Centers for Disease Control and Prevention
MMWR
Morbidity and Mortality Weekly Report
Weekly / Vol. 66 / No. 39
October 6, 2017

Retention in Medical Care Among Insured Children with Diagnosed HIV Infection — United States, 2010–2014

Mary R. Tanner, MD[1,2]; Tim Bush, MS[1]; Steven R Nesheim, MD[1]; Paul J. Weidle, PharmD[1]; Kathy K. Byrd, MD[1]

In 2014, an estimated 2,477 children aged <13 years were living with diagnosed human immunodeficiency virus (HIV) infection in the United States (*1*). Nationally, little is known about how well children with a diagnosis of HIV infection are retained in medical care. CDC analyzed insurance claims data to evaluate retention in medical care for children in the United States with a diagnosis of HIV infection. Data sources were the 2010–2014 MarketScan Multi-State Medicaid and MarketScan Commercial Claims and Encounters databases. Children aged <13 years with a diagnosis of HIV infection in 2010 were identified using *International Classification of Diseases, Ninth Revision, Clinical Modification* (ICD-9-CM) diagnostic billing codes for HIV or acquired immunodeficiency syndrome (AIDS), resulting in Medicaid and commercial claims cohorts of 163 and 129 children, respectively. Data for each child were evaluated during a 36-month study period, counted from the date of the first claim containing an ICD-9-CM code for HIV or AIDS. Each child's consistency of medical care was assessed by evaluating the frequency of medical visits during the first 24 months of the study period to see if the frequency of visits met the definition of retention in care. Frequency of medical visits was then assessed during an additional 12-month follow-up period to evaluate differences in medical care consistency between children who were retained or not retained in care during the initial 24-month period. During months 0–24, 60% of the Medicaid cohort and 69% of the commercial claims cohort were retained in care, among whom 93% (Medicaid) and 85% (commercial claims) were in care during months 25–36. To identify areas for additional public health action, further evaluation of the objectives for national medical care for children with diagnosed HIV infection is indicated.

National goals for HIV prevention and care include increasing retention in HIV care (*3*). One indicator for assessing progress toward this goal uses laboratory data reported to the National HIV Surveillance System to track retention in care for adults with diagnosed HIV infection; however, children with diagnosed HIV infection are not included in this assessment (*3*,*4*). The Health Resources and Services Administration evaluates retention in care by assessing the frequency of HIV-related medical visits (*2*). Health insurance claims databases contain information about medical encounters and have been used to assess retention in care for adults with diagnosed HIV infection (*5*,*6*). However, little information is available about retention in care metrics for children with diagnosed HIV infection in the United States.

Data from the 2010–2014 MarketScan Multi-State Medicaid and MarketScan Commercial Claims and Encounters databases were analyzed. MarketScan databases contain de-identified, patient-level health data, including inpatient, outpatient, and pharmaceutical services claims. A unique enrollee identifier is assigned to each client, allowing persons to be tracked across different types of data over multiple years (*5*). The MarketScan Medicaid databases include pooled Medicaid data

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

from 6–12 unidentified geographically dispersed states (*5*). MarketScan Commercial claims databases contain medical data from employees and their spouses and dependents who are covered by employer sponsored private health insurance in the United States (*6*).

Two cohorts were defined from the Medicaid and commercial claims MarketScan databases, using the following eligibility criteria: 1) an ICD-9-CM diagnostic billing code for HIV or AIDS in 2010; 2) aged <13 years in 2010; 3) enrollment in the relevant insurance program for ≥10 months out of each 12-month period during months 0–24; and 4) one or more outpatient visits with a physician, nurse practitioner, or physician's assistant during the first 6 months of the study period. Children with an ICD-9-CM code for HIV infection or AIDS on only one date were excluded. The study period for each subject extended 36 months from the date of the first claim containing an ICD-9-CM code for HIV infection or AIDS. Using a standard metric, retention in care was defined as at least one medical visit in each 6-month period during months 0–24, with a minimum of 60 days from the first medical visit in the prior 6-month period to the last medical visit in the subsequent 6-month period (*2*). During months 25–36, being in care was defined as having at least one medical visit in each 6-month period with a minimum of 60 days from the first medical visit in the prior 6-month period to the last medical visit in the subsequent 6-month period. This clinic-based definition was used to assess medical visits with clinical providers. Medical visits were counted in the analysis if they were associated with a qualifying outpatient visit Current Procedural Terminology (CPT) code and a provider type code indicating a visit with a physician, nurse practitioner, or physician's assistant or were associated with a consultation CPT code and a provider type code indicating a visit with an infectious diseases specialist. Visits associated with only a facility type code (e.g., acute care hospital) were not counted.

Demographic characteristics were described, and the unweighted proportion of children in each cohort who were retained in care during months 0–24 was determined. Each cohort was further categorized into retained and not retained subgroups, and within each subgroup, the unweighted proportions of children who met the definition for being in care during months 25–36 were determined. Records for persons not enrolled in the relevant insurance program for ≥10 months during months 25–36 were excluded.

Univariate logistic regression analyses were used to determine odds ratios (ORs) and 95% confidence intervals (CIs) to assess associations between available demographic factors and retention in care during months 0–24. Reference groups for regression were the following: male sex; age ≤1 year (compared with all other ages); basis of Medicaid eligibility was child (compared with blind/disabled individual, foster care child, and eligibility status unknown); and, for the Medicaid database, white race (compared with black, Hispanic, and other). Information on race/ethnicity was not available in the commercial claims database.

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

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

Cohorts consisted of 163 children from 4,713,171 unique persons in the Medicaid database, and 129 children from 45,239,752 unique persons in the commercial claims database (Table 1). The Medicaid cohort was predominately black (65%) and included equal proportions of males and females and a higher proportion of children aged 6–10 years (37%) (Table 2). The commercial claims cohort also had nearly equal proportions of males and females but similar proportions of children in all age groups. The most common basis of Medicaid eligibility category was child (not a child of unemployed adult or a foster care child). All children in the commercial claims cohort had a relationship to primary beneficiary category of child/other.

During months 0–24, 60% of the Medicaid cohort and 69% of the commercial claims cohort were retained in care (Figure). In the Medicaid cohort, 148 children remained in the study after month 24, and 117 (79%) were in care during months 25–36. Ninety-three percent of children in the retained in care subgroup and 59% of children in the not-retained subgroup were in care during months 25–36. In the commercial claims cohort, 91 children remained in the study after month 24, and 64 (70%) were in care during months 25–36. Eighty-five percent of children in the retained in care subgroup and 32% of children in the not-retained subgroup were in care during months 25–36, although a high rate of loss from the commercial claims cohort occurred after month 24 (Figure).

Compared with the reference groups, the basis of Medicaid eligibility categories of blind/disabled and foster child were associated with increased odds of retention in care (OR = 2.45, 95% CI = 1.09–5.53 and OR = 3.40, 95% CI = 1.16–9.99, respectively). In the commercial claims cohort, age ≤1 year was associated with decreased odds of retention in care (OR = 0.38, 95% CI = 0.16–0.88). No other covariates were significantly associated with retention in care in either cohort.

TABLE 1. Selection criteria for study cohorts of Medicaid and commercially insured children aged <13 years with diagnosed human immunodeficiency virus infection — MarketScan Multi-State Medicaid and Commercial Claims and Encounters databases, United States, 2010

Criteria	No. children	
	Multi-State Medicaid database	Commercial Claims database
Unique persons in the 2010 MarketScan database	4,713,171	45,239,752
Persons aged <25 years with an ICD-9-CM code for HIV or AIDS in 2010*	1,937	3,036
Children aged <13 years in 2010	403	370
Continuously enrolled months 0–24†	278	228
Qualifying outpatient visit in first 6 months of months 0–24§	198	158
Excluded¶,**	35	29
Total study population	**163**	**129**

Abbreviations: AIDS = acquired immunodeficiency syndrome; CPT = current procedural terminology; HIV = human immunodeficiency virus; ICD-9-CM = *International Classification of Diseases, Ninth Revision, Clinical Modification*.

* ICD-9-CM code for HIV or AIDS: inpatient or outpatient service claims that listed one or more ICD-9-CM diagnostic codes indicating HIV infection (042, V08, or 079.53). For ICD-9-CM code 795.71 (nonspecific serologic evidence of HIV), children were included only if another qualifying ICD-9-CM code was assigned to them over the course of the study period because code 795.71 is sometimes used to designate HIV-exposed infants before loss of maternal antibodies.

† Continuously enrolled was defined as enrolled in the relevant insurance program for ≥10 months out of each 12-month measurement period.

§ Qualifying outpatient visit criteria: an outpatient claim with a physician, nurse practitioner, or physician's assistant in the first 6 months of the study period. A qualifying visit had to meet one of the following criteria: 1) Outpatient Office Visit CPT code 99201, 99202, 99203, 99204, 99205, 99212, 99213, 99214, 99215, G0463, or T1015, and Physician, Nurse practitioner or Physician's Assistant Provider Type code 200–460, 825, or 845, or 2) Consultation CPT code 99241, 99242, 99243, 99244, or 99275 and Infectious Diseases Specialist Provider Type code 285 or 448.

¶ Medicaid: excluded because 1) ICD-9-CM code for HIV/AIDS on only one date (n = 31) and 2) Implausible Basis of Eligibility code was "adult, not based on employment," "aged individual," or "adult unemployed" (n = 4).

** Commercial Claims: excluded because 1) ICD-9-CM code for HIV/AIDS on only one date (n = 27) and 2) Implausible Relationship to Employee code was "employee" or "spouse" (n = 2).

Discussion

The proportions of children aged <13 years with diagnosed HIV infection who met the standard metric for retention in care for both the Medicaid and commercial claims cohorts were similar to those described in analyses conducted with

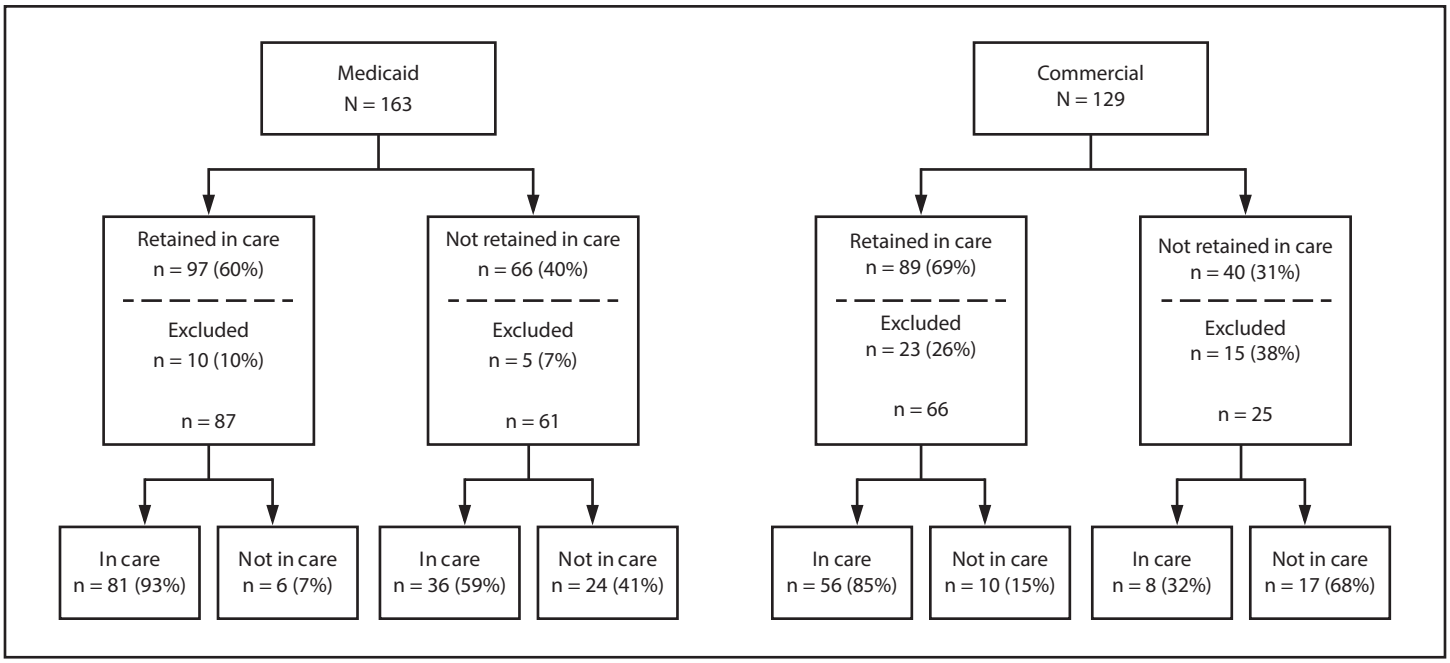
TABLE 2. Characteristics of Medicaid and commercially insured children aged <13 years with diagnosed human immunodeficiency virus infection — MarketScan Multi-State Medicaid and Commercial Claims and Encounters databases, United States, 2010

Characteristic	No. (%)	
	Medicaid (N = 163)	Commercial (N = 129)
Sex		
Male	81 (50)	66 (51)
Female	82 (50)	63 (49)
Race/Ethnicity*		
White	19 (12)	NA
Black	106 (65)	NA
Hispanic	7 (4)	NA
Other	31 (19)	NA
Age group (yrs)		
≤1	32 (20)	29 (22)
2–5	37 (23)	37 (29)
6–10	60 (37)	34 (26)
11–12	34 (21)	29 (22)
Medicaid (basis of Medicaid eligibility)		
Blind/Disabled	38 (23)	NA
Foster care child	22 (14)	NA
Child (not child of unemployed adult or foster care child)	92 (56)	NA
Unknown	11 (7)	NA
Commercial (relationship to primary beneficiary)		
Child/Other	NA	129 (100)
Dependent relationship unknown	NA	0

Abbreviation: NA = not available.

* No race/ethnicity information is collected within the MarketScan Commercial Claims and Encounter database.

FIGURE. Retention in care* and in-care† status among Medicaid§ and commercially¶ insured children aged <13 years with diagnosed human immunodeficiency virus infection — United States, 2010–2014**



* Retention in care defined as one or more qualifying outpatient visit in successive 6-month periods over 24 months (months 0–24), with ≥60 days between visits.
† In care is defined as having one or more qualifying outpatient visit during each 6-month period (months 25–36), with ≥60 days between visits.
§ MarketScan Medicaid Multi-State databases.
¶ MarketScan Commercial Claims and Encounters databases.
** Records of children who were not enrolled in insurance for ≥10 months out of each 12-month period were excluded.

insurance claims data for adults with diagnosed HIV infection (*5*,*6*). Among the 148 children in the Medicaid cohort after month 24, 117 (79%) were in care during months 25–36, including 59% of children who were not retained in care during months 0–24. This finding illustrates that failure to meet the retention in care definition (i.e., at least one medical visit in successive 6-month periods over 24 months) does not necessarily mean loss to follow-up, although it might suggest gaps in consistency of medical care.

Taken together with national surveillance data that indicate low rates of Stage 3 HIV (AIDS) diagnoses and deaths among children with diagnosed HIV infection (*1*), the need for additional public health attention to pediatric HIV care might not be immediately evident from these results. However, the proportions of children not meeting a retention in care definition based on 6-month interval clinic visits were unexpected in light of the frequency of medical visits recommended in many pediatric HIV care scenarios. U.S. Department of Health and Human Services pediatric HIV treatment guidelines recommend medical assessments every 3–4 months for the first 2 years of antiretroviral therapy (ART), and suggest that there is value in maintaining this frequency for all children with a diagnosis of HIV infection, although some experts might increase the time between assessments for certain stable patients (*7*). Gaps in medical care can result in missed or delayed opportunities for disease prevention (e.g., vaccinations) and might be associated with periods of reduced ART adherence, which could increase the risk for development of antiretroviral resistance (*8*), an issue of particular importance to children with diagnosed HIV infection, given their need for lifelong ART. Overall, the fact that >25% of children with diagnosed HIV infection did not meet the retention in care definition suggests that portions of this medically vulnerable population are not receiving the recommended frequency of medical care.

