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Hepatitis Awareness Month and Testing Day — May 2019

May is designated as Hepatitis Awareness Month, and May 19 is Hepatitis Testing Day. Hepatitis B and hepatitis C, the most common types of viral hepatitis in the United States, can cause chronic infections, and many persons remain unaware of their infection until serious complications occur. In 2016, an estimated 862,000 and 2.4 million persons were living with hepatitis B and hepatitis C, respectively, despite availability of a vaccine and effective treatment for hepatitis B and a cure for hepatitis C (*1*,*2*).

Although hepatitis A is preventable through vaccination, multiple states have had outbreaks since 2016, with unprecedented large numbers of cases and person-to-person spread (primarily among persons who use drugs or experience homelessness). A report in this issue of *MMWR* summarizes this resurgence of hepatitis A among unvaccinated adults at risk (*3*).

New cases of hepatitis C are also increasing; during 2010–2016, they increased 3.5-fold, mostly among young adults (*4*). Recent increases in viral hepatitis infections, many attributed to surges in injection-drug use (*4*), highlight the importance of acknowledging and combatting the infectious disease consequences of the nation's opioid crisis.

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Increase in Hepatitis A Virus Infections — United States, 2013–2018

Monique A. Foster, MD[1]; Megan G. Hofmeister, MD[1]; Benjamin A. Kupronis, MPH[1]; Yulin Lin, MD[1]; Guo-Liang Xia, MD[1]; Shaoman Yin, PhD[1]; Eyasu Teshale, MD[1]

Hepatitis A virus (HAV) is primarily transmitted fecal-orally after close contact with an infected person (*1*); it is the most common cause of viral hepatitis worldwide, typically causing acute and self-limited symptoms, although rarely liver failure and death can occur (*1*). Rates of hepatitis A had declined by approximately 95% during 1996–2011; however, during 2016–2018, CDC received approximately 15,000 reports of HAV infections from U.S. states and territories, indicating a recent increase in transmission (*2*,*3*). Since 2017, the vast majority of these reports were related to multiple outbreaks of infections among persons reporting drug use or homelessness (*4*). In addition, increases of HAV infections have also occurred among men who have sex with men (MSM) and, to a much lesser degree, in association with consumption of imported HAV-contaminated food (*5*,*6*). Overall, reports of hepatitis A cases increased 294% during 2016–2018 compared

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with 2013–2015. During 2016–2018, CDC tested 4,282 specimens, of which 3,877 (91%) had detectable HAV RNA; 565 (15%), 3,255 (84%), and 57 (<1%) of these specimens were genotype IA, IB, or IIIA, respectively. Adherence to the Advisory Committee on Immunization Practices (ACIP) recommendations to vaccinate populations at risk can help control the current increases and prevent future outbreaks of hepatitis A in the United States (*7*).

Hepatitis A infections among persons who meet the Council of State and Territorial Epidemiologists (CSTE) hepatitis A case definition (https://wwwn.cdc.gov/nndss/conditions/hepatitis-a-acute/) are notified to CDC through the National Notifiable Diseases Surveillance System (NNDSS). Cases reported to CDC through NNDSS during 2013–2018 were used to calculate percent change (2013–2015 versus 2016–2018) by state and mapped using RStudio software (version 1.2.1335; RStudio, Inc.). Serum specimens from CSTE confirmed cases submitted to the CDC laboratory were tested for HAV RNA by polymerase chain reaction, and isolated virus was amplified to characterize a 315–base-pair fragment of the VP1/P2B region, which defines the genotype of the virus.

Overall, reports of hepatitis A cases increased 294% during 2016–2018 compared with 2013–2015 (Figure). Eighteen states had lower case counts during 2016–2018 compared with 2013–2015. Nine states and Washington, DC had an increase of approximately 500%. During 2013–2018, 4,508 HAV anti-immunoglobulin M–positive specimens underwent additional testing at CDC. During 2013–2015, 226 specimens underwent additional testing, of which 197 (87%) had detectable HAV RNA; of the RNA-positive specimens, 76 (39%), 121 (61%), and 0 (0%) tested positive for a genotype IA, IB, or IIIA viral strain, respectively. In comparison, 4,282 specimens were tested by CDC during 2016–2018, of which 3,877 (91%) had detectable HAV RNA; 565 (15%), 3,255 (84%), and 57 (<1%) of these specimens were genotype IA, IB, or IIIA, respectively.

Discussion

The number of hepatitis A infections reported to CDC increased during 2016–2018, along with the number of specimens from infected persons submitted to CDC for additional testing. In the past, outbreaks of hepatitis A virus infections occurred every 10–15 years and were associated with asymptomatic children (*8*). With the widespread adoption of universal childhood vaccination recommendations (https://www.cdc.gov/mmwr/preview/mmwrhtml/rr5507a1.htm), asymptomatic children are no longer the main drivers of hepatitis A outbreaks (*3*,*9*). Although the overall incidence rate of HAV infections has decreased within all age groups, a large population of susceptible, unvaccinated adults who were not infected by being exposed to the virus during childhood remain vulnerable to infection by contaminated foods (typically imported from countries with endemic HAV transmission) and recently, on a much larger scale, through behaviors that increase risk for infection in certain vulnerable populations, such as drug use (*3*).

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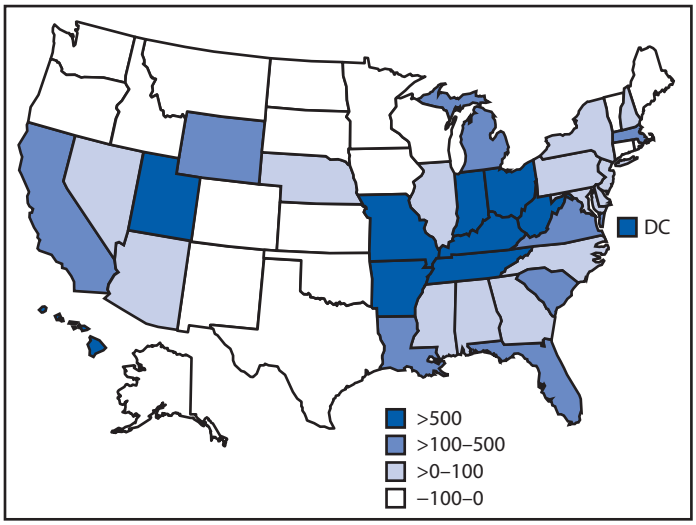
FIGURE. Percent change in reported hepatitis A infections, by state — National Notifiable Diseases Surveillance System, United States, 2013–2015 and 2016–2018*



Abbreviation: DC = District of Columbia.
* 2017 and 2018 case counts are provisional.

Increasingly, molecular epidemiology is employed by public health laboratories to better characterize hepatitis A transmission patterns. When combined with reliable epidemiologic data, these laboratory data can be used to identify transmission networks and confirm the source of exposure during common-source outbreaks, facilitating prompt and effective public health response. Historically, genotype IA has been the most common genotype circulating in North and South America. During 2013–2018, HAV genotype IB predominated in the United States. Increasing numbers of genotype IIIA were seen, a genotype that is considered rare in the United States.

Decreasing new infections from hepatitis A virus can be achieved and sustained by maintaining a high level of population immunity through vaccination. There is no universal vaccination recommendation for adults in the United States; however, ACIP does recommend vaccination for adults who plan travel to HAV-endemic countries, MSM, persons who use drugs, persons with chronic liver disease, and recently, persons experiencing homelessness (*7*). Continued efforts to increase hepatitis A vaccination coverage among the ACIP-recommended risk groups is vital to halting the current hepatitis A outbreaks and reducing overall hepatitis A incidence in the United States.

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Summary

What is already known about this topic?

Hepatitis A is a vaccine-preventable viral infection of the liver that is primarily transmitted through consumption of microscopic amounts of feces.

What is added by this report?

During 2016–2018, reports of hepatitis A infections in the United States increased by 294% compared with 2013–2015, related to outbreaks associated with contaminated food items, among men who have sex with men, and primarily, among persons who report drug use or homelessness.

What are the implications for public health practice?

