Centers for Disease Control and Prevention
MMWR
Morbidity and Mortality Weekly Report
Weekly / Vol. 67 / No. 48
December 7, 2018

Uterine Cancer Incidence and Mortality — United States, 1999–2016

S. Jane Henley, MSPH[1]; Jacqueline W. Miller, MD[1]; Nicole F. Dowling, PhD[1]; Vicki B. Benard, PhD[1]; Lisa C. Richardson, MD[1]

Uterine cancer is one of the few cancers with increasing incidence and mortality in the United States, reflecting, in part, increases in the prevalence of overweight and obesity since the 1980s (*1*). It is the fourth most common cancer diagnosed and the seventh most common cause of cancer death among U.S. women (*1*). To assess recent trends in uterine cancer incidence and mortality by race and ethnicity, CDC analyzed incidence data from CDC's National Program of Cancer Registries (NPCR) and the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program and mortality data from the National Vital Statistics System (*2*). Most recent data available are through 2015 for incidence and through 2016 for mortality. Uterine cancer incidence rates increased 0.7% per year during 1999–2015, and death rates increased 1.1% per year during 1999–2016, with smaller increases observed among non-Hispanic white (white) women than among women in other racial/ethnic groups. In 2015, a total of 53,911 new uterine cancer cases, corresponding to 27 cases per 100,000 women, were reported in the United States, and 10,733 uterine cancer deaths (five deaths per 100,000 women) were reported in 2016. Uterine cancer incidence was higher among non-Hispanic black (black) and white women (27 cases per 100,000) than among other racial/ethnic groups (19–23 per 100,000). Uterine cancer deaths among black women (nine per 100,000) were higher than those among other racial/ethnic groups (four to five per 100,000). Public health efforts to help women achieve and maintain a healthy weight and obtain sufficient physical activity can reduce the risk for developing cancer of the endometrium (the lining of the uterus), the most common uterine cancer. Abnormal vaginal bleeding, including bleeding between periods or after sex or any unexpected bleeding after menopause, is an important symptom of uterine cancer (*3*). Through programs such as CDC's Inside Knowledge* campaign, promoting awareness among women and health care providers of the need for timely evaluation of abnormal vaginal bleeding can increase the chance that uterine cancer is detected early and treated appropriately.

Data on new cases of invasive uterine cancer† diagnosed during 1999–2015 were obtained from population-based cancer registries affiliated with NPCR and SEER. Data from all registries met data quality criteria for U.S. Cancer Statistics§

* https://www.cdc.gov/cancer/knowledge/.

† Uterine cancer cases were defined as microscopically confirmed invasive cancers of the corpus uteri (*International Classification of Diseases for Oncology, Third Edition* [ICD-O-3] site codes C54.0 [isthmus uteri], C54.1 [endometrium], C54.2 [myometrium], C54.3 [fundus uteri], C54.8 [overlapping lesion of corpus uteri], C54.9 [corpus uteri]) and uterus, not otherwise specified (C55.9), excluding cases that were identified by autopsy or death certificate only. Only cases defined as malignant under *International Classification of Diseases for Oncology, Second Edition* (ICD-O-2) and ICD-O-3 were included in this report. Uterine cancer deaths were defined as deaths from cancers of corpus uteri (*International Classification of Diseases for Oncology, Tenth Edition* [ICD-10] codes C54.0–C54.3, C54.8, C54.9) and uterus, not otherwise specified (C55.9).

§ https://www.cdc.gov/cancer/uscs/index.htm.

INSIDE

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

in 2015, and data from 48 states met these criteria each year during 1999–2015, covering 98% of the U.S. population.[¶] Uterine cancers were classified by histologic type (endometrioid carcinoma, other carcinoma, carcinosarcoma, and sarcoma).[**] Stage at diagnosis (localized, regional, distant, or unknown) was characterized using SEER Summary Stage.[††] Data on uterine cancer deaths during 1999–2016 were based on death certificate information reported to state vital statistics offices and compiled into a national file through the National Vital Statistics System, covering 100% of the U.S. population. Data were examined by five mutually exclusive racial/ethnic groups: white, black, non-Hispanic American Indian/Alaska Native (AI/AN), non-Hispanic Asian/Pacific Islander (API), and Hispanic; as well as by histologic type, stage at diagnosis, and year of diagnosis or death.

Population estimates for rate denominators were a modification of annual county population estimates by age, sex, bridged-race, and ethnicity produced by the U.S. Census Bureau in collaboration with CDC's National Center for Health Statistics and with support from the National Cancer Institute.[§§] Annual incidence and death rates per 100,000 women were age-adjusted to the 2000 U.S. standard population. Average annual percent change (AAPC) was used to quantify changes in incidence rates during 1999–2015 and death rates during 1999–2016 and was calculated using joinpoint regression, which allowed different slopes for three periods; years at which slope changed could vary.[¶¶] To determine whether AAPC was significantly different from zero, a t-test was used for zero joinpoints, and a z-test was used for ≥1 joinpoint. Rates were considered to increase if AAPC >0 ($p<0.05$) and to decrease if AAPC <0 ($p<0.05$); otherwise rates were considered stable. All statistical tests were two-sided.

In 2015, 53,911 new microscopically confirmed uterine cancer cases, corresponding to 27 cases per 100,000 women, were reported in the United States (Table). Uterine cancer incidence was higher among white women and black women (27 cases per 100,000 in each group) compared with AI/AN and Hispanic women (23 each) and API women (19). Overall,

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

** Subtypes were classified by ICD-O-3 histology codes as endometrioid carcinomas (8380); other carcinomas (8000–8379, 8381–8790, 8981, 9700–9701); carcinosarcomas (8950 [Müllerian tumors], 8951, 8980); and sarcomas (8800–8932, 8934–8941, 8959–8975, 9141–9582).

†† Cases diagnosed in 1999 were classified using SEER Summary Stage 1977, cases diagnosed during 2000 to 2003 were classified using SEER Summary Stage 2000, and cases diagnosed during 2004 to 2015 were classified using Derived SEER Summary Stage 2000. https://seer.cancer.gov/tools/ssm.

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

¶¶ https://surveillance.cancer.gov/joinpoint/.

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 2018;67:[inclusive page numbers].

TABLE. Number and rate* of invasive uterine cancer cases (2015) and deaths (2016),† by selected characteristics — United States§

			Racial/Ethnic group¶									
	Overall		White		Black		American Indian/ Alaska Native		Asian/Pacific Islander		Hispanic	
Characteristic	No. (%)	Rate	No. (%)	Rate	No. (%)	Rate	No. (%)	Rate	No. (%)	Rate	No. (%)	Rate
Incidence	53,911 (100)	26.5	39,768 (100)	27.0	6,105 (100)	26.5	324 (100)	23.1	2,053 (100)	19.2	5,114 (100)	23.2
Stage at diagnosis**												
Localized	36,021 (67)	17.7	27,393 (69)	18.7	3,359 (55)	14.5	219 (68)	15.6	1,369 (67)	12.8	3,395 (66)	15.2
Regional	11,273 (21)	5.5	8,144 (20)	5.4	1,506 (25)	6.5	64 (20)	4.6	424 (21)	4.0	1,057 (21)	4.9
Distant	4,698 (9)	2.3	3,010 (8)	2.0	997 (16)	4.4	26 (8)	1.9	196 (10)	1.9	449 (9)	2.2
Unknown	1,919 (4)	1.0	1,221 (3)	0.8	243 (4)	1.1	15 (5)	1.1	64 (3)	0.6	213 (4)	1.0
Histologic type												
Endometrioid carcinoma	36,425 (68)	17.9	28,261 (71)	19.3	2,870 (47)	12.4	219 (68)	15.8	1,386 (68)	12.9	3,351 (66)	14.9
Other carcinoma	12,676 (24)	6.1	8,685 (22)	5.7	2,032 (33)	8.8	79 (24)	5.4	477 (23)	4.5	1,224 (24)	5.8
Carcinosarcoma	2,714 (5)	1.3	1,625 (4)	1.0	719 (12)	3.1	13 (4)	1.0	85 (4)	0.8	259 (5)	1.3
Sarcoma	1,790 (3)	1.0	1,013 (3)	0.8	425 (7)	1.9	9 (3)	0.6	92 (4)	0.9	237 (5)	1.0
Histologic type diagnosed at distant stage												
Endometrioid carcinoma	1,124 (3)	0.6	845 (3)	0.6	135 (5)	0.6	7 (3)	0.5	53 (4)	0.5	81 (2)	0.4
Other carcinoma	2,288 (18)	1.1	1,452 (17)	0.9	500 (25)	2.2	14 (18)	1.0	88 (18)	0.9	219 (18)	1.1
Carcinosarcoma	609 (22)	0.3	353 (22)	0.2	163 (23)	0.7	—††	—	25 (29)	0.2	65 (25)	0.3
Sarcoma	643 (36)	0.3	339 (33)	0.3	191 (45)	0.9	—	—	28 (30)	0.3	81 (34)	0.4
Deaths	10,733	5.0	7,391	4.6	2,048	9.0	52	3.7	378	3.5	841	4.0

Sources: CDC's National Program of Cancer Registries; National Cancer Institute's Surveillance; Epidemiology; and End Results program; and CDC's National Center for Health Statistics National Vital Statistics System.

* Per 100,000 women, age-adjusted to the 2000 U.S. standard population.

† Uterine cancer cases were defined as microscopically confirmed cancers of the corpus uteri (*International Classification of Diseases for Oncology, Third Edition* [ICD-O-3] site codes C54.0 [isthmus uteri], C54.1 [endometrium], C54.2 [myometrium], C54.3 [fundus uteri], C54.8 [overlapping lesion of corpus uteri], C54.9 [corpus uteri]), and uterus, not otherwise specified (C55.9), excluding cases that were identified by autopsy or death certificate only. Only cases defined as malignant under *International Classification of Diseases for Oncology, Second Edition* (ICD-O-2) and ICD-O-3 were included in this report. Histologic types were classified by ICD-O-3 histology codes, and include endometrioid carcinomas (8380); other carcinomas (8000–8379, 8381–8790, 8981, 9700–9701); carcinosarcomas (8950 [Müllerian tumors], 8951, 8980); and sarcomas (8800–8932, 8934–8941, 8959–8975, 9141–9582). Uterine cancer deaths were defined as deaths from cancers of corpus uteri (*International Classification of Diseases 10th Edition* [ICD-10] codes C54.0–C54.3, C54.8, C54.9) and uterus, not otherwise specified (C55.9).

§ Cancer incidence compiled from cancer registries that meet the data quality criteria in 2015, covering 100% of the U.S. population. Cancer mortality data cover 100% of the U.S. population.

¶ Mutually exclusive racial/ethnic groups are based on information about race/ethnicity that was collected separately and combined for this report. White, black, American Indian/Alaska Native, and Asian/Pacific Islander race categories are all non-Hispanic. Hispanic persons can be any race. Data are not presented for those with unknown or other race or unknown ethnicity.

** A localized cancer is one that is confined to the primary site, a regional cancer is one that has spread directly beyond the primary site or to regional lymph nodes, and a distant cancer is one that has spread to other organs.

