EPIDEMIOLOGIC SUMMARIES OF SELECTED GENERAL COMMUNICABLE DISEASES IN CALIFORNIA, 2009-2012

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Acknowledgements

We thank the California health care providers including the clinicians, infection control practitioners, hospital administrators, and laboratorians who reported the cases to their local health departments.

We thank the local public health workers including the local Health Officers, communicable disease controllers, epidemiologists, health data managers, public health nurses, morbidity clerks and support staff for providing these data to the California Department of Public Health (CDPH) and for their daily contributions to communicable disease surveillance and outbreak investigations.

We thank the current and former CDPH program staff and subject matter experts for their expertise and collaboration and for their contributions to communicable disease surveillance and outbreak investigations.

Background

The California Department of Public Health (CDPH) maintains a mandatory, passive reporting system for a list[1] of communicable disease cases and outbreaks. Health care providers and laboratories are mandated to report cases or suspected cases of these communicable diseases to their local health department (LHD). LHDs are also mandated to report these cases to CDPH.

These Technical Notes describe the definitions, methods, and limitations used to summarize the epidemiology of selected communicable diseases reported to CDPH[2]. In particular, these selected communicable diseases come from the general communicable diseases not covered by the categorical programs for tuberculosis, sexually transmitted diseases, HIV/AIDS, and vaccine- preventable diseases, all of which produce regular summaries of their diseases.

The distribution of information on the health of the community is a core function and essential service of public health. The data in the epidemiologic summaries provide important health information on the magnitude and burden of communicable diseases in California. Bearing in mind their limitations, these data can contribute toward identifying high risk groups needing preventive actions and tracking the effectiveness of control and prevention measures.

Materials and methods

Case data sources and inclusion criteria

For the 2009-2012 Epidemiologic Summaries of Selected General Communicable Diseases in California, we extracted data on communicable disease cases with an estimated onset date from 2009 through 2012 from California Confidential Morbidity Reports that were submitted to CDPH by May 4, 2013 which met the surveillance case definitions (see below). Similarly, due to inherent delays in case reporting, data for 2012 contained in these summaries are provisional and may differ from data published in future reports.

CDPH reviewed detailed clinical and laboratory data provided on disease-specific case history forms to determine if surveillance case definitions were met. LHDs applied surveillance criteria for diseases that did not require a case history form by regulation (campylobacteriosis, coccidioidomycosis, cryptosporidiosis, giardiasis, salmonellosis, and shigellosis).

We extracted data on foodborne and waterborne outbreaks with estimated onset dates from 2009 through 2012 from outbreak report forms submitted to CDPH by May 4, 2013 for the Epidemiologic Summary of Foodborne Disease Outbreaks in California, 2009 - 2012. These reports were the source for the number of outbreak-associated cases for each disease.

Population data source

For the 2009-2012 Summaries, we used State of California, Department of Finance population, projections, and estimations data[(3-7)].

Definitions

In general, we defined a case as laboratory and/or clinical evidence of infection or disease in a person that satisfied the most recent communicable disease surveillance case definition published by the United States (US) Centers for Disease Control and Prevention (CDC) or by the Council of

State and Territorial Epidemiologists (CSTE)[8 & 9]. Surveillance case definitions are described in individual disease summaries. By California regulation, an animal case was one that was determined, by a person authorized to do so, to have rabies or plague.

We defined the estimated onset date for each case as the date closest to the time when symptoms first appeared. Because date of onset may not be recorded, the estimated date of onset can range from the first appearance of symptoms to the date the report was made to CDPH. For diseases with insidious onset (for instance, coccidioidomycosis), estimated onset was more frequently drawn from the diagnosis date.

We defined single race-ethnicity categories as follows: Hispanic (of any, including unknown, race); White, non-Hispanic; Black, non-Hispanic; Asian/ Pacific Islander, Native American; and Other or multi-race. Cases with unknown race and ethnicity were listed as unknown.

We defined regions of California by collapsing counties with similar geography, demography and economic conditions as described by the Public Policy Institute of California[10]. Regions included the Far North (Butte, Colusa, Del Norte, Glenn, Humboldt, Lake, Lassen, Mendocino, Modoc, Nevada, Plumas, Shasta, Sierra, Siskiyou, Sutter, Tehama, Trinity, and Yuba Counties); Sacramento Metro (El Dorado, Placer, Sacramento, and Yolo Counties); Sierra (Alpine, Amador, Calaveras, Inyo, Mariposa, Mono, and Tuolumne Counties); Bay Area (Alameda, Contra Costa, Marin, Napa, San Francisco, San Mateo, Santa Clara, Solano, and Sonoma Counties); San Joaquin Valley (Fresno, Kern, Kings, Madera, Merced, San Joaquin, Stanislaus, and Tulare Counties); Central Coast: (Monterey, San Benito, San Luis Obispo, Santa Barbara, and Santa Cruz Counties); Inland Empire: (Riverside and San Bernardino Counties); South Coast: (Los Angeles, Orange, and Ventura Counties); and San Diego (Imperial and San Diego Counties). We defined Southern California as the counties comprising the Inland Empire, South Coast, and San Diego regions. All other counties comprised Northern California.

We defined a rate as unreliable if the relative standard error was 23 percent or more (a threshold recommended by the National Center for Health Statistics). The formulas used to calculate the relative standard error were:

- Incidence rate (IR) = Number of cases/population x 100,000
- Standard error (SE) = IR/√number of cases
- Relative standard error = SE/IR x 100

Data analyses

We reported case totals and rates per 100,000 population (unless otherwise indicated) stratified by estimated year of onset, age, and geographic residence. We calculated geographic-based rates by county, region, and bisection of the State (Northern or Southern California). Cases reported from the City of Berkeley were included in Alameda County and cases from the Cities of Long Beach and Pasadena were included in Los Angeles County.

To reduce the level of random error, we expanded the time and geographic range for incidence rates when few cases or small populations were identified. We produced multiple-year average rates and region-specific (rather than county-specific) rates, as needed. We calculated relative standard errors for all county-specific rates.

Because a substantial portion of race/ethnicity data were missing (disease-specific range: 12 to 50 percent), we did not calculate race/ethnicity specific incidence rates. However, because race/ethnicity can be an important marker for complex social, economic, and political

factors that influence health, we presented the distribution of single race/ethnicity categories among cases with complete information.

We evaluated the temporal trends in incidence rates for selected diseases using Poisson regression models. Values of $p < 0.05$ were considered statistically significant. Analyses were conducted using SAS Release 9.1 (SAS Institute, Inc, Cary North Carolina) and maps were created using ArcGIS version 9.3 (ESRI, Inc, Redlands, California).

Limitations

Data quality

CDPH relied on LHDs to apply surveillance and counting criteria for campylobacteriosis, coccidioidomycosis, cryptosporidiosis, giardiasis, salmonellosis, and shigellosis. It is possible that some cases did not meet surveillance case definitions or counting criteria.

Deaths

We presented the number of cases reported to CDPH Division of Communicable Diseases Control as having died with their disease. There is no standardized method for determining whether a communicable disease caused or contributed to the death for the purposes of reporting here. Deaths may have occurred after the report was filed (and thus not reported). The numbers of deaths and case- fatality ratios reported should be interpreted with caution.

Completeness of reporting

The numbers of disease cases in this report are likely to underestimate the true magnitude of disease. Among factors that may contribute to under-reporting are: delays in notification, limited collection or appropriate testing of specimens, health care-seeking behavior among ill persons, limited resources and competing priorities in LHDs, and lack of reporting by clinicians and laboratories. Among factors that may contribute to increased reporting are disease severity, the availability of new or less expensive diagnostic tests, changes in the case definition by CDC or CDPH, recent media or public attention, and active surveillance activities.

During the surveillance period 2009-2012, CDC and CDPH conducted active surveillance in Alameda, Contra Costa, and San Francisco Counties through the California Emerging Infections Program (CEIP). CEIP conducted active laboratory-based surveillance for *Salmonella*, *Shigella*, *Campylobacter*, *Escherichia coli* O157, Shiga toxin- producing *E. coli* (STEC) non-O157, *Listeria monocytogenes*, *Yersinia*, *Vibrio*, *Cryptosporidium*, and *Cyclospora* infection and active physician-based surveillance of pediatric hemolytic uremic syndrome (HUS) through a network of nephrologists in the catchment area. Therefore, cases of these diseases might be more completely reported in these counties.

Because outbreak-related case reports were not always identified as such on the Confidential Morbidity Report, it was not possible to ascertain the proportion of outbreak-related cases that were also reported as individual cases in the passive reporting system. Additionally, case definitions used to classify probable outbreak-related cases may not meet the more specific criteria required for individual case reporting. Therefore, outbreak-related cases may not be included in the total number of cases reported for each disease and outbreak-related cases reported in the probable classification may not meet surveillance reporting criteria.

Small numbers and rate variability

All rates, even those based on full population counts, are subject to random error. Random error may be substantial when the number of cases is small (e.g., less than 20) and can make it impossible to distinguish random fluctuations from true changes in the underlying risk of disease. Rates and proportions based on small numbers should be interpreted with caution.

Rate comparisons

Incidence rate comparisons between geographic entities and over time should be done with caution. Because not all LHDs reported age data, the rates in this report are not age-adjusted. Additionally, the limitations previously listed (especially the completeness of reporting and random variability of rates) should be considered when interpreting and comparing incidence rates.

References

[1]California Code of Regulations, Title 17, Sections 2500 and 2505 https://archive.cdph.ca.gov/HealthInfo/Documents/Reportable_Diseases_Conditions.pdf

[2]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001 – 2008, & 2009- 2012. https://archive.cdph.ca.gov/data/statistics/Pages/EpiSummariesCDsCA-01-08.aspx

[3]State of California, Department of Finance,Race/ Ethnic Population with Age and Sex Detail, 2000– 2050. Sacramento, CA, July 2007.

[4]State of California, Department of Finance, Race/Hispanic Population with Age and Gender Detail, 2000–2010. Sacramento, CA, September 2012.

[5]State of California, Department of Finance, E-4 Population Estimates for Cities, Counties and the State, 2000 – 2010, with 2000 & 2010 Census Counts. Sacramento, California, November 2012

[6]State of California, Department of Finance, P-3 State and County Population Projections by Race/Ethnicity, Detailed Age, and Gender, 2010 – 2060. Sacramento, California, January 2013

[7]State of California, Department of Finance, E-4 Population Estimates for Cities, Counties, and the State, 2011 – 2013, with 2010 Census Benchmark. Sacramento, California, May 2013

[8]Centers for Disease Control and Prevention. National Notifiable Diseases Surveillance System, Case Definitions for Infectious Conditions Under Public Health Surveillance http://www.cdc.gov/osels/ph_surveillance/nndss/phs/infdis2011.htm

[9] 2012 Case Definitions: Nationally Notifiable Conditions Infectious and Non-Infectious Case. (2012). Atlanta, GA: Centers for Disease Control and Prevention http://wwwn.cdc.gov/nndss/document/2012case%20definitions.pdf

[10]Johnson, H. *'A State of Diversity: Demographic Trends in California's Regions', California Counts*, Vol 3, No 1, Public Policy Institute of California, San Francisco, California 2002 http://www.ppic.org/content/pubs/cacounts/CC_502HJCC.pdf

Updated by Farzaneh Tabnak, PhD. 01/21/2014

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 93 confirmed and 5 probable cases of brucellosis with estimated illness onset dates from 2009 through 2012. This corresponds to an incidence rate of 0.07 per 100,000 population per year.

- Brucellosis incidence decreased by 33.3 percent from 2009 (24 cases; 0.06 per 100,000 population) to 2011 (15 cases; 0.04 per 100,000 population), but increased by 150.0% from 2011 to 2012 (37 cases; 0.10 per 100,000 population). During the surveillance period, one (1.0 percent) case-patient was reported to have died with brucellosis.

- Brucellosis incidence rates over the four-year surveillance period were highest among persons 75 to 84 years of age (0.25 per 100,000 population per year) and persons 85 years of age and older (0.20 per 100,000 population per year). The ratio of male to female case-patients was 1.1:1.0.

- Hispanic (84.2 percent) ethnicity was reported more frequently for brucellosis case-patients than would be expected based on the overall proportion in California (37.8 percent).

- Avoiding consumption of unpasteurized dairy products (e.g., milk, cheese), wearing protective clothing and washing hands thoroughly when handling livestock reproductive tissues (e.g., aborted fetuses, placentas), and using appropriate respiratory protection when working with livestock or their tissues in a confined space (e.g., slaughterhouse, laboratory) may provide the best opportunities for prevention of brucellosis among those persons at highest risk.

Background

Brucella spp. are uncommon but important bacterial zoonotic pathogens in the United States (US), causing an estimated 100 to 200 cases of human illness each year. Since 1954, the U.S. Department of Agriculture's National Brucellosis Eradication Program has significantly reduced the prevalence of *Brucella* in domestic livestock through routine testing, culling, and vaccination. Domestic cattle in California have been brucellosis-free since 1997. However, brucellosis remains an important zoonotic disease in other countries where domestic animal health programs are suboptimal. Consuming raw cow or goat milk products illegally imported from other countries is the most common route of exposure in California. Contact through broken skin with infected animal reproductive tissues and fluids, or inhalation of bio-aerosols, can also lead to infection, most notably in occupational settings such as livestock ranches, laboratories, slaughterhouses, meat-packing industry, and veterinary settings. Persons who harvest and dress certain wild animals (e.g., boar) may also be exposed to *Brucella* spp. Person- to-person transmission is extremely rare. *Brucella* spp. are listed among the U.S. Centers for Disease Control and Prevention (CDC) category B bioterrorism agents.

Brucellosis has a variable and sometimes prolonged incubation period (5 days to 6 months) and often presents as a nonspecific febrile syndrome (acute or insidious onset of fever, night sweats, fatigue, headache, and arthralgia). If treatment is delayed, patients may experience recurrent or 'undulant' fevers and possibly focal infections in bones, joints, liver, kidney, spleen, brain, or heart

valves[1,2].

We describe here the epidemiology of confirmed and probable human brucellosis cases in California with estimated illness onset from 2009 through 2012 that were reported to CDPH by August 27, 2014. Data for 2012 are provisional and may differ from data in future publications. The epidemiologic description of brucellosis for the 2001-2008 surveillance period was previously published in the Epidemiologic Summary for Brucellosis in California, 2001—2008[3]. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes[4].

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to immediately report known or suspected cases of brucellosis to their local health jurisdiction. Laboratories must immediately communicate by telephone with the CDPH Microbial Diseases Laboratory for instructions whenever a specimen for laboratory diagnosis of suspected human brucellosis is received. Laboratories must also report to the local health jurisdiction where the health care provider who first submitted the specimen is located, when laboratory testing yields evidence suggestive of *Brucella* sp.

California regulations also require local health officers to immediately report to CDPH cases of brucellosis. CDPH officially counted cases that satisfied the CDC surveillance case definition.

CDC defines a confirmed case as one with an illness clinically characterized by acute or insidious onset of fever, and one or more of the following: night sweats, arthralgia, headache, fatigue, anorexia, myalgia, weight loss, arthritis/spondylitis, meningitis, or focal organ involvement (endocarditis, orchitis/epididymitis, hepatomegaly, splenomegaly), along with definitive laboratory evidence of *Brucella* infection. Definitive laboratory evidence of *Brucella* infection included either culture and identification of *Brucella* sp. from clinical specimens or evidence of a fourfold or greater rise in *Brucella* antibody titer between acute- and convalescent-phase serum specimens obtained greater than or equal to 2 weeks apart. A probable case is defined as clinically compatible illness and either an epidemiologic link to a confirmed case or presumptive laboratory evidence (supportive serology using the agglutination method or detection of *Brucella* DNA by PCR)[5].

Epidemiology of brucellosis in California

CDPH received reports of 93 confirmed and 5 probable cases of brucellosis with estimated illness onset dates from 2009 through 2012. This corresponds to an incidence rate of 0.07 per 100,000 Californians per year.

Brucellosis incidence decreased by 33.3 percent from 2009 (24 cases; 0.06 per 100,000 population) to 2011 (15 cases; 0.04 per 100,000 population), but increased by 150.0% from 2011 to 2012 (37 cases; 0.10 per 100,000 population), almost reaching the peak observed in 2001 (39 cases; 0.11 per 100,000 population) [Figure 1]. During the surveillance period, one (1.0 percent) case-patient was reported to have died with brucellosis.

Brucellosis incidence rates over the four-year surveillance period were highest among persons 75 to 84 years of age (0.25 per 100,000 population per year) and those 85 years of age and older (0.20 per 100,000 population per year) [Figure 2]. Among brucellosis case-patients with complete information on race/ethnicity (96.9 percent), Hispanic ethnicity (84.2 percent) was reported more frequently than would be expected based on the overall proportion in California (37.8 percent) [Figure 3]. The ratio of male to female cases was 1.1:1.0.

Brucellosis incidence rates for brucellosis during the four-year surveillance period were similar in Northern California (0.07 per 100,000 population per year) and Southern California (0.06 per 100,000 population per year). However, incidence rates for the Central Coast (0.19 per 100,000 population per year), San Diego (0.12 per 100,000 population per year), and Sacramento (0.12 per 100,000 population per year) regions were higher than other regions in the state [Figure 4].

Figure 1. California brucellosis case counts and incidence rates

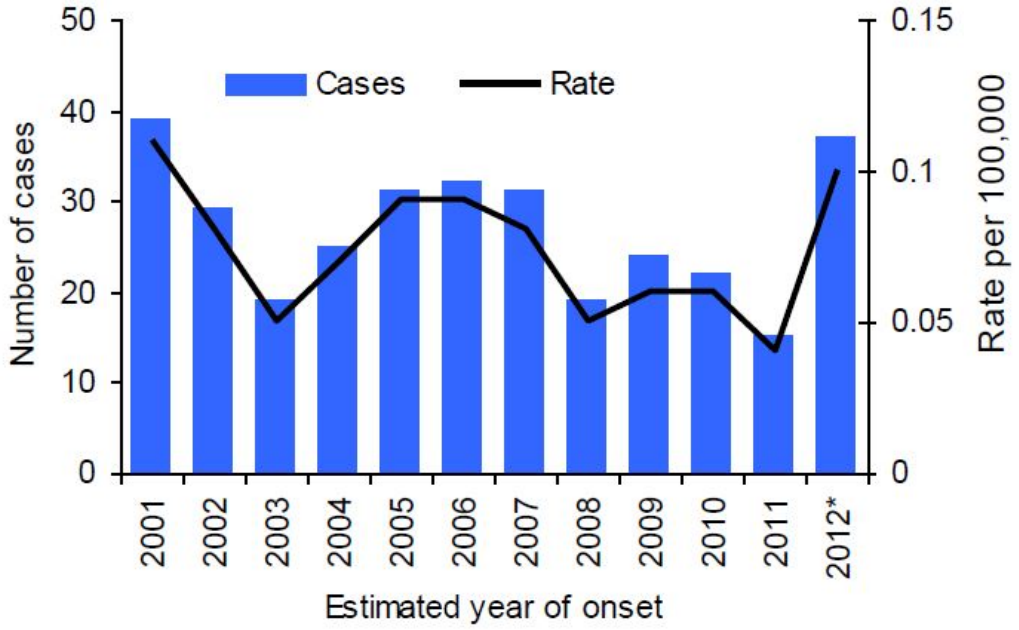


Figure 2. California brucellosis incidence rates by age 2009-2012*

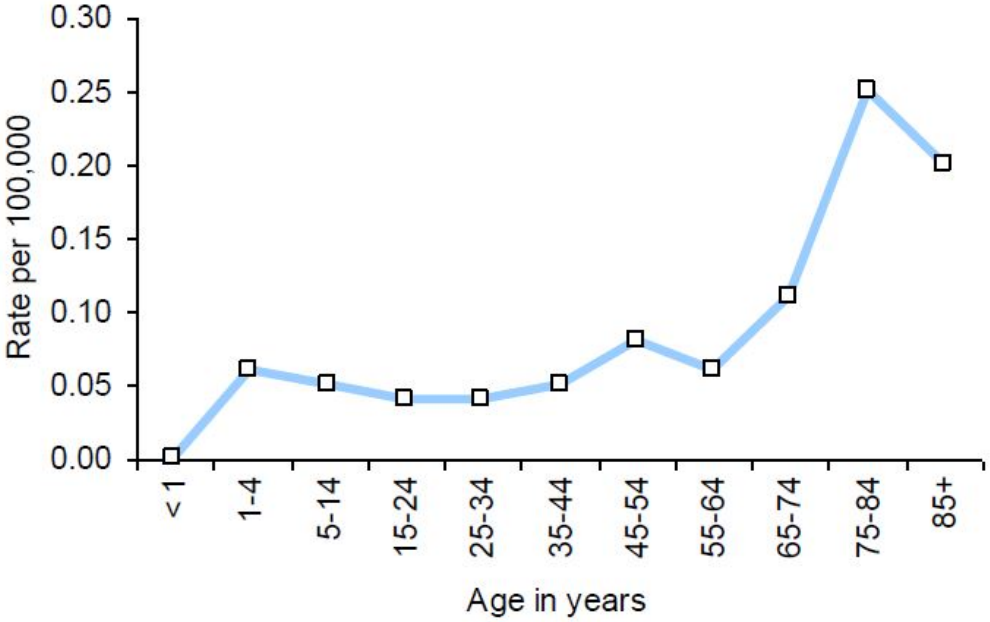
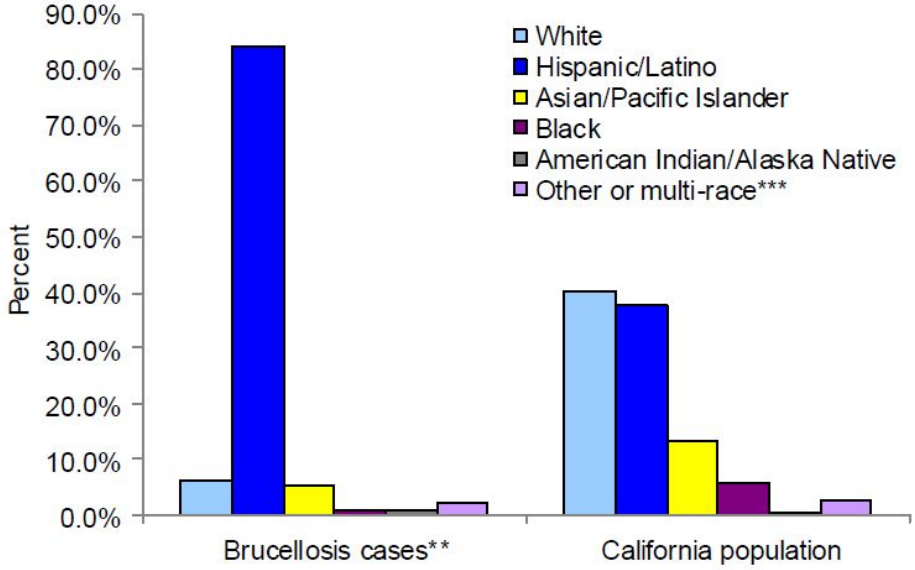


Figure 3. California brucellosis cases and population by race/ethnicity 2009-2012*



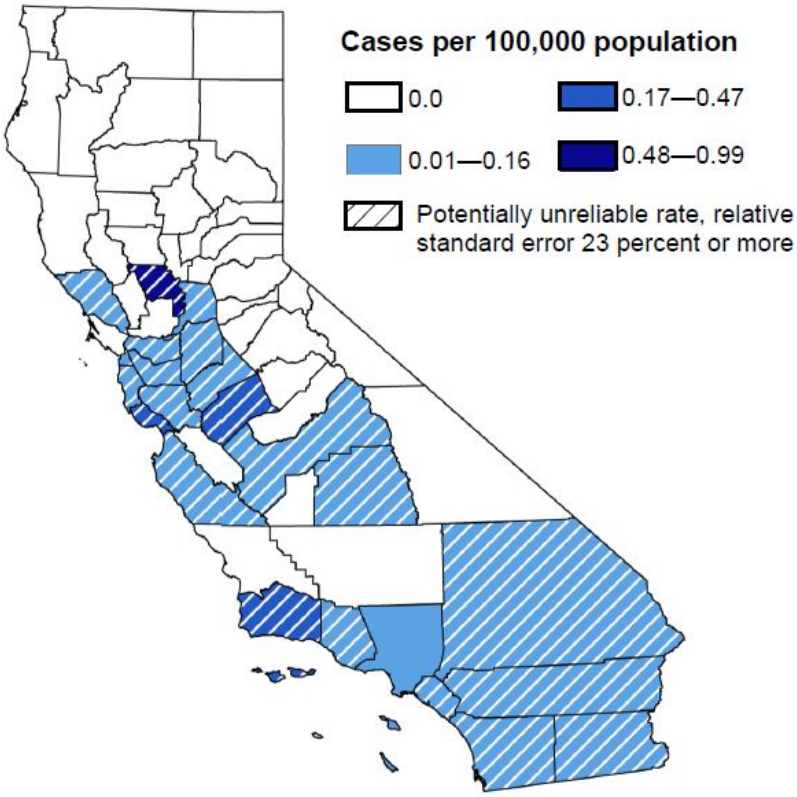
Notes for Figures 1-4

*2012 data are provisional

** Unknowns were excluded

*** Includes cases who identified "other" as their race and Californians ('population') who identified more than one race

Figure 4. California county-specific brucellosis incidence rates 2009 - 2012*



Notes for Figures 1-4

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** Unknowns were excluded

*** Includes cases who identified "other" as their race and Californians ('population') who identified more than one race

Comment

Brucellosis incidence in 2012 was 2.5 times that observed in 2011 nearing the peak observed in 2001. Brucellosis in California occurred disproportionately more frequently among persons of Hispanic ethnicity during surveillance period 2009 through 2012. This finding is consistent with the previously described report for surveillance period 2001 through 2008[3]. The highest rates of cases with estimated illness onset dates from 2009 through 2012 were among persons 75 to 84 years and those 85 years of age and older. Whereas, the highest rate of cases with estimated onset dates during 2001– 2008 was among persons 75 and 84 years of age[3]. The further shift of the disease toward older population and the higher frequency of the disease among persons of Hispanic ethnicity underscore the importance of prevention and control of brucellosis in California's aging and Hispanic population.

Animal brucellosis control programs (vaccination and/or test-and-slaughter of infected animals) are central to preventing

human cases. Avoiding consumption of unpasteurized dairy products (e.g., milk, cheese), wearing protective clothing and washing hands thoroughly when handling livestock reproductive tissues (e.g., aborted fetuses, placentas), and using appropriate respiratory protection when working with livestock or their tissues in a confined space (e.g., slaughterhouse, laboratory) may provide the best opportunities for prevention of brucellosis among those persons at highest risk[1,2].

Prepared by Farzaneh Tabnak, Alyssa Nguyen, Janice Kim, Curtis Fritz, Duc J Vugia, Infectious Diseases Branch

References and resources

1 CDC brucellosis information website
http://www.cdc.gov/brucellosis/exposure/expecting-mothers.html

2 CDPH brucellosis information website
http://www.cdph.ca.gov/HealthInfo/discond/Pages/Brucellosis.aspx

3 Epidemiological Summaries of Selected General Communicable Diseases in California, 2001—2008: Brucellosis
http://www.cdph.ca.gov/programs/sss/Documents/Epi-Summaries-CA-2001-2008-083111.pdf#page=9

4 Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001 - 2008 and 2009 - 2012: Technical Notes
http://www.cdph.ca.gov/programs/sss/Documents/TechnicalNotes01-08and09-12.pdfx

5 Brucellosis (*Brucella* spp.) 2010 Case Definition
http://wwwn.cdc.gov/nndss/script/casedef.aspx?CondYrID=625&DatePub=1/1/2010%2012:00:00%20AM

Last updated 10/30/2014

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 27,346 cases of confirmed and probable campylobacteriosis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 18.3 cases per 100,000 Californians.
- Campylobacteriosis annual incidence rate increased by 34.0 percent from 2009 (15.9 per 100,000) to 2012 (21.3 per 100,000).
- During the surveillance period, 29 (0.1 percent) case-patients were reported to have died with campylobacteriosis.
- Average annual campylobacteriosis incidence rates during the surveillance period were highest among children under 1 year of age (34.0 per 100,000) and 1 to 4 years of age (40.4 per 100,000). Incidence rates among all ages rose from 2009 to 2012 but increased the greatest among adults 75-84 years of age by 54.0 percent (from 16.3 to 25.1 per 100,000).
- From 2009 through 2012, CDPH received reports of 10 outbreaks of foodborne campylobacteriosis in California involving 132 cases.
- Decreasing contamination of poultry meat and dairy products, and educating consumers may provide the best opportunities for preventing and controlling campylobacteriosis.

Background

Campylobacter is among the most commonly reported enteric bacterial pathogens in the United States (US) causing an estimated 845,000 foodborne illnesses, 8,463 hospitalizations, and 76 deaths each year.[1] The US Centers for Disease Control and Prevention (CDC) estimates that for every reported case of campylobacteriosis, there are 30 more undiagnosed incidents.[1,2] The leading source of infection is foodborne, usually from consumption of contaminated animal products, particularly raw or undercooked poultry meat, and drinking of unpasteurized milk or contaminated water. Exposure to infected animals and their environments can also result in infection. Foodborne outbreaks of *Campylobacter* are relatively uncommon, in part because the organism does

Acute illness, usually gastroenteritis characterized by diarrhea, abdominal cramping and fever, occurs after an incubation period of 2 to 5 days, and usually lasts 1 week. Severe illness and death may rarely occur, particularly among immunocompromised persons. Complications, including Guillain-Barré syndrome and reactive arthritis, may also occur.[5] The recent emergence of human and animal *Campylobacter* isolates with fluoroquinolone resistance has led to restrictions on the use of some fluoroquinolones in poultry in the US.[6]

This report describes the epidemiology of confirmed and probable campylobacteriosis cases in California with estimated illness onset from January 2009 through December 2012 that were reported to CDPH by April 2015. Data for 2012 are provisional and may differ from data in future publications. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes.[7] The epidemiologic description of campylobacteriosis for the 2001-2008 period can be found in the *Epidemiologic*

Summary of Campylobacteriosis in California, 2001- 2008.[8]

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report any cases of campylobacteriosis to their local health department within one working day of identification or immediately by telephone if an outbreak is suspected. Laboratories are also required to report laboratory testing results suggestive of *Campylobacter* infection to either the California Reportable Disease Information Exchange (CalREDIE) via electronic laboratory reporting or to the local health department; reporting must occur within one working day after the health care provider has been notified.

Local health officers are required by regulation to report to CDPH cases of campylobacteriosis. CDPH counted cases that satisfied the CDC/Council of State and Territorial Epidemiologists' surveillance case definition of a confirmed or probable case. During the surveillance period, CDC defined a confirmed case as one with *Campylobacter* isolated from a clinical specimen including asymptomatic and extraintestinal infections.[9] A probable case was one with clinically-compatible illness and an established epidemiologic link to a laboratory-confirmed case.

Epidemiology of campylobacteriosis in California

CDPH received reports of 27,346 cases of campylobacteriosis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 18.3 cases per 100,000 Californians. Reported campylobacteriosis incidence rates increased by 34.0 percent from 2009 (15.9 per 100,000) to 2012 (21.3 per 100,000) [Figure 1]. During the surveillance period, 29 (0.1 percent) case-patients were reported to have died with campylobacteriosis.

Average annual campylobacteriosis incidence rates during the surveillance period were highest among children under 1 year of age (34.0 per 100,000) and 1 to 4 years of age (40.4 per 100,000). Incidence rates among all ages rose from 2009 to 2012 but increased the greatest among adults 75-84 years of age by 54.0 percent (from 16.3 to 25.1 per 100,000) [Figure 2]. The ratio of male to female cases was 1.2:1.0. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (58.3 percent). Of campylobacteriosis cases with complete data, reported race/ethnicities are roughly similar in proportions to the overall demographic profile of California [Figure 3].

Forty-eight (82.8 percent) of 58 counties reported average annual incidence rates for the surveillance period that were above the *Healthy People 2020* objective. Average annual incidence rates for the surveillance period were 1.9 times higher in Northern California (24.9 per 100,000) than Southern California (13.2 per 100,000). From 2009 to 2012, incidence rates for Southern California increased by 45.9 percent (from 11.1 to 16.2 per 100,000) and rates for Northern California increased by 26.1 percent (from 22.2 to 28.0 per 100,000). County-specific incidence rates for the surveillance period ranged from 0.0 to 55.0 per 100,000 persons [Figure 4].

From 2009 through 2012, CDPH received reports of 10 confirmed outbreaks of foodborne campylobacteriosis in California involving 132 cases. One multi-county outbreak involved 33 confirmed case-patients and was associated with drinking unpasteurized milk.

Figure 1. California campylobacteriosis case counts and incidence rates by estimated year of illness onset, 2009—2012*

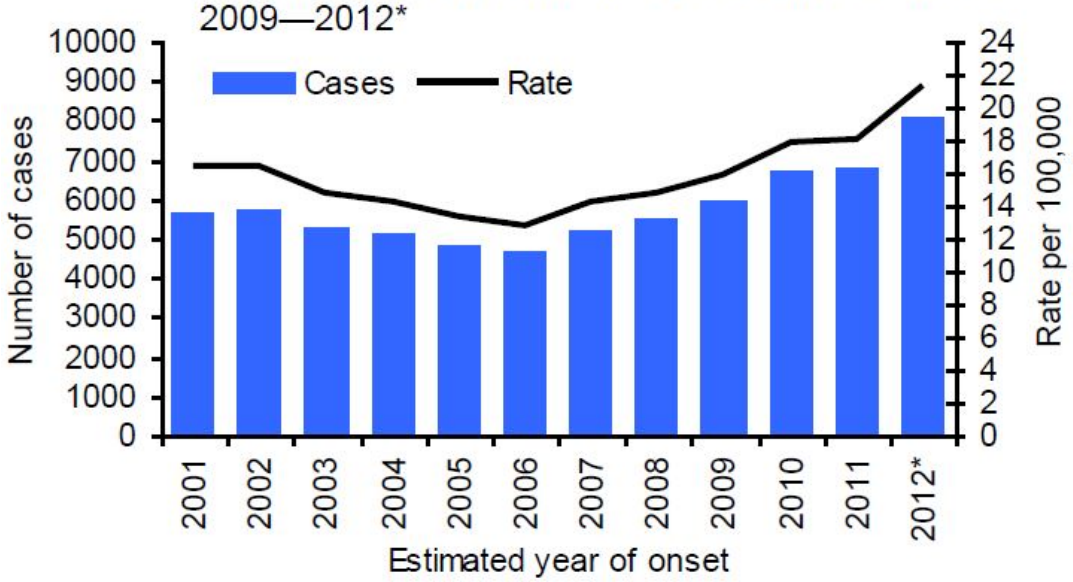


Figure 2. California campylobacteriosis incidence rates by age groups, 2009—2012*

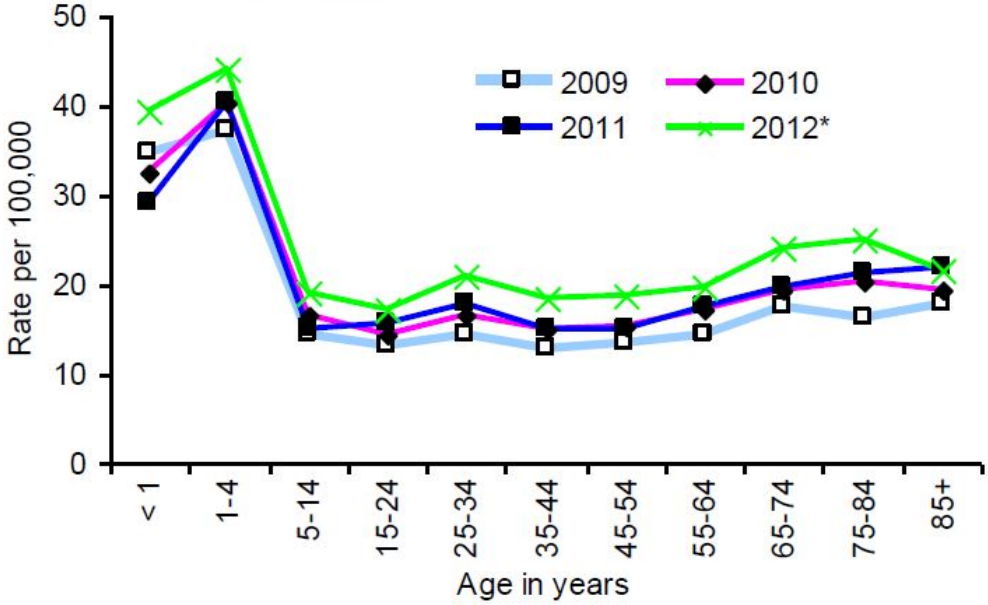
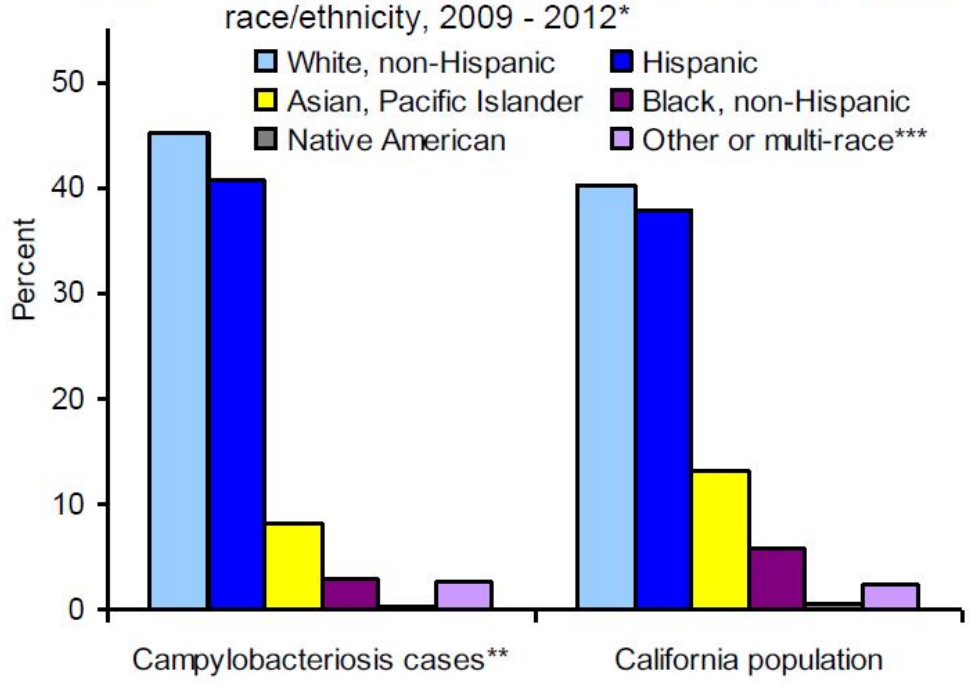


Figure 3. California campylobacteriosis cases and population by race/ethnicity, 2009 - 2012*



Notes for Figures 1-3

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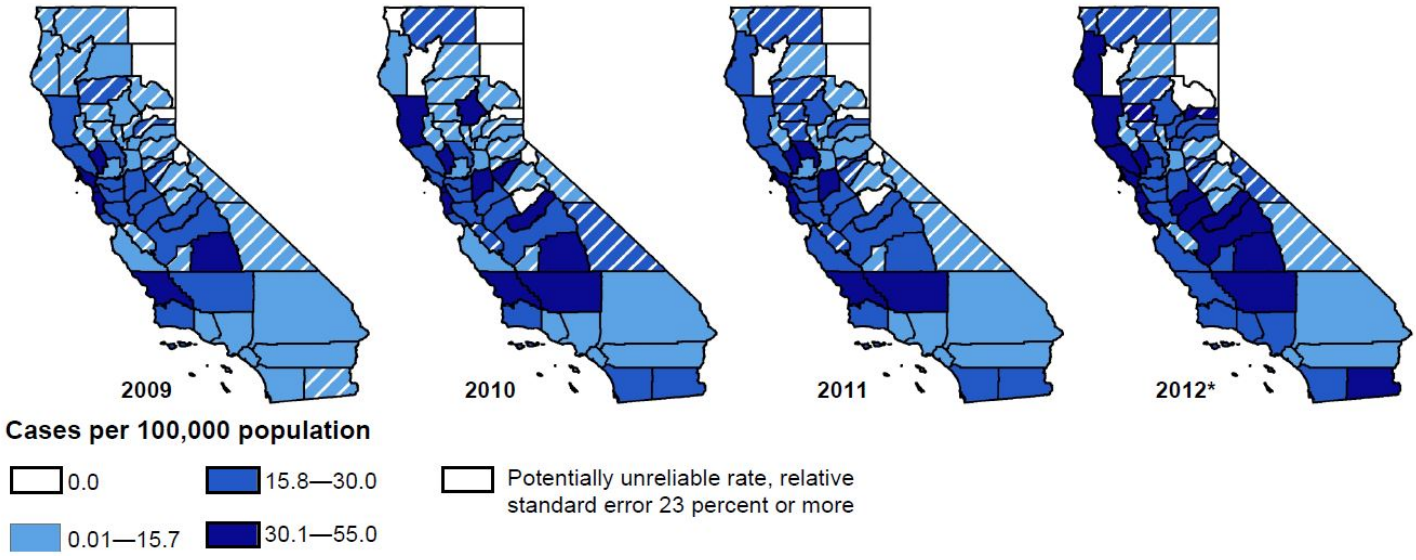
**Unknowns were excluded

***Includes cases who identified 'other' as their race and Californians ('population') who identified more than one race

Figure 4. California county-specific campylobacteriosis incidence rates



Comment

Between 2009 and 2012, California has experienced an increase in campylobacteriosis incidence with the highest rate occurring in 2012 (21.3 per 100,000). The reason for this recent increase is unknown. Continued monitoring of annual rates is needed.