The finding that a Medicaid eligibility categorization of blind/disabled individual or foster care child was associated with improved odds of being retained in care might be related to increased health care needs and indications for more frequent medical follow-up in both groups (*5*,*9*). The finding that age ≤1 year was associated with decreased odds of retention in care in the commercial claims cohort is surprising, particularly in light of the fact that pediatric HIV treatment guidelines recommend urgent ART and frequent medical follow-up for children < 1 year of age (*7*). This study is not able to identify the reasons for these associations; further investigation into the causes of nonretention in pediatric HIV care is needed.

The findings in this report are subject to at least five limitations. First, because restricting analyses to HIV primary care

Summary

What is already known about this topic?

In 2014, an estimated 2,477 children aged <13 years in the United States were living with diagnosed human immunodeficiency virus (HIV) infection. Guidelines for treating children living with diagnosed HIV infection recommend medical visit frequency of every 3 to 4 months for the first 2 years after care initiation. Nationally, little is known about how well children with diagnosed HIV infection are retained in medical care.

What is added by this report?

Applying a clinic-based standard definition of retention in care (≥1 medical visit in successive 6-month periods over 24 months, with ≥60 days between visits), CDC analyzed insurance claims data and estimated that among children with diagnosed HIV infection aged <13 years, 60% of Medicaid-insured children and 69% of commercially insured children were retained in medical care. The retention in care proportions for both cohorts are similar to retention in care findings from analyses conducted with insurance claims data for HIV-diagnosed adults.

What are the implications for public health practice?

A substantial proportion of the medically vulnerable population of children with diagnosed HIV infection might not be receiving the recommended frequency of medical care. Further investigation into the causes of nonretention in pediatric HIV care is indicated to identify possible areas for public health action.

visits was not possible, office visits might have been for non–HIV-related issues, which might have caused overestimation of HIV-specific medical care. Second, characteristics of the provider type code used to define outpatient visits could have resulted in an underestimation of retention. Encounters were only counted as visits if they were associated with a provider type code indicating a visit with a physician, nurse practitioner, or physician's assistant; encounters associated only with the type of facility (e.g., acute care hospital) were not counted. Encounters associated only with facility codes were more common in the Medicaid database. Third, results from the Medicaid and commercial claims cohorts cannot be directly compared because of underlying differences in the databases and the inability to exclude overlap between database populations. Fourth, these findings are based on unweighted proportions and might not be generalizable to the larger population of children with diagnosed HIV infection. Finally, these data do not permit determination of possible causes of nonretention.

The pediatric population with HIV infection has unique needs and challenges. Children's physiologic maturity and developmental stage affect treatment decisions, and children with HIV infection require close follow-up as they grow and mature (*7*). Children also represent a vulnerable population because they are dependent on their caregivers, and their need for long-term ART makes optimizing medication management important (*10*). The national goal for retention in care for persons aged ≥13 years living with HIV infection, using laboratory tests as a proxy for care visits, is 90% (*3*). Although there is no specific goal for children aged ≤13 years, no reason exists for why children should have a lower retention in care target than adults. Evaluating pediatric retention in care by analyzing results from laboratory testing might provide additional information about retention in care for children with diagnosed HIV infection in the United States. In addition, further investigation into the causes of nonretention in pediatric HIV care is indicated to identify possible areas for public health action.

Conflict of Interest

No conflicts of interest were reported.

[1]Division of HIV/AIDS Prevention, National Center for HIV/AIDS, Viral Hepatitis, STD, and TB Prevention, CDC; [2]Epidemic Intelligence Service, CDC.

Corresponding author: Mary R. Tanner, mtanner@cdc.gov, 404-639-6376.

References

1. CDC. Diagnoses of HIV infection in the United States and dependent areas, 2015. HIV surveillance report, vol. 27. Atlanta, GA: CDC; 2016. https://www.cdc.gov/hiv/pdf/library/reports/surveillance/cdc-hiv-surveillance-report-2015-vol-27.pdf
2. Health Resources Services Administration. HIV/AIDS Bureau performance measures. Washington, DC: US Department of Health and Human Services, Health Resources Services Administration; 2017. https://hab.hrsa.gov/deliverhivaidscare/coremeasures.pdf
3. Secretary's Minority AIDS Initiative Fund. National HIV/AIDS strategy for the United States: updated to 2020. Indicator supplement. Washington, DC: US Department of Health and Human Services, Secretary's Minority AIDS Initiative Fund; 2016. https://files.hiv.gov/s3fs-public/nhas-indicators-supplement-dec-2016.pdf
4. CDC. Monitoring selected national HIV prevention and care objectives by using HIV surveillance data—United States and 6 dependent areas, 2014. HIV Surveillance Supplemental Report, vol. 21, no. 4. Atlanta, GA: CDC; 2016. https://www.cdc.gov/hiv/pdf/library/reports/surveillance/cdc-hiv-surveillance-supplemental-report-vol-21-4.pdf
5. Byrd KK, Furtado M, Bush T, Gardner L. Evaluating patterns in retention, continuation, gaps, and re-engagement in HIV care in a Medicaid-insured population, 2006–2012, United States. AIDS Care 2015;27:1387–95. https://doi.org/10.1080/09540121.2015.1114991
6. Byrd KK, Furtado M, Bush T, Gardner L. Reengagement in care after a gap in HIV care among a population of privately insured persons with HIV in the United States. AIDS Patient Care STDS 2016;30:491–6. https://doi.org/10.1089/apc.2016.0188
7. Panel on Antiretroviral Therapy and Medical Management of HIV-Infected Children. AIDSinfo: guidelines for the use of antiretroviral agents in pediatric HIV infection. Washington, DC: US Department of Health and Human Services, Office of AIDS Research Advisory Council; 2017. https://aidsinfo.nih.gov/contentfiles/lvguidelines/pediatricguidelines.pdf
8. Gardner EM, Burman WJ, Steiner JF, Anderson PL, Bangsberg DR. Antiretroviral medication adherence and the development of class-specific antiretroviral resistance. AIDS 2009;23:1035–46. https://doi.org/10.1097/QAD.0b013e32832ba8ec

9. Szilagyi MA, Rosen DS, Rubin D, Zlotnik S; Council on Foster Care, Adoption, and Kinship Care; Committee on Adolescence; Council on Early Childhood. Health care issues for children and adolescents in foster care and kinship care. Pediatrics 2015;136:e1131–40. https://doi.org/10.1542/peds.2015-2656

10. Hazra R, Siberry GK, Mofenson LM. Growing up with HIV: children, adolescents, and young adults with perinatally acquired HIV infection. Annu Rev Med 2010;61:169–85. https://doi.org/10.1146/annurev.med.050108.151127

Human Adenovirus Surveillance — United States, 2003–2016

Alison M. Binder, MS[1]; Holly M. Biggs, MD[1]; Amber K. Haynes, MPH[1]; Christina Chommanard, MS, MPH[1]; Xiaoyan Lu, MS[1]; Dean D. Erdman, DrPH[1]; John T. Watson, MD[1]; Susan I. Gerber, MD[1]

Human adenoviruses (HAdVs) are nonenveloped, double-stranded DNA viruses in the family Adenoviridae; seven species (A–G) and >60 genotypes are known to cause human infection (*1*). Clinical manifestations associated with HAdV infection include fever, acute respiratory illness, gastroenteritis, and conjunctivitis. HAdV infection can be severe, particularly among immunocompromised patients, and can cause respiratory failure, disseminated infection, hemorrhagic cystitis, neurologic disease, and death (*1*,*2*). Illness tends to occur sporadically and without demonstrated seasonality. Outbreaks of HAdV have been reported globally in communities (*3*), and in closed or crowded settings, including dormitories, health care settings, and among military recruits, for whom a vaccine against HAdV type 4 (HAdV-4) and HAdV type 7 (HAdV-7) has been developed (*4*,*5*). CDC summarized HAdV detections voluntarily reported through the National Adenovirus Type Reporting System (NATRS) after initiation of surveillance in 2014 to describe trends in reported HAdVs circulating in the United States. Reporting laboratories were also encouraged to report available results for specimens collected before surveillance began. Overall, the number of reporting laboratories and HAdV type identifications reported to NATRS has increased substantially from the start of official reporting in 2014 through 2016; this report describes specimens collected during 2003–2016. The most commonly reported HAdV types were HAdV type 3 (HAdV-3) and HAdV type 2 (HAdV-2), although HAdV types reported fluctuated considerably from year to year. In the United States, information on recently circulating HAdV types is needed to inform diagnostic and surveillance activities by clinicians and public health practitioners. Routine reporting to NATRS by all U.S. laboratories with the capacity to type HAdVs could help strengthen this surveillance system.

NATRS is a passive laboratory-based surveillance system initiated in 2014 to coordinate reporting of laboratory identifications of HAdV types in the United States. Traditional typing techniques based on serologic methods have been largely replaced by molecular typing techniques including sequencing and conventional or real-time polymerase chain reaction, which can rapidly determine HAdV types. However, the number of public health and clinical laboratories with the capacity to type HAdVs using molecular techniques remains limited, and traditional methods are labor intensive and time consuming. Commercial molecular assays are more readily available; although some of these assays determine species, they do not identify specific types. Public health and clinical laboratories without the capacity to type HAdVs can send specimens of public health or clinical importance to CDC's Respiratory Virus Diagnostics Laboratory. All participating laboratories are encouraged to report HAdV typing data quarterly to NATRS accompanied by limited demographic, clinical, and laboratory data.

Eleven laboratories reported data to NATRS during 2014–2016, including the CDC Respiratory Virus Diagnostics Laboratory, public health laboratories from seven states, two clinical laboratories, and one U.S. Department of Defense laboratory. Data with specimen-collection years 2003–2013 represent retrospective data from reporting laboratories with the capacity to test and type before surveillance formally began in 2014. All typing data for specimens collected during 2003–2007 was provided by the CDC Respiratory Virus Diagnostics Laboratory. Five laboratories, including the CDC Respiratory Virus Diagnostics Laboratory, provided data for typed detections among specimens collected during 2008–2013 (ranging from two laboratories in 2008 to five in 2013). NATRS received reports for 2,138 HAdV detections among specimens collected during 2003–2016. Species and type were reported for 2,107 (98.6%) and 1,497 (70.0%) detections, respectively, representing 22 types (Table 1) from 32 states and the U.S.

TABLE 1. Number and percentage of human adenovirus (HAdV) detections, by species and type — National Adenovirus Type Reporting System, 32 states and the U.S. Virgin Islands, 2003–2016

HAdV species	HAdV type	No. (%) of detections
A	12	t3 (0.2)
	31	3 (0.2)
B	3*	341 (22.8)
	7*	127 (8.5)
	11	6 (0.4)
	14*	89 (5.9)
	21	34 (2.3)
	34	2 (0.1)
	35	14 (0.9)
C	1*	248 (16.6)
	2*	293(19.6)
	5	56 (3.7)
	6	20(1.3)
D	8	54 (3.6)
	15	1 (0.1)
	19	1 (0.1)
	22	1 (0.1)
	29	1 (0.1)
	37	12 (0.8)
	56	1 (0.1)
E	4*	185 (12.4)
F	41	5 (0.3)
Total	**22**	**1,497 (100)**

* One of the six most common types detected, accounting for 1,283 (85.5%) of reports.

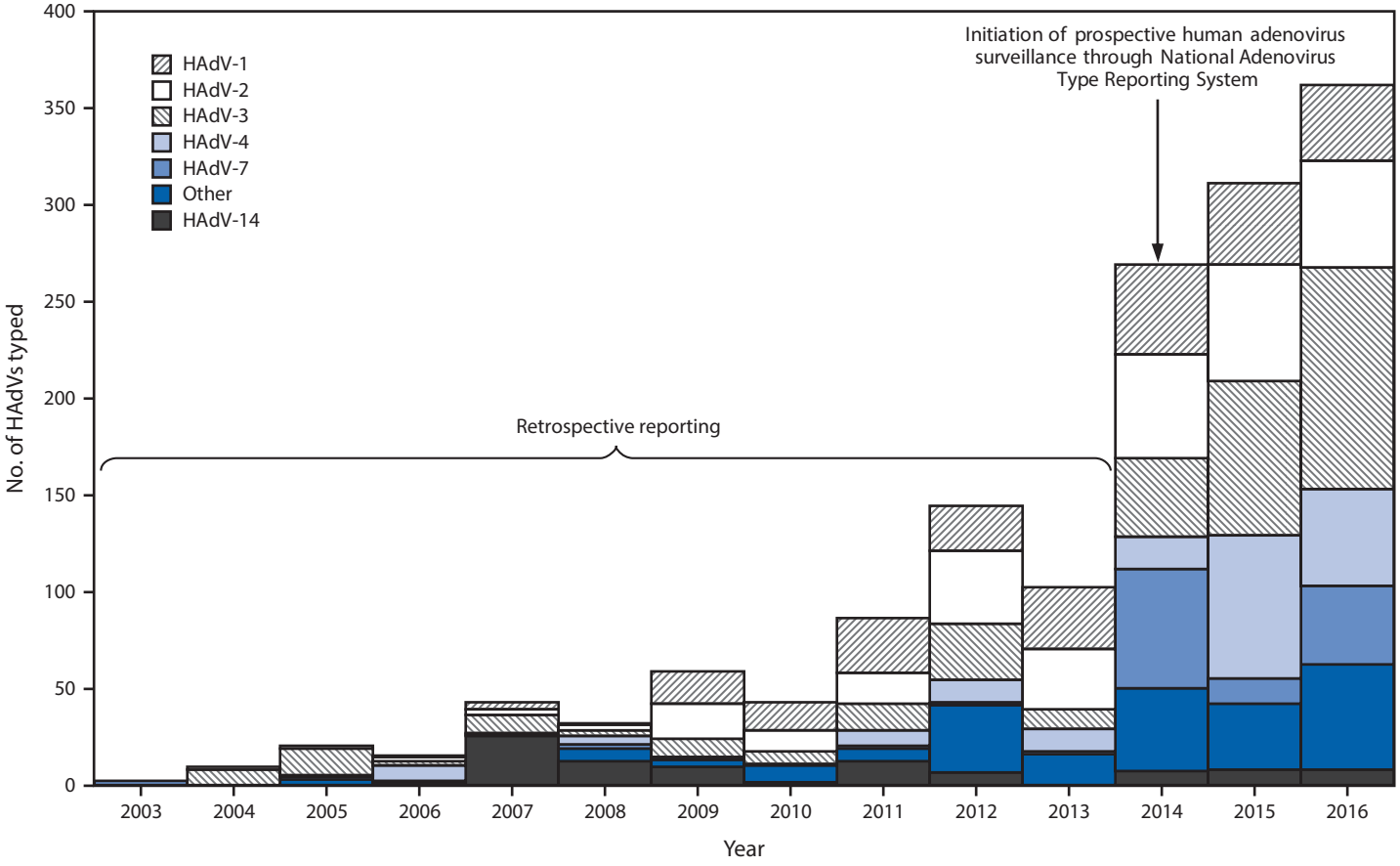
Virgin Islands, according to patient state of residence. The most commonly reported specimen types were respiratory specimens (N = 1,227; 82.0%), ocular swabs (61; 4.1%), and stool or rectal swabs (35; 1.8%). Species C (N = 683; 41%) and B (657; 40%) were the most commonly reported species during the 13-year period. The number of typed HAdV identifications reported by year of specimen collection ranged from two in 2003 to 269 in 2014 when data collection began and increased to 362 in 2016. HAdV-3 (N = 341; 22.8%), HAdV-2 (293; 19.6%), HAdV type 1 (HAdV-1) (248; 16.6%), HAdV-4 (185; 12.4%), HAdV-7 (127; 8.5%), and HAdV type 14 (HAdV-14) (89; 5.9%) accounted for 1,283 (85.5%) of the 1,497 reports with type identified (Figure) (Table 1). A single type was identified in 1,490 (99.5%) specimens, whereas detection of two types was identified in seven (0.5%) specimens. Year-to-year fluctuations in circulating types were evaluated using data reported during 2014–2016. HAdV-3, HAdV-2, HAdV-1, and HAdV-4 were among the most common types identified each year during this period; however, the frequency with which individual types were reported varied considerably from year to year (Table 2). The most commonly reported typing methods included serum neutralization (N = 591; 39.5%), full or partial genome sequencing (567; 37.9%), and real-time polymerase chain reaction (285; 19.0%).

