LYME DISEASE

CRUDE DATA	
Number of Cases	5
Annual Incidence [a]	
LA County [b]	0.05
California [c]	0.14
United States [c]	7.95
Age at Diagnosis	
Mean	29.8
Median	20
Range	3–82 years

[a]Cases per 100,000 population.

[b]Rates calculated based on less than 19 cases or events are considered unreliable.

[c]Calculated from Final 2014 Reports of Nationally Notifiable Infectious Diseases. MMWR 64(36):1019–1033.

DESCRIPTION

Lyme disease (LD) is caused by the spirochete *Borrelia burgdorferi*, which is transmitted to humans by the bite of *Ixodes* ticks; the vector in the Pacific coast states is the western blacklegged tick (*Ixodes pacificus*). This disease is rarely acquired in LAC due to the low prevalence of Lyme-infected ticks. Between 1985 and 2013, 0.2% of adult ticks and 0% of nymphal ticks tested positive for Lyme.[1] Most reported cases have been acquired in known endemic regions in the US. The most common clinical presentation is a distinctive circular rash called erythema migrans (EM). When EM is not present, other early symptoms such as fever, body aches, headaches, and fatigue are often unrecognized as indicators of LD. If untreated, patients may develop late stage symptoms such as aseptic meningitis, cranial neuritis, cardiac conduction abnormalities 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 antibiotics. Currently, there is no vaccine.

For purposes of surveillance, the CDC requires a confirmed case of LD to have:

- Physician-diagnosed EM that is at least 5 cm in diameter with known tick exposure (laboratory evidence is necessary without tick exposure), or
- At least one late manifestation of LD with supporting laboratory results.

Laboratory criteria for case confirmation include a positive culture for *B. burgdorferi* or demonstration of diagnostic IgM or IgG to *B. burgdorferi* in serum or cerebral spinal fluid. A coalition of several public health and medical organizations recommends a two-step serologic testing procedure for LD: an initial enzyme immunoassay or immunofluorescent antibody screening test, and if positive or equivocal, followed by IgM and IgG Western immunoblotting. [2]

Avoiding tick bite exposure is the primary means of preventing LD. 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 to 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; tucking shirts into pants; using tick repellant; 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 controlling ticks on pets.

2014 TRENDS AND HIGHLIGHTS

- LAC continues to document much lower rates (less than 1 per 100,000) than the national rate. Only 5 cases were reported in LAC in 2014 and since 2014, annual totals ranged between 1 and 11 reported cases.
- Cases were reported only through July, and occurred sporadically. Most cases in the previous 5 years occurred during the summer months (Figure 2).
- Most cases reported an outdoor exposure outside of LAC, but within the US (n=3, 60%) (Figure 3). These cases recalled exposure in New England and Midwestern states. Two cases with no travel outside California, reported outdoor exposure in Malibu and Torrance, but did not recall a tick bite. Only one case recalled a tick bite, which occurred in Maine.

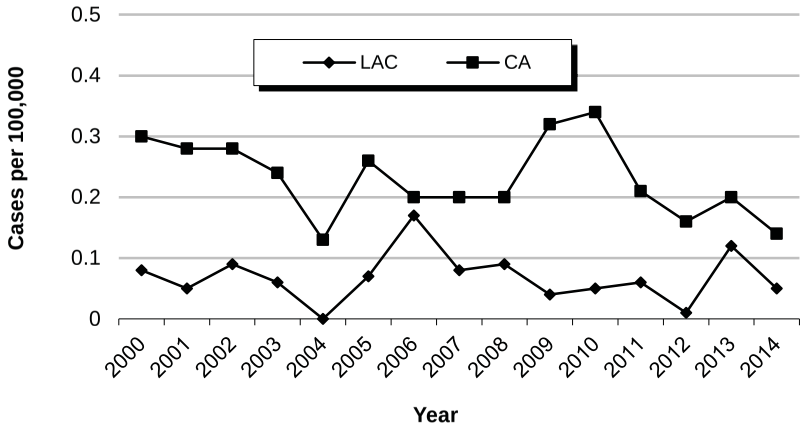
- There were 720 suspected cases of Lyme disease reported to LAC DPH in 2014, up from 564 reported in 2013. The large majority of these were reported as a result of positive laboratory results. Less than 1% of these reports met the CDC case definition for a confirmed case. The number of suspected cases of Lyme reported has increased since Lyme became laboratory reportable in 2006. However, the number of cases confirmed has remained relatively stable (Figure 1 and Figure 3). It is highly recommended that testing for Lyme occur within the context of appropriate clinical symptoms and outdoor exposure.

Reported Lyme Disease Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
Los Angeles County, 2010-2014

	2010 (N=5)			2011 (N=6)			2012 (N=1)			2013 (N=11)			2014 (N=5)		
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
1-4	0	-	-	0	-	-	0	-	-	1	9.1	-	1	20.0	-
5-14	1	20.0	-	0	-	-	0	-	-	3	27.3	-	1	20.0	-
15-34	2	40.0	-	1	16.7	-	0	-	-	5	45.5	-	2	40.0	-
35-44	1	20.0	-	0	-	-	0	-	-	0	-	-	0	-	-
45-54	0	-	-	3	50.0	-	0	-	-	1	9.1	-	0	-	-
55-64	1	2.0	-	1	16.7	-	0	-	-	1	9.1	-	0	-	-
65+	0	-	-	1	16.7	-	1	100.0	-	0	-	-	1	20.0	-
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Race/Ethnicity															
Asian	0	-	-	0	-	-	0	-	-	0	-	-	1	20.0	-
Black	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
Hispanic	1	20.0	-	0	-	-	0	-	-	0	-	-	0	-	-
White	4	80.0	-	6	100.0	-	1	100.0	-	8	72.7	-	3	60.0	-
Other	0	-	-	0	-	-	0	-	-	1	9.1	-	0	-	-
Unknown	0	-	-	0	-	-	0	-	-	1	9.1	-	1	20.0	
SPA															
1	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-
2	0	-	-	2	33.3	-	1	100.0	-	3	27.3	-	0	-	-
3	0	-	-	1	16.7	-	0	-	-	0	-	-	1	20.0	-
4	2	40.0	-	0	-	-	0	-	-	2	18.2	-	1	20.0	-
5	2	40.0	-	3	50.0	-	0	-	-	4	36.4	-	2	40.0	-
6	1	20.0	-	0	-	-	0	-	-	0	-	-	0	-	-
7	0	-	-	0	-	-	0	-	-	1	9.1	-	0	-	-
8	0	-	-	0	-	-	0	-	-	1	9.1	-	1	20.0	-
Unknown	0	-	-	0	-	-	0	-	-	0	-	-	0	-	-

*Rates were not calculated because rates calculated based on less than 19 cases or events are considered unreliable.

Figure 1. Incidence Rates of Lyme Disease LAC* and CA, 2000-2014



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

Figure 2. Reported Lyme Disease Cases by Month of Onset LAC, 2014

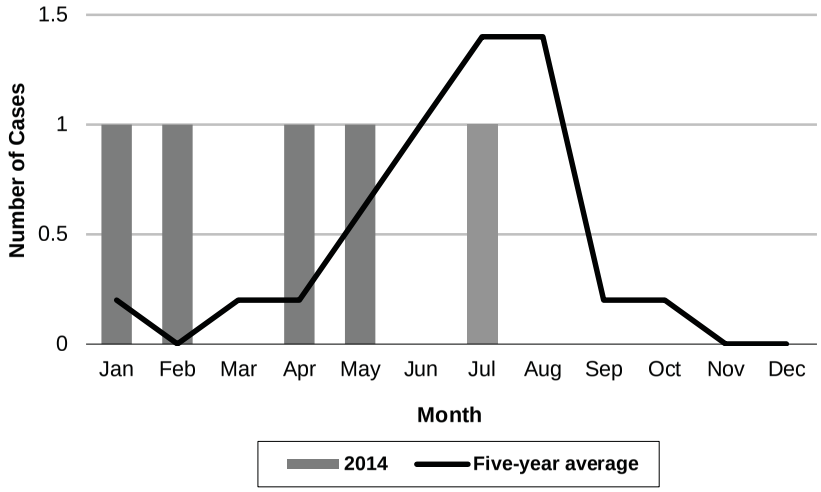
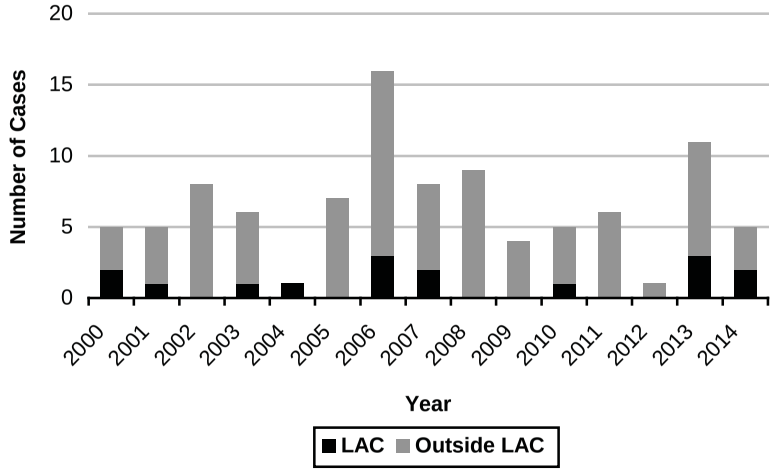


Figure 3. Locations of Tick and Outdoor Exposure in Lyme Disease Cases LAC, 2000-2014



LYME DISEASE

CRUDE DATA	
Number of Cases	11
Annual Incidence [a]	
LA County [b]	0.12
California	0.24
United States	8.67
Age at Diagnosis	
Mean	26.7
Median	28
Range	2-61

[a]Cases per 100,000 population.
[b]Rates calculated based on less than 19 cases or events are considered unreliable.
[c]Calculated from Final 2013 Reports of Nationally Notifiable Infectious Diseases. MMWR 63(32):702-716.

DESCRIPTION

Lyme disease (LD) is caused by the spirochete *Borrelia burgdorferi*, which is transmitted to humans by the bite of *Ixodes* ticks; the vector in the Pacific coast states is the western blacklegged tick (*Ixodes pacificus*). This disease is rarely acquired in Los Angeles County (LAC); most reported cases have been acquired in known endemic regions in the United States (US). The most common clinical presentation is a distinctive circular rash called erythema migrans (EM). When EM is not present, other early symptoms such as fever, body aches, headaches, and fatigue are often unrecognized as indicators of LD. If untreated, patients may develop late stage symptoms such as aseptic meningitis, cranial neuritis, cardiac conduction abnormalities 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 antibiotics. Currently, there is no vaccine.

For purposes of surveillance, the Centers for Disease Control and Prevention (CDC) require a confirmed case of LD to have:

- Physician-diagnosed EM that is at least 5 cm in diameter with known tick exposure (laboratory evidence is necessary without tick exposure), or
- At least one late manifestation of LD with supporting laboratory results.

Laboratory criteria for case confirmation include a positive culture for *B. burgdorferi* or demonstration of diagnostic IgM or IgG to *B.* burgdorferi in serum or cerebral spinal fluid. A coalition of several public health and medical organizations recommends a two-step serologic testing procedure for LD: an initial enzyme immunoassay or immunofluorescent antibody screening test, and if positive or equivocal, followed by IgM and IgG Western immunoblotting.[1]

Avoiding tick bite exposure is the primary means of preventing LD. 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 to 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; tucking shirts into pants; using tick repellant; 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 controlling ticks on pets.

2013 TRENDS AND HIGHLIGHTS

- The national incidence rose as high as 13.4 cases per 100,000 in 2009 and dropped to 7.1 cases per 100,000 by 2012. Though LAC documented the highest number of cases this year since 2006 (N=16) with 11 confirmed cases, the incidence in LAC in 2013 was 0.12 per 100,000 and remains well below the national and state rates (Figure 1).
- The peak number of cases occurred in August (n=5). Most cases occurred during the late spring and summer months, following the seasonal trend from the previous 5 years (Figure 2).
- Most cases reported an outdoor exposure outside of LAC (n=8, 73%) including several in the New England area, two cases in the

[1]Recommendations for Test Performance and Interpretation from the Second National Conference on Serologic Diagnosis of Lyme Disease. MMWR August 11, 1995/44(31);590-591, http://www.cdc.gov/mmwr/preview/mmwrhtml/00038469.htm.

Midwest and one case in Europe (Figure 3). Only one case recalled a tick bite, which occurred in LAC. The patient reports a tick bite on his property, located in the Hollywood-Wilshire Health District. The remaining two cases with LAC outdoor exposure reported activity in West and West Valley HDs, but did not recall insect bites.

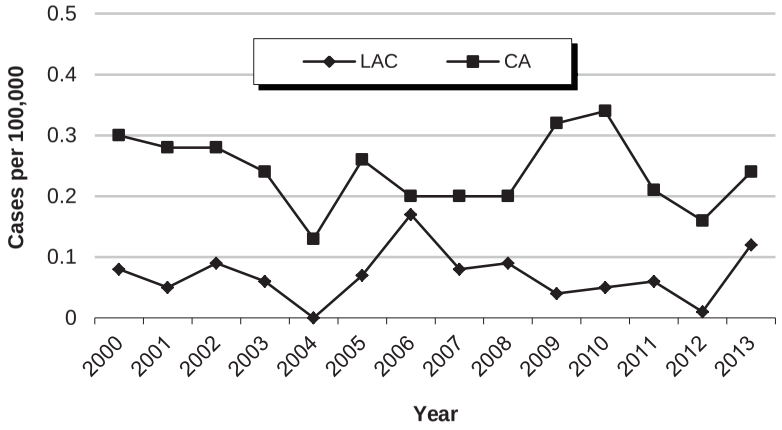
- There were 564 suspected cases of Lyme disease reported to LAC DPH in 2013. The large majority of these were reported as a result of positive laboratory results. Only 2% of these reports met the CDC case definition for a confirmed case. The number of suspected cases of Lyme reported has increased since Lyme became laboratory reportable in 2006. However, the number of cases confirmed has remained relatively stable (Figure 1 and Figure 3). It is highly recommended that testing for Lyme occur within the context of appropriate clinical symptoms and outdoor exposure in areas with known endemicity.

Reported Lyme Disease Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
Los Angeles County, 2009-2013

	2009 (N=4)			2010 (N=5)			2011 (N=6)			2012 (N=1)			2013 (N=11)		
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
1-4	0	0.0		0	0.0		0	0.0		0	0.0		1	9.1	
5-14	1	25.0		1	20.0		0	0.0		0	0.0		3	27	
15-34	0	0.0		2	40.0		1	16.7		0	0.0		5	45.5	
35-44	2	50.0		1	20.0		0	0.0		0	0.0		0	0.0	
45-54	0	0.0		0	0.0		3	50		0	0.0		1	9.1	
55-64	1	25.0		1	20.0		1	16.7		0	0.0		1	9.1	
65+	0	0.0		0	0.0		1	16.7		1	100		0	0.0	
Unknown	0	0.0		0	0.0		0	0.0		0	0.0				
Race/Ethnicity															
Asian	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
Black	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
Hispanic	0	0.0		1	20.0		0	0.0		0	0.0		0	0.0	
White	4	100		4	80.0		6	100		1	100		8	72.7	
Other	0	0.0		0	0.0		0	0.0		0	0.0		1	9.1	
Unknown	0	0.0		0	0.0		0	0.0		0			1	9.1	
SPA															
1	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
2	1	25.0		0	0.0		2	33.3		1	100		3	27.3	
3	0	0.0		0	0.0		1	16.7		0	0.0		0	0.0	
4	0	0.0		2	40.0		0	0.0		0	0.0		2	18.2	
5	1	25.0		2	40.0		3	50.0		0	0.0		4	36.4	
6	1	25.0		1	20.0		0	0.0		0	0.0		0	0.0	
7	0	0.0		0	0.0		0	0.0		0	0.0		1	9.1	
8	1	25.0		0	0.0		0	0.0		0	0.0		1	9.1	
Unknown	0	0.0		0	0.0		0	0.0		0			0	0.0	

*Rates were not calculated because rates calculated based on less than 19 cases or events are considered unreliable.

Figure 1. Incidence Rates of Lyme Disease LAC* and CA, 2000-2013



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

Figure 2. Reported Lyme Disease Cases by Month of Onset LAC, 2013

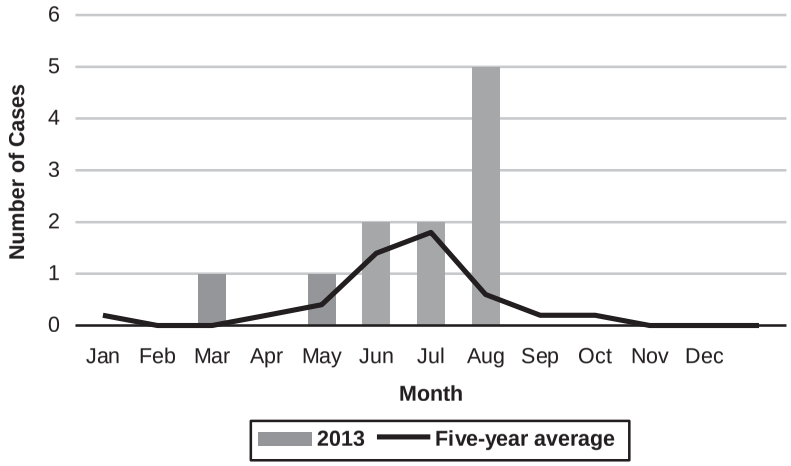
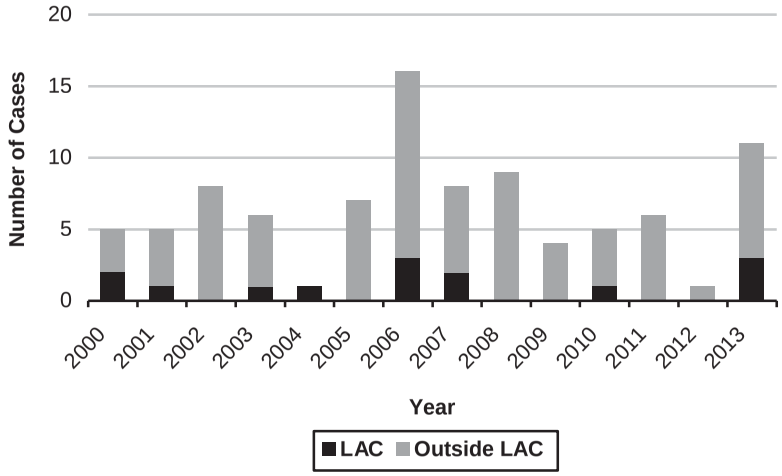


Figure 3. Locations of Tick and Outdoor Exposure in Lyme Disease Cases LAC, 2000-2013



LYME DISEASE

CRUDE DATA	
Number of Cases	1
Annual Incidence [a]	
LA County [b]	0.01
California	0.16
United States	7.1
Age at Diagnosis	
Mean	72
Median	72
Range	--

[a] Cases per 100,000 population.
[b] Rates calculated based on less than 19 cases or events are considered unreliable.
[c] Calculated from Final 2012 Reports of Nationally Notifiable Infectious Disease. MMWR 62(33);669-682.

DESCRIPTION

Lyme disease (LD) is caused by the spirochete *Borrelia burgdorferi*, which is transmitted to humans by the bite of *Ixodes* ticks; the vector in the Pacific coast states is the western blacklegged tick (*Ixodes pacificus*). This disease is rarely acquired in Los Angeles County (LAC); most reported cases have been acquired in known endemic regions in the United States (US). The most common clinical presentation is a distinctive circular rash called erythema migrans (EM). When EM is not present, other early symptoms such as fever, body aches, headaches, and fatigue are often unrecognized as indicators of LD. If untreated, patients may develop late stage symptoms such as aseptic meningitis, cranial neuritis, cardiac conduction abnormalities 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 antibiotics. Currently, there is no vaccine.

For purposes of surveillance, the Centers for Disease Control and Prevention (CDC) require a confirmed case of LD to have:

- Physician-diagnosed EM that is at least 5 cm in diameter with known tick exposure (laboratory evidence is necessary without tick exposure), or
- At least one late manifestation of LD with supporting laboratory results.

Laboratory criteria for case confirmation include a positive culture for *B. burgdorferi* or demonstration of diagnostic IgM or IgG to *B.* burgdorferi in serum or cerebral spinal fluid. A coalition of several public health and medical organizations recommends a two-step serologic testing procedure for LD: an initial enzyme immunoassay or immunofluorescent antibody screening test, and if positive or equivocal, followed by IgM and IgG Western immunoblotting.[1]

Avoiding tick bite exposure is the primary means of preventing LD. 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 to 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; tucking shirts into pants; using tick repellant; 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 controlling ticks on pets.

2012 TRENDS AND HIGHLIGHTS

- The national incidence rose as high as 13.4 cases per 100,000 in 2009 and dropped to 7.8 cases per 100,000 by 2011. The incidence in LAC in 2012 was 0.01 per 100,000 and has remained well below the national and state rates (Figure 1).
- The single confirmed case reported tick bite exposure in a highly endemic LD region outside of LAC (Massachussetts) (Figure 3).

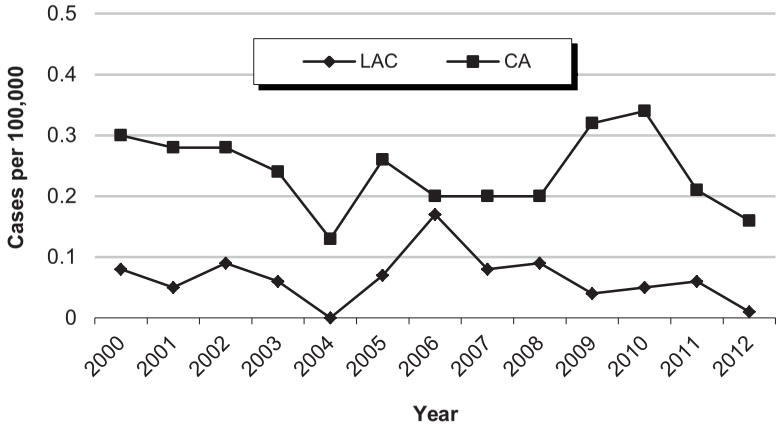
[1] Recommendations for Test Performance and Interpretation from the Second National Conference on Serologic Diagnosis of Lyme Disease. MMWR August 11, 1995/44(31);590-591, http://www.cdc.gov/mmwr/preview/mmwrhtml/00038469.htm.

Reported Lyme Disease Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
Los Angeles County, 2008-2012

	2008 (N=9)			2009 (N=4)			2010 (N=5)			2011 (N=6)			2012 (N=1)		
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
1-4	2	22.2		0	0.0		0	0.0		0	0.0		0	0.0	
5-14	1	11.1		1	25.0		1	20.0		0	0.0		0	0.0	
15-34	1	11.1		0	0.0		2	40.0		1	16.7		0	0.0	
35-44	1	11.1		2	50.0		1	20.0		0	0.0		0	0.0	
45-54	3	33.3		0	0.0		0	0.0		3	50		0	0.0	
55-64	0	0.0		1	25.0		1	20.0		1	16.7		0	0.0	
65+	1	11.1		0	0.0		0	0.0		1	16.7		1	100	
Unknown	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
Race/Ethnicity															
Asian	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
Black	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
Hispanic	0	0.0		0	0.0		1	20.0		0	0.0		0	0.0	
White	9	100		4	100		4	80.0		6	100		1	100	
Other	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
Unknown	0	0.0		0	0.0		0	0.0		0	0.0		0		
SPA															
1	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
2	2	22.2		1	25.0		0	0.0		2	33.3		1	100	
3	0	0.0		0	0.0		0	0.0		1	16.7		0	0.0	
4	1	11.1		0	0.0		2	40.0		0	0.0		0	0.0	
5	4	44.4		1	25.0		2	40.0		3	50.0		0	0.0	
6	0	0.0		1	25.0		1	20.0		0	0.0		0	0.0	
7	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
8	2	22.2		1	25.0		0	0.0		0	0.0		0	0.0	
Unknown	0	0.0		0	0.0		0	0.0		0	0.0		0		

*Rates were not calculated because rates calculated based on less than 19 cases or events are considered unreliable.

Figure 1. Incidence Rates of Lyme Disease LAC* and CA, 2000-2012



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

Figure 2. Reported Lyme Disease Cases by Month of Onset LAC, 2012

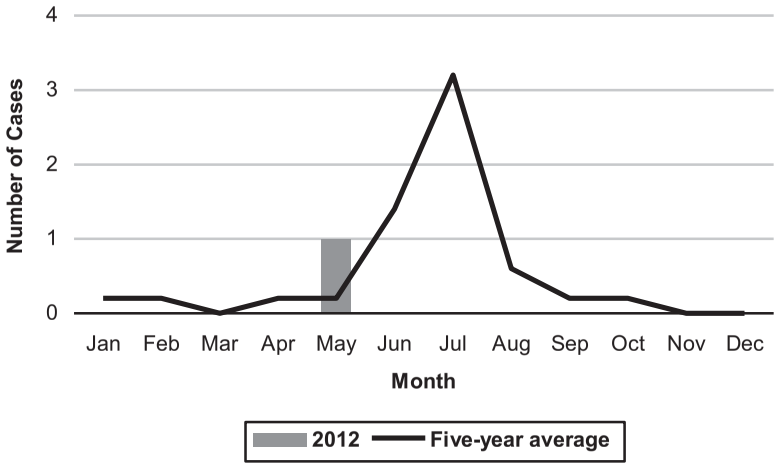
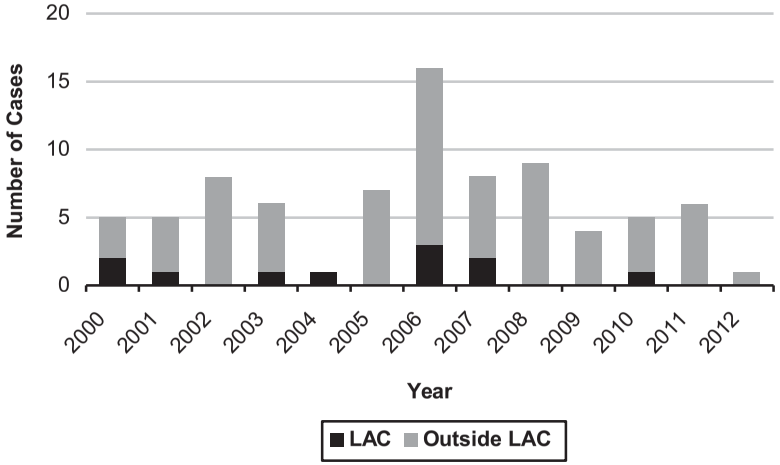


Figure 3. Locations of Tick and Outdoor Exposure in Lyme Disease Cases LAC, 2000-2012



LYME DISEASE

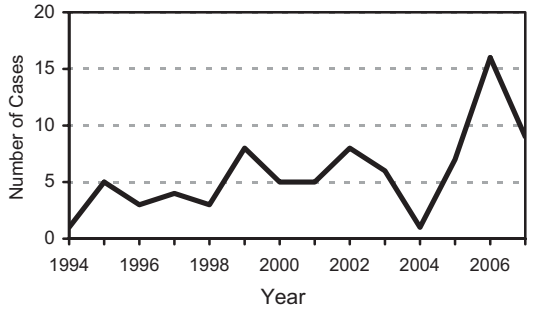
CRUDE DATA	
Number of Cases	9
Annual Incidence[a]	
LA County	0.09[b]
California	0.20[c]
United States	8.24[c]
Age at Diagnosis	
Mean	35
Median	31
Range	11-76 years

[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] Rates taken from CDC Lyme Disease page (http://www.cdc.gov/ncidod/dvbid/lyme/index.htm).

Figure 1
Lyme Disease Cases
by Year of Onset
LAC, 1994–2007



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. 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. Nevertheless, LD has been well documented to occur in counties throughout the state of California (CA) 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. 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 antibiotics. Currently, there is no vaccine.

For purposes of surveillance, the Centers for Disease Control and Prevention (CDC) requires a confirmed case of LD to have documented EM diagnosed by a healthcare provider that is at least 5cm in diameter or at least one late manifestation of LD 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; LD should primarily be diagnosed by a healthcare provider's consideration of the clinical presentation and history of tick exposure.

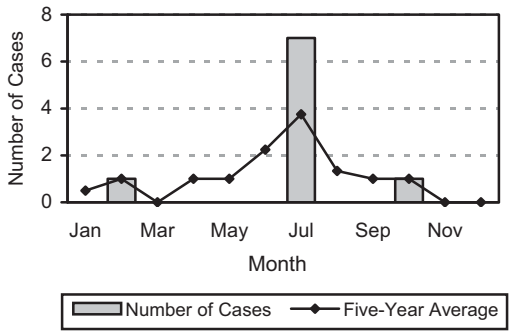
DISEASE ABSTRACT

- In 2007, the number of cases reported that met CDC surveillance criteria (n=9) dropped from an all-time high of 16 in 2006.
- The majority of cases (75%) 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: In 2007, only 9 Lyme cases met the CDC case definition, resembling numbers reported in years past. In 2006, there was nearly a 129% increase in cases (n=16) from the previous year (Figure 1). The number of cases reported with a possible exposure within LAC (n=2) continues to be low (Figure 3). Since 1994, the number of cases with possible exposure within LAC has ranged from 0 to 5.

Seasonality: The peak number of cases occurred in July (n=7) (Figure 2). As seen in the five-year average, July is the most commonly reported month of onset. 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.

