

## Chronic Obstructive Pulmonary Disease and Smoking Status — United States, 2017

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Cigarette smoking is the leading cause of chronic obstructive pulmonary disease (COPD) in the United States; however, an estimated one fourth of adults with COPD have never smoked (1). CDC analyzed state-specific Behavioral Risk Factor Surveillance System (BRFSS) data from 2017, which indicated that, overall among U.S. adults, 6.2% (age-adjusted) reported having been told by a health care professional that they had COPD. The age-adjusted prevalence of COPD was 15.2% among current cigarette smokers, 7.6% among former smokers, and 2.8% among adults who had never smoked. Higher prevalences of COPD were observed in southeastern and Appalachian states, regardless of smoking status of respondents. Whereas the strong positive correlation between state prevalence of COPD and state prevalence of current smoking was expected among current and former smokers, a similar relationship among adults who had never smoked suggests secondhand smoke exposure as a potential risk factor for COPD. Continued promotion of smoke-free environments might reduce COPD among both those who smoke and those who do not.

Data from 418,378 adult respondents to the 2017 BRFSS survey in the 50 states and the District of Columbia (DC) were analyzed. BRFSS is an annual state-based, random-digit-dialed cellular and landline telephone survey of the noninstitutionalized U.S. population aged  $\geq 18$  years and is conducted by state health departments in collaboration with CDC.\* Response rates for BRFSS are calculated using standards set by the American Association for Public Opinion Research (AAPOR) Response Rate Formula #4.† The response rate is the number of respondents who completed the survey as a proportion

of all eligible and likely eligible persons. The median survey response rate for all states and DC in 2017 was 45.9% and ranged from 30.6% to 64.1%.§ COPD was defined by an affirmative response to the question “Has a doctor, nurse, or other health professional ever told you that you had chronic obstructive pulmonary disease or COPD, emphysema, or chronic bronchitis?” Persons were considered to have never smoked if they reported never smoking or smoked less than 100 cigarettes during their lifetime. Former smokers had smoked at least 100 cigarettes in their life, but were not current smokers. Current smokers had smoked at least 100 cigarettes and currently smoked some days or every day.

§ [https://www.cdc.gov/brfss/annual\\_data/2017/pdf/2017-sdqr-508.pdf](https://www.cdc.gov/brfss/annual_data/2017/pdf/2017-sdqr-508.pdf).

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\* <https://www.cdc.gov/brfss/about/index.htm>.

† [http://www.aapor.org/AAPOR\\_Main/media/publications/Standard-Definitions20169theditionfinal.pdf](http://www.aapor.org/AAPOR_Main/media/publications/Standard-Definitions20169theditionfinal.pdf).



Age-specific and age-adjusted<sup>‡</sup> percentages and 95% confidence intervals (CIs) of adults with diagnosed COPD for all respondents and by smoking status were calculated for groups defined by selected sociodemographic characteristics, health characteristics, and state. Comparisons were made between these groups using t-tests with statistical significance set at  $p < 0.05$ . State-specific age-adjusted current smoking prevalence was compared with state-specific age-adjusted COPD prevalence using Pearson correlation for all respondents and groups defined by smoking status. All analyses were conducted using SAS-callable SUDAAN (version 11.0.1; RTI International) to account for the stratified, complex cluster sampling design of the survey.

Overall age-adjusted prevalence of COPD was 6.2% in 2017 and was higher among women, older adults, and American Indians/Alaska Natives. Prevalence was also higher among those with less education, those who lived in more rural counties, those with a history of asthma, those who were underweight or obese, those who reported no leisure-time physical activity in the past 30 days, and those with additional chronic conditions (Table 1). Similar patterns were observed irrespective of smoking status. Among all adults, age-adjusted prevalence of COPD ranged from 3.4% in Hawaii to 13.8% in West Virginia (Table 2) (Figure). Among current smokers, overall age-adjusted COPD prevalence was 15.2% and ranged from 7.8% in Hawaii to 25.9% in West Virginia. Among former smokers, age-adjusted COPD prevalence was 7.6% and ranged

from 4.7% in Hawaii to 15.1% in West Virginia. Among adults who never smoked, age-adjusted COPD prevalence was 2.8% and ranged from 1.6% in Minnesota to 6.0% in West Virginia. Among current smokers, COPD prevalence was highest in states in the Southeast and the Midwest. Among adults who never smoked, states with the highest COPD prevalence were concentrated in the Southeast. State-level prevalence of COPD among current smokers was strongly correlated with state-level current smoking prevalence (Pearson correlation coefficient = 0.69,  $p < 0.001$ ). State-level COPD prevalence among former smokers (Pearson correlation coefficient = 0.71,  $p < 0.001$ ) and among adults who had never smoked (Pearson correlation coefficient = 0.64,  $p < 0.001$ ) also were strongly correlated with state-level current smoking prevalence.

## Discussion

The higher COPD prevalences observed among women, older adults, American Indians/Alaska Natives, adults with less education, those with a history of asthma, and those residing in rural areas were consistent with results from previous studies (1–3). The geographic distribution also was consistent (1). These patterns were similar among adults who had never smoked. Although smoking tobacco is the main contributor to COPD in the United States, other factors might play a role in the development of COPD among nonsmokers, including secondhand smoke exposure, occupational and environmental exposures, and chronic asthma (4,5). Secondhand smoke exposure, in either childhood or as an adult, has been associated with

<sup>‡</sup>Age-adjusted to the 2000 U.S. standard population aged  $\geq 18$  years.

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TABLE 1. Age-specific and age-adjusted\* percentage of adults aged ≥18 years with COPD, by smoking status and selected characteristics — Behavioral Risk Factor Surveillance System, 2017

Characteristic	All adults	Current smokers	Former smokers	Never smokers
	% (95% CI)	% (95% CI)	% (95% CI)	% (95% CI)
<b>Overall</b>	6.2 (6.0–6.3)	15.2 (14.7–15.7)	7.6 (7.3–8.0)	2.8 (2.7–2.9)
<b>Sex</b>				
Men	5.5 (5.4–5.7)	12.4 (11.8–13.1)	6.6 (6.2–7.1)	2.3 (2.1–2.5)
Women	6.8 (6.6–7.0)	18.5 (17.8–19.3)	8.9 (8.4–9.5)	3.2 (3.0–3.4)
<b>Age group (yrs)</b>				
18–44	2.7 (2.5–2.8)	6.8 (6.2–7.3)	2.9 (2.5–3.4)	1.4 (1.3–1.6)
45–54	6.3 (6.0–6.7)	17.7 (16.4–19.1)	7.1 (6.4–8.0)	2.5 (2.2–2.9)
55–64	10.6 (10.2–11.0)	25.8 (24.4–27.3)	12.6 (11.9–13.5)	4.1 (3.7–4.5)
≥65	12.8 (12.5–13.2)	30.1 (28.5–31.8)	17.5 (16.8–18.3)	6.1 (5.7–6.5)
<b>Race/Ethnicity</b>				
White <sup>†</sup>	6.7 (6.5–6.8)	16.9 (16.3–17.5)	7.8 (7.5–8.2)	2.7 (2.5–2.8)
Black <sup>†</sup>	6.6 (6.1–7.1)	11.2 (10.0–12.6)	8.8 (7.3–10.5)	4.1 (3.6–4.7)
Hispanic	3.6 (3.2–3.9)	8.0 (6.5–9.8)	5.2 (4.2–6.3)	2.3 (1.9–2.6)
American Indian/Alaska Native <sup>†</sup>	11.9 (10.3–13.7)	21.6 (18.0–25.8)	10.9 (8.5–14.0)	5.7 (4.1–7.8)
Asian <sup>†</sup>	1.7 (1.2–2.5)	8.3 (4.3–15.3) <sup>§</sup>	1.5 (0.9–2.6)	1.2 (0.7–2.1)
Native Hawaiian/Pacific Islander <sup>†</sup>	3.3 (1.8–6.0) <sup>§</sup>	14.9 (7.3–27.9) <sup>§</sup>	4.3 (2.2–8.2) <sup>§</sup>	0.9 (0.4–2.1) <sup>§</sup>
Other/Multiracial <sup>†</sup>	9.3 (8.3–10.5)	19.4 (16.9–22.2)	10.3 (8.8–12.1)	3.9 (2.8–5.6)
<b>Education level</b>				
Less than high school diploma	10.4 (9.9–11.0)	20.0 (18.7–21.4)	12.5 (11.1–14.0)	4.0 (3.5–4.5)
High school diploma	7.4 (7.1–7.7)	14.9 (14.1–15.7)	8.8 (8.2–9.4)	3.5 (3.1–3.9)
Some college	6.5 (6.2–6.7)	14.6 (13.7–15.5)	7.3 (6.9–7.9)	3.3 (3.0–3.6)
College graduate	2.7 (2.6–2.9)	8.6 (7.8–9.6)	4.4 (3.9–4.9)	1.6 (1.4–1.7)
<b>Urban-rural status<sup>¶</sup></b>				
Large metropolitan center	4.8 (4.5–5.1)	11.8 (10.8–12.9)	6.0 (5.4–6.7)	2.6 (2.3–2.9)
Large fringe metropolitan	5.7 (5.4–6.0)	14.7 (13.6–15.9)	7.1 (6.4–7.8)	2.6 (2.4–2.9)
Medium metropolitan	6.5 (6.3–6.8)	16.1 (15.1–17.1)	8.0 (7.3–8.6)	2.9 (2.6–3.1)
Small metropolitan	7.3 (7.0–7.7)	17.0 (15.8–18.3)	9.1 (8.2–10.0)	3.0 (2.7–3.3)
Micropolitan	8.3 (7.9–8.8)	18.2 (17.0–19.4)	10.2 (9.2–11.2)	3.2 (2.8–3.5)
Noncore	8.5 (8.0–9.0)	18.8 (17.3–20.3)	9.7 (8.7–10.8)	3.6 (3.2–4.1)
<b>Ever had asthma</b>				
Yes	19.5 (19.0–20.1)	37.3 (35.9–38.9)	21.3 (20.2–22.5)	11.2 (10.6–11.9)
No	4.1 (4.0–4.2)	10.9 (10.4–11.4)	5.3 (5.0–5.6)	1.6 (1.5–1.8)
<b>Body mass index (BMI, kg/m<sup>2</sup>)</b>				
Underweight (BMI<18.5)	13.6 (11.8–15.5)	25.7 (22.3–29.5)	18.8 (13.5–25.6)	3.4 (2.1–5.5)
Normal weight (BMI = 18.5–24.9)	5.7 (5.5–6.0)	14.9 (14.1–15.8)	6.9 (6.3–7.5)	2.1 (1.8–2.4)
Overweight (BMI = 25.0–29.9)	4.9 (4.7–5.1)	12.6 (11.8–13.4)	6.5 (5.9–7.2)	2.0 (1.9–2.2)
Obesity (BMI≥30.0)	8.1 (7.8–8.4)	17.9 (16.9–19.0)	9.4 (8.8–10.0)	4.5 (4.2–4.8)
<b>Leisure-time physical activity<sup>**</sup></b>				
Yes	4.8 (4.7–5.0)	12.7 (12.1–13.3)	6.0 (5.7–6.3)	2.3 (2.2–2.5)
No	9.6 (9.3–9.9)	19.2 (18.3–20.2)	11.9 (11.0–12.9)	4.1 (3.8–4.4)
<b>Number of chronic conditions<sup>††</sup></b>				
None	2.5 (2.4–2.7)	6.5 (5.8–7.1)	3.7 (3.3–4.1)	1.2 (1.1–1.3)
1	5.8 (5.5–6.1)	13.4 (12.5–14.2)	6.5 (5.9–7.0)	2.7 (2.5–3.0)
2	12.6 (11.9–13.4)	24.5 (22.9–26.2)	13.5 (11.1–16.3)	6.4 (5.5–7.4)
3	20.2 (18.1–22.5)	32.1 (28.5–36.0)	22.6 (17.4–28.8)	11.7 (9.1–15.0)
≥4	34.4 (30.3–38.8)	45.7 (39.9–51.6)	42.6 (37.5–47.8)	25.3 (19.3–32.3)

**Abbreviations:** CI = confidence interval; COPD = chronic obstructive pulmonary disease.

\* Percentages for all characteristics except age group were age-adjusted to the 2000 U.S. standard population aged ≥18 years.

<sup>†</sup> Non-Hispanic.

<sup>§</sup> Unreliable estimate because relative standard error >0.3.

<sup>¶</sup> Classification based on the National Center for Health Statistics (NCHS) 2013 Urban-Rural Classification Scheme for Counties, which uses 2010 U.S. Census population data and the February 2013 Office of Management and Budget designations of metropolitan statistical area, micropolitan statistical area, or noncore area. [https://www.cdc.gov/nchs/data/series/sr\\_02/sr02\\_166.pdf](https://www.cdc.gov/nchs/data/series/sr_02/sr02_166.pdf).

\*\* Any leisure-time physical activity in the past 30 days.

<sup>††</sup> Chronic conditions include coronary heart disease (heart attack, angina, or coronary heart disease), stroke, diabetes, cancer, arthritis, kidney disease, and depressive disorder.

an increased risk for COPD-associated mortality (6). The 2006 Surgeon General's report on secondhand smoke concluded that although the evidence suggested a causal relationship between

exposure to secondhand smoke and COPD risk, there was insufficient evidence to state definitively that the relationship is causal (7).

