ACUTE COMMUNICABLE DISEASE CONTROL PROGRAM ANNUAL MORBIDITY REPORT AND SPECIAL STUDIES REPORT

2015





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Message from the Director 2015

It is my privilege to present the Acute Communicable Disease Control (ACDC) program's 2015 Annual Morbidity and Special Studies Report summarizing communicable disease trends and investigations for the year. As the Interim Director, I am honored to share these data on behalf of our staff that expertly manages surveillance and response for over 60 reportable communicable diseases and conditions. ACDC frequently is at the forefront addressing notable public health challenges through investigations requiring the use of innovative techniques and systems to best understand infectious diseases across a range of diverse domains (foodborne, vectorborne, bloodborne, healthcare-associated, antimicrobial-resistant pathogens, and select vaccine-preventable pathogens). In our efforts, we frequently partner with state and national agencies, and many of our findings have impact both locally and nationally, directing policy and prevention standards to protect the health of our communities. This was especially true during 2015.

2015 is the year that saw the conclusion of Ebola, the advent of Zika, the continued rise in multidrug resistant organisms (MDRO), and the continued emergence of West Nile virus as an annual health threat. Ebola virus disease was undoubtedly the most noteworthy public health emergency of the year. Beginning in March 2014, the disease rapidly spread through several West African nations resulting in the largest Ebola outbreak in history. While the vast majority of infections occurred in West Africa, this was the first Ebola outbreak to result in the spread of this disease to and in the U.S. In response, the Centers for Disease Control and Prevention (CDC) enacted a traveler monitoring program to identify those who were possibly exposed to the virus and to track their health while they were in the country, with the goal of preventing the spread of the virus if they had been infected. Local public health departments were responsible for monitoring the travelers within their jurisdiction, and this program ran for about 14 months, from October 21, 2014 through January 4, 2016. Over the entire course of the program, 269 travelers were referred to the Los Angeles County Department of Public Health (LAC DPH) for monitoring. Of these, medical assessments were deemed necessary for eight travelers, four met the criteria for Ebola testing. None tested positive, and all eight had a non-Ebola virus disease diagnosis. Throughout LAC DPH's response, ACDC served as the primary medical expert overseeing these potential cases. In addition, ACDC managed the brunt of the data assessment and analyses supporting this complex national requirement. ACDC also was instrumental in ensuring that our local hospitals were prepared to enact the necessary and rigorous infection control protocols. Ultimately, our preparedness activities resulted in establishing two Ebola assessment hospitals and two Ebola treatment hospitals. These critical partnerships were designed to serve local needs as well as the needs of neighboring jurisdictions. Even though the Ebola outbreak now has been controlled, these four facilities continue to work with LAC DPH to maintain their infection control preparedness to respond to other novel contagious diseases.

2015 also was notable for the rise in mosquito-borne diseases. West Nile virus (WNV), inflicted a substantial public health burden that year resulting in 300 confirmed cases and 24 deaths—the greatest number of local cases since the year that the disease first emerged in 2004 when 309 cases occurred and the most fatalities ever reported. 2015 also saw a peak in travel-associated diseases transmitted by *Aedes* mosquitoes. ACDC confirmed 30 cases of dengue and 107 cases of chikungunya virus during the year. 2015 also marked the first reports of Zika virus disease among our residents with six cases identified during the final two months of the year. ACDC greatly expanded surveillance activities and coordinated partnerships with the Public Health Laboratory, the Maternal, Child and Adolescent Health Program, and Children's Medical Services to address these burgeoning disease threats. We also enhanced our ongoing partnerships with the five mosquito and vector control agencies in LAC which have been vital in investigation and response.

ACDC is proud to be a national leader among local health departments in surveillance and response to infections from MDROs. In 2015, we led an outbreak investigation of multidrug resistant infections associated with a complex endoscope device. Through outreach to other facilities that used this device, we identified additional cases and outbreaks at other hospitals. Results were shared with CDC and the Food and Drug Administration, and presented at a national infectious diseases conference. Ultimately, our work resulted in new national recommendations for cleaning and disinfection of these devices. Beyond MDROs, the reduction of healthcare–acquired infections is a priority at ACDC. We continue to maintain our Hospital Outreach Unit, which fosters close partnerships with infection preventionists and other key staff at all 99 local hospitals. We collaborate to improve influenza vaccination rates among healthcare workers in Los Angeles County and have enacted programs to improve the identification, reporting, and prevention of healthcare–associated infections in areas beyond acute care hospital such as large clinics, ambulatory surgery centers, and skilled nursing facilities.

These events and more are detailed in ACDC's 2015 Annual Morbidity and Special Studies Report. We hope you find the report to be a useful resource for you and your organization.

Sincerely, Benjamin Schwartz, MD



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Acute Communicable Disease Control

The Acute Communicable Disease Control Program



(ACDC) serves a fundamental role in disease control and prevention in Los Angeles County (LAC). Our program leads surveillance,

investigation, and outbreak response for over 60 reportable communicable diseases, which account for considerable morbidity and mortality among LAC residents. In 2015, ACDC managed and confirmed over 4,000 reports of communicable diseases, many of which required further investigation and response (Table 1). Our findings are instrumental in the development of guidance and policy recommendations and inform prevention efforts for communicable diseases locally and nationally.

Enhancing surveillance for reportable diseases, ACDC manages an electronic laboratory-based reporting (ELR) system, receiving reports from participating laboratories not only for ACDC but also other county public health programs including TB, HIV/STD, and Immunization. In 2015, over 446,000 reports were processed through this system. ACDC's case surveillance system, visual confidential morbidity reporting (vCMR), has been identified as the enterprise surveillance and case management system for the LAC Department of Public Health (DPH). Care reports in vCMR include the range of conditions as in ELR as well as non-communicable diseases such as tracking of Fentanyl abuse reports for the Substance Abuse Prevention and Control program.

ACDC staff also serve as local experts in these diseases, providing vital guidance and recommendations for medical and community partners. Additionally, ACDC is the designated public health program responder for emerging infectious diseases such as Zika virus, Ebola, Middle East Respiratory Syndrome Coronavirus

Table 1.

ACDC-Managed Communicable Disease Reports for Selected Pathogens Los Angeles County, 2015

Disease	No. of Cases		
Gastrointestinal Disease			
<u>Salmonella</u>	1,144		
<u>E. coli</u> (Shiga toxin)	175		
<u>Shigella</u>	508		
Hepatitis A	33		
Vectorborne Diseases			
West Nile Virus	300		
<u>Dengue</u>	30		
<u>Malaria</u>	27		
<u>Typhus</u>	54		
Bloodborne Diseases			
Hepatitis B	50		
Respiratory Disease			
Influenza-Associated Deaths	54, 84		
Legionellosis	171		
Coccidioidomycosis	613		
Neuroinvasive Disease			
Viral Meningitis	367		
Meningococcal Disease	12		

* 2014-2015 season, 2015-2016 season, respectively.

(MERS-CoV), pandemic influenza, antimicrobial resistant organisms, and bioterrorism agents (e.g., smallpox, anthrax, and botulism). ACDC partners with local hospitals, healthcare facilities, and skilled nursing facilities to assist with infection control and outbreak response. ACDC physicians are available and on-call everyday (24/7) to ensure the health and safety of our communities.

The following are some highlights from ACDC's activities and accomplishments occurring during 2015.

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Ebola Outbreak Response

The outbreak of <u>Ebola viral disease (EVD)</u> in West Africa was the largest outbreak of EVD in history and the first Ebola outbreak that resulted in transmission of this disease in the U.S. The outbreak began in March 2014 with peak incidence of cases in three countries (Liberia, Guinea, and Sierra Leone) occurring between August and November of that year. Starting in October 2014, the U.S. required that all travelers from these three countries fly into one of five airports to be screened for possible EVD exposure and infection. In addition, on October 24, the Centers for Disease Control and



<u>Prevention (CDC)</u> announced that <u>all passengers from these countries would receive 21-day monitoring</u> by local public health departments while in the U.S. This nationwide requirement continued through 2015 and did not end until January 4, 2016. Over the full course of outbreak, the LAC DPH <u>monitored 269 travelers and oversaw in-</u> <u>depth medical assessments of 8 individuals who were symptomatic</u> (Figure 1). Of these, testing for possible EVD infection was deemed necessary for 4 individuals—all tested negative.



Figure 1.

Number of Travelers Monitored and Assessed for EVD by

Ensuring that our hospitals and medical communities were prepared to safely identify and care for to Ebola patients, maintaining rigorous infection protocols, was an important priority in 2015. Beginning in August 2014, LAC DPH's Health Officer requested that ACDC conduct outreach to local hospitals in anticipation of the worsening of the outbreak and due to an increase in the number of healthcare workers traveling to EVD affected countries on medical missions who were returning to the LAC. A survey was sent by email to hospital infection preventionists (IP) to assess which locations would voluntarily accept a suspect EVD patient in LAC. Letters from the Health Officer were sent to hospital IPs and Chief Executive Officers to encourage preparedness efforts and collaboration with LAC DPH. Of the 71 hospitals with emergency departments, LAC DPH conducted outreach to 51 (70%) through site visits, drill participation, and policy review. A total of 31 hospitals conducted preparedness drills with LAC DPH staff participation. As a result of our activities, two Ebola assessment preparedness hospitals and two Ebola treatment hospitals were established. These four facilities continue to work with LAC DPH to maintain preparedness for Ebola and other novel contagious diseases.

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ACDC conducted several EVD-related special projects during 2015 including <u>supervising the delivery of an infant</u> to a mother who was previously infected with EVD and subsequently developing detailed infection control guidelines for these events, <u>establishing a home EVD patient assessment protocol</u>, and conducting analyses of the demographics of travelers in LAC who required monitoring for EVD infection as well as <u>considering possible</u> <u>improvements to their daily febrile assessments</u>. These projects are summarized in **ACDC's 2015 Special Studies Report**.

Vectorborne Diseases

A sharp rise in vectorborne diseases inflicted substantial public health burden in LAC during 2015. That year, <u>300 West Nile</u> <u>virus (WNV) cases including 24 deaths</u> were identified among our residents. This was the greatest number of <u>WNV</u> infections and fatalities to occur locally since this disease first emerged in our area in 2004; <u>when WNV contributed to 309 cases and 13</u> fatalities (Figure 2). The increase in cases and the occurrence of a substantial number



of cases annually represents a change in the epidemiology of what initially had been a 4-year cycle of disease. <u>Historic high temperatures</u> and drought experienced in LAC in 2015 might have contributed to this increase in WNV cases.

Figure 3. Aedes species mosquito



While WNV is transmitted by the bite of infected *Culex* mosquitoes, which are indigenous to LAC, 2015 also saw the rise in travel-associated cases of diseases (dengue, chikungunya, and Zika) transmitted by *Aedes* mosquitoes (Figure 3). Invasive Aedes mosquitoes have been found in over 50 cities across LAC, although they presently do not spread this group of diseases. In 2015, 20 travel-associated <u>dengue</u> cases and 107 travel-associated <u>chikungunya</u> cases were identified in LAC. The year also marked the advent of travel-associated <u>Zika virus</u> infections among LAC residents with the first documented case identified in November. A total of six travel-associated cases were documented by the end of 2015. While Zika-infected *Aedes* mosquitoes

were not identified locally, the continuing expansion of this competent vector across the county, coupled with the ongoing occurrence of human cases, increases the potential that local disease transmission might occur.

ACDC also responded to two significant flea-borne disease outbreaks in 2015. In June, ACDC was alerted to three hospitalized <u>flea-borne typhus</u> cases among residents of a large mobile home community. ACDC coordinated a <u>multi-agency investigation of this outbreak</u> to identify additional cases, define and mitigate risk factors, and prevent further cases from occurring. In August, ACDC staff partnered with state and federal agencies when <u>plague</u>

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was <u>diagnosed for two persons who had visited Yosemite National Park in California</u>. One case was septicemic and the other bubonic. Subsequent environmental investigation identified probable locations of exposure for each patient and evidence of epizootic plague in other areas of the park. These projects are summarized in <u>ACDC's</u> <u>2015 Special Studies Report</u>.

Influenza Surveillance

Influenza seasons straddle the new year, typically beginning in October and extending through March. The 2014–2015 season was mild to moderately severe. Influenza A H3N2 was the predominant circulating strain with activity peaking the second week of January 2015 (as summarized in <u>ACDC's 2014 Special Studies Report</u>). This was followed by a subsequent slight increase in type B influenza in March 2015, which is common for LAC (Figure 4). During the 2014–2015 season, a total of 54 confirmed influenza-associated deaths (51 adult, 3 pediatric) were reported with the majority of fatalities occurring among those 65 years and older, which is consistent with other influenza A H3N2 seasons. During this season, the influenza A H1N1 pandemic strain was detected at the lowest level since its emergence in 2009—less than 1% of all subtyped influenza A nationally. Most notably, this season the influenza both locally and nationwide had the potential to be much more severe.



For the 2015–2016 season, the vaccine was modified leading to a better match the circulating influenza A H3N2 strain. Locally that season, activity was moderate overall, peaking in the final weeks of February 2016. Influenza A H1N1 was the predominant A strain circulating, though A and B viruses were almost equally represented throughout the season, which is very uncommon. A total of 84 confirmed influenza-associated deaths (81 adult, 3 pediatric) were reported. The CDC estimates that about 90% of all influenza-associated deaths occur among those 65 years and older;¹ however, during H1N1 seasons, the 20–60 years old age group accounts for a greater proportion of deaths as compared to H3N2 seasons. This shift in the ages of those impacted by influenza is reflected in LAC—especially when comparing the 2014–2015 versus the 2015–2016 seasons (Figure 5).

¹ www.cdc.gov/flu/about/disease/us_flu-related_deaths.htm

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Food and Waterborne Diseases

Foodborne disease surveillance, investigation, and prevention are core ACDC responsibilities, especially since these diseases result in significant morbidity—over 2,000 reportable cases were confirmed in 2015 alone. The extremely large area, population, and diversity of LAC makes investigating and preventing foodborne illnesses especially challenging. ACDC's investigations frequently require responding to complex situations, including outbreaks that span multiple jurisdictions, and potentially involving a range of food sources from differing cultures and backgrounds.

Many of ACDC's suspected foodborne disease investigations are initiated from information received by the public through our <u>foodborne illness report (FBIR) webpage</u>.² In 2015, we received 1,892 FBIRs. Two of our more notable investigations conducted that year were launched from FBIRs (see <u>ACDC's 2015 Special Studies Report</u>).

In late February through early March, <u>ACDC received three separate FBIRs from different parties describing similar</u> <u>gastrointestinal illness (GI) after dining at the same seafood buffet</u>. ACDC interviewed and collected specimens for testing from all 31 restaurant employees. Only one employee admitted to experiencing GI symptoms during the outbreak period; tests from that employee were negative for norovirus, *Shigella*, and *Salmonella*. Among the remaining employees, two tested positive for norovirus and one for *Salmonella*. Employees who tested positive were temporarily removed from work until cleared by LAC DPH. The restaurant was inspected by Environmental Health Services (EHS); two critical violations were identified, and the restaurant voluntarily closed to conduct a thorough cleaning and sanitization. Analyses of food consumption histories identified oysters as significantly

² <u>https://www.visualcmr.net/webvcmr/pages/public/pub_FBI_Report.aspx</u>

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associated with illness. The remaining oysters were red tagged and submitted for testing at the LAC DPH Public Health Laboratory which detected two separate strains of norovirus. The implicated oysters were harvested in another country and sold frozen in the U.S. Ultimately this investigation, along with corroboration from additional investigations of these imported oysters, prompted the Food and Drug Administration to take regulatory action with the importer to prevent others from potentially becoming ill with norovirus.

In September, ACDC received an FBIR from the public describing several individuals ill with GI symptoms after attending a corporate luncheon at an upscale LAC restaurant. While ACDC was interviewing the attendees, an LAC DPH public health nurse (PHN) notified ACDC of an employee of the restaurant who tested positive for Salmonella. With this information, ACDC alerted all Community Health Services (CHS) PHNs of this potential outbreak and requested them to be on the lookout for additional cases. PHNs subsequently identified eight additional cases connected to the restaurant. The following week, ACDC received eight more FBIRs reporting illness among individuals who ate at the implicated restaurant. Collectively, food and illness history questionnaires were completed on 81 individuals. Specimens were obtained from the restaurant employees—14 were positive for S. enteritidis with a matching PFGE pattern. Because this LAC restaurant is one of a larger chain, ACDC partnered with the CDC and identified cases in another state. ACDC's analyses identified a single food item as significantly associated with illness, however, the preparation of that item complicated our ability to definitively assign a single ingredient as being responsible for illness. In addition, we were unable to determine whether an ingredient or improper preparation contaminated the food. ACDC and CHS worked with the restaurant managers to ensure that the 14 employees who tested positive were either removed from work until they were cleared to return or were placed in duties that did not involve food handling. In addition, the restaurant owners and managers were educated about methods to prevent future Salmonella infections.

A change in enteric disease testing methods occurred in 2015 that will impact comparisons of foodborne disease incidence between years. This year, the LAC DPH Public Health Laboratory expanded its testing protocol to include both culture-based methods and polymerase chain reaction (PCR), which will lead to an increase in the identification of enteric diseases, particularly Shiga toxin-producing <u>Escherichia coli</u> (STEC).

Healthcare Outreach and infection Control

Prevention of healthcare associated infections continues to be a program priority. Unique among health departments in California, ACDC maintains a team of liaison Public Health Nurses (PHNs) within the <u>Healthcare</u> <u>Outreach Unit (HOU)</u> who work closely with the infection preventionists (IPs) at all 99 LAC hospitals to support improved disease identification, reporting, and prevention. Over a third of the hospitals in LAC invite our liaison PHNs to their infection control meetings to further the integration of public health goals into the hospital setting. Our efforts and have improved acceptance of influenza vaccination among healthcare workers in compliance with LAC DPH's Health Officer order. In addition, outreach has expanded to include non-hospital healthcare settings such as large clinics and ambulatory surgery settings. In 2015, <u>ACDC held its first symposium for skilled nursing facilities</u> to improve understanding, surveillance, and response to infectious diseases that are common in these settings. This event was so well-received it has become an annual event.

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One of the more notable and extensive investigations headed by the HOU in 2015 was in response to carbapenem-resistant Enterobacteriaceae (CRE) infections associated with endoscopic retrograde cholangiopancreatography (ERCP) procedures. These procedures have been described previously in the literature to be linked with outbreaks due to the complexity of the design of the scope, which can lead to bacterial contamination of difficult to clean areas, particularly within the elevator channel mechanism at the tip (Figure 6). ACDC's investigations began with a report in January 2015 of a cluster of patients who were carbapenemresistant Klebsiella pneumoniae (CRKP) culture positive after undergoing an ERCP procedure. Infection was associated with the use of specific endoscopes. This investigation prompted us to notify all LAC acute care hospitals urging active surveillance for CRE infections following ERCP procedures, including a retrospective review. Additional clusters were identified and reported, and ACDC initiated outbreak investigations at two additional hospitals.

Figure 6. Close-up view endoscope tip



Epidemiology and laboratory analyses suggest that the cause of these outbreaks were multifactorial, including that the complex design of the scope may have impeded effective cleaning, disinfection, and reprocessing. In January 2016, the duodenoscope manufacturer initiated a recall of one scope model for replacement of the elevator mechanism. In addition, several nationally recognized experts have since recommended options to enhance disinfection and reprocessing. The facilities experiencing these outbreaks were large, prestigious hospitals with robust infection prevention and control programs. Due to the design flaw of this instrument, hospitals could follow manufacturer guidelines and standard practices correctly and still experience scope-related infections. These investigations illustrate the importance of supportive, ongoing partnerships between hospitals and LAC DPH to ensure optimal surveillance and coordination of prevention activities and to ultimately improve patient safety.



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ACUTE COMMUNICABLE DISEASE CONTROL PROGRAM ANNUAL MORBIDITY REPORT OVERVIEW 2015

PURPOSE

The Acute Communicable Disease Control Program's (ACDC) Annual Morbidity Report of the Los Angeles County (LAC) Department of Public Health (DPH) is compiled to:

- 1. summarize annual morbidity from several acute communicable diseases occurring in LAC;
- 2. identify patterns of disease as a means of directing future disease prevention efforts;
- 3. identify limitations of the data used for the above purposes and to identify means of improving that data; and
- 4. serve as a resource for medical, public health, and other healthcare authorities at county, state and national levels.

<u>Note</u>: This report includes information on select vaccine preventable diseases (such as influenza and hepatitis A and B). For information on haemophilus influenzae, perinatal hepatitis B, measles, mumps, and pertussis, see LAC DPH's Immunization Program (www.publichealth.lacounty.gov/ip/index.htm). This report does <u>not</u> include information on tuberculosis, sexually transmitted diseases, or HIV and AIDS. Information regarding these diseases is available from their respective department programs (see LAC DPH website for more information at www.publichealth.lacounty.gov/index.htm).

LAC DEMOGRAPHIC DATA

LAC population estimates used for this report were created under contract for the County of Los Angeles, Internal Services Department.¹ The base population numbers came from the 2010 Census from which we extracted and aggregated data into the age, race-ethnicity, and sex categories required by the County. These numbers were updated to July 1, 2010, using city estimates from the California Department of Finance (DOF), Demographic Research Unit. July 1, 2015 population estimates were obtained by applying 5 years of birth, mortality and migration rates to the July 1, 2010 estimates. The estimates were also controlled to city and county level estimates from the California Department of Finance, Demographic Research Unit. The input datasets included Census Bureau decennial census enumerations and annual population estimates, DOF city and county estimates, and administrative records from the County of Los Angeles on registered voters, housing units, births and deaths. LAC population estimates used for this report are created by Hedderson Demographic Services and provided to the LAC Department of Public Health by Urban Research of the LAC Internal Services Department (ISD).

National and California State counts of reportable diseases can be obtained from the Centers for Disease Control and Prevention (CDC) Final 2015 Summary of Nationally Notifiable Infectious Diseases published in the Morbidity and Mortality Weekly Report (MMWR).²

Cities of Long Beach and Pasadena are separate reporting jurisdictions, as recognized by the California Department of Public Health, and as such these two cities maintain their own disease reporting systems. Therefore, disease episodes occurring among residents of Long Beach and Pasadena have been excluded

¹ County of Los Angeles, Internal Services Department, Information Technology Service, Urban Research-GIS Section, Population and Poverty Estimates of Los Angeles County Tract-City Splits by Age, Sex and Race-Ethnicity for July 1, 2015, Los Angeles, CA, April 15, 2016.

² CDC. *Notice to Readers:* Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306–1321. Available at: www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm



from LAC morbidity data, and their populations subtracted from LAC population data. Exceptions to this rule are noted in the text when they occur.

DATA SOURCES

Data on occurrence of communicable diseases in LAC were obtained through passive and sometimes active surveillance. Every healthcare provider or administrator of a health facility or clinic, and anyone in charge of a public or private school (of any grade level) knowing of a <u>case or suspected case</u> of a communicable disease is required to report it to the local health department as specified by the California Code of Regulations (Section 2500). Immediate reporting by telephone is also required for any <u>outbreak</u> or <u>unusual incidence</u> of infectious disease and any <u>unusual disease</u> not listed in Section 2500. Laboratories have separate requirements for reporting certain communicable diseases (Section 2505). Healthcare providers must also give detailed instructions to household members in regard to precautionary measures to be taken for preventing the spread of disease (Section 2514). Disease reporting standards sometime differ from those of both state and federal guidance. The most current version of LAC DPH's listing of reportable diseases and conditions is available on our website at: www.publichealth.lacounty.gov/acd/docs/DiseaseListOct2016.pdf

- 1. Passive surveillance relies on physicians, laboratories, and other healthcare providers to report diseases of their own accord to the DPH using the Confidential Morbidity Report (CMR) form, electronically, by telephone, or by facsimile.
- 2. Active surveillance entails ACDC staff regularly contacting hospitals, laboratories and other healthcare providers in an effort to identify all cases of a given disease.

DATA DESCRIPTION AND LIMITATIONS

Data in this report utilizes the following data descriptions, however, the report should be interpreted with caution of the notable limitations.

1. Underreporting

The proportion of cases that are not reported varies for each disease. Evidence indicates that for some diseases as many as 98% of cases are not reported.

2. Reliability of Rates

All vital statistics rates, including morbidity rates, are subject to random variation. This variation is inversely related to the number of events (observations, cases) used to calculate the rate. The smaller the frequency of occurrence of an event, the less stable its occurrence from observation to observation. As a consequence, diseases with only a few cases reported per year can have highly unstable rates. The observation and enumeration of these "rare events" is beset with uncertainty. The observation of zero events is especially hazardous.

To account for these instabilities, all rates in the ACDC Annual Morbidity Report based on less than 19 events are considered "unreliable". This translates into a relative standard error of the rate of 23% or more, which is the cut-off for rate reliability used by the National Center for Health Statistics.

In the Annual Morbidity Report, rates of disease for groups (e.g., Hispanic versus non-Hispanic) are said to differ significantly only when two criteria are met: 1) group rates are reliable and 2) the 95% confidence limits for these rates do not overlap. Confidence limits are calculated only those rates which are reliable.

3. Case Definitions

To standardize surveillance, CDC/CSTE (Council of State and Territorial Epidemiologists) case definition for infectious diseases under public surveillance³ is used with some exceptions as noted in the text of the

³ CDC. Case definitions for infectious conditions under public health surveillance. MMWR 1997; 46(RR10):1-55. Available at: https://www.cdc.gov/mmwr/preview/mmwrhtml/00047449.htm



individual diseases. Since verification by a laboratory test is required for the diagnosis of some diseases, cases reported without such verification may not be true cases. Therefore, an association between a communicable disease and a death or an outbreak possibly may not be identified.

- Onset Date versus Report Date Slight differences in the number of cases and rates of disease for the year may be observed in subsequent annual reports. Any such disparities are likely to be small.
- 5. <u>Population Estimates</u>

Estimates of the LAC population are subject to limitations. Furthermore, the population of LAC is in constant flux. Though not accounted for in census data, visitors and other non-residents may have an effect on disease occurrences.

6. Place of Acquisition of Infections

Some cases of diseases reported in LAC may have been acquired outside of the county. Geographical data is presented based on address of case, therefore, some disease rates may not accurately reflect the location where an infection was acquired.

7. Health Districts and Service Planning Areas

Since 1999, LAC is divided into eight "Service Planning Areas" (SPAs) for purposes of healthcare planning and provision of health services: SPA 1 Antelope Valley, SPA 2 San Fernando, SPA 3 San Gabriel, SPA 4 Metro, SPA 5 West, SPA 6 South, SPA 7 East, and SPA 8 South Bay. Each SPA is organized further into health districts (HDs). The SPAs are shown on the map included in this section. Due to variations in Community Health Services staffing, investigating District personnel can be different than the standard District of residence. Approximately 9% of County census tracts have been shifted in such a manner. For the purpose of this publication, case or outbreak location is consistently matched to the official District/SPA of record. A SPA map (last updated in 2012) is provided below and available at: www.publichealth.lacounty.gov/epi/images/GIS/SPA_HD_2012.pdf.

- 8. Race/Ethnicity Categories
 - Asian person having origins in any of the original peoples of the Far East, Southeast Asia, the Indian subcontinent, or the Pacific Islands.
 - Black person having origins in any of the black racial groups of Africa.
 - Hispanic/Latino person of Mexican, Puerto Rican, Cuban, Central or South American, or other Spanish culture or origin, regardless of race.
 - White person having origins in any of the original peoples of Europe, North Africa, or the Middle East.
 - Other includes persons that do not list themselves according to any of the above categories and those that note multiple race/ethnicity categories.

Because population data is not available for unknown, other, or multiple race categories, rate calculations for these groups are not possible.

STANDARD REPORT FORMAT

- 1. Crude data
 - **Number of Cases**: For most diseases, this number reflects new cases of the disease with an onset in the year of the report. If the onset was unknown, the date of diagnosis was used as proxy for onset.
 - Annual Incidence Rates in LAC: Number of new cases in the year of report divided by LAC census population (minus Long Beach and Pasadena) multiplied by 100,000.
 - Annual Incidence Rates in the United States (US) and California: The 2015 incidence rates for the US and California can be found in the CDC's Morbidity and Mortality Weekly Report (MMWR): Final Summary of Nationally Notifiable Infectious Diseases. Previous incidence reports are available at the CDC's MMWR site.
 - Mean Age at Onset: Average age of all cases.
 - Median Age at Onset: The age that represents the midpoint of the sequence of all case ages.



• Range of Ages at Onset: Ages of the youngest and oldest cases in the year of the report. For cases under one year of age, less than one (<1) was used.

2. Description

This includes the causative agent, mode of transmission, common symptoms, potential severe outcomes, susceptible groups, and/or vaccine-preventability; and other significant information (e.g., prevention and control methods) related to the disease.

3. <u>Trends and Highlights</u>

This provides a synopsis or the highlights of disease activity in the year of the report. This section may highlight trends, seasonality, significance related age, sex, race/ethnicity, and/or location of the disease.

4. Table

This is a main table for each disease chapter that includes numbers of reported cases, percentage, and rates per 100,000 by age group, race/ethnicity, and SPA of the reporting year and four years prior to the reporting year. Disease rates for <19 cases are omitted as the rates are unreliable.

5. Figures

Figures include disease incidence rates of the Los Angeles County and/or California (CA) and/or US. Some diseases may not include CA or US rates as the jurisdiction does not maintain surveillance of that particular disease. In separate figures, incidence rates or percent cases are expressed by age group, race/ethnicity, SPA, and/or month of onset. Some disease chapters have other type of figures or tables depending on the significance of that particular disease (e.g., percent cases by serotype, vaccination rates). When stratified data are presented in figures and/or tables these following facts are to be considered.

- Seasonality: Number of cases that occurred during each month of the reporting year.
- Age: Annual rate of disease for individual age groups. Race-adjusted rates are presented for some diseases.
- Sex: Male-to-female rate ratio of cases.
- **Race/Ethnicity**: Annual rate of disease for the four major racial groups. Cases of unknown race are excluded; thus, race-specific rates may be underestimates. Age-adjusted rates are presented for some diseases.
- Location: Location presented most often is the health district or SPA of residence of cases. Note that "location" refers to address of case and do not accurately reflect site of disease acquisition. Age-adjusted rates by location are presented for some diseases.



Los Angeles County Demographic Data 2015

Table A. Los Angeles County* Population by Year, 2010–2015					
Year Population % change					
2010	9 223 225				
2011	9,259,218	0.4%			
2012	9,296,158	0.4%			
2013	9,404,275	1.16%			
2014	9,452,968	0.52%			
2015	9,571,766	1.26%			

* Does not include cities of Pasadena and Long Beach.

Table B. Los Angeles County* Population by Age Group, 2015				
Age (in years)	Population	%		
<1	108,120	1.1%		
1–4	484,997	5.1%		
5–14	1,211,382	12.7%		
15–34	2,828,124	29.5%		
35–44	1,323,119	13.8%		
45–54	1,316,913	13.8%		
55-64	1,105,908	11.5%		
65+	1,193,203	12.5%		
Total	9,571,766	100.0%		

* Does not include cities of Pasadena and Long Beach.

Table C. Los Angles County* population by sex, 2015				
Sex Population %				
Male	4,725,060	49.4%		
Female	4,846,706	50.6%		
Total	9,571,766	100.0%		

* Does not include cities of Pasadena and Long Beach.

Table D. Los Angles County* population by race, 2015						
Race	Race Population %					
Asian	1,394,323	14.6%				
Black	785,325	8.2%				
Latino	4,689,432	49.0%				
White	2,684,584	28.0%				
Other**	18,102	0.2%				
Total	9,571,766	100.0%				

* Does not include cities of Pasadena and Long Beach. ** Includes American Indian, Alaskan Native, Eskimo and

Aleut.



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Table E. Los Angles County* population by health district and SPA, 2015**		
Health District Population		
SPA1	396,357	
Antelope valley	396,357	
SPA 2	2,228,821	
East Valley	462,314	
Glendale	348,193	
San Fernando	522,224	
West Valley	896,090	
SPA 3	1,655,477	
Alhambra	351,016	
El Monte	443,802	
Foothill	311,318	
Pomona	549,341	
SPA 4	1,167,286	
Central	350,463	
Hollywood Wilshire	501,237	
Northeast	315,586	
SPA 5	660,081	
West	660,081	
SPA 6	1,048,734	
Compton	286,423	
South	197,529	
Southeast	179,002	
Southwest	385,780	
SPA 7	1,322,943	
Bellflower	361,318	
East Los Angeles	207,037	
San Antonio	429,229	
Whittier	325,359	
SPA 8	1,092,067	
Inglewood	420,120	
Harbor	208,754	
Torrance	463,193	
Total	9,571,766	

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* Pasadena and Long Beach are separate health jurisdictions and as such are excluded from this table.
 ** Using 2010 Census estimates.







	Table F. List of Acronyms			
95%CI	95 percent confidence interval	HCV	Hepatitis C virus	
ACDC	Acute Communicable Disease Control	HD	Health District	
AIDS	Acquired Immunodeficiency Syndrome	Hib	Haemophilus influenzae, type b	
ALT	Alanine aminotransferase	HIV	Human Immunodeficiency Virus	
AR	Attack rate	IFA	Immunofluorescent Antibody	
CA	California	lgG	Immunoglobulin G	
CDC	Centers for Disease Control and Prevention	lgM	Immunoglobulin M	
CDPH	California Department of Public Health	LAC	Los Angeles County	
CHS	Community Health Services	MMR	Mumps-Measles-Rubella vaccine	
CMR	Confidential morbidity report	MMWR	Morbidity and Mortality Weekly Report	
CSF	Cerebral spinal fluid	MSM	Men who have sex with men	
CSTE	Council of State and Territorial Epidemiologists	N/A	Not available	
DPH	Department of Public Health	OR	Odds ratio	
DTaP	Diphtheria-tetanus-acellular pertussis	PCP	Pneumocystis carinii pneumonia	
DTP	Diphtheria-tetanus-pertussis vaccine	PCR	Polymerase Chain Reaction	
EHS	Environmental Health Services	PFGE	Pulsed Field Gel Electrophoresis	
EIA	Enzyme Immunoassay	PHBPP	Perinatal Hepatitis B Prevention Program	
GI	Gastrointestinal	RNA	Ribonucleic Acid	
GE	Gastroenteritis	RR	Rate ratio or relative risk	
HAART	Highly Active Antiretroviral Therapy	SNF	Skilled nursing facility	
HAV	Hepatitis A virus	sp. or spp.	Species	
HBIG	Hepatitis B Immunoglobulin	SPA	Service Planning Area	
HBsAg	Hepatitis B surface antigen	US	United States	
HBV	Hepatitis B virus	vCMR	Visual confidential morbidity report (software)	

The following abbreviations and acronyms may be found throughout this report.

LOS ANGELES COUNTY HEALTH DISTRICTS

AH	Alhambra	FH	Foothill	SE	Southeast
AV	Antelope Valley	GL	Glendale	SF	San Fernando
BF	Bellflower	HB	Harbor	SO	South
CE	Central	HW	Hollywood/Wilshire	SW	Southwest
CN	Compton	IW	Inglewood	то	Torrance
EL	East Los Angeles	NE	Northeast	WE	West
EV	East Valley	PO	Pomona	WV	West Valley
EM	El Monte	SA	San Antonio	WH	Whittier



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Table G.	Reported Cases of Selected Notifiable Diseases by Year of Onset
	Los Angeles County, 2010-2015

							Previous	5-Yr 95%
			Y	ear of On	set		5-year	upper
Disease	2010	2011	2012	2013	2014	2015	Average	Limit ^a
Amebiasis	119	86	99	57	64	62	85	130
Botulism	1	3	4	4	1	2	3	5
Brucellosis	7	6	4	10	7	8	7	11
Campylobacteriosis	1239	1259	1546	1703	1506	1623	1451	1798
Cholera ^b	0	0	0	0	0	4	0	0
Coccidioidomycosis ^b	235	304	327	362	426	613	331	455
Cryptosporidiosis	61	51	44	48	78	56	56	80
Cysticercosis	3	37	11	1	9	12	12	38
Dengue	1	0	2	2	32	30	7	32
Encephalitis ^b	51	59	75	79	92	136	71	100
Foodborne Outbreaks	17	22	21	12	24	23	19	28
Giardiasis	308	292	294	392	346	379	326	401
Hansen's Disease (Leprosy)	2	2	3	1	3	0	2	4
Hepatitis A	51	45	47	60	42	33	49	61
Hepatitis B	54	60	38	55	42	50	50	66
Hepatitis C	4	10	7	5	5	2	6	10
Hepatitis Unspecified	5	4	0	0	0	0	2	6
Legionellosis ^b	108	116	111	85	140	171	112	146
Listeriosis, Nonperinatal ^b	14	19	26	23	27	34	22	31
Listeriosis, Perinatal	4	6	7	4	5	3	5	7
Lyme Disease	5	6	1	11	5	4	6	12
Malaria ^b	25	22	19	16	21	27	21	26
Meningitis, Viral	570	317	303	355	400	367	389	578
Meningococcal Infections	26	37	12	17	11	12	21	40
Pneumococcal Disease, Invasive ^c	576	658	504	522	460	468	544	677
Psittacosis	0	0	0	0	0	0	0	0
Q-fever	1	0	3	2	1	5	1	3
Relapsing Fever	0	0	0	0	1	0	0	1
Rheumatic Fever, Acute	1	0	0	0	0	0	0	1
Salmonellosis	1142	900	1041	1010	1141	1144	1047	1224
Shiga Toxin-Producing E. Coli ^b	67	88	97	102	90	175	89	112
Shigellosis ^b	355	264	306	227	350	508	300	397
Staphylococcus Aureus Infection	28	44	24	26	17	9	28	45
Streptococcus, Group A Invasive	191	175	168	195	222	227	190	227
Strongyloidiasis	0	0	0	11	35	9	9	36
Taeniasis	4	5	6	4	3	2	4	6
Trichinosis	0	0	0	0	0	0	0	0
Tularemia	0	0	0	0	0	0	0	0
Typhoid Fever, Case	15	15	6	17	15	14	14	21
Typhoid Fever, Carrier	4	3	0	0	0	0	1	5
Typhus Fever	31	38	50	68	44	54	46	71
Vibrio	13	19	29	26	52	43	28	54
West Nile Virus ^b	4	63	174	165	218	300	125	279

^aThe normal distribution assumption may not apply to some rare diseases.

^b2015 data over 95% upper limit.

^cby specimen collection date.



Table H. Annual Incidence Rates of Selected Notifiable Diseases by Year of Onset Los Angeles County, 2010-2015

		Annual	l Incidence R	ate (Cases p	oer 100,000) ^b	
Disease	2010	2011	2012	2013	2014	2015
Amebiasis	1.29	0.93	1.06	0.61	0.68	0.65
Botulism	0.01	0.03	0.04	0.04	0.01	0.02
Brucellosis	0.08	0.06	0.04	0.11	0.07	0.08
Campylobacteriosis	13.43	13.60	16.63	18.11	15.93	16.96
Cholera	-	-	-	-	-	0.04
Coccidioidomycosis	2.55	3.28	3.52	3.85	4.51	6.40
Cryptosporidiosis	0.66	0.55	0.47	0.51	0.83	0.59
Cysticercosis	0.03	0.40	0.12	0.01	0.10	0.13
Dengue	0.01	-	0.02	0.02	0.34	0.31
Encephalitis	0.55	0.64	0.81	0.84	0.97	1.42
Giardiasis	3.34	3.15	3.16	4.17	3.66	3.96
Hansen's Disease (Leprosy)	0.02	0.02	0.03	0.01	0.03	-
Hepatitis A	0.55	0.49	0.51	0.64	0.44	0.34
Hepatitis B	0.59	0.65	0.41	0.58	0.44	0.52
Hepatitis C	0.04	0.11	0.08	0.05	0.05	0.02
Hepatitis Unspecified	0.05	0.04	-	-	-	-
Legionellosis	1.17	1.25	1.19	0.90	1.48	1.79
Listeriosis, Nonperinatal	0.15	0.21	0.28	0.24	0.29	0.36
Listeriosis, Perinatal ^a	3.23	4.95	5.71	3.34	4.11	2.58
Lyme Disease	0.05	0.06	0.01	0.12	0.05	0.04
Malaria	0.27	0.24	0.20	0.17	0.22	0.28
Meningitis, Viral	6.18	3.42	3.26	3.77	4.23	3.83
Meningococcal Infections	0.28	0.40	0.13	0.18	0.12	0.13
Pneumococcal Disease, Invasive	6.25	7.11	5.42	5.55	4.87	4.89
Psittacosis	-	-	-	-	-	-
Q-fever	0.01	-	0.03	0.02	0.01	0.05
Relapsing Fever	-	-	-	-	0.01	-
Rheumatic Fever, Acute	0.01	-	-	-	-	-
Salmonellosis	12.38	9.72	11.20	10.74	12.07	11.95
Shiga Toxin-Producing E. Coli	0.73	0.95	1.04	1.08	0.95	1.83
Shigellosis	3.85	2.85	3.29	2.41	3.70	5.31
Staphylococcus Aureus Infection	0.30	0.48	0.26	0.28	0.18	0.09
Streptococcus, Group A Invasive	2.07	1.89	1.81	2.07	2.35	2.37
Strongyloidiasis	-	-	-	0.12	0.37	0.09
Taeniasis	0.04	0.05	0.06	0.04	0.03	0.02
Trichinosis	-	-	-	-	-	-
Tularemia	-	-	-	-	-	-
Typhoid Fever, Case	0.16	0.16	0.06	0.18	0.16	0.15
Typhoid Fever, Carrier	0.04	0.03	-	-	-	-
Typhus Fever	0.34	0.41	0.54	0.72	0.47	0.56
Vibrio	0.14	0.21	0.31	0.28	0.55	0.45
West Nile Virus	0.04	0.68	1.87	1.75	2.31	3.13

^aRates for perinatal listeriosis were calculated as cases per 100,000 live births.



Table I. Five –Year Average of Notifiable Diseases by Month of Onset Los Angeles County, 2011-2015

Disease	Jan	Feb	Mar	Apr	Мау	June	July	Aug	Sept	Oct	Nov	Dec	Total
Amebiasis	6.0	5.0	9.2	4.0	6.0	7.4	7.4	5.6	5.2	4.8	5.8	5.8	73.6
Botulism	0.4	-	-	-	0.4	0.2	0.2	-	0.2	0.2	0.4	0.2	2.2
Brucellosis	0.2	0.2	-	0.8	0.4	0.4	-	0.6	0.2	0.2	0.2	-	7.0
Campylobacteriosis	48.2	27.4	29.4	35.8	49.0	52.2	69.2	73.4	59.4	63.2	58.8	39.2	1526.4
Cholera	-	-	-	-	-	-	0.2	-	0.2	-	-	-	0.8
Coccidioidomycosis	39.0	30.8	28.0	29.0	32.4	37.0	45.8	33.6	31.4	35.4	31.2	32.8	406.4
Cryptosporidiosis	3.4	2.8	3.8	4.4	3.8	3.8	5.0	9.0	3.8	1.8	2.8	2.6	55.4
Cysticercosis	0.4	0.4	0.6	0.6	0.2	0.2	-	0.2	-	-	0.2	0.2	5.4
Dengue	1.0	0.8	0.4	0.6	0.8	0.8	1.8	0.8	1.2	1.8	0.6	2.4	13.2
Encephalitis	2.2	2.0	3.6	2.4	2.4	1.8	6.2	11.6	29.6	16.6	4.2	1.6	88.2
Giardiasis	26.8	24.4	24.8	27.0	27.2	24.8	30.6	33.8	35.0	26.4	25.6	25.6	340.6
Hansen's Disease (Leprosy) ^a	-	-	-	-	-	-	-	-	-	-	-	-	-
Hepatitis A	3.0	3.4	3.2	4.4	5.4	4.2	3.8	4.6	3.8	4.4	2.4	2.8	45.4
Hepatitis B	5.6	2.8	4.6	4.2	4.6	3.2	3.6	4.2	3.8	3.6	6.0	2.6	49.0
Hepatitis C	0.8	0.8	0.4	0.6	0.2	0.8	0.2	0.4	0.8	0.8	-	-	5.8
Hepatitis Unspecified	-	-	-	-	-	-	-	-	-	-	-	-	0.8
Legionellosis	10.8	9.2	11.4	10.4	10.2	7.6	12.0	8.0	10.2	9.8	8.8	16.2	124.6
Listeriosis, Nonperinatal	1.4	0.8	1.4	1.2	2.0	2.4	2.6	3.0	3.2	2.4	1.4	1.4	25.8
Listeriosis, Perinatal	0.8	0.6	-	0.2	0.2	-	0.2	0.6	1.2	0.6	-	0.2	5.0
Lyme Disease	0.2	0.2	0.4	0.4	0.8	1.2	1.6	1.6	0.2	0.2	0.2	0.2	7.8
Malaria ^a	-	-	-	-	-	-	-	-	-	-	-	-	-
Meningitis, Viral	15.8	15.0	16.4	18.6	21.6	17.6	28.2	42.8	59.4	43.0	22.6	16.8	348.4
Meningococcal Infections	2.4	1.8	2.0	2.8	1.4	1.0	0.8	1.4	0.8	0.4	0.8	2.2	17.8
Pneumococcal Disease, Invasive ^b	89.6	83.4	64.0	45.2	36.8	31.4	19.4	17.0	21.0	23.6	32.6	59.4	523.4
Psittacosis	-	-	-	-	-	-	-	-	-	-	-	-	-
Q-fever	-	-	-	0.6	-	-	-	-	-	-	-	-	2.2
Relapsing Fever	-	-	-	-	-	-	-	0.2	-	-	-	-	0.2
Rheumatic Fever, Acute	-	-	-	-	-	-	-	-	-	-	-	-	-
Salmonellosis	59.4	51.8	67.6	69.2	97.2	88.8	116.6	121.0	118.4	92.8	68.4	52.4	1047.2
Shiga Toxin-Producing E. Coli	4.6	5.8	6.6	9.8	9.8	10.6	12.2	14.2	12.6	10.6	5.4	4.8	110.4
Shigellosis	14.8	14.6	16.4	15.0	26.0	21.6	31.2	39.2	42.4	39.8	31.0	23.4	331.0
Staphylococcus Aureus Infection	2.8	3.0	2.2	1.2	1.2	1.4	1.0	3.4	2.4	1.8	1.6	1.6	24.0
Streptococcus, Group A Invasive	24.0	18.8	21.4	19.0	16.6	15.6	12.4	9.0	9.2	15.2	14.2	18.4	194.4
Strongyloidiasis ^a	-	-	-	-	-	-	-	-	-	-	-	-	-
Taeniasis ^a	-	-	-	-	-	-	-	-	-	-	-	-	-
Trichinosis	-	-	-	-	-	-	-	-	-	-	-	-	-
Tularemia	-	-	-	-	-	-	-	-	-	-	-	-	-
Typhoid Fever, Case	1.4	1.4	0.4	1.2	1.2	1.2	1.6	1.6	0.6	0.6	1.4	0.6	13.4
Typhoid Fever, Carrier	-	-	0.2	-	0.2	-	-	-	-	-	-	-	0.6
Typhus Fever	3.6	1.2	1.4	1.2	5.4	5.4	5.4	7.8	6.0	6.2	4.6	2.4	50.8
Vibrio	0.8	-	0.2	1.2	1.4	3.2	4.8	6.2	4.2	2.0	1.4	1.4	33.8
West Nile Virus	-	-	-	-	-	0.2	12.4	43.8	81.6	38.8	7.2	-	184.0

^aNot applicable.

^bSpecimen collection date.



Table J. Number of Cases of Selected Notifiable Diseases by Age GroupLos Angeles County, 2015

Disease	<1	1-4	5-14	15-34	35-44	45-54	55-64	65+	Total ^a
Amebiasis	0	2	4	20	10	10	12	4	62
Botulism	0	0	0	0	0	0	2	0	2
Brucellosis	0	0	0	2	1	2	1	2	8
Campylobacteriosis	23	115	138	525	210	197	176	233	1623
Cholera	0	0	1	1	0	2	0	0	4
Coccidioidomycosis	0	4	7	96	98	127	109	172	613
Cryptosporidiosis	0	2	5	25	9	6	6	3	56
Cysticercosis	0	0	0	3	5	2	2	0	12
Dengue	0	0	0	8	6	9	6	1	30
Encephalitis	0	1	7	5	6	16	14	87	136
Giardiasis	0	14	20	126	76	66	47	29	379
Hansen's Disease (Leprosy)	0	0	0	0	0	0	0	0	0
Hepatitis A	0	0	1	12	9	3	4	4	33
Hepatitis B	0	0	0	10	14	18	5	3	50
Hepatitis C	0	0	0	1	0	1	0	0	2
Hepatitis Unspecified	0	0	0	0	0	0	0	0	0
Legionellosis	0	0	0	9	11	14	31	106	171
Listeriosis, Nonperinatal	0	0	0	1	3	5	4	21	34
Listeriosis, Perinatal ^b	0	0	0	2	1	0	0	0	3
Lyme Disease	0	0	0	3	1	0	0	0	4
Malaria	0	1	1	10	3	2	5	5	27
Meningitis, Viral	41	2	51	101	38	41	42	51	367
Meningococcal Infections	0	0	0	4	1	3	1	3	12
Pneumococcal Disease, Invasive	5	27	18	33	31	58	103	193	468
Psittacosis	0	0	0	0	0	0	0	0	0
Q-fever	0	0	0	1	1	2	0	1	5
Relapsing Fever	0	0	0	0	0	0	0	0	0
Rheumatic Fever, Acute	0	0	0	0	0	0	0	0	0
Salmonellosis	60	116	148	297	123	124	105	171	1144
Shiga Toxin-Producing E. Coli	5	44	24	42	14	14	15	17	175
Shigellosis	0	38	52	178	84	80	36	40	508
Staphylococcus Aureus Infection	0	0	0	1	1	1	2	4	9
Streptococcus, Group A Invasive	1	7	16	29	25	43	37	68	227
Strongyloidiasis	0	0	0	0	0	2	5	1	9
Taeniasis	0	0	0	0	1	0	1	0	2
Trichinosis	0	0	0	0	0	0	0	0	0
Tularemia	0	0	0	0	0	0	0	0	0
Typhoid Fever, Case	0	3	2	7	0	0	1	1	14
Typhoid Fever, Carrier	0	0	0	0	0	0	0	0	0
Typhus Fever	0	1	2	10	8	18	9	6	54
Vibrio	0	0	1	18	7	6	4	7	43
West Nile Virus	0	0	3	34	28	41	53	141	300

^aTotals include cases with unknown age.

^bMother's age.



