EPIDEMIOLOGIC SUMMARIES OF SELECTED GENERAL COMMUNICABLE DISEASES IN CALIFORNIA, 2001-2008

SURVEILLANCE AND STATISTICS SECTION
INFECTIOUS DISEASES BRANCH
DIVISION OF COMMUNICABLE DISEASE CONTROL
CENTER FOR INFECTIOUS DISEASES
CALIFORNIA DEPARTMENT OF PUBLIC HEALTH

Edmund G. Brown Jr.
Governor
State of California

Diana S. Dooley, Secretary
Health and Human Services Agency

Ronald W. Chapman, MD, MPH, Director
Department of Public Health

Table of Contents

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. In particular, we would like to acknowledge: Stanley Bissell, MS, Maria Teresa Bonafonte, MPH, PhD, Kate Brown, Kate Cummings, MPH, Daniel Gonzales, Melissa Moffett, Michelle Nguyen, Linda Sandoval, Shu Sebesta, Mark Starr, DVM, MPVM, Colleen Steward, Farzaneh Tabnak, MS, PhD, and Ying Yang, MS, of the Surveillance and Statistics Section; Jeff Higa, MPH, Akiko Kimura, MD, Bela Matyas, MD, MPH, Janet Mohle-Boetani MD, MPH, Jon Rosenberg, MD, Jennifer Schneider, MPH, and Charlotte Wheeler, MD, MPH, of the Disease Investigations Section; Claudia Erickson, MA, Sharon Ernst, James Glover, DVM, MPVM, MPH, Michele Jay-Russell, DVM, MPVM, and Ben Sun, DVM, MPVM, of the Veterinary Public Health Section; Curtis Fritz, DVM, PhD, MPVM, Anne Kjemtrup, DVM, PhD, MPVM, Vicki Kramer, PhD, and Jonathan Kwan, MS, of the Vector-Borne Disease Section; Meileen Acosta, MPH, Debra Gilliss, MD, MPH, Cynthia Jean Yen, MPH, Janice Kim, MD, MPH, Trish McLendon, MPH, and Hilary Rosen, MPH, of the Bioterrorism Epidemiology Section; Amy Karon, DVM, and Jean Yuan, MD, of the CDC Epidemic Intelligence Service; Janey Butner, and Duc Vugia, MD, MPH, of the Infectious Diseases Branch.We also thank Haydee Dabritz, PhD, Steve Arnon, MD, of the Infant Botulism Treatment and Prevention Program, Douglas Hatch, MD, MPH, James Watt, MD, MPH, of the Division of Communicable Disease Control, and Gilberto Chavez, MD, MPH, of the Center for Infectious Diseases.

Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001 - 2008: Technical Notes

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

We extracted data on communicable disease cases with an estimated onset date from 2001 through 2008 from California Confidential Morbidity Reports that were submitted to CDPH by May 8, 2009 and which met the surveillance case definitions (see below). Because of inherent delays in case reporting and depending on the length of follow-up clinical, laboratory and epidemiologic investigation, cases with eligible onset dates may be added or rescinded after the date of this report. Therefore, **data for 2008 contained in this report 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 2001 through 2008 from outbreak report forms submitted to CDPH by July 1, 2009. These reports were the source for the number of outbreak-associated cases for each disease.

Population data source

We used projections for state, county, and age-specific population totals that were published in: State of California, Department of Finance, *Race/ Ethnic Population with Age and Sex Detail, 2000–2050.* Sacramento, CA, July 2007.

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)[3]. 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 the surveillance period as 2001 through 2008.

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[4]. 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*); Sierras (*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 was missing (disease-specific range: 12 to 50 percent), we did not calculate 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 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 underreporting 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 cooperation of 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, 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.

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 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.

Acknowledgements

We thank California Communicable Disease Controllers and program staff for their continued excellence in communicable disease surveillance, investigation, and control.

We thank CDPH subject matter experts for their expertise and collaboration in producing the disease summaries. We would like to acknowledge the contributions to communicable disease surveillance and outbreak investigations made by the current and former members of the Infectious Diseases Branch. We thank Duc J Vugia, M.D., M.P.H. for his valuable comments throughout the development of the disease summaries.

References

[1]California Code of Regulations, Title 17, Sections 2500 and 2505
http://www.cdph.ca.gov/HealthInfo/Pages/ReportableDiseases.aspx

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

[3]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

[4]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

Last updated 08/30/2011

Prepared by Kate Cummings, MPH, and Farzaneh Tabnak, PhD, Infectious Diseases Branch

Epidemiologic Summary of Human Brucellosis in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 225 cases of brucellosis with estimated onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 0.08 per 100,000 Californians.
- Brucellosis incidence rates decreased by 54.5 percent from 2001 (0.11 per 100,000) to 2008 (0.05 per 100,000), although incidence rates rose from 2003 to 2007.
- Average brucellosis incidence rates during the surveillance period were higher among persons 55 to 64 years of age (0.10 per 100,000) and 65 to 74 years of age (0.15 per 100,000).
- Brucellosis cases reported Hispanic ethnicity (86.4 percent) more frequently than would be expected based on the overall proportion of Hispanics (35.3 percent) in California during the surveillance period.
- Avoiding consumption of unpasteurized milk and dairy products, limiting exposure to infected domestic animals, and education of higher risk groups (especially those in higher risk occupations) may provide the best opportunities for human brucellosis prevention and control.

Background

Brucella spp. are uncommon but important bacterial zoonotic pathogens in the United States (US), causing an estimated 100 to 200 cases per year. Consuming bovine or goat raw milk products, and contact through broken skin with infected animal tissues and fluids are leading sources of exposure in humans. Inhalation of bio-aerosols, notably in occupational settings such as laboratories, animal and veterinary settings, and accidental self-inoculation with animal vaccine strains can also result in infection. Person-to-person transmission is extremely rare. *Brucella* spp. are listed among the Centers for Disease Control and Prevention (CDC) category B bioterrorism (BT) agents.

Brucellosis has a variable and sometimes prolonged incubation period (5 days to 6 months) and often presents as a non-specific febrile syndrome (acute or insidious onset of fever, night sweats, undue fatigue, headache, and arthralgia). Brucellosis may occur in acute, chronic, and asymptomatic forms. Recurrent or 'undulant' fevers can occur if patients go untreated for long periods. Infections that last for more than 12 months can result in infections in bones, joints, liver, kidney, spleen, or heart valves.

We describe here the epidemiology of human brucellosis in California from 2001 through 2008. Data for 2008 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[1].

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report suspected cases of brucellosis 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 human brucellosis is received. Laboratories must also report to the local health department when laboratory testing yields evidence suggestive of *Brucella* spp; notification must occur within one hour after the health care provider has been notified.

California regulations also require local health officers to report to CDPH cases of brucellosis immediately by telephone. CDPH officially counted cases that satisfied the CDC surveillance case definition. CDC defined a confirmed case as one with an illness characterized by acute or insidious onset of fever, night sweats, undue fatigue, anorexia, weight loss, headache, and arthralgia and isolation of *Brucella* spp. from a clinical specimen or fourfold or greater rise in *Brucella* agglutination titer between serum specimens obtained at least 2 weeks apart and studied at the same laboratory, or demonstration by immunofluorescence of *Brucella* spp. in a clinical specimen. A probable case was one with clinically compatible illness and either an epidemiologic link to a confirmed case or supportive serology.

Epidemiology of brucellosis in California

CDPH received reports of 225 cases of brucellosis with estimated onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 0.08 per 100,000 Californians. Annual brucellosis

incidence rates decreased by 54.5 percent from 2001 (0.11 per 100,000) to 2008 (0.05 per 100,000), although rates rose from 2003 (0.05 per 100,000) to 2006 (0.09 per 100,000) [Figure 1]. During the surveillance period, 1 (0.4 percent) case was reported to have died with brucellosis.

Average annual brucellosis incidence rates during the surveillance period were higher among persons 55 to 64 years of age (0.10 per 100,000) and 65 to 74 years of age (0.15 per 100,000) [Figure 2]. The ratio of male to female cases was 1.0:1.0. During the surveillance period, brucellosis cases with complete information on race/ethnicity (88.0 percent of all cases) reported Hispanic ethnicity (86.4 percent) more frequently than would be expected based on the overall proportion of Hispanics (35.3 percent) in the California population.

Average annual incidence rates for brucellosis were similar in Northern California and Southern California. However, average incidence rates for the San Diego (0.14 per 100,00), San Joaquin Valley (0.11 per 100,000), and Central Coast (0.11 per 100,000) regions were higher than other regions in the state [Figure 3].

From 2001 through 2008, CDPH received reports of 2 outbreaks of foodborne brucellosis involving 7 cases. Both outbreaks were associated with consumption of imported unpasteurized cheese.

Comment

Brucellosis in California occurred more frequently among persons of Hispanic ethnicity. 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, limiting exposure to infected domestic animals, and education of higher risk groups (especially persons in higher risk occupations such as laboratory workers and veterinarians) may provide the best opportunities for human brucellosis prevention and control.

References and resources

[1]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf
CDPH brucellosis information website
http://www.cdph.ca.gov/HealthInfo/discond/Pages/Brucellosis.aspx

Last updated 10/22/2009
Prepared by Kate Cummings, MPH, Ben Sun, DVM, MPVM, and James Glover, MS, DVM, MPVM, MPH, Infectious Diseases Branch

Figure 1. California brucellosis case counts and incidence rates

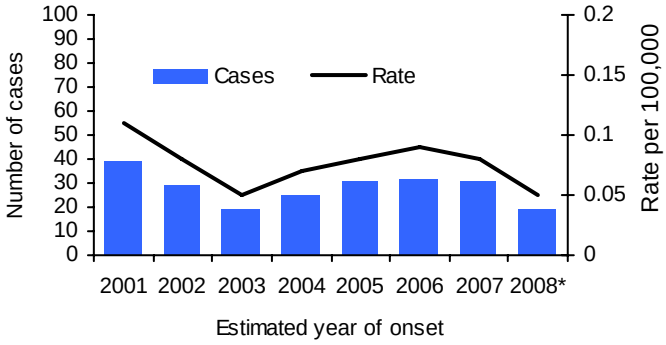


Figure 2. California brucellosis incidence rates by age 2001-2008*

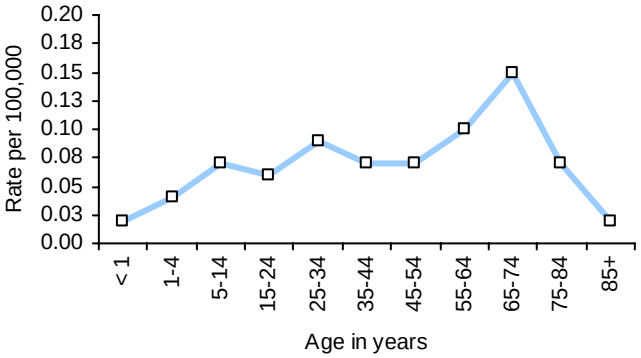
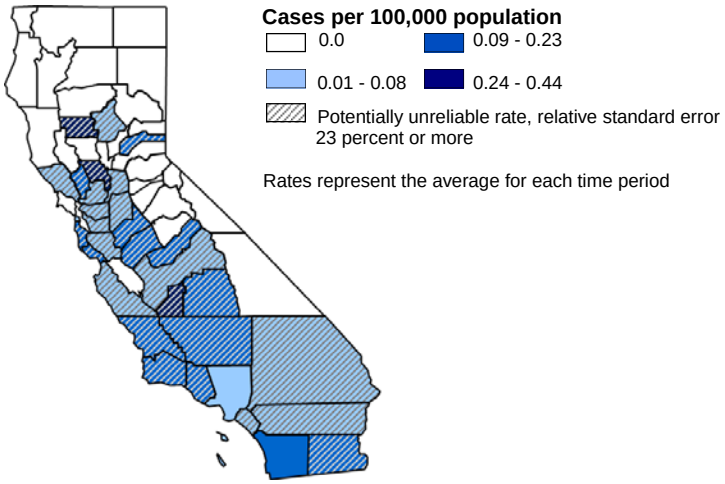


Figure 3. California county-specific brucellosis incidence rates 2001 - 2008*



Notes for Figures 1-3

*2008 data are provisional

Epidemiologic Summary of Campylobacteriosis in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 42,135 cases of campylobacteriosis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 14.4 cases per 100,000 Californians.
- Campylobacteriosis annual incidence rates decreased by 13.9 percent from 2001 (16.6 per 100,000) to 2008 (14.3 per 100,000).
- During the surveillance period, 76 (0.2 percent) cases were reported to have died with campylobacteriosis. Case fatality rates were 1.5 times higher in cases ≥ 65 years of age (0.3 percent) compared to cases < 65 years of age (0.2 percent).
- Average annual campylobacteriosis incidence rates during the surveillance period were higher among children under 1 year of age (33.0 per 100,000) and 1 to 4 years of age (34.1 per 100,000). Incidence rates among children under 1 year of age decreased by 37.5 percent from the combined years 2001 and 2002 (43.7 per 100,000) to the combined years 2007 and 2008 (27.3 per 100,000).
- From 2001 through 2008, CDPH received reports of 31 (16 confirmed, 15 suspected) outbreaks of foodborne campylobacteriosis in California involving 1,895 cases. One large outbreak associated with a dairy at a correctional facility involved 52 culture-confirmed cases and 1,592 clinically ill inmates.
- 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 2.4 million infections, 13,000 hospitalizations, and 100 deaths each year[1]. Handling and consuming food contaminated by infected animals, especially poultry, are the leading sources of *Campylobacter* infection. Consuming contaminated water or milk, and 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 not multiply in food products[2]. The national *Healthy People 2010* target objective for campylobacteriosis is no more than 12.3 new cases per 100,000 population.

Acute illness, usually gastroenteritis, occurs after an incubation period of 2 to 5 days, and usually lasts 1 week. Rarely, severe illness and death may occur, usually among the immunocompromised. Approximately one in 1,000 diagnosed *Campylobacter* infections can lead to Guillain-Barré syndrome. 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[3].

We describe here the epidemiology of campylobacteriosis in California from 2001 through 2008. Data for 2008 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

California Code of Regulations, Title 17, requires health care providers to report suspected cases of campylobacteriosis to their local health department within one working day of identification or immediately by telephone if an outbreak is suspected. Campylobacteriosis is not included in state regulations requiring notification by laboratories to local health officials.

Local health officers are required by regulation to report to CDPH cases of campylobacteriosis. CDPH officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC) surveillance case definition, including both confirmed and probable classifications. During the surveillance period, CDC defined a confirmed case as one with *Campylobacter*

isolated from a clinical specimen including asymptomatic and extraintestinal infections. 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 42,135 cases of campylobacteriosis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 14.4 cases per 100,000 Californians. Campylobacteriosis incidence rates decreased by 13.9 percent from 2001 (16.6 per 100,000) to 2008 (14.3 per 100,000). During the surveillance period, 76 (0.2 percent) cases were reported to have died with campylobacteriosis.

Average annual campylobacteriosis incidence rates during the surveillance period were higher among children under 1 year of age (33.0 per 100,000) and 1 to 4 years of age (34.1 per 100,000). Incidence rates among children under 1 year of age decreased by 37.5 percent from the combined years 2001 and 2002 (43.7 per 100,000) to the combined years 2007 and 2008 (27.3 per 100,000). In contrast, incidence rates increased by 34.7 percent among persons 75 years of age and older (from 12.1 to 16.3 per 100.000). 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 (40.7 percent). However, campylobacteriosis cases with complete data reported Hispanic ethnicity more frequently than would be expected based on the overall demographic profile of California [Figure 3].

Thirty-five (60.3 percent) of 58 counties reported average annual incidence rates for the surveillance period that were above the *Healthy People 2010* objective. Average annual incidence rates for the surveillance period were 2.0 times higher in Northern California (20.3 per 100,000) than Southern California (10.0 per 100,000). From 2001 to 2008, incidence rates for Southern California decreased by 14.7 percent (from 11.6 to 9.9 per 100,000) and rates for Northern California decreased by 14.8 percent (from 23.0 to 19.6 per 100,000). County-specific incidence rates for each two-year interval of the surveillance period ranged from 0.0 to 49.9 per 100,000 persons [Figure 4].

From 2001 through 2008, CDPH received reports of 31 (16 confirmed, 15 suspected) outbreaks of foodborne campylobacteriosis in California involving

Figure 1. California campylobacteriosis case counts and incidence rates

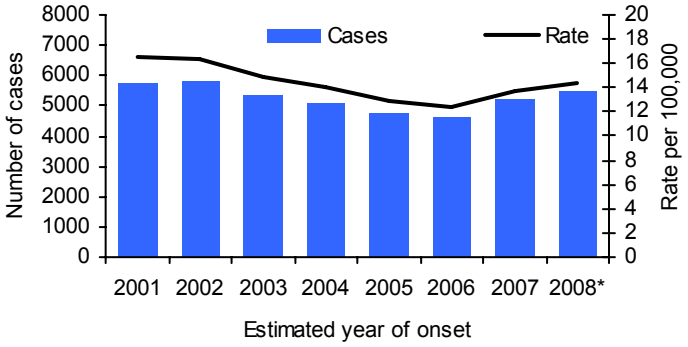


Figure 2. California campylobacteriosis incidence rates by age and time period

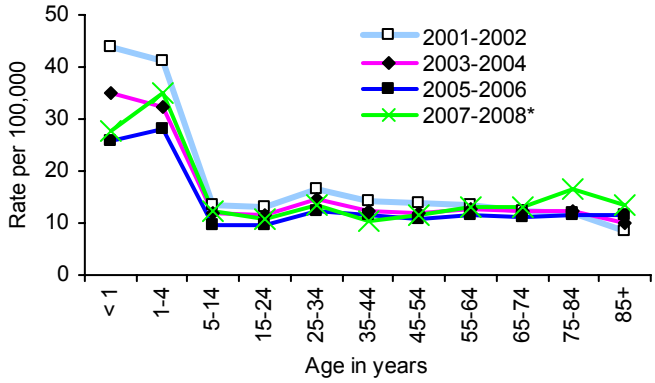
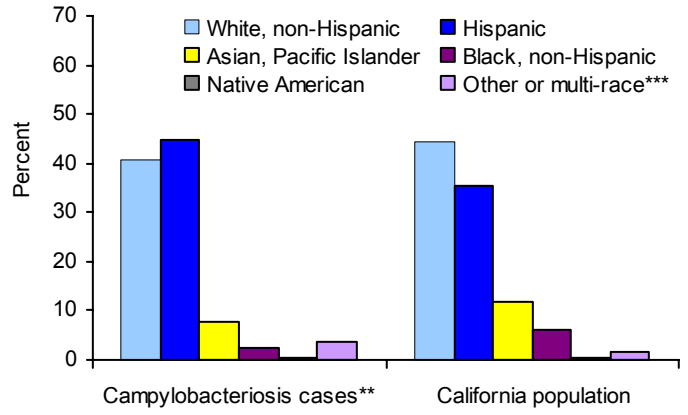


Figure 3. California campylobacteriosis cases and population by race/ethnicity 2001 - 2008*



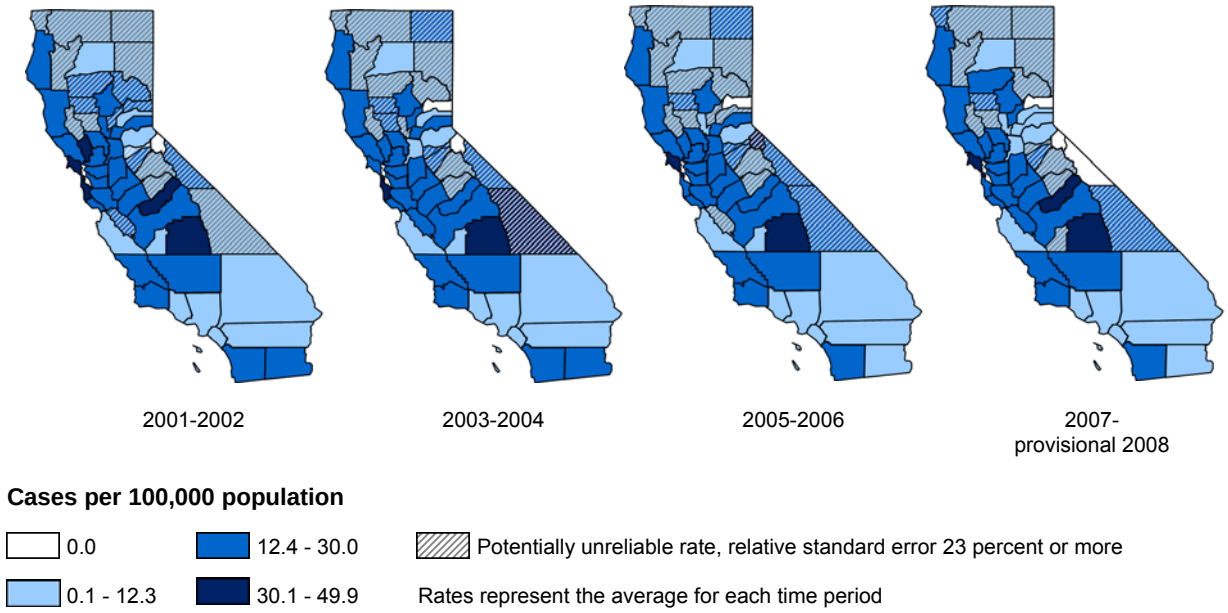
Notes for Figures 1-3

*2008 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 campylobacteriosis incidence rates



1,895 cases. Of 6 outbreaks with a confirmed vehicle, dairy products (3), poultry (2), and vegetables (2) were implicated. The largest confirmed outbreak occurred in 2006 and was associated with drinking pasteurized milk from a dairy at a correctional facility. It involved 52 culture-confirmed cases and an additional 1,592 clinical infections in inmates. The majority of these clinically ill inmates did not appear to be reported as individual cases in the passive reporting system (and may not have met the surveillance case classification criteria).

Comment

California has experienced a decrease in campylobacteriosis incidence from 2001 to 2006 although the *Healthy People 2010* target (12.3 per 100,000) was not achieved. Recent increases in case rates in 2007 and 2008, especially in the elderly, are difficult to interpret given they occurred over such a brief period of time. Continued monitoring of annual rates is needed.

Decreasing the contamination of poultry meat and dairy products, and consumer education may provide the best opportunities for preventing and controlling campylobacteriosis. The outbreak involving a dairy at a correctional facility underscores the opportunities for large scale outbreaks in these settings.

References and resources

[1]Mead PS, Slutsker L, Dietz V et al. Food-related illness and death in the United States. Emerg Infect Dis 1999;5:607-25. http://www.cdc.gov/ncidod/eid/Vol5no5/pdf/mead.pdf

[2] Gallay A, Bousquet V, Siret V. et al. Risk factors for acquiring sporadic *Campylobacter* infection in France: results from a national case-control study. J Infect Dis 2008;197:1477-1483.

[3]Nelson JM, Chiller TM, Powers JH, Angulo FJ. Fluoroquinolone-resistant *Campylobacter* species and the withdrawal of fluoroquinolones from use in poultry: A public health success story. Clin Infect Dis 2007;44:977-80.

[4]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

California Department of Public Health campylobacteriosis information web page http://www.cdph.ca.gov/HealthInfo/discond/Pages/Campylobacteriosis.aspx

Last updated 9/29/2009

Prepared by Kate Cummings, MPH, Amy Karon, DVM, and Akiko Kimura MD, Infectious Diseases Branch

Epidemiologic Summary of Cholera in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 5 cases of cholera with estimated onset dates from 2001 through 2008.
- Cholera stayed constant at 1 case per year during surveillance years 2003 to 2007.
- The median age among the cases was 45 years (range: 27 to 55 years).
- Three (60 percent) of the cholera cases were Asian, Pacific Islanders (60 percent). The ratio of male to female cases was 0.3:1.0.
- All of the cases had traveled outside of the U.S. and their illness onsets were within 2 days from their return. The majority of them (80 percent) reportedly did not have any education in cholera prevention before travel.
- When traveling to areas with epidemic cholera, following simple precautions such as drinking boiled or bottled water and eating thoroughly cooked foods may provide the best opportunities for prevention.

Background

Vibrio cholerae is the causative agent of cholera, a bacterial enteric disease. After an incubation period of a few hours to 5 days, cholera is characterized by sudden onset, profuse watery diarrhea and vomiting. Cholera has been rare in industrialized countries with modern sewage and water treatment systems, but it is still common in the Indian subcontinent and sub-Saharan Africa. The infection is often mild and with proper and timely rehydration the disease case fatality rate is less than 1 percent. Severe disease, however, occurs in about 1 in 20 infected persons and in untreated cases rapid dehydration, shock, and death can occur. The fatality rate may exceed 50 percent in severe dehydrated cases.

The cornerstone of cholera treatment is timely and adequate rehydration and replacement of electrolytes. The United States (US) travelers to areas with epidemic cholera may become exposed to cholera bacteria from ingesting contaminated foods or drinks. Contaminated seafood brought into the U.S. has previously been a source of cholera cases.

We describe here the epidemiology of cholera in California from 2001 through 2008. Data for 2008 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[1]. Because of the small numbers of reported cases, incidence rates were not calculated.

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report suspected cases of cholera to their local health department immediately by telephone. Cholera is not included in state regulations requiring notification by laboratories to local health officials. Local health officers are required by regulation to report to CDPH cases of cholera. CDPH officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC) surveillance case definition. During the surveillance period, CDC defined a confirmed case as one with illness characterized by diarrhea and/or vomiting and isolation of toxigenic (i.e., cholera toxin-producing) *Vibrio cholerae* O1 or O139 from stool or vomitus, or serologic evidence of recent infection as laboratory criteria.

Epidemiology of cholera in California

The California Department of Public Health (CDPH) received reports of 5 cases of cholera with estimated onset dates from 2001 through 2008. These cases were reported in years 2003 through 2007. (Figure 1)

The median age among cases was 45 years (range: 27 to 55 years). Three (60 percent) of the cholera cases were Asian, Pacific Islanders and 4 (80 percent) were females.

The predominant reported illness symptom among the cases was diarrhea. Abdominal cramps, fever, dehydration and muscle pain were among some of the other symptoms reported. *Vibrio cholerae* O1 was the species isolated from the stool of all of the cases.

Two of the cases were reported by San Francisco County and 1 each was reported by the Alameda, Contra Costa, and Orange Counties. All of the cases had traveled outside of the U.S. and their illness onsets were within 2 days from their return. The majority of them (80 percent) reportedly did not have any education in cholera prevention measures before travel.

Comment

From 2001 through 2008, 5 cases of cholera were reported in California. All of these cases had traveled outside of the U.S. where they probably were exposed to cholera bacteria. When traveling to areas with epidemic cholera following simple precautions such as drinking boiled or bottled water and eating thoroughly cooked foods may provide the best opportunities for prevention.

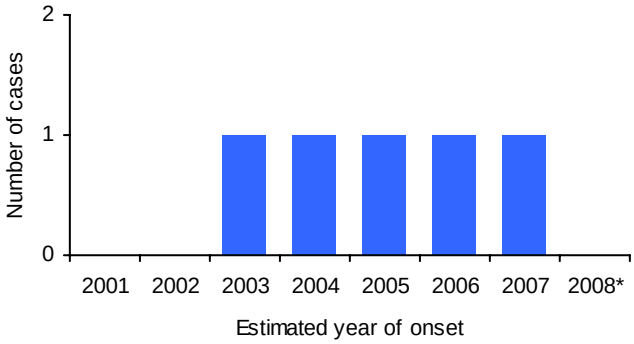
References and resources

[1]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

Last updated 11/02/2009

Prepared by Farzaneh Tabnak, MS, PhD, Kate Cummings, MPH, and Duc Vugia, MD, MPH, Infectious Diseases Branch

Figure 1. California cholera case counts



Notes for Figure 1
*2008 data are provisional

Epidemiologic Summary of Coccidioidomycosis in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 18,776 cases of coccidioidomycosis with estimated symptom onset dates from 2001 through 2008.
- Annual rates of coccidioidomycosis increased by 91.3 percent from 2001 (4.25 per 100,000) to 2006 (8.13 per 100,000) but decreased by 25.3 percent from 2006 to 2008 (6.07 per 100,000).
- During the surveillance period, 265 (1.4 percent) cases were reported to have died with coccidioidomycosis.
- During the surveillance period, the highest average annual incidence rate occurred among persons 45 to 54 years of age (8.81 per 100,000).
- Average annual incidence rates for the surveillance period were highest in Kern (155.0 per 100,000), Kings (70.6 per 100,000), Tulare (35.0 per 100,000), San Luis Obispo (31.7 per 100,000), Fresno (30.4 per 100,000) and Madera (14.9 per 100,000) Counties.
- During the surveillance period, CDPH received reports of increased cases in at least 1 federal and 2 state correctional institutions in the Central Valley and report of 1 point-source outbreak. In this 2007 outbreak, 10 of 12 civilian construction workers developed symptoms of coccidioidomycosis after excavating soil during an underground pipe installation on a military base in Monterey and San Luis Obispo Counties.
- 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.

