Centers for Disease Control and Prevention

MMWR

Morbidity and Mortality Weekly Report

Weekly / Vol. 66 / No. 28

July 21, 2017

Racial and Ethnic Differences in Homicides of Adult Women and the Role of Intimate Partner Violence — United States, 2003–2014

Emiko Petrosky, MD[1]; Janet M. Blair, PhD[1]; Carter J. Betz, MS[1]; Katherine A. Fowler, PhD[1]; Shane P.D. Jack, PhD[1]; Bridget H. Lyons, MPH[1]

Homicide is one of the leading causes of death for women aged ≤44 years.* In 2015, homicide caused the death of 3,519 girls and women in the United States. Rates of female homicide vary by race/ethnicity (*1*), and nearly half of victims are killed by a current or former male intimate partner (*2*). To inform homicide and intimate partner violence (IPV) prevention efforts, CDC analyzed homicide data from the National Violent Death Reporting System (NVDRS) among 10,018 women aged ≥18 years in 18 states during 2003–2014. The frequency of homicide by race/ethnicity and precipitating circumstances of homicides associated with and without IPV were examined. Non-Hispanic black and American Indian/Alaska Native women experienced the highest rates of homicide (4.4 and 4.3 per 100,000 population, respectively). Over half of all homicides (55.3%) were IPV-related; 11.2% of victims of IPV-related homicide experienced some form of violence in the month preceding their deaths, and argument and jealousy were common precipitating circumstances. Targeted IPV prevention programs for populations at disproportionate risk and enhanced access to intervention services for persons experiencing IPV are needed to reduce homicides among women.

CDC's NVDRS is an active state-based surveillance system that monitors characteristics of violent deaths, including homicides. The system links three data sources (death certificates, coroner/medical examiner reports, and law enforcement reports) to create a comprehensive depiction of who dies from violence, where and when victims die, and factors perceived to contribute to the victim's death (*3*). This report includes NVDRS data from 18 states during 2003–2014 (all available years).† Five racial/ethnic categories§ were used for this analysis: white, black, American Indian/Alaska Native

* CDC's Web-based Injury Statistics Query and Reporting System (WISQARS). https://www.cdc.gov/injury/wisqars/index.html.

† In 2003, the National Violent Death Reporting System (NVDRS) began data collection with six states (Maryland, Massachusetts, New Jersey, Oregon, South Carolina, and Virginia) participating; seven states (Alaska, Colorado, Georgia, North Carolina, Oklahoma, Rhode Island, and Wisconsin) joined in 2004, four (California, Kentucky, New Mexico, and Utah) in 2005, and two (Ohio and Michigan) in 2010. California did not collect statewide data and concluded participation in 2009. Ohio collected statewide data starting in 2011 and Michigan starting in 2014. CDC provides funding for state participation, and the ultimate goal is for NVDRS to expand to include all 50 states, U.S. territories, and the District of Columbia.

§ Information on race and ethnicity are recorded as separate items in NVDRS consistent with U.S. Department of Health and Human Services (HHS) and Office of Management and Budget standards for race/ethnicity categorization. HHS guidance on race/ethnicity is available at https://aspe.hhs.gov/datacncl/standards/ACA/4302/index.shtml.

INSIDE

747 Surveillance for Silicosis Deaths Among Persons Aged 15–44 Years — United States, 1999–2015
753 Progress Toward Measles Elimination — Bangladesh, 2000–2016
758 Notes from the Field: Cluster of Acute Flaccid Myelitis in Five Pediatric Patients — Maricopa County, Arizona, 2016
761 Notes from the Field: *Cronobacter sakazakii* Infection Associated with Feeding Extrinsically Contaminated Expressed Human Milk to a Premature Infant — Pennsylvania, 2016
763 Notes from the Field: Hospital Contact Investigation for a Patient Who Developed a Zoster Vaccine–Related Rash — Maryland, February 2015
765 Announcement
766 QuickStats

Continuing Education examination available at https://www.cdc.gov/mmwr/cme/conted_info.html#weekly.

U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

(AI/AN), Asian/Pacific Islander (A/PI), and Hispanic. Persons categorized as Hispanic might have been of any race. Persons categorized as one of the four racial populations were all non-Hispanic. Analyses were limited to female decedents aged ≥18 years. IPV-related deaths were defined as those involving intimate partner homicides (i.e., the victim was an intimate partner [e.g., current, former, or unspecified spouse or girlfriend] of the suspect), other deaths associated with IPV, including victims who were not the intimate partner (i.e., family, friends, others who intervened in IPV, first responders, or bystanders), or jealousy. Deaths where jealousy, such as in a lovers' triangle, was noted as a factor were included only when they involved an actual relationship (versus unrequited interest). Violence experienced in the preceding month refers to all types of violence (e.g., robbery, assault, or IPV) that was distinct and occurred before the violence that killed the victim; there did not need to be any causal link between the earlier violence and the death itself (e.g., victim could have experienced a robbery by a stranger 2 weeks before being killed by her spouse).

Rates were calculated using intercensal and postcensal bridged–race population estimates compiled by CDC's National Center for Health Statistics and were age-adjusted to the 2010 standard U.S. population of women aged ≥18 years (*4*). Sociodemographic characteristics and precipitating circumstances across racial/ethnic groups were examined using chi-square and Fisher's exact tests. Two-sided p-values <0.05 were considered statistically significant. Differences in victim and incident characteristics by race/ethnicity were examined using chi-square and Fisher's exact tests with posthoc pairwise comparisons of significant results; Bonferroni correction was applied to account for multiple comparisons.

From 2003 through 2014, a total of 10,018 female homicides were captured by NVDRS; among these, 1,835 (18.3%) were part of a homicide-suicide incident (i.e., suspect died by suicide after perpetrating homicide). Homicide victims ranged in age from 18 to 100 years. The overall age-adjusted homicide rate was 2.0 per 100,000 women. By race/ethnicity, non-Hispanic black women had the highest rate of dying by homicide (4.4 per 100,000), followed by AI/AN (4.3), Hispanic (1.8), non-Hispanic white (1.5), and A/PI women (1.2).

Approximately one third of female homicide victims (29.4%) were aged 18–29 years (Table 1); a larger proportion of non-Hispanic black and Hispanic victims were in this youngest age group than were non-Hispanic white and A/PI victims (p<0.01). The largest proportion of victims were never married or single at the time of death (38.2%); this proportion was highest among non-Hispanic black victims (59.2%; p<0.01). One third of victims had attended some college or more; history of college attendance was highest among non-Hispanic white (36.8%) and A/PI victims (46.2%; p<0.01). Approximately 15% of women of reproductive age (18–44 years) were pregnant or ≤6 weeks postpartum. Firearms were used in 53.9% of female homicides, most commonly among non-Hispanic black victims (57.7%; p<0.01). Sharp instrument (19.8%); hanging, suffocation, or strangulation (10.5%); and blunt instrument (7.9%) were other common

The *MMWR* series of publications is published by the Center for Surveillance, Epidemiology, and Laboratory Services, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30329-4027.

Suggested citation: [Author names; first three, then et al., if more than six.] [Report title]. MMWR Morb Mortal Wkly Rep 2017;66:[inclusive page numbers].

Centers for Disease Control and Prevention

Brenda Fitzgerald, MD, *Director*
William R. Mac Kenzie, MD, *Acting Associate Director for Science*
Joanne Cono, MD, ScM, *Director, Office of Science Quality*
Chesley L. Richards, MD, MPH, *Deputy Director for Public Health Scientific Services*
Michael F. Iademarco, MD, MPH, *Director, Center for Surveillance, Epidemiology, and Laboratory Services*

***MMWR* Editorial and Production Staff (Weekly)**

Sonja A. Rasmussen, MD, MS, *Editor-in-Chief*
Charlotte K. Kent, PhD, MPH, *Executive Editor*
Jacqueline Gindler, MD, *Editor*
Teresa F. Rutledge, *Managing Editor*
Douglas W. Weatherwax, *Lead Technical Writer-Editor*
Soumya Dunworth, PhD, Kristy Gerdes, MPH, Teresa M. Hood, MS, *Technical Writer-Editors*

Martha F. Boyd, *Lead Visual Information Specialist*
Maureen A. Leahy, Julia C. Martinroe, Stephen R. Spriggs, Tong Yang, *Visual Information Specialists*
Quang M. Doan, MBA, Phyllis H. King, Paul D. Maitland, Terraye M. Starr, Moua Yang, *Information Technology Specialists*

***MMWR* Editorial Board**

Timothy F. Jones, MD, *Chairman*
Matthew L. Boulton, MD, MPH
Virginia A. Caine, MD
Katherine Lyon Daniel, PhD
Jonathan E. Fielding, MD, MPH, MBA
David W. Fleming, MD
William E. Halperin, MD, DrPH, MPH
King K. Holmes, MD, PhD
Robin Ikeda, MD, MPH
Rima F. Khabbaz, MD
Phyllis Meadows, PhD, MSN, RN
Jewel Mullen, MD, MPH, MPA
Jeff Niederdeppe, PhD
Patricia Quinlisk, MD, MPH
Patrick L. Remington, MD, MPH
Carlos Roig, MS, MA
William L. Roper, MD, MPH
William Schaffner, MD

TABLE 1. Number and percentage* of homicides of females aged ≥18 years, by victim and incident characteristics — National Violent Death Reporting System, 18 states,† 2003–2014

Characteristic	No. (%)					
	Total (N = 10,018)	White, non-Hispanic (n = 5,206)	Black, non-Hispanic (n = 3,514)	American Indian/ Alaska Native (n = 240)	Asian/Pacific Islander (n = 236)	Hispanic§ (n = 822)
Age group (yrs)						
18–29¶	**2,947 (29.4)**	1,113 (21.4)**,††,§§	1,359 (38.7)¶¶,***	87 (36.3)¶¶	59 (25.0)**,§§	329 (40.0)¶¶,***
30–39¶	**2,179 (21.8)**	990 (19.0)**,§§	829 (23.6)§§,¶¶	56 (23.3)	59 (25.0)	245 (29.8)**,¶¶
40–49¶	**2,071 (20.7)**	1,126 (21.6)	704 (20.0)	52 (21.7)	46 (19.5)	143 (17.4)
50–59¶	**1,293 (12.9)**	824 (15.8)**,§§	352 (10.0)¶¶	25 (10.4)	31 (13.1)	61 (7.4)¶¶
≥60¶	**1,528 (15.3)**	1,153 (22.1)**,††,§§	270 (7.7)¶¶,***	20 (8.3)¶¶,***	41 (17.4)**,††,§§	44 (5.4)¶¶,***
Marital status						
Married, civil union, or domestic partnership¶	**3,156 (32.0)**	1,999 (38.9)**,††,§§,***	751 (21.9)§§,¶¶,***	51 (21.4)¶¶,***	121 (51.7)**,††,§§,¶¶	234 (28.7)**,¶¶,***
Never married or single¶	**3,766 (38.2)**	1,183 (23.0)**,††,§§	2,035 (59.2)††,§§,¶¶,***	118 (49.6)**,¶¶,***	52 (22.2)**,††,§§	378 (46.4)**,¶¶,***
Separated, divorced or widowed¶	**2,938 (29.8)**	1,954 (38.0)**,††,§§,***	651 (18.9)††,§§,¶¶	69 (29.0)**,¶¶	61 (26.1)¶¶	203 (24.9)**,¶¶
Education†††						
<High school graduate or GED equivalent¶	**2,143 (24.5)**	982 (21.2)**,††,§§	749 (25.6)§§,¶¶	75 (32.5)¶¶,***	39 (18.6)††,§§	298 (39.8)**,¶¶,***
High school graduate or GED equivalent¶	**3,672 (41.9)**	1,952 (42.1)	1,261 (43.0)	105 (45.5)	74 (35.2)	280 (37.4)
Some college or more¶	**2,946 (33.6)**	1,707 (36.8)**,††,§§	921 (31.4)††,§§,¶¶,***	51 (22.1)**,¶¶,***	97 (46.2)**,††,§§	170 (22.7)**,¶¶,***
Pregnancy status§§§						
Pregnant or ≤6 weeks postpartum¶	**298 (15.2)**	120 (12.9)**	134 (18.6)¶¶	7 (13.2)	6 (14.3)	31 (14.6)
Method						
Firearm¶	**5,234 (53.9)**	2,681 (53.4)**,††,***	1,975 (57.7)††,§§,¶¶,***	90 (38.8)**,§§,¶¶	92 (40.0)**,¶¶	396 (49.4)**,††
Sharp instrument¶	**1,918 (19.8)**	878 (17.5)**,§§,***	715 (20.9)§§,¶¶,***	49 (21.1)	70 (30.4)**,¶¶	206 (25.7)**,¶¶
Hanging, suffocation, strangulation¶	**1,017 (10.5)**	542 (10.8)	325 (9.5)§§	15 (6.5)	32 (13.9)	103 (12.9)**
Blunt instrument¶	**770 (7.9)**	453 (9.0)**,††,§§	216 (6.3)††,¶¶	40 (17.2)**,§§,¶¶,***	16 (7.0)††	45 (5.6)††,¶¶
Other (single method)¶	**765 (7.9)**	467 (9.3)**,††	189 (5.5)††,¶¶	38 (16.4)**,§§,¶¶	20 (8.7)	51 (6.4)††
IPV¶¶¶						
IPV-related¶,****	**4,442 (55.3)**	2,446 (56.8)**	1,360 (51.3)§§,¶¶	112 (55.4)	118 (57.8)	406 (61.0)**

Abbreviations: GED = General Education Development; IPV = intimate partner violence.

* Excludes decedents with missing, unknown, and other race/ethnicity (n = 61). Percentages might not sum to 100% because of rounding.

† Alaska, Colorado, Georgia, Kentucky, Maryland, Massachusetts, Michigan, New Jersey, New Mexico, North Carolina, Ohio, Oklahoma, Oregon, Rhode Island, South Carolina, Utah, Virginia, and Wisconsin.

§ Includes persons of any race.

¶ Characteristic with a statistically significant result.

** Significantly different from non-Hispanic black females.

†† Significantly different from American Indian/Alaska Native females.

§§ Significantly different from Hispanic females.

¶¶ Significantly different from non-Hispanic white females.

*** Significantly different from Asian/Pacific Islander females.

††† "<High school graduate/GED equivalent" includes 11th grade and below. "High school graduate/GED equivalent" includes 12th grade. "Some college or more" includes some college credit, associate's degree, master's degree, doctorate, and professional degrees.

