ACUTE COMMUNICABLE DISEASE CONTROL PROGRAM

ANNUAL MORBIDITY REPORT

AND

SPECIAL STUDIES REPORT

2006

Los Angeles County
Department of Public Health

Public Health
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Acute Communicable Disease Control Program
Annual Morbidity Report
2006

● EXECUTIVE SUMMARY ●

In Los Angeles County (LAC), more than 80 diseases and conditions, as well as unusual disease occurrences and outbreaks, are reportable by law. Acute Communicable Disease Control (ACDC) is the lead program for the surveillance and investigation of most communicable diseases—responsibilities exclude tuberculosis, sexually transmitted diseases, and HIV or AIDS. Surveillance is primarily passive, with reports submitted via facsimile, mail, or telephone by providers and hospitals and electronically from several laboratories. Reporting urgency varies according to disease and ranges from immediate reporting by telephone to the LAC Department of Public Health (DPH) to reporting required within 7 days of identification.

In addition to disease surveillance and investigation, ACDC sets policy and procedures for DPH activities related to infectious and communicable disease prevention and control. Our program interprets and enforces state and federal laws and regulations, and interfaces with other jurisdictions, programs and agencies responsible for public health. ACDC frequently provides consultation to the medical community on issues of communicable and infectious diseases and education to medical professionals.

ACDC has several units and special projects, each with unique goals and objectives for the surveillance and control of communicable disease:

- **Food and Water Safety Unit**: The aim of this unit is to decrease morbidity related to food and waterborne pathogens through surveillance of reported diseases and foodborne illness reports, to detect outbreaks and monitor trends. Pathogens of special interest include *Listeria*, norovirus and *Salmonella* and *E. coli*.

Los Angeles County:
A description of our community

LAC is one of the nation's largest counties, covering over 4,000 square miles. While LAC enjoys fairly temperate, year-round weather, it encompasses a wide variety of geographic areas including mountain ranges, arid deserts, and over 80 miles of ocean coastline. Accordingly, one challenge of disease surveillance, response and control is responding to its enormous size. LAC presently has the largest population (nearly 10 million) of any county in the US and is exceeded by only eight states. LAC is densely populated, with over one-fourth of the state's population. LAC is home to approximately 100 hospitals with 74 emergency departments, more than 30,000 licensed physicians, over 450 subacute healthcare facilities, and about 25 thousand retail food purveyors.

Another challenge is the extensive diversity of our population coupled with a high level of immigration. Nearly half of our residents are Latino (48%), around one-third white (30%), and around one in ten are Asian (13%) or black (9%). Residents report over 90 languages as their primary spoken language. There is also substantial economic diversity within our county; while LAC is world renowned for its areas of wealth and privilege, there is also considerable poverty. The 2000 US census recorded over 1.5 million residents (nearly 16% of LAC's population) living in poverty.

LAC is a major port of entry for immigrants to the US. According to a 1999 survey, almost one-third of respondents stated they were born outside of the US. In 2002, an Immigration and Naturalization Report found that California was home to the largest number of legal immigrants to the US, and over one-third of these immigrants reported settling in LAC. The population is also highly mobile. In terms of air travel alone, each year roughly 55 million travelers come through the Los Angeles International airport (over 40 million domestic and 14 million international flights yearly)—making it the nation's 3rd busiest airport.

- **Vectorborne Diseases and Central Nervous System Infections Unit**: This unit conducts surveillance and provides disease consultation for a variety of vectorborne and zoonotic diseases (e.g., West Nile virus, plague), meningococcal disease, and other causes of encephalitis and meningitis. The Varicella Surveillance Project, a special research project, is also part of this unit.

- **Hospital Outreach Unit:** This unit assists hospitals and other healthcare facilities with outbreak investigations and provides consultation on infection control issues. It strives to enhance communication with hospitals by interacting with infection control professionals, emergency departments, and laboratories.

- **Bloodborne Pathogens and Antimicrobial Resistance Unit**: Conducts surveillance and investigations of the viral hepatidities, MRSA, and invasive disease caused by pneumococcus and group A streptococcus.

- **Immunization Program**: Its mission is to improve immunization coverage levels to prevent the occurrence of vaccine-preventable diseases. Activities include surveillance for vaccine-preventable diseases, outbreak investigation and control, perinatal hepatitis B case management, immunization coverage assessments, professional education and training, community outreach and education, partnerships with child health advocates and organizations, vaccine management and distribution (especially influenza vaccines), assuring delivery of immunization services in DPH and community facilities, immunization registry development, health services research, and sponsorship of an Immunization Roundtable.

- **Electronic Data Collection Section**: The aim of this section is to enhance surveillance and epidemiology capacity to improve disease reporting and improve detection of unusual occurences. Activities include syndromic surveillance and electronic reporting from laboratories.

- **Planning, Evaluation & Response Section** is responsible for activities related to cross-cutting ACDC and bioterrorism performance measures, communicable disease annual reports, strategic planning, health education, and consequential epidemiology (application of public health research and aims to improve health outcomes). This section also plans, evaluates, trains, and educates internal and external partners in response to a potential or actual biologic incident which may be the result of bioterrorism.

Additional information about ACDC is available at:
www.lapublichealth.org/acd/index.htm.

Emerging and Re-Emerging Infectious Diseases—Los Angeles County, 2006

New diseases emerge, conditions once thought gone reemerge, and existing diseases acquire added prominence. While West Nile virus (WNV) was undoubtedly one of the more notable infectious diseases to emerge in recent years, its local impact continued to decline in 2006, with just 16 human WNV infections reported, including 1 case of encephalitis, 4 cases of meningitis, 8 cases of WNV fever, and 3 asymptomatic blood donors; there were no associated deaths. WNV environmental surveillance in mosquitoes, dead birds, and sentinel chickens documented that WNV has become enzootic in Los Angeles County (LAC). Arbovirus experts speculate local weather conditions and aggressive mosquito abatement efforts as well as personal behaviors, such as increased use of mosquito repellant and avoidance of risky areas at prime mosquito times, play a role in the decline of human infections.

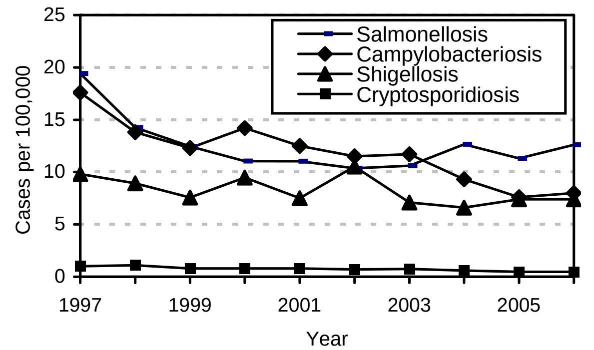
Emerging and Re-Emerging Diseases

West Nile virus infection continued to decreased compared to 2004; the virus is now enzootic to our region and human cases of illness can be expected annually.

Food- and Waterborne Diseases

Investigation of cases and outbreaks of diseases spread by food and water sources make up a large portion of activities conducted by ACDC. Overall, food- and waterborne diseases have declined since the mid-1990's and stabilized at lower rates as shown in Figure 1; also see separate reports on campylobacteriosis, cryptosporidiosis, listeriosis, salmonellosis, shigellosis, typhoid fever, and vibriosis for details. The declining trend in reported cases is most evident with the bacterial diseases campylobacteriosis and shigellosis, and mirrors national trends depicting sustained decreases among many foodborne illnesses, particularly those of bacterial origin. While the underlying causes for these local and national trends are not known, the implementation of control measures at every level are believed to be important factors in the reduction of food and water-related illnesses. On a national level, these include the expansion of federal food safety and inspection services with particular attention to fresh produce safety. Locally, a highly publicized restaurant grading system implemented in LAC in 1998 may have also improved food safety through education of food handlers and the public regarding best practices to reduce foodborne disease.

Figure 1
Selected Food and Waterborne Diseases
Incidence Rates by Year
LAC, 1997–2006



The LAC 2006 salmonellosis crude rate rose slightly compared to 2005 (Figure 1), and has remained below the national rate since 1998 after an overall decrease of more than 100% since 1994. The national incidence of salmonellosis has also been decreasing, but at a much slower rate than seen in LAC in the previous 10 years. Although many food items and both potable and recreational water sources have been implicated in the transmission of salmonella, salmonellosis is most commonly associated with eggs, poultry, and fresh produce. Another prominent source is contact with reptiles, either directly or through surfaces or other people exposed to reptiles. In 2006, at least 104 (8.6%) of LAC salmonellosis cases reported contact with turtles, lizards or snakes.

In 2006, there were 37 foodborne disease outbreaks representing 425 individuals with illness; this represented 15% more outbreaks than in 2005, but fewer persons were affected. While the overall incidence of most of these diseases has been decreasing, food- and waterborne diseases continue to account for considerable morbidity and mortality—thousands of preventable infections continue to occur yearly. The majority of people affected by these illnesses improve without treatment or complications. However, some infections may be invasive, especially among children, the elderly and those with certain chronic medical conditions (e.g., the immunocompromised), leading to hospitalization and death. Further efforts are needed to improve food and water quality and to educate food industry and the public about proper food storage, handling, and preparation.

The community-wide outbreak of hepatitis A that started in August of 2005 did not decrease to base-line levels until July of 2006. There were two outbreaks of acute hepatitis A in 2006. The first occurred in a bar in the south bay area in May and consisted of eight patrons. Transmission was believed to be due to a contaminated ice chest that patrons had access to. After Environmental Health closed down the bar and the owner corrected sanitation and food hygiene practices, no more cases were identified. The second outbreak, also affecting eight people, occurred in September, 2006. Eight people who ate at a single restaurant in Pomona in August were diagnosed with acute hepatitis A in September. Despite an active investigation, including a case-control study and Environmental Health inspections, no food or worker source could be found.

Vaccine Preventable Diseases

Surveillance for influenza is being scrutinized closely and enhanced in light of pandemic preparations. Working with the syndromic surveillance team, analyses specific for influenza-like illness surveillance have been devised, especially in children under 5 who are often considered harbingers of influenza activity in the community. A pilot surveillance system was established with the Los Angeles Unified School District (LAUSD); the results tallied well with other standard surveillance systems for influenza including emergency department data and viral isolates. To keep the public and healthcare professionals abreast of influenza related activities, a newsletter was developed to be distributed weekly during the standard influenza season (October-April). It includes results of our varied surveillance systems as well as breaking information from the US government or research.

Vaccine Preventable Diseases

- Immunization levels in LAC continue to be among the highest for large urban areas in the United States.
- However, a resurgence of reported mumps cases occurred nationally in 2006.

Vaccine-preventable disease incidence has decreased dramatically due to immunizations. Keeping young children current with their immunizations has historically been considered the most efficient method available to prevent disease incidence in children and control disease incidence among adults. Immunization levels in LAC among children 19-35 months of age continue yearly to exceed the national Healthy People year 2010 goal of 80% and are among the highest levels for large urban areas nationally. Despite these successful strides in vaccination coverage levels, select vaccine-preventable diseases have shown a resurgence in recent years. After a 30-year record high of reported pertussis cases in 2005 due in conjunction to the historical 3-5 year cyclical trend of increasing pertussis rates, improved recognition and reporting, and adolescents and adults comprising a larger proportion of cases, case numbers decreased in 2006. However, a pertussis outbreak occurred at a local university in 2006, continuing the trend of increased cases identified among adolescents/adults. In addition, during January to October 2006, a multi-state mumps outbreak occurred in the Midwest area of the United States, primarily affecting the 18-24 year age group; a high proportion of whom were college students. This outbreak had a profound impact on mumps surveillance nationwide and doubled the number of reports received in LAC in 2006, as compared to previous years. Although measles is no longer considered endemic in the United States, global travel and the endemic presence of measles in other countries continue to produce cases in the United States. In 2006, a lab-confirmed measles case was identified in LAC and rash onset occurred within 18 days of traveling outside of the United States. Due to a personal beliefs exemption, the case had never received any MMR vaccine.

In light of this resurgence in cases, controlling the incidence of ten unique vaccine-preventable diseases continues to be a challenge for the LAC Immunization Program and requires a multi-level plan of attack. Efforts are already in place to increase the usage of vaccines among adults and adolescents (i.e., Tdap, MMR, varicella, hepatitis B), while maintaining high childhood vaccine coverage levels. In addition, a sensitive surveillance and case management system specific to the epidemiology of each disease is required. For example, by employing a multi-lingual enhanced case management system, nearly all infants (98% and 97%, respectively) exposed to hepatitis B in 2006 received dose one of the hepatitis B vaccine and HBIG within 24 hours of birth. The future success of controlling vaccine-preventable disease incidence in LAC will depend on high vaccine coverage levels among children, adolescents, and adults along with the use of sophisticated and sensitive surveillance systems for each disease.

Hospital Outbreaks and Outreach

The Hospital Outreach Unit (HOU) is an integral component of the public health link to infection control professionals and community healthcare agencies. The unit incorporates five liaison public health nurses (LPHN), two program specialist PHNs, an epidemiology analyst, and a medical epidemiologiest who interface with infection control professionals at 104 licensed acute care hospitals educating them on disease reporting and promoting hospital implementation of web-based and emergency department surveillance to enhance early detection of potential critical situation. The team identifies and responds to potential risks and threats during hospital outbreaks and assist with investigations. The scope has expanded to include non-hospital healthcare settings, such as large clinics and jail medical services.

As in past years, the most common cause of reported hospital outbreaks was scabies. This was followed in number by outbreaks of *Acinetobacter baumanii* and methicillin-resistant *Staphylococcus areaus* (MRSA) infections. In 2006, the most common outbreaks in skilled nursing and other sub-acute health facilities were due to gastroenteritis and scabies, similar to previous years. For the first time the new highly toxigenic strain of *Clostridium difficile* (B1/NAP1) was confirmed in Los Angeles County. Selected hospital outbreak investigation summaries are available in ACDC's 2006 Morbidity Report.

Healthcare associated infections (HAI) have generated a great deal of attention locally and across the United States for several years. In response, California approved Senate Bill 739 in 2006, which imposes reporting requirements and establishes the California HAI Advisory Committee to monitor and prevent hospital-acquired infections. The HOU is working with the California Department of Public Health as a part of this advisory committee to make recommendations related to reporting of hospital acquired infections, use of national guidelines, and public reporting of process measures for preventing the spread of HAI.

Prevention and control of HAI must include collaborating with subacute nursing facilities (SNF) and other healthcare facilities. In 2005 and 2006, ACDC initiated a SNF needs assessment to assess general communicable disease reporting knowledge, infection control practices, identify knowledge gaps and elicit training needs. Based on these findings, ACDC is exploring collaboration with LACDPH Health Facilities Inspection Division and the Hospital Association of Southern California (HASC) on the best way to address training needs.

The HOU continues to work with governmental and specialty organizations to standardize guidelines for the cleaning and disinfection of semi-critical devices. In 2006, HOU investigated two outbreaks that implicated improper and/or inconsistent disinfection and cleaning practices of reusable medical devices. The first outbreak involved an adult ICU and *Escherichia coli* found on the transesophageal echocardiography (TEE) probe, a flexible endoscope used to visualize the heart. The second outbreak involved a neonatal ICU where *Pseudomonas aeruginosa* was discovered on a laryngoscope blade. In both outbreaks, instrument cleaning was in violation of the facility's established cleaning and disinfection policy (see 2006 Special Studies Report for detailed article).

Bioterrorism Surveillance, Preparedness and Response

In 2001, the mandated list of reportable diseases was modified to provide greater emphasis on diseases deemed likely indicators of bioterrorism activity (i.e. anthrax, botulism, brucellosis, plague, smallpox, tularemia, and viral hemorrhagic fevers). Education to strengthen awareness and understanding of disease and outbreak reporting continued throughout 2006, and ACDC provided tailored educational materials related to disease reporting to healthcare providers in LAC.

Bioterrorism Preparedness

In 2006, BT-related surveillance projects were further expanded and integrated into public health. These systems were shown to be useful indicators of morbidity and mortality.

The achievements of ACDC's bioterrorism surveillance and preparedness sections during 2006 were the continued integration of early detection system activities into routine public health operations. Emergency department syndromic surveillance, which includes detecting major trends from baseline patterns of illness that may potentially identify bioterrorist-related activity, was continued with the addition of several local hospitals. Our syndromic surveillance proved capable of detecting patterns of illness and community outbreaks and complemented traditional disease surveillance activities. Volume data from the ReddiNet® system for emergency department visits during influenza season strongly correlated with virologic test results. Nurse call line, coroner data, and over-the-counter medications data also complement our early event detection system.

vCMR (Visual Confidential Morbidity Report) is an advanced electronic reporting system for all communicable diseases. It manages the life-cycle of a disease incident from the initial date of onset to the final resolution. The system has been fully operational since May 2000. It features a disease, outbreak, foodborne illness, and community reporting module used by infection control practitioners as well as an extensive electronic laboratory reporting module.

To align with CDC-sponsored initiatives such as the Public Health Information Network (PHIN) and National Electronic Disease Surveillance System (NEDSS), the vCMR custom development solution was scaled up to support broader integration of disease reporting and expansion of standards-based electronic data exchange capabilities. In September 2005, vCMR was converted to a full web-based application using Microsoft.NET technology.

ELR (Electronic Laboratory Reporting): Automated electronic reporting of communicable diseases from laboratories to public health has been shown to yield more complete and rapid reporting of disease. Results are sent to public health as soon as they are available rather than days later. LAC began receiving ELR in 2002, and since early 2006 have pursued efforts to recruit and implement many additional public and private labs. We currently have live feeds from six (6) laboratories representing 10 hospitals and two independent labs. We have six labs currently in testing and a dozen more poised to begin testing in 2007. Establishing electronic lab reporting is a very time consuming process and on average takes roughly 8 to 12 months to implement.

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OVERVIEW

ACUTE COMMUNICABLE DISEASE CONTROL PROGRAM ANNUAL MORBIDITY REPORT **2006**

PURPOSE

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

1. summarize annual morbidity from several acute communicable diseases occurring in Los Angeles County (LAC);
2. assess the effectiveness of established communicable disease control programs;
3. identify patterns of disease as a means of directing future disease prevention efforts;
4. identify limitations of the data used for the above purposes and to identify means of improving that data; and
5. serve as a resource for medical and public health authorities at county, state and national levels.

Note: The 2006 ACDC Annual Morbidity Report does not include information on tuberculosis, sexually transmitted diseases, or HIV and AIDS. Information regarding these diseases is available from their respective departments (see the LAC Public Health website for more information at lapublichealth.org/phcommon/public/unitinfo/unitdirlist.cfm?ou=ph).

LAC DEMOGRAPHIC DATA

LAC population estimates used for this report are created by the Population Estimates and Projections System (PEPS) provided to the LAC DHS, Public Health by Urban Research. The LAC population is based on both estimates and projections that are adjusted when real relevant numbers become available (e.g., DMV records, Voters' registry, school enrollment and immigration records etc.).

National and California state counts of reportable diseases were obtained from the Centers for Disease Control and Prevention (CDC) Final 2006 Reports of Nationally Notifiable Infectious Diseases.[1] This report also includes US Census population estimates—these were used to calculate national and California rates of disease. According to that report, the population of the US in 2006 was 296,410,000 and the population of California was 36,132,000.

Long Beach and Pasadena are separate reporting jurisdictions, as recognized by the California Department of Health Services, 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 from LAC morbidity data, and their populations subtracted from LAC population data. Exceptions to this rule are noted in the text when they occur.

1. CDC. Notice to Readers: Final 2006 reports of nationally notifiable infectious diseases. MMWR 2007; 56(33):853–863. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/mm5633a4.htm

Table A. Los Angles County* population by year, 2001–2006

Year	Population	% change
2001	9,122,861	
2002	9,253,109	1.4%
2003	9,398,128	1.6%
2004	9,535,937	1.5%
2005	9,582,956	0.5%
2006	9,644,738	0.6%

* Does not include cities of Pasadena and Long Beach.

Table B. Los Angles County* population by age group, 2006

Age (in years)	Population	%
<1	144,825	1.5%
1–4	580,257	6.0%
5–14	1,474,646	15.3%
15–34	2,791,126	28.9%
35–44	1,506,357	15.6%
45–54	1,299,772	13.5%
55–64	868,327	9.0%
65+	979,428	10.2%
Total	**9,644,738**	**100.0%**

* Does not include cities of Pasadena and Long Beach.

Table C. Los Angles County* population by sex, 2006

Sex	Population	%
Male	4,771,987	49.5%
Female	4,872,751	50.5%
Total	**9,644,738**	**100.0%**

* Does not include cities of Pasadena and Long Beach.

Table D. Los Angles County* population by race, 2006

Race	Population	%
Asian	1,270,774	13.2%
Black	843,479	8.8%
Latino	4,624,005	47.9%
White	2,877,851	29.8%
Other**	28,629	0.3%
Total	**9,644,738**	**100.0%**

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

Table E. Los Angles County* population by health district and SPA, 2006

Health District	Population
SPA1	**347,823**
Antelope valley	347,823
SPA 2	**2,146,515**
East Valley	457,254
Glendale	353,559
San Fernando	460,426
West Valley	875,276
SPA 3	**1,720,297**
Alhambra	358,154
El Monte	477,775
Foothill	314,365
Pomona	570,003
SPA 4	**1,260,196**
Central	370,009
Hollywood Wilshire	540,747
Northeast	349,440
SPA 5	**636,309**
West	636,309
SPA 6	**1,041,685**
Compton	292,780
South	187,713
Southeast	179,218
Southwest	381,974
SPA 7	**1,379,540**
Bellflower	369,513
East Los Angeles	225,069
San Antonio	450,428
Whittier	334,530
SPA 8	**1,112,373**
Inglewood	435,627
Harbor	209,567
Torrance	467,179
Total	**9,644,738**

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

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, kindergarten, boarding school, or preschool knowing of a **case or suspected case** 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 **outbreak** or **unusual incidence** of infectious disease and any **unusual disease** 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).

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 physicians in an effort to identify all cases of a given disease.

DATA LIMITATIONS

This report should be interpreted in light of the following 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., Latino versus non-Latino) 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 case definition for infectious diseases under public surveillance[2] is used with some exceptions as noted in the text of the 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.

2 CDC. Case Definitions for Infectious Conditions under Public Health Surveillance," MMWR 1997; 46(RR10):1-55. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/00047449.htm

4. 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. Population Estimates
   Estimates of the LAC population are subject to many errors. 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. This may be especially true for many of the diseases common in Latino and Asian populations. Therefore, some disease rates more accurately reflect the place of diagnosis than the location where an infection was acquired.

7. Health Districts and Service Planning Areas
   Since 1999, Los Angeles County 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).

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.
   - **American Indian** – person having origins in any of the original peoples of North America and who maintain cultural identification through tribal affiliation or community recognition.
   - **Black** – person having origins in any of the black racial groups of Africa.
   - **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.

STANDARD REPORT FORMAT

1. Crude data.
   - **Number of Cases**: For most diseases, this number reflects new cases of the disease with an onset in 2006. If the onset was unknown, the date of diagnosis was used.
   - **Annual Incidence Rates in LAC**: Number of new cases in 2006 divided by LAC census population (minus Long Beach and Pasadena) multiplied by 100,000.
   - **Annual Incidence Rates in the US and California**: 2006 incidence rates for the US and California were taken from the previously cited CDC publication, Morbidity and Mortality Weekly Report (MMWR). The MMWR records diseases by date of report rather than date of onset.
   - **Mean Age at Onset**: Arithmetic 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 2006. For cases under one year of age, less than one (<1) was used.

2. Etiology
   This includes the causative agent, mode of spread, common symptoms, potential severe outcomes, susceptible groups, and vaccine-preventability.

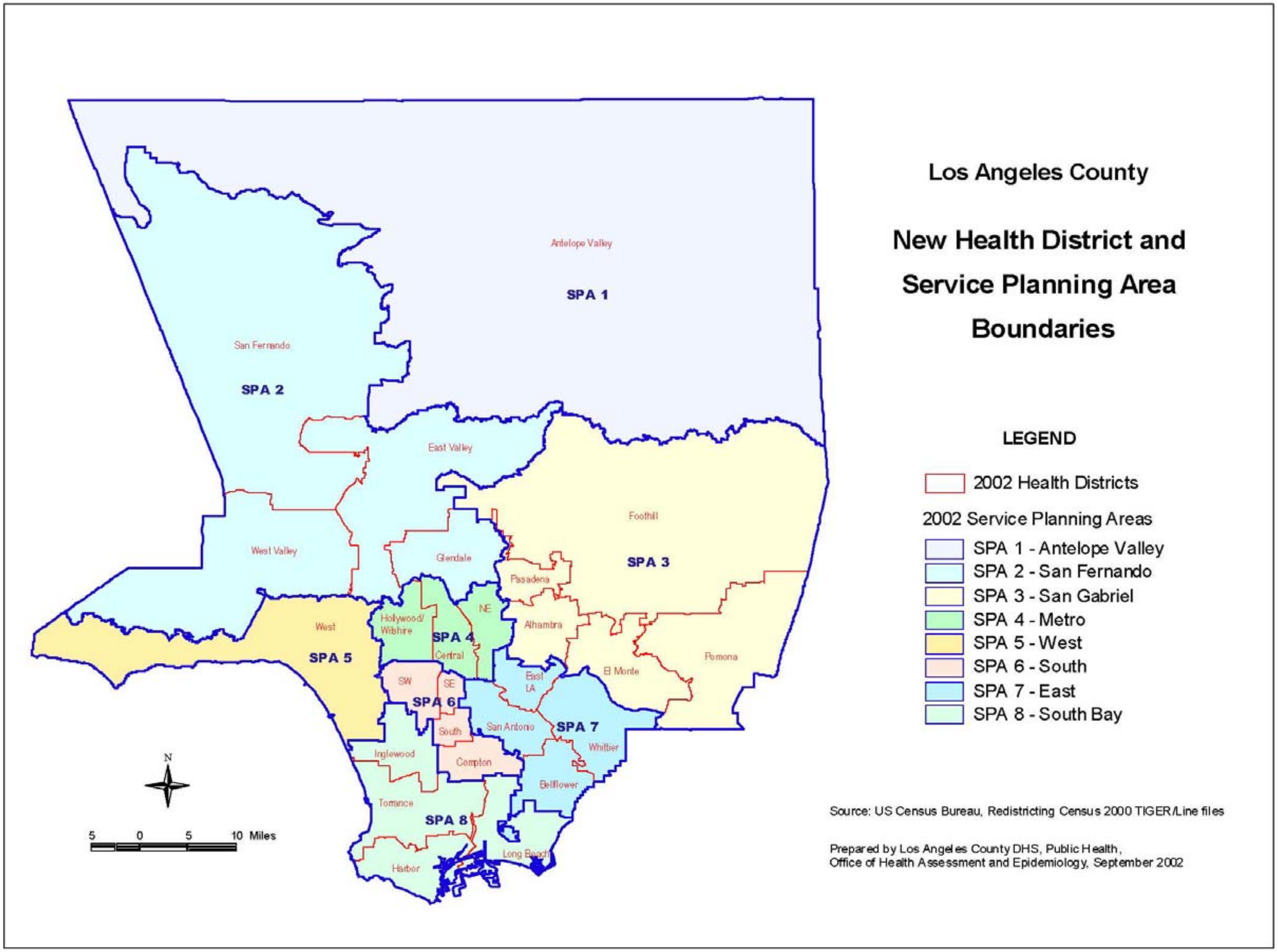
3. Disease Abstract
   This provides a synopsis or the highlights of disease activity in 2006.

4. Stratified Data
   - **Trends**: Any trends in case characteristics during recent years.
   - **Seasonality**: Number of cases that occurred during each month of 2006.
   - **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 five 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" rarely refers to the site of disease acquisition. Age-adjusted rates by location are presented for some diseases.

5. Prevention
   If applicable, includes a description of county programs and other measures that address the disease.

6. Comments
   Describes miscellaneous information not fitting easily into above categories, as well as elaboration of some findings of interest.

7. Additional Resources
   Provides agencies, phone numbers, websites, and other resources on the subject.


Los Angeles County
New Health District and
Service Planning Area
Boundaries
LEGEND
2002 Health Districts
2002 Service Planning Areas
SPA 1 - Antelope Valley
SPA 2 - San Fernando
SPA 3 - San Gabriel
SPA 4 - Metro
SPA 5 - West
SPA 6 - South
SPA 7 - East
SPA 8 - South Bay
Source: US Census Bureau, Redistricting Census 2000 TIGER/Line files
Prepared by Los Angeles County DHS, Public Health,
Office of Health Assessment and Epidemiology, September 2002
Antelope Valley
SPA 1
San Fernando
SPA 2
East Valley
West Valley
Glendale
Foothill
SPA 3
Pasadena
Alhambra
El Monte
Pomona
Hollywood/
Wilshire
SPA 4
NE
Central
West
SPA 5
SW
SE
SPA 6
South
East
LA
San Antonio
SPA 7
Whittier
Inglewood
Compton
Bellflower
Torrance
SPA 8
Harbor
Long Beach
N
5 0 5 10 Miles

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

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	**IgG**	Immunoglobulin G
CDC	Centers for Disease Control and Prevention	**IgM**	Immunoglobulin M
CDHS	California Department of Health Services	**LAC**	Los Angeles County
CMR	Confidential morbidity report	**MMR**	Mumps-Measles-Rubella vaccine
CSF	Cerebral spinal fluid	**MMWR**	Morbidity and Mortality Weekly Report
CSTE	Council of State and Territorial Epidemiologists	**MSM**	Men who have sex with men
DHS	Department of Health Services	**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)

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	**TO**	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

TABLES OF NOTIFIABLE DISEASES

Table G. Reported Cases of Selected Notifiable Diseases by Year of Onset
Los Angeles County, 2001-2006

Disease	Year of Onset 2001	2002	2003	2004	2005	2006	Previous 5-year Average	5-Yr 95% upper Limit[a]
Amebiasis	139	102	121	114	114	94	118	142
Botulism	2	2	0	3	8	2	3	8
Brucellosis	9	11	7	4	8	5	8	12
Campylobacteriosis	1141	1067	1100	884	725	775	983	1290
Cholera	0	0	1	0	0	0	0	1
Coccidioidomycosis	68	76	73	133	214	196	113	222
Cryptosporidiosis	77	62	71	56	45	48	62	84
Cysticercosis	37	18	12	8	15	11	18	38
Dengue	5	7	0	5	10	2	5	12
E. *coli* O157:H7	31	31	27	18	13	12	24	38
Encephalitis	41	61	38	133	72	46	69	136
Foodborne outbreaks	48	29	25	40	32	49	35	51
Giardiasis	446	441	401	320	313	376	384	497
Haemophilus influenzae type b	5	4	0	2	3	5	3	6
Hansen's Disease (Leprosy)	2	11	9	9	2	2	7	14
Hepatitis A	542	438	374	321	480	364	431	583
Hepatitis B	44	32	73	72	57	62	56	87
Hepatitis C	1	3	0	5	3	4	2	6
Hepatitis unspecified [b]	1	0	1	0	4	7	1	4
Kawasaki syndrome [b]	33	34	35	42	56	75	40	57
Legionellosis	18	25	21	15	31	24	22	33
Listeriosis, nonperinatal	27	14	17	21	25	25	21	30
Listeriosis, perinatal [b]	3	7	3	6	3	12	4	8
Lyme disease [b]	5	8	6	0	7	16	5	11
Malaria	46	38	60	51	45	33	48	62
Measles	8	0	0	1	0	1	2	8
Meningitis, viral	378	466	899	807	527	373	615	1011
Meningococcal infections	58	46	32	28	37	46	40	61
Mumps	17	16	10	5	10	10	12	20
Pertussis	103	172	130	156	439	150	200	439
Psittacosis	1	0	0	0	0	1	0	1
Q-fever	1	4	0	4	0	1	2	5
Relapsing fever [b]	0	1	0	0	0	2	0	1
Rheumatic fever, acute	6	0	0	1	0	0	1	6
Rubella	0	0	0	0	1	0	0	1
Salmonellosis	1006	956	995	1205	1085	1217	1050	1223
Shigellosis	684	974	669	625	710	524	732	975
Strongyloidiasis	0	0	0	0	0	0	0	0
Tetanus [b]	2	2	1	2	0	4	1	3
Trichinosis [b]	0	0	0	0	0	1	0	0
Tularemia	0	0	1	0	0	0	0	1
Typhoid fever, case	17	33	16	13	12	17	18	33
Typhoid fever, carrier	1	6	2	3	4	3	3	7
Typhus fever	8	11	12	8	9	10	10	13
Vibrio	15	14	13	26	14	18	16	26

[a] The normal distribution assumption may not apply to some rare diseases.

[b] 2006 data over 95% upper limit.

**Table H. Annual Incidence Rates of Selected Notifiable Diseases by Year of Onset
Los Angeles County, 2001-2006**

Disease	Annual Incidence Rate (Cases per 100,000)[b]					
	2001	2002	2003	2004	2005	2006
Amebiasis	1.52	1.10	1.29	1.20	1.19	0.97
Botulism	0.02	0.02	-	0.03	0.08	0.02
Brucellosis	0.10	0.12	0.07	0.04	0.08	0.05
Campylobacteriosis	12.50	11.50	11.70	9.27	7.57	8.04
Cholera	-	-	0.01	-	-	-
Coccidioidomycosis	0.75	0.82	0.78	1.39	2.23	2.03
Cryptosporidiosis	0.84	0.67	0.75	0.59	0.47	0.50
Cysticercosis	0.41	0.19	0.13	0.08	0.16	0.11
Dengue	0.05	0.08	-	0.05	0.10	0.02
E. *coli* O157:H7	0.34	0.33	0.29	0.19	0.14	0.12
Encephalitis	0.45	0.66	0.40	1.39	0.75	0.48
Giardiasis	4.89	4.75	4.26	3.36	3.27	3.90
Haemophilus influenzae type b	0.05	0.04	-	0.02	0.03	0.05
Hansen’s Disease (Leprosy)	0.02	0.12	0.10	0.09	0.02	0.02
Hepatitis A	5.94	4.72	3.98	3.37	5.01	3.77
Hepatitis B	0.48	0.34	0.78	0.76	0.59	0.64
Hepatitis C	0.01	0.03	-	0.05	0.03	0.04
Hepatitis unspecified	0.01	0.00	0.01	-	0.04	0.07
Kawasaki syndrome	0.36	0.37	0.37	0.44	0.58	0.78
Legionellosis	0.20	0.27	0.22	0.16	0.32	0.25
Listeriosis, nonperinatal	0.30	0.15	0.18	0.22	0.26	0.26
Listeriosis, perinatal[a]	2.05	4.96	2.12	4.25	2.14	8.47
Lyme disease	0.05	0.09	0.06	-	0.07	0.17
Malaria	0.50	0.41	0.64	0.53	0.47	0.34
Measles	0.09	-	-	0.01	-	0.01
Meningitis, viral	4.14	5.02	9.56	8.46	5.50	3.87
Meningococcal infections	0.64	0.50	0.34	0.29	0.39	0.48
Mumps	0.19	0.17	0.11	0.05	0.10	0.10
Pertussis	1.13	1.85	1.38	1.64	4.58	1.56
Psittacosis	0.01	-	-	-	-	0.01
Q-fever	0.01	0.04	-	0.04	-	0.01
Relapsing fever	-	0.01	-	-	-	0.02
Rheumatic fever, acute	0.07	-	-	0.01	-	-
Rubella	-	-	-	-	0.01	-
Salmonellosis	11.02	10.30	10.58	12.64	11.33	12.62
Shigellosis	7.50	10.50	7.11	6.55	7.41	5.43
Strongyloidiasis	-	-	-	-	-	-
Tetanus	0.02	0.02	0.01	0.02	-	0.04
Trichinosis	-	-	-	-	-	0.01
Tularemia	-	-	0.01	-	-	-
Typhoid fever, case	0.19	0.36	0.17	0.14	0.13	0.18
Typhoid fever, carrier	0.01	0.06	0.02	0.03	0.04	0.03
Typhus fever	0.09	0.12	0.13	0.08	0.09	0.10
Vibrio	0.16	0.15	0.14	0.27	0.15	0.19

[a] Rates for perinatal listeriosis were calculated as cases per 100,000 live births.

[b] Rates of disease based on less than 19 cases or events are considered "unreliable." A zero rate made from no events is especially hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

Table I. Five –Year Average of Notifiable Diseases by Month of Onset
Los Angeles County, 2002-2006

Disease	Jan	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	Nov	Dec	Total
Amebiasis	6.2	7.0	7.6	6.6	8.4	7.8	8.4	9.6	8.6	5.2	7.4	10.0	108.8
Botulism	0.0	0.4	0.4	0.2	0.0	0.4	0.2	0.2	0.2	0.0	0.6	0.2	2.8
Brucellosis	0.8	0.8	0.8	0.4	0.2	0.6	1.2	0.8	0.4	0.6	0.4	0.0	7.0
Campylobacteriosis	70.8	49.4	58.2	65.6	86.6	92.0	105.4	98.6	86.2	71.6	72.4	50.0	910.0
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.2
Coccidioidomycosis	11.2	8.0	9.2	9.2	9.6	8.8	10.4	13.6	15.4	12.2	13.0	9.4	139.4
Cryptosporidiosis	4.6	3.2	3.2	4.2	3.8	4.4	5.0	9.2	6.2	4.0	4.2	3.2	56.4
Cysticercosis	1.2	1.0	2.0	1.4	1.6	1.0	1.2	1.0	1.0	0.6	0.2	0.2	12.8
Dengue	0.0	0.4	0.0	0.0	0.0	0.4	1.0	1.2	0.8	0.4	0.0	0.0	4.4
E. *coli* O157:H7	1.0	0.6	0.8	1.0	1.2	1.6	3.6	4.2	2.8	2.0	0.8	0.4	20.0
Encephalitis	3.0	3.8	5.4	4.4	4.6	4.6	8.4	12.4	9.2	4.0	3.8	3.4	69.8
Giardiasis	27.2	17.8	28.2	25.6	26.2	27.8	35.2	36.2	35.0	30.4	26.0	22.8	369.4
Haemophilus influenzae type b	0.6	0.4	0.4	0.0	0.0	0.2	0.0	0.2	0.0	0.4	0.2	0.4	2.8
Hansen's Disease (Leprosy)[a]	-	-	-	-	-	-	-	-	-	-	-	-	-
Hepatitis A	45.0	35.4	27.2	22.0	26.6	20.0	24.6	28.4	36.6	38.6	40.0	32.4	395.4
Hepatitis B	6.4	7.6	5.6	6.2	7.2	5.8	4.0	4.4	2.8	6.2	6.8	6.8	70.8
Hepatitis C	0.6	0.6	0.6	0.4	0.4	0.6	0.6	0.4	0.4	0.8	0.2	0.8	7.2
Hepatitis unspecified	0.2	0.2	0.0	0.2	0.0	0.0	0.0	0.2	0.0	0.2	0.0	0.2	2.4
Kawasaki syndrome	6.4	6.0	4.8	3.6	3.8	5.0	2.6	3.6	3.0	2.2	4.0	3.0	48.0
Legionellosis	1.6	1.4	1.8	1.2	2.6	2.4	1.4	1.2	0.4	2.8	3.8	1.2	23.2
Listeriosis, nonperinatal	0.8	0.8	1.4	1.6	1.4	1.4	3.0	3.2	2.6	1.4	0.6	1.6	20.4
Listeriosis, perinatal	0.2	0.0	0.4	0.4	0.6	0.4	0.8	1.2	1.0	1.0	0.2	0.0	6.2
Lyme disease	0.2	0.2	0.0	0.2	0.2	1.8	1.6	0.8	0.4	0.4	0.0	0.0	5.8
Malaria[a]	-	-	-	-	-	-	-	-	-	-	-	-	-
Measles	0.2	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.4
Meningitis, viral	21.6	19.6	22.4	26.2	31.2	43.6	84.4	111.4	84.0	50.4	36.4	24.0	614.6
Meningococcal infections	6.2	4.8	3.0	4.6	2.2	2.6	2.2	1.8	1.4	2.6	2.8	3.4	37.8
Mumps	0.4	1.4	1.0	0.8	0.4	0.6	1.6	1.2	0.6	1.0	0.6	0.6	10.2
Pertussis	15.6	11.6	11.8	14.0	18.0	16.2	22.0	26.4	22.6	20.4	14.4	16.4	209.4
Psittacosis	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2
Q-fever	0.4	0.4	0.0	0.0	0.2	0.2	0.2	0.4	0.0	0.0	0.0	0.0	1.8
Relapsing fever	0.0	0.0	0.0	0.0	0.0	0.0	0.6	0.0	0.0	0.0	0.0	0.0	0.6
Rheumatic fever, acute	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.2
Rubella	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2
Salmonellosis	68.8	52.0	64.0	72.2	95.0	99.0	132.0	125.8	116.6	97.6	74.0	60.8	1091.4
Shigellosis	48.2	27.2	30.2	23.6	27.8	45.2	80.0	113.8	107.6	92.4	54.4	46.0	700.4
Strongyloidiasis	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
Tetanus	0.4	0.2	0.0	0.2	0.0	0.2	0.0	0.2	0.2	0.4	0.0	0.0	1.8
Trichinosis	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2
Tularemia	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.2
Typhoid fever, case	1.0	1.4	1.4	0.8	1.0	2.4	2.2	2.8	2.8	1.2	0.4	0.8	18.2
Typhoid fever, carrier	0.0	0.0	0.8	0.0	0.4	0.6	0.8	0.2	0.2	0.0	0.2	0.4	3.6
Typhus fever	0.6	0.0	0.0	0.2	1.0	1.0	1.4	1.2	1.4	1.2	0.8	0.4	9.2
Vibrio	0.2	0.4	0.6	0.4	1.2	1.8	4.0	2.6	1.4	1.8	1.6	0.4	17.0

[a] Not applicable.

Table J. Number of Cases of Selected Notifiable Diseases by Age Group
Los Angeles County, 2006

Disease	<1	1-4	5-14	15-34	35-44	45-54	55-64	65+	Total[a]
Amebiasis	0	0	5	28	26	18	9	8	94
Botulism	0	0	0	0	0	1	1	0	2
Brucellosis	0	0	0	4	0	1	0	0	5
Campylobacteriosis	21	91	97	207	105	81	68	105	775
Cholera	0	0	0	0	0	0	0	0	0
Coccidioidomycosis	1	1	3	51	30	42	32	36	196
Cryptosporidiosis	0	1	4	7	22	5	6	3	48
Cysticercosis	0	0	0	6	2	0	3	0	11
Dengue	0	0	0	1	0	1	0	0	2
E. *coli* O157:H7	0	5	3	4	0	0	0	0	12
Encephalitis	2	8	8	15	3	4	1	5	46
Giardiasis	0	47	66	105	66	47	29	15	376
Haemophilus influenzae type b	2	0	0	1	0	1	0	1	5
Hansen's Disease (Leprosy)	0	0	0	1	0	1	0	0	2
Hepatitis A	0	5	20	114	83	73	33	36	364
Hepatitis B	0	0	0	20	21	15	3	3	62
Hepatitis C	0	0	0	0	2	0	1	1	4
Hepatitis unspecified	0	0	1	2	1	1	0	1	7
Kawasaki syndrome	18	50	7	0	0	0	0	0	75
Legionellosis	0	0	0	1	2	2	5	14	24
Listeriosis, nonperinatal	0	0	0	2	1	4	6	12	25
Listeriosis, perinatal[b]	0	0	0	8	3	0	0	0	12
Lyme disease	0	0	3	7	2	2	1	1	16
Malaria	0	2	2	8	7	11	1	2	33
Measles	0	1	0	0	0	0	0	0	1
Meningitis, viral	71	14	47	111	53	42	23	10	373
Meningococcal infections	4	5	8	9	2	3	7	8	46
Mumps	0	1	2	2	1	3	1	0	10
Pertussis	58	14	33	21	8	7	6	3	150
Psittacosis	0	0	0	0	0	0	0	1	1
Q-fever	0	0	0	0	0	0	1	0	1
Relapsing fever	0	0	0	1	0	0	1	0	2
Rheumatic fever, acute	0	0	0	0	0	0	0	0	0
Rubella	0	0	0	0	0	0	0	0	0
Salmonellosis	100	221	208	251	105	112	80	140	1217
Shigellosis	5	118	134	111	71	39	17	29	524
Strongyloidiasis	0	0	0	0	0	0	0	0	0
Tetanus	0	0	0	1	1	0	1	1	4
Trichinosis	0	0	0	0	0	0	0	1	1
Tularemia	0	0	0	0	0	0	0	0	0
Typhoid fever, case	0	2	5	8	1	1	0	0	17
Typhoid fever, carrier	0	0	1	0	1	0	1	0	3
Typhus fever	0	0	1	1	5	0	1	2	10
Vibrio	0	0	1	5	3	3	3	3	18

[a] Totals include cases with unknown age.

[b] Mother's age.

Table K. Incidence Rates of Selected Notifiable Diseases by Age Group
Los Angeles County, 2006

Disease	Age-group Rates (Cases per 100,000)[b]							
	<1	1-4	5-14	15-34	35-44	45-54	55-64	65+
Amebiasis	-	-	0.3	1.0	1.7	1.4	1.0	0.8
Botulism	-	-	-	-	-	0.1	0.1	-
Brucellosis	-	-	-	0.1	-	0.1	-	-
Campylobacteriosis	14.5	15.7	6.6	7.4	7.0	6.2	7.8	10.7
Cholera	-	-	-	-	-	-	-	-
Coccidioidomycosis	0.7	0.2	0.2	1.8	2.0	3.2	3.7	3.7
Cryptosporidiosis	-	0.2	0.3	0.3	1.5	0.4	0.7	0.3
Cysticercosis	-	-	-	0.2	0.1	-	0.3	-
Dengue	-	-	-	-	-	0.1	-	-
E. *coli* O157:H7	-	0.9	0.2	0.1	-	-	-	-
Encephalitis	1.4	1.4	0.5	0.5	0.2	0.3	0.1	0.5
Giardiasis	-	8.1	4.5	3.8	4.4	3.6	3.3	1.5
Haemophilus influenzae type b	1.4	-	-	-	-	0.1	-	0.1
Hansen's Disease (Leprosy)	-	-	-	-	-	0.1	-	-
Hepatitis A	-	0.9	1.4	4.1	5.5	5.6	3.8	3.7
Hepatitis B	-	-	-	0.7	1.4	1.2	0.3	0.3
Hepatitis C	-	-	-	-	0.1	-	0.1	0.1
Hepatitis unspecified	-	-	0.1	0.1	0.1	0.1	-	0.1
Kawasaki syndrome	12.4	8.6	0.5	-	-	-	-	-
Legionellosis	-	-	-	-	0.1	0.2	0.6	1.4
Listeriosis, nonperinatal	-	-	-	0.1	0.1	0.3	0.7	1.2
Listeriosis, perinatal[a]	-	-	-	6.9	11.6	-	-	-
Lyme disease	-	-	0.2	0.3	0.1	0.2	0.1	0.1
Malaria	-	0.3	0.1	0.3	0.5	0.8	0.1	0.2
Measles	-	0.2	-	-	-	-	-	-
Meningitis, viral	49.0	2.4	3.2	4.0	3.5	3.2	2.6	1.0
Meningococcal infections	2.8	0.9	0.5	0.3	0.1	0.2	0.8	0.8
Mumps	-	0.2	0.1	0.1	0.1	0.2	0.1	-
Pertussis	40.4	2.4	2.2	0.8	0.5	0.5	0.7	0.3
Psittacosis	-	-	-	-	-	-	-	0.1
Q-fever	-	-	-	-	-	-	0.1	-
Relapsing fever	-	-	-	-	-	-	0.1	-
Rheumatic fever, acute	-	-	-	-	-	-	-	-
Rubella	-	-	-	-	-	-	-	-
Salmonellosis	69.0	38.1	14.1	9.0	7.0	8.6	9.2	14.3
Shigellosis	3.5	20.3	9.1	4.0	4.7	3.0	2.0	3.0
Strongyloidiasis	-	-	-	-	-	-	-	-
Tetanus	-	-	-	-	0.1	-	0.1	0.1
Trichinosis	-	-	-	-	-	-	-	0.1
Tularemia	-	-	-	-	-	-	-	-
Typhoid fever, case	-	0.3	0.3	0.3	0.1	0.1	-	-
Typhoid fever, carrier	-	-	0.1	-	0.1	-	0.1	-
Typhus fever	-	-	0.1	-	0.3	-	0.1	0.2
Vibrio	-	-	0.1	0.2	0.2	0.2	0.3	0.3

[a] Rates for perinatal listeriosis were calculated as cases per 100,000 live births.

[b] Rates of disease based on less than 19 cases or events are considered "unreliable." A zero rate made from no events is especially hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

Table L. Number of Cases of Selected Notifiable Diseases by Race/Ethnicity Los Angeles County, 2006

Disease	Asian	Black	Hispanic	White	Other[a]	Unknown
Amebiasis	10	2	32	39	2	2
Botulism	0	0	2	0	0	0
Brucellosis	0	0	5	0	0	0
Campylobacteriosis	92	34	336	302	4	6
Cholera	0	0	0	0	0	0
Coccidioidomycosis	15	27	68	75	3	2
Cryptosporidiosis	0	8	20	16	2	2
Cysticercosis	0	0	9	2	0	0
Dengue	0	0	1	1	0	0
E. *coli* O157:H7	1	0	3	8	0	0
Encephalitis	4	8	20	12	1	1
Giardiasis	36	26	137	149	7	5
Haemophilus influenzae type b	0	0	3	1	0	1
Hansen's Disease (Leprosy)	0	0	2	0	0	0
Hepatitis A	25	64	124	125	1	16
Hepatitis B	10	4	26	21	0	1
Hepatitis C	0	0	2	2	0	0
Hepatitis unspecified	2	0	2	1	0	1
Kawasaki syndrome	25	8	28	11	3	0
Legionellosis	6	3	5	10	0	0
Listeriosis, nonperinatal	3	1	8	13	0	0
Listeriosis, perinatal[b]	1	3	7	1	0	0
Lyme disease	1	0	2	11	1	1
Malaria	5	22	1	5	0	0
Measles	1	0	0	0	0	0
Meningitis, viral	29	33	195	101	5	9
Meningococcal infections	2	3	28	13	0	0
Mumps	3	0	3	3	0	1
Pertussis	8	4	79	59	0	0
Psittacosis	0	0	1	0	0	0
Q-fever	0	0	1	0	0	0
Relapsing fever	0	0	0	2	0	0
Rheumatic fever, acute	0	0	0	0	0	0
Rubella	0	0	0	0	0	0
Salmonellosis	138	95	609	351	4	20
Shigellosis	23	42	356	99	1	3
Strongyloidiasis	0	0	0	0	0	0
Tetanus	1	0	2	1	0	0
Trichinosis	1	0	0	0	0	0
Tularemia	0	0	0	0	0	0
Typhoid fever, case	7	0	8	1	0	1
Typhoid fever, carrier	1	0	2	0	0	0
Typhus fever	1	0	3	6	0	0
Vibrio	2	0	4	12	0	0

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

[b] Mother's race.

Table M. Incidence Rates of Selected Notifiable Diseases by Race/Ethnicity Los Angeles County, 2006

Disease	Race/Ethnicity Rates (Cases per 100,000)[b]			
	Asian	Black	Hispanic	White
Amebiasis	0.8	0.2	0.7	1.4
Botulism	-	-	-	-
Brucellosis	-	-	0.1	-
Campylobacteriosis	7.2	4.0	7.3	10.5
Cholera	-	-	-	-
Coccidioidomycosis	1.2	3.2	1.5	2.6
Cryptosporidiosis	-	0.9	0.4	0.6
Cysticercosis	-	-	0.2	0.1
Dengue	-	-	-	-
E. *coli* O157:H7	0.1	-	0.1	0.3
Encephalitis	0.3	0.9	0.4	0.4
Giardiasis	2.8	3.1	3.0	5.2
Haemophilus influenzae type b	-	-	0.1	-
Hansen's Disease (Leprosy)	-	-	-	-
Hepatitis A	2.0	7.6	2.7	4.3
Hepatitis B	0.8	0.5	0.6	0.7
Hepatitis C	-	-	-	0.1
Hepatitis unspecified	0.2	-	-	-
Kawasaki syndrome	2.0	0.9	0.6	0.4
Legionellosis	0.5	0.4	0.1	0.3
Listeriosis, nonperinatal	0.2	0.1	0.2	0.5
Listeriosis, perinatal[a]	6.6	29.4	7.7	4.2
Lyme disease	0.1	-	-	0.4
Malaria	0.4	2.6	-	0.2
Measles	0.1	-	-	-
Meningitis, viral	2.3	3.9	4.2	3.5
Meningococcal infections	0.2	0.4	0.6	0.5
Mumps	0.2	-	0.1	0.1
Pertussis	0.6	0.5	1.7	2.1
Psittacosis	-	-	-	-
Q-fever	-	-	-	-
Relapsing fever	-	-	-	0.1
Rheumatic fever, acute	-	-	-	-
Rubella	-	-	-	-
Salmonellosis	10.8	11.3	13.2	12.2
Shigellosis	1.8	5.0	7.7	3.4
Strongyloidiasis	-	-	-	-
Tetanus	0.1	-	-	-
Trichinosis	0.1	-	-	-
Tularemia	-	-	-	-
Typhoid fever, case	0.5	-	0.2	-
Typhoid fever, carrier	0.1	-	-	-
Typhus fever	0.1	-	0.1	0.2
Vibrio	0.2	-	0.1	0.4

[a] Rates for perinatal listeriosis were calculated as cases per 100,000 live births.

[b] Rates of disease based on less than 19 cases or events are considered "unreliable." A zero rate made from no events is especially hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

Table N. Number of Cases and Annual Incidence Rate of Selected Notifiable Diseases by Sex Los Angeles County, 2006

Disease	Male		Female	
	Cases	Rate (Cases per 100,000)[b]	Cases	Rate (Cases per 100,000)[b]
Amebiasis	54	1.1	35	0.7
Botulism	2	0.0	0	-
Brucellosis	1	0.0	4	0.1
Campylobacteriosis	440	9.2	335	6.9
Cholera	0	-	0	-
Coccidioidomycosis	134	2.8	62	1.3
Cryptosporidiosis	30	0.6	18	0.4
Cysticercosis	5	0.1	6	0.1
Dengue	2	0.0	0	-
E. *coli* O157:H7	6	0.1	6	0.1
Encephalitis	23	0.5	23	0.5
Giardiasis	255	5.3	115	2.4
Haemophilus influenzae type b	3	0.1	2	0.0
Hansen's Disease (Leprosy)	1	0.0	0	-
Hepatitis A	241	5.1	123	2.5
Hepatitis B	49	1.0	13	0.3
Hepatitis C	3	0.1	1	0.0
Hepatitis unspecified	4	0.1	3	0.1
Kawasaki syndrome	38	0.8	37	0.8
Legionellosis	13	0.3	11	0.2
Listeriosis, nonperinatal	13	0.3	12	0.2
Listeriosis, perinatal[a]	7	9.6	4	5.8
Lyme disease	7	0.1	9	0.2
Malaria	22	0.5	11	0.2
Measles	0	-	1	0.0
Meningitis, viral	191	4.0	182	3.7
Meningococcal infections	24	0.5	22	0.5
Mumps	6	0.1	4	0.1
Pertussis	58	1.2	92	1.9
Psittacosis	0	-	1	0.0
Q-fever	1	0.0	0	-
Relapsing fever	2	0.0	0	-
Rheumatic fever, acute	0	-	0	-
Rubella	0	-	0	-
Salmonellosis	583	12.2	631	12.9
Shigellosis	275	5.8	249	5.1
Strongyloidiasis	0	-	0	-
Tetanus	2	0.0	2	0.0
Trichinosis	0	-	1	0.0
Tularemia	0	-	0	-
Typhoid fever, case	8	0.2	9	0.2
Typhoid fever, carrier	2	0.0	1	0.0
Typhus fever	7	0.1	3	0.1
Vibrio	10	0.2	8	0.2

[a] Rates for perinatal listeriosis were calculated as cases per 100,000 live births.

[b] Rates of disease based on less than 19 cases or events are considered "unreliable." A zero rate made from no events is especially hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

Table O-1. Selected Notifiable Diseases
SPA 1. Antelope Valley Area
Los Angeles County, 2006

Disease	Frequency	Rate (Cases per 100,000)[b]
	Antelope	Antelope
Amebiasis	2	0.6
Botulism	0	-
Brucellosis	0	-
Campylobacteriosis	25	7.2
Cholera	0	-
Coccidioidomycosis	67	19.3
Cryptosporidiosis	4	1.2
Cysticercosis	2	0.6
Dengue	0	-
E. *coli* O157:H7	0	-
Encephalitis	5	1.4
Giardiasis	11	3.2
Haemophilus influenzae type b	0	-
Hansen's Disease (Leprosy)	0	-
Hepatitis A	3	0.9
Hepatitis B	2	0.6
Hepatitis C	0	-
Hepatitis unspecified	0	-
Kawasaki syndrome	1	0.3
Legionellosis	0	-
Listeriosis, nonperinatal	0	-
Listeriosis, perinatal[a]	1	1.3
Lyme disease	0	-
Malaria	0	-
Measles	0	-
Meningitis, viral	45	12.9
Meningococcal infections	2	0.6
Mumps	0	-
Pertussis	12	3.5
Psittacosis	0	-
Q-fever	0	-
Relapsing fever	0	-
Rheumatic fever, acute	0	-
Rubella	0	-
Salmonellosis	33	9.5
Shigellosis	6	1.7
Strongyloidiasis	0	-
Tetanus	0	-
Trichinosis	0	-
Tularemia	0	-
Typhoid fever, case	0	-
Typhoid fever, carrier	0	-
Typhus fever	0	-
Vibrio	0	-

[a] Rates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.

[b] Rates of disease based on less than 19 cases or events are considered "unreliable." A zero rate made from no events is especially hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

Table O-2. Selected Notifiable Diseases
SPA 2. San Fernando Area
Los Angeles County, 2006

Disease	Frequency					Rate (Cases per 100,000)[b]				
	EV	GL	SF	WV	TOTAL	EV	GL	SF	WV	TOTAL
Amebiasis	13	16	2	8	39	2.8	4.5	0.4	0.9	1.8
Botulism	0	0	0	0	0	-	-	-	-	-
Brucellosis	0	0	1	0	1	-	-	0.2	-	0.0
Campylobacteriosis	36	36	73	72	217	7.9	10.2	15.9	8.2	10.1
Cholera	0	0	0	0	0	-	-	-	-	-
Coccidioidomycosis	1	5	32	19	57	0.2	1.4	7.0	2.2	2.7
Cryptosporidiosis	2	0	3	8	13	0.4	-	0.7	0.9	0.6
Cysticercosis	0	0	0	1	1	-	-	-	0.1	-
Dengue	0	0	1	0	1	-	-	0.2	-	0.0
E. *coli* O157:H7	0	3	2	1	6	-	0.8	0.4	0.1	0.3
Encephalitis	5	0	3	0	8	1.1	-	0.7	-	0.4
Giardiasis	24	37	28	35	124	5.2	10.5	6.1	4.0	5.8
Haemophilus influenzae type b	0	0	1	0	1	-	-	0.2	-	0.0
Hansen's Disease (Leprosy)	0	0	0	0	0	-	-	-	-	-
Hepatitis A	9	15	6	28	58	2.0	4.2	1.3	3.2	2.7
Hepatitis B	3	3	3	6	15	0.7	0.8	0.7	0.7	0.7
Hepatitis C	0	0	0	0	0	-	-	-	-	-
Hepatitis unspecified	0	0	0	0	0	-	-	-	-	-
Kawasaki syndrome	1	4	5	4	14	0.2	1.1	1.1	0.5	0.7
Legionellosis	0	0	1	2	3	-	-	0.2	0.2	0.1
Listeriosis, nonperinatal	0	4	0	3	7	-	1.1	-	0.3	0.3
Listeriosis, perinatal[a]	0	0	0	1	1	-	-	-	0.5	0.2
Lyme disease	1	1	1	3	6	0.2	0.3	0.2	0.3	0.3
Malaria	0	1	1	3	5	-	0.3	0.2	0.3	0.2
Measles	0	0	0	1	1	-	-	-	0.1	0.0
Meningitis, viral	13	13	20	26	72	2.8	3.7	4.3	3.0	3.4
Meningococcal infections	3	2	2	4	11	0.7	0.6	0.4	0.5	0.5
Mumps	0	2	0	2	4	-	0.6	-	0.2	0.2
Pertussis	6	4	10	12	32	1.3	1.1	2.2	1.4	1.5
Psittacosis	0	0	0	1	1	-	-	-	0.1	0.0
Q-fever	0	0	0	0	0	-	-	-	-	-
Relapsing fever	0	0	1	0	1	-	-	0.2	-	0.0
Rheumatic fever, acute	0	0	0	0	0	-	-	-	-	-
Rubella	0	0	0	0	0	-	-	-	-	-
Salmonellosis	43	44	71	112	270	9.4	12.4	15.4	12.8	12.6
Shigellosis	19	17	26	25	87	4.2	4.8	5.6	2.9	4.1
Strongyloidiasis	0	0	0	0	0	-	-	-	-	-
Tetanus	0	0	0	0	0	-	-	-	-	-
Trichinosis	0	0	0	0	0	-	-	-	-	-
Tularemia	0	0	0	0	0	-	-	-	-	-
Typhoid fever, case	0	1	0	2	3	-	0.3	-	0.2	0.1
Typhoid fever, carrier	0	0	0	0	0	-	-	-	-	-
Typhus fever	0	2	0	1	3	-	0.6	-	0.1	0.1
VIbrio	0	0	0	2	2	-	-	-	0.2	0.1

[a] Rates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.

[b] Rates of disease based on less than 19 cases or events are considered "unreliable." A zero rate made from no events is especially hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

Table O-3. Selected Notifiable Diseases
SPA 3. San Gabriel Area
Los Angeles County, 2006

Disease	Frequency					Rate (Cases per 100,000)[b]				
	AH	EM	FH	PO	TOTAL	AH	EM	FH	PO	TOTAL
Amebiasis	3	0	1	2	6	0.8	-	0.3	0.4	0.3
Botulism	0	1	0	0	1	-	0.2	-	-	0.1
Brucellosis	1	0	0	0	1	0.3	-	-	-	0.1
Campylobacteriosis	28	8	20	36	92	7.8	1.7	6.4	6.3	5.3
Cholera	0	0	0	0	0	-	-	-	-	-
Coccidioidomycosis	4	0	3	4	11	1.1	-	1.0	0.7	0.6
Cryptosporidiosis	1	0	1	1	3	0.3	-	0.3	0.2	0.2
Cysticercosis	0	5	1	0	6	-	1.0	0.3	-	0.3
Dengue	0	0	0	0	0	-	-	-	-	-
E. *coli* O157:H7	1	1	0	1	3	0.3	0.2	-	0.2	0.2
Encephalitis	6	1	5	0	12	1.7	0.2	1.6	-	0.7
Giardiasis	4	8	16	18	46	1.1	1.7	5.1	3.2	2.7
Haemophilus influenzae type b	0	0	0	0	0	-	-	-	-	-
Hansen's Disease (Leprosy)	0	0	0	0	0	-	-	-	-	-
Hepatitis A	9	6	18	24	57	2.5	1.3	5.7	4.2	3.3
Hepatitis B	2	0	3	1	6	0.6	-	1.0	0.2	0.3
Hepatitis C	0	0	0	0	0	-	-	-	-	-
Hepatitis unspecified	3	0	0	0	3	0.8	-	-	-	0.2
Kawasaki syndrome	5	2	2	4	13	1.4	0.4	0.6	0.7	0.8
Legionellosis	0	1	2	1	4	-	0.2	0.6	0.2	0.2
Listeriosis, nonperinatal	4	2	1	1	8	1.1	0.4	0.3	0.2	0.5
Listeriosis, perinatal[a]	1	0	0	1	2	1.3	-	-	0.8	0.5
Lyme disease	0	0	0	0	0	-	-	-	-	-
Malaria	0	0	1	3	4	-	-	0.3	0.5	0.2
Measles	0	0	0	0	0	-	-	-	-	-
Meningitis, viral	22	5	16	35	78	6.1	1.0	5.1	6.1	4.5
Meningococcal infections	1	1	2	0	4	0.3	0.2	0.6	-	0.2
Mumps	0	0	0	0	0	-	-	-	-	-
Pertussis	4	1	3	13	21	1.1	0.2	1.0	2.3	1.2
Psittacosis	0	0	0	0	0	-	-	-	-	-
Q-fever	0	0	0	0	0	-	-	-	-	-
Relapsing fever	0	0	1	0	1	-	-	0.3	-	0.1
Rheumatic fever, acute	0	0	0	0	0	-	-	-	-	-
Rubella	0	0	0	0	0	-	-	-	-	-
Salmonellosis	47	27	54	61	189	13.1	5.7	17.2	10.7	11.0
Shigellosis	21	5	22	14	62	5.9	1.0	7.0	2.5	3.6
Strongyloidiasis	0	0	0	0	0	-	-	-	-	-
Tetanus	0	0	0	0	0	-	-	-	-	-
Trichinosis	0	0	1	0	1	-	-	0.3	-	0.1
Tularemia	0	0	0	0	0	-	-	-	-	-
Typhoid fever, case	2	0	4	1	7	0.6	-	1.3	0.2	0.4
Typhoid fever, carrier	0	0	0	0	0	-	-	-	-	-
Typhus fever	1	0	1	1	3	0.3	-	0.3	0.2	0.2
Vibrio	0	0	0	0	0	-	-	-	-	-

[a] Rates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.

[b] Rates of disease based on less than 19 cases or events are considered "unreliable." A zero rate made from no events is especially hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

Table O-4. Selected Notifiable Diseases
SPA 4. Metro Area
Los Angeles County, 2006

Disease	Frequency				Rate (Cases per 100,000)[b]			
	CE	HW	NE	TOTAL	CE	HW	NE	TOTAL
Amebiasis	2	12	3	17	0.5	2.2	0.9	1.3
Botulism	0	0	0	0	-	-	-	-
Brucellosis	0	0	0	0	-	-	-	-
Campylobacteriosis	20	49	29	98	5.4	9.1	8.3	7.8
Cholera	0	0	0	0	-	-	-	-
Coccidioidomycosis	9	4	1	14	2.4	0.7	0.3	1.1
Cryptosporidiosis	3	7	3	13	0.8	1.3	0.9	1.0
Cysticercosis	0	0	0	0	-	-	-	-
Dengue	0	0	0	0	-	-	-	-
E. *coli* O157:H7	0	1	0	1	-	0.2	-	0.1
Encephalitis	2	1	0	3	0.5	0.2	-	0.2
Giardiasis	11	36	10	57	3.0	6.7	2.9	4.5
Haemophilus influenzae type b	0	0	0	0	-	-	-	-
Hansen's Disease (Leprosy)	0	0	1	1	-	-	0.3	0.1
Hepatitis A	52	22	5	79	14.1	4.1	1.4	6.3
Hepatitis B	8	6	2	16	2.2	1.1	0.6	1.3
Hepatitis C	0	0	0	0	-	-	-	-
Hepatitis unspecified	0	0	0	0	-	-	-	-
Kawasaki syndrome	4	4	2	10	1.1	0.7	0.6	0.8
Legionellosis	0	4	3	7	-	0.7	0.9	0.6
Listeriosis, nonperinatal	2	2	1	5	0.5	0.4	0.3	0.4
Listeriosis, perinatal[a]	1	0	2	3	1.3	-	2.6	1.1
Lyme disease	2	3	0	5	0.5	0.6	-	0.4
Malaria	2	3	0	5	0.5	0.6	-	0.4
Measles	0	0	0	0	-	-	-	-
Meningitis, viral	8	6	9	23	2.2	1.1	2.6	1.8
Meningococcal infections	0	4	0	4	-	0.7	-	0.3
Mumps	1	0	1	2	0.3	-	0.3	0.2
Pertussis	2	10	2	14	0.5	1.8	0.6	1.1
Psittacosis	0	0	0	0	-	-	-	-
Q-fever	0	0	0	0	-	-	-	-
Relapsing fever	0	0	0	0	-	-	-	-
Rheumatic fever, acute	0	0	0	0	-	-	-	-
Rubella	0	0	0	0	-	-	-	-
Salmonellosis	53	82	44	179	14.3	15.2	12.6	14.2
Shigellosis	27	54	22	103	7.3	10.0	6.3	8.2
Strongyloidiasis	0	0	0	0	-	-	-	-
Tetanus	0	2	0	2	-	0.4	-	0.2
Trichinosis	0	0	0	0	-	-	-	-
Tularemia	0	0	0	0	-	-	-	-
Typhoid fever, case	0	0	0	0	-	-	-	-
Typhoid fever, carrier	0	1	0	1	-	0.2	-	0.1
Typhus fever	0	0	1	1	-	-	0.3	0.1
Vibrio	0	3	0	3	-	0.6	-	0.2

[a] Rates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.

[b] Rates of disease based on less than 19 cases or events are considered "unreliable." A zero rate made from no events is especially hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

Table O-5. Selected Notifiable Diseases
SPA 5. West Area
Los Angeles County, 2006

Disease	Frequency	Rate (Cases per 100,000)[b]
	West	West
Amebiasis	12	1.9
Botulism	0	-
Brucellosis	0	-
Campylobacteriosis	119	18.7
Cholera	0	-
Coccidioidomycosis	9	1.4
Cryptosporidiosis	2	0.3
Cysticercosis	0	-
Dengue	1	0.2
E. *coli* O157:H7	0	-
Encephalitis	1	0.2
Giardiasis	44	6.9
Haemophilus influenzae type b	1	0.2
Hansen’s Disease (Leprosy)	0	-
Hepatitis A	24	3.8
Hepatitis B	3	0.5
Hepatitis C	0	-
Hepatitis unspecified	0	-
Kawasaki syndrome	3	0.5
Legionellosis	1	0.2
Listeriosis, nonperinatal	4	0.6
Listeriosis, perinatal[a]	0	-
Lyme disease	2	0.3
Malaria	3	0.5
Measles	0	-
Meningitis, viral	10	1.6
Meningococcal infections	1	0.2
Mumps	2	0.3
Pertussis	11	1.7
Psittacosis	0	-
Q-fever	0	-
Relapsing fever	0	-
Rheumatic fever, acute	0	-
Rubella	0	-
Salmonellosis	104	16.3
Shigellosis	34	5.3
Strongyloidiasis	0	-
Tetanus	0	-
Trichinosis	0	-
Tularemia	0	-
Typhoid fever, case	2	0.3
Typhoid fever, carrier	0	-
Typhus fever	1	0.2
Vibrio	6	0.9

[a] Rates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.

[b] Rates of disease based on less than 19 cases or events are considered "unreliable." A zero rate made from no events is especially hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

Table O-6. Selected Notifiable Diseases
SPA 6. South Area
Los Angeles County, 2006

Disease	Frequency					Rate (Cases per 100,000)[b]				
	CN	SO	SE	SW	TOTAL	CN	SO	SE	SW	TOTAL
Amebiasis	2	0	1	1	4	0.7	-	0.6	0.3	0.4
Botulism	0	0	0	0	0	-	-	-	-	-
Brucellosis	1	0	0	0	1	0.3	-	-	-	0.1
Campylobacteriosis	12	13	19	19	63	4.1	6.9	10.6	5.0	6.0
Cholera	0	0	0	0	0	-	-	-	-	-
Coccidioidomycosis	6	4	3	3	16	2.0	2.1	1.7	0.8	1.5
Cryptosporidiosis	0	1	0	2	3	-	0.5	-	0.5	0.3
Cysticercosis	0	0	0	0	0	-	-	-	-	-
Dengue	0	0	0	0	0	-	-	-	-	-
E. *coli* O157:H7	0	0	0	0	0	-	-	-	-	-
Encephalitis	0	1	0	0	1	-	0.5	-	-	0.1
Giardiasis	5	4	9	16	34	1.7	2.1	5.0	4.2	3.3
Haemophilus influenzae type b	0	0	0	0	0	-	-	-	-	-
Hansen's Disease (Leprosy)	0	0	0	0	0	-	-	-	-	-
Hepatitis A	7	5	10	15	37	2.4	2.7	5.6	3.9	3.6
Hepatitis B	2	1	1	2	6	0.7	0.5	0.6	0.5	0.6
Hepatitis C	0	0	0	1	1	-	-	-	0.3	0.1
Hepatitis unspecified	0	0	0	0	0	-	-	-	-	-
Kawasaki syndrome	2	1	3	2	8	0.7	0.5	1.7	0.5	0.8
Legionellosis	0	0	0	0	0	-	-	-	-	-
Listeriosis, nonperinatal	0	0	0	1	1	-	-	-	0.3	0.1
Listeriosis, perinatal[a]	0	0	1	1	2	-	-	2.4	1.1	0.8
Lyme disease	0	0	0	0	0	-	-	-	-	-
Malaria	1	1	0	6	8	0.3	0.5	-	1.6	0.8
Measles	0	0	0	0	0	-	-	-	-	-
Meningitis, viral	9	7	2	13	31	3.1	3.7	1.1	3.4	3.0
Meningococcal infections	5	2	4	3	14	1.7	1.1	2.2	0.8	1.3
Mumps	0	0	0	0	0	-	-	-	-	-
Pertussis	8	1	4	4	17	2.7	0.5	2.2	1.0	1.6
Psittacosis	0	0	0	0	0	-	-	-	-	-
Q-fever	1	0	0	0	1	0.3	-	-	-	0.1
Relapsing fever	0	0	0	0	0	-	-	-	-	-
Rheumatic fever, acute	0	0	0	0	0	-	-	-	-	-
Rubella	0	0	0	0	0	-	-	-	-	-
Salmonellosis	42	15	32	53	142	14.3	8.0	17.9	13.9	13.6
Shigellosis	25	23	18	40	106	8.5	12.3	10.0	10.5	10.2
Strongyloidiasis	0	0	0	0	0	-	-	-	-	-
Tetanus	0	0	0	1	1	-	-	-	0.3	0.1
Trichinosis	0	0	0	0	0	-	-	-	-	-
Tularemia	0	0	0	0	0	-	-	-	-	-
Typhoid fever, case	0	0	0	1	1	-	-	-	0.3	0.1
Typhoid fever, carrier	0	0	0	0	0	-	-	-	-	-
Typhus fever	0	0	1	0	1	-	-	0.6	-	0.1
VIbrio	0	0	0	0	0	-	-	-	-	-

[a] Rates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.

[b] Rates of disease based on less than 19 cases or events are considered "unreliable." A zero rate made from no events is especially hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

Table O-7. Selected Notifiable Diseases
SPA 7. East Area
Los Angeles County, 2006

Disease	Frequency					Rate (Cases per 100,000)[b]				
	BF	EL	SA	WH	TOTAL	BF	EL	SA	WH	TOTAL
Amebiasis	1	2	2	2	7	0.3	0.9	0.4	0.6	0.5
Botulism	0	0	0	1	1	-	-	-	0.3	0.1
Brucellosis	0	0	2	0	2	-	-	0.4	-	0.1
Campylobacteriosis	20	16	26	32	94	5.4	7.1	5.8	9.6	6.8
Cholera	0	0	0	0	0	-	-	-	-	-
Coccidioidomycosis	1	3	2	3	9	0.3	1.3	0.4	0.9	0.7
Cryptosporidiosis	3	2	1	2	8	0.8	0.9	0.2	0.6	0.6
Cysticercosis	0	0	1	0	1	-	-	0.2	-	0.1
Dengue	0	0	0	0	0	-	-	-	-	-
E. *coli* O157:H7	0	0	0	1	1	-	-	-	0.3	0.1
Encephalitis	2	1	4	1	8	0.5	0.4	0.9	0.3	0.6
Giardiasis	14	5	7	4	30	3.8	2.2	1.6	1.2	2.2
Haemophilus influenzae type b	1	1	1	0	3	0.3	0.4	0.2	-	0.2
Hansen's Disease (Leprosy)	0	1	0	0	1	-	0.4	-	-	0.1
Hepatitis A	8	7	13	5	33	2.2	3.1	2.9	1.5	2.4
Hepatitis B	3	1	1	1	6	0.8	0.4	0.2	0.3	0.4
Hepatitis C	0	0	0	0	0	-	-	-	-	-
Hepatitis unspecified	0	0	1	0	1	-	-	0.2	-	0.1
Kawasaki syndrome	2	1	3	3	9	0.5	0.4	0.7	0.9	0.7
Legionellosis	1	1	2	3	7	0.3	0.4	0.4	0.9	0.5
Listeriosis, nonperinatal	0	0	0	0	0	-	-	-	-	-
Listeriosis, perinatal[a]	0	0	2	0	2	-	-	1.9	-	0.7
Lyme disease	0	0	0	0	0	-	-	-	-	-
Malaria	1	0	1	0	2	0.3	-	0.2	-	0.1
Measles	0	0	0	0	0	-	-	-	-	-
Meningitis, viral	10	5	36	8	59	2.7	2.2	8.0	2.4	4.3
Meningococcal infections	1	2	2	1	6	0.3	0.9	0.4	0.3	0.4
Mumps	1	0	1	0	2	0.3	-	0.2	-	0.1
Pertussis	13	2	5	7	27	3.5	0.9	1.1	2.1	2.0
Psittacosis	0	0	0	0	0	-	-	-	-	-
Q-fever	0	0	0	0	0	-	-	-	-	-
Relapsing fever	0	0	0	0	0	-	-	-	-	-
Rheumatic fever, acute	0	0	0	0	0	-	-	-	-	-
Rubella	0	0	0	0	0	-	-	-	-	-
Salmonellosis	36	49	58	32	175	9.7	21.8	12.9	9.6	12.7
Shigellosis	15	26	27	16	84	4.1	11.6	6.0	4.8	6.1
Strongyloidiasis	0	0	0	0	0	-	-	-	-	-
Tetanus	1	0	0	0	1	0.3	-	-	-	0.1
Trichinosis	0	0	0	0	0	-	-	-	-	-
Tularemia	0	0	0	0	0	-	-	-	-	-
Typhoid fever, case	1	0	2	0	3	0.3	-	0.4	-	0.2
Typhoid fever, carrier	0	0	2	0	2	-	-	0.4	-	0.1
Typhus fever	1	0	0	0	1	0.3	-	-	-	0.1
VIbrio	4	0	0	2	6	1.1	-	-	0.6	0.4

[a] Rates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.

[b] Rates of disease based on less than 19 cases or events are considered "unreliable." A zero rate made from no events is especially hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

Table O-8. Selected Notifiable Diseases
SPA 8. South Bay Area
Los Angeles County, 2006

Disease	Frequency				Rate (Cases per 100,000)[b]			
	HB	IW	TO	TOTAL	HB	IW	TO	TOTAL
Amebiasis	0	3	4	7	-	0.7	0.9	0.6
Botulism	0	0	0	0	-	-	-	-
Brucellosis	0	0	0	0	-	-	-	-
Campylobacteriosis	23	20	22	65	11.0	4.6	4.7	5.8
Cholera	0	0	0	0	-	-	-	-
Coccidioidomycosis	2	3	7	12	1.0	0.7	1.5	1.1
Cryptosporidiosis	0	1	0	1	-	0.2	-	0.1
Cysticercosis	0	0	1	1	-	-	0.2	0.1
Dengue	0	0	0	0	-	-	-	-
E. *coli* O157:H7	0	0	1	1	-	-	0.2	0.1
Encephalitis	3	3	2	8	1.4	0.7	0.4	0.7
Giardiasis	4	9	14	27	1.9	2.1	3.0	2.4
Haemophilus influenzae type b	0	0	0	0	-	-	-	-
Hansen's Disease (Leprosy)	0	0	0	0	-	-	-	-
Hepatitis A	22	10	13	45	10.5	2.3	2.8	4.0
Hepatitis B	3	1	2	6	1.4	0.2	0.4	0.5
Hepatitis C	2	0	0	2	1.0	-	-	0.2
Hepatitis unspecified	0	0	1	1	-	-	0.2	0.1
Kawasaki syndrome	2	7	8	17	1.0	1.6	1.7	1.5
Legionellosis	0	1	0	1	-	0.2	-	0.1
Listeriosis, nonperinatal	0	0	0	0	-	-	-	-
Listeriosis, perinatal[a]	0	1	0	1	-	1.0	-	0.4
Lyme disease	1	1	1	3	0.5	0.2	0.2	0.3
Malaria	0	2	4	6	-	0.5	0.9	0.5
Measles	0	0	0	0	-	-	-	-
Meningitis, viral	15	7	30	52	7.2	1.6	6.4	4.7
Meningococcal infections	0	2	2	4	-	0.5	0.4	0.4
Mumps	0	0	0	0	-	-	-	-
Pertussis	5	1	10	16	2.4	0.2	2.1	1.4
Psittacosis	0	0	0	0	-	-	-	-
Q-fever	0	0	0	0	-	-	-	-
Relapsing fever	0	0	0	0	-	-	-	-
Rheumatic fever, acute	0	0	0	0	-	-	-	-
Rubella	0	0	0	0	-	-	-	-
Salmonellosis	37	44	42	123	17.7	10.1	9.0	11.1
Shigellosis	14	16	11	41	6.7	3.7	2.4	3.7
Strongyloidiasis	0	0	0	0	-	-	-	-
Tetanus	0	0	0	0	-	-	-	-
Trichinosis	0	0	0	0	-	-	-	-
Tularemia	0	0	0	0	-	-	-	-
Typhoid fever, case	1	0	0	1	0.5	-	-	0.1
Typhoid fever, carrier	0	0	0	0	-	-	-	-
Typhus fever	0	0	0	0	-	-	-	-
Vibrio	0	0	1	1	-	-	0.2	0.1

[a] Rates for perinatal listeriosis were calculated as cases per 100,000 women aged 15 to 44 years.