Background

Coccidioidomycosis (also known as Valley Fever) results from directly inhaling spores of the dimorphic fungus *Coccidioides spp.* (*Coccidioides immitis* and *Coccidioides posadasii*) 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.

Coccidioidomycosis may occur in acute, chronic, and asymptomatic forms. Following an incubation period of 1 to 4 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 disseminated disease include African-Americans and Filipinos, those with immunocompromised conditions, and women in the third trimester of pregnancy. Disseminated or extrapulmonary coccidioidomycosis in an HIV-infected person is an AIDS defining condition.

We describe here the epidemiology of coccidioidomycosis in California from 2001 through 2008. Data for 2008 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[1]. Because coccidioidomycosis may occur as a chronic condition, 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, 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.

California regulations also require local health officers to report to CDPH cases of coccidioidomycosis. CDPH officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC) surveillance case definition. From 2001 through 2007, CDC defined 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 evidence of coccidioidal antibodies in serum or cerebrospinal fluid by detection of coccidioidal immunoglobulin (Ig) M, or a rising titer of coccidioidal IgG, or coccidioidal skin-test conversion from negative to positive after onset of clinical illness. Clinical illness included 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. In 2008, laboratory evidence concerning IgG was revised to detection of IgG alone, with or without evidence of a rising titer.

Epidemiology of coccidioidomycosis in California

CDPH received reports of 18,776 cases of coccidioidomycosis with estimated symptom onset dates from 2001 through 2008. Annual rates of coccidioidomycosis increased by 91.3 percent from 2001 (4.25 per 100,000) to 2006 (8.13 per 100,000) and decreased by 25.3 percent from 2006 to 2008 (6.07 per 100,000). During the surveillance period, 265 (1.4 percent) cases were reported to have died with coccidioidomycosis.

During the surveillance period, the highest average annual incidence rate occurred among persons 45 to 54 years of age (8.81 per 100,000). Incidence rates among persons 1 to 74 years of age increased from the combined years 2001 and 2002 to the combined years 2005 and 2006 and then decreased in the years 2007 and 2008 [Figure 2]. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (35.0 percent). However, cases with complete data reported Hispanic ethnicity and Black, non-Hispanic race/ethnicities more frequently than would be expected based on the overall demographic profile of California [Figure 3]. The ratio of male to female cases was 1.9:1.0.

Average annual incidence rates for the surveillance period were highest in Kern (155.0 per 100,000), Kings (70.6 per 100,000), Tulare (35.0 per 100,000), San Luis Obispo (31.7 per 100,000), Fresno (30.4 per 100,000) and Madera (14.9 per 100,000) Counties which are established *Coccidioides*-endemic areas. Although 76.4 percent of cases resided or were incarcerated in these 6 counties at the time of onset, only 6 California counties reported no cases during the entire surveillance period.

Figure 1. California coccidioidomycosis case counts and incidence rates

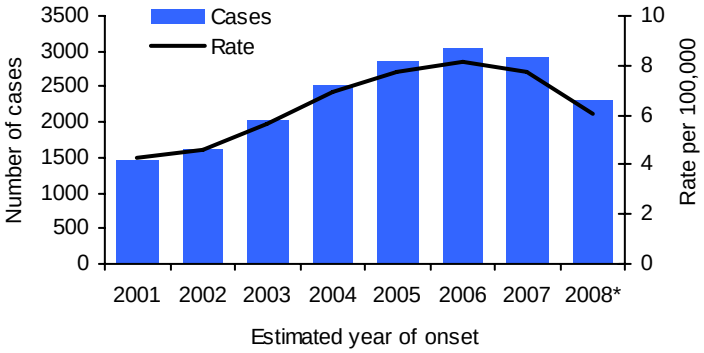


Figure 2. California coccidioidomycosis incidence rates by age and time period

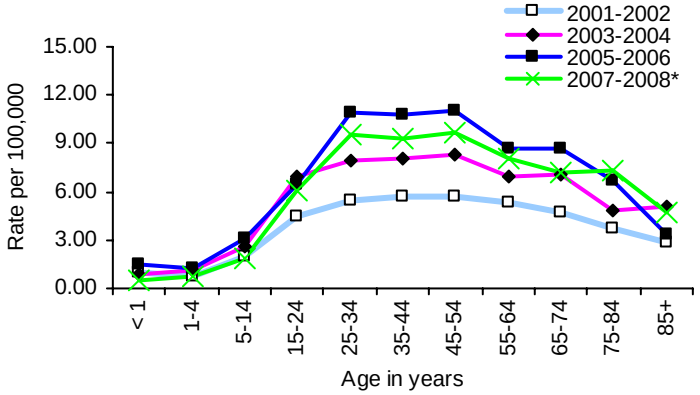
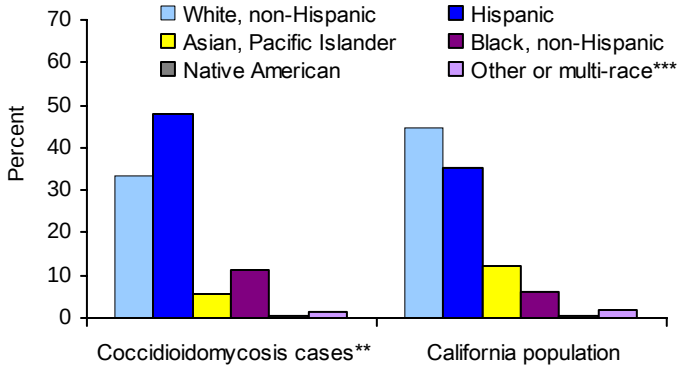


Figure 3. California coccidioidomycosis cases and population by race/ethnicity 2001 - 2008*



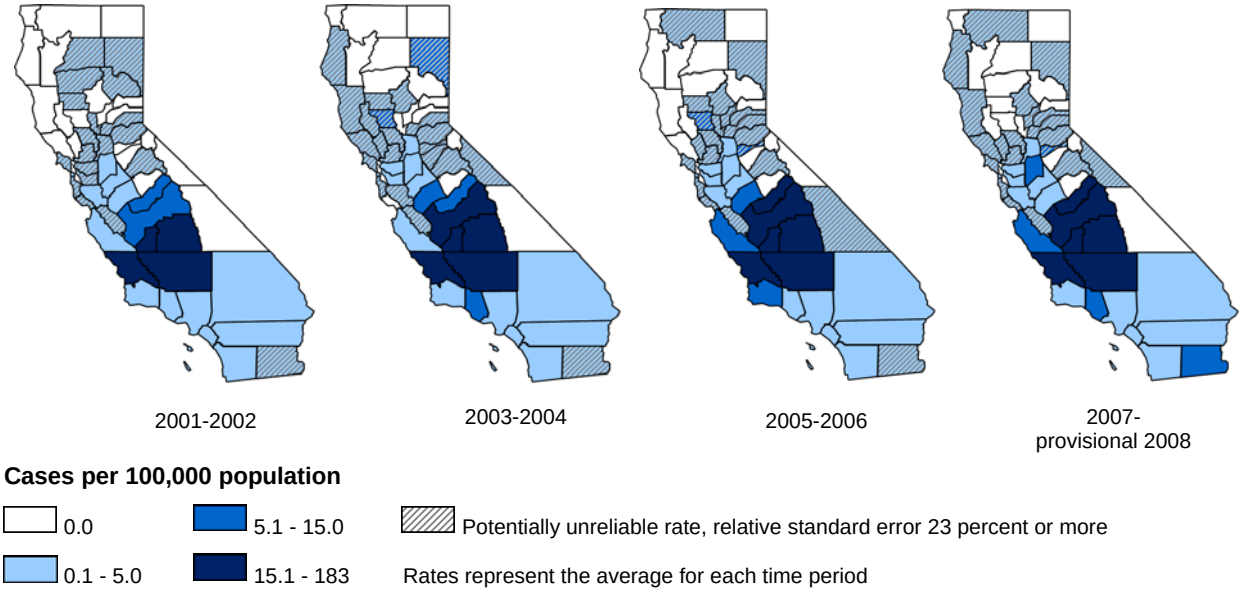
Notes for Figures 1-3

*2008 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 coccidioidomycosis incidence rates



During the surveillance period, CDPH received reports of increased cases in at least 1 federal and 2 state correctional institutions in the Central Valley. Taft Correctional Institution, a federal prison in Kern County, Avenal State Prison in Kings County, and Pleasant Valley State Prison in Fresno County reported increased numbers of cases among inmates in 2003, 2004, and 2005, respectively. CDPH also received report of 1 point-source outbreak of coccidioidomycosis in 2007. Ten of 12 civilian construction workers developed symptoms of coccidioidomycosis after excavating soil during an underground pipe installation on a military base in Monterey and San Luis Obispo Counties[2]. Eight cases had serologically-confirmed disease and 1 developed disseminated disease involving the skin.

Comment

From 2001 to 2006, coccidioidomycosis incidence rates nearly doubled in California – these increases have been described in more depth elsewhere[3]. Similar increases have been detected in other *Coccidioides* endemic areas such as Arizona. The causes of these increases are not well understood but climatic and environmental factors favorable to *Coccidiodes* proliferation and airborne release, and increases in non-immune populations in endemic areas may be contributing factors. Some of the recent increases in coccidioidomycosis in California may be attributable to increased cases among prison inmates[3,4].

Coccidioidomycosis is highly endemic in the San Joaquin Valley but remains an important public health and diagnostic consideration in all California counties. 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[3].

References and resources

[1]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf
[2]Cummings KC, McDowell A., Wheeler C, McNary J, Das R, Vugia DJ, Mohle-Boetani JC. Point-source outbreak of coccidioidomycosis in construction workers. Epidemiol Infect 2009 (in press).
[3]Centers for Disease Control and Prevention. Increase in coccidioidomycosis -- California, 2000-2007. MMWR 2009;58:105-9.
[4]Pappagianis D. Coccidioidomycosis in California state correctional institutions. Ann NY Acad Sci 2007;1111:1087 -9.

Prepared by Kate Cummings, MPH, Amy Karon, DVM, MPH, Charlotte Wheeler, MD, Duc J Vugia MD, MPH, Ying Yang, and Farzaneh Tabnak, MS, PhD, Infectious Diseases Branch

Last updated 11/05/2009

Epidemiologic Summary of Cryptosporidiosis in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 2,129 cases of cryptosporidiosis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 0.73 per 100,000 Californians.
- Annual cryptosporidiosis incidence rates increased by 30.9 percent from 2001 (0.68 per 100,000) to 2006 (0.89 per 100,000), and then decreased by 21.3 percent from 2006 to 2008 (0.70 per 100,000).
- During the surveillance period, 18 (0.9 percent) cases were reported to have died with cryptosporidiosis.
- Average annual cryptosporidiosis incidence rates during the surveillance period were higher among children 1 to 4 years of age (1.26 per 100,000), and among adults 35 to 44 years of age (1.29 per 100,000).
- The ratio of male to female cases was higher among adults 25 to 54 years of age (2.9:1.0) than among children 1 to 14 years of age (1.4:1.0). Cryptosporidiosis cases occurred more frequently in the months of August and September (30.2 percent of all cases).
- From 2001 through 2008, CDPH received reports of 7 cryptosporidiosis outbreaks involving 395 cases.
- 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

Cryptosporidium species is an important enteric parasitic pathogen in the United States (US), causing an estimated 300,000 infections per year[1]. Leading sources of *Cryptosporidium* infection include direct contact with an infected person or animal, ingestion of water or food contaminated by human or animal feces, and travel to highly-endemic areas. *Cryptosporidium* is resistant to current methods of water purification. In the US, it is the most frequently recognized cause of reported recreational water-associated outbreaks, particularly in disinfected venues, and is a recognized cause of drinking water-associated outbreaks[2]. Outbreaks in child care settings are also commonly reported.

Acute illness, usually gastroenteritis including watery diarrhea, occurs after an incubation period of 1 to 12 days. Symptoms in immunocompetent persons usually last 1 to 2 weeks. Infected persons who are immunodeficient, including those with HIV, may develop chronic, fulminant disease. Chronic intestinal cryptosporidiosis is an AIDS defining condition. Asymptomatic infections in people and animals are a frequent source of *Cryptosporidium* transmission.

We describe here the epidemiology of cryptosporidiosis in California from 2001 through 2008. Data for 2008 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[3].

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 must also notify the local health department when laboratory testing yields evidence suggestive of *Cryptosporidia*; notification 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 cryptosporidiosis. CDPH officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC) surveillance case definition. Although the CDC case definition was revised in 2009, from 2001 through 2008, CDC

defined a confirmed case as one with *Cryptosporidium* oocysts in stool by microscopic examination, or in intestinal fluid or small-bowel biopsy specimens; or oocyst or sporozoite antigens detected by immunodiagnostic methods or by PCR methods when routinely available; or demonstration of reproductive states in tissue preparations. Both symptomatic and asymptomatic laboratory confirmed infections were included among cases.

Epidemiology of cryptosporidiosis in California

CDPH received reports of 2,129 cases of cryptosporidiosis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 0.73 per 100,000 Californians. Annual cryptosporidiosis incidence rates increased by 30.9 percent from 2001 (0.68 per 100,000) to 2006 (0.89 per 100,000), although year-to-year changes were not uniform [Figure 1]. Incidence rates then decreased by 21.3 percent from 2006 to 2008 (0.70 per 100,000). During the surveillance period, 18 (0.9 percent) cases were reported to have died with cryptosporidiosis.

Average annual cryptosporidiosis incidence rates were higher in children 1 to 4 years of age (1.26 per 100,000), and in adults 35 to 44 years of age (1.29 per 100,000). Adults 35 to 44 years of age comprised 27.8 percent of cases. Incidence rates increased from the combined years of 2001 and 2002 to the combined years 2007 and 2008 for all age groups except children under 1 year of age and adults 35 to 44 years of age [Figure 2]. In this latter age group, incidence rates in men decreased by 54.1 percent (from 2.90 to 1.33 per 100,000) whereas the incidence rate in women increased by 128.6 percent (from 0.28 to 0.64 per 100,000).

The overall ratio of male to female cases was 2.0:1.0 although the ratio was 1.4:1.0 among children 1 to 14 years of age and 2.9:1.0 among adults 25 to 54 years of age. Cryptosporidiosis cases occurred more frequently in the months of August and September (30.2 percent of all cases). This seasonal pattern was evident among cases 1 to 14 years of age (53.7 percent occurred in August and September) and female cases 25 to 54 years of age (29.0 percent) but not in male cases 25 to 54 years of age (19.6 percent).

Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (38.9 percent). However, cryptosporidiosis cases with complete data reported White, non-Hispanic race/

Figure 1. California cryptosporidiosis case counts and incidence rates

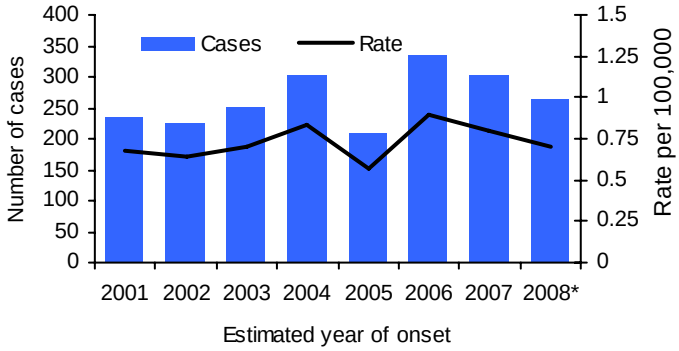


Figure 2. California cryptosporidiosis incidence rates by age and time period

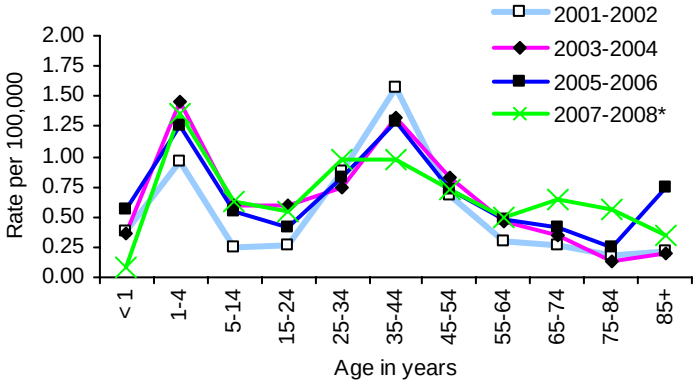
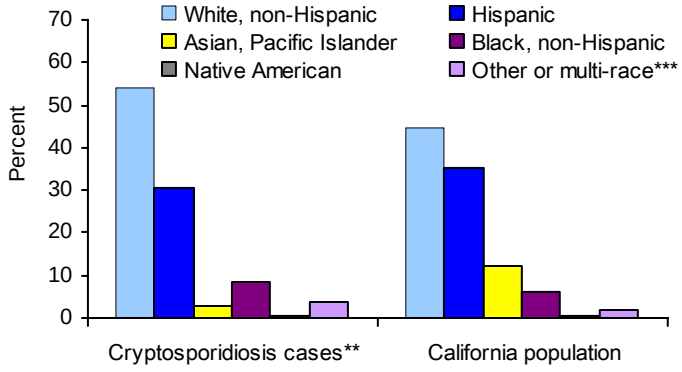


Figure 3. California cryptosporidiosis cases and population by race/ethnicity 2001 - 2008*



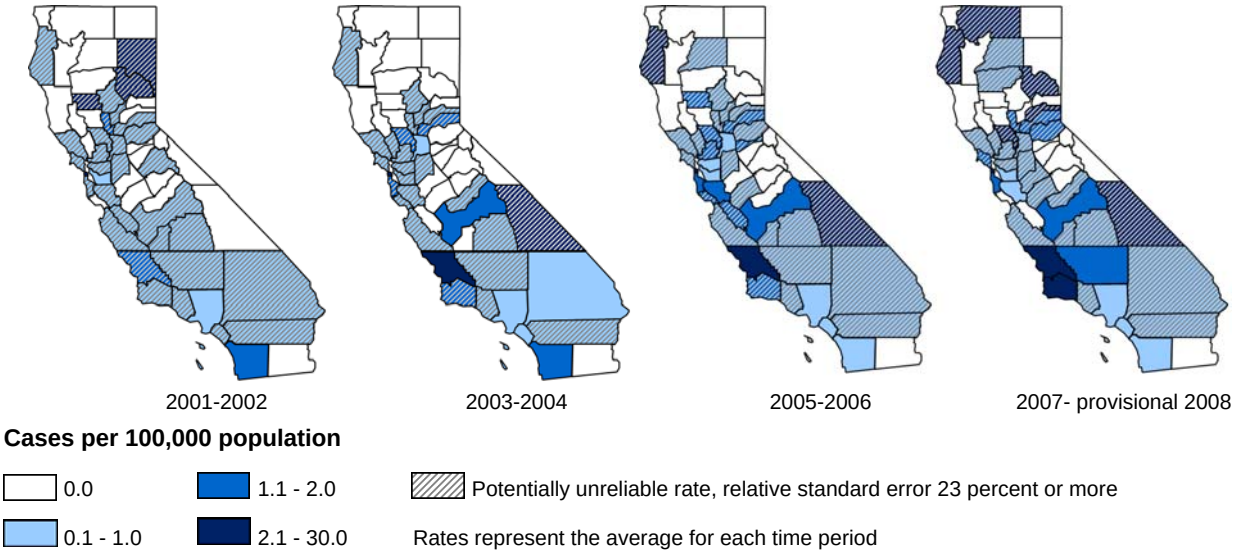
Notes for Figures 1-3

*2008 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 cryptosporidiosis incidence rates



ethnicity more frequently than would be expected based on the overall demographic profile of California [Figure 3].

Average incidence rates for the surveillance period were 1.6 times higher in Northern California (0.93 per 100,000) than in Southern California (0.57 per 100,000). From 2001 to 2008, cryptosporidiosis incidence rates increased by 64.1 percent (from 0.64 to 1.05 per 100,000) in Northern California but decreased by 39.4 percent (from 0.71 to 0.43 per 100,000) in Southern California.

From 2001 through 2008, CDPH received reports of 7 outbreaks of cryptosporidiosis involving 395 cases. One outbreak took place in a child care setting and the remaining 6 took place in recreational water settings. Four (66.7 percent) of 6 recreational water outbreaks had illness onsets in August or September. The largest outbreak occurred in 2004 at a water park and involved 59 culture-confirmed cases and an additional 277 clinically ill persons. Non-laboratory confirmed, clinically ill patients were not included in the official case count as they did not meet the CDC surveillance case definition.

Comment

California experienced an increase in cryptosporidiosis incidence rates from 2001 to 2006, followed by a modest decrease thereafter. Similar to national trends, cryptosporidiosis cases in children and women occurred more frequently during warmer months and may be associated with recreational water exposures[2]. Whether recent increases in children and selected adults reflect increases in disease diagnosis and reporting or disease activity is unclear and requires additional study.

Cryptosporidium presents special challenges to public health because of its extreme infectiousness combined with its resistance to chlorine disinfection. 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.

References and resources

[1]Mead PS, Slutsker L, Dietz V et al. Food-related illness and death in the United States. Emerg Infect Dis 1999;5:607-25. http://www.cdc.gov/ncidod/eid/Vol5no5/pdf/mead.pdf

[2]Centers for Disease Control and Prevention. Cryptosporidiosis surveillance - United States, 2003-2005. MMWR 2007;56(SS-7):1-10.

[3]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

Last updated 10/22/2009

Prepared by Kate Cummings, MPH, Charlotte Wheeler, MD, and Duc J Vugia MD, MPH, Infectious Diseases Branch

Epidemiologic Summary of Foodborne Botulism in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 26 cases of foodborne botulism with estimated onset dates from 2001 through 2008.
- During the surveillance period, 1 (3.9 percent) case was reported to have died with foodborne botulism.
- The ratio of male to female cases was 1.9:1.0.
- From 2001 through 2008, CDPH received reports of 4 outbreaks of foodborne botulism involving 11 cases. The largest outbreak occurred in 2004 and involved 4 male correctional inmates who consumed 'pruno', an alcoholic drink made illicitly in prison.
- Ensuring appropriate practices in food preparation and preservation and educating the public may provide the best opportunities for preventing and controlling 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. botulimum.* Despite the presence of bacteria and toxin in the stools of infected persons, person-to-person transmission has not been documented. *C. botulimum* toxin is listed among the Centers for Disease Control and Prevention (CDC) category A bioterrorism (BT) agents.

Botulism is a neuroparalytic illness. Neurologic symptoms generally begin 12 to 36 hours after ingestion of toxin. 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, 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 foodborne botulism in California from 2001 through 2008. Data for 2008 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[1]. 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 report to CDPH cases of foodborne botulism immediately 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 26 cases of foodborne botulism with estimated onset dates from 2001 through 2008. Annual foodborne botulism case counts remained level from 2001 to 2008 [Figure 1]. During the surveillance period, 1 (3.9 percent) case was reported to have died with foodborne botulism.

During the surveillance period, the number of foodborne botulism cases was highest among persons 35 to 44 years of age [Figure 2]. There were no cases in children under 15 years of age. The ratio of male to female cases was 1.9:1.0. Foodborne botulism cases with complete information on race/ethnicity (88.5 percent) reported Asian, Pacific Islander or Black, non-Hispanic race/ethnicities more frequently than would be expected based on the overall demographic profile of California [Figure 3]. Fourteen counties reported at least 1 case during the surveillance period. These counties were distributed throughout the state so that every region of the state reported at least 1 case. Riverside County reported the most cases (5); 4 were associated with an outbreak in a correctional facility described below.

From 2001 through 2008, CDPH received reports of 4 outbreaks of foodborne botulism involving 11 cases. The largest outbreak occurred in 2004 and involved 4 male correctional inmates who consumed 'pruno', an alcoholic drink made illicitly in prison[2]. Although not part of this outbreak, a male inmate in a second facility developed botulism after making and consuming pruno in 2005. All 5 inmates survived. The remaining outbreaks were associated with salmon (2 cases), home-canned carrots (2 cases), and home-fermented tofu (2 cases).

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. The outbreak identified in a correctional facility highlights a novel vehicle that can potentially place large numbers of persons at risk for botulism. Ensuring appropriate practices in food preparation and preservation and educating the public may provide the best opportunities for preventing and controlling foodborne botulism.

References and resources

[1]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

[2]Vugia DJ, Mase SR, Cole B, Stile J, Rosenberg J, Velasquez L, Radner A, Inami G. Botulism from drinking pruno. Emerg Infect Dis. 2009;15:69-71.

Last updated 10/22/2009

Prepared by Kate Cummings, MPH, Debra Gilliss MD, MPH, and Duc Vugia MD, MPH, Infectious Diseases Branch

Figure 1. California foodborne botulism case counts

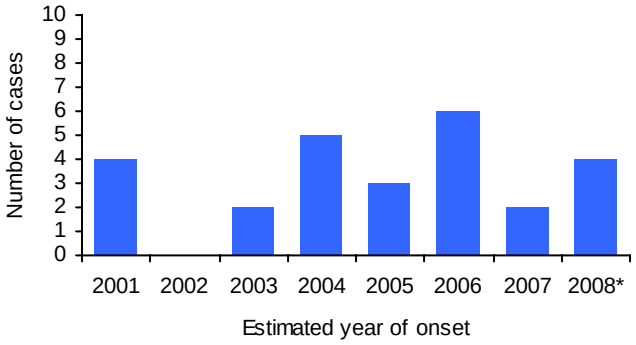


Figure 2. California foodborne botulism cases by age, 2001-2008*

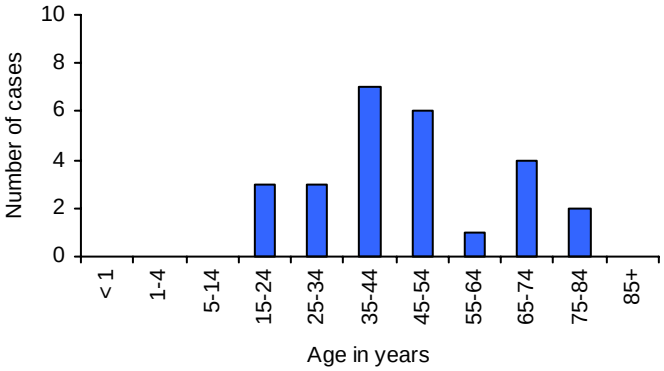
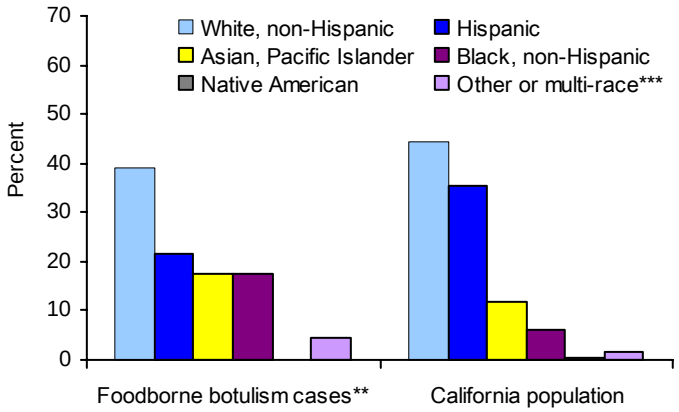


Figure 3. California foodborne botulism cases and population by race/ethnicity, 2001 - 2008*



Notes for Figures 1-3

*2008 data are provisional

**Unknowns were excluded

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

Epidemiologic Summary of Foodborne Disease Outbreaks in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 1,375 foodborne disease outbreaks (FBDOs) involving 24,280 cases with symptom onsets from 2001 through 2008. Of these 1,375 outbreaks, 26 involved exposures and cases in more than 1 California county and an additional 13 involved exposures and cases in more than 1 state.

- From 2001 through 2007, the annual numbers of reported outbreaks remained relatively level (average 180 outbreaks per year, range: 151 to 210 outbreaks) but decreased by 25.2 percent from 2007 (155) to 2008 (116).