§§§ Includes only females of reproductive age (18–44 years) with known pregnancy status (n = 1,957).

¶¶¶ Includes only decedents where circumstances were known (n = 8,028).

**** Includes cases with victim-suspect relationship of intimate partner (current, former, or unspecified spouse or girlfriend), other deaths associated with IPV, or IPV-related jealousy/lovers' triangle.

mechanisms. Over half of all female homicides (55.3%) for which circumstances were known were IPV-related. A larger percentage of IPV-related female homicides were perpetrated by male suspects than were non-IPV-related homicides (98.2% versus 88.5%, respectively; p<0.01).

Circumstance information was known for all 4,442 IPV-related homicides and 3,586 (64.3%) non-IPV-related homicides and was examined further. Among IPV-related homicides, 79.2% and 14.3% were perpetrated by a current or former intimate partner, respectively (Table 2). Approximately one in 10 victims experienced some form of violence in the month preceding their death. However, only 11.2% of all IPV-related homicides were precipitated by another crime; 54.4% of these incidents involved another crime in progress. The most frequently reported other precipitating crimes were assault/homicide (45.6%), rape/sexual assault (11.1%), and burglary

TABLE 2. Number and percentage* of homicides of females aged ≥18 years, by race/ethnicity, victim's relationship to suspect, and precipitating circumstances† for intimate partner violence (IPV)–related deaths — National Violent Death Reporting System, 18 states,§ 2003–2014

	No. (%)					
Characteristic	Total (N = 4,442)	White, non-Hispanic (n = 2,446)	Black, non-Hispanic (n = 1,360)	American Indian/ Alaska Native (n = 112)	Asian/Pacific Islander (n = 118)	Hispanic¶ (n = 822)
Victim-suspect relationship**						
Current intimate†† partner	**3,417 (79.2)**	1,927 (81.0)§§	1,007 (76.6)¶¶	88 (81.5)	94 (81.0)	301 (75.8)
Former intimate partner††	**618 (14.3)**	322 (13.5)	198 (15.1)	13 (12.0)	11 (9.5)	74 (18.6)
Other††,***	**278 (6.4)**	129 (5.4)§§	109 (8.3)¶¶	7 (6.5)	11 (9.5)	22 (5.5)
Circumstances						
Victim experienced violence in the past month†††	**265 (11.2)**	147 (10.8)	66 (9.9)	10 (16.7)	9 (12.9)	33 (15.6)
Precipitated by another crime	**496 (11.2)**	261 (10.7)	166 (12.2)	10 (8.9)	13 (11.0)	46 (11.3)
Crime in progress§§§	**270 (54.4)**	137 (52.5)	93 (56.0)	7 (70.0)	7 (53.8)	26 (56.5)
Argument preceded victim's death††	**1,320 (29.7)**	660 (27.0)¶¶¶	420 (30.9)¶¶¶	36 (32.1)	42 (35.6)	162 (39.9)§§,¶¶
Jealousy/lovers' triangle††	**516 (11.6)**	262 (10.7)¶¶¶	143 (10.5)¶¶¶	21 (18.8)	13 (11.0)	77 (19.0)§§,¶¶

* Includes only decedents with one or more circumstances present: n = 4,442 (100%) IPV-related female homicides.
† The sum of percentages in columns exceeds 100% because more than one circumstance could have been present per decedent.
§ Alaska, Colorado, Georgia, Kentucky, Maryland, Massachusetts, Michigan, New Jersey, New Mexico, North Carolina, Ohio, Oklahoma, Oregon, Rhode Island, South Carolina, Utah, Virginia, and Wisconsin.
¶ Includes persons of any race.
** Victim-suspect relationship known for 4,313 (97.1%) IPV-related female homicides.
†† Characteristic with statistically significant results.
§§ Significantly different from non-Hispanic black females.
¶¶ Significantly different from non-Hispanic white females.
*** Includes nonintimate partner victims of IPV-related female homicide (e.g., friend, family member, etc.).
††† Variable collected for homicides since 2009. Denominator is IPV-related female homicides during 2009–2014 (n = 2,369).
§§§ Denominator includes only those decedents involved in an incident that was precipitated by another crime.
¶¶¶ Significantly different from Hispanic females.

TABLE 3. Number and percentage* of homicides of females aged ≥18 years, by race/ethnicity, victim's relationship to suspect and precipitating circumstances† for nonintimate partner violence (IPV)–related deaths — National Violent Death Reporting System, 18 states,§ 2003–2014

	No. (%)					
Characteristic	Total (N = 3,586)	White, non-Hispanic (n = 1,859)	Black, non-Hispanic (n = 1,291)	American Indian/ Alaska Native (n = 90)	Asian/Pacific Islander (n = 86)	Hispanic¶ (n = 260)
Victim-suspect relationship**						
Acquaintance††	**439 (19.7)**	188 (14.9)§§	190 (29.0)¶¶	16 (24.2)	9 (14.3)	36 (20.7)
Stranger††	**349 (15.7)**	176 (13.9)***,†††	103 (15.7)	10 (15.2)	18 (28.6)¶¶	42 (24.1)¶¶
Other person, known to victim	**339 (15.2)**	195 (15.4)	103 (15.7)	9 (13.6)	8 (12.7)	24 (13.8)
Parent††	**337 (15.2)**	237 (18.7)§§,†††	79 (12.0)¶¶	4 (6.1)	7 (11.1)	10 (5.7)¶¶
Other††	**760 (34.2)**	469 (37.1)§§	181 (27.6)¶¶	27 (40.9)	21 (33.3)	62 (35.6)
Circumstances						
Precipitated by another crime††	**1,492 (41.6)**	788 (42.4)	526 (40.7)***	37 (41.1)	49 (57.0)§§,†††	92 (35.4)***
Crime in progress§§§	**1,002 (67.2)**	535 (67.9)	345 (65.6)	25 (67.6)	33 (67.3)	64 (69.6)
Argument preceded victim's death††	**1,357 (37.8)**	659 (35.4)§§	531 (41.1)¶¶,***	43 (47.8)***	22 (25.6)§§,¶¶¶	102 (39.2)

* Denominator includes only decedents with one or more circumstances present: n = 3,586 (64.3%) non-IPV related homicides.
† The sum of percentages in columns exceeds 100% because more than one circumstance could have been present per decedent.
§ Alaska, Colorado, Georgia, Kentucky, Maryland, Massachusetts, Michigan, New Jersey, New Mexico, North Carolina, Ohio, Oklahoma, Oregon, Rhode Island, South Carolina, Utah, Virginia, and Wisconsin.
¶ Includes persons of any race.
** Victim-suspect relationship known for 2,224 (62.0%) non-IPV-related female homicide victims.
†† Characteristic with a statistically significant result.
§§ Significantly different from non-Hispanic black females.
¶¶ Significantly different from non-Hispanic white females.
*** Significantly different from Asian/Pacific Islander females.
††† Significantly different from Hispanic females.
§§§ Denominator includes only those decedents involved in an incident that was precipitated by another crime.
¶¶¶ Significantly different from American Indian/Alaska Native females.

(9.9%). In 29.7% of IPV-related homicides, an argument preceded the victim's death; this occurred more commonly among Hispanic victims than among non-Hispanic black and white victims. Approximately 12% of IPV-related homicides were associated with jealousy; this circumstance was also documented more commonly among Hispanic victims than among non-Hispanic black and white victims.

Among non-IPV related female homicides with known suspects, the victim's relationship to the suspect was most often that of acquaintance (19.7%), stranger (15.7%), another person known to the victim in which the exact nature of the relationship or prior interaction was unclear (15.2%), or parent (15.2%) (Table 3). Non-Hispanic black victims were significantly more likely to be killed by an acquaintance (29.0%) than were non-Hispanic white victims (14.9%). A/PI and Hispanic victims were significantly more likely to be killed by a stranger (28.6% and 24.1%, respectively) than were non-Hispanic white victims (13.9%). Fewer than 2% of non-IPV related homicide victims experienced violence during the preceding month (data not shown). However, a substantial percentage of these homicides (41.6%) were precipitated by another crime; 67.2% of these incidents involved another crime in progress. The type of other precipitating crime was most frequently robbery (31.1%), assault/homicide (21.3%), burglary (12.2%), or rape/sexual assault (11.2%). Female homicides involving A/PI victims were more likely to be precipitated by another crime (57.0%) than were homicides involving non-Hispanic black (40.7%) and Hispanic (35.4%) victims. In 37.8% of non-IPV related homicides, an argument preceded the victim's death, more commonly among AI/AN (47.8%) and non-Hispanic black (41.1%) victims than among A/PI (25.6%) victims.

Discussion

Homicide is the most severe health outcome of violence against women. Findings from this study of female homicides from NVDRS during 2003–2014 indicate that young women, particularly racial/ethnic minority women, were disproportionately affected. Across all racial/ethnic groups of women, over half of female homicides for which circumstances were known were IPV-related, with >90% of these women being killed by their current or former intimate partner.

Strategies to prevent IPV-related homicides range from protecting women from immediate harm and intervening in current IPV, to developing and implementing programs and policies to prevent IPV from occurring (*5*). IPV lethality risk assessments conducted by first responders have shown high sensitivity in identifying victims at risk for future violence and homicide (*6*). These assessments might be used to facilitate immediate safety planning and to connect women with other services, such as crisis intervention and counseling, housing, medical and legal advocacy, and access to other community resources (*6*). State statutes limiting access to firearms for persons under a domestic violence restraining order can serve as another preventive measure associated with reduced risk for intimate partner homicide and firearm intimate partner homicide (*7*). Approximately one in 10 victims of IPV-related homicide experienced some form of violence in the preceding month, which could have provided opportunities for intervention. Bystander programs, such as Green Dot,[¶] teach participants how to recognize situations or behaviors that might become violent and safely and effectively intervene to reduce the likelihood of assault (*8*). In health care settings, the U.S. Preventive Services Task Force recommends screening women of childbearing age for IPV and referring women who screen positive for intervention services.[**] Approximately 15% of female homicide victims of reproductive age (18–44 years) were pregnant or postpartum, which might or might not be higher than estimates in the general U.S. female population, requiring further examination.

Approximately 40% of non-Hispanic black, AI/AN, and Hispanic female homicide victims were aged 18–29 years. Argument and jealousy were common precipitating factors for IPV-related homicides. Teaching safe and healthy relationship skills is an important primary prevention strategy with evidence of effectiveness in reducing IPV by helping young persons manage emotions and relationship conflicts and improve their problem-solving and communication skills (*5*). Preventing IPV also requires addressing the community- and system-level factors that increase the risk for IPV; neighborhoods with high disorder, disadvantage, and poverty, and low social cohesion are associated with increased risk of IPV (*5*), and underlying health inequities caused by barriers in language, geography, and cultural familiarity might contribute to homicides, particularly among racial/ethnic minority women (*9*).

The findings in this report are subject to at least five limitations. First, NVDRS data are available from a limited number of states and are therefore not nationally representative. Second, race/ethnicity data on death certificates might be misclassified, particularly for Hispanics, A/PI, and AI/AN (*10*). Third, the female homicide victims in this dataset were more likely to be never married or single and less likely to have attended college than the general U.S. female population[††]; although this is likely attributable to the relatively younger age distribution of homicide victims in general,[§§] this requires further

¶ http://www.livethegreendot.com.

** https://www.uspreventiveservicestaskforce.org/Page/Document/RecommendationStatementFinal/intimate-partner-violence-and-abuse-of-elderly-and-vulnerable-adults-screening.

†† https://www.census.gov/acs/www/data/data-tables-and-tools/.

§§ https://www.cdc.gov/nchs/data/nvsr/nvsr65/nvsr65_04.pdf.

Summary

What is already known about this topic?

Homicide is one of the leading causes of death for women aged ≤44 years, and rates vary by race/ethnicity. Nearly half of female victims are killed by a current or former male intimate partner.

What is added by this report?

Homicides occur in women of all ages and among all races/ethnicities, but young, racial/ethnic minority women are disproportionately affected. Over half of female homicides for which circumstances were known were related to intimate partner violence (IPV). Arguments and jealousy were common precipitating circumstances among IPV-related homicides. One in 10 victims of IPV-related homicide were reported to have experienced violence in the month preceding their deaths.

What are the implications for public health practice?

Racial/ethnic differences in female homicide underscore the importance of targeting intervention efforts to populations at risk and the conditions that increase the risk for violence. IPV lethality risk assessments might be useful tools for first responders to identify women at risk for future violence and connect them with life-saving safety planning and services. Teaching young persons safe and healthy relationship skills as well as how to recognize situations or behaviors that might become violent are effective IPV primary prevention measures.

examination. Fourth, not all homicide cases include detailed suspect information; in this analysis, 85.3% of cases included information on the suspect. Finally, information about male corollary victims of IPV-related homicide (i.e., other deaths associated with IPV, including male victims who were not the intimate partner) were not included in this analysis. Therefore, the full scope of IPV-related homicides involving women is not captured.

The racial/ethnic differences in female homicide underscore the importance of targeting prevention and intervention efforts to populations at disproportionately high risk. Addressing violence will require an integrated response that considers the influence of larger community and societal factors that make violence more likely to occur.

Acknowledgments

Linda Dahlberg, PhD, Keming Yuan, MS, Division of Violence Prevention, National Center for Injury Prevention and Control, CDC.

Conflict of Interest

No conflicts of interest were reported.

[1]Division of Violence Prevention, National Center for Injury Prevention and Control, CDC.

Corresponding author: Emiko Petrosky, xfq7@cdc.gov, 770-488-4399.