Consuming contaminated poultry is heavily cited as the leading source of *Campylobacter* infection. Efforts have been taken to address this issue. In 2011, the United States Department of Agriculture (USDA) implemented the first-ever performance standard for detection of *Campylobacter* in poultry by setting a maximum percentage of samples that test positive at slaughterhouses.[10] Further measures were proposed in 2015 to increase the frequency of testing at these facilities.[11] Both efforts are predicted to reduce the presence of *Campylobacter* in poultry but because the pathogen cannot be entirely eradicated from the food-borne source, consumers must be educated in safe food handling and preparation methods to reduce risk. Decreasing the contamination of poultry meat and dairy products, and consumer education may provide the best opportunities for preventing and controlling campylobacteriosis.

References and resources

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and campylobacter 2010. http://www.usda.gov/wps/portal/usda/usdahome?contentidonly=true&contentid=2010/05/0246.xml

[11]United States Department of Agriculture. USDA proposes new measures to reduce salmonella and campylobacter in poultry products 2015. http://www.usda.gov/wps/portal/usda/usdahome?contentidonly=true&contentid=2015/01/0013.xml

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Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 16,108 incident cases of coccidioidomycosis with estimated symptom onset dates from 2009 through 2012. The annual number of incident cases peaked at 5,182 in 2011, the highest annual number since the increasing trend from 2001.
- Annual rates of coccidioidomycosis increased by 67.7 percent from 2009 (2,399 case-patients; 6.5 per 100,000 population) to 2012 (4,094 case-patients; 10.9 per 100,000). During 2001-2012 the highest annual incidence rate was in 2011 with 13.9 per 100,000 population.
- From 2009 through 2012, 213 (1.3 percent) case- patients were reported to have died with coccidioidomycosis.
- The highest average annual incidence rate occurred among persons in age group 45 to 54 years of age (14.8 per 100,000).
- Average annual incidence rates were highest in Kern (205.1 per 100,000), Kings (191.7 per 100,000), Fresno (64.5 per 100,000), San Luis Obispo (47.2 per 100,000), Tulare (39.2 per 100,000) and Madera (20.7 per 100,000) counties.
- During 2009-2012, CDPH received report of one point-source outbreak. In this 2009 outbreak, three organ donor recipients developed symptoms of coccidioidomycosis after receiving organs from a donor in Los Angeles County who was later determined to have coccidioidomycosis on post-mortem specimen testing.
- To decrease the risk of infection, persons living, working, or traveling in coccidioidomycosis endemic areas, especially those at increased risk for disseminated disease, should limit their exposure to outdoor dust as much as possible. It is important that healthcare providers be alert for coccidioidomycosis among patients who live in or have traveled to endemic areas.

Background

Coccidioidomycosis (also known as Valley Fever) results from directly inhaling spores of the dimorphic fungus *Coccidioides* spp. (*Coccidioidesimmitis* and *Coccidioidesposadasii*) from soil or airborne dust. *Coccidioides* is not transmitted directly from person-to- person. Although *Coccidioides* grows in localized areas of the southwest United States (US), the southern San Joaquin Valley is the major region of endemicity in California.

Of those infected with coccidioidomycosis, approximately 60 percent may be asymptomatic. Following an incubation period of 1 to 3 weeks, clinical manifestations occur in 40 percent of infected persons and range from influenza-like illness to severe pneumonia, and rarely, disseminated disease. Disseminated infection, which can be fatal, most commonly involves skin and soft tissues, bones, and the central nervous system. Persons at increased risk for severe disease include African-Americans, Filipinos, Hispanics, pregnant women, adults 60 years of age and older, and people with weakened immune systems[1,2,3].

We describe the epidemiology of reported coccidioidomycosis in California from 2009 through 2012. Data for 2012 are provisional and may differ from data in future publications. The epidemiological description of coccidioidomycosis for the 2001-2008 period can be found in the Epidemiologic Summary of Coccidioidomycosis in California, 2001—

2008[4]. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes[5]. We included in this summary only the first report of coccidioidomycosis per person during the surveillance period.

California reporting requirements and surveillance case definition

California Code of Regulations (CCR), Title 17, requires health care providers to report suspected cases of coccidioidomycosis to their local health department within 7 days or immediately by telephone if an outbreak is suspected. Since 2010, CCR, Title 17, Section 2505 has also mandated laboratories to report to the local health jurisdiction.

California regulations also require local health officers to report to CDPH cases of coccidioidomycosis. CDC defines a confirmed case as one with clinically compatible illness and at least one of the following: culture, histopathologic, or molecular evidence of *Coccidioides* species, or positive serologic test for coccidioidal antibodies in serum, cerebrospinal fluid, or other body fluids by: detection of coccidioidal immunoglobulin M (IgM) by immunodiffusion, enzyme immunoassay (EIA), latex agglutination, or tube precipitin; or detection of coccidioidal immunoglobulin G (IgG) by immunodiffusion, EIA, or complement fixation; or coccidioidal skin-test conversion from negative to positive after onset of clinical signs and symptoms. Clinical illness includes one or more of the following: influenza-like signs and symptoms, pneumonia or other pulmonary lesion, erythema nodosum or multiforme rash, involvement of the bones, joints, or skin by dissemination, meningitis, or involvement of viscera or lymph nodes.

Epidemiology of coccidioidomycosis in California

CDPH received reports of 16,108 incident cases of coccidioidomycosis with estimated symptom onset dates from 2009 through 2012. The annual number of incident cases peaked at 5,182 in 2011, the highest annual number since the increasing trend from 2001 (Figure 1). Annual rates of coccidioidomycosis increased by 67.7 percent from 2009 (2,399 case-patients; 6.5 per 100,000 population) to 2012 (4,094 case-patients; 10.9 per 100,000). During 2001-2012, the highest annual incidence rate was in 2011 with 13.9 per 100,000 population (Figure 1). From 2009 through 2012, 213 (1.3 percent) case-patients were reported to have died with coccidioidomycosis.

The highest average annual incidence rate occurred among persons 45 to 54 years of age (14.8 per 100,000) (Figure 2). Incidence rates by race/ethnicity were not calculated due to the substantial missing data (44.4 percent). However, cases with complete data reported Hispanic ethnicity and Black (non-Hispanic) race more frequently than would be expected based on the overall demographic profile of California (Figure 3). The ratio of male to female case-patients was 2.0:1.0.

Average annual incidence rates from 2009 through 2012 were highest in Kern (205.1 per 100,000), Kings (191.7 per 100,000), Fresno (64.5 per 100,000), San Luis Obispo (47.2 per 100,000), Tulare (39.2 per 100,000) and Madera (20.7 per 100,000) counties (Figure 4) which are established *Coccidioides*-endemic areas. Approximately 73.6 percent of case-patients resided or were incarcerated in these six counties at the time of symptom onset. There were eleven counties that reported no cases during 2009-2012.

Since 2009, CDPH received report of one point- source outbreak; three organ donor recipients developed symptoms of coccidioidomycosis after receiving organs from a donor in Los Angeles County who was later determined to have coccidioidomycosis on post-mortem specimen testing.

Figure 1. California coccidioidomycosis case counts and incidence rates

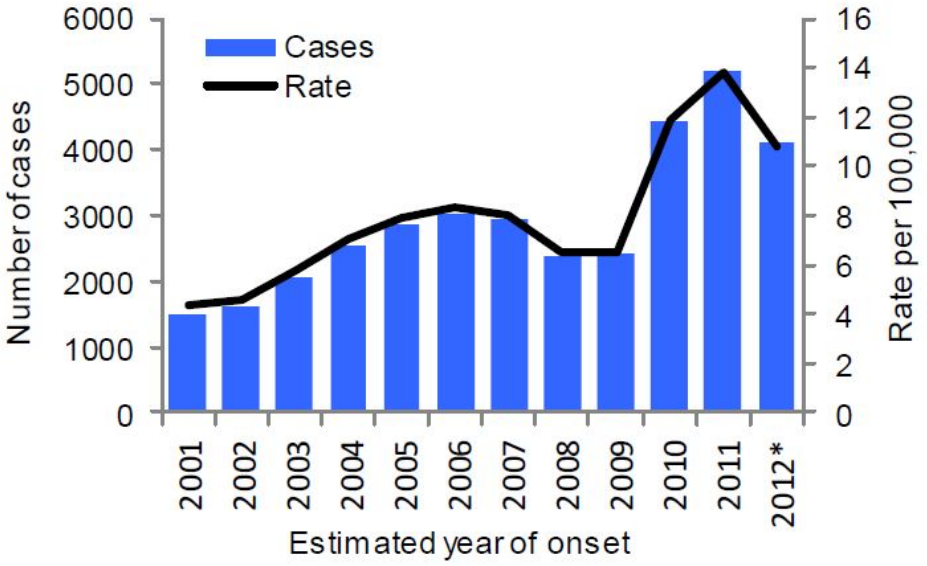


Figure 2. California coccidioidomycosis incidence rates by age and time period

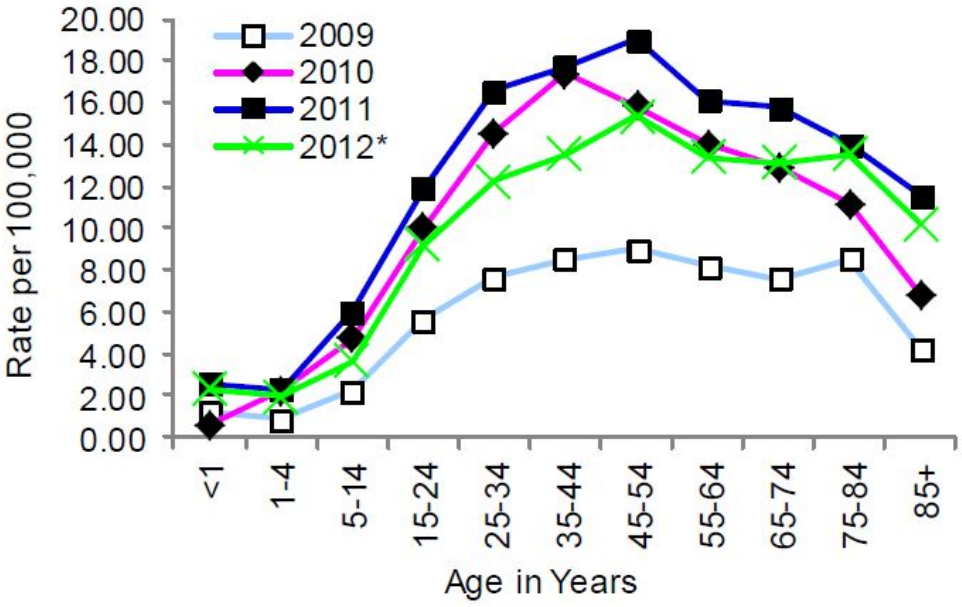
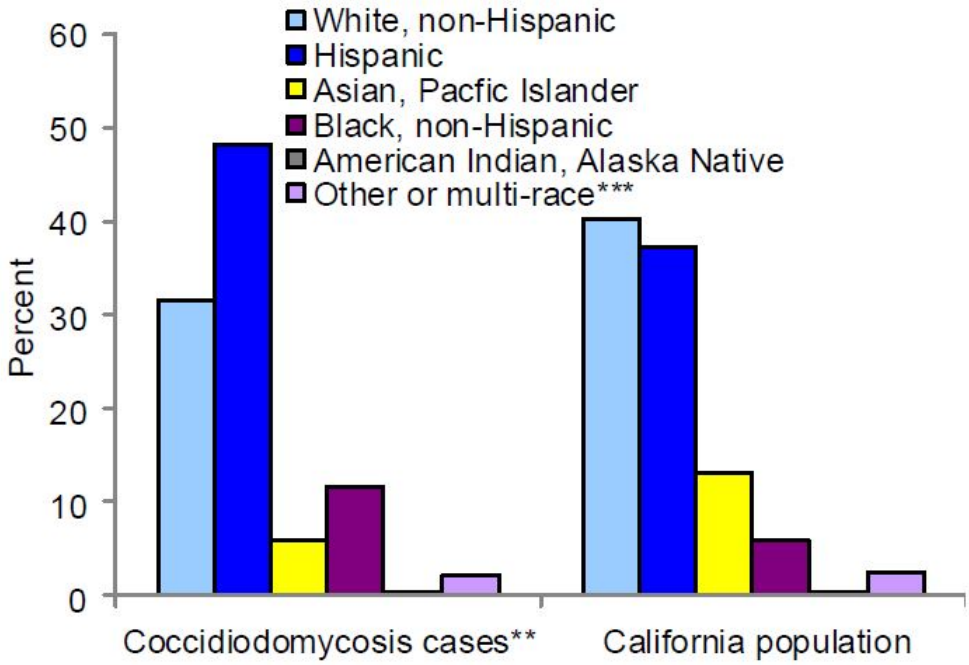


Figure 3. California coccidioidomycosis cases and population by race/ethnicity 2009—2012*



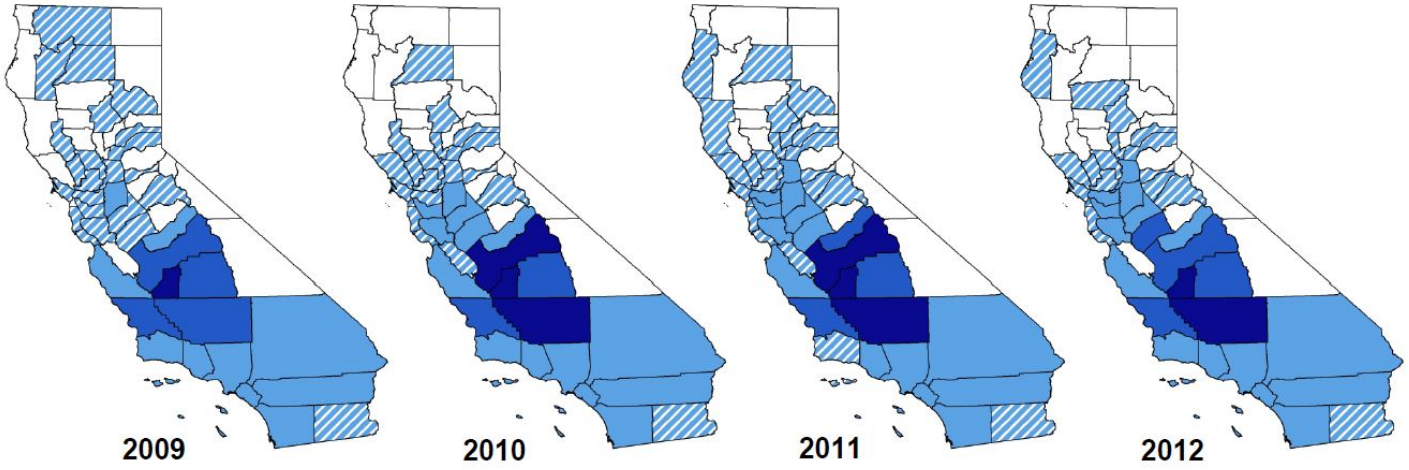
Notes for Figures 1-3

*2012 data reported as of May 4, 2013 and are provisional

**Unknowns were excluded

***includes cases who identified 'other' as their race and Californians ('population') who identified more than one race

Figure 4. California county-specific coccidioidomycosis incidence rates



Comment

Coccidioidomycosis annual incidence rates increased by 67.7 percent from 2009 to 2012. There was a peak in the incidence rate in 2011, which was the highest rate since the increasing trend from 2001. Age group, race/ethnicity, gender, and county epidemiologic profiles of incident cases with estimated onset dates from 2009 through 2012 remained fairly consistent with those with estimated onset dates from 2001 through 2008 as described previously.[4]

The causes of these increases are not well understood but climatic and environmental factors favorable to *Coccidioides* proliferation and airborne release, and increases in non-immune populations in endemic are- as may be contributing factors. The initiation of mandated laboratory reporting in 2010 could partly account for the increase in reported cases during 2011. However, some highly endemic counties were already using laboratory-based reporting. Coccidioidomycosis is highly endemic in the San Joaquin Valley and remains an important public health problem in California. There is currently no vaccine; efforts to develop a vaccine are ongoing. To decrease the risk of infection, persons living, working, or traveling in coccidioidomycosis endemic areas, especially those at increased risk for disseminated disease, should limit their exposure to out- door dust as much as possible. It is important that healthcare providers be alert for coccidioidomycosis among patients who live in or have traveled to endemic areas[1,2,3,6].

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Infectious Diseases

Branch Last updated

01/21/2014

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 1,537 cases of confirmed and probable cryptosporidiosis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 1.0 case per 100,000 Californians.
- Annual cryptosporidiosis incidence rates for each of the four years of 2009 through 2012 was higher than annual rates in the previous years of 2001 through 2008. The annual incidence rate peaked at 1.2 per 100,000 (463 case-patients) in 2009 and declined to 0.9 per 100,000 (351 case-patients) in 2012.
- Average annual cryptosporidiosis incidence rates during the surveillance period were highest among seniors 85 years and older (1.4 per 100,000), adults 35-44 years of age (1.2 per 100,000), and children 1–4 years of age (1.1 per 100,000).
- Incidence rates for males (1.03 per 100,000) and females (1.01 per 100,000) were similar. Females 65-74 years of age had the highest incidence rate (1.7 per 100,000), nearly twice that of males of the same age group.
- Cryptosporidiosis cases occurred more frequently in summer months of July and August (26 percent of all cases).
- During 2009-2012, one waterborne outbreak of cryptosporidiosis was reported with two confirmed case- patients exposed to a swimming pool in a rehabilitation facility.
- Decreasing human or animal fecal contamination of recreational or drinking water, education on hand hygiene and safe sexual practices, and targeted education of high risk groups likely offer the best opportunities for reducing cryptosporidiosis.

Background

Cryptosporidiosis is a worldwide diarrheal disease caused by intestinal infection with the microscopic parasite *Cryptosporidium.* The US Centers for Disease Control and Prevention (CDC) estimated that *Cryptosporidium* species cause 748,000 infections per year in the US[1,2]. Leading causes of *Cryptosporidium* infection include ingestion of untreated drinking water, contact with livestock, international travel to endemic areas, and contact with infected persons. In the US, it is the most frequently recognized cause of reported recreational water-associated outbreaks and is a recognized cause of drinking water and foodborne-associated outbreaks. Asymptomatic infections in people and animals are a frequent source of *Cryptosporidium* transmission[1].

Symptoms of cryptosporidiosis include diarrhea, stomach cramps, nausea, and dehydration, which can lead to weight loss. Illness begins 2 to 10 days after exposure and can last 1 to 2 weeks, although people with weakened immune systems may develop serious, chronic, and sometimes fatal illness[3]. On the other hand, some infections are asymptomatic.

This report describes the epidemiology of confirmed and probable cryptosporidiosis infections in California with estimated onset dates from January 1, 2009 through December 31, 2012 reported by April 2015. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to the *Technical Notes*[4]. The epidemiologic description of cryptosporidiosis for the 2001-2008 surveillance period can be found in the

Epidemiologic Summary of Cryptosporidiosis in California, 2001-2008[5].

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report suspected cases of cryptosporidiosis to their local health department within one working day of identification or immediately by telephone if an outbreak is suspected. Laboratories are also required to report laboratory testing results suggestive of *Cryptosporidia* infection to either the California Reportable Disease Information Exchange (CalREDIE) (via electronic laboratory reporting) or the local health department; reporting must occur within one working day after the health care provider has been notified.

Local health officers are required by regulation to report cases of cryptosporidiosis to CDPH, following the CDC/Council of State and Territorial Epidemiologists' (CSTE) surveillance case definition. The CDC/CSTE case definition was revised in 2009 and underwent minor revisions in 2011 and 2012 (For information on revisions see CSTE surveillance case definitions[6]). The 2012 case definitions for confirmed and probable cases are as follows:

Confirmed: a case diagnosed with *Cryptosporidium* spp. infection based on evidence of *Cryptosporidium* organisms or DNA in stool, intestinal fluid, tissue samples, biopsy specimens, or other biological sample by certain laboratory methods with a high positive predictive value (e.g., direct fluorescent antibody [DFA] test, polymerase chain reaction [PCR], enzyme immunoassay [EIA], OR light microscopy of stained specimens).

Probable: a case diagnosed with *Cryptosporidium* spp. only by antigen screening test method, such as immunochromatographic card/rapid card test; or a laboratory test of unknown method; OR a case of gastrointestinal illness characterized by diarrhea and one or more of the following: diarrhea duration of 72 hours or more, abdominal cramping, vomiting, or anorexia that is epidemiologically linked to a confirmed case of *Cryptosporidium* spp. infection[6].

Epidemiology of cryptosporidiosis in California

CDPH received reports of 1,537 cases of confirmed and probable cryptosporidiosis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 1.0 per 100,000 Californians. Annual cryptosporidiosis incidence rates for each of the four years of 2009 through 2012 were higher than annual rates in the previous years of 2001 through 2008. The annual incidence rate peaked at 1.2 per 100,000 (463 case-patients) in 2009 and declined to 0.9 per 100,000 (351 case-patients) in 2012 [Figure 1]. During the surveillance period, 2 cases (0.1 percent) were reported to have died with cryptosporidiosis.

During 2009-2012, the average annual cryptosporidiosis incidence rate was highest among seniors 85 years and older (1.4 per 100,000), with an incidence rate 2.8 times that of the 5-14 years age group (0.5 per 100,000), the group with the lowest rate. Children 1–4 (1.1 per 100,000) and adults 35-44 (1.2 per 100,000) years of age also had higher rates compared to other age groups.

Incidence rates for males (1.03 per 100,000) and females (1.01 per 100,000) were similar. Females 65-74 years of age had the highest incidence rate (1.7 per 100,000), nearly twice that of males of the same age group. Cryptosporidiosis cases occurred more frequently in warmer months, with July and August accounting for 26 percent of all cases. This seasonal pattern was generally evident among all age groups and genders. Incidence rates by race/ethnicity were not calculated due to the substantial portion of

missing data (47 percent). However, cryptosporidiosis cases with complete data indicate the proportion of cases in which White, non-Hispanic race/ethnicity was reported was larger than the proportion of White, non-Hispanic residents in California [Figure 3].

Average incidence rates for the surveillance period were 2.6 times higher in Northern California (1.6 per 100,000) than in Southern California (0.6 per 100,000). From 2009 to 2012, cryptosporidiosis incidence rates decreased by 40 percent (from 2.2 to 1.3 per 100,000) in Northern California but increased by 19 percent (from 0.55 to 0.65 per 100,000) in Southern California.

From 2009 through 2012, CDPH received a report of one waterborne outbreak of cryptosporidiosis involving two confirmed case-patients exposed to a swimming pool in a rehabilitation facility.

Figure 1. California cryptosporidiosis case counts and incidence rates, 2001-2012

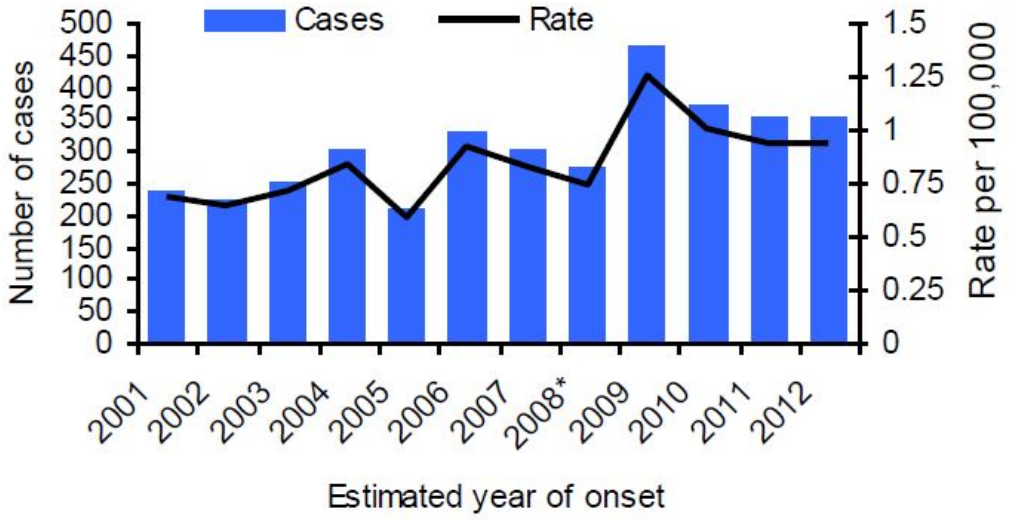


Figure 2. California cryptosporidiosis incidence rates by age and year, 2009-2012

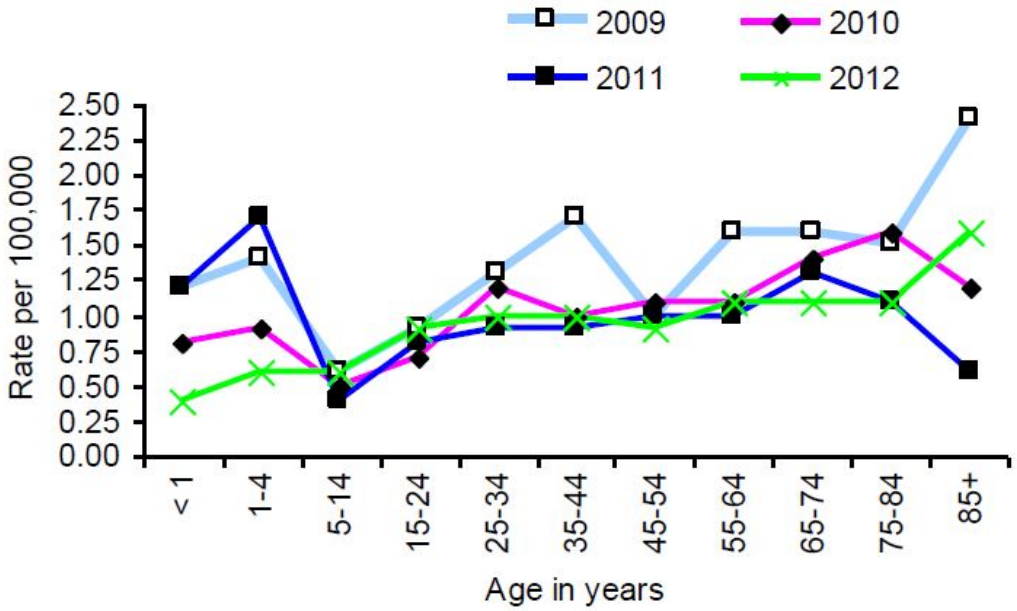
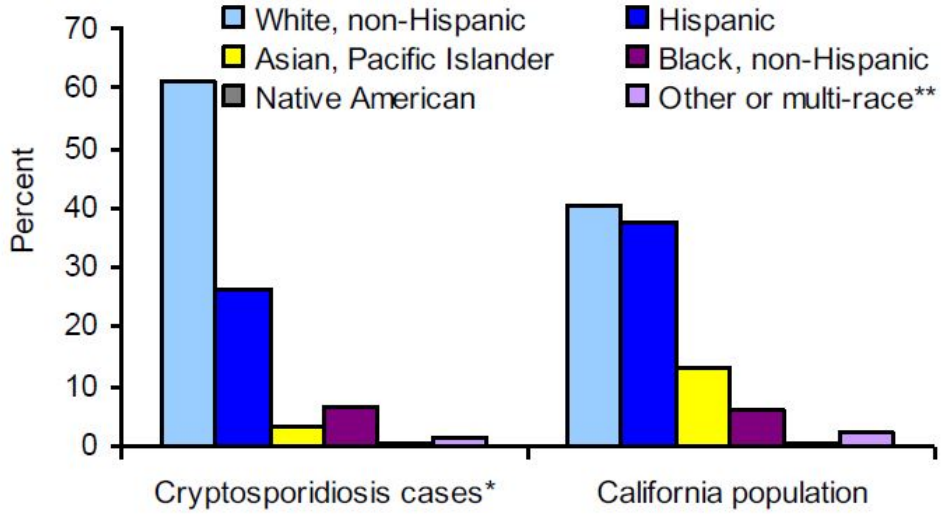


Figure 3. California cryptosporidiosis cases and population by race/ethnicity*, 2009- 2012

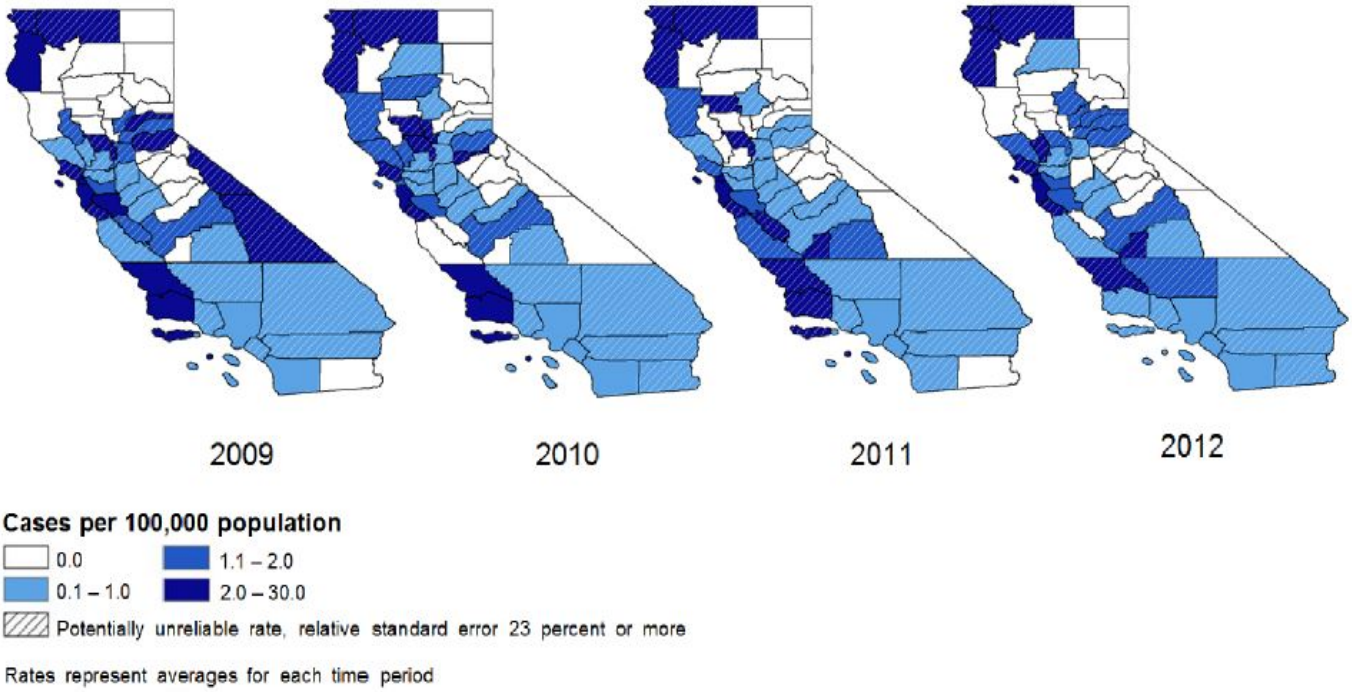


Notes for Figures 1-3

*Unknowns were excluded

**Includes cases who identified 'other' as their race and Californians ('population') who identified more than one race

Figure 4. California county-specific cryptosporidiosis incidence rates



Comment

Annual cryptosporidiosis incidence rates for each of the four years of 2009 through 2012 were higher than annual rates in the previous years of 2001-2008, with a pick-up in 2009 followed by a modest decrease thereafter. An increasing number of tests with high positive-predictive value are available, and since 2012 these tests have been used to distinguish confirmed and probable cases. However, probable and confirmed cases were combined for the purpose of this report, so these changes in testing and case definitions are less likely to affect the apparent trends among these data.

Similar to national trends, cryptosporidiosis incidence rates were highest among young children and seniors. Cases occurred more frequently during warmer months and may be associated with recreational water exposures[1]. Age group, race/ethnicity, gender, and county epidemiologic profiles of incident cases remained fairly consistent between those with onset dates from 2009 through 2012 and those with onset between 2001 through 2008[4].

Cryptosporidium presents special challenges to public health because of its low infectious dose combined with its resistance to chlorine disinfection. Decreasing human or animal fecal contamination of recreational or drinking water, information regarding hand hygiene and safe sexual practices, and targeted education of high-risk groups likely offer the best opportunities for reducing cryptosporidiosis.

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Last updated: 1/25/2016

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Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 43 confirmed and 61 probable cases of cysticercosis with estimated dates of illness onset in 2009 through 2012. This corresponds to an incidence rate of 0.07 per 100,000 population per year.
- Cysticercosis incidence decreased by 33.3 percent from 2009 (34 cases; 0.09 per 100,000 population) to 2012 (24 cases; 0.06 per 100,000 population).
- Incidence rates during the surveillance period were highest among persons 25 to 34 years of age (0.14 per 100,000 population per year) and 35 to 44 years of age (0.10 per 100,000 population per year).
- Hispanic ethnicity was reported more frequently (89.7 percent) for cysticercosis case-patients than expected from the proportion of Hispanics in the general California population (37.8 percent).
- Identification of a patient with cysticercosis warrants evaluation of other household members for both cysticercosis and taeniasis to rule out possible local transmission.