- The etiologic agent responsible for causing the outbreak was confirmed in 392 (28.5 percent), suspected in 832 (60.5 percent), and undetermined in 151 (11.0 percent) outbreaks.

- From 2001 through 2008, CDPH received reports of 392 confirmed outbreaks involving 10,999 cases (45.3 percent of all reported cases). Etiologic agents included bacteria (53.6 percent), viruses (37.2 percent), chemical agents (7.4 percent), and parasites (1.8 percent).

- Among confirmed FBDOs, norovirus caused the most outbreaks (141) and involved the most cases (4,372).

- Reducing the occurrence of FBDOs requires coordination between public health and agricultural and food industries. These efforts, along with additional research and consumer education, may offer the best opportunities for controlling and preventing FBDOs.

Background

Foodborne diseases incur significant morbidity and mortality in the United States (US), causing an estimated 76 million illnesses, 325,000 hospitalizations, and 5,000 deaths each year[1]. Foodborne disease outbreaks (FBDO) contribute to this burden and are important sentinel public health events. Etiologic agents that cause FBDOs include bacteria (including bacterial toxins), viruses, chemicals (toxins and metals), and parasites. The clinical syndromes associated with outbreaks vary by etiologic agent but can range from mild to life threatening illnesses. There are 2 national objectives to reduce outbreaks of infections caused by key foodborne bacteria. These *Healthy People 2010* target objectives are to reduce the numbers of annual *Escherichia coli* O157:H7 and *Salmonella* serotype Enteritidis outbreaks in the US to, respectively, 11 and 22.

We describe here the epidemiology of FBDOs in California from 2001 through 2008. Data for 2008 are provisional and may differ from results in future publications. For a complete discussion of the definitions, methods and limitations associated with this report, please refer to Technical Notes[2].

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report suspected FBDOs to their local health department immediately by telephone. A FBDO is defined by California regulation as an incident in which two or more persons experience similar illness after ingestion of a common food, and epidemiologic analysis implicates the food as the source of the illness. Additionally, one case of botulism or chemical poisoning constitutes an outbreak if laboratory studies identify the causative agent in food. Two or more suspected cases of foodborne disease from separate households suspected to have the same source of illness (in the absence of epidemiologic analysis) are considered suspected FBDOs.

California regulations also require local health officers to report FBDOs to CDPH. CDPH officially counted FDBOs that satisfied Centers for Disease Control and Prevention (CDC) surveillance case definitions[3] with some modifications. While outbreak definitions are agent-specific, a confirmed etiology generally required laboratory evidence of a specific etiologic agent in two or more cases[3]. CDPH also

classified an etiology as *suspected* if, in the absence of laboratory-confirmation, clinical and epidemiologic characteristics and the incubation period suggested an etiologic category. The etiology was unknown or undetermined when the criteria for an outbreak were met (≥2 cases with similar illness after ingesting a common food) but the clinical features of the illness did not suggest a specific etiologic category. CDPH counted aggregate estimates of total persons ill reported on the standardized CDC Electronic Foodborne Outbreak Reporting System form. Some cases included in this report may not have met CDC surveillance case definitions for individual case reporting[2].

We considered an outbreak to be multicounty if exposures and cases involved more than 1 California county and multistate if exposure and cases involved more than 1 state.

Epidemiology of FBDOs in California

CDPH received reports of 1,375 FBDOs involving 24,280 cases with symptom onsets from 2001 through 2008. The annual numbers of reported outbreaks were: 178 in 2001, 210 in 2002, 188 in 2003, 177 in 2004, 151 in 2005, 200 in 2006, 155 in 2007, and 116 in 2008 [Figure 1]. The number of reported outbreaks decreased by 25.2 percent from 2007 (155) to 2008 (116) although this may, in part, reflect incomplete reporting for 2008. A total of 26 (1.9 percent) outbreaks involving 2,474 (10.2 percent) cases were multicounty and an additional 13 (0.9 percent) involving 220 (0.9 percent) cases were multistate.

FBDOs by etiologic agent and confirmation status

Of reported etiologic agents, 605 (44.0 percent) were bacterial, 535 (38.9 percent) were viral, 76 (5.5 percent) were chemical, 8 (0.6 percent) were parasitic, and 151 (11.0 percent) were unknown or undetermined. The etiologic agent was confirmed in 392 (28.5 percent), suspected in 832 (60.5 percent), and undetermined in 151 (11.0 percent) outbreaks [Figure 2]. Etiologic agents were confirmed in 210 (34.7 percent) reported bacterial outbreaks, 146 (27.3 percent) reported viral outbreaks, 29 (38.2 percent) reported chemical outbreaks, and 7 (87.5 percent) of reported parasitic outbreaks. The etiologic agent was confirmed in 224 (40.6 percent) of 552 outbreaks involving more than 10 cases and 167 (20.3 percent) of 821 outbreaks involving 10 or fewer cases.

Figure 1. Reported outbreaks by year and confirmation status

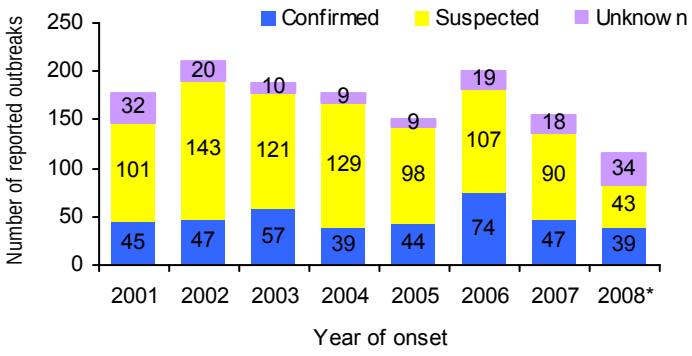


Figure 2. Reported outbreaks by etiology and level of confirmation 2001 - 2008*, excluding 151 outbreaks of unknown etiology

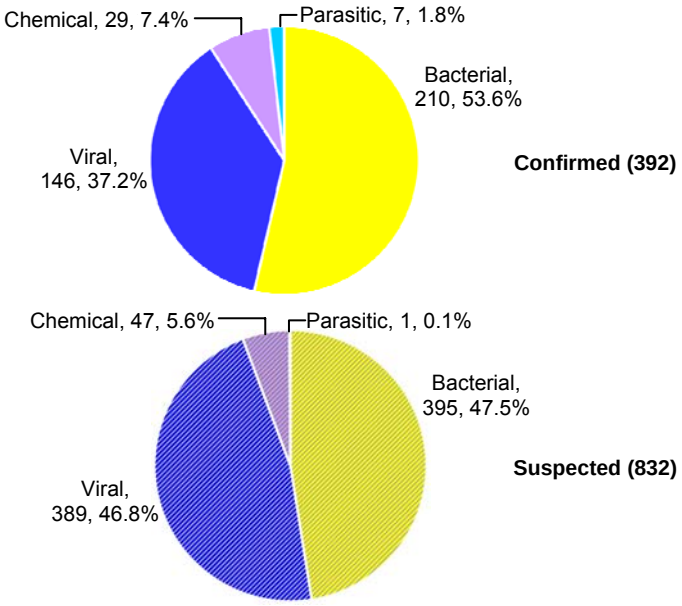
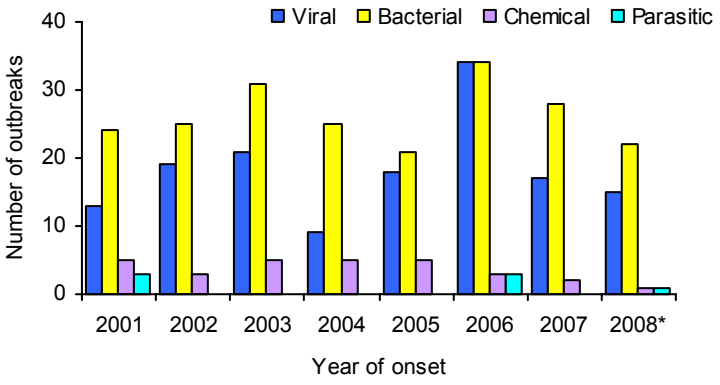


Figure 3. Confirmed outbreaks by etiologic agent and year



*2008 data are provisional

Table 1. Confirmed foodborne disease outbreaks and cases reported to CDPH by etiologic agent, 2001-2008*

	Outbreaks		Outbreak cases		
	Number	Percent	Total	Percent	Median per outbreak
All agents	**392**	**100.0**	**10,999**	**100.0**	**12**
Bacterial pathogens	**210**	**53.6**	**6,155**	**56.0**	**10.0**
Botulism, foodborne	4	1.0	11	0.1	2.0
Brucella	2	0.5	7	0.1	3.5
Campylobacter spp.	16	4.1	1,809	16.4	5.0
Clostridium perfringens	7	1.8	151	1.4	9.0
Shiga toxin-producing Escherichia coli	22	5.6	437	4.0	8.0
Listeria monocytogenes	1	0.3	28	0.3	-
Multiple agents	1	0.3	7	0.1	-
Salmonella	125	31.9	3,106	28.2	12.0
Shigella	19	4.8	417	3.8	9.0
Staphylococcus aureus	6	1.5	112	1.0	16.0
Vibrio spp (non-cholerae)	6	1.5	56	0.5	6.5
Yersina Enterocolitica	1	0.3	14	0.1	-
Chemical agents	**29**	**7.4**	**186**	**1.7**	**4.0**
Ciguatoxin	1	0.3	4	< 0.1	-
Gemylotoxin	1	0.3	10	0.1	-
Mushroom toxin	3	0.8	13	0.1	6.0
Scombrotoxin	24	6.1	159	1.4	3.0
Parasitic pathogens	**7**	**1.8**	**173**	**1.6**	**18.0**
Cyclospora cayetanensis	1	0.3	59	0.5	-
Giardia intestinalis	2	0.5	80	0.7	40.0
Paragonimus	1	0.3	18	0.2	-
Trichinella spiralis	3	0.8	16	0.1	7.0
Viral pathogens	**146**	**37.2**	**4,485**	**40.8**	**22.0**
Hepatitis A	4	1.0	51	0.5	12.0
Multiple agents	1	0.3	62	0.6	-
Norovirus	141	36.0	4,372	39.7	22.0

*2008 data are provisional
Numbers in bold are grand and subtotals

FBDOs with confirmed etiologies

Confirmed FBDOs (392) involved 10,999 cases (45.3 percent of all reported cases) [Table 1]. Etiologic agents included bacteria (53.6 percent), viruses (37.2 percent), chemical agents (7.4 percent), and parasites (1.8 percent) although these proportions varied by year of outbreak onset [Figure 3]. Among confirmed outbreaks, 24 (6.1 percent) were multicounty outbreaks of salmonellosis (12), *Escherichia coli* O157:H7 *infection* (6), campylobacteriosis (2), trichinellosis (2), scombroid (1), and norovirus infection (1). An additional 13 (3.9 percent) were multistate outbreaks of salmonellosis (9), and *Escherichia coli* O157:H7 infections (4).

The class of etiologic agents that accounted for the largest number of confirmed outbreaks and cases was bacterial agents [Figure 2, Table 1]. However, among specific etiologic agents, norovirus caused the most outbreaks (36.0 percent) and involved the most cases (39.7 percent). Among confirmed bacterial agents, *Salmonella* species caused the most outbreaks (125). During the surveillance period, the average annual numbers of *Escherichia coli* O157:H7 and *Salmonella* serotype Enteritidis

outbreaks were, respectively, 2.8 and 3.2.

A total of 155 (39.5 percent) confirmed outbreaks had an implicated food vehicle that was confirmed by either epidemiologic or laboratory evidence. The most commonly confirmed foods were vegetables and fruits (35, 22.6 percent), complex mixed food items such as burritos, sandwiches, or coleslaw (31, 20.0 percent), and meats (30, 19.4 percent) including beef, chicken, pork and bear. The proportion of FBDOs involving complex mixed food items was higher among FBDOs caused by viral agents (32.7 percent) than among FBDOs caused by bacterial agents (15.8 percent).

Notable outbreaks

In 2006, a multicounty outbreak of campylobacteriosis involving 1,644 cases (52 culture-confirmed) was linked to consumption of pasteurized milk. Cases were reported from 11 prison facilities. Pasteurized milk was traced to a dairy at a prison facility. *Campylobacter* spp. was not recovered from milk samples but laboratory testing of retention samples from the outbreak period had notably high levels of bacterial contamination. The outbreak was likely associated with contamination of pasteurized milk rather than inadequate pasteurization.

Also in 2006, a multistate outbreak of *E. coli* O157:H7 involving 205 cases, 103 hospitalizations, 30 cases of hemolytic uremic syndrome, and 3 deaths was linked to consumption of fresh, bagged, baby spinach[5]. Cases were reported from 26 states and Canada including 2 cases from California. Contaminated spinach was traced to 1 processing plant and 4 growing fields in California. Isolates of the outbreak strain were recovered from cattle feces, feral swine feces, surface waters, and soil sediment samples in or near the fields.

Comment

During the surveillance period, CDPH received an average of 170 FBDO reports each year although this is likely a fraction of the outbreaks that actually occurred. The identification, investigation, and reporting of FBDOs outbreaks represent a complex chain of events and under-reporting of outbreaks is well established[3,4]. The likelihood that an outbreak will be recognized and reported depends, among other things, on its size, severity, and scope.

During the surveillance period, fewer than 1 in every 3 California FBDOs had a laboratory-confirmed etiologic agent. Confirming the specific etiology of FBDOs provides critical information for developing focused control measures. Limited collection and/or testing of specimens during an outbreak can delay or impede the investigation[4].

Among confirmed outbreaks reported to CDPH, norovirus caused the most outbreaks and the most illnesses. FBDOs of norovirus infections and of salmonellosis tended to be large (based on the median number of cases per outbreak) and frequent. FBDOs of giardiasis tended to be large but infrequent. The multicounty outbreak of campylobacteriosis associated with pasteurized milk was notably large.

Outbreak investigations provide an important opportunity to understand the epidemiology of foodborne illnesses, identify needed illness prevention measures, and assess and build preparedness capacity for infectious disease emergencies. Reducing the occurrence of FBDOs requires coordination between public health and agricultural and food industries. These efforts, along with additional research and consumer education, may offer the best opportunities for controlling and preventing FBDOs.

References and additional resources

[1]Mead PS, Slutsker L, Dietz V et al. Food-related illness and death in the United States. Emerg Infect Dis 1999;5:607-25. http://www.cdc.gov/ncidod/eid/Vol5no5/pdf/mead.pdf

[2]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008 Technical notes http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

[3]Centers for Disease Control and Prevention. Surveillance for foodborne-disease outbreaks - United States, 1998-2002. MMWR 2006;55:SS-10.

[4]Jones TF, Imhoff B, Samuel M et al. Limitations to successful investigation and reporting of foodborne outbreaks: an analysis of foodborne disease outbreaks in FoodNet catchment areas, 1998-1999. 2004;38(Suppl 3)S297-302.

[5]Jay MT, Cooley M, Carychao D et al. *Escherichia coli* O157:H7 in feral swine near spinach fields and cattle, central California coast. 2007;13:1908-11.

Last update: 10/22/2009

Prepared by Kate Cummings, MPH, Charlotte Wheeler, MD, MPH, and Duc Vugia, MD, MPH, Infectious Diseases Branch

Epidemiologic Summary of Giardiasis in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 18,993 cases of giardiasis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average incidence rate of 6.5 cases per 100,000 Californians.
- Giardiasis incidence rates decreased by 42.0 percent from 2001 (8.8 per 100,000) to 2008 (5.1 per 100,000).
- Average giardiasis incidence rates during the surveillance period were higher among children 1 to 4 years of age (18.4 per 100,000) and 5 to 14 years of age (7.3 per 100,000), and among adults 35 to 44 years of age (7.4 per 100,000).
- From 2001 through 2008, CDPH received reports of 4 outbreaks of suspected waterborne giardiasis involving 86 cases. Of these outbreaks, suspected sources included a water source at a summer camp, a water dispenser at a commercial gym, and contact with water at a waste water treatment facility. Additionally, CDPH received reports of 2 foodborne outbreaks of giardiasis involving a total of 80 cases.
- Ensuring safe recreational and drinking water and educating the public about risk reduction measures, including good hygiene practices in child-care and recreational water settings, may provide the best opportunities for reducing giardiasis.

Background

Giardia intestinalis is the most commonly reported enteric parasite in the United States (US), causing an estimated 2 million infections, 5,000 hospitalizations, and 10 deaths each year[1]. Leading sources of *Giardia* infection include direct contact with an infected person (especially children in day care settings and among men who have sex with men) and ingestion of water contaminated by human or animal feces. Contaminated food is a less frequent source of infection. In the US, *Giardia* is an important cause of diarrheal illness outbreaks associated with recreational and drinking water, in part, because of its low infectious dose and moderate chlorine resistance. While animal contamination of drinking water has occurred, zoonotic transmission of giardiasis is not known to be a major source of human infection. There is no national *Healthy People 2010* target objective for giardiasis.

Acute illness, usually gastroenteritis, occurs after an incubation period of 3 to 25 days and can be associated with protracted symptoms and communicability. Occasionally, chronic intestinal symptoms develop and although extraintestinal invasion is rare, reactive arthritis can occur. *Giardia* is moderately resistant to chlorine and can survive for weeks in cold water.

We describe here the epidemiology of giardiasis in California from 2001 through 2008. Data for 2008 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[2].

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 working days of identification or immediately by telephone if an outbreak is suspected. Giardiasis is not included in state regulations requiring notification by laboratories to local health officials.

Local health officers are required by regulation to report to CDPH cases of giardiasis. CDPH officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC) surveillance case definition, including confirmed and probable classifications. During the surveillance period, CDC defined a confirmed case as one with detection of

Giardia intestinalis cysts in stool specimens by microscopic examination using staining methods or direct fluorescent antibody assays; or detection, by the same assays, of trophozoites in stool specimens, duodenal fluid, or small-bowel tissue; or detection of antigens in stool specimens by immunodiagnostic testing including asymptomatic infections. A probable case was one with clinically-compatible illness and an established epidemiologic link to a laboratory-confirmed case.

Epidemiology of giardiasis in California

CDPH received reports of 18,993 cases of giardiasis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average incidence rate of 6.5 cases per 100,000 Californians. Giardiasis incidence rates decreased by 42.0 percent from 2001 (8.8 per 100,000 population) to 2008 (5.1 per 100,000) ($p < 0.001$) although most of that decline occurred from 2001 to 2003 [Figure 1]. During the surveillance period, 35 (0.2 percent) cases were reported to have died with giardiasis during the surveillance period.

The average giardiasis incidence rates for the surveillance period were higher among children 1 to 4 years of age (18.4 per 100,000) and 5 to 14 years of age (7.3 per 100,000) and among adults 35 to 44 years of age (7.4 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 (39.3 percent). However, giardiasis cases with complete data reported White non-Hispanic race/ethnicity more frequently than would be expected based on the overall demographic profile of California [Figure 3].

Average incidence rates for the surveillance period were 2.1 times higher in Northern California (9.3 per 100,000) than in Southern California (4.4 per 100,000). In Northern California, the average rate decreased by 43.2 percent from the combined years of 2001 and 2002 (11.8 per 100,000) to the combined years of 2007 and 2008 (6.7 per 100,000) [Figure 4]. In Northern California, the San Francisco Bay Area, Far North, and Sierras regions had the highest average incidence rates during the surveillance period. In Southern California, the average rate decreased by 7.8 percent from the combined years of 2001 and 2002 (5.1 per 100,000) to the combined years of 2007 and 2008 (4.7 per 100,000).

Figure 1. California giardiasis case counts and incidence rates

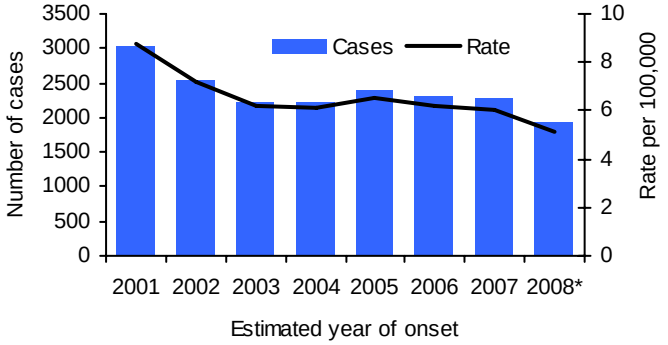


Figure 2. California giardiasis incidence rates by age and time period

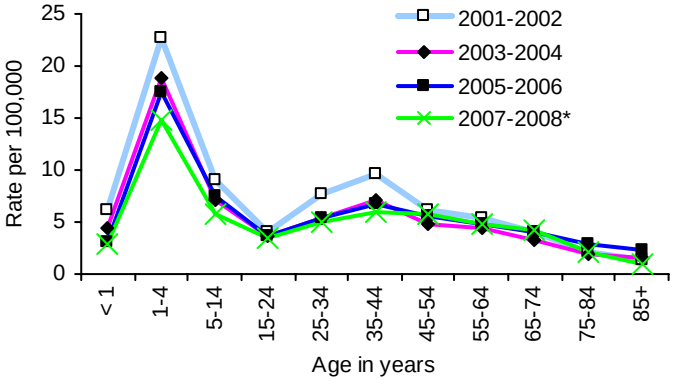
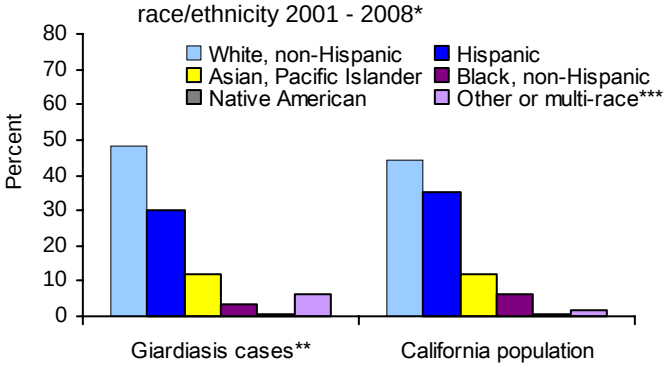


Figure 3. California giardiasis cases and population by race/ethnicity 2001 - 2008*



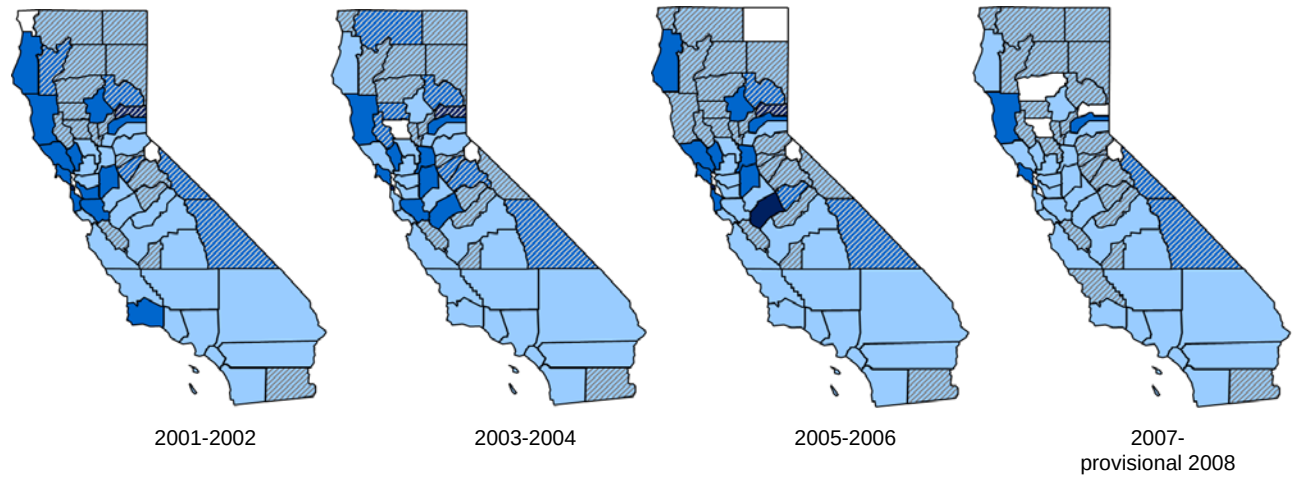
Notes for Figures 1-3

*2008 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



From 2001 through 2008, 6 outbreaks of suspected waterborne or foodborne giardiasis were reported to CDPH. Four suspected waterborne outbreaks involved 85 cases. Two of these 4 were suspected to be associated with a water filtration system at a Boy Scout camp and a water dispenser at a commercial gym. Two foodborne outbreaks of giardiasis involving a total of 80 cases were also reported; neither had a vehicle identified.

Comment

During the surveillance period, the highest annual number of giardiasis cases (3,049) was reported in 2001. California has experienced a significant decline in giardiasis incidence from 2001 to 2008. Reasons for this decrease are unknown, but declines might be associated with changes in laboratory testing and disease reporting practices, or changes in actual disease incidence. Continued monitoring of annual rates is needed.

Reducing giardiasis in California will require continued coordination between public health and drinking and recreational water quality control enforcement agencies. Educating the public about risk reduction measures, including safe hygiene practices for child care settings, recreational water settings, and for drinking water may provide the best opportunities for reducing giardiasis. Hand and personal hygiene practices in all settings, and safe sexual practices are also important for preventing and controlling this disease.

References and resources

[1]Mead PS, Slutsker L, Dietz V et al. Food-related illness and death in the United States. Emerg Infect Dis 1999;5:607-25.
http://www.cdc.gov/ncidod/eid/Vol5no5/pdf/mead.pdf

[2]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

Last update 8/17/2009

Prepared by Kate Cummings, MPH, and Amy Karon, DVM, Infectious Diseases Branch

Epidemiologic Summary of Hantavirus Pulmonary Syndrome (HPS) in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 16 cases of hantavirus pulmonary syndrome (HPS) with estimated onset dates from 2001 through 2008. The highest numbers of cases were in 2003 (5) and 2006 (4).
- During the surveillance period, 4 (25.0 percent) cases were reported to have died with HPS.
- The median age among cases was 45 years (range: 12 to 74 years) and the highest number of cases occurred among persons 55 to 64 years of age (4 cases).
- HPS cases reported White non-Hispanic (68.8 percent), Hispanic (25.0 percent) and Native American (6.3 percent) race/ethnicities. The ratio of male to female cases was 1.7:1.0.
- Avoiding contact with rodents and their excreta are primary strategies for reducing the risk of hantavirus exposure and provide the best opportunities for HPS prevention and control.

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). 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 is 30 to 40 percent.

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 the HPS cases in the US. Its reservoir, the deer mouse, is prevalent in undeveloped areas throughout the western US and will readily enter homes and buildings in search of food or nesting material. There is no national *Healthy People 2010* target objective for HPS.

We describe here the epidemiology of HPS in California from 2001 through 2008. Data for 2008 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[1]. Because of the small numbers of reported cases, incidence rates were not calculated.

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report suspected cases of hantavirus infections to their local health department immediately by telephone. HPS is not included in state regulations requiring notification by laboratories to local health officials.

Local health officers are required by regulation to report to CDPH cases of hantavirus infections. CDPH officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC) surveillance case definition for HPS. During the surveillance period, CDC defined a confirmed case as one with clinically compatible illness and laboratory confirmation. Clinically compatible illness included one or more of these clinical features: (i) a febrile illness (i.e., temperature greater than 101.0^{o} F [greater than 38.3^{o} C]) characterized by bilateral diffuse interstitial edema that may radiographically resemble ARDS, with respiratory compromise requiring supplemental oxygen, developing within 72 hours of hospitalization, and occurring in a previously healthy person or/and (ii) an unexplained respiratory illness resulting in death, with an autopsy examination demonstrating noncardiogenic pulmonary edema without an identifiable cause. Laboratory confirmation included 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 16 cases of HPS with estimated onset dates from 2001 through 2008. The highest numbers of cases were in 2003 and 2006 and the lowest numbers were in 2002 and 2007. There were no reported cases in 2001, 2005, and 2008 [Figure1]. During the surveillance period, 4 (25.0 percent) cases were reported to have died with HPS.

The number of HPS cases during the surveillance period was highest among persons 55 to 64 years of age [Figure 2]. The median age among cases was 45 years (range: 12 to 74 years). HPS cases reported White non-Hispanic (68.8 percent), Hispanic (25.0 percent) and Native American (6.3 percent) race/ethnicities. The ratio of male to female cases was 1.7:1.0.