**TABLE 2. Age-adjusted\* percentage of adults aged ≥18 years with diagnosed COPD, by smoking status and state — Behavioral Risk Factor Surveillance System, 2017**

State	Total (N = 418,378)	Current smokers (n = 61,855)		Former smokers (n = 118,692)		Never smoked (n = 237,831)	
	% with COPD (95% CI)	% of total (95% CI)	% with COPD (95% CI)	% of total (95% CI)	% with COPD (95% CI)	% of total (95% CI)	% with COPD (95% CI)
<b>Total</b>	<b>6.2 (6.0–6.3)</b>	<b>16.9 (16.6–17.1)</b>	<b>15.2 (14.7–15.7)</b>	<b>23.0 (22.8–23.3)</b>	<b>7.6 (7.3–8.0)</b>	<b>60.1 (59.8–60.4)</b>	<b>2.8 (2.7–2.9)</b>
Alabama	10.1 (9.2–11.2)	22.0 (20.5–23.6)	22.7 (19.7–25.9)	22.1 (20.8–23.5)	12.2 (10.2–14.5)	55.9 (54.2–57.6)	4.3 (3.6–5.1)
Alaska	6.3 (4.8–8.2)	20.8 (18.2–23.6)	14.0 (9.5–20.1)	26.4 (24.2–28.8)	5.1 (3.6–7.2)	52.8 (49.9–55.7)	3.2 (1.9–5.2)
Arizona	5.9 (5.5–6.4)	15.9 (15.1–16.8)	13.9 (12.4–15.6)	23.3 (22.5–24.2)	7.9 (6.7–9.3)	60.8 (59.7–61.9)	2.6 (2.3–3.1)
Arkansas	9.3 (8.1–10.8)	23.4 (21.1–26.0)	21.4 (17.7–25.6)	24.3 (22.2–26.6)	12.0 (7.6–18.4)	52.2 (49.5–54.9)	3.6 (2.7–4.7)
California	4.4 (3.9–4.9)	11.6 (10.6–12.7)	11.0 (8.7–13.9)	21.7 (20.6–22.8)	6.7 (5.4–8.3)	66.7 (65.3–68.0)	2.2 (1.8–2.7)
Colorado	4.2 (3.7–4.7)	14.7 (13.7–15.7)	12.1 (10.2–14.3)	25.4 (24.3–26.5)	4.9 (4.1–5.9)	59.9 (58.7–61.2)	1.7 (1.3–2.1)
Connecticut	5.3 (4.7–5.9)	13.4 (12.3–14.6)	14.7 (12.2–17.7)	24.4 (23.3–25.5)	7.2 (5.6–9.3)	62.2 (60.7–63.6)	2.6 (2.0–3.3)
DC	5.8 (5.0–6.7)	14.8 (13.5–16.2)	15.5 (11.6–20.4)	19.5 (18.1–21.0)	6.1 (4.7–8.0)	65.7 (63.9–67.5)	2.9 (2.3–3.7)
Delaware	7.3 (6.2–8.5)	18.0 (16.2–20.0)	19.2 (15.4–23.6)	23.7 (21.8–25.8)	8.8 (6.7–11.3)	58.2 (55.9–60.5)	2.5 (1.7–3.5)
Florida	7.1 (6.3–8.0)	16.8 (15.5–18.1)	15.7 (13.5–18.2)	22.4 (21.2–23.7)	8.2 (6.6–10.3)	60.8 (59.1–62.4)	3.9 (2.9–5.1)
Georgia	6.8 (6.1–7.6)	17.8 (16.4–19.2)	16.4 (13.6–19.6)	20.0 (18.8–21.3)	9.4 (6.8–12.9)	62.2 (60.6–63.9)	3.4 (2.8–4.1)
Hawaii	3.4 (3.0–3.9)	13.5 (12.4–14.8)	7.8 (5.8–10.5)	25.8 (24.3–27.2)	4.7 (3.7–5.9)	60.7 (59.1–62.3)	1.9 (1.5–2.4)
Idaho	4.7 (4.1–5.5)	14.8 (13.4–16.4)	13.1 (10.3–16.5)	23.0 (21.4–24.6)	5.6 (4.3–7.3)	62.2 (60.2–64.2)	2.1 (1.5–2.8)
Illinois	6.4 (5.7–7.3)	15.7 (14.4–17.2)	15.2 (12.5–18.4)	22.5 (21.1–23.9)	7.7 (6.1–9.7)	61.8 (60.1–63.5)	2.9 (2.3–3.7)
Indiana	8.0 (7.5–8.6)	22.5 (21.5–23.6)	18.3 (16.6–20.1)	23.9 (22.9–24.9)	8.5 (7.5–9.5)	53.6 (52.4–54.8)	3.3 (2.8–3.9)
Iowa	5.9 (5.3–6.5)	17.9 (16.8–19.1)	16.4 (14.1–19.0)	24.0 (22.9–25.2)	8.1 (5.8–11.3)	58.0 (56.7–59.4)	2.2 (1.8–2.8)
Kansas	6.2 (5.8–6.6)	18.0 (17.3–18.8)	16.3 (14.9–17.8)	23.8 (23.0–24.5)	7.9 (7.1–8.7)	58.2 (57.3–59.1)	2.4 (2.1–2.7)
Kentucky	11.3 (10.2–12.5)	25.5 (23.9–27.2)	23.7 (20.7–26.9)	24.6 (23.0–26.1)	11.3 (9.4–13.5)	49.9 (48.2–51.7)	4.3 (3.4–5.4)
Louisiana	8.4 (7.4–9.5)	23.8 (22.1–25.6)	16.4 (13.5–19.7)	22.0 (20.5–23.5)	11.2 (9.0–13.9)	54.2 (52.3–56.1)	3.5 (2.8–4.4)
Maine	6.5 (5.8–7.3)	18.7 (17.2–20.3)	16.4 (14.1–18.9)	29.0 (27.5–30.5)	8.9 (6.7–11.7)	52.3 (50.6–54.1)	1.9 (1.4–2.5)
Maryland	5.4 (4.8–6.0)	14.1 (13.1–15.2)	14.0 (11.7–16.7)	20.9 (19.9–21.9)	6.3 (5.3–7.5)	65.0 (63.7–66.3)	2.7 (2.2–3.3)
Massachusetts	5.0 (4.3–5.8)	14.1 (12.7–15.6)	15.2 (11.8–19.3)	23.5 (22.0–25.2)	5.7 (4.5–7.2)	62.4 (60.4–64.2)	1.8 (1.3–2.5)
Michigan	8.0 (7.3–8.6)	20.4 (19.3–21.5)	18.6 (16.5–20.9)	25.3 (24.2–26.4)	8.4 (7.3–9.6)	54.3 (53.0–55.6)	3.3 (2.8–4.0)
Minnesota	4.0 (3.7–4.4)	14.7 (14.0–15.5)	10.5 (9.1–12.1)	25.6 (24.7–26.4)	5.1 (4.4–5.9)	59.7 (58.7–60.7)	1.6 (1.3–2.0)
Mississippi	7.5 (6.6–8.5)	22.9 (21.0–24.9)	15.4 (12.5–18.8)	20.7 (19.1–22.3)	8.9 (6.9–11.3)	56.4 (54.3–58.6)	3.3 (2.5–4.2)
Missouri	7.9 (7.1–8.6)	21.6 (20.2–23.2)	19.1 (16.6–21.8)	24.8 (23.4–26.3)	8.6 (7.3–10.0)	53.6 (51.8–55.3)	3.1 (2.5–3.7)
Montana	5.7 (4.9–6.5)	18.4 (16.9–20.0)	12.9 (10.3–15.9)	25.8 (24.2–27.5)	7.1 (5.8–8.6)	55.8 (53.9–57.7)	2.3 (1.7–3.0)
Nebraska	5.3 (4.8–5.8)	15.9 (14.9–16.9)	14.6 (12.6–16.9)	24.0 (22.9–25.1)	6.4 (5.4–7.5)	60.1 (58.8–61.4)	2.2 (1.8–2.7)
Nevada	6.5 (5.5–7.6)	17.5 (15.6–19.6)	14.4 (10.9–18.8)	22.7 (20.8–24.8)	7.9 (5.9–10.5)	59.8 (57.4–62.2)	3.2 (2.3–4.4)
New Hampshire	6.0 (5.2–7.0)	17.0 (15.2–19.0)	16.4 (13.0–20.6)	28.3 (26.5–30.1)	7.2 (5.7–9.1)	54.7 (52.5–56.9)	2.5 (1.9–3.3)
New Jersey	5.8 (5.1–6.5)	14.1 (12.9–15.4)	12.8 (10.6–15.3)	23.9 (22.6–25.3)	6.3 (5.2–7.7)	62.0 (60.4–63.6)	3.6 (2.8–4.6)
New Mexico	5.6 (4.9–6.4)	17.9 (16.4–19.4)	13.2 (10.7–16.3)	22.9 (21.4–24.4)	7.0 (5.5–9.0)	59.3 (57.4–61.0)	2.5 (1.9–3.2)
New York	5.0 (4.5–5.5)	14.4 (13.5–15.4)	11.9 (10.2–13.8)	22.1 (21.1–23.2)	5.8 (5.0–6.8)	63.5 (62.2–64.7)	2.8 (2.3–3.3)
North Carolina	7.3 (6.4–8.2)	17.5 (16.0–19.1)	16.4 (13.4–20.0)	24.9 (23.3–26.5)	7.7 (6.3–9.4)	57.7 (55.8–59.6)	3.5 (2.8–4.5)
North Dakota	4.8 (4.2–5.4)	18.9 (17.6–20.3)	12.5 (10.4–15.1)	25.2 (23.9–26.6)	4.8 (3.9–5.9)	55.8 (54.2–57.5)	1.8 (1.4–2.4)
Ohio	7.6 (6.9–8.2)	22.1 (20.8–23.4)	16.7 (14.7–18.8)	23.4 (22.2–24.5)	9.5 (8.1–11.1)	54.6 (53.1–56.0)	2.9 (2.4–3.6)
Oklahoma	8.1 (7.3–8.9)	20.5 (19.1–22.0)	17.7 (15.3–20.4)	23.8 (22.5–25.2)	10.6 (9.0–12.5)	55.7 (54.0–57.4)	3.2 (2.6–3.9)
Oregon	4.9 (4.3–5.6)	16.7 (15.4–18.1)	12.6 (10.3–15.5)	24.5 (23.2–25.9)	6.2 (4.7–8.0)	58.8 (57.1–60.4)	2.0 (1.5–2.6)
Pennsylvania	5.9 (5.3–6.7)	19.7 (18.3–21.1)	11.6 (9.5–14.0)	25.6 (24.2–27.0)	8.8 (7.2–10.7)	54.8 (53.1–56.5)	2.2 (1.7–2.9)
Rhode Island	7.0 (6.1–8.1)	15.5 (13.9–17.3)	16.2 (13.0–20.0)	26.7 (25.0–28.4)	10.2 (7.8–13.2)	57.8 (55.7–59.8)	2.5 (1.9–3.4)
South Carolina	7.2 (6.6–7.9)	19.7 (18.5–20.9)	16.9 (14.9–19.2)	25.2 (24.1–26.4)	8.0 (6.5–9.8)	55.1 (53.7–56.5)	3.5 (2.9–4.2)
South Dakota	4.4 (3.6–5.4)	20.6 (18.5–22.8)	10.2 (7.3–14.2)	25.0 (23.0–27.1)	5.0 (3.8–6.7)	54.5 (52.1–56.8)	2.0 (1.3–2.9)
Tennessee	8.9 (8.0–9.8)	23.3 (21.6–25.1)	19.7 (17.2–22.5)	22.8 (21.3–24.3)	9.9 (8.2–11.9)	54.0 (52.0–55.9)	3.7 (2.8–4.8)
Texas	4.8 (4.1–5.7)	16.0 (14.5–17.5)	13.3 (10.3–17.1)	19.9 (18.5–21.4)	6.3 (4.7–8.4)	64.1 (62.2–65.9)	2.4 (1.8–3.2)
Utah	4.1 (3.6–4.6)	9.0 (8.3–9.8)	12.3 (9.9–15.3)	15.6 (14.7–16.5)	6.1 (5.0–7.4)	75.4 (74.3–76.4)	2.4 (2.0–2.9)
Vermont	5.7 (5.1–6.4)	17.3 (15.8–18.9)	17.3 (14.6–20.4)	27.5 (26.0–29.1)	6.2 (5.1–7.5)	55.2 (53.3–57.0)	1.9 (1.5–2.4)
Virginia	6.6 (5.9–7.4)	16.8 (15.7–18.0)	16.2 (13.8–19.0)	23.1 (21.9–24.3)	9.1 (6.8–11.9)	60.1 (58.7–61.6)	2.9 (2.4–3.6)
Washington	5.4 (5.0–6.0)	13.8 (13.0–14.7)	15.5 (13.3–17.9)	26.3 (25.4–27.3)	7.1 (6.0–8.3)	59.8 (58.7–61.0)	2.0 (1.7–2.4)
West Virginia	13.8 (12.7–15.0)	28.1 (26.4–29.9)	25.9 (23.3–28.8)	24.4 (22.9–25.9)	15.1 (12.6–18.0)	47.5 (45.7–49.4)	6.0 (5.0–7.3)
Wisconsin	4.7 (4.0–5.5)	16.7 (15.2–18.2)	14.0 (11.1–17.4)	25.0 (23.4–26.6)	4.9 (3.9–6.2)	58.4 (56.5–60.2)	1.9 (1.4–2.6)
Wyoming	6.1 (5.3–6.9)	19.2 (17.6–21.0)	12.9 (10.3–16.1)	25.1 (23.5–26.8)	8.7 (7.1–10.6)	55.7 (53.7–57.7)	2.3 (1.8–3.0)

