



WEST NILE VIRUS

| CRUDE DATA | |
|-------------------------------|-------------|
| Number of Cases ^a | 153 |
| Annual Incidence ^b | |
| LA County ^a | 1.59 |
| California ^c | 0.88 |
| United States ^c | 0.60 |
| Age at Diagnosis | |
| Mean | 60.5 |
| Median | 62 |
| Range | 17-92 years |

^aIncludes asymptomatic infections

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

^cCalculated from: CDC. *Notice to Readers: Final 2016 Reports of Nationally Notifiable Infectious Diseases and Conditions Weekly* / January 6, 2018 / 65(52). Available at: https://www.cdc.gov/mmwr/volumes/65/wr/mm6552md.htm?s_cid=mm6552md_w

DESCRIPTION

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

WNV-infected birds can develop high levels of the virus in their bloodstream, and mosquitoes (especially *Culex* species) become infected by biting them. Those mosquitoes can then infect more birds as well as people, horses, and other mammals. However, humans, horses, and other mammals are “dead-end” hosts because they do not develop high enough levels of virus in their bloodstream to be able to pass the virus on to other biting mosquitoes.

About 20% of persons infected will develop WNV fever with symptoms that include fever, headache, rash,

muscle weakness, fatigue, nausea, vomiting, and occasionally lymph node swelling. Fewer than 1% will develop a more severe illness, manifesting as WNV neuro-invasive disease (NID), including meningitis, encephalitis, and acute flaccid paralysis. WNV-associated meningitis usually involves fever, headache, and stiff neck. WNV-associated encephalitis is commonly associated with fever, altered mental status, headache, and seizures. Encephalitis usually necessitates a high level of specialized medical care. Long-term neurological and cognitive sequelae are not uncommon. Studies have found that only 37% of hospitalized NID patients achieve full recovery by one year [1].

After being infected with WNV, most people sustain a viremia and may remain asymptomatic. Starting in 2003, blood products have been screened for WNV utilizing polymerase chain reaction (PCR) testing to prevent transmission of WNV from asymptomatic blood donors to recipients. Organ donors are also screened by nucleic acid tests (NAT) and serology to prevent transplant-associated transmission. Additional routes of transmission that can occur include vertical transmission, transmission through breast milk, and occupational exposure.

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

- Apply insect repellent to exposed skin,
- When possible, wear long-sleeved shirts and long pants outdoors, especially for long periods of time,
- Stay indoors at dawn, dusk, and in the early evening, which are peak biting times for *Culex* mosquitoes, and
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water.



This will reduce the number of places mosquitoes can lay their eggs and breed.

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

- DEET (N,N-diethyl-m-toluamide),
- Picaridin (KBR 3023), and
- Oil of lemon eucalyptus IR3535 (3-[N-Butyl-N-acetyl]-aminopropionic acid, ethyl ester)

2016 TRENDS AND HIGHLIGHTS

- There were 153 cases in 2016, a 49% decrease from the previous year. However, this was the fifth consecutive year in which LAC experienced above the overall average incidence (Figure 1). Previously, LAC demonstrated a cyclical pattern, peaking every four years.
- There were 35 cases (23%) of WNV fever and 108 cases (71%) of NID (Figure 2). There were 10 asymptomatic donors (7%) reported from local blood banks. Of 143 reported symptomatic WNV infections, six were fatal (4.2%). The six fatalities were aged 50 to 88 years old (median 76.5 years), and all but one had contributing medical history including hypertension and diabetes. The remaining case, the youngest fatality, denied any prior medical conditions.
- The age range of all infections was 17-92 years old with the largest proportion ≥ 65 years old (n=71, 46.4%). Incidence increased with age (Figure 3).

- The top three counts of WNV by SPA were SPAs 2 (San Fernando Valley, n=86, 56.2%), 3 (San Gabriel Valley, n=22, 14.4%), and 4 (Central LA, n=11, 7.2%) (Figure 5). In 2016, residents within the city of Los Angeles reported the most WNV infections (n=18, 12%) followed by Van Nuys (n=10, 7%) and North Hollywood (n=6, 4%).
- In 2016, WNV infections occurred from July to November with the last case experiencing symptom onset on the 29th of November. Peak onset in 2016 occurred in August (n=75, 49%). The five-year average indicates September as the month with the most frequent onset peak (Figure 6).
- Though WNV is primarily transmitted by infected mosquitoes, a case of transfusion-associated WNV infection was documented in 2016. The patient received blood products collected throughout the month of July from 30 donors, nearly all from the southern California region. Donor blood is screened in pools including multiple donors (MP-NAT) until the seasonal risk of WNV increases and individual screening is triggered (ID-NAT). The implicated donor's unit tested negative during the initial screen but tested positive 81 days post-donation. The blood bank will re-evaluate criteria for triggering individual testing [2].

REFERENCES

1. Klee, A., Maldin, B., Edwin, B., et al. Long-Term Prognosis for Clinical West Nile Virus Infection. *Emerg Infect Dis*, 10 (8): 1405-1411.
2. Groves, J.A., Shafi, H., Nomura, J.H., et al. (2017). A probable case of West Nile virus transfusion transmission. *Transfusion*.



**Reported WNV Infections and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
LAC, 2012-2016**

| | 2012 (N=174) | | | 2013 (N=165) | | | 2014 (N=218) | | | 2015 (N=300) | | | 2016 (N=153) | | |
|-----------------------|--------------|------|------------------|--------------|------|------------------|--------------|------|------------------|--------------|------|------------------|--------------|------|------------------|
| | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 |
| Age Group | | | | | | | | | | | | | | | |
| <1 | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - |
| 1-4 | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - |
| 5-14 | 2 | 1.1 | 0.2 | 6 | 3.6 | 0.5 | 0 | - | - | 3 | 1.0 | 0.2 | 0 | - | - |
| 15-34 | 24 | 13.8 | 0.9 | 19 | 11.5 | 0.7 | 23 | 10.6 | 0.8 | 34 | 11.3 | 1.2 | 13 | 8.5 | 0.5 |
| 35-44 | 17 | 9.8 | 1.3 | 15 | 9.1 | 1.1 | 15 | 6.9 | 1.1 | 28 | 9.3 | 2.1 | 14 | 9.2 | 1.1 |
| 45-54 | 33 | 19.0 | 2.6 | 34 | 20.6 | 2.6 | 44 | 20.2 | 3.4 | 41 | 13.7 | 3.1 | 26 | 17.0 | 2.0 |
| 55-64 | 34 | 19.5 | 3.3 | 46 | 27.9 | 4.5 | 55 | 25.2 | 5.2 | 53 | 17.7 | 4.8 | 29 | 19.0 | 2.6 |
| 65+ | 64 | 36.8 | 5.8 | 45 | 27.3 | 4.1 | 81 | 37.2 | 7.2 | 141 | 47.0 | 11.8 | 71 | 46.4 | 5.8 |
| Unknown | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - |
| Race/Ethnicity | | | | | | | | | | | | | | | |
| Asian | 9 | 5.2 | 0.7 | 6 | 3.6 | 0.4 | 11 | 5.0 | 0.8 | 7 | 2.3 | 0.5 | 8 | 5.2 | 0.6 |
| Black | 3 | 1.7 | 0.4 | 3 | 1.8 | 0.4 | 3 | 1.4 | 0.4 | 5 | 1.7 | 0.6 | 2 | 1.3 | 0.3 |
| Hispanic | 59 | 33.9 | 1.3 | 50 | 30.3 | 1.1 | 73 | 33.5 | 1.6 | 110 | 36.7 | 2.3 | 40 | 26.1 | 0.8 |
| White | 91 | 52.3 | 3.4 | 80 | 48.5 | 3.0 | 97 | 44.5 | 3.6 | 142 | 47.3 | 5.3 | 77 | 50.3 | 2.9 |
| Other | 2 | 1.1 | - | 2 | 1.2 | - | 0 | - | - | 1 | 0.3 | - | 3 | 2.0 | - |
| Unknown | 10 | 5.7 | - | 24 | 14.5 | - | 34 | 15.6 | - | 35 | 11.7 | - | 23 | 15.0 | - |
| SPA | | | | | | | | | | | | | | | |
| 1 | 10 | 5.7 | 2.6 | 15 | 9.1 | 3.8 | 2 | 0.9 | 0.5 | 4 | 1.3 | 1.0 | 3 | 2.0 | 0.8 |
| 2 | 73 | 42.0 | 3.4 | 62 | 37.6 | 2.9 | 60 | 27.5 | 2.7 | 92 | 30.7 | 4.1 | 86 | 56.2 | 3.8 |
| 3 | 47 | 27.0 | 2.9 | 23 | 13.9 | 1.4 | 34 | 15.6 | 2.1 | 46 | 15.3 | 2.8 | 22 | 14.4 | 1.3 |
| 4 | 18 | 10.3 | 1.6 | 6 | 3.6 | 0.5 | 28 | 12.8 | 2.4 | 41 | 13.7 | 3.5 | 11 | 7.2 | 0.9 |
| 5 | 8 | 4.6 | 1.3 | 2 | 1.2 | 0.3 | 24 | 11.0 | 3.7 | 30 | 10.0 | 4.5 | 5 | 3.3 | 0.8 |
| 6 | 2 | 1.1 | 0.2 | 4 | 2.4 | 0.4 | 13 | 6.0 | 1.3 | 15 | 5.0 | 1.4 | 5 | 3.3 | 0.5 |
| 7 | 13 | 7.5 | 1.0 | 24 | 14.5 | 1.8 | 45 | 20.6 | 3.4 | 59 | 19.7 | 4.5 | 9 | 6.0 | 0.7 |
| 8 | 3 | 1.7 | 0.3 | 29 | 17.6 | 2.7 | 11 | 5.0 | 1.0 | 13 | 4.3 | 1.2 | 9 | 6.0 | 0.8 |
| Unknown | 0 | - | - | 0 | - | - | 1 | 0.5 | - | 0 | - | - | 3 | 2.0 | - |

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



Figure 1. Incidence Rates* of West Nile Virus LAC, 2004-2016

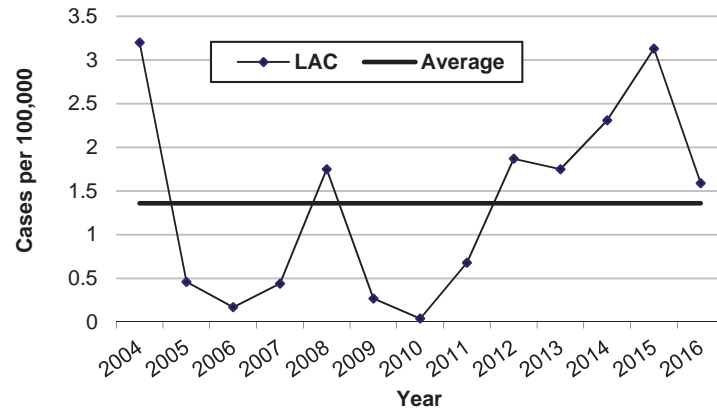


Figure 2. Percentage of West Nile Virus Infections by Presentation LAC, 2016 (N=153)

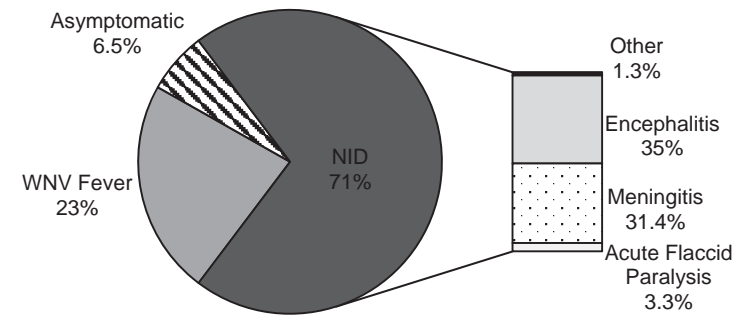


Figure 3. Incidence Rates* of West Nile Virus Infection by Age Group LAC, 2016 (N=153)

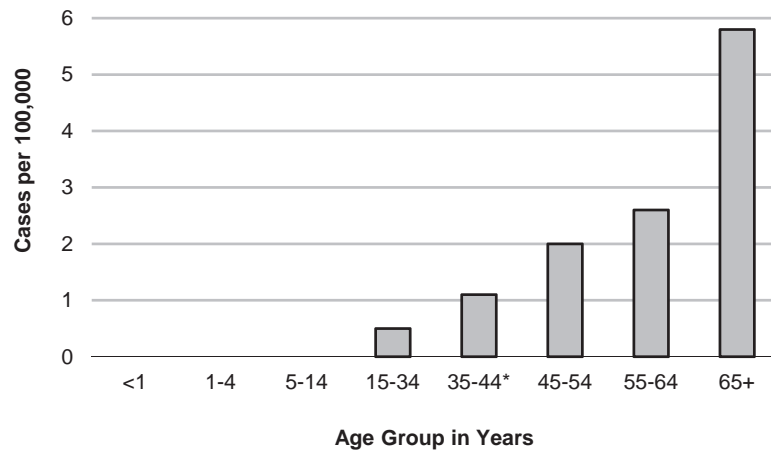
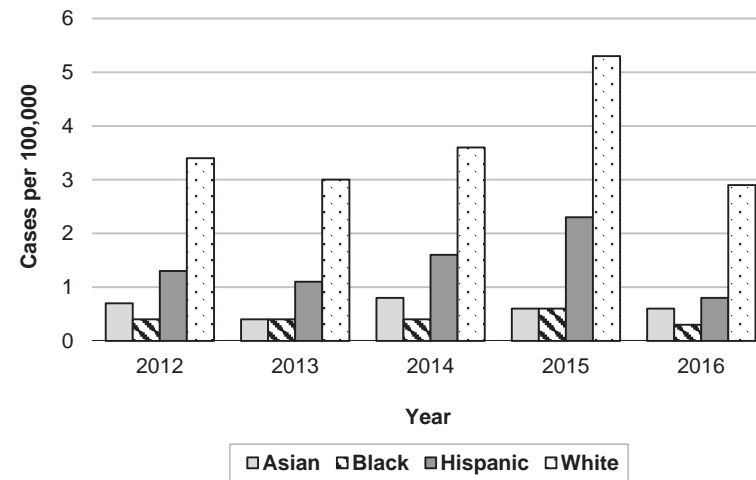
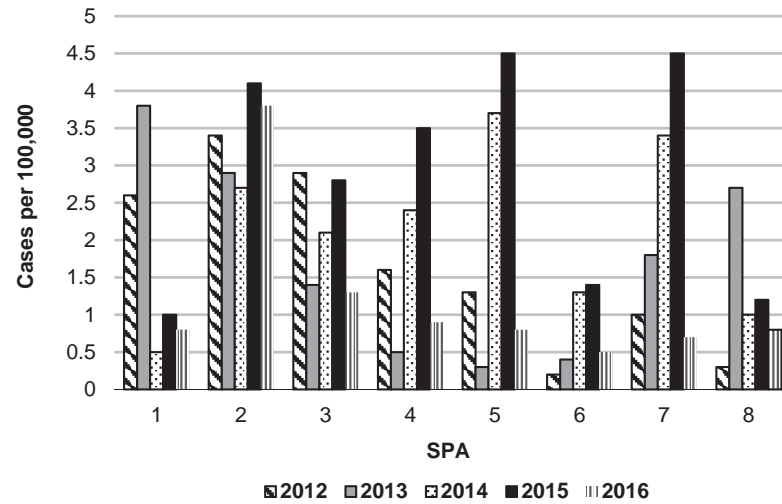


Figure 4. West Nile Virus Incidence* by Race/Ethnicity LAC, 2012-2016

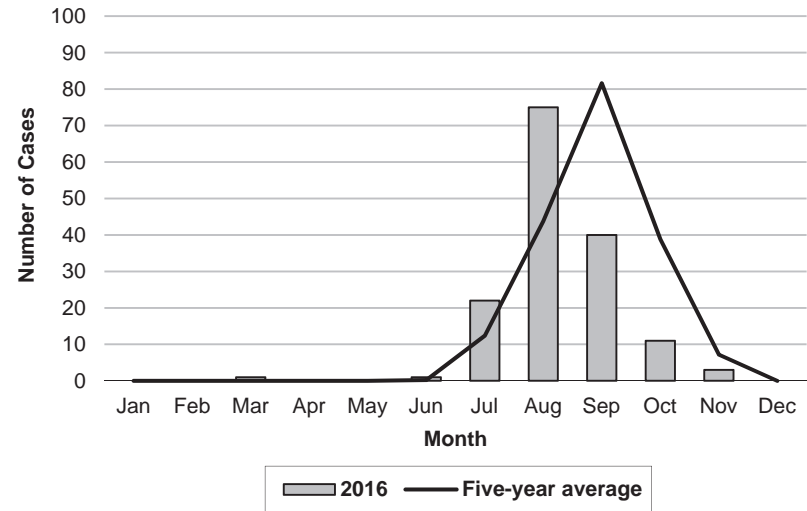




**Figure 5. Incidence Rates* of West Nile Virus by SPA
LAC, 2012-2016**

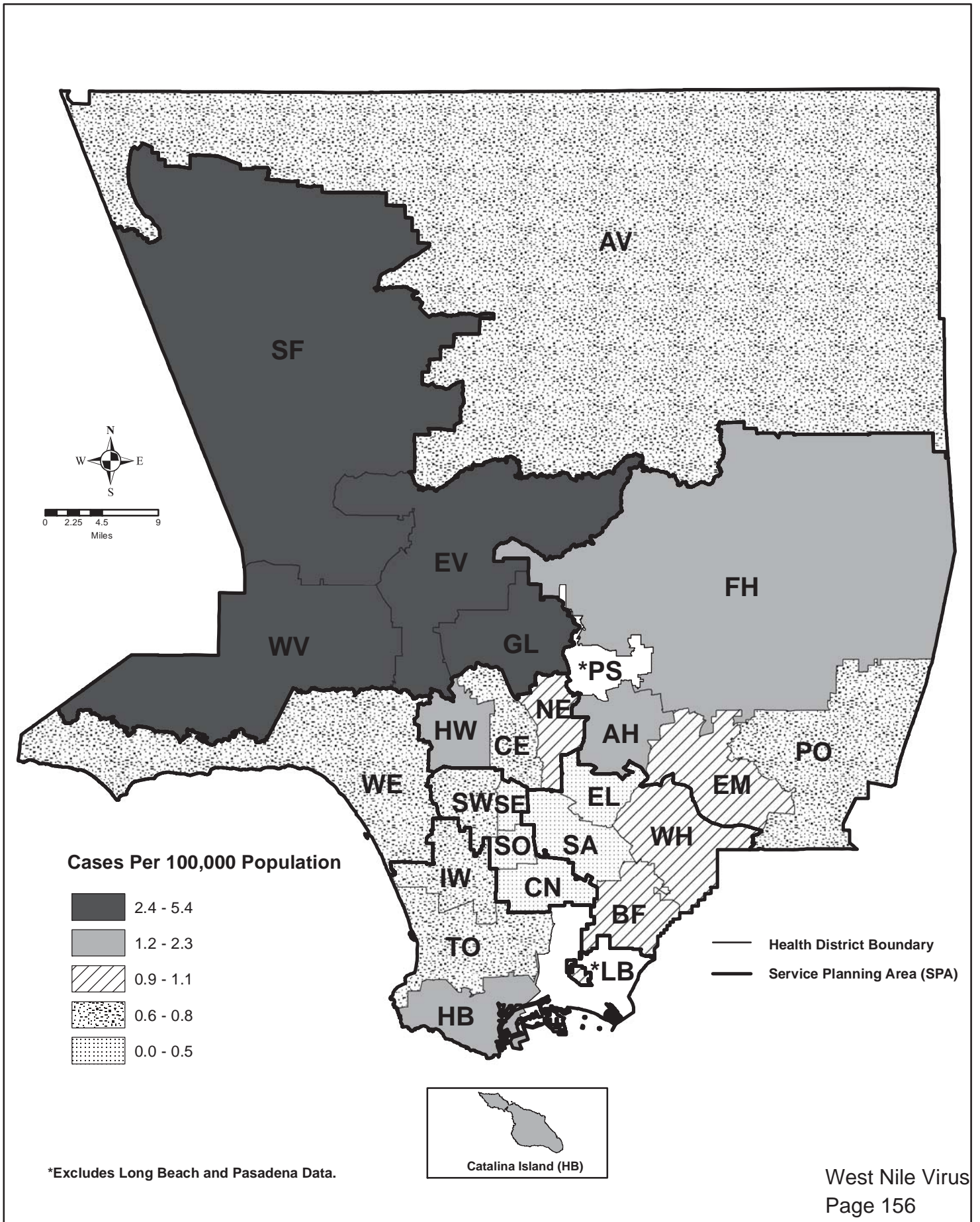


**Figure 6. Reported West Nile Virus Infections
by Month of Onset
LAC, 2016 (N=153)**



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

Map 14. West Nile Virus Rates by Health District, Los Angeles County, 2016*





WEST NILE VIRUS

| CRUDE DATA | |
|-------------------------------|-------------|
| Number of Cases ^a | 300 |
| Annual Incidence ^b | |
| LA County ^a | 3.13 |
| California ^c | 2.00 |
| United States ^c | 0.68 |
| Age at Diagnosis | |
| Mean | 60 |
| Median | 63 |
| Range | 12–98 years |

^aIncludes asymptomatic infections

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

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

Available at:

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

DESCRIPTION

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

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

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

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

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

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

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

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



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

2015 TRENDS AND HIGHLIGHTS

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

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

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



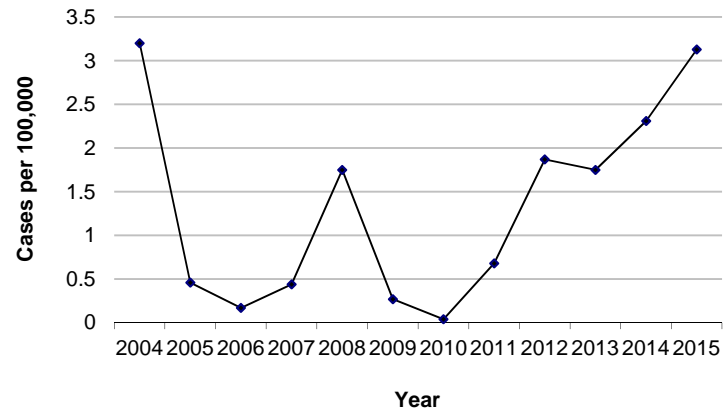
**Reported WNV Infections and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
LAC, 2011-2015**

| | 2011 (N=63) | | | 2012 (N=174) | | | 2013 (N=165) | | | 2014 (N=218) | | | 2015 (N=300) | | |
|-----------------------|-------------|------|------------------|--------------|------|------------------|--------------|------|------------------|--------------|------|------------------|--------------|------|------------------|
| | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 |
| Age Group | | | | | | | | | | | | | | | |
| <1 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 |
| 1-4 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 |
| 5-14 | 1 | 1.6 | 0.1 | 2 | 1.1 | 0.2 | 6 | 3.6 | 0.5 | 0 | 0.0 | 0.0 | 3 | 1.0 | 0.2 |
| 15-34 | 5 | 7.9 | 0.2 | 24 | 13.8 | 0.9 | 19 | 11.5 | 0.7 | 23 | 10.6 | 0.8 | 34 | 11.3 | 1.2 |
| 35-44 | 3 | 4.8 | 0.2 | 17 | 9.8 | 1.3 | 15 | 9.1 | 1.1 | 15 | 6.9 | 1.1 | 28 | 9.3 | 2.1 |
| 45-54 | 16 | 25.4 | 1.2 | 33 | 19.0 | 2.6 | 34 | 20.6 | 2.6 | 44 | 20.2 | 3.4 | 41 | 13.7 | 3.1 |
| 55-64 | 17 | 27.0 | 1.8 | 34 | 19.5 | 3.3 | 46 | 27.9 | 4.5 | 55 | 25.2 | 5.2 | 53 | 17.7 | 4.8 |
| 65+ | 21 | 33.3 | 2.0 | 64 | 36.8 | 5.8 | 45 | 27.3 | 4.1 | 81 | 37.2 | 7.2 | 141 | 47.0 | 11.8 |
| Unknown | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - |
| Race/Ethnicity | | | | | | | | | | | | | | | |
| Asian | 1 | 1.6 | 0.1 | 9 | 5.2 | 0.7 | 6 | 3.6 | 0.4 | 11 | 5.0 | 0.8 | 7 | 2.3 | 0.5 |
| Black | 1 | 1.6 | 0.1 | 3 | 1.7 | 0.4 | 3 | 1.8 | 0.4 | 3 | 1.4 | 0.4 | 5 | 1.7 | 0.6 |
| Hispanic | 26 | 41.3 | 0.5 | 59 | 33.9 | 1.3 | 50 | 30.3 | 1.1 | 73 | 33.5 | 1.6 | 110 | 36.7 | 2.3 |
| White | 30 | 47.6 | 1.0 | 91 | 52.3 | 3.4 | 80 | 48.5 | 3.0 | 97 | 44.5 | 3.6 | 142 | 47.3 | 5.3 |
| Other | 2 | 3.2 | - | 2 | 1.1 | - | 2 | 1.2 | - | 0 | 0.0 | 0.0 | 1 | 0.3 | - |
| Unknown | 3 | 4.8 | - | 10 | 5.7 | - | 24 | 14.5 | - | 34 | 15.6 | - | 35 | 11.7 | - |
| SPA | | | | | | | | | | | | | | | |
| 1 | 1 | 1.6 | 0.3 | 10 | 5.7 | 2.6 | 15 | 9.1 | 3.8 | 2 | 0.9 | 0.5 | 4 | 1.3 | 1.0 |
| 2 | 39 | 61.9 | 1.8 | 73 | 42.0 | 3.4 | 62 | 37.6 | 2.9 | 60 | 27.5 | 2.7 | 92 | 30.7 | 4.1 |
| 3 | 16 | 25.4 | 0.9 | 47 | 27.0 | 2.9 | 23 | 13.9 | 1.4 | 34 | 15.6 | 2.1 | 46 | 15.3 | 2.8 |
| 4 | 1 | 1.6 | 0.1 | 18 | 10.3 | 1.6 | 6 | 3.6 | 0.5 | 28 | 12.8 | 2.4 | 41 | 13.7 | 3.5 |
| 5 | 1 | 1.6 | 0.2 | 8 | 4.6 | 1.3 | 2 | 1.2 | 0.3 | 24 | 11.0 | 3.7 | 30 | 10.0 | 4.5 |
| 6 | 1 | 1.6 | 0.1 | 2 | 1.1 | 0.2 | 4 | 2.4 | 0.4 | 13 | 6.0 | 1.3 | 15 | 5.0 | 1.4 |
| 7 | 4 | 6.3 | 0.3 | 13 | 7.5 | 1.0 | 24 | 14.5 | 1.8 | 45 | 20.6 | 3.4 | 59 | 19.7 | 4.5 |
| 8 | 0 | 0.0 | 0.0 | 3 | 1.7 | 0.3 | 29 | 17.6 | 2.7 | 11 | 5.0 | 1.0 | 13 | 4.3 | 1.2 |
| Unknown | 0 | - | - | 0 | - | - | 0 | - | - | 1 | 0.5 | - | 0 | - | - |