Increasing vaccination among groups at risk for hepatitis A infection might halt ongoing outbreaks and prevent future outbreaks.

[1]Division of Viral Hepatitis, National Center for HIV, Viral Hepatitis, STD, and TB Prevention, CDC.

Corresponding author: Monique A. Foster, ydg9@cdc.gov, 404-718-8561.

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Disparities in Incidence of Human Immunodeficiency Virus Infection Among Black and White Women — United States, 2010–2016

Erin L.P. Bradley, PhD[1,2]; Austin M. Williams, PhD[3]; Shana Green, PhD[1,2]; Ashley C. Lima, PhD[1,2]; Angelica Geter, PhD[1,2]; Harrell W. Chesson, PhD[3]; Donna Hubbard McCree, PhD[1]

Incident human immunodeficiency virus (HIV) infections among adolescent females and women declined during 2010–2016, with the largest decrease (21%) occurring among black women (*1*). However, in 2016, although black women accounted for 13% of the U.S. female population, 60% of new HIV infections among women were in black women, indicating persisting disparities (*1*). CDC used the population attributable proportion (PAP) disparity measure to describe the proportional decrease in HIV infection among black and white women combined that would be realized if the group with the higher rate (blacks) had the same rate as did the group with the lower rate (whites) (*2*). Analyses indicated that an estimated 3,900 of 4,200 (93%) incident HIV infections among black women in 2016 would not have occurred if rates were the same for black and white women. The PAP disparity measure decreased from 0.75 in 2010 to 0.70 in 2016, suggesting that if incidence rates for black women were the same as those for white women, the annual number of incident HIV infections among black and white women would have been 75% lower in 2010 and 70% lower in 2016. Continued efforts are needed to identify and address social and structural determinants associated with HIV-related disparities to eliminate these disparities and decrease HIV incidence among black women.

CDC calculated the PAP disparity measure to assess trends in HIV infection disparities among black and white women in the United States from 2010 to 2016. HIV incidence and prevalence estimates for women and adolescent females aged ≥13 years from an HIV Supplemental Surveillance Report (*1*) were used to compare estimated incidence with the incidence had there been no racial disparity between blacks and whites (black-white disparity). The PAP disparity measure was calculated as the number of excess incident infections among black females divided by the total number of estimated incident infections among black and white females combined. Excess incident infections were determined as the estimated number of incident infections among black females minus the hypothetical number of incident infections (infections among black females in the absence of a black-white rate disparity). The hypothetical number of incident infections was obtained by dividing the HIV incidence rate in white females by 100,000 and then multiplying by the number of HIV-negative black females. To increase precision in the analyses because incident infection counts in the surveillance report were rounded to the nearest hundred, the estimated number of incident HIV infections was derived by dividing the surveillance report rate by 100,000, then multiplying by the number of females aged ≥13 years. Rates of HIV infection were defined as the estimated number of incident infections divided by the number of HIV-negative females aged ≥13 years, then multiplied by 100,000. This calculation was carried out for each year from 2010 to 2016. To assess changes in the PAP disparity measure between the beginning and the end of the study period, a z-statistic was calculated to test for statistically significant differences between the 2010 and 2016 measures. The z-statistic was calculated as the average difference between the 2016 and 2010 PAP disparity measures in the simulated data divided by the standard error of those differences. Simulations consisted of 10,000 calculations of the annual PAP measures, each using a random draw of the HIV incidence rate from a normal distribution (approximated using the relative standard errors from the surveillance report) (*3*).

From 2010 to 2016, the estimated incidence of HIV infection among black women and adolescent females decreased from 32.5 per 100,000 persons to 24.4; the rate among white women and adolescent females did not differ in 2016 (1.6) compared with that in 2010 (1.6) and ranged from 1.4–1.7 during that time. The PAP disparity measure decreased from 0.75 in 2010 to 0.70 in 2016. This change suggests that if incidence rates for black women were the same as were those for white women, the annual number of incident cases of HIV infection among black and white women would have been 75% lower in 2010 and 70% lower in 2016 (Table). The 7% decrease in the PAP disparity measure from 2010 to 2016 ($p = 0.15$) indicates that the percentage of incident HIV infections attributable to racial disparities between black and white women decreased by about 7% over this period. Thus, in 2016, an estimated 3,900 of 4,200 (93%) incident HIV infections among black women would not have occurred if rates were the same for black and white women.

Discussion

The declines in incidence of HIV infection among black women and adolescent females signal some progress toward reducing racial disparities among women, and these findings are consistent with previous research that indicated reductions in racial/ethnic disparities in diagnosis of HIV infection among

TABLE. Population attributable proportion (PAP) for human immunodeficiency virus (HIV) incidence among black and white women and adolescent females aged ≥13 years, by race — United States, 2010–2016

Year	No. of incident HIV infections* (rate†)		Excess infections among blacks	PAP§	% Change 2010 to 2016¶	P-value
	Blacks	Whites				
2010	5,300 (32.5)	1,400 (1.6)	5,000	0.75	−7	0.15
2011	5,000 (30.7)	1,300 (1.5)	4,800	0.75		
2012	4,700 (28.6)	1,300 (1.5)	4,500	0.74		
2013	4,400 (26.0)	1,200 (1.4)	4,100	0.74		
2014	4,000 (23.4)	1,300 (1.5)	3,700	0.70		
2015	4,100 (23.7)	1,500 (1.7)	3,800	0.68		
2016	4,200 (24.4)	1,400 (1.6)	3,900	0.70		

* Number of incident infections from an HIV Surveillance Report (https://www.cdc.gov/hiv/library/reports/hiv-surveillance.html). Incident infection counts rounded to the nearest hundred.

† Infections per 100,000 population. To increase precision in the analyses, rates were calculated as the estimated number of incident HIV infections not rounded to the nearest hundred (surveillance report rate divided by 100,000, multiplied by the number of females aged ≥13 years) divided by the number of HIV-negative females aged ≥13 years, then multiplied by 100,000.

§ The PAP disparity measure reflects the percentage of HIV infections attributable to racial disparities in HIV incidence between black and white women and adolescent females aged ≥13 years. The PAP measure was calculated as the number of excess incident infections among black females divided by the total number of estimated incident infections among black and white females. Excess incident infections among black females refers to the estimated number of incident infections among black women minus the hypothetical number of incident infections that would have occurred among black women if their HIV incidence rate were the same as that of white women. The hypothetical number of incident infections in the absence of a black-white disparity in rates was calculated by dividing the HIV incidence rate in white females by 100,000 and multiplying by the HIV-negative black female population.

¶ The percent change from 2010 to 2016 was calculated as the difference between the 2016 and 2010 PAP values, divided by the 2010 PAP value.

women during 2010–2014 using different measures of disparity (absolute rate difference, diagnosis disparity ratio, and index of disparity) (*4*). However, notable black-white disparities among women persist. In 2016, an estimated 93% of incident HIV infections among black women would not have occurred if the incidence rate for black women were as low as the rate for white women. Estimates of the annual PAP disparity measure during 2010–2016 suggest that eliminating black-white disparities in incident HIV infections among women and adolescent females would have achieved a decrease in overall incidence among black and white women of 75% in 2010 and 70% in 2016. This finding highlights the contribution of racial/ethnic disparities to overall HIV infection rates among women and adolescent females and underscores the importance of further reducing, or eliminating, these differences.

Reducing and monitoring HIV-related disparities are important national goals (*5*). Tailored strategies to reduce disparities in incidence among women should address social and structural determinants, including inequitable access to health care, HIV-related stigma, and comparatively high background prevalence of certain sexually transmitted infections (*6*,*7*), that increase the risk for HIV infection among black women. Because most HIV infections among black women occur through heterosexual transmission (*1*), strategies that also effectively engage heterosexual and bisexual men are important. Social and structural determinants create or sustain disparities in HIV infection, treatment, and care. For example, compared with their white counterparts, black women and men experience longer delays in diagnosis (*8*) and are less likely to be virally suppressed (i.e., <200 copies of viral RNA per mL of blood) (*9*,*10*). Targeted measures that address reducing transmission through viral suppression and preventing acquisition through biomedical and behavioral interventions (e.g., preexposure prophylaxis [PrEP] and condom use; and providing adequate treatment once HIV infection is diagnosed) will play important roles in reducing disparities.

