

## Healthy and Safe Swimming Week — May 22–28, 2017

Healthy and Safe Swimming Week highlights measures that swimmers, parents of young swimmers, aquatic facility (e.g., swimming pool and support infrastructure) operators, residential pool or hot tub/spa owners, beach managers, and public health officials can take to maximize the health benefits of water-based physical activity while minimizing the risk for recreational water-associated illness and injury. A public health communications toolkit is available at <https://www.cdc.gov/healthywater/observances/hss-week/response-tools-public-health.html>.

The theme of this year's observance is Diarrhea and Swimming Don't Mix. *Cryptosporidium*, a parasite that causes profuse, watery diarrhea, has emerged as the leading etiology of recreational water-associated outbreaks, particularly those associated with aquatic facilities (1). This issue of *MMWR* includes a report on *Cryptosporidium* molecular characterization, highlighting its utility in investigating these outbreaks (2).

In July 2016, CDC released the 2016 Model Aquatic Health Code (MAHC) (<https://www.cdc.gov/mahc/editions/current.html>). This national guidance can be voluntarily adopted by state and local jurisdictions to minimize the risk for public aquatic facility-associated illness and injury. The MAHC guidance reflects biennial input from public health professionals and other stakeholders through the Council for the MAHC (<https://www.cmahc.org>).

### References

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2. Hlavsa MC, Roellig DM, Seabolt MH, et al. Using molecular characterization to support investigations of aquatic facility-associated outbreaks of cryptosporidiosis—Alabama, Arizona, and Ohio, 2016. *MMWR Morb Mortal Wkly Rep* 2017;66:493–7.

## Using Molecular Characterization to Support Investigations of Aquatic Facility–Associated Outbreaks of Cryptosporidiosis — Alabama, Arizona, and Ohio, 2016

Michele C. Hlavsa, MPH<sup>1</sup>; Dawn M. Roellig, PhD<sup>1</sup>; Matthew H. Seabolt, MS<sup>1</sup>; Amy M. Kahler, MS<sup>1</sup>; Jennifer L. Murphy, PhD<sup>1</sup>; Taishayla K. McKitt, MPH<sup>2</sup>; Evelyn F. Geeter, MBA<sup>2</sup>; Ron Dawsey, MPH<sup>2</sup>; Sherri L. Davidson, MPH<sup>2</sup>; Thuy N. Kim, MPH<sup>2</sup>; Theresa H. Tucker<sup>2</sup>; Sally Ann Iverson, DVM<sup>3,4,5</sup>; Brenna Garrett<sup>4</sup>; Nicole Fowle<sup>3</sup>; Jennifer Collins, MPH<sup>3</sup>; Gregory Epperson<sup>6</sup>; Scott Zusy<sup>6</sup>; Joli R. Weiss, PhD<sup>4</sup>; Ken Komatsu, MPH<sup>4</sup>; Edwin Rodriguez<sup>7</sup>; J. Gage Patterson, MS<sup>8</sup>; Rebecca Sunenshine, MD<sup>3,9</sup>; Brandi Taylor<sup>10</sup>; Katie Cibulskas<sup>10</sup>; Lynn Denny, MPH<sup>10</sup>; Keoni Omura, MS<sup>11</sup>; Boris Tsorin PhD<sup>11</sup>; Kathleen E. Fullerton, MPH<sup>1</sup>; Lihua Xiao, PhD<sup>1</sup>

Cryptosporidiosis is a nationally notifiable gastrointestinal illness caused by parasitic protozoa of the genus *Cryptosporidium*, which can cause profuse, watery diarrhea that can last up to 2–3 weeks in immunocompetent patients

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and can lead to life-threatening wasting and malabsorption in immunocompromised patients. Fecal-oral transmission of *Cryptosporidium* oocysts, the parasite's infectious life stage, occurs via ingestion of contaminated recreational water, drinking water, or food, or following contact with infected persons or animals, particularly preweaned bovine calves (1). The typical incubation period is 2–10 days. Since 2004, the annual incidence of nationally notified cryptosporidiosis has risen approximately threefold in the United States (1). *Cryptosporidium* also has emerged as the leading etiology of nationally notified recreational water-associated outbreaks, particularly those associated with aquatic facilities (i.e., physical places that contain one or more aquatic venues [e.g., pools] and support infrastructure) (2). As of February 24, 2017, a total of 13 (54%) of 24 states reporting provisional data detected at least 32 aquatic facility-associated cryptosporidiosis outbreaks in 2016. In comparison, 20 such outbreaks were voluntarily reported to CDC via the National Outbreak Reporting System for 2011, 16 for 2012, 13 for 2013, and 16 for 2014. This report highlights cryptosporidiosis outbreaks associated with aquatic facilities in three states (Alabama, Arizona, and Ohio) in 2016. This report also illustrates the use of CryptoNet, the first U.S. molecularly based surveillance system for a parasitic disease, to further elucidate *Cryptosporidium* chains of transmission and cryptosporidiosis epidemiology. CryptoNet data can be used to optimize evidence-based prevention strategies. Not swimming when ill with diarrhea is key to preventing and controlling aquatic facility-associated cryptosporidiosis

outbreaks (<https://www.cdc.gov/healthywater/swimming/swimmers/steps-healthy-swimming.html>).

## Alabama

On August 12, 2016, the Alabama Department of Public Health received a report of 35 persons who developed gastrointestinal symptoms after visiting an Alabama aquatic facility. Case-finding efforts identified 23 outbreak-associated cases. Three (13%) patients had laboratory-confirmed *Cryptosporidium* infection; molecular characterization by CryptoNet of one *Cryptosporidium* specimen identified it as the *C. hominis* IfA12G1R5 subtype. Data collected using an outbreak-specific questionnaire completed for 15 patients indicated the median incubation period was 8 days (range = 5–17 days) after visiting the aquatic facility on July 31. The limited number of completed questionnaires provided insufficient statistical power to determine outbreak risk factors. On August 15, investigators collected filter backwash and water samples directly from the facility's aquatic venues; on August 16, facility operators hyperchlorinated the aquatic venues, raising the free available chlorine concentration for a prolonged period to achieve 3-log<sub>10</sub> (99.9%) *Cryptosporidium* inactivation. Microscopy and real-time polymerase chain reaction (PCR) testing (3) did not detect *Cryptosporidium* in the filter backwash or water samples. An inspection found facility operation and maintenance in compliance with local standards, including water disinfectant concentration and pH standards.

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No fecal incident was known to have occurred in any of the facility's aquatic venues before the outbreak. However, it was recommended the facility develop policies on fecal incident response and maintain a fecal/vomit incident response log.\*

## Arizona

On August 2, 2016, the Arizona Department of Health Services was notified of a cluster of gastrointestinal illness among players on a Coconino County Little League team and family members; 36 (71%) of 51 persons became ill 6–7 days after visiting a Maricopa County aquatic facility on July 22. Molecular characterization by CryptoNet of four *Cryptosporidium* specimens from the Little League cohort identified all four as the *C. hominis* IfA12G1R5 subtype. Maricopa County Department of Public Health simultaneously detected increased laboratory reporting of cryptosporidiosis as of mid-July. Multiple patients reported visits to the same Maricopa County aquatic facility. To determine the magnitude of the outbreak, the counties interviewed cryptosporidiosis patients, focusing on possible risk factors, particularly recreational water exposures. During July 1–October 31, 2016, a total of 352 laboratory-confirmed cryptosporidiosis cases were detected statewide, compared with a median of 46 total cases (range = 42–62) detected annually during 2011–2015. Among 317 interviewed patients, 204 (64%) reported recreational water exposure at 86 public aquatic venues, 74 (86%) of which were in Maricopa County. Environmental health practitioners of affected counties worked with facility operators to hyperchlorinate identified aquatic venues. Among 247 Maricopa County patients interviewed, 43 (17%) reported swimming while symptomatic at a median of one venue (range = 1–3).

## Ohio

During 2012–2015, the Ohio Department of Health and local public health partners detected a median of 399 cryptosporidiosis cases annually statewide (range = 324–571). In 2016, annual incidence increased nearly fivefold to 1,940 cases. Ten (42%) of 24 cryptosporidiosis outbreaks detected in Ohio in 2016† were associated with aquatic venues. Assessing patients' recreational water exposures to determine the magnitude of individual recreational water-associated outbreaks was complicated by individual patients reporting multiple exposures during their incubation period. Among six *Cryptosporidium* specimens from patients affected by

these outbreaks, all were identified by CryptoNet as the *C. hominis* IdA19 subtype, which has rarely been identified in the United States. Five specimens were from a university sports team's members; the sixth specimen was from a patient with no epidemiologic link to the university sports team except visiting the same waterpark. The matching subtype and epidemiologic link led the Ohio Department of Health to classify the 26 cases in the university sports team as part of the waterpark-associated outbreak; these cases previously had been thought to be associated with a different outbreak.

## Discussion

This report highlights cryptosporidiosis outbreaks associated with aquatic facilities in three states in 2016. CryptoNet genotyping (18S PCR-RFLP) to determine *Cryptosporidium* species and subtyping (gp60 PCR and sequencing) to determine subtype (4) supported and strengthened the Alabama, Arizona, and Ohio outbreak investigations. First, molecular characterization identified or confirmed epidemiologic links among individual outbreak-associated cases. Second, and perhaps more importantly, *C. hominis* was repeatedly identified as the outbreak etiology. Given that individual *Cryptosporidium* species can have unique host ranges, identifying the *Cryptosporidium* species can provide insight into possible exposures and outbreak sources. Identifying *C. hominis* as the etiology of these outbreaks indicates a human source of contamination and underscores the need to engage swimmers and parents of young swimmers in efforts to prevent and control aquatic facility-associated cryptosporidiosis outbreaks.

Most *Cryptosporidium* species are indistinguishable by traditional diagnostic tests (microscopy or immunoassays); only molecular diagnostic methods, such as those used by CryptoNet, can distinguish these species and their subtypes. *C. hominis* IfA12G1R5 subtype was identified as the etiology in the Alabama and Arizona outbreak investigations. This subtype was initially identified in small numbers of specimens from sporadic (i.e., not outbreak-associated) cryptosporidiosis cases in the United Kingdom and Australia (5–7). In the United States, it was first seen in specimens from patients with acquired immunodeficiency syndrome (AIDS) and was responsible for a 2009 Oregon cryptosporidiosis outbreak associated with the care of an AIDS patient. Since 2013, it has emerged as the dominant *C. hominis* subtype among sporadic and outbreak-associated cases with *Cryptosporidium* subtyping data; 107 (36.6%) of 292 *Cryptosporidium* specimens from sporadic cases in 2016 were identified as the *C. hominis* IfA12G1R5 subtype.

To better understand the implication of identifying this subtype, molecular characterization of *Cryptosporidium* specimens needs to shift from predominantly supporting outbreak investigations to becoming nationally systematic. In 2010, CDC

\*An example of an aquatic venue water-contamination-response log is available at <https://www.cdc.gov/healthywater/pdf/swimming/pools/water-contamination-response-log.pdf>.

†During 2012–2015, the Ohio Department of Health and local public health partners annually detected a median of six (range = 3–7) cryptosporidiosis outbreaks, a median of one (range = 0–2) were associated with recreational water.

launched CryptoNet (<https://www.cdc.gov/parasites/crypto/cryptonet.html>), the first U.S. molecularly based surveillance system for a parasitic disease. Formal collaborations with state public health partners began in mid-2015. The objectives are to efficiently integrate CryptoNet into existing infrastructure when possible (e.g., merging the CryptoNet BioNumerics infrastructure into that of PulseNet) and to regularly analyze molecular characterization and epidemiologic data for each nationally notified case of cryptosporidiosis to further elucidate *Cryptosporidium* chains of transmission and cryptosporidiosis epidemiology (e.g., geographic and temporal changes in the distribution of *Cryptosporidium* species and their subtypes and associated exposures). Achieving these objectives requires overcoming barriers to successful molecular characterization and sharing epidemiologic data by 1) increasing the positive predictive value of rapid diagnostic tests (i.e., decreasing the frequency of false positive results) (8), 2) shifting away from fixing specimens in formalin (which precludes molecular characterization), 3) advancing molecular diagnostics from single-gene to multilocus or whole-genome sequencing (which will increase discriminatory power), and 4) increasing state capacity to collect and share epidemiologic data with CDC.

The emergence of *Cryptosporidium* as the leading etiology of aquatic facility-associated outbreaks results from the parasite's extreme chlorine tolerance. Free available chlorine inactivates most infectious pathogens within minutes at CDC-recommended concentrations of at least 1 ppm<sup>§</sup>; however, *Cryptosporidium* oocysts can survive for days (9). As the Alabama outbreak investigation indicates, even properly operated and maintained aquatic venues can be sites of *Cryptosporidium* transmission. In addition, cyanuric acid (a stabilizer added to prevent chlorine depletion by the sun's ultraviolet light) has been found to substantially delay chlorine inactivation of *Cryptosporidium* (9). Consequently, in July 2016, CDC issued revised recommendations for hyperchlorination (<https://www.cdc.gov/healthywater/swimming/aquatics-professionals/fecalresponse.html>) when responding to diarrheal incidents in public aquatic venues (i.e., high-risk *Cryptosporidium* contamination events) and aquatic facility-associated cryptosporidiosis outbreaks. These recommendations are also included in CDC's 2016 Model Aquatic Health Code (<https://www.cdc.gov/mahc/editions/current.html>). This national guidance can be adopted voluntarily by state and local jurisdictions and aquatic facilities to minimize the risk for public aquatic facility-associated illness and injury, particularly cryptosporidiosis.