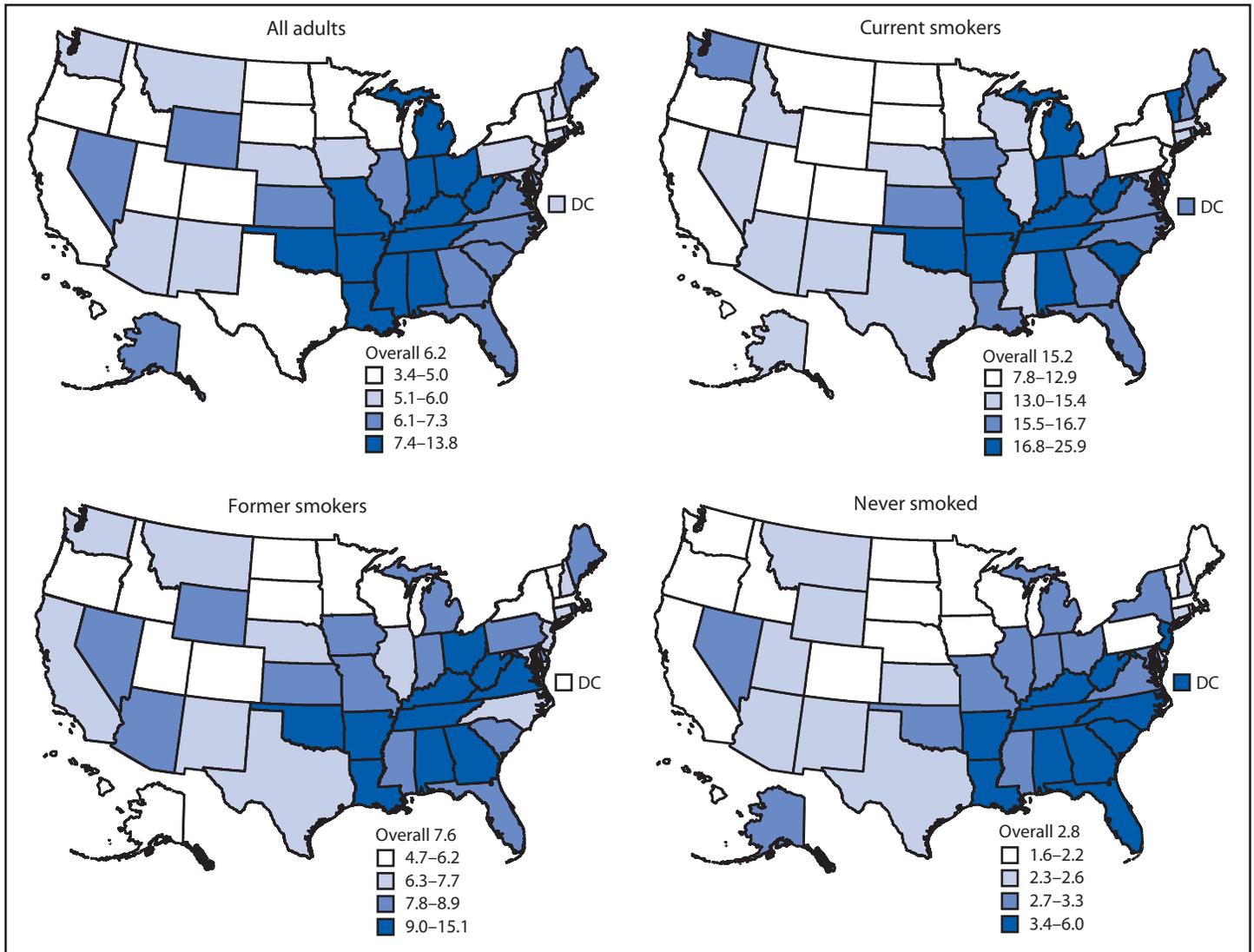
**Abbreviations:** CI = confidence interval; COPD = chronic obstructive pulmonary disease; DC = District of Columbia.

\* Age-adjusted to the 2000 U.S. standard population aged ≥18 years.

In the current analysis, the geographic distribution of high COPD prevalence was similar for current smokers and adults who never smoked. There is also a strong correlation between state-level prevalences of COPD among adults who never

smoked and state-level prevalence of current smoking. This could reflect that in certain regions adults who never smoked might be more likely to be exposed to secondhand smoke. Among the states in the highest quartile for COPD among

FIGURE. Age-adjusted\* percentage of U.S. adults with chronic obstructive pulmonary disease (COPD), overall and by current or previous smoking status — Behavioral Risk Factor Surveillance System, 2017



Abbreviation: DC = District of Columbia.

\* Age-adjusted to the 2000 U.S. standard population aged  $\geq 18$  years.

adults who never smoked, only New Jersey had laws banning smoking in private worksites, restaurants, and bars as of December 31, 2017; the remainder of states in that quartile either had no smoke-free laws or laws banning smoking in only one or two venues.\*\*

The findings in this report are subject to at least seven limitations. First, COPD status was based on self-report, not on medical records or diagnostic tests, and might be subject to recall and social desirability biases. Second, physicians might be more likely to diagnose COPD and other smoking-related

diseases in states with high smoking rates, whereas COPD might be more likely to remain undiagnosed in states with lower smoking rates. Third, smoking status also was based on self-report and might be subject to social desirability bias. Fourth, because the data were cross-sectional, causality could not be examined. Fifth, e-cigarette use was not examined in this report. There were no other measures of exposure to secondhand smoke or other indoor or outdoor air pollutants or history of respiratory infections, all of which might contribute to COPD risk. Sixth, BRFSS surveys noninstitutionalized adults and does not include adults who live in long-term care facilities, prisons, and other facilities; therefore, findings are not generalizable to those populations. Finally, state BRFSS

\*\* There were no changes from December 31, 2017, to March 31, 2019. <https://www.cdc.gov/statesystem/smokefreeindoorair.html>.

response rates were relatively low, which might lead to selection bias.

Population-based strategies for smoking prevention and control have the potential to decrease the prevalence of COPD in the United States. Such strategies include tobacco product price increases, mass media antismoking campaigns, comprehensive smoke-free laws, and barrier-free access to evidence-based cessation interventions.<sup>††</sup> Comprehensive smoke-free laws not only help protect nonsmokers from secondhand smoke exposure, but they can also promote adoption of voluntary smoke-free rules in private settings (e.g., homes and automobiles) and reduce smoking prevalence through increased cessation and decreased initiation.<sup>§§</sup> Clinicians can play a key role in increasing access to and use of cessation therapies, including counseling and Food and Drug Administration-approved cessation medications.<sup>¶¶</sup> Current clinical guidelines recommend screening all patients for tobacco use at every visit (8); however, clinicians should be mindful that not all COPD is necessarily caused by smoking and should use spirometry for diagnosis in patients with COPD symptoms (9), regardless of their smoking history.

<sup>††</sup> [https://www.cdc.gov/tobacco/stateandcommunity/best\\_practices/index.htm](https://www.cdc.gov/tobacco/stateandcommunity/best_practices/index.htm).

<sup>§§</sup> [https://www.cdc.gov/tobacco/basic\\_information/secondhand\\_smoke/](https://www.cdc.gov/tobacco/basic_information/secondhand_smoke/).

<sup>¶¶</sup> <https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/tobacco-use-in-adults-and-pregnant-women-counseling-and-interventions1>.

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## References

1. CDC. Chronic obstructive pulmonary disease among adults—United States, 2011. *MMWR Morb Mortal Wkly Rep* 2012;61:938–43.
2. Ford ES, Croft JB, Mannino DM, Wheaton AG, Zhang X, Giles WH. COPD surveillance—United States, 1999–2011. *Chest* 2013;144:284–305. <https://doi.org/10.1378/chest.13-0809>

## Summary

### What is already known about this topic?

Cigarette smoking is the primary risk factor for chronic obstructive pulmonary disease (COPD) in the United States; an estimated one fourth of adults with COPD have never smoked. Higher COPD prevalence has been observed in southeastern and Appalachian states.

### What is added by this report?

Geographic and sociodemographic patterns of COPD prevalence were similar among current smokers, former smokers, and adults who had never smoked.

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

Population-based strategies for smoking prevention and control, including comprehensive smoke-free policies, have the potential to decrease COPD prevalence, including among nonsmokers. Clinicians should offer cessation support to patients who smoke and consider COPD in symptomatic patients, regardless of smoking history.

3. Croft JB, Wheaton AG, Liu Y, et al. Urban-rural county and state differences in chronic obstructive pulmonary disease—United States, 2015. *MMWR Morb Mortal Wkly Rep* 2018;67:205–11. <https://doi.org/10.15585/mmwr.mm6707a1>
4. Salvi SS, Barnes PJ. Chronic obstructive pulmonary disease in non-smokers. *Lancet* 2009;374:733–43. [https://doi.org/10.1016/S0140-6736\(09\)61303-9](https://doi.org/10.1016/S0140-6736(09)61303-9)
5. US Department of Health and Human Services. The health consequences of smoking—50 years of progress: a report of the Surgeon General. Atlanta, GA: US Department of Health and Human Services, CDC; 2014. <https://www.surgeongeneral.gov/library/reports/50-years-of-progress/full-report.pdf>
6. Diver WR, Jacobs EJ, Gapstur SM. Secondhand smoke exposure in childhood and adulthood in relation to adult mortality among never smokers. *Am J Prev Med* 2018;55:345–52. <https://doi.org/10.1016/j.amepre.2018.05.005>
7. US Department of Health and Human Services. The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, CDC; 2006. <https://www.ncbi.nlm.nih.gov/books/NBK44324/>
8. Fiore MC, Jaen CR, Baker TB, et al. Treating tobacco use and dependence: 2008 update. Clinical practice guideline. Rockville, MD: US Department of Health and Human Services, Public Health Service; 2008. <https://www.ncbi.nlm.nih.gov/books/NBK63952/>
9. Qaseem A, Wilt TJ, Weinberger SE, et al.; American College of Physicians; American College of Chest Physicians; American Thoracic Society; European Respiratory Society. Diagnosis and management of stable chronic obstructive pulmonary disease: a clinical practice guideline update from the American College of Physicians, American College of Chest Physicians, American Thoracic Society, and European Respiratory Society. *Ann Intern Med* 2011;155:179–91. <https://doi.org/10.7326/0003-4819-155-3-201108020-00008>

## Trends in the Laboratory Detection of Rotavirus Before and After Implementation of Routine Rotavirus Vaccination — United States, 2000–2018

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Before the introduction of rotavirus vaccine in the United States in 2006, rotavirus infection was the leading cause of severe gastroenteritis among U.S. children (1). To evaluate the long-term impact of rotavirus vaccination on disease prevalence and seasonality in the United States, CDC analyzed national laboratory testing data for rotavirus from laboratories participating in CDC's National Respiratory and Enteric Viruses Surveillance System (NREVSS) during the prevaccine (2000–2006) and postvaccine (2007–2018) periods. Nationally, the median annual percentage of tests positive for rotavirus declined from 25.6% (range = 25.2–29.4) in the prevaccine period to 6.1% (range = 2.6–11.1) in the postvaccine period. When compared with the prevaccine period, the postvaccine period saw declines in the annual peak in rotavirus positivity from a median of 43.1% (range = 43.8–56.3) to a median of 14.0% (range = 4.8–27.3) and in the season duration from a median of 26 weeks (range = 23–27) to a median of 9 weeks (range = 0–18). In the postvaccine period, a biennial pattern emerged, with alternating years of low and high rotavirus activity. Implementation of the rotavirus vaccination program has substantially reduced prevalence of the disease and altered seasonal patterns of rotavirus in the United States; these changes have been sustained over 11 seasons after vaccine introduction. Ongoing efforts to improve coverage and on-time vaccination (2) can help maximize the public health impact of rotavirus vaccination.

NREVSS is a voluntary laboratory-based passive surveillance system that collects data on eight respiratory viruses and three enteric viruses, including rotavirus (3). Each week, participating laboratories report to CDC the aggregate number of rotavirus tests performed and the number of those that had positive results. A reporting year begins in July (epidemiologic week 27) and ends in June (epidemiologic week 26) of the following year. Peak rotavirus activity is defined as the highest proportion of tests positive for rotavirus during a single week in a given reporting year. The beginning and end of the rotavirus season are defined as the first and last, respectively, of 2 consecutive weeks in which  $\geq 10\%$  of the tests are positive for rotavirus. Historically, rotavirus disease exhibited a winter-spring seasonality, with the season beginning in December–January and ending in April–May (4).

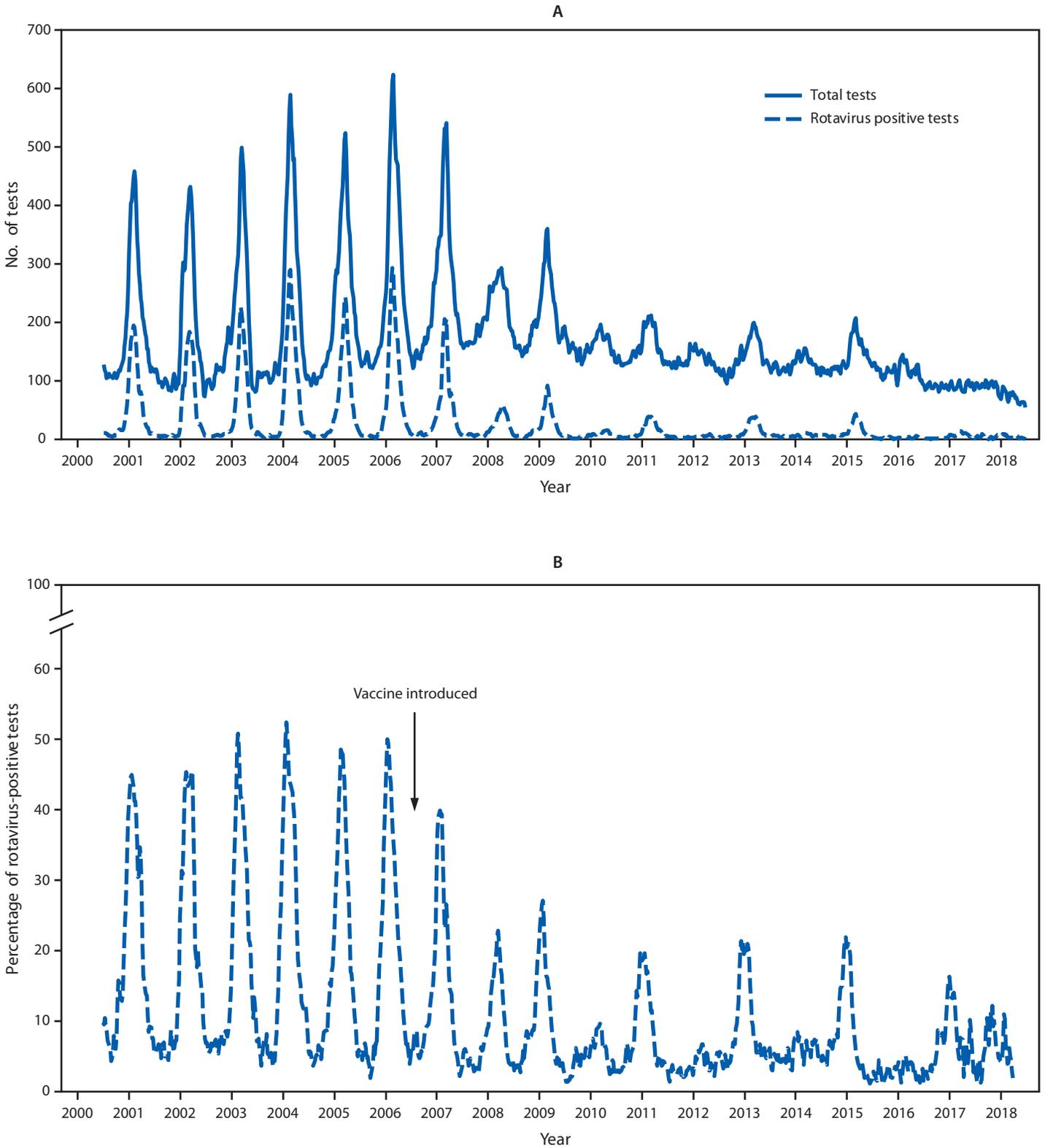
Results of all enzyme immunoassay (EIA) tests for rotavirus conducted during July 2000–June 2018 were obtained from laboratories participating in NREVSS. Data from the

first reporting year after vaccine introduction (July 2006–June 2007), which is considered a transitional year with low vaccination coverage, were excluded from the analysis. To examine trends in rotavirus testing and detection during the prevaccine and postvaccine periods, analyses were restricted to the 23 laboratories that continuously reported rotavirus testing results for  $\geq 26$  weeks of each reporting year during July 2000–July 2018. Data were aggregated by week and are presented using a 3-week moving average for the total number of rotavirus tests performed and the number of positive test results. Trends in testing practices over time were evaluated using the Spearman rank order correlation for the annual number of tests conducted and the Cochran–Armitage test for trend for the annual proportions of tests that were positive for rotavirus. SAS software (version 9.4; SAS Institute) was used for all statistical analyses. To compare the rotavirus season, duration, and peak activity between the prevaccine and postvaccine periods, data from all reporting laboratories (annual range = 57–223) were analyzed. When analyzing the biennial trend in rotavirus seasonality, data from the first 2 reporting years after vaccine introduction (July 2006–June 2008) were excluded from the analysis.