Figure 2
Lyme Disease Cases
by Month of Onset
LAC, 2007



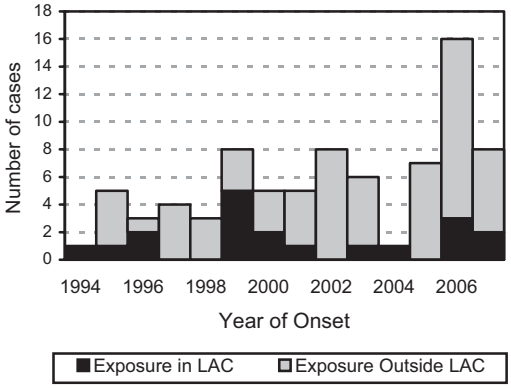
Age: The mean age of cases in 2007 was 35 (median = 31 years) with a range of 11-76 years old. Nationally, LD is most common among persons aged 5-19 years and 30 years and older.

Sex: The male to female ratio was 1:2. Nationally, LD occurs more frequently among males.

Race/Ethnicity: Of those cases in which race/ethnicity were known, most were white (n=4, 67%). There was one Hispanic (17%) and one Asian (17%). The remaining were unknown (n=3).

Location: LD does not commonly occur in ticks in LAC, most cases were likely exposed to infected ticks while outside of the county. However, two of eight cases with a known history (25%) reported no travel outside of LAC within three months of their onset of EM rash (Figure 3). These cases occurred among residents from SPAs 2 and 7.

Figure 3
Lyme Disease Cases
by Location of Exposure
LAC, 1994–2007



Disease Severity: Most cases (n=8, 89%) demonstrated EM. Rash sizes ranged from 5-14 cm, with a mean of 8.5 cm and median of 9.5 cm. Three cases (33%) also reported symptoms characteristic of late LD—one with swelling of joints, another with lymphocytic meningitis, and an additional with atrioventricular block.

Risk Factors: Only three cases of eight with a known history (38%) recalled a tick bite within three months of their onset. Six cases (75%) reported travel outside of LAC prior to their onset of symptoms (Figure 3). Of those, one (17%) recalled incurring the tick bite during their travels. All six traveled to parts of the eastern US, where LD is known to be highly endemic. Of the two that remained within LAC, one

hiked often in the Malibu canyon areas and the other camped in the Angeles National Forest; both recalled tick bites. One case could not be interviewed for epidemiological information.

COMMENTS

The number of suspected LD cases in LAC residents reported each year to LAC DPH by clinicians and laboratories has climbed from 20 to 30 in past years to over a hundred in 2007. The vast majority 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, with the exception of 2006, has ranged from none to nine cases a year.

Changes in reporting processes may have increased the number of suspected cases reported to LAC DPH in recent years. 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.

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 controlling ticks on pets.

RESOURCES

Centers for Disease Control and Prevention, general information—
http://www.cdc.gov/ncidod/dvbid/lyme/index.htm

Centers for Disease Control and Prevention, lyme disease statistics—
http://www.cdc.gov/ncidod/dvbid/lyme/ld_statistics.htm

Centers for Disease Control and Prevention (2007). Lyme disease—United States, 2003–2005. *Morbidity and Mortality Weekly Report*, 56(23), 573–576.

Shapiro, E.D. & Gerber, M.A. (2000). Lyme disease. *Clinical Infectious Diseases*, 31(2), 533-542.

Steere, A.C. (2001). Lyme disease. *New England Journal Medicine*, 345(2), 115–125.

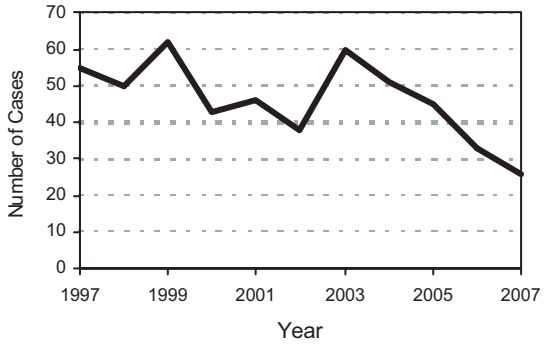
MALARIA

CRUDE DATA	
Number of Cases	26
Annual Incidence	
LA County	0.27
California	0.51[a]
United States	0.50[b]
Age at Onset	
Mean	37
Median	31
Age Range	14–63 years

[a] Calculated based on the number of cases reported in Malaria Surveillance - United States, 2006 issue of MMWR (57(SS05);24-39), and the state population estimate from the 2006 American Community Survey (www.census.gov).

[b] Malaria Surveillance - United States, 2006 issue of MMWR (57(SS05); 24-39),

Figure 1
Malaria
Cases by Year of Onset
LAC, 1997–2007



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. For the purpose of surveillance, confirmation of malaria requires the demonstration of parasites in thick or thin blood smears, regardless of whether the person experienced previous episodes of malaria.

Before the 1950's malaria was endemic in the southeastern US. Now, it is usually acquired outside the continental 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. Malaria surveillance is maintained to detect locally acquired cases that could indicate the reintroduction of transmission and to monitor patterns of resistance to antimalarial drugs. The last occurrence of locally acquired malaria in California (CA) was in 1988–89, when thirty migrant workers were reported in San Diego with *P. vivax* infection. Since then, local transmission has not occurred in southern CA due to the inadequate number of people infected with the malaria parasite required 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 residents who took some form of antimalarial chemoprophylaxis during travel to a malaria-endemic region has dropped to a low of 6%.

STRATIFIED DATA

Trends: In 2007, there were 26 reported cases compared to 33 reported the previous year — a 21% decrease. Over half of the cases (n=14, 54%) were infected with *P. falciparum* in 2007 (Figure 2), less than the proportion affected in 2006 (n=21, 64%).

Age: The median age of infection has decreased markedly from 40 years in 2006 to 31 years in 2007. The mean age was 37 years (range: 14–63 years). The largest number of cases (n=11, 42%) occurred in the 15–34 year age group (Figure 3). In 2006 the largest number occurred in the 45–54 year age group.

Sex: The ratio of male-to-female cases was three to one (2.25:1).

Race/Ethnicity: The majority of reported malaria cases occurred among blacks, which included African-Americans and African immigrants (n=11, 48%). Seven cases (30%) were reported among Asians and four (17%) among Hispanics. Only one case occurred in a white person. Three cases had unknown race and ethnicity. Since the early 1990s, blacks have had the highest proportion of reported malaria cases, with the exception of year 2003, where whites outnumbered blacks.

Disease Severity: There were no deaths from malarial infection in 2007. However, most (n=18, 75%) required hospitalization and several experienced severe complications, mainly with falciparum malaria, including two with renal failure and one with cerebral malaria. Two cases had unknown hospitalization status. The mean length of hospitalization for sixteen cases with known admission and discharge dates was 5.3 days and ranged from 1 to 28 days.

Figure 2
Malaria Cases by Species
LAC, 2007 (n=26)

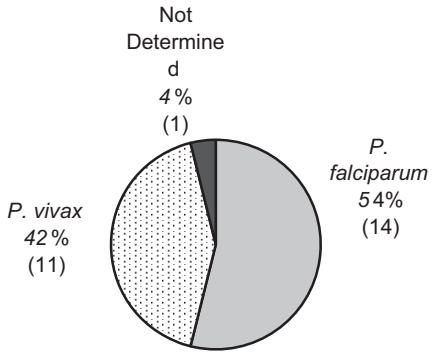
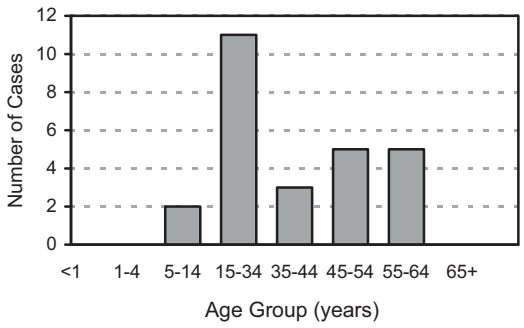


Figure 3
Malaria
Cases by Age Group
LAC, 2007 (n=26)



Transmission and Risk Factors: All twenty-three cases with known travel status reported recent travel to a foreign country. Africa remains the most common region visited (n=11, 48%). Reports of travel to Nigeria, usually the most frequently reported country by far, decreased from 16 in 2006 to 4 in 2007, the same number who travelled to India (Table 1). Among cases with a known reason for travel (n=16), the most commonly reported reason was visiting friends and relatives (n=9, 56%). Refugees and immigrants made up 13% (n=2) of cases with known travel reasons. Purpose of travel was reported for only 62% of cases.

Among the 18 cases with reported US residence and known prophylaxis usage, only one individual (6%) took prophylaxis (Table 2). This is the lowest rate of usage recorded in recent years. Information on antimalarial prophylaxis usage was available for 21 cases (81%), of which a total of three cases (14%) took some form of prophylaxis. None of those who took prophylaxis reported taking their medication correctly as prescribed (one unknown).

Table 1. Malaria Cases by Country of Acquisition and *Plasmodium* Species, 2007

Country of Acquisition	*P. falciparum*	*P. vivax*	Not Determined	Total
Africa	**10**	**0**	**1**	**11**
Congo	1	0	0	1
Ghana	0	0	1*	1
Liberia	1**	0	0	1
Nigeria	4	0	0	4
Sierra Leone	3	0	0	3
Uganda	1	0	0	1
Asia/Oceania	**1**	**6**	**0**	**7**
India	1	3	0	4
Pakistan	0	2	0	2
Papua New Guinea	0	1	0	1
Latin America	**1**	**4**	**0**	**5**
Dominican Republic	1	0	0	1
Guatemala	0	3	0	3
Peru	0	1	0	1
Unknown	**2**	**1**	**0**	**3**
Overall Total	**14**	**11**	**1**	**26**

* Case also traveled to Benin and Togo
** Case also traveled to Ghana

Table 2. Prophylaxis Use Among US Residents with Malaria, 2007

Reason for Travel	Total Cases (n)	Prophylaxis Use (n)	(%)
Pleasure	11	1	6
Work	2	0	0
Other/Unknown	5	0	0
Total	**18**	**1**	**6**

No cases reported a history of prior malaria infection within the past twelve months. No cases were reported as being acquired through blood transfusion or transplantation.

COMMENTS

The number of cases reported in recent years 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). The reasons for the overall decrease in malaria cases are unknown but it can be partially attributed to a decrease of incoming refugees from malaria endemic countries. 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 2007, refugees and immigrants made up only 13%.

Information on travel and prophylaxis is obtained by interviewing patients. The data are 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.

PREVENTION

Prevention methods for malaria include 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.

ADDITIONAL RESOURCES

Centers for Disease Control and Prevention. Available at: http://www.cdc.gov/malaria

Centers for Disease Control and Prevention (1990). Transmission of *Plasmodium vivax* malaria—San Diego County, California, 1988 and 1989. *Morbidity and Mortality Weekly Report*, 39(6), 91-94. Retrieved October 15, 2008, from the CDC Web site: http://www.cdc.gov/mmwr/preview/mmwrhtml/00001559.htm

Centers for Disease Control and Prevention (2006). Malaria surveillance—United States, 2004. *Morbidity and Mortality Weekly Report*, 55(SS04), 23-37. Retrieved October 15, 2008, from the CDC Web site: http://www.cdc.gov/mmwr/preview/mmwrhtml/ss5504a2.htm?s_cid=ss5504a2_e

MEASLES

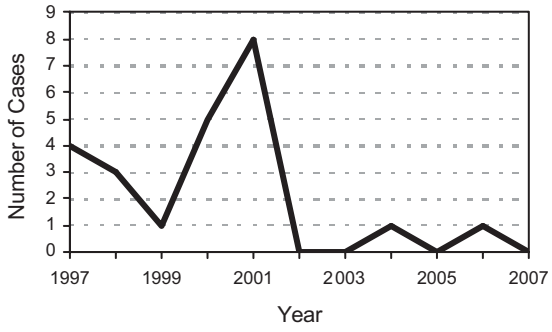
CRUDE DATA	
Number of Cases	0
Annual Incidence[a]	
LA County	0[b]
California	0.01[b,c]
United States	0.01[b]

[a] Cases per 100,000 population

[b] Rates based on less than 19 observations are unreliable.

[c] Calculated from Final 2007 Reports of Nationally Notifiable Infectious Diseases issues of MMWR (57: 901, 903-913).

Figure 1
Measles
Reported Cases by Year of Onset
LAC, 1997-2007



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 81 measles suspect reports received at the LAC Immunization Program, there were no confirmed measles cases identified in LAC during 2007, marking the fourth time this has occurred in over 40 years.
- During 2007, 4 measles cases were reported in California.

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, no serological evidence of measles immunity, or no documentation of physician-diagnosed measles. 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 or MMRV vaccine.
- All foreign travelers who are not immune to measles should be vaccinated, ideally 2 weeks prior to travel.
- Unvaccinated infants 6 months of age and older should be vaccinated if they are traveling outside of the US.

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, 2005, and 2007 no confirmed cases of measles were identified in LAC, marking only four 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 US.

COMMENTS

In the year 2000, the CDC stated that measles was no longer endemic in the US. High vaccination coverage, a highly effective vaccine, and diligent public health surveillance activities have contributed to the limited number of measles cases nationwide. However, even a limited number of cases serve as a reminder that measles can and still does occur in the US. The risk of imported disease remains because the virus continues to circulate in other parts of the world, putting unvaccinated individuals at risk for measles infection. During 2007, large measles outbreaks were reported in Japan, Canada, the United Kingdom, and Switzerland. In May, another state's Department of Health identified a measles case that had traveled from another country to attend an event. The subsequent public health investigation identified 102 California residents that were possibly exposed to the case, 7 of whom were residents of LAC. All 7 LAC residents reported a history of vaccination or previous disease. None developed measles-like symptoms. In another 2007 situation, a different state's Department of Health identified a measles case in a child who had traveled on an international flight from another country and traveled to multiple cities in the US. Six cases of measles were linked to the index case through exposures during travel, in the airport, and during an event. Five of the seven cases had no documented measles vaccination. While no LAC measles cases were identified in association with any of the exposures in Japan, Canada, the United Kingdom, Switzerland, and the two states, 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 93% of children 19-35 months of age in LAC are vaccinated against measles.

Because LAC is in many ways a "gateway" to the US for travelers, it is important that an effective measles surveillance system be maintained in LAC. The public health department depends on healthcare providers and laboratories to identify measles cases and report them in a timely manner. Routinely reminding reporting facilities about the reporting requirements dictated by the California Code of Regulations, Title 17, Section 2500 is an activity that should continue to be implemented. In addition, healthcare providers can play an important role in preventing further transmission by promoting appropriate pre-travel vaccination and by being aware of travel history when evaluating symptomatic patients. The possibility of measles should also be considered in persons with exposure to travelers or exposure to measles in their community (e.g., in healthcare, school, daycare, or household settings). In addition, since measles is highly contagious it is essential that appropriate airborne infection control measures be followed stringently with all suspect measles cases.

CASE INVESTIGATION

The LAC Immunization Program 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 2-28 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:

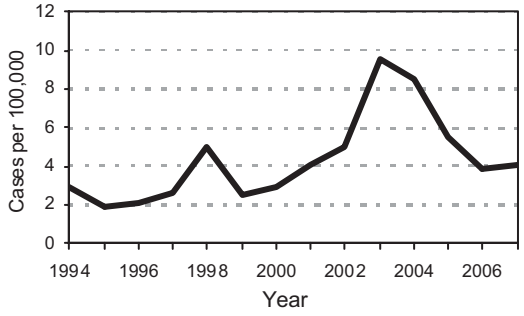
- National Center for Immunization and Respiratory Diseases – http://www.cdc.gov/vaccines
- Immunization Action Coalition – http://www.immunize.org
- LAC Immunization Program – http://www.lapublichealth.org/ip

MENINGITIS, VIRAL

CRUDE DATA	
Number of Cases	395
Annual Incidence[a]	
LA County	4.1
United States	N/A
Age at Onset	
Mean	27
Median	25
Range	0–84 years

[a] Cases per 100,000 population.

Figure 1
Viral Meningitis
Incidence Rates by Year of Onset
LAC, 1994–2007



DESCRIPTION

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

DISEASE ABSTRACT

- The incidence of viral meningitis has continued to be low compared to the peak in 2003 (Figure 1).
- WNV infection contributed to 4% of all reported cases of viral meningitis.
- Heightened surveillance conducted in late 2007 probably contributed to increased identification of viral meningitis cases caused by enterovirus as well as the overall number of cases.

Trends: In 2007, there were a total of 395 reported cases of viral meningitis, representing an annual incidence of 4.1 per 100,000. Though this is a small increase compared to the previous year when 373 cases were reported at an incidence of 3.9 cases per 100,000, this is a marked decrease from previous years when incidence was as high as 9.6 cases per 100,000 (2003) (Figure 1).

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 five-year average in Figure 2 where around a hundred cases are seen each month from July through September. This trend is also seen in 2007, peaking in August with 59 cases (Figure 2).

Figure 2
Viral Meningitis
Cases by Month of Onset
LAC, 2007

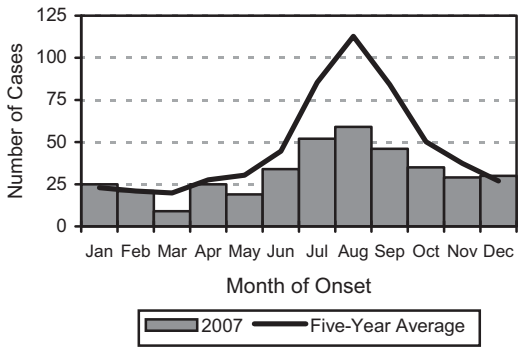


Figure 3
Viral Meningitis
Incidence Rates by Age Group
LAC, 2007 (N=395)

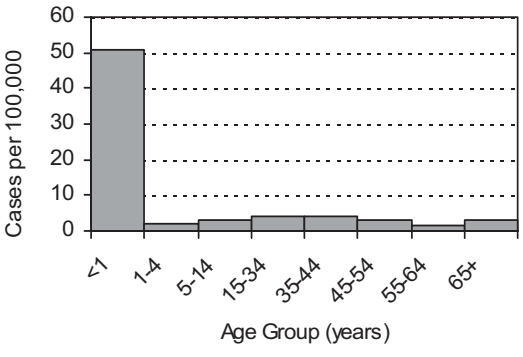
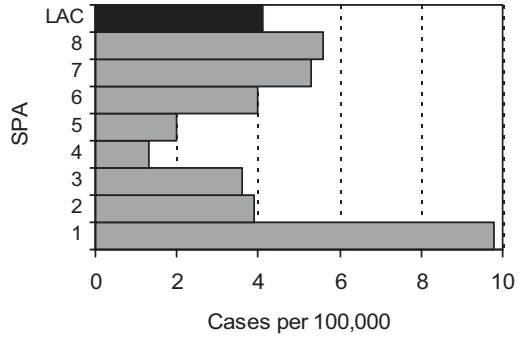


Figure 4
Viral Meningitis
Incidence Rates by SPA
LAC, 2007 (N=395)



Age: Infants less than 1 year old continued to have the highest age-group specific rate at 50.7 cases per 100,000 (Figure 3).

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

Race/Ethnicity: The incidence rates across race and ethnicity groups ranged from 2.3 to 3.9 cases per 100,000, the lowest occurring in Asian/Pacific Islanders. The rates were similar among Hispanics, whites, and blacks (data not shown).

Location: The highest incidence of viral meningitis continued to occur in SPA 1 (9.8 per 100,000).

Clinical Presentation: The case fatality rate remained low; three deaths were reported in 2007 (less than one percent case fatality rate). Of the 70 cases in which an etiology was identified, 49 (70%) were caused by an enterovirus. More cases of WNV meningitis were reported (n=14, 20% among those with known etiologies) than in 2006. They accounted for 4% of all reported cases in 2007 but only 1% in 2006. The viral etiologies of 82% of cases in 2007 remain unknown.

COMMENTS

The highest incidence in LAC in 2007, 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=357,142) of all SPAs in LAC, it continually carries the highest rates of viral meningitis in LAC. Reasons for this trend are unknown.

In late 2007, an increased level of activity of coxsackie B1 virus, a type of enterovirus, was associated with severe neonatal disease and multiple deaths in LAC and other areas of the US. Though none of the deaths was associated with viral meningitis in LAC, this enterovirus can be associated with the syndrome as well as encephalitis, myelitis, and myopericarditis. It has an epidemic pattern of circulation, with increases usually lasting 2 to 3 years. As a result of the increase, LAC requested all hospitals in the county to report all enterovirus-positive cases of severe or fatal myocarditis, aseptic meningitis, or sepsis-like febrile illness that occurred among children during June through November 2007. Surveillance for viral meningitis is generally passive; this change in procedures may explain the slight rise in reported meningitis cases caused by enterovirus, as well as the overall number of viral meningitis cases for 2007. In 2006, only 4% of reported cases (n=15) had an etiology identified. Sixty percent of those cases (n=9) were caused by an enterovirus. This year, 18% (n=70) of reported cases had known etiologies and 70% (n=49) were enteroviruses. Active surveillance is being continued in 2008.

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.

With passive surveillance, 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. 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 antibiotics plus fewer and shorter hospital admissions.

PREVENTION

Good personal hygiene, especially hand washing and avoiding contact with oral secretions of others, is the most practical and effective preventive measure.

ADDITIONAL RESOURCES

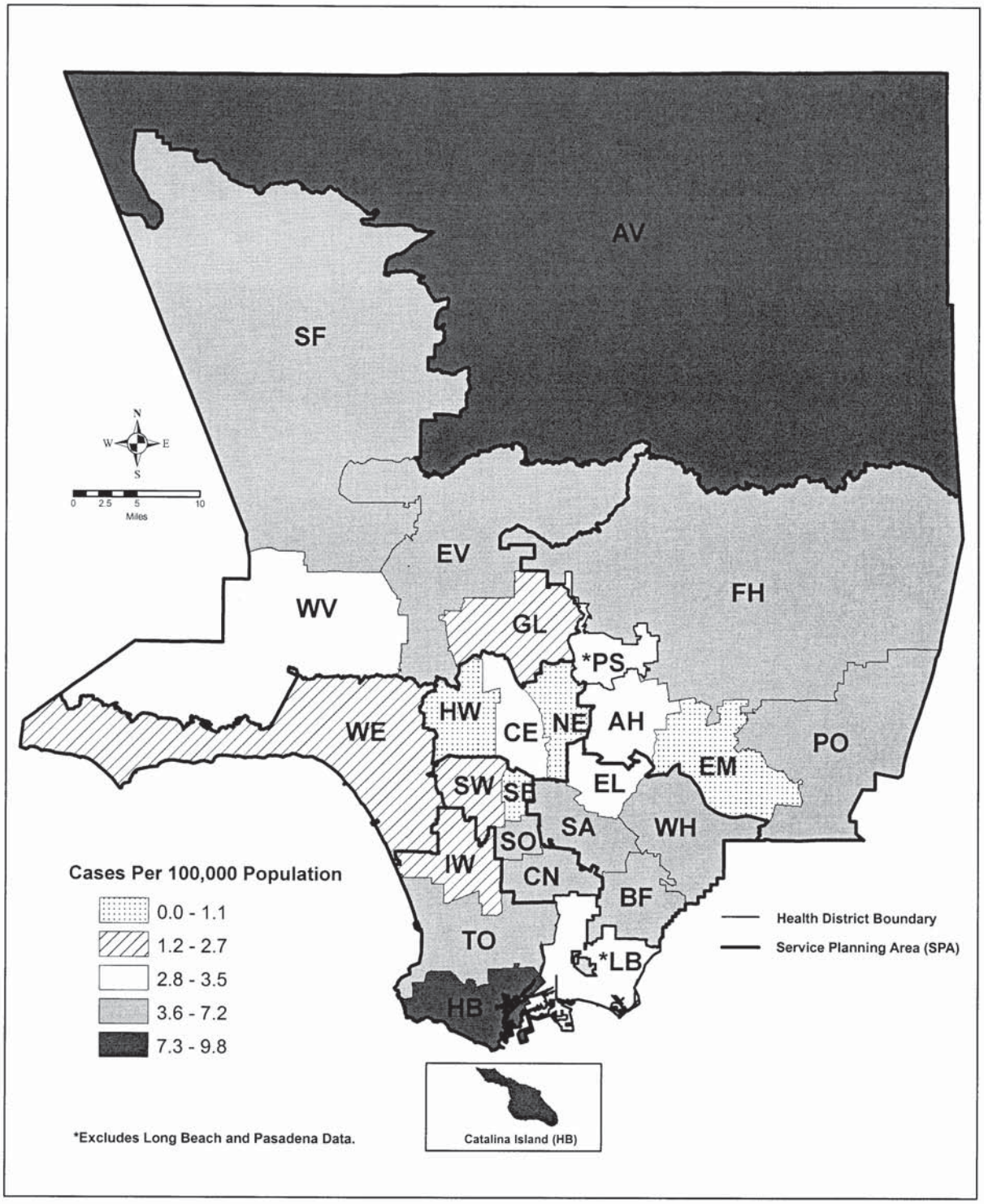
Centers for Disease Control and Prevention (2008). National Center for Immunization and Respiratory Diseases, Division of Bacterial Diseases, Viral (Aseptic) Meningitis at:
http://www.cdc.gov/meningitis/viral/viral-faqs.htm

Centers for Disease Control and Prevention (2008). National Center for Immunization and Respiratory Diseases, Division of Viral Diseases, Non-Polio Enterovirus Infections at:
http://www.cdc.gov/ncidod/dvrd/revb/enterovirus/non-polio_entero.htm

Centers for Disease Control and Prevention (2008). Increased detections and severe neonatal disease associated with coxsackievirus B1 infection—United States, 2007. *Morbidity and Mortality Weekly Report*, 57(20), 553-556. Available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5720a4.htm

Centers for Disease Control and Prevention (2003). Outbreaks of aseptic meningitis associated with echoviruses 9 and 30 and preliminary reports on enterovirus activity—United States, 2003. *Morbidity and Mortality Weekly Report*, 52(32), 761-764. Available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5232a1.htm

Map 10. Meningitis, Viral
Rates by Health District, Los Angeles County, 2007*



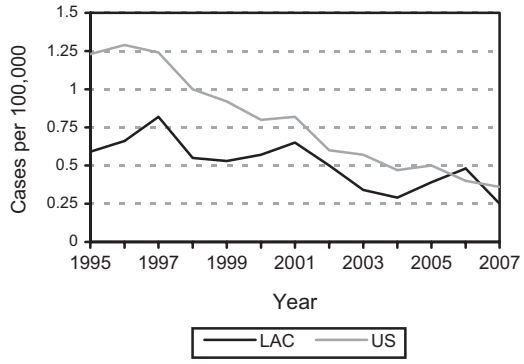
MENINGOCOCCAL DISEASE

CRUDE DATA	
Number of Cases	24
Annual Incidence[a]	
LA County	0.25
California	0.48[b]
United States	0.36[b]
Age at Diagnosis	
Mean	31
Median	28
Range	0–85 years

[a] Cases per 100,000 population.

[b] Calculation based on the MMWR 2007 Final Report of Nationally Notifiable Infectious Diseases and the 2007 estimate of populations at www.census.gov.

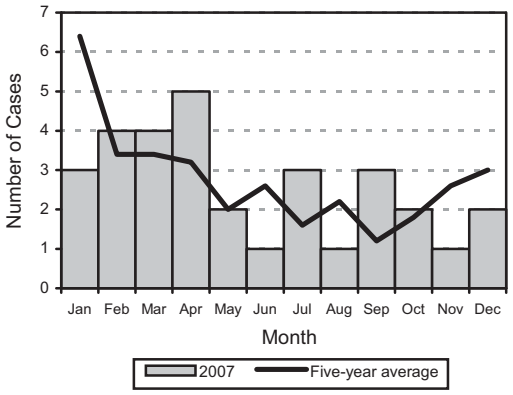
Figure 1
Meningococcal Disease
Incidence Rates by Year of Onset
LAC and US, 1995–2007



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.

Figure 2
Meningococcal Disease
by Month of Onset
LAC, 2007 (N=24)



For the purpose of surveillance, Los Angeles County Department of Public Health (LAC DPH) defines a confirmed case invasive meningococcal disease 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.

DISEASE ABSTRACT

- Confirmed invasive meningococcal disease cases decreased by 50% in 2007 compared to 2006 with 24 and 46 cases reported, respectively.
- Three deaths were documented in 2007 compared to 1 in 2006.
- There were 17 (71%) culture-confirmed cases: 5 (29%) from CSF, 9 (53%) from blood, and 3 from

both CSF and blood (18%). Twenty-one (88%) cases were serogrouped: 5 (24%) were identified as serogroup B, 8 (38%) serogroup C, 6 (29%) serogroup Y, 1 (5%) was W135, and 1 CSF isolate was untypeable.

- No outbreaks were documented in 2007.

STRATIFIED DATA

Trends: The incidence of invasive meningococcal disease decreased by nearly 50% to 0.25 per 100,000 population in 2007 (N=24) from 0.48 per 100,000 in 2006 (N=46) (Figure 1). Seventy-one percent (n=17) of the cases were culture-confirmed in 2007 compared to 83% (n=38) in 2006. The incidence rate has been slowly decreasing in LAC since 2003 and is below the national rate of 0.33 per 100,000 estimated for 2007. Despite the decrease in cases, more deaths were documented in 2007: three deaths (13%) compared to one in 2006 (2%).

Seasonality: Most cases were reported during winter and early spring (Figure 2). There were no cases reported in October and November.