References

1. Logan JE, Smith SG, Stevens MR. Homicides—United States, 1999–2007. MMWR Suppl 2011;60:67–70.
2. Catalano S, Smith E, Snyder H, Rand M. Selected findings: female victims of violence. Washington, DC: US Department of Justice, Bureau of Justice Statistics; 2009. https://www.bjs.gov/content/pub/pdf/fvv.pdf
3. Blair JM, Fowler KA, Jack SPD, Crosby AE. The National Violent Death Reporting System: overview and future directions. Inj Prev 2016;22(Suppl 1):i6–11. https://doi.org/10.1136/injuryprev-2015-041819
4. National Center for Health Statistics. Healthy people 2010: general data issues. Age adjustment. Hyattsville, MD: US Department of Health and Human Services, CDC, National Center for Health Statistics; 2011. https://www.cdc.gov/nchs/data/hpdata2010/hp2010_general_data_issues.pdf
5. Niolon PH, Kearns M, Dills J, et al. Preventing intimate partner violence across the lifespan: a technical package of programs, policies and practices. Atlanta, GA: US Department of Health and Human Services, CDC, National Center for Injury Prevention and Control; 2017. https://www.cdc.gov/violenceprevention/pdf/ipv-technicalpackages.pdf
6. Messing JT, Campbell J, Sullivan Wilson J, Brown S, Patchell B. The lethality screen: the predictive validity of an intimate partner violence risk assessment for use by first responders. J Interpers Violence 2017;32:205–26.
7. Zeoli AM, Webster DW. Effects of domestic violence policies, alcohol taxes and police staffing levels on intimate partner homicide in large US cities. Inj Prev 2010;16:90–5. https://doi.org/10.1136/ip.2009.024620
8. Coker AL, Bush HM, Fisher BS, et al. Multi-college bystander intervention evaluation for violence prevention. Am J Prev Med 2016;50:295–302. https://doi.org/10.1016/j.amepre.2015.08.034
9. Smedley BD, Stith AY, Nelson AR, eds.; Institute of Medicine (US) Committee on Understanding and Eliminating Racial and Ethnic Disparities in Health Care. Unequal treatment: confronting racial and ethnic disparities in health care. Washington, DC: National Academies Press; 2003.
10. Arias E, Schauman WS, Eschbach K, Sorlie PD, Backlund E. The validity of race and Hispanic origin reporting on death certificates in the United States. Vital Health Stat 2 2008;148:1–23.

Surveillance for Silicosis Deaths Among Persons Aged 15–44 Years — United States, 1999–2015

Jacek M. Mazurek, MD, PhD[1]; John M. Wood, MS[1]; Patricia L. Schleiff, MS[1]; David N. Weissman, MD[1]

Silicosis is usually a disease of long latency affecting mostly older workers; therefore, silicosis deaths in young adults (aged 15–44 years) suggests acute or accelerated disease.* To understand the circumstances surrounding silicosis deaths among young persons, CDC analyzed the underlying and contributing causes† of death using multiple cause-of-death data (1999–2015) and industry and occupation information abstracted from death certificates (1999–2013). During 1999–2015, among 55 pneumoconiosis deaths of young adults with *International Classification of Diseases, Tenth Revision* (ICD-10) code J62 (pneumoconiosis due to dust containing silica),§ 38 (69%) had code J62.8 (pneumoconiosis due to other dust containing silica), and 17 (31%) had code J62.0 (pneumoconiosis due to talc dust) listed on their death certificate. Decedents whose cause of death code was J62.8 most frequently worked in the manufacturing and construction industries and production occupations where silica exposure is known to occur. Among the 17 decedents who had death certificates listing code J62.0 as cause of death, 13 had certificates with an underlying or a contributing cause of death code listed that indicated multiple drug use or drug overdose. In addition, 13 of the 17 death certificates listing code J62.0 as cause of death had information on decedent's industry and occupation; among the 13 decedents, none worked in talc exposure–associated jobs, suggesting that their talc exposure was nonoccupational. Examining detailed information on causes of death (including external causes) and industry and occupation of decedents is essential for identifying silicosis deaths associated with occupational exposures and reducing misclassification of silicosis mortality.

Various occupationally associated pulmonary diseases are linked to exposure to silica and silicates, a large class of minerals that includes talc (hydrous magnesium silicate) and other nonfibrous silicate minerals (*1*). Silicosis is caused by inhaling respirable crystalline silica. Occupational exposure to airborne respirable silica particles has been associated with work in mining, quarrying, tunneling, construction, sandblasting, masonry, foundry operations, glass manufacture, ceramic and pottery production, and cement and concrete production and with work with certain materials in dental laboratories (*2*). Newly emerging occupations and tasks, including fabricating and installing quartz-containing engineered stone products and extracting natural gas by hydraulic fracturing also place workers at risk for silicosis.¶ Approximately 2.3 million workers might be exposed to respirable crystalline silica in the United States.**

Exposure to talc causes talcosis (talco-silicosis or talco-asbestosis if talc is contaminated with silica or asbestos fibers, respectively); inhalation of talc usually results from occupational exposures during talc mining and milling and during production of ceramics, pharmaceuticals, paint, paper, cosmetics, plastics, roofing, rubber, insecticides, and other products (*3*). Although only 240 workers were employed in talc mining in the United States during 2015 (the number of workers exposed to talc in milling and secondary industries is unknown), 803,000 metric tons of talc were used in various products that year.†† Nonoccupational exposure to talc dust has been associated with use of cosmetic talcum powder (*4*) and, importantly, with illicit intravenous or inhalation administration of talc-containing legal or illegal drugs, including marijuana, methamphetamine, methadone, promethazine, cocaine, diazepam, acetaminophen, meperidine, pentazocine, oxymorphone, and heroin (*3*,*5*–*7*).

To investigate silicosis deaths among young adults, ICD-10 codes for underlying and contributing causes of death from the 1999–2015 National Center for Health Statistics' multiple cause-of-death mortality data were analyzed to provide detailed information on the circumstances surrounding pneumoconiosis deaths among young adults caused by dust containing silica. Time trends were assessed using a linear

* Chronic silicosis, the most common form of silicosis, occurs after exposure to relatively low silica concentrations for >10 years. Accelerated silicosis occurs after 5–10 years of exposure to higher silica levels, and acute silicosis can occur after only weeks or months of exposure to extremely high silica concentrations.

† Records included on the entity axis that reflect the placement of each condition on the certificate for each decedent (https://www.cdc.gov/nchs/data/dvs/2b_2016.pdf); each record includes codes for one underlying cause of death (the disease or injury that initiated the chain of events that led directly and inevitably to death) and up to 20 contributing causes of death. https://webappa.cdc.gov/ords/norms-glossary.html.

§ *International Classification of Diseases, Tenth Revision* (ICD-10) code J62, pneumoconiosis due to dust containing silica category, is further subdivided into pneumoconiosis due to talc dust (J62.0) and pneumoconiosis due to other dust containing silica (J62.8). http://apps.who.int/classifications/icd10/browse/2016/en#/J62.

¶ https://www.cdc.gov/niosh/topics/silica.

** https://www.osha.gov/silica/.

†† https://minerals.usgs.gov/minerals/pubs/mcs/2017/mcs2017.pdf.

regression model. Twenty-one states provided copies of actual death certificates[§§] from 1999 through 2013; usual industry and occupation entries were abstracted from these certificates and were coded using the National Institute for Occupation Safety and Health's Industry and Occupation Computerized Coding System.[¶¶]

During 1999–2015, a total of 55 young adult decedents had ICD-10 code J62 assigned as either the underlying or a contributing cause of death, including 38 (69%) with ICD-10 subcategory J62.8 listed as the underlying (27) or a contributing (11) cause of death. The mean age of these 38 decedents was 38.6 years; most were males (95%), white (82%), non-Hispanic (74%), and born in the United States (71%) (Table 1). None of these 38 deaths involved multiple drug use or drug overdose; three (8%) had received subcutaneous silicone injections.[***]

Seventeen (31%) of the 55 decedents had subcategory J62.0 listed as the underlying (11) or contributing (6) cause of death. The mean age of these decedents was 37.5 years; slightly more than half (9) were male, 13 were white, 15 were non-Hispanic, and all were born in the United States. Thirteen of these 17 deaths involved multiple drug use and drug overdose. [†††] The number of pneumoconiosis deaths due to other dust containing silica and due to talc dust among young adults remained stable during 1999–2015 (Table 1).

To evaluate industry and occupation of decedents with a diagnosis of silicosis, CDC obtained death certificates for 47 young adult decedents reported during 1999–2013 from 21 states[§§§] who had ICD-10 code J62 assigned as the underlying or contributing cause of death. Industry and occupation entries recorded on death certificates were reviewed, including 34 (97%) certificates for 35 deaths with any mention of pneumoconiosis due to other dust containing silica and all certificates for 13 deaths with any mention of pneumoconiosis due to talc dust during 1999–2013. Among the 35 decedents with a diagnosis of pneumoconiosis due to other dust containing silica, the majority were associated with working in the manufacturing (e.g., cut stone and stone product manufacturing industry) (12 [34%]) and construction (7 [20%]) industry sectors; 11 (31%) were working in production (e.g., crushing, grinding, polishing, mixing, and blending workers) occupations; five (14%) in construction and extraction occupations; and three (9%) as brickmasons and blockmasons (Table 2). These industries and occupations have well-established associations with exposure to crystalline silica (*2*). Among the 13 decedents whose death certificates included any mention of pneumoconiosis due to dust containing talc, none was employed in an industry or occupation traditionally associated with exposure to talc. Ten of these 13 decedents were assigned codes indicating multiple drug use or drug overdose. Among these 10 decedents, three worked in the health care and social assistance industry (offices of dentists, ambulatory health care services, and general medical and surgical hospitals) (Table 3).

Discussion

Among 55 deaths in young adults reported for 1999–2015 with ICD-10 code J62 assigned as either the underlying or a contributing cause of death, 13 were coded as subcategory J62.0, indicating exposure to talc dust, and in most of these cases, the underlying or contributing cause-of-death codes also indicated multiple drug use or drug overdose. These deaths likely represent nonoccupational pulmonary talcosis caused by illicit inhalation or intravenous administration of talc-contaminated drugs (*3*,*5*–*7*). Eight of the 13 pneumoconiosis deaths attributed to talc dust were associated with multiple drug use and drug overdose occurred during 2010–2015, and coincided with the expanding epidemic of drug overdose deaths in the United States (*8*).

[§§] CDC requested death certificates from 22 states for 48 young (aged 15–44 years) decedents reported for 1999–2013 who had ICD-10 code J62 (pneumoconiosis due to dust containing silica) assigned as the underlying or contributing cause of death. Among these, 47 (received from 21 states) were available for review. Seven additional deaths with code J62 assigned to cause of death among young adults were identified in the National Center for Health Statistics multiple cause-of-death data for 2014 and 2015 (when the new data became available).

[¶¶] Industry and occupation entries were coded using North American Industry Classification System and 2010 Standard Occupational Classification codes. https://www.cdc.gov/niosh/topics/coding/overview.html.

[***] Three (8%) decedents had the underlying cause of death coded as pneumoconiosis due to other dust containing silica (J62.8) and had one or more contributing causes of death coded as the following: T65.8 (toxic effect of other and unspecified substances); T80.9 (complications following infusion, transfusion, and therapeutic injection); Y56.3 (topical agents primarily affecting skin and mucous membrane and ophthalmological, otorhinolaryngological, and dental drugs); X49 (accidental poisoning by and exposure to other and unspecified chemicals and noxious substances). All three decedents received subcutaneous silicone injections.

[†††] Five (29%) decedents had the underlying cause of death coded as X42 (accidental poisoning by and exposure to narcotics and psychodysleptics [hallucinogens], not elsewhere classified; n = 1) or X44 (accidental poisoning by and exposure to other and unspecified drugs, medicaments and biological substances; n = 4). Eight (47%) decedents had one or more contributing causes of death coded as F19 (multiple drug use and use of other psychoactive substances, including F19.1 [harmful use] or F19.9 [unspecified mental and behavioral disorder]); T39 (poisoning by nonopioid analgesics, antipyretics and antirheumatics, including T39.8 [other nonopioid analgesics and antipyretics, not elsewhere classified]); T40 (poisoning by narcotics and psychodysleptics [hallucinogens], including T40.2 [other opioids], T40.3 [methadone], T40.6 [other and unspecified narcotics]); T42 (poisoning by antiepileptic, sedative-hypnotic and antiparkinsonism drugs), including T42.4 [benzodiazepines], T42.6 [other antiepileptic and sedative-hypnotic drugs]); or X40 (accidental poisoning by and exposure to nonopioid analgesics, antipyretics, and antirheumatics).

[§§§] Alabama, Arizona, Arkansas, California, Georgia, Illinois, Indiana, Kansas, Kentucky, Louisiana, Michigan, Minnesota, Mississippi, New Jersey, New Mexico, North Carolina, Ohio, Pennsylvania, Tennessee, Texas, and Washington.

TABLE 1. Pneumoconiosis deaths due to dust containing silica (ICD-10 category J62),* among persons aged 15–44 years (n = 55), by patient characteristics, year of death, and ICD-10 subcategory (J62.0 or J62.8†) — United States, 1999–2015

	J62.0		J62.8	
Characteristic	Underlying or contributing cause	Underlying cause	Underlying or contributing cause	Underlying cause
Total	**17**	**11**	**38**	**27**
Sex				
Male	9	7	36	26
Female	8	4	2	1
Race				
White	13	9	31	22
Black	3	2	6	4
Other	1	0	1	1
Ethnicity				
Hispanic	2	1	10	8
Non-Hispanic	15	10	28	19
Education				
≤8 grade	0	0	5	4
9–12	1	1	6	3
High school diploma	8	5	10	9
Some college	1	0	2	2
College degree	2	1	1	0
Unknown	5	4	14	9
Marital status				
Married	6	5	18	13
Single/Divorced	11	6	19	13
Unknown	0	0	1	1
Place of birth				
United States	17	11	27	18
Outside United States	0	0	11	9
Year of death				
1999	1	1	2	1
2000	0	0	5	5
2001	0	0	1	1
2002	1	1	4	3
2003	3	2	3	3
2004	0	0	3	0
2005	0	0	2	1
2006	2	1	4	2
2007	0	0	1	1
2008	0	0	2	2
2009	0	0	1	1
2010	0	0	1	0
2011	1	1	3	2
2012	0	0	0	0
2013	5	3	3	3
2014	1	0	1	0
2015	3	2	2	2
p-value§	0.21	0.41	0.09	0.23

Abbreviation: ICD-10 = *International Classification of Diseases, Tenth Revision.*

* Decedents with the ICD-10 code J62, pneumoconiosis due to dust containing silica category assigned to their underlying or contributing causes of death.

† ICD-10 code J62 is further divided into subcategories: J62.0 = pneumoconiosis due to talc dust; J62.8 = pneumoconiosis due to other dust containing silica.

§ For 1999–2015 time trend (time trends examined using a first-order autoregressive linear regression model).

Summary

What is already known about this topic?

Various preventable occupational pulmonary diseases are associated with exposure to respirable particles of crystalline silica and other silicate materials, one of which is talc (hydrous magnesium silicate). Detailed information on the circumstances surrounding deaths of silicosis decedents is needed to better target intervention and prevention measures.