Data from the 23 laboratories that continuously reported rotavirus testing results during 2000–2018 demonstrated a decline in both rotavirus testing and percent positivity in the postvaccine era compared with the prevaccine era (Figure 1) (Table). The number of rotavirus tests declined by approximately one third, from an annual median of 10,845 (range = 9,105–13,257) in the prevaccine era to an annual median of 7,357 (range = 4,270–11,143;  $p < 0.001$ ) in the postvaccine era; the number of tests positive for rotavirus declined approximately 85%, from an annual median of 2,778 (range = 2,385–3,479) in the prevaccine era to an annual median of 411 (range = 159–1,231) in the postvaccine era ( $p < 0.001$ ). Mirroring the trends in the number of positive tests, the median annual proportion of tests positive for rotavirus declined 76%, from 25.6% (range = 25.2%–29.4%) in the prevaccine era to 6.1% (range = 2.6%–11.1%; all  $p$ -values  $< 0.001$ ) in the postvaccine era.

Analysis of data from all reporting laboratories indicated that rotavirus test positivity during peak activity declined by approximately two thirds, from an annual median of 43.1% (range = 43.8–56.3) in the prevaccine era to 14.0% (range = 4.8–27.3) in the postvaccine era (Table). In addition,

FIGURE 1. Total number of rotavirus tests and positive rotavirus tests (A) and percent positivity (B) among the 23 continuously reporting National Respiratory and Enteric Virus Surveillance System (NREVSS) laboratories\* — NREVSS, United States, 2000–2018



\* Data were aggregated by week and are presented using a 3-week moving average for the total number of rotavirus tests performed and the number of positive test results.

**TABLE. Rotavirus seasonality and testing results, by years — National Respiratory and Enteric Virus Surveillance System, United States, 2000–2018.**

Years	Season beginning, wk no.	Peak		Season end, wk no.	Season duration (wks)	No. of rotavirus tests performed*	Positive rotavirus tests* No. (%)	% Change in rotavirus tests performed* <sup>§</sup>	% Decline in positive rotavirus tests*
		Wk no.	% Positive tests						
2000–2006 <sup>†</sup>	50	9	43.1	24	26	10,845	2,778 (25.6)	Referent	Referent
2007–2008	9	17	17.3	21	12	11,143	1,034 (9.3)	2.7	62.8
2008–2009	4	11	25.3	21	17	11,078	1,231 (11.1)	2.1	55.7
2009–2010	— <sup>¶</sup>	18	10.9	—	—	8,345	411 (4.9)	–23.1	85.2
2010–2011	3	11	23.4	21	18	8,152	734 (9.0)	–24.8	73.6
2011–2012	—	22	12.2	—	—	7,129	244 (3.4)	–34.3	91.2
2012–2013	1	13	27.3	18	17	7,357	718 (9.8)	–32.2	74.2
2013–2014	—	21	11.3	—	—	6,687	352 (5.3)	–38.3	87.3
2014–2015	3	11	25.1	16	13	7,448	724 (9.7)	–31.3	73.9
2015–2016	—	20	4.8	—	—	6,145	159 (2.6)	–43.3	94.3
2016–2017	9	13	21.7	19	10	4,708	287 (6.1)	–56.6	89.7
2017–2018	—	17	10.3	—	—	4,270	235 (5.5)	–60.6	91.5

\* Testing data from the 23 laboratories that continuously reported rotavirus test results during 2000–2018.

<sup>†</sup> Median data are reported for the prevaccine seasons spanning 2000–2006.

<sup>§</sup> Compared with number of tests performed during 2000–2006.

<sup>¶</sup> Dashes indicate not applicable because seasonal start and end thresholds were not reached.

in the postvaccine era, the rotavirus season began later in the year, and the annual median season duration was reduced from 26 weeks (range = 23–27) in the prevaccine era to 9 weeks (range = 0–18) in the postvaccine era.

In the postvaccine period, a biennial pattern emerged, with alternating years of low and high rotavirus activity (Figure 1). In low-activity years after vaccine introduction, the 10% test positivity threshold for the start of the season was never reached, peak activity occurred later in the year (weeks 17–22), and the median peak activity was 8.7% (range = 4.8–12.2) (Figure 2). In contrast, in the high-activity years after vaccine introduction, the median season duration was 17 weeks (range = 10–18), peak activity occurred earlier in the year (weeks 11–13), and the median peak test positivity was 25.1% (range = 21.7%–27.3%). Over time, rotavirus activity and seasonality have remained relatively consistent in the low- and high-activity years; however, the season duration during high-activity years has slowly decreased.

The number of laboratories reporting EIA test results for rotavirus to NREVSS in the postvaccine era has gradually declined over time, from a high of 223 laboratories reporting 29,198 EIA test results (2008–09 season) to 109 laboratories reporting 14,737 EIA test results (2017–18 season). However, in recent years, the number of laboratories reporting polymerase chain reaction (PCR) tests for rotavirus has increased, from 19 laboratories reporting 16,490 PCR test results (2014–15 season) to 80 laboratories reporting 87,775 PCR test results (2017–18 season).

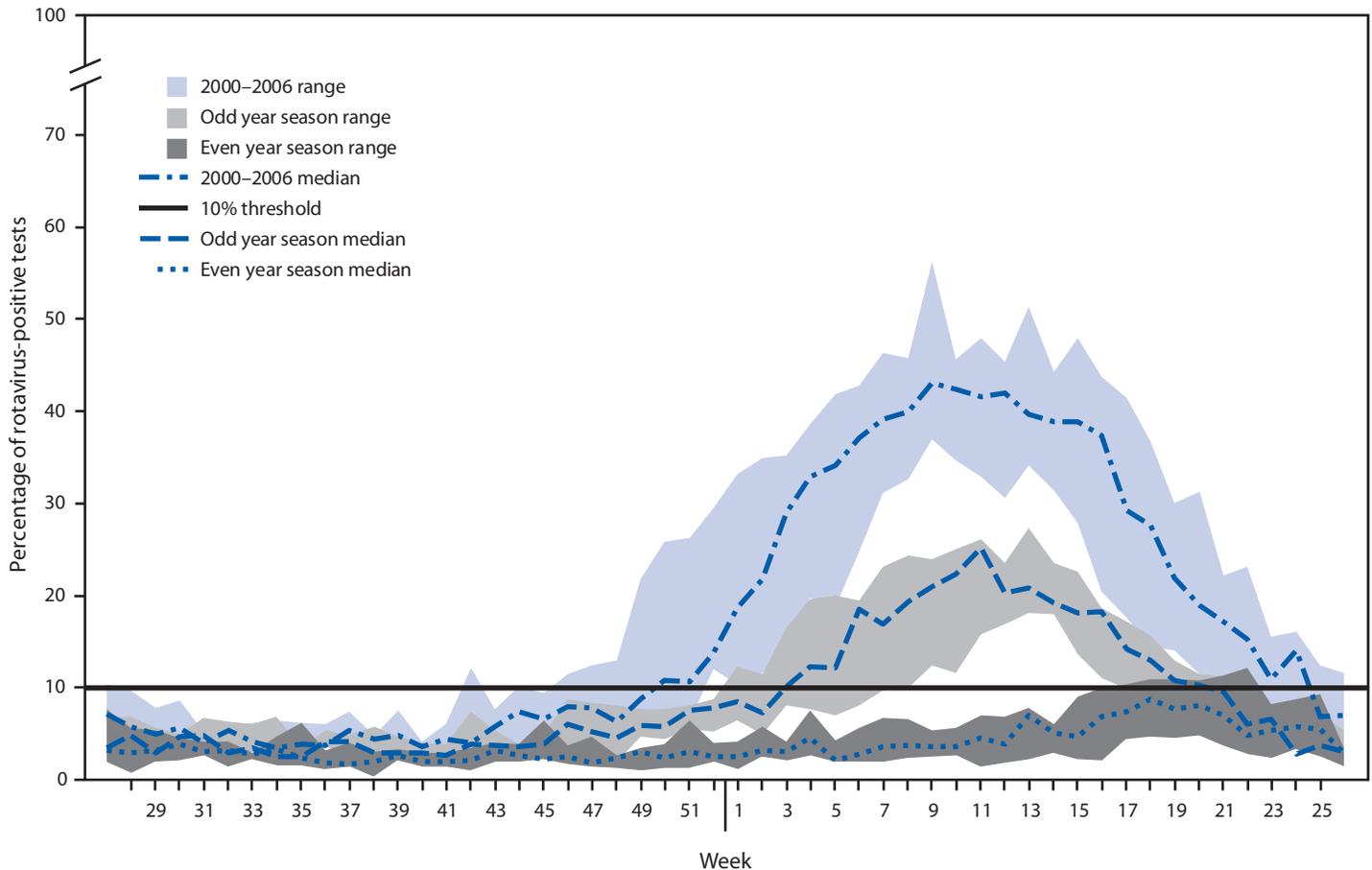
### Discussion

Implementation of the rotavirus vaccination program has markedly reduced the prevalence of rotavirus disease in the

United States. In all postvaccine-era seasons from 2007–08 to 2017–18, rotavirus activity consistently fell below the 2000–2006 baseline, and seasons were shorter in duration compared with those during the prevaccine era, which further attests to the long-term benefits of the rotavirus vaccination program. Some of the observed changes in rotavirus activity that occurred after vaccine introduction could be due to concurrent changes in rotavirus testing practices as PCR-based multipathogen detection assays are increasingly used. Although this shift in testing practices might explain some of the declines observed in the total number of tests performed and the number of positive tests, the substantial reductions in the proportion of rotavirus tests that were positive (which is less affected by changes in testing practices alone), supports attribution of the declines to the effects of vaccination.

Introduction of rotavirus vaccine has also modified the seasonality of rotavirus disease in the United States, with a biennial trend emerging in the postvaccine era beginning in the 2008–09 rotavirus season. Since vaccine introduction, coverage has slowly increased and completed coverage has plateaued at approximately 70%. The lower coverage of rotavirus compared with other childhood vaccines might be explained in part by the fact that rotavirus vaccine does not offer the same opportunity for catch-up because the first dose must be given by age 15 weeks, and the series must be completed by age 8 months (2,5). The biennial trend observed in the United States could be attributed to this low vaccination coverage, with the number of susceptible children accumulating in low rotavirus activity years, resulting in a higher number of susceptible children and a subsequent rotavirus outbreak during the following season (6). Countries that rapidly achieved and maintained rotavirus vaccination coverage of 90%–95%, such

**FIGURE 2. Rotavirus season duration and peak activity for prevaccine (2000–2006) and postvaccine years (2008–2018), stratified by even/odd year season,\* by week of season — National Respiratory and Enteric Virus Surveillance System, United States 2000–2018**



\* Odd year seasons: 2008–09; 2010–11; 2012–13; 2014–15; 2016–17. Even year seasons: 2009–10; 2011–12; 2013–14; 2015–16; 2017–18.

as the United Kingdom, have experienced a sustained decline in rotavirus activity without the biennial trend observed in the United States (7). As vaccination coverage and on-time vaccination continue to improve in the United States, the seasonality of rotavirus disease can be monitored to see whether the biennial trend continues.

The findings in this report are subject to at least three limitations. First, aggregate NREVSS data are reported, without demographic or clinical characteristics (including vaccination status), precluding examination of these characteristics. Second, these data were collected from a passive surveillance system composed of a convenience sample of laboratories and might not be representative of all those in the United States. Finally, because rotavirus testing does not affect clinical management (which focuses on rehydration and syndromic management), testing practices vary from site to site and year to year, which might affect data comparability (1,6). However,

NREVSS data have advantages, including the ability to describe trends in rotavirus activity in the United States in a timely fashion. In addition, NREVSS data have consistently aligned with rotavirus-related U.S. hospital discharge data and active surveillance data (8–10).

Rotavirus vaccination has resulted in a significant and sustained reduction of disease prevalence and has modified the seasonality of rotavirus disease in the United States. To maximize the public health impact of rotavirus vaccination, efforts to improve coverage and on-time vaccination should continue.

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

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

Before the introduction of rotavirus vaccine in the United States in 2006, rotavirus infection was the leading cause of severe gastroenteritis among U.S. children.

**What is added by this report?**

Implementation of the U.S. rotavirus vaccination program reduced the annual proportion of positive rotavirus tests, reduced peak rotavirus activity, and shortened the duration of the rotavirus season. Biennial seasonal patterns that emerged after vaccine introduction have continued with alternating years of low and high rotavirus activity.

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

Ongoing efforts to improve coverage and on-time vaccination can help maximize the public health impact of rotavirus vaccination.

