ACUTE COMMUNICABLE DISEASE CONTROL

ANNUAL MORBIDITY REPORT AND SPECIAL STUDIES REPORT 2001



County of Los Angeles DEPARTMENT OF HEALTH SERVICES



2001 ACUTE COMMUNICABLE DISEASE MORBIDITY REPORT

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PURPOSE

The Acute Communicable Disease Control **Annual Morbidity Report** of Los Angeles County's Department of Health Services, Public Health is compiled to:

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

Note: The 2001 ACDC Annual Morbidity Report does **not** include information regarding the following diseases: tuberculosis, sexually transmitted diseases, or adult HIV. Information regarding these diseases is available from their respective departments.

LAC DEMOGRAPHIC DATA

Population figures from the Census 2000 were used for both the 2000 and the 2001 data because estimates of the 2001 population were unavailable at the time of printing; Figures used for calculating the 1991-1999 disease rates in this report were derived from the Regional Population Model (RPM) file developed by the County of Los Angeles, Chief Administrative Office, Urban Research Division for the Population Estimation and Projection System Consortium. These population estimates were projected from 1990 MARS file (Modified Age, Race, and Sex) produced by the US Census Bureau and modified by local death rates, migration rates, and fertility rates within age, sex and racial/ethnic groups. Estimates for 1991-1998 were recently revised and are different than in the previous annual reports. Note also that the population estimates for the end of the 1990s were larger than the population in 2000 as determined by the 2000 census.

Long Beach and Pasadena are separate reporting jurisdictions recognized by California Department of Health Services and maintain their own disease reporting systems. Therefore, disease episodes occurring among residents of these two cities have been excluded from county morbidity data, and their populations subtracted from county population data. Exceptions to this rule are noted in the text when they occur.

National and California state counts of reportable diseases were obtained from the Centers for Disease Control and Prevention (CDC), Final 2001 Reports of Notifiable Diseases, *Morbidity and Mortality Weekly Report* 2002/51(32);710. The *MMWR* report also includes Bureau of the Census 2001 population estimates for the United States and the State of California; those figures were used to calculate national and California rates of disease. According to that report, the population of the US in 2001 was 284,796,000, and that of California was 34,501,000

Population estimates for Los Angeles County (not including Pasadena and Long Beach) used in this report are listed in Table A for 2001 as well as for the previous five years. Population data also are given by age, sex, race and health district for 2001 (Tables B-E). Additional disease cases identified after publication of prior annual reports are included in summary tables. Thus, for overall case totals and disease rates from prior years, the current data are

considered more accurate than those in prior annual reports.

Table A. Los Angeles County^aPopulation by Year, 1996-2001

Year	Population
1996	8,870,468
1997	8,970,692
1998	9,069,516
1999	9,171,507
2000	8,920,107
2001	8,920,107

a Does not include cities of Pasadena and Long Beach.

Table B.Los Angeles CountyaPopulation by Age Group, 2001

Age Group in Years	Population
	133 736
1-4	555 869
5-14	1,430,786
15-34	2,775,914
35-44	1,422,494
45-54	1,077,872
55-64	655,476
65+	867,960
Total	8,920,107

^a Does not include cities of Pasadena and Long Beach.

Table D. Los Angeles County^a Population by Race, 2001

Race	Population
Asian Black Hispanic White Other ^b	1,073,615 815,584 4,031,883 2,752,132 23,507
Total	8,920,107 ^c

a Does not include cities of Pasadena and Long Beach.

b Other includes only American Indian, Alaskan Native, Eskimo and Aleut.

^C Total does not include two or more races.

Table C. Los Angeles County^a Population by Sex, 2001

Sex	Population
Male	4,410,029
Female	4,510,078
Total	8,920,107

^aDoes not include cities of Pasadena and Long Beach.

Health District	Population
Alhambra	337,432
Antelope Valley	318,607
Bellflower	347,236
Central	331,894
Compton	273,360
East Los Angeles	202,121
East Valley	421,682
El Monte	437,113
Foothill	294,292
Glendale	334,152
Harbor Hollywood-Wilshire	195,632
Hollywood-Wilshire	502,310
Inglewood	405,766
Northeast	309,879
Pomona	529,997
San Antonio	423,862
San Fernando	392,875
South	165,096
Southeast	156,616
Southwest	359,982
Torrance	434,976
West	613,191
West Valley	820,045
Whittier	311,991
Total	8,920,107

Table E. Los Angeles County Populationaby Health District, 2001

^aPasadena and Long Beach are separate public health jurisdictions and are excluded from this table.

DATA SOURCES

Data on occurrence of communicable diseases in LAC were obtained through passive and sometimes active surveillance. Every health-care provider or administrator of a health facility or clinic, and anyone in charge of a public or private school, kindergarten, boarding school, or preschool knowing of a <u>case or suspected case</u> of a communicable disease is required to report it to the local health department as specified by the California Code of Regulations (Section 2500). Immediate reporting by telephone is also required for any <u>outbreak</u> or <u>unusual</u> <u>incidence</u> of infectious disease and any <u>unusual disease</u> not listed in Section 2500. Laboratories have separate requirements for reporting certain communicable diseases (Section 2505). Health-care providers must also give detailed instructions to household members in regard to precautionary measures to be taken for preventing the spread of disease (Section 2514).

- 1. Passive surveillance relies on physicians, laboratories, and other health-care providers to report diseases of their own accord to the Department of Health Services (DHS) using the Confidential Morbidity Report (CMR) form, electronically, by telephone, or by facsimile.
- Active surveillance entails ACDC staff regularly contacting hospitals, laboratories and physicians in an effort to identify all cases of a given disease. In 2001, ACDC did active surveillance for pediatric cases of acquired immunodeficiency syndrome. In addition, ACDC staff contacted schools, hospitals, nursing homes, student health centers and sentinel physicians to collect reports of vaccine-preventable diseases and to investigate outbreaks.

DATA LIMITATIONS

This report should be interpreted in light of the following notable limitations:

1. <u>Underreporting</u>.

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

2. <u>Reliability of Rates</u>.

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

To account for these instabilities, all rates in the ACDC Annual Morbidity Report based on less than 19 events are considered "unreliable." This translates into a relative standard error of the rate of 23% or more, which is the cut-off for rate reliability used by the National Center for Health Statistics. Therefore, rates based on less than 19 events will not be reported because their standard errors and reliability cannot be determined. Readers may calculate the rates on their own using standard population tables. In the Annual Morbidity Report, rates of disease for groups (e.g., Latino versus non-Latino) are said to differ significantly only when two criteria are met: 1) group rates are reliable and 2) the 95% confidence limits for these rates do not overlap. Confidence limits are calculated only those rates which are reliable.

3. <u>Case Fatality Rates</u>.

Some deaths from communicable diseases may not appear on LAC's Vital Records computer files. Deaths are filed with only underlying cause of death indicated. Any contributing or otherwise significant conditions, including communicable diseases, are not indicated in the computer record. Also, case-fatality percent is based on deaths that occurred in 2001 regardless of year of disease onset; therefore, fatality data should be interpreted with caution.

4. <u>Case Definitions</u>.

To standardize surveillance, "Case Definitions for Infectious Conditions under Public Health Surveillance," *MMWR* 1997;46(RR-10):1-57 is used with some exceptions as noted in the text of the individual diseases. Since verification by a laboratory test is required for the diagnosis of some diseases, cases reported without such verification may not be true cases. Therefore, an association between a communicable disease and a death or an outbreak possibly may not be identified.

5. <u>Onset date versus report date</u>.

Some cases of disease occurring in 2001 were not reported until after this annual report was completed. Slight differences in the number of cases and rates of disease for 2001 may be observed in subsequent annual reports. Any such disparities are likely to be small.

6. <u>Population Estimates</u>.

Estimates of the LAC population are subject to many errors. Population data for 1991 through 1999 were derived from the 1990 census using a sophisticated estimation model developed in 1999. These independent population estimates facilitate trend analysis. The 2000 census data was used to calculate disease rates in 2000 and 2001 since the population estimates for 2001 were not available at the time of printing. Furthermore, the population of LAC is in constant flux. Though not accounted for in census data, visitors and other non-residents may have an effect on disease occurrences.

7. <u>Place of acquisition of infections</u>.

Some cases of diseases reported in LAC may have been acquired outside of the county. This may be especially true for many of the diseases common in Latino and Asian populations. Therefore, some disease rates more accurately reflect the place of diagnosis than the location where an infection was acquired. Malaria is a good example of such a disease since no known locally acquired cases of malaria occurred in 2001.

8. <u>Health Districts and Service Planning Areas</u>.

In 1994, the following health district boundaries changed: Central, Compton, Glendale, Inglewood, Northeast, San Fernando, West, and Torrance. San Fernando Health District was split into Antelope Valley and San Fernando Health Districts. In 1999, the 24 individual health districts were grouped into eight Service Planning Areas (SPA): SPA 1, Antelope Valley; SPA 2, San Fernando Valley; SPA 3, San Gabriel; SPA 4, Metro; SPA 5, West; SPA 6, South; SPA 7, East; and SPA 8, South Bay.

9. <u>Race/Ethnicity category changes</u>.

The five major racial/ethnic categories and their definitions as used in this report are as follows:

- Asian person having origins in any of the original peoples of the Far East, Southeast Asia, the Indian subcontinent, or the Pacific Islands.
- American Indian person having origins in any of the original peoples of North America and who maintain cultural identification through tribal affiliation or community recognition.
- Black person having origins in any of the black racial groups of Africa.
- Latino person of Mexican, Puerto Rican, Cuban, Central or South American, or other Spanish culture or origin, regardless of race.
- White person having origins in any of the original peoples of Europe, North Africa, or the Middle East.

STANDARD REPORT FORMAT

- 1. <u>Crude Data</u>.
 - **Number of Cases**: For most diseases, this number reflects new cases of the disease with an onset in 2001. If the onset was unknown, the date of diagnosis was used.
 - Annual Incidence Rates in LAC: Number of new cases in 2001 divided by 2000 county census population (minus Long Beach and Pasadena) multiplied by 100,000.
 - Annual Incidence Rates in the US and California: 2001 incidence rates for the US and California were taken from the previously cited *Morbidity and Mortality Weekly Report*. The *MMWR* records diseases by date of report rather than date of onset.
 - Mean Age at Onset: Arithmetic average age of all cases.
 - Median Age at Onset: The age that represents the midpoint of the sequence of all case ages.
 - **Range of Ages at Onset**: Ages of the youngest and oldest cases in 2001. For cases under one year of age, less than one (<1) was used.
 - **Case Fatality**: Number of deaths in 2001 due to disease (when data were available) divided by the number of new cases of the disease in 2001, expressed as a percentage. Note that deaths may be due to infections acquired prior to 2001.
- 2. <u>Etiology</u>.

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

3. <u>Disease Abstract</u>.

This provides a synopsis or the highlights of disease activity in 2001.

- 4. <u>Stratified Data</u>.
 - **Trends**: Any trends in case characteristics during recent years.
 - Seasonality: Number of cases that occurred during each month of 2001.
 - Age: Annual rate of disease for individual age groups. Race-adjusted rates are presented for some diseases.
 - Sex: Male-to-female rate ratio of cases.
 - **Race/Ethnicity**: Annual rate of disease for the five major racial groups. Cases of unknown race are excluded; thus, race-specific rates may be underestimates.

Age-adjusted rates are presented for some diseases.

• Location: Location presented most often is the health district or SPA of residence of cases. Note that "location" rarely refers to the site of disease acquisition. Age-adjusted rates by location are presented for some diseases.

5. <u>Comments</u>.

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

- 6. <u>Prevention</u>. Includes a description of county programs and other measures that address the disease.
- 7. <u>Additional Resources</u>. Provides agencies, phone numbers, websites, and other resources on the subject.

CHANGES IN DISEASE INCIDENCE

Incidence rates for several diseases monitored by Acute Communicable Disease Control in 2001 were markedly different from those in 2000. The percent change in incidence during 2001 compared to 2000 is presented in Table F for those diseases where at least 15 cases were reported in either 2000 or 2001, and substantial change was observed. Reasons for these changes are discussed in the individual disease reports.

Disease	2000	2001	Percent
DISEase	2000	2001	Change (76)
Meningitis, viral	2.95	4.24	43.73
Listeriosis, nonperinatal	0.21	0.30	42.86
Legionellosis	0.16	0.20	25.00
Amebiasis	1.22	1.56	27.87
Kawasaki syndrome	0.39	0.27	-30.77
Hepatitis B	0.73	0.49	-32.88
Hepatitis A	9.41	6.08	-35.39
Mumps	0.33	0.19	-42.42
Typhus	0.19	0.09	-52.63

Table F. Percent Change in Incidence of Selected Notifiable Communicable Diseases, Los Angeles County, 2001

LIST OF ACRONYMS

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

95%CI	95 percent confidence interval	Hib	Haemophilus influenzae, type b
ACDC	Acute Communicable Disease Control	HIV	Human immunodeficiency virus
AIDS	Acquired immunodeficiency syndrome	lgG	Immunoglobulin G
AR	Attack rate	lgM	Immunoglobulin M
CDC	Centers for Disease Control and	LAC	Los Angeles County
CDHS	Prevention California Dept. of Health Services	MMR	Mumps-Measles-Rubella vaccine
CMR	Confidential morbidity report	MMWR	Morbidity & Mortality Weekly Report
CSF	Cerebral spinal fluid	N/A	Not available
DHS	Department of Health Services	NLV	Norwalk-like virus
DTaP	Diphtheria-tetanus-acellular pertussis	OR	Odds ratio
DTP	Diphtheria-tetanus-pertussis vaccine	РСР	Pneumocystis carinii pneumonia
EHS	Environmental Health Services	PHBPP	Perinatal Hepatitis B Prevention Prgm.
GI and GE	gastrointestinal and gastroenteritis	RR	Rate ratio or Relative risk
HAV	Hepatitis A virus	SNF	Skilled nursing facility
HBIG	Hepatitis B Immunoglobulin	sp. or spp.	Species
HBsAg	Hepatitis B surface antigen	SPA	Service Planning Area
HBV	Hepatitis B virus	US	United States
HCV	Hepatitis C virus	VCMR	Visual confidential morbidity report
HD	Health District		

Health Districts

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



Year of Onset5-yearupper biseaseDisease199619971998199920002001AverageLimitaAmebiasis214149157134109139153221Botulism43130225Brucellosis1262349512Campylobacteriosis17331535121710891273114113691826Cholera00300013Cocidioidomycosis7650534858685777Cryptosporidiosis143859371687792145Dengue02533536E. coli O157:H7 ^b 1820221227312029Encephalitis3442483940483354Giardiasis9567866725925094467031009Haemophilus influenzae type b3107016412Hansen's Disease (Leprosy)9181310921219Hepatitis A13641582940112083954211691704Hepatitis B24810992666544116					Previous	5-Yr 95%			
Disease199619971998199920002001AverageLimitAmebiasis214149157134109139153221Botulism43130225Brucellosis1262349512Campylobacteriosis17331535121710891273114113691826Cholera00300013Coccidioidomycosis7650534858685777Cryptosporidiosis143859371687792145Dengue02533536E. coli O157:H7 ^b 1820221227312029Encephalitis3442483949414253Foodborne outbreaks1240343940483354Giardiasis9567866725925094467031009Haemophilus influenzae type b3107016412Hansen's Disease (Leprosy)9181310921219Hepatitis A13641582940112083954211691704Hepatitis B24810992666544	-	4000	4007	Yea	r of Onset	2000	2004	5-year	upper
Amebiasis214149157134109139153221Botulism43130225Brucellosis1262349512Campylobacteriosis17331535121710891273114113691826Cholera00300013Coccidioidomycosis7650534858685777Cryptosporidiosis143859371687792145Cysticercosis3434242843373345Dengue02533536E. coli O157:H7 ^b 1820221227312029Encephalitis3442483949414253Foodborne outbreaks1240343940483354Giardiasis9567866725925094467031009Haemophilus influenzae type b3107016412Hansen's Disease (Leprosy)9181310921219Hepatitis A13641582940112083954211691704Hepatitis B24810992666544116 </th <th>Disease</th> <th>1990</th> <th>1997</th> <th>1998</th> <th>1999</th> <th>2000</th> <th>2001</th> <th>Average</th> <th>Limit</th>	Disease	1990	1997	1998	1999	2000	2001	Average	Limit
Botulism43130225Brucellosis1262349512Campylobacteriosis17331535121710891273114113691826Cholera00300013Coccidioidomycosis7650534858685777Cryptosporidiosis143859371687792145Cysticercosis3434242843373345Dengue02533536E. coli O157:H7b1820221227312029Encephalitis3442483949414253Foodborne outbreaks1240343940483354Giardiasis9567866725925094467031009Haemophilus influenzae type b3107016412Hansen's Disease (Leprosy)9181310921219Hepatitis B24810992666544116249	Amebiasis	214	149	157	134	109	139	153	221
Brucellosis1262349512Campylobacteriosis17331535121710891273114113691826Cholera00300013Coccidioidomycosis7650534858685777Cryptosporidiosis143859371687792145Cysticercosis3434242843373345Dengue02533536E. coli O157:H7b1820221227312029Encephalitis3442483949414253Foodborne outbreaks1240343940483354Giardiasis9567866725925094467031009Hasen's Disease (Leprosy)9181310921219Hepatitis A13641582940112083954211691704Hepatitis B24810992666544116249	Botulism	4	3	1	3	0	2	2	5
$\begin{array}{c cccc} Campylobacteriosis & 1733 & 1535 & 1217 & 1089 & 1273 & 1141 & 1369 & 1826 \\ Cholera & 0 & 0 & 3 & 0 & 0 & 0 & 1 & 3 \\ Coccidioidomycosis & 76 & 50 & 53 & 48 & 58 & 68 & 57 & 77 \\ Cryptosporidiosis & 143 & 85 & 93 & 71 & 68 & 77 & 92 & 145 \\ Cysticercosis & 34 & 34 & 24 & 28 & 43 & 37 & 33 & 45 \\ Dengue & 0 & 2 & 5 & 3 & 3 & 5 & 3 & 6 \\ E. \ coli \ O157:H7^b & 18 & 20 & 22 & 12 & 27 & 31 & 20 & 29 \\ Encephalitis & 34 & 42 & 48 & 39 & 49 & 41 & 42 & 53 \\ Foodborne \ outbreaks & 12 & 40 & 34 & 39 & 40 & 48 & 33 & 54 \\ Giardiasis & 956 & 786 & 672 & 592 & 509 & 446 & 703 & 1009 \\ Haemophilus \ influenzae \ type \ b & 3 & 10 & 7 & 0 & 1 & 6 & 4 & 12 \\ Hansen's \ Disease \ (Leprosy) & 9 & 18 & 13 & 10 & 9 & 2 & 12 & 19 \\ Hepatitis \ A & 1364 & 1582 & 940 & 1120 & 839 & 542 & 1169 & 1704 \\ Hepatitis \ B & 248 & 109 & 92 & 66 & 65 & 44 & 116 & 249 \end{array}$	Brucellosis	12	6	2	3	4	9	5	12
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Campylobacteriosis	1733	1535	1217	1089	1273	1141	1369	1826
$\begin{array}{c cccc} Coccidioidomycosis & 76 & 50 & 53 & 48 & 58 & 68 & 57 & 77 \\ Cryptosporidiosis & 143 & 85 & 93 & 71 & 68 & 77 & 92 & 145 \\ Cysticercosis & 34 & 34 & 24 & 28 & 43 & 37 & 33 & 45 \\ Dengue & 0 & 2 & 5 & 3 & 3 & 5 & 3 & 6 \\ E. {\it coli} O157:H7^{\rm b} & 18 & 20 & 22 & 12 & 27 & 31 & 20 & 29 \\ Encephalitis & 34 & 42 & 48 & 39 & 49 & 41 & 42 & 53 \\ Foodborne outbreaks & 12 & 40 & 34 & 39 & 40 & 48 & 33 & 54 \\ Giardiasis & 956 & 786 & 672 & 592 & 509 & 446 & 703 & 1009 \\ {\it Haemophilus influenzae type b} & 3 & 10 & 7 & 0 & 1 & 6 & 4 & 12 \\ Hansen's Disease (Leprosy) & 9 & 18 & 13 & 10 & 9 & 2 & 12 & 19 \\ Hepatitis A & 1364 & 1582 & 940 & 1120 & 839 & 542 & 1169 & 1704 \\ Hepatitis B & 248 & 109 & 92 & 66 & 65 & 44 & 116 & 249 \end{array}$	Cholera	0	0	3	0	0	0	1	3
Cryptosporidiosis143859371687792145Cysticercosis3434242843373345Dengue02533536E. coli O157:H7b1820221227312029Encephalitis3442483949414253Foodborne outbreaks1240343940483354Giardiasis9567866725925094467031009Haemophilus influenzae type b3107016412Hansen's Disease (Leprosy)9181310921219Hepatitis B24810992666544116249	Coccidioidomycosis	76	50	53	48	58	68	57	77
Cysticercosis 34 34 24 28 43 37 33 45 Dengue02533536E. coli O157:H7b1820221227312029Encephalitis3442483949414253Foodborne outbreaks1240343940483354Giardiasis9567866725925094467031009Haemophilus influenzae type b3107016412Hansen's Disease (Leprosy)9181310921219Hepatitis A13641582940112083954211691704Hepatitis B24810992666544116249	Cryptosporidiosis	143	85	93	71	68	77	92	145
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Cysticercosis	34	34	24	28	43	37	33	45
E. coli O157:H7b1820221227312029Encephalitis3442483949414253Foodborne outbreaks1240343940483354Giardiasis9567866725925094467031009Haemophilus influenzae type b3107016412Hansen's Disease (Leprosy)9181310921219Hepatitis A13641582940112083954211691704Hepatitis B24810992666544116249	Dengue	0	2	5	3	3	5	3	6
Encephalitis3442483949414253Foodborne outbreaks1240343940483354Giardiasis9567866725925094467031009Haemophilus influenzae type b3107016412Hansen's Disease (Leprosy)9181310921219Hepatitis A13641582940112083954211691704Hepatitis B24810992666544116249	E. coli O157:H7 ^b	18	20	22	12	27	31	20	29
Foodborne outbreaks1240343940483354Giardiasis9567866725925094467031009Haemophilus influenzae type b3107016412Hansen's Disease (Leprosy)9181310921219Hepatitis A13641582940112083954211691704Hepatitis B24810992666544116249	Encephalitis	34	42	48	39	49	41	42	53
Giardiasis9567866725925094467031009Haemophilus influenzae type b3107016412Hansen's Disease (Leprosy)9181310921219Hepatitis A13641582940112083954211691704Hepatitis B24810992666544116249	Foodborne outbreaks	12	40	34	39	40	48	33	54
Haemophilus influenzae type b3107016412Hansen's Disease (Leprosy)9181310921219Hepatitis A13641582940112083954211691704Hepatitis B24810992666544116249	Giardiasis	956	786	672	592	509	446	703	1009
Hansen's Disease (Leprosy)9181310921219Hepatitis A13641582940112083954211691704Hepatitis B24810992666544116249	Haemophilus influenzae type b	3	10	7	0	1	6	4	12
Hepatitis A 1364 1582 940 1120 839 542 1169 1704 Hepatitis B 248 109 92 66 65 44 116 249	Hansen's Disease (Leprosy)	9	18	13	10	9	2	12	19
Hepatitis B 248 109 92 66 65 44 116 249	Hepatitis A	1364	1582	940	1120	839	542	1169	1704
	Hepatitis B	248	109	92	66	65	44	116	249
Hepatitis C $10 23 12 21 10 11 15 26$	Hepatitis C	10	23	12	21	10	1	15	26
Hepatitis unspecified 28 17 13 9 11 1 16 29	Hepatitis unspecified	28	17	13	9	11	1	16	29
Kawasaki syndrome 20 26 33 29 35 24 29 39	Kawasaki syndrome	20	26	33	29	35	24	29	39
Legionellosis 12 32 20 16 14 18 19 33	Legionellosis	12	32	20	16	14	18	19	33
Listeriosis, nonperinatal 30 18 24 21 19 27 22 31	Listeriosis, nonperinatal	30	18	24	21	19	27	22	31
Listeriosis, perinatal 5 8 7 12 8 3 8 12	Listeriosis, perinatal	5	8	7	12	8	3	8	12
Lyme disease 3 4 2 8 7 5 5 9	Lyme disease	3	4	2	8	7	5	5	9
Malaria 62 55 50 62 43 46 54 69	Malaria	62	55	50	62	43	46	54	69
Measles ^b 2 4 3 1 5 8 3 6	Measles ^b	2	4	3	1	5		3	6
Meningitis viral 185 228 443 226 263 378 269 446	Meningitis, viral	185	228	443	226	263	378	269	446
Meningacoccal infections 59 74 50 49 53 58 57 75	Meningococcal infections	59	74	50	49	53	58	57	75
Mumps 37 39 21 24 29 17 30 44	Mumps	37	39	21	24	29	17	30	44
Pertussis 120 32 77 238 102 103 114 248	Pertussis	120	32	77	238	102	103	114	248
	Psittacosis	0	1	0	1	0	1	0	
	Q-fever	0	0	1	0	1	1	0	1
Relapsing fever 0 0 1 0 0 1	Relapsing fever	Õ	õ	0	1	0	0	0	1
Rheumatic fever, acute ^b 2 1 0 1 1 6 1 2	Rheumatic fever, acute ^b	2	1	Õ	1	1	6	1	2
	Rubella	5	5	Õ	O	3	Õ	3	7
Salmonellosis 1771 1696 1253 1101 990 1006 1362 1980	Salmonellosis	1771	1696	1253	1101	990	1006	1362	1980
Shigellosis 1132 857 784 669 849 684 858 1157	Shigellosis	1132	857	784	669	849	684	858	1157
Strongyloidiasis 4 1 5 5 1 0 3 7	Strongyloidiasis	4	1	5	5	1	0	3	7
	Tetanus	1	4	2	2	0	2	2	4
	Trichinosis	0	2	3	0	Õ	0	1	3
	Tularemia	2	1	0	0	0	0	1	2
Typhoid fever case 31 27 17 20 21 17 23 33	Typhoid fever case	21	27	17	20	21	17	22	22
Typhoid fever carrier $4 \ 1 \ 12 \ 4 \ 6 \ 1 \ 5 \ 13$	Typhoid fever carrier	4	<u>-</u> ' 1	12	20	6	1	5	13
Typhus fever 16 14 7 6 17 8 12 21	Typhus fever	16	14	7	т А	17	, 8	12	21
Vibrio 22 29 30 3 13 15 19 39	Vibrio	22	29	, 30	3	13	15	19	39

Table G. Reported Cases of Selected Notifiable Diseases by Year of OnsetLos Angeles County, 1996-2001

^aThe normal distribution assumption may not apply to some rare diseases. ^b2001 data over 95% upper limit.

Table H. Annual Incidence Rates of Selected Notifiable Diseases by Year of Onset Los Angeles County, 1996-2001

		Annual I	ncidence Rat	e (Cases per	100,000) ^b	
Disease	1996	1997	1998	1999	2000	2001
Amebiasis	2.41	1.66	1.73	1.46	1.22	1.56
Botulism	0.05	0.03	0.01	0.03	-	0.02
Brucellosis	0.14	0.07	0.02	0.03	0.04	0.10
Campylobacteriosis	19.54	17.11	13.42	11.87	14.27	12.79
Cholera	-	-	0.03	-	-	-
Coccidioidomycosis	0.86	0.56	0.58	0.52	0.65	0.76
Cryptosporidiosis	1.61	0.95	1.03	0.77	0.76	0.86
Cysticercosis	0.38	0.38	0.26	0.31	0.48	0.41
Dengue	-	0.02	0.06	0.03	0.03	0.06
E. coli O157:H7	0.20	0.22	0.24	0.13	0.30	0.35
Encephalitis	0.38	0.47	0.53	0.43	0.55	0.46
Foodborne outbreaks	0.14	0.45	0.37	0.43	0.45	-
Giardiasis	10.78	8.76	7.41	6.45	5.71	5.00
Haemophilus influenzae type b	0.03	0.11	0.08	-	0.01	0.07
Hansen's Disease (Leprosy)	0.10	0.20	0.14	0.11	0.10	0.02
Hepatitis A	15.38	17 64	10.36	12 21	9 41	6.08
Hepatitis B	2.80	1.22	1.01	0.72	0.73	0.49
Hepatitis C	0.11	0.26	0.13	0.23	0.11	0.01
Henatitis unspecified	0.32	0.19	0.14	0.10	0.12	0.01
Kawasaki syndrome	0.23	0.10	0.36	0.32	0.39	0.01
Legionellosis	0.20	0.36	0.22	0.02	0.00	0.20
Listeriosis nonnerinatal	0.11	0.00	0.26	0.23	0.10	0.20
Listeriosis perinatal	3.18	5.31	4 74	8 26	5.46	2.05
Lyme disease	0.03	0.04	0.02	0.09	0.08	0.06
Malaria	0.00	0.61	0.55	0.68	0.00	0.52
Measles	0.02	0.04	0.03	0.00	0.06	0.02
Meningitis viral	2.09	2 54	4 88	2 46	2 95	4 24
Meningococcal infections	0.67	0.82	0.55	0.53	0.59	0.65
Mumps	0.07	0.43	0.23	0.26	0.33	0.00
Pertussis	1.35	0.36	0.85	2 59	1 14	1 15
Psittacosis	-	0.00	-	0.01	-	0.01
Q-fever	-	-	0.01	-	0.01	0.01
Relansing fever	-	-	-	0.01	-	-
Rheumatic fever acute	0.02	0.01	-	0.01	0.01	0.07
Rubella	0.02	0.06	_	-	0.03	
Salmonellosis	19.00	18 91	13.82	12 00	11 10	11 28
Shinellosis	12.76	9.55	8 64	7 29	9.52	7.67
Strongyloidiasis	0.05	0.00	0.06	0.05	0.02	-
Tetanus	0.00	0.04	0.00	0.00	-	0.02
Trichinosis	0.01	0.04	0.02	0.02	_	0.02
Tularemia	0.02	0.02	0.00	-	-	
Typhoid fever case	0.02	0.01	0 1 9	0.22	0.24	0 1 9
Typhoid fever, carrier	0.00	0.00	0.13	0.22	0.27	0.13
Typhile fever	0.00	0.01	0.13	0.04	0.07	0.01
Vibrio	0.10	-	0.00	0.07	0.15	0.03
	0.20	-	0.00	0.00	0.10	0.17

^aRates for perinatal listeriosis were calculated as cases per 100,000 live births. ^bRates of disease based on less than 20 cases or events are considered "unreliable." A zero rate made from no events is especially hazardous and are not reported here, except with a dash ("-"). Conclusions drawn from unreliable rates should be made with caution, if they are to be made at all.