Age: The age-specific incidence rates declined in all age groups with the exception of the 35-44 year old group. Infants <1 year decreased in 2007 (2.0 versus 2.8 per 100,000) compared to 2006. The rates among 15-34 years were also lower (0.2 versus 0.3 per 100,000). The rate among adults > 65 also decreased in 2007 (0.8 versus 0.2 per 100,000).

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

Race/Ethnicity: Invasive meningococcal cases were reported most frequently in Hispanics (n=11, 46%) followed by whites (n=9, 38%), blacks (n=3, 13%), and Asians (n=1, <1%). The incidence rates by race/ethnicity are unstable.

Location: Cases were reported from all eight Service Planning Areas (SPA). No significance noted.

COMMENTS

As a part of public health meningococcal disease surveillance, clinical laboratories are requested to send isolates of every culture-confirmed case to the LAC Public Health Laboratory (PHL) for serotyping**.** In 2007, 21 isolates were serogrouped: 16 (76%) were culture-confirmed and 4 (19%) isolates were serogrouped using whole blood or CSF PCR. The remaining cases (n=3, 14%) had positive CSF antigen tests or gram stains. Most isolates were serogroup C, 8 (38%), followed by serogroup Y (n=6, 29%), serogroup B (n=5, 24%), and 1 (5%) isolate was W135. A larger proportion of isolates were serogroup

Figure 3
Meningococcal Disease
Incidence Rates by Age Group
LAC, 2007

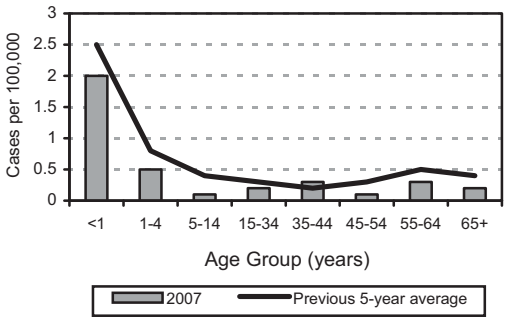


Figure 4
Meningococcal Disease
Incidence Rates by Race/Ethnicity
LAC, 2007

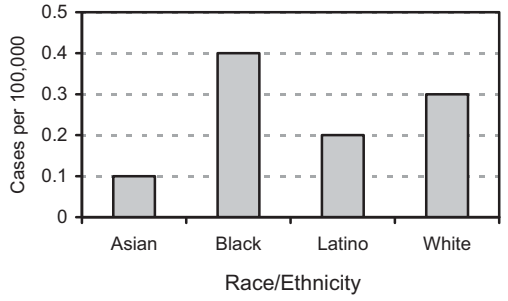
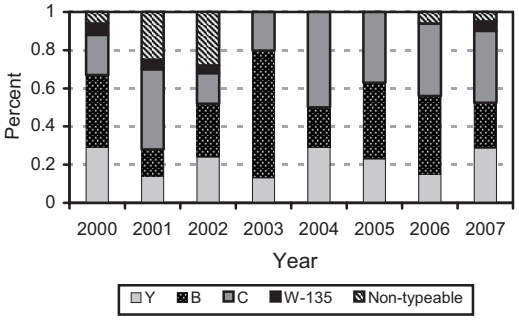


Figure 5
Meningococcal Disease
by Serogroup
LAC, 2000–2007



C compared to previous surveillance years (Figure 5). The mean and median ages of the vaccine preventable cases (n=15) were 33.4 and 21 years, respectively, and ranged from 0–85 years. Non-vaccine preventable serogroup B cases (n=5) had a mean age of 16, median age of 18, and range of 0–39. 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.

LAC DPH and the California Department of Public Health have continued to conduct 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.

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) daycare center contacts, and c) anyone directly exposed to the patient's oral secretions (e.g., through kissing, mouth-to-mouth resuscitation, endotracheal intubation, or endotracheal tube management). Because the rate of secondary disease for close contacts is highest during the first few days after onset of disease in the primary patient, antimicrobial chemoprophylaxis should be administered as soon as possible (ideally within 24 hours after the case is identified). Conversely, chemoprophylaxis administered 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 LAC DPH, Community Health Services.

In 2004, a new quadrivalent meningococcal conjugate (MCV4), Menactra®, was approved for use in the U.S. 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.

ADDITIONAL RESOURCES

Centers for Disease Control and Prevention (2007). Active Bacterial Core Surveillance Report, Emerging Infections Program Network, *Neisseria meningitidis*, 2007-*Provisional*. Retrieved December 15, 2008, from the CDC Web site: http://www.cdc.gov/ncidod/dbmd/abcs/survreports/MEN_2007_provisional.pdf

Centers for Disease Control and Prevention (2005). Prevention and control of meningococcal disease. Recommendations of the Advisory Committee on Immunization Practices. *Morbidity and Mortality Weekly Report*, 54(RR07), 1-21. Retrieved October 29, 2008, from the CDC Web site: http://www.cdc.gov/mmwr/PDF/rr/rr5407.pdf

Centers for Disease Control and Prevention (2007). Recommended immunization schedules for persons aged 0-18 years—United States, 2007. *Morbidity and Mortality Weekly Report*, 55(51), Q1-4. Retrieved October 29, 2008, from the CDC Web site: http://www.cdc.gov/mmwr/PDF/wk/mm5551-Immunization.pdf

Raghunathan, P.L., Bernhardt, S.A., & Rosenstein, N.E. (2004). Opportunities for control of meningococcal disease in the United States. *Annual Review of Medicine*, 55, 333-353.

MUMPS

CRUDE DATA	
Number of Cases	5
Annual Incidence[a]	
LA County	0.05[b]
California	0.12[c]
United States	0.27[c]
Age at Diagnosis	
Mean	35.2 years
Median	44.0 years
Range	6.0–53.0 years

[a] Cases per 100,000 population.
[b] Rates based on less than 19 observations are unreliable.
[c] Calculated from Final 2007 Reports of Nationally Notifiable Infectious Diseases issues of MMWR (57: 901, 903-913).

Figure 1
Mumps
Incidence Rates by Year of Onset
LAC and US, 1997-2007

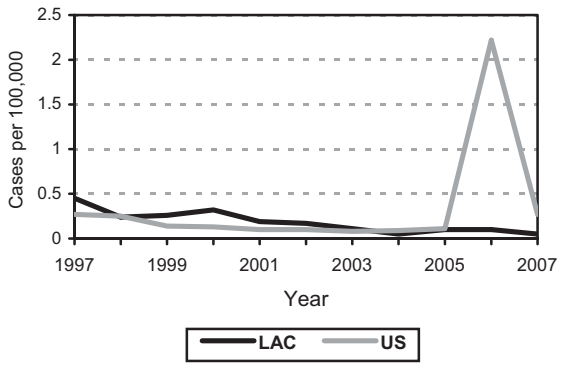
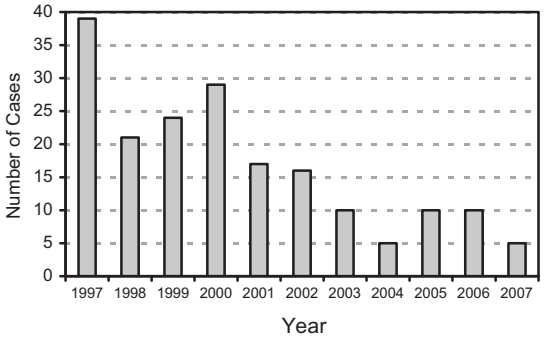


Figure 2
Mumps
Cases by Year of Onset
LAC, 1997-2007



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

- Compared to 2006, there was a 41.7% decrease in the number of suspect mumps reports.
- Of 60 suspect mumps reports received at the LAC Immunization Program during 2007, only five were identified as confirmed mumps cases.

Table 1. Mumps Cases by Case Classification, 2007 vs. 2006

	Confirmed		Probable	
	2007	2006	2007	2006
Number of Cases	5	10	13	63
Age at Onset				
Mean	35.2	31.5	19.9	16.0
Median	44.0	32.0	10.5	9.0
Range	6.0 – 53.0	3.0 – 56.0	3.0 – 64.0	1.0 – 55.0

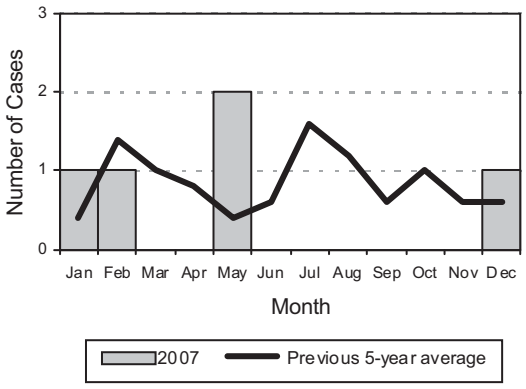
IMMUNIZATION RECOMMENDATIONS

- Mumps disease can be 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 mumps-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, no serological evidence of mumps immunity, or no documentation of physician-diagnosed mumps. 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.
- Approximately 90% of those who receive two doses of the current live attenuated mumps vaccine develop immunity.
- Women should not become pregnant within 4 weeks of vaccination.
- Individuals who are severely immunocompromised for any reason should not be given MMR or MMRV vaccine.
- All foreign travelers who are not immune to measles should be vaccinated, ideally 2 weeks prior to travel.
- Unvaccinated infants 6 months of age and older should be vaccinated if they are traveling out of the country.

STRATIFIED DATA

Trends: Since 1997, the annual number of LAC mumps cases has decreased by 87% (Figure 2). This decline reflects the effectiveness of the MMR vaccine in reducing the incidence of disease in the general population. Although the greater media attention and general public awareness related to the 2006 multi-state mumps outbreak resulted in a large number of suspect case reports (n=103) in 2006, only 10% (n=10) were confirmed cases and 61% (n=63) were probable cases. In 2007, there was a decrease in the number of suspect case reports (n=60). Among the 60 suspect cases, 8% (n=5) were identified as confirmed and 30% (n=18) as probable cases. However, since 2006 it should be noted that vaccination history and negative laboratory results have been considered irrelevant 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 in 2006-2007 would have been

Figure 3
Mumps
Cases by Month of Onset
LAC, 2007



classified as false prior to 2006 because they had documentation of 2 doses of MMR vaccine and/or negative laboratory results.

Seasonality: Historically, mumps incidence peaks during the winter and summer seasons. However, suspect mumps cases are reported throughout the year (Figure 3).

Age: Similar to previous years, 80% (n=4) of all confirmed cases in 2007 were in persons over the age of 15 (Figure 4). Children and young adults are more likely to have been fully immunized. Table 1 indicates that probable cases in the last couple years were on average younger than the confirmed cases.

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

Race/Ethnicity: Three of the confirmed cases were Asian and two were Hispanic.

Location: Confirmed cases were reported in SPA 1, 2, 3, 6, and 7 (Figure 5). None of the cases was epidemiologically linked to another 2007 case, but the case in SPA 2 was epidemiologically linked to a 2006 case with onset in December.

Figure 4
Mumps
Cases by Age Group
LAC, 2007

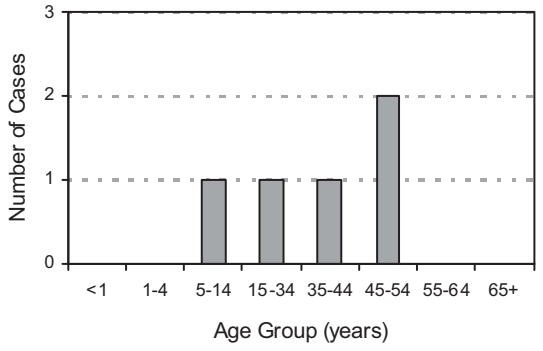
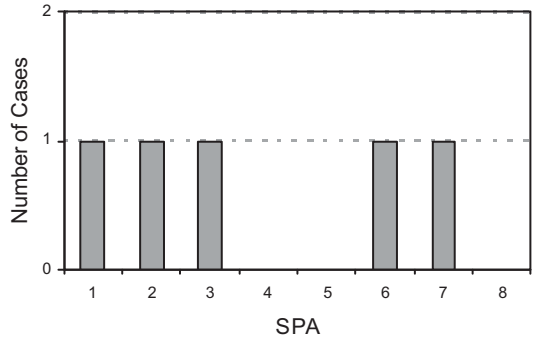


Figure 5
Mumps
Cases by Service Planning Area
LAC, 2007



COMMENTS

The 2006 multi-state mumps outbreak, which resulted in more than 6,000 reported mumps cases, had a profound impact on mumps surveillance nationwide. Vaccine efficacy was reevaluated, the case definition was slightly revised, and laboratory test guidelines were changed. Changes in case classifications also resulted in a large number of suspect cases that would have been classified as false prior to 2006 being classified as probable after 2006. Greater media attention and general public awareness also significantly increased the number of mumps reports.

During 2007, mumps outbreaks were also reported internationally. The state of Maine reported at least seven confirmed mumps cases, prompting some universities to exclude from classes students who were not up to date with their mumps vaccinations. The Maine outbreak was believed to be linked to outbreaks in the Canadian provinces of New Brunswick, Nova Scotia, Prince Edward Island, and Alberta. Internationally, 232 mumps cases were identified in an Ethiopian refugee camp between August 1 and November 9, 2007. The United States was in the process of resettling approximately 1,000 refugees from this camp and notified state health departments of potential imported mumps cases. However, LAC did not receive any notifications of imported mumps cases.

While there were no outbreaks (i.e., 3 epidemiologically linked cases) reported in LAC, there was one situation that required close monitoring. Two LAC cases (a father with onset in January 2007 and his son with onset in December 2006) were discovered to be epidemiologically linked to a laboratory-confirmed case in another state (with onset in January 2007). The father and son had traveled internationally in December 2006. Multiple family, friend, and work contacts were identified. The continued identification of cases in LAC and in other parts of the world indicates that more work needs to be done to increase

vaccination coverage and prevent further transmission. It should be noted that not all cases of parotitis are due to mumps. Sporadic cases among highly immunized populations are most likely caused by other agents such as 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. Determination of epidemiological linkages, MMR vaccination status, and appropriate laboratory testing (mumps IgM antibody assay and viral culture) will help ensure that only true mumps cases are reported.

Cluster Identification: None of the confirmed cases in 2007 were epidemiologically linked to each other. As described above, one case was linked to a 2006 LAC case and a 2007 laboratory-confirmed case in another state. The index case from this cluster of three cases was exposed in another country. An additional 2007 case (not related to the cluster) was also exposed in another country and was linked to a 2008 LAC case.

Vaccination Status: Only one of the confirmed cases was fully immunized with two doses of MMR vaccine. The remaining four cases did not know or remember their vaccination status.

Laboratory Confirmation: Eighty percent (n=4) of the confirmed cases had supporting laboratory confirmation. One case was epidemiologically linked to a 2007 laboratory-confirmed case in another state.

ADDITIONAL RESOURCES

Additional information is available at:

- National Center for Immunization and Respiratory Diseases – http://www.cdc.gov/vaccines
- Immunization Action Coalition – http://www.immunize.org
- LAC Immunization Program – http://www.lapublichealth.org/ip

PERTUSSIS (WHOOPING COUGH)

CRUDE DATA	
Number of Cases	69
Annual Incidence[a]	
LA County	0.71
California	1.62[b]
United States	3.49[b]
Age at Diagnosis	
Mean	10.9 years
Median	4.0 years
Range	10 days – 59 years

[a] Cases per 100,000 population.

[b] Calculated from Final 2007 Reports of Nationally Notifiable Infectious Diseases issues of MMWR (57: 901, 903-913).

Figure 1
Pertussis Incidence Rates
by Year of Onset
LAC and US, 1997-2007

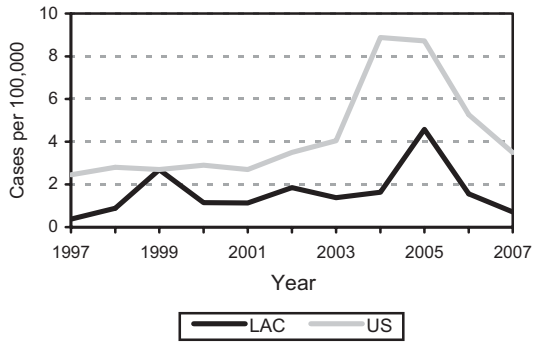
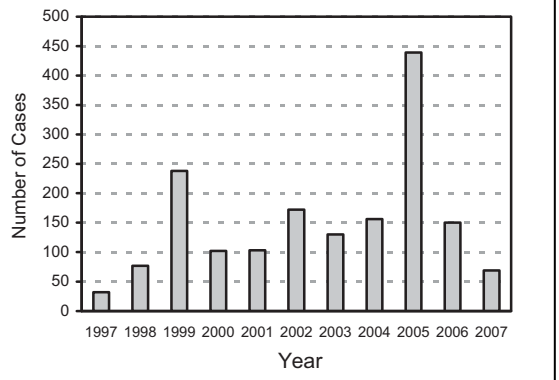


Figure 2
Pertussis Cases by Year of Onset
LAC, 1997-2007



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 *Bordetella pertussis* culture or PCR.

DISEASE ABSTRACT

- Only 69 cases were reported in 2007 (0.71 cases per 100,000), which is the lowest number of reported cases and incidence rate since 1997.
- One pertussis-related death occurred in 2007, marking the twelfth death within the last 10 years.
- Of the 2007 cases, 82% were not adequately immunized that could have been fully protected against pertussis (7 months to 64 years old).

IMMUNIZATION RECOMMENDATIONS

- A pertussis-containing vaccine should be administered at 2, 4, 6, 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: Typically, the summer months have the highest pertussis incidence in LAC (Figure 3). However, in 2007, there were peaks in the winter months of March and December. March accounted for 15% (n=10) of cases; six of the cases were a part of two separate household clusters. December accounted for 20% (n=14) of cases; three of the cases had epidemiological linkages to other cases with December onset dates. The onset of cases was distributed fairly uniformly throughout the rest of the year.

Age: Although the majority of reported cases are still in children <1 year of age, the proportion of cases in the <1 age group is slightly smaller in 2007 (45%) compared to the previous five year average (51%). As seen nationally, cases are slightly increasing among adolescents and adults, as evidenced by the fact that 30% (n=21) of the cases were over 14 years of age in 2007 compared to an average of 26% (n=54) in the previous five years (Figure 4). Increased recognition and diagnosis of pertussis in older age groups has contributed to the increase in reported cases among adolescents and adults.

Sex: The male-to-female case ratio was approximately 1:1.4.

Race/Ethnicity: After adjusting for the age differential in the cases, incidence rates in 2007 for blacks, Hispanics, 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, for which whites had the highest incidence rate at 6.1 cases per 100,000. The incidence rates for Hispanics and whites were approximately equivalent to the total LAC rate. However, the LAC population proportion of whites (30%) is much lower than that for Hispanics (48%).

Location: For the first time in over five years, West SPA 5 had the highest incidence rate of 1.2 cases per 100,000 (n=8); two of the cases in SPA 5 were epidemiologically linked. The second highest incidence rate occurred in South Bay SPA 8 with 0.9 cases per 100,000 (n=10); 60% (n=6) of the 10 cases were epidemiologically linked to cases living within two unrelated households.

Figure 3
Pertussis Cases by Month of Onset
LAC, 2007

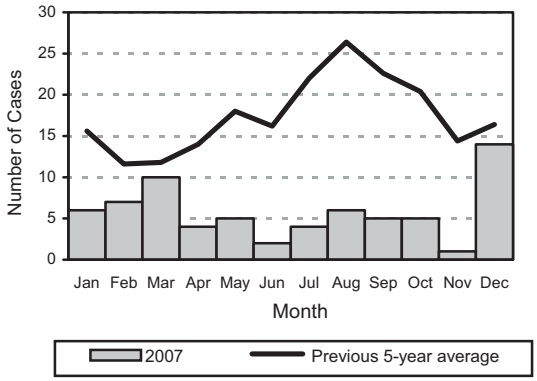


Figure 4
Pertussis Cases by Age Group
LAC, 2007

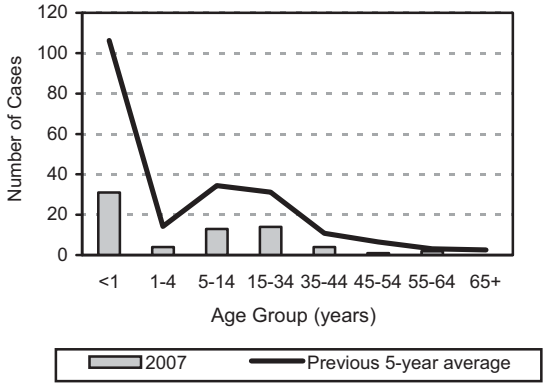
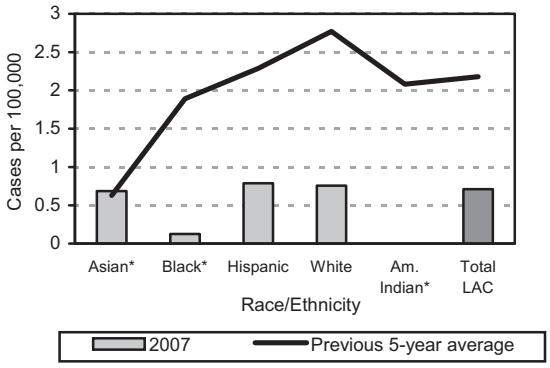


Figure 5
Pertussis Age-Adjusted Incidence Rates
by Race/Ethnicity, LAC, 2007



*Rates unreliable due to small number of observations.

COMMENTS

During 2007, pertussis received some media attention due to school outbreaks in the Virgin Islands and South Carolina. No LAC were identified in association with either of these outbreaks nor were there any outbreaks in LAC.

Historically, pertussis incidence peaks every 3 to 5 years. The last peak in incidence occurred in 2005, which was the same year the two Tdap vaccines for adolescents and adults were licensed. Following the cyclical nature of pertussis incidence, a high incidence would not be expected in 2007. However, compared to the last 10 years, an incidence of 0.71 cases per 100,000 in 2007 is unusually low. The decrease in pertussis activity is not likely to be due to increased use of pertussis vaccine. According to the most recent National Immunization Survey (NIS) data, vaccine coverage levels for 4+ doses of DTaP among children 19-35 months of age in LAC has consistently been above 80%; 85% in 2006 compared with an average of 83.9% during the previous 5 years (2001-2005). An NIS telephone survey conducted from May to August 2007 found that Tdap vaccine coverage levels among adults aged 18 to 64 years of age is only 2.1%. Additional surveillance and epidemiological studies will be needed to monitor the impact of Tdap vaccination on pertussis incidence following its 2005 licensure. However, it is clear that more work needs to be done to increase Tdap vaccination rates.

Trends: The epidemiology of pertussis in LAC is shifting to different age groups, racial/ethnic groups, and geographic areas (LAC IP, 2007). During the winter quarters preceding the most recent peak incidence years (1999, 2002, and 2005), more LAC cases were reported among adolescents 10 to 19 years of age. whites are contributing more of the LAC adolescent/adult cases. The geographic face of pertussis is also shifting outside of the historical high morbidity areas for vaccine-preventable diseases in central Los Angeles to surrounding areas with higher proportions of whites (West Los Angeles, South Bay, and Antelope Valley).

Laboratory Confirmation: More than half of the reported cases (59%, n=41) were laboratory confirmed by either *B. pertussis* culture or PCR. Culture is considered the gold standard laboratory test because it is the most specific of all the laboratory tests for pertussis. While the PCR test's rapidity and sensitivity can greatly aid in the diagnosis of pertussis, specificity can be poor with high rates of false-positive results.

Vaccination Status: Of the 38 cases who could have had full immunity from vaccination (7 months to 64 years old), only 18% (n=7) were fully up to date. Of these 38 cases, 68% (n=26) were 10 years of age or older. Although the 26 cases would have been eligible for Tdap vaccine, none had received Tdap.

Less than one fifth of all cases (19%, n=13) were younger than two months of age and were too young to receive pertussis vaccine. Approximately 26% (n=18) of cases were between 2–6 months of age. Of these, 56% (n=10) were up to date with pertussis vaccination for their age, but would not have developed full immunity against pertussis. Of the 12 children who could have had full immunity from childhood DTaP vaccination (7 months to 9 years old), 58% (n=7) 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.

Complications/Hospitalizations: Approximately 39% (n=27) were hospitalized, with an average hospital stay of 7 days (range 1-16 days). Among the hospitalized cases, 93% (n=25) were less than one year of age. Of the 6 cases who developed pneumonia, 83% (n=5) were infants less than one year of age.

Case Fatalities: There was one pertussis-related death in 2007. The fatality occurred in a Hispanic female infant who was less than 1 month of age. The principal diagnosis in the discharge/death summary was cardiorespiratory arrest. The female infant died 18 days after cough onset. The infant was in contact with 3 family members who were also coughing around the same time. During the first 13 days of her illness, the patient sought hospital care and was discharged both times without a pertussis diagnosis. During the third hospital visit 4 days later, she was admitted into the pediatric intensive care unit with a diagnosis of hypoxemia, respiratory failure, lymphocytosis, and suspect pertussis. A PCR test detected *Bordetella pertussis* DNA and azithromycin treatment was initiated. The patient expired the next day. Earlier consideration of pertussis may have prevented death.

REFERENCES

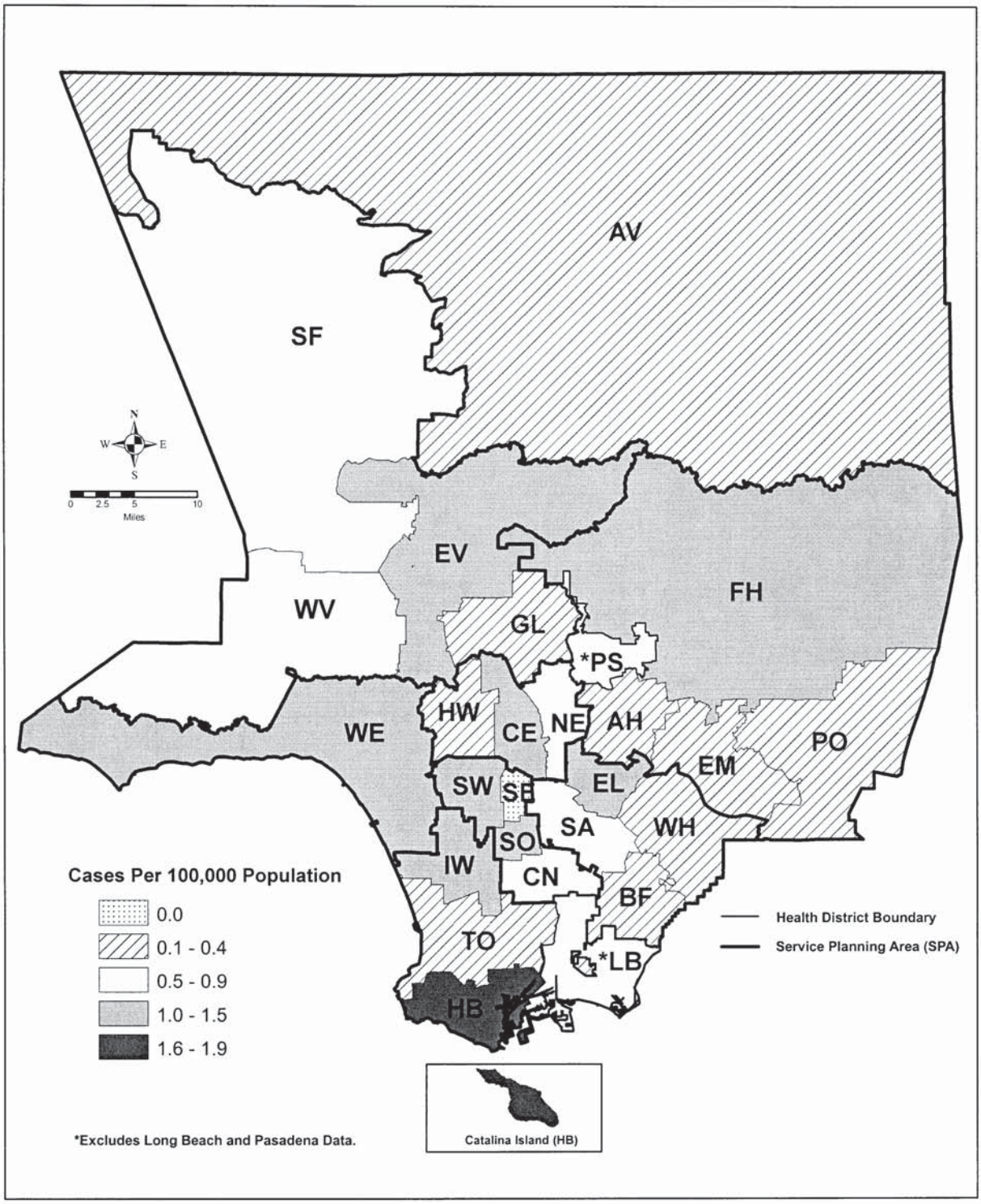
Los Angeles County Immunization Program (2007). The endemic and cyclical nature of pertussis disease morbidity: The evolving epidemiology of a unique vaccine-preventable disease in Los Angeles County. Los Angeles County Department of Public Health.

ADDITIONAL RESOURCES

Additional information is available at:

- National Center for Immunization and Respiratory Diseases – http://www.cdc.gov/vaccines
- Immunization Action Coalition – http://www.immunize.org
- LAC Immunization Program – http://www.lapublichealth.org/ip

Map 11. Pertussis
Rates by Health District, Los Angeles County, 2007*



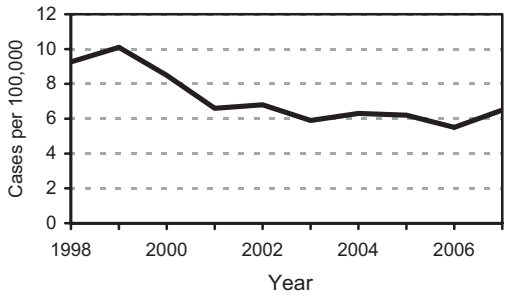
PNEUMOCOCCAL DISEASE, INVASIVE

CRUDE DATA	
Number of Cases	625
Annual Incidence[a]	
LA County	6.5
United States	14.0[b]
Age at Diagnosis	
Mean	51
Median	56
Range	0 days -100 years

[a] Cases per 100,000 population.