What is added by this report?

During 1999–2015, among 55 decedents aged 15–44 years who had pneumoconiosis due to dust containing silica assigned as either the underlying or contributing cause of death, 38 (69%) were assigned pneumoconiosis due to other dust containing silica, and 17 (31%) were assigned pneumoconiosis due to talc dust. Decedents with pneumoconiosis due to other dust containing silica had manufacturing or construction industry frequently listed as the occupation on their death certificates; both industries are well known to be associated with exposures to silica-containing dust. Among 17 decedents with pneumoconiosis due to talc dust, 13 (76%) involved multiple drug use or drug overdose and none worked in talc exposure-associated jobs.

What are the implications for public health practice?

Among deaths in persons aged 15–44 years attributed to pneumoconiosis due to dust containing silica, nearly one third had pneumoconiosis due to talc dust. Most of these cases likely represent nonoccupational exposure to talc. Examining detailed information on causes of death, including external causes, along with industry and occupation of decedents is essential for identifying silicosis deaths associated with occupational exposures and reducing misclassification of silicosis mortality.

The remaining two thirds of silicosis deaths were coded as J62.8. Among silicosis deaths reported for 1999–2013, manufacturing or construction industries, both of which are known to be associated with exposures to silica-containing dust, were frequently listed on death certificates for these decedents. Three decedents had a history of subcutaneous silicone injections and likely were erroneously assigned code J62.8 as the underlying cause of death.

The findings in this report are subject to at least five limitations. First, no information on silica exposure intensity or duration is listed on death certificates. Silicosis-associated deaths in young adults should be considered sentinel cases, potentially resulting from high exposures that cause short latency to disease onset and rapid disease progression. Second, lifetime occupational histories of decedents were not collected, and the usual industry and occupation listed on death certificates might not accurately represent the industry or occupation where the hazardous silica exposure occurred. However, there is a generally good agreement of industry and occupation information on death certificates compared with

TABLE 2. Deaths due to other dust containing silica (ICD-10 subcategory J62.8),* among persons aged 15–44 years (n = 35), by year of death, age, industry and occupation,† and assignment of code J62.8 as the underlying cause — United States, 1999–2013

Year of death	Age (yrs)	Industry	Occupation	J62.8 code listed as the underlying cause
1999	40	Cut stone and stone product manufacturing	Crushing, grinding, and polishing machine setters, operators, and tenders	Yes
1999	43	Commercial and institutional building construction	Cement masons and concrete finishers	No
2000	32	Cut stone and stone product manufacturing	Etchers and engravers	Yes
2000	34	Unknown, blank, inadequate information	Sandblaster§	Yes
2000	41	Manufacturing	Production workers, all other	Yes
2000	41	Nonmetallic mineral mining and quarrying	Loading machine operators, underground mining	Yes
2000	43	Services to buildings and dwellings	Janitors and cleaners, except maids and housekeeping cleaners	Yes
2001	39	Construction of buildings	Brickmasons and blockmasons	Yes
2002	40	All other miscellaneous chemical product and preparation manufacturing	Industrial production managers	No
2002	41	Masonry contractors	Brickmasons and blockmasons	Yes
2002	43	Vitreous china, fine earthenware, and other pottery product manufacturing	Production workers, all other/Machine feeders	Yes
2002	44	Unknown, blank, inadequate information	Unknown, blank, inadequate information	Yes
2003	22	Construction	Construction laborers	Yes
2003	31	Retail trade	First-line supervisors of retail sales workers	Yes
2003	35	Unknown, blank, inadequate information	Unknown, blank, inadequate information	Yes
2004	41	Cut stone and stone product manufacturing	Laborers and freight, stock, and material movers, hand	No
2004	42	Nonmetallic mineral product manufacturing	Production workers, all other	No
2004	44	Ferrous metal foundries	Crushing, grinding, and polishing machine setters, operators, and tenders	No
2005	36	Tile and terrazzo contractors	Brickmasons and blockmasons	Yes
2005	41	Cement and concrete product manufacturing	Production workers, all other	No
2006	38	Unknown, blank, inadequate information	Unknown, blank, inadequate information	Yes
2006	41	Cut stone and stone product manufacturing	Crushing, grinding, and polishing machine setters, operators, and tenders	Yes
2006	42	Agencies, brokerages, and other insurance related activities	Social workers, all other	No
2006	43	Unknown, blank, inadequate information	Unknown, blank, inadequate information	No
2007	34	Electric power generation, transmission and distribution	Painting, coating, and decorating workers	Yes
2008	43	Cut stone and stone product manufacturing	Etchers and engravers	Yes
2008¶	34	Unknown, blank, inadequate information	Unknown, blank, inadequate information	Yes
2009	32	Janitorial services	Janitors and cleaners, except maids and housekeeping cleaners	Yes
2010	37	Nonpaid workers	Did not work	No
2011	34	General freight trucking	Heavy and tractor-trailer truck drivers	No
2011	35	Manufacturing	Production workers, all other	Yes
2011	44	Unknown, blank, inadequate information	Unknown, blank, inadequate information	Yes
2013	36	Construction	Industrial production managers	Yes
2013	41	Construction of buildings	Operating engineers and other construction equipment operators	Yes
2013	44	Other miscellaneous durable goods merchant wholesalers	Industrial truck and tractor operators	Yes

Abbreviation: ICD-10 = *International Classification of Diseases, Tenth Revision.*

* Assigned as either the underlying or contributing cause of death in the National Center for Health Statistics (NCHS) multiple cause-of-death data.

† Usual industry and occupation entries on death certificates for 34 (97%) of 35 pneumoconiosis deaths caused by other dust containing silica reported for 1999–2013 were available for review and coded using North American Industry Classification System and 2010 Standard Occupational Classification codes (https://wwwn.cdc.gov/niosh-nioccs/default.aspx).

§ Because industry was not known, this occupation could be coded as 1) cleaners of vehicles and equipment, 2) crushing, grinding, and polishing machine setters, or 3) operators, and tenders construction laborers.

¶ Death certificate was unavailable for review; information on year of death, age, and codes assigned as the underlying cause of death from the NCHS multiple cause-of-death data.

that from other sources (*9*). Third, industry and occupation information was only available for 40 (83%) and 42 (88%) decedents, respectively, who were included in reports during 1999–2013. Fourth, pneumoconiosis as a cause of death might have been misclassified or under- or overreported. Finally, increased recognition of drug-related deaths, improvements in testing, and reporting of deaths involving drug use might have contributed to the high frequency of reported multiple drug use and drug overdose among pneumoconiosis deaths due to talc. The continuing occurrence of pneumoconiosis deaths due to other dust containing silica indicates the need for maintaining measures to limit workplace exposure to respirable

TABLE 3. Deaths due to talc dust (ICD-10 subcategory J62.0)* among persons aged 15–44 years (n = 13), by year of death, age, industry and occupation,† and assignment of code indicating multiple drug use or drug overdose§ — United States, 1999–2013

Year of death	Age (yrs)	Industry	Occupation	Multiple drug use or drug overdose codes listed
1999	37	Nonpaid workers	Homemakers	Yes
2002	41	Voluntary health organizations	Secretaries and administrative assistants, except legal, medical, and executive	No
2003	37	Glass and glass product manufacturing	Glaziers	Yes
2003	40	Offices of dentists	Dental laboratory technicians	Yes
2003	41	Ambulatory health care services	Emergency medical technicians and paramedics	Yes
2006	19	Nonpaid workers	Students	No
2006	40	Nonpaid workers	Did not work	Yes
2011	41	Construction	Operating engineers and other construction equipment operators	Yes
2013	34	Computer systems design and related services	Computer occupations, all other	No
2013	36	Unknown, blank, inadequate information	Driver/sales workers	Yes
2013	36	All other specialty trade contractors	Construction managers	Yes
2013	43	Nonpaid workers	Did not work	Yes
2013	44	General medical and surgical hospitals	Registered nurses	Yes

Abbreviation: ICD-10 = *International Classification of Diseases, Tenth Revision*.

* Assigned as either the underlying or contributing cause of death in the National Center for Health Statistics (NCHS) multiple cause-of-death data.

† Usual industry and occupation entries on death certificates for all 13 (100%) pneumoconiosis deaths due to talc dust reported for 1999–2013 were available for review and coded using North American Industry Classification System and 2010 Standard Occupational Classification codes (https://wwwn.cdc.gov/niosh-nioccs/default.aspx).

§ ICD-10 codes indicating multiple drug use of drug overdose: X42 (accidental poisoning by and exposure to narcotics and psychodysleptics [hallucinogens], not elsewhere classified); X44 (accidental poisoning by and exposure to other and unspecified drugs, medicaments and biologic substances); F19 (multiple drug use and use of other psychoactive substances [F19.1 (harmful use)], [F19.9 (unspecified mental and behavioral disorder)]); T39 (poisoning by nonopioid analgesics, antipyretics and antirheumatics, including T39.8 [other nonopioid analgesics and antipyretics, not elsewhere classified]); T40 (poisoning by narcotics and psychodysleptics [hallucinogens] [T40.2 (other opioids), T40.3 (methadone), T40.6 (other and unspecified narcotics)]); T42 (poisoning by antiepileptic, sedative-hypnotic and antiparkinsonism drugs, including T42.4 [benzodiazepines]) and T42.6 [other antiepileptic and sedative-hypnotic drugs]); X40 (accidental poisoning by and exposure to nonopioid analgesics, antipyretics, and antirheumatics) listed as the underlying or a contributing cause in the NCHS multiple cause-of-death data.

crystalline silica. Primary prevention of pneumoconioses relies on elimination or effective control of exposures (https://www.cdc.gov/niosh/topics/hierarchy/). Effective silicosis prevention strategies for employers are available from the Occupational Safety and Health Administration (https://www.osha.gov/silica/) and CDC (https://www.cdc.gov/niosh/topics/silica). The occurrence of pneumoconiosis deaths due to talc associated with multiple drug use and drug overdose reinforces the need for a multifaceted, collaborative clinical, public health, public safety, and law enforcement approach to the drug overdose epidemic (*8*). Examining detailed information on causes of death, including external causes, along with industry and occupation of decedents, is essential for identifying silicosis deaths associated with occupational exposures and reducing misclassification of silicosis mortality.

Conflict of Interest

No conflicts of interest were reported.

Acknowledgments

Center for Health Statistics, Alabama Department of Public Health; Bureau of Vital Records, Arizona Department of Health; Vital Records Branch, Arkansas Department of Health; Center for Health Statistics and Informatics, California Department of Public Health; State Office of Vital Records, Georgia Department of Public Health; Division of Vital Records, Illinois Department of Public Health; Division of Vital Records, Indiana State Department of Health; Kansas Department of Health and Environment; Office of Vital Statistics, Kentucky Department for Public Health; Center for Records and Statistics, Louisiana Department of Health and Hospitals; Division for Vital Records and Health Statistics, Michigan Department of Health and Human Services; Office of Vital Records, Minnesota Department of Health; Office of Vital Records and Health Statistics, Mississippi State Department of Health; Office of Vital Statistics and Registry, New Jersey Department of Health; Bureau of Vital Records and Health Statistics, New Mexico Department of Health; Vital Records, North Carolina Department of Health and Human Services; Vital Statistics, Ohio Department of Health; Bureau of Health Statistics and Registries, Pennsylvania Department of Health; Bureau of Health Statistics and Registries, Pennsylvania Department of Health; Office of Healthcare Statistics, Tennessee Department of Health; Texas Department of State Health Services; Center for Health Statistics, Washington State Department of Health; Rose A. Rudd, Division of Unintentional Injury Prevention, National Center for Injury Prevention and Control, CDC; Carole A. Rehm, Respiratory Health Division, National Institute for Occupational Safety and Health, CDC.

[1]Respiratory Health Division, National Institute for Occupational Safety and Health, CDC.

Corresponding author: Jacek M. Mazurek, jmazurek1@cdc.gov, 304-285-5983.

References

1. Craighead JE, Kleinerman J, Abraham JZ, et al.; Silicosis and Silicate Disease Committee. Diseases associated with exposure to silica and nonfibrous silicate minerals. Arch Pathol Lab Med 1988;112:673–720.
2. National Institute for Occupational Safety and Health. NIOSH hazard review: health effects of occupational exposure to respirable crystalline silica. Atlanta, GA: US Department of Health and Human Services, CDC, National Institute for Occupational Safety and Health; 2002. DHHS (NIOSH) Publication No. 2002–129. https://www.cdc.gov/niosh/docs/2002-129/pdfs/2002-129.pdf
3. Hollinger MA. Pulmonary toxicity of inhaled and intravenous talc. Toxicol Lett 1990;52:121–7, discussion 117–9. https://doi.org/10.1016/0378-4274(90)90145-C
4. van Huisstede A, Noordhoek Hegt V, Otte-Holler I, Looijen-Salamon M, Rudolphus A. Talcosis due to abundant use of cosmetic talcum powder. Eur Respir Rev 2010;19:165–8. https://doi.org/10.1183/09059180.00001310
5. Scheel AH, Krause D, Haars H, Schmitz I, Junker K. Talcum induced pneumoconiosis following inhalation of adulterated marijuana, a case report. Diagn Pathol 2012;7:26. https://doi.org/10.1186/1746-1596-7-26
6. Baylor PA, Sobenes JR, Vallyathan V. Interstitial pulmonary fibrosis and progressive massive fibrosis related to smoking methamphetamine with talc as filler. Respir Care 2013;58:e53–5.
7. Siddiqui MF, Saleem S, Badireddi S. Pulmonary talcosis with intravenous drug abuse. Respir Care 2013;58:e126–8. https://doi.org/10.4187/respcare.02402
8. Rudd RA, Seth P, David F, Scholl L. Increases in drug and opioid-involved overdose deaths—United States, 2010–2015. MMWR Morb Mortal Wkly Rep 2016;65:1445–52. https://doi.org/10.15585/mmwr.mm655051e1
9. Steenland K, Beaumont J. The accuracy of occupation and industry data on death certificates. J Occup Med 1984;26:288–96.

