

National Diabetes Month — November 2018

November is National Diabetes Month. In the United States, approximately 30 million persons are living with diabetes and 84 million with prediabetes (1). Persons with prediabetes are at high risk for developing type 2 diabetes, heart disease, and stroke (2). Likewise, women who have had gestational diabetes (diabetes during pregnancy) are at increased risk for developing type 2 diabetes later in life (2). However, type 2 diabetes can be prevented or delayed through a structured lifestyle change program that promotes weight loss, healthy eating, and increased physical activity (2). A report on changes in gestational diabetes in the United States is included in this issue of *MMWR* (3).

CDC plays a crucial role in preventing type 2 diabetes and diabetes complications. CDC released an action guide to help pharmacists reach persons at risk for type 2 diabetes who could benefit from the National Diabetes Prevention Program (<https://www.cdc.gov/diabetes/prevention>). In collaboration with partners, CDC launched “Your Health with Joan Lunden and CDC,” a broadcast miniseries that explores prediabetes and diabetes issues (<https://www.cdc.gov/diabetestv>), and the first national prediabetes awareness campaign (<https://www.DoIHavePrediabetes.org>) to encourage persons to learn their prediabetes risk. More information is available at <https://www.cdc.gov/diabetes>.

References

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Prevalence and Changes in Preexisting Diabetes and Gestational Diabetes Among Women Who Had a Live Birth — United States, 2012–2016

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Diabetes during pregnancy increases the risk for adverse maternal and infant health outcomes. Type 1 or type 2 diabetes diagnosed before pregnancy (preexisting diabetes) increases infants’ risk for congenital anomalies, stillbirth, and being large for gestational age (1). Diabetes that develops and is diagnosed during the second half of pregnancy (gestational diabetes) increases infants’ risk for being large for gestational age (1) and might increase the risk for childhood obesity (2); for mothers,

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gestational diabetes increases the risk for future type 2 diabetes (3). In the United States, prevalence of both preexisting and gestational diabetes increased from 2000 to 2010 (4,5). Recent state-specific trends have not been reported; therefore, CDC analyzed 2012–2016 National Vital Statistics System (NVSS) birth data. In 2016, the crude national prevalence of preexisting diabetes among women with live births was 0.9%, and prevalence of gestational diabetes was 6.0%. Among 40 jurisdictions with continuously available data from 2012 through 2016, the age- and race/ethnicity-standardized prevalence of preexisting diabetes was stable at 0.8% and increased slightly from 5.2% to 5.6% for gestational diabetes. Preconception care and lifestyle interventions before, during, and after pregnancy might provide opportunities to control, prevent, or mitigate health risks associated with diabetes during pregnancy.

NVSS collects data for all live births in 50 states, New York City,* and District of Columbia (DC).† The U.S. Standard Certificate of Live Birth (birth certificate) uniformly documents birth-related information across jurisdictions and was revised in 2003 to include distinct fields for preexisting and gestational diabetes; the National Center for Health Statistics recommends information about these conditions be collected

* New York City birth data are reported separately from New York state birth data and are not included in New York state estimates.

† NVSS also collects information for U.S. territories (American Samoa, Guam, Northern Mariana Islands, Puerto Rico, and U.S. Virgin Islands); however, data for these areas are not included in this report.

from prenatal care records, labor and delivery forms, or delivery records.§ The birth certificate also includes information on maternal characteristics, which might be self-reported or collected from medical records.¶ The revised birth certificate was implemented in 40 jurisdictions as of 2012** (representing 86.3% of live births to U.S. residents) and in all jurisdictions as of January 2016.

The national prevalences of preexisting and gestational diabetes were calculated for U.S. resident mothers who had a live birth in 2016. Crude prevalences were calculated overall

§ The National Center for Health Statistics guidance for completing the 2003 revision of the U.S. Standard Certificate of Live Birth recommends information on preexisting diabetes and gestational diabetes be collected from the following sources, in order: the prenatal care record, labor and delivery nursing admission source form, admission history and physical form, or delivery record. <https://www.cdc.gov/nchs/data/dvs/GuidetoCompleteFacilityWks.pdf>.

¶ Most maternal demographic information is collected by self-report, whereas most maternal medical and health data are collected from medical records. Additional information is available from the National Center for Health Statistics guidance for completing the 2003 revision of the U.S. Standard Certificate of Live Birth (https://www.cdc.gov/nchs/data/dvs/birth_edit_specifications.pdf); however, guidance might vary by jurisdiction because recommendations may be modified to better suit each jurisdiction's needs.

** The 40 jurisdictions that adopted the revised birth certificate by 2012 are California, Colorado, Delaware, District of Columbia, Florida, Georgia, Idaho, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maryland, Massachusetts, Michigan, Minnesota, Missouri, Montana, Nebraska, Nevada, New Hampshire, New Mexico, New York, New York City, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, South Carolina, South Dakota, Tennessee, Texas, Utah, Vermont, Washington, Wisconsin, and Wyoming.

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and by selected maternal characteristics among women with complete information for each particular characteristic^{††}; chi-square tests were used to evaluate differences by characteristic. To examine changes in prevalence of preexisting and gestational diabetes, jurisdiction-specific prevalences were calculated for U.S. resident mothers with a live birth during 2012–2016 and who were residing in jurisdictions that adopted the revised birth certificate by January 1 of the year in which they gave birth; women with missing data on diabetes status (<1%) were excluded from this portion of the analysis. Jurisdiction-specific prevalences were calculated for each year after directly standardizing to the distribution of age and race/ethnicity of U.S. resident mothers with live births in 2012 because these characteristics vary by jurisdiction and are nonmodifiable determinants of diabetes. For 40 jurisdictions with data available from 2012 to 2016 (n = 17,050,514 women; 86% of U.S. resident women with live births during 2012–2016), differences in standardized prevalences between 2012 and 2016 were calculated for each jurisdiction and for all jurisdictions combined; differences were assumed to be independent and were evaluated using the z-statistic. P-values <0.05 were considered statistically significant.

In 2016, the crude national prevalences of preexisting and gestational diabetes were 0.9% and 6.0%, respectively (Table 1); prevalence varied by all characteristics examined (p<0.05). For example, by race/ethnicity, the prevalence of preexisting diabetes was highest among American Indian/Alaska Native women (2.1%) and Native Hawaiian/Pacific Islander women (1.8%), and the prevalence of gestational diabetes was highest among non-Hispanic Asian women (11.1%). The prevalences of both preexisting and gestational diabetes varied by prepregnancy body mass index (BMI): among underweight women, the prevalences of preexisting diabetes and gestational diabetes were 0.3% and 2.9%, respectively; whereas among women with class III obesity, the respective prevalences were 3.2% and 13.9%.

After standardizing for age and race/ethnicity, the 2016 prevalence of preexisting diabetes ranged from 0.5% in California to 1.7% in West Virginia (Table 2) (Figure); prevalence of gestational diabetes ranged from 3.4% in DC to 9.2% in South Dakota (Table 2) (Figure). From 2012 to 2016, among the 40 jurisdictions with continuously available data, the standardized prevalence of preexisting diabetes was stable at 0.8% (Table 2). Statistically significant increases in the prevalence of preexisting diabetes were observed in eight jurisdictions (range = 0.1% [California] to 0.3% [Georgia]); a

^{††} Overall, <1% of live births had missing information on diabetic status. For maternal characteristics, among women with complete information on diabetic status, missing data ranged from <1% for nativity to 2.8% for trimester of entry into prenatal care.

TABLE 1. Unadjusted prevalences of preexisting diabetes and gestational diabetes among women with a live birth, by selected maternal characteristics — United States, 2016

Characteristic*	No.†	% Preexisting diabetes	% Gestational diabetes
Total	3,942,094	0.9	6.0
Age group (yrs)			
<20	211,827	0.4	1.9
20–24	803,153	0.5	3.3
25–29	1,148,057	0.7	5.1
30–34	1,110,010	1.0	7.0
35–39	546,995	1.4	9.6
≥40	122,052	2.1	12.8
Race and Hispanic origin[§]			
White, non-Hispanic	2,054,437	0.7	5.3
Black, non-Hispanic	558,044	1.2	4.8
Asian, non-Hispanic	254,326	0.9	11.1
Hispanic	917,822	1.0	6.6
American Indian/Alaska Native	31,375	2.1	9.2
Native Hawaiian/Pacific Islander	9,337	1.8	8.4
More than one race	80,836	0.9	5.8
Nativity			
U.S.-born	3,024,356	0.8	5.2
Not U.S.-born	909,638	0.9	8.4
Education			
Less than high school	537,990	1.1	6.2
High school graduate	978,917	0.9	5.5
Some college	1,128,682	1.0	6.2
College graduate	784,655	0.6	5.9
More than college	460,768	0.6	6.0
Payment source for delivery			
Medicaid	1,668,864	1.0	5.9
Private	1,936,143	0.8	6.2
Other [¶]	313,437	0.7	5.1
Trimester entry into prenatal care			
First	2,955,378	0.9	6.2
Second	639,593	0.8	5.6
Third or none	235,409	0.7	4.6
Parity			
Nulliparous	1,498,458	0.8	5.2
Primiparous	1,263,445	0.8	5.9
Multiparous	1,165,053	1.0	7.1
Prepregnancy body mass index**			
Underweight	134,392	0.3	2.9
Normal weight	1,699,751	0.4	3.6
Overweight	997,977	0.8	6.1
Obesity Class I	548,092	1.3	8.8
Obesity Class II	266,105	2.0	11.2
Obesity Class III	187,689	3.2	13.9

* Statistically significant (p<0.05) differences in the distribution of preexisting diabetes, gestational diabetes (or no diabetic conditions) were observed by all maternal characteristics.

† The number of women within a characteristic group (e.g., age group) might not sum to the total number of women because of missing information.

§ Race and Hispanic origin are reported separately on the birth certificate. Women reporting Hispanic origin were categorized as Hispanic regardless of their race. Categories represent single-race reporting (i.e., mothers reported only one race); mothers reporting more than one race were categorized as “More than one race.”

¶ Includes insurance provided by TRICARE or the Indian Health Service.

** Prepregnancy body mass index (BMI; kg/m²) classified as underweight (BMI <18.5), normal weight (BMI 18.5–24.9), overweight (BMI 25.0–29.9), obesity class I (BMI 30.0–34.9), obesity class II (35.0–39.9), and obesity class III (BMI ≥40.0).