Background

Cysticercosis is one of two forms of infection caused by the tapeworm, *Taenia solium*. *Taenia solium* is a parasite of both humans and pigs, and requires both species to complete its life cycle. People who eat pork containing *T. solium* cysts (cysticerci) develop an intestinal infection with the adult tapeworm called taeniasis. Persons with taeniasis pass tapeworm eggs in their feces. When these eggs are consumed by a pig, the eggs hatch in the intestine, releasing larvae that migrate and encyst in tissues. Cysticercosis occurs when people ingest tapeworm eggs directly, and, as in pigs, the larvae migrate out of the intestine and into muscle and other organs and tissues. Neurocysticercosis occurs when larvae form cysticerci in the brain, causing headache, seizures, signs of intracranial hypertension, or psychiatric disturbances.[1, 2]

Worldwide, *T. solium* cysticercosis and taeniasis are endemic in developing countries in Latin America, sub- Saharan Africa, and Asia. In the United States, regulations and food animal husbandry practices have virtually eliminated the parasite from commercial pork. Neurocysticercosis cases diagnosed in the U.S. have been mainly among immigrants from Mexico and Latin America; however, occasional disease acquired within the U.S., including in California, has been reported.[3]

We describe here the epidemiology of confirmed and probable cysticercosis cases in California with estimated onset dates from January 2009 through December 2012 reported to CDPH by March 2015. Data for 2012 are provisional and may differ from data in future publications. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes.[4]

California reporting requirements and surveillance case definition

During the surveillance period, a confirmed case was defined as clinically compatible illness with at least one of the following confirmatory laboratory/imaging criteria:

- *Taenia solium* identified by microscopic examination in cysticerci excised from tissues; or
- identification of cysticerci by computerized tomography (CT) scan, magnetic resonance imaging (MRI), or X-ray; and positive result on Centers for Disease Control and Prevention (CDC) immunoblot assay

A probable case was defined as clinically compatible illness with at least one of the following supportive laboratory/imaging criteria:

- identification of calcified cystic lesions in tissue by CT scan, MRI, or X-ray; or
- positive result on CDC immunoblot assay

Epidemiology of cysticercosis in California

CDPH received reports of 43 confirmed and 61 probable cases of cysticercosis with estimated illness onset dates from 2009 through 2012. This corresponds to an incidence rate of 0.07 per 100,000 population per year.

Reported cysticercosis incidence decreased by 33.3 percent from 2009 (34 cases; 0.09 per 100,000 population) to 2012 (24 cases; 0.06 per 100,000 population) [Figure 1]. During the surveillance period, two (1.9 percent) fatal cases were reported.

Reported cysticercosis incidence rates during the by estimated year of illness onset, surveillance period were highest among persons 25 to 34 years of age (0.14 per 100,000 population per year) and 35 to 44 years of age (0.10 per 100,000 population per year) [Figure 2]. The ratio of male to female case-patients was 1.6:1.0. Cysticercosis cases with complete information on race/ethnicity (93.3 percent of all cases) reported Hispanic (89.7 percent) ethnicity more frequently than would be expected based on the proportion of Hispanics (37.8) in the California general population [Figure 3].

The incidence for cysticercosis was 1.3 times higher in Southern California (0.08 per 100,000 population per year) than in Northern California (0.06 per 100,000 population per year). However; the highest incidence during the surveillance period was in the Central Coast (0.14 per 100,000 population per year) [Figure 4].

Figure 1. California cysticercosis case counts and incidence rates by estimated year of illness onset, 2001-2012*

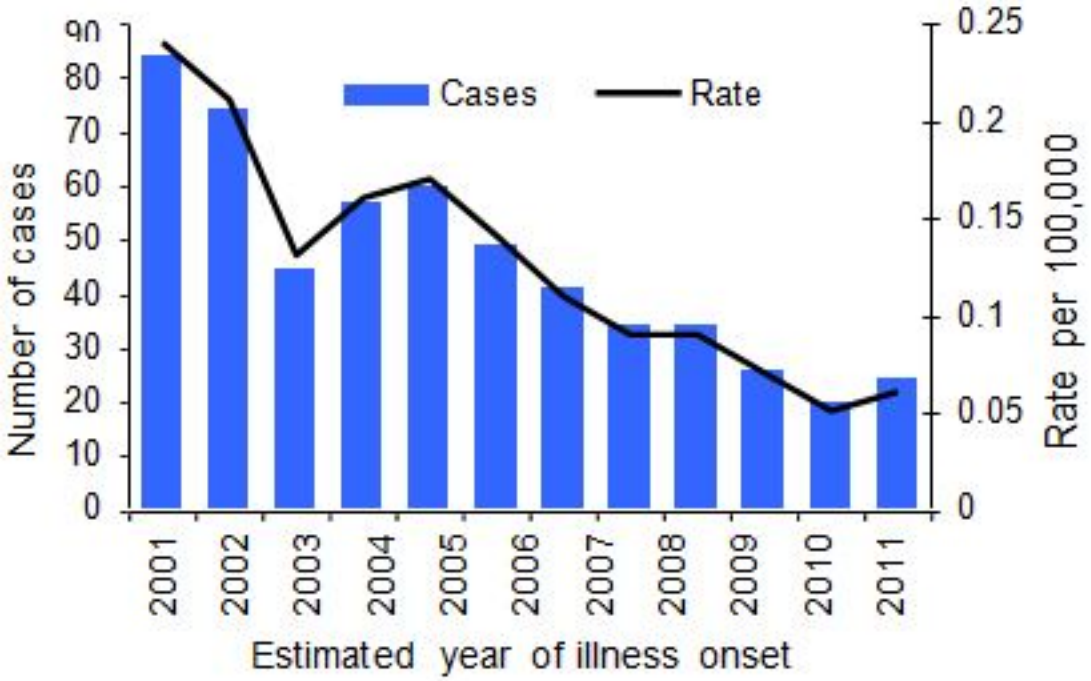


Figure 2. California cysticercosis incidence rates by age groups, 2009-2012*

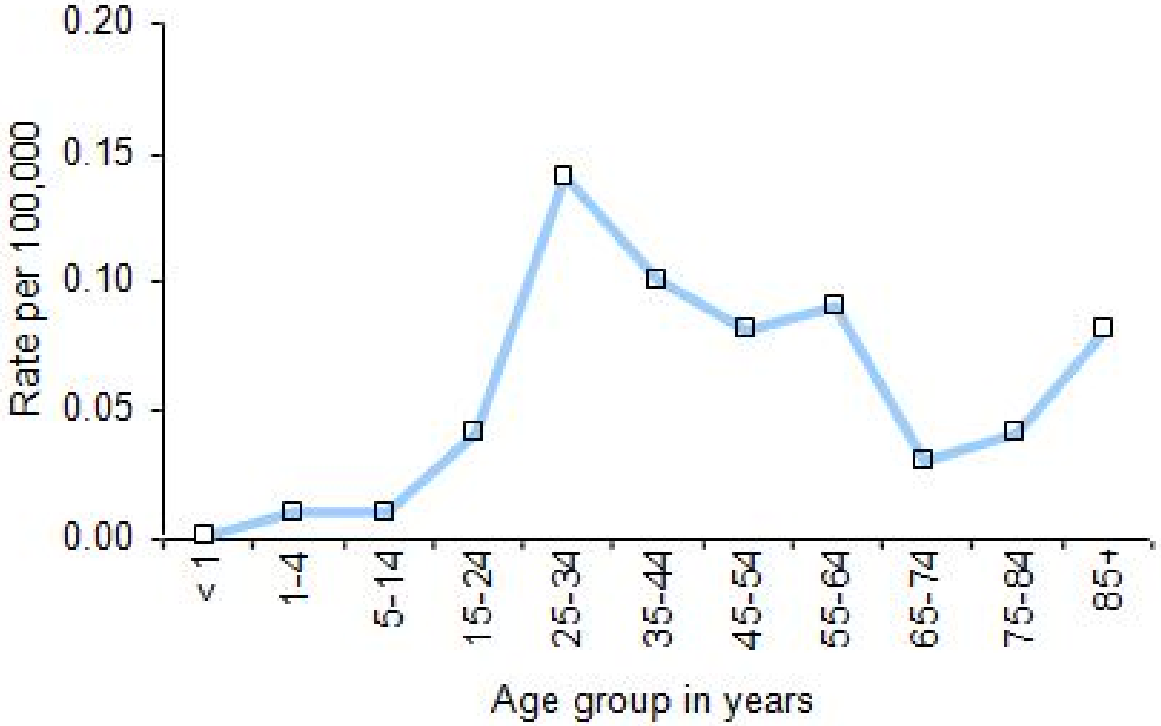
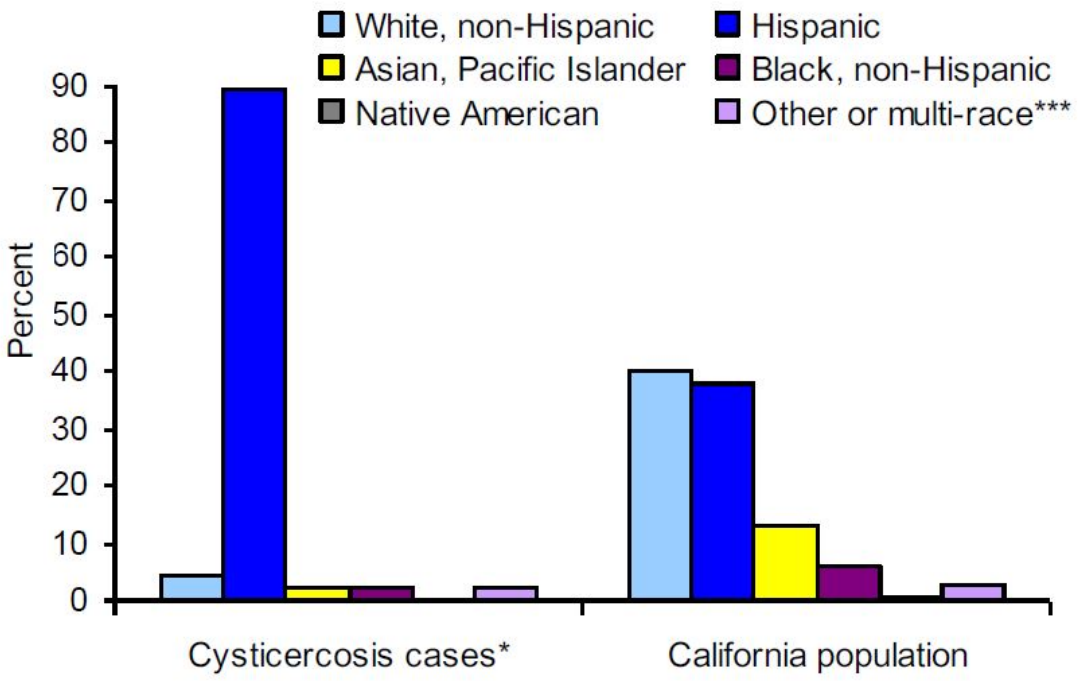


Figure 3. California cysticercosis cases and population by race/ethnicity, 2009-2012*



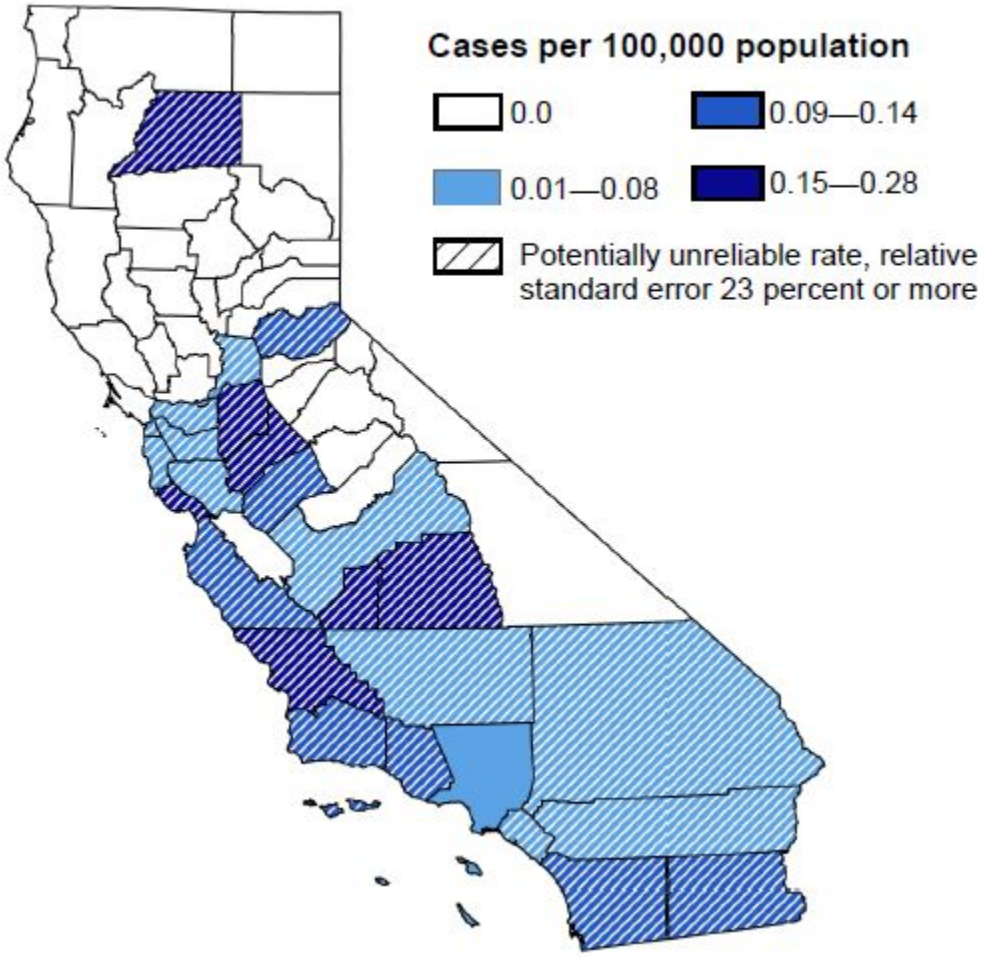
Notes for Figures 1-4

*2012 data are provisional

** Unknowns were excluded

*** Includes cases who identified "other" as their race and Californians ('population') who identified more than one race

Figure 4. California county-specific cysticercosis incidence rates, 2009-2012*



Comment

Overall, reported incidence of cysticercosis in California has decreased since 2001. The higher frequency of the disease among persons of Hispanic ethnicity is consistent with the historical finding of this disease being more common among immigrants from Latin America. Because cysticercosis is a result of ingestion of *T. solium* eggs from the feces of an infected human, washing hands with soap and water after using the toilet and before handling food is important in the prevention of this disease. Persons with taeniasis should refrain from preparing food until treatment has cleared their infection.

Because clinical manifestations of cysticercosis depend on both the number of encysted larvae and the particular tissues affected, infection may remain subclinical for months to years. Calcified cysts may be recognized only as incidental findings on imaging studies. Because of the potentially protracted period between exposure and diagnosis the circumstances leading to infection for a given case-patient are frequently difficult to identify and past the point of purposeful public health intervention. Nevertheless, identification of a patient with cysticercosis warrants evaluation of other household members for both cysticercosis and taeniasis to identify persons who may have acquired infection from, or been exposed to the same source as, known household case-patients.

For travelers visiting endemic countries, intestinal infection with *T. solium* (taeniasis) and cysticercosis can be prevented by avoiding pork that has not been thoroughly cooked and foods that may be contaminated with human feces.

References and resources

[1]CDC Parasites - Cysticercosis: (http://www.cdc.gov/parasites/cysticercosis/index.html)

[2]CDPH Cysticercosis (Pork Tapeworm, Taeniasis): (http://www.cdph.ca.gov/HealthInfo/discond/Pages/Cysticercosis.aspx)

[3]Cantey PT, Coyle CM, Sorvillo FJ, Wilkins PP, Starr MC, Nash TE. Neglected parasitic infections in the United States: Cysticercosis. Am J Trop Med Hyg 2014; 90:805-809.

[4]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001 - 2008 and 2009 - 2012: Technical Notes (http://www.cdph.ca.gov/programs/sss/Documents/TechnicalNotes01-08and09-12.pdf)

Last updated: 10/01/2015

Prepared by Farzaneh Tabnak, Alyssa Nguyen, Edward Powers, Curtis Fritz, Lennox Din, Pouya Khankhanian, Duc J Vugia, Infectious Diseases Branch

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 6 confirmed cases of foodborne botulism with estimated illness onset dates from 2009 through 2012.
- During the surveillance period, 1 (16.7 percent) case-patient was reported to have died with foodborne botulism.
- The ratio of male to female cases was 2.0:1.0.
- From 2009 through 2012, CDPH received re- ports of one "probable" case in a patient who shared a meal with a person who was laboratory confirmed. Two additional patients were reported as "probable" foodborne botulism cases as they shared a meal and both developed clinical findings consistent with botulism but neither was laboratory confirmed and other possible causes of their symptoms could not be ruled out.
- Ensuring appropriate practices in food preparation and preservation and public education about botulism may provide the best opportunities to prevent and control foodborne botulism.

Background

Clostridium botulinum neurotoxin is a rare but important food intoxicant in the United States (US). This potent toxin is produced by *C. botulinum,* an anaerobic, spore-forming bacterium that is ubiquitous in the environment. Foodborne botulism follows ingestion of preformed toxin in foods contaminated by *C. botulinum.* Despite the presence of bacteria and toxin in the stools of infected persons, person-to- person transmission has not been documented. *C. botulinum* toxin is listed among the Centers for Dis- ease Control and Prevention (CDC) category A bioterrorism agents.[1]

Botulism is a neuroparalytic illness. Early symptoms may include double/blurred vision, drooping eyelids, slurred speech, difficulty swallowing, dry mouth, and muscle weakness. Neurologic symptoms generally begin 12 to 36 hours after ingestion of toxin and can progress to a symmetric, descending flaccid paralysis that begins in the cranial nerves.

Untreated, botulism can progress to respiratory paralysis and death. If administered early in the course of illness, botulism antitoxin can stop the progression of, but cannot reverse paralysis. Antitoxin is available exclusively from public health authorities.

We describe here the epidemiology of confirmed food- borne botulism case-patients with estimated illness onset from 2009 through 2012 in California. Case-patients were reported as of October 22, 2014. The epidemiologic description of food botulism for the 2001-2008 surveillance period was previously published in the Epidemiologic Summary for Food Botulism in California, 2001- 2008.[2] For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes.[3] Because of the small numbers of cases, we did not calculate incidence rates.

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report

suspected cases of botulism to their local health department immediately by telephone. In the event that a commercial food product is suspected as the source, special instructions will be given by CDPH. Laboratories must immediately communicate by telephone with the CDPH Microbial Diseases Laboratory for instruction whenever a specimen for laboratory diagnosis of suspected botulism is received. Laboratories must report to the local health department when laboratory testing yields evidence suggestive of *C. botulinum;* notification must occur within one hour after the health care provider has been notified.

California regulations require local health departments to immediately report to CDPH cases of foodborne botulism by telephone. CDPH officially counted cases that satisfied the CDC surveillance case definition. CDC defined a confirmed case of foodborne botulism as one with clinically compatible illness and either (i) laboratory confirmation including detection of botulinum toxin in serum, stool, or patient's food or isolation of *C. botulinum* from stool, or (ii) a history of consuming the same food as persons with laboratory-confirmed botulism. A probable case was one with clinically compatible illness and an epidemiologic exposure (e.g., ingestion of a home-canned food within the previous 48 hours). California regulations defined one case of botulism as a foodborne outbreak if laboratory studies identified the causative agent in food.

Epidemiology of foodborne botulism in California

CDPH received reports of 6 cases of confirmed food- borne botulism with estimated illness onset dates from 2009 through 2012. Annual foodborne botulism case counts showed a decrease from 2009 to 2012 [Figure 1]. During the surveillance period, 1 (16.7 percent) case-patient was reported to have died with foodborne botulism.

During the surveillance period, the number of foodborne botulism cases was highest among persons 45-54 and over 85 years of age [Figure 2]. There were no cases reported for individuals under 45 years of age. The ratio of male to female cases was 2.0:1.0. Foodborne botulism cases reported White, non-Hispanic race/ethnicity more frequently than would be expected based on the overall demographic profile of California. [Figure 3]. Six counties reported at least 1 confirmed case-patients during the surveillance period.

From 2009 through 2012, CDPH received reports of one “probable” case in a patient who shared a meal with a person who was laboratory confirmed. Two additional patients were reported as “probable” foodborne botulism cases as they shared a meal and both developed clinical findings consistent with botulism but neither was laboratory confirmed and other possible causes of their symptoms could not be ruled out.

Figure 1. California foodborne botulism case counts, 2001-2012

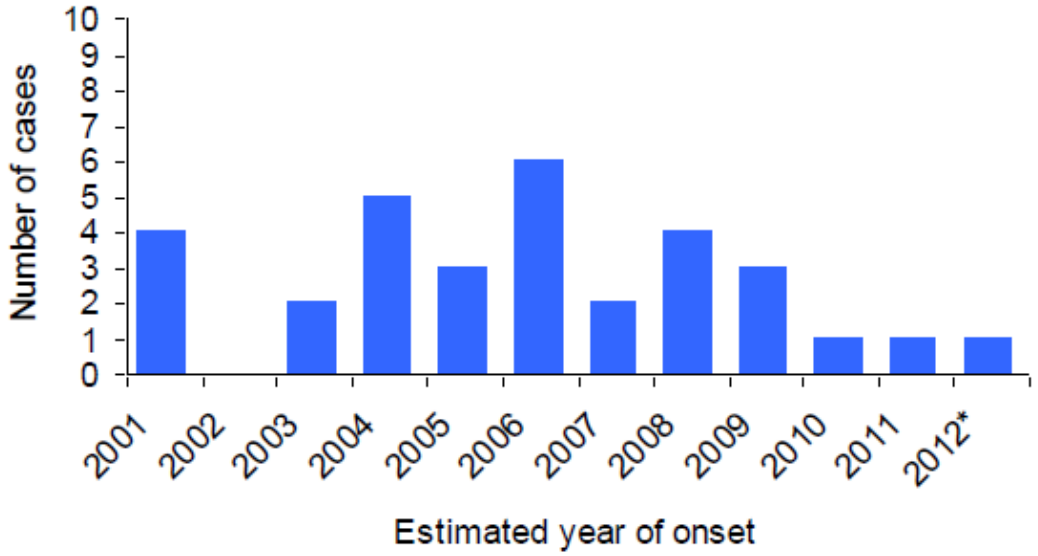


Figure 2. California foodborne botulism cases by age, 2001-2012

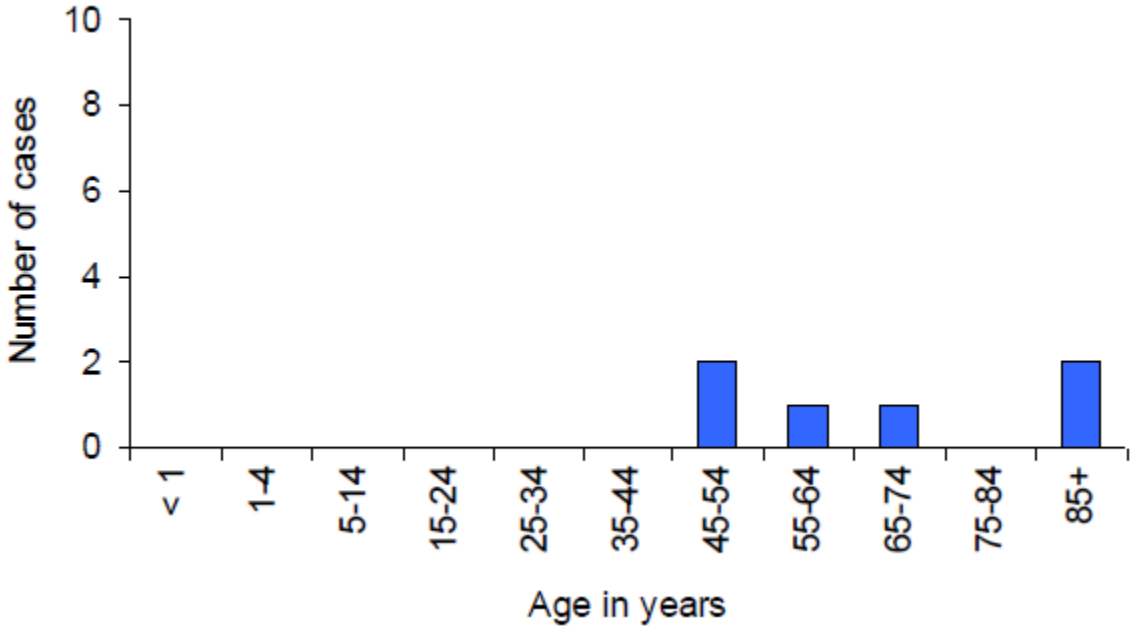
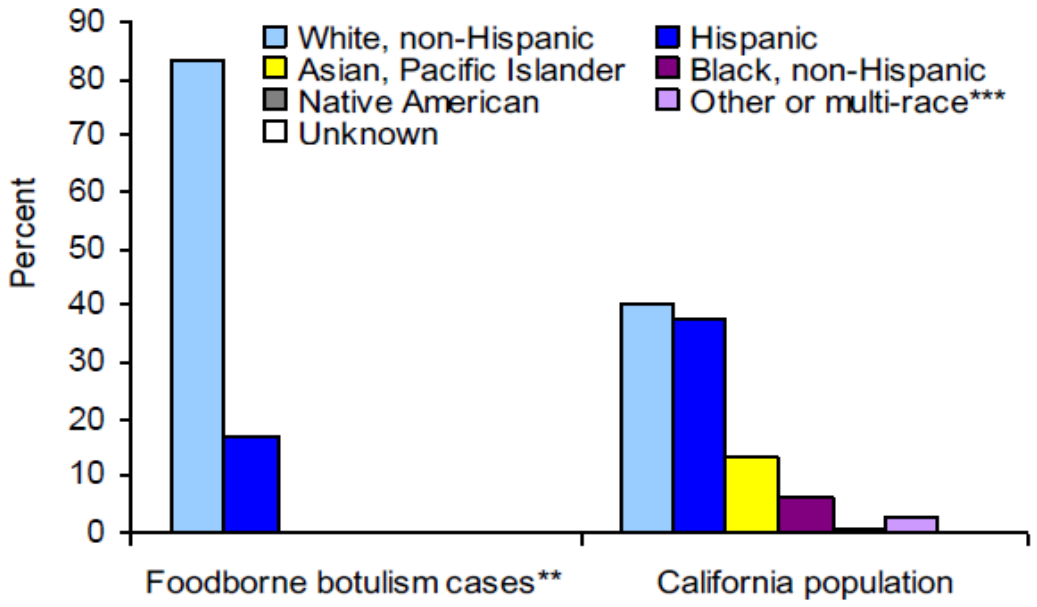


Figure 3. California foodborne botulism cases and population by race/ethnicity, 2009-2012



Notes for Figures 1-3

**Unknowns were excluded

***Includes cases who identified 'other' as their race and Californians ('population') who identified more than one race

Comment

Although foodborne botulism remained a rare occurrence in California, each case represented a medical and public health emergency. Surveillance and response to foodborne botulism is intensive because the contaminated food item must be identified and removed from distribution (whether it is commercial or homemade) without delay. Foodborne botulism has often been from home-canned foods.[4, 5] Patients having symptoms of foodborne botulism, should immediately seek medical care. USDA has information and guidelines on canning foods at home.[6] Ensuring appropriate practices in food preparation and preservation and public education about botulism may provide the best opportunities to prevent and control foodborne botulism.

Prepared by Farzaneh Tabnak, Debra Gilliss, Romana Teodora Dumitras, Duc J Vugia, Infectious Diseases Branch

Last updated 10/30/2014

References and resources

1 CDC botulism website—General Information and Resources (http://www.cdc.gov/nczved/divisions/dfbmd/diseases/botulism/ consumers.html)

2 Epidemiologic Summary of Foodborne Botulism in California, 2001 - 2008 (http://www.cdph.ca.gov/programs/sss/Documents/Epi-Summaries-CA- 2001-2008-083111.pdf#page=25)

3 Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001 - 2008 and 2009 - 2012: Technical Notes (http://www.cdph.ca.gov/programs/sss/Documents/TechnicalNotes01- 08and09-12.pdf)

4 CDPH botulism website (http://www.cdph.ca.gov/HealthInfo/discond/Pages/Botulism.aspx)

5 CDC Home Canning and Botulism (http://www.cdc.gov/features/homecanning/)

6 National Center for Home Food Preservation —USDA Complete Guide to Home Canning, 2009 revision (http://nchfp.uga.edu/publications/publications_usda.html)

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 7,080 cases of giardiasis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 4.73 cases per 100,000 Californians.
- Giardiasis incidence rates were stable during the 2009-2012 surveillance period. Incidence rates ranged from 4.59 per 100,000 (1,724 cases) in 2011 to 4.83 per 100,000 (1,791 cases) in 2009.
- Average annual giardiasis incidence rates during the surveillance period were highest among children 1 to 4 years of age (9.75 per 100,000) and adults 35 to 74 years of age (5.08 per 100,000).
- No outbreaks of giardiasis were reported to have occurred during 2009 through 2012.
- To prevent and control giardiasis, people should practice good hygiene, particularly at childcare facilities, avoid drinking or swallowing untreated or poorly treated water or use ice made from such water, avoid eating raw or uncooked foods when traveling in countries with poor food and water treatment, and prevent contact and contamination with feces during sex.

Background

Giardiasis is a worldwide diarrheal disease caused by the parasite *Giardia intestinalis* (a.k.a. *Giardia lamblia* or *Giardia duodenalis*). In the United States, giardiasis is the most frequently diagnosed intestinal parasitic disease[1], with an estimated 1.2 million cases occurring annually[2]. In recent years, the incidence rate of giardiasis cases reported to the US Centers for Disease Control and Prevention (CDC) appears to be decreasing[3]. *Giardia* may be found in water, soil, food, or on surfaces that have been contaminated with feces from infected persons or animals. People become infected after accidentally swallowing *Giardia* by drinking untreated contaminated surface or well water, by eating contaminated foods, or by having contact with an infected person or contaminated surfaces.

Symptoms of giardiasis include diarrhea, gas, stomach cramps, and dehydration which can lead to weight loss. Illness begins 1 to 3 weeks after exposure and can last 2 to 6 weeks.1 Some infections, however, are asymptomatic.

This report describes the epidemiology of confirmed and probable giardiasis infections in California with estimated symptom onset dates from January 1, 2009 through December 31, 2012 reported by December 2014. Data for 2012 are provisional and may differ from data in future publications. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to the Technical Notes[4]. The epidemiologic description of giardiasis for the 2001-2008 surveillance period can be found in the Epidemiologic Summary of Giardiasis in California, 2001-2008[5].

California reporting requirements and surveillance case definitions

California Code of Regulations, Title 17, requires health care providers to report

suspected cases of giardiasis to their local health department within seven calendar days of identification or immediately by telephone if an outbreak is suspected. Laboratories are also required to report laboratory testing results suggestive of *Giardia* infection to either the California Reportable Disease Information Exchange (CalREDIE) (via electronic laboratory reporting) or the local health department; reporting must occur within one working day after the health care provider has been notified.

Local health officers are required by regulation to report to CDPH cases of giardiasis. CDPH counted cases that satisfied the CDC/Council of State and Territorial Epidemiolgists' surveillance case definition of a confirmed or probable case. Through 2010, CDC defined a confirmed case as one with laboratory detection of *Giardia intestinalis* organisms, antigen, or DNA in stool, intestinal fluid, tissue samples, biopsy specimens or other biological sample. Beginning in 2011, laboratory detection of *Giardia intestinalis* along with clinically-compatible illness (as characterized by gastrointestinal symptoms such as diarrhea, abdominal cramps, bloating, weight loss, or malabsorption) was necessary to classify a case as confirmed. During the surveillance period, a probable case was defined as one with clinically-compatible illness and an established epidemiological link to a laboratory-confirmed case[6].

Epidemiology of giardiasis in California

CDPH received reports of 7,080 cases of giardiasis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 4.73 cases per 100,000 Californians. Following a decline in giardiasis incidence rates during the 2001-2008 surveillance period, rates were stable during the 2009-2012 surveillance period. Incidence rates ranged from 4.59 per 100,000 (1,724 cases) in 2011 to 4.83 per 100,000 (1,791 cases) in 2009 [Figure 1].

Average annual giardiasis incidence rates for the surveillance period were highest among children 1 to 4 years of age (9.75 per 100,000) and adults 35 to 74 years of age (5.08 per 100,000, not shown) [Figure 2]. The ratio of male to female cases was 1.7:1.0. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (47.6 percent). However, giardiasis cases with complete data reported White non-Hispanic race/ethnicity more frequently than would be expected and Hispanic ethnicity less frequently than would be expected based on the overall demographic profile of California [Figure 3].

County-specific incidence rates during the surveillance period ranged from 0 to 35.86 per 100,000 [Figure 4]. Average annual incidence rates for the surveillance period were 1.5 times higher in Northern California (5.77 per 100,000) than in Southern California (3.93 per 100,000). San Diego (8.08 per 100,000), Bay Area (8.01 per 100,000), and Far North (5.87 per 100,000) regions reported the highest average annual incidence rates during the surveillance period.

No waterborne or foodborne outbreaks of giardiasis were reported to CDPH to have occurred during the 2009-2012 surveillance period.

Figure 1. California giardiasis case counts and incidence rates by estimated year of illness onset.

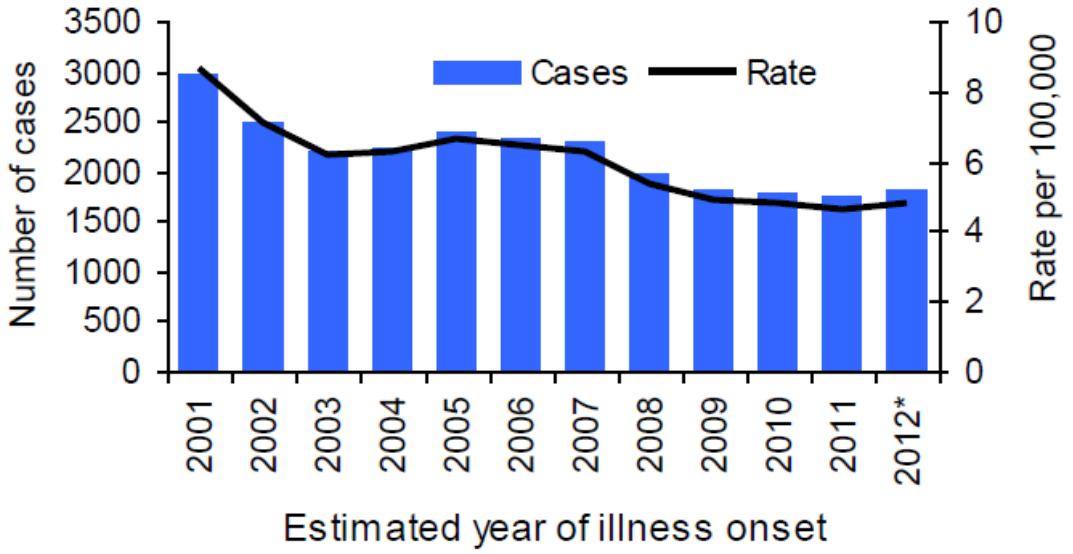


Figure 2. California giardiasis incidence rates by age group and estimated year of illness onset.