Table K. Incidence Rates of Selected Notifiable Diseases by Age Group Los Angeles County, 2015

Disease <1				Age-gro	oup Rates (Cases per	100,000) ^ь		
Amebiasis - 0.4 0.3 0.7 0.8 0.8 1.1 0.3 Botulism - - - - 0.2 - Brucellosis - - 0.1 0.1 0.2 0.1 0.2 Campylobacteriosis 21.3 23.7 11.4 18.6 15.9 15.0 15.9 19.5 Cholera - - 0.1 - - 0.2 - - Coccidioidomycosis - 0.8 0.6 3.4 7.4 9.6 9.9 14.4 Cryptosporidiosis - 0.4 0.4 0.9 0.7 0.5 0.5 0.3 Cysticercosis - - 0.1 0.4 0.2 0.2 - Dengue - - 0.3 0.5 0.7 0.5 0.1 Encephalitis - 0.2 0.6 0.2 0.5 1.2 1.3 7.3 Giardiasis - 2.9 1.7 4.5 5.7 5.0 4.2 <td< th=""><th>Disease</th><th><1</th><th>1-4</th><th>5-14</th><th>15-34</th><th>35-44</th><th>45-54</th><th>55-64</th><th>65+</th></td<>	Disease	<1	1-4	5-14	15-34	35-44	45-54	55-64	65+
Botulism - - - - 0.1 0.1 0.2 - Brucellosis - - - 0.1 0.1 0.2 0.1 0.2 Campylobacteriosis 21.3 23.7 11.4 18.6 15.9 15.0 15.9 19.5 Cholera - - 0.1 - - 0.2 - - Coccidioidomycosis - 0.8 0.6 3.4 7.4 9.6 9.9 14.4 Cryptosporidiosis - 0.4 0.4 0.9 0.7 0.5 0.5 0.3 Cysticercosis - - 0.1 0.4 0.2 0.2 - Dengue - - 0.3 0.5 0.7 0.5 0.1 Encephalitis - 0.2 0.6 0.2 0.5 1.2 1.3 7.3 Giardiasis - 2.9 1.7 4.5 5.7 5.0 4.2 2.4 Hansen's Disease (Leprosy) - - - -	Amebiasis	-	0.4	0.3	0.7	0.8	0.8	1.1	0.3
Brucellosis0.10.10.20.10.2Campylobacteriosis21.323.711.418.615.915.015.919.5Cholera0.10.2Coccidioidomycosis-0.80.63.47.49.69.914.4Cryptosporidiosis-0.40.40.90.70.50.50.3Cysticercosis0.10.40.20.2-Dengue0.30.50.70.50.1Encephalitis-0.20.60.20.51.21.37.3Giardiasis-2.91.74.55.75.04.22.4Hansen's Disease (Leprosy)0.40.70.20.40.3Hepatitis B0.41.11.40.50.3Hepatitis C0.41.11.40.50.3Hepatitis UnspecifiedListeriosis, Nonperinatal0.20.40.41.8Listeriosis Perinatal ^a 0.20.41.8	Botulism	-	-	-	-	-	-	0.2	-
Campylobacteriosis21.323.711.418.615.915.015.919.5Cholera-0.10.2Coccidioidomycosis-0.80.63.47.49.69.914.4Cryptosporidiosis-0.40.40.90.70.50.50.3Cysticercosis0.10.40.20.2-Dengue0.30.50.70.50.1Encephalitis-0.20.60.20.51.21.37.3Giardiasis-2.91.74.55.75.04.22.4Hansen's Disease (Leprosy)Hepatitis A0.10.40.70.20.40.3Hepatitis C0.41.11.40.50.3Hepatitis UnspecifiedListeriosis, Nonperinatal0.20.40.41.8	Brucellosis	-	-	-	0.1	0.1	0.2	0.1	0.2
Cholera - - 0.1 - - 0.2 - - Coccidioidomycosis - 0.8 0.6 3.4 7.4 9.6 9.9 14.4 Cryptosporidiosis - 0.4 0.4 0.9 0.7 0.5 0.5 0.3 Cysticercosis - - 0.1 0.4 0.2 0.2 - Dengue - - 0.3 0.5 0.7 0.5 0.1 Encephalitis - 0.2 0.6 0.2 0.5 1.2 1.3 7.3 Giardiasis - 2.9 1.7 4.5 5.7 5.0 4.2 2.4 Hansen's Disease (Leprosy) - - - - - - - Hepatitis A - - 0.1 0.4 0.7 0.2 0.4 0.3 Hepatitis C - - - - - - - - Legionellosis - - - 0.3 0.8 1.1 <	Campylobacteriosis	21.3	23.7	11.4	18.6	15.9	15.0	15.9	19.5
Coccidioidomycosis - 0.8 0.6 3.4 7.4 9.6 9.9 14.4 Cryptosporidiosis - 0.4 0.4 0.9 0.7 0.5 0.5 0.3 Cysticercosis - - 0.1 0.4 0.2 0.2 - Dengue - - 0.3 0.5 0.7 0.5 0.1 Encephalitis - 0.2 0.6 0.2 0.5 1.2 1.3 7.3 Giardiasis - 2.9 1.7 4.5 5.7 5.0 4.2 2.4 Hansen's Disease (Leprosy) - - - - - - - Hepatitis A - - 0.1 0.4 0.7 0.2 0.4 0.3 Hepatitis C -	Cholera	-	-	0.1	-	-	0.2	-	-
Cryptosporidiosis - 0.4 0.4 0.9 0.7 0.5 0.5 0.3 Cysticercosis - - 0.1 0.4 0.2 0.2 - Dengue - - 0.3 0.5 0.7 0.5 0.1 Encephalitis - 0.2 0.6 0.2 0.5 1.2 1.3 7.3 Giardiasis - 2.9 1.7 4.5 5.7 5.0 4.2 2.4 Hansen's Disease (Leprosy) - - - - - - - Hepatitis A - - 0.1 0.4 0.7 0.2 0.4 0.3 Hepatitis B - - - 0.4 1.1 1.4 0.5 0.3 Hepatitis C - - - 0.4 1.1 1.4 0.5 0.3 Hepatitis Unspecified - - - - - - - - - - - - - - - - -	Coccidioidomycosis	-	0.8	0.6	3.4	7.4	9.6	9.9	14.4
Cysticercosis - - - 0.1 0.4 0.2 0.2 - Dengue - - 0.3 0.5 0.7 0.5 0.1 Encephalitis - 0.2 0.6 0.2 0.5 1.2 1.3 7.3 Giardiasis - 2.9 1.7 4.5 5.7 5.0 4.2 2.4 Hansen's Disease (Leprosy) - - - - - - - Hepatitis A - - 0.1 0.4 0.7 0.2 0.4 0.3 Hepatitis B - - - 0.4 1.1 1.4 0.5 0.3 Hepatitis C - - - - 0.1 - </td <td>Cryptosporidiosis</td> <td>-</td> <td>0.4</td> <td>0.4</td> <td>0.9</td> <td>0.7</td> <td>0.5</td> <td>0.5</td> <td>0.3</td>	Cryptosporidiosis	-	0.4	0.4	0.9	0.7	0.5	0.5	0.3
Dengue - - 0.3 0.5 0.7 0.5 0.1 Encephalitis - 0.2 0.6 0.2 0.5 1.2 1.3 7.3 Giardiasis - 2.9 1.7 4.5 5.7 5.0 4.2 2.4 Hansen's Disease (Leprosy) - - - - - - - Hepatitis A - - 0.1 0.4 0.7 0.2 0.4 0.3 Hepatitis B - - 0.1 0.4 0.7 0.2 0.4 0.3 Hepatitis C - - - 0.4 1.1 1.4 0.5 0.3 Hepatitis Unspecified -	Cysticercosis	-	-	-	0.1	0.4	0.2	0.2	-
Encephalitis - 0.2 0.6 0.2 0.5 1.2 1.3 7.3 Giardiasis - 2.9 1.7 4.5 5.7 5.0 4.2 2.4 Hansen's Disease (Leprosy) - - - - - - - Hepatitis A - - 0.1 0.4 0.7 0.2 0.4 0.3 Hepatitis B - - 0.1 0.4 1.1 1.4 0.5 0.3 Hepatitis C - - - 0.4 1.1 1.4 0.5 0.3 Hepatitis Unspecified - - - - - - - - Legionellosis - - - 0.3 0.8 1.1 2.8 8.9 Listeriosis, Nonperinatal - - - - 0.2 0.4 0.4 1.8	Dengue	-	-	-	0.3	0.5	0.7	0.5	0.1
Giardiasis - 2.9 1.7 4.5 5.7 5.0 4.2 2.4 Hansen's Disease (Leprosy) -	Encephalitis	-	0.2	0.6	0.2	0.5	1.2	1.3	7.3
Hansen's Disease (Leprosy) -	Giardiasis	-	2.9	1.7	4.5	5.7	5.0	4.2	2.4
Hepatitis A - - 0.1 0.4 0.7 0.2 0.4 0.3 Hepatitis B - - - 0.4 1.1 1.4 0.5 0.3 Hepatitis C - - - 0.4 1.1 1.4 0.5 0.3 Hepatitis C - - - - 0.1 - - Hepatitis Unspecified - - - - - - - Legionellosis - - - 0.3 0.8 1.1 2.8 8.9 Listeriosis, Nonperinatal - - - 0.2 0.4 0.4 1.8 Listeriosis Perinatal ^a - - - 2.2 3.7 - -	Hansen's Disease (Leprosy)	-	-	-	-	-	-	-	-
Hepatitis B - - 0.4 1.1 1.4 0.5 0.3 Hepatitis C - - - - 0.1 - - Hepatitis Unspecified - - - - 0.1 - - Legionellosis - - 0.3 0.8 1.1 2.8 8.9 Listeriosis, Nonperinatal - - - 0.2 0.4 0.4 1.8 Listeriosis, Perinatal ^a - - - 2.2 3.7 - -	Hepatitis A	-	-	0.1	0.4	0.7	0.2	0.4	0.3
Hepatitis C - - - - 0.1 - - Hepatitis Unspecified -	Hepatitis B	-	-	-	0.4	1.1	1.4	0.5	0.3
Hepatitis UnspecifiedLegionellosis0.30.81.12.88.9Listeriosis, Nonperinatal0.20.40.41.8Listeriosis, Perinatal ^a 2.23.7	Hepatitis C	-	-	-	-	-	0.1	-	-
Legionellosis - - 0.3 0.8 1.1 2.8 8.9 Listeriosis, Nonperinatal - - - 0.2 0.4 0.4 1.8 Listeriosis, Perinatal ^a - - - 2.2 3.7 - -	Hepatitis Unspecified	-	-	-	-	-	-	-	-
Listeriosis, Nonperinatal 0.2 0.4 0.4 1.8	Legionellosis	-	-	-	0.3	0.8	1.1	2.8	8.9
Listeriosis Perinatal ^a 2,2 3,7	Listeriosis, Nonperinatal	-	-	-	-	0.2	0.4	0.4	1.8
	Listeriosis, Perinatal ^a	-	-	-	2.2	3.7	-	-	-
Lyme Disease 0.1 0.1	Lyme Disease	-	-	-	0.1	0.1	-	-	-
Malaria - 0.2 0.1 0.4 0.2 0.2 0.5 0.4	Malaria	-	0.2	0.1	0.4	0.2	0.2	0.5	0.4
Meningitis, Viral 37.9 0.4 4.2 3.6 2.9 3.1 3.8 4.3	Meningitis, Viral	37.9	0.4	4.2	3.6	2.9	3.1	3.8	4.3
Meningococcal Infections 0.1 0.1 0.2 0.1 0.3	Meningococcal Infections	-	-	-	0.1	0.1	0.2	0.1	0.3
Pneumococcal Disease, Invasive 4.6 5.6 1.5 1.2 2.3 4.4 9.3 16.2	Pneumococcal Disease, Invasive	4.6	5.6	1.5	1.2	2.3	4.4	9.3	16.2
Psittacosis	Psittacosis	-	-	-	-	-	-	-	-
Q-fever 0.1 0.2 0.0 0.1	Q-fever	-	-	-	-	0.1	0.2	0.0	0.1
Relapsing Fever	Relapsing Fever	-	-	-	-	-	-	-	-
Rheumatic Fever, Acute	Rheumatic Fever, Acute	-	-	-	-	-	-	-	-
Salmonellosis 55.5 23.9 12.2 10.5 9.3 9.4 9.5 14.3	Salmonellosis	55.5	23.9	12.2	10.5	9.3	9.4	9.5	14.3
Shiga Toxin-Producing E. Coli 4.6 9.1 2.0 1.5 1.1 1.1 1.4 1.4	Shiga Toxin-Producing E. Coli	4.6	9.1	2.0	1.5	1.1	1.1	1.4	1.4
Shigellosis - 7.8 4.3 6.3 6.1 3.3 3.4	Shigellosis	-	7.8	4.3	6.3	6.3	6.1	3.3	3.4
Staphylococcus Aureus Infection 0.1 0.1 0.2 0.3	Staphylococcus Aureus Infection	-	-	-	-	0.1	0.1	0.2	0.3
Streptococcus, Group A Invasive 0.9 1.4 1.3 1.0 1.9 3.3 3.3 5.7	Streptococcus, Group A Invasive	0.9	1.4	1.3	1.0	1.9	3.3	3.3	5.7
Strongyloidiasis 0.2 0.5 0.1	Strongyloidiasis	-	-	-	-	-	0.2	0.5	0.1
Taeniasis 0.1 - 0.1 -	Taeniasis	-	-	-	-	0.1	-	0.1	-
Trichinosis	Trichinosis	-	-	-	-	-	-	-	-
Tularemia	Tularemia	-	-	-	-	-	-	-	-
Typhoid Fever, Case - 0.6 0.2 0.2 0.1 0.1	Typhoid Fever, Case	-	0.6	0.2	0.2	-	-	0.1	0.1
Typhoid Fever, Carrier	Typhoid Fever, Carrier	-	-	-	-	-	-	-	-
Typhus Fever - 0.2 0.2 0.4 0.6 1.4 0.8 0.5	Typhus Fever	-	0.2	0.2	0.4	0.6	1.4	0.8	0.5
Vibrio 0.1 0.6 0.5 0.5 0.4 0.6	Vibrio	-	-	0.1	0.6	0.5	0.5	0.4	0.6
West Nile Virus 0.2 1.2 2.1 3.1 4.8 11.8	West Nile Virus	-	-	0.2	1.2	2.1	3.1	4.8	11.8

^aRates for perinatal listeriosis were calculated as cases per 100,000 live births.



Table L.	Number of Cases of Selected Notifiable Diseases by Race/Ethnicity
	Los Angeles County, 2015

Disease	Asian	Black	Hispanic	White	Other ^a	Unknown
Amebiasis	4	4	16	37	0	1
Botulism	0	0	0	0	0	2
Brucellosis	0	0	3	1	0	4
Campylobacteriosis	43	25	210	264	39	1042
Cholera	0	0	1	0	0	3
Coccidioidomycosis	47	111	201	217	13	24
Cryptosporidiosis	4	2	16	21	0	13
Cysticercosis	0	0	12	0	0	0
Dengue	4	0	16	3	2	5
Encephalitis	4	3	51	62	1	15
Giardiasis	17	14	104	238	4	2
Hansen's Disease (Leprosy)	0	0	0	0	0	0
Hepatitis A	11	1	11	9	1	0
Hepatitis B	5	9	17	17	0	2
Hepatitis C	0	0	2	0	0	0
Hepatitis Unspecified	0	0	0	0	0	0
Legionellosis	11	29	49	76	3	3
Listeriosis, Nonperinatal	6	0	9	13	1	5
Listeriosis, Perinatal ^b	0	0	2	1	0	0
Lyme Disease	0	0	1	2	0	1
Malaria	3	12	3	3	0	6
Meningitis, Viral	21	24	174	106	8	34
Meningococcal Infections	0	2	6	4	0	0
Pneumococcal Disease, Invasive	29	87	132	119	14	87
Psittacosis	0	0	0	0	0	0
Q-fever	0	0	1	0	0	4
Relapsing Fever	0	0	0	0	0	0
Rheumatic Fever, Acute	0	0	0	0	0	0
Salmonellosis	102	68	589	383	2	0
Shiga Toxin-Producing E. Coli	13	11	72	74	2	3
Shigellosis	17	60	213	215	3	0
Staphylococcus Aureus Infection	0	1	3	3	0	2
Streptococcus, Group A Invasive	5	14	29	52	3	124
Strongyloidiasis	0	0	6	1	0	2
Taeniasis	0	1	0	0	0	1
Trichinosis	0	0	0	0	0	0
Tularemia	0	0	0	0	0	0
Typhoid Fever, Case	8	0	4	2	0	0
Typhoid Fever, Carrier	0	0	0	0	0	0
Typhus Fever	3	4	20	24	1	2
Vibrio	2	1	8	14	1	17
West Nile Virus	7	5	110	142	1	35

^aOther includes Native American and any additional racial group that cannot be categorized as Asian, Black, Hispanic, and White.

^bMother's race.



Table M.	Incidence Rates of Selected	Notifiable Diseases by Race/Ethnicity
	Los Angeles	County, 2015

	Rad	(Cases per 100,000)	b	
Disease	Asian	Black	Hispanic	White
Amebiasis	0.3	0.5	0.3	1.4
Botulism	-	-	-	-
Brucellosis	-	-	0.1	-
Campylobacteriosis	3.1	3.2	4.5	9.8
Cholera	-	-	-	-
Coccidioidomycosis	3.4	14.1	4.3	8.1
Cryptosporidiosis	0.3	0.3	0.3	0.8
Cysticercosis	-	-	0.3	-
Dengue	0.3	-	0.3	0.1
Encephalitis	0.3	0.4	1.1	2.3
Giardiasis	1.2	1.8	2.2	8.9
Hansen's Disease (Leprosy)	-	-	-	-
Hepatitis A	0.8	0.1	0.2	0.3
Hepatitis B	0.4	1.1	0.4	0.6
Hepatitis C	-	-	-	-
Hepatitis Unspecified	-	-	-	-
Legionellosis	0.8	3.7	1.0	2.8
Listeriosis, Nonperinatal	0.4	-	0.2	0.5
Listeriosis, Perinatal ^a	-	-	3.4	4.5
Lyme Disease	-	-	-	0.1
Malaria	0.2	1.5	0.1	0.1
Meningitis. Viral	1.5	3.1	3.7	3.9
Meningococcal Infections	-	0.3	0.1	0.1
Pneumococcal Disease. Invasive	2.1	11.1	2.8	4.4
Psittacosis	-	-	-	-
Q-fever	-	-	-	-
Relapsing Fever	-	-	-	-
Rheumatic Fever, Acute	-	-	-	-
Salmonellosis	7.3	8.7	12.6	14.3
Shiga Toxin-Producing E. Coli	0.9	1.4	1.5	2.8
Shigellosis	1.2	7.6	4.5	8.0
Staphylococcus Aureus Infection	-	0.1	0.1	0.1
Streptococcus, Group A Invasive	0.4	1.8	0.6	1.9
Strongyloidiasis	-	-	0.1	-
Taeniasis	-	0.1	-	-
Trichinosis	-	-	-	-
Tularemia	-	-	-	-
Typhoid Fever, Case	0.6	-	0.1	0.1
Typhoid Fever, Carrier	-	-	-	-
Typhus Fever	0.2	0.5	0.4	0.9
Vibrio	0.1	0.1	0.2	0.5
West Nile Virus	0.5	0.6	2.3	5.3

^aRates for perinatal listeriosis were calculated as cases per 100,000 live births.



Table N. Number of Cases and Annual Incidence Rate of Selected Notifiable Diseases by Sex Los Angeles County, 2015

	Mal	e	Fema	ale
Disease	Cases	Rate (Cases per 100,000) ^b	Cases	Rate (Cases per 100,000) ^b
Amebiasis	46	1.0	16	0.3
Botulism	2	0.0	0	-
Brucellosis	5	0.1	3	0.1
Campylobacteriosis	854	18.1	759	15.7
Cholera	4	0.1	0	-
Coccidioidomycosis	393	8.3	220	4.5
Cryptosporidiosis	37	0.8	16	0.3
Cysticercosis	7	0.1	2	0.0
Dengue	12	0.3	18	0.4
Encephalitis	91	1.9	45	0.9
Giardiasis	276	5.8	103	2.1
Hansen's Disease (Leprosy)	0	-	0	-
Hepatitis A	15	0.3	18	0.4
Hepatitis B	39	0.8	11	0.2
Hepatitis C	1	0.0	1	0.0
Hepatitis Unspecified	0	-	0	-
Legionellosis	88	1.9	83	1.7
Listeriosis, Nonperinatal	12	0.3	20	0.4
Listeriosis, Perinatal ^a	0	-	2	3.5
Lyme Disease	3	0.1	1	0.0
Malaria	17	0.4	10	0.2
Meningitis, Viral	211	4.5	153	3.2
Meningococcal Infections	7	0.1	5	0.1
Pneumococcal Disease, Invasive	265	5.6	203	4.2
Psittacosis	0	-	0	-
Q-fever	5	0.1	0	-
Relapsing Fever	0	-	0	-
Rheumatic Fever, Acute	0	-	0	-
Salmonellosis	545	11.5	599	12.4
Shiga Toxin-Producing E. Coli	70	1.5	99	2.0
Shigellosis	339	7.2	169	3.5
Staphylococcus Aureus Infection	6	0.1	3	0.1
Streptococcus, Group A Invasive	123	2.6	87	1.8
Strongyloidiasis	2	0.0	6	0.1
Taeniasis	1	0.0	0	-
Trichinosis	0	-	0	-
Tularemia	0	-	0	-
Typhoid Fever, Case	7	0.1	7	0.1
Typhoid Fever, Carrier	0	-	0	-
Typhus Fever	29	0.6	25	0.5
Vibrio	28	0.6	14	0.3
West Nile Virus	198	4.2	102	2.1

^aRates for perinatal listeriosis were calculated as cases per 100,000 live births.



Table O-1. Selected Notifiable Diseases SPA 1. Antelope Valley Area Los Angeles County, 2015

	Frequency	Rate (Cases per 100,000) ^b
Disease	Antelope	Antelope
Amebiasis	0	-
Botulism	0	-
Brucellosis	0	-
Campylobacteriosis	66	16.7
Cholera	0	-
Coccidioidomycosis	169	42.6
Cryptosporidiosis	0	-
Cysticercosis	0	-
Dengue	3	0.8
Encephalitis	4	1.0
Giardiasis	9	2.3
Hansen's Disease (Leprosy)	0	-
Hepatitis A	0	-
Hepatitis B	2	0.5
Hepatitis C	0	-
Hepatitis Unspecified	0	-
Legionellosis	4	1.0
Listeriosis, Nonperinatal	0	-
Listeriosis, Perinatal ^a	0	-
Lyme Disease	1	0.3
Malaria	1	0.3
Meningitis, Viral	27	6.8
Meningococcal Infections	1	0.3
Pneumococcal Disease. Invasive	18	4.5
Psittacosis	0	-
Q-fever	0	-
Relapsing Fever	0	-
Rheumatic Fever, Acute	0	-
Salmonellosis	35	8.8
Shiga Toxin-Producing E. Coli	4	1.0
Shigellosis	4	1.0
Staphylococcus Aureus Infection	0	-
Streptococcus, Group A Invasive	4	1.0
Strongyloidiasis	0	-
Taeniasis	0	-
Trichinosis	0	-
Tularemia	0	-
Typhoid Fever, Case	0	-
Typhoid Fever, Carrier	0	-
Typhus Fever	0	-
Vibrio	2	0.5
West Nile Virus	4	1.0

^aRates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.


Table O-2.Selected Notifiable DiseasesSPA 2.San Fernando AreaLos Angeles County, 2015

-		Fre	equency	,		Rate (Cases per 100,000) ^b								
Disease	EV	GL	SF	wv	TOTAL		EV	GL	SF	wv	TOTAL			
Amebiasis	8	1	2	5	16		1.7	0.3	0.4	0.6	0.7			
Botulism	0	0	2	0	2		-	-	0.4	-	0.1			
Brucellosis	0	0	0	0	0		-	-	-	-	-			
Campylobacteriosis	87	56	87	186	416		18.8	16.1	16.7	20.8	18.7			
Cholera	0	1	1	1	3		-	0.3	0.2	0.1	0.1			
Coccidioidomycosis	24	13	65	55	157		5.2	3.7	12.4	6.1	7.0			
Cryptosporidiosis	4	1	14	5	24		0.9	0.3	2.7	0.6	1.1			
Cysticercosis	1	0	1	2	4		0.2	-	0.2	0.2	0.2			
Dengue	3	2	2	5	12		0.6	0.6	0.4	0.6	0.5			
Encephalitis	15	18	6	13	52		3.2	5.2	1.1	1.5	2.3			
Giardiasis	14	7	13	33	67		3.0	2.0	2.5	3.7	3.0			
Hansen's Disease (Leprosy)	0	0	0	0	0		-	-	-	-	-			
Hepatitis A	3	1	2	2	8		0.6	0.3	0.4	0.2	0.4			
Hepatitis B	1	2	0	11	14		0.2	0.6	-	1.2	0.6			
Hepatitis C	0	0	1	0	1		-	-	0.2	-	0.0			
Hepatitis Unspecified	0	0	0	0	0		-	-	-	-	-			
Legionellosis	6	6	10	16	38		1.3	1.7	1.9	1.8	1.7			
Listeriosis, Nonperinatal	0	3	0	5	8		-	0.9	-	0.6	0.4			
Listeriosis, Perinatal ^a	0	0	0	0	0		-	-	-	-	-			
Lyme Disease	0	0	0	1	1		-	-	-	0.1	0.0			
Malaria	1	1	1	3	6		0.2	0.3	0.2	0.3	0.3			
Meningitis, Viral	18	11	19	20	68		3.9	3.2	3.6	2.2	3.1			
Meningococcal Infections	0	1	1	2	4		-	0.3	0.2	0.2	0.2			
Pneumococcal Disease, Invasive	14	7	15	36	72		3.0	2.0	2.9	4.0	3.2			
Psittacosis	0	0	0	0	0		-	-	-	-	-			
Q-fever	0	0	0	1	1		-	-	-	0.1	0.0			
Relapsing Fever	0	0	0	0	0		-	-	-	-	-			
Rheumatic Fever, Acute	0	0	0	0	0		-	-	-	-	-			
Salmonellosis	62	42	47	113	264		13.4	12.1	9.0	12.6	11.8			
Shiga Toxin-Producing E. Coli	10	5	14	13	42		2.2	1.4	2.7	1.5	1.9			
Shigellosis	27	7	12	28	74		5.8	2.0	2.3	3.1	3.3			
Staphylococcus Aureus Infection	0	1	0	0	1		-	0.3	-	-	0.0			
Streptococcus, Group A Invasive	9	9	12	24	54		1.9	2.6	2.3	2.7	2.4			
Strongyloidiasis	1	0	0	0	1		0.2	-	-	-	0.0			
Taeniasis	1	0	1	0	2		0.2	-	0.2	-	0.1			
Trichinosis	0	0	0	0	0		-	-	-	-	-			
Tularemia	0	0	0	0	0		-	-	-	-	-			
Typhoid Fever, Case	2	1	0	4	7		0.4	0.3	-	0.4	0.3			
Typhoid Fever, Carrier	0	0	0	0	0		-	-	-	-	-			
Typhus Fever	2	5	2	1	10		0.4	1.4	0.4	0.1	0.4			
Vibrio	0	1	2	8	11		-	0.3	0.4	0.9	0.5			
West Nile Virus	32	31	8	21	92		6.9	8.9	1.5	2.3	4.1			

^aRates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.



Table O-3. Selected Notifiable Diseases SPA 3. San Gabriel Area Los Angeles County, 2015

_			Frequer	ncy		Rate (Cases per 100,000) ^b							
Disease	AH	EM	FH	PO	TOTAL	АН	EM	FH	PO	TOTAL			
Amebiasis	1	2	0	0	3	0.3	0.5	-	-	0.2			
Botulism	0	0	0	0	0	-	-	-	-	-			
Brucellosis	0	1	0	1	2	-	0.2	-	0.2	0.1			
Campylobacteriosis	46	54	45	72	217	13.1	12.2	14.5	13.1	13.1			
Cholera	0	0	0	0	0	-	-	-	-	-			
Coccidioidomycosis	8	9	3	16	36	2.3	2.0	1.0	2.9	2.2			
Cryptosporidiosis	2	0	1	4	7	0.6	-	0.3	0.7	0.4			
Cysticercosis	0	1	0	0	1	-	0.2	-	-	0.1			
Dengue	1	1	1	1	4	0.3	0.2	0.3	0.2	0.2			
Encephalitis	2	3	4	10	19	0.6	0.7	1.3	1.8	1.1			
Giardiasis	10	4	8	12	34	2.8	0.9	2.6	2.2	2.1			
Hansen's Disease (Leprosy)	0	0	0	0	0	-	-	-	-	-			
Hepatitis A	1	1	0	3	5	0.3	0.2	0.0	0.5	0.3			
Hepatitis B	1	1	3	1	6	0.3	0.2	1.0	0.2	0.4			
Hepatitis C	0	0	0	0	0	-	-	-	-	-			
Hepatitis Unspecified	0	0	0	0	0	-	-	-	-	-			
Legionellosis	4	5	7	6	22	1.1	1.1	2.2	1.1	1.3			
Listeriosis, Nonperinatal	1	2	4	3	10	0.3	0.5	1.3	0.5	0.6			
Listeriosis, Perinatal ^a	0	0	1	0	1	-	-	0.8	-	0.1			
Lyme Disease	0	0	0	0	0	-	-	-	-	-			
Malaria	0	0	1	1	2	-	-	0.3	0.2	0.1			
Meningitis, Viral	7	12	20	32	71	2.0	2.7	6.4	5.8	4.3			
Meningococcal Infections	0	0	0	0	0	-	-	-	-	-			
Pneumococcal Disease, Invasive	14	18	13	19	64	4.0	4.1	4.2	3.5	3.9			
Psittacosis	0	0	0	0	0	-	-	-	-	-			
Q-fever	0	1	0	0	1	-	0.2	-	-	0.1			
Relapsing Fever	0	0	0	0	0	-	-	-	-	-			
Rheumatic Fever, Acute	0	0	0	0	0	-	-	-	-	-			
Salmonellosis	43	47	36	70	196	12.3	10.6	11.6	12.7	11.8			
Shiga Toxin-Producing E. Coli	3	4	3	9	19	0.9	0.9	1.0	1.6	1.1			
Shigellosis	6	13	5	9	33	1.7	2.9	1.6	1.6	2.0			
Staphylococcus Aureus Infection	0	0	2	0	2	-	-	0.6	-	0.1			
Streptococcus, Group A Invasive	4	10	7	10	31	1.1	2.3	2.2	1.8	1.9			
Strongyloidiasis	1	0	0	0	1	0.3	-	-	-	0.1			
Taeniasis	0	0	0	0	0	-	-	-	-	-			
Trichinosis	0	0	0	0	0	-	-	-	-	-			
Tularemia	0	0	0	0	0	-	-	-	-	-			
Typhoid Fever, Case	1	0	0	1	2	0.3	-	-	0.2	0.1			
Typhoid Fever, Carrier	0	0	0	0	0	-	-	-	-	-			
Typhus Fever	7	3	3	9	22	2.0	0.7	1.0	1.6	1.3			
Vibrio	0	1	3	1	5	-	0.2	1.0	0.2	0.3			
West Nile Virus	5	7	12	22	46	1.4	1.6	3.9	4.0	2.8			

^aRates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.



Table O-4. Selected Notifiable Diseases SPA 4. Metro Area Los Angeles County, 2015

_		Freque	ency		Rate (Cases per 100,000)⁵							
Disease	CE	нพ	NE	TOTAL	CE	нพ	NE	TOTAL				
Amebiasis	6	13	3	22	1.7	2.6	1.0	1.9				
Botulism	0	0	0	0	-	-	-	-				
Brucellosis	0	1	1	2	-	0.2	0.3	0.2				
Campylobacteriosis	49	133	48	230	14.0	26.5	15.2	19.7				
Cholera	0	1	0	1	-	0.2	-	0.1				
Coccidioidomycosis	20	21	16	57	5.7	4.2	5.1	4.9				
Cryptosporidiosis	1	7	0	8	0.3	1.4	-	0.7				
Cysticercosis	2	0	0	2	0.6	-	-	0.2				
Dengue	1	0	2	3	0.3	-	0.6	0.3				
Encephalitis	3	9	2	14	0.9	1.8	0.6	1.2				
Giardiasis	22	83	5	110	6.3	16.6	1.6	9.4				
Hansen's Disease (Leprosy)	0	0	0	0	-	-	-	-				
Hepatitis A	2	7	0	9	0.6	1.4	-	0.8				
Hepatitis B	1	4	1	6	0.3	0.8	0.3	0.5				
Hepatitis C	0	0	0	0	-	-	-	-				
Hepatitis Unspecified	0	0	0	0	-	-	-	-				
Legionellosis	8	11	4	23	2.3	2.2	1.3	2.0				
Listeriosis, Nonperinatal	2	1	2	5	0.6	0.2	0.6	0.4				
Listeriosis, Perinatal ^a	0	0	0	0	-	-	-	-				
Lyme Disease	0	1	0	1	-	0.2	-	0.1				
Malaria	0	1	5	6	-	0.2	1.6	0.5				
Meningitis, Viral	6	9	16	31	1.7	1.8	5.1	2.7				
Meningococcal Infections	1	1	1	3	0.3	0.2	0.3	0.3				
Pneumococcal Disease, Invasive	30	27	12	69	8.6	5.4	3.8	5.9				
Psittacosis	0	0	0	0	-	-	-	-				
Q-fever	0	0	1	1	-	-	0.3	0.1				
Relapsing Fever	0	0	0	0	-	-	-	-				
Rheumatic Fever, Acute	0	0	0	0	-	-	-	-				
Salmonellosis	36	58	37	131	10.3	11.6	11.7	11.2				
Shiga Toxin-Producing E. Coli	4	18	4	26	1.1	3.6	1.3	2.2				
Shigellosis	40	131	23	164	11.4	20.2	7.3	14.0				
Staphylococcus Aureus Infection	0	1	1	2	-	0.2	0.3	0.2				
Streptococcus, Group A Invasive	14	16	4	34	4.0	3.2	1.3	2.9				
Strongyloidiasis	1	0	2	3	0.3	-	0.6	0.3				
Taeniasis	0	0	0	0	-	-	-	-				
Trichinosis	0	0	0	0	-	-	-	-				
Tularemia	0	0	0	0	-	-	-	-				
Typhoid Fever, Case	1	1	2	4	0.3	0.2	0.6	0.3				
Typhoid Fever, Carrier	0	0	0	0	-	-	-	-				
Typhus Fever	3	2	3	8	0.9	0.4	1.0	0.7				
Vibrio	3	0	1	4	0.9	-	0.3	0.3				
West Nile Virus	8	20	13	41	2.3	4.0	4.1	3.5				

^aRates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.



Table O-5. Selected Notifiable Diseases SPA 5. West Area Los Angeles County, 2015

	Frequency	Rate (Cases per 100,000) ^b
Disease	West	West
Amebiasis	14	2.1
Botulism	0	-
Brucellosis	0	-
Campylobacteriosis	219	33.2
Cholera	0	-
Coccidioidomycosis	25	3.8
Cryptosporidiosis	4	0.6
Cysticercosis	0	-
Dengue	4	0.6
Encephalitis	11	1.7
Giardiasis	77	11.7
Hansen's Disease (Leprosy)	0	-
Hepatitis A	3	0.5
Hepatitis B	1	0.2
Hepatitis C	0	-
Hepatitis Unspecified	ů 0	-
l egionellosis	16	24
Listeriosis Nonnerinatal	3	0.5
Listoriosia, Poripotala	0	-
	1	0.0
Lyme Disease	1	0.2
Maningitia Viral	1	0.2
Meninguis, virai	20	3.0
Meningococcal Infections		0.2
Prieumococcal Disease, invasive	20	3.9
Psillacosis	0	-
Q-lever Delensing Four	0	-
Relapsing Fever	0	-
Rheumatic Fever, Acute	0	-
Salmonellosis	114	17.3
Shiga Toxin-Producing E. Coll	31	4.7
Shigellosis Stanbulassasua Auraua Infaction	78	11.8
Staphylococcus Aureus Intection	0	-
Streptococcus, Group A Invasive	15	2.3
Strongyloidiasis	0	-
l aeniasis Triching dia	0	-
Trilanansia	0	-
	0	-
Typnola Fever, Case	1	0.2
i yphoid Fever, Carrier	0	-
i ypnus Fever	1	0.2
	7	1.1
West Nile Virus	30	4.5

^aRates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.



Table O-6. Selected Notifiable Diseases SPA 6. South Area Los Angeles County, 2015

		F	requenc	у		Rate (Cases per 100,000) ^b							
Disease	CN	SO	SE	SW	TOTAL	c	N SC) SE	SW	TOTAL			
Amebiasis	1	1	1	1	4	0	3 0.	5 0.6	0.3	0.4			
Botulism	0	0	0	0	0		-		-	-			
Brucellosis	2	0	0	0	2	0	7		-	0.2			
Campylobacteriosis	37	30	28	43	138	12	9 15.	2 15.6	11.1	13.2			
Cholera	0	0	0	0	0		-		-	-			
Coccidioidomycosis	18	10	7	22	57	6	3 5.	1 3.9	5.7	5.4			
Cryptosporidiosis	0	0	0	5	5		-		1.3	0.5			
Cysticercosis	0	0	2	1	3		-	- 1.1	0.3	0.3			
Dengue	0	0	0	2	2		-		0.5	0.2			
Encephalitis	1	2	0	0	3	0	1 1.	0 -	-	0.3			
Giardiasis	3	4	5	10	22	1	0 2.	0 2.8	2.6	2.1			
Hansen's Disease (Leprosy)	0	0	0	0	0		-		-	-			
Hepatitis A	1	0	0	0	1	0	3		-	0.1			
Hepatitis B	2	2	0	3	7	0	7 1.	0 -	0.8	0.7			
Hepatitis C	0	0	0	0	0		-		-	-			
Hepatitis Unspecified	Ō	Ō	Ō	Ō	0		-		-	-			
Legionellosis	9	2	1	7	19	3	1 1.	0.6	1.8	1.8			
Listeriosis, Nonperinatal	1	0	1	0	2	0	3	- 0.6	-	0.2			
Listeriosis, Perinatal ^a	0 0	Õ	Ō	1	1		-		0.6	0.2			
Listenesis, i ennatar	0	0	0	0	0		_		_	_			
Malaria	0	0	0	3	3		-		0.8	03			
Meningitis Viral	17	3	4	10	43	5	- 0 1	 5 22	1.0	0.3			
Meningacoccal Infections	1	0	-	13	+3	0	3 1.		4.9				
Proumococcal Disease Invasive	15	16	13	33	77	5	2 2 8	 1 73	0.5 8.6	73			
Psittacosis	0	0	0	0	,,	5	2 0.		0.0	1.5			
O-fever	0	0	1	1	2		-	- 06	03	0.2			
	0	0	0	0	0		_	- 0.0	0.5	0.2			
Phoumatic Fover Acute	0	0	0	0	0		-						
Salmonellosis	27	16	26	58	127	Q	- 1 8	 1 1/15	15.0	12.1			
Shiga Toxin-Producing F. Coli	5	10	20	2	10	1	7 0. 7 0	5 11	0.5	10			
	13	7	13	23	56	1	7 0. 5 3	5 73	0.5 6.0	5.3			
Staphylococcus Aureus Infection	0	0	0	23	1		5 5.		0.0	0.1			
Steptylococcus Adreus Intection	6	3	6	1/	20	2	- 1	5 31	0.5	2.0			
Strongyloidiasis	0	0	0	14	29	2			0.3	2.0			
	0	0	0	0	0		-		0.5	0.1			
Trichinosis	0	0	0	0	0		-		-	-			
Tularamia	0	0	0	0	0		-						
Tunboid Fovor Caso	0	0	0	0	0		-		-	-			
Typhoid Fever, Case	0	0	0	0	0		-		-	-			
Typhus Favor	0	0	0	0	0		_		-	-			
Vibrio	1	1	2	0	4	^	3 0		-	-			
West Nile Virus	4	5	2	⊿	4 15	1	ου. Δ 2	5 1.1	10	0.4			
	4	5	2	4	10		4 2.	J I.I	1.0	1.4			

^aRates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.



Table O-7. Selected Notifiable Diseases SPA 7. East Area Los Angeles County, 2015

Disease BF EL SA WH TOTAL BF EL SA WH TOTAL Amebiasis 0 1 0 0 1 - 0.5 0.7 Botulism 0 0 0 0 0 0.7 Brucellosis 0 1 0 1 2 0.5 0.3 0.2 Campylobacteriosis 32 31 57 45 165 8.9 15.0 13.3 13.8 12.5 Cholera 0 0 0 0 0 - - - - - Coccidioidomycosis 19 11 23 11 64 5.3 5.3 5.4 3.4 4.8 Cryptosporidiosis 1 2 0 0 3 0.3 1.0 - - 0.2 Dengue 0	
Amebiasis 0 1 0 0 1 - 0.5 - - 0.7 Botulism 0 0 0 0 0 0 - <th>Disease</th>	Disease
Botulism 0 0 0 0 0 0 -<	Amebiasis
Brucellosis 0 1 0 1 2 - 0.5 - 0.3 0.2 Campylobacteriosis 32 31 57 45 165 8.9 15.0 13.3 13.8 12.8 Cholera 0 0 0 0 0 - 0.2 - 0.2 - 0.2 - 0.2 - 0.2 - 0.2 - 0.2 - 0.2 - 0.2 - 0.2 - 0.2 - 0.2 0.2 0.2 0.2	Botulism
Campylobacteriosis 32 31 57 45 165 8.9 15.0 13.3 13.8 12.8 Cholera 0 0 0 0 0 0 0 - 0.2 - 0.2 </td <td>Brucellosis</td>	Brucellosis
Cholera 0 0 0 0 0 0 - 0.2 2 0 3 1.0 - - 0.2 0.2 - 0.2 0.2 - 0.2 0.2 - 0.2	Campylobacteriosis
Coccidioidomycosis 19 11 23 11 64 5.3 5.3 5.4 3.4 4.8 Cryptosporidiosis 1 2 0 0 3 0.3 1.0 - - 0.2 Cysticercosis 0 1 1 0 2 - 0.5 0.2 - 0.2 Dengue 0 0 0 1 1 - - - 0.3 0.4 Encephalitis 7 5 7 7 26 1.9 2.4 1.6 2.2 2.0	Cholera
Cryptosporidiosis 1 2 0 0 3 0.3 1.0 - - 0.2 Cysticercosis 0 1 1 0 2 - 0.5 0.2 - 0.2 Dengue 0 0 0 1 1 - - - 0.3 0.1 Encephalitis 7 5 7 7 26 1.9 2.4 1.6 2.2 2.0	Coccidioidomycosis
Cysticercosis 0 1 1 0 2 - 0.5 0.2 - 0.2 Dengue 0 0 0 1 1 - - 0.3 0.1 Encephalitis 7 5 7 7 26 1.9 2.4 1.6 2.2 2.0	Cryptosporidiosis
Dengue000110.30.1Encephalitis7577261.92.41.62.22.0	Cysticercosis
Encephalitis 7 5 7 7 26 1.9 2.4 1.6 2.2 2.0	Dengue
	Encephalitis
Giardiasis 12 2 10 4 28 3.3 1.0 2.3 1.2 2.1	Giardiasis
Hansen's Disease (Leprosy) 0 0 0 0 0	Hansen's Disease (Leprosy)
Hepatitis A 1 1 0 4 6 0.3 0.5 - 1.2 0.5	Hepatitis A
Hepatitis B 2 1 3 2 8 0.6 0.5 0.7 0.6 0.6	Hepatitis B
Hepatitis C 0 0 0 0 0	Hepatitis C
Hepatitis Unspecified 0 0 0 0 0 0	Hepatitis Unspecified
Legionellosis 10 2 3 7 22 2.8 1.0 0.7 2.2 1.7	Legionellosis
Listeriosis, Nonperinatal 0 1 0 2 3 - 0.5 - 0.6 0.2	Listeriosis. Nonperinatal
Listeriosis Perinatal ^a 0 0 0 0 0	Listeriosis Perinatal ^a
	Lyme Disease
	Malaria
Meningitis Viral 23 3 23 22 71 6.4 1.4 5.4 6.8 5.4	Meningitis, Viral
Meningacoccal Infections 1 0 0 0 1 0.3 0	Meningococcal Infections
Pneumococcal Disease. Invasive 15 9 24 11 59 4.2 4.3 5.6 3.4 4.5	Pneumococcal Disease. Invasive
	Psittacosis
Q-fever 0 0 0 0 0	Q-fever
Relapsing Fever 0 0 0 0 0 0	Relapsing Fever
Rheumatic Fever, Acute 0 0 0 0 0 0	Rheumatic Fever, Acute
Salmonellosis 50 22 42 48 162 13.8 10.6 9.8 14.8 12.2	Salmonellosis
Shiga Toxin-Producing E. Coli 8 0 7 5 20 2.2 - 1.6 1.5 1.5	Shiga Toxin-Producing E. Coli
Shigellosis 12 20 16 7 55 3.3 9.7 3.7 2.2 4.2	Shigellosis
Staphylococcus Aureus Infection 0 0 0 0 0 0	Staphylococcus Aureus Infection
Streptococcus. Group A Invasive 5 5 6 5 21 1.4 2.4 1.4 1.5 1.6	Streptococcus, Group A Invasive
Strongyloidiasis 0 2 1 0 3 - 1.0 0.2 - 0.2	Strongyloidiasis
	Taeniasis
	Trichinosis
Tularemia 0 0 0 0 0	Tularemia
Typhoid Fever, Case 0 0 0 0 0 0	Typhoid Fever, Case
Typhoid Fever, Carrier 0 0 0 0 0	Typhoid Fever, Carrier
Typhus Fever 3 0 2 1 6 0.8 - 0.5 0.3 0.5	Typhus Fever
Vibrio 1 2 1 2 6 0.3 1.0 0.2 0.6 0.5	Vibrio
West Nile Virus 17 4 20 18 59 4.7 1.9 4.7 5.5 4.5	West Nile Virus

^aRates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.



Table O-8. Selected Notifiable Diseases SPA 8. South Bay Area Los Angeles County, 2015

		Frequ	ency		Rat	e (Cases	per 100,0)00) ^ь
Disease	НВ	IW	то	TOTAL	НВ	IW	то	TOTAL
Amebiasis	0	1	1	2	-	0.2	0.2	0.2
Botulism	0	0	0	0	-	-	-	-
Brucellosis	0	0	0	0	-	-	-	-
Campylobacteriosis	39	59	74	172	18.7	14.0	16.0	15.7
Cholera	0	0	0	0	-	-	-	-
Coccidioidomycosis	8	16	20	44	3.8	3.8	4.3	4.0
Cryptosporidiosis	0	1	2	3	-	0.2	0.4	0.3
Cysticercosis	0	0	0	0	-	-	-	-
Dengue	0	1	0	1	-	0.2	-	0.1
Encephalitis	3	1	3	7	1.4	0.2	0.6	0.6
Giardiasis	4	7	21	32	1.9	1.7	4.5	2.9
Hansen's Disease (Leprosy)	0	0	0	0	-	-	-	-
Hepatitis A	0	0	1	1	-	-	0.2	0.1
Hepatitis B	1	3	2	6	0.5	0.7	0.4	0.5
Hepatitis C	1	0	0	1	0.5	-	-	0.1
Hepatitis Unspecified	0	0	0	0	-	-	-	-
Legionellosis	10	8	9	27	4.8	1.9	1.9	2.5
Listeriosis, Nonperinatal	1	2	0	3	0.5	0.5	-	0.3
Listeriosis. Perinatal ^a	0	0	1	1	-	-	0.6	0.2
Lvme Disease	0	0	0	0	-	-	-	-
Malaria	0	6	0	6	-	1.4	-	0.5
Meningitis. Viral	12	7	14	33	5.7	1.7	3.0	3.0
Meningococcal Infections	0	0	0	0	-	-	-	-
Pneumococcal Disease, Invasive	12	20	29	61	5.7	4.8	6.3	5.6
Psittacosis	0	0	0	0	-	-	-	-
Q-fever	0	0	0	0	-	-	-	-
Relapsing Fever	0	0	0	0	-	-	-	-
Rheumatic Fever, Acute	0	0	0	0	-	-	-	-
Salmonellosis	29	42	44	115	13.9	10.0	9.5	10.5
Shiga Toxin-Producing E. Coli	6	7	10	23	2.9	1.7	2.2	2.1
Shigellosis	10	21	12	43	4.8	5.0	2.6	3.9
Staphylococcus Aureus Infection	0	1	0	1	-	0.2	-	0.1
Streptococcus, Group A Invasive	4	10	12	26	1.9	2.4	2.6	2.4
Strongyloidiasis	0	0	0	0	-	-	-	-
Taeniasis	0	0	0	0	-	-	-	-
Trichinosis	0	0	0	0	-	-	-	-
Tularemia	0	0	0	0	-	-	-	-
Typhoid Fever, Case	0	0	0	0	-	-	-	-
Typhoid Fever, Carrier	0	0	0	0	-	-	-	-
Typhus Fever	2	1	4	7	1.0	0.2	0.9	0.6
Vibrio	0	1	3	4	-	0.2	0.6	0.4
West Nile Virus	6	3	4	13	2.9	0.7	0.9	1.2

^aRates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.



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CRUDE DATA											
Disease	Dengue	Chikungunya	Zika								
Number of Cases	30	107	6								
Annual Incidence ^a											
LA County	-	-	-								
California	-	-	-								
United States	-	-	-								
Age at Diagnosis											
Mean	42	50	30								
Median	46	54	28								
Range	15–73 years	13–84 years	16–54 years								

AEDES MOSQUITO-BORNE DISEASES

^aNot applicable as there is no local transmission.

DESCRIPTION

Dengue, chikungunya, and Zika are the three most important vector-borne diseases affecting travelers in LAC. The main vectors that transmit all three diseases are the *Aedes aegypti* and *A. albopictus* mosquitoes. These diseases are mainly found in the tropical and subtropical areas of the world. Though both *Aedes* species mosquitoes have been found in LAC, these diseases are not currently found in mosquitoes in LAC and local transmission has not been documented.

The best methods to prevent infection from *Aedes* mosquito-borne diseases are to eliminate mosquito breeding sources and avoid mosquito bites. People visiting or residing in regions where there is risk of *Aedes* mosquito-borne disease should take precautions by using mosquito repellants, wearing protective clothing, staying in screened dwellings, and using air conditioning when available.

There is no prophylactic medicine or vaccine available to prevent dengue, chikungunya, or Zika.

Dengue

Dengue, a flavivirus related to the West Nile virus (WNV) and Zika virus, is the most common vector-borne viral disease in the world. Infection

with dengue virus has a range of clinical presentations from asymptomatic infection to severe systemic febrile illness. Treatment is supportive.

No cases of dengue acquired within the continental US were reported between 1946 and 1980. Since 1980, locally-acquired outbreaks have been documented in Texas, Florida, and most recently in Hawaii in 2015. Concern for the reemergence of dengue in Florida, Texas, and Hawaii as well as increases in dengue among returning US travelers over the past 20 years has prompted heightened vigilance among the medical and public health communities.

Dengue was added to the list of Nationally Notifiable Infectious Conditions in 2009 though it has been a notifiable condition in California and LAC for several decades. Confirmation of dengue requires a clinically compatible case be laboratory confirmed with testing of paired serological specimens or molecular testing. Probable cases require only a single serologically positive specimen.

Chikungunya

The most common symptoms of chikungunya virus infection are fever and joint pain. Other symptoms may include headache, muscle pain, joint swelling, or rash. Treatment is supportive.



Outbreaks have occurred in countries in Africa, Asia, Europe, and the Indian and Pacific Oceans. In late 2013, chikungunya virus was found for the first time in the Americas on islands in the Caribbean. On July 16, 2014, the first locally acquired cases in the continental US was identified in Florida.

For purposes of surveillance, confirmation of chikungunya requires a clinically compatible case be laboratory confirmed with testing of paired serological specimens or molecular testing. Probable cases require only a single serologically positive specimen.

Zika

Unlike dengue and chikungunya viruses, infected persons can also spread Zika to their sexual partners, though this method of transmission is much less likely than transmission due to mosquito bites. In addition, Zika can be passed from a pregnant woman to her fetus. Infection during pregnancy can cause microcephaly and other adverse pregnancy and birth outcomes.

Most persons infected with Zika are asymptomatic. Only 20% of infected persons experience symptoms. The most common symptoms of Zika virus disease are: fever, diffuse macular papular rash, joint pain, and conjunctivitis. Other symptoms include muscle pain, headache, pain behind the eyes, and vomiting. The illness is usually mild with symptoms lasting from several days to a week. Severe disease requiring hospitalization is uncommon. Increased reports of Guillain-Barré syndrome, a rare post-infectious central nervous system condition, has been linked to previous infection with Zika. Deaths from Zika are rarely reported.

Zika virus was first discovered in 1947 with the first human cases detected in 1952. Since then, outbreaks of Zika have been reported in tropical Africa, Southeast Asia, and the Pacific Islands. Zika outbreaks have probably occurred in many locations. In May 2015, the Pan American Health Organization (PAHO) issued an alert regarding the first confirmed Zika virus infections in Brazil. By December 2015, Puerto Rico reported its first confirmed Zika virus case. Locally transmitted Zika was not documented in the US in 2015. During 2015, confirmed cases were those with clinically compatible illness, epidemiological risk factors, and either a positive RT-PCR (reverse transcriptase polymerase chain reaction) urine or plasma specimen indicating Zika infection or a single positive serological specimen confirmed by a plaque reduction neutralization test (PRNT). Probable cases did not have a confirmatory PRNT and may show infection with Zika and other flaviviruses, dengue or chikungunya.

2015 TRENDS AND HIGHLIGHTS

Dengue

A similar number of cases was reported this year compared to last year (30 vs. 32 cases, 1). respectively) (Figure The proportion confirmed also remained the same (22% in 2014 and 20% in 2015). Prior to this, only one to two cases have been confirmed cases per year. The increase in confirmed cases can be attributed to the increase in laboratory evaluation for both dengue and chikungunya due to the emergence of chikungunya in the Americas in 2014. Further increases are likely with the additional emergence of Zika in the Americas in 2015. dengue Because is clinically and epidemiologically similar to both chikungunva and Zika, it is recommended that diagnostic tests for all three arbovirals be conducted together. All local cases identified in 2015 reported recent travel to countries and regions endemic for dengue including those in Central and South America, Asia, and the South Pacific. The most frequent travel destination was El Salvador (n=13, 43%), followed by Mexico (n=4, 13%) (Figure 2).

Chikungunya

- The number of chikungunya cases more than doubled from 50 in 2014 to 107 in 2015. However, the number of confirmed cases remained the same (n=17), comprising only 16% of cases. Prior to 2014, the last reported case of chikungunya in a LAC resident occurred in 2007 in a traveler to India. A large outbreak on the Asian subcontinent was occurring during that time.
- Most cases (n=41, 38%) reported travel to Mexico, followed by El Salvador (n=24, 22%) and Guatemala (n=23, 21%) (Figure 2). In 2014, none had reported travel to Mexico. The remaining cases traveled to other countries in Central America, the Caribbean, South America, the South Pacific, and India.



Zika

The first documented case of travel-associated Zika infection in LAC had an onset of illness in late November 2015 and had traveled to El Salvador. A total of six cases were documented in LAC residents by the end of the year. One third (n=2) of the cases had confirmed laboratory evidence by RT-PCR or PRNT, and five were female patients. The high proportion of females represent the interest and priority in diagnosing females who are pregnant or of child-bearing age. Both asymptomatic and pregnant women with possible Zika exposure from travel or sexual exposure are prioritized for testing. Half of LAC Zika cases reported travel to El Salvador, the remaining cases traveled to Mexico (n=1) and Guatemala (n=2).

Summary

- Dengue, chikungunya, and Zika virus infection can affect persons of all ages; however, the mean ages varied for each disease (42.2, 49.6, and 30.2 years, respectively). The largest proportion of Zika cases by far occurred among 15-34 year old patients (n=5, 83%) (Figure 3). Similar to the disproportionate number of females documented with Zika, the high proportion of cases in this age group likely represent the interest and priority in diagnosing those of child-bearing age. In contrast, both dengue and chikungunya presented most frequently among 45-54 year olds, roughly 30% of cases.
- Aedes mosquito-borne diseases affected mostly individuals of Hispanic/Latino race/ethnicity. Specifically, 53%, 85%, and 83% of dengue, chikungunya, and Zika cases, respectively, were Hispanic/Latino (Figure 5). This trend is likely due both to a high proportion of Hispanics/Latinos in

LAC and their frequency of travel to countries from which they or their families originate.

- Cases of dengue and chikungunya occurred in nearly all months of the year. Chikungunya cases peaked in July and August with 33 and 22 cases, respectively, comprising over half of the total annual cases. Seasonal patterns of both dengue and chikungunya are likely a result of travel patterns among LAC residents (Figure 6). The first autochthonous cases of Zika in El Salvador were detected mid-November. LAC's first case, who traveled to El Salvador, had onset later that month.
- Local infestations of A. aegypti have been • documented in LAC since 2014 and A. albopictus since 2011 in a number of cities in the central and eastern parts of LAC. With the vectors of dengue, chikungunya, and Zika present in the county, there is heightened concern and vigilance for possible local transmission of these diseases. Several cases of these diseases have occurred in residents living in cities with documented Aedes infestations (Table 1). Most cases occurred in residents of the city of Los Angeles, which is geographically expansive. However, Aedes mosquitoes have been limited to neighborhoods in the eastern part of the city boundaries.
- With the Zika outbreaks throughout Central America, South America, and the Caribbean Islands, LAC DPH has enhanced collaboration with vector control districts in the county. Cases of Zika, dengue, and chikungunya are shared with vector control agencies in order to enhance surveillance for *Aedes* sp. mosquitos and to encourage local clean-up efforts by residents.















Figure 5. Aedes Mosquito-Borne Diseases, by Race/Ethnicity* LAC, 2015 100% 90% Percentage of Cases 80% 70% 60% 50% 40% 30% 20% 10% 0% Asian Black Hispanic White □Dengue □Chikungunya ■Zika

*Excludes Other and unknown.

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							0	67						Hunti
20							-14-	-14-					-	Pacoi
15							0	0						Santa
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														*(Nitio

Number of Cases

Table 1. <i>Aedes</i> Mosquito-Borne Diseases, Cities of Residence with ≥1 Cases, LAC 2015												
	Dengue N=30	Chikungunya N=107	Zika N=6									
City	n (%)	n (%)	n (%)									
Los Angeles*	7 (23)	37 (35)	3 (50)									
El Monte*	0 (0)	4 (4)	0 (0)									
Lancaster	3 (10)	4 (4)	0 (0)									
Huntington Park*	0 (0)	3 (3)	0 (0)									
Pacoima	0 (0)	3 (3)	0 (0)									
Santa Monica	0 (0)	3 (3)	0 (0)									
Wilmington	0 (0)	3 (3)	0 (0)									
Bell	0 (0)	2 (2)	0 (0)									
Downey*	0 (0)	2 (2)	0 (0)									
North Hollywood	5 (17)	2 (2)	0 (0)									
Palmdale	0 (0)	2 (2)	0 (0)									
South Gate*	0 (0)	2 (2)	0 (0)									
South Pasadena	0 (0)	2 (2)	0 (0)									

*Cities with documented Aedes infestations.





AMEBIASIS

CRUDE DATA										
Number of Cases	62									
Annual Incidence ^a										
LA County	0.65									
California⁵	N/A									
United States ^b	N/A									
Age at Diagnosis										
Mean	39									
Median	39									
Range	3–84 years									

^aCases per 100,000 population

^bData not available

DESCRIPTION

Amebiasis is caused by the protozoan parasite *Entamoeba histolytica*. Cysts shed in human feces may contaminate food or drinking water. It also can be transmitted from person-to-person through fecal-oral spread. The incubation period for amebiasis is 1-4 weeks.

Although anyone can have this disease, it is more common in people who live in tropical areas with poor sanitary conditions. In the US, amebiasis is most common in:

- People who have traveled to tropical places that have poor sanitary conditions,
- Immigrants from tropical countries that have poor sanitary conditions,
- People who live in institutions that have poor sanitary conditions, and
- Men who have sex with men (MSM).

Intestinal disease is often asymptomatic. When symptoms occur, they may range from acute abdominal pain, fever, chills, and bloody diarrhea to mild abdominal discomfort with diarrhea alternating with constipation. Extraintestinal infection occurs when organisms become bloodborne, leading to amebic abscesses in the liver, lungs, or brain. Complications include colon perforation.

Visual inspection of stool for ova and parasites in the microbiology laboratory cannot differentiate between pathogenic E. histolytica and nonpathogenic E. dispar. Clinicians frequently order stool inspection for ova and parasites for persons with enteric symptoms, particularly those who have been involved in recreational activities (e.g., hiking), travel, persons with HIV, and MSM. Within LAC, stool ova and parasite specimens are frequently collected on new refugees as part of established CDC health screening guidelines despite the lack of significant gastrointestinal symptoms. Since many clinicians only obtain visual inspection of stool for ova and parasites more specific without pursuing Enzvme Immunoassay (EIA) stool antigen testing, which can differentiate between E. histolytica and E. dispar, many reports may be of persons infected with the non-pathogenic E. dispar, leading to an overestimation of E. histolytica infection.

Cases of amebiasis are reportable at the state. Local level and surveillance is enhanced through electronic laboratory reporting, which captures EIA, microscopic, or serologically confirmed amebiasis cases from selected participating hospital and commercial laboratories.

Proper hand hygiene before meals and after using the restroom is a major way to prevent infection and transmission of amebiasis. Persons who care for diapered/incontinent children and adults should ensure that they properly wash their hands. Individuals with diarrheal illness should avoid swimming in recreational waters to prevent transmission to others. Fecal exposure during sexual activity, anal intercourse, and oral-anal sexual practices should also be avoided. There is no vaccine available for disease prevention.

2015 TRENDS AND HIGHLIGHTS

- In 2013, the LAC DPH's protocol changed to count only symptomatic persons with suspected gastrointestinal and/or extraintestinal amebiasis with laboratory evidence of *E. histolytica*. In 2015, the LAC DPH continued to count only laboratory confirmed symptomatic infections as confirmed cases of *Entamoeba histolytica*.
- Amebiasis disease incidence rate slightly decreased in LAC from 0.68 cases per 100,000 in 2014 to 0.65 cases per 100,000 in



2015. There was a 42% decrease in the incidence from a mean of 1.13/100,000 in 2010-2012 to 0.65/100,000 in 2015 (Figure 1). This decrease in incidence is most likely due to the change in case definition that occured in 2013.

- In 2015, there were no reports of cases with extraintestinal infection with evidence of amoebic abscesses in the liver.
- The greatest incidence of amebiasis was in 55-64 age group (1.1 cases per 100,000) followed by those 35-44 and 45-54 age group (0.8 cases per 100,000) (Figure 2).
- Comparing race/ethnicity, the greatest incidence of amebiasis occurred among whites (1.4 cases per 100,000) (Figure 6).
- The highest amebiasis incidence rates was documented within SPA 5 (2.1 per 100,000) and SPA 4 had the second highest incidence of cases (1.9 per 100,000). The higher incidence in SPA 4 may be attributable to a

high number MSM in that region (Figure 4). Across the remaining six SPAs, the incidence of amebiasis cases were consistent, which suggested an even geographical distribution of cases.

- The number of cases peaked in March, consistent with the previous five-year average (Figure 5).
- Consistent with previous years, males comprised the majority (74%) of reported cases in 2015. The incidence rate of males was three times greater than females, with 1.0 and 0.3 cases per 100,000, respectively.
- Risk factor information was available for all cases reported in 2015. More than one risk factor was identified for several cases. The most frequently reported risk factor was contact with animals, predominantly exposure to dogs (37%), followed by travel to another country (32%), MSM (31%), and exposure to recreational water (14%).