TABLE 2. Standardized* prevalence of preexisting and gestational diabetes among women with a live birth, by jurisdiction and year — United States, 2012–2016

Jurisdiction	Percentage of women with preexisting diabetes						Percentage of women with gestational diabetes					
	2012	2013	2014	2015	2016	% Difference, 2012 to 2016 (95% CI) [†]	2012	2013	2014	2015	2016	% Difference, 2012 to 2016 (95% CI) [†]
	— [§]	— [§]	— [§]	— [§]	— [§]		— [§]	— [§]	— [§]	— [§]	— [§]	
Alabama	— [§]	— [§]	1.1	1.1	1.1	— [§]	— [§]	— [§]	4.6	4.8	5.3	— [§]
Alaska	— [§]	0.6	1.0	1.0	0.9	— [§]	— [§]	7.2	6.9	6.7	8.3	— [§]
Arizona	— [§]	— [§]	0.8	0.8	0.8	— [§]	— [§]	— [§]	5.7	6.9	6.9	— [§]
Arkansas	— [§]	— [§]	1.0	1.0	1.0	— [§]	— [§]	— [§]	5.2	5.4	5.6	— [§]
California	0.4	0.4	0.4	0.4	0.5	0.1 (0.0 to 0.1) [†]	4.2	4.4	4.7	4.6	4.6	0.4 (0.4 to 0.5) [†]
Colorado	0.7	0.8	0.7	0.6	0.7	0.0 (-0.1 to 0.1)	4.2	4.4	4.2	4.2	4.3	0.1 (-0.1 to 0.4)
Connecticut	— [§]	— [§]	— [§]	— [§]	0.8	— [§]	— [§]	— [§]	— [§]	— [§]	5.7	— [§]
Delaware	1.0	0.9	0.8	0.8	0.9	-0.2 (-0.4 to 0.1)	7.5	6.9	7.9	7.2	7.2	-0.3 (-1.0 to 0.5)
District of Columbia	0.6	0.8	0.8	0.5	0.8	0.1 (-0.2 to 0.4)	2.9	3.0	2.8	2.5	3.4	0.5 (-0.3 to 1.3)
Florida	0.8	0.8	0.7	0.7	0.8	-0.1 (-0.1 to 0.0)	5.0	4.6	4.4	4.3	4.4	-0.5 (-0.7 to -0.4) [†]
Georgia	0.7	0.8	0.9	1.0	0.9	0.3 (0.2 to 0.4) [†]	4.0	4.1	3.8	3.8	4.7	0.7 (0.5 to 0.8) [†]
Hawaii	— [§]	— [§]	0.6	0.5	0.5	— [§]	— [§]	— [§]	3.3	4.5	3.8	— [§]
Idaho	0.7	0.7	0.7	0.7	1.0	0.3 (-0.0 to 0.6)	5.7	6.3	5.6	6.6	5.8	0.1 (-0.7 to 0.9)
Illinois	0.8	0.8	0.7	0.8	0.9	0.1 (0.0 to 0.1)	5.9	6.2	6.0	6.3	6.3	0.3 (0.2 to 0.5)
Indiana	1.1	1.0	1.0	1.0	1.0	-0.0 (-0.2 to 0.1)	6.7	6.2	6.1	6.2	6.9	0.1 (-0.2 to 0.4)
Iowa	1.0	1.0	1.2	1.2	1.3	0.2 (0.0 to 0.4) [†]	7.2	8.0	7.8	8.3	8.4	1.1 (0.6 to 1.6) [†]
Kansas	0.8	0.9	0.9	0.8	0.8	-0.0 (-0.2 to 0.1)	5.8	5.7	6.0	6.0	6.4	0.5 (0.2 to 0.9) [†]
Kentucky	1.1	1.0	1.1	1.1	1.1	0.1 (-0.1 to 0.2)	6.2	5.9	6.1	6.0	6.4	0.2 (-0.2 to 0.6)
Louisiana	0.8	0.9	0.9	1.0	1.0	0.2 (0.1 to 0.3) [†]	5.0	6.1	6.0	5.9	5.9	0.9 (0.5 to 1.2) [†]
Maine	— [§]	— [§]	1.0	1.0	0.9	— [§]	— [§]	— [§]	6.5	6.0	6.2	— [§]
Maryland	0.8	0.8	0.8	0.8	0.8	0.0 (-0.1 to 0.1)	4.8	5.0	5.6	5.9	5.9	1.1 (0.8 to 1.3) [†]
Massachusetts	0.7	0.8	0.7	0.8	0.8	0.1 (-0.0 to 0.2)	5.2	4.8	4.8	5.2	4.8	-0.4 (-0.6 to -0.1) [†]
Michigan	0.9	0.8	0.8	0.8	0.9	0.1 (-0.0 to 0.2)	6.2	5.4	5.5	5.4	5.5	-0.7 (-1.0 to -0.5) [†]
Minnesota	1.1	0.9	1.0	1.0	0.9	-0.1 (-0.3 to 0.0)	7.0	7.1	6.7	6.7	7.1	0.1 (-0.2 to 0.5)
Mississippi	— [§]	0.8	1.0	0.8	0.8	— [§]	— [§]	4.9	4.5	4.3	4.3	— [§]
Missouri	0.8	0.8	0.8	1.0	0.8	0.0 (-0.1 to 0.2)	6.0	5.8	5.9	6.2	6.8	0.8 (0.4 to 1.1) [†]
Montana	0.7	0.5	1.0	0.9	0.9	0.2 (-0.3 to 0.7)	2.8	4.0	4.6	5.2	4.7	1.8 (0.9 to 2.8) [†]
Nebraska	0.9	1.0	1.0	1.0	1.0	0.1 (-0.1 to 0.3)	5.8	6.4	5.7	6.0	6.5	0.7 (0.3 to 1.2) [†]
Nevada	0.9	0.8	0.9	0.9	1.0	0.1 (-0.1 to 0.2)	5.1	5.6	5.5	5.4	5.9	0.8 (0.4 to 1.1) [†]
New Hampshire	0.7	0.8	0.8	0.7	0.7	-0.0 (-0.3 to 0.3)	7.3	6.6	6.9	5.2	5.5	-1.9 (-3.0 to -0.7) [†]
New Jersey	— [§]	— [§]	— [§]	— [§]	0.8	— [§]	— [§]	— [§]	— [§]	— [§]	5.9	— [§]
New Mexico	0.8	0.9	0.9	0.9	0.8	-0.0 (-0.3 to 0.2)	3.4	3.5	4.3	4.4	4.7	1.4 (0.9 to 1.9) [†]
New York	0.7	0.7	0.8	0.7	0.8	0.2 (0.1 to 0.3) [†]	5.2	5.3	5.7	6.0	6.3	1.1 (1.0 to 1.3) [†]
New York City [¶]	0.5	0.5	0.5	0.5	0.5	0.0 (-0.0 to 0.1)	3.9	3.7	4.3	5.2	5.9	2.0 (1.8 to 2.2) [†]
North Carolina	0.8	0.8	0.8	0.9	1.0	0.2 (0.1 to 0.3) [†]	5.9	5.8	5.6	5.5	5.8	-0.0 (-0.2 to 0.2)
North Dakota	0.8	0.7	0.7	1.2	0.8	-0.0 (-0.4 to 0.4)	5.2	5.6	5.3	6.5	6.2	1.0 (-0.0 to 2.1)
Ohio	1.0	1.0	1.0	1.0	1.1	0.1 (-0.0 to 0.2)	7.6	7.8	7.6 ⁷	8.0	8.2	0.6 (0.3 to 0.9) [†]
Oklahoma	1.2	0.8	0.9	0.9	0.9	-0.4 (-0.5 to -0.2) [†]	4.3	4.5	4.7	4.8	5.1	0.9 (0.5 to 1.2) [†]
Oregon	1.0	0.9	1.0	0.8	1.0	0.1 (-0.1 to 0.2)	7.5	8.0	8.1	8.0	8.1	0.6 (0.1 to 1.0) [†]
Pennsylvania	0.8	0.8	0.8	0.8	0.8	-0.0 (-0.1 to 0.0)	5.5	5.4	5.6	5.5	5.5	0.1 (-0.2 to 0.3)
Rhode Island	— [§]	— [§]	— [§]	0.7	0.8	— [§]	— [§]	— [§]	— [§]	6.7	6.1	— [§]
South Carolina	1.0	1.1	0.9	0.9	1.0	0.0 (-0.1 to 0.2)	5.9	6.5	7.3	7.0	7.1	1.1 (0.8 to 1.5) [†]
South Dakota	0.7	0.8	0.8	0.7	0.7	0.1 (-0.3 to 0.4)	6.1	7.1	8.5	8.4	9.2	3.2 (2.1 to 4.3) [†]
Tennessee	1.0	1.1	1.3	1.2	1.2	0.2 (0.1 to 0.3) [†]	7.0	6.7	6.1	6.2	6.1	-0.9 (-1.2 to -0.6) [†]
Texas	0.7	0.7	0.7	0.7	0.6	-0.0 (-0.1 to 0.0)	4.2	4.0	4.5	4.5	4.6	0.4 (0.3 to 0.5) [†]
Utah	0.7	0.7	0.7	0.9	0.7	0.0 (-0.1 to 0.2)	4.8	5.0	5.6	6.4	6.4	1.6 (1.1 to 2.1) [†]
Vermont	0.6	0.8	1.1	1.2	1.0	0.3 (-0.3 to 1.0)	4.4	6.3	4.2	4.0	4.3	-0.1 (-1.5 to 1.2)
Virginia	— [§]	0.6	0.5	0.7	0.7	— [§]	— [§]	4.2	4.8	5.1	5.3	— [§]
Washington	0.8	0.8	0.8	0.9	0.9	0.1 (0.1 to 0.2) [†]	6.7	6.7	7.0	7.6	7.8	1.0 (0.8 to 1.3) [†]
West Virginia	— [§]	— [§]	2.0	1.5	1.7	— [§]	— [§]	— [§]	6.7	7.1	7.2	— [§]
Wisconsin	1.1	1.2	1.0	1.0	1.1	0.1 (-0.1 to 0.2)	7.0	7.1	7.0	6.9	6.6	-0.4 (-0.7 to -0.1) [†]
Wyoming	0.9	0.9	0.8	1.0	0.6	-0.3 (-1.0 to 0.3)	3.3	3.3	3.7	4.6	3.8	0.5 (-0.4 to 1.3)
40 jurisdictions with data during 2012–2016**	0.8	0.8	0.8	0.8	0.8	0.1 (0.0 to 0.1)[†]	5.2	5.2	5.4	5.5	5.6	0.4 (0.4 to 0.5)[†]

Abbreviation: CI = confidence interval.

* Standardized to the age and race/ethnicity distribution of U.S. resident mothers delivering in 2012.

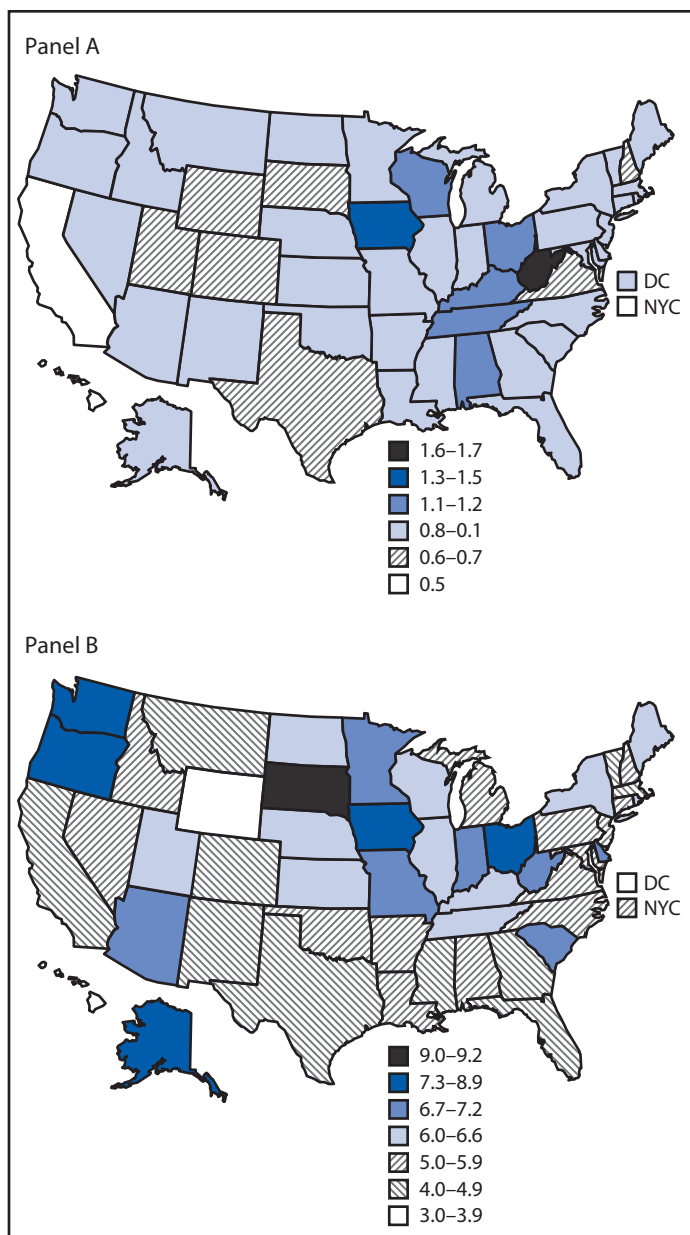
† Statistically significant (p<0.05) difference from 2012 to 2016.