The findings in this report are subject to at least five limitations. First, estimates of HIV incidence are subject to model assumptions and data completeness (*1*). Second, only one measure of disparity was used, limiting a more comprehensive analysis of racial/ethnic disparities in incidence of HIV infection among women and adolescent females. Using other measures of disparity could provide alternative results. Third, the p-value calculated for the 7% change in the PAP might be overestimated because it assumed no correlation in the error of estimated incidence within racial groups over time. This implies that the error in estimating the 2010 incidence among black women is unrelated to the error in estimating the 2016 incidence among black women. Fourth, although the PAP disparity measure has a straightforward interpretation and quantifies excess HIV infections among black females, this study does not yield additional insight into what structural or policy changes are needed to eliminate disparities. Finally, incidence in only two racial groups was compared, whereas disparities might exist among other racial/ethnic groups.

Despite these limitations, findings from the PAP disparity measure analyses enhance the measurement of HIV disparities among women and adolescent females by quantifying the number of incident HIV infections that might have been prevented in the absence of racial disparities. This information lends support for strengthening HIV prevention and care efforts for heterosexual black females and males to continue progress

Summary

What is already known about this topic?

Rates of human immunodeficiency virus (HIV) infection among all women have declined since 2010, but rates among black women remain higher than do those among white women.

What is added by this report?

A population attributable proportion analysis found that in 2016, an estimated 3,900 of 4,200 (93%) incident HIV infections among black women would not have occurred if the incidence for black women were the same as that for white women.

What are the implication for public health practice?

Reducing racial disparities among women is needed to achieve broader HIV control goals. Addressing social and structural determinants of health and applying tailored strategies to reduce HIV incidence in black women and their partners are important elements to achieving health equity.

toward closing the gap in racial disparities in HIV infection among women. Such gains are needed to achieve the U.S. Department of Health and Human Services' goal of ending the HIV epidemic in the United States by 2030* and prevent deaths related to acquired immunodeficiency syndrome.

* https://www.hiv.gov/federal-response/ending-the-hiv-epidemic/overview.

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Felicia Hardnett, Division of HIV/AIDS Prevention, National Center for HIV/AIDS, Viral Hepatitis, STD, and TB Prevention, CDC.

Corresponding author: Donna Hubbard McCree, zyr1@cdc.gov, 404-639-1834.

[1]Divsion of HIV/AIDS Prevention, National Center for HIV/AIDS, STD and TB Prevention CDC; [2]Oak Ridge Institute for Science and Education, Oak Ridge, Tennessee; [3]Division of STD Prevention, National Center for HIV/AIDS, STD and TB Prevention, CDC.

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Racial Disparities in Mortality Associated with Systemic Lupus Erythematosus — Fulton and DeKalb Counties, Georgia, 2002–2016

S. Sam Lim, MD[1]; Charles G. Helmick, MD[2]; Gaobin Bao, MPH[1]; Jennifer Hootman, PhD[2]; Rana Bayakly, MPH[3]; Caroline Gordon, MD[4]; Cristina Drenkard, MD, PhD[1]

Systemic lupus erythematosus (SLE) is a chronic, systemic autoimmune disease with often nonspecific symptoms that can lead to a delay in diagnosis. The disease disproportionately affects women and minorities. Blacks with SLE also have more severe disease and develop it at an earlier age (*1*). Despite an increase in the 5-year survival rate from 50% in 1955 to approximately 90% in the 2000s, attributed largely to advances in management of SLE (*2*), premature mortality among SLE patients persists, often as a result of disease severity, infections, and cardiovascular disease. Because existing SLE mortality estimates based on death certificate data are known to underestimate SLE deaths (*3*), SLE mortality was analyzed using 2002–2004 data from the population-based Georgia Lupus Registry (*1*). Incident and prevalent SLE cases matched to the National Death Index through 2016 identified 97 and 401 deaths, respectively. Standardized mortality ratios adjusted for age group, sex, and race were two to three times higher among persons with SLE relative to expected deaths in the general population. Blacks had significantly higher cumulative mortality than did whites, and blacks with both incident and prevalent cases were significantly younger at death (mean age 51.8 and 52.3 years, respectively) than were whites (mean age 64.4 and 65.0 years, respectively). Whites had lower mortality after diagnosis than did blacks; among incident cases, mortality among whites did not occur until 5 years after SLE diagnosis, whereas blacks had significantly and persistently higher mortality from the time of diagnosis. There were no significant differences by sex. Current CDC-supported efforts encourage early detection, diagnosis, and treatment, and enhanced self-management skills to mitigate racial disparities and improve outcomes overall among persons with SLE.

The Georgia Lupus Registry (*1*) was designed to collect data on all residents of two Georgia counties (Fulton and DeKalb) in the Atlanta metropolitan area with large black and white populations. The public health surveillance exemption to the Health Insurance Portability and Accountability Act Privacy Rule (https://www.hhs.gov/hipaa/for-professionals/privacy/index.html) allowed investigators to obtain protected health information (PHI) without written consent of the patient. Application of this exemption enabled investigators to ascertain all potential cases, determine whether potential cases met case definition criteria, and provide enough information to prevent duplicate counting of patients examined in multiple facilities. PHI was stored securely, and its use was limited to authorized research personnel, maximizing the use of deidentified data whenever feasible.

The primary sources of potential cases included hospitals, rheumatologists, nephrology groups, and dermatology groups in and around the two counties. Administrative databases were queried retrospectively for billing codes for lupus and related conditions. Secondary sources included laboratories (including pathology laboratories) and queries in other population databases (*1*). Abstractors were trained and underwent regular quality assessments. The study was reviewed and approved by the Institutional Review Boards at Emory University and the Georgia Department of Public Health. CDC determined this study did not meet the definition of human subjects research (public health practice). SLE prevalence was estimated for 2002 and incidence for 2002–2004 from the Georgia Lupus Registry. Denominator data for the two counties were obtained from postcensal population estimates. Age-adjusted estimates and 95% confidence intervals were calculated based on the standard 2000 projected age distribution (*1*).

A case of SLE was defined as meeting either the 1997 update of the 1982 American College of Rheumatology (ACR) revised classification criteria (meeting four or more of the 11 criteria*) (*4*,*5*) or an alternative definition (three of the ACR criteria plus a documented diagnosis of SLE by the patient's board-certified rheumatologist). All incident and prevalent SLE cases were matched to the National Death Index through 2016. Cause of death codes were available but not analyzed because of poor reliability regarding SLE attribution (*3*). Standardized mortality ratios were calculated as the ratio of observed deaths among persons with prevalent SLE to expected deaths in the general county populations; subgroups were compared using the same age group, sex, and race categories. The number of expected deaths was calculated by multiplying the death rate of the general population in Fulton and DeKalb counties by the total number of SLE patients in each group. There were too few deaths to calculate standardized mortality ratios for the incident SLE group. Cumulative mortality used Kaplan-Meier survival analysis for both incident and prevalent cases to determine the percentage

* The 11 criteria are as follows: malar rash, discoid rash, photosensitivity, oral ulcers, arthritis, serositis (pericarditis or pleuritis), renal disorder, neurologic disorder, hematologic disorder, immunologic disorder, and antinuclear antibody. https://www.rheumatology.org/Portals/0/Files/1997%20Update%20of%201982%20Revised.pdf.

of SLE patients dying since their diagnosis (*1*). Analyses were performed using SAS software (version 9.4; SAS Institute).