Amebiasis12.09.611.211.012.211.610.212.610.411.87.66.4126.6Botulism0.20.00.20.00.00.20.60.00.00.00.40.21.8Brucellosis0.00.40.00.40.40.40.20.40.21.20.40.24.2Campylobacteriosis78.871.874.4101.2128.2140.0134.8121.2117.0106.890.461.01225.6Cholera0.00.00.00.00.00.00.00.00.20.20.20.20.00.6Coccidioidomycosis4.82.83.04.23.63.05.03.24.25.23.63.045.6Cryptosporidiosis6.23.24.63.65.25.27.49.28.87.05.05.871.2Cysticercosis2.81.82.43.23.81.01.63.01.62.41.02.42.7Dengue0.60.00.00.40.60.60.40.00.40.00.23.23.2E. coli O157:H72.00.41.20.61.22.45.82.23.01.21.40.622.0Encephalitis3.43.42.83.43.42.22.02.23.4	Disease	Jan	Feb	Mar	Apr	Мау	June	July	Aug	Sept	Oct	Nov	Dec	Total ^a
Botulism0.20.00.20.00.00.20.60.00.00.00.40.21.8Brucellosis0.00.40.00.40.40.40.40.20.40.21.20.40.24.2Campylobacteriosis78.871.874.4101.2128.2140.0134.8121.2117.0106.890.461.01225.6Cholera0.00.00.00.00.00.00.00.00.00.00.20.20.20.00.6Coccidioidomycosis4.82.83.04.23.63.05.03.24.25.23.63.045.6Cryptosporidiosis6.23.24.63.65.25.27.49.28.87.05.05.871.2Cysticercosis2.81.82.43.23.81.01.63.01.62.41.02.42.7Dengue0.60.00.00.40.60.60.40.00.40.60.23.23.23.23.43.42.23.23.01.21.40.622.0E. coli O157:H72.00.41.20.61.22.45.82.23.01.21.40.622.0Encephalitis3.43.42.83.43.42.22.02.23.43.42.43.22.4<	Amebiasis	12.0	9.6	11.2	11.0	12.2	11.6	10.2	12.6	10.4	11.8	7.6	6.4	126.6
Brucellosis0.00.40.00.40.40.40.20.40.21.20.40.24.2Campylobacteriosis78.871.874.4101.2128.2140.0134.8121.2117.0106.890.461.01225.6Cholera0.00.00.00.00.00.00.00.00.20.20.20.20.00.6Coccidioidomycosis4.82.83.04.23.63.05.03.24.25.23.63.045.6Cryptosporidiosis6.23.24.63.65.25.27.49.28.87.05.05.871.2Cysticercosis2.81.82.43.23.81.01.63.01.62.41.02.42.7Dengue0.60.00.00.40.60.60.40.00.40.00.23.2E. coli O157:H72.00.41.20.61.22.45.82.23.01.21.40.622.0Encephalitis3.43.42.83.43.42.22.02.23.42.83.22.434.6Giardiasis41.835.444.045.648.445.256.067.665.648.241.831.4571.0Haemophilus influenzae type b0.80.60.40.40.40.40.20	Botulism	0.2	0.0	0.2	0.0	0.0	0.2	0.6	0.0	0.0	0.0	0.4	0.2	1.8
Campylobacteriosis 78.8 71.8 74.4 101.2 128.2 140.0 134.8 121.2 117.0 106.8 90.4 61.0 1225.6 Cholera 0.0 <t< td=""><td>Brucellosis</td><td>0.0</td><td>0.4</td><td>0.0</td><td>0.4</td><td>0.4</td><td>0.4</td><td>0.2</td><td>0.4</td><td>0.2</td><td>1.2</td><td>0.4</td><td>0.2</td><td>4.2</td></t<>	Brucellosis	0.0	0.4	0.0	0.4	0.4	0.4	0.2	0.4	0.2	1.2	0.4	0.2	4.2
Cholera 0.0 <	Campylobacteriosis	78.8	71.8	74.4	101.2	128.2	140.0	134.8	121.2	117.0	106.8	90.4	61.0	1225.6
Coccidioidomycosis 4.8 2.8 3.0 4.2 3.6 3.0 5.0 3.2 4.2 5.2 3.6 3.0 45.6 Cryptosporidiosis 6.2 3.2 4.6 3.6 5.2 5.2 7.4 9.2 8.8 7.0 5.0 5.8 71.2 Cysticercosis 2.8 1.8 2.4 3.2 3.8 1.0 1.6 3.0 1.6 2.4 1.0 2.4 2.7 Dengue 0.6 0.0 0.0 0.4 0.6 0.6 0.4 0.0 0.4 0.0 0.0 0.2 3.2 E. coli O157:H7 2.0 0.4 1.2 0.6 1.2 2.4 5.8 2.2 3.0 1.2 1.4 0.6 22.0 Encephalitis 3.4 3.4 2.8 3.4 3.4 2.2 2.0 2.2 3.4 3.4 3.4 2.2 2.0 2.2 3.4 3.4 2.4 3.4 3.4 2.2 2.0 2.2 3.4 3.4 2.4 3.6 3.2	Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.2	0.2	0.0	0.6
Cryptosporidiosis 6.2 3.2 4.6 3.6 5.2 5.2 7.4 9.2 8.8 7.0 5.0 5.8 71.2 Cysticercosis 2.8 1.8 2.4 3.2 3.8 1.0 1.6 3.0 1.6 2.4 1.0 2.4 2.7 Dengue 0.6 0.0 0.0 0.4 0.6 0.6 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.0 0.2 3.2 E. coli O157:H7 2.0 0.4 1.2 0.6 1.2 2.4 5.8 2.2 3.0 1.2 1.4 0.6 22.0 Encephalitis 3.4 3.4 2.8 3.4 3.4 2.2 2.0 2.2 3.4 2.4 3.4 3.4 2.4 2.6 3.2 2.4 34.6 Giardiasis 41.8 35.4 44.0 <	Coccidioidomycosis	4.8	2.8	3.0	4.2	3.6	3.0	5.0	3.2	4.2	5.2	3.6	3.0	45.6
Cysticercosis 2.8 1.8 2.4 3.2 3.8 1.0 1.6 3.0 1.6 2.4 1.0 2.4 2.7 Dengue 0.6 0.0 0.0 0.4 0.6 0.6 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.4 0.0 0.0 0.2 3.2 E. coli O157:H7 2.0 0.4 1.2 0.6 1.2 2.4 5.8 2.2 3.0 1.2 1.4 0.6 22.0 Encephalitis 3.4 3.4 2.8 3.4 3.4 2.2 2.0 2.2 3.4 2.8 3.2 2.4 34.6 Giardiasis 41.8 35.4 44.0 45.6 48.4 45.2 56.0 67.6 65.6 48.2 41.8 31.4 571.0 Haemophilus influenzae type b 0.8 0.6 0.4 0.4 0.4 0.2 0.2 0.4 0.6 0.4 0.6 1.0 1.4 0.6 1.2 10.6	Cryptosporidiosis	6.2	3.2	4.6	3.6	5.2	5.2	7.4	9.2	8.8	7.0	5.0	5.8	71.2
Dengue 0.6 0.0 0.4 0.6 0.6 0.4 0.0 0.4 0.0 0.0 0.2 3.2 E. coli O157:H7 2.0 0.4 1.2 0.6 1.2 2.4 5.8 2.2 3.0 1.2 1.4 0.6 22.0 Encephalitis 3.4 3.4 2.8 3.4 3.4 2.2 2.0 2.2 3.4 2.8 3.2 2.4 34.6 Giardiasis 41.8 35.4 44.0 45.6 48.4 45.2 56.0 67.6 65.6 48.2 41.8 31.4 571.0 Haemophilus influenzae type b 0.8 0.6 0.4 0.4 0.4 0.2 0.2 0.4 0.2 0.6 0.0 4.6	Cysticercosis	2.8	1.8	2.4	3.2	3.8	1.0	1.6	3.0	1.6	2.4	1.0	2.4	2.7
E. coli O157:H7 2.0 0.4 1.2 0.6 1.2 2.4 5.8 2.2 3.0 1.2 1.4 0.6 22.0 Encephalitis 3.4 3.4 2.8 3.4 3.4 2.2 2.0 2.2 3.4 2.8 3.4 3.4 2.2 2.0 2.2 3.4 2.8 3.2 2.4 34.6 Giardiasis 41.8 35.4 44.0 45.6 48.4 45.2 56.0 67.6 65.6 48.2 41.8 31.4 571.0 Haemophilus influenzae type b 0.8 0.6 0.4 0.4 0.4 0.2 0.2 0.4 0.2 0.6 0.0 4.6	Dengue	0.6	0.0	0.0	0.4	0.6	0.6	0.4	0.0	0.4	0.0	0.0	0.2	3.2
Encephalitis 3.4 3.4 2.8 3.4 3.4 2.2 2.0 2.2 3.4 2.8 3.2 2.4 34.6 Giardiasis 41.8 35.4 44.0 45.6 48.4 45.2 56.0 67.6 65.6 48.2 41.8 31.4 571.0 Haemophilus influenzae type b 0.8 0.6 0.4 0.4 0.4 0.2 0.2 0.4 0.2 0.6 0.0 4.6	E. coli O157:H7	2.0	0.4	1.2	0.6	1.2	2.4	5.8	2.2	3.0	1.2	1.4	0.6	22.0
Giardiasis 41.8 35.4 44.0 45.6 48.4 45.2 56.0 67.6 65.6 48.2 41.8 31.4 571.0 Haemophilus influenzae type b 0.8 0.6 0.4 0.4 0.4 0.2 0.2 0.4 0.2 0.6 0.0 4.6 Haemophilus influenzae (Leptropy) 0.6 0.8 0.8 0.4 1.0 1.0 1.4 1.0 0.6 1.2 10.6	Encephalitis	3.4	3.4	2.8	3.4	3.4	2.2	2.0	2.2	3.4	2.8	3.2	2.4	34.6
Haemophilus influenzae type b 0.8 0.6 0.4 0.4 0.4 0.4 0.2 0.2 0.4 0.2 0.6 0.0 4.6	Giardiasis	41.8	35.4	44.0	45.6	48.4	45.2	56.0	67.6	65.6	48.2	41.8	31.4	571.0
Hanson's Disease (Lapropu) 0.6 0.8 0.8 0.4 1.0 1.0 1.4 1.0 0.6 1.4 0.6 1.2 10.6	Haemophilus influenzae type b	0.8	0.6	0.4	0.4	0.4	0.4	0.2	0.2	0.4	0.2	0.6	0.0	4.6
Taliseli's Disease (Lepiusy) 0.0 0.0 0.0 0.4 1.0 1.0 1.4 1.0 0.0 1.4 0.0 1.2 10.0	Hansen's Disease (Leprosy)	0.6	0.8	0.8	0.4	1.0	1.0	1.4	1.0	0.6	1.4	0.6	1.2	10.6
Hepatitis A 73.0 71.4 89.6 74.2 77.6 66.2 68.4 91.0 104.6 85.2 60.4 45.4 907.0	Hepatitis A	73.0	71.4	89.6	74.2	77.6	66.2	68.4	91.0	104.6	85.2	60.4	45.4	907.0
Hepatitis B 9.0 10.6 9.6 9.8 10.6 7.2 10.6 6.4 9.4 6.2 7.0 4.8 101.2	Hepatitis B	9.0	10.6	9.6	9.8	10.6	7.2	10.6	6.4	9.4	6.2	7.0	4.8	101.2
Hepatitis C 2.2 1.6 1.4 1.2 1.8 1.8 2.0 1.8 2.2 0.8 1.0 1.6 19.4	Hepatitis C	2.2	1.6	1.4	1.2	1.8	1.8	2.0	1.8	2.2	0.8	1.0	1.6	19.4
Hepatitis unspecified 1.2 0.2 0.8 0.8 0.8 0.2 1.0 0.4 0.2 0.0 0.4 6.8	Hepatitis unspecified	1.2	0.2	0.8	0.8	0.8	0.8	0.2	1.0	0.4	0.2	0.0	0.4	6.8
Kawasaki syndrome 1.4 2.4 3.2 2.0 2.4 2.2 1.4 1.8 1.4 1.8 0.8 2.4 23.2	Kawasaki syndrome	1.4	2.4	3.2	2.0	2.4	2.2	1.4	1.8	1.4	1.8	0.8	2.4	23.2
Legionellosis 1.0 1.8 0.8 1.2 0.2 1.2 1.6 1.0 1.6 2.8 2.8 1.2 17.2	Legionellosis	1.0	1.8	0.8	1.2	0.2	1.2	1.6	1.0	1.6	2.8	2.8	1.2	17.2
Listeriosis, nonperinatal 1.2 1.4 1.0 0.6 2.2 5.0 1.8 1.8 2.8 1.8 1.2 1.0 21.8	Listeriosis, nonperinatal	1.2	1.4	1.0	0.6	2.2	5.0	1.8	1.8	2.8	1.8	1.2	1.0	21.8
Listeriosis, perinatal 0.6 0.2 0.4 1.2 0.8 1.2 0.8 0.8 0.6 0.4 0.4 0.2 7.6	Listeriosis, perinatal	0.6	0.2	0.4	1.2	0.8	1.2	0.8	0.8	0.6	0.4	0.4	0.2	7.6
Lyme disease 0.2 0.0 0.4 0.4 0.4 0.6 0.4 1.0 0.6 0.4 0.0 0.2 4.6	Lyme disease	0.2	0.0	0.4	0.4	0.4	0.6	0.4	1.0	0.6	0.4	0.0	0.2	4.6
Malaria 3.6 4.0 4.0 3.6 4.4 4.0 5.8 7.0 3.8 3.0 1.6 3.8 48.6	Malaria	3.6	4.0	4.0	3.6	4.4	4.0	5.8	7.0	3.8	3.0	1.6	3.8	48.6
Measles 0.2 0.4 0.8 0.6 0.6 0.6 0.4 0.4 0.0 0.2 0.0 0.0 4.2	Measles	0.2	0.4	0.8	0.6	0.6	0.6	0.4	0.4	0.0	0.2	0.0	0.0	4.2
Meningitis, viral 13.8 10.6 13.4 15.0 20.4 29.4 31.4 38.0 42.2 30.4 19.2 14.4 278.2	Meningitis, viral	13.8	10.6	13.4	15.0	20.4	29.4	31.4	38.0	42.2	30.4	19.2	14.4	278.2
Meningococcal infections 10.2 6.4 6.8 6.2 3.4 5.2 3.0 2.0 1.8 2.0 1.6 4.2 52.8	Meningococcal infections	10.2	6.4	6.8	6.2	3.4	5.2	3.0	2.0	1.8	2.0	1.6	4.2	52.8
Mumps 4.2 2.4 2.4 1.4 2.8 3.4 1.0 0.6 2.2 2.4 1.4 1.8 26.0	Mumps	4.2	2.4	2.4	1.4	2.8	3.4	1.0	0.6	2.2	2.4	1.4	1.8	26.0
Pertussis 5.6 6.0 4.0 7.8 8.6 10.2 11.6 14.2 13.4 11.6 6.4 11.0 110.4	Pertussis	5.6	6.0	4.0	7.8	8.6	10.2	11.6	14.2	13.4	11.6	6.4	11.0	110.4
Psittacosis 0.0 0.0 0.0 0.2 0.0 0.2 0.0 0.0 0.0 0.0	Psittacosis	0.0	0.0	0.0	0.2	0.0	0.2	0.0	0.0	0.0	0.0	0.2	0.0	0.6
Q-rever 0.2 0.0 0.0 0.2 0.2 0.0 0.0 0.0 0.0 0.0	Q-fever	0.2	0.0	0.0	0.2	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.6
Relapsing fever 0.0	Relapsing fever	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.2
	Rheumatic fever, acute	0.0	0.2	0.4	0.0	0.4	0.0	0.0	0.2	0.0	0.0	0.0	0.0	1.0
		70.0	0.2	70.0	0.0	0.2	107.0	120.4	120.0	107.4	112.6	0.2	0.0	0.0
Salmonellosis 76.0 59.0 76.2 67.6 99.0 107.2 126.4 130.0 127.4 113.0 92.0 00.0 1102.4	Salmonellosis	70.0 62.4	59.0 21.2	10.Z	01.0	99.0 45.6	107.Z	120.4	130.0	127.4	74.0	92.0 52.0	25.0	744.6
Singeliosis 05.4 51.2 55.0 56.0 45.0 55.4 51.2 121.0 56.4 74.0 55.6 55.0 744.0	Shigeliosis	03.4	0.4	35.0	30.0	40.0	0.0	97.2	121.0	90.4	74.0	0.0	35.0	1 44.0
		0.2	0.4	0.2	0.2	0.2	0.0	0.2	0.2	0.0	0.2	0.0	0.0	1.0
Teiching 0.2 0.0 0.2 0.4 0.2 0.0 0.4 0.0 0.2 0.0 0.0 0.2 1.0	Trichingsie	0.2	0.0	0.2	0.4	0.2	0.0	0.4	0.0	0.2	0.0	0.0	0.2	1.0
	Tuloromia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	Turboid fovor 2000	1.0	1.2	0.0	2.0	2.6	24	1 /	0.0 1 /	0.0 2 /	1.6	0.0	0.0	10.2
Typhold level, Gase 1.0 1.2 2.0 2.0 2.0 2.4 1.4 1.4 2.4 1.0 0.2 0.0 19.0	Typhoid fever, case	0.4	1.Z	2.0 0.4	2.U 0.0	2.0 0.6	2.4 0.0	1.4 ∩ 2	1.4 0.4	∠.4 ∩ 2	0.1	0.2	0.0	19.0
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	Typhus fovor	0.4	0.4	0.4	0.0 0.2	1.0	1.0	0.Z 1 0	0.4 0.2	0.2	1.6	1.4	0.Z	0.0 0.2
$Vibrio \qquad 0.6 0.6 0.2 0.0 1.0 1.2 1.0 0.0 0.0 1.0 1.2 1.0 9.0 9.0 9.0 1.0 1.2 1.0 9$	Vibrio	0.4	0.0	0.2	0.0	0.6	2.8	3.8	24	2.0	2.0	0.2	0.0	9.0 15.6

Table I. Five –Year Average of Notifiable Diseases by Month of OnsetLos Angeles County, 1997-2001

Amebiasis 0 7 16 44 32 12 17 6 139 Botulism 0 0 0 0 1 1 0 0 2 Brucellosis 0 0 3 2 0 1 0 3 2 Campylobacteriosis 44 161 173 326 148 112 82 95 1141 Cholera 0 <t< th=""><th>Disease</th><th><1</th><th>1-4</th><th>5-14</th><th>15-34</th><th>35-44</th><th>45-54</th><th>55-64</th><th>65+</th><th>Total</th></t<>	Disease	<1	1-4	5-14	15-34	35-44	45-54	55-64	65+	Total
Botulism 0 0 0 0 1 1 0 0 2 Brucellosis 0 0 3 2 0 1 0 3 9 Campylobacteriosis 44 161 173 326 148 112 82 95 1141 Cholera 0	Amebiasis	0	7	16	44	32	12	17	6	139
Brucellosis 0 0 3 2 0 1 0 3 9 Campylobacteriosis 44 161 173 326 148 112 82 95 1141 Cholera 0 <t< td=""><td>Botulism</td><td>0</td><td>0</td><td>0</td><td>0</td><td>1</td><td>1</td><td>0</td><td>0</td><td>2</td></t<>	Botulism	0	0	0	0	1	1	0	0	2
Campylobacteriosis 44 161 173 326 148 112 82 95 1141 Cholera 0	Brucellosis	0	0	3	2	0	1	0	3	9
Cholera 0 </td <td>Campylobacteriosis</td> <td>44</td> <td>161</td> <td>173</td> <td>326</td> <td>148</td> <td>112</td> <td>82</td> <td>95</td> <td>1141</td>	Campylobacteriosis	44	161	173	326	148	112	82	95	1141
Coccidioidomycosis 0 0 19 13 17 10 9 68 Cryptosporidiosis 0 1 3 23 35 9 2 3 77 Cysticercosis 0 0 0 15 8 7 4 2 37 Dengue 0 0 0 2 1 2 0 0 2 3 27	Cholera	0	0	0	0	0	0	0	0	0
Cryptosporidiosis 0 1 3 23 35 9 2 3 77 Cysticercosis 0 0 0 15 8 7 4 2 37 Dengue 0 0 0 2 1 2 0 0 5	Coccidioidomycosis	0	0	0	19	13	17	10	9	68
Cysticercosis 0 0 0 15 8 7 4 2 37 Dengue 0 0 0 2 1 2 0 0 5 Dengue 0 0 0 2 1 2 0 0 5	Cryptosporidiosis	0	1	3	23	35	9	2	3	77
Dengue 0 0 0 2 1 2 0 0 5	Cysticercosis	0	0	0	15	8	7	4	2	37
	Dengue	0	0	0	2	1	2	0	0	5
	E. <i>coli</i> O157:H7	0	4	11	6	2	4	1	3	31
Encephalitis 2 7 8 9 4 4 4 3 41	Encephalitis	2	7	8	9	4	4	4	3	41
Giardiasis 6 78 118 91 82 38 18 10 446	Giardiasis	6	78	118	91	82	38	18	10	446
Haemophilus influenzae type b 2 1 0 1 1 6	<i>Haemophilus influenzae</i> type b	2	1	0	1	0	0	1	1	6
Hansen's Disease (Leprosy) 0 0 0 1 0 0 1 2	Hansen's Disease (Leprosy)	0	0	0	1	0	0	0	1	2
Hepatitis A 0 23 110 163 114 49 31 50 542	Hepatitis A	0	23	110	163	114	49	31	50	542
Hepatitis B 0 0 0 17 17 6 2 2 44	Hepatitis B	0	0	0	17	17	6	2	2	44
Hepatitis C 0 0 0 1 0 0 0 1	Hepatitis C	0	0	0	1	0	0	0	0	1
Hepatitis unspecified 0 0 0 0 1 0 0 1	Hepatitis unspecified	0	0	0	0	1	0	0	0	1
Kawasaki syndrome 7 15 2 0 0 0 24	Kawasaki syndrome	7	15	2	0	0	0	0	0	24
Legionellosis 0 0 0 1 0 9 5 3 18	Legionellosis	0	0	0	1	0	9	5	3	18
Listeriosis, nonperinatal 0 0 2 6 4 3 3 9 27	Listeriosis, nonperinatal	0	0	2	6	4	3	3	9	27
Listeriosis, perinatal 0 0 0 2 1 0 0 3	Listeriosis, perinatal	0	0	0	2	1	0	0	0	3
Lyme disease 0 1 0 1 2 0 0 5	Lyme disease	0	1	0	1	2	0	0	0	5
Malaria 1 0 2 14 11 11 2 5 46	Malaria	1	0	2	14	11	11	2	5	46
Measles 1 2 2 2 1 0 0 6	Measles	1	2	2	2	1	0	0	0	8
Meningitis, viral 68 25 92 100 56 13 8 7 378	Meningitis, viral	68	25	92	100	56	13	8	7	378
Meningococcal infections 5 6 5 23 4 4 3 8 58	Meningococcal infections	5	6	5	23	4	4	3	8	58
Mumps 0 4 8 4 1 0 0 0 17	Mumps	0	4	8	4	1	0	0	0	17
Pertussis 71 1 13 12 5 0 0 1 103	Pertussis	71	1	13	12	5	0	0	1	103
Psittacosis 0 0 0 0 1 0 0 0 1	Psittacosis	0	0	0	0	1	0	0	0	1
Q-fever 0 0 0 0 0 0 1 1	Q-fever	0	0	0	0	0	0	0	1	1
Relapsing rever 0 0 0 0 0 0 0 0 0 0 0	Relapsing fever	0	0	0	0	0	0	0	0	0
	Rheumatic fever, acute	0	0	3	2	1	0	0	0	6
		0	0	0	0	0	0	0	0	1000
Saimoneilosis 96 195 172 205 102 88 57 88 1000	Salmonellosis	96	195	172	205	102	88	57	88	1006
Snigeliosis 12 154 164 171 94 53 19 17 684	Shigeliosis	12	154	164	1/1	94	53	19	17	684
	Strongyloidiasis	0	0	0	0	0	0	0	0	0
	Trichingsia	0	0	0	0	0	0	0	2	2
	Tularamia	0	0	0	0	0	0	0	0	0
	Turbeid fever esse	0	0	0	0	0	0	0	1	17
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	Typhold level, case	0	2	1	1	3	2	1		17
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	Typhus fovor	0	0	U 1	1	0	U 2	0	U 1	1
Vibrio 0 0 0 1 1 2 3 0 1 7	Vibrio	0	0	۱ 0	I ⊿	2	3 2	6	0	/ 15

Table J. Number of Cases of Selected Notifiable Diseases by Age GroupLos Angeles County, 2001

^aTotals include cases with unknown age.

	Age-group Rates (Cases per 100,000) ^b							
Disease	<1	1-4	5-14	15-34	35-44	45-54	55-64	65+
Amebiasis	-	1.3	1.1	1.6	2.2	1.1	2.6	0.7
Botulism	-	-	-	-	0.1	0.1	-	-
Brucellosis	-	-	0.2	0.1	-	0.1	-	0.3
Campylobacteriosis	32.9	29.0	12.1	11.7	10.4	10.4	12.5	10.9
Cholera	-	-	-	-	-	-	-	-
Coccidioidomycosis	-	-	-	0.7	0.9	1.6	1.5	1.0
Cryptosporidiosis	-	0.2	0.2	0.8	2.5	0.8	0.3	0.3
Cysticercosis	-	-	-	0.5	0.6	0.6	0.6	0.2
Dengue	-	-	-	0.1	0.1	0.2	-	-
E. coli O157:H7	-	0.7	0.8	0.2	0.1	0.4	0.2	0.3
Encephalitis	1.5	1.3	0.6	0.3	0.3	0.4	0.6	0.3
Giardiasis	4.5	14.0	8.2	3.3	5.8	3.5	2.7	1.2
Haemophilus influenzae type b	1.5	0.2	-	-	-	-	0.2	0.1
Hansen's Disease (Leprosy)	-	-	-	-	-	-	-	0.1
Hepatitis A	-	4.1	7.7	5.9	8.0	4.5	4.7	5.8
Hepatitis B	-	-	-	0.6	1.2	0.6	0.3	0.2
Hepatitis C	-	-	-	-	-	-	-	-
Hepatitis unspecified	-	-	-	-	0.1	-	-	-
Kawasaki syndrome	5.2	2.7	0.1	-	-	-	-	-
Legionellosis	-	-	-	-	-	0.8	0.8	0.3
Listeriosis, nonperinatal	-	-	0.1	0.2	0.3	0.3	0.5	1.0
Listeriosis, perinatal	-	-	-	1.7	4.2	-	-	-
Lyme disease	-	0.2	-	-	0.1	-	-	-
Malaria	0.7	-	0.1	0.5	0.8	1.0	0.3	0.6
Measles	0.7	0.4	0.1	0.1	0.1	-	-	-
Meningitis, viral	50.8	4.5	6.4	3.6	3.9	1.2	1.2	0.8
Meningococcal infections	3.7	1.1	0.3	0.8	0.3	0.4	0.5	0.9
Mumps	-	0.7	0.6	0.1	0.1	-	-	-
Pertussis	53.1	0.2	0.9	0.4	0.4	-	-	0.1
Psittacosis	-	-	-	-	0.1	-	-	-
Q-fever	-	-	-	-	-	-	-	0.1
Relapsing fever	-	-	-	-	-	-	-	-
Rheumatic fever, acute	-	-	0.2	0.1	0.1	-	-	-
Rubella	-	-	-	-	-	-	-	-
Salmonellosis	71.8	35.1	12.0	7.4	7.2	8.2	8.7	10.1
Shigellosis	9.0	27.7	11.5	6.2	6.6	4.9	2.9	2.0
Strongyloidiasis	-	-	-	-	-	-	-	-
Tetanus	-	-	-	-	-	-	-	0.2
Trichinosis	-	-	-	-	-	-	-	-
Tularemia	-	-	-	-	-	-	-	-
Typhoid fever, case	-	0.4	0.1	0.3	0.2	0.2	0.2	0.1
Typhoid fever, carrier	-	-	-	-	-	-	-	-
Typhus fever	-	-	0.1	-	0.1	0.3	-	0.1
Vibrio	-	-	-	0.1	0.1	0.3	0.9	-

Table K. Incidence Rates of Selected Notifiable Diseases by Age GroupLos Angeles County, 2001

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

Disease	Asian	Black	Hispanic	White	Other ^a	Unknown
Amebiasis	7	7	56	38	0	31
Botulism	0	0	1	1	0	0
Brucellosis	0	0	8	0	0	1
Campylobacteriosis	97	46	608	373	9	8
Cholera	0	0	0	0	0	0
Coccidioidomycosis	11	11	24	14	3	5
Cryptosporidiosis	1	4	20	28	3	21
Cysticercosis	0	0	37	0	0	0
Dengue	2	0	2	1	0	0
E. coli O157:H7	0	6	6	19	0	0
Encephalitis	7	3	21	10	0	0
Giardiasis	12	10	194	162	14	54
Haemophilus influenzae type b	0	0	3	1	0	2
Hansen's Disease (Leprosy)	2	0	0	0	0	0
Hepatitis A	37	28	214	185	67	11
Hepatitis B	5	10	15	12	1	1
Hepatitis C	0	0	0	1	0	0
Hepatitis unspecified	1	0	0	0	0	0
Kawasaki syndrome	4	0	12	7	0	1
Legionellosis	1	0	2	15	0	0
Listeriosis, nonperinatal	5	0	6	16	0	0
Listeriosis, perinatal	1	0	2	0	0	0
Lyme disease	1	0	0	4	0	0
Malaria	3	22	12	7	0	2
Measles	3	1	3	1	0	0
Meningitis, viral	15	26	130	113	4	90
Meningococcal infections	2	6	22	28	0	0
Mumps	1	1	12	0	0	3
Pertussis	4	8	59	31	0	1
Psittacosis	0	1	0	0	0	0
Q-fever	1	0	0	0	0	0
Relapsing fever	0	0	0	0	0	0
Rheumatic fever, acute	1	0	1	1	0	3
Rubella	0	0	0	0	0	0
Salmonellosis	114	76	497	271	13	35
Shigellosis	18	27	466	153	6	14
Strongyloidiasis	0	0	0	0	0	0
Tetanus	0	0	0	1	1	0
Trichinosis	0	0	0	0	0	0
Tularemia	0	0	0	0	0	0
Typhoid fever, case	6	1	8	0	1	1
Typhoid fever, carrier	0	0	1	0	0	0
Typhus fever	0	0	2	4	0	2
Vibrio	3	0	10	1	0	1

Table L. Number of Cases of Selected Notifiable Diseases by Race/EthnicityLos Angeles County, 2001