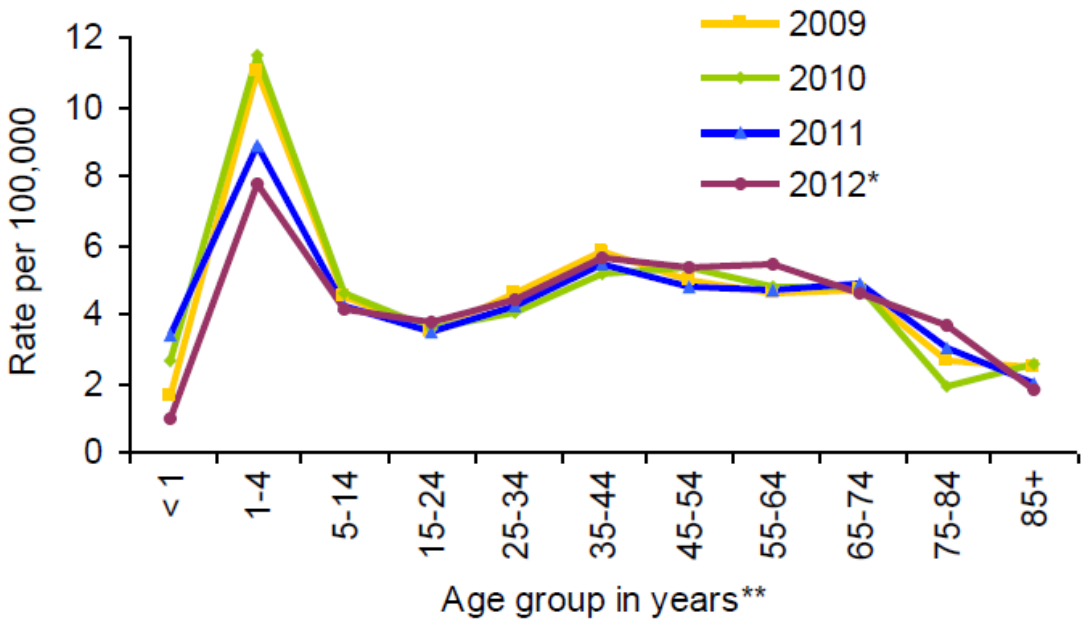
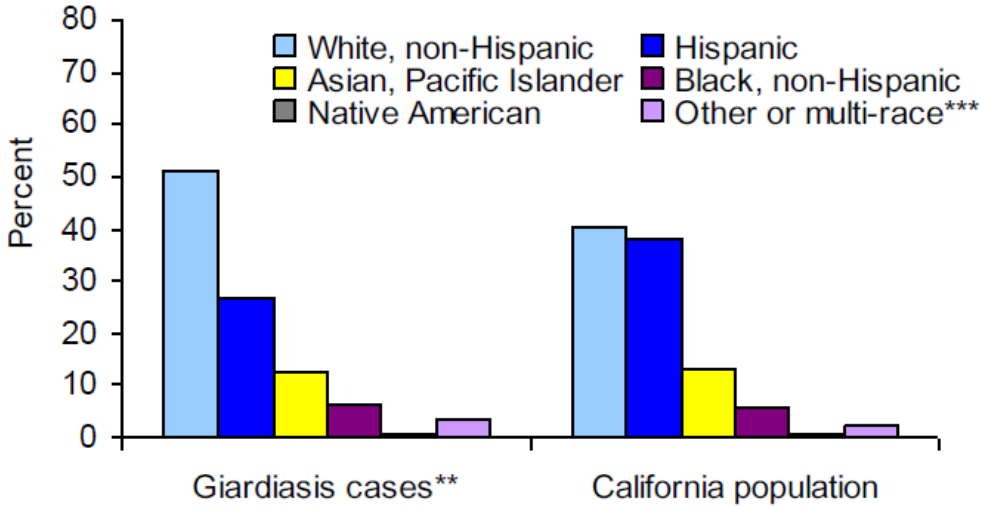


Figure 3. California giardiasis cases and population by race/ethnicity, 2009-2012



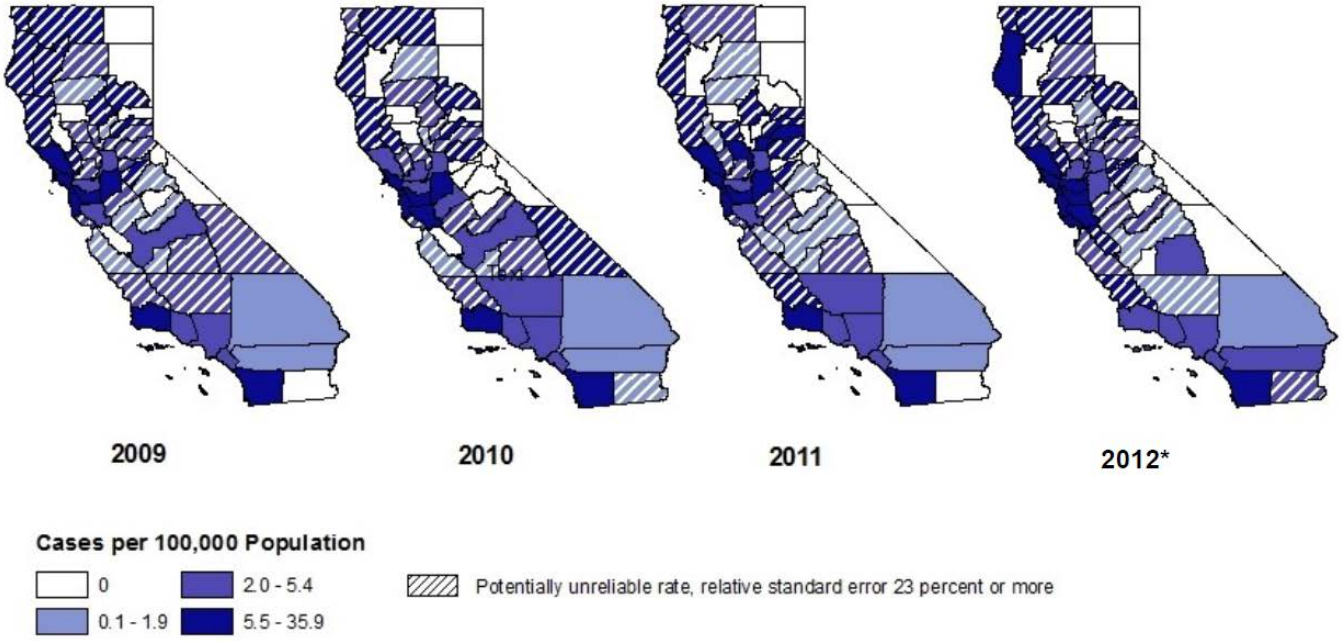
Notes for Figures 1-4

*2012 data are provisional

**Unknowns were excluded

***Includes cases who identified 'other' as their race and Californians ('population') who identified more than one race

Figure 4. California county-specific giardiasis incidence rates by estimated year of illness onset



Comment

Incidence rates of reported giardiasis infection among Californians were stable from 2009 through 2012. *Giardia* infections are often not diagnosed and not reported, so rates may be underestimated[2,3].

The age distribution of reported cases incident in California during 2009-2012 remained fairly consistent with that of 2001-2008[5]. Comparable to national trends, California children 1 to 4 years of age experienced the highest rates of giardiasis[3].

To prevent and control infection with *Giardia*, people should practice good hygiene, particularly at child- care facilities, avoid drinking or swallowing untreated or poorly treated water or use ice made from such water, avoid eating raw or uncooked foods when traveling in countries with poor food and water treatment, and prevent contact and contamination with feces during sex.[7]

References and resources

[1] Centers for Disease Control and Prevention. Parasites – *Giardia*. http://www.cdc.gov/parasites/giardia/

[2] Scallan E, Hoekstra RM, Angulo FJ et al. Foodborne illness acquired in the United States—major pathogens. Emerg Infect Dis. 2011 Jan; 17(1):7-15.

[3] Painter JE, Gargano JW, Collier SA et al. Giardiasis surveillance -- United States, 2011-2012. MMWR Surveill Summ. 2015 May 1;64 Suppl 3:15-25.

[4] Epidemiologic Summaries of Selected General Communicable Diseases in California, 2009-2012: Technical Notes.

https://archive.cdph.ca.gov/programs/sss/Documents/ TechnicalNotes01-08and09-12.pdf

[5] Epidemiological Summaries of Selected General Communicable Diseases in California, 2001-2008: Giardiasis. https://archive.cdph.ca.gov/data/statistics/Pages/EpiSummariesCDsCA-01-08.aspx

[6] National Notifiable Diseases Surveillance System, Case Definitions, Giardiasis. Centers for Disease Control and Prevention, 2015. http://wwwn.cdc.gov/nndss/conditions/giardiasis/

[7] Centers for Disease and Prevention. Parasites – *Giardia*. Prevention & Control: General Public. http://www.cdc.gov/parasites/giardia/prevention-control-general- public.html

Last updated 9/18/2015

Prepared by Kirsten Knutson, Farzaneh Tabnak, and Duc J Vugia, Infectious Diseases Branch

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 15 cases of hantavirus pulmonary syndrome (HPS) with onset dates from 2009 through 2012. The number of cases was highest in 2012 (8 cases).
- During summer 2012, 10 hantavirus infections were identified among overnight visitors to Yosemite National Park, including 8 California residents--6 with HPS and 2 with milder illnesses.
- During the surveillance period, 3 (20.0 percent) case-patients were reported to have died with HPS.
- The median age among HPS case-patients was 42 years (range: 13 to 61 years) and the highest number of cases occurred among persons 35 to 44 years of age (6 cases).
- HPS case-patients reported White non-Hispanic (53.3 percent), Hispanic (20.0 percent), Asian (20.0 percent) and non-Hispanic unknown (6.7 percent) race/ethnicities. The ratio of male to female case-patients was 2:1.
- The primary strategy for reducing the risk of hantavirus exposure is to avoid contact with rodents and their excreta.

Background

Hantavirus pulmonary syndrome (HPS) is a rodent- borne viral disease that was first recognized in 1993 when an outbreak of severe respiratory illnesses occurred among residents of the southwestern United States (US)[1]. HPS is an acute respiratory illness characterized by a flu-like prodrome consisting of fever, chills, myalgias, headaches, and gastrointestinal symptoms, followed by often severe cardiopulmonary dysfunction resembling adult respiratory distress syndrome (ARDS). Nationwide, the case-fatality ratio for HPS during 2009-2012 was 37.2 percent[2].

Hantaviruses are maintained in rodents which shed the virus in their urine and feces; humans become infected when rodent excreta are stirred into the air and inhaled. Sin Nombre virus is the hantavirus that causes the majority of HPS cases in the U.S. Its reservoir, the deer mouse, is prevalent in undeveloped areas throughout the western U.S. and will readily enter homes and buildings in search of food or nesting material.

We describe here the epidemiology of HPS in California from 2009 through 2012. Two cases of hantavirus infection with onset in 2012 that did not meet the HPS surveillance case definition were not included in the analysis and summary. The epidemiological description of HPS for the 2001-2008 surveillance period can be found in the Epidemiologic Summary of HPS in California, 2001- 2008[3]. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes[4]. Because of the small numbers of reported cases, incidence rates were not calculated.

California reporting requirements and surveillance case definition

California Code of Regulations (CCR), Title 17, requires health care providers to report

suspected cases of hantavirus infection to their local health department immediately by telephone. Since 2011, CCR, Title 17, Section 2505 has required laboratories to notify local health officials of test results suggestive of HPS.

California regulations also require local health officers to report to CDPH cases of hantavirus infections. CDPH officially counted cases of HPS that satisfied the U.S. Centers for Disease Control and Prevention (CDC) surveillance case definition for HPS[5]. CDC defines a confirmed case as one with clinically compatible illness and laboratory confirmation. Clinically compatible illness includes one or more of these clinical features: (i) a febrile illness (i.e., temperature greater than 101.0 ºF [greater than 38.3 ºC]) corroborated by bilateral diffuse interstitial edema or a clinical diagnosis of ARDS or radiographic evidence of noncardiogenic pulmonary edema, or unexplained respiratory ill- ness resulting in death, and occurring in a previously healthy person, or (ii) an unexplained respiratory illness resulting in death, with an autopsy examination demonstrating noncardiogenic pulmonary edema without an identifiable cause. Laboratory confirmation includes detection of hantavirus-specific immunoglobulin (Ig) M or rising titers of hantavirus-specific IgG, or detection of hantavirus-specific ribonucleic acid sequence by polymerase chain reaction in clinical specimens, or detection of hantavirus antigen by immunohistochemistry.

Epidemiology of HPS in California

CDPH received reports of 15 cases of HPS in California residents with illness onset dates from 2009 through 2012. The highest number of cases was in 2012 (8) and the lowest number was in 2011 (0) [Figure1]. During the surveillance period, 3 (20.0 percent) case-patients were reported to have died with HPS.

The number of HPS cases during the surveillance period was highest among persons 35 to 44 years of age [Figure 2]. The median age among case-patients was 42 years (range: 13 to 61 years). HPS patients reported White non-Hispanic (53.3 percent), Hispanic (20.0 percent), Asian (20.0 percent) and non-Hispanic unknown (6.7 percent) race/ ethnicities. The ratio of male to female case-patients was 2:1.

Eleven case-patients were residents of Northern California and 4 were residents of Southern California. The counties of Alameda (2), Mono (3) and Nevada (2) were the only counties to report more than 1 case.

Public health investigations of HPS cases that occurred during 2009 through 2012 revealed that the likely sites of exposure for 7 case-patients were lodgings within Yosemite National Park in the central Sierra Nevada (Mariposa and Tuolumne counties). One of these patients had illness onset during 2010, and the other 6 had illness onsets during 2012. The likely exposure sites for the other 8 case- patients with illness onsets during 2009 through 2012 were the eastern Sierra Nevada (Mono and Inyo counties) for 4 patients, the northern Sierra Nevada (Nevada County) for 2 patients, the southern California high desert for 1 patient, and undetermined for 1 patient.

Figure 1. California HPS case counts by onset year

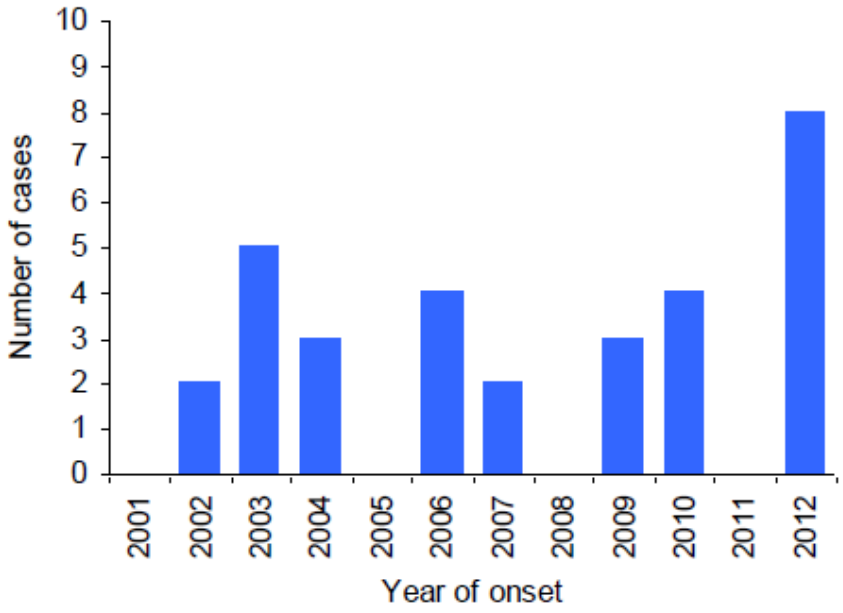
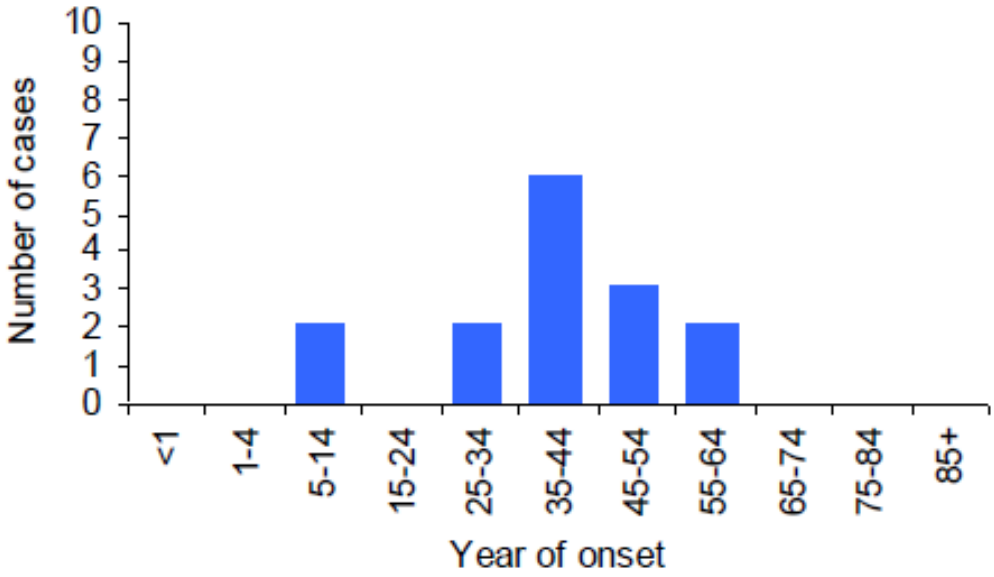


Figure 2. California HPS case counts by age-group, 2009-2012



Comment

Before 2012, the number of California HPS cases remained relatively steady, with an annual average 2.3 cases during 2009-2011, and an annual average 2.0 cases during 2001-2008. The increased number of cases in 2012 was chiefly attributable to several hantavirus infections with onset during July or August 2012 identified among visitors to Yosemite National Park. In addition to 6 HPS cases in California residents, other hantavirus

infections in 2012 among visitors to Yosemite National Park included 1 Pennsylvania resident with HPS, 1 West Virginia resident with HPS, and 2 California residents with hantavirus illnesses that did not meet the HPS case definition[6].

Hantavirus infections are associated with domestic, occupational, or recreational activities that bring humans into contact with rodents and their excreta, usually in rural settings[7]. Eight California residents developed hantavirus illnesses (6 with HPS) after visiting Yosemite National Park in 2012: 7 patients lodged in insulated, double-walled tent cabins in the Yosemite Valley, and 1 patient lodged in standard tent cabins elsewhere in the Park[8,9]. Other HPS case-patients in California reported working in or cleaning confined, poorly ventilated areas around their home or work place--such as storage buildings, sheds, or basements--prior to onset. Follow-up investigations indicated that at least 7 case-patients in California may have been exposed at either their residence or their worksite, underscoring the propensity for deer mice to enter areas of human activity.

The primary strategy for reducing the risk of hantavirus exposure is to avoid contact with rodents and their excreta. Useful measures include preventing rodents from entering buildings, eliminating current rodent infestations, and proper respiratory protection when working in poorly ventilated areas contaminated with rodent excreta.

References and resources

1 Hantavirus Cardiopulmonary Syndrome. California Department of Public Health. http://www.cdph.ca.gov/healthinfo/discond/Pages/HantavirusPulmonarySyndrome.aspx

2 Annual U.S. HPS Cases and Case-fatality, 1993-2012. Centers for Disease Control and Prevention. (http://www.cdc.gov/hantavirus/surveillance/annual-cases.html)

3 Epidemiological Summaries of Selected General Communicable Diseases in California, 2001-2008: Hantavirus Pulmonary Syndrome. (http://www.cdph.ca.gov/data/statistics/Pages/EpiSummariesCDsCA-01-08.aspx)

4 Epidemiologic Summaries of Selected General Communicable Diseases in California, 2009-2012: Technical Notes.

5 National Notifiable Diseases Surveillance System, Case Definitions, Hantavirus. Centers for Disease Control and Prevention. (http://wwwn.cdc.gov/NNDSS/script/conditionsummary.aspx?CondID=76)

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9 Update: Yosemite National Park Continues Response to Hantavirus Cases. National Park Service. (http://www.nps.gov/yose/parknews/hanta0812c.htm)

Last updated 2/19/2014

Prepared by Kirsten Knutson, Curtis L. Fritz, and Farzaneh Tabnak, Infectious Diseases Branch

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 885 cases of legionellosis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.59 cases per 100,000 Californians.
- From 2009 (167 cases, 0.45 per 100,000) through 2012 (247 cases, 0.65 per 100,000), legionellosis incidence rates increased by 44.4 percent. From 2001 (57 cases; 0.17 per 100,000) through 2012, the annual rates increased by 282.4 percent. The highest annual incidence rate occurred in 2011 (251 cases; 0.67 per 100,000).
- From 2009 through 2012, 82 (9.3 percent) reported case-patients died with legionellosis.
- Average legionellosis incidence rates increased with increasing age and were highest among adults 85 years of age and older (3.6 per 100,000).
- Average incidence rates for the surveillance period were 2.6 times higher in southern California (0.81 per 100,000) compared to northern California (0.31 per 100,000).
- In 2009, there were two outbreaks involving two cases each in southern California from recreational water exposure.
- Further study may help determine if an increasing population of older persons and other at risk individuals, improved detection such as increased use of urine legionella antigen testing and reporting (endorsement of more timely and sensitive surveillance), or some combination thereof contributed to the steady increase in legionellosis incidence rates in California.

Background

Legionella is an important respiratory bacterial pathogen in the United States (US). The national incidence rate has increased 192% from 0.39 per 100,000 population in 2000 to 1.15 per 100,000 in 2009[1].It was estimated that *Legionella* caused between 8,000 and 18,000 cases of community-acquired pneumonias requiring hospitalization each year[2]. People get legionellosis from inhaling or aspirating contaminated water aerosols. Legionellae are ubiquitous in manmade and fresh water-environments where they replicate within free-living amoebae. Warm temperatures and biofilms support bacterial growth, and hot-water and aircirculation systems, hot tubs, and decorative fountains have been implicated exposure sources in community-based outbreaks. *L. pneumophila* serogroup 1 is the most frequently identified serogroup among reported cases (the causative agent in 60% to 80% of patients)[3]. Most cases are now diagnosed by urine antigen, which is highly specific but only for *L. pneumophila* serogroup 1, so that disease caused by other serogroups or species is less likely to be diagnosed.

Legionellosis is associated with two clinically and epidemiologically distinct syndromes. Pontiac fever is a self-limited, nonpneumonic, influenza-like illness whereas Legionnaires' disease is a common cause of serious bacterial pneumonia. The vast majority of reported legionellosis cases are Legionnaires' disease. Although most cases occur sporadically, outbreaks have been identified in nosocomial and community based settings. Since its addition to national outbreak surveillance in 2001, *Legionella* has been the

most commonly reported pathogen associated with drinking water outbreaks. Persons at increased risk for legionellosis include those of advanced age and deficient immune status[4,5].

We describe here the epidemiology of legionellosis in California from 2009 through 2012. Data for 2012 are provisional and may differ from data in future publications. The epidemiological description of legionellosis for the 2001–2008 periods can be found in the Epidemiologic Summary of Legionellosis in California, 2001–2008[6]. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes[7].

California reporting requirements and surveillance case definitions

California Code of Regulations (CCR), Title 17, Section 2500 requires health care providers to report suspected cases of legionellosis to their local health department within seven working days of identification or immediately by telephone if an outbreak is suspected. In late 2006, revised regulations required clinical and reference laboratories to notify the local health department when laboratory testing yielded evidence suggestive of *Legionella* within one working day after the health care provider has been notified. Since 2010, CCR, Title 17, Section 2505 has also mandated all laboratories to report *Legionella* spp. (antigen or culture) to the local health jurisdictions within one working day after the health care provider or other person authorized to receive the report has been notified.

Local health officers are required by regulation to report to CDPH cases of legionellosis. CDPH officially counts cases that meet the 2005 U.S. Centers for Disease Control and Prevention (CDC)/Council of State and Territorial Epidemiologists' surveillance case definition. During the surveillance period, CDC defined a confirmed case as one with clinically compatible illness and either culture isolation of any *Legionella* organism from respiratory secretions, lung tissue, pleural fluid, or other normally sterile fluid; detection of *L. pneumophila* serogroup 1 antigen in urine; or at least a four-fold increase in serum antibody titer for *L. pneumophila* serogroup [1].

Epidemiology of legionellosis in California

CDPH received reports of 885 cases of legionellosis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.59 cases per 100,000 Californians. Legionellosis incidence rates increased by 44.4 percent from 0.45 per 100,000 (167 cases) in 2009 to 0.65 per 100,000 (247 cases) in 2012. From 2001 through 2012, the annual rates increased by 282.4 percent (from 57 cases, 0.17 per 100,000 in 2001). The highest annual incidence rate occurred in 2011 (251 cases; 0.67 per 100,000) [Figure 1]. During the surveillance period, 82 (9.3 percent) case-patients were reported to have died with legionellosis.

Average legionellosis incidence rates increased with increasing age and were highest among adults 85 years of age and older (3.6 per 100,000) [Figure 2]. The ratio of male to female cases was 2:1. Incidence rates by race/ethnicity were not calculated due to missing data (7.2 percent). However, legionellosis cases with complete data reported White and Black/African American non-Hispanic race/ethnicities more frequently than would be expected based on the demographic profile of California [Figure 3].

Average incidence rates for the surveillance period were 2.6 times higher in southern California (0.81 per 100,000) compared to northern California (0.31 per 100,000). From 2009 through 2012, incidence rates increased by 43.3 percent in southern California (from0.60 to 0.86 per 100,000) and by 52 percent in

northern California (from 0.25 to 0.38 per 100,000).

In 2009, there were two outbreaks involving two cases each; these outbreaks were attributed to the colonization of recreational water facilities at an apartment community and a fitness center in southern California.

Figure 1. California legionellosis case counts and incidence rates

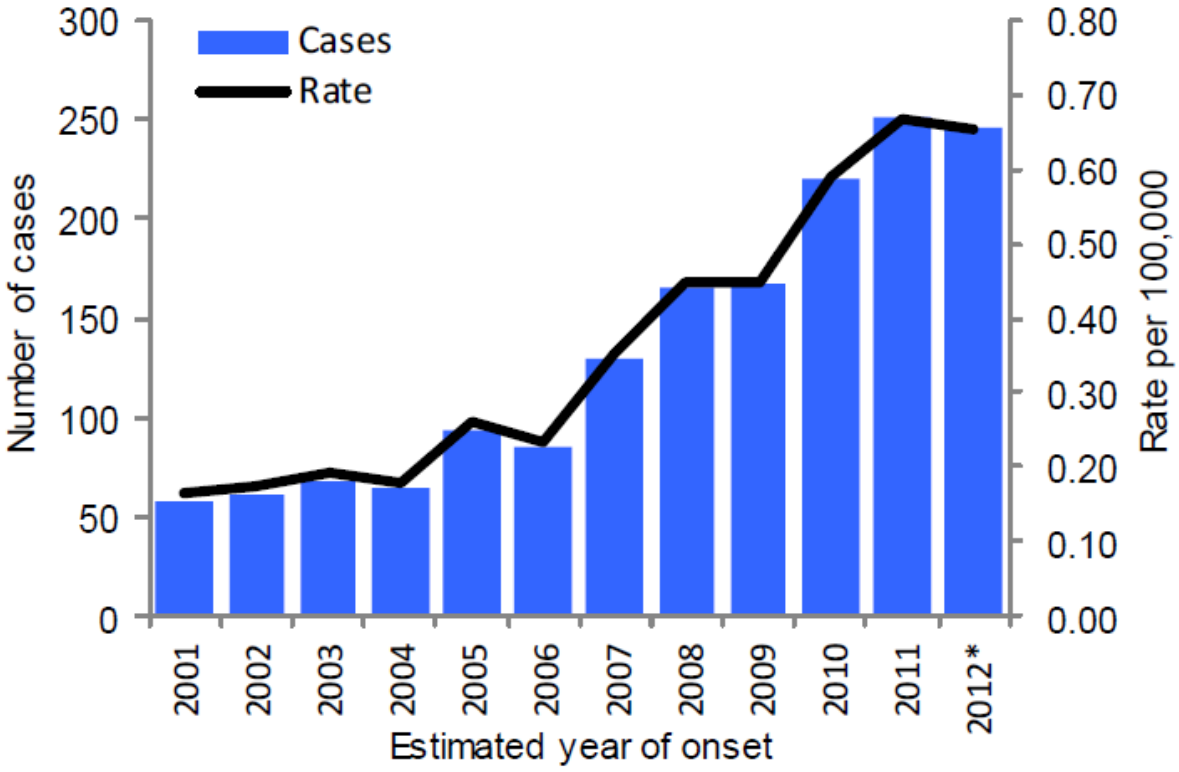


Figure 2. California legionellosis incidence rates by age and time period

Figure 2. California legionellosis incidence rates by age and time period

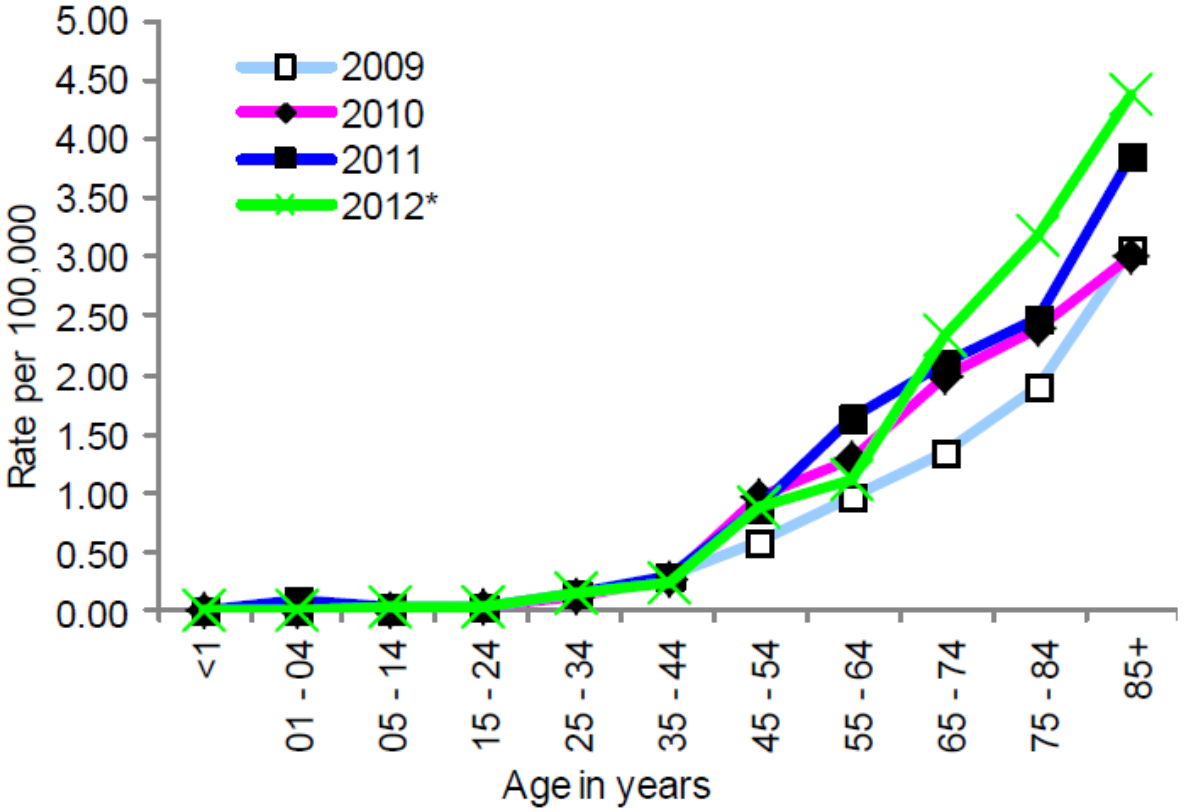
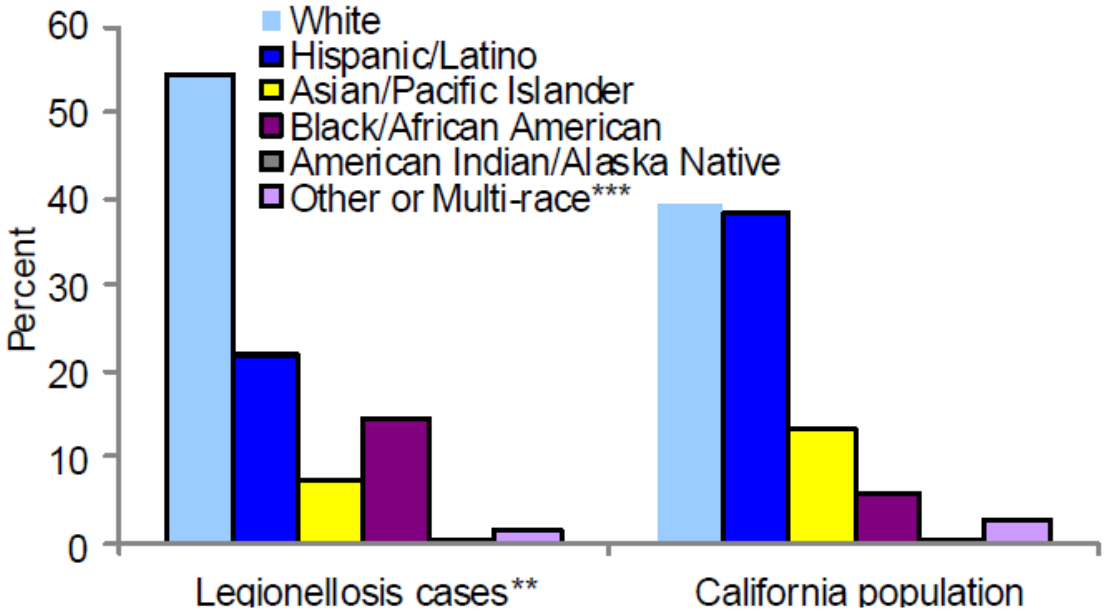


Figure 3. California legionellosis cases and population by race/ethnicity 2009 - 2012*



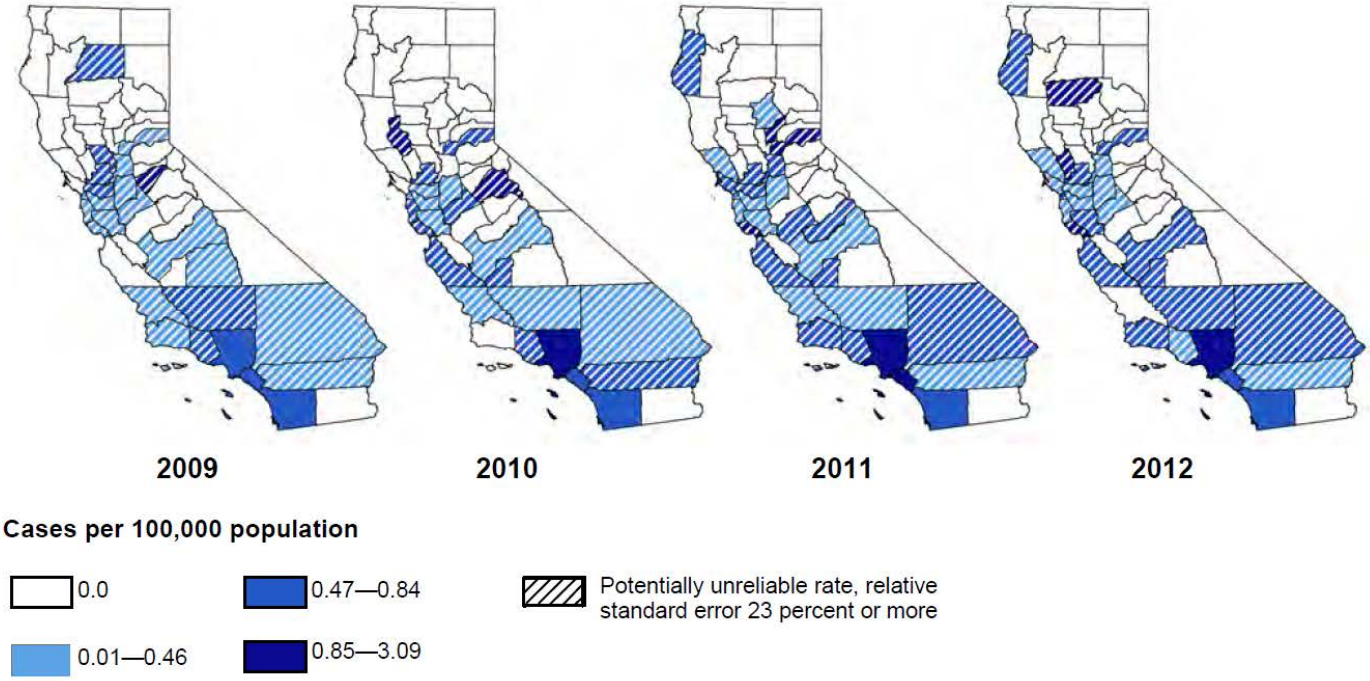
Notes for Figures 1-3

*2012 data are provisional

**Unknowns were excluded

***Includes cases who identified 'other' as their race and Californians ('population') who identified more than one race

Figure 4. California county-specific legionellosis incidence rates



Comments

During the surveillance period, the highest annual number of legionellosis cases was reported in 2011. California experienced a significant increase in reported legionellosis incidence rates from 2001 through 2012. The increase in incidence rates was also noticeable from 2009 to 2012. Similar increases have also been noted nationally[1,8,9]. Age group, race/ethnicity, gender, and county epidemiologic profiles of incident cases with estimated onset dates from 2009 through 2012 remained fairly consistent with those with estimated onset dates from 2001 through 2008 as described previously. California reported two legionellosis outbreaks in 2009. In the U.S. during 2007-2008, legionellosis was the most frequently reported etiology among drinking water-associated outbreaks that were mostly attributed to untreated or inadequately treated ground water and majority were occurred in public water systems[10].

Prevention efforts targeting against pathogens, infrastructure problems, and water sources associated with waterborne disease outbreaks are key to reduce legionellosis-associated waterborne outbreaks. Further study may help determine if an increasing population of older persons and other at risk individuals, improved detection such as increased use of urine legionella antigen testing and reporting, or some combination thereof contributed to the steady increase in legionellosis incidence rates in California.