<sup>§</sup>At water pH 7.2–7.8 and temperature 77°F (25°C).

## Summary

### What is already known about this topic?

*Cryptosporidium* has emerged as the leading etiology of recreational water-associated outbreaks, particularly those associated with aquatic facilities (places that contain one or more aquatic venues [e.g., swimming pools, interactive water play venues or water playgrounds, or hot tubs/spas] and support infrastructure [e.g., chemical storage space]).

### What is added by this report?

Most *Cryptosporidium* species are indistinguishable by traditional diagnostic tests (microscopy or immunoassays); only molecular diagnostic methods, such as those used by CryptoNet, the first U.S. molecularly based surveillance system for a parasitic disease, can distinguish these species and their subtypes. Given that individual *Cryptosporidium* species can have unique host ranges, identifying the *Cryptosporidium* species can provide insight into possible exposures and outbreak sources. In the summer of 2016, when detection of cryptosporidiosis outbreaks increased, CryptoNet supported outbreak investigations by further elucidating *Cryptosporidium* chains of transmission.

### What are the implications for public health practice?

Regular analysis of molecular characterization and epidemiologic data through CryptoNet for each nationally notified cryptosporidiosis case can further elucidate *Cryptosporidium* chains of transmission and cryptosporidiosis epidemiology (e.g., by monitoring geographic and temporal changes in the distribution of *Cryptosporidium* species and their subtypes and associated exposures). CryptoNet data can then be used to optimize development of evidence-based prevention strategies. Not swimming when ill with diarrhea is key to preventing and controlling aquatic facility-associated cryptosporidiosis outbreaks (<https://www.cdc.gov/healthywater/swimming/swimmers/steps-healthy-swimming.html>). State and local jurisdictions and aquatic facilities can voluntarily adopt recommendations in CDC's Model Aquatic Health Code (<https://www.cdc.gov/mahc/editions/current.html>) to prevent and control *Cryptosporidium* transmission in public aquatic venues.

Preventing *Cryptosporidium* contamination of water in an aquatic venue would prevent *Cryptosporidium* transmission more efficiently than remediating actions once contamination occurs. This means that public health agencies and the aquatics sector need to collaborate on engaging swimmers, who are the source of contamination, in prevention efforts. Young swimmers aged <5 years are more likely to contaminate the water because they are more likely to have inadequate toileting and hygiene skills; therefore, prevention efforts should focus on their parents. As the Arizona outbreak investigation demonstrated, patients continue to swim while symptomatic. The key healthy swimming message to the public to prevent

contamination is “Don’t swim or let your kids swim if sick with diarrhea.” Health care providers should also instruct cryptosporidiosis patients not to go back into the water until they have been diarrhea-free for 2 weeks.<sup>¶</sup> Healthy swimming promotion campaigns conducted before the summer swim season could reduce the risk for outbreaks caused by *Cryptosporidium* and other enteric pathogens (10), while optimizing the health benefits of water-based physical activity ([https://www.cdc.gov/healthywater/swimming/swimmers/health\\_benefits\\_water\\_exercise.html](https://www.cdc.gov/healthywater/swimming/swimmers/health_benefits_water_exercise.html)).

<sup>¶</sup>The additional 2 weeks are recommended only for patients whose diarrhea is known to be caused by *Cryptosporidium*, because diarrhea caused by *Cryptosporidium* can repeatedly wax and wane before complete resolution, and cryptosporidiosis patients can continue to excrete infectious *Cryptosporidium* oocysts, typically for up to 2 weeks, after symptoms completely resolve.

<sup>1</sup>Division of Foodborne, Waterborne, and Environmental Diseases, National Center for Emerging and Zoonotic Infectious Diseases, CDC; <sup>2</sup>Alabama Department of Public Health; <sup>3</sup>Maricopa County Department of Public Health, Arizona; <sup>4</sup>Arizona Department of Health Services; <sup>5</sup>Epidemic Intelligence Service, CDC; <sup>6</sup>Maricopa County Environmental Services Department, Arizona; <sup>7</sup>Coconino County Public Health Services District, Arizona; <sup>8</sup>Arizona State Public Health Laboratory; <sup>9</sup>Career Epidemiology Field Officer Program, CDC; <sup>10</sup>Ohio Department of Health; <sup>11</sup>Ohio Department of Health Laboratory.

Corresponding author: Michele Hlavsa, [acz3@cdc.gov](mailto:acz3@cdc.gov), 404-718-4695.

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## Inhalational Chlorine Injuries at Public Aquatic Venues — California, 2008–2015

Jason A. Wilken, PhD<sup>1,2</sup>; Michele DiMaggio<sup>3</sup>; Matthew Kaufmann<sup>4</sup>; Kevin O'Connor, PA<sup>1</sup>; Svetlana Smorodinsky, MPH<sup>1</sup>; Christina Armatas, MD<sup>1</sup>; Tracy Barreau<sup>1</sup>; Richard Kreutzer, MD<sup>1</sup>; Lino Ancheta<sup>3</sup>

In June 2015, personnel from California's Contra Costa Health Services Environmental Health and Hazardous Materials (hazmat) divisions were alerted to a possible chemical release at a swimming pool in an outdoor municipal water park. Approximately 50 bathers were in the pool when symptoms began; 34 (68%) experienced vomiting, coughing, or eye irritation. Among these persons, 17 (50%) were treated at the scene by Contra Costa's Emergency Medical Services (EMS) and released, and 17 (50%) were transported to local emergency departments; five patients also were evaluated later at an emergency department or by a primary medical provider. Environmental staff members determined that a chemical controller malfunction had allowed sodium hypochlorite and muriatic acid (hydrochloric acid) solutions to be injected into the main pool recirculation line while the recirculation pump was off; when the main recirculation pump was restarted, toxic chlorine gas (generated by the reaction of concentrated sodium hypochlorite and muriatic acid) was released into the pool. A review of 2008–2015 California pesticide exposure records identified eight additional such instances of toxic chlorine gas releases at public aquatic venues caused by equipment failure or human error that sickened 156 persons. Chemical exposures at public aquatic venues can be prevented by proper handling, storage, and monitoring of pool chemicals; appropriate equipment operation and maintenance; training of pool operators and staff members on pool chemical safety; and reporting of chemical exposures.

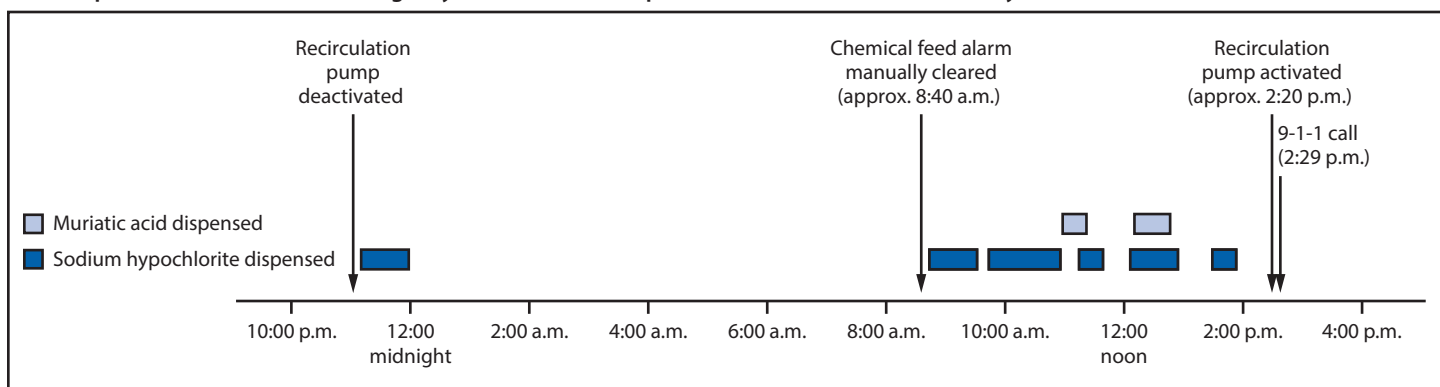
On June 18, 2015, at 2:29 p.m., an initial 9-1-1 call reported 10–12 persons experiencing vomiting or respiratory symptoms at one of five swimming pools at an outdoor municipal water park in Contra Costa County. Contra Costa EMS and fire department personnel were dispatched. At 2:42 p.m., fire personnel requested that hazmat personnel assist in incident response, but at 2:44 p.m., the request was cancelled after fire personnel determined that there was no active chemical leak. At 3:07 p.m., fire personnel again requested hazmat personnel to investigate a possible chemical leak. Hazmat staff members arrived at the water park at 4:14 p.m. and Environmental Health personnel arrived at 4:30 p.m.; both integrated into a fire department–led incident command structure. EMS personnel evaluated and transported patients to local emergency departments. Among the 17 patients transported to an emergency department, 16 (94%) were released the same day;

one patient who was experiencing tachycardia and wheezing was admitted for monitoring and breathing treatments and discharged the next day. Hazmat staff members reviewed the pool chemical controller data and performed air monitoring around the perimeter of the water park, within the immediate vicinity of the affected pool, and at the chemical storage building. Environmental Health staff members measured the free chlorine concentrations and pH of each pool and interviewed municipal water park employees and the pool maintenance contractor.

Hazmat personnel did not detect chlorine in the air during sampling conducted >2 hours after the initial 9-1-1 call. The free chlorine concentration measured in the water of the affected pool >2 hours after the initial 9-1-1 call was 10.5–13.5 ppm, and the pH was 6.8. Both measurements were in violation of California regulations, which allow a maximum of 10 ppm free chlorine and a pH range of 7.2–7.8 (1). The free chlorine concentrations also violated the manufacturer's label instructions that allow a concentration of no greater than 4 ppm in pool water. The pH of the water in two of the other four pools at the park was also <7.0. Environmental Health ordered that the water park and the pool where the exposure incident occurred be immediately closed and remain closed until Environmental Health completed a review of the park's remediation plan for the pool (2).

During normal operations, pool water was drawn from the pool by a recirculation pump. A chemical controller regulated feed of muriatic acid and sodium hypochlorite solutions into the recirculation line, diluting these chemicals and allowing them to mix safely. (Sodium hypochlorite provides the chlorine necessary to inactivate infectious pathogens, and muriatic acid maintains the pool pH within a range that maximizes the chlorine's effectiveness.) The chemical controller was equipped with a rotary flow sensor, interlocked with an accompanying overfeed alarm to prevent chemical feed in the absence of recirculation flow. Review of the chemical controller data identified a recorded zero flow rate of the main recirculation pump (Figure) for approximately 16 hours beginning at 10:40 p.m. on June 17, the day before the incident. During these 16 hours leading up to the incident, the chemical controller also recorded intermittent chemical dispensing of sodium hypochlorite (approximately 81 gallons over a total period of 218 minutes) and muriatic acid (approximately 2 gallons over a total period

**FIGURE. Timeline of events showing deactivation and reactivation of recirculation pump, release of sodium hypochlorite and muriatic acid into the pool water,\* and call to emergency services — Municipal Water Park, Contra Costa County, California, June 17–18, 2015**



\* Caused by an unknown equipment failure.

of 39 minutes) into the recirculation line in the absence of recirculation flow caused by an unknown equipment failure. This allowed these concentrated chemicals to mix and generate toxic chlorine gas. At approximately 8:40 a.m., for unknown reasons, the over-feed alarm was turned off by aquatic staff members. At about 2:20 p.m., aquatic staff members turned on the main recirculation pump, and approximately 10 minutes later EMS received a 9-1-1 call from water park employees.

To characterize such chemical exposures at public aquatic facilities, investigators reviewed California Department of Pesticide Regulation (CDPR) Pesticide Episode Notification Record\* extracts recorded during 2008–2015. Eight additional toxic chlorine releases with multiple persons injured by each release were identified, and Pesticide Episode Closing Reports of each of these incidents were reviewed. Medical records were obtained for nine incidents occurring in 2015 (Table). Among all nine incidents, a total of 155 persons (median = 16 per incident, range = 2–34) were symptomatic (primarily respiratory symptoms, vomiting, and eye irritation), 121 (78%) patients were transported to an emergency department or were evaluated by their primary medical provider (median = 11 per incident, range = 2–27), and five of 70 (7%) persons for whom information on hospitalization was available required hospital admission (median = 1 per incident, range = 0–2). Factors contributing to these incidents included one or more of the following: chemical controller failures, valve failures, or human error. A feature noted in all events was that chemicals were inappropriately dispensed while main recirculation pumps

were deactivated, causing the chemicals to mix at concentrations that resulted in the generation of toxic chlorine gas. In seven of the nine incidents, main recirculation pumps were turned on while bathers were present in pools.

