

Fatal Abusive Head Trauma Among Children Aged <5 Years — United States, 1999–2014

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In the United States, abusive head trauma (AHT) is one of the leading causes of maltreatment fatalities among infants and children, accounting for approximately one third of these deaths (1). Monitoring trends in AHT and evaluating prevention strategies have historically been difficult because of differences in AHT definitions used in research and surveillance. CDC's case definition for AHT and data from the National Vital Statistics System were used to examine the trends in fatal AHT during 1999–2014 using Joinpoint trend analysis software. During this period, AHT resulted in nearly 2,250 deaths among U.S. resident children aged <5 years. Whereas rates were relatively stable during 1999–2009, there was a statistically significant average annual decline of 13.0% in fatal AHT rates during 2009–2014. The fatal AHT rates in 2013 and 2014 (0.41 and 0.43 per 100,000 children aged <5 years, respectively) were the lowest in the 16-year study period. Although this decline in AHT deaths is encouraging, more can be done to prevent AHT, including family-based interventions and policies that create safe, stable, nurturing relationships and environments for children.

Comprehensive mortality data from the National Vital Statistics System (2) were used to identify fatal AHT* using the CDC case definition (3), and more broadly, to identify fatal assault-related traumatic brain injury (TBI)[†] among U.S. resident children aged <5 years during 1999–2014.

* Fatal abusive head trauma is defined as a death caused by an injury to the skull or intracranial contents of an infant or child aged <5 years attributable to inflicted blunt impact and/or violent shaking, and excludes deaths from injuries resulting from neglectful supervision and deaths from gunshot or stab wounds and penetrating trauma. Definite/presumptive fatal AHT cases have an external cause of injury code indicating assault or maltreatment. Probable fatal AHT cases have an undetermined external cause of injury code.

[†] Fatal traumatic brain injury is defined as a death caused by a bump, blow or jolt to the head, or by a penetrating injury, that disrupts normal brain function, and includes intentional gunshot wounds and stab wounds. These deaths can be classified as assault-related or nonassault-related.

Cases were identified based on the *International Classification of Diseases, 10th revision* (ICD-10) external cause/intent and nature-of-injury (body region and type of injury) codes, in accordance with established case definitions for AHT and TBI (3,4) (Figure). Only TBI (4) cases with an underlying cause consistent with assault (i.e., death record indicates assault as the intent of injury) were included in this analysis (Figure). Fatal assault-related TBI cases were then further classified by injury codes. Injury codes indicating blunt impact or violent shaking were classified as AHT, while injury codes indicating neglectful supervision, gunshot or stab wounds, and penetrating trauma were classified as assault-related TBI without AHT. Fatal AHT cases were further classified as definite or presumptive if the external cause of injury codes indicated assault or

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maltreatment, or probable if the external cause of injury codes were listed as undetermined. These assault-related TBI cases were further classified according to whether or not AHT was simultaneously indicated, to examine whether the reported decline in AHT deaths was offset by an increase in deaths identified as assault-related TBI exclusive of AHT, which might suggest that the decline in AHT deaths could have resulted from a change in coding of cases from AHT to assault-related TBI exclusive of AHT.

Both definite or presumptive and probable fatal AHT cases were included in the trend analysis. To examine whether or not cases that would have been coded as AHT were later being coded as assault-related TBI exclusive of AHT, death records that included an underlying cause code indicating assault and any nature-of-injury code indicating TBI were classified as assault-related TBI. Death records that did not list an underlying cause that broadly indicated injury were excluded from the analysis.

Yearly incidence rates were calculated using annual case counts and U.S. Census Bureau population estimates for children aged <5 years. To evaluate an apparent downward trend in annual rates of fatal AHT during the latter part of the analysis period (2009–2014), a negative binomial rate regression model allowing for an arbitrary shift in trend was fit to the data. The modeling process followed the general framework to test for significant changes in trend employed in the National Cancer Institute Joinpoint Regression Program (5), extended to compensate for potential overdispersion in the annual case

counts. The method allows for the description of changing trends over successive segments of time, and the increase or decrease within each time segment.

During 1999–2014, a total of 2,018 (90%) of 2,247 AHT deaths were classified as definite or presumptive, ranging from a high of 97% in 2001 to 81% in 2013. Nearly all definite or presumptive AHT deaths were simultaneously identified as assault-related TBI deaths (four deaths involving maltreatment, one each in 2003, 2004, 2005, and 2010, were not classified as assault-related). No probable AHT deaths were identified as assault-related TBI deaths (because the “probable” component of the AHT case definition excludes assault). During 1999–2009, annual rates of fatal AHT ranged from 0.68 per 100,000 children aged <5 years in 2001 to 0.88 per 100,000 in 2000 and 2009 (Table), with a modeled trend indicating a nonsignificant average annual increase of 0.04% ($p = 0.96$). During 2009–2014, annual rates of fatal AHT declined, with a modeled trend indicating a statistically significant average annual decrease of 13.0% ($p < 0.01$). Notably, the decline in deaths identified as AHT during this later period was not offset by an increase in deaths identified as assault-related TBI exclusive of AHT (Table), suggesting a real decline in AHT. During 2008–2014, the annual rate of fatal assault-related TBI (total) declined 28% from 2.25 to 1.62 per 100,000 children aged <5 years; from 2009 to 2014, this decline was almost entirely because of the decline in fatal AHT, as the rate of fatal assault-related TBI without AHT remained relatively stable during this period.

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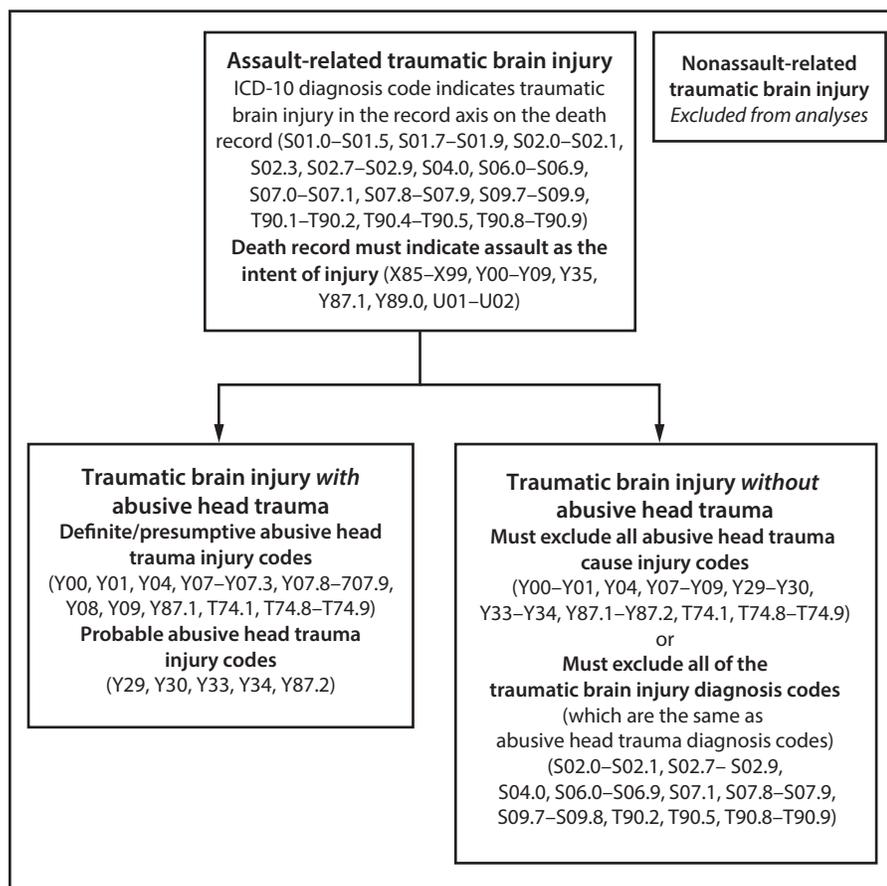
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FIGURE. Classification of fatal assault-related traumatic brain injury* with and without abusive head trauma† among children aged <5 years — United States, 1999–2014



Abbreviation: ICD = *International Classification of Diseases*.

* Fatal traumatic brain injury is defined as a death caused by a bump, blow, or jolt to the head, or by a penetrating injury that disrupts normal brain function, and includes intentional gunshot wounds and stab wounds. These deaths can be classified as assault-related or nonassault-related.

† Fatal abusive head trauma is defined as a death caused by an injury to the skull or intracranial contents of an infant or child aged <5 years attributable to inflicted blunt impact and/or violent shaking, and excludes deaths from injuries resulting from neglectful supervision and deaths from gunshot or stab wounds and penetrating trauma.