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6 Epidemiological Summaries of Selected General Communicable Diseases in California, 2001-2008: Legionellosis http://www.cdph.ca.gov/programs/sss/Documents/Epi-Summaries-CA-2001-2008- 083111.pdf#page=39

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Last updated: 03/13/2014

Prepared by Farzaneh Tabnak, Alyssa Nguyen, Lauren Lee, and Duc Vugia, Infectious Diseases Branch

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 424 cases of listeriosis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.28 cases per 100,000 Californians.
- During 2009-2012, listeriosis incidence rates in California were relatively stable, ranging from a minimum of 0.26 per 100,000 in 2009 and 2010 to a maximum of 0.32 per 100,000 in 2011.
- During the surveillance period, 60 (14.2 percent) case-patients were reported to have died with listeriosis.
- Average annual listeriosis incidence rates during the surveillance period were highest among adults 65 years of age or older (1.22 per 100,000) and children under 1 year of age (0.94 per 100,000).
- From 2009 through 2012, there were 5 multi-state foodborne outbreaks of listeriosis involving more than 28 California case-patients (as of February 2016).
- Improving the safety of food, such as soft cheeses and raw produce, and educational outreach to high risk consumers such as pregnant women, the immunocompromised, and adults 65 years of age or older may provide the best opportunities for reducing listeriosis.

Background

In the United States (US), listeriosis is an uncommon but serious foodborne illness associated with an estimated 1,600 infections and more than 1,400 hospitalizations annually. Listeriosis is a leading cause of foodborne-related mortality in the US, with infection resulting in more than 250 deaths each year[1]. Listeriosis is caused by the bacteria *Listeria monocytogenes*, which is ubiquitous in the environment in soil, vegetation, and untreated water and can infect various animals. The national *Healthy People 2020* target objective for listeriosis is for an incidence rate lower than 0.20 new cases per 100,000 population.

Consuming foods contaminated with *Listeria* is the leading source of infection. *Listeria* has been found in raw foods, including unpasteurized milk and milk products, uncooked meats, and produce, and has also been found in foods that became contaminated after processing, such as ready-to-eat meats and soft cheeses[2]. Cooking and pasteurizing kills *Listeria*, but unlike other foodborne pathogens, *Listeria* will multiply in refrigerated temperatures[3].

More than 90% of *Listeria* infections occur in immunocompromised persons, adults 65 years and older, and pregnant women and their newborns[3]. Onset of symptoms after exposure can range from as little as one day to more than two months[4]. Symptoms can vary but include gastroenteritis, fever, head and muscle aches, stiff neck and convulsions. Severe illness can result in meningoencephalitis, septicemia, and death. Most case-patients experience severe, invasive

illness: immune-compromised persons and adults 65 years and older are at greatest risk. Although infected pregnant women often experience only a mild illness, infection during pregnancy can lead to premature delivery, miscarriage, stillbirth, or serious infection in the newborn[3].

This report describes the epidemiology of confirmed *Listeria* infections in California with estimated symptom onset dates from January 1, 2009 through December 31, 2012. A description of listeriosis outbreaks involving California case-patients that occurred during 2009-2012 is also included. The year in which an outbreak occurred was defined as the earliest date of illness onset among the case-patients involved in outbreak. A multi-state outbreak with the patients' year of illness onset ranging from 2010 to 2015 was included in the outbreak discussion. Both listeriosis cases and outbreaks were reported by February 2016. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to the Technical Notes[5]. The epidemiologic description of listeriosis for the 2001-2008 surveillance period can be found in the Epidemiologic Summary of Listeriosis in California, 2001-2008[6].

California reporting requirements and surveillance case definitions

California Code of Regulations, Title 17, requires health care providers to report suspected cases of listeriosis to their local health department within one working day of identification or immediately by telephone if an outbreak is suspected. Laboratories are also required to report laboratory testing results suggestive of *Listeria* infection to either the California Reportable Disease Information Exchange (CalREDIE) (via electronic laboratory reporting) or the local health department; reporting must occur within one working day after the health care provider has been notified.

California regulations require local health officers to report cases of listeriosis to CDPH. Cases were counted as confirmed by CDPH based on the Centers for Disease Control and Prevention (CDC)/Council of State and Territorial Epidemiologists' surveillance case definition of a confirmed case. During the surveillance period, a confirmed case of listeriosis was defined as one with *L. monocytogenes* isolated from a normally sterile site or, in the setting of a miscarriage or stillbirth, isolation of *L. monocytogenes* from placental or fetal tissue[7].

Epidemiology of listeriosis in California

CDPH received reports of 424 cases of listeriosis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.28 cases per 100,000 Californians. Incidence rates during the 2009-2012 surveillance period were relatively stable, fluctuating within the same range as rates during the previous surveillance period [Figure 1]. Incidence rates ranged from a minimum rate of 0.26 per 100,000 (97 and 98 cases) in 2009 and 2010 to a maximum rate of 0.32 per 100,000 (121 cases) in 2011. During the surveillance period, 60 (14.2 percent) case-patients were reported to have died with listeriosis.

Average annual listeriosis incidence rates during 2009-2012 were highest among adults 65 years of age or older (1.22 per 100,000, not shown) and children under 1 year of age (0.94 per 100,000) [Figure 2].

The ratio of female to male cases was 1.3:1.0. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (14.4 percent). However, listeriosis cases with complete information reported White non-Hispanic and Asian/Pacific Islander race/ethnicities more frequently than would be expected and Hispanic ethnicity less frequently than would be expected based on the demographic profile of California [Figure 3]. County-specific average annual listeriosis incidence rates during the surveillance period ranged from 0 to 0.93 per 100,000 [Figure 4]. Average annual incidence rates were similar in Northern California (0.31 per 100,000) and Southern California (0.26 per 100,000). During 2009 through 2012, 19 counties reported average annual incidence rates that were above the Healthy People 2020 target objective.

From 2009 through 2012, there were 5 foodborne outbreaks of listeriosis involving more than 28 California case-patients. For each of the outbreaks, California was one of multiple states where exposure occurred. Among 4 outbreaks with a confirmed food vehicle, soft cheese (made with pasteurized and unpasteurized milk) was implicated in 3 outbreaks, and whole cantaloupe was implicated in 1 outbreak.

One of the 5 multistate *Listeria* outbreaks described above involved 34 case-patients reported from 10 states with illness onset ranging from 2010 to 2015 (including 21 California case-patients, 2 of whom died). This outbreak was first identified by CDC in 2015 when a cluster of patients were found to be infected with a rare strain of *Listeria*. Advanced genetic testing (whole genome sequencing (WGS)) subsequently linked earlier cases to the outbreak. Illnesses were associated with the consumption of various types of pasteurized cheeses sold under multiple brand names by a company in California. Environmental samples taken from the company's production facility in 2010 and 2015 matched the patient laboratory specimens by WGS. Four California patients had illness onset during the 2009-2012 surveillance period.

Figure 1. California listeriosis case counts and incidence rates by estimated year of illness onset

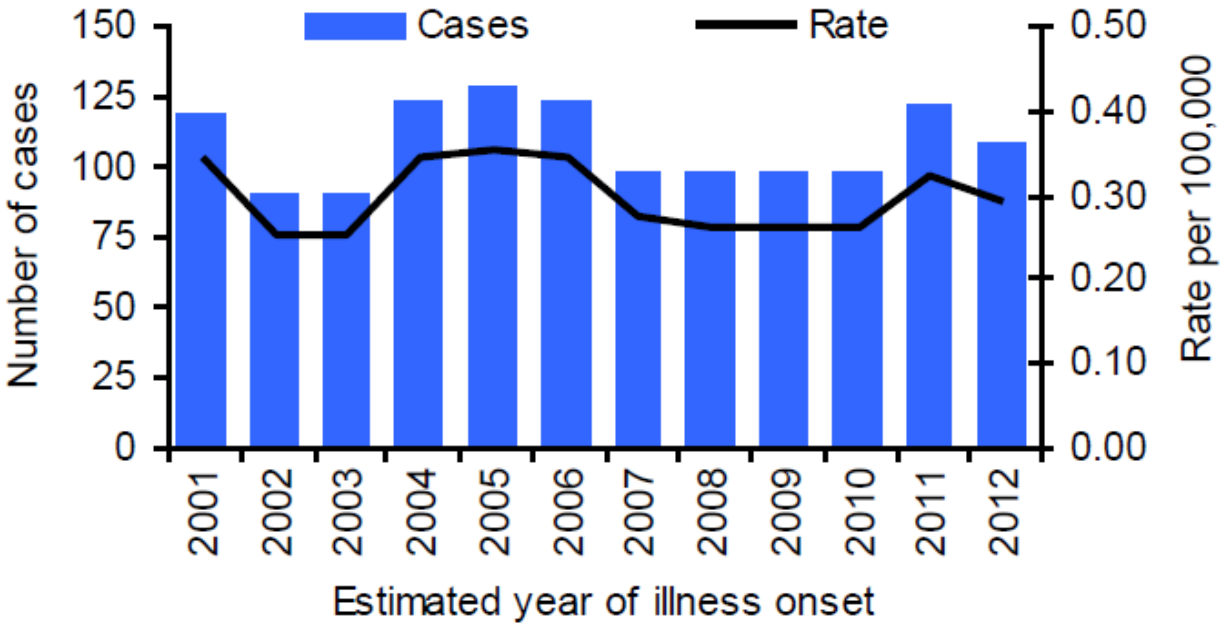


Figure 2. California listeriosis average annual incidence rates by age group, 2009—2012

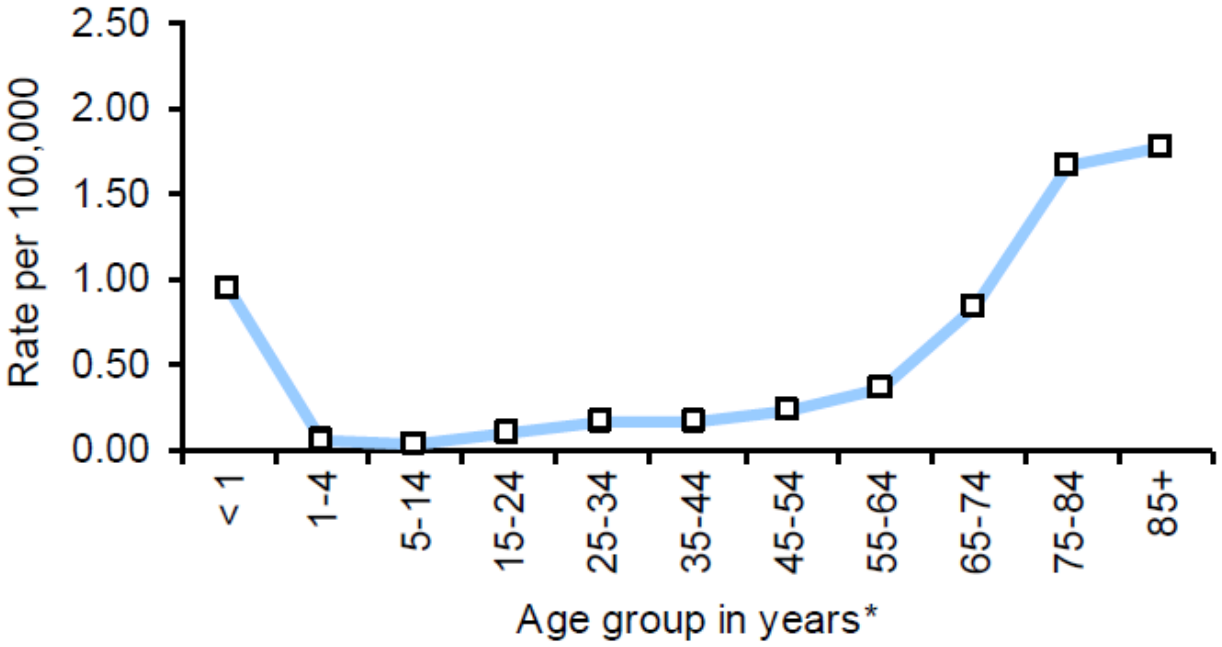


Figure 3. California listeriosis cases and population by race/ethnicity, 2009 - 2012

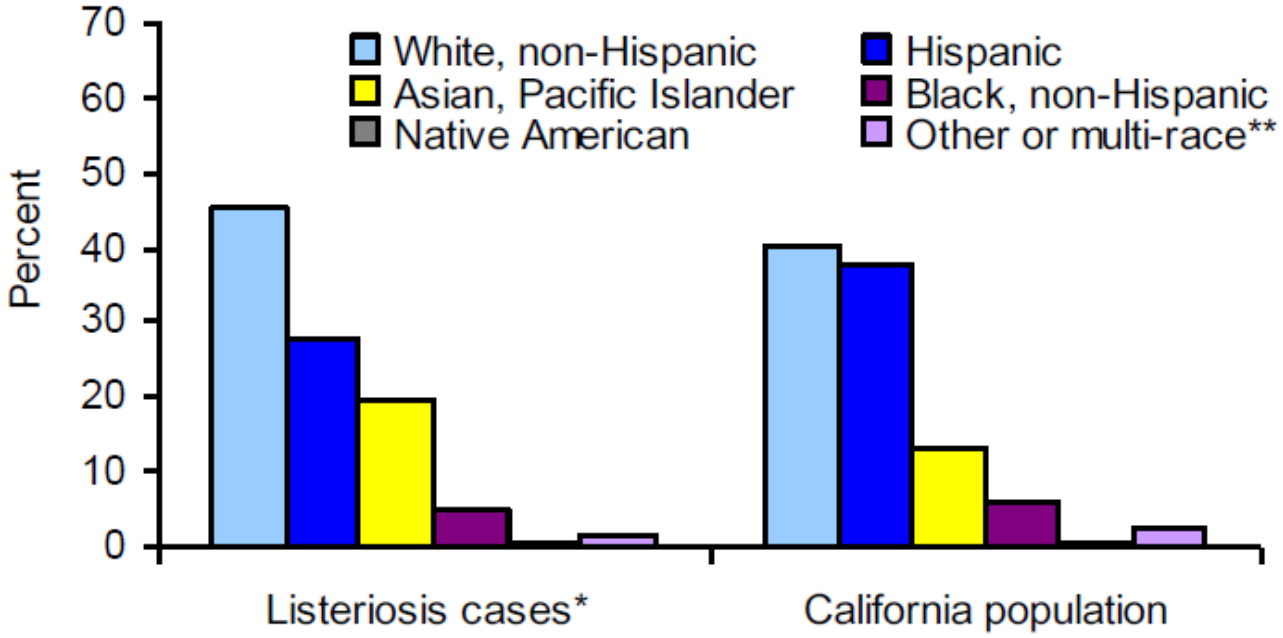
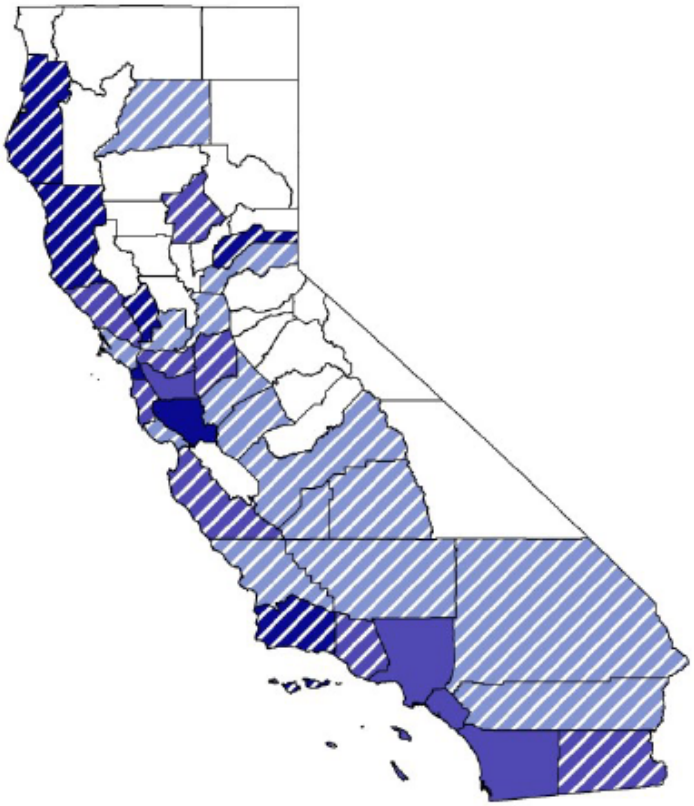


Figure 4. California county-specific listeriosis average annual incidence rates, 2009—2012



Cases per 100,000 population

- 0.0
- 0.1 - 0.2
- 0.3 - 0.5
- 0.6 - 0.9
- Potentially unreliable rate, relative standard error 23 percent or more

Comment

Incidence rates of reported listeriosis among Californians were relatively stable from 2009 through 2012. Each year during the surveillance period, the statewide incidence rate of listeriosis was greater than the national Healthy People 2020 target objective.

The age and gender distribution of reported cases incident in California during 2009-2012 remained fairly consistent with that of 2001-2008, although children under 1 year of age had a slightly lower incidence during this surveillance period[6]. Comparable to national trends, California children under 1 year of age and adults 65 years of age or older experienced the highest rates of listeriosis[8].

Improving the safety of foods, such as soft cheeses and raw produce, and educational outreach to high-risk consumers such as pregnant women, the immunocompromised, and adults 65 years of age and over may provide the best opportunities for reducing the incidence of listeriosis. Additionally, continued surveillance of human infections, especially in combination with enhanced molecular characterization of infecting strain types, may help detect dispersed, previously unrecognized disease clusters.

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Last updated 7/18/2016

Prepared by Kirsten Knutson, Farzaneh Tabnak, Hilary Rosen and Akiko Kimura, Infectious Diseases Branch

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received 374 reports of Lyme disease with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.25 cases per 100,000 Californians.
- Lyme disease incidence rates were relatively stable during the 2009-2012 surveillance period. Incidence rates declined from 0.28 per 100,000 (102 cases) in 2009 to 0.21 per 100,000 (81 cases) in 2012.
- Average annual Lyme disease incidence rates during the surveillance period were highest among adults 55 to 64 years of age (0.35 per 100,000) and children 5 to 14 years of age (0.34 per 100,000).
- Counties of the Far North (1.64 per 100,000) and Central Coast (0.86 per 100,000) regions reported the highest average annual incidence rates.
- Avoiding exposure to vector ticks provides the best opportunity for preventing and controlling Lyme disease. If potential exposure is unavoidable, important risk reduction measures include using both protective clothing and tick repellents, checking the entire body for ticks daily, and prompt removal of attached ticks.

Background

Lyme disease is caused by the bacteria Borrelia burgdorferi which is transmitted to humans by the bite of an infected tick. Lyme disease is the most common tick-borne infection in North America, with nearly 30,000 cases reported in the United States (US) each year. Over 95 percent of cases occur in the Northeastern, mid-Atlantic, and upper mid-Western states.1The most common initial sign of Lyme disease is a red, usually painless expanding rash (erythema migrans) that appears within 30 days after the bite of an infected tick. Other early symptoms include flu-like body aches, fatigue, fever, chills and swollen lymph nodes. If not treated, some patients can develop neurologic conditions or cardiac abnormalities during the next few weeks, or more severe central nervous and musculoskeletal disease up to several months later. Lyme disease is diagnosed based on symptoms, physical findings (e.g., erythema migrans), history of engaging in outdoor activity during the incubation period in areas where vector ticks are known to occur, and supportive laboratory testing. Most cases of Lyme disease can be treated successfully with oral or intravenous antibiotics.[1-2]

We describe here the epidemiology of confirmed and probable Lyme disease cases in California with estimated illness onset from 2009 through 2012 reported to CDPH by November 2015. Data for 2012 are provisional and may differ from data in future publications. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to the Technical Notes.[3] The epidemiologic description of Lyme disease for the 2001-2008 surveillance period can be found in the *Epidemiologic Summary of Lyme disease in California, 2001-2008*.[4]

California reporting requirements and surveillance case definitions

California Code of Regulations, Title 17, requires health care providers to report suspected cases of Lyme disease to their local health department within seven calendar days of identification. Laboratories are also required to report laboratory testing results suggestive of *B. burgdorferi* infection to either the California Reportable Disease

Information Exchange (CalREDIE) (via electronic laboratory reporting) or the local health department; reporting must occur within one working day after the health care provider has been notified.

California regulations also require local health officers to report to CDPH cases of Lyme disease. CDPH counted cases that satisfied the US Centers for Disease Control and Prevention (CDC)/Council of State and Territorial Epidemiologists' surveillance case definition of a confirmed or probable case.[5] During the surveillance period, a confirmed case was defined as one with: (i) a physician diagnosed erythema migrans of at least 5 cm diameter with either a known exposure or laboratory evidence of infection or (ii) at least one objective late manifestation (i.e., musculoskeletal, cardiovascular, or neurological) and laboratory evidence of infection. A probable case was defined as any other case of physician-diagnosed Lyme disease that had laboratory evidence of infection. Laboratory evidence of infection included: (1) a positive culture of *B. burgdorferi* or (2) two-tiered testing (a sensitive enzyme immunoassay (EIA) or immunofluorescence antibody assay (IFA) followed by a Western blot) interpreted using established criteria, where a positive IgM test result was sufficient only when the test was performed within 30 days from symptom onset, and a positive IgG test result was sufficientat any point during the patient's illness or (3) single-tier IgG immunoblot seropositivity interpreted using established criteria.[6-8] Beginning in 2011, laboratory evidence of infection could also include the demonstration of antibody production against *B. burgdorferi* in cerebrospinal fluid (CSF) via EIA or IFA, evidenced by a higher titer of antibody in CSF then in serum.

Epidemiology of Lyme disease in California

CDPH received reports of 374 cases of Lyme disease with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.25 cases per 100,000 Californians. Incidence rates during the 2009-2012 surveillance period were relatively stable, showing moderate fluctuations [Figure 1]. Incidence rates ranged from a minimum of 0.21 per 100,000 (81 cases) in 2012 to a maximum of 0.28 per 100,000 (102 cases) in 2009. During the surveillance period, no case-patients were reported to have died by the time of case report. Average annual incidence rates for the surveillance period were highest among adults 55 to 64 years of age (0.35 per 100,000) and children 5 to 14 years of age (0.34 per 100,000) [Figure 2]. The ratio of male to female cases was 1.2:1.0. Rates by race/ethnicity were not calculated due to the substantial portion of missing data (35.6 percent). However, Lyme disease cases with complete data reported White non- Hispanic race/ethnicity (84.6 percent) more frequently than would be expected (40.2 percent) based on the overall demographic profile of California [Figure 3].

County-specific average annual incidence rates from 2009 through 2012 ranged from 0 to 7.86 per 100,000, with Mendocino (5.40 per 100,000) and Humboldt (4.64 per 100,000) counties showing the highest average rates [Figure 4]. Average annual incidence rates for the surveillance period were 3.9 times higher in Northern California (0.43 per 100,000) than in Southern California (0.11 per 100,000). The Far North (1.64 per 100,000) and Central Coast (0.86 per 100,000) regions reported the highest average annual incidence rates during the surveillance period.

A total of 182 (48.7 percent) cases had estimated illness onsets during the months of June through August.

Figure 1. California Lyme disease case counts and incidence rates by estimated year of illness onset, 2001-2012*

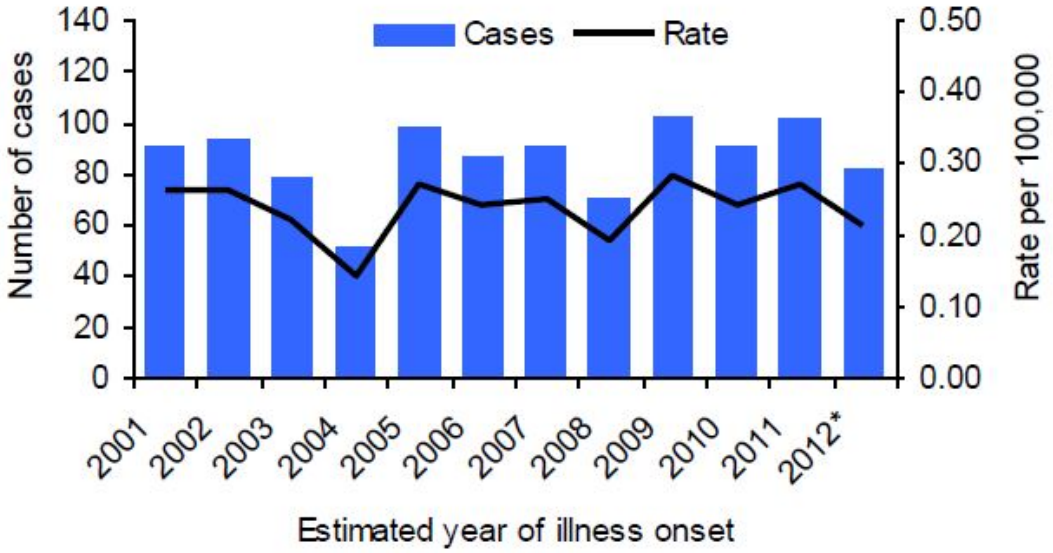


Figure 2. California Lyme disease incidence rates by age group, 2009 - 2012*

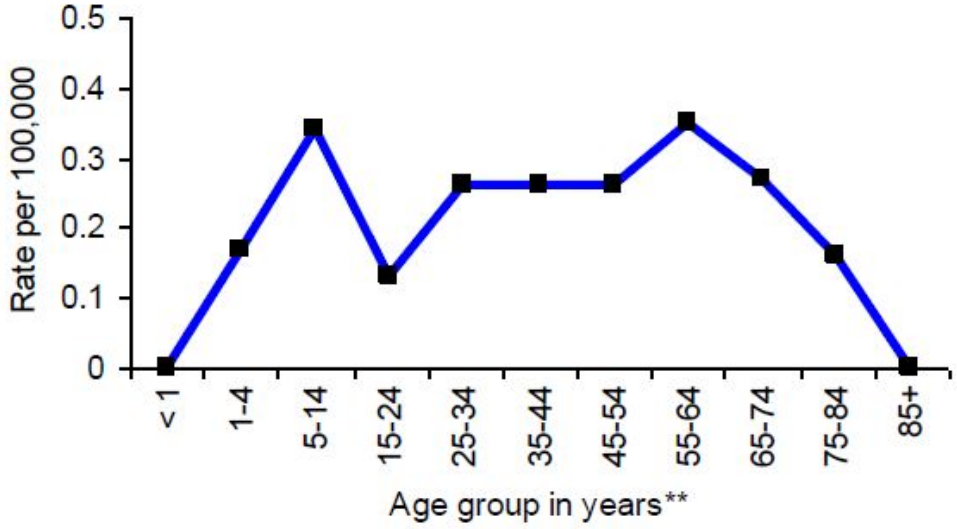
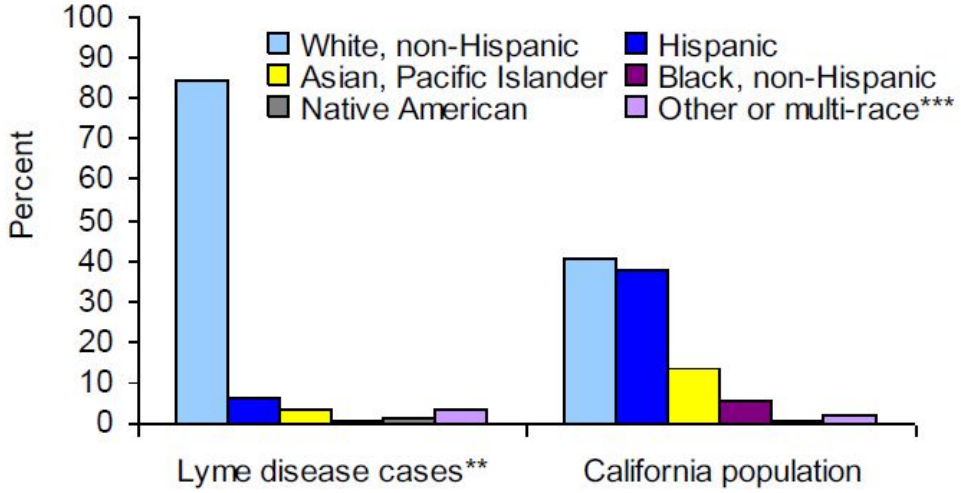


Figure 3. California Lyme disease cases and population by race/ethnicity, 2009-2012*



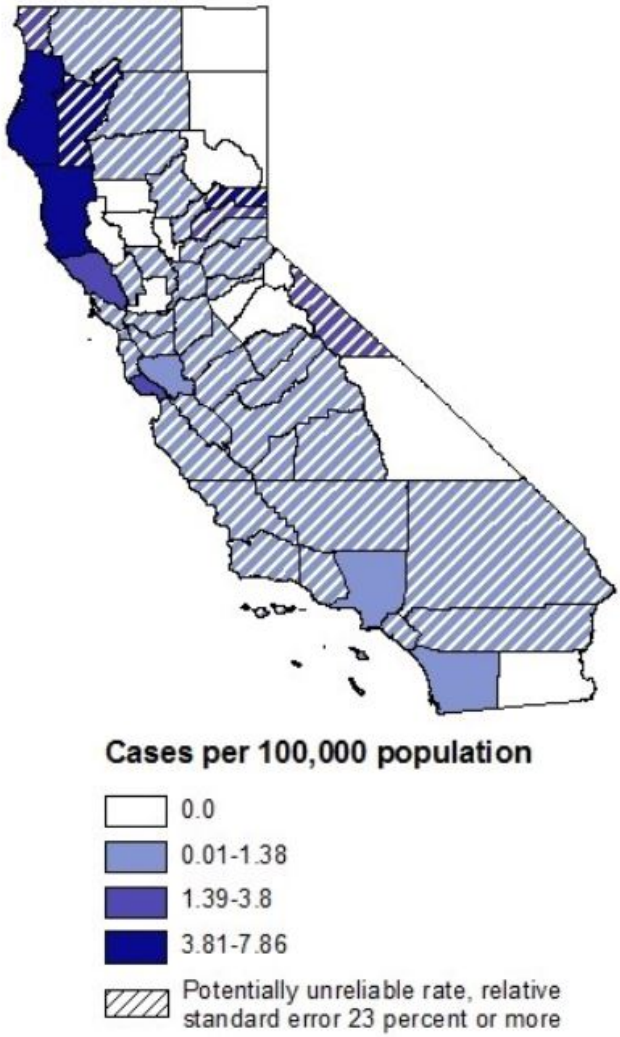
Notes for Figures 1-4

*2012 data are provisional

**Unknowns were excluded

***Includes cases who identified 'other' as their race and Californians ('population') who identified more than one race

Figure 4. California county-specific Lyme disease incidence rates, 2009 - 2012*



Comment

Incidence rates of reported Lyme disease during the 2009 through 2012 surveillance period were relatively stable. The race/ethnicity, gender, and geographic distribution and temporal pattern of Lyme disease cases incident in California from 2009 through 2012 remained fairly consistent with that of the 2001 through 2008 surveillance period. Conversely, children ages 5 to 14 years of age had a higher incidence rate during 2009 through 2012 than during the previous surveillance period.4 The bimodal age distribution of California cases is similar to the age distribution of cases nationwide.[9]

Some Lyme disease case-patients that reside in low- incidence states, such as California, may have been exposed to the bacteria when traveling to other states where incidence is higher.13 However, the western black-legged tick, which spreads Lyme disease in the western US, has been found in many wooded and grassy areas in California. People are most commonly exposed to the Lyme disease bacteria by the immature nymphal

tick which is active in the spring and early summer; a range of 5-15% of nymphal western black-legged ticks in California are infected with *B. burgdorferi*.[2]

Lyme disease prevention is best effected by avoiding areas where ticks occur, or if potential exposure is unavoidable, using both protective clothing and tick repellents, checking the entire body, clothing and pets for ticks daily, and promptly removing attached ticks.[1]

References and resources

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Prepared by Kirsten Knutson, Alyssa Nguyen, Farzaneh Tabnak and Anne Kjemtrup, Infectious Diseases Branch

Last updated: 7/12/2016

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 69 cases of Q fever with estimated illness onset dates from 2009 through 2012. This corresponds to an incidence rate of 0.05 per 100,000 population per year.
- Q fever incidence increased by 25.0 percent from 2009 (15 cases; 0.04 per 100,000 population) to 2012 (20 cases; 0.05 per 100,000 population). Although the increase during this surveillance period was small, the highest incidence (0.06 per 100,000 population in 2011) equaled the highest observed in the previous surveillance period (0.06 per 100,000 population in 2006 and in 2008).
- The Q fever incidence rate during the surveillance period was highest among persons 55 to 64 years of age (0.12 per 100,000 population per year).
- The ratio of male to female case-patients in 2009 to 2012 was 2.3:1.
- By region, incidence rates were highest in the Sacramento Metro (0.13 per 100,000 population per year) and San Joaquin Valley (0.13 per 100,000 population per year) regions.
- Persons in higher risk occupations, such as farmers and veterinarians, should limit contact with infected animals, their tissues, and their environments to reduce the opportunity for exposure to Q fever bacteria.

Background

Coxiella burnetii is a bacterial zoonotic pathogen that is widespread throughout the United States and the world. Transmission occurs chiefly through inhalation of aerosolized reproductive fluids from infected animals (especially parturient goats, sheep, and cattle). Contact with other animal fluids (e.g., milk, urine), inhalation of aerosolized particulates from contaminated environmental materials (e.g., hay, dust), and bites by infected ticks may also result in infection.[1,2] *C. burnetii* is listed among the Centers for Disease Control and Prevention (CDC) category B bioterrorism agents.[3]

Q fever has an incubation period of 2 to 3 weeks. Clinical manifestations vary widely in severity and symptoms, according to the route and size of inoculum and host factors. Up to one-half of infections are asymptomatic. Acute Q fever presents most commonly as an influenza-like febrile syndrome; pneumonia and hepatitis are other presentations of acute Q fever. Less than five percent of infections proceed to chronic Q fever, which manifests most frequently as endocarditis in patients with preexisting cardiac pathology (e.g., valvular disease). Most cases of acute Q fever are self-limited and patients recover in 1 to 2 weeks without complication. Treatment with tetracycline antimicrobials is recommended for patients with, or at increased risk for, chronic Q fever [1,2,4].

We describe here the epidemiology of human Q fever cases reported in California from 2009 through 2012. Cases that met criteria for confirmed or probable acute and chronic infection were included. Data for 2012 are provisional and may differ from data in future publications. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to the Technical Notes.[5] The

epidemiological description of Q fever for the 20012008 surveillance period can be found in the Epidemiologic Summary of Q fever in California, 2001- 2008.[6]

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report suspected cases of Q fever to their local health department within one working day of identification. Since 2010, CCR, Title 17, Section 2505 has required laboratories to notify local health officials of test results suggestive of Q fever.

California regulations also require local health officers to report to CDPH cases of Q fever. CDPH officially counted cases that satisfied the CDC/Council of State and Territorial Epidemiologists' surveillance case definition for Q fever.7 During the surveillance period, CDC defined a confirmed acute case as one with (i) clinically compatible illness or an epidemiological link and (ii) laboratory confirmation defined as ≥4-fold change in IgG antibody titer to *C. burnetii* phase II antigen in paired serum specimens, or isolation of *C. burnetii* from a clinical specimen by culture, or demonstration of *C. burnetii* DNA in a clinical specimen by amplification of a specific target by polymerase chain reaction assay or by immunohisto-chemical methods. A probable acute case was one with clinically compatible illness and supportive serology, defined as a single titer of IgG Phase II antibody phase antigen.

Epidemiology of Q Fever in California

CDPH received reports of 69 cases of Q fever with estimated illness onset dates from 2009 through 2012, corresponding to an incidence rate of 0.05 per 100,000 population per year. Q fever incidence increased by 25.0 percent from 2009 (15 cases; 0.040 per 100,000 population) to 2012 (20 cases; 0.05 per 100,000 population). During 2001-2012, the incidence peaked at 0.06 per 100,000 population in 2006, 2008, and 2011 [Figure 1].

Of 2009-2012 incident cases, 11 (15.9 percent) met the case definition of a confirmed case and 58 (84.1 percent) were determined to be probable cases. One case-patient was reported to have died with probable Q fever.

The Q fever incidence rate for the four-year surveillance period was highest among persons 55 to 64 years of age (0.12 per 100,000 population per year) [Figure 2]. The ratio of male to female case-patients was 2.3:1. Incidence rates by race/ethnicity were not calculated because data were not available for 23.2 percent of reported cases. However, for Q fever cases with complete information on race/ethnicity, White non-Hispanic was over-represented (49.1 percent) and Asian/Pacific Islander (5.7 percent) under-represented compared to their respective proportions in the California population. The Q fever incidence rate was higher in Northern California (0.07 per 100,000 population per year) than in Southern California (0.03 per 100,000 population per year). For the four-year surveillance period, incidence rates reported from the regions of the Sacramento Metro (0.13 per 100,000 population per year) and San Joaquin Valley (0.13 per 100,000 population per year) were the highest in the state.