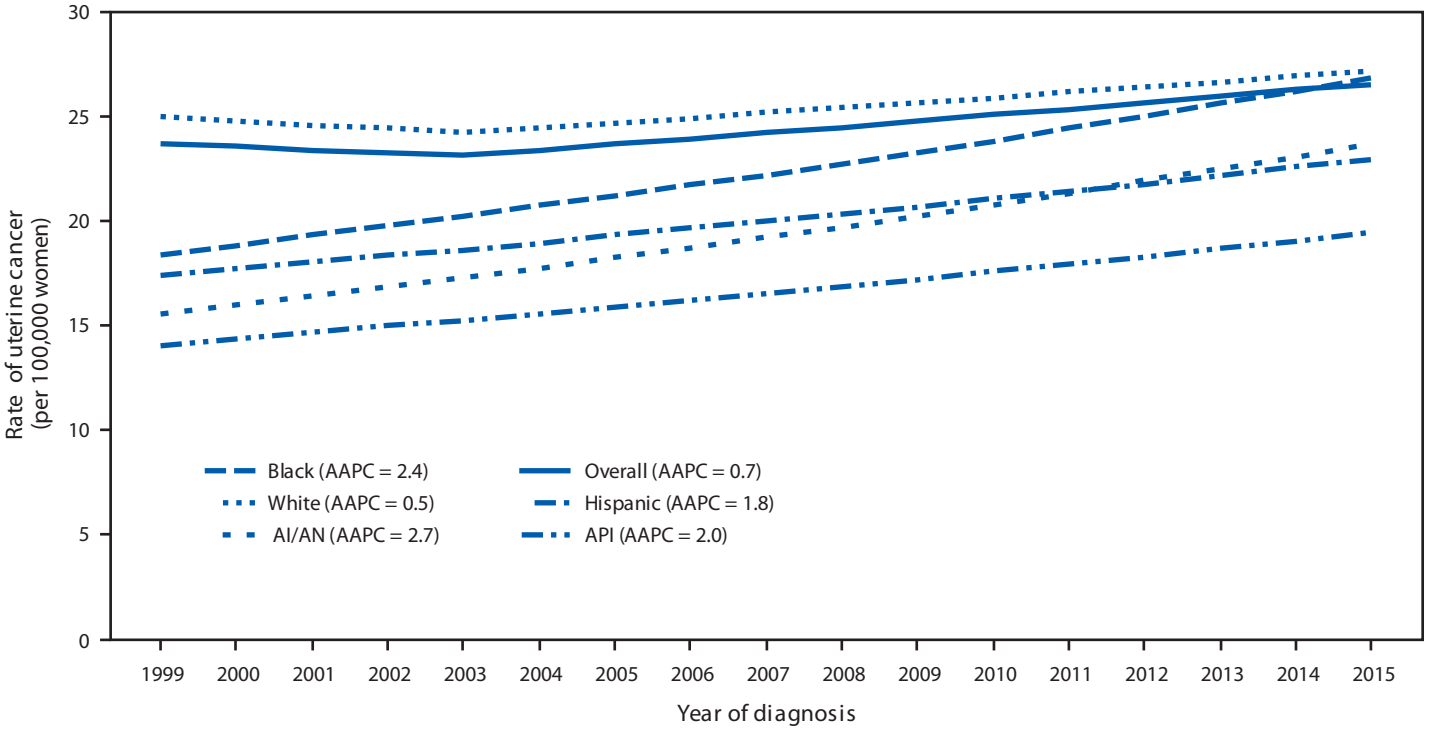
†† Dashes indicate that statistic could not be calculated because fewer than six cases were reported.

endometrioid carcinomas were the most common uterine cancers (68%). However, endometrioid carcinomas accounted for 47% of uterine cancers among black women, who had a higher percentage of other carcinomas, carcinosarcomas, and sarcomas than did women in other racial/ethnic groups. Approximately two thirds of uterine cancers were diagnosed at a localized stage among white (69%), AI/AN (68%), API (67%), and Hispanic women (66%), compared with 55% among black women. A higher proportion of sarcomas were diagnosed at distant stage (36%) than were endometrioid carcinomas (3%), other carcinomas (18%), or carcinosarcomas (22%). The proportion of uterine cancers diagnosed at distant stage was higher among black women than among women of other racial/ethnic groups, overall (16% versus 8%–10%) and for each histologic type, particularly sarcoma (45% versus 30%–34%).

During 1999–2015, uterine cancer incidence rates increased 12%, about 0.7% per year on average, with larger increases observed among AI/AN (53%; AAPC = 2.7%), black (46%; 2.4%), API (38%; 2.0%), and Hispanic (32%; 1.8%) women than among white women (9%; 0.5%) (Figure 1). During 1999–2015, incidence rates of endometrioid carcinomas increased 4.5% per year, other carcinomas decreased 4.5% per year, carcinosarcomas increased 1.9% per year, and sarcoma incidence remained stable (Supplementary Figure, https://stacks.cdc.gov/view/cdc/60809).

In 2016, 10,733 uterine cancer deaths, corresponding to five deaths per 100,000 women, were reported in the United States (Table). Uterine cancer death rates among black women (nine deaths per 100,000) were higher than those among white (five), AI/AN (four), API (four), and Hispanic (four) women. During 1999–2016, uterine cancer death rates increased 21%, approximately 1.1% per year on average, with larger increases among API (52%; AAPC = 2.5%), Hispanic (33%; 1.7%), and black (29%; 1.5%) women, than among white women (18%; 1.0%); rates were stable among AI/AN women (Figure 2).

FIGURE 1. Trends* in age-adjusted uterine cancer incidence rates,† by racial/ethnic group§ — United States,¶ 1999–2015



Sources: CDC's National Program of Cancer Registries and the National Cancer Institute's Surveillance, Epidemiology, and End Results program.
Abbreviations: AAPC = average annual percent change; AI/AN = American Indian/Alaska Native; API = Asian/Pacific Islander.
* Trends were measured with AAPC in rates and were considered to increase or decrease if $p<0.05$; otherwise, rates were considered stable. AAPC is the weighted average of the annual percent change over the period 1999–2015 using a Joinpoint regression model (up to 2 joinpoints).
† Per 100,000 women, age-adjusted to the 2000 U.S. standard population. Uterine cancers were defined as microscopically confirmed cancers of the corpus uteri (*International Classification of Diseases for Oncology, Third Edition* [ICD-O-3] site codes C54.0–C54.3, C54.8, C54.9) and uterus, not otherwise specified (C55.9), excluding cases that were identified by autopsy or death certificate only. Only cases defined as malignant under *International Classification of Diseases for Oncology, Second Edition* (ICD-O-2) and ICD-O-3 were included in this report.
§ Mutually exclusive racial/ethnic groups are based on information about race/ethnicity that was collected separately and combined for this report. White, black, AI/AN, and API race categories are all non-Hispanic. Hispanic persons can be any race.
¶ Cancer incidence compiled from cancer registries that meet the data quality criteria for each year during the period 1999–2015, covering 98% of the U.S. population.

Discussion

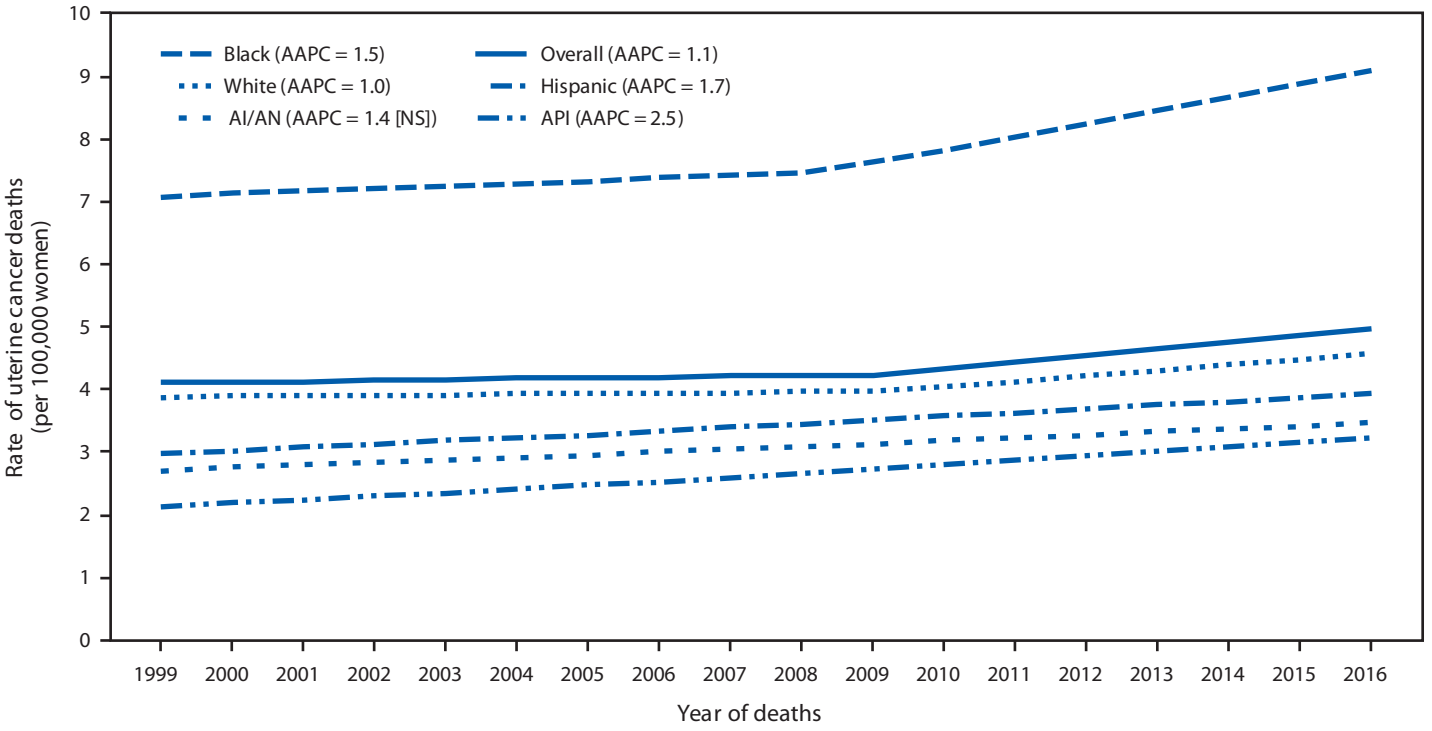
This report indicates that the rate of new uterine cancer cases increased during 1999–2015, with larger increases observed among black, AI/AN, API, and Hispanic women than among white women. This contrasts with the recent decreases in incidence rates that have been observed for many cancer types, such as lung and colorectal cancers (*1*). One contributing factor to increasing uterine cancer incidence could be excess body weight; women who are overweight (body mass index [BMI] = 25.0–29.9 kg/m^2) or have obesity (BMI ≥30 kg/m^2) are approximately two to four times as likely to develop endometrial cancer as are women with healthy weight (*4*). During 2013–2016, approximately 40% of women in the United States had obesity, including 56% of black women and 49% of Hispanic women.*** The U.S. Preventive Services Task Force recommends that clinicians offer or refer adults with obesity to intensive, multicomponent behavioral interventions.††† Community-based strategies to promote healthy body weight include helping persons meet dietary and physical activity guidelines by supporting healthy eating and active living in such settings as communities, worksites, schools, and early care and education facilities (*4*). Other factors such as insufficient physical activity, increasing prevalence of diabetes, and decreasing use of estrogen plus progestin menopausal hormone therapy might also contribute to increases in endometrial cancer incidence (*5*).

This report also found that uterine cancer death rates were higher in 2016 than in 1999 and that black women were approximately twice as likely to die from uterine cancer as were women in other racial/ethnic groups. As with other cancers,

*** https://www.cdc.gov/nchs/hus.

††† https://www.uspreventiveservicestaskforce.org/BrowseRec/Index/browse-recommendations.