Ten cases were residents of Northern California and 6 were residents of Southern California. The counties of Los Angeles (2), Mono (2), San Bernardino (2), and San Diego (2) were the only counties to report more than 1 case. Public health investigation of HPS cases revealed that the likely sites of exposure were the eastern Sierra Nevada (Mono, Inyo, Alpine Counties) for 7 cases, the northern Sierra Nevada (El Dorado, Nevada, Sierra, Plumas Counties) for 4 cases, the southern California mountains and deserts for 3 cases, and undetermined for 2 cases.

Figure 1. California HPS case counts by estimated onset year

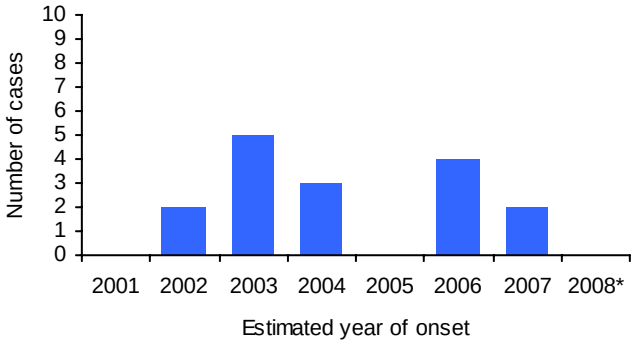
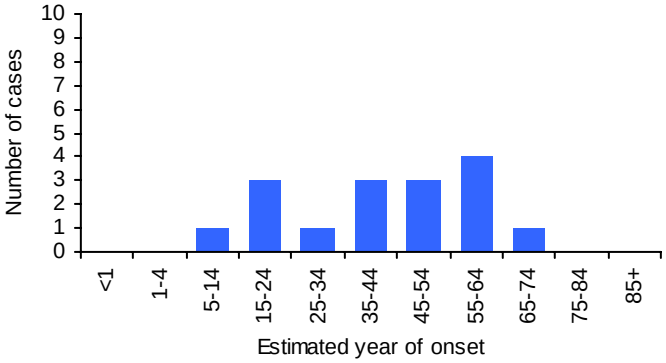


Figure 2. California HPS case counts by age-group



Notes for Figures 1 and 2
*2008 data are provisional

Comment

HPS infections are associated with domestic, occupational, or recreational activities that bring humans into contact with rodents and their excreta, usually in rural settings[2]. Many HPS cases 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. A substantial proportion of cases were residents of or visitors to the eastern Sierra Nevada; 3 cases were residents of coastal counties who became ill after camping in the eastern Sierra. Finally, follow-up investigations indicated that at least 4 cases may have been exposed at either their residence or their worksite, underscoring the ubiquity with which infected mice occur in some areas.

Avoiding contact with rodents and their excreta are primary strategies for reducing the risk of hantavirus exposure and provide the best opportunities for HPS prevention and control. Useful measures include preventing rodents from entering buildings, eliminating current rodent infestations, and proper respiratory protection when working in poorly ventilated areas infested with rodent excreta.

References and resources

[1] Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

[2] Hantavirus Pulmonary Syndrome—United States: Updated Recommendations for Risk Reduction. MMWR July 2002, 51 (RR09); 1-12.

Last updated 10/22/2009

Prepared by Farzaneh Tabnak, MS, PhD, Kate Cummings, MPH, and the Vector-borne Disease Section, Infectious Diseases Branch

Epidemiologic Summary of Infant Botulism in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 276 cases of infant botulism with onset dates from 2001 through 2008. This corresponds to an average incidence rate of 6.3 per 100,000 California resident live births.
- Infant botulism incidence rates steadily increased from the lowest of 4.0 per 100,000 live births in 2002 to the highest of 7.7 per 100,000 live births in 2005. Incidence rates decreased thereafter (to 6.4 per 100,000 live births in 2007 and 6.3 per 100,000 live births in 2008).
- Infant botulism cases with complete information on race/ethnicity were reported to be White non-Hispanic (49.6 percent) more frequently than would be expected based on the overall demographic profile of California resident live births (29.3 percent).
- During the surveillance period, just 32 (55.2 percent) of 58 counties reported at least 1 case.
- Honey is the only known avoidable source of *C. botulism* spores. Children less than 12 months of age should not be fed any foods containing honey.

Background

Infant botulism is a rare but serious paralytic illness in infants that results when swallowed spores of the causative bacterium *Clostridium botulinum* activate in the large intestine. These bacteria are commonly found in soil and dust and form spores allowing them to survive in dormant state. Ingested spores germinate, grow and release botulinum toxin in the gut. The illness is characterized by constipation, poor feeding, difficulty swallowing, lethargy, expressionless face, loss of head control and may be followed by progressive weakness, impaired respiration, and death. The case fatality rate among hospitalized infant botulism cases is less than 1 percent; however it is higher in areas of the world without access to hospitals with pediatric intensive care units.

Infant botulism occurs after the swallowing of food or dust that contains botulinum spores.

Honey can contain spores of *C. botulinum* and has been a source of infection for infants. Hence, infants under 1 year of age should not be fed honey.

We describe here the epidemiology of infant botulism in California from 2001 through 2008. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes[1].

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report suspected cases of botulism, including infant botulism, to their local health department immediately by telephone. Infant botulism is included in the state regulations requiring telephone notification within one hour, followed by a written electronic facsimile transmission or electronic mail within one working day, by laboratories to local health officials.

Local health officers are required by regulation to report cases of infant botulism to CDPH, which tabulated confirmed cases that satisfied the Centers for Disease Control and Prevention (CDC) surveillance case definition. During the surveillance period, CDC defined a confirmed case as an illness of an infant less than 1 year of age, characterized by constipation, poor feeding, and “failure to thrive” that may be followed by progressive weakness, impaired respiration, and death, in which botulinum toxin was identified either in serum or stool, or in which *C. botulinum* was isolated from feces.

Epidemiology of infant botulism in California

CDPH received reports of 276 cases of infant botulism with onset dates from 2001 through 2008. This corresponds to an average incidence of 6.3 per 100,000 California live births. Infant botulism incidence steadily increased from the lowest of 4.0 per 100,000 live births in 2002 to the highest of 7.7 per 100,000 live births in 2005. Incidence decreased to the average

rate in 2007 and 2008 (6.4 and 6.3 per 100,000 live births, respectively) [Figure 1].

The ratio of male to female cases was 0.9:1.0. Infant botulism cases with complete information on race/ethnicity (>98 percent of all cases) were reported to be White non-Hispanic (49.6 percent) more frequently than would be expected based on the overall demographic profile of California resident live births (29.3 percent).

During the surveillance period, just 32 (55.2 percent) of 58 counties reported at least 1 case. Only rates reported by the counties of Los Angeles (7.3 per 100,000 live births) and San Bernardino (10.8 per 100,000 live births) were statistically reliable [Figure 2].

Figure 1. California infant botulism case counts and incidence rates

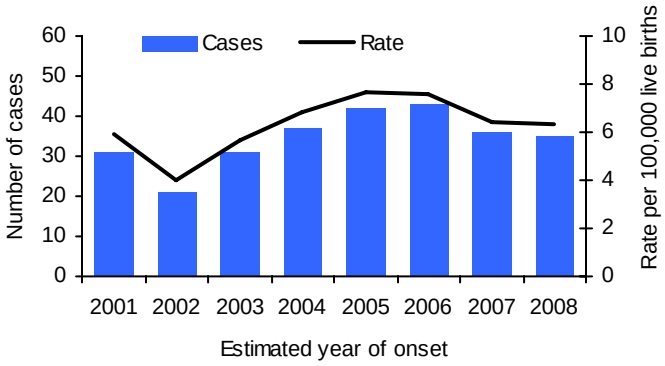
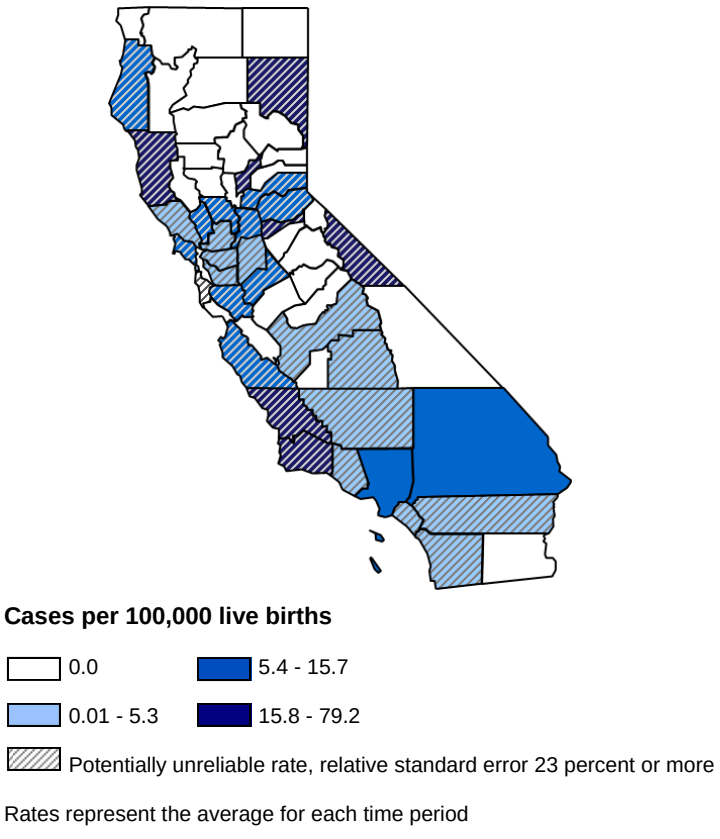


Figure 2. California county-specific infant botulism incidence rates 2001 - 2008



Comment

During the surveillance period, the highest annual number of infant botulism cases was reported in 2006. Infant botulism cases occurred throughout the state. Honey can contain spores of *C. botulinum* and has been a source of infection for infants. Infants under 1 year of age should not be fed any foods containing honey.

Human Botulism Immune Globulin Intravenous (BabyBIG®), a public service orphan drug created and developed by CDPH 1988-2003, was licensed by the FDA for the treatment of infant botulism in 2003. BabyBIG® is available through the CDPH Infant Botulism Treatment and Prevention program (24/7 telephone 510-231-7600; http://www.infantbotulism.org).

References and resources

[1]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

Last updated 07/09/2010

Prepared by Farzaneh Tabnak, MS, PhD, Haydee Dabritz, PhD, Kate Cummings, MPH, and Stephen Arnon, MD,MPH Infectious Diseases Branch and Infant Botulism Treatment & Prevention Program

Epidemiologic Summary of Legionellosis in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 725 cases of legionellosis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 0.25 cases per 100,000 Californians.
- Legionellosis incidence rates increased by 175.0 percent from 2001 (0.16 per 100,000) to 2008 (0.44 per 100,000).
- During the surveillance period, 69 (9.5 percent) reported cases died with legionellosis.
- Average legionellosis incidence rates during the surveillance period increased with increasing age and were highest among adults 75 to 84 years of age (1.22 per 100,000).
- Average incidence rates for the surveillance period were 1.8 times higher in Southern California (0.30 per 100,000) compared to Northern California (0.17 per 100,000).
- One nosocomial outbreak of legionellosis involving 18 cases was reported in Southern California in 2002.
- Further study may help determine if increased legionellosis incidence rates in California represent a true increase in disease activity, detection, reporting, or some combination thereof.

Background

Legionella is an important respiratory bacterial pathogen in the United States (US), causing between 8,000 and 18,000 cases of community-acquired pneumonias requiring hospitalization each year[1]. Inhaling or aspirating contaminated water aerosols are the leading sources of infection. *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 air-circulation 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. Most cases are now diagnosed by urine antigen, which is highly specific 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 generally 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. There is no national *Healthy People 2010* target objective for legionellosis.

We describe here the epidemiology of legionellosis in California from 2001 through 2008. Data for 2008 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[2].

California reporting requirements and surveillance case definitions

California Code of Regulations, Title 17, requires health care providers to report suspected cases of legionellosis to their local health department within one working day 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.

Local health officers are required by regulation to report to CDPH cases of legionellosis. CDPH officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC) 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. From 2001 through 2004, CDC criteria also included detection of *L. pneumophila* serogroup 1 by direct fluorescent antibody staining.

Epidemiology of legionellosis in California

CDPH received reports of 725 cases of legionellosis with estimated symptom onset dates from 2001 through 2008. This corresponded to an average incidence rate of 0.25 cases per 100,000 Californians. Legionellosis incidence rates increased by 175.0 percent from 2001 (0.16 to 100,000) to 2008 (0.44 per 100,000) ($p < 0.001$) [Figure 1]. During the surveillance period, 69 (9.5 percent) cases were reported to have died with legionellosis.

During the surveillance period, average legionellosis incidence rates increased with increasing age and were highest among adults 75 to 84 years of age (1.22 per 100,000). Incidence rates increased from 2001 to 2008 in all persons over 14 years of age [Figure 2] but was most pronounced among the elderly. 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 (13.7 percent). However, legionellosis cases with complete data reported White non-Hispanic race/ethnicity more frequently than would be expected based on the demographic profile of California [Figure 3].

Average incidence rates for the surveillance period were 1.8 times higher in Southern California (0.30 per 100,000) compared to Northern California (0.17 per 100,000). From 2001 to 2008, incidence rates

Figure 1. California legionellosis case counts and incidence rates

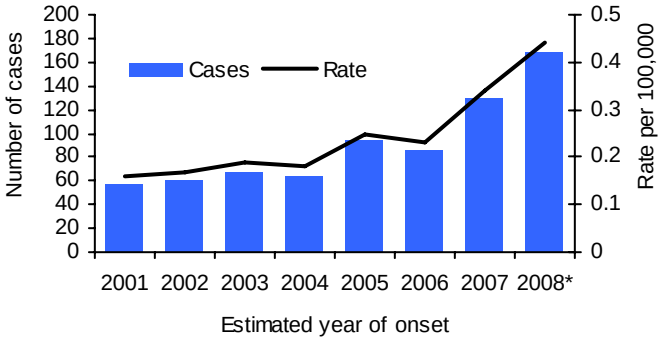


Figure 2. California legionellosis incidence rates by age and time period

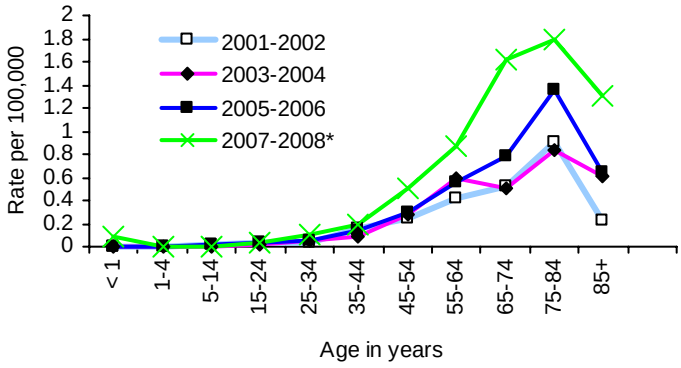
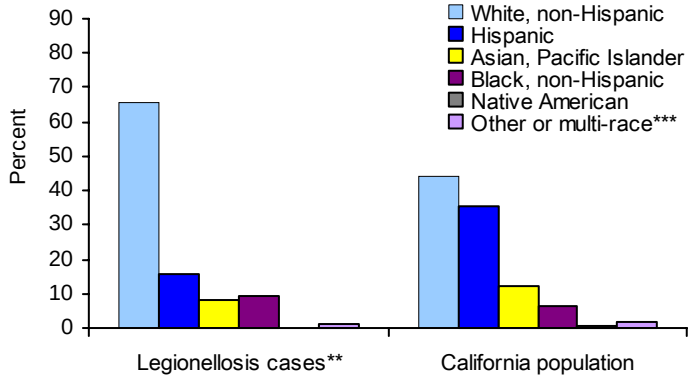


Figure 3. California legionellosis cases and population by race/ethnicity 2001 - 2008*



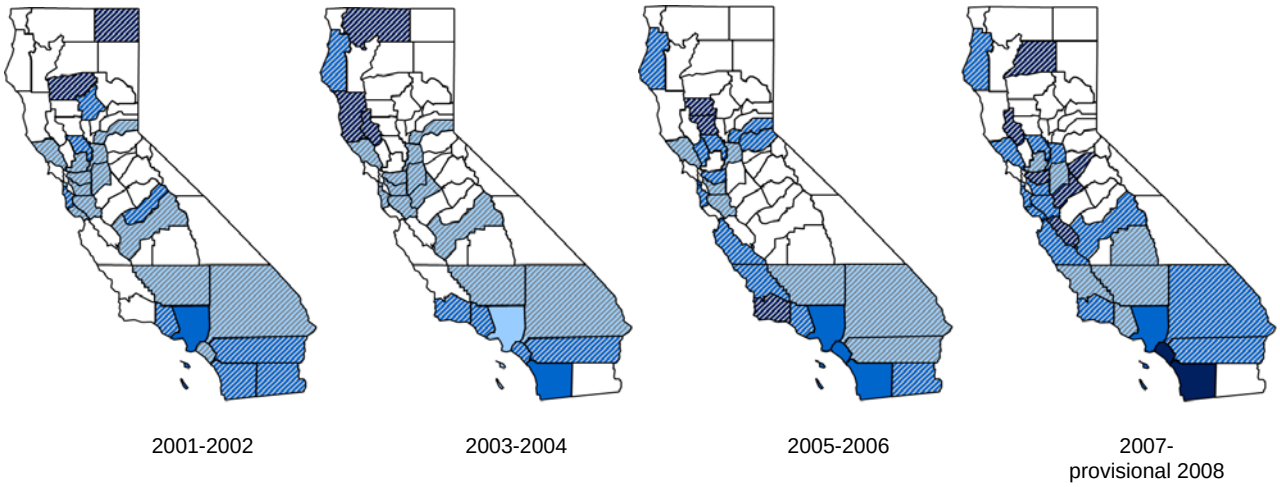
Notes for Figures 1-3

*2008 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



increased by 135.0 percent in Southern California (from 0.20 to 0.47 per 100,000) and by 141.7 percent in Northern California (from 0.12 to 0.29 per 100,000). The Central Coast, San Joaquin Valley, and South Coast regions reported the greatest overall increases [Figure 4].

One nosocomial outbreak of legionellosis involving 18 cases was reported in a Southern California acute care hospital in 2002 and was attributed to colonization of the potable water system.

Comment

During the surveillance period, the highest annual number of legionellosis cases (168) was reported in 2008. California has experienced a significant increase in reported legionellosis incidence rates from 2001 to 2008 An abrupt increase in reported legionellosis cases from 2002 through 2005 has also been noted nationally[3,4]. Expanded regulatory requirements for laboratory-based reporting may partially explain the increases in California in 2007 and 2008. However, increases in national legionellosis incidence rates were not clearly associated with changes in either diagnostic or physician or laboratory reporting practices. Further study is needed to determine if increased rates in California represent an increase in disease activity, detection, reporting, or some combination thereof.

References and resources

[1] Marston BJ, Plouffe JF, File TM JR et al.; Community-Based Pneumonia Incidence Study Group. Incidence of community-acquired pneumonia requiring hospitalization: results of a population-based active surveillance study in Ohio. Arch Intern Med 1997;157:1709-18.
[2]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf
[3] Neil K, Berkelman R. Increasing incidence of legionellosis in the United States, 1990-2005: changing epidemiologic trends. Clin infect Dis 2008;47:591-9.
[4] Ng V, Tang P, Fisman DN. Our evolving understanding of legionellosis epidemiology: learning to count. Clin Infect Dis 2008;47:600-2.

Centers for Disease Control and Prevention
http://www.cdc.gov/legionella/index.htm

Last updated: 8/17/2009

Prepared by Kate Cummings, MPH, Jon Rosenberg, MD, and Duc Vugia, MD, MPH, Infectious Diseases Branch

Epidemiologic Summary of Listeriosis in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 868 cases of listeriosis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average incidence rate of 0.30 cases per 100,000 Californians.
- Annual listeriosis incidence rates in California decreased by 26.5 percent from 2001 (0.34 per 100,000) to 2008 (0.25 per 100,000). However, incidence rates were elevated from 2004 through 2006 (average rate: 0.34 per 100,000) and then decreased thereafter.
- During the surveillance period, 73 (8.4 percent) cases were reported to have died with listeriosis during the surveillance period.
- Average listeriosis incidence rates during the surveillance period were highest among children under 1 year of age (1.65 per 100,000), and adults 65 years of age or older (1.17 per 100,000). Among Californians 15 to 44 years of age, the average rate of listeriosis was 3.6 times higher in women (0.25 per 100,000) than in men (0.07 per 100,000).
- From 2001 through 2008, CDPH received reports of 2 foodborne outbreaks of listeriosis (1 confirmed, 1 suspected) involving 84 cases. The confirmed outbreak involved 28 cases and was associated with delicatessen turkey meat.
- Improving the safety of processed meats and educational outreach to high-risk consumers such as pregnant women, the immunocompromised, and the elderly may provide the best opportunities for reducing listeriosis.

Background

In the United States (US), listeriosis is an uncommon but important foodborne illness and is associated with an estimated 2,500 severe infections and 500 deaths each year[1]. *Listeria monocytogenes* is ubiquitous in the environment and immunocompetent persons may have only a mild acute febrile illness or gastroenteritis. However, listeriosis is a leading cause of foodborne-related deaths in the US because of the severity of illness among certain populations. The elderly, immunocompromised persons, and pregnant women and neonates are at increased risk for severe illness including meningo-encephalitis and/or septicemia. Infected pregnant women may experience only a mild illness but infection can lead to premature delivery, miscarriage, stillbirth, or serious infection in the newborn.

Consuming contaminated foods, including unpasteurized milk products and ready-to-eat meats, is the leading source of infection. Outbreaks of listeriosis have been associated with deli meats and unpasteurized milk products including Mexican-style fresh soft cheese[2]. On rare occasions, pasteurized milk has been implicated in outbreaks[3]. Unlike other foodborne pathogens, *Listeria* will multiply in refrigerated temperatures. The national *Healthy People 2010* target objective for listeriosis is no more than 0.25 new cases per 100,000 people.

We describe here the epidemiology of listeriosis in California from 2001 through 2008. Data for 2008 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 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. Clinical and reference laboratories must also notify the local health department when laboratory testing yields evidence suggestive of *Listeria*; notification 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 listeriosis. CDPH officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC) surveillance case definition. During the surveillance period, CDC defined a confirmed case 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.

Epidemiology of listeriosis in California

CDPH received reports of 868 cases of listeriosis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average incidence rate of 0.30 cases per 100,000 Californians. Incidence rates decreased by 26.5 percent from 2001 (0.34 per 100,000) to 2008 (0.25 per 100,000) [Figure 1]. However, incidence rates were elevated from 2004 through 2006 (average rate: 0.34 per 100,000) and then decreased thereafter. During the surveillance period, 73 (8.4 percent) cases were reported to have died with listeriosis.

Average listeriosis incidence rates were highest among children under 1 year of age (1.65 per 100,000), and adults 65 years of age or older (1.17 per 100,000). Incidence rates among children under 1 year of age decreased by 71.8 percent from the combined years 2001 and 2001 (2.27 per 100,000) to the combined years 2007 and 2008 (0.64 per 100,000) [Figure 2]. The ratio of female to male cases was 1.2:1.0. Among Californians 15 to 44 years of age, the average incidence rate of listeriosis was 3.6 times higher in women (0.25 per 100,000) than in men (0.07 per 100,000). Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (21.7 percent). However, listeriosis cases with complete information reported race/ethnicities that were similar to those expected based on the demographic profile of California [Figure 3].

Average incidence rates for the surveillance period were similar in Northern California and Southern California. During the years 2007 and 2008 combined, 16 counties reported incidence rates (range: 0.30 per 100,000 - 1.66 per 100,000) that were above the *Healthy People 2010* target objective [Figure 4].

From 2001 through 2008, CDPH received reports of 1 confirmed and 1 suspected foodborne outbreak of

Figure 1. California listeriosis case counts and incidence rates

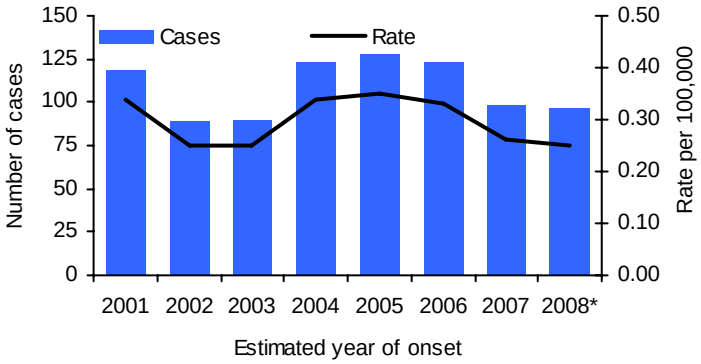


Figure 2. California listeriosis incidence rates by age and time period

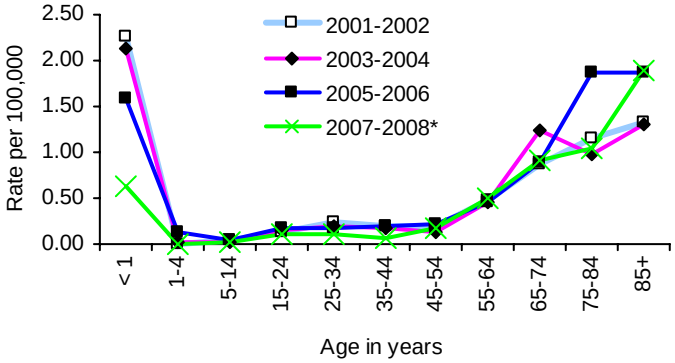
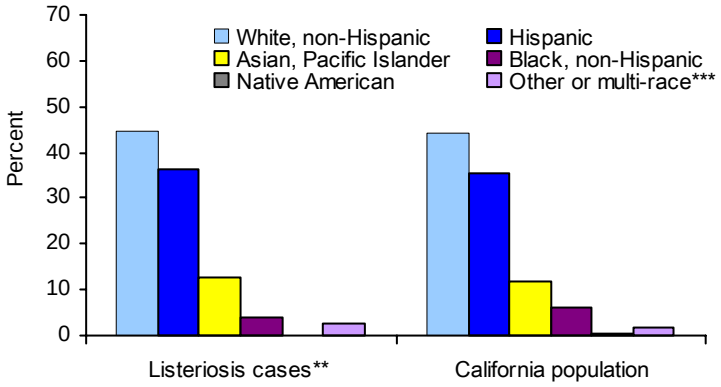


Figure 3. California listeriosis cases and population by race/ethnicity 2001 - 2008*



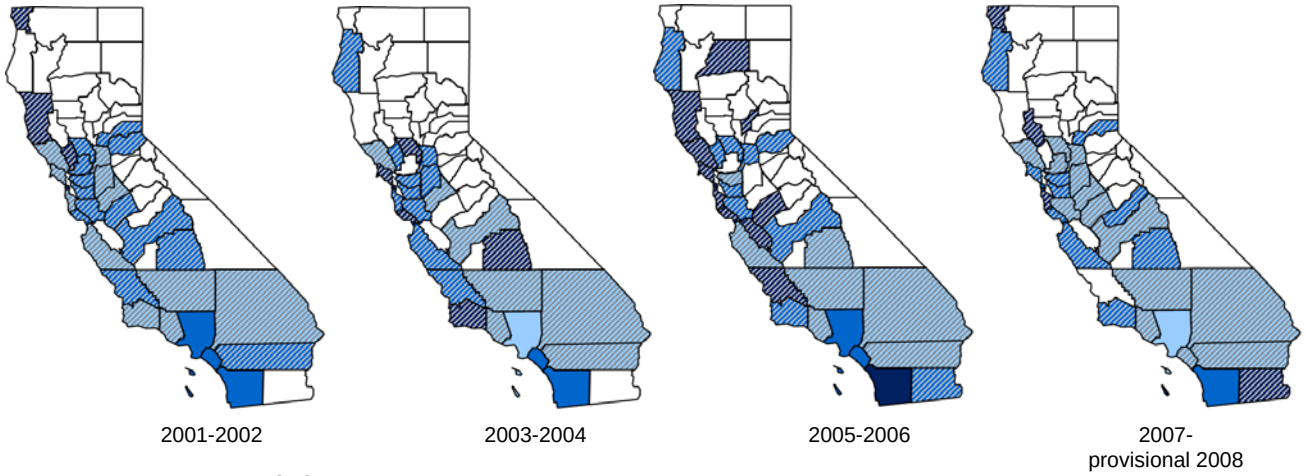
Notes for Figures 1-3

*2008 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 listeriosis incidence rates



listeriosis involving 84 cases. The confirmed outbreak occurred in 2001, involved 28 cases, and was associated with delicatessen turkey meat.