**References**

1. Aliabadi N, Tate JE, Haynes AK, Parashar UD. Sustained decrease in laboratory detection of rotavirus after implementation of routine vaccination—United States, 2000–2014. *MMWR Morb Mortal Wkly Rep* 2015;64:337–42.
2. Cortese MM, Parashar UD. Prevention of rotavirus gastroenteritis among infants and children: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep* 2009;58(No. RR-2).
3. CDC. The National Respiratory and Enteric Virus Surveillance System (NREVSS). US Department of Health and Human Services, CDC; 2019. <https://www.cdc.gov/surveillance/nrevss/index.html>
4. Turcios RM, Curns AT, Holman RC, et al.; National Respiratory and Enteric Virus Surveillance System Collaborating Laboratories. Temporal and geographic trends of rotavirus activity in the United States, 1997–2004. *Pediatr Infect Dis J* 2006;25:451–4. <https://doi.org/10.1097/01.inf.0000214987.67522.78>
5. Pindyck T, Tate JE, Parashar UD. A decade of experience with rotavirus vaccination in the United States—vaccine uptake, effectiveness, and impact. *Expert Rev Vaccines* 2018;17:593–606. <https://doi.org/10.1080/14760584.2018.1489724>
6. Tate JE, Haynes A, Payne DC, et al. Trends in national rotavirus activity before and after introduction of rotavirus vaccine into the national immunization program in the United States, 2000 to 2012. *Pediatr Infect Dis J* 2013;32:741–4. <https://doi.org/10.1097/INF.0b013e31828d639c>
7. Atchison CJ, Stowe J, Andrews N, et al. Rapid declines in age group-specific rotavirus infection and acute gastroenteritis among vaccinated and unvaccinated individuals within 1 year of rotavirus vaccine introduction in England and Wales. *J Infect Dis* 2016;213:243–9. <https://doi.org/10.1093/infdis/jiv398>
8. Cortese MM, Tate JE, Simonsen L, Edelman L, Parashar UD. Reduction in gastroenteritis in United States children and correlation with early rotavirus vaccine uptake from national medical claims databases. *Pediatr Infect Dis J* 2010;29:489–94. <https://doi.org/10.1097/INF.0b013e3181d95b53>
9. Yen C, Tate JE, Wenk JD, Harris JM 2nd, Parashar UD. Diarrhea-associated hospitalizations among US children over 2 rotavirus seasons after vaccine introduction. *Pediatrics* 2011;127:e9–15. <https://doi.org/10.1542/peds.2010-1393>
10. Payne DC, Staat MA, Edwards KM, et al.; New Vaccine Surveillance Network (NVSN). Direct and indirect effects of rotavirus vaccination upon childhood hospitalizations in 3 US counties, 2006–2009. *Clin Infect Dis* 2011;53:245–53. <https://doi.org/10.1093/cid/cir307>

## Update: Influenza Activity in the United States During the 2018–19 Season and Composition of the 2019–20 Influenza Vaccine

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Influenza activity\* in the United States during the 2018–19 season (September 30, 2018–May 18, 2019) was of moderate severity (*I*). Nationally, influenza-like illness (ILI)<sup>†</sup> activity began increasing in November, peaked during mid-February, and returned to below baseline in mid-April; the season lasted 21 weeks,<sup>§</sup> making it the longest season in 10 years. Illness attributed to influenza A viruses predominated, with very little influenza B activity. Two waves of influenza A were notable during this extended season: influenza A(H1N1)pdm09 viruses from October 2018 to mid-February 2019 and influenza A(H3N2) viruses from February through May 2019. Compared with the 2017–18 influenza season, rates of hospitalization this season were lower for adults, but were similar for children. Although influenza activity is currently below surveillance baselines, testing for seasonal influenza viruses and monitoring for novel influenza A virus infections should continue year-round. Receiving a seasonal influenza vaccine each year remains the best way to protect against seasonal influenza and its potentially severe consequences.

### Virus Surveillance

U.S. World Health Organization (WHO) collaborating laboratories and National Respiratory and Enteric Virus Surveillance System laboratories, which include both clinical and public health laboratories throughout the United States, contribute to virologic surveillance for influenza. During September 30, 2018–May 18, 2019, clinical laboratories tested 1,145,555 specimens for influenza virus; among these, 177,039 (15.5%) tested positive, including 167,529 (95.0%) for influenza A and 9,510 (5.0%) for influenza B. The percentage of

specimens testing positive for influenza each week ranged from 1.7% to 26.2%.

Nationally, the percentage of clinical laboratory–tested specimens positive for influenza virus peaked during the weeks ending February 9–March 16 (surveillance weeks 6–11) (range = 25.1%–26.2%). Regionally,<sup>¶</sup> the week of peak clinical laboratory influenza positivity varied, ranging from the week ending December 15, 2018 (week 50) to the week ending March 16, 2019 (week 11).

Public health laboratories tested 80,993 specimens during September 30, 2018–May 18, 2019; among these specimens, 42,303 (52.2%) were positive for influenza viruses, including 40,624 (96.0%) that were positive for influenza A and 1,679 (4.0%) for influenza B. Among the 38,995 seasonal influenza A viruses subtyped, 22,084 (56.6%) were influenza A(H1N1)pdm09, and 16,991 (43.6%) were influenza A(H3N2). Influenza B lineage information was available for 1,105 (65.8%) influenza B viruses; 406 (36.7%) of those were B/Yamagata lineage, and 699 (63.3%) were B/Victoria lineage. Whereas influenza A(H1N1)pdm09 viruses accounted for the majority of circulating viruses nationwide from October 2018 to mid-February 2019, influenza A(H3N2) viruses were detected more frequently than were A(H1N1)pdm09 viruses beginning in late February nationally (Figure 1) and in all 10 U.S. Health and Human Services (HHS) regions by the end of March 2019. For the season overall, influenza A(H3N2) viruses predominated in HHS Regions 4, 6, and 7, and influenza A(H1N1)pdm09 viruses predominated in the remaining seven regions.

Among 38,564 (91.2%) patients whose test results were positive for seasonal influenza virus by public health laboratories and for whom age data were available, 4,844 (12.6%) were

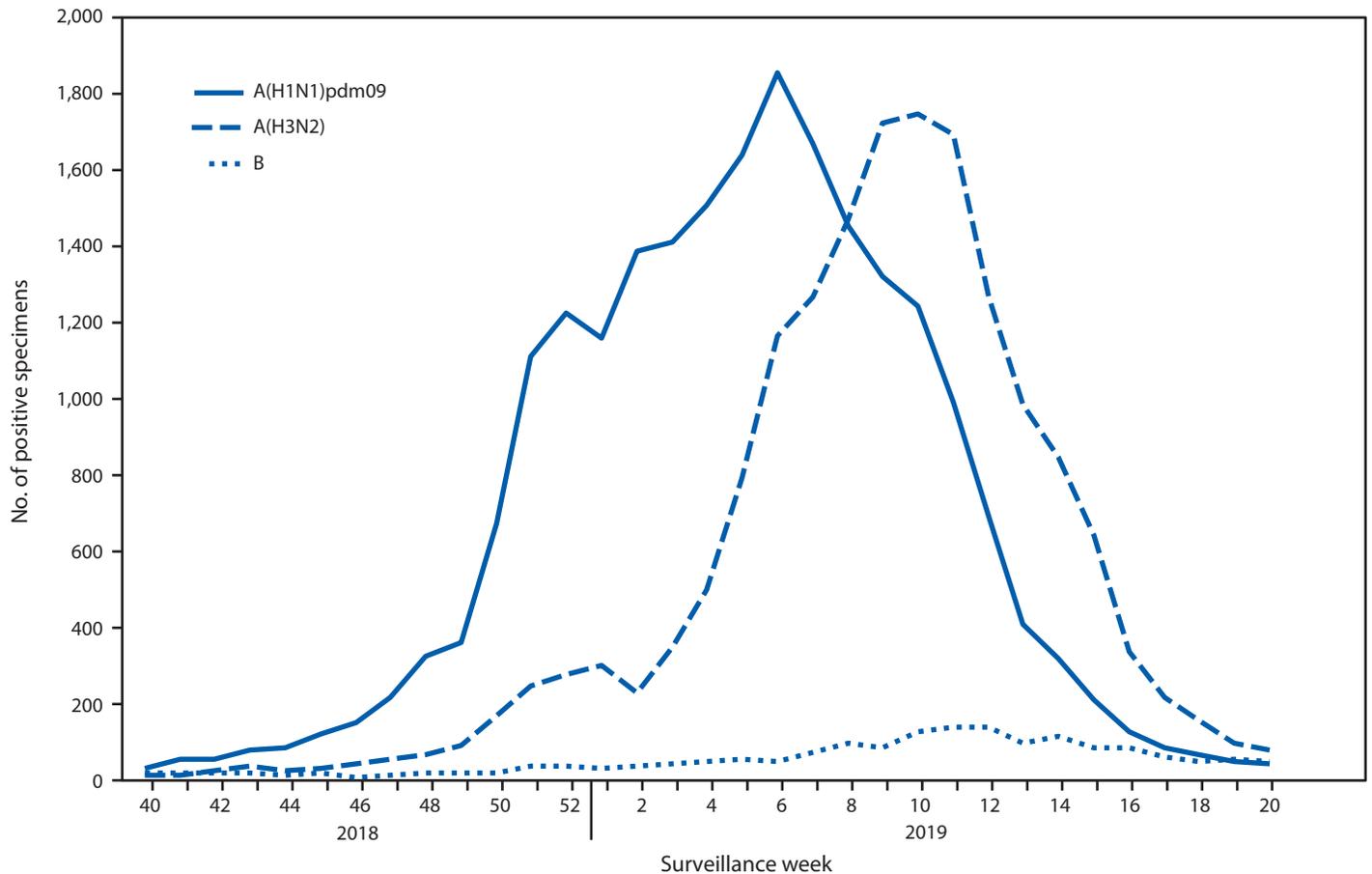
\*The CDC influenza surveillance system collects five categories of information from eight data sources: 1) virus surveillance (U.S. World Health Organization collaborating laboratories, the National Respiratory and Enteric Virus Surveillance System, and novel influenza A virus case reporting); 2) outpatient 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>.

<sup>†</sup> Defined as a fever (temperature  $\geq 100^{\circ}\text{F}$  [ $\geq 37.8^{\circ}\text{C}$ ]), oral or equivalent) and cough or sore throat, without a known cause other than influenza.

<sup>§</sup> Data as of June 14, 2019.

<sup>¶</sup> The 10 regions include the following jurisdictions: *Region 1*: Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, and Vermont; *Region 2*: New Jersey, New York, Puerto Rico, and U.S. Virgin Islands; *Region 3*: Delaware, District of Columbia, Maryland, Pennsylvania, Virginia, and West Virginia; *Region 4*: Alabama, Florida, Georgia, Kentucky, Mississippi, North Carolina, South Carolina, and Tennessee; *Region 5*: Illinois, Indiana, Michigan, Minnesota, Ohio, and Wisconsin; *Region 6*: Arkansas, Louisiana, New Mexico, Oklahoma, and Texas; *Region 7*: Iowa, Kansas, Missouri, and Nebraska; *Region 8*: Colorado, Montana, North Dakota, South Dakota, Utah, and Wyoming; *Region 9*: Arizona, California, Hawaii, Nevada, American Samoa, Commonwealth of the Northern Mariana Islands, Federated States of Micronesia, Guam, Marshall Islands, and Palau; *Region 10*: Alaska, Idaho, Oregon, and Washington.

**FIGURE 1. Number\* of respiratory specimens testing positive for influenza reported to CDC by public health laboratories, by influenza virus type, subtype,<sup>†</sup> and surveillance week — United States, September 30, 2018–May 18, 2019<sup>§</sup>**



\* N = 40,674.

<sup>†</sup> 1,629 influenza A viruses not subtyped are excluded.

<sup>§</sup> As of June 14, 2019.

aged 0–4 years; 12,508 (32.4%) were aged 5–24 years; 13,382 (34.7%) were aged 25–64 years; and 7,830 (20.3%) were aged ≥65 years. Influenza A(H1N1)pdm09 virus was the most frequently reported virus among persons aged 0–4 years (57.1%) and 25–64 years (63.2%), whereas influenza A(H3N2) virus was the most commonly reported virus among persons aged 5–24 years (48.8%) and ≥65 years (51.3%). The age group with the largest proportion of reported influenza B viruses (6.3%) was persons aged 5–24 years.

### Antigenic and Genetic Characterization of Influenza Viruses

Genetic characterization was carried out using next-generation sequencing, and the genomic data were analyzed and submitted to public databases (GenBank: <https://www.ncbi.nlm.nih.gov/genbank> or EpiFlu: <https://www.gisaid.org/>). Antigenic characterizations were carried out by hemagglutination inhibition

assays or virus neutralization–based focus reduction assays to evaluate whether genetic changes in circulating viruses affected antigenicity; substantial differences could affect vaccine effectiveness. CDC genetically characterized 2,750 influenza viruses collected and submitted\*\* by U.S. laboratories since September 30, 2018, including 1,251 influenza A(H1N1)pdm09 viruses, 1,024 influenza A(H3N2) viruses, and 475 influenza B viruses. A subset of these viruses also was antigenically characterized. Phylogenetic analysis of the hemagglutinin (HA) gene segments from the 1,251 characterized A(H1N1)pdm09 viruses determined that all belonged to genetic subclade 6B.1A, which evolved from clade 6B.1. Among 331 antigenically characterized A(H1N1)pdm09 viruses, 318 (96.1%) were well inhibited (reacting at titers that were within fourfold of the homologous

\*\* Association of Public Health Laboratories. Influenza Virologic Surveillance Right Size Roadmap. [https://www.aphl.org/AboutAPHL/publications/Documents/ID\\_July2013\\_Influenza-Virologic-Surveillance-Right-Size-Roadmap.pdf](https://www.aphl.org/AboutAPHL/publications/Documents/ID_July2013_Influenza-Virologic-Surveillance-Right-Size-Roadmap.pdf).

virus titer) by ferret antisera raised against A/Michigan/45/2015 (6B.1), the cell culture–propagated reference virus representing the A(H1N1)pdm09 component for the 2018–19 Northern Hemisphere influenza vaccines.