Figure 1. California Q Fever case counts and incidence rates

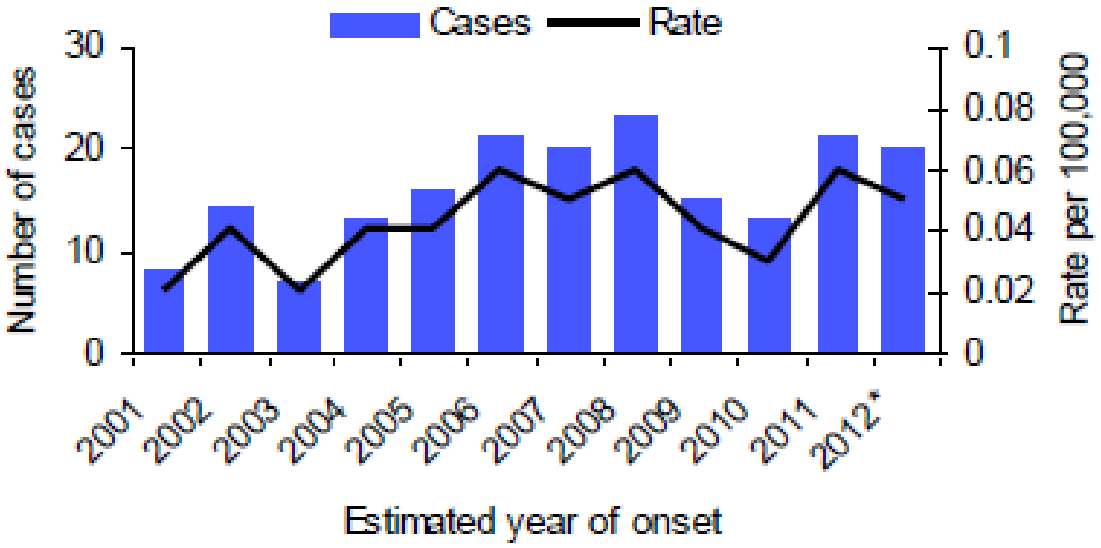
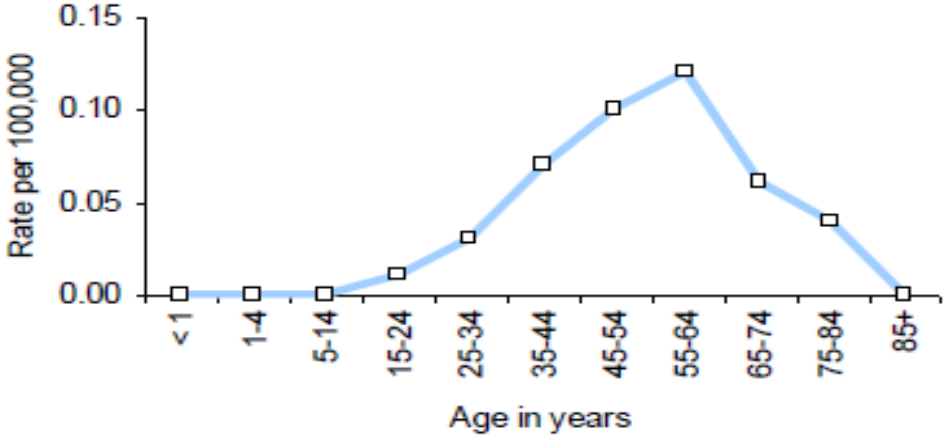


Figure 2, California Q Fever incidence rates by age group, 2009 - 2012*



Notes for Figures 1-2
*2012 data are provisional

Comment

There was a small increase in the incidence of Q fever in California during 2009 to 2012. However, the highest incidence 0.06 per 100,000 population in 2011 equaled that of the previous surveillance period of 2001-2008, during which incidence increased significantly. The age and race/ethnicity distribution of cases during 2009-2012 remained fairly consistent with that of 2001-2008.[6] In contrast, while more men than women became ill with Q fever during both surveillance periods, a greater proportion of the 2009-2012 case-patients were female than in 2001-2008. There were no known outbreaks of Q fever in California during 2009-2012.

Individuals who have routine direct contact with ruminants on farms, at slaughterhouses, or in research facilities are at an increased risk of exposure to *C. burnetii*.[4] Limiting exposure to infected animals and their environments (especially livestock birthing areas), and educating higher risk groups (especially persons in higher risk occupations) may provide the best opportunities for human Q fever prevention and control.

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http://www.cdc.gov/qfever/index.html

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Last updated 11/4/2014

Prepared by Kirsten Knutson and Curtis L. Fritz, Infectious Diseases Branch

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 876 animal rabies cases from 2009 through 2012. Reported animal cases increased in California by 11.5 percent from 226 in 2009 to 252 in 2012.

- Among animal rabies cases, the most frequently reported species were bats (723, 82.5 percent), skunks (95, 10.8 percent), and foxes (51, 5.8 percent).

- The annual number of rabid bats reported to CDPH increased by 61.0 percent from 141 in 2009 to 227 in 2012. Rabid bats were most frequently reported from the South Coast (31.4 percent of 723), Bay Area (14.2 percent), and Far North (12.0 percent) regions.

- The annual number of rabid skunks reported to CDPH decreased by 63.6 percent from 44 in 2009 to 16 in 2012. Rabid skunks were most frequently reported from the Central Coast (42.1 percent of 95), Sacramento Metro (30.5 percent), and Sierra (10.5 percent) regions.

- During 2009-2012, 2 human cases of rabies were reported to CDPH. One case-patient had an unknown exposure in Humboldt County and the other had contact with a bat in Contra Costa County.

- Appropriate domestic and wild animal management, animal vaccination programs, identification and medical management of persons exposed to potentially rabid animals, public education about strategies to avoid animal bites, and avoiding wild animal contact provide the best opportunities for reducing rabies in humans and animals.

Background

Human rabies is an uncommon but important viral zoonotic disease in the United States (US); between 1 and 8 cases are reported annually[1]. In the US, rabies is identified more frequently in certain wild animal species than in domestic animals.

Distinct strain variants of rabies virus are maintained in populations of bats and skunks in California. Contact with the saliva of a rabid animal by direct bite is the most typical means of transmission of rabies virus. Rarely, rabies can be transmitted through contact of infectious saliva with open wounds or mucous membranes, and via transplant of organs and tissues from an undiagnosed donor.

Incubation of rabies in humans is variable and sometimes prolonged (7 days to 6 years). After an initial prodromal phase (headache, fever, malaise, anxiety, and non-specific neuropathies), patients rapidly progress to severe encephalomyelitis. Rabies is almost invariably fatal; no treatment protocol has proved reliably effective once clinical signs appear. Guidance on public health investigation and management of potentially exposed humans, and on surveillance and management of animals subject to rabies in California, are available elsewhere[1,2].

We describe here the epidemiology of animal and human rabies in California from 2009 through 2012. The epidemiological description of animal and human rabies for the 2001-2008 period can be found in the Epidemiologic Summary of Animal and Human Rabies in California, 2001-2008[3]. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes[4].

California reporting requirements and surveillance case definitions

California Code of Regulations, Title 17, requires health care providers to report cases of suspected human or animal rabies to the local health officer (LHO) immediately by telephone. Laboratories must also notify the LHO when testing yields evidence suggestive of rabies; written notification must occur within one working day after the health care provider has been notified. Additionally, regulations require that all persons must notify the LHO if they have knowledge of persons or animals bitten by a potentially rabid animal, persons bitten by any mammal, or the whereabouts of an animal suspected to have rabies. In areas declared by CDPH to be rabies areas, the LHO must also be notified of any person who is bitten by an animal of a species subject to rabies, whether or not the animal is suspected of having rabies[2]. During the surveillance period, all counties in California were declared rabies areas.

California regulations require LHOs to report to CDPH cases of human and animal rabies. For the surveillance period, CDPH officially counted cases that satisfied the surveillance case definition published by the U.S. Centers for Disease Control and Prevention (CDC). CDC defined a case of animal rabies as one with a positive direct fluorescent antibody test (preferably performed on central nervous system tissue) or isolation of rabies virus in cell culture or in a laboratory animal. A human rabies case was defined as a patient for whom Lyssavirus was detected by direct fluorescent antibody in a clinical specimen (preferably the brain or the nerves surrounding hair follicles in the nape of the neck), or Lyssavirus was isolated in cell culture or in a laboratory animal from saliva or central nervous system tissue, or Lyssavirus specific antibody was detected in the cerebrospinal fluid (CSF) or serum by indirect fluorescent antibody (IFA) test or an antibody titer greater than or equal to 5 (complete neutralization), or Lyssavirus RNA was detected in saliva, CSF, or tissue using reverse transcriptase-polymerase chain reaction (RT-PCR).

Epidemiology of rabies in California

Animal cases

During the surveillance period, CDPH received reports of 876 animal rabies cases. Animal cases occurred in bats (723, 82.5 percent), skunks (95,10.8 percent), foxes (51, 5.8 percent), dogs (3, 0.3 percent), cats (1, 0.1 percent), raccoons (1, 0.1 percent), coyote (1, 0.1 percent), and cattle (1, 0.1 percent).

The annual number of rabid animals reported to CDPH increased by 11.5 percent from 226 in 2009 to 252 in 2012 [Figure 1]. The annual number of rabid bats increased by 61.0 percent from 141 in 2009 to 227 in 2012 [Figure 2]. The annual number of rabid skunks decreased by 63.6 percent from 44 in 2009 to 16 in 2012, and the annual number of rabid foxes decreased by 85.4 percent from 41 in 2009 to 6 in 2012.

Rabid bats were most frequently reported from the South Coast (31.4 percent of 723), Bay Area (14.2 percent), and Far North (12.0 percent) regions. The regions with the greatest number of bat cases reported remained constant from the combined years of 2001 through 2008 to the combined years of 2009 through 2012; however, the highest proportion of reported cases shifted from the Bay Area to the South Coast region [Figure 3]. Rabid skunks were most frequently reported from the Central Coast (42.1 percent of 95), Sacramento Metro (30.5 percent), and Sierra (10.5 percent) regions. From the combined years of 2001 through 2008 to the combined years of 2009 through 2012, the Central Coast region persisted in having the highest proportion of reported rabid skunk cases [Figure 3].

Human cases

During the surveillance period, 2 human cases

of rabies were reported in California, one of which was fatal. The first case-patient, in 2011, was a female child from Humboldt County who survived. Diagnosis was by serology, and no rabies virus was recovered for strain typing. The circumstances of exposure were unknown. The second case-patient, in 2012, was an adult male who had contact with a bat in Contra Costa County and was diagnosed after onset, hospitalization, and death outside of the United States. The rabies virus recovered from this patient was identified as a Mexican free-tailed bat variant.

Figure 1. Number of reported animal rabies cases in California by year

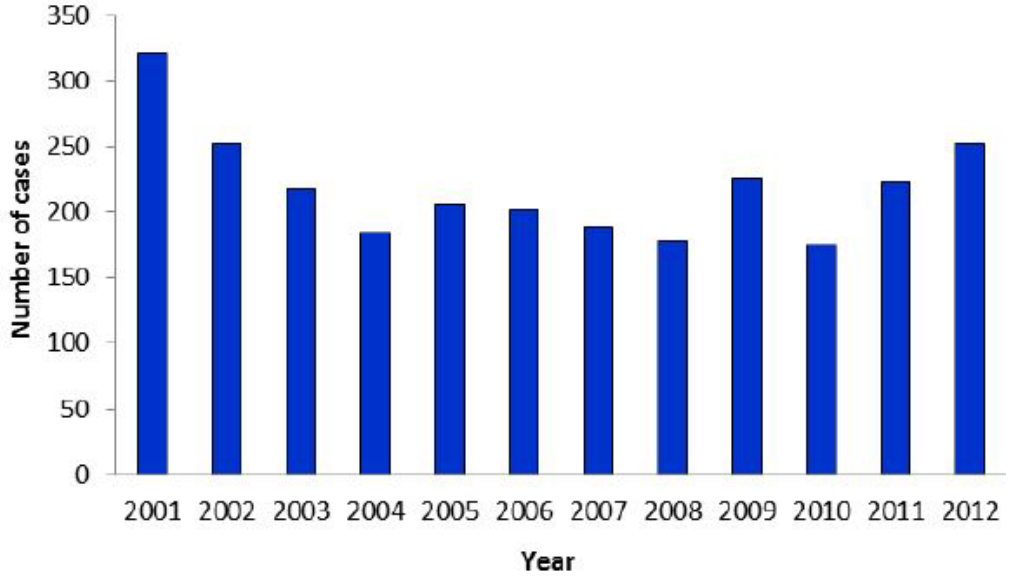


Figure 2. Reported animal cases of rabies in California by species and year

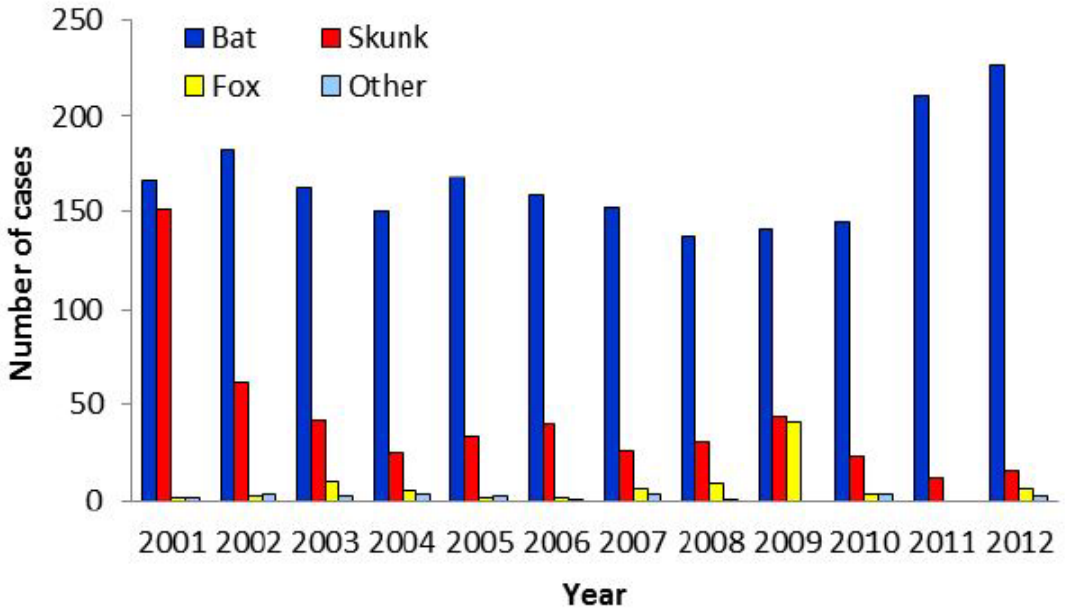
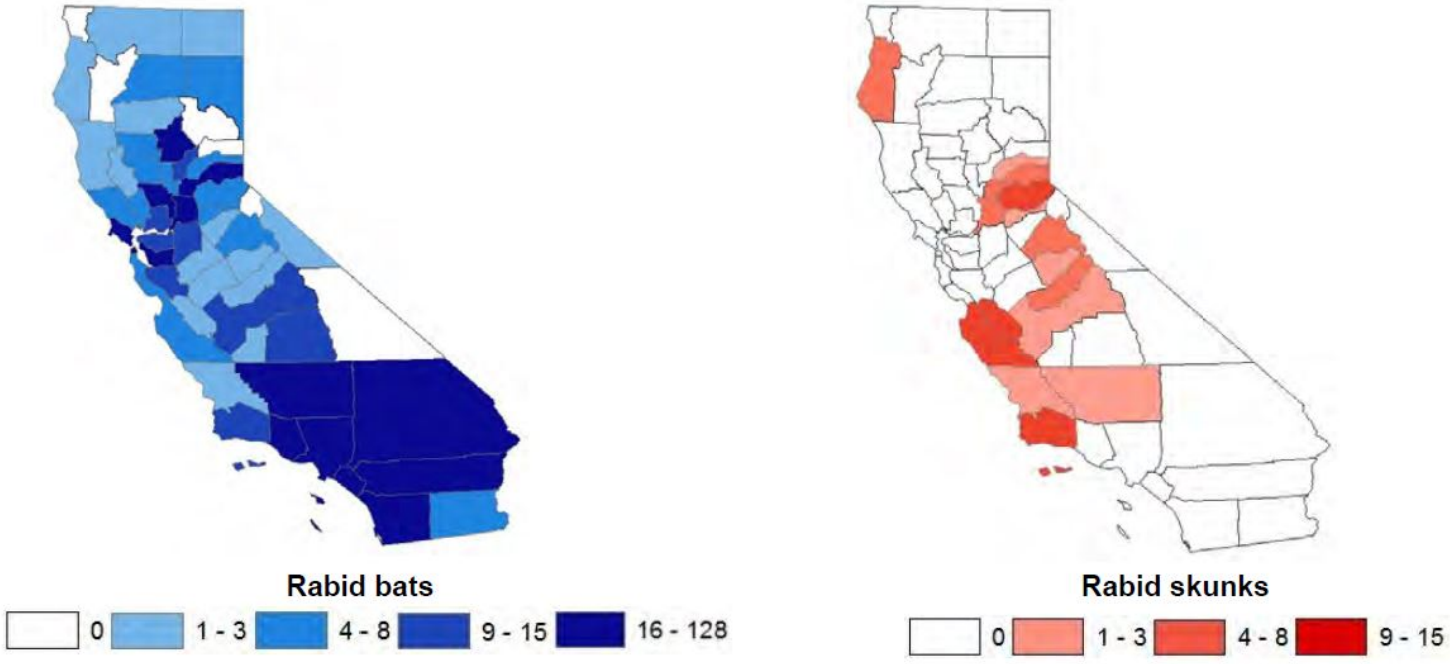


Figure 3. Reported rabid bats and skunks in California by location found, 2009-2012



Comment

Human rabies remained rare in California during the surveillance period. Rabies continues to be an almost invariably fatal disease; the recovery of the 2011 case-patient was exceptional and only the third known instance of an unvaccinated patient surviving rabies[5]. California's 2012 case-patient was the first California case with medical management and diagnosis abroad[6]. The number of rabid wild animals reported to CDPH increased during the surveillance period 2009-2012 (876) compared to the previous four-year surveillance period, 2005-2008 (773). The greatest increase occurred in foxes with a 168.4 percent increase; 80 percent of all 2009-2012 rabid foxes occurred in 2009 during an epidemic in Humboldt County. There was an 18 percent increase in reported rabid bats between the period 2005-2008 and 2009-2012, with the greatest increase in the South Coast region (139 percent).

Appropriate domestic and wild animal management, animal vaccination programs, assessment and medical management of persons exposed to potentially rabid animals, public education about strategies to avoid animal bites, and minimizing contact with wild animals provide the best strategies for reducing rabies in humans and animals.

References and resources

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Last updated: 02/10/2014

Prepared by Elizabeth Roberts, Curtis Fritz, Farzaneh Tabnak, acknowledging Susan Williams for data compilation

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 18,664 cases of non-typhoidal salmonellosis infections with estimated symptom onset dates from 2009 through 2012, corresponding to an average annual incidence rate of 12.47 cases per 100,000 population.
- During 2009-2012, salmonellosis incidence rates were relatively stable. The minimum rate occurred in 2011 (10.74 per 100,000) and the maximum rate occurred in 2010 (13.56 per 100,000).
- Average annual incidence rates were highest among children under 1 year of age (54.90 per 100,000) and 1 to 4 years of age (36.47 per 100,000), followed by children 5 to 14 years of age (14.20 per 100,000) and adults 65 years of age or older (13.27 per 100,000).
- From 2009 through 2012, CDPH received reports of 69 outbreaks of foodborne salmonellosis involving more than 900 California case-patients.
- Preventing contamination and cross-contamination during the processing and production of foods, combined with education of consumers and foodhandlers about food safety may provide the best opportunities for preventing and controlling salmonellosis.

Background

Salmonella is among the most commonly reported enteric bacterial pathogens in the United States, causing an estimated 1.2 million infections, 23,000 hospitalizations, and 450 deaths each year.[1,2] Non-typhoidal *Salmonella* is a commonly identified etiology in foodborne disease outbreaks, though most salmonellosis cases are not associated with outbreaks. From 2009 through 2012, the *Salmonella* serotypes most frequently isolated from human cases nationally were *S. enteritidis, S. typhimurium, S. newport,* and *S. javiana.*[3] The national Healthy People 2020 target objective for salmonellosis is for an incidence rate lower than 11.4 new cases per 100,000 population.

Consuming foods directly or indirectly contaminated with the feces of infected animals is the leading source of *Salmonella* infections. However, direct contact with infected people, consumption of foods handled by ill persons or exposure to infected animals and their environments (notably birds, petting zoo or farm animals, and reptiles such as pet turtles) may also result in infection.

Acute illness, usually gastroenteritis, occurs after an incubation period of 12 to 72 hours, and lasts 4 to 7 days; treatment with antibiotics is not usually necessary.[4] Some patients, especially young children, the elderly, and immunocompromised persons, may develop severe illness and require hospitalization. Rarely, *Salmonella* can cause invasive disease, including meningitis, pneumonia, and sepsis; death can result. Reactive arthritis is a rare long-term complication[5].

This report describes the epidemiology of non-typhoidal salmonellosis infections in California from January 1, 2009 through December 31, 2012 reported by December 2014. Data for 2012 are provisional and may differ from data in future publications. For a complete discussion of the definitions, methods, and limitations associated with

this report, please refer to the Technical Notes.[6] The epidemiologic description of non-typhoidal salmonellosis for the 2001-2008 surveillance period can be found in the *Epidemiologic Summary of Salmonellosis in California, 2001-2008*[7].

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report suspected cases of salmonellosis to their local health department within one working day of identification or immediately by telephone if an outbreak is suspected. Laboratories are also required to report laboratory testing results suggestive of *Salmonella* infection to either the California Reportable Disease Information Exchange (CalREDIE) (via electronic laboratory reporting) or the local health department; reporting must occur within one working day after the health care provider has been notified. A culture of the organism upon which the diagnosis of salmonellosis was established must be submitted to the local public health laboratory and then onto the State Microbial Diseases Laboratory for definitive identification and serotyping.

Local health officers are required by regulation to report to CDPH cases of salmonellosis. CDPH counted cases that satisfied the Centers for Disease Control and Prevention (CDC)/Council of State and Territorial Epidemiologists' surveillance case definition of a confirmed or probable case. During the surveillance period, a confirmed case was defined as one from whom *Salmonella* (excluding *S. typhi*) was isolated from a clinical specimen, including laboratory-confirmed asymptomatic and extraintestinal infections. A probable case had clinically compatible illness and an established epidemiologic link to a laboratory-confirmed case.[8]

Epidemiology of salmonellosis in California

CDPH received reports of 18,664 cases of non-typhoidal salmonellosis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 12.47 cases per 100,000 population. Incidence rates during the 2009-2012 surveillance period were relatively stable, showing moderate fluctuations similar to those observed since 2001 [Figure1]. Rates declined from the surveillance period's maximum in 2010 of 13.56 per 100,000 to the minimum in 2011 of 10.74 per 100,000. In 2012, incidence rates increased to 12.36 per 100,000. During the surveillance period, 76 (0.4 percent) case-patients were reported to have died by the time of case report. Case fatality rates were greatest among case-patients 65 years of age or older (1.6 percent).

Average annual salmonellosis incidence rates for the surveillance period were highest among children under 1 year of age (54.90 per 100,000) and 1 to 4 years of age (36.47 per 100,000), followed by children 5 to 14 years of age (14.20 per 100,000) and adults 65 years of age or older (13.27 per 100,000, not shown) [Figure 2]. Incidence rates were most variable over time among children under 1 year: during 2009-2012, rates ranged from 66.73 per 100,000 in 2009 to 43.83 per 100,000 in 2011.

The ratio of male to female case-patients was 1.0:0.9. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (23.0 percent). However, salmonellosis cases with complete data reported Hispanic ethnicity slightly more frequently than would be expected based on the demographic profile of California [Figure 3].

County-specific incidence rates during the

surveillance period ranged from 0 to 33.96 per 100,000 [Figure 4]. Average annual incidence rates for the surveillance period were higher in Northern California (13.63 per 100,000) than Southern California (11.57 per 100,000). The Bay Area (15.35 per 100,000), San Joaquin Valley (13.64 per 100,000) and Central Coast (12.83 per 100,000) regions reported the highest average annual incidence rates during the surveillance period.

From 2009 through 2012, there were 69 foodborne outbreaks of salmonellosis involving more than 900 California case-patients.[9] There was no discernable trend in the number of outbreaks. The most common serotypes reported among outbreaks were *S. typhimurium* (12 outbreaks, 168 California case-patients), *S. enteritidis* (9 outbreaks, more than 140 California case-patients), *S. heidelberg* (9 outbreaks, 111 California case-patients), and *S. newport* (6 outbreaks, 42 California case-patients). Exposure was confined to California for 40 (58.0 percent) of the outbreaks (638 California case-patients were involved), while for 29 (42.0 percent) outbreaks, California was one of multiple states where exposure occurred (at least 262 California case-patients were involved in these multi-state outbreaks). Among 49 (71.0 percent) outbreaks with a confirmed food vehicle, the most common types of foods implicated were multiple-ingredient foods (8, 16.3 percent), fruits (6, 12.2 percent), turkey (5, 10.2 percent), chicken (4, 8.2 percent), pork (4, 8.2 percent), and sprouts (4, 8.2 percent).[10] A notable outbreak involving California residents was a large multi-state *S. enteritidis* outbreak in 2010 that included nearly 2000 reported cases nationwide and was associated with consumption of shell eggs from a company in Iowa.[11]

Figure 1. California salmonellosis case counts and incidence rates by estimated year of illness onset

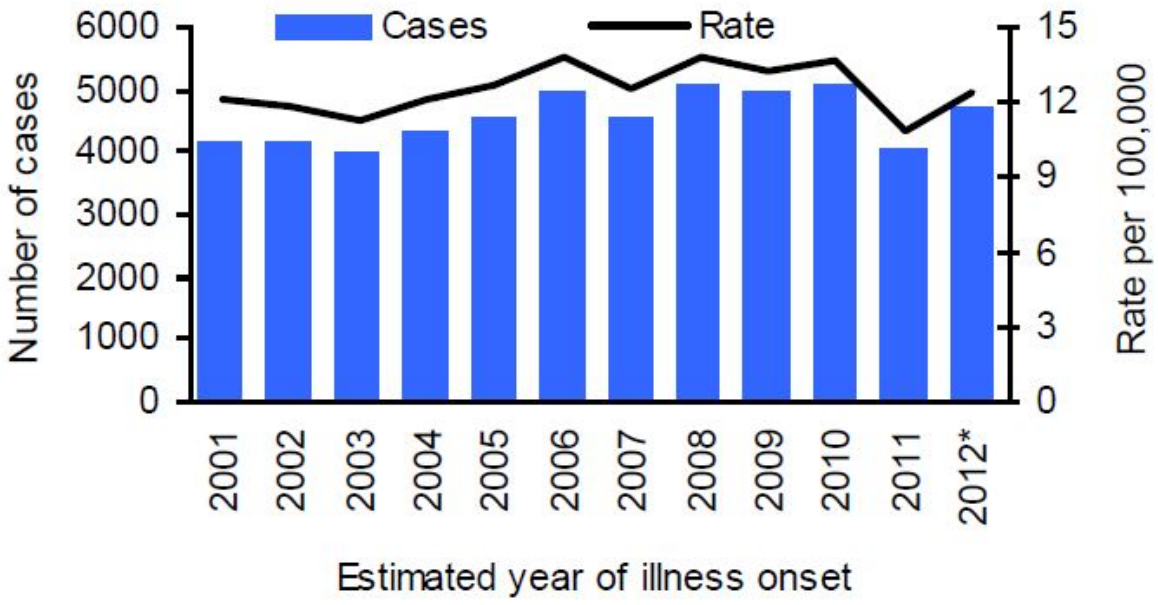


Figure 2. California salmonellosis incidence rates by age group and estimated year of illness onset

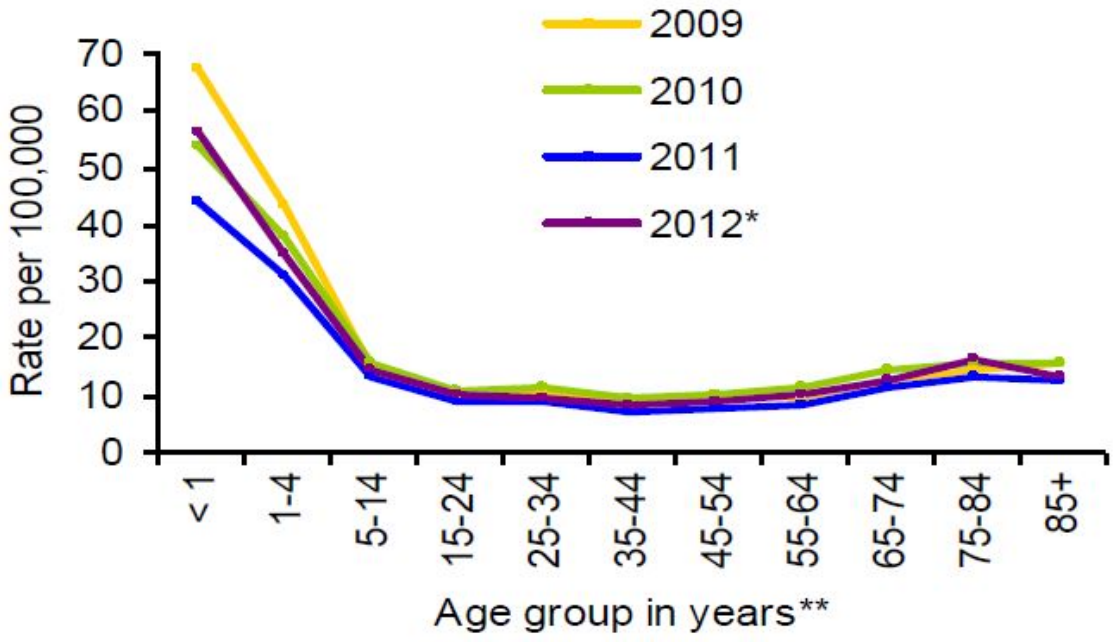
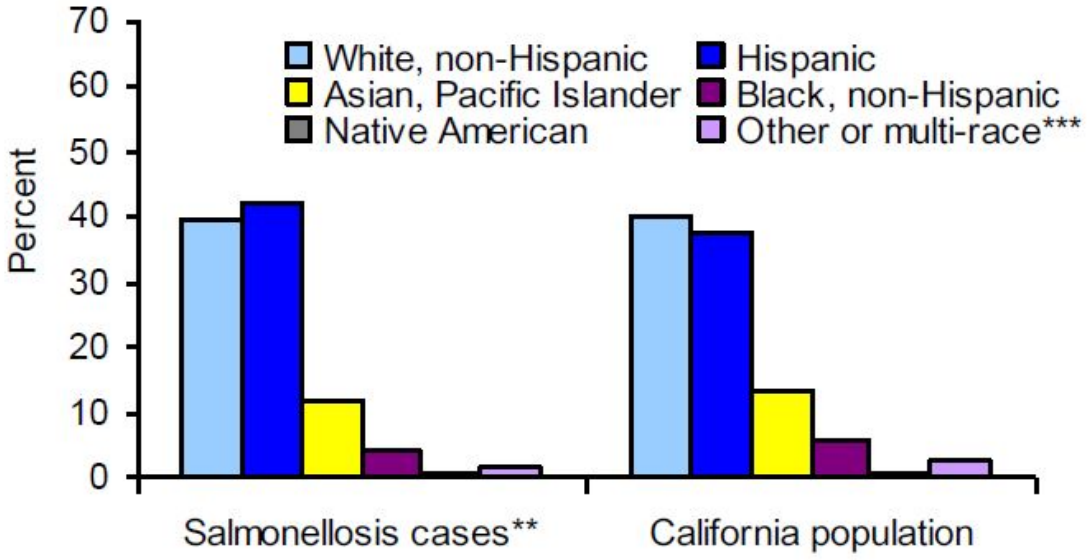


Figure 3. California salmonellosis cases and population by race/ethnicity, 2009 - 2012*



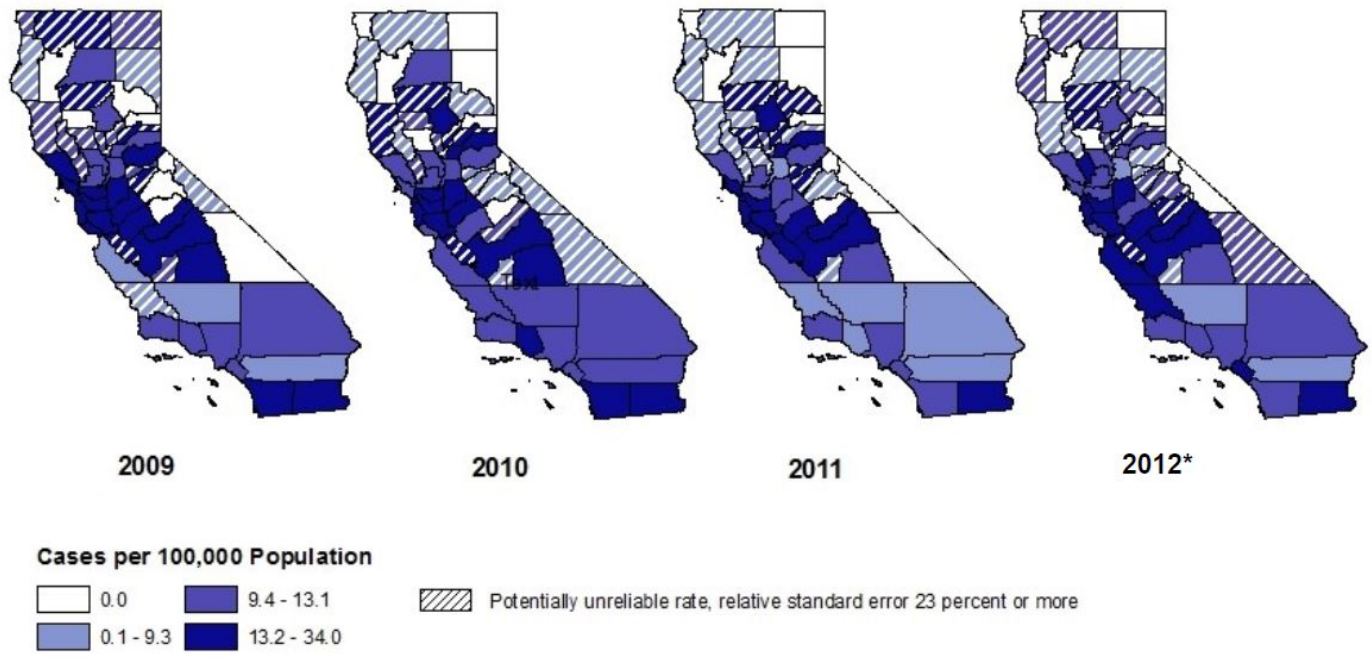
Notes for Figures 1-4

*2012 data are provisional

**Unknowns were excluded

***Includes cases who identified 'other' as their race and

Figure 4. California county-specific salmonellosis incidence rates by estimated year of illness onset



Comment

Incidence rates of reported salmonellosis infection among Californians were relatively stable from 2009 through 2012, with minor fluctuations in rates similar to the previous surveillance period. The rate in 2011 was the lowest in more than a decade. However, rates increased at the end of the surveillance period to essentially equal the average annual rate during the 2001-2008 surveillance period. The statewide 2009-2012 average annual incidence rate of salmonellosis was greater than the national Healthy People 2020 target objective, though California met the target in 2011. Salmonellosis infections are often not diagnosed and not reported, so rates may be underestimated.[2,12]

The age, race/ethnicity and gender distribution of cases incident in California from 2009 through 2012 remained fairly consistent with that of 2001 through 2008.[7] Incidence rates by age group, though, particularly in children under 1 year old, varied more during the 2009-2012 surveillance period than during the previous surveillance period.

Compared to salmonellosis incidence rates reported nationally during 2009 through 2012, rates reported among Californians were lower each year. However, the distribution by age group of incident cases in California and those reported nationally was similar: children under 5 years of age experienced the highest rates of salmonellosis.[13-16] Also during the surveillance period, three of the four serotypes most commonly involved in California salmonellosis outbreaks *S. typhimurium*, *S. enteritidis* and *S. newport* were the three serotypes most frequently isolated from lab-confirmed *Salmonella* infections nationally.[3]

Preventing contamination and cross-contamination during the processing and production of foods, including both foods of animal origin and produce, combined with education of consumers and foodhandlers on food safety may provide the best opportunities for preventing and controlling salmonellosis.

References and resources

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[7]Epidemiological Summaries of Selected General Communicable Diseases in California, 2001-2008: Salmonellosis. http://www.cdph.ca.gov/data/statistics/Pages/EpiSummariesCDsCA- 01-08.aspx

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Last updated 9/17/2015

Prepared by Kirsten Knutson, Farzaneh Tabnak, Akiko Kimura, Jeff Higa, and Debra Gilliss, Infectious Diseases Branch.

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of the following cases with estimated symptom onset dates from 2009 through 2012:
  - 1,007 cases of *E. coli* O157 (average annual incidence rate of 0.67 cases per 100,000),
  - 674 cases of *E. coli* non-O157 (average annual incidence rate of 0.45 cases per 100,000), and
  - 165 cases of hemolytic uremic syndrome (HUS) (average annual incidence rate of 0.11 cases per 100,000).

- Reported incidence rates of *E. coli* non-O157 infection increased six-fold from 2009 through 2012 to essentially equal *E. coli* O157 infection rates by 2012 (0.79 and 0.76 per 100,000 rates of *E. coli* O157 and non-O157 infection, respectively).

- The average annual incidence rates for the four-year surveillance period were highest among:
  - *E. coli* O157 patients 1 to 4 years of age (3.47 per 100,000), 5 to 14 years of age (1.24 per 100,000), and children less than 1 year (0.89 per 100,000),
  - *E. coli* non-O157 patients 1 to 4 years of age (3.55 per 100,000) and children less than 1 year old (1.88 per 100,000), and
  - HUS patients 1 to 4 years of age (1.01 per 100,000), 5 to 14 years of age (0.23 per 100,000), and children less than 1 year (0.20 per 100,000).