### Discussion

An estimated >50 million persons swim for sport or recreation in the United States each year (3). Proper management of chemical disinfection of pool water is essential to prevent transmission of infectious pathogens. In 2012, an estimated 4,876 visits to emergency departments occurred after pool chemical-associated health events, such as those highlighted in this report (4).

Compliance with California and by Contra Costa County regulations might have prevented the incident described in this report. In addition to regulations defining limits for free chlorine concentrations and pH in pool water, the California Code of Regulations also requires that chemical feeder equipment shall “be maintained and repaired according to manufacturer’s specifications” (5). The California Code of Regulations also requires the recirculation pump to be in operation whenever the public pool is available for use (6). However, no federal agencies regulate public aquatic facility design, construction, operation, and maintenance; rather, these regulations are written, enacted, implemented, and enforced by state and local jurisdictions. CDC and the New York Department of Health have spearheaded development of the Model Aquatic Health Code (MAHC; <https://www.cdc.gov/mahc/editions/current.html>), a science- and best practices-based resource for preventing public aquatic facility-associated illness and injury. MAHC development and maintenance is the result of an ongoing collaboration among federal, state, and local public health officials, and representatives from the aquatics sector. MAHC is updated biennially (<https://www.cmahc.org/>), most

\* Pesticide Episode Notification Records are California Department of Pesticide Regulation (CDPR) enforcement forms completed by staff members at CDPR’s Enforcement Regional Office, which provides oversight to counties when they are either notified by the county of an exposure incident or by the CDPR Pesticide Illness Surveillance Program of an exposure incident that meets the priority status as defined by a cooperative agreement with Environmental Protection Agency Region 9.

TABLE. Chlorine gas exposure incidents at public aquatic venues — California, 2008–2015

Date	County	Pool location/ type	No. persons			Identified cause
			Symptomatic	Evaluated at ED/PMP	Admitted to hospital ≥24 hours	
Oct 2008	Orange	High school swimming	6	6	0	Chemical metering device failed; recirculation pump restarted while bathers in pool
Jun 2009	Los Angeles		7	7	0	Chemical valves not closed during replacement of recirculation pump; recirculation pump replaced and activated while bathers in pool
Jul 2010	Los Angeles	Municipal	30*	17	—†	Attempted to prime a dry chemical line while bathers in pool
Aug 2010	San Mateo	Municipal	2	2	1	Unknown
Nov 2010	Santa Clara		19	11	—†	Flow switch monitor failure; recirculation pump restarted while bathers in pool
Aug 2011	Sacramento	Privately owned	24	24	—†	Chemical controller manually bypassed; recirculation pump restarted while bathers in pool
Jun 2015	Shasta	Privately owned fitness facility	28	27	2	Chemical valve failure; recirculation pump restarted while bathers in pool
Jun 2015	Contra Costa		34	22	1	Chemical controller failure and chemical controller manually bypassed; recirculation pump restarted while bathers in the pool
Oct 2015	Contra Costa	Municipal High school swimming	5	5	1	Chemical controller failure; recirculation pump restarted while bathers in the pool

**Abbreviations:** ED = emergency department; PMP = primary medical provider.

\* Approximate number.

† California Department of Pesticide Regulation records did not indicate whether patients were admitted to a hospital.

recently in 2016. State or local jurisdictions can voluntarily use MAHC sections as a resource and a guide to prevent illness and injury associated with public aquatic venues. MAHC section 5.7.3.5.1.3 (Fail Proof Safety Features) states that equipment must be unable to feed chemicals in the absence of recirculation flow, and MAHC section 4.7.3.2.1.3 (Interlock Controls and No or Low Flow Deactivation) describes criteria for automatic shutoff of chemical feeders by an interlock in the case of interruption of recirculation flow. In the 2015 incident described in this report, according to the manufacturer's specifications, the chemical feeder interlock should have shut off all chemical feeding while the recirculation pump was off but an unknown equipment failure allowed pool chemicals to be delivered intermittently during a 16-hour period. In addition, for unknown reasons members of the aquatic staff turned off the alarm.

The findings in this report are subject to at least three limitations. First, chlorine concentrations in the water and air can fluctuate, and sampling >2 hours after the incident likely did not reflect concentrations at the time the symptoms occurred. Second, in the retrospective analysis, only those incidents reported to CDPR could be identified; other incidents might have occurred and were not reported to CDPR. Finally, medical records associated with incidents before 2015 were not obtained for this analysis.

Despite these limitations, this investigation revealed that the public health impact of toxic chlorine gas releases might be reduced or mitigated by following practices recommended

in MAHC, and the incident described above might have been prevented by the additional following steps. Public aquatic facilities can perform regular challenge tests of chemical feeder interlock systems (e.g., regular measurement of time for the interlock to shut off chemical feeders after the recirculation pump turns off), and conduct these tests when no bathers are in the pool. An audible or visual alarm system can be incorporated to indicate when the recirculation pump is off. It is important that all bathers be evacuated from the aquatic venue if the recirculation pump is off (regardless of reason) or when chemical feeders are deactivated, and that they not be allowed to reenter the aquatic venue until the cause of recirculation pump deactivation has been identified and corrected. Furthermore, it is important that bathers not be allowed to reenter the aquatic venue until water quality measurements return to concentrations allowed by standards. Aquatic staff members can be trained to recognize the signs and symptoms of chlorine exposure and how to respond if noted. Public aquatic venues can develop, train, and test emergency action plans including notification of all applicable local regulatory agencies and emergency response teams. Hazardous materials and environmental health personnel can be promptly integrated into responses to potential chemical exposures at public aquatic venues to identify contributing factors, which can be addressed in future prevention efforts.



**Summary****What is already known about this topic?**

Equipment failure and human error at public aquatic venues can lead to toxic chlorine gas releases and have negative health impacts on bathers and aquatic staff members.

**What is added by this report?**

A multiagency investigation identified both equipment failure and human error as root causes of a toxic chlorine gas release and resulting exposures at a Contra Costa County, California municipal water park. A review of 2008–2015 California Department of Pesticide Regulation records identified contributing factors of toxic chlorine gas exposures at public aquatic venues, including equipment failure, human error, and restarting of a recirculation pump while bathers were present in pools.

**What are the implications for public health practice?**

Toxic chlorine gas releases at public aquatic venues can be prevented by regular testing of chemical control failsafe features, proper training of aquatic facility staff members, and by following standardized policies and procedures, including evacuating bathers from the pool before a recirculation pump is restarted. State or local jurisdictions can voluntarily use CDC's Model Aquatic Health Code (<https://www.cdc.gov/mahc/editions/current.html>) as a resource and guide of standardized, evidence-based regulations designed to prevent injuries and illness at public aquatic venues.

<sup>1</sup>Division of Environmental and Occupational Disease Control, California Department of Public Health, Richmond, California; <sup>2</sup>Career Epidemiology Field Officer Program, Field Services Branch, Division of State and Local Readiness, Office of Public Health Preparedness and Response, CDC; <sup>3</sup>Contra Costa Environmental Health, Martinez, California; <sup>4</sup>Hazardous Materials Division, Contra Costa Health Services, Martinez, California.

Corresponding author: Jason A. Wilken, [jason.wilken@cdph.ca.gov](mailto:jason.wilken@cdph.ca.gov), 510-620-3622.

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## Disparities in Diabetes Deaths Among Children and Adolescents — United States, 2000–2014

Sharon Saydah, PhD<sup>1</sup>; Giuseppina Imperatore, MD<sup>1</sup>; Yiling Cheng, PhD<sup>1</sup>; Linda S. Geiss, MS<sup>1</sup>; Ann Albright, PhD<sup>1</sup>

Diabetes is a common chronic disease of childhood affecting approximately 200,000 children and adolescents in the United States (1). Children and adolescents with diabetes are at increased risk for death from acute complications of diabetes, including hypoglycemia and diabetic ketoacidosis (2,3); in 2012, CDC reported that during 1968–2009, diabetes mortality among U.S. persons aged  $\leq 19$  years declined by 61% (4). CDC observed disparities by race during 1979–2004, with black children and adolescents dying from diabetes at twice the rate of white children and adolescents (5). However, no previous study has examined Hispanic ethnicity. CDC analyzed data from the National Vital Statistics System for deaths among persons aged 1–19 years in the United States during 2000–2014, with diabetes listed as the underlying cause of death overall, and for Hispanic, non-Hispanic white (white), and non-Hispanic black (black) children and adolescents. During 2012–2014, black children and adolescents had the highest diabetes death rate (2.04 per 1 million population), followed by whites (0.92) and Hispanics (0.61). There were no statistically significant changes in diabetes death rates over the study period, but disparities persisted among racial/ethnic groups. Death from diabetes in children and adolescents is potentially preventable through increased awareness of diabetes symptoms (including symptoms of low blood sugar), earlier treatment and education related to diabetes, and management of diabetes ketoacidosis. Continued measures are needed to reduce diabetes mortality in children and understand the cause of racial and ethnic disparities.

Diabetes mortality among persons aged 1–19 years during 2000–2014 was examined using information from death certificates filed in all 50 states and the District of Columbia (DC) and collected by CDC's National Center for Health Statistics. Hispanic ethnicity was collected on death certificates for all 50 states and DC starting in 1997. A diabetes death was defined as one with an *International Classification of Diseases, Tenth Revision* underlying cause of death code of E10–E14. Annual U.S. Census estimates for persons aged 1–19 years (<https://wonder.cdc.gov/wonder/help/cmfm.html#Population>) were used as the denominators. Mortality estimates were obtained from the CDC Wonder online database (<https://wonder.cdc.gov/mortSQL.html>). To produce stable mortality estimates, diabetes death rates were analyzed in 3-year intervals. Infants

(children aged  $< 1$  year) were excluded because methods for calculating neonatal and postnatal mortality rates differ from those for children aged  $\geq 1$  year. Race/ethnicity was categorized into the following groups: non-Hispanic black (black), non-Hispanic white (white), Hispanic, and all races/ethnicities (all children and adolescents). Hispanic includes all Hispanic origins, and persons who are Hispanic can be of any race. There were too few deaths among the other race/ethnicity groups to produce reliable estimates for those groups.

Joinpoint regression was used based on 3-year intervals to analyze trends using Hudson's algorithm, which includes time as a continuous variable (6). Joinpoint regression uses permutation tests to identify points where linear trends change significantly in direction or magnitude (i.e., joinpoints). The rate of change was tested for each trend to determine whether it was significantly different from zero, and each trend was described in the final model by an annual percentage change with a 95% confidence interval (CI). The National Cancer Institute's Joinpoint software was used (<https://surveillance.cancer.gov/joinpoint/>). Rate ratios and 95% CIs were calculated to compare racial/ethnic groups in each 3-year interval.

The total number of deaths from diabetes among all U.S. persons aged 1–19 years decreased from 265 (1.15 per 1 million) during 2000–2002 to 228 (0.97 per 1 million) during 2012–2014 (Table) (Figure). During 2012–2014, the highest diabetes death rates in this age group (2.04 per 1 million population) was among blacks, and the lowest was among Hispanics (0.61); death rates among whites were intermediate between blacks and Hispanics (0.92). From 2000–2002 to 2012–2014, the annual percentage change in diabetes death rate among all children and adolescents was -1.7%. From 2000–2002 to 2012–2014, the annual percentage change was 0.6% among Hispanics, -2.9% among blacks, and -0.92% among whites. None of these changes was significantly different from zero. There were no significant joinpoints, consistent with a straight line.