Discussion

Based on CDC’s recommended definition for AHT for public health surveillance (3), the rates of fatal AHT remained relatively stable during 1999–2009, followed by a significant decline during 2009–2014. The fatal AHT rates in 2013 and 2014 (0.41 and 0.43 per 100,000 children aged <5 years, respectively) were the lowest rates reported during the 16-year study period. This is the first documentation of a decline in AHT rates after 2009. These encouraging results are consistent with downward trends in other indices of child maltreatment and data systems, such as the recent analysis by the Children’s Bureau of the National Data Archive on Child Abuse and Neglect, which found that the number of children experiencing maltreatment decreased 3.8 percent during 2009–2013 (6).

Examining both definite or presumptive and probable fatal AHT cases illustrates that although definite or presumptive cases declined, probable cases did not increase, suggesting that the observed decline in definite or presumptive cases does not represent a change in case classification. In addition, data on fatal assault-related TBI (with and exclusive of AHT) similarly illustrate that classification of cases did not change over time from AHT to assault-related TBI exclusive of AHT. Sensitivity of clinical ascertainment of signs and symptoms associated with AHT might have systematically decreased or the coding of death data might have systematically changed over time. However, the nearly consistent annual number of injury-related death records listing an AHT-related cause code and constant annual rates of assault-related TBI exclusive of AHT during 2009–2014 suggest that such systematic changes are unlikely.

The findings in this report are subject to at least two limitations. First, the mortality data are based on coding of death certificates, which could result in undercounting AHT cases. However, any recent potential undercounting might be offset by enactment of the Child and Family Services Improvement and Innovation Act in 2011, which requires states to describe the data sources used to compile information on deaths attributable to child abuse or neglect. Because of this law, many states reported increased counts of child fatalities caused by abuse or neglect, and implemented child death reviews or expanded their scope. By 2012, all 50 states, the District of Columbia, and Guam had implemented child death reviews. Child death reviews have been identified as a key source of information for case identification and embody a process in which teams representing multiple disciplines, including medical examiners and juvenile justice experts, meet to share and discuss case information on child deaths to understand how and why they occur and how they might be prevented.[§] Second, this analysis cannot definitively determine the reasons for the decline in fatal AHT.

[§] <https://www.childdeathreview.org>.

TABLE. Annual rates of fatal AHT and fatal assault-related TBI per 100,000 children aged <5 years — United States, 1999–2014

Year	Fatal AHT rates (No. deaths)			Fatal assault-related TBI rates (No. deaths)		
	Definite or presumptive*	Probable†	Total	TBI and AHT	TBI without AHT	Total
1999	0.75 (143)	§ (11)	0.80 (154)	0.75 (143)	0.84 (161)	1.59 (304)
2000	0.83 (159)	§ (9)	0.88 (168)	0.83 (159)	1.09 (209)	1.92 (368)
2001	0.66 (128)	§ (4)	0.68 (132)	0.66 (128)	1.28 (247)	1.94 (375)
2002	0.77 (149)	§ (7)	0.80 (156)	0.77 (149)	1.14 (221)	1.90 (370)
2003	0.79 (154)	§ (10)	0.84 (164)	0.78 (153)	1.24 (242)	2.02 (395)
2004	0.62 (122)	§ (16)	0.70 (138)	0.61 (121)	1.21 (239)	1.82 (360)
2005	0.67 (134)	0.11 (22)	0.78 (156)	0.67 (133)	1.07 (213)	1.74 (346)
2006	0.70 (140)	§ (14)	0.77 (154)	0.70 (140)	1.33 (265)	2.03 (405)
2007	0.74 (149)	§ (19)	0.83 (168)	0.74 (149)	1.26 (253)	2.00 (402)
2008	0.65 (132)	0.12 (25)	0.77 (157)	0.65 (132)	1.60 (325)	2.25 (457)
2009	0.77 (155)	0.12 (24)	0.88 (179)	0.77 (155)	1.32 (267)	2.08 (422)
2010	0.52 (106)	§ (13)	0.59 (119)	0.52 (105)	1.25 (252)	1.77 (357)
2011	0.59 (119)	§ (11)	0.65 (130)	0.59 (119)	1.28 (258)	1.87 (377)
2012	0.44 (87)	§ (18)	0.53 (105)	0.44 (87)	1.27 (254)	1.71 (341)
2013	0.33 (66)	§ (15)	0.41 (81)	0.33 (66)	1.30 (258)	1.63 (324)
2014	0.38 (75)	§ (11)	0.43 (86)	0.38 (75)	1.24 (247)	1.62 (322)

Abbreviations: AHT = abusive head trauma; TBI = traumatic brain injury.

* Definite or presumptive fatal AHT cases are classified as death caused by an injury to the skull of an infant or child aged <5 years attributable to inflicted blunt impact and/or violent shaking, with an external cause of injury code indicating assault or maltreatment.

† Probable fatal AHT cases are classified as death caused by an injury to the skull of an infant or child aged <5 years attributable to inflicted blunt impact and/or violent shaking with an undetermined external cause of injury code.

§ Incidence rates based on counts <20 are not considered statistically stable and are not presented.

Fatal abusive head trauma, like all forms of child maltreatment, is preventable. An important step in the prevention of AHT and child maltreatment is the ongoing, systematic collection of data that help guide and monitor prevention approaches. Using CDC's uniform definitions of AHT and child maltreatment are important steps in strengthening surveillance. Data collected from surveillance systems, in combination with information on the implementation and results of interventions and policies, can help shape continuing public health efforts to prevent AHT.

Although the decrease in fatal AHT during 2009–2014 is encouraging, additional efforts are needed to prevent AHT. Prevention of child maltreatment requires understanding and addressing behavioral and environmental characteristics that increase and reduce the risk for child maltreatment. There is growing evidence that child maltreatment prevention strategies, such as those that change interactions, including those between parents and children, parents and other caregivers, and parents and health care providers are effective interventions (7). CDC's *Essentials for Parenting Toddlers and Preschoolers* (<http://www.cdc.gov/parents/essentials/index.html>) is an evidence-informed online resource based on decades of research about effectively promoting positive parenting and preventing child maltreatment, using various approaches, including videos and interactive practice exercises, to help caregivers build healthy relationships with their children aged >3 years. Community-level strategies offer additional critical components of preventing child maltreatment by modifying social and economic factors that put infants and young children at

risk for violence. Promising community-level strategies include strengthening economic supports for families and improving family-friendly work policies, such as the availability of paid parental leave (8). Societal level strategies aim to shift cultural norms surrounding parenting through public engagement and education campaigns to reframe thinking about child abuse. Communities can use CDC's *Preventing Child Abuse and Neglect: A Technical Package for Policy, Norm, and Programmatic Activities* (<http://www.cdc.gov/violenceprevention/pdf/can-prevention-technical-package.pdf>) and CDC's *Essentials for Childhood Framework: Steps to Create Safe, Stable, Nurturing Relationships and Environments for All Children* (<http://www.cdc.gov/violenceprevention/childmaltreatment/essentials.html>) to promote safe, stable, nurturing relationships and environments to prevent child maltreatment and assure that all children reach their full potential. This framework encourages communities to consider building and coordinating relationships between traditional and nontraditional partners (e.g., public health and business), collaboratively identify and implement child maltreatment prevention strategies, and monitor impact on morbidity and mortality.

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References

Summary

What is already known about this topic?

In the United States, abusive head trauma (AHT) is one of the leading causes of child maltreatment fatalities, accounting for approximately one third of these deaths. CDC developed a formal case definition for fatal AHT to facilitate consistent tracking over time and evaluation of interventions focused on prevention.

What is added by this report?

During 1999–2014, AHT resulted in nearly 2,250 deaths among U.S. resident children aged <5 years. During 2009–2014, annual rates of fatal AHT declined significantly, with an average annual decrease of 13.0%, and there was no evidence that cases were simply being classified differently during this time. This is the first report of a decline in AHT rates after 2009. The fatal AHT rates in 2013 and 2014 were 0.41 per 100,000 children aged <5 years and 0.43 per 100,000, respectively, the lowest rates in the 16-year study period.

What are the implications for public health practice?

Communities can use evidence-based approaches, such as family-based interventions, and CDC's *Essentials for Childhood Framework: Steps to Create Safe, Stable, Nurturing Relationships and Environments for All Children and Preventing Child Abuse and Neglect: A Technical Package for Policy, Norm and Programmatic Activities* to promote safe, stable, nurturing relationships and environments for children. Ongoing surveillance for AHT, in combination with information on the implementation and results of interventions and policies, can help shape prevention.