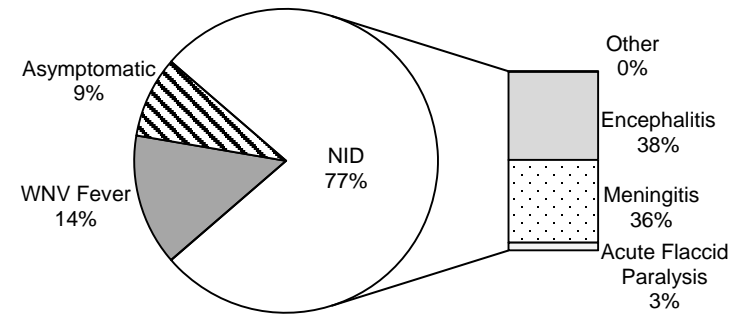
*Rates calculated based on less than 19 cases or events are considered unreliable.



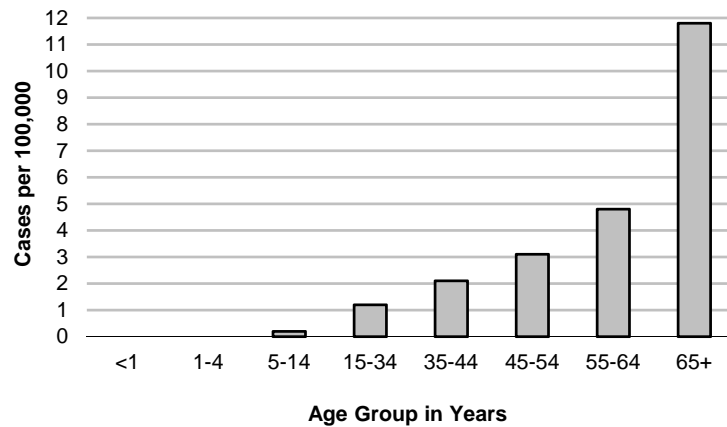
**Figure 1. Incidence Rates* of WNV
LAC, 2004-2015**



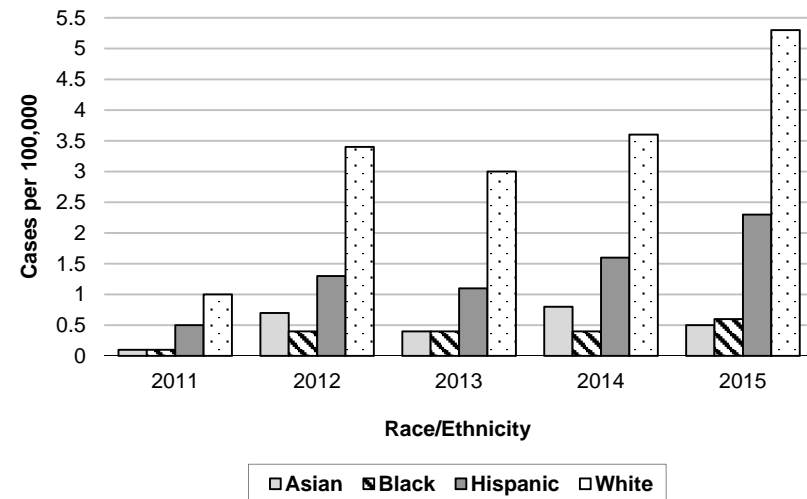
**Figure 2. Percentage of WNV Infections
by Presentation
LAC, 2015 (N=300)**



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



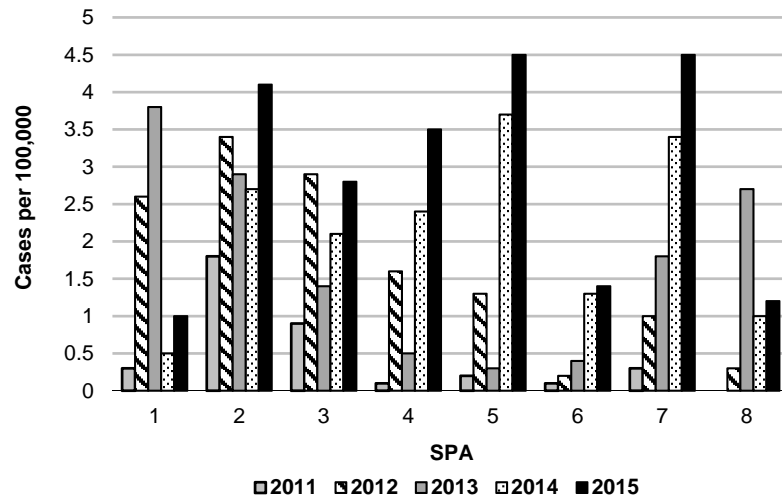
**Figure 4. WNV Incidence* by Race/Ethnicity
LAC, 2011-2015**



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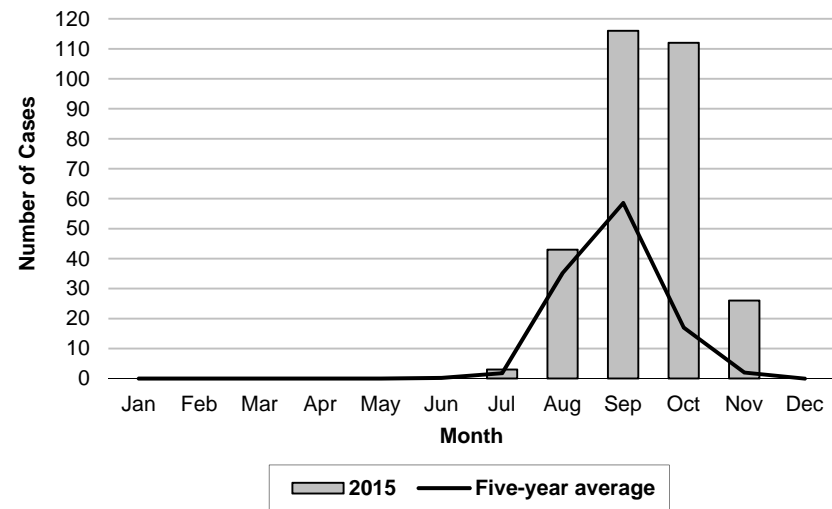


**Figure 5. Incidence Rates* of WNV by SPA
LAC, 2011-2015**

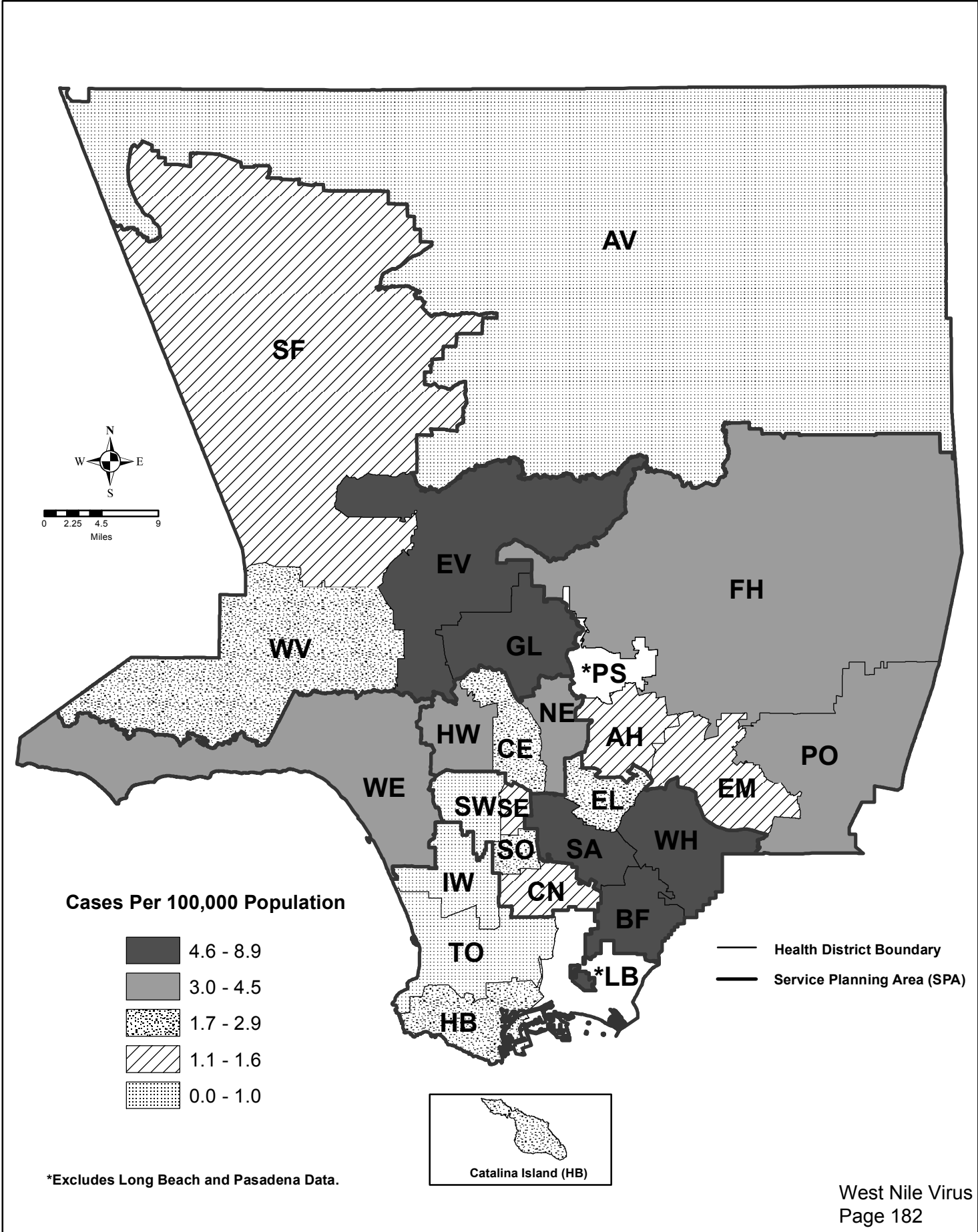


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**Figure 6. Reported WNV Infections by Month of Onset
LAC, 2015 (N=300)**



Map 16. West Nile Virus Rates by Health District, Los Angeles County, 2015*





WEST NILE VIRUS

| CRUDE DATA | |
|-------------------------------|-------------|
| Number of Cases ^a | 218 |
| Annual Incidence ^b | |
| LA County ^a | 2.31 |
| California ^c | 2.11 |
| United States ^c | 0.69 |
| Age at Diagnosis | |
| Mean | 58.7 |
| Median | 59 |
| Range | 17–94 years |

^aIncludes asymptomatic infections.

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

^cCalculated from Final 2014 Reports of Nationally Notifiable Infectious Diseases. MMWR 64(36):1019–1033.

DESCRIPTION

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

Normally transmitted by mosquitoes (usually *Culex* or *Anopheles* species) between bird reservoir hosts, humans are incidentally infected with the virus when bitten by an infected mosquito. About 20% of persons infected will develop WNV fever with symptoms that include fever, headache, rash, muscle weakness, fatigue, nausea and vomiting, and occasionally lymph node swelling. Fewer than 1% will develop more severe illness, manifesting as WNV neuro-invasive disease (NID), including meningitis, encephalitis, and acute flaccid paralysis. WNV-associated meningitis usually involves fever, headache, and stiff neck, and has a good prognosis. WNV-associated encephalitis is commonly associated with fever, altered mental status, headache, and seizures, and usually requires a high level of specialized medical care. Long-term neurological and cognitive sequelae are not uncommon.

After being infected with WNV, most people sustain a viremia and may remain asymptomatic or eventually develop symptoms. In 2002, asymptomatic blood

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

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

- Apply insect repellent to exposed skin. A higher percentage of DEET in a repellent will provide longer protection. DEET concentrations higher than 50% do not increase the length of protection.
- When possible, wear long-sleeved shirts and long pants when outdoors for long periods of time.
- Stay indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times.
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water. This will reduce the number of places mosquitoes can lay their eggs and breed.

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



- DEET (N,N-diethyl-m-toluamide)
- Picaridin (KBR 3023)
- Oil of lemon eucalyptus IR3535 (3-[N-Butyl-N-acetyl]-aminopropionic acid, ethyl ester).

2014 TRENDS AND HIGHLIGHTS

- The incidence of WNV infections reported in 2014 (2.31 per 100,000 population) is the second highest documented in LAC since WNV appeared in 2003 (Figure 1). A high number of cases each year from 2012 to 2014 suggests that the 4-year periodicity previously seen may no longer be characteristic.
- Of 199 reported symptomatic WNV infections, there were 43 cases of WNV fever and 156 NID cases (74 with meningitis, 67 encephalitis, and 15 acute flaccid paralysis) (Figure 2). Seven WNV-associated deaths were reported among symptomatic cases (4%). Nineteen asymptomatic donors (9%) were reported from local blood banks (Figure 2).
- The mean age of all reported infections was 58.7 years with the largest proportion in the 65 years old and over age group (28%). Incidence increased as age increased with no cases reported among children <15 years old (Figure 3). This likely reflects the increased occurrence of NID and more severe infection with increasing age.
- The highest incidence of reported WNV infections occurred among whites and persons of Hispanic/Latino race/ethnicity (3.6 and 1.6 per 100,000, respectively); disease in these populations consistently has been higher than for blacks and Asians.
- The male to female ratio was 1:8:1.
- WNV infections were distributed widely across all SPAs this year. The highest number of people infected with WNV continued to be residents of the San Fernando Valley area (28%), though geographic differences were less than in previous years (Figure 5). Record counts of human infections were documented in SPAs 4, 5, 6, and 7. In fact, the highest incidence rate occurred in the SPA 5 (western LAC area) with 3.7 cases per 100,000 (n=24).
- This year, human WNV infections occurred from July to November, with the last case experiencing illness onset on November 30, 2014 (Figure 6). This is the latest onset ever documented for LAC and continues a trend in which WNV transmission occurs through November. The human WNV season in LAC previously ran from June through the end of October. Peak onset in 2014 occurred in September, similar to the five-year average.
- Statewide in 2014, 892 human WNV infections were reported and nationally, 2,521 cases were reported.



**Reported West Nile Virus Infections and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
Los Angeles County, 2010-2014**

| | 2010 (N=4) | | | 2011 (N=63) | | | 2012 (N=174) | | | 2013 (N=165) | | | 2014 (N=218) | | |
|-----------------------|------------|------|------------------|-------------|------|------------------|--------------|------|------------------|--------------|------|------------------|--------------|------|------------------|
| | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 |
| Age Group | | | | | | | | | | | | | | | |
| <1 | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - |
| 1-4 | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - |
| 5-14 | 0 | - | - | 1 | 1.6 | 0.1 | 2 | 1.1 | 0.2 | 6 | 3.6 | 0.5 | 0 | - | - |
| 15-34 | 1 | 25.0 | 0.0 | 5 | 7.9 | 0.2 | 24 | 13.8 | 0.9 | 19 | 11.5 | 0.7 | 23 | 10.6 | 0.8 |
| 35-44 | 0 | - | - | 3 | 4.8 | 0.2 | 17 | 9.8 | 1.3 | 15 | 9.1 | 1.1 | 15 | 6.9 | 1.1 |
| 45-54 | 1 | 25.0 | 0.1 | 16 | 25.4 | 1.2 | 33 | 19.0 | 2.6 | 34 | 20.6 | 2.6 | 44 | 20.2 | 3.4 |
| 55-64 | 0 | - | - | 17 | 27.0 | 1.8 | 34 | 19.5 | 3.3 | 46 | 27.9 | 4.5 | 55 | 25.2 | 5.2 |
| 65+ | 2 | 50.0 | 0.2 | 21 | 33.3 | 2.0 | 64 | 36.8 | 5.8 | 45 | 27.3 | 4.0 | 81 | 37.2 | 7.2 |
| Unknown | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - |
| Race/Ethnicity | | | | | | | | | | | | | | | |
| Asian | 0 | - | - | 1 | 1.6 | 0.1 | 9 | 5.2 | 0.7 | 6 | 3.6 | 0.4 | 11 | 5.0 | 0.8 |
| Black | 0 | - | - | 1 | 1.6 | 0.1 | 3 | 1.7 | 0.4 | 3 | 1.8 | 0.4 | 3 | 1.4 | 0.4 |
| Hispanic | 1 | 25.0 | 0.01 | 26 | 41.3 | 0.5 | 59 | 33.9 | 1.3 | 50 | 30.3 | 1.1 | 73 | 33.5 | 1.6 |
| White | 3 | 75.0 | 0.1 | 30 | 47.6 | 1.0 | 91 | 52.3 | 3.4 | 80 | 48.5 | 3.0 | 97 | 44.5 | 3.6 |
| Other | 0 | - | - | 2 | 3.2 | - | 2 | 1.1 | - | 2 | 1.2 | - | 0 | 0.0 | - |
| Unknown | 0 | - | - | 3 | 4.8 | - | 10 | 5.7 | - | 24 | 14.5 | - | 34 | 15.6 | - |
| SPA | | | | | | | | | | | | | | | |
| 1 | 0 | - | - | 1 | 1.6 | 0.3 | 10 | 5.7 | 2.6 | 15 | 9.1 | 3.8 | 2 | 0.9 | 0.5 |
| 2 | 0 | - | - | 39 | 61.9 | 1.8 | 73 | 42.0 | 3.4 | 62 | 37.6 | 2.9 | 60 | 27.5 | 2.7 |
| 3 | 2 | 50.0 | 0.1 | 16 | 25.4 | 0.9 | 47 | 27.0 | 2.9 | 23 | 13.9 | 1.4 | 34 | 15.6 | 2.1 |
| 4 | 0 | - | - | 1 | 1.6 | 0.1 | 18 | 10.3 | 1.6 | 6 | 3.6 | 0.5 | 28 | 12.8 | 2.4 |
| 5 | 0 | - | - | 1 | 1.6 | 0.2 | 8 | 4.6 | 1.3 | 2 | 1.2 | 0.3 | 24 | 11.0 | 3.7 |
| 6 | 0 | - | - | 1 | 1.6 | 0.1 | 2 | 1.1 | 0.2 | 4 | 2.4 | 0.4 | 13 | 6.0 | 1.3 |
| 7 | 2 | 50.0 | 0.1 | 4 | 6.3 | 0.3 | 13 | 7.5 | 1.0 | 24 | 14.5 | 1.8 | 45 | 20.6 | 3.4 |
| 8 | 0 | - | - | 0 | - | - | 3 | 1.7 | 0.3 | 29 | 17.6 | 2.7 | 11 | 5.0 | 1.0 |
| Unknown | 0 | - | - | 0 | - | - | 0 | - | - | 0 | - | - | 1 | 0.5 | - |

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



Figure 1. Incidence Rates* of West Nile Virus LAC, 2004-2014

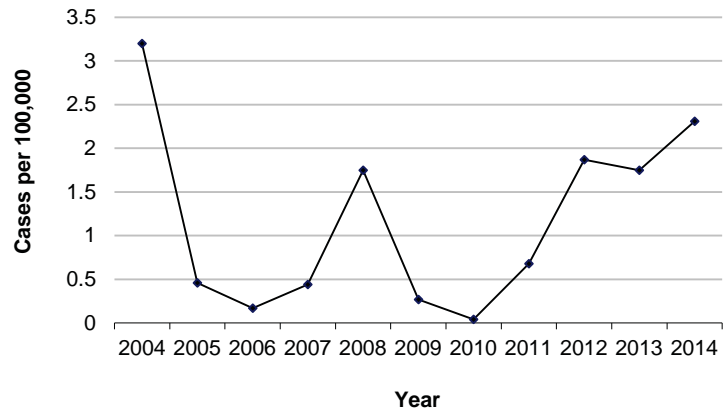


Figure 2. Percentage of West Nile Virus Infections by Presentation LAC, 2014 (N=218)

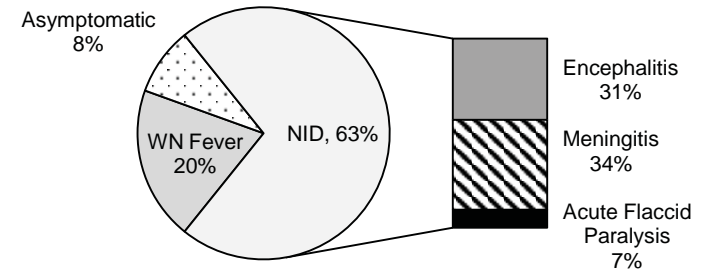


Figure 3. Incidence Rates* of West Nile Virus Infection by Age Group LAC, 2014 (N=218)

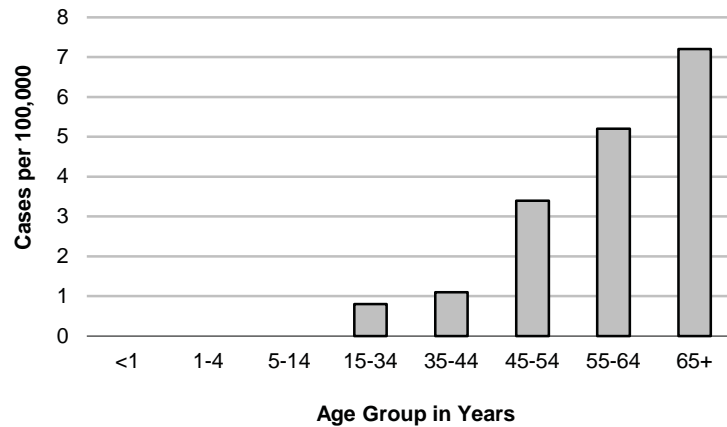
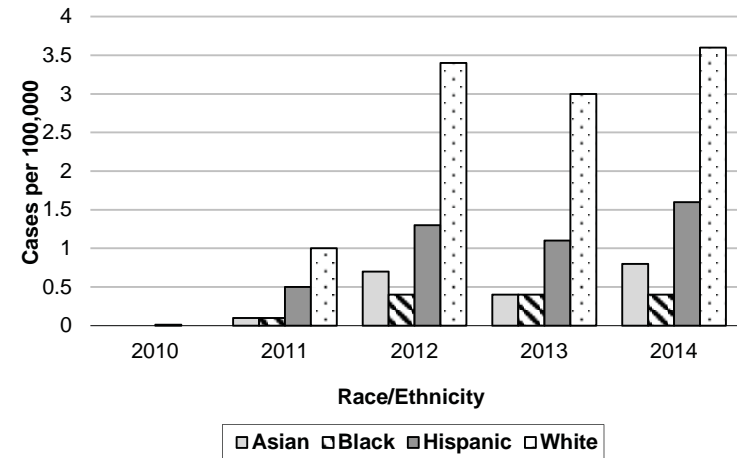


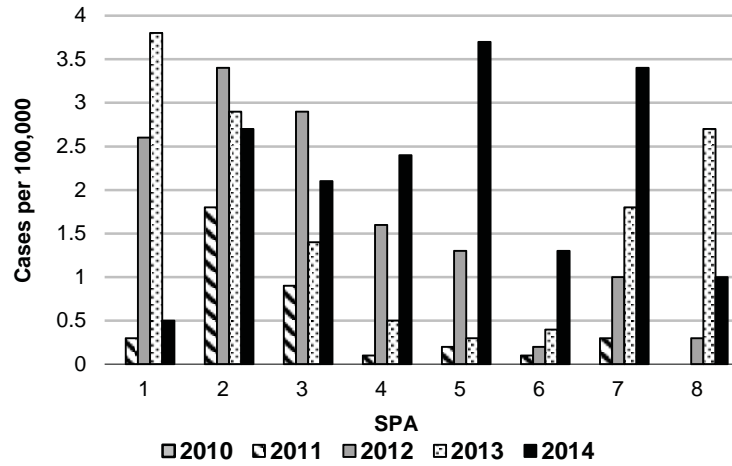
Figure 4. West Nile Virus Incidence* by Race/Ethnicity LAC, 2010-2014



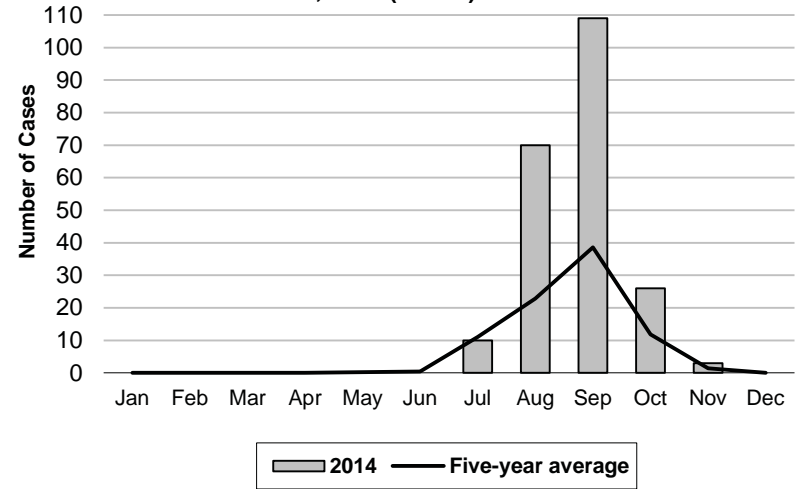
*Rates calculated based on less than 19 cases or events are considered unreliable.