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

	Race/Eth	Race/Ethnicity Rates (Cases per 100,000) ^b						
Disease	Asian	Black	Hispanic	White				
Amebiasis	0.7	0.9	1.4	1.4				
Botulism	-	-	-	-				
Brucellosis	-	-	0.2	-				
Campylobacteriosis	9.0	5.6	15.1	13.6				
Cholera	-	-	-	-				
Coccidioidomycosis	1.0	1.3	0.6	0.5				
Cryptosporidiosis	0.1	0.5	0.5	1.0				
Cysticercosis	-	-	0.9	-				
Dengue	0.2	-	-	-				
E. coli O157:H7	-	0.7	0.1	0.7				
Encephalitis	0.7	0.4	0.5	0.4				
Giardiasis	1.1	1.2	4.8	5.9				
Haemophilus influenzae type b	-	-	0.1	-				
Hansen's Disease (Leprosy)	0.2	-	-	-				
Hepatitis A	3.4	3.4	5.3	6.7				
Hepatitis B	0.5	1.2	0.4	0.4				
Hepatitis C	-	-	-	-				
Hepatitis unspecified	0.1	-	-	-				
Kawasaki syndrome	0.4	-	0.3	0.3				
Legionellosis	0.1	-	-	0.5				
Listeriosis, nonperinatal	0.5	-	0.1	0.6				
Listeriosis perinatal	7 1	-	22	-				
Lyme disease	0.1	-		0.1				
Malaria	0.3	27	0.3	0.3				
Measles	0.3	0.1	0.0	-				
Meningitis viral	1 4	3.2	3.2	4 1				
Meningococcal infections	0.2	0.2	0.5	1.1				
Mumps	0.1	0.1	0.3	-				
Pertussis	0.4	0.1	13	1 1				
Psittanosis	- 0.4	0.5	-					
	0.1	0.1	_	_				
Relansing fever	0.1		_					
Recuration fever acute	0.1							
Rubella	0.1			_				
Salmonellosis	10.6	03	12.3	- 0.8				
Shinollosis	17	3.3	12.5	5.0				
Strongyloidiacia	1.7	5.5	11.0	5.0				
	-	-	-	-				
Trichinggia	-	-	-	-				
Tularamia	-	-	-	-				
Turbaid fovor and	-	-	-	-				
Typhola lever, case	0.0	0.1	0.2	-				
i ypnoid iever, carrier	-	-	-	-				
rypnus rever	-	-	-	0.1				
	0.3	-	0.2	-				

Table M. Incidence Rates of Selected Notifiable Diseases by Race/Ethnicity Los Angeles County, 2001

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

Table N. Number of Cases and Annual Incidence Rate of Selected Notifiable Diseases by Sex Los Angeles County, 2001

		Male	Female			
		Rate (Cases per		Rate (Cases per		
Disease	Cases	100,000) 5	Cases	100,000) ⁵		
Amebiasis	86	2.0	52	1.2		
Botulism	1	0.0	1	0.0		
Brucellosis	4	0.1	5	0.1		
Campylobacteriosis	611	13.9	521	11.6		
Cholera	0	-	0	-		
Coccidioidomycosis	51	1.2	15	0.3		
Cryptosporidiosis	67	1.5	8	0.2		
Cysticercosis	19	0.4	17	0.4		
Dengue	3	0.1	2	0.0		
E. coli O157:H7	19	0.4	12	0.3		
Encephalitis	24	0.5	17	0.4		
Giardiasis	244	5.5	195	4.3		
Haemophilus influenzae type b	4	0.1	2	0.0		
Hansen's Disease (Leprosy)	1	0.0	1	0.0		
Hepatitis A	359	8.1	183	4.1		
Hepatitis B	31	0.7	13	0.3		
Hepatitis C	1	0.0	0	-		
Hepatitis unspecified	1	0.0	0	-		
Kawasaki syndrome	9	0.2	12	0.3		
Legionellosis	13	0.3	5	0.1		
Listeriosis, Nonperinatal	10	0.2	17	0.4		
Listeriosis, perinatal	0		3	2.1		
Lyme disease	3	0.1	2	0.0		
Malaria	20	0.5	26	0.6		
Measles	3	0.1	5	0.1		
Meningitis viral	200	4.5	172	3.8		
Meningococcal infections	34	0.8	24	0.5		
Mumps	8	0.0	9	0.0		
Pertussis	53	12	50	11		
Psittacosis	1	0.0	0	-		
Q-fever	1	0.0	0	-		
Relapsing fever	O	-	ů – Č	-		
Rheumatic fever acute	1	0.0	4	0.1		
Rubella	0	-	O	-		
Salmonellosis	466	10.6	540	12.0		
Shigellosis	386	8.8	293	6.5		
Strongyloidiasis	000	-	0	-		
Tetanus	1	0.0	1	0.0		
Trichinosis	0	-	, O	-		
Tularemia	0	-	n n	-		
Typhoid fever case	10	0.2	7	0.2		
Typhoid fever carrier	10	0.0	0	- 0.2		
Typhus fever	л Д	0.0	4	0.1		
Vibrio	13	0.3	2	0.0		

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

Table O-1. Selected Notifiable Diseases SPA 1. Antelope Valley Area Los Angeles County, 2001

	Frequency	Rate (Cases per 100,000) ^b
Disease	Antelope	Antelope
Amebiasis	2	0.6
Botulism	0	-
Brucellosis	0	-
Campylobacteriosis	39	12.2
Cholera	0	-
Coccidioidomycosis	7	2.2
Cryptosporidiosis	0	-
Cysticercosis	0	-
Dengue	0	-
E. coli O157:H7	1	0.3
Encephalitis	0	-
Giardiasis	14	4.4
Haemophilus influenzae type b	0	-
Hansen's Disease (Leprosy)	0	-
Hepatitis A	14	4.4
Hepatitis B	5	1.6
Hepatitis C	0	-
Hepatitis unspecified	0	-
Kawasaki syndrome	1	0.3
Legionellosis	0	-
Listeriosis, nonperinatal	0	-
Listeriosis, perinatal	0	-
Lyme disease	0	-
Malaria	1	0.3
Measles	0	-
Meningitis, viral	13	4.1
Meningococcal infections	1	0.3
Mumps	1	0.3
Pertussis	3	0.9
Psittacosis	0	-
Q-fever	0	-
Relapsing fever	0	-
Rheumatic fever, acute	0	-
Rubella	0	-
Salmonellosis	24	7.5
Shigellosis	10	3.1
Strongyloidiasis	0	-
Tetanus	0	-
Trichinosis	0	-
Tularemia	0	-
Typhoid fever, case	0	-
Typhoid fever, carrier	0	-
Typhus fever	0	-
Vibrio	0	<u> </u>

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

Table O-2.Selected Notifiable DiseasesSPA 2.San Fernando AreaLos Angeles County, 2001

_			Freque	ency		Rate (Cases per 100,000) ^b				
Disease	EV	GL	SF	wv	TOTAL	EV	GL	SF	wv	TOTAL
Amebiasis	8	11	11	10	40	1.9	3.3	2.8	1.2	2.0
Botulism	0	0	0	0	0	-	-	-	-	-
Brucellosis	0	0	0	1	1	-	-	-	0.1	0.1
Campylobacteriosis	47	42	66	97	252	11.1	12.6	16.8	11.8	12.8
Cholera	0	0	0	0	0	-	-	-	-	-
Coccidioidomycosis	3	0	6	11	20	0.7	-	1.5	1.3	1.0
Cryptosporidiosis	1	2	0	5	8	0.2	0.6	-	0.6	0.4
Cysticercosis	0	0	3	7	10	-	-	0.8	0.9	0.5
Dengue	0	0	0	0	0	-	-	-	-	-
E. coli O157:H7	0	3	1	3	7	-	0.9	0.3	0.4	0.4
Encephalitis	0	1	6	6	13	-	0.3	1.5	0.7	0.7
Giardiasis	22	54	15	39	130	5.2	16.2	3.8	4.8	6.6
Haemophilus influenzae type b	0	0	1	0	1	-	-	0.3	-	0.1
Hansen's Disease (Leprosy)	0	0	0	1	1	-	-		0.1	0.1
Hepatitis A	38	29	29	53	149	9.0	8.7	7.4	6.5	7.6
Hepatitis B	0	4	0	5	9	-	1.2	-	0.6	0.5
Hepatitis C	0	0	0	0	0	-	-	-	-	-
Hepatitis unspecified	0	0	0	0	0	-	-	-	-	-
Kawasaki syndrome	0	0	1	4	5	-	-	0.3	0.5	0.3
Legionellosis	0	1	2	0	3	-	0.3	0.5	-	0.2
Listeriosis, nonperinatal	0	9	1	1	11	-	2.7	0.3	0.1	0.6
Listeriosis, perinatal	0	0	0	0	0	-	-	-	-	-
Lyme disease	0	0	0	2	2	-	-	-	0.2	0.1
Malaria	1	1	1	4	7	0.2	0.3	0.3	0.5	0.4
Measles	0	0	0	1	1	-	-	-	0.1	0.1
Meningitis, viral	8	10	34	15	67	1.9	3.0	8.7	1.8	3.4
Meningococcal infections	3	3	0	(13	0.7	0.9	-	0.9	0.7
Mumps	1	0	1	3	5	0.2	-	0.3	0.4	0.3
Pertussis	4	5	4	12	25	0.9	1.5	1.0	1.5	1.3
Psittacosis	0	0	0	0	0	-	-	-	-	-
Q-tever	0	0	0	0	0	-	-	-	-	-
Relapsing rever	0	0	0	0	0	-	-	-	-	-
Rneumatic tever, acute	0	0	2	3	5	-	-	0.5	0.4	0.3
	0	0	10	100	0	-	-	-	-	-
Saimonellosis	24	34	40	106	204	5.7	10.2	10.2	12.9	10.4
Shigellosis	24	28	21	40	113	5.7	8.4	5.3	4.9	5.7
Strongyloidiasis	0	0	0	0	0	-	-	-	-	-
Trichinggio	1	0	0	1	2	0.2	-	-	0.1	0.1
Tuloromio	0	0	0	0	0	-	-	-	-	-
Turboid fovor	0	U 4	U 4	0	0	-	-	-	-	-
Typhoid fever, case	0		1	3	5	-	0.3	0.3	0.4	0.3
Typhola lever, carrier	0	0	0	0	0	-	-	-	-	-
i yprius iever	0	0	0	0	0	-	-	-	-	-
OITO	U	1	U	U	1	-	0.3	-	-	0.1

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

Table O-3. Selected Notifiable Diseases SPA 3. San Gabriel Area Los Angeles County, 2001

_			Freq	uency		Rate (Cases per 100,000) ^b				
Disease	AH	EM	FH	PO	TOTAL	AH	EM	FH	PO	TOTAL
Amebiasis	3	1	5	8	17	0.9	0.2	1.7	1.5	1.1
Botulism	0	0	0	1	1	-	-	-	0.2	0.1
Brucellosis	0	0	1	0	1	-	-	0.3	-	0.1
Campylobacteriosis	44	7	35	54	140	13.0	1.6	11.9	10.2	8.8
Cholera	0	0	0	0	0	-	-	-	-	-
Coccidioidomycosis	0	0	1	2	3	-	-	0.3	0.4	0.2
Cryptosporidiosis	2	1	1	3	7	0.6	0.2	0.3	0.6	0.4
Cysticercosis	0	0	2	4	6	-	-	0.7	0.8	0.4
Dengue	0	0	1	0	1	-	-	0.3	-	0.1
E. coli O157:H7	0	0	3	3	6	-	-	1.0	0.6	0.4
Encephalitis	2	2	4	3	11	0.6	0.5	1.4	0.6	0.7
Giardiasis	25	5	14	25	69	7.4	1.1	4.8	4.7	4.3
Haemophilus influenzae type b	0	0	0	0	0	-	-	-	-	-
Hansen's Disease (Leprosy)	0	1	0	0	1	-	0.2	-		0.1
Hepatitis A	16	4	16	28	64	4.7	0.9	5.4	5.3	4.0
Hepatitis B	0	0	5	3	8	-	-	1.7	0.6	0.5
Hepatitis C	0	0	0	0	0	-	-	-	-	-
Hepatitis unspecified	0	0	0	0	0	-	-	-	-	-
Kawasaki syndrome	2	0	2	5	9	0.6	-	0.7	0.9	0.6
Legionellosis	0	0	0	2	2	-	-	-	0.4	0.1
Listeriosis, nonperinatal	4	0	1	1	6	1.2	-	0.3	0.2	0.4
Listeriosis, perinatal	0	0	0	1	1	-	-	-	0.8	0.3
Lyme disease	0	0	0	0	0	-	-	-	-	-
Malaria	2	1	0	3	6	0.6	0.2	-	0.6	0.4
Measles	0	0	0	0	0		-	-		-
Meningitis, viral	19	8	36	41	104	5.6	1.8	12.2	7.7	6.5
Meningococcal infections	2	1	2	8	13	0.6	0.2	0.7	1.5	0.8
Mumps	0	0	0	1	1	-	-	-	0.2	0.1
Pertussis	3	2	3	(15	0.9	0.5	1.0	1.3	0.9
Psittacosis	0	0	0	0	0	-	-	-	-	-
Q-fever	0	0	0	0	0	-	-	-	-	-
Relapsing fever	0	0	0	0	0	-	-	-	-	-
Rheumatic fever, acute	0	0	0	0	0	-	-	-	-	-
	0	0	0	0	0	-	-	-	-	-
Salmonellosis	47	20	43	62	172	13.9	4.6	14.6	11.7	10.8
Shigellosis Strang and sidia size	19	4	27	30	80	5.6	0.9	9.2	5.7	5.0
Strongyloidiasis	0	0	0	0	0	-	-	-	-	-
Tetanus Triatia acia	0	0	0	0	0	-	-	-	-	-
	0	0	0	0	0	-	-	-	-	-
Turatemia	0	0	0	0	0	-	-	-	-	-
i yphoid fever, case	0	U	U	4	4	-	-	-	0.8	0.3
i yphoid fever, carrier	0	U	0	U	0	-	-	-	-	-
i ypnus tever	4	U	U	U	4	1.2	-	-	-	0.3
VIDrio	1	0	0	0	1	0.3	-	-	-	0.1

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

Table O-4. Selected Notifiable Diseases SPA 4. Metro Area Los Angeles County, 2001

_		F	requency	/	Rate (Cases per 100,000) ^b					
Disease	CE	HW	NE	TOTAL	CE	HW	NE	TOTAL		
Amebiasis	2	14	4	20	0.6	2.8	1.3	1.7		
Botulism	1	0	0	1	0.3	-	-	0.1		
Brucellosis	0	0	1	1	-	-	0.3	0.1		
Campylobacteriosis	37	78	40	155	11.1	15.5	12.9	13.5		
Cholera	0	0	0	0	-	-	-	-		
Coccidioidomycosis	3	2	2	7	0.9	0.4	0.6	0.6		
Cryptosporidiosis	13	18	2	33	3.9	3.6	0.6	2.9		
Cysticercosis	3	3	1	7	0.9	0.6	0.3	0.6		
Dengue	0	1	0	1	-	0.2	-	0.1		
E. <i>coli</i> O157:H7	0	1	1	2	-	0.2	0.3	0.2		
Encephalitis	3	0	1	4	0.9	-	0.3	0.3		
Giardiasis	22	30	6	58	6.6	6.0	1.9	5.1		
Haemophilus influenzae type b	0	1	1	2	-	0.2	0.3	0.2		
Hansen's Disease (Leprosy)	0	0	0	0	-	-	-	-		
Hepatitis A	31	72	8	111	9.3	14.3	2.6	9.7		
Hepatitis B	4	6	0	10	1.2	1.2	-	0.9		
Hepatitis C	0	1	0	1	-	0.2	-	0.1		
Hepatitis unspecified	0	0	0	0	-	-	-	-		
Kawasaki syndrome	0	0	1	1	-	-	0.3	0.1		
Legionellosis	0	3	0	3	-	0.6	-	0.3		
Listeriosis, nonperinatal	1	1	1	3	0.3	0.2	0.3	0.3		
Listeriosis, perinatal	0	0	0	0	-	-	-	-		
Lyme disease	0	0	0	0	-	-	-	-		
Malaria	2	2	1	5	0.6	0.4	0.3	0.4		
Measles	0	2	0	2	-	0.4	-	0.2		
Meningitis, viral	7	6	3	16	2.1	1.2	1.0	1.4		
Meningococcal infections	2	6	2	10	0.6	1.2	0.6	0.9		
Mumps	0	1	2	3	-	0.2	0.6	0.3		
Pertussis	4	8	9	21	1.2	1.6	2.9	1.8		
Psittacosis	0	0	0	0	-	-	-	-		
Q-fever	0	1	0	1	-	0.2	-	0.1		
Relapsing fever	0	0	0	0	-	-	-	-		
Rheumatic fever, acute	0	1	0	1	-	0.2	-	0.1		
Rubella	0	0	0	0	-	-	-	-		
Salmonellosis	37	66	39	142	11.1	13.1	12.6	12.4		
Shigellosis	24	99	38	161	7.2	19.7	12.3	14.1		
Strongyloidiasis	0	0	0	0	-	-	-	-		
Tetanus	0	0	0	0	-	-	-	-		
Trichinosis	0	0	0	0	-	-	-	-		
Tularemia	0	0	0	0	-	-	-	-		
Typhoid fever, case	4	2	0	6	1.2	0.4	-	0.5		
Typhoid fever, carrier	0	1	0	1	-	0.2	-	0.1		
Typhus fever	1	0	3	4	0.3	-	1.0	0.3		
Vibrio	0	1	1	2	-	0.2	0.3	0.2		

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

Table O-5. Selected Notifiable Diseases SPA 5. West Area Los Angeles County, 2001

	Frequency	Rate (Cases per 100,000) ^b
Disease	West	West
Amebiasis	24	3.9
Botulism	0	-
Brucellosis	0	-
Campylobacteriosis	109	17.8
Cholera	0	-
Coccidioidomycosis	6	1.0
Cryptosporidiosis	5	0.8
Cysticercosis	1	0.2
Dengue	0	- · · · · · · · · · · · · · · · · · · ·
E. coli O157:H7	2	0.3
Encephalitis	1	0.2
Giardiasis	51	8.3
Haemophilus influenzae type b	0	-
Hansen's Disease (Leprosy)	0	0.0
Hepatitis A	56	9.1
Henatitis B	0	-
Henatitis C	Û	-
Henatitis unspecified	0 0	
Kawasaki syndrome	0 0	-
Legionellosis	1	0.2
Listeriosis nonnerinatal	2	0.2
Listeriosis, norperinatal	2	0.5
Listenosis, perinatai	2	- 03
Malaria	2	13
Mooslos	8	1.5
Moningitic virol	5	0.2
Moningacaccal infactions	3	0.8
Mumpe	5	0.5
Bertuesia	11	- 1 0
Peittacocio	11	1.0
C fovor	0	-
Q-level Relansing fovor	0	-
Relapsing level Bhoumatic fouer couto	0	-
Riteumatic level, acute	0	-
	0	- 11.2
Spigollogia	61	11.5
Shiyellosis	01	9.9
Tetenue	0	-
Trichingoig	0	-
Tuloromio	U	-
Turbaid favor and	U	-
Typhola lever, case	1	0.2
Typhold lever, carrier	U	-
	0	-
OTALV	2	0.3

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

Table O-6. Selected Notifiable Diseases SPA 6. South Area Los Angeles County, 2001

	Frequency						Rate (Cases per 100,000) ^b					
Disease	CN	SO	SE	sw	TOTAL		CN	SO	SE	SW	TOTAL	
Amebiasis	1	6	0	4	11		0.4	3.6	-	1.1	1.2	
Botulism	0	0	0	0	0		-	-	-	-	-	
Brucellosis	3	2	0	0	5		1.1	1.2	-	-	0.5	
Campylobacteriosis	30	25	38	40	133		11.0	15.1	24.3	11.1	13.9	
Cholera	0	0	0	0	0		-	-	-	-	-	
Coccidioidomycosis	1	1	0	3	5		0.4	0.6	-	0.8	0.5	
Cryptosporidiosis	0	0	2	4	6		-	-	1.3	1.1	0.6	
Cysticercosis	1	0	5	2	8		0.4	-	3.2	0.6	0.8	
Dengue	0	0	0	0	0		-	-	-	-	-	
E. <i>coli</i> O157:H7	1	0	0	1	2		0.4	-	-	0.3	0.2	
Encephalitis	2	0	0	1	3		0.7	-	-	0.3	0.3	
Giardiasis	14	8	7	14	43		5.1	4.8	4.5	3.9	4.5	
Haemophilus influenzae type b	1	0	1	0	2		0.4	-	0.6	-	0.2	
Hansen's Disease (Leprosy)	0	0	0	0	0		-	-	-	-	-	
Hepatitis A	18	10	9	13	50		6.6	6.1	5.7	3.6	5.2	
Hepatitis B	2	2	1	3	8		0.7	1.2	0.6	0.8	0.8	
Hepatitis C	0	0	0	0	0		-	-	-	-	-	
Hepatitis unspecified	0	0	0	0	0		-	-	-	-	-	
Kawasaki syndrome	0	0	0	0	0		-	-	-	-	-	
Legionellosis	0	0	0	1	1		-	-	-	0.3	0.1	
Listeriosis, nonperinatal	0	0	0	0	0		-	-	-	-	-	
Listeriosis, perinatal	0	0	0	1	1		-	-	-	1.2	0.4	
Lyme disease	0	0	0	0	0		-	-	-	-	-	
Malaria	2	1	0	2	5		0.7	0.6	-	0.6	0.5	
Measles	0	0	0	0	0		-	-	-	-	-	
Meningitis, viral	19	6	6	9	40		7.0	3.6	3.8	2.5	4.2	
Meningococcal infections	0	0	0	3	3		-	-	-	0.8	0.3	
Mumps	2	0	0	0	2		0.7	-	-	-	0.2	
Pertussis	1	4	3	3	11		0.4	2.4	1.9	0.8	1.2	
Psittacosis	0	0	1	0	1		-	-	0.6	-	0.1	
Q-fever	0	0	0	0	0		-	-	-	-	-	
Relapsing fever	0	0	0	0	0		-	-	-	-	-	
Rheumatic fever, acute	0	0	0	0	0		-	-	-	-	-	
Rubella	0	0	0	0	0		-	-	-	-	-	
Salmonellosis	26	24	23	57	130		9.5	14.5	14.7	15.8	13.6	
Shigellosis	31	19	11	42	103		11.3	11.5	7.0	11.7	10.8	
Strongyloidiasis	0	0	0	0	0		-	-	-	-	-	
Tetanus	0	0	0	0	0		-	-	-	-	-	
Trichinosis	0	0	0	0	0		-	-	-	-	-	
Tularemia	0	0	0	0	0		-	-	-	-	-	
Typhoid fever, case	0	0	1	0	1		-	-	0.6	-	0.1	
Typhoid fever, carrier	0	0	0	0	0		-	-	-	-	-	
Typhus fever	0	0	0	0	0		-	-	-	-	-	
VIbrio	3	0	0	1	4		1.1	-	-	0.3	0.4	

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

Table O-7. Selected Notifiable Diseases SPA 7. East Area Los Angeles County, 2001

			Free	quency			Rate (Cases per 100,000) ^b					
Disease	BF	EL	SA	₩Н	TOTAL	В	F	EL	SA	wн	TOTAL	
Amebiasis	5	8	3	4	20		1.4	4.0	0.7	1.3	1.6	
Botulism	0	0	0	0	0		-	-	-	-	-	
Brucellosis	0	1	0	0	1		-	0.5	-	-	0.1	
Campylobacteriosis	30	38	62	23	153	8	3.6	18.8	14.6	7.4	11.9	
Cholera	0	0	0	0	0		-	-	-	-	-	
Coccidioidomycosis	3	0	2	7	12	().9	-	0.5	2.2	0.9	
Cryptosporidiosis	0	0	3	3	6		-	-	0.7	1.0	0.5	
Cysticercosis	0	1	3	1	5		-	0.5	0.7	0.3	0.4	
Dengue	0	0	1	0	1		-	-	0.2	-	0.1	
E. <i>coli</i> O157:H7	2	0	0	2	4	().6	-	-	0.6	0.3	
Encephalitis	0	0	2	0	2		-	-	0.5	-	0.2	
Giardiasis	7	9	21	6	43	4	2.0	4.5	5.0	1.9	3.3	
Haemophilus influenzae type b	1	0	0	0	1	().3	-	-	-	0.1	
Hansen's Disease (Leprosy)	0	0	0	0	0		-	-	-	-	-	
Hepatitis A	15	7	16	15	53	4	1.3	3.5	3.8	4.8	4.1	
Hepatitis B	0	0	0	3	3		-	-	-	1.0	0.2	
Hepatitis C	0	0	0	0	0		-	-	-	-	-	
Hepatitis unspecified	0	0	0	0	0		-	- -	-	-	-	
	0	1	1	2	4		-	0.5	0.2	0.6	0.3	
	2	0	1	0	2	(0.0	-	-	-	0.2	
	0	0	1	2 1	3		-	-	0.2	0.0	0.2	
Listenosis, perinatai	0	0	0	0	1		-	-	-	1.4	0.5	
Malaria	2	0	0	0	2	(16	_	_		0.2	
Maaslas	2	0	0	0	2).0) 6	_	_	_	0.2	
Meningitis viral	30	4	28	g	71		3.6	20	6.6	29	5.5	
Meningococcal infections	1	0	4	1	6) 3	2.0	0.0	0.3	0.5	
Mumps	Ö	Ő	0	0	Ő		-	-	- 0.0	- 0.0		
Pertussis	2	2	2	2	8	().6	1.0	0.5	0.6	0.6	
Psittacosis	0	0	0	0	0		-	-	-	-	-	
Q-fever	Õ	Õ	Õ	Õ	0		-	-	-	-	-	
Relapsing fever	0	0	0	0	0		-	-	-	-	-	
Rheumatic fever, acute	0	0	0	0	0		-	-	-	-	-	
Rubella	0	0	0	0	0		-	-	-	-	-	
Salmonellosis	35	41	39	32	147	1().1	20.3	9.2	10.3	11.4	
Shigellosis	14	18	36	24	92	4	1.0	8.9	8.5	7.7	7.2	
Strongyloidiasis	0	0	0	0	0		-	-	-	-	-	
Tetanus	0	0	0	0	0		-	-	-	-	-	
Trichinosis	0	0	0	0	0		-	-	-	-	-	
Tularemia	0	0	0	0	0		-	-	-	-	-	
Typhoid fever, case	0	0	0	0	0		-	-	-	-	-	
Typhoid fever, carrier	0	0	0	0	0		-	-	-	-	-	
Typhus fever	0	0	0	0	0		-	-	-	-	-	
VIbrio	0	1	1	1	3		-	0.5	0.2	0.3	0.2	

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

Table O-8. Selected Notifiable Diseases SPA 8. South Bay Area Los Angeles County, 2001

_		Fr	equency	/	Rate (Cases per 100,000) ^b					
Disease	НВ	IW	то	TOTAL	НВ	IW	то	TOTAL		
Amebiasis	2	2	1	5	1.0	0.5	0.2	0.5		
Botulism	0	0	0	0	-	-	-	-		
Brucellosis	0	0	0	0	-	-	-	-		
Campylobacteriosis	61	41	56	158	31.2	10.1	12.9	15.2		
Cholera	0	0	0	0	-	-	-	-		
Coccidioidomycosis	1	5	2	8	0.5	1.2	0.5	0.8		
Cryptosporidiosis	2	4	3	9	1.0	1.0	0.7	0.9		
Cysticercosis	0	0	0	0	-	-	-	-		
Dengue	0	0	2	2	-	-	0.5	0.2		
E. coli O157:H7	1	1	5	7	0.5	0.2	1.1	0.7		
Encephalitis	2	1	4	7	1.0	0.2	0.9	0.7		
Giardiasis	14	8	13	35	7.2	2.0	3.0	3.4		
Haemophilus influenzae type b	0	0	0	0	-	-	-	-		
Hansen's Disease (Leprosy)	0	0	0	0	-	-	-	-		
Hepatitis A	13	18	13	44	6.6	4.4	3.0	4.2		
Hepatitis B	0	0	1	1	-	-	0.2	0.1		
Hepatitis C	0	0	0	0	-	-	-	-		
Hepatitis unspecified	0	0	0	0	-	-	-	-		
Kawasaki syndrome	1	0	2	3	0.5	-	0.5	0.3		
Legionellosis	2	1	2	5	1.0	0.2	0.5	0.5		
Listeriosis, nonperinatal	1	1	0	2	0.5	0.2	-	0.2		
Listeriosis, perinatal	0	0	0	0	-	-	-	-		
Lyme disease	0	0	1	1	-	-	0.2	0.1		
Malaria	2	5	2	9	1.0	1.2	0.5	0.9		
Measles	0	0	2	2	-	-	0.5	0.2		
Meningitis, viral	12	16	26	54	6.1	3.9	6.0	5.2		
Meningococcal infections	1	3	5	9	0.5	0.7	1.1	0.9		
Mumps	2	3	0	5	1.0	0.7	-	0.5		
Pertussis	3	6	0	9	1.5	1.5	-	0.9		
Psittacosis	0	0	0	0	-	-	-	-		
Q-fever	0	0	0	0	-	-	-	-		
Relapsing fever	0	0	0	0	-	-	-	-		
Rheumatic fever, acute	0	0	0	0	-	-	-	-		
Rubella	0	0	0	0	-	-	-	-		
Salmonellosis	37	41	40	118	18.9	10.1	9.2	11.4		
Shigellosis	16	32	15	63	8.2	7.9	3.4	6.1		
Strongyloidiasis	0	0	0	0	-	-	-	-		
Tetanus	0	0	0	0	-	-	-	-		
Trichinosis	0	0	0	0	-	-	-	-		
Tularemia	0	0	0	0	-	-	-	-		
Typhoid fever, case	0	0	0	0	-	-	-	-		
Typhoid fever, carrier	0	0	0	0	-	-	-	-		
Typhus fever	0	0	0	0	-	-	-	-		
Vibrio	0	1	1	2	-	0.2	0.2	0.2		

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


AMEBIASIS

CRUDE DATA		
Number of Cases	139	
Annual Incidence ^a		
LA County	1.56	
United States	N/A	
Age at Diagnosis		
Mean	34	
Median	34	
Range	2-86 years	
Case Fatality		
LA County	0.0%	
United States	N/A	



a Cases per 100,000 population.

DESCRIPTION

Amebiasis is caused by the protozoan parasite Entamoeba histolytica. Cysts shed in human feces may contaminate food or drinking water or be transferred sexually, on hands, or on fomites. Recreational waters such as lakes and pools may also serve as transmission vehicles, since cysts are relatively chlorine-resistant. Intestinal disease is often asymptomatic. Symptoms may range from acute abdominal pain, fever, chills, bloody and diarrhea to mild abdominal diarrhea discomfort with alternating with constipation. Extraintestinal infection occurs when organisms become bloodborne, leading to amebic abscesses in the liver, lungs or brain. Complications include colonic perforation. There is no vaccine. The most commonly ordered parasite test (microscopy of stool for ova and parasites) cannot distinguish *E. histolytica* from *E. dispar*, a non-pathogenic amebic species.