Although there were no statistically significant changes in diabetes death rates from 2000–2002 to 2012–2014, disparities persisted among racial/ethnic groups. During 2000–2002, the diabetes death rate ratio for blacks compared with whites was 2.36 and for blacks compared with Hispanics was 3.69 (Table). This disparity was still present during 2012–2014,

**TABLE. Deaths from diabetes per 1 million children and adolescents aged 1 to 19 years, by race and ethnicity and race/ethnicity rate ratios — United States, 2000–2014**

Characteristic	No. (95% CI)					Absolute change (95% CI)*	Annual percentage change (95% CI)
	2000–2002	2003–2005	2006–2008	2009–2011	2012–2014		
<b>Total no. deaths</b>	<b>265</b>	<b>285</b>	<b>251</b>	<b>231</b>	<b>228</b>	<b>-37</b>	<b>—</b>
<b>Deaths and rates of death</b>							
All racial/ethnic groups†	1.15 (1.01 to 1.29)	1.22 (1.08 to 1.36)	1.06 (0.93 to 1.19)	0.97 (0.85 to 1.1)	0.97 (0.84 to 1.1)	0.18 (-0.38 to 0.02)	1.67 (-3.39 to 0.09)
Hispanic	0.65 (0.42 to 0.95)	0.52 (0.33 to 0.78)	0.69 (0.48 to 0.96)	0.73 (0.52 to 0.99)	0.61 (0.42 to 0.85)	-0.04 (-0.04 to 0.28)	0.63 (-4.08 to 5.57)
Black	2.39 (1.91 to 2.95)	2.72 (2.21 to 3.32)	2.26 (1.80 to 2.80)	1.43 (1.07 to 1.88)	2.04 (1.60 to 2.57)	-0.35 (-1.04 to 0.35)	-2.89 (-9.17 to 3.83)
White	1.01 (0.85 to 1.18)	1.10 (0.93 to 1.27)	0.97 (0.81 to 1.14)	1.01 (0.84 to 1.18)	0.92 (0.75 to 1.08)	-0.10 (-0.33 to 0.14)	-0.92 (-2.82 to 1.02)
<b>Rate ratios (95% CI)§</b>							
Black to white	2.36 (1.80 to 3.08)§	2.47 (1.92 to 3.19)§	2.32 (1.76 to 3.05)§	1.42 (1.03 to 1.95)§	2.22 (1.66 to 2.98)§	—	—
Black to Hispanic	3.69 (2.38 to 5.73)§	5.26 (3.41 to 8.30)§	3.28 (2.20 to 4.89)§	1.97 (1.30 to 2.98)§	3.36 (2.24 to 5.04)§	—	—
White to Hispanic	1.57 (1.03 to 2.38)§	2.13 (1.37 to 3.30)§	1.41 (0.97 to 2.06)	1.39 (0.98 to 1.99)	1.51 (1.03 to 2.21)§	—	—

**Abbreviation:** CI = confidence interval.

\* From 2000–2002 to 2012–2014.

† Hispanic persons can be of any race; black and white refer to non-Hispanic persons.

§ Rate ratio is statistically significantly different from 1.0.

when the diabetes death rate for blacks was 2.22 times that of whites and 3.36 times that of Hispanics. Hispanics had the lowest diabetes death rates during all periods. Diabetes death rate for whites was 1.57 (95% CI = 1.03, 2.38) and 1.51 (95% CI = 1.03, 2.20) times that of Hispanics during 2000–2002 and 2012–2014, respectively.

### Discussion

During 2012–2014, among U.S. persons aged 1–19 years, 228 diabetes-related deaths (approximately one per 1 million population) occurred. It is encouraging that, despite increases in diabetes prevalence and incidence among children and adolescents during the 14 years from 2000 to 2014, there was no significant increase in diabetes mortality. However, significant racial/ethnic disparities in diabetes deaths among persons aged 1–19 years persisted. In particular, the death rates among blacks remained approximately twice as high as those of whites and Hispanics, whereas Hispanics had the lowest rates of diabetes mortality during all periods. Among children and adolescents, diabetes deaths are likely caused by acute complications of diabetes (3). Therefore, it would be expected that the highest diabetes-associated mortality would occur among racial/ethnic groups with the highest diabetes incidence and prevalence. The incidence of type 1 diabetes for children and adolescents was higher among whites than among blacks in 2011 (7), and the prevalence of childhood and adolescent diabetes among whites was higher than among blacks during this same period (8). In contrast, this analysis found that diabetes mortality was higher among black children and adolescents than among whites. Reasons for these disparities in deaths from diabetes are likely complex. Possible explanations could include differences in

### Summary

#### What is already known about this topic?

Diabetes in children and adolescents is a serious chronic disease. Young persons with diabetes are at risk for death from acute complications of the disease.

#### What is added by this report?

In this first report of diabetes mortality among Hispanic persons aged 1–19 years and comparison with mortality among white and black children and adolescents, there were no statistically significant changes in diabetes death rates from 2000–2002 to 2012–2014. Despite the higher prevalence and incidence of reported diabetes among whites than among blacks, blacks had approximately a twofold increased risk for diabetes death compared with whites and over a threefold increased risk compared with Hispanics.

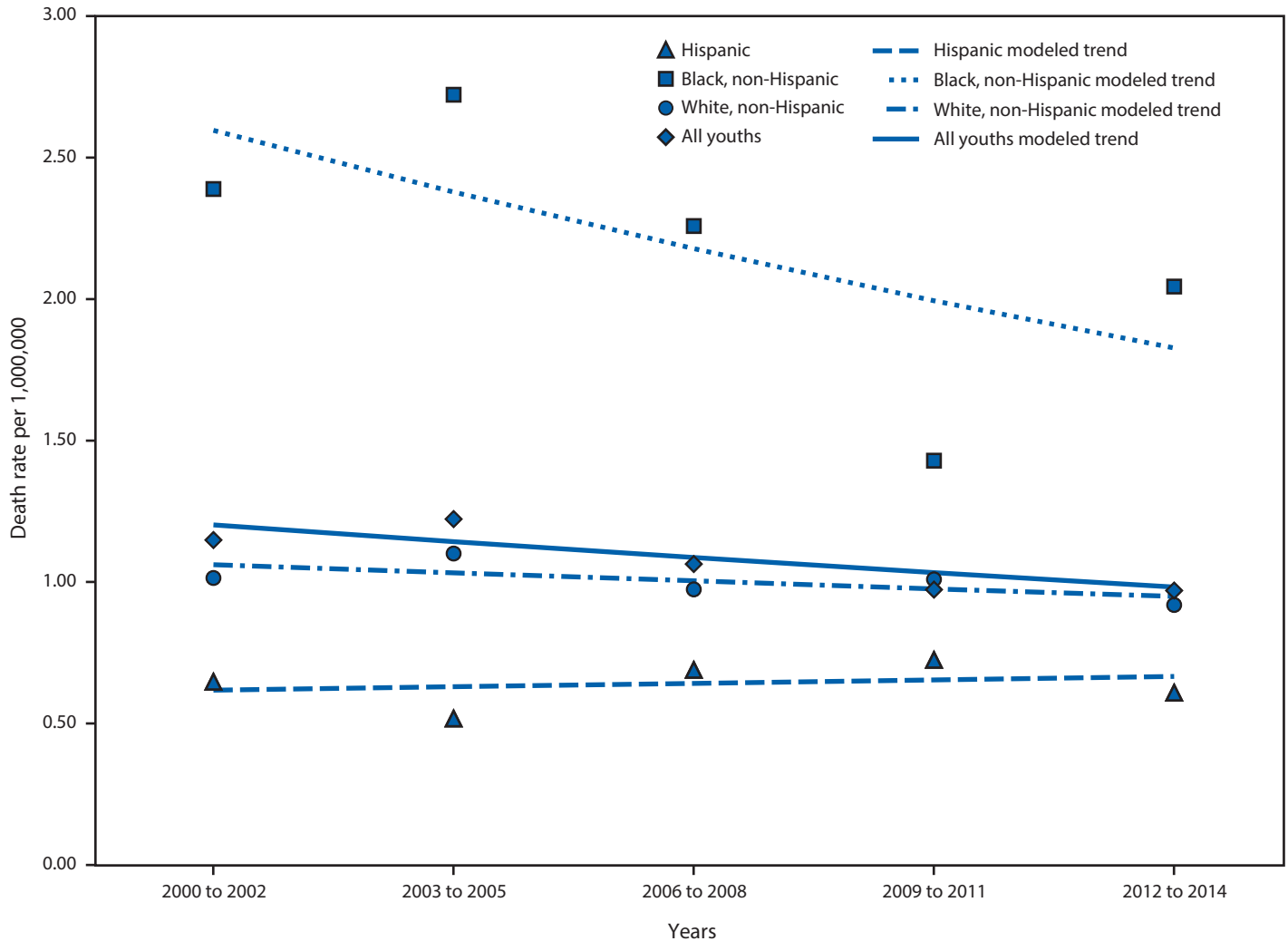
#### What are the implications for public health practice?

Deaths from diabetes in young persons are potentially preventable. The continued existence of racial/ethnic disparities in diabetes mortality in this age group adds information about Hispanics. Further research to identify health care factors and behaviors that contribute to diabetes mortality in children and adolescents might be helpful in understanding the reasons for disparities by race/ethnicity and focusing future prevention efforts.

access to health care, health services, diabetes self- and parent-management education, and diabetes care (2,9,10).

The findings in this report are subject to at least three limitations. First, because the small number of diabetes deaths precluded more detailed analysis, multiple years of deaths were combined for reliable estimates, which made it difficult to discern subtle changes in trends. Whereas a previous report observed an increase in diabetes mortality among persons aged

FIGURE. Three-year annual average diabetes death rates\* per 1 million among persons aged 1–19 years, by race/ethnicity — United States, 2000–2014



\* Symbols indicate observed points; lines indicate modeled trends. There were no significant modeled joinpoints, which is consistent with a straight line.

10–19 years (4), the small number of deaths in some racial/ethnic groups prevented stratification of the results by age. However, over the 2000–2014 period, only 14% of the deaths occurred among children aged 1–9 years (data not shown). The small number of deaths also precluded analysis by Hispanic subgroups. Second, it is also not known whether diabetes death rates differed by diabetes type. Type 1 diabetes is the most common diabetes type among children and adolescents, and its prevalence varies by race/ethnicity: among persons aged 10–19 years with diabetes, 94.5% of whites, 62.4% of blacks, and 64.8% of Hispanics have type 1 diabetes (8). However, in this study, information on the death certificate indicating diabetes type was only available for 24% of all diabetes deaths among persons aged 1–19 years from 2000 to 2014, precluding

analysis by diabetes type. Finally, there is a potential for misclassification of race/ethnicity on the death certificate.

This is the first time diabetes mortality among Hispanic children and adolescents has been reported and compared with mortality among whites and blacks. The findings indicate that although the diabetes mortality among children and adolescents has not changed significantly in the United States, disparities by race/ethnicity persist and warrant further research and investigation so that targeted interventions for prevention of diabetes deaths among children and adolescents can be developed and implemented.

<sup>1</sup>National Center for Chronic Disease Prevention and Health Promotion, Division of Diabetes Translation, CDC.

Corresponding author: Sharon Saydah, SSaydah@cdc.gov, 301-458-4183.

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# Hepatitis B Surface Antigen Screening Among Pregnant Women and Care of Infants of Hepatitis B Surface Antigen–Positive Mothers — Guam, 2014

Winston E. Abara, MD, PhD<sup>1,2</sup>; Susan Cha, PhD<sup>1,3</sup>; Tasneem Malik, MSN, MPH<sup>3</sup>; Mia S. DeSimone, MD<sup>4,5</sup>; Bernadette Schumann, MPA<sup>6</sup>; Esther Mallada<sup>6</sup>; Michael Klemme<sup>7</sup>; Vince Aguon, MPA<sup>6</sup>; Anne Marie Santos<sup>6</sup>; Melissa Collier, MD<sup>2</sup>; Mary Kamb, MD<sup>3</sup>

Hepatitis B virus (HBV) infection is endemic among adults in the U.S. territory of Guam (1,2). Perinatal HBV transmission, which occurs at birth from an infected mother to her newborn infant, is a major mode of HBV transmission and maintains HBV endemicity (3). Approximately 90% of HBV-infected infants will develop chronic HBV infection, and approximately 25% of those will die prematurely from liver failure or hepatocellular carcinoma (4,5). Since 1988, the Advisory Committee on Immunization Practices has recommended that all pregnant women be screened for hepatitis B surface antigen (HBsAg), an indicator of HBV infection, and that infants of women who screen positive (HBsAg-positive women) receive postexposure prophylaxis (PEP) (hepatitis B vaccine and hepatitis B immunoglobulin [HBIG]). When received within 12 hours of birth, PEP is 85%–95% effective in preventing perinatal HBV transmission (5,6). Hepatitis B vaccine provides long-term active immunity to HBV infection and HBIG provides short-term passive immunity to HBV infection until the infant responds to the vaccine (5). Hepatitis B vaccine was introduced into the routine universal infant vaccination schedule in Guam in 1988 (1).

Data for this analysis were obtained from the medical records of pregnant women who delivered live-born infants at Guam Memorial Hospital in 2014. This hospital is the largest delivery hospital in Guam and accounted for approximately 73% of all recorded births in 2014. Among 2,478 live-born infants delivered at this hospital during 2014, a sample of 971 (39%) was randomly selected. After excluding one infant from each of the five sets of twins in the selected sample, the final analytical sample consisted of 966 mother-infant pairs. Prenatal medical records of mothers of all 966 infants and vaccination records of infants of HBsAg-positive women were reviewed. Maternal demographic and clinical care data as well as information on the administration of hepatitis B vaccine and HBIG to infants of HBsAg-positive women were collected using a standardized chart abstraction tool. Descriptive analyses and frequencies were performed to calculate the prevalence of prenatal HBsAg screening, HBsAg positivity, demographic characteristics, prenatal care among pregnant women and the administration of hepatitis B vaccination and HBIG to infants of HBsAg-positive women. Receipt of prenatal care was defined as having  $\geq 1$  prenatal care visit before admission for delivery,

and prenatal HBsAg screening was defined as documentation of testing for HBsAg at any time before birth, including during the delivery admission.

