

C. perfringens, a sporogenic gram-positive bacterium, has been rarely reported as the source of transfusion-associated sepsis (*4*). Disinfectants used for skin antisepsis during blood collection are not sporicidal and might be ineffective in removing *C. perfringens* from skin. *K. pneumoniae,* a gram-negative bacterium, is a common pathogen among transfusion-related fatalities (*9*). Both pathogens might not be inactivated by pathogen inactivation§ (*10*) but might have been detected with the modified culture strategies described above, which are not routinely practiced in the United States.

Blood collection services could consider implementing enhanced safety interventions to reduce further the risk for bacterial contamination of platelets. Clinicians could consider bacterial contamination when patients develop sepsis during or after a platelet transfusion and rapidly investigate these transfusion reactions.

* Control of Bacterial Contamination of Platelets, 21 C.F.R. Sect. 606.145 (2017). https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/CFRSearch.cfm?fr=606.145; Donation suitability requirements, 21 C.F.R., Sect.630.30 (2017). https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/CFRSearch.cfm?fr=630.30.

† https://www.fda.gov/downloads/BiologicsBloodVaccines/BloodBloodProducts/ApprovedProducts/SubstantiallyEquivalent510kDeviceInformation/UCM551535.pdf.

§ https://www.fda.gov/downloads/BiologicsBloodVaccines/BloodBloodProducts/ApprovedProducts/PremarketApprovalsPMAs/UCM427512.pdf.

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Conflict of interest

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Update of Recommendations for Use of Once-Weekly Isoniazid-Rifapentine Regimen to Treat Latent *Mycobacterium tuberculosis* Infection

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Treatment of latent tuberculosis infection (LTBI) is critical to the control and elimination of tuberculosis disease (TB) in the United States. In 2011, CDC recommended a short-course combination regimen of once-weekly isoniazid and rifapentine for 12 weeks (3HP) by directly observed therapy (DOT) for treatment of LTBI, with limitations for use in children aged <12 years and persons with human immunodeficiency virus (HIV) infection (*1*). CDC identified the use of 3HP in those populations, as well as self-administration of the 3HP regimen, as areas to address in updated recommendations. In 2017, a CDC Work Group conducted a systematic review and meta-analyses of the 3HP regimen using methods adapted from the Guide to Community Preventive Services. In total, 19 articles representing 15 unique studies were included in the meta-analysis, which determined that 3HP is as safe and effective as other recommended LTBI regimens and achieves substantially higher treatment completion rates. In July 2017, the Work Group presented the meta-analysis findings to a group of TB experts, and in December 2017, CDC solicited input from the Advisory Council for the Elimination of Tuberculosis (ACET) and members of the public for incorporation into the final recommendations. CDC continues to recommend 3HP for treatment of LTBI in adults and now recommends use of 3HP 1) in persons with LTBI aged 2–17 years; 2) in persons with LTBI who have HIV infection, including acquired immunodeficiency syndrome (AIDS), and are taking antiretroviral medications with acceptable drug-drug interactions with rifapentine; and 3) by DOT or self-administered therapy (SAT) in persons aged ≥2 years.

Systematic Review

A CDC Work Group including epidemiologists, health scientists, physicians from CDC's Tuberculosis Elimination program, and a CDC library specialist, was convened to conduct the systematic literature review using methods adapted from the Guide to Community Preventive Services (*2*,*3*). The library specialist used a systematic search strategy to identify and retrieve intervention studies on the use of 3HP to treat LTBI that were published from January 2006 through June 2017 and indexed in the MEDLINE, Embase, CINAHL, Cochrane Library, Scopus, and Clinicaltrials.gov databases. To identify missed studies, reference lists from included articles were reviewed, and CDC's TB experts were consulted. This review included English language articles that met the following criteria: 1) the study design was randomized controlled trial, quasi-experimental, observational cohort, or other design with a concurrent comparison group; 2) the target population included, but was not restricted to, persons aged ≥12 years, children aged 2–11 years, or persons with HIV infection; and 3) outcomes reported were prevention of TB disease, treatment completion, adverse events while on 3HP, discontinuation as a result of adverse events while on 3HP, or death while on 3HP.