	20	011 (N=	86)	2	012 (N	=99)	2	013 (N:	=57)	20)14 (N=	=64)	2015 (N=0		=62)
			Rate/			Rate/			Rate/			Rate/		(Rate/
	No.	(%)	100,000	No.	(%)	100,000	No.	(%)	100,000	No.	(%)	100,000	No.	(%)	100,000
Age Group															
<1	1	1.2	0.7	0	-	-	0	-	-	2	3.1	1.7	0	-	-
1-4	1	1.2	0.2	1	1.0	0.2	0	-	-	1	1.6	0.2	2	3.2	0.4
5-14	4	4.7	0.3	5	5.1	0.4	0	-	-	3	4.7	0.2	4	6.5	0.3
15-34	26	30.2	0.9	33	33.3	1.2	18	31.6	0.6	19	29.7	0.7	20	32.3	0.7
35-44	17	19.8	1.2	24	24.2	1.8	13	22.8	1	17	26.6	1.3	10	16.1	0.8
45-54	15	17.4	1.1	18	18.2	1.4	21	36.8	1.6	12	18.8	0.9	10	16.1	0.8
55-64	9	10.5	0.9	9	9.1	0.9	3	5.3	0.3	4	6.3	0.4	12	19.4	1.1
65+	13	15.1	1.2	9	9.1	0.8	2	3.5	0.2	6	9.4	0.5	4	6.5	0.3
Race/															
Ethnicity															
Asian	1	1.2	0.1	6	6.1	0.5	3	5.3	0.2	5	7.8	0.4	4	6.5	0.3
Black	7	8.1	0.8	4	4.0	0.5	2	3.5	0.3	7	10.9	0.9	4	6.5	0.5
Hispanic	40	46.5	0.8	39	39.4	0.9	17	29.8	0.4	26	40.6	0.6	16	25.8	0.3
White	27	31.4	0.9	33	33.3	1.2	34	59.6	1.3	23	35.9	0.9	37	59.7	1.4
Other	2	2.3	-	0	-	-	0	-	-	0	-	-	0	-	-
Unknown	9	10.5	-	17	17.2	-	1	1.8	-	3	4.7	-	1	1.6	-
SPA															
1	0	-	-	1	1.0	0.3	1	1.8	0.3	2	3.1	0.5	0	-	-
2	25	29.1	1.1	29	29.3	1.4	21	36.8	1	13	20.3	0.6	16	25.8	0.7
3	7	8.1	0.4	4	4.0	0.2	5	8.8	0.3	7	10.9	0.4	3	4.8	0.2
4	20	23.3	1.6	25	25.3	2.2	13	22.8	1.1	19	29.7	1.7	22	35.5	1.9
5	6	7.0	0.9	8	8.1	1.3	8	14.0	1.2	7	10.9	1.1	14	22.6	2.1
6	13	15.1	1.2	13	13.1	1.3	3	5.3	0.3	4	6.3	0.4	4	6.5	0.4
7	10 10	11.6	0.7	15	15.2	1.2	3	5.3	0.2	7	10.9	0.5	1	1.6	0.1
8	4	4.7	0.4	4	4.0	0.4	3	5.3	0.3	5	7.8	0.5	2	3.2	0.2
Unknown	1	1.2	-	0	-	-	0	-	-	0	-	-	0	-	-

Reported Amebiasis Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011–2015

*Rates calculated based on less than 19 cases or events are considered unreliable.



Figure 1. Incidence Rates of Amebiasis CA and LAC, 2006-2015*



* CA data not avaialable after 2010.

Figure 3. Percent Cases of Amebiasis by Race/Ethnicity LAC, 2015 (*N=62)



* Other includes Native American and any additional racial/ethnic group that cannot be categorized as Asian, Black, Hispanic, and White.



Figure 2. Incidence Rates of Amebiasis by Age Group LAC, 2015 (N=62)









Figure 5. Reported Amebiasis Cases by Month of Onset LAC, 2015 (N=62)

Figure 6. Incidence Rate of Amebiasis by Race/Ethnicity LAC, 2011-2015, 2015 (N=62)



⊠2011	2012	■2013	⊠2014	■2015	

Map 1. Amebiasis Rates by Health District, Los Angeles County, 2015*





CAMPYLOBACTERIOSIS

CRUDE DATA										
Number of Cases	1,623									
Annual Incidence ^a										
LA County	16.96									
California⁵	21.21									
United States ^₅	16.97									
Age at Diagnosis										
Mean	38									
Median	35									
Range	0–103 years									

^aCases per 100,000 population

^bCalculated from: CDC. *Notice to Readers*: Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306– 1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

Campylobacteriosis is a bacterial disease caused by several species of Gram-negative bacilli including Campylobacter jejuni, C. upsaliensis, C. coli, and C. fetus. It is usually transmitted through ingestion of organisms in undercooked poultry or other meat, contaminated food, water, or raw milk or occasionally through contact with infected animals. The incubation period is two to five days. Common symptoms include watery or bloody diarrhea, fever, abdominal cramps, mvalgia, and nausea. Sequelae include Guillain-Barré syndrome and Reiter syndrome, both of which are rare.

To reduce the likelihood of contracting campylobacteriosis, all food derived from animal sources, particularly poultry, should be thoroughly cooked. Cross contamination may be avoided by making sure utensils, counter tops, cutting boards, and sponges are cleaned or do not come in contact with raw poultry or meat or their juices. Hands should be thoroughly washed before, during, and after food preparation. The fluids from raw poultry or meat should not be allowed to drip on other foods in the refrigerator or in the shopping cart. It is especially important to wash hands and avoid cross contamination of infant foods, bottles, and eating utensils. It is recommended to consume only pasteurized milk, milk products, or juices. In addition, it is important to wash hands after coming in contact with any animal or its environment.

2015 TRENDS AND HIGHLIGHTS

- There was a 6.3% increase in the incidence of campylobacteriosis from the previous year and a 25.9% increase from 2010 (Figure 1).
- The highest rates were among children aged 1 to 4 (23.7 per 100,000) followed by persons aged <1 years (21.3 per 100,000) (Figure 2).
- SPA 5 had the highest rate (33.2 per 100,000), which is consistent with previous years (Figure 3).
- No outbreaks of campylobacteriosis were detected in 2015.
- Routine interviewing of campylobacteriosis cases was discontinued in 2010; however, surveillance of reported cases continues in order to monitor for clusters and review foodborne illness reports that have a diagnosis of campylobacteriosis.



	2011(N=1,259)		2012 (N=1,546)			2013 (N=1,703)			201	4 (N=1	,506)	2015 (N=1,623)			
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	16	1.3	11.5	46	3.0	38.7	45	2.6	37.2	27	1.8	22.8	23	1.4	21.3
1-4	158	12.6	27.2	136	8.8	28.6	159	9.3	32.7	118	7.8	24.2	115	7.1	23.7
5-14	146	11.6	11.0	181	11.7	15.1	173	10.2	14.3	159	10.6	13.2	138	8.5	11.4
15-34	366	29.1	12.4	418	27.0	15.1	495	29.1	17.5	437	29.0	15.5	525	32.4	18.6
35-44	133	10.6	9.2	169	10.9	12.8	182	10.7	13.7	192	12.8	14.5	210	12.9	15.9
45-54	142	11.3	10.5	186	12.0	14.5	185	10.9	14.3	175	11.6	13.5	197	12.1	15.0
55-64	114	9.1	11.9	163	10.5	16.0	177	10.4	17.2	155	10.3	14.6	176	10.8	15.9
65+	172	13.7	16.2	238	15.4	21.5	281	16.5	25.3	239	15.9	14.6	233	14.4	19.5
Unknown	12	1.0	-	9	0.6	-	6	0.4	-	4	0.3	-	6	0.4	0.3
Race/Ethnicity															
Asian	28	2.2	2.1	37	2.4	2.8	46	2.7	3.4	61	4.1	4.4	43	2.7	3.1
Black	21	1.7	2.5	34	2.2	4.4	46	2.7	5.9	39	2.6	5.0	25	1.5	3.2
Hispanic	157	12.5	3.3	161	10.4	3.6	167	9.8	3.6	219	14.5	4.8	210	12.9	4.5
White	119	9.5	4.2	228	14.8	8.6	386	22.7	14.5	272	18.1	10.2	264	16.4	9.8
Other	14	1.1	-	11	0.7	-	32	1.9	-	25	1.7	-	39	2.4	-
Unknown	920	73.1	-	1075	69.5	-	1026	60.3	-	888	59.0	-	1042	64.2	-
SPA															
1	46	3.7	12.3	36	2.3	9.3	41	2.4	10.5	55	3.7	14.0	66	4.1	16.7
2	347	27.6	15.7	362	23.4	16.9	401	23.6	18.4	388	25.8	17.7	416	25.6	18.7
3	164	13.0	9.5	200	12.9	12.4	220	12.9	13.5	217	14.4	13.2	217	13.4	13.1
4	156	12.4	12.4	234	15.1	20.8	292	17.2	25.6	198	13.2	17.2	230	14.2	19.7
5	142	11.3	21.5	228	14.8	35.7	218	12.8	33.7	189	12.6	29.0	219	13.5	33.2
6	123	9.8	11.5	140	9.1	13.8	175	10.3	17.0	136	9.0	13.2	138	8.5	13.2
7	136	10.8	9.9	179	11.6	13.8	180	10.6	13.7	137	9.1	10.4	165	10.2	12.5
8	145	11.5	12.9	157	10.2	14.7	172	10.1	16.0	185	12.3	17.1	172	10.6	15.7
Unknown	0	-	-	10	0.7	-	4	0.2	-	1	0.1	-	0	-	-

Reported Campylobacteriosis Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

*Rates calculated based on less than 19 cases or events are considered unreliable. Data provided in section race/ethnicity is incomplete.





Figure 1. Reported Campylobacteriosis Rates by Year LAC, 2005-2015



Figure 2. Reported Campylobacteriosis Rates by Age Group LAC, 2015 (N=1623)



Figure 3. Reported Campylobacteriosis Rates by SPA LAC, 2015 (N=1623)



AV SF Miles EV FH GL WV *PS ĆE РÓ WE EM SWSE Ε WH ISO Ŵ CN Cases Per 100,000 Population BF 18.8 - 33.2 **Health District Boundary** ΤO Service Planning Area (SPA) 15.7 - 18.7 14.1 - 15.6 HΒ 13.0 - 14.0 0.0 - 12.9 Catalina Island (HB) *Excludes Long Beach and Pasadena Data. Campylobacteriosis Page 44

Map 2. Campylobacteriosis Rates by Health District, Los Angeles County, 2015*



COCCIDIOIDOMYCOSIS

CRUDE DATA										
Number of Cases	613									
Annual Incidence ^a										
LA County	6.40									
California ^b	7.80									
United States ^b	3.44									
Age at Diagnosis										
Mean	52									
Median	52									
Range	1–99 years									

^aCases per 100,000 population

^bCalculated from: CDC. *Notice to Readers*: Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306– 1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

Coccidioidomycosis, also called Valley Fever, is a fungal disease transmitted through the inhalation of Coccidioides immitis spores that are carried in dust. Environmental conditions conducive to an increased occurrence of coccidioidomycosis include arid to semi-arid regions, dust storms, hot summers, warm winters, and sandy, alkaline soil. The fungus is endemic in the southwestern US (including Southern California) and parts of Mexico and South America. Most infected people exhibit no symptoms or have mild respiratory illness, but a few individuals develop severe illness such as pneumonia, meningitis, or dissemination to other parts of the body. Among the wide range of clinical presentations, only the most severe cases are usually diagnosed and reported to the health department. Blacks, Filipinos, pregnant women, vouna (age <5 vears), elderly, and immunocompromised individuals are at higher risk for severe disease. Currently, no safe and vaccine effective or drug to prevent coccidioidomycosis exists. Prevention lies mainly in dust avoidance and control (e.g., planting grass in dusty areas, putting oil on roadways, wetting down soil, air conditioning homes, wearing masks or respirators). Other options may be to warn people at high risk for severe disease not to travel to endemic areas when conditions are most dangerous for exposure.

Recovery from the disease confers lifelong immunity to reinfection, providing the rationale for development of a vaccine for prevention of symptomatic or serious forms of the disease. Increasing exposure and risk associated with construction, a growing naïve population in the endemic area, and antifungal treatments that have side effects and are not uniformly effective validate the need for prevention efforts.

2015 TRENDS AND HIGHLIGHTS

- The overall LAC incidence rate for coccidioidomycosis has continued to increase over the last ten years, and has tripled since 2010.
- No US data were available in year 2010 (Figure 1).
- Those over the age of 65 experienced the most cases (28%), with an incidence rate of 14.4 cases per 100,000 (Figure 2).
- Males represented 64% of cases; females 33%.
- Incidence rates were the highest among Blacks at 14.1 per 100,000, which has almost tripled from 5.3 per 100,000 since 2014 (Figure 4).
- SPA 1 has consistently reported the highest incidence rate of coccidioidomycosis in LAC; in 2015, the incidence rate was 42.6 per 100,000, which has doubled from last year's rate of 26.2 per 100,000 (Figure 5).
- July had the most cases at 14% of the total cases (n=83). However, there are no marked seasonal differences in rates based on data from the past 5 years, other than a modest decrease in late-winter and early-spring (Figure 6).



	2011 (N=304)		2012 (N=327)			2013 (N=362)			2014 (N=426)			2015 (N=613)			
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	-	-	0	-	-	1	0.3	0.8	0	-	-	0	-	-
1-4	1	0.3	0.2	3	0.9	0.6	0	-	0.6	1	0.2	0.2	4	0.7	0.8
5-14	3	1.0	0.2	3	0.9	0.3	6	1.7	0.5	4	0.9	0.3	7	1.1	0.6
15-34	62	20.4	2.1	68	20.8	2.5	67	18.5	2.4	68	16.0	2.4	96	15.7	3.4
35-44	35	11.5	2.4	53	16.2	4.0	55	15.2	4.1	61	14.3	4.6	98	16.0	7.4
45-54	67	22.0	5.0	84	25.7	6.5	86	23.8	6.7	91	21.4	7.0	127	20.7	9.6
55-64	54	17.8	5.6	46	14.1	4.5	73	20.2	7.1	93	21.8	8.8	109	17.8	9.9
65+	82	27.0	7.7	70	21.4	6.3	74	20.4	6.7	108	25.4	9.5	172	28.1	14.4
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	23	7.6	1.7	26	8.0	2.0	30	8.3	2.2	33	7.7	2.4	47	7.7	3.4
Black	48	15.8	5.6	46	14.1	5.9	50	13.8	6.4	42	9.9	5.3	111	18.1	14.1
Hispanic	94	30.9	2.0	133	40.7	2.9	104	28.7	2.3	139	32.6	3.0	201	32.8	4.3
White	134	44.1	4.7	121	37.0	4.6	132	36.5	5.0	175	40.8	6.6	217	35.4	8.1
Other	1	0.3	-	0	-	-	5	1.4	-	3	0.7	-	13	2.1	-
Unknown	4	1.3	-	1	0.3	-	41	11.3	-	34	8.0	-	24	3.9	-
SPA															
1	93	30.6	24.9	74	22.6	19.1	74	20.4	18.9	103	24.2	26.2	169	27.6	42.6
2	86	28.3	3.9	72	22.0	3.4	83	22.9	3.8	125	29.3	5.7	157	25.6	7.0
3	13	4.3	0.7	25	7.6	1.5	38	10.5	2.3	44	10.3	2.7	36	5.9	2.2
4	26	8.6	2.1	53	16.2	4.7	46	12.7	4.0	30	7.0	2.6	57	9.3	4.9
5	17	5.6	2.6	18	5.5	2.8	22	6.1	3.4	21	4.9	3.2	25	4.1	3.8
6	29	9.5	2.7	37	11.3	3.6	38	10.5	3.7	42	9.9	4.1	57	9.3	5.4
7	20	6.6	1.5	34	10.4	2.6	29	8.0	2.2	30	7.0	2.3	64	10.4	4.8
8	18	5.9	1.6	14	4.3	1.3	25	6.9	2.3	29	6.8	2.7	44	7.2	4.0
Unknown	2	0.7	-	0	-	-	7	1.9	-	2	0.5	-	4	0.7	-

Reported Coccidioidomycosis Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011–2015

*Rates calculated based on less than 19 cases or events are considered unreliable.





4

5

% Female

SPA

6

--- Previous 5-year average (Female)

7

8

60%

40%

30%

20%

10%

0%

CZZZZI % Male

1

2

Previous 5-year average (Male)

3

Percent 50%

Figure 4. Coccidioidomycosis Incidence Rates by Race/Ethnicity LAC, 2011-2015









Figure 6. Reported Coccidioidomycosis Cases

Map 3. Coccidioidomycosis Rates by Health District, Los Angeles County, 2015*







CRYPTOSPORIDIOSIS

CRUDE DATA									
Number of Cases	56								
Annual Incidence ^a									
LA County	0.59								
California⁵	0.95								
United States ^b	3.03								
Age at Diagnosis									
Mean	34								
Median	32								
Range	1–89 years								

^aCases per 100,000 population

^bCalculated from: CDC. *Notice to Readers*: Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306– 1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

Cryptosporidiosis is fecal-orally transmitted when cysts of the parasite *Cryptosporidium spp.* are ingested. The parasite is protected by an outer shell that allows it to survive outside the body for long periods of time and makes it very tolerant to chlorine disinfection.

While this parasite can be spread in several different ways, drinking contaminated water (drinking water and recreational water) is the most common way to spread the parasite. This parasite also can be transmitted through contact with animals. Another common cause is unprotected sexual contact, particularly among men who have sex with men (MSM). The usual incubation period is 2-10 days with typical symptoms of watery diarrhea, abdominal cramps, and low-grade fever. However, asymptomatic infection is also common. Symptoms last up to two weeks in healthy individuals. Those who have a weakened immune system may experience prolonged illness. Immunocompromised individuals (e.g., HIV/AIDS patients, cancer patients, and transplant patients), young children, and pregnant women are at risk for more severe illness.

Proper hand hygiene before meals and after using the restroom is a major way to prevent infection and transmission of cryptosporidiosis. Hand washing is also important for individuals who come in contact with diapered/incontinent children and adults. Persons should avoid drinking untreated water that may be contaminated. Persons with diarrhea should not go swimming in recreational waters in order to prevent transmission to others. Fecal exposure during sexual activity such as anal intercourse and oral-anal sexual practices should also be avoided.

2015 TRENDS AND HIGHLIGHTS

- The incidence of cryptosporidiosis cases in LAC decreased from 0.83 to 0.59 cases per 100,000 in 2014 and 2015 respectively. However, no trend exists over the last decade (Figure 1).
- The greatest incidence of cryptosporidiosis was in persons 15–34 years old (0.9 cases per 100,000) followed by those 35–44 years old (0.9 cases per 100,000) (Figure 2).
- The greatest incidence of cryptosporidiosis was in Whites (0.8 cases per 100,000) followed by Hispanic, Asians, and Blacks, respectively (0.3 cases per 100,000) (Figure 6).
- SPA 2 had the highest incidence rate, 1.1 cases per 100,000 (Figure 4). The reasons for this outcome are unclear since routine interviews of cryptosporidiosis cases were discontinued beginning October 1, 2015.
- Information on race and/or risk factors are incomplete. However, surveillance continues to monitor for clusters and review of cryptosporidiosis with positive laboratory reports.
- The number of reported cases peaked in August, which was consistent with the previous five years and is consistent with risk factors such as exposure to recreational water, hiking, and travel, which occur more commonly in the summer (Figure 5).
- The male to female ratio for 2015 was almost 2:1 compared with 2014 when the ratio was approximately 3:2. Males have consistently comprised the larger proportion of cases.
- No outbreaks of cryptosporidiosis were detected in 2015.



	2011 (N=51)		2012 (N=44)			2013 (N=48)			20)14 (N=	=78)	2015 (N=56)			
			Rate/			Rate/			Rate/	No	(0/)	Rate/	No	(0/)	Rate/
	No.	(%)	100,000	No.	(%)	100,000	No.	(%)	100,000	NO.	(%)	100,000	NO.	(%)	100,000
Age Group															
<1	0	-	-	0	0	0	0	-	-	0	-	-	0	-	-
1-4	3	5.9	0.5	2	4.5	0.4	1	2.1	0.2	2	2.6	0.4	2	3.6	0.4
5-14	6	11.8	0.5	4	9.1	0.3	2	4.2	0.2	5	6.4	0.4	5	8.9	0.4
15-34	16	31.4	0.5	13	29.5	0.5	16	33.3	0.6	29	37.2	1.0	25	44.6	0.9
35-44	10	19.6	0.7	8	18.2	0.6	8	16.7	0.6	17	21.8	1.3	9	16.1	0.7
45-54	6	11.8	0.4	8	18.2	0.6	14	29.2	1.1	15	19.2	1.2	6	10.7	0.5
55-64	3	5.9	0.3	4	9.1	0.4	2	4.2	0.2	5	6.4	0.5	6	10.7	0.5
65+	7	13.7	0.7	4	9.1	0.4	5	10.4	0.5	4	5.1	0.4	3	5.4	0.3
Unknown	0	-	-	1	2.3	-	0	-	-	1	1.3	-			
Race/Ethnicity															
Asian	3	5.9	0.2	1	2.3	0.1	2	4.2	0.1	5	6.4	0.4	4	7.1	0.3
Black	6	11.8	0.7	1	2.3	0.1	12	25.0	1.5	12	15.4	1.5	2	3.6	0.3
Hispanic	11	21.6	0.2	9	20.5	0.2	7	14.6	0.2	22	28.2	0.5	16	28.6	0.3
White	20	39.2	0.7	19	43.2	0.7	24	50.0	0.9	34	43.7	1.3	21	37.5	0.8
Other	0	-	-	0	-	-	2	4.2	-	2	2.6	-	0	-	-
Unknown	11	21.6	-	14	31.8	-	1	2.1	-	3	3.8	-	13	23.2	-
SPA															
1	6	11.8	1.6	5	11.4	1.3	4	8.3	1.0	3	3.8	0.8	0	-	-
2	15	29.4	0.7	12	27.3	0.6	15	31.3	0.7	23	29.5	1.1	24	42.9	1.1
3	4	7.8	0.2	7	15.9	0.4	4	8.3	0.2	5	6.4	0.3	7	12.5	0.4
4	8	15.7	0.7	6	13.6	0.5	6	12.5	0.5	21	26.9	1.8	8	14.3	0.7
5	5	9.8	0.8	6	13.6	0.9	6	12.5	0.9	4	5.1	0.6	4	7.1	0.6
6	4	7.8	0.4	1	2.3	0.1	5	10.4	0.5	6	7.7	0.6	5	8.9	0.5
7	1	2.0	0.5	1	2.3	0.1	3	6.3	0.2	8	10.2	0.6	3	5.4	0.2
8	1	2.0	0.1	3	6.8	0.3	5	10.4	0.5	7	9.0	0.6	3	5.4	0.3
Unknown	7	13.7	-	3	6.8	-	0	-	-	1	1.3	-	2	3.6	-

Reported Cryptosporidiosis Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011–2015

*Rates calculated based on less than 19 cases or events are considered unreliable.









Other includes Native American and any additional racial/ethnic group that cannot be categorized as Asian, Black, Hispanic, and White.



Figure 2. Incidence Rates of Cryptosporidiosis by Age Group, LAC, 2015 (N=56)

Figure 4. Incidence Rates of Cryptosporidiosis by SPA LAC, 2015 (N=56)







*Date of onset missing on 19 out of 56 cases.

Figure 5. Reported Cryptosporidiosis Cases by Month of Onset, LAC, 2015 (N*=56)



Figure 6. Incidence Rates of Cryptosporidiosis by Race/Ethnicity LAC, 2011-2015

AV SF 4.5 Miles EV FH GL WV *PS HW AH CE PO WE EM SWSE EI WΗ SO SA ÍŴ CN Cases Per 100,000 Population BF 1.0 - 2.7 TO **Health District Boundary** 0.5 - 0.9 Service Planning Area (SPA) 0.3 - 0.4 HB 0.1 - 0.2 0.0 Catalina Island (HB) *Excludes Long Beach and Pasadena Data. Cryptosporidiosis Page 55

Map 4. Cryptosporidiosis Rates by Health District, Los Angeles County, 2015*




ENCEPHALITIS

CRUDE	DATA
Number of Cases	136
Annual Incidence ^a	
LA County	1.42
Californiab	N/A
United States ^b	N/A
Age at Diagnosis	
Mean	60
Median	63
Range	0–94 years

^aCases per 100,000 population

^bNot nationally notifiable

DESCRIPTION

Encephalitis, an inflammation of parts of the brain, spinal cord, and meninges, causes headache, stiff neck, fever, and altered mental status. It can result from infection of a number of different agents including viral, parasitic, fungal, rickettsial, and bacterial pathogens as well as chemical agents. Public health conducts passive surveillance of encephalitis cases and is limited to cases with suspected or confirmed viral and bacterial etiologies, which includes primary and postinfectious encephalitis but excludes individuals with underlying human immunodeficiency virus (HIV) infection. Of special concern are arthropod-borne viruses (i.e., arboviruses), which are maintained in nature through biological transmission between susceptible vertebrate hosts by blood feeding arthropods (mosquitoes, ticks, and certain mites and gnats). All arboviral encephalitides are zoonotic, meaning that they are maintained in complex life cycles involving a nonhuman vertebrate primary host and a primary arthropod vector. Arboviruses have a global distribution. The five main viral agents of encephalitis in the United States are West Nile virus (WNV), eastern equine encephalitis virus (EEEV), western equine encephalitis virus (WEEV), Saint Louis encephalitis virus (SLEV), and La Crosse encephalitis virus (LACV). All of these are transmitted by mosquitoes, thus can be prevented by personal protection and mosquito control (see WNV chapter).

- A total of 136 cases of encephalitis were confirmed in 2015 compared to 92 cases reported in 2014. The increase in encephalitis was most likely due to the increase in WNVassociated encephalitis cases. The 2015 surveillance year had the second highest number of total WNV infections (n=300) cases since the first LAC WNV outbreak (n=309), which occurred in 2004 (see WNV chapter).
- Most laboratory confirmed encephalitis cases (n=114, 84%) were due to underlying WNV infection. WNV-associated encephalitis is the most frequently reported etiology for viral encephalitis in the US. Cases of WNV encephalitis were reported from late July through late November. October, the peak month of encephalitis reports coincided with the WNVinfection peak in 2015 (Figure 4). A total of 17 (15%) of WNV-associated cases died, 0.2% mortality rate.
- Herpes virus encephalitis associated with herpes simplex virus was the second most common etiology for reported encephalitis cases (n=3, 2%).
- A total of 19 (14%) encephalitis cases were considered to be due to an unknown viral etiology based on review of medical records.
- The greatest incidence of encephalitis was in persons ≥65 years old (7.3 cases per 100,000) followed by those 55-64 years old (1.3 cases per 100,000 population). The peak incidence in persons ≥65 years old corresponds to age as a risk factor for WNV-associated neuroinvasive disease. The average age of WNV encephalitis cases in 2015 was 69.4 years.
- The highest encephalitis incidence rates were documented within SPA 2 (2.3 cases per 100,000) and SPA 7 (2.0 cases per 100,000) (Figure 3). The SPAs with the highest incidence rates for WNV-associated encephalitis were SPA 2 (1.9 cases per 100,000) and SPA 3 (1.7 cases per 100,000).



	2011 (N=59)			2012 (N=75)			2013 (N=79)			20)14 (N=	=92)	2015 (N=136)		
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	3	5.1	2.1	1	1.3	0.8	1	1.3	0.8	1	1.1	0.8	0	-	-
1-4	4	6.8	0.7	3	4.0	0.6	4	5.1	0.8	2	2.2	0.4	1	0.7	0.2
5-14	10	16.9	0.8	8	10.7	0.7	7	8.9	0.6	4	4.3	0.3	7	5.1	0.6
15-34	8	13.6	0.3	6	8.0	0.2	6	7.6	0.2	5	5.4	0.2	5	3.7	0.2
35-44	2	3.4	0.1	0	0	-	1	1.3	0.1	3	3.3	0.2	6	4.4	0.5
45-54	9	15.3	0.7	9	12.0	0.7	13	16.5	1.0	10	10.9	0.8	16	11.8	1.2
55-64	8	13.6	0.8	12	16.0	1.2	19	24.1	1.9	23	25.0	2.2	14	10.3	1.3
65+	15	25.4	1.4	36	48.0	3.2	28	25.3	2.5	44	47.8	3.9	87	64.0	7.3
Unknown	0	-	-	0	-	-	8	10.1	-	0	-	-	-	-	-
Race/Ethnicity															
Asian	0	0	-	8	10.7	0.6	6	7.6	0.4	8	8.7	0.6	4	2.9	0.3
Black	4	6.8	0.5	3	4.0	0.4	2	2.5	0.3	3	3.3	0.4	3	2.2	0.4
Hispanic	33	55.9	0.7	23	30.7	0.5	20	25.3	0.4	24	26.1	0.5	51	37.5	1.1
White	14	23.7	0.5	31	41.3	1.2	36	45.6	1.4	40	43.5	1.5	62	45.6	2.3
Other	1	1.7	-	5	6.7	-	3	3.8	-	0	-	-	1	0.7	-
Unknown	7	11.9	-	5	6.7	-	12	15.2	-	17	18.5	-	15	11.0	-
SPA															
1	2	3.4	0.5	6	8.0	1.5	6	7.6	1.5	1	1.1	0.3	4	2.9	1.0
2	20	33.9	0.9	22	29.3	1.0	27	34.2	1.2	21	22.8	1.0	52	38.2	2.3
3	9	15.3	0.5	24	32.0	1.5	11	13.9	0.7	14	15.2	0.9	19	14.0	1.1
4	4	6.8	0.3	10	13.3	0.9	3	3.8	0.3	12	13.0	1.0	14	10.3	1.2
5	1	1.7	0.2	2	2.7	0.3	2	2.5	0.3	11	12.0	1.7	11	8.1	1.7
6	4	6.8	0.4	4	5.3	0.4	3	3.8	0.3	5	5.4	0.5	3	2.2	0.3
7	8	13.6	0.6	5	6.7	0.4	11	13.9	0.8	18	19.6	1.4	26	19.1	2.0
8	5	8.5	0.4	2	2.7	0.2	13	16.5	1.2	9	9.8	0.8	7	5.1	0.6
Unknown	6	10.2	-	0	-	-	3	3.8	-	1	1.1	-	0	-	-

Reported Encephalitis Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015





*See text for limitations.

2

3

2.5

2

1.5

1

0.5

0

1

Cases per 100,000



8

Figure 2. Percent Cases of Encephalitis by Race/Ethnicity LAC, 2015 (*N=136)







4

SPA

5

6

7

Encephalitis Page 59





Figure 5. Reported Encephalitis Cases by Race/Ethnicity LAC, 2011-2015

Map 5. Encephalitis Rates by Health District, Los Angeles County, 2015*







GIARDIASIS

CRUDE	DATA
Number of Cases	379
Annual Incidence	
LA County ^a	4.00
California ^b	5.49
United States ^b	4.51
Age at Diagnosis	
Mean	39
Median	38
Range	1–90 years

^aCases per 100,000 population

^bCalculated from: CDC. *Notice to Readers*: Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306– 1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

Giardiasis is an intestinal infection caused by the zoonotic protozoan parasite Giardia intestinalis (previously G. lamblia). Giardia cysts shed in animal or human feces may contaminate food or drinking water or be transferred on hands or fomites. Recreational waters such as lakes and pools may also serve as vehicles of transmission. Incubation can range from 3-25 days or longer, but the median incubation time is 7-10 days. While often asymptomatic, symptoms can include sulfurous burps, chronic diarrhea, frequent loose and pale greasy stools, bloating, cramps, fatigue, and weight loss. Complications are rare but may include malabsorption of fats and fat-soluble vitamins. Children attending day care represent a reservoir of disease in developed countries. There is no vaccine.

To prevent transmission of giardiasis, individuals should wash their hands before eating, after using the toilet, and after changing diapers. People should shower before recreational water use and avoid accidental swallowing of recreational water. Persons with diarrhea should avoid swimming in recreational waters in order to prevent transmission to others. Fecal exposure during sexual activity such as anal intercourse and oral-anal sexual practices should also be avoided.

- In 2015, only laboratory confirmed symptomatic *Giardia* infections continued to be counted as confirmed cases of giardiasis in LAC.
- Giardiasis disease incidence slightly increased in LAC from 3.7 cases per 100,000 in 2014 to 4.0 cases per 100,000 in 2015 (Figure 1).
- The highest age-specific incidence rate occurred among adults 35-44 years old with 5.7 cases per 100,000. In 2013 and 2014, the incidence was also highest among 35-44 year olds. From 2010-2012, the highest incidence was among 1-4 year olds (Figure 2).
- Whites continue to have the highest race/ethnicity-specific incidence rates compared to other races (Figure 3). The greatest proportion of cases were reported among Whites (n=238, 63%) and Hispanics (n=104, 27%) (Figure 3).
- SPA 5 reported the highest incidence rate of giardiasis with 11.7 cases per 100,000 in 2015 (Figure 5). The most common risk factors reported among these cases were travel to another country and contact with animals.
- The number of cases reported in 2015 peaked from August to September, which was consistent with the previous five-year average (Figure 6).
- Males have consistently accounted for a larger proportion of cases. In 2015, males accounted for 73% and females 27% of cases. The incidence rate of giardiasis in males was 5.8 per 100,000 and females was 2.1 cases per 100,000.
- Complete risk factor data were available for all cases. More than one risk factor was identified for many cases. The most frequently reported risk factor was contact with animals (42%), predominantly dogs. Travel to another country was also frequently reported (28%) followed by MSM (men who have sex with men) activity (27%) and exposure to recreational waters (19%). Other reported risk factors included hiking (10%), camping (6%), and recently arrived immigrant or refugee status (3%).



	20	2011 (N=292)		201	2 (N=2	94)	20	13 (N=3	392)	201	14 (N=3	346)	20	15 (N=:	379)
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	1	0.3	0.7	0	-	-	3	0.7	2.5	0	-	-	0	-	-
1-4	22	7.5	3.8	30	10.2	6.3	20	5.1	4.1	19	5.5	3.9	14	3.7	2.9
5-14	39	13.4	2.9	29	9.9	2.4	41	10.5	3.4	27	7.8	2.2	20	5.3	1.7
15-34	84	28.8	2.8	86	29.3	3.1	114	29.1	4.0	96	27.7	3.4	126	33.2	4.5
35-44	49	16.8	3.4	52	17.7	3.9	65	16.6	4.9	70	20.2	5.3	76	20.1	5.7
45-54	44	15.1	3.3	39	13.3	3	72	18.4	5.6	63	18.2	4.8	66	17.4	5.0
55-64	29	9.9	3	35	11.9	3.4	51	13.0	5.0	42	12.1	4.0	47	12.4	4.2
65+	23	7.9	2.2	22	7.5	2	26	6.6	2.3	29	8.4	2.6	29	7.7	2.4
Unknown	1	0.3	-	1	0.3	-	0	-	-	0	-	-	1	0.3	-
Race/Ethnicity															
Asian	20	6.8	1.5	18	6.1	1.4	25	6.4	1.8	24	6.9	1.7	17	4.5	1.2
Black	18	6.2	2.1	17	5.8	2.2	27	6.9	3.5	25	7.2	3.2	14	3.7	1.8
Hispanic	89	30.5	1.9	84	28.6	1.9	124	31.6	2.7	113	32.7	2.5	104	27.4	2.2
White	146	50.0	5.1	125	42.5	4.7	210	53.6	7.9	175	50.6	6.6	238	62.8	8.9
Other	2	0.7	-	1	0.3	-	2	0.5	-	3	0.9	-	4	1.1	-
Unknown	17	5.8	-	49	16.7	-	4	1.0	-	6	1.7	-	2	0.5	-
SPA															
1	8	2.7	2.1	5	1.7	1.3	9	2.3	2.3	10	2.9	2.5	9	2.4	2.3
2	102	34.9	4.6	96	32.7	4.5	95	24.2	4.4	89	25.7	4.1	67	17.7	3.0
3	22	7.5	1.3	27	9.2	1.7	50	12.8	3.1	26	7.5	1.6	34	9.0	2.1
4	47	16.1	3.7	57	19.4	5.1	71	18.1	6.2	82	23.7	7.1	110	29.0	9.4
5	37	12.7	5.6	39	13.3	6.1	49	12.5	7.6	46	13.3	7.1	77	20.3	11.7
6	20	6.8	1.9	17	5.8	1.7	39	9.9	3.8	24	6.9	2.3	22	5.8	2.1
7	26	8.9	1.9	25	8.5	1.9	42	10.7	3.2	31	9.0	2.4	28	7.4	2.1
8	28	9.6	2.5	28	9.5	2.6	37	9.4	3.4	38	11.0	3.5	32	8.4	2.9
Unknown	2	0.7	-	0	-	-	0	-	-	0	-	-	0	-	-

Reported Giardiasis Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015





Giardiasis Page 65





Figure 6. Reported Giardiasis Cases by Month of Onset LAC, 2015 (N=379)



2015 — Previous 5-year average

Map 6. Giardiasis Rates by Health District, Los Angeles County, 2015*







HEPATITIS A

CRUDE	DATA
Number of Cases	33
Annual Incidence ^a	
LA County	0.34
California ^b	0.46
United States ^₅	0.43
Age at Diagnosis	
Mean	41
Median	39
Range	7–92 years

^aCases per 100,000 population

^b Calculated from: CDC. *Notice to Readers:* Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306– 1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

Hepatitis A virus (HAV), an RNA virus, is a vaccine-preventable disease transmitted fecalorally, person-to-person, or through vehicles such as food. In the US, among adults with identified risk factors, the majority of cases are among men who have sex with other men (MSM), persons who use illegal drugs, and international travelers. Sexual and household contacts of HAV-infected persons are also at increased risk of getting the disease.

The average incubation period is 28 days (range 15–50 days). Signs and symptoms of acute hepatitis A include fever, fatigue, loss of appetite, nausea, vomiting, abdominal pain, dark urine, clay-colored bowel movements, joint pain, and jaundice. Many cases, especially in children, are mild or asymptomatic. Recovery usually occurs within one month. Infection confers life-long immunity.

Hepatitis A vaccination is the most effective means of preventing HAV transmission among persons at risk of infection. Hepatitis A vaccination is recommended for:

- 1) All children between their first and second birthdays (12-23 months old),
- Anyone ≥1 year old traveling to or working in countries with high or intermediate prevalence of hepatitis A,
- Children and adolescents 2-18 years old who live in states or communities where routine vaccination has been implemented because of high disease incidence,
- 4) MSM,
- 5) People who use street drugs,
- 6) People with chronic liver disease,
- 7) People who are treated with clotting factor concentrates,
- 8) People who work with HAV-infected primates or HAV in research laboratories, and
- Households adopting a child or caring for an adopted child from a country where hepatitis A is common.

LAC DPH uses the CDC Council of State and Territorial Epidemiologists 2012 case definition for acute hepatitis A to standardize surveillance of this infection. A case of hepatitis A is defined as a person with:

- An acute illness with discrete onset of symptoms,
- 2) Jaundice or elevated alanine aminotransferase (ALT) levels, and
- Either IgM anti-HAV positive or an epidemiologic link to a person who has laboratory confirmed hepatitis A.

- The 2015 incidence rate of acute hepatitis A was lower than that in 2014 (0.3 per 100,000 versus 0.4 per 100,000, respectively (Figure 1)).
- The incidence rate was highest among those between 35–44 year olds (0.7 per 100,000) followed by 15–34 year olds and 55–64 year olds (0.4 per 100,000), respectively (Figure 2).
- Similar to the previous years, in 2015, the highest incidence rate was seen in Asians (0.8 per 100,000) (Figure 3).
- The male-to-female ratio was 15:18.
- A total of four SPAs had incidence rates greater than the overall county incidence rate of 0.3 per 100,000. These areas are SPA 2 (0.4 per 100,000), SPA 4 (0.8 per 100,000),



SPA 5 (0.5 per 100,000), and SPA 7 (0.5 per 100,000) (Figure 4).

• Risk factors were identified in 70% (n=23) of the 33 confirmed cases including some cases with multiple risk factors. Recent travel

outside of the US (n=13, 39%) was the most frequently reported risk factor followed by household travel (n=6, 18%), consumption of raw shellfish (n=7, 21%), and MSM (n=1, 3%) (Figure 5).



Reported Hepatitis A Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=45)			2012 (N=47)			2013 (N=60)				2014 (N=4	2)	2015 (N=33)		
	No.	(%)	Rate/ 100,000	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
1-4	1	2.2	0.2	0	-	-	0	-	-	0	-	-	0	-	-
5-14	3	6.7	0.2	3	6.4	0.3	2	3.3	0.2	1	2.4	0.1	1	3.0	0.1
15-34	18	40.0	0.6	24	51.1	0.9	22	36.7	0.8	17	40.5	0.6	12	36.4	0.4
35-44	11	24.4	0.8	9	19.1	0.7	12	20.0	0.9	9	21.4	0.7	9	27.3	0.7
45-54	5	11.1	0.4	3	6.4	0.2	8	13.3	0.6	0	0.0	0.0	3	9.1	0.2
55-64	3	6.7	0.3	5	10.6	0.5	13	21.7	1.3	8	19.0	0.8	4	12.1	0.4
65+	4	8.9	0.4	3	6.4	0.3	3	5.0	0.3	7	16.7	0.6	4	12.1	0.3
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	13	28.9	1.0	8	17.0	0.6	15	25.0	1.1	11	26.2	0.8	11	33.3	0.8
Black	2	4.4	0.3	0	0.0	0.0	1	1.7	0.1	4	9.5	0.5	1	3.0	0.1
Hispanic	8	17.8	0.2	20	42.6	0.4	18	30.0	0.4	14	33.3	0.3	11	33.3	0.2
White	22	48.9	0.8	14	29.8	0.5	26	43.3	1.0	12	28.6	0.5	9	27.3	0.3
Other	0	-	-	0	-	-	0	-	-	1	2.4	-	1	3.0	-
Unknown	0	-	-	5	10.6	-	0	-	-	0	-	-	0	-	-
SPA															
1	2	4.4	0.5	2	4.3	0.5	3	5.0	0.8	2	4.8	0.5	0	-	-
2	17	37.8	0.8	17	36.2	0.8	17	28.3	0.8	12	28.6	0.5	8	24.2	0.4
3	10	22.2	0.6	4	8.5	0.2	5	8.3	0.3	5	11.9	0.3	5	15.2	0.3
4	6	13.3	0.5	8	17.0	0.7	8	13.3	0.7	12	28.6	1.0	9	27.3	0.8
5	2	4.4	0.3	4	8.5	0.6	9	15.0	1.4	1	2.4	0.2	3	9.1	0.5
6	3	6.7	0.3	0	0.0	0.0	1	1.7	0.1	4	9.5	0.4	1	3.0	0.1
7	1	2.2	0.1	7	14.9	0.5	12	20.0	0.9	3	7.1	0.2	6	18.2	0.5
8	4	8.9	0.4	5	10.6	0.5	5	8.3	0.5	3	7.1	0.3	1	3.0	0.1
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-









Figure 3. Hepatitis A Incidence Rates* by Race/Ethnicity LAC, 2012-2015



Figure 4. Incidence Rates* of Hepatitis A by SPA LAC, 2015 (N=33)







Figure 5. Hepatitis A Reported Risk Factors* LAC, 2015 (N=23)

*Includes cases with multiple risk factors





HEPATITIS B, ACUTE (NONPERINATAL)

CRUI	DE DATA
Number of	50
Cases	50
Annual	
Incidence ^a	
LA County	0.52
California ^b	0.41
United States ^₅	1.05
Age at Diagnosis	
Mean	44
Median	43
Range	23–84 years

^aCases per 100,000 population

^bCalculated from: CDC. *Notice to Readers*: Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306–1321. Available at: www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

Hepatitis B is a DNA virus transmitted through activities that involve percutaneous or mucosal contact with infectious blood or bodily fluids. This is often through injection drug use, sexual contact with an infected person, or contact from an infected mother to her infant during birth. Transmission also occurs among household contacts of a person with hepatitis B. Healthcare-associated transmission of hepatitis B is documented in the US and should be considered in persons without traditional risk factors.

Symptoms occur in less than half of those acutely infected and begin an average of 90 days (range 60–150 days) after exposure. They can include: fever, fatigue, loss of appetite, nausea, vomiting, abdominal pain, dark urine, clay-colored bowel movements, joint pain, and jaundice. Approximately 2-10% of adults infected with hepatitis B virus (HBV) are unable to clear the virus within six months and become chronic carriers. Death from cirrhosis or liver cancer occurs in an estimated 15–25% of those with chronic infection. Overall, hepatitis B is more prevalent and infectious than HIV.

The absence of acute hepatitis B in persons under 19 years old in the US is evidence of the successful immunization strategy to eliminate HBV transmission. This strategy includes: screening all pregnant women and providing immunoprophylaxis to infants of HBV-infected women, routine immunization of all infants, and catch-up vaccination of all previously unvaccinated children <19 years old.

Adult vaccination is recommended for high risk groups including: men who have sex with men (MSM), those with history of multiple sex partners, injection drug users, persons seeking treatment for sexually transmitted diseases, household and sex contacts of persons with chronic HBV infections, healthcare workers, persons with chronic liver disease, persons with HIV, hemodialysis patients, and unvaccinated adults with diabetes mellitus 19-59 years old.

For the purpose of surveillance, LAC DPH uses the 2012 CDC Council of State and Territorial Epidemiologists (CSTE) case definition for acute hepatitis B. The criteria include:

- 1) Discrete onset of symptoms,
- 2) Jaundice or elevated alanine aminotransferase (ALT) levels >100 IU/L, and
- 3) HBsAg positive and anti-HBc IgM positive, (if done).

In 2012, the CDC CSTE modified the acute hepatitis B case definition to include documented seroconversion cases (documented negative HBV test result within six months prior to HBV diagnosis) without the acute clinical presentation.

- The 2015 incidence rate slightly increased from the previous year (0.5 per 100,000 versus 0.4 per 100,000) (Figure 1).
- The incidence rate was highest among those between 45–54 years old (1.4 per 100,000) (Figure 2).
- The male-to-female ratio was 39:11.
- Similar to the previous year, Blacks had the highest incidence rate in 2015 (1.1 per 100,000) (Figure 3).
- Three SPAs had incidence rates greater than the overall county rate of 0.5 per 100,000: SPA 2 (0.6 per 100,000), SPA 6 (0.7 per 100,000), and SPA 7 (0.6 per 100,000) (Figure 4).
- Risk factors were identified in 50% (n=25) of the 50 confirmed cases (including some cases with multiple risk factors). Of those with identified risk factors, the most frequently reported risk factor was unprotected sexual contact (n=10,



40%). Half of these cases reported having multiple sexual partners (n=5, 20%) followed by patients who had dental procedures done in a non-healthcare facility outside of the country (n=2, 8%), IV drug use (n=1, 4%), consumption of raw shellfish (n=1, 4%), and source of infection could not be determined (n=5, 20%).

In September, ACDC investigated a breach in infection control in an acute care facility dialysis unit after being notified by the facility's infection preventionist (IP) that an HIV-infected patient had a documented HBV seroconversion following the dialysis of a known chronically infected HBV patient. The investigation revealed that a technician had not performed

recommended bleaching procedure the between the two patients as well as the subsequent five patients that had used the same dialysis machine. The IP immediately initiated infection prevention measures and ensured that the dialysis machine was not used further until adequate disinfection was completed. The six exposed patients were screened for HBV IgM and HBV through polymerase chain reaction (PCR). ACDC did not identify any additional HBV cases related to this breach. The five subsequent patients that were potentially exposed showed no evidence of HBV infection in follow-up testing.

Reported Hepatitis B, Acute, (Nonperinatal) Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=60)			2012 (N=38)			2013 (N=55)				2014 (N=42	2)	2015 (N=50)		
_	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
1-4	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
5-14	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
15-34	12	20.0	0.4	10	26.3	0.4	20	36.4	0.7	5	11.9	0.2	10	20.0	0.4
35-44	10	16.7	0.7	13	34.2	1.0	15	27.3	1.1	16	38.1	1.2	14	28.0	1.1
45-54	21	35.0	1.6	10	26.3	0.8	12	21.8	0.9	14	33.3	1.1	18	36.0	1.4
55-64	12	20.0	1.2	3	7.9	0.3	5	9.1	0.5	3	7.1	0.3	5	10.0	0.5
65+	5	8.3	0.5	2	5.3	0.2	3	5.5	0.3	4	9.5	0.4	3	6.0	0.3
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	3	5.0	0.2	1	2.6	0.1	6	10.9	0.4	3	7.1	0.2	5	10.0	0.4
Black	13	21.7	1.5	5	13.2	0.6	12	21.8	1.5	6	14.3	0.8	9	18.0	1.1
Hispanic	19	31.7	0.4	13	34.2	0.3	21	38.2	0.5	20	47.6	0.4	17	34.0	0.4
White	23	38.3	0.8	14	36.8	0.5	15	27.3	0.6	10	23.8	0.4	17	34.0	0.6
Other	0	-	-	0	-	-	0	-	-	1	2.4	-	0	-	-
Unknown	2	3.3	-	5	13.2	-	1	1.8	-	2	4.8	-	2	4.0	-
SPA															
1	0	-	-	2	5.3	0.5	1	1.8	0.3	2	4.8	0.5	2	4.0	0.5
2	13	21.7	0.6	5	13.2	0.2	9	16.4	0.4	12	28.6	0.5	14	28.0	0.6
3	8	13.3	0.5	8	21.1	0.5	9	16.4	0.6	1	2.4	0.1	6	12.0	0.4
4	15	25.0	1.2	9	23.7	0.8	9	16.4	0.8	11	26.2	1.0	6	12.0	0.5
5	1	1.7	0.2	3	7.9	0.5	7	12.7	1.1	1	2.4	0.2	1	2.0	0.2
6	10	16.7	0.9	2	5.3	0.2	10	18.2	1.0	6	14.3	0.6	7	14.0	0.7
7	3	5.0	0.2	6	15.8	0.5	6	10.9	0.5	6	14.3	0.5	8	16.0	0.6
8	8	13.3	0.7	3	7.9	0.3	2	3.6	0.2	3	7.1	0.3	6	12.0	0.5
Unknown	2	3.3	-	0	-	-	2	3.6	-	0	-	-	0	-	-



Figure 2. Incidence Rates* of Acute Hepatitis B by Age

Group

Figure 1. Incidences Rates* of Acute Hepatitis B LAC, CA, and US, 2010-2015

*Rates based on fewer than 19 cases are unreliable



HEPATITIS C, ACUTE

CRUDE	CRUDE DATA												
Number of Cases	2												
Annual Incidence ^a													
LA County	0.02ª												
California⁵	0.15												
United States ^b	0.76												
Age at Diagnosis													
Mean	39												
Range	25–53 years												

^aRates calculated based on less than 19 cases or events are considered unreliable

^bCalculated from: CDC. *Notice to Readers:* Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306– 1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

The hepatitis C virus (HCV) is an RNA virus primarily transmitted though percutaneous exposure to infectious blood. Traditional risk factors include: injection drug use (IDU), receipt of a blood transfusion prior to 1992, needle-stick injuries in healthcare settings, birth to infected mothers, multiple sexual partners, tattoos or body-piercing, and hemodialysis. HIV infection is associated with increased risk of infection among men who have sex with men (MSM). Household or familial contact does not appear to increase the risk of transmission of hepatitis C. An estimated 30% of cases have no identifiable exposure risk. Healthcare-related transmission has been documented and should be considered in persons without identified traditional risk factors. HCV is the most common chronic bloodborne infection in the US.

The average incubation period is 4–12 weeks (range 2–24 weeks). Up to 85% of persons with newly acquired HCV infection are asymptomatic. When symptoms occur, they can include: fever, fatigue, loss of appetite, nausea, vomiting, abdominal pain, dark urine, clay-colored bowel movements, joint pain, and jaundice. After acute infection, 15–25% of persons appear to resolve their infection while chronic infection develops in

75–85% of persons. Long-term medical complications occur decades after initial infection including cirrhosis, liver failure, and hepatic cancer.

Primary prevention activities are recommended for prevention and control of HCV infection including: screening and testing of blood donors and persons born 1945-1965, viral inactivation of plasma-derived products, risk-reduction counseling and screening of persons at risk for HCV infection, and routine practice of injection safety in healthcare settings. There is no vaccine or post-exposure prophylaxis for HCV, and vaccines for hepatitis A and B do not provide immunity against hepatitis C. Curative therapy for HCV is available for all HCV genotypes. Limitations to therapy include cost, access to care, and meeting clinical criteria for treatment.

For the purpose of surveillance, LAC DPH uses the 2012 the CDC Council of State and Territorial Epidemiologists (CSTE) criteria for acute hepatitis C:

- 1) Discrete onset of symptoms,
- 2) Jaundice or alanine aminotransferase (ALT) levels >400IU/L,
- a) anti-HCV screening test positive with signal to cut-off ratio predictive of true positive,

b) HCV RIBA positive, or

c) Nucleic acid test (NAT) for HCV RNA positive, and

4) No evidence of either acute hepatitis A or B disease.

In 2012, the CDC/CSTE acute hepatitis C case definition also included documented seroconversion cases as acute hepatitis C cases (documented negative HCV test result within six months prior to HCV diagnosis).

- In 2015, there were only two cases reported, which was lower than that of 2014 with five cases. The rates of acute hepatitis C have been consistently low the past several years.
- The two cases in 2015 were in 15–34 (n=1, 50%) and 45–54 year olds (n=1, 50%) (Figure 2).
- Both cases were Hispanic.



- The CDC/CSTE revised the case definitions for acute and chronic hepatitis C, effective January 1, 2016.
- Risk factors were identified in 50% (n=1) of the confirmed cases interviewed. The

confirmed case had a medical procedure and dental work done 6–12 months prior to the HCV diagnosis.



Reported Hepatitis C, Acute Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=10)		2012 (N=7)			2013 (N=5)				2014 (N=	5)	2015 (N=2)			
	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
1-4	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
5-14	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
15-34	4	40.0	0.1	4	57.1	0.1	2	40.0	0.1	2	40.0	0.1	1	50.0	0.0
35-44	2	20.0	0.1	1	14.3	0.1	1	20.0	0.1	2	40.0	0.2	0	0.0	0.0
45-54	1	10.0	0.1	2	28.6	0.2	1	20.0	0.1	1	20.0	0.1	1	50.0	0.1
55-64	1	10.0	0.1	0	0.0	0.0	1	20.0	0.1	0	0.0	0.0	0	0.0	0.0
65+	2	20.0	0.2	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	1	10.0	0.1	0	0.0	0.0	0	0.0	0.0	1	20.0	0.1	0	0.0	0.0
Black	0	0.0	0.0	1	14.3	0.1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
Hispanic	6	60.0	0.1	3	42.9	0.1	1	20.0	0.0	2	40.0	0.0	2	100.0	0.0
White	2	20.0	0.1	2	28.6	0.1	4	80.0	0.2	2	40.0	0.1	0	0.0	0.0
Other	0	-	-	1	14.3	-	0	-	-	0	-	-	0	-	-
Unknown	1	10.0-	0.1	0	-	-	0	-	-	0	-	-	0	-	-
SPA															
1	0	0.0	0.0	2	28.6	0.5	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
2	1	10.0	0.0	1	14.3	0.0	1	20.0	0.0	3	60.0	0.1	1	50.0	0.0
3	2	20.0	0.1	0	0.0	0.0	1	20.0	0.1	2	40.0	0.1	0	0.0	0.0
4	3	30.0	0.2	1	14.3	0.1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
5	1	10.0	0.2	1	14.3	0.2	1	20.0	0.2	0	0.0	0.0	0	0.0	0.0
6	0	0.0	0.0	1	14.3	0.1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
7	2	20.0	0.1	0	0.0	0.0	1	20.0	0.1	0	0.0	0.0	0	0.0	0.0
8	1	10.0	0.1	1	14.3	0.1	1	20.0	0.1	0	0.0	0.0	1	50.0	0.1
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-







*Rates based on less than 19 cases are considered unreliable.



LEGIONELLOSIS

CRUDE D	ΑΤΑ
Number of Cases	171
Number of Deaths	18
Annual Incidence ^a	
LA County	1.79
California ^b	1.16
United States ^b	1.89
Age at Diagnosis	
Mean	68
Median	70
Range	15–97 years

^aCases per 100,000 population

^bCalculated from: CDC. *Notice to Readers*: Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306– 1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

Legionellosis is a bacterial infection with two distinct clinical forms: 1) Legionnaires' disease (LD), the more severe form characterized by pneumonia, and 2) Pontiac fever, an acute, self-limited influenza-like illness without pneumonia. Legionella bacteria are common inhabitants of aquatic systems that thrive in warm environments. The majority (90%) of LD cases are caused by Legionella pneumophila serogroup 1 (LP1), although at least 46 Legionella species and 70 serogroups have been identified. Transmission occurs through inhalation of aerosolized water containing the bacteria or by aspiration of contaminated water. Person-to-person transmission does not occur. The case-fatality rate for LD ranges from 10-15% but can be higher in outbreaks occurring in a hospital setting. People of any age may get LD. However, the disease most often affects older persons, particularly those who are heavy smokers or have chronic underlying diseases such as diabetes mellitus, congestive heart failure or lung disease or have immune systems that are suppressed by illness or medication.

The implementation of water safety measures to control the risk of transmission of *Legionella* to susceptible hosts in hospitals, hotels, and public

places with water-related amenities remains the primary means of reducing LD. Approaches include periodic inspection of water sources, distribution systems, heat exchangers, and cooling towers. Prevention strategies include appropriate disinfection, monitoring, and maintenance of both cold and hot water systems and setting the hot water temperature to \geq 50°C to limit bacterial growth. All healthcare-associated LD case reports are investigated to identify potential outbreak situations. Early recognition and investigation is crucial for timely implementation of control measures.