Comment

During the surveillance period, the highest annual number of listeriosis cases (128) was reported in 2005. From 2001 to 2008, annual incidence rates have decreased modestly. However, why incidence rates were higher during the period 2004 through 2006 is unexplained. Increases during this period were also reported nationally. The average annual incidence for the surveillance period (0.30 per 100,000) was 16.7 percent above the *Healthy People 2010* target objective (0.25 per 100,000) although the incidence rate in 2008 (0.25 per 100,000) met the target. As expected, incidence rates were elevated in the very young and the elderly. Although we did not have data on pregnancy, we noted increased incidence rates among women of childbearing age.

Improving the safety of processed meats and educational outreach to high-risk consumers such as pregnant women, the immunocompromised, and the elderly may provide the best opportunities for reducing 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.

References and resources

[1]Mead PS, Slutsker L, Dietz V et al. Food-related illness and death in the United States. Emerg Infect Dis 1999;5:607-25.
http://www.cdc.gov/ncidod/eid/Vol5no5/pdf/mead.pdf

[2]Centers for Disease Control and Prevention. Outbreak of listeriosis associated with homemade Mexican-style cheese -- North Carolina, October 2000-January 2001. MMWR 2001:50(26);560-2.

[3]Centers for Disease Control and Prevention. Outbreak of Listeria monocytogenes infections associated with pasteurized milk from a local dairy -- Massachusetts, 2007. MMWR 2008:57(40);1097-1100.

[4]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

Centers for Disease Control and Prevention listeriosis information webpage
http://www.cdc.gov/nczved/dfbmd/disease_listing/listeriosis_gi.html

Last update 8/17/2009

Prepared by Kate Cummings, MPH, and Akiko Kimura, MD, Infectious Diseases Branch

Epidemiologic Summary of Lyme disease in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received 660 reports of Lyme disease with estimated symptom onset dates from 2001 through 2008. This corresponded to an average annual incidence rate of 0.23 cases per 100,000 Californians.
- Counties of the north coast and the western Sierra Nevada reported the highest county-specific 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 20,000 cases reported in the United States (US) in 2006[1]. Over 90 percent of cases occur in the northeastern, mid Atlantic, and upper mid western states.

The most common initial sign of Lyme disease is a red, expanding rash (erythema migrans) that appears 3 to 30 days after the bite of an infected tick. If not treated, 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.

We describe here the epidemiology of Lyme disease in California from 2001 through 2008. Data for 2008 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[2].

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 7 working days of identification or immediately by telephone if an outbreak is suspected. Starting in 2005, laboratories must report to the local health department when laboratory testing yields evidence suggestive of *B. burgdorferi* infection; notification 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 officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC) surveillance case definition. From 2001 through 2007, CDC defined a confirmed case as (i) physician diagnosed erythema migrans of at least 5 cm diameter or (ii) at least 1 objective late manifestation (i.e., musculoskeletal, cardiovascular, or neurological) and either isolation of *B. burgdorferi* from a clinical specimen or demonstration of diagnostic levels of immunoglobulin (Ig) M or IgG antibodies to *B. burgdorferi* in serum or cerebrospinal fluid (CSF). A two-test approach (a sensitive enzyme immunoassay or immunofluorescence antibody assay followed by a Western blot) was recommended but not required. In 2008, CSTE defined a confirmed case as: (i) physician diagnosed erythema migrans with either a known exposure or laboratory evidence of infection or (ii) at least 1 objective late manifestation and laboratory evidence of infection. In 2008, laboratory evidence of infection included: (1) a positive culture of *B. burgdorferi* or (2) two-tiered testing interpreted using established criteria or (3) single-tier IgG immunoblot seropositivity interpreted using established criteria.

Epidemiology of Lyme disease in California

CDPH received 660 reports of Lyme disease with estimated symptom onset dates from 2001 through 2008. This corresponded to an average annual incidence rate of 0.23 cases per 100,000

Californians. Rates remained relatively level from 2001 to 2008 with the exception of a temporary decrease in 2004 (to 0.14 per 100,000) [Figure 1].

Average annual incidence rates for the surveillance period were highest among persons 45 to 54 years of age (0.32 per 100,000), 55 to 64 years of age (0.30 per 100,000), and 65 to 74 years of age (0.31 per 100,000). The ratio of male to female cases was 1.0:1.0. Rates by race/ethnicity were not calculated due to the substantial portion of missing data (24.4 percent). However, Lyme disease cases with complete data reported White non-Hispanic race/ethnicity (85.2 percent) more frequently than would be expected (44.5 percent) based on the overall demographic profile of California.

Trinity, Mendocino, Humboldt, Mono, Sierra, and Nevada Counties reported the highest average rates during the surveillance period [Figure 3]. Of 393 cases for whom likely location of exposure was reported, 178 (45.3%) were likely exposed outside California in another state or country. A total of 308 (46.7 percent) cases had estimated onsets during the months of June through August.

Comment

The western black-legged tick, the main Lyme disease vector in the western US, has been found in many wooded and grassy areas in California. People are most commonly exposed to the Lyme disease agent by the immature nymphal tick which is active in the spring and early summer; an average of 5-15% of nymphal western black-legged ticks in California are infected with *B. burgdorferi*. 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 for ticks daily, and promptly removing attached ticks[3].

References and resources

[1] CDC. Surveillance for Lyme Disease - United States, 1992 -2006. MMWR 2008;57:1-9.
[2]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf
[3]CDPH Lyme disease information webpage:
http://www.cdph.ca.gov/HealthInfo/discond/Pages/LymeDisease.aspx

Last updated: 11/02/2009

Prepared by Kate Cummings, MPH, and the Vector-borne Disease Section, Infectious Diseases Branch

Figure 1. California Lyme disease case counts and incidence rates

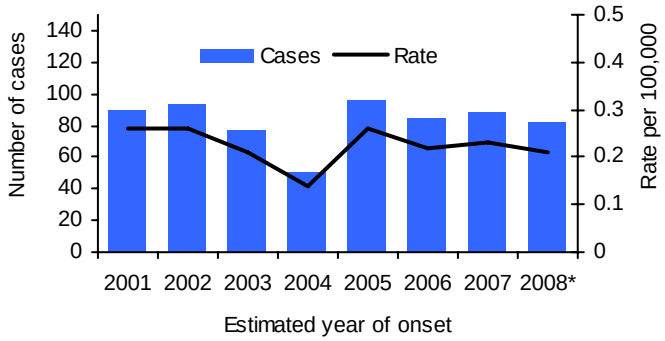


Figure 2. California Lyme disease incidence rates by age, 2001 - 2008*

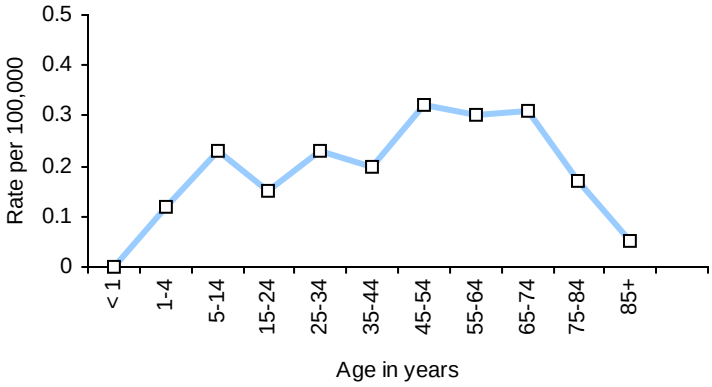
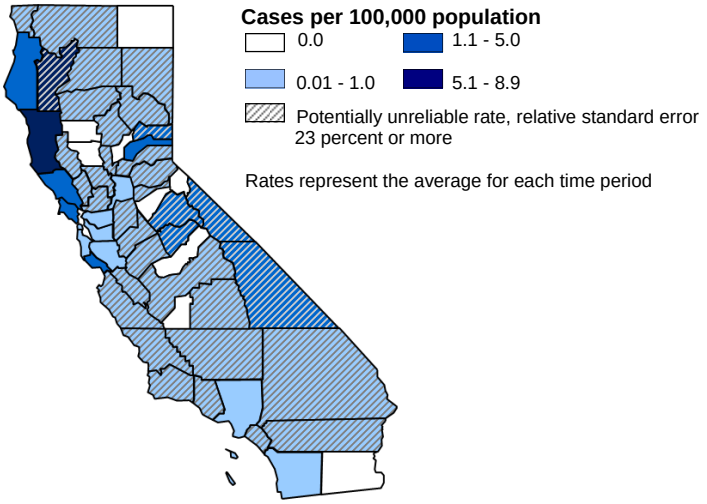


Figure 3. California county-specific Lyme disease incidence rates, 2001 - 2008*



Notes for Figures 1-2

*2008 data are provisional

Epidemiologic Summary of Human Q Fever in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 120 cases of Q Fever with estimated onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 0.04 per 100,000 Californians.
- Annual Q Fever incidence rates increased by 150.0 percent from 2001 (0.02 per 100,000) to 2008 (0.05 per 100,000).
- Average annual Q Fever incidence rates during the surveillance period were higher among persons 55 to 64 years of age (0.10 per 100,000).
- The ratio of male to female cases was 4.7:1.0.
- Average annual incidence rates were higher in the regions of: the Sierras (0.33 per 100,00), Inland Empire (0.33 per 100,00), and San Joaquin Valley (0.11 per 100,000).
- 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.

Background

Coxiella burnetii is an uncommon but important bacterial zoonotic pathogen in the United States (US). Inhaling bio-aerosols generated from infected animals (especially parturient goats, sheep, and cattle) and their byproducts is a leading source of human exposure. Contact with bio-droplets and fomites, and consumption of raw milk products may also result in infection. *C. burnetii* is listed among the Centers for Disease Control and Prevention (CDC) category B bioterrorism (BT) agents.

Q Fever has a variable incubation period (usually 3 to 30 days) that is dose-dependent, may manifest as a non-specific febrile syndrome, pneumonia, or hepatitis, and can occur in acute, chronic, and asymptomatic forms. Most cases resolve without complication. Chronic Q Fever occurs in fewer than 1 percent of cases and may be accompanied by severe endocarditis or granulomatous hepatitis. There is no vaccine licensed in the United States to protect against Q Fever.

We describe here the epidemiology of human Q Fever in California from 2001 through 2008. Data for 2008 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[1].

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 7 calendar days of identification or immediately by telephone if an outbreak is suspected.

California regulations also require local health officers to report to CDPH cases of Q Fever. CDPH officially counted cases that satisfied the CDC surveillance case definition. From 2001 through 2007, CDC defined a confirmed case as with one with (i) clinically compatible illness or an epidemiological link and (ii) laboratory confirmation defined as ≥4 fold change in antibody titer to *C. burnetii* phase I or II antigen in paired serum specimens, or isolation of *C. burnetii* from a clinical specimen by culture, or demonstration of *C. burnetii* in a clinical specimen by detection of antigen or nucleic acid. A probable case was one with supportive serology and clinically compatible illness or an epidemiologic link to a confirmed case. In 2008, CDC revised the surveillance case definition to distinguish acute from chronic Q Fever cases and to include laboratory detection of *C. burnetii* DNA in a clinical specimen by amplification of a specific target by polymerase chain reaction assay or by immunohistochemical methods.

Epidemiology of Q Fever in California

CDPH received reports of 120 cases of Q Fever with estimated onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 0.04 per 100,000 Californians. Q Fever incidence rates increased by 150.0 percent from 2001 (0.02 per 100,000) to 2008 (0.05 per 100,000) ($p < .001$) [Figure 1]. During the surveillance period, no cases were reported to have died with Q Fever.

Average annual Q Fever incidence rates for the surveillance period were highest among persons 55 to 64

years of age (0.10 per 100,000) [Figure 2]. The ratio of male to female cases was 4.7:1.0. During the surveillance period, Q Fever cases with complete information on race/ethnicity (80.3 percent) reported White non-Hispanic race/ethnicities (59.8 percent) more frequently than would be expected based on the overall proportion of White non-Hispanics (44.4 percent) in the California population.

Average annual incidence rates for Q Fever were higher in Northern California (0.05 per 100,000) than in Southern California (0.03 per 100,000). For the surveillance period, average annual incidence rates reported from the regions of the Sierras (0.33 per 100,00), Inland Empire (0.33 per 100,00), and San Joaquin Valley (0.11 per 100,000) were the highest in the state [Figure 3].

Comment

California has experienced a significant increase in the incidence of human Q Fever from 2001 to 2008. Regions of California with higher Q Fever incidence rates are areas associated with large numbers of commercial and backyard goat and sheep flocks.

Nationally reported Q Fever, introduced in 1999, increased by 714 percent from 2000 (21 cases) to 2007 (171 cases)[2,3]. Classification of *C. burnetii* as a BT agent and the subsequent introduction of national reporting has likely been accompanied by increased diagnostic suspicion and reporting. Further study is needed to determine the fraction of California's increases that were attributable to increases in disease activity, detection, and/or reporting.

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.

References and resources

[1]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf
[2]Karakousis PC et al. Chronic Q Fever in the United States. J Clin Microbiol 2006;44:2283-7.
[3]CDC. Surveillance of notifiable diseases - United States, 2007. MMWR 2009;56:1-94.
CDPH Q Fever information website:
http://www.cdph.ca.gov/HealthInfo/discond/Pages/QFever.aspx

Last updated 10/22/2009
Prepared by Kate Cummings, MPH, James Glover, MS, DVM, MPVM, MPH, and Ben Sun, DVM, MPVM, Infectious Diseases Branch

Figure 1. California Q Fever case counts and incidence rates

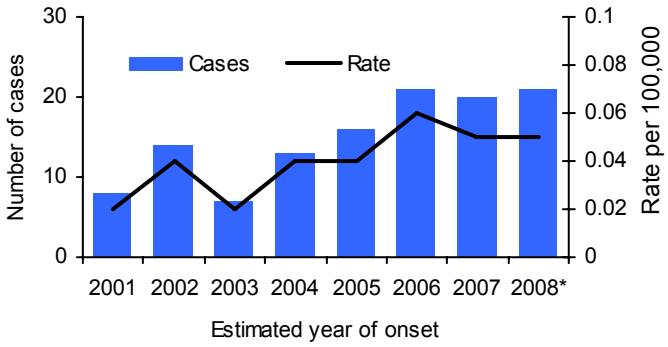


Figure 2. California Q Fever incidence rates by age, 2001-2008*

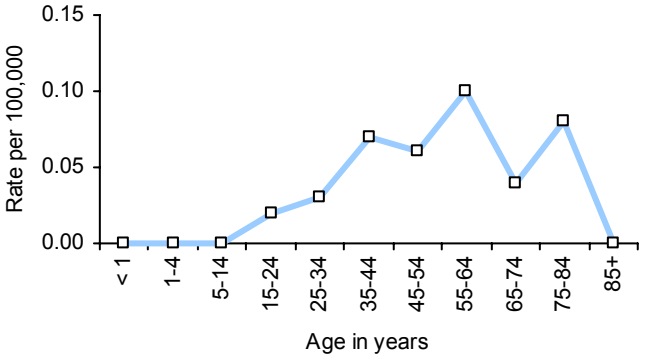
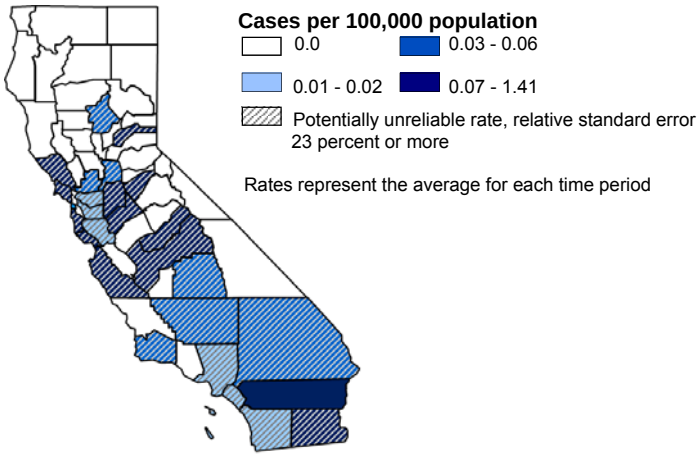


Figure 3. California county-specific Q Fever incidence rates, 2001 - 2008*



Notes for Figures 1-3
*2008 data are provisional

Epidemiologic Summary of Animal and Human Rabies in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 1,747 animal rabies cases from 2001 through 2008. Reported animal cases decreased in California by 44.5 percent from 2001 (321) to 2008 (178).
- Among animal rabies cases, the most frequently reported species were bats (1,276, 73.0 percent), skunks (410, 23.5 percent), foxes (39, 2.3 percent), and cats (11, 0.6 percent).
- The annual number of rabid bats reported to CDPH decreased by 17.5 percent from 2001 (166) to 2008 (137). Rabid bats were most frequently reported from the Far North (17.7 percent of 1,276), Bay Area (21.3 percent), Sacramento Metro (13.7 percent), and South Coast (13.7 percent) regions.
- The annual number of rabid skunks reported to CDPH decreased by 79.5 percent from 2001 (151) to 2008 (31). Rabid skunks were most frequently reported from the Central Coast (32.4 percent of 410), Far North (25.1 percent), and Sacramento Metro (17.8 percent) regions.
- During the surveillance period, 6 human cases of rabies were reported to CDPH. Four of 6 human rabies cases resulted from exposures that occurred outside of the US and 2 resulted from bat exposures in California.
- Appropriate domestic and wild animal management, animal vaccination programs, public health and medical management of persons exposed to potentially rabid animals, public education about animal risk reduction strategies, and avoiding wild animal contact may 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 common in some wild animal species but is rare in domestic animals. The primary animal reservoirs for rabies in California are bats and skunks; each has its own species-specific rabies virus variants. Contact with the saliva of a rabid animal by direct bite is the leading source of rabies virus exposure in humans. Although rare, rabies can be transmitted by contact through open wounds or mucus membranes with infected animal saliva. Transmission of virus has been documented through human organ and corneal transplant (from undiagnosed donor to recipient).

Human rabies has a variable and sometimes prolonged incubation period (7 days to 6 years). After an initial non-specific febrile prodromal phase (headache, fever, malaise, a sense of apprehension, and indefinite sensory changes), patients rapidly progress to an almost invariably fatal acute encephalomyelitis. Although uncommon, human rabies retains its public health significance because of the lethality of human infections. Guidance on public health investigation and management of potentially exposed humans, and surveillance and management of animals subject to rabies in California are described elsewhere[1,2].

We describe here the epidemiology of animal and human rabies in California from 2001 through 2008. Data for 2008 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[3].

California reporting requirements and surveillance case definition

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 laboratory 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 a mammal, or the

whereabouts of an animal suspected to have rabies. In areas declared by CDPH to be rabies areas, persons must also report to the LHO information regarding persons 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. CDPH officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC) surveillance case definition. 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 one with detection by direct fluorescent antibody of viral antigens in a clinical specimen (preferably the brain or the nerves surrounding hair follicles in the nape of the neck), or isolation in cell culture or in a laboratory animal of rabies virus from saliva, cerebrospinal fluid (CSF), or central nervous system tissue, or identification of a rabies-neutralizing antibody titer greater than or equal to 5 (complete neutralization) in the serum or CSF of an unvaccinated person.

Epidemiology of rabies in California

Animal cases

During the surveillance period, CDPH received reports of 1,747 animal rabies cases. Animal cases occurred in bats (1,276, 73.0 percent), skunks (410, 23.5 percent), foxes (39, 2.3 percent), cats (11, 0.6 percent), dogs (4, 0.2 percent), equine (2, 0.1 percent), raccoons (2, 0.1 percent), coyote (1, < 0.1 percent), opossum (1, < 0.1 percent), and rabbit (1, < 0.1 percent).

The annual number of animal cases reported to CDPH decreased by 44.5 percent from 2001 (321) to 2008 (178) [Figure 1]. The annual number of rabid bats decreased by 17.5 percent from 2001 (166) to 2008 (137) [Figure 2]. The annual number of rabid skunks decreased by 79.5 percent from 2001 (151) to 2008 (31).

Rabid bats were most frequently reported from the Far North (17.7 percent of 1,276), Bay Area (21.3 percent), Sacramento Metro (13.7 percent), and South Coast (13.7 percent) regions. The number of cases reported from most geographic regions remained level from the combined years of 2001 through 2004 to the combined years of 2005

Figure 1. Number of reported animal rabies cases in California

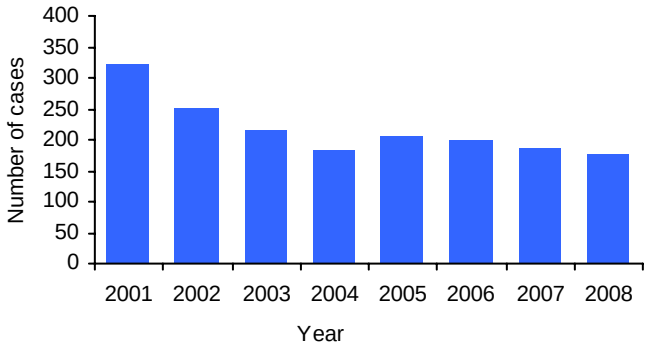


Figure 2. Reported animal cases of rabies in California by species and year

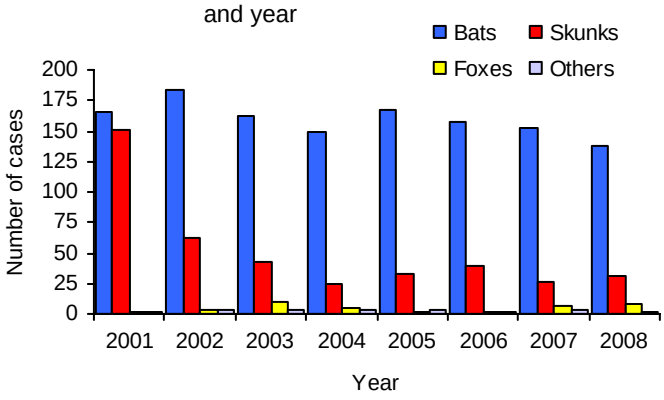
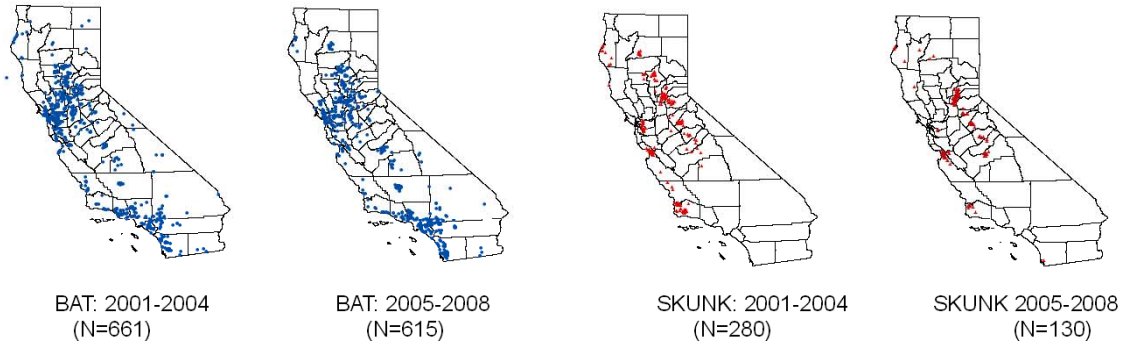


Table 1. Reported human rabies cases in California

Year	Age	Sex	County	Rabies virus variant
2001	72	Male	San Die-go*	Dog, Philippines
2002	28	Male	Glenn	Bat, Mexican free-tailed
2003	66	Male	Trinity	Bat, Silver haired
2004	22	Male	Los Angeles*	Dog, El Salvador
2006	11	Male	San Joaquin*	Dog, Philippines
2008	16	Male	Santa Barbara*	Bat, Mexican free-tailed

* Imported cases; exposure occurred outside of the California

Figure 3. Reported rabid bats and skunks in California by location found, 2001 - 2008



through 2008 [Figure 3]. In contrast, rabid skunks were most frequently reported from the Central Coast (32.4 percent of 410), Far North (25.1 percent), and Sacramento Metro (17.8 percent) regions. From the combined years of 2001 through 2004 to the combined years of 2005 through 2008, the regions with the greatest percentage decreases in reported skunk rabies were the Bay Area (88.5 percent decrease from 26 to 3 cases), the Far North (78.8 percent decrease from 85 to 18 cases), and the Central Coast (53.9 percent decrease from 91 to 42 cases) [Figure 3].

Human cases

During the surveillance period, 6 human cases of rabies were reported in California; as expected all died [Table 1]. All were males with a median age of 25 years (range: 11 to 72 years). Race/ethnicities were White, non-Hispanic (2, 33.3 percent), Hispanic (2, 33.3 percent), and Asian-Pacific Islander (2, 33.3 percent). Cases resided in the regions of the Far North (2, 33.3 percent), San Joaquin Valley (1, 16.7 percent), Central Coast (1, 16.7 percent), South Coast (1, 16.7 percent), and San Diego (1, 16.7 percent). Four (66.7 percent) of 6 human rabies cases resulted from exposures that occurred outside of the US. The remaining 2 cases were associated with California bat variants.

California's most recent human case occurred in a 16-year-old male who entered the state illegally from Oaxaca, Mexico and became ill 1 day after his arrival[4]. The patient had a history of a dog and a fox bite. Patient specimens yielded virus closely related to bat rather than dog virus variant. At least 20 persons received postexposure prophylaxis because of exposure to the case.

Comment

Human rabies remained rare in California during the surveillance period. California's 2008 case was the first imported human rabies case in the US that was not associated with a canine rabies virus variant. Although the number of rabid wild animals (especially skunks) reported to CDPH decreased during the surveillance period, it remains unclear whether these decreases represent changes in disease activity, detection, testing, or reporting. Because animal public health surveillance is largely passive, resource limitations may have influenced case detection and reporting.

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

References and resources

[1]Centers for Disease Control and Prevention. Human rabies prevention - - United States, 2008. MMWR 2008;57;1-26,28.
http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5703a1.htm
[2]CDPH rabies information page:
http://ww2.cdph.ca.gov/HealthInfo/discond/Pages/rabies.aspx
[3]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf
[4]Centers for Disease Control and Prevention. Imported human rabies - California, 2008. MMWR 2009;58:713-6.
http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5826a1.htm

Last updated 10/22/2009

Prepared by Kate Cummings, MPH, James Glover, MS, DVM, MPVM, MPH, and Ben Sun, DVM, MPVM, Infectious Diseases Branch

Epidemiologic Summary of Salmonellosis in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 35,885 cases of non-typhoidal salmonellosis with estimated symptom onset dates from 2001 through 2008.
- Salmonellosis incidence rates increased by 9.1 percent from 2001 (12.1 per 100,000) to 2008 (13.2 per 100,000).
- During the surveillance period, 167 (0.5 percent) reported cases died with salmonellosis. Case fatality rates were 6.7 times higher in cases ≥ 65 years of age (2.0 percent) compared to cases < 65 years of age (0.3 percent).
- Average annual incidence rates were higher among children under 1 year of age (63.4 per 100,000) and 1 to 4 years of age (37.4 per 100,000). From 2001 to 2008, incidence rates increased by 52.8 percent among children 1 to 4 years of age (from 35.6 to 54.4 per 100,000).
- From 2001 through 2008, CDPH received reports of 146 (125 confirmed, 21 suspected) outbreaks of foodborne salmonellosis involving 3,422 cases.
- Preventing contamination during the production and processing of human foods, including fresh fruits and vegetables, combined with consumer education 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 1.4 million infections, 16,000 hospitalizations, and 550 deaths each year[1]. Consuming foods directly or indirectly contaminated by infected animals is the leading source of *Salmonella* infections. However, exposure to ill persons or infected animals and their environments (notably turtles and other reptiles and petting zoo or farm animals) may also result in infection. The most frequent *Salmonella* serovars isolated from human cases nationally have included *S*. Enteritidis, *S*. Typhimurium, *S*. Newport, *S*. Heidelberg, and *S*. Javiana. Non-typhoidal *Salmonella* is a commonly identified etiology in foodborne disease outbreaks, though most salmonellosis cases are reported as sporadic. The national *Healthy People 2010* target objective for salmonellosis is no more than 6.8 new cases per 100,000 population.