Among the 966 women in this sample, 752 (78%) were Pacific Islanders, 197 (21%) were Asian, 11 (1%) were white, and two (<1%) were Hispanic (Table). The mean and median age at delivery was 27 years (range = 15–45 years); 542 (56.1%) women were aged >25 years at delivery. Information on prenatal HBsAg screening was available for 936 (97%) women, 905 (97%) of whom received prenatal HBsAg screening. Overall, 857 (89%) women received prenatal care; among this group, prenatal HBsAg screening information was available for 834 (97%) women, 818 (98%) of whom were screened for HBsAg. Among the 106 (11%) women who did not receive prenatal care, prenatal HBsAg screening data were available for 102 (96%); among these women, 87 (85%) were screened for HBsAg upon admission for delivery. The odds of receiving HBsAg screening among women who received prenatal care was significantly higher than among those who did not receive prenatal care (odds ratio = 8.82,  $p < 0.001$ ).

Among 899 women with available HBsAg screening result data, 18 (2%) were HBsAg-positive, of whom 14 were Pacific Islanders and four were Asian. Sixteen (89%) HBsAg-positive women were aged >25 years of age at delivery (born before the introduction of hepatitis B vaccine into the routine immunization program in 1988), and were therefore less likely to have been vaccinated against hepatitis B as infants; hepatitis B vaccination status of mothers was not available. All 18 infants born to HBsAg-positive women received hepatitis B vaccination within 12 hours of delivery and 17 of 18 received HBIG.

## Discussion

The prevalence of prenatal HBsAg screening in this hospital-based random sample of women with a live birth during 2014 in Guam (97%) was similar to the 94% prevalence estimate in the continental United States in 2010 (7); however, the 2.0% HBsAg positivity prevalence in this sample is approximately 13 times higher than the 0.14% maternal prevalence estimate among U.S.-born Pacific Islander and Asian women and approximately twice the 0.9% maternal prevalence estimate in the continental United States (7,8). Despite the high HBsAg prevalence in this sample, all infants born to HBsAg-positive

**TABLE. Demographic characteristics, prenatal hepatitis B surface antigen (HBsAg) screening, prenatal care received, and screening results among a random sample of pregnant women with live-born deliveries, and receipt of hepatitis B virus postexposure prophylaxis among infants of HBsAg-positive mothers — Guam Memorial Hospital, Guam, 2014 (N = 966)**

Characteristic	No. (%) <sup>*</sup>
<b>Race/Ethnicity (N = 962)</b>	
Pacific Islander	752 (78.2)
Asian	197 (20.5)
White	11 (1.1)
Hispanic	2 (0.2)
<b>Prenatal HBsAg screening received<sup>†</sup> (N = 936)</b>	
Yes	905 (96.7)
No	31 (3.3)
<b>Prenatal care received<sup>§</sup> (N = 963)</b>	
Yes	857 (89.0)
No	106 (11.0)
<b>Prenatal HBsAg screening among women with prenatal care (N = 834)</b>	
Yes	818 (98.1)
No	16 (1.9)
<b>Prenatal HBsAg screening among women without prenatal care (N = 102)</b>	
Yes	87 (85.3)
No	15 (14.7)
<b>Maternal HBsAg screening results (N = 899)</b>	
HBsAg-positive	18 (2.0)
HBsAg-negative	881 (98.0)
<b>Receipt of postexposure HBV prophylaxis among infants born to HBsAg-positive women<sup>¶</sup> (N = 18)</b>	
Received HB vaccine within 12 hrs of delivery	18 (100)
Received HBIG within 12 hrs of delivery	17 (94)
<b>Age at delivery, yrs (N = 966)</b>	
Mean	27.2
Median	27.0
Range (SD)	15–45 (6.2)
>25 yrs (all mothers [N = 966])	542 (56.1)
>25 yrs (HBsAg-positive mothers [N = 18])	16 (88.9)

**Abbreviations:** HBIG = hepatitis B immune globulin; HBV = hepatitis B virus; SD = standard deviation.

<sup>\*</sup> Except as noted.

<sup>†</sup> Includes women screened during prenatal care and women without prenatal care who were screened upon admission for delivery.

<sup>§</sup> At least one prenatal care visit before delivery.

<sup>¶</sup> Limited to infants born to HBsAg-positive mothers.

women received hepatitis B vaccine, and all but one infant received HBIG. Most of the HBsAg-positive women included in the sample were born before hepatitis B vaccine was introduced into Guam's universal infant vaccination schedule; although catch-up vaccination programs were implemented in later years, many women might have been missed. However, infant and childhood hepatitis B vaccination coverage has significantly increased in Guam and the other U.S.-affiliated Pacific Islands since the introduction of universal infant hepatitis B vaccination (9). The estimated hepatitis B vaccine coverage among children in this region is 98%; consequently, the risk for perinatal HBV transmission is likely to decrease in future birth cohorts (9).

## Summary

### What is already known about this topic?

Hepatitis B virus (HBV) infection is endemic in the U.S. territory of Guam, and perinatal transmission is a major mode of transmission. The Advisory Committee on Immunization Practices recommends that all pregnant women be screened for hepatitis B surface antigen (HBsAg) in each pregnancy and that infants of HBsAg-positive women receive postexposure prophylaxis (PEP) with hepatitis B vaccine and hepatitis B immune globulin (HBIG) within 12 hours of birth to reduce the risk for perinatal HBV transmission.

### What is added by this report?

In a hospital-based random sample of women with a live birth during 2014 in Guam, HBsAg seroprevalence (2.0%) was approximately 13 times higher than that among U.S.-born Pacific Islander and Asian women (0.14%) and approximately twice the overall U.S. maternal prevalence estimate (0.9%). Approximately 90% of HBsAg-positive women were born before introduction of universal infant hepatitis B vaccination. Among women who had at least one prenatal care visit, 98% received prenatal HBsAg screening, compared with 85% of women who did not receive prenatal care. All infants of HBsAg-positive women received hepatitis B vaccine and all but one infant received HBIG.

### What are the implications for public health practice?

Prenatal HBsAg screening facilitates prompt identification of HBsAg-positive pregnant women and mitigates the risk for perinatal HBV transmission. Timely administration of PEP to infants of HBsAg-positive women is important to prevent perinatal HBV transmission.

The findings in this report are subject to at least two limitations. First, although this hospital accounted for approximately three quarters of births registered in 2014 in Guam, the data came from only one hospital; therefore, the prevalence of prenatal HBsAg screening, maternal HBsAg positivity, hepatitis B vaccination, and HBIG administration cannot be generalized to all health care facilities in Guam. Second, no postvaccination serologic testing data for the 18 infants born to HBsAg-positive mothers were available to assess HBV infection and immune response status, although administration of hepatitis B vaccine and HBIG within 12 hours of birth is reported to be 85%–95% effective in preventing perinatal transmission (5).

The prevalence of HBsAg screening among women who had at least one prenatal care visit (98%) was significantly higher than that among women who did not (85%). Prenatal screening of all pregnant women for HBsAg is a critical component of the HBV elimination strategy in the United States and its territories (6), especially in areas with a high prevalence of HBV infection in adults. Prenatal screening facilitates the timely identification of HBsAg-positive women and ensures that PEP is available to their infants immediately after delivery,

thus reducing the likelihood of infants becoming chronically infected and serving as reservoirs for continued HBV transmission (5,10). Prenatal care is important for prenatal HBSAg screening in Guam. Fully implementing systemic and institutional hospital policies that require documentation of maternal HBsAg status in hospital maternity records and the administration of PEP to all infants of HBsAg-positive mothers will ensure that all infants at risk receive PEP and that the risk for perinatal HBV transmission is reduced (7).

<sup>1</sup>Epidemic Intelligence Service, CDC; <sup>2</sup>Division of Viral Hepatitis, CDC; <sup>3</sup>Division of Sexually Transmitted Diseases Prevention, CDC; <sup>4</sup>Division of Scientific Education and Professional Development, CDC; <sup>5</sup>Emory University School of Medicine, Atlanta, Georgia; <sup>6</sup>Guam Department of Public Health and Social Services; <sup>7</sup>Guam Memorial Hospital Authority.

Corresponding author: Winston Abara, wabara@cdc.gov, 404-639-8132.

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# Updated Recommendations for Use of MenB-FHbp Serogroup B Meningococcal Vaccine — Advisory Committee on Immunization Practices, 2016

Monica E. Patton, MD<sup>1</sup>; David Stephens, MD<sup>2</sup>; Kelly Moore, MD<sup>3</sup>; Jessica R. MacNeil, MPH<sup>1</sup>

Two serogroup B meningococcal (MenB) vaccines are currently licensed for use in persons aged 10–25 years in the United States. The two vaccines are MenB-FHbp (Trumenba, Pfizer, Inc.) (1) and MenB-4C (Bexsero, GlaxoSmithKline Biologicals, Inc.) (2). In February 2015, the Advisory Committee on Immunization Practices (ACIP) recommended use of MenB vaccines among certain groups of persons aged ≥10 years who are at increased risk for serogroup B meningococcal disease\* (Category A) (3), and in June 2015, ACIP recommended that adolescents and young adults aged 16–23 years may be vaccinated with MenB vaccines to provide short-term protection against most strains of serogroup B meningococcal disease (Category B<sup>†</sup>) (4). Consistent with the original Food and Drug Administration (FDA) licensure for the two available MenB vaccines, ACIP recommended either a 3-dose series of MenB-FHbp or a 2-dose series of MenB-4C. Either MenB vaccine can be used when indicated; ACIP does not state a product preference. The two MenB vaccines are not interchangeable; the same vaccine product must be used for all doses in a series. In April 2016, changes to the dosage and administration of MenB-FHbp were approved by FDA to allow for both a 2-dose series (administered at 0 and 6 months) and a 3-dose series (administered at 0, 1–2, and 6 months) (5,6). In addition, the package insert now states that the choice of dosing schedule depends on the patient's risk for exposure and susceptibility to serogroup B meningococcal disease. These recommendations are regarding use of the 2- and 3-dose schedules of MenB-FHbp vaccine (Trumenba) and replace previous ACIP recommendations for use of MenB-FHbp vaccine published in 2015 (3,4). Recommendations regarding use of MenB-4C (Bexsero) are unchanged (3,4).

\*Persons with persistent complement component deficiencies (including inherited or chronic deficiencies in C3, C5–C9, properdin, factor D, factor H, or who are taking eculizumab [Solaris]); persons with anatomic or functional asplenia (including sickle cell disease); microbiologists routinely exposed to isolates of *Neisseria meningitidis*; persons identified as at increased risk because of a serogroup B meningococcal disease outbreak.

<sup>†</sup>Category A recommendations are made for all persons in an age- or risk-factor-based group. Category B recommendations are made for individual clinical decision making. <https://www.cdc.gov/vaccines/acip/recs/grade/about-grade.html>.

## Methods

The ACIP Meningococcal Vaccines Work Group identified studies of the comparative immunogenicity, safety, and antibody persistence of 2- and 3-dose schedules of MenB-FHbp vaccine by consulting with the manufacturer and searching PubMed using the search terms “meningococcal serogroup B vaccine,” “Trumenba,” and “MenB-FHbp.” One relevant published clinical trial (7) and unpublished data from the same trial (Pfizer, unpublished data<sup>§</sup>) were identified that compared immunogenicity and safety of 2- and 3-dose schedules of MenB-FHbp vaccine. Additionally, unpublished data were identified (Pfizer, unpublished data<sup>¶</sup>) for participants in the same trial who were enrolled in an extension study designed to evaluate antibody persistence annually for 48 months and response to a single booster dose approximately 48 months after the primary series. The Work Group reviewed published and unpublished immunogenicity and safety data from the clinical trial and unpublished antibody persistence data and booster dose response data. The type and quality of evidence supporting the use of MenB vaccines in adolescents and young adults (including college students) and persons at increased risk for serogroup B meningococcal disease were evaluated previously using the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) framework (3,4,8,9). Summaries of the Work Group discussions and data reviewed were presented to ACIP in June and October 2016, and recommendations were approved by the voting ACIP members at the October 2016 meeting (detailed meeting minutes are available at <https://www.cdc.gov/vaccines/acip/meetings/meetings-info.html>).

## MenB-FHbp Immunogenicity

Previous ACIP policy statements have described the assessments of MenB-FHbp immunogenicity data for persons aged ≥10 years that supported FDA licensure (3,4,8,9). The immunogenicity of 3-dose versus 2-dose MenB-FHbp schedules in

<sup>§</sup> Unpublished data, study B1971012, <https://clinicaltrials.gov/ct2/show/NCT01299480>, ACIP meeting October 2016.