§ A dash indicates revised birth certificates were not available by January 1 of that year for that jurisdiction.

¶ Natality data from New York City are reported separately and are not included in New York state estimates.

** Among the 40 jurisdictions with data during 2012–2016, the sample sizes were 3,391,723 (2012); 3,378,197 (2013); 3,435,616 (2014); 3,434,815 (2015); and 3,410,163 (2016).

FIGURE. Standardized* prevalence of preexisting (panel A) and gestational (panel B) diabetes among women who had a live birth — United States, 2016



Abbreviations: DC = District of Columbia; NYC = New York City.

* Standardized to age and race/ethnicity distribution of U.S. resident mothers delivering in 2012.

significant decrease was observed only for Oklahoma (0.4%). From 2012 to 2016, the standardized prevalence of gestational diabetes increased from 5.2% to 5.6%. Statistically significant increases in the prevalence of gestational diabetes were observed in 22 jurisdictions (range = 0.3% [Illinois] to 3.2% [South Dakota]); significant decreases were observed in six jurisdictions (range = 0.4% [Massachusetts] to 1.9% [New Hampshire]).

Discussion

In 2016, the crude national prevalences of preexisting and gestational diabetes were 0.9% and 6.0%, respectively.^{§§} From 2012 to 2016 among 40 jurisdictions with continuously available data, the age- and race/ethnicity-standardized prevalence of preexisting diabetes remained stable (<0.1 percentage point change), and the prevalence of gestational diabetes increased by 0.4 percentage point. Changes in preexisting and gestational diabetes reported here extend findings from two studies using hospital discharge data from 19 states; these studies found the age-adjusted prevalence of preexisting diabetes increased from 0.7% to 0.9% from 2000 to 2010, and the prevalence of gestational diabetes increased from 3.7% to 5.8% (4,5). Observed increases in the prevalence of preexisting and gestational diabetes might reflect, in part, recent increases in the prevalence of prepregnancy obesity.^{¶¶} Estimates of preexisting diabetes may be leveling off compared to what has been seen in recent years. The high prevalence of gestational diabetes in Asian women is consistent with previous literature (5). Preconception care and lifestyle interventions before, during, and after pregnancy might provide opportunities to control, prevent, or mitigate health risks associated with diabetes during pregnancy.

Preconception care refers to health care before pregnancy that optimizes a woman's health and pregnancy-related outcomes, should a pregnancy occur.^{***} Preconception care provides an opportunity to reinforce the importance of diabetes management among reproductive-aged women with type 1 or type 2 diabetes and might reduce adverse pregnancy outcomes by improving glycemic control before critical developmental stages of the fetus early in pregnancy (6). Because prepregnancy overweight and obesity are strongly associated with developing gestational diabetes, preconception care offers an opportunity to provide all women with recommended BMI screening and to refer women with obesity to intensive multicomponent behavioral interventions.^{†††}

Gestational diabetes strongly predicts the development of future type 2 diabetes (3). Women with gestational diabetes are recommended to receive testing for type 2 diabetes 4–12 weeks postpartum and, if diabetes is detected, referred for follow-up

^{§§} Findings are consistent with prevalence estimates reported by the National Center for Health Statistics (https://www.cdc.gov/nchs/data/nvsr/nvsr67/nvsr67_01.pdf).

^{¶¶} <https://www.cdc.gov/mmwr/volumes/66/wr/mm665152a3.htm>.

^{***} <https://www.cdc.gov/mmwr/preview/mmwrhtml/rr5506a1.htm>.

^{†††} The U.S. Preventive Services Task Force recommends screening all adults for obesity and referrals for patients with obesity to intensive, multicomponent behavioral interventions (<https://www.uspreventiveservicestaskforce.org/Page/Document/RecommendationStatementFinal/obesity-in-adults-screening-and-management>).

care; lifelong monitoring is recommended for women with normal results.^{§§§} Although national estimates of postpartum diabetes testing are unavailable, some studies report suboptimal testing rates (7), suggesting missed opportunities to provide health care for women with diabetes and those at risk for developing diabetes.

Structured lifestyle change programs that promote a healthy diet and increase physical activity, such as CDC-recognized programs coordinated through the National Diabetes Prevention Program, reduce the risk for type 2 diabetes in nonpregnant populations at high risk.^{¶¶¶} During the first half of pregnancy, lifestyle interventions might reduce the risk for developing gestational diabetes; however, additional research is needed to understand the most successful intervention designs (8). Among women who had gestational diabetes but did not develop type 2 diabetes after pregnancy, postpartum lifestyle interventions have been found to reduce postpartum weight retention and improve markers of insulin resistance (9). Importantly, postpartum mothers face unique barriers to engaging in lifestyle interventions, including childcare responsibilities and time constraints (9).

The findings in this report are subject to at least five limitations. First, prevalences of preexisting and gestational diabetes might be underestimated because of underreporting or incomplete birth certificate information, the degree of which might vary by jurisdiction, or because this study was limited to live births; studies indicate sensitivity of identifying preexisting diabetes from birth certificates ranges from 47%–52%, whereas sensitivity for identifying gestational diabetes ranges from 46%–83% (10). Second, recommendations for gestational diabetes screening changed in 2014, and diagnostic criteria might vary by individual practice; consequently, differences in prevalence over time or by jurisdiction might reflect variations in screening or diagnostic practices. Third, analyses examining changes over time were limited to 40 jurisdictions with available data and, as a result, do not represent the entire U.S. population of women giving birth. Fourth, differences in standardized prevalences between the two times do not necessarily imply a steady rate of change during the entire period, which might not reflect actual variation observed. Finally, some statistically significant findings might be driven by large sample sizes and might not reflect a meaningful change.

In 2016, the national prevalences of preexisting and of gestational diabetes were 0.9% and 6.0%, respectively, and

Summary

What is already known about this topic?

Diabetes diagnosed before (preexisting diabetes) and during (gestational diabetes) pregnancy increases the risk for adverse infant and maternal health outcomes. Recent prevalence and trend estimates for these conditions have not been reported.

What is added by this report?

In 2016, the national prevalences of preexisting and gestational diabetes were 0.9% and 6.0%, respectively. Among 40 jurisdictions, the age- and race/ethnicity-standardized preexisting diabetes prevalence was stable at 0.8%, and the gestational diabetes prevalence increased from 5.2% to 5.6%.

What are the implications for public health practice?

Changes in preexisting and gestational diabetes suggest strategies before, during, and after pregnancy are needed to prevent, control, or mitigate risks associated with these conditions.

prevalences of both conditions increased slightly from 2012 to 2016; notably, standardized prevalences and changes over time varied by jurisdiction. Preconception care and lifestyle interventions before, during, and after pregnancy might prevent, control, or mitigate risks associated with diabetes during pregnancy.

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Hepatitis A Virus Outbreaks Associated with Drug Use and Homelessness — California, Kentucky, Michigan, and Utah, 2017

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During 2017, CDC received 1,521 reports of acute hepatitis A virus (HAV) infections from California, Kentucky, Michigan, and Utah; the majority of infections were among persons reporting injection or noninjection drug use or homelessness. Investigations conducted by local and state health departments indicated that direct person-to-person transmission of HAV infections was occurring, differing from other recent, large HAV outbreaks attributed to consumption of contaminated commercial food products. Outbreaks with direct HAV transmission among persons reporting drug use or homelessness signals a shift in HAV infection epidemiology in the United States, and vaccination of these populations at high risk can prevent future outbreaks.

Epidemiologic Investigation

Outbreak cases were defined as those meeting the 2012 CDC-Council of State and Territorial Epidemiologists' (CSTE) definition of acute hepatitis A infection,* having a specimen matching an outbreak strain, or an epidemiologic link to a previously identified case. Local and state health department personnel reviewed clinical charts and interviewed patients using standard questionnaires that evaluated risk factors associated with infection, including recent drug use, sexual history, housing status, recent international travel, and contact with another person with HAV infection.

Among states reporting increases in HAV infections to CDC outside or inside the National Notifiable Disease Surveillance System, only California, Kentucky, Michigan, and Utah reported sustained within-state transmission. This report includes outbreaks that occurred during 2017 in these four states. Additional cases reported from other states were excluded because they were attributed to HAV exposure during travel to one of the four outbreak states, and because prolonged, ongoing transmission did not occur in the other states.

During 2017, a total of 1,521 outbreak-associated HAV cases were reported from California, Kentucky, Michigan, and Utah, with 1,073 (71%) hospitalizations and 41 (3%) deaths (Table 1). Among patients for whom clinical or laboratory records were available for review, 42 (3%) had confirmed or probable hepatitis B virus coinfection, and 341 (22%) had

confirmed or probable hepatitis C virus coinfection. Overall, 866 (57%) patients reported drug use, homelessness, or both (Table 2). Among all cases, 818 (54%) had an indication for hepatitis A vaccination before becoming infected (i.e., using drugs or being men who had sex with men [MSM]) as recommended by the Advisory Committee on Immunization Practices (ACIP) (1).

Laboratory Investigation

When available, serum specimens from patients who met the CSTE case definition were sent to CDC's Division of Viral Hepatitis laboratory for HAV RNA isolation, genotyping, and genetic characterization. HAV RNA was extracted from immunoglobulin M antibody-positive serum samples and used to amplify and Sanger-sequence a 315-base-pair fragment of the VP1/P2B region (2). During 2017, 1,169 specimens from outbreak-associated cases from the four affected states were sent to CDC for additional testing. A total of 1,054 (90%) specimens had HAV confirmed by polymerase chain reaction, 1,014 (96%) of which tested positive for a genotype 1b viral strain. The strains circulating in California, Kentucky, and Utah were genetically different from those circulating in Michigan.

Public Health Response

CDC worked with affected local and state health departments to apply control measures through health advisories, public education, and vaccination clinics that provided outreach and vaccination to the targeted populations. Vaccine was administered in jails, emergency departments, syringe exchange programs, drug treatment facilities, and homeless shelters. In certain jurisdictions, investigation teams also visited homeless encampments to educate and vaccinate unsheltered homeless groups. Although reporting of new outbreak cases in California has ended, new case investigations continue in Kentucky, Michigan, and Utah. Vaccination campaigns also continue for MSM and persons who use drugs or report homelessness in the affected states.

Discussion

After the introduction of hepatitis A vaccine in 1996, the incidence of reported HAV infection steadily decreased in the United States until 2011 and then stabilized at an annual

* <https://wwwn.cdc.gov/nndss/conditions/hepatitis-a-acute/case-definition/2012>.