**Figure 5. Incidence Rates* of West Nile Virus by SPA
LAC, 2010-2014**

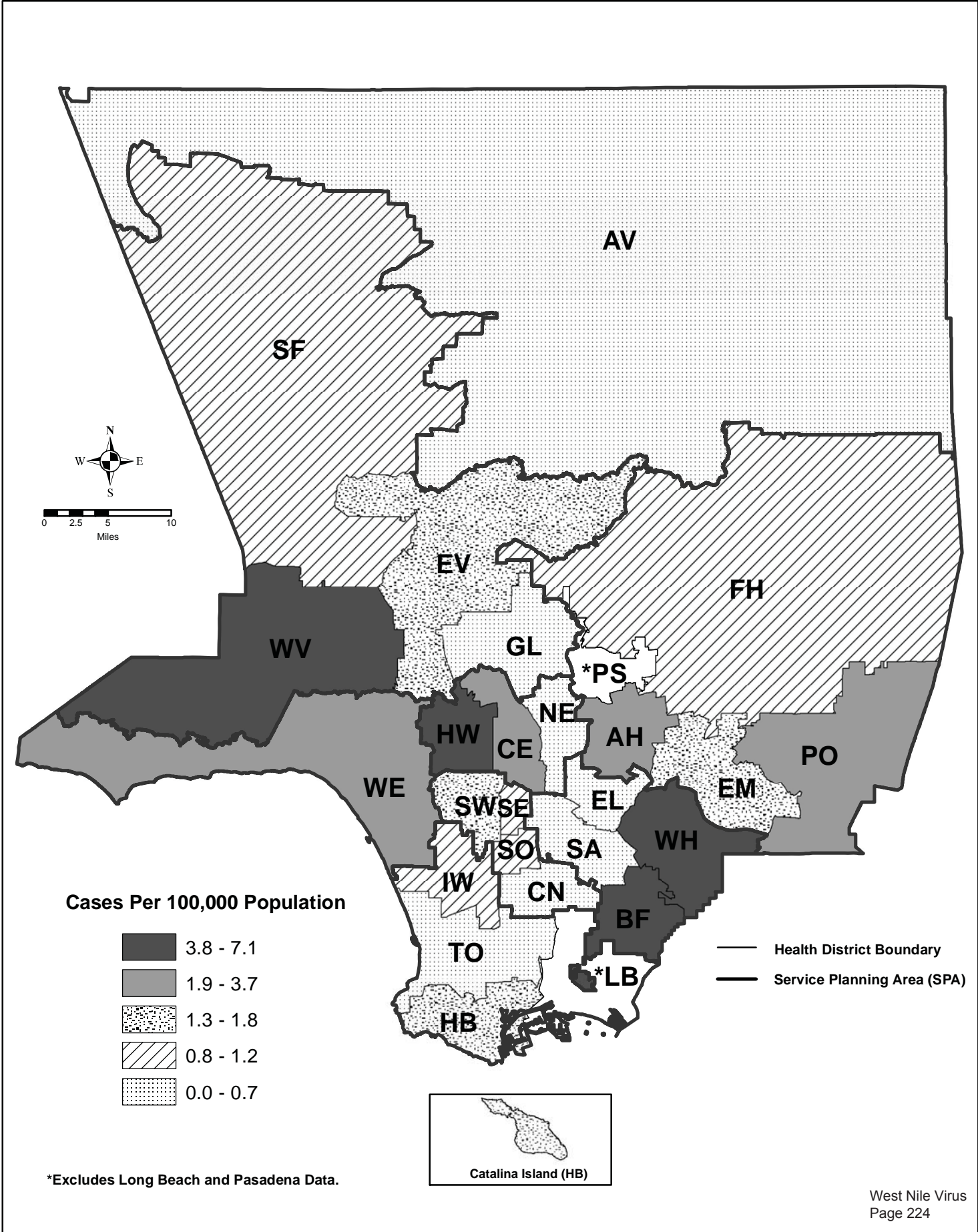


**Figure 6. Reported West Nile Virus Infections
by Month of Onset
LAC, 2014 (N=218)**



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

Map 16. West Nile Virus Rates by Health District, Los Angeles County, 2014*





WEST NILE VIRUS

| CRUDE DATA | |
|-------------------------------|------|
| Number of Cases ^a | 165 |
| Annual Incidence ^b | |
| LA County ^a | 1.75 |
| California ^c | 1.00 |
| United States ^c | 0.79 |
| Age at Diagnosis | |
| Mean | 54.2 |
| Median | 56.5 |
| Range | 7-92 |

^aIncludes asymptomatic infections.

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

^cCalculated from Final 2013 Reports of Nationally Notifiable Infectious Diseases. MMWR 63(32):702-716.

DESCRIPTION

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

Normally transmitted by mosquitoes (usually *Culex* or *Anopheles* species) between bird reservoir hosts, humans are incidentally infected with the virus when bitten by an infected mosquito. About 20% of persons infected will develop WNV fever with symptoms that include fever, headache, rash, muscle weakness, fatigue, nausea and vomiting, and occasionally lymph node swelling. Fewer than 1% will develop more severe illness, manifesting as WNV neuro-invasive disease (NID), including meningitis, encephalitis, and acute flaccid paralysis. WNV-associated meningitis usually involves fever, headache, and stiff neck, and has a good prognosis. WNV-associated encephalitis is commonly associated with fever, altered mental status, headache, and seizures, and usually necessitates a high level of specialized medical care. Long-term neurological and cognitive sequelae are not uncommon.

After being infected with WNV, most people sustain a viremia and may remain asymptomatic or eventually develop symptoms. In 2002, asymptomatic blood

donors were documented to transmit WNV to blood product recipients. Beginning 2003, blood products have been screened for WNV utilizing polymerase chain reaction (PCR) testing. To date, there have been no blood transfusion-associated secondary WNV infections from asymptomatic WNV-infected blood donors from Los Angeles (LAC) residents. However, four cases of WNV-associated infection including three cases of NID were documented from a LAC organ donor in 2011, not known to be infected with WNV infection at the time of organ donation. Additional routes of transmission that can occur include vertical transmission transplacentally, through breast milk, and occupational exposure.

Vector management programs are the most effective approach to prevention and control of WNV and other arboviral diseases. These programs include surveillance for WNV activity in mosquito vectors, birds, horses, other animals, and humans; and implementation of appropriate mosquito control measures to reduce mosquito populations when necessary. When virus activity is detected in an area, residents are advised to increase measures to reduce contact with mosquitoes. Currently, there is no human vaccine available against WNV but several vaccines are under development. Important preventive measures against WNV include the following:

- Apply insect repellent to exposed skin. A higher percentage of DEET in a repellent will provide longer protection. DEET concentrations higher than 50% do not increase the length of protection.
- When possible, wear long-sleeved shirts and long pants when outdoors for long periods of time.
- Stay indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times.
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water. This will reduce the number of places mosquitoes can lay their eggs and breed.

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

- DEET (N,N-diethyl-m-toluamide)



- Picaridin (KBR 3023)
- Oil of lemon eucalyptus IR3535 (3-[N-Butyl-N-acetyl]-aminopropionic acid, ethyl ester).

2013 TRENDS AND HIGHLIGHTS

- The incidence of WNV infections reported in 2013 (1.75 per 100,000 population), as in 2012 (1.87 per 100,000), is among the highest documented in LAC since WNV appeared in 2003 (Figure 1).
- Of 144 reported symptomatic WNV infections, there were 40 cases of WNV fever and 104 NID cases (52 with meningitis, 46 encephalitis, and 6 acute flaccid paralysis) (Figure 2). Nine WNV-associated deaths were reported among symptomatic cases (6%). Twenty-one asymptomatic donors (13%) were reported from local blood banks (Figure 2).
- The mean age of infected cases was 54 years with the largest proportion in the 55 to 64 years age group (n=46, 28%). This was closely followed by the 65 years and older age group (N=44, 27%). Incidence increased as age increased (Figure 3).
- Most WNV infections were of white or Hispanic/Latino race/ethnicity (n=80, 49%, and n=50, 30%, respectively).
- The male to female ratio was 2.4:1.
- In contrast to previous years, WNV infections occurred in all SPAs. The highest number of people infected with WNV resided in the San Fernando Valley area (n=62). However, the highest incidence rate occurred in the Antelope Valley area with 3.8 cases per 100,000 (n=15) (Figure 5). This is the second year in a row in which the Antelope Valley had an unusually active WNV season. A substantial increase in documented human infections also occurred in SPA 8, increasing from 0 to 3 infections per year previously to 29 infections in 2013.
- This year, human WNV infections occurred from July to November, with the last case occurring on 11/5/2013. The human WNV season in LAC often runs from June through the end of October. Peak onset in 2013 occurred in September, similar to the five-year average (Figure 6). Statewide, 379 human symptomatic cases were reported. Nationally, 2469 were reported.



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

| | 2009 (N=25) | | | 2010 (N=4) | | | 2011 (N=63) | | | 2012 (N=174) | | | 2013 (N=165) | | |
|-----------------------|-------------|------|------------------|------------|------|------------------|-------------|------|------------------|--------------|------|------------------|--------------|------|------------------|
| | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 |
| Age Group | | | | | | | | | | | | | | | |
| <1 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 |
| 1-4 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 |
| 5-14 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 | 2 | 1.1 | 0.2 | 6 | 3.6 | 0.5 |
| 15-34 | 5 | 20.0 | 0.2 | 1 | 25.0 | 0.0 | 5 | 7.9 | 0.2 | 24 | 13.8 | 0.9 | 19 | 11.5 | 0.7 |
| 35-44 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 3 | 4.8 | 0.2 | 17 | 9.8 | 1.3 | 15 | 9.1 | 1.1 |
| 45-54 | 10 | 50.0 | 0.7 | 1 | 25.0 | 0.1 | 16 | 25.4 | 1.2 | 33 | 19.0 | 2.6 | 34 | 20.6 | 2.6 |
| 55-64 | 4 | 16.0 | 0.4 | 0 | 0.0 | 0.0 | 17 | 27.0 | 1.8 | 34 | 19.5 | 3.3 | 46 | 27.9 | 4.5 |
| 65+ | 6 | 24.0 | 0.6 | 2 | 50.0 | 0.2 | 21 | 33.3 | 2.0 | 64 | 36.8 | 5.8 | 45 | 27.3 | 4.0 |
| Unknown | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | |
| Race/Ethnicity | | | | | | | | | | | | | | | |
| Asian | 1 | 4.0 | 0.1 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 | 9 | 5.2 | 0.7 | 6 | 3.6 | 0.4 |
| Black | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 | 3 | 1.7 | 0.4 | 3 | 1.8 | 0.4 |
| Hispanic | 5 | 20.0 | 0.1 | 1 | 25.0 | 0.01 | 26 | 41.3 | 0.5 | 59 | 33.9 | 1.3 | 50 | 30.3 | 1.1 |
| White | 16 | 64.0 | 0.5 | 3 | 75.0 | 0.1 | 30 | 47.6 | 1.0 | 91 | 52.3 | 3.4 | 80 | 48.5 | 3.0 |
| Other | 0 | 0.0 | | 0 | 0.0 | | 2 | 3.2 | | 2 | 1.1 | | 2 | 1.2 | |
| Unknown | 3 | 12.0 | | 0 | 0.0 | | 3 | 4.8 | | 10 | 5.7 | | 24 | 14.5 | |
| SPA | | | | | | | | | | | | | | | |
| 1 | 12 | 48.0 | 3.3 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.3 | 10 | 5.7 | 2.6 | 15 | 9.1 | 3.8 |
| 2 | 9 | 36.0 | 0.4 | 0 | 0.0 | 0.0 | 39 | 61.9 | 1.8 | 73 | 42.0 | 3.4 | 62 | 37.6 | 2.9 |
| 3 | 2 | 8.0 | 0.1 | 2 | 50.0 | 0.1 | 16 | 25.4 | 0.9 | 47 | 27.0 | 2.9 | 23 | 13.9 | 1.4 |
| 4 | 1 | 4.0 | 0.1 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 | 18 | 10.3 | 1.6 | 6 | 3.6 | 0.5 |
| 5 | 1 | 4.0 | 0.2 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.2 | 8 | 4.6 | 1.3 | 2 | 1.2 | 0.3 |
| 6 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 | 2 | 1.1 | 0.2 | 4 | 2.4 | 0.4 |
| 7 | 0 | 0.0 | 0.0 | 2 | 50.0 | 0.1 | 4 | 6.3 | 0.3 | 13 | 7.5 | 1.0 | 24 | 14.5 | 1.8 |
| 8 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 3 | 1.7 | 0.3 | 29 | 17.6 | 2.7 |
| Unknown | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | |

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



Figure 1. Incidence Rates* of West Nile Virus LAC, 2004-2013

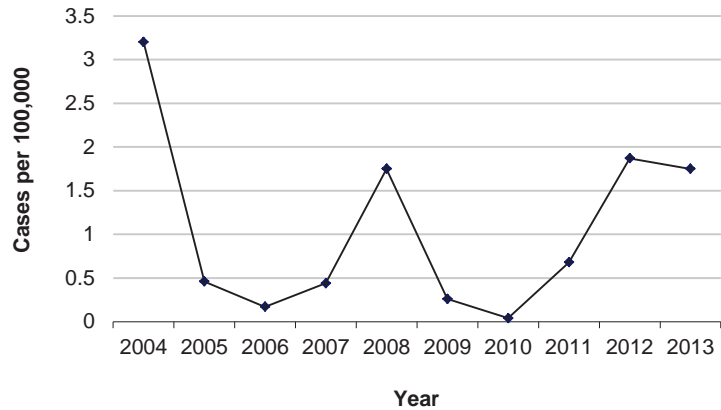
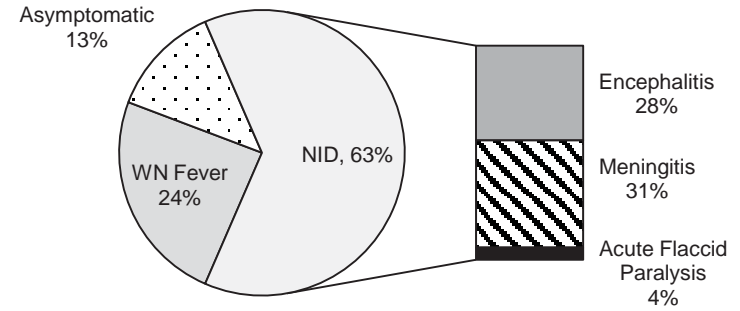


Figure 2. Percent Cases of West Nile Virus by Presentation LAC, 2013 (N=165)



*R

Figure 3. Incidence Rates* of West Nile Virus by Age Group LAC, 2013 (N=165)

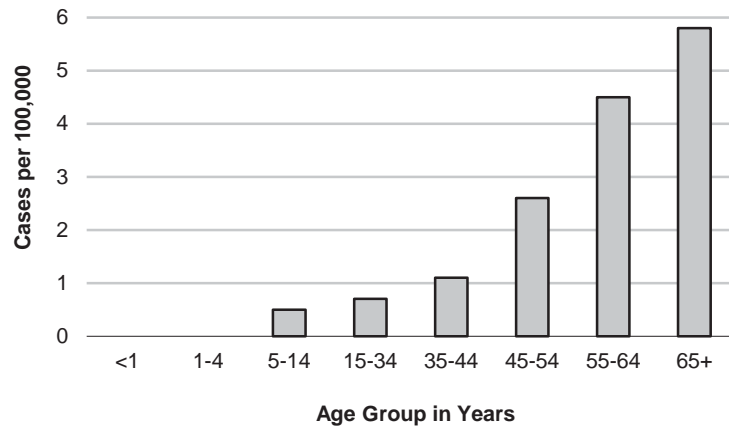
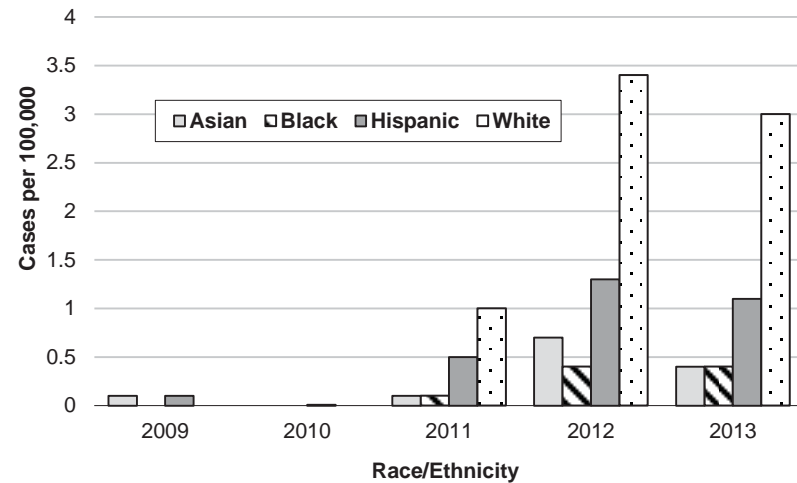
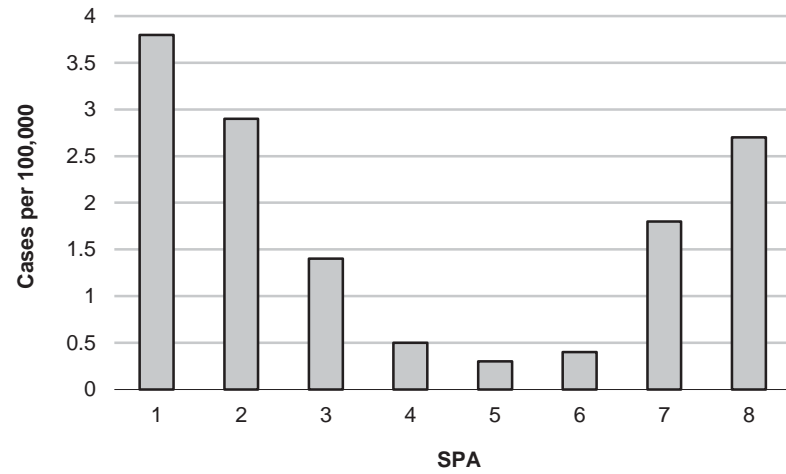


Figure 4. West Nile Virus Incidence* by Race/Ethnicity LAC, 2009-2013

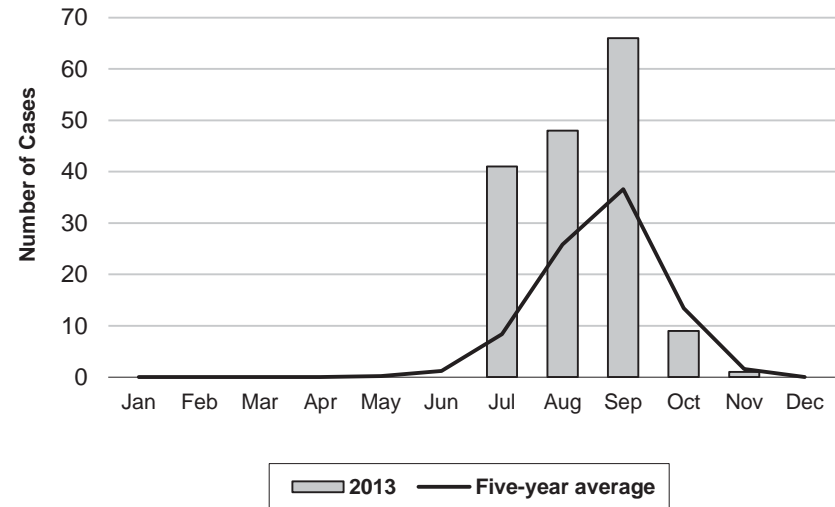




**Figure 5. Incidence Rates* of West Nile Virus by SPA
LAC, 2013 (N=165)**

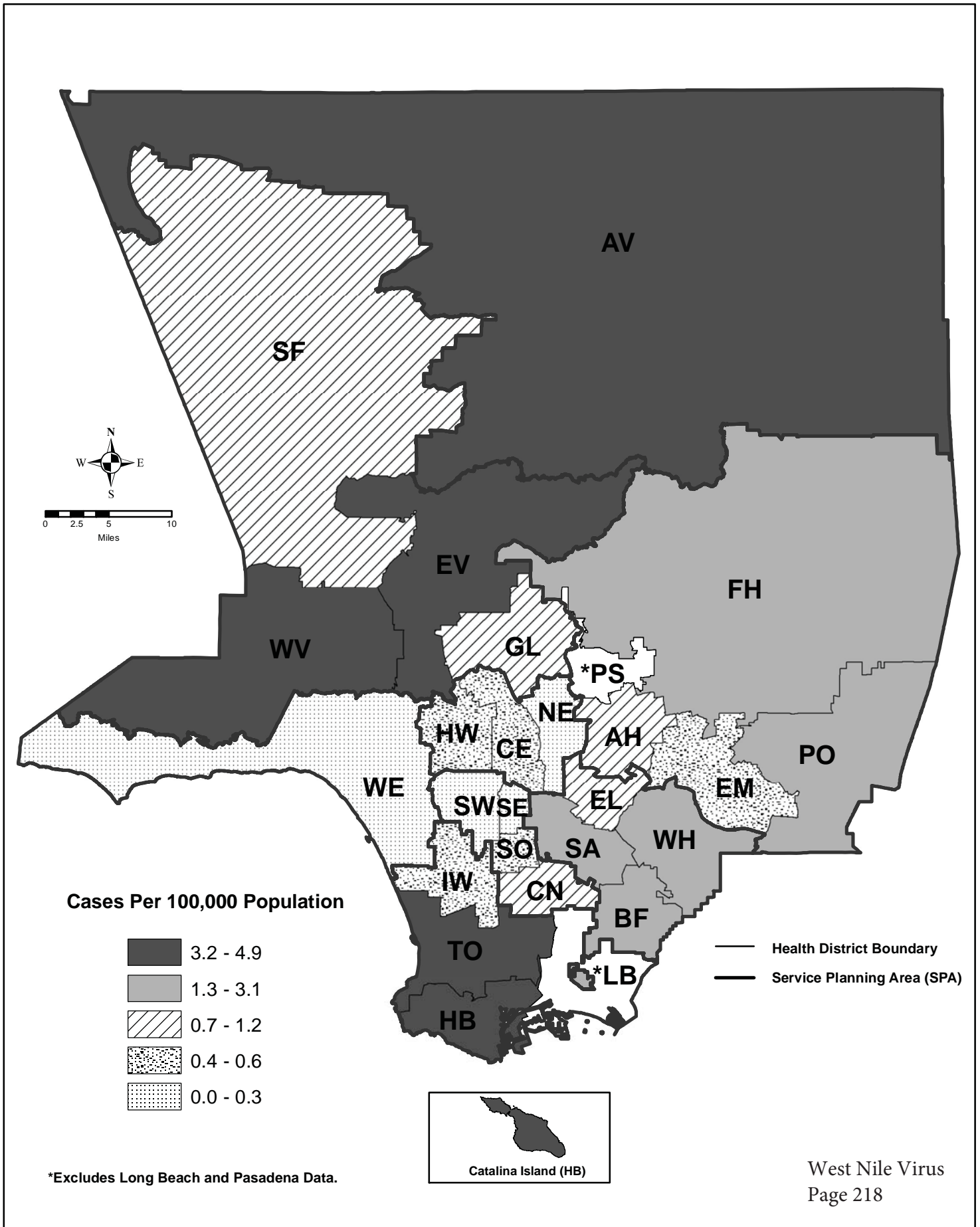


**Figure 6. Reported West Nile Virus Cases by Month of Onset
LAC, 2013 (N=165)**



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

Map 17. West Nile Virus Rates by Health District, Los Angeles County, 2013*





WEST NILE VIRUS

| CRUDE DATA | |
|-------------------------------|-------|
| Number of Cases ^a | 174 |
| Annual Incidence ^b | |
| LA County ^a | 1.87 |
| California ^c | 1.27 |
| United States ^c | 1.82 |
| Age at Diagnosis | |
| Mean | 56.8 |
| Median | 59 |
| Range | 10-94 |

^aIncludes asymptomatic infections.