<sup>¶</sup> Unpublished data, study B1971033, <https://clinicaltrials.gov/ct2/show/NCT01543087>, ACIP meeting October 2016.

adolescents and young adults was evaluated in a clinical trial conducted in Europe among 1,450 persons aged 11–18 years (7) (Pfizer, unpublished data). Participants were randomly assigned to one of five groups. Group 1 received MenB-FHbp at months 0, 1, and 6 and received a saline injection at month 2; group 2 received MenB-FHbp at months 0, 2, and 6 and saline at month 1; group 3 received MenB-FHbp at months 0 and 6 and saline at months 1 and 2; group 4 received MenB-FHbp at months 0 and 2 and saline at months 1 and 6; group 5 received MenB-FHbp at months 2 and 6 and saline at months 0 and 1 (referred to as 0, 4 months below). Serum bactericidal antibody activity, measured using human complement (hSBA) was used as a correlate of protection to assess vaccine immunogenicity (10,11). Immunogenicity in the trial was assessed as the percentage of subjects who achieved an hSBA titer greater than or equal to the lower limit of quantification of the assay (hSBA titer  $\geq 1:8$ ) to each of the four selected serogroup B meningococcal strains tested (7,12). For purposes of this evaluation, immunogenicity was assessed as the proportion of subjects who achieved an hSBA titer  $\geq 1:8^{**}$  to all four selected strains tested (composite response) (Pfizer, unpublished data).

Among the 3-dose schedules evaluated, 83.1% of subjects in group 1 (0, 1, 6 months) and 81.7% of subjects in group 2 (0, 2, 6 months) had a composite response (hSBA titer  $\geq 1:8$ ) to all four strains tested at 1 month following the third dose (Table) (Pfizer, unpublished data). Among the 2-dose schedules, group 3 (0, 6 months) had the highest percent of responders, 73.5%; 58.9% of subjects in group 5 (0, 4 months) and 56.8% of subjects in group 4 (0, 2 months) had a composite response to all four strains tested at 1 month following the second dose. In addition, whereas geometric mean antibody titers (GMTs) were higher to all four strains tested among subjects who received 3 doses compared with those who received 2 doses, group 3 (0, 6 months) had the highest GMTs among all 2-dose schedules (7).

### MenB-FHbp Antibody Persistence

Antibody persistence data through 48 months and response to a single booster dose at approximately 48 months were evaluated for participants aged 11–18 years in the clinical trial described who also enrolled in an extension study (Pfizer, unpublished data). The percentage of subjects with protective titers to all four of the serogroup B meningococcal strains tested was evaluated at 1, 12, 18, 24, 36, and 48 months following completion of the aforementioned 2-dose and 3-dose schedules and at 1 month following the booster dose at 48 months. An hSBA titer  $\geq 1:4$  was considered protective, a

\*\* Lower limit of quantification for the MenB strain expressing FHbp A22 was hSBA titer  $\geq 1:16$ .

**TABLE. Percentage of persons\* aged 11–18 years who achieved an hSBA titer  $\geq 1:8^{\dagger}$  against all four selected serogroup B meningococcal strains tested<sup>§</sup> (composite response) at 1 month (m) following completion of a 3-dose (0, 1, 6 months; and 0, 2, 6 months) or 2-dose (0, 6 months; 0, 2 months; and 0, 4 months) series of MenB-FHbp**

Series	Group	hSBA titer $\geq 1:8^{\dagger}$ against all four serogroup B strains <sup>§</sup> (%)	95% confidence interval
3-dose series	Group 1 (0, 1, 6 m)	83.1	78.6–86.9
	Group 2 (0, 2, 6 m)	81.7	77.3–85.7
2-dose series	Group 3 (0, 6 m)	73.5	68.5–78.1
	Group 4 (0, 2 m)	56.8	52.5–61.0
	Group 5 (0, 4 m)	58.9	49.0–68.3

**Abbreviation:** hSBA = serum bactericidal antibody activity, measured using human complement.

\* Number of subjects in the evaluable immunogenicity population = 1,450.

<sup>†</sup> hSBA titer  $\geq 1:16$  for the serogroup B strain expressing FHbp (factor H binding protein) A22.

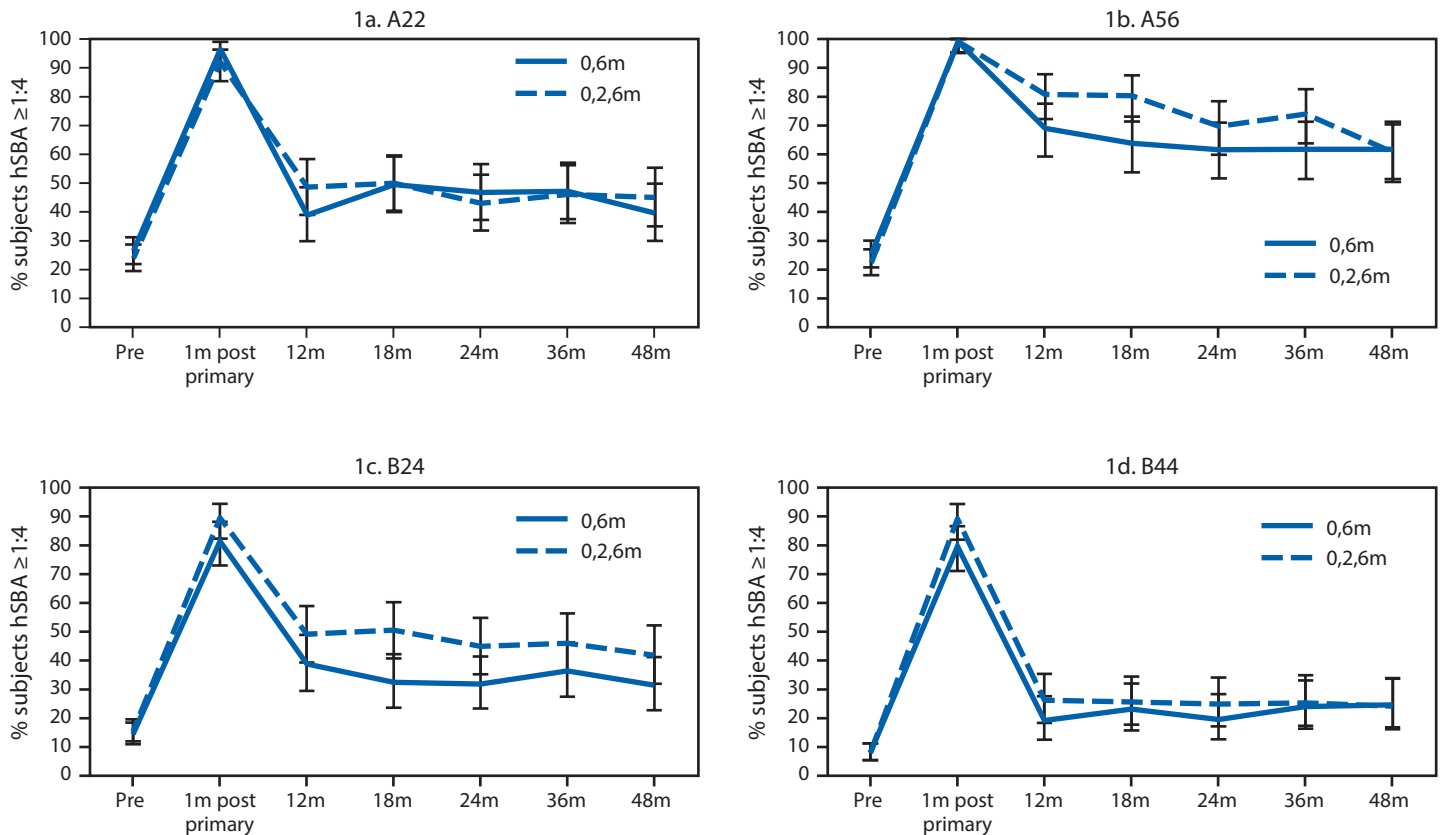
<sup>§</sup> Serogroup B meningococcal strains expressing FHbp of subfamily A (A22, A56) and subfamily B (B24, B44).

lower level of activity than the hSBA titer of  $\geq 1:8$  used to assess immunogenicity. Among subjects enrolled in the extension study who received the 2-dose (0, 6 month) schedule and the 3-dose (0, 2, 6 month) schedule, the percentages of subjects with protective hSBA titers to the four selected strains did not statistically differ at any time point (Figure 1). At 1 month following completion of the primary series, 78.9%–98.9% of subjects who received the 2-dose schedule and 86.5%–99.1% of subjects who received the 3-dose schedule had protective hSBA titers to the four selected strains. For both groups, the percentage of subjects with protective antibodies declined sharply at 12 months after completion of the primary series and remained stable through 48 months after vaccination (Figure 1). The hSBA responses and GMTs following a single booster dose at approximately 48 months after primary vaccination for the group that received the 2-dose schedule were not statistically different from those of the group that received the 3-dose schedule (Figure 2).

### MenB-FHbp Safety

MenB-FHbp safety data have been reported previously (3,4,8,9). No significant increased risk for serious adverse events has been identified among >4,250 subjects aged 10–25 years in seven clinical trials who received at least 1 dose of MenB-FHbp (4,6,8,9). The most common adverse reactions observed in the 7 days after receipt of MenB-FHbp were pain at the injection site ( $\geq 85\%$  of subjects), fatigue ( $\geq 40\%$ ), headache ( $\geq 35\%$ ), myalgia ( $\geq 30\%$ ), and chills ( $\geq 15\%$ ). Safety and tolerability profiles were similar among subjects aged 11–18 years who were randomly assigned either a 3-dose or 2-dose series of MenB-FHbp (6,7).

**FIGURE 1.** Persistence of hSBA responses  $\geq 1:4$ \* against four selected serogroup B meningococcal strains† in subjects aged 11–18 years,‡ up to 48 months (m) after completion of a 2-dose (0, 6 months) or 3-dose (0, 2, 6 months) series of MenB-FHbp



**Abbreviation:** hSBA = serum bactericidal antibody activity, measured using human complement.

\* Expressed as a percentage, with error bars representing 95% confidence intervals.

† Serogroup B meningococcal strains expressing FHbp (factor H binding protein) of subfamily A (A22, A56) or subfamily B (B24, B44).

‡ Number of subjects for persistence time points: 0, 6 m = 99–116; 0, 2, 6 m = 92–114.

## ACIP Recommendations

These recommendations are regarding use of the 2- and 3-dose schedules of MenB-FHbp vaccine (Trumenba) and replace previous ACIP recommendations for use of MenB-FHbp vaccine published in 2015 (3,4). Recommendations regarding use of MenB-4C (Bexsero) are unchanged (3,4).

**Persons aged  $\geq 10$  years at increased risk for serogroup B meningococcal disease (Category A recommendation).** For persons at increased risk for meningococcal disease and for use during serogroup B meningococcal disease outbreaks, 3 doses of MenB-FHbp should be administered at 0, 1–2, and 6 months to provide earlier protection and maximize short-term immunogenicity. However, if the second dose of MenB-FHbp is administered at an interval of  $\geq 6$  months, a third dose does not need to be administered.

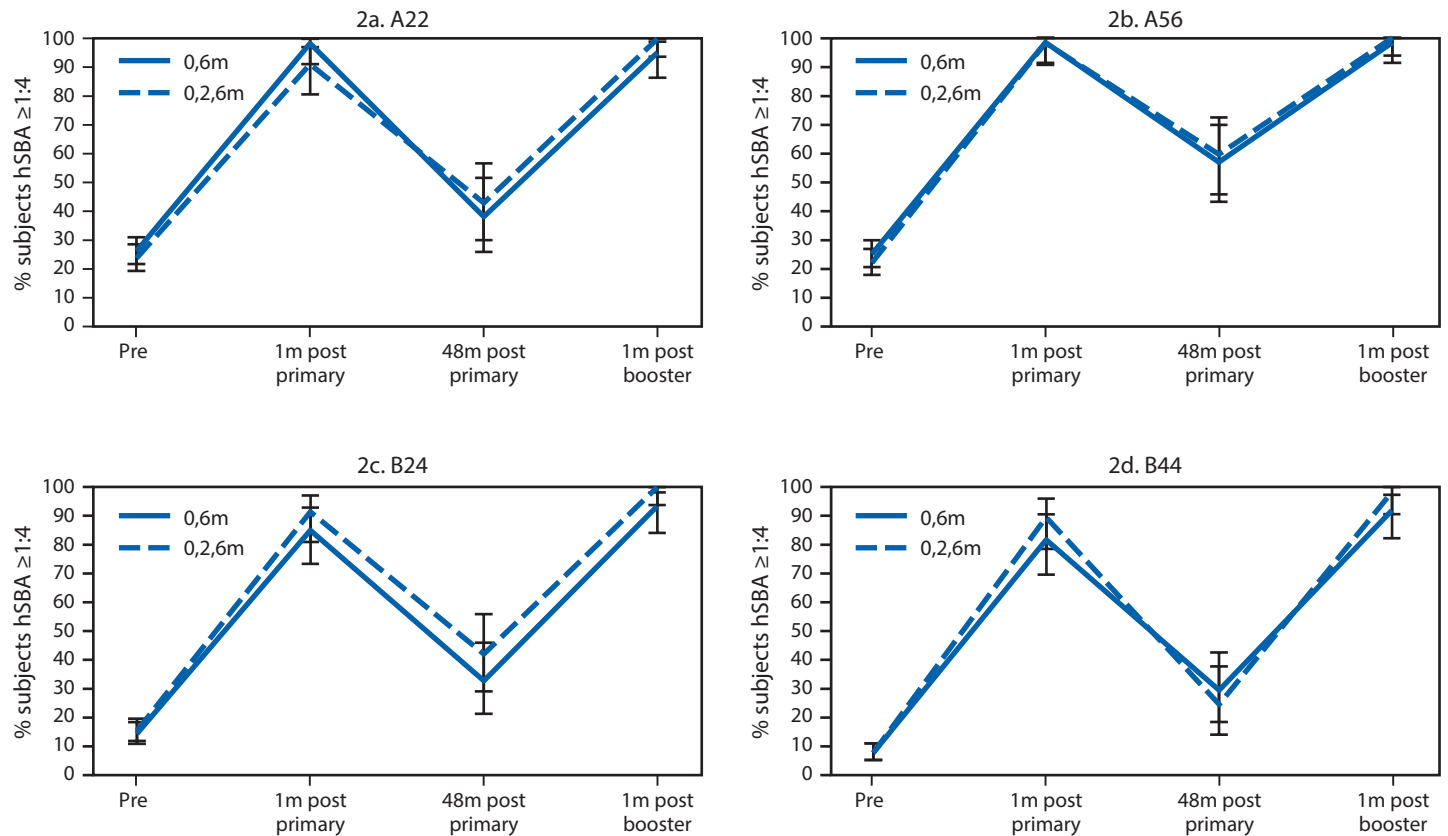
**Adolescents and young adults aged 16–23 years (Category B recommendation).** When given to healthy adolescents who are not at increased risk for meningococcal disease, 2 doses of

MenB-FHbp should be administered at 0 and 6 months. If the second dose of MenB-FHbp is administered earlier than 6 months after the first dose, a third dose should be administered at least 4 months after the second dose.