Discussion

Type-based HAdV surveillance in the United States has three objectives: 1) to monitor patterns of circulation for HAdV types over time; 2) to assist with recognition and confirmation of outbreaks associated with circulating types; and 3) to inform development or use of diagnostics tests, therapeutics, and vaccines. After initiation of NATRS in 2014, this is the first report describing national trends in HAdV type circulation in the United States, and fluctuations in frequency of HAdV types during 2014–2016.

During 2003–2016, the six most commonly reported types were HAdV-3, HAdV-2, HAdV-1, HAdV-4, HAdV-7, and HAdV-14, which have all been detected in association with acute respiratory illness worldwide (*2*). HAdV-7 was recognized in the United States as an important cause of severe respiratory illness among adults in a community outbreak in Oregon in 2014 (*3*). Nearly half of HAdV-7 detections (n = 62; 48.8%)

FIGURE. Distribution of human adenovirus species (HAdVs) and types, by year of specimen collection* — National Adenovirus Type Reporting System, 32 U.S. states and the U.S. Virgin Islands, 2003–2016



* Frequency of the most common HAdV types reported after surveillance initiation in 2014 varied by year of specimen collection.

TABLE 2. Number and percentage of human adenovirus detections, by species, type, and year of specimen collection* — National Adenovirus Type Reporting System, 32 states and the U.S. Virgin Islands, 2003–2016

Retrospectively reported to NATRS		Reported to NATRS after surveillance initiation							
2003–2013 (n = 555)		2014 (n = 269)		2015 (n = 311)		2016 (n = 362)		Total NATRS 2003–2016 (N = 1,497)	
Species/Type	No. (%)	Species/Type	No. (%)	Species/Type	No. (%)	Species/Type	No. (%)	Species/Type	No. (%)
C1	124 (22.3)	B7	62 (23)	B3	80 (25.7)	B3	115 (31.8)	**B3**	**341 (22.8)**
B3	121 (21.8)	C2	54 (20.1)	E4	74 (23.8)	C2	55 (15.2)	**C2**	**293 (19.6)**
C2	105 (18.9)	C1	46 (17.1)	C2	60 (19.3)	E4	50(13.8)	**C1**	**248 (16.6)**
D8	66 (11.9)	B3	41 (15.2)	C1	42 (13.5)	B7	41 (11.3)	**E4**	**185 (12.4)**
B14	45 (8.1)	E4	16 (5.9)	C5	17 (5.5)	C1	39 (10.8)	**B7**	**127 (8.5)**
B7	33 (5.9)	C5	14 (5.2)	B7	13 (4.2)	B21	15 (4.1)	**B14**	**89 (5.9)**
C5	16 (2.9)	B21	11 (4.1)	B14	8 (2.6)	D8	13 (3.6)	**C5**	**56 (3.7)**
E4	11 (2)	B14	7 (2.6)	C6	7 (2.3)	C5	9 (2.5)	**D8**	**54 (3.6)**
B21	11 (2)	D8	5 (1.9)	D8	3 (1)	B14	8 (2.2)	**B21**	**34 (2.3)**
A12	6 (1.1)	D37	5 (1.9)	B21	3 (1)	C6	6 (1.7)	**C6**	**20 (1.3)**
B35	5 (0.9)	C6	4 (1.5)	D37	2 (0.6)	B35	3 (0.8)	**B35**	**14 (0.9)**
B11	3 (0.5)	A31	2 (0.7)	F41	1 (0.3)	D37	3 (0.8)	**D37**	**12 (0.8)**
F41	3 (0.5)	D19	1 (0.4)	D15	1 (0.3)	F41	2 (0.6)	**B11**	**6 (0.4)**
C6	2 (0.4)	D22	1 (0.4)	B35	—†	D29	1 (0.3)	**F41**	**5 (0.3)**
D37	2 (0.4)	B35	—†	A12	—†	B34	1 (0.3)	**A12**	**3 (0.2)**
A31	1 (0.2)	F41	—†	A31	—†	D56	1 (0.3)	**A31**	**3 (0.2)**
B34	1 (0.2)	A12	—†	B11	—†	A12	—†	**B34**	**2 (0.1)**
D15	—†	B11	—†	D19	—†	A31	—†	**D15**	**1 (0.1)**
D19	—†	D15	—†	D22	—†	B11	—†	**D19**	**1 (0.1)**
D22	—†	D29	—†	D29	—†	D15	—†	**D22**	**1 (0.1)**
D29	—†	B34	—†	B34	—†	D19	—†	**D29**	**1 (0.1)**
D56	—†	D56	—†	D56	—†	D22	—†	**D56**	**1 (0.1)**

Abbreviation: NATRS = National Adenovirus Type Reporting System.
* HAdV types reported after frequency of the most common surveillance initiation in 2014 varied by year of specimen collection.
† Data not reported.

in NATRS occurred during 2014 (Table 2) as a result of sampling and typing of specimens collected as part of the Oregon outbreak investigation. Before 2014, HAdV-7 was uncommonly reported among civilian populations, although respiratory illness outbreaks had been reported among military recruits (*6*). Respiratory illness associated with HAdV-4 also has been well documented among military recruits, but has been less commonly reported among civilian populations (*7*).

A live, oral vaccine against HAdV-4 and HAdV-7 was given to all U.S. military members from 1971 to 1999. After depletion of the vaccine in 1999, HAdV-4 reemerged as the main cause of febrile respiratory illness among military service members, especially among those in initial entry (i.e., basic) training. Subsequent reintroduction of HAdV-4 and HAdV-7 vaccine at all U.S. initial entry training sites in late 2011 led to declines in overall rates of respiratory illness and in incidence of adenovirus infections(*8*,*9*). The prevalence of respiratory illness associated with HAdV-4 and HAdV-7 in non-military congregate populations that might potentially benefit from HAdV vaccination is not known.

Other common HAdV types reported to NATRS include HAdV-14, which was first documented in North American military populations in 2006 (*2*,*10*). After HAdV-4 and HAdV-7 vaccine re-introduction in 2011, HAdV-14 surpassed HAdV-4 and HAdV-7 as the most prevalent HAdV type reported in these settings, although the actual number of cases of HAdV-14 did not increase (*8*). By 2007, outbreaks of respiratory illness because of HAdV-14 in multiple civilian populations were also documented (*4*). Among the 89 HAdV-14 detections reported to NATRS during 2003–2016, the highest number (25) and percentage (28.1%) were reported during 2007, likely coinciding with specimens collected during documented outbreaks of HAdV-14 among community and military populations and in health care settings in certain states during this period.

The findings in this report are subject to at least four limitations. First, NATRS is a passive system that relies on voluntary participation from laboratories, and data might be biased by outbreak investigations and nonrandom sample selection for typing; therefore, types reported might not be representative of the HAdV types circulating nationally or regionally. Second, NATRS collects limited clinical information, restricting the interpretation of trends in HAdV disease associated with circulating types. Third, although quarterly reporting to NATRS is encouraged and allows for retrospective outbreak documentation, not all participating laboratories submit timely data, limiting the ability to detect outbreaks of HAdVs in real-time. Finally, although the number of laboratories with the capacity to test for specific HAdV types and report to NATRS is

Summary

What is already known about this topic?

Human adenoviruses (HAdVs) can cause a wide spectrum of clinical illness, ranging from asymptomatic infections to severe illnesses and death. Approximately 60 HAdV genotypes have been identified to date, and they are associated with different clinical illnesses, including respiratory illness, gastroenteritis, and conjunctivitis. Surveillance for circulating HAdV types in the United States is passive and voluntary but might be useful to inform diagnostic and surveillance activities by clinicians and public health practitioners.

What is added by this report?

Based on data from the National Adenovirus Type Reporting System, the most commonly reported types of HAdVs during 2003–2016 in the United States were HAdV types 1, 2, 3, 4, 7, and 14, which accounted for 85.5% (n = 1,283) of all types reported. Year-to-year fluctuations in HAdV types circulating in the United States varied considerably, likely reflecting increases in testing in response to recognized HAdV outbreaks.

What are the implications for public health practice?

HAdV type-based surveillance data can be used to determine patterns of circulation for individual HAdV types in the United States, assist with the recognition and documentation of outbreaks associated with circulating types, and guide development of new diagnostic tests, therapeutics, and vaccines.

increasing, the relatively small number of reporters limits the reliability and generalizability of these results.

NATRS monitors patterns of HAdV circulation in the United States based on voluntary laboratory reporting of detections by type. Understanding currently circulating HAdV types and improving the reliability and generalizability of surveillance data relies upon voluntary reports to NATRS from public health and clinical laboratories. The long-term sustainability of NATRS depends on building and maintaining the capacity to identify and type HAdVs among public health and clinical laboratories; improving timeliness of reporting by currently participating laboratories; and increasing the number of participating laboratories.

Acknowledgments

Participating public health and clinical laboratories reporting to National Adenovirus Type Reporting System.

Conflict of Interest

No conflicts of interest were reported.

[1]Division of Viral Diseases, National Center for Immunization and Respiratory Disease, CDC.

Corresponding author: Alison M. Binder, abinder@cdc.gov, 404-718-6446.

References

1. Lion T. Adenovirus infections in immunocompetent and immunocompromised patients. Clin Microbiol Rev 2014;27:441–62. https://doi.org/10.1128/CMR.00116-13
2. Lynch JP 3rd, Kajon AE. Adenovirus: epidemiology, global spread of novel serotypes, and advances in treatment and prevention. Semin Respir Crit Care Med 2016;37:586–602. https://doi.org/10.1055/s-0036-1584923
3. Scott MK, Chommanard C, Lu X, et al. Human adenovirus associated with severe respiratory infection, Oregon, USA, 2013–2014. Emerg Infect Dis 2016;22:1044–51. https://doi.org/10.3201/eid2206.151898
4. CDC. Acute respiratory disease associated with adenovirus serotype 14—four states, 2006-2007. MMWR Morb Mortal Wkly Rep 2007;56:1181–4.
5. Lamson DM, Kajon A, Shudt M, Girouard G, St George K. Detection and genetic characterization of adenovirus type 14 strain in students with influenza-like illness, New York, USA, 2014–2015. Emerg Infect Dis 2017;23:1194–7. https://doi.org/10.3201/eid2307.161730
6. Metzgar D, Osuna M, Kajon AE, Hawksworth AW, Irvine M, Russell KL. Abrupt emergence of diverse species B adenoviruses at US military recruit training centers. J Infect Dis 2007;196:1465–73. https://doi.org/10.1086/522970
7. Kandel R, Srinivasan A, D'Agata EM, Lu X, Erdman D, Jhung M. Outbreak of adenovirus type 4 infection in a long-term care facility for the elderly. Infect Control Hosp Epidemiol 2010;31:755–7. https://doi.org/10.1086/653612
8. Radin JM, Hawksworth AW, Blair PJ, et al. Dramatic decline of respiratory illness among US military recruits after the renewed use of adenovirus vaccines. Clin Infect Dis 2014;59:962–8. https://doi.org/10.1093/cid/ciu507
9. Clemmons NS, McCormic ZD, Gaydos JC, Hawksworth AW, Jordan NN. Acute respiratory disease in U.S. army trainees 3 years after reintroduction of adenovirus vaccine 1. Emerg Infect Dis 2017;23:95–8. https://doi.org/10.3201/eid2301.161297
10. Kajon AE, Lu X, Erdman DD, et al. Molecular epidemiology and brief history of emerging adenovirus 14-associated respiratory disease in the United States. J Infect Dis 2010;202:93–103. https://doi.org/10.1086/653083

Update: Influenza Activity — United States and Worldwide, May 21–September 23, 2017

Lenee Blanton, MPH[1]; David E. Wentworth, PhD[1]; Noreen Alabi, MPH[1]; Eduardo Azziz-Baumgartner, MD[1]; John Barnes, PhD[1]; Lynnette Brammer, MPH[1]; Erin Burns, MA[1]; C. Todd Davis, PhD[1]; Vivien G. Dugan, PhD[1]; Alicia M. Fry, MD[1]; Rebecca Garten, PhD[1]; Lisa A. Grohskopf, MD[1]; Larisa Gubareva, PhD[1]; Krista Kniss, MPH[1]; Stephen Lindstrom, PhD[1]; Desiree Mustaquim, MPH[1]; Sonja J. Olsen, PhD[1]; Katherine Roguski, MPH[1]; Calli Taylor, MPH[1]; Susan Trock, DVM[1]; Xiyan Xu, MD[1]; Jacqueline Katz, PhD[1]; Daniel Jernigan, MD[1]

During May 21–September 23, 2017,* the United States experienced low-level seasonal influenza virus activity; however, beginning in early September, CDC received reports of a small number of localized influenza outbreaks caused by influenza A(H3N2) viruses. In addition to influenza A(H3N2) viruses, influenza A(H1N1)pdm09 and influenza B viruses were detected during May–September worldwide and in the United States. Influenza B viruses predominated in the United States from late May through late June, and influenza A viruses predominated beginning in early July. The majority of the influenza viruses collected and received from the United States and other countries during that time have been characterized genetically or antigenically as being similar to the 2017 Southern Hemisphere and 2017–18 Northern Hemisphere cell-grown vaccine reference viruses; however, a smaller proportion of the circulating A(H3N2) viruses showed similarity to the egg-grown A(H3N2) vaccine reference virus which represents the A(H3N2) viruses used for the majority of vaccine production in the United States. Also, during May 21–September 23, 2017, CDC confirmed a total of 33 influenza variant virus† infections; two were influenza A(H1N2) variant (H1N2v) viruses (Ohio) and 31 were influenza A(H3N2) variant (H3N2v) viruses (Delaware [1], Maryland [13], North Dakota [1], Pennsylvania [1], and Ohio [15]). An additional 18 specimens from Maryland have tested presumptive positive for H3v and further analysis is being conducted at CDC.

United States

The U.S. Influenza Surveillance System§ is a collaboration between CDC and federal, state, local, and territorial partners and uses eight data sources to collect influenza information,¶ six of which operate year-round. U.S. World Health Organization (WHO) and National Respiratory and Enteric Virus Surveillance System laboratories, which include both public health and clinical laboratories throughout the United States, contribute to virologic surveillance for influenza. During May 21–September 23, 2017, clinical laboratories in the United States tested 153,397 respiratory specimens for influenza viruses, 3,785 (2.5%) of which were positive (Figure 1). Among these, 1,885 (49.8%) were positive for influenza A viruses, and 1,900 (50.2%) were positive for influenza B viruses. Public health laboratories in the United States tested 6,431 respiratory specimens collected during May 21–September 23, 2017. Among these, 1,536 were positive for influenza (Figure 2), including 842 (54.8%) that were positive for influenza A viruses, and 694 (45.2%) that were positive for influenza B viruses. Influenza B viruses were more commonly reported from late May through late June, and influenza A viruses have predominated since early July. Among the 828 (98.3%) influenza A viruses subtyped by public health laboratories, 715 (86.4%) were influenza A(H3N2) and 113 (13.6%) were influenza A(H1N1)pdm09 virus. Among the 537 (77.4%) influenza B viruses for which lineage was determined, 398 (74.1%) belonged to the B/Yamagata lineage and 139 (25.9%) belonged to the B/Victoria lineage.

During May 21–September 23, the weekly percentage of outpatient visits to health care providers for influenza-like illness** from the U.S. Outpatient Influenza-Like Illness Surveillance Network remained below the national baseline†† of 2.2%, ranging from 0.7% to 1.2%. Based on data from CDC's National Center for Health Statistics Mortality Surveillance

* Data as of September 29, 2017.

† Influenza viruses that circulate in swine are called swine influenza viruses when isolated from swine, but are called variant influenza viruses when isolated from humans. Seasonal influenza viruses that circulate worldwide in the human population have important antigenic and genetic differences from influenza viruses circulating in swine.