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

^cCalculated from Final 2012 Reports of Nationally Notifiable Infectious Disease. MMWR 62(33);669-682.

DESCRIPTION

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

Normally transmitted by mosquitoes (usually *Culex* or *Anopheles* species) between bird reservoir hosts, humans are incidentally infected with the virus when bitten by an infected mosquito. About 20% of persons infected will develop WNV fever with symptoms that include fever, headache, rash, muscle weakness, fatigue, nausea and vomiting, and occasionally lymph node swelling. Fewer than 1% will develop more severe illness, manifesting as WNV neuro-invasive disease (NID), including meningitis, encephalitis, and acute flaccid paralysis. WNV-associated meningitis usually involves fever, headache, and stiff neck, and has a good prognosis. WNV-associated encephalitis is commonly associated with fever, altered mental status, headache, and seizures, and usually necessitates a high level of specialized medical care. Long-term neurological and cognitive sequelae are not uncommon.

After being infected with WNV, most people sustain a viremia and may remain asymptomatic or eventually

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

Prevention and control of WNV and other arboviral diseases are most effective with vector management programs. These programs include surveillance for WNV activity in mosquito vectors, birds, horses, other animals, and humans; and implementation of appropriate mosquito control measures to reduce mosquito populations when necessary. When virus activity is detected in an area, residents are advised to increase measures to reduce contact with mosquitoes. Currently, there is no human vaccine available against WNV but several vaccines are under development. Important preventive measures against WNV include the following:

- Apply insect repellent to exposed skin. A higher percentage of DEET in a repellent will provide longer protection. DEET concentrations higher than 50% do not increase the length of protection.
- When possible, wear long-sleeved shirts and long pants when outdoors for long periods of time.
- Stay indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times.
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water. This will reduce the number of places mosquitoes can lay their eggs and breed.

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

DEET (N,N-diethyl-m-toluamide)



Picaridin (KBR 3023)

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

2012 TRENDS AND HIGHLIGHTS

- The number of WNV infections reported in 2012 (N=174) is the second highest number documented in LAC since WNV appeared in 2003 (Figure 1). A peak number of cases has occurred every four years – 2004, 2008, and 2012. These peak years have been characterized by differences in demographic and environmental trends (see ACDC Special Studies Report).
- Of 157 reported symptomatic WNV infections, there were 39 cases of WNV fever and 118 NID cases (67 with meningitis, 47 encephalitis, and 4 acute flaccid paralysis) (Figure 2). Six WNV-associated deaths were reported among symptomatic cases (3.8%). Seventeen asymptomatic donors (9.7%) were reported from local blood banks, organ procurement agencies, and a cord blood bank.
- The mean age of cases was 56.8 years with the largest proportion of cases in the 65 years and older age group (n=64, 37%). Incidence increased as age increased (Figure 3).
- Most cases were of Hispanic/Latino or white race/ethnicity (n=59, 34%, and n=91, 52%, respectively).
- The male to female ratio was 1.7:1.
- WNV cases occurred in all SPAs. The highest number of cases resided in the San Fernando (n=73) and San Gabriel Valley (n=47) areas. Though relatively fewer cases occurred in the Antelope Valley area (n=10), the incidence rate (2.6 per 100,000) was almost just as high (Figure 5).
- Peak onset occurred in September (Figure 6). The last case had an onset on 11/25/2012, which is the latest onset ever documented in LAC. Statewide, 471 human cases were reported in 31 counties. Nationally, the number of WNV cases reported, 5387, is also the highest since 2003.



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

| | 2008 (N=170) | | | 2009 (N=25) | | | 2010 (N=4) | | | 2011 (N=63) | | | 2012 (N=174) | | |
|-----------------------|--------------|------|------------------|-------------|------|------------------|------------|------|------------------|-------------|------|------------------|--------------|------|------------------|
| | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 |
| Age Group | | | | | | | | | | | | | | | |
| <1 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 |
| 1-4 | 1 | 0.6 | 0.2 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 |
| 5-14 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 | 2 | 1.1 | 0.2 |
| 15-34 | 19 | 11.2 | 0.7 | 5 | 20.0 | 0.2 | 1 | 25.0 | 0.0 | 5 | 7.9 | 0.2 | 24 | 13.8 | 0.9 |
| 35-44 | 15 | 8.8 | 1.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 3 | 4.8 | 0.2 | 17 | 9.8 | 1.3 |
| 45-54 | 34 | 20.0 | 2.5 | 10 | 50.0 | 0.7 | 1 | 25.0 | 0.1 | 16 | 25.4 | 1.2 | 33 | 19.0 | 2.6 |
| 55-64 | 36 | 21.2 | 3.9 | 4 | 16.0 | 0.4 | 0 | 0.0 | 0.0 | 17 | 27.0 | 1.8 | 34 | 19.5 | 3.3 |
| 65+ | 65 | 38.2 | 6.4 | 6 | 24.0 | 0.6 | 2 | 50.0 | 0.2 | 21 | 33.3 | 2.0 | 64 | 36.8 | 5.8 |
| Unknown | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | |
| Race/Ethnicity | | | | | | | | | | | | | | | |
| Asian | 6 | 3.5 | 0.5 | 1 | 4.0 | 0.1 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 | 9 | 5.2 | 0.7 |
| Black | 5 | 2.9 | 0.6 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 | 3 | 1.7 | 0.4 |
| Hispanic | 68 | 40.0 | 1.5 | 5 | 20.0 | 0.1 | 1 | 25.0 | 0.01 | 26 | 41.3 | 0.5 | 59 | 33.9 | 1.3 |
| White | 75 | 44.1 | 2.6 | 16 | 64.0 | 0.5 | 3 | 75.0 | 0.1 | 30 | 47.6 | 1.0 | 91 | 52.3 | 3.4 |
| Other | 3 | 1.8 | 12.2 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 2 | 3.2 | | 2 | 1.1 | |
| Unknown | 13 | 7.6 | | 3 | 12.0 | | 0 | 0.0 | | 3 | 4.8 | | 10 | 5.7 | |
| SPA | | | | | | | | | | | | | | | |
| 1 | 5 | 2.9 | 1.4 | 12 | 48.0 | 3.3 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.3 | 10 | 5.7 | 2.6 |
| 2 | 37 | 21.8 | 1.7 | 9 | 36.0 | 0.4 | 0 | 0.0 | 0.0 | 39 | 61.9 | 1.8 | 73 | 42.0 | 3.4 |
| 3 | 61 | 35.9 | 3.5 | 2 | 8.0 | 0.1 | 2 | 50.0 | 0.1 | 16 | 25.4 | 0.9 | 47 | 27.0 | 2.9 |
| 4 | 12 | 7.1 | 0.9 | 1 | 4.0 | 0.1 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 | 18 | 10.3 | 1.6 |
| 5 | 1 | 0.6 | 0.2 | 1 | 4.0 | 0.2 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.2 | 8 | 4.6 | 1.3 |
| 6 | 6 | 3.5 | 0.6 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 | 2 | 1.1 | 0.2 |
| 7 | 44 | 25.9 | 3.2 | 0 | 0.0 | 0.0 | 2 | 50.0 | 0.1 | 4 | 6.3 | 0.3 | 13 | 7.5 | 1.0 |
| 8 | 4 | 2.4 | 0.4 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 3 | 1.7 | 0.3 |
| Unknown | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | |

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



Figure 1. Incidence Rates* of West Nile Virus LAC, 2004-2012

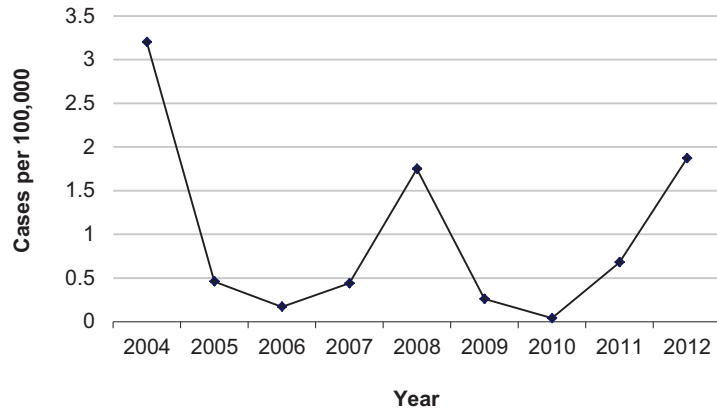
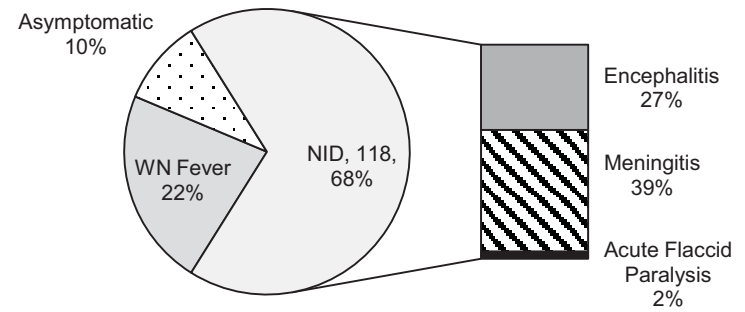


Figure 2. Percent Cases of West Nile Virus by Presentation LAC, 2012 (N=174)



*R

Figure 3. Incidence Rates* of West Nile Virus by Age Group LAC, 2012 (N=174)

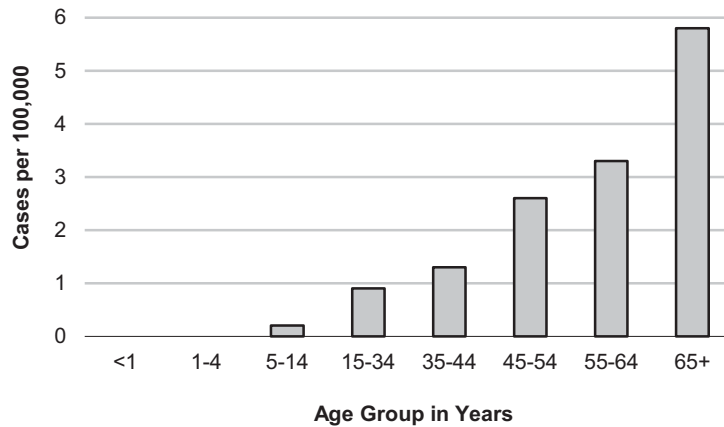
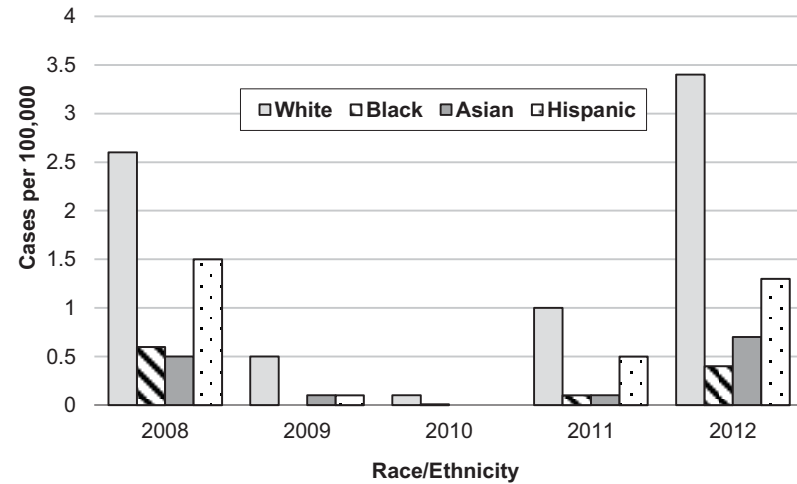
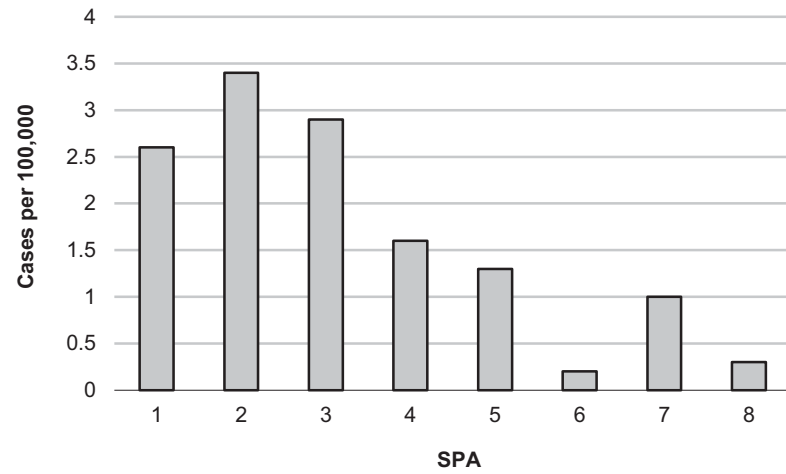


Figure 4. West Nile Virus Incidence* by Race/Ethnicity LAC, 2008-2012

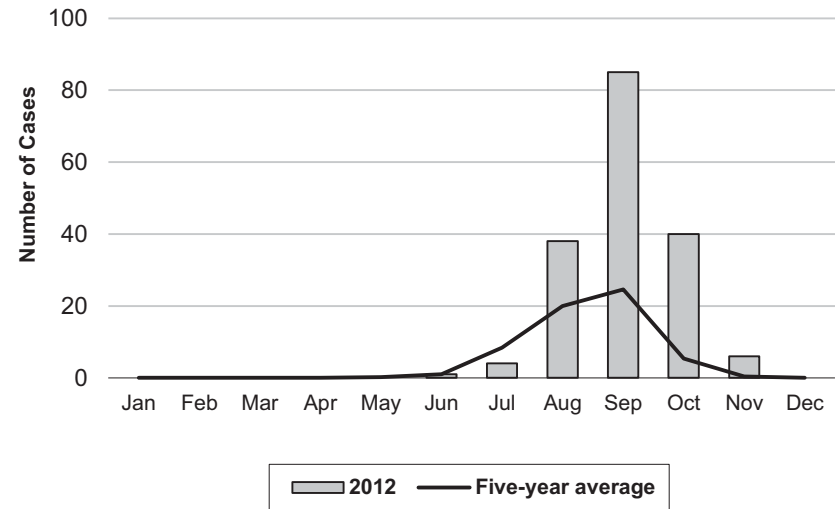




**Figure 5. Incidence Rates* of West Nile Virus by SPA
LAC, 2012 (N=174)**

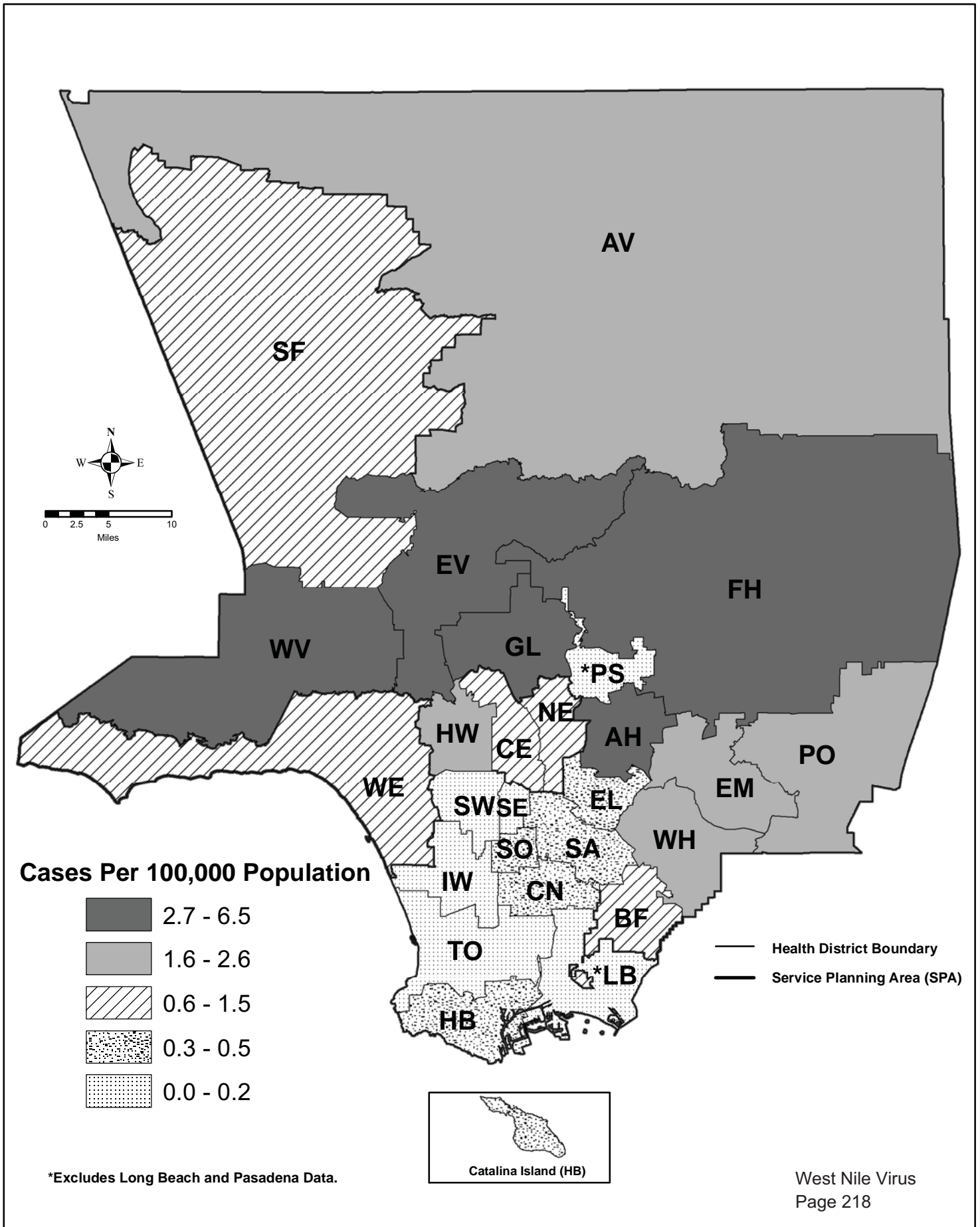


**Figure 6. Reported West Nile Virus Cases by Month of Onset
LAC, 2012 (N=174)**



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

Map 14. West Nile Virus Rates by Health District, Los Angeles County, 2012*





WEST NILE VIRUS

| CRUDE DATA | |
|-------------------------------|-------|
| Number of Cases | 63 |
| Annual Incidence ^a | |
| LA County | 0.64 |
| California | 0.42 |
| United States | 0.23 |
| Age at Diagnosis | |
| Mean | 57.1 |
| Median | 57 |
| Range | 14-88 |

^aCases per 100,000 population.

^bCalculated from Final 2011 Reports of Nationally Notifiable Infectious Disease. MMWR 61(32);625-637.

DESCRIPTION

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

Normally transmitted by mosquitoes (usually *Culex* or *Anopheles* species) between bird reservoir hosts, humans are incidentally infected with the virus when bitten by an infected mosquito. About 20% of persons infected will develop WNV fever with symptoms that include fever, headache, rash, muscle weakness, fatigue, nausea and vomiting, and occasionally lymph node swelling. Fewer than 1% will develop more severe illness, manifesting as WNV neuro-invasive disease (NID), including meningitis, encephalitis, and acute flaccid paralysis. WNV-associated meningitis usually involves fever, headache, and stiff neck, and has a good prognosis. WNV-associated encephalitis is commonly associated with fever, altered mental status, headache, and seizures, and usually necessitates a high level of specialized medical care. Long-term neurological and cognitive sequelae are not uncommon.

Since most persons infected with WNV will not develop clinical illness or symptoms, transmission via blood donation is problematic. Beginning 2003, blood

products have been screened for WNV utilizing polymerase chain reaction (PCR) testing.

To date, there have been no blood transfusion-associated secondary WNV infections from asymptomatic WNV-infected blood donors from Los Angeles (LAC) residents. However, four cases of WNV-associated infection including three cases of NID were documented from a LAC organ donor, not known to be infected with WNV infection at the time of organ donation. Additional routes of transmission that can occur include vertical transmission transplacentally, occupational exposure, and through breast milk.

Prevention and control of WNV and other arboviral diseases are most effective with vector management programs. These programs include surveillance for WNV activity in mosquito vectors, birds, horses, other animals, and humans; and implementation of appropriate mosquito control measures to reduce mosquito populations when necessary. When virus activity is detected in an area, residents are advised to increase measures to reduce contact with mosquitoes. Currently, there is no human vaccine available against WNV but several vaccines are under development. Important preventive measures against WNV include the following:

- Apply insect repellent to exposed skin. A higher percentage of DEET in a repellent will provide longer protection. DEET concentrations higher than 50% do not increase the length of protection.
- When possible, wear long-sleeved shirts and long pants when outdoors for long periods of time.
- Stay indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times.
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water. This will reduce the number of places mosquitoes can lay their eggs and breed.

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

DEET (N,N-diethyl-m-toluamide)
 Picaridin (KBR 3023)
 Oil of lemon eucalyptus.