Two reviewers from the CDC Work Group independently screened citations obtained from the search and retrieved full-text articles in the relevant literature to be synthesized. Using a standard data abstraction form, the reviewers abstracted data on intervention characteristics, outcomes of interest, demographics, benefits, harms, considerations for implementation, and evidence gaps. Each study was also assessed for threats to internal and external validity per Guide to Community Preventive Services standards (*2*,*3*). Any disagreement between reviewers was resolved by consensus of the CDC Work Group members.

The CDC Work Group reviewed 292 citations retrieved from the librarian's search. Of these, 30 full-text articles were ordered and screened for inclusion. No eligible studies including children aged <2 years were identified. In total, 19 articles representing 15 unique studies were included in the meta-analysis. Findings from the meta-analysis indicated that 3HP is as safe and effective as other recommended LTBI regimens and achieves significantly higher treatment completion rates. Complete results of the systematic review and meta-analysis have been published elsewhere (*4*). Overall, the majority of included studies were of greatest design suitability and good quality of execution, as defined by the Guide to Community Preventive Services (*2*,*3*). Issues related to poor reporting of appropriate analytic methods and possible selection bias were the most common limitations assigned to the body of evidence.

Recently published randomized control trials that were heavily weighted in the meta-analyses and drug interaction studies (*5*–*9*) are summarized as follows:

Study of 3HP in children. A large randomized clinical trial of 3HP administered by DOT, which included children aged 2–17 years, demonstrated that 3HP was as well-tolerated and as effective as 9 months of daily isoniazid (9H) for preventing TB (*5*). The trial also reported that 3HP was safe and had higher treatment completion rates than 9H (*5*). Data on the

safety and pharmacokinetics of rifapentine in children aged <2 years are not available.

Studies of 3HP in persons with HIV infection, including AIDS. In 2011, CDC recommended the 3HP regimen for treatment of LTBI in persons with HIV infection, including AIDS, who are otherwise healthy and who are not taking antiretroviral medications (*1*). Since that time, additional data confirm not only the effectiveness of 3HP in persons with HIV infection who are not taking antiretroviral therapy, but also demonstrate the absence of clinically significant drug interactions between once-weekly rifapentine and either efavirenz or raltegravir in persons with HIV infection who are treated with those antiretroviral medications (*4*,*6*–*8*).

Study of self-administered therapy. A randomized clinical trial demonstrating noninferior treatment completion and safety of 3HP-SAT compared with 3HP-DOT in persons aged ≥18 years in the United States provides the primary evidence on 3HP administration by SAT (*9*). The 3HP-SAT regimen has not been studied in randomized controlled trials in persons aged <18 years.

Expert Consultation

In July 2017, CDC met with nine non-CDC subject matter experts in TB and LTBI diagnosis, treatment, prevention, surveillance, epidemiology, clinical research, pulmonology, pediatrics, HIV/AIDS, public health programs, and patient advocacy. CDC presented the systematic review results and proposed recommendations to the experts, who provided 1) individual perspectives on the review; 2) experience with implementation of the 3HP regimen in various settings and populations; and 3) individual viewpoints on the proposed updates. Subject matter experts from programs prescribing 3HP described benefits of this regimen, including increased acceptance and completion of treatment. Some experts reported that several health departments are currently using 3HP, with high treatment completion, in children as young as age 2 years. Some noted that the 2011 recommendation to administer 3HP by DOT limits use of the regimen. In December 2017, CDC solicited input from ACET and members of the public for incorporation into the final recommendations.

With regard to pediatric use, the 2011 recommendations had included limited use of the 3HP regimen for treatment of LTBI in children aged <12 years (*1*). New data on efficacy and safety of 3HP in children were determined sufficient to recommend the 3HP regimen for treatment of LTBI in children aged ≥2 years (*4*).

Concerning patients with HIV infection, information about interactions between specific antimycobacterial agents, including rifamycins (e.g., rifampin, rifabutin, and rifapentine) and antiretroviral agents, is available in the U.S. Department of Health and Human Services Guidelines for the Use of Antiretroviral Agents in HIV-1-Infected Adults and Adolescents. These frequently updated guidelines include a section addressing management of LTBI in persons with HIV coinfection and tables with information on drug interactions.* Use of concomitant LTBI treatment and antiretroviral agents should be guided by clinicians experienced in the management of both conditions.