TABLE 1. Demographic and clinical characteristics of hepatitis A outbreak-associated cases, by state — four states, 2017

Characteristic	California	Kentucky	Michigan	Utah	Total
Total cases, no.	682	59	632	148	1,521
Male, no. (%)	471 (69)	39 (66)	412 (65)	97 (66)	1,019 (67)
Median age, yrs (range)	42 (5–87)	36 (1–84)	41 (<1–90)	38 (22–83)	—
Earliest onset, date	01/17/2017	08/29/2017	01/05/2017	05/08/2017	—
Outcome					
Hospitalized, no. (%)	442 (65)	45 (76)	508 (80)	78 (53)	1,073 (70)
Died, no. (%)	21 (3)	0	20 (3)	0	41 (3)
Comorbidities					
Hepatitis B infection, no. (%)	10 (1)	4 (7)	16 (3)	12 (8)	42 (3)
Hepatitis C infection, no. (%)	116 (17)	29 (49)	165 (26)	31 (21)	341 (22)

TABLE 2. Risk exposures of hepatitis A outbreak-associated patients, by state — four states, 2017

Reported risk exposure	No. (%)				
	California	Kentucky	Michigan	Utah	Total
Homelessness and drug use	247 (36)	27 (46)	64 (10)	75 (51)	395 (26)
Homelessness only	65 (10)	3 (5)	7 (1)	5 (3)	78 (5)
Homelessness, drug use unknown	43 (6)	2 (3)	2 (0.3)	5 (3)	51 (3)
Drug use only	67 (10)	11 (19)	165 (26)	28 (19)	265 (17)
Drug use, homelessness unknown	11 (2)	1 (2)	58 (9)	7 (5)	77 (5)
Neither homelessness nor drug use	190 (28)	13 (22)	286 (45)	15 (10)	504 (33)
Men who have sex with men	18 (3)	4 (7)	61 (10)	1 (0.7)	81 (5)
Unknown	59 (9)	2 (3)	27 (4)	13 (9)	114 (8)

* Percentage totals sum >100 because of men who had sex with men being included independently and as part of “homelessness,” “drug use,” and “neither homelessness nor drug use” categories.

average of approximately 1,600 reported cases, mostly among international travelers returning from countries with endemic HAV or as part of foodborne outbreaks (1,3). HAV outbreaks among illicit drug users were common in the prevaccine era; during the mid-1980s, drug users accounted for >20% of all HAV cases reported to CDC (3,4). However, large community outbreaks within this population rarely occurred after 1996, when hepatitis A vaccine was first recommended for persons who use illicit drugs (3,4).

Person-to-person transmission of HAV between those who report drug use or homelessness can result from unsafe sanitary conditions or specific sexual contact or practices, or it can be parenterally transmitted through contaminated needles or other injection paraphernalia (4–6). Transient housing, economic instability, limited access to health care, and distrust of government services make outbreaks among affected populations more difficult to control, requiring tailored comprehensive public health interventions that address their specific circumstances and needs (5–7).

During 2016, U.S. hospitalization and mortality rates associated with HAV infections were 42% and 0.7%, respectively (3). Increased hospitalization and mortality rates observed in the 2017 HAV outbreaks might be attributable to preexisting illnesses, including chronic hepatitis B and hepatitis C infections, other comorbidities, age, and risk behaviors common among persons reporting drug use and homelessness (e.g., heavy alcohol use) (8).

Increasingly, investigations of HAV infections are using molecular epidemiology to confirm outbreaks (2). Laboratory data, when combined with reliable epidemiologic data, can be effective in understanding transmission networks, particularly among populations distrustful of investigators. The majority of surveillance specimens tested by CDC’s laboratory before 2017 were genotype 1a, the most common genotype in North and South America, but expansion of genotype 1b attributed to the current outbreaks is leading to increased detection of this previously uncommon genotype (2,9).

Vaccination rates among existing ACIP-identified risk groups are unknown but are believed to be low (10). On October 24, 2018, ACIP voted unanimously to add “homelessness” as an indication for ACIP-recommended HAV vaccination (1).[†] Although the outbreak has ended in California, hepatitis A outbreaks among persons reporting drug use or homelessness continue in Kentucky, Michigan, and Utah, and, as of October 12, 2018, >7,000 outbreak associated cases have been reported from 12 states.[§]

Increasing vaccination coverage among all at-risk groups recommended by ACIP to receive hepatitis A vaccine might halt ongoing outbreaks and prevent future large community outbreaks (1).

[†] <https://www.cdc.gov/vaccines/acip/meetings/downloads/agenda-archive/agenda-2018-10-508.pdf>.

[§] <https://www.cdc.gov/hepatitis/outbreaks/2017March-HepatitisA.htm>.

CDC has recommended that local health jurisdictions experiencing HAV outbreaks among persons who report drug use or homelessness ensure procedures are in place for identifying these risk factors and that these groups are vaccinated against HAV infection.[¶] State and local health departments and CDC should be notified of any new suspected clusters of acute HAV infections.

[¶] <https://emergency.cdc.gov/han/han00412.asp>.

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Summary

What is already known about this topic?

Hepatitis A is a vaccine-preventable viral infection of the liver that is commonly transmitted through consumption of microscopic amounts of feces. Outbreaks of hepatitis A infections are infrequent in the United States and are typically associated with contaminated food items.

What is added by this report?

During 2017, California, Kentucky, Michigan, and Utah reported 1,521 hepatitis A infections, mostly among persons who reported drug use or homelessness, signaling a shift in hepatitis A epidemiology from point-source outbreaks associated with contaminated food to large community outbreaks with person-to-person transmission.

What are the implications for public health practice?

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

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Violence Victimization, Substance Use, and Suicide Risk Among Sexual Minority High School Students — United States, 2015–2017

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Youths identifying as lesbian, gay, bisexual, or another non-heterosexual identity (sexual minority youths) report more violence victimization, substance use, and suicide risk than do heterosexual youths (1). These disparities are generally attributed to minority stress (the process through which stigma directed toward sexual minorities influences health outcomes) (2,3). Sexual minority youths might experience negative outcomes associated with minority stress differently across sexual identities, but to date, no nationally representative study has examined differences in victimization, substance use, and suicide risk within sexual minority youth. Using pooled data from the 2015 and 2017 national Youth Risk Behavior Surveys (YRBS), relationships between sexual identity groups and victimization, substance use, and suicide risk were evaluated with sex-stratified logistic regression models. Compared with heterosexual students, bisexual females and all sexual minority males reported more victimization; lesbian and bisexual females reported more use of alcohol, cigarettes, and marijuana; and all sexual minority youths reported elevated high-risk substance use and suicide risk. Programmatic efforts to reduce and prevent victimization, substance use, and suicide risk among sexual minority youths might benefit from consideration of issues within group differences.

Analyses use pooled data from the 2015 and 2017 cycles of the national YRBS, a biennial, school-based survey of U.S. high school students. YRBS uses a three-stage cluster sample design to select a nationally representative sample of students in grades 9–12 attending public and private schools (4). The overall response rate for the 2015 and 2017 YRBS was 60% (both years), and the sample sizes were 15,624 and 14,765, respectively. The combined analytic sample included 30,389 students. Data were weighted to yield nationally representative estimates. Survey procedures protected students' privacy through anonymous/voluntary participation, using local parental permission procedures. CDC's Institutional Review Board approved data collection.

Students were grouped into one of four sexual identity categories (gay/lesbian, bisexual, not sure, and heterosexual) based on their response to "Which of the following best describes you?" Seven items assessed victimization: 1) felt unsafe at or traveling to or from school in past 30 days; 2) ever forced to have sexual intercourse; and in past 12 months, 3) threatened or injured with a weapon at school; 4) experienced sexual dating

violence; 5) experienced physical dating violence; 6) was bullied at school; and 7) was electronically bullied. Three items assessed lifetime substance use of cigarettes, alcohol, and marijuana, and five items assessed lifetime high-risk substance use (cocaine, heroin, methamphetamines, ecstasy, and inhalants). Five items assessed suicide risk in the past 12 months: 1) felt sad or hopeless, 2) considered attempting suicide, 3) made a suicide plan, 4) attempted suicide, and 5) had a suicide attempt treated by a doctor or nurse.

Analyses used statistical software to account for complex sampling design. Unadjusted prevalence estimates with 95% confidence intervals (CIs) were calculated using Taylor series linearization. Sex-stratified logistic regression models, controlling for race/ethnicity and school grade produced adjusted prevalence ratios (APRs) with heterosexual students serving as the referent group. Models were tested for effect modification by sex (i.e., Wald F statistic for interaction between sex and sexual identity) to determine if associations between sexual identity and victimization, substance use, and suicide risk varied by sex. Post-hoc linear contrast t-tests were used to assess additional between-group differences in prevalence of outcomes across sexual identity. Statistical tests were considered significant if p-values were <0.05 or 95% CIs did not include 1.0.

Compared with heterosexual females, bisexual females reported a higher prevalence of feeling unsafe at or traveling to or from school (APR = 1.6), being threatened/injured with a weapon (2.2), having experienced forced sex (2.8), sexual dating violence (1.7), physical dating violence (1.9), bullying at school (1.7), and electronic bullying (2.3) (Table 1). Bisexual females were more likely than were lesbians to report sexual dating violence, and more likely than both lesbian and females not sure of their sexual identity to experience forced sex, bullying at school, and electronic bullying. Males who were gay, bisexual, or not sure of their sexual identity had a higher likelihood than did heterosexual males of reporting all seven indicators of violence victimization. Prevalence estimates and significant effect modifications by sex indicate that associations between sexual minority status and victimization were stronger for males than for females for experiencing forced sex, sexual dating violence, physical dating violence, being bullied at school, and being electronically bullied.

TABLE 1. Unadjusted prevalence and adjusted prevalence ratios of experiences of violence victimization, by sex and sexual identity — National Youth Risk Behavior Survey, United States, 2015–2017*