[b] National projection of IPD incidence from Active Bacterial Core Surveillance areas data, 2007 (CDC, 2007).

Figure 1
Invasive Pneumococcal Disease
Incidence Rates by Year of Culture
LAC, 1998–2007



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 identified 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 the most common bacterial cause 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 increased slightly in LAC in 2007.
- The overall percentage of penicillin nonsusceptible infections has increased slightly. The percentage of penicillin nonsusceptible isolates increased or remained the same for all age groups except for cases aged 5-14 years and 45-54 years.
- The highest incidence of IPD continued to be among blacks.

STRATIFIED DATA

Trends: IPD occurred at an incidence rate of 6.5 per 100,000 in 2007 (N=625), an increase from the previous year (5.5 per 100,000, N=533) (Figure 1).

Seasonality: The seasonal trend in 2007 followed the typical peak for IPD in winter months, dropping in the spring and summer months (Figure 2).

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

Age: The age of IPD cases ranged from birth to 100 years old with a mean of 51 years and median of 56 years. The incidence rate increased or stayed the same from 2006 in all age groups. The incidence rate has increased consistently since 2005 in persons aged 55-64 years. As expected, the highest rate (21 cases per 100,000) occurred in cases aged 65 years and older (Figure 3).

Race/Ethnicity: The incidence decreased among whites and blacks and increased among Latinos and Asians. The highest incidence of IPD occurred among blacks (8.2 cases per 100,000). This rate was more than twice as high as that of whites and approximately three times as high as that of Latinos and Asians (Figure 4).

Disease Severity: Hospitalization status was known for 71% of cases. Of these cases, 94% were hospitalized. Hospitalization was more frequent in cases 65 years and older (99%) and occurred less in children under 5 years (80%). The overall case fatality was 14%, similar to 2006 and higher than the national case fatality of 10.3% (CDC, 2007). Adults aged 35-44 years had the highest case fatality (34%) of all age groups followed by cases aged 55-64 years (18%) and cases older than 65 years (14%).

Antibiotic Susceptibility: Antibiotic resistance information was provided for 93% of cases. The percentage of isolates nonsusceptible to penicillin has increased slightly compared to the previous 5 years. The same is true of isolates nonsusceptible to erythromycin and cefotaxime. The percentage of isolates nonsusceptible to trimethoprim-sulfamethoxazole (TMP-SMZ) decreased to 20% in 2007.

The percentage of cases with penicillin nonsusceptible S. pneumoniae (PNSP) isolates

Figure 2
IPD Cases By Month of Culture
LAC, 2007

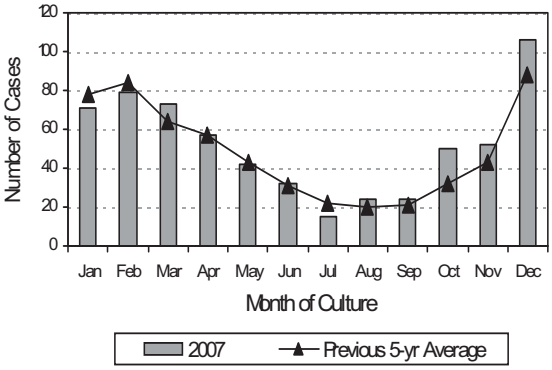


Figure 3
Incidence Rates of IPD Cases by Age
LAC, 2005-2007

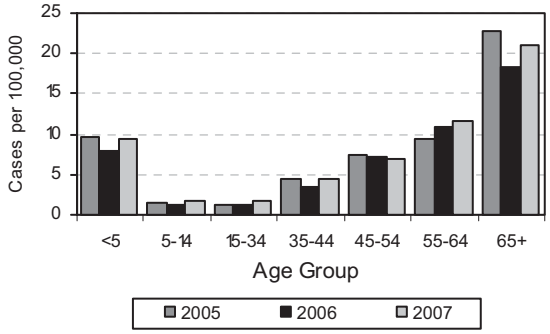
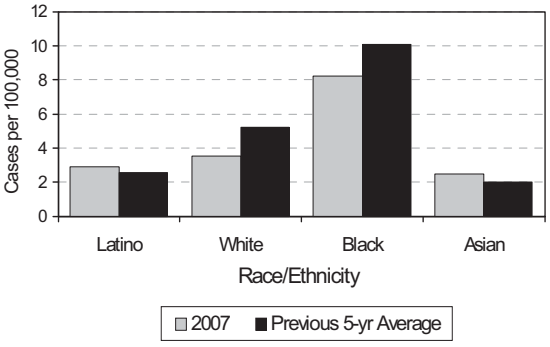


Figure 4
Incidence Rates of IPD Cases by Race/Ethnicity
LAC, 2007



has increased for cases aged < 5 years, 15-34 years, and 35-44 years, as well as cases over 65 years of age. Cases aged 5-14 years and 45-54 experienced a decrease in PNSP (Figure 6). The percentage of PNSP isolates has remained about the same for adults 55-64 years old.

COMMENTS

In 2007 IPD increased in all age groups except for adults aged 45-54 years. Surprisingly, adults aged 35-44 years had the highest case fatality (34%) of all age groups. Cases <5 years showed the highest percentage of PNSP isolates. The percentage of PNSP isolates increased or remained the same for all age groups with the exception of cases aged 5-14 years and 45-54 years which experienced a considerable decrease in the percentage of PNSP.

In LAC, incidence of IPD in blacks (8.2 cases per 100,000) is over two times that of whites and about 3 times that of Latinos and Asians. The black-to-white rate ratio is similar to the ratio found nationally; however, the incidence rates in Los Angeles County for both whites and blacks are lower than the national incidence rates (national rates: 12 and 24 cases per 100,000 respectively) (CDC, 2007).

Laboratories are the source for many of the IPD case reports to ACDC: 58% of cases were reported by laboratories only. Many of the limitations in the data are due to the limited access laboratories have to patient information. Race/ethnicity data and outcome status, in particular, are often missing from laboratory reported cases. Only 54% of reports contained race/ethnicity data and 37% 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.

Figure 5
IPD Isolates Nonsusceptible to Selected Antimicrobial Agents
LAC, 2007

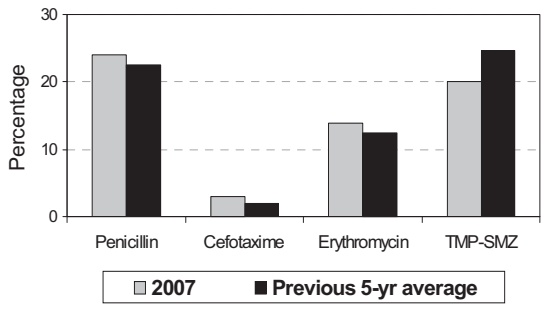
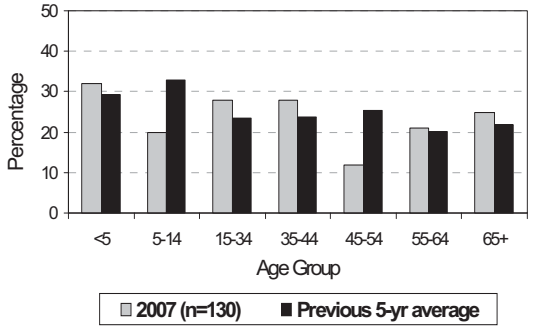


Figure 6
PNSP Isolates by Age
LAC, 2007



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 under 2 years, and for children up to 5 years 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 >2 years at high risk of IPD. For children aged 2 to 5 years at high risk of invasive pneumococcal infections, ACIP recommends the 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 (CDC, 1997).

REFERENCES

Centers for Disease Control and Prevention (1997). Prevention of pneumococcal disease: recommendations of the Advisory Committee on Immunization Practices. *Morbidity and Mortality Weekly Report*, 46(RR08), 1–24.

Centers for Disease Control and Prevention (2007). Active Bacterial Core Surveillance Reports, Emerging Infections Program Network, *Streptococcus Pneumoniae 2007*. Available at: http://www.cdc.gov/ncidod/dbmd/abcs/survreports/SPNEUMO_2007_provisional.pdf

ADDITIONAL RESOURCE

Flannery, B., Schrag, S., Bennett, N.M., Lynfield, R., Harrison, L.H., Reingold, A., et al. (2004). Impact of childhood vaccination on racial disparities in invasive *Streptococcus pneumoniae* infections. *Journal of the American Medical Association*, 291(18), 2197-2203.

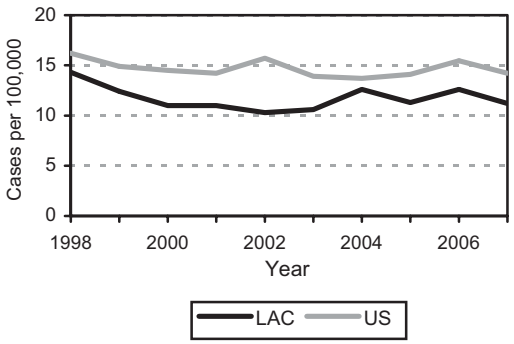
SALMONELLOSIS

CRUDE DATA	
Number of Cases	1081
Annual Incidence[a]	
LA County	11.2
California	11.0[b]
United States	14.2[b]
Age at Diagnosis	
Mean	27.9
Median	22
Range	<1-101

[a] Cases per 100,000 population.

[b] Calculated from Final 2007 Reports of Nationally Notifiable Infectious diseases issue of MMWR (57:901, 903-913).

Figure 1
Salmonellosis
Incidence Rates by Year
LAC and US, 1998–2007



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 2007 salmonellosis crude rate decreased 11% when compared to 2006 (Figure1). This rate is comparable to the state rate and remains below the national rate.
- *Salmonella* serotype e*nteritidis* was again the most common serotype in 2007. However, the percent of change was -10% due to a continued decrease in the total number of isolates (Table 1).
- Five outbreaks were investigated in 2007, compared to nine in 2006.
- SPA 6 had the highest rate (12.6 per 100,000) of salmonellosis during 2007.

STRATIFIED DATA

Trends: The rate of salmonellosis cases for LAC in 2007 was 11.2 cases per 100,000 population, an 11% decrease from the 2006 rate of 12.6 but similar to the 2005 rate of 11.3 (Figure 1). This rate remains below the national rate. Reasons for this decrease are unknown. 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 2007 rate decreases to 10.6 per 100,000 population.

Salmonella Serotypes: For the fourth year, *S. enteritidis* was the number one serotype, however, the incidence has continued to decrease to 24.2% of total isolates serotyped.

Table 1. Most Frequent *Salmonella* Serotypes—LAC, 2006–2007

Serotype	2006 (N=1217)		2007 (N=1011)*		
	No.	Percent	No.	Percent	%Change
Enteritidis	328	26.9	245	24.2	-10.0
*Typhimurium***	173	14.2	146	14.4	+1.4
Newport	76	6.2	76	7.5	+21.0
Heidelberg	49	4.0	58	5.7	+42.5
Agona	21	1.7	46	4.5	+164.7
Montevideo	47	3.9	28	2.8	-28.2
Oranienburg	27	2.2	25	2.4	+9.0
I 4,5,12:i:--	48	3.9	23	2.3	-41.0
Blockley	1	0.08	22	2.2	+2650.0
Braenderup	23	1.9	19	1.9	0

* Includes only serotyped isolates. (three cases for 2007 had two different serotypes of *Salmonella*)
** Includes *S. typhimurium* var. 05 negative (formally var. *copenhagen*)

No commonalities were identified among 2007 *S. blockley* cases.

Seasonality: In 2007, incidence peaked in August (Figure 2) and was greater than the five-year average. Incidence was also greater than the five-year average for the months of April, July, and November. There were outbreaks recorded for the months of March, June, August and November (Table 2).

Age: As shown in Figure 3, the highest age group rates of infection occurred among infants aged less than one year (66.9 per 100,000 population) followed by children aged 1-4 years (31.7 per 100,000 population). This is typical for salmonellosis. In 2007, the rate for infants aged less than one year was slightly higher than the five-year average.

Hospitalization: In 2007, 19.7% of cases were hospitalized for more than 24 hours, compared to 19% in 2006. Ages ranged from less than 1 year to 101 years. The average age of the hospitalized patient was 38 years and the median age was 37 years.

Sex: The male-to-female rate ratio was 1:1.2.

Figure 2
Salmonellosis
Cases by Month of Onset
LAC, 2007

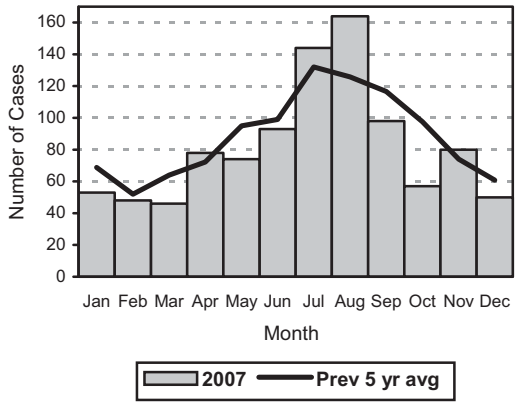
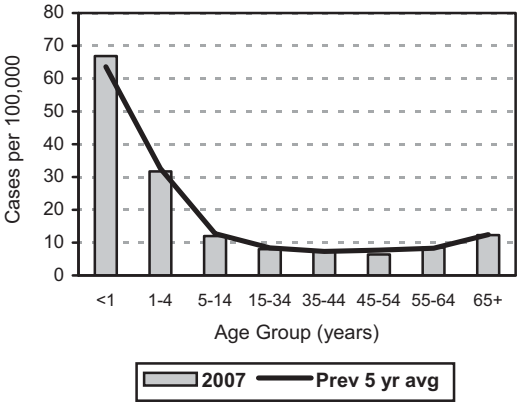


Figure 3
Salmonellosis
Incidence Rates by Age Group
LAC, 2007



Race/Ethnicity: Again, the highest age-adjusted rate was among whites (13.1 per 100,000 population), followed by Hispanics (11.4 per 100,000 population) then Asians (10.4 per 100,000 population), and blacks (7.8 per 100,000 population, Figure 4). The rates for whites and Asians were higher than the five-year average (12.7 and 8.9 per 100,000, respectively). The rates for Hispanics and blacks were lower than the five-year average (11.9 and 10.0 per 100,000, respectively).

Location: Harbor Health District in SPA 8 had the highest district rate with 17.0 cases per 100,000. The lowest district rate was in El Monte Health District (SPA 3) with 4.4 cases per 100,000. Of all SPAs, SPA 6 had the highest rate with 12.6 cases per 100,000 (Figure 5).This increase may be due to the changing demographics in the area. SPA 8 had the lowest rate at 10.1 cases per 100,000. No single SPA had a rate significantly higher or lower than LAC average.

Figure 4
Salmonellosis
Age-Adjusted Rates by
Race/Ethnicity
LAC, 2007

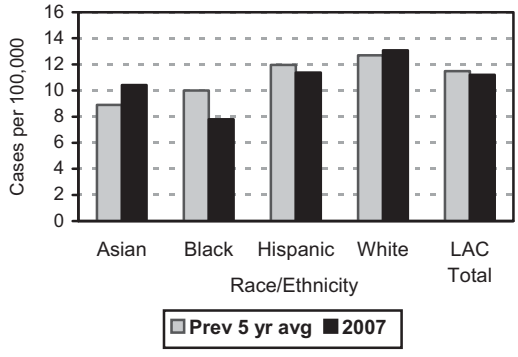


Figure 5
Salmonellosis
Incidence Rates by
Service Planning Area
LAC, 2007

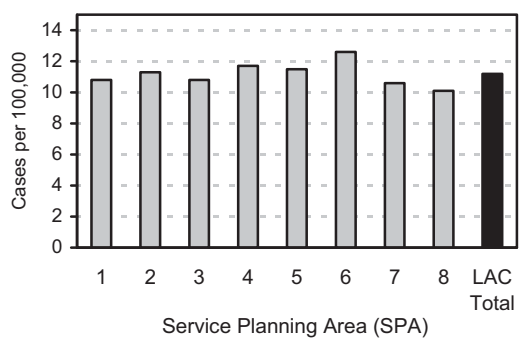


Table 2. Salmonellosis Outbreaks in LAC, 2007

Onset Month	Outbreak Setting	Total # Ill	Culture Positive	Serotype	Suspect Vehicle	Suspect Source
March	Community	6	6	*S.* Agona	Unknown food vehicle	Undetermined
March	Community	3	3	*S.* Montevideo	Sprouts	Sprouts
June	Church	15	4	*S.* Heidelberg	Homemade Food	Cross Contamination/Raw Poultry
August	Restaurant	40	18	*S. enteritidis*	Eggs Benedict	Shell Eggs
November	Private Home	11	7	*S. enteritidis*	Unknown food vehicle	Unknown food source
TOTAL		75	38			

COMMENTS

After a peak in 1994, from 1995 through 2000, a steady decline occurred in the LAC rate of salmonellosis. The LAC rate has been relatively stable or ranged between 10-13 since 2002 (Figure 2). Continued surveillance is necessary to determine long term trends.

Travel was noted as a risk factor for 16.3% of cases (n=176); of those 33.5% traveled domestically. Of those who traveled outside of the United States, 44.5% (n=52) traveled to Mexico.

There were five salmonellosis outbreaks during 2007 compared to nine identified in 2006. Two outbreaks were serotype e*nteritidis*, the others involved multiple serotypes (Table 2). Outbreak-related cases (both confirmed and presumptive) made up 7% of total cases in 2007 compared to 4.3% of total cases in 2006. This year *Salmonella Enteritidis*, the predominant serotype for 2007, was found to be the cause for two outbreaks with a total of 51 cases. Only one salmonellosis outbreak investigation cited restaurant food as a source compared to three in 2006. The use of PFGE and comparison of PFGE patterns with other laboratories through PulseNet, the national molecular subtyping network, continues to help identify potentially related clusters within LAC.

Salmonellosis was reported as a contributing cause of death in two people, both of whom had underlying health problems such as cancer and chronic disease. These cases were 80 years of age or older.

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.

Reptile-associated salmonellosis (RAS) has been a consistent problem in LAC and nationally for 15 years. In 2007, 10.5% of cases (n = 113) had some type of reptile exposure, most of which were turtle related. Despite press releases, pamphlets and periodic sweeps of areas where turtles are sold, these

animals remain popular and many people are not aware of laws controlling their sale. When RAS cases occur, District Public Health Nurses should educate case patients and their families on the risk related to reptiles. Emphasis is on the following:

- Always wash hands thoroughly with soap and water after handling reptiles or their cages and equipment;
- Owners and potential purchasers of reptiles should be educated about the risk of acquiring salmonellosis from these animals;
- Persons at increased risk for infection, such as children less than 5 years of age and imunocompromised persons should avoid both direct and indirect contact with reptiles;
- Reptiles are inappropriate pets for households with children less than 5 years of age and immunocompromised persons. If expecting a new child, remove pet reptiles from the home before the child arrives and thoroughly clean the home;
- Reptiles should not be kept in preschools and child care facilities.

ADDITIONAL RESOURCES

General information about salmonellosis—
http://www.cdc.gov/nczved/dfbmd/disease_listing/salmonellosis_gi.html

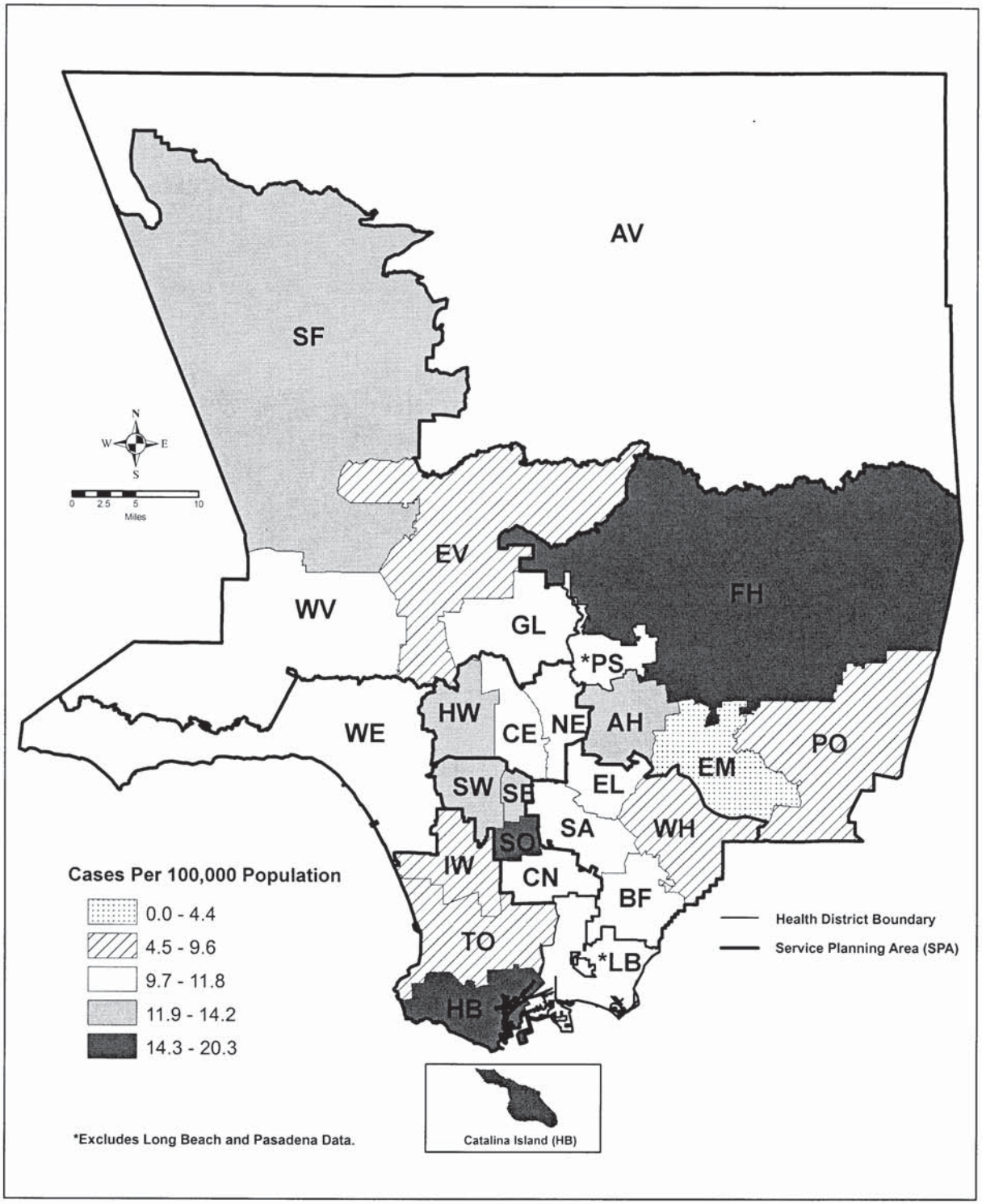
General information and reporting information about this and foodborne diseases in LAC—
www.lapublichealth.org/acd/food.htm

Reptile-associated salmonellosis information—http://www.lapublichealth.org/acd/Diseases/Reptiles.htm

Centers for Disease Control and Prevention (2003). Reptile-associated salmonellosis--selected states 1998-2002. *Morbidity and Mortality Weekly Report*, 52(49), 1206-1209.

Centers for Disease Control and Prevention (2004). Salmonellosis associated with pet turtles--Wisconsin and Wyoming, 2004. *Morbidity and Mortality Weekly Report*, 54(9), 223-226.

Map 12. Salmonellosis
Rates by Health District, Los Angeles County, 2007*



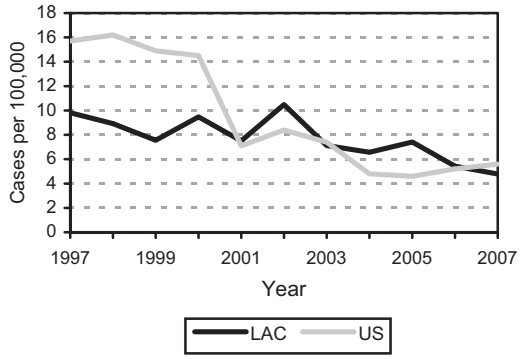
SHIGELLOSIS

CRUDE DATA	
Number of Cases	463
Annual Incidence[a]	
LA County	4.78
California	3.25[b]
United States	5.6[b]
Age at Diagnosis	
Mean	24.65
Median	21
Range	<1– 98

[a] Cases per 100,000 population.

[b] Calculated from Final 2007 Reports of Nationally Notifiable Infectious diseases issue of MMWR (57:901, 903-913).

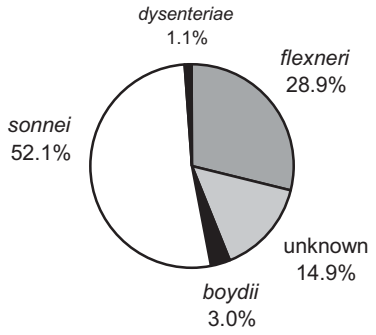
Figure 1
Shigellosis
Incidence Rates by Year of Onset
LAC and US, 1997–2007



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, 2007 (N=463)



DISEASE ABSTRACT

- There was an 11.6% decrease in reported cases in 2007.
- Three shigellosis-associated outbreaks were investigated in 2007.
- In 2007, incidence peaked in July and other months stayed below the five-year average through the entire year (Figure 3). This was due primarily to a large outbreak and several family clusters during the month of July. The rate of travel related cases that occurred from July through September decreased to 44% when compared to 60% in 2006.

STRATIFIED DATA

Trends: There was an 11.6% decrease in the number of cases during 2007. This is lowest rate in over twenty years. The rate in LAC continues to decline since peaking in 2005 (Figure 1).

Serotypes: In 2007, *S. sonnei* (n=241; 52.1%) represented a smaller percentage of case when compared to 2006 (n=315; 60%) but remains the dominant serotype. Other serotypes identified during 2007 include: *S. flexneri* (n=134), *S. dysenteriae* (n=5), and *S. boydii* (n=14) (Figure 2).

Age: Infants less than 1 year (8.7 per 100,000) and children 1–4 (17.3 per 100,000) had the highest rates. The rate for children aged 1-4 years was significantly higher than all other age groups but below the five-year average. Infants had the highest rates above the five-year average (Figure 4). The rates for adults between the ages of 45 and 65+ were significantly lower than the county average.

Race/Ethnicity: During 2007, Hispanics aged 1-4 years again had the highest age-adjusted rate (Figure 5). Hispanic children aged <1, 1-4 and 5-14 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 Hispanics may be possible causes. Black adults aged 45-55 years, had a higher rate than other ethnicities.

Location: The rates for SPA 6 (7.65 per 100,000) and SPA 4 (6.89 per 100,000) were significantly higher than the county average (4.77 per 100,000). The increase in SPA 6 is consistent with previous years. The rate for SPA 8 (2.5 per 100,000) was significantly lower than the county average. The three outbreaks involved cases from all SPAs except for SPA 1. The majority of men who have sex with men (MSM) cases (50%) were seen in SPA 4.

Severity of Illness: Fourteen percent of shigellosis cases (n=66) were hospitalized for at least two days. There were no shigellosis-associated deaths reported.

Risk Factors: Exposure to a case inside or outside the household (21%) and foreign travel (16%) were the most commonly reported potential sources of infection. The majority of foreign travel–associated illness (42%) involved visiting Mexico. Four of the 14 *S.boydii* cases reported travel to Mexico, Pakistan, and within the US. Two of the five *S. dysenteriae* traveled to Peru and Egypt during the incubation period. In 2007, three percent of cases were in MSM compared to five percent in 2006.

Figure 3
Shigellosis
Cases by Month of Onset
LAC, 2007

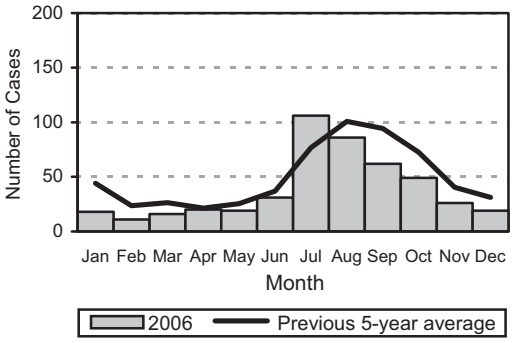


Figure 4
Shigellosis
Incidence Rates by Age Group
LAC, 2007

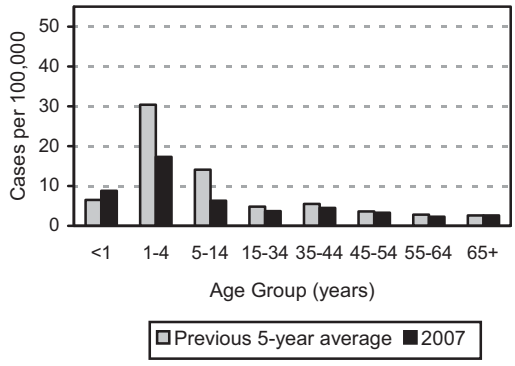
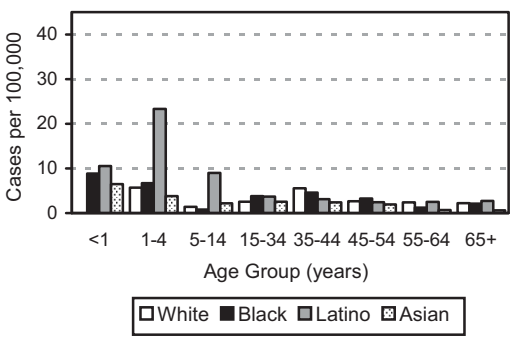


Figure 5
Shigellosis Incidence Rates
by Age Group and Race
LAC, 2007 (N=463)



COMMENTS

There were three shigellosis outbreaks investigated in 2007, all were laboratory confirmed. One was a community outbreak involving a day care setting, the second was a foodborne outbreak involving a restaurant, and the third involving a board and care facility. There was no source identified in any of the outbreaks that were investigated.