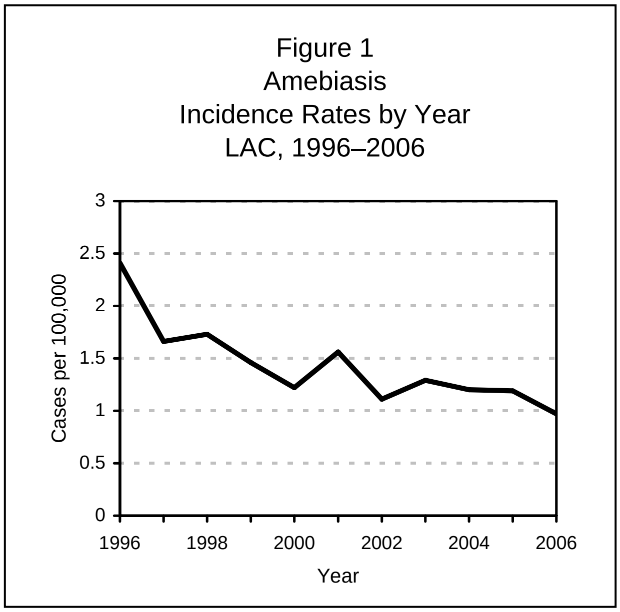
[b] Rates of disease based on less than 19 cases or events are considered "unreliable." A zero rate made from no events is especiallly hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

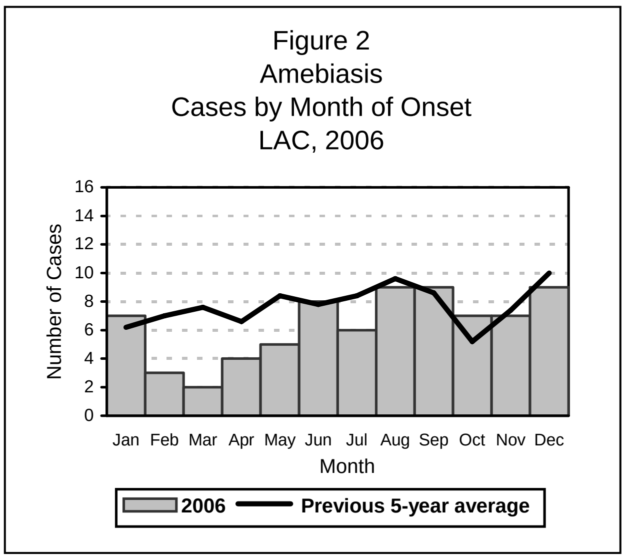
DISEASE SUMMARIES
2006

AMEBIASIS

CRUDE DATA	
Number of Cases	94
Annual Incidence[a]	
LA County	0.97
United States	N/A
Age at Diagnosis	
Mean	39.9
Median	40
Range	5-87 years

[a] Cases per 100,000 population.





DESCRIPTION

Amebiasis is caused by the protozoan parasite *Entamoeba histolytica*. Cysts shed in human feces may contaminate food or drinking water or be transferred sexually, on hands, or fomites. Incubation period is 1 to 4 weeks. Recreational waters such as lakes and pools may also serve as transmission vehicles, since cysts are relatively chlorine-resistant. While intestinal disease is often asymptomatic, symptoms 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 colonic perforation. There is no vaccine. The most commonly ordered parasite test (microscopy of stool for ova and parasites) cannot distinguish *E. histolytica* from *E. dispar*, a non-pathogenic amebic species. There is an available EIA test, however, that can distinguish between the two.

DISEASE ABSTRACT

- Amebiasis incidence has decreased substantially over the past 10 years. In 2006 the rate decreased from 1.19 per 100,000 to 0.97 per 100,000.
- Decreasing numbers of refugees and immigrants from endemic regions or a reduction in testing may account for the decrease in cases.
- No amebiasis outbreaks were reported during 2006.

STRATIFIED DATA

Trends: After a small increase in 2003, the 2006 amebiasis incidence rate decreased still further to 0.97 per 100,000 (Figure 1).

Seasonality: Amebiasis incidence usually peaks in the summer months; however, in 2006 the incidence rose in the summer months and remained elevated through December (Figure 2).

Age: While amebiasis is ubiquitous, it is a disease more often diagnosed among adults (Figure 3). About two-thirds of the cases reported in LAC during 2006 were among those aged 15–54 (n=72, 77%). Amebiasis is rare among those below age 5 and especially rare among those below age 2. Dysentery in infants is typically due to Shigella.

Sex: Males (57%) continue to be slightly more likely to contract amebiasis than females, with a ratio of 1.74:1.

Race/Ethnicity: In 2006, whites had the highest rate, closely followed by Asians and Latinos (Figure 4). The rate for Asians increased from 0.4 per 100,000 in 2005 to 0.8 per 100,000 in 2006. The rate for blacks decreased from 0.8 per 100,000 in 2005 to 0.2 per 100,000 in 2006.

Location: Three SPAs had rates greater than the county mean rate: SPA 2 (1.8 per 100,000), SPA 4 (1.3 per 100,000) and SPA 5 (1.9 per 100,000).

Risk factors: Many of the cases (n=33, 35%) were recent immigrants (less than 6 months) and 20 cases (21%) reported recent foreign travel.

Figure 3
Amebiasis
Incidence Rates by Age Group
LAC, 2006

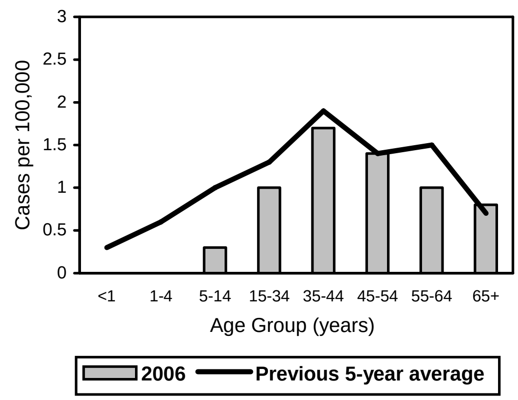
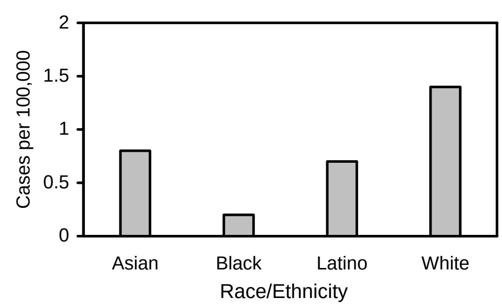


Figure 4
Amebiasis
Incidence Rates by Race/Ethnicity
LAC, 2006



COMMENTS

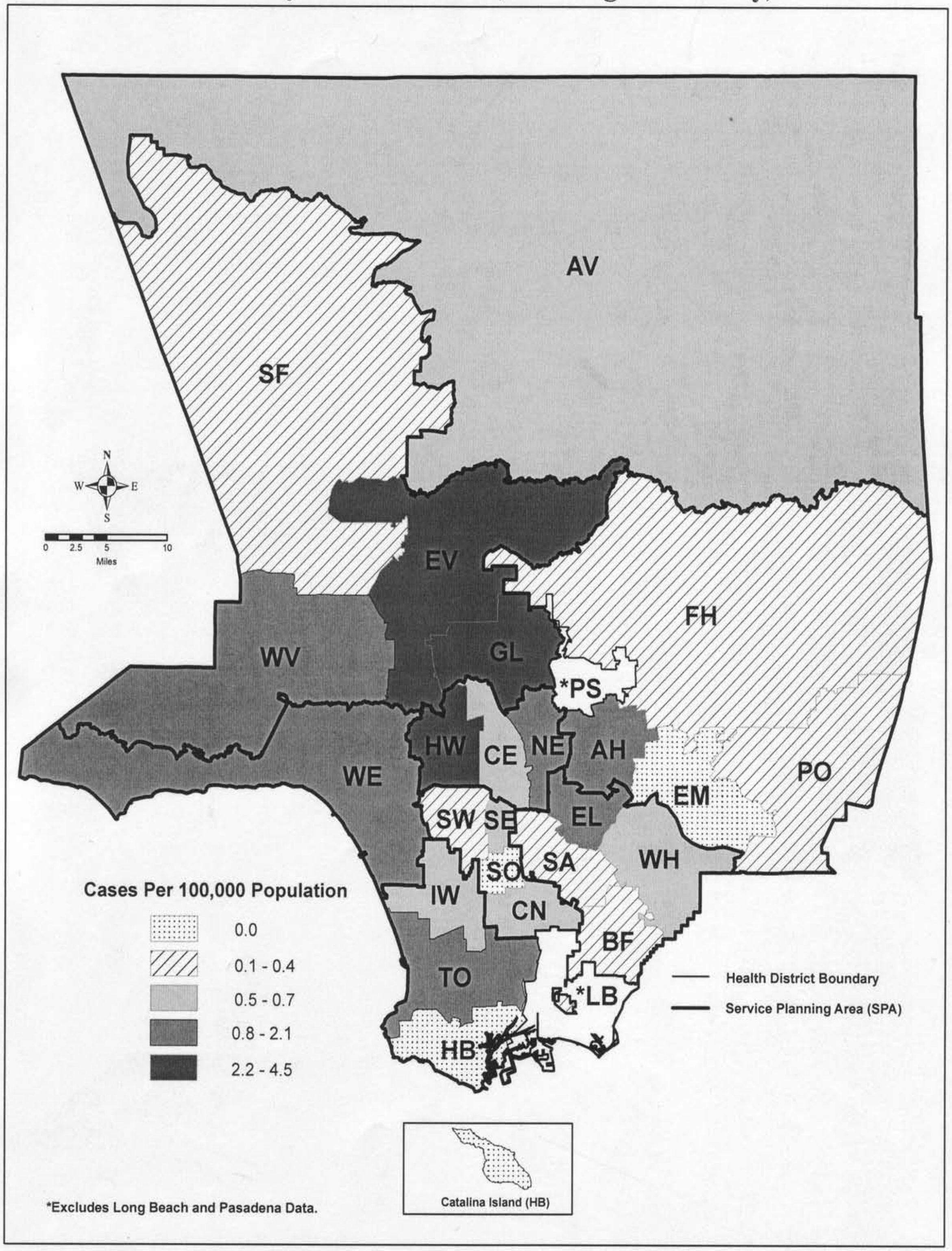
Amebiasis is no longer nationally reportable, so there are no current national rates for comparison. The disease remains reportable in California because a large proportion of the population travels to endemic countries in Asia and Central America. The impact of new tests that distinguish *E. histolytica* from *E. dispar* is unknown since such tests are rarely ordered. It is believed that many reported amebiasis cases are actually not infected with pathogenic *E. histolytica*.

ADDITIONAL RESOURCES

Amebiasis - Health Information for International Trave, 2008:
wwwn.cdc.gov/travel/yellowBookCh4-Amebiasis.aspx

More CDC Information on Amebiasis:
www.cdc.gov/ncidod/dpd/parasites/amebiasis/default.htm

Map 2. Amebiasis
Rates by Health District, Los Angeles County, 2006*

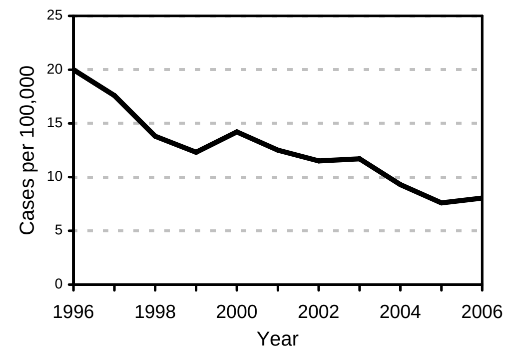


CAMPYLOBACTERIOSIS

CRUDE DATA	
Number of Cases	775
Annual Incidence[a]	
LA County	8.0
United States	N/A
Age at Diagnosis	
Mean	34.16
Median	32
Range	0–98

[a] Cases per 100,000 population.

Figure 1
Campylobacteriosis
Incidence Rates by Year of Onset
LAC, 1996–2006



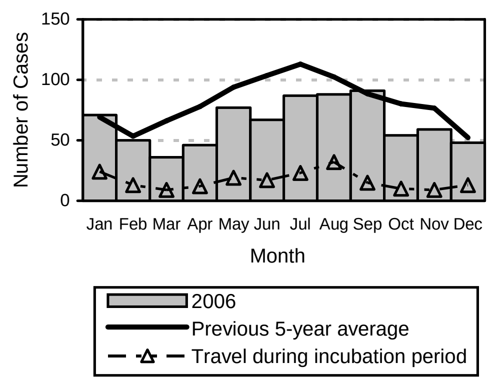
DESCRIPTION

Campylobacteriosis is a bacterial disease caused by Gram-negative bacilli transmitted through ingestion of organisms in undercooked poultry or other meat, contaminated food, water or raw milk, or contact with infected animals. The incubation period is 2–5 days. Common symptoms include watery or bloody diarrhea, fever, abdominal cramps, myalgia, and nausea. Species include *C. jejuni, C. upsaliensis, C. coli* and *C. fetus*. Sequelae include Guillain-Barré syndrome and Reiter syndrome, which occur in a limited number of cases.

DISEASE ABSTRACT

- There was a 6.9% increase in the incidence of campylobacteriosis in 2006.
- In 2006, overall age-adjusted rates were highest for whites.
- One outbreak of campylobacteriosis was investigated in 2006.

Figure 2
Campylobacteriosis
Cases and Travel History by
Month of Onset
LAC, 2006



STRATIFIED DATA

Trends: The incidence of campylobacteriosis increased by 6.9% in 2006. After two years of relative stability in 2002 and 2003, the rate of campylobacteriosis decreased significantly from 11.7 cases per 100,000 to 9.3 in 2004 and 7.6 in 2005 ($p < 0.05$). In 2006, the rated increased slightly to 8.0 cases per 100,000. Continued surveillance is needed to identify any new trend.

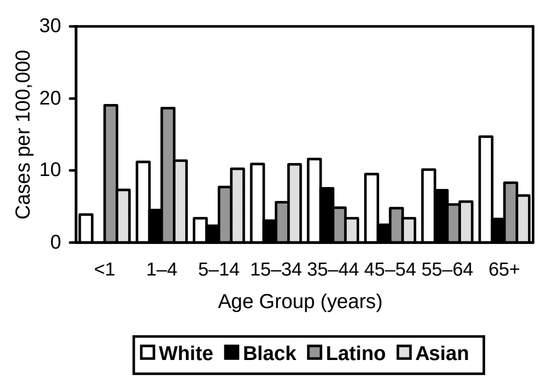
Seasonality: With the exception of January and September, monthly incidence decreased when compared to the previous five-year average. Incidence increased in the spring and summer as seen in other years. Peaks during these seasons may be associated with the increase in travel. Travel is a risk factor for infection since it is most likely associated with an increase in eating at restaurants—which is a risk factor for this disease. Risk also increases when traveling to countries where food safety is questionable. In 2006, 197 cases (25.4%) reported travel during the incubation period. Of these, 30% traveled within the US. Mexico was the most commonly named (33.5%) travel destination outside the US, although other locations in Central and South America and Europe were named frequently. In 2006, overall incidence peaked in September and travel related incidence peaked in August (Figure 2).

Age: The highest rates continued to be among infants aged <1 year and children, aged 1–4 years (Figure 3). These age groups had significantly higher rates than any other age group but the rates were lower than the previous five-year average. In developed countries, children younger than five years and young adults have the highest incidence of this disease.

Sex: The male-to-female rate ratio was 1.3:1. The preponderance of male cases is typical and the reason for this is not known [1]. Among men above the age of fifteen, only 1.3% reported sexual contact with other men (MSM).

Race/Ethnicity: The highest overall age-adjusted rate was in whites (9.96 cases per 100,000 population); this was a decrease from 2005 (11 per 100,000). In 2006 the age-adjusted rate for Latinos was stable (7.0) although Latinos had similar incidence to whites. Age-adjusted rates for Asians (7.7) and blacks (4.0) increased. Latino infants and children have the highest age adjusted rates when compared to other races by age group. Asians showed a higher rate for several age groups (Figure 3).

Figure 3
Campylobacteriosis Incidence Rates
by Age Group and Race/Ethnicity
LAC, 2006 (N=775)



Location: SPA 2 again had the highest number of cases at 217 (10.1 per 100,000), and SPA 5 had the highest rate with 18.7 per 100,000 (N= 119). The higher rate in SPA 5 is consistent with previous years and is significantly higher than any other SPA.

Severity of Illness: Thirteen percent of campylobacteriosis cases (N=101) were hospitalized for at least two days. Two campylobacteriosis-associated deaths occurred in a 78 year-old male and a 52 year-old male. Both deaths were associated with multiple medical problems including a history of liver and lung cancer. Although, there is no active surveillance of disease sequelae, there was one report of Guillain-Barré syndrome (GBS) subsequent to a campylobacteriosis diagnosis. Fifteen percent of campylobacteriosis cases were immunocompromised (N=120). Reasons for immunosuppression included HIV, AIDS, diabetes, leukemia, kidney and liver transplant, lupus, cancer, and recent diagnosis of cancer with treatment.

PREVENTION

To reduce the likelihood of contracting campylobacteriosis, all food derived from animal sources should be thoroughly cooked, particularly poultry. 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.

COMMENTS

Consuming raw milk or raw milk products was a risk factor for twelve sporadic cases; four of these cases consumed the milk or product while traveling outside the US and two consumed unpasteurized cheese brought back from Mexico.

There was one campylobacteriosis outbreaks investigated in 2006. This outbreak was travel related, involving a missionary group. There were two confirm cases in this outbreak.

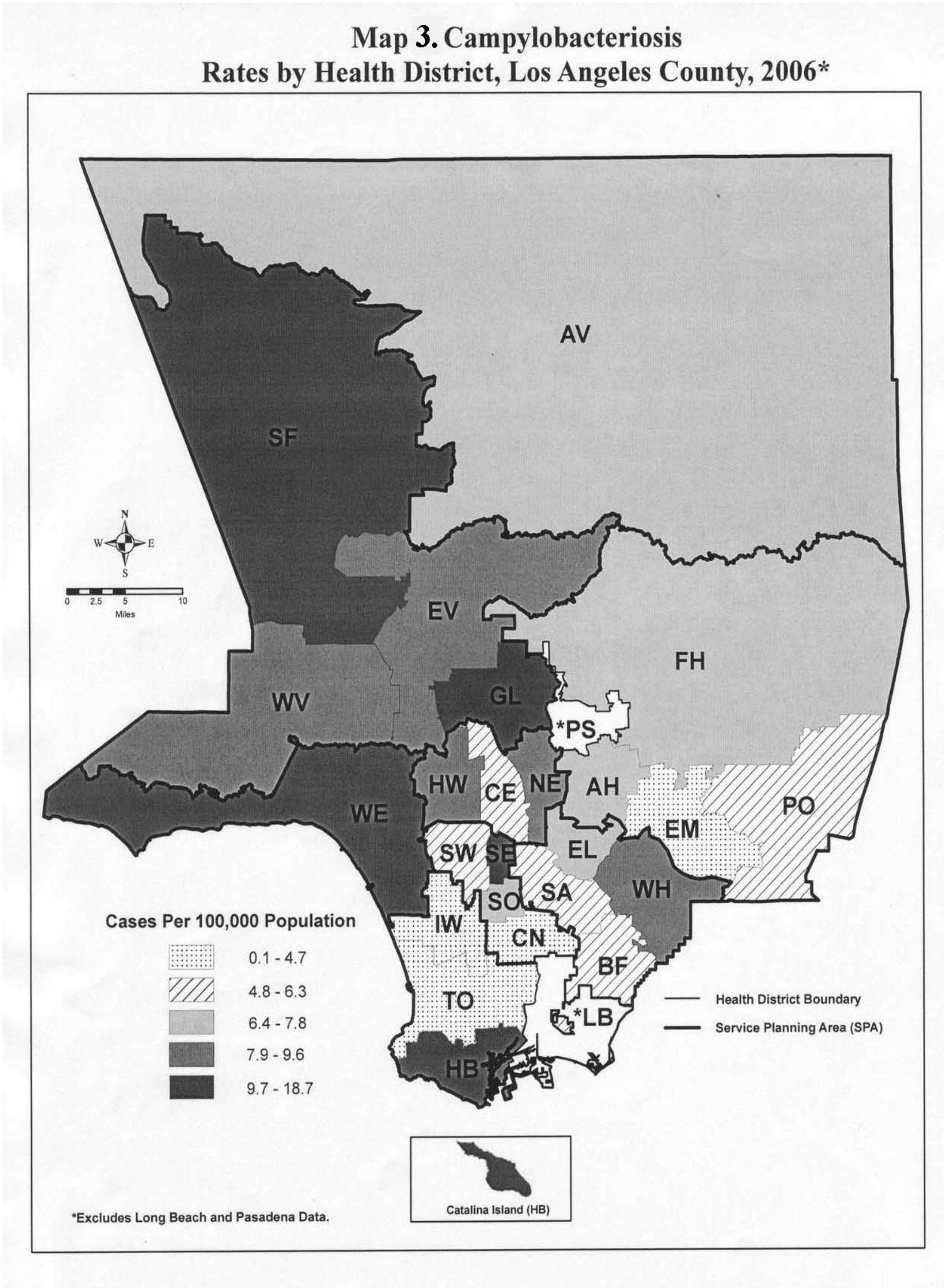
REFERENCES

1. Allos BM. Campylobacter jejuni infections: update on emerging issues and trends. Clin Infect Dis 2001; 32(8):1201–1206.

ADDITIONAL RESOURCES

Disease information is available from the CDC at:
www.cdc.gov/ncidod/dbmd/diseaseinfo/campylobacter_g.htm

General information and reporting information about this and other foodborne diseases in LAC is available at: www.lapublichealth.org/acd/food.htm

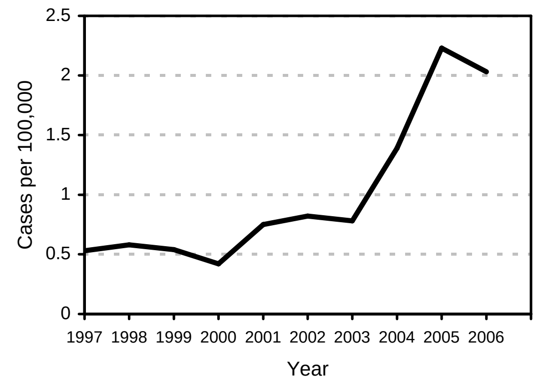

Map 3. Campylobacteriosis
Rates by Health District, Los Angeles County, 2006*
AV
SF
EV
FH
WV
GL
*PS
HW
CE
NE
AH
WE
PO
EM
SW
SE
EL
SA
WH
SO
IW
CN
BF
TO
*LB
HB
N
W
E
S
0
2.5
5
10
Miles
Cases Per 100,000 Population
0.1 - 4.7
4.8 - 6.3
6.4 - 7.8
7.9 - 9.6
9.7 - 18.7
Health District Boundary
Service Planning Area (SPA)
Catalina Island (HB)
*Excludes Long Beach and Pasadena Data.

COCCIDIOIDOMYCOSIS

CRUDE DATA	
Number of Cases	196
Annual Incidence[a]	
LA County	2.03
California	8.67[b]
United States	3.01[b]
Age at Diagnosis	
Mean	46.8
Median	48
Range	3-88 years

[a] Cases per 100,000 population.
[b] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

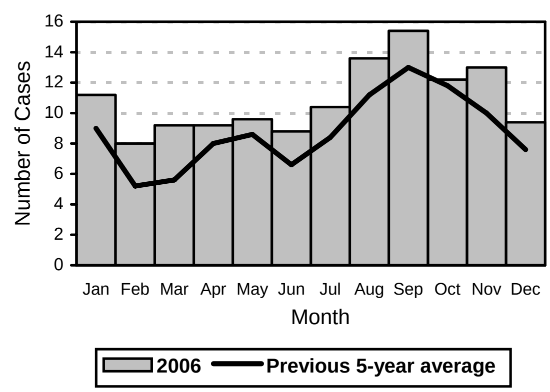
Figure 1
Coccidioidomycosis
Incidence Rates by Year of Onset
LAC, 1997–2006



DESCRIPTION

Coccidioidomycosis, or Valley Fever, is a common fungal disease transmitted through the inhalation of *Coccidioides immitis spores* that are carried in dust. Environmental conditions conducive to an increased occurrence of coccidioidomycosis are as follows: arid to semi-arid regions, dust storms, lower altitude, hotter summers, warmer winters, and sandy, alkaline soils. It is endemic in the southwestern US and parts of Mexico and South America. Southern California is a known endemic area.

Figure 2
Coccidioidomycosis
by Month of Onset
LAC, 2006 (N=196)



Most infected individuals exhibit no symptoms or have a mild respiratory illness, but a few individuals develop a severe illness such as pneumonia, meningitis, or dissemination when the fungus spreads to many parts of the body. Because of the wide range of clinical presentations, only the most severe cases are usually reported to the health department. Laboratory diagnosis is made by identifying the fungus through microscopic examination, culture, serologic testing or DNA probe. Blacks, Filipinos, pregnant women, the very young (<5 years), elderly, and immunocompromised individuals are at high risk for severe disease.

DISEASE ABSTRACT

- The incidence rate for coccidioidomycosis has been increasing since 2000, when it was at its lowest point in 10 years (1997-2006) in LAC.
- Cost in terms of disease severity and hospitalization is substantial.
- The incidence of coccidioidomycosis this year is slightly lower than last year. Adults, males, blacks, and residents of the San Fernando and Antelope Valleys are at higher risk for disease.

STRATIFIED DATA

Trends: The incidence rate was 2.03 cases per 100,000 population which decreased from last year's rate of 2.23 per 100,000 population (Figure 1).

Seasonality: The highest number of cases per month was observed in the 3rd and 4th quarters, although the highest numbers of cases have typically been seen in the 3rd quarter. The number of cases per month through most of 2006 was above the five-year average (Figure 2). Cases commonly occur in the summer after a rainy winter or spring, especially after wind and dust storms.

Age: Cases were predominantly in the adult age groups. The greatest numbers of cases reported were in persons aged 15-34 and 45-54 years (Figure 3). The greatest incidence rate was in the 65+ age group (3.7/100,000). The youngest case was less than 1 year of age. The mean age for males was 45 years and for females, 49 years (Figure 3).

Sex: The male-to-female rate ratio was 2.2:1. Males had an overall higher incidence rate, which is consistent with previous years. The gender difference is likely due to occupational and recreational dust exposure of males, although this is not clearly evident from the information collected. No female cases reported being pregnant.

Race/Ethnicity: The highest incidence rate of 3.2 cases per 100,000 was observed among blacks compared to the other race/ethnic groups. Whites had an incidence rate of 2.6 (n = 75), Latinos 1.5 (n= 68), Asians 1.2 (n=15) and others (n=3). Race was unknown in 2 cases (Figure 4).

Location: SPA 1 (Antelope Valley Health District) had the highest number of coccidioidomycosis cases (n=67), within SPA 2. San Fernando Valley had 32 cases and West Valley had 19 cases. SPA 1 and 2 cases combined comprise 60% of the total. This has added significance because the incidence rate per 100,000 in Antelope Valley is 19.3, San Fernando Valley 7.2 and West Valley 2.2. These districts are more arid than the rest of the county, thus have higher risk.

Travel: Travel history was available for 158 cases. Of those with a travel history, 53% (n=83) reported travel within four weeks before onset of illness, while 47% (n=75) reported no travel. Of those traveling, many reported multiple travel destinations: 42% (n=35) traveled within California including San Fernando Valley, Central Valley, and adjacent

Figure 3
Coccidioidomycosis
Incidence by Age and Sex
LAC, 2006 (N=196)

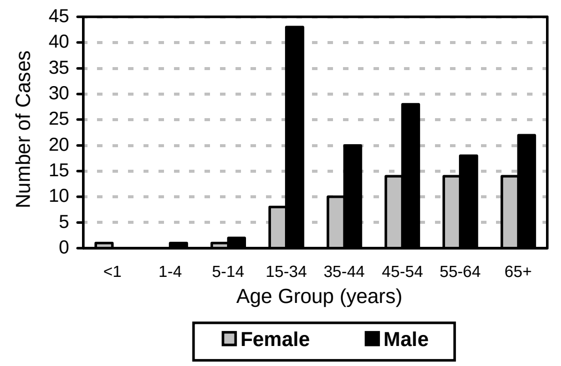


Figure 4
Coccidioidomycosis
Percent Cases by Race/Ethnicity
LAC, 2006

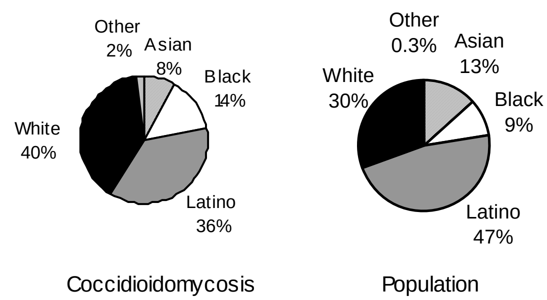
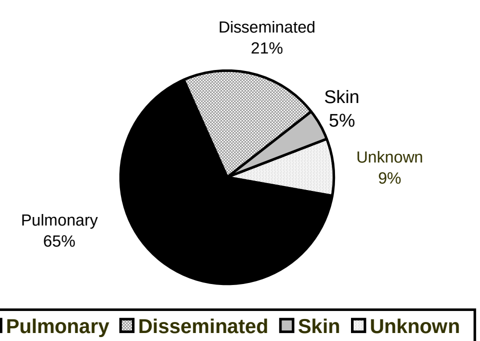


Figure 5
Coccidioidomycosis
by Infection Site
LAC, 2006 (N=196)



counties of Bakersfield and Oxnard; 52% (n=43) traveled outside California to Las Vegas, Arizona, Texas, Mexico, and South America, and 6% (n=5) reported travel both within and outside of California to other locations. The fungus is known to be endemic in most of these areas.

Underlying Disease: Out of 146 cases assessed, 97 cases (66%) reported having an underlying disease: 24% (n=35) diabetes, 7% (n=10) malignancy, 2% (n=3) HIV, 3% (n=4) organ transplants, and 28% (n=41) coded as other (e.g. asthma, kidney problems, sickle cell anemia). No disease history was reported in 36% (n=53) of cases. Some cases had multiple underlying diseases.

Severity of Disease: Sites of infection were reported as primary pulmonary 65% (n=128), disseminated 21% (n=41), meningitis 0.5% (n=1), skin 5% (n=9), and 9% (n=17) of the case infection sites were not indicated (Figure 5). Of the cases, 47 were culture-confirmed and 113 cases were diagnosed by serological, histopathological, or molecular testing. Some cases had multiple labs available for diagnosis. Of the 178 cases where information was available, 71% (n=127) were hospitalized.

COMMENTS

In LAC, the 2006 incidence for coccidioidomycosis was slightly lower than the previous year. Though there is a plateau in the rate of cases this year, overall, the rate has been increasing since 2000. The dramatic increase began in the fall of 2003 and the wildfires in southern California may have been a contributor by destroying vegetation and increasing dust exposure. This, followed by seasonal warm temperatures, drought, and Santa Ana winds are ideal conditions for disseminating *Coccidioides immitis* spores. Although the number of cases reported is small compared to other diseases, the costs in terms of disease severity, hospitalization, and mortality are great. Also, more young adults and adults ages 45-64, especially males are affected instead of the very young and old, who are normally at high risk for illness, which may reflect a higher outdoor recreational or occupational exposure in these groups.

As in past years, residents of the Antelope Valley and the West Valley are at higher risk for severe disease because these districts are more arid than the rest of the county. These areas of the county have seen a rapid growth in population. It is hypothesized that the influx of a naïve population living in areas of heavy construction, greatly increased risk for disease in a cocci endemic area.

The increased number of cases reported in SPA 2 may be due to a reporting bias. It was determined that cases were not being reported consistently from a single source, and after education was provided on reporting guidelines and procedures, an increase in the number of reports was noted.

PREVENTION / INTERVENTION

Currently, no safe and effective vaccine or drug to prevent coccidioidomycosis exists. Prevention lies mainly in dust 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 individuals who are at high risk for severe disease not to travel to endemic areas when conditions are most dangerous for exposure.

Although coccidioidomycosis cannot be readily prevented, improved understanding of the epidemiology of this disease can assist in developing more effective prevention strategies. To increase awareness among Antelope Valley residents and healthcare workers, a series of presentations on the epidemiology, clinical symptoms, diagnosis and treatment of coccidioidomycosis were provided.

Nikkomycin Z is an experimental compound that has been shown to exhibit antifungal properties by inhibiting chitin synthesis. Funding to continue clinical trials of nikkomycin Z is being sought, but even if the fund-raising efforts are successful, the drug is unlikely to be available for general use for another five to seven years.

Currently, vaccine research is being conducted in part by the state of California.

ADDITIONAL RESOURCES

Bussum LV. National Fire Weather Report, 2003. Report available at the National Weather Service, Boise, ID website: http://fire.boi.noaa.gov/FIREWX/AnnualReport/2003NationalReport.pdf

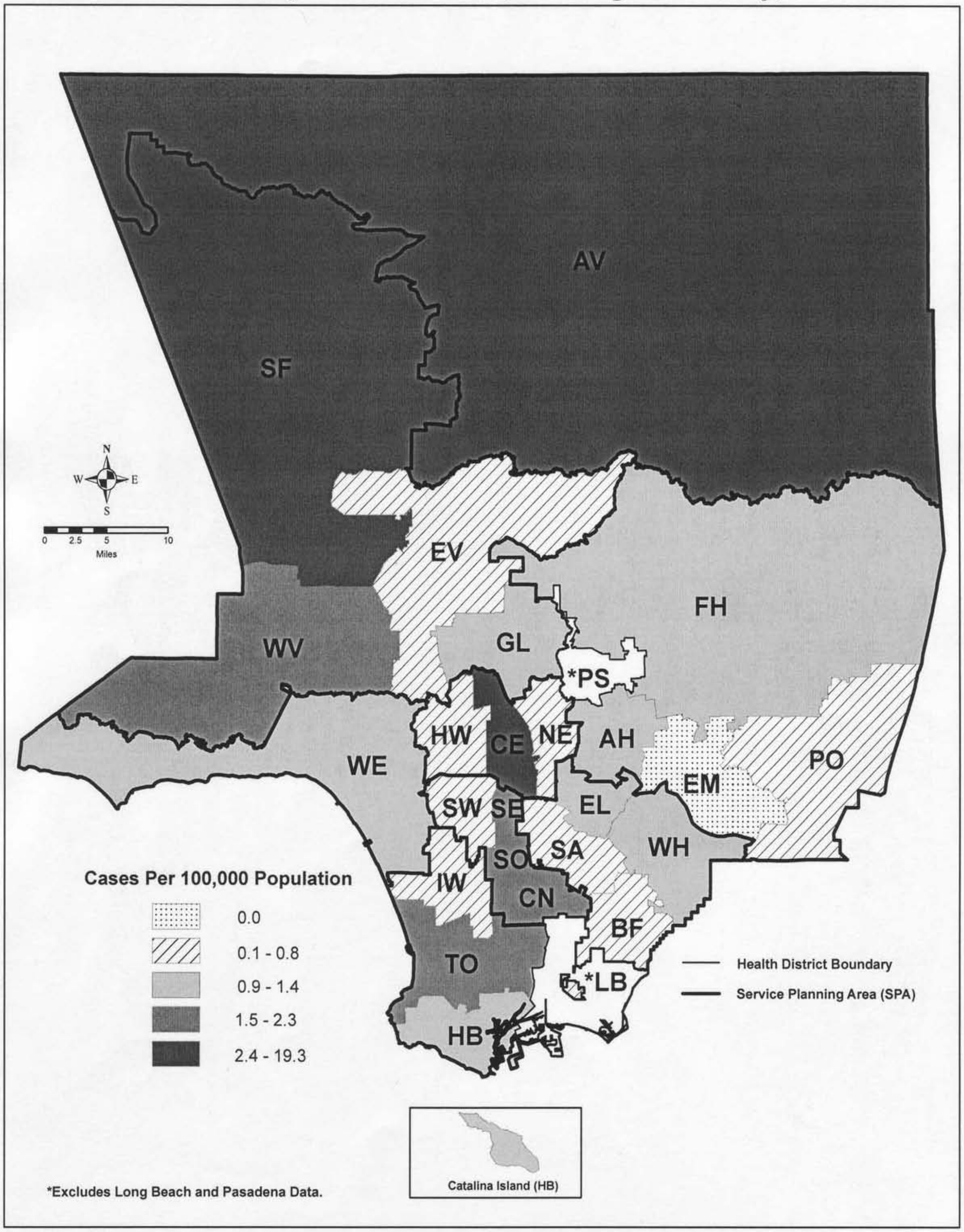
CDC website: www.cdc.gov/ncidod/dbmd/diseaseinfo/coccidioidomycosis_t.htm

Kirkland TN, Fierer J. Coccidioidomycosis: a reemerging infectious disease. Emerg Infect Dis 1996; 2(3):192–199.

Valdivia L, Nix D, Wright M, et al. Coccidioidomycosis as a common cause of community-acquired pneumonia. Emerg Infect Dis 2006; 12(8):958-969.

Valley Fever Center for Excellence website: www.vfce.arizona.edu/NAE-home.htm

Map 4. Coccidioidomycosis
Rates by Health District, Los Angeles County, 2006*

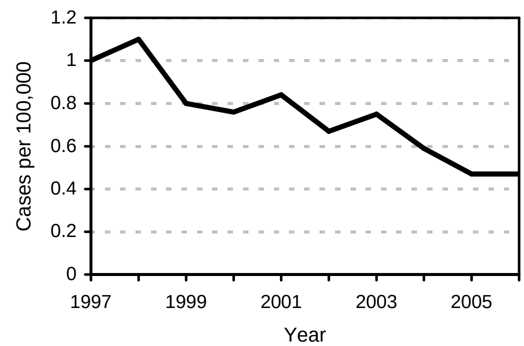


CRYPTOSPORIDIOSIS

CRUDE DATA	
Number of Cases	48
Annual Incidence[a]	
LA County	0.47
California	0.94[b]
United States	2.05[b]
Age at Diagnosis	
Mean	40
Median	39
Range	3-89

[a] Cases per 100,000 population.
[b] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

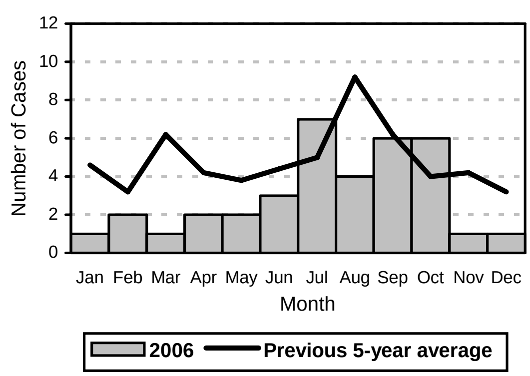
Figure 1
Cryptosporidiosis
Incidence Rates by Year of Onset
LAC, 1997–2006



DESCRIPTION

Cryptosporidiosis is fecal-orally transmitted when cysts of the parasite *Cryptosporidium parvum* are ingested. Common causes include unprotected sexual contact, particularly among men who have sex with men (MSM), and by swallowing contaminated recreational or untreated water. 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 2 weeks in healthy individuals. Those who have a weakened immune system may experience prolonged illness. Immunocompromised individuals (e.g., HIV/AIDS patients, cancer patients, transplant patients), young children and pregnant women are at risk for more severe illness.

Figure 2
Cryptosporidiosis
Cases by Month of Onset
LAC, 2006 (N=48)



DISEASE ABSTRACT

- The incidence rate for this disease decreased from 0.59 per 100,000 in 2004 to 0.47 per 100,000 in 2006. The incidence of this disease has remained the same for 2005 and 2006 and is the lowest incidence rate in the past ten years. The last outbreak of this disease occurred during 1988.
- HIV infection and AIDS are the most common identified risk factors for cryptosporidiosis. Cryptosporidiosis has been an AIDS-defining disease since 1983. The number of reported cases has decreased since the advent of highly active antiretroviral therapy.

STRATIFIED DATA

Trends: The rate of cryptosporidiosis (0.47 cases per 100,000) remained the same in 2006 (Figure 1).

Seasonality: In 2006, there was a peak in July, although the previous 5-year average peak was in August (Figure 2).

Age: The 35-44 age group had the highest incidence rate followed by the 55-64 and 45-64 age groups (Figure 3).

Sex: The male-to-female ratio was 5:3 (18 females). This marks a noticeable increase in the number of female cases from 2005 (n=7).

Race/Ethnicity: Blacks had the highest incidence rate (Figure 4), followed by whites and Latinos. Race was unknown for 2 cases (4.2%). The rate for blacks decreased from 1.2 per 100,000 in 2005 to 0.9 per 100,000 in 2006. There were no cases among Asians in 2006.

Location: Location information was available for all 48 cases. Hollywood-Wilshire (HW) Health District had the highest incidence rate, 1.3 per 100,000 (n=7), followed closely by Antelope Valley (AV) Health District, which had 1.2 per 100,000 (n=4).

Risk Factors: Complete risk factor data was not available for all cases; 10 cases (21%) were either unable to be located or refused to be interviewed (Figure 5). HIV infection and AIDS accounted for 46% of the cases. Animal contact (38%) and recent international travel (25%) were the other most common risk factors following HIV status. Many cases had more than one risk factor.

Figure 3
Cryptosporidiosis
Incidence Rates by Age Group
LAC, 2006

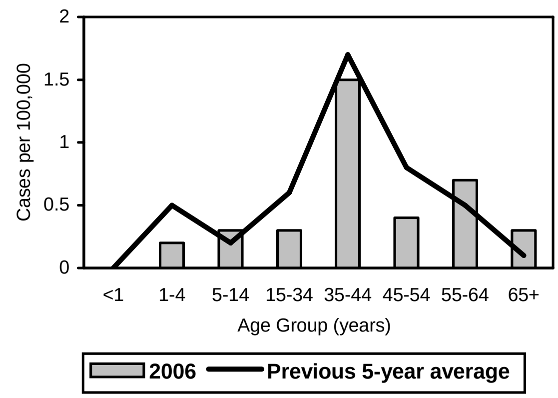


Figure 4
Cryptosporidiosis
Incidence Rate by Race/Ethnicity
LAC, 2006

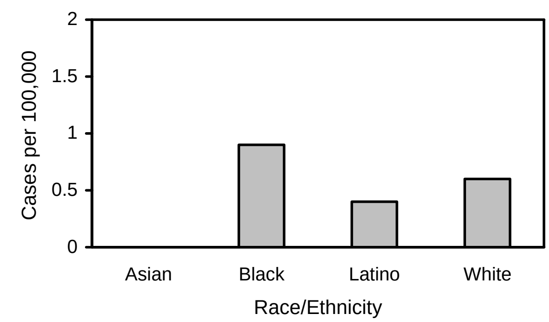
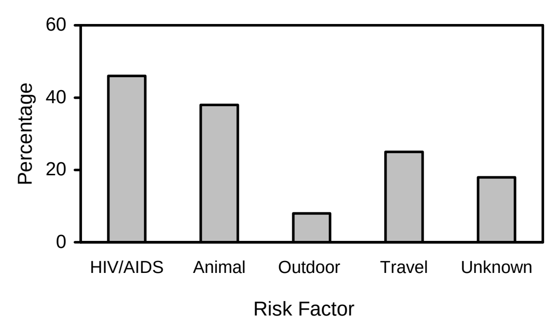


Figure 5
Cryptosporidiosis
by Reported Risk Factor
LAC, 2006



COMMENTS

Risk factors were self-reported and were not proven to be the actual source of infection. A large percentage (n=22, 46%) of the cryptosporidiosis cases were among HIV positive males. In 2006, the majority of male HIV cases were Latino (n=11, 50%). In 2005 and 2004 the majority of cases were black (44% and 45% respectively). However, these changes are not statistically significant due to the small number of cases.

Cryptosporidiosis can become a chronic infection among immunocompromised patients and cases are often reported multiple times; however, within this report, cases are counted only once. There has not been an outbreak of cryptosporidiosis in LAC since 1988, which involved contaminated swimming pool water [1].

RESOURCES

1. Sorvillo FJ, Fujioka K, Nahlen B, Tormey MP, Kebabjian R, Mascola L. Swimming-associated cryptosporidiosis. Am J Public Health 1992; 82(5):742-744.

ADDITIONAL RESOURCES

General disease information is available from the CDC at:
www.cdc.gov/ncidod/dpd/parasites/cryptosporidiosis/default.htm

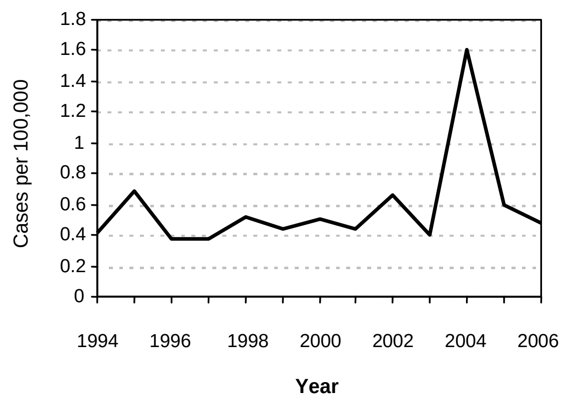
General information and reporting information about this and other foodborne diseases in LAC is available at: www.lapublichealth.org/acd/food.htm

ENCEPHALITIS

CRUDE DATA	
Number of Cases[a]	46
Annual Incidence[b]	
LA County	0.48
California	N/A
United States	N/A
Age at Onset	
Mean	25.1
Median	15.5
Range	0–80 years

[a] Excludes AIDS encephalopathy cases.
[b] Cases per 100,000 population.

Figure 1
Encephalitis
Incidence Rates by
Year of Onset
LAC 1994-2006



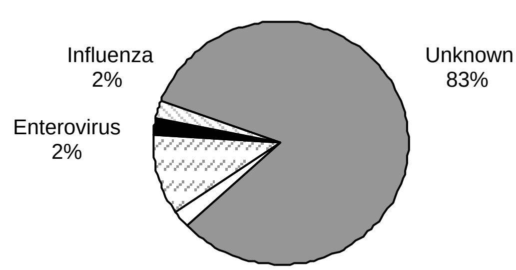
DESCRIPTION

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 with a number of different agents including viral, parasitic, fungal, rickettsial, bacterial and chemical. Public health surveillance is limited to cases of suspected or confirmed viral etiology, which includes primary and post-infectious encephalitis—but excludes individuals with underlying human immunodeficiency virus (HIV) infection. Of special concern is arboviral (mosquito-borne) encephalitis, which can be prevented by personal protection and mosquito control (See West Nile virus section). Arthropod-borne viruses (i.e., arboviruses) are viruses that 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, being maintained in complex life cycles involving a nonhuman vertebrate primary host and a primary arthropod vector. Arboviral encephalitides have a global distribution. There are five main viral agents of encephalitis in the United States: West Nile virus (WNV), eastern equine encephalitis (EEE) virus, western equine encephalitis (WEE) virus, St. Louis encephalitis (SLE) virus and La Crosse (LAC) virus, all of which are transmitted by mosquitoes.

DISEASE ABSTRACT

- In 2006, 46 viral encephalitis cases were reported. The incidence of viral encephalitis decreased from 0.59 cases per 100,000 population in 2005 to 0.48 cases per 100,000 population in 2006 (Figure 1).
- The number of reported encephalitis cases declined in 2006 by 22% compared to 2005 when 56 cases were reported. The underlying etiologies of encephalitis were identified in only 11 (24%) cases and included: 1 WNV (see WNV Report for details), 8 herpes simplex virus (HSV), 1 influenza, and 1 enterovirus (Figure 2). No deaths were reported.
- The majority of encephalitis cases occurred in children <15 years old (n=18, 39%), followed by adolescents and young adults ages 15-34 years (n=15, 33%);

Figure 2
Encephalitis by Etiology
LAC, 2006 (N=46)



adults between 35-64 years (n=8, 17%), and those more than 65 years (n=4, 9%).

- Latinos had the greatest number of encephalitis cases (n=20, 45%), followed by whites (n=11, 25%), blacks (n=8, 18%), and Asians (n=4, 9%).
- The number of reported encephalitis cases was highest in SPA 3 (n=12, 0.7 per 100,000), followed by SPAs 7 and 8 (n=8 each, 0.8 and 0.9 per 100,000, respectively), and SPA 2 (n=7, 0.3 per 100,000).

The annual incidence of acute encephalitis reported in the medical literature varies from 3.5–7.4 cases per 100,000 person-years. In 2006, the overall LAC viral encephalitis rate of 0.48 per 100,000 person-years was slightly lower than the 2005 incidence rate (.59 cases per 100,000) and rates quoted in surveillance literature. Rationale for the lower rate may be far fewer cases of WNV-associated encephalitis reported in 2006 compared to 2005; misclassification of encephalitis cases as meningitis; and underreporting of hospitalized encephalitis cases, since all reporting is passive. The case fatality from encephalitis has ranged from a high of 38% in 1997 to a low of 0% in 2006 and remains lower than the 2005 overall state case fatality rate of 12% reported by the California Encephalitis Project. The higher encephalitis mortality rate reported by the California Encephalitis Project, a California Department of Health Services' research project, may be biased as more severely ill individuals are more likely to be included in this data source. Further, cases are often reported before the final outcome of the patient is known and so the LAC record of mortality may be incomplete.

Of particular public health concern in LAC are the arthropod-borne viral (arboviral) encephalitides, SLE, WEE and WNV encephalitis, endemic to California. Since 1985, sporadic cases of SLE have been reported each year following an outbreak of 16 cases in 1984. The last confirmed SLE case in LAC was in 1997. The potential for another SLE outbreak exists, as sporadic cases in previous years and identification of SLE virus in sentinel chicken populations indicate that the virus remains endemic in LAC. Beginning in 2001, arboviral disease surveillance has included WNV, in addition to SLE and WEE.

In 2006, only 1 of 16 (6%) documented WNV infections had a clinical history compatible with encephalitis. This case was laboratory-confirmed WNV and thought to be locally acquired. In 2006, far fewer WNV associated encephalitis cases were seen compared to 2005 and 2004 when 13 and 48 cases were noted in respective years. This is consistent with overall surveillance data showing a continued decline in WNV infections over the past three years. Like SLE virus, WNV is transmitted principally by *Culex* species mosquitoes. Enhanced surveillance for early detection of virus activity in birds and mosquitoes will be crucial to guide control measures in 2006.

Prevention measures for arboviral infections consist of personal protection, screens on windows, avoiding mosquito-infested areas, especially at dusk when most mosquitoes are active, wearing protective clothing and use of insect repellants containing DEET, oil of eucalyptus and Picaridin. Elimination of standing water and proper maintenance of ponds and swimming pools decrease the available sites for hatching and maturation of mosquito larvae. Five local mosquito abatement districts monitor and control populations of these insects, especially in areas used by the public (See WNV section).

Future Directions: Surveillance for WNV infection in humans, mosquitoes, sentinel chickens, and dead birds will continue throughout the state of CA. and LAC. Research is underway to develop a WNV vaccine and treatment for humans. No human vaccine is available for SLE, WEE, and WNV. A human vaccine exists for Japanese Encephalitis.

Licensed equine (horse) vaccines are available for WEE, EEE, and WN viruses.

ADDITIONAL RESOURCES

Glaser CA, Gilliam S, Schnurr D, et al. In search of encephalitis etiologies: diagnostic challenges in the California Encephalitis Project, 1998–2000. Clin Infect Dis 2003; 36(6):731–742.

Khetsuriani N, Holman RC, Anderson LJ. Burden of encephalitis-associated hospitalizations in the United States, 1988–1997. Clin Infect Dis 2002; 35(2):175–182.

Johnston RT. Acute Encephalitis. Clin Infect Dis 1996; 23:219–226.

Nicolosi A, Hauser WA, Beghi E, Kurland LT. Epidemiology of central nervous system infections in Olmsted County, Minnesota, 1950–1981. J Infect Dis 1986; 154(3):399–498.

Trevejo RT. Acute encephalitis hospitalizations, California, 1990-1999: unrecognized arboviral encephalitis? Emerg Infect Dis 2004; 10(8):1442-1449.

For information on mosquito-borne encephalitis: www.cdc.gov/ncidod/dvbid/arbor/index.htm.

For information for consumers: www.nlm.nih.gov/medlineplus/encephalitis.html

For more detailed information such as causal information and effective management strategies: www.postgradmed.com/issues/1998/03_98/guti.htm

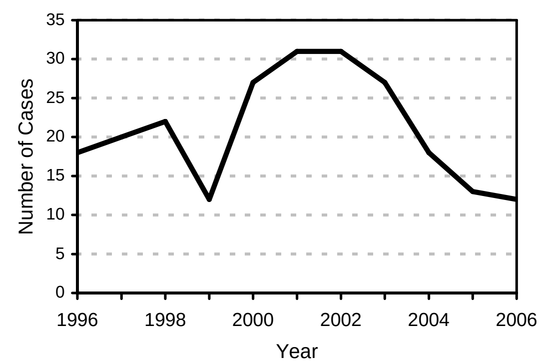
Information about case investigation of encephalitis in LAC is available at: www.lapublichealth.org/acd/procs/b73/b73index.htm

ESCHERICHIA COLI O157:H7 / HEMOLYTIC UREMIC SYNDROME

CRUDE DATA	
Number of Cases	12
Annual Incidence[a]	
LA County	--- [b]
California	25
United States	
Age at Diagnosis	
Mean	10.4
Median	7
Range	1-27 years

[a] Cases per 100,000 population.
[b] Rates based on less than 19 observations are unreliable.

Figure 1
Escherichia coli O157:H7
Cases by Year of Onset
LAC 1996–2006*



DESCRIPTION

Escherichia coli O157:H7, a Gram-negative bacillus, is a specific serotype of the Shiga toxin producing class of *E. coli* (STEC) and the most common such serotype in the US. Incubation period is 2-8 days. Shiga toxins cause abdominal cramps and watery diarrhea, often developing into bloody diarrhea; fever is uncommon. Likely modes of transmission include foodborne (e.g., undercooked ground beef, fresh produce, unpasteurized juice, and raw milk) and person-to-person (e.g., day-care settings). There also have been outbreaks associated with exposure to animals and their environments and recreational water exposure. All *E.coli* O157:H7 isolates are confirmed and fingerprinted by the Los Angeles County Public Health Laboratory and submitted to the national Pulse-Net database.

Hemolytic uremic syndrome (HUS) is a clinical diagnosis often associated with *E. coli* O157:H7. Children younger than five years of age are at highest risk for HUS, a clinical complication consisting of hemolytic anemia, thrombocytopenia, and kidney failure. Adults may develop thrombotic thrombocytopenic purpura after STEC infection.

DISEASE ABSTRACT

- There was a decrease in confirmed cases in 2006.
- There were no LAC outbreaks in 2006.

STRATIFIED DATA

Trends: After peaking in 2001 and 2002, rates of *E.coli* O157:H7 infection have been steadily decreasing. This is the third year with fewer than twenty cases in LAC since 1999 (Figure 1). There were eight cases of HUS in addition to the 12 cases of O157.

Seasonality: In 2006, 58% of confirmed cases occurred during the summer with a peak of three cases in September (Figure 2). This is consistent with the 5-year average, although the peak was later in the summer months.

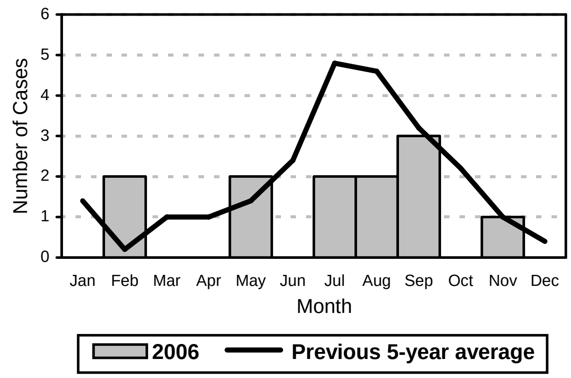
Age: In 2006, there were more cases in children (67%; n=8) than in adults. There were two family clusters involving siblings. One family cluster involving two siblings both with O157:H7 isolated and the second family cluster involved one sibling with O157:H7 and the other with HUS only (without lab confirmation of 0157:H7 infection). All other cases were sporadic and not linked to an outbreak.

Sex: The male to female ratio was 1:1.

Race/Ethnicity: Eight cases were reported in whites, three in Latinos, and one in Asians. There were no confirmed cases among blacks.

Location: SPA 2 had six confirmed cases, all unrelated. SPA 3 had three cases but they were unrelated. SPAs 5, 7, and 8 had one case each.

Figure 2
Esherichia coli O157:H7
Cases by Month of Onset
LAC, 2006



Severity of Illness: Most cases (75%; n=9) reported bloody diarrhea and abdominal cramps, and only two cases reported having fever (mean temperature was 101.0^{0}F). Two cases (16%) required hospitalization. There were no reported deaths in confirmed cases.

Risk Factors: In the week prior to onset, cases with available information reported the consumption of raw milk (8%), ground beef (25%), steak (25%), fast food (75%) or food from other types of restaurants (16%). Eight percent (N= 1) traveled to Texas. One confirmed case received antibiotic therapy, which increases the risk of HUS, but did not develop HUS.

HUS: In 2006, there were eight reported HUS cases without lab confirmation of *E. coli* O157:H7 infection. All eight cases were one to four years of age. All cases required hospitalization with no deaths. No cases reported any recent antibiotic therapy prior to the onset of HUS. Two cases required dialysis. No case reported the consumption of raw milk; however consumption of ground beef (50%) steak (25%), cider (12%), and lettuce (12%) was reported. No travel was reported.

COMMENTS

There were six cases of other STEC (non-O157:H7) reported with different serotypes. There were no outbreaks related to *E. coli* O157:H7 in LAC during 2006.

Collaborative efforts among physicians, laboratories and the health department are important for enhancement of surveillance. Physicians should request testing for *E. coli* O157:H7 or Shiga toxin on all bloody stools, and consider *E. coli* O157:H7 in their diagnoses by asking about consumption of high-risk foods, attendance at day-care centers or farms, and exposure to other individuals with diarrhea. The collection of detailed food histories is important to understand underlying sources of infection. All cases of HUS should be reported immediately and physicians should request stool testing for *E.coli* O157:H7 for these patients.

PFGE has been helpful in detecting clusters of *E. coli* O157:H7. PulseNet is a nationwide network of laboratories that perform PFGE, or “DNA fingerprinting” of foodborne bacteria. This network permits rapid comparison of fingerprint patterns to identify clusters and enhance outbreak investigation.

PREVENTION

Increased public education to prevent STEC infection is needed. Information should focus on safe food handling practices, proper hygiene and identifying high-risk foods and activities both in the 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 water during swimming or wading. Careful handwashing is essential, especially before eating and after handling raw beef products or coming in contact with or being around animals. The strengthening of national food processing regulations to decrease contamination is also important to reduce infection.

ADDITIONAL RESOURCES

General information about this disease can be found at:
www.cdc.gov/ncidod/diseases/submenus/sub_ecoli.htm.

Foodborne disease active surveillance is available from FoodNet (CDC) at: www.cdc.gov/foodnet.

Information from the Gateway to Government Food Safety is available at: www.foodsafety.gov.

Information about outbreaks (nationwide) is available from the Outbreak Response and Surveillance Team of the CDC at: www.cdc.gov/foodborneoutbreaks/index.htm.

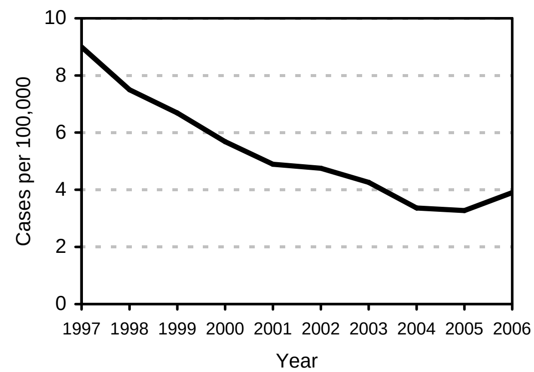
General information and reporting information about this and other foodborne diseases in LAC is available at: www.lapublichealth.org/acd/food.htm.

GIARDIASIS

CRUDE DATA	
Number of Cases	376
Annual Incidence[a]	
LA County	3.9
California	6.37[b]
United States	6.39[b]
Age at Diagnosis	
Mean	31
Median	30
Range	<1–89 years

[a] Cases per 100,000 population.
[b] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

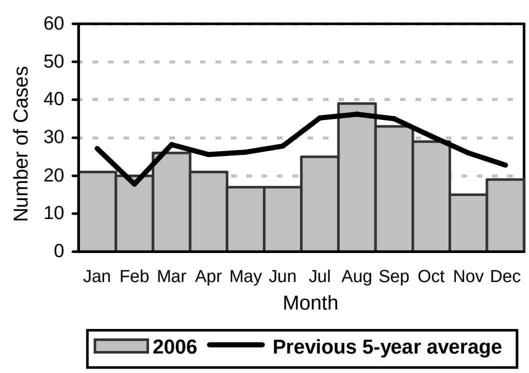
Figure 1
Giardiasis
Incidence Rates by Year of Onset
LAC, 1997–2006



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 usually 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 in day care represent a reservoir of disease in developed countries. There is no vaccine.

Figure 2
Giardiasis
Cases by Month of Onset
LAC, 2006



DISEASE ABSTRACT

- The incidence of reported Giardiasis in Los Angeles County has dropped dramatically over the past 10 years, and has remained low for the past 4 years.
- The incidence of Giardiasis in 2006 increase from 2005 (3.3 per 100,000) by 18%, primarily due to a 34% increase in the incidence rate for men (from 3.8 to 5.3 per 100,000).
- Incidence tends to increase during summer months when high-risk activities such as recreational water exposure also increase.

STRATIFIED DATA

Trends: Giardiasis incidence in LAC remains low in 2006 relative to the last 10 years, and the incidence has been reduced by over 50% since 1997 (Figure 1).

Seasonality: The number of cases typically increases during summer months when recreational exposure is more likely (i.e., swimming in infected pools, lakes, etc.) (Figure 2).

Age: As in previous years, the highest age-specific incidence rate occurred among children aged 1–4 years (8.1 cases per 100,000) (Figure 3).

Sex: Males are more then twice as likely to contract *Giardia* than females in 2006 (2.2:1), an increase from that seen in 2005 (1.4:1). The incidence for men in 2006 (5.3 per 100,000) increased from 2005 (3.8 per 100,000) by 34% where as the rate for woman dropped by 12% (2.7 to 2.4 per 100,000).

Race/Ethnicity: Whites continue to have higher race/ethnicity specific incidence rates (5.2 per 100,000) than other races (Figure 4).

Location: SPA 5 (West Area) had the highest reported incidence (6.9 per 100,000) followed by SPA 2 (San Fernando Area) (5.8 per 100,000).

Figure 3
Giardiasis
Incidence Rates by Age Group
LAC 2006

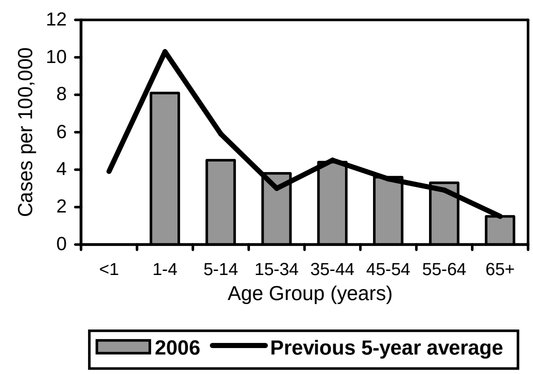
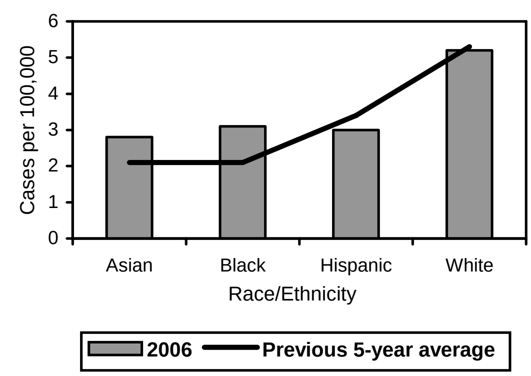


Figure 4
Giardiasis
Incidence Rates by Race/Ethnicity
LAC, 2006



COMMENTS

There has been a considerable decline in incidence of *Giardia* over the past decade. While the specific reasons for this decrease are unknown, several factors may have contributed including advances in food and water safety as well as improved education about safety regarding recreational water (i.e., avoiding drinking lake and pool water, keeping babies in diapers and individuals with diarrhea from swimming in public facilities). There was no outbreak reported in 2006.

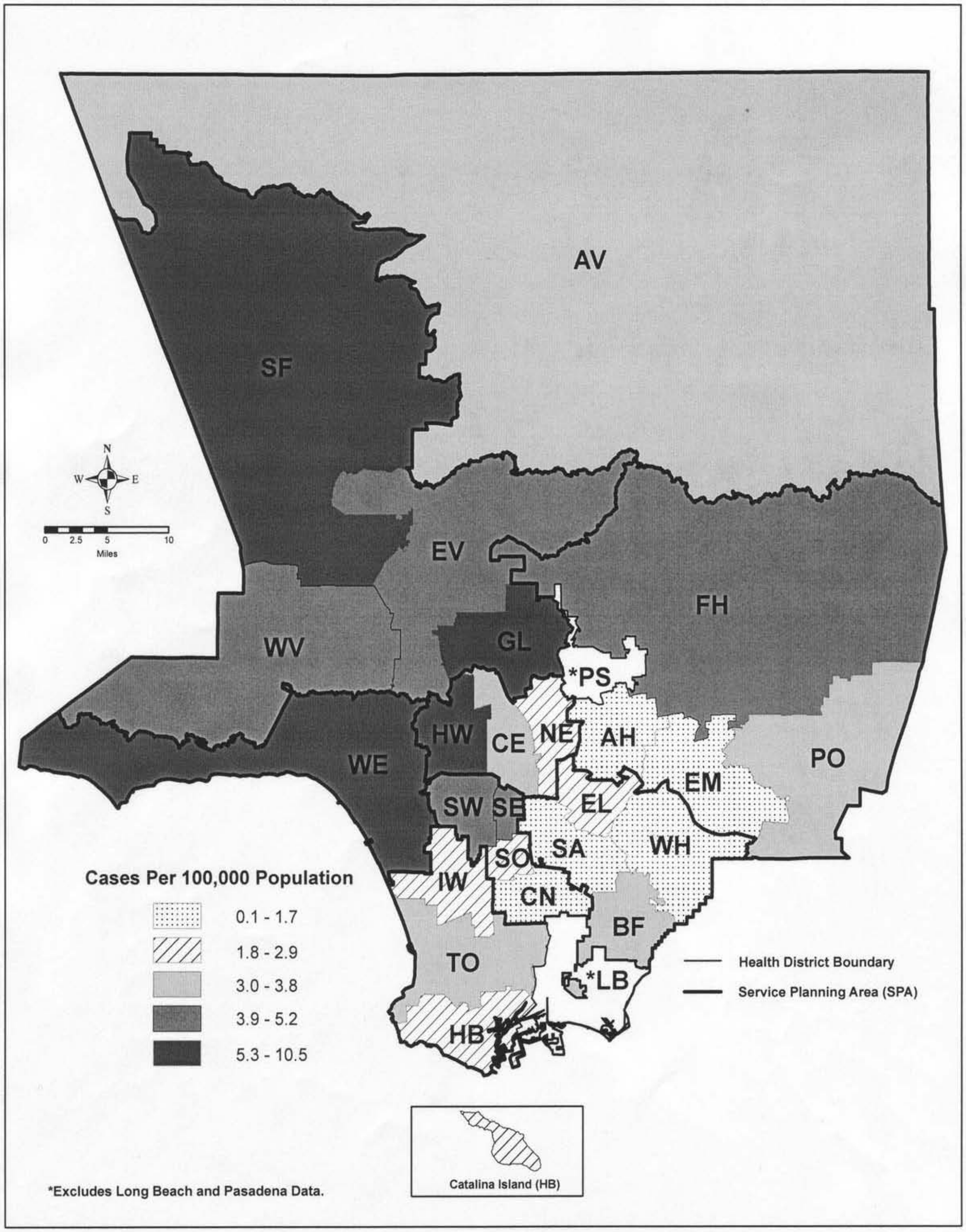
ADDITIONAL RESOURCES

CDC. Giardiasis surveillance--United States, 1992–1997. MMWR 2000; 49(SS07):1–13. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/ss4907a1.htm

CDC. Parasitic Disease Information Fact Sheet—Giardiasis. Available at: www.cdc.gov/ncidod/dpd/parasites/giardiasis/factsht_giardia.htm

CDC. Surveillance for foodborne-disease outbreaks--United States, 1998—2002. MMWR 2006; 55(SS10):1-34. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/ss5510a1.htm

Map 5. Giardiasis
Rates by Health District, Los Angeles County, 2006*



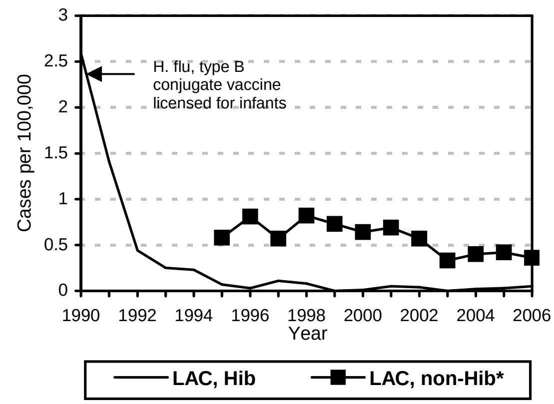
*Excludes Long Beach and Pasadena Data.

HAEMOPHILUS INFLUENZAE INVASIVE DISEASE

CRUDE DATA	
Number of Cases	66
Annual Incidence[a]	
LA County	0.68
California	0.11[b,c]
United States	0.82[c]
Age at Diagnosis	
Mean	59.0
Median	66.0
Range	<1 – 98.0

[a] Cases per 100,000 population.
[b] Cases per 100,000 persons, aged less than 30 years. In California, *H. influenzae* among persons > 29 years of age is not reportable.
[c] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

Figure 1
H. influenzae Invasive Disease
Incidence Rates by Year
LAC, 1990–2006

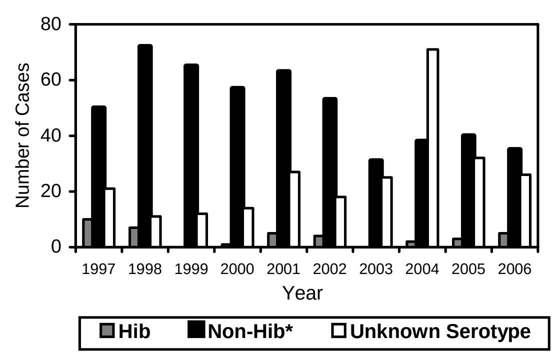


* Surveillance became mandated in 1995. Excludes unknown serotypes

DESCRIPTION

Haemophilus influenzae is a Gram-negative coccobacillus that can cause both invasive and non-invasive disease. *H. influenzae* invasive disease includes meningitis, sepsis, pneumonia, cellulitis, and septic arthritis. Currently, the disease primarily affects infants and the elderly, as well as immunocompromised individuals and those who have abnormal splenic function. *H. influenzae* can be transmitted by respiratory secretions of individuals colonized in the oropharynx with the organism. There are six encapsulated, typeable strains (a–f) and unencapsulated, nontypeable strains of *H. influenzae*. Prior to the introduction of the H. *influenzae* type b (Hib) conjugate vaccine in 1990, most cases of invasive disease in children were caused by type b. *H. influenzae* type b is the only serotype that is vaccine-preventable and for which chemoprophylaxis is effective.

Figure 2
H. influenzae Invasive Disease
Cases by Year of Onset
LAC, 1997-2006



* Surveillance became mandated in 1995. Excludes unknown

DISEASE ABSTRACT

- Of the 5 Hib cases identified in 2006, none were completely vaccinated.
- The epidemiology of H. *influenzae* invasive disease is now being shaped by non-Hib and unknown serotypes (Table 1, Figure 2, Figure 3).
- Like previous years, non-Hib incidence peaked during the months of January to March.

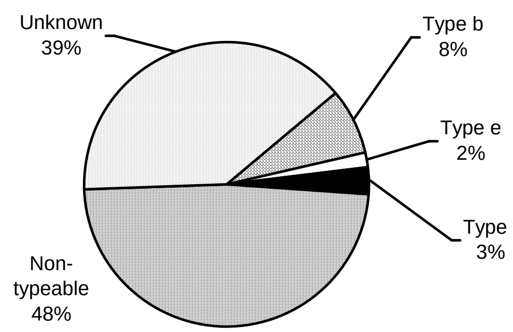
Table 1: *H. influenzae* Crude Data by Serotype, 2006 vs. Previous 5-Year Average

	B		Non-Hib		Unknown type	
	2006	Previous 5-Year Average	2006	Previous 5-Year Average	2006	Previous 5-Year Average
No. of Cases	5	2.8	35	45.2	26	36.0
Age at Onset						
Mean	30.8	23.4	58.4	41.4	65.3	61.8
Median	34.0	14.0	71.0	40.5	65.0	67.3
Range	<1 – 73.0	1.0 – 60.5	<1 – 92.0	<1 - 92.4	18.0 – 98.0	10 – 97.2
LAC Case Fatality	0%	14.3%	11.4%	6.6%	7.7%	6.3%

IMMUNIZATION RECOMMENDATIONS

- All infants, including those born prematurely, can receive a primary series of conjugate Hib vaccine beginning at 2 months of age. The number of doses in the series depends on the brand of vaccine used. A booster is recommended at 12-15 months regardless of which brand of vaccine is used for the primary series.
- Individuals older than 59 months of age do not need Hib vaccination unless they have a health condition that puts them at increased risk for invasive Hib disease.

Figure 3
H. influenzae Invasive Disease
Percent Cases by Serotype
LAC, 2006 (N=66)



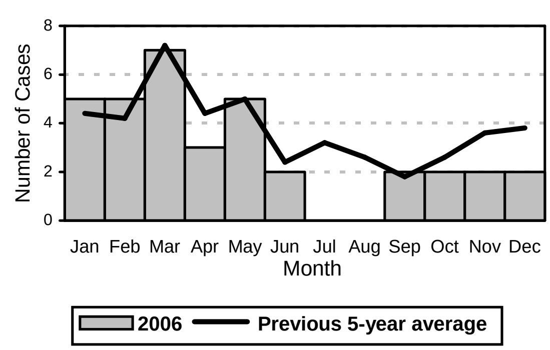
STRATIFIED DATA

Seasonality: The 5 Hib cases had disease onset in January (n=1), February (n=1), October (n=1), and December (n=2). Similar to previous years a temporal pattern has been evidenced in LAC, with a peak in non-Hib cases during the months of January to March. These three months accounted for 48.6% (n=17) of the non-Hib cases (Figure 4).

Sex: The male-to-female ratio of Hib, non-Hib, and unknown serotype cases was 1.5:1, 1.3:1, and 1.2:1 respectively.

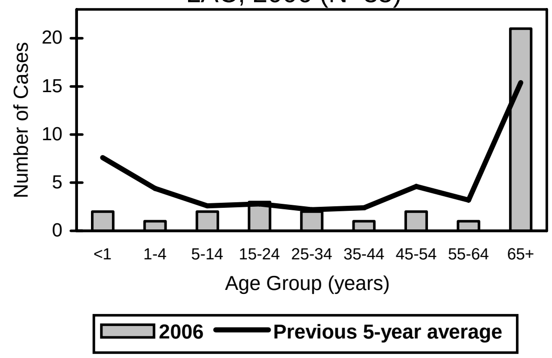
Age: Of the 5 Hib cases, two were less than 6 months of age, while the remaining three were 34, 47, and 73 years of age. The number of non-Hib cases by age in 2006 followed the trend of the previous five years – the 65+ age group (60%, n=21) remaining the most affected by non-Hib invasive disease (Figure 5). Only 9% (n=3) of non-Hib cases were under the age of 5. Of the 26 cases with unknown serotype, 96% (n=25) were over the age of 30 and were not actively investigated for serotype as detailed in LAC's priority investigation criteria. In addition, 50% (n=13) of these unknown serotype cases were in the 65+ age group.

Figure 4
H. influenzae Invasive Disease
Cases Due to Non-Hib Serotype
by Month of Onset
LAC, 2006 (n=35)



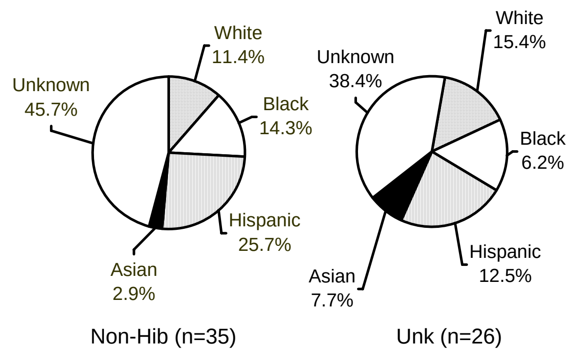
Race/Ethnicity: Two of the Hib cases were Hispanic, one was white, one was black, and one's race was unknown. Among the non-Hib cases where the race/ethnicity was known (n=19), Hispanics accounted for 47% (n=9) of the cases, followed by blacks (n=5, 26%), whites (n=4, 21%) and Asians (n=1, 5%). Among the unknown serotype cases of whom race/ethnicity was identified (n=16), 38% were among Hispanics (n=6), followed by whites (n=4, 25%), blacks (n=4, 25%), and Asians (n=2, 12%). (Figure 6.)

Figure 5
H. influenzae Invasive Disease
Serotype Non-Hib Cases by Age Group
LAC, 2006 (N=35)



Location: The 5 Hib cases resided in SPA 2 (n=1), SPA 5 (n=1), and SPA 7 (n=3). The number of non-Hib cases per SPA ranged from 3 to 7. SPA 6 accounted for 7 non-Hib cases. San Fernando Valley (SPA 2) accounted for 6 cases. San Gabriel Valley (SPA 3) and East (SPA 7) accounted for 5 non-Hib cases each. South Bay (SPA 8) had 4 cases followed by Metro (SPA 4) and West (SPA 5) with 3 cases each. An additional 5% (n=2) of non-Hib cases had no identified SPA. The number of unknown serotype cases per SPA ranged from 2 to 7, with SPA 5 accounting for 7 cases. SPA 2 accounted for 4 cases. SPA 4 and SPA 6 accounted for 3 cases each followed by SPA 1, SPA 3, SPA 7, and SPA 8 with 2 cases each. One unknown serotype case did not have a residence indicated.

Figure 6
H. influenzae Invasive Disease
Percent Cases by Race/Ethnicity
LAC, 2006



COMMENTS

The only cases of *H. influenzae* disease investigated in LAC in 2006 are those in persons less than 30 years of age. Contacts of these cases are investigated and chemoprophylaxis is given when appropriate.

Rates of invasive Hib disease in children have decreased to extremely low levels since Hib vaccines became available in 1990. Among the 66 H. *influenzae* cases, only 5 (8%) were Hib cases and only 2 (3%) were less than 30 years of age. None of the cases had any known exposure to a confirmed/suspected case. Four Hib cases were hospitalized indicating the severity of type B disease.

Only one of the Hib cases (the 5-month-old) was vaccinated. Although the child was not up-to-date with immunizations, the child was too young to have completed a three-dose primary vaccination series and would not have developed protective antibody levels.

<u>Case Fatalities</u>: There were six fatalities among *H. influenzae* cases: four were non-Hib cases and two were unknown serotypes. One of the fatalities was a premature baby of a substance-abusing mother who subsequently died from various complications. The other five fatalities (83%) were in persons over the age of 30 so the cases were not investigated for further details. However, information on complications was provided for three cases; two of the cases had pneumonia and one had meningitis. Males accounted for four of the six (66.7%) case fatalities. Three of the fatalities were Hispanic, two were white, and one was of unknown race/ethnicity.

ADDITIONAL RESOURCES

Additional information about *Haemophilus* influenzae invasive disease is available at:

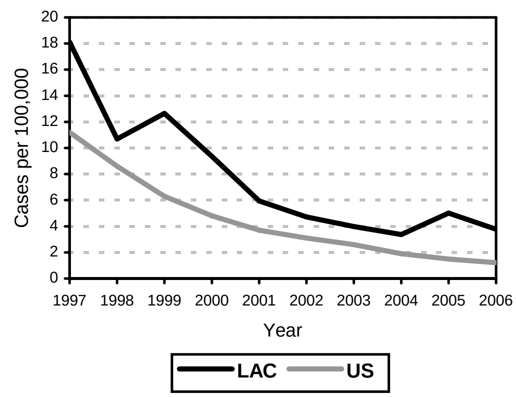
- National Immunication Program – www.cdc.gov/vaccines
- Immunization Action Coalition – www.immunize.org
- LAC Immunization Program – www.lapublichealth.org/ip
- Acute Communicable Disease Control Program – http://lapublichealth.org/acd/procs/b73/b73index.htm

HEPATITIS A

CRUDE DATA	
Number of Cases	364
Annual Incidence[a]	
LA County	3.77
California	2.75[b]
United States	1.21[b]
Age at Diagnosis	
Mean	41
Median	41
Range	1-100 years

[a] Cases per 100,000 population.
[b] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

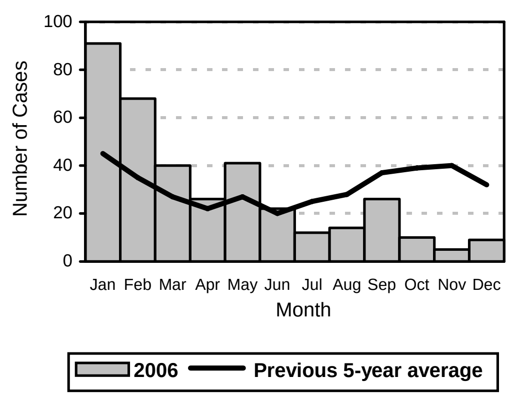
Figure 1
Hepatitis A
Incidence Rates by Year
LAC and US, 1996–2006



DESCRIPTION

Hepatitis A virus (HAV), a RNA-virus of the Picornaviridae family, is a vaccine-preventable disease transmitted fecal-orally, person-to-person, or through vehicles such as food. Signs and symptoms of acute hepatitis A include fever, malaise, dark urine, anorexia, nausea, and abdominal discomfort, followed by jaundice. Many cases, especially in children, are mild or asymptomatic. Sexual and household contacts of HAV-infected persons are at increased risk for getting the disease. The average incubation period is 28 days (range 15–50 days). Recovery usually occurs within one month. Infection confers life-long immunity.

Figure 2
Hepatitis A
Cases by Month of Onset
LAC, 2006



ACDC uses the CDC/CSTE criteria for acute hepatitis A to standardize surveillance of this infection. The criteria include: 1) an acute illness with discrete onset of symptoms and 2) jaundice or elevated aminotransferase levels, and 3) appropriate lab tests to confirm laboratory criteria for acute hepatitis A diagnosis: IgM anti-HAV positive, or a case meets the clinical case definition and has an epidemiologic link with a person who has laboratory confirmed hepatitis A (i.e., a household or sexual contact of an infected person during the 15–50 days before the onset of symptoms).

DISEASE ABSTRACT

- The incidence rate of acute hepatitis A has decreased from the previous year (5.01 to 3.77 per 100,000) (Figure1).
- The hepatitis A incidence rate in blacks and in those between the ages of 35-54 increased in 2006 from 2005.
- There were two outbreaks of hepatitis A in 2006.

STRATIFIED DATA

Trends: The hepatitis A incidence rate was 3.77 cases per 100,000 in 2006 which was lower than last year (Figure 1).

Seasonality: Historically, there is an increase of hepatitis A cases in summer to early autumn, but 2006 was different than the previous five-year average (Figure 2).