- In 2015, there were 171 cases reported (incidence of 1.8/100,000), which was 22% higher than that in 2014 (Figure 1).
- No cases of Pontiac fever were reported.
- The case fatality rate decreased from 15% in 2014 to 10.5% in 2015.
- The most affected age group in LAC was persons <u>>65</u> years old (Figure 2), which is consistent over a five-year period.
- SPA 8 had the highest incidence this year followed by SPA 5 (Figure 3).
- The greatest number of cases was reported in December, which was consistent over the past five years (Figure 4).
- The highest incidence rate occurred among Blacks (3.7 per 100,000) followed by Whites (2.8 per 100,000) (Figure 5).
- Travelers staying overnight in commercial lodging during the incubation period accounted for 9.4% of cases in 2015 compared to 5.0% of cases in 2014. No LAC resident was linked to any multi-state outbreaks reported by CDC.
- Healthcare-associated legionellosis cases in a skilled nursing facility (SNF) decreased from 5.7% to 3.5% of cases with one fatality and from 2.1% to 1.2% of cases in an assisted living facility with one fatality. Healthcare-associated legionellosis cases in an acute care facility decreased from 8.6% to 4.1% of cases.
- One outbreak investigation involved two confirmed cases and one suspect case of legionellosis from the same SNF. All three cases were positive for the *Legionella pneumophila* serogroup 1 urinary antigen. One environmental sample of water collected at the SNF was positive for *Legionella pneumophila* serogroup 1. Additional environmental samples of water were collected at the facility by an environmental



consulting firm, and three were positive for *Legionella pneumophila* serogroup 1. Recommendations were to use disposable water filters and to work with LAC DPH Environmental Health (EH) and an outside consultant to implement a permanent water maintenance plan.

One nosocomial legionellosis outbreak investigation involved two cases in a rehabilitation/ convalescent hospital. Both patients were positive for the *Legionella pneumophila* serogroup 1 urinary antigen. One environmental sample of water collected was positive for *Legionella pneumophila* serogroup 1, and one environmental sample was positive for *Legionella anisa*. Recommendations were to continue working with EH and to follow their suggestion to implement a permanent water maintenance plan.

- One legionellosis outbreak investigation . involved two cases in an assisted living facility. Both residents were positive for the Legionella pneumophila serogroup 1 urinary antigen. The facility also had an ongoing norovirus outbreak at the time of the legionellosis investigation. All environmental samples collected during the site visit were negative Legionella. for Recommendations were made to continue working with EH and to follow their suggestion to implement a permanent water maintenance plan.
- One case was associated with an outbreak in a prison located outside of LAC.



Reported Legionellosis Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	20	011 (N=1	16)	20	12 (N=1	11)	20	013 (N=8	85)	20	14 (N=14	40)	20	15 (N=1)	71)
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
1-4	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
5-14	0	0.0	0.0	1	0.9	0.1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
15-34	5	4.3	0.2	4	3.6	0.1	3	3.5	0.1	3	2.1	0.1	9	5.3	0.3
35-44	7	6.0	0.5	6	5.4	0.5	4	4.7	0.3	11	7.9	0.8	11	6.4	0.8
45-54	21	18.1	1.6	21	18.9	1.6	12	14.1	0.9	17	12.1	1.3	14	8.2	1.1
55-64	22	19.0	2.3	18	16.2	1.8	19	22.4	1.9	29	20.7	2.7	31	18.1	2.8
65+	61	52.6	5.8	61	55.0	5.5	47	55.3	4.2	80	57.1	7.1	106	62.0	8.9
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	8	6.9	0.6	7	6.3	0.5	7	8.2	0.5	16	11.4	1.2	11	6.4	0.8
Black	20	17.2	2.3	16	14.4	2.1	16	18.8	2.1	21	15.0	2.7	29	17.0	3.7
Hispanic	37	31.9	0.8	32	28.8	0.7	24	28.2	0.5	39	27.9	0.8	49	28.7	1.0
White	47	40.5	1.6	49	44.1	1.8	34	40.0	1.3	62	44.3	2.3	76	44.4	2.8
Other	2	1.7	-	5	4.5	-	1	1.2	-	0	-	-	3	1.8	-
Unknown	2	1.7	-	2	1.8	-	3	3.5	-	2	1.4	-	3	1.8	-
SPA															
1	2	1.7	0.5	3	2.7	0.8	2	2.4	0.5	3	2.1	0.8	4	2.3	1.0
2	19	16.5	0.9	21	18.9	1.0	27	31.8	1.2	46	32.9	2.1	38	22.2	1.7
3	15	12.9	0.9	17	15.3	1.1	8	9.4	0.5	16	11.4	1.0	22	12.9	1.3
4	13	11.2	1.0	13	11.7	1.2	18	21.2	1.6	23	16.4	2.0	23	13.5	2.0
5	8	6.9	1.2	10	9.0	1.6	6	7.1	0.9	12	8.6	1.8	16	9.4	2.4
6	23	19.8	2.2	17	15.3	1.7	9	10.6	0.9	10	7.1	1.0	19	11.1	1.8
7	15	12.9	1.1	14	12.6	1.1	3	3.5	0.2	14	10.0	1.1	22	12.9	1.7
8	19	16.4	1.7	14	12.6	1.3	12	14.1	1.1	14	10.0	1.3	27	15.8	2.5
Unknown	2	1.7	0.5	2	1.8	-	0	-	-	2	1.4	-	0	-	-



Figure 2. Incidence Rates of Legionellosis by Age Group LAC, 2011 - 2015



Figure 3. Incidence Rates of Legionellosis by SPA LAC, 2011-2015



Figure 4. Reported Legionellosis Cases by Month of Onset LAC, 2015 (N=171)







Figure 5. Legionellosis Rates by Race/Ethnicity LAC, 2011- 2015

Map 7. Legionellosis Rates by Health District, Los Angeles County, 2015*





LISTERIOSIS, NONPERINATAL

CRUDE DATA									
Number of Cases	34								
Annual Incidence ^a									
LA County ^b	0.36								
California	N/A								
United States	N/A								
Age at Diagnosis									
Mean	67								
Median	71								
Range	32–97 years								

^aCases per 100,000 population

^bRates calculated based on less than 19 cases or events are considered unreliable

DESCRIPTION

Listeriosis is a disease caused by infection with Listeria monocytogenes, a gram-positive rod bacteria found in soil throughout the environment. Listeriosis is often caused by ingestion of foods contaminated with L. monocytogenes such as raw fruits and vegetables, cold cuts, deli meats, and unpasteurized dairy products. The disease affects primarily persons of advanced age, pregnant women, newborns, and adults with weakened immune systems. On rare occasions, people without these risk factors have also contracted listeriosis. Symptoms of listeriosis include: fever, muscle aches, and sometimes nausea or diarrhea. If infection spreads, sepsis or meningitis can occur, which may be fatal. Infected pregnant women may experience only a mild, flulike illness; however, infection during pregnancy can lead to miscarriage or stillbirth, premature delivery, or infection of the newborn.

In general, listeriosis may be prevented by thoroughly cooking raw food from animal sources and avoiding unpasteurized milk or foods made from unpasteurized milk. Individuals at risk for severe outcomes from infection should follow additional recommendations including avoiding soft cheeses and leftover foods or ready-to-eat foods such as deli meats and hot dogs. Deli meats should be cooked until steaming hot before eating.

- Whites comprised 38% of all nonperinatal listeriosis cases followed by Hispanics (26%) and Asians (18%) (Figure 3). In 2015, the proportion of cases among Whites increased by 23% compared to 2014. This year, there were not any reported cases that identified as Black.
- In 2015, three nonperinatal listeriosis cases were part of a nationwide outbreak associated with a Middle Eastern cheese producer. One LAC case was a match for an outbreak associated with onions, but exposure could not be confirmed.
- This year, the number of cases <u>>65</u> years old more than doubled since 2011. Advanced age increases the risk of developing listeriosis.
- Regionally, the greatest number of listeriosis were in SPA 3 (Figure 4) with an incidence rate of 0.6 per 100,000. SPA 2, which historically has a large percentage of cases, had the same incidence rate as that in 2014 (0.4 per 100,000).
- The occurrence of listeriosis cases in 2015 peaked in June (Figure 5) while the fiveyear average peaked at the end of summer and the start of fall.
- Individuals with pre-existing health conditions are disproportionately affected. The majority of cases (n=25, 74%) had one or more other medical conditions before receiving a diagnosis of listeriosis.
- There were five deaths due to nonperinatal listeriosis, resulting in a case-fatality rate of 14.7%. These cases had underlying diseases including cancer, diabetes, kidney disease, and hypertension.



Reported Listeriosis, Nonperinatal Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011(N=19)			2012 (N=26)		2013 (N=23)			2014 (N=27)			2015 (N=34)			
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	/Rate 100,000	No.	(%)	/Rate 100,000	No.	(%)	/Rate 100,000
Age Group															
<1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
1-4	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
5-14	0	0.0	0.0	1	3.8	0.1	0	0.0	0.0	1	3.7	0.1	0	0.0	0.0
15-34	0	0.0	0.0	1	3.8	0.0	0	0.0	0.0	0	0.0	0.0	1	2.9	0.0
35-44	0	0.0	0.0	0	0.0	0.0	1	4.3	0.1	2	7.4	0.2	3	8.8	0.2
45-54	4	21.1	0.3	8	30.8	0.6	3	13.0	0.2	1	3.7	0.1	5	14.7	0.4
55-64	5	26.3	0.5	1	3.8	0.1	3	13.0	0.3	3	11.1	0.3	4	11.8	0.4
65+	10	52.6	0.9	15	57.7	1.4	16	69.6	1.4	20	74.1	1.8	21	61.8	1.8
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	2	10.5	0.1	5	19.2	0.4	7	30.4	0.5	9	33.3	0.7	6	17.6	0.4
Black	0	0.0	0.0	1	3.8	0.1	1	4.3	0.1	1	3.7	0.1	0	0.0	0.0
Hispanic	4	21.1	0.1	8	30.8	0.2	8	34.8	0.2	10	37.0	0.2	9	26.5	0.2
White	13	68.4	0.5	11	42.3	0.4	6	26.1	0.2	4	14.8	0.2	13	38.2	0.5
Other	0	-	-	0	-	-	0	-	-	0	-	-	1	-	-
Unknown	0	-	-	1	-	-	1	-	-	3	-	-	5	-	-
SPA															
1	0	0.0	0.0	1	3.8	0.3	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
2	5	26.3	0.2	9	34.6	0.4	7	30.4	0.3	9	33.3	0.4	8	23.5	0.4
3	4	21.1	0.2	2	7.7	0.1	2	8.7	0.1	5	18.5	0.3	10	29.4	0.6
4	1	5.3	0.1	3	11.5	0.3	4	17.4	0.4	2	7.4	0.2	5	14.7	0.4
5	4	21.1	0.6	5	19.2	0.8	1	4.3	0.2	2	7.4	0.3	3	8.8	0.5
6	0	0.0	0.0	3	11.5	0.3	2	8.7	0.2	3	11.1	0.3	2	5.9	0.2
7	2	10.5	0.1	0	0.0	0.0	5	21.7	0.4	2	7.4	0.2	3	8.8	0.2
8	3	15.8	0.3	3	11.5	0.3	2	8.7	0.2	4	14.8	0.4	3	8.8	0.3
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-



Figure 1. Reported Cases of Nonperinatal Listeriosis LAC, 2006-2015

Figure 3. Percent Cases of Nonperinatal Listeriosis by Race/Ethnicity, 2015 LAC (N=34)



Figure 4. Reported Cases of Nonperinatal Listeriosis by SPA LAC, 2015 (N=34)

Figure 2. Reported Cases of Nonperinatal Listeriosis





Listeriosis, Nonperinatal Page 91


Figure 5. Reported Nonperinatal Listeriosis Cases by Month of Onset LAC, 2015 (N=34)





LISTERIOSIS, PERINATAL

CRUDE DATA									
Number of Cases	3								
Annual Incidence ^a									
LA County ^b	2.58								
California	N/A								
United States	N/A								
Age at Diagnosis									
Mean	33								
Median	N/A								
Range	N/A								

^aCases per 100,000 live births

^bRates calculated based on less than 19 cases or events are considered unreliable

DESCRIPTION

Listeriosis is a disease caused by infection with *Listeria monocytogenes*, a gram-positive rod bacteria found in soil throughout the environment. Listeriosis is often caused by ingestion of foods contaminated with *L. monocytogenes*. Foods often associated with *Listeria* contamination include raw fruits and vegetables, undercooked meat such as beef, pork, poultry, and pâté, cold cuts, and unpasteurized dairy products such as milk, milk products, and soft cheeses (Mexican-style, Brie, feta, blue-veined, Camembert).

Pregnant women are susceptible because pregnancy causes a suppression of the immune system. The pregnant mother may only experience a mild febrile illness but can transmit the infection to the fetus. Symptoms of listeriosis include fever, muscle aches, and sometimes nausea or diarrhea. Infections during pregnancy can lead to miscarriage, stillbirth, premature delivery, or infection of the newborn. Often, *Listeria* can be isolated from both the mother and infant. Pregnant women should avoid foods associated with *Listeria*, particularly cheeses sold by street vendors or obtained from relatives/friends in countries where food processing quality assurance is unknown. Leftover foods or ready-toeat foods such as hot dogs should be cooked until steaming hot before eating.

Prevention strategies include education during prenatal checkups, outreach to Latino communities more likely to consume soft cheese, and food safety notices at food and deli markets.

- In 2015, there were three perinatal motherinfant pairs with listeriosis. Two cases were Hispanic, and one case was White. All three cases were single gestations.
- Two mothers were not diagnosed with listeriosis, but their infants tested positive. One mother tested positive, but *Listeria* was not cultured from her infant.
- Maternal ages were 20-45 years old with a mean of 33 years old.
- The number of perinatal listeriosis cases in 2015 is consistent with the range of incidence of listeriosis over the past ten years (2006– 2015, excluding the increase in 2006 when there were 12 cases (Figure 1)).
- Hispanic women had the highest number of cases of perinatal listeriosis, consistent with the past five years, except 2012 when non-Hispanic White mothers comprised the majority of cases (Figure 2). Incidence of perinatal listeriosis remains consistent among Hispanic mothers. There have been no cases of perinatal listeriosis in Black expectant mothers since 2006.
- One of the mothers reported eating cold cuts, and two reported eating soft cheeses while pregnant.
- All three mothers were hospitalized and released. There were no maternal or neonatal deaths.



Reported Perinatal Listeriosis Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=6)			:	2012 (N=7))		2013 (N=4))	2	2014 (N=5)		2015 (N=3)		
	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000	No.	(%)	/Rate 100,000	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
1-4	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
5-14	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
15-34	3	50.0	3.1	4	57.1	4.2	4	100.00	4.3	3	60.0	3.2	2	66.7	2.2
35-44	3	50.0	12.3	3	42.9	11.7	0	0.0	0.0	2	40.0	7.3	1	33.3	3.7
45-54	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
55-64	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
65+	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
Unknown	0	-	-	0	0.0	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	2	33.3	13.1	1	14.3	5.4	0	0.0	0.0	1	20.0	4.6	0	0.0	0.0
Black	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
Hispanic	3	50.0	4.1	2	28.6	2.8	3	75.0	4.4	2	40.0	3.0	2	66.7	3.4
White	1	16.7	4.6	4	57.1	18.6	1	25.0	4.5	1	20.0	4.5	1	33.3	4.5
Other	0	-	-	0	-	-	0	-	-	0	0.0	0.0	0	-	-
Unknown	0	-	-	0	-	-	0	-	-	1	20.0	-	0	-	-
SPA															
1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
2	0	0.0	0.0	2	28.6	0.2	1	25.0	0.2	1	20.0	0.2	0	0.0	0.0
3	3	50.0	0.4	2	28.6	0.3	1	25.0	0.3	1	20.0	0.3	1	33.3	0.1
4	0	0.0	0.0	1	14.3	0.2	0	0.0	0.0	1	20.0	0.4	0	0.0	0.0
5	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
6	1	16.7	0.2	0	0.0	0.0	0	0.0	0.0	1	20.0	0.4	1	33.3	0.2
7	0	0.0	0.0	1	14.3	0.2	1	25.0	0.3	0	0.0	0.0	0	0.0	0.0
8	2	33.3	0.4	1	14.3	0.2	1	25.0	0.4	1	20.0	0.5	1	33.3	0.2
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-



Figure 1. Reported Cases of Perinatal Listeriosis







MENINGITIS, VIRAL

CRUDE DATA										
Number of Cases	367									
Annual Incidence ^a										
LA County	3.83									
Age at Diagnosis										
Mean	35									
Median	32									
Range	0–94 years									

^aCases per 100,000 population

DESCRIPTION

Viruses are the major cause of aseptic meningitis syndrome, a term used to define any meningitis (infectious or noninfectious), particularly one with a cerebrospinal fluid lymphocytic pleocytosis for which a cause is not apparent after initial evaluation and routine stains and cultures do not support a bacterial or fungal etiology. Viral meningitis can occur at any age but is most common among the very young. Symptoms are characterized by sudden onset of fever, severe headache, stiff neck, photophobia, drowsiness, confusion, nausea, and vomiting and usually last from seven to ten days.

The most common cause of viral meningitis is nonpolio enteroviruses, which are not vaccinepreventable and account for 85-95% of all cases in which a pathogen is identified. Transmission of enteroviruses may be by fecal-oral, respiratory, or other route specific to the etiologic agent. Other viral agents that can cause viral meningitis include herpes simplex virus (HSV), varicellazoster virus (VZV), mumps virus, lymphocytic choriomeningitis virus, human immunodeficiency virus, adenovirus, parainfluenza virus type 3, influenza virus, measles virus, and arboviruses such as West Nile virus (WNV).

Antiviral agents are available for HSV and VZV; however, in most cases, only supportive measures are available for the treatment of viral meningitis. Recovery is usually complete and associated with low mortality rates. Several types of viral meningitis cases are vaccine-preventable including those caused by VZV, mumps, influenza, and measles. Good personal hygiene, especially hand washing and avoiding contact with oral secretions of others, is the most practical and effective preventive measure for non-vaccine preventable causes.

- In 2015, viral/aseptic meningitis incidence declined from 4.2 cases per 100,000 in 2014 to 3.8 cases per 100,000. There had been a rise in incidence each year between 2012 and 2014 (Figure 1).
- SPA 1 (Antelope Valley) continued to report the highest rate of viral meningitis in LAC with 6.8 cases per 100,000 in 2015 followed by SPA 3 (San Gabriel Valley) with 4.3 cases per 100,000 (Figure 2).
- The distribution of viral/aseptic meningitis by age groups remains similar to previous years with the <1 year old age group experiencing the highest age-specific incidence rate at 37.9 per 100,000 (Figure 3).
- The peak number of cases occurred in September (n=71, 19%) and follows the typical seasonal trend for enteroviral and WNV meningitis, which comprise the majority of viral meningitis cases (Figure 4).
- The etiologies of 174 cases were identified (47%). Of those, nearly two-thirds (n=107, 61%) were identified as WNV, and one quarter (n=44, 25%) were identified as enterovirus (Figure 6).
- Four fatalities were reported (1%). Of these, three were associated with WNV, and the fourth was due to an unknown etiology. No outbreaks were documented.



Reported Viral Meningitis Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	20	11 (N=31	17)	20'	12 (N=30)3)	20	13 (N=3	55)	20	14 (N=40)0)	2015 (N=367)			
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	
Age Group																
<1	33	10.4	23.6	28	9.2	23.5	43	12.1	35.6	47	11.8	39.7	41	11.2	37.9	
1-4	6	1.9	1.0	4	1.3	0.8	9	2.5	1.8	8	2.0	1.6	2	0.5	0.4	
5-14	53	16.7	4.0	24	7.9	2.0	57	16.1	4.7	54	13.5	4.5	51	13.9	4.2	
15-34	102	32.2	3.5	93	30.7	3.4	105	29.6	3.7	114	28.5	4.0	101	27.5	3.6	
35-44	39	12.3	2.7	45	14.9	3.4	27	7.6	2.0	43	10.8	3.3	38	10.4	2.9	
45-54	41	12.9	3.0	40	13.2	3.1	44	12.4	3.4	43	10.8	3.3	41	11.2	3.1	
55-64	24	7.6	2.5	32	10.6	3.1	35	9.9	3.4	42	10.5	4.0	42	11.4	3.8	
65+	18	5.7	1.7	37	12.2	3.3	31	8.7	2.8	44	11.0	3.9	51	13.9	4.3	
Unknown	1	0.3	-	0	-	-	4	1.1	-	5	1.3	-	0	-	-	
Race/Ethnicity																
Asian	21	6.6	1.6	23	7.6	1.7	21	5.9	1.5	22	5.5	1.6	21	5.7	1.5	
Black	37	11.7	4.3	36	11.9	4.7	26	7.3	3.3	26	6.5	3.3	24	6.5	3.1	
Hispanic	147	46.4	3.1	131	43.2	2.9	158	44.5	3.4	186	46.5	4.0	174	47.4	3.7	
White	78	24.6	2.7	86	28.4	3.2	88	24.8	3.3	99	24.8	3.7	106	28.9	3.9	
Other	7	2.2	-	10	3.3	-	19	5.4	-	12	3.0	-	8	2.2	-	
Unknown	27	8.5	-	17	5.6	-	43	12.1	-	55	13.8	-	34	9.3	-	
SPA																
1	33	10.4	8.8	18	5.9	4.6	29	8.2	7.4	33	8.3	8.4	27	7.4	6.8	
2	67	21.1	3.0	63	20.8	2.9	67	18.9	3.1	73	18.3	3.3	68	18.5	3.1	
3	75	23.7	4.3	68	22.4	4.2	64	18.0	3.9	97	24.3	5.9	71	19.3	4.3	
4	14	4.4	1.1	16	5.3	1.4	32	9.0	2.8	34	8.5	3.0	31	8.4	2.7	
5	15	4.7	2.3	10	3.3	1.6	7	2.0	1.1	14	3.5	2.1	20	5.4	3.0	
6	26	8.2	2.4	29	9.6	2.9	43	12.1	4.2	38	9.5	3.7	43	11.7	4.1	
7	48	15.1	3.5	57	18.8	4.4	56	15.8	4.3	71	17.8	5.4	71	19.3	5.4	
8	35	11.0	3.1	36	11.9	3.4	52	14.6	4.8	37	9.3	3.4	33	9.0	3.0	
Unknown	4	1.3	-	6	2.0	-	5	1.4	-	3	0.8	-	3	0.8	-	



Figure 3. Incidence Rates of Viral Meningitis by Age Group LAC, 2011-2015





Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec Month

E 2015 — Five-year average

*35 cases missing onset date.







Figure 6. Percent Cases of Viral Meningitis by Etiology, LAC, 2015 (N=367)



Map 8. Meningitis, Viral Rates by Health District, Los Angeles County, 2015*







MENINGOCOCCAL DISEASE

CRUDE DATA										
Number of Cases	12									
Annual Incidence ^a										
LA County	0.13									
California⁵	0.12									
United States ^b	0.12									
Age at Diagnosis										
Mean	47									
Median	46									
Range	16–84 years									

^aCases per 100,000 population.

^bCalculated from: CDC. *Notice to Readers*: Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306– 1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

Meningococcal disease (MD) occurs most often as meningitis, an infection of the cerebrospinal fluid (CSF), or meningococcemia, an infection of the bloodstream. It is transmitted through direct or droplet contact with nose or throat secretions of persons colonized in the upper respiratory tract with Neisseria meningitidis bacteria. Common symptoms include sudden onset of fever, headache, nausea, vomiting, stiff neck, petechial rash, and lethargy, which can progress to overwhelming sepsis, shock, and death within hours. Despite effective antibiotic therapy, the mortality rate remains between 10-15%. Long-term sequelae include significant neurologic or orthopedic complications such as deafness or amputation. Meningococcal disease affects all age groups but occurs most often in infants. Of the 13 serogroups, A, B, C, Y, and W-135 are responsible for causing nearly all cases of meningococcal disease.

For the purpose of surveillance, the LAC DPH defines reports of invasive meningococcal disease as confirmed when *N. meningitidis* has been isolated from or evidenced by polymerase chain reaction (PCR) analysis in a normally sterile site (e.g., blood or CSF). In the absence of a positive culture, reports are defined as probable if the *N. meningitidis* antigen

is detected by immunohistochemistry or latex agglutination. Reports are classified as suspected cases when they present with clinical diagnosis of purpura fulminans or demonstrate gram-negative diplococci by gram staining [1].

Both suspected clinical cases of MD and laboratory findings consistent with MD are immediately reportable to the public health department. All cases are investigated by public health nurses within the district corresponding to the home of residence. A standardized case report is completed. In December 2012, in addition to the standardized case report form, a supplemental form documenting additional risk factors was included in the investigation. Additional risk factors such as sexual history (men who have sex with men [MSM]) and travel history were documented due to the ongoing outbreak of MD among MSM in New York City in 2011-2012 [2].

A total of four vaccines have been made available in the US that protect against serogroups A. C. Y. W-135. A guadrivalent unconjugate and polysaccharide meningococcal vaccine (MPSV4) is licensed for persons >55 years old and \geq 2 years old when a quadrivalent conjugate polysacharide vaccine is not available. Two guadrivalent conjugate vaccines, MenACWY-D and MenACWY-CRM, are licensed for use in persons 2-55 years old. MenACWY-D is also licensed for used in children 9-23 months old. Both vaccines are recommended for all adolescents between 11-18 years old, preferably at 11 or 12 years old, and for those 2-55 years old who are at increased risk for meningococcal disease. An additional booster dose is needed if the primary dose was given before 16 years old. Routine vaccination is recommended for college freshman living in dormitories, persons at increased risk for meningococcal disease. An additional conjugate vaccine, Hib-MenCY-TT, has been licensed for infants 6 weeks to 18 months old but only protects against serogroups C and Y disease [3]. Two serogroup B vaccines, MenB-FHbp and MenB-4C, were approved for use in persons 10-25 years old [4].

In addition to ACIP recommended groups, DPH has recommended meningococcal vaccination for MSM at increased risk for IMD since 2014. The vaccine should be offered to:

All HIV-infected gay/MSM

• Gay/MSM, regardless of HIV status, who regularly have close or intimate contact with multiple partners or who seek partners through the use of digital applications ("apps"), particularly those who share cigarettes or marijuana or use illegal drugs

Antimicrobial chemoprophylaxis of close contacts of sporadic cases of MD remains the primary means for prevention of MD among close contacts. This includes:

- a) Household members,
- b) Daycare center contacts, and

c) Anyone directly exposed to the patient's oral secretions (e.g., through kissing, mouth-to-mouth resuscitation, endotracheal intubation, or endotracheal tube management)

Because the rate of secondary disease for close contacts is highest during the first few days after onset of disease in the primary patient, antimicrobial chemoprophylaxis should be administered as soon as possible—ideally within 24 hours after the case is identified. Conversely, chemoprophylaxis administered >10 days after onset of illness in the index case-patient is probably of limited or no value. Prophylactic treatment and follow-up of close contacts are routinely handled by the LAC DPH Community Health Services.

2015 TRENDS AND HIGHLIGHTS

- The incidence of MD in LAC has followed the national incidence for the past decade and continues to decrease from a peak of 0.6 cases per 100,000 in 2001 to 0.1 cases per 100,000 in 2015 (Figure 1). In 2015, LAC documented one of the lowest incidence and case counts with only 12 cases.
- There were no cases reported among persons less than 15 years old in 2015. The highest number of cases (n=6, 55%) occurred among those 15-34 years old (Figure 2). This has been the trend for previous five years. However, in a typical distribution curve depicting incidence for MD, the peak incidence occurs among infants <1 year old. There have been no cases of MD in children <1 year old since 2010.
- The monthly onset of disease deviated from the typical seasonal trend of peaking in the winter season. The highest numbers of cases occurred in January and April with four cases each (Figure 4).
- Culture confirmation was obtained for 10 of the 12 cases (83%), and of these, seven (70%) were cultured from blood, two (20%) from blood and CSF, and one (10%) from synovial fluid.

- Two cases were not serotyped: one due to the specimen being discarded and the other due to a negative culture and PCR test (case was diagnosed by gram stain). The majority of serotyped cases were serogroup B (n=6, 60%), two (20%) were serogroup C, and two (20%) were serogroup W-135 (Figure 6). The proportion of serogroup B cases in LAC has increased since 2013. Seven outbreaks of serogroup B disease have occurred on college campuses since 2009 in the US. However, the incidence of serogroup B disease in young adults 18-23 years old remains low (0.1 per 100,000), and no outbreaks or increases in college students or that age group have been documented within LAC [5].
- No fatalities were documented this year. In contrast, LAC documented a 27% case fatality rate (n=3) in 2014.
- Though no outbreaks occurred within LAC, a serogroup B case who reported travel to and exposure to students at the University of Oregon was confirmed to have a strain indistinguishable from the strain associated with an outbreak occurring there.
- An increase of MD among MSM occurred between October 2012 and September 2014 (n=13). Due to increases in fatalities and HIV co-morbid cases, among other factors, LAC DPH recommended vaccination against MD among certain risk groups in the MSM community beginning April 2014. The number of MD cases among MSM has declined since then with only one case in 2015. However, LAC DPH continues to endorse the recommendation.

References

- Centers for Disease Control and Prevention. National Notifiable Disease Surveillance System. Meningococcal Disease (*Neisseria meningitidis*), 2015 Case Definition. https://wwwn.cdc.gov/nndss/conditions/meni ngococcal-disease/case-definition/2015/. Accessed: June 8, 2016.
- Centers for Disease Control and Prevention. Notes from the field: serogroup C invasive meningococcal ease among men who have sex with men – New York City, 2010-2012. Morbidity and Mortality Weekly Report. 4 Jan 2013; 61(51): 1048.
- Centers for Disease Control and Prevention. Morbidity and Mortality Weekly Report. Prevention and Control of Meningococcal Disease, Recommendations of the Advisory Committee on Immunization Practices (ACIP). 22 Mar 2013, 62 (2): 1-28.



- Centers for Disease Control and Prevention. Morbidity and Mortality Weekly Report. Use of Serogroup B Meningococcal Vaccines in Persons Aged ≥10 Years at Increased Risk for Serogroup B Meningococcal Disease: Recommendations of the Advisory Committee on Immunization Practices (ACIP). 2015. 12 Jun 2015, 64 (22): 608-12.
- 5. Centers for Disease Control and Prevention. Morbidity and Mortality Weekly Report. Use of Serogroup B Meningococcal Vaccines in Adolescents and Young Adults: Recommendations of the Advisory Committee on Immunization Practices (ACIP), 2015. 23 Oct 2015, 64 (41): 1171-6.



Reported Meningococcal Disease Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=37)			20	12 (N=1	2)	2013 (N=17)			20	14 (N=1	1)	2015 (N=12)		
	No.	(%)	Rate/ 100,000	No.	(%)	/Rate 100,000	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
1-4	1	2.7	0.2	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
5-14	1	2.7	0.1	0	0.0	0.0	1	5.9	0.1	0	0.0	0.0	0	0.0	0.0
15-34	12	32.4	0.4	4	33.3	0.1	7	41.2	0.2	6	54.5	0.2	4	33.3	0.1
35-44	10	27.0	0.7	0	0.0	0.0	3	17.6	0.2	1	9.1	0.1	1	8.3	0.1
45-54	3	8.1	0.2	2	16.7	0.2	2	11.8	0.2	3	27.3	0.2	3	25.0	0.2
55-64	5	13.5	0.5	2	16.7	0.2	1	5.9	0.1	1	9.1	0.1	1	8.3	0.1
65+	5	13.5	0.5	4	33.3	0.4	3	17.6	0.3	0	0.0	0.0	3	25.0	0.3
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	4	10.8	0.3	2	16.7	0.2	0	-	-	2	18.2	0.1	0	0.0	0.0
Black	12	32.4	1.4	2	16.7	0.3	4	23.5	0.5	2	18.2	0.3	2	16.7	0.3
Hispanic	11	29.7	0.2	5	41.7	0.1	6	35.3	0.1	6	54.5	0.1	6	50.0	0.1
White	10	27.0	0.3	3	25.0	0.1	6	35.3	0.2	1	9.1	0.0	4	33.3	0.1
Other	0	-	-	0	-	-	1	5.9	-	0	-	-	0	-	-
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
SPA															
1	1	2.7	0.3	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	1	8.3	0.3
2	9	24.3	0.4	2	16.7	0.1	5	29.4	0.2	3	27.3	0.1	4	33.3	0.2
3	2	5.4	0.1	0	0.0	0.0	1	5.9	0.1	1	9.1	0.1	0	0.0	0.0
4	5	13.5	0.4	5	41.7	0.4	4	23.5	0.4	6	54.5	0.5	3	25.0	0.3
5	1	2.7	0.2	2	16.7	0.3	2	11.8	0.3	0	0.0	0.0	1	8.3	0.2
6	9	24.3	0.8	3	25.0	0.3	1	5.9	0.1	0	0.0	0.0	2	16.7	0.2
7	4	10.8	0.3	0	0.0	0.0	3	17.6	0.2	0	0.0	0.0	1	8.3	0.1
8	6	16.2	0.5	0	0.0	0.0	1	5.9	0.1	1	9.1	0.1	0	0.0	0.0
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-



*Rates calculated based on less than 19 cases or events are considered unreliable.



Figure 3. Meningococcal Disease Cases by Race/Ethnicity, LAC, 2011-2015





Figure 4. Reported Meningococcal Disease Cases by Month of Onset, LAC, 2015 (N=12)



Figure 5. Meningococcal Disease Cases by SPA LAC, 2011-2015



Figure 6. Meningococcal Disease by Serogroup LAC, 2011–2015

*Among cases with known serogroup.



PNEUMOCOCCAL DISEASE, INVASIVE

CRUDE	DATA						
Number of Cases	468						
Annual Incidence ^a							
LA County	4.89						
California⁵	N/A						
United States ^c	5.03						
Age at Diagnosis							
Mean	56						
Median	60						
Range	0–100 years						

^aCases per 100,000 population ^bNot potifiable

^bNot notifiable

^eCalculated from: CDC. *Notice to Readers:* Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306– 1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm <u>Note</u>: LA County utilizes passive surveillance in all age groups. Passive surveillance is not comparable to US rates due to difference in surveillance methodology.

DESCRIPTION

Invasive pneumococcal disease (IPD) is a leading cause of illness in young children and causes considerable illness and death in the elderly. The infectious agent *Streptococcus pneumoniae* is spread by direct and indirect contact with respiratory secretions and can cause pneumonia, bacteremia, meningitis, and death. *S. pneumoniae* is one of the most common bacterial causes of community acquired pneumonia and otitis media (ear infections). However, these non-invasive forms of infection (except bacteremic community acquired pneumonia) are not counted in LAC surveillance. Therefore, the data presented in this report underestimate all disease caused by *S. pneumoniae* in LAC.

ACDC has been tracking IPD as part of a special antibiotic resistance surveillance project since late 1995 and added IPD to its list of reportable diseases in October 2002. Cases are defined as LAC residents with a positive *S. pneumoniae* isolate collected from a normally sterile site (e.g., blood, cerebrospinal fluid).

In 2010, ACDC was awarded a grant from the CDC to evaluate the effectiveness of the 13-valent pneumococcal conjugate vaccine (Prevnar13®) among children 2-59 months old. This led to substantial improvements in IPD surveillance data quality for surveillance years 2010 to 2014. However, decreases in funding and staff resources have led to declines in data quality for surveillance year 2015.

Pneumococcal isolates from persons with IPD are sent to the LAC Public Health Laboratory to assess antimicrobial susceptibility, determined by disk or dilution diffusion. Minimum inhibitory concentration (MIC) breakpoints used by participating laboratories are based on standards developed by the Clinical and Laboratory Standards Institute. For this report, an isolate of *S. pneumoniae* is considered non-susceptible to an antibiotic if the results indicate intermediate or high-level resistance.

Two effective vaccines are available to prevent pneumococcal disease. First, Prevnar13[®] is recommended for all children 2-59 months old, children \geq 6 years old with certain risk factors for invasive pneumococcal infections, and adults \geq 65 years old. Second, the 23-valent pneumococcal polysaccharide vaccines (Pnu-Imune[®]23 and Pneumovax[®]23) are recommended for all adults \geq 65 years old and those <2 years old who are at high risk of IPD.

- The incidence rate this year of 4.9 cases per 100,000 people was lower than the average annual incidence of 5.8 cases per 100,000 people over the past five years (range 4.9-7.1 cases per 100,000) (Figure 1) and is similar to last year's rate (4.9 cases per 100,000).
- Mortality in 2015 (n=42 deaths, 15.6%) was fairly consistent compared to the annual mortality during the past five years, which ranged from 12.8% to 17.3% among cases with known disease outcome.
- In 2015, 84% of reported cases were hospitalized, which is similar to the previous five-year average of 93%.
- Incidence rates decreased amongst all age groups, compared to the previous five-year average (Figure 2). Among cases <1 year old, the incidence rate decreased 39% (from 7.5 to 4.6



cases per 100,000). This age group is part of the target population for the 13-valent pneumococcal conjugate vaccine released in the spring of 2010. The decrease in incidence in this age group is indicative of vaccine effectiveness (Figure 2).

- All age groups decreased despite no or little conjugate vaccination, indicating decreased transmission in the overall population (Figure 2).
- Cases ≥65 years old and 55-64 years old had the highest incidence rates (16.2 and 9.3 cases per 100,000, respectively) (Figure 2). High rates among the elderly may be indicative of lower vaccination rates among the elderly (≥65 years old) compared to children >5 years old. Alternatively, the elderly may be affected by different serotypes than are contained in the vaccines. More research is required to further assess this.
- Incidence rates decreased across all race/ethnic groups (Figure 3) compared to the previous five years.
- Consistent with previous years, the 2015 incidence rate in Blacks was substantially higher than rates among all other race/ethnic groups (Figure 3).
- Similar to previous years, SPA 6 had the highest incidence rate of IPD (7.3 cases per 100,000) (Figure 4). Compared to the rest of LAC, SPA 6 historically has had a high number of Hispanics and Blacks in addition to high numbers of individuals with low income and lack of access to

care. These may be contributory factors for the high number of cases in this SPA. More data is needed to study this [1, 2].

- In all SPAs, the incidence rate was lower than that of the previous five-year average. The largest incidence rate decrease among the SPAs occurred in SPA 5. This incidence rate decreased by 25% (from 5.2 to 3.9 cases per 100,000) compared to the previous five-year average (Figure 4).
- The percentage of isolates susceptible to penicillin, erythromycin, cefotaxime, ceftriaxone, levofloxacin, and TMP-SMZ was fairly consistent with the previous five years (Figure 6).

References

- 1. Accessed on 7/21/2015 from the Los Angeles County Department of Public Health, LA HealthDataNow!: https://dgs.publichealth.lacounty.gov/
- Senterfitt JW, Long A, Shih M, Teutsch SM. How Social and Economic Factors Affect Health. Social Determinants of Health, Issue no.1. Los Angeles: Los Angeles County Department of Public Health; January 2013.
- Active Bacterial Core Surveillance Reports from 2005 to 2014 from the Centers for Disease Control and Prevention's Division of Bacterial Diseases. Report available at: www.cdc.gov/abcs/reports-findings/survreports.html



Reported Invasive Pneumococcal Disease Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=658)		201	12 (N=50	04)	20	13 (N=52	25)	201	4 (N=4	60)	2015 (N=468)			
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	7	1.1	5.9	13	2.6	10.9	7	1.3	5	7	1.5	5.9	5	1.1	4.6
1-4	36	5.5	7.5	24	4.8	5.1	24	4.6	4.9	18	3.9	3.7	27	5.8	5.6
5-14	31	4.7	2.6	17	3.4	1.4	23	4.4	1.9	12	2.6	1.0	18	3.8	1.5
15-34	64	9.7	2.3	32	6.3	1.2	32	6.1	1.1	31	6.7	1.1	33	7.1	1.2
35-44	57	8.7	4.3	38	7.5	2.9	40	7.6	2.9	42	9.1	3.2	31	6.6	2.3
45-54	107	16.3	8.3	82	16.3	6.4	63	12.0	4.9	65	14.1	5.0	58	12.4	4.4
55-64	128	19.5	12.9	89	17.7	8.7	108	20.6	10.5	97	21.1	9.1	103	22.0	9.3
65+	227	34.5	21.5	209	41.5	18.8	228	43.4	20.5	188	40.9	16.6	193	41.2	16.2
Unknown	1	0.2	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	49	7.4	3.7	36	7.1	2.7	32	6.1	2.3	34	7.4	2.5	29	6.2	2.1
Black	130	19.8	16.8	96	19.0	12.4	96	18.3	12.2	70	15.2	8.9	87	18.6	11.1
Hispanic	244	37.1	5.4	192	38.1	4.2	209	39.8	4.5	161	35.0	3.5	132	28.2	2.8
White	234	35.6	8.8	172	34.1	6.5	174	33.1	6.5	154	33.5	5.8	119	25.4	4.4
Other	0	-	-	0	-	-	0	-	-	15	3.3	-	14	3.0	-
Unknown	1	0.2	-	8	1.6		14	2.7	-	26	5.7	-	87	18.6	-
SPA															
1	31	4.7	8.0	18	3.6	4.6	25	4.8	6.4	16	3.5	4.1	18	3.8	4.5
2	117	17.8	5.5	111	22.0	5.2	99	18.9	4.5	102	22.2	4.7	72	15.4	3.2
3	85	12.9	5.3	79	15.7	4.9	75	14.3	4.6	66	14.3	4.0	64	13.7	3.9
4	87	13.2	7.8	72	14.3	6.4	66	12.6	5.8	55	12.0	4.8	69	14.7	5.9
5	49	7.4	7.7	28	5.6	4.4	20	3.8	3.1	25	5.4	3.8	26	5.6	3.9
6	86	13.1	8.5	72	14.3	7.1	74	14.1	7.2	60	13.0	5.8	77	16.5	7.3
7	81	12.3	6.3	54	10.7	4.1	73	13.9	5.5	56	12.2	4.3	59	12.6	4.5
8	94	14.3	8.9	57	11.3	5.3	75	14.3	6.9	53	11.5	4.9	61	13.0	5.6
Unknown	28	4.3	-	13	2.6	-	18	3.4	-	27	5.9	-	22	4.7	-



*US incidence rate estimate from Active Bacterial Core

Figure 3. Annual Incidence Rates of Invasive Pneumococcal Disease by Race/Ethnicity, LAC, 2010-2015*



*For 2010, 2011, 2012, 2013, 2014, and 2015 total numbers of cases (and percent with race-ethnicity missing) were 576 (4%), 658 (0%), 504 (2%), 525 (3%), 460 (10%), and 468 (19%), respectively.





Figure 4. Annual Incidence Rates of Invasive Pneumococcal Disease by SPA, LAC, 2010-2015





Figure 5. Invasive Pneumococcal Disease Cases by Month of

*Range of number of isolates tested 2010-2015: Cefotaxime (212-316), Ceftriaxone (328-431), Erythromycin (218-363), Levofloxacin (240-367), Penicillin (391-596), and TMP-SMZ (193-279).



Map 9. Pneumococcal Disease, Invasive (IPD) Rates by Health District, Los Angeles County, 2015*



SALMONELLOSIS

CRUDE DATA										
Number of Cases	1,144									
Annual Incidence ^a										
LA County	11.95									
California⁵	14.21									
United States ^b	17.15									
Age at Diagnosis										
Mean	34									
Median	31									
Range	<0–95 years									

^aCases per 100,000 population

^bCalculated from: CDC. *Notice to Readers*: Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly*/November 25, 2016/65(46);1306–1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

Salmonellosis is caused by the gram-negative bacillus Salmonella enterica, of which there are more than 2,500 serotypes. This disease is transmitted by the fecal-oral route, from animal or human, and with or without intermediary contamination of foodstuffs. The most common symptoms include diarrhea, fever, headache, abdominal pain, nausea and sometimes vomiting. Occasionally, the clinical course is that of enteric fever or septicemia. Asymptomatic infections may occur. The incubation period is usually 12 to 36 hours for gastroenteritis and longer and variable for other manifestations. Communicability lasts as long as organisms are excreted, usually 2-5 weeks, but may last for months to years. Healthy people are susceptible, but persons especially at risk are those who are on antacid therapy, who have recently taken or are taking broad-spectrum antibiotic therapy or immunosuppressive therapy, or who have had gastrointestinal surgery, neoplastic disease, or other debilitating conditions. Severity of the disease is related to the serotype, the number of organisms ingested, and host factors. Immunocompromised persons such as those with cancer or HIV infection are at risk for recurrent Salmonella septicemia. Occasionally, the organism may localize anywhere in the body, causing abscesses, osteomyelitis, arthritis, meningitis, endocarditis, pericarditis, pneumonia, or pyelonephritis. LAC DPH's review of investigation reports indicates that many cases engaged in high-risk food handling behaviors such as consumption of raw or undercooked meats, use of raw eggs, not washing hands and/or cutting boards after handling raw poultry or meat, and having contact with reptiles. Travel is also a risk factor for salmonellosis. LAC cases report domestic, national, and international travel.

- Three LAC salmonellosis outbreaks were investigated by ACDC in 2015; all were foodborne outbreaks. For more information see the Foodborne Illness Outbreak summary in this ACDC Annual Morbidity Report 2015.
- By age group, the highest incidence rate (55.5 cases per 100,000) was seen in those who were less than one year old (Figure 2).
- In 2015 and in prior years, the highest incidence rates by race/ethnicity occurred among Whites and Hispanics (Figure 3).
- Incidence rates by SPA ranged from 8.8 in SPA 1 to 17.3 in SPA 5 (Figure 4).
- Travel was reported by 17.9% of the cases. Approximately one-third of the cases (34.6%) traveled to Mexico or countries other than Mexico (31.7%).
- Reptile-associated salmonellosis accounted for 5.9% of cases in 2015. Among these cases, 67.1% were related to turtle exposures, and 20.9% were related to lizard exposures. In addition, 17 LAC residents were part of a national outbreak related to reptile-associated salmonellosis exposures.
- Nearly one-fourth (23.0%) of cases were hospitalized for two or more days.
- There were eight deaths in persons diagnosed with salmonellosis. Ages ranged from 56 to 80 years with a mean of 69 and median of 72 years. All eight cases had comorbidities.



Reported Salmonellosis Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=900)			20	12 (N=1,04	¥1)	2013 (N=1,010)			201	4 (N=1,1	41)	2015 (N=1,144)		
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	61	6.8	43.7	73	7.0	61.4	59	5.8	48.8	62	5.4	52.4	60	5.2	55.5
1-4	134	14.9	23.1	153	14.7	32.2	141	14.0	29.0	162	14.2	33.2	116	10.1	23.9
5-14	148	16.4	11.1	158	15.2	13.2	185	18.3	15.3	181	15.9	15.0	148	12.9	12.2
15-34	186	20.7	6.3	224	21.5	8.1	227	22.5	8.0	248	21.7	8.8	297	26.0	10.5
35-44	93	10.3	6.5	95	9.1	7.2	89	8.8	6.7	110	9.6	8.3	123	10.8	9.3
45-54	86	9.6	6.4	108	10.4	8.4	82	8.1	6.3	111	9.7	8.5	124	10.8	9.4
55-64	86	9.6	8.9	88	8.5	8.6	84	8.3	8.2	99	8.7	9.3	105	9.2	9.5
65+	106	11.8	10.0	142	13.6	12.8	143	14.2	12.9	168	14.7	14.8	171	14.9	14.3
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	64	7.1	4.8	92	8.8	7.0	73	7.2	5.3	140	12.3	10.2	102	8.9	7.3
Black	53	5.9	6.2	56	5.4	7.2	69	6.8	8.9	67	5.9	8.5	68	5.9	8.7
Hispanic	464	51.6	9.8	503	48.3	11.1	538	53.3	11.7	575	50.4	12.5	589	51.5	12.6
White	279	31.0	9.7	247	23.7	9.3	318	31.5	12.0	344	30.1	12.9	383	33.5	14.3
Other	8	0.9	-	11	1.1	-	5	0.5	-	10	0.9	-	2	0.2	-
Unknown	32	3.6	-	132	12.7	-	7	0.7	-	5	0.4	-	0	-	-
SPA															
1	24	2.7	6.4	38	3.7	9.8	40	4.0	10.2	29	2.5	7.4	35	3.1	8.8
2	215	23.9	9.7	228	21.9	10.6	262	25.9	12.1	238	20.9	10.9	264	23.1	11.8
3	161	17.9	9.3	164	15.8	10.1	155	15.3	9.5	235	20.6	14.3	196	17.1	11.8
4	80	8.9	6.4	162	15.6	14.4	106	10.5	9.3	130	11.4	11.3	131	11.5	11.2
5	70	7.8	10.6	71	6.8	11.1	74	7.3	11.4	62	5.4	9.5	114	10.0	17.3
6	107	11.9	10.0	109	10.5	10.7	109	10.8	10.6	142	12.4	13.7	127	11.1	12.1
7	122	13.6	8.9	145	13.9	11.2	155	15.3	11.8	176	15.4	13.4	162	14.2	12.2
8	117	13.0	10.4	123	11.8	11.5	109	10.8	10.1	129	11.3	11.9	115	10.1	10.5
Unknown	4	0.4	-	1	0.1	-	0	-	-	0	-	-	0	-	-



Figure 1. Reported Salmonellosis Rates by Year LAC, CA, and US, 2005-2015



Figure 3. Reported Salmonellosis by Race/Ethnicity LAC, 2015 (N=1144)



*Other includes Native American and any additional racial/ethnic group that cannot be categorized as Asian, Black, Hispanic, or White.

Figure 4. Reported Salmonellosis Rates by SPA LAC, 2015 (N=1144)







Figure 5. Reported Salmonellosis Cases by Month of Onset LAC, 2015 (N=1144)



Map 10. Salmonellosis Rates by Health District, Los Angeles County, 2015*





SHIGA-TOXIN PRODUCING ESCHERICHIA COLI (STEC)

CRUDE DATA	STEC						
Number of Cases	175						
Annual Incidence ^a							
LA County	1.83						
California ^{b, c}	2.37						
United States ^{b, c}	2.20						
Age at Diagnosis							
Mean	27						
Median	21						
Range	0–89 years						

^aCases per 100,000 population

^bCalculated from: CDC. *Notice to Readers:* Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly*/November 25, 2016/65(46);1306–1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm ^cIncudes E.coli O157:H7; shiga toxin-positive, serogroup non-O157: and Shiga toxin-positive, not serogrouped. All cases are now reported as STEC (Shiga toxin producing *E.coli*) in order to simplify the reporting process

DESCRIPTION

Escherichia coli is a gram-negative bacillus with numerous serotypes. Several of these produce Shiga toxin and are called STEC. Gastrointestinal infection with a Shiga toxin-producing serotype causes abdominal cramps and watery diarrhea, often developing into bloody diarrhea; fever is uncommon. The incubation period is 2-8 days. These organisms naturally occur in the gut of many animals. Likely modes of transmission to humans from animals include foodborne (e.g., undercooked ground beef, raw milk, fresh produce, and contaminated unpasteurized juice), direct exposure to animals and their environments, and exposure to recreational water contaminated with animal or human feces. Person-to-person transmission such as between siblings or within a daycare center is also well documented.

The most common STEC serotype in the US is *E. coli* O157:H7, but several other serotypes occur and cause illness. A positive test for Shiga toxin in stool as well as cultures of STEC are reportable to LAC DPH. All reported positive STEC broths or isolates are confirmed and serotyped by the LAC Public Health Laboratory.

Hemolytic uremic syndrome (HUS) is a disorder consisting of hemolytic anemia, kidney failure, and thrombocytopenia. It is diagnosed clinically and is most frequently associated with recent infection from *E. coli* O157:H7 but may also be caused by other serotypes. Children younger than five years old are at highest risk for HUS. Adults may develop a related condition called thrombotic thrombocytopenic purpura (TTP) after STEC infection.

Increased public education to prevent STEC infection is important. Information should focus on safe food handling practices, proper hygiene, and identifying high-risk foods and activities at home and while eating out. To avoid infection, beef products should be cooked thoroughly. Produce, including pre-washed products, should be thoroughly rinsed prior to eating. In addition, one should drink only treated water and avoid swallowing recreational water. Careful handwashing is essential, especially before eating and after handling raw beef products or coming in contact with or being around animals. Strengthening of national food processing regulations is also important to reduce contamination.

- In 2015, the increased use of new technology to perform bacterial testing was implemented. Polymerase chain reaction (PCR) and real-time PCR were used rather than the traditional culture method. This likely contributed to the increase in cases.
- There were 175 cases reported, and 53% (n=92) of these cases were confirmed by PCR testing.
- The highest incidence rate by age was observed in the 1-4 years old age group (9.1 per 100,000), which has consistently had the highest incidence rate (Figure 2).
- In 2015, White cases had the highest incidence rate of all race/ethnicity groups (2.8 per 100,000) followed by Hispanics (1.5 per 100,000) (Figure 6).
- SPA 5 had the highest rate (4.7 per 100,000) followed by SPA 4 (2.2 per 100,000) (Figure 4).
- Two cases were reported with HUS and were laboratory confirmed with STEC serotype, one



was O157:H7, and the other was STEC (non O157:H7). No deaths occurred.

• There was one LAC outbreak of O157:H7 in 2015 involving a petting zoo investigated by

ACDC. ACDC participated in three multistate cluster investigations.



Reported Shiga-toxin Producing *Escherichia coli* (STEC) Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA, LAC, 2011-2015

	2011 (N=88)			2012 (N=97)			2013 (N=102)			2014 (N=90)			2015 (N=175)		
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	8	9.1	6.7	6	6.2	5.0	5	4.9	4.1	1	1.1	0.8	5	2.9	4.6
1-4	36	40.9	7.5	42	43.3	8.8	43	42.2	8.8	42	46.7	8.6	44	25.1	9.1
5-14	14	15.9	1.2	15	15.5	1.3	17	16.7	1.4	17	18.9	1.4	24	13.7	2.0
15-34	15	17.0	0.5	16	16.5	0.6	24	23.5	0.8	10	11.1	0.4	42	24.0	1.5
35-44	4	4.5	0.3	4	4.1	0.3	4	3.9	0.3	4	4.4	0.3	14	8.0	1.1
45-54	0	0.0	0.0	5	5.2	0.4	3	2.9	0.2	8	8.9	0.6	14	8.0	1.1
55-64	5	5.7	0.5	6	6.2	0.6	1	1.0	0.1	4	4.4	0.4	15	8.6	1.4
65+	6	6.8	0.6	3	3.1	0.3	5	4.9	0.5	4	4.4	0.4	17	9.7	1.4
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	6	6.8	0.5	6	6.2	0.5	2	2.0	0.1	5	5.6	0.4	13	7.4	0.9
Black	3	3.4	0.4	4	4.1	0.5	5	4.9	0.6	3	3.3	0.4	11	6.3	1.4
Hispanic	50	56.8	1.1	50	51.5	1.1	57	55.9	1.2	54	60.0	1.2	72	41.1	1.5
White	28	31.8	1.1	34	35.1	1.3	36	35.3	1.4	25	27.8	0.9	74	42.3	2.8
Other	0	-	-	0	-	-	0	-	-	0	-	-	2	1.1	-
Unknown	1	1.1	-	3	3.1	-	2	2.0	-	3	3.3	-	3	1.7	-
SPA															
1	3	3.4	0.8	1	1.0	0.3	5	4.9	1.3	2	2.2	0.5	4	2.3	1.0
2	18	20.5	0.8	27	27.8	1.3	29	28.4	1.3	23	25.6	1.1	42	24.0	1.9
3	11	12.5	0.7	12	12.4	0.7	12	11.8	0.7	20	22.2	1.2	19	10.9	1.1
4	9	10.2	0.8	13	13.4	1.2	11	10.8	1.0	8	8.9	0.7	26	14.9	2.2
5	8	9.1	1.3	8	8.2	1.3	12	11.8	1.9	2	2.2	0.3	31	17.7	4.7
6	11	12.5	1.1	9	9.3	0.9	13	12.7	1.3	7	7.8	0.7	10	5.7	1.0
7	21	23.9	1.6	15	15.5	1.2	13	12.7	1.0	17	18.9	1.3	20	11.4	1.5
8	7	8.0	0.7	12	12.4	1.1	7	6.9	0.6	11	12.2	1.0	23	13.1	2.1
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-









Figure 3. Percent Cases of Shiga Toxin-Producing *E. coli* by Race/Ethnicity, LAC, 2015 (N=175)



65+





Figure 6. Reported Shiga Toxin-Producing Cases by

Race/Ethnicity, LAC, 2010-2015



Figure 5. Reported Shiga Toxin-Producing *E. coli* Cases by Serotype Month of Onset, LAC, 2015 (N=175)





Map 11. Shiga Toxin-Producing *E. Coli* Rates by Health District, Los Angeles County, 2015*



SHIGELLOSIS

CRUDE DATA							
Number of Cases	508						
Annual Incidence ^a							
LA County	5.31						
California ^b	5.68						
United States ^b	7.34						
Age at Diagnosis							
Mean	34						
Median	33						
Range	1–92 years						

^aCases per 100,000 population

^bCalculated from: CDC. *Notice to Readers:* Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306– 1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

Shigellosis is caused by a gram-negative bacillus with four main serogroups: Shigella dysenteriae (group A), S. flexneri (group B), S. boydii (group C), and S. sonnei (group D). The incubation period is 1-3 days. Humans are the definitive host. Fecal-oral transmission occurs when individuals fail to thoroughly wash their hands after defecation and then spread infective particles to others. This occurs either directly by physical contact including sexual behaviors or indirectly by contaminating food. Infection may occur with ingestion of as few as ten organisms. Common symptoms include diarrhea, fever, nausea, vomiting, and tenesmus. Stool may contain blood or mucous. In general, elderly. immunocompromised, and malnourished people are more susceptible to severe outcomes from infection.