Certain sexual practices—especially those in which there is direct contact with fecal material—are a potential source of infection. There were 12 shigellosis cases reported in MSM in 2007. No links could be established among these cases. *S. flexneri* (83%) was again the predominant serotype in 2007 for this risk group; in 2002 the predominant MSM serotype was *S. sonnei* (56%).

PREVENTION

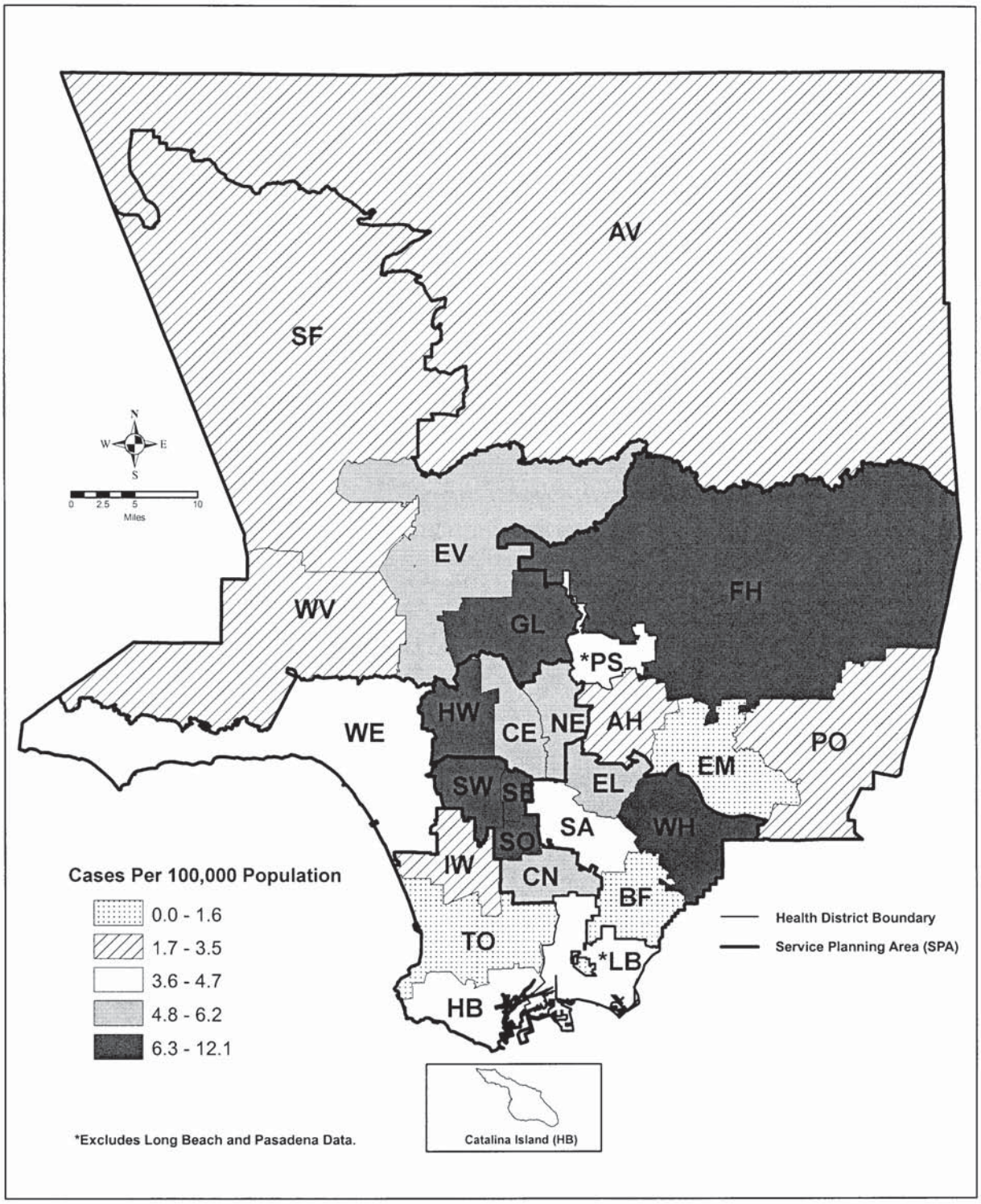
Hand washing is vital in preventing this disease. Young children or anyone with uncertain hygiene practices should be monitored to promote compliance. Hand washing is especially important when out in crowded areas such as amusement parks or shopping malls. Children should not be allowed to swim or wade 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.

ADDITIONAL RESOURCES

CDC General Information – http://www.cdc.gov/nczved/dfbmd/disease_listing/shigellosis_gi.html

LAC General Information – http://www.lapublichealth.org/acd/Diseases/Shigellosis.htm

Map 13. Shigellosis
Rates by Health District, Los Angeles County, 2007*



INVASIVE GROUP A STREPTOCOCCUS (IGAS)

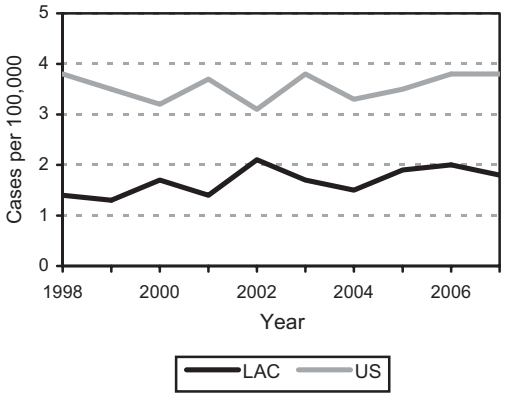
CRUDE DATA	
Number of Cases	171
Annual Incidence[a]	
LA County	1.8
California	---[c]
United States[b]	3.8[b]
Age at Diagnosis	
Mean	52
Median	54
Range	2 months–97 years

[a] Cases per 100,000 population.

[b] National projection of IGAS incidence from Active Bacterial Core Surveillance areas data, 2006 [1]. Data available beginning in 1997.

[c] Not notifiable.

Figure 1
IGAS
Incidence Rates by Year of Onset
LAC and US[b], 1998–2007



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 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 superficial 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

- The 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 1.8 per 100,000 (N=171) during 2007, similar to 2006 where 2.0 cases per 100,000 (N=197) were reported (Figure 1).

Seasonality: Although cases were observed throughout the year, a winter/spring seasonality commonly associated with streptococcal pharyngitis was observed during the spring and winter months. The number of cases in 2007 peaked in May (Figure 2) whereas the highest number of cases for the previous 5 years occurred in April.

Age: The age of cases ranged from 12 months to 97 years with a mean of 52 years and median of 54 years. For all age groups, the incidence rate was equal to or lower than the previous 5-year average with the exception of cases aged 55-64 years. In this group the 2007 incidence rate was slightly higher than the average incidence rate for the previous 5-years. The highest rate of cases occurred in those aged 65 years and older (Figure 3).

Gender: The male-to-female rate ratio decreased from 2:1 in 2005 and 2006 to 1.3:1 in 2007.

Race/Ethnicity: Race/ethnicity was known for 87% of cases. The percentage of cases that were black increased from 14% (n=23) in 2006 to 22% (n=33) in 2007. The incidence rate among blacks was the highest overall and increased from 2.7 per 100,000 in 2006 to 3.9 per 100,000 in 2007 (data not shown).

Location: The incidence rates for SPAs 2, 5, and 6 were higher compared to LAC overall with the highest rate occurring in SPA 6 (3.3 cases per 100,000). The incidence rates for all other SPAs were lower than that of LAC overall (Figure 4). However, stratification of cases by SPA produced small numbers and unstable rates for all SPAs except SPAs 2, 3, and 6.

Clinical Presentation: IGAS cases presented most often with cellulitis and bacteremia (Table 1). The number of cases presenting with pneumonia increased from 9% in 2006 to 13% in 2007. The percentage of cases with STSS (12%) and necrotizing fasciitis (7%) remained approximately equal. Clinical presentation data was available for 88% of cases.

The case fatality rate increased from 10% in 2005 and 14% in 2006 to 17% in 2007. This rate exceeds the 2007 national estimate of 11% (CDC). Of the 15 cases that met the criteria for STSS and for which outcome information was available, 7 (47%) died.

Risk Factors: Risk factor information was collected for 86% of cases, 33% of which reported no risk factors. Diabetes was reported more than any other risk factor (25%), followed by chronic heart disease (19%), alcohol abuse (14%), history of blunt trauma (10%), and malignancy (8%). Alcohol abuse and history of blunt trauma were more common in cases less than 50 years while

Figure 2
IGAS
Cases by Month of Onset
LAC, 2007

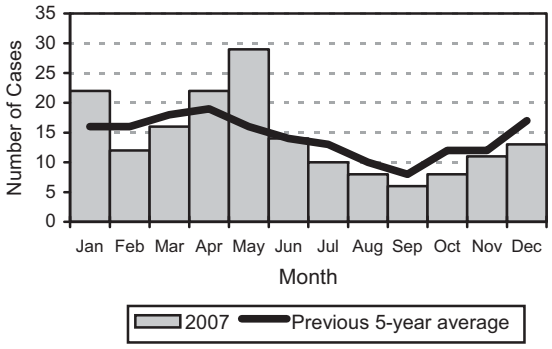
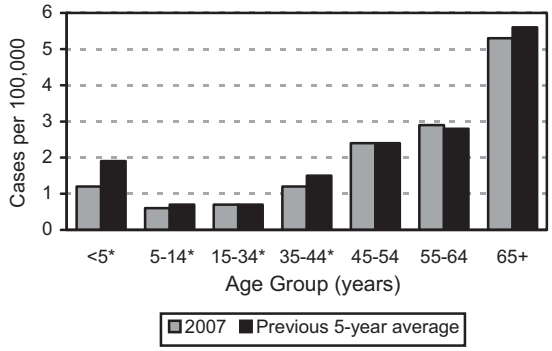
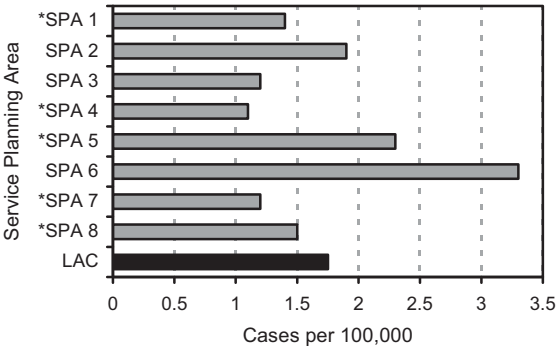


Figure 3
IGAS Incidence Rates by Age Group
LAC, 2007 (n=166)



* Small numbers prduced unstable rates in these age groups.

Figure 4
IGAS Rates by Service Planning Area
LAC, 2007 (n=166)



* Small numbers produced unstable rates for these SPAs.

diabetes, chronic heart disease, and malignancy were more prevalent in cases older than 50 years (data not shown).

Table 1. Frequency and Percentage of IGAS Clinical Syndromes, LAC, 2007

Syndrome	Number	Percent*
Cellulitis	42	28
Bacteremia (without focus)	37	25
STSS	18	12†
Non-Surgical Wound Infection	17	11
Pneumonia	20	13
Necrotizing Fasciitis	10	7
Other	51	34

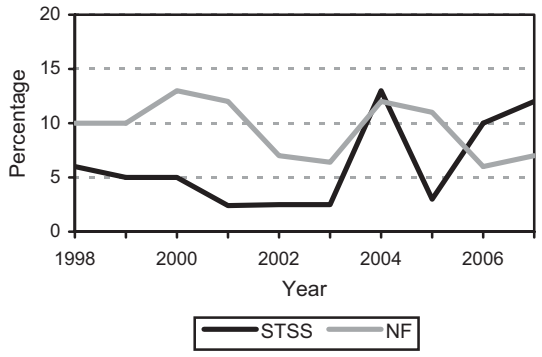
* Overlapping syndromes will total over 100%.
† Denominator data is slightly different for STSS than other syndromes (n=153 for STSS, n=150 for all other syndromes).

COMMENTS

The incidence rate of IGAS has remained relatively unchanged. However, certain demographic groups, including persons aged 65 years and older and blacks, remain at greater risk of infection. In previous years, SPA 5 had the highest incidence rate in the county. However, in 2007, the highest incidence rate was seen in SPA 6 (3.3 cases per 100,000). This change reflects both a decrease in incidence in SPA 5 as well as an increase in incidence in SPA 6. It is uncertain whether this was due to reporting bias or if true changes in incidence of IGAS occurred in these SPAs.

While the percentage of STSS and necrotizing fasciitis cases remained about the same as 2006, the overall case fatality rate increased. Of the 18 STSS cases in 2007, the outcome was known for 15 cases (83%). Of these cases, 7 were fatal (47%). The high case fatality associated with STSS suggests that IGAS case fatality is strongly affected by STSS incidence.

Figure 5
IGAS Clinical Presentation of STSS and NF
LAC, 1998–2007



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.

PREVENTION

The spread of IGAS can be prevented by good hand washing. CDC guidelines for good hand washing can be found at http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5605a4.htm. All wounds should be kept clean and monitored for signs of infection such as redness, swelling, pus, and pain. A person should seek medical care if any signs of wound infection are present especially if accompanied by fever. High risk groups such as diabetics are encouraged to seek medical care sooner particularly if experiencing fever, chills, and any redness on the skin.

REFERENCE

Centers for Disease Control and Prevention (2007). Active Bacterial Core Surveillance Reports from 1997 to 2007-*Provisional*. Report available at: www.cdc.gov/ncidod/dbmd/abcs/survreports.htm

ADDITIONAL RESOURCES

- General Information – http://www.cdc.gov/ncidod/dbmd/diseaseinfo/groupastreptococcal_g.htm
- National Institutes of Health – http://www.niaid.nih.gov/factsheets/strep.htm
- IGAS in Los Angeles County –

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

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

IGAS Publications:

American Academy of Pediatrics Committee on Infectious Diseases (1998). Severe invasive group A streptococcal infections: a subject review. *Pediatrics*, 101(1), 136-140.

Centers for Disease Control and Prevention (2002). Prevention of invasive group A streptococcal disease among household contacts of case patients and among postpartum and postsurgical patients. *Clinical Infectious Diseases*, 35(8), 950-959.

O'Brien, K.L., Beall, B., Barrett, N.L., Cieslak, P.R., Reingold, A., Farley, M.M., et al. (2002). Epidemiology of invasive group A streptococcal disease in the United States, 1995-1999. *Clinical Infectious Diseases*, 35(3), 268-276.

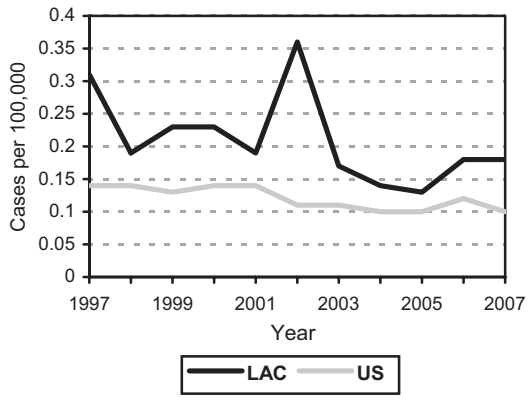
Kaul, R., McGeer, A., Low, D.E., Green, K., Schwartz, B. (1997) Population-based surveillance for group A streptococcal necrotizing fasciitis: clinical features, prognostic indicators, and microbiologic analysis of seventy-seven cases. *American Journal of Medicine*, 103(1), 18-24.

TYPHOID FEVER, ACUTE

CRUDE DATA	
Number of Cases	17
Annual Incidence[a]	
LA County	0.18[b]
California	0.16[c]
United States	0.10[c]
Age at Diagnosis	
Mean	36.4
Median	31.0
Range	13-75

[a] Cases per 100,000 population.
[b] Rates based on less than 19 observations are unreliable.
[c] Calculated from Final 2007 Reports of Nationally Notifiable Infectious diseases issue of MMWR (57:901, 903-913).

Figure 1
Acute Typhoid Fever
Incidence Rates by Year of Onset
LAC* and US, 1997–2007

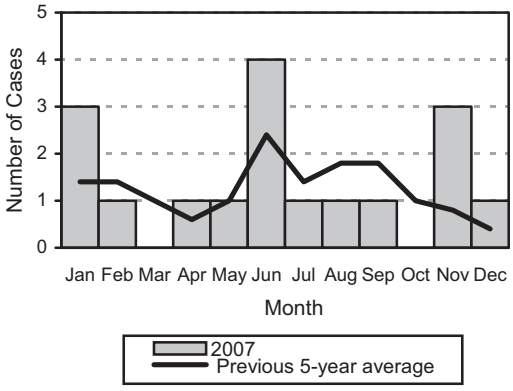


* Rates based on less than 20 observations are unreliable.

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, 2007



DISEASE ABSTRACT

- Travel was the most common risk factor identified in LAC; 82.3% of cases reported travel to typhoid endemic countries. One case recently immigrated from an endemic country.
- Fifty-three percent of cases were Asian in 2007.

STRATIFIED DATA

Trends: The yearly incident has decreased after a peak in 2002 however, there was an increase in cases in 2006 but remains stable in 2007.

Age: In 2007, 59% of acute cases were in adults consistent with the five-year average (Figure 3).

Race/Ethnicity: In 2007, acute typhoid cases occurred in Asians and Latinos. There was one white case reported (Figure 4). Black cases are rare. In 2007, Asian cases increased compared to the five-year average.

Figure 3
Acute Typhoid Fever by Age Group
LAC, 2007

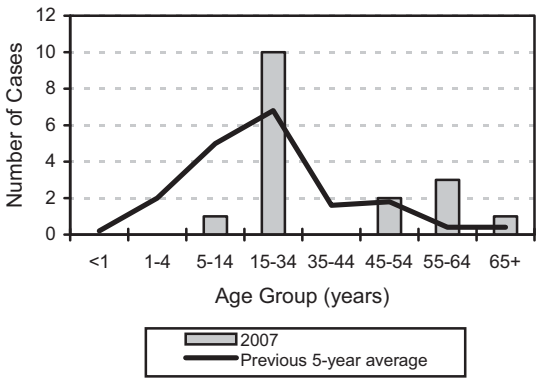
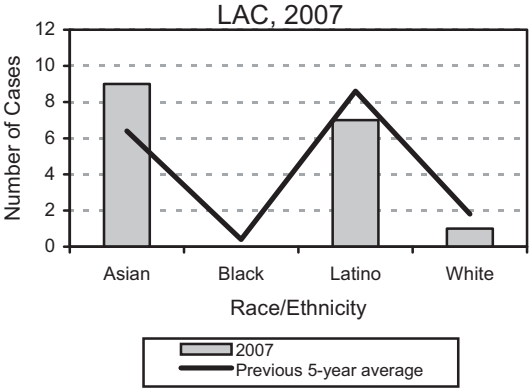


Figure 4
Acute Typhoid Fever by
Race/Ethnicity
LAC, 2007



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 at appropriate temperature; bottled water should be used for drinking as well as for brushing teeth and making ice. Vaccination should be considered when traveling in high endemic areas. LAC tests household contacts of confirmed cases for *S. typhi* to identify any previously undiagnosed carriers or cases.

COMMENTS

The majority of cases (n=14, 82.3%) traveled to endemic areas outside the US; Pakistan, India, Bangladesh, Philippines, and Cambodia were reported travel destinations. Some of the cases (n=6, 35%) traveled to India. Typhoid fever may have been a contributing cause to of death in one case.

ADDITIONAL RESOURCES

CDC General Information – http://www.cdc.gov/ncidod/dbmd/diseaseinfo/typhoidfever_g.htm

CDC Traveler's Health Information – http://wwwn.cdc.gov/travel/yellowBookCh4-Typhoid.aspx

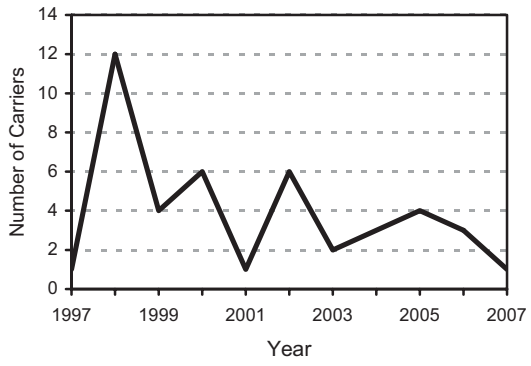
LAC General Information – http://www.lapublichealth.org/acd/Diseases/TyphoidCase.htm

TYPHOID FEVER, CARRIER

CRUDE DATA	
Number of New Carriers	1
Total Number of Carriers	10
Annual Incidence[a]	
LA County	N/A[b]
United States	N/A
Age at Diagnosis	
Mean	N/A
Range	N/A

[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, 1997–2007



DESCRIPTION

The chronic typhoid carrier state can occur following symptomatic or subclinical infections of *Salmonella typhi*. Chronic carriers of typhoid are, by definition, asymptomatic. Transmission may occur person-to-person or by ingestion of food or water contaminated by the urine or feces of acute cases or carriers. Humans are the only known reservoir for S. 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 was one new carrier of typhoid fever identified in 2007.
- All typhoid carriers are monitored semi-annually and reported to the state registry. During 2007, no carriers of typhoid were closed at the state level. A total of 10 carriers remained under case management in LAC at the end of 2007.

COMMENTS

The single new carrier was foreign born. Previously unknown carriers are sometimes identified when testing household contacts to a new acute typhoid cases for S. *typhi.* The single new carrier was not associated with any acute cases. The carrier was identified during a cholecystectomy.

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

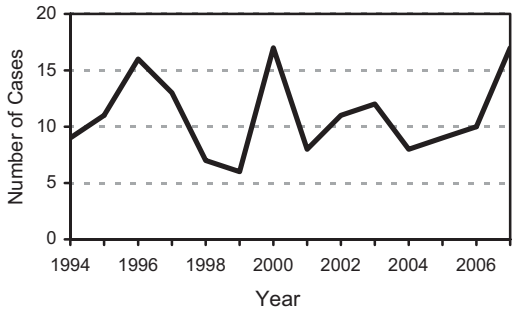
CDC General Information – http://www.cdc.gov/ncidod/dbmd/diseaseinfo/typhoidfever_g.htm
LAC General Information – http://www.lapublichealth.org/acd/Diseases/TyphoidCarrier.htm

TYPHUS FEVER

CRUDE DATA	
Number of Cases	17
Annual Incidence[a]	
LA County	0.18[b]
United States	N/A
Age at Onset	
Mean	39
Median	46
Range	4–65 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, 1994–2007



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.

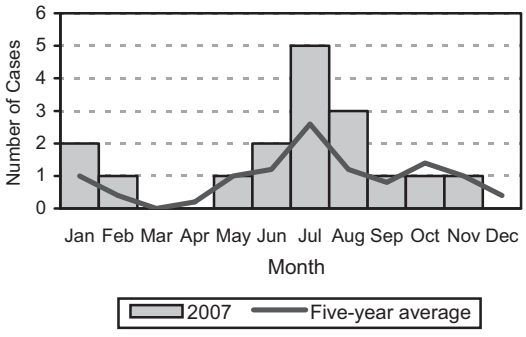
DISEASE ABSTRACT

- The number of cases reported in 2007 (n=17) is a 70% increase from 2006 and continues a rise since 2004.
- No outbreaks occurred. However, two cases were linked to visiting a park in San Marino.
- There continues to be increased reports of typhus in LAC Health Districts where typhus has not historically been often seen.

STRATIFIED DATA

Trends: Seventeen cases were reported in 2007, a 70% increase from 2006 (n=10). This number is equivalent to the highest ever reported to LAC DPH when seventeen was also reported in 2000 (Figure 1).

Figure 2
Murine Typhus
Cases by Month of Onset
LAC, 2007



Seasonality: In 2007, a substantial number of cases occurred in July and August (Figure 2). 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.

Age: In 2007, the mean and median ages were 39 and 46 years, respectively. Ages of cases ranged from 4 to 65 years; the largest number of cases occurred in those between 45 and 54 years old (n=6, 35%) (data not shown).

Sex: There were almost twice as many cases reported among females as males. The male-to-female case ratio was 1:1.8. In the past in LAC, gender had been distributed evenly.

Race/Ethnicity: Most cases were of white race/ethnicity (n=12, 71%). One case each (6%) occurred in a Hispanic and an Asian (data not shown). Two cases (12%) had unknown race/ethnicity information.

Location: More than half of the cases (n=11, 65%) 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 six cases resided in the West (n=4, 24%), Torrance (n=1, 6%), and Bellflower (n=1, 6%) 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. Almost half of the cases in 2007(n=8, 47%) 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 keeping pets or observing other types of small mammals (e.g., rats, opossums) on their residential property, and thus may have had exposure to animals that carry fleas. The single case that denied having pets or seeing animals around his residence resided near Griffith Park and had substantial foliage around his home.

COMMENTS

The rise in confirmed cases in 2007 continues an increase seen since 2004. No outbreaks occurred; however, a cluster of two cases were reported with onset in July and was linked to visiting a park in San Marino. The occurrence of typhus in localities where typhus is not usually seen (e.g., West and Bellflower Health Districts) also substantially contribute to the number seen this year. Results from a CDPH/CDC study of fleas collected from opossums from the Long Beach/Orange County outbreak in 2006 indicate that *R. felis* may be the main infectious agent in those jurisdictions. It is possible that *R. felis* is a main infectious agent in adjacent LAC areas as well. On the other hand, 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.

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.

ADDITIONAL RESOURCES

General information about typhus fever is available from the ACDC website at:
http://www.lapublichealth.org/acd/vectormurine.htm

Publications:

Azad, A.F., Radulovic, S., Higgins, J.A., Noden, B.H. & Troyer, J.M. (2007). Flea-borne rickettsioses: ecologic considerations. *Emerging Infectious Diseases*, 3(3), 319–327.

Civen, R. & Ngo, V. (2008). Murine typhus: an unrecognized suburban vector-borne disease. *Clinical Infectious Diseases*, 46, 913-918.

Sorvillo, F.J., Gondo, B., Emmons, R., Ryan, P., Waterman, S.H., Tilzer, A., et al. (1993). A suburban focus of endemic typhus in Los Angeles County: association with seropositive domestic cats and opossums. *American Journal of Tropical Medicine and Hygiene*, 48(2), 269–273.

Williams, S.G., Sacci, J.B., Schriefer, M.E., Andersen, E.M., Fujioka, K.K., Sorvillo, F.J., et al. (1992). Typhus and typhuslike rickettsiae associated with opossums and their fleas in Los Angeles County, California. *Journal of Clinical Microbiology*, 30(7), 1758–1762.

VIBRIOSIS

CRUDE DATA	
Number of Cases	14
Annual Incidence[a]	
LA County	0.15
United States	0.20
Age at Diagnosis	
Mean	44
Median	43
Range	14–86 years

[a] Cases per 100,000 population.

Figure 1
Selected Vibrio Species
Cases by Year of Onset
LAC, 1997–2007

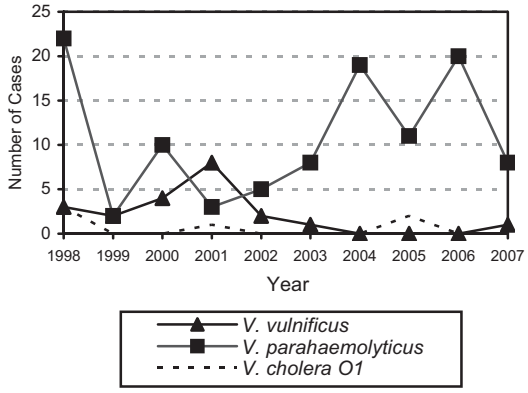
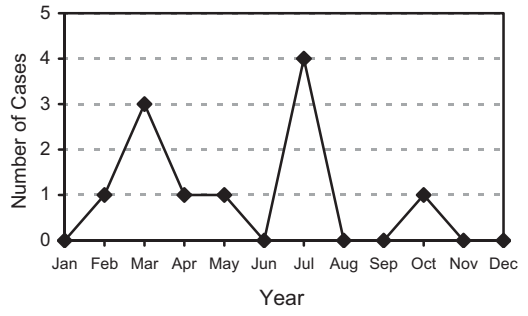


Figure 2
Vibriosis
Cases by Month of Onset
LAC, 2007



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. parahæmolyticus*, 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.

DISEASE ABSTRACT

- Thirteen cases of vibriosis were reported in 2007, a significant decrease from 33 cases reported in 2006. None was fatal.
- There were four cases of *V. alginolyticus* infections, two of which were related to recreational water exposures, one of which was a work-related injury, and one case whose risk factors could not be identified. There was one case of *V. cholerae* non-O1/non-O139 sepsis; risk factors were undetermined. There was one case of *V. vulnificus* in a man who had eaten seafood. No cases of toxigenic *V. cholerae* O1/O139 were reported in 2007.