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Sodium in Store and Restaurant Food Environments — Guam, 2015

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Compared with the United States overall, Guam has higher mortality rates from cardiovascular disease and stroke (1). Excess sodium intake can increase blood pressure and risk for cardiovascular disease (2,3). To determine the availability and promotion of lower-sodium options in the nutrition environment, the Guam Department of Public Health and Social Services (DPHSS) conducted an assessment in September 2015 using previously validated tools adapted to include sodium measures. Stores (N = 114) and restaurants (N = 63) were randomly sampled by region (north, central, and south). Data from 100 stores and 62 restaurants were analyzed and weighted to account for the sampling design. Across the nine product types assessed, lower-sodium products were offered less frequently than regular-sodium products ($p < 0.001$) with <50% of stores offering lower-sodium canned vegetables, tuna, salad dressing, soy sauce, and hot dogs. Lower-sodium products were also less frequently offered in small stores than large (two or more cash registers) stores. Reduced-sodium soy sauce cost more than regular soy sauce ($p < 0.001$) in stores offering both options in the same size bottle. Few restaurants engaged in promotion practices such as posting sodium information (3%) or identifying lower-sodium entrées (1%). Improving the availability and promotion of lower-sodium foods in stores and restaurants could help support healthier eating in Guam.

In 2010, the Pacific Islands Health Officers Association declared a regional state of health emergency in the U.S.-Affiliated Pacific Islands because of an epidemic of noncommunicable diseases.* Globalization and increasing dependence on “cheap, energy-dense, high-fat foreign foods” have been cited as contributing factors in the increasing incidence of obesity and cardiovascular disease in the region (4,5). Approximately 162,000 persons live on the island of Guam, which is about 30 miles long and ranges from 4 miles to 12 miles wide.† Although some regional food manufacturing (e.g., bread) occurs, most food is imported by freighter, and much of the food supply is processed to improve shelf life (6). Recognizing that the nutrition environment could be contributing to chronic disease, Guam DPHSS and its partners created the *Non-Communicable Disease Strategic Plan 2014–2018* (7) to address several nutrition objectives, including reducing sodium

consumption. Guam DPHSS, with technical assistance from CDC, conducted this nutrition environment assessment to obtain data in support of the plan’s nutrition objectives.

To identify stores and restaurants for assessment, sampling frames were created using business listings from Guam DPHSS, Division of Environmental Health, Guam Supplemental Nutrition Assistance Program (SNAP), and the local phone book. Venues were classified by region and assigned identification numbers; a random-number generator determined the order of selection of venues in each region. Three large stores, where many residents obtained groceries, were deliberately (i.e., nonrandomly) placed into the sample. Included venues were open to the public with a permanent, nonmobile structure (i.e., no food trucks or other mobile venues). Stores were included if they sold three or more of five staple foods (milk, eggs, bread, produce, or meat/fish). Restaurants were included if they had a breakfast, lunch, or dinner menu with ≥ 5 entrées in any single category.

Stores were classified as small (one cash register) or large (two or more cash registers). Restaurants were classified as sit-down, fast-food, or fast-casual, with the second and third categories combined. Assessments used the previously validated Nutrition Environment Measures Surveys for stores and for restaurants,§ after modification to better reflect the local diet and to better assess sodium. In the style of the Nutrition Environment Measures Surveys for stores, foods were classified as “lower-sodium” with a corresponding “regular-sodium” counterpart to compare available consumer choices. Lower-sodium foods were classified according to Food and Drug Administration guidelines for sodium labeling,¶ with “no salt” foods containing no ingredient that is sodium chloride and <5mg sodium per labeled serving; “low-sodium” foods containing ≤ 140 mg sodium per serving; and “reduced-sodium” foods containing at least 25% less sodium per serving than an appropriate reference food. Lower-sodium foods assessed included no-salt-added canned vegetables; low-sodium tuna, chips, salad dressing, and bread; and reduced-sodium Spam (≤ 410 mg/serving sodium), soy sauce (≤ 690 mg/serving), hot dogs (≤ 305 mg/serving), and instant noodles (≤ 730 mg/serving). Soy sauce was an a priori selection for pricing assessment.

* <http://www.pihoa.org/initiatives/policy/ncds.php>.

† <https://www.cia.gov/library/publications/the-world-factbook/geos/gq.html>.

§ <http://www.med.upenn.edu/nems/about.shtml>.

¶ <http://www.fda.gov/Food/GuidanceRegulation/GuidanceDocumentsRegulatoryInformation/LabelingNutrition/ucm064911.htm>.

In all assessed venues, managers or owners were invited to participate in interviews regarding barriers to and supports for offering healthy food. However, some managers were not present and some declined to participate because of time or language barriers, leaving a convenience sample of approximately half of managers (50 store managers and 31 restaurant managers) who completed the sodium-related questionnaires.

Descriptive statistics were used to examine the availability and pricing of lower-sodium foods in stores, to assess sodium-related practices in restaurants, and to describe manager perceptions. Store and restaurant analyses were weighted to account for the survey design and disproportionate sampling across regions, but the convenience sample of managers was not weighted.

Lower-sodium alternatives typically were less available than regular-sodium product types (Table 1). Fewer than half of surveyed stores offered no-salt-added canned vegetables (27%), low-sodium tuna (10%) or salad dressing (8%), or reduced-sodium soy sauce (43%) or hot dogs (18%). Small stores were less likely than large stores to offer certain lower-sodium products ($p < 0.001$). In stores that sold both reduced-sodium soy sauce and regular soy sauce in the same size bottle ($n = 30$), reduced-sodium soy sauce was significantly more expensive ($\$6.35 \pm 0.49$) than regular-sodium soy sauce ($\$5.43 \pm 0.41$), normalized to median (20-ounce) volume ($p < 0.001$). Three fourths of store managers agreed that they should increase the availability of lower-sodium foods in their neighborhoods, but

78% did not believe that their stores offered a large selection of lower-sodium products (Table 2).

No surveyed restaurants promoted sodium reduction in displays or table tents, and few posted sodium information at the point-of-purchase (3%), identified lower-sodium menu items (1%), had salt-substitute shakers on tables (7%), or had reduced-sodium soy sauce bottles on tables (3%) (Table 3). Approximately one quarter of restaurants had saltshakers (27%) on tables, and sit-down restaurants were more likely to have saltshakers on tables than fast-food restaurants ($p < 0.001$). The majority of restaurant managers surveyed (65%) were aware of efforts on Guam to reduce sodium intake; all managers who reported awareness were supportive of these efforts (100%) (Table 2).

Discussion

This report is the first of its kind to examine sodium in the nutrition environment of Guam, including availability and promotion of lower-sodium foods in stores and restaurants. In surveyed stores, lower-sodium foods were less widely available than foods with higher sodium content, and small stores were significantly less likely than large stores to offer certain lower-sodium products. Few surveyed restaurants engaged in promotion practices such as labeling sodium content on menus, but many managers reported support for sodium reduction efforts.

These findings are consistent with those from a recent report from American Samoa, another U.S.-affiliated Pacific Island, which found that healthful foods were infrequently available in

TABLE 1. Percentage of all stores, large stores, and small stores offering at least one variety of lower-sodium food, and mean total number of products available per store, by sodium content and food type — Guam, 2015*

Type of food	Lower-sodium (%)			Regular-sodium (%)		
	All stores (N = 100)	Large stores (n = 34)	Small stores (n = 66)	All stores (N = 100)	Large stores (n = 34)	Small stores (n = 66)
Canned vegetables	26.6 [†]	41.2 [§]	18.3	95.5	100 [§]	92.9
Tuna	9.6 [†]	22.5 [§]	2.2	59.0	85.2 [§]	44.0
Chips	74.7 [†]	89.3 [§]	66.3	93.3	93.2	93.3
Salad dressing	8.2 [†]	22.5 [§]	0	51.0	83.6 [§]	32.5
Bread	69.2 [†]	86.5 [§]	59.4	90.7	100 [§]	85.5
Spam	64.4 [†]	74.4	58.8	85.4	89.3	83.2
Soy sauce	42.8 [†]	73.7 [§]	25.2	91.2	97.1 [§]	87.8
Hot dogs	18.4	35.9 [§]	8.4	79.4	81.3	78.4
Instant noodles	76.9	90.8 [§]	68.9	97.9	97.1	98.4
No. products per store, of nine stores assessed (mean ± SE)						
Total	3.9[¶] ± 0.18	5.4^{**} ± 0.29	3.1 ± 0.16	7.4 ± 0.14	8.3^{**} ± 0.20	7.0 ± 0.16

Abbreviation: SE = standard error.