Experience/Sex	Heterosexual [†]		Lesbian/Gay		Bisexual		Not sure		Interaction by sex p-value
	% (95% CI)	% (95% CI)	APR (95% CI)	% (95% CI)	APR (95% CI)	% (95% CI)	APR (95% CI)		
Felt unsafe at or traveling to/from school[§]									
Females	5.8 (5.0–6.7)	11.0 (7.5–15.9)	1.9 [¶] (1.3–2.9)	9.6 (8.0–11.6)	1.6 [¶] (1.3–2.0)	9.4 (6.7–13.0)	1.6 [¶] (1.1–2.2)	0.135	
Males	4.7 (4.2–5.4)	18.0 (10.9–28.2)	3.7 [¶] (2.4–5.8)	10.6 (6.9–15.8)	2.4 [¶] (1.6–3.7)	11.2 (7.6–16.2)	2.3 [¶] (1.5–3.5)		
Threatened or injured with a weapon at school**									
Females	3.7 (3.2–4.3)	5.9 (3.3–10.3)	1.5 (0.8–2.7)	8.6 (6.9–10.7)	2.2 [¶] (1.7–2.8)	6.1 (3.8–9.8)	1.6 (1.0–2.7)	0.210	
Males	6.5 (5.9–7.2)	15.1 (9.3–23.6)	1.9 [¶] (1.2–2.8)	11.6 (7.6–17.5)	1.8 [¶] (1.2–2.8)	17.2 (12.5–23.1)	2.5 [¶] (1.8–3.4)		
Experienced forced sexual intercourse^{††}									
Females	8.8 (7.7–10.1)	15.4 (11.3–20.6)	1.7 ^{¶,§§} (1.2–2.4)	23.9 (21.2–26.8)	2.8 ^{¶,¶¶} (2.4–3.2)	11.4 (8.0–16.2)	1.3 (0.9–1.8)	0.000	
Males	2.5 (2.1–2.9)	17.1 (11.2–25.3)	6.6 [¶] (4.4–9.8)	8.1 (4.7–13.4)	3.3 [¶] (2.0–5.6)	12.6 (8.9–17.4)	4.7 [¶] (3.1–7.0)		
Experienced sexual dating violence^{***}									
Females	12.0 (10.9–13.3)	10.8 (7.1–16.0)	1.0 ^{§§} (0.6–1.5)	20.8 (17.5–24.6)	1.7 [¶] (1.4–2.1)	18.0 (13.1–24.2)	1.5 [¶] (1.1–2.1)	0.000	
Males	3.3 (2.8–3.9)	24.0 (13.1–39.7)	5.5 [¶] (3.0–10.1)	12.6 (7.6–20.1)	3.9 [¶] (2.3–6.6)	14.7 (9.6–22.0)	4.5 [¶] (2.8–7.2)		
Experienced physical dating violence^{†††}									
Females	9.0 (7.9–10.2)	13.8 (9.1–20.5)	1.5 (0.9–2.3)	17.5 (15.0–20.2)	1.9 [¶] (1.6–2.3)	13.6 (10.0–18.2)	1.5 [¶] (1.1–2.1)	0.008	
Males	6.0 (5.4–6.7)	24.2 (14.6–37.3)	3.2 [¶] (2.0–4.9)	14.5 (8.5–23.8)	2.5 [¶] (1.4–4.2)	21.6 (14.6–30.9)	3.3 [¶] (2.1–5.2)		
Bullied at school^{§§§}									
Females	21.9 (20.5–23.5)	25.6 (19.5–32.8)	1.3 ^{§§} (1.0–1.7)	36.1 (32.0–40.4)	1.7 ^{¶,¶¶} (1.5–1.8)	22.6 (18.8–26.9)	1.0 (0.9–1.2)	0.001	
Males	14.6 (13.7–15.5)	27.7 (20.1–36.9)	2.0 [¶] (1.5–2.8)	33.5 (27.1–40.7)	2.3 [¶] (1.8–2.8)	26.2 (21.3–31.7)	1.8 [¶] (1.4–2.2)		
Electronically bullied^{¶¶¶}									
Females	19.6 (18.4–20.9)	21.4 (15.9–28.1)	1.2 ^{§§} (0.9–1.6)	30.9 (27.3–34.8)	1.6 ^{¶,¶¶} (1.4–1.8)	22.1 (17.8–27.1)	1.2 (0.9–1.4)	0.000	
Males	8.8 (8.0–9.6)	17.7 (12.6–24.3)	1.9 ^{¶,¶¶} (1.3–2.7)	26.3 (20.6–32.8)	3.0 [¶] (2.3–3.9)	20.1 (15.4–25.8)	2.2 [¶] (1.7–3.0)		

Abbreviations: APR = adjusted (for race/ethnicity and grade) prevalence ratio; CI = confidence interval.

* Statistical significance is indicated when $p < 0.05$ or 95% CI does not include 1.0.

[†] Referent group is heterosexual students.

[§] Did not go to school because they felt unsafe at school or on their way to or from school on at least 1 day during the 30 days before the survey.

[¶] Significantly different from heterosexual students.

** Threatened or injured with a weapon on school property during the 12 months before the survey.

^{††} Ever physically forced to have sexual intercourse when they did not want to.

^{§§} Significantly different from bisexual students.

^{¶¶} Significantly different from students who are not sure of their sexual identity.

^{***} Being forced to do sexual things they did not want to do by someone they were dating or going out with one or more times during the 12 months before the survey, among students who dated or went out with someone during the 12 months before the survey.

^{†††} Being physically hurt on purpose by someone they were dating or going out with one or more times during the 12 months before the survey, among students who date or went out with someone during the 12 months before the survey.

^{§§§} Bullied on school property during the 12 months before the survey.

^{¶¶¶} Bullied through texting, Instagram, Facebook, or other social media during the 12 months before the survey.

Lesbians reported a higher prevalence of using cigarettes (APR = 1.8) and marijuana (1.5) than did heterosexual females (Table 2). Compared with heterosexual females, bisexual females had a higher prevalence of use of cigarettes (1.8), alcohol (1.2), and marijuana (1.6); females who were not sure of their sexual identity reported a higher prevalence of using cigarettes (1.2), but a lower prevalence of using alcohol (0.9). The prevalences of reported use of cocaine, heroin, methamphetamines, ecstasy, and inhalants were higher among lesbian, bisexual, and females not sure of their sexual identity than among heterosexual females. Among male students, compared with those identifying as heterosexual, bisexuals had a higher prevalence of reported cigarette use (1.4), and those identifying as not sure had a lower reported prevalence of marijuana use (0.8). The reported prevalences of using cocaine, heroin, methamphetamines, ecstasy, and inhalants were higher among

gay and bisexual males and males who were not sure of their sexual identity than among heterosexual males. Prevalence estimates and significant effect modifications by sex indicate that associations between sexual minority status and cigarettes/marijuana use were stronger for females than for males.

All sexual minority females reported a higher prevalence of feeling sad or hopeless, considering attempting suicide, making a suicide plan, and attempting suicide than did heterosexual females (Table 3). The reported prevalence of a suicide attempt treated by a doctor or nurse was higher among lesbian (APR = 3.7) and bisexual females (3.7) than among heterosexual females. Compared with heterosexual males, all sexual minority males had higher prevalences of all five indicators of suicide risk. Prevalence estimates and significant effect modification indicate that the association between sexual identity and attempted suicide was stronger for males than for females.

TABLE 2. Unadjusted prevalence and adjusted prevalence ratios of lifetime substance use behaviors by sex and sexual identity — National Youth Risk Behavior Survey, United States, 2015–2017*

Substance use behavior/Sex	Heterosexual [†]	Lesbian/Gay		Bisexual		Not sure		Interaction by sex p-value
	% (95% CI)	% (95% CI)	APR (95% CI)	% (95% CI)	APR (95% CI)	% (95% CI)	APR (95% CI)	
Cigarettes								
Females	26.4 (23.7–29.4)	46.8 (39.3–54.3)	1.8 ^{§,¶} (1.5–2.1)	47.1 (42.6–51.7)	1.8 ^{§,¶} (1.6–2.0)	32.1 (26.4–38.5)	1.2 [§] (1.0–1.5)	0.007
Males	32.1 (29.9–34.4)	38.4 (29.9–47.8)	1.2 (1.0–1.5)	43.8 (37.4–50.4)	1.4 ^{§,¶} (1.2–1.6)	32.4 (26.3–39.1)	1.0 (0.8–1.2)	
Alcohol								
Females	63.8 (61.4–66.2)	68.1 (61.3–74.2)	1.1 ^{§,¶} (1.0–1.2)	78.1 (74.3–81.4)	1.2 ^{§,¶} (1.2–1.3)	54.9 (48.4–61.2)	0.9 [§] (0.8–1.0)	0.064
Males	60.2 (58.6–61.7)	64.2 (55.2–72.3)	1.1 (0.9–1.2)	66.5 (58.3–73.9)	1.1 [¶] (1.0–1.2)	55.1 (47.8–62.1)	0.9 (0.78–1.1)	
Marijuana								
Females	34.5 (32.1–37.0)	55.1 (47.3–62.7)	1.5 ^{§,¶} (1.3–1.8)	55.6 (51.9–59.2)	1.6 ^{§,¶} (1.5–1.8)	36.0 (30.2–42.2)	1.1 (0.9–1.3)	0.000
Males	38.1 (35.9–40.3)	43.5 (34.2–53.3)	1.1 [¶] (0.9–1.4)	37.4 (29.6–45.9)	1.0 (0.8–1.2)	31.3 (25.8–37.3)	0.8 [§] (0.7–1.0)	
Cocaine								
Females	3.0 (2.6–3.5)	6.4 (4.2–9.7)	2.2 [§] (1.4–3.4)	6.3 (5.0–7.9)	2.2 [§] (1.6–2.9)	6.5 (4.2–9.8)	2.2 [§] (1.4–3.4)	0.069
Males	5.2 (4.5–5.9)	21.4 (13.7–31.8)	3.6 [§] (2.5–5.0)	12.1 (7.5–18.9)	2.5 [§] (1.5–4.1)	17.6 (12.7–23.7)	3.0 [§] (2.1–4.4)	
Heroin								
Females	0.7 (0.5–1.0)	4.3 (2.4–7.6)	5.8 [§] (3.0–11.1)	2.2 (1.5–3.2)	3.0 [§] (1.9–4.8)	2.8 (1.4–5.8)	3.9 [§] (1.8–8.6)	0.169
Males	1.7 (1.3–2.1)	13.4 (7.2–23.7)	6.1 [§] (4.1–9.3)	8.1 (4.4–14.3)	5.2 [§] (3.0–9.1)	14.4 (10.3–19.7)	8.1 [§] (5.5–11.9)	
Methamphetamines								
Females	1.2 (1.0–1.6)	6.4 (3.4–11.9)	4.9 [§] (2.5–9.7)	4.4 (3.2–5.9)	3.6 [§] (2.4–5.3)	3.8 (2.3–6.2)	2.8 [§] (1.6–5.1)	0.334
Males	2.5 (2.1–3.0)	18.7 (10.7–30.6)	6.2 [§] (4.0–9.6)	9.4 (5.6–15.2)	3.9 [§] (2.3–6.6)	14.4 (9.7–20.9)	5.2 [§] (3.3–8.1)	
Ecstasy								
Females	2.7 (2.2–3.3)	4.8 (3.0–7.4)	1.8 [§] (1.1–3.0)	7.3 (5.9–9.0)	2.8 [§] (2.1–3.7)	5.4 (3.5–8.4)	2.0 [§] (1.2–3.3)	0.088
Males	4.6 (4.0–5.3)	15.9 (10.7–23.0)	3.2 [§] (2.2–4.7)	15.4 (10.6–21.8)	3.6 [§] (2.4–5.3)	16.4 (11.3–23.3)	3.5 [§] (2.3–5.3)	
Inhalants								
Females	5.2 (4.6–6.0)	11.3 (7.3–16.9)	2.3 [§] (1.4–3.5)	12.5 (10.3–15.1)	2.4 [§] (1.9–2.9)	13.8 (9.6–19.4)	2.6 [§] (1.8–3.7)	0.176
Males	5.5 (5.0–6.1)	20.0 (11.7–32.1)	3.2 [§] (2.1–5.0)	14.9 (10.5–20.7)	2.8 [§] (1.9–4.0)	22.2 (17.1–28.2)	3.9 [§] (2.9–5.1)	

Abbreviations: APR = adjusted (for race/ethnicity and grade) prevalence ratio; CI = confidence interval.

* Statistical significance is indicated when $p < 0.05$ or 95% CI does not include 1.0.

[†] Referent group is heterosexual students.

[§] Significantly different from heterosexual students.

[¶] Significantly different from students who are not sure of their sexual identity.

** Significantly different from bisexual students.

Discussion

These findings demonstrate that sexual minority youths generally experience disparities in health outcomes attributed to minority stress when compared with heterosexual youths. Furthermore, some critical differences exist among sexual minority youths.