STRATIFIED DATA

Seasonality: Among reported vibriosis cases with distinct onset dates, the majority (62%, n=8) 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 11 years old. The average age of cases was 44 years, median age was 36 years (Table 1).

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, 2007

Species	No. of cases	Race (no. of cases)	Mean Age, years (range)	Sex Ratio M:F
V. parahæmolyticus	8	Asian (3), Hispanic(5), white (12), black (0)	45 (14-86)	0.81:1
V. cholerae non-O1/O139	1	Asian (1)	79 (79)	0:1
V. alginolyticus	4	Hispanic (2), white (2)	54.5 (54-55)	2:0
V. vulnificus	1	Hispanic (1)	69 (69)	1:0
V. furnissii	0	n/a	n/a	0:0

Species-specific Risk Factors:

Vibrio parahæmolyticus
Eight cases of *V. parahæmolyticus* were reported during 2007. All 8 were identified through stool culture. Four reported eating seafood recently, with three specifying raw oysters.

***Vibrio cholerae* non-O1/O139**
One case of non-toxigenic *V. cholerae* gastroenteritis was reported in 2007. Its risk factors could not be determined. The case denied eating raw seafood, and had not travelled internationally in years.

Vibrio alginolyticus
Two *V. alginolyticus* infections were wound infections, one of which was caused by a work injury. The other wound infection was in a man suffering from complications due to diabetes. Two *V. alginolyticus* infections were in girls who had recreational seawater exposure.

COMMENTS

In LAC, risk of *Vibrio* infection can be reduced by not eating raw fish and shellfish. In 2007, there was a dramatic reduction in *V. parahæmolyticus* cases from the previous year. This is probably a result of close oversight by oyster harvesters in Washington State, following the *V. parahæmolyticus* outbreak in 2006. 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. However in 2007 males and females were equally to cite recreational water exposures as a risk factor for their infections.

There was a higher number of *V. alginolyticus* cases in 2007 than in the previous three years. The two cases that occurred in females were related to recreational water exposure. Warmer summer temperatures may be affecting the growth of *Vibrio alginolyticus* in the water.

ADDITIONAL RESOURCES

Mouzin, E., Mascola, L., Tormey, M.P. & Dassey, D.E. (1997). Prevention of *Vibrio vulnificus* infections. Assessment of regulatory educational strategies. *Journal of American Medical Association*, 278(7), 576-578. Retrieved November 10, 2008, from the JAMA Web site: http://jama.ama-assn.org/cgi/content/abstract/278/7/576

More information on Centers for Disease Control and Prevention — http://www.cdc.gov/nczved/dfbmd/disease_listing/vibriov_gi.html

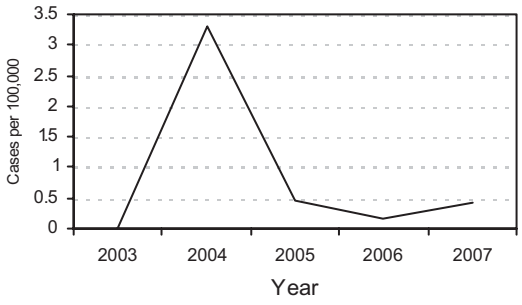
WEST NILE VIRUS

CRUDE DATA	
Number of Cases	43
Annual Incidence[a]	
LA County	0.44
California[b]	1.04
United States[b]	1.20
Age at Diagnosis	
Mean	61.5
Median	62
Range	15–94 years

[a] Cases per 100,000 population.

[b] Incidence calculated with 2007 population estimates from www.census.gov.

Figure 1
West Nile Virus Disease
Incidence Rates by Year of Onset
LAC, 2003-2007



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.

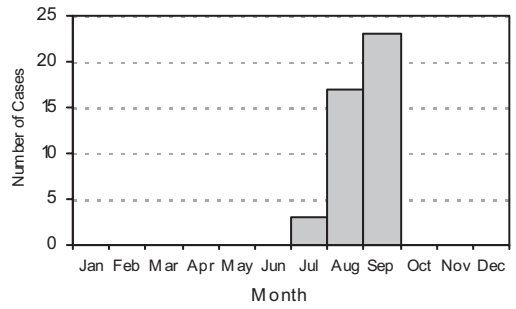
The life cycle of the virus involves the transmission of the virus between mosquitoes and bird reservoir hosts. Humans are incidentally infected when bitten by an infected mosquito, usually a *Culex* or *Anopheles* species. The incubation period for human infection is 2 to 14 days. Birds, especially 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.

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. Additional routes of transmission that have been documented include transplantation of WNV-infected organs, 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. 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. Approximately one in 150 patients will develop more severe illness, manifesting as WNV neuro-invasive disease (NID). WNV NID includes encephalitis, meningitis, and acute flaccid paralysis (AFP). WNV-associated encephalitis is commonly associated with 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.

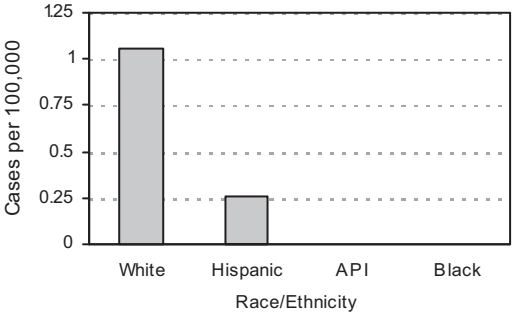
Figure 2
West Nile Virus Disease
Cases by Month of Onset
LAC, 2007 (N=43)



DISEASE ABSTRACT

- The overall incidence of reported WNV infections in 2007 was 0.44 cases per 100,000 population, rising from a low of 0.17 in 2006 when only 16 cases were confirmed (Figure 1).
- Case fatalities (n=5) occurred for the first time since 2004.
- Meningitis continues to be the most commonly reported clinical condition, comprising 32% (n=14) of cases.
- Most WNV infections occurred in persons residing in San Fernando Valley.

Figure 3
West Nile Virus Disease
Incidence Rates by Race/Ethnicity
LAC, 2007



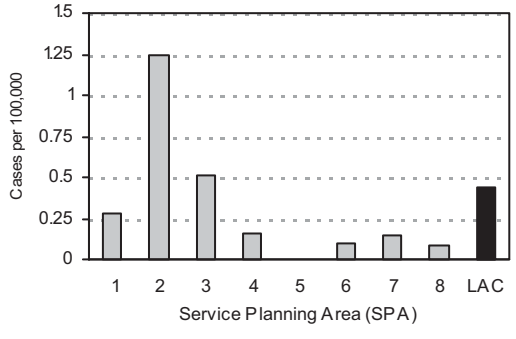
STRATIFIED DATA

Trends: WNV infection, including in asymptomatic blood donors, occurred at an incidence rate of 0.44 per 100,000 population in 2007. Both the total number and incidence of WNV infection has decreased dramatically since 2004 when 309 cases were confirmed at an incidence of 3.3 cases per 100,000 population (incidence re-calculated with updated population estimates) (Figure 1).

Seasonality: Onset of cases occurred July through October and peaked in September (Figure 2). Since 2004, the onset of WNV cases has been limited to July through October.

Age: The median age was 62 years (range: 15–94 years). The highest incidence occurred in the 65 and over age group (1.9 per 100,000) (data not shown).

Figure 4
West Nile Virus Disease
Cases by Service Planning Area
LAC, 2007



Almost all cases (n=40, 93%) were at least 45 years old.

Sex: Over three times as many male WNV cases were reported than female cases, a rate ratio of 3.4:1. The incidence rates were 0.68 cases and 0.20 cases per 100,000, respectively.

Race/Ethnicity: In 2007, WNV cases occurred only in whites and Hispanics, with whites accounting for the greatest proportion of reported cases (72%) as well as the highest incidence rates of infection (n=31, 1.1 per 100,000). Hispanics comprised 28% of cases (n=12, 0.26 per 100,000) (Figure 3).

Location: The greatest number of reported WNV cases were reported from SPA 2, the San Fernando Valley area (n=27, 1.3 per 100,000). The second highest incidence occurred in SPA 3, the San Gabriel Valley area (n=9, 0.51 per 100,000). WNV occurred sparsely and sporadically in the remaining SPA locations (Figure 4).

Disease Severity: The WNV infections reported presented most frequently as neuroinvasive disease (n=28, 65%); 12 were diagnosed as encephalitis, 14 as meningitis, and 2 as acute flaccid paralysis. A substantial number of infections were asymptomatic blood donors (n=7, 16%). Of those symptomatic cases, 86% (n=31) were hospitalized. Five fatalities (12%) occurred in 2007, the first since 2004 when 14 deaths (5% of cases) were reported. Four of the deaths were diagnosed with encephalitis and one with WNV fever.

COMMENTS

The first symptomatic WNV case in LAC associated with 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. In response to the outbreak, LAC DPH added WNV infection to its list of reportable diseases by authority of the Health Officer under California Code of Regulations, Title 17, Sections 2511 and 2505. Physicians and laboratories are required to report all positive laboratory findings of WNV tests to the DPH within one working day.

The following years presented a markedly different picture, with numbers declining to a low of 16 in 2006. This year, however, over twice as many cases were reported. The rise in cases, as well as the continued detection of positive mosquito pools, dead birds and other reservoir animals, has demonstrated that WNV remains endemic in the LAC and southern CA region. As the number of cases has fluctuated greatly from year to year (ranging from 16 to 43 since 2005), the baseline level of cases expected for this region remains to be seen. 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.

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.

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 (SGVMVCD)
- Los Angeles County West Vector Control District (LACWVCD)
- Antelope Valley Mosquito and Vector Control District (AVMVCD)
- Compton Creek Mosquito Abatement District (CCMAD)

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: http://www.cdc.gov/ncidod/dvbid/westnile/index.htm
- California Department of Health Services: http://www.westnile.ca.gov
- Acute Communicable Disease Control Program, Los Angeles County Public Health: http://www.lapublichealth.org/acd/index.htm
- Vector Management Environmental Health, Los Angeles County Public Health: http://www.lapublichealth.org/eh/index.htm
- For additional information on EPA-registered repellants: http://www.epa.gov/pesticides/factsheets/insectrp.htm

Mosquito and Vector Control District Websites:

- Greater Los Angeles County Vector Control District: http://www.glacvcd.org
- West Los Angeles Vector Control District: http://www.lawestvector.org
- San Gabriel Valley Mosquito and Vector Control District: http://www.sgvmosquito.org
- Antelope Valley Mosquito and Vector Control District: http://www.avmosquito.org
- Mosquito and Vector Control Association of California: http://www.mvcac.org

DISEASE OUTBREAK SUMMARIES

2007

COMMUNITY-ACQUIRED DISEASE OUTBREAKS

ABSTRACT

- In 2007, 130 community-acquired disease outbreaks accounted for 1,690 cases of illness (Figure 1).
- Schools were the most common setting of community-acquired outbreaks (46%).
- The number of reported outbreaks (130) was a decreased from 2006 and below the previous 7-year average (142).

DATA

Disease outbreaks are defined as clusters of illness that occur in a similar time or place, or case numbers above baseline for a specified population or location. 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 chapter) or facilities where medical care is provided (see Healthcare Associated Outbreaks chapter).

Varicella caused most community-acquired outbreaks in LAC (32%) gastroenteritis (GE) of various etiologies, followed by ectoparasites (scabies and pediculosis) were the second and third most common cause of outbreaks, comprising 25% and 18% of all outbreaks respectively (Figure 2, Table 1). Collectively accounting for 75% of all community-acquired outbreaks in 2007, the dominance of these three disease categories is similar to past years (75% in 2006 and 2005 and 72% in 2004).

GE outbreaks, specifically caused by norovirus, had the highest incident-specific case average attributed to the seven confirmed norovirus outbreaks (mean of 35 cases per outbreak), followed by 16 undetermined GE 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 presumptive diagnosis. Important to note in 2007, due to a documented increase in county-wide norovirus activity, a reduction in collecting diagnostic viral specimens was instituted. Larger outbreaks might have been more likely to warrant additional laboratory testing. While the overall number of outbreaks for 2007 decreased from the previous year, the number of GE outbreaks (both norovirus confirmed and clinically suspect) went up in 2007. These figures highlight the increased circulation of norovirus and reflect the ease this agent can be transmitted from person-to-person in

Figure 1
Community Acquired Outbreaks
Number of Persons Affected
LAC, 2000–2007

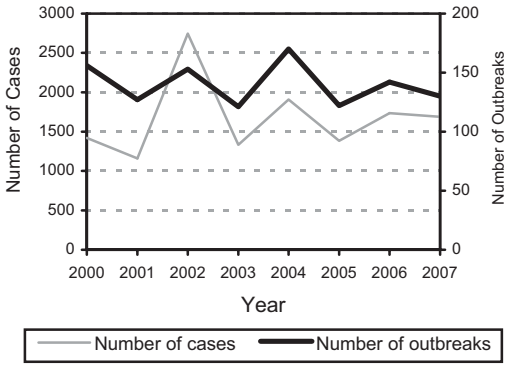
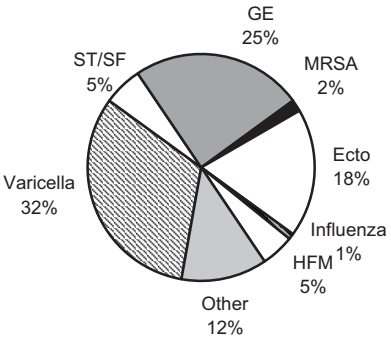
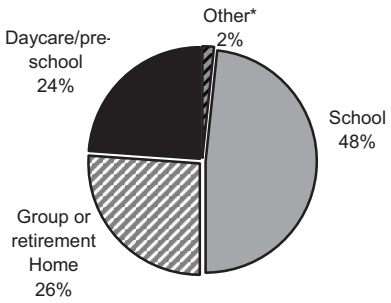


Figure 2
Community-Acquired Outbreaks by
Type of Disease* LAC, 2007 (N=130)



* ST/SF=strep throat/scarlet fever; Ecto=ectoparasites; HFM=Hand, foot & mouth; GE=Gastroentestinal; Other=unknown respiratory, RSV, impetigo, unknown febrile illness.

Figure 3
Community Outbreaks by Setting
LAC, 2007 (N=130)

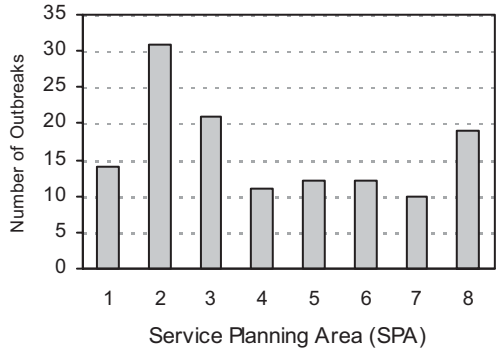


* Other includes: juvenile camp, detention center

community settings. (Table 1).

The most common outbreak settings (Figure 3) for illness transmission were schools [elementary schools (51), middle schools (8), after-school care (1), high schools (1), and universities (2)] accounting for 48% of all outbreaks. The predominance of reported outbreaks affecting children in school settings can be seen over the last several years. Settings with young children in daycare or pre-school accounted for an additional 20%. Group and retirement home settings were the second most common site of community-acquired outbreaks reported in 2007, accounting for 26% of all outbreaks. The 2006 year also reported high impact in this setting (30%). This recent two year figures more than doubles the previous five-year-average percentage of 13% ranging from 11% to 16%.

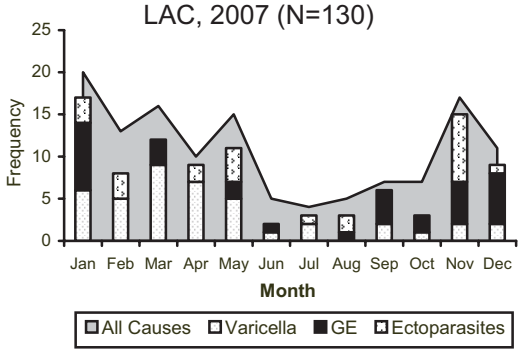
Figure 4
Community Outbreaks by SPA
LAC, 2007 (N=130)



Outbreaks were reported from all eight SPAs (Figure 4). SPA 2, in the San Fernando Valley, had the most outbreaks (31) for 2007.

The chart of community-acquired outbreaks by onset month (Figure 5) shows a bimodal distribution. Varicella outbreaks predominated the early months of the year. GE occurred throughout the year, but tended towards the cooler months with outbreaks focused in the winter, spring and fall. 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, 2007 (N=130)



COMMENTS

There was a decrease in the number of outbreaks and outbreak associated cases in 2007 from the prior year; however, the number of outbreaks in 2007 was closer to the median 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 Reports section). In 2007, eight varicella outbreaks were identified in the Antelope Valley Health District (SPA 1), where the LACDHS Varicella Acute Surveillance Project is in place, which tied SPA 2 for most reported outbreaks of varicella.
Community-acquired outbreaks result in 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 (70% in pre-school, elementary, middle, or after-school). Varicella, pediculosis (head lice), and gastroenteritis were most common in this young group. The second age group affected by outbreaks is in the older population associated with group-home settings (26%). 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 increased norovirus activity during 2007.

Table 1. Community-Acquired Outbreaks by Disease— LAC, 2007

Disease	No. of outbreaks	No. of cases	Cases per outbreak (average)	Cases per outbreak (range)
Varicella	42	515	12	5-48
Scarlet fever/strep throat	7	56	8	4-15
Scabies	14	82	6	2-16
Hand, foot & mouth disease	6	28	5	2-9
Pediculosis	10	116	12	3-26
GE illness - Norovirus	7	246	35	13-59
GE illness - Shigella	2	6	3	2-4
GE illness - Unknown	23	375	16	3-38
Fifth disease	5	46	9	5-16
Conjunctivitis	4	48	12	3-20
MRSA	2	13	7	6-7
Influenza B	1	4	4	4
Other*	7	115	22	4-45
Total	**130**	**1,690**	**13**	**2–59**

* Includes: unknown respiratory, RSV, impetigo, unknown febrile illness.

Table 2. Community-Acquired Outbreaks by Disease and Setting — LAC, 2007

Disease	Group Home[a]	School[b]	Preschool or Daycare	Other[c]	Total
Varicella	0	40	1	1	**42**
Scarlet fever/strep throat	0	4	3	0	**7**
Scabies	13	1	0	0	**14**
Hand, foot & mouth disease	0	1	5	0	**6**
Pediculosis	0	5	5	0	**10**
GE illness - Norovirus	3	1	3	0	**7**
GE illness - Shigella	1	0	1	0	**2**
GE illness - Unknown	15	2	6	0	**23**
Fifth disease (Parvovirus)	0	5	0	0	**5**
Conjunctivitis	0	1	3	0	**4**
MRSA	1	1	0	0	**2**
Influenza B	1	0	0	0	**1**
Other	0	2	4	1	**7**
Total	**34**	**63**	**31**	**2**	**130**

[a] Includes centers for retirement, assisted living, rehabilitation, and shelter.
[b] Includes elementary (51), middle (7), after-school (1), high schools (1) and university (2).
[c] Includes juvenile camp, detention center.

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 or drink (CDC, 1996).

The system used by LAC DPH for detection of foodborne outbreaks begins with a Foodborne Illness Report (FBIR) from individuals or healthcare providers. 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.

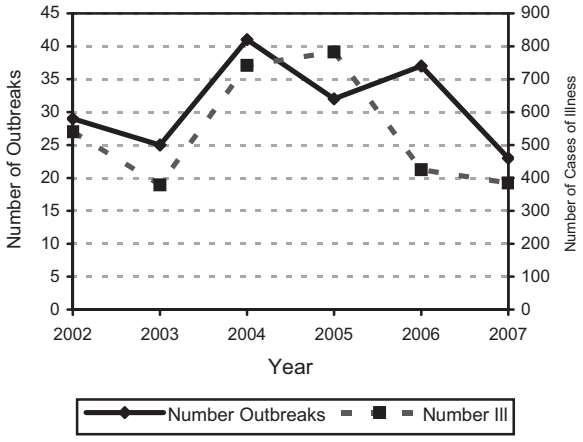
ACDC's Food Safety Unit and F&M review all FBIRs and investigate reports with the greatest public health importance. An epidemiologic investigation will typically be initiated when there are illnesses in multiple households, multiple reports from the same establishment in a short period of time, or ill individuals who attended a large event with the potential for others to become ill. The objective of each investigation is to determine the agent of infection, determine extent of the outbreak, identify a food vehicle or processing error, and take any actions needed to protect the public's health.

OVERVIEW

In 2007 there were 16% fewer FBIRs reported than in 2006 (1700 versus 2019). The F&M program contacted each individual making the FBIR, and performed a site inspection on 32% of FBIR reports that were deemed high priority (n=537). The remaining FBIRs were referred to district Environmental Health F&M inspectors or another agency for follow-up.

Of the 31 investigations conducted by ACDC that were suspected to be foodborne in 2007, 30 were conducted by the Food Safety Unit. Of the 31 investigations, 26 were initiated by FBIR complaints and 5 were initiated through other surveillance activities. Ten of the 31 outbreaks were determined to be person-to-person transmission of norovirus in a food setting and not considered to be food-related (32%). The remaining 21 outbreaks determined to be foodborne are summarized here. These 21 outbreaks encompassed 385 cases of foodborne illness with an average of 7 persons per outbreak (range 2-70 cases) (Figure 1).

Figure 1
Foodborne Outbreaks
Number of Persons Affected
LAC, 2002–2007



Seasonality: Foodborne outbreak investigations occurred throughout the year in 2007, with no seasonal pattern (Figure 2).

Implicated Food Vehicles: A food vehicle was epidemiologically implicated in 57% of foodborne outbreaks (n=12). Implicated food

items included produce (n=7), egg dishes (n=2), poultry (n=1), fish (n=1) and molé with multiple ingredients (n=1).

Agent: An agent was identified in 90% of foodborne outbreak investigations (n=19). Agents were confirmed in 57% of these outbreaks (n=12), which was an improvement over previous years (Figure 3). Reasons for no laboratory testing include lack of cooperation, delayed notification and cases being out of town or unavailable.

The most prevalent agents identified in foodborne outbreaks in 2007 were bacterial (43%, n=9), which included Salmonella (n=5), Campylobacter (n=1), Shigella (n=1), and bacterial toxin (n=2). The number of outbreaks determined to be caused by a bacterial agent in 2007 was comparable to that seen in 2006 (9 versus 8).

Norovirus was also a common agent laboratory confirmed and suspected in outbreak investigations in 2007 (38%, n=8). The number of foodborne outbreaks where norovirus was identified in 2007 was down 70% from what was seen in 2006 (8 versus 27), indicating a milder norovirus season in 2007. The LAC Public Health Laboratory tests human specimens for norovirus using the reverse transcription-polymerase chain reaction (RT-PCR) method. Results are used for confirming the etiology agent of an outbreak and not for diagnosing individual cases.

Contributing Factors: An ill food handler was identified in three outbreaks (14%). Two of these outbreaks involved the same food handler. Two food handlers were identified with hepatitis A in one outbreak. The Public Health Department provided notification and prophylaxis to persons potentially exposed to foods prepared by the ill food handlers. No patrons reported illness and no additional cases of hepatitis A were found related to the restaurant.

Outbreak Location: The most common locations for reported foodborne outbreaks were restaurants (52%, n=11) followed by food that was brought or catered to a work place (19%, n=4). Other locations include places of worship, schools, and fairs. The largest number of outbreaks were reported from SPA 2 (24%) (Table 1). There was one multi-district and one multi-county outbreak, but there were no outbreaks that involved multiple states.

Figure 2
Foodborne Outbreak Investigations
by Month of Onset
LAC, 2007 (N=21)

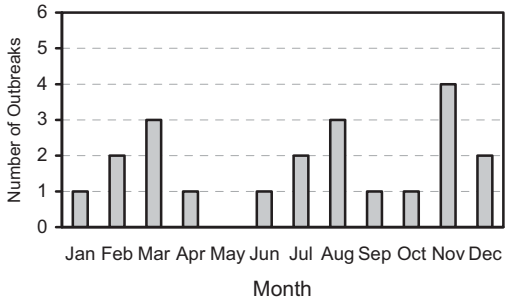


Figure 3
Foodborne Outbreaks
% with Suspected/Confirmed Etiologic Agent
LAC, 2003–2007

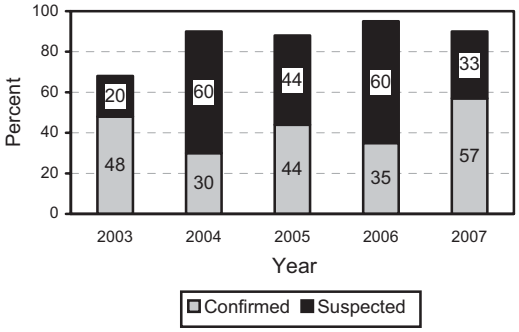


Figure 4
Foodborne Outbreaks
by Etiologic Agent Category
(Lab Confirmed and Suspect)
LAC, 2003–2007

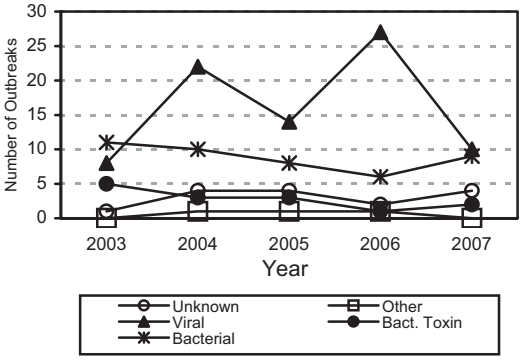


Table 1. Frequency of Foodborne Outbreaks by Location, 2007 (N=21)

SPA	Frequency	Percent
1	2	10%
2	5	24%
3	3	14%
4	4	19%
5	1	5%
6	1	5%
7	1	5%
8	3	14%
Multi-district	1	5%
Multi-county	1	5%
Multi-state	0	0%
Total	21	106%

INVESTIGATION HIGHLIGHTS

There were fewer FBIRs received from consumers by ACDC and fewer foodborne outbreaks investigation in 2007 than occurred in 2006. The reduction in FBIRs from consumers in 2007 may indicate a true decrease in foodborne illness in the community, but the actual cause is unknown. 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 as well.

The largest foodborne outbreak investigated this year by the Food Safety Unit involved two separate LAC restaurants with 89 cases identified. Laboratory results confirmed the agent in this outbreak as norovirus. The outbreak was attributed to an ill food handler working at both locations. Case control analysis of food items eaten at both restaurants implicated green salad or fruit salad items.

A large shigellosis outbreak occurred at a restaurant with most of the 72 cases identified as residing in LAC. Laboratory results confirmed the agent in this outbreak as *Shigella sonnei.* Case control analysis of food items eaten implicated pre-made salads or leafy greens in this outbreak.
A large salmonellosis outbreak occurred at a restaurant in the city of Los Angeles, with 39 cases identified. Laboratory results confirmed the outbreak as *S. enteridid*is. A hollandaise sauce made with shell eggs was implicated in the case control food analysis.

A bacterial toxin outbreak occurred among persons eating precooked fried chicken at an office luncheon with 15 cases identified. The chicken eaten at the event tested positive for high levels of bacteria (*C. perfringens)* at the LAC Public Health Laboratory. The outbreak was most likely due to mishandling of the food by event organizers.

ACDC along with LAC Community Health Services also investigated a report of two food handlers ill with Hepatitis A from two separate catering companies. Notification and prophylaxis were provided to persons potentially exposed to foods prepared by the ill food handlers. No cases of hepatitis A were subsequently reported.

Table A. Foodborne Outbreaks in LAC, 2007 (N=21)

	Agent	Species	Confirmed/ Suspected	Source	Setting	OB#	Cases	HD
1	Norovirus		Confirmed	Undetermined	Workplace	31	35	31
2	Norovirus		Confirmed	Salads	Restaurant	161	20	86
3	Norovirus		Confirmed	Salads	Restaurant	163	29	62
4	Norovirus		Suspected	Undetermined	Restaurant	70	15	5
5	Norovirus		Suspected	Undetermined	Restaurant	149	7	34
6	Norovirus		Suspected	Berries	Restaurant	181	30	86
7	Norovirus		Suspected	Undetermined	Workplace	197	11	91
8	Norovirus		Suspected	Undetermined	Restaurant	3	7	79
9	Salmonella	montevideo	Confirmed	Sprouts	Community	99	3	multi
10	Salmonella	heidelberg	Confirmed	Molé	Church	114	15	69
11	Salmonella	agona	Confirmed	Undetermined	Community	98	6	5
12	Salmonella	enteritidis	Confirmed	Eggs hollandaise	Restaurant	129	39	34
13	Salmonella	enteritidis	Confirmed	Mac Cheese	Residence	179	14	79
14	Bacterial toxin	C. perfringens	Confirmed	Chicken	Workplace	1	15	9
15	Bacterial toxin		Suspected	Beans	Residence	62	25	6
16	Campylobacter	jejuni	Confirmed	Watermelon	Fair	130	4	19
17	Shigella	sonnei	Confirmed	Wonton Salad/ Spinach	Restaurant	136	72	5
18	Tetrototoxin		Suspected	Fish	Restaurant	63	2	91
19*	Hepatitis A		Confirmed	N/A	Restaurant	46	2	84
20	Undetermined		N/A	Undetermined	Restaurant	120	5	34
21	Undetermined		N/A	Undetermined	Workplace	135	16	25

* Investigation conducted by ACDC, EHFM and DPHN

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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

Centers for Disease Control and Prevention:

- Foodborne and Diarrheal Diseases Branch – http://www.cdc.gov/enterics/
- Outbreak Response and Surveillance Team – http://www.cdc.gov/foodborneoutbreaks/
- FoodNet – http://www.cdc.gov/foodnet/
- Norovirus Information – http://www.cdc.gov/foodborneoutbreaks/

Other national agencies:

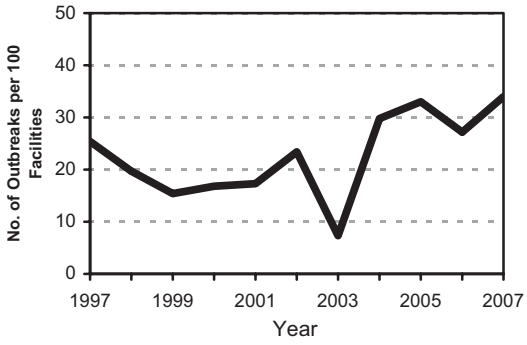
- FDA Center for Food Safety and Applied Nutrition – http://www.cfsan.fda.gov/
- Gateway to Government Food Safety Information – http://www.foodsafety.gov/

HEALTHCARE ASSOCIATED OUTBREAKS: ACUTE CARE HOSPITALS, CLINICS AND PROVIDER OFFICES

DEFINITION

Healthcare associated outbreaks occur in acute care hospitals, clinics, or other types of healthcare facilities. This chapter will discuss outbreaks in aforementioned healthcare settings excluding subacute settings such as skilled nursing facilities (see separate chapter in this report). Outbreaks in such settings are defined as clusters of nosocomial (health-facility acquired) 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.