Phylogenetic analysis of the HA gene segments of 1,204 sequenced influenza A(H3N2) viruses indicated cocirculation of multiple clades/subclades. Circulating viruses possessed HA gene segments that belonged to clade 3C.2a (66; 6.4%), subclade 3C.2a1 (201; 19.6%), or clade 3C.3a (757; 73.9%). The frequency of 3C.3a viruses increased from 12.7% of the A(H3N2) viruses collected and sequenced by November 2018 to 81.9% of those collected and sequenced during December 2018–May 2019. Among the 505 A(H3N2) viruses antigenically characterized by focus reduction assays with ferret antisera, 191 (37.8%) were well inhibited by ferret antisera raised against A/Singapore/INFIMH-16-0019/2016 (3C.2a1), a cell culture–propagated reference virus representing the A(H3N2) component of 2018–19 Northern Hemisphere influenza vaccines. However, only 43 (11%) of the 388 viruses tested were well inhibited by antiserum raised against egg-propagated A/Singapore/INFIMH-16-0019/2016 reference virus, likely because of egg-adaptive amino acid changes in the HA protein of the egg-propagated virus. Three hundred fourteen (62.2%) viruses were poorly inhibited by ferret antiserum raised against cell culture–propagated A/Singapore/INFIMH-16-0019/2016 reference virus (at titers that were reduced eightfold or more when compared with the homologous virus); among those viruses, 312 (99.4%) belonged to clade 3C.3a, the prevalence of which increased throughout the season.

Phylogenetic analysis of 203 influenza B/Yamagata lineage viruses determined that the HA gene segments belonged to clade Y3. All 178 B/Yamagata lineage viruses antigenically characterized were well inhibited by ferret antiserum raised against cell culture–propagated B/Phuket/3073/2013, the reference virus representing the B/Yamagata lineage component of quadrivalent vaccines for the 2018–19 Northern Hemisphere influenza season.

Multiple genetically and antigenically distinct B/Victoria lineage viruses cocirculated during the 2018–19 season. Viruses with a two-amino acid deletion (162–163) in the HA protein belong to subclade V1A.1, and viruses with a three-amino acid deletion (162–164) in the HA protein belong to subclade V1A-3Del. Among the 272 influenza B/Victoria lineage viruses sequenced and phylogenetically analyzed, the HA gene segment belonged to genetic clade V1A (40; 14.7%), subclade V1A.1 (137; 50.4%), or subclade V1A-3Del (95; 34.9%). Among 191 B/Victoria lineage viruses antigenically characterized, 147 (79.1%) were well inhibited by ferret antiserum raised against cell culture–propagated B/Colorado/06/2017-like V1A.1 reference virus representing the B/Victoria lineage component of the vaccines for the 2018–19 Northern Hemisphere

influenza season. Among the 44 (20.9%) viruses that reacted poorly, 17 were antigenically related to the previous vaccine virus B/Brisbane/60/2008 and belonged to clade V1A, and 27 belonged to subclade V1A-3Del.

## Antiviral Susceptibility of Influenza Viruses

Testing of seasonal influenza A(H1N1)pdm09, influenza A(H3N2), and influenza B viruses for resistance to the neuraminidase inhibitors oseltamivir, zanamivir, and peramivir is performed at CDC using next-generation sequencing analysis, a functional assay (2), or both. Neuraminidase sequences of viruses are examined for the presence of amino acid substitutions previously associated with reduced or highly reduced inhibition by any of the three neuraminidase inhibitors.<sup>††</sup> The amino acid substitution H275Y in A(H1N1)pdm09 viruses is considered clinically relevant because of the frequency of occurrence and the availability of clinical data demonstrating a reduced treatment efficacy; however, other amino acid substitutions have been observed less frequently and caused reduced susceptibility *in vitro*, but with less clear clinical significance (2).

A total of 2,699 influenza virus specimens, including 1,240 influenza A(H1N1)pdm09, 1,016 influenza A(H3N2), 252 influenza B/Victoria, and 191 influenza B/Yamagata viruses collected in the United States since October 1, 2018, were tested for resistance to oseltamivir, zanamivir, and peramivir. Five (0.3%) influenza A(H1N1)pdm09 viruses had the amino acid substitution H275Y and displayed highly reduced inhibition by oseltamivir and peramivir. In addition, four (0.3%) influenza A(H1N1)pdm09 viruses displayed some reduction in inhibition by oseltamivir, and two influenza B viruses (0.4%) from different lineages had the amino acid substitution H273Y and displayed highly reduced inhibition by peramivir.

During the 2018–19 influenza season, CDC began to test seasonal influenza viruses for resistance to the PA cap-dependent endonuclease inhibitor baloxavir using next-generation sequencing analysis, a phenotypic assay (3), or both. PA protein sequences were examined for the presence of amino acid substitutions previously associated with decreased susceptibility or resistance to baloxavir (3).

Among 2,673 influenza virus specimens, including 1,213 influenza A(H1N1)pdm09, 1,007 influenza A(H3N2), 255 influenza B/Victoria, and 198 influenza B/Yamagata viruses collected in the United States since October 1, 2018, and tested genetically for resistance to baloxavir, none contained amino acid substitutions in the PA protein previously associated with decreased susceptibility to baloxavir. All 191 influenza viruses tested by a phenotypic assay were susceptible to baloxavir.

<sup>††</sup> [https://www.who.int/influenza/gisrs\\_laboratory/antiviral\\_susceptibility/NAI\\_Reduced\\_Susceptibility\\_Marker\\_Table\\_WHO.pdf?ua](https://www.who.int/influenza/gisrs_laboratory/antiviral_susceptibility/NAI_Reduced_Susceptibility_Marker_Table_WHO.pdf?ua).

## Composition of the 2019–20 Influenza Vaccines

Vaccine recommendations were made based on factors including data from global influenza virologic and epidemiologic surveillance, genetic characterization, antigenic characterization, and the candidate vaccine viruses that are available for production. WHO recommended the Northern Hemisphere 2019–20 influenza vaccine composition (4), and the Food and Drug Administration's Vaccines and Related Biologic Products Advisory Committee subsequently made the influenza vaccine composition recommendation for the United States (5,6). Both agencies recommend that influenza trivalent vaccines contain an A/Brisbane/02/2018 A(H1N1)pdm09-like virus, an A/Kansas/14/2017 A(H3N2)-like virus, and a B/Colorado/06/2017-like (B/Victoria lineage) virus. The quadrivalent vaccine recommendation included the trivalent vaccine viruses and a B/Phuket/3073/2013-like (B/Yamagata lineage) virus. The A(H1N1)pdm09 and A(H3N2) recommendations are an update to the 2018–19 Northern Hemisphere vaccines. The decision to update the A(H1N1)pdm09 component was made because of genetic and antigenic characterization data using individual postvaccination human sera, which demonstrated significantly reduced titers (eightfold or greater) to recent 6B.1A viruses, compared with the titers against the A/Michigan/45/2015 vaccine virus (5). The decision to update the A(H3N2) component was made to address antigenic drift of the virus with emergence and spread of A/Kansas/14/2017-like viruses (3C.3a) (6).

## Outpatient Illness Surveillance

Nationally, the weekly percentage of outpatient visits for ILI to health care providers participating in the U.S. Outpatient Influenza-like Illness Surveillance Network (ILINet) was at or above the national baseline<sup>§§</sup> level of 2.2% for 21 consecutive weeks (weeks 47–15) during the 2018–19 season (Figure 2). The percentage of outpatient ILI visits peaked at 5.1% during the week ending February 16, 2018 (week 7).

ILINet data are used to produce a weekly jurisdiction-level measure of ILI activity,<sup>¶¶</sup> ranging from minimal to high. The

<sup>§§</sup> The national and regional baselines are the mean percentages of visits for ILI during noninfluenza weeks for the previous three seasons plus two standard deviations. Noninfluenza weeks are defined as periods of  $\geq 2$  consecutive weeks during which each week accounted for  $< 2\%$  of the season's total number of specimens that tested positive for influenza. National and regional percentages of patient visits for ILI are weighted according to state population. Use of the national baseline for regional data is not recommended.

<sup>¶¶</sup> Activity levels are derived from the percentage of outpatient visits in a jurisdiction attributed to ILI and are compared with the average percentage of ILI visits that occur during weeks with little or no influenza virus circulation. Activity levels range from minimal, corresponding to ILI activity from outpatient clinics at or below the average, to high, corresponding to ILI activity from outpatient clinics much higher than the average. Because the clinical definition of ILI is nonspecific, not all ILI is caused by influenza; however, when combined with laboratory data, the information on ILI activity provides a clearer picture of influenza activity in the United States.

number of jurisdictions reporting high ILI activity peaked during the week ending February 23, 2019 (week 8) when 33 (61%) of 54 jurisdictions (50 states, New York City, the District of Columbia, Puerto Rico, and U.S. Virgin Islands) experienced high ILI activity.

## Geographic Spread of Influenza Activity

State and territorial epidemiologists report the geographic distribution of influenza in their jurisdictions through a weekly influenza activity code.<sup>\*\*\*</sup> During the 2018–19 season, the peak number of jurisdictions reporting widespread activity in a single week was 50 (93%); this occurred during week 8 (week ending February 23, 2019).

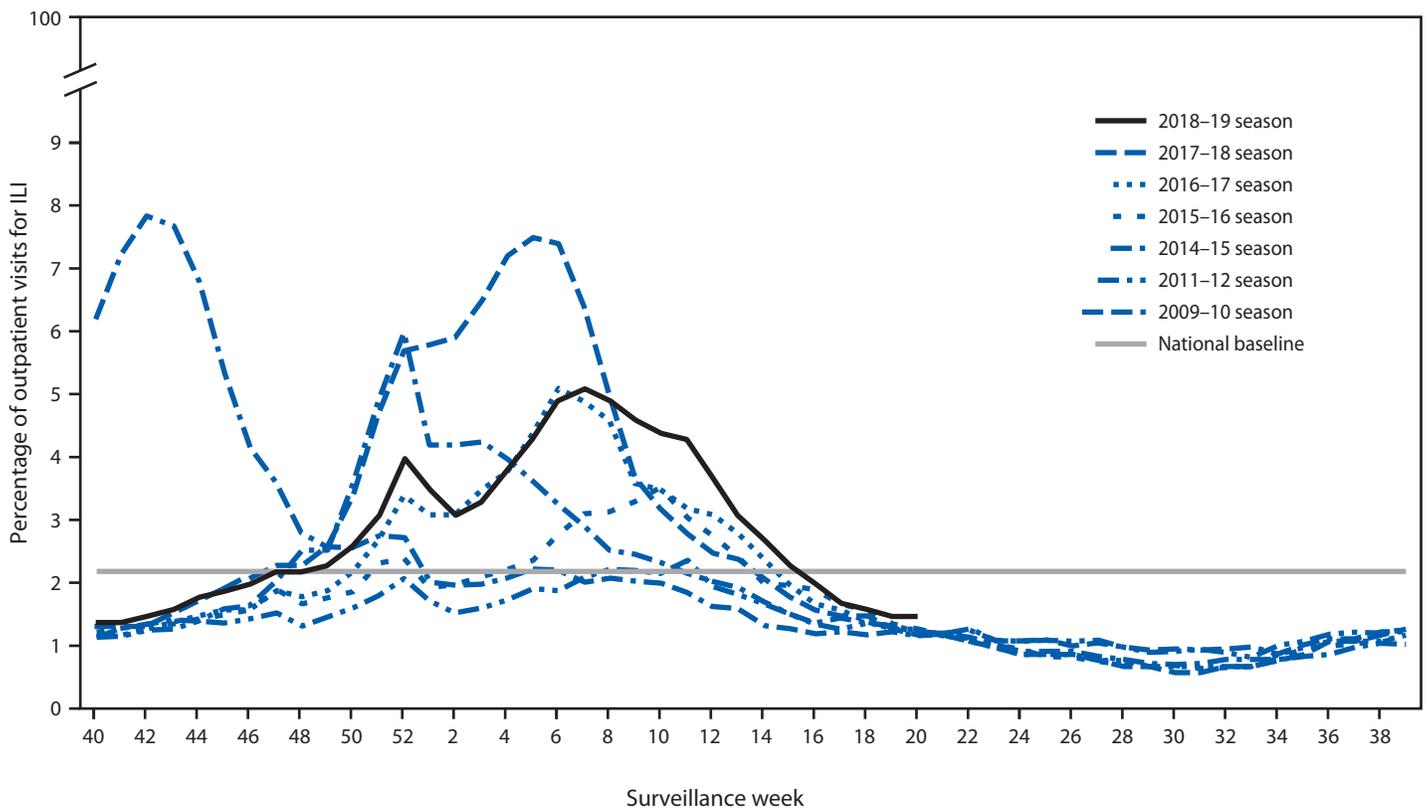
## Influenza-Associated Hospitalizations

CDC monitors hospitalizations associated with laboratory-confirmed influenza infections through the Influenza Hospitalization Surveillance Network (FluSurv-NET),<sup>†††</sup> which covers approximately 27 million persons (9% of the U.S. population). During October 1, 2018–April 30, 2019, a total of 18,847 laboratory-confirmed influenza-related hospitalizations were reported (cumulative incidence for all age groups = 65.3 per 100,000 population). The overall peak

<sup>\*\*\*</sup> Levels of activity are 1) no activity; 2) sporadic: isolated laboratory-confirmed influenza cases or a laboratory-confirmed outbreak in one institution, with no increase in activity; 3) local: increased ILI, or two or more institutional outbreaks (ILI or laboratory-confirmed influenza) in one region of the state, with recent laboratory evidence of influenza in that region; virus activity no greater than sporadic in other regions; 4) regional: increased ILI activity or institutional outbreaks (ILI or laboratory-confirmed influenza) in two or more outbreaks, but fewer than half of the regions in the state with recent laboratory evidence of influenza in those regions; and 5) widespread: increased ILI activity or institutional outbreaks (ILI or laboratory-confirmed influenza) in at least half of the regions in the state, with recent laboratory evidence of influenza in the state.

<sup>†††</sup> FluSurv-NET conducts population-based surveillance for laboratory-confirmed, influenza-associated hospitalizations in children and adolescents aged  $< 18$  years (since the 2003–04 influenza season) and adults aged  $\geq 18$  years (since the 2005–06 influenza season). FluSurv-NET covers approximately 70 counties in the 10 Emerging Infections Program states (California, Colorado, Connecticut, Georgia, Maryland, Minnesota, New Mexico, New York, Oregon, and Tennessee) and additional Influenza Hospitalization Surveillance Project (IHSP) states. IHSP began during the 2009–10 season to enhance surveillance during the 2009 H1N1 pandemic. IHSP sites included Idaho, Iowa, Michigan, Oklahoma, and South Dakota during the 2009–10 season; Idaho, Michigan, Ohio, Oklahoma, Rhode Island, and Utah during the 2010–11 season; Michigan, Ohio, Rhode Island, and Utah during the 2011–12 season; Iowa, Michigan, Ohio, Rhode Island, and Utah during the 2012–13 season; and Michigan, Ohio, and Utah during the 2013–14, 2014–15, 2015–16, and 2016–17 seasons. Cumulative unadjusted incidence rates are calculated using CDC's National Center for Health Statistics population estimates for the counties included in the surveillance catchment area. Laboratory confirmation is dependent on clinician-ordered influenza testing, and testing for influenza often is underused because of the poor reliability of rapid test results and greater reliance on clinical diagnosis for influenza. Therefore, cases identified as part of influenza hospitalization surveillance likely are an underestimation of the actual number of persons hospitalized with influenza.