FIGURE 2. Trends* in age-adjusted uterine cancer death rates,† by racial/ethnic group§ — United States, 1999–2016



Source: CDC's National Center for Health Statistics National Vital Statistics System.
Abbreviations: AAPC = average annual percent change; AI/AN = American Indian/Alaska Native; API = Asian/Pacific Islander; NS = not significant.
* Trends were measured with AAPC in rates and were considered to increase or decrease if $p<0.05$; otherwise rates were considered stable. AAPC is the weighted average of the annual percent change over the period 1999–2016 using a Joinpoint regression model (up to 2 joinpoints).
† Per 100,000 women, age-adjusted to the 2000 U.S. standard population. Uterine cancer deaths were defined as deaths from cancers of corpus uteri (*International Classification of Diseases 10th Edition* [ICD-10] codes C54.0–C54.3, C54.8, C54.9) and uterus, not otherwise specified (C55.9).
§ Mutually exclusive racial/ethnic groups are based on information about race/ethnicity that was collected separately and combined for this report. White, black, AI/AN, and API race categories are all non-Hispanic. Hispanic persons can be any race.

the odds of surviving uterine cancer are much higher when it is detected at an early stage, when treatment is more effective (*5*). The 5-year relative survival estimate for localized uterine cancer is 80%–90% compared with <30% for distant uterine cancer (*5*). This report found that black women were more likely to receive a diagnosis at distant stage and with more aggressive histologic types than were other women, which might in part account for the higher death rate among black women.

Although population-based screening tests are recommended for several cancers, including breast, cervical, colorectal, and lung cancers, at present, population-based screening tests are not recommended for uterine cancer (*6*). An important early symptom of uterine cancer is abnormal vaginal bleeding, including bleeding between periods or after sex or any unexpected bleeding after menopause (i.e., any bleeding except intermittent bleeding within 1 year after cessation of menses or cyclic bleeding associated with use of cyclic postmenopausal hormone therapy) (*3*). Approximately 90% of women with uterine cancer report abnormal vaginal bleeding (*6*). A lower percentage of women with uterine sarcomas have abnormal vaginal bleeding (approximately 56%) or nonspecific symptoms, such as pelvic pain (22%); consequently, a higher percentage of sarcomas are not detected until the cancer has already spread (*7*). Uterine cancer outcomes could be improved by increasing awareness among women that abnormal vaginal bleeding should be evaluated promptly by a health care provider. It is also important for health care providers to perform timely evaluation and necessary follow-up of women's concerns and symptoms (*8*). Transvaginal ultrasonography or endometrial tissue sampling are appropriate for initial evaluation of postmenopausal bleeding; further evaluation could include hysteroscopy combined with endometrial sampling (*8*). To help women make informed choices, health care providers can educate women about different procedural options (including surgical choices); discuss the benefits and risks of each procedure; and discuss the risk for cancer (*9*). CDC's Inside Knowledge campaign attempts to raise awareness among women and health care providers about uterine cancer and other gynecologic cancers. Inside Knowledge uses

a multimedia approach to ensure campaign messages reach the broadest audience possible.

The findings in this report are subject to at least five limitations. First, reporting of race and ethnicity uses data from medical records and death certificates, which might be inaccurate in some cases, especially among AI/AN; ongoing procedures are used to ensure that this information is as accurate as possible.[§§§] Second, improved pathologic classification of tumors over time might influence rates and trends. Third, broad groups were used for histologic type, which might mask varying levels of tumor behavior. Fourth, in clinical practice, uterine cancers are commonly staged on the basis of histologic type using the International Federation of Gynecology and Obstetrics system (*6*); however, because this information is not routinely collected in cancer registries, this report used SEER Summary Stage to stage cancers. Finally, rate denominators were not adjusted for hysterectomy prevalence and might include women who did not have an intact uterus and were not at risk for uterine cancer, thus underestimating the actual rate among women at risk, particularly black women, who have higher rates of hysterectomy (*10*).

Multifactorial efforts at individual, community, clinical, and systems levels to help women achieve and maintain a healthy weight and obtain sufficient physical activity might reduce the risk for developing uterine cancer. Promoting awareness among women and health care providers of the need for timely evaluation of abnormal vaginal bleeding can increase the chance that uterine cancer is detected early and treated appropriately.

[§§§] https://www.cdc.gov/cancer/npcr/uscs/technical_notes/interpreting/race.htm.

Acknowledgments

State and regional cancer registry and health department personnel.

Corresponding author: S. Jane Henley, shenley@cdc.gov, 770-488-4157.

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

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

Summary

What is already known about this topic?

Uterine cancer is one of the few cancers with increasing incidence and mortality.

What is added by this report?

During 1999–2015 and 1999–2016, uterine cancer incidence and mortality rates increased 0.7% and 1.1% per year, respectively, with black women disproportionately affected.

What are the implications for public health practice?

Health care providers and community programs can help women achieve and maintain a healthy weight and get enough physical activity, which can reduce the risk for endometrial cancer, the most common uterine cancer. Promoting awareness of the need for timely evaluation of abnormal vaginal bleeding (between periods, after sex, or after menopause), an important symptom of uterine cancer, increases the chance for early detection and treatment.

References

1. Cronin KA, Lake AJ, Scott S, et al. Annual report to the nation on the status of cancer, part I: national cancer statistics. Cancer 2018;124:2785–800. https://doi.org/10.1002/cncr.31551
2. US Cancer Statistics Working Group. United States cancer statistics: 1999–2015 cancer incidence and mortality data. Atlanta, GA: US Department of Health and Human Services, CDC; 2018. https://www.cdc.gov/cancer/npcr/uscs/index.htm
3. Null DB, Weiland CM, Camlibel AR. Postmenopausal bleeding—first steps in the workup. J Fam Pract 2012;61:597–604.
4. Steele CB, Thomas CC, Henley SJ, et al. Vital signs: trends in incidence of cancers associated with overweight and obesity—United States, 2005–2014. MMWR Morb Mortal Wkly Rep 2017;66:1052–8. https://doi.org/10.15585/mmwr.mm6639e1
5. Cote ML, Ruterbusch JJ, Olson SH, Lu K, Ali-Fehmi R. The growing burden of endometrial cancer: a major racial disparity affecting black women. Cancer Epidemiol Biomarkers Prev 2015;24:1407–15. https://doi.org/10.1158/1055-9965.EPI-15-0316
6. Kurman R, Carcangiu M, Herrington S, Young R, editors. World Health Organization classification of tumours of female reproductive organs. Lyon, France: IARC Press; 2014.
7. Prat J, Mbatani N. Uterine sarcomas. Int J Gynaecol Obstet 2015;131(Suppl 2):S105–10. https://doi.org/10.1016/j.ijgo.2015.06.006
8. American College of Obstetrics and Gynecologists Committee on Gynecologic Practice. ACOG committee opinion no. 734 summary: the role of transvaginal ultrasonography in evaluating the endometrium of women with postmenopausal bleeding. Obstet Gynecol 2018;131:945–6. https://doi.org/10.1097/AOG.0000000000002626
9. American College of Obstetrics and Gynecologists Committee on Ethics. ACOG committee opinion no. 439: informed consent. Obstet Gynecol 2009;114:401–8. https://doi.org/10.1097/AOG.0b013e3181b48f7f
10. Siegel RL, Devesa SS, Cokkinides V, Ma J, Jemal A. State-level uterine corpus cancer incidence rates corrected for hysterectomy prevalence, 2004 to 2008. Cancer Epidemiol Biomarkers Prev 2013;22:25–31. https://doi.org/10.1158/1055-9965.EPI-12-0991

Public Health Response to an Avian Influenza A(H7N8) Virus Outbreak in Commercial Turkey Flocks — Indiana, 2016

Jennifer A. Brown, DVM[1]; Reema Patel, MPH[1]; Lynn Maitlen, MSN[2]; Donna Oeding[2]; Karen Gordon[1]; Joshua L. Clayton, PhD[1]; Shawn Richards[1]; Pam Pontones, MA[1]; James Brewer, MPA, MS[1]; Sara Blosser, PhD[1]; Joan Duwve, MD[1]

In January 2016, highly pathogenic avian influenza (HPAI) A(H7N8) virus and low pathogenicity avian influenza (LPAI) A(H7N8) virus were detected in commercial turkey flocks in Dubois County, Indiana. The Indiana State Department of Health (ISDH) and the Dubois County Health Department (DCHD) coordinated the public health response to this outbreak, which was the first detection of HPAI A(H7N8) in any species (*1*). This response was the first to fully implement unpublished public health monitoring procedures for HPAI responders that were developed by the U.S. Department of Agriculture (USDA) and CDC in 2015 (Sonja Olsen, CDC, personal communication, October 2017). No cases of zoonotic avian influenza infection in humans were detected during the outbreak.

Investigation and Results

On January 15, 2016, ISDH was notified by the Indiana State Board of Animal Health that HPAI A(H7N8) virus had been confirmed in a commercial turkey flock in Dubois County, Indiana. In accordance with USDA guidelines (*2*), the State Board of Animal Health promptly began active surveillance for HPAI in commercial poultry flocks within a radius of 6.2 miles (10 km) of the infected premises. By January 16, avian influenza H7 virus was detected in nine additional commercial turkey flocks; eight of these were confirmed as LPAI A(H7N8), and testing was inconclusive for one. Two additional poultry flocks were classified as dangerous contact premises because of their proximity to infected premises (*2*). The circulating HPAI and LPAI strains were suspected to be closely related; the State Board of Animal Health therefore elected to depopulate all 10 avian influenza H7-infected premises and both dangerous contact premises (a total of 414,223 birds). Depopulation was accomplished primarily by premises owners and industry representatives with the assistance of volunteer offenders from the Indiana Department of Correction (IDOC), whose participation was approved by the IDOC deputy commissioner. Offenders were compensated using the standard IDOC pay schedule and underwent medical clearance, N95 respirator fit testing, and job-specific training that included information about the zoonotic potential of avian influenza viruses. Depopulation was completed by January 16 for the index flock and by January 20 for the remaining flocks. The majority of poultry carcasses were disposed of by in-barn composting, and infected premises were cleaned and disinfected (*2*,*3*). Repopulation of all infected premises and dangerous contact premises was permitted as of May 1.

ISDH and DCHD recommended that responders be monitored during the response and for 10 days after their last possible exposures for influenza-like illness (ILI), defined as either 1) self-reported fever with cough or sore throat, or 2) conjunctivitis with or without additional symptoms. All responders received instructions to seek medical attention and contact public health authorities if they developed ILI during the 10-day period. USDA monitored federal employees, contractors, and subcontractors who participated in the response. ISDH and DCHD monitored state and local responders, using adapted, unpublished USDA/CDC public health monitoring procedures for HPAI A(H5) that were first circulated in September 2015 and later updated in November 2015. Responders were classified into three risk categories: 1) no risk, 2) low but not zero risk, and 3) some risk. Responders with low but not zero risk were those exposed to infected or potentially infected birds or their environments while using appropriate personal protective equipment (PPE) (*3*) as well as persons who were not exposed to birds or their environments but who worked or resided on infected premises; these persons were contacted by telephone on their first and last day of monitoring. Responders with some risk were those exposed to infected or potentially infected birds or their environments who did not use appropriate PPE or had a breach in PPE; these persons were actively monitored, with daily contact by visit, telephone call, text, or e-mail.