Figure 1
Healthcare Associated
Acute Care Outbreaks
LAC, 1997–2007



ABSTRACT

Confirmed acute care hospital outbreaks increased 25% from 2006 to 2007 to the highest number in 10 years.

Table 1. Number of Reported Outbreaks in Acute Care Hospitals, Clinics and Provider Offices—LAC, 2003-2007

	YEAR				
Type of Facility	**2003**	**2004**	**2005**	**2006**	**2007**
Acute Care Hospitals	8	31	34	28	35
Clinic	0	0	0	0	1
Provider Office	0	0	0	0	2
Total	**8**	**31**	**34**	**28**	**38**

Acute Care Hospitals: There were 35 outbreaks reported in acute care hospitals in 2007 (Table 1). Thirty-four percent (n=12) occurred in a unit providing intensive or focused specialized care (e.g., NICU, cardio-thoracic unit, telemetry) (Table 2). Nine percent (n=3) occurred in the psychiatric or behavioral unit and 14% (n=5) occurred in a subacute unit located within the acute care hospital. Scabies outbreaks continues to account for the majority of acute care outbreaks (n=14, 40%). Forty-six percent (n=16) of acute care outbreaks were of bacterial etiology (Table 3). Drug resistant organisms such as methicillin-resistant *Staphylococcus aureus* (MRSA), *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, and vancomycin-resistant enterococci (VRE) were responsible for 11 outbreaks (31%) in 2007, with more than half attributed to *Acinetobacter baumannii* (n=7). In 2007, the etiologic agents contributing the largest number of cases in acute care outbreaks were *C. difficile* (n=179, 34%) followed by *A. baumannii* (n =141 or 27%).

Clinics/Provider Offices: Three outbreaks occurred in an outpatient clinic or private provider office associated with an acute care hospital. The ectoparasite *Sarcoptes scabiei* caused one outbreak with the majority of cases (n=9, 33%) (Table 4). Inadequate cleaning and disinfection of a reusable medical device

resulted in two outbreaks (67%) caused by multiple bacterial organisms, such as *Pseudomonas aeruginosa*, *Klebsiella oxytoca* and *Enterobacter cloacae.*

Table 2. Acute Care Hospital Outbreaks by Unit—LAC, 2007

Outbreak Location	No. of Outbreaks
Cardio-thoracic	1
Hematology-Oncology	1
Intensive Care – Adult	5
Intensive Care- Neonatal	2
Medical-Surgical	4
Psychiatric	3
Sub-acute Unit within a Hospital	5
Telemetry	3
Transitional Care	2
Multiple Units	9
Total	**35**

Table 3. Acute Care Hospital Outbreaks by Disease/Condition—LAC, 2007

Disease/Condition/ Etiologic Agent	No. of Outbreaks	No. of Cases
Acinetobacter baumannii	7	141
Aspergillosis	2	7
Clostridium difficile	4	179
MRSA	1	5
Norovirus	2	46
Pseudomonas aeruginosa	2	13
Scabies	14	85
Vancomycin-resistant Enterococci (VRE)	1	28
Unknown Gastroenteritis	2	25
Total	**35**	**529**

Table 4. Clinic And Provider Office Outbreaks by Disease/Condition, LAC, 2007

Disease/Condition/ Etiologic Agent	No. of Outbreaks	No. of Cases
Endophthalmitis	1	4
Scabies	1	9
Multiple bacteria	1	6
Total	**3**	**19**

COMMENTS

Nurses, doctors, respiratory therapists and other members of the health care community believe that patient safety is high priority. However, study after study has demonstrated that this belief does not always transfer to appropriate behavior of health care workers providing direct patient care, as evidenced by an increasing number of health care associated infections (HAIs) worldwide (Cookson, et al., 1999; Mah, et al, 2006). California is among 27 states to enact legislation designed to protect the public by mandating hospitals and related health facilities (e.g., ambulatory surgical centers, dialysis centers) to disclose HAI rates (McGiffert, 2006). In 2006, Senate Bill (SB) 739 was approved, which directs hospitals to evaluate and augment existing infectious disease control programs and implement new standards to prevent HAI. A statewide advisory committee was established on July 1, 2007 to provide guidance to hospitals and ensure compliance with the chosen process measures (central line insertion practices, influenza vaccination of employees and patients, and surgical antimicrobial prophylaxis). Hospitals are mandated to report these measures through the California Department of Public Health and the National Healthcare Safety Network (NHSN) of the Centers for Disease Control and Prevention (CDC) (CDPH, 2007). ACDC is an active participant in the statewide HAI advisory committee and continues to work with state and local providers on implementation and compliance requirements.

Multi-drug resistant Acinetobacter outbreaks reported to LAC increased 600% from 2003 (n=1) as compared to 2007 (n=7). Overall, LAC experienced a 50% increase in outbreaks due to multi-drug

resistant organisms. In 2007, there were 11 multi-drug resistant organism (MDRO) outbreaks reported as compared to eight MDRO outbreaks reported in 2006. *Pseudomonas aeruginosa, Acinetobacter baumannii,* and vancomycin-resistant Enterococci are all pathogens that cause significant morbidity and/or mortality in the immunocompromised hospitalized patient. MDRO outbreaks are not unique to LAC and have increased nationwide, capturing the attention of state legislatures. Recently, MRSA has received a considerable amount of public attention. News media accounts dramatically report the profound and devastating impact an MRSA infection can have on the patient and family (Engel, 2008; PRNewswire, 2008). For the hospitalized patient, acquisition of an MDRO nosocomial infection lengthens hospital days and greatly increases the patient's risk of a negative outcome (Siegel, et al., 2006).

The majority of reported acute care facility outbreaks (n=14) were caused by the ectoparasite *Sarcoptes scabiei*. While rarely the cause of serious morbidity or mortality and usually characterized as a nuisance disease, the economic costs incurred to successfully manage an outbreak can be high (De beer, et al., 2006). *Clostridium difficile* (*C. difficile*) is an organism we continue to see in the LAC hospital population. It was responsible for the greatest number of cases (n=179) reported in hospital outbreaks in 2007. Several outbreaks occurred in an outpatient setting affiliated with an acute care hospital, where a re-usable medical device utilized during a diagnostic or therapeutic procedure was implicated in two of these outbreaks. We determined that staff improper cleaning and disinfection practices contributed to both outbreaks (see 2007 Special Studies Report)

The ACDC Hospital Outreach Unit's Liaison Public Health Nurses (LPHNs) continue to collaborate with partners in the hospital, clinic and other health care settings on the mission to enhance emerging infectious disease preparedness and increase communicable disease and outbreak reporting. Established relationships are maintained with the hospital Infection Preventionist to communicate essential health information that can be disseminated quickly throughout the facility. Among LPHN responsibilities are to make at least an annual visits to their assigned hospital and attend monthly hospital infection control committee (ICC) meetings, if invited. In 2007, the LPHNs conducted 215 hospital visits to update the hospital profile and distribute pandemic influenza, hand washing and related communicable disease education materials. As of end of 2007, the LPHNs are invited to ICC meetings at 14 acute care hospitals.

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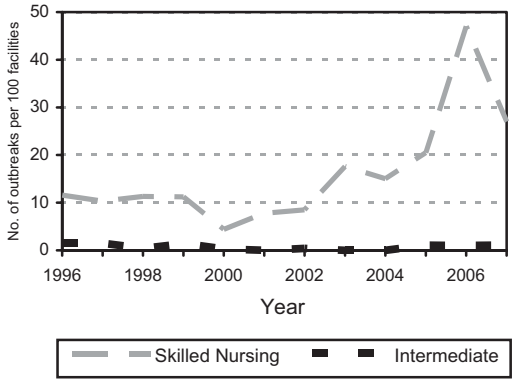
HEALTHCARE - ASSOCIATED OUTBREAKS
SUB-ACUTE CARE FACILITIES

DEFINITION

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

The sub-acute care facilities include skilled nursing facilities, intermediate care facilities and psychiatric care facilities. Skilled nursing facilities provide continuous skilled nursing care to patients on an extended basis. Intermediate care facilities also provide skilled nursing care to patients, but the care is not continuous. Psychiatric facilities provide 24-hour inpatient care for patients with psychiatric needs.

Figure 1
Sub-acute Facility Outbreaks
LAC, 1996–2007



ABSTRACT

- Total confirmed sub-acute care associated outbreaks decreased 34% from 176 outbreaks in 2006 to 116 outbreaks in 2007. This was largely due to a decrease in outbreaks of gastroenteritis.
- In 2007, the number of skilled nursing facility outbreaks decreased 36% from an unusually high outbreak year in 2006 (Table 1). The rate of skilled nursing facility outbreaks decreased from 47 per 100 facilities in 2006 to 27 per 100 facilities in 2007 (Figure 1).
- There was no change in the number of outbreaks in intermediate care facilities from 2006 to 2007. This is the first year in which intermediate care and psychiatric facilities are examined as separate categories in the annual report.

Table 1. Number of Reported Outbreaks in Sub-acute Healthcare Facilities, LAC, 2003–2007

	YEAR				
Type of Facility	**2003**	**2004**	**2005**	**2006**	**2007**
Intermediate Care Facilities	0	0	0	3	3
Psychiatric Care Facilities	-	-	-	-	3
Skilled Nursing Facilities	75	63	76	173	110
Total	75	63	76	176	116

Intermediate Care Facilities: Reported intermediate care facility outbreaks did not change in 2007, with 3 outbreaks in 2007 as compared to 3 in 2006. Scabies accounted for 33% of total cases in 2007 (Table 2).

Table 2. Intermediate Care Facility (ICF) Outbreaks by Disease/Condition—LAC, 2007

Disease/Condition	No. of Outbreaks	No. of Cases
Scabies	1	3
Unknown Rash	1	4
Varicella (Chickenpox)	1	2
Total	3	9

Psychiatric Facilities: In 2007, there were 3 outbreaks in psychiatric facilities, all of which were unspecified gastroenteritis (Table 3).

Table 3. Psychiatric Care Facility Outbreaks by Disease/Condition—LAC, 2007

Disease/Condition	No. of Outbreaks	No. of Cases
Unspecified Gastroenteritis	3	20
Total	3	20

Skilled Nursing Facilities: Reported skilled nursing facility outbreaks decreased by 36% in 2007, with 110 outbreaks in 2007, as compared to 173 outbreaks in 2006. Unspecified gastroenteritis was the most commonly reported outbreak disease, accounting for 49% of outbreaks in 2007 and 74% of cases. Scabies was the second most commonly reported outbreak disease for 2007.

Table 4. Skilled Nursing Facility (SNF) Outbreaks by Disease/Condition—LAC, 2007

Disease/Condition	No. of Outbreaks	No. of Cases
Gastroenteritis • unspecified (n=40) • norovirus (n=14)	54	1100
Scabies	43	181
Unknown Rash	7	98
Respiratory illness • unspecified (n=4) • influenza (n=2)	6	103
Total	110	1482

COMMENTS

Los Angeles County skilled nursing facilities (SNFs) experienced a decrease in the total number of reported gastrointestinal outbreaks, both due to norovirus and unspecified causes, in 2007. SNFs accounted for 117 outbreaks of gastroenteritis involving 2,428 cases in 2006, compared with 54 outbreaks involving 1,100 cases in 2007. Scabies outbreaks and total cases also declined slightly; in 2007, there was a 10% decrease from 48 outbreaks (338 cases) in 2006 to 43 outbreaks (181 cases).

The formation of the Water and Sub-acute Care Unit within the Acute Communicable Disease Control Program (ACDC) in 2007 has permitted focus on working directly with the SNFs in LAC and LAC DPH Community Health Services (CHS) staff to assess communicable disease issues in licensed health facilities, as well as conduct outbreak surveillance of facilities, excluding acute care. In addition, this is the first year in which intermediate care and psychiatric facilities are examined separately in the annual report; previous years had incorporated outbreak information for these and skilled nursing facilities into one healthcare-associated outbreak report. Due to this, some of the trend information for previous years may be skewed by intermediate care and psychiatric care facilities.

In 2007, ACDC initiated a needs assessment of area health officers, area medical directors, nurse managers, public health nurse supervisors and public health nurses (PHNs) in the 24 health districts to determine training needs, assess the interaction with SNFs in their district and identify ways in which ACDC could improve upon the health facilities outbreak investigation data collection form. Respondents reported interest in information targeted to PHNs regarding multi-drug resistant organisms, outbreak investigations and norovirus. Additional information describing the survey results can be found in the 2007 Special Studies Report "Survey of Community Health Services' Interactions with Skilled Nursing Facilities in Los Angeles County".

Based on feedback from the survey, a revised health facilities outbreak investigation data collection form was developed that captures information that was not previously captured on the older form, such as facility census, information on treatment and prophylaxis, number of specimens collected, and laboratory results. The revised form will aid PHNs during outbreak investigations and will capture the most pertinent information for ACDC to use in epidemiological analysis. Currently the form is being piloted in two health districts and a final form for use by all districts is planned by the end of 2008.

The survey also identified multi-drug resistant organisms (MDROs) as a topic on which PHNs and facilities needed additional information. As a result, a summary of recommended infection control guidelines for the prevention and control of multi-drug resistant organisms in long term care (LTC) facilities was updated. These guidelines are specific to LTC facilities in LAC and emphasize the use of standard precautions for all patients, as well as contact precautions, when appropriate. The updated guidelines were distributed to each of the SNFs and ICFs that are licensed in LAC and to CHS staff.

Lastly communication and collaboration with the Health Facilities Inspection Division is continuing to improve. ACDC conducted joint trainings with Health Facilities Inspection Division on infection prevention and communicable diseases to SNF staff.

PREVENTION

The majority of outbreaks in sub-acute care facilities are caused by agents that are spread via person-to-person contact. Thus, appropriate hand hygiene by staff and residents is a crucial infection control measure. It is also important for staff to implement use of isolation precautions when indicated.

RESOURCE

Burwell, L. and MacColl, L. (2007). Survey of community health services' interactions with skilled nursing facilities in Los Angeles County. *Acute Communicable Disease Control Program 2007 Special Studies Report*, 25-28.

Acute Communicable Disease Control
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2007

LOS ANGELES COUNTY
DEPARTMENT OF PUBLIC HEALTH
ACUTE COMMUNICABLE DISEASE CONTROL PROGRAM
2007

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ACUTE COMMUNICABLE DISEASE CONTROL
2007 ANNUAL MORBIDITY REPORT

Disease Summaries Contributors

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- Coccidiodomycosis .....Merle Baron, RN, BSN, PHN
- Cryptosporidiosis..…… ... ……Lauren Burwell, MD
- Encephalitis……..…….....Rachel Civen, MD, MPH
- *Escherichia coli* O157:H7.……... Leticia Martinez, RN, BSN, MPA
- Giardiasis……Patricia Marquez, MPH
- *Haemophilus Influenzae*…… Dulmini Kodagoda, MPH
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- Hepatitis B, Acute (Non-perinatal)…… …Susan Hathaway, RN, BSN, MPH
- Hepatits B, Perinatal…….. ..... Kim Moore, RN, BSN, PHN
- Hepatitis C, Acute…… Susan Hathaway, RN, BSN, MPH
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- Legionellosis .....Juliet Bugante, RN, BSN, PHN
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- Listeriosis, Perinatal…… .....Jennifer Beyer, MPH
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- Meningococcal Disease……... .....Rachel Civen, MD, MPH
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- Pertussis (Whooping Cough)…….. Dulmini Kodagoda, MPH
- Pneumococcal Disease, Invasive……..... Melissa Higdon, MPH
- Salmonellosis……..……Rita Bagby, RN, MSN, PHN
- Shigellosis……Leticia Martinez, RN, BSN, MPA
- Streptococcus, Group A Invasive Disease (IGAS)…… .... ……Melissa Higdon, MPH
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- Typhoid Fever, Carrier……..Leticia Martinez, RN, BSN, MPA
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ACUTE COMMUNICABLE DISEASE CONTROL PROGRAM PUBLICATIONS 2007

Bancroft, E.A. (2007). Antimicrobial resistance: it's not just for hospitals. *Journal of American Medical Association*, 298(15), 1803-1804.

Chaves, S.S., Gargiullo, P., Zhang, J.X., Civen, R., Guris, D., Mascola, L. & Seward, J.F. (2007). Loss of vaccine-induced immunity to varicella over time. *New England Journal of Medicine*, 356(11), 1121-1129.

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Mascola L., Terashita D., Sircar K., et al. (2007). Increased detections and severe neonatal disease associated with Coxsackievirus B1 infection--United States, 2007. *Morbidity and Mortality Weekly Report*, 57(20), 553-556.

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Watson, B., Civen, R., Reynolds, M., Heath, K., Perella, D., Carbajal, T., Mascola, L., Jumaan, A., Zimmerman, L., James, A., Quashi, C. & Schmid, S. (2007). Validity of self-reported varicella disease history in pregnant women attending prenatal clinics. *Public Health Reports*, 22, 499-506.

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Pinner, T., Watts-Troutman, W., Barr, M. Knocking down barriers and evaluating pneumococcal vaccination policy in Los Angeles County comprehensive health centers. Los Angeles County Department of Public Health Nurse Practice Conference and Awards Luncheon, Los Angeles, CA, May 2007.

LYME DISEASE

CRUDE DATA	
Number of Cases	6
Annual Incidence [a]	
LA County [b]	0.06
California	0.2
United States	7.8
Age at Diagnosis	
Mean	48.7
Median	47
Range	15-71

[a] Cases per 100,000 population.
[b] Rates calculated based on less than 19 cases or events are considered unreliable.

DESCRIPTION

Lyme disease (LD) is caused by the spirochete *Borrelia burgdorferi*, which is transmitted to humans by the bite of *Ixodes* ticks; the vector in the Pacific coast states is the western blacklegged tick (*Ixodes pacificus*). This disease is rarely acquired in Los Angeles County (LAC); most reported cases have been acquired in known endemic regions in the United States (US). The most common clinical presentation is a distinctive circular rash called erythema migrans (EM). When EM is not present, other early symptoms such as fever, body aches, headaches, and fatigue are often unrecognized as indicators of LD. If untreated, patients may develop late stage symptoms such as aseptic meningitis, cranial neuritis, cardiac conduction abnormalities 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 antibiotics. Currently, there is no vaccine.

For purposes of surveillance, the Centers for Disease Control and Prevention (CDC) require a confirmed case of LD to have:

- Physician-diagnosed EM that is at least 5 cm in diameter with known tick exposure (laboratory evidence is necessary without tick exposure), or
- At least one late manifestation of LD with supporting laboratory results.

Laboratory criteria for case confirmation include a positive culture for *B. burgdorferi* or demonstration of diagnostic IgM or IgG to *B.* burgdorferi in serum or cerebral spinal fluid. A coalition of several public health and medical organizations recommends a two-step serologic testing procedure for LD: an initial enzyme immunoassay or immunofluorescent antibody screening test, and if positive or equivocal, followed by IgM and IgG Western immunoblotting.[1]

Avoiding tick bite exposure is the primary means of preventing LD. 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 to 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; tucking shirts into pants; using tick repellant; 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 controlling ticks on pets.

2011 TRENDS AND HIGHLIGHTS

- Even as the national incidence increased (from 6.0 per 100,000 in 1999 to 9.9 per 100,000 in 2010), the incidence in LAC (0.06 per 100,000) has remained relatively stable and well below the national and state rates (Figure 1).
- Of the six confirmed cases of LD, all were likely exposed in highly endemic LD regions outside of LAC (Figure 3).
- Three cases (50%) recalled an insect bite prior to onset of EM rash, two of whom reported the insect as a tick.

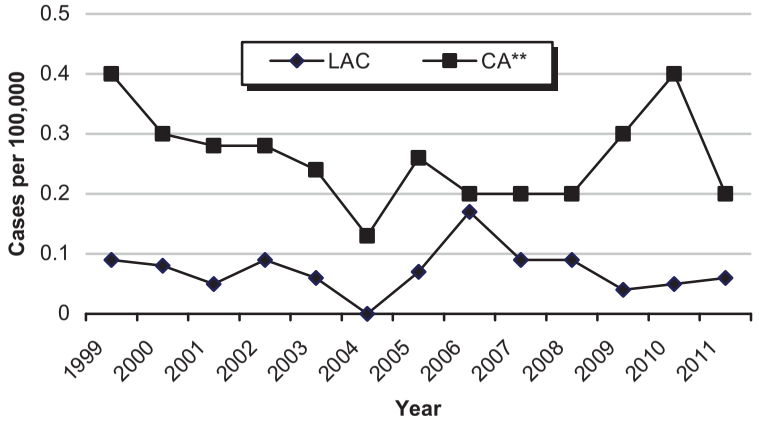
[1] Recommendations for Test Performance and Interpretation from the Second National Conference on Serologic Diagnosis of Lyme Disease. MMWR August 11, 1995/44(31);590-591, http://www.cdc.gov/mmwr/preview/mmwrhtml/00038469.htm.

Reported Lyme Disease Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
Los Angeles County, 2007-2011

	2007 (N=8)			2008 (N=9)			2009 (N=4)			2010 (N=5)			2011 (N=6)		
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
1-4	0	0.0		2	22.2		0	0.0		0	0.0		0	0.0	
5-14	2	25.0		1	11.1		1	25.0		1	20.0		0	0.0	
15-34	3	37.5		1	11.1		0	0.0		2	40.0		1	16.7	
35-44	0	0.0		1	11.1		2	50.0		1	20.0		0	0.0	
45-54	2	25.0		3	33.3		0	0.0		0	0.0		3	50	
55-64	0	0.0		0	0.0		1	25.0		1	20.0		1	16.7	
65+	1	12.5		1	11.1		0	0.0		0	0.0		1	16.7	
Unknown	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
Race/Ethnicity															
Asian	1	12.5		0	0.0		0	0.0		0	0.0		0	0.0	
Black	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
Hispanic	1	12.5		0	0.0		0	0.0		1	20.0		0	0.0	
White	3	37.5		9	100		4	100		4	80.0		6	100	
Other	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
Unknown	3	37.5		0	0.0		0	0.0		0	0.0		0	0.0	
SPA															
1	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
2	3	37.5		2	22.2		1	25.0		0	0.0		2	33.3	
3	1	12.5		0	0.0		0	0.0		0	0.0		1	16.7	
4	2	25.0		1	11.1		0	0.0		2	40.0		0	0.0	
5	2	25.0		4	44.4		1	25.0		2	40.0		3	50.0	
6	0	0.0		0	0.0		1	25.0		1	20.0		0	0.0	
7	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
8	0	0.0		2	22.2		1	25.0		0	0.0		0	0.0	
Unknown	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	

*Rates were not calculated because rates calculated based on less than 19 cases or events are considered unreliable.

Figure 1. Incidence Rates of Lyme Disease LAC* and CA, 1999-2011



*Rates calculated based on less than 19 cases or events are considered unreliable.
**Beginning 2008, CA case count includes probable cases.

Figure 2. Reported Lyme Disease Cases by Month of Onset LAC, 2011

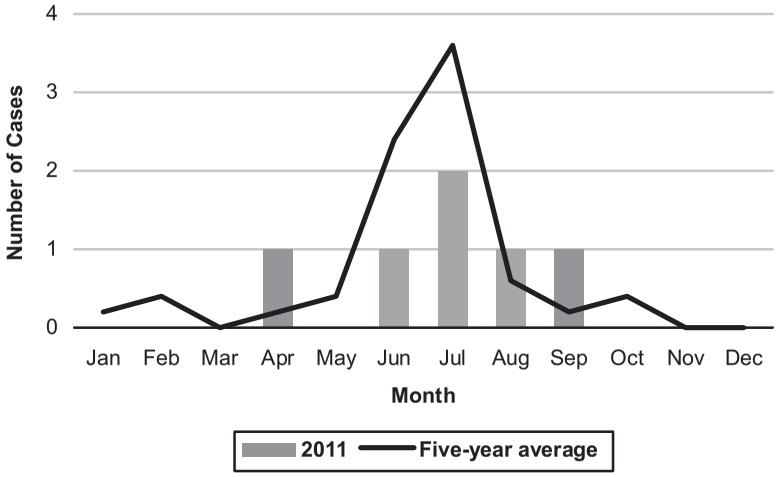
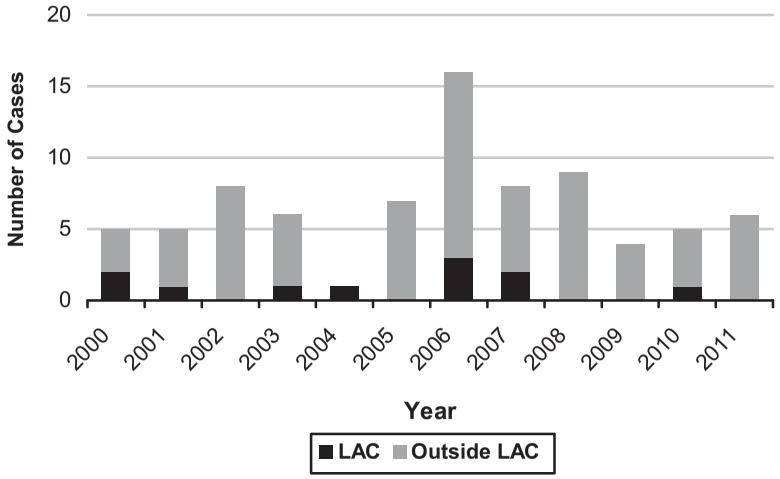


Figure 3. Locations of Tick and Outdoor Exposure in Lyme Disease Cases LAC, 2000-2011



LYME DISEASE

CRUDE DATA	
Number of Cases	5
Annual Incidence [a]	
LA County [b]	0.05
California [c]	--
United States [c]	--
Age at Diagnosis	
Mean	32.6
Median	33
Range	6-56

[a] Cases per 100,000 population.
[b] Rates calculated based on less than 19 cases or events are considered unreliable.
[c] See Final Summary of Nationally Notifiable Infectious Diseases, United States on MMWR website http://www.cdc.gov/mmwr/mmwr_nd/index.html.

DESCRIPTION

Lyme disease (LD) is caused by the spirochete *Borrelia burgdorferi*, which is transmitted to humans by the bite of *Ixodes* ticks; the vector in the Pacific coast states is the western blacklegged tick (*Ixodes pacificus*). This disease is rarely acquired in Los Angeles County (LAC); most reported cases have been acquired in known endemic regions in the United States (US). The most common clinical presentation is a distinctive circular rash called erythema migrans (EM). When EM is not present, other early symptoms such as fever, body aches, headaches, and fatigue are often unrecognized as indicators of LD. If untreated, patients may develop late stage symptoms such as aseptic meningitis, cranial neuritis, cardiac conduction abnormalities 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 antibiotics. Currently, there is no vaccine.

For purposes of surveillance, the Centers for Disease Control and Prevention (CDC) requires a confirmed case of LD to have:

- Physician-diagnosed EM that is at least 5 cm in diameter with known tick exposure (laboratory evidence is necessary without tick exposure), or
- At least one late manifestation of LD with supporting laboratory results.

Laboratory criteria for case confirmation include a positive culture for *B. burgdorferi* or demonstration of diagnostic IgM or IgG to *B.* burgdorferi in serum or cerebral spinal fluid. A coalition of several public health and medical organizations recommends 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[1].

Avoiding tick bite exposure is the primary means of preventing LD. 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 to 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; tucking shirts into pants; using tick repellant; 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 controlling ticks on pets.

2010 TRENDS AND HIGHLIGHTS

- Even as the national incidence increases (from 6.0 per 100,000 in 1999 to 9.9 per 100,000 in 2009), the incidence in LAC (0.05 per 100,000) has remained relatively stable and well below the national rate (Figure 1).
- Of the five confirmed cases of LD, four cases were likely exposed in highly endemic LD regions of the US. One case did not have exposure outside of LAC; this case presented with physician-diagnosed EM.
- Only one case (20%) recalled a tick bite prior to onset of rash.