DISEASE ABSTRACT

- Amebiasis rates rose very slightly in all racial and most age groups, but are below the average of the previous 5 years.
- Decreasing numbers of refugees and immigrants from endemic regions or reduction in testing may explain the rate fall.
- No amebiasis outbreaks were reported in 2001.

STRATIFIED DATA

Trends: The 2001 amebiasis incidence rate and number of cases rose slightly but remained below rates and counts seen prior to 1999 (Figure 1).

Seasonality: Cases were more common in the summer, as is seen historically (Figure 2).

Age: Compared to 2000 rates, most age group rates remained stable (data not shown). An increase in cases aged 55 to 64 represents 17 cases in 2001 compared to 7 cases in 2000; for children 1 to 4 years of age, this represents 7 and 3 cases, respectively (Figure 3). White cases were older than Latinos (means of 37 and 32 years, respectively), but the difference was not significant.

Sex: The male-to-female rate ratio remains skewed toward males at 1.6:1. White cases were three times more likely to be male than were Latino cases (p=0.01).

Race/Ethnicity: Rates were unchanged for White and slightly lower for Latino cases compared to the previous 5 years (Figure 4). Race was unknown for 22% of cases (N=31). Numbers of cases in Asians and Blacks were too low to be compared.

Location: Districts with rates at least double the county mean rate were East Los Angeles (4.0 per 100,000), West (3.9), South (3.6), and Glendale (3.3).

COMMENTS

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

ADDITIONAL RESOURCES

Amebiasis - Health Information for International Travel, 2001-2002. available at: www.cdc.gov/travel/diseases/amebiasis.htm







CAMPYLOBACTERIOSIS

CRUDE DATA			
Number of Cases	1,141		
Annual Incidence ^a			
LA County	12.8		
United States	N/A		
Age at Diagnosis			
Mean	29		
Median	27		
Range	<1-93 years		
Case Fatality			
LA County	<1%		
United States	11%		



a Cases per 100,000 population.

DESCRIPTION

Campylobacteriosis is a bacterial disease that is transmitted through ingestion of contaminated foods of animal origin, especially raw or undercooked poultry, or contaminated water. Common symptoms include watery or bloody diarrhea, fever, abdominal cramps, myalgia, and nausea. Species include *C. jejuni, C. upsaliensis, C. coli* and *C. fetus*. Sequelae include Guillain-Barré syndrome and Reiter syndrome, which occur in a limited number of cases.

DISEASE ABSTRACT

- The campylobacteriosis rate decreased 7% in 2001 after a 16% increase in 2000.
- In 2000, the majority of cases were reported in Whites; in 2001 the majority of cases were reported in Latinos.
- Incidence is highest in infants and children. There was an increase seen in Latino infants.

STRATIFIED DATA

Trends: Figure 1 shows the highest incidence rate in 1996, followed by a decline in rates from 1997 to 1999. In 2000 there was a slight increase and in 2001 the downward trend continued to a rate of 12.8 per 100,000.



Seasonality: As in previous years, the number of cases increased in the spring and summer, with incidence peaking April through October (Figure 2).

Age: The previous five-year average showed the highest rate was among infants aged < 1 year and children, aged 1 - 4 years. In 2001 infants aged < 1 year had the highest rate when rates were age adjusted (Figure 3).

Sex: The male-to-female ratio was 1:0.8.

Race/Ethnicity: In 2001, the rate among Latinos (15.1) was higher than the rate among Whites (13.6). In 2000, both Whites and Latinos had increasing rates, but Whites had the higher rate. Latino infants aged < 1 year had the highest age adjusted rate in 2001. Rates for White, Asian and Black infants decreased (Figure 4). This trend was not seen in 2000. The reason for this is unknown.

Location: SPA 2 had 22% (251) of all reported cases. SPA 8 and SPA 4 each had 14% (158 and 157 respectively) of reported cases. The higher number of cases seen in districts in the western and coastal areas of LAC is consistent with previous years.

Severity of Illness: Eleven percent (122) of reported campylobacteriosis cases were hospitalized. There were two campylobacteriosis-associated deaths in 2001. Both deaths were in patients with multiple medical problems. There were four reports of Guillain-Barré syndrome (GBS) subsequent to campylobacteriosis diagnosis. One study reported that C. jejuni infection was identified in up to 41% of GBS patients.¹





Comments: In 2001,19% (214) of cases reported traveling both inside and outside the US during the incubation period. Travel may be associated with visiting countries where food safety is questionable. Travel may also be a marker for eating in restaurants more often.

There was one reported campylobacteriosis outbreak (N = 3) in 2001. This outbreak was associated with consumption of an ethnic dish consisting of intentionally undercooked chicken.

Seventy percent of all reported cases were identified as *C. jejuni*. Although 28.7 % of reported cases were not serotyped in 2001, one study reported that *C. upsaliensis* was the second most frequently identified species in LAC in 1998.² This species is commonly associated with cats and dogs. The serotypes *C. coli, C.fetus, C. gracilis and C. laridis* comprised the remaining 2 % of reported cases.

Prevention: All foodstuffs derived from animal sources should be thoroughly cooked, particularly poultry. Cross contamination may be avoided by making sure utensils, counter tops, cutting boards and sponges are cleaned or do not come in contact with raw poultry or meat. Hands must be thoroughly washed before, during and after food preparation. The juices from

raw poultry or meat must not be allowed to drip on other foods in the refrigerator or in the shopping cart.

REFERENCES

- 1. Hahn, A. Guillan-Barre syndrome. Lancet 1998;352:9128.
- 2. Labarca, J, Sturgeon, J, Borenstein, L, et al. *Campylobacter upsaliensis*: another pathogen for consideration in the United States. Clin Inf Dis 2002;34:e59-60.

ADDITIONAL RESOURCES

Disease information is available at: www.cdc.gov/ncidod/abmd/diseaseinfo/campylobacter_g.htm

Information about this disease in LAC is available at: www.lapublichealth.org



COCCIDIOIDON	IYCOSIS
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CRUDE DATA		
Number of Cases	58	
Annual Incidence ^a		
LA County	0.7	
California ^b	4.4	
United States	1.4	
Age at Diagnosis		
Mean	46	
Median	44	
Range	2-84 years	
Case Fatality		
LA County	7.0%	
United States	N/A	



a Cases per 100,000 population.

b California Department of Health Services Surveillance and Statistics Section.

DESCRIPTION

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

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



DISEASE ABSTRACT

- 2001 incidence rate for coccidioidomycosis increased from last year, which was at its lowest point in ten years in LAC.
- Cost in terms of disease severity and hospitalization was substantial. The case fatality rate
 was lower although the incidence of coccidioidomycosis was greater than last year. Adults,
 males, Blacks, and residents of the West Valley, Antelope Valley and San Fernando Valley
 are at higher risk for disease.

STRATIFIED DATA

Trends: The 2001 incidence rate increased to 0.65 cases per 100,000 population from a tenyear low of 0.41 observed in 2000 (Figure 1).

Seasonality: In 2001, the highest number of cases per month was observed in January (9 cases), October (8 cases), June (6 cases), and September (6 cases). For more than half of the year, the number of cases per month was above the previous five-year average (Figure 2). Although not reflected in LAC's cases, cases commonly occur in the summer after a rainy winter or spring, especially after wind and dust storms. Perhaps because of LAC's temperate climate, the fluctuation of cases per month is not great.



Age: For 2001, males once again have the highest incidence in almost all age groups. The greatest numbers of cases reported were in persons aged 15-34 and 35-44 years, followed by the 45-54 year old age group (Figure 3). There were only two cases under 18 years of age; one was a child under 5 years.

Sex: The male-to-female rate ratio was 4.5:1. The mean age for males was 47 years and for females it was 41. The gender difference is likely due to occupational and recreational dust

exposure of males although this is not clearly evident from the information collected (Figure 3). No female cases reported being pregnant.

Race/Ethnicity: A higher incidence rate was observed among Blacks (1.72 cases per 100,000) compared to the other groups, although the rates were unstable due to small numbers. Latinos had the greatest number of cases with 22 (Figure 4).

Location: West Valley District had the highest number of cases (10) followed by Antelope Valley (6) and San Fernando (6).

Travel: Of the 29 cases where travel was known, 15 reported travel within four weeks before onset of illness: 8 traveled within California



(Sacramento, San Joaquin Valley, Palm Springs, San Diego,) and 7 traveled outside California to Arizona, Guatemala, New Mexico, Mexico, and South Carolina. Coccidioidomycosis is known to be endemic in all these areas except South Carolina. The case that traveled to South Carolina also resided in an endemic area.

Immunosuppression: Of 13 cases with known immunosuppression, 5 cases had HIV, 3 were diabetics, 3 had a malignancy, and 2 had other diseases (chronic bronchitis and multiple sclerosis). One HIV case also had diabetes and another had syphilis.

Severity of Disease: Of the cases reported, sites of infection were reported as 58% primary pulmonary, 9% disseminated, 9% skin, 9% other (abdominal abscess, sternal mass, knee, and lymph nodes), and 2% meningitis; in 13% of the cases infection site was unknown (Figure 5). Sixty-seven percent (39) of cases were culture-confirmed and the majority remaining were diagnosed by serological or molecular evidence. Of the 47 cases where information was available, 87% (41) were hospitalized. Four cases died. The 2001 case fatality rate (7%) decreased 56% from last year.



COMMENTS

A documented peak occurred in 1992 to 1994 probably as a result of a 5 year drought (1986-1990) with heavy rainfall in 1991, 1992, and 1993. It appears that the organisms competing with *C. immitis* decrease in the soil during a drought and, after a heavy rain, the dormant *C. immitis* multiplies to a higher density because of lack of competing organisms. Also, there was increased media attention and reporting because of a Simi Valley outbreak and increased dust exposure related to the Northridge earthquake in 1994.

PREVENTION

There is no safe and effective vaccine or drug to prevent coccidioidomycosis; prevention lies mainly in dust control such as planting grass in dusty areas, putting oil on roadways, wetting down soil, air conditioning homes, and wearing masks or respirators. Other options may be to warn individuals who are at high risk for severe disease not to travel to endemic areas when conditions (dusty) are most dangerous for exposure.

ADDITIONAL RESOURCES

Chin, J. (Ed.). (2000). <u>Control of Communicable Diseases Manual</u>. Washington, DC: United Book Press, Inc.

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

Kirkland TN, Fierer J. Coccidioidomycosis: A reemerging infectious disease. Emerg Infect Dis 1996 Jul-Sep;2(3):192-9.

CRYP1	FOSPORI	DIOSIS
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CRUDE DATA		
Number of Cases	77	
LA County	0.9	
United States	1.3	
Age at Diagnosis		
Mean	39	
Median	37	
Range	4-75	
Case Fatality		
LA County	0.0%	
United States	N/A	



a Cases per 100,000 population.

DESCRIPTION

Cryptosporidiosis is transmitted by the fecaloral route (for example, swallowing contaminated recreational or untreated water, and eating raw or undercooked contaminated food) by ingestion of cysts of the parasite Cryptosporidium parvum. The usual incubation period is 2-10 days with typical symptoms of watery diarrhea, abdominal cramps, and lowgrade fever; however, asymptomatic infection is also common. Symptoms last up to 2 weeks in healthy individuals. Those who have a weakened immune system may experience prolonged illness. Immunocompromised individuals (e.g., HIV/AIDS patients, cancer patients, transplant patients), young children and pregnant women are at risk for more severe illness.



DISEASE ABSTRACT

- The incidence rate has been dropping since 1997.
- HIV positivity is the most common identified risk factor for cryptosporidiosis. Cryptosporidiosis has been an AIDS-defining disease since 1983, and reported cases have fallen since the advent of highly active antiretroviral therapy.

STRATIFIED DATA

Trends: The rate of cryptosporidiosis (0.9 cases per 100,000) rose slightly in 2001 (Figure 1).

Seasonality: In 2001, there was a peak in August compared to the previous 5 year average peak seen in September (Figure 2).

Age: The majority of cases was in those aged 35-44 years.

Sex: The male-to-female rate ratio was 8.5:1, This is due to the high rate of cryptosporidiosis in men who have sex with men.

Race/Ethnicity: Whites had the highest number of cases (34) and the highest rate (Figures 4, 5). Blacks and Latinos had the same rate of disease. Only 1 case occurred among Asians. This variable was unknown for 10 cases (13%).

Location: Location information was available for 75 cases. Twenty-five percent of cases (19) lived in Hollywood-Wilshire Health District, followed by 19% (14) in Central Health District.

COMMENTS

Data on HIV status was available for 55 cases. Of these, 80% (44) reported being HIV positive. Cryptosporidiosis has been an AIDS-defining disease since 1983 and reported cases have fallen since the advent of highly active antiretroviral therapy.

Of those cases where information was available. immigration (recent or otherwise) and animal contact were the two highest risk factors besides HIV status (33%). Twenty percent of cases had a history of travel. Outdoor activities camping, hiking, swimming or fishing were reported among 20% of cases. A contaminated water supply was indicated in 12% of cases where information was available. Further details such as type of animal or nature of animal exposure, swimming location, country visited, or date of immigration were not provided for most cases. has not been an There outbreak of cryptosporidiosis in LAC since 1988. The source of that outbreak was contaminated swimming pool water (Sorvillo et al).







ADDITIONAL RESOURCES

Sorvillo FJ, Fujioka K, Nahlen B, Tormey MP, Kebabjian R, Mascola L. Swimming-associated cryptosporidiosis. Am J Public Health 1992;82(5):742-4.

www.cdc.gov/ncidod/dpd/parasites/cryptosporidiosis/default.htm

www.lapublichealth.org/acd/procs/b73/b73index.htm

CYSTICERCOSIS

CRUDE DATA			
Number of Cases	37		
Annual Incidence ^a			
LA County	0.4		
United States	N/A		
Age at Diagnosis			
Mean	41		
Median	40		
Range	18-71 years		
Case Fatality			
LA County	10.8%		
United States	N/A		



a Cases per 100,000 population.

DESCRIPTION

Cysticercosis is infection with the larval form of the pork tapeworm *Taenia solium*. It is caused by ingestion of tapeworm eggs excreted in human feces. The larvae that hatch from the eggs are called cysticerci and they may invade any organ; the most serious manifestation is neurocysticercosis, infection in the central nervous system, including the brain, meninges, spinal column, and eye. Pork tapeworm – taeniasis – infection is caused by consuming uncooked pork infested with viable larvae.

DISEASE ABSTRACT

- Most cases occur in persons born in foreign countries where the pork tapeworm is endemic.
- All reported cases this year were born in Latin American countries, principally Mexico.

STRATIFIED DATA

Trends: The rate of reported disease has remained stable for the past five years. However, previous studies have demonstrated extensive under reporting of this disease. The illness is lifelong and may lead to multiple medical examinations and hospitalizations.

Seasonality: There is no seasonality to disease onset.

Age: Cases ranged in age from 18 to 71 years; the greatest number of cases was in the age group 15-34, while the highest rates were seen in the middle age groups (Figure 2). Though rates



among adult age groups appears to have doubled compared to recent years, the number of cases represented is small. Most acute infections are asymptomatic; symptoms develop months or years later due to the inflammatory response made against the larvae or the resulting space-occupying lesions in the central nervous system. Thus fewer cases are diagnosed in children because of the typical latency between infection and development of clinical symptoms leading to diagnosis.

Sex: Of the 37 reported cases, 19 (51%) were male; the male:female rate ratio was 1:1.

Race/Ethnicity: All 37 cases (100%) were Latino (0.9 cases per 100,000). Thirty-two cases (87%) were foreign born: 22 were born in Mexico, 5 in El Salvador, 3 in Guatemala, and 1 each in Ecuador and Nicaragua.

Location: Since the incubation period may be decades long, it is generally not possible to pinpoint the time or place of infection of cases born or traveling frequently outside the US.

Comment: Epidemiologic and clinical information was obtained on 30 cases (81%).

Symptoms: All reported cases were neurocysticercosis. The most common presenting symptoms were headache (65%) and seizures (51%) (Figure 3).

Diagnosis: Most cases were diagnosed by either MR or CT scan; 29 of 29 scans performed were interpreted as consistent with the disease. While such scans are not usually definitive, the diagnosis of cysticercosis can be made with reasonable accuracy given typical appearance, number, and location of brain lesions. Serology may be helpful, especially in individuals without a history of exposure in endemic parts of the world.



Serologic tests were significant in 13 of 14 cases where obtained. Biopsy is the definitive method of diagnosis; it was positive in the only case for which it was performed. One of the four fatal cases was diagnosed post mortem; the others died having been diagnosed and treated previously.

Public Health Impact: Stool examinations for ova and parasites (O&P) were performed on only 20 of 30 cases (67%) from which interviews were obtained. Thirteen cases submitted three or more specimens; one pork tapeworm carrier was identified. Since tapeworm carriers are capable of autoinfection, patients with radiologic evidence of recent (active) cysticercal lesions may carry a tapeworm in their own gut. While treatment successfully eradicates intestinal tapeworm and negates the need to screen the patient further, O&P testing may provide information critical to identifying the source of infection.

ADDITIONAL RESOURCES

CDC Cysticercosis Fact Sheet available at: www.cdc.gov/ncidod/dpd/parasites/cysticercosis/factsht_cysticercosis.htm

Neurocysticercosis in Radiographically Imaged Seizure Patients in US Emergency Departments. Emerg Infec Dis 2002;8:608-13. www.cdc.gov/ncidod/EID/vol8no6/01-0377.htm

Review of Cysticercosis. Neurosurg Focus 2002;12(6).

ENCEPHALITIS

CRUDE DATA		
Number of Cases	41	
Annual Incidence ^a		
LA County	0.5	
California	N/A	
United States	N/A	
Age at Diagnosis		
Mean	26	
Median	19	
Range	0-90 years	
Case Fatality		
LA County ^b	22.0%	
United States	N/A	



a Cases per 100,000 population.

b Excludes AIDS encephalopathy cases.

DESCRIPTION

Encephalitis, an inflammation of the brain, causes headache, stiff neck, fever and altered mental status. It can result from infection with a number of different agents including viral, parasitic, rickettsial, bacterial and chemical. Public health surveillance is limited to cases of suspected or confirmed viral etiology, and includes primary and postinfectious encephalitis. Of special concern is arboviral (mosquito borne) encephalitis, which can be prevented by personal protection and mosquito control. The etiologies of cases with known cause reported in 2001 are shown in Figure 1.



DISEASE ABSTRACT

- The 2001 incidence of viral encephalitis, 0.46 cases per 100,000 population, remains in the range seen during non-epidemic years (Figure 1).
- 17 of the 41 cases (44%) were in children less than 14 years of age, 15 cases were in those from 15 to 44 and 11 cases occurred in those more than 55 years.
- There were 24 male cases (59%).
- Cases of encephalitis occurred throughout LAC; SPA 2 had 13 cases, followed by SPA 3 with 11 and SPA 8 with 7 cases
- The etiology of encephalitis was not identified in 70% of cases.

COMMENTS

Although the Public Health Laboratory provides free testing of clinical samples for arboviral encephalitis, and the California DHS has an encephalitis study that provides free testing for numerous agents, few samples are submitted, and the etiologic agent for most cases is not identified. In 2001, the etiology was unknown for 70% of reported cases. Determining the etiology of encephalitis allows public health to follow disease trends, to notify the community of increased disease risk and to implement prevention efforts.

Of particular public health concern in LAC are the arthropod-borne (arboviral) encephalitides, especially those due to St. Louis encephalitis (SLE) and Western equine encephalitis (WEE) viruses. Since 1985, sporadic cases of SLE have been reported, following an outbreak of 16 cases in 1984. The last confirmed SLE case in LAC was in 1997. The potential for another SLE outbreak exists, as sporadic cases in previous years and identification of SLE in sentinel animal populations indicate that the virus is now endemic in LAC. Beginning in 2001, surveillance has included West Nile (WN) virus, which also can cause encephalitis, especially in the elderly. The annual mosquito-borne encephalitis surveillance program consists of surveillance for equine cases of WEE and WN viral infections, monitoring of mosquito populations, laboratory testing of mosquitoes for WEE, SLE and WN viruses, and twice monthly testing of sentinel chicken flocks for SLE, WEE and WN virus seroconversion. In addition, in 2001, the LAC PHL began to develop human testing for West Nile virus.

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

Future Directions: Research is underway in development of a WNV vaccine for humans. No human vaccine is available for SLE and WEE. Since SLE and WN are both flaviviruses, vaccination or infection with WNV may offer cross protective antibodies for SLE.

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

ADDITIONAL RESOURCES

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

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

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

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

Meningitis Foundation of America, Inc. 7155 Shadeland Station, Suite 190 Indianapolis, IN 46256-3922 <u>support@musa.org</u> or <u>www.musa.org</u> Tel: 800-668-1129 or 317-595-6383; FAX 317-595-6370

National Institute of Allergy and Infectious Diseases (NIAID) National Institutes of Health 31 Center Drive, Rm. 7A50 MSC 2520 Bethesda, MD 20892-2520 www.niad.hih.gov or Tel: 301-496-5717

ESCHERICHIA COLI 0157:H7 / HEMOLYTIC UREMIC SYNDROME

CRUDE DATA		
Number of Cases	31	
Annual Incidence ^a		
LA County	0.4	
California ^b	0.7	
United States ^b	1.2	
Age at Diagnosis		
Mean	26	
Median	15	
Range	3-88 years	
Case Fatality		
LA County	0.0%	
United States	N/A	



a Cases per 100,000 population.

^b Data via the National Electronic Telecommunications System for Surveillance.

DESCRIPTION

Escherichia coli O157:H7, a gram-negative bacillus, is a specific serotype of the Shiga-toxin producing class of *E. coli* (STEC). Shigatoxins cause abdominal cramps and watery diarrhea developing into bloody diarrhea; fever is uncommon. The common modes of transmission are foodborne (e.g. undercooked ground beef, unpasteurized juice, raw milk) and person-to-person (e.g. day-care settings). There have been outbreaks associated with recreational water exposure.

Children under five are at highest risk for hemolytic uremic syndrome (HUS), a clinical complication which consists of hemolytic anemia, thrombocytopenia, and kidney failure. Adults may get thrombotic thrombocytopenic purpura (TTP) after infection.

DISEASE ABSTRACT

- In 2001, LAC saw the largest number of *E. coli* O157:H7 cases within the last six years. The reason for this increase is not known.
- No outbreaks were identified in LAC during 2001.

STRATIFIED DATA

Trends: In 2001, there was a 17 % increase in the rate of *E. coli* O157:H7 cases. This increase is consistent with a trend of increasing incidence that started in 1996 but was interrupted in 1999. The reason for the drop in reported cases in 1999 is not known.

Seasonality: In 2001, the greatest number of cases was again observed during the summer with a peak in July (8 cases). This could be due to increased consumption of ground beef during

barbeques, cooking involving hamburger, travel and recreational water exposure.

Age: The greatest number of cases was seen in persons aged 5 -14 years. The number of cases in this age group and persons aged15-34 years and 45 to 54 years were higher than the five-year average.

Sex: The male to female rate ratio was 1.3:1.

Race/Ethnicity: The highest percentage of cases was seen among Whites (61%), followed by Latinos (19%), and Blacks (19%). There were no cases reported among Asians in 2001.



Location: SPA 8 had the highest rate at 0.7 per 100,000 population. SPA 3 and SPA 5 had rates of 0.4 per 100,000 population and 0.3 per 100,000 population respectively.

COMMENTS

E. coli O157:H7 was first recognized as an important human pathogen causing foodborne illness in 1982. In 1994, LAC requested laboratories and health care providers to voluntarily report suspected *E. coli* O157:H7 cases. Mandatory reporting of *E. coli* O157:H7 cases in California was instituted in July 1995.

In 2001, in LAC, there was one case with confirmed *E. coli* O157:H7 and HUS. There was one case with confirmed *E. coli* O157:H7 and TTP. There were five cases of HUS and two cases of HUS and TTP without confirmation of *E. coli* O157:H7.

Reported cases had symptoms of diarrhea (100%), abdominal cramps (97%), bloody diarrhea (84%), nausea (68%), vomiting (65%), and fever (48%; mean temperature was 101.4). One case was on antibiotics the week prior to onset. Fifty-five percent of reported cases were hospitalized. There were no deaths.

There were no outbreaks detected within LAC. No cases were associated with outbreaks in other jurisdictions.

Collaborative efforts among physicians, laboratories and the health department are important for enhancement of surveillance activities. Physicians should consider *E. coli*



O157:H7 in their diagnoses by asking about consumption of high-risk foods, attendance at daycare centers or farms, and exposure to other individuals with diarrhea. It is important that physicians request testing for *E. coli* O157:H7 on all bloody stools. Lab analysis through PulseNet has been notable in detecting clusters of *E. coli* O157:H7. PulseNet is a nationwide network that performs PFGE or "DNA fingerprinting" of foodborne bacteria. This network permits rapid comparison of the fingerprint patterns to identify clusters and enhance outbreak investigation.

PREVENTION

The public needs increased education regarding food handling practices, proper hygiene and high-risk foods. To avoid infection, it is recommended to cook beef products thoroughly, drink only treated water, and avoid swallowing water during swimming or wading. Collection of detailed food histories and strengthening of national food processing regulations to decrease contamination should be emphasized.

ADDITIONAL RESOURCES

Information from the Foodborne and Diarrheal Diseases Branch of the CDC is available at: www.cdc.gov/ncidod/dbmd/foodborne.htm

Information about outbreaks (nationwide) is available from the Outbreak Response and Surveillance Unit of the CDC at: www.cdc.gov/ncidod/dbmd/outbreak

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

Information from Center for Food Safety and Applied Nutrition is available at: <u>www.vm.cfsan.gov/list.html</u>

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

GIARDIASIS

CRUDE DATA		
446		
5.0		
N/A		
24		
19		
0-86 years		
0.0%		
N/A		



a Cases per 100,000 population.

DESCRIPTION

Giardiasis is an intestinal infection caused by the zoonotic protozoan parasite Giardia intestinalis (previously G. lamblia). Giardia cysts shed in animal or human feces may contaminate food or drinking water or be transferred on hands or fomites; recreational waters such as lakes and pools may also serve as vehicles of transmission. The disease is usually asymptomatic. Symptoms can include chronic diarrhea, bloating, cramps, fatigue, and weight loss. Complications are rare, but may include malabsorption of fats and fat-soluble vitamins. Children in day care represent a reservoir of disease in developed countries. There is no vaccine.



DISEASE ABSTRACT

- Chiefly a pediatric disease; rates fell in all categories analyzed.
- Significant numbers of cases occur among adults.
- Risk factors are not summarized routinely.

STRATIFIED DATA

Trends: The annual rate and number of reported cases have fallen for more than ten years, with the lowest rate on record recorded in 2001 (Figure 1). Rates fell in all age group, sex, and race/ethnic categories.

Seasonality: Rates are typically higher in the summer, consistent with increased recreational exposure to pools, lakes, and similar water venues (Figure 2).

Age: The age-specific incidence of giardiasis was greatest in children aged 1-4 years (14 per 100,000) followed by children aged 5-14 years (8.2 per 100,000) (Figure 3). Among Whites, there was no age difference between male and female cases; however, among Latino cases females were significantly older than males (p=0.02).

Sex: The male-to-female rate ratio was 1.3:1.

Race/Ethnicity: As in past years, rates for Latinos and Whites were substantially higher than those of Asians and Blacks (Figure 4). White cases were more likely to be male, compared to Latino cases.

Location: The rate reported by Glendale District (16.2 per 100,000) was nearly twice as great as the next highest district rate, West District, at 8.3 per 100,000. Glendale District cases did not differ by age group or sex from all other districts, but were more likely to be White, a reflection of that district's makeup.

COMMENTS

There were no outbreaks reported.

ADDITIONAL RESOURCES



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

Surveillance for Waterborne-Disease Outbreaks -- United States, 1997-1998. MMWR May 26, 2000/49(SS04):1-35. Available at: www.cdc.gov/epo/mmwr/review/mmwrhtml/ss4904a1.htm





HAEMOPHILUS INFLUENZAE INVASIVE DISEASE

CRUDE DATA		
Number of Cases	95	
Annual Incidence ^a		
LA County	1.1	
California ^b	0.2	
United States	0.5	
Age at Diagnosis		
Mean	50	
Median	53	
Range	0-95 years	
Case Fatality		
LA County	6.0%	
United States	N/A	

^a Cases per 100,000 population.

b Cases per 100,000 persons, aged less than 30 years. In California,

H. influenzae among persons > 29 years of age is not reportable.

DESCRIPTION

Haemophilus influenzae (H. influenzae) is a gram-negative coccobacillus that can cause both invasive and non-invasive disease. H. influenzae invasive disease includes meningitis, sepsis, pneumonia, cellulitis, and septic arthritis. The disease primarily affects infants and the elderly, as well as immunocompromised individuals and those who have abnormal splenic function. H. influenzae can be transmitted by respiratory secretions of individuals colonized with the organism. There are six encapsulated, typable





strains (a-f) and unencapsulated, nontypable strains of *H. influenzae*. Prior to the introduction of the H. *influenzae* type b (Hib) conjugate vaccine in 1990, most cases of invasive disease in children were caused by type b. *H. influenzae* type b is the only serotype that is vaccine-preventable.

DISEASE ABSTRACT

- The widespread use of the Hib vaccine since 1990 has dramatically decreased the incidence of *H. influenzae* type b disease in LAC.
- The epidemiology of H. *influenzae* invasive disease is now being shaped by non-b and unknown serotypes.

Table 1: H. <i>Influenzae</i> Crude Date by Serotype, 2001			
	В	Non-b	Unknown type
Number of Cases	6	64	25
LAC Incidence	N/A	0.72	0.28
Age at Onset			
Mean	43	48	59
Median	47	50	63
Range	Birth - 81	Birth - 95	Birth - 93
LAC Case Fatality	0	9%	0

Table 1:	: H .	influenzae	Crude	Date	by	Ser	otype	, <mark>200</mark> 1	
							-		

IMMUNIZATION RECOMMENDATIONS

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

STRATIFIED DATA

Seasonality: All Hib cases and more than half of non-Hib cases (58%) occurred from January to May; there was an additional Fall peak. This is consistent with the bimodal temporal pattern of disease noted in the US.

Sex: The male-to-female ratio of non-Hib and Hib cases was 1.2:1 and 2:1, respectively.

Age: The number of cases by age follows the trend of previous years except for the 45-54 vear age group, where an increase in non-Hib cases was evidenced in 2001. The 65+ age group remains the most affected by non-Hib invasive disease over the last six years (Figure 5). The majority of non-Hib cases in 2001 were individuals older than 45 years (n=34, 53%), with 61% of the cases identified as a non-typeable serotype. Four of the six Hib cases were older than 24 years. Of the 25 cases with unknown serotype, 23 (92%) were over the age of 30 and were not actively investigated for serotype as detailed in LAC's priority investigation criteria.