Although it is difficult to estimate the total number of responders, the number of daily on-site personnel peaked at 516 on day 4 of the response, most of whom were federal employees or contractors. DCHD, IDOC, and other local health departments conducted risk assessment and monitoring for 166 state and local responders. These included 93 farm workers or residents, 67 officers and offenders from state correctional facilities, and six local USDA employees who had completed their response activities. Among these 166 responders, 74 (45%) were monitored for some risk exposures, 67 (40%) were monitored for low but not zero risk exposures, four (2%) were monitored with no risk status recorded, seven (4%) had no exposure, five (3%) declined to be monitored, and nine (5%) were lost to follow-up. Among the 145 persons who were monitored, 14 (10%) reported current or recent ILI symptoms,

12 (86%) of whom were tested during January 16–22, 2016, with a median of 1 day from onset to medical evaluation. Nine patients had nasopharyngeal/oropharyngeal specimens collected, one had a conjunctival specimen collected, and two had both types of specimens collected. Specimens were tested for influenza A virus by real-time reverse transcription–polymerase chain reaction at the ISDH Laboratories; all 12 patients tested negative for influenza A virus.

Public Health Response

On January 15, 2016, Indiana activated its Emergency Operations Center with staffing consistent with the Federal Emergency Management Agency Emergency Support Functions (ESFs) appropriate to the response (ESF-1 = Transportation, ESF-5 = Emergency Management, ESF-8 = Public Health and Medical, ESF-10 = Oil and Hazardous Waste, ESF-11 = Food, Agriculture and Natural Resources, and ESF-13 = Law Enforcement).* The Indiana State Board of Animal Health was the lead state agency for this response, and ISDH provided public health and medical services support. DCHD led the local response, including monitoring for exposed county residents, in cooperation with Memorial Hospital in Jasper, Indiana. The State Incident Management Team was deployed to Dubois County to establish a unified command post in conjunction with USDA. ISDH deployed a field liaison to the unified command post to communicate with other state and local agencies. ISDH supported several missions from the state Emergency Operations Center, including distribution of N95 respirators and laboratory testing supplies and placement of the antiviral medication oseltamivir at the local hospital.

ISDH's major actions during the public health response included developing a demobilization packet for responders, issuing a Health Alert Network advisory to Dubois County and surrounding counties with recommendations for health care providers, establishing syndromic surveillance queries in the Indiana Public Health Emergency Surveillance System to detect community-acquired cases, and developing recommendations for the use of antivirals.

Discussion

Public health monitoring procedures for H7N8 responders were successfully implemented during this outbreak; no cases of zoonotic avian influenza infection were detected. The risk for zoonotic transmission in this outbreak was thought to be low at the time. No human infections with influenza A(H7N8) viruses had ever been reported,† and preliminary genetic analyses did not suggest enhanced virulence or transmission in mammals (*4*). However, other avian influenza H7 viruses have caused human infections, including severe respiratory illnesses (*5*,*6*), and human coinfection with an avian influenza A virus and a human influenza A virus presents a theoretical risk for emergence of a novel influenza A virus through genetic reassortment (*7*). The HPAI virus in this outbreak was suspected to have emerged as a result of spontaneous mutation in a circulating LPAI virus; this hypothesis was supported by later genetic analyses (*1*). A study conducted after the outbreak found that the HPAI virus exhibited enhanced virulence in mouse and ferret models, but that only the LPAI strain was transmissible; however, transmissibility in mammals and capacity to rapidly acquire increased virulence is a concerning combination of characteristics (*8*).

The unpublished USDA/CDC public health monitoring procedures that were adapted for use in this outbreak were developed for responders to an HPAI A(H5) outbreak. HPAI and LPAI are differentiated based on genetic features and the extent to which these viruses produce morbidity and mortality in poultry (*9*). The classification does not predict the probability of zoonotic transmission or severity of human illness (*6*); in fact, LPAI is capable of causing severe morbidity and mortality in humans (*10*). Given this and that many responders worked on both HPAI-infected and LPAI-infected premises, the same guidance was used for all H7N8-infected flocks.

The USDA/CDC public health monitoring plan is currently being updated (James Kile, CDC, personal communication, August 2018). The updated plan will allow for passive monitoring of persons wearing PPE and responding to certain influenza A H5 and H7 viruses that have caused outbreaks in the United States but have no history of causing human infections (e.g., the H7N8 virus described in this report). The updated plan will also cover all avian influenza viruses of public health concern, including both HPAI and LPAI viruses.

Several challenges to human health monitoring were identified during this outbreak. First, receipt of contact information for responders by the local health department was delayed in the initial stages because of the urgency and complexity of the animal health response. Complete information for exposed responders was not received by DCHD until 5 days into the response (on January 20), although preliminary information was provided earlier. Second, tears in Tyvek suits were reportedly common because of the nature of animal handling activities; this could have resulted in misclassification of disease exposure risk. Finally, mobilization of a large number of responders within a short period raised concerns that PPE and monitoring recommendations were not being implemented consistently.

Enhanced communication and information sharing among local, state, and federal agencies would improve identification of exposed persons and coordination of specimen collection,

* https://emilms.fema.gov/is230c/fem0104160text.htm.
† https://www.cdc.gov/flu/news/avian-influenza-h7n8-update.htm.

Summary

What is already known about this topic?

Prolonged or close contact with birds infected with avian influenza (AI) virus increases the risk for zoonotic infection in humans. Monitoring exposed persons for 10 days might facilitate early detection and reporting of zoonotic AI.

What is added by this report?

Monitoring procedures for highly pathogenic AI (HPAI) responders were successfully implemented during a 2016 outbreak of HPAI A(H7N8) in commercial turkey flocks in Indiana. No human cases of AI were identified.

What are the implications for public health practice?

Collaboration among local, state, and federal partners is essential during AI outbreak responses. Monitoring should be considered for all responders who had contact with infected birds or their environments, regardless of whether personal protective equipment was worn.

testing, and medical care for ill responders. This could be accomplished by 1) effectively communicating public health needs and recommendations to all stakeholders in the response; 2) identifying processes for early identification of exposed persons (e.g., badging systems); 3) designating a local/state/federal public health department liaison to be embedded in the unified command post to facilitate coordinated implementation of human health monitoring, including obtaining names and contact information for responders; and 4) conducting daily debriefings with safety officers in the incident command system to identify injuries or breaches in PPE that could elevate responder risk. In future outbreaks, ISDH will also recommend that responders to outbreaks of AI viruses of public health concern entering the exclusion zone (hot zone) and contamination reduction zone (warm zone) in infected premises (*2*) undergo active monitoring for 10 days after the last date of exposure, whether or not they were wearing appropriate PPE. This adjustment is expected to facilitate identification of personnel requiring monitoring and ensure that even responders with unrecognized or unreported breaches in PPE will be monitored.

Acknowledgments

Amanda Billman, Alexandra Boland, Lee Christenson, Stephanie Dearth, Mark Glazier, Zac Jones, Judy Lovchik, Megan Lytle, Craig Smith, Jill Stauffer, Taryn Stevens, Indiana State Department of Health; Maria Cooper, Kyle Shipman, Indiana State Board of Animal Health; James Basinger, Indiana Department of Correction; Sonja Olsen, Lenee Blanton, National Center for Immunization and Respiratory Diseases, CDC; Jane Rooney, Animal and Plant Health Inspection Service, U.S. Department of Agriculture.

Corresponding author: Jennifer A. Brown, jenbrown@isdh.in.gov, 317-233-7272.

[1]Indiana State Department of Health; [2]Dubois County Health Department, Jasper, Indiana.

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

References

1. Killian ML, Kim-Torchetti M, Hines N, Yingst S, DeLiberto T, Lee DH. Outbreak of H7N8 low pathogenic avian influenza in commercial turkeys with spontaneous mutation to highly pathogenic avian influenza. Genome Announc 2016;4:e00457-16. https://doi.org/10.1128/genomeA.00457-16
2. US Department of Agriculture. Highly pathogenic avian influenza response plan: the red book. Riverdale, MD: US Department of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services; 2017. https://www.aphis.usda.gov/animal_health/emergency_management/downloads/hpai_response_plan.pdf
3. US Department of Agriculture. Highly pathogenic avian influenza. Riverdale, MD: US Department of Agriculture, Animal and Plant Health Inspection Service; 2018. https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/emergency-management/fadprep-hpai
4. US Department of Agriculture. Epidemiologic and other analyses of Indiana HPAI/LPAI-affected poultry flocks: March 18, 2016 report. Fort Collins, CO: US Department of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services; 2016. https://www.aphis.usda.gov/animal_health/animal_dis_spec/poultry/downloads/Epidemiologic-Analysis-March-18-2016.pdf
5. Belser JA, Bridges CB, Katz JM, Tumpey TM. Past, present, and possible future human infection with influenza virus A subtype H7. Emerg Infect Dis 2009;15:859–65. https://doi.org/10.3201/eid1506.090072
6. Kile JC, Ren R, Liu L, et al. Update: increase in human infections with novel Asian lineage avian influenza A(H7N9) viruses during the fifth epidemic—China, October 1, 2016–August 7, 2017. MMWR Morb Mortal Wkly Rep 2017;66:928–32. https://doi.org/10.15585/mmwr.mm6635a2
7. Zhu Y, Qi X, Cui L, Zhou M, Wang H. Human co-infection with novel avian influenza A H7N9 and influenza A H3N2 viruses in Jiangsu province, China. Lancet 2013;381:2134. https://doi.org/10.1016/S0140-6736(13)61135-6
8. Sun X, Belser JA, Pulit-Penaloza JA, et al. Pathogenesis and transmission assessments of two H7N8 influenza A viruses recently isolated from turkey farms in Indiana using mouse and ferret models. J Virol 2016;90:10936–44. https://doi.org/10.1128/JVI.01646-16
9. Swayne DE, Pantin-Jackwood M. Pathogenicity of avian influenza viruses in poultry. Dev Biol (Basel) 2006;124:61–7.
10. Kang M, Lau EHY, Guan W, et al. Epidemiology of human infections with highly pathogenic avian influenza A(H7N9) virus in Guangdong, 2016 to 2017. Euro Surveill 2017;22:30568. https://doi.org/10.2807/1560-7917.ES.2017.22.27.30568

Exposure to Secondhand Smoke Among Nonsmokers — United States, 1988–2014

James Tsai, MD[1]; David M. Homa, PhD[1]; Andrea S. Gentzke, PhD[1]; Margaret Mahoney, JD[1]; Saida R. Sharapova, MD[1]; Connie S. Sosnoff, MS[2]; Kevin T. Caron[2]; Lanqing Wang, PhD[2]; Paul C. Melstrom, PhD[1]; Katrina F. Trivers, PhD[1]

Exposure to secondhand smoke from burning tobacco products can cause sudden infant death syndrome, respiratory infections, ear infections, and asthma attacks in infants and children, and coronary heart disease, stroke, and lung cancer in adult nonsmokers (*1*). There is no risk-free level of secondhand smoke exposure (*2*). CDC analyzed questionnaire and laboratory data from the National Health and Nutrition Examination Survey (NHANES) to assess patterns of secondhand smoke exposure among U.S. nonsmokers. The prevalence of secondhand smoke exposure among U.S. nonsmokers declined substantially during 1988–2014, from 87.5% to 25.2%. However, no change in exposure occurred between 2011–2012 and 2013–2014, and an estimated one in four nonsmokers, or approximately 58 million persons, were still exposed to secondhand smoke during 2013–2014. Moreover, marked disparities persisted across population groups. Exposure prevalence was highest among nonsmokers aged 3–11 years (37.9%), non-Hispanic blacks (50.3%), and those who were living in poverty (47.9%), in rental housing (38.6%), or with someone who smoked inside the home (73.0%), or among persons who had less than a high school education (30.7%). Comprehensive smoke-free laws and policies for workplaces and public places and smoke-free rules for homes and vehicles can further reduce secondhand smoke exposure among all nonsmokers.