In 2011, CDC recommended use of the 3HP regimen by DOT (*1*). Treatment completion rates are highest when the regimen is administered by DOT (*4*). However, the burden and expense of DOT is greater than that for SAT (*9*). During the expert consultation and again during review by ACET, some subject matter experts strongly recommended permitting use of SAT, when combined with clinical monitoring, in children aged ≥2 years. Based on this expert opinion, ACET formally recommended expansion of the option of parentally administered SAT to children. Some experts still prefer DOT for treating LTBI in children aged 2–5 years, in whom risk for TB progression and severe disease is higher than that in older children and adults. Health care providers should make joint decisions about SAT with each individual patient (and parent or legal guardian), considering program resources and the patient's age, medical history, social circumstances, and risk factors for progression to severe TB disease. Subject matter experts stressed the importance of educating providers and patients about 3HP.

Recommendations

Based on evidence on effectiveness, safety, and treatment completion rates from the systematic review, and after consideration of viewpoints from TB subject matter experts and input from ACET and the public, CDC continues to recommend 3HP for treatment of LTBI in adults and now recommends use of 3HP 1) in persons with LTBI aged 2–17 years; 2) in persons with LTBI who have HIV infection, including AIDS, and are taking antiretroviral medications with acceptable drug-drug interactions with rifapentine; and 3) by DOT or SAT in persons aged ≥2 years.

The health care provider should choose the mode of administration (DOT versus SAT) based on local practice, individual patient attributes and preferences, and other considerations, including risk for progression to severe forms of TB disease. Use of concomitant LTBI treatment and antiretroviral agents should be guided by clinicians experienced in the management of both conditions (Box 1).

* https://aidsinfo.nih.gov/guidelines/html/1/adult-and-adolescent-arv/367/overview.

BOX 1. Updated recommendations for once-weekly isoniazid-rifapentine for 12 weeks (3HP) for the treatment of latent tuberculosis infection

CDC continues to recommend use of the short-course combination regimen of once-weekly isoniazid-rifapentine for 12 weeks (3HP) for treatment of latent tuberculosis infection (LTBI) in adults. With regard to age limits, HIV infection, and administration of the treatment, CDC now also recommends the following:

- use of 3HP in persons aged 2–17 years;
- use of 3HP in persons with LTBI who are living with human immunodeficiency virus (HIV) infection, including acquired immunodeficiency syndrome (AIDS) and taking antiretroviral medications with acceptable drug-drug interactions with rifapentine*; and
- use of 3HP by directly observed therapy (DOT) or self-administered therapy (SAT) in persons aged ≥2 years; the health care provider should choose the mode of administration (DOT versus SAT) based on local practice, individual patient attributes and preferences, and other considerations, including risk for progression to severe forms of tuberculosis disease.

* https://aidsinfo.nih.gov/guidelines/html/1/adult-and-adolescent-arv/367/overview.

Patient monitoring and adverse events. Hepatic enzymes and other blood tests should be performed for certain patients before initiation of 3HP therapy (Box 2). Approximately 4% of all patients using 3HP experience flu-like or other systemic drug reactions, with fever, headache, dizziness, nausea, muscle and bone pain, rash, itching, red eyes, or other symptoms (*4*,*10*). Approximately 5% of persons discontinue 3HP because of adverse events, including systemic drug reactions (*4*,*10*); these reactions typically occur after the first 3–4 doses, and begin approximately 4 hours after ingestion of medication (*10*). Hypotension and syncope have been reported rarely (two cases per 1,000 persons treated) (*4*,*10*). If symptoms suggestive of a systemic drug reaction occur, patients should stop 3HP while the cause is determined. Symptoms usually resolve without treatment within 24 hours. Neutropenia and elevation of liver enzymes occur uncommonly (*4*,*10*). CDC recommends that health care providers educate patients to report adverse events. Patient use of symptom checklists might facilitate timely recognition and reporting.†

Rifapentine is a rifamycin compound; like rifampin, it induces metabolism of many medications. CDC recommends monitoring of patients when 3HP is prescribed with interacting medications (e.g., methadone or warfarin). Rifapentine can reduce the effectiveness of hormonal contraceptives; therefore, women who use hormonal birth control should be advised to add, or switch to, a barrier method. Women should be advised to inform their health care provider if they decide to try to become pregnant or become pregnant during 3HP treatment.