Race/Ethnicity: In cases where the race/ethnicity was known, the majority of Hib and non-Hib cases were reported among Latinos (n=3, n=19, respectively) followed by Whites (n=1, n=10, respectively). (See Figure 6.)



Location: Hib cases were not clustered.

COMMENTS

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

Rates of invasive Hib disease in children have decreased to extremely low levels since Hib vaccines became available. None of the cases were in infants too young to have been immunized or in children with medical conditions, which might predispose them to Hib disease.

Case Fatalities: There were six fatalities among H. influenzae cases: Five were due to non-typable strains and the other case was typed as H. *aegyptius*. Five cases had sepsis and one case had pneumonia. All but one case was over the age of 34 years and males accounted for four of the total case fatalities. Two of the fatalities were Latino, two were White, and two were of unknown race. The residences of all the cases were uniformly distributed throughout LAC.

ADDITIONAL RESOURCES

Information about immunization is available through the National Immunization Program at: <u>www.cdc.gov/nip</u> and the Immunization Action Coalition at: <u>www.immunize.org</u>





Information specific the LAC is available from the LAC DHS Immunization Program at: <u>www.lapublichealth.org/ip</u> and the LAC DHS Acute Communicable Disease Control Unit at: <u>www.lapublichealth.org/acd/procs/b73/b73index.htm</u>
HEPATITIS A

CRUDE DATA		
Number of Cases	542	
Annual Incidence ^a		
LA County	6.1	
California	7.9	
United States	3.7	
Age at Diagnosis		
Mean	33	
Median	32	
Range	1-89 years	
Case Fatality		
LA County	<1%	
United States	N/A	



a Cases per 100,000 population.

DESCRIPTION

Hepatitis A virus (HAV), a RNA-virus of the Picornaviridae family, is a vaccine-preventable disease usually transmitted by fecal-oral route, person-to-person, or through vehicles such as food. Signs and symptoms of HAV include fever, malaise, dark urine, anorexia, nausea, and abdominal discomfort, followed by jaundice. Many cases, especially in children, are mild or asymptomatic. Sexual and household contacts of HAV-infected persons are at increased risk for getting the disease.



DISEASE ABSTRACT

- The annual incidence rate of HAV in LAC shows a dramatic decrease in 2001.
- With the exception of a decrease in Latinos from 5-14 years, age, race, and gender characteristics were similar to the last five years.
- More cases occurred in summer and fewer cases in winter.
- Hospitalization rates were highest among children and young adults.

STRATIFIED DATA

Trends: The rate of HAV in LAC has remained steady, around 9 per 100,000, since 1998. From 1993-1997, it had ranged between 10-15 per 100,000 (Figure 3). In 2001, there were 542 cases reported.

Seasonality: Historically, there is an increase of HAV cases in the summer and decrease in the winter. This was observed again in 2001 (Figure 2).

Age: The overall mean age for HAV cases in LAC 2001 was 33 years. The mean age differed significantly by race and ethnic groups. The mean age for Latinos was 20



years while, White, Asian and Black cases had mean ages of 40, 48, and 46 years, respectively. Overall race mean remained similar compared to 2000. The age specific rate remained highest in children age 5-14 years with a rate of 8.0 per 100,000 (Figure 3).

Sex: The overall HAV male-to-female rate ratio was 2:1. Among Latino cases, the male-to-female rate ratio was 1.5:1, while among White, Asian, and Black cases, incidence rates ratios were higher among males, at 3:1, 2:1, and 3:1, respectively.

Race/Ethnicity: Overall hepatitis A crude rate decreased for all ethnic groups in 2001(n=542). The highest rate in 2001 was among Whites (6.7 per 100,000), followed by Latinos (5.3). The rates for Asian (3.4) and Blacks (3.4) were remained lower (Figure 4).

Location: Map 5 shows district-specific HAV rates for 2001. The highest rate was the Hollywood-Wilshire district (14.3 cases per 100,000 population), closely followed by Central (9.3), Burke (9.1), East Valley (9.0), and Glendale (8.7). Looking at distribution by Service Planning Area (SPA, Figure 5) SPAS 4, and 5 have the highest rates (9.7 and 9.1 per 100,000, respectively), while SPAs 3,7, and 8 have rates significantly lower than the county average.



Severity of Illness: Among all HAV cases in 2001, there were three fatalities (case-fatality rate=0.5%) aged 15-62 years. Over half (66%) reported jaundice and 10% were hospitalized for their illness. Hospitalization was most prevalent among children and young adults–with increased liver enzymes and jaundice reported by over 50% who were hospitalized.

Risk Factors: Recent travel (n=136, 25%) outside of the US was the most common risk factor reported in 2001, followed by MSM (18.6%), eating raw shellfish (11.8%), and being a contact to case (10.5%) while 40% did not report any risk factor. Among travelers, Latin-American (South-Central America) destinations (74%) were the most frequently cited.

COMMENTS

There was a significant decrease in the number of cases of HAV reported in LAC since 1997. This decrease may be due to a cyclic nature of Hepatitis A. Other potential reasons for the decrease may be the result of LAC Department of Health Service following the Advisory Council on Immunization Practices (ACIP) recommendation of HAV vaccine for children, greater public awareness or improved hygiene and food sanitation. Under reporting and underdiagnosis by physicians cannot be excluded as a reason for the decrease.

HAV is a mandated laboratory reportable disease in LAC. The 542 HAV cases reported in 2001 were confirmed with a laboratory test for the IgM antibody to HAV, which indicates acute infection. Studies have shown that many children who acquired HAV were asymptomatic and not tested for HAV-IgM. Even when these children's laboratory results were confirmed IgM positive, many private health care providers and laboratories may not report HAV cases to county health officials. Therefore, support and encouragement for physician reporting and compliance with the ACIP recommendations should continue.

Most cases of HAV result from person-toperson transmission during community-wide outbreaks in areas with high and intermediate rates of HAV. In LAC, there were no outbreaks of HAV reported in 2001.

PREVENTION



5

Recent travel

*Includes cases identifying multiple risks.

Eating raw shellfish

10

11.8

15

Percent

18.6

20

Contacts

MSM/Bisexual

25

30

25

14

12

10

8

6

4

2

0

Cases per 100,000

Risk I

0

Figure 5 Hepatitis A Rates

by Planning Area

LAC, 2001

In LAC, most infections result from international travel, contact with a household member or sexual partner who has HAV. Casual contact, such as that in the office, factory, or school setting, does not spread the virus. Good personal hygiene and proper sanitation can prevent HAV. Immune globulin is recommended for certain short-term pre-exposure situations and post-exposure prophylaxis.

Since 1995, vaccines have been available for the permanent prevention of HAV infection in persons aged 2 years and older. In 1999, the ACIP recommended universal childhood vaccination in states and communities (including LAC) with rates equal to or greater than twice the national average (20 cases per 100,000) during 1987-1997. LAC began providing the vaccine to children aged 2-18 since August 1999. Over the past year, there was a legislative effort to require HAV immunization for all children entering kindergarten and preschool in California. Although it was not successful, it is possible that such a law will be enacted in the future.

Widespread post-exposure prophylaxis with immune globulin is used to control outbreaks in LAC. Since HAV vaccination has become available and in more routine use, it has been recommended by ACIP (CDC, 1999) that outbreaks of HAV could be effectively controlled through vaccine use, leading to a sustained reduction in disease incidence.

ADDITIONAL RESOURCES

B-73 Communicable Diseases Control, A Manual of Departmental Rules, Regulations and Control Procedures at: <u>www.lapublichealth.org/acd/procs/manual.htm</u>

General information about hepatitis is available at: www.cdc.gov/ncidod/diseases/hepatitis/slideset/bibliography.htm

www.cdc.gov/ncidod/diseases/hepatitis/a/index.htm



HEPATITIS B, ACUTE (NON-PERINATAL)

CRUDE DATA		Figure 1
Number of Cases Annual Incidence ^a	44	Acute Hepatitis B Incidence Rates by Year LAC and US. 1992 - 2001
LA County	0.5	14
United States	N/A	8 12 1
Age at Diagnosis		
Mean	39	
Median	38	
Range	18-73 years	8 2
Case Fatality		0
LA County	0.0%	Year
United States	N/A	LACUS

a Cases per 100,000 population.

DESCRIPTION

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

DISEASE ABSTRACT

- For a full discussion of perinatal HBV cases, see the following report describing perinatal HBV. The number of non-perinatal acute cases for 2001 was 44, which was a 32% decrease from the 65 cases in 2000.
- All acute cases were adults aged 18 years or older and the majority of cases were young, adult males.
- Cases have been decreasing among all age groups since 2000.
- Having multiple partners, predominately among men who have sex with men



(MSM), remains the most frequently identified risk factor (Figure 5). Nearly half of all acute cases deny all risk factors.

STRATIFIED DATA

Seasonality: None

Age: Cases ranged in age from 18 to 73, with a median age of 38. An increase was seen in cases among adults aged 35-44 (Figure 3). Adults aged 18-44 accounted for 37% of cases.

Sex: The male-to-female rate ratio was 2.3:1. The number of cases in males exceeded those in females in all ethnic groups except in Asians.

Race/Ethnicity: The highest rates were seen in Blacks (1.23 per 100,000) followed by Asians (.7 per 100,000) and Whites (.55 per 100,000), respectively (Figure 4).

Location: SPA 4 (Metro) and SPA 2 (San Fernando) had the most cases (n=9), respectively, followed by SPA 3 (San Gabriel, n=8), SPA 8 (South Bay/Long Beach, n=6), SPA 1 (Antelope Valley, n=5), SPA 6 (South Area, n=4), and SPA 7 (East LA Area, n=3). The West Area (SPA 5) had no reported cases.

COMMENTS

The substantial decrease in the number of nonperinatal acute hepatitis B cases in 2001 is likely attributable to the changes in the criteria for investigation and classification rather than a true reduction in infection. Surveillance for hepatitis B is passive and dependent solely upon reports from providers and laboratories. Additional information is obtained through patient interview and further investigation. Only when a case report meets both the clinical case definition and is laboratory confirmed can it then be diagnosed as an acute case. However, the majority of these case reports do not provide supporting clinical or demographic information, thus presenting difficulties for public health follow-up.

Decreasing rates of acute hepatitis B since 1992 in those under age 15 suggest that the strategy to reduce hepatitis B among infants and children through prophylaxis of newborns of chronic carrier mothers and universal hepatitis B immunization of all infants is succeeding.

Excluding perinatal cases, in 2001, there were



44 cases designated as acute hepatitis B following investigation. All were aged 18 years or older (see perinatal hepatitis B report for hepatitis B in infants). Based on crude frequencies of reported risk factors by both men and women, MSM and people with multiple sexual partners continue to be at greatest risk for hepatitis B; thus, preventive efforts should continue to focus on these high risk populations.

Health care workers are also at substantial occupational risk of acquiring the hepatitis B virus. Over 12,999 cases of HBV infection occur among health care workers each year in the US and 200 die. The risk of acquiring HBV after needle stick exposure to an HBV carrier is estimated to range from 27% to 43%.

There were 12,931 chronic hepatitis B reports in LAC in 2001; 51% were in younger adults aged 18-44 years. Chronic cases, unlike acute cases, are not routinely investigated or interviewed, so risk factor information is unavailable.

The current approach of vaccination for adolescents and others at high risk, as well as education aimed at eliminating, reducing, or mitigating high-risk behaviors in sexually active adults, should continue. Ongoing improvements in data collection and analysis will provide a more accurate description of this infection in the future.

ADDITIONAL RESOURCES

Epidemiology and Prevention of Viral Hepatitis slide set available at: www.cdc.gov/ncidod/diseases/hepatitis/slideset/hep_b/slide1.htm

CDC Publications regarding viral hepatitis at: www.cdc.gov/ncidod/diseases/hepatitis/resource/pubs.htm

General information available at: www.cdc.gov/ncidod/diseases/hepatitis/b/index.htm and www.hepb.org

Immunization information available at: www.immunize.org

Information regarding the control and reporting of hepatitis B in LAC is available at the LAC DHS Acute Communicable Disease Control website: www.lapublichealth.org/acd/procs/b73/b73index.htm

HEPATITIS E	B, PERINATAL
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a Not available.

DESCRIPTION

Hepatitis B is a vaccine-preventable disease transmitted through parenteral or mucous membrane exposure to the blood and other body fluids of individuals infected with the hepatitis B virus (HBV), a DNA-virus of the Hepadnaviridae family. It is also transmitted from mother to infant during birth. Within LAC, it is estimated that over 40% of infants born to Hepatitis B surface antigen (HBsAg) positive women will become infected without prophylaxis. An estimated 90% of infants who become infected by perinatal transmission will have chronic HBV infection and up to 25% will die of chronic liver disease as adults. Hepatitis B vaccination and one dose of hepatitis B immune globulin (HBIG), administered within 24 hours after birth, are 85-95% effective in preventing both HBV infection and the chronic carrier state. The Immunization Program's Perinatal Hepatitis B Prevention Program (PHBPP) conducts case management of chronic HBsAg-positive pregnant women, their newborns, and household contacts.

DISEASE ABSTRACT

- The majority of HBsAg-positive women giving birth were born in areas of the world with high or intermediate levels of endemic hepatitis B disease.
- Of infants born to HBsAg-positive mothers, 97% were immunized within 24 hours of birth.
- Of those responding to a survey 3 to 9 months after the full vaccination series was completed, 88% of infants were found to be protected against HBV, 6% were still susceptible, and 6% were found to have been infected with HBV.

STRATIFIED DATA

Trends: In 2001, 733 infants (including 10 sets of twins) were born to 723 HBsAgpositive women. The incidence of infants born to HBsAg-positive mothers was essentially unchanged from 2000 (Figure 1).

Race/Ethnicity: The majority of the cases were among Asian/Pacific Islanders (API). Five hundred twenty-five (73%) of the women were API, 108 (15%) were Latino, 47 (6%) were Black, 28 (4%) were White, and 15 (2%) were Other (Figure 2). Of API women, 277 (53%) were Chinese, 86 (16%) Vietnamese, 63 (12%) Filipino, 59 (11)% Korean, and 40 (8%) other API women from Samoa, Tonga, Japan, Laos, Burma, Indonesia, and India.



Age: The age-range of mothers was 15-50 years of age with a median age of 31 years.

Location: The majority of the HBsAg-positive mothers resided in SPA 3 (n=308, 43%) primarily because of the large Asian/Pacific Islander constituency. An additional 15% resided in SPA 4 (n=111), followed by 90 mothers in SPA 2, 65 in SPA 7, 60 in SPA 8, 52 in SPA 6, 23 in SPA 5, 9 in SPA 1, and 5 where residence could not be confirmed.

Countries of Origin: The majority of the HBsAg-positive women giving birth were born outside of the US. Six hundred and forty-two (89%) were non-US born. Of these women, 548 (85%) were born in areas of the world with high or intermediate levels of endemic hepatitis B disease, such as Southeast Asia, Central Asia, India, the Middle East, Africa, Eastern Europe, South Pacific Islands, and Central and South America.

CASES COMPLETED FOR FOLLOW-UP IN 2001

In 2001, follow-up was completed for 712 women, their 718 newborns, and 1,066 household contacts. Fifty-two mothers were excluded (26 mothers miscarried, 5 transferred/moved out of LAC prior to delivery, 10 were unable to be located before delivery and 11 were retested and found to be HBsAg negative). Case managers made numerous attempts to complete follow up of infants and household contacts; therefore, some of the cases completed in 2001 were reported in 1999 and 2000.

Case management protocol includes (1) educating pregnant HBsAg-positive women about HBV disease, transmission, and infant vaccinations, (2) identifying and referring household contacts for screening and vaccination, (3) notifying hospitals of the expected deliveries and requesting that the hospitals return documentation after the infant-s birth with the dates and times of the administration of hepatitis B vaccine #1 and HBIG, (4) notifying the infant-s health care provider about the need for hepatitis B vaccine #2 at 1-2 months and hepatitis B vaccine #3 at six months of age, (5) reminding parents about these needed vaccinations, and (6) sending postvaccination serology letters to pediatric health care providers.

Infant Immunoprophylaxis Completion Rates: Of 718 eligible infants (including 6 sets of twins), 94% received both hepatitis B vaccine #1 and HBIG within 12 hours of birth, while 98% received hepatitis B vaccine #1 and 97% received HBIG within 24 hours of birth. Six hundred sixty-four infants (94%) received HBIG and a complete three-dose series of hepatitis B vaccine (Table 1).

	-		
Hepatitis B Immunoprophylaxis	Number of Infants	Percent*	
Infants who received hepatitis B vaccine #1 within 12 hours of birth	671	94%	
Infants who received hepatitis B vaccine #1 within 24 hours of birth	695	98%	
Infants who received HBIG within 12 hours of birth	667	94%	
Infants who received HBIG within 24 hours of birth	692	97%	
Infants who completed HBIG/3-dose hepatitis B vaccine series	664	93%	

Table 1. Summary of Infant Hepatitis	B Immunoprophylaxis, LAC, 2	2001
(N=71	18)	

* Percent of infants receiving hepatitis B immunoprophylaxis out of 718 infants born to 712 HBsAg+ mothers who completed follow-up in 2001. Total includes infants who moved out of LAC prior to 6 months of age and prior to completion of the 3-dose hepatitis B vaccine.

Household and Sexual Contacts Completion

Rates: A household contact was defined as an individual with anticipated continuous household exposure for greater than one year (often limited to nuclear family). Of 1,066 household and sexual contacts identified, 617 (58%) had already been vaccinated against hepatitis B, and 166 (16%) were known to have serologic evidence of hepatitis B infection. Of the remaining 283 (27%) contacts, 154 (14%) were screened for serologic evidence of hepatitis B infection or immunity, while 116 (11%) refused screening or vaccination, were lost to follow-up, or moved; 1% were vaccinated without screening. Of the 138 household contacts who were serologically screened, 91 (66%) had positive markers for



hepatitis B and therefore did not need vaccine. Forty-seven (34%) of the screened household contacts were seronegative that is, susceptible to hepatitis B infection (Figure 3). At the time of completion of case management for the HBsAg-positive mothers, 38 (81%) of the susceptible household contacts had completed all three doses of hepatitis B vaccine.

Post-vaccination serology results: Postvaccination serology testing of infants born to HBsAgpositive mothers is recommended 3 to 9 months after completing immunoprophylaxis to verify vaccine failure or success. Letters requesting postvaccination serology results were mailed to pediatric health care providers of infants tracked by the PHBPP. The postvaccination serology results of 217 infants (27%) whose follow-up was completed in 2001 were received. Of these, 191 (88%) had antibodies to hepatitis B surface antigen indicating protection against HBV, 13 (6%) were HBsAg-positive and infected, and 13 (6%) were negative for both markers and revaccination was recommended.

ADDITIONAL RESOURCES

Viral Hepatitis B Virus slide set. www.cdc.gov/ncidod/diseases/hepatitis/slideset/hep_b/slide_1.htm

CDC Publications: Viral Hepatitis. Available at www.cdc.gov/ncidod/diseases/hepatitis/resource/pubs.htm

Viral Hepatitis B. www.cdc.gov/ncidod/diseases/hepatitis/b/index.htm

Immunization Action Coalition. www.immunize.org

Hepatitis B Foundation. www.hepb.org/

LAC, Acute Communicable Disease Control. www.lapublichealth.org/acd/procs/b73/b73index.htm

LAC DHS, Immunization Program at: www.lapublichealth.org/ip

HEPATITIS C, ACUTE

CRUDE DATA		
Number of Cases Annual Incidence LA County	1 N/A ^a	
Age at Diagnosis Mean	N/A ^b	
Median Range	N/A ^b N/A ^b	
LA County United States	0.0% N/A	

^aRates based on less than 20 observations are unreliable.

^bNot available.

DESCRIPTION

The Hepatitis C virus (HCV) is the most common blood-borne infection in the US. This RNA virus is one of at least 5 different viruses associated with liver disease that is predominantly transmitted through contact with contaminated blood and blood products. Sexual and perinatal transmission of HCV appears to occur less frequently, but its epidemiology has yet to be fully elucidated. People at risk include anyone who has had a blood transfusion prior to 1989, IV drug users, hemodialysis patients, infants born to infected mothers, those with multiple sexual partners, health care workers who suffer needle-stick accidents and people with tattoos or body-piercings. However, an estimated 30% have no identifiable history of exposure to the virus and household/familial contact is not considered a risk factor for the transmission of hepatitis C. There is no vaccine available for HCV and vaccines for hepatitis A and B do not provide immunity against hepatitis C.

DISEASE ABSTRACT

- 11,379 HCV chronic cases were reported in 2001.
- This is a 7.5% increase from 10,044 cases reported in 2000.
- Only 1 case was confirmed as acute hepatitis C out of 26 in depth investigations.
- The acute case occurred in a 25 year-old white who had a surgical procedure in the 6 months before onset of symptoms; no blood products were involved.

COMMENTS

In the US, the annual estimated number of acute HCV infections has declined during the past decade from 180,000 to 35,000. Primary prevention efforts concentrate mainly on risk-behavior

modification - specifically, avoiding contact with contaminated blood. An estimated 3.9 million Americans are currently infected with HCV, and an estimated 8,000-10,000 deaths each year result from HCV-associated chronic liver disease. HCV infection affects persons of all ages, but most acute cases of hepatitis C and the highest seroprevalence of HCV infection are found among young, male adults. The highest proportion both of incident cases and of prevalent infections is among whites.

Conducting surveillance for acute hepatitis C is difficult. There are stringent criteria set forth by the CDC and the CSTE which are required in order to be diagnosed with acute hepatitis C. It is important to differentiate acute hepatitis C from chronic cases because public health can learn about current patterns of transmission and acquisition of hepatitis C only from acute cases. With more widespread use of HCV testing increasingly larger numbers of persons with a positive anti-HCV (antibody to HCV) test are being reported to state and local health departments. Most of these reports represent chronic disease from past drug use or blood transfusions. Because there is no serologic marker for acute hepatitis C, additional investigation is required to determine if these reports represent acute infection, chronic infection, repeated testing of a person previously reported, or a false-positive result.

The current CDC/CSTE definition for acute hepatitis C requires that a person have evidence of jaundice or an onset date of symptoms within six months of diagnosis and have the following laboratory results:

- A positive anti-HCV test confirmed by a more specific test (RIBA or RNA by polymerase chain reaction) or an EIA signal to cutoff ratio of
 <u>></u>3.8;
- Serum alanine aminotransferase (ALT) greater than 7 times the upper limit of normal;
- No evidence of either acute hepatitis A or B disease.

Such stringent criteria explain why it is hard to classify the HCV cases reported to LAC DHS as acute hepatitis C; most of the anti-HCV reports are not accompanied by results of the other laboratory tests. LAC DHS does not have the resources to follow-up on every case of anti-HCV reported. Only those cases that are reported with additional laboratory information are investigated; therefore the count of acute hepatitis C cases are an underestimate of the actual number of acute hepatitis C cases in LAC. Furthermore, there has been a recent change in the ALT levels necessary to be considered as an acute HCV case. Since 2000, the serum ALT levels have been raised from 2.5 times the upper limit of normal (ALT>120U/L) to 7 times the upper limit of normal (ALT>280 U/L). Thus the number of acute hepatitis C cases decreased from 10 in 2000 to 1 in 2001. LAC DHS is exploring ways to improve surveillance for hepatitis C in cases and better understand the epidemiology of acute hepatitis C in LAC.

Universal blood product screening in 1990 and heat-inactivation of other blood concentrates since 1987 have dramatically reduced recipient-associated cases of hepatitis C. This action leaves reduction of high-risk behaviors as the chief further means to prevent transmission. Education aimed at reducing high-risk behaviors for hepatitis B and HIV transmission B such as sharing injection drug equipment B should have additional benefit in reducing hepatitis C cases. Testing should be offered routinely to persons most likely to be infected with HCV who might require medical management, and testing should be accompanied by appropriate counseling and medical follow-up. Once chronic infection has occurred, consuming alcohol and becoming co-infected with HIV or other hepatitis A or B viruses can accelerate the progression of hepatitis C disease to cirrhosis, liver failure, and hepatocellular carcinoma. Additional funding is

necessary to study the feasibility of hepatitis B vaccine into existing programs that provide drug/alcohol treatment as well as HIV screening and treatment.

The most important areas for future research include developing less toxic treatments and finding better ways to identify those who are infected. New studies, including evaluations of the newest combination treatment in patients who haven't responded to other treatments or who had to stop those treatments due to side effects still need to be conducted.

ADDITIONAL RESOURCES

American Liver Foundation website: www.liverfoundation.org/

International Liver Foundation website: www.hepfi.org/

CDC website: www.cdc.gov/ncidod/diseases/hepatitis/

Information regarding the rules, regulations and control of hepatitis C within LAC is available from the Acute Communicable Disease Control website at: www.lapublichealth.org/acd/procs/b73/b73index.htm

INVASIVE GROUP A STREPTOCOCCUS (IGAS)

CRUDE DATA		
Number of Cases	127	
LA County United States	1.4 N/A	
Age at Diagnosis		
Mean	45	
Median	46	
Range	0-95 years	
Case Fatality		
LA County	8.7%	
United States	N/A	



a Cases per 100,000 population.

DESCRIPTION

Group A *Streptococcus*, *Streptococcus pyogenes*, causes invasive, noninvasive (e.g. "strep throat"), and nonsuppurative (e.g. acute rheumatic fever and poststreptococcal glomerulonephritis) diseases. Invasive group A streptococcal (IGAS) disease is defined as isolation of *Streptococcus pyogenes* from a normally sterile body site (e.g., blood, cerebrospinal fluid, bone, joint fluid, or from tissue collected during surgical procedures). IGAS includes the following potentially overlapping clinical syndromes:

- Streptococcal toxic shock syndrome (STSS) characterized by early shock and multiorgan system failure;
- Necrotizing fasciitis (NF) necrosis of subcutaneous soft tissue and skin with signs of severe systemic disease;
- Sterile site infections that do not meet the clinical criteria for STSS or NF, including bacteremia without an apparent focus of infection, and focal infections (e.g., meningitis, pneumonia, peritonitis, osteomyelitis, septic arthritis, and deep soft tissue infections) with or without bacteremia.

A case was defined as isolation of group A *streptococcus* (GAS) from a normally sterile site or from a nonsterile site (e.g., wound culture) in conjunction with NF or STSS. Case patients who had positive results of blood culture for GAS but for whom no clinical syndrome was identified on the initial report were categorized as having only bacteremia without a source. Case patients were categorized as having NF or STSS if the diagnosis was made by the treating physician with or without fulfillment of the case definitions for these syndromes.

DISEASE ABSTRACT

- A small cluster of fatal IGAS cases occurring among children during a four-week period was investigated and the cases were determined to be unrelated
- There has been no sustained increase in the incidence of IGAS in LAC since 1997.
- There was one cluster of 4 cases of IGAS that occurred in a hospital burn unit (see Special Reports).

Table 1: Frequency of IGAS, STSS and NF – LAC, 1994-2001			
	IGAS	STSS	NF
Year	Ν	N (% of IGAS)	N (% of IGAS)
1994	83	29 (35)	18 (22)
1995	103	16 (16)	17 (17)
1996	175	9 (5)	13 (7)
1997	205	7 (3)	9 (4)
1998	128	8 (6)	13 (10)
1999	114	6 (5)	11 (10)
2000	154	8 (5)	20 (13)
2001	127	3 (2.4)	15 (12)

STRATIFIED DATA

Trends: The number of reported cases decreased 18% from 154 cases in 2000 to 127 cases in 2001 and was below the peak seen in 1996 and 1997 (Figure 1). The year-to-year variation has been substantial, ranging from 83 reported cases in 1994 to a high of 205 cases in 1997 (Table 1).

Seasonality: Cases occurred throughout the year. The pronounced winter/spring seasonality commonly associated with streptococcal pharyngitis was not observed (Figure 2).

Age: The mean age of cases was 45 years and the median was 46 years (range newborn to 95 years). The incidence varied substantially by age (Figure 3), and was highest among infants aged less than 1 year (6.0 cases per 100,000 population), followed by those aged >65 years (3.6 cases per 100,000).

Gender: The male-to-female rate ratio was 1.4:1. More detailed case investigation, including collection of risk factor data such as injection drug use, might serve to explain the gender disparity.



Race/Ethnicity: Race/ethnicity was known for 69 (54%) cases; of these, 25 (38%) were White, 31 (47%) were Latino, 5 (7%) were Black, and 4 (6%) were Asian and 4 (6%) were other.

Clinical Syndromes: The distribution of clinical syndromes among cases is shown in Table 2. The majority of cases (87, 69%) were categorized as bacteremia without an apparent source, followed by necrotizing fasciitis (15 cases. 12%), soft tissue infections, not NF (10 cases, 8%), meningitis (5 cases, 4%), septic arthritis (4 cases, 3%), pneumonia (3 cases, 2%), osteomyelitis (2 cases, 1.6%), and other (3 cases). There were 11 known deaths for an overall IGAS case fatality rate of 8.7 %. This value is lower than the approximately 12% case fatality rate observed in population-based IGAS studies, and probably represents incomplete outcome data in LAC in 2001.



Of the 15 cases of NF, the mean age was 45 years, the median was 49 years and the range was 5 years to 72 years. Nine were male. All case patients underwent surgical debridement and four required amputation. There was one known death.

Location: The crude incidence rate for IGAS was highest in Service Planning Area (SPA) 6 (2.2 cases per 100,000 population), compared with a mean of 1.4 per 100,000 for all of LAC (Figure

4). However, many of the rates are unstable because they are based on small numbers of reported cases.

Cluster of Fatal IGAS Cases Among **Children:** Between January 19 and February 18, 2001, three fatal cases of STSS occurred among children aged five years (2 cases) and fourteen years (1 case). Multi-limb necrotizing fasciitis accompanied by STSS developed in one of the cases aged 5 years following varicella; for the other two cases, there was no apparent focus of infection. Epidemiologic investigation along with molecular typing of case isolates by pulsed-field ael electrophoresis of GAS isolates showed no relationship among the cases.



COMMENTS

IGAS disease is not a mandated reportable disease in California. Following a cluster of severe IGAS infections among previously healthy children in 1993, the Acute Communicable Disease Control Unit requested reporting of IGAS disease from laboratories, hospitals, and healthcare providers in LAC. Since 1994, surveillance methods have varied from passive during 1994 and 1995, to stimulated passive as part of a special project between 1996-1999 (Communicable Disease Active Surveillance Project [CDAS]), and passive once again since 2000. The CDAS project was primarily laboratory based and laboratories continue to be the principal reporting sources for IGAS. Consequently, information pertaining to clinical presentation, race/ethnicity

and outcome is often incomplete. In 2001, case investigation to collect more detailed demographic, clinical and outcome data was conducted on selected cases known to involve NF, STSS or other severe manifestations.