Progress Toward Measles Elimination — Bangladesh, 2000–2016

Sudhir Khanal, MPH[1]; Rajendra Bohara, MD[2]; Stephen Chacko, MD[2]; Mohammad Sharifuzzaman, MSc[2]; Mohammad Shamsuzzaman, PhD[3]; James L. Goodson, MPH[4]; Alya Dabbagh, PhD[5]; Katrina Kretsinger, MD[5]; Deepak Dhongde, MSc[1]; Jayantha Liyanage, MD[1]; Sunil Bahl, MD[1]; Arun Thapa, MD[1]

In 2013, at the 66th session of the Regional Committee of the World Health Organization (WHO) South-East Asia Region (SEAR), a regional goal was established to eliminate measles and control rubella and congenital rubella syndrome* by 2020 (*1*). WHO-recommended measles elimination strategies in SEAR countries include 1) achieving and maintaining ≥95% coverage with 2 doses of measles-containing vaccine (MCV) in every district, delivered through the routine immunization program or through supplementary immunization activities (SIAs)†; 2) developing and sustaining a sensitive and timely measles case-based surveillance system that meets targets for recommended performance indicators; and 3) developing and maintaining an accredited measles laboratory network (*2*). In 2014, Bangladesh, one of 11 countries in SEAR, adopted a national goal for measles elimination by 2018 (*2*,*3*). This report describes progress and challenges toward measles elimination in Bangladesh during 2000–2016. Estimated coverage with the first MCV dose (MCV1) increased from 74% in 2000 to 94% in 2016. The second MCV dose (MCV2) was introduced in 2012, and MCV2 coverage increased from 35% in 2013 to 93% in 2016. During 2000–2016, approximately 108.9 million children received MCV during three nationwide SIAs conducted in phases. During 2000–2016, reported confirmed measles incidence decreased 82%, from 34.2 to 6.1 per million population. However, in 2016, 56% of districts did not meet the surveillance performance target of ≥2 discarded nonmeasles, nonrubella cases§ per 100,000 population. Additional measures that include increasing MCV1 and MCV2 coverage to ≥95% in all districts with additional strategies for hard-to-reach populations, increasing sensitivity of measles case-based surveillance, and ensuring timely transport of specimens to the national laboratory will help achieve measles elimination.

Immunization Activities

In Bangladesh, MCV1, administered at age 9 months, was introduced nationwide¶ in 1989 (*4*), and MCV2, administered at age 15 months, in 2012. Administrative vaccination coverage** data are reported each year from the 64 districts in Bangladesh to the National Immunization Programme, where they are aggregated and reported to WHO and UNICEF through the Joint Reporting Form (JRF). WHO and UNICEF use reported administrative coverage and available survey results to generate annual estimates of vaccination coverage through routine services (*5*). As noted previously, in Bangladesh estimated coverage for MCV1 and MCV2 increased significantly (Figure). A routine vaccination coverage survey†† implemented in 2015 estimated 92% national coverage for MCV1 and 81% for MCV2.

During 2005–2006, a nationwide SIA using monovalent measles vaccine reached 36.0 million children aged 9 months–10 years (1.5 million during the first phase in 2015 and 34.2 million during the second phase in 2006) with >100% administrative coverage (*6*). A nationwide SIA in 2010 using monovalent measles vaccine reached 18.1 million children aged 9–59 months and achieved 100% administrative coverage. In 2014, a nationwide SIA using measles-rubella vaccine reached 53.6 million children aged 9 months–14 years and achieved >100% administrative coverage; 63 of 64 districts reported administrative coverage >95%. The post-SIA coverage survey estimated 90% vaccination coverage during the SIA.

* Measles elimination is defined as the absence of endemic measles cases for a period of ≥12 months, in the presence of adequate surveillance. Rubella/Congenital Rubella Syndrome control is defined as 95% reduction in disease burden from 2008 levels.

† Supplementary Immunization Activities (SIAs) are immunization campaigns, typically carried out using two targeted age ranges. An initial, nationwide catch-up SIA targets all children aged 9 months–14 years, with the goal of eliminating measles susceptibility in the population. Periodic follow-up SIAs then target all children born since the last SIA and generally are conducted every 2–4 years and target children aged 9–59 months; the goal of a follow-up SIA is to eliminate any measles susceptibility that has accumulated in recent birth cohorts and to protect children who did not respond to the first dose of measles vaccine.

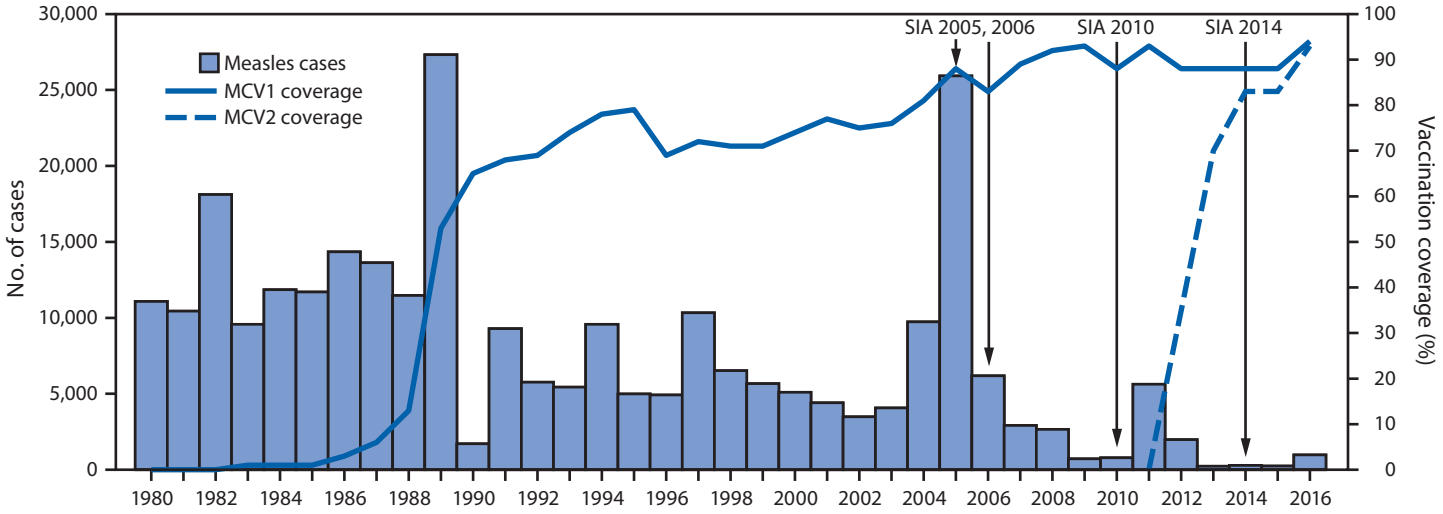
§ Suspected cases that have been investigated and discarded as a nonmeasles and nonrubella case using laboratory testing in a proficient laboratory or epidemiologic linkage to a laboratory-confirmed outbreak of another communicable disease that is neither measles nor rubella. It is estimated that the baseline rates of such discarded nonmeasles, nonrubella cases is two per 100,000 populations.

¶ The Expanded Programme on Immunization (EPI) began in 1979 as a pilot project and expanded in phases: the first phase (1985) included eight out of 490 thanas (a former administrative subdivision in urban areas of Bangladesh, equivalent to the current subdistrict [Upazila]); the second phase (1988) included 190 subdistricts. By the end of 1989 the EPI was expanded to the entire country.

** Administrative vaccination coverage is the number of vaccine doses administered divided by the estimated target population.

†† Bangladesh regularly conducts 30-cluster EPI immunization coverage surveys to evaluate routine and SIA coverage and is supported by partners. The last EPI coverage survey for which the results are available was conducted in 2015.

FIGURE. Aggregated measles cases,* estimated coverage† with the first and second dose of measles-containing vaccine (MCV1 and MCV2), and supplementary immunization activities (SIAs)§,¶,,†† — Vaccine Preventable Disease Surveillance Report, Bangladesh, 1980–2016**



* Laboratory-confirmed, epidemiologically linked and clinically compatible cases with fever; rash; and cough, coryza or conjunctivitis; reported as of Dec 2015 to World Health Organization (WHO) South-East Asia Region (2016 Joint Reporting Form [JRF]).

† 1990–2015 coverage data from WHO/UNICEF estimates of national immunization coverage as published in July 2016; for 2016, coverage data are from country official estimates (submitted through 2016 JRF).

§ National measles catch-up SIA targeted children aged 9 mos–10 yrs, implemented in two phases: 1) Sep 2005, targeting 1,481,321 children; 2) Feb 2006, targeting 34,199,590 children. Overall administrative coverage >100%.

¶ National measles follow-up SIA targeted children aged 9–59 mos, conducted Feb 14–28, 2010, targeting 18,136,066 children.

** National measles-rubella catch-up SIA targeted children aged 9 mos–14 yrs, conducted during Jan 25–Feb 13, 2014 targeting 51,745,231 children.

†† Specific SIA dates are as follows: Sep 3–22, 2005; Feb 25–Mar 16, 2006; Feb 14–28, 2010; Feb 13–Mar 25, 2014.

Surveillance Activities and Measles Incidence

In 2003, laboratory-supported case-based surveillance for suspected measles§§ was implemented in Bangladesh by adapting the existing acute flaccid paralysis surveillance system for polio detection; data are provided from 143 active and 625 passive surveillance sites in all 64 districts. In addition, aggregated measles cases¶¶ are reported by all health facilities through the National Health Management Information System and have been reported annually through the JRF since 2000. The difference in number of cases reported annually by these two parallel systems has decreased since 2013 (Table 1). Measles virus genotyping began in Bangladesh in 2014.

During 2013–2016, years for which data on key surveillance performance indicators*** (*7*) were available, the discarded nonmeasles, nonrubella rate increased nationally from 1.1 to 1.9 per 100,000 population; percentage of districts reporting at least two discarded nonmeasles, nonrubella cases per 100,000 population increased from 19% to 44%; percentage of suspected cases with adequate investigation initiated within 48 hours of notification increased from 87% to 94%; and the percentage of serology results reported by the laboratory within 4 days of specimen receipt increased from 82% to 94% (Table 2).

During 2000–2016, incidence of measles cases reported through the JRF decreased 84%, from 40.0 to 6.0 per million

§§ In Bangladesh, a suspected measles case is defined as an illness in any person a clinician suspects of having measles infection, or in any person with fever and maculopapular rash and cough, coryza, or conjunctivitis.

¶¶ National measles case data are reported to WHO South-East Asia Region Office) through the World Health Organization/UNICEF Joint Reporting Form (JRF) aggregate reporting annually. Bangladesh uses administrative data reported through the national Health Management Information system (HMIS) to report in the JRF. The HMIS receives aggregated data from all the health facilities in the country, including private and public clinics and hospitals.

*** Key surveillance performance indicators include 1) ≥2 discarded nonmeasles, nonrubella cases per 100,000 population at the national level per year; 2) ≥2 discarded nonmeasles, nonrubella cases per 100,000 per year in ≥80% of subnational administrative units; 3) an adequate investigation conducted within 48 hours of notification for ≥80% of suspected measles cases; 4) adequate specimens for detecting acute measles and rubella infection collected and tested in a proficient laboratory from ≥80% of suspected cases; 5) receipt of ≥80% of specimens at the laboratory within 5 days of collection; 6) laboratory reporting of ≥80% of serology results within 4 days of specimen receipt; and 7) on-time reporting of measles and rubella data to the national level by ≥80% of surveillance units.

TABLE 1. Measles incidence,* number of reported measles cases by case classification, age group, and vaccination status — Vaccine Preventable Disease Surveillance Report, Bangladesh, 2001–2016

	WHO/UNICEF JRF aggregate reporting†		Measles case-based reporting§													
			No. of measles case classification					Age of confirmed measles cases, No. (%)					MCV doses received by confirmed measles cases, No. (%)			
Year	No. of reported measles cases	Incidence (cases/ million population)	Suspected¶	Confirmed**	Laboratory-confirmed	Epi-linked	Clinically compatible	<9 mos	9 mos–4 yrs	5–9 yrs	10–14 yrs	≥15 yrs	≥2	1	Zero	Unknown
2000	5,098	40.0	—	—	—	—	—	—	—	—	—	—	—	—	—	—
2001	4,414	34.2	—	—	—	—	—	—	—	—	—	—	—	—	—	—
2002	3,484	26.6	—	—	—	—	—	—	—	—	—	—	—	—	—	—
2003	4,067	30.6	721	640	56	584	—	46 (7.2)	303 (47.3)	212 (33.1)	59 (9.2)	20 (3.1)	—	169 (26.4)	436 (68.1)	35 (5.5)
2004	9,743	71.3	6,612	5,517	318	5,199	—	524 (9.5)	2662 (48.3)	1653 (30.0)	448 (8.1)	230 (4.2)	—	2,189 (39.7)	2,554 (46.3)	774 (14.0)
2005	25,934	186.4	27,539	14,877	739	14,138	—	1358 (9.1)	5670 (38.1)	4889 (32.9)	1763 (11.9)	1197 (8.1)	5 (0.0)	8,103 (54.5)	4,973 (33.4)	1,796 (12.1)
2006	6,192	43.7	7,820	3,058	169	2,889	—	306 (10.0)	1069 (34.9)	1076 (35.2)	392 (12.8)	215 (7.0)	1,085 (35.5)	1,146 (37.5)	773 (25.3)	54 (1.8)
2007	2,924	20.3	14,482	6	6	—	—	—	3 (50.0)	2 (33.3)	1 (16.7)	—	2 (33.3)	4 (66.7)	—	—
2008	2,660	18.1	8,308	139	16	123	5	12 (8.6)	77 (55.4)	33 (23.7)	7 (5.0)	10 (7.2)	2 (1.4)	68 (48.9)	69 (49.6)	—
2009	718	4.9	14,896	78	35	43	212	8 (10.3)	47 (60.3)	9 (11.5)	9 (11.5)	5 (6.4)	1 (1.3)	27 (34.6)	50 (64.1)	—
2010	788	5.3	14,745	66	51	15	440	7 (10.6)	24 (36.4)	19 (28.8)	3 (4.5)	13 (19.7)	27 (40.9)	17 (25.8)	22 (33.3)	—
2011	5,625	37.4	14,696	5,329	1,930	3,399	741	359 (6.7)	1820 (34.2)	1426 (26.8)	460 (8.6)	1264 (23.7)	1427 (26.8)	1,559 (29.3)	2343 (43.9)	—
2012	1,986	13.1	8,291	1,793	715	1,078	599	211 (11.8)	551 (30.7)	361 (20.1)	176 (9.8)	494 (27.6)	381 (21.2)	601 (33.5)	656 (36.6)	155 (8.6)
2013	237	1.5	5,229	200	77	123	325	32 (16.0)	70 (35.0)	45 (22.5)	14 (7.0)	39 (19.5)	28 (14.0)	88 (44.0)	63 (31.5)	21 (10.5)
2014	289	1.9	3,041	288	145	143	175	41 (14.2)	145 (50.3)	58 (20.1)	15 (5.2)	29 (10.1)	56 (19.4)	100 (34.7)	131 (45.5)	1 (0.3)
2015	240	1.5	3,435	250	158	92	64	32 (12.8)	125 (50.0)	42 (16.8)	22 (8.8)	29 (11.6)	87 (34.8)	81 (32.4)	75 (30.0)	7 (2.8)
2016	972	6.0	4,291	972	618	354	81	149 (15.3)	543 (55.9)	161 (16.6)	27 (2.8)	92 (9.5)	123 (12.7)	262 (27.0)	559 (57.5)	28 (2.9)

Abbreviations: epi = epidemiologically; JRF = Joint Reporting Form; MCV = measles-containing vaccine; WHO = World Health Organization.