Because altered dosing might reduce effectiveness or safety, patients on 3HP SAT should be encouraged to record medication intake and report deviations from the prescribed regimen. Persons on 3HP regimens should be evaluated monthly (in person or by telephone) to assess adherence and adverse effects.

Additional studies are needed to understand the pharmacokinetics, safety, and tolerance of 3HP in children aged <2 years; adherence and safety of 3HP-SAT in persons aged <18 years; and safety of 3HP during pregnancy (*4*).

† Examples of patient's medication intake log and symptoms checklists are available at https://www.cdc.gov/tb/publications/pamphlets/12-doseregimen.htm.

BOX 2. Guidance to health care providers during treatment of latent tuberculosis infection (LTBI) with a combination regimen of isoniazid and rifapentine in 12 once-weekly doses (3HP)

- Evaluate all patients for active tuberculosis disease both before and during treatment of LTBI.
- Inform the patient or parents or legal guardians about possible adverse effects and instruct them to seek medical attention when symptoms of possible adverse reaction first appear; particularly drug hypersensitivity reactions, rash, hypotension, or thrombocytopenia.
- Conduct monthly evaluations to assess treatment adherence and adverse effects, with repeated patient education regarding adverse effects at each visit.
- Order baseline hepatic chemistry blood tests (at least aspartate aminotransferase [AST]) for patients with the following specific conditions: human immunodeficiency virus infection, liver disorders, postpartum period (≤3 months after delivery), regular alcohol use, injection drug use, or use of medications with known possible interactions.
- Conduct blood tests at subsequent clinical encounters for patients whose baseline testing is abnormal and for others at risk for liver disease. Discontinue 3HP if a serum AST concentration is ≥5 times the upper limit of normal in the absence of symptoms or ≥3 times the upper limit of normal in the presence of symptoms.
- In case of a possible severe adverse reaction, discontinue 3HP and provide supportive medical care. Conservative management and continuation of 3HP under observation can be considered in the presence of mild to moderate adverse events as determined by health care provider.

Any LTBI treatment–associated adverse effect leading to hospital admission or death should be reported by health care providers to local or state health departments for inclusion in the National Surveillance for Severe Adverse Events Associated with Treatment for LTBI (e-mail: ltbidrugevents@cdc.gov). Serious drug side effects, product quality problems, and therapeutic failures should be reported to the Food and Drug Administration's MedWatch program (https://www.fda.gov/Safety/MedWatch/HowToReport/default.htm) or by telephoning 1-800-FDA-1088.

Additional information regarding 3HP is available at https://www.cdc.gov/tb/publications/ltbi/ltbiresources.htm. Questions also can be directed to CDC's Division of Tuberculosis Elimination by e-mail (cdcinfo@cdc.gov) or by telephoning 800-CDC-INFO (800-232-4636).

Conflict of Interest

No conflicts of interest were reported.

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Notes from the Field

Domestically Acquired Verona Integron-Mediated Metallo-β-Lactamase-Producing Enterobacteriaceae — Indiana, 2016–2017

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Beginning in January 2016, Verona integron-mediated metallo-ß-lactamase (VIM) producing carbapenem-resistant Enterobacteriaceae (CRE) were identified in Indiana. CRE are an emerging antibiotic-resistant public health threat. CRE spread might be largely due to the emergence of carbapenemase-producing CRE (CP-CRE). Carbapenemases are generally encoded on mobile genetic elements that are easily transferred between bacterial strains, greatly increasing their potential for spread (*1–3*). Furthermore, CP-CRE pose a risk because of their extensive drug resistance, increased associated mortality, and national lack of public health laboratory capacity for detection prior to 2016 (*2,4,5*).

The geographic distribution of carbapenemases varies globally. In the United States, the carbapenemase most frequently identified among Enterobacteriaceae is *Klebsiella pneumoniae* carbapenemase; others are less common and are most often identified in patients who recently received health care outside the United States. For example, VIM is frequently identified in southern Europe and Southeast Asia; however, it is infrequently reported from the United States (*1–3*).