## CDC Guidance for Use

There are two MenB vaccines licensed for use in the United States among persons aged 10–25 years. Either MenB vaccine can be used when indicated; ACIP does not state a product preference. The two MenB vaccines are not interchangeable; the same vaccine product must be used for all doses in a series. The minimum interval between any 2 doses of MenB vaccine is 4 weeks. On the basis of available data and expert opinion, MenB-FHbp or MenB-4C may be administered concomitantly with other vaccines indicated for this age, but at a different anatomic site, if feasible. ACIP will consider MenB vaccine booster doses for persons at increased risk for serogroup B meningococcal disease as data become available.

**FIGURE 2.** Persistence of hSBA responses  $\geq 1:4^*$  and GMTs<sup>†</sup> against four selected serogroup B meningococcal strains<sup>§</sup> at 48 months (m) in subjects aged 11–18 years<sup>¶</sup> after completion of a 2-dose (0, 6 months) or 3-dose (0, 2, 6 months) series of MenB-FHbp, and hSBA responses  $\geq 1:4$  and GMTs to a booster dose of MenB-FHbp at approximately 48 months after primary vaccination



**Abbreviations:** GMTs = geometric mean antibody titers; hSBA = serum bactericidal antibody activity, measured using human complement.

\* Expressed as a percentage, with error bars representing 95% confidence intervals.

† GMTs were as follows.

**A22 (0, 6 m):** Pre 6.4, 1m post primary 55.8, 48m post primary 15.3, 1m post booster 140.0; (0, 2, 6m): Pre 6.3, 1m post primary 59.5, 48m post primary 15.4, 1m post booster 119.1.

**A56 (0, 6m):** Pre 6.7, 1m post primary 143.1, 48m post primary 15.8, 1m post booster 358.0; (0, 2, 6m): Pre 6.1, 1m post primary 191.2, 48m post primary 17.4, 1m post booster 370.8.

**B24 (0, 6m):** Pre 5.0, 1m post primary 29.2, 48m post primary 7.8, 1m post booster 86.0; (0, 2, 6m): Pre 5.1, 1m post primary 30.5, 48m post primary 9.1, 1m post booster 80.3.

**B44 (0, 6m):** Pre 4.5, 1m post primary 35.5, 48m post primary 5.3, 1m post booster 84.6; (0, 2, 6m): Pre 4.5, 1m post primary 50.2, 48m post primary 5.3, 1m post booster 117.6.

<sup>§</sup> Serogroup B meningococcal strains expressing FHbp (factor H binding protein) of subfamily A (A22, A56) or subfamily B (B24, B44).

<sup>¶</sup> Number of subjects for persistence time points: 0, 6 m = 58–62; 0, 2, 6 m = 57–58.

No randomized controlled clinical trials have been conducted to evaluate the use of MenB vaccines in pregnant or lactating women. As stated in previous ACIP reports on MenB vaccines, vaccination should be deferred in women known to be pregnant or lactating unless the woman is at increased risk for serogroup B meningococcal disease, and, after consultation with her health care provider, the benefits of vaccination are considered to outweigh the potential risks. Additional information for health care providers and parents can be found at <https://www.cdc.gov/meningococcal>.

## Precautions and Contraindications

Before administering serogroup B meningococcal vaccines, health care providers should consult the package inserts for precautions, warnings, and contraindications (6,13). Adverse events occurring after administration of any vaccine should be reported to the Vaccine Adverse Event Reporting System (VAERS). Reports can be submitted to VAERS online, by fax, or by mail. Additional information about VAERS is available by telephone (1–800–822–7967) or online (<https://vaers.hhs.gov>).

## References

## Summary

## What is currently recommended?

Two serogroup B meningococcal (MenB) vaccines are currently licensed for use among persons aged 10–25 years in the United States: MenB-FHbp (Trumenba) and MenB-4C (Bexsero). The Advisory Committee on Immunization Practices (ACIP) currently recommends routine use of MenB vaccines among persons aged ≥10 years who are at increased risk for serogroup B meningococcal disease (Category A recommendation), including persons who have persistent complement component deficiencies; persons who have anatomic or functional asplenia; microbiologists who routinely are exposed to isolates of *Neisseria meningitidis*; and persons identified to be at increased risk because of a serogroup B meningococcal disease outbreak. Adolescents and young adults aged 16–23 years may also be vaccinated with MenB vaccines to provide short-term protection against most strains of serogroup B meningococcal disease (Category B recommendation). Consistent with the original Food and Drug Administration (FDA) licensure for the MenB vaccines, ACIP recommended either a 3-dose series of MenB-FHbp or a 2-dose series of MenB-4C. Either MenB vaccine can be used when indicated; however, they are not interchangeable, and the same product must be used for all doses.

## Why are the recommendations being modified now?

Changes to the dosage and administration of MenB-FHbp were approved by FDA to include both a 3-dose series (administered at 0, 1–2, and 6 months) and a 2-dose series (administered at 0 and 6 months).

## What are the new recommendations?

These updated recommendations are regarding use of the 2- and 3-dose schedules of MenB-FHbp vaccine (Trumenba). For persons at increased risk for meningococcal disease and for use during serogroup B meningococcal disease outbreaks, ACIP recommends that 3 doses of MenB-FHbp be administered at 0, 1–2, and 6 months. When given to healthy adolescents who are not at increased risk for meningococcal disease, ACIP recommends that 2 doses of MenB-FHbp should be administered at 0 and 6 months. Recommendations regarding use of MenB-4C vaccine (Bexsero) are unchanged. Either MenB vaccine can be used when indicated; however, they are not interchangeable, and the same product must be used for all doses in a series.

## Acknowledgments

ACIP members (membership roster for June 2016–July 2017, available at <https://www.cdc.gov/vaccines/acip/committee/members.html>); ACIP Meningococcal Vaccines Work Group.

<sup>1</sup>Division of Bacterial Diseases, National Center for Immunization and Respiratory Diseases, CDC; <sup>2</sup>Advisory Committee on Immunization Practices Meningococcal Vaccines Work Group, Emory University School of Medicine, Atlanta, Georgia; <sup>3</sup>Advisory Committee on Immunization Practices Meningococcal Vaccines Work Group, Tennessee Department of Health and Vanderbilt University School of Medicine, Nashville, Tennessee.

Corresponding author: Jessica R. MacNeil, [jmacneil@cdc.gov](mailto:jmacneil@cdc.gov).

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## Notes from the Field

### Ongoing Transmission of *Candida auris* in Health Care Facilities — United States, June 2016–May 2017

Sharon Tsay, MD<sup>1,2</sup>; Rory M. Welsh, PhD<sup>1</sup>; Eleanor H. Adams, MD<sup>3</sup>; Nancy A. Chow, PhD<sup>1</sup>; Lalitha Gade, MPharm<sup>1</sup>; Elizabeth L. Berkow, PhD<sup>1</sup>; Eugénie Poirot, PhD<sup>2,4</sup>; Emily Lutterloh, MD<sup>3,5</sup>; Monica Quinn, MS<sup>3</sup>; Sudha Chaturvedi, PhD<sup>3,5</sup>; Janna Kerins, VMD<sup>2,6</sup>; Stephanie R. Black, MD<sup>6</sup>; Sarah K. Kemble, MD<sup>6</sup>; Patricia M. Barrett, MSD<sup>7</sup>; Kerri Barton, MPH<sup>8</sup>; D.J. Shannon, MPH<sup>9</sup>; Kristy Bradley, DVM<sup>10</sup>; Shawn R. Lockhart, PhD<sup>1</sup>; Anastasia P. Litvintseva, PhD<sup>1</sup>; Heather Moulton-Meissner, PhD<sup>11</sup>; Alicia Shugart, MA<sup>11</sup>; Alex Kallen, MD<sup>11</sup>; Snigdha Vallabhaneni, MD<sup>1</sup>; Tom M. Chiller, MD<sup>1</sup>; Brendan R. Jackson, MD<sup>1</sup>

In June 2016, CDC released a clinical alert about the emerging, and often multidrug-resistant, fungus *Candida auris* and later reported the first seven U.S. cases of infection through August 2016 (1). Six of these cases occurred before the clinical alert and were retrospectively identified. As of May 12, 2017, a total of 77 U.S. clinical cases of *C. auris* had been reported to CDC from seven states: New York (53 cases), New Jersey (16), Illinois (four), Indiana (one), Maryland (one), Massachusetts (one), and Oklahoma (one) (Figure). All of these cases were identified through cultures taken as part of routine patient care (clinical cases). Screening of close contacts of these patients, primarily of patients on the same ward in health care facilities, identified an additional 45 patients with *C. auris* isolated from one or more body sites (screening cases), resulting in a total of 122 patients from whom *C. auris* has been isolated.

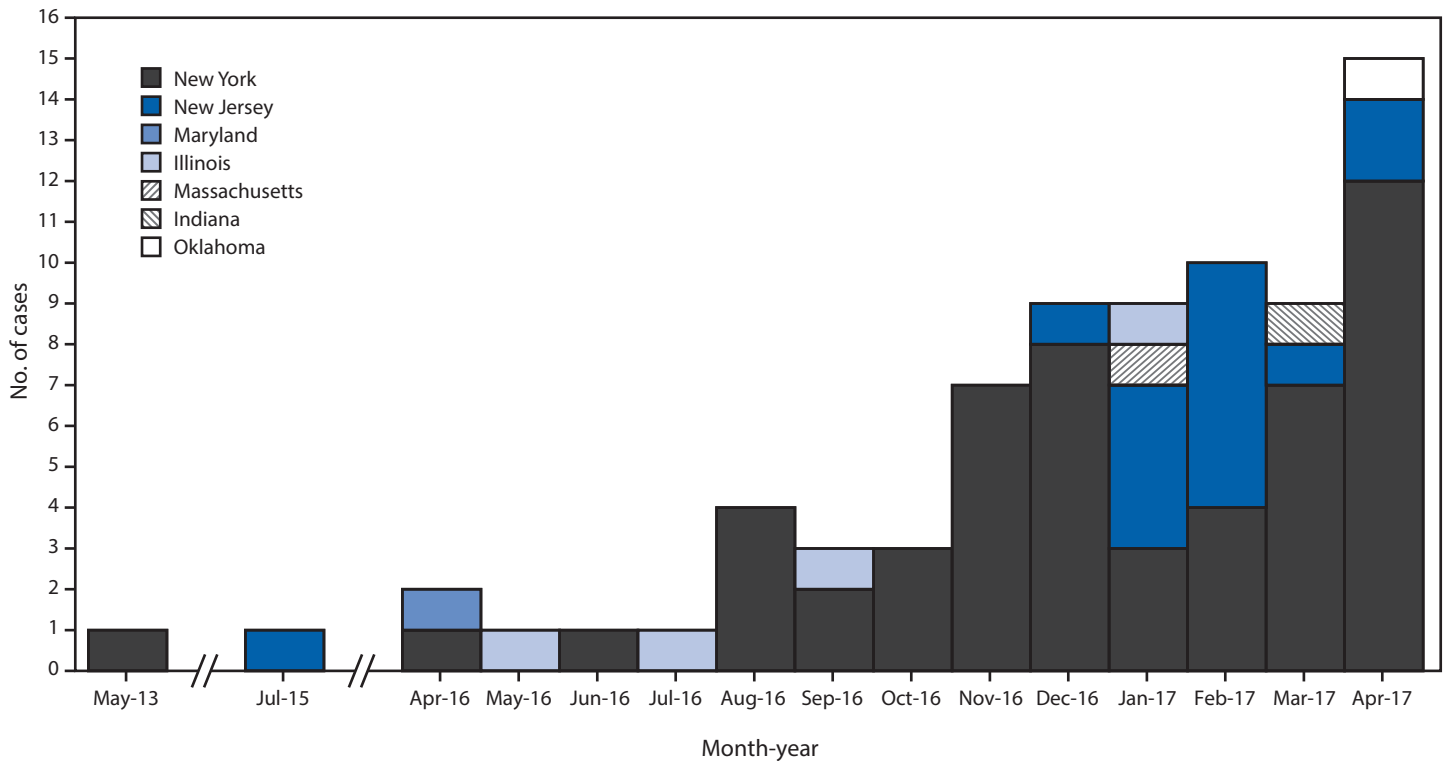
Among the 77 clinical cases, median patient age was 70 years (range = 21–96 years), and 55% were male. *C. auris* was cultured from the following sites: blood (45 isolates), urine (11), respiratory tract (eight), bile fluid (four), wound (four), central venous catheter tip (two), bone (one), ear (one), and a jejunal biopsy (one). Antifungal susceptibility testing at CDC of the first 35 clinical isolates revealed that 30 (86%) isolates were resistant to fluconazole (minimum inhibitory concentration [MIC] >32), 15 (43%) were resistant to amphotericin B (MIC ≥2), and one (3%) was resistant to echinocandins (MIC >4). Most (69, 90%) clinical cases were identified in the New York City metropolitan area (53 in New York and 16 in New Jersey). Nearly all patients had multiple underlying medical conditions and extensive health care facility exposure. Epidemiologic links have been found between most cases. In Illinois, three cases were associated with the same long-term care facility. In New York and New Jersey, cases were identified in multiple acute care hospitals, but further investigation found most had overlapping stays at interconnected long-term care facilities and acute care hospitals