* Measles incidence calculated based on reported confirmed measles cases and population by countries through WHO/UNICEF JRF.

† National measles case data as reported to WHO South-East Asia Region Office (SEARO) as of December 2015 through the WHO/UNICEF JRF. Bangladesh uses administrative data reported through the national Health Management Information system (HMIS) to report in the JRF. The HMIS receives aggregated data from all the health facilities in the country, including private and public clinics and hospitals.

§ Data from case-based measles surveillance through the Vaccine Preventable Diseases surveillance network reported to WHO SEARO as of December 2016.

¶ An illness in any person a clinician suspects of having measles infection or in any person with fever and maculopapular rash and cough, coryza, or conjunctivitis.

** Includes laboratory-confirmed and epidemiologically linked cases. An epidemiologically linked case is one that meets the clinical case definition and is linked epidemiologically to a laboratory-confirmed or another epidemiologically confirmed case.

(Table 1). After implementation of nationwide measles SIAs in 2005–2006 and 2010, the number of confirmed measles cases decreased from 14,877 (2005) to 66 (2010), but increased to 5,329 in 2011, (Table 1). Following MCV2 introduction in 2012 and the nationwide measles-rubella catch-up SIA in 2014, confirmed measles cases declined from 1,793 in 2012 to 250 in 2015. In 2016, a program assessment was conducted using the WHO Programmatic Risk Assessment Tool for measles (*8*), and found eight districts at very high risk for measles transmission, 13 at high risk, 24 at medium risk, and 19 at low risk. In 2016, the number of confirmed measles cases increased, with 21 outbreaks in Sylhet Division and Cox's Bazar District in the southeastern part of the country. Overall, 972 confirmed cases were reported. Outbreak response immunization activities targeting 100,000 children aged 9–59 months were conducted in two districts in December 2016. An outbreak investigation in affected areas revealed persistent low coverage with MCV1 and MCV2 through routine immunization (RI) and during the 2014 SIA. In addition, procedures for isolating measles cases were not followed, and nosocomial transmission of measles virus occurred in multiple health care facilities.

Genotype results were available for two cases each in 2014 and 2015; all were genotype B3. No results were available for 2016.

Discussion

During 2000–2016, after increasing MCV1 and MCV2 coverage and three SIAs, confirmed measles incidence in Bangladesh decreased 84% (*9*). In 2016, however, an outbreak occurred, and transmission has continued into 2017, revealing gaps in both RI and SIA coverage. The national vaccination

TABLE 2. National measles case-based surveillance performance indicator targets and progress toward meeting them — Vaccine Preventable Disease Surveillance Report, Bangladesh, 2013–2016

Indicator	Target	Year			
		2013	2014	2015	2016
Reporting rate of discarded nonmeasles cases at the national level per year	≥2	1.1	1.4	1.8	1.9
Percentage of subnational administrative units (districts) reporting at least two discarded nonmeasles cases per 100,000 population per year	≥80	19	18	36	44
Percentage of suspected measles* cases adequately investigated† within 48 hours of notification	≥80	87	90	92	94
Percentage of suspected cases with adequate specimens§ tested for measles in a proficient laboratory¶	≥80	84	90	98	99
Percentage of results reported by the laboratory within 4 days of specimen receipt**	≥80	82	99	87	94
Percentage of weekly surveillance units reporting to the national level on time	≥80	85	89	91	97

* An illness in any person a clinician suspects of having measles infection, or in any person with fever and maculopapular rash and cough, coryza, or conjunctivitis.
† Includes collection of all the following data elements about each suspected case of measles or rubella: patient name or identifiers, place of residence, place of infection (at least to district level), age (or date of birth), sex, date of rash onset, date of specimen collection, measles-rubella vaccination status, date of last measles-rubella or measles-mumps-rubella vaccination, date of notification, date of investigation, and travel history.
§ A blood specimen collected within 28 days of the onset of rash.
¶ A World Health Organization (WHO)-accredited laboratory that has an established quality assurance program or one with oversight by a WHO-accredited laboratory.
** Changed to 4 days from 7 day in 2015.

coverage survey conducted in 2015 found the following most common reasons for a child being unvaccinated or partially vaccinated: 1) caretakers were too busy with other priorities, 2) caretakers did not remember to bring the child for vaccination, and 3) lack of information about when to bring the child for vaccination. These findings indicated the need for intensified social mobilization activities to strengthen RI, and a communication campaign is planned for 2017–2018.

In 2003, laboratory-supported measles case-based surveillance was implemented in Bangladesh by adapting the existing acute flaccid paralysis surveillance system for polio detection. Measles case-based surveillance indicators reflected underreporting and low sensitivity of the suspected measles case definition. Case-based surveillance sensitivity could be increased by expanding case-based surveillance reporting sites from acute flaccid paralysis reporting units to all health facilities in the country and by using the broad definition of "fever and maculopapular rash" (*10*). In addition, specimens for genotyping need to be collected from more chains of transmission to better track transmission pathways and identify outbreak sources.

Summary

What is already known about this topic?

Before 2000, estimated coverage with the routine first dose of measles-containing vaccine (MCV1) in Bangladesh was ≤75% nationally; no districts had ≥95% MCV1 coverage, and measles was a major cause of child death.

What is added by this report?

In 2014, a goal was set for measles elimination in Bangladesh by 2018. During 2000–2016, estimated MCV1 coverage increased from 74% to 94%. The routine second dose of measles-containing vaccine (MCV2) was introduced nationwide in 2012, and MCV2 coverage increased from 35% in 2013 to 93% in 2016. Approximately 108.9 million children were vaccinated during supplemental immunization activities (SIAs) in 2005–2006, 2010 and 2014. Annual reported measles incidence decreased 84%, from 40.0 to 6.0 per million population. Challenges to achieving elimination include low coverage with MCV1 and MCV2 and suboptimal performance of the measles case-based surveillance system.

What are the implications for public health practice?

Achieving ≥95% 2-dose measles vaccination coverage in all districts will require strengthening routine immunization services through innovative approaches and implementation of periodic high-quality SIAs. Improved measles case-based surveillance performance and increased surveillance sensitivity are needed for rapid case detection and outbreak preparedness and response.

Infection prevention and control practices and isolation of measles cases in health care facilities needs strengthening to prevent nosocomial infection.

The findings in this report are subject to at least two limitations. First, administrative coverage resulted in overestimates of vaccination coverage through erroneous inclusion of SIA doses or doses administered to children outside the target age group, inaccurate estimates of the target population size, and inaccurate reports of the number of doses delivered. Second, surveillance data might substantially underestimate disease incidence, because not all patients seek care, and not all patients who seek care are reported.

The endorsement of the measles elimination goal in the comprehensive 2014–2018 plan for immunization in Bangladesh provides an opportunity to achieve and maintain measles elimination, by strengthening routine immunization services through innovative approaches, conducting high quality nationwide follow-up SIAs, enhancing case-based surveillance, and identifying opportunities for synergies with other public health programs. In 2015, the National Verification Committee for Measles Elimination was established, according to the global framework for the verification of progress toward measles elimination (*7*). As measles elimination nears, it will be necessary to conduct annual risk assessments and develop risk

mitigation plans, conduct an immediate nationwide follow-up measles-rubella SIA to address current immunity gap among children aged 9–59 months, and build capacity for epidemiologic investigations and outbreak preparedness and response to rapidly identify and contain outbreaks.

Conflict of Interest

No conflicts of interest were reported.

[1]Immunization and Vaccine Development, World Health Organization (WHO) South-East Asia Regional Office, Delhi, India; [2]Immunization Preventable Disease, Bangladesh Country Office, WHO; [3] Epidemiology and Surveillance, Directorate General of Health Services, Ministry of Health, Bangladesh; [4]Global Immunization Division, Center for Global Health, CDC; [5]Immunization and Vaccine Development, WHO Headquarters, Geneva, Switzerland.

Corresponding author: Sudhir Khanal, khanals@who.int.

References

1. World Health Organization Regional Office of South-East Asia. Resolution of the WHO Regional Committee for South Asia on measles elimination and rubella/congenital rubella syndrome control (SEA/RC66/R5). New Delhi, India: World Health Organization, Regional Office for South East Asia; 2013. http://www.searo.who.int/about/governing_bodies/regional_committee/rc66-r5.pdf?ua=1
2. World Health Organization Regional Office of South-East Asia. Strategic plan for measles elimination and rubella and congenital rubella syndrome control in the South-East Asia Region—2014–2020. New Delhi, India: World Health Organization, Regional Office for South East Asia; 2014. http://www.searo.who.int/entity/immunization/documents/sear_mr_strategic_plan_2014_2020.pdf_
3. Government of People's Republic of Bangladesh, Ministry of Health and Family Welfare. Comprehensive multi-year plan of the national immunization program of Bangladesh 2014–2018. Dhaka, Bangladesh: Government of People's Republic of Bangladesh, Ministry of Health and Family Welfare; 2014.
4. Talukdar LR, Basu RN, Shareef M, Khan MR, Nasiruddin NH. The near miracle: how immunization services are delivered in Bangladesh. In: Huq M, ed. Near miracle in Bangladesh. Dhaka, Bangladesh: Dhaka University Press Limited; 1991:57–74.
5. World Health Organization; UNICEF. WHO/UNICEF estimates of national immunization coverage (WUENIC). Geneva, Switzerland: World Health Organization; New York, NY: UNICEF; 2015. http://www.who.int/immunization/monitoring_surveillance/data/en/
6. World Health Organization. Supplementary immunization activities. Geneva, Switzerland: World Health Organization; 2017. http://www.who.int/immunization/monitoring_surveillance/data/Summary_Measles_SIA_Jan2000_Dec2017.xls?ua=1
7. World Health Organization Regional Office of South-East Asia. Guidelines on verification of measles elimination and rubella/congenital rubella syndrome control in the WHO South-East Asia Region. New Delhi, India: World Health Organization Regional Office for South-East Asia; 2016. http://www.searo.who.int/entity/immunization/documents/mr_guidelines.pdf
8. Lam E, Schluter WW, Masresha BG, et al. Development of a district-level programmatic assessment tool for risk of measles virus transmission. Risk Anal 2015. Epub May 15, 2015.
9. World Health Organization. Measles vaccines: WHO position paper. Wkly Epidemiol Rec 2009;84:349–60.
10. Thapa A, Khanal S, Sharapov U, et al. Progress toward measles elimination—South-East Asia Region, 2003–2013. MMWR Morb Mortal Wkly Rep 2015;64:613–7.

Notes from the Field

Cluster of Acute Flaccid Myelitis in Five Pediatric Patients — Maricopa County, Arizona, 2016

Sally A. Iverson, DVM[1,2,3]; Scott Ostdiek, MD[4]; Siru Prasai, MD[2]; David M. Engelthaler, PhD[5]; Melissa Kretschmer, MA[2]; Nicole Fowle[2]; Harlori K. Tokhie, MD[4]; Janell Routh, MD[6]; James Sejvar, MD[7]; Tracy Ayers, PhD[6]; Jolene Bowers, PhD[5]; Shane Brady, MPH[3]; Shannon Rogers, MS[6]; W. Allan Nix, PhD[6]; Ken Komatsu, MPH[3]; Rebecca Sunenshine, MD[2,8]; AFM Investigation Team

In 2016, CDC saw an increase in cases of acute flaccid myelitis (AFM); 144 persons in 37 states and the District of Columbia were confirmed to have AFM. After investigations in California (*1*) and Colorado (*2*) in 2014, CDC characterized AFM as an acute flaccid paralysis (AFP) distinguishable by magnetic resonance imaging (MRI) abnormalities of the gray matter of the anterior and posterior spinal cord segments, involving one or more spinal segments (*3*). Although certain viruses (e.g., nonpoliovirus enteroviruses, adenoviruses, and West Nile virus) can cause rare cases of AFP, and findings from the 2014 outbreak investigations indicated that enterovirus D68 (EV-D68) was temporally associated with AFM, no viral etiology for AFM has been definitively established (*3*). In September 2016, an acute care hospital in Arizona notified the Maricopa County Department of Public Health (MCDPH) of a suspected case of AFM and subsequent cluster of 11 children who were evaluated with similar neurologic deficits; differential diagnoses included transverse myelitis and AFM. The Maricopa County Department of Public Health, in cooperation with the Arizona Department of Health Services, CDC, the Translational Genomics Research Institute (TGen, Flagstaff, Arizona), and the acute care hospital, initiated an investigation to confirm AFM cases and identify an etiology.

The 2015 Council of State and Territorial Epidemiologists and CDC case definition for probable AFM requires acute onset of flaccid limb weakness and cerebrospinal fluid (CSF) pleocytosis (CSF white blood cell [WBC] count >5/mm^3 when corrected for red blood cells). A confirmed case must have an MRI demonstrating lesions restricted primarily to the gray matter of the spinal cord, in addition to acute onset of flaccid limb weakness (*4*). Based on medical chart abstraction and review of the MRI images, a CDC neurology subject matter expert verified four confirmed cases of AFM and one probable case. Among the six patients whose cases did not meet the AFM confirmed or probable case definition, two had focal limb weakness and pleocytosis (CSF WBC = 7/mm^3 and 22/mm^3, respectively), but MRI results indicated alternative etiologies (acute disseminated encephalomyelitis and neuromyelitis optica, respectively). The case that met the probable case definition had pleocytosis (CSF WBC = 7/mm^3), but MRI findings were inconsistent with AFM, and no other plausible diagnosis was identified.