* Weighted results are presented. Stores include both large (two or more cash registers) and small (one cash register) venues that are open to the public and sell at least three of the following: milk, eggs, produce, bread, or meat/fish. Lower-sodium refers to either no-salt-added (canned vegetables), low-sodium (tuna, chips, salad dressing, or bread with ≤ 140 mg/serving sodium), or reduced-sodium (Spam with ≤ 410 mg/serving, soy sauce with ≤ 690 , hot dogs with ≤ 305 mg, or instant noodles with ≤ 730 mg), depending on the product.

[†] Indicates significant difference ($p < 0.05$) between availability of lower-sodium versus regular-sodium options among all stores. Ideally, McNemar's test for paired proportions would be used for categorical comparisons within stores, but could not be performed with survey procedures. Survey-adjusted chi-square tests were used, which produced more conservative results than paired comparisons without survey adjustment.

[§] Indicates significant difference ($p < 0.05$) in availability of the same type of food (e.g., no-salt-added canned vegetables) in large versus small stores.

[¶] Indicates significant difference ($p < 0.05$) in total number of lower-sodium versus regular-sodium options among all stores.

** Indicates significant difference ($p < 0.05$) in total number of lower-sodium products or total number of regular-sodium products in large versus small stores.

TABLE 2. Store and restaurant manager perceptions and practices regarding lower-sodium foods — Guam, 2015*

Perceptions and practices	Stores (n = 50) (%)	Restaurants (n = 31) (%)
Store/restaurant manager-reported supports and barriers (agree/strongly agree)		
My customers look for low-salt or low-sodium foods	18.0	41.9
Customers ask for low-salt or low-sodium foods	34.0	51.6
I should increase the availability of low-salt or low-sodium foods in this neighborhood	78.0	71.0
Suppliers of low-salt or low-sodium foods are hard to find	36.0	29.0
Customers don't like low-sodium or low-salt foods, so they don't buy them	22.0	9.7
There is a large selection of low-salt or low-sodium products in my store	22.0	—
Restaurant manager perceptions and practices (answered "yes")		
Is sodium information for menu items posted for customers to see?	—	6.5
Are lower-sodium products specifically purchased for use by customers or by the chef?	—	54.8
Are lower-sodium products used routinely in cooking?	—	64.5
Are any vegetables routinely served without added sauce, sodium, or salad dressing?	—	54.8
Are you aware of the efforts on Guam to decrease people's salt intake?	—	64.5
If yes, are you supportive of the efforts to decrease people's salt intake?	—	100.0
If no, would you be supportive of efforts to decrease people's salt intake?	—	72.7

* Unweighted percentages are presented.

TABLE 3. Percentage of restaurants with sodium-related promotional practices, by type of restaurant — Guam, 2015*

Type of promotion	Restaurants (%)		
	Total all types (N = 62)	Sit-down (n = 43)	Fast-casual/Fast-food (n = 19)
Signs, table tents, or displays promote sodium reduction	0.0	0.0	0.0
Sodium information is available at point-of-purchase or on the menu	3.2	0.0	12.0
Menus have special icons/labels or separate sections identifying lower-sodium items	0.8	1.1	0.0
Salt-substitute shakers on tables	6.7	9.1	0.0
Saltshakers on tables	27.4	37.5 [†]	0.0
Reduced-sodium soy sauce bottles on tables	3.2	0.0	12.0
Regular soy sauce bottles on tables	28.7	34.9	12.0

* Weighted percentages are presented.

[†] Statistically significant difference ($p < 0.05$) between restaurant types.

stores and restaurants and often cost more and were promoted less (5). The present results provide context and data for the Guam *Non-Communicable Disease Strategic Plan 2014–2018*, which targets sodium reduction. For example, efforts in Guam have promoted removing saltshakers from tables in restaurants, and nearly three quarters of assessed restaurants did not have saltshakers on tables. While only a small percentage of sodium intake in the continental United States might come from a saltshaker (8), recent surveys in the Pacific islands indicate that the main sources of dietary sodium are salt and monosodium glutamate added during cooking and at the table (9). Other planned efforts in Guam include working with grocers, restaurants, and schools to increase availability of low-salt foods and meals (7). Lowering sodium content in the food supply is a recommended strategy for population-wide sodium reduction (9,10).

This report is subject to at least four limitations. First, only a small selection of foods could be assessed. Efforts were made to focus on local foods that are important contributors of sodium in the diet (10), and that had both lower-sodium and regular versions for comparison. Second, price comparisons related to

sodium content were restricted to soy sauce, commonly used instead of salt in cooking. Because most small stores did not have displayed prices, the number of price comparisons were limited to reduce the number of requests to store staff. Third, manager interviews were subject to selection and response bias. For example, non-English speaking managers might have been less likely to participate, although translators were used when possible. Selection bias was less likely in observational assessments of stores and restaurants; no stores and only two restaurants refused participation in the environmental assessment. Finally, sample weights could not fully account for the probability of selection of each venue, because of duplication in the sample frame. Selection probability depended on the number of times a venue was listed in the sample frame, but overall rates of duplication within the sample frame were unknown. Estimated weights were calculated with the assumption that the probability of duplication was equal for all stores in a region. Among stores (where duplication was most common), weighted and unweighted data yielded similar results.

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

Guam has higher rates of heart disease and stroke mortality than the U.S. average. The nutrition environment might contribute to the incidence of disease, as excess sodium intake is associated with hypertension and cardiovascular disease risk.

What is added by this report?

Lower-sodium food options are less commonly available than regular-sodium products in Guam, particularly in small stores, and few restaurants currently engage in supportive practices such as menu labeling for sodium content. Most interviewed restaurant and store managers reported a desire to improve access to lower-sodium foods in their neighborhoods.

What are the implications for public health practice?

There is room for improvement in the availability and promotion of lower-sodium foods in stores and restaurants in Guam. Restaurant and store managers might be willing to engage with the public health community in support of sodium reduction efforts, one objective of Guam's *Non-Communicable Disease Strategic Plan, 2014–2018*.

Efforts to reduce sodium in the food supply need to involve manufacturers, vendors, and the public (9). Most managers interviewed reported a desire to improve access to lower-sodium foods in their neighborhoods and might support future sodium-reduction efforts. Improving the availability, pricing, and promotion of lower-sodium foods in stores and restaurants could help to decrease sodium intake on Guam.

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Possible Zika Virus Infection Among Pregnant Women — United States and Territories, May 2016

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Zika virus is a cause of microcephaly and brain abnormalities (1), and it is the first known mosquito-borne infection to cause congenital anomalies in humans. The establishment of a comprehensive surveillance system to monitor pregnant women with Zika virus infection will provide data to further elucidate the full range of potential outcomes for fetuses and infants of mothers with asymptomatic and symptomatic Zika virus infection during pregnancy. In February 2016, Zika virus disease and congenital Zika virus infections became nationally notifiable conditions in the United States (2). Cases in pregnant women with laboratory evidence of Zika virus infection who have either 1) symptomatic infection or 2) asymptomatic infection with diagnosed complications of pregnancy can be reported as cases of Zika virus disease to ArboNET* (2), CDC's national arboviral diseases surveillance system. Under existing interim guidelines from the Council for State and Territorial Epidemiologists (CSTE), asymptomatic Zika virus infections in pregnant women who do not have known pregnancy complications are not reportable. ArboNET does not currently include pregnancy surveillance information (e.g., gestational age or pregnancy exposures) or pregnancy outcomes. To understand the full impact of infection on the fetus and neonate, other systems are needed for reporting and active monitoring of pregnant women with laboratory evidence of possible Zika virus infection during pregnancy. Thus, in collaboration with state, local, tribal, and territorial health departments, CDC established two surveillance systems to monitor pregnancies and congenital outcomes among women with laboratory evidence of Zika virus infection[†] in the United States and territories: 1) the U.S. Zika Pregnancy Registry (USZPR),[§] which monitors pregnant women residing in U.S. states and all U.S. territories except Puerto Rico, and 2) the Zika Active Pregnancy Surveillance System (ZAPSS),

which monitors pregnant women residing in Puerto Rico. As of May 12, 2016, the surveillance systems were monitoring 157 and 122 pregnant women with laboratory evidence of possible Zika virus infection from participating U.S. states and territories, respectively. Tracking and monitoring clinical presentation of Zika virus infection, all prenatal testing, and adverse consequences of Zika virus infection during pregnancy are critical to better characterize the risk for congenital infection, the performance of prenatal diagnostic testing, and the spectrum of adverse congenital outcomes. These data will improve clinical guidance, inform counseling messages for pregnant women, and facilitate planning for clinical and public health services for affected families.