2011 TRENDS AND HIGHLIGHTS

- The number of WNV infections reported in 2011 (N=63) was nearly 16 times that of the number reported the previous year, bouncing back from an all-time low of four cases in 2010. This reaffirms that WNV remains entrenched in the ecology of Los Angeles County.
- Of 58 reported symptomatic WNV infections, there were 17 cases of WNV fever and 41 (65%) neuro-invasive disease cases (21 meningitis, 15 encephalitis, and 5 acute flaccid paralysis). Five asymptomatic blood donors were reported from local blood banks. Four (6%) WNV- associated deaths were reported.
- An LAC resident organ donor with WNV fever, unknown at time of organ donation to four recipients, resulted in three cases of encephalitis (with two deaths) and one asymptomatic WNV infection in a liver recipient from LAC. LAC Department of Public Health worked with Centers for Disease Control and Prevention, the local Southern California organ procurement agency, and other institutions to ensure no additional organs were transplanted from this donor. Additional testing of the organ donor tissue included the lymph nodes and spleen confirmed WNV infection by PCR.



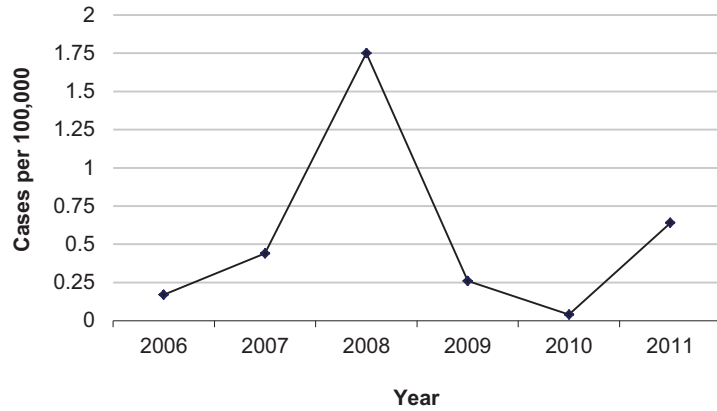
**Reported West Nile Virus Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
Los Angeles County, 2007-2011**

| | 2007 (N=43) | | | 2008 (N=170) | | | 2009 (N=25) | | | 2010 (N=4) | | | 2011 (N=63) | | |
|-----------------------|-------------|------|------------------|--------------|------|------------------|-------------|------|------------------|------------|------|------------------|-------------|------|------------------|
| | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 |
| Age Group | | | | | | | | | | | | | | | |
| <1 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 |
| 1-4 | 0 | 0.0 | 0.0 | 1 | 0.6 | 0.2 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 |
| 5-14 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 |
| 15-34 | 3 | 7.0 | 0.1 | 19 | 11.2 | 0.7 | 5 | 20.0 | 0.2 | 1 | 25.0 | 0.0 | 5 | 7.9 | 0.2 |
| 35-44 | 0 | 0.0 | 0.0 | 15 | 8.8 | 1.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 3 | 4.8 | 0.2 |
| 45-54 | 9 | 20.9 | 0.7 | 34 | 20.0 | 2.5 | 10 | 50.0 | 0.7 | 1 | 25.0 | 0.1 | 16 | 25.4 | 1.2 |
| 55-64 | 12 | 27.9 | 1.4 | 36 | 21.2 | 3.9 | 4 | 16.0 | 0.4 | 0 | 0.0 | 0.0 | 17 | 27.0 | 1.8 |
| 65+ | 19 | 44.2 | 1.9 | 65 | 38.2 | 6.4 | 6 | 24.0 | 0.6 | 2 | 50.0 | 0.2 | 21 | 33.3 | 2.0 |
| Unknown | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | | |
| Race/Ethnicity | | | | | | | | | | | | | | | |
| Asian | 0 | 0.0 | 0.0 | 6 | 3.5 | 0.5 | 1 | 4.0 | 0.1 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 |
| Black | 0 | 0.0 | 0.0 | 5 | 2.9 | 0.6 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 |
| Hispanic | 12 | 27.9 | 0.3 | 68 | 40.0 | 1.5 | 5 | 20.0 | 0.1 | 1 | 25.0 | 0.01 | 26 | 41.3 | 0.5 |
| White | 29 | 67.4 | 1.0 | 75 | 44.1 | 2.6 | 16 | 64.0 | 0.5 | 3 | 75.0 | 0.1 | 30 | 47.6 | 1.0 |
| Other | 0 | 0.0 | 0.0 | 3 | 1.8 | 12.2 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 2 | 3.2 | |
| Unknown | 2 | 4.7 | | 13 | 7.6 | | 3 | 12.0 | | 0 | 0.0 | | 3 | 4.8 | |
| SPA | | | | | | | | | | | | | | | |
| 1 | 1 | 2.3 | 0.3 | 5 | 2.9 | 1.4 | 12 | 48.0 | 3.3 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.3 |
| 2 | 27 | 62.8 | 1.3 | 37 | 21.8 | 1.7 | 9 | 36.0 | 0.4 | 0 | 0.0 | 0.0 | 39 | 61.9 | 1.8 |
| 3 | 9 | 20.9 | 0.5 | 61 | 35.9 | 3.5 | 2 | 8.0 | 0.1 | 2 | 50.0 | 0.1 | 16 | 25.4 | 0.9 |
| 4 | 2 | 4.7 | 0.2 | 12 | 7.1 | 0.9 | 1 | 4.0 | 0.1 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 |
| 5 | 0 | 0.0 | 0.0 | 1 | 0.6 | 0.2 | 1 | 4.0 | 0.2 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.2 |
| 6 | 1 | 2.3 | 0.1 | 6 | 3.5 | 0.6 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 1.6 | 0.1 |
| 7 | 2 | 4.7 | 0.1 | 44 | 25.9 | 3.2 | 0 | 0.0 | 0.0 | 2 | 50.0 | 0.1 | 4 | 6.3 | 0.3 |
| 8 | 1 | 2.3 | 0.1 | 4 | 2.4 | 0.4 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 |
| Unknown | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | | |

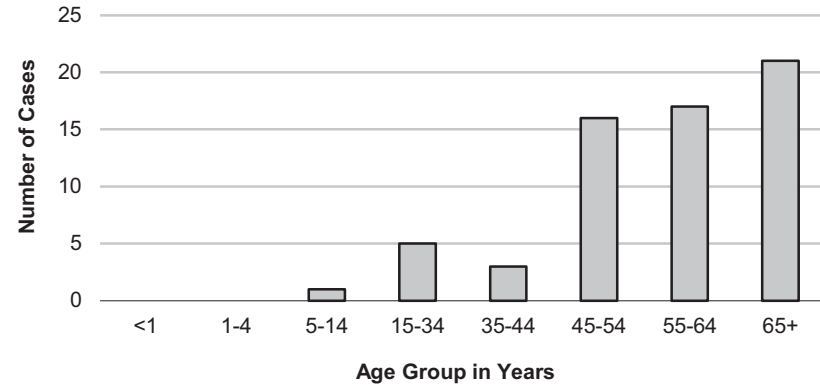
*Rates calculated based on less than 19 cases or events are considered unreliable.



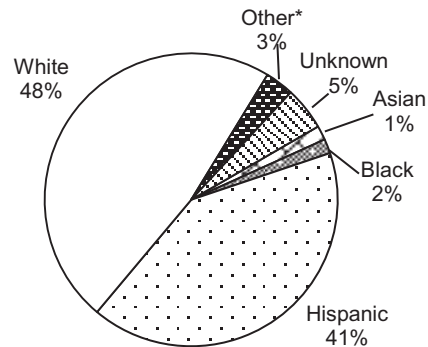
**Figure 1. Incidence Rates of West Nile Virus
LAC, 2006-2011**



**Figure 2. Incidence Rates of West Nile Virus by Age Group
LAC, 2011 (N=63)**



**Figure 3. Percent Cases of West Nile Virus by
Race/Ethnicity
LAC, 2011 (N=63)**



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

**Figure 4. Incidence Rates of West Nile Virus by SPA
LAC, 2011 (N=63)**

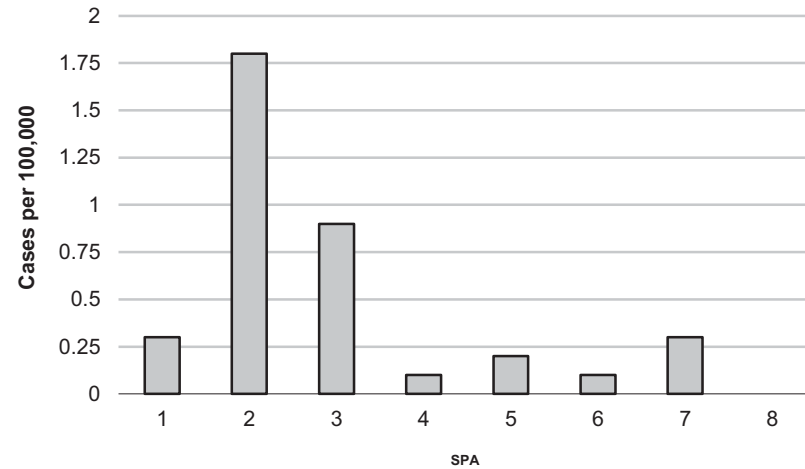




Figure 5. Reported West Nile Virus Cases by Month of Onset LAC, 2011 (N=63)

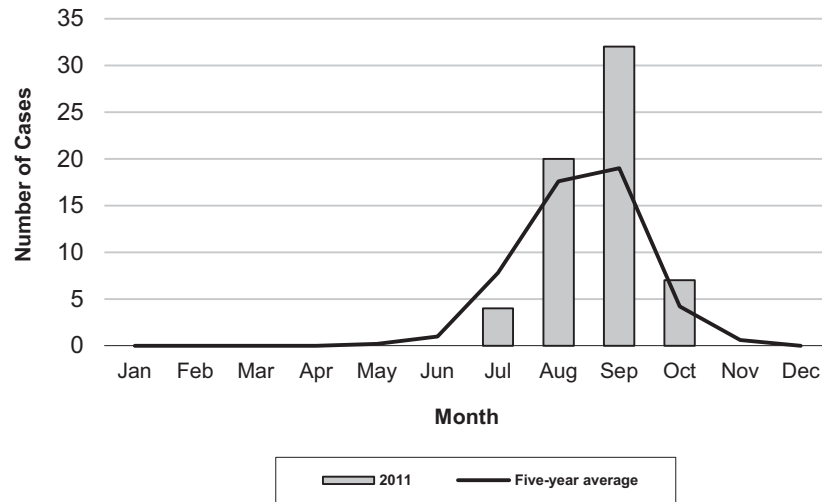
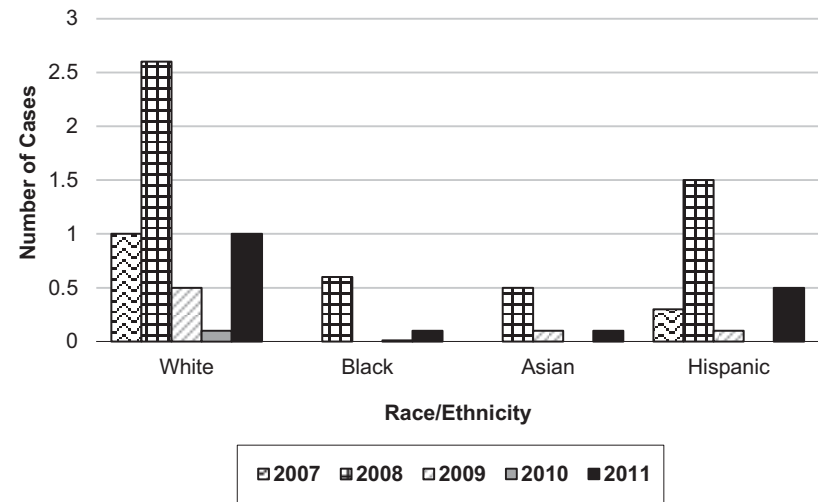
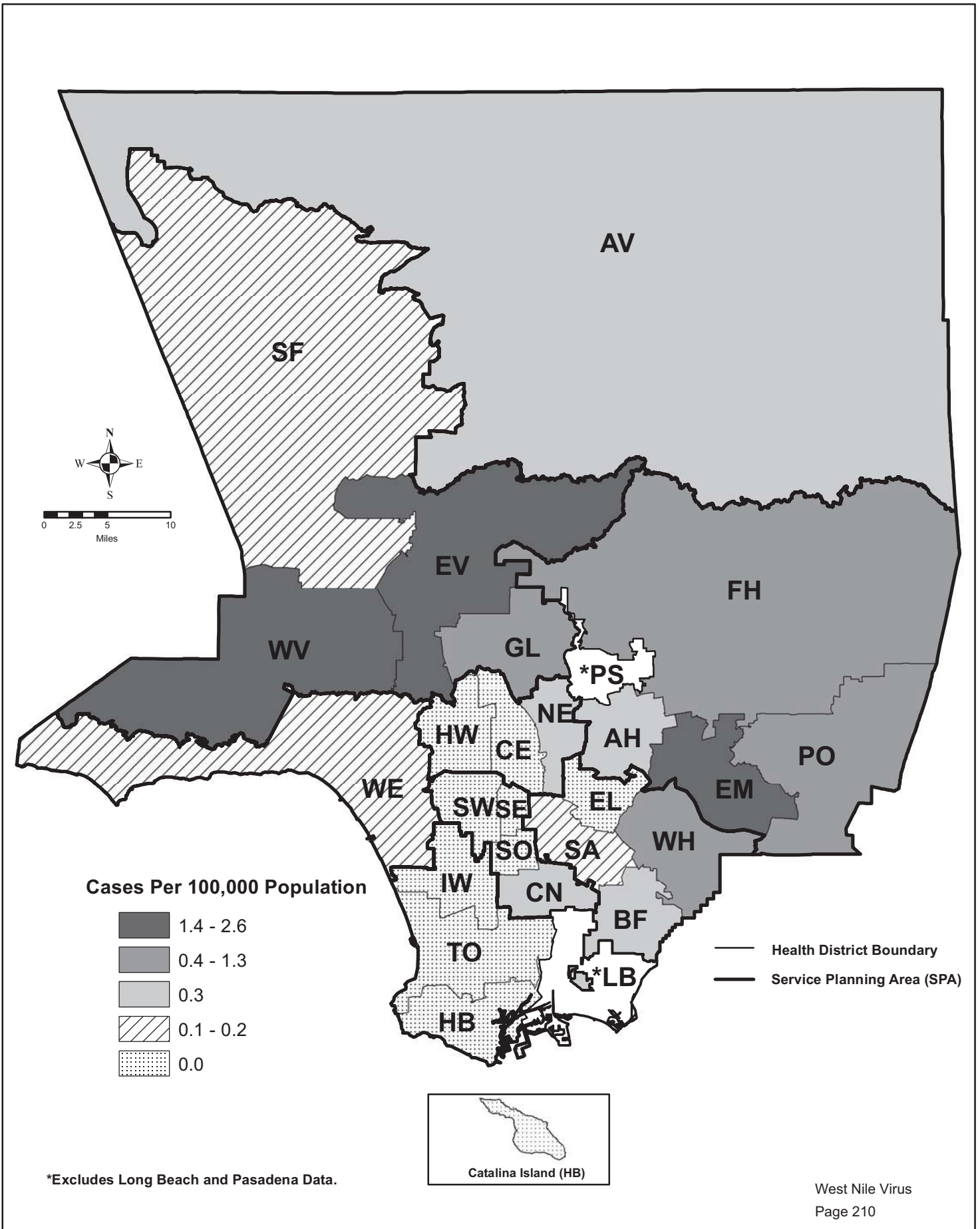


Figure 6. West Nile Virus Incidence by Race/Ethnicity LAC, 2007-2011



Map 16. West Nile Virus Rates by Health District, Los Angeles County, 2011*





WEST NILE VIRUS

| CRUDE DATA | |
|-------------------------------|-------------------|
| Number of Cases | 4 |
| Annual Incidence ^a | |
| LA County | 0.04 ^b |
| California | -- |
| United States | -- |
| Age at Diagnosis | |
| Mean | 55.5 |
| Median | 49 |
| Range | 26-78 |

^aCases per 100,000 population.

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

^cSee Final Summary of Nationally Notifiable Infectious Diseases, United States on MMWR website http://www.cdc.gov/mmwr/mmwr_nd/index.html.

DESCRIPTION

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

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

Since most persons infected with WNV will not develop clinical illness or symptoms, transmission via blood donation is problematic. Beginning 2003, blood products have been screened for WNV utilizing polymerase chain reaction (PCR) testing.

No transmission associated with blood products has been reported in LAC. Additional routes of transmission that have been documented include transplantation of WNV-infected organs, vertical transmission transplacentally, occupational exposure, and through breast milk.

Prevention and control of WNV and other arboviral diseases are most effective with vector management programs. These programs include surveillance for WNV activity in mosquito vectors, birds, horses, other animals, and humans; and implementation of appropriate mosquito control measures to reduce mosquito populations when necessary. When virus activity is detected in an area, residents are advised to increase measures to reduce contact with mosquitoes. Currently, there is no human vaccine available against WNV but several vaccines are under development. Important preventive measures against WNV include the following:

- Apply insect repellent to exposed skin. A higher percentage of DEET in a repellent will provide longer protection. DEET concentrations higher than 50% do not increase the length of protection.
- When possible, wear long-sleeved shirts and long pants when outdoors for long periods of time.
- Stay indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times.
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water. This will reduce the number of places mosquitoes can lay their eggs and breed.

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

DEET (N,N-diethyl-m-toluamide)
Picaridin (KBR 3023)
Oil of lemon eucalyptus.



2010 TRENDS AND HIGHLIGHTS

- The number of WNV infections reported in 2010 (n=4) was at an all time low since its introduction to California in 2003.
- WNV manifested as neuro-invasive disease in three reported infections (75%): two meningitis and one encephalitis. No WNV-associated deaths were reported.
- There was markedly less WNV activity in the LAC environment in 2009-2010, as measured in dead birds and mosquitoes.



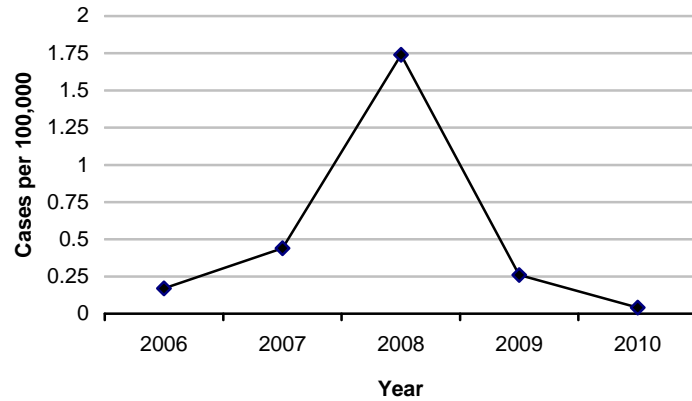
**Reported West Nile Virus Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
Los Angeles County, 2006-2010**

| | 2006 (N=16) | | | 2007 (N=43) | | | 2008 (N=170) | | | 2009 (N=25) | | | 2010 (N=4) | | |
|-----------------------|-------------|------|------------------|-------------|------|------------------|--------------|------|------------------|-------------|------|------------------|------------|------|------------------|
| | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 |
| Age Group | | | | | | | | | | | | | | | |
| <1 | 0 | 0.0 | | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | |
| 1-4 | 0 | 0.0 | | 0 | 0.0 | 0.0 | 1 | 0.6 | 0.2 | 0 | 0.0 | 0.0 | 0 | 0.0 | |
| 5-14 | 0 | 0.0 | | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | |
| 15-34 | 2 | 12.5 | | 3 | 7.0 | 0.1 | 19 | 11.2 | 0.7 | 5 | 20.0 | 0.2 | 1 | 25.0 | |
| 35-44 | 5 | 31.3 | | 0 | 0.0 | 0.0 | 15 | 8.8 | 1.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | |
| 45-54 | 3 | 18.8 | | 9 | 20.9 | 0.7 | 34 | 20.0 | 2.5 | 10 | 50.0 | 0.7 | 1 | 25.0 | |
| 55-64 | 3 | 18.8 | | 12 | 27.9 | 1.4 | 36 | 21.2 | 3.9 | 4 | 16.0 | 0.4 | 0 | 0.0 | |
| 65+ | 3 | 18.8 | | 19 | 44.2 | 1.9 | 65 | 38.2 | 6.4 | 6 | 24.0 | 0.6 | 2 | 50.0 | |
| Unknown | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | |
| Race/Ethnicity | | | | | | | | | | | | | | | |
| Asian | 1 | 6.3 | | 0 | 0.0 | 0.0 | 6 | 3.5 | 0.5 | 1 | 4.0 | 0.1 | 0 | 0.0 | |
| Black | 0 | 0.0 | | 0 | 0.0 | 0.0 | 5 | 2.9 | 0.6 | 0 | 0.0 | 0.0 | 0 | 0.0 | |
| Hispanic | 2 | 12.5 | | 12 | 27.9 | 0.3 | 68 | 40.0 | 1.5 | 5 | 20.0 | 0.1 | 1 | 25.0 | |
| White | 13 | 81.3 | | 29 | 67.4 | 1.0 | 75 | 44.1 | 2.6 | 16 | 64.0 | 0.5 | 3 | 75.0 | |
| Other | 0 | 0.0 | | 0 | 0.0 | 0.0 | 3 | 1.8 | 12.2 | 0 | 0.0 | 0.0 | 0 | 0.0 | |
| Unknown | 0 | 0.0 | | 2 | 4.7 | | 13 | 7.6 | | 3 | 12.0 | | 0 | 0.0 | |
| SPA | | | | | | | | | | | | | | | |
| 1 | 0 | 0.0 | | 1 | 2.3 | 0.3 | 5 | 2.9 | 1.4 | 12 | 48.0 | 3.3 | 0 | 0.0 | |
| 2 | 9 | 56.3 | | 27 | 62.8 | 1.3 | 37 | 21.8 | 1.7 | 9 | 36.0 | 0.4 | 0 | 0.0 | |
| 3 | 4 | 25.0 | | 9 | 20.9 | 0.5 | 61 | 35.9 | 3.5 | 2 | 8.0 | 0.1 | 2 | 50.0 | |
| 4 | 3 | 18.8 | | 2 | 4.7 | 0.2 | 12 | 7.1 | 0.9 | 1 | 4.0 | 0.1 | 0 | 0.0 | |
| 5 | 0 | 0.0 | | 0 | 0.0 | 0.0 | 1 | 0.6 | 0.2 | 1 | 4.0 | 0.2 | 0 | 0.0 | |
| 6 | 0 | 0.0 | | 1 | 2.3 | 0.1 | 6 | 3.5 | 0.6 | 0 | 0.0 | 0.0 | 0 | 0.0 | |
| 7 | 0 | 0.0 | | 2 | 4.7 | 0.1 | 44 | 25.9 | 3.2 | 0 | 0.0 | 0.0 | 2 | 50.0 | |
| 8 | 0 | 0.0 | | 1 | 2.3 | 0.1 | 4 | 2.4 | 0.4 | 0 | 0.0 | 0.0 | 0 | 0.0 | |
| Unknown | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | |

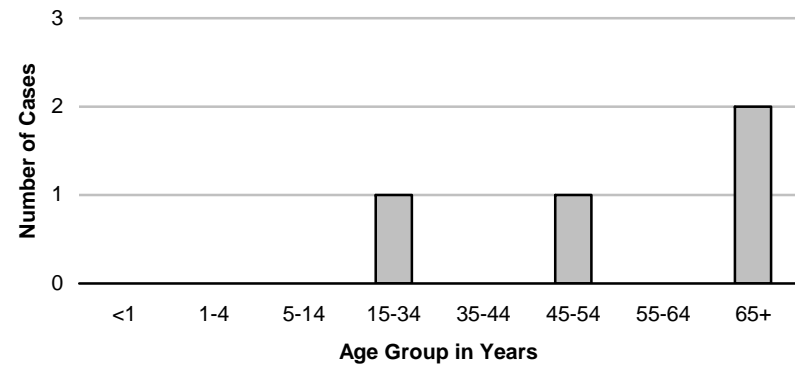
*Rates calculated based on less than 19 cases or events are considered unreliable.