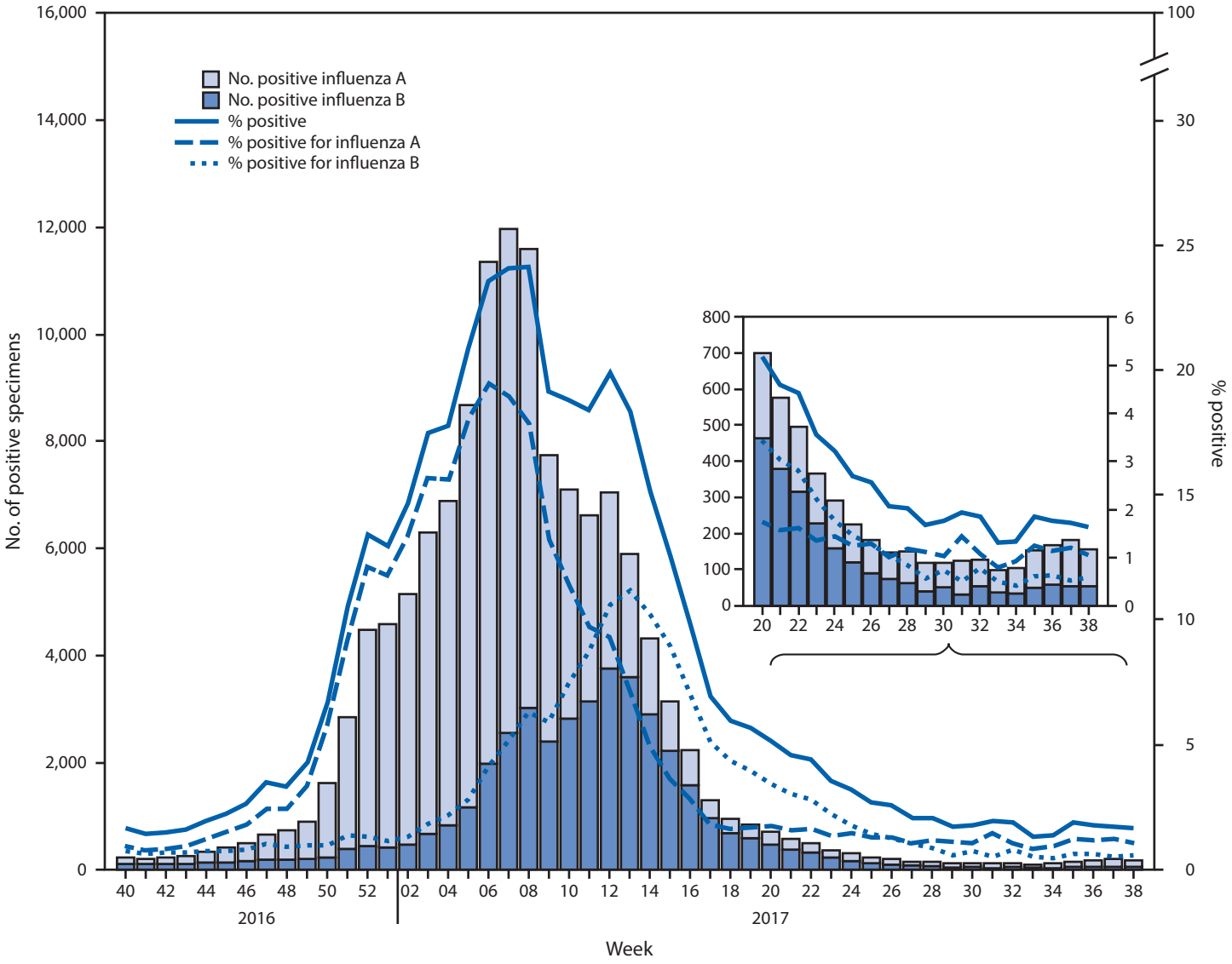
§ The CDC influenza surveillance system collects five categories of information from eight data sources: 1) viral surveillance (U.S. World Health Organization collaborating laboratories, the National Respiratory and Enteric Virus Surveillance System, and novel influenza A virus case reporting); 2) outpatient illness surveillance (U.S. Outpatient Influenza-Like Illness Surveillance Network); 3) mortality (the National Center for Health Statistics Mortality Surveillance System and influenza-associated pediatric mortality reports); 4) hospitalizations (FluSurv-NET, which includes the Emerging Infections Program and surveillance in three additional states); and 5) summary of the geographic spread of influenza (state and territorial epidemiologist reports). https://www.cdc.gov/flu/weekly/fluactivitysurv.htm.

¶ https://www.cdc.gov/flu/weekly/overview.htm.

** Defined as a fever (temperature ≥100°F [≥37.8°C]), oral or equivalent, and cough and/or sore throat, without a known cause other than influenza.

†† The national and regional baselines are the mean percentage of visits for influenza-like illness (ILI) during noninfluenza weeks for the previous three seasons plus two standard deviations. Noninfluenza weeks are defined as periods of ≥2 consecutive weeks in which each week accounted for <2% of the season's total number of specimens that tested positive for influenza in public health laboratories. National and regional percentages of patient visits for ILI are weighted based on state population. Use of the national baseline for regional data is not appropriate.

FIGURE 1. Number* and percentage of respiratory specimens testing positive for influenza reported by clinical laboratories, by influenza virus type and surveillance week — United States, October 2, 2016–September 23, 2017†



* 131,519 (12.3%) of 1,067,211 tested were positive during October 2, 2016–September 23, 2017.
† As of September 29, 2017.

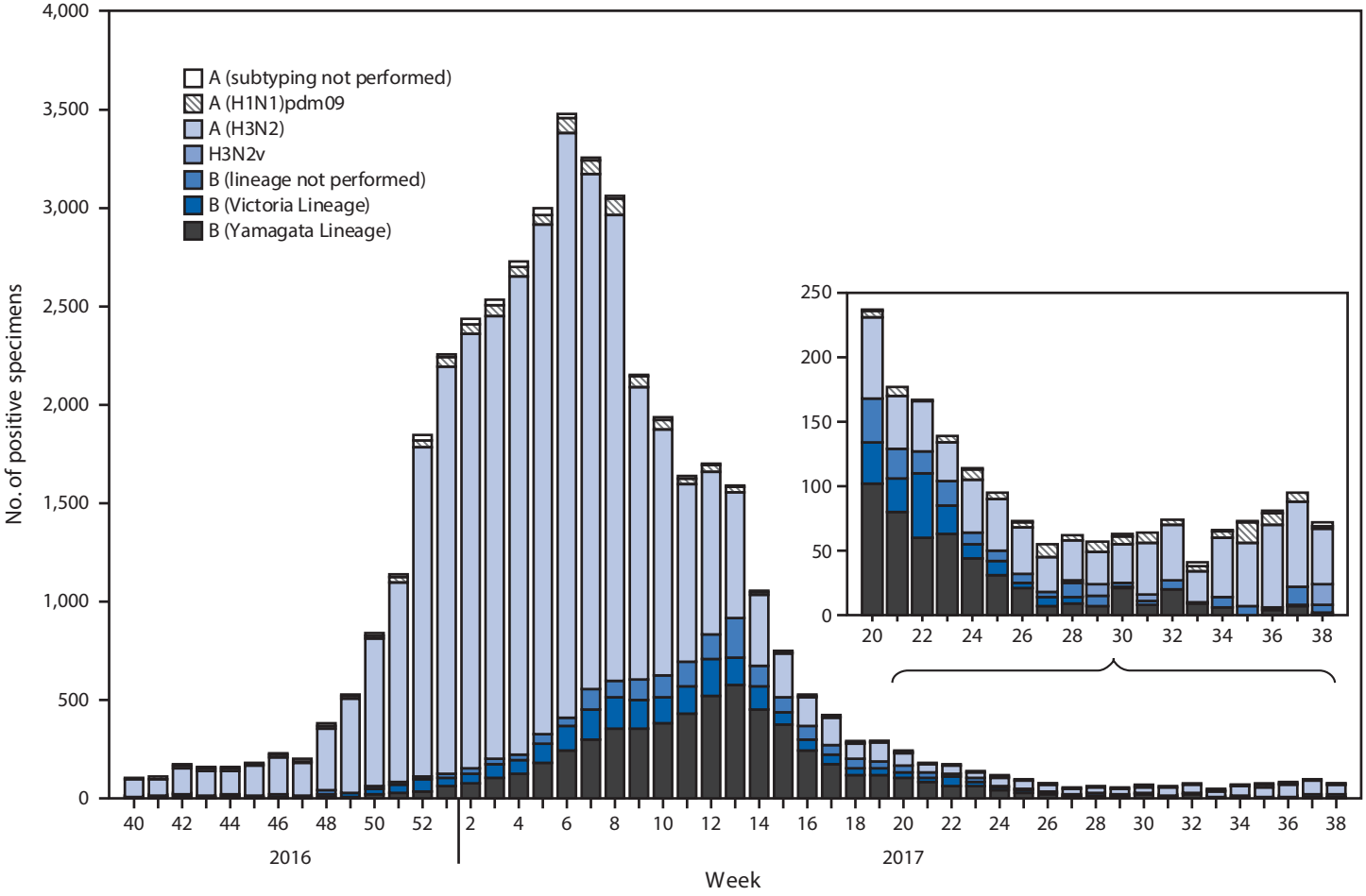
System, the percentage of deaths attributed to pneumonia and influenza did not exceed the epidemic threshold[§§] and ranged from 5.1% to 6.1%. Four influenza-associated pediatric deaths occurring during May 21–September 23 were reported; two were associated with an influenza A(H3N2) virus, one was associated with an influenza A(H1N1)pdm09 virus, and one was associated with an influenza B virus.

[§§] The seasonal baseline proportion of pneumonia and influenza (P&I) deaths is projected using a robust regression procedure, in which a periodic regression model is applied to the observed percentage of deaths from P&I that were reported by the National Center for Health Statistics Mortality Surveillance System during the preceding 5 years. The epidemic threshold is set at 1.645 standard deviations above the seasonal baseline.

Novel Influenza A Virus Infections

Fifty-one human infections with novel influenza A viruses were reported in the United States during May 21–September 23, 2017. All of these were variant virus infections (human infections with influenza viruses that normally circulate in swine). Thirty-one have been sequenced and are H3N2v viruses reported from five states (Delaware [1], Maryland [13], North Dakota [1], Pennsylvania [1], and Ohio [15]) and two were H1N2v viruses, both from Ohio. The remaining 18 viruses have tested presumptive positive for H3v at the Maryland public health laboratory and further confirmatory testing is being performed by CDC. All 51 patients reported

FIGURE 2. Number* of respiratory specimens testing positive for influenza reported by public health laboratories, by influenza virus type, subtype/lineage, and surveillance week — United States, October 2, 2016–September 23, 2017†



* N = 42,875.
† As of September 29, 2017.

exposure to swine in a fair setting during the week preceding illness onset. Swine influenza A viruses were identified from respiratory specimens collected from pigs at multiple fairs. Forty-seven of the 51 patients were children aged <18 years and four patients were adults aged ≥50 years. Three of the 51 patients were hospitalized. All other patients are recovering or have fully recovered from their illness. No human-to-human transmission of these viruses has been identified.

The viruses detected in Maryland, Ohio, North Dakota, and Pennsylvania all had a hemagglutinin (HA) gene derived from a seasonal human H3N2 virus that was likely introduced into swine by reverse zoonosis (i.e., humans infecting swine) in 2010. These viruses were closely related to H3N2 viruses known to circulate in the U.S. swine population, as well as to variant virus infections detected in Ohio and Michigan during 2016 (*1*). Further analysis of the variant viruses detected in the most recent cases from Maryland is being performed at CDC. One of the H1N2v viruses had an HA gene from the alpha sublineage of the classical swine H1 HA lineage (*2*). This is the second alpha sublineage H1N2v virus detected since the mid-1990s. The second H1N2v virus had an HA gene representative of the delta 2 sublineage circulating in swine. The HA and neuraminidase (NA) genes are closely related to 2016/2017 swine influenza viruses from the United States. The NA genes of both H3N2v and H1N2v viruses are related to human H3N2 viruses that likely entered the North American swine population around 2002 and have remained the predominant NA found in contemporary swine influenza viruses.

Worldwide

CDC serves as a WHO Collaborating Center for Surveillance, Epidemiology, and Control of Influenza, one of six WHO Collaborating Centers for Influenza in the WHO Global Influenza Surveillance and Response System (GISRS).¶¶ CDC,

¶¶ http://www.who.int/influenza/gisrs_laboratory/collaborating_centres/en.

along with other international public health partners, provides surveillance and virus characterization data to WHO.*** The timing of influenza activity around the world varies by region,††† and areas with similar influenza transmission patterns are grouped by influenza transmission zones.§§§

Reports from GISRS during May 21–September 23 suggested that typical seasonal patterns of influenza activity occurred in temperate climate Southern Hemisphere countries (*3*). Influenza activity began to increase in late April in the temperate countries of South America, late May in Southern Africa, early June in New Zealand, and early July in Australia. Influenza activity peaked in mid-June in temperate South America, the beginning of July in Southern Africa and New Zealand, and in mid-August in Australia, although elevated activity continued through September in Southern Africa and Australia. Influenza A(H3N2) viruses predominated across the Southern Hemisphere countries, with some reported influenza B viruses cocirculating in temperate South America, New Zealand, and Australia. In temperate-climate countries of Europe and North America, influenza activity was low, with influenza B viruses predominating.

In countries with tropical influenza seasonality, influenza activity levels and the predominant virus varied by country. In Central America and the Caribbean, activity was low and influenza A(H3N2) and influenza B viruses predominated. In tropical South America, influenza activity decreased from May 21 through September 23; influenza A(H3N2) viruses predominated with some influenza B viruses reported. Sporadic influenza virus detections were reported in Eastern and Western Africa, with influenza A(H1N1)pdm09, influenza A(H3N2), and influenza B viruses cocirculating. In Eastern Asia, high levels of influenza activity were reported in Southern China, Hong Kong special administrative region (SAR), and Taiwan beginning in July, peaked in mid-August, and decreased through September 23; influenza A(H3N2) viruses predominated. In Southern Asia, influenza A(H1N1)pdm09 viruses predominated, with elevated activity reported in India, Nepal, and the Maldives. Influenza activity in Southeast Asia was elevated in August and September. Influenza A(H1N1)pdm09 viruses predominated in the Philippines and Myanmar. Influenza A(H3N2), influenza A(H1N1)pdm09, and influenza B viruses cocirculated in Singapore, and influenza A(H1N1)pdm09 and influenza B viruses cocirculated in Vietnam.

During May 23–September 13, WHO reported 100 laboratory-confirmed human infections with avian influenza viruses, all from China, including 99 Asian lineage avian influenza A(H7N9) infections, and one influenza A(H9N2) infection.¶¶¶ A total of 764 human infections, including 283 (37%) deaths, with Asian lineage avian influenza A(H7N9) virus were reported to WHO from more provinces, regions, and municipalities in China during the fifth epidemic than in the previous four epidemics combined (*4*).

Genetic and Antigenic Characterization of Influenza Viruses

The 2017–18 influenza vaccine virus components were selected in March 2017, during one of two biannual WHO-sponsored vaccine consultation meetings to review influenza data generated by GISRS laboratories. The recommended Northern Hemisphere 2017–18 trivalent influenza vaccine composition consists of an A/Michigan/45/2015 (H1N1) pdm09-like virus, an A/Hong Kong/4801/2014 (H3N2)-like virus, and a B/Brisbane/60/2008-like (B/Victoria lineage) virus. An additional influenza B virus (B/Phuket/3073/2013-like [B/Yamagata lineage]) was recommended for quadrivalent vaccines.**** These recommendations reflect an update to the A(H1N1)pdm09 virus component to a more contemporary influenza A(H1N1)pdm09 virus (an A/California/7/2009 (H1N1)pdm09-like virus was replaced with an A/Michigan/45/2015 (H1N1)pdm09-like virus), compared with the recommendation for the Northern Hemisphere 2016–2017 influenza season and are the same as the vaccine virus recommendations made for the 2017 Southern Hemisphere influenza vaccine.