Case information was obtained from hospital infection control staff for 46 cases, either from the initial report or case follow-up; the remainder of the cases were reported by laboratories only, either by means of laboratory culture reports or Confidential Morbidity Report forms. Information regarding race/ethnicity, clinical syndromes and outcomes was usually not available for cases reported only by laboratories. Consequently, it is likely that the number of deaths and the occurrence of nonbacteremic clinical manifestations are underestimated.

With the exceptions of eliminating varicella as a risk factor for IGAS through vaccination, and preventing nosocomial transmission (especially in obstetrical and surgical settings) and outbreaks in childcare centers, opportunities for public health intervention of IGAS are limited. The extent to which each reported case of IGAS should be investigated must be weighed in the context of available public health resources. However, at a minimum, the completeness of reporting should be assessed in order to make meaningful year-to-year comparisons.

ADDITIONAL RESOURCES

- The Working Group on Severe Streptococcal infections. Defining the group A streptococcal toxic shock syndrome. Rationale and consensus definition. *JAMA* 1993;269:390-1.
- Davies HD, McGeer A, Schwarz B, et al. Invasive group A streptococcal infections in Ontario, Canada. *N Engl J Med* 1996;335:545-54.
- Kaul R, McGeer A, Low D, et al. Population-based surveillance for group A streptococcal necrotizing fasciitis: clinical features, prognostic indicators, and microbiologic analysis of seventy-seven cases. *Am J Med* 1997;103:18-24.
- American Academy of Pediatrics. Committee on Infectious Diseases. Severe invasive group A streptococcal infections: a subject review. *Pediatrics.* 1998;101:136-40.
- Zurawski CA, Bardsley MS, Beall B, et al. Invasive group A streptococcal disease in metropolitan Atlanta: a population-based assessment. *Clin Infec* Dis 1998;27:150-7.
- Laupland KB, Davies HD, Low DE, et al. Invasive group A streptococcal disease in children and association with varicella-zoster virus infection. Ontario Group A Streptococcal Study Group. *Pediatrics* 2000;105(5):E60.
- O'Brien KL, Beall B, Barret NL, et al. Epidemiology of invasive group A streptococcal disease in the United States, 1995-1999. *Clin Infec Dis* 2002;36:268-276.

LEGIONELLOSIS

CRUDE DATA		
Number of Cases	18	
LA County	N/A ^b	
United States	0.4	
Age at Diagnosis		
Mean	55	
Median	50	
Range	22-78 years	
Case Fatality		
LA County	1.6%	
United States	N/A	

a Cases per 100,000 population.

^b Rates based on less than 20 observations are unreliable.

DESCRIPTION

Legionellosis is a bacterial infection with two distinct clinical forms: Legionnaires' disease (LD), the more severe form characterized by pneumonia; and Pontiac fever, an acute-onset, self-limited flu-like illness without pneumonia. Legionella bacteria are common inhabitants of aquatic systems and thrive in warm environments. Ninety percent of cases of LD caused by Legionella pneumophila. are although at least 11 other species and a number of serogroups are known to cause disease in humans. Transmission occurs through inhalation of aerosols containing the





bacteria or by aspiration of contaminated water. Person-to-person transmission does not occur. The case fatality rate for LD ranges between 5%-15% or even higher in outbreaks occurring in the hospital setting. People of any age may get LD, but the disease most often affects middle-aged to older persons, particularly those who are heavy smokers, have chronic lung disease, or whose immune system is suppressed by illness or medication.

DISEASE ABSTRACT

- As in previous years, the incidence of legionellosis in LAC was well below national levels.
- Two definite and three possible nosocomial cases in separate facilities were reported in 2001; no outbreaks were identified.
- There were no cases of Pontiac fever.

STRATIFIED DATA

Trends: Eighteen reported cases met the CDC surveillance case definition for LD in 2001, remaining well below the all-time high number of 32 cases reported in 1997 (Figure 1).

Seasonality: Thirteen cases (72%) occurred during the summer and autumn months of June through November (Figure 2). This is consistent with national surveillance data, possibly representing increased exposure related to travel and/or contaminated air-cooling systems during warmer months.

Age: The mean age of reported cases was 55 years, the median age was 50 years, and the range was 22-78 years.

Gender: The male-to-female case ratio was 2.6:1. Disproportionately higher rates of legionellosis among males has been a consistent finding in LAC in previous years and is consistent with national surveillance data. Both cigarette smoking and older age are recognized risk factors for LD. An explanation often offered to explain the gender disparity in LD is the higher prevalence of cigarette smoking among males in the older age groups. The gender disparity in prevalence of smoking in the older age groups is expected to narrow or disappear in the near future, as it has among younger age groups.

Race/Ethnicity: The distribution by race/ethnicity of legionellosis in 2001 was remarkable for the predominance of cases among Whites; 14 of the 18 cases occurred among Whites, 3 among Latinos, and 1 among Asians. None of the cases was Black.

Location: There was no apparent clustering by district of residence; no more than two cases each occurred among residents of the following health districts: Hollywood-Wilshire, Bellflower, Pomona, San Fernando, West, Southwest, Glendale, Harbor, Torrance, and Inglewood Health Districts.

COMMENTS

At least one recognized risk factor for LD was present in 11 of the 14 (79%) cases for whom risk factors were known and included heavy smoking and/or chronic pulmonary disease (4 cases), malignancy (4 cases), chronic renal disease (1 case), AIDS (2 cases), and diabetes (1 case). Three case patients had traveled out of LAC during the incubation period, one to Hong Kong, one to Thailand, and one to Nevada.

Four cases were confirmed by culture alone, two by both culture and urine antigen testing, and 12 by urine antigen testing alone. *Legionella pneumophila* serogroup 1 was implicated in all but one of the cases, possibly reflecting increased use of urine antigen testing, which is specific for Lp1.

Two definite and three possible nosocomial cases were investigated in 2001. The affected facilities were located in Central, Northeast, San Fernando, and Hollywood-Wilshire Health Districts. CDC guidelines for investigation of nosocomial legionellosis were followed in each facility and no additional cases were identified.

The number of cases of legionellosis in LAC remains lower than expected based on national surveillance data and other epidemiologic studies. Empiric treatment for community-acquired pneumonia without specific testing for Legionella, inappropriate laboratory testing (use of a

single serologic antibody titer testing), and underreporting by physicians are possible explanations.

ADDITIONAL RESOURCES

Guidelines:

- Centers for Disease Control and Prevention. Guidelines for Prevention of Nosocomial Pneumonia. Morbid Mortal Weekly Rep. 1997;(RR-1):1-79.
 www.cdc.gov/ncidod/diseases/hip/pneumonia/pneu_mmw.htm
- Allegheny County Health Department. Approaches to prevention and control of Legionella infection in Allegheny County health care facilities. 2nd ed. Pittsburgh, PA: Allegheny County Health Department. 1997:1-15. <u>www.legionella.org</u>
- State of Maryland, Department of Health and Mental Hygiene. Report of the Maryland Scientific working Group to Study Legionella in Water Systems in Healthcare Institutions. June 14, 2000, Baltimore, Maryland. <u>www.dhmh.state.md.us/html/legionella.htm</u>
- ASHRAE. Guideline 12-2000. Minimizing the risk of legionellosis associated with building water systems. American Society of Heating, Refrigerating and Air-Conditioning Engineers, Atlanta, GA., 1999. www.ASHRAE.org or www.baltimoreaircoil.com/index1.html
- LAC Department of Health Services. Legionellosis: Taking the Mystery out of Laboratory Diagnosis. The Public's Health. 2001;1(3) 4. Available at: www.lapublichealth.org/wwwfiles/ph/ph/TPH_October_2001.pdf

Reviews:

- Stout JE, Yu VL; Legionellosis. N Engl J Med 1997;337:682-687.
- Breiman RF, Butler JC: Legionnaires' disease: clinical, epidemiological, and public health perspectives. Semin Respir Infect 1998;13:84-89.

Selected Articles:

- Yu VL: Resolving the controversy on environmental cultures for Legionella: A modest proposal. Infect Control Hosp Epidemiol 1998;19:893-7.
- Lin YS, Stout JE, Yu VL, Vidic RD: Disinfection of water distribution systems for Legionella. Semin Respir Infect 1998 Jun;13:147-59.

CRUDE DATA Figure 1 Nonperinatal Listeriosis Number of Cases 27 Cases by Year of Onset Annual Incidence LAC. 1992-2001 0.30^a LA County 35 0.21^a **United States** 30 Number of Cases Age at Diagnosis 25 Mean 48 20 Median 50 15 Range 9-94 years 10 Case Fatality 5 LA County 7.0% n 1992 1993 1994 1995 1996 1997 1998 1999 2000 2001 United States N/A Year

LISTERIOSIS, NONPERINATAL

a Rates include both nonperinatal and perinatal cases.

DESCRIPTION

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

DISEASE ABSTRACT

- In June 2001 there was a foodborne listeriosis outbreak with 13 cases in LAC affecting previously healthy individuals with gastroenteritis, associated with turkey deli meat. This outbreak accounted for nearly half of the cases in 2001.
- The nonperinatal listeriosis incidence rate increased in 2001, due to the June outbreak, following a two-year decline in cases.
- The majority of cases occurred in persons >65 years of age, followed by those 15-24 years of age.
- Most cases were White, followed by Latinos. No cases were reported in Blacks.

STRATIFIED DATA

Trends: The incidence of nonperinatal listeriosis increased in 2001, following a two-year decline

in incidence, with 27 cases (Figure 1).

Seasonality: The highest number of reported cases occurred in June (n=13) as a result of an outbreak associated with turkey deli meat. There were smaller peaks seen in May (n=4) followed by October (n=3) (Figure 2).

Age: Age greater than 65 is considered a risk factor for nonperinatal listeriosis. Thirty-three percent of cases were older than 65 years in 2001, resulting in a decrease compared to 2000 (47%) and 1999 (57%). There was an increase in the younger age groups in 2001. There were two cases in the 5-14 year group and six cases in the 15-34 year group, which is not typically seen in LAC. Five of these younger cases were associated with the June outbreak (Figure 3).

Sex: The male-to-female incidence ratio was 0.59:1.

Race/Ethnicity: In 2001, Whites had the highest incidence of nonperinatal listeriosis (n=16). Latinos had the second highest number (n=6) followed by Asians (n=5). All 6 of the June outbreak cases were White. There were no Black cases reported in 2001 (Figure 4).

Location: Glendale Health District had the highest incidence (n=9); 5 of the 9 cases were part of the June outbreak. Alhambra Health District had the next highest incidence (n=4); 1 of the cases was part of the June outbreak.

Predisposing Conditions and Medical Risk Factors: Nine (33%) of 26 cases were older than 65 years of age, 7 (26%) were diagnosed with cancer, 6 (22%) had diabetes, and 8 (30%) had no identified risk factors. Of the eight with no identified risk factors, six were part of the June outbreak (Table 1).

Outcome: Two (7%) of 27 cases in 2001 died.

Culture Sites: *Listeria monocytogenes* was isolated from blood (n=14), stool (n=8), CSF (n=6), and once each from inguinal tissue and peritoneal fluid. Three cases had LM isolated from two locations.







COMMENTS

There was a foodborne *Listeria monocytogenes* outbreak with 28 individuals experiencing gastroenteritis associated with turkey deli meat, 6 were lab-confirmed cases; only lab-confirmed cases were counted as cases in this report. The outbreak occurred following a catered birthday party. Two other events catered by the same caterer were also associated with this outbreak. See 2001 Special Reports for a complete summary of this outbreak.

Table 1: Predisposing Factors in Cases of Nonperinatal Listeriosis, LAC, 2001		
Medical Conditions	Number	Percent
Age >65 years	9	33
Cancer	7	26
Diabetes	6	22
Steroid Use	4	15
Kidney Disease	4	15
Prior Antibiotic Use	3	11
No Identified Risk Factors	8	30

All LM isolates are now typed by pulsed field gel electrophoresis (PFGE). There were no LAC outbreaks or LAC cases associated with a multi-jurisdictional outbreak identified in this manner in 2001.

PREVENTION

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

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

ADDITIONAL RESOURCES

www.cdc.gov/ncidod/dbmd/diseaseinfo/listeriosis_g.htm

Information regarding the rules, regulations and control of this disease within LAC is available from the Acute Communicable Disease Control website at: www.lapublichealth.org/acd/procs/b73/b73index.htm

LISTERIOSIS, PERINATAL



a Rates include both nonperinatal and perinatal cases.

DESCRIPTION

Listeriosis is a disease transmitted primarily through consumption of food contaminated with *Listeria monocytogenes* (*LM*), a gram-positive bacterium. *LM* is found in soil and water, and may contaminate raw foods, such as uncooked meats and vegetables, as well as processed foods that become contaminated after processing, such as soft cheeses and cold cuts. Unpasteurized (raw) milk or foods made from unpasteurized milk may also contain the bacterium.

Infected pregnant women may experience only mild flu-like symptoms or may be asymptomatic. A perinatal listeriosis case is defined as a pregnant woman, fetus or neonate with infection of a sterile site with *LM*. Neonatal/infant listeriosis is divided into early onset (0-6 days after birth) and late onset (7-42 days after birth). Infection during pregnancy may lead to premature birth, stillbirth, or septicemia and/or meningitis in the neonate, even if the mother is asymptomatic. There is no vaccine to prevent listeriosis.

DISEASE ABSTRACT

- The perinatal listeriosis incidence has been declining since 1999, and is the lowest seen in LAC over the last 10 years.
- All three cases in 2001 were early onset, and they all survived.

STRATIFIED DATA

Trends: The 2001 perinatal listeriosis incidence has decreased annually since 1999 (Figure 1).

Seasonality: There were too few cases to look for seasonality. Two of the cases occurred in April and one case in July.

Age: The three women were ages 23, 31, and 35. Two delivered prematurely at 31 and 33 weeks while third was 38 weeks.

Sex: All three infants born to 2001 cases were female.

Race/Ethnicity: Two of the cases were Latino, and one was Asian.

Location: The three perinatal cases were from three different health districts; Pomona, Southwest and Whittier.

Type of Delivery: The method of delivery was Caesarian section for two cases and vaginal for one case. Two cases delivered prematurely at 31 and 33 weeks; the third delivered at 38 weeks gestitation.

Outcome: All three infants and mothers survived.

Culture Sites: Sites of *LM* isolation were from blood, placenta, and tracheal aspirate. No *LM* isolations were from amniotic fluid (Table 1). All three infants were blood culture positive for *LM*. Two mothers had positive blood cultures, with one having a positive tracheal fluid culture as well. The third mother had a positive placental culture.

Onset: In 2001, all cases were classified as early-onset.

From Mothers and mants, LAC, 2001				
	Mother	Mother (n=3)		(n=3)
Culture Site	Number	Percent	Number	Percent
Blood	2	67	3	100
Placenta	1	33	0	0
Tracheal Aspirate	1	33	1	33
Amniotic Fluid	0	0	N/A	

Table 1: Frequency (%)* of Listeria monocytogenes IsolatesFrom Mothers and Infants, LAC, 2001

* Percentages may exceed 100% as cultures were obtained from more than one site in some cases.

COMMENTS

Cases of perinatal listeriosis have decreased compared to the number of cases in 1999 and 2000. Incidence by race has shifted; however the numbers are small. Latino mothers (n=2) now have the highest incidence whereas White mothers (n=4) had the highest incidence in 2000. There were no perinatal cases associated with outbreaks in 2001.

All isolates of LM are now typed by pulsed-field gel electrophoresis (PFGE), a technique to

detect matching strains of various pathogenic agents. When matches between isolates from patients or foods are detected, an investigation may be initiated. In addition, a solitary case occurring locally can be linked by PFGE results to an outbreak occurring on a wider geographical scale. In 2001, there were no cases of *LM* in LAC associated with a multi-jurisdictional outbreak identified in this manner.

PREVENTION

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

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

LYME DISEASE

CRUDE DATA		
Number of Cases Annual Incidence ^a	5	
LA County	N/A ^b	
United States	6.0	
Age at Diagnosis		
Mean	38	
Median	42	
Range	2-82 years	
Case Fatality		
LA County	0.0%	
United States	N/A	

a Cases per 100,000 population.

^b Rates based on less than 20 observations are unreliable.

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 small rodents, with deer as a secondary reservoir. Ticks that feed on 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 such



as fever, body aches, headaches and fatigue may be not be recognized as Lyme disease 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 available.

The diagnosis of Lyme disease may be difficult because many other diseases cause 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 2001, five reported cases of Lyme disease met CDC surveillance criteria.
- Three cases were male.
- Four cases reported exposure outside LAC.

COMMENTS

Lyme disease is now the most frequently reported vector-borne disease in the US. Lyme disease is reported infrequently in LAC. Since Lyme disease became reportable in 1989, 48 reported cases have met the CDC surveillance criteria. Sixteen cases (33%) 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 common 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 the Acute Communicable Disease Control Unit, 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. The 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 can be taken to prevent tick bites. These measures 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

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 Jul 12;345(2):115-125.

Sood SK. Lyme disease. Pediatr Infect Dis J. 1999;18:913-25.

CDC WEB page: www.cdc.gov/ncidod/dvbid/lyme/index.htm

CA DHS Brochure:

www.dhs.ca.gov/ps/dcdc/disb/pdf/Lyme%20Disease%20brochure%20final.pdf
MALARIA

CRUDE DATA			
Number of Cases	46		
Annual Incidence ^a			
LA County	0.53		
United States	0.54		
Age at Diagnosis			
Mean	40		
Median	43		
Range	<1-75 years		
Case Fatality			
LA County	0.0%		
United States	N/A		



a Cases per 100,000 population.

DESCRIPTION

Malaria is caused by four species of the genus *Plasmodium*: *P. vivax* (PV); *P. falciparum* (PF); *P. malariae* (PM); and *P. ovale* (PO). PF can cause cerebral malaria and sometimes death. Malaria is acquired from the bite of an infective female *Anopheles* mosquito. Malaria is not transmitted locally in LAC, although a vector, *Anopheles hermsi*, exists here.

DISEASE ABSTRACT

- The incidence of malaria in LAC remained stable at 43 cases in 2000 to 46 in 2001.
- The percent of malaria cases who were US residents increased from 56% in 2000 to 63% in 2001.
- The percent of malaria cases who were recent immigrants or visitors to the US decreased from 44% in 2000 to 26% in 2001.
- Of US resident cases, 24.1% had taken some form of prophylaxis.

STRATIFIED DATA

Species Frequency: The infecting malarial species was identified for 45 cases (98%) (Figure 4). Most cases were infected with PF (53%) or PV (41%). There was one unspecified case (2%) and one case of mixed PF and PM infection (2%).



Seasonality: In 2001 May and July had the most cases of malaria. As is typical, the fall and winter months had fewer cases compared to spring and summer months (Figure 2). These fluctuations in malaria cases by month are probably due to travel.

Age: Most cases of malaria were in persons aged 15 to 24 years. This was due to more travel in this age group.

Sex: The rate ratio of male-to-female cases was 1.3:1.

Race/Ethnicity: Cases were highest among African nationals/Black Americans (Figure 5). Most Latino cases were immigrants, individuals visiting the US, or persons whose residency status was unknown.

Location: West District had the most cases (8). Inglewood had 5 cases and West Valley had 4.

COMMENTS

In LAC, malaria is a disease that is related to travel and immigration. There is no documentation of malaria being transmitted locally, but a competent vector is found in LAC. Local transmission has not occurred here perhaps due to the dry weather and lack of a concentrated group of people circulating the parasite.

Malaria cases among immigrants and foreign nationals overestimate the risk to local residents. Residency and/or reason for travel were available for 41 of 46 cases of malaria (Table 1). Sixty-three percent (29/46) of Omalaria cases were LAC residents who traveled abroad either for work or vacation. Thirty-seven percent (17/46) were recent immigrants, individuals visiting the US, or those whose residency status was unknown. The reason for this drop in malaria cases overall is probably due to fewer people emigrating from malarial regions. There were fewer immigrants this year (percentage-wise) that contributed to malaria cases. The number of malaria cases overall is still far below the







numbers of cases seen throughout the late 1970s through mid- 1980s (yearly average from 1979-1986 = 133 reported cases/year).

Among malaria cases in US residents traveling abroad, Africa remains the most common region visited and Nigeria the most frequent destination. This is mostly due to naturalized Nigerians visiting relatives still living in Nigeria. Thirty-nine percent (18/46) of all reported malaria cases were from individuals who had traveled to or were coming from African countries. Since the early 1990s Blacks/African nationals have been the ethnic group with the highest incidence of malaria in LAC. Figure 62 shows that Blacks make up only 8% of the population of LAC but account for 48% of cases of malaria. Before the 1990s, immigrants/refugees from Central America and Southeast Asia made up the majority of all malaria cases seen in LAC. Forty-one percent of cases (7/17) who were recent immigrants, visitors to the US, or whose residency status was unknown were from Central America and Mexico. Thirty-five percent (6/17) were from African countries.

Antimalarial prophylaxis history was available for 27 of the 29 US resident cases (Table 1). S Only seven individuals (24%) took prophylaxis. A higher percentage of work-related cases took prophylaxis compared to tourist cases (50 vs. 22%). However, appropriateness of prophylaxis and adherence to regime was unknown, and group size was small.

A low percentage of US residents and recent immigrants had a previous history of malaria this year compared to previous years (Table 2), most likely due to a change in reporting format on the epidemiologic form. For 2001, a history of malaria was only documented if it was within the previous 12 months, rather than at any time in the past. Under the new definition, 4% (2/46) of cases had a previous malaria history.

ADDITIONAL RESOURCES

CDC website: www.cdc.gov/ncidod/diseases/submenus/sub_malaria.htm

Foreign Travel by US Residents		Recent Immigration, Residency Status Unknown, or Visit to US by Non-US Residents		
Region/Country	Number of Cases (Species) ^a	Country	Number of Cases (Species) ^a	
Africa Cameroon Central African Republic ^b Gabon Ghana Ivory Coast Kenya Mozambique Nigeria Uganda ^c	1(1PF) 1(1PF) 1(1PO) 3(1PF,2PV) 1(1PF) 1(1PF) 1(1PF) 11(11PF) 2(1PF,1PF&PM)	Congo Ethiopia Guinea Nigeria Uganda	1(1PV) 1(1PV) 1(1PF) 2(2PF) 1(1PF)	
Latin America Belize Ecuador Guatemala Honduras	1(1PV) 1(1PV) 1(1PV) 2(2PV)	Central America El Salvador Guatemala ^d	1(1PV) 4(4PV) 2(2PV)	
Asia/Oceania India Indonesia Unknown	1(1PF) 1(1PV) 0	India Indonesia Unknown	1(PV) 1(1N) 2(2PV)	
Total	29		17	

Table 1. Malaria Cases by Species, Residency Status, and Travel Exposure B LAC, 2001

^a PF = N = not determined, *P. falciparum*, PM = *P. malariae*, PO = *P. ovale, and* PV = *P. vivax.* ^b Case also traveled through India and Cambodia. ^c Case also traveled through Sudan.

d Case also traveled through Mexico.

Table 2. Malaria Cases by Residency Status, Reason for Travel, Malaria Prophylaxis and Previous Malaria History B LAC, 2001

	US Residents			Non-US Residents	
	Total US Residents	Total USTravel forTravel forResidentsWorkPleasure		Recent Immigrant or Foreign Visitor to US	
Prophylaxis (%)	7/29 (24)	1/2 (50)	6/27 (22)	0 *	
Previous malaria within last year (%)	1/29 (3)	0/3 (0)	1/24 (4)	1/17 (6)	

* Natives of malaria-endemic countries generally do not take pre-exposure prophylaxis.

MEASLES

CRUDE DATA			
Number of Cases	0		
Appual Incidence	δ		
Annual incluence			
LA County	N/A ^a		
California	0.11		
United States	0.04		
Age at Diagnosis			
Mean	15		
Median	12		
Range	1-38 years		
Case Fatality			
LA County	0.0%		
United States	N/A		



^a Rates based on less than 20 observations are unreliable.

DESCRIPTION

Measles is a vaccine-preventable disease caused by a paramyxovirus. Measles 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 positive IgM titers or a four-fold increase between acute and convalescent IgG titers.

DISEASE ABSTRACT

- The annual number of measles cases in 2001 was at its highest since 1995.
- During 2002, 7 of the 8 cases identified were over the age of one year and could have been prevented by Measles-Mumps-Rubella (MMR) vaccine, given in accordance with current Advisory Committee on Immunization Practices (ACIP) recommendations.

STRATIFIED DATA

Trends: According to Figure 1, the number of confirmed measles cases has decreased significantly after a record high in 1993. Although absolute numbers are low, the number of measles cases has been increasing since 1999, reversing a 5 year decline and stabilizing trend.

Sex: The male-to-female ratio of the cases was 1:1.7

Race/Ethnicity: Three cases were Latino, three were Asian, one was White, and one was Black.

Seasonality: All cases occurred in the first two quarters of 2001, consistent with the temporal pattern of measles in temperate areas (Figure 2).

Age: One case was <1 year, two were between 1-4 years, two were between 5-14 years, two were between 15-34, and one was between 35-44 years of age.

Location: Cases were geographically distributed throughout LAC health districts, with one case identified in West, one in West Valley, two in Hollywood Wilshire, two in Bellflower, and two in Torrance.



COMMENTS

Four of the eight measles cases had not been vaccinated for the following reasons: the first case did not receive the MMR vaccine because of a liver transplant, the second case was a child who was too young to receive the vaccine, the third case was a 12-month-old child who did not receive timely vaccination due to traveling, and the last case was an adult male who did not receil ever receiving measles-containing vaccine.

In only one instance was there evidence of secondary spread from the eight measles cases reported in 2001. This involved spread from an un-immunized adult case to an infant who was too young to have been immunized. This cluster of two cases occurred in the Bellflower area of LAC during March. The rarity of secondary cases is a result of the high measles immunization coverage levels generally present among LAC children.

Vaccination Status: Of the eight cases, four claimed to be vaccinated against measles and only two had documentation. However, of these two latter cases, one was given the vaccine prior to the recommended time period, which may not have resulted in lasting immunity.

Importation Status: Four of the eight cases had travel to or arrival from other countries or states within 18 days of rash onset. One had traveled to Japan during the incubation period, which is consistent with disease onset. Another case was a student from Korea visiting the US. A third case had lived in the Philippines since childhood and had recently moved back to California. The final case had recently traveled to Tijuana, Mexico and was not protected by vaccination because of medical concerns with liver transplantation.

Hospitalization: Four cases were hospitalized, with two cases having stayed for 4-5 days. None of the cases was pregnant and no complications were reported.

ADDITIONAL RESOURCES

National Immunization Program at: <u>www.cdc.gov/nip</u>

Immunization Action Coalition at: www.immunize.org

LAC Department of Health Services, Immunization Program at: www.lapublichealth.org/ip

Acute Communicable Disease Program at: www.lapublichealth.org/acd/procs/b73/b73index.htm

CRUDE DATA			
Number of Cases	378		
LA County	4.2		
Age at Diagnosis	N/A		
Mean Median	20 14		
Range	<1-100 years		
Case Fatality			
LA County	N/A		
United States	N/A		

MENINGITIS, VIRAL



a Cases per 100,000 population.

DESCRIPTION

Viral meningitis, also referred to as aseptic meningitis, is a clinical syndrome in which no etiologic agent is identified on bacterial culture or examination of cerebrospinal fluid. When viral culture is done, an enterovirus is the organism most often detected. Transmission may be fecal-oral, respiratory or by another route specific to the etiologic agent. Viral meningitis can occur at any age but is most common among the very young. Symptoms, which usually last from 7 to 10 days, are characterized by sudden onset of fever, severe headache, stiff neck, photophobia, drowsiness or confusion. nausea and vomiting. Treatment is usually supportive although antiviral agents may be available; recovery is usually complete. Enteroviruses, the etiologic agents commonly associated with viral meningitis, are not vaccinepreventable [except for polioviruses].



DISEASE ABSTRACT

- In 2001, reports of viral meningitis increased by 45% from 2000.
- The summer seasonal increase continued later into the year compared with the previous 5 year average (Figure 2).

- No unusual viral etiologies, associated cases, or clusters were reported in 2001.
- The highest age-group specific rate (50.8 per 100,000) continued to be seen in infants aged less than1 year (Figure 3).

COMMENTS

Surveillance for viral meningitis is passive and only outbreaks, not individual cases, are investigated. The number of cases reported annually is considered to be significantly lower than the actual burden of disease. In 2001, there was a 45% increase in the number of cases reported. Reasons for the increase, whether real, or the result of improved reporting or other unknown factors, were not apparent. A similar unexplained increase was seen in 1998.

Information about the causative agents of viral meningitis is rarely included with case reports because viral cultures and RT-PCR tests are



not routinely performed. When an etiology is determined, an enterovirus, most of which are transmitted through the fecal-oral route, is the most frequently identified agent. Improvements in molecular testing capabilities should lead to faster diagnoses and changes in the management of viral meningitis.

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

ADDITIONAL RESOURCES

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

CDC, Respiratory and Enteric Viruses Branch, Non-polio Enterovirus Infections at: www.cdc.gov/ncidod/dvrd/entrvirs.htm

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



MENINGOCOCCAL DISEASE

CRUDE DATA			
Number of Cases	58		
Annual Incidence ^a			
LA County	0.7		
California	0.9		
United States	0.8		
Age at Diagnosis			
Mean	30		
Median	22		
Range	<1-84 years		
Case Fatality			
LA County	16.0%		
United States	N/A		



a Cases per 100,000 population.

DESCRIPTION

Meningococcal disease, occurring most often as meningococcal meningitis or meningococcemia, is transmitted through direct or droplet contact with nose or throat secretions of a person infected with the Neisseria meningitidis bacterium. It affects all age groups but occurs most often in infants. Common symptoms include sudden onset of fever, headache, nausea and vomiting, stiff neck and lethargy, which can progress to overwhelming sepsis, shock and death within hours. In LAC, a confirmed case is one with clinically compatible signs and symptoms and recovery of the organism from a normally sterile site, usually the blood or cerebrospinal fluid. A presumptive case is one with clinically compatible signs and symptoms, and a positive bacterial antigen test on CSF, or identification of gram-negative diplococci from a normally sterile site. N. meningitidis serogroups B, C and Y are the serogroups commonly seen in LAC and the US. Serogroups A, C, Y and W-135 are vaccine-preventable; a vaccine for seroaroup B.

DISEASE ABSTRACT

- Meningococcal disease cases increased but overall incidence remained low.
- A cluster of cases occurred among nightclub patrons.
- Serogroup C predominated.

STRATIFIED DATA

Trends: The number of cases increased but remained relatively low in comparison to previous years (Figure 1). Serogroup C replaced B as the predominant serogroup identified. Cases among older teenagers increased (see "Age" below).

Seasonality: Cases were characteristically highest during winter and early spring, with over 50% occurring in the first quarter of the year (Figure 2).