During 2002–2004, a total of 336 incident SLE cases were identified; these SLE patients were demographically similar to the patients in 1,353 cases with prevalent SLE in 2002 (87%–90% female, 74%–76% black, and 23% white) but were older at SLE diagnosis (mean age 40.6 years) than were patients with prevalent SLE (34.6 years). Among patients with prevalent and incident SLE, 401 and 97 deaths, respectively, occurred through 2016. Standardized mortality ratios using 2002–2016 data were 2.3–3.3 times higher for persons with prevalent SLE relative to expected deaths in the general population (Table). Black females with prevalent SLE were three times more likely to die than were black females in the general population (standardized mortality ratio = 3.38). Cumulative mortality was significantly higher among blacks than among whites for both incident (Figure 1) and prevalent (Figure 2) SLE; death occurred at a younger age among blacks with incident SLE cases (mean age = 51.8 ± 17.5 years) and prevalent SLE cases (mean 52.3 ± 15.9 years) than it did among whites (64.4 ± 18.9 years and 65.0 ± 16.3 years, respectively) ($p<0.001$). Mortality among whites was markedly lower in the years immediately following diagnosis compared with mortality among blacks; among incident cases, no deaths were observed among whites until 5 years after SLE diagnosis, whereas mortality among blacks was persistently higher from the time of diagnosis. In addition, whites with SLE had the same cumulative mortality proportion (9%) in 10 years as that observed in blacks in 2 years (Figure 1). There were no significant differences by sex.

TABLE. Standardized mortality ratios for patients with prevalent cases of systemic lupus erythematosus (SLE) from 2002 to 2016, adjusted by age, sex, and black/white race* — Georgia Lupus Registry

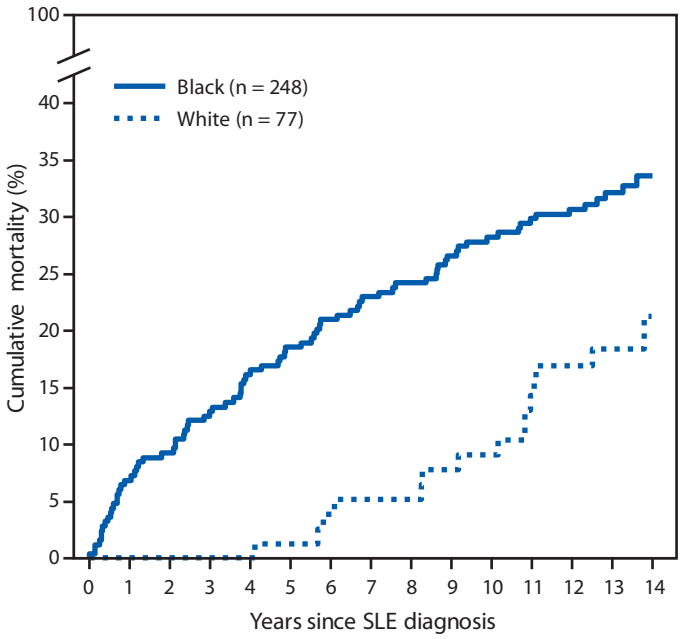
Characteristic	No. of SLE patients (%)	Deaths		Standardized mortality ratio (95% CI)
		Observed	Expected	
Overall (black and white†)	**1,335 (100)**	**400**	**128**	**3.12 (2.83–3.44)**
Sex				
Male	135 (10.1)	51	17	2.98 (2.27–3.92)
Female	1,200 (89.9)	349	111	3.14 (2.83–3.49)
Race				
Black	1,024 (76.7)	324	97	3.34 (3.00–3.72)
White	311 (23.3)	76	31	2.43 (1.94–3.04)
Race/Sex (total = 1,200)				
Black female	924 (77.0)	287	85	3.38 (3.01–3.79)
White female	276 (23.0)	62	26	2.36 (1.84–3.02)

Abbreviation: CI = confidence interval.

* Age on July 1 was used for adjustment. The standardized mortality ratio is a ratio between the observed number of deaths in those with SLE and the number of deaths expected, based on age, sex, and race specific rates in Fulton and DeKalb counties. CIs are based on a generalized estimating equation model with Poisson distribution.

† Eighteen persons who were not identified as black or white, including one who died, were excluded.

FIGURE 1. Cumulative mortality* of incident systemic lupus erythematosus (SLE) cases diagnosed during 2002–2004, by black/white race — Georgia Lupus Registry, 2002–2016



* Cumulative mortality for incident SLE cases was calculated using Kaplan-Meier survival analysis to indicate the probability of SLE patients dying at a specified time since diagnosis. Difference p = 0.008, by log rank test.

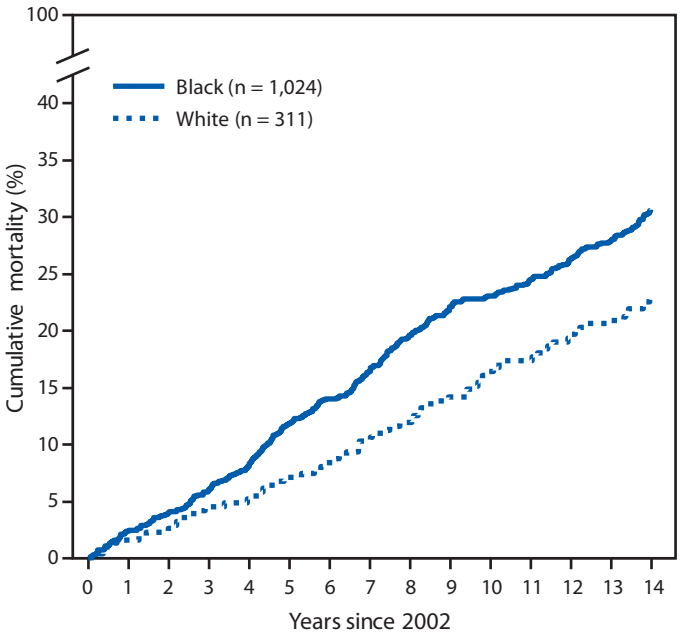
Discussion

Despite increasing awareness of SLE and advancements in treatment (*6*), mortality among persons with SLE remains high, with the highest standardized mortality ratio among black females. The effect of this racial disparity in mortality is further underscored by the fact that the prevalence of SLE in blacks is three times that in whites (*1*).

These findings are similar to those reported in a 2002 study, which also found a higher incidence and prevalence among women and blacks, but the current study used more accurate methods to ascertain cases (*7*). A recent nationwide study using causes of death from 1968 through 2013 obtained from death certificate data in CDC's WONDER database (https://wonder.cdc.gov) showed that age-standardized mortality rates decreased over time among SLE patients but remained high relative to non-SLE mortality, with the highest mortality rates in women, blacks, and residents of the South and West U.S. Census regions (*8*). Both of these studies depended solely on death certificates to identify cases of SLE, which only capture an estimated 40%–60% of SLE cases (*3*,*9*).

The findings in this report are subject to at least four limitations. First, racial identity was assigned based primarily on the physician's assessment as documented in the medical record, which might not reflect the patient's self-identity. Second, some cases might have

FIGURE 2. Cumulative mortality* of prevalent systemic lupus erythematosus (SLE) cases diagnosed in 2002, by black/white race — Georgia Lupus Registry, 2002–2016



* Cumulative mortality for prevalent SLE cases was calculated using Kaplan-Meier survival analysis to indicate the probability of SLE patients dying at a specified time since 2002. Difference p = 0.025, by log rank test.

been missed in the original registry. Third, there might be variability in SLE diagnosis by rheumatologists, and undiagnosed cases were not sought. Finally, these results might not be generalizable outside the two counties. Strengths of the current study include the use of a population-based lupus registry identifying nearly all validated SLE cases in the two-county area and the long follow-up period, resulting in data on more SLE deaths than would be identified by death certificate diagnoses alone.

Prioritizing the identification of reversible mortality factors and developing strategies to address them could aid in mitigating racial disparities and improving outcomes overall in SLE. The first-ever National Public Health Agenda for Lupus (*10*) describes a plan to address lupus from a public health perspective. Other CDC-supported, population-based lupus registries and longitudinal follow-up activities include examining natural history, treatment, access to care, and disparities as potential factors in SLE mortality and progression (https://www.cdc.gov/lupus/funded/lupus-studies.htm). The Lupus Foundation of America and the American College of Rheumatology are working together to encourage early detection and treatment of lupus, enhance the self-management skills of patients with lupus, and improve health care providers' ability to make accurate diagnoses. Additional information is available at https://www.cdc.gov/lupus/funded/awareness.htm.