**FIGURE 2. Percentage of outpatient visits for influenza-like illness (ILI)\* reported to CDC, by surveillance week — U.S. Outpatient Influenza-like Illness Surveillance Network, 2018–2019† influenza season and selected previous influenza seasons**



\* Defined as fever (temperature of  $\geq 100^{\circ}\text{F}$  [ $\geq 37.8^{\circ}\text{C}$ ], oral or equivalent) and cough or sore throat, without a known cause other than influenza.

† As of June 14, 2019.

occurred during the week ending March 16, 2019 (week 11). The hospitalization rate was highest among persons aged  $\geq 65$  years, who accounted for approximately 47% of reported influenza-associated hospitalizations. By age group, the cumulative hospitalization rate per 100,000 population was 72.0 among children aged 0–4 years, 20.4 among children and adolescents aged 5–17 years, 25.8 among adults aged 18–49 years, 80.7 among adults aged 50–64 years, and 221.7 among adults aged  $\geq 65$  years. Among all influenza-associated hospitalizations, 17,993 (95.5%) were associated with influenza A virus, 727 (3.9%) with influenza B virus, 41 (0.2%) with influenza A virus and influenza B virus coinfection, and 86 (0.5%) with influenza virus for which the type was not determined. Among 6,360 (35.3%) with influenza A subtype information, 3,367 (52.9%) were influenza A(H1N1)pdm09 viruses, and 2,993 (47.1%) were influenza A(H3N2) viruses.

Complete medical chart abstraction data in FluSurv-NET will not be finalized until later in 2019; however, as of June 13, 2019, data were available for 7,531 (40.0%) hospitalized adults and children with laboratory-confirmed influenza. Among 6,399

hospitalized adults with information on underlying medical conditions, 92.6% had at least one reported underlying medical condition that placed them at high risk<sup>§§§</sup> for influenza-associated complications. The most commonly reported underlying medical conditions among adults were cardiovascular disease (45.0%), metabolic disorders (42.9%), obesity (39.4%), and chronic lung disease (29.9%). Among 1,132 hospitalized children with such information, 55.0% had at least one underlying

<sup>§§§</sup> Persons at higher risk include 1) children aged  $< 2$  years; 2) adults aged  $\geq 65$  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  $\leq 18$  years who are receiving long-term aspirin therapy; 7) American Indians/Alaska Natives; 8) persons with extreme obesity (i.e., body mass index  $\geq 40$ ); and 9) residents of nursing homes and other chronic care facilities.

medical condition; those most commonly reported were asthma (27.1%) and neurologic disorder (14.7%). Among 759 hospitalized females aged 15–44 years with information on pregnancy status, 152 (28.7%) were pregnant.

### Pneumonia and Influenza-Associated Mortality

CDC tracks pneumonia and influenza (P&I)–attributed deaths through CDC’s National Center for Health Statistics (NCHS) Mortality Surveillance System data. The percentages of deaths attributed to P&I are released 2 weeks after the week of death to allow for collection of sufficient data to produce a stable P&I mortality percentage. During the 2018–19 season, according to NCHS data, the proportion of deaths attributed to P&I was at or above the epidemic threshold<sup>\*\*\*</sup> for 10 weeks during the weeks ending January 5–26, 2019 (weeks 1–4), the weeks ending February 16–March 2, 2019 (weeks 7–9), and the weeks ending March 16–30, 2019 (weeks 11–13). Nationally, mortality attributed to P&I peaked two times at 7.7% during the weeks ending February 23 (week 8) and March 16, 2019 (week 11).

### Influenza-Associated Pediatric Mortality

During September 30, 2018–May 18, 2019, 116 laboratory-confirmed influenza-associated pediatric deaths were reported to CDC from Chicago, New York City, and 41 states. Two deaths occurred in non-U.S. residents. Twenty-five (22%) of the deaths were associated with influenza A(H3N2) infection, 43 (37%) with influenza A(H1N1)pdm09, 39 (34%) with an influenza A virus for which no subtyping was performed, eight (7%) with an influenza B virus, and one (1%) with an influenza virus for which the type was not determined. The mean age of the pediatric deaths reported this season was 6.1 years (range = 2 months–17 years); 75 (66%) children died after admission to the hospital. Among the 104 children with a known medical history, 53 (51%) had at least one underlying medical condition recognized by the Advisory Committee on Immunization Practices (ACIP) as placing them at high risk for influenza-related complications. Among the 89 children who were eligible for influenza vaccination (age  $\geq$  6 months at date of onset) and for whom vaccination status was known, 30 (34%) had received at least 1 dose of influenza vaccine before illness onset (25 were fully vaccinated according to 2018 ACIP recommendations, and five had received 1 of 2 recommended doses).

<sup>\*\*\*</sup> The seasonal baseline proportion of 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.

### Severity Assessment

In 2017, CDC implemented a new methodology to classify influenza season severity using three indicators: 1) the percentage of visits to outpatient clinics for ILI (from ILINet); 2) the rates of influenza-associated hospitalizations (from FluSurv-Net); and 3) the percentage of deaths resulting from pneumonia or influenza (from NCHS) (*1*). This approach uses data from past influenza seasons to calculate three intensity thresholds (<https://www.cdc.gov/flu/professionals/classifies-flu-severity.htm>). These intensity thresholds represent the historic chance that surveillance system data exceeded a certain threshold. CDC then classifies the severity of the current influenza season by determining which intensity threshold was exceeded by at least two of the peak values from these indicators. The severity of the 2018–19 season was thus classified as moderate overall, as well as by age group (for children and adolescents, adults, and older adults).

### Preliminary Estimates of Influenza Burden

CDC uses the cumulative rates of influenza-associated hospitalizations reported through FluSurv-NET and a mathematical model<sup>\*\*\*\*</sup> to estimate the number of persons who have been symptomatically ill with influenza who had a medical visit, were hospitalized, or died related to influenza. Using data available from October 1, 2018, to May 4, 2019, CDC estimates that influenza virus infection has caused 37.4 million–42.9 million symptomatic illnesses; 17.3 million–20.1 million medical visits; 531,000–647,000 hospitalizations; and 36,400–61,200 deaths in the United States.

### Discussion

The 2018–19 U.S. influenza season differed from recent seasons in that there were two waves of influenza A activity of similar magnitude during the season. Influenza A(H1N1)pdm09 viruses predominated overall and represented the most frequently detected influenza A virus from October 2018 to mid-February 2019; influenza A(H3N2) viruses were reported more frequently than were A(H1N1)pdm09 viruses from late February through mid-May 2019. The predominant influenza A virus also differed by geographic region and age group. In contrast to the number of influenza A viruses reported, the number of influenza B viruses reported was low, compared with previous seasons, accounting for 4% of influenza viruses reported by public health laboratories.

The 2018–19 influenza season was longer than recent influenza seasons, and ILI activity was at or above baseline for 21 consecutive weeks. Compared with hospitalization

<sup>\*\*\*\*</sup> <https://www.cdc.gov/flu/about/burden/preliminary-in-season-estimates.htm>.

rates during the previous five influenza seasons, the 2018–19 cumulative influenza-associated hospitalization rate (65.3 per 100,000 population) was most similar to rates observed during 2014–15 (64.1) and 2016–17 (62.0) and well below those observed during 2017–18 (102.9). Hospitalization rates for children aged <17 years exceeded those during the 2013–14 through 2016–17 seasons and were similar to those during the 2017–18 season, whereas hospitalization rates for adults aged 18–64 years exceeded those in 2013–14 through 2016–17 but were less than those during the 2017–18 season. For persons aged ≥65 years, this season's hospitalization rates were below those observed during the three most recent H3N2-predominant seasons (2014–15, 2016–17, and 2017–18) but higher than the two H1N1-predominant seasons (2013–14 and 2015–16). Compared with P&I-attributed mortality during the previous five seasons, 2018–19 P&I-attributed mortality was most similar to the 2015–16 season and was lower than that during the other four seasons.

Most of the influenza A(H1N1)pdm09 viruses characterized (using hemagglutination inhibition tests with ferret antisera) were antigenically similar to the cell culture–propagated reference virus representing the 2018–19 Northern Hemisphere influenza vaccine virus, but considerable genetic diversity among currently circulating influenza A(H1N1)pdm09 viruses belonging to clade 6B.1A was observed. The increased circulation of clade 3C.3a viruses strongly contributed to the increasing proportion of A(H3N2) viruses that were antigenically distinct from the reference virus representing the A(H3N2) vaccine component of the 2018–19 Northern Hemisphere vaccines. Viruses from clade 3C.3a were well inhibited by ferret antisera raised against recent 3C.3a cell culture–propagated reference viruses, including A/Kansas/14/2017, the reference virus representing the A(H3N2) component for the 2019–20 Northern Hemisphere influenza vaccines (4). All B/Yamagata lineage viruses and the majority of B/Victoria lineage viruses tested were antigenically similar to the reference viruses representing the components of vaccines for the 2018–19 Northern Hemisphere influenza season. However, B/Victoria lineage subclade V1A-3Del viruses, which were antigenically distinct from the B/Victoria lineage vaccine virus, were more frequently reported in the United States toward the end of the season. The majority (>99%) of influenza viruses collected and tested since October 1, 2018, were susceptible to oseltamivir and peramivir, and all tested viruses were susceptible to zanamivir and baloxavir.

Since the 2010–11 season, CDC estimates that during each influenza season, influenza virus infection has caused 9.3 million–49 million symptomatic illnesses, 4.3 million–23 million medical visits, 140,000–960,000 hospitalizations, and

## Summary

### What is already known about this topic?

CDC collects, compiles, and analyzes data on influenza activity and viruses in the United States.

### What is added by this report?

The 2018–19 influenza season was a moderate severity season with two waves of influenza A activity of similar magnitude during the season: A(H1N1)pdm09 predominated from October 2018 to mid-February 2019, and A(H3N2) activity increased from mid-February through mid-May.

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

Receiving a seasonal influenza vaccine each year remains the best way to protect against seasonal influenza and its potentially severe consequences. Testing for seasonal influenza viruses and monitoring for emergence of antigenic drift variant viruses should continue year-round.

12,000–79,000 deaths.<sup>††††</sup> Preliminary estimates for the 2018–19 season fall within these ranges.

Receiving a seasonal influenza vaccine each year remains the best way to protect against seasonal influenza and its potentially severe consequences. Although seasonal influenza activity is currently below baseline, influenza illnesses are often reported during the summer. Influenza should be suspected in ill travelers returning from countries with ongoing influenza activity. Variant influenza infections associated with exposure to swine during animal exhibitions are reported each summer (7). Suspected variant influenza infections should be referred to state public health departments for testing. Treatment as soon as possible with influenza antiviral medications is recommended for patients with confirmed or suspected influenza who have severe, complicated, or progressive illness; who require hospitalization; or who are at high risk for influenza-associated complications (8). Providers should not rely on less sensitive assays such as rapid antigen detection influenza diagnostic tests to inform treatment decisions. Four influenza antiviral drugs are approved by the Food and Drug Administration for treatment of acute uncomplicated influenza within 2 days of illness onset and are recommended for use in the United States during the 2018–19 season: oseltamivir, zanamivir, peramivir, and baloxavir.

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 online (<https://www.cdc.gov/flu>).

<sup>††††</sup> <https://www.cdc.gov/flu/about/burden/past-seasons.html>.

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## References

1. Biggerstaff M, Kniss K, Jernigan DB, et al. Systematic assessment of multiple routine and near real-time indicators to classify the severity of influenza seasons and pandemics in the United States, 2003–2004 through 2015–2016. *Am J Epidemiol* 2018;187:1040–50. <https://doi.org/10.1093/aje/kwx334>
2. World Health Organization. Laboratory methodologies for testing the antiviral susceptibility of influenza viruses: neuraminidase inhibitor (NAI). Geneva, Switzerland: World Health Organization; 2019. [https://www.who.int/influenza/gists\\_laboratory/antiviral\\_susceptibility/nai\\_overview/en/](https://www.who.int/influenza/gists_laboratory/antiviral_susceptibility/nai_overview/en/)
3. Gubareva LV, Mishin VP, Patel MC, et al. Assessing baloxavir susceptibility of influenza viruses circulating in the United States during the 2016/17 and 2017/18 seasons. *Euro Surveill* 2019;24. <https://doi.org/10.2807/1560-7917.ES.2019.24.3.1800666>
4. World Health Organization. Recommended composition of influenza virus vaccines for use in the 2019–2020 northern hemisphere influenza season. Geneva, Switzerland: World Health Organization; 2019. [https://www.who.int/influenza/vaccines/virus/recommendations/2019\\_20\\_north/en/](https://www.who.int/influenza/vaccines/virus/recommendations/2019_20_north/en/)
5. Food and Drug Administration Center for Biologics Evaluation and Research. Vaccines and Related Biological Products Advisory Committee meeting announcement, March 6–7, 2019. Silver Spring, MD: Department of Health and Human Services, Food and Drug Administration; 2019. <https://www.fda.gov/advisory-committees/advisory-committee-calendar/vaccines-and-related-biological-products-advisory-committee-march-6-7-2019-meeting-announcement>
6. Food and Drug Administration Center for Biologics Evaluation and Research. Vaccines and Related Biological Products Advisory Committee meeting announcement, March 22, 2019. Silver Spring, MD: Department of Health and Human Services, Food and Drug Administration; 2019. <https://www.fda.gov/advisory-committees/advisory-committee-calendar/vaccines-and-related-biological-products-advisory-committee-march-22-2019-meeting-announcement>
7. 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>
8. 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).