- During the surveillance period, 108 (10.7 percent) *E. coli* O157 infections and 9 (1.3 percent) *E. coli* non-O157 infections progressed to HUS.

- From 2009 through 2012, there were 14 confirmed foodborne outbreaks of Shiga toxin-producing *E. coli* (STEC) involving 54 California case-patients. Thirteen (92.9 percent) of the outbreaks were confirmed to have been caused by *E. coli* O157, and 1 (7.1 percent) outbreak was caused by *E. coli* non-O157.

- Preventing contamination and cross-contamination during the processing and production of foods, avoiding raw and unpasteurized dairy products and juices, combined with consumer education may pro-vide the best opportunities for preventing and controlling *E. coli* O157 and non-O157 infections and HUS.

Background

Shiga toxin-producing *Escherichia coli* (STEC) are important enteric bacterial pathogens in the United States (US), causing an estimated 265,000 infections, more than 3600 hospitalizations, and 30 deaths each year[1]. These diarrhea- causing *E. coli* are named for the potent cytotoxins (Shiga toxins 1 and 2) they produce. *E. coli* O157 is the most frequently reported STEC serogroup in the US, causing approximately 95,000 infections, mostly by serotype *E. coli* O157:H7. The many other STEC serogroups, referred to in this report collectively as *E. coli* non-O157, cause approximately 170,000 infections nationwide each year[2].

Exposure to the feces of a contaminated animal (STEC live in ruminant animals, like cattle) or an infected human can result in illness. Ingesting or handling contaminated food is a common cause of STEC, but illness can also result from direct contact with contaminated animals or their environments, consuming contaminated beverages, or direct exposure to infected people or their personal items[2,3].

Acute illness, usually gastroenteritis, typically occurs after an incubation period of 3 to 4 days, but may occur anywhere from 1 to 10 days after exposure. Illness may be more severe in young children and elderly patients. Overall, *E. coli* O157 appears to be more likely to cause severe illness than *E. coli* non-O157, though illness severity is also affected by the virulence characteristics of the infecting strain[2].

The national Healthy People (HP) 2020 target objective for *E. coli* O157 incidence is for an incidence rate lower than 0.60 cases per 100,000 population. There is no HP 2020 objective for *E. coli* non-O157 incidence.

About 5 to 10 percent of STEC case- patients develop hemolytic uremic syndrome (HUS), a delayed, life-threatening complication of a STEC infection. HUS is a disease characterized by hemolytic anemia, acute kidney failure, and often a low platelet count, and is the leading cause of short-term acute renal failure in US children[4]. Progression to HUS occurs on average 7 days after symptom onset, and may be delayed until after STEC infection has cleared[2]. Most cases the of HUS are caused by *E. coli* O157, but *E. coli* non-O157 can also cause HUS[4,5].

For surveillance purposes, post-diarrheal HUS cases without laboratory evidence of an STEC infection are presumed to be related to an undetected STEC infection. The national HP 2020 target objective for HUS incidence is for an incidence rate lower than 1 case per 100,000 children under 5 years of age. Described in this report is the epidemiology of *E. coli* O157 and *E. coli* non-O157 infections in California from January 1, 2009 through December 31, 2012 reported by December 4, 2014. The epidemiology of HUS is also described, including HUS cases in which STEC was identified and post-diarrheal HUS cases without laboratory evidence of an STEC infection. Data for 2012 are provisional and may differ from data in future publications. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to the Technical Notes[6]. The epidemiological description of STEC infections and HUS for the 2001-2008 surveillance period can be found in the Epidemiologic Summary of STEC-related infections and illnesses in California, 2001-2008[7].

California reporting requirements and surveillance case definitions

California Code of Regulations, Title 17, requires health care providers to report suspected cases of *E. coli* O157 infection, *E. coli* non-O157 (since late 2006) infection, and post-diarrheal HUS to their local health department immediately by telephone. Clinical and reference laboratories are also required to report laboratory testing results suggestive of E. coli O157 or *E. coli* non-O157 infection to either the California Reportable Disease Information Exchange (CalREDIE) (via electronic laboratory reporting) or the local health department; reporting must occur within one working day after the health care provider has been notified.

California regulations require local health officers to report to CDPH cases of *E. coli* O157 and *E. coli* non-O157 infection, and post-diarrheal HUS. California officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC)/Council of State and Territorial

Epidemiologists' (CSTE) surveillance case definition of a confirmed or probable case[8]. During 2009 through 2012, the confirmed and probable case definitions for STEC infections were:

- A confirmed case was one with isolation of STEC from a clinical specimen. Serotype O157:H7 isolates were assumed to be Shiga toxin-producing, while for all other serotypes, evidence of toxin production or the presence of Shiga toxin genes was required.
- A probable case was one with isolation of *E. coli* O157 from a clinical specimen without confirmation of H antigen or Shiga toxin production, or a clinically compatible case that either was epidemiologically linked to a confirmed or probable case or had an elevated antibody titer to a known Shiga toxin-producing *E. coli* serotype. The confirmed and probable case definitions for HUS were:
- A confirmed case was one with anemia with microangiopathic changes or renal injury evidenced by either hematuria, proteinuria, or elevated creatinine levels that began within three weeks of onset of acute or bloody diarrhea.
- A probable case was one with laboratory evidence of HUS but an unclear history of diarrhea or a case that met all criteria for a confirmed case but did not have confirmed microangiopathic changes.

Cases of Shiga toxin detected in feces without further culture confirmation or serogroup identification are also reportable according to California regulation. This requirement was added in late 2006 because some commercial laboratories now test for Shiga toxin without subsequently confirming identification by culture or other means. However, Shiga toxin detected in feces without culture confirmation is not designated by CDC as nationally notifiable and lacks a standard CDC/ CSTE case definition, so is not described in this report.

Epidemiology of STEC Infections and HUS

E. coli O157 Infections

CDPH received reports of 1,007 cases of *E. coli* O157 infection with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.67 cases per 100,000 Californians. Since 2001, incidence rates fluctuated moderately: during 2001-2008, the rate ranged from 0.52 to 0.86 per 100,000, and during 2009-2012, the rate increased from 0.58 per 100,000 in 2010 to 0.79 per 100,000 in 2012 [Figure 1]. A total of 108 (10.7 percent) *E. coli* O157 infections progressed to HUS by the time of case report [Figure 2]. Of 299 *E. coli* O157 case-patients under 5 years of age, 55 (18.4 percent) developed HUS (not shown). During the surveillance period, two (0.2 percent) *E. coli* O157 case-patients were reported to have died by the time of case report.

During 2009-2012, the average annual incidence rates for *E. coli* O157 infection were highest among children 1 to 4 years of age (3.47 per 100,000), 5 to 14 years of age (1.24 per 100,000), and children less than 1 year old (0.89 per 100,000) [Figure 3]. The ratio of male to female case-patients was 0.9:1.0. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (18.7 percent). However, *E. coli* O157 cases with complete data reported White non-Hispanic race/ethnicity more frequently than would be expected based on the demographic profile of California [Figure 4].

County-specific average annual incidence rates of *E. coli* O157 infection during the surveillance period ranged from 0 to 7.86 per 100,000 [Figure 5]. Average annual incidence rates were 3.4 times higher in Northern California (1.12 per 100,000) than in Southern California (0.33 per 100,000). The Far North (1.85 per 100,000), San Joaquin Valley (1.23 per 100,000) and Central Coast (1.19 per 100,000) regions reported the highest average annual incidence rates during the surveillance period.

E. coli Non-O157 Infections

CDPH received reports of 674 cases of *E. coli* non-O157 infection with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.45 cases per 100,000 Californians. Incidence rates for *E. coli* non-O157 infections increased by nearly 600 percent from 2009 (40 cases; 0.11 per 100,000) to 2012 (288 cases; 0.76 per 100,000). In contrast, rates were stable from 2006 (when reporting was first required in California) to the beginning of the current surveillance period (0.10 per 100,000 in 2006, 2007, and 2008) [Figure 1]. A total of 9 (1.3 percent) *E. coli* non-O157 infections progressed to HUS by the time the case was reported [Figure 2], and 2 (0.3 percent) case-patients were reported to have died.

The average annual incidence rates for *E. coli* non-O157 infection during the surveillance period were highest among children 1 to 4 years of age (3.55 per 100,000) and children less than 1 year old (1.88 per 100,000) [Figure 3]. The ratio of male to female case-patients was 0.9:1.0. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (13.2 percent). However, *E. coli* non-O157 cases with complete data reported Hispanic race/ethnicity more frequently than would be expected based on the demographic profile of California [Figure 4].

County-specific average annual incidence rates of *E.coli* non-O157 during the surveillance period ranged from 0 to 1.83 per 100,000. Average annual incidence rates were nearly the same in Northern California (0.46 per 100,000) and Southern California (0.44 per 100,000) [Figure 5]. The Central Coast (1.06 per 100,000) and San Diego (0.59 per 100,000) regions reported the highest *E. coli* non-O157 average annual incidence rates during the surveillance period.

HUS

CDPH received reports of 165 patients with HUS with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.11 cases per 100,000 Californians. Rates remained stable during 2009 to 2012 (range: 0.10 to 0.13 per 100,000), and have varied little since the 2001-2008 surveillance period (range: 0.09 to 0.14 per 100,000) [Figure 6]. The majority of HUS (108, 65.5 percent) diagnoses were associated with a laboratory-confirmed *E. coli* O157 infection, 9 (5.5percent) HUS patients had an associated laboratory-confirmed *E. coli* non-O157 infection, and 48 (29.1 percent) HUS patients did not have laboratory evidence of an STEC infection [Figure 2]. Two (1.2 percent) HUS case-patients were reported to have died (one patient with a laboratory-confirmed *E. coli* O157 infection and one patient with a non-O157 infection, as described above).

Average annual HUS incidence rates were highest among children 1 to 4 years of age (1.01 per 100,000), 5 to 14 years of age (0.23 per 100,000), and less than 1 year (0.20 per 100,000) [Figure 3]. The ratio of male to female patients was 0.7:1.0. Incidence rates by race/ethnicity were not calculated due to missing data (15.2 percent). However, HUS patients reported White non-Hispanic race/ethnicity

more frequently than would be expected based on the demographic profile of California [Figure 4]. The average annual incidence rate for HUS for the surveillance period was 3.8 times higher in Northern California (0.19 per 100,000) than in Southern California (0.05 per 100,000) [Figure 5]. The San Joaquin Valley, (0.26 per 100,000), Far North (0.25 per 100,000) and Central Coast (0.19 per 100,000) regions reported the highest average annual incidence rates during the surveillance period.

STEC Outbreaks

According to CDC's National Outbreak Reporting System data[9], from 2009 through 2012, there were 14 foodborne outbreaks of STEC involving 54 California residents. Thirteen (92.9 percent) of the outbreaks were confirmed to have been caused by *E. coli* O157, and 1 (7.1 percent) outbreak was caused by *E. coli* non-O157. Of the 14 outbreaks, 10 (71.4 percent) involved cases exposed in multiple states (27 California residents were part of these multi-state outbreaks) and 4 (28.6 percent) outbreaks were confined to California (involving 27 case- patients). Among 13 (92.9 percent) outbreaks with a confirmed food vehicle, the most commonly implicated types of foods were beef (5, 38.5 percent) and vegetable row crops (4, 30.8 percent)[10]. The largest multi-state E. coli outbreak involving California residents occurred in 2009 and included 77 case-patients from more than ten states whose infection with *E. coli* O157:H7 was associated with consumption of cookie dough; 5 (6.5 percent) case-patients were CA residents. The largest outbreak confined to California occurred in 2012 and involved 12 cases of *E. coli* O157:H7 infection associated with romaine lettuce consumption. The lone *E. coli* non-O157 outbreak was a multi- state outbreak of *E. coli* O121 associated with consumption of a frozen meal; one California case-patient was involved.

Figure 1. California *E. coli* O157 and *E. coli* non-O157 infection case counts and incidence rates by estimated year of illness onset

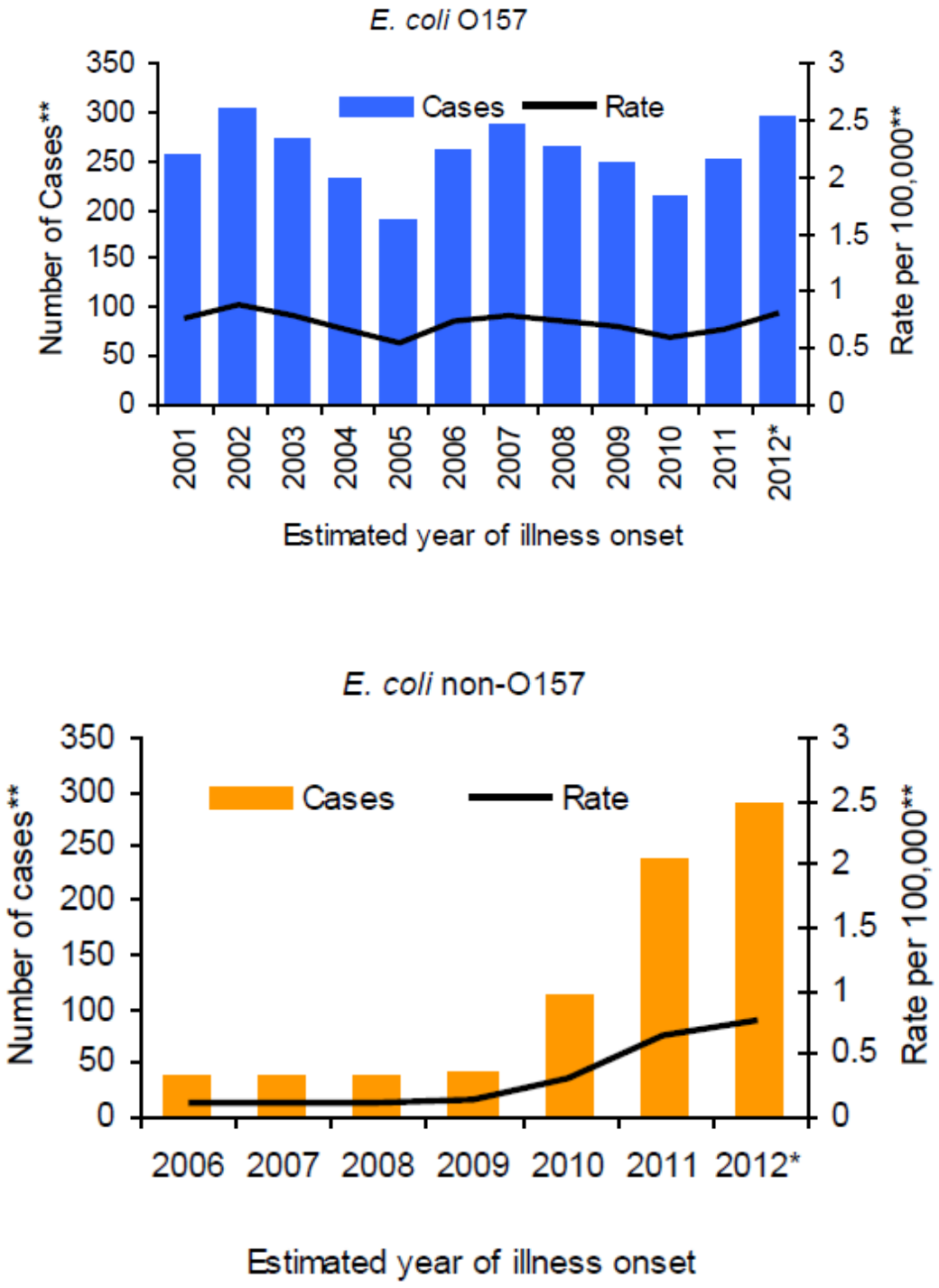


Figure 2. Venn diagram of California cases of *E. coli* O157 and *E. coli* non-O157 infection and Hemolytic Uremic Syndrome (HUS), 2009-2012*

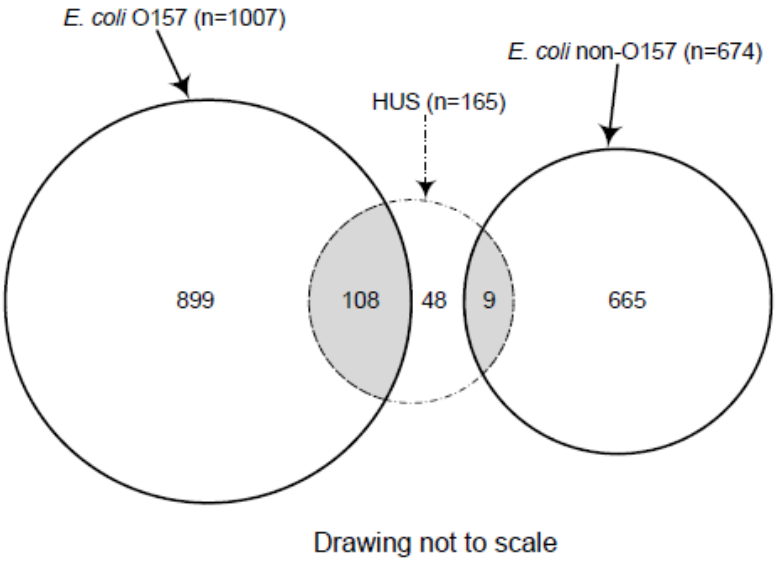


Figure 3. California *E. coli* O157 and *E. coli* non-O157 infection and HUS average annual incidence rates by age group, 2009-2012*

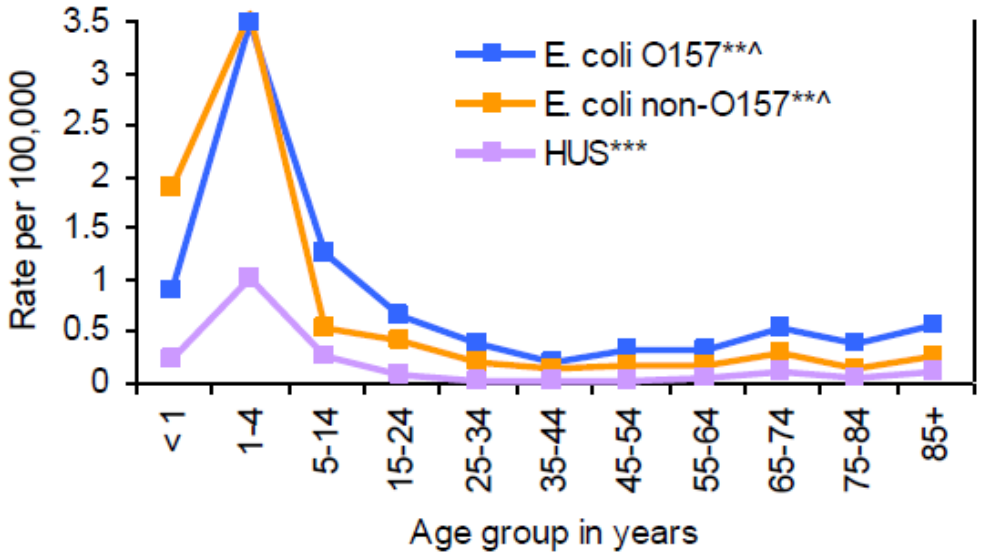
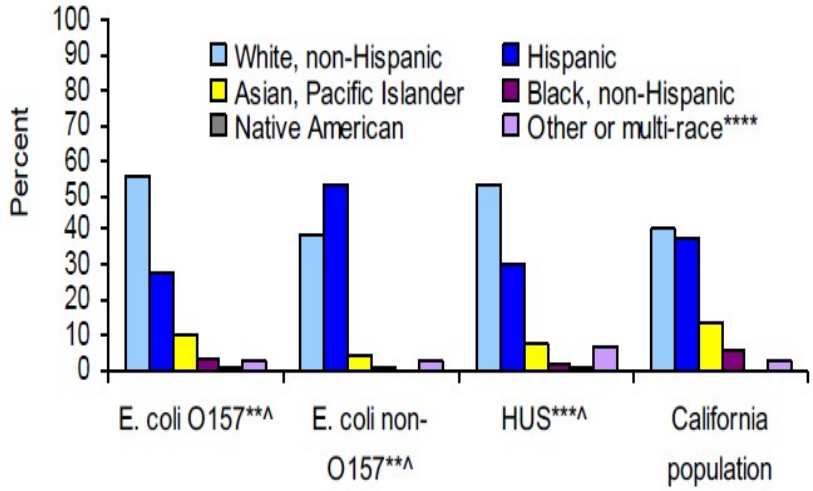


Figure 4. California *E. coli* O157 and *E. coli* non-O157 infection and HUS cases and population by race/ethnicity, 2009-2012*



Notes for Figures 1-6

*2012 data are provisional

**Includes cases accompanied by HUS

***Includes cases with laboratory evidence of STEC

****Includes cases who identified 'other' as their race or more than one race and Californians ('population') who identified more than one race

^Unknowns were excluded

Figure 5. California county-specific *E. coli* O157 and *E. coli* non-O157 infection and HUS average annual incidence rates, 2009-2012*

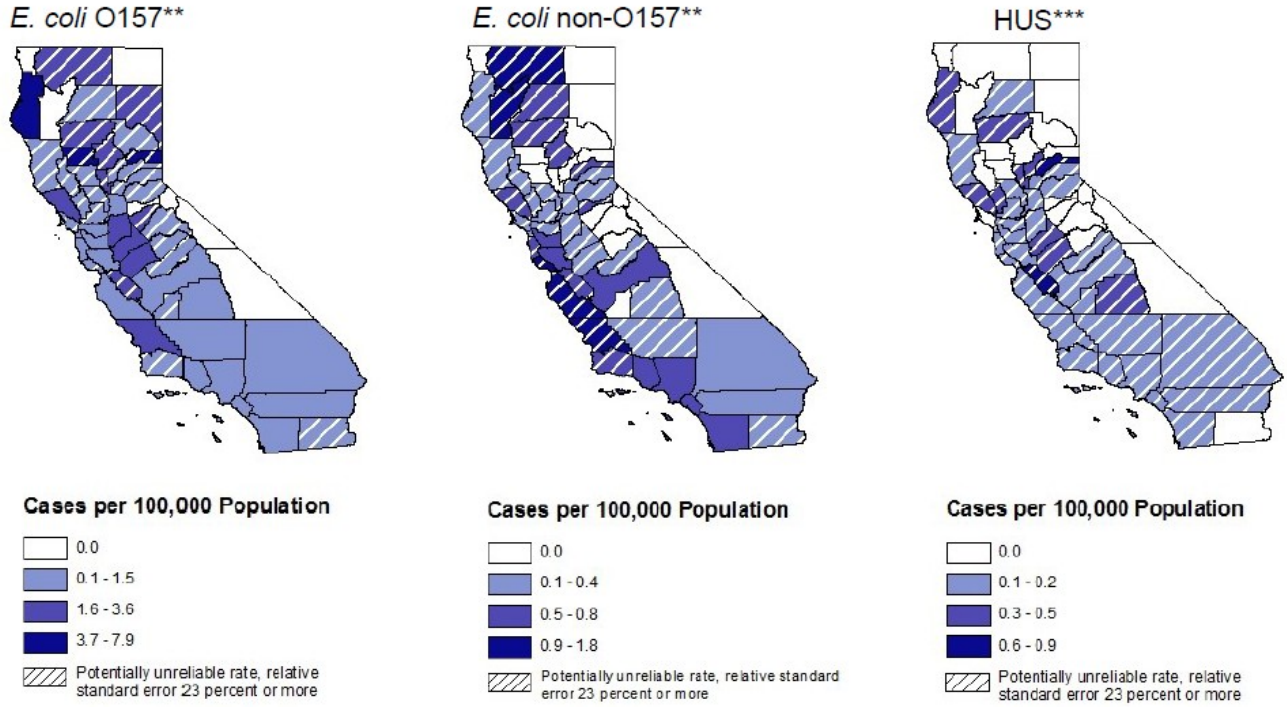
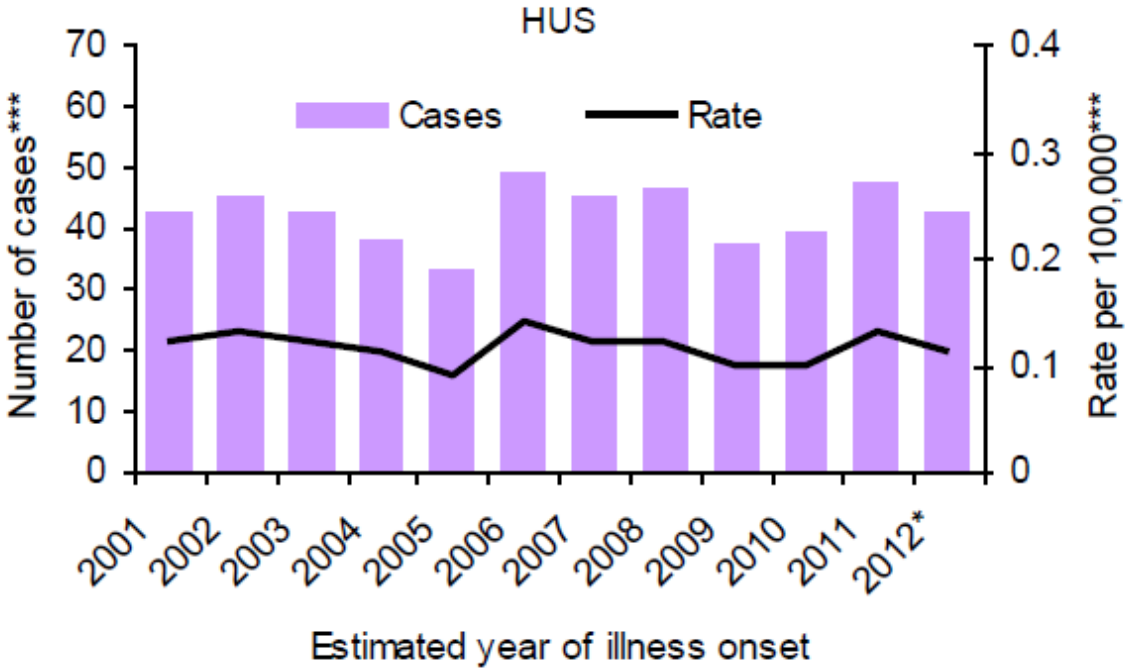


Figure 6. California HUS case counts and incidence rates by estimated year of illness onset



Comment

During 2009 through 2012, incidence rates of reported *E. coli* O157 infection among Californians fluctuated moderately but trended upwards towards the end of the surveillance period. The statewide average annual incidence rate of *E. coli* O157 infection for the surveillance period, 0.67 per 100,000, was just above the national Healthy People 2020 target objective of 0.60 per 100,000. *E. coli* O157 incidence rates among Californians during 2009-2012 were similar to those reported nationally[11].

Incidence rates of *E. coli* non-O157 infection, which became reportable in California in 2006, increased six-fold from 2009 to 2012 to essentially equal *E. coli* O157 rates by the end of the surveillance period (0.79 and 0.76 per 100,000 of *E. coli* O157 and non-O157, respectively, in 2012). This marked increase may be influenced by several factors, including increased use of Shiga toxin testing by clinical laboratories, growing awareness of reporting requirements for *E. coli* non-O157, and increasing numbers of Shiga toxin positive specimens forwarded to a public health laboratory for culture and identification. The rise in incidence may also be due to a true increase in *E. coli* non-O157 infections, due to yet undefined demographic and environmental risk factors. A rise in rates was also experienced in the US overall; *E. coli* non-O157 incidence rates, particularly in 2011 and 2012, were similar to those reported in the US[11].

HUS incidence rates among Californians were relatively stable during 2009 through 2012 (average annual incidence rate of 0.11

cases per 100,000). Nearly 10 percent of all *E. coli* O157 and non-O157 infections reported during the surveillance period progressed to HUS by the time of their report.

However, as in the US overall, the majority of HUS diagnoses in California were associated with a laboratory-confirmed *E. coli* O157 infection, while only a small proportion were associated with a laboratory-confirmed E. coli non-O157 infection[1,5].

Also similar to national trends, California children ages 1 to 4 years experienced the highest rates of *E. coli* O157 and *E. coli* non-O157 infection, as well as of HUS[11,12]. A slightly greater proportion (18.4 percent) of California *E. coli* O157 case-patients under 5 years of age had a HUS diagnosis than did nationally (about 15 percent)[12].

However, the HUS annual average incidence rate in California children under 5 years (0.85 per 100,000) was below the HP 2020 target objective of 1 case per 100,000. During the surveillance period, the higher rate of *E. coli* O157 infections and HUS in Northern California compared to Southern California is notable. The reason for the difference in rates is unknown, and may reflect regional differences in demographics or exposures. Further investigation may be warranted.

The moderately fluctuating trend over time and annual average rate of both *E. coli* O157 infection and HUS among Californians during this surveillance period were similar to those of the last surveillance period. The age and race/ethnicity distribution of *E. coli* 0157 and HUS cases during 2009-2012 remained fairly consistent with that of 2001-2008.

Rates of *E. coli* O157 and *E. coli* non-O157 infection may be underestimated for several reasons. Patients often do not seek medical attention or provide samples for diagnostic testing. Clinical specimens may not be tested properly, and isolates may not be forwarded to a public health laboratory for serotyping and strain typing. Laboratories and health care providers may not report STEC infections to local health departments.

Surveillance depends on the complete, timely, and accurate collection of data. In order to capture the burden of STEC infections in California and to develop a comprehensive public health response, it is crucial that clinical laboratories routinely test all stool specimens collected from patients with symptoms consistent with acute bacterial enteritis for the presence of Shiga toxin and attempt to culture STEC. Suspect STEC specimens must be sent to a public health laboratory for serogrouping and subtyping.

Preventing contamination and cross-contamination during the processing and production of foods, including beef and fresh fruits and vegetables, avoiding raw and unpasteurized dairy products and juices, combined with consumer education may provide the best opportunities for preventing and controlling *E. coli* O157 and non-O157 infections and HUS.

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Last updated 8/6/2015

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Acknowledging Jeff Higa for interpretation of outbreak data

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 4,186 cases of shigellosis with estimated illness onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 2.8 cases per 100,000.
- Shigellosis incidence rates remained stable during this period, ranging from 2.8 per 100,000 in 2009 to 2.9 per 100,000 in 2012.
- Average annual shigellosis incidence rates were highest among children 1–4 years of age (9.0 per 100,000) and 5–14 years of age (3.9 per 100,000), followed by adults 35–44 years of age (2.9 per 100,000).
- From 2009 through 2012, 13 outbreaks of shigellosis were reported: 3 foodborne outbreaks with unknown or suspected food source and 10 outbreaks of unknown source, 9 of which had day care/ preschool/elementary school settings.
- Of reported cases with known species (74.8 percent), *S. sonnei* (66.4 percent), and *S. flexneri* (31.5 percent) infections were most common.
- Public health measures such as early diagnosis and reporting of cases, education on hand hygiene, and targeted education for high-risk groups likely offer the best opportunities for reducing disease transmission.

Background

Shigella is a commonly reported enteric bacterial pathogen in the United States (US), estimated to cause nearly half a million illnesses, with more than 5,400 hospitalizations, and 38 deaths each year[1]. *Shigella* infection is restricted to humans and is predominantly transmitted from person to person through direct or indirect fecal-oral contact. Other sources of infection include ingestion of contaminated food and drinking or recreational water, and sexual contact (especially among men who have sex with men). *Shigella* species include *S. dysenteriae*, *S. flexneri*, *S. boydii* and *S. sonnei*. *Shigella sonnei* is predominant in industrialized countries whereas *S. flexneri* is predominant in developing countries[2,3].

Acute illness, usually gastroenteritis, occurs after an incubation period of 1 to 3 days. The severity of shigellosis varies by patient age and by infecting species and characterized by diarrhea, fever, nausea, cramps, and tenesmus. *S. dysenteriae* is associated with the most severe illnesses, whereas most people with *S. sonnei* infection have self-limited illness. Postinfectious arthritis is a rare complication of *Shigella* infection, especially with *S. flexneri* infection. Populations at increased risk of infection include young children, men who have sex with men, persons with human immunodeficiency virus (HIV) infection[4,5], and international travelers. Although most shigellosis appears to be sporadic cases, large outbreaks of *Shigella* have occurred, particularly in crowded settings where personal hygiene may be difficult, such as custodial institutions and child

care facilities. Pointsource outbreaks due to contaminated food or water have also occurred.

Recently, the US Centers for Disease Control and Prevention (CDC) has declared antibiotic-resistant *Shigella* a public health threat in the United States that requires a multi-pronged approach to reduce spread[6]. Increasing numbers of *Shigella* isolates have demonstrated resistance to antimicrobial agents, including ciprofloxacin. This has been associated with both international travel and domestic acquisition[6,7,8,9.]

We describe here the epidemiology of shigellosis case-patients in California with estimated illness onset from January 2009 through December 2012 reported to CDPH by December 4, 2014. Data for 2012 are provisional and may differ from data in future publications. The epidemiologic description of shigellosis for the 2001-2008 surveillance period can be found in the *Epidemiologic Summary of Shigellosis in California, 2001-2008*[10]. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes[11].

California reporting requirements and surveillance case definition

California Code of Regulations Title 17 requires health care providers to report suspected cases of shigellosis to their local health department within one working day of identification or immediately by telephone if an outbreak is suspected. Clinical and reference laboratories are also required to report either to the California Reportable Disease Information Exchange (CalREDIE) (via electronic laboratory reporting) or to the local health department when laboratory testing yields evidence suggestive of *Shigella*; reporting must occur within one working day after the health care provider has been notified.

Local health officers are required by California regulation to report to CDPH cases of shigellosis. CDPH officially counted cases that satisfied the CDC surveillance case definition, including both confirmed and probable case classifications. During the surveillance period, CDC defined a confirmed case as one with *Shigella* isolated from a clinical specimen, including asymptomatic and extra-intestinal infections. A probable case was one with clinically compatible illness and an established epidemiologic link to a laboratory-confirmed case, or a member of a risk group defined by public health authorities during an outbreak.

Epidemiology of shigellosis in California
CDPH received reports of 4,186 cases of shigellosis with estimated illness onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 2.8 cases per 100,000. Incidence rates remained stable during this period, ranging from 2.8 per 100,000 (1,055 case-patients) in 2009 to 2.9 per 100,000 (1,096 case-patients) in 2012 [Figure 1]. During the surveillance period, 8 (0.2 percent) case-patients were reported to have died.

Annual shigellosis incidence rates for the surveillance period were highest among children 1–4 years of age (9.0 per 100,000) and 5-14 years of age (3.9 per 100,000), followed by adults 35-44 years of age (2.9 per 100,000) [Figure 2]. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (17.5 percent). However, shigellosis cases with complete data reported Hispanic ethnicity more frequently than would be expected based on the overall demographic profile of California [Figure 3].

The average annual incidence rate for the surveillance period was 6.5 percent higher in Northern California (2.9 per 100,000) than in Southern California (2.7 per 100,000). County-specific incidence rates for the report period ranged from 0 to 14.9 county-specific shigellosis incidence rates, 2009–2012 per 100,000 residents [Figure 4]. The highest rates occurred in Imperial (year 2010: 28.1 per 100,000) and San Francisco (year 2012: 17.6 per 100,000) counties.

From 2009 through 2012, CDPH received reports of 13 outbreaks of shigellosis: 3 foodborne outbreaks with unknown or suspected food source and 10 outbreaks of unknown source, 9 of which had day care/ preschool/elementary school settings.

From 2009 through 2012, 3,132 (74.8 percent) cases had a *Shigella* isolate with the species identified and reported. Among these, *S. sonnei* (2,081; 66.4 percent), and *S. flexneri* (987; 31.5 percent) infections comprised of the majority of the reported casepatients. Median age of *S. sonnei* case-patients was 26 years and infections were slightly higher among males than females (male to female ratio: 1.3:1.0), whereas *S. flexneri* case-patients' median age was 33 and their male to female ratio was 2.2:1.0. This is consistent with national findings, where *S. sonnei* (71.7 percent), and *S. flexneri* (18.4 percent) were also the most common species identified and reported in the U.S.[2].