Bisexual female high school students experienced more disparities in victimization than did females who identified as heterosexual, lesbian, or not sure of their sexual identity. This finding differs from research indicating that lesbian and bisexual females experience similar rates of peer victimization (5), but supports study findings that bisexual female adults experience heightened risk for sexual violence (6). Among males, all sexual minority students were more likely than were heterosexual students to have experienced violence victimization. Associations between sexual minority status and victimization were stronger among males than among females, consistent with previous findings on gender differences among sexual minority youths (5). Violence prevention programs might need to consider the unique social stressors faced by bisexual females and sexual minority males.

Lesbian and bisexual female students reported higher levels of use of alcohol, cigarettes, and marijuana than did heterosexuals, whereas few differences across sexual identity for these substances were found among male students. These results are consistent with findings from previous research suggesting that sexual minority females are at increased risk for using alcohol, cigarettes, and marijuana (7). All sexual minority students were more likely than were heterosexual students to engage in high-risk substance use (i.e., less prevalent substances with a high risk of adverse outcomes), suggesting a need for increased primary and secondary prevention of high-risk substance use for sexual minority youths.

Suicide risk was higher among sexual minority students, regardless of sex or sexual identity, than among heterosexual students, which is consistent with the broader literature on suicide risk and sexual minority youths (8). The ubiquity of elevated APRs for suicidal thoughts and behaviors among sexual minority youths reinforces the important need for suicide prevention programming that is relevant and efficacious for this group.

The findings in this report are subject to at least five limitations. First, data are cross-sectional and indicate only association, not causation. Second, whether “not sure” students

TABLE 3. Unadjusted prevalence and adjusted prevalence ratios of suicide risk behaviors by sex and sexual identity — National Youth Risk Behavior Survey, United States, 2015–2017

Suicide risk behaviors/Sex	Heterosexual*		Lesbian/Gay		Bisexual		Not sure		Interaction by sex p-value
	% (95% CI)	% (95% CI)	APR (95% CI)	% (95% CI)	APR (95% CI)	% (95% CI)	APR (95% CI)		
Sad/Hopeless†									
Females	36.1 (34.0–38.3)	59.4 (51.2–67.2)	1.7 ^{§,¶,***} (1.5–1.9)	69.3 (66.1–72.4)	1.9 ^{§,¶} (1.8–2.1)	51.0 (44.7–57.2)	1.4 [§] (1.3–1.6)	0.385	
Males	19.0 (17.9–20.2)	39.4 (32.3–47.0)	2.0 [§] (1.7–2.5)	49.1 (41.7–56.5)	2.6 ^{§,¶} (2.2–3.1)	38.3 (32.2–44.8)	2.0 [§] (1.6–2.4)		
Considered attempting suicide††									
Females	18.3 (17.2–19.5)	45.8 (37.7–54.2)	2.6 ^{§,¶} (2.1–3.1)	49.7 (46.0–53.3)	2.7 ^{§,¶} (2.5–3.0)	34.5 (28.6–40.8)	1.9 [§] (1.6–2.2)	0.242	
Males	10.4 (9.7–11.2)	28.0 (20.7–36.8)	2.7 ^{§,¶} (2.0–3.7)	40.6 (32.9–48.8)	4.0 ^{§,¶} (3.3–4.8)	27.1 (22.1–32.7)	2.6 [§] (2.1–3.2)		
Made a suicide plan^{§§}									
Females	14.3 (13.1–15.6)	38.0 (30.6–46.0)	2.7 ^{§,¶} (2.2–3.3)	42.0 (38.5–45.6)	2.9 ^{§,¶} (2.6–3.3)	27.9 (23.2–33.2)	1.9 [§] (1.6–2.3)	0.330	
Males	8.4 (7.7–9.3)	23.3 (17.8–29.7)	2.6 ^{§,¶} (2.0–3.5)	31.7 (24.7–39.6)	3.9 ^{§,¶} (3.0–5.0)	22.6 (17.8–28.3)	2.7 [§] (2.1–3.5)		
Attempted suicide^{¶¶}									
Females	7.7 (6.7–8.9)	24.3 (17.4–32.8)	3.2 ^{§,¶} (2.3–4.5)	28.3 (24.7–32.3)	3.6 ^{§,¶} (3.0–4.2)	12.4 (9.5–16.1)	1.5 [§] (1.1–2.1)	0.012	
Males	4.3 (3.8–4.9)	13.1 (7.7–21.2)	3.0 ^{§,¶,††} (1.9–4.9)	23.2 (17.0–30.7)	5.5 ^{§,¶} (4.1–7.5)	14.9 (10.9–20.0)	3.3 [§] (2.3–4.8)		
Suicide attempt treated by a doctor or nurse^{***}									
Females	2.4 (1.9–3.0)	8.7 (4.6–15.7)	3.7 [§] (1.9–7.1)	9.2 (7.4–11.4)	3.7 ^{§,¶} (2.8–5.0)	4.2 (2.4–7.0)	1.6 (0.9–2.9)	0.545	
Males	1.4 (1.1–1.8)	4.9 (2.4–9.6)	3.7 [§] (1.8–7.5)	5.6 (2.7–11.1)	4.2 [§] (2.2–7.9)	5.3 (2.7–10.0)	3.5 [§] (1.6–7.8)		

Abbreviations: APR = adjusted (for race/ethnicity and grade) prevalence ratio; CI = confidence interval.

* Referent group.

† Felt sad or hopeless almost every day for ≥2 weeks in a row so that they stopped doing some usual activities during the 12 months before the survey.

§ Significantly different from heterosexual students.

¶ Significantly different from students who are not sure of their sexual identity.

** Significantly different from bisexual students.

†† Seriously considered attempting suicide during the 12 months before the survey.

§§ Made a plan about how they would attempt suicide during the 12 months before the survey.

¶¶ Attempted suicide one or more times during the 12 months before the survey.

*** Attempted suicide that resulted in an injury, poisoning, or overdose that had to be treated by a doctor or nurse during the 12 months before the survey.

were not sure of their identity, the usefulness of the response options for their identity, or the meaning of the question itself is unclear. Third, YRBS responses are self-reported, and might be subject to reporting bias; although the extent of underreporting or overreporting cannot be determined, the survey questions generally demonstrate good test-retest reliability (4). Fourth, the national YRBS does not currently ask about gender identity, and thus the prevalence of these outcomes for transgender students cannot be assessed with these data. Finally, CDC collects YRBS data in schools; because students at highest risk for victimization, substance use, and suicide risk might have dropped out, these findings might underestimate the observed associations with sexual minority status (9).

These findings serve to highlight the variation in victimization, substance use, and suicide risk among high school students by sexual identity. Prevention efforts might benefit from acknowledging within-group differences among sexual minority youths, and by updating and tailoring intervention strategies accordingly (e.g., the recently released suite of CDC technical packages for violence prevention*). More work is needed to understand whether programs promoting safe and supportive environments in schools and communities or stable, nurturing relationships could be better designed to address the within-group differences among sexual minority youths based on their sexual identity

* <https://www.cdc.gov/violenceprevention/pub/technical-packages.html>.

Summary

What is already known about this topic?

Sexual minority youths are at increased risk for certain adverse health outcomes.

What is added by this report?

Analysis of 2015 and 2017 Youth Risk Behavior Survey data identified within-group differences in victimization, substance use, and suicide risk among sexual minority high school students. Compared with heterosexuals, females who are bisexual and males who are gay, bisexual, or not sure of sexual identity reported more victimization; lesbian and bisexual females reported more alcohol, cigarette, and marijuana use; and all sexual minorities reported elevated high-risk substance use and suicide risk behavior.

What are the implications for public health practice?

Consideration of the different experiences of victimization, substance use, and suicide risk by sexual identity might help to inform interventions.

(10). Finally, professional development for educators and health providers working with sexual minority youths might benefit from content clearly articulating the differences between lesbian/gay, bisexual, and not sure identities, particularly as they relate to victimization, substance use, and suicide risk (10). Future research is necessary to determine the ideal components of such programs.

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Update: Recommendations of the Advisory Committee on Immunization Practices for Use of Hepatitis A Vaccine for Postexposure Prophylaxis and for Preexposure Prophylaxis for International Travel

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Postexposure prophylaxis (PEP) with hepatitis A (HepA) vaccine or immune globulin (IG) effectively prevents infection with hepatitis A virus (HAV) when administered within 2 weeks of exposure. Preexposure prophylaxis against HAV infection through the administration of HepA vaccine or IG provides protection for unvaccinated persons traveling to or working in countries that have high or intermediate HAV endemicity. The Advisory Committee on Immunization Practices (ACIP) Hepatitis Vaccines Work Group conducted a systematic review of the evidence for administering vaccine for PEP to persons aged >40 years and reviewed the HepA vaccine efficacy and safety in infants and the benefits of protection against HAV before international travel. The February 21, 2018, ACIP recommendations update and supersede previous ACIP recommendations for HepA vaccine for PEP and for international travel. Current recommendations include that HepA vaccine should be administered to all persons aged ≥12 months for PEP. In addition to HepA vaccine, IG may be administered to persons aged >40 years depending on the provider's risk assessment. ACIP also recommended that HepA vaccine be administered to infants aged 6–11 months traveling outside the United States when protection against HAV is recommended. The travel-related dose for infants aged 6–11 months should not be counted toward the routine 2-dose series. The dosage of IG has been updated where applicable (0.1 mL/kg). HepA vaccine for PEP provides advantages over IG, including induction of active immunity, longer duration of protection, ease of administration, and greater acceptability and availability.

Introduction

Postexposure prophylaxis (PEP) with hepatitis A (HepA) vaccine or immune globulin (IG) effectively prevents infection with hepatitis A virus (HAV) when administered within 2 weeks of exposure (1,2). The efficacy of IG or vaccine when administered >2 weeks after exposure has not been established.

Previous ACIP* recommendations for PEP included HepA vaccine for persons aged 1–40 years and IG for persons outside this age range; if IG was not available for persons aged >40 years, HepA vaccine could be administered (1).

Preexposure prophylaxis against HAV infection through the administration of HepA vaccine or IG is also recommended for unvaccinated persons traveling to or working in countries that have high or intermediate HAV endemicity (3). Because HepA vaccine is not licensed for use in children aged <1 year, IG has historically been recommended for travelers in this age group; however, IG cannot be administered simultaneously with measles, mumps, and rubella (MMR) vaccine, which is also recommended for infants aged 6–11 months traveling internationally from the United States (4–6).

This report provides recommendations for PEP use of HepA vaccine and IG, and use of HepA vaccine and IG for preexposure protection for persons who will be traveling internationally, including infants aged 6–11 months. This report updates and supersedes previous ACIP recommendations for HepA vaccine for PEP and for international travel (1).

Methods

During November 2016–February 2018, the ACIP Hepatitis Work Group[†] held monthly conference calls to review and

* Recommendations for routine use of vaccines in children, adolescents, and adults are developed by the Advisory Committee on Immunization Practices (ACIP). ACIP is chartered as a federal advisory committee to provide expert external advice and guidance to the Director of CDC on use of vaccines and related agents for the control of vaccine-preventable diseases in the civilian U.S. population. Recommendations for routine use of vaccines in children and adolescents are harmonized to the greatest extent possible with recommendations made by the American Academy of Pediatrics (AAP), the American Academy of Family Physicians (AAFP), and the American College of Obstetricians and Gynecologists (ACOG). Recommendations for routine use of vaccines in adults are harmonized with the recommendations of AAFP, ACOG, and the American College of Physicians (ACP). ACIP recommendations approved by the CDC Director become agency guidelines on the date published in the *Morbidity and Mortality Weekly Report* (MMWR). <https://www.cdc.gov/vaccines/acip>.