Onset dates for the four confirmed cases occurred during August 19–September 15, 2016. All four patients had preceding respiratory (three patients) or gastrointestinal illness (one patient), with onset dates for those illnesses occurring during August 14–September 13. Their illness began a median of 2 days (range = 2–5 days) before onset of focal limb weakness; three patients experienced tactile or measured fever preceding onset of neurologic symptoms (Table). Among patients with confirmed cases, focal limb weakness was present in a single limb (one case), three limbs (two cases), and four limbs (one case). Two patients with confirmed cases and one patient with a probable case had a prior medical history of asthma, and a third patient with a confirmed case reported a family history of asthma. The investigation team conducted hypothesis-generating interviews with all confirmed AFM patients and their proxies. Three of the four patients with confirmed cases were residents of Maricopa County, and no epidemiologic links were detected among the four patients. None of the patients had traveled to an area with ongoing Zika virus transmission in the month prior to symptom onset.

CSF was collected from all four patients with confirmed AFM. Median CSF WBC count was 133/mm^3 (range = 50–207), and initial viral testing at the hospital included CSF reverse transcription–polymerase chain reaction (RT-PCR) assays for enterovirus (three patients) and West Nile virus (WNV) (two patients), polymerase chain reaction (PCR) assay for herpes simplex virus (two patients), and enzyme immunoassay to detect immunoglobulin M (IgM) or immunoglobulin G (IgG) for WNV (three patients). All results were negative. All four CSF specimens were negative on TGen amplicon sequencing assay (*5*,*6*) using primers based on the 2014 circulating EV-D68 virus. Results of microbiome analysis by metagenomic sequencing of RNA and 16S rRNA gene sequencing of DNA extracted from CSF revealed bacterial sequencing dominated by *Propionibacterium*, which is a normal component of the skin flora and most likely represents a contaminant (*7*). Serum collected from one patient at initial evaluation was negative for WNV IgM and IgG on a hospital immunoassay; serum collected from the same patient 47 days after onset of focal limb weakness and from two additional patients (19 and 26 days after onset of focal limb weakness) were negative for WNV

TABLE. Characteristics of five pediatric patients with acute flaccid myelitis* — Maricopa County, Arizona, October 2016

Characteristic	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Case status	Confirmed	Confirmed	Confirmed	Confirmed	Probable
Age at onset (yrs)	3.5	10	4	9	12
Sex	Boy	Girl	Girl	Girl	Girl
Onset of focal limb weakness	August 23, 2016	August 19, 2016	September 15, 2016	September 8, 2016	August 27, 2016
Onset of preceding respiratory or gastrointestinal illness	August 21, 2016 (respiratory)	August 14, 2016 (respiratory)	September 13, 2016 (respiratory)	September 6, 2016 (gastrointestinal)	August 17, 2016 (respiratory)
Presence of fever (tactile† or measured)	Yes	No	Yes	Yes	No
Limbs affected (region)	1 (LUE)	4	3 (BUE, RLE)	4	1 (LUE)
Cranial nerve features and timing	None	Facial droop subsequent to onset of limb weakness	Facial droop subsequent to onset of limb weakness	Facial droop before onset of limb weakness	Diplopia concurrent with limb weakness
Patient and family history of asthma	Asthma and family history of asthma	Asthma	None	Family history of asthma	Mild asthma, seasonal allergies, food allergies, eczema
Corticosteroid history	Maintenance inhaled fluticasone; oral budesonide for asthma exacerbation August 15–19	Maintenance inhaled fluticasone; oral prednisolone for asthma exacerbation beginning August 14	None	Oral prednisolone for treatment of Bell's palsy beginning September 5	Maintenance inhaled fluticasone
Magnetic resonance imaging (MRI) findings	T2 signal abnormalities in anterior and posterior columns of the central gray cervical cord	T2 signal abnormality with anterior and posterior involvement, contiguous through multiple levels of the cord	T2 signal abnormality in the anterior horn of the central gray cord	Anterior horn signal abnormality extending four cervical levels	Normal MRI result
Cerebrospinal fluid/white blood cell/mm^3	50	150	207	115	7
Nasopharyngeal swab polymerase chain reaction results from TGen	Positive for EV-D68	Positive for EV-D68	Positive for EV-D68	Unavailable	Unavailable
Stool specimen testing results	Negative enterovirus/parechovirus by RT-PCR	Negative viral culture and enterovirus/parechovirus by RT-PCR	Negative viral culture and enterovirus/parechovirus by RT-PCR	Positive for coxsackievirus A10 by Sanger sequencing of the VP1 region	Unavailable

Abbreviations: BUE = bilateral upper extremities; EV = enterovirus; LUE = left upper extremity; RLE = right lower extremity; RT-PCR = reverse transcription–polymerase chain reaction; T2 = T2 weighted image; TGen = Translational Genomics Research Institute (Flagstaff, Arizona); VP1 = viral protein 1.
* Four with confirmed cases and one with a probable case.
† Felt warm to the touch, according to parent.

IgM and St. Louis encephalitis IgM at the Arizona State Public Health Laboratory.

Three of the four patients had nasopharyngeal (NP) swabs available from initial evaluation that were forwarded to CDC; one specimen was positive for enterovirus/rhinovirus on a panviral respiratory PCR panel at the admitting hospital laboratory and for EV-D68 at CDC. RNA extracted from NP swabs from all three patients was positive by the TGen amplicon sequencing test for EV-D68 (GenBank Bioproject); an NP specimen from a patient who did not meet the AFM confirmed or probable case definitions also was positive for EV-D68 by the same assay.

Stool specimens were collected from two patients at the time of initial evaluation; vital cultures of these specimens were negative on viral. One available specimen and three additional specimens, collected 28, 47, and 63 days after onset of focal limb weakness, were sent to CDC for four enterovirus/parechovirus RT-PCR assays. A stool specimen, collected at day 28 from the patient who did not have an NP swab available, was positive for coxsackievirus A10.

This cluster of AFM at one children's acute care hospital is the largest cluster identified to date in Arizona and is part of a nationally identified increase in AFM cases. Although no statewide surveillance system specific to AFM is available, this

cluster was detected by physician reporting, highlighting the need for physicians to remain vigilant for this emerging disease and to report cases that fit the AFM case definition to their local health department. Metagenomic analyses identified EV-D68 in NP swabs from the three patients for whom specimens were available, along with a specimen from a patient who did not meet the AFM case definition; therefore, no single etiology or risk factor was associated with only confirmed cases.

Patient and family history of asthma was the most common comorbidity reported among confirmed AFM cases and should be considered in future case investigations. Expanded analysis of infectious, postinfectious, and noninfectious etiologies might provide further insight into the mechanism of AFM.

Acknowledgments

Kathryn Putman; Jennifer Adair, Bernny Apodaca, Phoenix Children's Hospital, Neurology Department; Phoenix Children's Hospital Infection, Department of Infection Prevention and Control; Arizona State Public Health Laboratory, Virology Section.

AFM Investigation Team

Tammy Sylvester, Maricopa County Department of Public Health, Arizona; Veronica Harrison, Translational Genomics Research Institute, Flagstaff, Arizona; Jennifer Heim, MD, Phoenix Children's Hospital, Arizona; Susan Robinson, MPH, Arizona Department of Health Services; Gholamabbas A. Ostovar, MD, Maricopa Integrated Health System, Arizona; Kathryn Fitzpatrick, Arizona State Public Health Laboratory, Virology Section.

Conflict of Interest

David Engelthaler and Jolene Bowers have a provisional patent application in progress for a real-time polymerase chain reaction assay for the detection of Enterovirus D68 in complex specimens. No other conflicts of interest were reported.

[1]Epidemic Intelligence Service, CDC; [2]Maricopa County Department of Public Health, Phoenix, Arizona; [3]Arizona Department of Health Services; [4]Phoenix Children's Hospital, Arizona; [5]Translational Genomics Research Institute, Flagstaff, Arizona; [6]Division of Viral Diseases, National Center for Immunization and Respiratory Diseases, CDC; [7]Division of High-Consequence Pathogens and Pathology, National Center for Emerging and Zoonotic Infectious Diseases, CDC; [8]Career Epidemiology Field Officer Program, CDC; [9]Maricopa Integrated Health System, Phoenix, Arizona.

Corresponding author: Sally A. Iverson, lyu3@cdc.gov, 602-359-0424.

References

1. CDC. Notes from the field: acute flaccid myelitis among persons aged ≤21 years—United States, August 1–November 13, 2014. MMWR Morb Mortal Wkly Rep 2015;63:1243–4.
2. Pastula DM, Aliabadi N, Haynes AK, et al. Acute neurologic illness of unknown etiology in children—Colorado, August–September 2014. MMWR Morb Mortal Wkly Rep 2014;63:901–2.
3. Sejvar JJ, Lopez AS, Cortese MM, et al. Acute flaccid myelitis in the United States, August–December 2014: results of nationwide surveillance. Clin Infect Dis 2016;63:737–45. https://doi.org/10.1093/cid/ciw372
4. Council of State and Territorial Epidemiologists (CSTE). CSTE position statement: standardized case definition for acute flaccid myelitis. Atlanta, GA: Council of State and Territorial Epidemiologists; 2015. http://c.ymcdn.com/sites/www.cste.org/resource/resmgr/2015PS/2015PSFinal/15-ID-01.pdf
5. Colman RE, Anderson J, Lemmer D, et al. Rapid drug susceptibility testing of drug-resistant *Mycobacterium tuberculosis* isolates directly from clinical samples by use of amplicon sequencing: a proof-of-concept study. J Clin Microbiol 2016;54:2058–67. https://doi.org/10.1128/JCM.00535-16
6. Bowers JR, Lemmer D, Sahl JW, et al. KlebSeq, a diagnostic tool for surveillance, detection, and monitoring of *Klebsiella pneumoniae*. J Clin Microbiol 2016;54:2582–96. https://doi.org/10.1128/JCM.00927-16
7. Grice EA, Segre JA. The skin microbiome. Nat Rev Microbiol 2011;9:244–53.

Notes from the Field

Cronobacter sakazakii Infection Associated with Feeding Extrinsically Contaminated Expressed Human Milk to a Premature Infant — Pennsylvania, 2016

Anna Bowen, MD[1]; Harold C. Wiesenfeld, MD [2,3]; Jennifer L. Kloesz, MD[2,3]; A. William Pasculle, ScD[4]; Andrew J. Nowalk, MD, PhD[5]; LuAnn Brink, PhD[6]; Elisa Elliot, PhD[7]; Haley Martin[1]; Cheryl L. Tarr, PhD[1]

In April 2016, a female infant was born via Cesarean delivery at 29 estimated gestational weeks and had a birthweight of 3 pounds (1,405 grams). Her clinical course in the neonatal intensive care unit was unremarkable until she developed signs of sepsis at age 21 days. Cultures of blood and cerebrospinal fluid yielded *Cronobacter sakazakii*, a gram-negative pathogenic bacillus. Despite treatment with ampicillin and cefepime, she developed seizures; brain imaging revealed liquefaction necrosis of the entire left cerebral hemisphere and right frontal lobe. The infant developed spastic cerebral palsy and global developmental delay and required a ventriculoperitoneal shunt and a gastrostomy feeding tube.

The infant had been fed pasteurized donor human milk and expressed maternal milk (EMM) during the first week after birth; thereafter, she received EMM mixed with a commercial liquid human milk fortifier. Maternal milk was expressed using a dedicated bedside hospital breast pump and the mother's personal breast pump throughout the infant's hospitalization. The infant did not receive powdered infant formula products.

Clinicians and microbiologists from the hospital, Allegheny County Health Department, the Food and Drug Administration, and CDC investigated the source of the infection. Items and materials tested included the personal and hospital breast pump kits; samples of frozen EMM from the personal breast pump; hand-expressed maternal milk; lanolin used to treat the mother's breasts; human milk fortifier, caffeine citrate, vitamin D, and iron supplements from lots given to the infant; tap water, faucet and sink surfaces from the hospital bedside and home kitchen; two wash basins from the home kitchen; and maternal stool samples. *C. sakazakii* was cultured from the valves of the personal breast pump kit, 11 frozen EMM samples collected using that pump kit during 7 separate days before illness onset, and the drain of the kitchen sink at the mother's home. Cultures of the personal breast pump kit and the 11 frozen EMM samples each yielded 2–5 additional gram-negative bacteria; other items did not yield pathogens. Except for the EMM isolate with the earliest collection date, pulsed-field gel electrophoresis patterns of all EMM and clinical *C. sakazakii* isolates were indistinguishable.

The mother reported typically soaking the collection kit from her personal breast pump in soapy water in a wash basin for ≤5 hours without scrubbing or sanitizing. She then rinsed, air-dried, and stored the kit in a plastic zip-top bag until the next use. The collection kit from the hospital breast pump was washed immediately and thoroughly air-dried at the bedside. The mother did not report symptoms or signs of mastitis.

C. sakazakii can cause sepsis and severe meningitis, particularly among infants (*1*). *Cronobacter* infections have been traced to contaminated powdered infant formula, and only once has a source unrelated to powdered infant formula been reported to be associated with an infant infection (*1*,*2*). However, *Cronobacter* can be found in other food products and the environment, and some infected infants did not consume powdered infant formula (*1*,*3*). This case of *C. sakazakii* infection caused by consumption of extrinsically contaminated expressed human milk led to meningitis, brain necrosis, and marked developmental delays. Human milk is the ideal food for nearly all infants and is associated with decreased risk for many illnesses; however, microorganisms can multiply rapidly in expressed human milk (*4*). Although many women report good hygiene while expressing milk (*5*), expressed human milk is frequently contaminated with pathogens (*6*,*7*), most likely because of suboptimal hygiene practices associated with milk expression. Although the source of contamination in this case is unknown, a breast pump kit became contaminated with *C. sakazakii* and was not adequately cleaned or sanitized, leading to contamination of the milk expressed with this kit on several days. Human milk contaminated during or after expression can put infants at risk for infection with various pathogens, including *C. sakazakii* (*7*). CDC has developed guidance to optimize breast pump hygiene.* Clinicians should provide detailed recommendations about hygienic expression and handling of human milk to parents who plan to feed EMM to their infants. Settings in which mothers might need to pump their milk, such as hospitals and workplaces, should facilitate hygienic expression and handling of human milk.

Conflict of Interest

No conflicts of interest were reported.

* https://www.cdc.gov/healthywater/hygiene/healthychildcare/infantfeeding/breastpump.html.