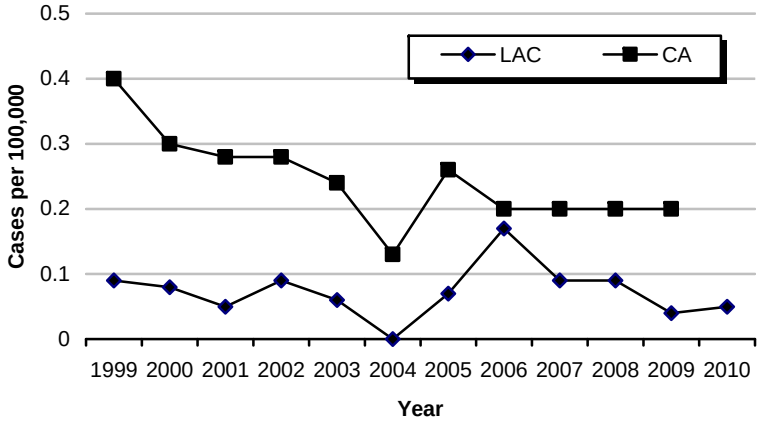
[1] Recommendations for Test Performance and Interpretation from the Second National Conference on Serologic Diagnosis of Lyme Disease. MMWR August 11, 1995/44(31);590-591, http://www.cdc.gov/mmwr/preview/mmwrhtml/00038469.htm.

Reported Lyme Disease Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
Los Angeles County, 2006-2010

	2006 (N=16)			2007 (N=8)			2008 (N=9)			2009 (N=4)			2010 (N=5)		
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
1-4	0	0.0		0	0.0		2	22.2		0	0.0		0	0.0	
5-14	3	18.8		2	25.0		1	11.1		1	0.25		1	0.2	
15-34	7	43.8		3	37.5		1	11.1		0	0.0		2	0.4	
35-44	2	12.5		0	0.0		1	11.1		2	0.50		1	0.2	
45-54	2	12.5		2	25.0		3	33.3		0	0.0		0	0.0	
55-64	1	6.3		0	0.0		0	0.0		1	0.25		1	0.2	
65+	1	6.3		1	12.5		1	11.1		0	0.0		0	0.0	
Unknown	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
Race/Ethnicity															
Asian	1	6.3		1	12.5		0	0.0		0	0.0		0	0.0	
Black	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
Hispanic	2	12.5		1	12.5		0	0.0		0	0.0		1	0.2	
White	11	68.8		3	37.5		9	100.		4	100		4	0.8	
Other	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
Unknown	2	12.5		3	37.5		0	0.0		0	0.0		0	0.0	
SPA															
1	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	
2	6	37.5		2	25.0		2	22.2		1	0.25		0	0.0	
3	0	0.0		1	12.5		0	0.0		0	0.0		0	0.0	
4	5	31.3		2	25.0		1	11.1		0	0.0		2	0.4	
5	2	12.5		2	25.0		4	44.4		1	0.25		2	0.4	
6	0	0.0		0	0.0		0	0.0		1	0.25		1	0.2	
7	0	0.0		1	12.5		0	0.0		0	0.0		0	0.0	
8	3	18.8		0	0.0		2	22.2		1	0.25		0	0.0	
Unknown	0	0.0		0	0.0		0	0.0		0	0.0		0	0.0	

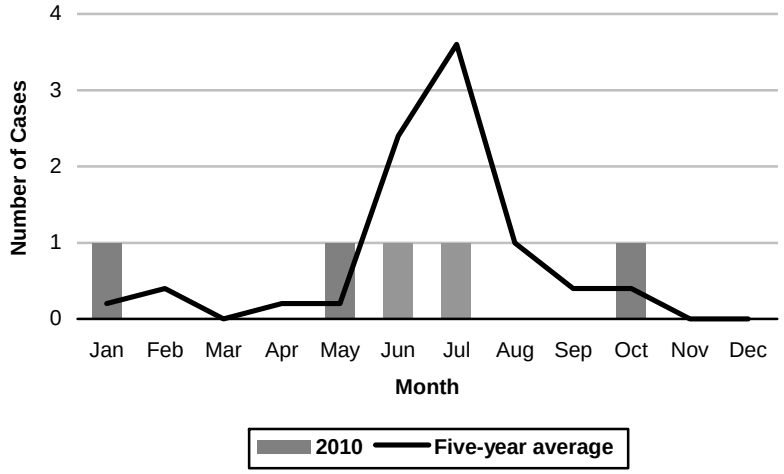
*Rates calculated based on less than 19 cases or events are considered unreliable.

Figure 1. Incidence Rates of Lyme Disease
LAC* and CA, 1999-2010



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

Figure 2. Reported Lyme Disease Cases by Month of Onset
LAC, 2010



LYME DISEASE

CRUDE DATA	
Number of Cases	4
Annual Incidence[a]	
LA County	0.04 [b]
California[c]	0.2
United States[c]	9.6
Age at Diagnosis	
Mean	35.8
Median	38
Range	7-56

[a]Cases per 100,000 population.
[b]Rates calculated based on less than 19 cases or events are considered unreliable.
[c]Calculated from Final 2008 Reports of Nationally Notifiable Infectious Disease. MMWR 58(31);856-857;859-869.

DESCRIPTION

Lyme disease (LD) is caused by a bacterium, *Borrelia burgdorferi*, which is transmitted to humans by the bite of *Ixodes* ticks; the vector in the Pacific coast states is the western blacklegged tick (*Ixodes pacificus*). This disease is rarely acquired in Los Angeles County (LAC), and most reported cases have been acquired outside of LAC from known endemic regions in the United States (US). The most common clinical presentation is a distinctive circular rash called erythema migrans (EM). 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 develop 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 antibiotics. Currently, there is no vaccine.

For purposes of surveillance, the Centers for Disease Control and Prevention (CDC) requires a confirmed case of LD to have documented EM diagnosed by a healthcare provider that is at least 5cm in diameter or at least one late manifestation of LD 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. If indicated, a coalition of several public health and medical organizations recommends 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[1].

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 to 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 controlling ticks on pets.

2009 TRENDS AND HIGHLIGHTS

- Even as the national incidence increases (from 6.3 per 100,000 in 2000 to 9.6 per 100,000 in 2008), the incidence in LAC (0.04 per 100,000) has remained relatively stable and well below the national rate (Figures 1 and 2).
- All cases in 2009 (n=4) reported a travel history to an endemic area outside of LAC.
- One case (25%) recalled a tick bite prior to onset of rash.
- Onset of symptoms continues to be limited to the summer months of June through August (Figure 3).

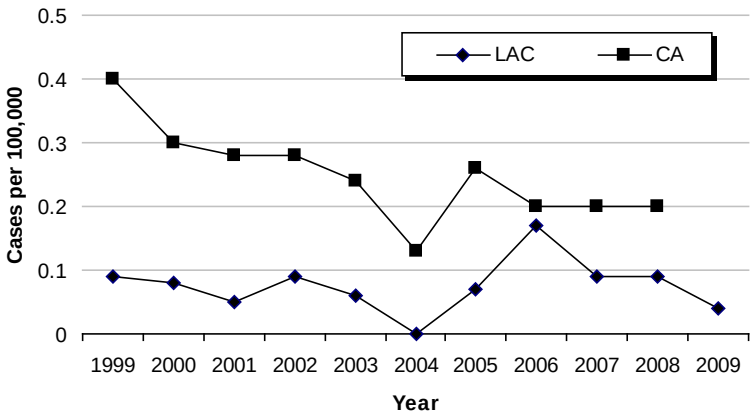
[1]Notice to Readers: Recommendations for Test Performance and Interpretation from the Second National Conference on Serologic Diagnosis of Lyme Disease. MMWR August 11, 1995/44(31);590-591, http://www.cdc.gov/mmwr/preview/mmwrhtml/00038469.htm.

Reported Lyme Disease Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
Los Angeles County, 2005-2009

	2005 (N=7)			2006 (N=17)			2007 (N=8)			2008 (N=9)			2009 (N=4)		
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0	0
1-4	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	2	22.2	0.4	0	0	0
5-14	1	14.3	0.1	3	17.6	0.2	2	25.0	0.1	1	11.1	0.1	1	25.0	0.1
15-34	2	28.6	0.1	7	41.2	0.3	3	37.5	0.1	1	11.1	0.0	0	0	0
35-44	1	14.3	0.1	2	11.8	0.1	0	0.0	0.0	1	11.1	0.1	2	50.0	0.1
45-54	1	14.3	0.1	2	11.8	0.2	2	25.0	0.2	3	33.3	0.2	0	0	0
55-64	1	14.3	0.1	1	5.9	0.1	0	0.0	0.0	0	0.0	0.0	1	25.0	0.1
65+	1	14.3	0.1	1	5.9	0.1	1	12.5	0.1	1	11.1	0.1	0	0	0
Unknown	0	0.0		1	5.9		0	0.0		0	0.0		0	0	
Race / Ethnicity															
Asian	1	14.3	0.1	1	5.9	0.1	1	12.5	0.1	0	0.0	0.0	0	0	0
Black	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0	0
Hispanic	4	57.1	0.1	1	5.9	0.0	1	12.5	0.0	0	0.0	0.0	0	0	0
White	0	0.0	0.0	13	76.5	0.5	3	37.5	0.1	9	100.	0.3	4	100	0.1
Other	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0	0
Unknown	2	28.6		2	11.8		3	37.5		0	0.0		0	0	
SPA															
1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0	0
2	2	28.6	0.1	6	35.3	0.3	2	25.0	0.1	2	22.2	0.1	1	25.0	0.0
3	0	0.0	0.0	0	0.0	0.0	1	12.5	0.1	0	0.0	0.0	0	0	0
4	1	14.3	0.1	5	29.4	0.4	2	25.0	0.2	1	11.1	0.1	0	0	0
5	2	28.6	0.3	2	11.8	0.3	2	25.0	0.3	4	44.4	0.6	1	25.0	0.2
6	0	0.0	0.0	1	5.9	0.1	0	0.0	0.0	0	0.0	0.0	1	25.0	0.1
7	0	0.0	0.0	1	5.9	0.1	1	12.5	0.1	0	0.0	0.0	0	0	0
8	2	28.6	0.2	1	5.9	0.1	0	0.0	0.0	2	22.2	0.2	1	25.0	0.1
Unknown	0	0.0		1	5.9		0	0.0		0	0.0		0	0	

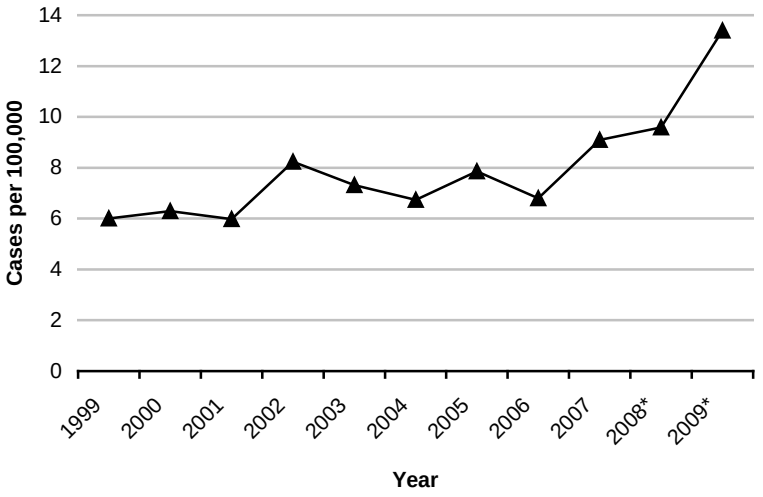
*Rates calculated based on less than 19 cases or events are considered unreliable.

Figure 1. Incidence Rates of Lyme Disease
LAC* and CA, 1999-2009



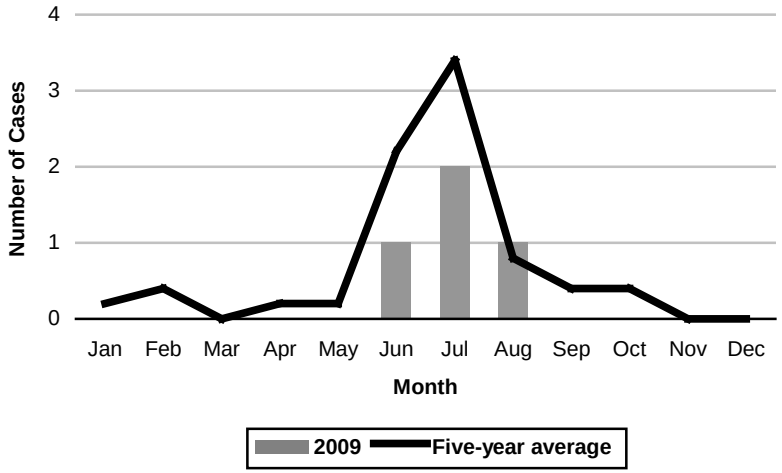
*Rates calculated based on less than 19 cases or events are considered unreliable.

Figure 2. Incidence Rates of Lyme Disease
US, 1999-2009



*Includes probable cases.

Figure 3. Reported Lyme Disease Cases by Month of Onset
LAC, 2009



LYME DISEASE

CRUDE DATA	
Number of Cases	9
Annual Incidence[a]	
LA County	0.09[b]
California[c]	0.20
United States[c]	9.6
Age at Diagnosis	
Mean	31.4
Median	43
Range	3-65

[a]Cases per 100,000 population.
[b]Rates calculated based on less than 19 cases or events are considered unreliable.
[c]Calculated from Final 2008 Reports of Nationally Notifiable Infectious Disease. MMWR 58(31);856-857;859-869.

DESCRIPTION

Lyme disease (LD) is caused by a bacterium, *Borrelia burgdorferi*, which is transmitted to humans by the bite of *Ixodes* ticks; the vector on the Pacific coast states is the western blacklegged tick (*Ixodes pacificus*). This disease is rarely acquired in Los Angeles County (LAC), and most reported cases have been acquired outside of LAC from known endemic regions in the United States (US). The most common clinical presentation is a distinctive circular rash called erythema migrans (EM). 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 develop 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 antibiotics. Currently, there is no vaccine.

For purposes of surveillance, the Centers for Disease Control and Prevention (CDC) requires a confirmed case of LD to have documented EM diagnosed by a healthcare provider that is at least 5cm in diameter or at least one late manifestation of LD 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. If indicated, a coalition of several public health and medical organizations recommends 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[1].

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 controlling ticks on pets.

2008 TRENDS AND HIGHLIGHTS

- Even as the national incidence increases (from 6.3 per 100,000 in 2000 to 9.1 per 100,000 in 2007), the incidence in LAC (0.09 per 100,000) has remained stable and well below the national rate (Figures 1 and 3).
- All cases in 2008 (n=9) reported a travel history to an endemic area outside of LAC.
- Fifty-six percent (n=5) recalled a tick bite prior to onset of rash.
- Onset of symptoms continues to be limited to the summer months of June through August (Figure 2).

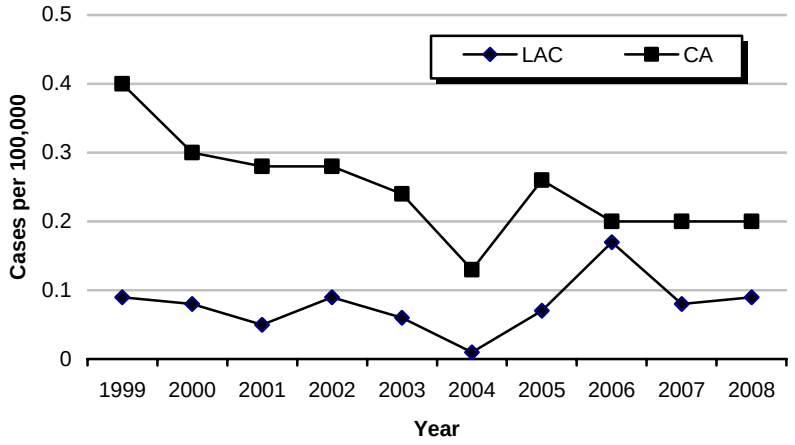
[1]Notice to Readers Recommendations for Test Performance and Interpretation from the Second National Conference on Serologic Diagnosis of Lyme Disease. MMWR August 11, 1995/44(31);590-591, http://www.cdc.gov/mmwr/preview/mmwrhtml/00038469.htm.

Reported Lyme Disease Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
Los Angeles County, 2004-2008

	2004 (N=1)			2005 (N=7)			2006 (N=17)			2007 (N=8)			2008 (N=9)		
	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000	No.	(%)	Rate/ 100,000
Age Group															
<1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
1-4	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	2	22.2	0.4
5-14	0	0.0	0.0	1	14.3	0.1	3	17.6	0.2	2	25.0	0.1	1	11.1	0.1
15-34	1	100.	0.0	2	28.6	0.1	7	41.2	0.3	3	37.5	0.1	1	11.1	0.0
35-44	0	0.0	0.0	1	14.3	0.1	2	11.8	0.1	0	0.0	0.0	1	11.1	0.1
45-54	0	0.0	0.0	1	14.3	0.1	2	11.8	0.2	2	25.0	0.2	3	33.3	0.2
55-64	0	0.0	0.0	1	14.3	0.1	1	5.9	0.1	0	0.0	0.0	0	0.0	0.0
65+	0	0.0	0.0	1	14.3	0.1	1	5.9	0.1	1	12.5	0.1	1	11.1	0.1
Unknown	0	0.0		0	0.0		1	5.9		0	0.0		0	0.0	
Race / Ethnicity															
Asian	0	0.0	0.0	1	14.3	0.1	1	5.9	0.1	1	12.5	0.1	0	0.0	0.0
Black	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
Hispanic	0	0.0	0.0	4	57.1	0.1	1	5.9	0.0	1	12.5	0.0	0	0.0	0.0
White	1	100.	0.0	0	0.0	0.0	13	76.5	0.5	3	37.5	0.1	9	100.	0.3
Other	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
Unknown	0	0.0		2	28.6		2	11.8		3	37.5		0	0.0	
SPA															
1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
2	0	0.0	0.0	2	28.6	0.1	6	35.3	0.3	2	25.0	0.1	2	22.2	0.1
3	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	1	12.5	0.1	0	0.0	0.0
4	0	0.0	0.0	1	14.3	0.1	5	29.4	0.4	2	25.0	0.2	1	11.1	0.1
5	1	100.	0.2	2	28.6	0.3	2	11.8	0.3	2	25.0	0.3	4	44.4	0.6
6	0	0.0	0.0	0	0.0	0.0	1	5.9	0.1	0	0.0	0.0	0	0.0	0.0
7	0	0.0	0.0	0	0.0	0.0	1	5.9	0.1	1	12.5	0.1	0	0.0	0.0
8	0	0.0	0.0	2	28.6	0.2	1	5.9	0.1	0	0.0	0.0	2	22.2	0.2
Unknown	0	0.0		0	0.0		1	5.9		0	0.0		0	0.0	

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

Figure 1. Incidence Rates of Lyme Disease
LAC* and CA, 1999-2008



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

Figure 2. Reported Lyme Disease Cases by Month of Onset
LAC, 2008

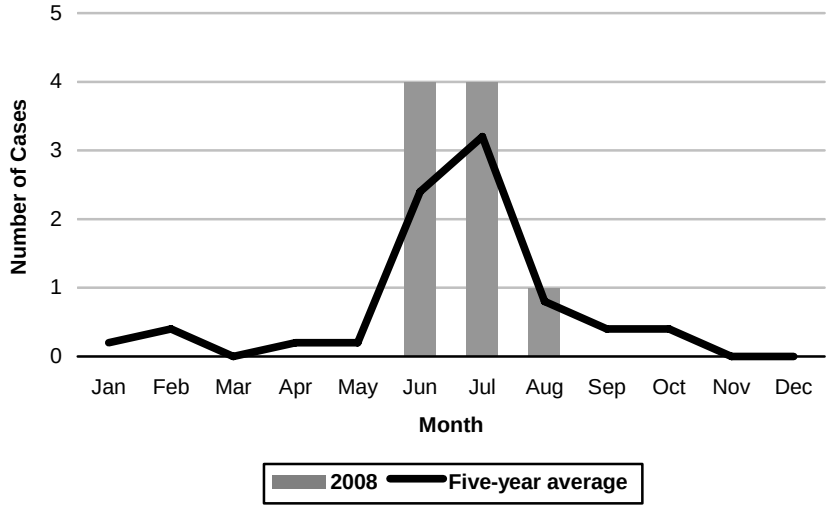
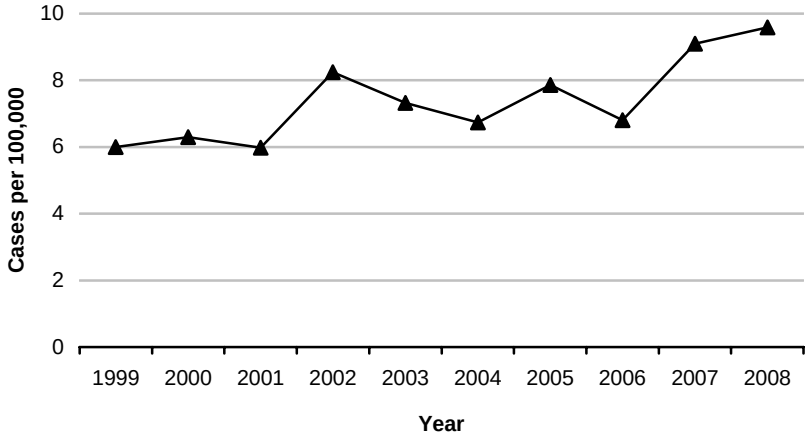


Figure 3. Incidence Rates of Lyme Disease
US, 1999-2008

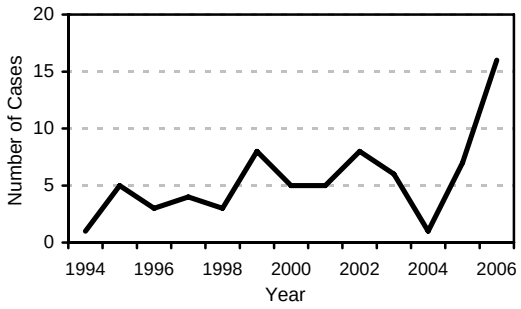


LYME DISEASE

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 1
Lyme Disease Cases
by Year of Onset
LAC, 1994–2006



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.

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.

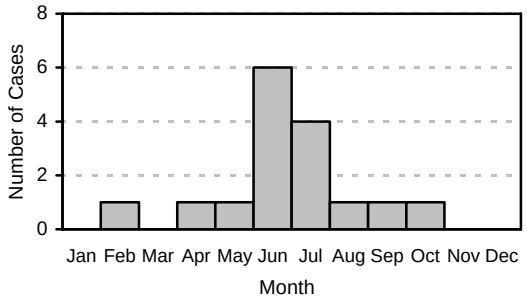
Age: The average age of cases in 2006 was 33, the median was 28.5, and the ages ranged from 8–69 years old. Nationally, LD is most common among persons aged 5–19 years and 30 years and older.

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%).

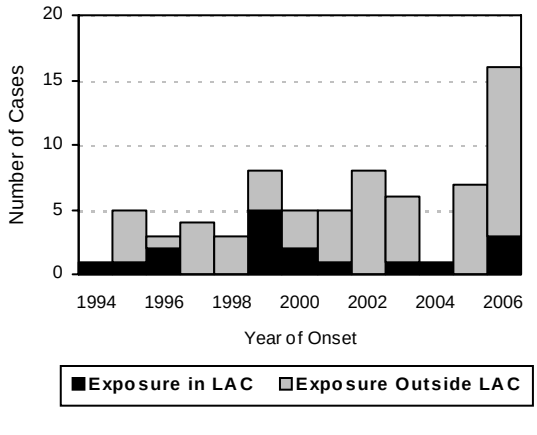
Location: LD does not commonly occur in ticks in LAC, most cases were likely exposed to infected ticks while outside of the county. However, three cases (19%) reported no history of travel outside of LAC within three months of their onset of EM rash (Figure 3). These cases occurred among residents from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

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



*Previous 5-yr average not available.

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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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.
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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

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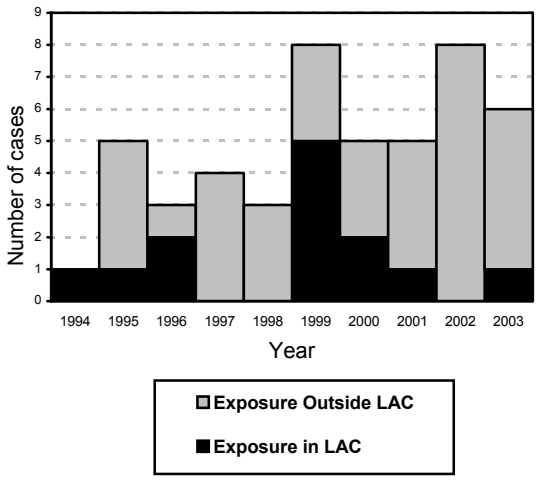
LYME DISEASE

CRUDE DATA	
Number of Cases	6
Annual Incidence[a]	
LA County	---[b]
California	0.25
United States	7.39
Age at Diagnosis	
Mean	37
Median	31
Range	6–55 years
Case Fatality	
LA County	0.0%
United States	N/A

[a] Cases per 100,000 population.
[b] Rates based on fewer than 20 cases are unreliable.

Figure 1
Lyme Disease
Exposure Site by Year 1994–2003



DESCRIPTION

Lyme disease is caused by a bacterium, *Borrelia burgdorferi*, transmitted to humans by the bite of the western blacklegged tick (*Ixodes pacificus*). This disease is not common in LAC. The reservoir is in 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 classic rash is called erythema migrans, an expanding "bull's eye" rash, which is the first sign in about 60–90% of patients (usually at the site of the tick bite.). The incubation period is from 3–32 days. However, early symptoms (e.g., fever, body aches, headaches and fatigue) are often unrecognized and patients may present with later manifestations. These include aseptic meningitis, cranial neuritis, cardiac arrhythmias and arthritis of the large joints. Early disease is treated with a short course of oral antibiotics, while later manifestations may require longer treatment with oral or intravenous (IV) antibiotics. Currently, there is no vaccine.

The diagnosis of Lyme disease may be difficult because other diseases can cause early symptoms of fever, body aches, headaches, and fatigue. Laboratory tests are available, but they are often not sensitive, specific or consistent.

Lyme disease may be cured by early diagnosis and treatment with antibiotics. Untreated disease causing long-term illness and complications may occur, requiring longer treatment with oral or IV antibiotics.

DISEASE ABSTRACT

- In 2003, 6 reported cases met CDC surveillance criteria. Four were male and two were female.
- All cases except 1 reported exposure outside LAC. The reported one LAC Lyme case noted tick exposure in Malibu.

COMMENTS

Lyme disease is now the most frequently reported vectorborne disease in the US; however, it is reported infrequently in LAC. Since Lyme disease became reportable in 1989, 48 reported cases have met the CDC surveillance criteria. Sixteen cases (28%) were exposed to ticks inside LAC. Although transmission of Lyme disease does occur in LAC, it is believed to be rare because the western blacklegged tick is not the most common tick in LAC, and only 1–2% of western blacklegged ticks in California are infected with the bacterium that causes Lyme disease. The tick must be attached for a minimum of 48 hours for transmission to occur. Although DHS has been testing ticks and reservoir animals for the past eleven years, 1999 was the first year for which ticks were confirmed to carry *B. burgdorferi* by culture.

When a case of Lyme disease is reported to the DHS, an investigation is initiated by ACDC, which includes collection of information from the physician and the patient. Vector Management staff determine the probable site of tick exposure and initiate field studies. Field studies include collection of ticks and samples from animals to test for Lyme disease.

Although Lyme disease occurs rarely in LAC, personal protective measures are recommended to prevent tick bites. These include: using insect repellents containing DEET, wearing long pants and long-sleeved clothing, wearing light-colored clothing (so that ticks can be spotted more easily) and walking in the center of a trail to avoid overhanging grass or brush.

Future Directions

The vaccine made by SmithKline Beecham (LYMErix) was taken off the market in 2001 due to poor sales and possible side effects and complications. Efforts are being made to develop a new vaccine.

ADDITIONAL RESOURCES

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

A brochure regarding Lyme disease is from the California DHS is avilable at:
www.dhs.ca.gov/ps/dcdc/disb/pdf/Lyme%20Disease%20brochure%20final.pdf

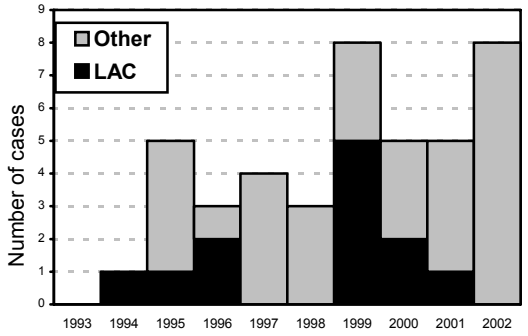
Publications:

- Nadelman RB and Wormser GP. Lyme borreliosis. Lancet. 1998; 352: 557–65.
- Barbour AG. Lyme Disease: The Cause, the Cure, the Controversy. 1996. The Johns Hopkins University Press, Baltimore, MD.
- Steere AC. Lyme disease. N Engl J Med. 2001; 345(2): 115–125.
- Sood SK. Lyme disease. Pediatr Infect Dis J. 1999; 18: 913–25.

LYME DISEASE

CRUDE DATA	
Number of Cases	8
Annual Incidence[a]	
LA County	---[b]
California	0.3
United States	8.4
Age at Diagnosis	
Mean	37
Median	36
Range	7–67 years
Case Fatality	
LA County	0.0%
United States	N/A

Figure 1
Lyme Disease
Exposure Site by Year
LAC and US, 1993–2002



[a] Cases per 100,000 population.
[b] Rates based on fewer than 20 cases are unreliable.

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DISEASE ABSTRACT

- In 2002, 8 reported cases met CDC surveillance criteria. Most (n=6, 75%) were female.
- All cases reported exposure outside LAC.

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