Age: Rates of meningococcal disease are characteristically highest among infants and children aged 1-4 years. In 2001, rates in these age groups were 5.2/100,000 and 1.2/100,000, respectively. Combined, these two age groups accounted for 19% of all cases. The rate among all age groups remained about the same (Figure 3). The rate in persons aged 15-19 years, a subset of the 15-34 year old group (0.6/100,000), was 1.5/100,000. Cases in this age group accounted for 17% of all cases. There has been an increasing incidence in this age group in recent years, associated with outbreaks among high school as well as freshman college students - especially those living in dormitories (see Comments below). This trend has been seen around the country in recent years.

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

Race/Ethnicity: In 2001, the highest rate (1.01/100,000), and number of cases (n=28), occurred among Whites. Among Latinos the rate increased slightly (0.54/100,000) (n=22), but in Blacks it decreased by 50% (0.73/100,000) (n=6). Among Asians, who historically have the lowest rates, the rate (0.18/100,000) was unchanged from 2000 (n=2) (Figure 4).

Location: The highest number of cases occurred in SPAs 2 (n=13), 3 (n=13) and 4 (n=10). Rates were highest in SPAs 4 and 8 (0.9/100,000 respectively), and SPA 3 (0.8/100,000).

COMMENTS

In 2001, *N. meningitidis* was confirmed by culture in 49 of 58 cases: 27 (56%) from blood, 10 (20%) from cerebrospinal fluid (CSF), 8 (16%) from both blood and CSF, 2 (4%) from synovial fluid, and 1 each (2%) from vitreous fluid and tracheal aspirate (Figure 5). Although tracheal aspirate is not usually considered





□ 2000 ■ 2001

sterile, and the patient, a 25 day old infant, had evidence of respiratory syncytial virus infection as well, this was considered a meningococcal case because the physician considered the infant too sick for RSV infection only. The Public Health Laboratory performed serogroup identification on isolates from 41 confirmed cases. Serogroup identification was made in 32 cases. Of all isolates submitted, 12% were serogroup B; 37% were serogroup C; 24% were serogroup Y; and 5% were serogroup W-135 (Figure 6). Nine case isolates (22%) were nontypeable. In 16% of cases, serogroup information was not obtained.

There was continued public concern about meningococcal disease among high school and college students in 2001. Several high school clusters occurred in northern California and other states. None occurred in LAC. However, there was a cluster of cases among young men of college age and three unrelated cases in college students. The cluster occurred among three unacquainted men, aged 19-22 years, who attended a popular nightclub on the same night, along with several hundred others. Two of the cases were confirmed as Serogroup C meningococcal disease. The third case was presumptive based on Gram-negative cocci in cerebrospinal fluid and clinical signs and symptoms. Active surveillance in LAC and adjacent counties did not identify additional cases. The college cases, two in unimmunized college freshmen and the third in a graduate student whose immunization status was not determined, occurred at different universities,. Serogroup C was identified in two cases, stimulating renewed interest in meningococcal immunization among students.

PREVENTION

In 2001, at least 47% of the cases and 44% of the deaths from meningococcal disease in LAC were caused by serogroups covered by the vaccine, and thus potentially preventable. Currently, a one-dose polysaccharide vaccine for meningococcal disease, effective against serogroups A,C,Y, and W-135, is available in the U.S., and research continues on a vaccine effective against serogroup B disease. Meningococcal vaccine is routinely given to military recruits, and is recommended for those with terminal complement deficiencies or asplenia, travelers to endemic or epidemic areas, and certain lab personnel. The Advisory Committee on Immunization Practices (ACIP) recommends that college students, especially freshmen and those living in dormitories, be informed about meningococcal disease and the benefits of the



vaccine.

ADDITIONAL RESOURCES

Centers For Disease Control and Prevention Website: <u>www.cdc.gov/mmwr/PDF/rr/rr4907.pdf</u> Prevention and control of meningococcal disease and college students: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR* 2000;49 (RR-7):1-20.

Centers For Disease Control and Prevention Website:

www.cdc.gov/epo/mmwr/preview/mmwrhtml/00046263.htm

Control and Prevention of Meningococcal Disease: Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR* 1997; 46(RR-5):1-51

Riedo FX, Plikaytis BD, Broome CV. Epidemiology and prevention of meningococcal disease. *Pediatr Infect Dis J* 1995;14:643-57.

Rosenstein NE, Perkins BA, Stephens DS, Popovic T, Hughes JM. Meningococcal disease. *N Engl J Med* 2001;344:1378-88.

MUMPS

CRUDE DATA			
Number of Cases	17		
Annual Incidence ^a			
LA County	0.2 ^b		
California	0.1		
United States	0.1		
Age at Diagnosis			
Mean	12		
Median	7		
Range	1-39 years		
Case Fatality			
LA County	0.0%		
United States	N/A		

a Cases per 100,000 population.

^b Rates based on less than 20 observations are unreliable.

DESCRIPTION

Mumps is a vaccine-preventable disease caused by an RNA paramyxovirus that is transmitted by direct contact with respiratory droplets. 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. Although single probable or confirmed cases are reportable, only outbreaks of two or more cases are investigated.

DISEASE ABSTRACT

• The incidence of mumps cases in LAC has been steadily declining since 1991 (Figure 1).

IMMUNIZATION RECOMMENDATIONS

• Two doses of mumps-containing vaccine, usually given as Measles-Mumps-Rubella (MMR),

are normally required to achieve immunity. The first dose is recommended at 12 months of age. The second dose can be given as early as four weeks after the first dose, but is usually given at ages 4 to 6 years. Vaccination is recommended for those who have no prior MMR, particularly if they are in a high-risk setting. Over 95% of those who receive the current live attenuated mumps vaccine develop immunity.

- Women should not become pregnant within one month of vaccination.
- Individuals who are severely immunocompromised for any reason should not be given MMR vaccine.

STRATIFIED DATA

Trends: Since 1991, the incidence of mumps has decreased by 78%.

Seasonality: Historically case reports have peaked during the winter and spring seasons. However, the onset date of 35% of the reported cases in 2001 was in May and June (Figure 3).

Age: Persons, aged less than 14 years, accounted for 71% of all reported cases in 2001. Of all the cases, four were between 1-4 years, eight were between 5-14 years, four were between 15-34 years, and one was between 35-44 years of age.

Sex: The male-to-female ratio of the cases was 1:1.3

Race/Ethnicity: About 70% of the reported mumps cases occurred among Latinos, although this ethnic group accounts for 45% of LAC population in 2001. Asians and Blacks accounted for one case each, and three cases were of an unspecified race/ethnicity.

Location: Cases were reported in six of the 8 Service Planning Areas, and ten of the 24 health districts. San Fernando (SPA2) and South Bay (SPA 8) accounted for 59% (n=10) of reported cases, with West Valley and Inglewood districts reporting 6 of these 10 cases (Figure 4).



COMMENTS

Most reported individual (non-outbreak related) and non-lab confirmed clinical mumps cases in highly immunized populations are most likely caused by other agents such as coxsackie and parainfluenza group 3 viruses. Recurrent parotitis can also result from non-infectious etiologies.



Cluster Identification: No cases were epidemiologically linked.

Vaccination Status: Of the 17 cases, only seven claimed to have received a first dose of MMR with an additional four cases having received a second dose of MMR. However, no documentation was obtained on any of the cases.

ADDITIONAL RESOURCES

National Immunization Program at: www.cdc.gov/ip

Immunization Action Coalition at: www.immunize.org

LAC Department of Health Services, Immunization Program at: www.lapublichealth.org/ip

PERTUSSIS (WHOOPING COUGH)

CRUDE DATA			
Number of Cases	103		
Annual Incidence ^a	100		
LA County	1.2		
California	2.0		
United States	1.2		
Age at Diagnosis			
Mean	6 years		
Median	2 months		
Range	1 day – 69 years		
Case Fatality			
LA County	2.0%		
United States	N/A		



a Cases per 100,000 population.

DESCRIPTION

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



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

DISEASE ABSTRACT

- The majority of reported cases in 2001 were reported in children less than one year of age. Infants less than two months of age accounted for more than half of these cases.
- Eleven cases had complications from pertussis including pneumonia, seizures, and death in two cases.
- More than half of the cases reported in 2001 were susceptible to pertussis by reason of absent or waning immunization.

IMMUNIZATION RECOMMENDATIONS

- A pertussis-containing vaccine should be given at 2 months, 4 months, 6 months, 15-18 months, and 4-6 years of age to provide protection against the disease.
- Immunity conferred by the pertussis component of the DTP/DTaP vaccine decreases over time, with little or no protection 5-10 years following the last dose.
- Currently, there is no pertussis vaccine booster available for adults.

STRATIFIED DATA

Seasonality: Typically, the summer months have the highest pertussis incidence in LAC (Figure 3). However, the number of 2001 cases steadily increased as the year progressed, peaking in mid summer and late fall/winter.

Age: As evidenced in previous years, approximately 69% (n=71) of reported cases in 2001 were reported in children less than one year of age. Infants less than two months of age and infants 2-6 months of age accounted for 58% and 38% of these cases, respectively. This is consistent with the national trend which has shown the highest annual pertussis incidence occurring among infants aged less than one year.

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

Race/Ethnicity: After adjusting for age, racial group rates were similar to the previous 5 year average except for Whites which increased 62% from the previous 5 year average (Figure 5). Since 1998, the rate among Whites has been steadily increasing.

Location: Number of cases per SPA ranged from 3 to 25. San Fernando SPA 2 and Metro SPA 4 reported most cases. The clustering of cases in specific geographic areas is influenced in part by the active reporting efforts of local hospitals.







COMMENTS

Because immunity induced by pertussis vaccine decreases over time, adolescents and adults can develop infection and serve as a source of transmission to infants who are not adequately immunized. Adults and adolescents with pertussis are more likely to have mild or atypical disease, so they often go undiagnosed. Future licensure and widespread use of an acellular pertussis booster vaccine for adolescents and adults should significantly decrease the incidence of pertussis in children, as well as its complications.

Trends: Pertussis incidence in LAC has peaked every 3-4 years since 1991 with the highest incidence in 30 years occurring in 1999 (n=238). The number of pertussis cases in 2001 was similar to that of 2000.

Laboratory Confirmation: Forty-five percent (n=46) of reported cases were laboratory confirmed by a positive culture or PCR. The other 55% were either epidemiologically linked to a confirmed case, or met the clinical criteria for pertussis.

Vaccination Status: Forty percent (n=41) of cases were less than two months of age and were too young to receive pertussis vaccine. Only 17% (n=18) of cases were 15 years of age or older; so even if they were fully immunized in early childhood, they would not have had complete immunity against pertussis in 2001. Thus, 57% percent of the cases reported in 2001 were susceptible to pertussis.

Twenty-six percent (n=27) of cases were between 2-6 months of age. Of these, 44% were up to date with pertussis vaccination for their age, but would not have developed full immunity against pertussis. Of the children who could have had full immunity from vaccination (7 months to 15 years old), 50 (75%) were fully up to date. Two of eight patient-cases were not immunized due to religious/philosophical exemptions, one child had medical exclusion, and the other five had undisclosed reasons.

Complications/Hospitalization: Sixty-one cases (59%) were hospitalized, with an average hospital stay of nine days (range 1-34 days). All but one of the hospitalized cases were less than one year of age. Of the eight cases who developed pneumonia, six were infants aged less than nine months and two were 15 years old. The one case with seizures was reported in a two-week-old infant. Two infants aged less than one year died from complications of pertussis.

ADDITIONAL RESOURCES

National Immunization Program at: www.cdc.gov/nip

Immunization Action Coalition at: www.immunize.org

LAC DHS, Immunization Program at: www.lapublichealth.org/ip



SAL	MON	ELL	OSIS
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CRUDE DATA			
Number of Cases	1,006		
Annual Incidence ^a			
LA County	11.3		
United States	14.2		
Age at Diagnosis			
Mean	25		
Median	19		
Range	<1-89 years		
Case Fatality			
LA County	1.0%		
United States	N/A		

a Cases per 100,000 population.

DESCRIPTION

Salmonellosis is caused by a bacterium, Salmonella enterica, of which there are at least 2,400 serotypes. It is transmitted by the fecaloral route, from animal or human, with or without intermediarv contamination of foodstuffs. The most common symptoms include fever, headache, abdominal pain, diarrhea, nausea, and sometimes vomiting. Occasionally the clinical course is that of enteric fever or septicemia. Asymptomatic infections occur. The incubation period is usually 12-36 hours for gastroenteritis, longer



and variable for other manifestations of salmonellosis. Communicability lasts as long as organisms are excreted, usually from 2-5 weeks, but may last for several months to years. Even 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, those who have had gastrointestinal surgery, achlorhydria, 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

• Six of ten identified Salmonella outbreaks in 2001 were caused by contaminated imported

produce.

- Five of the six produce related outbreaks were multi-county or multi-state outbreaks.
- Pulsed-field gel electrophoresis (PFGE) is now commonly used to identify clusters of the same serotype, as well as assist in the identification of outbreak-related cases.

STRATIFIED DATA

Trends: The incidence rate of reported salmonellosis cases in 2001 was 11.3 cases per 100,000 population. This is essentially unchanged from 2000, and is less than the national incidence rate of 14.2 per 100,000 population. A change occurred in the way cases were counted in 2001. Beginning in 2001, "presumptive cases", i.e. cases meeting a case definition and who have an epidemiological link to a lab confirmed case, were counted for the first time. A presumptive case is not lab confirmed. If the presumptive cases were not included in the total, the rate would have decreased to 10.8 per 100,000 in 2001

Serotypes of Salmonella: Despite a 4% decrease in *Salmonella* Enteritidis (SE) cases in 2001, SE still makes up 23% of all *Salmonella* isolates. Increases in *S.* Newport, *S.* Heidelberg, and *S.* Poona cases are partially explained by outbreaks. The reason for the increases in *S.* Oranienburg, *S.* Infantis, and *S.* Braenderup cases is unknown (Table 1).

	-		•••		
	N	2000 = 963 *	N	2001 = 949 *	Percent of Percent
Serotype	No.	Percent	No.	Percent	Change
Enteritidis	233	24	216	23	-4
Typhimurium **	175	18	159	17	-6
Newport	53	5.5	66	7.0	+27
Heidelberg	35	3.6	60	6.3	+75
Agona	28	2.9	24	2.5	-14
Thompson	34	3.5	21	2.2	-37
Oranienburg	16	1.7	20	2.1	+24
Infantis	17	1.8	20	2.1	+17
Braenderup	11	1.1	19	2.0	+82
Poona	13	1.3	18	1.9	+46

Table 1. Most Frequent Salmonella Serotypes,	LAC,	2000 -	· 2001
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* Denominator N = only those isolates which were serotyped.

** Includes S. Typhimurium var. Copenhagen and degraded form.

Seasonality: In 2001, early peaks were seen in May and June, due in part to produce-related outbreaks. The traditional late summer/early fall peak did not occur (Figure 2).

Age: As in past years, the highest age-specific rates of infection occurred among infants, aged less than 1 year (71.8 per 100,000 population) followed by children aged 1 - 4 years (35.1 per 100,000 population).

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

Race/Ethnicity: The highest age-adjusted rate was in Latinos (12.3 cases per 100,000), followed by Asians (10.6), Whites (9.8), and Blacks (9.3) (Figure 4). The rates rose the most in Latinos; 3 outbreaks in 2002 involved mostly Latinos.

Location: East LA Health District had the highest incidence rate (20.3 per 100,000 population), followed by Southwest (15.8) and Foothill (14.6). By Service Planning Area, SPA 6 (13.6) and SPA 4 (12.4) had rates higher than the county average, while SPA 1 had the lowest rate (7.5 per 100,000).

COMMENTS

During 2002 there were 10 reported outbreaks of salmonellosis in LAC. Outbreak-related cases accounted for 5% of all culture-confirmed salmonellosis cases and 8% of total cases reported in 2001. In vears since 1994, Salmonella most Enteritidis (SE) was the etiologic agent identified in the majority of outbreaks in LAC. In 2001, SE caused only one outbreak. SE continues to be the predominant serotype in LAC despite having decreased by 4%. Since 1995, fresh produce, such as alfalfa sprouts and cantaloupe, has increasingly been recognized as a source of salmonellosis. In 2002, 6 of the 10 salmonellosis outbreaks were caused by contaminated produce; 5 of these were multi-state or multi-county outbreaks. All were attributed to produce imported from Mexico. Most of these outbreaks occurred in late spring or early summer, and this may be one reason why cases peaked earlier than the usual late summer and early fall increase. The use of PFGE and the sharing of PFGE patterns with other laboratories through PulseNet, the national molecular subtyping network for foodborne disease, has helped identify related clusters and outbreaks within LAC, as well as multi-county and multi-state outbreaks caused by a common food product.

Salmonellosis was reported as a contributing cause of death in 10 people, all of whom had underlying health problems such as cancer, renal disease, diabetes, AIDS and chronic liver disease. Nine of 10 were hospitalized with symptoms probably caused by salmonellosis: 5 had sepsis with positive blood cultures; 2 had positive urine cultures with urosepsis; 3 had a positive stool





LAC

Total

8

2

1

3

Service Planning Area (SPA)

cultures with diarrhea.

PREVENTION

Each report 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 eggs and meats, not washing hands and/or cutting boards after handling raw poultry or meat, and not maintaining food at proper temperature to prevent bacterial growth. These investigations demonstrate a need for public education on proper handling and preparation of animal-derived foods, especially eggs. Also, health education targeted at specific racial/ethnic groups is necessary; for example, 47% of salmonellosis cases having reptile contact were Hispanic. In addition, now that fresh produce has been recognized as a source of salmonellosis (6 outbreaks in LAC for 2001 were associated with fresh produce), washing of fresh fruits and vegetables prior to consumption is advised.

Table 2: Salmonellosis Outbreaks in LAC, 2001							
Onset	Outbreak	Total	Culture	_	Suspect	Suspect	
Month	Setting	# III	Positive	Serotype	Vehicle	Source	
Feb	Private Home	6	2	STVC	Biltong (South African style beef jerky)	Unknown	
Feb*	Various	7	7	SK	Alfalfa Sprouts	Alfalfa Sprouts	
Mar*	Various	3	3	E1:e,h:-	Cantaloupe	Cantaloupe	
Apr	Preschool	12	3	SH	Person to Person	Unknown	
Apr*	Various	7	7	SP	Cantaloupe	Cantaloupe	
May	Various	8	8	SN	Cilantro	Cilantro	
June*	Various	6	6	SP	Pre-cut Melon	Pre-cut Melon	
June**	Private Home (Catered)	21	2	SE	Chicken Salad	Unknown	
Sept*	Various	6	6	SS	Green Grapes	Green Grapes	
Oct	Various	8	8	SI	Unknown	Unknown	
TOTAL	_	84	52				

SE = Salmonella Enteritidis

SH= Salmonella Heidelberg

SI= Salmonella Istanbul

SK= Salmonella Kottbus

SN = Salmonella Newport

SP = Salmonella Poona

SS= Salmonella Senftenberg

STVC = Salmonella Typhimurium var Copenhagen

* Multi-State or Multi-County outbreak; number of cases listed represents LAC cases only.

** This outbreak involved both Pasadena and LAC jurisdictions. The 2 confirmed cases are Pasadena residents; 14 presumptive

cases are LAC residents.

*** Multi-County, Multi-State, outbreaks with no specific setting.



SHIGELLOSIS

CRUDI		
Number of Cases	684	
LA County	7.7	Inciden LA
California	6.2	25
United States	7.1	8 20
Age at Diagnosis		
Mean	21	
Median	16	
Range	<1-90 years	<u> </u>
Case Fatality		0 1992 1993
LA County	0.0%	
United States	N/A	



a Cases per 100,000 population.

DESCRIPTION

Shigellosis is caused by a gram-negative bacillus with four serogroups: *Shigella dysenteriae* (group A), *S. flexneri* (group B), *S. boydii* (group C) and *S. sonnei* (group D). 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 depending on the serogroup. In general, the elderly, the immunocompromised, and the malnourished are more susceptible to severe disease outcomes.

DISEASE ABSTRACT

- In general, shigellosis rates have continued to fall.
- In LAC, most cases occur sporadically. No outbreaks were seen in childcare and institutions in 2001.

STRATIFIED DATA

Trends: Shigellosis rates declined between 1994 and 1999, and then increased in 2000 (9.0 per 100,000). This increase was attributed to the large multi-state outbreak that year and high rates of disease in men who have sex with men (MSM). Although there continues to be high rates among MSM, in 2001 the mere that the descent of the second sec



2001, the overall LAC rate decreased 18% to a rate comparable to the 1999 rate.

Serotypes: S. sonnei continues to be the most common serotype seen in LAC followed by S. flexneri (Figure No 2).

Seasonality: As in previous years, the highest number of cases was seen in spring and summer, peaking in August (Figure 3).

Age: Although the rate for children aged 1-4 years is lower than the five-year average, it is still higher than any other age group. Rates for persons aged 35-44 years and 45-54 years are higher than the previous 5 year average and may be due to cases among MSM.

Race/Ethnicity: In 2001 Latino children aged 1-4 years had the highest age adjusted rate. This is probably due to more travel to endemic countries, families with more small children and overcrowded living conditions.

Sex: The male-to-female rate ratio for all shigellosis was 1.25:1. For cases 14 years and younger, the ratio was 0.9:1. For cases 15 years and older, the ratio was 2:1; this difference may be due to cases among MSM.

Location: The rate for SPA 4 was significantly higher than county average. The high incidence in SPA 4 is due to the number of MSM cases reported in the Hollywood Wilshire health district (33 cases).

Severity of Illness: Fourteen percent of reported shigellosis cases were hospitalized. There were no shigellosis related deaths reported in 2001.

Risk Factors: Exposure during travel (18%) and exposure to a case inside or outside the household (20%) were the most commonly reported potential sources. Other reported potential risks included participation in an outdoor activity (e.g., hiking, camping, swimming) and drinking untreated water. Swimming or wading in areas not designated for this activity was associated with several cases of shigellosis.



COMMENTS

Indirect exposure by consumption of food contaminated by an ill individual is a potential source. There were two restaurant-related shigellosis outbreaks reported in 2001. Both were caused by *S. sonnei* (described under Foodborne Outbreaks in the Disease Outbreaks section of this report).

Certain sexual practices – those in which there is direct contact with fecal material – can be a potential source of infection. In 2001, *S. sonnei* (81%) continued to be the predominant serotype among MSM cases. A cluster of PFGE-related *S. sonnei* cases in MSM occurred in March 2001. No links were established among these cases.

Two percent of LAC case isolates were serotyped as Provisional SH108. This unusual serotype has been associated with travel or having visitors from Mexico.

Prevention: Careful handwashing is important in preventing this disease. Children should not be allowed to swim or wade while ill with diarrhea. Swimming or wading in areas not designated for such activities should be avoided, especially in areas where there are no toileting or handwashing facilities. In LAC, cases and symptomatic contacts in sensitive occupations or situations (e.g., food handling, healthcare workers) are routinely removed from work or the situation until they are negative on stool specimens tested in the Public Health Laboratory.

ADDITIONAL RESOURCES

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



TYPHOID FEVER, ACUTE

CRUDE DATA					
Number of Cases	17				
Annual Incidence ^a					
LA County	0.2 ^b				
California	0.2				
United States	0.1				
Age at Diagnosis					
Mean	32				
Median	32				
Range	2-90 years				
Case Fatality					
LA County	0.0%				
United States	N/A				



a Cases per 100,000 population.

b 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 common than diarrhea), bradycardia, enlargement of the spleen, and rose spots on the trunk. Humans are the only known reservoir for *S. typhi*.

DISEASE ABSTRACT

- In LAC, 82% of the acute typhoid fever cases were associated with recent immigration and foreign travel.
- Most cases were reported among Asians, followed by Latinos.
- In 2001, no cases were linked to previously unknown carriers.

STRATIFIED DATA

Trends: The rate of reported typhoid fever cases remained steady after decreasing for ten years. Annual incidence had declined from 0.67 in 1990 to 0.22 in 1999. In 2000, the incidence rate was 0.23. In 2001, the rate was 0.18.



Seasonality: In LAC, the majority of cases (65%) had onset in Spring. Most cases occur in late

spring and summer, coinciding with holidays and school vacation (Figure 2).

Age: In 2001, persons aged 15-34 years continued to have the highest incidence (Figure 3). This may be because persons in this age group travel or immigrate more.

Sex: The male-to-female rate ratio was 1.25:1. This slight male preponderance is typical.

Race/Ethnicity: Acute typhoid fever cases continue to be seen primarily in Asians, who accounted for 59% of cases (Figure 4). Latinos had the second highest incidence with 29% of cases. This trend may be related to individuals traveling to their countries of origin (see comments related to travel).

Location: Eighty-eight percent of cases were seen in SPAs 6, 5 and 4.

COMMENTS

Fourteen cases (82%) were associated with travel to endemic areas outside the US. Of these cases, 9 apparently acquired disease in Asia and 5 acquired disease in Mexico or Central America.

Three cases (18%) denied foreign travel or having recent visitors from areas outside the US. It is presumed they became infected in LAC. Household contacts were tested for *S. typhi* and no source of infection was identified.

PREVENTION

Handwashing after using the toilet, before preparing or serving food, and before and after caring for others is important in preventing the spread of typhoid. When traveling to locations where sanitary practices are uncertain, foods should be thoroughly cooked and served hot; bottled water should be used for drinking as well as for brushing teeth and making ice. Vaccination should be considered when traveling in areas of high endemicity.

ADDITIONAL RESOURCES

General disease information is available at: www.cdc.gov/ncidod/dbmd/diseaseinfo/typhoidfever_g.htm

Traveler's health information is available at: www.cdc.gov./travel/diseases/typhoid.htm




TYPHOID FEVER, CARRIER

CRUDE DATA				
Number of New				
Carriers	1			
Annual Incidence ^a				
LA County	N/A			
United States	N/A			
Age at Diagnosis				
Mean	N/A			
Median	N/A			
Range	N/A			
Case Fatality				
LA County	0.0%			
United States	N/A			



a Cases per 100,000 population.

DESCRIPTION

The chronic typhoid carrier state can occur after symptomatic or subclinical infections of *Salmonella typhi*. Among untreated cases, 10% will shed bacteria for three months after initial onset of symptoms and 2-5% will become chronic carriers. The chronic carrier state occurs most commonly among women in middle age.

DISEASE ABSTRACT

- During 2001, a total of 18 carriers were under case management in LAC. Only one new typhoid carrier was identified in 2001.
- Four carriers were successfully treated and cleared with antibiotics.
- Two previously known carriers moved into LAC from other jurisdictions.

COMMENTS

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

ADDITIONAL RESOURCES

Disease Information is available at: www.cdc.gov/ncidod/dbmd/diseaseinfo/typhoidfever_g.htm

TYPHUS

CRUDE DATA				
Number of Cases	8			
Annual Incidence ^a	0			
LA County	N/A ^b			
United States	N/A			
Age at Diagnosis				
Mean	42			
Median	44			
Range	6-73 years			
Case Fatality				
LA County	0.0%			
United States	N/A			

a Cases per 100,000 population.

b Not calculated. Rates based on less than 20 observations are unreliable.

DESCRIPTION

Typhus (murine typhus, endemic typhus) is caused by the bacteria, Rickettsia typhi and *R. felis.* They are transmitted through the bite or contact with feces of an infected flea. Most reported cases of typhus live in the foothills of Reservoir central LAC. animals are predominantly rats and other small mammals that live in areas with heavy foliage. Symptoms include fever, severe headache, chills, and myalgia. A fine, macular rash may appear three to five days after onset. complications Occasionally, such as





pneumonia or hepatitis may occur. Fatalities are uncommon, occurring in less than 1% of cases. The disease is mild in young children. Typhus is not vaccine preventable, but can be treated with antibiotics.

DISEASE ABSTRACT

- Cases occur more often in summer and fall. In 2001, four of eight reported cases occurred in June and July.
- Seventy-five percent of cases (6/8) were hospitalized.
- There were 4 male cases and 4 female cases.
- Whites led with 4 cases followed by Latinos with 3, there was 1 Asian case and one case of unknown race.

LOCATION

In 2001 four cases were residents in Alhambra, two lived in Central, and two resided in the Northeast health districts. Typhus is endemic in the foothills of central LAC. Cases have been reported from Silver Lake, Echo Park, Eagle Rock, Glendale Hills, Pasadena and Altadena. Animals from these areas have tested positive for typhus group *Rickettsia*. The reasons for this localized endemic area are unclear.

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. Four of the six cases recalled being bitten by a flea and most did observe small mammals such as rats, opossums, dogs and cats in their yards, and thus had exposure to animals that carry fleas. One case reported contact with an opossum. Typhus cannot be transmitted from person to person.

COMMENTS

Each case of endemic typhus is carefully interviewed regarding potential exposures. If possible, field studies of the property where exposure occurred and surrounding areas in the neighborhood are conducted. 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 endemic typhus difficult. Thus, diagnosis of endemic typhus depends on the clinical acumen of the treating physician, and is often confirmed after the patient has recovered. Accurate reporting of typhus or suspect typhus cases is important to identify endemic areas in LAC which can be monitored for the presence of disease in the animal populations and to institute control measures. Treatment with antibiotics hastens recovery and lessens the chance of complications.

PREVENTION

Typhus infection can be prevented through flea control measures implemented on pets and around the yard. Foliage in the yard should be kept trim 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

Azad AF, Radulovic S, Higgins JA, Noden BH and Troyer JM. Flea-borne rickettsioses: ecologic considerations. *Emerg Infect Dis* 1997;3:319-27.

Sorvillo FJ, Gondo B, Emmons R, et al. A suburban focus of endemic typhus in Los Angeles County: association with seropositive domestic cats and opossums. *Am J Trop Med Hyg* 1993;48:269-73.

Williams SG, Sacci JB Jr, Schriefer ME, et al. Typhus and typhuslike rickettsiae associated with opossums and their fleas in Los Angeles County, California. *J Clin Microbiol* 1992;30:1758-62.

Acute Communicable Disease Control website: <u>www.lapublichealth.org/acd/vectormurine.htm</u>

VIBRIOSIS

CRUD	E DATA	Figure 1
Number of Cases Annual Incidence ^a	15	Selected Vibrio species Cases by Year of Onset LAC, 1993 - 2001*
LA County	N/A ^b	25
United States	N/A ^b	
Age at Diagnosis		5 1 1 1
Mean	42	
Median	48	
Range	16-64 years	1993 1994 1995 1996 1997 1998 1999 2000 2001
Case Fatality		V. vulnificus – V. parahaemolyticus
LA County	4.0% ^c	V. cholera-01
United States	varies by species	*Data for 1992 not available.

a Cases per 100,000 population.

 $^{\mbox{b}}$ Not calculated. Rates based on less than 20 observations are unreliable.

^C Deaths from *V. vulnificus* - this species had a 50% case fatality.