Age: The overall mean age for hepatitis A cases in 2006 was 41 years. The mean age differed significantly by race and ethnic groups. The mean age for Latinos was 30 years while Asian, black, and white cases had mean ages of 43, 46, and 48 years, respectively. Historically, the age-specific rate has been highest in children aged 5-14 years and 65 and older. However, in 2006, the rate was highest among those 35-54 years (Figure 3).

Sex: The hepatitis A cases male-to-female rate ratio was 2:1. Among Asian cases, the male-to-female rate ratio was 1:1, while among Latino, white, and black cases, incidence rate ratios were higher among males, at 1.2:1, 2.5:1, and 4:1 respectively.

Race/Ethnicity: Compared to the previous five-year average, the incidence rate for blacks is for the first time higher than other races (7.6 per 100,000), followed by whites (4.3), Latinos (2.7), and Asians (2.0), respectively (Figure 4).

Location: Of the eight SPAs across LAC, two had rates that were greater than the overall county mean rate for this disease: SPA 4 (6.3 per 100,000) and SPA 8 (4.0 per 100,000) (Figure 5).

Severity of Illness: Among all hepatitis A cases in 2006, there was one reported fatality. Twenty-seven percent (n=98) of hepatitis A cases were hospitalized. The age of those hospitalized ranged from 1 to 92 years, with a median age of 43.

Risk Factors: Of the 364 confirmed cases, 88% were interviewed by public health nurses for risk factors. Risk factors were identified for only 40% (n=128) of the cases (including some cases with multiple risk factors). Of those with identified risk factors, recent travel outside of the US (n=59, 46%) was the most common risk factor reported in 2006, followed by eating raw shellfish (n=45, 35%), and being in contact with another case (n=26, 20%), and MSM (n=18, 14%), respectively (Figure 6). Among travelers, Mexico and Central American destinations (75%) were most frequently cited.

Figure 3
Hepatitis A Incidence Rates
by Age Group LAC, 2006 (N=364)*

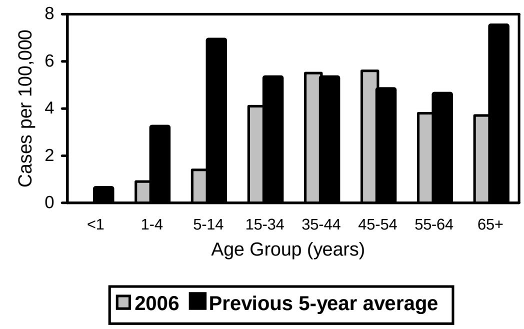


Figure 5 Hepatitis A Incidence Rates
by SPA LAC, 2006

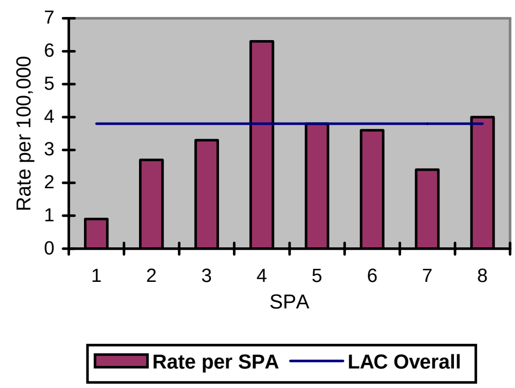
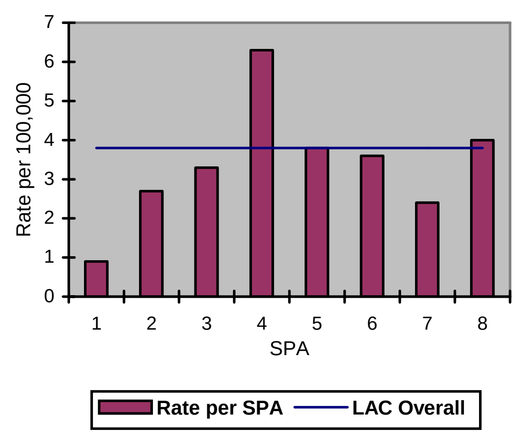


Figure 5 Hepatitis A Incidence Rates
by SPA LAC, 2006

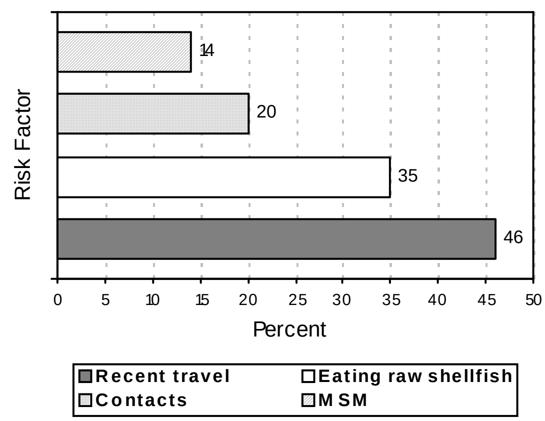


PREVENTION

Effective strategies for decreasing the number of hepatitis A cases in LAC include adding hepatitis A vaccine to the children's immunization program and public health nurses providing immune globulin (IG) to close contacts of cases and educating clients about the importance of hand hygiene on reducing infections when cases of acute hepatitis A are reported to Public Health.

Post-exposure prophylaxis with IG is used to control outbreaks in LAC. It has been suggested that outbreaks of HAV could also be effectively interrupted through vaccine use, leading to sustained reduction in disease incidence.

Figure 6
Hepatitis A Reported Risk Factors
LAC, 2006



***Includes cases identifying multiple risks.**

COMMENTS

Rates of acute hepatitis A have varied widely in the past several years, despite an overall decline of acute hepatitis A in the US. What follows is an account of the changes in the true incidence of disease, coupled with a change in case definitions, that explain the fluctuations in acute hepatitis A in LAC.

Prior to 1998, the highest rate of acute hepatitis A occurred in those 5-14 years in LAC, especially among Latino children. However, with the inclusion of hepatitis A vaccine into Vaccine for Children's Program in 1999, the rate of acute hepatitis A in children decreased. The decrease of hepatitis A in children was the major source of the decrease of hepatitis A in the population as a whole from 1999-2004 in LAC. With the decrease in the rate of hepatitis A in children, the number of cases in adults also decreased but increased as a proportion of the total number of cases of hepatitis A in LAC.

In LAC, prior to 2005, hepatitis A cases were often counted as "acute" even if the only information received about the patient was a positive IgM test. However, many other jurisdictions have documented "false positive" results on the IgM test, especially in the elderly who often receive screening tests despite lack of symptoms or medical indication. Therefore, since January 1, 2005, we have been consistently applied the CDC/CSTE criteria to all reported cases of acute hepatitis A The effect of consistently applying this more stringent case definition, which includes clinical and laboratory findings in addition to a single serological test, was to remove those reported cases who lacked evidence of clinical symptoms or liver damage. Utilizing the standardized case definition, the rate of acute hepatitis A dropped even more than the expected drop due to the use of the vaccine. The number of cases in all age groups, especially those aged >65 years, decreased. This was expected as many of the initial reports in the older adult population, based on a single positive laboratory test, were felt to be due to over aggressive screening and not due to newly acquired infection.

However, from August 2005 to July 2006, LAC sustained a 12 month community-wide outbreak of acute hepatitis A. The overall rate increased from 3.37 in 2004 to 5.01 in 2005, despite a more restrictive case definition of acute hepatitis A. If the new definition had not been implemented in 2005, it is anticipated that the 2005 and 2006 incidence rate of acute hepatitis A during the community outbreak would have been even larger. Even so, it is remarkable that the rate increased during this time of steadily decreasing rates nationwide and in California. While the outbreak affected most race/ethnicity groups and geographic regions of the county, the proportion of hepatitis A cases increased in blacks and in those ages 15-54 years. Furthermore, 11% of the cases during this time period occurred in the homeless, a population which is estimated to comprise only 1% of county's total population.

As the community-wide outbreak came to an end during the summer of 2006, the rate of hepatitis A again fell to below historical levels. This can be best appreciated in Figure 2 where the number of cases reported each month from July to December is below the previous 5-year average. The discrepancy between the July to December 2006 cases and the previous 5-year average is large because the previous 5-year average is calculated including cases that were considered “confirmed” under the previous, less restrictive, case definition used before 2005 as well as outbreak cases that occurred during August to December 2005. (See 2005 Special Studies Report on Acute Hepatitis A for more information.)

In LAC, prior to 2005, the age-specific rate has been highest in children aged 5-14 years and 65 and older. However, using the CDC/CSTE acute hepatitis A criteria, in 2006 the rate was highest among those 35-54 years (Figure 2), consistent with 2005.

During the outbreak period of 2005 (August through December), cases in blacks increased. This trend continued into the first half of 2006. The hepatitis A incidence rates among the blacks was almost double that compared to the previous 5-year average (7.6 versus 3.2 per 100,000).

There were 11% (n= 41) of acute cases identified as homeless. 42% of them were black males (n=17). After identifying the homeless as a disproportionately affected group, LAC DPH did an outreach project to collaborate with the downtown homeless organizations to provide education and hepatitis A vaccine for food service providers at the downtown Skid Row area.

In 2006, ACDC investigated two hepatitis A outbreaks, associated with licensed food and drink establishments. The first was reported in June 2006; 7 cases with onset in May were identified in patrons of a cocktail lounge. After investigation by DPH Environmental Health Food and Milk Program, District Public Health staff, and ACDC, a contaminated ice chest was a suspected source. The ice was probably contaminated by a patron who had been diagnosed with acute hepatitis A in April and was known to take ice and drinks from the public chest with his bare hands. In September 2006, 8 cases of acute hepatitis A were identified in patrons who ate at a restaurant in Pomona during August. A case-control study was unable to identify the source of the outbreak. It is most likely that an intermittent source of hepatitis A, such as an asymptomatic food worker or contaminated food product, or an external contamination of publicly available food (such as the salsa bar) was the source of this outbreak. Outbreaks of hepatitis A without a clear source being identified are commonly reported.

PREVENTION

International travel was the most common risk factor reported in 2006, followed by eating raw shellfish and contact with a household member or sexual partner who had HAV, and MSM. Therefore, it is important to educate travelers, consumers of raw shellfish, and MSM about hepatitis A vaccinations. Sustaining and further reducing hepatitis A incidence can be achieved by improving vaccination coverage in all US children starting at 2 years of age. Increased awareness of the public about the mode of hepatitis transmission and the importance of good personal hygiene may also lead to a significant reduction in disease incidence.

ADDITIONAL RESOURCES

General information about hepatitis is available from the CDC at:
www.cdc.gov/ncidod/diseases/hepatitis/a/index.htm

<u>Publications</u>:

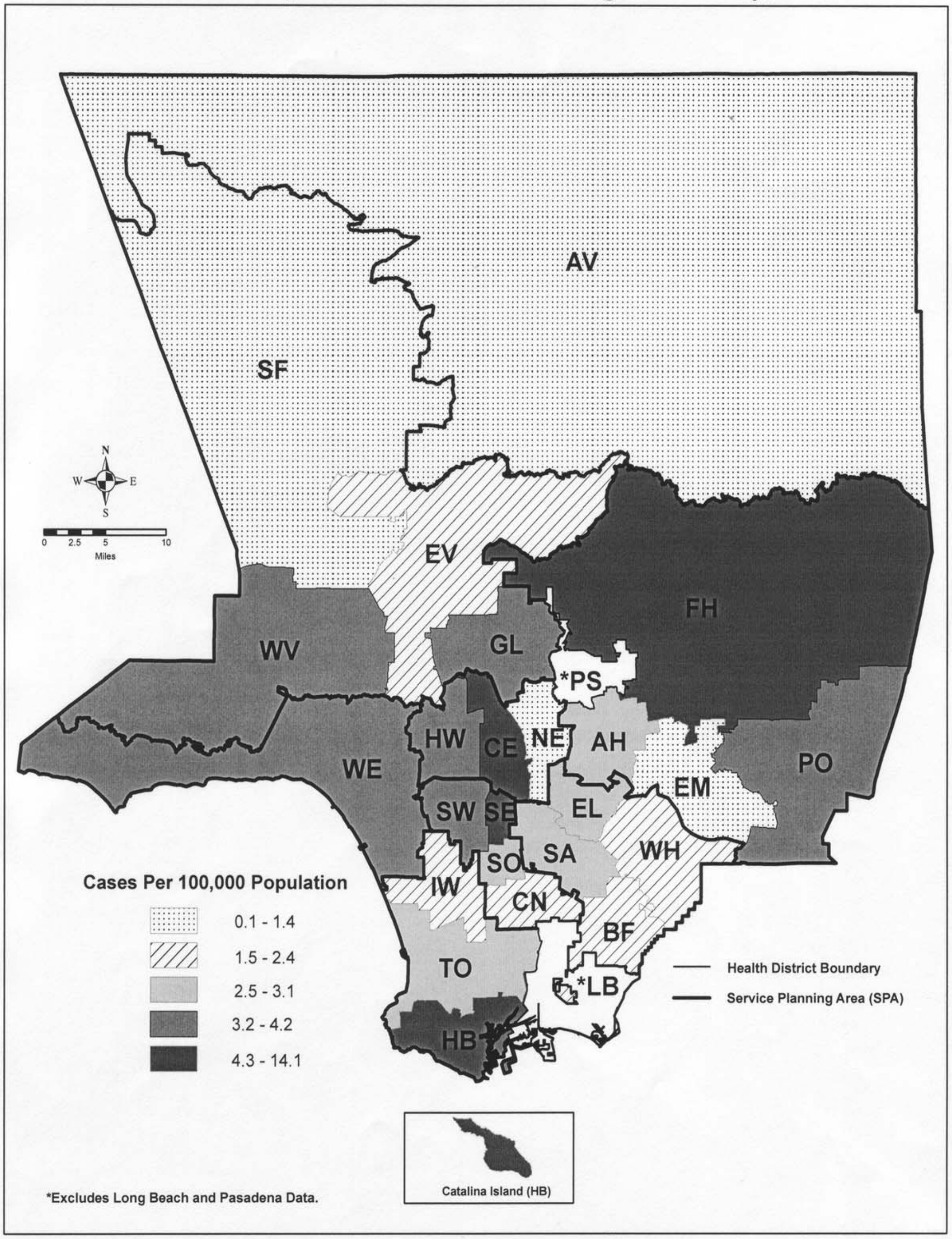
CDC. Prevention of hepatitis A through active or passive immunization: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 2006; 55(RR07):1-23.
Available at: www.cdc.gov/mmwr/preview/mmwrhtml/rr5507a1.htm

CDC. Surveillance for acute viral hepatitis--United States, 2005. MMWR 2007; 56(SS03):1-24.
Available at: www.cdc.gov/mmwr/preview/mmwrhtml/ss5603a1.htm

CDC. Hepatitis A outbreak associated with green onions at a restaurant--Monaca, Pennsylvania, 2003. MMWR 2003; 52(47):1155-1157. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/mm52d1121a1.htm
CDC. Positive test results for acute hepatitis A virus infection among persons with no recent history of acute hepatitis--United States, 2002-2004. MMWR 2005; 54(18):453-456. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/mm5418a1.htm

CDC. Foodborne transmission of hepatitis A--Massachusetts, 2001. MMWR 2003; 52(24):565-567. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/mm5224a2.htm

Map 5. Hepatitis A
Rates by Health District, Los Angeles County, 2006*

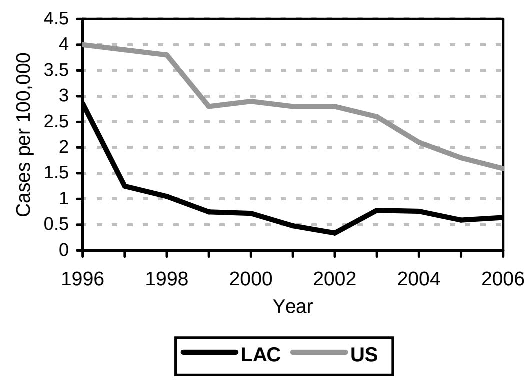


HEPATITIS B, ACUTE (NON-PERINATAL)

CRUDE DATA	
Number of Cases	62
Annual Incidence[a]	
Los Angeles	0.64
California	1.18[b]
United States	1.59[b]
Age at Diagnosis	
Mean	41
Median	41
Range	15-84 years

[a] Cases per 100,000 population.
[b] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

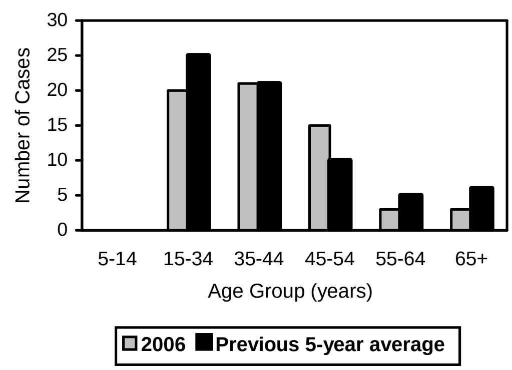
Figure 1
Hepatitis B
Incidence Rates by Year
LAC and US, 1996–2006



DESCRIPTION

Hepatitis B is a vaccine-preventable disease transmitted through parenteral or mucous membrane exposure (via sex or drugs) to the blood and other bodily fluids of individuals infected with the hepatitis B virus (HBV), a DNA-virus of the Hepadnaviridae family. It is also spread from mother to child at birth or soon after birth. Symptoms, which occur in less than half of those acutely infected, may be very mild and flu-like: anorexia, nausea, fatigue, abdominal pain, muscle or joint aches, jaundice and mild fever. Approximately 2–10% of adults infected with HBV are unable to clear the virus within six months and become chronic carriers. Death from cirrhosis or liver cancer is estimated to occur in 15–25% of those with chronic infection. Overall, hepatitis B is more prevalent and infectious than HIV.

Figure 2
Acute Hepatitis B
Number Cases by Age Group
LAC, 2006



For the purpose of surveillance, ACDC uses the CDC/CSTE criteria for acute hepatitis B. The criteria include: 1) discrete onset of symptoms and 2) jaundice *or* elevated aminotransferase levels, and 3) appropriate laboratory tests to confirm acute hepatitis B diagnosis (i.e., HBsAg positive or anti-HBc IgM positive, if done, and anti-HAV IgM negative, if done).

DISEASE ABSTRACT

- The incidence rate for acute hepatitis B slightly increased from the previous year (Figure 1); there were 62 cases confirmed for 2006 versus 57 cases in 2005.
- The greatest numbers of confirmed acute cases were in persons aged 15-44 years and the majority of cases were males.
- Multiple sex partners, predominately in MSM, remain the most frequently identified risk factor.
- No outbreaks were reported in 2005.

STRATIFIED DATA

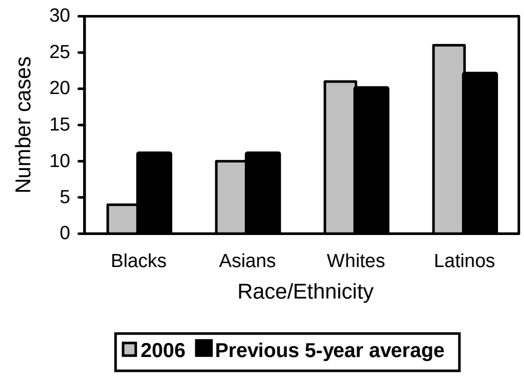
Seasonality: None.

Age: Cases ranged in age from 15 to 84 years (the median age was 41) with 66% occurring in those aged under 45 years (Figure 2).

Sex: The male-to-female rate ratio was 3.8:1. The number of cases in males exceeded those in females in all ethnic groups.

Race/Ethnicity: The highest number of cases was seen in Latinos (n=26) which is consistent with the previous five-year average, followed by whites (n=21), Asians (n=10), and blacks (n=4) respectively (Figure 3).

Figure 3
Acute Hepatitis B
Number cases by Race/Ethnicity*
LAC, 2006



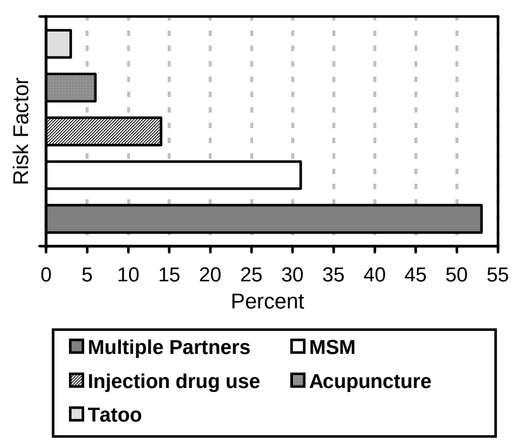
* Excludes perinatal cases.

Location: SPA 4 (n=16) had the most cases, followed by SPA 2 (n=15), SPA 3 (n=6), SPA 6 (n=6), SPA 7(n=6), SPA 8 (n=6), SPA 5 (n=3), and SPA 1 (n=2) respectively.

Severity of Illness: Among all acute HBV cases in 2006, there were no fatalities reported.

Risk Factors: Risk factors were identified in 58% (N=36) of confirmed cases (including some cases with multiple risk factors). Of those with risk factors, multiple sexual partners (n=19, 53%) was the most common risk factor reported, followed by MSM (n=11, 31%), injection drug use (n=5, 14%), acupuncture (n=2, 6%), and tattoo (n=1, 3%) (Figure 4).

Figure 4
Hepatitis B Reported Risk Factors*
LAC, 2006



*Includes cases identifying multiple risks.

COMMENTS

In LAC, there were 403 cases initially reported to have acute hepatitis B in comparison to the 381 cases reported for 2005. In both years, the percentage of cases that met the CDC/CSTE criteria for confirmation was similar (~15%). Most cases that are not confirmed as meeting the CDC/CSTE criteria are missing documentation of clear evidence of liver involvement (e.g., the liver enzyme levels are normal or missing).

In 2006, all acute hepatitis B cases were aged 15 years or older. Sixty-six percent were in younger adults aged 15-44 years. People with multiple sexual partners and MSM continue to be at risk for hepatitis B; thus, preventive efforts including education and vaccinations should continue to focus on these high-risk populations. In LAC, hepatitis B vaccine is provided to high-risk groups at the Public Health District Health Centers at no charge in an effort to reduce hepatitis B incidence.

Only 58% of the cases had an identified risk factor for acute hepatitis B. LAC DPH will use a new risk factor form in 2007 and it is hoped that better identification of risk factors, to aid in prevention programs, will follow.

PREVENTION

Decreasing rates of acute hepatitis B in children under age 19 is evidence of the successful immunization strategy to eliminate HBV transmission in LAC. The immunization strategy includes: preventing perinatal HBV transmission by screening all pregnant women for HBsAg and providing immunoprophylaxis to infants of HBV-infected women, routine immunization of all infants, and catch-up vaccination of all previously unvaccinated children aged < 19 years.

New strategies are needed to reduce high-risk behaviors and provide resources for low-cost hepatitis B immunization particularly for adults with the highest rates of transmission. Development and implementation of such strategies is possible through collaboration between public health, community-based organizations, and other agencies that serve target populations. Additionally, promoting hepatitis health education aims at eliminating, reducing, or mitigating high-risk behaviors in sexually active adults and increasing awareness and knowledge in the community.

ADDITIONAL RESOURCES

CDC Viral Hepatitis B - www.cdc.gov/ncidod/diseases/hepatitis/slideset/index.htm

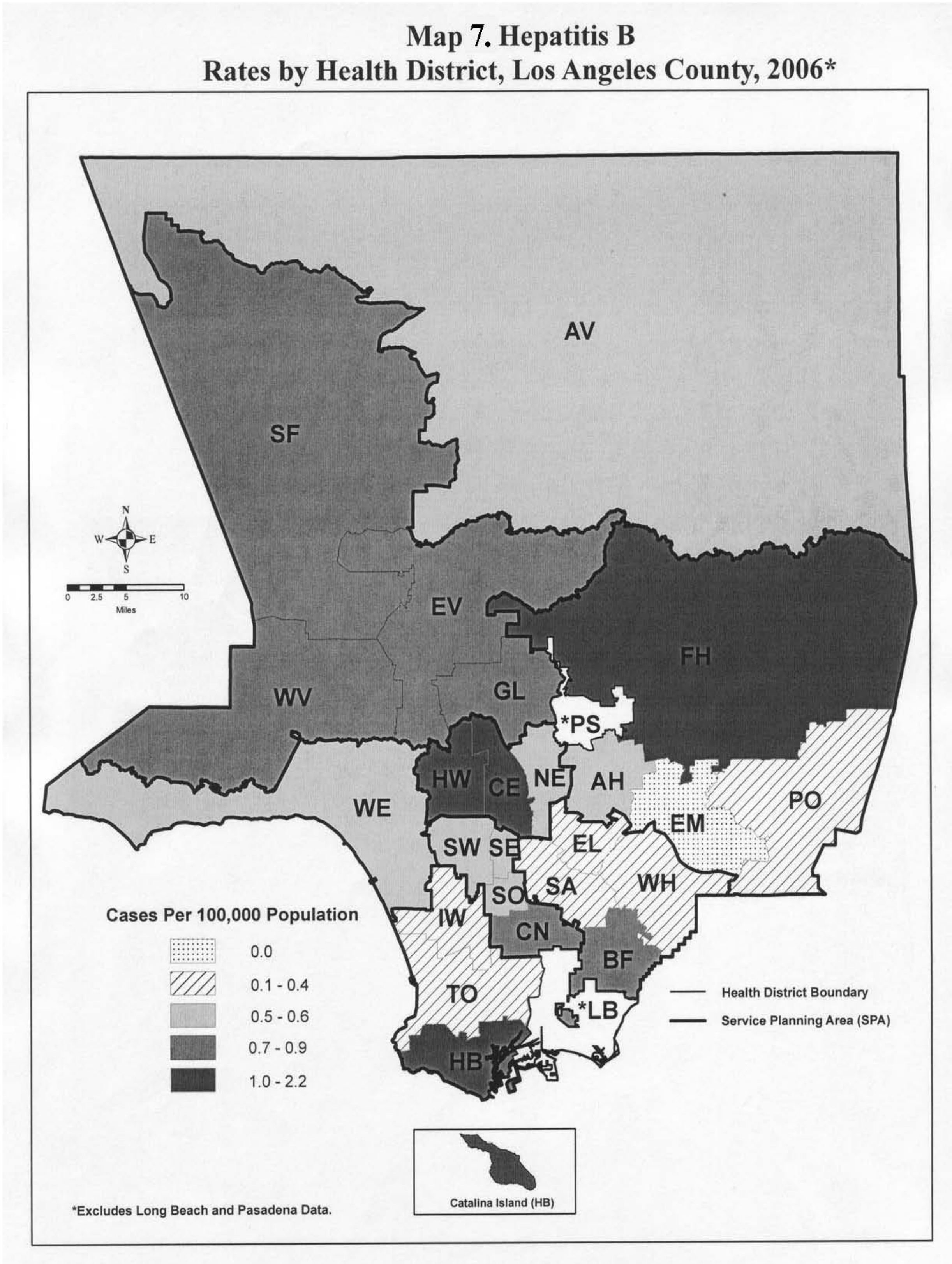
CDC Viral Hepatitis Resource Center - www.cdc.gov/ncidod/diseases/hepatitis/resource/index.htm#pubs

Hepatitis B Vaccine Information - www.cdc.gov/ncidod/diseases/hepatitis/b/factvax.htm

<u>Publications</u>:

Transmission of hepatitis B virus among persons undergoing blood glucose monitoring in long-term care facilities--Mississippi, North Carolina, and Los Angeles County, California, 2003-2004. MMWR 2005; 54(9):220-223. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/mm5409a2.htm

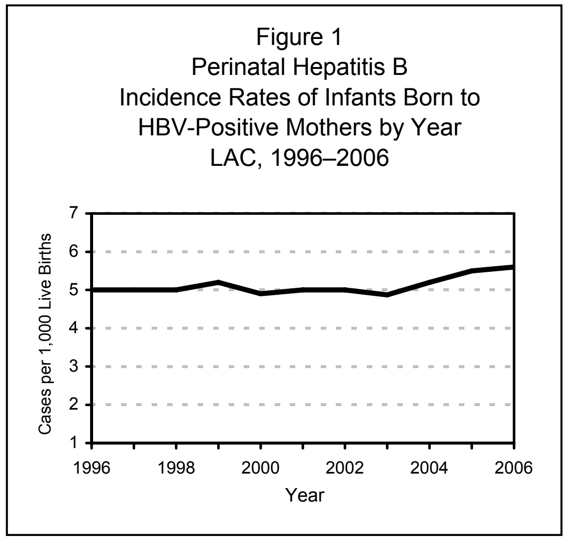
Transmission of hepatitis B and C viruses in outpatient settings--New York, Oklahoma, and Nebraska, 2000-2002. MMWR 2003; 52(38):901-906. Available at: www.cdc.gov/mmwr/PDF/wk/mm5238.pdf


Map 7. Hepatitis B
Rates by Health District, Los Angeles County, 2006*
AV
SF
EV
WV
GL
FH
*PS
HW
CE
NE
AH
WE
EM
PO
SW
SE
EL
SO
SA
WH
IW
CN
BF
TO
*LB
HB
N
W
E
S
0
2.5
5
10
Miles
Cases Per 100,000 Population
0.0
0.1 - 0.4
0.5 - 0.6
0.7 - 0.9
1.0 - 2.2
Health District Boundary
Service Planning Area (SPA)
Catalina Island (HB)
*Excludes Long Beach and Pasadena Data.

HEPATITIS B, PERINATAL

CRUDE DATA	
Number of Infants Born to HBsAg Positive Mothers	795
Incidence of Exposure[a]	
LA County	5.6
United States	N/A
Age at Diagnosis	
Mean	N/A
Median	N/A
Range	N/A

[a] Number of Infants born to HBsAg-positive mothers per 1,000 live births.



DESCRIPTION

Hepatitis B is a vaccine-preventable disease transmitted through parenteral or mucous membrane exposure to blood and other body fluids of individuals infected with the hepatitis B virus (HBV). It is also transmitted from mother to infant during birth. Within LAC, it is estimated that over 40% of infants born to hepatitis B surface antigen (HBsAg) positive women will become infected without prophylaxis. An estimated 90% of infants who become infected by perinatal transmission develop chronic HBV infection and up to 25% will die from chronic liver disease as adults. Postexposure prophylaxis with hepatitis B vaccine and hepatitis B immune globulin (HBIG) administered 12-24 hours after birth, followed by completion of a 3-dose vaccine series, has been demonstrated to be 85–95% effective in preventing acute and chronic HBV infection in infants born to mothers who are positive for both HBsAg and hepatitis B e-antigen. Postvaccination serologic testing is recommended at age 9–18 months after completing immunoprophylaxis to verify vaccine success or failure. The LAC Immunization Program's Perinatal Hepatitis B Prevention Program (PHBPP) conducts enhanced case management of HBsAg-positive pregnant women, their newborns, and household contacts.

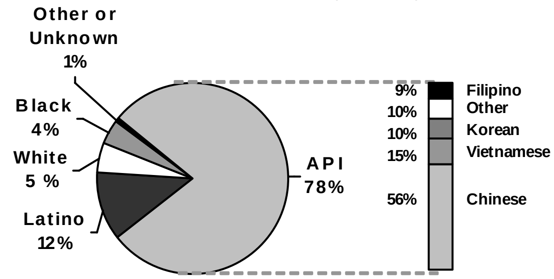
DISEASE ABSTRACT

- The majority of HBsAg-positive women giving birth were born in areas of the world with high or intermediate levels of endemic hepatitis B disease (e.g., Asia, Africa, Eastern Europe, Independent States of the former Soviet Union, Middle East, Pacific Islands, and several Central and South American countries).
- Of infants born to HBsAg-positive mothers, 98% received hepatitis B vaccine and 97% received HBIG within 24 hours of birth.
- Among those infants whose pediatric health care providers responded to a survey after the completion of the full vaccination series, 92% of infants were protected against HBV, 5% were still susceptible, and 3% were infected with HBV.
- The incidence of exposure of infants born to HBsAg-positive mothers increased by 2% from 5.5 to 5.6 per 1,000 infants born in 2006.

STRATIFIED DATA

Trends: In 2006, 795 infants (including 12 sets of twins) were born to 783 HBsAg-positive women. The incidence of exposure of infants born to HBsAg-positive mothers increased by 2% from 5.5 to 5.6 per 1,000 infants born in 2006. (Figure 1).

Figure 2
Perinatal Hepatitis B
by Maternal Race/Ethnicity
LAC, 2006 (N=795)



Race/Ethnicity: The majority of the cases were among Asian/Pacific Islanders (API). Six hundred-nine (78%) of the women were API, 92 (12%) were Latino, 45 (5%) were white, 32 (4%) were black, and 5 (1%) were classified as other or unknown ethnic group (Figure 2). Of API women, over half were Chinese (n=345, 56%). The remaining API women included: Vietnamese (n=90, 15%), Korean (n=60, 10%), Filipino (n=55, 9%), and others from various countries (e.g., Cambodia, Thailand, Samoa, Tonga, Japan, Burma, Indonesia; India, Argentina, and Panama (n=59, 10%).

Age: The age-range of mothers was 15–44 years of age with a median age of 31 years.

Location: The majority of the HBsAg-positive mothers (n=392, 50%) resided in SPA 3, which has a large Asian constituency. An additional 13% resided in SPA 4 (n=100), followed by SPA 2 (n=97, 12%), SPA 7 (n=57, 7%), SPA 8 (n=52, 7%), SPA 6 (n=43, 5%), SPA 5 (n=37, 5%), and SPA 1 (n=5, 1%).

Countries of Origin: The majority (n=714, 91%) of the HBsAg-positive women giving birth were born outside of the US. Of these women, 644 (90%) were known to be born in areas of the world with high or intermediate levels of endemic hepatitis B disease, such as Asia, Africa, Eastern Europe, Independent States of the former Soviet Union, Middle East, Pacific Islands, and several Central and South American countries.

ENHANCED CASE MANAGEMENT

In 2006, enhanced case management was completed for 791 HBsAg-positive mothers, their 798 newborns, and 1,341 households. Case managers made numerous attempts to complete follow-up of mothers, infants and household contacts. The majority (72%, n=569) of the HBsAg-positive mothers were reported in 2005. An additional 15% were reported in 2004 (n=122) followed by 2006 (n=99, 13%) with one case reported in 2003. One hundred thirty mothers were excluded for infant follow-up (86 mothers miscarried, terminated or had fetal demise, 9 transferred/moved out of LAC or were unable to be located before delivery, and 35 were retested and found to be HBsAg negative).

Enhanced case management protocol includes:

1. Educating pregnant HBsAg-positive women about HBV disease and transmission,
2. Identifying and referring household contacts for screening and vaccination,
3. Notifying hospitals of the expected deliveries and requesting that the hospitals return documentation after the infant's birth with the dates and times of the administration of hepatitis B vaccine #1 and HBIG,
4. Notifying the infant's health care provider about the need for hepatitis B vaccine #2 at 1 to 2 months and hepatitis B vaccine #3 at six months of age,
5. Reminding parents about these needed vaccinations, and
6. Sending postvaccination serology letters to pediatric health care providers.

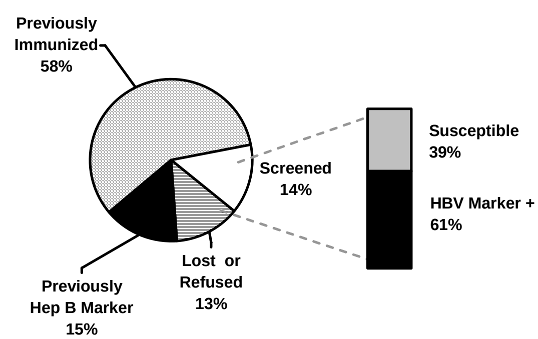
Infant Immunoprophylaxis Completion Rates: Of 798 eligible infants (including 7 sets of twins) born to 791 mothers, nearly all received the hepatitis B vaccine #1 (n=780, 98%) and HBIG (n= 770, 97%) within 24 hours of birth. The majority of infants (n=748, 94%) received HBIG and a complete three-dose series of hepatitis B vaccine (Table 1).

Table 1. Summary of Infant Hepatitis B Immunoprophylaxis, LAC—2006 (N=798)

Hepatitis B Immunoprophylaxis	# of Infants	Percent*
Received hepatitis B vaccine #1 ≤ 12 hours after birth	769	96%
Received hepatitis B vaccine #1 ≤ 24 hours after birth	780	98%
Received HBIG ≤ 12 hours after birth	760	95%
Received HBIG ≤ 24 hours after birth	770	97%
Completed HBIG/3-dose hepatitis B vaccine series	748	94%

* Percent of infants receiving hepatitis B immunoprophylaxis out of a total 798 infants born to 791 HBsAg+ mothers who completed follow-up in 2006.

Figure 3
HBV Marker Status of Household Contacts to Cases Closed
LAC, 2006 (N=1,341)



Household and Sexual Contacts Completion Rates: A household contact was defined as an individual with anticipated continuous household exposure for greater than one year (often limited to nuclear family). Of 1,341 household and sexual contacts identified, 778 (58%) had already been vaccinated against hepatitis B, and 198 (15%) were known to have serologic evidence of hepatitis B infection. Of the remaining 365 (27%) contacts, 192 (14%) were screened for serologic evidence of hepatitis B infection or immunity, while 173 (13%) refused screening or vaccination, were lost to follow-up, or moved. Of the 192 (14%) household contacts that were serologically screened, 118 (61%) had positive markers for hepatitis B and therefore did not need vaccine. The remaining 74 (39%) household contacts were seronegative, and therefore, susceptible to hepatitis B infection (Figure 3). At the time of completion of case management for the HBsAg-positive mothers, 56 (75%) of these susceptible household contacts had completed all three doses of hepatitis B vaccine.

Postvaccination Serology Results: Postvaccination serology testing of infants born to HBsAg-positive mothers is recommended 3 to 18 months after completing immunoprophylaxis to verify efficacy of the hepatitis B immunoprophylaxis. Letters requesting post-vaccination serology results were mailed to pediatric health care providers of infants tracked by the PHBPP. Post-vaccination serology results were received for 180 infants screened in 2006. Of these, 166 (92%) had antibodies to hepatitis B surface antigen indicating protection against HBV, 5 (3%) were HBsAg-positive and infected, and 9 (5%) were negative for both markers and revaccination was recommended.

ADDITIONAL RESOURCES

Information from the CDC:

- General information – www.cdc.gov/vaccines/vpd-vac/hepb/
- Publications – www.cdc.gov/ncidod/diseases/hepatitis/resource/pubs.htm
- Perinatal hepatitis B vaccine recommendations - www.cdc.gov/mmwr/PDF/rr/rr5416.pdf

Additional information:

- Immunization Program's PHBPP website - http://lapublichealth.org/ip/perinatalhepB/
- Hepatitis B Foundation – www.hepb.org
- Asian Liver Center - http://liver.stanford.edu
- Immunization Action Coalition – www.immunize.org

HEPATITIS C, ACUTE

CRUDE DATA	
Number of Cases	4
Annual Incidence LA County California United States	 0.04[a] 0.07[b] 0.26[b]

[a] Rates based on fewer than 19 cases are unreliable.
[b] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

DESCRIPTION

The Hepatitis C virus (HCV) is the most common bloodborne infection in the US. This RNA virus is predominantly transmitted through contact with contaminated blood and blood products via injection drug use. Sexual and perinatal transmission of HCV appears to occur less frequently. People at risk include: anyone who has had a blood transfusion prior to 1989, IV drug users, hemodialysis patients, infants born to infected mothers, those with multiple sexual partners, health care workers who suffer needle-stick accidents, and people with tattoos or body-piercing. However, an estimated 30% have no identifiable history of exposure to the virus. Household or familial contact is not considered a risk factor for the transmission of hepatitis C. There is no vaccine available for HCV and vaccines for hepatitis A and B do not provide immunity against hepatitis C.

Symptoms of acute infections can include jaundice, fatigue, anorexia, nausea, or vomiting; however, up to 85% of acute infections have mild or no symptoms and usually go undetected. After acute infection, 15%-25% of persons appear to resolve their infection without sequelae as defined by sustained absence of HCV RNA in serum and normalization of ALT levels. Chronic HCV infection develops in most persons (75%-85%) with persistent or fluctuating ALT elevations indicating active liver diseases developing in 60%-70% of chronically infected persons. In the remaining 30%-40% of chronically infected persons, ALT levels are normal. No clinical or epidemiologic features among patients with acute infection have been found to be predictive of either persistent infection or chronic liver disease [1]. Most studies have reported that medical complications occur decades after initial infection including cirrhosis, liver failure, and hepatic cancer.

ACDC uses the CDC/CSTE criteria for acute hepatitis C to standardize surveillance of this infection. The criteria include discrete onset of symptoms and:

1. A positive HCV test (antibody test EIA) confirmed by a more specific test (RIBA or detection of the HCV-RNA antigen by polymerase-chain reaction [PCR]) or an EIA signal to cutoff ratio of $\geq$3.8;
2. Serum alanine aminotransferase (ALT) greater than 400; and
3. No evidence of either acute hepatitis A or B disease.

The purpose of standardizing surveillance is to allow ACDC to more accurately monitor trends in hepatitis C, compare local data with state and national data, and improve identification of risk groups, and develop and evaluate prevention programs.

DISEASE ABSTRACT

- There were four cases of confirmed acute hepatitis C in 2006, compare to 3 cases confirmed in 2005.
- No fatal cases of acute hepatitis C were reported in 2006.
- All cases were white.

STRATIFIED DATA

Seasonality: None.

Age: Cases ranged in age from 43 to 85 years (the median age was 51; the mean age was 58).

Sex: In 2006, the male-to-female rate ratio was 3:1, which differed compared to the previous year (1:2 in 2005)

Race/Ethnicity: In 2006, all cases were white. It remained the same as the previous year.

Location: SPA 8 (n=2) had the most cases, followed by SPA 6 (n=1) and homeless (n=1), respectively.

Risk Factors: Of the four confirmed acute cases, risk factors were identified in 50% (n=2) of the cases (including some cases with multiple risk factors). The most commonly identified risk factor for infection were multiple sexual partners and injection drug use (n=2), followed by MSM (n=1) and being in contact with another case (n=1), respectively.

COMMENTS

There were 158 cases initially reported to have acute hepatitis C in 2006 as compared to 79 cases reported in 2005. Upon further investigation, only four, 3% (n=3; 4% in 2005) met the acute hepatitis C surveillance criteria. The stringent criteria for acute hepatitis C illustrates the difficulty of counting initially reported cases as confirmed acute hepatitis C for surveillance purposes. Therefore, it is likely that this data reflects an under-identification of acute hepatitis C in those cases reported to Public Health. Furthermore, since most people have no symptoms or limited, non-specific symptoms in the acute stage of hepatitis C and therefore never diagnosed or reported to Public Health, there are likely many more new cases of acute hepatitis C in Los Angeles County each year.

There were limitations to the data collected. The data did not provide enough information for monitoring trends in transmission patterns. Half of the cases denied having risk factors for infection. The two cases that reside in SPA 8 (Harbor HD) lived in the same census tract. After further investigation, no link could be established between these cases.

Although the number of new cases of acute hepatitis C has declined over the past 5 years, there is still a substantial burden of disease on the population from chronic hepatitis C. It is very important for improvements on monitoring changes in acute disease incidence and risk factors for infection be used to assess comprehensively the burden of disease caused by HCV infection in LA County. LAC DPH will use a new risk factor form starting in 2007 and it is hoped that better identification of risk factors, to aid in prevention programs, will follow.

PREVENTION

Universal blood product screening in 1990 and heat-inactivation of other blood concentrates initiated in 1987 have dramatically reduced recipient-associated cases of hepatitis C. This leaves the reduction of high-risk behaviors as the primary recommendation for preventing transmission; especially, since there is no effective vaccine or post-exposure prophylaxis. Educational efforts aimed at reducing high-risk behaviors (e.g., sharing injection drug equipment, engaging in unprotected sex), may help to reduce new hepatitis C cases. Additional education provided to those who already have hepatitis C is important because alcohol consumption and co-infection with HIV can accelerate the progression of cirrhosis and hepatocellular carcinoma. Furthermore, patients with chronic hepatitis C should be encouraged to receive hepatitis A and B vaccine and evaluated for severity of their liver diseases and for possible treatment.

REFERENCES

1. CDC. Recommendation for prevention and control of hepatitis C virus (HCV) infection and HCV related chronic disease. MMWR 1998; 47(RR19):1-39. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/00055154.htm

ADDITIONAL RESOURCES

Further information about hepatitis is available from:

- American Liver Foundation – www.liverfoundation.org
- Hepatitis Foundation International – www.hepfi.org/living/index.htm
- CDC – www.cdc.gov/ncidod/diseases/hepatitis

Publications:
CDC. Guidelines for laboratory testing and result reporting of antibody to hepatitis C virus. MMWR 2003; 52(RR03):1-16. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/rr5203a1.htm

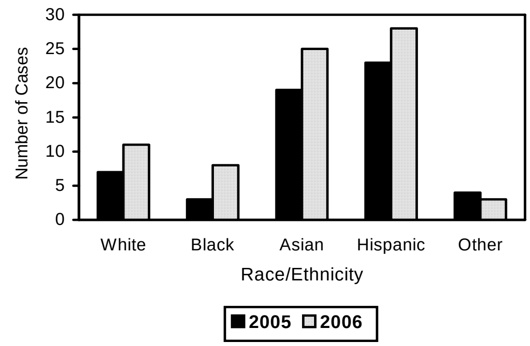
CDC. Surveillance for acute viral hepatitis--United States, 2005. MMWR 2007; 56(SS03):1-24. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/ss5603a1.htm

KAWASAKI SYNDROME

CRUDE DATA	
Number of Cases	75
Annual Incidence[a]	
LA County	0.78
United States	N/A
Age at Diagnosis	
Mean	2.3
Median	2
Range	3 m/o – 8 y/o

[a] Cases per 100,000 population 2006 LAC Census Estimates.

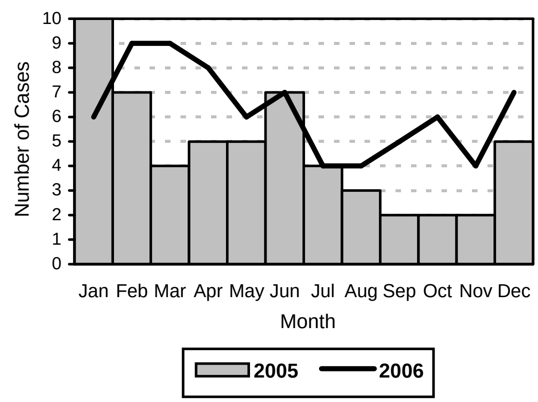
Figure 3
Kawasaki Syndrome
Incidence by Race/Ethnicity
LAC, 2005-2006



DESCRIPTION

Kawasaki Syndrome (KS), also called mucocutaneous lymph node syndrome (MLNS), was first described by Dr. Tomisaku Kawasaki in Japan in 1967 and emerged in the US in the 1970s. Several regional outbreaks have been reported since 1976. This is an illness that affects children usually under 5 years of age. It occurs more often in boys than girls (ratio of about 1.5:1). This is an acute febrile illness that causes an autoimmune inflammation of the blood vessels throughout the body, leading to vessel wall injury with potentially fatal complications affecting the heart and its larger arteries. In the US, it is a major cause of heart disease in children. The etiology is unknown and is considered a noncontagious infection. In the US, the mortality rate is approximately 1%. The diagnosis is clinical, and by CDC case definition, a KS patient must have fever lasting 5 or more days without any other reasonable explanation and must satisfy at least four of the following criteria:

Figure 2
Kawasaki Syndrome
Cases by Month of Onset
LAC, 2005-2006



- bilateral conjunctival injection;
- oral mucosal changes (erythema of lips or oropharynx, strawberry tongue, or drying or fissuring of the lips);
- peripheral extremity changes (edema, erythema, generalized or periungual desquamation)
- rash and;
- cervical lymphadenopathy > 1.5 cm diameter.

Although laboratory findings are nonspecific for KS, they may assist in establishing the diagnosis [3]. Chest X-ray and a series of echocardiograms and electrocardiograms are additional important tests to follow up coronary aneurysm or arteritis. The course of KS can be divided into three clinical phases: acute febrile phase, subacute phase, and convalescent phase [3]. KS is usually treated with a combination of aspirin (typically, 80-100 mg/kg/day in four doses) and IVIG (intravenous gamma globulin 2 gm/kg, a single infusion over 8 to 12 hours). Early treatment can prevent the processes that lead to coronary artery disease.

DISEASE ABSTRACT

- The incidence of KS in LAC increased 34% in 2006 (N=75) compared to 2005 (N=56).
- The recurrent cases were reported in 4% (n=3) of confirmed cases (N=75) in 2006.
- In 2006, coronary artery aneurysm was reported in 5% (n= 4) of cases with IVIG treatment (n=74).

STRATIFIED DATA

Trends: A total of 75 confirmed cases met the CDC surveillance case definition in 2006. There is a continued increase in the number of reported cases from 2001 to 2006 (Figure 1).

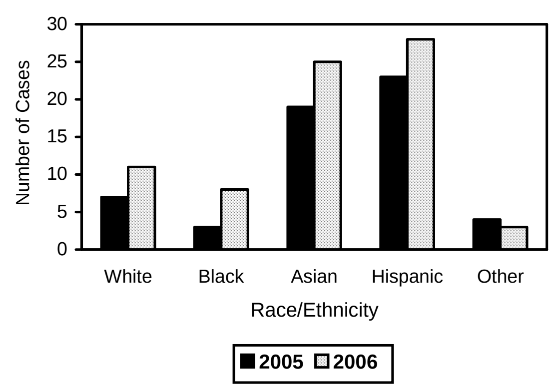
Seasonality: KS occurs year-round, but more cases are reported in winter and spring (Figure 2).

Age: 91% (n=68) of confirmed cases (N=75) were reported in children under 5 years old. Mean age was 2.3 years old, median was 2 years old. The range of age was from 3 months to 8 years old.

Gender: The male-to-female ratio was 1.03:1, unusual to previous reports. 51% (n=38) of confirmed cases were boys, 49% (n=37) of confirmed cases were girls. Descriptive studies show this disease has been approximately 1.5 times more common in boys than in girls.

Race/Ethnicity: The incidence rate for Asians (2.0 per 100,000 population, n=25) was higher compared to other racial groups, as it has been in past years. The incidence rates of other racial groups increased in 2006; black (0.9 per 100,000 population, n=8), Hispanic (0.6 per 100,000 population, n=28), white (0.4 per 100,000 population, n=11), Other (n=3) (Figure 3).

Figure 3
Kawasaki Syndrome
Incidence by Race/Ethnicity
LAC, 2005-2006



Location: The highest rate was found in SPA 8 (1.5 per 100,000 population, n=17), South Bay Area in LAC. The lowest rate was found in SPA 1 (0.3 per 100,000 population, n=1), Antelope Valley Area in LAC. SPA 3 (0.8 per 100,000, n=13), SPA 4 (0.8 per 100,000 population, n=10), SPA 6 (0.8 per 100,000 population, n=8), SPA 2 (0.7 per 100,000 population, n=14), SPA 7 (0.7 per 100,000 population, n=9), SPA 5 (0.5 per 100,000 population, n=5) incidence rates were noted.
Note: Incidence rate for cases less than 20 is unreliable.

Risk Factors: Unknown according to CDC [1] and other research reports.

Prevention: There is no known measure that will prevent KS. However, early treatment with intravenous gamma globulin (IVIG) and aspirin has been found to decrease the incidence of sequelae, the most serious of which is coronary artery aneurysm.

Prognosis: Most patients with KS will recover completely, but about 1-2% will die as a result of blood clots forming in the coronary arteries, or as a result of a heart attack without proper treatment.

COMMENTS

There were three recurrent cases (4%) similar to previously reported rates. All three cases of recurrent cases developed cardiac complications including coronary artery aneurysm. Additional studies on the etiology and pathogenesis of KS are needed to allow for improved diagnosis, treatment, and prevention. In November 2006, a new study refuted an earlier study. The new study finds no link between KS and a newly discovered coronavirus [2]. Atypical or incomplete cases in infants are not counted as confirmed cases because they do not meet the CDC case definition criteria. ACDC uses the documentation of admission, history and physical, discharge summary, and the result of the echocardiogram submitted by infection control professionals to determine possible KS cases.

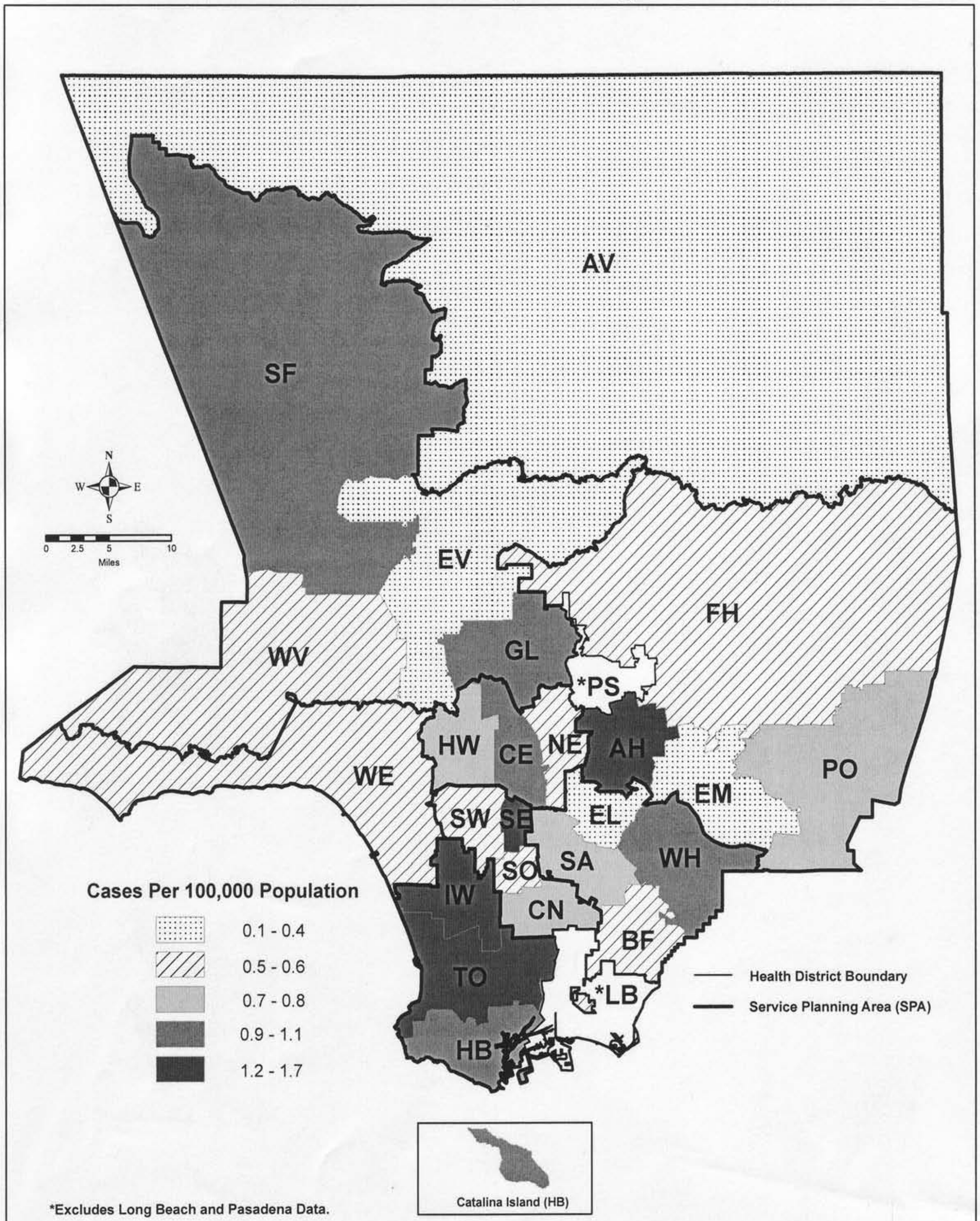
REFERENCES

1. CDC. Kawasaki Syndrome--United States. MMWR 1983; 32(7):98-100.
2. New study finds no link between Kawasaki disease and newly discovered coronavirus. Retrieved from EurekAlert website: www.eurekalert.org/pub_releases/2006-11/idso-nsf112006.php.
3. Taubert KA, Shulman ST. Kawasaki disease. Am Fam Physician 1999; 59(11):3093-3102, 3107-3108.

ADDITIONAL RESOURCE

Burns JC. The riddle of Kawasaki disease. N Engl J Med 2007; 356(7):659-661.

Map 8. Kawasaki Syndrome Rates by Health District, Los Angeles County, 2006*



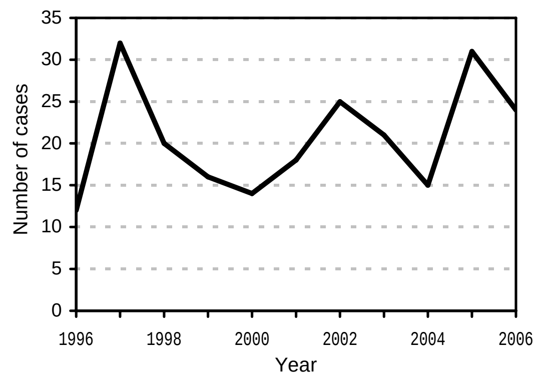
*Excludes Long Beach and Pasadena Data.

LEGIONELLOSIS

CRUDE DATA	
Number of Cases	24
Annual Incidence	
LA County	0.25
California	0.27[a]
United States	0.96[a]
Age at Diagnosis	
Mean	64
Median	67
Range	26–87 years

[a] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

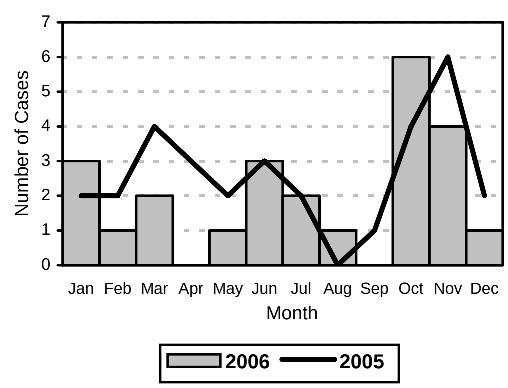
Figure 1
Legionellosis
Cases by Year of Onset
LAC, 1996–2006



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-onset, self-limited flu-like illness without pneumonia. Legionella bacteria are common inhabitants of aquatic systems that thrive in warm environments. Ninety percent of cases of LD are caused by *Legionella pneumophila serogroup 1,* although at least 46 Legionella species and 70 serogroups have been identified. Transmission occurs through inhalation of aerosols containing the bacteria or by aspiration of contaminated water, and water birthing. 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, but the disease most often affects middle-aged and older persons, particularly those who are heavy smokers, have chronic lung disease, or whose immune systems are suppressed by illness or medication.

Figure 2
Legionellosis
Cases by Month of Onset
LAC, 2005–2006



DISEASE ABSTRACT

- The incidence of legionellosis in LAC is decreasing.
- Two unrelated nosocomial cases (1 definite and 1 possible) were reported in 2006.
- No cases of Pontiac fever were reported in 2006.
- The case fatality rate decreased from 16% to 4% in 2005 and 2006, respectively.

STRATIFIED DATA

Trends: A total of 24 reported cases met the CDC surveillance case definition for LD in 2006. This is lower than the peak incidence of 32 cases reported in 1997 in which a community outbreak occurred (Figure 1).

Seasonality: Cases occurred throughout the year, with a peak in October—this peak was unrelated to nosocomial incidents.

Age: Consistent with the expected higher frequency among older persons, the mean age of reported cases was 64 years, the median age was 67 years, and the age range was 26-87 years.

Fatality: In 2006, the fatality rate decreased to 4% (1/24) compared to 16% (5/31) in 2005. The age of the expired case was 73 years.

Gender: There were 13 (54%) male cases and 11 (46%) female cases.

Race: The majority of cases 42% (n=10) occurred in whites. The next most reported racial group was Asians 25% (n=6), Hispanics 21% (n= 5), followed by blacks 12% (n=3).

Ethnicity: The majority of cases reported were among non-Hispanics 79% (n=19), as compared to Hispanics 21% (n= 5).

COMMENTS

In 2006, 22 (92%) LD cases were diagnosed by Legionella urinary antigen, 2 (8%) were diagnosed by direct fluorescent antibody (DFA) staining, and none by BAL/sputum culture, or serologic antibody titers. As in 2005, the Legionella urinary antigen was the most frequently used method to diagnose LD due to the ease of its use and specificity. This test also facilitates diagnosis; therefore, is very useful for prompt initiation of treatment by clinicians. However, this diagnostic test will only consistently screen for Legionella pneumophila serogroup 1. Not using culture to detect infection could result in an incomplete surveillance of legionellosis. LAC encourages all providers who suspect a case of nosocomial legionella to include culture for diagnosis so further testing of the isolate may be performed. Serological testing is not commonly used due to its low sensitivity and needs further research to determine its reliability. This diagnostic method offers minimal impact to patients for their therapeutic management because seroconversion occurs later during the course of infection.

Legionnaire's disease is more prevalent during summer and early fall. The more favorable weather conditions could explain increased exposure risk during outdoor and recreational activities (i.e., hot tubs, cruise ships, hotels, swimming pools, etc). However, data show that LD is equally distributed throughout the year. Outbreaks of LD continue to occur worldwide and surveillance is in full force. There were 4 travel related cases this year 17% (n=4). These cases were found to be unrelated to any outbreak case through collaboration with the Centers for Disease Control and Prevention and the California Department of Health Services..

One definite nosocomial and one possible nosocomial LD case were reported in LAC in 2006 by different medical facilities. Each medical facility conducted eight weeks of prospective active surveillance and six months of retrospective review to detect other possible cases of nosocomial related LD. No additional LD cases were found in either situation.

The number of LD cases in LAC has decreased despite improvements in reporting, monitoring, and ease of diagnostic methods. In 2006, there were 41 reported cases, compared to 43 cases in 2005, a 5% decrease. There are no specific reasons for the decline in the number of cases that met the case definition, but the following are considered to be some of the factors that may have contributed to this decline: 1) clinical awareness continues to be low despite the number of years since LD was first detected; 2) clinicians are still not familiar with the timing of serology collection of single titers to meet the laboratory criteria of case definition; 3) cases may have been missed due to convalescent samples being taken prematurely or not at all; 4) challenge of calling providers to order tests, although the individual is willing to submit; and 5) individual is unwilling to test due to financial reasons and/or don't see the importance of follow-up after treatment. For surveillance to be more effective and to help identify future trends of the disease and possible changing epidemiology, clinicians should consider LD as a differential diagnosis in patients who present with atypical or nosocomial pneumonia. Legionella will be made a mandatory laboratory reportable disease in 2007.

Reasons for the decrease in the case fatality rate are unknown. It is hypothetically possible that an increase in surveillance has resulted in the increased finding of more mild disease though this is not supported by our total number of reported cases.

ADDITIONAL RESOURCES

Guidelines:
CDC. Guidelines for environmental infection control in health-care facilities: recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). MMWR 2003; 52(RR10):1-42. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/rr5210a1.htm

CDC. Guidelines for preventing health-care associated pneumonia, 2003: recommendations of CDC and the Healthcare Infection Practices Advisory Committee (HICPAC). MMWR 2004; 53(RR3):1-36. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/rr5303a1.htm

Squier CL, Stout JE, Krsytofiak S, et al. A proactive approach to prevention of health care-acquired Legionnaires' disease: the Allegheny County (Pittsburg) experience. Am J Infect Control 2005; 33(6):360-367.

State of Maryland, Department of Health and Mental Hygiene. Report of the Maryland scientific working group to study legionella in water systems in healthcare institutions. June 14, 2000. Report available at: www.dhmh.state.md.us/html/legionella.htm

LAC Department of Health Services. Legionellosis: taking the mystery out of laboratory diagnosis. The Public's Health 2001; 1(3):4-5. Available at: www.lapublichealth.org/wwwfiles/ph/ph/ph/TPH_October_2001.pdf

Reviews:
- Stout JE, Yu VL. Hospital-acquired Legionnaires' disease: new developments. Curr Opin Infect Dis 2003; 16(4):337-341.
- Sabria M, Yu VL. Hospital-acquired legionellosis: solutions for a preventable infection. Lancet Infect Dis 2002; 2(6):368-373.

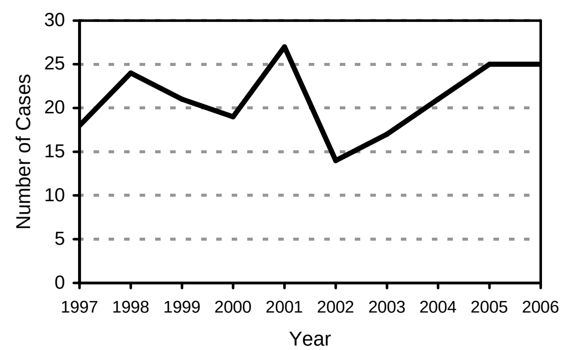
Selected Articles:
- Benin AL, Benson RF, Besser RE. Trends in Legionnaires' disease, 1980- 1998: declining mortality and new patterns of diagnosis. Clin Infect Dis 2002; 35(9):1039-1046.
- Garbino J, Bornand JE, Uckay I, Fonseca S, Sax H. Impact of positive legionella urinary antigen test on patient management and improvement of antibiotic use. J Clin Pathol 2004; 57(12):1302-1305.
- Franzin L, Scolfaro C, Cabodi D, Valera M, Tovo PA. *Legionella pneumophila* pneumonia in a newborn after water birth: a new mode of transmission. Clin Infect Dis 2001; 33(9):e103-104.
- Fields BS, Benson RF, Besser RE. Legionella and Legionnaires' disease: 25 years of investigation. Clin Microbiol Rev 2002; 15(3):506-526.

LISTERIOSIS, NONPERINATAL

CRUDE DATA	
Number of Cases	25
Annual Incidence[a]	
LA County	0.26
United States	N/A
Age at Diagnosis	
Mean	62.96
Median	64
Range	20–90 years

[a] Cases per 100,000 population.

Figure 1
Nonperinatal Listeriosis
Cases by Year of Onset
LAC, 1997–2006



DESCRIPTION

Listeriosis is a disease transmitted primarily through consumption of food contaminated with *Listeria monocytogenes,* a Gram-positive bacterium. *L. monocytogenes* is found in soil and water, and can contaminate raw foods (e.g., uncooked meats and vegetables), as well as processed foods that become contaminated after processing (e.g., soft cheeses and cold cuts). Unpasteurized (raw) milk and foods made from unpasteurized milk may also contain the bacterium. Common symptoms of listeriosis include fever, muscle aches, headache, nausea, diarrhea, and neck stiffness. A case of nonperinatal listeriosis is one that occurs in persons other than pregnant women and/or their fetuses, neonates, or infants up to 42 days after birth. Historically, nonperinatal listeriosis presents as meningoencephalitis and/or septicemia, primarily affecting elderly and immunocompromised persons, such as those with cancer or HIV, and those on immunosuppressive therapy.

DISEASE ABSTRACT

- In 2006, 25 nonperinatal listeriosis cases were reported, the same as the previous year (2005, n=25) (Figure 1).
- There were two case fatalities in 2006. As in 2005, these fatalities were more likely due to severe underlying disease (i.e., cancer, liver disease).
- Although one multi-state cluster was identified by PulseNet, no food source was identified. Additionally, there were no confirmed foodborne listeriosis outbreaks during 2006.

STRATIFIED DATA

Trends: Since 2002 (N=14), the number of nonperinatal listeriosis cases has been increasing (Figure 1). In 2006 there were 25 cases of nonperinatal listeriosis; the same as 2005.

Seasonality: Listeriosis typically follows a seasonal trend with most cases occurring during the summer months. During the previous five years, the highest incidence of cases occurred during July and August. This year's trend was different in that there were two peaks (one in July and another in October) (Figure 2).

Age: Advanced age is considered a risk factor for nonperinatal listeriosis. In 2006, 48% (n=12) of nonperinatal listeriosis cases were 65 years of age or older - an increase from 2005 (36%, n=9). In 2006, the median age of nonperinatal listeriosis cases was 64 years, markedly higher than the median age of 54 years in 2005. The majority of cases in 2006 were over the age of 45 years.

Sex: Similar to previous years, more males (n=13) than females (n=12) contracted nonperinatal listeriosis; though due to the relatively small number of cases, the difference in the infection rate between the two sexes is probably not significant.

Race/Ethnicity: In 2006, whites and Latinos had the highest numbers of incident cases of nonperinatal listeriosis (n=12, 48%, and n=9, 43%, respectively) (Figure 4). Since 2004, the annual numbers of Latino cases has remained the same. In 2006 there was a significant increase in white cases.

Location: Geographic information was known for all 25 of the cases. During 2006, there was no significant clustering of cases by location.

Predisposing Conditions and Medical Risk Factors: In 2006, 72% (n=18) of the nonperinatal cases occurred in adults older than 65 years of age. In addition, 56% had cancer; 36% had history of gastrointestinal disease; 32% had recent chemotherapy; 20% had kidney disease; 20% had recent antibiotic use; and 20% had recent steroid use. Twenty-two (88%) of nonperinatal cases had two or more medical risk factors. One case had no known risk factors for listeriosis (Table 1).

High-risk Foods: For high-risk foods routinely investigated, 25% of cases reported eating

Figure 2
Nonperinatal Listeriosis
Cases by Month of Onset
LAC, 2006

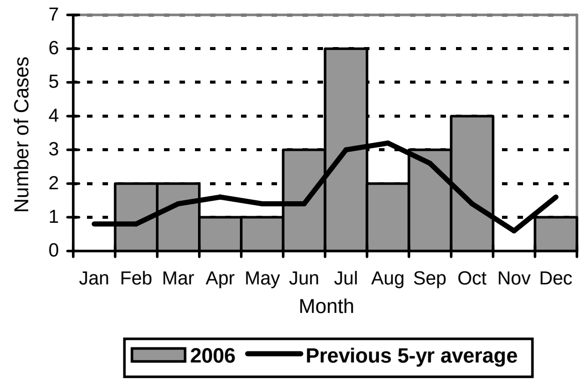


Figure 3
Nonperinatal Listeriosis Incidence
by Age Group
LAC, 2006

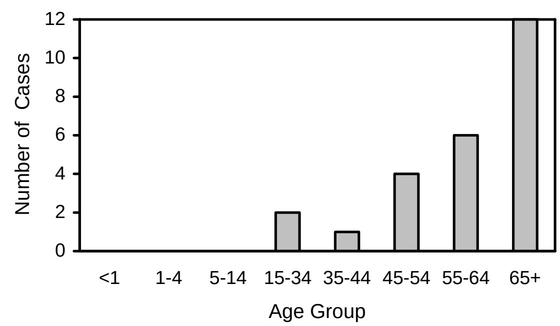
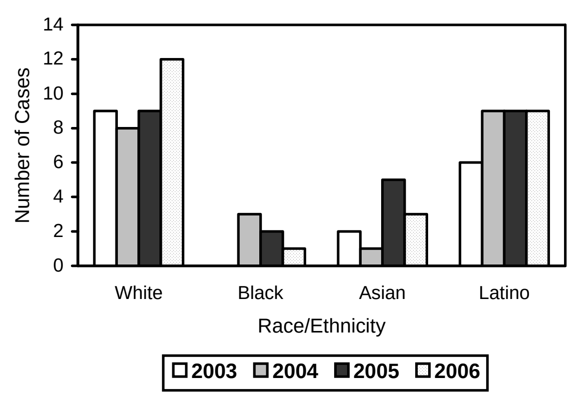


Figure 4
Nonperinatal Listeriosis
Incidence by Race/Ethnicity
LAC, 2003-2006



Mexican or soft cheese; 16% cold cuts or deli meats; 16% other cheese (non-Mexican-style cheese; non-soft cheese); 16% raw fruits; and 20% raw vegetables (Table 2).

Outcome: Two (8%) of the 25 cases in 2006 died. These cases were not of advanced age but were severely immunocompromised with cancer and liver disease.

Culture Sites: *L. monocytogenes* was isolated from blood only in 22 (88%) cases, CSF in two (8%) cases, and one culture drawn from ascitic fluid.

PFGE-identified Clusters: All *L. monocytogenes* isolates are analyzed by pulsed field gel electrophoresis (PFGE). Two cases matched a PulseNet pattern which was part of a cluster with cases from New York, Ohio and Texas.

Table 1. Predisposing Factors in Cases of Nonperinatal Listeriosis—LAC, 2006

Medical Conditions	Number	Percent
Age >65 years	18	72
Cancer	14	56
Gastrointestinal Disease	9	36
Chemotherapy	8	32
Kidney Disease	5	20
Prior Antibiotic Use	5	20
Steroid Use	5	20
Autoimmune Disease	4	16
Liver Disease	4	16
Lung Disease	4	16
Antacid Use	3	12
Chronic Alcoholism	3	12
Diabetes	3	12
Radiation Therapy	3	12
Other Immunosuppressive Therapy	2	8
No Identified Risk Factors	1	4

PREVENTION

In general, listeriosis may be prevented by thoroughly cooking raw food from animal sources, such as beef, pork, or poultry; washing raw fruits and vegetables thoroughly before eating; and keeping uncooked meats separate from raw produce and cooked foods. Avoiding unpasteurized milk or foods made from unpasteurized milk, and washing hands, knives, and cutting boards after handling uncooked foods also may prevent listeriosis.

Table 2. High-risk Foods among Cases of Nonperinatal Listeriosis—LAC, 2006

Risk foods	Number	Percent
Raw Vegetables	5	20
Raw Fruit	4	16
Cold Cuts/Deli-Meats	4	16
Soft Cheese	4	16
Other Cheese	4	16
Mexican Style Cheese	3	12

Persons at high risk for listeriosis include the elderly, those with cancer, HIV, diabetes, weakened immune systems, and those on immunosuppressive therapy. These individuals should follow additional recommendations: avoid soft cheeses such as feta, brie, camembert, blue-veined, and Mexican-style cheese. Hard cheeses, processed cheeses, cream cheese, cottage cheese, or yogurt need not be avoided all together; however, individuals with severely compromised immune systems and/or several disease risk factors should avoid them. Leftover foods or ready-to-eat foods, such as hot dogs and deli meats, should be cooked until steaming hot before eating. Finally, although the risk of listeriosis associated with foods from deli counters is relatively low, immunosuppressed persons should avoid these foods or thoroughly reheat cold cuts before eating.

ADDITIONAL RESOURCES

General disease information is available from the CDC at:
www.cdc.gov/ncidod/dbmd/diseaseinfo/listeriosis_g.htm

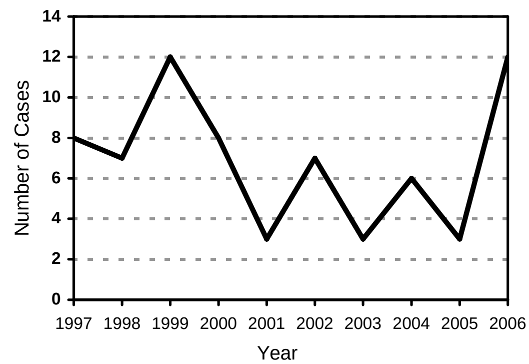
General information and reporting information about this and other foodborne diseases in LAC is available at: www.lapublichealth.org/acd/food.htm

LISTERIOSIS, PERINATAL

CRUDE DATA	
Number of Cases[a]	12
Annual Incidence[b] LA County United States	 8.47[c] N/A
Age at Onset Maternal: Mean Median Range	 28.36 years 30 years 16-38 years
Infant Gestational: Mean Median Range	 31 weeks 31 weeks 22-37 weeks

[a] Cases are mother-infant pairs.
[b] Rates for perinatal listeriosis were calculated as cases per 100,000 live births.
[c] Rates based on less than 19 observations are unreliable.

Figure 1
Perinatal Listeriosis
Cases by Year of Onset
LAC, 1997–2006



DESCRIPTION

Perinatal listeriosis is a disease transmitted transplacentally from infected pregnant women; these women may experience only mild flu-like symptoms or may be asymptomatic. A perinatal listeriosis case is defined as a mother-infant pair in which one or both persons has a positive *Listeria monocytogenes* culture from a normally sterile site. Neonatal/infant listeriosis is often categorized into early onset (0–6 days after birth) and late onset (7–42 days after birth). Infection during pregnancy may lead to premature birth, stillbirth, or septicemia and/or meningitis in the neonate—even if the mother is asymptomatic. There is no vaccine to prevent listeriosis.

DISEASE ABSTRACT

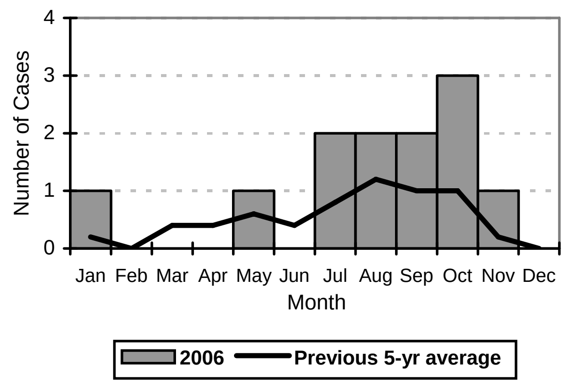
- Perinatal listeriosis increased markedly from three cases in 2005 to 12 cases in 2006 (Figure 1). The 12 cases included ten single births and one set of twins.
- Eight cases were born ill at varying lengths of gestation. Two cases resulted in fetal demise at 22 and 31 weeks gestation. The outcomes of the remaining two cases were unknown due to inability to contact the family for follow-up.

STRATIFIED DATA

Trends: Since 2001, the number of perinatal listeriosis has fluctuated, ranging from 3 to 12 cases, with a marked increase from three cases in 2005 to 12 cases in 2006 (Figure 1).

Seasonality: In 2006, the seasonality of perinatal listeriosis was slightly, though insignificantly, later than the average annual incidence of the previous five years. Perinatal listeriosis cases peaked in October during 2006 (Figure 2).

Figure 2
Perinatal Listeriosis
Cases by Month of Onset
LAC, 2006



Age: During 2006, the average maternal and gestational ages of perinatal cases at disease onset (28 years and 31 weeks, respectively) were higher compared to those in 2005 although the overall five year trend remains unchanged.

Sex: In 2006, seven infants were identified as male and five as female. The male to female ratio was 1.4:1. In 2005 the male to female ratio was unknown. During 2004 and 2003, the male to female ratios were 2:3 and 2:1, respectively.

Race/Ethnicity: In 2006, 58.3% (n=7) of the cases were Latino, which is similar to years past. There was an increase in black cases from 0 cases in 2005 to 3 in 2006 (25%). The remaining cases were white (n=1, 8.3%) and Asian (n=1, 8.3%). However, due to small numbers of cases, it is difficult to draw conclusions from this information.

Location: In 2006, three cases resided in SPA 4 (Central and Northeast health districts), SPAs 3, 6 and 7 had two cases each. Additionally, one case resided in each of SPAs 1, 2 and 8. In 2005, reported perinatal cases were from only SPA 4 and 6.

Type of Delivery: Five infants (42%) were delivered by caesarian section. Two infants (17%) were delivered vaginally. The mode of delivery for the remaining infants is unknown.

Outcome: There were no maternal fatalities. Two (33%) neonates were stillborn – one at 22 and one at 31 weeks of gestation. Eight infants (67%) were delivered sick at varying weeks of gestation ranging from 25-37 weeks of gestation. The outcomes of the other two infants are unknown.

Culture Sites: Listeriosis was culture confirmed in six maternal and ten neonatal isolates. Among culture-positive mothers, five (83%) mothers had *L. monocytogenes* isolated from blood, one mother had *L. monocytogenes* isolated from peritoneal fluid. Of the ten neonatal isolates, six (60%) had *L. monocytogenes* isolated from blood; the other isolates were from wound, amniotic fluid, gastric aspirate and sputum.

Maternal Clinical Signs/Outcomes: In 2006, ten mothers had fever (91%). Temperatures were recorded for five mothers with an average temperature of 102°F. Signs and symptoms were unknown for one case. Similar to the previous three years no mothers had meningitis.

Onset: In 2006, 12 neonates/infants (100%) were categorized as early onset cases in which the disease onset is 0 to 6 days after birth.

High-risk Foods: Six cases (50%) reported eating at least one potentially high-risk food. All six ate Mexican-style cheese; the other risk foods included: soft cheeses (n=2), raw fruits (n=3) and raw vegetables (n=3) (Table 1).

Risk factors: Four mothers (36%) had known predisposing medical risk factors other than pregnancy. Those factors included use of iron supplements, chronic anemia, and gestational diabetes.

PREVENTION

L. monocytogenes is found in soil and water. Animals can carry *Listeria* without appearing ill, which can result in contaminated foods of animal origin, such as meats and dairy products. In particular, studies have implicated unpasteurized milk or milk products; soft cheeses (Mexican-style, Brie, Feta, blue-veined, Camembert); undercooked meat, such as beef, pork, poultry, and pâté; and cold cuts from deli counters. Pregnant women should avoid these foods. In particular, cheese sold by street vendors, or obtained from relatives/friends in other countries where food processing quality assurance is unknown should be avoided by pregnant women.

Table 1. High-risk Foods among Cases of Perinatal Listeriosis—LAC, 2006

Risk foods	Number	Percent
Mexican-style Cheese	6	50
Raw Fruit	3	25
Raw Vegetables	3	25
Soft Cheese	2	17
Other Cheese	0	0
Cold Cuts/ Deli Meats	0	0
Yeast Products	0	0
Raw Milk	0	0

In addition, fruits and vegetables should be thoroughly washed. Uncooked meats should be stored separately from vegetables, cooked foods, and ready-to-eat foods. Hands, utensils, and cutting boards should be washed after handling uncooked foods. Leftover foods or ready-to-eat foods, such as hot dogs, should be cooked until steaming hot before eating. Finally, although the risk of listeriosis associated with foods from deli counters is relatively low, pregnant women may choose to avoid these foods or thoroughly reheat cold cuts before eating.

Given the seasonality of perinatal listeriosis, prevention strategies should take effect before April. Possible preventive methods include education during prenatal checkups, outreach to Hispanic/Latino communities, and food safety notices at food and deli markets.

COMMENTS

Incidence of perinatal listeriosis in LAC increased to 12 cases in 2006. Prevention efforts should be targeted towards Hispanic and black women, especially since Hispanics are the fastest growing segment of the LAC population. There were no perinatal cases associated with outbreaks in 2006.

All isolates of *L. monocytogenes* are typed by pulsed-field gel electrophoresis (PFGE), a technique to detect matching strains of various pathogenic agents. When matches between isolates from patients or foods are detected, an investigation may be initiated. In addition, a solitary case occurring locally can be linked by PFGE results to an outbreak occurring on a wider geographical scale. In 2006, there were no cases of *L. monocytogenes* in LAC associated with a multi-jurisdictional outbreak identified in this manner.