Summary

What is already known about this topic?

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease that disproportionately affects women and minorities. The 5-year survival rate of patients with SLE has been improving.

What is added by this report?

Using improved methods by following SLE patients carefully defined in a population-based registry, standardized mortality ratios were two to three times higher in persons with SLE than in the general population. Compared with whites with SLE, cumulative SLE mortality was significantly higher among blacks, with deaths occurring sooner after diagnosis and at a mean age approximately 13 years younger.

What are the implications for public health practice?

Current CDC-supported efforts to encourage early detection, diagnosis, and treatment, and to enhance self-management skills might mitigate racial disparities and improve overall outcomes in SLE.

Corresponding author: S. Sam Lim, sslim@emory.edu, 404-712-2945.

[1]Division of Rheumatology, Department of Medicine, Emory University, Atlanta, Georgia; [2]Division of Population Health, National Center for Chronic Disease Prevention and Health Promotion, CDC; [3]Georgia Department of Public Health; [4]Rheumatology Research Group, Institute of Inflammation and Ageing, University of Birmingham, United Kingdom.

All authors have completed and submitted the ICMJE form for disclosure of potential conflicts of interest. No potential conflicts of interest were disclosed.

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Vital Signs: Pregnancy-Related Deaths, United States, 2011–2015, and Strategies for Prevention, 13 States, 2013–2017

Emily E. Petersen, MD[1]; Nicole L. Davis, PhD[1]; David Goodman, PhD[1]; Shanna Cox, MSPH[1]; Nikki Mayes[1]; Emily Johnston, MPH[1]; Carla Syverson, MSN[1]; Kristi Seed[1]; Carrie K. Shapiro-Mendoza, PhD[1]; William M. Callaghan, MD[1]; Wanda Barfield, MD[1]

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Abstract

Background: Approximately 700 women die from pregnancy-related complications in the United States every year.

Methods: Data from CDC's national Pregnancy Mortality Surveillance System (PMSS) for 2011–2015 were analyzed. Pregnancy-related mortality ratios (pregnancy-related deaths per 100,000 live births; PRMRs) were calculated overall and by sociodemographic characteristics. The distribution of pregnancy-related deaths by timing relative to the end of pregnancy and leading causes of death were calculated. Detailed data on pregnancy-related deaths during 2013–2017 from 13 state maternal mortality review committees (MMRCs) were analyzed for preventability, factors that contributed to pregnancy-related deaths, and MMRC-identified prevention strategies to address contributing factors.

Results: For 2011–2015, the national PRMR was 17.2 per 100,000 live births. Non-Hispanic black (black) women and American Indian/Alaska Native women had the highest PRMRs (42.8 and 32.5, respectively), 3.3 and 2.5 times as high, respectively, as the PRMR for non-Hispanic white (white) women (13.0). Timing of death was known for 87.7% (2,990) of pregnancy-related deaths. Among these deaths, 31.3% occurred during pregnancy, 16.9% on the day of delivery, 18.6% 1–6 days postpartum, 21.4% 7–42 days postpartum, and 11.7% 43–365 days postpartum. Leading causes of death included cardiovascular conditions, infection, and hemorrhage, and varied by timing. Approximately sixty percent of pregnancy-related deaths from state MMRCs were determined to be preventable and did not differ significantly by race/ethnicity or timing of death. MMRC data indicated that multiple factors contributed to pregnancy-related deaths. Contributing factors and prevention strategies can be categorized at the community, health facility, patient, provider, and system levels and include improving access to, and coordination and delivery of, quality care.

Conclusions: Pregnancy-related deaths occurred during pregnancy, around the time of delivery, and up to 1 year postpartum; leading causes varied by timing of death. Approximately three in five pregnancy-related deaths were preventable.

Implications for Public Health Practice: Strategies to address contributing factors to pregnancy-related deaths can be enacted at the community, health facility, patient, provider, and system levels.

Introduction

Approximately 700 women die annually in the United States from pregnancy-related complications (*1*). Significant racial/ethnic disparities in pregnancy-related mortality exist; black women have a pregnancy-related mortality ratio approximately three times as high as that of white women (*2*,*3*). Better understanding is needed on the circumstances surrounding pregnancy-related deaths and strategies to prevent future deaths.

This report describes the timing and characteristics of pregnancy-related deaths in the United States using 2011–2015 national CDC Pregnancy Mortality Surveillance System (PMSS) data. Data from 13 state maternal mortality review committees (MMRCs) during 2013–2017 were used to determine the percentage of pregnancy-related deaths that were preventable and factors that contributed to the deaths. MMRC-identified strategies for prevention are reported.

Methods

PMSS was established in 1986 by CDC and the American College of Obstetricians and Gynecologists (ACOG) to evaluate the causes of death and risk factors associated with pregnancy-related deaths. PMSS methodology has been described previously (*2*); CDC's Division of Reproductive Health requests that all states, the District of Columbia, and New York City send death certificates, linked live birth or fetal death certificates, and additional data when available, on deaths that occurred during

Summary

What is already known about this topic?

Approximately 700 women die annually in the United States from pregnancy-related complications.

What is added by this report?

Among pregnancy-related deaths for which timing was known, 31.3% deaths occurred during pregnancy, 16.9% on the day of delivery, 18.6% on days 1–6 postpartum, 21.4% on days 7–42 postpartum, and 11.7% on days 43–365 postpartum. Leading causes of death varied by timing relative to the end of pregnancy. Approximately three in five pregnancy-related deaths were preventable. Contributing factors can be categorized at the community, health facility, patient, provider, and system levels.

What are the implications for public health practice?

Most pregnancy-related deaths are preventable, demonstrating the need to identify and implement strategies to address the multiple contributing factors.

pregnancy or within 1 year after delivery. Information on individual deaths are reviewed by medically trained epidemiologists to determine the pregnancy-relatedness and cause (*4*). A death is determined to be pregnancy-related if the death was caused by a pregnancy complication, a chain of events initiated by pregnancy, or the aggravation of an unrelated condition by the physiologic effects of pregnancy. Cause of death coding includes the following 10 mutually exclusive categories: hemorrhage; infection; amniotic fluid embolism; thrombotic pulmonary or other embolism (i.e., air, septic, or fat); hypertensive disorders of pregnancy (i.e., preeclampsia or eclampsia)*; anesthesia complications; cerebrovascular accidents†; cardiomyopathy; other cardiovascular conditions (e.g., congenital heart disease, ischemic heart disease, cardiac valvular disease, hypertensive heart disease, and congestive heart failure); and other noncardiovascular medical conditions (e.g., endocrine, hematologic, immunologic, and renal).

Pregnancy-related death data from PMSS for 2011–2015 were analyzed. The pregnancy-related mortality ratio (PRMR) is the number of pregnancy-related deaths per 100,000 live births. PRMRs were calculated by race/ethnicity, age, marital status, education, and year. Birth data, used for determining the number of live births, were obtained from U.S. natality files from the National Center for Health Statistics (*5*). SAS (version 9.4; SAS Institute) was used for all analyses.

Cause and timing of pregnancy-related deaths were analyzed. Timing of death was identified as "during pregnancy" when keywords on the death certificate noted the death was during pregnancy or the pregnancy checkbox option "pregnant at the time of death" was checked. Otherwise, timing of death in relation to the end of pregnancy was determined by comparing date of death on the death certificate with date of live birth or fetal death on linked birth or fetal death certificates. The specific timing of postpartum deaths was classified as unknown if there was no linked birth or fetal death certificate.

Data shared by 13 state MMRCs for deaths that occurred during 2013–2017§ were analyzed. Using a standardized data collection system, each multidisciplinary MMRC reviewed available data sources (e.g., medical records, social service records, autopsy reports, and vital records) to determine preventability, factors that contributed to the death, and prevention strategies to address contributing factors. Deaths attributable to suicide, drug overdose, homicide, and unintentional injury were excluded from analyses. MMRCs used the following definition of preventability: "a death is considered preventable if the committee determines that there was some chance of the death being averted by one or more reasonable changes to patient, community, provider, health facility, and/or system factors" (*6*). Percentage of deaths determined by MMRCs to have been preventable were calculated, and chi-squared tests were used to assess whether preventability differed by race/ethnicity or by timing of death. Thematic analyses of MMRC-identified factors that might have contributed to deaths and strategies to prevent future deaths also were conducted.