Most influenza vaccines licensed in the United States, with the exception of cell culture–based inactivated influenza vaccine (ccIIV4) and recombinant influenza vaccines (RIV3 and RIV4) are produced through propagation of candidate vaccine viruses (CVVs) in eggs. Historically, CVVs provided to manufacturers have been egg-derived. Egg propagation of influenza viruses, particularly influenza A(H3N2) viruses, often leads to genetic changes that might have antigenic implications. The vaccine viruses selected for the Northern Hemisphere 2017–18

*** http://www.who.int/influenza/gisrs_laboratory/en/.

††† In temperate climates, the onset and peak of influenza activity might vary substantially from one influenza season to the next, but generally begins to increase in the late fall. In the Northern Hemisphere's temperate regions, annual epidemics of influenza typically occur during October–February, but the peak of influenza activity can occur as late as April or May. In temperate regions of the Southern Hemisphere, influenza activity typically peaks during May through August. Although temperate regions of the world experience a seasonal peak in influenza activity, influenza viruses can be isolated year-round. The timing of seasonal peaks in influenza activity in tropical and subtropical countries varies by region. Multiple peaks of activity during the same year have been seen in some areas and influenza infection can occur year-round.

§§§ http://www.who.int/influenza/gisrs_laboratory/flunet/en/.

¶¶¶ The list of WHO monthly risk assessment summaries for human infections with avian influenza viruses is available at http://www.who.int/influenza/human_animal_interface/HAI_Risk_Assessment/en/ and WHO disease outbreak news reports are available at http://www.who.int/csr/don/en/.

**** http://www.who.int/influenza/vaccines/virus/recommendations/2017_18_north/en/.

vaccine were representative of most, but not all, circulating influenza viruses at that time, and had the fewest and least substantial egg-adapted changes. In August 2016, the Food and Drug Administration approved the use of cell-derived CVVs for inclusion in ccIIV4.[††††] For the 2017–18 season, the influenza A(H3N2) component of this vaccine is manufactured using a cell-derived CVV. The other components of this vaccine are manufactured using egg-derived CVVs. Production of influenza vaccines using cell-grown CVVs and cell-based technology can circumvent antigenic changes that might be associated with egg propagation, particularly for influenza A(H3N2) viruses.[§§§§]

Data obtained from antigenic characterization are important in the assessment of the similarity between reference vaccine viruses and circulating viruses. In vitro antigenic characterization data acquired through hemagglutination inhibition (HI) assays or virus neutralization assays are used to assess whether genetic changes in circulating viruses affect antigenicity, which could affect vaccine effectiveness. Since the 2014–15 season, many influenza A(H3N2) viruses lack sufficient hemagglutination titers for antigenic characterization using hemagglutination inhibition assays. Therefore, representative influenza A(H3N2) viruses are selected for antigenic characterization using the virus neutralization focus reduction assay to assess the ability of various antisera to neutralize infectivity of the test viruses. For nearly all influenza-positive surveillance samples received by CDC, next generation sequencing (NGS), which employs genomic enrichment practices (*5*–*7*), adapted by CDC, Nextera library preparation (Illumina, San Diego, California) and NGS using MiSeq (Illumina, San Diego, California), is performed to determine the genetic identity of circulating viruses. The genomic data are analyzed and submitted to public databases (GenBank or GISAID EpiFlu). CDC has antigenically or genetically characterized 877 influenza viruses collected and submitted by U.S. and international laboratories since May 21, 2017, including 117 influenza A(H1N1)pdm09 viruses, 495 influenza A(H3N2) viruses, and 265 influenza B viruses.

Phylogenetic analysis of the HA genes from the A(H1N1) pdm09 viruses collected since May 21, 2017, showed that all but one were in subclade 6B.1, and one virus belonged to clade 6B (Figure 3). All A(H1N1)pdm09 viruses were antigenically similar (analyzed using HI with ferret antisera) to the 6B.1 virus A/Michigan/45/2015, the recommended influenza A(H1N1) pdm09 reference virus for the 2017 Southern Hemisphere and 2017–18 Northern Hemisphere influenza vaccines.

Four hundred ninety-five influenza A(H3N2) viruses collected globally since May 21, 2017, were sequenced, and phylogenetic analysis of the HA genes illustrated that multiple clades/subclades were cocirculating (Figure 3). The HA genes showed extensive diversity and belonged to clades 3C.2a or 3C.3a, with 3C.2a predominating (Figure 3). The 3C.2a and the 3C.2a1 subclade circulated in approximately equal proportions. A representative set of 153 influenza A(H3N2) viruses (51 international and 102 United States) were antigenically characterized, and most (97%) A(H3N2) viruses were well-inhibited (reacting at titers of less than or equal to fourfold of the homologous virus titer) by ferret antisera raised against A/Michigan/15/2014 (3C.2a), a cell propagated A/Hong Kong/4801/2014-like reference virus representing the A(H3N2) component of the 2017 Southern Hemisphere and 2017–18 Northern Hemisphere influenza vaccines. A smaller proportion (33%) of influenza A(H3N2) viruses were well-inhibited by antiserum raised against egg-propagated A/Hong Kong/4801/2014 reference virus representing the A(H3N2) vaccine component, which is likely because of egg-adaptive amino acid changes in the HA of the egg-propagated virus.

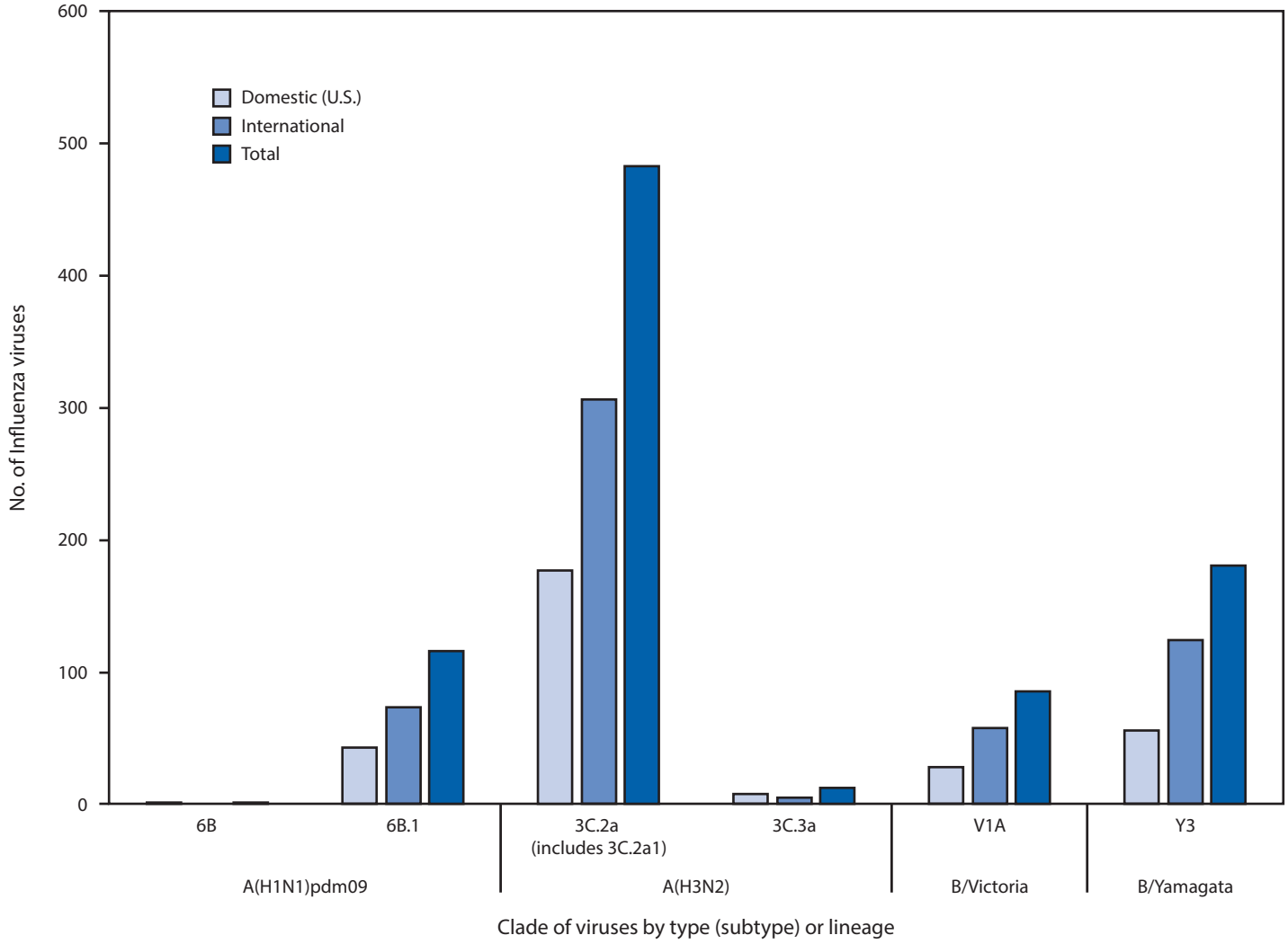
A total of 85 influenza B/Victoria-lineage viruses were phylogenetically analyzed, and all HA genes belonged to genetic clade V1A, the same genetic clade as the vaccine reference virus, B/Brisbane/60/2008. However, two deletion subclades were detected in 2017. One subclade has a 6-nucleotide deletion (encoding amino acids 162 and 163) and the other subclade has a 9-nucleotide deletion (encoding amino acids 162, 163 and 164). The 162–163 double deletion in the HA was detected in viruses circulating in multiple countries, with the majority identified in the United States, although the three viruses with 162–164 triple deletion were only detected in Hong Kong SAR, China. Thirty-nine (72%) B/Victoria lineage viruses were well-inhibited by ferret antisera raised against MDCK-propagated B/Brisbane/60/2008 reference virus, representing the B/Victoria lineage component of the 2017 Southern Hemisphere and 2017–2018 Northern Hemisphere influenza vaccines. However, 28% of B/Victoria lineage viruses reacted poorly with ferret antisera raised against MDCK-propagated B/Brisbane/60/2008, which correlated with the 162–163 double deletion and the 162–164 triple deletion in the HA.

Phylogenetic analysis of 180 influenza B/Yamagata-lineage viruses indicate that the HA genes belonged to clade Y3 (Figure 3). A total of 99 representative influenza B/Yamagata-lineage viruses (59 international and 40 United States) were antigenically characterized, and all were antigenically similar to B/Phuket/3073/2013, the reference vaccine virus representing the influenza B/Yamagata-lineage component of the 2017 Southern Hemisphere and 2017–18 Northern Hemisphere quadrivalent vaccines.

[††††] https://www.fda.gov/downloads/BiologicsBloodVaccines/Vaccines/ApprovedProducts/UCM522280.pdf.

[§§§§] https://virologyj.biomedcentral.com/track/pdf/10.1186/1743-422X-4-42?site=virologyj.biomedcentral.com.

FIGURE 3. Genetic characterization of U.S. and international viruses collected during May 21, 2017–September 23, 2017*



* As of September 29, 2017.

Composition of the 2018 Southern Hemisphere Influenza Vaccine

The WHO recommendations for influenza vaccine composition for the 2018 Southern Hemisphere season were made at the WHO Vaccine Consultation meeting September 25–28, 2017, in Melbourne, Australia. The recommended components for the 2018 Southern Hemisphere influenza trivalent vaccines are an A/Michigan/45/2015 (H1N1)pdm09-like virus, an A/Singapore/INFIMH-16-0019/2016 (H3N2)-like virus, and a B/Phuket/3073/2013-like (B/Yamagata lineage) virus (*8*). For quadrivalent vaccines, an additional component, B/Brisbane/60/2008-like (B/Victoria lineage) virus, is recommended (*8*). This represents a change in the influenza A(H3N2) component and a change in the influenza B lineage included in the trivalent vaccine compared with the composition of the 2017 Southern Hemisphere and 2017–18 Northern Hemisphere influenza vaccine formulation. The H3N2 component was updated to address the egg-adaptive changes that occurred with the egg-propagated A/Hong Kong/4801/2014 reference virus and to better represent genetic changes seen in recently circulating H3N2 viruses.

Antiviral Resistance of Influenza Viruses

The WHO Collaborating Center for Surveillance, Epidemiology, and Control of Influenza at CDC tested 486 influenza virus specimens collected during May 21–September 23, 2017, from the United States and worldwide for resistance to the influenza neuraminidase inhibitor antiviral medications currently approved for use against seasonal influenza: oseltamivir, zanamivir, and peramivir. A total of 75 influenza A(H1N1)pdm09 viruses (37 international and 38 United States) were tested, and all were sensitive to these drugs.

All 231 influenza A(H3N2) viruses (60 international and 171 United States) and all 180 influenza B viruses (98 international and 82 United States) tested were also sensitive to all three recommended antiviral medications. High levels of resistance to the adamantanes (amantadine and rimantadine) persist among influenza A(H1N1)pdm09 and influenza A(H3N2) viruses. Adamantane drugs continue not to be recommended for use against influenza at this time.

Discussion

During May 21–September 23, 2017, influenza A(H3N2), influenza A(H1N1)pdm09, and influenza B viruses co-circulated worldwide. In the United States, influenza B viruses predominated from late May through late June. Influenza A viruses were most commonly reported beginning in early July. The majority of the influenza viruses collected from the United States and other countries during that time were characterized antigenically and genetically as being similar to the cell-grown reference viruses representing the 2017 Southern Hemisphere and 2017–18 Northern Hemisphere influenza vaccine viruses. Antigenic and genetic characterization of circulating influenza viruses can give an indication of the influenza vaccine's ability to produce an immune response against the wide array of influenza viruses cocirculating, but vaccine effectiveness studies are needed to determine how much protection has been provided to the population by vaccination. Influenza A(H1N1) pdm09 viruses were detected at low levels from May 21 to September 23, and virus characterization data indicate no substantial genetic or antigenic changes, even among viruses from regions that experienced higher A(H1N1)pdm09 activity. Influenza A(H3N2) viruses have predominated in many countries in the Southern Hemisphere and in the United States since early July. Virus characterization data suggest extensive genetic diversity among circulating viruses, but limited evidence of substantial antigenic drift. To date, a predominant subclade of A(H3N2) viruses with substantial antigenic drift has yet to emerge, and extensive genetic variation exists in the circulating virus population. Among the influenza B viruses for which lineage was determined, influenza B/Yamagata viruses predominated across the United States from May 21 through September 23, and virus characterization data indicate no substantial genetic or antigenic changes. Two subgroups of antigenically distinct influenza B/Victoria viruses, represented by the double or triple deletion viruses, were detected; the majority of the double deletion viruses were identified in the United States, while all three triple deletion viruses were identified only in Hong Kong SAR, China. Nevertheless, such antigenically distinct viruses represented a minority of B/Victoria viruses circulating globally during this period. Close monitoring of these viruses is required to better assess their potential impact on public health. Although influenza B viruses circulate throughout the influenza season, they frequently circulate later in the season than do influenza A viruses and often result in a second peak of influenza activity, often in the late winter and spring in the United States and other Northern Hemisphere countries (*3*).

Annual influenza vaccination is the best method for preventing influenza and its potentially severe complications (*9*). In the United States, annual influenza vaccination is recommended for all persons aged ≥6 months who do not have contraindications. Annual influenza vaccination is recommended regardless of whether the vaccine composition has changed because immunity from vaccination wanes over time and might decline below protective levels after one season. Optimally, vaccination should occur before the onset of influenza activity in the community. If possible, vaccination should be offered by the end of October and should continue to be offered as long as influenza viruses are circulating and unexpired vaccine is available. Children aged 6 months through 8 years who require 2 doses should receive their first dose as soon as possible after vaccine becomes available, and the second dose ≥4 weeks later. For 2017-18 season, manufacturers have projected they will supply the United States with as many as 151 to 166 million doses of injectable influenza vaccine; approximately 119 million of this will be quadrivalent vaccine. As of September 15, 2017, approximately 73 million doses had already been distributed.