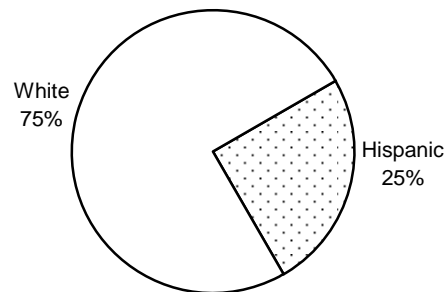
**Figure 1. Incidence Rates of West Nile Virus
LAC, 2006-2010**



**Figure 2. Incidence Rates of West Nile Virus by Age Group
LAC, 2010 (N=4)**

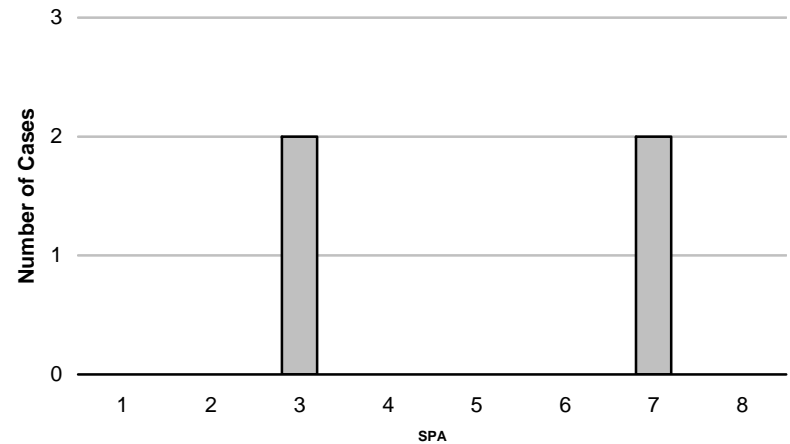


**Figure 3. Percent Cases of West Nile Virus by
Race/Ethnicity
LAC, 2010 (N=4)**



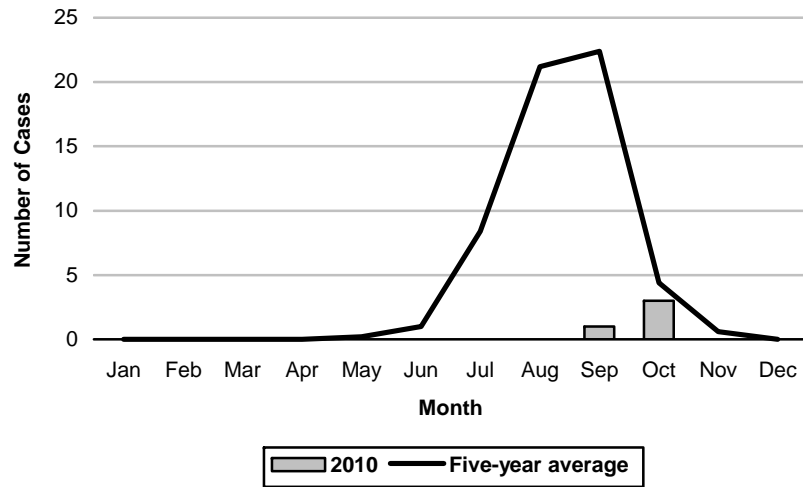
* Other includes Native American and any additional racial/ethnic group that cannot be categorized as Asian, black, Hispanic, or white.

**Figure 4. Incidence Rates of West Nile Virus by SPA
LAC, 2010 (N=4)**

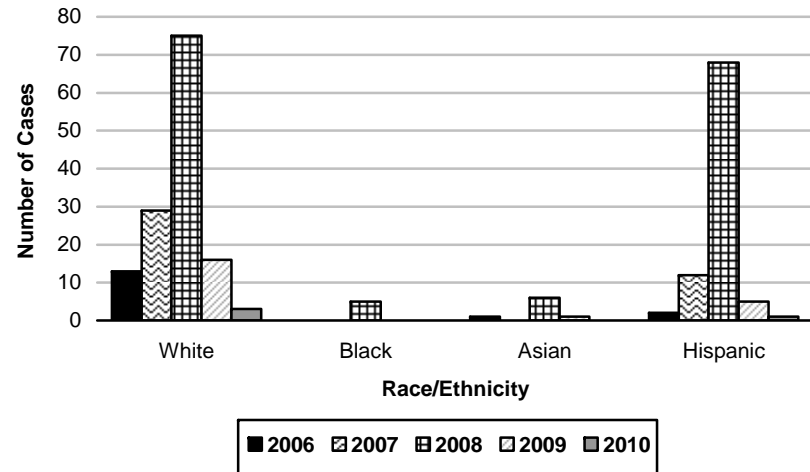




**Figure 5. Reported West Nile Virus Cases by Month of Onset
 LAC, 2010 (N=4)**



**Figure 6. West Nile Virus Incidence by Race/Ethnicity
 LAC, 2006-2010**





WEST NILE VIRUS

| CRUDE DATA | |
|-------------------------------|-------|
| Number of Cases | 25 |
| Annual Incidence ^a | |
| LA County | 0.26 |
| California ^b | 1.22 |
| United States ^b | 0.45 |
| Age at Diagnosis | |
| Mean | 53.4 |
| Median | 53 |
| Range | 15-87 |

^aCases per 100,000 population.

^bCalculated from Final 2008 Reports of Nationally Notifiable Infectious Disease. MMWR 58(31);856-857;859-869.

DESCRIPTION

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

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

Since most persons infected with WNV will not develop clinical illness or symptoms, blood donation is problematic. Beginning 2003, blood donors were screened for WNV infection utilizing polymerase chain reaction (PCR) testing.

No transmission associated with blood products has been reported in LAC. Additional routes of transmission that have been documented include transplantation of WNV-infected organs, transplacental (mother-to-child), occupational exposures, and through breast milk.

Prevention and control of WNV and other arboviral diseases is most effective with vector management programs. These programs include surveillance for WNV activity in mosquito vectors, birds, horses, other animals, and humans; and implementation of appropriate mosquito control measures to reduce mosquito populations when necessary. When virus activity is detected in an area, residents are advised to increase measures to reduce contact with mosquitoes. Currently, there is no human vaccine available against WNV but several vaccines are under development. Important preventive measures against WNV include the following:

- Apply insect repellent to exposed skin. A higher percentage of DEET in a repellent will provide longer protection. DEET concentrations higher than 50% do not increase the length of protection.
- When possible, wear long-sleeved shirts and long pants when outdoors for long periods of time.
- Stay indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times.
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water. This will reduce the number of places mosquitoes can lay their eggs and breed.

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

DEET (N,N-diethyl-m-toluamide)
Picaridin (KBR 3023)
Oil of lemon eucalyptus.



2009 TRENDS AND HIGHLIGHTS

- The number of WNV infections reported in 2009 (n=25) decreased by 85% compared to 2008 (n=170)
- WNV manifested as neuro-invasive disease in 15 reported infections (60%): 9 meningitis and 6 encephalitis. There were five asymptomatic infections identified through blood donor screening and one WNV-associated death.
- Unlike previous years in which the highest incidences were reported from the San Fernando Valley or the San Gabriel Valley regions, most cases occurred in the Antelope Valley in 2009 (Figure 4).
- The WNV season shifted one month earlier in 2009 compared to the previous five-year average with onsets occurring May through September (Figure 5).



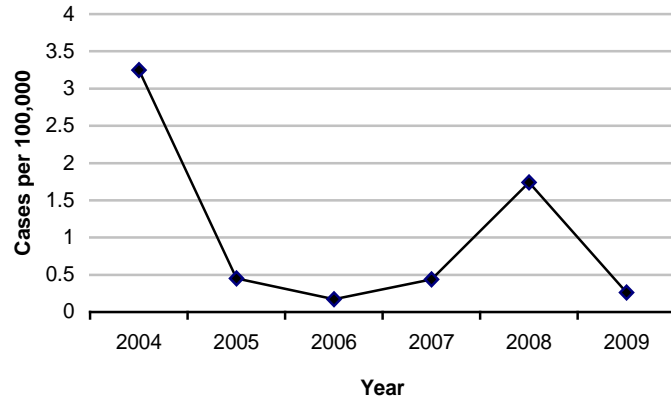
**Reported West Nile Virus Cases and Rates* per 100,000 by Age Group, Race/Ethnicity, and SPA
Los Angeles County, 2005-2009**

| | 2005 (N=43) | | | 2006 (N=16) | | | 2007 (N=43) | | | 2008 (N=170) | | | 2009 (N=25) | | |
|-----------------------|-------------|------|------------------|-------------|------|------------------|-------------|------|------------------|--------------|------|------------------|-------------|------|------------------|
| | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 |
| Age Group | | | | | | | | | | | | | | | |
| <1 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0 | 0 |
| 1-4 | 1 | 2.3 | 0.2 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 0.6 | 0.2 | 0 | 0 | 0 |
| 5-14 | 1 | 2.3 | 0.1 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0 | 0 |
| 15-34 | 7 | 16.3 | 0.2 | 2 | 12.5 | 0.1 | 3 | 7.0 | 0.1 | 19 | 11.2 | 0.7 | 5 | 20.0 | 0.2 |
| 35-44 | 4 | 9.3 | 0.3 | 5 | 31.3 | 0.3 | 0 | 0.0 | 0.0 | 15 | 8.8 | 1.0 | 0 | 0 | 0 |
| 45-54 | 8 | 18.6 | 0.6 | 3 | 18.8 | 0.2 | 9 | 20.9 | 0.7 | 34 | 20.0 | 2.5 | 10 | 50.0 | 0.7 |
| 55-64 | 8 | 18.6 | 1.0 | 3 | 18.8 | 0.3 | 12 | 27.9 | 1.4 | 36 | 21.2 | 3.9 | 4 | 16.0 | 0.4 |
| 65+ | 14 | 32.6 | 1.5 | 3 | 18.8 | 0.3 | 19 | 44.2 | 1.9 | 65 | 38.2 | 6.4 | 6 | 24.0 | 0.6 |
| Unknown | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0 | |
| Race/Ethnicity | | | | | | | | | | | | | | | |
| Asian | 2 | 4.7 | 0.2 | 1 | 6.3 | 0.1 | 0 | 0.0 | 0.0 | 6 | 3.5 | 0.5 | 1 | 4.0 | 0.1 |
| Black | 1 | 2.3 | 0.1 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 5 | 2.9 | 0.6 | 0 | 0 | 0 |
| Hispanic | 17 | 39.5 | 0.4 | 2 | 12.5 | 0.0 | 12 | 27.9 | 0.3 | 68 | 40.0 | 1.5 | 5 | 20.0 | 0.1 |
| White | 22 | 51.2 | 0.8 | 13 | 81.3 | 0.5 | 29 | 67.4 | 1.0 | 75 | 44.1 | 2.6 | 16 | 64.0 | 0.5 |
| Other | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 3 | 1.8 | 12.2 | 0 | 0 | 0 |
| Unknown | 1 | 2.3 | | 0 | 0.0 | | 2 | 4.7 | | 13 | 7.6 | | 3 | 12.0 | |
| SPA | | | | | | | | | | | | | | | |
| 1 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 2.3 | 0.3 | 5 | 2.9 | 1.4 | 12 | 48.0 | 3.3 |
| 2 | 18 | 41.9 | 0.8 | 9 | 56.3 | 0.4 | 27 | 62.8 | 1.3 | 37 | 21.8 | 1.7 | 9 | 36.0 | 0.4 |
| 3 | 4 | 9.3 | 0.2 | 4 | 25.0 | 0.2 | 9 | 20.9 | 0.5 | 61 | 35.9 | 3.5 | 2 | 8.0 | 0.1 |
| 4 | 0 | 0.0 | 0.0 | 3 | 18.8 | 0.2 | 2 | 4.7 | 0.2 | 12 | 7.1 | 0.9 | 1 | 4.0 | 0.1 |
| 5 | 1 | 2.3 | 0.2 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 0.6 | 0.2 | 1 | 4.0 | 0.2 |
| 6 | 2 | 4.7 | 0.2 | 0 | 0.0 | 0.0 | 1 | 2.3 | 0.1 | 6 | 3.5 | 0.6 | 0 | 0 | 0 |
| 7 | 12 | 27.9 | 0.9 | 0 | 0.0 | 0.0 | 2 | 4.7 | 0.1 | 44 | 25.9 | 3.2 | 0 | 0 | 0 |
| 8 | 6 | 14.0 | 0.5 | 0 | 0.0 | 0.0 | 1 | 2.3 | 0.1 | 4 | 2.4 | 0.4 | 0 | 0 | 0 |
| Unknown | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0 | |

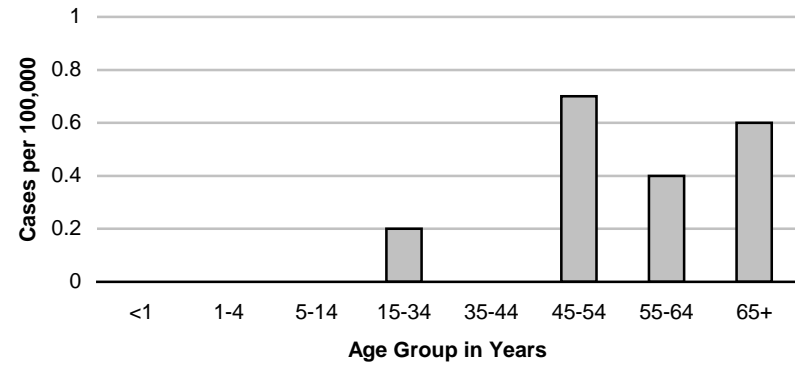
*Rates calculated based on less than 19 cases or events are considered unreliable.



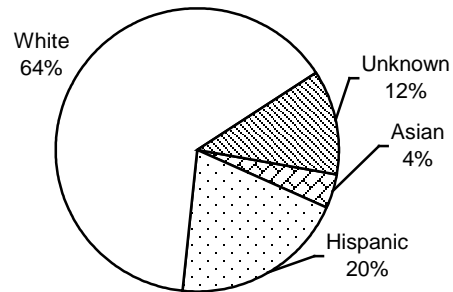
**Figure 1. Incidence Rates of West Nile Virus
LAC, 2004-2009**



**Figure 2. Incidence Rates of West Nile Virus by Age Group
LAC, 2009 (N=25)**

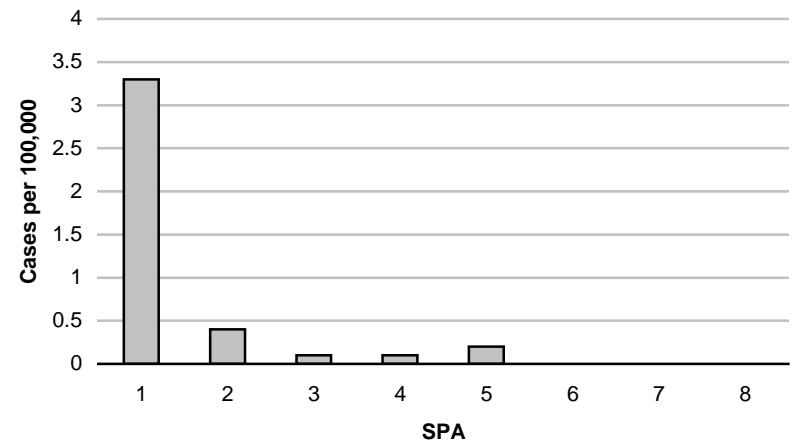


**Figure 3. Percent Cases of West Nile Virus by
Race/Ethnicity
LAC, 2009 (N=25)**



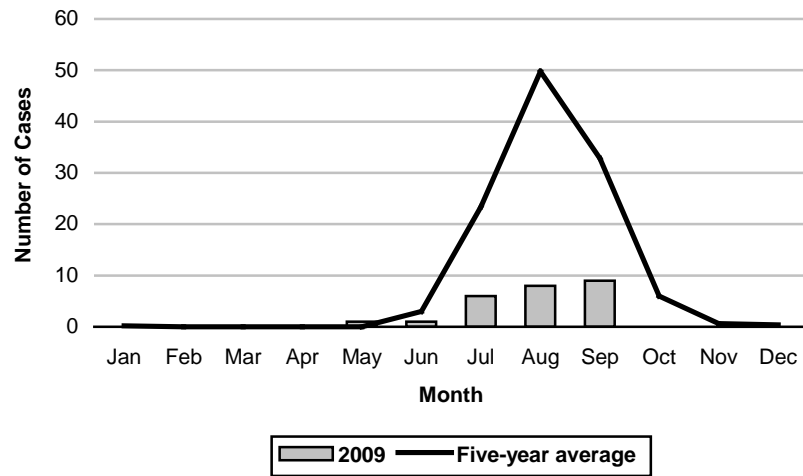
* Other includes Native American and any additional racial/ethnic group that cannot be categorized as Asian, black, Hispanic, or white.

**Figure 4. Incidence Rates of West Nile Virus by SPA
LAC, 2009 (N=25)**

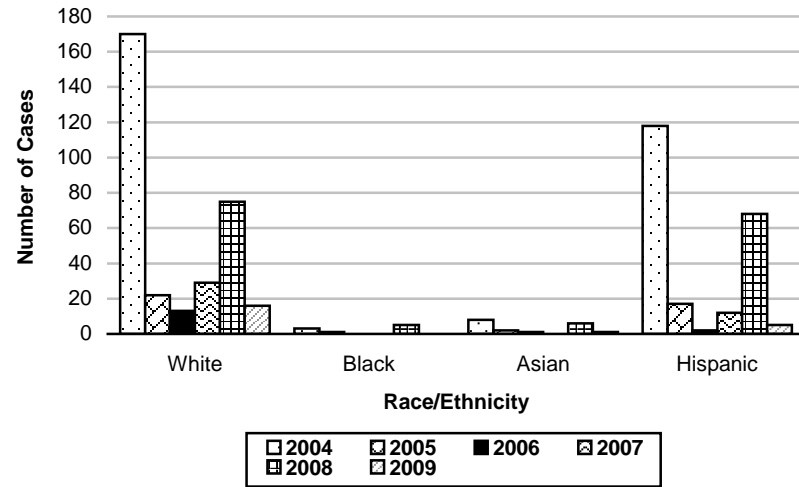




**Figure 5. Reported West Nile Virus Cases by Month of Onset
 LAC, 2009 (N=25)**



**Figure 6. West Nile Virus Incidence by Race/Ethnicity
 LAC, 2004-2009**





WEST NILE VIRUS

| CRUDE DATA | |
|-------------------------------|------|
| Number of Cases | 170 |
| Annual Incidence ^a | |
| LA County | 1.74 |
| California ^b | 1.22 |
| United States ^b | 0.45 |
| Age at Diagnosis | |
| Mean | 57 |
| Median | 59 |
| Range | 4-94 |

^aCases per 100,000 population.

^bCalculated from Final 2008 Reports of Nationally Notifiable Infectious Disease. MMWR 58(31);856-857;859-869.

DESCRIPTION

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

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

Most persons who become infected with WNV will not develop clinical illness or symptoms, which can be problematic in blood donation. Beginning 2003, blood donors were screened for WNV infection utilizing polymerase chain reaction (PCR) testing.

No transmission associated with blood products has been reported in LAC. Additional routes of transmission that have been documented include transplantation of WNV-infected organs, transplacental (mother-to-child), occupational exposures, and through breast milk.

Prevention and control of WNV and other arboviral diseases is most effective with vector management programs. These programs include surveillance for WNV activity in mosquito vectors, birds, horses, other animals, and humans; and implementation of appropriate mosquito control measures to reduce mosquito populations when necessary. When virus activity is detected in an area, residents are advised to increase measures to reduce contact with mosquitoes. Currently, there is no human vaccine available against WNV but several vaccines are under development. Important preventive measures against WNV include the following:

- Apply insect repellent to exposed skin. A higher percentage of DEET in a repellent will provide longer protection. DEET concentrations higher than 50% do not increase the length of protection.
- When possible, wear long-sleeved shirts and long pants when outdoors for long periods of time.
- Stay indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times.
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water. This will reduce the number of places mosquitoes can lay their eggs and breed.

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

DEET (N,N-diethyl-m-toluamide)
Picaridin (KBR 3023)
Oil of lemon eucalyptus.



2008 TRENDS AND HIGHLIGHTS

- The number of WNV infections reported in 2008 (n=170) increased almost four times from the previous year (n=43), including 22 asymptomatic donors and six deaths.
- WNV manifested as neuro-invasive disease in 105 reported infections (62%) including 54 meningitis, 49 encephalitis, and 2 AFP.
- As occurred in 2004, the highest incidence was reported from Service Planning Area (SPA) 3, the San Gabriel Valley (Figure 4).
- The WNV season has extended slightly with onset as early as June and as late as November, whereas in most previous years, onset was limited to July through October (Figure 5).



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

| | 2004 (N=309) | | | 2005 (N=43) | | | 2006 (N=16) | | | 2007 (N=43) | | | 2008 (N=170) | | |
|-----------------------|--------------|------|------------------|-------------|------|------------------|-------------|------|------------------|-------------|------|------------------|--------------|------|------------------|
| | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 | No. | (%) | Rate/ 100,000 |
| Age Group | | | | | | | | | | | | | | | |
| <1 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 |
| 1-4 | 0 | 0.0 | 0.0 | 1 | 2.3 | 0.2 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 0.6 | 0.2 |
| 5-14 | 10 | 3.2 | 0.7 | 1 | 2.3 | 0.1 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 |
| 15-34 | 32 | 10.4 | 1.1 | 7 | 16.3 | 0.2 | 2 | 12.5 | 0.1 | 3 | 7.0 | 0.1 | 19 | 11.2 | 0.7 |
| 35-44 | 46 | 14.9 | 3.1 | 4 | 9.3 | 0.3 | 5 | 31.3 | 0.3 | 0 | 0.0 | 0.0 | 15 | 8.8 | 1.0 |
| 45-54 | 70 | 22.7 | 5.7 | 8 | 18.6 | 0.6 | 3 | 18.8 | 0.2 | 9 | 20.9 | 0.7 | 34 | 20.0 | 2.5 |
| 55-64 | 59 | 19.1 | 7.4 | 8 | 18.6 | 1.0 | 3 | 18.8 | 0.3 | 12 | 27.9 | 1.4 | 36 | 21.2 | 3.9 |
| 65+ | 91 | 29.4 | 9.6 | 14 | 32.6 | 1.5 | 3 | 18.8 | 0.3 | 19 | 44.2 | 1.9 | 65 | 38.2 | 6.4 |
| Unknown | 1 | 0.3 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | |
| Race/Ethnicity | | | | | | | | | | | | | | | |
| Asian | 8 | 2.6 | 0.6 | 2 | 4.7 | 0.2 | 1 | 6.3 | 0.1 | 0 | 0.0 | 0.0 | 6 | 3.5 | 0.5 |
| Black | 3 | 1.0 | 0.4 | 1 | 2.3 | 0.1 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 5 | 2.9 | 0.6 |
| Hispanic | 118 | 38.2 | 2.6 | 17 | 39.5 | 0.4 | 2 | 12.5 | 0.0 | 12 | 27.9 | 0.3 | 68 | 40.0 | 1.5 |
| White | 170 | 55.0 | 5.8 | 22 | 51.2 | 0.8 | 13 | 81.3 | 0.5 | 29 | 67.4 | 1.0 | 75 | 44.1 | 2.6 |
| Other | 7 | 2.3 | 25.2 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 3 | 1.8 | 12.2 |
| Unknown | 3 | 1.0 | | 1 | 2.3 | | 0 | 0.0 | | 2 | 4.7 | | 13 | 7.6 | |
| SPA | | | | | | | | | | | | | | | |
| 1 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 2.3 | 0.3 | 5 | 2.9 | 1.4 |
| 2 | 79 | 25.6 | 3.7 | 18 | 41.9 | 0.8 | 9 | 56.3 | 0.4 | 27 | 62.8 | 1.3 | 37 | 21.8 | 1.7 |
| 3 | 113 | 36.6 | 6.6 | 4 | 9.3 | 0.2 | 4 | 25.0 | 0.2 | 9 | 20.9 | 0.5 | 61 | 35.9 | 3.5 |
| 4 | 14 | 4.5 | 1.1 | 0 | 0.0 | 0.0 | 3 | 18.8 | 0.2 | 2 | 4.7 | 0.2 | 12 | 7.1 | 0.9 |
| 5 | 2 | 0.6 | 0.3 | 1 | 2.3 | 0.2 | 0 | 0.0 | 0.0 | 0 | 0.0 | 0.0 | 1 | 0.6 | 0.2 |
| 6 | 8 | 2.6 | 0.8 | 2 | 4.7 | 0.2 | 0 | 0.0 | 0.0 | 1 | 2.3 | 0.1 | 6 | 3.5 | 0.6 |
| 7 | 74 | 23.9 | 5.4 | 12 | 27.9 | 0.9 | 0 | 0.0 | 0.0 | 2 | 4.7 | 0.1 | 44 | 25.9 | 3.2 |
| 8 | 5 | 1.6 | 0.5 | 6 | 14.0 | 0.5 | 0 | 0.0 | 0.0 | 1 | 2.3 | 0.1 | 4 | 2.4 | 0.4 |
| Unknown | 14 | 4.5 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | | 0 | 0.0 | |