Figure 1. California shigellosis case counts and incidence rates by estimated illness onset, 2001–2012

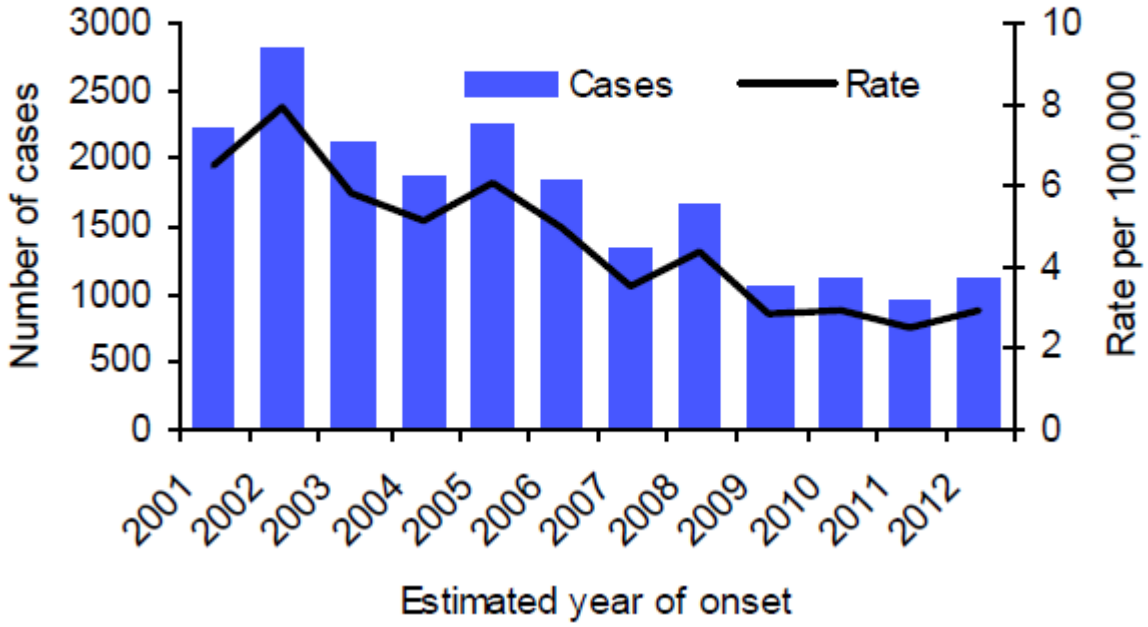


Figure 2. California shigellosis incidence rates by age group and time period, 2009–2012

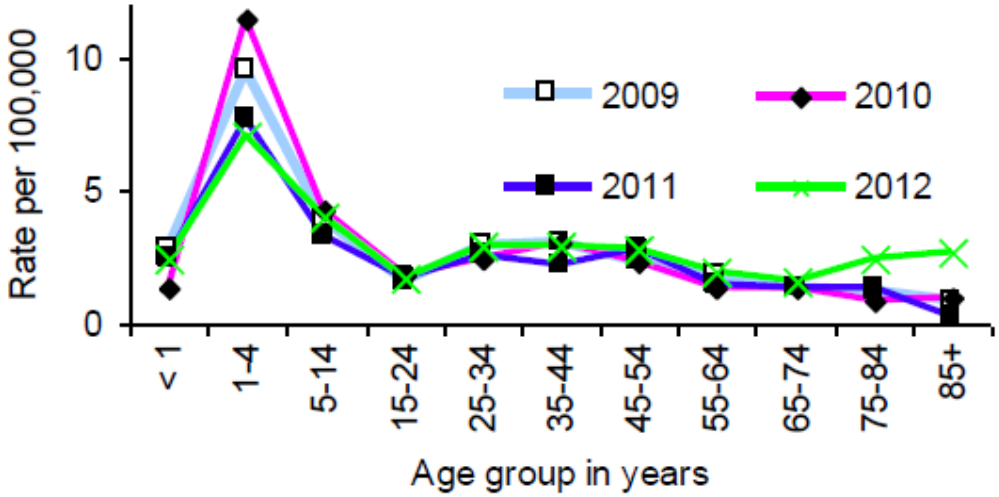
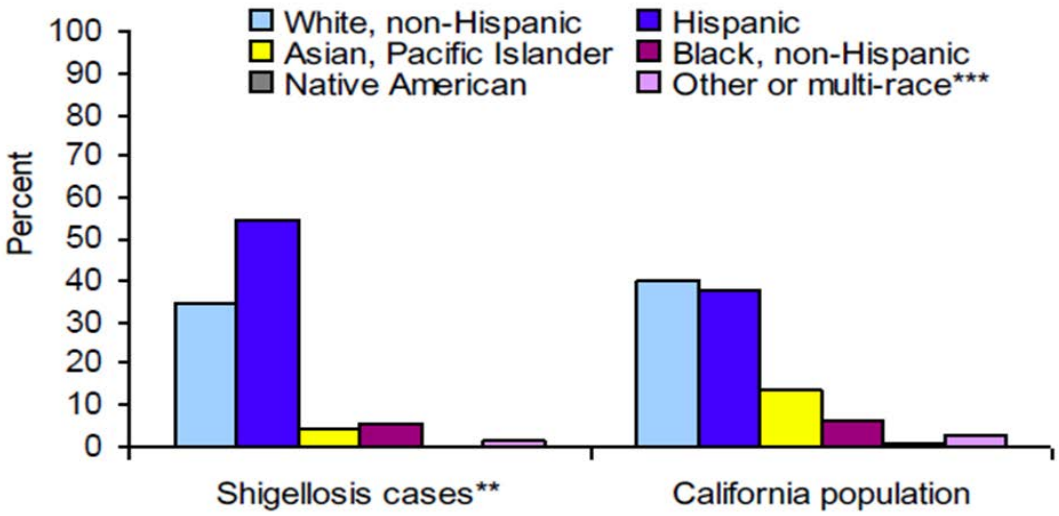


Figure 3. California shigellosis cases and population by race/ethnicity, 2009- 2012*



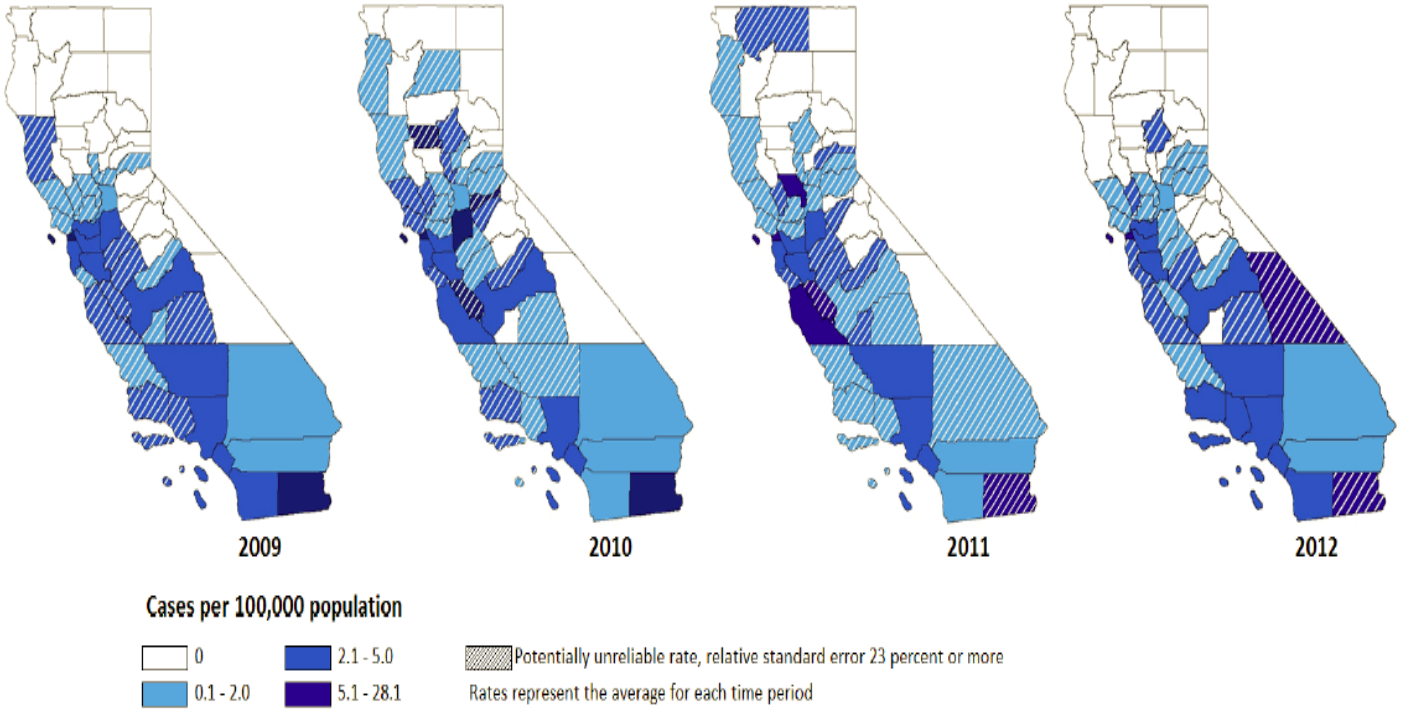
Notes for Figures 1-3

*2012 data are provisional

**Unknowns were excluded

***Includes cases who identified 'other' as their race and Californians ('population') who identified more than one race

Figure 4. California county-specific shigellosis incidence rates, 2009–2012



Comment

Incidence was relatively constant during the 2009– 2012 surveillance period. *S. sonnei* and *S. flexneri* were the most frequently identified species and were associated with different epidemiologic characteristics. Age group, race/ethnicity, and gender epidemiologic profiles of incident cases with estimated onset dates from 2009 through 2012 remained fairly consistent with those with estimated illness onset dates from 2001 through 2008, as previously described[10]. Public health measures such as early diagnosis and reporting of cases, education on hand hygiene (washing hands with soap and water for everyone, particularly in group settings such as childcare facilities), and targeted education for high-risk groups likely offer the best opportunities for reducing disease transmission.

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Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 537 cases of non-cholera vibriosis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.36 cases per 100,000 Californians.
- Non-cholera vibriosis incidence rates decreased from 2009 (0.37 per 100,000) to 2011 (0.28 per 100,000), followed by a 75-percent increase in 2012 (0.49 per 100,000).
- During the surveillance period, the highest incidence rate of non-cholera vibriosis was among adults 25 to 34 and 45 to 54 years of age (0.51 per 100,000). Average incidence rates were 1.8 times higher in men (0.46 per 100,000) compared to women (0.26 per 100,000).
- The highest reported incidence rates for the surveillance period were reported by the San Diego (0.73 per 100,000), San Francisco Bay Area (0.63 per 100,000), and Central Coast (0.56 per 100,000) regions.
- From 2009 through 2012, CDPH received reports of 4 foodborne outbreaks of non-cholera vibriosis involving a total of 26 California cases. Additionally, a 2012 multi-state outbreak accounted for two more cases in California. Consumption of raw or undercooked oysters was the implicated exposure in all five outbreaks.
- Ensuring that shellfish beds are routinely monitored for the presence of *Vibrio spp*, that shellfish are handled safely during and after harvest, and educating consumers about the risks of consuming raw or undercooked shellfish provide the best opportunities for reducing non-cholera vibriosis.

Background

Several non-cholera *Vibrio* species are important enteric bacterial pathogens, accounting for an estimated 80,000 illnesses, 500 hospitalizations, and 100 deaths each year in the United States (US).[1,2] *Vibrio* species are natural inhabitants of marine coastal and estuarine environments, and their populations increase during the warm summer months. In the US, the non-cholera *Vibrio* species of greatest concern are *V. parahaemolyticus* and *V. vulnificus*.

V. parahaemolyticus is the most commonly reported *Vibrio* infection, but *V. vulnificus* is associated with the greatest mortality. Consuming raw or undercooked shellfish is the most common cause of non-cholera vibriosis.[3-5] Exposing wounds to contaminated warm seawater or raw shellfish harvested from such waters can also cause skin or soft tissue *Vibrio* infection.[6]

V. parahaemolyticus infection causes acute gastroenteritis with fever that usually occurs after an incubation period of 24 hours. Symptoms usually last 1 to 7 days and are often self-limited. *V. vulnificus* can cause systemic disease that is frequently fatal, especially in persons with immunocompromising conditions, particularly those with chronic liver disease.[7] We describe here the epidemiology of confirmed and probable non-cholera vibriosis cases in California with estimated illness onset from 2009 through 2012 that were reported to CDPH by April 21, 2015. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes.[8] The epidemiologic description of non-cholera vibriosis for the 2001–2008 surveillance period can be found in the *Epidemiologic Summary of Non-Cholera Vibriosis in California, 2001—2008*.[9]

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report suspected cases of *Vibrio* infection to their local health department within one working day of identification or immediately by telephone if an outbreak is suspected. Clinical and reference laboratories are also required to report laboratory testing results suggestive of *Vibrio s*pecies to either the California Reportable Diseases Information Exchange (CalREDIE) (via electronic laboratory reporting) or the local health department*;* notification should occur within one working day after the health care provider has been notified of the laboratory testing result.

Local health officers are required by regulation to report to CDPH cases of non-cholera vibriosis. CDPH counted cases that satisfied the US Centers for Disease Control and Prevention (CDC)/Council of State and Territorial Epidemiologists' surveillance case definition of a confirmed or probable case. During the surveillance period, CDC defined a confirmed case of non-cholera vibriosis as one with isolation of *Vibrio spp.* other than toxigenic *Vibrio cholerae* O1 or O139 from a clinical specimen. A probable case had clinically-compatible illness and an epidemiologic link to a confirmed case, but no laboratory confirmation.

Epidemiology of non-cholera vibriosis in California

CDPH received reports of 535 confirmed and 2 probable cases of non-cholera vibriosis with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.36 cases per 100,000 Californians. Non-choleravibriosis incidence rates decreased from 2009 (0.37 per 100,000) to 2011 (0.28 per 100,000), followed by a 75-percent increase in 2012 (0.49 per 100,000) [Figure 1]. During the surveillance period majority of reported cases were *V. parahaemolyticus*. Four cases of *V. vulnificus* were reported between 2009-2012*.* The non-cholera vibriosis incidence rate during the surveillance period was highest among adults, 25 to 34 and 45 to 54 years of age (0.51 per 100,000) [Figure 2]. The incidence rates were 1.8 times higher in men (0.46 per 100,000) compared to women (0.26 per 100,000). Incidence rates by race/ethnicity were not calculated due to the substantial portion (20.5% percent) of missing data. However, non-cholera vibriosis cases with complete information reported White non-Hispanic race/ethnicity more frequently than would be expected based on the overall demographic profile of California [Figure 3].

Incidence rates for the surveillance period were 1.3 times higher in Northern California (0.42 per 100,000) than in Southern California (0.32 per 100,000). However, from 2009 to 2012, rates for Southern California increased by 37.5% percent (from 0.32 to 0.44 per 100,000) whereas rates for Northern California increased by 31.0% percent (from 0.42 to 0.55 per 100,000). In Northern California, the highest incidence rate (0.55 per 100,000) occurred in year 2012. The 3 geographic regions of California with the highest rates for the surveillance period were San Diego (0.73 per 100,000), the San Francisco Bay Area (0.63 per 100,000), and the Central Coast (0.56 per 100,000) [Figure 4].

From 2009 through 2012, CDPH received reports of 4 foodborne outbreaks of non-cholera vibriosis, involving 24 confirmed and 2 probable cases. Additionally, a 2012 multistate outbreak accounted for two more cases in California. Consumption of raw or undercooked oysters was the implicated exposure in all five outbreaks. The largest California outbreak occurred in 2009, involved 15 persons with *V. parahaemolyticus* infections, and was associated with consumption of imported raw oysters from oyster beds in Canada.

Figure 1. California non-cholera vibriosis case counts and incidence rates, 2001-2012*

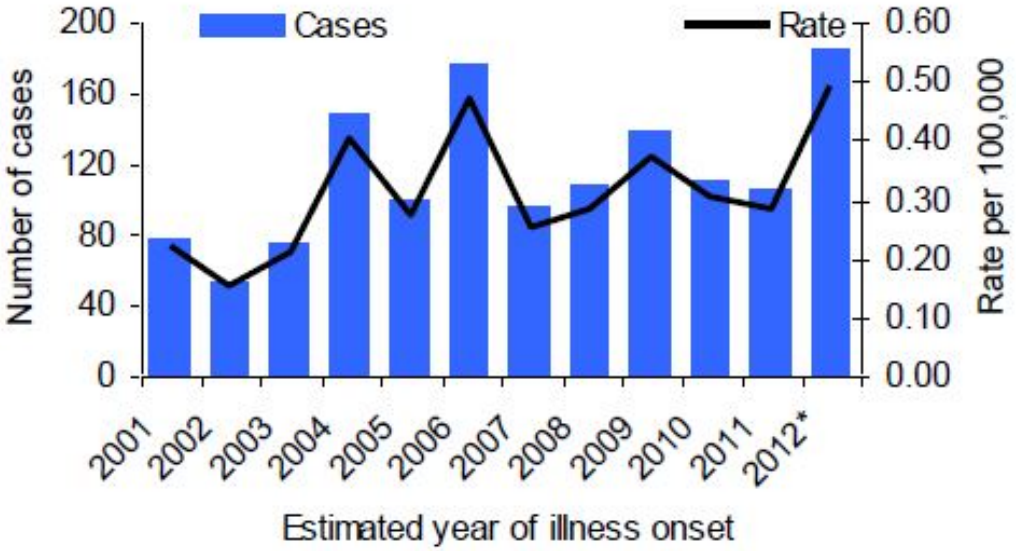


Figure 2. California non-cholera vibriosis by incidence rates by age-group and time period, 2009-2012*

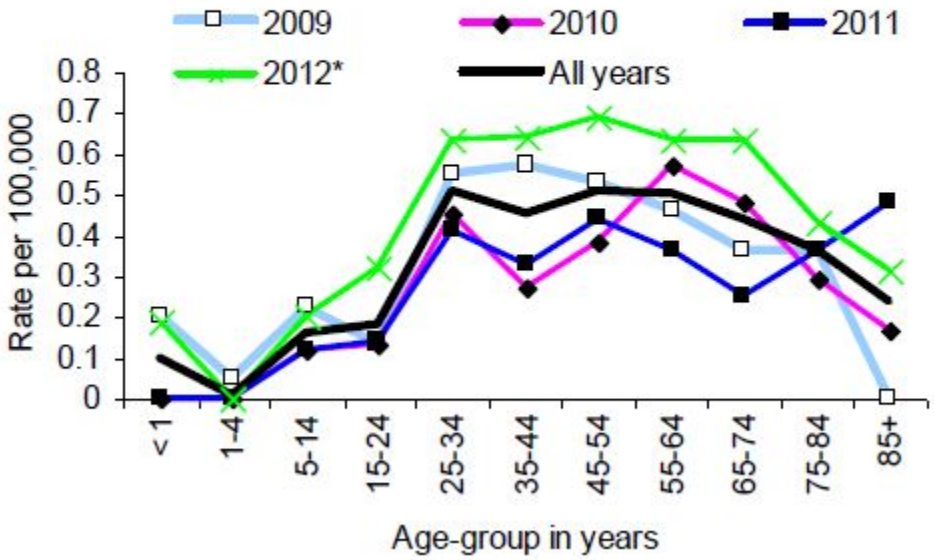
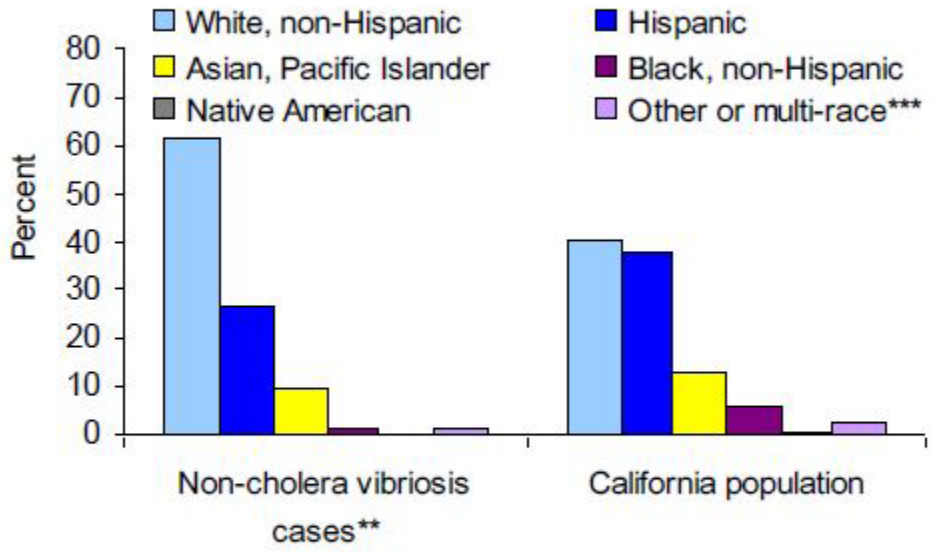


Figure 3. California non-cholera vibriosis cases and population by race/ethnicity, 2009 - 2012*



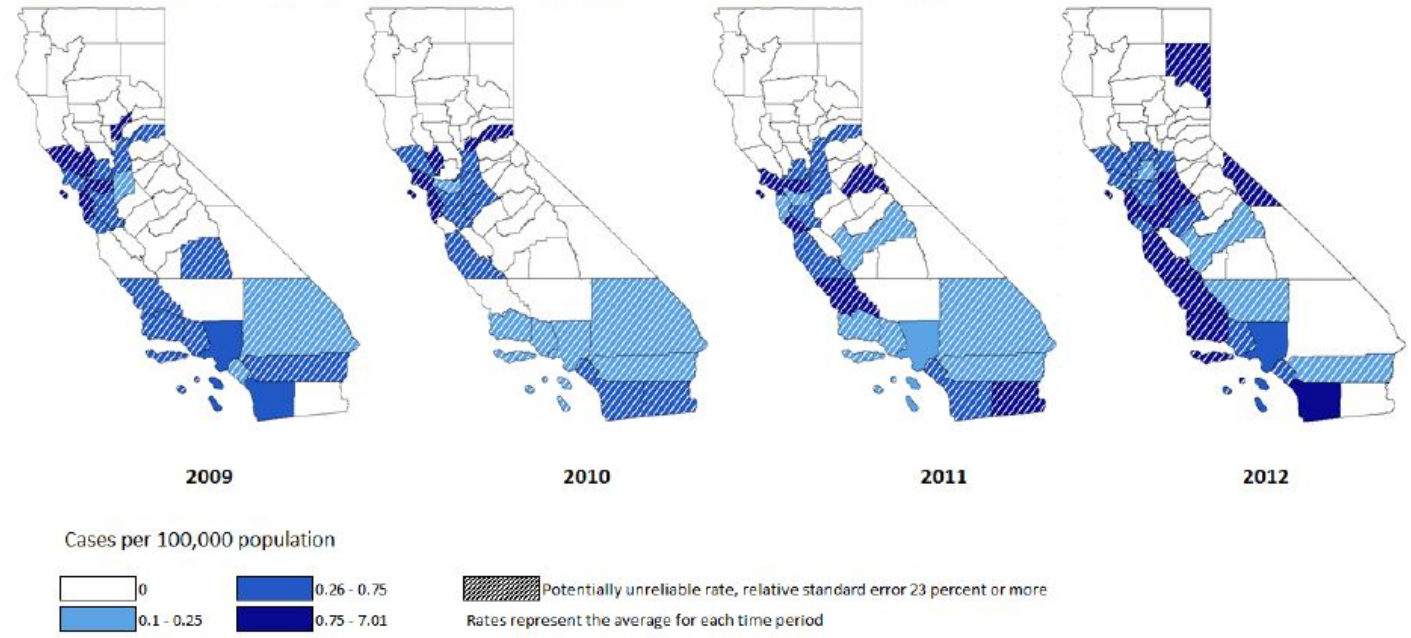
Notes for Figures 1-3

* 2012 data are provisional

** Unknowns were excluded

*** Includes cases who identified 'other' as their race and Californians ('population') who identified more than one race

Figure 4. California county-specific non-cholera vibriosis incidence rates, 2009-2012*



Comment

During the surveillance period, the highest incidence rate of reported non-cholera vibriosis among Californians was in 2012; most infection were due to *V. parahaemolyticus*. The geographic regions of California with the highest rates were San Diego, the San Francisco Bay Area, and the Central Coast, where *Vibrio* infections occurred in people who consume raw or undercooked shellfish.

Vibrio infections are often underdiagnosed, partly due to laboratories not routinely using media that are selective for vibriosis.[10] The recent increase in availability and use of culture-independent diagnostic tests (CIDT) is moving the clinical detection of bacterial infections, including vibriosis, away from culture based methods.[11] While CIDT allows for more rapid diagnosis, the lack of isolates that are currently needed to distinguish among strains and subtypes makes monitoring of trends and rapid detection of outbreaks difficult. Ensuring that a positive CIDT result is followed by culture confirmation is critical in the detection, investigation, and prevention of foodborne disease outbreaks.

Ensuring that shellfish beds are routinely monitored for the presence of enteric pathogens, that shellfish are handled safely during and after harvest, and educating consumers about the risks of consuming raw or undercooked shellfish and about potential exposure from open-wound contact with warm seawater provide the best opportunities for reducing non-cholera vibriosis.

Immunocompromised individuals, especially those with chronic liver disease, are at highest risk for severe *Vibrio* infection and should be targeted for education. Physicians should maintain a high index of suspicion in persons with gastroenteritis or sepsis and a history of raw shellfish consumption. Physicians suspecting vibriosis should also notify the laboratory of their suspicions so that the appropriate selective culture medium can be used to isolate the organism.

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Last updated: 5/19/2016

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 50 confirmed cases of wound botulism with estimated illness onset dates from 2009 through 2012. This corresponds to an annual incidence rate of 0.03 per 100,000 Californians.
- Wound botulism incidence rates remained relatively level in 2009 and 2010 and decreased by 50 percent from 2010 (16 case-patients; 0.04 per 100,000) to 2012 (7 case-patients; 0.02 per 100,000).
- During the surveillance period, 1 (2.0 percent) case-patient was reported to have died with wound botulism.
- The ratio of male to female cases was 4.0:1.0.
- Rapid diagnosis and treatment, including administration of botulinum antitoxin, may provide the best opportunities for minimizing the morbidity and mortality associated with wound botulism. Educating injecting drug users to seek medical care if typical symptoms develop may enable more timely administration of antitoxin.

Background

Clostridium botulinum toxin is a rare but potent neurotoxin. It is produced by *C. botulinum,* an anaerobic, spore-forming bacterium that is ubiquitous in the environment. Wound botulism is caused by *C. botulinum* colonization of a wound and in situ toxin production. Wound botulism occurred mainly in the setting of traumatic injury until the early 1990's when California began experiencing an epidemic of wound botulism among injecting drug users.[1,2] *C. botulinum* toxin is listed among the Centers for Disease Control and Prevention (CDC) category A bioterrorism agents.[3]

Wound botulism is a neuroparalytic illness. Initial neurologic symptoms may appear up to 2 weeks after the wound is infected. Illness can progress to a symmetric, descending flaccid paralysis that begins in the cranial nerves. Untreated, botulism can progress to respiratory paralysis and death. If administered early in the course of illness, botulinum antitoxin can stop the progression of, but cannot reverse paralysis. Antitoxin is available exclusively from public health authorities.

We describe here the epidemiology of confirmed wound botulism cases in California with estimated illness onset from 2009 through 2012. Cases reported as of September 10, 2014 are included. The epidemiologic description of wound botulism for the 2001-2008 surveillance period was previously published in the Epidemiologic Summary for Wound Botulism in California, 2001- 2008.[4] Data for 2012 are provisional and may differ from data in future publications. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes.[5]

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report suspected cases of botulism to their local health department immediately by telephone. Laboratories must immediately communicate by telephone with the CDPH Microbial Diseases Laboratory for instruction whenever a specimen for laboratory diagnosis of suspected botulism is received. Laboratories must report to the local health department when laboratory testing yields evidence suggestive of *C. botulinum;* notification must occur within one hour after the health care provider has been notified.

California regulations require local health departments to report to CDPH cases of wound botulism immediately by telephone. CDPH officially counted cases that satisfied the CDC surveillance case definition. CDC defined a confirmed case of wound botulism as clinically compatible illness and detection of botulinum toxin in serum, or isolation of *C. botulinum* from the wound in a patient who has no suspected exposure to contaminated food and who has a history of a fresh, contaminated wound during the 2 weeks before onset of symptoms, or a history of injection drug use within the 2 weeks before onset of symptoms.

Epidemiology of wound botulism in California

CDPH received reports of 50 cases of wound botulism with estimated illness onset dates from 2009 through 2012. This corresponds to an annual incidence rate of 0.03 per 100,000 Californians. Wound botulism incidence rates remained relatively level in 2009 and 2010, and decreased by 50 percent from 2010 (16 case- patients; 0.04 per 100,000) to 2012 (7 case-patients; 0.02 per 100,000) [Figure 1]. During the surveillance period, 1 (2.0 percent) case-patient was reported to have died with wound botulism.

Incidence rates of wound botulism were highest among persons 45 to 54 years of age [Figure 2]. There were no case-patients under 16 years of age or over 65.

The majority of confirmed incidents of wound botulism were among intravenous drug users (49 case-patients; 98%). The ratio of male to female cases was 4.0:1.0. Among wound botulism cases with complete information on race/ethnicity (94 percent), Hispanic ethnicity (66 percent) was reported much more frequently than would be expected based on the overall demographic profile of California [Figure 3].

During the surveillance period, 15 counties reported at least 1 case of wound botulism. These counties were distributed throughout the state so that all but 2 regions of the state (the Sierras and Far North) reported at least 1 case. The Inland Empire (0.04 per 100,000), San Diego (0.05 per 100,000), and Bay Area (0.05 per 100,000) regions reported the highest annual incidence rates.

Figure 1. California wound botulism case counts and incidence rates, 2001–2012*

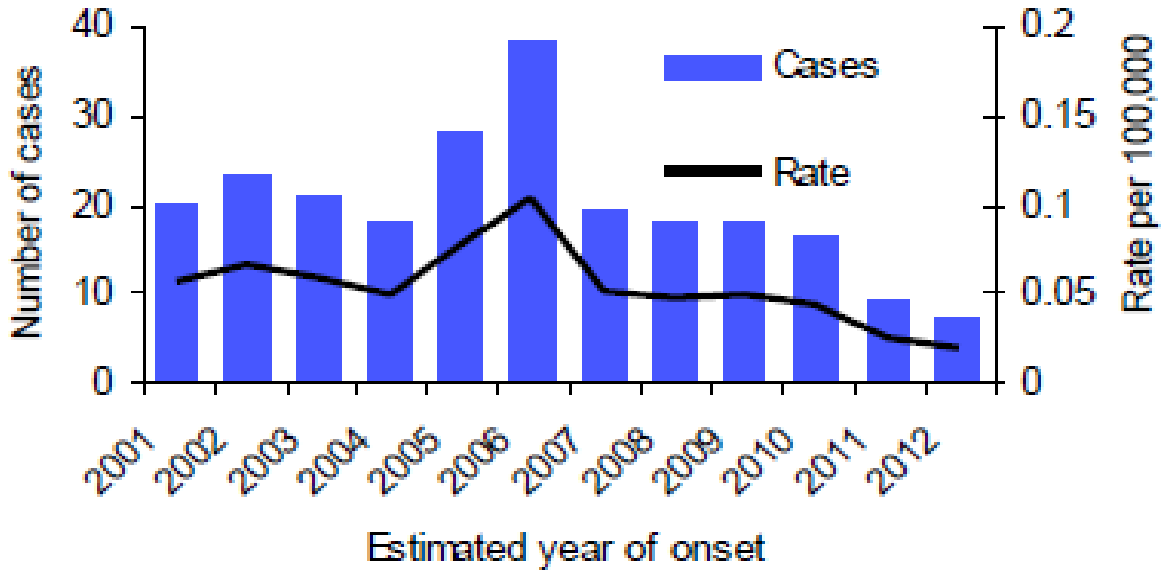


Figure 2. California wound botulism incidence rates by age, 2009-2012*

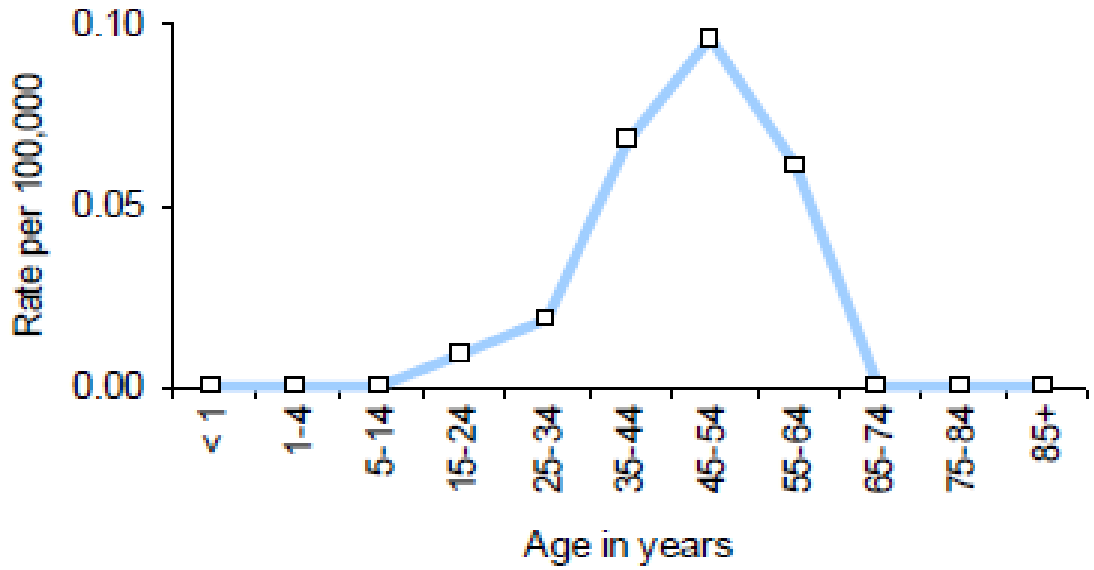
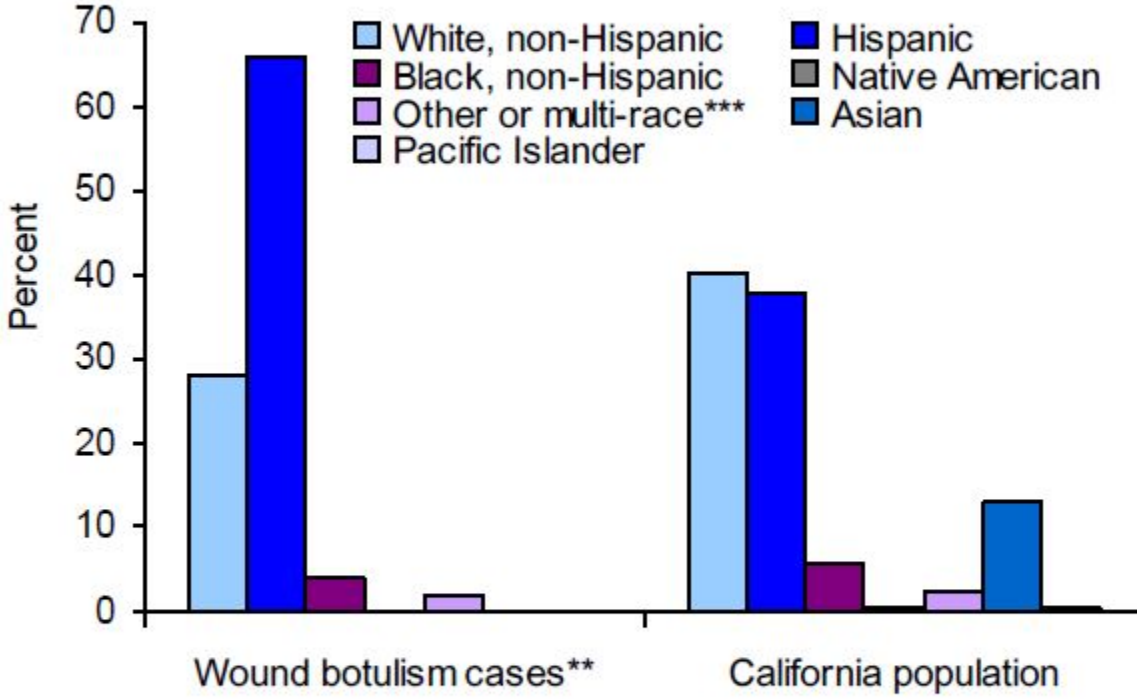


Figure 3. California wound botulism cases and population by race/ethnicity, 2009 - 2012*



Notes for Figures 1-3

*2012 data are provisional

**Unknowns were excluded

***Includes cases who identified 'other' as their race and Californians ('population') who identified more than one race

Comment

Although wound botulism remained a rare occurrence in California, each case represented a medical and public health emergency. Cases occurred almost exclusively among injecting drug users. A declining trend was observed in incidence of wound botulism during this surveillance period.

Rapid diagnosis and treatment, including administration of botulinum antitoxin, may provide the best opportunities for minimizing the morbidity and mortality associated with wound botulism. Additionally, educating injecting drug users to seek medical care if typical symptoms develop may enable more timely administration of antitoxin.

References and resources

1 Werner SB, Passaro D, McGee J, Schechter R, Vugia DJ. Wound botulism in California, 1951-98: Recent Epidemic in Heroin Injectors. Clin Infect Diseases 2000;31:1018-24.

2 Yuan J1, Inami G, Mohle-Boetani J, Vugia DJ: Recurrent wound botulism among injection drug users in California. Clin Infect Dis. 2011 Apr 1;52(7):862-6.

3 CDC botulism website
http://www.cdc.gov/nczved/divisions/dfbmd/diseases/botulism/professional.html; and
http://www.bt.cdc.gov/agent/Botulism/clinicians/

4 Epidemiologic Summary of Wound Botulism in California, 2001 - 2008.
http://www.cdph.ca.gov/programs/sss/Documents/Epi-Summaries-CA-2001-2008-083111.pdf#page=81

5 Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001 - 2008 and 2009 - 2012: Technical Notes
http://www.cdph.ca.gov/programs/sss/Documents/TechnicalNotes01-08and09-12.pdf

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