Multiple influenza vaccines are approved and recommended for use and are being distributed during the 2017–18 season, including egg-based trivalent and quadrivalent inactivated influenza vaccines (IIV3 and IIV4), adjuvanted trivalent egg-based inactivated influenza vaccines (aIIV3), high-dose trivalent egg-based inactivated influenza vaccines (HD-IIV3), quadrivalent cell culture–based inactivated influenza vaccines (ccIIV4), and recombinant trivalent and quadrivalent influenza vaccines (RIV3 and RIV4). Two available intramuscular vaccines are approved for administration by jet injector for persons aged 18 through 64 years. One IIV4 formulation is approved for intradermal administration. There is no preferential recommendation for one licensed and recommended influenza vaccine product over another for persons for whom more than one licensed, recommended product is available (*9*). Currently available influenza vaccines, with the exceptions of RIV3, RIV4, and ccIIV4, are prepared by propagation of virus in embryonated eggs (*9*). Egg propagation of influenza A(H3N2) viruses often leads to genetic changes that have antigenic implications. For the 2017–18 season inactivated vaccines, all influenza A(H1N1) and A(H3N2) and both influenza B components will be egg-derived, with the exception of ccIIV4, for which the influenza A(H3N2) virus component will, for the first time, be a cell-derived vaccine virus component (*9*).

This represents a first step toward producing a totally egg-independent inactivated virus vaccine. Recombinant technology is used in the production of RIV3 and RIV4; therefore they are manufactured without the use of influenza viruses or eggs. The use of egg-independent vaccine technologies is likely to provide vaccines that more precisely represent the antigenic characteristics of circulating viruses and have the potential to offer improved protection. Because of the low effectiveness of live attenuated intranasal influenza vaccine (LAIV4) against influenza A(H1N1)pdm09 viruses in the United States during the 2013–14 and 2015–16 seasons, for the 2017–18 season, the Advisory Committee on Immunization Practices and CDC renewed the recommendation that LAIV4 should not be used (*9*).

Although vaccination is the best method for preventing and reducing the impact of influenza, antiviral medications provide a valuable adjunct. Treatment with influenza antiviral medications as early as possible in the course of illness is recommended for patients with confirmed or suspected influenza (either seasonal influenza or novel influenza virus infection) who have severe, complicated, or progressive illness; who require hospitalization; or who are at high risk for influenza-related complications[¶¶¶¶] (*10*). Treatment is most effective when given early in the illness, especially within 48 hours of illness onset; providers should not delay treatment until test results become available and should not rely on insensitive assays such as found with some rapid antigen detection influenza diagnostic tests to determine treatment decisions (*10*).

Fifty-one infections with variant influenza viruses were reported from five states during summer 2017. Most of these infections occurred in children with prior direct contact with pigs at agricultural fairs, highlighting the importance of preventive actions,[*****] especially for young children or persons at high risk for serious influenza complications. Although community transmission of these viruses has not been identified, the potential for them to develop the ability to transmit efficiently from person to person remains a concern. Testing for seasonal influenza viruses and monitoring for novel influenza A virus infections should continue year-round. Health care providers also are reminded to consider novel influenza virus infections in persons with influenza-like illness and swine or poultry exposure, or with severe acute respiratory infection after travel to areas where avian influenza viruses have been detected. Providers should alert the local public health department if novel influenza virus infection is suspected. Clinical laboratories using a commercially available influenza diagnostic assay that includes influenza A virus subtype determination should contact their state public health laboratory to facilitate transport and additional testing of any specimen that is positive for influenza A, but for which the subtype cannot be determined. Public health laboratories should immediately send influenza A virus specimens that they cannot subtype using standard methods to CDC and submit all specimens that are otherwise unusual as soon as possible after identification. Early identification and investigation of human infections with novel influenza A viruses are critical to ensure timely risk assessment so that appropriate public health measures can be taken.

Summary

What is already known about this topic?

CDC collects, compiles, and analyzes data on influenza activity year-round in the United States. Timing of influenza activity and predominant circulating influenza viruses vary by season.

What is added by this report?

Worldwide, influenza activity during May 21–September 23, 2017, followed typical seasonality and in the United States overall, low levels of seasonal influenza activity were detected. The majority of influenza viruses genetically and antigenically characterized at CDC were similar to the reference viruses representing the recommended components for the 2017–18 vaccine. A small subset of antigenically distinct influenza B/Victoria viruses was detected.

What are the implications for public health practice?

In the United States, an annual influenza vaccination is recommended for all persons aged ≥6 months and can reduce the likelihood of becoming ill with influenza and transmitting the virus to others. Annual influenza vaccination offers optimal protection regardless of whether the vaccine composition has changed since the previous season, because immunity wanes over time. Although vaccination is the best method for preventing and reducing the impact of influenza, antiviral medications are an important adjunct. Early treatment with influenza antiviral medications is recommended for patients with confirmed or suspected influenza (either seasonal influenza or novel influenza virus infection) who have severe, complicated, or progressive illness; who require hospitalization; or who are at high risk for influenza-related complications. Testing for seasonal influenza viruses and monitoring for novel influenza A virus infections should continue year-round.

[¶¶¶¶] Persons at high risk include 1) children aged <2 years; 2) adults aged ≥65 years; 3) persons with chronic pulmonary conditions (including asthma), cardiovascular disease (except hypertension alone), renal, hepatic, hematologic (including sickle cell) disease, metabolic disorders (including diabetes mellitus), or neurologic and neurodevelopmental conditions (including disorders of the brain, spinal cord, peripheral nerves, and muscles, such as cerebral palsy, epilepsy [seizure disorders], stroke, intellectual disability [mental retardation], moderate to severe developmental delay, muscular dystrophy, or spinal cord injury); 4) persons with immunosuppression, including that caused by medications or by human immunodeficiency virus infection; 5) women who are pregnant or postpartum (within 2 weeks after delivery); 6) persons aged ≤18 years who are receiving long-term aspirin therapy; 7) American Indians/Alaska Natives; 8) persons with extreme obesity (i.e., body mass index ≥40); and 9) residents of nursing homes and other chronic care facilities.

[*****] https://www.cdc.gov/flu/swineflu/variant/preventspreadfactsheet.htm.

Influenza surveillance reports for the United States are posted online weekly (https://www.cdc.gov/flu/weekly). Additional information regarding influenza viruses, influenza surveillance, influenza vaccine, influenza antiviral medications, and novel influenza A infections in humans is available at https://www.cdc.gov/flu.

Acknowledgments

State, county, city, and territorial health departments and public health laboratories; U.S. World Health Organization collaborating laboratories; National Respiratory and Enteric Virus Surveillance System laboratories; U.S. Outpatient Influenza-Like Illness Surveillance Network sites; National Center for Health Statistics, CDC; FluNet, World Health Organization; Angie Foust, Wendy Sessions, Anwar Isa Abd Elal, Elisabeth Blanchard, Priya Budhathoki, Thomas Rowe, Lizheng Guo, LaShondra Berman, Shannon Emery, Janná Murray, Ji Liu, Bo Shu, Brian Lynch, Marisela Rodriguez, Ewelina Lyszkowicz, Shoshona Le, Malania Wilson, Juliana DaSilva, Alma Trujillo, Thomas Stark, Samuel Shepard, Sujatha Seenu, Ha Nguyen, Vasiliy Mishin, Juan De la Cruz, Roxana Cintron, Norman Hassell, Influenza Division, National Center for Immunization and Respiratory Diseases, CDC.

Conflict of interest

Jacqueline M. Katz reports U.S. Patent 6,196,175 (issued January 2, 2001) for "Preparation and use of recombinant influenza A virus M2 construct vaccine" and U.S. Patent 8,163,545 (issued April 26, 2012) for "An effective vaccine against pandemic strains of influenza viruses." No other conflicts of interest were reported.

[1]Influenza Division, National Center for Immunization and Respiratory Diseases, CDC.

Corresponding author: Lenee Blanton, lblanton@cdc.gov, 404–639–3747.

References

1. Bowman AS, Walia RR, Nolting JM, et al. Influenza A(H3N2) virus in swine at agricultural fairs and transmission to humans, Michigan and Ohio, USA, 2016. Emerg Infect Dis 2017;23:1551–5. https://doi.org/10.3201/eid2309.170847
2. Anderson TK, Macken CA, Lewis NS, et al. A phylogeny-based global nomenclature system and automated annotation tool for H1 hemagglutinin genes from swine influenza A viruses. MSphere 2016;1:e00275–16. https://doi.org/10.1128/mSphere.00275-16
3. Azziz Baumgartner E, Dao CN, Nasreen S, et al. Seasonality, timing, and climate drivers of influenza activity worldwide. J Infect Dis 2012;206:838–46. https://doi.org/10.1093/infdis/jis467
4. 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
5. Zhou B, Donnelly ME, Scholes DT, et al. Single-reaction genomic amplification accelerates sequencing and vaccine production for classical and Swine origin human influenza a viruses. J Virol 2009;83:10309–13. https://doi.org/10.1128/JVI.01109-09
6. Zhou B, Wentworth DE. Influenza A virus molecular virology techniques. Methods Mol Biol 2012;865:175–92. https://doi.org/10.1007/978-1-61779-621-0_11
7. Zhou B, Lin X, Wang W, et al. Universal influenza B virus genomic amplification facilitates sequencing, diagnostics, and reverse genetics. J Clin Microbiol 2014;52:1330–7. https://doi.org/10.1128/JCM.03265-13
8. World Health Organization. Recommended composition of influenza virus vaccines for use in the 2018 southern hemisphere influenza season. Geneva, Switzerland: World Health Organization; 2017. http://www.who.int/influenza/vaccines/virus/recommendations/2018_south/en/
9. Grohskopf LA, Sokolow LZ, Broder KR, et al. Prevention and control of seasonal influenza with vaccines: recommendations of the Advisory Committee on Immunization Practices—United States, 2017–18 influenza season. MMWR Recomm Rep 2017;66:1–20. https://doi.org/10.15585/mmwr.rr6602a1
10. Fiore AE, Fry A, Shay D, Gubareva L, Bresee JS, Uyeki TM. Antiviral agents for the treatment and chemoprophylaxis of influenza—recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep 2011;60(No. RR-1).

Vital Signs: Trends in Incidence of Cancers Associated with Overweight and Obesity — United States, 2005–2014

C. Brooke Steele, DO[1]; Cheryll C. Thomas, MSPH[1]; S. Jane Henley, MSPH[1]; Greta M. Massetti, PhD[1]; Deborah A. Galuska, PhD[2]; Tanya Agurs-Collins, PhD[3]; Mary Puckett, PhD[1]; Lisa C. Richardson, MD[1]

On October 3, 2017, this report was posted as an MMWR Early Release on the MMWR website (https://www.cdc.gov/mmwr).

Abstract

Background: Overweight and obesity are associated with increased risk of at least 13 different types of cancer.

Methods: Data from the United States Cancer Statistics for 2014 were used to assess incidence rates, and data from 2005 to 2014 were used to assess trends for cancers associated with overweight and obesity (adenocarcinoma of the esophagus; cancers of the breast [in postmenopausal women], colon and rectum, endometrium, gallbladder, gastric cardia, kidney, liver, ovary, pancreas, and thyroid; meningioma; and multiple myeloma) by sex, age, race/ethnicity, state, geographic region, and cancer site. Because screening for colorectal cancer can reduce colorectal cancer incidence through detection of precancerous polyps before they become cancerous, trends with and without colorectal cancer were analyzed.

Results: In 2014, approximately 631,000 persons in the United States received a diagnosis of a cancer associated with overweight and obesity, representing 40% of all cancers diagnosed. Overweight- and obesity-related cancer incidence rates were higher among older persons (ages ≥50 years) than younger persons; higher among females than males; and higher among non-Hispanic black and non-Hispanic white adults compared with other groups. Incidence rates for overweight- and obesity-related cancers during 2005–2014 varied by age, cancer site, and state. Excluding colorectal cancer, incidence rates increased significantly among persons aged 20–74 years; decreased among those aged ≥75 years; increased in 32 states; and were stable in 16 states and the District of Columbia.

Conclusions: The burden of overweight- and obesity-related cancer is high in the United States. Incidence rates of overweight- and obesity-related cancers except colorectal cancer have increased in some age groups and states.

Implications for Public Health Practice: The burden of overweight- and obesity-related cancers might be reduced through efforts to prevent and control overweight and obesity. Comprehensive cancer control strategies, including use of evidence-based interventions to promote healthy weight, could help decrease the incidence of these cancers in the United States.

Introduction

In 2013–2014, approximately one third of adults in the United States were overweight (body mass index [BMI] 25.0–29.9 kg/m^2) and approximately one third had obesity (BMI ≥30 kg/m^2) (*1*). Approximately half of U.S. residents are unaware that adults who are overweight or have obesity are at increased risk for cancer (*2*,*3*). The International Agency for Research on Cancer (IARC) states that there is sufficient evidence for an association with excess body fatness, including overweight, obesity, and weight gain, and at least 13 cancers (*3*). These cancers include adenocarcinoma of the esophagus; cancers of the breast (in postmenopausal women), colon and rectum, endometrium (corpus uterus), gallbladder, gastric cardia, kidney (renal cell), liver, ovary, pancreas, and thyroid; meningioma, and multiple myeloma. Overweight and obesity might increase cancer risk through induction of metabolic and endocrine abnormalities, including increases in inflammation and levels of insulin, insulin-like growth factor, and sex hormones (*4*).

Data compiled for the United States Cancer Statistics (USCS) data set (https://nccd.cdc.gov/uscs/) were used to calculate incidence rates in 2014 and trends during 2005–2014 for cancers associated with overweight and obesity (overweight- and obesity-related cancers). In this report, overweight- and obesity-related cancers were defined as those classified by IARC as having sufficient evidence for an association with excess body fatness.

Methods

The USCS is a compilation of data from multiple sources that is used to report official federal cancer statistics through the USCS web-based report. The USCS data set includes

Key Points

- Overweight and obesity are associated with increased risk of at least 13 different types of cancer.
- Overweight- and obesity-related cancers accounted for 40% of all cancers diagnosed in 2014.
- About 55% of cancers diagnosed in women and 24% of those diagnosed in men are overweight- and obesity-related cancers.
- The incidence of overweight- and obesity-related cancers (excluding colorectal cancer) increased significantly among persons aged 20–74 years during 2005–2014.
- The findings emphasize the importance of intensifying nationwide efforts to prevent and treat overweight and obesity.
- Multilevel approaches to comprehensive cancer control that address social determinants of health and include evidence-based interventions that address healthy weight and other cancer risk factors might help reduce the burden of cancer and other chronic diseases.
- Additional information is available at https://www.cdc.gov/vitalsigns/.

cancer incidence data from CDC's National Program of Cancer Registries (NPCR) and the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program. Data on new cancer cases diagnosed during 2005–2014 were obtained from population-based cancer registries affiliated with NPCR and SEER programs in each state and the District of Columbia (DC). Data from DC and all states met USCS publication criteria for 2014, covering 100% of the U.S. population; all states except Nevada met USCS publication criteria each year during 2005–2014, covering approximately 99% of the U.S. population.* Cancer site for cases was classified by anatomic site and histology.† Only cases of invasive cancer were included. Postmenopausal breast cancer was defined as breast cancer diagnosed in women aged ≥50 years.

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 and with support from National Cancer Institute, and aggregated to the state and national levels.§ Race bridging is a method used to make multiple-race and single-race data collection systems sufficiently comparable to permit estimation and analysis of race-specific statistics (https://www.cdc.gov/nchs/data/series/sr_02/sr02_135.pdf). Ninety-five percent confidence intervals for rates are presented to allow for informal comparisons among rates, without specifying a referent group. Joinpoint regression (https://surveillance.cancer.gov/joinpoint/), which allowed different slopes for more than one period, was used to calculate changes in rates; trends were quantified by average annual percent change. Because screening for colorectal cancer can reduce colorectal cancer incidence through detection of precancerous polyps before they become cancerous (https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/colorectal-cancer-screening2?ds), trends with and without colorectal cancer were analyzed. Rates were estimated by sex, age, race/ethnicity, and U.S. Census region. Trends were age-adjusted and estimated by cancer site, sex, and state. To examine the impact of the change in rates, the number of cases expected during 2006–2014 if rates had remained at 2005 levels was subtracted from the actual number of cases during this period.