ADDITIONAL RESOURCES

General disease information is available from the CDC at:
www.cdc.gov/ncidod/dbmd/diseaseinfo/listeriosis_g.htm

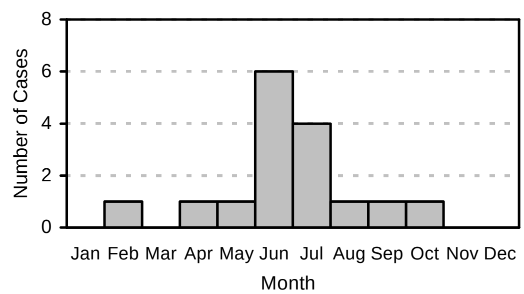
General information and reporting information about this and other foodborne diseases in LAC is available at: www.lapublichealth.org/acd/food.htm

LYME DISEASE

CRUDE DATA	
Number of Cases	16
Annual Incidence[a]	
LA County	0.17[b]
California	0.24[c]
United States	6.72[c]
Age at Diagnosis	
Mean	33
Median	28.5
Range	8–69 years

[a] Cases per 100,000 population. Exposure may have occurred outside of indicated jurisdiction.
[b] Incidence rates based on counts less than 19 are unreliable.
[c] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

Figure 2
Lyme Disease Cases
by Month of Onset*
LAC, 2006



DESCRIPTION

Lyme disease (LD) is caused by a bacterium, *Borrelia burgdorferi*, which is transmitted to humans by the bite of the western blacklegged tick (*Ixodes pacificus*). This disease is not common in Los Angeles County (LAC). From 1996 through 2005, the LAC incidence of LD was estimated at 0.05 per 100,000 persons—equivalent to one case for every 2 million residents per year [1]. Most of these cases were acquired outside of LAC from known endemic regions in the United States (US); each year only 0 to 5 cases report possible tick exposure within LAC. In contrast, the incidence in Connecticut, one of the most endemic states in the US, was 51.56 per 100,000 in 2005 [2,3]. Nevertheless, LD has been well documented to occur in counties throughout the state of California (CA) — Trinity County in northern California reported an incidence of 19.23 per 100,000 in 2005 [1] — and has been a reportable disease in the state since 1989.

The reservoir is small rodents, with deer as a secondary reservoir. Ticks that feed from infected rodents or deer may then transmit the disease to humans, who are accidental hosts. The most common clinical presentation is a distinctive circular rash called erythema migrans (EM) that usually appears at the site of the bite within 3-32 days of a tick bite exposure. EM resembles a rapidly expanding red bull's eye and occurs in 60-90% of cases. If there is no rash, other early symptoms such as fever, body aches, headaches, and fatigue are often unrecognized as indicators of LD. If untreated, patients may present with late stage symptoms such as aseptic meningitis, cranial neuritis, cardiac arrhythmias and arthritis of the large joints. Early disease is treated with a short course of oral antibiotics, while late symptom manifestations may require longer treatment with oral or intravenous (IV) antibiotics. Currently, there is no vaccine.

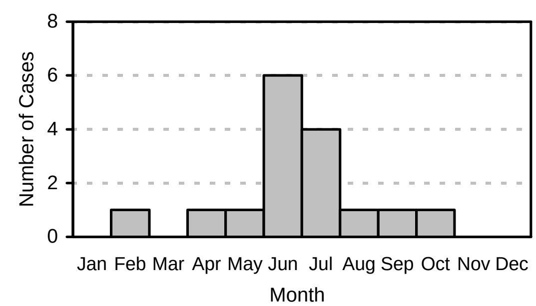
Because the EM rash is unique to LD and can distinguish it from other diseases with similar early symptoms, its presentation precludes the need for further testing. For purposes of surveillance, the Centers for Disease Control and Prevention (CDC) requires a confirmed case of LD to have documented EM that is at least 5cm in diameter or at least one late manifestation of LD diagnosed by a healthcare provider with supporting laboratory results. Laboratory criteria for case confirmation include the isolation of *B. burgdorferi* from a clinical specimen or demonstration of diagnostic IgM or IgG to *B. burgdorferi* in serum or cerebral spinal fluid. Currently available serological tests, however, are often not sensitive, specific or consistent; and LD should primarily be diagnosed by a healthcare provider's consideration of the clinical presentation and history of tick exposure. If indicated, the CDC, Food and Drug Administration, the Association of State and Territorial Public Health Laboratory Directors, and the American College of Physicians currently recommend a two-step serologic testing procedure for LD: an initial enzyme immunoassay (EIA) or immunofluorescent antibody (IFA) screening test, and if positive or equivocal, followed by IgM and IgG Western immunoblotting [4].

DISEASE ABSTRACT

- In 2006, there was a 129% increase in reported cases that met CDC surveillance criteria; most likely due to increases of LD seen in the eastern US.
- The majority of cases (81%) in 2006 reported exposure outside the county. The prevalence of probable LAC-acquired infection remains low and consistent with surveillance data from the previous 13 years.

Trends: The number of cases has increased by nearly 129% from 7 confirmed cases in 2005 to 16 in 2006 (Figure 1). This number is twice as high as any year in which LAC has recorded incidence of LD. However, the number of cases reported with a possible exposure within LAC (n=3) remains similar to previous years. Since 1994, cases with possible exposure within LAC has ranged from 0 to 5.

Figure 2
Lyme Disease Cases
by Month of Onset*
LAC, 2006



*Previous 5-yr average not available.

Seasonality: There was a peak number of cases occurring in the summer months of June (n=6) and July (n=4) (Figure 2). A similar peak occurred in 2005 in July (n=2) and August (n=2). Ticks may be active at any time of the year but the highest risk of infection occurs from March through August. The seasonal peak may be a reflection of both tick activity and human outdoor activity.

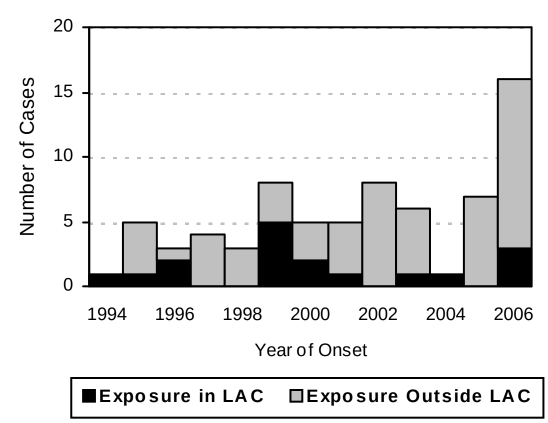
Age: The average age of cases in 2006 was 33, the median was 28.5, and the ages ranged from 8–69 years old. Nationally, LD is most common among persons aged 5–19 years and 30 years and older.

Sex: The male to female ratio was 0.78:1. Nationally, LD occurs more commonly among males.

Race/Ethnicity: Of those cases in which race/ethnicity were known, most were white (n=11, 78%). There were two Latinos (14%) and one Asian (7%).

Location: LD does not commonly occur in ticks in LAC, most cases were likely exposed to infected ticks while outside of the county. However, three cases (19%) reported no history of travel outside of LAC within three months of their onset of EM rash (Figure 3). These cases occurred among residents from SPAs 2, 5, and 8.

Figure 3
Lyme Disease Cases
by Location of Exposure
LAC, 1994–2006



Disease Severity: Most cases (n=13, 81%) demonstrated EM. Rash sizes ranged from 5–20cm, with a mean of 10.25cm and median of 10cm. Five cases (31%) experienced swelling of one or a few joints, a symptom characteristic of late LD, two of them in combination with EM. One case experienced an additional late symptom: a facial nerve palsy consistent with a cranial neuropathy.

Risk Factors: Many of the cases (n=10, 63%) recalled a tick bite within three months of their onset. Thirteen cases (81%) reported travel outside of LAC prior to their onset of symptoms (Figure 3). Of the thirteen, nine (69%) recalled incurring the tick bite during their travels. The remaining either denied or could not recall a tick bite. However, published studies show that few patients - only about one third – can recall being bitten by a tick [5]. All traveled to areas where LD is known to be highly endemic: 11 to the eastern US and 2 to Europe – Sweden, in particular. Of the three that remained within LAC, one had traveled to northern California, where LD is more common, over three months before the onset of her EM rash. She could not recall a tick bite. Only one case with no history of travel recalled a tick bite near her residence - a rural area of the San Fernando health district (SPA 2).

PREVENTION

Since GlaxoSmithKline Pharmaceuticals removed the LYMErix® vaccine off the market in February 2002, avoiding tick bite exposure is the primary means of preventing Lyme disease. The risk of acquiring infection with LD increases when the tick has attached to the body for at least 24 hours. Tips for preventing exposure from tick bites include checking the body regularly for prompt removal of attached ticks; wearing light-colored clothing so that ticks can be easily seen; wearing long pants and long-sleeved shirts and tucking pants into boots or socks, and tucking shirts into pants; using tick repellant and treating clothing with products containing permethrin; staying in the middle of trails when hiking to avoid contact with bushes and grasses where ticks are most common; and checking for and controling ticks on pets.

COMMENTS

Each year only 20 to 30 suspected LD cases from LAC residents are reported to LAC DPH by clinicians and laboratories. Many of these reports do not meet the CDC definition for a confirmed case because laboratory tests are often ordered for patients with vague symptoms not consistent with LD. Indeed, the number of cases eventually confirmed in LAC has ranged from none to eight cases a year. However, in 2006 twice the number of confirmed cases typically seen in a single year in LAC was reported. It is likely that this increase reflects increases in LD in the ten states where it is most prevalent (located in the northeastern, mid-Atlantic, and north-central areas of the US), occurring since it became a nationally notifiable disease in 1991 [3]. During the period of 2003–2005, these ten states accounted for 93% of cases nationwide and had an average annual incidence rate per 100,000 persons of 29.1 in 2003, 26.8 in 2004, and 31.6 in 2005. A considerable proportion of cases from LAC, 69% during 2006, reported travel to these highly endemic areas. The number of cases confirmed with possible exposure within LAC remains similar to previous years.

Furthermore, changes in reporting processes may have increased the number of suspected cases reported to LAC DPH. In 2005, Lyme disease became a laboratory reportable disease in California. As soon as March of that year, a commercial laboratory began reporting positive LD results to LAC through an automated electronic reporting system. A second commercial laboratory was added to the automated reporting system in February 2006. The magnitude at which laboratory and electronic reporting may have affected reporting and confirmation of LD in LAC is unknown and will require further study.

The increase in confirmed cases highlights the complicated issues in the diagnosis and surveillance of LD that can result in both overdiagnosis and underreporting. One challenge to surveillance is the misdiagnosis of EM, which occurs even in the highly endemic eastern states [6]. One might expect that the misdiagnosis of EM could be even greater in non-endemic or low endemic areas of the country such as LAC where clinicians have not had as much clinical experience with LD. Not only do the early and late symptoms of LD resemble those of many other diseases, but also the laboratory tests available are often inaccurate in diagnosing LD. Laboratory diagnostic tests may not reliably detect the infection early in the course of disease or can be interpreted incorrectly. Despite this, the surveillance of LD in LAC is heavily based on positive laboratory reports; and reports are confirmed only after consultation with the healthcare provider as well as the patient regarding symptoms and tick exposure. The response rate of healthcare providers in requests for confirmation has not been fully investigated; it most likely varies from year to year and could affect the trends in confirmed LD cases.

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1. California Department of Health Services. 2005 Annual Report. Report available at: www.dhs.ca.gov/ps/dcdc/disb/disbindex.htm
2. CDC. Lyme disease statistics. Report available at: www.cdc.gov/ncidod/dvbid/lyme/ld_statistics.htm
3. CDC. Lyme disease--United States, 2003–2005. MMWR 2007; 56(23):573–576.
4. Fritz CL, Vugia DJ. Clinical issues in Lyme borreliosis: a California perspective. Infect Dis Rev 2001; 3(3):111-122.
5. Gerber MA, Shapiro ED, Burke GS, Parcells VJ, Bell GL. Lyme disease in children in southeastern Connecticut. N Engl J Med 1996; 335(17):1270-1274.
6. Feder HM, Whitaker DL. Misdiagnosis of erythema migrans. Am J Med 1995; 99(4):412-419.

ADDITIONAL RESOURCES

More information about Lyme disease is available from the CDC at:
www.cdc.gov/ncidod/dvbid/lyme/index.htm

A brochure on Lyme disease from the California Department of Public Health is available at:
www.cdph.ca.gov/healthinfo/discond/Documents/Lyme/LymeDiseaseBrochure2005.pdf

PUBLICATIONS

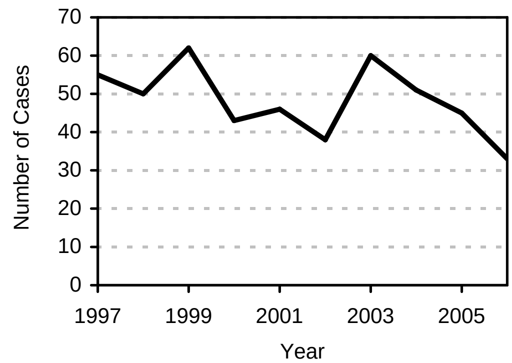
1. Nadelman RB, Wormser GP. Lyme borreliosis. Lancet 1998; 352(9127):557–565.
2. Barbour AG. Lyme Disease: The Cause, the Cure, the Controversy. Baltimore, MD: The Johns Hopkins University Press; 1996.
3. Steere AC. Lyme disease. N Engl J Med 2001; 345(2):115–125.
4. Sood SK. Lyme disease. Pediatr Infect Dis J 1999; 18(10):913–925.
5. Shapiro ED, Gerber MA. Lyme disease. Clin Infect Dis 2000; 31(2):533-542.

MALARIA

CRUDE	
Number of Cases	33
Age at Onset Mean Median Age Range	 38 40 3–69 years
Annual Incidence LA County California United States	 0.34 0.43[a] 0.50[a]

[a] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

Figure 1
Malaria
Cases by Year of Onset
Los Angeles County, 1997–2006



DESCRIPTION

Human malaria is an acute or subacute febrile illness caused by one or more protozoan parasites that infect humans: *Plasmodium vivax*, *P. falciparum, P. malariae,* and *P. ovale*. The disease is transmitted by the bite of an infected *Anopheles sp.* mosquito and is characterized by episodes of chills and fever every 2–3 days. *P. falciparum* is found primarily in tropical regions and poses the greatest risk of death because it invades red blood cells of all stages and is often drug-resistant. The more severe symptoms of *P. falciparum* include jaundice, shock, renal failure, and coma. Each case of malaria requires the demonstration of parasites in thick or thin blood smears, regardless of whether the person experienced previous episodes of malaria while outside the country.

Malaria is usually acquired outside the continental United States (US) through travel and immigration and is rarely transmitted within the US. Although there is no recent documentation of malaria being transmitted locally, a particular mosquito, *A. hermsi*, exists here and is capable of transmitting the parasite. In 1988–89, the last autochthonous cases in California (CA) occurred in San Diego among thirty migrant workers infected with *P. vivax*. Since then, local transmission has not occurred in southern CA due to the inadequate number of people infected with the malaria parasite necessary to sustain disease transmission. Additionally, the mosquito capable of transmitting malaria is very rare.

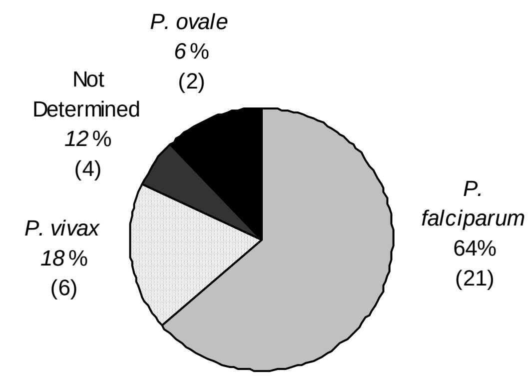
DISEASE ABSTRACT

- The number of malaria cases in LAC has continued to decrease since its peak in 2003.
- The percentage of US travelers who took some form of antimalarial chemoprophylaxis during travel to a malaria-endemic region has increased since the previous year to 52%. Almost all who took prophylaxis reported complete compliance with the regimen.

STRATIFIED DATA

Trends: In 2006, there were 33 reported cases compared to 45 reported the previous year: a 27% decrease. This continued a decline in cases that began in 2003 when 60 cases were reported (Figure 1). Most cases (n=21, 64%) were infected with *P. falciparum* in 2006 (Figure 2), similar to the proportion affected in 2005 (n=29, 65%).

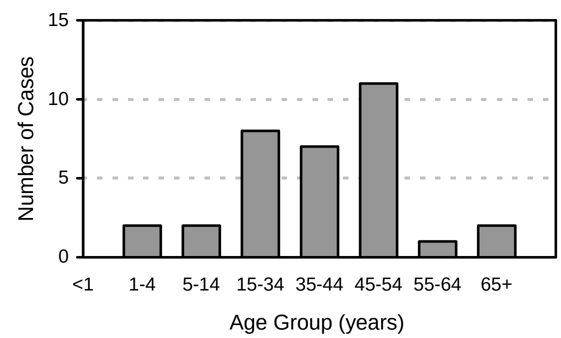
Figure 2
Malaria Cases by Species
Los Angeles County, 2006 (n=33)



Seasonality: Seasonality for malaria was not determined. Malaria is acquired abroad and is independent of LAC weather or seasonal patterns.

Age: The mean age of infection has increased in 2006 to 38 (range: 3–69 years); the median age was 40. The largest number of cases (n=11, 33%) occurred in an older age group than previous years (45–54 years). In 2005 the largest number occurred in the 15–24 year age group (Figure 3).

Figure 3
Malaria
Cases by Age Group
LAC, 2006 (n=33)



Sex: The ratio of male-to-female cases was three to one (3:1).

Race/Ethnicity: The majority of reported malaria cases occurred among blacks, which included African-Americans and African immigrants (n=22, 67%). Five cases each (15%) were reported among Asians and whites. Only one case (3%) of Latino ethnicity was reported. Since the early 1990s, blacks have had the highest proportion of reported malaria cases, with the exception of year 2003, where whites outnumbered blacks. Race and ethnicity were known for all cases.

Disease Severity: There were no deaths or severe complications associated with malarial infection in 2006, however, most (n=24, 73%) required hospitalization. The mean length of hospitalization was 2.7 days and ranged from 1 to 7 days.

Transmission and Risk Factors: All cases reported recent travel to a foreign country, with Africa continuing to be the most common region visited. Twenty-three (70%) reported malaria cases were from individuals who were traveling to or coming from African countries. Reports of travel to Nigeria, the most frequently reported country, increased from 9 in 2005 to 16 in 2006 (n=16) (Table 1). The most commonly reported reason for travel was visiting friends and relatives (n=19, 71%). Refugees and immigrants made up only 7% (n=2) of cases with known travel reasons. Purpose of travel was reported for 82% of cases.

Among the 21 cases that reported US residency prior to their most recent travel, 11 individuals (52%) took prophylaxis, which was at least twice as high a rate of usage compared to the previous two years. Information on antimalarial prophylaxis usage was available for 20 (95%). Almost all (n=10) took their medication correctly as prescribed. When stratified by purpose of travel, the proportion of prophylaxis usage among cases was higher in those who traveled for work than for pleasure (67% vs. 53%) (Table 2). Traveling for work in 2006 included individuals who traveled as part of volunteer service or for a scientific conference. Tourism and visiting friends and family were classified as traveling for pleasure. Prophylaxis usage among travelers for work (67%) has remained similar to that found in 2005 (60%). Usage among travelers for pleasure has increased markedly from 12% in 2005 to 53% in 2006.

Table 1. Malaria Cases by Country of Acquisition and *Plasmodium* Species— LAC, 2006

Country of Acquisition	*P. falciparum*	*P. vivax*	*P. ovale*	Not Determined	Total
Africa	**19**	**1**	**2**	**1**	**23**
- Cameroon	2	0	0	0	2
- Ghana	1	0	0	0	1
- Nigeria	12	1	2	1	16
- Sierra Leone	2	0	0	0	2
- Uganda	2	0	0	0	2
Asia/Oceania	**1**	**3**	**0**	**2**	**6**
- India	0	1	0	1	2
- Indonesia	1	0	0	0	1
- Papua New Guinea*	0	2	0	0	2
- Vanuatu	0	0	0	1	1
Latin America	**0**	**2**	**0**	**1**	**3**
- Guatemala	0	1	0	0	1
- Honduras	0	1	0	0	1
- Mexico	0	0	0	1	1
Unknown	**1**	**0**	**0**	**0**	**1**
Overall Total	**21**	**6**	**2**	**4**	**33**

*One case also traveled to Indonesia and Guatemala in addition to Papua New Guinea.

Table 2. Prophylaxis Use Among US Travelers with Malaria, 2006

Reason for Travel	Total Cases (N)	Prophylaxis Use (N)	(%)
Pleasure	17	9	53
Work	3	2	67
Other/Unknown	1	0	0
Total	**21**	**11**	**52**

Seven of 27 cases (26%) reported a history of infection with malaria in the twelve months prior to their most recent episode. The species of the prior infections were not identified for any cases. No cases were acquired through blood transfusion or transplantation.

PREVENTION

Prevention method of malaria includes avoiding mosquito bites or, once already infected, preventing the development of disease by using antimalarial drugs as prophylaxis. Travelers to countries where malaria is endemic should take precautions by taking the appropriate antimalarial prophylaxis as prescribed; using mosquito repellants, utilizing bednets, and wearing protective clothing; as well as avoiding outdoor activities between dusk and dawn when mosquito activity is at its peak.

COMMENTS

The reason for the overall decrease in malaria cases is most likely due to a decrease in overseas travel and incoming refugees from malaria endemic countries. The number of malaria cases overall is far below the number of cases seen throughout the late 1970s through 1986 (an average of 133 malaria cases reported annually from 1979-1986). Prior to the 1990s, refugees and immigrants from Central America and Southeast Asia made up the majority of all malaria cases seen in LAC. In contrast in 2006, refugees and immigrants made up only 7%.

Information on travel and prophylaxis is obtained by interviewing patients. The data is limited by the patients' ability to recall this information. It is also limited by the small size of the case population, particularly when stratified by multiple variables.

ADDITIONAL RESOURCES

Additional information about malaria is available from the CDC at: www.cdc.gov/malaria/

CDC. Malaria surveillance--United States, 2004. MMWR 2006; 55(SS04):23-37. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/ss5504a2.htm?s_cid=ss5504a2_e

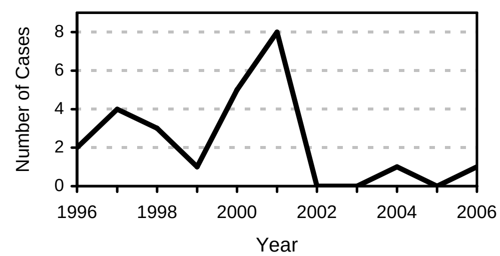
CDC. Transmission of *Plasmodium vivax* malaria--San Diego County, California, 1988 and 1989. MMWR 1990; 39(6):91-94. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/00000814.htm

MEASLES

CRUDE DATA	
Number of Cases	1
Annual Incidence[a]	
LA County	0.01[b]
California	---[c]
United States	0.01[d]

[a] Cases per 100,000 population.
[b] Rates based on less than 19 observations are unreliable.
[c] No reported cases.
[d] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

Figure 1
Measles
Reported Cases by Year of Onset
LAC, 1996-2006



DESCRIPTION

Measles is a vaccine-preventable disease caused by a paramyxovirus and is transmitted by contact with respiratory droplets or by airborne spread. Common signs and symptoms of measles include fever, cough, conjunctivitis, runny nose, photophobia, Koplik spots, and a generalized maculopapular rash. Severe complications are rare, but can include acute encephalitis and death from respiratory or neurologic complications. Immunocompromised individuals are more likely to develop complications. All persons who have not had the disease or who have not been successfully immunized are susceptible. The minimum clinical criteria for measles are fever of at least 101°F, a generalized rash lasting at least three days, and either cough, coryza, conjunctivitis, or photophobia. A case is confirmed by a positive IgM titer or a four-fold increase in acute and convalescent IgG titers.

DISEASE ABSTRACT

- From 64 measles suspect reports received at the LAC Immunization Program, there was only one confirmed measles case identified in LAC during 2006.
- During 2006, 6 measles cases were reported in California. Since all recent measles cases have been imported, an effective measles surveillance system needs to be maintained.

IMMUNIZATION RECOMMENDATIONS

- Measles disease can be effectively prevented by Measles-Mumps-Rubella (MMR) or Measles-Mumps-Rubella-Varicella (MMRV) vaccine, given in accordance with recommendations from the CDC's Advisory Committee on Immunization Practices (ACIP).
- Usually, two doses of measles-containing vaccine are given via MMR or MMRV vaccine. The first dose is recommended at 12 months of age. The second dose can be given as early as four weeks after the first dose, but is usually given at ages 4 to 6 years.
- Vaccination is recommended for those born in 1957 or later who have no prior MMR vaccination or history of disease. Proof of immunization with two MMR doses is recommended for health care workers and persons attending post secondary educational institutions as well as others who work or live in high-risk settings.
- Over 95% of those who receive the current live attenuated measles vaccine develop immunity.
- Although the titer of vaccine-induced antibodies is lower than that following natural disease, both serologic and epidemiologic evidence indicate that vaccine-induced immunity appears to be long-term and probably life-long in most individuals.
- Women should not become pregnant within 4 weeks of vaccination.
- Individuals who are severely immunocompromised for any reason should not be given MMR vaccine.

STRATIFIED DATA

Trends: Over the past 10 years, the number of confirmed measles cases has decreased significantly (Figure 1). Although absolute numbers are low, the number of reported measles cases started increasing in 1999. In 2002, 2003, and 2005, no confirmed cases of measles were identified in LAC, marking only three times this has occurred in more than 40 years. The single cases in 2004 and 2006 were imported cases, whose rash onsets occurred within 18 days of traveling outside of the United States.

Sex: Female.

Race/Ethnicity: Asian.

Seasonality: Rash onset in January.

Age: The case was 3 years of age.

Location: The case resides in SPA 2 (San Fernando HD) but the illness was not linked to local transmission. The case acquired measles while traveling to and from India and developed clinical symptoms of measles within 18 days of returning to the United States.

Vaccination Status: Due to a personal beliefs exemption, the case did not receive any MMR vaccine.

Laboratory Confirmation: The case was confirmed with a positive IgM antibody titer.

Complications: The case survived but was hospitalized for 4 days with dehydration and pneumonia.

COMMENTS

It is important to be reminded that while measles is no longer considered to be endemic in the United States, the virus continues to circulate in other parts of the world putting susceptible individuals at risk of measles infection. LAC's single measles case this year was identified in January. As previously mentioned, the case was an imported case who was unvaccinated. In March, the Colorado Health Department notified the LAC Immunization Program of their imported case who had 2 LAC contacts. In April, CDC notified local health departments of a Venezuelan measles case who was infectious while attending a conference in Chicago. Later that same month, the LAC Immunization Program was notified of an Australian case who was incubating measles during a 4-hour layover at LAX airport. In June, the CDC released a media advisory regarding a measles outbreak in Germany, which notified World Cup games attendees of potential exposure. Then from July to August, a multi-state investigation identified 3 measles cases associated with the adoption of children in China. Only one of the cases had documentation of having received 2 doses of a measles-containing vaccine. While no LAC measles cases were identified in association with any of the exposures in Colorado, Chicago, LAX, Germany, or China, the potential disease exposures serve as a reminder that we must continue to sustain high measles vaccine coverage levels. According to the most recent National Immunization Survey data, over 90% of children 19-35 months of age in LAC are vaccinated against measles. In addition, ensuring that travelers are immune to measles can minimize the importation of measles. Healthcare providers can play an important role in pre- and post-travel-related health screenings by promoting appropriate pre-travel vaccination and by being aware of travel history when evaluating symptomatic patients.

It is important that an effective measles surveillance system be maintained in LAC. For surveillance to be effective, suspected cases must be reported to the health department in a timely manner. The 2006 LAC case is a prime example of delayed reporting. Although healthcare providers suspected measles and ordered the appropriate laboratory tests, the case was not reported to the health department. Furthermore, the final diagnosis of "not measles" was made before final lab results were even available. When the labs were determined to be positive, the laboratory reported the results to the LAC Immunization Program. However, 22 days had passed since symptom onset. This is problematic because the maximum incubation period for measles is 18 days and the maximum communicability period is 4 days after onset. The extended reporting lag time led to delayed or missed opportunities for effective public health intervention. Fortunately, all contacts were immune to measles and no other cases were identified. In response to this situation, the LAC Immunization Program called the reporting facility to remind them that measles cases should be reported within one working day of identification of the suspected case, regardless of whether lab results are ready. Routinely reminding reporting facilities about the reporting mandates by the California Code of Regulations, Title 17, Section 2500 is an activity that should continue to be implemented.

In 2006, the 64 suspect measles reports came from a variety of sources. Half (n=32) of the suspect cases were first reported by laboratories, 17.2% (n=11) were reported by hospitals/clinics, 17.2% (n=11) were reported by school nurses, and the remaining 15.6% (n=10) were reported by other sources, including the state health department, other counties, and workplaces. Among the 64 suspect cases, 39.1% (n=25) had febrile-rash illnesses that were ruled out because they did not meet the minimum clinical criteria for measles. Thirty-seven of the 64 suspect cases (57.8%) had laboratory studies performed. For 5 of the 37 cases, testing was conducted due to clinical suspicion of measles; results were negative for 4 cases, ruling out measles as the cause of illness. The remaining 32 patients tested were reported to the health department by laboratories due to false positive lab results. Further investigation revealed that the individuals were asymptomatic and that measles antibody tests were performed to test for immunity as part of a routine physical examination, school entrance requirement, or employee health requirement.

It is the policy of the LAC Immunization Program to immediately investigate all suspect measles cases that are reported in order to verify diagnosis, medical history information, immunization status, and past travel history. Physicians and suspect cases are contacted directly by phone to verify the diagnosis and determine if the minimum clinical criteria for measles classification have been met. If a measles report involves a school or a sensitive setting like a health care facility, a school nurse or a medical administrator is contacted to assist in investigative efforts and to immediately implement isolation procedures necessary for preventing the spread of the disease. Susceptible contacts are identified and offered MMR vaccination to prevent natural measles occurrence. If vaccine is contraindicated, immune globulin (IG) may be given instead. IG is recommended for infants less than 6-months of age, pregnant women, and immunocompromised individuals.

Both clinical examination and laboratory tests are important in the diagnostic confirmation of the disease. Blood specimen collections are arranged for serological analysis by public health nurses or Immunization Program surveillance staff if physicians have not ordered them. The testing laboratory is contacted to obtain measles IgM and IgG antibody levels. Detection of both types of antibodies is important in disease testing. Measles IgM antibodies are detectable from 2 to 28 days after rash onset. The presence of IgG antibodies in the serum indicates prior exposure to measles, either by natural means or by immunization. In the absence of an IgM test, a four-fold rise in measles IgG antibody titers between an acute serum specimen and a convalescent specimen at 2 weeks later usually indicates current or recent measles infection.

In summary, the decline in the number of measles cases in LAC is attributable to the effectiveness of the MMR vaccine, diligent surveillance activities, and the success of the various outreach and educational programs implemented by the LAC Immunization Program and others to improve vaccination coverage rates in the county.

ADDITIONAL RESOURCES

Additional information about measles is available at:

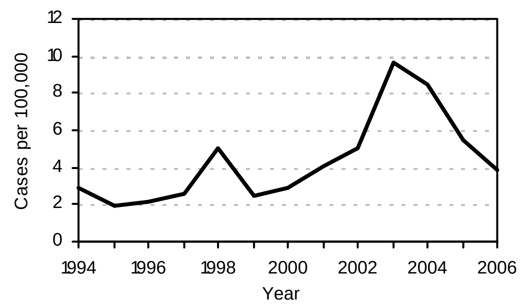
- National Immunization Program – www.cdc.gov/vaccines
- Immunization Action Coalition – www.immunize.org
- LAC Immunization Program – www.lapublichealth.org/ip

MENINGITIS, VIRAL

CRUDE DATA	
Number of Cases	373
Annual Incidence[a]	
LA County	3.9
United States	N/A
Age at Onset	
Mean	25
Median	24
Range	0–85 years

[a] Cases per 100,000 population.

Figure 1
Viral Meningitis
Incidence Rates by Year of Onset
LAC, 1994–2006



DESCRIPTION

Viruses are the major cause of aseptic meningitis syndrome, a term used to define any meningitis (infectious or noninfectious), particularly one with a 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 or confusion, nausea and vomiting and usually last from 7 to 10 days.

Nonpolio enteroviruses, the most common cause of viral meningitis, are not vaccine-preventable and account for 85% to 95% of all cases in which a pathogen is identified. Estimates from the Centers for Disease Control and Prevention (CDC) indicate that 10 to 15 million symptomatic enteroviral infections occur annually in the United States, which includes 30,000 to 75,000 cases of meningitis. Transmission of enteroviruses may be fecal-oral, respiratory or by another route specific to the etiologic agent.

Other viral agents that can cause viral meningitis include herpes simplex virus, varicella-zoster virus, mumps virus, lymphocytic choriomeningitis virus, human immunodefieciency virus, adenovirus, parainfluenza virus type 3, influenza virus, measles virus and arboviruses, such as West Nile virus (WNV). Since its arrival in Southern California in 2003, WNV should be considered an important cause of viral meningitis, especially during the summer and fall among adults; and the appropriate diagnostic tests should be obtained. Treatment for most forms of viral meningitis is supportive; recovery is usually complete and associated with low mortality rates. Antiviral agents are available for treatment of viral meningitis due to several herpes viruses: herpes simplex virus-1 (HSV-1), HSV-2, and varicella-zoster virus.

Supportive measures, and to a lesser extent antiviral agents, are the usual treatments for viral meningitis. Good personal hygiene, especially hand washing and avoiding contact with oral secretions of others, is the most practical and effective preventive measure.

DISEASE ABSTRACT

- The incidence of viral meningitis has continued to decrease since its peak in 2003 (Figure 1). The seasonal peak, usually very high, is seen only weakly this year (Figure 2).

- WNV infection contributed to fewer cases of viral meningitis in 2006 (1% of all cases) compared to 2005 (3%).
- No outbreaks were reported.

Trends: In 2006, there were a total of 373 cases of viral meningitis compared to 530 in 2005, representing a 30% decrease from 2005. The annual incidence also decreased, dropping from 5.5 per 100,000 in 2005 to 3.9 per 100,000 in 2006. This continues a decreasing trend from a peak incidence of 9.6 cases per 100,000 in 2003.

Seasonality: Enteroviruses demonstrate a seasonality in temperate climates that typically peaks in the late summer and early fall. WNV follows a similar pattern. The onset of viral meningitis cases in LAC usually follow this trend closely, as seen in the previous 5-year average in Figure 2 where approximately a hundred cases are seen each month from July through September. This trend appeared weakly in 2006, however, peaking in August with 46 cases (Figure 2).

Age: Infants less than 1 year old continued to have the highest age-group specific rate at 49 cases per 100,000 (Figure 3).

Sex: The male to female rate ratio of cases was nearly 1:1.

Race/Ethnicity: The incidence rates across race and ethnicity groups ranged from 2.3 to 4.2 cases per 100,000, the lowest occurring in Asian/Pacific Islanders. The rates were similar among Latinos, Whites, and Blacks (data not shown).

Location: The highest incidence of viral meningitis continued to occur in SPA 1 (13 per 100,000); the lowest in SPA 5 (1.6 per 100,000) (Figure 4). However, because SPA 5 had such a low case count (n=10), the calculated incidence rate is unstable.

Clinical Presentation: The case fatality rate remained low; only two deaths were reported in 2006 (less than one percent case fatality rate). Of the 15 cases in which an etiology was identified, 9 (60%) were caused by an enterovirus. WNV infection has been less prevalent as a cause of viral meningitis than in 2005. Only 27% of cases (n=4) in which an

Figure 2
Viral Meningitis
Cases by Month of Onset
LAC, 2006

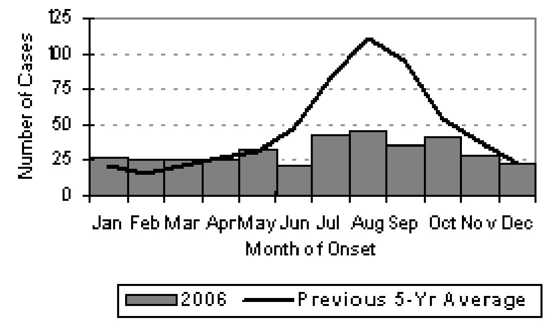


Figure 3
Viral Meningitis
Incidence Rates by Age Group
LAC, 2006 (N=373)

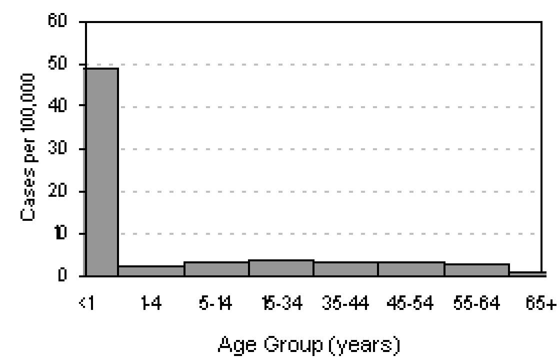
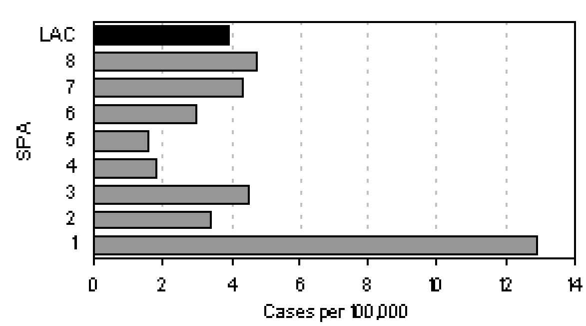


Figure 4
Viral Meningitis
Incidence Rates by SPA
LAC, 2006 (N=373)



etiology was known, or 1% of all cases, were associated with WNV infection. However, the viral etiology is not investigated in all cases; the etiologies of 96% of cases in 2006 remain unknown.

COMMENTS

The highest incidence in LAC in 2006, as well as for previous years, occurred among children less than one and those with residence in SPA 1 (Antelope Valley). It is common for small children who are not yet toilet trained to transmit enteroviruses—the most frequently identified etiology of viral meningitis — to other children or to adults who change their diapers, as these viruses can be found in the stool of infected persons. Though SPA 1 has the smallest population (n=342,804) of all SPAs in LAC, it continually carries the highest rates of viral meningitis in LAC. Reasons for this trend are unknown.

The low incidence in 2006 continues a decreasing trend since a substantial peak in 2003. That peak coincided with national and regional outbreaks, including California, which occurred due to serotypes of enteroviruses that are associated with an epidemic circulation pattern. Individual enterovirus serotypes have different temporal patterns of circulation; and the changes in predominant serotypes can be accompanied by large-scale outbreaks. However, no predictable patterns exist for these serotypes or for viral meningitis in general. There is significant yearly variation and no long-term trends have been identified.

The emergence of WNV in LAC in 2003 and subsequent introduction of WNV surveillance have not markedly affected the trend in overall viral meningitis annual incidence rates. Since 2003, increased reporting of viral meningitis and testing for underlying WNV infection have been encouraged among health care providers and hospital infection control practitioners. However, the peak incidence of viral meningitis in LAC did not correspond with the peak incidence of WNV, which occurred in 2004. Further, WNV meningitis only contributed 10% of cases at its highest incidence in 2004 and has decreased considerably since then.

Because surveillance for viral meningitis is passive, the number of cases reported annually is considered to be substantially lower than the actual burden of disease. Investigations are initiated only for outbreaks, not individual cases. No outbreaks occurred in 2006. Information about the causative agents of viral meningitis is rarely included with case reports because viral cultures and nucleic acid-based tests, such as PCR analysis of the cerebral spinal fluid, are not routinely performed at most medical facilities. Improvements in molecular testing capabilities should lead to faster diagnoses and more appropriate management of viral meningitis including less use of inappropriate antibiotics and fewer and shorter hospital admissions.

ADDITIONAL RESOURCES

CDC. Respiratory and Enteric Viruses Branch, Viral (Aseptic) Meningitis at:
www.cdc.gov/ncidod/dvrd/revb/enterovirus/viral_meningitis.htm

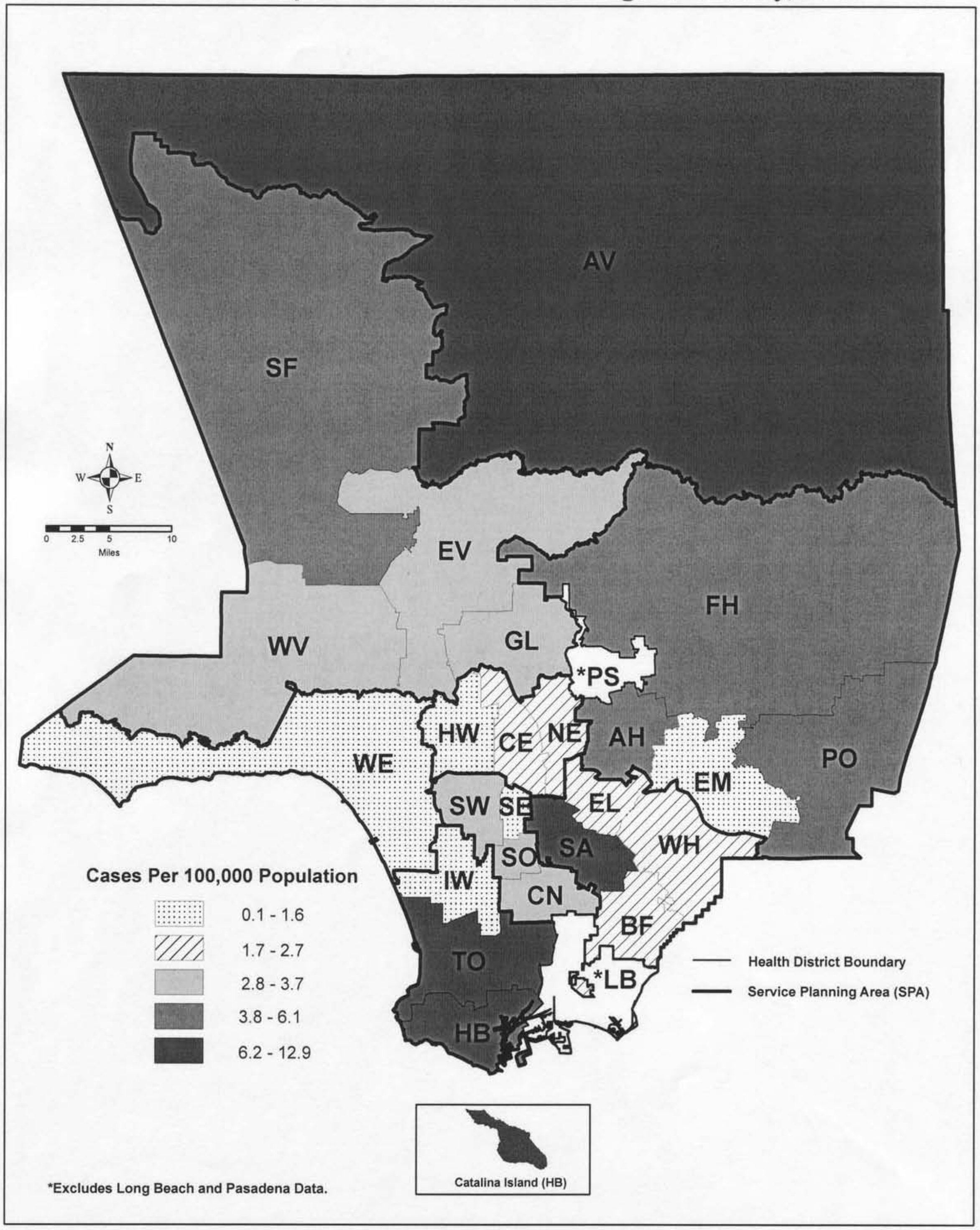
CDC. Respiratory and Enteric Viruses Branch, Non-Polio Enterovirus Infections at:
www.cdc.gov/ncidod/dvrd/revb/enterovirus/non-polio_entero.htm

Association of State and Territorial Directors of Health Promotion and Public Health Education, Infectious Facts, Viral Meningitis at: www.astdhpphe.org/infect/vmenin.html

CDC. Outbreaks of aseptic meningitis associated with echoviruses 9 and 30 and preliminary reports on enterovirus activity--United States, 2003. MMWR 2003; 52(32):761-764. Available at:
www.cdc.gov/mmwr/preview/mmwrhtml/mm5232a1.htm

CDC. Enterovirus surveillance--United States, 2002–2004. MMWR 2006; 55(6):153-156. Available at:
www.cdc.gov/mmwr/preview/mmwrhtml/mm5506a3.htm

Map 9. Meningitis, Viral
Rates by Health District, Los Angeles County, 2006*

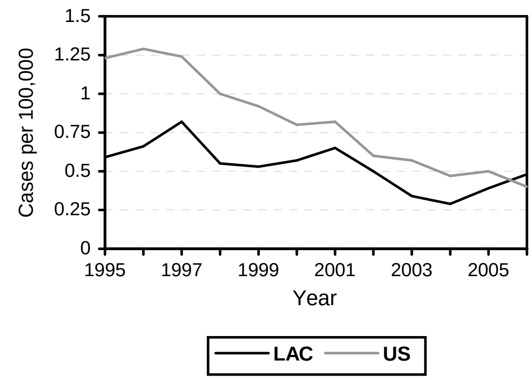


MENINGOCOCCAL DISEASE

CRUDE DATA	
Number of Cases	46
Annual Incidence[a]	
LA County	0.48
California	0.51[c]
United States[b]	0.40[c]
Age at Diagnosis	
Mean	32
Median	18.5
Range	<0–82 years

[a] Cases per 100,000 population.
[b] Based on 2005 population estimates and the Active Bacterial Core Surveillance Report.
[c] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

Figure 1
Meningococcal Disease
Incidence Rates by Year of Onset
LAC and US, 1995–2006

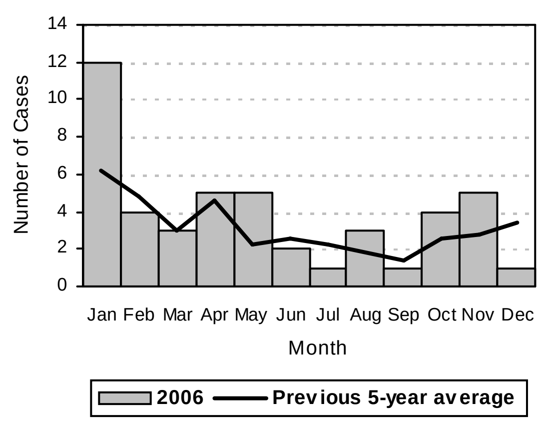


DESCRIPTION

Meningococcal disease 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 the *Neisseria meningitidis* bacterium. Common symptoms include sudden onset of fever, headache, nausea, vomiting, stiff neck, petichial rash and lethargy which can progress to overwhelming sepsis, shock and death within hours. Long-term sequelae include significant neurologic or orthopedic complications such as deafness or amputation secondary to disseminated intravascular coagulation and thromboses. Meningococcal disease affects all age groups but occurs most often in infants. Of the 12 serogroups, only A, C, Y, and W-135 are vaccine-preventable.

For the purpose of surveillance, the LAC DPH defines reports of invasive meningococcal disease as confirmed when *N. meningitidis* has been isolated from a normally sterile site (e.g., blood or CSF). In the absence of a positive culture, reports are defined as probable in the setting of clinical symptoms consistent with invasive meningococcal disease and when there is evidence of the bacteria in a normally sterile site by gram staining, polymerase chain reaction (PCR) analysis, or CSF antigen test.

Figure 2
Meningococcal Disease
by Month of Onset
LAC, 2006 (N=46)



DISEASE ABSTRACT

- Confirmed invasive meningococcal disease cases increased by 24% in 2006 compared to 2005 with 46 and 37 cases reported, respectively.
- Fewer deaths were documented in 2006: one death compared to two in 2005.
- There were 38 (83%) culture-confirmed cases:

11 (29%) from CSF, 22 (58%) from blood, and 5 (13%) from both blood and CSF (Figure 5). Thirty-four (74%) cases were serogrouped: 14 were identified as serogroup B (41%), 13 serogroup C, 5 serogroup Y, and 2 untypeable.
- A cluster of two cases reported in a high school prompted mass distribution of antimicrobial prophylaxis to students and staff .

STRATIFIED DATA

Trends: The incidence of invasive meningococcal disease increased by 23% to 0.48 per 100,000 population in 2006 (N=46) from 0.39 per 100,000 in 2005 (N=37) (Figure 1). Eighty-three percent (n=38) of cases were culture-confirmed in 2006 compared to 93% in 2005. The incidence rate has been slowly increasing in LAC since 2003 and is above the national rate of 0.35 per 100,000 estimated for 2005. Despite the increase, fewer deaths were documented in 2006: one death (2%) compared to two in 2005 (5%).

Seasonality: Most cases were reported during winter and early spring (Figure 2).

Age: The incidence rates among infants <1 year increased in 2006 (2.8 versus 2.1 per 100,000) compared to 2005. The rates among 15-34 years were similar to last year (0.3 versus 0.4 per 100,000). The rate among adults 55-64 increased slightly in 2006 (0.8 versus 0.6 per 100,000).

Sex: The male-to-female rate ratio was 1.1:1.

Race/Ethnicity: Invasive meningococcal cases were reported most frequently in Latinos (n=28, 61%) followed by whites (n=13, 28%), blacks (n=3, 6%), and Asians (n=2, 4%). The incidence rates by race/ethnicity are noted in Figure 4.

Location: Cases were reported from all eight Service Planning Areas (SPA). The number of cases was highest in SPA 6 (n=14) and SPA 2 (n=11), followed by SPA 7 (n=6); and finally SPAs 4, 5, 8 with 4 cases each.

Figure 3
Meningococcal Disease
Incidence Rates by Age Group
LAC, 2006

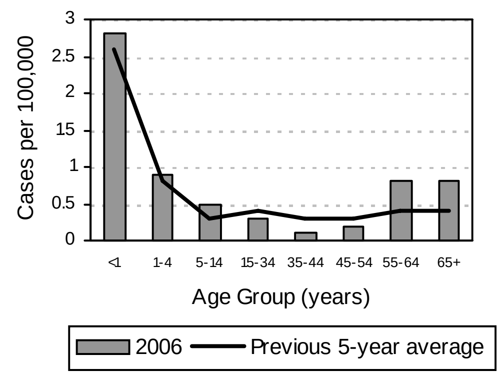
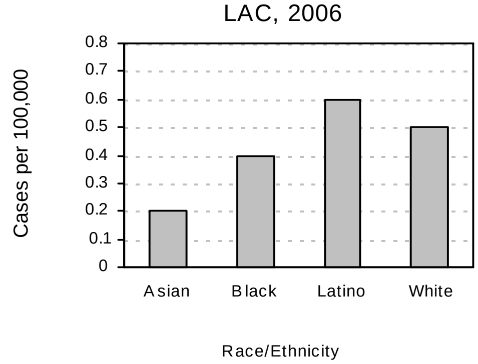


Figure 4
Meningococcal Disease
Incidence Rates by Race/Ethnicity
LAC, 2006



PREVENTION

Antimicrobial chemoprophylaxis of close contacts of sporadic cases of meningococcal disease remains the primary means for prevention of meningococcal disease. Close contacts include a) household members, b) day care 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 greater than 14 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 respective health district in LAC.

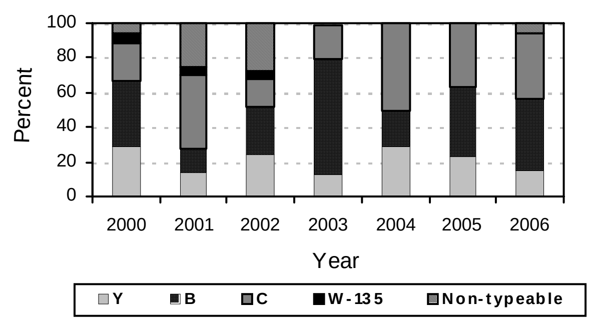
In 2004, a new quadrivalent meningococcal conjugate (MCV4), Menactra®, was approved for use in the United States. This vaccine protects against serogroups A, C, Y, and W-135, the same serogroups as MPSV4, but provides longer lasting immunity. MCV4 is recommended for use in persons aged 11 to 55 years, although the use of MPSV4 is acceptable when MCV4 is not available. Generally, only a single dose of either vaccine is recommended. As of 2006, MCV4 is part of the childhood vaccination schedule and recommended for all children between ages 11-12 years. Additionally, unvaccinated college freshman who live in dormitories are at higher risk for meningococcal disease and should be vaccinated with MCV4.

Although no noticeable changes were found with respect to the serogroup distribution of invasive meningococcal isolates from 2005 to 2006 and the introduction of MCV4 in 2004, enhanced surveillance for invasive *N. meningitidis* infections remains important (Figure 5). LAC DPH and the California Department of Health Services (CDHS) have continued to participate in enhanced meningococcal disease surveillance with the goals of (1) monitoring the epidemiology changes of meningococcal disease; (2) assisting with identification and management of cases and outbreaks; (3) assessing vaccine effectiveness; (4) ascertaining the usefulness of PCR in culture negative cases, particularly in patients treated with antibiotics prior to culture; and (5) helping contribute to improvements in the overall diagnosis and management of invasive meningococcal disease.

An analysis of two years of statewide meningococcal surveillance data is expected to be published in the coming year.

COMMENTS

Figure 5
Meningococcal Disease
by Serogroup
LAC, 2000–2006



As a part of public health meningococcal disease surveillance, for every culture-confirmed case reported to DPH, clinical laboratories are requested to send isolates to the LAC Public Health Laboratory (PHL) for serotyping**.** In 2006, the LAC PHL received 34 case isolates (89% of all culture-confirmed cases) for serogroup identification. Of these, 14 (41%) were serogroup B; 13 (38%) serogroup C; and 5 (15%) serogroup Y, and 2 (6%) were not typeable (Figure 5). As in 2004 and 2005, no serogroup W-135 isolates were identified. Whereas, in 2005 of the 25 isolates that were serogrouped, 10 (40%) were serogroup B, 10 (40%) serogroup C, and 5 (20%) serogroup Y. Therefore, the distribution of serogroups did not change substantially between 2005 and 2006 (Figure 5). The mean and median ages of the vaccine preventable cases were 44.2 and 55 years, respectively, and ranged from 0–82 years. Non-vaccine preventable serogroup B cases had a mean age of 25.7, a median age of 17.5 and range of 0–56. With greater widespread use of the MCV4 vaccine, the incidence of serogroups C, Y, and W-135 is expected to decline. However, due to the lack of universal vaccine protection against invasive meningococcal disease, clinicians must still maintain diagnostic clinical acumen.

Two students from the same high school in SPA 2 were reported with serogroup B meningococcal disease: one was a confirmed meningococcemia diagnosed by culture and the other a probable meningitis diagnosed by PCR. The cluster prompted to set up a point of distribution (POD) clinic at the high school where the cases attended. Antimicrobial prophylaxis was provided over a period of two days by LAC DPH to 2861 students and staff. Full details of this investigation are detailed in an accompanying 2006 Special Studies Report.

ADDITIONAL RESOURCES

CDC. Recommended immunization schedules for persons aged 0-18 years—United States, 2007. MMWR 2007; 55(51):Q1-4. Available at: www.cdc.gov/mmwr/PDF/wk/mm5551-Immunization.pdf

CDC. Active Bacterial Core Surveillance Report, Emerging Infections Program Network, *Neisseria meningitidis*, 2005. Available at: www.cdc.gov/ncidod/dbmd/abcs/survreports/mening05.pdf.

CDC. Prevention and control of meningococcal disease. Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 2005; 54(RR07):1-21. Available at: www.cdc.gov/mmwr/PDF/rr/rr5407.pdf

Meningococcal Disease Prevention Plan, Division of Communicable Disease, California Department of Health Services. Available at: www.dhs.ca.gov/ps/dcdc/disb/pdf/Meningococcal%20Plan%20Final%202003.pdf

CDC. Control and prevention of meningococcal disease. Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 2000; 49(RR07):1–10. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/rr4907a1.htm

CDC. Prevention and control of meningococcal disease and meningococcal disease and college students. Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 2000; 49(RR07):1–10. Available at: www.cdc.gov/mmwr/PDF/rr/rr4907.pdf

Raghunathan PL, Bernhardt SA, Rosenstein NE. Opportunities for control of meningococcal disease in the United States. Annu Rev Med 2004; 55:333-353.

MUMPS

CRUDE DATA	
Number of Cases	10
Annual Incidence[a]	
LA County	0.10[b]
California	0.09[c]
United States	2.22[c]
Age at Diagnosis	
Mean	31.5 years
Median	32.0 years
Range	3.0 – 56.0 years

[a] Cases per 100,000 population.
[b] Rates based on less than 19 observations are unreliable.
[c] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

Figure 1
Mumps
Incidence Rates by Year of Onset
LAC and US, 1996-2006

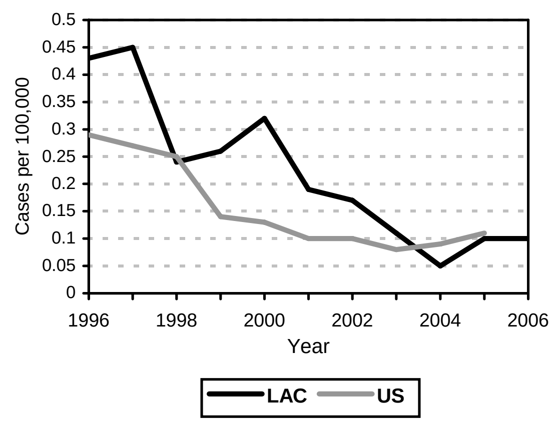
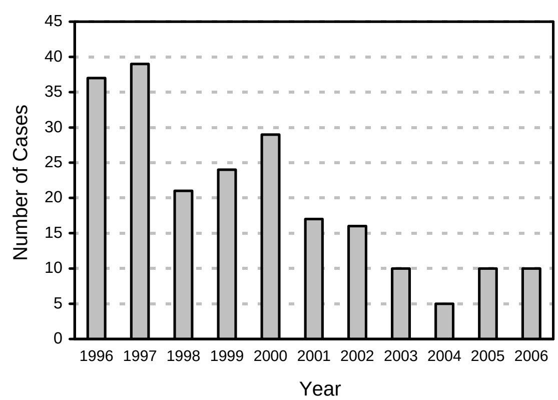


Figure 2
Mumps
Cases by Year of Onset
LAC, 1996-2006



DESCRIPTION

Mumps is a vaccine-preventable disease caused by an RNA paramyxovirus that is transmitted by direct contact with respiratory droplets from infected persons. Symptoms begin 14–18 days after exposure, with a range of 12–25 days, and include swelling of salivary glands, fever, and inflammation of the testes in teenage and adult males. Up to 20% of infected individuals may be asymptomatic. Sequelae include encephalitis, meningitis, orchitis, arthritis, and deafness. In addition, pregnant women who contract mumps are at increased risk of spontaneous abortions. Most reported cases are diagnosed based on clinical symptoms and do not have supporting laboratory confirmation (*i.e.*, positive IgM titer, significant increase between acute and convalescent IgG titers, or culture confirmation). The minimum clinical criteria for mumps is an acute onset of unilateral or bilateral swelling of the parotid or other salivary gland lasting $\geq$2 days without other apparent cause. Although single probable or confirmed cases are reportable, only outbreaks of two or more cases are investigated.

DISEASE ABSTRACT

- Greater media attention and public awareness of mumps following the multi-state mumps outbreak in the Midwest in 2006 resulted in twice as many suspect mumps reports compared to 2005.
- Of 103 suspect mumps reports received at the LAC Immunization Program during 2006, only 10 were identified as confirmed mumps cases.
- During 2006, there were 21 reported cases in CA, of which 48% were reported in LAC.

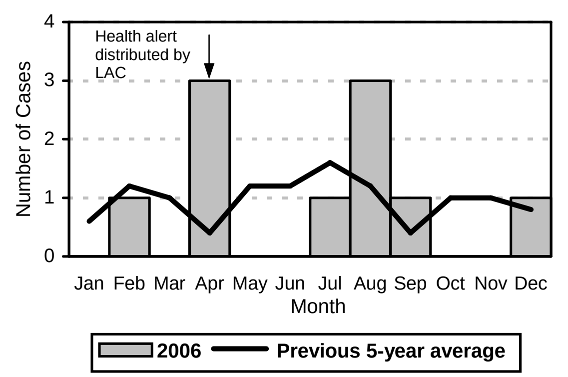
IMMUNIZATION RECOMMENDATIONS

- Two doses of mumps-containing vaccine, usually given as Measles-Mumps-Rubella (MMR), are normally recommended to achieve immunity. The first dose is recommended at 12 months of age. The second dose can be given as early as four weeks after the first dose, but is usually given at ages 4 to 6 years. Vaccination is recommended for those who have no prior MMR, particularly if they are in a high-risk setting.
- Approximately 90% of those who receive two doses of the current live attenuated mumps vaccine develop immunity.
- Generally, persons can be considered immune to mumps if they were born before 1957, have serologic evidence of mumps immunity, have documentation of physician-diagnosed mumps, or have documentation of vaccination with at least one dose of live mumps vaccine on or after their first birthday.
- Women should not become pregnant within 4 weeks of vaccination.
- Individuals who are severely immunocompromised for any reason should not be given MMR vaccine.

STRATIFIED DATA

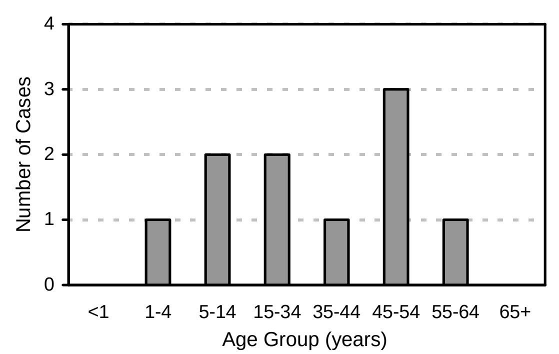
Trends: Since 1995, the annual number of cases of mumps has decreased by 76% (Figure 2). This decline reflects the effectiveness of the MMR vaccine in reducing the incidence of disease in the general population. The 2006 multi-state mumps outbreak in the Midwest area of the United States resulted in greater media attention and general public awareness of mumps. In LAC, twice as many suspect cases were reported in 2006 (n=103) compared to 2005 (n=50). Among the 103 suspect cases, 10 were identified as confirmed and 63 as probable cases. However, it should be noted that vaccination history and negative lab results were considered noncontributory in 2006 by the California Department of Health Services based upon studies conducted by the CDC during the Midwest outbreak. Thus, a large number of the probable cases this year would have been classified as false in previous years because they had documentation of 2 doses of MMR vaccine and/or negative lab results.

Figure 3
Mumps
Cases by Month of Onset
LAC, 2006



Seasonality: Historically, mumps incidence peaks during the winter and spring seasons. However, mumps cases have been reported throughout the year. In 2006, cases occurred throughout the year with peaks in April (n=3) and August (n=3). The summer months of July, August, and September accounted for 50% (n=5) of confirmed cases (Figure 3). The first MMWR report on the Iowa outbreak occurred in late March. LAC followed up with a health alert in early April subsequently increasing the number of suspect mumps reports.

Figure 4
Mumps
Cases by Age Group
LAC, 2006



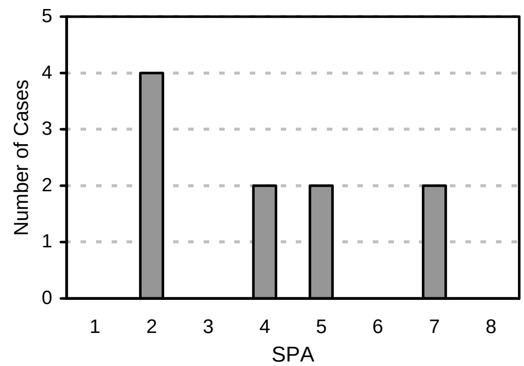
Age: Similar to previous years, 70% (n=7) of all confirmed cases in 2006 were in persons over the age of 15 (Figure 4). Children and young adults are more likely to have been fully immunized.

Sex: The male-to-female ratio of the confirmed cases was 1.5:1.

Race/Ethnicity: More than half of the confirmed mumps cases occurred among non-Latinos. There were 4 white cases, 3 Hispanic cases, 2 Asian cases, and 1 as unspecified race/ethnicity (data now shown).

Location: Confirmed cases were reported in four of the eight SPAs (Figure 5). Four of the cases (40%) resided in San Fernando Valley (SPA 2). Metro (SPA 4), West (SPA 5), and East (SPA 7) reported two cases each. None of the cases was epidemiologically linked to another 2006 case, although there were cases linked to 2007 cases (details in the Comments section below).

Figure 5
Mumps
Cases by Service Planning Area
LAC, 2006



COMMENTS

During January to October 2006, more than 5,700 mumps cases were reported in the United States, including more than 2,500 cases from the multi-state outbreak in the Midwest area. The predominant age group affected in the Midwest outbreak was the 18-24 year age group; a high proportion of whom were college students. The close-contact environment of college dormitories may have facilitated transmission of the mumps virus. The Midwest outbreak had a profound impact on mumps surveillance nationwide. On April 7, the Immunization Program released a health alert urging Los Angeles County healthcare providers to be vigilant about mumps. Greater media attention and general public awareness also increased the number of mumps reports. Vaccine efficacy was reevaluated, the case definition was slightly revised, and laboratory test guidelines were revised.

The efficacy of the mumps component of the MMR vaccine was reevaluated. Efficacy was estimated to be approximately 80% after one dose and approximately 90% after two doses. Thus, individuals who received 2 doses may still be susceptible to mumps. In the United States, where mumps vaccination coverage is high, most mumps cases will likely occur in persons who have received 2 doses [1].

In April 2006, the California Department of Health Services (CDHS) updated mumps surveillance guidelines and specimen collection guidelines for mumps virus testing. In addition, a mumps case report form was created and introduced for use in reporting probable and confirmed mumps cases to the state. Most notably, the CDHS also changed the classification of mumps cases. Prior to 2006, suspect mumps cases that received 2 doses of MMR vaccine were classified as false cases (regardless of clinical symptoms). In 2006, a suspect mumps case that met the clinical case definition (regardless of MMR vaccination history), is not laboratory-confirmed, and is not epidemiologically-linked to another probable or confirmed case was classified as a probable mumps case.

The value of mumps serological testing in previously vaccinated individuals was also questioned. In vaccinated individuals, the IgM response is highly variable and may be absent. In addition, it may not be possible to observe a 4-fold rise between acute and convalescent IgG titers. Thus, it was determined that a negative lab result, especially in previously vaccinated individuals, did not rule out mumps. Urine cultures were also no longer recommended because of lack of sensitivity. There are concerns with relying only on clinical classification of a mumps case. A clinical diagnosis of mumps may be unreliable since agents other than the mumps virus can cause parotitis. Parotitis can also be caused by parainfluenzae virus types 1 and 3, influenza A virus, Coxsackie A virus, echovirus, lymphocytic choriomeningitis virus, human immunodeficiency virus, and other non-infectious causes such as drugs, tumors, immunologic diseases, and obstruction of the salivary duct. As a result of the new case definition and laboratory test guidelines, a large number of suspect cases that would have been classified as false prior to 2006 were classified as probable in 2006.

Cluster Identification: None of the confirmed cases in 2006 were epidemiologically linked to each other. One case was linked to a 2005 case. Another case was exposed in the Phillipines and was subsequently linked to two cases with onset in 2007. None of the cases reported traveling to the states involved in the Midwest outbreak.

Vaccination Status: Only two of the confirmed cases were fully immunized with 2 doses of MMR vaccine. One case (age 3) had received 1 dose of MMR vaccine but was up-to-date for his age. The remaining 7 cases did not know or remember their vaccination status.

Laboratory Confirmation: Ninety percent (n=9) of the confirmed cases had supporting laboratory confirmation. One case was epidemiologically linked to a 2007 lab-confirmed case in another state.

REFERENCE

1. CDC. Brief report: update: mumps activity--United States, January 1–October 7, 2006. MMWR 2006; 55(42):1152-1153.

ADDITIONAL RESOURCES

Additional information is available at:

- National Immunization Program – www.cdc.gov/vaccines
- Immunization Action Coalition – www.immunize.org
- LAC Immunization Program – www.lapublichealth.org/ip

PERTUSSIS (WHOOPING COUGH)

CRUDE DATA	
Number of Cases	150
Annual Incidence[a]	
LA County	1.56
California	4.84[b]
United States	5.27[b]
Age at Diagnosis	
Mean	13.6 years
Median	6.0 years
Range	8 days–89 years

[a] Cases per 100,000 population.
[b] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

Figure 1
Pertussis
Incidence Rates by Year of Onset
LAC and US, 1996-2006

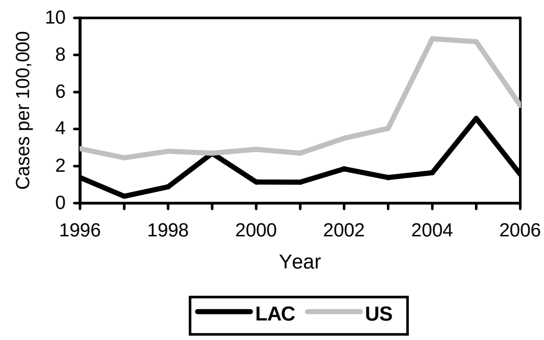
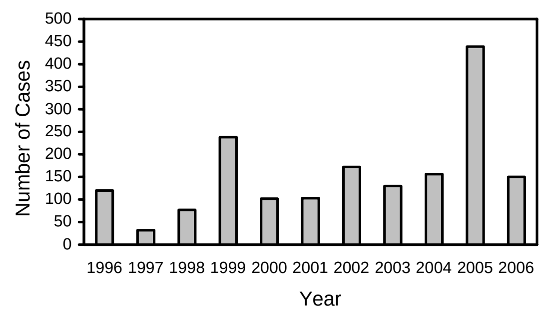


Figure 2
Pertussis
Cases by Year of Onset
LAC, 1996-2006



DESCRIPTION

Pertussis, commonly known as whooping cough, is a vaccine-preventable disease spread by close contact with the respiratory secretions of infected individuals. Typical symptoms include paroxysmal coughing, inspiratory whooping, and post-tussive vomiting. Complications include pneumonia, seizures, and encephalopathy. Infants under 1 year of age are at highest risk for developing severe complications.

The minimum clinical criteria for pertussis is a cough lasting at least two weeks with paroxysms of coughing, inspiratory "whoop," or post-tussive vomiting, without other apparent causes. Pertussis is confirmed by either positive *B. pertussis* culture or PCR.

DISEASE ABSTRACT

- Following a record-high of 438 cases in 2005, 150 cases were reported in 2006, which is similar to pre-2005 baseline levels.
- Preceding their illness, less than half of the cases in 2006 indicated contact to a person who had a prolonged cough.
- Of the 2006 cases that could have been fully immunized and protected against pertussis, approximately one fourth were not adequately immunized.

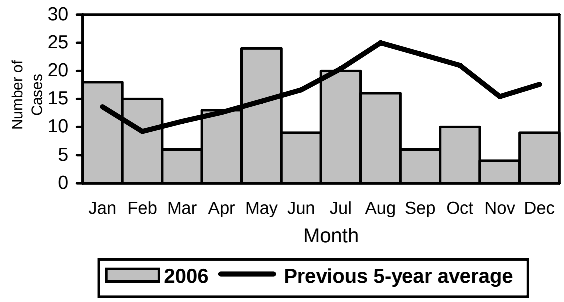
IMMUNIZATION RECOMMENDATIONS

- A pertussis-containing vaccine should be administered at 2 months, 4 months, 6 months, 15–18 months, and 4–6 years of age to provide protection against the disease.
- Immunity conferred by the pertussis component of the DTP/DTaP vaccine decreases over time, with some vaccinated individuals becoming susceptible to pertussis 5–10 years following their last dose.
- In Spring 2005, 2 Tdap vaccines were licensed for use in adolescents and adults, one for persons aged 10-18 years (Boostrix, GlaxoSmithKline) and the other for persons aged 11-64 years (ADACEL, Sanofi Pasteur).

STRATIFIED DATA

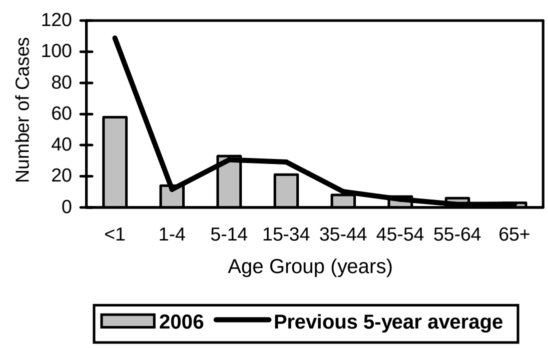
Seasonality: Following the record-high number of cases reported in 2005, a higher number of cases were reported during the first 5 months of 2006 compared to the previous five-year average. The number of cases peaked in May, which accounted for 16% (n=24) of cases. (Note: The only LAC pertussis outbreak in 2006 occurred in May and involved 4 cases.) From June to December, the number of 2006 cases was lower than the previous five-year average during this same time period. Typically, the summer months have the highest pertussis incidence in LAC. In 2006, 46% (n=69) of reported cases had disease onset during the months of May, June, July, and August. (Figure 3)

Figure 3
Pertussis
Cases by Month of Onset
LAC, 2006



Age: Although the majority of reported cases are still in children less than one year of age, the proportion of cases in the <1 age group is smaller in 2006 (39%) compared to the previous five year average (54%). As expected, cases are increasing among adolescents and adults, as evidenced by the fact that 30% (n=45) of the cases were over 14 years of age (Figure 4) in 2006 compared to an average of 24% (n=49) in the previous five years. Increased recognition and diagnosis of pertussis in older age groups has contributed to the increase in reported cases among adolescents and adults.

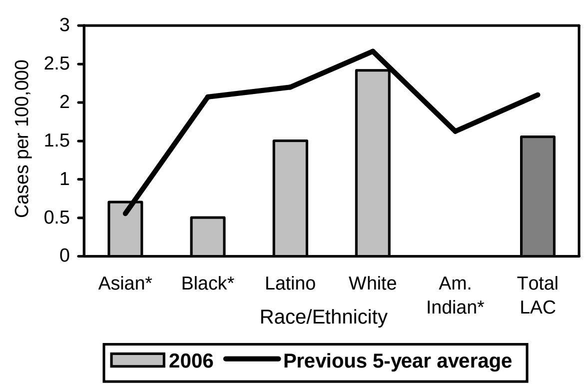
Figure 4
Pertussis
Cases by Age Group
LAC, 2006



Sex: The male-to-female case ratio was approximately 1:1.6.

Race/Ethnicity: After adjusting for the age differential in the cases, incidence rates in 2006 for blacks, Latinos, whites, and American Indians were lower than the previous 5-year averages (Figure 5). However, it should be noted that the previous 5-year average is influenced by the high incidence rates reported in 2005. Only whites had a higher incidence rate than the total LAC rate. The incidence rate for Latinos was approximately equivalent to the total LAC rate. However, the LAC population proportion of whites (30%) is much lower than that for Latinos (48%).

Figure 5
Pertussis
Age-Adjusted Incidence Rates by
Race/Ethnicity
LAC, 2006



*Rates unreliable due to small number of observations.

Location: For the second year in a row, Antelope Valley (SPA 1) had the highest incidence rate of 3.5 cases per 100,000 (n=12). Of the 12 cases reported in SPA 1, 42% (n=5) were epidemiologically linked to cases living within two households. The second highest incidence rate occurred in East (SPA 7) with 2.0 cases per 100,000 (n=27), followed by West (SPA 5) with 1.7 cases per 100,000 (n=11), South (SPA 6) with 1.6 cases per 100,000 (n=17), San Fernando Valley (SPA 2) with 1.5 cases per 100,000 (n=32), South Bay (SPA 8) with 1.4 cases per 100,000 (n=16), San Gabriel Valley (SPA 3) with 1.2 cases per 100,000 (n=21), and Metro (SPA 4) with 1.1 cases per 100,000 (n=14).

At the health district level, Bellflower (n=13) and Antelope Valley (n=12) had the highest incidence rates, each reporting 3.5 cases per 100,000. Compton had 2.7 cases per 100,000 (n=8), followed by Harbor with 2.4 cases per 100,000 (n=5). The lowest incidences rates were in El Monte and Inglewood health districts, each reporting only 1 case and an incidence rate of 0.2 cases per 100,000.

COMMENTS

In 2005, two Tdap vaccines were newly licensed for use in adolescents and adults. The Immunization Program conducted multiple intervention activities (*i.e.*, health alerts, fact sheets, a symposium) to increase the community's awareness of pertussis cases in individuals of all ages. In addition, LAC experienced a significantly high incidence rate of 4.5 cases per 100,000, which was consistent with similar increases throughout California and the United States. It was also observed that more cases were being reported among adolescents and adults. Whether the increase in pertussis incidence represented a true increase in disease or improved recognition and reporting remains unclear.

During 2006, the Immunization Program continued to promote the Tdap vaccines. Because they have yet to be provided to the population at large, data on the impact of the vaccines is not yet available. No county-wide pertussis-specific intervention activities were conducted. The only outbreak of pertussis occurred at a local university where four epidemiologically-linked cases were identified, prompting the health district to conduct a Tdap vaccination clinic in which 201 faculty and students were vaccinated. Although the 2006 incidence rate in LAC decreased to pre-2005 baseline levels (1.56 cases per 100,000), adolescents and adults now comprise a larger proportion of cases. As discussed previously in this report, infants less than one year of age no longer make up the overwhelming majority of cases. However, infants still account for the majority of complications/hospitalizations. The only fatal case in 2006 occurred in an infant less than 2 months of age. Thus, in order to protect the population at large, it is critical that high DTaP and Tdap coverage rates are achieved in LAC.

<u>Trends</u>: Pertussis incidence normally peaks every 3 to 5 years. Between 1990 and 1999, there was an annual average of 101 cases reported, with the highest incidence occurring in 1999 (n=238). During 2000-2004, an annual average of 133 cases was reported, with the highest incidence occurring in 2002 (n=172). In 2005, 439 cases were reported, which was the highest number of cases reported in more than 35 years. In 2006, 150 cases were reported, which is comparable to pre-2005 baseline levels.

<u>Laboratory Confirmation</u>: More than half of the reported cases (55%, n=83) were not laboratory confirmed by either *B. pertussis* culture or PCR.

<u>Vaccination Status</u>: Less than one fifth of cases (14%, n=21) were younger than two months of age and were too young to receive pertussis vaccine. About 43% (n=65) of cases were 10 years of age or older; so even if they were fully immunized in early childhood, they would not have had complete immunity against pertussis in 2006 and would thus be eligible for Tdap vaccine.

Approximately 23% (n=35) of cases were between 2–6 months of age. Of these, 80% (n=28) were up-to-date with pertussis vaccination for their age, but would not have developed full immunity against pertussis. Of the 29 children who could have had full immunity from vaccination (7 months to 9 years old), 21 (72%) were fully up to date. The previous 5-year trend has indicated that, on average, 65% of cases 7 months to 9 years of age were adequately immunized.

<u>Complications/Hospitalizations</u>: Approximately 37% (n=55) were hospitalized, with an average hospital stay of 7 days (range 1-24 days). Among the hospitalized cases, 85% (n=47) were less than one year of age. Of the 15 cases who developed pneumonia, 8 (53%) were infants less than 1 year of age. One of the 15 cases with pneumonia, a child in the 1-4 year age group, also developed seizures. One additional case in the 1-4 year age group developed seizures.

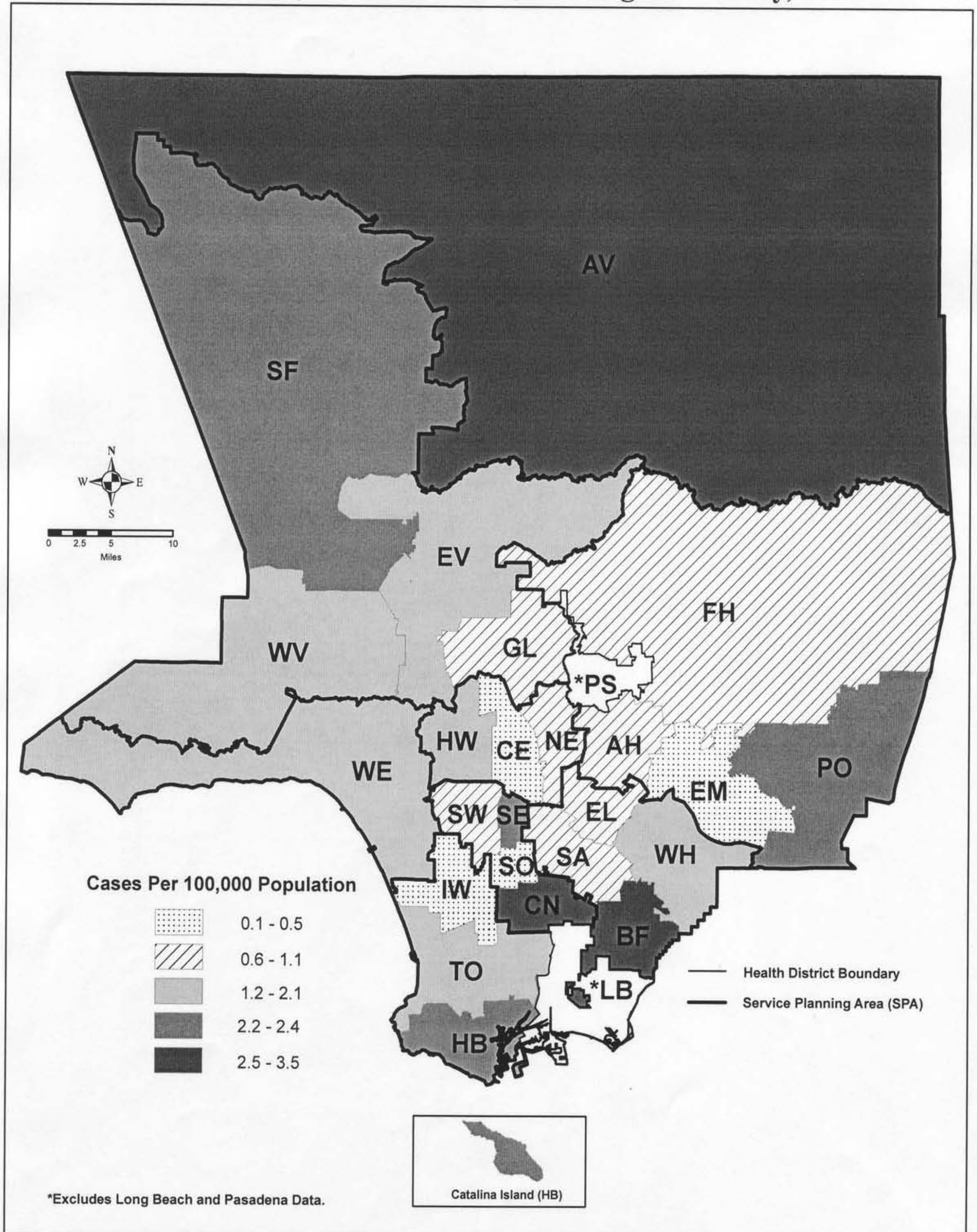
Case Fatalities: There was one pertussis-related death in 2006. The fatality occurred in a Hispanic female infant who was less than 2 months of age and was too young to receive pertussis vaccine. The principal diagnosis in the discharge/death summary was caradiorespiratory failure. The female infant died 6 days after cough onset and a PCR test detected *Bordetella* pertussis DNA. The infant was exposed to her twin brother whose cough onset was 3 weeks prior. Earlier consideration of pertussis for the brother would have initiated the administration of appropriate chemoprophylaxis to close contacts. Disease and death may have been prevented in this female infant.