Results

During 2011–2015, a total of 3,410 pregnancy-related deaths occurred in the United States; the overall PRMR was 17.2 pregnancy-related deaths per 100,000 live births. The highest PRMRs were in women who were black (42.8) and American Indian/Alaska Native (32.5); these PRMRs were 3.3 and 2.5 times as high, respectively, as were those in white women (13.0) (Table 1). The PRMR was highest among women aged ≥35 years and women who were not married. The overall PRMR fluctuated by year, ranging from 15.9 (2012) to 18.0 (2014).

When combined, cardiovascular conditions were responsible for >33% of pregnancy-related deaths; these conditions include cardiomyopathy (10.8%), other cardiovascular conditions (15.1%), and cerebrovascular accidents (7.6%). Other leading causes of pregnancy-related death included other noncardiovascular medical conditions (14.3%), infection (12.5%), and obstetric hemorrhage (11.2%). The cause of death could not be determined for 6.7% of pregnancy-related deaths.

* Deaths caused by hypertension that was not preeclampsia, eclampsia, or gestational hypertension were categorized in the "other cardiovascular conditions" category.

† Deaths caused by cerebrovascular accidents that were a result of preeclampsia or eclampsia were classified in the "hypertensive disorders of pregnancy" category; otherwise, deaths were classified in the "cerebrovascular accidents" category.

§ Arizona (2016), Colorado (2014–2015), Delaware (2013–2017), Florida (2017), Georgia (2013–2014), Hawaii (2015–2016), Illinois (2015), Mississippi (2016–2017), North Carolina (2014–2015), Ohio (2013–2016), South Carolina (2014–2017), Tennessee (2017), and Utah (2015–2016).

Timing of death was known for 2,990 (87.7%) pregnancy-related deaths. Among these deaths, 937 (31.3%) occurred during pregnancy, 506 (16.9%) on the day of delivery, 556 (18.6%) 1–6 days postpartum, 640 (21.4%) 7–42 days postpartum, and 351 (11.7%) 43–365 days postpartum (Table 2). Timing of deaths did not significantly differ between black and white women for most periods; however, a greater proportion of deaths among black women (14.9%) occurred 43–365 days postpartum compared to the proportion of deaths among white women (10.2%) that occurred during the same period (p<0.01).

Distribution of timing of death varied by cause of death (Table 2). Most deaths caused by amniotic fluid embolism occurred on the day of delivery or within 6 days postpartum. Approximately 60% of deaths caused by hypertensive disorders of pregnancy occurred 0–6 days postpartum, whereas those caused by cerebrovascular accidents occurred most frequently 1–42 days postpartum. Deaths caused by cardiomyopathy most commonly occurred 43–365 days postpartum; deaths caused by other cardiovascular conditions occurred most commonly during pregnancy and within 42 days postpartum.

The leading causes of death also varied by time relative to the end of pregnancy. During pregnancy, other noncardiovascular and other cardiovascular conditions were the leading causes of death (Figure); on the day of delivery, hemorrhage and amniotic fluid embolism were the major causes of death. Hemorrhage, hypertensive disorders of pregnancy, and infection were leading causes of death during the first 6 days postpartum. From 6 weeks postpartum (43 days) through the end of the first year (365 days), cardiomyopathy was the leading cause of death.

Among 251 pregnancy-related deaths evaluated for preventability by the 13 MMRCs, a determination was made for 232 (92.4%). Among these, 139 (60.0%) were determined to be preventable deaths. Preventability did not significantly differ between black and white women (p = 0.4), or between Hispanic and white women (p = 0.7), with 57.4% of deaths among black women, 62.7% among white women, and 58.3% among Hispanic women determined to be preventable. Preventability was also similar by timing of pregnancy-related death (59.0% during pregnancy, 53.3% during delivery, 57.1% 1–6 days postpartum, 66.7% 7–42 days postpartum, and 61.9% 43–365 days postpartum; [p = 0.8]).

MMRCs identified an average of three to four contributing factors and two to three prevention strategies per pregnancy-related death. Contributing factors were thematically coded as community factors (e.g., unstable housing and limited access to transportation); health facility factors (e.g., limited experience with obstetric emergencies and lack of appropriate personnel or services); patient factors (e.g., lack of knowledge of warning signs and nonadherence to medical regimens); provider factors (e.g., missed or delayed diagnosis and lack of continuity of care); and system-level factors (e.g., inadequate access to care and poor case coordination) (Table 3). MMRC-identified prevention strategies addressing community factors included expanding clinical office hours and the number of providers who accept Medicaid, prioritizing pregnant and postpartum women for temporary housing programs, and improving access to transportation. Actions addressing health facility factors included implementing obstetric emergency protocols and simulation training, providing telemedicine for facilities without on-site obstetric expertise, and implementing systems to foster communication among multiple providers. Although patient-level contributing factors were commonly identified, prevention strategies to mitigate these factors are often reliant upon providers and health systems. For example, prevention strategies to address patient-level factors included improving patient education materials and providing home health and patient support services. Provider-level prevention strategies included offering provider education to reduce missed or delayed diagnoses, implementing a maternal early warning system (*7*), and improving hand-off communication between obstetricians

TABLE 1. Pregnancy-related deaths, by sociodemographic characteristics — Pregnancy Mortality Surveillance System, United States, 2011–2015

Characteristic	No. of pregnancy-related deaths	Pregnancy-related mortality ratio*
Total	**3,410**	**17.2**
Race/Ethnicity† (N = 3,400)		
White	1,385	13.0
Black	1,252	42.8
American Indian/Alaska Native	62	32.5
Asian/Pacific Islander	182	14.2
Hispanic	519	11.4
Age group (yrs) (N = 3,409)		
<20	158	11.3
20–24	543	12.1
25–29	751	13.2
30–34	799	15.3
35–39	706	28.7
≥40	452	76.5
Highest level of education (N = 2,938)		
Less than high school	572	19.8
High school graduate	1,090	24.2
Some college	775	14.8
College graduate or higher	501	9.4
Marital status (N = 3,371)		
Married	1,543	13.1
Not married	1,828	22.8
Year		
2011	702	17.8
2012	627	15.9
2013	679	17.3
2014	718	18.0
2015	684	17.2

* Number of pregnancy-related deaths per 100,000 live births.
† Women identified as white, black, American Indian/Alaska Natives, or Asian/Pacific Islanders were not Hispanic. Hispanic women could be of any race.

TABLE 2. Pregnancy-related deaths, by cause of death and time of death relative to the end of pregnancy — Pregnancy Mortality Surveillance System, United States, 2011–2015*

Cause of death†	Time of death relative to the end of pregnancy§ No. (%) attributed to each cause (row %)					Total no. of deaths
	During pregnancy	Day of delivery	1–6 days postpartum	7–42 days postpartum	43–365 days postpartum	
Hemorrhage	72 (21.9)	123 (37.4)	105 (31.9)	27 (8.2)	2 (0.6)	**329**
Infection	117 (32.5)	17 (4.7)	83 (23.1)	121 (33.6)	22 (6.1)	**360**
Amniotic fluid embolism	12 (6.9)	114 (65.9)	42 (24.3)	4 (2.3)	1 (0.6)	**173**
Thrombotic pulmonary or other embolism	115 (40.9)	24 (8.5)	41 (14.6)	69 (24.6)	32 (11.4)	**281**
Hypertensive disorders of pregnancy	23 (10.8)	41 (19.3)	94 (44.3)	44 (20.8)	10 (4.7)	**212**
Anesthesia complications	2 (20.0)	3 (30.0)	3 (30.0)	2 (20.0)	0	**10**
Cerebrovascular accidents	68 (29.8)	9 (3.9)	49 (21.5)	79 (34.6)	23 (10.1)	**228**
Cardiomyopathy	48 (15.6)	21 (6.8)	25 (8.1)	75 (24.4)	138 (45.0)	**307**
Other cardiovascular conditions	173 (37.6)	65 (14.1)	61 (13.3)	110 (23.9)	51 (11.1)	**460**
Other noncardiovascular medical conditions	225 (52.7)	61 (14.3)	27 (6.3)	59 (13.8)	55 (12.9)	**427**
Unknown	82 (40.4)	28 (13.8)	26 (12.8)	50 (24.6)	17 (8.4)	**203**
Total	**937 (31.3)**	**506 (16.9)**	**556 (18.6)**	**640 (21.4)**	**351 (11.7)**	**2,990**

* Deaths in which timing of death was unknown were excluded (n = 420).
† Cause of death categories are mutually exclusive.
§ Time of death might be distant from onset of disease or initial event leading to death.

and other providers. MMRC-identified prevention strategies addressing system-level factors included developing policies to ensure that women deliver at a health facility with an appropriate level of maternal care and extending Medicaid coverage for pregnant women to include 1 year of postpartum care.