Zika virus disease and congenital Zika virus infection are defined by the interim CSTE case definition and include confirmed and probable cases with laboratory evidence of infection (2). The clinical criteria for Zika virus disease include the presence of one of four symptoms (fever, rash, arthralgia, and conjunctivitis), or Guillain-Barré syndrome, or an adverse pregnancy outcome (fetal loss, or in utero findings of microcephaly or intracranial calcifications) in a symptomatic or asymptomatic mother with compatible illness or epidemiologic risk factors for Zika virus infection. Clinical criteria for Zika virus congenital infection in infants include microcephaly, intracranial calcifications, or other central nervous system abnormalities (2). Jurisdictions report cases meeting these criteria to ArboNET. Although jurisdictions can report asymptomatic infection in pregnant women without pregnancy complications to ArboNET, this reporting is at the discretion of the local jurisdiction and is not universal. Current ArboNET reporting includes cases of Zika virus disease that meet the interim CSTE case definition.

For the purposes of the USZPR and ZAPSS, laboratory evidence of possible Zika virus infection is defined as a positive Zika virus real-time reverse transcription–polymerase chain reaction (rRT-PCR) test result (i.e., a confirmed case of Zika virus infection) or an equivocal or presumptive positive Zika virus immunoglobulin M (IgM) antibody capture enzyme-linked immunosorbent assay (ELISA) test result (3–5).[¶] Plaque reduction neutralization testing (PRNT) performed

* <http://www.cdc.gov/westnile/resourcepages/survresources.html>.

[†] In the surveillance systems, laboratory evidence of Zika virus infection is defined as a positive Zika virus real-time reverse transcription–polymerase chain reaction test or a positive Zika virus immunoglobulin M (IgM) antibody test using the CDC IgM antibody capture enzyme-linked immunosorbent assay (ELISA). Plaque reduction neutralization testing (PRNT) performed in conjunction with the IgM ELISA must have Zika PRNT titers ≥ 10 .

[§] <http://www.cdc.gov/zika/hc-providers/registry.html>.

[¶] <http://www.cdc.gov/zika/hc-providers/diagnostic.html>; <http://www.cdc.gov/zika/hc-providers/qa-pregnant-women.html>.

in conjunction with the IgM ELISA must have Zika PRNT titers ≥ 10 for inclusion. Pregnant women who meet laboratory criteria are included in the surveillance systems whether they report symptoms or not. Women are included retrospectively if laboratory evidence of congenital Zika virus infection is identified in fetal tissues, the placenta, or the infant.

The USZPR was initiated primarily to monitor outcomes in pregnant women returning from travel to areas with local Zika virus transmission (6). To date the majority of cases in pregnant women reported to USZPR are associated with travel, but it also includes cases of sexual transmission (7) and local transmission from the U.S. territories. ZAPSS was developed separately for Puerto Rico to conduct enhanced surveillance in pregnant women at risk for Zika virus infection as a result of ongoing local Zika virus transmission. Using USZPR and ZAPSS, CDC will report the number of pregnant women with laboratory evidence of possible Zika virus infection weekly on its website. Data reported by noon Eastern Standard Time each Thursday (for this report, May 12, 2016) will be verified and reported in aggregate the following Thursday. Reporting is subject to a lag of 1 week to verify data from each participating jurisdiction. Reports from Arizona and Idaho have not yet been verified and are excluded from the current report.

As of May 12, 2016, combined data from USZPR and ZAPSS include 279 reports of pregnant women with laboratory evidence of possible Zika virus infection, including 157 pregnant women residing in U.S. states and the District of Columbia (Figure 1) and 122 residing in U.S. territories (Figure 2). As of May 11, 2016, 113 pregnant women meeting clinical criteria for Zika virus disease were reported to CDC through ArboNET, 48 in U.S. states, and 65 in U.S. territories.

Among the 157 pregnant women from U.S. states and the District of Columbia monitored through USZPR, 73 (49%)** reported clinical symptoms consistent with Zika virus disease. Among these symptomatic pregnant women, 64 (88%) reported rash, 36 (49%) arthralgia, 37 (51%) fever, and 17 (23%) conjunctivitis. Among all pregnancies included from U.S. states, Zika virus nucleic acid detection by rRT-PCR was reported in 39 (25%).

Among 122 pregnant residents of the U.S. territories†† being monitored in USZPR or ZAPSS, 80 (66%)§§ reported clinical symptoms consistent with Zika virus disease. Among these symptomatic women, 60 (75%) reported rash, 29 (36%) arthralgia, 27 (34%) fever, and 15 (19%) conjunctivitis. Among all women included from U.S. territories, Zika virus nucleic acid detection by rRT-PCR in serum was identified in 67 (55%).

** Eight missing information on symptom status.

†† All U.S. territories are participating.

§§ One missing information on symptom status.

Discussion

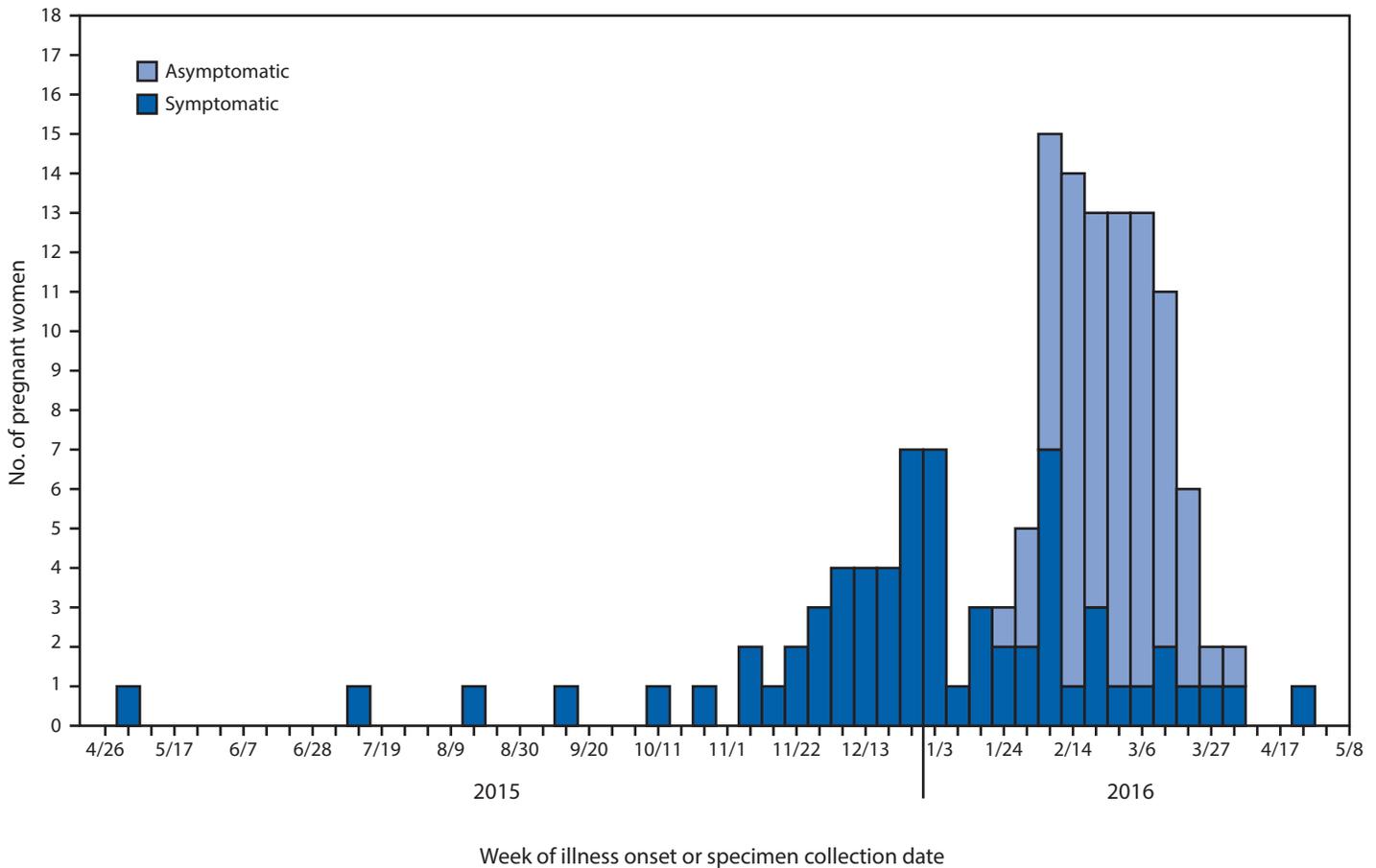
Through the establishment of these pregnancy surveillance systems, CDC, in collaboration with state, local, tribal, and territorial partners, is reporting and actively monitoring pregnant women with laboratory evidence of possible Zika virus infection. These surveillance systems monitor pregnant women at risk for adverse congenital outcomes attributable to possible Zika virus infection. Including pregnant women with laboratory evidence of possible Zika virus infection but without a reported history of symptoms more than doubles the number of pregnancies being monitored, compared with pregnancies meeting the interim CSTE case definition and reported by ArboNET.