Acute illness, usually gastroenteritis, occurs after an incubation of 12 to 72 hours, and lasts 4 to 7 days; treatment with antibiotics is not generally recommended. Rarely, complications such as septicemia, arthritis, meningitis, or pneumonia may occur, especially among immunocompromised persons and those in the extremes of age. The recent emergence of *Salmonella* serovars with resistance to first-line antibiotics could limit effective therapeutic options in the future.

We describe here the epidemiology of salmonellosis in California from 2001 through 2008. Data for 2008 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[2].

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. 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. California regulations

require that any illness in which organisms of the genus *Salmonella* (except the typhoid bacillus) have been isolated from feces, blood, urine or pathological material be reported as a *Salmonella* infection. CDPH officially counted such cases including asymptomatic and extraintestinal infections.

Epidemiology of salmonellosis in California

CDPH received reports of 35,885 cases of non-typhoidal salmonellosis with estimated symptom onset dates from 2001 through 2008. Incidence rates increased by 9.1 percent from 2001 (12.1 per 100,000) to 2008 (13.2 per 100,000) [Figure 1]. During the surveillance period, 167 (0.5 percent) cases were reported to have died with salmonellosis during the surveillance period. Case fatality rates were 6.7 times higher in cases ≥ 65 years of age (2.0 percent) compared to cases < 65 years of age (0.3 percent).

Average annual salmonellosis incidence rates for the surveillance period were higher among children under 1 year of age (63.4 per 100,000), 1 to 4 years of age (37.4 per 100,000), and 5 to 14 years of age (12.5 per 100,000) followed by adults 65 years of age or older (11.5 per 100,000). From 2001 to 2008, annual incidence rates increased by 52.8 percent among children 1 to 4 years of age (from 35.6 to 54.4 per 100,000) which was largely driven by an outbreak of *S*. Javiana in Los Angeles County in late 2008. In contrast, incidence rates decreased by 6.9 percent among children under 1 year of age from the combined years of 2001 and 2002 (66.7 per 100,000) to the combined years of 2007 and 2008 (62.1 per 100,000) [Figure 2]. The ratio of male to female cases was 1.0:1.0. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (27.3 percent). However, salmonellosis cases with complete data reported Hispanic ethnicity more frequently than would be expected based on the overall demographic profile of California [Figure 3].

Fifty (86.2 percent) of 58 counties reported average annual salmonellosis incidence rates for the surveillance period that were above the *Health People 2010* objective. Average annual incidence rates for the surveillance period were higher in Northern California (13.5 per 100,000) than Southern California (11.3 per 100,000). However, from 2001 to 2008, Southern California rates increased by 21.8 percent (from 10.1 to 12.3 per 100,000) whereas Northern California rates

Figure 1. California salmonellosis case counts and incidence rates

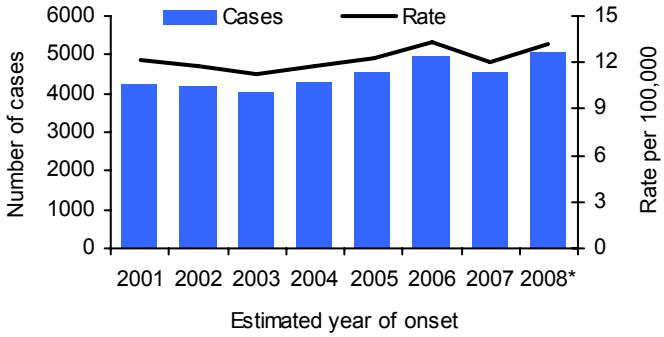


Figure 2. California salmonellosis incidence rates by age and time period

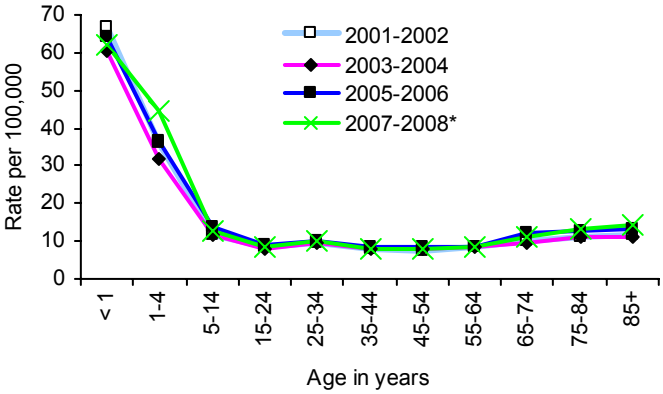
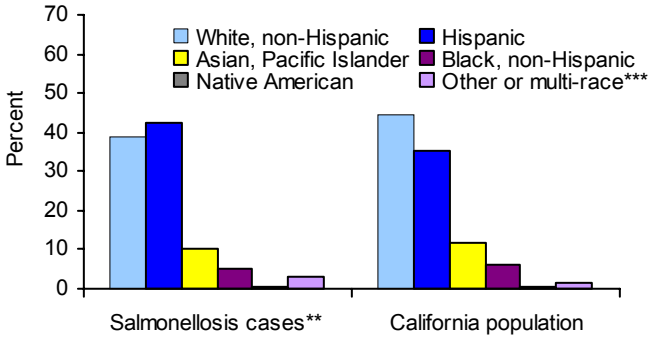


Figure 3. California salmonellosis cases and population by race/ethnicity 2001 - 2008*



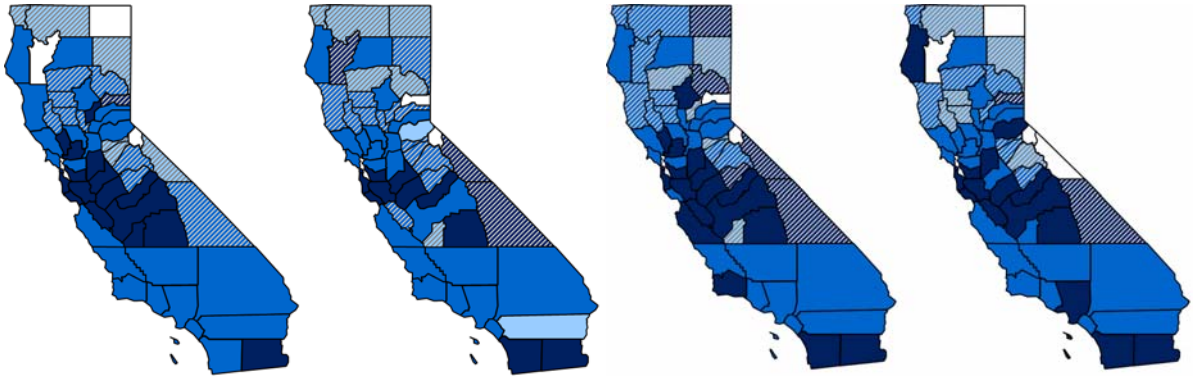
Notes for Figures 1-3

*2008 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 salmonellosis incidence rates



2001-2002 | 2003-2004 | 2005-2006 | 2007-provisional 2008

Cases per 100,000 population

0.0 | 0.1 - 6.8 | 6.9 - 13.6 | 13.7 - 29.0 | Potentially unreliable rate, relative standard error 23 percent or more | Rates represent the average for each time period

decreased by 9.7 percent (from 14.5 to 13.1 per 100,000). County-specific incidence rates for each two-year interval of the surveillance period ranged from 0 to 29.0 per 100,000 persons [Figure 4].

From 2001 through 2008, CDPH received reports of 146 (125 confirmed, 21 suspected) outbreaks of foodborne salmonellosis involving 3,422 cases. The most common serovars reported among outbreaks were *S*. Enteritidis (26), S. Typhimurium (20), S. Heidelberg (15), and S. Newport (12). While the majority of outbreaks involved a single county, 12 (8.2 percent) involved exposures and cases in more than 1 county and an additional 9 (6.2 percent) involved exposures and cases in more than 1 state. Among 56 (38.4 percent) outbreaks with a confirmed vehicle, the most common types of foods implicated were fruits and vegetables (12, 21.4 percent) and poultry (10, 17.9 percent).

Comment

Similar to national data[3], the average annual incidence rate for salmonellosis in California during the surveillance period was nearly twice the national *Health People 2010* target objective (6.8 per 100,000). And, as reported nationally[3], the declines in salmonellosis incidence rates in California that occurred from 1996 to 1999 (from 20 to 13 cases per 100,000 persons) appear to have plateaued. Why rates have leveled off is unclear and remains the subject of continued national attention and research.

Preventing contamination during the production and processing of human foods, including fresh fruits and vegetables, combined with consumer education may provide the best opportunities for preventing and controlling salmonellosis.

References and resources

[1]Mead PS, Slutsker L, Dietz V et al. Food-related illness and death in the United States. Emerg Infect Dis 1999;5:607-25.
http://www.cdc.gov/ncidod/eid/Vol5no5/pdf/mead.pdf

[2]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

[3]Centers for Disease Control and Prevention. Preliminary FoodNet data on the incidence of infection with pathogens transmitted commonly through food --- 10 states, 2008. MMWR 2009:58 (13);333-7.
http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5813a2.htm

California Department of Public Health salmonellosis information web page
http://www.cdph.ca.gov/HealthInfo/discond/Pages/Salmonellosis.aspx

Last updated 12/16/2009

Prepared by Kate Cummings, MPH, Jeff Higa, MPH, and Akiko Kimura, MD, Infectious Diseases Branch

Epidemiologic Summary of Shiga toxin-producing *Escherichia coli (STEC)*-related infections and illnesses in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 2,067 cases of *E. coli* O157:H7 infection with estimated onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 0.7 cases per 100,000 Californians.
- Average annual *E. coli* O157:H7 incidence rates for the surveillance period were higher among children 1 to 4 years of age (3.30 per 100,000), and 5 to 14 years of age (1.29 per 100,000). During the surveillance period, 179 *E. coli* O157:H7 infections progressed to hemolytic uremic syndrome (HUS) and 10 cases died with their infections.
- CDPH received reports of 336 cases of HUS with estimated symptom onset dates from 2001 through 2008. This corresponds to an average annual HUS incidence rate of 0.11 cases per 100,000 Californians.
- Average annual HUS incidence rates were higher among children 1 to 4 years of age (1.04 per 100,000), and 5 to 14 years of age (0.21 per 100,000). During the surveillance period, 11 cases were reported to have died with HUS and 179 cases were accompanied by a laboratory-confirmed *E. coli* O157:H7 infection.
- From 2001 through 2008, CDPH received reports of 22 confirmed and 2 suspected foodborne outbreaks of STEC in California involving 501 case-patients.
- Despite advances in food safety, STEC infections have remained level in California.

Background

Shiga toxin-producing *Escherichia coli* (STEC) are important enteric bacterial pathogens in the United States (US), causing an estimated 110,000 infections, 3300 hospitalizations, and 91 deaths each year[1]. These diarrhea-causing *E. coli* are named for the potent cytotoxins (Shiga toxins 1 and 2) they produce. Among the many STEC serotypes, *E. coli* O157:H7 is the most frequently reported. Handling or consuming food contaminated by infected animals, especially cattle, are the leading sources of STEC infections. Direct exposure to infected persons or infected animals and their environments can also result in infection. The national *Healthy People 2010* target objective for *E. coli* O157:H7 is fewer than 1 new case per 100,000 population.

Acute illness, usually gastroenteritis, occurs after an incubation period of 3 to 4 days. About 8 percent of infections progress to hemolytic uremic syndrome (HUS). HUS is a delayed, life-threatening complication and is a leading cause of acute renal failure in US children. Approximately 85 percent of childhood HUS is preceded by an STEC infection. The fraction of HUS cases attributable to an antecedent STEC infection is large and onset of HUS may be delayed until after the STEC infection has cleared. Therefore, for surveillance purposes, post-diarrheal HUS cases without laboratory evidence of an STEC infection are presumed to be related to an undetected STEC infection.

We describe here the epidemiology of *E. coli* O157:H7 and HUS in California from 2001 through 2008. We also describe the numbers of cases in California in 2008 that were infections of non-O157:H7 STEC serogroups or were Shiga toxin fecal screening test positive with no further laboratory confirmation. Data for 2008 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[2].

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:H7 and HUS to their local health department immediately by telephone. Laboratories are also required to notify the local health

department within one working day after the health care provider has been notified that laboratory testing yielded evidence suggestive of *E. coli* O157:H7.

California regulations were expanded in late 2006 to require reporting of non-O157 STEC infections and cases in which Shiga toxin was detected in feces without further culture confirmation or serogroup identification. This latter requirement, which was considered fully implemented in 2008, was added because some commercial laboratories now test for Shiga toxin without subsequently confirming identification by culture or other means.

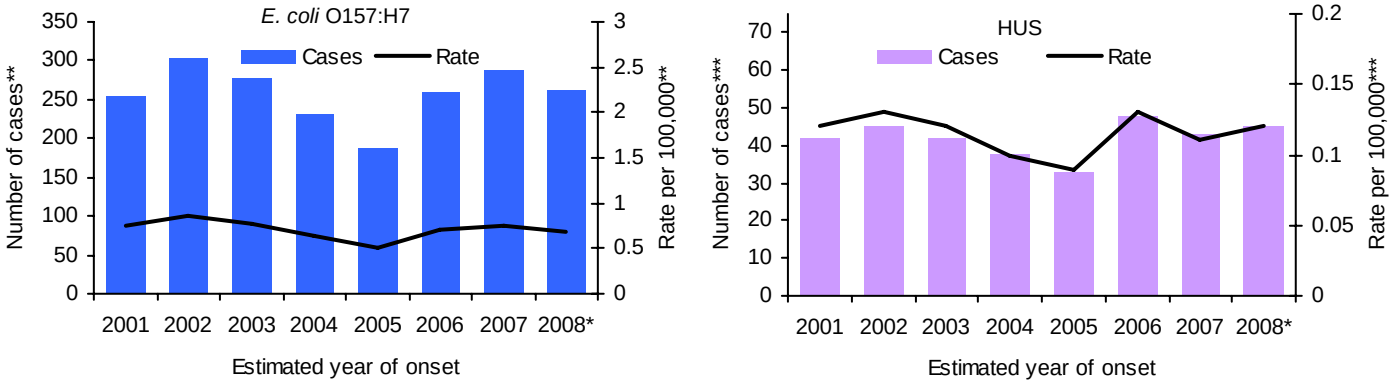
California regulations require local health officers to report to CDPH cases of STEC-related infections. California officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC) surveillance case definition with minor modifications. CDC defined a confirmed case as one with isolation of STEC from a clinical specimen. CDC assumed serotype O157:H7 isolates were Shiga toxin-producing. For all other serotypes, CDC required evidence of toxin production or the presence of Shiga toxin genes. Although CDC also included a probable classification for case reporting, in practice, CDPH only counted confirmed cases. Although not included in the CDC case definition, CDPH began counting cases with Shiga toxin present in stool (in the absence of culture confirmation) to comply with regulatory changes in California. The CDC defined a confirmed case of HUS as 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 one that met all criteria for a confirmed case but did not have confirmed microangiopathic changes. CDPH counted both confirmed and probable HUS cases.

Epidemiology of E. coli O157:H7

CDPH received reports of 2,067 cases of *E. coli* O157:H7 infection with estimated onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 0.71 cases per 100,000 Californians. Incidence rates remained stable during the surveillance period (range: 0.51 to 0.86 per 100,000) [Figure 1]. A total of 179 infections progressed to HUS (8.7 percent) at the time of report [Figure 2] and 10 (0.5 percent) cases died with their infections. *E. coli* O157:H7 average annual incidence rates during the surveillance period were higher among children 1 to 4 years of age (3.30 per 100,000), and 5 to 14 years of age (1.29 per 100,000) [Figure 3]. The ratio of male to female cases was 0.9:1.0. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (22.8 percent). However, *E. coli* O157:H7 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].

Twenty-eight (48.3 percent) of 58 counties had an *E. coli* O157:H7 average annual incidence rate for the surveillance period that was above the national *Healthy People 2010* target [Figure 5]. Average

Figure 1. California *E. coli* O157:H7 and hemolytic uremic syndrome (HUS) case counts and incidence rates



annual incidence rates were 3.2 times higher in Northern California (1.16 per 100,000) than in Southern California (0.36 per 100,000). The Sierra (2.35 per 100,000), San Joaquin Valley (1.31 per 100,000) and Bay Area (1.18 per 100,000) regions reported the highest average incidence rates during the surveillance period.

From 2001 through 2008, CDPH received reports of 24 (22 confirmed, 2 suspected) foodborne outbreaks of STEC involving 501 cases. Only 1 outbreak was suspected to have been caused by a non-O157:H7 STEC. While the majority of outbreaks involved a single county, 6 (25.0 percent) involved exposures and residents in multiple counties and an additional 4 (16.7 percent) involved exposures and residents in multiple states. Among 13 (54.2 percent) confirmed outbreaks with a confirmed vehicle, the most common types of foods implicated were meats (7, 53.9 percent) and fruits and vegetables (4, 30.8 percent). The largest confirmed outbreak occurred in 2007, and involved 124 cases of *E. coli* O157:H7 infection associated with beef tri-tip.

Epidemiology of HUS

CDPH received reports of 336 cases of HUS with estimated symptom onset dates from 2001 through 2008. The average annual HUS incidence rate was 0.11 cases per 100,000 Californians and rates remained stable from 2001 to 2008 (range: 0.09 to 0.13 per 100,000) [Figure 1]. During the surveillance period, 11 (3.3 percent) cases were reported to have died with HUS and 179 (53.3 percent) cases were accompanied by a laboratory-confirmed *E. coli* O157:H7 infection [Figure 2]. Average annual HUS incidence rates were higher among children 1 to 4 years of age (1.04 per 100,000), and 5 to 14 years of age (0.21 per 100,000) [Figure 3]. The ratio of male to female cases was 0.8:1.0. Incidence rates by race/ethnicity were not calculated due to missing data (15.7 percent). However, HUS cases reported White non-Hispanic race/ethnicity more frequently than would be expected based on the demographic profile of California [Figure 4].

Similar to *E. coli* O157:H7 rates, the average annual incidence rate for HUS for the surveillance period was 3.0 times higher in Northern California (0.18 per 100,000) than in Southern California (0.06 per 100,000) [Figure 4]. From 2001 to 2008, incidence rates increased by 17.6 percent in Northern California (from 0.17 to 0.20 per 100,00) but decreased by 33.3 percent in Southern California (from 0.09 to 0.06 per 100,000).

Figure 2. Venn diagram of California *E. coli* O157:H7 and HUS cases 2001 - 2008*

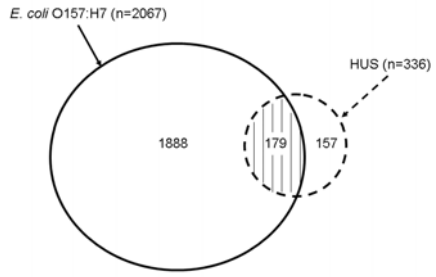


Figure 3. California *E. coli* O157:H7 and HUS incidence rates by age 2001-2008*

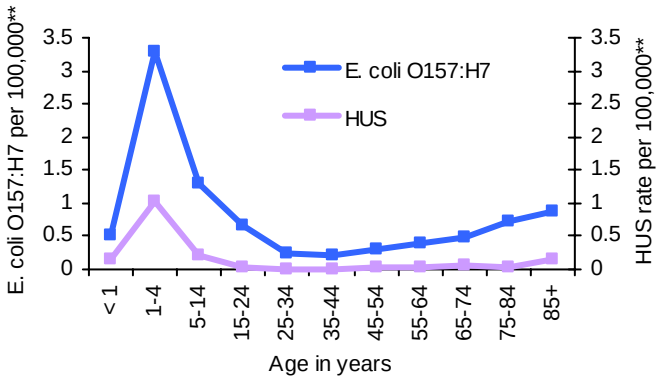
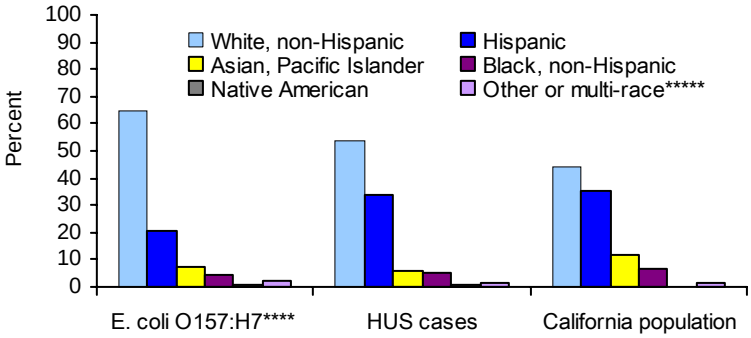


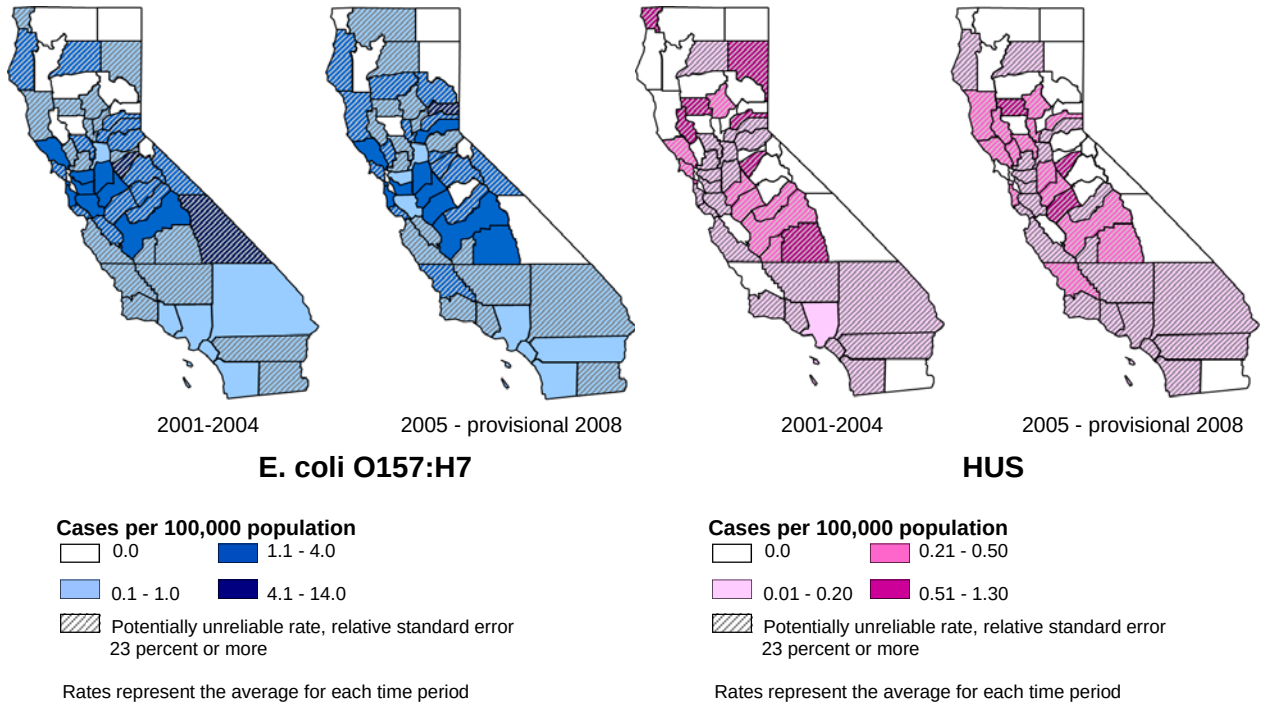
Figure 4. California *E. coli* O157:H7 and HUS cases and population by race/ethnicity 2001-2008*



Notes for Figures 1-5

*2008 data are provisional
** Includes cases accompanied by HUS
***Includes cases with laboratory evidence of STEC
****Unknowns were excluded
*****Includes cases who identified 'other' as their race and Californians ('population') who identified more than one race

Figure 5. California county-specific *E. coli* O157:H7 and HUS incidence rates



Non-O157:H7 STEC cases 2008

CDPH received reports of 350 cases of STEC related infections and illnesses with estimated symptom onset dates in 2008. Of these cases, 36 (10.3 percent) were culture-confirmed as non-O157:H7 serogroups, 35 (10.0 percent) were Shiga toxin fecal screening test positive without further culture confirmation or identification, 263 (75.1 percent) were *E. coli* O157:H7 including 29 cases that were accompanied by HUS, and 16 (4.6 percent) were HUS without further laboratory evidence of STEC infection.

Comment

During the surveillance period, average annual incidence rates of *E. coli* O157:H7 infection and HUS were stable with only modest fluctuations. The statewide average annual incidence rate of *E. coli* O157:H7 infection for the surveillance period was just below the national *Healthy People 2010* target objective of 1 case per 100,000. However, many counties in Northern California reported average incidence rates above that threshold.

Despite advances in food safety, STEC infections have remained level during the surveillance period in California. Preventing contamination during the production and processing of foods, including beef and fresh fruits and vegetables, combined with consumer education may provide the best opportunities for preventing and controlling STEC-related infections and illnesses.

References and resources

[1]Mead PS, Slutsker L, Dietz V et al. Food-related illness and death in the United States. Emerg Infect Dis 1999;5:607-25.
http://www.cdc.gov/ncidod/eid/Vol5no5/pdf/mead.pdf

[2]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

California Department of Public Health information web page
http://www.cdph.ca.gov/HealthInfo/discond/Pages/EscherichiacoliO157H7.aspx

Last updated 9/29/2009

Prepared by Kate Cummings, MPH, Charlotte Wheeler, MD, MPH, and Farzaneh Tabnak, PhD, Infectious Diseases Branch.

Epidemiologic Summary of Shigellosis in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 15,997 cases of shigellosis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 5.5 cases per 100,000 Californians.
- Shigellosis incidence rates decreased by 32.8 percent from 2001 (6.4 per 100,000) to 2008 (4.3 per 100,000).
- During the surveillance period, 30 (0.2 percent) cases were reported to have died with shigellosis. Case fatality rates were 6.5 times higher in cases ≥ 65 years of age (1.3 percent) compared to cases < 65 years of age (0.2 percent).
- Average annual shigellosis incidence rates were higher among children 1 to 4 years of age (21.3 per 100,000), and 5 to 14 years of age (8.8 per 100,000).
- From 2001 through 2008, CDPH received reports of 23 outbreaks (19 confirmed, 4 suspected) of foodborne shigellosis involving 472 cases.
- *S. sonnei* (68.5 percent), and *S. flexneri* (28.5 percent) infections were most common and varied, respectively by median age (12 years vs. 26 years) and by the ratio of male to female cases (1.0:1.0 vs. 1.6:1.0).
- Early diagnosis and reporting of cases, and education on hand hygiene and safe sexual practices are cornerstones of disease control.

Background

Shigella is a commonly reported enteric bacterial pathogen in the United States (US), causing an estimated 500,000 infections, 6,000 hospitalizations, and 70 deaths each year[1]. *Shigella* infection is restricted to humans and is efficiently and predominantly transmitted from person-to-person through direct or indirect fecal-oral contact. Other sources of infection include 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*. *S. sonnei* is predominate in industrialized countries whereas *S. flexneri* is predominate in developing countries. There is no national *Healthy People 2010* target objective for shigellosis.

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. *S. dysenteriae* is associated with life threatening complications including toxic megacolon and hemolytic uremic syndrome. Post-infectious arthritis is a rare, late complication of *S. flexneri* infection. Although most shigellosis appear to be sporadic cases, large outbreaks of *Shigella* have occurred in crowded settings where personal hygiene may be difficult (custodial institutions, day care centers), and in association with contaminated food or water. Other persons at increased risk of infection include men who have sex with men, persons with human immunodeficiency virus (HIV) infection[2], and international travelers. Increasing resistance to antimicrobial agents has been noted among nationally-reported infections acquired domestically and abroad.

We describe here the epidemiology of shigellosis in California from 2001 through 2008. Data for 2008 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[3].

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 supsected. Clinical and

reference laboratories must also notify the local health department when laboratory testing yields evidence suggestive of *Shigella*; notification 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 shigellosis. CDPH officially counted cases that satisfied the Centers for Disease Control and Prevention (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 extraintestinal infections. A probable case was one with clinically compatible illness and an established epidemiologic link to a laboratory-confirmed case.

Epidemiology of shigellosis in California

CDPH received reports of 15,997 cases of shigellosis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 5.5 cases per 100,000 Californians. Incidence rates decreased by 32.8 percent from 2001 (6.4 per 100,000) to 2008 (4.3 per 100,000) ($p < 0.001$) [Figure 1]. During the surveillance period, 30 (0.2 percent) cases were reported to have died with shigellosis during the surveillance period. Case fatality rates were 6.5 times higher in cases ≥ 65 years of age (1.3 percent) compared to cases < 65 years of age (0.2 percent).