Hand washing is vital in preventing this disease. Children or anyone with uncertain hygiene practices should be monitored to promote compliance. Hand washing is especially important when in crowded areas. Children with diarrhea, especially those in 147 diapers, should not be allowed to swim or wade in public swimming areas. In LAC, cases and symptomatic contacts in sensitive situations or occupations (e.g., food handlers, daycare, and healthcare workers) are routinely removed from work or the situation until their stool specimen cultures are negative when tested by the LAC Public Health Laboratory (PHL).

- The incidence of shigellosis cases in LAC increased from 3.7 cases per 100,000 in 2014 to 5.3 cases per 100,000 in 2015 (Figure 1).
- The highest incidence rate by age was observed in 1-4 year olds (7.8 per 100,000) followed by 15-34 year olds and 35-44 year olds at 6.3 per 100, 000 for both (Figure 2). The 1-4 year olds have consistently had the highest incidence rate.
- In 2015, White cases had the highest incidence rate of all race/ethnicity groups (8.0 per 100,000) (Figure 6) followed by Blacks (7.6 per 100,000). In prior years, rates were similar among Whites and Hispanics with decreased rates among Asians.
- SPA 4 sustained the highest rate (14.0 per 100,000) followed by SPA 5 (11.8 per 100,000) (Figure 4). The incidence of shigellosis cases in SPA 5 increased to 11.8 cases per 100,000 in 2015 from 2.1 cases per 100,000 in 2011. The increase in SPA 5 can be attributed to a large community outbreak.
- In 2015, the percentage of shigellosis cases hospitalized for at least two days was consistent with previous years (n=113; 22%). The number of cases among men who have sex with men (MSM) in 2015 decreased to 18% (n=90) from 24% (n=84) in 2014. There was one death reported with comorbidities.
- There were two shigellosis-associated outbreaks investigated in 2015 by the LAC DPH Community Health Services.


Reported Shigellosis Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=264)		201	12 (N=30	06)	20 ⁻	13 (N=2	27)	20	14 (N=3	50)	201	5 (N=50	08)	
	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000	No.	(%)	/Rate 100,000	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	4	1.5	2.9	4	1.3	3.4	1	0.4	0.8	2	0.6	1.7	0	-	-
1-4	30	11.4	5.2	32	10.5	6.7	26	11.5	5.3	30	8.6	6.1	38	7.5	7.8
5-14	37	14.0	2.8	54	17.6	4.5	49	21.6	4.1	51	14.6	4.2	52	10.2	4.3
15-34	80	30.3	2.7	68	22.2	2.5	55	24.2	1.9	85	24.3	3.0	178	35.0	6.3
35-44	41	15.5	2.8	39	12.7	2.9	31	13.7	2.3	64	18.3	4.8	84	16.5	6.3
45-54	44	16.7	3.3	31	10.1	2.4	30	13.2	2.3	57	16.3	4.4	80	15.7	6.1
55-64	15	5.7	1.6	25	8.2	2.5	19	8.4	1.9	30	8.6	2.8	36	7.1	3.3
65+	12	4.5	1.1	52	17.0	4.7	15	6.6	1.4	31	8.9	2.7	40	7.9	3.4
Unknown	1	0.4	-	1	0.3	-	1	0.4	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	4	1.5	0.3	2	0.7	0.2	5	2.2	0.4	17	4.9	1.2	17	3.3	1.2
Black	24	9.1	2.8	29	9.5	3.7	25	11.0	3.2	19	5.4	2.4	60	11.8	7.6
Hispanic	149	56.4	3.1	153	50.0	3.4	107	47.1	2.3	167	47.7	3.6	213	41.9	4.5
White	78	29.5	2.7	104	34.0	3.9	82	36.1	3.1	132	37.7	5.0	215	42.3	8.0
Other	0	-	-	0	-	-	2	0.9	-	1	0.3	-	3	0.6	-
Unknown	9	3.4	-	18	5.9	-	6	2.6	-	14	4.0	-	0	-	-
SPA															
1	7	2.7	1.9	3	1.0	0.8	4	1.8	1.0	5	1.4	1.3	4	0.8	1.0
2	40	15.2	1.8	52	17.0	2.4	39	17.2	1.8	59	16.9	2.7	74	14.6	3.3
3	32	12.1	1.8	26	8.5	1.6	16	7.0	1.0	29	8.3	1.8	33	6.5	2.0
4	82	31.1	6.5	85	27.8	7.6	58	25.6	5.1	108	30.9	9.4	164	32.3	14.0
5	14	5.3	2.1	48	15.7	7.5	18	7.9	2.8	25	7.1	3.8	78	15.4	11.8
6	38	14.4	3.6	37	12.1	3.6	44	19.4	4.3	40	11.4	3.9	56	11.0	5.3
7	24	9.1	1.7	33	10.8	2.5	33	14.5	2.5	43	12.3	3.3	55	10.8	4.2
8	26	9.8	2.3	22	7.2	2.1	15	6.6	1.4	41	11.7	3.8	43	8.5	3.9
Unknown	1	0.4	-	0	-	-	0	-	-	0	-	-	1	0.2	-



12

10

8

6

4 2

0

Cases per 100,000





Figure 4. Reported Shigellosis Rates by SPA LAC, 2015 (N=508)







Figure 6. Shigellosis Incidence by Race/Ethnicity LAC, 2011-2015

Figure 5. Reported Shigellosis Cases by Month of Onset LAC, 2015 (N=508)

AV SF Miles EV FH WV *PS HW AH CE PO WE EM SWSE NH SO SA IW ÇN Cases Per 100,000 Population BF 7.4 - 20.2 **Health District Boundary** Service Planning Area (SPA) 4.6 - 7.3 3.0 - 4.5 1.8 - 2.9 0.0 - 1.7

Catalina Island (HB)

Map 12. Shigellosis Rates by Health District, Los Angeles County, 2015*

*Excludes Long Beach and Pasadena Data.

Shigellosis Page 131





SEVERE STAPHYLOCOCCUS AUREUS INFECTION IN PREVIOUSLY HEALTHY PERSONS

CRUDE	DATA					
Number of Cases	9					
Annual Incidence						
LA County ^a	0.09					
California⁵	0.20					
United States°	N/A					
Age at Diagnosis						
Mean	57					
Median	58					
Range	34–78 years					

^aCases per 100,000 population

^bSee Yearly Summary Reports of Selected General Communicable Diseases in California at:

https://www.cdph.ca.gov/data/statistics/Documents/YearlySu mmaryReportsofSelectedGeneralCommDiseasesinCA2011-2015.pdf

°Not notifiable

DESCRIPTION

Staphylococcus aureus (S. aureus) is bacteria that can cause a number of diseases as a result of infection of various tissues of the body. S. aureus-related illness can range from mild and requiring no treatment to severe and potentially fatal. It is a common cause of skin infections such as boils, abscesses, and cellulitis. It can also cause invasive skin and soft-tissue infection, necrotizing fasciitis, musculoskeletal infection, and osteomyelitis. Infection can result in severe illness including bacteremia, sepsis, pneumonia, empyema, and necrotizing pneumonia.

Certain groups of people are at greater risk including people with chronic conditions such as diabetes, cancer, vascular disease, and lung disease. Those who are intravenous drug users, with skin injuries or disorders, with intravenous catheters, with surgical incisions, and with weakened immune systems due to disease or to immune-suppressing medications have an increased risk of developing *S. aureus* infections. In February 2008, in response to the significant public health burden and potential severity of community-associated *S. aureus* infections, the CDPH added severe cases of *S. aureus* infections including methicillin-resistant *S. aureus* (MRSA) to the state list of reportable diseases and conditions. This is not a nationally notifiable disease.

For surveillance purposes, a case of communityassociated severe *S. aureus* infection is defined as a laboratory-confirmed *S. aureus* infection in a person resulting in admission to an intensive care unit (ICU). Additionally, this definition includes laboratory-confirmed infections in deaths who had not been hospitalized or had surgery, dialysis, or residency in a long-term care facility in the year prior to illness. Lastly, this definition includes laboratory-confirmed infections in those who did not have an indwelling catheter or percutaneous medical device at the onset of illness. If any of these conditions were present, the case would be considered healthcareassociated.

S. aureus is one of the most common bacterial causes of skin infections that result in a visit to a doctor or hospital. However, most of these infections do not result in ICU admission or death. Therefore, the data presented in this report underestimate all disease caused by this organism in LAC.

2015 TRENDS AND HIGHLIGHTS

- Cases <u>>65</u> years old had the highest rate (0.3 per 100,000) in 2015.
- Blacks, Hispanics, and Whites had the same rate (0.1 per 100,000) (Figure 2) while 22.2% of cases had no designated race/ethnicity.
- The male to female ratio in 2015 was 2:1.
- The incidence rate was highest in SPA 4 (0.2 per 100,000) (Figure 3).
- Cases were distributed throughout the year, peak months being January, February, and August (Figure 4).
- Nearly one-fourth (n=2, 22.2%) of the reported cases were MRSA infections (Figure 5).
- The most frequently reported risk factors were alcohol abuse, diabetes, obesity, and intravenous drug use (Table 1).



• Pneumonia, septic shock, septic emboli, and endocarditis were among the common presentations for *S. aureus* infections (Table 2).



Reported Severe Staphylococcus Aureus Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=44)		4)	20	12 (N=2	4)	20)13 (N=2	26)	20	14 (N=1	7)	20	015 (N=	9)
	No.	(%)	Rate/ 100,000	No.	(%)	/Rate 100,000	No.	(%)	/Rate 100,000	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0	0.0	1	4.2	0.8	1	3.8	0.8	0	0.0	0.0	0	0.0	0.0
1-4	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
5-14	2	4.5	0.2	1	4.2	0.1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
15-34	6	13.6	0.2	3	12.5	0.1	7	26.9	0.2	3	17.6	0.1	1	11.1	0.0
35-44	6	13.6	0.4	2	8.3	0.2	2	7.7	0.2	3	17.6	0.2	1	11.1	0.1
45-54	9	20.5	0.7	3	12.5	0.2	6	23.1	0.5	3	17.6	0.2	1	11.1	0.1
55-64	8	18.2	0.8	5	20.8	0.5	5	19.2	0.5	3	17.6	0.3	2	22.2	0.2
65+	13	29.5	1.2	9	37.5	0.8	5	19.2	0.5	5	29.4	0.4	4	44.4	0.3
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	7	15.9	0.5	4	16.7	0.3	3	11.5	0.2	4	23.5	0.3	0	0.0	0.0
Black	3	6.8	0.4	4	16.7	0.5	5	19.2	0.6	2	11.8	0.3	1	11.1	0.1
Hispanic	17	38.6	0.4	4	16.7	0.1	10	38.5	0.2	3	17.6	0.1	3	33.3	0.1
White	15	34.1	0.5	10	41.7	0.4	8	30.8	0.3	0	0.0	0.0	3	33.3	0.1
Other	1	2.3	-	1	4.2	-	0	-	-	1	5.9	-	0	-	-
Unknown	1	2.3	-	1	4.2	-	0	-	-	7	41.2	-	2	22.2	-
SPA															
1	0	0.0	0.0	2	8.3	0.5	1	3.8	0.3	0	0.0	0.0	0	0.0	0.0
2	12	27.3	0.5	1	4.2	0.0	6	23.1	0.3	2	11.8	0.1	1	11.1	0.0
3	7	15.9	0.4	8	33.3	0.5	1	3.8	0.1	6	35.3	0.4	2	22.2	0.1
4	2	4.5	0.2	2	8.3	0.2	4	15.4	0.4	5	29.4	0.4	2	22.2	0.2
5	5	11.4	0.8	1	4.2	0.2	2	7.7	0.3	1	5.9	0.2	0	0.0	0.0
6	11	25.0	1.0	5	20.8	0.5	5	19.2	0.5	1	5.9	0.1	1	11.1	0.1
7	5	11.4	0.4	4	16.7	0.3	3	11.5	0.2	0	0.0	0.0	0	0.0	0.0
8	1	2.3	0.1	0	0.0	0.0	2	7.7	0.2	2	11.8	0.2	1	11.1	0.1
Unknown	1	2.3	-	1	4.2	-	2	7.7	-	0	-	-	2	22.2	-







Figure 2. Severe *S. aureus* Infection Incidence Rates* by Race/Ethnicity, LAC, 2014-2015



Figure 3. Incidence Rates* of Severe *S. aureus* Infection by SPA LAC, 2014-2015







Month

rol ort

Figure 5. Percent Cases of Severe S. aureus Infection by Methicillin-Resistance Type, LAC, 2015 (N=9)



*MRSA=Methicillin Resistance *Staphylococcus aureus* **MSSA=Methicillin Sensitive *Staphylococcus aureus*

	201 N = 1	4 17	201 N =	5 9
	N	%*	Ν	%*
Alcohol Abuse	1	6	3	33
Diabetes	6	35	2	22
Obesity	0	0	2	22
IVDU	0	0	2	22
Chronic Dermatitis	1	6	1	11
Current Smoker	3	18	1	11
Emphysema	3	18	1	11
HIV/AIDS	2	12	1	11
None	2	12	1	11
Unknown	0	0	1	11
Chronic Renal	0	0	1	11
Malignancy-Hem	0	0	1	11
Other	5	29	0	0
Intravenous Drug Use	3	18	0	0
Malignancy-Solid	2	12	0	0
Liver Disease	0	0	0	0

Table 1. Severe S. aureus Medical Conditions by Date of

Table 2. Frequency and Percentage of Severe *S. aureus* Clinical Syndromes, LAC, 2015 (N=9)

Syndrome	<u>Number</u>	Percent*
Pneumonia	2	22.2
Bacteremia (without focus)	1	11.1
Septic Shock	2	22.2
Endocarditis	2	22.2
Septic emboli	2	22.2
Other	1	11.1
Meningitis	1	11.1

*Overlapping syndromes will total over 100%.

*Overlapping risk factors will total over 100%.

Onset, 2014-2015 2014 2015





INVASIVE GROUP A STREPTOCOCCUS (IGAS)

CRUE	CRUDE DATA										
Number of Cases	227										
Annual Incidenceª											
LA County	2.37										
California ^b	N/A										
United States ^{b, c}	N/A										
Age at Diagnosis											
Mean	50										
Median	53										
Range	0–100 years										

^aCases per 100,000 population

^b Not notifiable

[°] National projection of IGAS incidence from Active Bacterial Core Surveillance Areas data, 2015. Not available as of January 2017.

DESCRIPTION

Invasive group A streptococcal disease (IGAS) is caused by the group A beta-hemolytic Streptococcus pyogenes bacterium. Transmission is by direct contact or occasionally by indirect contact with infectious material. Illness manifests as various clinical syndromes including bacteremia without focus, sepsis, cutaneous wound or deep soft-tissue infection, septic arthritis, and pneumonia. IGAS is the most frequent cause of necrotizing fasciitis and is commonly known as "flesh eating bacteria." IGAS occurs in all age groups but more frequently occurs among elderly people. Infection can result in severe illness or even death.

For surveillance purposes in LAC, a case of IGAS is defined as isolation of *S. pyogenes* from a normally sterile body site (e.g., blood, cerebrospinal fluid, synovial fluid, or from tissue collected during surgical procedures) or from a non-sterile site if associated with streptococcal toxic shock syndrome (STSS) or necrotizing fasciitis (NF). IGAS cases are characterized as

STSS if the diagnosis fulfills the CDC or Council of State and Territorial Epidemiologists case definition for this syndrome or as NF if the diagnosis was made by the treating physician.

S. pyogenes more commonly causes noninvasive disease that presents as strep throat and skin infections. However, these diseases are not counted in LAC surveillance of invasive disease; therefore, the data presented in this report underestimates all disease caused by *S. pyogenes* in LAC.

The spread of IGAS can be prevented by good hand washing. The CDC provides guidelines for hand washing

(www.cdc.gov/mmwr/preview/mmwrhtml/rr5605 a4.htm). Wounds should be kept clean and monitored for signs of infection such as redness, swelling, pus, and pain. A person should seek medical care if any signs of wound infection are present, especially if accompanied by fever. High risk groups such as diabetics are encouraged to seek medical care sooner if experiencing fever, chills, and any redness on the skin.

2015 TRENDS AND HIGHLIGHTS

- The incidence rate of reported IGAS was 2.4 cases per 100,000 during 2015, which is the highest in the last 10 seasons (Figure 1).
- Whites had the highest rate of IGAS this year (1.9 per 100,000), which is consistent with 2014. In 2015, rates for Blacks slightly increased to 1.8 from 1.3. A little over half of cases (n=124, 54.6%) had no race/ethnicity designation, which is consistent with 2014.
- SPA 4 and 6 had the highest incidence rate at 2.9 and 2.8 cases per 100,000, respectively (Figure 4). Other SPAs remained relatively consistent from 2011-2015.
- In 2015, the number of reported cases peaked in January with 27 cases followed by 26 cases in April. August and September had the lowest number of reported cases with 11 and 10 cases, respectively (Figure 5). The number of reported cases throughout the year was higher overall than the previous five-year average and higher than any other individual year since 2005 (Figure 1).



- IGAS cases presented most often with bacteremia (without focus) and cellulitis (Table 1).
- Consistent with the past several years, diabetes was reported more than any other risk factor (30%) followed by history of blunt trauma (11%). Although one-third of cases (33%) reported having none of the traditional risk factors (Table 2).
- The most common risk factor in the category of other was obesity (2%).
- Although the number of cases in 2015 is highest over the last five-year period (2011-2015), this increase may be attributable to an increase in reporting due to the development of more efficient electronic reporting systems.



Reported Invasive Group A Streptococcus Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	20	11 (N=	175)	20	12 (N=	168)	20	13 (N=	195)	20	14 (N=	222)	20	15 (N=	227)
	No.	(%)	Rate/ 100,000												
Age Group															
<1	1	0.6	0.7	3	1.8	2.5	5	2.6	4.1	7	3.2	5.9	1	0.4	0.9
1-4	6	3.4	1.0	5	3.0	1.1	4	2.1	0.8	7	3.2	1.4	7	3.1	1.4
5-14	10	5.7	0.8	7	4.2	0.6	10	5.1	0.8	16	7.2	1.3	16	7.0	1.3
15-34	16	9.1	0.5	27	16.1	1.0	29	14.9	1.0	34	15.3	1.2	29	12.8	1.0
35-44	28	16.0	1.9	20	11.9	1.5	20	10.3	1.5	24	10.8	1.8	25	11.0	1.9
45-54	32	18.3	2.4	31	18.5	2.4	41	21.0	3.2	43	19.4	3.3	43	18.9	3.3
55-64	36	20.6	3.7	35	20.8	3.4	31	15.9	3.0	35	15.8	3.3	37	16.3	3.3
65+	46	26.3	4.3	39	23.2	3.5	54	27.7	4.9	56	25.2	4.9	68	30.0	5.7
Unknown	0	-	-	1	0.6	-	1	0.5	-	0	-	-	1	0.4	-
Race/Ethnicity															
Asian	13	7.4	1.0	8	4.8	0.6	8	4.1	0.6	6	2.7	0.4	5	2.2	0.4
Black	22	12.6	2.6	24	14.3	3.1	29	14.9	3.7	10	4.5	1.3	14	6.2	1.8
Hispanic	49	28.0	1.0	58	34.5	1.3	29	14.9	0.6	29	13.1	0.6	29	12.8	0.6
White	45	25.7	1.6	44	26.2	1.7	50	25.6	1.9	51	23.0	1.9	52	22.9	1.9
Other	0	-	-	2	1.2	-	5	2.6	-	11	5.0	-	3	1.3	-
Unknown	46	26.3	-	32	19.0	-	74	37.9	-	115	51.8	-	124	54.6	-
SPA															
1	3	1.7	0.8	0	0.0	0.0	4	2.1	1.0	5	2.3	1.3	4	1.8	1.0
2	34	19.4	1.5	32	19.0	1.5	38	19.5	1.7	38	17.1	1.7	54	23.8	2.4
3	22	12.6	1.3	17	10.1	1.1	23	11.8	1.4	49	22.1	3.0	31	13.7	1.9
4	31	17.7	2.5	38	22.6	3.4	33	16.9	2.9	44	19.8	3.8	34	15.0	2.9
5	14	8.0	2.1	10	6.0	1.6	18	9.2	2.8	11	5.0	1.7	15	6.6	2.3
6	22	12.6	2.1	24	14.3	2.4	23	11.8	2.2	25	11.3	2.4	29	12.8	2.8
7	20	11.4	1.5	17	10.1	1.3	16	8.2	1.2	21	9.5	1.6	21	9.3	1.6
8	28	16.0	2.5	21	12.5	2.0	24	12.3	2.2	24	10.8	2.2	26	11.5	2.4
Unknown	1	0.6	-	9	5.4	-	16	8.2	-	5	2.3	-	13	5.7	-





Figure 1. Incidence Rates of Invasive Group A

* Active Bacterial Core Surveillance Reports from 2000 to 2015 from the Centers for Disease Control and Prevention's Division of Bacterial Diseases. Report available at: www.cdc.gov/abcs/reportsfindings/surv-reports.html

Figure 3. Invasive Group A Streptococcus Incidence Rates* by



*Rates based on fewer than 19 cases are unreliable





Figure 4. Incidence Rates* of Invasive Group A Streptococcus by SPA LAC, 2015 (N=227)



*Rates based on fewer than 19 cases are unreliable





Table 1. Frequency and Percentage of 2015 (N=1	IGAS Clinical Syn 57)	dromes, LAC,
<u>Syndrome</u>	Number	Percent*
Cellulitis	56	35.7
Bacteremia (without focus)	43	27.4
STSS	32	20.4
Other	18	11.5
Pneumonia	11	7.0
Necrotizing Fasciitis	11	7.0
Non-surgical wound infection	5	3.2
Osteomyelitis	4	2.5
*Overlapping syndromes will total over **Cases with unknown symptoms excl	[.] 100%. uded.	

Table 2. Percentage of IGAS Risk Factors Based on Date of Onset Between 1/1/2013-12/31/2015									
	2013	2014	2015						
Risk Factors*	(N =195)	(N =182)	(N=141)						

Risk Factors*	(N =195)	(N =182)	(N=141)							
	%**	%**	%**							
Alcohol Abuse	13	8	7							
Chronic Heart Disease	14	15	9							
Chronic Lung Disease	6	6	6							
Cirrhosis	5	7	0							
Diabetes	28	30	30							
History of Blunt Trauma	17	4	11							
HIV/AIDS	2	2	4							
IV Drug Use	7	2	2							
Malignancy	13	9	7							
Other	15	7	0							
None	30	29	33							
*Overlapping risk factors will total over 100%.										



Map 13. Streptococcus, Group A Invasive Rates by Health District, Los Angeles County, 2015*



TYPHOID FEVER, ACUTE AND CARRIER

ACUTE TYPHOID CRUDE DATA											
Number of Cases	14										
Annual Incidence ^a											
LA County ^b	0.15										
California ^c	0.14										
United States ^c	0.11										
Age at Diagnosis											
Mean	21										
Median	25										
Range	2–79 years										

^aCases per 100,000 population

^bRates based on less than 19 observations are considered unreliable

^cCalculated from: CDC. *Notice to Readers*: Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306– 1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

Typhoid fever, or enteric fever, is an acute systemic disease caused by the gram-negative bacillus *Salmonella typhi*. Transmission may occur person to person or by ingestion of food or water contaminated by the urine or feces of acute cases or carriers. Common symptoms include persistent fever, headache, malaise, anorexia, constipation (more commonly than diarrhea), bradycardia, enlargement of the spleen, and rose spots on the trunk. Humans are the only known reservoir for *S. typhi*. Vaccines are available to those at high risk from close exposure to a typhoid carrier in the house or travel to developing foreign countries.

Among untreated acute cases, 10% will shed bacteria for three months after initial onset of symptoms and 2-5% will become chronic typhoid carriers. Some carriers are diagnosed by positive tissue specimen. Chronic carriers are by definition asymptomatic.

Hand washing after using the toilet, before preparing or serving food, and before and after direct or intimate contact with others is important in preventing the spread of typhoid. When traveling to locations where sanitary practices are uncertain, foods should be thoroughly cooked, and bottled water should be used for drinking, brushing teeth, and making ice. Vaccination should be considered when traveling to endemic areas. LAC DPH screens household contacts of confirmed cases for *S. typhi* to identify any previously undiagnosed carriers or cases. A modified order of isolation restricts a carrier from engaging in a sensitive occupation or situation. LAC DPH monitors compliance with such isolation order and offers the case a chance to clear the infection with antibiotics.

2015 TRENDS AND HIGHLIGHTS

- In 2015, all acute typhoid cases reported travel to countries with endemic typhoid fever.
- Asians (n=8; 57%) accounted for the largest proportion of acute cases followed by Hispanic cases (n=4, 29%) (Figure 3). Asians had the highest incidence rate of all the race/ethnicity groups (0.6 cases per 100,000).
- SPA 2 and 4 both had the highest incidence rates for acute typhoid fever (0.3 cases per 100,000). SPA 2 reported the largest proportion of cases (n=7, 50%) followed by SPA 4 (n=4, 29%).
- During 2015, cases were observed throughout the year; however, more cases are typically observed during the summer months. Cases peaked above the five-year average in January, July, and November (Figure 5).
- LAC DPH monitors existing carriers who are listed on the state typhoid registry until they are cleared of infection (Figure 6). No new carriers were reported since 2012.
- No paratyphoid cases were reported in 2015.



Reported Acute Typhoid Fever Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=15)		20	012 (N=	6)	20	13 (N=1	7)	20	014 (N=1	5)	20	15 (N=1	4)	
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	1	6.7	0.7	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
1-4	0	0.0	0.0	0	0.0	0.0	3	17.6	0.6	0	0.0	0.0	3	21.4	0.6
5-14	1	6.7	0.1	1	16.7	0.1	3	17.6	0.2	2	13.3	0.2	2	14.3	0.2
15-34	6	40.0	0.2	3	50.0	0.1	7	41.2	0.2	7	46.7	0.2	7	50.0	0.2
35-44	2	13.3	0.1	1	16.7	0.1	1	5.9	0.1	2	13.3	0.2	0	0.0	0.0
45-54	3	20.0	0.2	1	16.7	0.1	2	11.8	0.2	2	13.3	0.2	0	0.0	0.0
55-64	1	6.7	0.1	0	0.0	0.0	1	5.9	0.1	1	6.7	0.1	1	7.1	0.1
65+	1	6.7	0.1	0	0.0	0.0	0	0.0	0.0	1	6.7	0.1	1	7.1	0.1
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	7	46.7	0.5	2	33.3	0.2	12	70.6	0.9	10	66.7	0.7	8	57.1	0.6
Black	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
Hispanic	8	53.3	0.2	4	66.7	0.1	5	29.4	0.1	5	33.3	0.1	4	28.6	0.1
White	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	2	14.3	0.1
Other	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
SPA															
1	1	6.7	0.3	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
2	4	26.7	0.2	1	16.7	0.0	2	11.8	0.1	1	6.7	0.0	7	50.0	0.3
3	0	0.0	0.0	1	16.7	0.1	6	35.3	0.4	5	33.3	0.3	2	14.3	0.1
4	4	26.7	0.3	2	33.3	0.2	3	17.6	0.3	4	26.7	0.3	4	28.6	0.3
5	3	20.0	0.5	0	0.0	0.0	2	11.8	0.3	0	0.0	0.0	1	7.1	0.2
6	1	6.7	0.1	0	0.0	0.0	1	5.9	0.1	2	13.3	0.2	0	0.0	0.0
7	1	6.7	0.1	1	16.7	0.1	0	0.0	0.0	1	6.7	0.1	0	0.0	0.0
8	1	6.7	0.1	1	16.7	0.1	3	17.6	0.3	2	13.3	0.2	0	0.0	0.0
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-

Reported Typhoid Fever Carrier Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=3)			2012 (N=0)			2013 (N=0)			20	014 (N=0))	2015 (N=0)		
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
1-4	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
5-14	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
15-34	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
35-44	1	33.3	0.1	0	-	-	0	-	-	0	-	-	0	-	-
45-54	1	33.3	0.1	0	-	-	0	-	-	0	-	-	0	-	-
55-64	1	33.3	0.1	0	-	-	0	-	-	0	-	-	0	-	-
65+	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Black	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Hispanic	3	100.0	0.1	0	-	-	0	-	-	0	-	-	0	-	-
White	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Other	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
SPA															
1	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
2	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
3	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
4	1	33.3	0.1	0	-	-	0	-	-	0	-	-	0	-	-
5	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
6	1	33.3	0.1	0	-	-	0	-	-	0	-	-	0	-	-
7	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
8	1	33.3	0.1	0	-	-	0	-	-	0	-	-	0	-	-
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-



0.25

0.2

0.15

0.1

0.05

0

Cases per 100,000

----• LAC ----

Figure 1. Reported Acute Typhoid Fever Rates by Year LAC and US, 2005-2015

— US



Figure 2. Acute Typhoid Fever Cases by Age Group LAC, 2015 (N=14)

Figure 3. Reported Acute Typhoid Fever Cases by Race/Ethnicity LAC, 2015 (N=14)

Year



Figure 4. Reported Acute Typhoid Fever Cases by SPA LAC, 2015 (N=14)









TYPHUS FEVER

CRUDE DATA											
Number of Cases	54										
Annual Incidence ^a											
LA County	0.56										
California⁵	0.20										
United States ^c	N/A										
Age at Diagnosis											
Mean	45										
Median	47										
Range	4–82 years										

^aCases per 100,000 population

^bSee Yearly Summary Reports of Selected General Communicable Diseases in California at: https://www.cdph.ca.gov/data/statistics/Documents/YearlySu mmaryReportsofSelectedGeneralCommDiseasesinCA2011-2015.pdf

°Not notifiable

DESCRIPTION

Fleaborne typhus (murine typhus and endemic typhus) is caused by the bacteria Rickettsia typhi and Rickettsia felis and is transmitted through contact with feces that is discharged when an infected flea bites. Reservoir animals are predominantly feral cats, opossums, and rats. In LAC, most reported cases of typhus have historically occurred in residents of the foothills of central LAC. However, since 2006, the distribution of typhus has expanded to other regions of LAC. Symptoms include fever, severe headache, chills, and myalgia. A fine, macular rash may appear three to five days after onset. Occasionally, complications such as pneumonia or hepatitis may occur. Fatalities are uncommon, occurring in less than 1% of cases, but increase with age. The disease is typically mild in young children. Typhus is not vaccine preventable but can be treated with antibiotics.

Because fleaborne typhus is not a nationally reportable disease, there is no national case definition. In California, a standard case definition was developed beginning in 2012 because of expansion of this disease into new regions including Long Beach and Orange County. Cases included in LAC surveillance have, at minimum, a single high IgM or IgG titer positive for *Rickettsia typhi* along with the appropriate symptoms.

Typhus infection can be prevented through flea control measures implemented on pets. Foliage in the yard should be trimmed so that it does not harbor small mammals. Screens can be placed on windows and crawl spaces to prevent entry of animals and their fleas into the house.

2015 TRENDS AND HIGHLIGHTS

- LAC continues to document higher numbers of typhus compared to the previous decade with 54 cases in 2015. The case count began rising in 2010 with 31 cases and peaked in 2013 with 68 cases (Figure 1). Most reported cases were hospitalized (n=46, 85%), indicating that mild cases may not be diagnosed and reported. Our surveillance then likely underestimates the true number of cases.
- In 2015, the mean age of cases was 45 years old. Infections in children five years old and younger were rare.
- The highest number of typhus cases occurred in SPA 3 (n=22, 41%), which historically has had high case counts (Figure 3). With the exception of SPA 1, typhus cases continue to exist in all SPAs, indicating that typhus has established itself in new areas.
- This year, the peak number of cases occurred earlier than the typical seasonal curve with the highest monthly case count in June (n=11, 20%) (Figure 4). However, cases were documented in all months of the year. Physicians and residents should be aware that there is year-round risk of typhus infection in LAC.
- Only 11 cases (20%) recalled having flea exposure. Three cases reported exposure to animals directly due to occupational activities including a geologist, a day laborer, and a construction worker.
- Over half of cases reported an exposure to cats at or around their home (n=31, 57%) and about one third (n=17, 31%) to feral cats, in particular (Table 1). Reported exposure to cats has increased in the last few years (Figure 5). Overall exposure to cats increased from 26% of cases in 2010 to 68% of cases in 2014. Feral cat exposure was extracted from interview notes beginning

2012 and occurred in 33% of cases in 2015, accounting for over half of all cat exposures.

- The increase in cases of typhus in LAC may be due to a number of factors including the natural relocation of host animals (possums and feral cats) to regions not previously enzootic for typhus, changes in weather that favor flea survival, increased testing and reporting due to better educated physicians, and increased reporting to LAC DPH by electronic laboratory reporting.
- In 2015, a cluster of fleaborne typhus cases occurred among residents of a mobile home community in the San Gabriel Valley. ACDC coordinated a multi-agency investigation including Environmental Health, San Gabriel Valley Mosquito and Vector Control District, and Veterinary Public Health as well as

private organizations to determine the extent of the outbreak, to identify risk factors, and to implement control measures. A total of five outbreak cases of fleaborne typhus with symptom onsets from April 9 to June 5 were identified. Observed risk factors included an overabundance of fleas that were associated with opossums and free-roaming feral cats. These animals were sustained by ample amounts of domestic pet food that was left outdoors by the community's residents. A variety of control measures were implemented including enacting flea control within the mobile home park, reducing the feral cat population, and encouraging flea control for domesticated dogs and cats (see Special Studies report for details of this investigation).



Reported Fleaborne Typhus Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=38)		2012 (N=50)			2013 (N=68)			2	2014 (N=44	·)	2015 (N=54)			
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
1-4	1	2.6	0.2	0	0.0	0.0	1	1.5	0.2	1	2.3	0.2	1	1.9	0.2
5-14	3	7.9	0.2	6	12.0	0.5	5	7.4	0.4	1	2.3	0.1	2	3.7	0.2
15-34	5	13.2	0.2	11	22.0	0.4	16	23.5	0.6	10	22.7	0.4	10	18.5	0.4
35-44	5	13.2	0.3	13	26.0	1.0	12	17.6	0.9	6	13.6	0.5	8	14.8	0.6
45-54	9	23.7	0.7	10	20.0	0.8	13	19.1	1.0	10	22.7	0.8	18	33.3	1.4
55-64	9	23.7	0.9	4	8.0	0.4	13	19.1	1.3	8	18.2	0.8	9	16.7	0.8
65+	6	15.8	0.6	6	12.0	0.5	8	11.8	0.7	8	18.2	0.7	6	11.1	0.5
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	1	2.6	0.1	0	0.0	0.0	3	4.4	0.2	3	6.8	0.2	3	5.6	0.2
Black	2	5.3	0.2	2	4.0	0.3	1	1.5	0.1	0	0.0	0.0	4	7.4	0.5
Hispanic	9	23.7	0.2	15	30.0	0.3	24	35.3	0.5	17	38.6	0.4	20	37.0	0.4
White	23	60.5	0.8	25	50.0	0.9	35	51.5	1.3	17	38.6	0.6	24	44.4	0.9
Other	0	-	-	3	6.0	-	1	1.5	-	1	2.3	-	1	1.9	-
Unknown	3	7.9	-	5	10.0	-	4	5.9	-	6	13.6	-	2	3.7	-
SPA															
1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
2	9	23.7	0.4	5	10.0	0.2	6	8.8	0.3	3	6.8	0.1	10	18.5	0.4
3	13	34.2	0.7	18	36.0	1.1	20	29.4	1.2	17	38.6	1.0	22	40.7	1.3
4	5	13.2	0.4	13	26.0	1.2	18	26.5	1.6	5	11.4	0.4	8	14.8	0.7
5	5	13.2	0.8	6	12.0	0.9	5	7.4	0.8	6	13.6	0.9	1	1.9	0.2
6	0	0.0	0.0	4	8.0	0.4	7	10.3	0.7	3	6.8	0.3	0	0.0	0.0
7	5	13.2	0.4	3	6.0	0.2	4	5.9	0.3	5	11.4	0.4	6	11.1	0.5
8	1	2.6	0.1	1	2.0	0.1	8	11.8	0.7	5	11.4	0.5	7	13.0	0.6
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-









Figure 4. Fleaborne Typhus Cases by Month of Onset LAC, 2015 (N=54)







*Hash marked bars denotes exposure to any type of cat including feral cats.

Table 1. Animal Exposure* of Fleaborne Typhus Cases, LAC, 2015 (N=54)												
	At or around At or arou											
	Home	Employment										
	n (%)	n (%)										
Cat	31 (57)	3 (6)										
Feral Cat	17 (31)	3 (6)										
Dog	30 (56)	1 (2)										
Opossum	19 (35)	1 (2)										
Rodent	8 (15)	4 (7)										

*Cases may report more than one exposure and in both the home and employment location.

AV SF Miles ΈV FH ŴŃ GL *PS_ NE HW CE AH PO ŴÉ EM SWSE E SA ISO ÍŴ CN Cases Per 100,000 Population BF 1.1 - 2.0 TO **Health District Boundary** Service Planning Area (SPA) 1.0 0.5 - 0.9 HΒ 0.1 - 0.4 0.0 Catalina Island (HB) *Excludes Long Beach and Pasadena Data. Typhus Fever Page 156

Map 14. Typhus Fever Rates by Health District, Los Angeles County, 2015*



VIBRIOSIS

CRUDE DATA											
Number of Cases	43										
Annual Incidence ^a											
LA County ^₅	0.45										
California ^c	0.61										
United States ^c	0.41										
Age at Diagnosis											
Mean	42										
Median	37										
Range	8–89 years										

^aCases per 100,000 population

^bRates calculated based on less than 19 cases or events are considered unreliable

^cCalculated from: CDC. *Notice to Readers*: Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly* / November 25, 2016 / 65(46);1306– 1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

Vibriosis is an infection caused by commashaped, gram-negative bacteria of the genus Vibrio. Vibriosis most commonly presents as acute diarrhea but may also occur as a wound infection or septicemia. Vibriosis is transmitted by ingesting food or water contaminated with Vibrio or by contact between open wounds and contaminated water. The most common species that cause vibriosis are V. parahæmolyticus, V. alginolyticus, V. vulnificus, and V. choleræ. Two serotypes of V. choleræ (O1 and O139) may cause cholera, an acute, life-threatening diarrheal illness. Infection may be mild or without symptoms, but sometimes it can be severe. Approximately 1 in 20 infected persons develop severe disease characterized by profuse watery diarrhea, vomiting, and leg cramps. In these

persons, rapid loss of bodily fluids can lead to dehydration and shock. Without treatment, death can occur within hours. This disease can spread rapidly in areas with inadequate treatment of sewage and drinking water. Vibriosis is commonly associated with consumption of raw or undercooked seafood, particularly shellfish. Many vibriosis patients have had recent history of travel to developing countries.

2015 TRENDS AND HIGHLIGHTS

- The number of vibriosis cases reported to LAC increased each year from 2010 to 2014 (13 to a high of 52 cases). The number of cases decreased in 2015 compared to 2014 (Figure 1).
- The majority of vibriosis cases were 15-34 year olds (Figure 2).
- SPA 2 had the most confirmed cases of vibriosis in 2015 (Figure 4). In all regions of LAC, consumption of raw oysters or other seafood were significant sources of vibriosis.
- Typically, vibriosis cases peak during June through August because *Vibrio* flourishes in rising water temperatures (Figure 5).
- *V. parahæmolyticus* was the most common etiologic agent isolated (n=32). Almost one-third (n=10) *V. parahæmolyticus* cases reported eating oysters prior to onset.
- A minority of cases (n=7) reported foreign travel. Foreign countries reported included Mexico and the Philippines.
- There were five confirmed cases of *V*. *alginolyticus*. The majority of these cases (n=3) had a history of recreational water exposure.
- There were three confirmed cases of *V. fluviali*. All of these cases had unknown exposure.
- A small number of cases (n=3) had a *Vibrio* species that was not identified.
- There were no vibriosis deaths in 2015.



Reported Vibriosis Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=19)		2012 (N=29)			2013 (N=26)			20	014 (N=	52)	2015 (N=43)			
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
1-4	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
5-14	1	5.3	0.1	3	10.3	0.3	3	11.5	0.2	2	3.8	0.2	1	2.3	0.1
15-34	5	26.3	0.2	7	24.1	0.3	4	15.4	0.1	18	34.6	0.6	18	41.9	0.6
35-44	3	15.8	0.2	4	13.8	0.3	7	26.9	0.5	13	25.0	1.0	7	16.3	0.5
45-54	5	26.3	0.4	7	24.1	0.5	6	23.1	0.5	6	11.5	0.5	6	14.0	0.5
55-64	3	15.8	0.3	4	13.8	0.4	2	7.7	0.2	7	13.5	0.7	4	9.3	0.4
65+	2	10.5	0.2	4	13.8	0.4	4	15.4	0.4	6	11.5	0.5	7	16.3	0.6
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	0	0.0	0.0	2	6.9	0.2	3	11.5	0.2	4	7.7	0.3	2	4.7	0.1
Black	1	5.3	0.1	1	3.4	0.1	0	0.0	0.0	3	5.8	0.4	1	2.3	0.1
Hispanic	8	42.1	0.2	11	37.9	0.2	6	23.1	0.1	16	30.8	0.3	8	18.6	0.2
White	9	47.4	0.3	15	51.7	0.6	15	57.7	0.6	12	23.1	0.5	14	32.6	0.5
Other	0	-	-	0	-	-	0	-	-	0	-	-	1	2.3	-
Unknown	1	5.3	-	0	-	-	2	7.7	-	17	32.7	-	17	39.5	-
SPA															
1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	2	3.8	0.5	2	4.7	0.5
2	4	21.1	0.2	6	20.7	0.3	7	26.9	0.3	11	21.2	0.5	11	25.6	0.5
3	2	10.5	0.1	3	10.3	0.2	3	11.5	0.2	5	9.6	0.3	5	11.6	0.3
4	4	21.1	0.3	4	13.8	0.4	5	19.2	0.4	9	17.3	0.8	4	9.3	0.3
5	1	5.3	0.2	6	20.7	0.9	5	19.2	0.8	9	17.3	1.4	7	16.3	1.1
6	3	15.8	0.3	3	10.3	0.3	2	7.7	0.2	6	11.5	0.6	4	9.3	0.4
7	2	10.5	0.1	3	10.3	0.2	0	0.0	0.0	3	5.8	0.2	6	14.0	0.5
8	2	10.5	0.2	4	13.8	0.4	4	15.4	0.4	5	9.6	0.5	4	9.3	0.4
Unknown	1	5.3	-	0	-	-	0	-	-	2	3.8	-	0	-	-



Figure 3. Percent Cases of Vibriosis by Race/Ethnicity LAC, 2015 (N=43)





Hispanic 19% White 33% Asian 5% Other 2% Unknown 40%







WEST NILE VIRUS

CRUDE DATA												
Number of Cases ^a	300											
Annual Incidence ^b												
LA County ^a	3.13											
California⁰	2.00											
United States [°]	0.68											
Age at Diagnosis												
Mean	60											
Median	63											
Range	12–98 years											

^aIncludes asymptomatic infections

^bCases per 100,000 population. CA and US rates do not include asymptomatic infections

^cCalculated Calculated from: CDC. *Notice to Readers:* Final 2015 Reports of Nationally Notifiable Infectious Diseases and Conditions *Weekly*/November 25, 2016/65(46);1306–1321. Available at:

www.cdc.gov/mmwr/volumes/65/wr/mm6546a9.htm

DESCRIPTION

West Nile virus (WNV) is a flavivirus related to the viruses that cause Japanese encephalitis (JE) and Saint Louis encephalitis (SLE). Indigenous to Africa, Asia, Europe, and Australia, WNV was first detected in North America in New York City in 1999. Since then, human and non-human WNV has been documented as an enzootic disease throughout the continental US, Canada, and Mexico.

Normally transmitted by mosquitoes (usually Culex or Anopheles species) between bird reservoir hosts, humans are incidentally infected with the virus when bitten by an infected mosquito. About 20% of persons infected will develop WNV fever with symptoms that include fever, headache, rash, muscle weakness, fatigue, nausea, vomiting, and occasionally lymph node swelling. Fewer than 1% will develop more severe illness, manifesting as WNV neuro-invasive disease (NID), including meningitis, encephalitis, and acute flaccid paralysis. WNV-associated meningitis usually involves fever, headache, and stiff neck and has a good prognosis. WNV-associated encephalitis is commonly associated with fever, altered mental status, headache, and seizures and usually necessitates a high level of specialized medical

care. Long-term neurological and cognitive sequelae are not uncommon.

After being infected with WNV, most people sustain a viremia and may remain asymptomatic or eventually develop symptoms. In 2002, asymptomatic blood donors were documented to transmit WNV to blood product recipients. Beginning in 2003, blood products have been screened for WNV utilizing polymerase chain reaction (PCR) testing. To date, there have been no blood transfusion-associated secondary WNV infections from asymptomatic WNV-infected blood donors from LAC residents. However, four cases of WNV-associated infection, including three with NID, were documented to be transmitted from an LAC organ donor in 2011 who was not known to be infected with WNV infection at the time of organ donation. Additional routes of transmission that can occur include vertical transmission transplacentally, breast milk, and occupational exposure.

Vector management programs are the most effective tools to prevent and control WNV and other arboviral diseases. These programs include surveillance for WNV activity in mosquitoes, birds, horses, other animals, and humans and implementation of appropriate mosquito control measures to reduce mosquito populations when necessary. Currently, there is no human vaccine available for WNV, but several vaccines are under development. Important preventive measures against WNV include the following:

- Apply insect repellant to exposed skin
- When possible, wear long-sleeved shirts and long pants outdoors, especially for long periods of time
- Stay indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water. This will reduce the number of places mosquitoes can lay their eggs and breed

A wide variety of insect repellent products are available. CDC recommends the use of products containing active ingredients that have been registered with the US Environmental Protection Agency (EPA) for use as repellents applied to skin and clothing. Products containing these active ingredients typically provide longer-lasting protection than others:

- DEET (N,N-diethyl-m-toluamide)
- Picaridin (KBR 3023)



• Oil of lemon eucalyptus IR3535 (3-[N-Butyl-N-acetyl]-aminopropionic acid, ethyl ester)

2015 TRENDS AND HIGHLIGHTS

- The incidence of WNV infections reported in 2015 (3.1 per 100,000 population) was the second highest documented in LAC since WNV first appeared in 2003 (Figure 1).
- Of 274 reported symptomatic WNV infections, there were 42 cases of WNV fever and 232 cases of NID (n=114, 38% encephalitis, n=107, 36% meningitis, n=10, 3% acute flaccid paralysis, and n=1, 0% other) (Figure 2). There were 24 WNV-associated deaths reported among symptomatic cases (9%). There were 26 asymptomatic donors (9%) reported from local blood banks (Figure 2).
- The mean age of all infections was 60 years old with the largest proportion ≥65 years old (n=141, 47%). Incidence increased with age (Figure 3).
- Similar to previous years, Whites and Hispanics comprised the majority of WNV infections (n=142, 47% and n=110, 37%, respectively).
- The male to female ratio was 1.9:1.
- WNV infections were distributed widely across all SPAs this year. The largest number of WNV infections continued to be identified in SPA 2, the San Fernando Valley area (n=92, 31%)

(Figure 5). Record counts of human infections were also documented in SPAs 2, 4, 5, and 7 compared to previous years. However, both SPA 5 (western LAC area) and SPA 7 (eastern LAC area) had the highest WNV incidence rates with 4.5 cases per 100,000 (n=30 and n=59, respectively).

- Starting in mid-October of 2015, the weekly WNV Epidemiology Report documented WNV infections by city (see www.publichealth.lacounty.gov/acd/docs/West %20Nile/WNVepi2015.pdf). This change was implemented to focus prevention messages to specific cities at higher risk for WNV infection. In 2015, residents within LA had the most WNV infections (n=66, 22%) followed by Glendale (n=21, 7%) and North Hollywood (n=18, 6%). In 2016, the weekly WNV Surveillance Report will divide the city of LA into established neighborhoods with 20,000 to 40,000 residents.
- In 2015, WNV infections occurred from July to November with the last case experiencing illness onset on November 20, 2015. Peak onset in 2015 occurred in September (n=116, 39%), which was similar to the previous fiveyear average (Figure 6). Notably, October (n=112, 37%) closely followed this trend. This may be attributable to October 2015 being the warmest October on record to date. Statewide, 860 infections were reported. A total of 2,060 infections were reported nationwide.



Reported WNV Infections and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA LAC, 2011-2015

	2011 (N=63)		2012 (N=174)			2013 (N=165)			20	14 (N=2	18)	2015 (N=300)			
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	/Rate 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
1-4	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
5-14	1	1.6	0.1	2	1.1	0.2	6	3.6	0.5	0	0.0	0.0	3	1.0	0.2
15-34	5	7.9	0.2	24	13.8	0.9	19	11.5	0.7	23	10.6	0.8	34	11.3	1.2
35-44	3	4.8	0.2	17	9.8	1.3	15	9.1	1.1	15	6.9	1.1	28	9.3	2.1
45-54	16	25.4	1.2	33	19.0	2.6	34	20.6	2.6	44	20.2	3.4	41	13.7	3.1
55-64	17	27.0	1.8	34	19.5	3.3	46	27.9	4.5	55	25.2	5.2	53	17.7	4.8
65+	21	33.3	2.0	64	36.8	5.8	45	27.3	4.1	81	37.2	7.2	141	47.0	11.8
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	1	1.6	0.1	9	5.2	0.7	6	3.6	0.4	11	5.0	0.8	7	2.3	0.5
Black	1	1.6	0.1	3	1.7	0.4	3	1.8	0.4	3	1.4	0.4	5	1.7	0.6
Hispanic	26	41.3	0.5	59	33.9	1.3	50	30.3	1.1	73	33.5	1.6	110	36.7	2.3
White	30	47.6	1.0	91	52.3	3.4	80	48.5	3.0	97	44.5	3.6	142	47.3	5.3
Other	2	3.2	-	2	1.1	-	2	1.2	-	0	0.0	0.0	1	0.3	-
Unknown	3	4.8	-	10	5.7	-	24	14.5	-	34	15.6	-	35	11.7	-
SPA															
1	1	1.6	0.3	10	5.7	2.6	15	9.1	3.8	2	0.9	0.5	4	1.3	1.0
2	39	61.9	1.8	73	42.0	3.4	62	37.6	2.9	60	27.5	2.7	92	30.7	4.1
3	16	25.4	0.9	47	27.0	2.9	23	13.9	1.4	34	15.6	2.1	46	15.3	2.8
4	1	1.6	0.1	18	10.3	1.6	6	3.6	0.5	28	12.8	2.4	41	13.7	3.5
5	1	1.6	0.2	8	4.6	1.3	2	1.2	0.3	24	11.0	3.7	30	10.0	4.5
6	1	1.6	0.1	2	1.1	0.2	4	2.4	0.4	13	6.0	1.3	15	5.0	1.4
7	4	6.3	0.3	13	7.5	1.0	24	14.5	1.8	45	20.6	3.4	59	19.7	4.5
8	0	0.0	0.0	3	1.7	0.3	29	17.6	2.7	11	5.0	1.0	13	4.3	1.2
Unknown	0	-	-	0	-	-	0	-	-	1	0.5	-	0	-	-




Year

Figure 3. Incidence Rates* of WNV Infection by Age Group, LAC, 2015 (N=300)



*Rates calculated based on less than 19 cases or events are considered unreliable.









□Asian
Black
Hispanic
White



*Rates calculated based on less than 19 cases or events are considered unreliable.

Figure 6. Reported WNV Infections by Month of Onset

Map 15. West Nile Virus Rates by Health District, Los Angeles County, 2015*





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COMMUNITY-ACQUIRED DISEASE OUTBREAKS

ABSTRACT

- In 2015, 334 community-acquired disease outbreaks accounted for 4,787 cases (Figure 1).
- Most (81%) of all outbreaks were from only three general disease categories: ectoparasites (38%), gastroenteritis (GE) (27%), and respiratory (16%) (Figure 2, Table 1).
- Three outbreak settings accounted for almost all (97%) of the reported outbreaks: preschools (44%), schools (31%) with the majority in elementary schools, and residential/assisted living settings (22%) (Figure 3, Table 2).
- Hand, foot, and mouth (HFM) disease declined from 39% in 2014 to 8% in 2015.



Only six outbreaks (2%) were caused by disease conditions that are individually reportable (Tables 1, 2).

DATA

А disease outbreak is an infection/infestation clustered in place and time with case numbers above expected for specified population or location. а Depending on the nature of the outbreak, the responsibility for the investigation is held by either ACDC or Community Health Services with ACDC providing as-needed consultation. The outbreaks reported in this section do not include outbreaks associated with food (see the Foodborne Outbreaks section) facilities specifically or regulated/licensed to provide medical care (see the Healthcare Associated Outbreaks section).



Ectoparasites Head lice and scabies were the top reported outbreak etiologies (n=128) for this category. Head lice (pediculosis) dominates the ectoparasite category with 100 reported outbreaks. Averaging six



cases per outbreak, head lice tends to occur in the youngest age groups with 92 of the 100 outbreaks in school settings, either preschool (72) or elementary school (20). Reporting of head lice outbreaks has increased steadily over the past five years (annual outbreak counts of 21, 33, 49, 50, and 80 from years 2010 to 2014, respectively), which has had an effect on the overall outbreak annual trends (Figure 5). While LAC DPH does not monitor whether lice are resistant to over-thecounter treatments, the prevalence of these resistant lice (also called "super lice"), which require prescriptions for treatment, has been increasing.



Scabies outbreaks were more common in the older risk group with 21 of 28 (75%) reported in residential/assisted-living settings (Table 2). Reported scabies outbreaks had a sharp increase in 2015 compared to the previous five-year average of 10 outbreaks annually.

The 89 GE outbreaks in 2015 were primarily caused by either an undetermined etiology (71) or norovirus (16); there were 2 reported *Shigellosis* outbreaks. GE outbreaks had the highest case per outbreak counts; norovirus outbreaks had a mean of 44 cases per outbreak (median 34), and unspecified GE outbreaks had

31 cases per outbreak (median 21) (Table 1). Many of the GE outbreaks of undetermined etiology had characteristics similar to the confirmed norovirus outbreaks, but specimens were not available for testing. The relative ability to obtain stool specimens from older individuals in a residential/assisted living facility compared with children in a school setting may be a factor why the majority (75%) of norovirus were confirmed in the former setting (Table 2). The GE figures for 2015 highlight the continuing circulation of norovirus and reflect the ease this agent can be transmitted from person-to-person in community settings.





Reported respiratory illness outbreaks, were seen predominately in the first part of 2015; 64% were in the first two months of the year (Figure 4). Most (67%) of the 18 confirmed influenza outbreaks occurred in January, and all were in the first 3 months. The number of confirmed influenza outbreaks as compared to those with an unspecified etiology (26) improved to the highest in 6 years. Respiratory outbreaks averaged 12 cases per outbreak, with one influenza outbreak in a preschool with 30 reported cases.

The graph of community-acquired outbreaks by report month (Figure 4) further illustrates the impact of

GE, ectoparasite, and respiratory outbreaks. These three disease categories accounted for majority of outbreaks each month throughout the year.

Looking at annual disease trend data, Figure 5 shows the three predominate etiologies over the years. The only two years where the three did not provide over 70% was 2012 and 2014, years with reporting surges in HFM.

Outbreaks were reported from all eight SPAs (Figure 6). SPA 3 (San Gabriel, 111) and SPA 2 (San Fernando, 63) have consistently had the most outbreaks over the past three years.

COMMENTS

Outbreaks are most often reported from locations with the ability to recognize an unusual occurrence of illness/infestation in a group of individuals and have a procedure in place/knowledge to report to the local health department. Thus, most community outbreaks are reported from schools, including preschools, and residential facilities.



Defining a cluster of illness as an outbreak can be problematic. With rare exception, a minimum of two cases occurring in time and exposure are required. An additional measure—cases above the usual number or background— also is used to define an outbreak situation. When ambiguity exists regarding whether



the number of cases are usual or unusual, the situation is typically labeled an outbreak. For the LAC DPH, all initial reports are considered suspect and are rapidly investigated. Even in situations where an outbreak designation is not met, rapid public health intervention can result in the mitigation of future cases and establishment of good relationships with facilities that may need public health assistance in the future.

There is a strong relationship between outbreak setting and the disease being reported. Characteristics of community-acquired outbreaks result from interactions among particular age groups, locations, and specific diseases. It is the epidemiologic characteristics of the three that lead to disease transmission and a potential outbreak. The predominance of outbreaks reported among children in educational settings (preschool to university) has been recognized in previous Annual Reports. In the preschool setting, pediculosis accounted for 49% of all preschool outbreak reports. While reduced from the previous year, HFM was the next highest incidence (16%). While illness is often linked to schools, in some cases, the true association with the school might be solely related to where the illness was identified and the reporting source rather than where the exposure/transmission occurred. Children who share a school setting often have other social interactions that could also account for the infection or infestation (e.g., sleepovers, parties, play dates, after school care, sports camps, etc.). However, regardless of the original exposure source, once cases are identified, schools need to be vigilant to prevent further transmission and can be greatly aided by the expertise of public health nurses in this effort.