Discussion

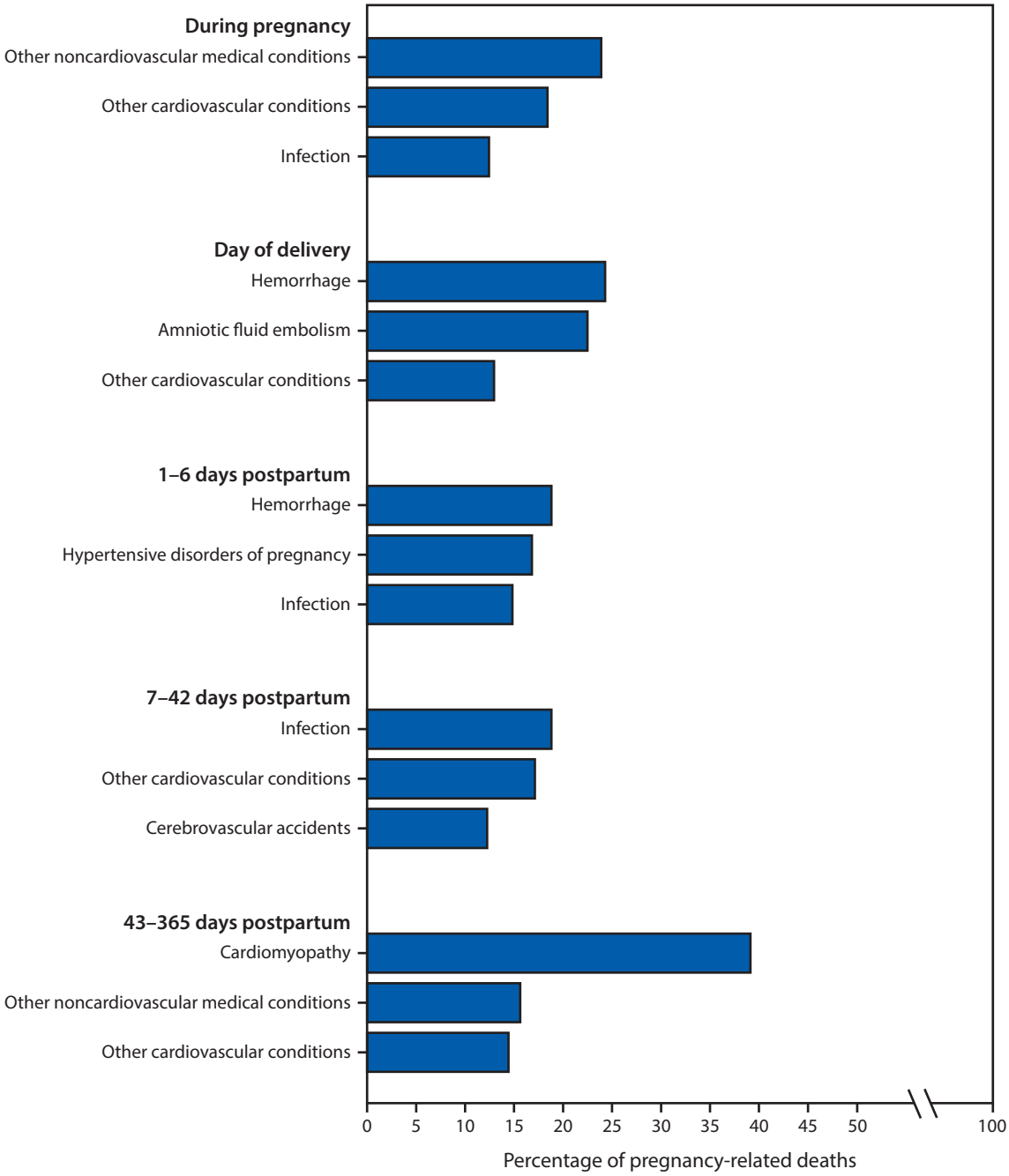
Pregnancy-related deaths occur not only during delivery but also during pregnancy and up to 1 year postpartum. The leading causes of pregnancy-related deaths varied by timing of death. Acute obstetric emergencies such as hemorrhage and amniotic fluid embolism most commonly occurred on the day of delivery, whereas deaths caused by hypertensive disorders of pregnancy and thrombotic pulmonary embolism most commonly occurred 0–6 days postpartum, and during pregnancy and 1–42 days postpartum, respectively. Cardiomyopathy was the most common cause of death in the late postpartum period (43–365 days postpartum). The higher proportion of pregnancy-related deaths in the late postpartum period among black women is likely attributable to higher proportion of pregnancy-related deaths due to cardiomyopathy among these women (*8*). Approximately three in five pregnancy-related deaths were determined by MMRCs to be preventable, and preventability did not differ significantly by race/ethnicity or timing of death. Recognizing the major causes of death by timing can help identify opportunities for intervention.

These data demonstrate the need to address the multiple factors that contribute to pregnancy-related deaths during pregnancy, labor and delivery, and postpartum. No single intervention is sufficient; reducing pregnancy-related deaths requires reviewing and learning from each death, improving women's health, and reducing social inequities across the life span, as well as ensuring quality care for pregnant and postpartum women (*9*). Throughout the preconception, pregnancy, and postpartum periods, providers and patients can work together to optimally manage chronic health conditions (*10*). Standardized approaches to addressing obstetric emergencies can be implemented in all hospitals that provide delivery services. The Alliance for Innovation on Maternal Health (AIM) has provided sets of bundled guidance to provide for such standardization.¶ Implementation of this guidance is often supported by perinatal quality collaboratives, state-based initiatives that aim to improve the quality of care for mothers and infants (*11*). Ensuring that pregnant women at high risk for complications receive care in facilities prepared to provide the required level of specialized care also can improve outcomes; professional organizations have developed criteria for recommended levels of maternal care (*12*). CDC has created the Levels of Care Assessment Tool (LOCATe) for public health decision makers to evaluate risk-appropriate care (*13*). In the postpartum period, follow-up care is critical for all women, particularly those with chronic medical conditions and complications of pregnancy (e.g., hypertensive disorders of pregnancy). ACOG recommends that postpartum women have contact with obstetric providers within the first 3 weeks postpartum and recognizes postpartum care as an ongoing process tailored to each woman's individual needs (*14*).

The findings in this report are subject to at least four limitations. First, errors in reported pregnancy status on the death certificates have been described, potentially leading to overestimation or underestimation of the number of pregnancy-related deaths (*15*). Second, data for specified race or Hispanic-origin groups other than non-Hispanic white and non-Hispanic black

¶ https://safehealthcareforeverywoman.org/aim-supported-patient-safety-bundles.