NHANES is a program of studies designed to assess the health and nutritional status of children and adults in the United States (*3*). Participants are recruited using a household-based, multistage, stratified sampling scheme designed to represent the noninstitutionalized civilian U.S. population (*3*).* NHANES includes a home interview and physical examination at a mobile examination center where biologic specimens are collected for laboratory testing, including serum cotinine, an indicator of recent nicotine exposure (*4*,*5*).† Questionnaire and laboratory data were collected from participants (or their guardians) aged ≥4 years during NHANES III 1988–1994 and aged ≥3 years during biennial NHANES 1999–2014. Interview response rates ranged from 71.0% (2013–2014) to 86.0% (1988–1994). Response rates for mobile examination center samples ranged from 68.5% (2013–2014) to 80.0% (2001–2002) (*3*). An established standard range of serum cotinine of 0.05–10 ng/mL was used to define secondhand smoke exposure among nonsmokers and to allow for historical comparisons (*6*,*7*).§ Nonsmokers were defined as 1) children aged 4–11 years (NHANES III 1988–1994) and children aged 3–11 years (NHANES 1999–2014) with serum cotinine ≤10 ng/mL; 2) adolescents aged 12–19 years with serum cotinine ≤10 ng/mL and who did not report smoking within the preceding 30 days or use of any nicotine-containing product within the preceding 5 days at mobile examination center interview; and 3) adults aged ≥20 years with serum cotinine ≤10 ng/mL and who did not report being a current smoker during household interview or use of any nicotine-containing product within the preceding 5 days at mobile examination center interview.¶

To assess prevalence of secondhand smoke exposure during 1988–2014, the percentage of persons with serum cotinine levels 0.05–10 ng/mL for each survey cycle was calculated among nonsmokers overall by age group (3–11, 12–19, and ≥20 years), and, among children aged 3–11 years, by race and Hispanic origin** (non-Hispanic white, non-Hispanic black,

* Stand-alone NHANES were conducted in 1976–1980 and 1988–1994 (Phase 1: 1988–1991; Phase 2: 1991–1994); since 1999, NHANES has been a continuous study conducted for 2-year periods. https://www.cdc.gov/nchs/nhanes/index.htm.

† Serum cotinine is based on blood samples collected by venipuncture. Since 1988, data are collected and laboratory analyses are performed using an isotope dilution liquid chromatography tandem mass spectrometry method. Laboratory analytic and quality assurance and quality control procedures are described in detail. https://wwwn.cdc.gov/nchs/data/nhanes/2013-2014/manuals/2013_MEC_Laboratory_Procedures_Manual.pdf.

§ During 1988–2000, the laboratory limit of detection for serum cotinine was 0.05 ng/mL. Since 2001–2002, the laboratory limit of detection for serum cotinine has been 0.015 ng/mL.

¶ Based on response to the question "During the past 5 days, did (you/he/she) use any product containing nicotine including cigarettes, pipes, cigars, chewing tobacco, snuff, nicotine patches, nicotine gum, or any other product containing nicotine?" For 2013–2014, questions assessed included "During the past 5 days, including today, did you smoke cigarettes, pipes, cigars, little cigars or cigarillos, water pipes, hookahs, or e-cigarettes?" "During the past 5 days, including today, did (you/he/she) use any smokeless tobacco (chewing tobacco, snuff, snus, or dissolvables)?" and "During the past 5 days, including today, did you use any nicotine replacement therapy products such as nicotine patches, gum, lozenges, inhalers, or nasal sprays?" Serum cotinine concentrations >10 ng/mL are associated with recent active smoking.

** Because of the NHANES sample design, only data for non-Hispanic whites, non-Hispanic blacks, and Mexican Americans are available across all survey cycles. However, all racial and ethnic groups are included in the reported estimates for the total population and subgroups by sex, age, poverty, education, housing, and status of living with anyone who smoked inside the home.

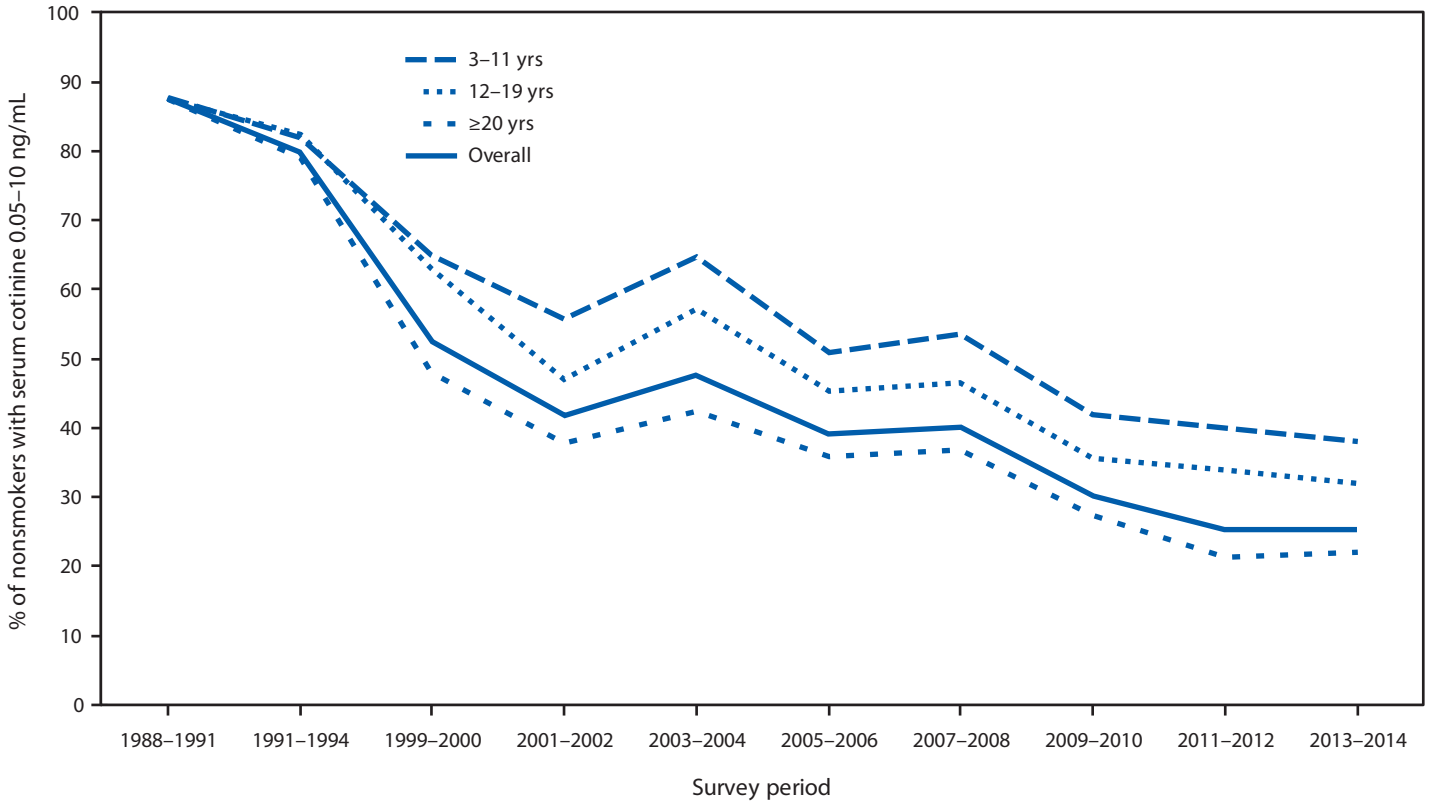
or Mexican American). During 2013–2014, percentages and 95% confidence intervals of secondhand smoke exposure were computed among nonsmokers by age, sex, race and Hispanic origin, poverty, education, housing status, and whether the participant lived with someone who smoked inside the home. Percentage differences within each subgroup were assessed using chi-squared tests, with statistical significance defined as $p<0.05$. Estimated numbers of persons exposed to secondhand smoke during 2013–2014 were calculated according to population estimates from the American Community Survey.[††] Data were weighted using examination sample weights to account for the complex survey design and differential probability of sample selection, nonresponse, and noncoverage.

From 1988–1991 to 2013–2014, the prevalence of secondhand smoke exposure declined 71.2% among U.S. nonsmokers, from 87.5% to 25.2%. Secondhand smoke exposure declined from 87.8% to 37.9% among children aged 3–11 years (56.8% decrease), from 87.4% to 32.0% among adolescents aged 12–19 years (63.4% decrease), and from 87.4% to 22.0% among adults aged ≥20 years (74.8% decrease) (Figure 1). Among nonsmokers aged 3–11 years, secondhand smoke exposure declined from 86.4% to 37.8% among non-Hispanic whites (56.3% decrease), from 94.5% to 66.1% among non-Hispanic blacks (30.1% decrease), and from 84.4% to 22.2% among Mexican Americans (73.7% decrease) (Figure 2). From 2011–2012 to 2013–2014, no statistically significant change occurred in the prevalence of secondhand smoke exposure among U.S. nonsmokers.

During 2013–2014, the prevalence of secondhand smoke exposure was significantly higher among children aged 3–11 years (37.9%) than among adults aged ≥20 years (22.0%) (Table), among non-Hispanic blacks (50.3%) than among non-Hispanic whites (21.4%) and Mexican Americans (20.0%), and among persons living below the poverty level (47.9%) compared with those living at or above the poverty level (21.2%). By education, among persons aged ≥25 years, the prevalence of secondhand smoke exposure was highest among those with less than a high school education (30.7%) and lowest among those with a college degree or higher (10.8%). The prevalence of secondhand smoke exposure was significantly higher among persons who rented (38.6%) than among those

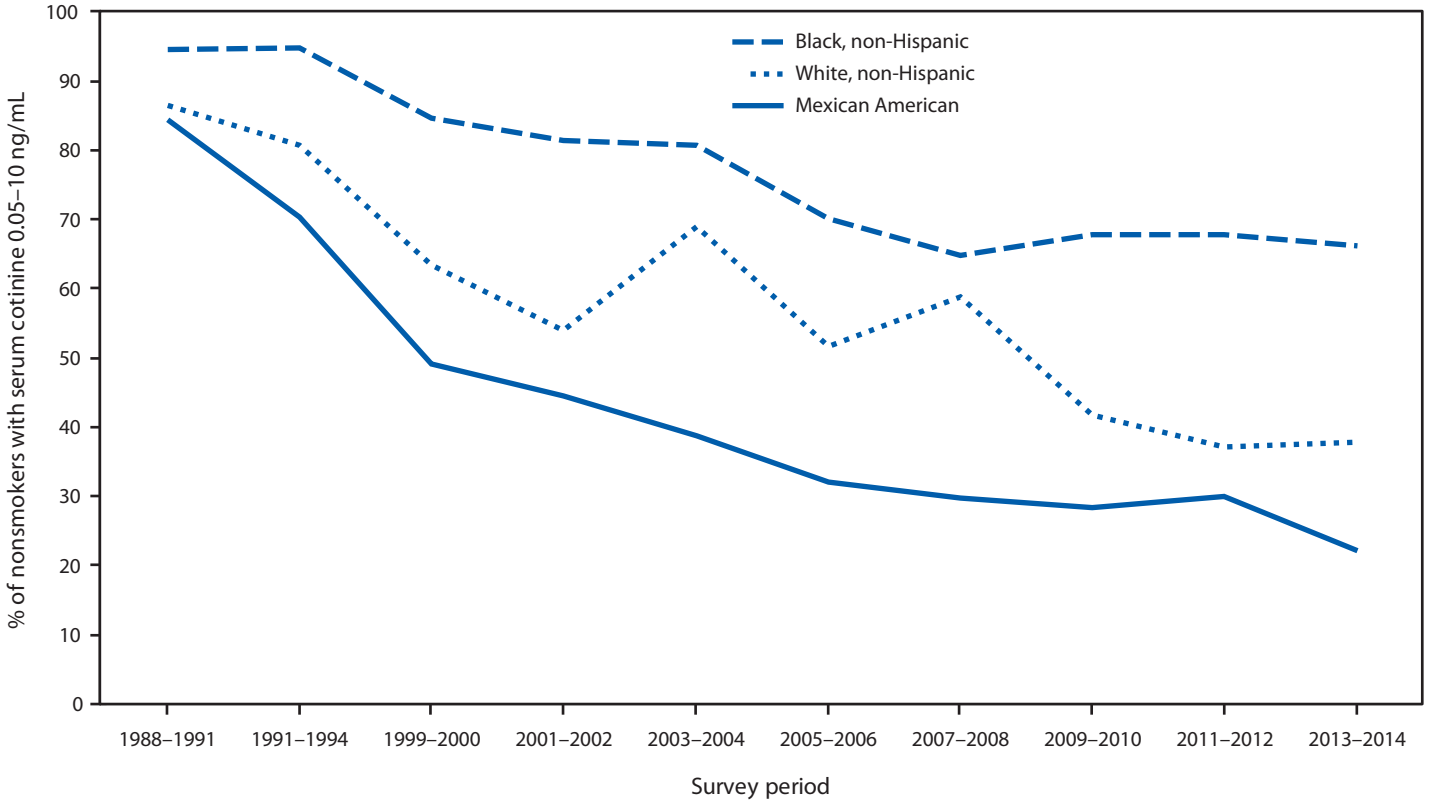
[††] https://wwwn.cdc.gov/nchs/data/nhanes3/ResponseRates/2013-2014_ACS_Control_Total.xlsx.