The second most affected age group is an older population associated with residential/assisted living settings. In this older age category, scabies and GE each accounted for about a third (36%) of all outbreaks (Table 2). Nearly all of the confirmed norovirus outbreaks (75%) were in residential/assisted living sites.



Table 1. Community-Acquired Outbreaks by Disease				
Disease	No. of outbreaks	No. of cases	Cases per outbreak mean/median	Cases per outbreak (range)
Gastroenteritis:				
Norovirus	16	699	44/34	11-111
Shigella	2	23	12/12	9-14
Salmonella	0	0	0	0
<i>E</i> . coli	0	0	0	0
GE-Unknown	71	2205	31/21	3-133
Respiratory:				
Influenza	18	298	17/17	3-30
Streptococcal	7	50	7/10	2-14
Legionellosis	2	4	2/2	2
RespUnknown	26	272	10/9	3-26
Ectoparasites:				
Pediculosis	100	645	6/4	2-46
Scabies	28	197	7/5	2-25
Others:				
Hand, Foot & Mouth Disease	28	187	7/5	2-24
Typhus	1	6	6	6
Conjunctivitis	17	85	5/4	2-11
Varicella	1	5	5/5	5
Fifth disease	3	20	7/3	2-15
Other*	14	91	7/4	2-26
Total	224	1707	1 4 /6	2 4 2 2

 Total
 334
 4787
 14/6
 2-133

 * Includes Wound Botulism (1), Unknown rash (4), Staph (2), scarlet fever (1), Roseola (2), febrile unspecified (2), pinworm (1) and warts (1).



Table 2. Community-Acquired Outbreaks by Disease and Setting					
Disease	Residential/ assisted living	School ^a	Preschool or Daycare	Other ^b	TOTAL
Gastroenteritis:					
Norovirus	12	2	2	0	16
Shigella	0	1	1	0	2
Salmonella	0	0	0	0	0
<i>E</i> . coli	0	0	0	0	0
GE Illness-Unknown	21	32	18	0	71
Respiratory:					
Influenza	7	8	3	0	18
Streptococcal	0	6	1	0	7
Legionellosis	1	0	0	1	2
Respiratory-Unknown	7	14	5	0	26
Ectoparasites:					
Pediculosis	0	25	72	3	100
Scabies	21	2	2	3	28
Other:					
Hand, Foot & Mouth Disease	0	5	23	0	28
Typhus	0	0	0	1	1
Conjunctivitis	0	0	16	1	17
Varicella	0	0	1	0	1
Fifth disease	0	2	1	0	3
Other	4	5	3	2	14
Tota	I 73	102	148	11	334

^a Includes elementary (85) middle school (4) high school (12), and universities (1).
 ^b Includes rehab residence programs (4), trailer park (2), parks (2), girls residential programs (1), and day programs for disabled (1).



FOODBORNE OUTBREAKS 2015

DESCRIPTION

Foodborne outbreaks are caused by a variety of bacterial, viral, parasitic pathogens, and toxic substances. To be considered a foodborne outbreak, both the California Department of Public Health (CDPH) and the Centers for Disease Control and Prevention (CDC) require the occurrence of two or more cases of a similar illness resulting from the ingestion of a common food [1].

The surveillance system used by LAC DPH for detection of foodborne outbreaks typically begins with a Foodborne Illness Report (FBIR). FBIRs can be submitted by calling the LAC DPH Communicable Disease Reporting System Hotline (888-397-3993) or via the internet¹. The FBIR system monitors complaints from residents, illness reports associated with commercial food facilities, and foodborne exposures uncovered during disease-specific case investigations such as salmonellosis, shigellosis, and toxigenic *E. coli* including shiga toxin-producing *E. coli* (STEC). LAC Environmental Health Service's (EHS) Wholesale Food and Safety Program (WFS) investigates each FBIR by contacting the reporting individual and assessing the public health importance and need for expanded follow-up. When warranted, a thorough inspection of the facility is conducted. This public health action is often sufficient to prevent additional foodborne illnesses.

ACDC's Food Safety Unit also reviews all FBIRs. Joint investigations are conducted on possible foodborne outbreaks of public health importance. Typically, an epidemiologic investigation will be initiated when there are illnesses in multiple households, multiple reports against the same establishment in a short period of time, or there are ill individuals who attended a large event with the potential for others to become ill. The objective of each investigation is to determine the extent of the outbreak, identify a food vehicle or processing error, determine the agent of infection, and take actions to protect the public's health.

RESULTS

A total of 1,892 FBIRs were received in 2015, more than the 1,454 FBIRs received in 2014. Public reporting via the web accounted for 49% of FBIRs this year. WFS contacted each person making the FBIR and performed a site inspection on 26% of FBIR reports that were deemed high priority. The majority (55%) of the complaints were referred to district EHS offices, and 7% were referred to other EHS specialty programs (Vehicle Inspection, Street Vending Compliance, Drinking Water, etc.), other LAC departments (Department of Weights and Measures), or agencies outside LAC (other local health jurisdictions, state agencies, federal agencies). There were 101 FBIRs (17%) on which WFS did not take action or were duplicates.

The ACDC Food Safety Unit conducted 23 outbreak investigations this year. Of these, 21 outbreaks were initiated by FBIR complaints, and two were initiated through other surveillance activities. Of the 23

¹ www.visualcmr.net/webvcmr/pages/public/pub_FBI_Report.aspx



investigations, 3 (13%) were not considered to be foodborne because the evidence collected during the investigations did not support a foodborne source (Table 1). These outbreaks were due to norovirus, which can easily be spread person-to-person in a food setting if one guest is sick when attending. Another reason for these investigations not being considered to be foodborne outbreaks was because the illness pattern (epidemic curve) was consistent with person-to-person spread rather than point source infection. Determining whether a food item was the source in these outbreaks can be challenging as well as time and resource consuming.

The 20 outbreaks determined to be foodborne are listed in Table 1 and summarized below. These outbreaks represent 252 cases of foodborne illness (Figure 1), 5 hospitalizations, and no deaths. Outbreaks occurred throughout the year (Figure 2).

Etiology of Foodborne Outbreaks

<u>Cooked food items</u> Of the five outbreaks where a food item was found to be associated with illness, two involved cooked food items (outbreaks 274 and 297). One outbreak was due to norovirus (outbreak 274). Although norovirus is not usually associated with cooked food items, the dish associated with this outbreak may have been improperly handled during the plating or the service of the food or contaminated garnish items such as parsley.



The other outbreak (outbreak 297) involving a cooked food item was caused by *Salmonella*. It is likely that an item on the plate was not cooked and contaminated the implicated food item.

Uncooked food items

There were three outbreaks involving an uncooked food item (outbreaks 106, 169, and 279). In two of these, the etiologic agent was suspected to be a calicivirus such as norovirus. These items included raw oysters (outbreak 106) and guacamole (outbreak 169). For outbreak 106, the appeared have been oysters to contaminated prior to retail. The mode of contamination is less clear with outbreak 169. It is possible that the food was contaminated by a sick relative who made





or handled the food. Food handlers from the banquet facility were tested and had negative laboratory results.

The other outbreak involving uncooked food items was outbreak 279. This outbreak was suspected to be caused by a bacterial toxin.

Foodborne Agents

An etiological agent was identified in 22 of the 23 outbreak investigations this year and confirmed in 43% (n=10) (Figure 3). A viral agent was responsible for 18 outbreaks, bacterial agents for three outbreaks, and bacterial toxins for one outbreak (Figure 3).

Norovirus Outbreaks

Norovirus was confirmed or suspected in 18 foodborne outbreaks this year (78%), which is much more than was observed in 2014 but still considerably less than the peak number observed in 2006 (N=27).



There were two large, laboratory-confirmed foodborne norovirus outbreaks this year. The first (outbreak 106) involved at least 21 cases who ate at an all-you-can-eat-sushi restaurant in LAC. The incubation times were consistent with a point-source outbreak, and raw oysters were significantly associated with illness. Four patrons and two employees tested positive for norovirus. The oysters also tested positive for norovirus.

The second large laboratory-confirmed norovirus outbreak involved 26 cases who ate food served during an office meeting (outbreak 274). The symptoms and incubation periods were consistent with a point-source outbreak. Coconut shrimp and tempura vegetables were significantly associated with becoming ill. However, cooked food items such as these are unusual vehicles for norovirus.

Bacterial Outbreaks

Salmonella was confirmed in two outbreaks this year (outbreaks 145 and 297). The first salmonellosis outbreak (outbreak 145) occurred in persons eating at a restaurant that serves Mexican-style cuisine. A total of 13 confirmed and 10 probable cases ate at the restaurant during the same time period, but no common food item was identified. However, eight food handlers tested positive for *Salmonella*.



The second salmonellosis outbreak was also due to *Salmonella* Enteriditis. A total of 42 persons ate at the restaurant and became ill; 11 sought medical care and tested positive for *S*. Enteriditis. The suspected food item was truffle oil used as a garnish for many dishes.

There was one outbreak in which *Campylobacter* was the suspected etiology. Three people ate at the same restaurant and became ill. Only one was tested and found to be positive for *Campylobacter*. No food item was implicated.

Other Foodborne Outbreaks

There was one outbreak in which a bacterial toxin was identified as the source (outbreak 279). Nine cases ate together at a social gathering that was catered by an LAC caterer. The symptoms and duration of illness reported by cases were consistent with the ingestion of a toxin secreted by bacteria such as *B. cereus* [2]. Although the etiology of this outbreak was not laboratory-confirmed, the incubation times of cases are consistent with a point source exposure involving a bacterial toxin with exposure occurring at the time that the attendees reported eating food at the gathering. California sushi rolls, an unlikely source of bacterial toxin, were significantly associated with becoming ill.

Outbreak Locations

Exposure locations for reported foodborne outbreaks included restaurants (10), the workplace (4), a residence (3), a banquet hall (5), and a hotel. This year SPA 2 reported the largest number of outbreaks (31%) (Table 2). This is consistent with SPA 2 reporting the largest proportion of foodborne outbreaks since 2010, except in 2014.

State and National Investigations Involving LAC

ACDC staff assisted state and federal investigators with 55 *Salmonella*, 7 STEC, 4 *Shigella*, and 9 *Listeria* cluster investigations that required additional investigation such as specialized interviews, product traceback, and extra laboratory testing.



	Table 1. 1 boubbine Oubleak investigation 2013 (N=23)						
	Agent	Laboratory Confirmed*	OB#	Setting	# Cases	HD	Food Implicated
1	Norovirus	No	20	Restaurant	3	West	none
2	Unknown	No	101	Restaurant	5	West Valley	none
3	Norovirus	Yes	106	Restaurant	21	Pomona	oysters
4	Norovirus	No	109	Banquet Hall	12	Whittier	none
5	Norovirus	No	129	Residence	25	San Fernando	none
6	Salmonella Enteriditis	Yes	145	Restaurant	23	West	none
7	Norovirus	Yes	151	Restaurant	8	West	none
8	Campylobacter	No	179	Restaurant	3	Torrance	none
9	Norovirus	Yes	168	Restaurant	9	San Fernando	None
10	Norovirus	No	169	Banquet Hall	23	West	Guacamole
11	Norovirus	No	174	Restaurant	16	Pomona	none
12	Norovirus	No	201	Residence	3	San Fernando	none
13	Norovirus	No	270	Restaurant	5	Bellflower	none
14	Norovirus	Yes	274	Workplace	26	Central	coconut shrimp, tempura vegetables
15	Bacterial Toxin	No	279	Banquet Hall	9	San Fernando	California sushi roll
16	Norovirus	No	284	Workplace	4	Central	none
17	Norovirus	No	287	Workplace	4	San Fernando	none
18	Salmonella Enteriditis	Yes	297	Restaurant	42	Hollywood/ Wilshire	truffle mushroom croquette
19	Norovirus	Yes	350	Banquet Hall	14	El Monte	none
20	Norovirus	No	401	Restaurant	4	Bellflower	none
21	Norovirus	No	417	Workplace	4	West	none
22	Norovirus	No	419	Hotel	9	West	none
23	Norovirus	No	436	Banquet Hall	4	Glendale	none

Table 1. Foodborne Outbreak Investigation 2015 (N=23)

*Etiology of the outbreak was confirmed with two or more patrons having positive laboratory results for the infectious agent.



Table 2. Frequency of Foodborne Outbreaks by	
Service Planning Area or Location, LAC, 2015 (N=23)	

SPA	Frequency	Percent
1	0	0%
2	7	31%
3	3	13%
4	3	13%
5	6	26%
6	0	0%
7	3	13%
8	1	4%

ADDITIONAL RESOURCES

LAC resources

- Communicable Disease Reporting System Hotline: (888) 397-3993 Fax: (888) 397-3779
- For reporting and infection control procedures consult the LAC DPH ACDC www.publichealth.lacounty.gov/acd/index.htm

<u>CDC</u>

- Division of Foodborne, Waterborne, and Environmental Diseases (DFWED) www.cdc.gov/ncezid/dfwed/
- Outbreak Response and Surveillance Team www.cdc.gov/foodsafety/outbreaks/index.html
- FoodNet www.cdc.gov/foodnet
- Norovirus Information www.cdc.gov/norovirus/index.html

Other national agencies

- FDA Center for Food Safety and Applied Nutrition www.fda.gov/AboutFDA/CentersOffices/OfficeofFoods/CFSAN/
- Gateway to Government Food Safety Information
 www.FoodSafety.gov

REFERENCES

- 1. Centers for Disease Control and Prevention. Surveillance for foodborne disease outbreaks United States, 2006. *MMWR*. 2009;58(22):609-615.
- Food and Drug Administration. <u>Bad Bug Book, Foodborne Pathogenic Microorganisms and Natural Toxins</u>. Second Edition. [*Bacillus cereus* and other *Bacillus* species, pp 96-99]. 2012. Accessible online at: www.fda.gov/downloads/Food/FoodbornellInessContaminants/UCM297627.pdf



HEALTHCARE-ASSOCIATED OUTBREAKS GENERAL ACUTE CARE HOSPITALS

DEFINITION

This chapter will discuss healthcare-associated outbreaks and situation events that occurred within the general acute care hospital setting on any patient unit, sub-acute or specialty area, within the facility (surgical suites or procedure rooms). An outbreak in such settings is defined as a cluster of infections or colonizations related in time and place or occurring above a baseline or threshold level for a defined area of a facility, including the entire facility, specific unit, or ward. Baseline is relative to what is normally observed in a particular setting.

A situation event is defined as a cluster of infections or colonizations in the setting of a



general acute care hospital that may not clearly meet all outbreak criteria defined above or that requires additional information to determine if an outbreak has occurred.

ABSTRACT

There were 19 confirmed outbreaks reported in acute care hospitals in 2015 (Figure 1). Most (n=13, 68%) occurred in a unit providing intensive or focused specialized care (long-term acute care, pediatric subacute, and neonatal intensive care unit (NICU). Two outbreaks involved patients who became carbapenem-resistant *Klebsiella pneumoniae* (CRKP) positive after an endoscopic retrograde cholangiopancreatography (ERCP) procedure (Table 2). Similarly, a situation event involved patients who became *Pseudomonas aeruginosa (P. aeruginosa*) positive after an ERCP procedure (Table 4). One-third (37%, n=7) of acute care hospital outbreaks were of bacterial etiology, often from a multidrug-resistant organism (MDRO) such as *Acinetobacter baumannii (A. baumannii)* as shown in Table 2 and Figure 2. Scabies accounted for the greatest number of outbreaks (n=8) followed by *A. baumannii* (n=2) and CRKP (n=2). There were four situation events investigated in acute care hospitals in 2015 (Table 4).

Table 1. General Acute Care Hospital Outbreaks by Unit LAC, 2015 (N=19)		Table 2. General Acute Care Hospital Outbreaks by Disease/Condition/Etiologic Agent			
Outbreak Location	No. of Outbreaks		<u>, 2015</u>	NI	
Hematology/oncology	1	Etiologic Agent	NO. Of Outbreaks	NO. Of Cases	
Intensive Care – Adult	1	A. baumannii	2	8	
Intensive Care- Neonatal	4	B. cereus	1	4	
Liver transplant	1	E. coli	1	5	
Long-term acute care	6	Enterobacter Klebsiella pneumoniae.	1	4	
Multiple units	3	carbapenem-resistant	2	20	
Skilled Nursing Facility	2	Mucormycosis	1	8	
Sub-acute Unit within a Hospital - Pediatrics	1	Norovirus	1	16	
Total	19	MIKSA Davi	1	6	
		RSV	1	2	
		Scabies	8	51	
		Total	19	124	

Table 3. General Acute Care Hospital Situation Events by Unit LAC, 2015 (N=4)			
Outbreak Location	No. of Events		
Multiple Units	3		
Medical/Surgical 1			
Total	4		

Table 4. General Acute Care Hospital Situation Events by Disease/Condition LAC, 2015					
Disease/Condition/ Etiologic Agent	No. of Events	No. of Cases			
Candida albicans	1	4			
Klebsiella pneumoniae, carbapenem resistant	1	2			
Pseudomonas aeruginosa	1	16			
Unknown GI	1	17			
Total	4	39			





COMMENTS

Infections due to antibiotic resistant bacteria, particularly multidrug-resistant bacteria, have increased worldwide. Strategic prevention and control efforts to combat and prevent these infections were developed that recommend a multi-pronged approach that incorporates antimicrobial stewardship (AS) in hospital infection prevention and control programs [1, 2, 3].

In Los Angeles County (LAC), 103 acute care hospital outbreaks were reported between 2011 and 2015. Of these, 43% (n=44) were caused by a multidrug-resistant organism (MDRO). In 2015, 7 of 19 reported outbreaks were caused by a MDRO (Table 2). In California, hospitals were mandated to implement an antimicrobial stewardship policy to develop a process for evaluating the judicious use of antibiotics and develop a physician supervised multidisciplinary committee (SB 1311, Health and Safety Code, 1288.85). In March 2015, The White House introduced the National Action Plan for Combating Antibiotic-Resistant Bacteria with a "primary purpose to guide action by public health …in a common effort to address urgent and serious drug-resistant threats..." [4].

LAC Department of Public Health (DPH) implemented several strategies to collaborate with hospitals on preventing and understanding antibiotic resistance. First, LAC DPH established the LAC Healthcare-Associated Infections and Antimicrobial Resistance Committee, a multidisciplinary partnership to address the issue. Community partners include infectious disease physicians and pharmacists, infection preventionists, LAC Emergency Medical Services, the Public Health Laboratory, dialysis services, clinical microbiologists and academic researchers. A work plan was developed with several objectives that align with California Department of Public Health (CDPH) AS goals, including the development of a standardized



community antibiogram, improving AS practices in hospitals, and improving inter-facility communication when patients diagnosed with antibiotic-resistant organisms are transferred to another facility.

Another HAI prevention strategy was to provide targeted, non-punitive infection control assessments to hospitals. The assessments are held on-site at selected hospitals and conducted by ACDC public health nurses and epidemiologists. The goal of the assessments is to better characterize the current status of HAI prevention, identify common gaps, and determine how ACDC can work collaboratively with LAC healthcare facilities to address those gaps.

Contaminated duodenoscopes were implicated in two CRKP outbreaks investigated by ACDC and one (*P. aeruginosa*) situation event consultation that occurred out of jurisdiction. The duodenoscope is a complex reusable medical device that requires high-level cleaning, disinfection, and reprocessing. It is used during an ERCP procedure to visualize the liver, gallbladder, pancreas, and biliary tract. Multiple reports of outbreaks after ERCP have been documented in the literature [5, 6]. Improper cleaning, disinfection, and/or reprocessing was frequently cited as the cause of the outbreaks, although we did not observe these practices during our investigations. In March 2015, the US Food and Drug Administration (FDA) proposed that "the complex design of the duodenoscope may impede effective reprocessing" [7]. Guidance and recommendations on duodenoscope cleaning, disinfection, and reprocessing were provided to hospitals and providers by government agencies (Centers for Disease Control and Prevention (CDC), FDA, and CDPH), professional infection control associations (Association for Professionals in Infection Control (APIC) and Society for Healthcare Epidemiology of America (SHEA)), gastrointestinal professional organizations (American Society for Gastrointestinal Endoscopy), and the duodenoscope manufacturer.

REFERENCES

- 1. Moody, J, Cosgrove, S and Olmsted, R et al. Antimicrobial stewardship: A collaborative partnership between infection preventionists and Health care epidemiologists, 2012: vol.40, p.94-5. Available at: www.apic.org/Resource_/TinyMceFileManager/Practice_Guidance/APIC_SHEA_Antimicrobial_Stew ardship_Position_Statement.pdf Accessed July 20, 2016.
- 2. Sandora, Thomas J. and Goldmann, Donald A., Preventing Lethal Hospital Outbreaks of Antibiotic-Resistant Bacteria, 2012: N Engl J Med; 367:2168-2170
- Weiner, Lindsey M., Fridkin, Scott K., Aponte-Torres, Z. et al. Vital Signs: Preventing Antibiotic-Resistant Infections in Hospitals—United States, 2014, US Dept. of Health and Human Services/CDC Morbidity and Mortality Weekly Report (MMWR), March 11, 2016, Vol. 65, No.9.
- 4. National Action Plan for Combating Antibiotic-Resistant Bacteria, March 2015, the White House, Washington. www.obamawhitehouse.archives.gov/blog/2015/03/27/our-plan-combat-and-prevent-antibiotic-resistant-bacteria
- Kristen A Wendorf, Meagan Kay, Christopher Baliga, et al. (2015). Endoscopic Retrograde Cholangiopancreatography—Associated AmpC *Escherichia coli* Outbreak. Infection Control & Hospital Epidemiology, 36, pp 634-642 doi:10.1017/ice.2015.66
- 6. William A. Rutala and David J. Weber (2015). ERCP Scopes: What Can We Do to Prevent Infections? Infection Control & Hospial Epidemiology, 36, pp 643-648 doi:10.1017/ice.2015.98



 U.S. Food and Drug Administration, Design of Endoscopic Retrograde Cholangiopancreatography (ERCP) Duodenoscopes May Impede Effective Cleaning: FDA Safety Communication, March 4, 2015. Updated Information for Healthcare Providers Regarding Duodenoscopes (downloads/MedicalDevices/DeviceRegulationandGuidance/ReprocessingofReusableMedicalDevice s/UCM436588.pdf. Accessed at www.fda.gov/MedicalDevices/Safety/AlertsandNotices/ucm434871.htm.





HEALTHCARE-ASSOCIATED OUTBREAKS SUB-ACUTE CARE FACILITIES

DEFINITION

Healthcare-associated outbreaks are defined as clusters of infections in healthcare settings related in time and place, or occurring above a baseline or threshold level for a facility, specific unit, or ward. Baseline is defined as what is normally observed in a particular setting.

The sub-acute care facilities include freestanding dialysis centers, skilled nursing facilities (SNF), intermediate care facilities and psychiatric care facilities. SNFs provide continuous skilled nursing care and supportive care to patients whose primary need is for availability of skilled nursing on an extended



basis. Intermediate care facilities also provide inpatient care to patients who have need for skilled nursing supervision and need supportive care, but who do not require continuous nursing care. Psychiatric care facilities provide 24-hour inpatient care for patients with psychiatric care needs.

ABSTRACT

- The total number of confirmed sub-acute care associated outbreaks in 2015 increased by 13% (85 to 96 outbreaks) from the previous year.
- In 2015, the number of SNF outbreaks reported increased by 15% (from 82 to 94 outbreaks) from the previous year (Table 1). The rate of SNF outbreaks was 24 per 100 facilities in 2015 compared with 21 per 100 in 2014. (Figure 1).
- Outbreaks occurred in intermediate care facilities, psychiatric care facilities, and SNFs in 2015 (Table 1).



Table 1. Number of Reported Outbreaks in Sub-Acute Healthcare Facilities LAC, 2011–2015					
	YEAR				
Type of Facility	2011	2012	2013	2014	2015
Intermediate Care Facilities	4	2	1	3	1
Psychiatric Care Facilities	3	3	1	-	1
Dialysis Centers	1	-	-	-	-
Skilled Nursing Facilities	102	119	96	82	94
Total	110	124	98	85	96

Intermediate Care Facilities: One unknown GE outbreak with 36 cases was reported in an intermediate care facility in 2015.

Psychiatric care facilities: One scabies outbreak with 43 cases was reported in a psychiatric care facility in 2015.

SNFs: A total of 94 outbreaks were reported by SNFs. Rash illness outbreaks were the most frequently reported outbreak category, with 36 (38%) outbreaks and 392 associated cases. In 2014, GE outbreaks were most frequently reported with 36 (44%) outbreaks with 763 cases.

Table 2. All Sub-Acute Healthcare Facilities Outbreaks by Disease/Condition LAC, 2015						
Disease/Condition	No. of Outbreaks	No. of Cases				
Gastroenteritis	(N=29)	(N=592)				
Unspecified	17	269				
Norovirus	11	315				
Clostridium difficile	1	8				
Rash Illness	(N=36)	(N=435)				
Atypical Scabies	7	17				
Scabies	16	294				
Unknown Rash	13	124				
Respiratory Illness	(N=31)	(N=632)				
Unspecified	6	113				
Influenza	23	514				
Legionella	2	5				
Total	96	1,659				

COMMENTS

In 2015, the total number of outbreaks within sub-acute care facilities increased by 13% as compared to the previous year. Rash illness was the most frequently reported outbreak category (37%), and respiratory illness outbreaks contributed the greatest number of outbreak-associated illnesses (38%).



The total number of reported rash illness outbreaks increased by 14% in 2015 compared to 2014 from 31 to 36 outbreaks. Of these reported outbreaks occurring in 2015, most (44%) were due to atypical scabies. SPA 3 reported the most rash illness outbreaks (n=12, 33%) followed by SPA 7 (n=11, 30%).

The total number of reported respiratory outbreaks increased by 61% (from 12 to 31 outbreaks) as compared to the previous year. The mismatch in the influenza A (H3N2) component of the vaccine most likely accounted for the increase in respiratory outbreaks. The 2014-2015 seasonal vaccine was not a good match to the dominant circulating strain and vaccine efficacy (VE) against A (H3N2) viruses was estimated at 18% (95% confidence interval (CI): 6%-29%); however, VE against influenza B was estimated at 45% (95% CI: 14%-65%).¹ A total of 31 respiratory outbreaks were investigated causing 632 cases of outbreak-associated illness. Of the 31 outbreaks, 23 (74%) were caused by influenza virus, six (19%) were due to unknown etiologies, and two (6%) were caused by *Legionella*. Respiratory outbreaks were classified as influenza if there was at least one case of laboratory-confirmed influenza in the setting of a cluster of influenza-like illness within a 48-72 hour period.

GE illness outbreaks in 2015 included 11 (38%) laboratory-confirmed norovirus, 17 (59%) unknown GE, and 1 (3%) *Clostridium difficile* outbreak. SPA 3 consistently reported the most GE outbreaks of any Los Angeles County Department of Public Health (LAC DPH) SPA since 2008 with 12 (41%). The reasons for these associations are possibly due to continuous outreach activities to SNFs in SPA 3 on reporting requirements and prevention and control of GE outbreaks using the "Norovirus Outbreak Prevention Toolkit", which was developed in 2012 with ACDC and SPA 3 LAC DPH nurses, and a higher number of SNFs in this SPA compared to other SPAs. Per the Centers for Disease Control and Prevention (CDC), health care facilities, including nursing homes and hospitals, are the most commonly reported settings for norovirus outbreaks in the US and other industrialized countries. Over half of all norovirus outbreaks reported in the US occur in long-term care facilities. The virus can be introduced into healthcare facilities by infected patients—who may or may not be showing symptoms—or by staff, visitors, or contaminated foods. The duration of outbreaks in these settings can be quite long, sometimes lasting months. Illness can be more severe, occasionally even fatal, in hospitalized or nursing home patients compared with otherwise healthy people.²

Sub-acute facility outbreaks were investigated and documented from all LAC SPAs in 2015. The greatest proportion of outbreaks were investigated within SPA 3 with 23 (24%) followed by SPA 2 with 19 (20%). This was consistent with outbreak reports in previous years.

PREVENTION

The majority of outbreaks in sub-acute care facilities are caused by agents spread by person-to-person contact. Thus, appropriate hand hygiene practice by staff and residents is a crucial infection control measure. Influenza vaccination for sub-acute facility staff and residents as well as proper hand washing,

¹ CDC, Situation Update: Summary of Weekly FluView Report http://www.cdc.gov/flu/weekly/summary.htm

² CDC. Norovirus U.S. Trends and Outbreaks www.cdc.gov/norovirus/trends-outbreaks.html



administrative controls, utilization of appropriate antiviral treatment and prophylaxis for facility residents and staff, and isolation are essential in the prevention of seasonal influenza.

In 2014, ACDC created the Skilled Nursing Facility (SNF) Outreach Program (OP). The purpose of this program is to support our public health mission to prevent and control communicable diseases in SNF settings. ACDC continues to engage in collaborations with stakeholders, provide assistance and health education, and develop resources to prevent infections, strengthen outbreak detection and response, and address other acute communicable disease issues in SNFs.

As part of influenza prevention efforts, ACDC SNF OP sent a reminder letter to SNFs prior to the 2014-2015 influenza season to comply with the Health Officer Order (HOO), issued October 2, 2013, which mandates that healthcare personnel in acute care hospitals, long term care facilities, and intermediate care facilities in LAC be vaccinated against influenza or wear a protective mask. A toolkit for influenza vaccination programs in SNFs was developed (www.publichealth.lacounty.gov/acd/SNFToolKit.htm). In addition, in 2015, ACDC partnered with Community Health Services (CHS) to develop the manual Influenza Outbreak Prevention and Control Guidelines for Skilled Nursing Facilities (www.publichealth.lacounty.gov/acd/InfluenzaOBGuidelines.htm). The purpose of this manual is to provide a standardized guidance for CHS when conducting influenza and respiratory outbreak investigations in SNFs. Printed guidelines were distributed to CHS Public Health Nurses (PHN) and SNFs during outreach activities.

To assist facilities with management of scabies outbreaks, LAC DPH's *Scabies Prevention and Control Guidelines Acute and Long-Term Care Facilities* was initially developed in 2009 and updated in 2015. The printed guidelines were distributed to CHS PHNs and SNFs during outreach activities and is available at: www.publichealth.lacounty.gov/acd/Diseases/ScabiesToolkit.htm.

In 2015, SPA 3 PHNs continued outreach to SNFs in SPA 3 using the "Norovirus Outbreak Prevention Toolkit", which was developed in the spring of 2012 by ACDC in collaboration with CHS, Health Facilities Inspection Division, Licensing and Certification Program, and Environmental Health in response to an increasing number of GE outbreaks reported by sub-acute facilities. The online toolkit is available at www.publichealth.lacounty.gov/acd/docs/Norovirus/NoroToolkit2012.pdf.

In collaboration with CHS, ACDC presented the 2015 Symposium on Infection Prevention and Control in Skilled Nursing Facilities on September 1, 2015 at the California Endowment. The symposium was designed to provide PHNs, infection preventionists, and administrators with resources and strategies to prevent and control common communicable disease outbreaks within SNFs such as influenza, norovirus, and scabies in SNFs. At the symposium, ACDC provided printed copies of many useful resources and materials including the *Influenza Outbreak Prevention and Control Guidelines for SNFs, Norovirus Outbreak Prevention Toolkit*, and Scabies Prevention and Control Guidelines for Acute and Long-Term Care Facilities.



LOS ANGELES COUNTY DEPARTMENT OF PUBLIC HEALTH ACUTE COMMUNICABLE DISEASE CONTROL PROGRAM 2015*

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ACUTE COMMUNICABLE DISEASE CONTROL 2015 ANNUAL MORBIDITY REPORT

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•	Legionellosis	Juliet Bugante, RN, PHN
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•	Listeriosis, Nonperinatal	Michelle Chan, MPH
•	Listeriosis, Perinatal	Michelle Chan, MPH
•	Meningitis, Viral	Van Ngo, MPH
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ACUTE COMMUNICABLE DISEASE CONTROL SPECIAL STUDIES REPORT 2015

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BOTULISM CASE REPORT SUMMARY LOS ANGELES COUNTY, 2015

Botulism is a rare but serious and potentially fatal paralytic illness caused by a nerve toxin produced by the bacterium Clostridium botulinum. The bacterial spores which causes botulism are common in both soil and water and produce botulinum toxin when exposed to low oxygen levels and certain temperatures. There are five main kinds of botulism: 1) Foodborne botulism can happen by eating foods that have been contaminated with botulinum toxin. Common sources of foodborne botulism are homemade foods that have been improperly canned, preserved, or fermented. Though uncommon, store-bought foods also can be contaminated with botulinum toxin, 2) Wound botulism can happen if the spores of the bacteria get into a wound and make a toxin. People who inject drugs have a greater chance of getting wound botulism. Wound botulism has also occurred in people after a traumatic injury, such as a motorcycle accident, or surgery, 3) Infant botulism can happen if the spores of the bacteria get into an infant's intestines. The spores grow and produce the toxin which causes illness. 4) Adult intestinal toxemia (also known as adult intestinal toxemia) botulism is a very rare kind of botulism that can happen if the spores of the bacteria get into an adult's intestines, grow, and produce the toxin (similar to infant botulism). Although we don't know why people get this kind of botulism, people who have serious health conditions that affect the gut may be more likely to get sick, 5) Latrogenic botulism can happen if too much botulinum toxin is injected for cosmetic reasons, such as for wrinkles, or medical reasons, such as for migraine headaches.

Because botulism infections may be fatal, they are considered medical emergencies and suspected cases are mandated to be reported to the Los Angeles County Department of Public Health (LAC DPH) immediately by telephone. The California Department of Public Health's (CDPH) Division of Communicable Disease Control is responsible for the investigation and surveillance of infant botulism cases identified in the county and across the state. LAC DPH is responsible for reporting suspected cases of infant botulism to CDPH's Infant Botulism Treatment and Prevention Program¹ for their investigation. Specialized antitoxin is used to treat botulism, which can only be released when authorized by LAC DPH or CDPH. Testing for case confirmation can be conducted at the LAC DPH Public Health Laboratory.

The number of confirmed botulism cases in LAC fluctuates from year to year. For the past 5 years, an average of three cases were confirmed annually.

In 2015, two associated cases of suspected botulism were reported in LAC: one was classified as probable (Case 1) and the other as confirmed (Case 2). Both cases had wound botulism, lived in the same sober living house, and reportedly used heroin together including using shared needles. Case 2 had onset of symptoms 11 days after Case 1's symptom onset. Botulinum toxin A was detected by mouse bioassay in a serum specimen from Case 2. The serum for Case 1, collected approximately 3 weeks after initial onset

¹ Infant Botulism Treatment and Prevention Program. Division of Communicable Disease Control, California Department of Public Health. www.infantbotulism.org.



of symptoms, was negative for botulinum toxin. However, because Case 1 had clinically compatible symptoms and was epidemiologically linked with Case 2, Case 1 was classified as a probable case.

In 2015, ACDC also received three other reports of suspected botulism which were ultimately not classified as cases. One had a history of injection drug use; serum testing was negative by matrix-assisted laser desorption/ionization-time of flight (MALDI-TOF), as a result an alternate diagnosis of myasthenia gravis was assigned. Another suspected case had a history of crystal methamphetamine use, but denied injection use. For this suspected case, serum testing by both mouse bioassay and MALDI-TOF were negative. The third suspected case had no identified risk factors. Serum testing by MALDI-TOF and serum/stool testing by mouse bioassay were negative, and EMG results were determined not to be consistent with botulism.

Upon notification and review of case history and symptoms, LAC DPH authorized the release and use of botulism antitoxin for all five suspected botulism cases reported in 2015.



MONITORING WEST AFRICAN TRAVELERS FOR EBOLA VIRUS DISEASE IN LOS ANGELES COUNTY: A COMPLETE REVIEW

OVERVIEW

The outbreak of Ebola virus disease (EVD) in West Africa was the largest outbreak of EVD in history, and the first Ebola outbreak which resulted in transmission of this disease in the US. The outbreak in West Africa began in March 2014. However, implementation of a nationwide monitoring system in the US did not begin until a West African traveler was diagnosed in Dallas, Texas with EVD in September of that year, and EVD subsequently spread to two nurses who treated this patient.

Starting in October 2014, US government officials responded by initiating questioning of airplane passengers from West Africa for possible EVD exposure and screening these travelers for fever. This occurred at five US airports in New York, New Jersey, Illinois, Virginia, and Georgia. Combined, these five airports receive more than 94% of passengers from Guinea, Liberia, and Sierra Leone, the three countries that were most affected during this EVD outbreak. On October 21, the Department of Homeland Security announced that all passengers from Liberia, Sierra Leone, and Guinea would be required to fly into one of those five airports. On October 23, the Centers for Disease Control and Prevention (CDC) announced that all passengers from these countries also would receive 21-day monitoring while in the US [1].

On October 21, 2014, the Los Angeles County Department of Public Health (LAC DPH) was notified of the first traveler to our jurisdiction. Traveler monitoring for EVD ultimately ended on January 4, 2016. This report provides a summary of the entire Ebola traveler monitoring effort in Los Angeles County (LAC).

METHODS

In order to assess traveler risk of developing EVD and to implement daily symptom monitoring, LAC DPH created the EVD Exposure Risk Assessment Form and the EVD Daily Symptom Monitoring Log based on guidance materials released by the CDC. CDC guidance also was used to assign travelers to one of four risk groups: no identifiable risk, low risk, some risk, and high risk [2]. Initial data was collected on travelers by the US Customs and Border Protection and the CDC during a screening process at one of the five airports accepting travelers from Ebola affected countries. Data were then received by LAC DPH through the California Department of Public Health (CDPH). Upon notification, LAC DPH personnel visited the travelers and conducted an interview to complete the EVD Risk Assessment Form. Travelers were then monitored daily by district public health nurses for EVD symptoms for up to 21 days after the travelers' last potential exposure to EVD. The primary EVD symptom assessed was fever, but symptoms monitored also included: severe headache, abdominal pain, diarrhea, vomiting, muscle pain, weakness or fatigue, and unexplained bleeding or bruising (hemorrhage). Low risk travelers were contacted daily by a LAC DPH Public Health Nurse (PHN) by telephone. Travelers that were determined to be at some risk for developing EVD were contacted daily by LAC DPH staff either in-person or through video conferencing. None of the travelers in LAC were determined to be at high risk for developing EVD. Travelers who reported fever or other symptoms of EVD were evaluated by an LAC DPH physician to determine whether further follow-up or



EVD testing was necessary. If the traveler met the criteria, they were tested for EVD by polymerase chain reaction (PCR) at the LAC DPH Public Health Laboratories.

Early in the response, the initial paper based protocol was merged into an electronic surveillance system which centralized the data and allowed for conducting queries. Analyses were performed using SAS[®] and Microsoft Access. Surveillance data were summarized daily and reports were disseminated to key stakeholders, which described LAC DPH's ongoing traveler monitoring activities and the current health status of those being monitored.

This report covers the entire traveler monitoring period, which started on October 21, 2014 and ended on January 4, 2016.

RESULTS

Over the full course of the US response, 269 travelers were referred to LAC DPH for monitoring. Of these, 20 travelers were not monitored, either because it was determined that they were never exposed to EVD or because they were not residents of LAC (Figure 2). Of the 249 travelers monitored by LAC DPH, 40 (16%) reported EVD-related symptoms during at least one monitoring event. In nearly all cases, symptoms resolved quickly and without need for further assessment. LAC DPH determined that medical assessment was needed for eight travelers, however only four met the criteria for EVD testing—none of those tested were positive for EVD, and all eight medically assessed travelers had a non-EVD diagnosis (Figure 2).

Most of the travelers that LAC DPH monitored came from Sierra Leone (120, 48%), followed by Liberia (69, 28%), and Guinea (47, 19%). Six travelers (2%) reported travel from two EVD-affected countries. The largest proportion of travelers (82, 33%) were in an EVD-affected area for business, followed by travel for vacation or visiting family (63, 25%). Many of the travelers LAC DPH monitored (49, 20%) were permanent residents of one of the EVD-affected areas (Table 1).

Of the 249 travelers: 193 were monitored for the full 21-day infectious period, 32 were not monitored for the full period either because they left the country or because CDPH authorized ending their monitoring. A total of 24 travelers transferred to other jurisdictions during their monitoring period. Only two travelers to LAC (0.8%) had contact with an EVD case within their incubation period. The majority of travelers in LAC (238, 96%) were low risk for their entire monitoring period, four were some risk, and seven were considered to be some risk for part of their monitoring period and later were downgraded to low risk (Table 1). None of the travelers to LAC were considered at high risk for developing EVD. Travelers were mostly male (144, 58%), only one traveler was pregnant, and 11 (4%) were under age 18.

CONCLUSION

LAC DPH was able to adapt existing surveillance systems to meet the needs of the Ebola response. Through this system, LAC DPH was able to detect symptomatic travelers, determine need for further assessment and activate a countywide response as necessary. The protocols and data systems that were established were able to effectively monitor travelers over the entire duration of the outbreak. Response staff were also able to effectively transmit timely information to key LAC DPH staff and other stakeholders. Clear and


frequent communication between LAC DPH, CDPH, and CDC partners was vital to the success of our response, and allowed for the flexibility necessary to adapt to this quickly changing situation with wide reaching public health implications. In addition, the systems LAC DPH developed and the lessons learned have been instrumental in our response to other emerging diseases, including Zika.

REFERENCES

- CDC. Notes on the Interim U.S. Guidance for Monitoring and Movement of Persons with Potential Ebola Virus Exposure. www.cdc.gov/vhf/ebola/exposure/monitoring-and-movement-of-personswith-exposure.html
- 2. CDC. Epidemiologic Risk Factors to Consider when Evaluating a Person for Exposure to Ebola Virus. www.cdc.gov/vhf/ebola/exposure/risk-factors-when-evaluating-person-for-exposure.html

Table 1. Characteristics of Travelers Monitored for EVD LAC, 2014-2016						
	Frequency	Percent				
Gender						
Male	144	58				
Female	105	42				
Affected Areas Visited						
Guinea	47	19				
Guinea and Sierra Leone	3	1				
Liberia	69	28				
Liberia and Sierra Leone	3	1				
Mali	7	3				
Sierra Leone	120	48				
EVD Risk						
Low	238	96				
Some	4	2				
Some, Low	7	3				
High	0	0				
Reason for Travel to EVD-Affected Area						
Business	82	33				
Visiting Family or Vacation	63	25				
Ebola-Response or Humanitarian Aid	37	15				
Permanent Resident of Affected Area	49	20				
>1 reason	4	2				
Other	14	6				









* Travelers were not monitored by LAC DPH if they were not residents of LAC, or if risk assessments determined that they did not have exposure to EVD.



PREGNANCY, LABOR, AND DELIVERY AFTER EBOLA VIRUS DISEASE AND IMPLICATIONS FOR INFECTION CONTROL IN OBSTETRIC SERVICES, US²

OVERVIEW

Many of the survivors of the 2014–2015 epidemic of Ebola virus disease (EVD) in West Africa were women of childbearing age. Limited clinical and laboratory data exist that describe these women's pregnancies and outcomes. We report the case of an EVD survivor who became pregnant and delivered her child in the United States (US), and we discuss implications of this case for infection control practices in obstetric services. Hospitals in the US must be prepared to care for EVD survivors.

The 2014–2015 epidemic of Ebola virus disease (EVD), which was centered in West Africa, is the largest EVD epidemic in history. Vertical transmission of Ebola virus (EBOV) from mother to fetus can occur during acute Ebola infection, leading to intrauterine fetal death, stillbirth, or neonatal death [1–5]. Little is known about the risk for vertical transmission of EBOV from women to their neonates outside of the acute infectious period. EBOV has been found in breast milk during acute disease [6], and a study documenting two discordant mother–child pairs postulated that breast feeding of one infant may have led to infection of the infant [7]. EBOV has been found in immune-privileged sites, ocular fluid and semen, many months after onset of infection [8–13]. It is possible that other immune-privileged sites such as the central nervous system (CNS) may also contain EBOV many months after onset of infection. In addition, acutely infected pregnant women have had high amounts of Ebola viral nucleic acid persist in the amniotic fluid following clearance of viremia; however, it is not known whether this amniotic fluid is infectious [2]. Some theoretical concern exists that during labor and delivery or obstetric anesthetic procedures (e.g., spinal anesthesia), contact with products of conception or cerebrospinal fluid from EVD survivors may pose an infectious risk [6,14–18].

As of March 9, 2016, an estimated 17,323 persons worldwide have survived EVD, and among them are \approx 5,000 women of childbearing age [19]. Survivors will require medical care for routine illnesses, surgical services, dental work, and management of disease sequelae [20,21]. In addition, many of the female survivors who are of reproductive age will require obstetric care. Some of these survivors may come to the US, and hospitals and healthcare workers must be prepared to provide care in a manner that promotes patient dignity and comfort, prevents stigmatization, and ensures that all patients receive appropriate, high-quality medical care [22–24]. However, limited preparations have been made for follow-up care for EVD survivors, including those needing obstetric care, and minimization of possible stigma and fear. We describe the case of an EVD survivor who delivered a healthy neonate in a community hospital in the US 14 months after acute EBOV infection, and we discuss the implications of the findings from this case for infection control in obstetric services.

² Published as: Kamali A, Jamieson DJ, Kpaduwa J, et al. Pregnancy, Labor, and Delivery after Ebola Virus Disease and Implications for Infection Control in Obstetric Services, United States. *Emerging Infectious Diseases*. 2016;22(7):1156-1161.



CLINICAL COURSE

EVD Course

A 29-year old physician from West Africa became ill with EVD in late July 2014. She had contracted the virus from an EVD patient whom she had cared for from July 20 until his death on July 25. On July 29, the woman began feeling unwell, noting arthralgia and myalgia, which she self-treated with antimalarial medications. On August 1, she had fever, and on August 3, she began vomiting and had diarrhea. The woman was admitted to an Ebola treatment center (ETC) and isolated after results of an EBOV real-time reverse transcription PCR (rRT-PCR) were positive for EBOV RNA (cycle threshold unknown). According to the woman, she spent 13 days in the ETC, where she was treated with oral rehydration fluids, acetaminophen, and a second course of antimalarial medications. She was discharged from the ETC on August 16, after showing negative results on two EBOV rRT-PCRs. After her recovery, the woman noted some fatigue, anorexia, arthralgia, and alopecia; she did not report any sleep disturbances, headaches, or vision problems. Symptoms resolved 2–3 months later.

Pregnancy, Labor, and Delivery

Eight months before her EVD diagnosis, the patient had had a spontaneous abortion at ten weeks gestation. In January 2015, 22 weeks after her last negative EBOV rRT-PCR, she became pregnant again. For this second pregnancy, the estimated date of delivery was established on the basis of an 11.5-week ultrasound that was consistent with the patient's last menstrual period. The patient received routine prenatal care in West Africa. At 25 weeks gestation, she traveled to Kern County, CA, US, and a detailed anatomy ultrasound was performed in Los Angeles County (LAC), CA, which demonstrated normal fetal development.

The hospital identified staff members who were willing to assist during labor and delivery for the patient, and at 40 weeks and one day of gestation, labor was induced to ensure that those staff members were present. The patient was given two vaginal doses of misoprostol, oxytocin was administered, and labor progressed normally. The patient was given epidural anesthesia for pain control and had a normal vaginal delivery of a female neonate (weight 4,128 g) with Apgar scores of 8 and 9 at one and five mins of age, respectively. The patient had a second-degree perineal laceration, which was repaired.

The patient and her neonate were discharged from the hospital at 36 h postpartum. They returned for routine follow-up seven days postpartum and were monitored for six weeks following delivery, after which they traveled home to West Africa.

Infection Control and Personal Protective Equipment, Public Health Response

Two weeks before the patient's delivery date, her US obstetrician contacted the California Department of Public Health (CDPH; Richmond, CA, US) and the Centers for Disease Control and Prevention (CDC; Atlanta, GA, US) to determine if there were any special precautions needed for infection control; the CDPH notified the LAC DPH (Los Angeles, CA, US). Because the patient was healthy and had fully recovered from EVD ≈4 months before becoming pregnant, all public health agencies agreed that she presented an extremely low risk for transmission of EBOV. Nevertheless, it was deemed appropriate that public health officials play an



active role in assessing and guiding management of the patient. The LAC DPH and CDC collaborated with the hospital's healthcare providers, nursing directors, laboratory director, environmental services staff, anesthesiologists, and hospital administration to address concerns and review the care plan, including plans for any complications such as the need for cesarean delivery or the development of peripartum fever.

Hospital infection control procedures were reviewed in person with hospital staff. In review of these policies, no additional precautions were recommended above the standard precautions and policies currently used for all deliveries at the hospital. Several hospital staff members not directly involved in patient care expressed discomfort about working while an EVD survivor was admitted. To reassure these staff members, the patient was kept in one room during labor, delivery, and after delivery. No changes were made to the policies for environmental cleaning or waste disposal.

Hospital staff raised concerns about the possibility of EBOV being harbored in immune-privileged sites (e.g., cerebrospinal fluid) in EVD survivors; thus, they expressed their concerns about a theoretical risk for EBOV transmission [6,14–17]. This patient did not show signs or symptoms of CNS involvement during her acute illness or during her pregnancy, which likely indicated a decreased risk of any latent EBOV reservoir in her CNS. Thus, it was considered likely that epidural or spinal anesthesia for this patient would not pose an infectious risk to staff. Hospital staff also noted the often imperfect adherence to use of personal protective equipment (PPE) during labor and delivery; thus, they voiced concern over this patient's history of EVD because large volumes of blood and amniotic fluid are often encountered in typical, uncomplicated vaginal deliveries [25]. As a result of these concerns, many discussions were held regarding what PPE should be used during labor and delivery. Standard precautions should always be applied in all medical settings, including labor and delivery; however, neither CDC nor the American College of Obstetricians and Gynecologists had tailored recommendations for PPE specifically for vaginal or cesarean deliveries for any patients. Thus, CDC and LAC DPH developed a preliminary set of recommendations for the patient's providers regarding the use of PPE (Table 1 and 2) during and after labor and delivery to ensure that standard precautions were implemented. These PPE recommendations were discussed with the providers in the days before the delivery, and staff members were able to ask for clarification and ensure that materials were readily available. These PPE recommendations did not differ from standard precautions, but they explicitly discussed which PPE to use for casual contact, vaginal examinations, labor and delivery, anesthesia, and postpartum care. Routine hand hygiene, use of barriers for mucous membrane protection, and use of double gloves for procedures that involve sharps were emphasized.

Laboratory Assessment

One week before delivery, EBOV rRT-PCR testing was performed on the patient's blood by the LAC DPH laboratory and the CDC Viral Special Pathogens Branch; both results were negative. As expected, EBOV serum antibodies were detected by ELISA ($IgG \ge 1:1600$, IgM negative).

After obtaining written informed consent from the patient, healthcare staff obtained the following during and after delivery: vaginal secretions, amniotic fluid (vaginal pool), cord blood, placenta, umbilical cord,



breast milk (colostrum collected 16 h after delivery), and oral and ear swab samples from the neonate. Cord blood, colostrum, amniotic fluid, and swab samples were kept refrigerated until processed or frozen on dry ice for shipment to CDC. A placental sample was frozen in a sterile specimen cup, and samples of placenta and umbilical cord were placed in buffered formalin and shipped at room temperature to CDC. EBOV rRT-PCR testing was performed on all of these specimens at the LAC DPH and CDC laboratories by using assays specific for nucleoprotein and 40 viral protein genes.

Placenta, amniotic fluid, and cord blood samples and ear and oral swab samples from the neonate were negative by EBOV rRT-PCR. Attempts were made to recover virus from placenta, amniotic fluid, cord blood, and colostrum at CDC, but no virus was recovered (Table 3). Amniotic fluid, cord blood, and colostrum were tested by ELISA for IgM and IgG against EBOV antigens [26]. Cord blood was negative for IgM and had an IgG titer of \geq 1:1600. Amniotic fluid and colostrum were negative for IgM and IgG. The placenta and umbilical cord were histologically normal, and no EBOV antigen was detected by immunohistochemistry [27], including in maternal and fetal endothelial cells and leukocytes.

CONCLUSIONS

We describe the delivery of a healthy baby to an EVD survivor who became pregnant 22 weeks after clearance of viremia and resolution of post-EVD sequelae (i.e., fatigue, anorexia, arthralgia). At six weeks follow-up, before returning to West Africa, the mother and baby were doing well. Given that the mother did not exhibit any signs or symptoms of post-EVD sequelae during her pregnancy, we did not expect to find any EBOV by rRT-PCR in any specimens obtained, and none was detected. It is somewhat surprising that we did not detect EBOV IgG in the colostrum; however, studies of antibodies for other infections have found that levels of IgG and IgM in colostrum are much lower than those in serum [28], and this might also be true for antibodies against EBOV.

Although we did not detect EBOV RNA in this patient during pregnancy, women who are pregnant during acute EBOV infection usually transmit virus to the fetus and may pose an infectious risk to healthcare providers and others during delivery or abortion [3]. EBOV can readily cross the placenta, and pathologic examination of placental tissues of patients with confirmed EVD have demonstrated EBOV antigen in the trophoblasts, syncytiotrophoblasts, and circulating maternal macrophages [4]. EBOV RNA has been demonstrated in amniotic fluid, fetal meconium, vaginal secretions, umbilical cord, buccal swab samples from neonates, and peripheral blood samples from neonates, including those of mothers with cleared viremia [29,30].

The immune effects of pregnancy in the context of EVD have not been well documented [3]; however, alterations in the immune system do occur during pregnancy [31], which during acute EBOV infection likely increases the risk for a poor outcome including spontaneous abortion and neonatal death. Unlike the CNS, eye, and male testis, the genital tract of a nongravid female is not traditionally considered an immune-privileged site [32–34]. Laboratory data that demonstrate the absence of EBOV or the presence of antibodies in post-EVD pregnancies are lacking; however, on the basis of epidemiological evidence in the field of multiple uneventful deliveries in West Africa and of the laboratory-analyzed case reported



here, no evidence currently exists that EBOV can persist in the female genital tract. Any perceived risk must be mitigated to ensure that patients are not stigmatized and receive appropriate care. The authors concur with current guidelines by the World Health Organization, which state that women who have recovered from EVD are not infectious, should receive routine prenatal care, and their labor and delivery should be performed using standard PPE for protection against blood and bodily fluids [35].

The normal pregnancy for the patient described in this study and her delivery of a healthy neonate offer reassurance that women who become pregnant after recovery from EVD pose little risk for transmission of EBOV to the baby or others. Many more EVD survivors will become pregnant and deliver, and some may do so in the US. Many other survivors will require routine medical care, including care for post-EVD syndrome. Lessons learned from this patient, specifically those addressing concerns about potential risks for virus transmission, may be applied to future patients. However, each survivor who seeks medical care will likely need to be assessed individually to determine possible risks for transmitting virus [16,18]. Over the course of the public health involvement in this case, it became evident that, although standard precautions should routinely be used in all labor and delivery settings, written guidelines for labor and delivery may be useful given the heightened concern for a theoretical disease transmission risk. We hope that the preliminary recommendations for PPE use during labor and delivery in the case discussed here will provide a template for other professional organizations to create guidelines for use in all labor and delivery settings.

REFERENCES

- 1. Akerlund E, Prescott J, Tampellini L. Shedding of Ebola virus in an asymptomatic pregnant woman. N Engl J Med. 2015;372:2467–9.
- 2. Baggi FM, Taybi A, Kurth A, Van Herp M, Di Caro A, Wolfel R, Management of pregnant women infected with Ebola virus in a treatment centre in Guinea, June 2014. Euro Surveill. 2014;19:20983.
- 3. Black BO, Caluwaerts S, Achar J. Ebola viral disease and pregnancy. Obstet Med. 2015;8:108–13.
- Muehlenbachs A, de la Rosa Vazquez O, Bausch DG, Schafer IJ, Paddock CD, Bergeron E, Ebola virus disease in pregnancy: histopathologic and immunohistochemical findings. Presented at: Abstracts of the American Society of Tropical Medicine and Hygiene 64th Annual Meeting; 2015 Oct 25–29; Philadelphia, PA, USA. Abstract LB-5108.
- 5. Mupapa K, Mukundu W, Bwaka MA, Kipasa M, De Roo A, Kuvula K, Ebola hemorrhagic fever and pregnancy. J Infect Dis. 1999;179(Suppl 1):S11–2.
- 6. Bausch DG, Towner JS, Dowell SF, Kaducu F, Lukwiya M, Sanchez A, Assessment of the risk of Ebola virus transmission from bodily fluids and fomites. J Infect Dis. 2007;196(Suppl 2):S142–7.
- 7. Moreau M, Spencer C, Gozalbes JG, Colebunders R, Lefevre A, Gryseels S, Lactating mothers infected with Ebola virus: EBOV RT-PCR of blood only may be insufficient. Euro Surveill. 2015;20:21017.
- 8. Deen GF, Knust B, Broutet N, Sesay FR, Formenty P, Ross C, Ebola RNA persistence in semen of Ebola virus disease survivors—preliminary report. N Engl J Med. Epub 2015 Oct 14.
- 9. Mate SE, Kugelman JR, Nyenswah TG, Ladner JT, Wiley MR, Cordier-Lassalle T, Molecular evidence of sexual transmission of Ebola virus. N Engl J Med. 2015. Epub 2015 Oct 14.