Limiting surveillance to symptomatic women with confirmed or probable Zika virus disease or to women already affected by an adverse pregnancy outcome excludes a substantial proportion of women with asymptomatic and possible Zika virus infection during pregnancy. In contrast, the broader case definition used for the USZPR and ZAPSS surveillance systems might overestimate Zika virus infection among women screened for infection because of crossreactivity with dengue and other flaviviruses, particularly among residents of U.S. territories and travelers with a history of prior flavivirus infection or flavivirus vaccination (8), or nonspecific reactivity.

Case reports indicate that fetuses and infants of pregnant women with asymptomatic Zika virus infection might be at risk for microcephaly and other severe brain defects (9,10). Following pregnant women with laboratory evidence of possible Zika virus infection in the surveillance system, regardless of symptoms, allows better characterization of the full impact and consequences of infection to the mother and her offspring, and might allow for better stratification of risk for adverse congenital outcomes (1). An important role of the USZPR and ZAPSS surveillance systems is evaluating the range of outcomes associated with Zika virus infection during pregnancy. Pregnancy outcomes are currently being monitored and will be shared in future reports. It is critical that health care providers inform state, local, tribal, and territorial health departments of any pregnant women with laboratory evidence of possible Zika virus infection under their care.

The findings in this report are subject to at least three limitations. First, data provided to the jurisdictions and CDC regarding symptoms and symptom onset might not be accurate or complete because of variability in recall by patients or data available to jurisdictions. Second, only pregnant women who are tested for Zika virus infection are included, thereby

FIGURE 1. Week of illness onset for symptomatic pregnant women or specimen collection date* for asymptomatic pregnant women^{†,§} with laboratory evidence[¶] of possible Zika virus infection, by symptom status (N = 142)** — 48 states^{††} and the District of Columbia, April 26, 2015–May 12, 2016



Abbreviations: ELISA = enzyme-linked immunosorbent assay; IgM = immunoglobulin M; PRNT = plaque reduction neutralization test.

* Date of onset of symptoms or testing.

[†] Specimen collection dates for asymptomatic pregnant women might not coincide with the period of exposure or infection with Zika virus.

[§] CDC issued updated interim guidelines on February 5, 2016, to include recommending serologic testing of asymptomatic pregnant women 2–12 weeks after travel to an affected area.

[¶] Laboratory evidence of possible Zika virus infection is defined as a positive Zika virus real-time reverse transcription–polymerase chain reaction test or a positive Zika virus IgM ELISA test; if PRNT is performed in conjunction with the IgM ELISA, Zika PRNT titers must be ≥ 10 for inclusion.

** Excludes 15 women with missing symptom status or missing date of symptom onset.

^{††} Figure includes data for U.S. states from the U.S. Zika Pregnancy Registry, excluding Arizona and Idaho.

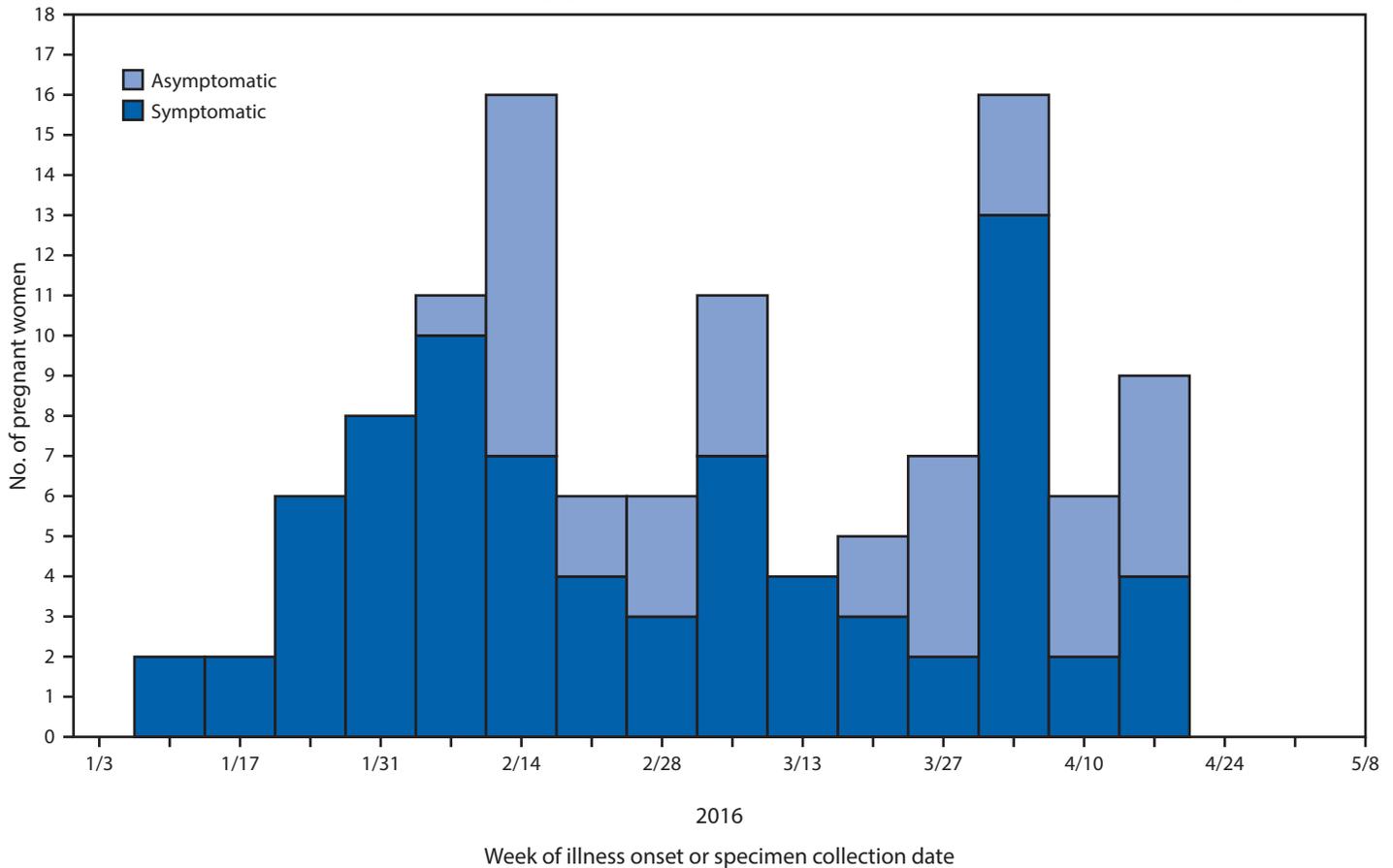
potentially underestimating the prevalence of infection and outcomes among all pregnant women. Finally, all states are not included in the USZPR, possibly affecting the representativeness of these data with regard to all pregnant women identified with a possible Zika virus infection.

One challenge of this Zika virus outbreak is the lack of understanding of the magnitude of risk and spectrum of outcomes associated with Zika virus infection during pregnancy. The USZPR and ZAPSS are surveillance systems established to enumerate and describe pregnancies with

Zika virus infection and risk for adverse outcomes associated with infection during pregnancy. Findings from these U.S. surveillance systems are expected to improve understanding of Zika virus infection during pregnancy, enhance risk assessment and counseling of pregnant women and families, advance clinical care, and assist states and territories to anticipate and plan needed resources and increase prevention efforts.^{¶¶}

^{¶¶} <http://www.cdc.gov/zika/pregnancy/question-answers.html>.

FIGURE 2. Week of illness onset for symptomatic pregnant women or specimen collection date* for asymptomatic pregnant women^{†,§} with laboratory evidence[¶] of possible Zika virus infection, by symptom status (N = 115) — U.S. territories,^{††} January 3, 2016–May 12, 2016**



Abbreviations: ELISA = enzyme-linked immunosorbent assay; IgM = immunoglobulin M; PRNT = plaque reduction neutralization test.

* Date of onset of symptoms or testing.

[†] Specimen collection dates for asymptomatic pregnant women might not coincide with the period of exposure or infection with Zika virus.