FIGURE 1. Percentage of nonsmokers aged ≥3 years* with evidence of secondhand smoke exposure (serum cotinine levels 0.05–10 ng/mL), by age group — National Health and Nutrition Examination Survey (NHANES), United States, 1988–2014



* Nonsmokers aged ≥4 years for NHANES III 1988–1994.

FIGURE 2. Percentage of nonsmokers aged 3–11 years* with evidence of secondhand smoke exposure (serum cotinine levels 0.05–10 ng/mL), by race and Hispanic origin† — National Health and Nutrition Examination Survey (NHANES), United States, 1988–2014



* Nonsmokers aged ≥4 years for NHANES III 1988–1994.
† Because of sample design, racial and Hispanic origin categories were limited to non-Hispanic whites, non-Hispanic blacks, and Mexican Americans across all survey cycles.

who owned their homes (19.2%). In addition, the prevalence among persons who lived with anyone who smoked inside the home (73.0%) was significantly higher than it was among those who did not (22.3%).

Among the estimated 58.0 million nonsmokers who were exposed to secondhand smoke during 2013–2014, approximately 36.7 million were adults, 9.1 million were adolescents, and 14.0 million were children.§§ This includes 6.8 million non-Hispanic whites, 3.3 million non-Hispanic blacks, and 1.5 million Mexican Americans.¶¶

Discussion

Although secondhand smoke exposure among U.S. nonsmokers declined from 87.5% to 25.2% during 1988–2014, progress has stalled in recent years. One in four nonsmokers were still exposed to secondhand smoke during 2013–2014, and disparities in exposure prevalence persisted across demographic groups. Prevalence remained highest among children aged 3–11 years, non-Hispanic blacks, and persons living in poverty, in rental housing, and with someone who smoked inside the home. Enhanced and equitable implementation of comprehensive smoke-free laws and policies for workplaces and public places and smoke-free rules for homes and vehicles can further reduce secondhand smoke exposure among all nonsmokers (*2*).***

The decline in secondhand smoke exposure among U.S. nonsmokers is likely due to decreasing cigarette smoking rates, increased awareness of the risks for secondhand smoke exposure, and the adoption of comprehensive smoke-free laws prohibiting smoking in workplaces and public places in many states and localities (*1*,*8*,*9*). During 2011–2014, the percentage of nonsmokers exposed to secondhand smoke did not decline significantly across most demographic subgroups (*6*). This lack of decline could be attributable to slowed adoption of statewide comprehensive smoke-free laws during this period (*10*).

§§ Numbers do not sum to total because of rounding.
¶¶ Children of all racial ethnic groups are included in the total population estimate.

*** https://www.thecommunityguide.org/search/secondhand.

Summary

What is already known about this topic?

Exposure to secondhand tobacco smoke can cause sudden infant death syndrome, respiratory infections, ear infections, and asthma attacks in infants and children, and coronary heart disease, stroke, and lung cancer in adult nonsmokers.

What is added by this report?

Although secondhand smoke exposure among U.S. nonsmokers declined from 87.5% to 25.2% during 1988–2014, one in four nonsmokers, including 14 million children, were exposed to secondhand smoke during 2013–2014.

What are the implications for public health practice?

Continued measures to implement comprehensive smoke-free laws in workplaces and public places, adoption of smoke-free home and vehicle rules, and educational interventions warning about the risks for secondhand smoke exposure can further reduce secondhand smoke exposure.

Nonetheless, to date, 27 states and the District of Columbia have comprehensive smoke-free laws, and progress in smoke-free law adoption has occurred at the local level in more recent years.[†††] Moreover, during 2015–2017, 199 localities adopted comprehensive smoke-free laws, and 21 additional localities have implemented such laws as of July 2018.[§§§] In addition, the U.S. Department of Housing and Urban Development adopted a rule requiring most public housing to be smoke-free by July 31, 2018, and Alaska adopted a statewide law in 2018 prohibiting smoking in workplaces and public places, although localities can opt out.[¶¶¶]

Disparities in secondhand smoke exposure persisted, with higher exposure among children aged 3–11 years (37.9%) and non-Hispanic blacks (50.3%) than among other age or racial and Hispanic origin subgroups. Variations in smoking prevalence, smoke-free policy coverage, and knowledge about the harms of secondhand smoke might have contributed to these disparities. These findings underscore the importance of continued measures to enhance smoke-free policy coverage, including educating parents and caregivers about the benefits of voluntarily prohibiting smoking in their homes and vehicles. These steps can reduce secondhand smoke exposure across all population groups, particularly those with the greatest exposure prevalence.

The findings in this report are subject to at least five limitations. First, smoking status was based on self-report and could be subject to social desirability and reporting biases. Some smokers might misrepresent their smoking status in surveys. Second, serum cotinine levels reflect recent exposure; thus, exposure misclassification might have occurred. Third, an established standard range of serum cotinine was used to define secondhand smoke exposure, which allowed historical comparisons. However, secondhand smoke exposure below this cutpoint might not have been measured. Fourth, serum cotinine might reflect secondhand exposure to other tobacco products such as e-cigarettes, which was not assessed in the survey. Finally, sample design limited the racial and Hispanic populations that could be assessed.

Although secondhand smoke exposure among U.S. nonsmokers has decreased considerably during the past two and a half decades, progress has stalled in recent years, and approximately one in four nonsmokers remains exposed to this preventable health hazard. In addition, disparities persist: 14.0 million children aged 3–11 years, including two of every three non-Hispanic black children, were still exposed during

TABLE. Percentage of nonsmokers aged ≥3 years with serum cotinine levels 0.05–10 ng/mL, by selected sociodemographic characteristics — National Health and Nutrition Examination Survey, United States, 2013–2014

Characteristic	% (95% CI)
Overall	25.2 (21.1–29.8)
Sex	
Male	27.1 (23.0–31.6)
Female	23.6 (19.0–28.9)
Age group (yrs)	
3–11	37.9 (31.2–45.0)
12–19	32.0 (24.9–39.9)
≥20	22.0 (18.4–26.1)
Race and Hispanic origin*	
White, non-Hispanic	21.4 (16.1–27.8)
Black, non-Hispanic	50.3 (44.8–55.8)
Mexican American	20.0 (16.1–24.6)
Poverty status	
Below poverty level[†]	47.9 (42.2–53.7)
At or above poverty level	21.2 (17.4–25.7)
Unspecified	23.3 (17.6–30.1)
Education[§]	
Less than high school diploma	30.7 (25.4–36.5)
High school diploma or equivalent	28.8 (21.7–37.0)
Some college or associate's degree	23.5 (19.2–28.5)
Bachelor's degree or higher	10.8 (8.1–14.3)
Housing	
Own	19.2 (15.0–24.3)
Rent	38.6 (33.9–43.5)
Other arrangement	35.9 (22.1–52.5)
Living with anyone who smoked inside the home	
Yes	73.0 (59.2–83.4)
No	22.3 (18.7–26.5)

Abbreviation: CI = confidence interval.
* Data by race and Hispanic origin were limited to the three racial and Hispanic origin groups available across all survey cycles (non-Hispanic white, non-Hispanic black, and Mexican American).
† Income-to-poverty ratio <1.0.
§ Assessed for persons aged ≥25 years.

††† https://chronicdata.cdc.gov/Legislation/STATE-System-Smokefree-Indoor-Air-Fact-Sheet/vgq2-kkcg.
§§§ https://no-smoke.org/wp-content/uploads/pdf/EffectivePopulationList.pdf.
¶¶¶ https://www.gpo.gov/fdsys/pkg/FR-2016-12-05/pdf/2016-28986.pdf; http://www.akleg.gov/basis/Bill/Text/30?Hsid=SB0063F.

2013–2014. Continued measures to implement comprehensive smoke-free laws in workplaces and public places, adoption of smoke-free home and vehicle rules, and educational interventions warning about the risks for secondhand smoke exposure can further reduce secondhand smoke exposure, especially among vulnerable populations.

Corresponding author: James Tsai, jxt9@cdc.gov, 770-488-5493.

[1]Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, CDC; [2]Division of Laboratory Sciences, National Center for Environmental Health, CDC.

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

References

1. US Department of Health and Human Services. The health consequences of smoking—50 years of progress: a report of the Surgeon General. Washington, DC: US Department of Health and Human Services; 2014. http://www.surgeongeneral.gov/library/reports/50-years-of-progress/full-report.pdf
2. US Department of Health and Human Services. The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General. Washington, DC: US Department of Health and Human Services; 2006. https://www.surgeongeneral.gov/library/reports/secondhandsmoke/fullreport.pdf
3. CDC. National Health and Nutrition Examination Survey. Hyattsville, MD: US Department of Health and Human Services, CDC; 2018. https://www.cdc.gov/nchs/nhanes/index.htm
4. CDC. Fourth national report on human exposure to environmental chemicals. Atlanta, GA: US Department of Health and Human Services, CDC; 2015. https://www.cdc.gov/biomonitoring/pdf/FourthReport_UpdatedTables_Feb2015.pdf
5. CDC, National Center for Health Statistics. NHANES 2013–2014 laboratory methods. Hyattsville, MD: US Department of Health and Human Services, CDC, National Center for Health Statistics; 2013. https://wwwn.cdc.gov/nchs/nhanes/continuousnhanes/labmethods.aspx?BeginYear=2013
6. Homa DM, Neff LJ, King BA, et al. Vital signs: disparities in nonsmokers' exposure to secondhand smoke—United States, 1999–2012. MMWR Morb Mortal Wkly Rep 2015;64:103–8.
7. Max W, Sung HY, Shi Y. Deaths from secondhand smoke exposure in the United States: economic implications. Am J Public Health 2012;102:2173–80. https://doi.org/10.2105/AJPH.2012.300805
8. American Nonsmokers' Rights Foundation. Summary of 100% smokefree state laws and population protected by 100% U.S. smokefree laws. Berkeley, CA: American Nonsmokers' Rights Foundation; 2018. http://no-smoke.org/wp-content/uploads/pdf/SummaryUSPopList.pdf
9. CDC. Best practices for comprehensive tobacco control programs—2014. Atlanta, GA: US Department of Health and Human Services, CDC; 2014. https://www.cdc.gov/tobacco/stateandcommunity/best_practices/index.htm
10. Holmes CB, King BA, Babb SD. Stuck in neutral: stalled progress in statewide comprehensive smoke-free laws and cigarette excise taxes, United States, 2000–2014. Prev Chronic Dis 2016;13:150409. https://doi.org/10.5888/pcd13.150409