Average annual shigellosis incidence rates for the surveillance period were higher among children 1 to 4 years of age (21.3 per 100,000) and 5 to 14 years of age (8.8 per 100,000) followed by adults 25 to 44 years of age (4.8 per 100,000). Average incidence rates associated with these same age groups demonstrated the greatest decreases from the combined years of 2001 and 2002 to the combined years of 2007 and 2008 (42.9 percent, 44.1 percent, and 50.8 percent, respectively) [Figure 2]. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (19.5 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].

Average annual incidence rates for the surveillance period were 11.8 percent higher in Southern

Figure 1. California shigellosis case counts and incidence rates

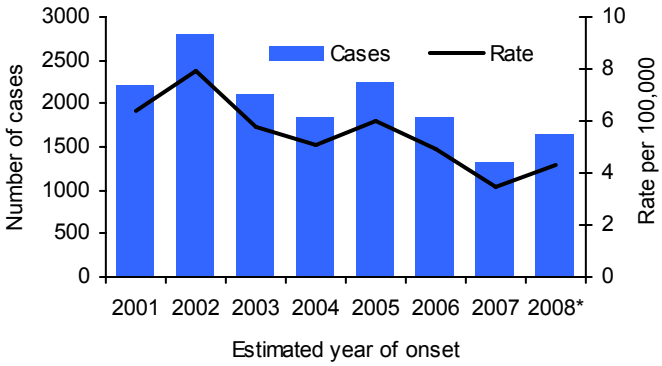


Figure 2. California shigellosis incidence rates by age and time period

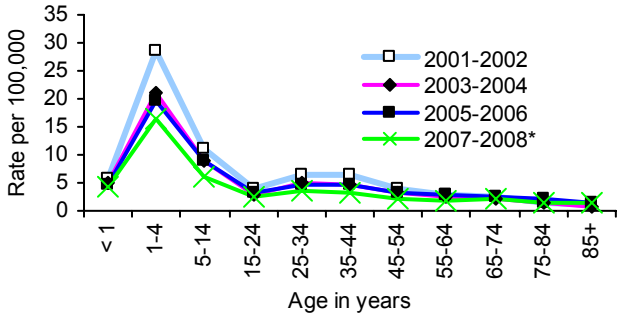
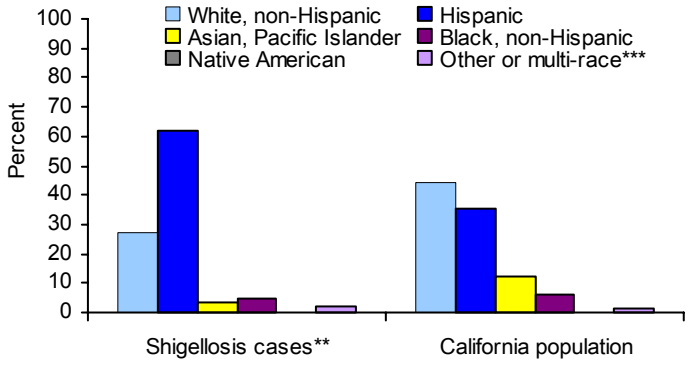


Figure 3. California shigellosis cases and population by race/ethnicity 2001 - 2008*



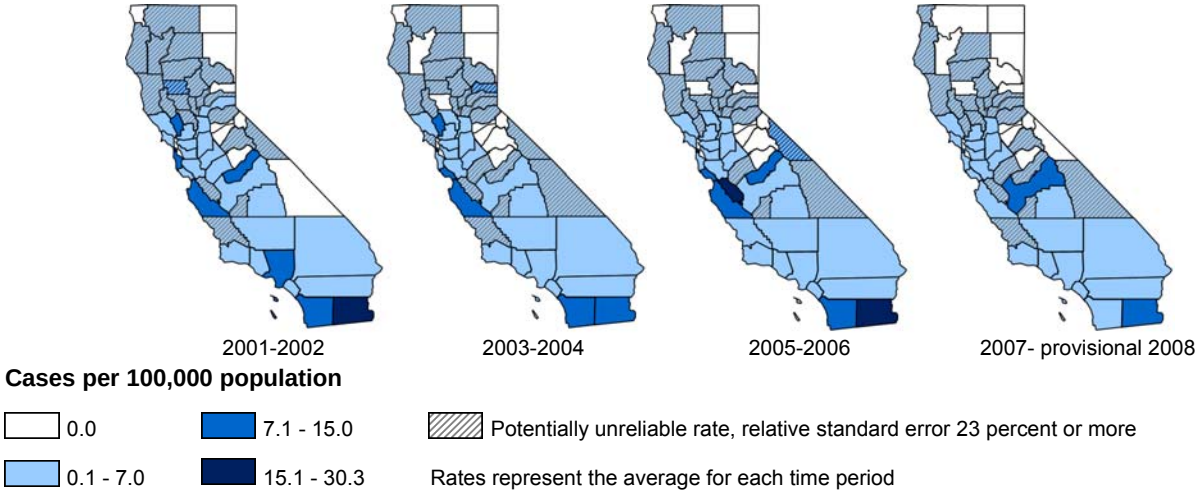
Notes for Figures 1-3

*2008 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



California (5.7 per 100,000) than in Northern California (5.1 per 100,000). County-specific incidence rates for each two-year interval of the report period ranged from 0 to 30.3 per 100,000 residents [Figure 4]. The highest rates occurred in San Francisco (30.3 per 100,000) and Imperial (28.0 per 100,000) counties during the years 2001 and 2002. From 2000 to 2001, San Francisco experienced a large, sustained community-based outbreak of *S. sonnei* outbreak among men who have sex with men[4].

From 2001 through 2008, CDPH received reports of 23 outbreaks (19 confirmed, 4 suspected) of foodborne shigellosis involving 472 cases.

From 2001 through 2008, 13,484 (84.3 percent) cases had a *Shigella* isolate with the species identified and reported. Among these, *S. sonnei* (9,237, 68.5 percent), and *S. flexneri* (3,836, 28.4 percent) infections were the most common species reported. *S. sonnei* cases tended to be younger (median age 12 years) and infections were equally distributed among both sexes (male to female ratio: 1.0:1.0). *S. flexneri* cases tended to be adult (median age 26 years) and male (male to female ratio: 1.6:1.0). By comparison, in the US, *S. sonnei* (71.7 percent), and *S. flexneri* (18.4 percent) were also the most common species identified and reported but differed by proportion[5].

Comment

From 2001 to 2008, there was a significant decrease in shigellosis cases in California although the rate of decline was not consistent from year to year. *S. sonnei* and *S. flexneri were* the most frequently identified species but were associated with different epidemiologic characteristics.

Public health measures such as early diagnosis and reporting of cases, education on hand hygiene and safe sexual practices, and targeted education for high-risk groups likely offer the best opportunities for reducing disease transmission.

References and additional resources

[1]Mead PS, Slutsker L, Dietz V et al. Food-related illness and death in the United States. Emerg Infect Dis 1999;5:607-25.
http://www.cdc.gov/ncidod/eid/Vol5no5/pdf/mead.pdf
[2]Aragón TJ, Vugia DJ, Shallow S, Samuel MC, et al. Case-control study of shigellosis in San Francisco: the role of sexual transmission and HIV infection. Clin Infect Dis 2007;44:327-34.
[3]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf
[4]Centers for Disease Control and Prevention. *Shigella* sonnei outbreak among men who have sex with men -- San Francisco, California, 2000-2001. Morb Mort Week Rep 2001;50(42):922-6.
[5]Gupta A, Polyak CS, Bishop RD, et al. Laboratory-confirmed shigellosis in the US, 1989-2002: Epidemiologic trends and patterns. Clin Infect Dis 2004;38:1372-7.

California Department of Public Health shigellosis information web page
http://www.cdph.ca.gov/HealthInfo/discond/Pages/Shigellosis.aspx

Last updated 9/29/2009

Prepared by Kate Cummings, MPH, and Akiko Kimura, MD, Infectious Diseases Branch

Epidemiologic Summary of Human Tularemia in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 16 cases of tularemia with estimated onset dates from 2001 through 2008.
- No cases were reported to have died with tularemia.
- The highest number of cases was reported among persons 5 to 14 years of age.
- The ratio of male to female cases was 1.3:1.0.
- Eleven (68.7 percent) cases had estimated onset months from May through August.
- Cases were predominantly reported from Northern California (12, 75.0 percent).
- Avoiding exposure to bites by ticks and blood-feeding flies (by insect repellent and by closely examining clothes and skin for ticks), and avoiding direct contact with wild animal tissues may provide the best opportunities for preventing and controlling human tularemia.

Background

Francisella tularensis is a rare but important bacterial zoonotic pathogen in the United States (US); between 100 and 200 incident cases of human tularemia are reported annually[1]. *F. tularensis* is divided into 4 subspecies, 2 of which occur in California. *F. tularensis* subsp. *tularensis* (Jellison type A) is highly virulent, has a case fatality rate of 5 to 15 percent, and is found only in North America. *F. tularensis* subsp. *holarctica* (Jellison type B), is less virulent, is infrequently fatal, and is found throughout the Northern Hemisphere. Because of its longevity in the environment, low inoculum dose (10 to 50 organisms), and multiple routes of transmission including inhalation of aerosol, *F. tularensis* is listed among the Centers for Disease Control and Prevention (CDC) category A bioterrorism agents.

Bites from infected arthropods (ticks or flies) and handling contaminated animal tissues are the leading means of human exposure. Consuming or direct contact with contaminated water, food, or soil, or inhaling bioaerosols can also result in human infection. Person-to-person transmission has not been documented.

Human tularemia can present with diverse clinical symptoms depending on the route of exposure, inoculum size, and infecting subspecies. The usual incubation period is 3 to 5 days. Primary disease presentations can include ulceroglandular, glandular, oculoglandular, oropharyngeal, pneumonic, typhoidal or septic forms. Infection can spread to multiple organ systems, including the lungs, liver, spleen, kidney, and lymphatic system.

We describe here the epidemiology of human tularemia in California from 2001 through 2008. Data for 2008 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[2]. Because of the small numbers of cases, incidence rates were not calculated.

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report suspected cases of human tularemia 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 human tularemia is received. Laboratories must report to the local health department when laboratory testing yields evidence suggestive of *F. tularensis*; notification must occur within one hour after the health care provider has been notified.

California regulations also require local health officers to report to CDPH cases of human tularensis immediately by telephone. CDPH officially counted cases that satisfied the CDC surveillance case definition. CDC defined a confirmed case as one with clinically compatible illness and isolation of *F. tularensis* in a clinical specimen or fourfold or greater change in serum antibody titer to *F. tularensis* antigen. A probable case was one with clinically compatible illness and (i) elevated serum antibody titer(s) to *F. tularensis* antigen (without documented fourfold or greater change) in a patient with no history of tularemia vaccination or (ii) detection of *F. tularensis* in a clinical specimen by fluorescent assay. Clinically compatible illnesses included ulceroglandular, glandular, oculoglandular, oropharyngeal, intestinal (pain, vomiting, diarrhea), pneumonic, or typhoidal

(febrile illness without early localizing signs and syndromes) presentations.

Epidemiology of human tularemia in California

CDPH received reports of 16 cases of tularemia with estimated onset dates from 2001 through 2008. Case counts rose from 2001 (1) to 2005 (4) and then decreased to 2008 (2) [Figure 1]. No cases were reported to have died with tularemia.

For the surveillance period, the highest number of cases was reported among persons 5 to 14 years of age [Figure 2]. The ratio of male to female cases was 1.3:1.0. Eleven (68.7 percent) cases had estimated onset months from May through August. Cases were reported from 10 counties including the counties of Alameda (4), Contra Costa (2), Los Angeles (2), Marin (1), Mendocino (2), Nevada (1), Sacramento (1), San Diego (1), Sonoma (1), and Ventura (1).

Commonly reported symptoms included fever (12), lymphadenopathy including cervical (5), axillary (2), submandibular (1), or unspecified (1), and wounds or ulcers on the arm/hand (5) or leg (1). *F. tularensis* was detected in 12 cases by culture (7), polymerase chain reaction (2), or direct fluorescent antibody (3). Specimens were blood (3), lymph node (5), or swab of cutaneous lesion (4). Of 7 isolates available for subtyping, 1 was biovar A and 6 were biovar B. Likely sources of infection were arthropod (tick or deer fly) bite (5), contact with rabbit or coyote tissues (3), and ingestion of nonpotable water (1); circumstances of exposure could not be determined for 7 cases. Three cases were likely exposed outside California in Utah (2) or Nevada (1).

Comment

During the surveillance period, tularemia remained a rare human infection in California. Human cases occurred more frequently during the spring and summer months. Two of 3 cases clearly associated with tick bites (a 5 year-old male from Alameda County and 6 year-old female from Marin County) were bitten at the same grounds in San Mateo County, one in 2004 and one in 2006. *F. tularensis* biovar Type B was detected in ticks field-collected from the grounds in 2006 and in a tick removed from the case in 2004.

Six cases had cutaneous lesions or ulcers from which *F. tularensis* was recovered, suggesting that the route of exposure was through a break in the skin. For some of these cases, an arthropod bite or direct contact with a mammal carcass were the apparent sources of infection, but others had no identifiable contact with infectious material. For example, 1 case with a wound on his finger reported that during his exposure period he had worked repairing fences on a ranch where cattle, pigs, and rabbits were present but he had no direct contact with them.

Figure 1. California human tularemia case counts

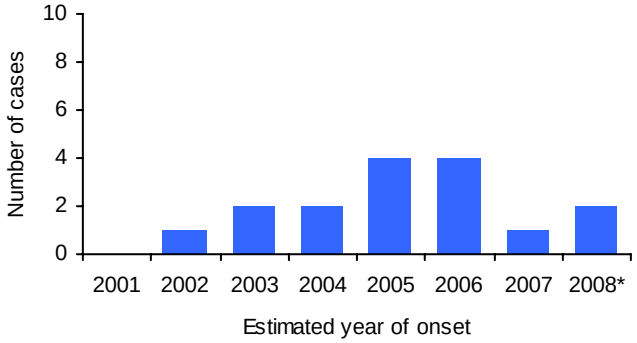
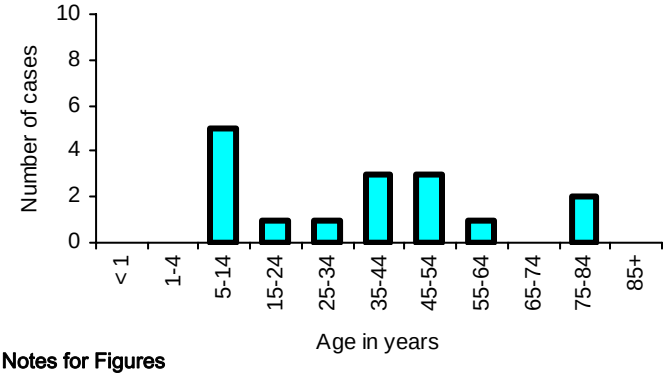


Figure 2. California human tularemia cases by age 2001-2008*



Notes for Figures

*2008 data are provisional

Avoiding exposure to bites by ticks and blood-feeding flies (by using insect repellent and by examining clothes and skin for ticks), and avoiding direct contact with wild animal tissues may provide the best the best opportunities for preventing and controlling human tularemia. Thoroughly washing injuries to the skin and covering new and existing wounds may help to reduce the risk of contamination with bacteria present in the environment.

References and resources

[1]Centers for Disease Control and Prevention. Tularemia - Missouri, 2000-2007. MMWR 2009;58:744-748.

[2]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

CDPH tularemia information website
http://www.cdph.ca.gov/HealthInfo/discond/Pages/Tularemia.aspx

Last updated 10/22/2009

Prepared by Kate Cummings, MPH, James Glover, MS, DVM, MPVM, MPH, Ben Sun, DVM, MPVM, and the Vector-borne Disease Section, Infectious Diseases Branch

Epidemiologic Summary of Typhoid Fever in California, 2001 - 2008

Key Findings and Public Health Messages

- California Department of Public Health (CDPH) received reports of 603 cases of typhoid fever with estimated symptom onset dates from 2001 through 2008. This corresponds to an average incidence rate of 0.21 cases per 100,000 Californians.
- Typhoid fever incidence rates decreased by 12.5 percent from 2001 (0.24 per 100,000) to 2008 (0.21 per 100,000).
- During the surveillance period, 2 (0.3 percent) cases died with typhoid fever.
- Average typhoid fever incidence rates during the surveillance period were highest among children 1 to 4 years of age (0.38 per 100,000) and adults 25 to 34 years of age (0.34 per 100,000).
- From 2001 through 2008, 43 persons were reported as chronic typhoid carriers. One (2.3 percent) carrier died. Chronic carriers were more likely than acute typhoid fever cases to report older age (median age 47 years vs. 26 years) and Hispanic race/ethnicity (48.8 percent vs. 19.9 percent).
- For international travelers, a typhoid vaccine and care in selecting foods and drinks are important prevention measures.

Background

Salmonella Typhi is an uncommon but important enteric and systemic bacterial pathogen in the United States (US), causing an estimated 400 cases per year. While uncommon in the US, typhoid fever is highly endemic in developing countries in Africa, Asia (especially Southeast Asia and the Indian subcontinent), and Central and South America. Most cases in the US are travelers returning from endemic areas. *S.* Typhi infection is restricted to humans, and food or water contaminated by the feces or urine of typhoid fever cases or carriers are the leading sources of exposure. There is no national *Healthy People 2010* target objective for typhoid fever.

Acute illness, usually gastroenteritis, occurs after an incubation that varies from 3 to over 60 days depending on size of the inoculum and host factors. Onset is often insidious. A carrier state may follow acute illness or mild or even subclinical infections. About 1.0 to 4.0 percent of untreated cases will become carriers and the chronic carrier state is more common among persons infected during middle age.

S. Typhi resistant to first-line drugs became so common by the 1990s, that fluoroquinolones became the drugs of choice for treatment. However, nalidixic acid-resistant *S*. Typhi, with decreased susceptibility to fluoroquinolones, and fluoroquinolone-resistance have now been reported in South and Southeast Asia. Two typhoid vaccines are currently available in the US. Both vaccines confer about 70.0 percent protection in older children and adults but neither is licensed for use in young children. Notably, vaccine-induced immunity provides little protection against large challenge doses.

We describe here the epidemiology of typhoid fever in California from 2001 through 2008. Data for 2008 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[1].

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers to report suspected cases of typhoid fever and carriers of *S*. Typhi to their local health department within one working day of identification or immediately by telephone if an outbreak is suspected. Laboratories must also notify the local health department when laboratory testing yields evidence suggestive of *S.* Typhi; notification

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 typhoid fever and carriers of *S.* Typhi. CDPH officially counted typhoid fever cases that satisfied the Centers for Disease Control and Prevention (CDC) surveillance case definition including confirmed and probable classifications. During the surveillance period, CDC defined a confirmed typhoid fever case as one with *S.* Typhi isolated from a clinical specimen. A probable case was one with clinically compatible illness and an established epidemiologic link to a laboratory-confirmed case during an outbreak. CDPH defined a convalescent typhoid carrier as a person who harbored typhoid bacilli for three or more months after onset of typhoid fever. A chronic carrier was: (a) a person who continued to excrete typhoid bacilli for more than 12 months after onset of typhoid fever or (b) (i) a person who gave no history of typhoid fever or who had the disease more than one year previously, and (ii) whose feces or urine were found to contain typhoid bacilli on two separate examinations at least 48 hours apart, confirmed by the CDPH Microbial Diseases Laboratory. CDPH defined other typhoid carriers as persons who had typhoid bacilli isolated from surgically removed tissues, organs, or draining lesions. If such persons continued to excrete typhoid bacilli for more than 12 months, he/she was a chronic typhoid carrier.

Epidemiology of typhoid fever in California

CDPH received reports of 603 cases of typhoid fever with estimated symptom onset dates from 2001 through 2008. This corresponds to an average incidence rate of 0.21 cases per 100,000 Californians. Typhoid fever incidence rates decreased by 12.5 percent from 2001 (0.24 per 100,000) to 2008 (0.21 per 100,000). [Figure 1]. During the surveillance period, 2 (0.3 percent) cases were reported to have died with typhoid fever.

Average typhoid fever incidence rates during the surveillance period were highest among children 1 to 4 years of age (0.38 per 100,000) and adults 25 to 34 years of age (0.34 per 100,000) [Figure 2]. The ratio of male to female cases was 1.0:1.0. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (31.3 percent). However, typhoid fever cases with complete information reported Asian race

Figure 1. California typhoid fever case counts and incidence rates

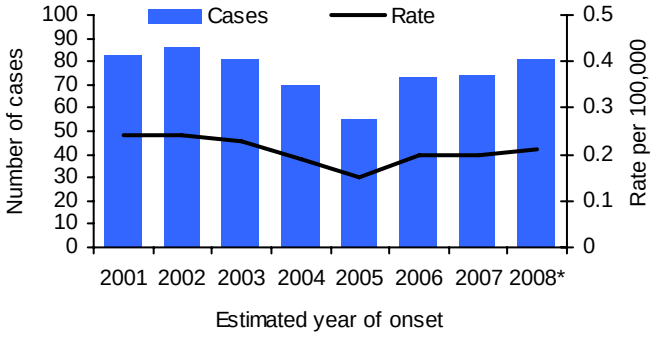


Figure 2. California typhoid fever incidence rates by age and time period

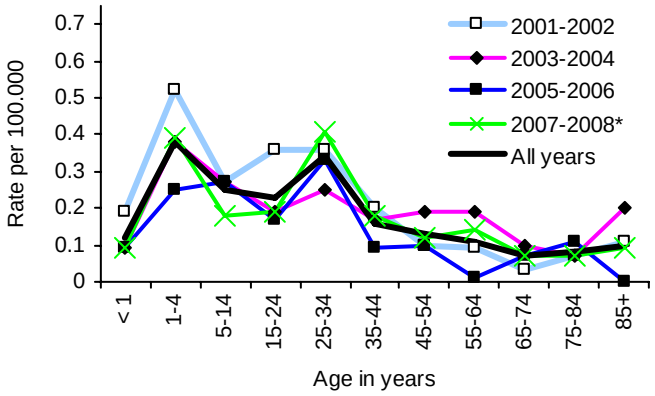
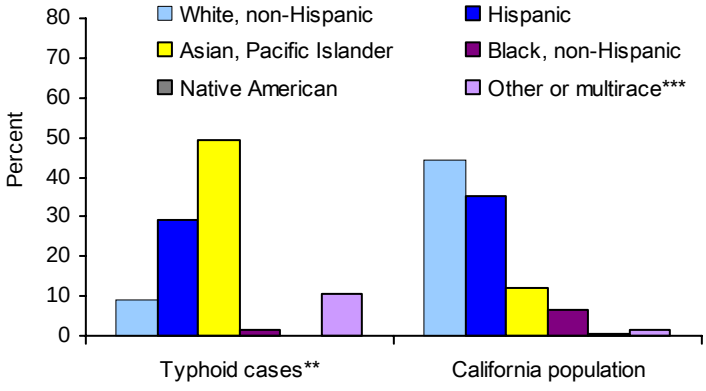


Figure 3. California typhoid fever cases and population by race/ethnicity 2001 - 2008*



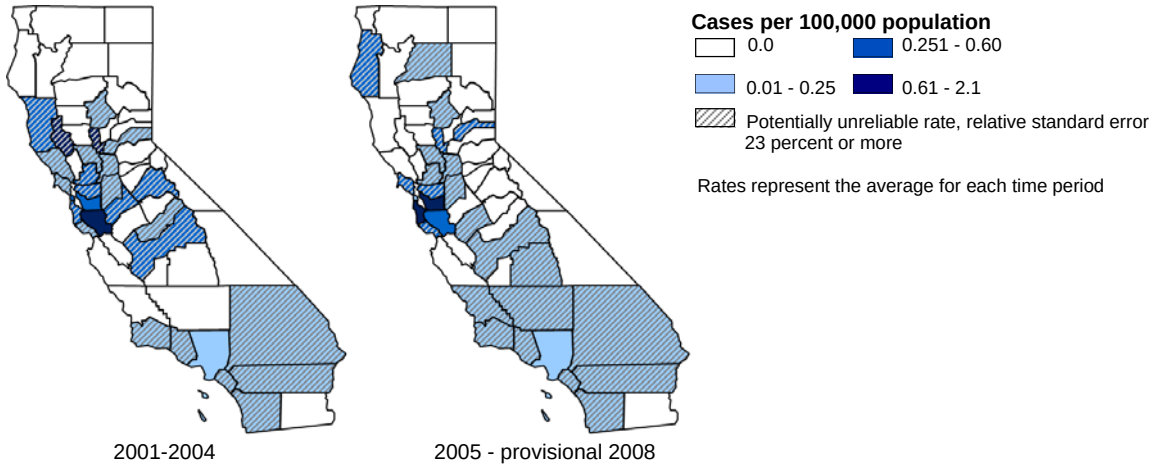
Legend for Figures 1-3

*2008 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 typhoid fever incidence rates



much more frequently than would be expected based on the overall demographic profile of California [Figure 3].

Although the rate of typhoid fever decreased modestly from 2001 to 2008 for the state as a whole, some counties of California reported increased rates. The average incidence rate in the Sacramento Metro region for the combined years of 2005 through 2008 (0.13 per 100,00) was 1.6 times higher than during the combined years of 2001 through 2004 (0.08 per 100,00) [Figure 4]. Of note, many of the counties in this region have small populations and the rates are somewhat unstable.

From 2001 through 2008, 43 Californians were newly reported typhoid carriers. One (2.3 percent) carrier died. Typhoid carriers were more likely than acute typhoid fever cases to report older age (median age: 47 years vs. 26 years), and Hispanic race/ethnicity (48.8 percent vs. 19.9 percent).

Comment

On average, 75 cases of typhoid fever were reported annually in California from 2001 through 2008. These cases occurred predominantly among international travelers. Although uncommon in California and the US, two recent typhoid fever outbreaks illustrate the potential threat to public health associated with this disease. The first outbreak occurred in Florida in 1998, involved at least 16 cases, and was epidemiologically-linked to consumption of drinks made with frozen mamey prepared in Guatemala and Honduras. The second occurred in Nepal in 2002, involved 5,963 multidrug-resistant cases, and was traced to the city's water supply. These outbreaks illustrate that *S*. Typhi can pose an outbreak threat here in the US and that drinking-water associated outbreaks can be very large.

Because of their public health importance, typhoid cases, contacts, and carriers in California are subject to special restrictions and public health supervision. California maintains a registry of all typhoid carriers. Convalescent carriers may be released from supervision by authority of the local health officer while chronic carriers can be released from local supervision only by authority of CDPH.

The recent global emergence of *S*. Paratyphi A, especially in southeast China, may have important implications for enteric fever control worldwide. *S*. Paratyphi A and *S.* Typhi are clinically indistinguishable. While treatment strategies are similar for both, *S.* Parapyphi is not included in currently licensed vaccines and therefore not part of this critical prevention strategy.

For international travelers, while a typhoid vaccine confers some degree of protection, ensuring the safety of food and water is still the most important protective measure.

Resources and References

[1]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

Last updated 8/17/2009

Prepared by Kate Cummings, MPH, and Duc J Vugia, MD, MPH, Infectious Diseases Branch

Epidemiologic Summary of Non-Cholera Vibriosis in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 828 cases of non-cholera vibriosis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average incidence rate of 0.28 cases per 100,000 Californians.
- Although non-cholera vibriosis incidence rates increased from 2001 (0.22 per 100,000) to 2008 (0.28 per 100,000), the highest annual rates were reported in 2004 (0.40 per 100,000) and 2006 (0.47 per 100,000).
- During the surveillance period, the highest average incidence rate of non-cholera vibriosis occurred among adults 55 to 64 years of age (0.46 per 100,000). Average incidence rates were 2.3 times higher in men (0.39 per 100,000) compared to women (0.17 per 100,000).
- The highest reported average incidence rates for the surveillance period were reported by the San Francisco Bay Area (0.56 per 100,000), San Diego (0.53 per 100,000), and Sacramento Metro (0.34 per 100,000) regions.
- From 2001 through 2008, CDPH received reports of 6 confirmed and 6 suspected outbreaks of foodborne non-cholera vibriosis involving a total of 93 cases. Consumption of raw or undercooked oysters and mussels was the most frequently implicated exposure.
- Ensuring that shellfish beds are routinely monitored, seafood products are handled safely during and after harvest, and that consumers are educated about the risks of consuming raw or undercooked seafood may provide the best opportunities for reducing non-cholera vibriosis.