In December 2015, the Indiana State Department of Health (ISDH) mandated reporting of CP-CRE, allowing for statewide identification and response to CP-CRE. To facilitate this reporting, the ISDH laboratories hosted CP-CRE workshops in which clinical laboratorians were trained in the detection of carbapenemases via currently available phenotypic testing methods. The ISDH laboratories provide CP-CRE characterization in real time, allowing for timely public health intervention. Upon detection of CP-CRE, the ISDH provides education and technical assistance to health care facilities to ensure rapid implementation of proper infection control procedures. Each patient from whom a CP-CRE isolate is identified is investigated by the local health department to characterize demographics and CP-CRE risk factors, including recent health care exposures and international travel during the preceding 6 months.

During January 2016–December 2017, 649 CP-CRE isolates were reported across Indiana, including nine VIM-producing CP-CRE (VIM-CRE) isolates from seven patients. VIM was the most commonly identified carbapenemase after *Klebsiella pneumoniae* carbapenemase. Seven different species were identified from the nine VIM-producing isolates; one patient was found be colonized or infected with three different VIM-producing organisms over a 15-month period (Table). All seven patients had overnight stays in Indiana health care facilities, and none had documented international travel in the 6 months preceding specimen collection.

Improved isolate submission and expanded capacity to detect carbapenemase-producing organisms have identified VIM-CRE as an emerging resistance problem in Indiana. All patients with VIM-CRE reported recent health care in Indiana but had not traveled outside the country, suggesting VIM transmission within Indiana health care facilities. Notably, although VIM remains one of the least frequently reported carbapenemases among CRE in the United States, Indiana and neighboring states account for 29 (71%) of the 41 VIM-CRE reported to CDC to date, suggesting possible regional emergence of this resistance mechanism (*6*). This finding highlights the important role of state public health laboratories in facilitating identification and reporting of CRE by clinical laboratories and in testing isolates to identify important CRE resistance mechanisms, including all five carbapenemases of major public health concern.* Although such testing has had limited availability in clinical and public health laboratories, recent CDC investments to create the Antibiotic Resistance Laboratory Network have increased carbapenemase testing and CRE screening nationwide. This testing will provide better understanding of CP-CRE epidemiology throughout the United States, including important regional differences in emerging carbapenemases (*6*). Once CP-CRE are identified, health department epidemiologists can work to ensure prompt implementation of infection control interventions. This collaboration between epidemiologists and laboratorians to identify, describe, and respond to emerging drug resistance is needed for containment efforts.

* *Klebsiella pneumoniae* carbapenemase (KPC), New Delhi metallo-beta-lactamase (NDM), Verona integron-mediated metallo-ß-lactamase (VIM), imipenemase (IMP), and oxacillinase-48-like carbapenemase (OXA-48).

Conflict of Interest

No conflicts of interest were reported.

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TABLE. Verona integron-mediated metallo-β-lactamase–producing carbapenem-resistant Enterobacteriaceae (N = 9) isolated from seven patients in health care facilities — Indiana, January 1, 2016–December 31, 2017

Patient	Age (yrs)	Sex	Specimen collection date	Specimen	Organism	Health care exposure history in 6 months preceding specimen collection	Antibiotic use in 6 months preceding specimen collection	Other resistant organisms identified in 6 months preceding specimen collection
1*	36	M	01/19/2016	Wound	*Proteus mirabilis*	ACH	Unknown	CRE, MDR-AB, ESBL
			01/27/2017	Urine	*Klebsiella pneumoniae*	ACH	Yes	CP-CRE, MDR-AB, MRSA
			03/24/2017	Urine	*Providencia rettgeri*	ACH	Yes	CP-CRE, MDR-AB, MRSA
2	28	M	03/21/2016	Wound	*Enterobacter cloacae* complex	ACH	Yes	MRSA
3	67	M	10/01/2016	BAL	*Enterobacter cloacae* complex	ACH, LTACH, LTCF	Yes	CRE, MRSA, MDR-PA, CDI
4	94	F	12/12/2016	Urine	*Klebsiella pneumoniae*	LTCF	Yes	VRE
5	36	F	08/08/2017	Sputum	*Citrobacter freundii complex*	ACH	Yes	None
6	75	M	09/01/2017	Urine	*Klebsiella pneumoniae*	ACH, LTACH	Unknown	None
7	56	F	11/28/2017	Urine	*Klebsiella oxytoca*	ACH	Yes	None

Abbreviations: ACH = acute care hospital; BAL = bronchoalveolar lavage; CDI = *Clostridioides difficile* infection; ESBL = extended-spectrum β-lactamase; F = female; LTACH = long term acute care hospital; LTCF = long term care facility; M = male; MDR-AB = multidrug-resistant *Acinetobacter baumannii* complex; MDR-PA = multidrug-resistant *Pseudomonas aeruginosa*; MRSA = methicillin-resistant *Staphylococcus aureus*; VRE = vancomycin-resistant *Enterococcus*.
* Single patient with multiple isolates.