[†] The ACIP Hepatitis Vaccines Work Group comprises professionals from academic medicine (family medicine, internal medicine, pediatrics, obstetrics, infectious disease, occupational health, and preventive medicine specialists), federal and state public health entities, and medical societies.

discuss relevant scientific evidence,[§] including the use of HepA vaccine and IG for PEP and the use of HepA vaccine for infants before some international travel. The ACIP Hepatitis Work Group evaluated the quality of evidence related to the benefits and harms of administering a dose of HepA vaccine for PEP for persons aged >40 years using the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) framework (<https://www.cdc.gov/vaccines/acip/recs/grade/table-refs.html>). Quality of evidence related to the benefits and harms of administering HepA vaccine for preexposure prophylaxis to infants aged 6–11 months who will be traveling internationally was not evaluated using the GRADE framework; instead, studies of HepA vaccine efficacy and safety in infants (7–9) and the benefits of protection against HAV before international travel were considered (3).

At the February 2018 ACIP meeting, the following proposed recommendations were presented to the committee: 1) HepA vaccines should be administered for PEP for all persons aged ≥12 months; in addition to HepA vaccine, IG may be administered to persons aged >40 years for PEP, depending on the provider's risk assessment; and 2) HepA vaccine should be administered to infants aged 6–11 months traveling outside the United States when protection against hepatitis A is recommended. After a period for public comment, the recommendations were approved unanimously by the voting ACIP members.[¶]

Summary of Key Findings

Prevention of HAV infection with HepA vaccine following exposure. A randomized, double-blind clinical trial of HepA vaccine in 1,090 HAV-susceptible persons aged 2–40 years who were contacts of persons with HAV infection suggested that performance of HepA vaccine administered <14 days after exposure approaches that of IG in healthy children and adults aged <40 years (1,10). Limited data are available comparing HepA vaccine and IG in healthy adults aged >40 years; available data indicate reduced response to HepA vaccine in older age groups compared with response in younger adults (11).

[§] In preparation for ACIP deliberation, the scientific literature was searched using PubMed and EMBASE databases for reports published from January 1, 1992, through January 7, 2017. Search terms included “hepatitis A vaccine” and “HAV vaccine” and excluded studies in nonhumans and articles on children and adolescents. To qualify as a candidate for inclusion in the review, a study had to include data within 2 weeks of the first dose of HepA vaccine. Studies were excluded if they reported data focused solely on children, did not provide information on ages of persons studied, did not include data on Havrix or Vaqta (the two single antigen HepA vaccines currently licensed in the United States), only included safety data or discussed vaccine introduction without providing new data on vaccine efficacy or seroprotection, or only reported data on persons with underlying health conditions.

[¶] 14 voted in favor, with none opposed, none abstained, and none recused.

GRADE quality of evidence summary for HepA vaccine for PEP in persons aged >40 years. The evidence assessing benefits and harms of administering a dose of HepA vaccine for PEP to prevent HAV infection in adults aged >40 years was determined to be GRADE evidence type 4 (i.e., evidence from clinical experience and observations, observational studies with important limitations, or randomized controlled trials with several major limitations) for benefits and type 3 (i.e., evidence from observational studies, or randomized controlled trials with notable limitations) for harms (<https://www.cdc.gov/vaccines/acip/recs/grade/table-refs.html>).

Prevention of HAV infection among infants aged 6–11 months who received HepA vaccine before travel. HepA vaccine was demonstrated to be safe and efficacious for infants as young as age 2 months (2,7–9), although vaccination of infants aged <12 months might result in a suboptimal immune response because of potential interference with passively acquired maternal antibody, which could decrease long-term immunity (7–9).

Rationale for Recommendations

Advantages of HepA vaccine for PEP. HepA vaccine for PEP provides numerous public health advantages compared with IG, including the induction of active immunity and longer duration of protection, ease of administration, and greater acceptability and availability (11). Previous recommendations favoring IG for adults aged >40 years were based on the premise that IG is more efficacious in this group; however, evidence of decreased IG potency (i.e., reduced titers of anti-HAV antibodies) (12) led to a recommendation for an increase in the IG dosage (0.1 mL/kg) for hepatitis A PEP in 2017, with a consequent increase in IG administration volume (6). In addition, when HAV exposure, and thus the need for PEP, is not clear (i.e., consumer of recalled food product or patron at a restaurant where a notification occurred), the benefit of IG compared with vaccine, which provides long-term protection, is less certain.

Before travel administration of HepA vaccine to infants aged 6–11 months. IG cannot be administered simultaneously with MMR vaccine because antibody-containing products such as IG can inhibit the immune response to measles and rubella vaccines for 3 months (4,6). However, because MMR vaccine is recommended for all infants aged 6–11 months traveling internationally from the United States and because measles in infancy is more severe than HAV infection in infancy, MMR vaccine should be administered preferentially to preexposure prophylaxis with IG for prevention of HAV

infection. Administration of HepA vaccine (indication for off-label use) and MMR vaccine to infants aged 6–11 months (7–9) provides protection against both HAV and measles and allows for simultaneous prophylactic administration (4,13).

Recommendations for Postexposure Prophylaxis Against HAV Infection

HepA vaccine should be administered to all persons aged ≥ 12 months for PEP. In addition to HepA vaccine, IG may be administered to persons aged >40 years, depending on the provider's risk assessment (Supplementary Text 1, <https://staging-stacks.cdc.gov/view/cdc/59777>). Recommendations for PEP have been updated to include HepA vaccine for all unvaccinated persons aged ≥ 12 months, regardless of risk group, and co-administration of IG when indicated (Table 1). The dosage of GamaSTAN S/D human IG for PEP (0.1 mL/kg) also has been updated (6). Persons who have recently been exposed to HAV and who have not received HepA vaccine previously should receive PEP as soon as possible, within 2 weeks of exposure (1).

Infants aged <12 months and persons for whom vaccine is contraindicated. Infants aged <12 months and persons for whom vaccine is contraindicated (persons who have had a life-threatening allergic reaction after a dose of HepA vaccine, or who have a severe allergy to any component of this vaccine) should receive IG (0.1 mL/kg) (6,14) instead of HepA vaccine, as soon as possible and within 2 weeks of exposure. MMR and varicella vaccines should not be administered sooner than 3 months after IG administration (4–6).

Immunocompetent persons aged ≥ 12 months. Persons aged ≥ 12 months who have been exposed to HAV within the past 14 days and have not previously completed the 2-dose HepA vaccine series should receive a single dose of HepA vaccine (Table 2) as soon as possible. In addition to HepA vaccine, IG (0.1 mL/kg) may be administered to persons aged >40 years depending on the providers' risk assessment (Supplementary Text 1, <https://staging-stacks.cdc.gov/view/cdc/59777>). For long-term immunity, the HepA vaccine series should be completed with a second dose at least 6 months after the first dose; however, the second dose is not necessary for PEP. A second dose should not be administered any sooner than 6 months after the first dose, regardless of HAV exposure risk.

Persons aged ≥ 12 months who are immunocompromised or have chronic liver disease. Persons who are immunocompromised or have chronic liver disease and who have been exposed to HAV within the past 14 days and have not previously completed the 2-dose HepA vaccination series should receive both IG (0.1 mL/kg) and HepA vaccine simultaneously in a different anatomic site (e.g., separate limbs) as soon as

TABLE 1. Recommendations for postexposure prophylaxis and preexposure protection, by age group and risk category

Indication/ Age group	Risk category/ Health status	Hepatitis A vaccine	Immune globulin
Postexposure prophylaxis			
<12 mos	Healthy	No	0.1 mL/kg*
12 mos–40 yrs	Healthy	1 dose [†]	None
>40 yrs	Healthy	1 dose [†]	0.1 mL/kg [§]
≥ 12 mos	Immunocompromised or chronic liver disease	1 dose [†]	0.1 mL/kg [¶]
≥ 12 mos	Vaccine contraindicated**	No	0.1 mL/kg
Preexposure protection^{††}			
<6 mos	Healthy	No	0.1–0.2 mL/kg ^{§§}
6–11 mos	Healthy	1 dose ^{¶¶}	None
12 mos–40 yrs	Healthy	1 dose ^{***}	None
>40 yrs	Healthy	1 dose ^{***}	0.1–0.2 mL/kg ^{§§,†††}
All ages	Immunocompromised or chronic liver disease	1 dose ^{***}	0.1–0.2 mL/kg ^{§§,†††}
>6 mos	Persons who elect not to receive vaccine or for whom vaccine is contraindicated	No	0.1–0.2 mL/kg ^{§§}

* Measles, mumps, and rubella vaccine should not be administered for at least 3 months after receipt of IG.

[†] A second dose is not required for postexposure prophylaxis; however, for long-term immunity, the hepatitis A vaccination series should be completed with a second dose at least 6 months after the first dose.

[§] The provider's risk assessment should determine the need for immune globulin administration. If the provider's risk assessment determines that both vaccine and immune globulin are warranted, HepA vaccine and immune globulin should be administered simultaneously at different anatomic sites

[¶] Vaccine and immune globulin should be administered simultaneously at different anatomic sites.

** Life-threatening allergic reaction to a previous dose of hepatitis A vaccine, or allergy to any vaccine component.

^{††} IG should be considered before travel for persons with special risk factors for either HAV infection or increased risk for complications in the event of exposure to HAV.

^{§§} 0.1 mL/kg for travel up to 1 month; 0.2 mL/kg for travel up to 2 months, 0.2 mL/kg every 2 months for travel of ≥ 2 months' duration.

^{¶¶} This dose should not be counted toward the routine 2-dose series, which should be initiated at age 12 months.

^{***} For persons not previously vaccinated with HepA vaccine, administer dose as soon as travel is considered, and complete series according to routine schedule.

^{†††} May be administered, based on providers' risk assessment.

possible after exposure (6,15–17) (Table 1). For long-term immunity, the HepA vaccination series should be completed with a second dose at least 6 months after the first dose; however, the second dose is not necessary for PEP. A second dose should not be administered any sooner than 6 months after the first dose, regardless of HAV exposure risk.

In addition to HepA vaccine, IG should be considered for postexposure prophylaxis for persons with special risk factors for either HAV infection or increased risk of complications in the event of an exposure to HAV (Table 3) (Supplementary Text 1, <https://staging-stacks.cdc.gov/view/cdc/59777>).

TABLE 2. Vaccines used to prevent hepatitis A virus (HAV) infection

Vaccine	Trade name (manufacturer)	Age group (yrs)	Dosage	Route	Schedule	Booster
Hepatitis A vaccine, inactivated	Havrix (GlaxoSmithKline)	1–18	0.5 mL (720 ELU)	IM	0, 6–12 mo	None
		≥19	1 mL (1,440 ELU)	IM	0, 6–12 mo	None
Hepatitis A vaccine, inactivated	Vaqta (Merck and Co.)	1–18	0.5 mL (25 U)	IM	0, 6–18 mo	None
		≥19	1 mL (50 U)	IM	0, 6–18 mo	None
Combined hepatitis A and B vaccine*	Twinrix (GlaxoSmithKline)	≥18 (primary)	1 mL (720 ELU HAV + 20 µg HBsAg)	IM	0, 1, 6 mo	None
		≥18 (accelerated)	1 mL (720 ELU HAV + 20 µg HBsAg)	IM	0, 7, 21–30 days	12 mo

Abbreviations: ELU = ELISA units of inactivated HAV; HBsAg = hepatitis B surface antigen; IM = intramuscular; U = units of HAV antigen.