Outbreak of Epidemic Keratoconjunctivitis Caused by Human Adenovirus Type D53 in an Eye Care Clinic — Los Angeles County, 2017

Kelsey OYong, MPH[1]; Marie Killerby, VetMB[2]; Chao-Yang Pan, MPH[3]; Thalia Huynh[3]; Nicole M. Green, PhD[4]; Debra A. Wadford, PhD[3]; Dawn Terashita, MD[1]

On June 22, 2017, the Los Angeles County Department of Public Health (LAC DPH) was notified of seven patients who were seen at an eye care clinic on June 8, 2017, and later developed symptoms of epidemic keratoconjunctivitis (EKC). EKC is a contagious, severe form of viral conjunctivitis that can cause pain and blurred vision for up to 4 weeks (*1*). LAC DPH conducted an investigation, which identified 17 patients with EKC, including 15 who had visited the optometry clinic and two who were household contacts of clinic patients. Observations in the clinic found deficiencies in disinfection of tonometers (an instrument connected to a slit lamp and used to test for glaucoma by measuring intraocular pressure) and multiuse eye drop administration. Staff member education and revision of disinfection practices interrupted further transmission. Patient specimens tested positive for human adenovirus (HAdV) type D53 (HAdV-53). As the first documented EKC outbreak associated with HAdV-D53 in the United States, this outbreak highlights the need for rigorous implementation of recommended infection prevention practices in eye care settings.

Investigation and Results

On June 22, 2017, hospital A reported a cluster of seven patients with EKC who had been seen at an affiliated optometry clinic to LAC DPH. Staff members who provide care at the clinic include three optometrists, one ophthalmologist, and three optometric assistants. The clinic has three exam rooms and sees an average of 1,300 patients each month. LAC DPH subsequently began an investigation into the cluster.

A case was defined as 1) diagnosis of EKC, adenoviral conjunctivitis, or viral conjunctivitis by an ophthalmologist or optometrist; or 2) laboratory confirmation of HAdV from a specimen collected by conjunctival swab in a person seen at the optometry clinic during June 5–July 3, 2017. A health care–linked case was defined as a case of EKC in a person who had visited the optometry clinic during June 7–July 3, 2017, and had symptom onset within 21 days of their visit. A household case was defined as an EKC case in a household or family contact of a patient with EKC.

All patients with EKC were symptomatic and self-referred to a health care provider. Review of optometry clinic medical records and telephone calls to patients did not identify additional cases. Among the 17 patients with EKC, 15 met the health care–linked case definition, including patient A, who appeared to be the source of introduction into the clinic (Figure). Two additional patients met the household case definition; both reported that a spouse was symptomatic before their own illness onset.

The median patient age was 62 years (range = 43–78 years), and 12 patients were women. No hospitalizations resulted from infection, although seven patients had more than one visit to a clinic, a hospital emergency department, or an urgent care center for symptoms. Patients had symptoms consistent with EKC, including eye redness (14) and discharge (13). The mean incubation period was 9 days (range = 5–19 days).

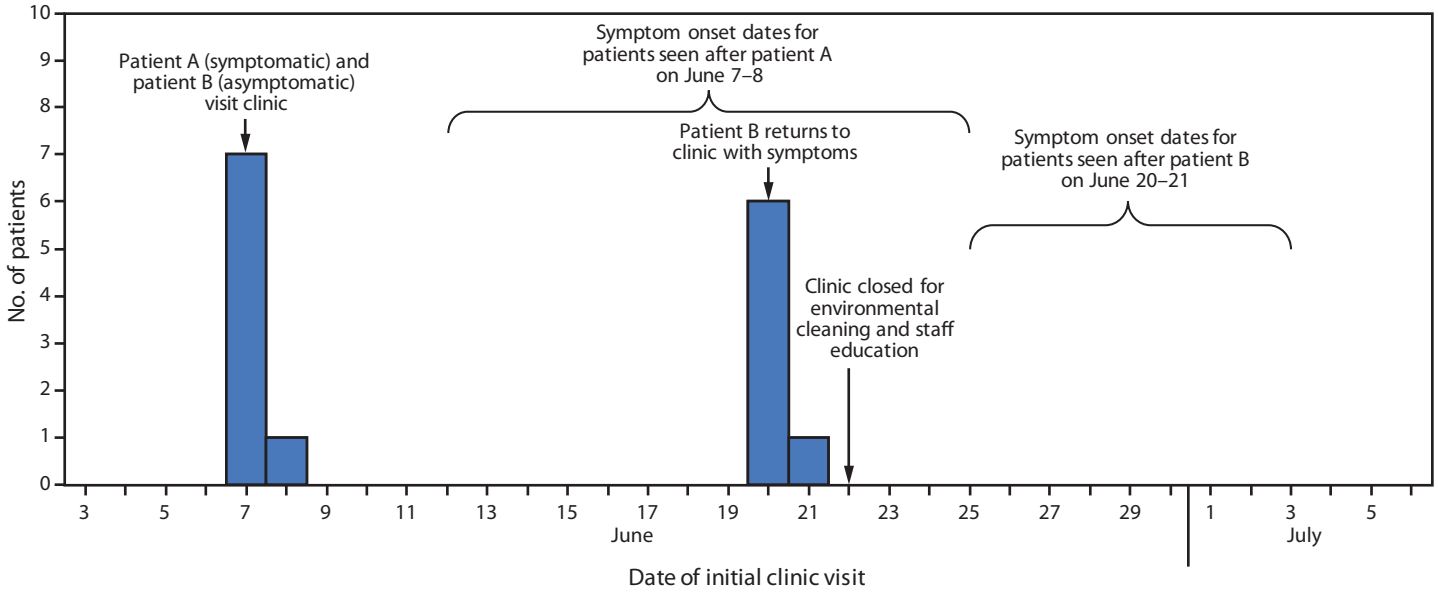
Review of health care–linked patient clinic visit dates preceding symptom onset revealed two apparent clusters. Patient A visited the clinic on June 7 with symptoms consistent with EKC, before the initial visits of seven additional patients on June 7 and June 8; these patients' EKC symptoms began during June 12–25. On June 20, a patient who went to the clinic on June 7 (patient B) returned to the clinic with EKC symptoms that had begun on June 14. Another seven patients visited the clinic after patient B on June 20 and June 21, before the onset of their EKC symptoms (June 26–July 3), consistent with transmission to these additional seven patients.

Medical chart review indicated common exposures among the 14 health care–linked patients: all were examined by the same optometrist in the same exam room after either patient A (June 7) or patient B (June 20) had been seen. No health care personnel reported EKC symptoms before or during the outbreak period. Among the 14 patients, other exposures included slit lamp contact (13), tonometry (12), and receipt of dilating eye drops from a multidose container (10). Use of multidose sodium fluorescein eye drops was reported for six patients in the first cluster and none in the second. During patient A's initial clinic visit on June 7, sodium fluorescein drops from a multiuse vial were administered, and a slit lamp examination was performed.

The clinic closed on June 22 for intensive environmental cleaning of clinic surfaces and equipment, instrument cleaning and disinfection, and to provide training to staff members on infection prevention. The clinic reopened the following day.

On June 23, LAC DPH conducted an announced site visit to inspect the premises, observe infection prevention practices, interview staff members, and review infection prevention

FIGURE. Health care–linked cases of epidemic keratoconjunctivitis (N = 15), by date of initial eye care clinic visit — Los Angeles County, California, June–July 2017



policies. Clinic patients typically proceed from the waiting area to one of three exam rooms, each with its own slit lamp with tonometer. Observations and staff member interviews indicated gaps in infection prevention practices, including use of eye drops from multidose vials on multiple patients, occasionally touching the eye or surrounding area, and reprocessing of tonometers using a 70% isopropyl alcohol wipe rather than the recommended 5–10-minute disinfecting soak with chlorine or ethyl alcohol.*

Conjunctival swab specimens from four symptomatic patients were sent to the LAC Public Health Laboratory for conventional and shell vial culture (used for adenovirus detection) (*2*) and detection by fluorescent monoclonal antibody staining; adenovirus was detected in two specimens. Specimens from an additional 11 patients were tested at the laboratory of hospital A, and adenovirus was identified in six by viral culture.

Specimens from the eight patients with positive adenovirus cultures were then submitted to the California Department of Public Health Viral and Rickettsial Disease Laboratory (VRDL) for HAdV detection and molecular typing by sequence analysis of the hypervariable region of the HAdV hexon gene and the HAdV group-specific region of the fiber gene (*3*,*4*). All eight patient specimens were positive for HAdV-D53. Subsequently, VRDL generated HAdV-D53 whole genome sequences from one patient specimen, which was nearly identical to a recently reported whole genome sequence of HAdV-D53 from Japan (GenBank sequence LC215428).

* https://www.cdc.gov/infectioncontrol/pdf/guidelines/disinfection-guidelines.pdf.

Discussion

HAdV-D53 has been recognized as an agent of EKC outbreaks in Japan since 1980 (*5*–*7*) and in Germany since 2005 (*8*). However, HAdV-D53 has not previously been reported to the U.S. National Adenovirus Type Reporting System, and this is the first reported EKC outbreak associated with HAdV-D53 in the United States.

Based on this investigation, it is believed that the virus was introduced to surfaces in the exam room by a symptomatic patient, and that subsequent lapses in infection prevention practices led to transmission to other patients. Previous studies have demonstrated that adenoviruses can persist on environmental surfaces for several weeks (*9*). Enhanced infection prevention practices, including staff member education on eye drop administration and longer slit lamp and tonometer disinfection times were implemented. No further cases were reported after July 3, 2017.

Previous similar EKC outbreaks have been linked to eye care clinics employing improper disinfection practices and lapses in hygienic protocols (*10*). To prevent EKC transmission in eye care settings, recommended practices include the use of disposable tonometer tips, disinfectants efficacious against adenoviruses for tonometers and slit lamps, and single-use eye drops when available. Use of recommended infection prevention practices is necessary to avoid EKC and other health care–associated infections.

Summary

What is already known about this topic?

Epidemic keratoconjunctivitis (EKC) associated with adenovirus is a frequent cause of outbreaks in eye care settings. Previous outbreaks have been associated with lapses in infection prevention.

What is added by this report?

This report details the first documented outbreak of adenovirus D53 EKC in the United States. Seventeen EKC cases were identified; after the primary case, all cases occurred in eye care clinic patients or their household contacts. Infection prevention lapses were associated with the outbreak, specifically improper ocular equipment disinfection.

What are the implications for public health practice?

By understanding the associated causes for transmission, health care practitioners and public health officials can target resources to ensure proper infection prevention practice.

Acknowledgments

Virology Section staff members, Los Angeles County Department of Public Health, Public Health Laboratory.

Corresponding author: Kelsey OYong, koyong@ph.lacounty.gov, 213-240-7941.

[1]Acute Communicable Disease Control Program, Los Angeles County Department of Public Health, Los Angeles, California; [2]National Center for Immunization and Respiratory Diseases, Division of Viral Diseases, CDC; [3]Viral and Rickettsial Disease Laboratory, California Department of Public Health, Richmond, California; [4] Public Health Laboratory, Los Angeles County Department of Public Health, Los Angeles, California.