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



Figure 1. Incidence Rates of West Nile Virus LAC, 2004-2008

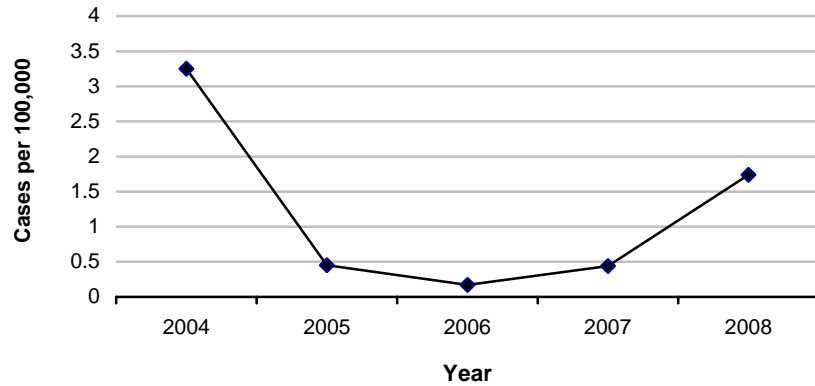


Figure 2. Incidence Rates of West Nile Virus by Age Group LAC, 2008

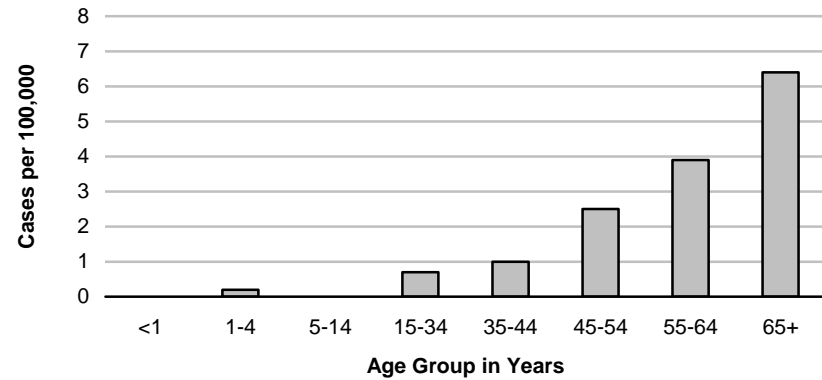
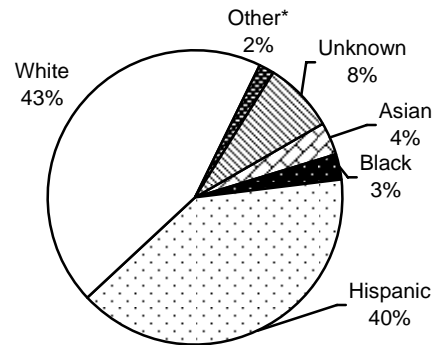


Figure 3. Percent Cases of West Nile Virus by Race/Ethnicity LAC, 2008



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

Figure 4. Incidence Rates of West Nile Virus by SPA LAC, 2008

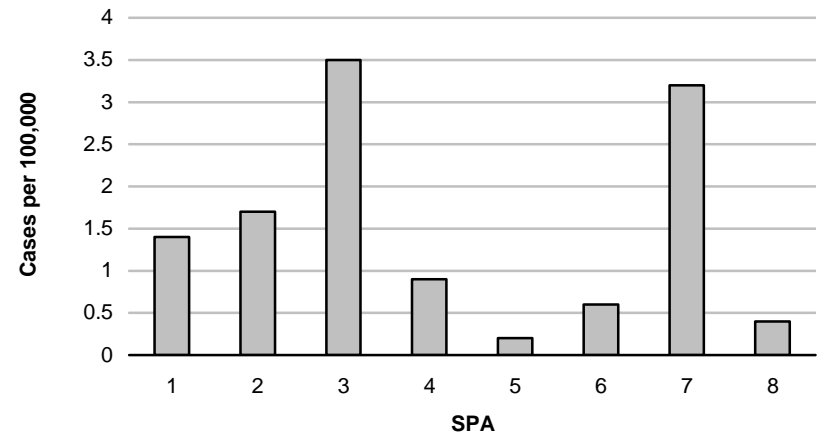




Figure 5. Reported West Nile Virus Cases by Month of Onset LAC, 2008

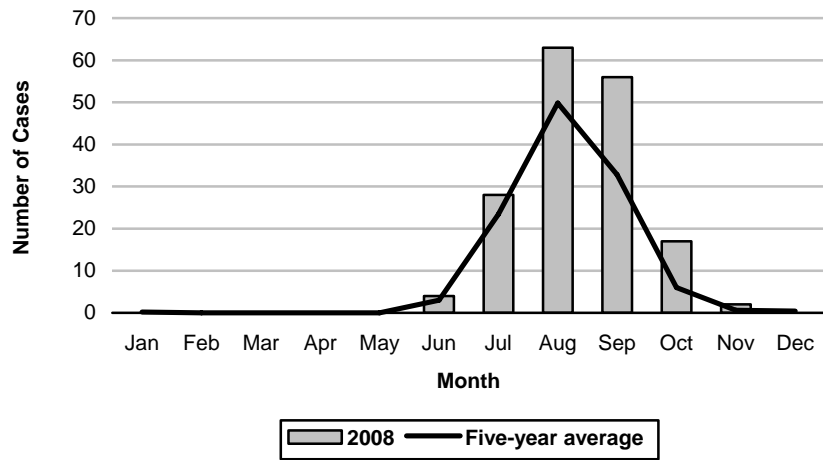
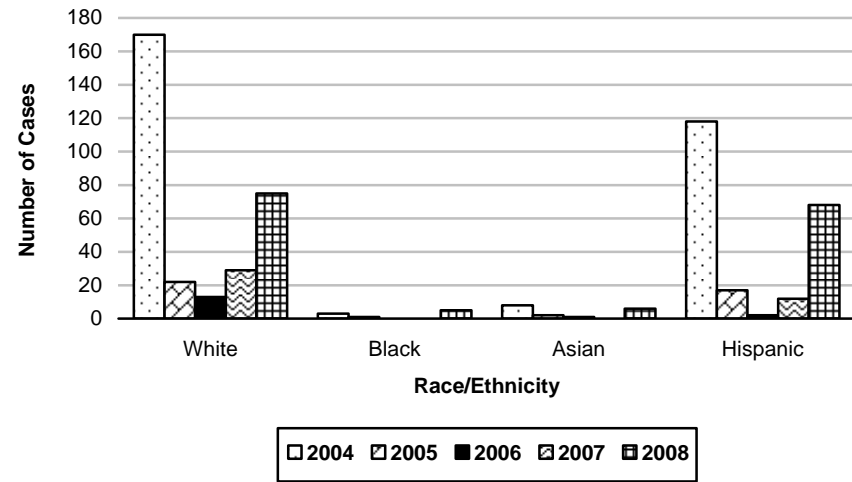


Figure 6. West Nile Virus Incidence by Race/Ethnicity LAC, 2004-2008



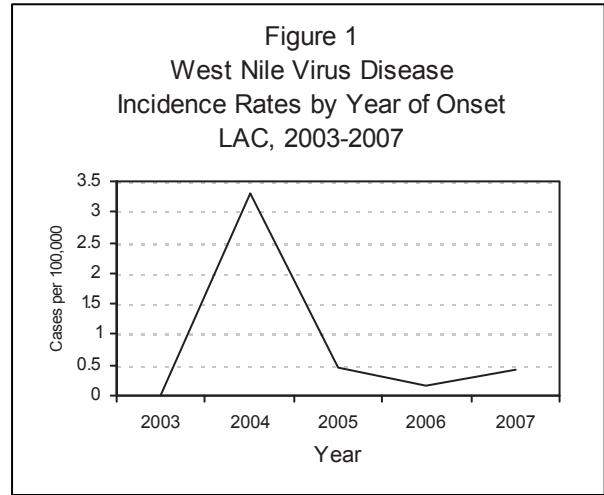


WEST NILE VIRUS

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

^a Cases per 100,000 population.

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



DESCRIPTION

Life Cycle and Epidemiology

West Nile virus (WNV) is a single-stranded RNA virus placed within the family Flaviviridae, genus Flavivirus. Within the genus Flavivirus, WNV has been serologically classified within the Japanese encephalitis (JE) virus antigenic complex, which includes the human pathogens JE, Murray Valley encephalitis, Saint Louis encephalitis (SLE), and Kunjin viruses.

WNV was indigenous to Africa, Asia, Europe, and Australia, and was introduced to North America in 1999, when it was first detected in New York City. The likely origin of the introduced strain was the Middle East, but the mode of introduction remains unknown. Since 1999, human and non-human WNV surveillance data has documented that WNV has extended its range through most of the continental United States as well as to Canada and Mexico.

The life cycle of the virus involves the transmission of the virus between mosquitoes and bird reservoir hosts. Humans are incidentally infected when bitten by an infected mosquito, usually a *Culex* or *Anopheles* species. The incubation period for human infection is 2 to 14 days. Birds, especially corvids such as the North American crow, are the optimal hosts for harboring and replicating the virus. Mosquitoes become infected when they feed on infected birds, which may circulate high level of viremia for several days. Infectious mosquitoes carry virus particles in their salivary glands and infect susceptible bird species during blood-meal feeding. Bird reservoirs will sustain an infectious viremia for 1 to 4 days.

In 2002, evidence of WNV transmission was shown to occur via the transfer of all blood product components including platelets, packed red blood cells, and plasma. Beginning 2003, blood donors were screened for WNV infection utilizing polymerase chain reaction (PCR) testing. Millions of units of blood were screened for WNV utilizing PCR based technology, testing donor mini-pools. Though asymptomatic donors have been identified as positive for WNV in LAC, no transmission associated with blood products has been reported. Additional routes of transmission that have been documented include transplantation of WNV-infected organs, transplacental (mother-to-child), occupational exposures, and through breast milk.



Clinical Infection and Diagnosis

Most persons who become infected with WNV will not develop clinical illness or symptoms. About 20% of persons infected will develop WNV fever with symptoms that include fever, headache, rash, muscle weakness, fatigue, nausea and vomiting, and occasionally lymph node swelling. Approximately one in 150 patients will develop more severe illness, manifesting as WNV neuro-invasive disease (NID). WNV NID includes encephalitis, meningitis, and acute flaccid paralysis (AFP). WNV-associated encephalitis is commonly associated with fever, altered mental status, headache, and seizures; WNV encephalitis usually necessitates high levels of specialized medical care. Focal neurologic deficits, including limb paralysis, cranial nerve palsies, Parkinsonian-like tremors, and other movement disorders have been observed. WNV-associated meningitis usually involves fever, headache, and stiff neck, and has a good prognosis.

DISEASE ABSTRACT

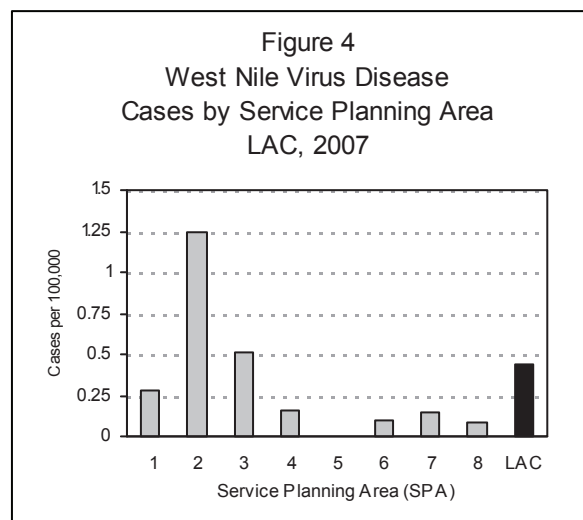
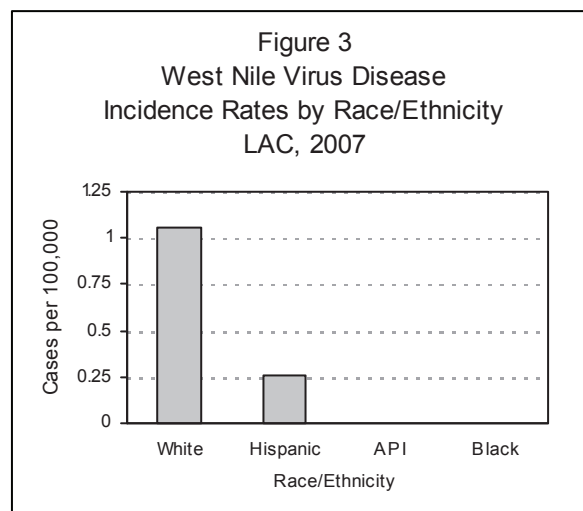
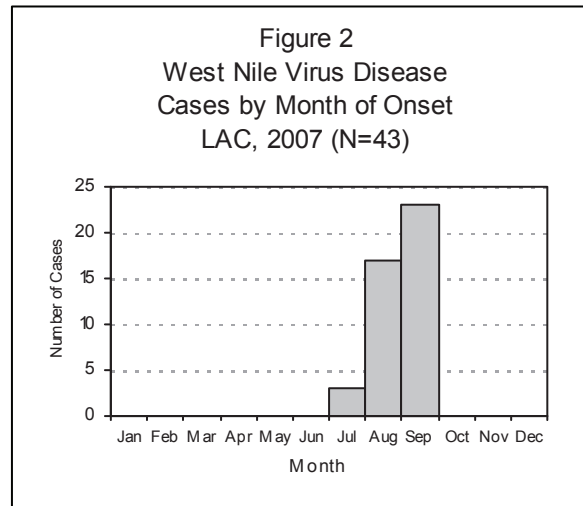
- The overall incidence of reported WNV infections in 2007 was 0.44 cases per 100,000 population, rising from a low of 0.17 in 2006 when only 16 cases were confirmed (Figure 1).
- Case fatalities (n=5) occurred for the first time since 2004.
- Meningitis continues to be the most commonly reported clinical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

STRATIFIED DATA

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

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

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





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

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

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

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

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

COMMENTS

The first symptomatic WNV case in LAC associated with environmental evidence was documented in 2003. In 2004, an outbreak of 309 WNV infections, including asymptomatic blood donors, with 14 deaths were reported in LAC — the most of any CA jurisdiction. In response to the outbreak, LAC DPH added WNV infection to its list of reportable diseases by authority of the Health Officer under California Code of Regulations, Title 17, Sections 2511 and 2505. Physicians and laboratories are required to report all positive laboratory findings of WNV tests to the DPH within one working day.

The following years presented a markedly different picture, with numbers declining to a low of 16 in 2006. This year, however, over twice as many cases were reported. The rise in cases, as well as the continued detection of positive mosquito pools, dead birds and other reservoir animals, has demonstrated that WNV remains endemic in the LAC and southern CA region. As the number of cases has fluctuated greatly from year to year (ranging from 16 to 43 since 2005), the baseline level of cases expected for this region remains to be seen. Sustained surveillance of humans, as well as other animals, will be required in the coming years to help guide public health officials in providing targeted health education to communities at particularly high risk.

PREVENTION

Prevention and control of WNV and other arboviral diseases is most effectively accomplished through integrated vector management programs. These programs include surveillance for WNV activity in mosquito vectors, birds, horses, other animals, and humans; and implementation of appropriate mosquito control measures to reduce mosquito populations when necessary. Additionally, when virus activity is detected in an area, residents are alerted and advised to increase measures to reduce contact with mosquitoes. Currently, there is no human vaccine available against WNV but several vaccines are under development. Important preventive measures against WNV include the following:

- Apply insect repellent to exposed skin. A higher percentage of DEET in a repellent will provide longer protection. DEET concentrations higher than 50% do not increase the length of protection.
- When possible, wear long-sleeved shirts and long pants when outdoors for long periods of time.
- Stay indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times.
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water. This will reduce the number of places mosquitoes can lay their eggs and breed.



A wide variety of insect repellent products are available. CDC recommends the use of products containing active ingredients which have been registered with the U.S. Environmental Protection Agency (EPA) for use as repellents applied to skin and clothing. EPA registration of repellent active ingredients indicates the materials have been reviewed and approved for efficacy and human safety when applied according to the instructions on the label. Of the active ingredients registered with the EPA, three have demonstrated a higher degree of efficacy in the peer-reviewed, scientific literature. Products containing these active ingredients typically provide longer-lasting protection than others:

DEET (N,N-diethyl-m-toluamide)
Picaridin (KBR 3023)
Oil of lemon eucalyptus

Oil of lemon eucalyptus [p.menthane 3, 8-diol (PMD)], a plant based repellent, is registered with EPA. In two recent scientific publications, when oil of lemon eucalyptus was tested against mosquitoes found in the US it provided protection similar to repellents with low concentrations of DEET.

VECTOR CONTROL

There are five local mosquito and vector control districts within LAC that provide mosquito abatement services to all areas of the county. They carry out mosquito and sentinel chicken surveillance, provide public information, and are critical to mosquito-borne disease control. They include:

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

These five local mosquito and vector control districts work closely with the ACDC to investigate confirmed and presumptive human cases of locally acquired mosquito-borne disease to identify mosquito breeding sites and to put into place appropriate control measures.

ADDITIONAL RESOURCES

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

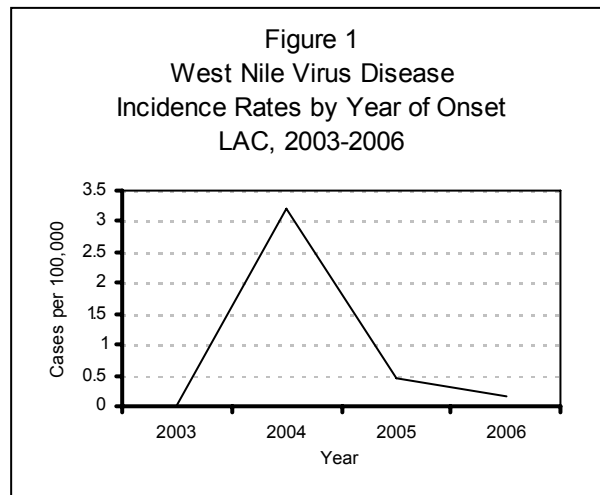
Mosquito and Vector Control District Websites:

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

WEST NILE VIRUS

| CRUDE DATA | |
|----------------------------|-------------|
| Number of Cases | 16 |
| Incidence LAC ^a | |
| LA County | 0.17 |
| California | N/A |
| United States | N/A |
| Age at Diagnosis | |
| Mean | 50.9 |
| Median | 50.5 |
| Range | 28–82 years |

^a Cases per 100,000 population.



DESCRIPTION

Life Cycle and Epidemiology

West Nile virus (WNV) is a single-stranded RNA virus placed within the family Flaviviridae, genus Flavivirus. Within the genus Flavivirus, WNV has been serologically classified within the Japanese encephalitis (JE) virus antigenic complex, which includes the human pathogens JE, Murray Valley encephalitis, Saint Louis encephalitis (SLE), and Kunjin viruses.

WNV was indigenous to Africa, Asia, Europe, and Australia, and was introduced to North America in 1999, when it was first detected in New York City. The likely origin of the introduced strain was the Middle East, but the mode of introduction remains unknown. Since 1999, human and non-human WNV surveillance data has documented that WNV has extended its range through most of the continental United States as well as to Canada and Mexico.

The life cycle of the virus involves the transmission of the virus between mosquitoes and bird reservoir hosts. Humans are incidentally infected when bitten by an infected mosquito, usually a *Culex* or *Anopheles* species. The incubation period for human infection is 2 to 14 days. Birds, especially corvids such as the North American crow, are the optimal hosts for harboring and replicating the virus. Mosquitoes become infected when they feed on infected birds, which may circulate high level of viremia for several days. Infectious mosquitoes carry virus particles in their salivary glands and infect susceptible bird species during blood-meal feeding. Bird reservoirs will sustain an infectious viremia for 1 to 4 days. Additional routes of transmission that have been documented include transplantation of WNV-infected organs, blood transfusions, transplacental (mother-to-child), occupational exposures, and through breast milk.

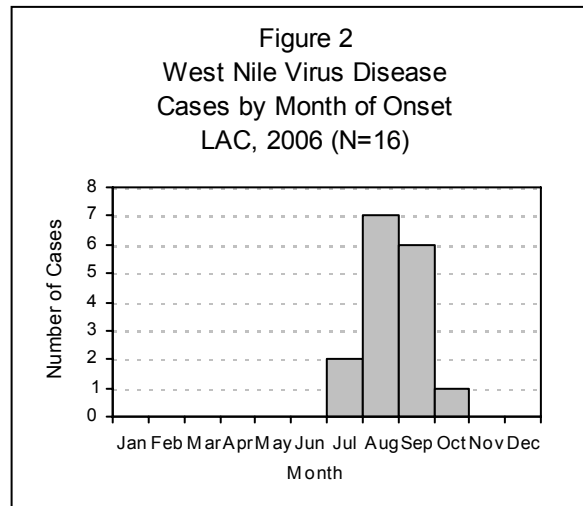
Clinical Infection and Diagnosis

Most persons who become infected with WNV will not develop clinical illness or symptoms. Approximately one in 150 patients will develop more severe illness, manifesting as WNV neuro-invasive disease (NID), and about 20% of persons infected will develop WNV fever with symptoms that include fever, headache, rash, muscle weakness, fatigue, nausea and vomiting, and occasionally lymph node swelling. WNV NID includes encephalitis, meningitis, and acute flaccid paralysis (AFP). WNV-associated encephalitis is commonly associated with the following symptoms: fever, altered mental status, headache, and seizures; WNV encephalitis usually necessitates high levels of specialized medical care. Focal

neurologic deficits, including limb paralysis, cranial nerve palsies, Parkinsonian-like tremors, and other movement disorders have been observed. WNV-associated meningitis usually involves fever, headache, and stiff neck, and has a good prognosis.

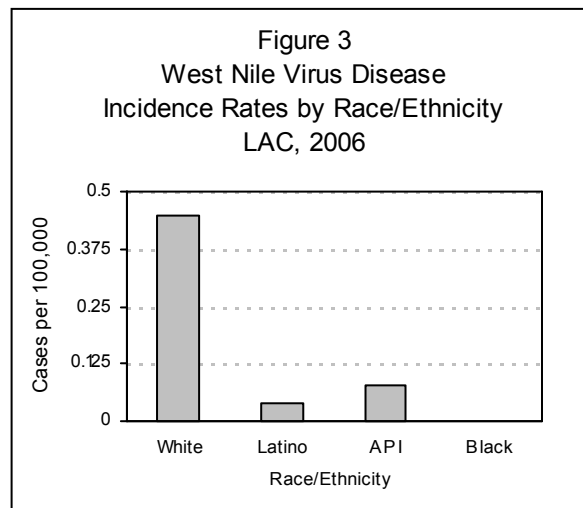
DISEASE ABSTRACT

- The overall incidence of reported WNV infections in 2006 was 0.17 cases per 100,000 population, far lower than the incidence rates of previous years, when 3.2 per 100,000 and 0.46 per 100,000 were confirmed in 2004 and 2005, respectively (Figure 1).
- There were no case fatalities in 2005 or 2006.
- Meningitis was the most commonly reported clinical condition as it was in 2005, comprising 25% (n=4) of cases. In 2005, meningitis comprised 34.8% of cases (n=15).
- There were few or no cases in children in both 2005 and 2006.
- Most WNV infections occurred in persons residing in San Fernando Valley.