* Combined hepatitis A and B vaccine (Twinrix) should not be used for postexposure prophylaxis.

TABLE 3. Categories of persons with increased risk for hepatitis A virus (HAV) infection or increased risk for complications in the event of exposure to HAV

Type of risk	Risk category	Examples
Increased risk for HAV infection	Close contacts of persons with HAV infection*	Household contacts Caretakers Sexual contacts
	Occupational risk	Persons working with nonhuman primates Persons working with HAV in a research laboratory
Increased risk for HAV-associated complications	Immunocompromised persons	Congenital or acquired immunodeficiency HIV infection Chronic renal failure/Undergoing dialysis Solid organ, bone marrow, or stem cell transplant recipients Persons with diseases requiring treatment with immunosuppressive drugs/biologics (e.g., tumor necrosis alpha inhibitors), long-term systemic corticosteroids, radiation therapy
	Chronic liver disease	Hepatitis B infection Hepatitis C infection Cirrhosis (any etiology) Fatty liver disease (hepatic steatosis) Alcoholic liver disease Autoimmune hepatitis Alanine aminotransferase (ALT) or aspartate amino transferase (AST) level more than twice the upper limit of normal or persistently elevated for 6 months

Abbreviation: HIV = human immunodeficiency virus.

* Excludes health care personnel using appropriate personal protective equipment.

Recommendations for Preexposure Protection Against HAV Infection for Travelers

Infants aged 6–11 months. HepA vaccine should be administered to infants aged 6–11 months traveling outside the United States when protection against HAV is recommended

Summary

What is already known about this topic?

Postexposure prophylaxis (PEP) with hepatitis A (HepA) vaccine or immune globulin (IG) prevents infection with hepatitis A virus when administered within 2 weeks of exposure. Measles, mumps, and rubella vaccine (MMR) is recommended for infants aged 6–11 months traveling outside the United States. IG cannot be administered simultaneously with MMR.

What is added by this report?

HepA vaccine is recommended for persons aged ≥12 months for PEP. Providers may also administer IG to adults aged >40 years, if indicated. The dosage of IG has been updated. Simultaneous administration of MMR and HepA vaccines is recommended for infants aged 6–11 months traveling internationally.

What are the implications for public health practice?

HepA vaccine for PEP provides advantages over IG, including induction of active immunity, longer duration of protection, ease of administration, and greater acceptability and availability.

(Table 1). The travel-related dose for infants aged 6–11 months should not be counted toward the routine 2-dose series. Therefore, the 2-dose HepA vaccination series should be initiated at age 12 months according to the routine, age-appropriate vaccination schedule.

Recommendations for preexposure protection against HAV for travelers aged <6 months and aged ≥12 months remain unchanged from previous recommendations (Table 1), except for the updated dosage of IG where applicable (Supplementary Text 2, <https://staging-stacks.cdc.gov/view/cdc/59778>) (6). For travel duration up to 1 month, 0.1 mL/kg of IG is recommended; for travel up to 2 months, the dose is 0.2 mL/kg, and for travel of ≥2 months, a 0.2 mL/kg dose should be repeated every 2 months for the duration of travel. All susceptible persons traveling to or working in countries that have high or intermediate HAV endemicity are at increased risk for infection and should be vaccinated or receive IG before departure (1,3).

Infants aged <6 months and travelers who elect not to receive vaccine or for whom vaccine is contraindicated.

Infants aged <6 months and travelers who elect not to receive vaccine or for whom vaccine is contraindicated should receive a single dose of IG before travel when protection against HAV is recommended. If travel is for ≥ 2 months' duration, a repeat dose of 0.2 mL/kg every 2 months should be administered (6).

Healthy persons aged 12 months–40 years. Healthy persons aged 12 months–40 years who are planning travel to an area with high or intermediate HAV endemicity and have not received HepA vaccine should receive a single dose of HepA vaccine as soon as travel is considered and should complete the 2-doses series according to the routine schedule.

Persons aged >40 years, immunocompromised persons, and persons with chronic liver disease. Persons with chronic liver disease as well as adults aged >40 years, immunocompromised persons, and persons with other chronic medical conditions planning to depart to an area with high or intermediate HAV endemicity in <2 weeks should receive the initial dose of HepA vaccine, and also simultaneously may be administered IG at a separate anatomic injection site (e.g., separate limbs) (Table 1) (6,15–17).

In addition to HepA vaccine, IG should be considered before travel for persons with special risk factors for either HAV infection or increased risk for complications in the event of an exposure to HAV (Table 3) (Supplementary Text 2, <https://staging-stacks.cdc.gov/view/cdc/59778>).

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Notes from the Field

Intestinal Colonization and Possible Iatrogenic Botulism in Mouse Bioassay–Negative Serum Specimens — Los Angeles County, California, November 2017

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Mouse bioassay (MBA) is the standard test for botulinum neurotoxin detection. Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) can be 10–100 times more sensitive than MBA (1), but is not yet widely available. This report describes two patients whose serum initially tested negative for botulinum neurotoxin by MBA and subsequently tested positive by MALDI-TOF MS. Los Angeles County Department of Public Health (LACDPH) routinely sends botulism test specimens to the CDC for MALDI-TOF MS while performing MBA in-house.

Case 1

In mid-November 2017, an elderly man with no serious medical problems was admitted to a hospital with dysarthria, dysphagia, and dyspnea of 3 days' duration. The day after admission (day 4 after symptom onset), he required endotracheal intubation and mechanical ventilation for respiratory failure. He developed ptosis, extraocular palsy, and quadriplegia. On day 16 (13 days into his hospitalization) LACDPH was consulted regarding suspected botulism and promptly released heptavalent botulinum antitoxin (HBAT). Clinicians subsequently decided not to administer HBAT because they suspected that refractory myasthenia gravis was the more likely diagnosis. A limited electromyography was nondiagnostic. Serum collected on day 16 was reported 8 days later to be negative for botulinum neurotoxin by MBA.

Interviews and home inspection did not identify high-risk foods or ill contacts, and the patient did not have any known risk factors for botulism, such as anatomic or functional bowel abnormalities or altered gastrointestinal flora associated with receipt of recent antimicrobials. The patient's neurologic status did not improve, and MALDI-TOF MS results, available on day 35 (19 days after serum collection), confirmed botulinum toxin type A. HBAT treatment was discussed after this result, and a clinical decision was made to hold off HBAT administration. Stool collected on day 48 was positive for botulinum neurotoxin type A by MBA on day 68. HBAT was subsequently administered within 24 hours of this result (day 69), but neurologic status remained unchanged. The patient died from ventilator-associated pneumonia on day 109.

The prolonged excretion of toxin-producing *Clostridium botulinum* is consistent with adult intestinal colonization botulism. Only one to two cases of adult intestinal colonization botulism are identified in the United States annually (2).

Case 2

In late November 2017, a middle-aged woman was evaluated at a hospital emergency department with dysphagia and dysphonia without respiratory distress or weakness. Five days earlier, she had received botulinum toxin A injections using Food and Drug Administration (FDA)-approved doses for cervical dystonia at office A under electromyography guidance. The emergency department physician consulted LACDPH regarding suspected iatrogenic botulism; LACDPH released antitoxin, which the patient received. The next day, her symptoms had improved, and she was discharged from the hospital. Serum collected the day of symptom onset, before HBAT administration, tested negative for botulinum neurotoxin by MBA (on day 5); however, MALDI-TOF MS results available on day 41 confirmed botulinum neurotoxin type A. Office A reported the adverse event* to FDA's MedWatch. Inspection of office A did not identify injection practice or dosing concerns. It is unknown whether the patient's dysphagia and dysphonia, which are localized effects expected from botulinum toxin administration, would have progressed to systemic signs and symptoms had HBAT not been administered.

Laboratory results for these patients highlight the reported increased sensitivity of MALDI-TOF MS compared with MBA, with potential implications for botulism surveillance. Because MALDI-TOF MS testing is not available locally at LACDPH, specimens must be shipped to CDC for testing, and results might not be available until 2–4 weeks later. Because paralysis can develop rapidly, HBAT should be administered empirically for most suspected botulism cases on the basis of clinical findings (3) to prevent progression or respiratory failure. Clinicians are required to report suspected cases of botulism immediately to local and state public health officials who are available around-the-clock for consultation, release of antitoxin, exposure identification, and guidance on laboratory testing and interpretation. For a patient with localized signs and symptoms after botulinum toxin injection, clinicians, in close consultation with public health officials, might consider monitoring the patient and administering HBAT if additional signs or symptoms of neurologic weakness suggesting systemic spread of toxin occur.

* Detection of botulinum toxin in serum after an FDA-approved botulinum toxin formulation administered at an approved dose for an approved indication.

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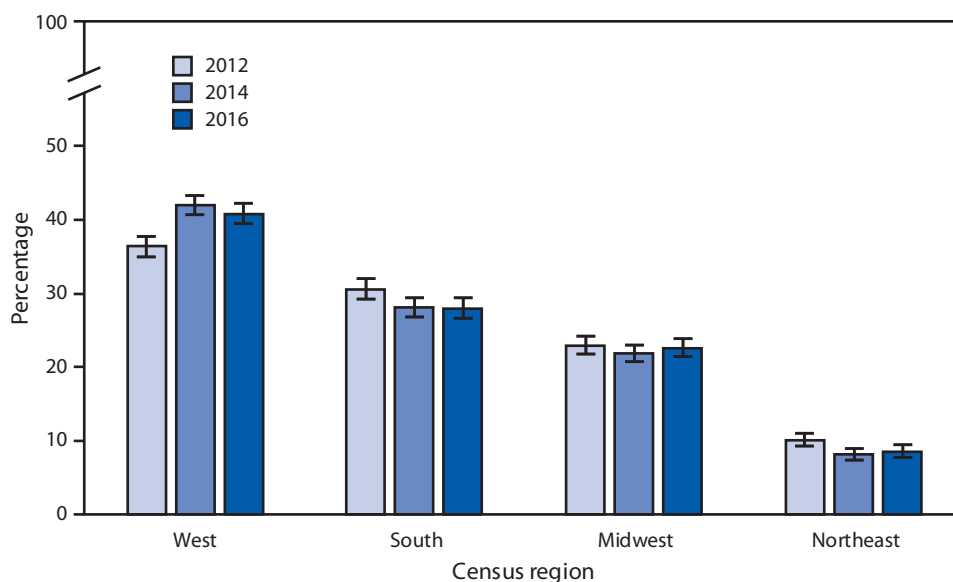
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QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Percentage* of Residential Care Communities,[†] by U.S. Census Region[§] — National Study of Long-Term Care Providers, 2012–2016



* With 95% confidence intervals indicated with error bars.

[†] Residential care communities include those that were state-regulated; had four or more beds; and provided room and board with at least two meals a day, around-the-clock on-site supervision, and help with personal care, such as bathing and dressing or health-related services such as medication management. Residential care communities licensed to exclusively serve the mentally ill or the intellectually or developmentally disabled populations were excluded.

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

During 2012–2016, the percentage of residential care communities located in the West increased from 36.4% to 40.8%. Throughout the period, a higher percentage of residential care communities were located in the West compared with other regions. The percentage of residential care communities declined from 30.6% in 2012 to 28% in 2016 in the South and from 10.1% to 8.6% in the Northeast. In the Midwest, the percentage was 22.9% in 2012 and 22.6% in 2016.

Source: National Study of Long-Term Care Providers, 2012–2016 data. <https://www.cdc.gov/nchs/nsltcp/index.htm>.

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