- 10. Varkey JB, Shantha JG, Crozier I, Kraft CS, Lyon GM, Mehta AK, Persistence of Ebola virus in ocular fluid during convalescence. N Engl J Med. 2015;372:2423–7.
- 11. Fischer WA II, Wohl DA. Confronting Ebola as a sexually transmitted infection. Clin Infect Dis. Epub 2016 Mar 1.2016;62:1272–6.
- 12. Howlett P, Brown C, Helderman T, Brooks T, Lisk D, Deen G, Ebola virus disease complicated by lateonset encephalitis and polyarthritis, Sierra Leone. Emerg Infect Dis. 2016;22:150–2.
- Rowe AK, Bertolli J, Khan AS, Mukunu R, Muyembe-Tamfum JJ, Bressler D, Clinical, virologic, and immunologic follow-up of convalescent Ebola hemorrhagic fever patients and their household contacts, Kikwit, Democratic Republic of the Congo. Commission de Lutte contre les Epidémies à Kikwit. J Infect Dis. 1999;179(Suppl 1):S28–35.
- 14. Barkhordarian A, Thames AD, Du AM, Jan AL, Nahcivan M, Nguyen MT, Viral immune surveillance: toward a TH17/TH9 gate to the central nervous system. Bioinformation. 2015;11:47–54.
- 15. Brainard J, Pond K, Hooper L, Edmunds K, Hunter P. Presence and persistence of Ebola or Marburg virus in patients and survivors: a rapid systematic review. PLoS Negl Trop Dis. 2016;10:e0004475.
- 16. Sonnenberg P, Field N. Sexual and mother-to-child transmission of Ebola virus in the postconvalescent period. Clin Infect Dis. 2015;60:974–5.
- Farge E, Giahyue JH. Female survivor may be cause of Ebola flare-up in Liberia. Reuters. 2015 Dec 17. In: Kaye D. 15 March news. Clin Infect Dis. 2016;62:i–ii.
- Centers for Disease Control and Prevention. Interim guidance for management of survivors of Ebola virus disease in US healthcare settings. 2016 [cited 2016 Mar 18].
 www.cdc.gov/vhf/ebola/healthcare-us/evaluating-patients/guidance-for-management-of-survivorsebola.html
- World Health Organization. Ebola data and statistics: situation summary 09, March 2016. 2016 Mar 11 [cited 2016 Mar 18]. <u>http://apps.who.int/gho/data/view.ebola-sitrep.ebola-summary-20160309?lang=en</u>
- 20. Bausch DG. Sequelae after Ebola virus disease: even when it's over it's not over. Lancet Infect Dis. 2015;15:865–6.
- 21. Clark DV, Kibuuka H, Millard M, Wakabi S, Lukwago L, Taylor A, Long-term sequelae after Ebola virus disease in Bundibugyo, Uganda: a retrospective cohort study. Lancet Infect Dis. 2015;15:905–12.
- 22. Kupferschmidt K. Infectious diseases. Surviving Ebola survival. Science. 2015;348:1406–7.
- 23. Minkoff H, Ecker J. Physicians' obligations to patients infected with Ebola: echoes of acquired immune deficiency syndrome. Am J Obstet Gynecol. 2015:212;456.e1–4.
- 24. Sprecher A. Handle survivors with care. N Engl J Med. 2015. Epub 2015 Oct 14.
- 25. Magann EF, Bass JD, Chauhan SP, Young RA, Whitworth NS, Morrison JC. Amniotic fluid volume in normal singleton pregnancies. Obstet Gynecol. 1997;90:524–8.
- 26. Ksiazek TG, West CP, Rollin PE, Jahrling PB, Peters CJ. ELISA for the detection of antibodies to Ebola viruses. J Infect Dis. 1999;179(Suppl 1):S192–8.
- 27. Martines RB, Ng DL, Greer PW, Rollin PE, Zaki SR. Tissue and cellular tropism, pathology and pathogenesis of Ebola and Marburg viruses. J Pathol. 2015;235:153–74.
- 28. Wheeler TT, Hodgkinson AJ, Prosser CG, Davis SR. Immune components of colostrum and milk—a historical perspective. J Mammary Gland Biol Neoplasia. 2007;12:237–47.



- 29. Bower H, Grass JE, Veltus E, Brault A, Campbell S, Basile AJ, Delivery of an Ebola virus–positive stillborn infant in a rural community health center, Sierra Leone, January 2015. Am J Trop Med Hyg. 2016;94:417–9.
- 30. Oduyebo T, Pineda D, Lamin M, Leung A, Corbett C, Jamieson DJ. A pregnant patient with Ebola virus disease. Obstet Gynecol. 2015;126:1273–5.
- 31. Kourtis AP, Read JS, Jamieson DJ. Pregnancy and infection. N Engl J Med. 2014;371:1077.
- 32. Clark GF, Schust DJ. Manifestations of immune tolerance in the human female reproductive tract. Front Immunol. 2013;4:26.
- 33. Mital P, Hinton BT, Dufour JM. The blood–testis and blood–epididymis barriers are more than just their tight junctions. Biol Reprod. 2011;84:851–8.
- 34. Muldoon LL, Alvarez JI, Begley DJ, Boado RJ, Del Zoppo GJ, Doolittle ND, Immunologic privilege in the central nervous system and the blood–brain barrier. J Cereb Blood Flow Metab. 2013;33:13–21.
- 35. World Health Organization. Interim guidance: Ebola virus disease in pregnancy: screening and management of Ebola cases, contacts and survivors. 2015 Sep 4 [cited 2015 Nov 11]. <u>http://apps.who.int/iris/bitstream/10665/184163/1/WHO_EVD_HSE_PED_15.1_eng.pdf?ua=1</u>



Table 1. Recommendations for use of PPE by healthcare workers during labor and delivery for a womanwho became pregnant after surviving EVD, US, 2015* PPE Gown Gloves Fluid-Fluid-resistant resistant, Face Face or midcalf boot Potential exposure mask shield Isolation impermeable[†] Single Double covers Casual contact with patient Performing duties for patient with intact membranes (e.g., delivering food or water, No No No No No No No talking with patient, adjusting external monitors) Performing duties for patient with ruptured No No No No No No No membranes; no touching of patient or bedding Noncasual contact with patient Touching patient with ruptured membranes or No No Yes No Yes No No bedding of patient with ruptured membranes Administering epidural Yes Yes Yes No No Yes Yes‡ Performing vaginal Yes Yes No Yes Yes No Yes‡ examination Performing obstetric No Yes Yes Yes Yes Yes No

*These PPE recommendations were developed for this particular patient and do not represent a formal recommendation. †Impermeable indicates that the material and construction have demonstrated resistance to synthetic blood and simulated bloodborne pathogens; fluid-resistant indicates demonstrated resistance to water (http://www.cdc.gov/niosh/npptl/topics/protectiveclothing/default.html).

‡To be used if membranes were ruptured.

procedures§

§Procedures include placement of fetal scalp electrode or intrauterine pressure catheter; manual removal of placenta; bimanual massage of uterus.



Table 2. Recommendations for use of PPE by healthcare workers during postpartum care of a woman who became pregnant after surviving EVD and during care of her neonate, US, 2015*

	PPE							
				Gloves		Fluid-		
Potential exposure	Face	Face	Isolation	Fluid-resistant or impermeablet	Single	Double	resistant, midcalf boot	
While caring for mother	mask	3111610	1301211011	Impermedbic	Olligie	Double	000013	
Before bedding/gown change	Yes	Yes	No	Yes	Yes	No	Yes	
After bedding/gown change (vaginal exam, perineal care)	No, unless splash likely	No, unless splash likely	Yes	No	Yes	No	No	
While caring for neonate								
Before bathing	Yes	Yes	No	Yes	Yes	No	Yes	
After bathing	No	No	No	No	Yes‡	No	No	

*These PPE recommendations were developed for this particular patient and do not represent a formal recommendation.

†Impermeable indicates that the material and construction have demonstrated resistance to synthetic blood and simulated bloodborne pathogens; fluid-resistant indicates demonstrated resistance to water (http://www.cdc.gov/niosh/npptl/topics/protectiveclothing/default.html).

‡To be used if exposure to fluids is likely.

Table 3. Laboratory test results for a woman who became pregnant after surviving EVD and for her neonate, US, 2015*						
Source	Time of sample collection	rRT-PCR	EBOV antibodies	Immunohistochemistry		
Maternal blood	1 week before delivery	Negative	IgG (1:1,600); IgM not detected	NA		
Cord blood	At delivery	Negative	IgG (1:1,600); IgM not detected	NA		
Amniotic fluid	At delivery	Negative	IgG; IgM not detected	NA		
Vaginal swab sample	At delivery	Negative	NA	NA		
Neonate ear swab sample	At delivery	Negative	NA	NA		
Neonate oral swab sample	At delivery	Negative	NA	NA		
Placenta	At delivery	Negative	NA	Negative for Ebola antigen		
Umbilical cord	At delivery	NA	NA	Negative for Ebola antigen		
Colostrum	1 day after delivery	Negative	IgG; IgM not detected	NA		

*NA, not applicable; rRT-PCR, real-time reverse transcription PCR.





THE UTILITY OF A MOBILE EBOLA ASSESSMENT FOR PERSONS UNDER INVESTIGATION

OVERVIEW

As hospitals in Los Angeles County (LAC) developed capabilities and were designated by the Centers for Disease Control and Prevention (CDC) as Ebola Assessment and Treatment Facilities, the Department of Public Health (DPH) referred people under investigation (PUI) as suspect Ebola cases to those facilities for evaluation. This process required standing-up special units with dedicated, trained staff and strict personal protective equipment and other requirements to prevent potential spread of infection. As a result, evaluating a PUI substantially disrupted hospital operations, critically ill patients needed to be moved, additional staff needed to be called in, and financial costs were substantial. Because of these challenges, hospitals exhibited some reluctance to evaluate patients. At the suggestion of medical staff at one of the Ebola treatment hospitals, LAC DPH initiated a process to explore the feasibility and plan for a mobile assessment of a PUI. Not only would this approach address the challenges associated with a hospital-based evaluation, it also would be less disruptive and faster for the patient while continuing to ensure an appropriate level of care.

ACTIVITIES

LAC DPH began by vetting the concept with the California Department of Public Health (CDPH) and CDC. Because no other jurisdictions had developed plans for mobile assessment, a cross-program, multidisciplinary team met to plan all aspects of the strategy with a primary goal of ensuring appropriate evaluation of the PUI and safety for the evaluation team. Participants in planning included staff from ACDC, Public Health Lab (PHL), Emergency Preparedness and Response Program, Environmental Health, Community Health Services, the Public Information Officer, Emergency Medical Services, fire and police departments, and the Ebola treatment hospital.

OUTCOMES

Ultimately, through the course of our response to the Ebola outbreak that ended in January 2016, this novel approach was used twice to evaluate PUIs during August 2015. An on-scene incident command post and staging area was established at a fire station near the PUIs' residence. The patients were evaluated by infectious disease staff in their home and specimens were obtained, packaged, and taken to the PHL where (negative) results were available within three hours. Mobile assessment proved to be effective, safe, rapid, and prevented the disruption of hospital healthcare services and provided a model for other jurisdictions in future public health emergency responses.

BARRIERS

Challenges to implementing mobile evaluation included discomfort of staff in full personal protective equipment (PPE) in Los Angeles summer heat, risks to privacy from neighbors, and adequacy of patients' homes as settings to conduct the evaluation safely and effectively. Because of the care with which planning was done, prior training of all staff who had participated in previous hospital-based assessments,



good collaboration between LAC DPH and hospital staff, and coordination with LAC emergency response agencies, all these challenges were overcome.



TOWARD AN INDIVIDUALIZED APPROACH TO DEFINE FEVER AMONG TRAVELERS FROM EBOLA-AFFECTED COUNTRIES OR PERSONS WITH EXPOSURE TO AN EBOLA PATIENT

OVERVIEW

Early detection of Ebola virus disease (EVD) is critical to preventing its spread. With the occurrence of EVD cases outside of West Africa, the US screened and monitored travelers from affected countries. Because fever is a key indicator of possible EVD among monitored travelers, high sensitivity in defining fever is critical.

We evaluated two novel methods that defined fever as a temperature increase of $\geq 1^{\circ}$ C (1.8°F) over baseline using data from 45 travelers monitored by the Los Angeles County Department of Public Health (LAC DPH) between October 20 and December 30, 2014. Individual baselines were defined as either the cumulative moving average of all temperatures before the peak measurement or the mean of the first six measurements.

Temperatures measured by travelers ranged from 33.2°C (91.8°F) to 37.3°C (99.1°F). Individuals' mean temperatures ranged from 35.3°C (95.6°F) to 36.9°C (98.4°F). Applying our proposed definitions, each individual's fever threshold would be less than the Center for Disease Control and Prevention's (CDC) reference level of 38.0°C (100.4°F), and for 62% would be less than that of the Dallas nurse who traveled with a temperature of 37.5°C (99.5°F) and later was diagnosed with EVD. While no traveler to Los Angeles County (LAC) developed EVD and sensitivity could not be calculated; nonetheless, a better method for determining a threshold for travelers would be helpful. One monitored traveler who was not diagnosed with EVD had a peak temperature 1.3°C (2.3°F) higher than the mean; thus, the specificity of our fever definition was 97.8%.

A limitation of this analysis is the relatively small number of persons monitored in California and for whom data are available. Analysis of data from other health departments would help refine the specificity estimate. This strategy may be useful not only for EVD but also other infectious conditions where temperature monitoring is done.

Early detection of persons with EVD is critical to preventing the spread of infection. As EVD cases have occurred outside of West Africa, screening and monitoring of travelers from affected countries have been implemented in several countries. In October 2014, US health officials began airport screening of travelers from affected countries. Initial screening includes identifying exposures and defining risk-level, measuring temperature and assessing other symptoms that may be compatible with EVD. Subsequent monitoring by the health department at the traveler's final destination includes twice daily temperature measurements and assessment of other symptoms for a 21-day period during which EVD becomes manifest among the large majority of infected people [1,2].



Fever is a key indicator in the detection of EVD as an early and common symptom among ill persons. Among 1,152 EVD patients in the West Africa outbreak, 87.1% had a measured temperature of >38°C (100.4°F) or a history of fever [2]. Among 103 persons in an earlier Democratic Republic of Congo outbreak, 93% were febrile [3]. The threshold for defining fever among travelers arriving from affected countries and for contacts of EVD patients in the US initially was defined as 38.6°C (101.5°F) but subsequently was lowered to 38.0°C (100.4°F) to increase sensitivity.

The suitability of this definition was questioned, however, when a nurse who cared for a US EVD patient traveled by airplane with a temperature of 37.5°C (99.5°F) and was later diagnosed with EVD [4]. For the CDC and state and local health departments monitoring travelers, fever detection is an important component of monitoring to protect public health and to maintain public confidence.

The widely used definition of 37.0°C (98.6°F) as normal body temperature and 38.0°C (100.4°F) as fever is based on an 1868 study by Wunderlich and Seguin [5]. More recent studies have challenged this definition, finding variation between individuals and systematic differences based on age, gender, time of day, and method of measurement [6-8]. For example, among 148 healthy Baltimore adults ages 18 through 40 years, 700 temperature measurements showed a mean temperature of 36.8°C (98.2°F) and a range from 35.6°C (96.1°F) to 38.2°C (100.8°F); women's temperature was significantly higher than that of men and temperatures in the morning were significantly lower than in the evening [6].

High sensitivity in defining fever is critical for early detection of EVD. An unrecognized case ("false negative" from monitoring) may transmit infection, expose additional persons posing a greater burden for public health agencies, and increase fear of EVD in the community. High specificity also is important given the resources required for diagnosis and the potential disruption of the healthcare system in evaluating a suspected case. Following a report showing low sensitivity of temperature cutoffs of 38.6°C (101.5°F) and 38.0°C (100.4°F) for Ebola among five patients who had serial temperature measurements, we re-evaluated our approach to defining fever among monitored travelers in LAC [9]. Another example that prompted our re-evaluation is the experience from Spain where an infected nurse assistant had "low-grade fever" <38.0°C (100.4°F) for several days before Ebola diagnosis [10].

Whereas using a single fever threshold is necessary when a person is evaluated for infection *de novo*, in a setting where serial measurements are obtained before illness occurs (e.g. where a person is being monitored), healthcare providers have the ability to refine the definition of fever as a difference from the individual's own baseline. In this report, we analyze data from travelers monitored by LAC DPH and the California Department of Public Health (CDPH) to evaluate two potential definitions of fever that may increase the sensitivity of EVD detection while remaining highly specific.



METHODS

During the EVD West Africa outbreak, the CDC informed CDPH of all people from an Ebola-affected countries traveling to the state. CDPH then forwarded traveler contact information to the local health department where the traveler would reside, and that health department monitored the traveler for fever and other Ebola-associated symptoms for 21 days following their last possible exposure, generally their departure from West Africa [11]. Travelers were given a digital oral thermometer on their entry to the US and asked to take their temperature twice daily, in the morning and evening, although specific times were not defined. Measured temperatures and other symptoms were recorded on a diary card and reported in a daily telephone call with the local health department. As a public health surveillance and emergency response activity, informed consent was not required to collect these data from persons being monitored. This study used anonymized data that was maintained in encrypted form and was approved as exempt research by the LAC DPH Institutional Review Board.

At the onset of monitoring for persons in LAC, public health nurses provided education to all adult travelers about how to take oral temperatures. At an initial home visit, travelers were asked to demonstrate taking their temperatures orally. Two children, ages 2 and 3 years, had axillary temperatures measured. Because temperatures from these children were low (with some measurements <34.0°C [93.2°F]) and variable, suggesting difficulty with accurate measurement using this method, they are excluded from the analysis.

Between October 20 and December 31, 2014, 47 travelers were monitored by the LAC DPH (n=38) and by other counties reporting to CDPH (n=9). Data from travelers with at least six temperature measurements are included in this report. For each traveler, we determined the overall mean temperature, the mean temperatures in the morning and evening, and the maximum temperature. We established an individual's baseline temperature in two ways: 1) as the mean of all temperatures before the person's maximum temperature (cumulative moving average, CMA), with a minimum of at least 6 measurements, and 2) as the mean of the first six temperatures recorded (first-6 mean). We calculated the specificity of definitions of fever as 1.0°C (1.8°F) higher than a person's CMA or first-6 mean temperatures. While sensitivity could not be assessed as none of the travelers were diagnosed with EVD, we determined individual and overall mean differences between our definitions of fever and that of the CDC (38.0°C [100.4°F]).

Data were entered into a Microsoft Excel 2010 file and analyzed using Excel and SAS software, version 9.3. Associations of temperature with time of day, age group, gender, and gender-specific age groups were assessed using a student t-test.

RESULTS

Data from 45 travelers who had six or more oral temperature measurements were analyzed. Overall, 1,335 measurements were recorded (mean 29.7 per person). No travelers were identified as having EVD. Ages ranged from 4 to 67 years, and 44 (97.8%) were age 20 years or greater; 66.7% were male. Compliance with measuring temperature was 98.7% (18 of 1,335 potential observations missing).



The temperatures measured and reported by travelers ranged from 33.2°C (91.8°F) to 37.3°C (99.1°F). Individuals' mean temperatures ranged from 35.3°C (95.6°F) to 36.9°C (98.4°F) (Figure 1). The mean and median of the individual mean temperatures were 36.3°C (97.3°F) and 36.4°C (97.5°F), respectively. The morning mean and the evening mean were not different (both 36.3°C [97.3°F]) (Figure 2). Women's mean temperature was higher than that among men (36.5°C [97.7°F] and 36.2°C [97.2°F], respectively, p=0.07). Among adults age 20 to 59 years, women had a significantly higher mean temperature than men (p<0.01). Individuals' maximum temperatures were on average 0.59°C (1.06°F) greater than their mean temperatures. The mean differences between mean and maximum temperatures for women and men were 0.61°C (1.10°F) and 0.51°C (0.92°F), respectively.

Applying a proposed definition of fever as at least 1.0° C (1.8° F) greater than an individual's mean temperature, using the CMA of all temperature measurements before the maximum value, the temperature cutoff for fever would be from 36.7° C (98.1° F) to 37.9° C (100.2° F). Thus, for all travelers, this threshold would be lower than CDC's 38.0° C (100.4° F) reference level. In addition, for 28 (62%) of 45 travelers, the threshold would be lower than the temperature at the time of travel (37.5° C (99.5° F]) of the Dallas nurse who later developed EVD. For one traveler, the maximum temperature was 1.3° C higher than the mean; thus, the specificity of our fever definition was 97.8%. This 52-year old male's reported temperatures ranged from 33.2° C (91.8° F) to 36.8° C (98.2° F) and eight of his 24 measurements were lower than 35.0° C (95.0° F). His mean temperature of 35.3° C (95.6° F) was lower than that of any other traveler.

Using the first six temperature measurements to define a person's baseline temperature yielded very similar results to defining a baseline as the mean of all measurements before their maximum temperature. Of 45 travelers with more than six measurements, for 23 (51%) the means using the two methods were the same, for 18 (40%) were within 0.1° C (0.2° F), for 3 (7%) were within 0.2° C (0.4° F), and for 1 (2%) was within 0.3° C (0.5° F). Where results differed, for 12 persons the first-6 mean was higher, and for 10 it was lower than the CMA. For two travelers, maximum temperatures exceeded the first-6 mean temperature by >1.0°C (1.8° F): one was the same traveler who exceeded the CMA threshold described above, and the other was a traveler whose maximum temperature was 1.0° C (1.8° F) over the first-6 mean baseline and 0.9° C (1.6° F) higher than the CMA baseline.

DISCUSSION

Early identification of EVD among travelers and case-contacts is a public health priority. Given the significance of fever as an early sign of illness and recognizing that people's baseline temperatures may substantially vary; it may be beneficial to explore fever definitions other than the classical single threshold identified almost 150 years ago. Based on the range of mean temperatures we observed, the increase among persons monitored in California required to exceed the CDC 38.0°C (100.4°F) threshold, ranged between 1.1°C (2.0°F) and 2.7°C (4.9°F). Where this increment is smaller, the specificity of this definition may be lower whereas where the difference is greater, the sensitivity would be lower. Fever due to infection occurs with the release of cytokines which act at the hypothalamic thermoregulatory center to elevate the temperature set point [12]. Thus, it is plausible that the temperature of people early in their Ebola illness varies with their baseline temperature and the elevation of their own temperature set point.



The temperature increase with infection has been shown to be less among the elderly [13] making a more sensitive fever threshold particularly important in this group.

To our knowledge, there are limited data on serial temperature measurements in persons early in the course of EVD. A description of the first case acquired in Europe associated with the West Africa epidemic noted "low-grade fever" (temperature <38°C [100.4°F]), which continued for three days, but the specific temperatures were not published [10]. A note about five EVD patients who had serial temperature measurements suggested sensitivities of 79% and 53% for cutoffs of 38.0°C (100.4°F) and 38.6°C (101.5°F), respectively [9]. However, this analysis assessed all temperatures measured during the course of their illness rather than focusing on temperatures at the time of presentation. Reviewing data from the five patients cited shows one of five with temperatures less than 38.0°C (100.4°F) during the first two days of their illness [14-16]. Data from the current EVD outbreak in West Africa may be available to better define the sensitivity of different fever thresholds at the onset of illness.

The performance of our two proposed definitions of fever was similar. For one false positive identified by both methods, the variability in temperature measurements and the frequency of temperatures less than 35.0°C (95.0°F) suggests measurement error. Intervention by a public health nurse reinforcing the proper way to take an oral temperature and elimination of very low measurements from calculating the baseline may increase accuracy. Applying the first-6 mean method would be easier for nursing staff since this value could be calculated after the first three days of monitoring and daily temperatures compared with this value. Because the CMA method requires recalculating the mean after each measurement, the monitoring process would be more complex. With either method, during the first three days before a baseline is established, using a single threshold for all persons monitored would be necessary. Based on our data and experiences from EVD among nurses from Dallas and Spain, an initial 37.5°C (99.5°F) threshold may be reasonable. Importantly, identifying a temperature that exceeds the threshold or identifying other EVD-compatible symptoms only signals the need for more evaluation including a thorough clinical and epidemiological assessment; thus, a "false positive" result for fever would lead to additional evaluation and potentially laboratory testing for Ebola.

A limitation of this analysis is the relatively small number of persons who have been monitored in California and for whom data are available. Further data from travelers we monitor and from those who are monitored by health departments elsewhere can be analyzed to refine the estimate of specificity. Because none of the travelers monitored developed EVD, we cannot quantify the increment in sensitivity of our fever definitions. Necessarily, sensitivity would be similar to or greater than the CDC reference level because each individual's cutoff would be equal to or below 38.0°C (100.4°F). Because we did not observe temperatures being measured and cannot ensure the correct placement of the thermometer, some temperatures may be falsely low, and the mean and range from our population may not be directly comparable with the data from Wunderlich [5] or Mackowiak [6] where temperatures were measured by healthcare personnel. We also did not collect data on the use of antipyretics or assess other factors that may have influenced temperature measurements. Finally, we emphasize that decisions about evaluating



a traveler for EVD should be based on a complete assessment including their exposure history, symptoms, and contextual factors such as ill contacts.

While the focus of this analysis is to develop and test hypotheses that may lead to improved early detection of EVD among travelers from outbreak-affected countries, this approach also may be relevant to other public health settings. It could be used for other emerging infections such as Severe Acute Respiratory Syndrome (SARS) or Middle East Respiratory Syndrome (MERS), for which travelers from specific countries or those who have had defined exposures may be monitored. For hospitalized patients where vital signs are regularly measured, graphing the temperature and identifying increases, which do not exceed an arbitrary cutoff, may trigger further investigation and diagnostic testing, increasing detection of nosocomial infection [17]. Finally, as the current EVD outbreak is likely to continue well into 2015, monitoring and early detection of this illness remain important.

REFERENCES

- CDC. Ebola virus disease: algorithm for evaluation of the returned traveler. http://www.cdc.gov/vhf/ebola/pdf/ebola-algorithm.pdf. (Accessed January 12, 2015)
- 2. WHO Ebola Response Team. Ebola virus disease in West Africa the first 9 months of the epidemic and forward projections. *N Engl J Med* 2014;371:1481-95.
- 3. Bwaka MA, Bonnet M-J, Calain P, et al. Ebola hemorrhagic fever in Kikwit, Democratic Republic of Congo: clinical observations in 103 patients. *J Infect Dis* 1999;179(suppl 1):S1–7.
- Levs J, Yan H. CDC: US health worker with Ebola should not have flown on commercial jet. Cable News Network http://www.cnn.com/2014/10/15/health/texas-ebola-outbreak/. (Accessed January 12, 2015)
- 5. Wunderlich CA, Seguin E. *Medical Thermometry and Human Temperature*. New York, NY: William Wood & Co, 1871.
- 6. Mackowiak PA, Wasserman SS, Levine MM. A critical appraisal of 98.6°F, the upper limit of normal body temperature, and other legacies of Carl Reinhold August Wunderlich. *JAMA* 1992;268:1578-80.
- 7. Adhi M, Hasan R, Norman F, Mahmood SF, Naqvi A, Rizvi A-U-H. Range for normal body temperature in the general population of Pakistan. *J Pak Med Assoc* 2008;58:580-84.
- 8. Lu S-H, Leasure A-R, Dai T-Y. A systematic review of body temperature variations in older people. *J Clin Nursing* 2009;19:4-16.
- 9. Dananche C, Benet T, Vanherns P. Ebola: fever definitions might delay detection in non-epidemic areas. *Lancet* 2014;284:1743.
- 10. Parra JM, Salmeron OJ, Velasco M. The first case of Ebola virus disease acquired outside Africa. N Engl J Med. 2014;371:2439-40.
- 11. CDC. Interim US guidance for monitoring and movement of persons with potential Ebola virus exposure. www.cdc.gov/vhf/ebola/exposure/monitoring-and-movement-of-persons-with-exposure.html (Accessed 5/12/15)
- 12. Dinarello CA. Cytokines as endogenous pyrogens. J Infect Dis 1999;179(suppl 2):S294-304, JID 1999.



- Kelly G. Body temperature variability (Part 1): a review of the history of body temperature and its variability due to site selection, biological rhythms, fitness and aging. *Altern Med Rev* 2006;11:278-93.
- 14. Emond RTD, Evans B, Bowen ETW, Lloyd G. A case of Ebola virus infection. *BMJ* 1977;2:541-44.
- 15. Formenty P, Hatz C, Le Guenno B, et al. Human infection due to Ebola virus, subtype Cote d'Ivoire: clinical and biologic presentation. *J Infect Dis* 1999;179(suppl 1):S48-53.
- Isaacson M, Sureau P, Courteille G, Pattyn SR. Clinical aspects of Ebola virus disease at the Ngaliema Hospital, Kinshasa, Zaire, 1976. In Pattyn SR, ed.: *Ebola Virus Hemorrhagic Fever*. Amsterdam: Elsiever, 1976;22-26.
- 17. Wlodaver CG, Fever notions of the misinformed: a quality improvement project. J Oklahoma State Med Assn 2014;107:21-22.

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Figure 1. Distribution of mean temperatures among 45 travelers from EVD affected countries being monitored by the LAC and CDPH







OUTBREAK OF *SALMONELLA* AT A RESTAURANT IN LOS ANGELES COUNTY: PART OF A MULTI-JURISDICTION OUTBREAK

OVERVIEW

On Thursday, 9/17/15, the Los Angeles County Department of Public Health (LAC DPH) received a foodborne illness report (FBIR) via the web.³ The initial complainant reported 15 out of 18 ill after eating on Friday, 9/11/15. Initial food items reported were salad, zucchini carpaccio, crostini, bread and olive oil, mushroom truffle croquette, risotto, and an apple tart. Symptoms included diarrhea, abdominal cramps, fevers, body aches, and headaches. ACDC initiated an outbreak investigation to determine the extent of the outbreak, risk factors for the disease, and steps needed to prevent further spread.

METHODS

- An outbreak-associated case was defined as a person eating at the FBIR-implicated restaurant between 9/6/15 and 9/13/15 who had:
 - 1) a stool, urine, or blood sample taken which grew Salmonella, or
 - 2) diarrhea and fever, or
 - 3) diarrhea and two of the following symptoms: nausea, fatigue, chills, fever, headache, body aches, or abdominal cramps.

An outbreak-associated control was defined as a person who ate at the restaurant during the same period of time but did not become ill with any gastrointestinal symptoms.

- LAC DPH Environmental Health Services (EHS) contacted the parties on the FBIR complaints to obtain contact information and preliminary information for all members.
- EHS conducted two inspections of the restaurant on 9/17/15 and 9/18/15.
- EHS requested contact information for all reservations made between 9/1/15 and 9/18/15.
- ACDC contacted the individuals who made reservations for case and control finding.
- ACDC created a food history and illness questionnaire for all the complainants from the FBIRs and interviewed them via telephone.
- ACDC collected data in MS Access and calculated frequency and distribution of symptoms among cases. Analyses of food items and combination of food items were also performed. All analyses were conducted using SAS 9.3 analysis software and MS Excel.
- ACDC sent out a health advisory to hospitals requesting to be notified of salmonellosis patients who could potentially be cases of the outbreak.
- ACDC created a separate questionnaire to interview employees on job duties, food history, and possible illnesses prior to the outbreak.
- ACDC, in conjunction with the District Public Health Nurses (PHNs), conducted a site visit on 9/18/15 to interview employees and initiate the process of stool collection.
- ACDC and EHS discussed food preparation with restaurant management and executive chef and obtained recipes with ingredient lists and invoices.

³ www.visualcmr.net/webvcmr/pages/public/pub_FBI_Report.aspx



- PHNs questioned all routinely reported *Salmonella* cases to determine if they had any connection to the LAC restaurant. Any new cases identified by the PHNs were additionally interviewed over the phone by ACDC with the outbreak food and illness history questionnaire.
- Employee stool samples were collected through the restaurant and received by PHNs at their District Health Centers.
- The LAC DPH Public Health Laboratory (PHL) tested all the employee stool specimens and provided results.
- PHL serotyped and determined the pulsed-field gel electrophoresis (PFGE) patterns for all the employee and case isolates.
- ACDC collaborated with Centers for Disease Control and Prevention (CDC) and Food and Drug Administration (FDA) via conference call and email to investigate the multistate outbreak and product trace back.

RESULTS

Setting

On Friday, 9/11/15, a group of employees went to an LAC restaurant for a company luncheon. This restaurant is a dine-in restaurant offering cuisine found in the Riviera and Coastal regions of France, Italy, and Spain. Some food items include ravioli, crostini, salad, lamb chops, risotto, zucchini carpaccio, branzino, truffle mushroom croquettes, and crème brulee. Wine and other alcoholic beverages are additionally available upon order. Patrons typically consume their food at the establishment; however, the restaurant also offers catering services. The restaurant is open seven days a week for lunch and dinner. On Saturday and Sunday, they serve brunch. It is frequented by families and friends who gather to share a meal or to celebrate special events. It is a popular location to hold special events such as weddings, baby showers, birthdays, and work luncheons. Employees are responsible for all the preparation and service of a majority of the food, but some food items come semi-prepared from a commissary in Long Island City, New York.

Among this LAC group, 15 out of 18 people eating at the restaurant reported becoming ill. EHS obtained line lists of the diners and ACDC interviewed luncheon attendees via telephone. Interviews were conducted with 13 of those individuals (87%). During this time, a public health nurse notified ACDC of an employee of the restaurant who tested culture positive for *Salmonella*. Subsequently, all CHS PHNs were notified of a potential outbreak. PHNs soon identified eight additional cases connected to the restaurant. ACDC made contact with all eight cases. In the following week, ACDC received eight more FBIRs identifying individuals who ate at the LAC restaurant and experienced illness between 9/6/15 and 9/13/15. Collectively, food and illness history questionnaires were completed on 81 individuals. Contact information for these individuals were obtained through FBIRs, reservation lists provided by the restaurant, and routinely reported cases PHNs identified as having recently eaten at the restaurant.

Out of the 81 individuals interviewed, 42 cases and 29 controls were identified. The remaining 10 individuals were ill, but did not meet case definition. Out of the 11 laboratory confirmed cases, 10 stool



samples were collected by the private medical facilities the cases visited, and one sample was collected by public health. All isolates of confirmed cases were forwarded to the PHL for serotyping and PFGE testing.

Cases: Restaurant Patrons

The median age of cases was 33 years, ranging from 19-85 years (Table 1). Cases were both male (21%) and female (79%). The controls included males (24%) and females (76%) with a median age of 35 years (range: 23-93 years) (Table 1). Main symptoms of cases included diarrhea (100%), abdominal cramps (98%), nausea (81%), fever (38%), and chills (71%) (Table 2). Illness onsets occurred between 9/6/15 and 9/19/15 (Figure 1). The median incubation period was 30 hours (range: 2 to 139 hours). The median duration was slightly longer than 4 days (range: 1 day to at least 14 days). A total of 11 restaurant patrons had confirmed positive *Salmonella* Enteritidis laboratory cultures with the PFGE pattern JEGX01.0008.

Food Analysis

The results of the statistical analysis of food items eaten by attendees are shown in Table 3. The truffle mushroom croquette (p-value <0.001) was eaten by 86% of cases and the tajine (p-value 0.016) was eaten by 36% of cases. Both food items were found to be significantly associated with illness.

Restaurant

Inspection

All patrons interviewed consumed the food at the restaurant. The inspection by EHS on 9/17/15 revealed violations such as an employee eating while preparing food and the absence of gloves while having contact with food. The restaurant voluntarily closed that day for cleaning. The restaurant disposed of all food items and brought in new food stock. A third party food safety consultant was hired to train staff and provide guidance on food safety matters. Also, a cleaning service company was hired to conduct a deep cleaning of equipment in the kitchen.

EHS also conducted a second site visit the next day, 9/18/15, which included a walk-through of the areas that were cleaned the day before. The restaurant was allowed to hold a special pre-booked event on the evening of 9/18/15. Food and employees for this special event were from a sister location not associated with the outbreak.

Employees

There were 121 employees reported to ACDC. Contact was made with all 121 employees through inperson interviews or self-administration of interview sheets distributed by upper management at the restaurant. Out of 121, 23 employees admitted to gastrointestinal symptoms. Stool samples were collected from any employee that handled food or reported being symptomatic within the last month. The PHL performed the test for results. A total of 14 employees had positive stool cultures for *S*. Enteritidis, with PFGE pattern JEGX01.0008.



ACDC and CHS worked with the restaurant managers to ensure that these 14 employees were either removed from the restaurant until they were cleared by standard procedures or were placed in duties that did not involve food handling.

DISCUSSION

This is a laboratory confirmed *S*. Enteritidis outbreak. The PHL, in conjunction with private labs, yielded a total of 25 positive *Salmonella* tests. Patrons and employees had identical serotypes and PFGE patterns. Patrons who tested positive were from separate dining groups and had eaten at the restaurant at different times or dates. Several cases were identified from routine *Salmonella* surveillance rather than foodborne illness reporting. Presumptive cases also reported severe symptoms such as ongoing diarrhea, fever, headaches, and body aches. Truffle mushroom croquettes and tajine were items found to be significantly associated with illness. Although the tajine resulted as significantly associated, 11 of the 12 individuals who ate tajine also ate the truffle mushroom croquette. In other words, the association of the tajine with illness is confounded by the consumption of the truffle mushroom croquette.

According to the CDC, *Salmonella* results in symptoms of diarrhea, fever, and abdominal cramps. Individuals generally become symptomatic 12 to 72 hours after being infected and remain so for approximately 4-7 days [1]. Children, elderly, immunocompromised, and individuals with severe symptoms may require hospitalization. Certain food items and meats are known to cause Salmonellosis when not properly heated. In particular, *S.* Enteriditis infection is most commonly associated with eggs, but other sources include raw milk, pork, beef, sprouts, and raw almonds [2]. In this outbreak investigation, the items mentioned above were not suspected to be the cause of infection.

The spread of *Salmonella* in this restaurant could have been through a contaminated ingredient used at multiple locations. Produce, for example, can be contaminated at the source before it is shipped through dirty irrigation water, manure, or animal contact. If *Salmonella* is able to contaminate one piece of a larger batch of produce, cross contamination would occur throughout the rest of the batch [3]. This restaurant, and its other locations, use ingredients that are pre-prepared in a commissary and then individually shipped out to each location. Particular to this investigation, black trumpet mushrooms were found to be one of those ingredients that are shipped to the commissary, dried, and then sent to the restaurants. The restaurant then prepares a puree by blending the dried mushrooms with oil, and the puree is used to garnish a few dishes including the truffle mushroom croquettes. Due to the absence of a heat kill step, it is possible the mushrooms, and therefore the puree, were contaminated before they were distributed. The CDC and FDA are involved in an ongoing multistate investigation with this restaurant and its commissary.

The cooking of the truffle mushroom croquettes also introduces a possible pathway for the spread of *Salmonella*. This item is partially prepared in the commissary and then finished in the kitchen of the restaurant. The commissary prepares a frozen truffle mushroom croquette mix that is shipped to each location. At the restaurant, the frozen mix is cut into cubes, dipped in flour and eggs, and fried. If the internal temperature of the croquette does not reach a minimum of 165°F, *Salmonella* may still survive.



Another source of the *Salmonella* could have been an infected food handler at the commissary. Infected individuals can excrete the bacteria in their feces for a few days or several weeks, depending on how quickly their bodies are able to rid the gastrointestinal tract of the illness [3]. *Salmonella* can remain in a person's system even after symptoms have resolved. Food handlers are possible sources of *Salmonella* due to the nature of their work [3-6]. Food handlers at the restaurant were most likely infected themselves when eating the contaminated food and were not the source. Food handlers were likely exposed due to the family style meals eaten on site every day. Also, because there was an outbreak of *Salmonella* with the same PFGE pattern at another location of this restaurant chain, it is more likely a food handler at the commissary would be implicated.

LIMITATIONS

Cases that are found through routine *Salmonella* surveillance occasionally have difficulties recalling when and what they ate. Persons may eat out frequently and the restaurant is one of many exposures. More time has also passed for these cases compared to the individuals who report foodborne illness. As a result, it is also harder to remember the date and time their symptoms first began. These are individuals who have already been diagnosed and may be ascertained several days after resolution of their symptoms.

PREVENTION

EHS educated restaurant owners and managers about sanitization and ways to prevent future *Salmonella* infections. The PHNs and ACDC educated all the restaurant workers and individual salmonellosis cases on the spread of *Salmonella* and the importance of staying home when ill to prevent spreading sickness.

CONCLUSION

This is a single outbreak that occurred among patrons who dined at this restaurant between 9/6/15 and 9/13/15. This outbreak occurred in a specific restaurant location but is part of a larger cluster nationwide. The agent *S*. Enteritidis was confirmed by laboratory results. No additional complaints or illnesses have been reported for this restaurant location since the restaurant has taken appropriate measures to remove all potential causes of this outbreak. ACDC in conjunction with EHS will monitor for future reports of foodborne illness.

REFERENCES

- 1. CDC. General Information on *Salmonella*. www.cdc.gov/*Salmonella*/general/index.html Last Accessed: April 20, 2015.
- CDC. Salmonella serotype Enteriditis. www.cdc.gov/nczved/divisions/dfbmd/diseases/salmonella_enteritidis/ Last Accessed: October 30, 2015.
- 3. Heymann, David et al. *Control of Communicable Disease*. Salmonellosis. Baltimore: United Book Press, Inc.; 2008.
- 4. Fatica, M, Schneider, K. *Salmonella* and produce: Survival in the plan environment and implication in food safety. Virulence. 2011 Nov/Dec 2:6, 573-579.



- 5. Greig JD, Todd EC, Bartleson CA et al. Outbreaks where food workers have been implicated in the spread of foodborne disease. Part 1. Description of the problem, methods, and agents involved. J Food Prot. 2007 July; 70(7):1752-61.
- 6. Hedican E, Hooker C, Jenkin T, et al. Restaurant *Salmonella* Enteritidis outbreak associated with an asymptomatic infected food worker. J Food Prot. 2009 Nov;72(11):2332-6.





Table 1. Patron Der	nogra	ohics			Table 2. Symptoms	(n=42)	
	Ca (n=	ses =42)	Co (ontrols n=29)			
	Ν	(%)	Ν	(%)	Symptom	Ν	%
Male	9	(21%)	7	(24%)	Fatigue	32	76%
Female	33	(79%)	22	(76%)	Nausea	34	81%
					Diarrhea	42	100%
Age Group (years)					Bloody Diarrhea	5	12%
<1	0	(0%)	0	(0%)	Body Aches	25	60%
1-4	0	(0%)	0	(0%)	Abdominal cramps	41	98%
5-9	0	(0%)	0	(0%)	Dizziness	18	43%
10-19	1	(2%)	0	(0%)	Chills	30	71%
20-49	31	(74%)	22	(76%)	Vomiting	12	29%
50-74	9	(21%)	3	(10%)	Headache	22	69%
>74	1	(2%)	1	(3%)	Fever	16	38%
Unknown	0	(0%)	3	(10%)	Fever > 102°F	0	0%
					Tingling	2	5%
Median age	33	3 years	3	5 years			
Age range	19-85	5 years	23-9	3 years	Median Duration=1.7	days (rar	nge 1-5 days)
					Median Incubation=3	4 hours (i	range 2-51 hours)



Table 3. Food Items Eaten

	Cases (N=42)		Controls (N=29)				
Food Item	Percent	n	Ν	Percent	n	N	p-value
Salad	17%	7	42	38%	11	29	0.043
Fig & Gorgonzola Risotto	10%	4	42	7%	2	29	0.696
Crostini	67%	28	42	69%	20	29	0.839
Truffle Mushroom Croquette	86%	36	42	17%	5	29	<0.001
Filet Mignon Salad	12%	5	42	14%	4	29	0.814
Caramelized Apple Tart	21%	9	42	10%	3	29	0.221
Zucchini Carpaccio	17%	7	42	10%	3	29	0.452
Lamb	21%	9	42	10%	3	29	0.221
Beef Carpaccio	2%	1	42	3%	1	29	0.789
Tajine	36%	15	42	10%	3	29	0.016
Bread	36%	15	42	38%	11	29	0.849
Olive Oil	38%	16	42	38%	11	29	0.989
Water	60%	25	42	90%	26	29	0.008
Ice	26%	11	42	38%	11	29	0.293
Truffle Risotto	2%	1	42	3%	1	29	0.789
Pot de Cream	10%	4	42	14%	4	29	0.576
Tartufo	7%	3	42	0%	0	29	0.141
Salmon	19%	8	42	10%	3	29	0.319
Paella	7%	3	42	7%	2	29	0.968
Buratta	5%	2	42	3%	1	29	0.787
Octopus	7%	3	42	3%	1	29	0.507
Sea bass	5%	2	42	3%	1	29	0.787



A CONFIRMED NOROVIRUS OUTBREAK ASSOCIATED WITH OYSTERS

OVERVIEW

From Friday, 2/27/15, to Tuesday, 3/3/15, the Los Angeles County Department of Public Health (LAC DPH) received three separate foodborne illness reports (FBIRs) via the web⁴ all describing illness after at the same restaurant. All three groups reported eating the buffet that includes a variety of raw seafood, sushi rolls, and side dishes. Gastrointestinal (GI) illness symptoms included vomiting, diarrhea, stomach cramps, and nausea. The ACDC initiated an outbreak investigation to determine the extent of the outbreak, risk factors for the disease, and steps needed to prevent further spread.

METHODS

- An outbreak-associated case was defined as a person eating at the restaurant between 2/22/15 and 3/1/15 who had:
 - a) a positive lab result of norovirus, or
 - b) diarrhea and vomiting, or
 - c) diarrhea or vomiting plus two or more additional GI symptoms including dizziness, nausea, abdominal cramps, fatigue, headaches, body aches, chills, and fever.

An outbreak-associated control was defined as a person who ate at the restaurant during the same period of time but did not become ill with any GI symptoms.

- LAC DPH Environmental Health Services (EHS) contacted the parties on the FBIR complaints to obtain contact information for all attendees.
- EHS conducted three inspections of the restaurant on 2/27/15, 3/4/15, and 3/6/15.
- EHS requested contact information for complaints of illness made directly to the restaurant between 2/22/15 and 3/1/15.
- ACDC created food history and illness questionnaires for all FBIR and restaurant complainants.
- ACDC called all members of the parties on the FBIRs and interviewed them via telephone. ACDC also called those who complained directly to the restaurant and interviewed patrons either over the phone or via a fillable questionnaire.
- ACDC interviewed and collected stool samples from all the restaurant employees.
- Oysters were tested for norovirus by the Gulf Coast Seafood Laboratory, Dauphin Island, Alabama.
- ACDC collected data in MS Access and calculated the frequency and distribution of symptoms among cases. An analysis of food items consumed was also performed. All analyses were conducted using SAS 9.3 analysis software and MS Excel.
- The Public Health Laboratory (PHL) performed laboratory tests for all the employees and patrons who submitted stool samples, checking for norovirus, *Salmonella*, and *Shigella*.

⁴ www.visualcmr.net/webvcmr/pages/public/pub_FBI_Report.aspx



RESULTS

Different groups (Groups A-D) were established to differentiate complainants based on food that was consumed and method of reporting illnesses. On Tuesday, 2/24/15, Group A gathered for a family dinner at a Los Angeles County (LAC) restaurant. This restaurant is an all you can eat buffet that includes large selections of seafood and sushi. Patrons order selected items from the menu. They then are served their chosen items and are allowed to order more food at no additional cost. Some reported food items included oysters, yellowtail, salmon, halibut, tempura, noodles, edamame, and scallops. EHS obtained a line list, and ACDC interviewed all 13 members of the group (100%) via telephone for their food and illness histories. Six cases and six controls were identified. One ill individual did not meet the case definition.

Group B dined that Saturday, 2/28/2015, and consumed items such as salmon rolls, oysters, tuna, and sashimi salad. This group comprised of friends from separate households. EHS obtained a line list, and ACDC called all the attendees. Interviews were completed for six out of ten (60%) individuals. All six interviewees met case definition. Stool samples were additionally collected from three of the patrons. Multiple attempts were made to contact the four non-respondents.

A member of Group C had reported food poisoning on Yelp.⁵ ACDC contacted this individual encouraging a report to the LAC DPH. This complainant complied but chose not to cooperate further with the investigation. As a result, contact information was not provided for this party. Six out of six individuals eating with this party on Saturday evening, 2/28/15, were reported ill. Food items reported were oysters and sushi. These individuals were not included in the analysis because they could not be interviewed.

Other patrons had reported their illnesses directly to the restaurant (Group D). EHS obtained a list of names and phone numbers from the restaurant operator, and ACDC called every person on the list. Phone interviews were completed for those with valid phone numbers. These individuals were then asked to forward an electronic copy of the questionnaire to their eating companions. This method was employed due to unwillingness of patrons to give additional contact information. Nine interviews were completed from this group, and one stool sample was collected. The percentage of interviews completed cannot be calculated because the denominator for many parties were unknown. General food items reported were oysters, fish, and sushi rolls. All nine people were identified as cases.

Cases: Restaurant Patrons

There were 21 individuals who met case definition. The median age of cases was 29 years, ranging from 24 to 65 years (Table 1). Cases were both male (43%) and female (57%). The controls also included males (50%) and females (50%) with a median age of 9 years (range 2-84 years). Symptoms of cases included diarrhea (81%), nausea (86%), abdominal cramps (76%), fatigue (90%), body aches (76%), chills (67%), vomiting (67%), and other gastrointestinal symptoms (Table 2). Illness onsets occurred between 2/24/15 and 3/2/15 (Figure 1). The median incubation period was 34 hours (range 1.5–51 hours). The median duration was 1.7 days (range 1–5 days). All four stool samples submitted by cases tested positive for the norovirus strains GI (one case) and GII (three cases). Samples were collected on 3/5/2015 and 3/6/2015,

⁵ www.yelp.com/la



which were two and three days after onset date (onset date for all four tested cases: 3/2/15). None of the cases tested positive for *Salmonella* or *Shigella*.

Food Analysis

The results of the analysis of food items eaten by the patrons are shown in Table 3. Food analysis was combined for all the groups because several food items were shared across parties. Additionally, since only Group A had controls, these controls could also be compared to the cases from the other groups (Groups B-D). Several food items calculated as significantly associated with illness. These included oysters, salmon, yellowtail, halibut, sea urchin, scallop, tuna, lobster roll, and water. The most significant food items were raw oysters and raw salmon. Raw oysters were consumed by all 21 cases (100%) and 0 controls (0%), and raw salmon was consumed by 20 cases (95%) and 0 controls (0%).

Restaurant A

Inspection

Restaurant A is a casual dining restaurant open seven days a week for lunch and dinner. It is a popular spot for family and friends to gather for a relatively inexpensive seafood meal. Items were consumed at the establishment. Patrons also are not able to bring leftover food out of the restaurant. The inspection by EHS on 2/27/15 revealed minor violations such as dirty equipment, improper food storage, and incorrect placement of cleaning chemicals. Two critical violations were noted. These included holding potentially hazardous food at unapproved temperatures and allowing employees to eat and drink in the food preparation area. An office hearing was also scheduled to discuss a plan for correction of violations. The oysters were voluntarily held from service and invoices were obtained for the oysters. At the inspection on 3/4/15, the restaurant voluntarily closed to do a thorough cleaning and sanitation of the restaurant. The remaining box of oysters was red tagged, and the oysters were collected for testing on 3/10/15. The restaurant met the necessary requirements to reopen on 3/6/15.

Food testing

Oysters from the suspect lot were obtained from the restaurant and submitted to the LAC PHL for norovirus testing. These were imported oysters from Korea and were shipped frozen. The PHL sent the oysters to the California Viral and Rickettsial Diseases Laboratory where a new test method was unable to detect norovirus. The oysters were then submitted to the Food and Drug Administration (FDA) Gulf Coast Seafood Laboratory where testing resulted in detection of norovirus. Two strains of norovirus, GI and GII, were found in the oysters. These are the same strains of norovirus found in the employees and patrons who tested positive for norovirus. The date and time the two employees actually became ill could not be confirmed.

Employees

There were 31 employees reported to ACDC, and ACDC made contact with all 31 employees (100%). One employee admitted to GI symptoms on 2/27/15. All other employees denied symptoms of GI illnesses in themselves and members of their household during the month preceding the outbreak. Stool samples were collected from the entire staff of 31 employees (100%). The PHL performed the laboratory tests for



all the employees. The one employee who reported illness tested negative for norovirus, *Shigella*, and *Salmonella*. For the remaining employees, two tested positive for norovirus and one for *Salmonella*. No employee tested positive for *Shigella*. ACDC and Community Health Services took the appropriate steps to temporarily remove these employees from work until they were cleared by standard procedures. All other workers yielded negative test results for norovirus, *Salmonella*, and *Shigella*.

DISCUSSION

This outbreak is consistent with an etiology of norovirus infection and was confirmed by laboratory testing. Six individuals (four patrons and two employees) tested positive for norovirus. The one employee positive for *Salmonella* was a server who did not touch food or raw fruits and vegetables. There is no evidence that this individual infected any patrons or other employees. While multiple food items were significantly associated with illness, statistical and laboratory evidence implicated the oysters. All ill individuals reported consumption of oysters, while individuals who were not sick did not eat oysters. The restaurant had also recently purchased oysters from a different distributor and started serving those oysters around the same time cases ate at the restaurant. There were no reports of illness from patrons who ate oysters prior to the switch of distributors.

The Centers for Disease Control and Prevention (CDC) cites that "norovirus outbreaks can occur from foods, such as oysters, fruits, and vegetables, which are contaminated at their source." Norovirus survives at cold temperatures and can easily be transmitted to humans via consumption of high risk foods not properly heated. People infected with norovirus can spread it through their feces and vomit when preparing food or contaminating common surfaces such as door knobs, tables, and restroom sinks. Having contact with a sick individual is another way to pick up the virus. It is highly contagious and can be transmitted even when symptoms are not present [1]. The incubation period for norovirus-associated gastroenteritis is usually between 24 and 48 hours with symptoms such as nausea, diarrhea, vomiting, and abdominal pain lasting 24-72 hours. It most commonly manifests itself from November to April but occurs year round.

LIMITATIONS

The food analysis is limited by the small number of controls included in the analysis. Having few cases and even fewer controls reduces statistical power. Having the responses of more controls would increase the chances of finding a statistically significant association between food items and illness.

PREVENTION

Wholesale Food and Safety, an EHS program, educated restaurant owners and managers about sanitization and ways to prevent future norovirus infections. Some recommendations included following guidelines for hand-washing, maintaining clean surfaces where patrons and employees frequent, and monitoring workers to ensure they are not handling food for at least 48 hours after symptoms have subsided [2]. Employees were educated on the importance of staying home from work when feeling ill. The restaurant was taught about the relationship between raw foods and norovirus as well as other



reservoirs of this virus that could be found in restaurants. ACDC additionally provided education to the restaurant managers and employees during a site visit.

CONCLUSION

This is a single outbreak that occurred among patrons who dined at Restaurant A between 2/22/15 and 3/1/15. The agent was laboratory confirmed as norovirus. The likely source of the outbreak was the frozen imported oysters, which were found to have two strains of norovirus. These two strains were also found in the patrons. No additional complaints or illnesses have been reported following this investigation. ACDC in conjunction with EHS will monitor for future reports of foodborne illness from Restaurant A.

REFERENCES

- 1. Centers for Disease Control and Prevention. Norovirus Overview. Website: www.cdc.gov/norovirus/about/overview.html. Last Accessed: April 1, 2015.
- 2. Heymann, David, et al., Control of Communicable Disease. Baltimore: United Book Press, Inc.; 2008.

Table 1. Patron Demographics						
	Cases (n=21)	Controls (n=6)				
	N (%)	N (%)				
Male	9 (43%)	3 (50%)				
Female	12 (57%)	3 (50%)				
Age Group (years)						
<1	0 (0%)	0 (0%)				
1-4	0 (0%)	3 (50%)				
5-9	0 (0%)	0 (0%)				
10-19	0 (0%)	1 (17%)				
20-49	15 (71%)	0 (0%)				
50-74	6 (29%)	1 (17%)				
>74	0 (0%)	1 (17%)				
Median age	29 years	9 years				
Age range	24-65 years	2-84 years				

Table 2. Symptoms (n=21)					
Symptom	Ν	%			
Fatigue	19	90%			
Nausea	18	86%			
Diarrhea	17	81%			
Bloody Diarrhea	1	5%			
Body Aches	16	76%			
Abdominal Cramps	16	76%			
Dizziness	15	71%			
Chills	14	67%			
Vomiting	14	67%			
Headache	13	62%			
Fever	5	24%			
Fever > 101°F	3	14%			
Median Duration=1.7 days (range 1-5 days)					
Median Incubation=34 hours (range 1.5 -51 hours)					




Table 3. Food Items Eate	n								
	Ca	ses (I	N=21)		Cor	Controls (N=6)			
Food Item	Percent	n	unk*	Ν	Percent	n	unk*	Ν	p-value
oysters	100%	21		21	0%	0		6	<0.0001
salmon	95%	20		21	0%	0		6	<0.0001
yellowtail	76%	16		21	0%	0		6	0.0008
halibut	48%	10		21	0%	0		6	0.0332
sea urchin	57%	12		21	0%	0		6	0.0130
scallop	43%	9		21	0%	0		6	0.0495
tuna	57%	12		21	0%	0		6	0.0130
sashimi salad	24%	5		21	0%	0		6	0.1855
avocado crab roll	52%	11		21	17%	1		6	0.1205
lobster roll	57%	12		21	0%	0		6	0.0130
spider roll	33%	7		21	0%	0		6	0.1003
California roll	48%	10		21	50%	3		6	0.918
eel and avocado roll	33%	7		21	0%	0		6	0.1003
caterpillar roll	5%	1	1	21	0%	0		6	0.7345
shrimp tempura	33%	7		21	50%	3		6	0.4559
veggie tempura	33%	7		21	33%	2		6	1.0000
octopus	10%	2		21	0%	0		6	0.4321
tofu	24%	5		21	50%	3		6	0.2153
seaweed	86%	18		21	67%	4		6	0.2895
rice	90%	19		21	67%	4		6	0.1477
udon	10%	2		21	100%	6		6	<0.0001
mussels	14%	3		21	33%	2		6	0.2895
eggs	10%	2		21	33%	2		6	0.1477
edamame	67%	14		21	50%	3		6	0.4559
mochi ice cream	38%	8		21	83%	5		6	0.0505
green tea ice cream	33%	7		21	0%	0		6	0.1003
water	90%	19		21	50%	3		6	0.0244
ice	76%	16		21	50%	3		6	0.2153
soda	10%	2		21	0%	0		6	0.4321
tea	38%	8		21	33%	2		6	0.8313
*unk=unknown: Number of respondents who cannot recall whether they consumed the food item. This number is subtracted from the denominator to calculate percent.									