ADDITIONAL RESOURCES

Additional information is available at:

- National Immunization Program – www.cdc.gov/vaccines
- Immunization Action Coalition – www.immunize.org
- LAC Immunization Program – www.lapublichealth.org/ip

Map 10. Pertussis
Rates by Health District, Los Angeles County, 2006*



*Excludes Long Beach and Pasadena Data.

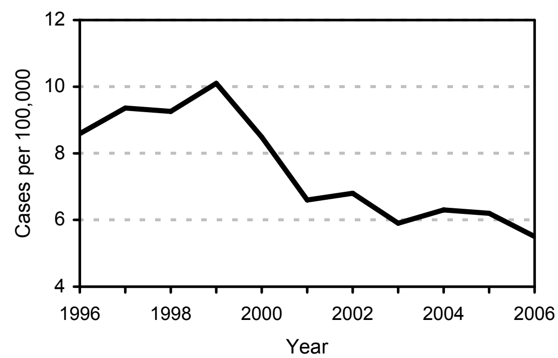
PNEUMOCOCCAL DISEASE, INVASIVE

CRUDE DATA	
Number of Cases	533
Annual Incidence[a]	
LA County	5.5
United States	14.0[b]
Age at Diagnosis	
Mean	52
Median	56
Range	0–101 years

[a] Cases per 100,000 population.

[b] National projection of IPD incidence from Active Bacterial Core Surveillance areas data, 2005 [1].

Figure 1
Invasive Pneumococcal Disease
Incidence Rates by Year of Culture
LAC, 1996–2006



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 discharge and attacks various parts of the body resulting in pneumonia, bacteremia, and meningitis. *S. pneumoniae* has become increasingly resistant to antibiotics during the last decade. Disease caused by *S. pneumoniae* is vaccine-preventable.

ACDC has followed IPD as a special 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 isolate for *S. pneumoniae* collected from a normally sterile site (e.g., blood, cerebral spinal fluid). Antibiotic susceptibility is determined by disk or dilution diffusion. Minimum inhibitory concentration (MIC) breakpoints utilized 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 nonsusceptible to an antibiotic if the results indicate intermediate or high-level resistance.

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 are not counted in LAC surveillance, therefore the data presented in this report underestimate all disease caused by *S. pneumoniae* in LAC.

DISEASE ABSTRACT

- The incidence rate decreased in LAC in 2006.
- There was no change in the overall percentage of penicillin nonsusceptible infections. However, an increase was observed in the 45-64 years age group while all other age groups remained approximately equal or decreased from 2005 (Figure 3).
- The highest incidence of IPD continued to be among blacks—the incidence rate of this group was at least twice as high as that of whites or Latinos (Figure 4).

STRATIFIED DATA

Trends: IPD occurred at an incidence rate of 5.5 per 100,000 in 2006 (N=533), a decrease from the previous year (6.2 per 100,000, N=590) (Figure 1).

Seasonality: The seasonal trend in 2006 followed the typical peak for IPD in the winter months, dropping in the spring and summer months (Figure 2).

Sex: The male-to-female rate ratio was 1.1:1. Males had a slightly higher incidence than females (6 vs. 5 cases per 100,000).

Age: The age of IPD cases ranged from birth to 101 years old with a mean of 52 years and median of 56 years. Compared to previous years, the incidence greatly decreased in children <1 year and in persons older than 65 years. A slight decrease was also observed in the 1-4 year age group. The distribution of incidence across the remaining age groups in 2006 remained similar to previous years (Figure 3).

Race/Ethnicity: The highest incidence of IPD occurred among blacks. With an incidence of 10.2 per 100,000, this rate was at least twice as high as that of whites or Latinos (Figure 4).

Disease Severity: During 2006, hospitalization status was known for 80% of the cases. Of these, 94% were hospitalized. Hospitalization was more frequent in cases older than 65 years (98%) and occurred less in children aged less than 5 years (78%). The overall case fatality was 14%, slightly higher than the national case fatality (11%) [1]. Most deaths occurred among adults 65 years and older (43% [n=16]); however, the 45–64 age group followed closely at 32% (n=12).

Antibiotic Susceptibility: Since 2004, there has been an increasing proportion of isolates nonsusceptible to trimethoprim-sulfamethoxazole (TMP-SMZ), increasing to 25% in 2006 (n=37). The percent of isolates nonsusceptible to penicillin and erythromycin remained the same as 2005, while cefotaxime increased (Figure 5). Almost all reported cases had antibiotic resistance information provided (95%).

Figure 2
IPD Cases By Month of Culture
LAC, 2006

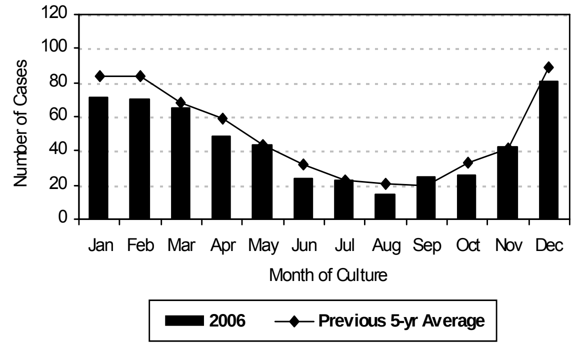


Figure 3
Incidence Rates of IPD Cases by Age
LAC, 2004-2006

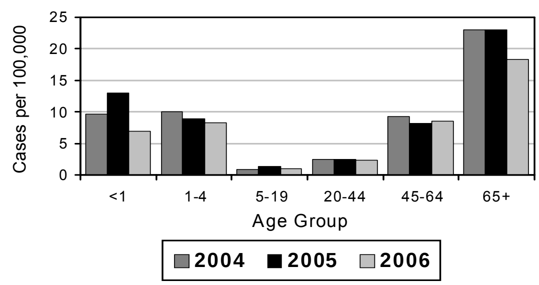
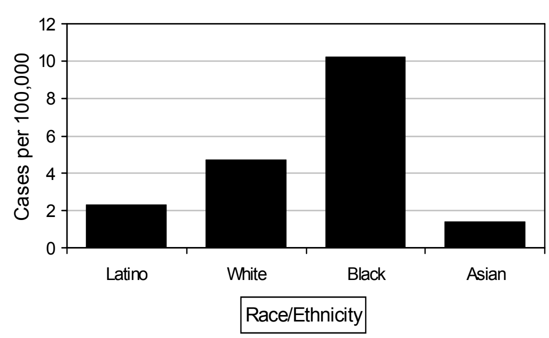


Figure 4
Incidence Rates of IPD Cases by Race/Ethnicity
LAC, 2006



The proportion of cases with penicillin nonsusceptible *S. pneumoniae* (PNSP) isolates decreased or remained the same in almost all age groups except in cases aged 45 to 64 years. In this age group there has been an increasing trend of greater nonsusceptibility to penicillin since 2004.

PREVENTION

Two effective vaccines are available for pneumococcal disease. Heptavalent pneumococcal conjugate vaccine (Prevnar®) is recommended by the Advisory Committee on Immunization Practices (ACIP) for all children less than age 2 years, and for children up to age 5 years who are at high risk of invasive pneumococcal infections. The 23-valent pneumococcal polysaccharide vaccines (Pnu-Imune®23 and Pneumovax®23) are recommended for all adults ≥65 years and those over age 2 years who are at high risk of invasive pneumococcal disease. For children aged 2 to 5 years who are at high risk of invasive pneumococcal infections, ACIP recommends use of pneumococcal conjugate vaccine followed at least 2 months later by the 23-valent pneumococcal polysaccharide vaccine. This regimen provides protection against a broader range of serotypes, although supporting data are limited [2].

Figure 5
IPD Isolates Nonsusceptible to Selected Antimicrobial Agents
LAC, 2004–2006

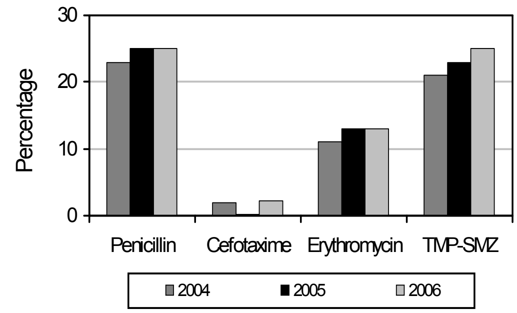
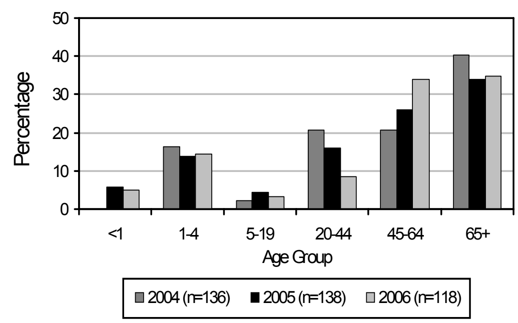


Figure 6
PNSP Isolates by Age
LAC, 2004–2006



COMMENTS

LAC experienced a decline of IPD in 2006, especially in those aged less than one year or older than 65 years. Though the overall proportion of PNSP isolates remained the same as 2005 (25%), a 30% increase of PNSP was observed in the 45 to 64 years age group, making it nearly equal to the proportion of PNSP isolates in the 65 years and older age group.

Incidence of IPD in blacks is over two times the incidence in whites or Latinos in LAC. The ratio of black-white incidence is similar to that found nationally; however, the incidence is much lower for both whites and blacks, which are 12 and 25 per 100,000 in the national population, respectively [1]. Interestingly, black IPD cases were more likely to be female (52%) and aged between 45 and 64 years (53%) when compared to non-blacks (45% female and 32% aged 45-64 years). Studies have indicated that the difference in incidence among blacks is associated with rates of breastfeeding, attendance in daycare, and underlying infections such as HIV [3].

Laboratories are the source for many of the IPD case reports to ACDC: 58% of cases were reported by laboratories only. Much of the limitations in the data are due to the minimal access that laboratories have to patient information. Race/ethnicity data and outcome status, in particular, are often missing from laboratory reported cases. Only 65% of case reports contained race/ethnicity data and 49% contained outcome status. The unavailability of outcome status is further exacerbated by the requirements of laboratory reporting procedures. Cases often are reported before the final outcome is known due to the requirement to report positive cultures within seven days. Therefore, case fatality rates may be unreliable.

REFERENCES

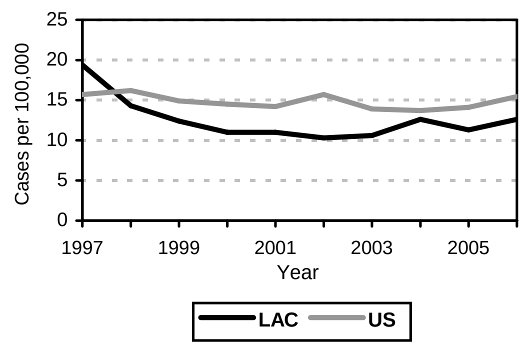
1. Active Bacterial Core Surveillance Reports from 1997 to 2005 from the CDC's Division of Bacterial and Mycotic Diseases. Report available at: www.cdc.gov/ncidod/dbmd/abcs/survreports.htm
2. CDC. Prevention of pneumococcal disease: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1997; 46(RR08):1–24.
3. Flannery B, Schrag S, Bennett NM, et al. Impact of childhood vaccination on racial disparities in invasive Streptococcus pneumoniae infections. JAMA 2004; 291(18):2197-2203.

SALMONELLOSIS

CRUDE DATA	
Number of Cases	1217
Annual Incidence[a]	
LA County	12.6
California	13.67[b]
United States	15.45[b]
Age at Diagnosis	
Mean	27.7
Median	22
Range	<1-95

[a] **Cases per 100,000 population.**
[b] Calculated from 2007 Summary of notifiable diseases issue of MMWR **(56:853-863).**

Figure 1
Salmonellosis
Incidence Rates by Year
LAC and US, 1997–2006



DESCRIPTION

Salmonellosis is caused by a 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, 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 12–36 hours for gastroenteritis, longer and variable for other manifestations. Communicability lasts as long as organisms are excreted, usually from 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, have recently taken or are taking broad-spectrum antibiotic therapy or immunosuppressive therapy, or those 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.

DISEASE ABSTRACT

- The LAC 2006 salmonellosis crude rate increased 11.5% when compared to 2005 (Figure1). This rate continues to remain below both the state and national rates.
- *Salmonella* serotype *enteritidis* was again the most common serotype in 2006. However, the percent of change was a decrease of 9.1 % due to a decrease in the total number of isolates (Table 1).
- Nine outbreaks were investigated in 2006, compared to four in 2005.
- SPA 5 continues to have the highest rate (16.3 per 100,000) of salmonellosis during 2006.

STRATIFIED DATA

Trends: The rate of salmonellosis cases for LAC in 2006 was 12.6 cases per 100,000 population, an 11.5% increase from the 2005 rate of 11.3 but similar to the 2004 rate of 12.6 (Figure 1). This rate remains below the national rate. Reasons for this increase are unknown but may be due to increases in the black and Asian population groups and an increase in the number of outbreaks investigated in 2006. ACDC continues to include "presumptive cases" those that meet a clinical case definition and have an epidemiological link to a laboratory confirmed case. If the presumptive cases are removed, the 2006 rate decreases to 12.3 per 100,000 population.

Salmonella Serotypes: For the third year, *S*. e*nteritidis* was the number one serotype, however, the incidence has decreased to 26.9% of total isolates serotyped.

Table 1. Most Frequent *Salmonella* Serotypes—LAC, 2005–2006

Serotype	2005 (N=1,032)*		2006 (N=1,217)*		
	No.	Percent	No.	Percent	%Change
Enteritidis	306	29.6	328	26.9	-9.1
Typhimurium**	150	14.6	173	14.2	-2.7
Newport	60	5.8	76	6.2	+7.4
Heidelberg	47	4.5	49	4.0	-11.6
I 4,5,12:i:-	32	3.1	48	3.9	+25.8
Montevideo	16	1.5	47	3.8	+149.0
Oranienburg	24	2.3	27	2.2	-4.6
Stanley	7	0.7	27	2.2	+227.0
Braenderup	22	2.1	23	1.9	-11.3
Infantis	11	1.1	23	1.9	+77.3
Mbandaka	16	1.5	23	1.9	+21.9

* Includes only serotyped isolates. (Eight cases for 2005 had two different serotypes of *Salmonella*)
** Includes *S.* Typhimurium var. Copenhagen.

Seasonality: In 2006, incidence again peaked in July (Figure 2) and was again dramatically greater than the five-year average. Incidence was also greater than the five-year average for the months of June, August, October, November and December. There were outbreaks recorded for the months of June, July, August, October and December (Table 2).

Age: As shown in Figure 3, the highest age group rates of infection occurred among infants aged less than one year (69.0 per 100,000 population) followed by children aged 1–4 years (38.1 per 100,000 population). This is typical for salmonellosis. The rate for all age groups except adults aged 35-44 years is higher than the five-year average.

Hospitalized: In 2006, 19% of cases were hospitalized for more than 24 hours, compared to 23.0% in 2005. Ages ranged from less than 1 year to 95 years. The average age of the hospitalized patient was 39.7 years and the median age was 39 years.

Sex: The male-to-female rate ratio was 1:1.06.

Figure 2
Salmonellosis
Cases by Month of Onset
LAC, 2006

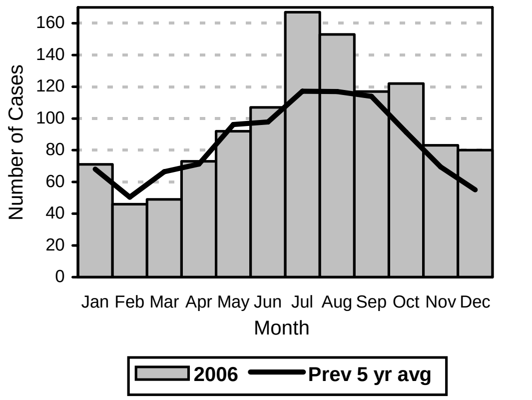
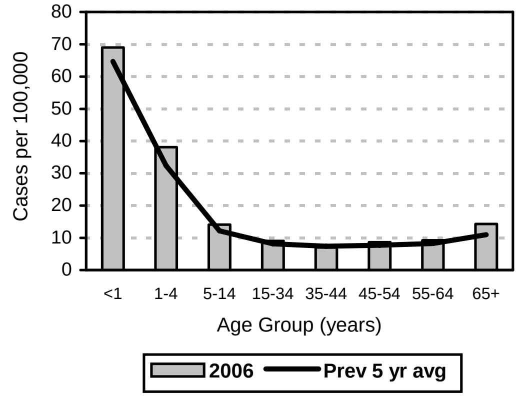


Figure 3
Salmonellosis
Incidence Rates by Age Group
LAC, 2006



Race/Ethnicity: Again, the highest age-adjusted rate was among whites (13.7 per 100,000 population), followed by Latinos (13.1 per 100,000 population) then Asians (12.9 per 100,000 population), and blacks (11.8 per 100,000 population, Figure 4). The rate for whites was lower than 2005 (15.4 per 100,000). The rates for Latinos, Asians and blacks were higher than 2005 (10.3, 9.1 and 8.9 per 100,000, respectively). This may be due to high numbers of family clusters in these populations and outbreaks that involved primarily Latino, Asian and black cases.

Location: East Los Angeles District in SPA 4had the highest district rate with 21.8 cases per 100,000. The lowest district rate was in El Monte Health District (SPA 3) with 5.7 cases per 100,000. Of all SPAs, SPA 5 again had the highest rate with 16.0 cases per 100,000. SPA 1 again had the lowest rate at 9.5 cases per 100,000 (Figure 5). All SPAs had an increase in rate with the exception of SPA 8. No single SPA had a rate significantly higher or lower than LAC average.

Figure 4
Salmonellosis
Age-Adjusted Rates by
Race/Ethnicity
LAC, 2005–2006

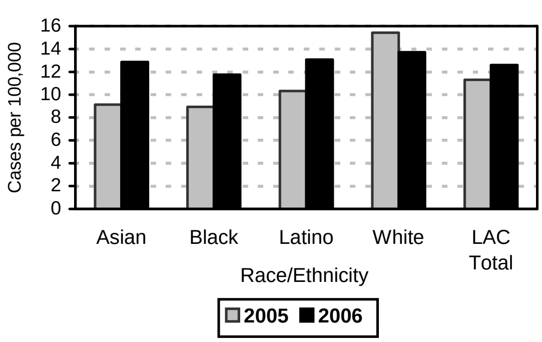
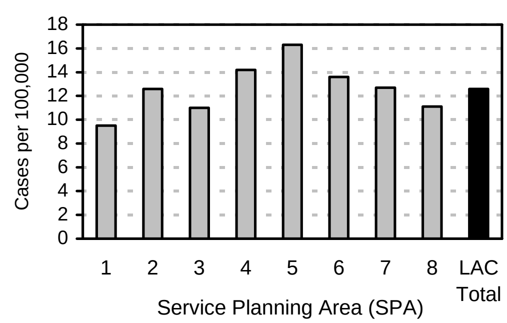


Figure 5
Salmonellosis
Incidence Rates by
Service Planning Area
LAC, 2006



PREVENTION

Each outbreak of salmonellosis is investigated and preventive measures are recommended. Review of investigation reports shows that many persons engage in high-risk food handling behaviors such as: consumption of raw or undercooked meats, or produce, use of raw eggs; not washing hands and/or cutting boards after handling raw poultry or meat; and having contact with reptiles. These investigations demonstrate a need for improved public education on proper handling and preparation of produce and animal-derived foods and the risk related to handling reptiles.

Health education targeted at specific high-risk groups is an ongoing necessity; for example, 26.4% of the salmonellosis cases in 2006 were in the infant through four-year age group. This age group has consistently been the highest risk group for LAC since 1982. When cases occur, District Public Health Nurses should educate parents and teachers in preschools and day care facilities. Emphasis is on the following:

- Washing hands for parents, teachers and preschoolers;
- Proper preparation of foods and formula for this age group; cross contamination is a common risk;
- Proper handling and cooking of uncooked meat, poultry and fish to prevent cross contamination;
- Keeping kitchen and utensils clean and preventing cross contamination;
- Avoiding reptile pets in the home, preschool and child care facilities and;
- Avoiding other pets that may carry *Salmonella*, such as baby chicks or ducklings.

Table 2. Salmonellosis Outbreaks in LAC, 2006

Onset Month	Outbreak Setting	Total # Ill	Culture Positive	Serotype	Suspect Vehicle	Suspect Source
January	Day care	7	6	*S. stanley*	Person-to-person	Probable reptile source with secondary transmission
March	Restaurant	4	4	*S. oranienburg*	Unknown food vehicle	Unknown food source
June	Banquet hall	20	3	*S. typhimurium*	Chicken skewers	Chicken
July	Staff party at bakery	5	5	*S. heidelberg*	Milkshake	Raw shell egg
August	Assisted living facility	2	2	*S. agona*	Unknown	Probable secondary transmission
September	Restaurant	3	2	*S. typhimurium var copenhagen*	Unknown food vehicle	Unknown food source
October	Health facility	2	2	*S. hiduddify*	Unknown	Probable reptile source with secondary transmission
October	Skilled nursing facility	2	2	*S. thompson*	Unknown	Probable secondary transmission
December	Banquet hall	7	4	*S. enteritidis*	Potato appetizer	Unknown ingredient
TOTAL		**52**	**30**			

COMMENTS

After a peak in 1994, starting in 1995 through 2000, a steady decline occurred in the LAC rate of salmonellosis. The LAC rate in 2004 had increased, but then adjusted down again in 2005 (Figure 2). The rate has again increased to a rate similar to 2004. Continued surveillance is necessary to determine trends.

Travel was noted as a risk factor for 16.8% of cases (n=204); 33% traveled domestically. Of those who traveled outside of the United States, 57% (n=77) traveled to Mexico. Exposure to a reptile was reported as a risk factor for 8.6% (n=104) of cases.

There were nine salmonellosis outbreaks during 2006 compared to four identified in 2005. Two outbreaks were serotype Typhimurium or a variation of that serotype, the others involved multiple serotypes (Table 2). Outbreak-related cases (both confirmed and presumptive) made up 4.3% of total cases in 2006 compared to 3.5% of total cases in 2005. This year *Salmonella enteritidis*, the predominant serotype for 2006, was found to be the cause for only one outbreak with a total of seven cases. Three of the nine salmonellosis outbreak investigations cited restaurant or catered food as a source. One investigation cited a drink made with raw shell eggs as a source for a group of employees at a bakery. The use of PFGE and comparison of PFGE patterns with other laboratories through PulseNet, the national molecular subtyping network for foodborne disease, continues to help identify potentially related clusters within LAC.

Salmonellosis was reported as a contributing cause of death in eight people, all of whom had underlying health problems such as cancer, immune deficiency, chronic tuberculosis, and chronic liver disease. Ages of these individuals ranged from 1 to 84 years.

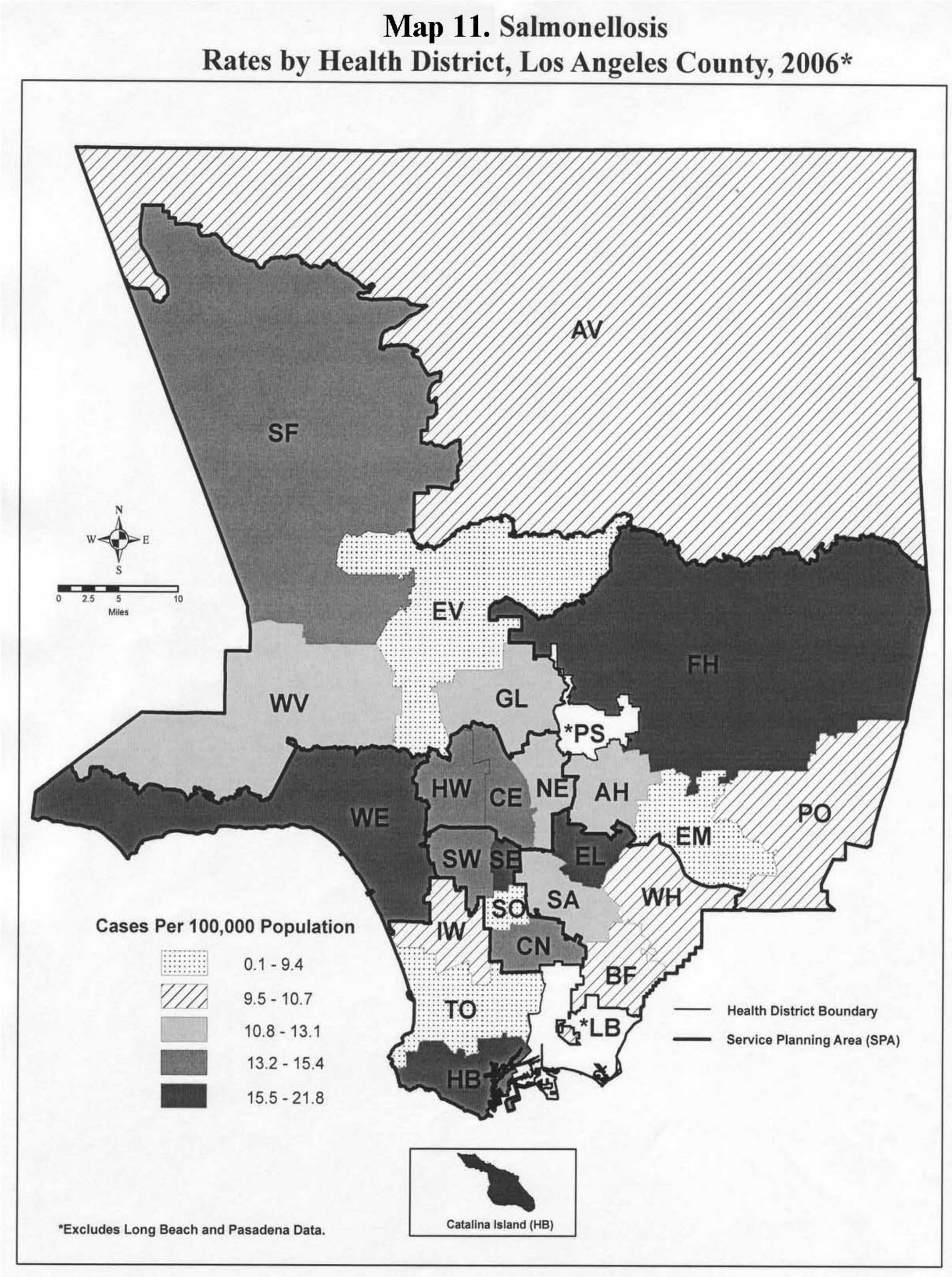
ADDITIONAL RESOURCES

General information about salmonellosis is available at:
www.cdc.gov/ncidod/dbmd/diseaseinfo/salmonellosis_g.htm

General information and reporting information about this and foodborne diseases in LAC is available at:
www.lapublichealth.org/acd/food.htm

CDC. Reptile-associated salmonellosis--selected states 1998-2002. MMWR 2003; 52(49):1206-1209.

CDC. Salmonellosis associated with pet turtles--Wisconsin and Wyoming, 2004. MMWR 2005; 54(9):223-226.



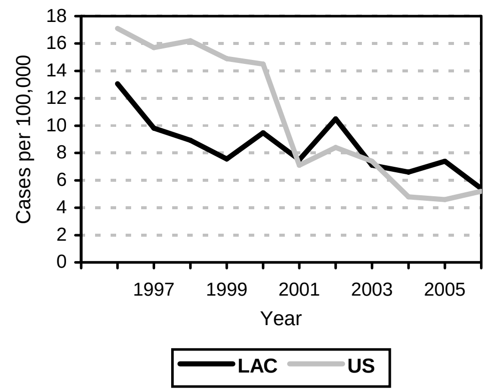
Map 11. Salmonellosis
Rates by Health District, Los Angeles County, 2006*
AV
SF
EV
FH
WV
GL
*PS
HW
CE
NE
AH
PO
WE
EM
SW
SE
EL
SO
SA
WH
IW
CN
BF
TO
*LB
HB
Cases Per 100,000 Population
0.1 - 9.4
9.5 - 10.7
10.8 - 13.1
13.2 - 15.4
15.5 - 21.8
Health District Boundary
Service Planning Area (SPA)
N
W
E
S
0
2.5
5
10
Miles
Catalina Island (HB)
*Excludes Long Beach and Pasadena Data.

SHIGELLOSIS

CRUDE DATA	
Number of Cases	524
Annual Incidence[a]	
LA County	5.4
California	5.18[b]
United States	5.23[b]
Age at Diagnosis	
Mean	23.3
Median	18
Range	<1– 98

[a] Cases per 100,000 population.
[b] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

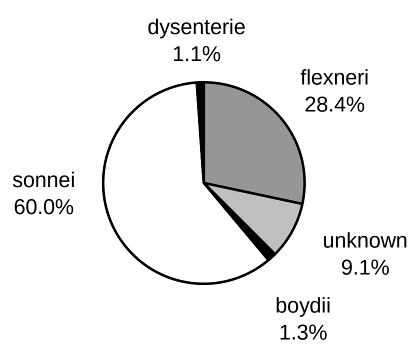
Figure 1
Shigellosis
Incidence Rates by Year of Onset
LAC and US, 1996–2006



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). Incubation period is 1-3 days. Human are the definitive host; transmission occurs when individuals fail to thoroughly wash their hands after defecation and spread infective particles to others, either directly by physical contact, including sexual behaviors, or indirectly by contaminating food. Infection may occur with ingestion of as few as 10 organisms. Common symptoms include diarrhea, fever, nausea, vomiting, and tenesmus. Stool may contain blood or mucous. In general, the elderly, the immunocompromised, and the malnourished are more susceptible to severe disease outcomes.

Figure 2
Shigellosis
Percent by Serogroup
LAC, 2006
(N=524)



DISEASE ABSTRACT

- There was a 35.5% decrease in reported cases in 2006.
- Two shigellosis-associated outbreaks were investigated in 2006.

STRATIFIED DATA

Trends: There was a 35.5% decrease in the number of cases during 2006. This is lowest rate in over twenty years. The LAC rate had been decreasing since a peak in 2002 (Figure 1), before peaking again in 2005. Although the 2006 rate may be an adjustment from the 2005 increase, continued surveillance is needed to identify an emerging trend.

Serotypes: In 2006, *S. flexneri* (n=149; 28.4%) represented a larger percentage than 2005 (n=122; 17.2%). *S. sonnei* remains the dominant serotype (n=315; 60%). Other serotypes identified during 2006 include: *S. boydii* (n=7) and *S. dysenterie* (n=6) (Figure 2).

Seasonality: In 2006, incidence peaked in August and stayed at or below the five-year average through the entire year (Figure 3). There were nine family clusters during the month of August. The rate of travel related cases that occurred from July through September decreased to 48% as compared to 60% in 2005.

Age: Children aged 1–4 years (20.3 per 100,000) and 5-14 (9.1 per 100,000) again had the highest rates; however, these rates were lower than the previous five-year average. The rate for children aged 1-4 years was significantly higher than all other age groups. Adults 65 years and older were the only age group to have a rate higher than the five-year average (Figure 4). This rate was still significantly lower than the county average.

Race/Ethnicity: During 2006, Latinos aged 1–4 years again had the highest age-adjusted rate (Figure 5). For the fourth year, Latino infants and children aged 5–14 had higher age adjusted rates compared to other race/ethnicities. This year, Latinos aged 65 years and older also had higher age-adjusted rates compared to other race/ethnicities. Overcrowding and living with extended family members in addition to the higher overall rate in Latinos may be possible causes. Blacks adults aged 45-55 years, had a higher rate than other ethnicity. All but one case among Latinos were male; of these male cases one self-reported as MSM and the others refused to disclose their sexual orientation.

Sex: The male-to-female rate ratio was 1.1:1. Men are still the preponderant group as reflected in the 2006 ratio.

Location: The rates for SPA 6 (10.2 per 100,000) and SPA 4 (8.2 per 100,000) were significantly higher than the county average (5.45 per 100,000). The increase in SPA 6 is consistent with previous years and may be due to changing demographics in that location. The two outbreaks involved cases from SPAs 3, 4, 5, and 6. The majority of MSM cases (66%) were seen in SPA 4.

Figure 3
Shigellosis
Cases by Month of Onset
LAC, 2006

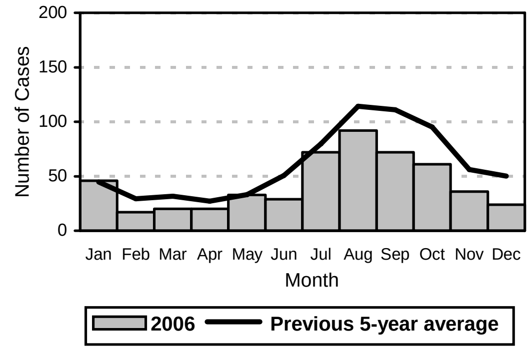


Figure 4
Shigellosis
Incidence Rates by Age Group
LAC, 2006

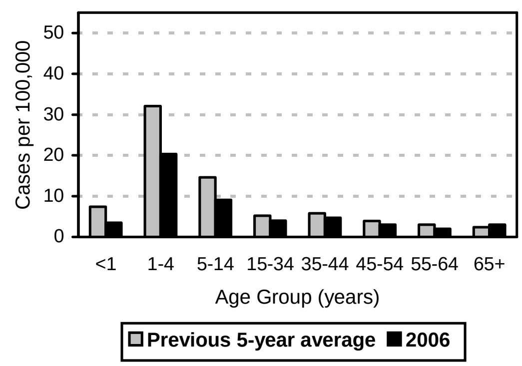
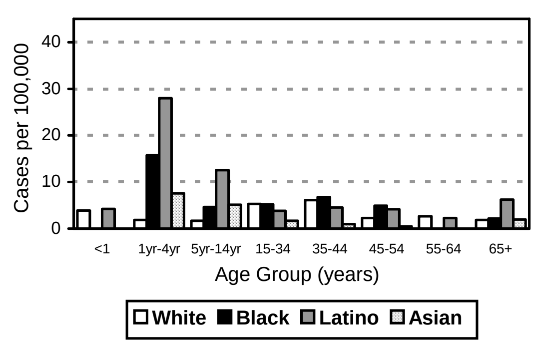


Figure 5
Shigellosis Incidence Rates
by Age Group and Race
LAC, 2006 (N=524)



Severity of Illness: Fifteen percent of shigellosis cases (n=79) were hospitalized for at least two days. There were two shigellosis-associated deaths reported; both cases were immunocompromised.

Risk Factors: Exposure to a case inside or outside the household (15%) and foreign travel (15%) were the most commonly reported potential sources of infection. The majority of foreign travel–associated illness (50%) involved visiting Mexico. Two of the seven *S.boydii* cases reported travel to Africa and India. Three of the six *S. dysenterie* traveled to India, Mexico, and Asia during the incubation period. One *S. dysenterie* case was found during contact follow-up of a typhoid case. In 2006, five percent of cases were in MSM compared to four percent in 2005.

PREVENTION

Careful hand washing is vital in preventing this disease. Young children or anyone with uncertain hygiene practices should be monitored to promote compliance. Hand washing is especially important when out in crowded areas such as amusement parks or shopping malls. Children should not be allowed to swim or wade while ill with diarrhea; ill children (exhibiting symptoms) in diapers should never be allowed in public swimming areas. Swimming or wading in areas not designated for such activities should be avoided, especially in areas where there are no toileting or hand washing facilities. In LAC, cases and symptomatic contacts in sensitive occupations or situations (e.g., food handling, daycare and healthcare workers) are routinely removed from work or the situation until they have culture negative stool specimens tested in the Public Health Laboratory.

COMMENTS

There were two shigellosis outbreaks investigated in 2006, both laboratory confirmed. One was a community outbreak involving a day care setting and the second was a foodborne outbreak involving a restaurant.

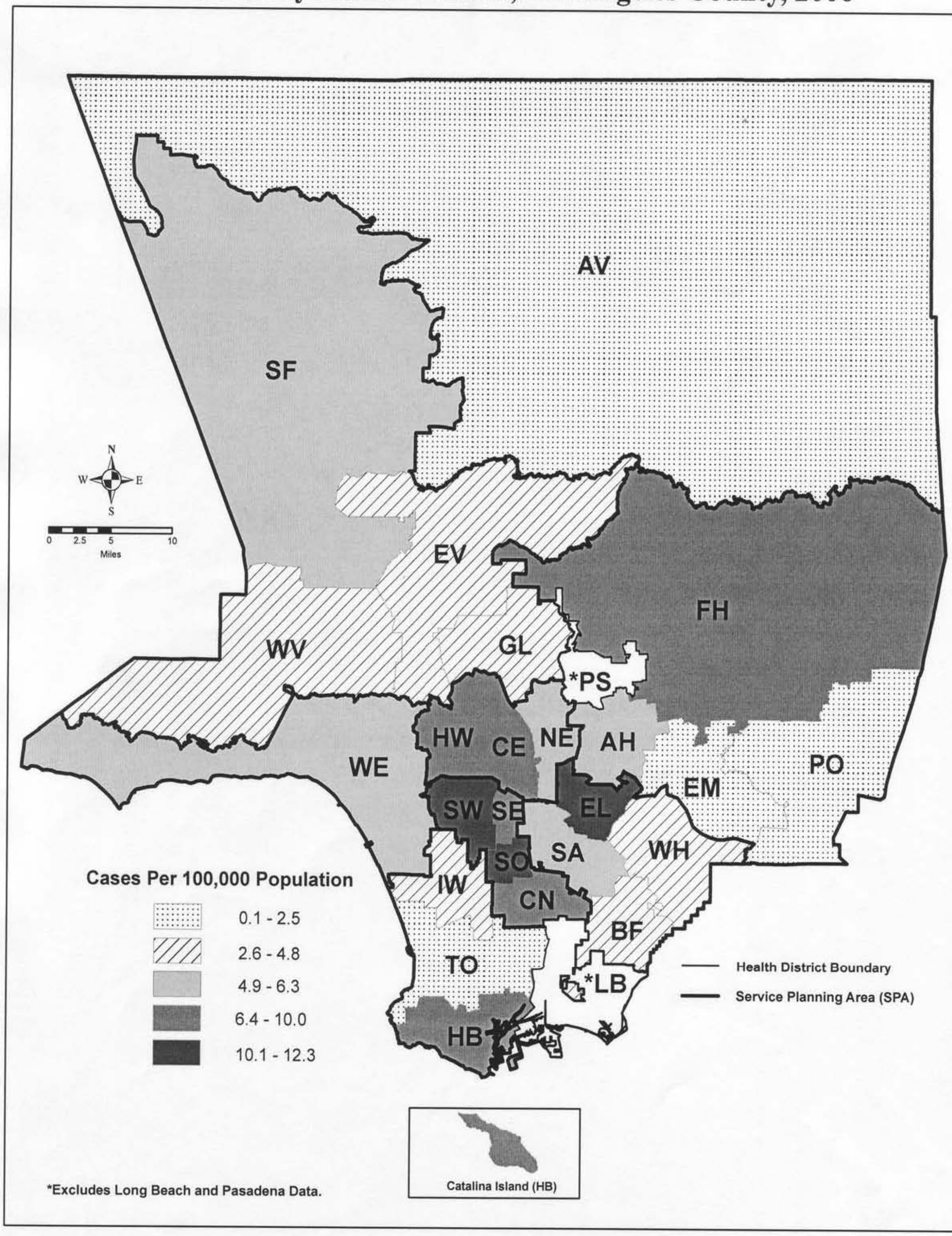
Certain sexual practices—especially those in which there is direct contact with fecal material—are a potential source of infection. There were 28 shigellosis cases reported in MSM in 2006. No links could be established among these cases. *S. flexneri* (55%) was again the predominant serotype in 2003 and 2004 for this risk group; in 2002 the predominant MSM serotype was *S. sonnei* (56%).

ADDITIONAL RESOURCES

General information about shigellosis is available at:
www.cdc.gov/ncidod/dbmd/diseaseinfo/shigellosis_g.htm

General information and reporting information about this and foodborne diseases in LAC is available at: www.lapublichealth.org/acd/food.htm

Map 12. Shigellosis
Rates by Health District, Los Angeles County, 2006*

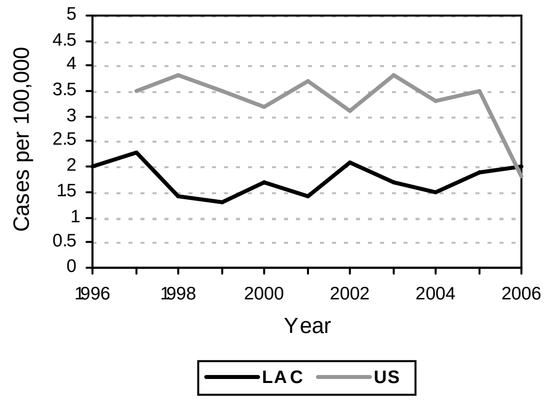


INVASIVE GROUP A STREPTOCOCCUS (IGAS)

CRUDE DATA	
Number of Cases	197
Annual Incidence[a]	
LA County	2.0
California	---[c]
United States[b]	1.82[d]
Age at Diagnosis	
Mean	49
Median	51
Range	1–96 years

[a] Cases per 100,000 population.
[b] National projection of IGAS incidence from Active Bacterial Core Surveillance areas data, 2005 [1]. Data available beginning in 1997.
[c] Not notifiable.
[d] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

Figure 1
IGAS
Incidence Rates by Year of Onset
LAC and US[b], 1996–2006



DESCRIPTION

Invasive Group A Streptococcal (IGAS) disease is caused by the group A beta-hemolytic *Streptococcus pyogenes* bacterium. Transmission is by direct or, rarely, indirect contact. Illness manifests as various clinical syndromes including bacteremia without focus, sepsis, cutaneous wound or deep soft-tissue infection, septic arthritis, and pneumonia. It is the most frequent cause of necrotizing fasciitis, commonly known as “flesh eating bacteria.” IGAS occurs in all age groups but more frequently among the very old. Infection can result in severe illness, including death.

For surveillance purposes in LAC, 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 (CSTE) case definitions for this syndrome; and as NF if the diagnosis was made by the treating physician.

S. pyogenes more commonly causes non-invasive 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.

DISEASE ABSTRACT

- STSS clinical presentation and case fatality rate has increased compared to previous years.
- No clusters or outbreaks were reported.

STRATIFIED DATA

Trends: The incidence rate of reported IGAS was 2.0 per 100,000 (N=197) during 2006, similar to 2005 where 1.9 cases per 100,000 (N=179) were reported (Figure 1).

Seasonality: Although cases were observed throughout the year, a winter/spring seasonality commonly associated with streptococcal pharyngitis was observed as the number of cases increased during the spring and winter months, peaking in April (Figure 2).

Age: The age of cases ranged from 1 to 96 years with a mean of 49 years and median of 51 years. In all age groups the rate of cases in 2006 was higher than the previous 5-year average, with the exception of the less than one year age group, where no cases were reported (4 to 10 reported cases in previous years). The highest rate of cases occurred in those aged 65 years and older (Figure 3).

Gender: Similar to 2005, the male-to-female ratio remained at 2:1 in 2006. In previous years the distribution was nearly equal.

Race/Ethnicity: Race/ethnicity was known for 81% of cases. There has been an increase in the percentage of white cases and a decrease in Latino cases. Similar to 2005, blacks had the highest reported incidence at 2.7 per 100,000 (data not shown).

Location: The incidence rate was highest in SPA 5 (3.3 cases per 100,000) compared to LAC overall (2.0 cases per 100,000). Incidence for SPAs 2, 4, and 8 were slightly higher than LAC overall, while SPAs 3 and 7 had lower rates (Figure 4). However, stratification of cases by SPA produced small numbers and unstable incidence rates for SPAs 1 and 7.

Clinical Presentation: IGAS cases presented most often with cellulitis and bacteremia (Table 1). STSS increased from 5 cases in 2005 (3%) to 18 cases in 2006 (10%) (Figure 5). However, necrotizing fasciitis and pneumonia decreased since 2005 (data not shown). Other syndromes reported include osteomyelitis (5%), septic arthritis (5%), and meningitis (2%). Clinical presentation data was available for 90% of cases.

The case fatality rate has increased from 9% in 2005 to 14% in 2006. This rate is equivalent to the national estimate [1].

Risk Factors: Nearly one third of IGAS cases reported no risk factors (30%). Diabetes was reported more than any other risk factor (24%), followed by history of blunt trauma (15%), alcohol abuse (14%), chronic heart disease (13%), and malignancy (13%). Alcohol abuse and history of blunt trauma were more common in younger cases less than 50 years while diabetes, chronic heart disease, and malignancy were more prevalent in cases older than 50 years (data not shown). Risk factor information was collected for 81% of cases.

Figure 2
IGAS
Cases by Month of Onset
LAC, 2006

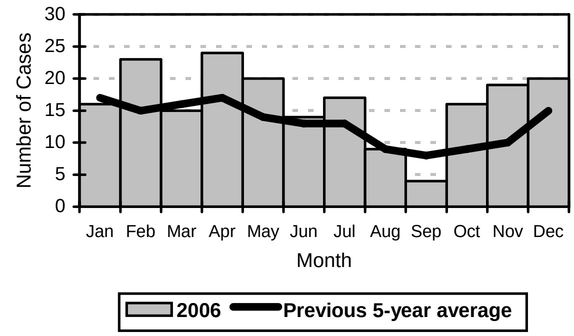


Figure 3
IGAS Incidence Rates by Age Group
LAC, 2006 (n=196)

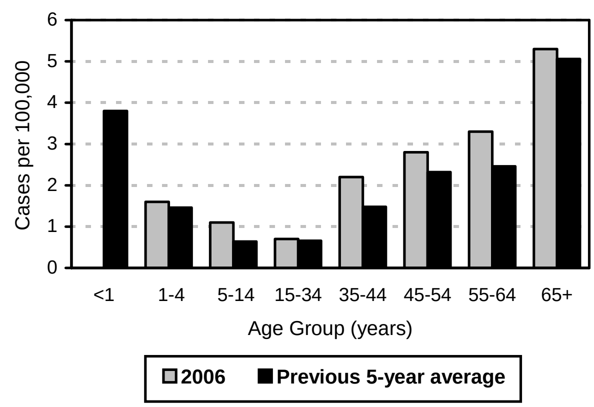
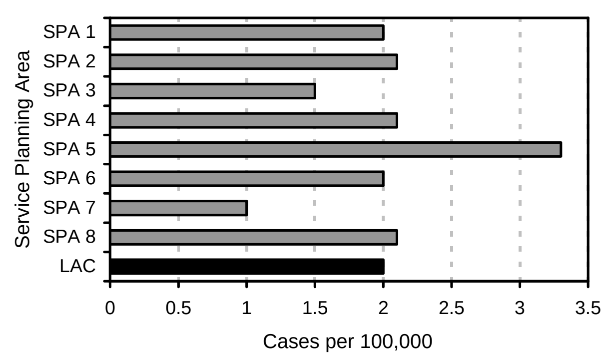


Figure 4
IGAS Rates by Service Planning Area
LAC, 2006 (n=185)



COMMENTS

Although the number of cases increased from 2005, the incidence remained the same at approximately 2 cases per 100,000. However, certain demographic groups in Los Angeles County were at greater risk of infection, including persons aged 65 years and older, blacks, and males. In addition, residents of SPA 5 continued to have the greatest incidence of IGAS disease compared to the rest of the county. It is unknown if this was due to reporting bias or if SPA 5 residents were at increased risk for IGAS infection.

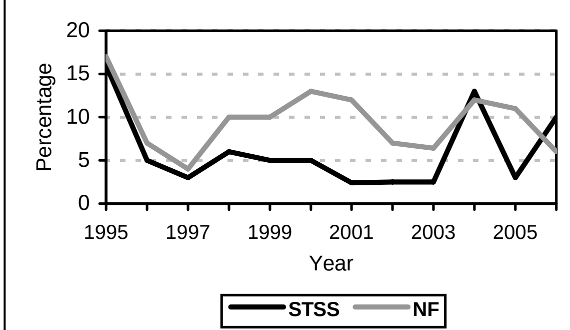
Table 1. Frequency and Percentage of IGAS Clinical Syndromes, LAC, 2006

Syndrome	Number	Percent*
Cellulitis	63	35
Bacteremia (without focus)	43	24
STSS	18	10
Non-Surgical Wound Infection	18	10
Pneumonia	16	9
Necrotizing Fasciitis	11	6
Other	50	28

*Overlapping syndromes will total over 100%.

The number of STSS cases in 2006 more than tripled from 2005 (18 vs. 5), which most likely accounted for the increase in case fatality. Of the 18 STSS cases in 2006, the outcome was known for 16 cases (89%). Of these cases, 10 were fatal (63%). In the past ten years, with the exception of 2004, the number of STSS cases ranged from three to eight (2-6%). In 2004, there were 17 STSS cases and the overall case fatality was 26% (73% among STSS cases). Interestingly, the majority of STSS cases in 2006 were male (83%) compared to 2004 where the majority were female (65%). The rise in STSS and case fatality in 2004 had been attributed possibly to changes in the reporting of IGAS during that year. However, as reporting methods have not changed and clinical presentation was known for approximately 90% of the cases each year from 2004 to 2006, the pattern of STSS and case fatality in 2004 and 2006 suggests not only that the increases were real trends but also that IGAS case fatality is strongly affected by STSS incidence.

Figure 5
IGAS Clinical Presentation of STSS and NF
LAC, 1995–2006



Although IGAS disease is not a mandated reportable disease in California, LAC DPH has required laboratories, hospitals, and healthcare providers to report IGAS disease since 1993. Surveillance has been predominately passive and information pertaining to patient demographics, clinical presentation, intervention, and outcome was often incomplete in the past. Complete IGAS reporting requires active case follow-up, particularly for STSS and NF as the classification of these syndromes requires more intensive review. In 2002, a new IGAS history form including a specific section for STSS reporting was developed and distributed to infection control professionals. Increased information about IGAS and its various clinical syndromes has been systematically collected since that time with increasing success.

ADDITIONAL RESOURCES

For more information about IGAS visit:

- www.cdc.gov/ncidod/dbmd/diseaseinfo/groupastreptococcal_g.htm
- National Institutes of Health – www.niaid.nih.gov/factsheets/strep.htm

For specific information about risk factors for IGAS in Los Angeles County 2004-2006 visit:

- Hageman L. Risk factors for invasive group A streptococcal disease. The Public's Health 2006; 6(9):8-9. Available at: www.lapublichealth.org/media/docs/TPH_NovDec_2006v4.pdf

- Bancroft EB, Lindsey H. Risk factors for invasive group A streptococcal disease in Los Angeles County, 2004-2006. Acute Communicable Disease Control Special Studies Report 2006:81-84. Available at: http://lapublichealth.org/acd/reports/spclrpts/spcrpt06/spcl06[1].new.pdf

IGAS Publications:

- Bancroft EB, Hageman L. Risk factors for invasive group A streptococcal disease in Los Angeles County, 2004-2006. Acute Communicable Disease Control Special Studies Report 2006:81-84. Available at: http://lapublichealth.org/acd/reports/spclrpts/spcrpt06/spcl06[1].new.pdf
- Prevention of invasive group A streptococcal disease among household contacts of case patients and among postpartum and postsurgical patients: recommendations from the Centers for Disease Control and Prevention. Clin Infect Dis 2002; 35(8):950-959.
- O'Brien KL, Beall B, Barret NL, et al. Epidemiology of invasive group A streptococcal disease in the United States, 1995-1999. Clin Infect Dis 2002; 35(3):268-276.
- American Academy of Pediatrics. Committee on Infectious Diseases. Severe invasive group A streptococcal infections: a subject review. Pediatrics 1998; 101(1):136-140.
- Kaul R, McGeer A, Low DE, Green K, Schwartz B. Population-based surveillance for group A streptococcal necrotizing fasciitis: clinical features, prognostic indicators, and microbiologic analysis of seventy-seven cases. Am J Med 1997; 103(1):18-24.

REFERENCE

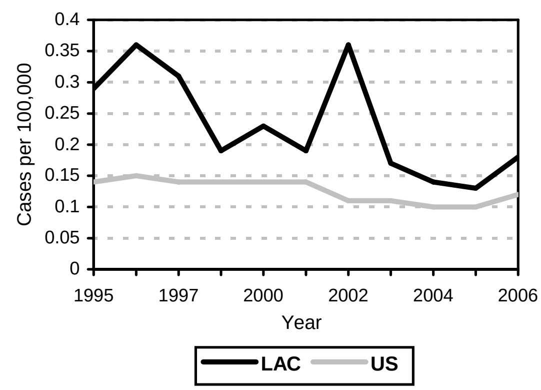
1. Active Bacterial Core Surveillance Reports from 1997 to 2005 from the Centers for Disease Control and Prevention's Division of Bacterial and Mycotic Diseases. Report available at: www.cdc.gov/ncidod/dbmd/abcs/survreports.htm

TYPHOID FEVER, ACUTE

CRUDE DATA	
Number of Cases	17
Annual Incidence[a]	
LA County	0.18[b]
California	0.21[c]
United States	0.12[c]
Age at Diagnosis	
Mean	18.70
Median	20.0
Range	1-48

[a] Cases per 100,000 population.
[b] Rates based on less than 19 observations are unreliable.
[c] Calculated from 2007 Summary of notifiable diseases issue of MMWR (56:853-863).

Figure 1
Acute Typhoid Fever
Incidence Rates by Year of Onset
LAC* and US, 1996–2006

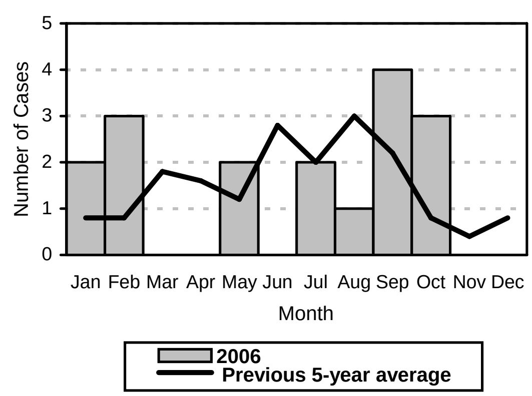


*Rates based on less than 20 observations are unreliable.

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 insidious onset of 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*. Vaccine is available to those at high risk or travelers.

Figure 2
Acute Typhoid Fever
Cases by Month of Onset
LAC, 2006



DISEASE ABSTRACT

- Travel was the most common risk factor identified in LAC; 76% of cases reported travel to typhoid endemic countries. One case recently immigration and one case visited from endemic countries.
- Fifty-eight percent of cases were Asian in 2006.

STRATIFIED DATA

Trends: The yearly incident has decreased after a peak in 2002. However, there were 41% more cases in 2006 compared to 2005.

Seasonality: In 2006, the number of cases peaked in September (Figure 2); however, no cases seemed to coincide with the winter holidays. Typhoid cases occur sporadically throughout the year and are not necessarily associated with traditional travel periods.

Age: In 2005, 75% of acute cases were in adults consistent with the five-year average (Figure 3). The age group of 15-34 years has consistently represented the highest percentage of cases in the past five years.

Sex: The male-to-female ratio was 1:1.1.

Race/Ethnicity: In 2006, acute typhoid cases occurred in Asians and Latinos as seen in 2005. There were no cases in Blacks or White (Figure 4). In 2006, Asian cases increased compared to the five-year average. Continued surveillance is needed to identify emerging trends. I

Location: In 2006, SPA 3 had the majority of cases (41%). This may be due to the rise in Asian population in SPA 3. SPA 2 and 7 had three cases each (18%). SPA 6 and 8 had one case each (6%). SPA 5 had two cases (12%) (data not shown).

Figure 3
Acute Typhoid Fever
by Age Group
LAC, 2006

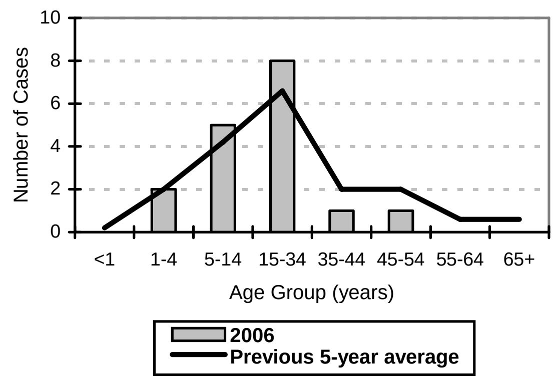
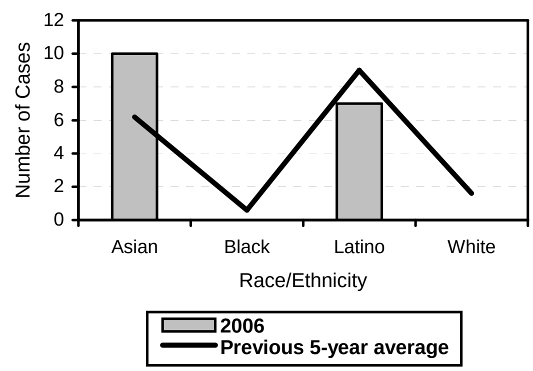


Figure 4
Acute Typhoid Fever
by Race/Ethnicity
LAC, 2006



PREVENTION

Handwashing after using the toilet, before preparing or serving food, and before and after caring for others is important in preventing the spread of typhoid. When traveling to locations where sanitary practices are uncertain, foods should be thoroughly cooked and served hot; bottled water should be used for drinking as well as for brushing teeth and making ice. Vaccination should be considered when traveling in areas of high endemicity. LAC tests household contacts of confirmed cases for *S. typhi* to identify any previously undiagnosed carriers or cases.

COMMENTS

The majority of cases (n=11, 65%) traveled to endemic areas outside the US; Mexico, India, Bangladesh, Indonesia, Philippines and Cambodia were reported travel destinations. One case was infected by previously undiagnosed carrier in the household.

ADDITIONAL RESOURCES

General information about typhoid fever available from CDC at:
www.cdc.gov/ncidod/dbmd/diseaseinfo/typhoidfever_g.htm

Traveler's health information is available at: wwwn.cdc.gov/travel/yellowBookCh4-Typhoid.aspx

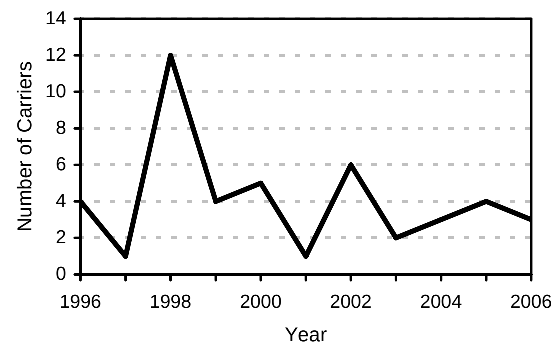
General information and reporting information about this and other diseases in LAC is available at:
www.lapublichealth.org/acd/food.htm

TYPHOID FEVER, CARRIER

CRUDE DATA	
Number of New Carriers	3
Total Number of Carriers	17
Annual Incidence[a]	
LA County	N/A[b]
United States	N/A
Age at Diagnosis	
Mean	40 years
Range	5-61 years

[a] Cases per 100,000 population.
[b] Rates based on less than 19 observations are unreliable.

Figure 1
Typhoid Fever Carriers
by Year of Detection
LAC, 1996–2006



DESCRIPTION

The chronic typhoid carrier state can occur following symptomatic or subclinical infections of *Salmonella typhi*. Among untreated cases, 10% will shed bacteria for three months after initial onset of symptoms and 2-5% will become chronic carriers. The chronic carrier state occurs most commonly among middle-aged women.

DISEASE ABSTRACT

- There were three new carriers identified in 2006.
- During 2006, three carriers were closed as lost to follow-up, leaving a total of 17 carriers under case management in LAC at the end of 2006.

COMMENTS

All new carriers were foreign born; two were male and one was female. Two previously unknown carriers were identified while testing household contacts to a new acute typhoid case, all in the same household. The other carrier was identified when presented to the hospital with fevers and tested positive for Campylobacter; subsequently the patient was found to have *S. typhi* infection.

Upon identification, each new carrier is added to the typhoid carrier registry. All carriers are visited semi-annually by a public health nurse to assess and emphasize compliance with a signed typhoid carrier agreement. Per state code, carriers are to remain under the supervision of the local health officer until cleared. Conditions for release from supervision are also mandated by state code. An approved public health laboratory must test the cultures for the purpose of release.

ADDITIONAL RESOURCES

Disease information is available from CDC at:
www.cdc.gov/ncidod/dbmd/diseaseinfo/typhoidfever_g.htm

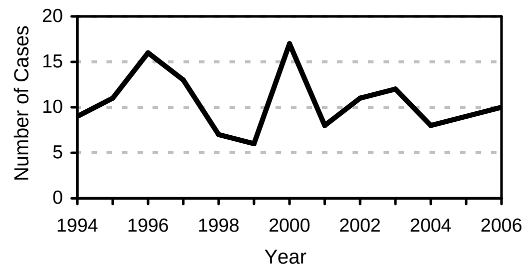
General information and reporting information about this and other diseases in LAC is available at:
www.lapublichealth.org/acd/food.htm

TYPHUS FEVER

CRUDE DATA	
Number of Cases	10
Annual Incidence[a]	
LA County	0.09[b]
United States	N/A
Age at Onset	
Mean	43
Median	40.5
Range	13–73 years

[a] Cases per 100,000 population.
[b] Rates based on less than 20 observations are unreliable.

Figure 1
Murine Typhus
Cases by Year of Onset
LAC, 1994–2006



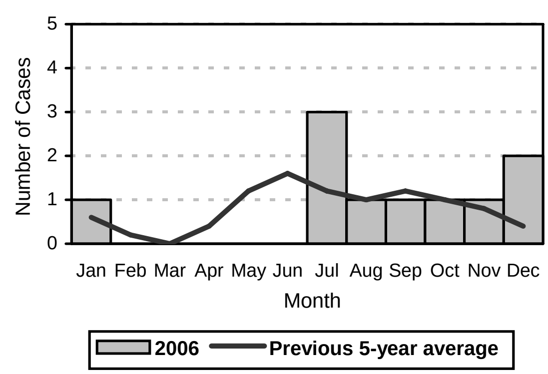
DESCRIPTION

Typhus fever (murine typhus, endemic typhus) is caused by the bacteria, *Rickettsia typhi* and *R. felis*, and transmitted through the bite or contact with feces of an infected flea. Reservoir animals are predominantly rats and opossums that live in areas with heavy foliage. In Los Angeles County (LAC), most reported cases of typhus occur in residents of the foothills of central 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 increases with age. The disease is typically mild in young children. Typhus infection is not vaccine preventable, but can be treated with antibiotics.

DISEASE ABSTRACT

- The number of cases reported in 2006 (n=10) falls within range of the number reported annually in previous years. No outbreaks occurred.
- Increased reports of typhus in unusual localities as well as those occurring in the Long Beach and Orange County jurisdictions indicate the endemic areas of typhus may be shifting.

Figure 2
Murine Typhus
Cases by Month of Onset
Los Angeles County, 2006



STRATIFIED DATA

Trends: The number of cases reported in 2006 (n=10) increased in compared to the 9 cases reported in 2005. However, the number of 2006 case report fall within the range of 8–12 cases reported annually in the previous five years (Figure 2).

Seasonality: Typhus fever is a seasonal disease and most cases will be seen in the summer and fall. Seasonality is mostly likely related to chance exposure to fleas relating to time spent outdoors with animal reservoirs of infection and their infected fleas. In 2006, most cases occurred during these times of the year; however, cases were also uncharacteristically reported throughout the fall and into December (Figure 2).

Age: In 2006, the mean and median ages were 43 and 40.5 years, respectively. Ages of cases ranged from 13 to 73 years; most cases occurred in those under 65 years (n=8, 80%) (data not shown).

Sex: There were at least twice as many cases reported in males as females. The male-to-female case ratio was 2.3:1. The gender distribution in previous years has been roughly equivalent.

Race/Ethnicity: Most cases were of white race/ethnicity (n=6, 60%). Three cases (30%) occurred in Latinos and one (10%) in an Asian (data not shown).

Location: Most cases (n=7, 70%) were residents of, or reported substantial recreational activity in, health districts around the foothills of central LAC or in the metropolitan area, localities which have historically been endemic for typhus fever. Mammalian reservoirs such as rats, opossum, and cats from these areas have been serologically positive for *R. typhus and R. felis*. The remaining three cases (30%) resided in the West, West Valley, and Bellflower health districts, and did not report any activity in the endemic localities.

Transmission and Risk Factors: Human infection most commonly occurs by introduction of infectious flea fecal matter into the bite site or into adjacent areas that have been abraded by scratching. Only 30% of the cases in 2006 (n=3) reported an exposure to fleas or flea bites within the 2 weeks prior to onset of illness. Of the cases that were not exposed to fleas, almost all reported observing other types of small mammals (e.g., rats, opossums, dogs and cats) on their residential property, and thus may have had exposure to animals that carry fleas. One case worked as a parking attendant in the downtown LA area and reported no exposure to animals or activity in the foothills of central LAC. Typhus infection cannot be transmitted from person to person.

PREVENTION

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

COMMENTS

Though the number of typhus fever cases confirmed in LAC in 2006 has not changed remarkably relative to previous years, the higher proportion of cases appearing in health districts in which typhus is not usually seen has shown that the endemic areas of typhus may be shifting. In addition to cases reported in unusual locations within the county, the public health departments of Long Beach and Orange County have also confirmed cases in their jurisdictions during the latter part of 2006, either for the very first time or the first in many years. However, the increase in reporting and confirmation may reflect increased awareness of endemic typhus due to media attention and alerts issued by these health departments.

When a diagnosis of typhus fever is confirmed by serology, each case is interviewed regarding potential exposures. If possible, field studies of the property where exposure occurred and surrounding areas in the neighborhood are conducted by an environmental health specialist. In addition, local residents are contacted and provided with education about typhus and prevention of the disease by controlling fleas and eliminating harborage for potentially typhus-infected animals that carry fleas.

The nonspecific clinical presentation and the lack of a definitive test during the acute phase of the illness make the early diagnosis of typhus fever difficult. Thus, diagnosis of typhus fever depends on the clinical acumen of the treating physician and often requires acute and convalescent serology, and so is frequently confirmed after the patient has recovered. Reporting of typhus or suspect typhus cases can help identify areas in LAC that may require monitoring for the presence of disease in the animal populations and the institution of control measures.

ADDITIONAL RESOURCES

General information about typhus fever is available from the ACDC website at:
www.lapublichealth.org/acd/vectormurine.htm

Publications:
Azad AF, Radulovic S, Higgins JA, Noden BH, Troyer JM. Flea-borne rickettsioses: ecologic considerations. Emerg Infect Dis 1997; 3(3):319–327.

Sorvillo FJ, Gondo B, Emmons R, et al. A suburban focus of endemic typhus in Los Angeles County: association with seropositive domestic cats and opossums. Am J Trop Med Hyg 1993; 48(2):269–273.

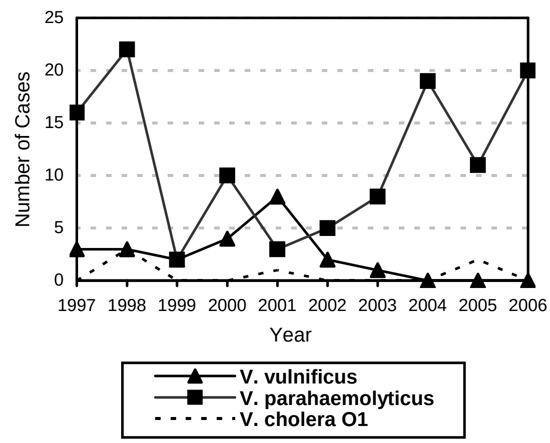
Williams SG, Sacci JB, Schriefer ME, et al. Typhus and typhuslike rickettsiae associated with opossums and their fleas in Los Angeles County, California. J Clin Microbiol 1992; 30(7):1758–1762.

VIBRIOSIS

CRUDE DATA	
Number of Cases	24
Annual Incidence[a]	
LA County	0.19
United States	N/A
Age at Diagnosis	
Mean	46
Median	43
Range	14–86 years

[a] Cases per 100,000 population.

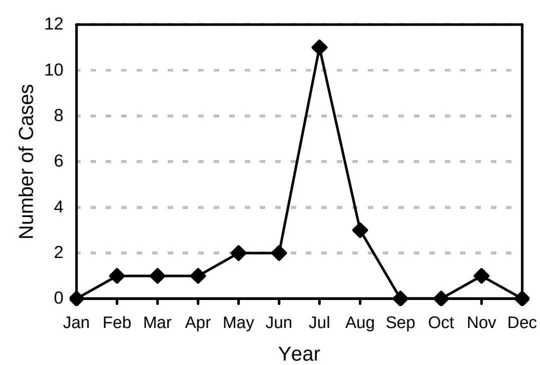
Figure 1
Selected Vibrio Species
Cases by Year of Onset
LAC, 1997–2006



DESCRIPTION

The genus Vibrio consists of Gram-negative, curved, motile rods, and contains about a dozen species known to cause human illness. Transmission is most often through ingestion via a foodborne route, but also from contact between broken skin and contaminated water. Presenting symptoms vary by species and mode of transmission. The Vibrio species of greatest public health importance in the US are: *V. vulnificus* which causes a primary septicemia and is often associated with oysters harvested in the Gulf of Mexico, and *V. parahaemolyticus*, which presents as gastrointestinal illness. Cholera, a potentially fatal diarrheal disease caused by *V. cholerae* serotypes O1 and O139, is rarely imported into the US.

Figure 2
Vibriosis
Cases by Month of Onset
LAC, 2006



DISEASE ABSTRACT

- Twenty-four cases of vibriosis were reported in 2006, an increase from 14 cases reported in 2005.
- No fatal cases of vibriosis were reported in 2006.
- No cases of *V. vulnificus* or toxigenic *V. cholerae* O1/O139 were reported in 2006. There were two cases of *V. alginolyticus* infections related to surfing injuries and one case of *V. furnissii* infection in a leg wound.

STRATIFIED DATA

Trends: Over the last 10 years, case reports of Vibrio infections peaked in 1998 with 36 cases (7 cases were part of an outbreak). Reported cases of *V. vulnificus* remained zero since 2004, a substantial decline compared to the 10-year peak of eight cases occurring during in 2001 (Figure 1). *V. cholerae* non-O1/non-O139 cases declined from two cases in 2005, down to one case in 2006.

Seasonality: Among reported vibriosis cases with distinct onset dates, the majority (64%, n=16) occurred between June and October (Figure 2). Vibrio infections typically increase during the summer months when ocean temperatures rise, allowing the bacteria to flourish.

Age: Vibrio cases were all adults except for one juvenile who was 14 years old. The average age of cases was 46 years (Table 1).

Sex: Slightly over half of the cases were male (52%, n=13, Table 1).

Race/Ethnicity: Reported cases were most often Non-Latino white (54%, n=14, Table 1), which is consistent with 2005. Latinos historically constituted a more significant proportion of all vibriosis cases.

Severity: For vibriosis cases with distinct onset and resolution dates (n=16), duration of illness averaged 8 days (range 1-43). Five cases required hospitalization.

Table 1. Vibrio Cases by Species, Race, Age and Sex—LAC, 2006

Species	No. of cases	Race (no. of cases)	Mean Age, years (range)	Sex Ratio M:F
V. parahaemolyticus	20	Asian (3), Latino (5), White (12), Black (0)	45 (14-86)	0.81:1
V. cholerae non-O1/O139	1	Latino (1)	67 (67)	0:1
V. alginolyticus	2	White (2)	54.5 (54-55)	2:0
V. furnissii	1	Latino (1)	61 (61)	1:0

Species-specific Risk Factors:

Vibrio parahaemolyticus
Twenty cases of *V. parahaemolyticus* were reported during 2006. All 20 were identified through stool culture. Seventeen reported eating seafood recently, with 12 specifying raw oysters. Of these 12, 11 were linked to contaminated oysters harvested in Puget Sound, WA.

***Vibrio cholerae* non-O1/O139**
One case of non-toxigenic *V. cholerae* gastroenteritis was reported in 2006. It was related to travel to Mexico.

Vibrio alginolyticus
Both *V. alginolyticus* infections were wound infections. The patients had been exposed to seawater via surfing injuries in separate incidents.

COMMENTS

In LAC, risk of Vibrio infection can be prevented or reduced by avoiding eating raw fish and shellfish. In 2006 there were no cases of *V. vulnificus* infection. This continued absence of cases is most likely due to a state-mandated oyster ban that took effect in 2003 banning Gulf Coast Oysters harvested between April 1st and October 31st. Oysters from Gulf Coast waters during warm months pose a higher risk for *V. vulnificus* contamination. Adult men may be more at risk for Vibrio infections because of their tendency to engage in behaviors exposing them to seawater and untreated water (such as surfing or river rafting) or to eat raw or partially cooked seafood, especially oysters.

ADDITIONAL RESOURCES

Mouzin E, Mascola L, Tormey MP, Dassey DE. Prevention of *Vibrio vulnificus* infections. Assessment of regulatory educational strategies. JAMA 1997; 278(7):576–578. Abstract available at: www.jama.ama-assn.org/cgi/content/abstract/278/7/576

Disease information regarding *Vibrio vulnificus* is available from the CDC at:
www.cdc.gov/ncidod/dbmd/diseaseinfo/vibriovulnificus_g.htm

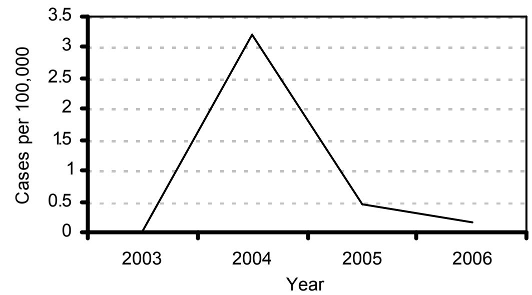
Disease information regarding *Vibrio parahaemolyticus* is available from the CDC at:
www.cdc.gov/ncidod/dbmd/diseaseinfo/vibrioparahaemolyticus_g.htm

WEST NILE VIRUS

CRUDE DATA	
Number of Cases	16
Incidence LAC[a]	
LA County	0.17
California	N/A
United States	N/A
Age at Diagnosis	
Mean	50.9
Median	50.5
Range	28–82 years

[a] Cases per 100,000 population.

Figure 1
West Nile Virus Disease
Incidence Rates by Year of Onset
LAC, 2003-2006



DESCRIPTION

Life Cycle and Epidemiology

West Nile virus (WNV) is a single-stranded RNA virus placed within the family Flaviviridae, genus Flavivirus. Within the genus Flavivirus, WNV has been serologically classified within the Japanese encephalitis (JE) virus antigenic complex, which includes the human pathogens JE, Murray Valley encephalitis, Saint Louis encephalitis (SLE), and Kunjin viruses.

WNV was indigenous to Africa, Asia, Europe, and Australia, and was introduced to North America in 1999, when it was first detected in New York City. The likely origin of the introduced strain was the Middle East, but the mode of introduction remains unknown. Since 1999, human and non-human WNV surveillance data has documented that WNV has extended its range through most of the continental United States as well as to Canada and Mexico.

The life cycle of the virus involves the transmission of the virus between mosquitoes and bird reservoir hosts. Humans are incidentally infected when bitten by an infected mosquito, usually a *Culex* or *Anopheles* species. The incubation period for human infection is 2 to 14 days. Birds, especially corvids such as the North American crow, are the optimal hosts for harboring and replicating the virus. Mosquitoes become infected when they feed on infected birds**,** which may circulate high level of viremia for several days. Infectious mosquitoes carry virus particles in their salivary glands and infect susceptible bird species during blood-meal feeding. Bird reservoirs will sustain an infectious viremia for 1 to 4 days. Additional routes of transmission that have been documented include transplantation of WNV-infected organs, blood transfusions, transplacental (mother-to-child), occupational exposures, and through breast milk.

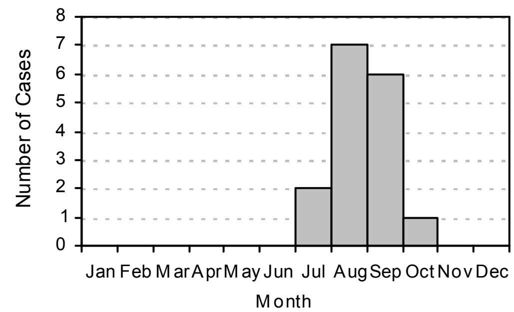
Clinical Infection and Diagnosis

Most persons who become infected with WNV will not develop clinical illness or symptoms. Approximately one in 150 patients will develop more severe illness, manifesting as WNV neuro-invasive disease (NID), and about 20% of persons infected will develop WNV fever with symptoms that include fever, headache, rash, muscle weakness, fatigue, nausea and vomiting, and occasionally lymph node swelling. WNV NID includes encephalitis, meningitis, and acute flaccid paralysis (AFP). WNV-associated encephalitis is commonly associated with the following symptoms: fever, altered mental status, headache, and seizures; WNV encephalitis usually necessitates high levels of specialized medical care. Focal neurologic deficits, including limb paralysis, cranial nerve palsies, Parkinsonian-like tremors, and other movement disorders have been observed. WNV-associated meningitis usually involves fever, headache, and stiff neck, and has a good prognosis.

DISEASE ABSTRACT

- The overall incidence of reported WNV infections in 2006 was 0.17 cases per 100,000 population, far lower than the incidence rates of previous years, when 3.2 per 100,000 and 0.46 per 100,000 were confirmed in 2004 and 2005, respectively (Figure 1).
- There were no case fatalities in 2005 or 2006.
- Meningitis was the most commonly reported clinical condition as it was in 2005, comprising 25% (n=4) of cases. In 2005, meningitis comprised 34.8% of cases (n=15).
- There were few or no cases in children in both 2005 and 2006.
- Most WNV infections occurred in persons residing in San Fernando Valley.

Figure 2
West Nile Virus Disease
Cases by Month of Onset
LAC, 2006 (N=16)



STRATIFIED DATA

Trends: WNV infection, including in asymptomatic blood donors, occurred at an incidence rate of 0.17 per 100,000 population in 2006. Both the total number and incidence of WNV infection decreased dramatically since 2004 when 309 cases were confirmed at an incidence of 3.2 cases per 100,000 population. In 2005, the incidence was 0.46 per 100,000 (n=43) (Figure 1).

Seasonality: Onset of cases occurred July through October and peaked in August (Figure 2). A similar epidemiologic symptom onset curve also occurred in 2005.

Age: The median age was 50.5 years (range: 28–82 years). For age groups ≥35 years, the incidence rates were similar (they ranged 0.2-0.4 cases per 100,000). There was more varied distribution in 2005 where incidence rates ranged from 0.3 cases per 100,000 among children under 10 to 11.6 cases per 100,000 in those greater than 80 years old.

Sex: A higher proportion of male WNV cases were reported than female cases. The incidence rates were 0.25 cases and 0.08 cases per 100,000, respectively.

Race/Ethnicity: Whites had the greatest proportion of reported cases (81%) as well as the highest incidence rates of infection (n=13, 0.45 per 100,000). Latinos accounted for 13% of cases (n=2, 0.04 per 100,000), and only 6% of reported cases occurred among Asian Pacific Islanders (n=1, 0.1 per 100,000). No cases in

Figure 3
West Nile Virus Disease
Incidence Rates by Race/Ethnicity
LAC, 2006

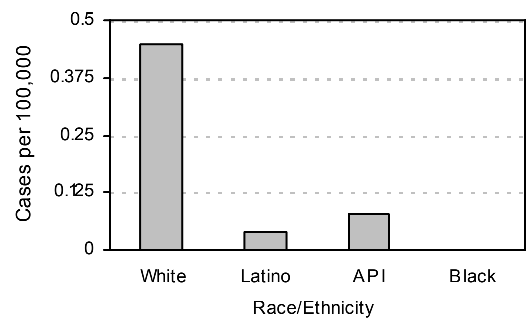
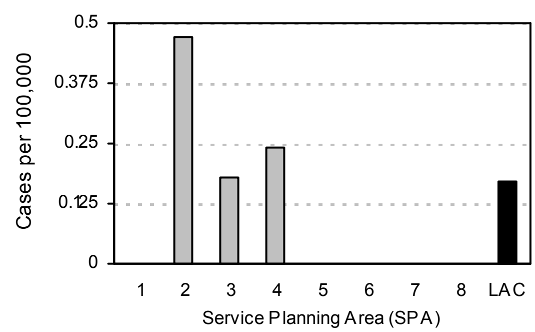


Figure 4
West Nile Virus Disease
Cases by Service Planning Area
LAC, 2006



blacks were reported (Figure 3).

Location: The greatest number of reported WNV cases were reported from SPA 2 (n=10, 0.47 per 100,000). WNV cases occurred in only two other areas: SPAs 3 and 4. WNV was distributed more widely in 2005, though SPA 2 also accounted for most cases.

PREVENTION

Prevention and control of WNV and other arboviral diseases is most effectively accomplished through integrated vector management programs. These programs include surveillance for WNV activity in mosquito vectors, birds, horses, other animals, and humans; and implementation of appropriate mosquito control measures to reduce mosquito populations when necessary. Additionally, when virus activity is detected in an area, residents are alerted and advised to increase measures to reduce contact with mosquitoes. Currently, there is no human vaccine available against WNV but several vaccines are under development. Important preventive measures against WNV include the following:

- Apply insect repellant to exposed skin. A higher percentage of DEET in a repellent will provide longer protection. DEET concentrations higher than 50% do not increase the length of protection.
- When possible, wear long-sleeved shirts and long pants when outdoors 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 which have been registered with the U.S. Environmental Protection Agency (EPA) for use as repellents applied to skin and clothing. EPA registration of repellent active ingredients indicates the materials have been reviewed and approved for efficacy and human safety when applied according to the instructions on the label. Of the active ingredients registered with the EPA, three have demonstrated a higher degree of efficacy in the peer-reviewed, scientific literature. 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

Oil of lemon eucalyptus [p.menthane 3, 8-diol (PMD)], a plant based repellant, is registered with EPA. In two recent scientific publications, when oil of lemon eucalyptus was tested against mosquitoes found in the US it provided protection similar to repellants with low concentrations of DEET.