DESCRIPTION

The genus *Vibrio* consists of gram-negative, curved, motile rods, and contains about a dozen species known to cause illness in man. Transmission is most often through ingestion of the organism via foodborne route, but also from contact with seawater–as in a break in the skin. Presenting symptoms vary by infecting species and mode of transmission. Cholera vaccine was once available in the US, but had not been recommended, due to limited protectiveness and potential for side effects. It is no longer available in the US. The vibrio species of greatest public health importance are: *V. vulnificus*, presenting as a primary septicemia and are often associated with oysters harvested in the Gulf of Mexico; *V. cholerae*- O1, most often travel associated; and *V. parahaemolyticus* usually seafood associated, both presenting as a gastrointestinal illness.

DISEASE ABSTRACT

- Fifteen cases of Vibrio species were reported in 2001, compared to the 18 cases the previous year.
- Eight cases of Vibrio vulnificus were reported, four died.

STRATIFIED DATA

Trends: Looking at the last ten years, case numbers of Vibrio infections peaked in 1998 with 36 reports. The reported cases of *Vibrio vulnificus* peaked in 2001, the highest number in a 10 year period.

Seasonality: Seventy- three percent (11/15) of Vibrio cases occurred in May and September. Historically, cases of vibrio infections increase during the warmer summer months.

Age: All cases were adults (Table 1).

Sex: Males cases were predominant across Vibrio species, especially for *Vibrio vulnificus*, (Table 1).

Race/ethnicity: Reported cases were most often in Latinos (Table 1).

Severity: Four fatal Vibrio cases were reported, all V. vulnificus.

Species	Race	Mean Age (range)	Sex Ratio M:F
V. parahaemolyticus (n=3)	Latino (1), Asian (1), Unknown (1)	48 years (31 - 59)	2:1
<i>V. cholerae</i> - O1 (n=1)	Latino (1)	27 years	1:0
V. vulnificus (N=8)	Latino (8)	51 years (36 - 64)	8:0
V. other species* (n=3)	Latino (1), Asian (2)	32 years (16 - 58)	2:1

Table 1: Vibrio Cases by Species, Race, Age and Sex – LAC, 2001

* Other species = V. alginolyticus (1), V. mimicus (1) and V. fluvialis (1).

Species specific risk factors:

- Vibrio vulnificus

Food history risk factor data was available on 6 of the 8 cases. All 6 of these cases reported seafood exposure and four could be specifically linked to Gulf Coast oyster consumption. All cases of *V. vulnificus* were in adult Latino males. Pre-existing medical risk factor data was documented in seven cases - six with liver disease, one diabetic. One individual had unknown prior medical history. Investigation of *V. vulnificus* can be hampered by lack of history, as cases may be too ill to give a reliable epidemiological history.

- Vibrio cholerae – O1

The single reported case of *V. cholerae* - O1 was cryptic and the case denied any risk factor data. In addition, the organism was isolated from blood rather than the usual stool culture.

COMMENTS

In LAC, risk from vibrioses can be prevented or reduced by avoiding seawater contamination of food (especially raw fish and shellfish) or drink. Infection with *V. vulnificus* is a particular risk for persons with pre-existing liver disease, frequently leading to soft tissue invasion, limb amputation, and a high case fatality. Adult males may be more at risk for Vibrio infections because of their tendency to engage in behaviors exposing them to seawater contamination or higher levels of raw or partially cooked seafood consumption, especially oysters.

PREVENTION

In LAC, risk from vibrioses can be prevented or reduced by avoiding seawater contamination of food (especially raw fish and shellfish) or drink.

General information on Vibrio infections can be found on web sites sponsored by the LAC Health Department, FDA and CDC.

Recent publication on V. vulnificus in Los Angeles: Mouzin E, Mascola L, Tormey M, et al.: Prevention of Vibrio vulnificus infections. Assessment of regulatory educational strategies. JAMA 1997 Aug 20; 278(7):576-578.



COMMUNITY-ACQUIRED DISEASE OUTBREAKS

ABSTRACT

- In 2001, 48 of 174 reported communityacquired outbreaks investigated were foodborne (see Foodborne Outbreak section). The remaining 127 community outbreaks consisted of 1,160 illnesses.
- Settings of community-acquired outbreaks primarily include schools and pre-schools.

DATA

Disease outbreaks are defined as clusters of illness that occur in a similar time or place, or unusual numbers of disease cases above baseline in a specified area. Depending on the nature of the outbreak, investigation responsibility is held by either ACDC or by Community Health Services with ACDC providing consultation. Figure 94 shows that since 1997 the annual number of reported cases has varied in unison with the annual number of outbreaks.

Most of the reported community outbreaks in LAC were due to varicella (35%) and the ectoparasites scabies and pediculosis (24%) (Figure 2).

Varicella and Gastroenteritis (GE) of undetermined etiology were the diseases with the highest number of cases. Also, GE outbreaks of viral & undetermined etiology had the highest number of cases per outbreak (Table 1).

The most common settings for illness transmission were schools (kindergarten or higher), accounting for 64%, and preschools (12%) (Figure 3).



Disease	Number of Outbreaks	Number of Cases	Avg. Cases per Outbreak	Range
Varicella	45	429	9	2-41
Pediculosis	14	136	10	2-25
Scabies	17	86	5	2-9
Hand, foot & mouth disease	10	76	7	2-21
Ringworm	10	47	5	1-10
GE illness ^a – undertermined etiology	11	267	24	4-108
GE illness – Salmonellosis	1	12	12	12
GE illness – Norwalk-like virus	1	10	10	10
Fifth disease	6	32	5	2-10
Scarlet Fever/Strep throat	7	37	5	4-8
Pertussis	1	4	4	4
Other ^b	4	24	2	2-5
Total	127	1,160	8.6	

Table 1. Community Cubicats by Disease Diagnosis, LAC, 200	Table 1. Communit	y Outbreaks b	y Disease Di	agnosis,	LAC, 2001
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^a Excludes foodborne outbreaks.
 ^b Other includes conjunctivitis (pink eye) and unspecified rash.

	Group		Pre-	Day-		
Disease	Home	School ^a	School	care	Other ^b	TOTAL
Pediculosis	0	12	2	0	0	14
Scabies	8	2	1	2	4	17
Varicella	3	39	3	0	0	45
Hand, foot and mouth disease	0	4	2	4	0	10
Fungal diseases	0	8	1	1	0	10
GE illness – viral GE	2	1	1	0	1	5
GE illness – bacterial etiology	0	0	1	0	0	1
GE illness – unknown etiology	0	2	3	2	0	7
Fifth disease	0	6	0	0	0	6
Scarlet fever / Strep throat	0	5	1	1	0	7
Pertussis	0	1	0	0	0	1
Other	1	1	0	2	0	4
					_	
TOTAL	14	81	15	12	5	127

Table 2. Community Outbreaks: Disease Diagnosis by Setting, LAC, 2001

^a Includes elementary school, high school, and a school for the developmentally disabled. ^b Includes jail, shelter and workplace.

COMMENTS

In previous years, the ectoparasites pediculosis and scabies were the predominant outbreak types reported in schools (41% in 1998). Varicella accounted for 27% of school outbreaks in 1998. In 1999, varicella (32%) surpassed ectoparasites (23%) as the leading outbreak type reported among schools. This dominance in varicella outbreaks among schools is seen again in 2001: varicella 35%, ectoparasites 24%. However, there was a drop in the total number of community-acquired outbreaks reported for 2001; only 127 were reported this year compared to 156 in 2000. This may be due to the increasing use of varicella vaccine for school-age children.

FOODBORNE OUTBREAKS

DESCRIPTION

Foodborne outbreaks are caused by a variety of bacterial, viral, and parasitic pathogens and toxic substances. To be considered a foodborne outbreak, CDC requires at minimum "the occurrence of two or more cases of a similar illness resulting from the ingestion of a common food."* There were no waterborne outbreaks reported in 2001. There were no foodborne outbreaks in health facilities.

The system used by LAC DHS for detection of foodborne outbreaks begins with the Foodborne Illness Report (FBIR), which 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, а thorough inspection of the facility is conducted. In 2001, 75% of FBIRs resulted in investigation of the facility; this is often sufficient public health additional foodborne action to prevent illnesses.

The ACDC Food and Water Safety Program also reviews all FBIRs. Typically, an epidemiologic investigation will be initiated there when are illnesses in multiple households, multiple reports from the same establishment with similar symptoms in a short period of time, and large events implicated with the potential for others to become ill.

DISEASE ABSTRACT

• In 2001, the number of outbreaks investigated was greater than in any of the previous four



^{*} Centers for Disease Control and Prevention: Surveillance for Foodborne - Disease Outbreaks - United States, 988-1992. MMWR 1996;45(SS-5):58.

years, whereas the overall number of cases of individual illness was lower than in each of the previous four years (Figure 1).

- A food item was more likely to be implicated for outbreaks involving a confirmed or suspected bacterial toxin or bacteria compared to outbreaks involving a confirmed or suspected viral agent or an unknown etiologic agent (Figure 5).
- Probable contributing factors were unable to be determined for over half of the outbreaks investigated (Figure 8).

DATA

Overview: Of the 1,466 FBIRs in 2001 from consumers eating food from establishments located in LAC, F&M investigated 1,093 (75%). Of the total FBIRs received, 511 (35%) were potential outbreaks, 356 (24%) were single reports of multiple illnesses within one household, 127 (9%) were single reports of two or more illnesses in multiple households, and 28 multiple reports (2%) were for same establishment. As always, ACDC investigates those foodborne outbreaks with the greatest public health importance. In 2001, ACDC investigated 48 outbreaks representing 590 cases of foodborne illness (Table 1; Figure 1). These outbreaks were caused by a variety of pathogens (Figure 2). The mean number of cases per foodborne outbreak was 12 (range 2 -56).

Seasonality: In 2001 there were peaks of foodborne outbreaks in February, April, and June (Figure 3).

Agent: Typical foodborne pathogens can be categorized according to characteristics of illness they have in common. The categories used in this report include five types of pathogens. Bacterial agents that cause infection include Salmonella, Shigella, Campvlobacter, Vibrio, and E.coli. Bacteria that elaborate toxins include Staphylococcus aureus, Clostridium perfringens, and Bacillus cereus. Viral gastroenteritis (Viral GE) includes the Norwalklike viruses (NLV) of the Caliciviridae family. The "other" category includes Hepatitis A virus, fish poisonings, and enteric parasites.



A specific pathogen was laboratory confirmed in 46% and epidemiologically suspected in 27% of foodborne outbreaks investigated (Figure 4). Nine outbreaks, all bacterial, were identified by routine disease surveillance (Table 2). Laboratory testing was conducted in 30 of the 48 foodborne outbreaks (63%). Reasons for no laboratory testing include lack of cooperation (n=9); unclear epidemiologic picture (n=2); and delayed notification (n=7).

Implicated Food Vehicles: In 60% of foodborne outbreak investigations, a food vehicle was epidemiologically implicated (Figure 5); an organism was isolated and confirmed from a food item in 5 of those outbreaks. In suspected bacterial outbreaks, the food vehicle was identified 93% of the time, while a food item was identified in only 31% of viral GE outbreaks. Implicated food vehicles are categorized in Figure 6. The largest proportion of outbreaks was caused by the meat/poultry/fish category (48%), followed by fruit/vegetable (24%). Two outbreaks had multiple implicated food items.

Food Preparation Site: Outbreak-associated food was most often prepared by a restaurant (69%) or was produce obtained directly from a farm (13%) (Figure 7).

Contributing Factors: In 22 of 48 outbreak investigations, probable contributing factors of the cause of the outbreak were found (Figure 8). More than one factor could be cited for each outbreak. The most frequent factors identified were contaminated raw products (27%), improper holding temperature (29%), and improperly cleaned utensils/equipment (8%). On average, 2 contributing factors were reported per outbreak.

Outbreak Location: The most common eating places for foodborne outbreaks were restaurants (44%), followed by private homes (25%) (Figure 9).

The geographic distribution of the outbreaks by SPA is summarized in Table 3. SPA 2 had the most foodborne outbreaks (15); SPA 6 had the least (1). Six outbreaks involved multiple







jurisdictions, one of which involved multiple SPAs, four of which involved multiple states and one was multi-national.

Viral GE Summary:

Thirteen of the 48 foodborne outbreaks (27%) investigated in 2001 were GE hypothesized to be caused by viruses. Laboratory testing was completed on 10 of these viral GE outbreaks, with six testing positive for NLV. Viral GE was suspected in the remaining seven outbreaks based on symptoms, incubation, duration of symptoms, secondary cases in households, and/or negative bacterial test results. A majority of the suspect viral GE outbreaks occurred in April. The mean number of cases per outbreak for 2001 was 15 cases. The average of the median duration of symptoms for each outbreak was 2.4 days. A majority of the viral GE outbreaks (69%) had an undetermined implicated food item. Restaurants were the most common food source for the 2001 viral GE outbreaks (69%). In 77% of the outbreaks, contributing factors were undetermined.

COMMENTS

Since 1999, the LAC Public Health Laboratory has been testing human specimens for NLV using the reverse transcription-polymerase chain reaction (RT-PCR) method. This method is still considered to be experimental and is only used to diagnose outbreaks as a whole, not for individual patients. There has been a marked increase in the number of viral GE and confirmed NLV outbreaks since 1999.

PulseNet is a public health network sponsored by the CDC that uses the collaboration of laboratories and health departments at local, state, and federal levels to detect outbreaks through pulsed-field gel electrophoresis (PFGE) of pathogens. The PFGE results are monitored for matching pathogen strains of various etiologic agents. When matches are detected, an investigation may be initiated. In addition, a solitary case occurring locally can be linked to a larger, previously identified outbreak occurring on a wider geographical scale. LAC was involved in the investigation of 5 of these multi-jurisdictional foodborne outbreaks in 2001.

Mild symptoms, long incubation periods, and poor public and medical community awareness of public health procedures may contribute to under-reporting of foodborne disease.

	Confirmed/			
Agent	Suspected	Serotype	Cases*	Jurisdictions / SPA
C. perfringens	Lab Confirmed		9	2
C. perfringens	Lab Confirmed		35	3
Campylobacter	Lab Confirmed	jejuni	3	5
Listeria	Lab Confirmed	monocytogenes	12	2
NLV	Lab Confirmed		38	3
NLV	Lab confirmed		7	3
NLV	Lab Confirmed		13	1
NLV	Lab Confirmed		10	2
NLV	Lab Confirmed		25	5
NLV	Lab Confirmed		11	5
Salmonella	Lab Confirmed	Poona	6	Multi-state
Salmonella	Lab Confirmed	Kotthus	7	Multi-state
Salmonella	Lab Confirmed	Senftenhera	6	Multi-state
Salmonella	Lab Confirmed	Poona	7	Multi-state/Canada
Salmonella	Lab Confirmed		1	Multi-state/Canada
Salmonella	Lab Confirmed		5	Nulli-State/Carlada
Salmonolla	Lab Confirmed	optoriditio	15	ວ ວ
Salmonolla	Lab Confirmed	Nowport	10	Multi cpo 2
Sambroid	Lab Confirmed	Newport	0	wuiii-spa 3
Scombroid	Lab Confirmed	connoi	∠ 10	8 5
Shigella	Lab Confirmed	sonnei	10	5
Shiyella	Lab confirmed	Sonnei	9	2
Staph			0	7
Bacterial	Suspected		3	5
Bacterial Taxin	Suspected		11	4
Bacterial Toxin	Suspected		31	2
Bacterial Toxin	Suspected		4	2
Bacterial Toxin	Suspected		4	3
Bacterial Toxin	Suspected		30	8
Viral Gastroenteritis	Suspected		5	4
Viral Gastroenteritis	Suspected		10	6
Viral Gastroenteritis	Suspected		8	1
Viral Gastroenteritis	Suspected		10	2
Viral Gastroenteritis	Suspected		19	8
Viral Gastroenteritis	Suspected		10	2
Viral Gastroenteritis	Suspected		30	2
Unknown			8	4
Unknown			4	2
Unknown			/	5
Unknown			12	8
Unknown			8	3
Unknown			56	1
Unknown			6	2
Unknown			9	2
Unknown			4	2
Unknown			26	2
Unknown			6	8
Unknown			6	2
Unknown			5	3

Table 1. Foodborne Outbreaks in LAC, 2001 (N=48)

* Include only LAC cases.

Outbreaks by Suspect/Confirmed Etiologic Agent "Type," 2001						
	Bacterial	Bacterial Toxin	Norwalk- Like Virus	Unknown	Total	
# OBs Investigated	14	8	13	13	48	
# OBs Tested	14	5	10	1	30	
# OBs Agent Confirmed	12	4	6		22	
# OBs Identified By PHL Surveillance	9				9	

Table 2. LAC Foodborne OutbreaksLaboratory Summary:Outbreaks by Suspect/Confirmed Etiologic Agent "Type," 2001

 Table 3: Frequency of Foodborne Outbreaks by

 Service Planning Area (SPA)/Jurisdictions, 2001

SPA	Frequency	Percent
1	2	4
2	16	32
3	7	15
4	3	6
5	7	15
6	1	2
7	2	4
8	5	10
Multi-SPA	1	2
Multi-State	3	2
Multi-State/Canada	2	8
Total	48	100

ADDITIONAL RESOURCES

LAC Communicable Disease Reporting System: Hotline: (888) 397-3993 Faxline: (888) 397-3779

LAC DHS Public Health Programs and Services: www.lapublichealth.org

LAC DHS Foodborne Disease Section in B-73 Manual: www.lapublichealth.org/acd/procs/b73/b73fh.pdf

CDC Foodborne and Diarrheal Diseases Branch: www.cdc.gov/ncidod/dbmd/foodborne/index.htm

Outbreak Response and Surveillance Unit: www.cdc.gov/ncidod/dbmd/outbreak/

FoodNet: <u>www.cdc.gov/foodnet/</u>

FDA - Center for Food Safety and Applied Nutrition: www.vm.cfsan.fda.gov/list.html

Gateway to Government Food Safety Information: www.FoodSafety.gov

HEALTHCARE ASSOCIATED OUTBREAKS

DEFINITION

Outbreaks in healthcare facilities are defined as clusters of nosocomial (health-facility acquired) or home-healthcare-associated infections related in time and place, or occurring above a baseline or threshold level for a facility, specific unit, or ward. Baseline is defined as what is normally observed in a particular setting.

ABSTRACT

- More health facility outbreaks were reported in 2001 compared to 2000, but the number was still lower than those reported for 1997-1999 (Figure 1; Table 1).
- Skilled nursing facilities (SNFs) in 2001 contributed to most of the increase from 2000.



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The most common cause of reported outbreaks was scabies.

Type of Healthcare Facility	1997	1998	1999	2000	2001
	No.	No.	No.	No.	No.
Acute Care Hospitals	31	24	18	20	19
Sub-acute Care					
Dialysis Facilities	0	0	0	0	1
Home Health Agencies	2	1	0	0	0
Intermediate Care/Psych	3	1	4	1	0
Skilled Nursing Facilities	40	41	41	19	35
Total	70	67	C 2	40	FF
lotal	76	07	03	40	55

Table 1. Reported Outbreaks Occurring in Healthcare Facilities LAC, 1997- 2001

Acute Care Hospitals:

In acute care hospitals, 19 outbreaks were reported in 2001 (Table 1), which was very similar to the incidence seen for the previous two years. Two hospitals reported more than one outbreak. Nosocomial scabies outbreaks fluctuated from 3 in 1998, to a low of 0 in 2000, increasing to 5 outbreaks in 2001 (Table 2). In 2001, patients with scabies were 31% of the cases reported from all the acute care hospital outbreaks, which was similar to the 35% seen in 1998. In 2001, the etiologic agents contributing the largest number of cases in acute care outbreaks were *Sarcoptes scabiei* (n=73), *Acinetobacter baumanni* (n=34), *Mycobacterium fortuitum* (n=22), and *Burkholderia cepacia* (n=20). There were also two outbreaks involving 48 cases with surgical site infections caused by multiple organisms.

	Number of	Number of
Disease/Condition/Etiologic Agent	Outbreaks	Cases
Scabios	5	73
Asperaillosis	2	73 Q
Rurkholderia cenacia	2	20
Surgical sites infections - mixed organisms	2	48
Acinetobacter baumanni	1	34
Enterobacter cloacae	1	5
Enterobacter gergoviae	1	10
Invasive Group A Streptococcus	1	4
Methicillin-resistant Staphylococcus aureus	1	4
Mycobacterium fortuitum colonization	1	22
Pseudomonas aeruginosa	1	2
Serratia marcescens	1	5
Total	19	236

Table 2. Acute Care Hospital Outbreaks by Disease/Condition LAC, 2001

Sub-acute Facilities:

During 2001, 35 outbreaks were reported in SNFs and 1 in a dialysis facility (Table 1). Two SNFs reported more than one outbreak. As in previous years, scabies outbreaks were the most frequently reported in sub-acute care settings (69%). The 4 outbreaks and 111 cases of gastroenteritis (unspecified or Norwalk-like virus) reported in 2001 was similar to 2000 (4 outbreaks, 102 cases) but less than the 9 outbreaks and 251 cases in 1999. In 2001, one outbreak due to crusted scabies was reported from a dialysis facility.

LAC, 2001		
	Number of	Number of
Disease/Condition	Outbreaks	Cases
Scabies (crusted, atypical, typical)	25	111
Gastroenteritis, unspecified	4	111
Chickenpox (Varicella)	2	6
Pediculosis	2	11
Conjunctivitis	1	13
Flu-like symptoms	1	12
Methicillin-resistant S. aureus	1	6
Total	36	270

Table 3. Sub-acute Care Setting* Outbreaks by Disease/Condition LAC, 2001

* Skilled-Nursing, Intermediate-Care/Psychiatric, Home Health, Dialysis Center

COMMENTS

Hospital infection control practitioners principally manage hospital outbreaks. ACDC becomes involved when hospitals report the outbreaks and ask for assistance with investigating some of the outbreaks, developing infection control policies, or facilitating specialized laboratory tests

(like pulsed-field gel electrophoresis or DNA "fingerprinting"). More extensive oversight is provided for outbreaks in facilities with minimal infection control resources and those diseases with higher morbidity or mortality potential. Community Health Services district staff have primary responsibility for disease investigations in sub-acute care settings. To assist healthcare facilities and district staff, ACDC produces and distributes guidelines in the management of scabies in healthcare facilities and the management of methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant *enterococcus* (VRE) in long-term care facilities. Developing strategies to prevent and control the emergence and spread of antibiotic-resistant bacteria is a priority in both sub-acute and acute care settings. In the future, ACDC would like to partner with health facilities to develop antibiotic prescribing guidelines and infection control practices.

ADDITIONAL INFORMATION

For more information on infection control and outbreaks in healthcare settings, go to the following website: www.cdc.gov/ncidod/hip/



PEDIATRIC ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS)

CRUDI	E DATA	
Number of Cases	4	Pedia
LA County California United States	0.01 ^b 0.04 ^b 0.10	30 25 20 20 15
Case Fatality LA County California United States	68.0% N/A N/A	



a Cases per 100,000 population.

 $^{\mbox{b}}$ Rates based on less than 20 observations are unreliable.

DESCRIPTION

Pediatric Acquired Immunodeficiency Syndrome^{*} (AIDS) is a severe disease syndrome in children less than 13 years of age. This syndrome represents the late clinical stage of infection with the human immunodeficiency virus (HIV). HIV attacks the body's immune system. When the immune system is weak it can no longer defend the body against diseases and tumors. Various infections called opportunistic infections develop. A woman who is HIV infected and does not receive treatment has about a 25% chance of passing HIV to her baby. This can happen in utero, during labor and delivery, and after delivery if the mother breast-feeds the baby. A woman who has HIV and is pregnant can help decrease the chance of transmission by taking ZDV (zidovudine, Retrovir®) during pregnancy and at the time of delivery; the baby should be given the same drug for six weeks after birth. When this is done the rate of transmission is decreased to 5-8%.

DISEASE ABSTRACT

• The number of children diagnosed with AIDS in Los Angles County declined from a peak of 27 in 1994, to a minimum of one in 1999. In 2001, four cases of pediatric AIDS were reported in LAC.

^{*} Centers for Disease Control and Prevention (1999). CDC Guidelines for National Human Immunodeficiency Virus Case Surveillance, including monitoring of human Immunodeficiency virus infection and acquired Immunodeficiency syndrome. MMWR 48 (RR – 13): 1-31.

Description	Case 1	Case 2	Case 3	Case 4
Place of birth	Mexico	Virginia	LAC	LAC
Gender	Male	Male	Female	Male
Race/ethnicity	Latino	Latino	Latino	Black
Date of AIDS diagnosis	03/2001	10/2001	07/2001	11/2001
AIDS defining illness	Cryptosporidiosis Isosporiasis	Cytomegalovirus retinitis	Pneumocystis carinii	Pneumocystis carinii
	M. tuberculosis		pneumonia pneumonia	
			Esophageal candidiasis	
Date mother was diagnosed with HIV	After child's birth	Before child's birth	After child's birth	After child's birth
Mode of delivery	Vaginal	Vaginal	Vaginal	Cesarean section
Mother received zidovudine (ZDV) during pregnancy, labor and delivery	No	Yes	No	No
Breastfed	Yes	No	Yes	Yes

Table 1: Description of Pediatric AIDS Cases Reported. LAC, 2001

COMMENTS

- The risk of perinatal HIV transmission can be maximally reduced by early prenatal care, screening of all pregnant women for HIV, and if found positive, appropriate treatment of mother and child, cesarean delivery and not breast-feeding.
- The standard HIV serological tests, including enzyme linked immunosorbent assay (ELISA) and Western blot immunoassay, are not useful in the diagnosis of HIV infection during infancy because of the confounding presence in infants' blood of transplacentally derived maternal antibody.
- In the U.S., the HIV DNA polymerese chain reaction (PCR) assay is the most widely used test for diagnosis of HIV infection during infancy.
- For the purposes of clinical decision making, an infant less than 18 months of age is considered HIV-infected if he/she is known to be HIV-seropositive, or was born to an HIV-infected mother, and has positive results on two separate direct tests for HIV (i.e., HIV culture, PCR, or p24 antigen detection).

ADDITIONAL RESOURCES

CDC and Prevention. Perinatal HIV Guideline Working Group. Public Health Services Task Force recommendations for use of antiretroviral drugs in pregnant HIV-1 transmission in the United States. MMWR Morb Mortal Wkly Rep. 1998;47 (RR-2):1-30. Update guideline available at <u>www.hivatis.org</u>

The Committee on Pediatric AIDS. Technical Report: Perinatal Human Immunodeficiency Virus testing and prevention of transmission. Pediatrics 2000; 106:e88 (1-12). Available at www.pediatrics.org

CDC and prevention. Guideline for national human Immunodeficiency virus case surveillance, including monitoring for human Immunodeficiency virus infection and acquired immunodeficiency syndrome. MMWR Morb Mortal Wkly Rep. 1999;48 (RR-13):1-27, 29-31. Available at www.cdc.gov/mmwr/preview/mmwrhtm/rr4813a1.htm

Centers for Disease Controls and Prevention. HIV/AIDS Surveillance Report, 2000; 12 (1). Available at www.cdc.gov/hiv/stats/hasrlink.htm

Centers for Disease Control and Prevention. Success in Implementing Public Health Guidelines to reduce Perinatal Transmission of HIV – Louisiana, Michigan, New Jersey, and South Carolina, 1993, 1995, and 1996. MMWR Morb Mortal Wkly Rep. 1998 Aug 28; 47(33). Available at www.cdc.gov/mmwr/preview/mmwrhtml/00054649.htm

HIV/AIDS Reporting System: www.cdc.gov/nchstp/hiv_aids/dhap.htm

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Disease Outbreak Summaries

Community-Acquired Disease Outbreaks	Heather Smith, MPH
Foodborne Outbreaks	Robin Seitzman, MPH
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Pediatric HIV/AIDS Reporting Project Report Pediatric Acquired Immunodeficiency Syndrome

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PUBLICATIONS, PRESENTATIONS AND ABSTRACTS

2001

PRESENTATIONS AND ABSTRACTS

Frederick T, Dominguez K, Mascola L. Trends in CD4 counts and AIDS-defining conditions (ADC's) in infants and children in Los Angeles County (LAC): A comparison of the pre and post HAART time periods. Presented at 8th Conference on Retroviruses and Opportunistic Infections. February 4-8, 2001; Chicago, Illinois.

Seward J, Watson B, Peterson C, Mascola L, et al. Decline in varicella incidence and hospitalizations in sentinel surveillance areas in the United States, 1995-2000. Presented at VZV Foundation Meeting. March 6, 2001; San Diego, California.

Maupin T, Goldman G, Peterson CL, Mascola L, et al. Knowledge, attitudes, and practices of healthcare providers regarding varicella vaccination in a sentinel surveillance area, 1996, 1997, and 1999. Presented at 2001 Pediatric Academic Societies Meeting. April 28-May 1, 2001. Baltimore, Maryland.

Galil K, Watson B, Peterson C, Mascola L, et al. Breakthrough-varicella cases since licensure in the Varicella Active Surveillance Project. Presented at 2001 Pediatric Academic Societies Meeting. April 28-May 1, 2001. Baltimore, Maryland.

Reporter R. Zweig R, Vogt J, Marks K, Mascola L. East Meets West: Campylobacteriosis associated with and ethnic specialty. Presented at 39th Annual Meeting of the Infectious Diseases Society of America. October 25-28, 2001; San Francisco, California.

Smith H., Reporter R, Rood M, Linscott A, Mascola L. Seroprevalence study for Antibody to ratborne pathogens and other agents among skid row residents–Los Angeles 2000. Presented at 39th Annual Meeting of the Infectious Diseases Society of America. October 25-28, 2001; San Francisco, California.

Zweig R, Frye D, Vogt J, Kabasele K, Mascola L. Rising rates and shifting serotypes of shigellosis among men who have sex with men–LAC, 1998-2000. Presented at 39th Annual Meeting of the Infectious Diseases Society of America. October 25-28, 2001; San Francisco, California.

Mascola L, Itano A, Peterson C. Invasive pneumococcal disease in Los Angeles County (LAC) children in the pre-conjugate vaccine era. Presented at 39th Annual Meeting of the Infectious Diseases Society of America. October 25-28, 2001; San Francisco, California.

Peterson C, Mascola L, Maupin T, Goldman G. Seward J. Varicella epidemiology: six years of active surveillance data following implementation of the varicella vaccination program. Presented at 39th Annual Meeting of the Infectious Diseases Society of America. October 25-28, 2001; San Francisco, California.

Bancroft A. E, Lee I, Yasuda L, Lehnkering E, Tormey M, Borenstein L, Harvey S, Mascola L. Outbreak of "Norwalk-like Virus" associated with foodhandlers: evidence of prolonged viral shedding using new DNA primers, Los Angeles, 2000. Presented at 39th Annual Meeting of the Infectious Diseases Society of America. October 25-28, 2001; San Francisco, California.

PUBLICATIONS

Buchholz U, Mascola L. Transmission, pathogenesis, and epidemilogy of *Listeria Monocytogenes*. Infect Dis Clin Practice. 2001;10:34-41.