[§] CDC issued updated interim guidelines on February 5, 2016, to include recommending serologic testing of asymptomatic pregnant living in an area with active Zika virus transmission in the first and second trimester.

[¶] Laboratory evidence of possible Zika virus infection is defined as a positive Zika virus real-time reverse transcription–polymerase chain reaction test or a positive Zika virus IgM ELISA test; if PRNT is performed in conjunction with the IgM ELISA, Zika PRNT titers must be ≥10 for inclusion.

** Excludes seven women with missing symptom status or missing date of symptom onset.

^{††} Figure includes data for U.S. territories from the U.S. Zika Pregnancy Registry and the Zika Active Pregnancy Surveillance System.

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Summary

What is already known about this topic?

Zika virus infection during pregnancy causes microcephaly and other serious brain abnormalities. However, the full range of outcomes of asymptomatic and symptomatic Zika virus infection during pregnancy are not yet well understood.

What is added by this report?

In February 2016, CDC, in collaboration with state, local, tribal, and territorial health departments, launched comprehensive surveillance systems to report and actively monitor pregnancies and congenital outcomes among symptomatic and asymptomatic women with laboratory evidence of possible Zika virus infection. As of May 12, 2016, there were 157 and 122 pregnant women with laboratory evidence of possible Zika virus infection residing in U.S. states and U.S. territories, respectively.

What are the implications for public health practice?

This report launches the weekly reporting of pregnant women with laboratory evidence of possible Zika virus infection in U.S. states and territories. Monitoring all pregnant women with possible Zika virus infection during pregnancy, whether asymptomatic or symptomatic, will enhance understanding of possible adverse outcomes and allow better estimates of the number of pregnancies at risk for adverse outcomes. This information will assist health care providers who counsel pregnant women and will facilitate planning services for affected families.

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Notes from the Field

Outbreak of Serogroup B Meningococcal Disease at a University — California, 2016

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On January 31, 2016, the Santa Clara County Public Health Department (SCCPHD) was notified of a suspected case of meningococcal disease in a university undergraduate student. By February 2, two additional suspected cases had been reported in undergraduate students living on the same campus. The index patient (patient A) required intensive care, whereas patients B and C had milder illness; there were no deaths. All three patients were part of overlapping social networks and had attended the same events during the week before the onset of patient A's symptoms, but whether they had direct contact with one another could not be verified. Serogroup B *Neisseria meningitidis* was identified in cerebrospinal fluid and blood from patient A and in blood from patient B. Serogroup B has been responsible for all U.S. college outbreaks of meningococcal disease since 2011 (1). Laboratory results for patient C were inconclusive.

The university student health center and a local hospital began providing ciprofloxacin chemoprophylaxis to students in the social networks of patient A on January 31, the day the case was reported. Expanded postexposure chemoprophylaxis to social network members (e.g., persons sharing social events) in addition to close contacts is recommended by the California Department of Public Health (CDPH) for single cases in crowded environments such as college campuses (2). As a result, patients B and C received ciprofloxacin after symptom onset but before they received their diagnoses, which might have prevented more severe disease. Additional students were targeted for chemoprophylaxis after cases in patients B and C were reported. A total of 436 students in the social networks of the three patients, which included social organizations and athletic teams, received ciprofloxacin.

After the second case was confirmed on February 2, SCCPHD and CDPH recommended that meningococcal serogroup B (MenB) vaccine be offered to the university student population. Two MenB vaccines are licensed in the United States, MenB-4C (Bexsero, GlaxoSmithKline, Middlesex,

United Kingdom) and MenB-FHbp (Trumenba, Pfizer, New York, New York). In 2015, the Advisory Committee on Immunization Practices (ACIP) recommended use of MenB vaccines during outbreaks and for persons at increased risk for meningococcal disease (3). In addition, MenB vaccines may be administered to any adolescent or young adult aged 16–23 years (4).

Federally funded MenB-4C vaccine was provided by CDPH at no cost. All 5,232 undergraduate students, as well as graduate students and faculty and staff members at increased risk for meningococcal disease, were advised to receive vaccine. Persons at increased risk were defined as persons with underlying health conditions as recommended by ACIP (3,5,6) and persons living in on-campus housing at the time of the outbreak (208 persons other than undergraduate students). During four vaccination clinics held February 4–8, a total of 4,921 persons received the first vaccine dose. Vaccination clinics for the second vaccine dose were held on March 18 and April 6–8, during which 4,731 persons were vaccinated (some of whom had not received the first dose). No additional cases in Santa Clara County were identified as of May 23, 2016.

The response to this outbreak was rapid, with the first vaccination clinic conducted <48 hours after the second case was confirmed. University officials had conducted a serogroup B meningococcal disease outbreak tabletop exercise in June 2015, and SCCPHD had updated their incident command system protocol in January 2016. Factors that might have contributed to the rapid response include availability of a licensed vaccine, high levels of preparedness and activation of incident command systems at both the university and SCCPHD, and close partnerships among the state and local health department and the university.

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Notes from the Field

Expanded Chemoprophylaxis Offered in Response to a Case of Meningococcal Meningitis in an Elementary School — Indiana, 2015

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On December 11, 2015, the Fort Wayne-Allen County (Indiana) Department of Health was notified by a local hospital laboratory of a suspected case of meningococcal meningitis based on Gram stain results of cerebrospinal fluid. The county health department interviewed close family members and friends of the patient to establish an infectious period, timeline of events, and possible exposures. Close medical and household contacts were offered chemoprophylaxis (1). This case was associated with an elementary school. The patient had intermittent, close, potentially face-to-face contact with many students, and was reported to have had a persistent, productive cough throughout the exposure period. In light of these unusual circumstances, and the fact that elementary school-aged children are not routinely vaccinated against meningococcal disease,* local and state health officials, with CDC support, decided to offer chemoprophylaxis to the patient's contacts. A total of 581 child and adult contacts were identified.

Local, state, and federal public and private health care providers partnered in planning and implementing the distribution of prophylaxis and developing a communication strategy to inform parents and the community. This public-private partnership resulted in a high level of compliance with public health recommendations and minimal disruption to families and the elementary school.

The chemoprophylaxis clinic took place at the school on December 15, 2015. After discussion with CDC, ciprofloxacin oral suspension was offered according to published guidelines as the preferred antibiotic because of its ease of administration as a single dose, the need for varying dosages because of the number of children weighing <55 pounds (<25 kg), and the concern that some children might not be able to swallow pills (1). Parkview Regional Medical Center, a local nonprofit hospital, provided pharmacy services to facilitate and distribute appropriate dosages of ciprofloxacin (20 mg/kg, orally, up to a maximum of 500 mg) (1) and to answer questions regarding potential drug interactions, indications, and safety. Ceftriaxone was offered to pregnant or lactating women, and ceftriaxone or rifampin was offered to persons with contraindications to or precautions for ciprofloxacin use (2).

* <http://www.cdc.gov/vaccines/schedules/index.html>.

Among 581 persons identified as contacts, 496 (85%) received chemoprophylaxis; among these persons, 449 (91%) received chemoprophylaxis at the clinic, including 335 students and 114 school faculty members or volunteers. In addition, 12 health care workers, eight close household contacts, and 27 peripheral event contacts received chemoprophylaxis outside of the clinic. The county health department offered home delivery of medication for persons who were unable to attend the clinic, and the hospital pharmacy also stored the medication at the hospital to facilitate pickup. Despite these efforts, 15% of contacts chose not to pick up the medication for undisclosed reasons. No immediate adverse events were reported from those who received ciprofloxacin or rifampin provided at the clinic.

On December 21, 2015, the patient was confirmed to have *Neisseria meningitidis* serogroup B infection by testing performed at CDC. The patient fully recovered, and no additional cases were identified.

Although chemoprophylaxis of persons other than close contacts is not routinely recommended in response to a single case of meningococcal meningitis, in unique circumstances when expanded meningococcal chemoprophylaxis is warranted, it is important to identify a well-defined target group and ensure that all persons within the target group receive antibiotics within a short time frame. In this case, the communication framework and professional relationships developed during the 2009 H1N1 influenza pandemic created a multidisciplinary infrastructure that facilitated the investigation and response. Providing the appropriate chemoprophylaxis for approximately 500 persons in 2 days with minimal school disruption is an indicator of the strength of the local health partnerships with schools, hospitals, and health care providers, and of the lessons learned from previous public health emergencies.

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Announcement

National High Blood Pressure Education Month — May 2016

May is National High Blood Pressure Education Month. High blood pressure (hypertension) is a major contributor to heart disease and stroke, two leading causes of death in the United States.* High blood pressure affects one third of U.S. adults, or approximately 75 million persons, yet approximately 11 million of these persons are not aware they have hypertension, and approximately 18 million are not being treated (unpublished data) (1,2).