In 2002, evidence of WNV transmission was shown to occur via the transfer of all blood product components including platelets, packed red blood cells, and plasma. Beginning 2003, blood donors were screened for WNV infection utilizing polymerase chain reaction (PCR) testing. Millions of units of blood were screened for WNV utilizing PCR based technology, testing donor mini-pools. Though asymptomatic donors have been identified as positive for WNV in LAC, no transmission associated with blood products has been reported.

COMMENTS

The first symptomatic WNV case in LAC with associated environmental evidence was documented in 2003. In 2004, an outbreak of 309 WNV infections, including asymptomatic blood donors, with 14 deaths were reported in LAC — the most of any CA jurisdiction. The following years have presented a markedly different picture. In 2005, the county only documented 43 infections and no deaths. The decline continued in 2006, during which only 16 cases and no deaths were reported.

In response to the 2004 WNV outbreak, LAC DPH specifically added WNV infection to its list of reportable diseases by authority of the Health Officer under California Code of Regulations, Title 17, Sections 2503 and 2505. Physicians and laboratories are required to report all positive laboratory findings of WNV to the DPH within one working day. Continued vector surveillance efforts have demonstrated that, despite the decline in incidence in LAC, WNV remains endemic (enzootic) in the LAC and southern CA region. Sustained surveillance of humans, as well as other animals, will be required in the coming years to help guide public health officials in providing targeted health education to communities at particularly high risk.

VECTOR CONTROL

There are five local mosquito and vector control districts within LAC that provide mosquito abatement services to all areas of the county. They carry out mosquito and sentinel chicken surveillance, provide public information, and are critical to mosquito-borne disease control. They include:

- Greater Los Angeles County Vector Control District (GLACVCD)
- San Gabriel Valley Mosquito and Vector Control District (SGVVCD)
- Los Angeles County West Vector Control District (LACWVCD)
- Antelope Valley Mosquito and Vector Control District (AVMVCD)
- Compton Creek Mosquito Abatement District

These five local mosquito and vector control districts work closely with the ACDC to investigate confirmed and presumptive human cases of locally acquired mosquito-borne disease to identify mosquito breeding sites and to put into place appropriate control measures.

ADDITIONAL RESOURCES

- Centers for Disease Control and Prevention: www.cdc.gov/ncidod/dvbid/westnile/index.htm
- California Department of Health Services: www.westnile.ca.gov
- Acute Communicable Disease Control Program, Los Angeles County Public Health: www.lapublichealth.org/acd/index.htm
- Vector Management Environmental Health, Los Angeles County Public Health: www.lapublichealth.org/eh/index.htm
- For additional information on EPA-registered repellants: www.epa.gov/pesticides/factsheets/insectrp.htm

Mosquito and Vector Control District Websites:

- Greater Los Angeles County Vector Control District: www.glacvcd.org
- West Los Angeles Vector Control District: www.lawestvector.org
- San Gabriel Valley Mosquito and Vector Control District: www.sgvmosquito.org
- Antelope Valley Mosquito and Vector Control District: www.avmosquito.org
- Mosquito and Vector Control Association of California: www.mvcac.org

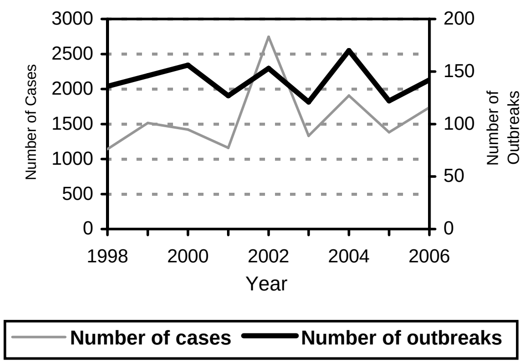
DISEASE OUTBREAK SUMMARIES 2006

COMMUNITY-ACQUIRED DISEASE OUTBREAKS

ABSTRACT

- In 2006, 142 community-acquired disease outbreaks accounted for 1,743 cases of illness (Figure 1).
- Schools were the most common setting of community-acquired outbreaks (46%).
- The number of reported outbreaks (142) surpassed the previous eight-year average of reported outbreaks (141).

Figure 1
Community Acquired Outbreaks
Number of Persons Affected
LAC, 1998–2006

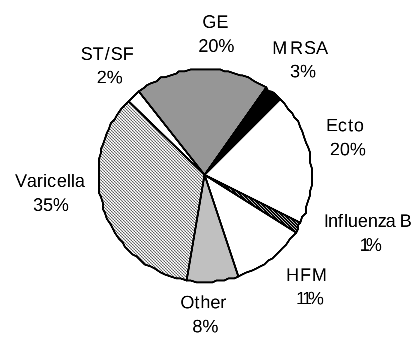


DATA

Disease outbreaks are defined as clusters of illness that occur in a similar time or place, or unusual case numbers above baseline in a specified area. Depending on the nature of the outbreak, investigation responsibility is maintained by either ACDC or Community Health Services with ACDC providing consultation as needed. The outbreaks reported in this section do not include outbreaks associated with food (see Foodborne Outbreaks section) or facilities where medical care is provided (see Healthcare Associated Outbreaks section).

Varicella caused most community-acquired outbreaks in LAC (35%). Gastroenteritis (GE) of various etiologies closely followed by ectoparasites (scabies and pediculosis) were in a near tie for second most common cause of outbreaks, each comprising 20% of all outbreaks (Figure 2, Table 1). Collectively accounting for 75% of all community-acquired outbreaks in 2006, the dominance of these three disease categories is similar to past years (75% in 2005 and 72% in 2004).

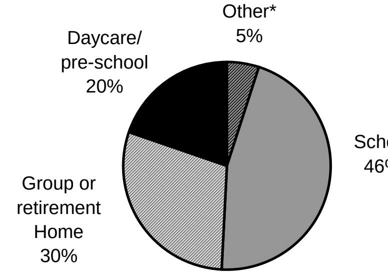
Figure 2
Community-Acquired Outbreaks by
Type of Disease* LAC, 2006 (N=142)



* ST/SF=strep throat/scarlet fever; Ecto=ectoparasites; HFM=Hand, foot &mouth; GE = Gastroentestinal; Other = MRSA, pertussis, ringworm, typhus, unknown resp., viral menigitis.

The agents causing the most cases per outbreak were norovirus (9 outbreaks, mean of 35 cases per outbreak), followed by GE of undetermined cause (16 outbreaks, mean of 15 cases per outbreak). While not laboratory confirmed, the signs and symptoms of these undetermined GE outbreaks were consistent with a norovirus etiology. Important to note in 2006, due to documented increase in county-wide norovirus activity, a reduction in collecting diagnostic viral specimens was instituted. These figures highlight the increased circulation of norovirus and reflect the ease this agent can be transmitted from person to person in community settings (Table 2).

Figure 3
Community Outbreaks by Setting
LAC, 2006 (N=142)

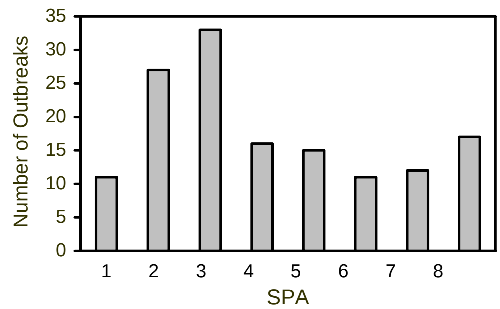


*Other includes: jail (2), vocational work setting (2), restaurant (2) and tattoo parlor (1)

The most common outbreak settings were schools [elementary schools (47), middle schools (13), after-school care (1), high schools (1), and universities (3)] accounting for 46% of all outbreaks. This is similar to 2005 when most outbreaks (60%) were associated with schools settings. Indeed, in most prior report years, the proportion of outbreaks in schools had always been greater than 50%; the prior five year average for schools is 59%. Group and retirement home settings were the second most common site of community-acquired outbreaks reported in 2006, with 30% of the outbreaks. Prior report years had group and retirement home setting consistently lower; the previous five-year-average percentage was 13%. Settings with young children in daycare or pre-school accounted for an additional 20%. (Figure 3).

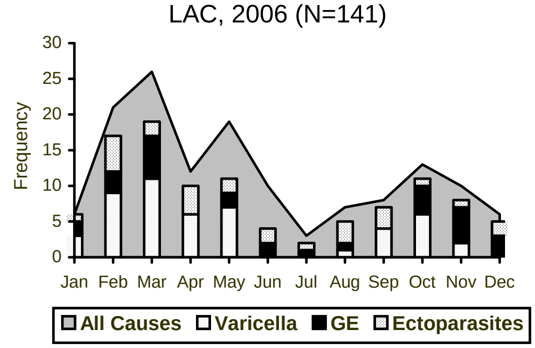
Outbreaks were reported from all 8 SPAs (Figure 4). SPA 3, in the San Gabriel and Pomona Valleys, had the most outbreaks reported in 2006.

Figure 4
Community Outbreaks by SPA
LAC, 2006 (N=142)



The chart of community-acquired outbreaks by onset month (Figure 5) shows a peak in the distribution for March. Varicella outbreaks tended to show a bimodal seasonality with reports occurring during the traditional school year and low numbers during the summer and winter break. GE occurred throughout the year, but tended towards the cooler months with outbreaks focused in the early spring and fall months. This cooler season predominance illustrates the importance of norovirus circulation during this reporting period.

Figure 5
Community Outbreaks by Selected
Diseases and Onset Month
LAC, 2006 (N=141)



COMMENTS

There was an increase in the number of outbreaks and outbreak associated cases reported in 2006 from the prior year; however, the number of outbreaks in 2006 was only one above the mean number of outbreaks for the last eight years. Varicella remained the most common cause of community-acquired outbreaks in LAC since 1999 (also see summary of the Varicella Project in the Special Studies Report section). In 2006, seven varicella outbreaks were identified in the Antelope Valley Health District (SPA 1), where the LAC DPH Varicella Surveillance Project is in place, but most outbreaks of varicella was identified in SPA 3 (n=11).

Community-acquired outbreaks result from an interaction among particular age groups, location and specific diseases. A profile emerges where the very young and early adolescent acquire infection/infestation at school (62% in pre-school, elementary, or middle school). Varicella, hand, foot and mouth disease (HFM), and pediculosis (head lice) were most common in this young group. The second age group affected by outbreaks is in the older population associated with group-home settings (30%). In this age category, GE and scabies are the most common causes (Table 2). The increased ranking of the group and retirement home as a setting for outbreaks was fueled by the increase norovirus activity during 2006.

An unusual outbreak in 2006 was caused by non-tuberculosis Mycobacterium chelonie associated with a tattoo parlor. As this outbreak differs from the usual outbreaks investigated, it has an expanded report located within the 2006 Special Studies Report Section.

Table 1. Community-Acquired Outbreaks by Disease—LAC, 2006

Disease	No. of outbreaks	No. of cases	Cases per outbreak (average)	Cases per outbreak (range)
Varicella	49	584	12	5-53
Scarlet fever/strep throat	3	44	15	3-21
Scabies	15	92	6	2-23
Hand, foot & mouth disease	16	164	10	2-67
Pediculosis	13	78	6	2-11
GE illness - Norovirus	9	311	35	13-79
GE illness - Shigella	1	2	2	2
GE illness - Salmonella	2	11	6	2-9
GE illness - Giardia	1	4	4	4
GE illness - Unknown	16	244	15	5-35
Fifth disease	2	14	7	5-9
Conjunctivitis	2	13	7	6-7
MRSA	4	19	5	3-9
Influenza B	2	18	9	8-10
Other*	7	145	21	3-119
Total	**142**	**1,743**	**12**	**2–119**

* Includes: pertussis, ringworm, Staphylococcus aureus, non-tuberculosis Mycobacterium chelonie, unknown respiratory

Table 2. Community-Acquired Outbreaks by Disease and Setting—LAC, 2006

Disease	Group Home[a]	School[b]	Preschool or Daycare	Other[c]	TOTAL
Varicella	0	47	2	0	**49**
Scarlet fever/strep throat	0	2	0	1	**3**
Scabies	14	0	0	1	**15**
Hand, foot & mouth disease	0	2	14	0	**16**
Pediculosis	1	6	5	1	**13**
GE illness - Norovirus	9	0	0	0	**9**
GE illness - Shigella	0	0	1	0	**1**
GE illness - Salmonella	1	0	1	0	**2**
GE illness - Rotavirus	0	0	1	0	**1**
GE illness - Unknown	13	0	3	0	**16**
Fifth disease (Parvovirus)	0	2	0	0	**2**
Conjunctivitis	2	0	0	0	**2**
MRSA	1	2	0	1	**4**
Influenza B	0	0	0	2	**2**
Other	1	4	1	1	**7**
Total	**42**	**65**	**28**	**7**	**142**

[a] Includes centers for retirement, assisted living, rehabilitation, and shelter.
[b] Includes elementary (n=47), middle (n=13), after-school (1), high schools (n=1) and University (3).
[c] Includes jail, vocational training sites, restaurants, tattoo parlor.

FOODBORNE OUTBREAKS

DESCRIPTION

Foodborne outbreaks are caused by a variety of bacterial, viral, and parasitic pathogens, as well as toxic substances. To be considered a foodborne outbreak, both the state and the CDC require at minimum the occurrence of two or more cases of a similar illness resulting from the ingestion of a common food.[1]

The system used by LAC DPH for detection of foodborne outbreaks begins with a Foodborne Illness Report (FBIR). This surveillance system monitors complaints from residents, illness reports associated with commercial food facilities, and foodborne exposures uncovered during disease-specific case investigations (e.g., *Salmonella, Shigella, Campylobacter*). LAC Environmental Health Services Food and Milk (F&M) Program investigates each FBIR by contacting the reporting individual and evaluating the public health importance and need for immediate follow-up. When warranted, a thorough inspection of the facility is conducted. This is often sufficient public health action to prevent additional foodborne illnesses.

ACDC's Food and Water Safety Program (F&WS) also review all FBIRs. ACDC investigates foodborne outbreaks with the greatest public health importance. An epidemiologic investigation will typically be initiated when there are illnesses in multiple households, multiple reports from the same establishment in a short period of time, or ill individuals who attended a large event with the potential for others to become ill. The objective of each investigation is to determine extent of the outbreak, identify a food vehicle or processing error, and determine the agent of infection.

INVESTIGATIONS SUMMARY

In 2006 there were 48 outbreak investigations performed jointly by F&WS and the F&M programs. Twenty-three percent of these investigations (n=11) were caused by person-to-person transmission of norovirus in a food setting and not considered to be food-related (Table 1).

For outbreaks identified as foodborne (n=37), an agent was determined in 95% of outbreaks (n=35). A majority of outbreaks were determined to be caused by a viral agent, with 16% lab confirmed and 57% suspected based on clinical and epidemiological information. Thirty-five percent of investigations identified a contributing factor in the preparation of the meal being investigated (n=13), with an ill food handler identified in 11% of outbreaks (n=4). A food item was implicated in 35% of investigations (n=13). Restaurants were the most commonly identified eating location (43%, n=16), and SPA 4 was the most commonly reported geographical area (24%, n=9).

The percent of foodborne outbreaks with suspected and confirmed viral etiology continues to increase, as exemplified by the large percent found in 2006. In addition, a new, more virulent strain of norovirus was identified in one outbreak (GII.4 Minerva), and may be responsible for the severity seen in recent outbreaks.

Table 1. Outbreak Investigations Summary

Investigations	Frequency	Percent
Total	48	100%
Person-to-person norovirus	11	23%
Foodborne outbreak identified	37	77%
Foodborne Outbreaks Identified	**Frequency**	**Percent**
Total	37	100%
Agent determined	35	95%
Bacterial (lab confirmed)	6	16%
Viral (lab confirmed)	6	16%
Viral (suspect norovirus)	21	57%
Contributing factor identified	13	35%
Ill food handler	3	8%
Food item implicated	13	35%
Seafood (sushi, tuna)	6	16%
Occurred at a restaurant	16	43%
Occurred in SPA 4	9	24%

1 CDC. Surveillance for foodborne disease outbreaks—United States, 1988–1992. MMWR 1996; 45(SS-5):58. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/00044241.htm

Overview: In 2006, there were 2012 FBIRs reported from consumers eating food from establishments located in LAC. Thirty-nine percent of reports (n=784) were investigated by the F&M program, and 23% (n=453) were referred to district inspectors or another agency for follow-up. The remaining 39% (n=775) were either duplicate reports on the same establishments, or contained incomplete or inaccurate complaint information for follow-up.

In 2006, 37 foodborne outbreaks were jointly investigated by the F&WS and F&M programs, representing 425 cases of foodborne illness, and an average of 9 persons per outbreak (range 1-57 cases) (Figure 1). One waterborne outbreak identified in 2006 occurred in a bar where hepatitis A infections were associated with ice served at the bar.

Seasonality: Foodborne outbreak investigations occurred throughout the season in 2006, with many outbreaks occurring in the late winter and spring months (Figure 2).

Figure 1
Foodborne Outbreaks
Number of Persons Affected
LAC, 2002–2006

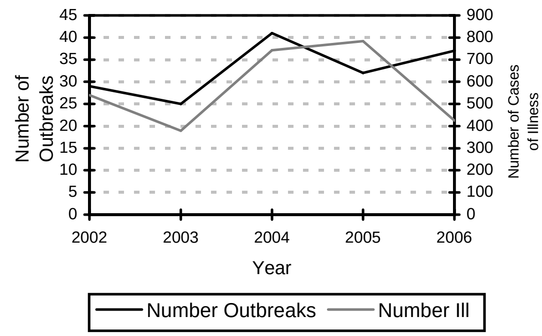


Figure 2
Foodborne Outbreak Investigations
by Month of Onset
LAC, 2006 (N=37)

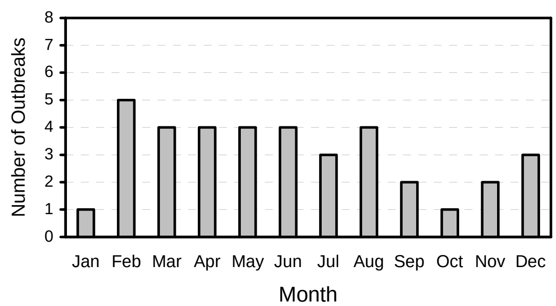
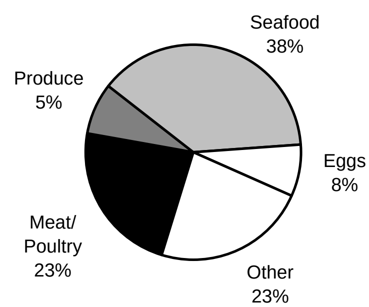
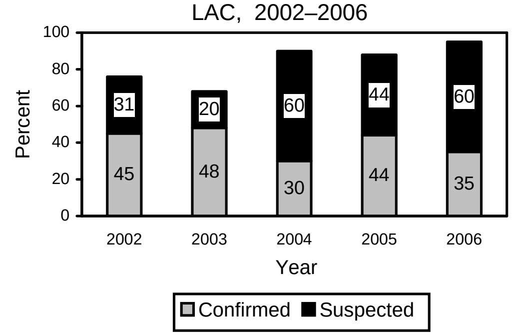


Figure 3
Foodborne Outbreaks
with Implicated Food Vehicles
LAC, 2006 (n=13)



Implicated Food Vehicles: A food vehicle was epidemiologically implicated in 35% of foodborne outbreaks (n=13), with an etiologic agent lab confirmed in a food item in one outbreak. A seafood product was the most commonly implicated item (38%, 2 sushi, 1 tuna, 2 tuna salad) followed by meat and poultry items (23%, 2 chicken, 1 beef) (Figure 3).

Figure 4
Foodborne Outbreaks
% with Suspected/Confirmed Etiologic Agent
LAC, 2002–2006



Agent: In 2006, an agent was laboratory confirmed in 35% of investigations (n=13), similar to previous years (Figure 4). Of the 60% of outbreaks with a suspect agent, 57% (n=21) were suspected to be norovirus based on based on symptoms onsets, symptoms durations, incubation period, duration of symptoms, secondary cases in households, and/or negative bacterial test results. Reasons for no laboratory testing include lack of cooperation, delayed notification and cases out of town/unavailable. Of foodborne outbreaks with a lab confirmed or suspect agent (n=35), 73% of with a lab confirmed or suspect agent (n=35), 73% of these investigations were viral etiology (n=27; 25 norovirus, 2 hepatitis A) and 16% of these were identified as bacterial (n=6; 5 salmonella, 1 campylobacter) (Figure 5). Foodborne outbreaks reports with a viral etiology appear to be increasing in more recent years.

Outbreak Location: The most common locations for reported foodborne outbreaks were restaurants (43%, n=16) followed by food that was brought or catered to a workplace (14%, n=5) or eaten at home (14%, n=5)(Figure 6). Other locations include places of worship, schools, and parks. The geographic distribution of the outbreaks by SPA is summarized in Table 2. SPA 4 reported the most outbreaks (24%, n=9), similar to that reported in 2005 (28%). There were several multi-district and one multi-county outbreak, but there were no outbreaks that involved multiple states.

Figure 5
Foodborne Outbreaks
by Etiologic Agent Category
(Lab Confirmed and Suspect)
LAC, 2002–2006

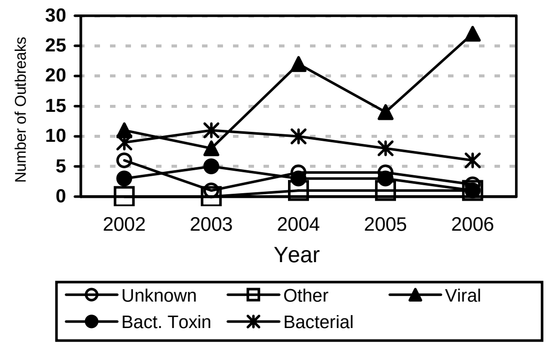


Figure 6
Foodborne Outbreaks
by Place Eaten
LAC, 2006 (N=37)

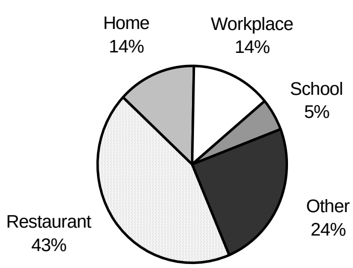
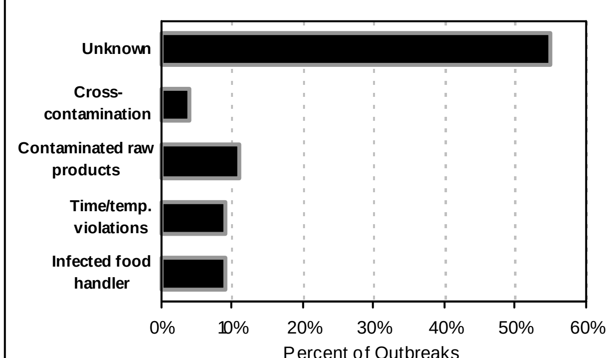


Table 2. Frequency of Foodborne Outbreaks by Location, 2006

SPA	Frequency	Percent
1	1	3%
2	6	16%
3	5	14%
4	9	24%
5	7	19%
6	0	0%
7	4	11%
8	3	8%
Multi-district	2	5%
Multi-county	1	3%
Multi-state	0	0%
Total	37	100%

Contributing Factors: In 2006, a contributing factor was identified in 35% of foodborne outbreak investigations (n=13) (Figure 7). The most frequent factors identified were potential contamination of raw food products (11%, n=4). An ill food handler was identified in 8% (n=3) of outbreaks, with one food handler lab confirmed with norovirus and 3 suspected to be norovirus based on symptoms.

Figure 7
Foodborne Outbreaks with Known
Probable Contributing Factors (N=37)
LAC, 2006



DISCUSSION

The percent of foodborne outbreaks with viral etiology continue to increase in recent years, as exemplified by the large percent found in 2006. In addition, a new, more virulent strain of norovirus (GII.4 Minerva) was identified in a large person-to-person outbreak affecting multiple jurisdictions. This outbreak affected 113 persons and resulted in 35 medical visits and 2 hospitalizations. According to the US Centers for Disease Control and Prevention (CDC), norovirus infection and illness reports have increased significantly, with the appearance of this new strain accounting for at least 60 percent of the outbreaks occurring this past winter 2005. Outbreaks have occurred across the US in a variety of settings, including colleges, prisons, elementary schools, cruise ships and long-term care facilities. The increased reports of norovirus illness prompted federal health officials to gather for an assessment of this new strain. The pathogen is named for the Minerva II cruise ship, where health officials first became aware of the particularly virulent norovirus strain during a shipboard outbreak in January 2006.

Since 1999, the LAC Public Health Laboratory has been testing human specimens for norovirus using the reverse transcription-polymerase chain reaction (RT-PCR) method. This method is still considered to be experimental and is only used to diagnose outbreaks as a whole, not for individual cases. There has been a marked increase in the number of viral GE and confirmed norovirus outbreaks since 1999.

To assist in the identification of national outbreaks, the PulseNet system is used to monitor for strains of various etiologic agent. PulseNet is a public health network sponsored by the CDC that uses the collaboration of laboratories and health departments at local, state, and federal levels to detect outbreaks through comparison of results of pulsed-field gel electrophoresis (PFGE) of pathogens. The PFGE are monitored for strains of various etiologic agents. When similar resulting patterns are detected, an investigation may be initiated. In addition, PFGE results can link solitary case occurring locally to a larger, previously identified outbreak occurring on a wider geographical scale (e.g., multistate *E. Coli* O157:H7 outbreak).

Persons with mild symptoms, long incubation periods, and poor public and medical community awareness of public health procedures may contribute to under-reporting of foodborne disease.

Table A. Foodborne Outbreaks in LAC, 2006 (N=37)

	Agent	Confirmed/ Suspected	Strain/Type	OB#	Source	Setting	Cases	HD
1	Norovirus	Lab Confirmed		174	Undetermined	Restaurant	14	Whittier
2	Norovirus	Lab Confirmed		178	Undetermined	Hotel Rest	26	Inglewood
3	Norovirus	Lab Confirmed		220	Tuna Salad	Restaurant	8	Central
4	Norovirus	Lab Confirmed*		148	Potato	Dining Hall	57	Central
5	Norovirus	Suspected		20	Undetermined	Residence	25	E. Valley
6	Norovirus	Suspected		48	Undetermined	Funeral	6	Multi
7	Norovirus	Suspected		54	Undetermined	Work Place	7	Central
8	Norovirus	Suspected		72	Undetermined	Restaurant	10	Antelope Valley
9	Norovirus	Suspected		74	Undetermined	Restaurant	8	Central
10	Norovirus	Suspected		90	Sushi	Restaurant	9	West
11	Norovirus	Suspected		95	Undetermined	Restaurant	17	San Fernando
12	Norovirus	Suspected		102	Undetermined	Restaurant	5	Pomona
13	Norovirus	Suspected		112	Undetermined	Restaurant	6	Whittier
14	Norovirus	Suspected		133	Undetermined	Work Place	6	San Fernando
15	Norovirus	Suspected		134	Undetermined	Work Place	6	West
16	Norovirus	Suspected		138	Undetermined	Work Place	7	Hollywood Wilshire
17	Norovirus	Suspected		167	Undetermined	School	19	Central
18	Norovirus	Suspected		172	Undetermined	School	4	West
19	Norovirus	Suspected		180	Undetermined	Residence	9	West
20	Norovirus	Suspected		235	Tuna Salad	Church	7	West
21	Norovirus	Suspected		238	Undetermined	Restaurant	12	Harbor
22	Norovirus	Suspected		38	Undetermined	Restaurant	8	Alhambra
23	Norovirus	Suspected*		37	Undetermined	Residence	13	Multi
24	Norovirus	Suspected		16	Undetermined	Residence	8	West
25	Norovirus	Suspected		162	Muffins	Retreat	19	San Antonio
26	Salmonella	Lab Confirmed	*typhimirum*	150	Chicken	Dining Hall	39	San Antonio
27	Salmonella	Lab Confirmed	*heidelberg*	161	Raw egg shake	Restaurant	8	West
28	Salmonella	Lab Confirmed	*oranienberg*	169	Undetermined	Restaurant	6	Central
29	Salmonella	Lab Confirmed	*typhimirum*	198	Chicken	Restaurant	5	Central
30	Salmonella	Lab Confirmed	*enteritidis*	236	Potato Puffs	Dining Hall	19	Foothill
31	Hepatitis A	Lab Confirmed		145	Ice	Bar	8	Harbor
32	Hepatitis A	Lab Confirmed		176	Undetermined	Restaurant		Pomona
33	Shigella	Lab confirmed	*sonnei*	9	Sushi	Restaurant	8	Central
34	Toxin	Lab confirmed	(Scombroid)	124	Tuna	Restaurant	2	West Valley
35	Bacterial Toxin	Suspected		217	Meatballs	Residence	7	Central
36	Unknown GI	Unknown		15	Undetermined	Work Place	7	Bellflower
37	Unknown GI	Unknown		96	Undetermined	Restaurant	13	Central

Table B. LAC Foodborne Outbreaks Laboratory Summary: Outbreaks Confirmed Etiologic Agent, 2006

Number of Outbreaks	Bacterial	Bacterial Toxin	Norovirus	Hep A	Unknown/ Other	Total
Investigated	6	1	27	2	2	37
Tested	6	1	4	2	2	13
Lab Confirmed	6	1	4	2	2	13

ADDITIONAL RESOURCES

LAC resources:

- Communicable Disease Reporting System
  Hotline: (888) 397-3993
  Faxline: (888) 397-3779
- For reporting and infection control procedures consult the LAC DHS Foodborne Disease Section in the B-73 Manual – http://lapublichealth.org/acd/procs/b73/b73index.htm

CDC:

- Foodborne and Diarrheal Diseases Branch – www.cdc.gov/foodborne
- Outbreak Response and Surveillance Team – www.cdc.gov/foodborneoutbreaks
- FoodNet – www.cdc.gov/foodnet
- Norovirus Information – www.cdc.gov/foodborneoutbreaks/

Other national agencies:

- FDA Center for Food Safety and Applied Nutrition – www.cfsan.fda.gov
- Gateway to Government Food Safety Information – www.FoodSafety.gov

HEALTHCARE ASSOCIATED OUTBREAKS

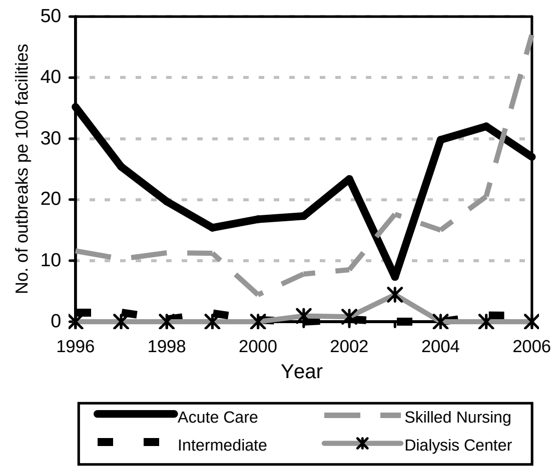
DEFINITION

Healthcare associated outbreaks are defined as clusters of nosocomial (health-facility acquired) or home-healthcare-associated infections 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.

ABSTRACT

- Confirmed healthcare associated outbreaks increased 81% from 2005 to 2006 and 123% from the mean number of outbreaks the previous 4 years.
- In 2006, skilled nursing facility (SNF) outbreaks were responsible for the entire increase in healthcare facility outbreaks, and increased 128% from 2005 (Table 1). This was largely due to a significant increase in gastrointestinal (GI) outbreaks.

Figure 1
Healthcare Associated Outbreaks
LAC, 1996–2006



Denominator data from the Electronic Licensing Management System(ELMS) database for LAC

Table 1. Number of Reported Outbreaks in Healthcare Facilities LAC, 2002–2006

	YEAR				
Type of Facility	**2002**	**2003**	**2004**	**2005**	**2006**
Acute Care Hospitals	26	8	31	34	28
Provider Offices	2	0	0	0	0
Dialysis Facilities	1	9	0	0	0
Intermediate Care/Psych	1	0	0	3	3
Skilled Nursing Facilities	37	75	63	76	173
TOTAL	**67**	**92**	**94**	**113**	**204**

Acute Care Hospitals: There were 28 outbreaks reported in acute care hospitals in 2006 (Table 1). Fifty percent (n=14) of these outbreaks occurred in a unit that required intensive or focused specialized care (e.g., NICU, cardio-thoracic unit, burn unit) (Table 2). Eighteen percent (n=5) occurred in the psychiatric or behavioral units within the acute care hospital. As in previous years, scabies accounted for the majority of acute care outbreaks (n=8 or 29%). Fifty percent (n=14) of acute care outbreaks were of bacterial etiology (Table 3). Multi-drug resistant organisms such as MRSA, *Stenotrophomonas maltophilia* and *Acinetobacter baumannii* accounted for 8 outbreaks in 2006. In 2006, the etiologic agents contributing the largest number of cases in acute care outbreaks were mold (n=85 or 24%), followed by scabies (n=83 or 25%).

Table 2. Acute Care Hospital Outbreaks by Unit—LAC, 2006

Outbreak Location	No. of Outbreaks
Intensive Care-Neonatal	7
Multiple Units	7
Psychiatric	5
Intensive Care - Adult	3
Medical-Surgical	2
Burn	1
Cardio-thoracic	1
Nursery, Intermediate Care	1
Telemetry	1
Total	**28**

Table 3. Acute Care Hospital Outbreaks by Disease/Condition—LAC, 2006

Disease/Condition/ Etiologic Agent	No. of Outbreaks	No. of Cases
Scabies	8	83
Acinetobacter baumannii	3	36
MRSA	3	12
Multiple Mold	2	85
Stenotrophomonas maltophilia	2	21
Unknown Gastroenteritis	2	27
Candida albicans	1	6
Elizabethkingia meningoseptica	1	25
Escherichia coli	1	8
Multiple Bacterial Organisms	1	3
Norovirus	1	8
Pseudomonas aeruginosa	1	11
Salmonellosis (Non-Typhoid)	1	3
Vancomycin-Resistant Enterococcus faecium	1	6
TOTAL	**28**	**334**

Skilled Nursing Facilities: Reported skilled nursing facility outbreaks increased by 128% in 2006, with 173 outbreaks in 2006, as compared to 76 outbreaks in 2005. Gastroenteritis, including unknown GI, and scabies were the most common causes (Table 4), together accounting for 95% of the total outbreaks in SNFs and 97% of the total cases.

Table 4. Skilled Nursing Facility (SNF) Outbreaks by Disease/Condition LAC, 2006

Disease/Condition	No. of Outbreaks	No. of Cases
Gastroenteritis • unspecified (n=1) • norovirus (n=60)	61	1574
Unknown Gastroenteritis	56	854
Scabies	48	338
Unknown Rash	4	60
Clostridium difficile	1	8
Salmonellosis (Non-Typhoid)	1	2
Respiratory illness • unspecified	1	2
Headlice	1	2
Total	**173**	**2840**

COMMENTS

Healthcare associated infections (HAI) have generated a great deal of attention in the US within the past few years, with a focus on public disclosure of HAI's in the acute care hospital setting [1]. There has been ongoing debate among stakeholders on how to best facilitate HAI disclosure, and no consistent process to disseminate the information, based on the same criteria, has been identified. In September 2006, California approved Senate Bill (SB) 739, which directs hospitals to evaluate and augment existing infectious disease control programs and implement new standards to prevent HAI. Implementation began July 1, 2007 and will be phased in over a three-year period. Major components of the bill are pandemic influenza preparation and planning, evaluating the judicious use of antibiotics and annual state reporting its implementation of specified infection surveillance and infection prevention process measures, and to develop, implement and evaluate compliance with policies and procedures to prevent surgical site infection, ventilator associated pneumonia, and subject to surveys by CDPH Licensing and Certification on compliance with new infection control procedures and reporting measures [2]. ACDC is working with the state and local providers regarding the requirements of this bill.

Los Angeles County experienced a slight decrease in the number of reported scabies outbreaks in both acute care and skilled nursing healthcare facilities from 2005 to 2006. In 2005, 13 scabies outbreaks (229 cases) were reported in acute care facilities, as compared to 8 scabies acute care facility outbreaks (83 cases) in 2006 (Table 3). Overall, SNF scabies outbreaks also decreased. In 2005, 55 scabies outbreaks (404 cases) were reported in SNFs, as compared to 48 (338 cases) SNF outbreaks in 2006, a decrease of 13%.

In 2005 and 2006, ACDC initiated a SNF needs assessment to assess general communicable disease reporting knowledge, ascertain staff infection control practices, and to identify knowledge gaps and elicit training needs. One hundred SNFs in Los Angeles County were randomly selected to participate in the needs assessment and fifty-nine (59%) responded to the survey questionnaire. All respondents (n=59) reported that they have an infection control policy and procedure manual and 51 (86%) reported that the manual is reviewed annually. At the time of the survey, fifty-eight respondents (98%) reported that they have at least one individual assigned to infection control activities that are trained in disease surveillance, prevention and control, and fifty-four (92%) reported that the individual assigned to infection control activities is a full-time employee. Eighty-six percent are interested in infection control and reportable disease training for their staff. Additional training topics identified include hand hygiene education, MRSA, *Clostridium difficile*, scabies and influenza management and control. Based on these findings, ACDC will explore collaboration with LAC DPH Health Facilities Inspection Division to address training needs.

ACDC investigated two outbreaks that implicated improper and/or inconsistent disinfection and cleaning practices of reusable medical devices. The first outbreak involved an adult ICU and *Escherichia coli* found on the transesophageal echocardiography (TEE) probe, a flexible endoscope used to visualize the heart. The second outbreak involved a neonatal ICU and *Pseudomonas aeruginosa* discovered on a laryngoscope blade. In both outbreaks, instrument cleaning was in violation of the facility's established cleaning and disinfection policy (see 2006 Special Studies Report for detailed article). Staff non-compliance with their facility's instrument cleaning and disinfection policies is frequently cited in the literature as a cause of hospital-acquired infection [3].

For several years throughout the US, cases and outbreaks of *Clostridium difficile* associated disease (CDAD) have increased. The CDC has verified that the new highly toxic strain of *Clostridium difficile* (*C. diff*) has been confirmed in LAC patients. This strain of *C. diff*, known as B1/NAP1, has been associated with high recurrence rate and fatality. The *C. diff* bacillus produces several exotoxins that can cause colitis, ileus, and even death. The new strain produces as much as16 times more toxin A and 23 times more toxin B, compared with the common strain. The bacillus is carried in feces and is transmitted through direct and indirect contact with the contaminated environment or hands of healthcare providers. Previously, the severely ill and elderly patients on prolonged antimicrobials were typically affected; however, more and more the disease is being found among the otherwise healthy population. Symptoms include watery bloody diarrhea, severe cramping, abdominal pain, and fever. An advisory was sent to hospital infection control professionals informing them that the virulent strain was now identified in LAC and to strengthen appropriate infection prevention and control measures to reduce nosocomial transmission. ACDC will continue to monitor the situation and encourage our healthcare partners to conduct surveillance by screening symptomatic patients appropriately to help identify future trends of the disease and its changing epidemiology.

Another disturbing trend is the increase in multidrug-resistant organisms (MDROs) that are seen in all healthcare settings, and of particular significance in hospitalized patients. Methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant enterococcus (VRE) and certain Gram-negative bacteria (e.g., *Acinetobacter baumannii* and *Elizabethkingia meningoseptica*), are resistant to multiple classes of antimicrobial agents [4]. The full economic impact of MDROs on acute healthcare facilities and society at large has yet to be determined; however, the literature cites challenges faced because "…so many variables and perspectives are involved". These challenges have many elements, including clinician prescribing practices and the appropriate and judicious use of antibiotics; development of new antimicrobial agents; surveillance for antimicrobial-drug resistance, implementing infection control measures; adapting laboratory methods for detecting new types of antimicrobial-drug resistance, education programs, and influencing drug choice [5]. ACDC will continue to monitor this shift and collaborate on control and prevention efforts with state and national organizations.

The ACDC Hospital Outreach Unit (HOU) is an integral component of the public health link to infection control professionals and community healthcare agencies. Team members continue to strengthen communication and collaboration between public health and the acute care hospitals to increase disease and outbreak reporting.

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Acute Communicable Disease Control Program

Special Studies Report

2006

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2006 SPECIAL STUDIES REPORT

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INTEGRATION OF PUBLIC HEALTH NURSING INTO A REGIONAL TERRORISM INTELLIGENCE CENTER

BACKGROUND

The public health nurse (PHN) enhances Los Angeles County (LAC) Public Health's ability to respond to bioterrorism (BT) and other emergencies through consultation, coordination, and training services to health professionals, law enforcement, government officials, and community agencies. The PHN integrates disease surveillance into the Los Angeles Joint Regional Intelligence Center (JRIC), a multi-agency terrorism intelligence task force that includes representatives from the FBI, local law enforcement, public health and fire departments by providing accurate disease information and ongoing analysis of all threats including biological terrorism. According to *Healthy People 2010*, two of the leading health indicators are injury and violence, and mental health. Los Angeles County Public Health originally committed a full-time PHN to the Los Angeles Terrorism Early Warning (TEW) Group in 2003, prior to the absorption of TEW into the larger JRIC operation. The PHN ensures integration of the public health discipline into law enforcement investigations by providing estimates of possible cases of suspicious disease occurrences or outbreaks, populations at risk, and potential deaths, as well as information on management of outbreaks such as prophylaxis, treatment, and isolation.

Figure 1. Los Angeles Joint Regional Intelligence Center (JRIC): a multidisciplinary intelligence center

JOINT REGIONAL INTELLIGENCE CENTER (JRIC)

The Los Angeles Joint Regional Intelligence Center is a multidisciplinary, intelligence center (Figure 1) composed of public health, fire services, FBI, police and sheriff departments working in partnership with other local, state, and federal programs to share and analyze information, disseminate intelligence, and assist with the coordination of resources for a unified response to a terrorism event. The JRIC grew from an established, local center called the Terrorism Early Warning Group (TEW) that provided LAC stakeholders with local terrorism analysis and advisories.

This framework is based on a core foundation of shared information and collaborative efforts which can be used as a tool for full integration of PHNs into the medical intelligence analysis function of the JRIC.

This new approach to integration facilitates the access to public health information and increases multi-disciplinary response capability to potential BT events. The PHN provides real-time disease information to the FBI, law enforcement and fire agencies at daily JRIC briefings and can facilitate the access to public health subject-matter experts.

PUBLIC HEALTH NURSE ROLE

The PHN functions as an intelligence analyst by researching and studying known terrorist groups in order to assess their ability to develop and deploy biological and chemical weapons. The PHN currently possesses a Department of Justice security clearance that assists with the collection and assessment of terrorist threat information that is generally unavailable to most public health practitioners. The majority of the information that is gathered to conduct this intelligence analysis is through the monitoring and assessing open media sources, law enforcement bulletins and non-classified disease intelligence sources. Due to the education and experience inherent to the public health profession, the PHN participates in the planning and coordination of public health bioterrorism and emergency preparedness drills and exercises, ongoing response protocol development, and the development of after action reports that facilitate the synchronization of public health and public safety responses to terrorist attacks.

In this new area of practice, the PHN serving as a terrorism and medical intelligence analyst also serves as a link between public health and many other experts with a variety of disciplinary skills to increase resource capabilities in response to possible BT attacks. This approach strengthens preparedness and response capacity of the LAC public health system, and strengthens the relationship among the JRIC partners and respective representatives.

The PHN fully integrates with the JRIC bioterrorism threat analysis and response planning resources to address interests and concerns of public health in the collaborative approach in response to bioterrorism. The PHN facilitates JRIC accesses to public health information and subject-matter expertise, enhances sharing information, improves mobilization of community partners, promotes disease surveillance and joint investigation, and maximizes capacity and response capability to counteract bioterrorism attacks. The PHN works with experts from other public health disciplines such as mental health, environmental health, toxics epidemiology, radiation management, veterinary science and communicable diseases to ensure a better understanding of the public health role in community preparedness, and to be an advocate for the public health mission within the JRIC. The JRIC staff may need the expert analysis of intelligence related to a potential chemical, biological or radiological attack to validate the credibility of that information, and to recommend potential courses of action.

The PHN who is integrated into a regional terrorism intelligence center adheres to both the LAC and Minnesota practice models, and to the following core components of public health nursing that are currently being practiced. The Minnesota Public Health Nursing Interventions Model [1] provides PHNs with a guide to formulate and implement public health nursing plans. The model describes levels of practice with the various types of interventions that optimize delivery of public health services in society. The PHN integrates the surveillance, disease and health event investigation interventions into the analysis process by observing anomalies that may indicate the presence of a biological attack while presenting these findings in a multi-disciplinary, collaborative environment.

The LAC Public Health Nursing Practice Model [2] provides PHNs with a comprehensive framework to function within an interdisciplinary environment. The model allows PHNs to use public health nursing knowledge and skills with knowledge from other disciplines to meet the changing health needs of the community. The PHN assesses the reportable disease and outbreak investigations, investigates the possible connection between these events and terrorism threat information for any feasible connection to terrorism, reports findings to appropriate health and law enforcement individuals, develops protocols and strategies for analyzing and sharing these findings, links appropriate health and law enforcement entities and evaluates the validity of these strategies. This team approach offers PHNs access to a new network of counterterrorism and homeland security professionals that facilitate reliable exchange of information and foster collaboration among agencies to improve the overall response capability of public health to BT attacks.

CONCLUSION

The challenges posed by terrorism, the necessary integration of public health expertise into the law enforcement/intelligence community, and the importance of partnership and multi-agency, multidisciplinary collaboration to achieve common goals has created a new area of specialization for 21st century public health nurses. PHNs continue to integrate public health expertise into the JRIC through the application of the nursing process and by linking law enforcement and fire services with public heath subject-matter experts. PHNs should become familiar with the subject of medical intelligence gathering, assessment and analysis in order to meet the challenges of countering the threat of bioterrorism in their local community.

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SUSPECTED SMALLPOX CASE INVESTIGATION TRAINING

BACKGROUND

Smallpox is a serious, contagious, and often fatal infectious disease that was declared eradicated worldwide in 1980 by the World Health Organization (WHO). However, the events of September 2001 and October 2001 raised concern that the smallpox (variola) virus could be used as an agent of biologic terrorism. Since there has been limited experience among public health personnel in responding to smallpox cases, four smallpox case investigation training sessions were provided to Los Angeles County (LAC) Department of Public Health (DPH) Smallpox Response Team members to increase their knowledge and skills in responding to these situations. These trainings were offered to build Los Angeles County's smallpox preparedness capacity in conducting investigations and outbreak control for suspected smallpox cases.

METHODS

From April 2006 through May 2006, four suspected smallpox case investigation trainings were offered by the LAC DPH Acute Communicable Disease Control (ACDC) Program, Bioterrorism Preparedness and Response Section. The trainings were conducted by two physician specialists, three health educators, a public health nurse, and a Federal Bureau of Investigation (FBI) special agent.

The training was organized into four components that consisted of a comprehensive lecture, a hands-on demonstration of the Smallpox Aid Response Kit (SPARK), which is a "go-bag" that contains supplies including: personal protective equipment (PPE), laboratory specimen collection kit and procedures, case investigation forms, smallpox rash evolution guide, a digital camera, and laptop with wireless connection.

The objectives of the training were as follows:

- List the three major diagnostic criteria for smallpox;
- List three of five minor diagnostic criteria for smallpox;
- State how smallpox is transmitted;
- State the notification process for a suspected smallpox case;
- State the infection control precautions needed for a suspected smallpox case;
- Describe the functional role as a member of a public health smallpox response team in the initial evaluation of a suspected smallpox case;
- Describe two mechanisms by which a terrorist attack could be perpetrated using the smallpox virus and its relevance to the assessment of a smallpox case and;
- Describe the importance of chain of custody in evidence collection and transport.

Training participants received a packet of materials that included the notification and call-down process, risk assessment for smallpox using the Centers for Disease Control and Prevention (CDC) criteria, guides for distinguishing smallpox from chickenpox, standardized procedures for specimen collection, guidelines for evaluating a rash illness suspicious for smallpox, PPE donning and removal procedures, worksheets for evaluating a suspect smallpox case(s), checklists for investigating a suspected smallpox case, clinical and non-clinical resource guides, and a list of the SPARK items. The chain of custody issues for specimen collection and joint investigation with DPH and the FBI were reviewed. A copy of The Joint Bioterrorism Investigation Memorandum of Understanding between DPH, FBI, and LAC Sheriff was then given to the participants.

The target audience for this training was DPH staff physicians, public health investigators, nurses, and other staff who would respond to assess and investigate suspect smallpox case(s) in LAC. Smallpox-vaccinated LAC public health workers were contacted by telephone and e-mail to participate in one of the four training sessions. Participants consisted of clinical (physicians and nurses) smallpox-vaccinated individuals and non-clinical vaccinated individuals.

To evaluate changes in the knowledge and skills of the participants, pre-tests and post-tests were administered at the training. The pre-test and post-test included 10 questions consisting of true/false and multiple choice questions. There was an additional section of three short answer clinical exam questions for clinical staff only. Analysis of the pre-tests and post-tests was conducted with the SAS Software program. In addition, pre-tests and post-tests were grouped by the four different sessions. In an effort to maintain confidentiality of the participants, names and identification numbers were not used.

The planning and evaluation process of the training were based on the Continuing Medical Education (CME) and Continuing Education Unit (CEU) activity guidelines. The California Medical Association approved the activity for 2.0 CME category 1 credits. The California Board of Registered Nursing approved the activity for 2.0 CEU contact hours. The CME Program Evaluation and LAC Public Health Nursing Administration Evaluation were conducted for all four training sessions. The evaluation measured if the training objectives were met and also requested general comments about the training. Participants were asked to submit their views on how the information will be applied to their public health duties to improve effectiveness. Further, participants were asked to list the two most pertinent pieces of information they learned from the training. To assist with future programs, participants were also asked to indicate future needs and topics they would like to have reviewed.

RESULTS

There were 45 clinical and 10 non-clinical staff participants that completed the training. Only pre-test and post-test pairs completed by clinical staff participants were analyzed (n=42). The Paired T-test was calculated for the pre-tests and post-tests utilizing the SAS Software program. Analysis revealed that there is a statistical significance between pre-tests and post-tests ($p<0.0001$). The mean scores out of 100% of the pre-tests were: Group I = 69.4, Group II = 69.4, Group III = 63.4, and Group IV = 70.4. A majority of participants scored below 70% on the pre-test.

An analysis of the post-test revealed improvement in knowledge with the following scores: Group I = 86.2, Group II = 86.2, Group III = 87.8, and Group IV = 90.2. Overall, post-test mean scores ranged from 86.2 % to 90.2% which increased from the pre-test mean score of 70%. Participants that were non-clinical staff members (n=10) did not participate in the clinical exam questions. Therefore, their results were not calculated in the mean scores of the pre-test and post-test.

Table 1. Pre-Test and Post-Test Results of Clinical Staff Participants

		Mean Scores			95% C.I.			
Groups	**# of participants (n=42*)**	**Pre-Test**	**Post-Test**	**Difference**	**Lower**	**Upper**	**T Value**	**P Value**
1	12	69.4	86.2	18.5	14.2	22.8	9.42	<0.0001
2	12	69.4	86.2	18.5	14.2	22.8	9.42	<0.0001
3	9	63.4	87.8	24.3	19.3	29.4	11.07	<0.0001
4	9	70.4	90.2	17.6	12.4	22.7	7.87	<0.0001

* Based on pairs matched for completed tests in order to conduct analysis.

CONCLUSION

Suspected smallpox case investigation trainings were provided to smallpox vaccinated public health workers in LAC to improve knowledge and skills in responding to suspect and initial smallpox case(s). A total of 45 clinical and 10 non-clinical public health staff members completed the training. The Paired T-test was calculated for the pre-tests and post-tests utilizing the SAS Software Program. Analysis revealed that there was a statistical significance between pre-tests and post-tests ($p<0.0001$). A majority of the participants scored below 70% on the pre-test. However, post-test scores showed improvement in knowledge with mean scores ranging from 86.2% to 90.2%.

The training included an overview of the notification and call-down process, risk assessment for smallpox using CDC criteria, systematic approach to evaluating a febrile vesicular or pustular rash illness using CDC diagnostic algorithms, and information about isolation and infection control precautions. Participants had the opportunity to become familiar with transmitting digital photos via wireless laptop. A hands-on demonstration of the Smallpox Aid Response Kit (SPARK), laboratory specimen collection procedure, and a discussion about the chain of custody issues for specimen collection were reviewed.

In evaluating the four training sessions, 92% to 100% of participants thought that the course objective to state the notification process for a suspected smallpox case was fully met. In addition, 85% to 100% of the participants thought that the objective to describe the functional role as a member of the public health response team in the initial evaluation of a suspected smallpox case was fully met. Some of the general comments about the training were: very well done, excellent overview, comprehensive, practical, instructive, and straight forward presentation with good information. Overall, a majority of the participants agreed that they would recommend future sessions, such as this, to their colleagues. In the near future, a yearly refresher course will be conducted as a self-study module so Smallpox Response Team members can maintain their skills and knowledge.

BOTULISM SUMMARY
LOS ANGELES COUNTY, 2006

Botulism is a rare but serious paralytic illness caused by a nerve toxin produced by the bacterium *Clostridium botulinum* (and rarely other species). There are three main kinds of botulism. Foodborne botulism is caused by eating foods that contain the botulism toxin. Wound botulism is caused by toxin produced from a wound infected with *Clostridium botulinum*. Infant botulism (also known as intestinal botulism) is caused by consuming the spores of the botulinum bacteria, which then grow in the intestines and release toxin. All forms of botulism can be fatal and are considered medical emergencies. Foodborne botulism can be especially dangerous because many people can be poisoned by eating a contaminated food.

A total of seven patients were reported with suspected botulism in 2006 to Los Angeles County (LAC) Department of Public Health (DPH), only two of which were confirmed with the disease (Table 1). Most suspects were male (n=6), most were Hispanic (n=6) and ages ranged from 10 to 63 years (mean=45). Four suspect cases were injection drug users (IDUs), including the two confirmed cases. Antitoxin was administered to four suspect cases based on their risk factors and presenting signs and symptoms.

The LAC Public Health Laboratory (PHL) performed analyses on specimens from five suspect cases. After investigation, only two cases were confirmed as wound botulism. This report excludes cases of infant botulism, which is monitored by the California State Department of Health Services (DHS).

CASE REPORTS

<u>Confirmed Wound Botulism (n=2)</u>: Two of the four cases of IDUs reported with possible botulism were confirmed; both were Hispanic males, and both were confirmed by demonstration of botulinum type A toxin in serum.

<u>Probable Wound Botulism (n=2)</u>: The other two IDUs were domestic partners who presented to hospital together with typical botulism signs and symptoms; both had obvious injection abscesses that were cultured. They were admitted for diagnostic work-up and treatment; wound cultures were obtained, but pre-treatment sera were not submitted for testing. The male was treated with antitoxin; a post-treatment serum sample was negative for botulinum toxin. The female suspect was admitted but not treated with antitoxin. Their wound cultures were negative for clostridia. They left the hospital against medical advice.

<u>Other Central Nervous System Disease (n=3)</u>: A 10 year-old boy with cerebral palsy had been receiving periodic therapeutic injections of BoTox® (toxin type A) to relieve muscle spasms. A month prior to report, the brand of toxin was changed to Myobloc® (toxin type B) without knowledge of the treating physician. These products are not bioequivalent (i.e., the same dosage has different physiological effects) and the dosage was not decreased accordingly. After the last treatment, the physician noted the onset of bilateral facial nerve weakness, ptosis, floppy neck, and lax palate, as well as noisy breathing; a full neurological assessment was made difficult by his preexisting disorder. He was being evaluated for sleep apnea when the pharmaceutical oversight was discovered. Serum tested five weeks after the last injection was negative for botulinum toxin; however the findings are consistent with medically induced (iatrogenic) botulism. Confusion between the two forms of therapeutic botulinum toxin has been noted previously, and package inserts for both products draw attention to this point.

Two patients reported with possible botulism were found to have another neurological disorder. A man was assessed for possible botulism but ultimately diagnosed with Guillan-Barré syndrome (GBS) after showing clinical improvement with administration of IVIG; he also had a history of a recent diarrheal illness, not uncommon with GBS. The final suspect had a clinical presentation compatible with botulism and no history of wounds or self injection; he was treated with antitoxin for possible foodborne botulism. Serum and stool were negative for toxin, and stool was negative for clostridia; no suspect foods were found in the home.

Table 1. Suspected Botulism Cases, LAC DPH, 2006

Age/ Sex	Race/ Ethnicity	Month of onset	Injection drug user	Serum test*	Stool test¶	Wound culture	Anti-toxin	Diagnosis
10 M	Asian	Dec. 05	N	Neg.	--	--	No	Cerebral palsy; possible iatrogenic botulism
62 M	Hispanic	Feb.	Y	Type A	--	--	Yes	Wound botulism, type A
47 F	Hispanic	Aug.	Y	--	--	Neg.	No	Probable wound botulism
47 M	Hispanic	Aug.	Y	--	--	Neg.	Yes	Probable wound botulism
43 M	Hispanic	Nov.	N	--	--	--	No	Guillain-Barré syndrome
52 M	Hispanic	Dec.	Y	Type A	--	Neg.	Yes	Wound botulism, type A
57 M	Hispanic	Dec.	N	Neg.	Neg.	--	Yes	Unknown

Pos – test was performed and result was positive
Neg – test was performed and result was negative
* Botulinum toxin screen by mouse bio-assay
¶ Botulinum toxin screen by mouse bio-assay; culture for clostridia.

COMMENTS

Botulism testing using the mouse bio-assay is available only in the LAC PHL and state or Centers for Disease Control and Prevention (CDC) laboratories. Antitoxin is available in California only upon release by designated public health physicians in ACDC or the California DHS. For these reasons, reporting of hospitalized cases is felt to be complete. However, under-detection of mild cases is possible.

Botulism is one of seven biological agents classified as “Category A” for bioterrorism preparedness, requiring the highest priority for reporting. Heightened concern over bioterrorism should lead to increased consultations with Public Health for possible botulism cases.

ACUTE *TRYPANOSOMA CRUZI* INFECTION IN ORGAN TRANSPLANT RECIPIENTS IN LOS ANGELES, CALIFORNIA, 2006

BACKGROUND

This report describes two cases of acute Chagas disease in heart transplant recipients at two separate local hospitals in Los Angeles County in February 2006. Chagas disease is a life-long infection caused by the parasite *Trypanosoma cruzi (T. cruzi).* Most infected persons are asymptomatic and undiagnosed. Triatomine (i.e., Reduviid or kissing) bugs transmit the parasite through infected feces. *T. cruzi* may also be transmitted congenitally or by an infected blood transfusion or organ transplantation. Although serologic testing of organ, tissue, and blood donors is performed in areas of Latin America where Chagas disease is endemic, there is no *T. cruzi* screening test licensed in the United States (U.S.). However, seroprevalence studies have documented the presence of *T. cruzi* antibodies in U.S. blood [1] and organ donor populations [2]. In the U.S., there is one previous report of *T. cruzi* transmission through solid organ transplantation where three organ recipients were infected [3].

CASE REPORTS

Case 1:

A 64 year-old male with idiopathic cardiomyopathy received a heart transplant on December 11, 2005. He was treated with enhanced immunosuppression in January 2006 for suspected organ rejection. On February 11, 2006, he was readmitted with anorexia, fever, and diarrhea of two weeks duration. A peripheral blood smear revealed *T. cruzi* trypomastigotes, blood cultures were positive for *T. cruzi*, and endomyocardial biopsy specimens contained amastigotes. The patient was interviewed about natural exposures, and the organ procurement and transplantation records were reviewed. He had no risk factors for pre-existing *T. cruzi* infection. He was seronegative for *T. cruzi* antibodies but positive for *T. cruzi* DNA by polymerase chain reaction (PCR), indicating recent infection. After initiation of nifurtimox therapy, his parasitemia rapidly cleared. However, the patient expired on April 30, 2006 from acute rejection.

To identify the source of infection, a trace-back was conducted on blood products transfused to the organ donor and heart recipient. All available blood donors tested negative for *T. cruzi* antibodies by immunofluorescence assay (IFA) and radioimmunoprecipitation assay (RIPA). The organ donor, who was born in the U.S. but had traveled to a Chagas-endemic area of Mexico, originally tested borderline positive for *T. cruzi* antibodies by IFA. The donor had received multiple blood products prior to his death; therefore it was believed that IFA might not be sensitive enough to pick up *T. cruzi* antibodies. A follow-up test using RIPA was done which confirmed the presence of *T. cruzi* antibodies.

Three additional patients received solid organs from the same donor. These patients remain *T. cruzi*-seronegative by IFA with no evidence of parasitemia by PCR. They continue to be monitored.

Case 2:

A 73 year-old male with ischemic cardiomyopathy received a heart transplant on January 3, 2006. The patient was re-admitted to the hospital on February 22, 2006 with complaints of fever, fatigue, and an abdominal rash. A thin blood smear revealed *T. cruzi* trypomastigotes, and blood cultures were positive for *T. cruzi*. Organ procurement and transplantation records were reviewed. The patient had no risk factors for pre-existing *T. cruzi* infection. He was seronegative and PCR-positive for *T. cruzi*, indicating recent infection.

The patient's rash and parasitemia resolved after 10 days of nifurtimox treatment. He remains hospitalized. Endomyocardial biopsies thus far have not revealed trypanosomes, and he remains seronegative by IFA for *T. cruzi*.

The source of infection was investigated with the same methods used for Case 1. All available blood donors tested seronegative for *T. cruzi*. The organ donor born in El Salvador first tested negative for *T. cruzi* antibodies by IFA but had a follow-up test with RIPA due to the amount of blood products transfused to the organ donor and possible hemodilution. The donor then tested positive for *T. cruzi* antibodies.

There were three other solid organ recipients from the same donor. These patients remain *T. cruzi*-seronegative by IFA with no evidence of parasitemia by PCR. They continue to be monitored.

These are the fourth and fifth cases of reported *T. cruzi* transmission through solid organ transplantation in the U.S. *T. cruzi* prevalence in the U.S. varies by region and may be higher than previously appreciated, especially in Los Angeles County, where a substantial proportion of donors have emigrated from Chagas-endemic countries. Because organ donors are frequently transfused, infection may be transmitted to recipients either by transfusion or transplant. Currently, there are no national policies recommending blood, organ or tissue donor screening for *T. cruzi*. Available diagnostic tests have variable sensitivity and specificity and there is no licensed screening test. However, evaluation of potential serologic tests for blood screening is currently being conducted.

Physicians and laboratorians should maintain an index of suspicion for *T. cruzi* infection in organ transplant recipients who exhibit fever in the absence of obvious opportunistic and bacterial infections. Acute Chagas disease in severely immunocompromised patients is of special concern, because the clinical course is often severe and rapidly progressive. If it is suspected, manual review of blood smears should be performed. Acute Chagas disease should be treated as early as possible in the course of infection with nifurtimox (obtained from the CDC Drug Service, telephone 404-639-3670) and benznidazole (not available in the U.S.).

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RESPONDING TO URGENT CASE REPORTS: TESTING THE LOS ANGELES COUNTY DISEASE REPORTING SYSTEM

Since the 9/11 event and subsequent anthrax attacks, strengthening the ability of Local Public Health Agencies (LPHAs) to detect and respond to bioterrorism as well as natural disease outbreaks has become a national priority. In response to this priority, the Centers for Disease Control and Prevention (CDC) issued guidance that clarified LPHA responsibilities for receiving and responding to urgent disease case reports [1]. This guidance detailed four primary recommendations: 1) a single, well-publicized telephone number to receive urgent case reports; 2) a phone triage system to process urgent case reports; 3) capabilities to receive urgent case reports 24 hours a day, 7 days a week and 4) a trained public health (PH) professional to respond within 30 minutes of receiving the report. Lacking from this guidance was the provision of tools or methods that LPHAs could use to evaluate and test their disease reporting system to identify areas that were working well and areas that needed improvement.

RAND Corporation developed a set of methods that could be used by LPHAs to evaluate their ability to respond to urgent case reports and assess their compliance with CDC recommendations. A pilot study using these methods was conducted by RAND in 2004 using several LPHAs across the country as test subjects. The study methods and results were published in 2005 [2]. Accompanying the report was a technical manual that LPHAs could use to perform similar evaluations of their own disease reporting systems. Using this manual as a guide, an evaluation of the Los Angeles County (LAC) Disease Reporting System was performed in early-2006.

BACKGROUND

Los Angeles County maintains a disease reporting system capable of receiving reports 24 hours a day, 7 days a week via an 888 toll-free disease reporting hotline (Figure 1). In addition to the hotline, urgent disease reports can also be called in directly to Acute Communicable Disease Control Program (ACDC) or Immunization Program (IP).

Calls received through the 888 toll-free number during normal business hours—Monday to Friday, 8am to 5pm—go directly to the LAC Department of Public Health Morbidity Unit. If a caller is requesting information or assistance related to infectious disease the call is transferred to ACDC. Calls are then triaged based on whether the caller is a healthcare provider and the exact nature of the call.

All calls received after-hours—Monday to Friday, 5 p.m. to 8 a.m., weekends, and holidays—are forwarded directly to the County Operator (serves as the answering service for *all* county departments). Healthcare providers with questions related to infectious disease are transferred to the Public Health physician on call (aka Administrator On Duty [AOD]). Public callers, however, are provided with requested information, but not typically transferred to the AOD.

METHODS

The RAND technical manual organizes the evaluation of a disease reporting system into four levels. The Level 1 test is designed to only test how quickly a response to an urgent disease report is received. Subsequent testing levels build on this basic test by evaluating other, more complex aspects of a disease reporting system.

A Level 1 test for LAC was planned for April 2006. Test callers were selected from a Public Health program unrelated to the county disease reporting system. Callers were required to attend a training session that gave an overview of the RAND study, explained the design of the test being conducted in LAC and provided specific training on how to perform test calls. This training included an instructional session as well as an interactive one. Once completed, callers signed up to perform between one to three test calls during the test month.

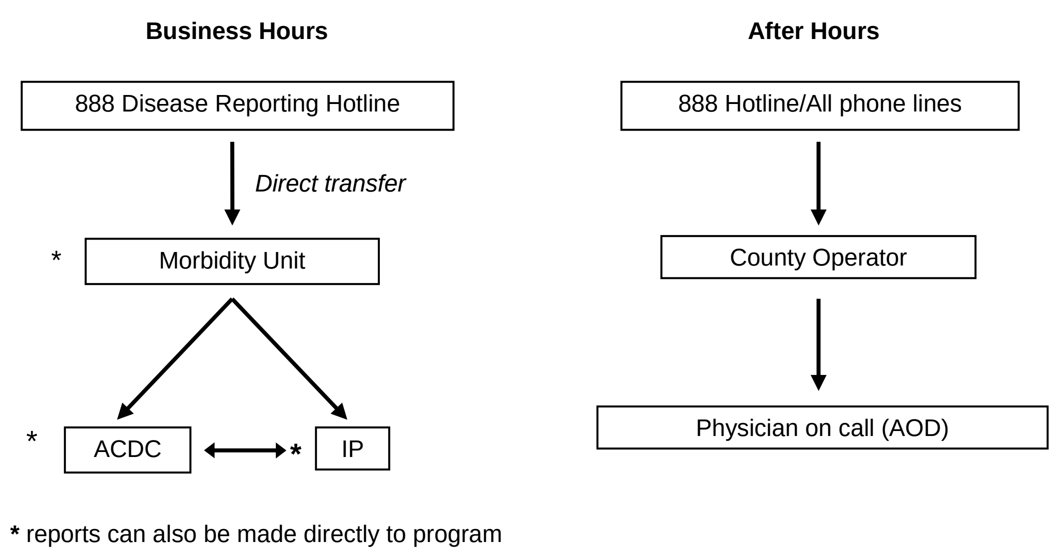
Each call process consisted of three phases: 1) initiating a call, 2) reaching an action officer[1] (AO) and 3) debriefing. A call was initiated when a test caller phoned the disease reporting system, used a lead-in (a short message designed to move the call to an AO) and asked to speak to an AO. The caller would either be transferred directly to the AO (a warm transfer) or be asked to leave a message for the AO (callback). Once the caller reached an AO and confirmed that the person was responsible for handling urgent disease case reports, the AO was "debriefed" (i.e., informed that the call was only a test and that no further action was required).

Test callers received a script to follow for each call initiation that had them pose as a healthcare worker trying to get information regarding a potential case of infectious disease. This disguise prevented the person receiving the call from knowing immediately that the call was a test. During the call, each caller would complete a worksheet to keep track of specific call details such as the exact time the call was initiated, how long the caller was on hold, if the caller reached an AO, whether they had a warm transfer or a callback and how long the entire call took from start to finish. Callers were also encouraged to make notes on anything else of interest that happened during the call.

Information collected during the test calls was used to measure several outcomes—if contact with an AO was made within 30 minutes of call initiation (where contact was treated as a yes/no variable); the time from call initiation to contact with an AO; and the percent of calls with warm transfers as opposed to callbacks.

The test of the disease reporting system was announced to physician staff, but the exact schedule of test calls was kept secret. Dates and times of test calls were varied throughout the month.

Figure 1. Disease Reporting Schematic
(urgent reports)



RESULTS

During the month of April 2006, a total of ten test calls were made to the disease reporting system. Contact with an AO was made within 30 minutes for eight calls (Table 1), while two calls yielded no contact. Response times for successful calls ranged from 4 to 15 minutes with a mean of 8.25 minutes

[1] For purposes of this test, an Action Officer (AO) is defined as a Public Health professional responsible for responding to public health emergencies at the time of the test call.

from initiating the phone call to reaching an AO. Of the eight successful calls, seven (88%) were warm transfers.

Two calls were not able to connect with an AO within the 30 minutes recommended by CDC. In the first call, the caller was transferred to an AO's voicemail instead of being transferred to an alternate AO who was available to speak with the person immediately. The voice outgoing message did, however, leave an alternate number to use in the case of an emergency. The test caller used this number, insisted on speaking with someone and eventually reached an AO within 30 minutes. The initial AO was out of the office for the entire day, although they did return the call the next business day.

The second call was made to the 888 toll-free disease reporting hotline at the end of the business day on a Friday. The phone rang numerous times without being answered and eventually went to a recorded message that asked the caller to "remain on the line for the next available agent". After remaining on hold for 15 minutes, the test caller ended the call. The caller made two additional attempts and was on hold for approximately eight minutes each time. A live person was never reached.

Table 1. Successful Call Line List

Call #	Type of Call	Time of Call	Out-come	Time on hold: County Operator	Time on hold: Morbidity Unit	Time on hold: ACDC/IP	Total Time to reach AO
1	After Hrs	Early Morning	WT	7 min	----	----	9 min
2	Business Hrs	Afternoon	WT	----	0 min	6 min	10 min
3	After Hrs	Late Evening	WT	2 min	----	----	4 min
4	Business Hrs	Afternoon	WT	----	0 min	5 sec	4 min
5	After Hrs	Afternoon	WT	3 min	----	----	5 min
6	After Hrs	Early Evening	CB	6 min	----	----	13 min
7	Business Hrs	Late Morning	WT	----	----	3 min	15 min
8	Business Hrs	Afternoon	WT	----	0 min	4 min	6 min

WT=Warm Transfer; CB=Callback

Improvements: At the end of the test period, call transfer protocols were reviewed with ACDC front office staff. Protocols were developed such that healthcare providers calling about a specific patient would not be forced to leave a message on voicemail, but would be transferred to a live person for assistance. In addition, all staff were encouraged to leave an alternate number on their voicemail so that in an emergency situation, callers have another option for reaching a live person.

Telephone services were contacted and asked to ensure that calls were being appropriately forwarded to the county operator at the conclusion of business hours. It was also clarified that staff must be available to answer phones in all county departments through 5pm on weekdays as the automatic transfer of phone calls to county operator does not occur until 5pm precisely.

DISCUSSION

The test of the LAC disease reporting system showed that the current system works very well. The county already had a system set up to receive reports 24 hours a day, 7 days a week and a toll-free hotline specific for receiving urgent disease case reports. While more than one number for disease reporting does exist, the 888 toll-free number has been well-publicized (e.g., rolodex inserts, phone stickers, pens, etc) by the county and is the number public callers and healthcare providers are given when asked where they can report cases of disease.

Most test calls reached an AO within 15 minutes; well under the 30 minute standard recommended by the CDC. The phone triage system functioned smoothly with most calls being transferred directly to an AO.

Test callers reported back that both Morbidity Unit and ACDC staff were pleasant and professional on the phone. While there were a few problems with the phone numbers, they were resolved quickly with minimal disruption.

Additional testing of the disease reporting system will be conducted over the next one to two years, with each subsequent test increasing in difficulty until the most comprehensive and complex test has been performed. Subsequently, tests varying in difficulty and scope will be conducted annually for quality assurance purposes.

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PEARLS OF SICKNESS: A MULTISTATE EPIDEMIC OF *VIBRIO PARAHÆMOLYTICUS* LINKED TO CONTAMINATED OYSTERS FROM WASHINGTON STATE

BACKGROUND

In June 2006, routine disease surveillance by the Los Angeles County (LAC) Department of Public Health (DPH) Acute Communicable Disease Control (ACDC) Program uncovered a sharp increase in the number of cases of *Vibrio parahæmolyticus* infection. *Vibrio parahæmolyticus (V. parahæmolyticus)* is a species of comma-shaped bacteria that thrives in seawater or brackish water. People commonly become infected with *V. parahæmolyticus* through ingestion of contaminated water or undercooked shellfish. Shellfish include oysters, mussels, clams and scallops. Symptoms of vibriosis include profuse diarrhea, fever, abdominal cramps, nausea, vomiting, headache and severe fatigue. Illness duration extends from 1 to 7 days and incubation ranges between 4 to 30 hours, but usually 12 to 24 hours [1].

The endemic rate for *V. parahæmolyticus* infection is approximately 15 cases per year, with most of those cases occurring between late May and early October [2]. However, starting in mid-June ACDC began to receive more reports of infection than expected. Due to the apparent swell in incidence, ACDC investigated the rising cases of *V. parahæmolyticus* infection and found they coincided with increased incidence of vibriosis in Washington State.

METHODS

<u>General investigation</u>: Cases of *V. parahæmolyticus* are reportable to ACDC and are tracked. For each report received, the physician listed on the report was contacted and interviewed about the case. Medical records such as history and physical, infectious disease consultation and discharge summary were requested from hospitals for hospitalized cases. Cases were interviewed about symptoms and risk factors, particularly consumption of certain seafood items. LAC Environmental Health Services investigated reports in which consumption of raw seafood was implicated.

<u>Case definition</u>: An outbreak case was defined as any person meeting all of the following three criteria:

1. Is a Los Angeles County resident with *V. parahæmolyticus* infection confirmed by the LAC-PHL.
2. Ate raw shellfish harvested from Puget Sound, WA between July 1 and July 20, 2006.
3. Had onset of gastrointestinal symptoms within 72 hours following ingestion of the shellfish..

<u>Environmental Health Inspection:</u> Because many of the vibriosis cases reported eating at restaurants throughout California, multiple environmental health jurisdictions were requested to assist in the investigation. In addition to LAC Environmental Health Services, the following counties participated in the investigation: San Diego, Santa Barbara, Orange County and San Francisco. Each county inspected the restaurants or vendors; confirmed receipt and sales of raw oysters; and copied invoices and shellfish tags to determine the source of the oysters.

RESULTS

<u>Cases</u>: ACDC obtained reports on 14 vibriosis cases infected with *V. parahæmolyticus* and residing in LAC between June 15 and August 15, 2006. Figure 1 shows onset dates for *V. parahæmolyticus* infections from July 1 to August 2; the time frame encompasses the vibriosis epidemic, but also includes endemic cases not related to oyster consumption. Eleven *V. parahæmolyticus* cases (79%) recalled eating raw oysters or scallops, while 3 cases denied eating raw oysters prior to onset of symptoms. Forty-seven percent of cases were male. Cases had a mean age of 48 years with a range of 25 to 86 years.

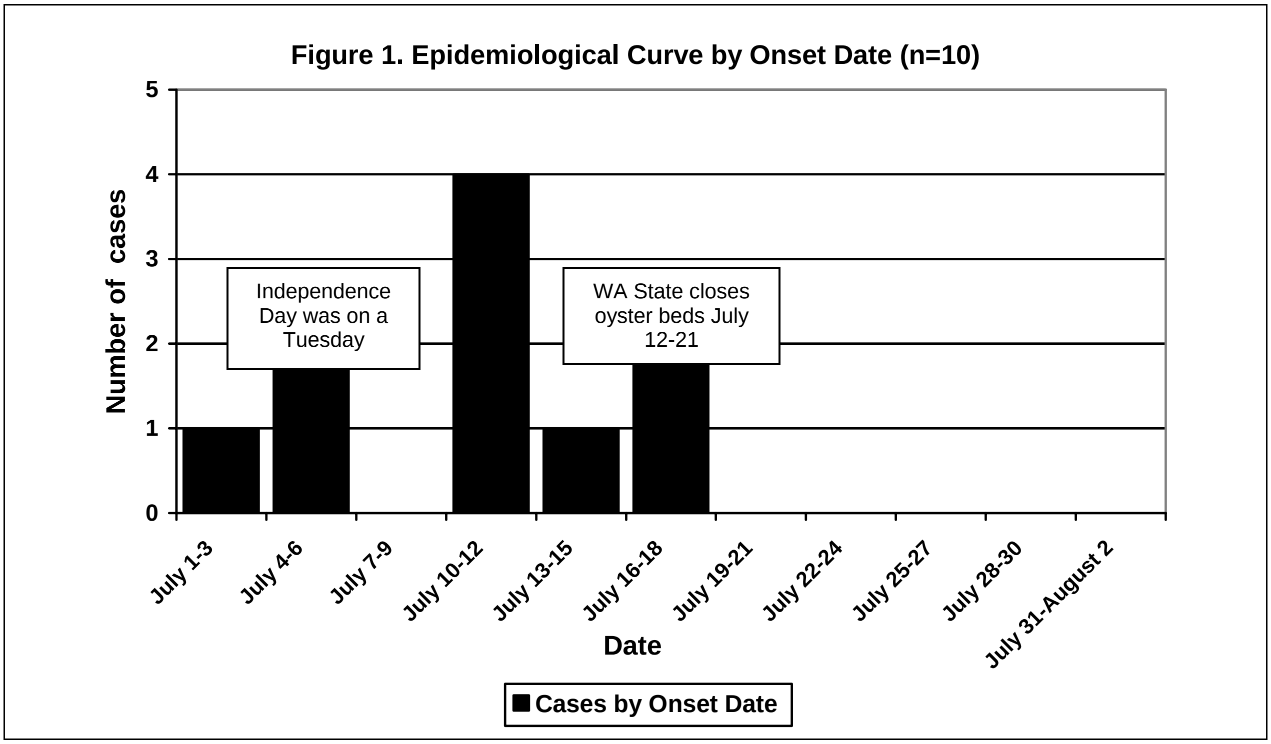

Table 1. Profile of Selected *V. parahæmolyticus* Cases

Age / Sex		Onset date	Implicated seafood	Purchase location
55	F	June 10, 2006	Raw scallops	Los Angeles – restaurant
47	F	July 2, 2006	Raw oysters	San Francisco – Pier 39
31	M	July 4, 2006	Raw oysters	San Diego – restaurant
25	M	July 10, 2006	Raw oysters	Century City – restaurant
48	F	July 10, 2006	Raw oysters	Santa Barbara – Stearns Wharf
43	M	July 10, 2006	Raw oysters	San Francisco – restaurant
34	F	July 14, 2006	Raw oysters	Los Angeles – supermarket
86	M	July 17, 2006	Raw oysters	Sonoma – private retreat
38	F	July 17, 2006	Raw oysters	Los Angeles – restaurant

Table 1 outlines the age, gender and onset date of selected vibriosis cases and shows the implicated seafood item and its point of purchase. The first case in Table 1 typifies LA County seasonal vibriosis case histories; a female case ate scallop ceviche at a Mexican restaurant, although she is not part of the epidemic. The first case linked to the epidemic was a woman who had visited the San Francisco Bay Area over the Independence Day weekend. She reported eating raw oysters at Fisherman's Wharf and subsequently became ill. The second case linked to this epidemic was a man who had traveled to San Diego with friends. He dined on raw oysters at a popular beachfront hotel as part of a large dining party. The third case linked to this epidemic was a young man who had eaten raw oysters at a restaurant in Century City in LAC. He was treated by a private physician and later diagnosed with vibriosis. One case linked to this epidemic became ill following home consumption of raw oysters. She and her partner purchased shucked oysters from a supermarket and ate them raw. Her partner experienced some gastrointestinal symptoms, but was not diagnosed with vibriosis. Three other cases were associated with raw oyster consumption outside of LAC; the remaining cases ate raw oysters at commercial food establishments in LAC.

Laboratory: The LAC PHL, bacteriology unit confirmed 12 cultures positive for *V. parahæmolyticus*. One case each was confirmed by Santa Cruz County and Orange County PHLs.

Environmental Health Investigations: LAC Environmental Health Service Food and Milk Program inspected one restaurant in Century City and one supermarket in Westwood based on case reports. Both establishments had sold raw oysters harvested from various beds in Puget Sound, Washington State.

Santa Barbara County Environmental Health inspected a restaurant on Stearns Wharf and confirmed their oyster supply had been harvested also in Puget Sound, Washington State.

San Francisco Environmental Health could not complete the inspection because the two cases reportedly ate at restaurants on Fisherman's Wharf, but neither could recall which specific restaurant.

San Diego County Environmental Health completed an outbreak investigation based on the report received from ACDC. The vibriosis case reported dining with a large group of friends at a hotel restaurant, and while he was the only person to be diagnosed with vibriosis, several members of his party became ill with similar symptoms following the meal. All of those who were ill reportedly ate raw oysters. The tags for that particular lot of oysters indicated they were harvested, again, from Puget Sound, Washington State.

Halt of Supply: Between July 12 and 21, 2006, the state of Washington issued public warnings and closed several oyster beds in Puget Sound in response to the public health threat. Following the closures of the oyster beds, the number of cases of vibriosis (including all *Vibrio* species) reported in Los Angeles County fell back to endemic levels.