Background

Non-*cholera Vibrio* species are uncommon but important enteric bacterial pathogens, causing an estimated 8,000 infections, 185 hospitalizations, and 57 deaths in the United States (US) each year[1]. *Vibrio* species are natural inhabitants of marine coastal and estuarine environments, and their populations increase dramatically during the warm summer months. In the US, *V. parahaemolyticus* is the most commonly reported *Vibrio* infection, but *V. vulnificus* is associated with severe morbidity and mortality. Consuming raw, undercooked, or cross-contaminated seafood, especially shellfish, is the most common cause of non-cholera vibriosis, but exposing wounds to contaminated warm seawater can also cause skin or soft tissue *Vibrio* infection. There is no national *Healthy People 2010* target objective for non-cholera vibriosis.

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. In contrast, *V. vulnificus* causes septicemia in persons with immunocompromising conditions, chronic liver disease, and alcoholism. Fifty percent of such patients with septicemia die, and the case-fatality rate exceeds 90% among patients who become hypotensive.

We describe here the epidemiology of non-cholera vibriosis in California from 2001 through 2008. Data for 2008 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[2].

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 notify the local health department when laboratory testing yields evidence suggestive of *Vibrio* species; notification should 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 non-cholera vibriosis. CDPH officially counted cases that satisfied the current

Centers for Disease Control and Prevention (CDC) surveillance case definition, including both confirmed and probable classifications. During the surveillance period, CDC defined a confirmed case 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 symptomatic illness and an epidemiologic link to a confirmed case.

Epidemiology of non-cholera vibriosis in California

CDPH received reports of 828 cases of non-cholera vibriosis with estimated symptom onset dates from 2001 through 2008. This corresponds to an average incidence rate of 0.28 cases per 100,000 Californians. Non-cholera vibriosis incidence rates increased from 2001 (0.22 per 100,000) to 2008 (0.28 per 100,000) although the highest annual rates occurred in 2004 (0.40 per 100,000) and 2006 (0.47 per 100,000) [Figure 1]. During the surveillance period, most reported cases were *V. parahaemolyticus*. Only 2 to 5 cases of *V. vulnificus* were reported each year except in 2001, when 13 cases of *V. vulnificus* were reported. During the surveillance period, 14 (1.7 percent of all) cases were reported to have died with non-cholera vibriosis. Among these 14 cases, 7 (50.0 percent) were *V. vulnificus*, 4 (28.6 percent) were *V. cholerae* non-01 or non-0139, and 3 (21.4 percent) were *V. parahaemolyticus*.

The average non-cholera vibriosis incidence rate during the surveillance period was highest among adults 55 to 64 years of age (0.46 per 100,000) [Figure 2]. The ratio of male to female cases was 2.3:1 and average incidence rates were 2.3 times higher in men (0.39 per 100,000) compared to women (0.17 per 100,000). Incidence rates by race/ethnicity were not calculated due to the substantial portion (28.3 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].

Average incidence rates for the surveillance period were 1.6 times higher in Northern California (0.36 per 100,000) than in Southern California (0.22 per 100,000). However, from 2001 to 2008, rates for Southern California increased by 71.4 percent (from 0.14 to 0.24 per 100,000) whereas rates for Northern California increased by 29.2 percent (from

Figure 1. California non-cholera vibriosis case counts and incidence rates

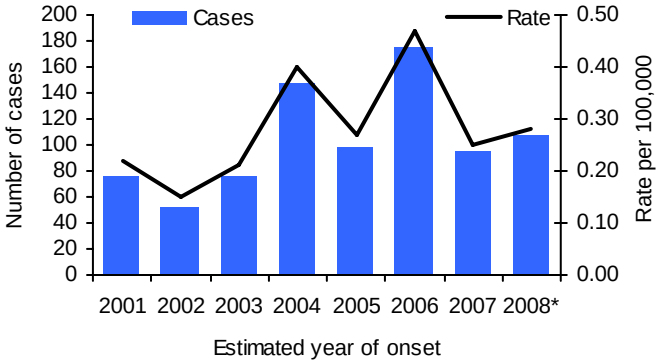


Figure 2. California non-cholera vibriosis by incidence rates by age and time period

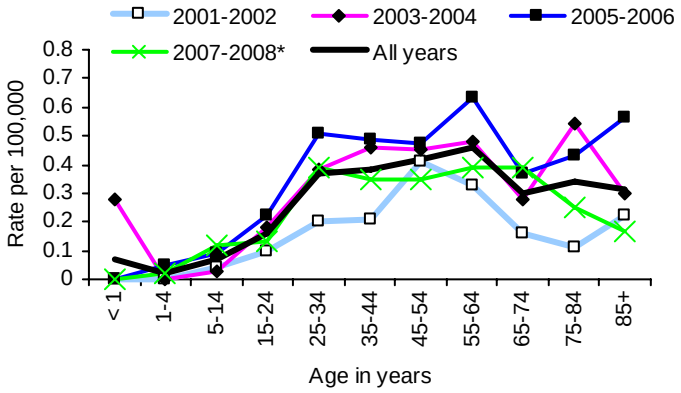
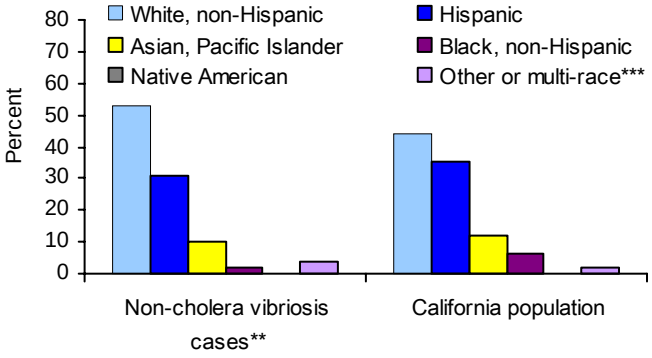


Figure 3. California non-cholera vibriosis cases and population by race/ethnicity 2001 - 2008*



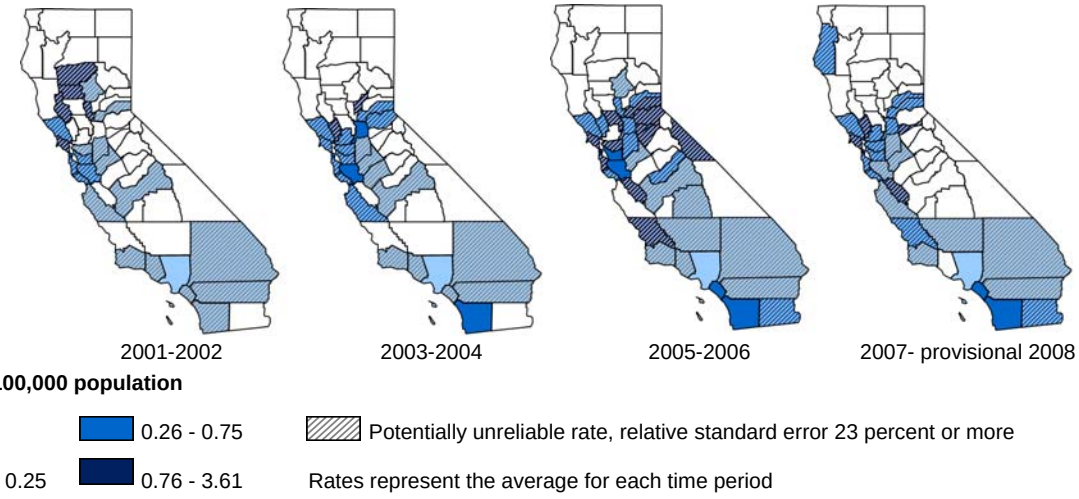
Notes for Figures 1-3

*2008 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



0.24 to 0.31 per 100,000). In Northern California, the highest incidence rate (0.51 per 100,000) occurred in 2005 and 2006. The 3 geographic regions of California with the highest rates for the surveillance period were the San Francisco Bay Area (0.56 per 100,000), San Diego (0.53 per 100,000), and Sacramento Metro (0.34 per 100,000) [Figure 4].

From 2001 through 2008, CDPH received reports of 6 confirmed and 6 suspected foodborne outbreaks of non-cholera vibriosis, involving 93 cases. Consumption of raw or undercooked oysters and mussels was the most frequently implicated exposure. The largest outbreak occurred in 2006, involved 27 persons with *V. parahaemolyticus* infections, and was associated with consumption of raw oysters.

Comment

During the surveillance period, the highest annual number of non-cholera vibriosis cases (176) was reported in 2006; most were due to *V. parahaemolyticus*. California experienced its highest incidence rates of the surveillance period in 2004 and 2006 and its largest outbreak in 2006. In 2006, at least three other states (New York, Oregon and Washington) also reported several large clusters of *V. parahaemolyticus* cases[3]. Traceback investigation linked contaminated oysters and clams that had been consumed by patients to harvest beds in Washington state and British Columbia. Previous non-cholera vibriosis outbreaks have coincided with large increases in sporadic cases nationally, and it is unclear whether the increase in sporadic cases in California in 2006 was related to these national clusters. In 2006, some shellfish harvest areas in Washington that were associated with outbreak cases had demonstrated acceptable *Vibrio* levels by routine testing[3]. Because *Vibrio* species multiply very rapidly and can reach infectious levels in seafood after harvest, shellfish bed monitoring is important but not sufficient to prevent illness[3].

Nevertheless, ensuring that shellfish beds are routinely monitored, seafood products are handled safely during and after harvest, and educating consumers about the risks of consuming raw or undercooked seafood may provide the best opportunities for reducing non-cholera vibriosis. Physicians should maintain a high index of suspicion in persons with gastroenteritis and a history of raw seafood consumption. Physicians suspecting vibriosis should also notify the laboratory of their suspicions so that the appropriate selective medium can be used to isolate the organism.

References and resources

[1]Mead PS, Slutsker L, Dietz V et al. Food-related illness and death in the United States. Emerg Infect Dis 1999;5:607-25.
http://www.cdc.gov/ncidod/eid/Vol5no5/pdf/mead.pdf

[2]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes
http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

[3]Centers for Disease Control and Prevention. Vibrio parahaemolyticus infectious associated with consumption of raw shellfish - three states, 2006. MMWR 2006;55:854-6.

Last updated 8/17/2009

Prepared by Kate Cummings, MPH, Amy Karon, DVM, and Duc J Vugia, MD, MPH, Infectious Diseases Branch

Epidemiologic Summary of West Nile Virus in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 2,765 cases of West Nile virus illness with estimated onset dates from 2003 through 2008.
- In 2002, one human case of West Nile virus was identified in southern California. However, further epidemiologic investigation determined that the individual likely acquired the infection in another state.
- Of the 2,765 cases reported during the surveillance period, 1,137 (41.1 percent) had a neuroinvasive form of illness (e.g., meningitis or encephalitis).
- Incidence rates of West Nile virus illness increased from the lowest of <0.1 per 100,000 in 2003 to the highest of 2.4 per 100,000 in 2005. Incidence rates have since fluctuated but have remained around 1 case per 100,000.
- Average annual incidence rates were highest among cases 65 to 84 years of age (3.0 per 100,000).
- West Nile virus cases with complete race/ethnicity data were reported to be White non-Hispanic (68.7 percent) more frequently than would be expected based on the overall demographic profile of Californians (44.2 percent).
- During the surveillance period, 48 (82.8 percent) of 58 counties reported at least 1 case.
- The best way to prevent West Nile virus infection is to avoid mosquito bites.

Background

West Nile virus is a mosquito-borne arbovirus occurring in Africa, Europe, west and central Asia, the Middle East, and most recently, North America. The virus was first detected in the United States in New York in 1999, and has since spread to 47 states including California. West Nile virus is typically transmitted through the bite of an infected mosquito. Mosquitoes become infected when they feed on infected birds.

Most persons infected with West Nile virus do not develop clinical illness or symptoms. However, approximately 20 percent of people develop symptoms compatible with what is known as West Nile fever. The West Nile fever syndrome can be variable, but common symptoms include fever, headache, muscle weakness, fatigue, or rash. About one in 150 people infected with West Nile virus develop severe neuroinvasive illness, e.g. meningitis, encephalitis, or acute flaccid paralysis. The incubation period for West Nile virus is commonly 3 to 14 days. There is no specific treatment. Though milder illness often improves without treatment, those cases with severe West Nile virus illness may require hospitalization and supportive care.

We describe here the epidemiology of human West Nile virus in California from 2003, when the first three locally acquired human cases were detected, through 2008. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to Technical Notes[1].

California reporting requirements and surveillance case definition

California Code of Regulations, Title 17, requires health care providers and laboratories to report cases of West Nile virus infection to their local health department via electronic transmission (including FAX), telephone, or mail within one working day of identification.

Local health officers are required by regulation to investigate provider or laboratory reports of West Nile virus infection and report any new cases of infection to CDPH. Asymptomatic infections, such as those detected in blood donors, are reportable but do not meet the surveillance case definition as defined by the Center for Disease Control and Prevention (CDC).

During the surveillance period, CDC defined a case of West Nile virus illness as meeting at least one of the clinical criteria for either neuroinvasive or non-neuroinvasive disease, and at least one of the laboratory criteria for diagnosis.

Cases of neuroinvasive disease had fever and at least one of the following: acutely altered mental status, other acute signs of central or peripheral neurologic

dysfunction, or pleocytosis associated with an illness clinically compatible with meningitis. Cases of non-neuroinvasive disease had fever, the absence of neuroinvasive disease, and the absence of a more likely clinical explanation for the illness.

CDC classified cases as confirmed if they met at least one of the following four laboratory criteria: (1) four-fold or greater change in virus-specific serum antibody titer; (2) isolation of virus from or demonstration of specific viral antigen or genomic sequences in tissue, blood, CSF, or other body fluid; (3) virus-specific immunoglobulin M (IgM) antibodies demonstrated in CSF by antibody capture enzyme immunoassay (EIA); or (4) virus-specific IgM antibodies demonstrated in serum by antibody-capture EIA and confirmed by demonstration of virus-specific serum immunoglobulin G (IgG) antibodies in the same or later specimen by another serologic assay (e.g. neutralization or hemagglutination inhibition).

CDC classified cases as probable if they had either (1) stable (less than or equal to a two-fold change) but elevated titer of virus-specific serum antibodies; or (2) virus-specific serum IgM antibodies detected by antibody-capture EIA but with no available results of a confirmatory test for virus-specific serum IgG antibodies in the same or a later specimen.

Since West Nile virus antibodies can cross-react with other flavivirus antibodies and since IgM antibodies can persist for up to one year, laboratory diagnosis of acute West Nile virus infection can often involve a multi-step process with multiple test assays. Caution should be used when interpreting laboratory reports.

Epidemiology of West Nile virus in California

CDPH received reports of 2,765 cases of locally acquired West Nile virus illness with estimated onset dates from 2003 through 2008[2]. This corresponds to an average annual incidence rate of 1.2 per 100,000 California residents. Incidence rates were highest in 2004 and 2005, when California was the epicenter of national West Nile virus activity. The incidence rates were 2.1 and 2.4 per 100,000 in 2004 and 2005, respectively. From 2006-2008, annual incidence rates decreased to an average of 1 case per 100,000 [Figure 1].

Of the 2,765 cases reported to CDPH, 1,137 (41.1 percent) were neuroinvasive disease. The ratio of male to female cases was 1.5:1.0. The median age of all cases was 52 years (range: 2-96 years). Average incidence rates for the combined years 2003-2004, 2005-2006, and 2007-2008 were highest

Figure 1. California West Nile virus case counts and incidence rates, 2003-2008*

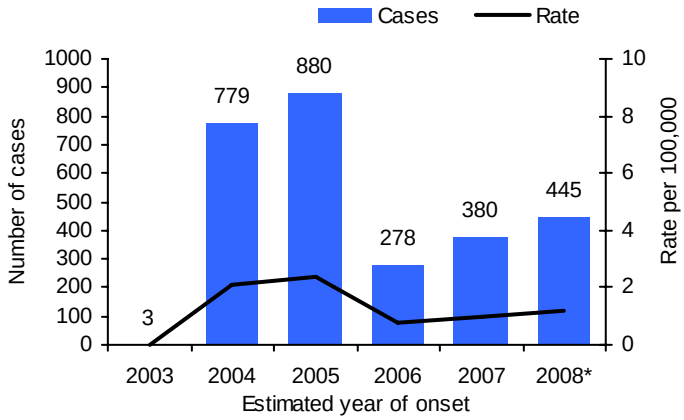


Figure 2. California West Nile virus incidence rates by age, 2003 - 2008*

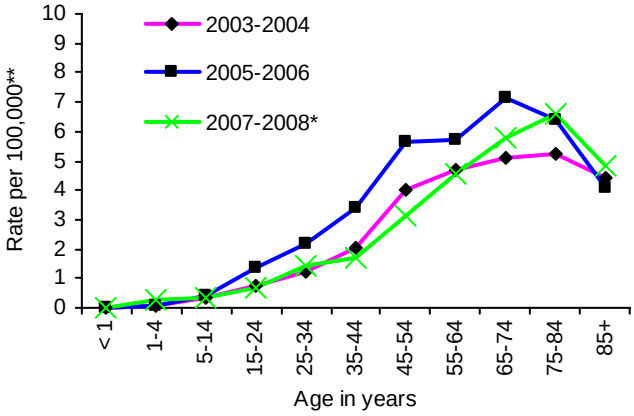
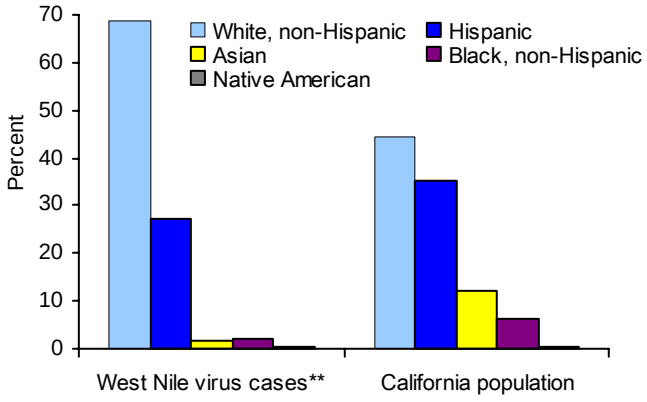


Figure 3. California West Nile virus cases and population by Race/ethnicity, 2003 - 2008*

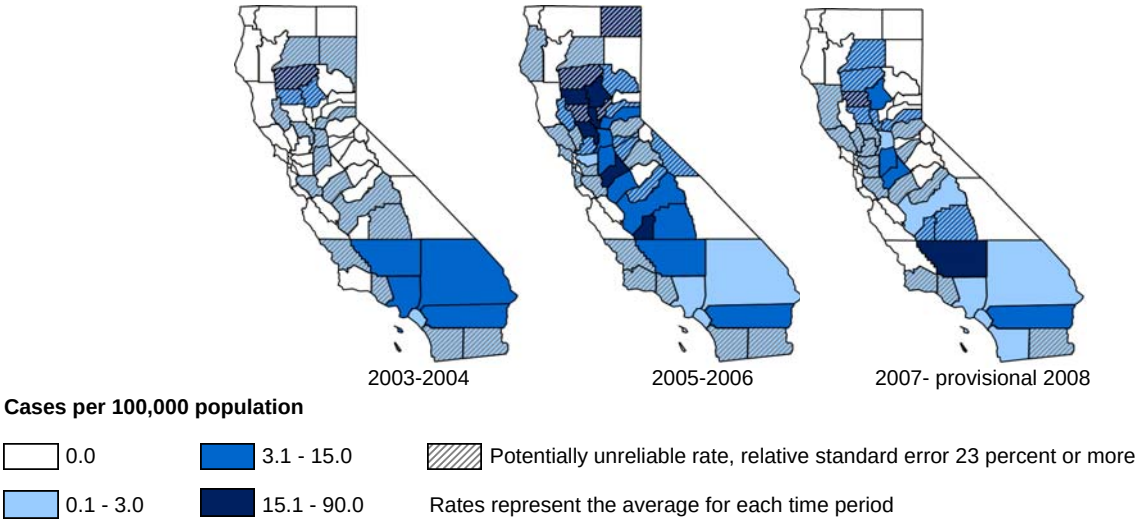


Notes for Figures 1-3

*2008 data are provisional

**Unknowns were excluded

Figure 4. California county-specific West Nile virus incidence rates, 2003 - 2008



among cases 75 to 84 years of age (6.1 per 100,000). Incidence rates among cases 75 to 84 years of age increased by 25.2 percent from the combined years 2003 and 2004 (5.3 per 100,000) to the combined years 2007 and 2008 (6.6 per 100,000) [Figure 2].

Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (23.9 percent). Of the West Nile virus cases with complete data reported, 68.7 percent were non-Hispanic White, 27.2 percent were Hispanic, 2.2 percent were non-Hispanic Black, 1.5 percent were Asian, and 0.4 percent were Native American [Figure 3]. White, non-Hispanic cases were reported more frequently than would be expected based on the overall demographic profile of California, while Hispanic, Asian, non-Hispanic Black and Native American cases were reported less frequently than would be expected.

In California, cases of West Nile virus illness typically occur during the summer and fall seasons. During the surveillance period, 88.7 percent of all reported cases experienced onset of symptoms in the months of June through September. Forty-eight (82.8 percent) of 58 counties reported at least 1 case during the surveillance period [Figure 4].

Comment

Being outdoors increases the risk of being bitten by an infected mosquito for all individuals. The best way to prevent West Nile virus infection is to avoid mosquito bites. Some ways to do this include using insect repellent, staying indoors during dawn and dusk (peak mosquito biting times), installing or fixing door and window screens, and draining standing water in and around the yard. Local and state public health and vector control agencies also work to prevent and control outbreaks of West Nile virus.

Some individuals appear to be at a greater risk for developing severe symptoms from West Nile virus infection. Among the cases reported in California, risk factors associated with developing neuroinvasive disease as compared to non-neuroinvasive disease included older age, male gender, and diabetes mellitus[3].

References and resources

[1]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

[2]California West Nile virus website http://www.westnile.ca.gov

[3]Jean CM, Honarmand S, Louie JK, Glaser CA. Risk factors for West Nile virus neuroinvasive disease, California, 2005. Emerg Infect Dis. 2007; 13(12):1918-20.

Last updated 05/05/2010

Prepared by Cynthia Jean Yen, MPH, Farzaneh Tabnak, MS, PhD, and Kate Cummings, MPH

Infectious Diseases Branch and Communicable Disease Emergency Response Branch

Epidemiologic Summary of Wound Botulism in California, 2001 - 2008

Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of 185 cases of wound botulism with estimated onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 0.06 per 100,000 Californians.
- Annual wound botulism incidence rates remained relatively level from 2001 to 2008 with the exception of an increase from 2004 (0.05 per 100,000) to 2006 (0.10 per 100,000).
- During the surveillance period, 3 (1.7 percent) cases were reported to have died with wound botulism.
- The ratio of male to female cases was 3.1:1.0.
- 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]. *C. botulimum* toxin is listed among the Centers for Disease Control and Prevention (CDC) category A bioterrorism (BT) agents.

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 wound botulism in California from 2001 through 2008. Data for 2008 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[2].

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. CDPH assumed that all injecting drug users had contaminated wounds even if the wounds were not apparent on physical exam.

Epidemiology of wound botulism in California

CDPH received reports of 185 cases of wound botulism with estimated onset dates from 2001 through 2008. This corresponds to an average annual incidence rate of 0.06 per 100,000 Californians. Annual wound botulism incidence rates remained relatively level from 2001 to 2008 with the exception of an increase from 2004 (0.05 per 100,000) to 2006 (0.10

per 100,000) [Figure 1]. During the surveillance period, 3 (1.7 percent) cases were reported to have died with wound botulism.

During the surveillance period, average annual incidence rates of wound botulism were highest among persons 45 to 54 years of age [Figure 2]. There were no cases in children under 15 years of age. The ratio of male to female cases was 3.1:1.0. Incidence rates by race/ethnicity were not calculated because of the substantial portion of missing data (14.0 percent). However, wound botulism cases with complete information on race/ethnicity reported Hispanic ethnicity more frequently and Asian, Pacific Islander race less frequently than would be expected based on the overall demographic profile of California [Figure 3].

During the surveillance period, 23 counties reported at least 1 case of wound botulism during the surveillance period. These counties were distributed throughout the state so that all but 1 region of the state (the Sierras) reported at least 1 case. The Sacramento Metro (0.14 per 100,000), Inland Empire (0.09 per 100,000), and Bay Area (0.08 per 100,000) regions reported the highest average annual incidence rates for the surveillance period.

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. The epidemic of wound botulism among California injecting drug users continued but did not appear to increase 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, et al. Wound botulism in California, 1951-98: Recent Epidemic in Heroin Injectors. Clin Infect Diseases 2000;31:1018-24.

[2]Epidemiologic Summaries of Selected General Communicable Diseases in California, 2001-2008: Technical Notes http://www.cdph.ca.gov/data/statistics/Documents/technicalnotes-episummary-aug2409.pdf

Last updated 10/22/2009

Prepared by Kate Cummings, MPH, Debra Gilliss MD, MPH, and Charlotte Wheeler, MD, MPH, Infectious Diseases Branch

Figure 1. California wound botulism case counts and incidence rates

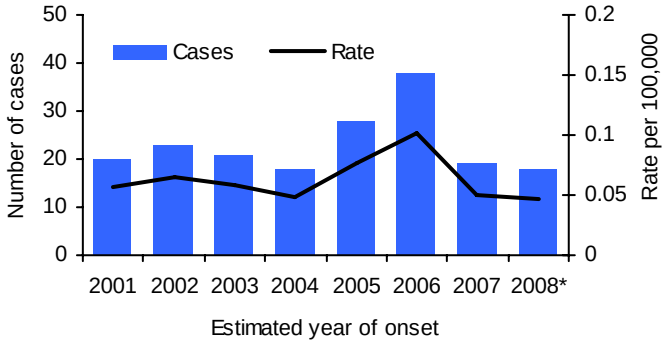


Figure 2. California wound botulism incidence rates by age, 2001-2008*

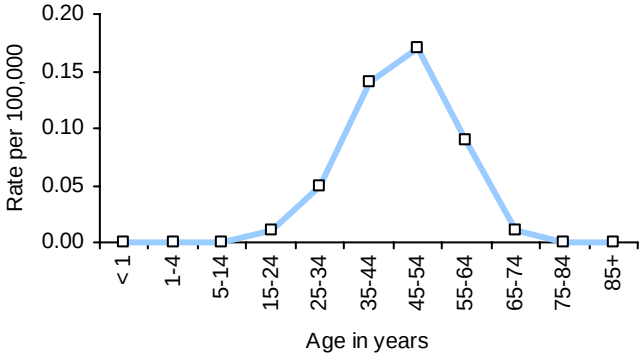
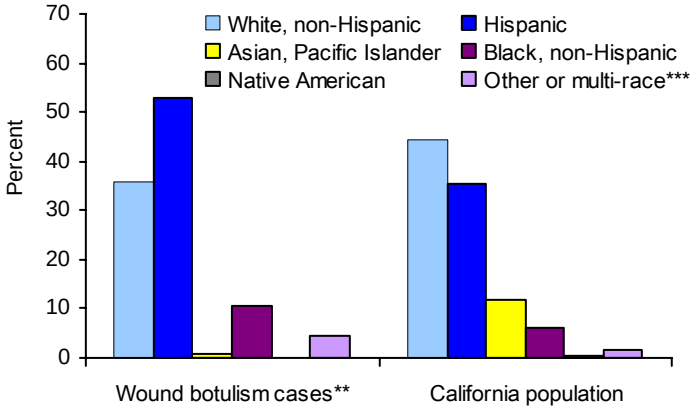


Figure 3. California wound botulism cases and population by race/ethnicity, 2001 - 2008*



Notes for Figures 1-3

*2008 data are provisional

**Unknowns were excluded

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

Report published October 2011

Surveillance and Statistics Section
Infectious Diseases Branch
Division of Communicable Disease Control
Center for Infectious Diseases
California Department of Public Health
1616 Capitol Avenue, MS 7306
P.O. Box 997377
Sacramento, CA 95899-7377
916-552-9720
www.cdph.ca.gov/programs/sss