Certain groups are at increased risk for hypertension, including minorities and some women. In the United States, African-American men and women have higher rates of hypertension than any other race or ethnicity (3), and they are also more likely to be hospitalized for hypertension. Women with high blood pressure who become pregnant are more likely to have complications during pregnancy than are women with normal blood pressure (4). Hypertension can harm the mother's kidneys and other organs, and it can cause low birthweight and early delivery. Certain types of hormonal birth control can also raise a woman's risk for high blood pressure (5).

Hypertension affects persons of all ages: approximately one in four men and nearly one in five women aged 35–44 years have hypertension (3). New research also indicates that having uncontrolled high blood pressure during midlife (aged 45–65 years) increases the risk for dementia later in life (6,7). Vascular dementia—one of the most common types of dementia—is usually caused by the impact of multiple strokes over time, including small “silent” strokes that occur unnoticed. Hypertension is the main cause of these strokes (6,7).

Most persons with uncontrolled hypertension have health insurance (82%) and see their providers at least twice a year (62%), but their hypertension remains undiagnosed (8). An

important goal of the Million Hearts initiative is to equip health care providers with evidence-based tools and resources to identify and connect with these patients with undiagnosed hypertension.

In recognition of National High Blood Pressure Education Month, CDC and Million Hearts urge patients and health care professionals to learn more about the risks for high blood pressure at any age and encourage health care professionals to take steps to identify and treat patients with undiagnosed hypertension. Health care professionals can take advantage of evidence-based strategies and interactive tools and resources at <http://millionhearts.hhs.gov>. Additional information about hypertension is available at <http://www.cdc.gov/bloodpressure>.

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* <https://www.healthypeople.gov/2020/topics-objectives/topic/heart-disease-and-stroke>.

Notice to Readers

Changes in the Presentation of Zika Virus Disease, Non-Congenital Infection, and Addition of Zika Virus Congenital Infection to Notifiable Diseases and Mortality Table I

The Executive Board of the Council of State and Territorial Epidemiologists has approved the additions of “Zika virus disease, non-congenital infection” and “Zika virus congenital infection” to the list of nationally notifiable conditions reportable to the National Notifiable Diseases Surveillance System (NNDSS) beginning in 2016 (1). Therefore, Zika virus disease, non-congenital infection data previously displayed for 2015 will no longer appear in Table I (Provisional cases of selected infrequently reported notifiable diseases [$<1,000$ cases reported during the preceding year], United States) of the *MMWR* Weekly Notifiable Diseases and Mortality Tables, because the condition was not considered nationally notifiable at that time. Instead, “NN” (not nationally notifiable) will appear in the 2015 column for total cases reported for previous years.

Additional Modifications to Table I

Data for Zika virus disease, non-congenital infection have been displayed under “Arboviral diseases” in Table I of the *MMWR* Weekly Notifiable Diseases and Mortality Tables since February 5, 2016 (2). These data will now be displayed in Table I as a separate condition in conjunction with Zika virus congenital infection. CDC and the U.S. States are still modifying the needed technical infrastructure to collect and transmit data for Zika virus congenital infections; the ability to receive and display this data are anticipated in early summer 2016. A Zika virus congenital infections heading is included in Table I now as a placeholder until the data become available to display.

The Zika virus disease data in Table I do not include data from the U.S. Territories, where most of the Zika virus disease cases are currently occurring. Therefore, Zika virus disease case counts displayed in Table I of the *MMWR* Weekly Notifiable Diseases and Mortality Tables will be different from case counts displayed in other reports that include data from U.S. Territories.

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Errata

Vol. 65, No. 19

In the report, “Prevalence of Doctor-Diagnosed Arthritis at State and County Levels — United States, 2014,” multiple errors occurred.

On page 491, the third sentence of the second paragraph should have read, “In 2014, 47 states, DC, and **Puerto Rico** had an age-standardized prevalence of doctor-diagnosed arthritis of $\geq 20\%$, and four states had an age-standardized prevalence of arthritis of $\geq 30\%$ (Table 2).”

On page 491, the first sentence under Discussion should have read, “In 2014 doctor-diagnosed arthritis was common in the 50 states and DC (age-standardized median prevalence = 24.0%), affecting at least one in five adults in 47 states, DC, and **Puerto Rico** and nearly one in three adults in four states.”

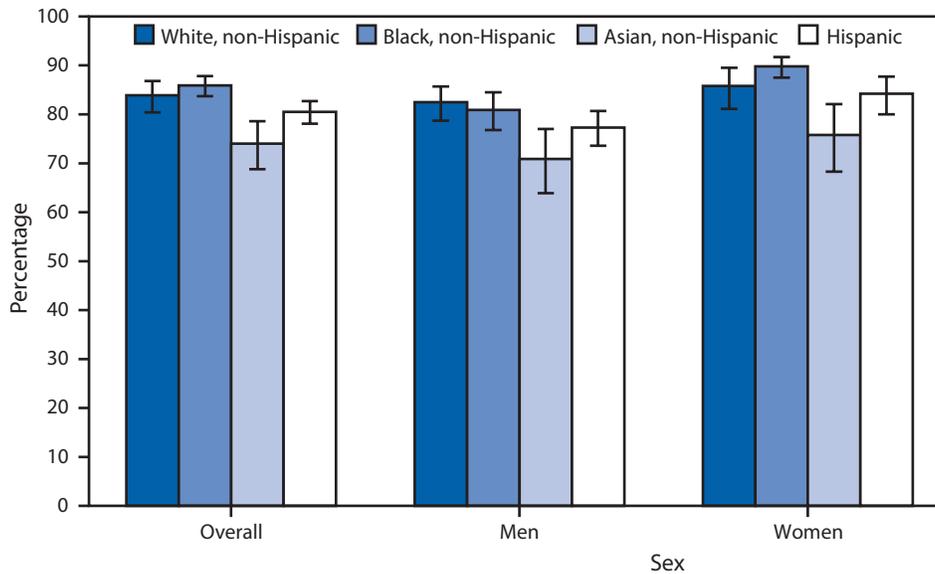
On page 492, in “TABLE 2. Weighted unadjusted and age-standardized prevalence of doctor-diagnosed arthritis* among adults aged ≥ 18 years, by state/area — Behavioral Risk Factor Surveillance System, United States, † 2014,” the values for Guam and Puerto Rico should have read as follows:

State/Area	No.	Weighted no. in population (in 1,000s) [§]	Unadjusted % (95% CI)	Age-standardized [¶] % (95% CI)
Guam	432	17	15.7 (13.9–17.6)	18.0 (16.2–20.0)
Puerto Rico	1,990	689	24.6 (23.3–5.8)	22.4 (21.3–23.5)

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Age-Adjusted Prevalence^{*,†} of Adults Aged ≥18 Years with Hypertension[§] Who Are Aware They Have Hypertension,[¶] by Sex and Race/Ethnicity — National Health and Nutrition Examination Survey, United States, 2011–2014



* With 95% confidence intervals indicated by error bars.

† Age-adjusted, using the subpopulation of persons aged ≥18 years with hypertension during 2007–2008.

§ Respondents were defined as having hypertension if their systolic blood pressure was ≥140 mm Hg or their diastolic blood pressure was ≥90 mm Hg, or they were currently taking medication to lower high blood pressure.

¶ Respondents with hypertension who answered “yes” to the question, “Have you ever been told by a doctor or health professional that you had hypertension, also called high blood pressure?”

For the period 2011–2014, 83.3% of adults aged ≥18 years with hypertension were aware of their hypertension status. Overall, a smaller percentage of non-Hispanic Asian adults (74.0%) with hypertension were aware of their status compared with non-Hispanic white (83.9%), non-Hispanic black (85.9%), and Hispanic adults (80.5%) with hypertension. This pattern generally was found for both men and women, with the exception of non-Hispanic Asian men and Hispanic men, where the difference was not significant. A larger percentage of non-Hispanic black and Hispanic women were aware of their hypertension condition compared with non-Hispanic black and Hispanic men, respectively.

Source: Nwankwo T, Yoon SS, Burt V, Gu Q. Hypertension among adults in the United States: National Health and Nutrition Examination Survey, 2011–2012. NCHS data brief no. 133; 2013. <http://www.cdc.gov/nchs/data/databriefs/db133.htm>.

CDC. National Health and Nutrition Examination Survey data. Hyattsville, MD: US Department of Health and Human Services, CDC, National Center for Health Statistics; 2013–2014. <http://www.cdc.gov/nchs/nhanes.htm>.

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Morbidity and Mortality Weekly Report

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