All authors have completed and submitted the ICMJE form for disclosure of potential conflicts of interest. Nicole Green reports ownership of stock in Abbot Laboratories and Illumina. No other potential conflicts of interest were disclosed.

References

1. Lu X, Joshi A, Flomenberg P. Adenoviruses. In: Kaslow RA, Stanberry LR, LeDuc JW, eds. Viral infections of humans. New York, NY: Springer; 2014:99–121.
2. Espy MJ, Hierholzer JC, Smith TF. The effect of centrifugation on the rapid detection of adenovirus in shell vials. Am J Clin Pathol 1987;88:358–60. https://doi.org/10.1093/ajcp/88.3.358
3. McCarthy T, Lebeck MG, Capuano AW, Schnurr DP, Gray GC. Molecular typing of clinical adenovirus specimens by an algorithm which permits detection of adenovirus coinfections and intermediate adenovirus strains. J Clin Virol 2009;46:80–4. https://doi.org/10.1016/j.jcv.2009.06.008
4. Lu X, Erdman DD. Molecular typing of human adenoviruses by PCR and sequencing of a partial region of the hexon gene. Arch Virol 2006;151:1587–602. https://doi.org/10.1007/s00705-005-0722-7
5. Engelmann I, Madisch I, Pommer H, Heim A. An outbreak of epidemic keratoconjunctivitis caused by a new intermediate adenovirus 22/H8 identified by molecular typing. Clin Infect Dis 2006;43:e64–6. https://doi.org/10.1086/507533
6. Aoki K, Ishiko H, Konno T, et al. Epidemic keratoconjunctivitis due to the novel hexon-chimeric-intermediate 22,37/H8 human adenovirus. J Clin Microbiol 2008;46:3259–69. https://doi.org/10.1128/JCM.02354-07
7. Kaneko H, Aoki K, Ishida S, et al. Recombination analysis of intermediate human adenovirus type 53 in Japan by complete genome sequence. J Gen Virol 2011;92:1251–9. https://doi.org/10.1099/vir.0.030361-0
8. Binder AM, Biggs HM, Haynes AK, et al. Human adenovirus surveillance—United States, 2003–2016. MMWR Morb Mortal Wkly Rep 2017;66:1039–42. https://doi.org/10.15585/mmwr.mm6639a2
9. Killerby ME, Stuckey MJ, Guendel I, et al. Notes from the field: epidemic keratoconjunctivitis outbreak associated with human adenovirus type 8—U.S. Virgin Islands, June–November 2016. MMWR Morb Mortal Wkly Rep 2017;66:811–2. https://doi.org/10.15585/mmwr.mm6630a3
10. King D, Johnson B, Miller D, et al. Adenovirus-associated epidemic keratoconjunctivitis outbreaks—four states, 2008–2010. MMWR Morb Mortal Wkly Rep 2013;62:637–41.

Notes from the Field:

An Outbreak of *Salmonella* Agbeni Infections Linked to Turtle Exposure — United States, 2017

Lia Koski, MPH[1,2]; Lauren Stevenson, MHS[1,3]; Jasmine Huffman[1]; Amy Robbins, MPH[4]; Julia Latash, MPH[5,6]; Enoma Omoregie, PhD[5]; Kelly Kline, MPH[7]; Megin Nichols, DVM[1]

In June 2017, PulseNet, the national molecular subtyping network for foodborne disease surveillance, identified 17 *Salmonella* Agbeni clinical isolates with indistinguishable *Xba*I enzyme pattern (outbreak strain) by pulsed-field gel electrophoresis. The same *Salmonella* Agbeni *Xba*I pattern was isolated from a turtle in 2015; in a 2016 investigation involving the same outbreak strain, 63% of patients reported contact with turtles (CDC, unpublished data, 2016). Despite prohibition of sale of small turtles (shell length less <4 inches) in the United States since 1975 (*1*), illness outbreaks associated with turtle contact continue to occur. Ill persons in previous *Salmonella* Poona and *Salmonella* Pomona outbreaks linked to turtles were geographically concentrated in the Southwest region of the United States (*2*,*3*). Turtle production is known to be higher in the Southeast region of the country (*2*). An outbreak investigation by CDC and health departments was initiated to identify the source of the 2017 illness outbreak.

A case was defined as isolation of *Salmonella* Agbeni with the outbreak strain from an ill patient during April–December 2017. State and local health officials interviewed patients to ascertain turtle exposure information, including details about the species of turtle and purchasing information. Purchase locations reported by patients were contacted for traceback information. Whole genome sequencing (WGS), using high quality single nucleotide polymorphism (hqSNP) analysis, was performed by CDC on clinical isolates from the 2017 outbreak, the 2016 illness cluster, and the turtle isolate from 2015 to characterize genetic relatedness.

Seventy-six cases were identified in 19 states in 2017; two thirds (67%) of patients resided in East Coast states (Connecticut, Delaware, Maryland, Massachusetts, New Jersey, New York, Pennsylvania, Rhode Island, and Virginia).* Patient ages ranged from <1–100 years (median = 21 years). Among 63 (83%) patients with information on hospitalization, 30 (48%) were hospitalized; no deaths were reported. Fifty-nine (78%) patients provided exposure information, including 23 (39%) who reported contact with turtles; among these, 14 (61%) specified small turtles. Among 12 patients who reported how the turtles were obtained, six purchased them from a street or roadside vendor, three purchased them from a retail store, two purchased them at festivals, and one reported receiving them as a gift. The traceback investigation did not identify a common turtle farm that supplied purchase locations. WGS hqSNP analysis indicated that the 2017 and 2016 clinical isolates and the 2015 turtle isolate were closely related, differing by 0–18 SNPs.

This salmonellosis outbreak was linked to contact with small turtles and was associated with a higher frequency of hospitalization (48%) than multistate foodborne pathogen outbreaks (27%) as well as recent *Salmonella* outbreaks linked to turtles (28%–33%) (*2*–*4*). The geographic distribution of patients differed from that of previous outbreaks, suggesting the need to better understand the breeding of turtles and distribution of turtle sales in the United States. WGS hqSNP analysis was used to link historic illnesses and turtle isolates to isolates from 2017 patients, supporting the hypothesis that turtles were the likely source of this outbreak. This outbreak indicates further need to educate consumers and retail store staff members regarding the ban on sale of small turtles and to educate consumers to prevent transmission of *Salmonella* from pets to humans.

Corresponding author: Lia Koski, lkoski@cdc.gov, 404-718-5887.

[1]Division of Foodborne, Waterborne, and Environmental Diseases, National Center for Emerging and Zoonotic Infectious Diseases, CDC; [2]Oak Ridge Institute for Science and Education, Oak Ridge, Tennessee; [3]CAITTA, Inc., Herndon, Virginia; [4]New York State Department of Health; [5]New York City Department of Health and Mental Hygiene; [6]CDC/CSTE Applied Epidemiology Fellowship; [7]Pennsylvania Department of Health.

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

References

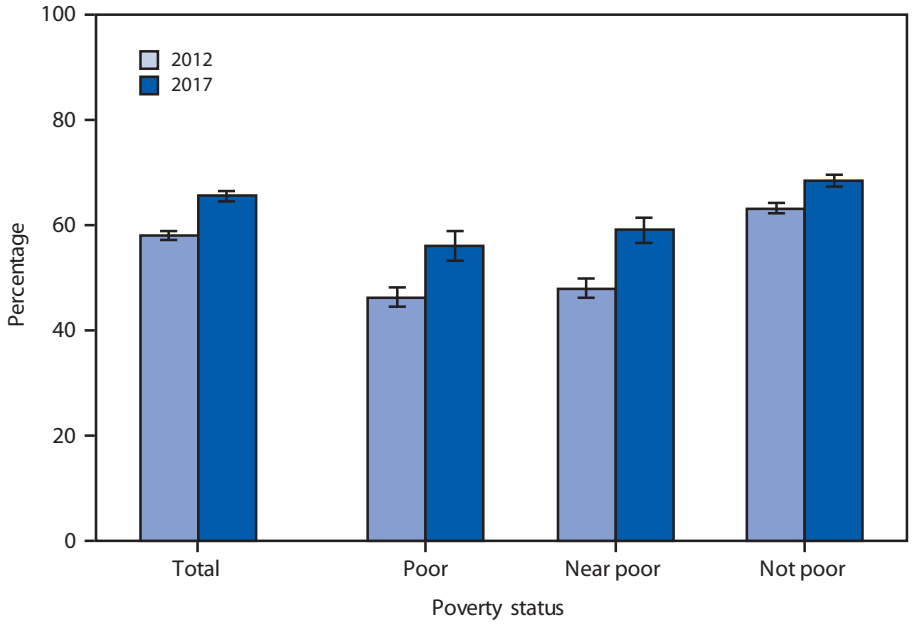
1. Turtles intrastate and interstate requirements, 21 C.F.R. Sect. 1240.62 (2011). https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/cfrsearch.cfm?fr=1240.62
2. Gambino-Shirley K, Stevenson L, Concepción-Acevedo J, et al. Flea market finds and global exports: four multistate outbreaks of human *Salmonella* infections linked to small turtles, United States—2015. Zoonoses Public Health 2018;65:560–8. https://doi.org/10.1111/zph.12466
3. Basler C, Bottichio L, Higa J, Prado B, Wong M, Bosch S. Multistate outbreak of human *Salmonella* Poona infections associated with pet turtle exposure—United States, 2014. MMWR Morb Mortal Wkly Rep 2015;64:804. https://doi.org/10.15585/mmwr.mm6429a7
4. CDC. Surveillance for foodborne disease outbreaks, United States, 2016: annual report. Atlanta, Georgia: US Department of Health and Human Services, CDC; 2018. https://www.cdc.gov/fdoss/pdf/2016_FoodBorneOutbreaks_508.pdf

*https://www.cdc.gov/salmonella/agbeni-08-17/map.html.

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Percentage* of Adults Aged 20–64 Years with a Blood Cholesterol Check by a Health Professional† During the Past 12 Months, by Poverty Status§ — National Health Interview Survey, 2012 and 2017¶



* With 95% confidence intervals shown with error bars.
† Based on a positive response to the question "During the past 12 months, have you had your blood cholesterol checked by a doctor, nurse, or other health professional?"
§ Poverty status is based on family income and family size using the U.S. Census Bureau's poverty thresholds. "Poor" persons are defined as those with incomes below the poverty threshold; "near poor" persons have incomes of 100% to <200% of the poverty threshold; and "not poor" persons have incomes of 200% of the poverty threshold or greater.
¶ 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 Sample Adult component.

The percentage of adults aged 20–64 years who had a blood cholesterol check by a health professional in the past 12 months increased from 58.0% in 2012 to 65.5% in 2017. From 2012 to 2017, there was an increase in the percentage of adults with a blood cholesterol check among poor (46.3% to 56.0%), near poor (47.9% to 59.0%), and not poor (63.2% to 68.5%) adults. In both years, not poor adults were more likely than poor and near poor adults to have had a blood cholesterol check.

Source: National Health Interview Survey, 2012 and 2017 data. https://www.cdc.gov/nchs/nhis.htm.

Reported by: Michael E. Martinez, MPH, MHSA, bmd7@cdc.gov, 301-458-4758; Tainya C. Clarke PhD.

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* at *https://www.cdc.gov/mmwr/index.html*.

Readers who have difficulty accessing this PDF file may access the HTML file at *https://www.cdc.gov/mmwr/index2018.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.

ISSN: 0149-2195 (Print)