FIGURE. Three most frequent causes of pregnancy-related deaths, by time relative to the end of pregnancy — Pregnancy Mortality Surveillance System, United States, 2011–2015



should be interpreted with caution because of inconsistencies in reporting these data on death certificates and surveys. Third, generally the pregnancy-relatedness cannot be determined in PMSS for injury deaths such as drug overdoses, suicides, or homicides, or for cancer-related deaths, because of limited information concerning death circumstances. As such, these types of death are often not included in the PRMR. For consistency among data sources, these conditions were not investigated in MMRC data, although MMRC data have found suicides and drug overdoses to be a leading underlying cause of pregnancy-related mortality (*6*). Most (75.0%) of these deaths occur in the late postpartum period. Finally, not all preventable deaths reported by MMRCs had a prevention strategy to address contributing factors; improving quality, completeness, and

TABLE 3. Maternal Mortality Review Committee–identified contributing factors and strategies to prevent future pregnancy-related deaths — Maternal Mortality Review Committees, 13 states, 2013–2017

Level	Contributing factor	Strategies to address contributing factor
Community	Access to clinical care	Expand office hours, increase number of providers who accept Medicaid, increase availability and use of group prenatal care programs
	Unstable housing	Prioritize pregnant and postpartum women for temporary housing programs
	Lack of, or inadequate, transportation options	Strengthen or build systems to link persons to affordable transportation, or provide vouchers for transport to medical appointments
		Improve availability of transportation services covered by Medicaid
	Obesity and associated chronic disease complications	Improve access to healthy foods and enhance efforts to educate and promote healthy eating habits and weight management strategies
Health facility	Limited experience with obstetric emergencies	Implement obstetric emergency simulation training for emergency department and obstetric staff members
		Ensure emergency department staff members ask about recent pregnancy history and consult with obstetrician on call if patient is pregnant or has recently been pregnant
	Lack of appropriate personnel or services	Provide telemedicine for facilities with no obstetric provider on-site
		Ensure Medicaid managed care organizations' contracts include sufficient access to specialists for patients at high risk
	Lack of guiding protocols or tools to help ensure quality care provision	Ensure sepsis, hemorrhage, and massive transfusion protocols are in place and followed by staff members
		Implement applicable patient safety bundles
		Implement systems to foster communication among multiple providers to ensure proper case coordination
		Implement protocols for using patient navigators
Patient/Family	Lack of knowledge of warning signs or need to seek care	Improve counseling and use of patient education materials on warning signs and when to seek care, such as AWHONN Save Your Life discharge instructions
		Implement a public education campaign to increase awareness of signs and symptoms of common complications
	Nonadherence to medical regimens or advice	Standardize patient education to ensure providers relay consistent messages and implement techniques for ensuring patient understanding, such as patient "teaching back" to the provider
		Make education materials available in the clinic and online
		Strengthen and expand access to patient navigators, case managers, and peer support
		Ensure access to interpreter services when needed
		Offer home health or social work follow-up services
Provider	Missed or delayed diagnosis	Repeat blood pressure measurement in a timely (and possibly manual) manner when initial blood pressure result is unexpected
		Offer provider education on cardiac conditions in pregnant and postpartum women
		Perform thorough evaluation of patients reporting pain and shortness of breath
	Inappropriate or delayed treatment	Only perform cesarean deliveries when medically indicated
		Implement a maternal early warning system
	Lack of continuity of care	Improve care transition communication among obstetrician-gynecologists and other primary and specialty care physicians
System	Inadequate receipt of care	Develop policies to ensure pregnant women are transported to a hospital with an appropriate level of maternal care
		Enlist state perinatal quality collaboratives to identify quality improvement procedures and periodic drills/simulation training for birth facilities, including obstetric emergency drills
		Design education initiatives for emergency department staff members on the care of pregnant and postpartum women
	Case coordination or management	Extend expanded Medicaid coverage eligibility for pregnant women to include 1 year of postpartum care
		Create quality improvement entity to manage outpatient care gaps and improve care coordination
		Implement a postpartum care transition bundle for better integration of services for women at high risk
		Develop procedures for all hospitals to improve documentation of abnormal test results, plan for follow-up care, and management of conditions
		Develop universal health record system that allows for sharing of medical records among hospitals
	Guiding policies, procedures, or standards not in place	Develop protocol for timely referrals and consults
		Ensure all hospitals within a health care system follow the same protocols and policies

Abbreviation: AWHONN = Association of Women's Health, Obstetric and Neonatal Nurses.

timeliness of MMRC data can translate into opportunities for prevention. MMRC-identified prevention strategies are based on comprehensive case review by a multidisciplinary group of clinical and nonclinical experts and might not always be drawn from published evidence-based interventions, in part because of a lack of programmatic and policy-based evidence. MMRCs' access to comprehensive medical and social service records highlights their unique and critical role in understanding all factors contributing to pregnancy-related deaths and using those data to identify strategies to potentially prevent future deaths and contribute to the evidence base.

Pregnancy-related deaths occur during pregnancy, around the time of delivery, and within 1 year postpartum; leading causes of death vary by timing of death. Most pregnancy-related deaths can be prevented. Comprehensive review of pregnancy-related deaths can identify contributing factors and opportunities to implement strategies for preventing future deaths.

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Corresponding author: Emily Petersen, fmd9@cdc.gov, 404-488-4316.

[1]Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion, CDC.

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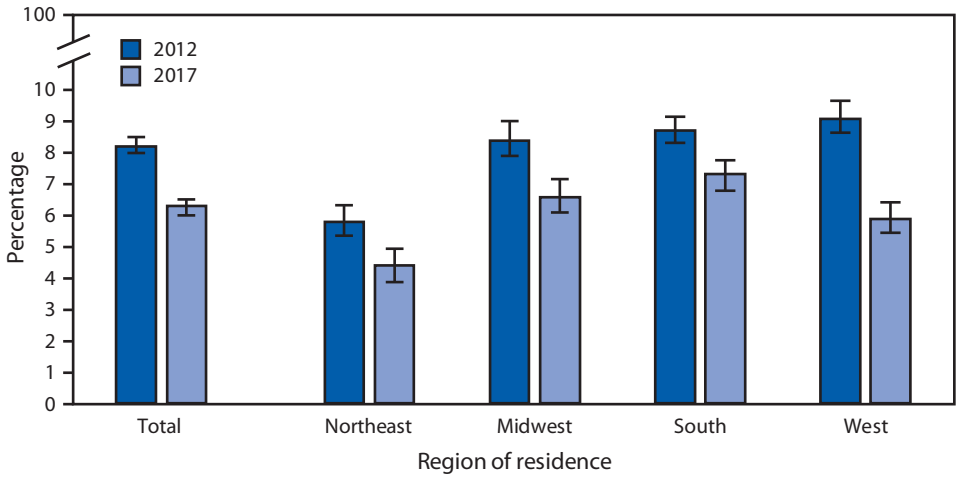
QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Age-Adjusted Percentages* of Persons of All Ages Who Delayed Seeking Medical Care in the Past 12 Months Because of Worry About Cost,† by U.S. Census Region§ of Residence — National Health Interview Survey,¶ 2012 and 2017



* With 95% confidence intervals indicated with error bars.

† Based on a response to the question "During the past 12 months, has [person] delayed seeking medical care because of worry about the cost?" This question excluded dental care. Respondents were asked the question regarding themselves and other family members of all ages living in the same household.

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

¶ Estimates are based on household interviews of a sample of the civilian, noninstitutionalized U.S. population and are derived from the National Health Interview Survey Family Core component. Estimates are age-adjusted using the projected 2000 U.S. population as the standard population and using five age groups: 0–11, 12–17, 18–44, 45–64, and ≥65 years.

The percentage of persons of all ages who delayed seeking medical care in the past 12 months because of worry about the cost decreased from 8.2% in 2012 to 6.3% in 2017, and this pattern was consistent in each U.S. Census region of residence. Delays in seeking medical care because of worry about the cost declined from 5.8% to 4.4% in the Northeast, from 8.4% to 6.6% in the Midwest, from 8.7% to 7.3% in the South, and from 9.1% to 5.9% in the West. In both 2012 and 2017, persons of all ages living in the Northeast were the least likely to delay medical care because of worry about the cost.

Sources: Summary Health Statistics for the U.S. Population, National Health Interview Survey, 2012. https://www.cdc.gov/nchs/data/series/sr_10/sr10_259.pdf.

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Reported by: Jacqueline W. Lucas, MPH, jbw4@cdc.gov, 301-458-4355.

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