Acknowledgments

Kristen Mertz, Robin Shaw, Barbara J. Grosch, Allegheny County Health Department, Pennsylvania; Barbara R. Hildebrand, Susann J. Guess, Magee-Womens Hospital of UPMC, Pittsburgh, Pennsylvania; Deborah Simonetti, UPMC Presbyterian Clinical Microbiology Laboratory, Pittsburgh, Pennsylvania; Allison Longenberger, Pennsylvania Department of Health; Southeast Regional Laboratory, Food and Drug Administration Philadelphia District Office; Jacqueline Hurd, Jonathan Jackson, Janet Pruckler, Rachael D. Aubert, Gillian McAllister, Maryann Turnsek, Maurice Curtis, Molly Freeman, Kelley Hise, Division of Foodborne, Waterborne and Environmental Diseases, National Center for Emerging and Zoonotic Infectious Diseases, CDC; Katherine Shealy, Erica Anstey, Jennifer Nelson, Cria Perrine, Division of Nutrition, Physical Activity, and Obesity, National Center for Chronic Disease Prevention and Health Promotion, CDC.

[1]Division of Foodborne, Waterborne and Environmental Diseases, National Center for Emerging and Zoonotic Infectious Diseases, CDC; [2]University of Pittsburgh, Pennsylvania; [3]Magee-Womens Hospital of UPMC, Pittsburgh, Pennsylvania; [4]UPMC Presbyterian Clinical Microbiology Laboratory, Pittsburgh, Pennsylvania; [5]Children's Hospital of Pittsburgh of UPMC, Pennsylvania; [6]Allegheny County Health Department, Pennsylvania; [7]Coordinated Outbreak Response and Evaluation Network, Food and Drug Administration, Silver Spring, Maryland.

Corresponding author: Anna Bowen, abowen@cdc.gov, 404-639-4636.

References

1. Food and Agriculture Organization of the United Nations; World Health Organization Department of Food Safety, Zoonoses and Foodborne Disease. *Enterobacter sakazakii* (*Cronobacter* spp.) in powdered follow-up formulae [meeting report]. Rome, Italy: United Nations, Food and Agriculture Organization; Geneva Switzerland: World Health Organization, Department of Food Safety, Zoonoses and Foodborne Disease; 2008. http://www.who.int/foodsafety/publications/micro/MRA_followup.pdf
2. Santos M, Silva C, Marangoni D, Pinto M, Moreira B. Detection of *Enterobacter sakazakii* sepsis outbreak in four hospitals in Rio de Janeiro, Brazil. Presented at the Fourth Decennial International Conference on Nosocomial and Healthcare-Associated Infections; Atlanta, Georgia; March 5–9, 2000.
3. Drudy D, O'Rourke M, Murphy M, et al. Characterization of a collection of *Enterobacter sakazakii* isolates from environmental and food sources. Int J Food Microbiol 2006;110:127–34. https://doi.org/10.1016/j.ijfoodmicro.2006.02.008
4. Lenati RF, O'Connor DL, Hébert KC, Farber JM, Pagotto FJ. Growth and survival of *Enterobacter sakazakii* in human breast milk with and without fortifiers as compared to powdered infant formula. Int J Food Microbiol 2008;122:171–9. https://doi.org/10.1016/j.ijfoodmicro.2007.11.084
5. Reyes-Foster BM, Carter SK, Hinojosa MS. Human milk handling and storage practices among peer milk-sharing mothers. J Hum Lact 2017;33:173–80. https://doi.org/10.1177/0890334416678830
6. Landers S, Updegrove K. Bacteriological screening of donor human milk before and after Holder pasteurization. Breastfeed Med 2010;5:117–21. https://doi.org/10.1089/bfm.2009.0032
7. Smith SL, Serke L. Case report of sepsis in neonates fed expressed mother's milk. J Obstet Gynecol Neonatal Nurs 2016;45:699–705. https://doi.org/10.1016/j.jogn.2016.05.006

Notes from the Field

Hospital Contact Investigation for a Patient Who Developed a Zoster Vaccine–Related Rash — Maryland, February 2015

Diana P. Le, MD[1]; Jamie Vega, DO[2]; Lucas A Johnson, MD[1]

On January 30, 2015, the public health department of a Maryland hospital was notified of a patient who developed a disseminated rash after receiving the live attenuated zoster vaccine, Zostavax (Merck). Zostavax is routinely provided to adults aged ≥50 years for the prevention of herpes zoster (shingles). The patient, a man aged 51 years, was evaluated in an outpatient clinic on postvaccination day 21, at which time physical exam revealed a nonpainful, nonpruritic, mixed maculopapular and vesicular rash (approximately 50 total lesions) involving the patient's face, torso, groin, and arms. The patient was born and raised in Greece and reported that he did not have varicella (chickenpox) as a child, and he did not recall previous vaccination against varicella. For 10 months before developing the rash, the patient received a weekly 10-mg dose of methotrexate for rheumatoid factor-negative spondyloarthropahy. He reported no contact with persons known or suspected to have active varicella or zoster and had no cough or other constitutional symptoms. Valacyclovir, an oral antiviral medication, was prescribed, and the patient was instructed to remain at home and avoid outside contacts until the lesions crusted over. All household contacts of the patient had documented evidence of receipt of 2 doses of varicella vaccine. Direct immunofluorescence testing and culture of vesicular fluid were positive for varicella zoster virus (VZV) on postvaccination day 24.

Although zoster is less contagious than varicella (particularly if the rash is appropriately covered), the unusual presentation of the rash raised concerns for varicella. A contact investigation was initiated by the hospital's public health department to identify health care worker contacts as well as clinic waiting room patient contacts who might have been exposed to the patient during the outpatient clinic visit. Eight health care workers were identified as having had face-to-face contact with the patient; all had documented evidence of VZV immunity by antibody titer or documentation of receipt of 2 doses of varicella vaccine. The patient spent approximately 25 minutes in the clinic waiting room, resulting in potential exposure of 18 persons. Among these persons, 15 had evidence of VZV immunity and the three who did not were offered vaccination. None of the unimmunized waiting room contacts was pregnant, had an immunocompromising medical condition, or was undergoing immunomodulatory treatment. In accordance with current guidelines, exposed health care workers were instructed to remain vigilant for signs or symptoms of varicella (i.e., fever, headache, or other constitutional symptoms and skin lesions) for 21 days after exposure and to immediately report symptoms to the hospital's public health department if any occurred (*1*). No cases of varicella were identified in potentially exposed patients or health care workers after 21 days of surveillance. After the conclusion of the contact investigation, vesicular fluid collected and sent for genotyping at time of the initial patient evaluation demonstrated vaccine-type (Oka/Merck) virus.

Development of a generalized rash following zoster vaccination is rare, but can occur. Circulation of wild type VZV has declined considerably during the era of varicella and zoster vaccines (*2*); however, given the delays and challenges in determining if a vesicular rash in a vaccine recipient is VZV, early institution of a contact investigation by clinicians and public health officials might mitigate the risk for VZV transmission. Potentially exposed contacts without evidence of VZV immunity should be vaccinated against varicella. Both varicella vaccination and varicella zoster immune globulin are most effective in preventing disease in susceptible persons when administered as soon as possible after exposure (*3*).

Health care settings should ensure that health care workers are immune to VZV. Documentation of immunity includes: 1) written documentation of vaccination with 2 doses of varicella vaccine; 2) laboratory evidence of immunity or laboratory confirmation of disease; 3) diagnosis or verification of a history of varicella disease by a health care provider; or 4) diagnosis or verification of a history of herpes zoster by a health care provider (*4*,*5*). This case highlights the importance of maintaining vigilance for unusual events following the use of live vaccines in persons who receive immunosuppressant medications, the importance of vaccination for primary prevention of communicable diseases in hospital settings, and the value of a robust occupational health program as a critical component of hospital infection control efforts.

Conflict of Interest

No conflicts of interest were reported.

[1]Uniformed Services University of the Health Sciences, Bethesda, Maryland; [2]Walter Reed National Military Medical Center, Bethesda, Maryland.

Corresponding author: Lucas A. Johnson, lucas.johnson@usuhs.edu, 757-953-6600.

References

1. CDC. Preventing varicella-zoster virus (VZV) transmission from zoster in healthcare settings. Atlanta, GA: US Department of Health and Human Services, CDC; 2014. https://www.cdc.gov/shingles/hcp/HC-settings.html#healthcare-personnel
2. Lopez AS, Zhang J, Marin M. Epidemiology of varicella during the 2-dose varicella vaccination program—United States, 2005–2014. MMWR Morb Mortal Wkly Rep 2016;65:902–5.
3. Lopez AS, Marin M. Strategies for the control and investigation of varicella outbreaks. Atlanta, GA: US Department of Health and Human Services, CDC; 2008. https://www.cdc.gov/chickenpox/outbreaks/downloads/manual.pdf
4. Marin M, Güris D, Chaves SS, Schmid S, Seward JF; Advisory Committee on Immunization Practices, CDC. Prevention of varicella: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep 2007;56(No. RR-4).
5. Shefer A, Atkinson W, Friedman C, et al.; Advisory Committee on Immunization Practices, CDC. Immunization of health-care personnel: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep 2011;60(No. RR-7).

Announcement

Community Preventive Services Task Force Recommendations for Multicomponent Interventions to Increase Breast, Cervical, and Colorectal Cancer Screening

The Community Preventive Services Task Force recently posted on its website new information regarding recommendations for multicomponent interventions for three different cancers: 1) "Increasing Cancer Screening: Multicomponent Interventions — Breast Cancer," https://www.thecommunityguide.org/findings/cancer-screening-multicomponent-interventions-breast-cancer; 2) "Increasing Cancer Screening: Multicomponent Interventions — Cervical Cancer," https://www.thecommunityguide.org/findings/cancer-screening-multicomponent-interventions-cervical-cancer; and 3) "Increasing Cancer Screening: Multicomponent Interventions — Colorectal Cancer," https://www.thecommunityguide.org/findings/cancer-screening-multicomponent-interventions-colorectal-cancer.

Established in 1996 by the U.S. Department of Health and Human Services, the task force is an independent, nonfederal panel of public health and prevention experts who are appointed by the director of CDC. The task force provides information for a wide range of persons who make decisions about programs, services, and other interventions to improve population health. Although CDC provides administrative, scientific, and technical support for the task force, the recommendations developed are those of the task force and do not undergo review or approval by CDC.

Erratum

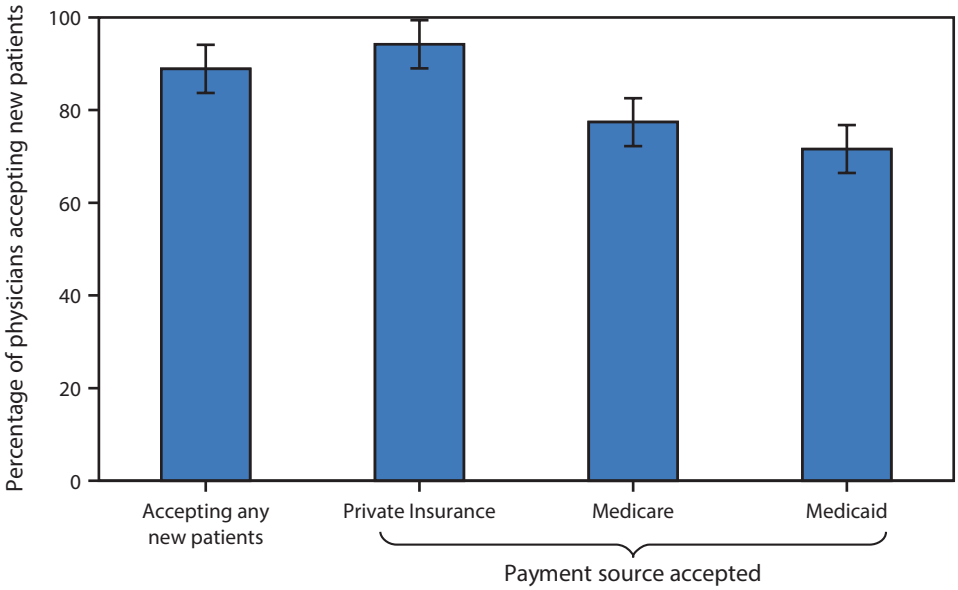
Vol. 66, No. 23

In the report, "Tobacco Use Among Middle and High School Students — United States, 2011–2016," an error occurred in the Table. The prevalence estimate for cigar smoking among high school males should read "**9.9** (8.6–11.2)."

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Percentage* of Office-Based Primary Care Physicians Accepting New Patients, by Source of Payment Accepted — National Electronic Health Records Survey, 2015



*With 95% confidence intervals indicated with error bars.

Overall, 88.9% of primary care physicians reported that they accepted new patients. However, acceptance varied by the patient's expected payment source: 94.2% of physicians accepting new patients accepted privately insured patients, 77.4% accepted new Medicare patients, and 71.6% accepted new Medicaid patients. The percentages of primary care physicians accepting new Medicaid or Medicare patients were significantly lower than the percentage of primary care physicians accepting new privately insured patients.

Source: National Electronic Health Records Survey, 2015 data. Data available through the NCHS Research Data Center at https://www.cdc.gov/rdc/index.htm; survey questionnaire available at https://www.cdc.gov/nchs/ahcd/ahcd_questionnaires.htm.

Reported by: Ninee Yang, PhD, nyang1@cdc.gov, 301-458-4689; Esther Hing, MPH.

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format. To receive an electronic copy each week, visit *MMWR*'s free subscription page at *https://www.cdc.gov/mmwr/mmwrsubscribe.html*. Paper copy subscriptions are available through the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone 202-512-1800.

Readers who have difficulty accessing this PDF file may access the HTML file at *https://www.cdc.gov/mmwr/index2017.html*. Address all inquiries about the *MMWR* Series, including material to be considered for publication, to Executive Editor, *MMWR* Series, Mailstop E-90, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30329-4027 or to mmwrq@cdc.gov.

All material in the *MMWR* Series is in the public domain and may be used and reprinted without permission; citation as to source, however, is appreciated.

Use of trade names and commercial sources is for identification only and does not imply endorsement by the U.S. Department of Health and Human Services.

References to non-CDC sites on the Internet are provided as a service to *MMWR* readers and do not constitute or imply endorsement of these organizations or their programs by CDC or the U.S. Department of Health and Human Services. CDC is not responsible for the content of these sites. URL addresses listed in *MMWR* were current as of the date of publication.

ISSN: 0149-2195 (Print)