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Notice to Readers

Ongoing Reanalysis of Suicide Rates Data by Occupational Group from Results Reported in *MMWR*

Recently, *MMWR* Editors were informed by the authors of "Suicide Rates by Occupational Group — 17 States, 2012" (*1*) that some results and conclusions might be inaccurate as a result of coding errors for certain occupational groups. The authors are undertaking a thorough reanalysis of the data. This notice is to alert readers about the coding errors while the reanalysis is conducted to assess the validity of results and conclusions in the publication.

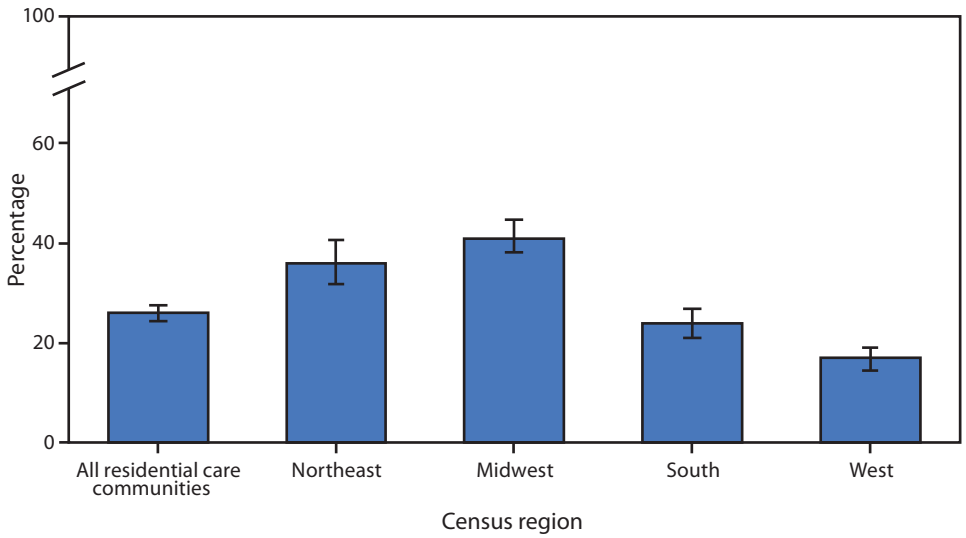
Reference

1. McIntosh WL, Spies E, Stone DM, Lokey CN, Trudeau AR, Bartholow B. Suicide rates by occupational group—17 states, 2012. MMWR Morb Mortal Wkly Rep 2016;65:641–5. https://doi.org/10.15585/mmwr.mm6525a1

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Percentage* of Residential Care Communities† That Use Electronic Health Records,§ by Census Region¶ — United States, 2016



* With 95% confidence intervals indicated with error bars.

† Residential care communities include those that were state-regulated; had four or more beds; and provided room and board with at least two meals a day, around-the-clock on-site supervision, and help with personal care, such as bathing and dressing or health-related services such as medication management. Residential care communities licensed exclusively to serve the mentally ill or the intellectually or developmentally disabled populations were excluded; residential care communities with missing data were excluded.

§ Respondents were asked "An Electronic Health Record is a computerized version of the resident's health and personal information used in the management of the resident's health care. Other than for accounting or billing purposes, does this residential care community use electronic health records?"

¶ The U.S. Census Bureau defines four regions comprising the following states: *Northeast*: Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont; *Midwest*: Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, and Wisconsin; *South*: Alabama, Arkansas, Delaware, District of Columbia, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, and West Virginia; *West*: Alaska, Arizona, California, Colorado, Hawaii, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, and Wyoming.

In 2016, 26% of residential care communities used electronic health records (EHRs). The percentage that used EHRs was 36% of communities in the Northeast, 41% of communities in the Midwest, 24% of communities in the South, and 17% of communities in the West.

Source: National Study of Long-Term Care Providers, 2016 data. https://www.cdc.gov/nchs/nsltcp/index.htm.

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