CARBAPENEM-RESISTANT ENTEROBACTERIACEAE INFECTIONS ASSOCIATED WITH ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY PROCEDURES LOS ANGELES COUNTY, 2015

OVERVIEW

Carbapenem-resistant Enterobacteriaceae (CRE) infections associated with endoscopic retrograde cholangiopancreatography (ERCP) procedures have been reported in the literature, and several outbreaks have been investigated. Previous reports have identified breaches in cleaning protocols, including bacterial contamination of difficult to clean areas [1]. Other investigations report finding no breach in cleaning and reprocessing protocols or defects in the implicated scopes [2]. The scope's design has been implicated as a source of potential contamination due to the complexity of the elevator channel and the difficulty in ensuring adequate cleaning and disinfection [3].

In 2015, the Los Angeles County Department of Public Health (LAC DPH) Acute Communicable Disease Control (ACDC) program investigated three outbreaks of ERCP associated multidrug resistant organism (MDRO) infections at three separate hospitals. Each hospital performs a high volume of ERCP procedures, serves as referral centers for other hospitals, and often sees medically complex, high-risk patients.

SUMMARY OF INVESTIGATIONS

Hospital A

In January 2015, the hospital infection preventionist (IP) notified ACDC of a cluster of patients who were carbapenem-resistant *Klebsiella pneumoniae* (CRKP) culture positive after undergoing an ERCP procedure. In mid-December 2014, an infectious disease physician alerted Infection Prevention and Control (IPC) to an unusual case of CRKP bacteremia in a patient shortly after undergoing ERCP. An investigation was initiated by the IPC, who requested a list of all 2014 CRE isolates identified by the laboratory. The laboratory identified 33 CRE positive patients in 2014, of which 23 were CRKP. Hospital A staff conducted a comprehensive investigation including extensive chart review of each case to identify potential risk factors, room locations, and IPC direct observation of duodenoscope reprocessing. The microbiology laboratory did further molecular testing on a subset of the CRKP isolates to determine relatedness. Molecular results were reviewed by IPC and further investigation was performed to determine the point source. Multiplex real-time PCR assay (rtPCR), which was used to detect carbapenemases, was negative for several CRKP.

A total of 15 patients met the case definition. A case was defined as a patient who was CRKP culture positive, infected or colonized at any site, who had an ERCP procedure between October 2014 and January 2015. Of these cases, three died during their hospitalization.

Initially, eight patients met the case definition, with clinical culture positive sites including blood (n=4), and abdominal sources including aspirate, drainage, or abscess (n=4). Seven isolates had identical sensitivity patterns and were resistant to carbapenems, aminoglycosides, penicillins, cephalosporins, and



fluoroquinolones and susceptible to colistin. One multiplex negative CRKP isolate underwent whole genome sequencing which identified the OXA-232 carbapenemase. Additional molecular testing by repetitive sequence-based polymerase chain reaction (repPCR) and high resolution melt analysis (HRM) was conducted by Hospital A's laboratory on CRE isolates from 17 patients in 2014 to determine relatedness. The unique carbapenemase OXA-232 strain was identified in CRKP isolates from ERCP-related patients (n=8). RepPCR and HRM results showed OXA-232 strains from all cases to be almost identical. When focusing on the strains that were highly related to each other, the only commonality between patients was ERCP during their hospitalization.

An index patient who was CRKP positive prior to their ERCP procedures in October 2014 was identified. This patient underwent multiple procedures with two duodenoscopes (duodenoscope 1 and duodenoscope 2). A total of 14 patients had subsequent ERCP exposure with duodenoscope 1; three additional patients had subsequent exposure to duodenoscope 2. There were no other common exposures identified among OXA-232 positive patients.

Once ERCP with duodenoscopes 1 and 2 was established as a risk factor for transmission of CRKP, patient notification was initiated by Hospital A. One hundred eighty-six (186) patients had ERCP with the implicated duodenoscopes between October 2014 and January 2015. Notification included phone calls and mailed letters informing of possible CRE exposure and offers to screen for CRE by rectal swab of all patients notified; 150 patients were screened, seven (5%) were positive for CRKP. Isolates from the surveillance cases were also identified as OXA-232.

Hospital A implemented many control measures including ceasing all ERCP procedures during the investigation, sequestering the two implicated duodenoscopes (1 and 2), assessing duodenoscope cleaning and disinfection process, culturing all seven adult duodenoscopes, reprocessing following manufacturer's guidelines, and sending duodenoscopes to a private company for additional ethylene oxide (EtO) gas sterilization. A Manufacturer and User Facility Device Experience report was submitted by Hospital A to the U.S. Food and Drug Administration (FDA). All seven duodenoscopes were cultured and all were negative for CRE.

A site visit was conducted by ACDC staff in February 2015, five days after the outbreak was reported. During this visit, duodenoscope cleaning and high level disinfection procedures were observed. Reprocessing was done by GI reprocessing technicians or GI registered nurses (RNs), both trained in reprocessing. Pre-cleaning was performed immediately after the procedure in the procedure room. The facility used an automated endoscope reprocessor. No breaches in technique to prevent infections were observed. Duodenoscopes were stored appropriately according to manufacturer instructions. Several consultations with the California Department of Public Health (CDPH), Centers for Disease Control and Prevention (CDC), and the FDA were conducted.



In late February 2015, ACDC sent an email to all acute care hospital IPs encouraging active surveillance for CRE infections following ERCP procedures, including a retrospective review. Additional clusters were identified and reported to LAC DPH.

Hospital B

In February 2015, the director of IPC at Hospital B notified ACDC of four patients with CRKP infections since September 2014 following ERCP in their facility. In response to recent media attention surrounding the investigation at Hospital A, Hospital B initiated a review of CRE infections following ERCP in their facility and identified the four patients. A case was defined as a patient who was CRKP positive from any site after ERCP at Hospital B. IPC conducted a comprehensive review of patient medical records, ERCP procedures and microbiology review for other CRKP positive patients who may have undergone ERCP.

Five patients met the case definition. All cases underwent at least one ERCP procedure prior to their positive culture; three cases underwent two or more procedures prior to their positive culture. Four cases were CRKP culture positive in clinical specimens including blood (n=2) and bile (n=2); the fifth case was positive in a surveillance rectal swab tested after patient notification was initiated; two cases died.

IPC identified one duodenoscope as having been used by all cases prior to their positive culture. This duodenoscope was used frequently as it was preferred by the gastroenterologist who performed the larger volume of procedures at Hospital B. Reprocessing of the duodenoscopes was performed using an automated endoscope reprocessor.

Isolates for four cases were available for testing, including the case identified through surveillance. RepPCR performed by an outside laboratory identified two cases to be greater than 98% similar and 95% similarity among all four case isolates tested. Pulsed-field gel electrophoresis (PFGE) analysis performed at the LAC DPH Public Health Laboratory (PHL) on the initial three isolates available indicated that two cases were genetically indistinguishable. Isolates from all three cases were identified as genetically related.

Multiple control measures were implemented by the facility, including removing the implicated duodenoscope from use, postponing all elective ERCP procedures, and culturing of all duodenoscopes. Hospital B duodenoscopes were cultured twice using the CDC Interim Sampling Method for the Duodenoscope – Distal End and Biopsy Channel. The 10 scopes cultured were negative for CRE; all but two grew other organisms, including *Bacillus spp.* and coagulase negative Staph. In addition to culturing, Hospital B sequestered duodenoscopes for 48 hours after culture to ensure all samples were negative prior to further use, with the exception of urgent or emergent cases. Additional duodenoscopes were ordered to accommodate the 48 hour wait period after culture, and elective ERCPs resumed two weeks later. Apart from the use of the implicated duodenoscope in their ERCP procedures, no other common suspected source of infection was identified among the five cases.



IPC initiated patient notification for ERCP patients who were exposed to the implicated duodenoscope from August 2014 to February 2015. Notification letters were mailed to patients and included an FAQ on CRE and duodenoscopes as well as the number to a hotline that was established specifically for patients who were notified to call in with questions. Of the 67 patients notified, 34 (51%) requested rectal swab kits, and one patient tested CRKP positive.

ACDC conducted a site visit on February 2015, four days after notification by Hospital B, and observed the method used to reprocess duodenoscopes. No breaches in practices to prevent the spread of infections were noted. We reviewed infection control practices, scope reprocessing manuals, technician training and competency materials, and related policies and procedures. Several consultations with CDPH, CDC, and the FDA were conducted.

Hospital C

In August 2015, ACDC was notified by IPC at Hospital C of three patients who became ill and were multidrug resistant *Pseudomonas aeruginosa* (MDR-PA) culture positive in July 2015 following ERCP procedures in the facility. Hospital C initiated an ERCP surveillance program in May 2015 in response to two ERCP related MDRO outbreaks in other LAC facilities and identified three patients with blood cultures positive for MDR-PA after ERCP. ACDC notified the appropriate local health jurisdiction (LHJ) who led the investigation, with ACDC participating in a consultative role.

A case was defined as a patient who had received an ERCP procedure, inpatient or outpatient, at Hospital C between January 2013 and August 2015 who presented with a positive MDR-PA culture from any site within 90 days of ERCP. A comprehensive investigation was initiated by IPC staff, ACDC, and the LHJ, including review of ERCP procedure logs, medical records, administrative records, microbiology and culture results from patients, duodenoscopes, and environmental samples.

Sixteen patients met the case definition; eleven cases died. All cases had ERCP procedures performed between January 2013 and August 2015 with one or more of the three duodenoscopes linked to the outbreak. All cases were MDR-PA culture positive from at least one body site, including wound (n=4), blood (n=9), and other sites (4). Isolates were sent to the LAC DPH PHL for PFGE testing. Duodenoscopes were sent to CDC Environmental and Applied Microbiology Laboratory for testing.

A total of 41 MDR-PA isolates from 29 patients, three duodenoscopes, and one environmental site were sent for strain testing by PFGE at the LAC DPH PHL. Test results showed 16 case isolates and 8 duodenoscope isolates from three different scopes were identified as indistinguishable or closely related. One distinct MDR-PA strain was identified by molecular epidemiology. No commonalities other than ERCP procedure were identified among the 16 cases. Per the LHJ request, Hospital C sent the three epidemiologically linked duodenoscopes to the CDC Environmental and Applied Microbiology Laboratory for testing. Using the CDC Interim Duodenoscope Surveillance Protocol as well as more aggressive sampling techniques and sonication, many types of bacteria were identified, including *Pseudomonas*



aeruginosa, Klebsiella pneumoniae, Citrobacter freundii, and others. Sampled sites that demonstrated growth included the instrument channel, distal tip, and elevator.

Control measures recommended by ACDC and LHJ included removing the three epidemiologically linked duodenoscopes from service, double high-level disinfection, repairing and maintaining the scope storage room, monitoring and recording temperature and humidity, ceasing use of canned compressed air during drying, and discontinuing use of plastic scope covers during storage. Hospital C initiated periodic culturing of scopes in July 2015 in response to the outbreaks at Hospital A and B. During the outbreak, the recommendation was made to culture each scope after reprocessing. Once control measures were implemented, no further transmission was identified.

Patient notification was initiated at the recommendation of ACDC and LHJ. Eighty-eight patients who received an ERCP procedure with any duodenoscope from January 2015 to August 2015 were notified and offered testing. Fifteen patients requested testing, and none were positive for *Pseudomonas aeruginosa*. In addition, ACDC and the LHJ recommended Hospital C obtain consent for future ERCP procedures, inpatient and outpatient, including a verbal and written detailed review of the risks of infection and notification of the outbreak.

A site visit was conducted in August 2015, one day after notification by Hospital C, by ACDC, LHJ, and CDPH Licensing and Certification staff. Clinical, surveillance, and microbiology data was reviewed with Hospital C staff. Staff also observed duodenoscope reprocessing and storage. Immediate recommendations were made for control measures and patient notification. A second visit was made in mid-September 2015 to observe implementation of initial recommendations regarding storage and reprocessing procedures as well as to obtain environmental cultures. Several consultations with CDPH, CDC, and FDA were conducted.

CONCLUSION

The epidemiology and lab analyses of these investigations suggest that the cause of these outbreaks is multifactorial, including that the complex design of the scope may impede effective cleaning, disinfection and reprocessing. In January 2016, the duodenoscope manufacturer initiated a recall of one scope model for replacement of the elevator mechanism [4]. In addition, several nationally recognized experts have recommended several options to enhanced reprocessing, including double high-level disinfection with periodic culturing of a sample of scopes and use of ethylene oxide sterilization after high-level disinfection [5]. The CDC, FDA, and CDPH provided guidance to hospitals and providers on duodenoscope reprocessing after ERCP. Professional associations that provide infection prevention and related information, e.g. the Association for Professionals in Infection Control (APIC) and the Society for Healthcare Epidemiology of America (SHEA) also provided reprocessing guidance.

Partnerships between hospitals performing ERCP procedures and LAC DPH are essential to ensuring optimal surveillance and coordination of prevention activities. The facilities experiencing these outbreaks were large, prestigious hospitals with robust infection prevention and control programs. Due to the design flaw of this instrument, hospitals could follow manufacturer guidelines and standard practices correctly



and still experience duodenoscope-related MDRO transmission. In addition, there may be other facilities with duodenoscope-related transmission of MDROs that may not have the expertise to conduct a complex investigation and implement effective prevention and control strategies. The involvement of LAC DPH in this issue is key to address these problems on a larger scale that will improve the safety of the patients these hospitals treat.

REFERENCES

- 1. Wendorf KA, Kay M, Baliga C, et.al. Endoscopic Retrograde Cholangiopancreatography-Associated AmpC Escherichia coli Outbreak. ICHE. 2015;36(6):634-642
- Epstein L, Hunter JC, Arwady MA, et.al. New Delhi Metallo-β-Lactamase-Producing Carbapenem-Resistant Escherichia coli Associated with Exposure to Duodenoscopes. JAMA. 2014;312(14):1447-1455
- 3. Rutala WA, Weber DJ. ERCP Scopes: What Can We Do to Prevent Infections? ICHE. 2015;36(6):643-64
- 4. http://medical.olympusamerica.com/sites/us/files/pdf/TJF-Q180V_reprocessing_compatible_materials_3.pdf
- 5. FDA Presentation "ERCP Scopes: What Can We Do To Prevent Infections?" William Rutala, Ph.D., MPH http://www.fda.gov/downloads/AdvisoryCommittees/UCM447146.pdf accessed October 5, 2016



2015 SYMPOSIUM ON INFECTION PREVENTION CONTROL IN SKILLED NURSING FACILITIES

OVERVIEW

On 9/1/15, the Los Angeles County Department of Public Health (LAC DPH) Acute Communicable Disease Control (ACDC) program held a symposium for key county skilled nursing facility (SNF) staff responsible for infectious disease outbreak prevention and control. Representatives from SNFs included directors of nursing, administrators, and infection preventionists. Due to the large number of SNFs in LAC, attendance was limited to two representatives per facility. The goals of the symposium were to improve partnerships between SNFs and LAC DPH as well as to improve understanding, surveillance, and response to many infectious diseases that impact SNFs. In addition, this symposium provided education on the National Healthcare Safety Network and antimicrobial stewardship.

SUMMARY

A total of 97 attendees from 63 local SNFs attended the day-long event. In addition, the event included 37 attendees from various LAC DPH programs (including ACDC, Community Health Services, and Health Facilities) and four medical representatives from agencies outside of DPH.

The topics for this event focused primarily on the prevention and control of infectious diseases that are common in SNF settings and greatly impact the vulnerable population cared for in these settings. The presenters were representatives from ACDC and Community Health Services, and the agenda was as follows:

PROGRAM						
7:30 am – 8:30 am	Registration					
	Breakfast & Coffee					
8:30 am – 9:00 am	Welcome & Introduction					
	Laurene Mascola, M.D., M.P.H., F.A.A.P.					
	Chief, Acute Communicable Control Program					
	Christine Wigen, M.D., M.P.H.					
	Medical Epidemiologist, Acute Communicable Disease Control Program					
9:00 am – 10:00 am	Prevention and Control of Influenza					
	Christine Wigen, M.D., M.P.H.					
	Medical Epidemiologist, Acute Communicable Disease Control Program					
10:00 am – 10:10 am	Break					
10:10 am – 11:10 am	Prevention and Control of Scabies					
	L'Tanya English, R.N., P.H.N., M.P.H.					
	Acute Communicable Disease Control Program					



11:10 am – 12:10 pm	Prevention and Control of Norovirus
	Rachel Civen, M.D., M.P.H.
	Medical Epidemiologist, Acute Communicable Disease Control Program
	Public Health's Role in Outbreak Investigations
	Veronica Caballero, R.N., P.H.N., B.S.N.
	Monrovia Health Center (SPA 3)
12:10 pm – 1:00 pm	Lunch
1:00 pm – 2:00 pm	National Healthcare Safety Network (NHSN) & Antimicrobial Stewardship
	Dawn Terashita, M.D., M.P.H.
	Medical Epidemiologist, Acute Communicable Disease Control Program
	Amanda Kamali, MD
	LCDR US Public Health Service
	Centers for Disease Control and Prevention
	Acute Communicable Disease Control Program
2:00 pm – 2:40 pm	Q & A Session
2:40 pm – 2:50 pm	Break
2:50 pm – 3:45 pm	Interactive Activities / Group Discussion
3:45 pm – 4:00 pm	Closing Remarks & Evaluations

In addition to presentations, each attendee received a binder with the following materials and manuals:

- Los Angeles County List of Reportable Diseases and Conditions
- Influenza Outbreak Prevention and Control Guidelines
- Scabies Prevention and Control Guidelines: Acute and Long-Term Care Facilities
- Norovirus Outbreak Prevention Toolkit
- Antimicrobial Stewardship Guidelines Pocket Card
- Health Education Materials for Influenza and Scabies
- Listing of Useful Resources and Websites

Many of these documents and materials were developed specifically for this event. These materials and an archive of the presentations and available on the ACDC website.⁶

Following the presentations, a panel question and answer session was held which provided further clarification on the day's topics. Next, all attendees participated in interactive activities. The goals of these activities were to provide an opportunity for representatives from the SNFs and LAC DPH to collaborate on issues related to infectious disease prevention and control in SNFs as well as to reinforce the guidance and recommendations that were provided during this meeting.

⁶ www.publichealth.lacounty.gov/acd/SNF.htm



Overall, the symposium was very well received and the representatives from the SNFs urged LAC DPH to hold additional trainings to provide further guidance on other topics including antibiotic resistant infections. ACDC plans to hold another symposium in 2016, and these trainings might become an annual event.





INFLUENZA SURVEILLANCE OVERVIEW: 2015–2016 SEASON SUMMARY

OVERVIEW

The 2015-2016 influenza season (October 4, 2015-May 21, 2016) in Los Angeles County (LAC) was moderate overall. Peak activity occurred during mid-February, substantially later compared to previous seasons where peak activity usually occurs from December to January. During the week of February 14-20, 2016 (surveillance week 7), percent positive tests for influenza reached a high of 31.4% for the season (Table 1). In addition, syndromic surveillance detected the highest proportion of visits to Emergency Departments for influenza-like-illness (ILI) that same week (Figure 1). The greatest number of influenza-associated deaths (IAD) also occurred during week 7. Overall IADs increased from last season (N=70), however did not surpass the number of deaths during the last A (H1N1) season of 2013-14. While influenza A (H1N1) viruses predominated, overall influenza A and B viruses were almost equally represented in laboratory surveillance testing throughout the season which is uncommon (Table 1).

California data show that influenza activity across the state was similar to what was seen in LAC, in terms of the timing of peak activity and representation of influenza A/B viruses [1]. Conversely, nationwide influenza activity peaked in mid-March (surveillance week 10), almost a month later than in LAC. Influenza A (H1N1) predominated throughout the season followed by a typical later season increase of influenza B viruses [2]. The majority of viruses characterized by the Centers for Disease Control and Prevention (CDC) were similar to the ones included in this season's vaccine, which resulted in an estimated vaccine efficacy of almost 60% [3].

SENTINEL LABORATORY DATA

Eight sentinel laboratories serving healthcare providers and institutions across LAC reported weekly influenza and other respiratory virus data this season. Although individual cases of influenza are not reportable to the LAC Department of Public Health (DPH), analyzing data from these sentinel labs provides a robust estimate of influenza and other respiratory virus activity in the county. This season a total of 50,640 respiratory isolate tests were reported to LAC DPH (Table 1). Figure 2 shows the distribution of percent positive rates of respiratory specimens by week. Influenza activity began to increase at the end of December, peaked mid-February, then tapered off in April. Other viruses co-circulated with influenza, contributing to the overall impact of respiratory illness in LAC.

INFLUENZA-ASSOCIATED DEATHS

A total of 70 IADs were confirmed in LAC this season. The majority of deaths (61%) occurred in those under 65 years old (median 59 years old), which is consistent with other A (H1N1) predominant seasons that more severely affect the <65 years old population (Table 2). More deaths overall were reported in LAC this season compared to last season. Of the three pediatric IADs reported this season, two had no past medical history identified, which highlights the potential for severe influenza outcomes in otherwise healthy children.



Figure 3 compares the distribution of LAC IADs by age-specific rates across the past seven influenza seasons. During A (H1N1) seasons, the 20-64 age group accounts for a greater proportion of IADs compared to A (H3N2) predominant seasons. Overall, the CDC estimates that about 90% of all IADs occur among adults 65 years and older [4].

RESPIRATORY OUTBREAKS

The total number of respiratory outbreaks confirmed in LAC decreased to 48, compared with 58 last season. The majority of respiratory outbreaks this season occurred in schools or pre-schools (46%), followed by skilled nursing facilities (SNFs) (29%) (Table 3). Respiratory outbreak definitions vary by setting; however, in general, clusters of ILI (fever >100° F with cough and/or sore throat) is cause for investigation.

Thirty-two respiratory outbreaks were confirmed in schools, daycare, and assisted living facilities. Of those, influenza was identified as the responsible pathogen in 11 outbreaks, with flu B accounting for the majority of them. In SNFs, influenza was identified in 11 of 14 respiratory outbreaks.

2016-2017 SEASONAL VACCINE

The World Health Organization and the Food and Drug Administration's Vaccines and Related Biologics Advisory Committee recommends that next season's influenza vaccine contain the following components:

- A/California/7/2009 (H1N1)pdm09-like virus
- A/Hong Kong/4801/2014 (H3N2)-like virus
- B/Brisbane/60/2008-like virus (B/Victoria lineage)
- B/Phuket/3073/2013-like virus (B/Yamagata lineage) (quadrivalent only)

These components represent a change in the A (H3N2) strain and the influenza B lineage included in the trivalent vaccine from the 2015-2016 vaccine. Influenza vaccination is the best way to protect yourself and others from getting influenza and potentially serious complications. Vaccination is recommended for everyone six months of age and older without contraindications.

The live attenuated influenza vaccine, also known as the "nasal spray vaccine", is no longer recommended and should not be used during the upcoming influenza season. This marks a significant change in the CDC's Advisory Committee on Immunization Practices (ACIP) recommendations for the 2016-2017 influenza vaccine. See the full report here: ACIP votes down use of LAIV for 2016-2017 flu season | CDC Online Newsroom | CDC

REFERENCES

- 1. CDPH Influenza Surveillance www.cdph.ca.gov/HealthInfo/discond/Documents/Week%2020%20-%20FINAL%20Report.pdf
- Influenza Activity United States, 2015–16 Season and Composition of the 2016–17 Influenza Vaccine | MMWR

www.cdc.gov/mmwr/volumes/65/wr/mm6522a3.htm?s_cid=mm6522a3_e

3. Flu Vaccine Nearly 60 Percent Effective | CDC Online Newsroom | CDC



www.cdc.gov/media/releases/2016/flu-vaccine-60-percent.html

4. Estimating Seasonal Influenza-Associated Deaths in the United States: CDC Study Confirms Variability of Flu | Seasonal Influenza (Flu) | CDC

www.cdc.gov/flu/about/disease/us_flu-related_deaths.htm

Table 1. LAC Influenza Surveillance Summary									
	2015-	2014-2015							
	Peak Week 7*	YTD ^{**}	9/1/14- 8/8/15						
Sentinel Laboratory Data									
Positive Flu Tests/Total Tests (Percent Positive Flu Tests)	960/3,059 (31.4%)	6,702/50,640 (13.2%)	5,752/48,405 (11.9%)						
Percent Flu A/B	50/50	51/49	81/19						
Outbreaks [†]									
Community Respiratory Outbreaks	1	22	21						
Influenza Confirmed Outbreaks	0	11	37						
Total	1	33	58						
Influenza-Associated Deaths ^{†‡}									
Pediatric Flu Deaths	0	3	3						
Adult Flu Deaths	11	67	51						
Total	11	70	54						
*Week 7 corresponds to February 14-20, 2016 **The influenza surveillence year spans October 4, 2015-May 21, 2016 (surveillance weeks 40-20) *Numbers are provisional and subject to change									

Confirmed influenza death is defined by a positive lab test, ILI symptoms, and clear progression from illness to death.









Table 2. Demographic Characteristics of Influenza Fatalities, LAC, 2009-2016										
		2015-16 ⁺ N(%)	2014-15 N (%)	2013-14 N(%)	2012-13 N (%)	2011-12 N (%)	2010-11 N (%)	2009-10 ^{††} N (%)		
	Median	59	82	56	68	64	45	48		
	Range	1-103	1-101	0-89	0-100	0-104	0-92	0-94		
A	0-5	2 (3)	1 (2)	1 (1)	5 (7)	2 (8)	4 (9)	3 (2)		
Age	6-17	1 (1)	2 (4)	3 (3)	3 (4)	2 (8)	2 (5)	10 (8)		
(years)	18-40	10 (14)	5 (9)	13 (12)	4 (6)	2 (8)	14 (33)	37 (29)		
	41-64	30 (43)	8 (15)	59 (56)	22 (31)	6 (25)	19 (44)	60 (47)		
	65+	27 (39)	39 (71)	30 (28)	36 (51)	12 (50)	4 (9)	17 (13)		
Candan	Male	38 (54)	29 (53)	67 (63)	35 (50)	10 (42)	20 (47)	57 (45)		
Genuer	Female	32 (46)	26 (47)	38 (36)	35 (50)	14 (58)	23 (53)	70 (55)		
	Hispanic	26 (37)	16 (29)	48 (45)	29 (41)	12 (50)	26 (60)	56 (44)		
	White Non-Hispanic	21 (30)	26 (47)	41 (39)	25 (36)	5 (21)	9 (21)	39 (31)		
Race	Asian/Pacifc Islander	13 (19)	8 (15)	7 (7)	6 (9)	3 (13)	4 (9)	9 (7)		
	Black	9 (13)	4 (7)	9 (8)	8 (11)	4 (17)	4 (9)	11 (9)		
	Native American	1 (1)	1 (2)	0	0	0	0	0		
Total Fata	lities	70	55	106	70	24	43	127		

+2015-16 season missing race data for one case

++2009-10 season is missing race data for several cases

Table 3. Characteristics of Confirmed Community Respiratory Outbreaks										
LAC, 2009-2016										
	2015-16	2014-15	2013-14	2012-13	2011-12	2010-11	2009-10			
	N (%)									
Total	48	58	29	73	39	60	436			
Location										
Skilled Nursing Facility (SNF)	14 (29)	25 (43)	12 (41)	23 (32)	12 (31)	7 (12)	25 (6)			
School or Pre-School	22 (46)	20 (34)	11 (38)	41 (56)	22 (56)	46 (77)	376 (86)			
Assisted Living	8 (17)	12 (21)	3 (10)	6 (8)	2 (5)	3 (5)	20 (5)			
Daycare/child care	2 (4)	1 (2)	1 (3)	3 (4)	3 (8)	3 (5)	6 (1)			
Other	2 (4)	0	2† (7)	0	0	1 (2)	9 (2)			
Etiology										
Influenza ++	22 (46)	37 (64)	7 (24)	17 (23)	6 (15)	18 (30)	74 (17)			
Other Respiratory (RSV, Rhinovirus, Strep)	2 (4)	1* (2)	0	1 (1)	7 (18)	4 (7)	0			
Respiratory unknown etiology	24 (50)	20 (34)	22 (76)	55 (75)	26 (67)	38 (63)	362 (83)			

⁺Same home for pregnant women and children

++Confirmed influenza outbreaks must include at least 1 positive lab test

*Both influenza and strep were detected in one outbreak





MEASLES IN A PATIENT WITH PRESUMED IMMUNITY—LOS ANGELES COUNTY, 20157

On February 14, 2015, patient A, aged 17 years, was seen in an emergency department for evaluation of reactive airway disease. In the waiting room at the same time were two siblings, aged six months, presenting with fever and rash; these two children (patients B and C) were later confirmed to have measles. Patient A began a five-day course of oral prednisone (50 mg per day); however, symptoms continued, and patient A returned to the emergency department the next day and received 125 mg of intravenous (IV) methylprednisolone. Patient A had documentation of receipt of two doses of measles, mumps, and rubella (MMR) vaccine at ages 12 months and four years.

A contact investigation was initiated by the hospital to identify all persons who might have been exposed to patient B or patient C. An infant aged 10 days was identified within the first six days of exposure and offered post-exposure prophylaxis with intramuscular (IM) immune globulin. A second infant was identified later and was outside of the window period for immune globulin. Patient A was not identified as a susceptible contact in the investigation because of the documented history of receipt of MMR vaccine. Patients B and C had returned to the hospital on February 17, before receiving a diagnosis of measles, and exposed three other susceptible children (two infants aged <12 months and a child aged three years with leukemia). One infant was offered the MMR vaccine, the other IM immune globulin, and the child with leukemia was offered IV immune globulin. On March 2, 16 days after the first emergency department visit, patient A was hospitalized for vomiting and dehydration. Patient A was also found to be febrile and to have a confluent papular rash that began on the face and spread to trunk and extremities and had small vesicular oral lesions. Measles was confirmed by laboratory testing, and patient A received supportive treatment with anti-emetics and IV fluids.

Patients A, B, and C were part of a measles outbreak originating at the Disney theme park in Orange County, California, in December 2014, which included 28 confirmed cases in Los Angeles County [1]. As of April 17, 2015, a total of 136 measles cases had been documented in California, and among those 10 patients had received at least one dose of the MMR vaccine, 13 had received two doses, and two had received three doses (1; Jennifer Zipprich, PhD, Kathleen Harriman, PhD, California Department of Public Health, personal communication, June 2015). Measles is highly contagious, and high levels of population immunity are required to prevent transmission to susceptible persons. MMR vaccine is highly effective, with a single dose conferring immunity in 92%–95% of persons [2]; however, because vaccine failures do occur, a second dose of measles vaccine has been routinely recommended since 1989 [3]. Complications associated with measles include pneumonia, otitis media, diarrhea, and encephalitis; post-exposure prophylaxis is recommended for all susceptible contacts [2,4]. The MMR vaccine, if administered within 72 hours of initial measles exposure, might provide some protection or modify the clinical course of disease. Persons who are at risk for severe illness and complications from measles who cannot receive the

⁷ Published as: Kamali A, Bagchi CP, Mendoza E, Wilson D, Schwartz B, Mascola L. Measles in a patient with presumed immunity—Los Angeles County, 2015. MMWR October 9, 2015 / 64(39);1123.



MMR vaccine, including infants aged <12 months, persons who are severely immunocompromised (including persons taking high-dose steroids for \geq 2 weeks), and persons with leukemia or lymphoma [2,5], should receive prophylaxis with immunoglobulin within six days of exposure.

Patient A had received two doses of MMR vaccine and did not meet criteria for being severely immunocompromised; however, this patient did develop measles after being exposed in the setting of a hospital emergency department to patients with laboratory-confirmed measles. Although it is not known whether patient A developed immunity to measles in response to the two administered doses of MMR vaccine or whether patient A had an unrecognized immunocompromising condition, the recent steroid use might have weakened the patient's immune response and rendered patient A susceptible to a wild measles strain. The diagnosis of measles in patient A highlights the concern that immunocompromised and susceptible persons might be exposed in a health care setting. More information is needed concerning the effect of immunomodulating drugs on vaccine-induced immunity to measles and other vaccine-preventable diseases.

REFERENCES

- 1. California Department of Public Health. Surveillance update. Available at www.cdph.ca.gov/HealthInfo/discond/Documents/Measles_update_4-17-2015_public.pdf
- 2. CDC. Prevention of measles, rubella, congenital rubella syndrome, and mumps, 2013: Summary recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep 2013;62(No. RR-4).
- 3. Rosen JB, Rota JS, Hickman CJ, et al. Outbreak of measles among persons with prior evidence of immunity, New York City, 2011. Clin Infect Dis 2014;58:1205–10.
- Perry RT, Halsey NA. The clinical significance of measles: a review. J Infect Dis 2004;189(Suppl 1):S4– 16.
- 5. California Department of Public Health. Measles investigation quicksheet. Available at www.cadph.ca.gov/programs/immunize/Documents/CDPHMeaslesInvestigationQuicksheet.pdf



INVESTIGATION OF AND RESPONSE TO TWO PLAGUE CASES YOSEMITE NATIONAL PARK, CALIFORNIA, USA, 2015⁸

OVERVIEW

In August 2015, plague was diagnosed for two persons who had visited Yosemite National Park in California, USA. One case was septicemic and the other bubonic. Subsequent environmental investigation identified probable locations of exposure for each patient and evidence of epizootic plague in other areas of the park. Transmission of *Yersinia pestis* was detected by testing rodent serum, fleas, and rodent carcasses. The environmental investigation and whole-genome multilocus sequence typing of *Y. pestis* isolates from the patients and environmental samples indicated that the patients had been exposed in different locations and that at least two distinct strains of *Y. pestis* were circulating among vector—host populations in the area. Public education efforts and insecticide applications in select areas to control rodent fleas probably reduced the risk for plague transmission to park visitors and staff.

INTRODUCTION

Plague is a zoonotic disease caused by the gram-negative bacterium *Yersinia pestis*; the organism's reservoir is rodents, and the vectors are fleas [1,2]. Transmission to humans can occur through bites by infected fleas or through handling *Y. pestis*—infected rodents [1,2]. Epidemics of plague still occur on the continents of Africa, Asia, and North and South America [3]. Plague was introduced to California in 1900 [1,4–6] where over the next 25 years it caused occasional outbreaks in rats commensally residing with humans in urban areas [2,4,6]. Shortly after its introduction, *Y. pestis* moved into wild rodent populations, establishing a sylvatic transmission cycle [7,8]. In subsequent decades, plague spread across California and other western states [9] periodically affecting humans [4–6, 10–13].

The California ground squirrel plays a major role in human exposure in California because its predominant flea species *Oropsylla montana* is a competent *Y. pestis* vector [1,2] that is often abundant on this rodent and in its burrows [14] and will readily bite humans [1,11]. Since the 1980s, evidence of *Y. pestis* transmission in rodents in the Sierra Nevada mountains has been generally restricted to locations at elevations >1,200 meters (California Department of Public Health, unpub. data, 1983–2015). Despite ongoing sylvatic transmission, human plague remains rare in the western United States [15–17], including in California where no cases have been confirmed since 2006 [18,19].

During the summer of 2015, the Los Angeles County Department of Public Health (LAC DPH) and the Georgia Department of Public Health reported two cases of plague in persons who had recently travelled to Yosemite National Park (Yosemite). The California Department of Public Health (CDPH), in collaboration with the US Centers for Disease Control and Prevention (CDC) and the National Park Service (NPS), investigated the increased *Y. pestis* transmission in Yosemite. We summarize the epidemiologic, laboratory, environmental findings, and the public health response.

⁸ Full article published as: Danforth M, Novak M, Peterson J, et al. Investigation of and Response to 2 Plague Cases, Yosemite National Park, California, USA, 2015. Emerg Infect Dis. 2016 Dec; 22(12): 2045–2053.



RESULTS

Environmental Findings

Plague risk assessments were conducted for nine locations in Yosemite and the surrounding national forests visited by the patients. Within the park, eight more sites were also evaluated for *Y. pestis* transmission and potential risk areas for transmission to humans.

Flea Control

Sites with evidence of recent *Y. pestis* transmission and an increased risk for human exposure were temporarily closed, and rodent burrows were treated with insecticide to reduce flea populations and protect wildlife and human health. The following five areas in Yosemite were identified for insecticide treatments: Crane Flat Campground, Glacier Point, Tuolumne Meadows Campground, Tamarack Flat Campground, and the Crane Flat–NatureBridge campus. In total, 16.3 kg of 0.05% deltamethrin was used per label instructions to treat an estimated 3,700 rodent burrows. Although time and logistical constraints precluded pre- and post-treatment flea evaluations at all locations, evidence from limited sampling suggested that the insecticide applications reduced the local flea populations.

Public Outreach

To further reduce the plague risk for Yosemite visitors and staff, NPS and collaborating agencies initiated an aggressive public education campaign. The campaign included three news releases issued August 6– 18, media interviews, and website alerts. The park newsletter, *The Yosemite Guide*, which was given to persons in every entering vehicle, included information about plague. Placards with plague information were posted at park entrances, locations with confirmed *Y. pestis* transmission, all campgrounds, and many day use locations and trailheads. Educational pamphlets were available to visitors at a variety of locations, including affected campgrounds.

DISCUSSION

In August 2015, these two cases of plague were linked to exposure in the internationally popular Yosemite National Park. The initial public health investigation and response with broad media coverage of the first case led to the rapid recognition and appropriate treatment of the second case-patient.

The investigation found little overlap in the travel itineraries of the two patients, and isolation of distinct strains of *Y. pestis* suggested that at least two *Y. pestis* strains were circulating among vector–host populations in the Yosemite area. In the only area visited by both patients, Yosemite Valley, no evidence of *Y. pestis* transmission in rodents was found, and *Y. pestis* has not been detected in the valley's rodent populations in recent decades (CDPH, unpub. data, 1984–2015). We were able to connect the exposure of patient 1 to epizootic transmission at the campground on the basis of the visual observations at Crane Flat Campground, the positive results for rodent serology and the pool of fleas collected there, and whole-genome MLST analysis of *Y. pestis* isolates from patient 1 and the flea pool. The most likely exposure site for patient 2 was Glacier Point, 20 km away, on the opposite side of Yosemite Valley. Although *Y. pestis*-



seropositive rodents were found at this location, we did not detect active infection in rodents or fleas and were therefore unable to directly link the patients' exposure to this site by whole-genome MLST.

The environmental investigation found evidence of *Y. pestis* transmission in disparate locations of the park, including epizootic activity in the Tuolumne Meadows area, \approx 41 and 25 km from Crane Flat and Glacier Point, respectively. Evidence of *Y. pestis* transmission in rodents was found at 4 of the 5 areas trapped. Of the eight species of rodents live trapped in Yosemite, *Y. pestis* antibodies were detected in only 5 (15.2%) of 33 lodgepole chipmunks and 3 (7.3%) of 41 California ground squirrels. However, *Y. pestis* was also isolated from golden-mantled ground squirrel and Douglas squirrel carcasses and a deer mouse flea, indicating broader zoonotic involvement.

The 2015 findings for Yosemite share some striking similarities with those associated with the only human plague case previously associated with Yosemite [20]. In 1959, a teenage boy became ill after camping along Yosemite Creek trail, \approx 5 km from Crane Flat Campground. Subsequent investigation by CDPH and CDC found evidence of a recent epizootic plague event that had decimated the rodent populations near the campsite. During this investigation, *Y. pestis* transmission was also documented in Tuolumne Meadows and at Lake Tenaya.

The rapid interagency investigation and public health response to these cases probably reduced the risk for plague among Yosemite visitors and staff. Critical risk-reduction measures included expanding the investigation to recreational sites beyond those visited by the patients and localized insecticide treatments at sites with *Y. pestis* transmission. Increased educational efforts informing the public about how to reduce their exposure to the cause of this potentially fatal disease contributed to the early diagnosis for patient 2 and to increased reports of finding dead rodents in the park, which led to detection of *Y. pestis* transmission at additional locations.

REFERENCES

- 1. Pollitzer R. Plague. Geneva: World Health Organization. 1954.
- Perry RD, Fetherston JD. *Yersinia pestis*—etiologic agent of plague. Clin Microbiol Rev. 1997;10:35– 66.
- 3. Gage KL, Kosoy MY. Natural history of plague: perspectives from more than a century of research. Annu Rev Entomol. 2005;50:505–28.
- 4. Link VB. A history of plague in United States of America. Public Health Monogr. 1955;26:1–120.
- 5. Caten JL, Kartman L. Human plague in the United States, 1900-1966. JAMA. 1968;205:333–6.
- 6. Kugeler KJ, Staples JE, Hinckley AF, Gage KL, Mead PS. Epidemiology of human plague in the United States, 1900-2012. Emerg Infect Dis. 2015;21:16–22.
- 7. Barnes A. Surveillance and control of bubonic plague in the United States. Symposium of the Zoological Society of London. 1982;50:237–70.
- Inglesby TV, Dennis DT, Henderson DA, Bartlett JG, Ascher MS, Eitzen E, et al. Working Group on Civilian Biodefense. Plague as a biological weapon: medical and public health management. JAMA. 2000;283:2281–90.



- 9. Antolin JF, Gober P, Luce B, Biggins DE, van Pelt WE, Seery DB, et al. The influence of sylvatic plague on North American wildlife at the landscape level, with special emphasis on black-footed ferret and prairie dog conservation. In: Transactions of the 67th North American Wildlife and Natural Resources Conference; 2002 Apr 3–7. Washington (DC): Wildlife Management Institute; 2002.
- 10. CDC. Human plague—four states, 2006. MMWR Morb Mortal Wkly Rep. 2006;55:940–3.
- 11. Craven RB, Maupin GO, Beard ML, Quan TJ, Barnes AM. Reported cases of human plague infections in the United States, 1970-1991. J Med Entomol. 1993;30:758–61.
- 12. Wong D, Wild MA, Walburger MA, Higgins CL, Callahan M, Czarnecki LA, et al. Primary pneumonic plague contracted from a mountain lion carcass. Clin Infect Dis. 2009;49:e33–8.
- 13. Lowell JL, Wagner DM, Atshabar B, Antolin MF, Vogler AJ, Keim P, et al. Identifying sources of human exposure to plague. J Clin Microbiol. 2005;43:650–6.
- 14. Lang JD. Factors affecting the seasonal abundance of ground squirrel and wood rat fleas (Siphonaptera) in San Diego County, California. J Med Entomol. 1996;33:790–804.
- 15. CDC. Imported plague—New York City, 2002. MMWR Morb Mortal Wkly Rep. 2003;52:725–8.
- Eisen RJ, Enscore RE, Biggerstaff BJ, Reynolds PJ, Ettestad P, Brown T, et al. Human plague in the southwestern United States, 1957-2004: spatial models of elevated risk of human exposure to *Yersinia pestis.* J Med Entomol. 2007;44:530–7 .10.1603/0022-2585(2007)44[530:HPITSU]2.0.CO;2
- 17. Kwit N, Nelson C, Kugeler K, Petersen J, Plante L, Yaglom H, et al. Human Plague United States, 2015. MMWR Morb Mortal Wkly Rep. 2015;64:918–9.
- 18. California Department of Public Health. Yearly summaries of selected general communicable diseases in California, 2001–2010. Sacramento (CA): The Department; 2015.
- 19. California Department of Public Health. Yearly summaries of selected general communicable diseases in California, 2011–2014. Sacramento (CA): The Department; 2015.
- 20. Murray KF, Kartman L. Plague in California during 1959. California Vector Views. 1959;6:66–7.



MULTI-AGENCY RESPONSE TO A FLEA-BORNE TYPHUS OUTBREAK ASSOCIATED WITH A MOBILE HOME COMMUNITY

BACKGROUND

Flea-borne typhus is an acute febrile illness caused by *Rickettsia typhi* or *R. felis*. Persons typically become infected when the feces of a carrier flea enters the body through a bite or other break in the skin [1]. Most infections present as self-limited illness; however, infection for some progress to a more serious febrile illness and require hospitalization [2,3]. Deaths have been documented but are rare [4].

In Los Angeles County (LAC), cats, opossums, and the cat flea (*Ctenocephalides felis*) maintain the suburban life cycle of flea-borne typhus [1,5,6]. The flea acquires the bacteria from small urban mammals such as opossums that can harbor these bacteria. Opossums, a peridomestic animal, carry large numbers of fleas and often inhabit areas near human habitation where there is readily available food and harborage. Fleas may move from opossums to domestic pets (dogs and cats) and then to humans where they cause infection.

Flea-borne typhus is not a nationally reportable condition, so the number of cases occurring in the US is unknown. Cases primarily occur in Texas, Hawaii, and California where typhus is endemic. Providers and laboratories are mandated to report suspect cases to their local public health departments in these places. The majority of California's cases occur in LAC. In 2014, 51 cases were reported in California; 44 (86%) were LAC residents. This number corresponds to an LAC incidence of 0.47 per 100,000 [7].

On June 16, 2015, a local hospital infection preventionist alerted the Acute Communicable Disease Control program (ACDC) of three hospitalized flea-borne typhus cases occurring from April 23, 2015 to June 9, 2015 among residents of a 95-unit mobile home community (MHC). ACDC coordinated a multi-agency investigation of this outbreak in order to identify additional cases, identify and mitigate risk factors, and prevent further cases from occurring.

METHODS

Risk Factor Identification

To assess for risk factors at the MHC, several multi-agency site investigations of the MHC were conducted from June through November 2015. These agencies included ACDC, Environmental Health (EH), Community Health Services (CHS), Veterinary Public Health (VPH), and San Gabriel Valley Mosquito and Vector Control District (SGV).

Community Outreach

Printed health education materials (Figure 1) in English and Spanish were distributed to residents, and a community outreach meeting was hosted at a location adjacent to the MHC. Meeting invitations, notification of the investigation, and educational pamphlets were distributed to residents in English and Spanish (Figure 2). The notification letter urged residents to contact ACDC if they had been ill with fever



and headache or rash anytime since March 1, 2015, one month before the earliest case onset. All residents who contacted ACDC were interviewed by an ACDC investigator using a standardized questionnaire, which included information on individual demographics, clinical signs and symptoms, and possible exposures. Those with persisting symptoms were referred to their personal healthcare provider. ACDC consulted these providers and coordinated collection of a serological sample and testing.

Case Review and Case Finding

Outbreak-associated cases were defined as persons with the following criteria and symptom onset between March 1 and August 31, 2015:

- residence within the MHC,
- fever with headache or rash, and
- positive *R. typhi* or *R. rickettsii* laboratory test (immunoglobulin M (IgM) >1:64 and/or immunoglobulin G (IgG)>1:64).

ACDC increased surveillance for additional flea-borne typhus cases linked to this MHC. Disease surveillance staff reviewed all cases reported to DPH from January 1 through August 31, 2015 for possible links to the MHC. ACDC also contacted laboratory directors from four acute care facilities that could have evaluated an MHC resident or persons residing within this geographical area for an acute febrile illness. ACDC requested that laboratory directors review data for positive *R. typhi* or *R. rickettsii* laboratory tests (IgM >1:64 and/or IgG>1:64) and submit results to ACDC. A Los Angeles Health Alert Network⁹ (LAHAN) notification was sent to emergency rooms and urgent care providers that served MHC residents and persons within this area. It requested that providers consider the possibility of flea-borne typhus in patients presenting with acute onset of fever, headache, rash, and myalgia. Clinicians were asked to collect serum specimens from suspect cases and to report suspect cases to ACDC.

To confirm etiology, available samples were transported to the LAC Public Health Laboratory (PHL). Samples were tested for *R. typhi* and *R. rickettsii* IgG and IgM via indirect immunofluorescence antibody testing (IFA).

RESULTS

Risk Factor Identification

On June 18, 2015, EH and SGV visited the 95-unit MHC. EH visited cases' residences and provided education regarding risk reduction. SGV inspected the entire grounds and identified multiple sanitation concerns: large numbers of free-roaming cats (>30), cat and dog feces throughout the grounds, pet food and water bowls outside residences, and an abundant flea population. Two opossums were trapped by SGV on June 18 and 22 wherein 615 and 1,087 fleas were identified, respectively, when combed. A pool of five fleas from each opossum was tested for rickettsial organisms by the Orange County Mosquito and Vector Control District. Fleas from the two opossums tested positive for *R. felis* via polymerase chain reaction (PCR), but *R. typhi* was not detected.

⁹ www.publichealth.lacounty.gov/eprp/lahan/lahan.htm

Multi-Agency Response to a Flea-Borne Typhus Outbreak Page 66



On June 24, 2015, SGV issued a summary abatement notice to the property owner and property manager but not the residents. The notice required the owner to remove all feces from the grounds, eliminate the availability of pet food outdoors, enforce property rules limiting the number of pets and requiring pet registration with management, provide bi-weekly flea abatement, and remove feral animals.

A follow-up site visit with representatives from ACDC, EH, SGV, VPH, and CHS was conducted on August 13, 2015. Consistent with the June site visit, investigators observed many free-roaming cats, dog and cat feces, and pet food left outside. There were several aspects that likely also increased the presence of feral cats and fleas. First, three community dumpsters were present, uncovered, and overfilled with refuse. In addition, the foundation supporting and surrounding the mobile home was damaged. This offered potential harborage for wildlife. Also, a noticeable flea population persisted despite flea abatement efforts by the management company.

SGV re-contacted management to reiterate the order for bi-weekly flea abatement by a private company. SGV monitored the flea population by placing six glue boards (16 cm x 11 cm in size) throughout the neighborhood on a bi-weekly basis to assess for the presence of fleas. At the start of September, an average of 14 fleas were trapped on the boards. In November 2015, two consecutive visits yielded an average of zero fleas collected, suggesting a sustained reduction in the presence of fleas.

Community Outreach

A total of three residents contacted ACDC in response to the investigation letter. One was referred to his primary care physician due to persisting symptoms consistent with the case definition. However, laboratory results determined that he did not meet the case definition.

The community meeting was held adjacent to the MHC on August 24, 2015 with representatives from ACDC, EH, SGV, VPH, CHS, city council, and the office of a state senator. Approximately 20 residents attended the community meeting. An ACDC physician presented information about flea-borne typhus and advice for reducing its transmission including instructing residents not to leave pet food outdoors. VPH distributed flea collars free of charge to attendees for their pet cats or dogs. CHS public health nurses performed free on-site blood draws and completed the standardized questionnaire for five attendees who reported experiencing symptoms consistent with flea-borne typhus since March 1, 2015. Two additional outbreak-associated cases were identified.

Case Review and Case Finding

Two additional outbreak cases were identified among MHC residents at the community meeting as described. However, no additional cases were identified within the geographic area of the MHC using case finding and provider outreach methods employed during the investigation. Follow up through December 2015 to ensure implemented control measures were effective yielded no additional cases.

A total of five confirmed flea-borne typhus cases were identified within the MHC; three initially reported by the hospital infection preventionist and two additional cases that were identified through



investigational activities and confirmed via IFA (Table 1). Initially, Case A's lab values did not meet the CDPH flea-borne typhus case definitions but was reclassified as a confirmed case due to the epidemiologic link to the MHC. Illness onset ranged over three months, from April through June 2015, but was unknown for the non-hospitalized cases. Cases were primarily female (4/5) with a median age of 48 (range 42-67). All cases owned at least one dog; two cases also owned at least one cat. Of the five cases, three were hospitalized for a total of 15 nights (average = 5). All five cases recovered without complication.

DISCUSSION

An outbreak of flea-borne typhus occurred in a LAC MHC in the summer of 2015, resulting in a total of five identified cases. It is likely that additional cases occurred as part of this outbreak but remain undetected due to the non-specific, typically mild presentation of this disease and the residents' limited access to health care.

In LAC, the incidence and geographic spread of typhus cases has increased over recent years. Total cases increased from 31 in 2010 (0.3 per 100,000) to a peak of 68 cases in 2013 (0.7 per 100,000), with a slight decrease to 44 cases in 2014 (0.5 per 100,000). Despite this overall increase, typhus clusters remain an unusual occurrence. Prior to this investigation, the last documented cluster in LAC occurred in 2005 [8].

The etiologic agent of flea-borne typhus has received increased debate. *R. typhi* is traditionally considered the etiologic agent of flea-borne typhus. However, *R. felis* was detected in the fleas obtained from opossums in our investigation. This suggests that the causative agent of this outbreak was possibly *R. felis*, a rickettsial agent that is serologically indistinguishable from *R. typhi* in humans due to cross-reactivity [9,10]. PCR testing of samples obtained from acutely ill patients is necessary to make the distinction between the two organisms; these samples were not available during our investigation [10]. *R. felis* serology tests are not commercially available nor is PCR testing for *R. typhi*. Future efforts should be made to acquire samples in acutely ill persons with suspected flea-borne typhus and tested via PCR for both *R. felis* and *R. typhi* by appropriate laboratories.

Limitations of this investigation included the amount of time required to coordinate the multiple agencies involved, which highlights the need to continually foster relationships with outside agencies. As a result, our on-site testing of residents occurred at a time when cases were no longer acutely ill. However, there was evidence that *R. felis* was still circulating in the community at the time of our involvement. Investigators successfully remediated that risk factor and improved overall environmental conditions.

Overall, this response demonstrated that the implementation of a multi-faceted intervention can interrupt the suburban transmission cycle of flea-borne typhus. Multiple interactions with the management were needed to sufficiently improve site conditions and decrease the flea population. More intimate engagement of community members and provision of pet flea control supplies was ultimately required in order to affect a change in the community. Infectious disease epidemiologists, community health providers, veterinarians, environmental health specialists, vector control experts, and city representatives were required to address the many factors contributing to the outbreak. One year post-



outbreak, we have received no additional reports of cases occurring in the MHC or surrounding area, suggesting that our efforts were successful in mitigating the outbreak.

REFERENCES

- 1. Civen R, Ngo V. Murine typhus: an unrecognized suburban vectorborne disease. *Clin. Infect. Dis.* 2008;46(6):913–918.
- 2. Fergie J, Purcell KM. Spontaneous splenic rupture in a child with murine yyphus. *ET J*. 2004;23(12):1171–1172.
- 3. Vallejo-Maroto I, Garcia-Morillo S, Wittel MB, et al. Aseptic meningitis as a delayed neurologic complication of murine typhus. *Clin. Microbiol. Infect.* 2002;8(12):826–827.
- 4. Dumler JS, Taylor JP, Walker DH. Clinical and laboratory features of murine typhus in south Texas, 1980 through 1987. *J. Am. Med. Assoc.* 1991;266(10):1365–1370.
- 5. Boostrom A, Beier MS, Macaluso JA, et al. Geographic association of Rickettsia felis-infected opossums with human murine typhus, Texas. *Emerg. Infect. Dis.* 2002;8(6):549–554.
- 6. Sorvillo FJ, Gondo B, Emmons R. A suburban focus of endemic typhus in Los Angeles County: association with seropositive domestic cats and opossums. *J. Trop. Med. Hyg.* 1993;48(2):269–273.
- Human Flea-Borne Typhus Cases in California. California Department of Public Health; (Accessed August 23, 2016).(https://www.cdph.ca.gov/HealthInfo/discond/Documents/FleaborneTyphusCaseCounts.pdf). (Accessed August 23, 2016).
- Acute Communicable Disease Control 2005 Special Reports. A suburban neighborhood outbreak of murine typhus, South Pasadena, May 2005.(http://www.publichealth.lacounty.gov/acd/reports/ spclrpts/spcrpt05/Murine_SS05.pdf). (Accessed September 1, 2016).
- 9. Raoult D, La Scola B, Enea M, et al. A flea-associated Rickettsia pathogenic for humans. *Emerg. Infect. Dis.* 2001;7(1):73–81.
- 10. Azad AF, Radulovic S, Higgins JA, et al. Flea-borne rickettsioses: ecologic considerations. *Emerg. Infect. Dis.* 1997;3(3):319–327.

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Figure 1. Community Meeting Invitation





Figure 2. Community Meeting Invitation



Table 1: Case Characteristics

Case	Age	Sex Cat Dog Onset Hospitalized		Hospitalized	Hospital	Hospital R. Typhi		R. Rickettsii			
	Group		Owner	Owner	Date		Nights				
								lgG	lgM	lgG	ΙgΜ
А	45-54	М	Yes	Yes	4/20/15	Yes	6	<64	1:64	N/A	N/A
В	35-44	F	No	Yes	4/9/15	Yes	4	1:128	≥1:256	N/A	N/A
С	65-74	F	Yes	Yes	6/5/15	Yes	5	1:128	1:128	N/A	N/A
D	45-54	F	No	Yes	Unknown	No	0	1:128	<64	1:64	<64
E	45-54	F	No	Yes	Unknown	No	0	1:64	<64	1:128	<64