Results

In 2014, approximately 631,604 persons in the United States received a diagnosis of an overweight- or obesity-related cancer (Table 1). This represents 40% of the nearly 1.6 million cancers diagnosed each year (55% of the 799,734 cancers among women and 24% of the 796,752 cancers among males). Overweight- and obesity-related cancer incidence rates were higher among older persons (ages ≥50 years) than younger persons and two thirds of cases occurred among persons aged 50–74 years. The overweight- and obesity-related cancer incidence rate was higher among females (218.1 per 100,000 population) than among males (115.0 per 100,000), partially because endometrial, ovarian, and postmenopausal female breast cancers accounted for 42% (268,091) of overweight- and obesity-related cancers. The rates also varied by race/ethnicity, with higher incidence among non-Hispanic blacks (black) and non-Hispanic whites (white) compared with other groups; however, black males and American Indian/Alaska Native males had higher incidence rates than did white males.

* Cancer registries demonstrated that cancer incidence data were of high quality by meeting five United States Cancer Statistics publication 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 inter-field computerized edits that test the validity and logic of data components (https://www.cdc.gov/cancer/npcr/uscs/technical_notes/criteria.htm).

† Cases were first classified by anatomic site using the *International Classification of Diseases for Oncology, Third Edition* (http://codes.iarc.fr/) then cases with hematopoietic histologies were classified using the *World Health Organization Classification of Tumours of Haematopoietic and Lymphoid Tissues, Fourth Edition* (http://www.bloodjournal.org/content/117/19/5019?sso-checked=true#T1).

§ 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/index.html).

TABLE 1. Number and annual age-adjusted rate* of overweight- and obesity-related invasive cancer cases,† by selected characteristics — United States,§ 2014

Characteristic	Total		Males		Females	
	No.	Rate (95% CI)	No.	Rate (95% CI)	No.	Rate (95% CI)
Total	**631,604**	**169.7 (169.3–170.1)**	**194,727**	**115.0 (114.5–115.5)**	**436,877**	**218.1 (217.4–218.7)**
Age group (yrs)						
<20	2,230	2.7 (2.6–2.8)	719	1.7 (1.6–1.8)	1,511	3.8 (3.6–4.0)
20–49	60,386	47.2 (46.8–47.6)	20,884	32.5 (32.1–32.9)	39,502	62.1 (61.4–62.7)
50–64	240,299	383.6 (382.0–385.1)	71,518	235.3 (233.6–237.0)	168,781	523.3 (520.8–525.8)
65–74	173,764	658.4 (655.3–661.5)	54,313	440.0 (436.3–443.7)	119,451	850.3 (845.5–855.2)
≥75	154,925	782.1 (778.2–786.0)	47,293	592.1 (586.8–597.5)	107,632	910.4 (904.9–915.8)
Race/Ethnicity¶						
White	470,789	170.9 (170.4–171.4)	144,456	114.2 (113.6–114.8)	326,333	222.3 (221.5–223.1)
Black	71,847	186.5 (185.1–188.0)	22,129	134.2 (132.3–136.1)	49,718	226.3 (224.3–228.4)
American Indian/Alaska Native	3,970	162.5 (157.2–167.9)	1,376	121.9 (115.1–129.0)	2,594	197.3 (189.5–205.3)
Asian/Pacific Islander	23,193	128.4 (126.7–130.1)	6,904	87.7 (85.6–89.9)	16,289	162.2 (159.7–164.8)
Hispanic	55,778	150.6 (149.3–152.0)	17,990	108.8 (107.1–110.6)	37,788	188.0 (186.0–189.9)
Census region††						
Northeast	127,436	185.3 (184.2–186.3)	37,739	122.8 (121.5–124.0)	89,697	239.2 (237.6–240.9)
Midwest	140,687	173.8 (172.9–174.8)	43,173	117.6 (116.5–118.7)	97,514	224.1 (222.7–225.6)
South	230,431	165.7 (165.0–166.4)	73,138	115.5 (114.7–116.4)	157,293	209.5 (208.4–210.5)
West	133,050	159.7 (158.8–160.6)	40,677	105.5 (104.5–106.6)	92,373	208.9 (207.5–210.3)

Abbreviation: CI = confidence interval.

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

† Overweight- and obesity-related cancers include adenocarcinoma of the esophagus; cancers of the breast [in postmenopausal women], colon and rectum, endometrium, gallbladder, gastric cardia, kidney, liver, ovary, pancreas, and thyroid; meningioma; and multiple myeloma.

§ Cancer incidence compiled from cancer registries that meet the data quality criteria for all invasive cancer sites combined (covering 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. The white, black, American Indian/Alaska Native, and Asian/Pacific Islander race categories are all non-Hispanic. Hispanic persons can be any race. Rates are not presented for those with unknown or other race or unknown ethnicity.

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

Incidence was highest in the Northeast compared with other U.S. Census regions.

Among cancers affecting both males and females, incidence rates in 2014 were higher among males than among females for colorectal cancer (44.1 per 100,000 versus 33.7 per 100,000), kidney cancer (20.9 versus 10.6), pancreatic cancer (14.4 versus 11.1), liver cancer (11.2 versus 3.4), adenocarcinoma of the esophagus (5.4 versus 0.8), multiple myeloma (7.5 versus 4.9), and gastric cardia cancer (3.6 versus 0.8) (Table 2). Females had higher rates than did males of thyroid cancer (21.3 versus 7.4) and gallbladder cancer (1.4 versus 0.8). Among the three overweight- and obesity-related cancers that affect females only, incidence rates were higher for postmenopausal breast cancer (92.6 per 100,000) than for endometrial cancer (26.5 per 100,000) and ovarian cancer (11.0 per 100,000). By site, incidence rates decreased significantly each year for meningioma (-3.8% per year), colorectal cancer (-2.9%), and ovarian cancer (-2.0%). Incidence rates increased significantly each year during this period for six cancers: thyroid cancer (4.0% per year), liver cancer (2.9%), gastric cardia cancer (1.2%), endometrial cancer (1.1%), pancreatic cancer (0.8%), and kidney cancer (0.7%). The incidence rates were stable for adenocarcinoma of the esophagus, gallbladder cancer, multiple myeloma, and postmenopausal breast cancer. The increase in risk for cancer per 1 kg/m^2 increase in BMI ranged from 1% each for thyroid and ovarian cancers to 9% for adenocarcinoma of the esophagus.

During 2005–2014, declines were observed in the overall incidence of overweight- and obesity-related cancers (-2%), colorectal cancer (-23%), and cancers not known to be related to overweight and obesity (-13%) (Table 2). Increased use of colorectal cancer screening tests likely contributed to the decline in colorectal cancer; when colorectal cancer was excluded from overweight- and obesity-related cancers, a 7% increase in overall incidence was observed. The trends varied substantially by age group: the rate for all overweight- and obesity-related cancers increased significantly among persons aged 20–49 years and 50–64 years, and decreased among those aged 65–74 years and ≥75 years; colorectal cancer rates declined in all age groups except in persons aged 20–49 years; and rates for overweight- and obesity-related cancers (excluding colorectal cancers) increased among all age groups except persons aged ≥75 years (Figure 1). Because of reductions in colorectal cancer rates, approximately 224,800 cases have been averted since 2005.

TABLE 2. Age-adjusted incidence of overweight- and obesity-related invasive cancer, changes in rates, and estimated percent increase in cancer risk associated with change in BMI, by cancer site and sex — United States,* 2005 and 2014

Cancer site	%	2005 Rate† (95% CI)	2014 Rate† (95% CI)	2005–2014 % Change in rates	2005–2014 Average annual percent change in rates§	% Increase in risk for cancer per 1 kg/m² increase in BMI¶
Breast [in postmenopausal women]	31	90.5 (90.1–91.0)	92.6 (92.2–93.0)	2	0.2	2
Colon and rectum	22	49.7 (49.4–49.9)	38.4 (38.2–38.6)	-23	-2.9§	2
Male		58.1 (57.7–58.5)	44.1 (43.7–44.4)	-24	-3.1§	
Female		43.1 (42.8–43.4)	33.7 (33.4–34.0)	-22	-2.8§	
Kidney (renal cell)	9	14.4 (14.2–14.5)	15.4 (15.2–15.5)	7	0.7§	5
Male		19.5 (19.3–19.7)	20.9 (20.7–21.1)	7	0.7§	
Female		10.2 (10.0–10.3)	10.6 (10.4–10.7)	4	0.4	
Endometrium (corpus uterus) (female only)	8	23.9 (23.7–24.1)	26.5 (26.3–26.8)	11	1.1§	8
Thyroid	8	10.3 (10.2–10.4)	14.4 (14.3–14.6)	40	4.0§	1
Male		5.3 (5.1–5.4)	7.4 (7.2–7.5)	40	4.0§	
Female		15.2 (15.0–15.4)	21.3 (21.1–21.5)	40	4.0§	
Pancreas	7	11.7 (11.6–11.9)	12.6 (12.5–12.7)	7	0.8§	2
Male		13.3 (13.1–13.5)	14.4 (14.2–14.5)	8	0.8§	
Female		10.5 (10.3–10.6)	11.1 (10.9–11.2)	6	0.7§	
Multiple myeloma	4	5.6 (5.5–5.7)	6.0 (6.0–6.1)	8	1.1	2
Male		6.9 (6.7–7.0)	7.5 (7.3–7.6)	9	1.2§	
Female		4.6 (4.5–4.8)	4.9 (4.8–5.0)	6	1.1§	
Liver	4	5.5 (5.4–5.6)	7.0 (7.0–7.1)	29	2.9§	5
Male		8.8 (8.6–8.9)	11.2 (11.0–11.3)	28	2.9§	
Female		2.7 (2.6–2.8)	3.4 (3.3–3.5)	26	2.5§	
Ovary (female only)	3	13.1 (12.9–13.2)	11.0 (10.8–11.2)	-16	-2.0§	1
Adenocarcinoma of the esophagus	2	2.9 (2.8–2.9)	2.9 (2.8–2.9)	-1	-0.5	9
Male		5.5 (5.4–5.7)	5.4 (5.2–5.5)	-3	-0.7§	
Female		0.8 (0.7–0.8)	0.8 (0.7–0.8)	2	-0.4	
Gastric cardia	1	1.9 (1.9–2.0)	2.1 (2.0–2.1)	8	1.2§	4
Male		3.4 (3.3–3.5)	3.6 (3.5–3.7)	7	1.1§	
Female		0.8 (0.7–0.8)	0.8 (0.8–0.9)	6	0.8§	
Gallbladder	1	1.1 (1.1–1.2)	1.1 (1.1–1.2)	-1	-0.1	5
Male		0.8 (0.7–0.8)	0.8 (0.8–0.8)	3	0.1	
Female		1.4 (1.4–1.5)	1.4 (1.3–1.5)	-1	-0.1	
Meningioma	<1	0.1 (0.1–0.2)	0.1 (0.1–0.1)	-29	-3.8§	4
Male		0.1 (0.1–0.1)	0.1 (0.1–0.1)	-17	-2.7§	
Female		0.2 (0.1–0.2)	0.1 (0.1–0.1)	-35	-4.0§	
All overweight- and obesity-related cancers	—	173 (173–174)	170 (169–170)	-2	-0.3§	—
All overweight- and obesity-related cancers except colorectal cancer	—	123 (123–124)	132 (131–132)	7	0.8§	—
Cancers not related to overweight and obesity	—	306 (305–306)	267 (267–268)	-13	-1.4§	—

Abbreviations: BMI = body mass index calculated as weight in kilograms/height in meters squared (kg/m²); CI = confidence interval.

* Cancer incidence compiled from cancer registries that meet the data quality criteria for all invasive cancer sites combined for all years during the period 2005 to 2014 (covering 99% of the U.S. population).

† Per 100,000 persons, age-adjusted to the 2000 U.S. standard population.

§ Significant at p<0.05. Trends were measured with average annual percent change in rates and were considered to increase or decrease if p<0.05; otherwise trends were considered stable.

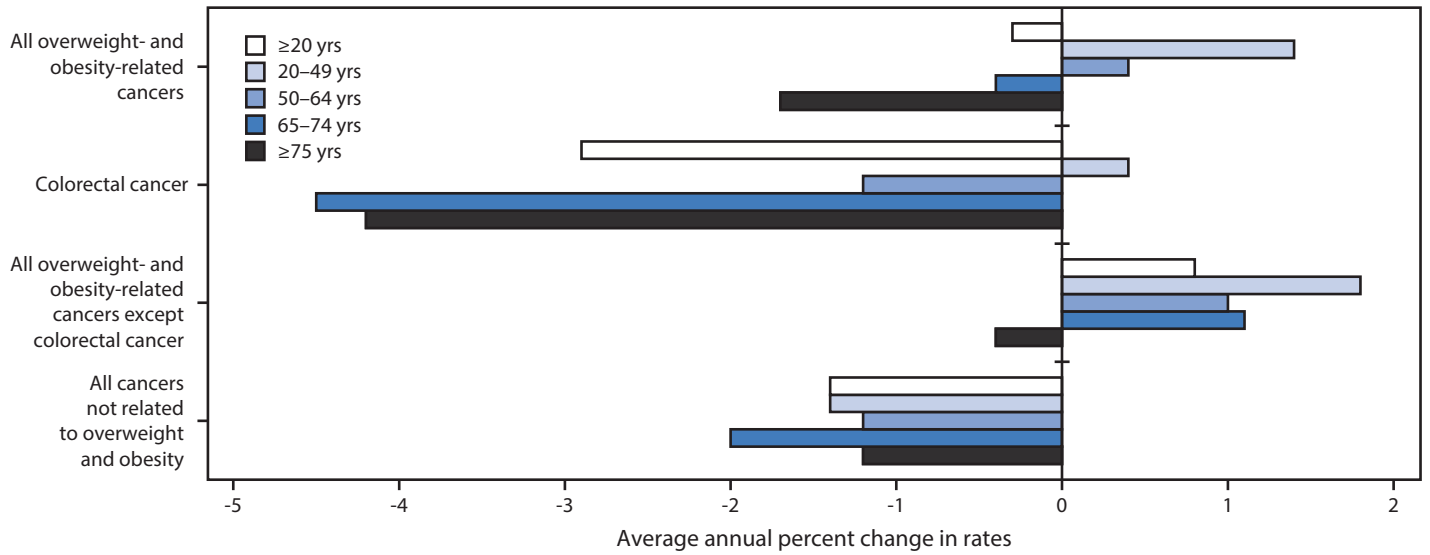
¶ Based on relative risk estimates from pooled epidemiologic studies (reviewed in http://www.nejm.org/doi/full/10.1056/NEJMsr1606602) and in the World Cancer Research Fund Continuous Update Project (http://www.wcrf.org/int/research-we-fund/continuous-update-project-cup).

However, during this same period, 211,800 excess cases from other overweight- and obesity-related cancers have occurred. Incidence rates of overweight- and obesity-related cancers (excluding colorectal cancer) increased significantly in 32 states (0.3%–1.8%), and did not change in 16 states and the District of Columbia (Figure 2).

Conclusions and Comments

Overweight- and obesity-related cancers accounted for 40% of all cancers diagnosed in 2014, and varied substantially across demographic groups. Endometrial, ovarian, and postmenopausal female breast cancers accounted for 42% of new cases of overweight-and obesity-related cancers in 2014,

FIGURE 1. Average annual percent change* in overweight- and obesity-related invasive cancer incidence rates† among adults — United States,§ 2005–2014



* Average annual percent change (AAPC) was calculated using joinpoint regression, which allowed different slopes for two periods; the year at which slopes changed could vary by age. All AAPCs were significantly different from zero ($p<0.05$) except for colorectal cancer in persons aged 20–49 years.