DISCUSSION

After thorough investigation, the LAC DPH determined that the epidemic of vibriosis due to infection with *V. parahæmolyticus* was caused by environmental contamination of oysters harvested from Puget Sound in Washington State. The summer of 2006 was one of the warmest recorded for the United States since 1895 [4]. As a result, water temperatures in Puget Sound were also above normal. *V. parahæmolyticus* tends to thrive in warmer conditions, which led to proliferation of the bacteria in the water. Oysters and other filter-feeding marine life concentrate the bacteria in their bodies, and if the shellfish are not cooked properly, the bacteria may cause illness.

The onset of four cases in LAC coincided with Independence Day celebrations, three of whom reported traveling outside of LAC for the holiday. This is significant because in some cultures oysters are a special occasion food item consumed during celebrations. Two peaks of disease incidence surround July 4, 2006. The holiday occurred on a Tuesday, which led to some people taking an extended weekend before or after the holiday. Several cases who became ill outside the holiday period had eaten oysters as part of other festivities including business meetings, family gatherings, parties and romantic liaisons.

LIMITATIONS

This investigation was limited by a few factors. There was recall bias among some cases due to the retrospective nature of the data collection. Several cases were unable to positively identify the restaurant where they had eaten oysters. The standardized questionnaire administered to the cases asked specifically about seafood and seawater exposure, which may have biased the cases' answers.

Despite the multiple health jurisdictions involved in this epidemic, no additional warnings regarding the consumption of raw oysters and other seafood were officially made in California by either state or local health departments. Epidemiological data related to this outbreak of *V. parahæmolyticus* from LAC and other parts of California were not included in a bulletin posted to the CDC Morbidity and Mortality Weekly Report [3]. Because the source of the contaminated oysters was in Puget Sound, much of the higher-level oversight of the investigation was covered by Washington state authorities.

RECOMMENDATIONS

While it is legal to sell and serve raw oysters in LAC, the DPH recommends that people do not consume raw or undercooked oysters. Current California health codes dictate that commercial food establishments that serve raw oysters originating from the Gulf of Mexico display prominent warnings in both English and Spanish, detailing the health risks associated with raw seafood consumption and sales of oysters from this region are restricted between April 1 and October 31 each year. However, there is no such regulation for oysters taken from other locations or for other raw shellfish.

Also, LAC DPH recommends that clinicians treating patients for profuse diarrhea and other symptoms consistent with vibriosis ask their patients about seafood consumption and recreational water exposure. If patients admit to a recent history of either activity, clinicians should take a stool specimen for culture. Culture for vibriosis species is not done routinely on all stool cultures. Vibriosis (stool) culture must be requested by Doctor's order and the lab has to use TCBS agar.

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2006 HUMAN HANTAVIRUS INFECTIONS IN LOS ANGELES COUNTY RESIDENTS

BACKGROUND

Hantavirus pulmonary syndrome (HPS) was first recognized in 1993 after an outbreak of acute respiratory failure in young people in the Four Corners area of southwestern United States (U.S.). The case fatality rate among these cases was 76% [1]. Fortunately, recent Centers for Disease Control and Prevention (CDC) surveillance data reports a decline in the case-fatality rate of 30 to 40% [2]. HPS is characterized by a febrile illness associated with bilateral diffuse interstitial edema of the lungs developing within 72 hours of hospitalization in a previously healthy person. The causative agent of HPS is Sin Nombre virus, a previously unknown hantavirus that was both documented in individuals with HPS in New Mexico and rodents within their dwellings [1]. Sin Nombre virus (SNV), a lipid enveloped single stranded RNA virus of the family Bunyaviridae, is genetically distinct from other known hantaviruses that cause hemorrhagic fever with renal syndrome in Europe and in Asia [3].

Several hantaviruses that are pathogenic for humans have been identified in the U.S. [3]. In general, each virus has a single primary rodent host. The deer mouse *Peromyscus maniculatus* (*P. maniculatus*) is the primary rodent host and reservoir for Sin Nombre virus (SNV). *P. maniculatus* has been found in almost every state and province in the U.S. and Canada, except in the southeastern U.S. and the Atlantic seaboard.

Hantavirus infection is invariably related to contact with rodent reservoirs, but the duration of contact with infectious materials and dose necessary for disease transmission are not well understood. Most human infection is felt to be acquired through inhalation of aerosolized feces, urine or saliva from the infected mice. Case-control studies have found that the most commonly associated risk of hantavirus infection is contact with rodent excreta. Because most contact with infectious materials results from the ubiquitous presence of rodents, determination of the exact exposure can be difficult. The estimated incubation period has ranged from 1 to 5 weeks [4].

Since HPS was first identified in 1993, the CDC has confirmed 438 cases of HPS reported from 30 states among residents of 32 states through March 2006 [2]. Most cases have been reported in the Southwest especially New Mexico, Colorado, and Arizona. However, 43 cases have been reported to the CDC from California (CA) as well. Most cases in CA have been documented on the CA-Nevada border in the Sierra Nevada mountain range. However, environmental surveillance data has shown *P. maniculatus* serologically positive for SNV infection throughout southern and northern CA. There are usually from 0 to 5 cases documented each year in CA. Although Los Angeles County (LAC) residents have been previously identified with SNV infection, the cases were thought to have acquired infection outside of LAC.

In 2006, two cases of fatal SNV infection were documented in LAC residents. The first case was most likely acquired in Mono County, CA in an area where the human HPS had been previously documented. The second case of HPS was most likely acquired in the Antelope Valley of LAC. Although hantavirus infection had been documented in deer mice from past annual environmental surveillance data in the Antelope Valley, no human cases of HPS had been previously documented.

CASE REPORTS

<u>Case 1</u>: A 52 year-old male with history of hypertension, sleep apnea, and morbid obesity presented to a medical center in Reno, Nevada (NV) with complaints of progressive shortness of breath, wheezing, coughing and increased sputum production for the past three weeks. Prior to seeking care in Reno, this patient had spent one month camping in the family trailer with his wife at Robinson Creek campground, located in the Sierra-Nevada Mountain Range within Mono County, California.

Upon initial evaluation at the medical facility in Reno, the patient was noted to have an O_2 saturation at 90% on 10L of oxygen with a chest radiograph showing hypo-inflation with atelactasis versus infiltrates.

The patient's admitting diagnoses included: chronic obstructive pulmonary disease, pneumonia, and rule-out myocardial infarction. Within five days of his hospitalization, it was apparent that both his renal and pulmonary functions were deteriorating. He developed renal failure, creatinine of 2.4 mg/dl with hematuria and also required mechanical ventilation. Additionally, he developed thrombocytopenia with a platelet count of 61,000. The patient was placed on broad spectrum antibiotics initially and cared for in the intensive care unit. In addition to routine blood, urine, and sputum culture, additional infectious disease work-up was requested to include: hantavirus serological testing, blood smears for *Borrelia* sp., urinary *Legionella* antigen, and serological testing for West Nile virus and Rocky Mountain Spotted Fever. All blood, urine, and sputum cultures showed no growth and serological testing was negative with the exception of hantavirus serology which was strongly positive. Acute hantavirus (SNV specific) titers initially performed at the State of Nevada Public Health Laboratory were notable for an IgG $\geq$ 1:6400 and IgM $\geq$ 1:6400, consistent with acute hantavirus pulmonary syndrome. No additional convalescent hantavirus titers were obtained on this patient. These serological results were additionally confirmed at State of California Viral and Ricketsial Disease Laboratory (VRDL). The patient ultimately died of fulminant respiratory failure within 11 days of admission. No autopsy was conducted on this case.

Case 2: On July 22, 2006, a previously healthy sixteen year-old Hispanic male was initially seen at an outpatient clinic with a one-day history of high fever and headache. His evaluation consisted of blood cultures, complete blood count, blood chemistries and a computerized tomography head scan. All tests were normal and the patient was discharged home. The patient was seen again on July 24, 2006 due to persistent fevers, progressive shortness of breath, and severe headache. His evaluation revealed a bilateral pneumonia, thrombocytopenia, elevated hemoglobin and severe hypotension. He was subsequently admitted to an inpatient medical center with a diagnosis of sepsis and pneumonia and later requiring mechanical ventilation. On July 26, 2006, he was seen by an infectious disease specialist who placed him on broad spectrum antibiotics and also treated with a new "sepsis" drug (Xigris). The working diagnoses included: adult respiratory distress syndrome (ARDS), septic shock, and rule out meningitis. A spinal tap was not performed due to low platelet counts. On July 26, 2006, the infection control practitioner from the inpatient medical center reported the case as an "unusual occurrence" to the Los Angeles County Department of Public Health Acute Communicable Disease Control (ACDC) Program. After reviewing the medical chart and additional discussion with the infectious disease specialist on this case, additional infectious disease work-up was recommended which included testing for: WNV, HIV, Hantavirus, and urinary *Legionella* antigen. All labs were unrevealing including blood cultures, serologic testing for WNV, HIV, other viral pathogens, as well as *Legionella*. The patient's serum specimen was sent to the state of California VRDL and acute serological results were strongly consistent with acute SNV infection with IgM $\geq$ 1:1600 and IgG $\geq$ 1:6400. Despite aggressive medical care, the patient died 19 days after his admission to the medical center.

During the six week period before the onset of his illness the patient completed his junior year of high school, and worked at a nearby fast food restaurant. He had quit his job at a local fast food restaurant the first week in July. His parents could not recall the patient complaining of seeing with rodent dropping or rats or mice during his time working. However, during the first week in July, his last days on the job were spent cleaning the store room behind the kitchen. Other summer activities included rabbit hunting, visits to a regional park and odd jobs gardening and painting were confined to the Antelope Valley. During this period he resided with his family in a mobile home and at a nearby friend's home, where no infestations of rodents were reported.

METHODS

Medical chart review was completed on the two reported cases of suspected HPS. The family of Case #2 was extensively interviewed by a physician from ACDC and investigators from the LAC Environmental Health Vector-borne Disease Surveillance Unit for possible sources of exposure to hantavirus. Serological testing for both human and rodents for hantavirus infection was conducted at the State of CA VDRL using ELISA methodology.

Case Definition: A confirmed case of HPS is a febrile illness characterized by bilateral interstitial pulmonary infiltrates and respiratory compromise usually requiring supplemental oxygen and clinically resembling acute respiratory disease syndrome (ARDS). The typical prodrome consists of fever, chills, myalgia, headache, and gastrointestinal symptoms. Typical clinical laboratory findings include hemoconcentration, left shift in the white blood cell count, neutrophilic leukocytosis, thrombocytopenia, and circulating immunoblasts [5].

The appropriate laboratory criteria for diagnosis are:

- detection of hantavirus-specific immunoglobulin M or rising titers of hantavirus-specific immunoglobulin G, or
- detection of hantavirus-specific ribonucleic acid sequence by polymerase chain reaction (PCR) in clinical specimens, or
- detection of hantavirus antigen by immunohistochemistry

A field investigation was conducted by California Department of Health Services (CDHS) Vector-borne Disease Section to determine the source of hantavirus-infected deer mice surrounding infection in Case #1. The LAC Environmental Health Vector-borne Disease Surveillance Unit conducted rodent trapping in multiple locations within the Antelope Valley that Case #2 frequented. Investigation consisted of trapping of rodents and obtaining serum from deer mice, *P. maniculatus*, and completing serological testing and PCR testing for hantavirus infection.

RESULTS

Human Serological Results: Only acute serological evaluations were obtained from both Case #1 and #2 during their initial evaluation. Both cases had strongly positive acute IgM and IgG consistent with recent hantavirus (SNV-specific) infection. The first case was found to have an IgM $\geq$ 1:6400 and IgG $\geq$ 1:6400 and the second had an IgM $\geq$ 1:1600 and IgG $\geq$ 1:6400.

Autopsy Findings and RT- PCR Evaluation: No autopsy was performed on Case #1. Autopsy on Case #2 revealed severe pulmonary edema consistent with ARDS and cerebral edema. Both pulmonary and renal tissues obtained at autopsy did not reveal hantavirus RNA upon RT-PCR.

Environmental Health Investigation

Case #1: On August 3, 2006 staff of the CDHS Vector-borne Disease Section (VBDS) initiated a site inspection and rodent surveillance at Robinson Creek Campground in Mono County. The first stop was the Bridgeport District Ranger Station on August 3. VBDS staff consulted with the District Ranger and campground hosts and informed them of the purpose of the visit. Several campground visitors expressed concern to VBDS staff about rodents that they had observed entering their campers or recreational vehicles. VBDS staff observed fresh mouse droppings under the sofa-bed of one guest's vehicle. VBDS staff offered safety tips on avoiding exposure to SNV to many campers.

Ninety folding traps were set at several campsites and inside buildings. A total of 81 rodents were collected. Serum specimens for SNV serologic testing were collected from 41 deer mice (*P. maniculatus*) and 2 mountain voles (*Microtus montanus*). Seven of the deer mice were trapped within the two campsites the case-patient occupied. Serum antibodies to SNV were detected in 19 of 41 deer mice and 1 of 2 voles, including 4 of the 7 trapped from the case-patient's campsites.

Case #2: On August 14-16, 2006, staff of the LAC Vector-borne Disease Surveillance Unit (VBDSU) visited the case-patient's residence to conduct visual evaluation and rodent surveillance. No rodents were captured in 20 traps set over-night at the patient's residence. Eight rodents, including three *P. maniculatus*, were captured in 53 traps set nearby at a friend's residence. Visual inspection of the friend's mother's worksite, and a regional park where the patient reportedly frequented revealed no evidence of rodent activity. Visual evaluation of the patient's worksite demonstrated nine surrounding habitats conducive to deer mouse presence.

LAC VBDSU conducted a second round of rodent surveillance on August 21-23, 2006. Six rodents, including one *Peromyscus* sp., were collected in 40 traps set in a field near the friend's residence. Two rodents, including one *Peromyscus* sp., were captured in 10 traps set near the patient's work site. No rodents were collected in 20 and 22 traps set at the patient's residential mobile home park and school, respectively.

Serum specimens from five *Peromyscus* sp. were collected and tested at VDRL. Only one of the five collected specimens was positive for hantavirus. The positive specimen was collected in a field adjacent to the residence of the patient's friend and was also found to positive for hantavirus by RT-PCR evaluation of pulmonary tissue of the deer mouse.

DISCUSSION AND PREVENTION

In 2006, two cases of hantavirus infection were confirmed in LAC residents. Both cases had onset dates in late spring and summer which is the usual peak period for hantavirus infection. The first case, most likely acquired in hantavirus exposure in Mono County, while the second case probably acquired infection within the Antelope Valley. Previous CA hantavirus pulmonary syndrome cases have been documented to have been acquired in Mono County, CA, however, this is the first time that human hantavirus infection has ever been documented within LAC.

The first case was documented in a known endemic area for hantavirus infection in the CA- Nevada border in the Sierra-Nevada Mountain Range. We can speculate that with the first case, exposure was probably peridomestic, likely associated with live deer mice and their excreta during a camping trip at Robinson Creek campground in Mono County. The second case was most likely acquired in the Antelope Valley area of LAC. Exposure probably occurred from rodents located at the patient's friends' residence. Field surveillance data documented one of five trapped deer mice (*Peromyscus* sp) had been infected with hantavirus by both serological and PCR testing. Although only one deer mouse was trapped that was positive after an extensive investigation, it is very possible that exposure could have been 4 to 8 weeks prior to the field investigation when the infected deer mouse population was at a much higher level.

Unfortunately, both cases were fatal. There is still no established antiviral therapy that has proven effective in the treatment of HPS. Treatment remains supportive with aggressive management in the intensive care unit with ventilator support and fluid management and use of ionotropic pressers agents as needed. Therefore, prevention of hantavirus exposure is critical. The best available approach to disease control and prevention is risk reduction through environmental modification and hygiene practices that deter rodents from colonizing the home and work environment, as well as safe cleanup of rodent waste and nesting material. Rodent control in and around the home remains the primary strategy in preventing hantavirus infection by undertaking such measures as keeping food and water covered and stored in rodent-proof container and keeping pet food and trash in rodent-proof containers. Additionally, various precautions outside the dwelling include disposing of trash, placing woodpile and stack of lumber at least 100 feet from the dwelling, and removing excess brush and shrubbery close to the home. Making homes rodent-proof is also an important preventive strategy. All gaps and holes inside and out of the home $\geq$¼ inch should be sealed. Gaps and holes are common around windows and doors and between the foundation of the home and ground. Further guidance to workers, campers and hikers with frequent exposure to rodents can be found in a recently MMWR devoted to HPS risk reduction [6].

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TRANSESOPHAGEAL ECHOCARDIOGRAPHY, INSUFFICIENT CLEANING PRACTICES AND LAX EQUIPMENT MAINTENANCE, AND *ESCHERICHIA COLI* - A BREAKDOWN IN INFECTION CONTROL

INTRODUCTION

Escherichia coli (*E. coli*) is a rod-shaped, gram-negative bacillus normally found in the lower gastrointestinal tract and is part of the normal intestinal flora. In hospital settings, *E. coli* most commonly causes urinary tract infections. Respiratory tract infections due to *E. coli* are uncommon, though there have been several published reports that chronicle *E. coli* pneumonia in the pediatric intensive care unit (ICU) [1]. Outbreaks of respiratory tract infections with gram-negative organisms have been increasingly reported due to contamination of medical equipment including bronchoscopes which are directly inserted into the respiratory tract. Transesophageal echocardiography (TEE) is normally done by inserting the instrument into the gastrointestinal tract (the esophagus) and is used during cardiac surgery to better visualize the posterior of the heart. The gastrointestinal tract is considered “dirty” and medical equipment should receive high-level disinfection.

On May 30, 2006, Los Angeles County (LAC) Department of Public Health, Acute Communicable Disease Control (ACDC) Program received a report from the hospital infection control professional that nine cardiac surgery patients were culture positive (blood or sputum) with *E. coli* infections that occurred in early May 2006. The positive cultures occurred from 1 to 4 days after surgery. This report describes the ensuing investigational study to determine the source of the outbreak.

METHODS

<u>Setting</u>: The study was conducted in a 370-bed acute care hospital in LAC which specializes in cardiology and orthopedic care.

<u>Cohort Study</u>: This was a hospital-based cohort study of individuals who underwent valve replacement, coronary artery bypass graft (CABG), both or any other cardiac procedure in May 2006. During this period, a total of 26 cardiac procedures were performed.

Cases were defined as patients who had a cardiac procedure in May that tested positive for *E. coli* within seven days of the procedure and had either a matching pulsed-field gel electrophoresis (PFGE) pattern or matching antibiotic susceptibility pattern. Controls were defined as patients who had a cardiac surgery procedure in May and did not test positive for *E. coli*.

A standardized chart abstraction tool was developed to collect information on demographics; culture results; pre-operative, operative, and post-operative procedures; surgical staff, medications, bed location, and ICU staff during and after the operation until the first positive culture for *E. coli* (cases) or for four days after surgery (controls).

The antibiotic susceptibility profiles of the *E. coli* infections in the cases were reviewed. Susceptibility to amikacin, cefazolin, cefepime, cefotaxime, ceftazidime, imipenem, nitrofurantoin and piperacillin/tazobactam was tabulated.

<u>Environmental</u>: Environmental surveillance cultures of the cardio-vascular ICU (CVICU) were obtained by hospital infection control staff from May 26 to June 2, 2006 and by Public Health staff. Cultures of the TEE equipment were obtained by hospital staff and LAC Public Health Laboratory (PHL) staff.

<u>Laboratory Investigation</u>: Available *E. coli* isolates from cardiac surgery patients and from environmental surveillance were submitted to the LAC PHL for microbiological analysis.

The LAC PHL completed PFGE analysis on *E. coli* clinical (case and control) and environmental isolates. PFGE was performed using the standardized methods of the PulseNet USA protocol [2]. PFGE pattern comparisons were performed visually and using BioNumerics software, version 4.0 (Applied Maths, Belgium).

Statistical Analysis: Data were analyzed using SAS, version 9.1 (Statistical Analysis Software, Cary, NC). Logistic regression was used to generate relative risks (RRs) and corresponding 95% confidence intervals (CIs) to evaluate potential risk factors. χ^2 test was used to compare groups while Fisher's exact test was used when appropriate. The mean surgery time was calculated and compared between cases and controls. A two-tailed *P* value of 0.05 or less was considered statistically significant.

Infection Control Measures/Investigation of Implicated Re-useable Medical Device: After the first site visit on May 31, 2006, ACDC issued interim recommendations including adding antibiotic coverage from gram-negative organisms for cardiac surgery patients, collecting surveillance cultures (sputum) on all intubated CVICU patients, collecting environmental cultures, and culturing the TEE equipment and removing it from use. CVICU and operating room procedures, infection control standards, and procedures for cleaning the TEE equipment were all assessed. When not in use, the TEE probe is stored in a closed case on top of the refrigerator in the cleaning room of the CV operating room (CVOR) office. The TEE equipment was visually inspected and the manufacturer was contacted regarding routine maintenance provided.

RESULTS

Cohort study: Of the nine case-patients seven had positive sputum cultures, one had a positive blood culture, and one had both a positive sputum and blood culture for *E. coli*. All the cultures occurred 1 to 4 days after surgery. All were treated with antibiotics after positive culture.

The distribution of ages and gender was similar between cases and control (Table 1). However, more controls were at home prior to surgery, had elective surgery than cases (Table 1), and did not have valve replacements. Cases also had a longer mean duration of surgery time (p=0.06) (Table 3).

Table 1. Characteristics of Post-Cardiac Surgery Patients with *Escherichia coli* Infection (Cases) versus without (Controls)

Variable	Cases (n=9)		Controls (n=17)		p-value
	n	(%)	n	(%)	
Age					
<50	-	-	1	5.9	0.1319
50-59	3	33.3	4	23.5	
60-69	2	22.2	6	35.3	
70-79	3	33.3	4	23.5	
80+	1	11.1	2	11.8	
Sex					
Male	4	44.4	13	76.5	0.1167
Female	5	55.6	4	23.5	
Prior Surgery Location					
Home	-	-	4	23.5	<0.0001
Ward	5	55.6	10	5.8	
Emergency Room	1	11.1	2	11.8	
Intensive Care Unit	2	22.2	-	-	
Other	1	11.1	1	5.9	
Procedure Type					
Valve	1	11.1	3	17.7	0.014
CABG	4	44.4	9	52.9	
Valve + CABG	2	22.2	-	-	
Other	2	22.2	5	29.4	
Status					
Urgent	2	22.2	3	17.6	<0.0001
Emergent	1	11.1	-	-	
Elective	4	44.4	12	70.6	
Other	-	-	1	5.9	
Missing	2	22.2	1	5.9	

* Values may not add up to totals due to missing values.

Table 3. Comparison of Procedure Duration for Cases and Controls

Procedure Duration	Cases	Controls	p-value
Mean (minutes)	351.4	270.8	0.055
Median	343	297	
Range	(300,455)	(75,414)	

Data for potential risk factors collected for cases and controls was analyzed to yield RRs and 95% CI (Table 2). None of the analyzed risk factors were statistically significant. Surgical staff, including surgeons, assistants, anesthesiologists, nurses, perfusionists, respiratory therapists and CVICU nurses were also analyzed, but no particular staff member emerged as a source of the infection. Pharmacy data for cases and controls was also analyzed, but did not yield a medication that may potentially be associated with the infection.

Table 2. Risk Ratios and Corresponding 95% Confidence Intervals (CI) For Potential Risk Factors for Patients with *Escherichia coli* Infection (Cases) versus without (Controls)

Risk Factor	RR	95% CI	P-value
Procedure Type			
Valve+CABG*	2.40	0.18,32.9	0.5122
CABG	1.33	0.10,17.1	0.8253
Valve	Referent	-	-
TEE			
Yes	0.47	0.08,2.6	0.3905
No	Referent	-	-
Bronchoscopy			
Yes	1.07	0.08,13.9	0.9579
No	Referent	-	-
OR Room			
14	1.80	0.29,11.2	0.3905
12	Referent	-	-
Surgery Status			
Urgent or emergent	3.00	0.42,21.3	0.2720
Elective	Referent	-	-
Vancomycin			
Yes	2.15	0.2,23.2	0.5268
No	Referent	-	-
TEE Post Surgery			
Yes	0.72	0.06,8.5	0.7956
No	Referent	-	-

* Includes "Other" category

<u>Environmental Cultures</u>: Twenty-three environmental cultures were collected by hospital staff, including the TEE probe, which was cultured on June 2 and again on June 8. The TEE probe tested positive for Klebsiella pneumoniae on June 2 and tested positive for *E. coli* on June 8. Four additional cultures were taken from the TEE probe, TEE gel, gel cap, and outside of the cap by PHL staff. All samples were sent to the PHL. All environmental cultures were negative for *E. coli* except for the TEE probe.

<u>Laboratory</u>: Thirteen clinical specimens (from *E. coli* positive CVICU patients in May and June) and one environmental specimen (TEE) were submitted to the PHL for PFGE testing. PFGE was performed using the standardized methods of the PulseNet USA protocol [2]. PFGE pattern comparisons were performed visually and using BioNumerics software, version 4.0 (Applied Maths, Belgium). Strain typing analysis revealed that three patient isolates and one infection control isolate (TEE) had an indistinguishable PFGE pattern with *Xba*I and *Bln*I enzymes. Three patient isolates were subtypes of the predominant strain type, differing by a total of one to four bands, and six isolates had band differences of $\geq$7, indicating that these six are not part of the outbreak [3].

<u>Infection Control Review</u>: The hospital had one TEE probe dedicated to the two cardiac surgery operating rooms. Cardiac surgery patients regularly had the TEE inserted at the beginning of a procedure and the scope remained inserted for the entire duration. The TEE probe was cleaned between each patient with disinfectant and recorded; however, incorrect recording and poor disinfection technique was observed. Visual inspection revealed cracks in the ring of the TEE (Figure 1, 2). The TEE probe was removed from patient care and returned to the manufacturer.

Figure 1.

Figure 2.

DISCUSSION

Reports of *E.coli* infections acquired in hospitals are typically described in the context of urinary tract [3] or ventilator-associated infections [4]. Respiratory tract infections due to *E. coli* are uncommon.

Here, a hospital outbreak of *E. coli* respiratory infections among post-cardiac patients due to a reusable medical device, the TEE probe, was described. After an extensive literature search, this is the only other outbreak due to the TEE equipment that could be gathered.

The TEE equipment is used to visualize the posterior aspect of the heart during cardiac surgery. Professional organizations, medical equipment manufacturers and disinfectant manufacturers all provide instructions on the cleaning and disinfection of these items. Reprocessing of flexible endoscopes is standard practice in many health care settings, and the appropriate cleaning, disinfection, storage and maintenance of these devices can be a lengthy and complicated process. Frequently, endoscopes have been linked to nosocomial outbreaks [5-7].

It is the responsibility of the facility to ensure that reusable medical devices are properly cleaned and disinfected prior to each patient use. In addition, staff must be trained (and retrained) in the proper use, cleaning, storage and maintenance of the device. Staff knowledge is crucial to the infection control bottom line, and annual competency should be documented.

It is critical that reusable medical devices are properly cleaned prior to disinfection. Rutala and Weber reference the Spaulding classification for reusable medical items as critical, semi-critical and non-critical on the basis of the degree of risk of infection [8]. The TEE equipment is considered a semi-critical item since it is in contact with mucous membranes, and high level disinfection using chemical disinfectants is the minimum requirement. Prior to disinfection, the item should be rinsed with sterile water, filtered water, or tap water, followed by an alcohol rinse. The item should be thoroughly dried prior to storage.

The hospital has a policy and procedure "Cleaning TEE Transducer" outlining the appropriate cleaning principles such as "…the transducer must be cleaned and inspected before and after each transesophageal echocardiography examination…should be inspected for perforations or tears in the outer casing…".

The TEE equipment consists of a transducer probe and a motor housing with articulation knobs followed by a cable ending at the connector. The probe is covered by a hard, black, smooth plastic with depth markings. The CVOR transducer showed visible fraying and deterioration in the area surrounding the outer aspect of the transducer probe neck, and fraying with a white string protruding from the inner aspect.

ACDC was initially told that the TEE is inspected quarterly on-site by the manufacturers' representative. However, the hospital was unable to provide documentation of the manufacturers' quarterly maintenance.

The Centers for Disease Control and Prevention (CDC) Guidelines for Environmental Infection Control in Health Care Facilities Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC) 2003 advises that "manufacturers should provide care and maintenance instructions specific to their equipment" [9].

After reviewing the literature, other than 1 report of 2 cases of *Legionella* after TEE, no other report of respiratory, or other infections, associated with TEE was found. Nosocomial infections in ICUs are almost always associated with the use of an invasive device [1]. Richards et al. found that infections at three major sites represented 68% of all reported infection (primary bloodstream, 28%; pneumonia, 21%; and UTIs, 15%); 84% of all episodes of nosocomial pneumonia were related to mechanical ventilation [1]. In another study, device-related sources were responsible for 43% of all hospital-acquired bacteremia [10].

In the analysis of the data, no one particular factor emerged as a probable risk factor. This was surprising, since after obtaining the PFGE results, which implicated the TEE probe as the point source, it was expected to be confirmed by the statistical analysis. A possible explanation may be that the results of the analysis depend solely on the quality of the data. Because of the busy nature of the OR and the many surgical procedures, procedures such as TEE may not be documented and recorded in patient medical charts. As a result, upon chart review, data may be inaccurate and may thus reflect in the final analysis. Since PFGE is the gold standard method and has high reproducibility and discriminatory power [11], the interpretation relied on the PFGE results, which were used for the typing of *E. coli* isolates.

The TEE probe was implicated as the cause of this outbreak due to multiple reasons, including the matching PFGE isolates from the TEE and the cardiac patients exposed to the TEE, the epidemiology of *E. coli* infection in the cardiac patients, the cracked surface of the TEE which would have allowed safe harbor for bacteria even during disinfection, and the correlation between exposure duration to TEE and the increased likelihood of *E.coli* infection.

Interestingly, though post-cardiac surgery patients began developing *E. coli* infections in the beginning of the year at this facility, the PFGE only showed that half of the patients with the same antibiotic resistance profile had the same PFGE. Additionally, two patients had one strain that matched exactly the outbreak strain and another isolate that differed by two bands. This may be attributed to multiple strains of *E. coli* that survived on the TEE; however, there were only one culture because the TEE was removed from use and cleaned by the time it was cultured.

Other notable findings include the rapidity of the *E. coli* growth; many patients were positive within a day of surgery. However, it is still not clear how the bacteria migrated from the esophagus or oropharynx to the trachea/bronchi given that the patients were intubated during the time that the TEE was in the patient and for those who remained intubated, there should have been a sufficient seal with the TEE to block the spread of oropharyngeal flora to the lungs. For those who were extubated, it is possible that their oropharynx was so contaminated by the bacteria with the TEE passing through their mouth that it was able to gain access to their lungs.

This study has several limitations. As previously mentioned, the quality of a study depends on the accuracy of its data. Selective survival bias may also exist in this study. The longer surgery time might be a function of the emergent nature of the surgeries for the case patients, who might have been more likely to have surgery after ICU stay, resulting in an increased susceptibility to *E. coli* infection.

This study highlights the importance of a close relationship between hospitals and their local health departments. ACDC was notified of the outbreak by an astute hospital infection control practitioner. Due to complete cooperation and frequent communication, the point source of the outbreak was quickly identified and suggested control measures were implemented, thereby preventing additional infections. This study also demonstrates the necessity for hospitals to maintain better surveillance, especially in this case where *E. coli* infections are unusual in cardiac surgery patients. It is also necessary for hospitals to review infection control policies and procedures for “semi-critical” equipment, since such equipment has been linked to outbreaks of extended-spectrum beta-lactamase, hepatitis B and C [12,13]. Lastly, hospitals need to examine their equipment for deterioration per the manufacturers’ recommendations and hospital policy. In fact, once the TEE was identified as the source of the outbreak, the hospital visually inspected other scopes at the facility and found that some had evidence of erosion that had not been reported previously and were removed from patient use.

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A CASE-CONTROL STUDY ON RISK FACTORS OF SKILLED NURSING FACILITIES FOR NOROVIRUS OUTBREAKS IN LOS ANGELES COUNTY, 1999–2005

BACKGROUND

Los Angeles County (LAC) experienced an upsurge in the number of norovirus (NV) outbreaks reported in skilled nursing facilities (SNFs) from 2002 to 2004 [1]. Noroviruses (also called caliciviruses and Norwalk-like viruses) are small, round, relatively hardy single-stranded RNA viruses that are estimated to cause 23 million cases of acute gastroenteritis and 50% of all foodborne outbreaks in the United States each year [2,3]. Although humans are its only reservoir, NV is highly contagious with an estimated infectious dose of 10 to 100 viral particles [2-4]. Outbreaks thus tend to occur in institutions and in crowded settings that facilitate person-to-person or fecal-oral transmission, such as in schools, restaurants, and nursing homes [1].

The steadily aging population, and a marked rise in the popularity of alternative living arrangements such as assisted living and continuing care retirement facilities among the healthy elderly in the past 10 to 15 years, suggest that the resident populations of nursing homes are comprised of older and sicker individuals than ever before [5,6]. Although the distinction is not always made, SNFs differ from traditional nursing homes in that SNF residents in general endure more severe health complications that necessitate more intense medical care and equipment. Although NV is not typically fatal, the symptoms—vomiting, diarrhea, stomach cramps, fever, and nausea—for as long as 24 to 60 hours may cause serious health complications for those within the already medically compromised SNF resident population [3].

While there is little to no documentation in the literature on predictors of NV in institutionalized settings, one study of respiratory and gastrointestinal (GI) illness outbreaks in New York State SNFs has suggested that facility size, staffing patterns, and employee sick leave policies are important predictors for NV outbreaks [7]. This study aims to examine these NV outbreak-associated factors in addition to staff-resident and resident-resident interactions in order to help determine possible prevention measures which may reduce susceptibility to NV outbreaks among LAC SNFs.

METHODS

SNFs in which a GI outbreak had occurred from July 1, 1999 to June 30, 2005 were identified by using the Visual Confidential Morbidity Report (VCMR) database of the Los Angeles County (LAC) Department of Public Health (DPH). Data from 1999 were used because this was when the Public Health Laboratory (PHL) initiated the usage of reverse transcriptase polymerase chain reaction (RT-PCR) techniques to test stool specimen samples for NV [3]. Missing data and data discrepancies were resolved using archived outbreak investigation paper records such as epidemiology forms completed by public health nurses (PHNs) and laboratory reports. Each SNF eligible for the study was checked against an established LAC DPH Acute Communicable Disease Control (ACDC) Program Hospital Outreach Unit database to ensure that the facility contained or was entirely a SNF.

Classification of case and control SNFs depended on outbreak definitions. A “NV confirmed” GI disease outbreak had at least one LAC PHL-confirmed stool sample positive for NV. A GI disease outbreak was “NV probable” if lab specimens were either not collected or found negative for NV, but the investigating PHN still implicated NV because of poor stool specimen quality and because the symptoms, duration, and incubation periods were consistent with NV. These criteria reflect that the viral loads in stool samples from infected individuals are not consistently detectable. SNFs with at least one confirmed or probable NV outbreak during the study period formed the case SNF population. Only SNFs that had participated in the reporting process at least once, for non-GI outbreaks, were selected as controls, reducing the possibility of including SNFs that had actually experienced but neglected to report NV outbreaks. One control SNF for each case SNF was cumulatively sampled from the pool of eligible control SNFs.

Introductory solicitation for study participation was conducted by telephone. An introduction letter comprised of a description of NV and of the study, along with a twenty-five question survey was mailed or faxed to each SNF that had agreed to participate. The questionnaire centered on: 1) facility, 2) staff, and 3) resident characteristics, as well as 4) infection control practices. While changes in any of these SNF characteristics would not necessarily guarantee that a NV outbreak would occur, they might affect the chance that NV is brought into the SNF and also, once introduced, the chance that the virus would gain a foothold and cause an outbreak.

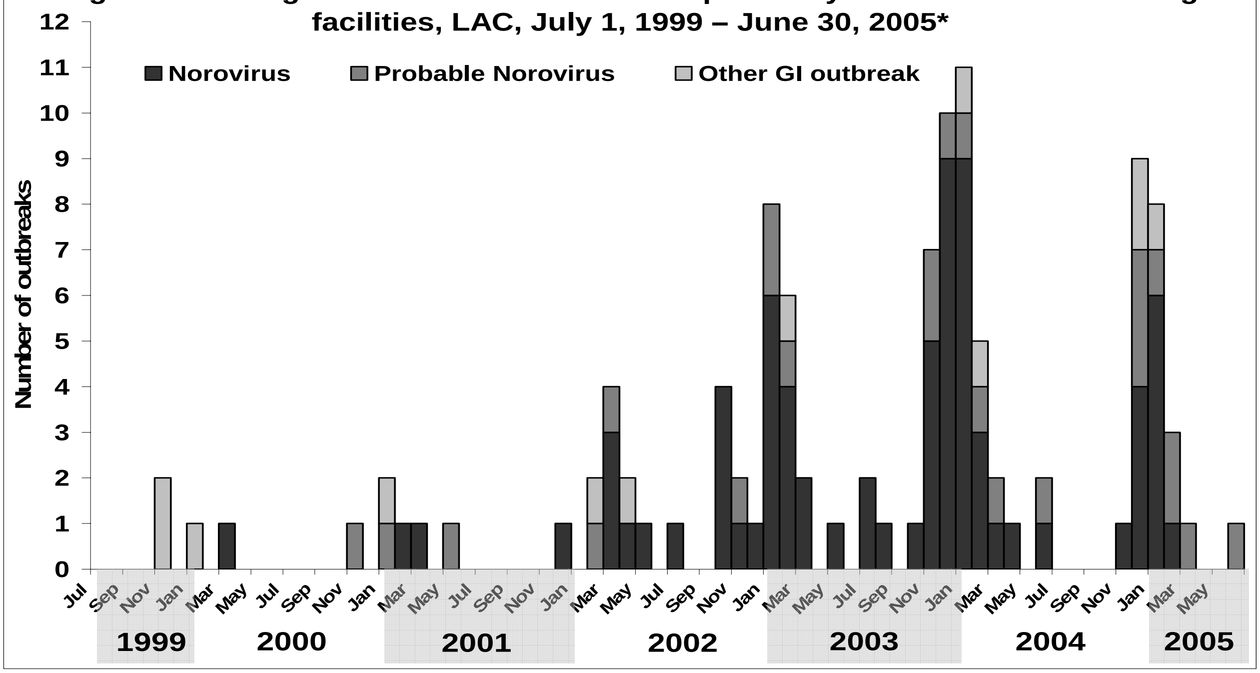
Questionnaires sent to case SNFs were pertinent to the month prior to their last reported NV outbreak, which in this study is referred to as the “surveyed month,” to capture the characteristics that may have precipitated the NV outbreaks in case SNFs. The “surveyed year” refers to the time period from July 1 to June 30, between which the corresponding surveyed month falls. Each control SNF was randomly assigned a month/year of interest to which its survey would pertain, such that the month/year would correspond to the surveyed month of a case SNF.

Questionnaires were administered and collected over a six-month period. Microsoft Access was used for database management. All data analysis was conducted using SAS Version 9.1. Continuous variables with non-normal distributions were analyzed for differences between case and control SNFs using the non-parametric Mann-Whitney Rank test, and continuous proportion variables were tested by computing z-statistics for tests of proportions. A statistical model was also created, based on the hypothesis that increasing the level of staff-resident and resident-resident mixing would increase the chance of a reported NV outbreak. Odds ratios and 95% confidence intervals (95% CI) were calculated for the predictors in this model, with case or control status as the outcome of interest, using logistic regression.

RESULTS

As of August 2005, there were 417 licensed LAC SNFs in the LACDPH ACDC Hospital Outreach Unit database. Of the 113 GI disease outbreaks reported among these LAC SNFs during July 1, 1999 to June 30, 2005, 75 of these were classified as confirmed NV outbreaks, 24 as probable NV outbreaks, and 14 were attributed to other types of GI diseases (Figure 1). Ninety-nine confirmed or probable NV outbreaks occurred in 76 SNFs. Of the 76 SNFs that reported at least one confirmed or probable NV outbreak during the study period, 74% experienced onsets after July 1, 2003.

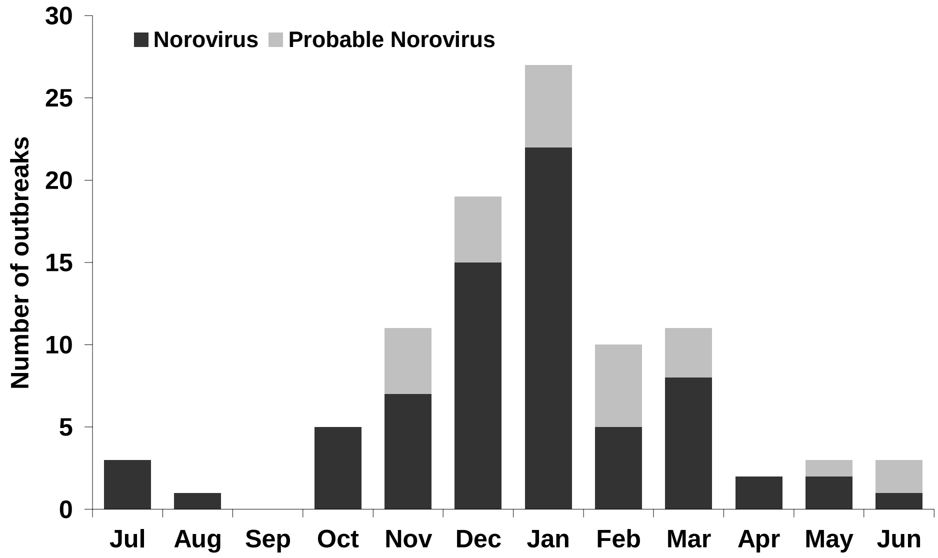
Figure 1. Viral gastrointestinal outbreaks reported by licensed skilled nursing facilities, LAC, July 1, 1999 – June 30, 2005*



*Onset dates were not available for all outbreaks – 110 of 113 viral GI outbreaks are shown (74 of 75 NV confirmed outbreaks, 24 of 24 probable NV confirmed outbreaks, and 12 of 14 other GI outbreaks). Some facilities reported multiple outbreaks.

The number of viral GI outbreaks generally increased over time, with three, five, 18, 34, and 31 reported viral outbreaks in 2000, 2001, 2002, 2003, and 2004, respectively (Figure 1). The proportion of NV confirmed or probable viral GI outbreaks reported by SNFs increased from 64% before 2002, to 92% after 2002. Outbreaks tended to occur during the winter months, from October through March with peaks in January and December (Figure 2).

Figure 2. Onset months for Norovirus outbreaks (N=101) reported by licensed skilled nursing facilities, LAC, July 1, 1999 – June 30, 2005.



Since five of the 76 unique SNFs meeting the case definition were no longer in operation by the time study participation was solicited, 71 SNFs formed the case population for this study, of which all agreed to participate in the study. Of 84 SNFs that met the control definition, 71 were randomly selected for recruitment into the study, of which sixty-eight (96%) agreed to participate. The response rate was higher in general for case SNFs compared to control SNFs—a total of 39 (55%) case facilities and 35 (51%) control facilities that initially agreed to participate in the study returned questionnaires. Administrators and Directors of Nursing were most commonly denoted as primary respondents. Case and control SNFs had similar distributions for the time periods (NV season or off-season, per year) of which the returned questionnaires were concerned, limiting the differential bias due to the seasonality of NV. The most commonly reported outbreak diseases of the control SNFs that returned the study questionnaire were scabies, pneumonia, and methicillin-resistant *Staphylococcus aureus* (MRSA).

Case and control SNFs were not substantially different when examining the numbers of staff by type during the surveyed months, except in that case SNFs employed a higher median of 11 food workers versus 8 in control SNFs during the month prior to the outbreak than (p=0.02). While only marginal differences were observed for other staff such as registered nurses (RNs), certified nursing assistants (CNAs), and custodial workers, case SNFs consistently employed numbers greater than (or equal to, in the case of custodial workers) those of control SNFs for each category. Staff-related facility stress indicators such as the numbers of new staff per surveyed month or year were not substantially different between case and control SNFs when examining individual indicators and were also too small to meaningfully compare (ranging from zero to three).

Case SNFs tended to have a greater number of rooms with a greater number of beds than control SNFs. Although the median numbers of residents reported during the surveyed months were the same for case and control SNFs (94), case SNFs reported a 1% greater median percentage of beds filled than did control SNFs (93% versus 92%), an indication that the number of residents with respect to SNF capacity, as opposed to the absolute number of residents, may better indicate the effects of crowding in the chance for a NV outbreak.

Case SNFs in general also reported greater resident-to-resident and resident-to-staff interaction opportunities than control SNFs. For instance, case SNFs reported a greater percentage than control SNFs of residents who utilized day rooms (63% versus 50%), wore diapers (75% versus 66%), and were handfed by staff (23% versus 22%). Case SNFs also reported a smaller percentage of residents who ate meals in their own rooms, versus eating in dining halls with other residents (17% versus 23%). As with the staff-related facility stress indicators, resident-related facility stress indicators such as the mean number of residents, new residents, or residents per nurse during the surveyed months compared to the surveyed years were not different for case and control SNFs.

In terms of infection control practices, case SNFs and control SNFs were similar in that custodial workers and CNAs cleaned diarrhea and vomitus more often than RNs in both case and control SNFs. NV education for staff was also similar between case and control SNFs. However, while almost all SNFs reported using gloves, more case SNFs than control SNFs reported not using masks (56% versus 42%), eye protection (82% versus 73%), aprons (62% versus 42%), and bleach or approved cleansers (51% versus 38%) to clean diarrhea and vomitus.

Table 1. Comparison of Infection Control Polices for Case and Control Skilled Nursing Facilities (SNFs) During the Month Prior to a Norovirus Outbreak, Los Angeles County, July 1, 1999 – June 30, 2005.

	No. SNFs (Baseline risk)**			Response rate*	
Characteristic	**Cases**	**Controls**	***p*** †	**Cases**	**Controls**
SNFs that lacked the following specific AGI (acute gastrointestinal illness) policies:					
Staff sent home if ill on job	8 (20.5)	8 (24.2)	0.78	39 (100)	33 (94)
Staff required to stay home if ill	9 (23.1)	9 (27.3)	0.79	39 (100)	33 (94)
Resident care (RNs, CNAs, other healthcare) staff assigned to AGI residents	26 (66.7)	21 (63.6)	0.81	39 (100)	33 (94)
Housekeeping staff assigned to AGI resident rooms	25 (64.1)	22 (68.8)	0.80	39 (100)	32 (91)
Unnecessary staff restricted from contact w/ AGI residents	14 (35.9)	19 (57.6)	0.10	39 (100)	33 (94)
AGI residents isolated	15 (38.5)	17 (48.6)	0.48	39 (100)	35 (100)
Movement of AGI residents restricted	8 (20.5)	11 (32.4)	0.29	39 (100)	34 (97)
Toilets of AGI residents sanitized	8 (20.5)	11 (32.4)	0.29	39 (100)	34 (97)
Carpets sanitized after soiled	20 (51.3)	19 (55.9)	0.81	39 (100)	34 (97)
Staff handwashing emphasized	6 (15.4)	9 (27.3)	0.25	39 (100)	33 (94)
Resident handwashing emphasized	3 (7.7)	5 (14.3)	0.46	39 (100)	35 (100)
Visitor handwashing emphasized	6 (15.4)	5 (14.7)	1.00	39 (100)	34 (97)

* Percentage that completed question of 39 case SNFs and 35 control SNFs that returned surveys
** Baseline risk is calculated as percentage of SNFs with characteristic; this calculation was affected when SNFs did not answer the pertinent question
† *p*-value reflects test of distribution similarity at $\alpha=0.05$

More control SNFs than case SNFs reported that sick pay was unavailable for RNs (26.5% versus 16.7%), CNAs (23.5% versus 16.2%), custodial workers (29.4% versus 18.9%), and food workers (29.4% versus 19.4%) during the surveyed months. Some responses to infection control practice questions, however, were opposite of what was expected. For instance, although having staff handwashing policies should reduce the number of illnesses brought into and transmitted within SNFs, more control SNFs than case SNFs reported lacking such policies during the surveyed month (Table 1). Except for policies requiring healthcare staff assignment to residents with acute gastrointestinal illness (AGI) and visitor handwashing, more control SNFs than case SNFs reported lacking policies meant to limit AGI.

Table 2. Bivariable (crude) and logistic regression (main effects) odd ratios of staff-resident and resident-resident interaction predictors for having a Norovirus outbreak among skilled nursing facilities (N=74), Los Angeles County, July 1, 1999 – June 30, 2005.

Predictors	Crude OR (95% CI)	Main effects OR (95% CI)*
Average daily resident census during year	1.021 (0.972-1.073)	0.910 (0.806-1.027)
No. residents using diapers during month	1.049 (0.989-1.113)	1.061 (0.939-1.199)
No. residents handfed by staff during month	1.081 (0.982-1.190)	1.034 (0.898-1.190)
No. residents bathed themselves during month	0.953 (0.842-1.079)	0.946 (0.820-1.091)
No. residents using day rooms during month	1.065 (0.995-1.140)	1.105 (1.003-1.217)
No. residents taking meals in own rooms during month	1.023 (0.951-1.100)	1.011 (0.908-1.126)
No. residents visited other residents during month	1.006 (0.906-1.117)	1.003 (0.865-1.189)

* ORs obtained using logistic regression, rescaled to reflect 5-unit changes
** For case SNFs, "month" refers to month prior to last reported NV outbreak. For control SNFs, "month" values were assigned to correspond to those of the case SNFs. "Year" refers to July 1-June 30, between which the "month" in question falls.

Table 2 gives the results of the logistic regression model, which demonstrates that increasing staff-resident interactions (having more residents requiring diapers and more residents requiring handfeeding by staff) could increase the risk of a NV outbreak in the following month. According to the model, if all other predictors were held constant, adding five more residents who use diapers into a SNF would increase that SNF's odds of reporting a NV outbreak by 6% in the next month. Conversely, increasing independence of residents from staff was also shown to have a protective effect: if the other model predictors were held constant, increasing the number of residents who bathed themselves by five would result in a five percent decrease in the odds of reporting a NV outbreak in the next month. The model thus demonstrates that increasing resident-resident interactions was associated with an increased risk of reporting NV outbreaks as well. Similarly, if all other predictors were held constant, increasing the number of residents who used day rooms and visited other residents by five would result in an 11% and 0.3% increase in the odds of reporting at least one confirmed or probable NV outbreak in the following month. Increasing the number of residents taking meals in their own rooms had a negligible effect. This may be because while residents who take meals in their rooms may interact with fewer residents, they may have greater interactions with staff. As expected, the model also demonstrates that increasing the number of residents who bathe themselves reduces the odds of reporting a NV outbreak.

DISCUSSION

The results of this study indicate that case SNFs might have had greater potential for NV outbreaks because they tended to have more residents per room than control SNFs. A greater median number of food service workers employed in case SNFs during the month prior to the outbreak (p=0.02) suggests that food service workers may act as a point of entry or transmission for NV into SNFs. To reduce this possibility, SNFs might establish or reinforce NV education and prevention practices among food workers, including those of outside companies contracted for food preparation and custodial services.

Although the availability of sick pay was associated with a reduction in the incidence of disease outbreaks, policies meant to limit AGI were comparable or counter intuitively present more frequently in case SNFs than control SNFs. The distributions of staff NV education were similar between case and control SNFs as well. While the effectiveness and frequency of the education might have differed between the two groups, this study suggests that policy establishment and norovirus education alone are not sufficient for effective infection control. Particularly in regards to using bleach and approved cleaners, the results of this study indicate that infection control practice is at least as important as having infection control policies to prevent NV. Control SNFs reported using additional protective equipment while cleaning diarrhea and vomitus more frequently than did case SNFs. In addition to wearing gloves while cleaning diarrhea and vomitus, using other types of protection may be substantially important for preventing NV outbreaks. Therefore, while employing a greater number of workers in direct contact with residents (namely RNs and CNAs) might increase the risk of NV transmission within SNFs, employing more workers may be beneficial if good infection control is practiced. In addition to establishing and implementing sick pay policies, AGI control policies [8], and NV education programs, the results of this study suggest several methods of prevention:

- Education and infection control training should occur in September in anticipation of the rise in outbreak incidence beginning in October. SNFs should train or retrain staff periodically to ensure knowledge and practice of effective infection control; reinforcement of infection control practices should follow in November and December to offset NV outbreak peaks in December and January.
- SNFs with many three-bed rooms should reinforce infection control practice, particularly just before and during the October to March NV season.
- Education on NV should include food workers and custodians, especially those of contracted outside companies as many SNFs use contractors for food service and facility maintenance.
- SNFs should provide adequate personal protective equipment such as gloves, masks, eye protection, and aprons as well as proper training in the use of this equipment. In addition, the training should reinforce the use of bleach and approved cleansers when cleaning up diarrhea and vomit from residents with AGI.
- Common rooms such as day rooms in SNFs need to be monitored and well-cleaned especially after incidents of vomiting and diarrhea.
- Practice of good hygiene by staff should be reinforced during staff-resident interactions such as diapering, bathing, and feeding to prevent NV from spreading to other residents.

In the planning of this study a sample size calculation using EpiInfo software indicated that, with 45 case SNFs and 45 control SNFs, if 67% of case SNFs had a risk factor for reporting a NV outbreak, only odds ratios of at least 4.0 would achieve statistical significance at the 95% confidence level. With at most 39 case and 35 control SNFs, statistical significance was not stressed in the presentation of the results of this study, as much was the comparison of numbers, medians, and percentages between case and control SNFs. While the selection criterion for control SNFs greatly reduced the pool of potential control SNFs, those selected into the study seemed representative of the case SNFs in terms of response rates and median numbers of beds. Although survey questions referred to specific periods of time in order to prevent collecting responses concerning the state of the SNFs after rather than before outbreaks, some SNFs acknowledged utilizing more recent data or guessing answers when record retrieval was difficult or impossible. The resulting temporal bias is most evident in the attenuated and even reversed-from-expected responses to questions related to AGI policies.

A prospective study on SNFs would provide better measurements of possible risk factors and predictors of NV outbreaks by limiting temporal and recall bias. Furthermore, predictors of outbreaks can be measured over time so that changes or trends in these factors might be analyzed as effects rather than studying the immediate predictor status before the outbreak. More importantly, time between the predictor and the onset of the outbreak can be measured or estimated more accurately. In this retrospective study, while the exposure-predictor questions referred to a month prior to the outbreak date, respondents might have provided answers referring to one day to thirty days prior, or even well before or after the reference date. Since prospective studies are generally resource intensive, such a study would ideally have multiple outcomes such as diseases common to SNFs. Nevertheless, further studies, retrospective or prospective, can improve prevention efforts, such as infection control practices and staff education, to reduce the number of outbreaks experienced by the SNF resident population.

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SALMONELLA HIDUDDIFY GASTROENTERITIS IN A NEWBORN NURSERY

BACKGROUND

There are over 2400 known serotypes of *Salmonella* [1] *Salmonella Hiduddify*, also known as *Salmonella I 6, 8:1z28:1, 5*, is an uncommon serotype in the United States. The PulseNet national data base does not contain any patterns for this serotype, other than those recently submitted by Los Angeles County (LAC). No PulseNet patterns were even a close match. This serotype was last identified in LAC twenty years ago in an infant case. One study reported finding this serotype in dogs in Nigeria and suggested these animals as a source for transmission of salmonellosis to humans and domestic animals [2].

On October 24, 2006, LAC Department of Public Health (DPH) Acute Communicable Disease Control (ACDC) Program was notified by the infection control professional (ICP) of two infant cases diagnosed with salmonellosis who were both cared for in the Level II nursery in an acute care hospital (Hospital A). On the same day ACDC initiated an investigation and worked together with the ICP to determine the extent of the outbreak, risk factors for disease, and any steps needed to prevent further infections.

METHODS

The hospital ICP reviewed the medical charts and provided clinical information to ACDC. District public health nurses (DPHNs) visited the cases and their families in the home and gathered data related to possible exposures and risk factors. Stool specimens were collected from caregivers and other family members for culture. A segment of reptile animal skin was also cultured from one case patient house. ACDC staff visited Hospital A to assess the physical layout of the nursery and gather additional information from staff. The LAC Public Health Laboratory (PHL) performed serotyping and molecular epidemiology using pulsed field gel electrophoresis (PFGE) on three isolates.

Case Definition: An outbreak-associated case was defined as an infant with culture-confirmed *Salmonella Hiduddify* (*S. Hiduddify*) infection who was cared for in the Level II nursery at Hospital A in October 2006.

RESULTS

A total of three confirmed cases of *S. Hiduddify* were identified, the two hospitalized newborns at Hospital A and a sibling of Infant #1. The two newborns met the case definition.

Infant #1: The infant was born at Hospital A by scheduled cesarean section on October 10, 2006. She was coupled in-room with her mother. The infant's father and two siblings were observed by hospital staff to visit frequently. The infant had a blood-streaked stool on October 12, 2006 and was subsequently moved to the Level II nursery and placed in contact isolation. The child was breast fed but also received premixed formula in individual-use bottles. The infant was treated and discharged home on October 20, 2006.

The home of Infant #1 was assessed and investigated by the DPHN. The father made drums in an adjoining workshop using animal skins, including reptile skins, imported from Africa. The skins were soaked and then stretched to construct the drums.

Stool cultures of the parents and siblings of Infant #1 detected the infant's one year-old sibling as positive for *S. Hiduddify*; the sibling had not been symptomatic. The infant's mother was positive for *S. I 9,12:a:*__(incomplete serotype); she reported having had symptoms of diarrhea and fever for two days in August 2006. The infant's father and a seven year-old sibling were negative on stool culture and asymptomatic. DPHNs educated the family regarding salmonellosis, stressing transmission prevention with emphasis on hygiene and possibility of contaminated clothing related to the handling of reptile skins in the home. A small sample of cleaned and dried skin, identified by the father as iguana skin, was provided by the father. The type of processing done on the skin before collection was unknown. This skin was cultured in the PHL for *Salmonella*; the result was negative.

Infant #2: The infant was born normal spontaneous vaginal delivery (NSVD) at Hospital B on October 14, 2006 and transferred to Hospital A on the same day due to respiratory problems. After spending three days in the neonatal intensive care unit (ICU), the infant was moved to the Level II nursery on October 17, 2006. She was breast fed but also had formula in 4 oz. bottles. This infant was discharged to home on October 19, 2006 but returned with fever and diarrhea the next day to the Hospital A emergency room.

The home of Infant #2 was also assessed and investigated by the DPHN. No other family members had been ill. There had been no travel or exposure to reptiles. Stool culture results for the infant's mother and father were negative. DPHNs educated the family regarding salmonellosis, stressing transmission prevention.

The two infants were together in the same nursery between October 17 and October 19, 2006. The ICP provided information on Level II nursery staffing. One medical team—four interns and one resident—cared for both babies during that time period. Five nurses cared for the infants; two nurses floated from the labor and delivery unit and one from the pediatric ICU.

No other infants were symptomatic in the Level II nursery. No hospital staff was symptomatic. The hospital infection control committee chair decided to test all infants who were in the Level II nursery between October 17 and October 19, 2006 and associated staff for *Salmonella*. Six infants and twenty-nine hospital staff members were tested; all results were negative. Not all staff members were tested due to intern rotations.

ACDC conducted a site visit on October 27, 2006 to review the layout of the Level II nursery. The actual room was being remodeled and was not in use at the time of the visit. Originally the room was set up in a horseshoe formation, with basinets being evenly spaced around a central room. Two or three nurses would be assigned to care for up to four infants. Two reclining sleeper chairs were placed in one section, away from the basinets; an electric breast pump was situated between the chairs. Parents were encouraged to stay with their infants and mothers to use the reclining chairs while holding and nursing their infants. When parents visit, they must wash hands for three minutes; they do not gown. Each mother has her own breast pump kit. The reclining chairs were not routinely cleaned after each use. Contact isolation does not require a one-to-one nursing ratio. Only premixed, portioned, ready-to-use formula is used at Hospital A. Per hospital staff, the families of the two infants were not observed to commingle. Both mothers did use the reclining chairs.

Three isolates were available for PFGE, including the isolates from the two cases, plus the isolate from the one year-old sibling of Infant #1. PFGE patterns for the three isolates were similar if not indistinguishable to each other using both Xba 1 and Bln 1 enzymes. PFGE differentiation could not be assessed because there were no patterns for comparison in the PulseNet national data base.

CONCLUSIONS

An outbreak of salmonellosis associated with Hospital A Level II nursery occurred during October 2006. This outbreak was identified by the hospital ICP.

S. Hiduddify is rare in California, but it is seen in Africa [2]; the origin of the animal skins used by the father of Infant #1 was West Africa. The negative culture of the skin sample did not rule out the possibility of other skins being the source of the infection. Based on the onset date and other available information, Infant #1 was infected during a family visit to the nursery and not at the time of birth. The father and siblings were asymptomatic, and only the one year-old sibling was positive for *S. Hiduddify*. It is possible that the one year-old infected Infant #1, while being held in the same bed or parent's lap or during manipulation of her diaper. Another possibility is that the father or mother was shedding the bacteria at the time of their visits. Although the mother had a different serotype she may have been carrying two serotypes of S*almonella*. She may have infected the infant during care or feeding and then cleared this serotype by the time public health screening was conducted.

Infant #1 was the likely source for Infant #2 with transmission occurring during care or via an item shared by the infants or the mothers. Outbreaks with transmission via contaminated equipment have been documented [3]. Person-to-person transmission via hospital staff and parents has also been documented [4] [5]. The parents may have had a role in transmission; however, they were not observed to commingle. Although infant formula has been the source of large *Salmonella* outbreaks in the past [6], it is unlikely that formula was the source of this outbreak based on the small number of cases and the type of formula used at this hospital.

ACDC provided Hospital A with recommendations to improve infection control practices among mothers and visiting families, as well as environmental cleaning of shared equipment and furniture.

LIMITATIONS

Limitations for this investigation include small number of cases, lack of information on PFGE differentiation, and incomplete histories on the culture-positive family members.

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AN OUTBREAK OF *ELIZABETHKINGIA MENINGOSEPTICA* ASSOCIATED WITH COLISTIN USE IN A RESPIRATORY HOSPITAL, LOS ANGELES COUNTY 2006

INTRODUCTION

Elizabethkingia meningoseptica is Gram-negative rod rarely found in the human microflora [1] but is ubiquitous in freshwater, saltwater, and soil [1,2]. It has most often been cited as a nosocomial infection among neonates [3-6]. Transmission is usually waterborne, often involves a medical device that has been contaminated or not adequately sterilized [7-12], and has been associated with infection in intensive care units [10-13]. Of particular concern, *E. meningoseptica* has demonstrated multi-drug resistance in previous studies [1,6,9,14-16].

On March 27, 2006, the Acute Communicable Disease Control (ACDC) Program of the Los Angeles County (LAC) Department of Public Health (DPH) was informed of an outbreak in a 69-bed respiratory acute-care hospital involving eight patients with positive cultures of *E. meningoseptica* since January 2006. The patient population was mostly ventilator-dependent and admitted for respiratory failure. Hospital infection control observed that eight of ten patients who received colistin were culture positive for *E. meningoseptica*.

METHODS

ACDC and the respiratory hospital collaborated to establish surveillance, collect data, and implement control measures. Hospital laboratory data since January 2005 was collected, and a standardized questionnaire was developed to review patient charts and medication lists. Active surveillance started in April 2006. Throughout surveillance, hospital infection control monitored hand hygiene and infection control practices among hospital staff. In addition, patient and staff cohorting, contact isolation precautions, terminal cleaning, hand hygiene education, and review of various procedures such as sterilization, routine cleaning, and pharmacy compounding were implemented. During the outbreak period, investigators conducted multiple unannounced site visits to the facility to assess compliance with these interventions.

To determine if the outbreak extended beyond the respiratory hospital, the Centers for Disease Control and Prevention (CDC) and California Department of Health Services (CDHS) were contacted. Through the Health Alert Network of LAC DPH an inquiry to all acute-care hospitals in LAC was issued to survey increases in *E. meningoseptica* between January 2005 and March 2006.

A case-control study was performed to determine the causes of the nosocomial outbreak. A case was defined as a patient within the respiratory hospital who had a positive culture of *E. meningoseptica* identified by the hospital laboratory and had no positive cultures of *E. meningoseptica* in the previous two months of hospital stay. The presence of new symptoms such as fever and increased white blood cell count distinguished infection from colonization. Comparing intensive care unit (ICU) admission, medical procedures, prior medications, co-infections, recent pathogens, and antibiotic resistance, an unmatched analysis using Mantel-Haenzel statistical calculations was performed to calculate odd ratios and 95% confidence intervals (95% CI). Fischer's exact tests were used to calculate 95% confidence intervals when numbers were less than six. Wilcoxon rank sum tests were performed to determine statistical differences between medians. SAS version 9.1 was used to perform statistical analyses, including multivariable logistic regression.

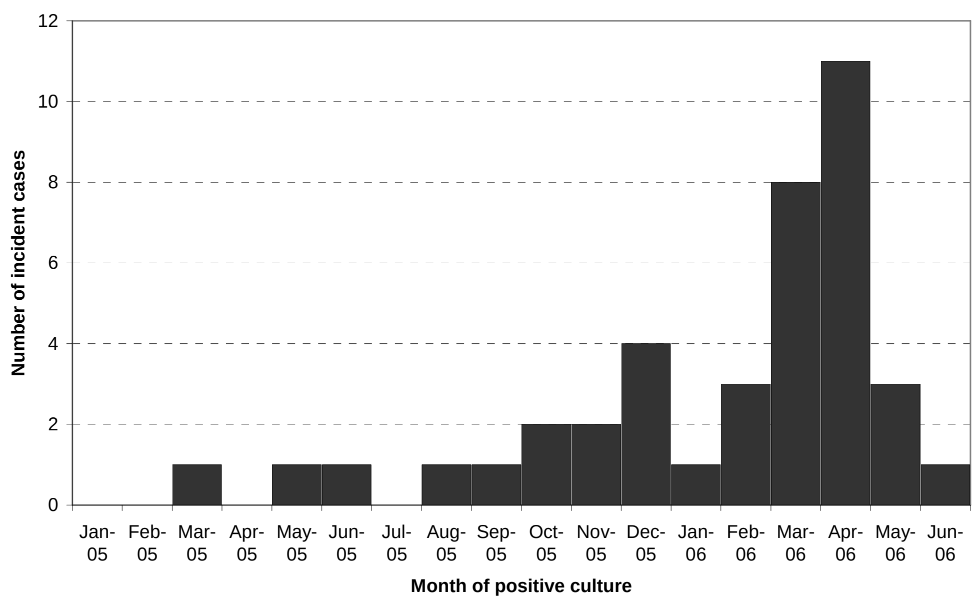
In addition, the LAC Public Health Laboratory (PHL) tested one isolate of *E. meningoseptica* for susceptibility to colistin, and from the respiratory hospital pharmacy, tested a sample of dry powder colistin, a pre-mixed colistin 3-cc syringe, a sterile 3-cc Safety Lok syringe, and a sample of the sterile water used for injections.

The Environmental Health Program (EH) of LAC DPH, Los Angeles Department of Water and Power (LA DWP), and ACDC collected multiple environmental samples to investigate possible sources of *E. meningoseptica*.

RESULTS

A total of 40 incident cases were identified by the hospital laboratory between January 2005 and June 2006 (Figure 1). One infected patient-case in December 2005 cleared infection in January but became colonized in April 2006. Thirty-five (87.5%) cases had positive sputum cultures, four (10.0%) cases had positive blood cultures, and one (2.5%) case had positive cultures from sputum and blood specimens. While small increases in incidence occurred in the winter of 2005, the occurrence of eight cases in March 2006 triggered notification of Public Health about the outbreak. With two deaths attributed to *E. meningoseptica*, case fatality was 7.1%. During unannounced site visits investigators observed respiratory therapy staff to be noncompliant with hand washing practices on multiple occasions. In addition, general infection control practices were inconsistent particularly with regard to contact precautions. Investigators noted that the use of gowns and gloves by hospital staff increased with awareness that they were being observed.

Figure 1. Number of incident cases (N=40) by month of positive culture in an outbreak of *Elizabethkingia meningoseptica* in respiratory hospital, Los Angeles County, 2005 - 2006.



CDC and CDHS confirmed that there were no concurrent outbreaks anywhere else with this organism, and survey results from 22 (21.4%) of 104 hospitals in LAC showed no evidence that the outbreak extended beyond the index hospital. Of the 22 responding hospitals, none observed any atypical increases of *E. meningoseptica* among their patients since January 2006 and 19 hospitals provided the number of patients with *E. meningoseptica* by the month of positive culture since January 2005. The total number of *E. meningoseptica* positive patients from the 19 hospitals was 24.

Outbreak investigators collected information on all patient-cases occurring in 2006, including both incidences of the patient-case who was infected in December 2005 but then colonized in April 2006. Among 27 patients there were 28 incident cases and 23 controls. Sixteen (69.6%) controls eventually became cases. Of the 28 cases, 19 (67.9%) were determined to be infected and nine (32.1%) colonized. Cases and controls were similar in age, gender, and factors for immunosuppression (Table 1).

Table 1. Characteristics of Cases (n=28) and Controls* (n=23), *Elizabethkingia meningoseptica* Outbreak in a Respiratory Hospital, Los Angeles County, Dec. 2005 – Jun. 2006

Characteristic	Cases (n=28)	Controls* (n=23)
Median** years of age at admission (range)	76 (48 - 89)	76 (62 - 89)
Females to males (ratio)	19 : 9 (2.1 : 1)	16 : 7 (2.3 : 1)
Diabetes (%)	16 (57.1%)	15 (65.2%)
Cancer (%)	10 (35.7%)	7 (30.4%)
Steroid medication (%)	5 (17.9%)	3 (13.0%)
Chemotherapy (%)	2 (7.1%)	1 (4.3%)

* 16 (69.6%) of 23 controls became cases.
** Wilcoxon rank sum score t approximation p-value=0.65.

Possible risk factors for colonization or infection by *E. meningoseptica* were analyzed in an unmatched case-control study (Table 2). Among the medical procedures that were possible sources of *E. meningoseptica*, only colistin, tracheotomy, and ICU admission had strong statistically significant associations with becoming a case. Patients receiving inhaled or injected colistin had 22.2 times greater odds of becoming a case (95% CI of 4.3 - 115.8). Tracheotomy patients had 11.8 times greater odds of becoming a case (95% CI of 1.3 - 551.5), and patients admitted to the ICU had 3.8 times greater odds of becoming a case (95% CI of 1.1 - 12.5).

Table 2. Possible Risk Factors for Colonization or Infection by *E. meningoseptica* (EM) of Infection Among 28 Cases and 23 Controls, EM Outbreak in a Respiratory Hospital, Los Angeles County, Dec. 2005 – Jun. 2006

	Number (%)		
Possible risk factors	Case, N=28	Controls, N=23	Odds ratio (95% CI)
Mechanical ventilator	27 (96.4)	18 (78.3)	7.5 (0.7 - 367.5)
Nebulizer	28 (100.0)	20 (87.0)	Undefined
Bronchoscope	7 (25.0)	4 (17.4)	1.6 (0.3 - 8.5)
Central line	17 (60.7)	9 (39.1)	2.4 (0.8 - 7.4)
Central vein catheter	10 (35.7)	6 (26.1)	1.6 (0.5 - 5.3)
Arterial catheter	0 (0.0)	0 (0.0)	Not applicable
Bladder catheter	3 (10.7)	1 (4.4)	2.6 (0.2 - 144.8)
Other catheter	6 (21.4)	3 (13.0)	1.8 (0.3 - 12.6)
Dialysis	5 (17.9)	2 (8.7)	2.3 (0.3 - 26.0)
Foley	26 (92.9)	20 (87.0)	2.0 (0.2 - 25.1)
Parenteral nutrition	3 (10.7)	0 (0.0)	Undefined
Gastrointestinal tube	21 (75.0)	14 (60.9)	1.9 (0.6 - 6.4)
Nasogastric tube	12 (42.9)	6 (26.1)	2.1 (0.6 - 7.0)
Tracheotomy	27 (96.4)	16 (69.6)	11.8 (1.3 - 551.5)
Tracheotomy care	27 (96.4)	19 (82.6)	5.7 (0.6 - 54.9)
Enteroscopy	9 (32.1)	5 (21.7)	1.7 (0.5 - 6.1)
Other respiratory procedures	6 (21.4)	1 (4.4)	6.0 (0.6 - 288.3)
ICU admission	16 (57.1)	6 (26.1)	3.8 (1.1 - 12.5)
Colisitin	19 (67.9)	2 (8.7)	22.2 (4.2 - 115.8)

All 47 environmental samples (30 surface samples, 17 water samples) and all colistin samples were negative for *E. meningoseptica*.

DISCUSSION

Increasing multi-drug resistance in Gram-negative bacteria, increasing prevalence of these bacteria, and the decline in the discovery of new antibiotics have led researchers to look at older drugs for effective treatment [17]. Polymyxins, particularly colistin, have been cited as the only available active antibiotics for multidrug-resistant Gram-negative bacteria such as *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, and *Klebsiella pneumoniae* [18-21]. Despite concerns of adverse effects of nephrotoxicity and neurotoxicity, use of colistin has increased and found success in the treatment of multidrug-resistant Gram-negative bacterial infections [22-24].

The finding that colistin had the strongest statistical association with *E. meningoseptica* in this outbreak was interesting. Because *E. meningoseptica* was not cultured from the colistin or any of the environmental samples, the source of *E. meningoseptica* remained unknown.

Although different studies have found *E. meningoseptica* to have varying susceptibilities to different antibiotics, the outbreak strain of *E. meningoseptica* was resistant to most of the antibiotics tested in these studies [4,6,14-16]; the *E. meningoseptica* strain cultured during this outbreak was resistant to colistin. Hence, as *E. meningoseptica* is rarely seen among nosocomial infections, the colistin seems to have acted as a selective factor that allowed *E. meningoseptica* to emerge in the respiratory hospital. Given that *E. meningoseptica* has appeared as a human pathogen only among people with lowered or under-developed immunity, been associated with outbreaks in ICUs, and manifested most frequently as pneumonia among non-neonates, the respiratory hospital that experienced this outbreak was an ideal setting for flourishment of *E. meningoseptica*. In a 1987 study, six weeks after polymyxin B was introduced to a medical/surgical ICU, nine patients over 2.5 months tested positive with *E. meningoseptica* [14]. Similarly, the source of the outbreak was not identified but polymxyin B was suspected to have caused a selective environment for the emergence of *E. meningoseptica*.

Although the source of *E. meningoseptica* in this setting was not identified, transmission by hospital staff was the most likely cause for this outbreak. General infection control practices were inconsistent particularly with regard to hand washing and contact precautions. Between April 22 and May 9, 2006 there was a 17-day period without a positive culture. However, compliance with contact precautions declined and four more cases occurred during May and June.

Dealing with *E. meningoseptica* in a hospital requires appropriate surveillance and infection control. Although distinguishing infection from colonization can be difficult because this pathogen occurs among the immunocompromised, identification of patients with *E. meningoseptica* allows cohorting of patients and staff to help prevent transmission. Physicians should be reminded to culture symptomatic patients when bacterial infection is suspected and prescribe antibiotics judiciously especially during outbreak situations. Moreover, antibiotic susceptibility tests of isolates should include new antibiotic therapies to ensure effectiveness of treatment. Also, environmental samples should be taken during outbreak situations as *E. meningoseptica* to determine a source of transmission. Most importantly, infection control policies and procedures should be reviewed, updated if necessary, and enforced among all staff.

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AN OUTBREAK OF *PSEUDOMONAS AERUGINOSA* IN A NEONATAL INTENSIVE CARE UNIT, LOS ANGELES COUNTY, 2006

BACKGROUND

Pseudomonas aeruginosa (PA) is a gram-negative rod-shaped bacterium that is commonly found in soil and water. It is one of the leading causes of hospital-acquired pneumonia in high-risk patient populations such as in intensive care units [1]. In the hospital setting, *PA* has been found to colonize both manual and sensored non-touch faucets, although at a higher rate in the latter [2,3].

On December 4, 2006, the Los Angeles County (LAC) Department of Public Health (DPH) Acute Communicable Disease Control (ACDC) Program was notified of eight infants in a local neonatal intensive care unit (NICU) tested positive for *PA* in a four-day period, from November 28, 2006 through December 1, 2006. An epidemiologic investigation was begun and continued through February 1, 2007.

METHODS

Setting: The neonatal intensive care unit (NICU), is a state-of-the-art facility with 28 beds. The unit is divided into 4 pods with 6 beds each and an isolation area containing space for 4 additional patients. The isolation area is separated from the main NICU pods by a staff hallway. Other areas of the unit include a nursing station, family waiting area, family scrub room, and staff and family restrooms.

Case Definition: A case was defined as a patient in the NICU that had a *PA* positive blood, nasopharyngeal (NP), endotrachael (ET) or rectal specimen culture that matched the outbreak strain by pulsed-field gel electrophoresis (PFGE) from November 28, 2006 through February 1, 2007.

Case Identification: Blood and respiratory cultures were taken on clinically symptomatic patients on November 28, 2006. NP or ET and rectal surveillance cultures were completed on all NICU patients not previously cultured on December 4, 2006 and on December 5, 2006. Surveillance cultures were collected two times a week through January 2, 2007. They continued once weekly through February 28, 2007. Rectal, blood, NP or ET cultures were collected on all patients upon NICU admission starting December 6, 2006.

Environmental Cultures: Numerous environmental cultures were obtained by the hospital and ACDC from NICU high-use areas and from devices used by patients and families. These included laryngoscope blades, sinks, multi-use nebulizers, isolate humidifiers, ventilators and ventilator circuit lines, a breast pump, and an ice-machine.

In addition, select medicaments (i.e., total parenteral nutrition, lipids, insulin, dopamine, dobutamine, ampicillin, cefotaxime, hydrocortisone, dextrose and surfactant) were cultured by the hospital.

Faucet Cultures: Restrictive flow devices (RFD) (aerators and non-aerated laminar flow devices) on faucet fixtures were removed and cultured throughout the hospital. Thirty-seven RFDs from patient areas outside the NICU (n=296) and 24 RFDs within the NICU (n=24) were cultured for *PA*. NICU RFDs cultured were from the nurses station, restrooms, family scrub room, and patient pods.

Water Cultures: Three water samples were collected from two different sensored non-touch faucets in common areas of the NICU and were analyzed for *PA* by the LAC PHL.

Molecular Epidemiologic Investigation: PFGE patterns were completed on all available patient, environmental, and faucet isolates by the LAC PHL.

Infection Control Evaluation and Measures: The unit was voluntarily closed to new admissions on December 4, 2006. Infection control measures implemented included: contact precautions for cases per hospital protocol; cohorting of cases and providing dedicated staff to each cohort; obtaining disinfected laryngoscope handles and blades from central cleaning; using single-dose medication vials/bottles when possible; re-educating NICU staff regarding infection control issues; completing terminal cleaning upon infant discharge; ensuring all staff member finger nails are short and without artificial nail applications; and using sterile water for bathing of infants.

During the temporary closure, emergency admissions were housed in the isolation suite until patient stabilization and transfer to another hospital NICU.

The unit re-opened on December 19, 2006 to new admissions.

RESULTS

Case Patient Characterization and Cultures: Isolates obtained from patient cultures taken between November 28, 2006 and December 5, 2006 showed that eight patients had matching PFGE patterns. Three of these patients died. An additional patient died who was found positive for *PA* during the outbreak period but whose isolate was unavailable for PFGE analysis.

PFGE patterns of patient isolates obtained revealed 4 different strains of *PA*. Eight patients had strain A (outbreak strain), 4 strain B, 1 strain C, and 1 strain D. Two *PA* positive patients did not have isolates available for PFGE analysis.

Environmental Cultures: Thirty-six environmental cultures obtained by the hospital were negative for *PA* with the exception of a laryngoscope blade, which was positive for *PA* and *Serratia marcescens*. PFGE analysis revealed that the laryngoscope positive strain matched the outbreak strain (Strain A). All medicaments tested for *PA* were negative.

Five of twenty-five (20%) environmental cultures collected by ACDC were positive for *PA*. These cultures were from five of five NICU sink basins. PFGE analysis on these positive cultures also revealed that they matched the outbreak strain.

Faucet Cultures: The culture results from 22 NICU RFDs revealed that 12 NICU non-aerator laminar flow devices were positive for *PA*. These included all the infrared sensored faucet non-aerator laminar flow devices from the pods (n=11) and the parents scrub room (n=1). All the non-sensored gooseneck faucet RFDs (n=5) were negative for *PA*. PFGE analysis revealed that 12 of 12 *PA* positive cultures matched the outbreak strain.

Six (n=37) infrared sensored faucet non-aerator laminar flow devices in patient areas outside the NICU were culture *PA* positive. Two of these were from the Labor and Delivery Unit located on the same floor as the NICU. The PFGE results on these cultures were unique and different from all other PFGE results. Four (n=30) were from faucets on other patient floors. PFGE analyses revealed that two of these positive cultures matched the outbreak strain. The remaining two positive cultures were unique and different from all other PFGE results and from each other.

Water Cultures: Water samples collected from two different sinks in common areas of the NICU on December 18, 2006 were negative for *PA*.

CONCLUSION AND FINAL RECOMMENDATIONS

Hospital staff stated that the cleaning practice for NICU laryngoscope blades included a tap water rinse and that this practice started in early 2006. At that time, NICU respiratory therapy staff began cleaning laryngoscope blades with a cleaning solution and tap water rinse rather than sending them to central supply for reprocessing. Healthcare Infection Control Practices Advisory Committee (HICPAC) guidelines, state that laryngoscope blades are considered semi-critical devices and should be cleaned with high-level disinfection [4].

The vehicle in this outbreak was likely a laryngoscope blade rinsed with tap water. The possible source was tap water as *PA* positive RFDs matching the outbreak strain were found on hospital wards outside the NICU. Laryngoscope blades, as multi-use devices, should undergo high-level disinfection between patient use and should not be rinsed with tap water.

Since the outbreak was investigated and infection control measures implemented, including using only blades from central supply that have undergone high-level disinfection, there have been no further cases of the outbreak strain detected. The role of non-aerated laminar flow devices and sensored faucets should be studied to determine their role in transmission of nosocomial infections.