within a limited geographic area. The case in Massachusetts was linked to the Illinois cases. The cases in Indiana and Oklahoma occurred in patients who had recently received health care in other countries.

Testing for *C. auris* colonization, using a composite swab of the groin and axilla, was conducted for 390 close contacts of the 77 patients in three states, primarily patients on the same ward in health care facilities because of the risk for environmental contamination and transmission from health care personnel. The two body sites tested were selected based on results of previous investigations. Forty-five (12%) colonized persons were identified (24 in New Jersey, 17 in New York, and four in Illinois). Contact Precautions were recommended for colonized patients in health care facilities. Nasal swabs also were collected from 184 (47%) contacts; two swabs (1%) were positive, both from patients with positive groin/axilla swabs. Environmental testing of patients' rooms identified *C. auris* from mattresses, beds, windowsills, chairs, infusion pumps, and countertops, indicating *C. auris* environmental contamination. *C. auris* was not isolated from rooms after thorough cleaning with a sodium hypochlorite-based disinfectant.

All *C. auris* isolates were forwarded to CDC for whole-genome sequencing and comparison with previously sequenced international isolates, which clustered into four distinct clades (2). Isolates from within each state were highly related. New York isolates, with the exception of one clinical and one screening case, were highly related to one another and grouped in the same clade as isolates from South Asia. Isolates in New Jersey also were similar to those from South Asia but were distinct from those in New York. Illinois isolates were nearly identical to one another and grouped with isolates from South America. These data suggest multiple introductions of *C. auris* into the United States followed by local transmission.

Ongoing investigation of U.S. *C. auris* cases provides epidemiologic and laboratory data suggesting that this fungus can spread within health care facilities and that interventions are needed to prevent transmission during this early stage of *C. auris* emergence. As of May 2017, recognized U.S. *C. auris* cases were concentrated in health care facilities in three separate geographic areas, and most cases were in chronically ill patients with long stays at high-acuity skilled nursing facilities (e.g., facilities providing mechanical ventilation). Apart from one case identified in 2013, clinical laboratories serving health care facilities with *C. auris* cases have not identified suspected *C. auris* isolates from before 2015 from retrospective microbiology record reviews, suggesting recent *C. auris* emergence

FIGURE. Number of health care–associated cases of *Candida auris* infection reported to CDC (N = 77) — seven states, May 2013–May 2017

in those locations. However, the disease might exist elsewhere, because some laboratories do not fully characterize *Candida* species or are otherwise unable to detect *C. auris*.

CDC has worked with state and local partners to develop and share infection control recommendations to help curb the spread of *C. auris* (3). Current recommendations for *C. auris*–colonized or infected patients include 1) use of Standard Precautions and Contact Precautions, 2) housing the patient in a private room, 3) daily and terminal cleaning of a patient's room with a disinfectant active against *Clostridium difficile* spores (an update from previous disinfectant recommendations) (4), and 4) notification of receiving health care facilities when a patient with *C. auris* colonization or infection is transferred. Accurate identification of *C. auris* and adherence to infection control practices, coupled with ongoing public health surveillance and investigations, are needed to halt the spread of *C. auris* in the United States.

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<sup>1</sup>Division of Foodborne, Waterborne, and Environmental Diseases, National Center for Emerging and Zoonotic Infectious Diseases, CDC; <sup>2</sup>Epidemic Intelligence Service, CDC; <sup>3</sup>New York State Department of Health; <sup>4</sup>Division of Epidemiology, New York City Department of Health and Mental Hygiene; <sup>5</sup>School of Public Health, University at Albany, State University of New York; <sup>6</sup>Chicago Department of Public Health; <sup>7</sup>New Jersey Department of Health; <sup>8</sup>Massachusetts Department of Public Health; <sup>9</sup>Indiana State Department of Health; <sup>10</sup>Oklahoma State Department of Health; <sup>11</sup>Division of Healthcare Quality Promotion, National Center for Emerging and Zoonotic Infectious Diseases, CDC.

Corresponding author: Sharon Tsay, [tsay@cdc.gov](mailto:tsay@cdc.gov), 404-718-5510.

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## Announcements

### Click It or Ticket Campaign — May 22–June 4, 2017

In 2015, a total of 22,441 passenger vehicle occupants died in motor vehicle crashes in the United States, representing a 6.6% increase from 2014. Among those who died, 48% were unrestrained by a seat belt (or an age- and size-appropriate car seat or booster seat for younger children) at the time of the crash, whereas only 14% of 38,152 passenger vehicle occupants who survived a crash where at least one person died were unrestrained (1). Using a seat belt is one of the most effective ways to prevent serious injury or death among older children, teens, and adults in the event of a crash (2). Despite the effectiveness of seat belts, millions of persons in the United States continue to travel unrestrained (3).

*Click It or Ticket* is a national campaign coordinated annually by the National Highway Traffic Safety Administration (NHTSA) to increase proper use of seat belts through safety education and strong law enforcement. *Click It or Ticket* takes place from May 22 to June 4, 2017. Law enforcement agencies across the nation will conduct intensive, high-visibility enforcement of seat belt laws, which has been demonstrated to be effective in increasing seat belt use (4). Enforcement is particularly encouraged from 6 p.m. until 5:59 a.m., because seat belt use is lower at night (1). Additional information and publication materials for the 2017 *Click It or Ticket* campaign are available from the NHTSA website at <https://www.traffic-safetymarketing.gov/get-materials/seat-belts/click-it-or-ticket>.

State-specific fact sheets on seat belt use, strategies to increase restraint use, and costs of crash deaths are available from CDC at <https://www.cdc.gov/motorvehiclesafety/seatbelts/states.html> and <https://www.cdc.gov/motorvehiclesafety/statecosts/index.html>. Additional information on preventing motor vehicle crash related injuries is available from CDC at <https://www.cdc.gov/motorvehiclesafety>.

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### World IBD Day — May 19, 2017

World IBD Day is recognized on May 19 to raise awareness of inflammatory bowel disease (IBD) and the two conditions that comprise it: Crohn's disease and ulcerative colitis, both of which cause chronic inflammation of the gastrointestinal tract. World IBD Day is sponsored by the European Federation of Crohn's and Ulcerative Colitis Associations, which includes the Crohn's & Colitis Foundation.

In 2015, approximately 3 million adults in the United States self-reported having a diagnosis of IBD, representing a large increase from <2 million in 1999 (1,2). Symptoms can include frequent diarrhea, abdominal pain, and bloody stools, as well as fever, weight loss, fatigue, and night sweats. The cause of IBD is not known, but genetic susceptibilities, problems with the immune system, and environmental exposures, such as smoking and certain microorganisms, all might play a role. Although to date, IBD cannot be prevented, there are ways to manage the symptoms and prevent complications. Additional information is available at <https://www.cdc.gov/ibd/>. Additional information on World IBD Day is available from the Crohn's & Colitis Foundation (<http://www.crohnscolitisfoundation.org/WorldIBDDay/>).

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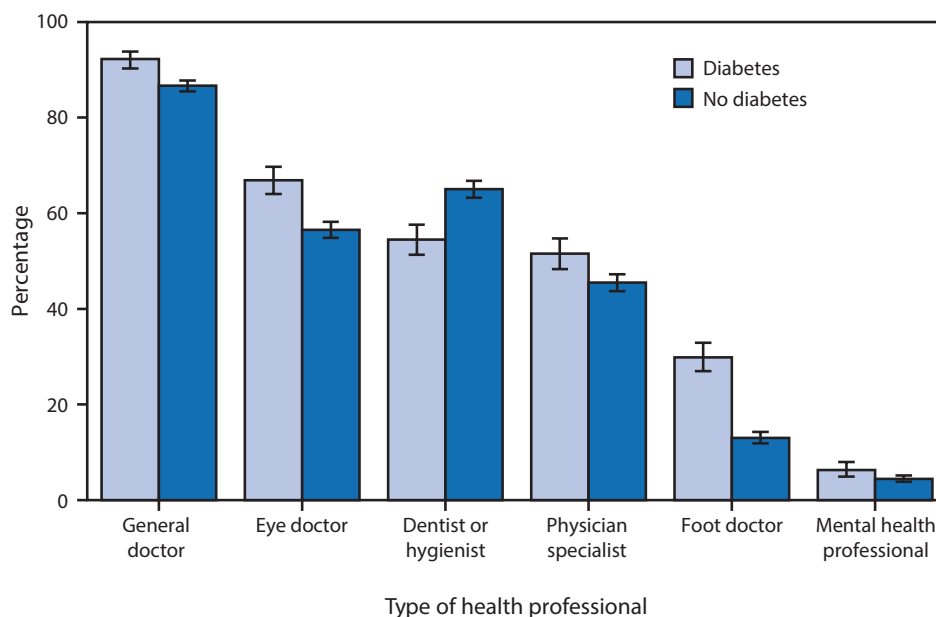
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## QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

## Percentage\* of Adults Aged $\geq 65$ Years Who Saw Selected Types of Health Professionals<sup>†</sup> in the Past 12 Months, by Diagnosed Diabetes Status<sup>§</sup> — National Health Interview Survey, 2015



\* With 95% confidence intervals indicated by error bars.

<sup>†</sup> Based on responses to the following questions: "During the past 12 months, have you seen or talked to any of the following health care providers about your own health? A general doctor who treats a variety of illnesses (a doctor in general practice, family medicine, or internal medicine)? An optometrist, ophthalmologist, or eye doctor (someone who prescribes eyeglasses)? A medical doctor who specializes in a particular medical disease or problem (other than obstetrician/gynecologist, psychiatrist, or ophthalmologist)? A foot doctor? A mental health professional such as a psychiatrist, psychologist, psychiatric nurse, or clinical social worker? About how long has it been since you last saw a dentist, including all types of dentists, such as orthodontists, oral surgeons, and all other dental specialists, as well as dental hygienists?"

<sup>§</sup> Diabetes status was determined by a positive response to the survey question, "Have you ever been told by a doctor or other health professional that you have diabetes or sugar diabetes?" Women were asked not to include diabetes occurring during pregnancy.

In 2015, adults aged  $\geq 65$  years with diagnosed diabetes were more likely than adults without diagnosed diabetes to report seeing general doctors (92.3% compared with 86.7%); eye doctors (66.9% compared with 56.6%); physician specialists (51.5% compared with 45.5%); foot doctors (29.9% compared with 13.0%) and mental health professionals (6.3% compared with 4.5%) in the past 12 months. Those with diabetes were less likely than those without diabetes to report seeing a dentist or dental hygienist in the past 12 months (54.5% compared with 65.0%).

**Source:** National Health Interview Survey, 2015 data. <https://www.cdc.gov/nchs/nhis.htm>.

**Reported by:** Sarah E. Lessem, PhD, [slessem@cdc.gov](mailto:slessem@cdc.gov), 301-458-4209; Robin P. Pendley, DrPH.





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