† Overweight- and obesity-related cancer (adenocarcinoma of the esophagus; cancers of the breast [in postmenopausal women], colon and rectum, endometrium, gallbladder, gastric cardia, kidney, liver, ovary, pancreas, and thyroid; meningioma; and multiple myeloma) rates were calculated with and without colorectal cancer because colorectal cancer screening can detect precancerous polyps before they become cancerous, which might affect cancer incidence.

§ Cancer incidence compiled from cancer registries that meet the data quality criteria for all invasive cancer sites combined for each year during the period 2005–2014 (covering 99% of the U.S. population).

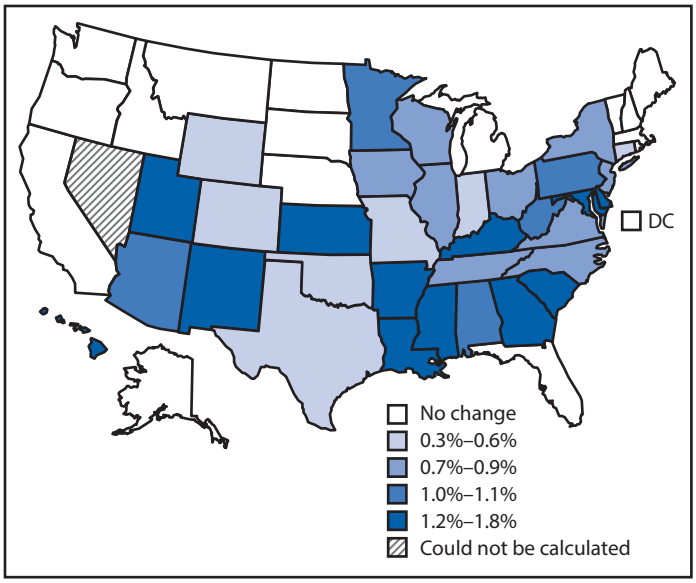
which is reflected in the higher overall incidence of overweight- and obesity-related cancers among females. For cancers that occurred among both males and females, however, the incidence of most cancers was higher in males.

Meningioma declined the most (-3.8% per year) during 2005–2014; however, this cancer accounted for <1% of overweight- and obesity-related cancers among males and females. The second largest decline was in the rate of colorectal cancer, which accounted for approximately 22% of overweight- and obesity-related cancers; this trend likely influenced the overall decline in the incidence of overweight- and obesity-related cancers during 2005–2014. National data have demonstrated an increase in colorectal cancer screening (*5*), which might have contributed to the decline in colorectal cancer incidence through detection of precancerous polyps, which can then be removed before becoming cancerous. When colorectal cancer was excluded from the trend analysis, overweight- and obesity-related cancer incidence increased among all age groups except persons aged ≥75 years. The increase in obesity-related cancer incidence coincides with an increase in the prevalence of obesity since 1960 in the United States with larger absolute percentage increases from 1960 to 2004 than from 2005 to 2014 (*1*). The prevalence of overweight during this later period remained stable. These historical and current trends in overweight and obesity and cancers related to excess weight reflect the continued need for public health strategies to prevent and control overweight and obesity in children and adults and help communities make it easier for people to be physically active and eat healthfully.

There is consistent evidence that a high BMI is associated with cancer risk. Persons who are overweight or have obesity are nearly twice as likely as are healthy-weight (BMI = 18.5–24.9 kg/m^2) persons to develop adenocarcinoma of the esophagus and cancers of the gastric cardia, liver, and kidney (*6*–*9*). Persons who have obesity are approximately 30% more likely to develop colorectal cancer than are persons with healthy weight (*10*). Women who are overweight or have obesity are approximately two to four times as likely as are women with healthy weight to develop endometrial cancer (*11*).

Observational studies have provided evidence that even a 5-kg (11 pound) increase in weight since early adulthood is associated with increased risk for overweight- and obesity-related cancers (*12*). Maintaining a healthy weight throughout life has been associated with a reduction in risk of these cancers (*3*). However, the population effect of weight loss interventions on cancer risk might not be observable for at least a decade (*4*). In studies evaluating the effect of weight change on risks for endometrial cancer and breast cancer after long-term follow-up, weight loss was associated with reduced risks for both types of cancer among postmenopausal women (*13*,*14*).

FIGURE 2. Average annual percent change in incidence of overweight- and obesity-related cancers,* by quartile — United States, 2005–2014



* Except colorectal cancer.

Without intensified nationwide efforts to prevent and treat overweight and obesity, the high prevalence of excess weight might impede further declines in overall cancer incidence (*15*). These efforts include investing in addressing both social and behavioral determinants of health, such as unemployment and disparities in education and housing, to achieve better population health (https://nam.edu/addressing-social-determinants-of-health-and-health-disparities-a-vital-direction-for-health-and-health-care/). Eating a healthy diet and engaging in sufficient physical activity are important components of behavioral strategies to maintain a healthy weight. Population-based strategies to prevent and reduce overweight and obesity include helping persons of all ages meet dietary (https://health.gov/dietaryguidelines/2015/guidelines) and physical activity (https://health.gov/PAGuidelines) guidelines by supporting healthy eating and active living in a variety of settings, including communities, worksites, schools, and early care and education facilities. Strategies to provide support for these settings have been recommended by a number of public health entities including CDC (https://www.cdc.gov/mmwr/preview/mmwrhtml/rr5807a1.htm), the National Academy of Medicine (*16*), and the Community Preventive Services Task Force (https://www.thecommunityguide.org/topic/obesity). Health care providers could encourage patients to maintain healthy weights throughout their lifespans. To help treat obesity, the U.S. Preventive Services Task Force recommends that clinicians screen all adults for obesity and either offer patients who have obesity intensive, multicomponent behavioral interventions or refer them to programs that offer these services (https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/obesity-in-adults-screening-and-management); similar recommendations exist for children aged ≥6 years (https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/obesity-in-children-and-adolescents-screening).

The CDC's National Comprehensive Cancer Control Program supports comprehensive cancer control efforts in all 50 states, DC, eight tribes and tribal organizations, and seven U.S. territories and Pacific Island jurisdictions; these efforts include policy, systems, and environmental changes that promote physical activity and healthy food options in communities. A review of cancer control plans implemented by grantees revealed that 89% include goals or strategies related to nutrition or physical activity to reduce cancer risk, with 82% including both (*17*). Other CDC programs, such as the State Public Health Action's Program, address diet, physical activity, and obesity more broadly (https://www.cdc.gov/nccdphp/dnpao/state-local-programs/state-public-health-action.html). Maintaining and strengthening these programmatic activities might help reduce the burden of overweight- and obesity-related cancer.

The findings in this report are subject to at least five limitations. First, the weights and BMI histories of cancer patients were not known. Second, because race and ethnicity data are abstracted from medical records, they are subject to misclassification (https://www.cdc.gov/cancer/npcr/uscs/technical_notes/interpreting/race.htm). Third, whereas IARC's most recent report was used to define overweight- and obesity-related cancer, this might underestimate the actual burden, because evidence is still accumulating related to the association of overweight and obesity with other cancers (*3*). Fourth, many different risk factors might contribute to development of overweight- and obesity-related cancers, such as genetic mutations; chronic infections; and tobacco, hormone, and alcohol use (*2*). Changes in these other risks, as well as in cancer screening rates, might have affected the number of cancer cases and the trends described in this report. Finally, although this report tracks overweight- and obesity-related cancers, it does not estimate what proportion of these cancers are attributable to overweight and obesity.

The incidence of overweight- and obesity-related cancers (excluding colorectal cancer) increased significantly among persons aged 20–74 years during 2005–2014, mirroring increases of obesity observed since 1960 (*1*). Multilevel approaches to comprehensive cancer control that address social determinants of health and include evidence-based interventions that address healthy weight and other cancer risk factors might help reduce the burden of cancer and other chronic diseases in the United States.

Conflict of Interest

No conflicts of interest were reported.

[1]Division of Cancer Prevention and Control, CDC; [2]Division of Nutrition, Physical Activity, and Obesity, CDC; [3]Division of Cancer Control and Population Sciences, National Cancer Institute, Rockville, Maryland.

Corresponding author: C. Brooke Steele, bsteele1@cdc.gov, 770-488-4261.

References

1. National Center for Health Statistics. Prevalence of overweight, obesity, and extreme obesity among adults aged 20 and over: United States, 1960–1962 through 2013–2014. Hyattsville, MD: US Department of Health and Human Services, CDC, National Center for Health Statistics; 2016. https://www.cdc.gov/nchs/data/hestat/obesity_adult_13_14/obesity_adult_13_14.htm
2. American Institute for Cancer Research. The AICR 2015 cancer risk survey report. Washington, DC: American Institute for Cancer Research; 2015. http://www.aicr.org/assets/docs/pdf/education/aicr-awareness-report-2015.pdf
3. Lauby-Secretan B, Scoccianti C, Loomis D, Grosse Y, Bianchini F, Straif K; International Agency for Research on Cancer Handbook Working Group. Body fatness and cancer—viewpoint of the IARC Working Group. N Engl J Med 2016;375:794–8. https://doi.org/10.1056/NEJMsr1606602
4. Renehan AG, Zwahlen M, Egger M. Adiposity and cancer risk: new mechanistic insights from epidemiology. Nat Rev Cancer 2015;15:484–98. https://doi.org/10.1038/nrc3967
5. White A, Thompson TD, White MC, et al. Cancer screening test use—United States, 2015. MMWR Morb Mortal Wkly Rep 2017;66:201–6. https://doi.org/10.15585/mmwr.mm6608a1
6. Hoyo C, Cook MB, Kamangar F, et al. Body mass index in relation to oesophageal and oesophagogastric junction adenocarcinomas: a pooled analysis from the International BEACON Consortium. Int J Epidemiol 2012;41:1706–18. https://doi.org/10.1093/ije/dys176
7. Chen Y, Liu L, Wang X, et al. Body mass index and risk of gastric cancer: a meta-analysis of a population with more than ten million from 24 prospective studies. Cancer Epidemiol Biomarkers Prev 2013;22:1395–408. https://doi.org/10.1158/1055-9965.EPI-13-0042
8. Chen Y, Wang X, Wang J, Yan Z, Luo J. Excess body weight and the risk of primary liver cancer: an updated meta-analysis of prospective studies. Eur J Cancer 2012;48:2137–45. https://doi.org/10.1016/j.ejca.2012.02.063
9. Wang F, Xu Y. Body mass index and risk of renal cell cancer: a dose-response meta-analysis of published cohort studies. Int J Cancer 2014;135:1673–86. https://doi.org/10.1002/ijc.28813
10. Ma Y, Yang Y, Wang F, et al. Obesity and risk of colorectal cancer: a systematic review of prospective studies. PLoS One 2013;8:e53916. https://doi.org/10.1371/journal.pone.0053916
11. Setiawan VW, Yang HP, Pike MC, et al.; Australian National Endometrial Cancer Study Group. Type I and II endometrial cancers: have they different risk factors? J Clin Oncol 2013;31:2607–18. https://doi.org/10.1200/JCO.2012.48.2596
12. Keum N, Greenwood DC, Lee DH, et al. Adult weight gain and adiposity-related cancers: a dose-response meta-analysis of prospective observational studies. J Natl Cancer Inst 2015;107:djv088. https://doi.org/10.1093/jnci/djv088
13. Luo J, Chlebowski RT, Hendryx M, et al. Intentional weight loss and endometrial cancer risk. J Clin Oncol 2017;35:1189–93. https://doi.org/10.1200/JCO.2016.70.5822
14. Eliassen AH, Colditz GA, Rosner B, Willett WC, Hankinson SE. Adult weight change and risk of postmenopausal breast cancer. JAMA 2006;296:193–201. https://doi.org/10.1001/jama.296.2.193
15. Eheman C, Henley SJ, Ballard-Barbash R, et al. Annual report to the nation on the status of cancer, 1975–2008, featuring cancers associated with excess weight and lack of sufficient physical activity. Cancer 2012;118:2338–66. https://doi.org/10.1002/cncr.27514
16. Institute of Medicine. Accelerating progress in obesity prevention: solving the weight of the nation. Washington, DC: National Academies Press; 2012.
17. Puckett M, Neri A, Underwood JM, Stewart SL. Nutrition and physical activity strategies for cancer prevention in current National Comprehensive Cancer Control Program plans. J Community Health 2016;41:1013–20. https://doi.org/10.1007/s10900-016-0184-8

Errata

Vol. 66, No. 26

In the report "Tobacco Use in Top-Grossing Movies — United States, 2010–2016," on page 683, the last sentence of the third paragraph of the Discussion should have read "During 2010–2016, approximately 24 states awarded approximately **$1.7 billion** in public subsidies, such as tax credits, to productions of movies with tobacco incidents, including youth-rated movies.**"

Vol. 66, No. 36

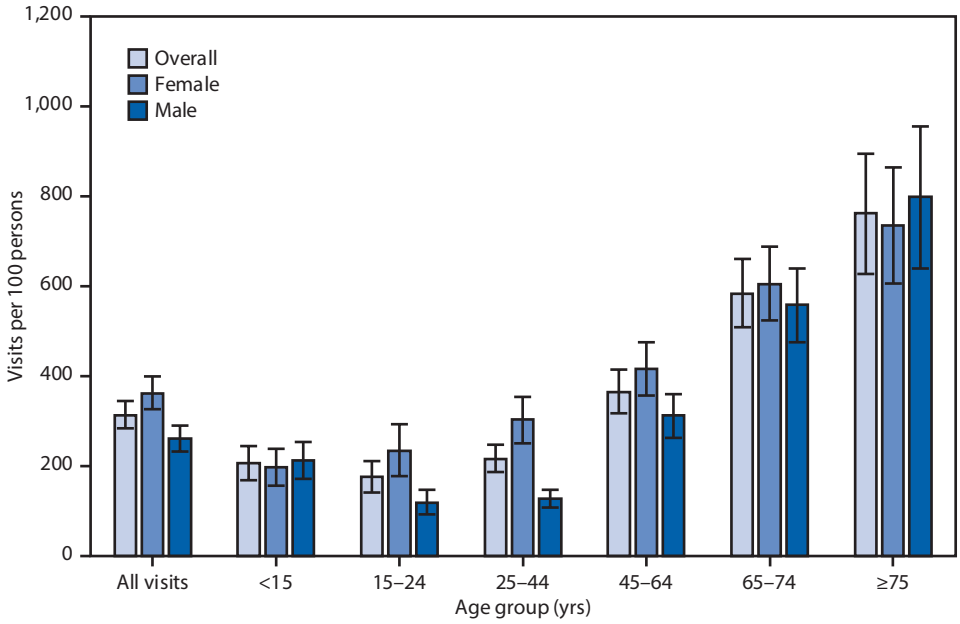
The announcement "Childhood Cancer Awareness Month — September 2017," on page 963, should have included the following as the third and last reference:

Oeffinger KC, Mertens AC, Sklar CA, et al.; Childhood Cancer Survivor Study. Chronic health conditions in adult survivors of childhood cancer. N Engl J Med 2006;355:1572–82.

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Rate of Visits* to Office-Based Physicians,† By Patient Age and Sex — National Ambulatory Medical Care Survey, United States, 2015



* With 95% confidence intervals indicated with error bars. Visit rates are based on the July 1, 2015, set of estimates of the civilian noninstitutional population of the United States as developed by the Population Division, U.S. Census Bureau.

† Based on a sample of visits to nonfederally employed office-based physicians who are primarily engaged in direct patient care. Physicians in the specialties of anesthesiology, pathology, and radiology were excluded from the survey.

In 2015, the visit rate to office-based physicians was 313 visits per 100 persons. The rate was higher for females (362 per 100) compared with males (262 per 100). For patients in age groups between 15 years and 64 years, the rate for females was higher than the rate for males; for those aged ≥65 years no difference by sex was found. Rates increased with age after the age of 15 years for males and females.

Source: National Center for Health Statistics, National Ambulatory Medical Care Survey, 2015. https://www.cdc.gov/nchs/ahcd/ahcd_questionnaires.htm.

Reported by: Jill J. Ashman, PhD, JAshman@cdc.gov, 301-458-4439; Pinyao Rui, MPH.

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

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

ISSN: 0149-2195 (Print)