## Nationwide Shortage of Tuberculin Skin Test Antigens: CDC Recommendations for Patient Care and Public Health Practice

CDC is expecting a 3–10 month nationwide shortage of Aplisol, a product of Par Pharmaceuticals, and one of two purified-protein derivative (PPD) tuberculin antigens licensed by the Food and Drug Administration (FDA) for use in performing tuberculin skin tests. This time frame is the manufacturer's current estimate and is subject to change. The manufacturer notified CDC that they anticipate an interruption of supply of Aplisol 5 mL (50 multidose vials) beginning in June 2019, followed by an interruption of the supply of Aplisol 1 mL (10 multidose vials) in November 2019. The expected shortage of Aplisol 1 mL could occur before November 2019 if demand increases before then. Information on the status of this supply interruption will be updated at FDA's Center for Biologics Evaluation and Research—Regulated Products: Current Shortages website (<https://www.fda.gov/vaccines-blood-biologics/safety-availability-biologics/cber-regulated-products-current-shortages>). This report includes CDC recommendations for mitigating a reduction in tuberculosis (TB) testing capability resulting from the anticipated Aplisol shortage (1).

Two types of immunological methods (tuberculin skin tests [TSTs] and interferon-gamma release assay [IGRA] blood tests) are used for detecting *Mycobacterium tuberculosis* infection. TSTs and IGRAs are used for the diagnosis of latent TB infection and can aid in the diagnosis of TB disease, but additional evaluation and testing is necessary to distinguish between latent TB infection and TB disease to determine the appropriate treatment (2). When findings such as chest radiography and mycobacterial cultures are sufficient for confirming or excluding a TB diagnosis, the results from a TST or an IGRA blood test might not be needed (2). However, most TB cases in the United States are diagnosed through a combination of findings, including results from one of these tests. When TB disease is strongly suspected, specific treatment should be initiated, regardless of results from TST or an IGRA blood test (3,4).

Two FDA-approved PPD tuberculin antigen products are available in the United States for use in performing TSTs: Tubersol (Sanofi-Pasteur) and Aplisol. In controlled studies, the concordance between the two products is high (5).

### Recommendations

CDC recommends the following three general approaches to mitigate a reduction in TB testing capability resulting from the expected shortage of Aplisol:

- Substitute IGRA blood tests for TSTs. Clinicians who use the IGRA blood tests should be aware that the criteria for test interpretation are different from the criteria for interpreting TSTs (3).
- Substitute Tubersol for Aplisol for skin testing. In studies, the two skin test products give similar results for most patients (5).
- Prioritize allocation of TSTs, in consultation with state and local public health authorities. Prioritization might require the deferment of testing some persons. CDC recommends testing only for persons who are at risk for TB (6–8). Groups at high risk for TB infection include 1) persons who are recent contacts exposed to persons with TB disease; 2) those born in or who frequently travel to countries where TB disease is common; 3) those who currently or previously lived in large group settings (such as homeless shelters or correctional facilities); 4) persons with compromised immune systems, including those with health conditions or taking medications that might alter immunity; and 5) children, especially those aged <5 years, if they are in one of the risk groups noted above.

Although overall test concordance is high, switching between PPD skin test products or TSTs and blood tests in serial testing might result in apparent conversions from negative to positive or reversions from positive to negative that might be attributable to inherent interproduct or intermethod discordance rather than change in *M. tuberculosis* infection status (3,9). Clinicians should assess test results based on the person's likelihood of infection and risk for progression to TB disease, if infected (2).

In settings with a low likelihood of TB exposure, the deferment of routine serial testing should be considered in consultation with public health and occupational health authorities. Annual TB testing of health care personnel is not recommended unless there is a known exposure or ongoing transmission (9).

## References

1. CDC Health Alert Network. Nationwide shortage of tuberculin skin test antigens: CDC recommendations for patient care and public health practice. Atlanta, GA: US Department of Health and Human Services, CDC; 2019. <https://emergency.cdc.gov/han/han00420.asp?deliveryName=DM1457>
2. Lewinsohn DM, Leonard MK, LoBue PA, et al. Official American Thoracic Society/Infectious Diseases Society of America/Centers for Disease Control and Prevention clinical practice guidelines: diagnosis of tuberculosis in adults and children. *Clin Infect Dis* 2017;64:e1–33. <https://doi.org/10.1093/cid/ciw694>
3. Mazurek GH, Jereb J, Vernon A, LoBue P, Goldberg S, Castro K; IGRA Expert Committee; CDC. Updated guidelines for using interferon gamma release assays to detect *Mycobacterium tuberculosis* infection—United States, 2010. *MMWR Recomm Rep* 2010;59(No. RR-5).
4. Nahid P, Dorman SE, Alipanah N, et al. Official American Thoracic Society/Centers for Disease Control and Prevention/Infectious Diseases Society of America clinical practice guidelines: treatment of drug-susceptible tuberculosis. *Clin Infect Dis* 2016;63:e147–95. <https://doi.org/10.1093/cid/ciw376>
5. Villarino ME, Burman W, Wang YC, et al. Comparable specificity of 2 commercial tuberculin reagents in persons at low risk for tuberculous infection. *JAMA* 1999;281:169–71. <https://doi.org/10.1001/jama.281.2.169>
6. American Thoracic Society; CDC. Targeted tuberculin testing and treatment of latent tuberculosis infection. *MMWR Recomm Rep* 2000;49(No. RR-6).
7. CDC. Guidelines for the investigation of contacts of persons with infectious tuberculosis; recommendations from the National Tuberculosis Controllers Association and CDC, and guidelines for using the QuantiFERON®-TB Gold test for detecting *Mycobacterium tuberculosis* infection, United States. *MMWR Recomm Rep* 2005;54(No. RR-15).
8. Bibbins-Domingo K, Grossman DC, Curry SJ, et al.; US Preventive Services Task Force. Screening for latent tuberculosis infection in adults: US Preventive Services Task Force recommendation statement. *JAMA* 2016;316:962–9. <https://doi.org/10.1001/jama.2016.11046>
9. Sosa LE, Njie GJ, Lobato MN, et al. Tuberculosis screening, testing, and treatment of U.S. health care personnel: recommendations from the National Tuberculosis Controllers Association and CDC, 2019. *MMWR Morb Mortal Wkly Rep* 2019;68:439–43. <https://doi.org/10.15585/mmwr.mm6819a3>

## Notes from the Field

### Meningeal and Pulmonary Tuberculosis on a Commercial Fishing Vessel — Hawaii, 2017

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In December 2016, U.S. Customs and Border Protection notified the CDC Honolulu Quarantine Station of a crewman on a commercial fishing vessel who was hospitalized with suspected tuberculosis (TB); the crewman, in his mid-30s, was unconscious, intubated, and dependent upon mechanical ventilation to maintain his respiratory status. He was a native of a high TB-burden country (one with TB incidence exceeding 10 cases per 100,000 population per year)\* in the Pacific region. Nine days earlier, he had been hospitalized in Hawaii following a 1-month history of headache, fever, night sweats, chills, fatigue, weight loss, breathing difficulties, and cough and recent onset of abdominal pain, vomiting, dizziness, and blurred vision. Brain computerized tomography (CT) and magnetic resonance imaging scans showed lesions in the left basal ganglia and left temporal lobe; chest CT showed multiple bilateral lung opacities with central cavitation. Pathology results from a lung biopsy demonstrated acid-fast bacilli with molecular and culture tests positive for *Mycobacterium tuberculosis* complex, susceptible to all first-line drugs. Cerebrospinal fluid demonstrated low glucose (23 mg/dL), elevated protein (247 mg/dL), and elevated white blood cell count (298 cells/uL) with a relative lymphocytic predominance (50%), consistent with TB meningitis. Testing for human immunodeficiency virus infection was negative, and the patient had no medical comorbidities. The Hawaii Department of Health (Hawaii DOH) was contacted to assist with the investigation.

Upon confirmation of infectious TB, U.S. Customs and Border Protection, accompanied by the Hawaii Harbor Police called the vessel to port for contact interviews and TB screening by the CDC Honolulu Quarantine Station and Hawaii DOH. The vessel's crew members included the captain, who was a U.S. citizen, and five crew members from the same country as the patient. A fisheries observer with the National Oceanic and Atmospheric Administration, who was a U.S. citizen, was identified as an additional contact. All seven contacts were evaluated by Hawaii DOH; none reported symptoms or signs of active TB. Four crew members had positive tuberculin skin test readings. These four persons were evaluated by Hawaii DOH and had

normal chest x-rays, indicating that they had latent TB infection (LTBI). All four were offered LTBI treatment by Hawaii DOH, but all declined. The patient received standard four-drug treatment while hospitalized, regained consciousness and survived respirator-weaning but remained neurologically incapacitated. He completed 12 months of TB treatment. After 426 days, he was repatriated to his country of origin.

Meningeal TB is the most severe form of TB and is associated with high morbidity (5%–24%) (e.g., stroke, seizure, hydrocephalus, vision impairment, and hearing impairment) and mortality (10%–20%), even in high-resource countries (1). Infection of the central nervous system occurs by hematogenous dissemination of *Mycobacterium tuberculosis* from the lungs (2). Neurologic complications are most common when diagnosis and treatment are delayed (2,3).

This case highlights the clinical importance of considering TB in persons from high TB-burden countries. Meningeal TB should be suspected in patients who have signs and symptoms of TB disease (e.g., fever, chills, night sweats) as well as neurologic disease (e.g., headache, blurred vision, dizziness) without a plausible alternative diagnosis by history, physical exam, and basic laboratory or radiographic studies. Once extrapulmonary TB is confirmed, an evaluation for pulmonary disease should proceed (4). This patient's delay of >1 month before being evaluated by a health care provider and his resultant neurologic incapacitation highlight the importance of early diagnosis and the challenges associated with accessing health care among mobile populations. Commercial, noncruise maritime crew members might be particularly vulnerable because of their remote locations, lack of onboard medical resources, and possible inadmissibility to the nearest port of entry. Crew members on commercial vessels are also at high risk for infection if exposed to communicable disease on board because of their close living and working conditions.

A number of factors affect initiation of LTBI therapy in mobile populations, including provider communication about treatment for LTBI versus active disease, barriers to access LTBI therapy, duration of treatment, financial and medical risk of medications, patient willingness to accept therapy, and provider willingness to prescribe treatment given the risk for loss to follow-up. Approximately 4%–6% of persons with untreated LTBI will develop infectious TB (4). Although the four contacts with LTBI declined treatment, this contact investigation demonstrates the feasibility of successful evaluation and management of TB close contacts even in challenging settings.

\* Countries with the highest number of incident TB cases and that account for the highest global percentage of new TB cases. <https://www.tbfacts.org/countries-tb/>.

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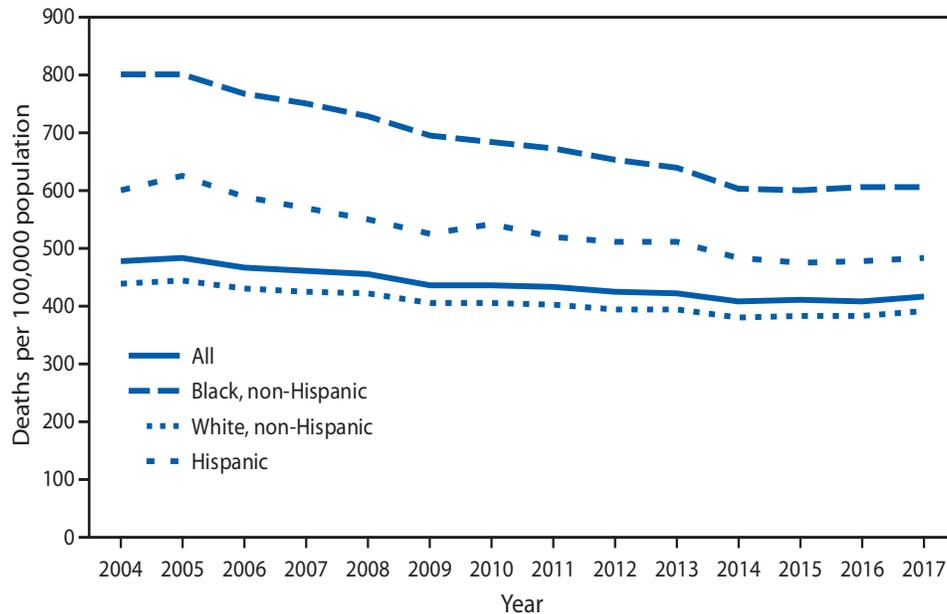
### References

1. Merkler AE, Reynolds AS, Gialdini G, et al. Neurological complications after tuberculous meningitis in a multi-state cohort in the United States. *J Neurol Sci* 2017;375:460–3. <https://doi.org/10.1016/j.jns.2017.02.051>
2. Rock RB, Olin M, Baker CA, Molitor TW, Peterson PK. Central nervous system tuberculosis: pathogenesis and clinical aspects. *Clin Microbiol Rev* 2008;21:243–61. <https://doi.org/10.1128/CMR.00042-07>
3. Brancusi F, Farrar J, Heemskerk D. Tuberculous meningitis in adults: a review of a decade of developments focusing on prognostic factors for outcome. *Future Microbiol* 2012;7:1101–16. <https://doi.org/10.2217/fmb.12.86>
4. Lewinsohn DM, Leonard MK, LoBue PA, et al. Official American Thoracic Society/Infectious Diseases Society of America/Centers for Disease Control and Prevention clinical practice guidelines: diagnosis of tuberculosis in adults and children. *Clin Infect Dis* 2017;64:e1–33. <https://doi.org/10.1093/cid/ciw694>

## QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

### Age-Adjusted Death Rates\* from Diabetes Mellitus† as Underlying or Contributing Cause Among Adults Aged ≥65 Years, by Race/Ethnicity — National Vital Statistics System, United States, 2004–2017



\* Deaths per 100,000 population, age-adjusted to the 2000 U.S. standard population.

† Diabetes mellitus deaths are identified with the *International Classification of Diseases, Tenth Revision* codes E10–E14.

During 2004–2017, the death rate from diabetes mellitus as underlying or contributing cause among adults aged ≥65 years decreased from 477.5 per 100,000 in 2004 to 418.1 in 2017. Throughout this period, the death rate was highest among non-Hispanic black adults and lowest among non-Hispanic white adults. During 2004–2017, the death rate decreased from 438.3 per 100,000 to 391.1 among non-Hispanic white adults, from 602.0 to 485.7 among Hispanic adults, and from 804.3 to 607.0 among non-Hispanic black adults.

Source: National Vital Statistics System, 2004–2017. <https://www.cdc.gov/nchs/nvss/deaths.htm>.

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