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FOLLOW-UP SURVEY OF SIDE EFFECTS OF SINGLE DOSE CIPROFLOXACIN FOR PROPHYLAXIS OF MENINGOCOCCAL DISEASE IN A LOS ANGELES COUNTY HIGH SCHOOL

BACKGROUND

On November 14, 2006, two cases of invasive meningococcal disease (MD) occurring in students attending the same high school (HS) were reported to the Los Angeles County (LAC) Department of Public Health Department (DPH). One case was culture-confirmed with *Neisseria meningitidis* serogroup B bacteremia and the other was later PCR-confirmed with serogroup B meningococcal meningitis (culture-negative). The two students did not know each other and did not share common classes, friends or school activities. Following the confirmation of these cases, the LAC DPH stood up two point-of-distribution (POD) clinics to dispense prophylaxis for students and teaching staff at the HS who may have had contact with these students. The first clinic was held on the evening of November 14th, and an additional clinic the morning of November 15th. Parents and students were notified about the clinics through the school's automated phone message system, internet page, and a letter to parents. School officials released the names of the two ill students during the first clinic after obtaining parental consent, in an effort to identify the direct contacts that would require prophylaxis. Despite this, over 3000 persons were evaluated and 2861 persons were provided with single-dose ciprofloxacin prophylaxis.

As part of the routine public health follow-up of individual suspected and confirmed cases of invasive meningococcal disease, all contacts are evaluated for prophylaxis and educated on the symptoms of invasive MD. Mass prophylaxis is usually not considered except in situations which meet the Centers for Disease Control and Prevention (CDC) criteria for meningococcal outbreaks, defined as three or more cases within a three month period occurring in an institutional setting such as a school or among military [1]. The decision to provide prophylaxis on a mass basis rather than only to known close contacts of the case must be weighed against the risk of high numbers of reports of serious side effects associated with the prophylactic antibiotic, including anaphylaxis, to local health facilities, as well as the possibility of antimicrobial resistance developing within a contained community. In this situation, the decision was made to provide prophylaxis to self-identified close contacts through a distribution clinic because neither student could be interviewed to identify close contacts in a timely manner; and the extent of *N. meningitidis* carriage in this population could not be ascertained. Further, ciprofloxacin is generally well tolerated, having been utilized successfully without adverse events in other HS settings in California where mass prophylaxis had been required [California CD Brief, March 4, 2001]. Moreover, *N. meningitidis* has been rarely observed to be resistant to ciprofloxacin.

The use of ciprofloxacin in the pediatric and adolescent population has been limited because irreversible joint damage has occurred as a side effect in juvenile animal studies. Despite this, ciprofloxacin has been commonly used for children and adolescents when other treatment is not an option. Irreversible joint damage has never been found to occur [3-6]. District public health personnel documented only two major adverse events immediately following the clinic—two (0.07%) students developed rash without anaphylaxis. However, a number of adverse events may have gone unreported.

Two weeks after the POD clinics were held, LAC DPH conducted a follow-up survey study of all students and teaching staff of the high school in order to quantify possible side effects related to single-dose ciprofloxacin in an adolescent population and to evaluate the reasons such a large number of students and staff chose to receive prophylaxis despite being at low risk. Such a study would detect any minor or unreported adverse events that were not documented during the clinic or by another healthcare provider. Further, the results of the study may help provide information for future public health responses to both institutional outbreaks of infectious disease as well as bioterrorism events.

METHODS

As part of school policy, parents were notified prior to student participation in a follow-up POD clinic survey. Parents, students, and teaching staff were notified of the upcoming survey one week in advance via an automated phone message system and an announcement on the school's webpage. The survey was distributed to all HS teaching staff and students during their homeroom period on November 28, 2006. Completed surveys were collected by HS staff through December 3, 2006. Survey data included: demographics, the date of POD clinic attendance, reasons for attendance, side effects of single dose 500 mg ciprofloxacin, type of contact with the case students, health status at the time of the clinics, perception of risk of a variety of health conditions, and knowledge of MD. Respondents were asked to rate the importance of reasons for clinic attendance on a scale from 1 to 5, 1 being not important and 5 being very important. They were asked to rate their perception of risk of various health conditions on a similar scale as previously noted. The health conditions included meningitis and ranged from rare conditions such as avian influenza (referred to as "bird flu" on the survey) and cancer to more common conditions such as being in a traffic accident. Part of their knowledge of MD was assessed by asking students to identify the correct modes of transmission of MD. Data were entered into Microsoft Access and analyzed with SAS 9.1. Because of the known differences in the side effects and attitudes between adults and adolescents, the student and staff were analyzed as two separate populations. The differences in proportions were evaluated by chi square analysis and Fisher's exact test.

RESULTS

Surveys were distributed to 2888 students in attendance the day of the survey and 105 teaching staff in 105 homeroom classes. A total of 1717 completed surveys were returned (n=1649, or 57%, of students and n=68, or 65%, of teaching staff). All parents allowed the participation of their child on the survey. Twenty-seven surveys were excluded (2%) from the analysis because they did not contain enough information due to missing or inappropriate answers. A majority of the returned surveys (n=1690, 98%) from students and staff were available for analysis. Of these, 1624 (96%) were completed by students and 66 (4%) were completed by staff. Only results from the analysis of student surveys will be presented in this report.

Among all students who completed the survey, 49% were male and 50% were female. Students were distributed evenly among ninth to eleventh grades (26% to 28%), but there were slightly fewer 12th graders (18%). This is significantly different from the distribution of students at the high school ($p<0.0001$). The race/ethnicity distribution was 49% white, 33% Asian, 8% Latino, 6% were mixed race or other, and 2% were black. The distribution of whites, Asians, and Latinos is also significantly different from that of the high school ($p<0.0001$). Most of the students who completed the survey (n=1445, 89%) attended the clinics. More females than males attended the clinics (91% versus 87%, $p=0.0038$) All racial/ethnic groups attended the clinics at similar proportions (85% to 91%), with the exception of blacks, with only 74% reporting clinic attendance ($p=0.0231$) (Table 1).

The mean ratings of reasons for attendance among students ranged from 2.13 for having "contact with one of the sick students" to 3.97 for "parents told me to". Only 24% of student respondents rated the importance of having contact with the ill students as a 4 or 5. "Heard about it in the media" was rated second to last at 2.56 with only 30% of students rating its importance at 4 or 5 (Table 2).

Table 1. Differences in Characteristics of Students Completing Survey and Attending POD Clinics

		All HS Students n (%) (n=2962)	Total n (%) (n=1624)	p-value	Surveyed Students: Attend POD Clinics n (%)* of Surveyed Students (n=1445)	Surveyed Students: Did Not Attend POD Clinics n (%)* of Surveyed Students (n=179)	p-value
Gender	Male	1469 (50)	795 (49)	0.8578	689 (87)	106 (13)	0.0038
	Female	1493 (50)	817 (50)		745 (91)	72 (9)	
	Unknown	--	12 (1)	--	11 (92)	1 (8)	--
Grade	9th	782 (26)	419 (26)	<0.0001	366 (87)	53 (13)	0.1922
	10th	735 (25)	440 (27)		385 (88)	55 (12)	
	11th	742 (25)	459 (28)		416 (91)	43 (9)	
	12th	703 (24)	292 (18)		266 (91)	26 (9)	
	Unknown	--	14 (1)	--	12 (86)	2 (14)	--
Race	Asian**	829 (28)	530 (33)	<0.0001§	480 (91)	50 (9)	0.0231
	Black***	--	27 (2)		20 (74)	7 (26)	
	Latino	237 (8)	135 (8)		117 (87)	18 (13)	
	White	1807 (61)	799 (49)		714 (89)	85 (11)	
	Mixed/Other***	89 (3)	104 (6)		88 (85)	16 (15)	
	Unknown	--	29 (2)	--	26 (90)	3 (10)	--

* Percentages tabulated across rows, not columns.
** Includes Filipinos in surveyed students but excludes Filipinos among all HS students.
*** Includes mixed race and American in surveyed students but excludes Black, American Indian, Filipino, and Pacific Islander among all HS students.
§ Chi square test performed only among Asian, Latino, and White race categories.

Of the 1445 students who attended the clinics, 1390 (96%) took the ciprofloxacin. Table 3 lists the main side effects experienced by 608 students (44%) after taking the antibiotic. Most (69%) were able to recall an onset time. Among these, 57% reporting experiencing side effects from one to six hours after ingesting the single dose of ciprofloxacin. The median onset time was three hours. A greater proportion of females reported side effects compared to males (49% versus 39%), (p=0.0002). The most common side effects reported were headache (20%) and stomachache (12%), followed by sore throat, restlessness and muscle pain (each at 6%). Other notable side effects occurring less commonly were nausea/vomiting (5%), itching (3%), rash (2%), difficulty breathing (2%), and one case of face swelling. No joint pain was reported.

Table 2. Reasons for Clinic Attendance among Students

Reason for Attendance	Mean Rating of Importance	% Rated 4 or 5
Parents told me to	3.97	71
Heard phone message/ Received letter from school	3.34	51
Fear of serious illness or death	3.24	48
Friends did it	2.87	36
Advised by physician	2.63	35
Heard about it in the media	2.56	30
Had contact with one of the sick students	2.13	24

There was a significant difference in the proportion that reported side effects in those already ill compared to those who were not ill (60% versus 40%, p>0.0001). The most common side effects among those who were not already ill at the time of the clinics included: headache (17%), stomachache (10%), followed by restlessness, muscle pain, sore throat and nausea/vomiting (each at 4%) (Table 3).

Table 3. Reported Side Effects among Students Who Took Single Dose Ciprofloxacin (500mg)*

	All n (%) (n=1390)	No Illness Report at Time of POD Clinics n (%) (n=1153)	Illness at Time of POD Clinics n (%) (n=237)
≥1 Side Effect	608 (44)	465 (40)	143 (60)
Fever	48 (3)	28 (2)	22 (9)
Cough	72 (5)	33 (3)	39 (16)
Sore Throat	83 (6)	44 (4)	39 (16)
Headache	281 (20)	191 (17)	90 (38)
Watery Eyes	40 (3)	26 (2)	14 (6)
Stomachache	166 (12)	116 (10)	50 (21)
Itching	40 (3)	27 (2)	13 (5)
Rash	21 (2)	10 (<1)	11 (5)
Diarrhea	33 (2)	21 (2)	12 (5)
Nausea/ Vomiting	67 (5)	42 (4)	25 (11)
Difficulty Breathing	22 (2)	11 (<1)	11 (5)
Muscle Pain	79 (6)	45 (4)	34 (14)
Anxiety	24 (2)	12 (<1)	12 (5)
Restlessness	80 (6)	51 (4)	29 (12)
Tired	32 (2)	29 (3)	0 (0)
Muscle Stiffness	5 (<1)	4 (<1)	1 (<1)
Face swelling	1 (<1)	1 (<1)	0 (0)

*Students can have more than one side effect

A considerable number of students completing the survey (n=282, 17%) reported experiencing symptoms from other illnesses at the time the POD clinics were set up (Table 3). This is the same prevalence of illness among students who attended the clinic and took the antibiotic. There was no significant difference in the prevalence of illness between students who attended and did not attend the POD clinics. Among those who took the antibiotic, coughing was mentioned most frequently (n=110, 8%) as a symptom experienced at the time of the clinic. Fifty-one (4%) mentioned a headache and 70 (5%) mentioned a stomachache (Table 4).

Table 4. Reported Symptoms from Illness Experienced by All Students at the Time of the POD Clinics*

Symptoms	All Students n (%) (n=1624)	Took Ciprofloxacin n (%) (n=1390)	Did Not Take Ciprofloxacin n (%) (n=282)
Total Ill	282 (17)	237 (17)	45 (16)
Fever	82 (5)	68 (5)	14 (6)
Cough	138 (9)	110 (8)	28 (12)
Headache	58 (4)	51 (4)	7 (3)
Stomachache	81 (5)	70 (5)	11 (5)
Sneezing	65 (4)	54 (4)	11 (5)

*Students can have more than one side effect

The majority of all student respondents (n=1223, 75%) had no contact with either of the cases. Only 50 (3%) reported sharing an item such as a cigarette, food or drink—activities that would put these students at highest risk for MD. The most frequent type of contact reported was being in the same class with the cases (n=158, 10%). Other types of contact listed included indirect relationships to the cases (e.g., friends of siblings) (n=67, 4%) and having casual direct contact with the cases (n=43, 3%).

Table 5 lists adverse health conditions, including meningitis, in decreasing order of mean rating of perceived risk. The students rated their risk of meningitis very low (mean of 1.49) relative to the other listed health conditions. Very few (5%) rated their risk as a 4 or 5.

Table 5. Perceived Risk of Various Health Conditions

Health Condition	Mean Rating of Perceived Risk	% Rated 4 or 5
Common cold	3.41	49
Other injury	2.86	31
Flu	2.68	27
Traffic accident	2.54	21
Food poisoning	2.14	12
Cancer	1.77	9
Obesity-related disease	1.73	10
Meningitis	1.49	5
Bird flu	1.35	3

Sixty-nine percent (n=1113) of student respondents reported not having knowledge of MD prior to the incident. These students attended the clinic in a larger proportion than those who reported having some knowledge of MD (90% versus 87%, p=0.032). Students who incorrectly identified touching objects touched by case students as a transmission mode attended the POD clinic more often (92% versus 86%, p =0.0007).

DISCUSSION

The POD clinics provided public health officials with a rare opportunity to detect side effects of single dose ciprofloxacin in a healthy adolescent population. The follow-up survey conducted two weeks after the clinics were held enabled documentation of a 44% overall rate of side effects, or a rate of 40% among students who were not already ill at the time of the clinic. These included both minor side effects as well as more serious ones that may have been related to anaphylaxis. The survey results also helped public health to deduce the main reasons for participation in a prophylaxis clinic involving a single dose of an oral antibiotic in a high school setting.

The overall frequency of side effects from ciprofloxacin reported in this adolescent population (44%) is similar to that reported for this age group in the Physicians Desk Reference (PDR), which reported a rate of 41% from a clinical trial among complicated urinary tract infection patients prescribed ciprofloxacin [2]. The frequencies of individual symptoms in this population differ substantially than what is listed in the PDR and other pediatric studies. The most commonly reported side effects associated with ciprofloxacin among children and adolescents are gastrointestinal (including nausea, diarrhea, vomiting, and abdominal pain), central nervous system (headache and restlessness), and dermatologic symptoms. This study reports headache in 17% of healthy students, stomachache in 10%, and no joint-related disorders. In the PDR, gastrointestinal symptoms occurred in 15% of patients, musculoskeletal symptoms in 9.3%, abdominal pain in 3.3%, and headache in less than 1% [2]. A few other pediatric studies have shown similar rates of gastrointestinal symptoms that have ranged up to 14.5%. Neurological symptoms, which may include headache, in these same studies, however, range only up to 4.8% [5]. Most other studies report much lower rates of specific symptoms: abdominal pain ranged from 1% to 5% and headaches from 0% to 4% [4-6]. The frequency of joint disorders in these studies, however, are higher than this findings and ranged from 1% to 22% [3-6].

Prior to the implementation of the survey, only two adverse events were documented—two students with rash who required oral Benadryl®. The survey revealed multiple other occurrences of rash and itching (2% and 3%, respectively) as well as breathing difficulties (2%) and a case of facial swelling—all possible anaphylactic reactions to ciprofloxacin which were not reported to public health prior to the survey. The frequency of these symptoms falls within range of other referenced pediatric studies. Itching and rash, for example, are seen in about 2% of patients in these published studies. Vomiting occurred in 2% to 5% of patients [2,4,6].

The high rates of adverse events seen in this study compared to previously published pediatric studies can be explained by the use of ill or hospitalized populations in these studies. In this patient setting, study participants are most likely in a controlled environment where interactions with substances commonly consumed by adolescents such as caffeine and nicotine are limited or nonexistent. Ciprofloxacin can act to increase the effects of caffeine in particular, and this most commonly reported symptoms are also known side effects of caffeine, including headache, stomachache or abdominal pain, and restlessness and anxiety [2]. In addition, the lack of serious illness in this study population may have promoted detailed recall of minor symptoms that may be overlooked or unimportant in an ill population. It has been documented that even among healthy persons who were not taking any medications, minor symptoms such as headache, fatigue, and drowsiness, are common [7]. Benign bodily symptoms such as these may be mistakenly attributed to side effects of medication. This phenomenon would be emphasized as the high school population was in the midst of the fall/winter “cold and flu season” and already experiencing a general illness rate of 17% at the time of the clinic.

Conversely, there is a superior ability to detect side effects in patient populations because of the availability of healthcare professionals and special monitoring. Further, the follow-up time in these patient population studies ranged from 20 days to 6 weeks, longer than the two week follow-up period of this study, enabling a greater window of time to detect side effects. These populations also underwent longer treatment courses and higher doses of ciprofloxacin whereas this student population took only one single dose.

Few associations were found to be significant that could explain the high rates of attendance and subsequent acceptance of antibiotic prophylaxis. A minority of surveyed students (25%) had any contact with the students, and much fewer (3%) had direct contact that may put them at risk for MD. Accordingly, having contact with the case students did not factor heavily in their decision to attend the clinic. Interestingly, experiencing current symptoms of illness was not a factor in either attendance or intake of antibiotic. Having better knowledge of meningitis and the methods of transmission was some indicator of attendance and antibiotic intake. Though the students understood that they were at low risk of meningitis, rating it nearly last only before avian influenza, a large majority of the student population attended the clinic. Because “fear of serious illness or death” was rated relatively high, it appears that students and staff felt that even at low risk, the consequence was serious enough to warrant prophylaxis.

Evidence suggests that school and public health officials may have inadvertently encouraged all students and staff to seek prophylaxis. Hearing the school’s telephone message or receiving the letter from the school administrators was rated among the highest as an important reason for attending the clinics. It has been suggested that parents and students were highly influenced by the advice of their personal physicians or the message given by the media, namely, that there was a “meningitis outbreak”, despite the fact that public health officials made it clear that one confirmed case and a suspected case did not meet the definition of an outbreak. However, “advised by a physician”, as well as “heard about it in the media” even more so, had lower mean ratings of importance. Furthermore, the health announcement the HS administration initially composed did not specifically focus enough on close contacts and may have also communicated heightened fear and risk. Though the names of the case students were released in order to limit attendance, they were announced to parents and students as the first clinic was already underway. Finally, the structure of the POD clinic itself did not alleviate the high attendance as it was designed more for distributing medication rather than assessing risk and need.

A major limitation of the study was the lack of a placebo group to determine if symptoms reported were a side effect of ciprofloxacin alone. This would not have been feasible or appropriate in a public health response setting without prior approval from an Institutional Review Board. In such a study, factors such as interactions with additional consumed substances or the background prevalence of illness would be controlled for. The survey was implemented two weeks after the clinic event, increasing recall bias of reported symptoms, particularly as most symptoms had an onset within six hours after ingestion of the antibiotic. The lag time in survey implementation may also have influenced the response rate of the survey: only 57% of students in attendance that day completed the survey. The surveyed students were not representative of the school as there were differences in rates of participation among grade levels and race/ethnicity groups. Lastly, the survey was self-administered without the presence of public health staff, which could have decreased the validity of many answers, especially the self-report of symptoms.

Despite these limitations, the results of this study fell within range of adverse events found in previous studies. As adverse events from ciprofloxacin in pediatric populations have often been studied in patient groups, this study added insight on how ciprofloxacin may affect a healthy population. Though the occurrence of side effects approached the higher range of published rates, the side effects were minor and most did not require medical attention. The lack of any joint-related side effects also further supports the safety of ciprofloxacin in the pediatric population as seen in previous studies, especially in the setting of single dose usage. These results provide a realistic assessment of the frequency and severity of side effects that would be useful for other situations of mass prophylaxis, for both common outbreaks as well as bioterrorism events.

Additionally, the results of this study indicate that parents and students are reasonable and rational in the face of the threat of a serious disease and are highly influenced by the advice of school officials. Public health officials must work closely with schools to explain the risk of disease and advise on appropriate prophylaxis distribution. Presenting a balanced message by communicating the risks of unnecessary use may encourage more prudent use of the antibiotic prophylaxis.

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PROPHYLAXIS OF HIGH SCHOOL STUDENTS WITH CIPROFLOXACIN FOLLOWING TWO CASES OF INVASIVE MENINGOCOCCAL INFECTION AT A LOS ANGELES COUNTY HIGH SCHOOL, NOVEMBER 14-15, 2006

Over the past 10 years, Los Angeles County (LAC) Department of Public Health (DPH) has confirmed 40 to 60 cases of invasive meningococcal disease annually. Outbreaks of invasive meningococcal disease, defined as three or more cases within three months [1] within a circumscribed community (e.g., school) or group of individuals sharing a common exposure, have been rare events in LAC, with the last outbreak noted in 2001 among attendees of a nightclub [2]. As part of routine public health follow-up, all contacts to both confirmed and suspected cases are evaluated for prophylaxis and educated on the symptoms of invasive meningococcal disease; meningococcal isolates are serotyped and may be genotyped by Pulsed Field Gel Electrophoresis (PFGE) at the LAC Public Health Laboratory.

On November 14, 2006, one culture-confirmed case of *Neisseria meningitidis* (*N. meningitides*) bacteremia (serogroup B) in a high school student and an additional case of suspected meningococcal meningitis (culture-negative) in a critically ill teenager were reported to the LAC DPH by the same community hospital. Medical record review and interviews with family members revealed that both teenagers had symptom onset on November 12, 2006. Both cases attended the same school, but did not know each other, share classes together, or participate in similar activities such as clubs and/or sports teams. Further diagnostic work-up revealed that the culture negative meningitis student had PCR-positive cerebral spinal fluid (CSF) for *N. meningitides*, serogroup B (California Microbial Disease Laboratory) despite negative blood and CSF cultures.

On the same day, after consultation with high school officials and California Department of Health Services Division of Communicable Disease Control, the LAC DPH held a point-of-distribution (POD) clinic at the students' school to dispense prophylaxis in anticipation of a large turnout. Two clinics were held, one on November 14 from 6 to 9 p.m. and an additional clinic the following day from 8 a.m. to 2 p.m. Parents and students were notified about the POD clinics through the school's automated phone message system, internet page, and a letter to parents, advising only close contacts of the students to obtain prophylaxis. Although the students' names were made public (after parental permission was granted) in an effort to identify only those students who had direct contact with the two ill students, over 3000 students and teachers were evaluated and 2861 persons were provided with prophylactic medication (ciprofloxacin 500 mg orally in a single dose). Two teens experienced allergic reactions—skin rash with itching—and were treated with diphenhydramine. Additionally, five students attending the POD were referred to local hospitals for evaluation of symptoms suggestive of meningitis; one received a lumbar puncture. No student had meningitis and all five were discharged.

Two weeks after completion of the POD, a follow-up survey was distributed to all students and staff at the school. The goals of the survey were to quantitate the possible side-effects related to single-dose ciprofloxacin in an adolescent population and to evaluate the reasons why so many students and school staff chose to receive prophylaxis despite being at low risk. The survey results are presented in a separate article within this Special Studies Report. At six weeks after the symptom onset of the cases, no additional meningococcal cases associated with this high school had been documented.

DISCUSSION

LAC DPH successfully held a POD clinic to provide antimicrobial prophylaxis rapidly to contacts of one culture-confirmed case of meningococcal bacteremia and one suspected case of meningococcal meningitis within 24 hours of notification to the DPH. Although ACDC recommended that prophylaxis be provided only to persons with close contact to the cases (e.g., shared drinks, cigarettes, secretion), nearly 2900 students and staff received prophylaxis. Factors that may have contributed to this very large participation included:

- the school administration composed the school health announcement to parents and students that was not specific enough to focus on close contacts;

- additionally, the county supervisor's office for this region issued a press release, advising LAC residents of a "meningitis outbreak", despite the fact that the DPH determined that one confirmed case and a suspected case did not meet the definition of an outbreak.

At the time of POD formation, it was not known if the case of bacterial meningitis was caused by *N. meningitis*, although PCR diagnostics revealed Group B meningococcus in the CSF of the suspect case by the time of the second clinic. This meningitis case remained culture-negative; thus PCR proved to be a very important diagnostic tool in providing bacteriologic and serogroup information.

Ciprofloxacin was chosen for prophylaxis because it can be administered in a single dose and is generally well tolerated. Despite experience from large setting, school outbreak prophylaxis distributions (California CD Brief, March 4, 2001) and its widespread use in the treatment of uncomplicated gonorrhea infection in adolescents [3], ciprofloxacin in adolescents is still not supported in the Pediatric Red Book [4,5] or the Physicians Desk Reference (PDR) [6]. The survey documented only two adverse events (0.07%) in students who developed rash without anaphylaxis and were successfully treated with diphenhydramine. This is less than the 1% frequency noted in the PDR [6].

By all accounts, the POD clinic was successfully and efficiently executed by public health officials, parents, and school administrators who participated and observed. The clinic's success can be attributed to recent bioterrorism-related preparedness exercises that have stressed rapid organization of POD clinics for vaccines and antibiotics. Other helpful factors included having school officials, a public health pharmacist, public health nurses, a public health medical director, and the health officer on site.

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MYCOBACTERIUM CHELONAE INFECTIONS FROM A TATTOO PARLOR LANCASTER, CALIFORNIA 2006

BACKGROUND

This report describes three cases of *Mycobacterium chelonae* infection in customers who received tattoos in February 2006 at the same tattoo parlor in Lancaster, California. *Mycobacterium chelonae* is rapid growing and ubiquitous in the environment such as soil and water, including tap water. It is associated with infections of the skin, lung, bone, joint, nervous system and eye. It manifests as localized soft-tissue and skeletal infections in otherwise health individuals and more disseminated disease in immunocompromised patients. Transmission can occur from incidental environmental inoculation such as subcutaneous exposure to contaminated water. This mode of exposure is possible during the act of tattooing.

The state of California currently has no tattooing regulations; however the Los Angeles County Code does have Department Regulations on Body Art requiring the use of specific dyes, inks and pigments and only sterile water as a dilutant. Most cities within Los Angeles County including Lancaster did not adopt the Los Angeles County Regulations. Additionally, tap water is sometimes used to dilute inks during the tattooing process. Mycobacterium species are common in tap water and may lead to subsequent skin infections. Therefore, this report has implications for future regulation of tattoo parlors.

METHODS

<u>Case Investigation</u>: On March 24, 2006 the Los Angeles County (LAC) Department of Public Health (DPH) Acute Communicable Disease Control (ACDC) Program received a report of a 25 year-old male patient with an arm abscess positive by fluorescent staining for mycobacterial species. The patient had recently obtained a tattoo on February 12, 2006. Onset of infection occurred two days later and consisted of painful, itchy red papules over the gray areas of his tattoo. He saw a physician multiple times and received antibiotics with no resolution. A skin biopsy was taken on March 15, 2006 and a culture was taken on March 27, 2006. The culture was positive for *Mycobacterium chelonae*.

The tattoo artist reported two other recent tattoo recipients with similar looking infections. Both received the tattoo from the same tattoo parlor and artist. The two recipients were notified, interviewed and skin biopsies were collected on March 30. Both cultures were positive for *Mycobacterium chelonae*. Two patients have been prescribed Clarithromycin (Biaxin) for six months and one patient has been prescribed Azithromyacin for three months. No physical limitations have been noted in any of the patients.

<u>Case Finding</u>: A client list from the tattoo parlor was obtained to commence case finding. No other clients have reported any symptoms consistent with Mycobacterium species infections to date. However, the client list was not complete and many of those listed were missing contact information. A health alert was also sent out to all physicians on March 24, 2006 to ask them to report any tattoo related skin infections from the Antelope Valley region. Additionally, a letter was written to all dermatologists, pediatricians, family care practitioners, urgent care doctors and internal medicine doctors in Antelope Valley asking them to report any tattoo related skin infections. There have been no other reports of skin infections related to tattoos from this tattoo parlor or others in the region.

<u>Environmental Investigation</u>: On March 24, 2006, ACDC Program inspected the tattoo parlor. Environmental specimens were obtained for culture using both agar-based media and 7h9 MGIT broth media. Photographs were taken of the tattoo parlor specifically in areas where contamination could occur. Specimens taken included the sink spout, the water cooler's spout, the tattoo instruments, the ink, receptacles for water used for cleaning instruments and diluting colors and other ointments used to clean and soothe the skin before and after tattooing. Environmental samples including the exterior and interior sink faucet, the sink drain and the water cooler's spout and basin were all positive for *Mycobacterium gordonae*. Other samples had no evidence of Mycobacterial species.

Additionally, an informal environmental inspection took place using the terms of Los Angeles County Code Title 11 chapter 11.36, Department Regulations for Body Art. This inspection was not official as tattoo parlors in the Antelope Valley are not registered or regulated by the LAC DPH Environmental Health Branch.

On March 31, 2006 a draft of the Environmental Health report of recommendations was sent to the tattoo parlor. Tattooists at the parlor were not registered as body art technicians in LAC and they do not have proof of having completed bloodborne pathogen disease transmission prevention training. There were ashtrays in the work area filled with cigarettes and there was no clear separation between the autoclave and ultrasonic device which may allow cross-contamination. The floors of the tattoo parlor were not smooth or cleanable and were not sanitized on a regular basis. There was no hand-wash sink in the workstation and no hot water under pressure provided in the sink in the bathroom.

An interview with the tattoo artist revealed that he used tap water to shade the paints when tattooing the three patients. Specifically, he may have used the drinking water from the water cooler or water from the bathroom sink of the tattoo parlor. Usually distilled water is used; however the artist stated that if no distilled water is available, other water might be used.

DISCUSSION

Los Angeles County regulations require sterile potable water to be used during the tattoo process only. Specifically section 525.00 states:

“(a) All inks, dyes, and pigments used to alter the color of skin in the conduct of body art shall be specifically manufactured for such purpose, approved, properly labeled as to its ingredients, manufacturer and lot number in accordance with applicable FDA requirements, and shall not be contaminated or adulterated. The mixing of such inks, dyes or pigments or their dilution with potable sterile water is acceptable, unless prohibited or not recommended by its/their manufacturer.

(b) Inks, dyes and pigments prepared by or at the direction of a body art technician for use in body art activity shall be made exclusively of non-toxic and non-contaminated ingredients approved by the department or FDA.”

However, as noted previously, this tattoo parlor is in Lancaster which has not adopted the ordinance nor is registered with the county. Only unincorporated regions of Los Angeles have adopted the ordinance. Additionally, although the state of California is currently drafting Body Art Regulations; these regulations do not contain guidelines for ink dilution.

At this time ACDC Program recommend that Lancaster and other incorporated areas adopt the Los Angeles County ordinance and register with the county and that the drafted State Body Art Regulations include guidelines on ink dilution. The tattoo parlor is slowly making changes and the tattoo artist who performed the tattoos that facilitated the infections is no longer working at this establishment. Although the exact cause of this outbreak was not determined, Mycobacterium species were found in two water sources. It is likely that this outbreak could have been prevented if the tattoo parlor was up to code and if only sterile water was used in the tattooing process.

UNIVERSITY PANDEMIC INFLUENZA PLANNING SUMMIT

OVERVIEW

On April 28, 2006, the Acute Communicable Disease Control (ACDC) program of the Los Angeles County (LAC) Department of Public Health Services (DPH) held a pandemic influenza planning summit with select representatives (e.g., student health center directors, risk management, directors of student affairs) of many Los Angeles-area universities. To focus and prioritize the scope of the summit, invitations were limited to universities that maintain on-campus housing. Representatives from 17 universities as well as key LAC DPH staff attended. Prior to attending, university representatives were asked to submit a brief survey summarizing the characteristics of their campus (Table 1) and whether they have included pandemic influenza as a part of their emergency preparedness planning.

The summit agenda consisted of three informative presentations followed by a tabletop discussion. The three presentations provided information on: differentiating seasonal, avian, and pandemic influenza; understanding issues specific to avian influenza; and, detailing advanced information and guidance on pandemic influenza and planning especially for universities. Guiding the subsequent tabletop discussion was a series of hypothetical pandemic influenza-related scenarios that may impact universities (Table 2). The tabletop provided a forum for the university representatives to suggest steps they might undertake before, during, and after an influenza pandemic. In addition, the tabletop served to generate suggestions for materials that LAC DPH can provide to assist universities with their pandemic influenza planning (Table 2). In addition, many informative handouts were also provided including: supplemental information on influenza and pandemic influenza, pandemic planning checklists, and lists of public health resources.

BACKGROUND

Pandemic influenza has the potential to cause tremendous impact on health and welfare globally, nationally, and locally. Recently, the need to prepare for a pending pandemic became more critical following the emergence of an Asian strain of avian influenza A H5N1 (commonly known as “bird flu”). The continuing spread of H5N1 in wildlife, and the continued animal outbreaks and human cases, has heightened concerns that this viral strain will eventually mutate and cause a pandemic. But unlike seasonal influenza, which circulates annually, and as such, has predetermined activities for preparation and response (i.e., established risk-groups and protocols for vaccination and treatment, etc.), pandemics are unpredictable—the onset, severity, and full range of characteristics of a pandemic are unknown. This inherent uncertainty, coupled with continual scientific advancements, responses, and changes in circumstances, greatly complicates planning.

There are many factors unique to universities, and the students that they serve, that make preparing for a potential pandemic a critical part of their emergency planning. Foremost is the fact that university students commonly live in close communal quarters (i.e., dormitories, sororities, etc.); these living arrangements typically include factors that can further the spread of illness such as sharing restrooms and eating in large-communal facilities. Moreover, college students do not typically maintain ideal hygiene and often engage in activities that can foster the spread of illnesses (i.e., sharing personal items, etc.). Accordingly, the introduction of a highly contagious illness, such as influenza, has the potential to spread rapidly, and within a short time, affect many. In addition, college campuses are known for uniting individuals from diverse countries—universities frequently invite visiting scholars, students often travel to unusual foreign lands—which can increase the likelihood of potentially introducing a novel illness.

Finally, another unique facet of universities is their system of centralized healthcare. Students (and sometimes staff) typically rely on the university student health center as their primary healthcare resource. In the event of a pandemic, the university student health center may be responsible for providing for the health and welfare of the majority of the students, and others, on their campus.

RESULTS

Prior to attending, university representatives were asked to submit a brief survey summarizing the characteristics of their campus and the steps they have completed to prepare for an influenza pandemic—of the 17 universities represented at the event, 14 university summaries were completed.

Student Profile: The universities represented at the summit are responsible for a large portion of the Los Angeles-area population—the combined enrollment from the 17 universities exceeds 200,000 students. In addition, large portions of those students live on-campus—on average well over 2,000 students live on each campus (Table 1). In addition, should travel be suspended, many students will likely be forced to

Table 1. Student Profile of Summit Participating Universities

Student Demographics	Total*	Mean	Median	Range (Min.–Max.)
Student Enrollment	184,198	14,169	8,300	350–35,625
Campus Residents	37,492	2,884	2,100	125–8,998
Out-of-State Students	19,933	1,661	1,272	100–5,000
International Students	14,362	1,104	500	10–6,881

* Since only 14 of the 17 schools completed the survey, the total numbers of student enrollment listed here underestimates the 17 universities represented at this event.

stay on campus—the universities attending this summit reported thousands of students are out-of-state residents or international students.

Emergency Planning: University representatives were asked whether their campus has established an all-hazards emergency plan—all 14 universities that responded noted that they have such a plan for their campus. However, when asked if their campus presently has a pandemic influenza plan (either separately or as an adjunct to their all-hazards plan) only one university said they did; eight universities stated that they had developed some pandemic planning, but their plans were presently incomplete; and, five universities stated that they presently had no pandemic influenza plan.

Student Health Center and Influenza Vaccination Profile: The participants were also asked the average number of primary care visits their student health center attends to yearly; on average, the health centers reported 16,678 student visits (median 10,000 visits), but there was also a very broad range in reported visits (range: 325 to 50,000 visits). The majority of the attending universities provide influenza vaccination for their students and others on campus. Only three of the 14 responding universities do not provide influenza vaccination. Of the remaining 11 schools, four provide vaccination free of charge, and seven charge a nominal fee. Of the universities that provide vaccination, most extend coverage to health center staff (76%), faculty (61%) and campus staff (53%).

Excerpts from the Tabletop Discussion: To guide the tabletop discussion, a series of four scenarios were developed describing different pandemic-influenza events that may affect universities (Table 2). The scenarios followed the World Health Organization's (WHO) pandemic phases[*] and were used to prompt discussion and debate regarding pandemic influenza planning and response. In addition, the scenarios helped to identify materials and resources that LAC DPH could provide to assist universities during these various events.

[*] As described on www.who.int/csr/disease/avian_influenza/phase/en/index.html.

The most common item that the representatives from the universities requested of LAC DPH was information and guidance—at every stage, the representatives noted that they would most value the expertise of LAC DPH to best respond to the scenario; the information should be easily accessed, simple to understand, and available in formats that can be easily disseminated for their use (i.e., in multiple languages, specific for students, specific for people who may travel, etc.).

Table 2. Tabletop Scenario and Suggestions for Pandemic Planning Assistance

Pandemic Phase:* Transmission Summary	Hypothetical Scenario Synopsis	Discussion Topics	Suggestions for Planning Assistance
3: No human-to-human transmission. No human cases in the US.	A student from your campus dies from influenza H5N1 infection while visiting family abroad. How do you quell fears and correct misinformation on your campus?	• What similar past campus events can provide guidance for responding to *this* event? • What are the key facets of this event that need to be included in campus communications? • What facets of pandemic planning should be instituted at this stage?	• Provide talking points (streamlined message maps) to inform and summarize the situation and frequent updates for posting on websites. • Provide updated contact information and relevant resources. • Develop educational materials and posters for health centers and other campus locations.
5: Large clusters of human-to-human transmission. Cases in the US. No cases on your campus.	Major human outbreaks from a novel influenza A virus have been identified. Outbreaks are occurring in neighboring cities, but not yet in LA, and not on your campus. What activities are paramount at this time?	• How can campuses health centers enhance their surveillance? • Should campuses activate their Incident Command Structure at this stage? • Should campuses stockpile antivirals and masks? If so, what are their strategies for their use? • What infection control practices should be recommended? Do campuses have methods of isolating sick students who reside on campus? • What methods of alternative education are available (i.e., web-based lessons, etc.)?	• In addition to the suggestions described for Scenario 1, establish a toll-free information hotline and create educational materials including responses to frequently asked questions and other talking points for campus representatives. • Develop posters to assist in identifying symptomatic patients and to request that they wear masks to limit the spread of illness due to coughing and/or sneezing. • Assist campuses in defining and implementing their Incident Command Structure to ensure ease of operations during the possible spread of illness to the campus.
6: Large clusters of human-to-human transmission. Cases in the US. Cases on your campus, including some of the summit participants.	The previously described pandemic-related illness has now reached the LA-area and your campus, including some of the representatives at the summit. How should activities change at this time?	• Because some of the summit participants were classified as "sick," their duties were described as well as any responsibilities that could not be performed if they were unexpectedly absent. • Other aspects of continuity of operations were discussed (i.e., what activities could and couldn't be redirected, what campus tasks and responsibilities were essential versus what could be postponed, etc.). • What supplies and/or preparations does your campus have for this type of emergency? • Would your campus be able to monitor absenteeism and illness? Are their any groups that may be overlooked?	• Since information and available resources will likely rapidly change, a centralized website (perhaps one with private access to maintain confidential information) would be a valuable tool for monitoring the epidemic and disseminating information including potential changes in treatment, affected groups, available materials.
Post-Pandemic: Peak in incident cases ended.	The first major wave of pandemic-related illness has subsided. Secondary waves of illness are likely. What activities should be conducted at this time?	• What resources are available on your campus that may be of use during this period (i.e., counseling services, etc.)? • At what point would your campus return to "business as usual"?	• Summary reports describing many facets of the pandemic (i.e., "lesson learned") would be valuable—especially if there are issues relevant to universities, their population and/or geographic area. • Guidance for how to prepare for future waves of illness and resources to assist in recovery and future response.

* World Health Organization Pandemic Phases (www.who.int/csr/disease/avian_influenza/phase/en/index.html).

DISCUSSION

Overall, the summit was very well-received—the participants were grateful for the opportunity to attend, were pleased with the materials and information that was provided, and requested future summits and updates as relevant. In response, LAC DPH developed a confidential university-specific web-portal to: store information (including the materials provided during the summit), allow universities to share information including their pandemic influenza plans, and post and respond to questions that may arise. In addition, LAC DPH has developed many educational materials such as posters to hang in student health centers to assist in identifying patients with novel respiratory viruses and to facilitate infection control. Finally, a follow-up summit was held six months later to provide updated information and materials.

A CASE OF PLAGUE IN URBAN LOS ANGELES

BACKGROUND

Plague was first recognized in the United States in San Francisco in 1900, and appeared in Los Angeles County in 1908. The disease was likely introduced to western United States ports via infected rats and humans who traveled on ships from Asia. Outbreaks in rats and subsequent human epidemics followed the introduction of plague to both San Francisco and Los Angeles. Since the early epidemics, sporadic human plague cases in California have been associated with epizootics (animal disease outbreaks), most commonly among California ground squirrels. No previous human cases have been associated with epizootics in wild rabbits in southern California. Despite the presence of sylvatic plague in many areas of the western United States, human infection in an urban setting without known risk behaviors is an urgent public health concern. The last previous human case of *Yersinia pestis* (*YP*) infection in Los Angeles County (LAC) occurred in 1984 in a veterinarian with established exposure to an ill cat.

CASE PRESENTATION

In April 2006, the Los Angeles County (LAC) Department of Public Health (DPH) received a report from an infectious disease (ID) physician of a positive blood culture for *Yersinia pestis* (*YP*) taken from a woman who lived in an urban area of Los Angeles. This 28 year-old previously healthy female was admitted to a local inpatient medical center with a three day history of fever and a severely painful right axillary swelling (bubo); she had no pulmonary symptoms. All chest radiographs were negative. Her preliminary diagnosis was “probable” abscess due to methcillin resistant *Staphylococcus aureus.*

On the third hospital day, the hospital laboratory reported to the clinician a presumptive identification of *YP* from an admission blood culture. Initially requiring aggressive therapy for shock, she improved enough after excision and drainage of the mass and antibiotic therapy to be discharged six days later. She recovered fully without sequelae. The case was queried in detail by ID consultants regarding any potential plague exposures. Beyond vaguely noting residential infestation with rodents and feral cats, she firmly denied any direct animal contact or travel outside of her densely urban locale. Within hours, LAC DPH was notified of the case by telephone and facsimile, which in turn notified the California state health department and the Federal Bureau of Investigation, because *YP* is category A bioterrorism agent.

METHODS

Case and contact interviews were conducted in person using a standardized questionnaire. The case and her family were interviewed repeatedly regarding potential exposures to animals and locations enzootic with *YP*; potential exposure sites were evaluated and animals were collected and tested for *YP*. Environmental investigations were conducted including interviews, general environmental assessment, trapping for animals, and serologic tests of animal serum. LAC Public Health Laboratory (PHL) tested the blood isolate by direct fluorescent antibody (DFA), polymerase chain reaction (PCR), and phage lysis. Sera from rabbits and deer mice were tested for plague at the California Department of Health Services (CDHS) Microbial Diseases Laboratory using a hemagglutinin assay (HA). Rabbit carcasses were tested for plague by the Centers for Disease Control and Prevention (CDC). Pulsed field gel electrophoresis (PFGE) analysis was done on human and animal *YP* isolates by CDC. Close contacts of the case and hospital staff were screened and offered prophylactic antibiotics.

RESULTS

In initial interview, the case denied any travel outside her immediate residence, except to walk her son to the local school. A second interview revealed that the case had visited a large park in Los Angeles that has many wild animals. In the early 1980s surveillance by LAC DPH in this park detected plague positive California mice, a ground squirrel and a Norway rat. The case was unsure of the dates she visited the park but thought it was 3 to 4 weeks prior to her onset, which was outside of the range for *YP* incubation period.

The blood isolate was positive for *YP* by DFA, PCR probes and the phage lysis test. Twelve hospital staff, including surgical residents and laboratory technicians, were screened by the hospital occupational health clinic and offered chemoprophylaxis because respiratory precautions were not taken during aspiration and excision of the bubo or during handling of the specimen in the laboratory. The household contacts were assessed by public health nursing staff—16 persons who lived on the premises were screened and offered antibiotic prophylaxis; 11 received doxycycline for 7 days, 5 received sulfamethoxazole/trimethoprim for 7 days; one person did not take prophylaxis as she was pregnant.

LAC DPH Environmental Health Vector Management staff assessed the property as not being good harborage for rats or ground squirrels, although feral cats were observed. Traps set for rodents inside and outside of the home yielded no competent *YP* vectors. Sera from two feral cats were tested and found negative for *YP*.

Day trapping activities in the local park frequented by the case yielded 34 California ground squirrels, which were flea infested. Serologic tests of squirrel sera showed no antibodies to *YP.*

After extensive re-questioning, the husband of the case reported that he and his friends hunted rabbits in the Mojave area of Kern County in early April 2006; the case did not go hunting and did not skin the rabbit but had handled the raw rabbit meat prior to cooking. On the hunting trip, the husband observed approximately 5 rabbits dead on the ground. A rabbit die-off in that region was also reported in May to California Department of Fish and Game by a local utility worker. Inspection of the hunting site by vector biologists from CDHS, Kern County Environmental Health and LAC Environmental Health revealed signs of a die-off at the time of hunting; five rabbits were obtained for testing. Trapping yielded 25 deer mice (*Peromyscus sp.*) and two jack rabbits. Five deer mice sera were positive by HA and one rabbit carcass was positive by DFA and culture for *YP*. PFGE results showed that the rabbit and human isolates had indistinguishable patterns and were unique when compared with 363 distinct patterns in the CDC database representing over 1,100 PFGE entries.

CONCLUSIONS

This confirmed human plague case was likely caused by handling the carcass of an infected wild rabbit collected in the area of a recent plague epizootic. Rabbits are known to transmit plague to humans, through either infected fleas or contact with blood when dressing a dead animal. Symptoms were compatible with bubonic plague and development of sepsis, but because the case resides in an urban area, plague was not in the initial differential diagnosis which resulted in inadequate infection control precautions during the hospital stay. Plague should be considered upon clinical assessment of persons who have been in an endemic area or have handled mammals taken from endemic areas. Repeated interviews may be needed to reveal risk factors when disease occurs in an non-endemic area. Public education regarding risk of plague in endemic areas is needed.

Bioterrorism was ruled out early in the investigation, as the case had limited exposure outside the home and an apparently natural infection. Nevertheless, the FBI was informed of the case and investigation as per protocol for cases infected with potential agents of bioterrorism.

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RISK FACTORS FOR INVASIVE GROUP A STREPTOCOCCAL DISEASE IN LOS ANGELES COUNTY, 2004-2006

BACKGROUND

Infection with group A streptococci (*Streptococcus pyogenes*) may result in several clinical presentations, ranging from non-invasive disease, such as strep throat, to invasive disease, where the bacteria invade a normally sterile site. Although readily treatable with antibiotics, severe invasive infections require prompt treatment to prevent devastating sequelae. Severe sequelae include necrotizing fasciitis (NF), otherwise known as “flesh eating disease,” and streptococcal toxic shock syndrome (STSS), which is characterized by a rapid onset of hypotension and multi-system involvement. Other clinical symptoms, often overlapping, include bacteremia, cellulitis, and pneumonia.

Invasive group A streptococcal (IGAS) infections cause substantial burden and mortality. In 2005, an estimated 10,400 cases and 1,350 deaths occurred in the United States [1]. The case fatality rate of IGAS infections is 12 to 13%, increasing to 30 to 80% in persons with severe infections [2]. Known risk factors include age, diabetes, Human Immunodeficiency Virus (HIV) infection, injection drug use (IDU), cardiovascular disease, and other chronic conditions [3].

The risk factors of Los Angeles County (LAC) IGAS cases were reviewed and compared to the prevalence of these risk factors in the general population to determine specific populations at greatest risk for IGAS infection. Based on this study in LAC, diabetes was the most prevalent risk factor observed in IGAS cases. Risk factors in older adults included chronic diseases, while risk factors in younger adults included alcoholism and blunt trauma. The prevalence of nosocomial IGAS infection, IDU, and HIV was lower in LAC IGAS cases when compared to national data.

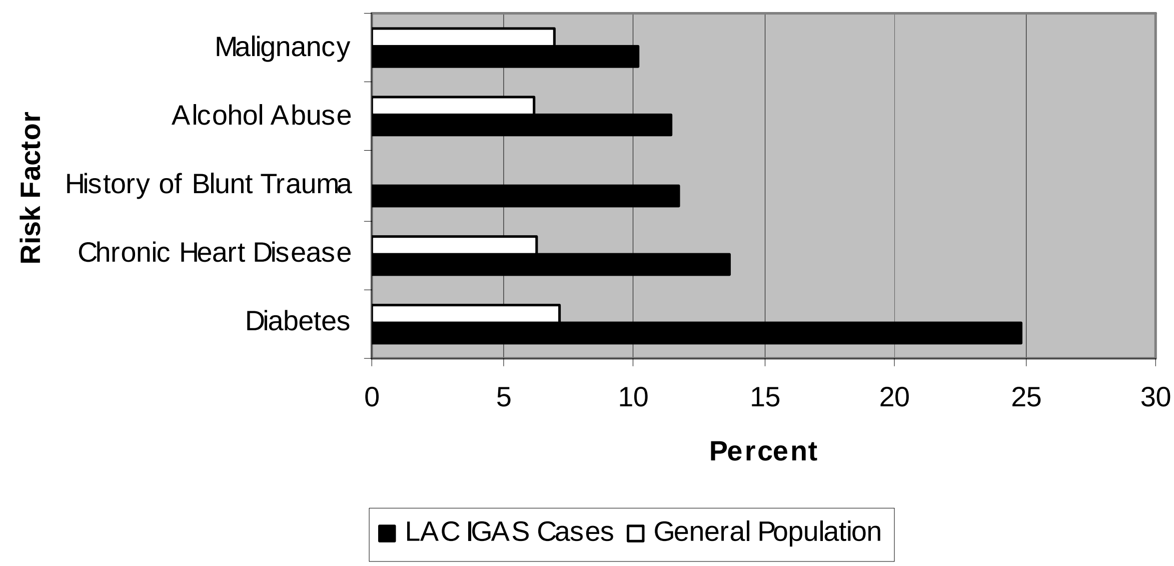
METHODS

IGAS is a reportable condition in LAC. An IGAS case is defined as a LAC resident who has *Streptococcus pyogenes* isolated from a normally sterile body site or from a non-sterile site if associated with STSS or NF. In 2004, a questionnaire was created to collect detailed demographic, clinical, and risk factor information for each reported case. IGAS cases reported and investigated by March 1, 2007 with disease onset from January 1, 2004 to December 31, 2006 were reviewed and analyzed to identify risk factors associated with IGAS infection. By univariate analysis, the prevalence of risk factors in LAC IGAS cases was compared to that of the general population, using data from multiple surveillance systems, including the Behavioral Risk Factor Surveillance System (BRFSS), the National Health Interview Survey (NHIS), the Los Angeles County Health Survey (LACHS), and US census.

RESULTS

From 2004 to 2006, a total of 516 cases were reported in LAC, with risk factor information available for 80% of the cases (n=410). From 2004 to 2005, the average incidence rate of IGAS infection in LAC was lower than the average rate reported in the United States (1.7 versus 3.5 cases per 100,000) [1]. However, the average case fatality rate from 2004 to 2005 was higher than the national average (18% versus 13%). During the three-year period, IGAS infection occurred more often in males (62%), adults aged 45 years and older (61%), Latinos (40%), and Whites (40%). Risk factors in older adults included chronic diseases, while risk factors in younger adults included alcoholism and blunt trauma. The most common risk factors reported included diabetes (25%), chronic heart disease (14%), blunt trauma (12%), alcohol abuse (12%), and malignancy (10%) (Figure 1). Specific trends and analyses are highlighted below.

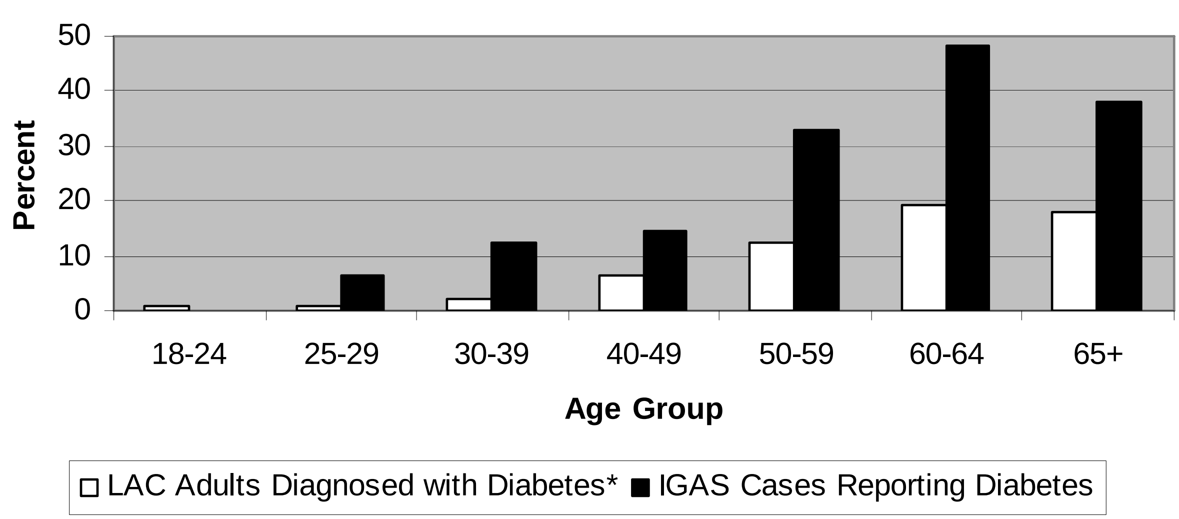
Figure 1. The Prevalence of Risk Factors in LAC IGAS Cases Compared to the General Population, 2004-2006



*No prevalence data available for blunt trauma in the general population.

Diabetes: From 2002 to 2003, seven percent of LAC adults reported being diagnosed with diabetes [4]. In contrast, the overall percentage of IGAS cases with diabetes was 3.5 times higher, as one in every four cases (25%) was also diabetic. The greatest number of IGAS cases with diabetes occurred in persons aged 45 years and older. However, in all racial groups (data not shown) and for persons over 25 years, the percentage of IGAS cases with diabetes was greater than the corresponding LAC diabetes prevalence by age (Figure 2) [4]. In particular, the percentage of IGAS infections in persons aged 25 to 39 years with diabetes was much higher than expected based on the underlying prevalence of diabetes in this age group.

Figure 2. Prevalence of Diabetes in LAC and IGAS Cases by Age



* 2002-03 Los Angeles County Health Survey; Office of Health Assessment and Epidemiology, Los Angeles County Department of Public Health.

Chronic Heart Disease and Malignancy: As the majority of IGAS infections occur in older adults, it is not surprising that many of the top reported risk factors include existing chronic diseases. Chronic heart disease was the second most reported risk factor (14%) and one in every three IGAS cases over 65 years reported this condition (33%). In contrast, the prevalence of coronary heart disease in the United States is lower (6%), with 18% of adults aged 65 to 74 years and 26% of persons over 75 years reporting coronary heart disease [5].

Overall, 10% of IGAS cases reported a malignancy compared to the national prevalence of 7% [5]. Interestingly, the percentage of IGAS cases with malignancy in LAC is higher in younger age groups and lower in older age groups when compared to the national cancer prevalence. Malignancy was reported in 7% of IGAS cases aged 20 to 44 years compared to the United States prevalence of 2% in those aged 18 to 44 years and was highest in persons aged 45 to 64 years (14% in LAC IGAS cases versus 8% in the United States). In IGAS cases 65 years and older, malignancy occurred in 13% of cases, which is lower than the national cancer prevalence of 19% in persons 65 to 74 years and 25% in persons 75 years and older. Additionally, the percent of female IGAS cases with malignancy (14%) was higher than both the national prevalence (7%) and the percentage of male IGAS cases with malignancy (8%).

Alcohol Abuse and Blunt Trauma: From 2004 to 2006, there has been an increase in the number of IGAS cases reporting a history of blunt trauma or alcohol abuse (data not shown). The majority of IGAS cases younger than 20 years have no risk factors reported (72%). However, a history of blunt trauma was the most reported risk factor in children IGAS cases aged 1 to 19 years, ranging from 32% in children aged 1 to 4 years to 27% in children aged 5 to 19 years.

In IGAS cases aged 20 to 44 years, alcohol abuse was reported more than any other risk factor (20%), more than double the percentage of Californians reporting heavy drinking in 2005 (10% in persons 18 to 24 years, 7% in persons 25 to 34 years, and 4% in persons 35 to 44 years) [6]. Among LAC adults, the percentage of males reporting alcohol abuse was more than 3 times higher than the percentage of females (16% versus 5%). Comparatively, in 2005, 8% of men and 5% of women reported heavy drinking in California [6].

Other: In contrast to what other studies have reported [3,7], HIV infection or IDU was infrequently observed in LAC IGAS cases. In one study, 7% of adult IGAS cases reported HIV infection and 24% reported a history of IDU [7]. In contrast, HIV and IDU were reported in 2% and 6% of LAC IGAS cases, respectively. In addition, only 2% of LAC IGAS cases were nosocomial, compared to 5% in the United States and 14% as reported in Canada [2].

DISCUSSION

By conducting IGAS surveillance in LAC, risk factors of persons presenting with IGAS infection can be identified which may assist in the timely diagnosis and treatment of these infections. With the recent increase in community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA), diagnosing IGAS in persons presenting with skin infections is challenging, especially since one of the most commonly prescribed antibiotics for CA-MRSA is not indicated for treating IGAS infections.

In LAC diabetes was the most prevalent risk factor, especially in adults aged 45 years and older. In older adults, risk factors for IGAS included chronic heart disease and malignancy, while a history of blunt trauma and alcohol abuse are reported more often in younger age groups. Physicians should recognize risk factors for IGAS infection and counsel their diabetic and older patients with chronic disease about their increased risk for IGAS and other infections. In addition, IGAS should be considered in younger patients, especially those with a history of trauma or alcoholism.

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USING SPATIAL SATSCAN™ STATISTICS IN SYNDROMIC SURVEILLANCE TO ENHANCE ILLNESS CLUSTER IDENTIFICATION

BACKGROUND

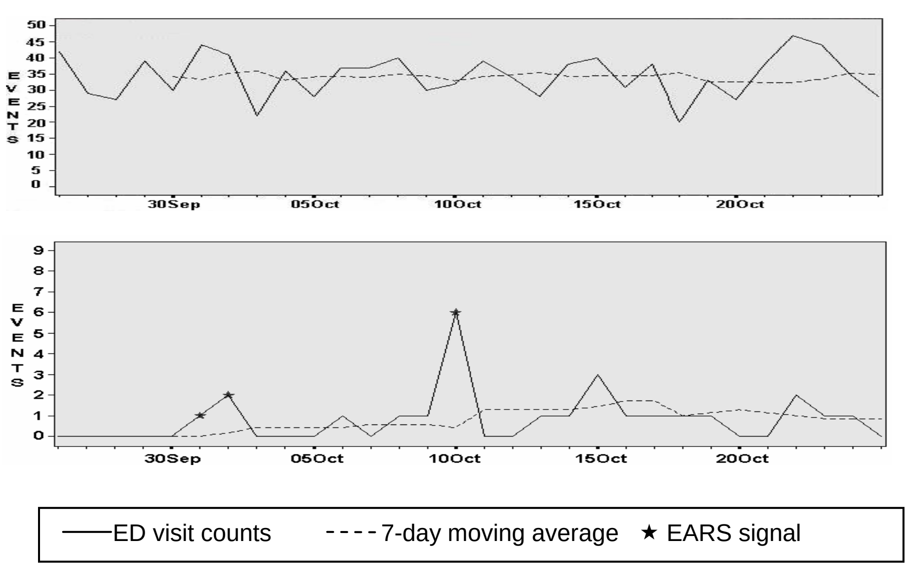
The Bioterrorism (BT) Surveillance Unit of the Los Angeles County (LAC) Department of Public Health, Acute Communicable Disease Control (ACDC) program conducts syndromic surveillance for early event detection and ongoing health events in near real-time. The syndromic surveillance system receives daily Emergency Department (ED) data representing over 40% of ED visits in LAC. These data are automatically classified into five major syndrome categories: gastrointestinal, neurological, rash, respiratory, and influenza-like illness. Syndrome-specific, ED-specific signals are generated when daily visits exceed thresholds determined by the Centers for Disease Control and Prevention (CDC)'s Early Aberration Reporting System (EARS) algorithm. In addition, SaTScan™ statistics are calculated using patient home zip codes to detect syndrome-specific spatial clusters. This report describes the utility of using both temporal and spatial analyses for assessing a rash signal and a neurological signal in 2006.

METHODS

Rash Signal: On October 10, 2006, syndromic surveillance detected a rash signal of six visits at one ED—two over the threshold (Figure 1). The small increase did not cause a substantial deviation in the total number of rash-related visits for all EDs. The line list, however, revealed that five of the patients resided in one zip code and synonymously cited chief complaints of "fever", "hair loss", and "rash." SaTScan™ analysis not only detected the rash cluster, but also served to emphasize that seeing five rash-related ED patients from this particular zip code on the specific date was extremely unusual (p=0.001) (Figure 2). Since the SaTScan™ cluster only included five rash patients, this implied that the sixth patient did not reside close enough to be included in the significant cluster. As also was insinuated by comparing chief complaints, this suggested that the sixth patient was probably an unrelated case.

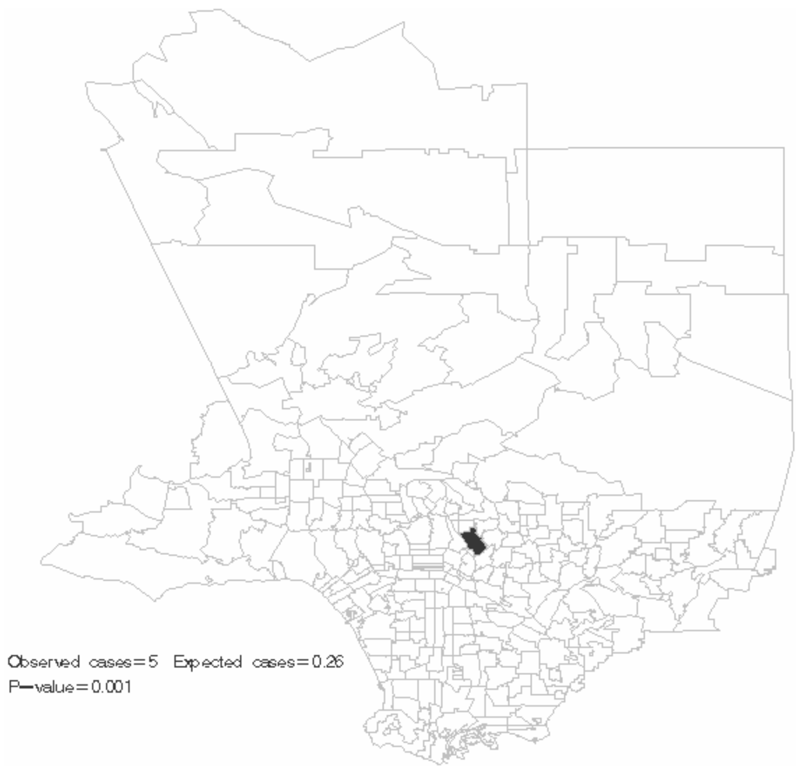
The subsequent ACDC Hospital Outreach Unit (HOU) investigation revealed that all five patients were diagnosed with scabies and were from the same household, consisting of a father, mother, and three children. All were treated and discharged with thorough scabies education and instructions to receive follow-up care from a primary medical physician.

Figure 1. Rash Syndrome ED Visit Counts for All Participating EDs (top) and in the Specific ED that Generated a Signal (bottom), with 7-Day Moving Average Trend Lines (dashed)



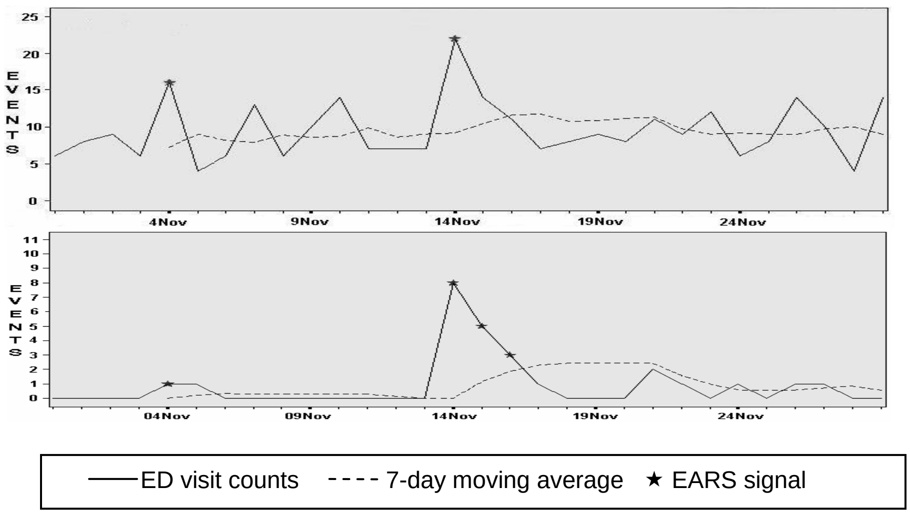
It is unknown how the patients were originally infected, but some or all had been symptomatic for weeks before visiting the ED. Although there was potential for a scabies outbreak, syndromic surveillance did not detect any rash-related unusual activity in subsequent days. The case was closed the following day, when rash syndrome counts returned to temporally and spatially normal levels.

Figure 2. Map of Rash Syndrome Cluster of Patient Residence Zip Codes on October 10, 2006 for All Participating EDs



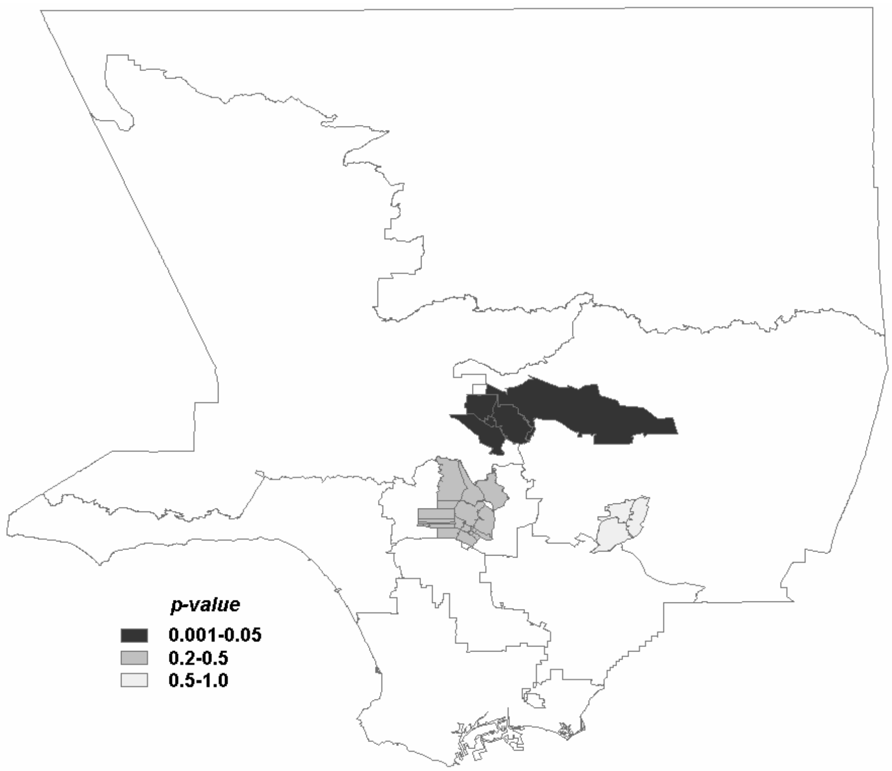
Neurological signal: On November 14, 2006, ACDC was alerted to a high school student who was symptomatic for meningitis. Syndromic surveillance subsequently detected five neurological syndrome visits over the threshold at one ED located in the vicinity of the high school (Figure 3). Unlike the scabies signal, this increase was large enough to cause a substantial aberration in the combined neurological syndrome counts across all EDs. Five of eight neurological syndrome patients were classified as meningitis-related due to having chief complaints which included “fever,” “headache,” or “meningitis.” Four patients cited “meningitis exposure.”

Figure 3. Neurological Syndrome ED Visit Counts for All Participating EDs (top) and in the ED Closest to the Event (bottom), with 7-Day Moving Average Trend Lines (dashed)



SaTScan™ also detected a substantial cluster of neurological syndrome patients from six adjacent zip codes in the vicinity of the high school on the same day (*p*=0.001) (Figure 4). Two additional neurological syndrome clusters were identified, albeit statistically weak (*p*≥0.2). Enhanced surveillance was thus expanded to neighboring EDs even if signals at those EDs were not detected. No additional meningitis-related visits were verified. Meanwhile, public health officials organized mass prophylaxis for all students potentially exposed to the index case. Eight more possibly meningitis-related visits to the same ED occurred over the next two days, of which five reported "meningitis exposure" and in some cases, specifically cited the high school in their chief complaint. The number of possible meningitis-related ED visits and SaTScan™ spatial statistics returned to normal on subsequent days, providing affirmation that a meningitis outbreak was successfully averted.

Figure 4. Map of Neurological Syndrome Cluster of Patient Residence Zip Codes on November 14, 2006 for All Participating EDs.



DISCUSSION

SaTScan™ is a tool for analyzing syndromic surveillance ED data that enhances ED-specific temporal (count-based) analysis. Since patient zip codes may not always correlate with which EDs were visited, SaTScan™ analysis may detect significant clustering in locations far from the hospital EDs at which temporal signals may be detected. It is also possible that SaTScan™ can detect substantial patient clusters when no ED-specific temporal signals are generated. This may occur if many people residing in an area become ill but choose to visit EDs in different locations.

Since SaTScan™ utilizes patient home zip code data, it may not be effective for detecting clusters if many zip code data are missing or if causative exposures took place far from home. However, when patient residence zip codes reflect the locations of their exposure, SaTScan™ may significantly improve the depiction of health events given by ED-specific temporal data alone. SaTScan™ not only corroborated the ED-specific rash signal, but also provided a quantitative basis with which the sixth rash patient could be excluded from the cluster. In the instance of the meningitis signal, SaTScan™ demonstrated its ability to help direct the locations to which surveillance should be expanded. Syndromic surveillance is thus amplified when SaTScan™ statistics are utilized in conjunction with ED-specific temporal signals to illustrate the spatial scope of health events and monitor subsequent days for secondary outbreaks.

SaTScan™ is a trademark of Martin Kulldorff. The SaTScanTM software was developed under the joint auspices of (i) Martin Kulldorff, (ii) the National Cancer Institute, and (iii) Farzad Mostashari of the New York City Department of Health and Mental Hygiene.

EMERGENCY DEPARTMENT SYNDROMIC SURVEILLANCE AND POPULATION-BASED HEALTH MONITORING IN LOS ANGELES COUNTY

BACKGROUND

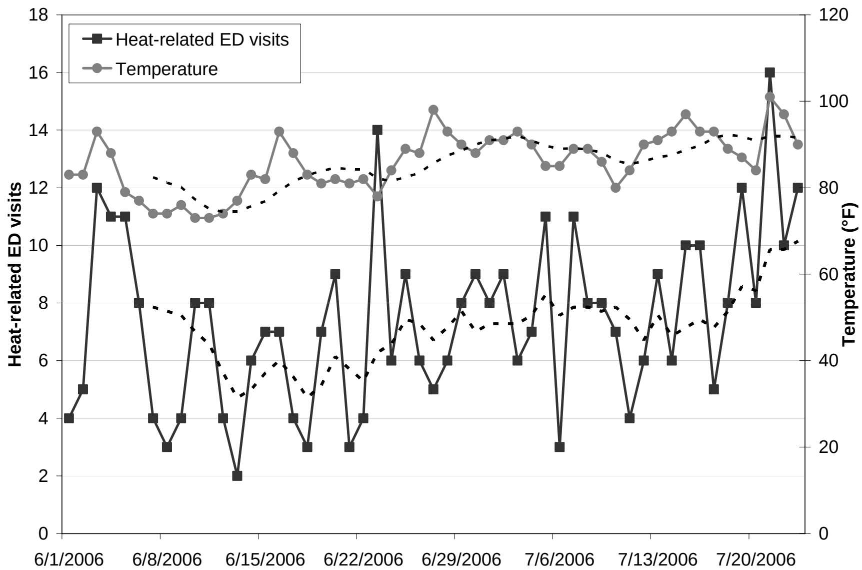
The Bioterrorism (BT) Surveillance Unit of the Los Angeles County (LAC) Department of Public Health (DPH), Acute Communicable Disease Control (ACDC) program analyzes Emergency Department (ED) data on a daily basis. The development of this system was primarily for early event detection and surveillance of ongoing health events in near real-time. Currently, the hospital EDs participating in syndromic surveillance monitor over 40% of the ED visits in LAC. Through an automated process, ED data from the previous day are collected and evaluated for aberrations in count and spatial distribution by utilizing the Centers for Disease Control and Prevention (CDC)'s Early Aberration Reporting System and SaTScan™ statistics. ED admitting chief complaints are classified using a SAS-based language processing code into five major syndrome categories: gastrointestinal, neurological, rash, respiratory, and influenza-like illness. Other complimentary systems used for surveillance include: Reddinet, which provides a daily tabulation of total ED visits from 65 participating hospital EDs, as well as ED-related hospital admittances, ICU admittances, and deaths; over the counter medicine sales provided by the Real-time Outbreak and Disease Surveillance laboratory; and LAC Coroners' mortality data. A daily report summarizing syndromic surveillance results and any signals generated is sent to key stakeholders seven days a week.

The syndromic surveillance system is automated, near real-time, population-based, and enables the surveillance of health indicators that would otherwise be difficult if not impossible for both hospital and ACDC staff. Typical usage of the system may be extended for various enhanced surveillance activities by creating additional syndrome categories tailored to specific illnesses or conditions. This report describes examples of how ED data was harnessed in 2006 to detect and monitor ED visits related to: 1) a summer heat wave, 2) a beach sewage spill, and 3) *E. coli* associated with contaminated bagged spinach. These examples demonstrate the flexibility of syndromic surveillance in capturing ED visits related to infectious and non-infectious, broadly defined and specific illnesses.

METHODS

Heat-related ED visits: While June 2006 was characterized by relatively normal temperatures for the month, July 2006 was the warmest July on record in many parts of California, during which a sustained heat wave caused a flood of ambulance calls, hospitalizations, and deaths due to heat-related illnesses [4]. Temperatures in LAC varied by region—downtown Los Angeles experienced 17 days during which maximum temperatures met or exceeded 90 degrees, while Woodland Hills experienced 24 days of triple digit temperatures [5].

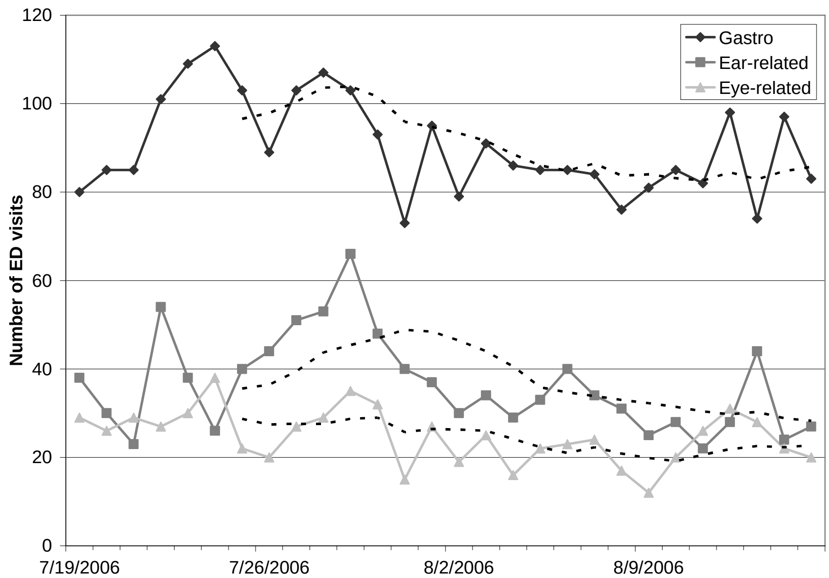
Figure 1. Daily Heat-Related ED Visits from 16 Hospital EDs in LAC, and Temperature Data from the Metro Area from June 1, 2006 to July 23, 2007, Overlaid by Seven-Day Moving Average Trend Lines



In order to estimate and track heat-related morbidity in LAC, the BT Unit monitored ED surveillance data from 16 hospitals in LAC to detect heat-related visits from June 1, 2006 to July 23, 2006. Patients with chief complaints containing key words such as: "heat exhaustion", "dehydration", "sun stroke", "hyperthermia", "overheat", "heat rash", and "feel hot" were classified as heat-related visits. Daily average temperatures for the LAC metro area were obtained from the website, weather.com and were analyzed for correlation with the number of heat-related ED visits. The ED data showed that the average number of heat-related visits per day substantially increased from 6.6 in June, to 8.3 in July (p=0.04). Daily heat-related ED visit counts were weakly correlated with temperature (r=0.35), although this may be in part because some heat-related ED visits may not have occurred on the day of exposure (Figure 1).

<u>Health monitoring following raw sewage spill</u>: On August 8, 2006, 20,000 to 30,000 gallons of raw sewage spilled near Ballona Creek and Marina Del Rey due to complications from a broken sewage line in Culver City, prompting the closure of two miles of beach [3]. While water tests indicated that bacteria returned to safe levels by August 10, 2006, beaches were not closed until 24 hours after the spillage ensued, exposing beachgoers to potentially high levels of bacteria. In response to this public health concern, syndromic surveillance was used to monitor increases in gastrointestinal, ear-related, or eye-related illnesses throughout LAC during this period, since these were the syndrome categories most likely to be experienced by those exposed to the sewage spill. Visits with chief complaints such as "otitis", "ear pain", and "ear ache" were classified into the ear-related category, while visits with chief complaints such as "conjunctivitis", "eye pain", "pink eye", and "red eye" were classified as eye-related visits. Patients under two years of age were excluded.

Figure 2. Daily ED visits in 17 Syndromic Surveillance Participant ED Hospitals from July 19, 2006 to August 15, 2006. Historical Data (Data Prior to August 8, 2006) was Retrospectively Plotted for Baseline Comparison, along with Seven-Day Moving Average Trend Lines



There did not appear to be any increasing trend in ear-related, eye-related, or gastrointestinal ED visits subsequent to the sewage spill (Figure 2). Although it is possible that the syndromic surveillance was not sensitive enough to detect a change in ED visits resulting from the spill, the simplest explanation is that a substantial increase in morbidity did not occur. Given that the sewage was diluted once entering the ocean, and given that many viruses are unstable in an ocean environment, it was unlikely that many individuals would develop illnesses from their exposure, much less develop illnesses so severe as to necessitate visits to the ED. Corroborating evidence of this was provided by the LAC Environmental Health Division's Food and Milk Program, which interviewed 23 of 30 individuals who submitted foodborne illness reports during the days following the sewage spill. All denied visiting LAC beaches within three days prior to the onset of their illness.

Spinach outbreak: A widely publicized national *E. coli* outbreak related to spinach consumption resulted in 204 infected individuals in 26 states as of October 18, 2006, in which there were 102 hospitalizations and three fatalities [1,2]. Although the epidemiologic investigation concluded that contaminated spinach was not distributed within California, the BT Unit proceeded to conduct surveillance of any ED visits in LAC that were potentially related to the outbreak. Syndromic surveillance analyzed data from September 15, 2006 to December 11, 2006. The chief complaint and diagnosis fields were tagged if they contained the words "*E. coli*" or "Spinach" or the ICD-9 codes for *E. coli*. In all, the syndromic surveillance system detected 13 spinach outbreak-related ED visits in seven EDs. Of these, eight visits were reported in September, followed by two visits each in October and November, and only one visit in December. No additional suspect ED visits were subsequently found. All 13 patients were followed up by the ACDC foodborne unit, and none were diagnosed with *E. coli* infection. Eventually, only two residents of Shasta and Riverside counties in California were confirmed positive for the *E. coli* strain related to the outbreak [1,6].

DISCUSSION

While syndromic surveillance was initially developed for early detection of bioterrorism events, it has also been proven to be useful as an overall monitor of the public's health. No other system was or is now capable of providing a depiction of the public health impact of the 2006 summer heat wave, sewage spill, and the multi-state *E. coli* outbreak on LAC residents; much less a temporal and spatial statistics-based assessment utilizing as much population-based data as was generated by the system, and in as near to real-time. Syndromic surveillance is also the only system capable of consistently generating and analyzing syndrome specific data without requiring additional steps for the hospital EDs once connected to the system. This may be especially important during a large-scale outbreak, for which classic methods of surveillance data collection (e.g., mandating the reporting of specific illnesses), may be time and resource expensive for both reporting medical providers and County epidemiologists who must manually tabulate incoming data.

Syndromic surveillance is not without its imperfections. Mild illnesses are difficult to capture, since they may not cause people to visit EDs. Other underlying medical conditions may cause symptoms similar to those of the illness of interest, and since only some hospital EDs transmit diagnosis data or provide a key that can be used to relocate patient records, it is currently impossible to completely eliminate misclassifications of syndrome categories. However, this should not affect the system's capability to assess changes in incidence, assuming that the same percentage of data is misclassified at any time when querying the same syndrome definition. For instance, assuming that the baseline number of heat-related ED visits established in June was applicable for July as well, syndromic surveillance was able to detect an increase in heat-related ED visits for the month, which corresponded with the increase in temperature. Syndromic surveillance also served in this capacity to provide assurances that the risks for potential outbreak events caused by the sewage spill and *E. coli* spinach contamination were stabilized.

Syndromic surveillance offers an easily configured and rapidly accessible population-based surveillance mechanism for illnesses that may otherwise not be rapidly quantifiable in LAC and surpasses other systems that cannot generate as much data as is collected and analyzed in as timely a manner to detect and monitor specific illnesses.

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VARICELLA OUTBREAK EPIDEMIOLOGY IN AN ACTIVE SURVEILLANCE SITE, 1995-2005

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ABSTRACT

We report on varicella disease outbreaks identified in an active surveillance site from 1995 to 2005 and describe trends and characteristics of the outbreaks. Cases of varicella were reported to the active surveillance project and outbreaks were defined retrospectively as ≥5 varicella cases epidemiologically linked to a common setting occurring within one incubation period. Outbreaks were grouped by calendar year. From 1995-1998 to 2002-2005, varicella outbreaks significantly decreased in number, from 236 to 46 ($p<0.001$); in size, median number of cases per outbreak from 15 to 9 ($p<0.001$); and duration, from 44.5 days to 30 days ($p<0.001$). The median age of outbreak cases increased from 6 to 9 years ($p<0.001$). The one-dose varicella vaccination program has been successful with decreasing the number of outbreaks and cases; however, challenges remain with controlling outbreaks among vaccinated persons and targeting vaccination efforts to susceptible older age groups.

This article has been accepted for publication in the Journal of Infectious Diseases supplement devoted to varicella.