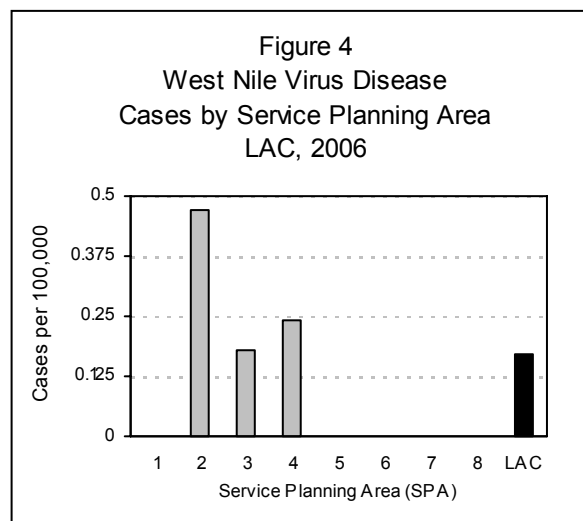
STRATIFIED DATA

Trends: WNV infection, including in asymptomatic blood donors, occurred at an incidence rate of 0.17 per 100,000 population in 2006. Both the total number and incidence of WNV infection decreased dramatically since 2004 when 309 cases were confirmed at an incidence of 3.2 cases per 100,000 population. In 2005, the incidence was 0.46 per 100,000 (n=43) (Figure 1).



Seasonality: Onset of cases occurred July through October and peaked in August (Figure 2). A similar epidemiologic symptom onset curve also occurred in 2005.

Age: The median age was 50.5 years (range: 28–82 years). For age groups ≥35 years, the incidence rates were similar (they ranged 0.2-0.4 cases per 100,000). There was more varied distribution in 2005 where incidence rates ranged from 0.3 cases per 100,000 among children under 10 to 11.6 cases per 100,000 in those greater than 80 years old.



Sex: A higher proportion of male WNV cases were reported than female cases. The incidence rates were 0.25 cases and 0.08 cases per 100,000, respectively.

Race/Ethnicity: Whites had the greatest proportion of reported cases (81%) as well as the highest incidence rates of infection (n=13, 0.45 per 100,000). Latinos accounted for 13% of cases (n=2, 0.04 per 100,000), and only 6% of reported cases occurred among Asian Pacific Islanders (n=1, 0.1 per 100,000). No cases in

blacks were reported (Figure 3).

Location: The greatest number of reported WNV cases were reported from SPA 2 (n=10, 0.47 per 100,000). WNV cases occurred in only two other areas: SPAs 3 and 4. WNV was distributed more widely in 2005, though SPA 2 also accounted for most cases.

PREVENTION

Prevention and control of WNV and other arboviral diseases is most effectively accomplished through integrated vector management programs. These programs include surveillance for WNV activity in mosquito vectors, birds, horses, other animals, and humans; and implementation of appropriate mosquito control measures to reduce mosquito populations when necessary. Additionally, when virus activity is detected in an area, residents are alerted and advised to increase measures to reduce contact with mosquitoes. Currently, there is no human vaccine available against WNV but several vaccines are under development. Important preventive measures against WNV include the following:

- Apply insect repellent to exposed skin. A higher percentage of DEET in a repellent will provide longer protection. DEET concentrations higher than 50% do not increase the length of protection.
- When possible, wear long-sleeved shirts and long pants when outdoors for long periods of time.
- Stay indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times.
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water. This will reduce the number of places mosquitoes can lay their eggs and breed.

A wide variety of insect repellent products are available. CDC recommends the use of products containing active ingredients which have been registered with the U.S. Environmental Protection Agency (EPA) for use as repellents applied to skin and clothing. EPA registration of repellent active ingredients indicates the materials have been reviewed and approved for efficacy and human safety when applied according to the instructions on the label. Of the active ingredients registered with the EPA, three have demonstrated a higher degree of efficacy in the peer-reviewed, scientific literature. Products containing these active ingredients typically provide longer-lasting protection than others:

- DEET (N,N-diethyl-m-toluamide)
- Picaridin (KBR 3023)
- Oil of lemon eucalyptus

Oil of lemon eucalyptus [p.menthane 3, 8-diol (PMD)], a plant based repellent, is registered with EPA. In two recent scientific publications, when oil of lemon eucalyptus was tested against mosquitoes found in the US it provided protection similar to repellents with low concentrations of DEET.

In 2002, evidence of WNV transmission was shown to occur via the transfer of all blood product components including platelets, packed red blood cells, and plasma. Beginning 2003, blood donors were screened for WNV infection utilizing polymerase chain reaction (PCR) testing. Millions of units of blood were screened for WNV utilizing PCR based technology, testing donor mini-pools. Though asymptomatic donors have been identified as positive for WNV in LAC, no transmission associated with blood products has been reported.

COMMENTS

The first symptomatic WNV case in LAC with associated environmental evidence was documented in 2003. In 2004, an outbreak of 309 WNV infections, including asymptomatic blood donors, with 14 deaths were reported in LAC — the most of any CA jurisdiction. The following years have presented a markedly different picture. In 2005, the county only documented 43 infections and no deaths. The decline continued in 2006, during which only 16 cases and no deaths were reported.

In response to the 2004 WNV outbreak, LAC DPH specifically added WNV infection to its list of reportable diseases by authority of the Health Officer under California Code of Regulations, Title 17, Sections 2503 and 2505. Physicians and laboratories are required to report all positive laboratory findings of WNV to the DPH within one working day. Continued vector surveillance efforts have demonstrated that, despite the decline in incidence in LAC, WNV remains endemic (enzootic) in the LAC and southern CA region. Sustained surveillance of humans, as well as other animals, will be required in the coming years to help guide public health officials in providing targeted health education to communities at particularly high risk.

VECTOR CONTROL

There are five local mosquito and vector control districts within LAC that provide mosquito abatement services to all areas of the county. They carry out mosquito and sentinel chicken surveillance, provide public information, and are critical to mosquito-borne disease control. They include:

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- Compton Creek Mosquito Abatement District

These five local mosquito and vector control districts work closely with the ACDC to investigate confirmed and presumptive human cases of locally acquired mosquito-borne disease to identify mosquito breeding sites and to put into place appropriate control measures.

ADDITIONAL RESOURCES

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

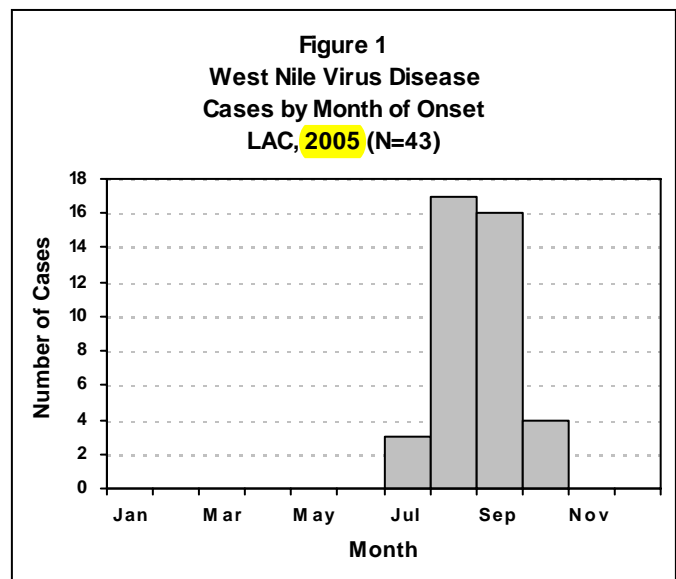
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- Antelope Valley Mosquito and Vector Control District: www.avmosquito.org
- Mosquito and Vector Control Association of California: www.mvacac.org

WEST NILE VIRUS

| CRUDE DATA | |
|----------------------------|------------|
| Number of Cases | 43 |
| Incidence LAC ^e | |
| LA County | 0.46 |
| California | 2.6 |
| United States | 1.1 |
| Age at Diagnosis | |
| Mean | 52 |
| Median | 56 |
| Range | 4-89 years |
| Case Fatality | 0 |
| LA County | 0% |
| California | 2% |
| United States | 4% |

^a Cases per 100,000 population and based on 2005 population estimates.



DESCRIPTION

LIFE CYCLE AND EPIDEMIOLOGY

West Nile virus (WNV) is a single-stranded RNA virus placed within the family Flaviviridae, genus Flavivirus. Within the genus Flavivirus, WNV has been serologically classified within the Japanese encephalitis (JE) virus antigenic complex, which includes the human pathogens JE, Murray Valley encephalitis, Saint Louis encephalitis (SLE), and Kunjin viruses.

WNV was indigenous to Africa, Asia, Europe, and Australia, and was introduced to North America in 1999, where it was first detected in New York City. The likely origin of the introduced strain was the Middle East, but the mode of introduction remains unknown. From 1999 through 2004, human and non-human WNV surveillance data has documented that WNV has extended its range through most of continental United States to include 47 states and the District of Columbia, Canada, and Mexico. In 2005, 3000 confirmed human WNV cases were reported nationally to the Centers for Disease Control and Prevention (CDC). California (CA) reported the greatest number of any state, 935 cases; Los Angeles County (LAC) reported 43 human cases.

The life cycle of the virus involves the transmission of the virus from a bird reservoir to humans via *Culex*, or *Anopheles* mosquitoes. Birds, especially, corvids such as the North American crow, are the optimal hosts for harboring and replicating the virus. Mosquitoes become infected when they feed on infected birds, which may circulate high level of viremia for several days. Infectious mosquitoes carry virus particles in their salivary glands and infect susceptible bird species during blood-meal feeding. Bird reservoirs will sustain an infectious viremia for 1 to 4 days. Additional routes of transmission that have been documented include transplantation of WNV-infected organs, blood transfusions, transplacental (mother-to-child), occupational exposures, and through breast milk.

CLINICAL INFECTION AND DIAGNOSIS

Most persons who become infected with WNV will not develop clinical illness or symptoms. Approximately one in 150 people will develop more severe illness, manifesting as WNV neuro-invasive disease (NID), and about 20% of persons infected will develop WNV fever with symptoms that include fever, headache, rash, muscle weakness, fatigue, nausea and vomiting, and occasionally lymph node swelling. WNV NID includes encephalitis, meningitis, and acute flaccid paralysis (AFP). WNV-associated encephalitis is commonly associated with the following symptoms: fever, altered mental status, headache, seizures, and usually necessitates high levels of specialized medical care. Focal neurologic deficits, including limb paralysis, cranial nerve palsies, Parkinsonian-like tremors, and movement disorders have also been observed. WNV-associated meningitis usually involves fever, headache, and stiff neck, and has an excellent prognosis.

DISEASE ABSTRACT

- The overall incidence of reported WNV infections including asymptomatic blood donors in 2005 was 0.46 cases per 100,000 population, far lower than the 2004 incidence of 3.2 cases per 100,000 (Figure 1).
- There were no case fatalities in 2005.
- WNV meningitis was the most commonly reported clinical condition, comprising 34.8% (15) of cases in 2005.
- The lowest incidence rates were found in children; there was a consistent increase in incidence after age 40 years.
- Non-Hispanic Whites had the highest proportion of symptomatic infections (49%, n=21) followed by Hispanics (40%, n=17) (Figure 2).
- Most WNV infections occurred in persons residing in San Fernando Valley.

STRATIFIED DATA

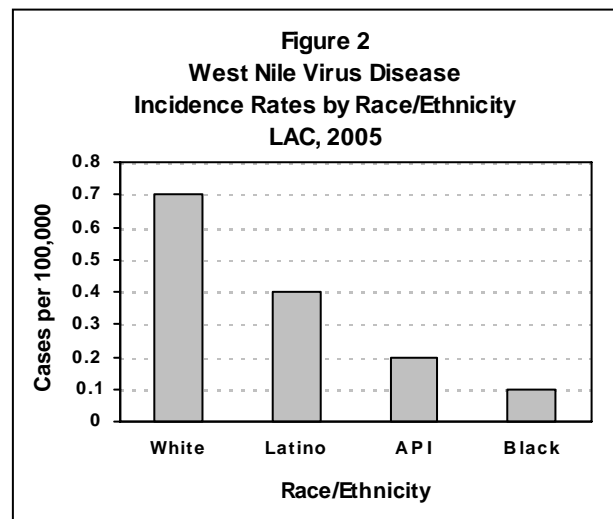
Trends: In 2003, the first WNV case with associated environmental evidence was documented in LAC. In 2004, 209 cases were document in LAC, the most of any CA jurisdiction. In 2005, both the total number of WNV cases and the incidence of WNV infection decreased dramatically with 43 cases reported, an incidence of 0.46 cases per 100,000 population.

Seasonality: Cases were reported from the beginning of summer (first week of July) through fall (last week of October). The peak onset of cases was the 2nd week in August (33 week) when 9 cases were reported (Figure 1).

Age: The median age for all WNV cases including asymptomatic blood donors was 43 years old (range: 18-57 years). The median age for both WNF and WNV meningitis was 47 years. The median age for the 13 encephalitis cases was 63 years old. The lowest incidence rates of WNV infection occurred among children under 10, 0.3 cases per 100,000, whereas the greatest incidence occurred in those > 80 years (11.6 cases per 100,000).

Sex: A higher proportion of male WNV cases were reported than female cases. The incidence rates were 0.5 cases and 0.4 cases per 100,000, respectively.

Race/Ethnicity: Whites had the both the greatest proportion of reported cases and highest incidence rates of infection, 49% of cases (n=21, 0.7 per 100,000), followed by Latinos, 40% of cases (n= 17, 0.4 per 100,000). Only 5% of reported cases occurred among Asian Pacific Islanders (n=2, 0.2 per 100,000) and only 1 case (1%) was reported in an African-American (0.1 per 100,000) (Figure 2).



Location: The greatest number of reported WNV cases were reported from SPA 2 (n=19, 0.9 per 100,000). Whereas, SPA 7 (n=12, 4.0 per 100,000) had the second largest number of reported cases but had the highest incidence WNV infection (Figure 3) of any of the service planning areas.

PREVENTION

Prevention and control of WNV and other arboviral diseases is most effectively accomplished through integrated vector management programs. These programs include surveillance for WNV activity in mosquito vectors, birds, horses, other animals, and humans, and implementation of appropriate mosquito control measures to reduce mosquito populations when necessary. Additionally, when virus activity is detected in an area, residents are alerted and advised to increase measures to reduce contact with mosquitoes. Currently, there is no human vaccine available against WNV but several vaccines are under development. Important preventive measures against WNV include the following:

- Apply insect repellent to exposed skin. A higher percentage of DEET in a repellent will provide longer protection. DEET concentrations higher than 50% do not increase the length of protection.
- When possible, wear long-sleeved shirts and long pants when outdoors for long periods of time.
- Stay indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times.
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water. This will reduce the number of places mosquitoes can lay their eggs and breed.

A wide variety of insect repellent products are available. CDC recommends the use of products containing active ingredients which have been registered with the U.S. Environmental Protection Agency (EPA) for use as repellents applied to skin and clothing. EPA registration of repellent active ingredients indicates the materials have been reviewed and approved for efficacy and human safety when applied according to the instructions on the label. Of the active ingredients registered with the EPA, three have demonstrated a higher degree of efficacy in the peer-reviewed, scientific literature. Products containing these active ingredients typically provide longer-lasting protection than others:

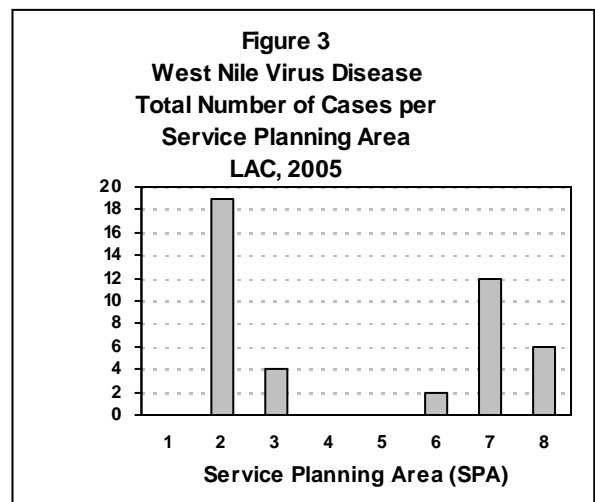
- DEET (N,N-diethyl-m-toluamide)
- Picaridin (KBR 3023)
- Oil of lemon eucalyptus

Oil of lemon eucalyptus [p.menthane 3, 8-diol (PMD)], a plant based repellent, is also registered with EPA. In two recent scientific publications, when oil of lemon eucalyptus was tested against mosquitoes found in the US it provided protection similar to repellents with low concentrations of DEET.

In 2002, WNV transmission was documented from all components of blood products including platelets, packed red blood cells, and plasma. Subsequently in 2003, all blood donors were screened for WNV infection utilizing polymerase chain reaction (PCR) testing. Millions of units of blood were screened for WNV utilizing PCR based technology, testing donor mini-pools. In 2005, WNV screening continued and no transmission associated with blood products were reported.

COMMENTS

In 2005, 43 human WNV cases were confirmed in LAC among hundreds of tested patients. WNV is considered endemic (enzootic) to LAC and Southern California. Sustained surveillance will be required in



the coming years, including surveillance among humans, dead birds, mosquito pools, and sentinel chickens. These activities guide public health officials in providing targeted health education to communities at particularly high risk. In response to the 2004 WNV outbreak, LAC DHS specifically added WNV infection to its list of reportable diseases by authority of the Health Officer under California Code of Regulations, Title 17, Sections 2503 and 2505. Physicians and laboratories are required to report all positive laboratory findings of WNV to the Department of Health Services within one (1) working day.

VECTOR CONTROL

There are five local mosquito and vector control districts within LAC that provide mosquito abatement services to all areas of the county. They carry out mosquito and sentinel chicken surveillance, provide public information, and are critical to mosquito-borne disease control. They include:

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These five local mosquito and vector control districts work closely with the ACDC to investigate confirmed and presumptive human cases of locally acquired mosquito-borne disease to identify mosquito breeding sites and to put into place appropriate control measures.

REFERENCES:

1. Campbell GL, Marfin AA, Lanciotti RS, Gubler DJ. West Nile virus. *Lancet Infect Dis* 2002 ;519-29.
2. Watson JT, Pertel PE, Jones RC, et al. Clinical characteristics and functional outcomes of West Nile fever. *Ann Intern Med.* 2004;141:360-365.
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6. Nash D, Mostasharia F, Fine A, Miller M, et al. The outbreak of West Nile virus infection in the New York City area in 1999. *N Eng J Med* 2001; 344:24: 1807-1814.

ADDITIONAL RESOURCES

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

Mosquito and Vector Control District Websites:

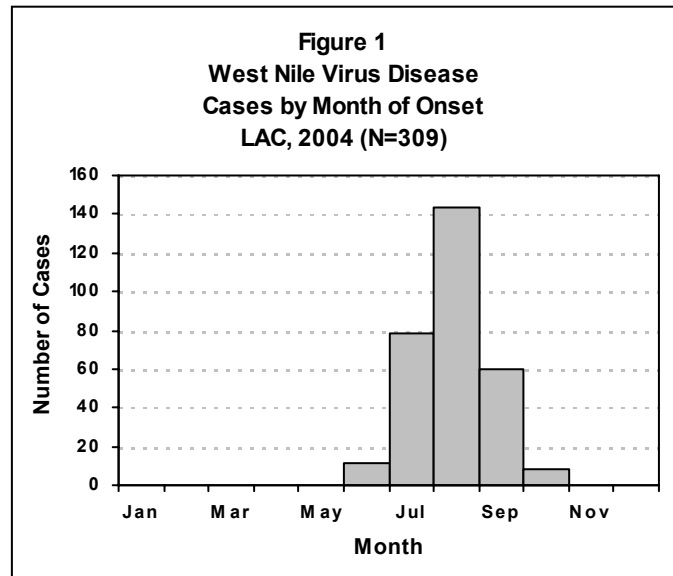
- Greater Los Angeles County Vector Control District: www.glacvcd.org/
- West Los Angeles Vector Control District: www.lawestvector.org/
- San Gabriel Valley Mosquito and Vector Control District: www.sgvmosquito.org/
- Antelope Valley Mosquito and Vector Control District: www.avmosquito.org/
- Mosquito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



WEST NILE VIRUS

| CRUDE DATA | |
|----------------------------|-------------|
| Number of Cases | 309 |
| Incidence LAC ^a | |
| LA County | 3.2 |
| California | 0.81 |
| United States | 0.39 |
| Age at Diagnosis | |
| Mean | 54 |
| Median | 54 |
| Range | <5–94 years |
| Case Fatality | 14 |
| LA County | 4.5% |
| California | 3.6% |
| United States | 3.9% |

^a Cases per 100,000 population and based on 2004 population estimates.



DESCRIPTION

LIFE CYCLE AND EPIDEMIOLOGY

West Nile virus (WNV) is a single-stranded RNA virus placed within the family Flaviviridae, genus Flavivirus. Within the genus Flavivirus, WNV has been serologically classified within the Japanese encephalitis (JE) virus antigenic complex, which includes the human pathogens JE, Murray Valley encephalitis, Saint Louis encephalitis (SLE), and Kunjin viruses.

WNV is indigenous to Africa, Asia, Europe, Middle East, and Australia, and was introduced to North America in 1999, where it was first detected in New York City. The likely origin of the introduced strain was the Middle East, but the mode of introduction remains unknown. From 1999 through 2004, human and non-human WNV surveillance data has documented that WNV has extended its range through most of continental United States to include 47 states and the District of Columbia, Canada, and Mexico. In 2004, 2539 confirmed human WNV cases were reported nationally to the Centers for Disease Control and Prevention (CDC). California (CA) reported the greatest number of any state, 779 cases; Los Angeles County (LAC) reported 309 human cases.

The life cycle of the virus involves the transmission of the virus from a bird reservoir to humans via *Culex*, or Anopheles mosquitoes. Birds, especially, corvids such as the *North American crow*, are the optimal hosts for harboring and replicating the virus. Mosquitoes become infected when they feed on infected birds, which may circulate high level of viremia for several days. Infectious mosquitoes carry virus particles in their salivary glands and infect susceptible bird species during blood-meal feeding. Bird reservoirs will sustain an infectious viremia for 1 to 4 days. Additional less frequent routes of transmission that have been documented include transplantation of WNV-infected organs, blood transfusions, transplacental (mother-to-child), occupational exposures, and through breast milk.



CLINICAL INFECTION AND DIAGNOSIS

Most persons who become infected with West Nile virus (WNV) will not develop clinical illness or symptoms. The incubation period for WNV infection can range from 2 to 14 days, although longer incubation periods have been documented in immunosuppressed persons. Approximately one in 150 people will develop severe illness, WNV neuro-invasive disease (NID), and about 20% of persons infected with WNV will develop WNV fever (WNF) with symptoms that can include fever, headache, rash, muscle weakness, fatigue, nausea and vomiting, and occasionally lymph node swelling. WNV NID includes: encephalitis, meningitis, and acute flaccid paralysis (AFP). WNV-associated encephalitis, the most severe form of NID, is commonly associated with the following symptoms: fever, altered mental status, headache, seizures, and usually necessitates high levels of specialized medical care. Focal neurologic deficits, including limb paralysis, cranial nerve palsies, Parkinsonian-like tremors, and movement disorders have also been observed. WNV-associated meningitis usually involves fever, headache, and stiff neck. WNV-associated poliomyelitis or acute flaccid paralysis (AFP) syndrome, is the least common among NID. AFP, initially described as atypical Guillian Barre Syndrome in New York City in 1999, was well documented in 2002 during the outbreak in Louisiana. Clinically this syndrome is characterized by the acute onset of asymmetric limb weakness or paralysis in the absence of sensory loss. The paralysis can occur in the absence of fever, headache, or other common symptoms associated with WNV infection. AFP is associated with significant short- and long-term illness and death.

WNV infection is suspected in a person based on clinical symptoms and patient history. Serologic laboratory testing is required to confirm a diagnosis. Diagnosis of acute infection requires the detection of IgM antibody. Serum IgM is usually positive within 5 to 14 days of illness in over 90% of cases and CSF IgM is positive within 7 days of onset. The most commonly performed serologic test is the IgM antibody-capture assay, the enzyme-linked immunosorbent assay (MAC-ELISA). Los Angeles County Public Health laboratory utilizes both IgM and IgG antibody-immunofluorescent Assay (IFA) as well as IgM enzyme immunoassays (EIA). The plaque-reduction neutralization test (PRNT), a cell culture based assay, is a confirmatory test performed at the CA Department of Health Services (DHS) Viral and Rickettsial Disease Laboratory (VRDL). PRNT can distinguish between arthropod-borne flaviviruses such as SLE and WNV. It can also be used to help distinguish false-positive results in an IgM antibody-capture enzyme-linked immunosorbent assay or other assays. In addition to PRNT and serologic testing, nucleotide based testing such as PCR is utilized for blood screening and for surveillance of WNV-infected mosquitoes and dead birds.

HISTORY OF WNV IN LOS ANGELES COUNTY

In 2002, a single human case of WNV-associated meningitis was confirmed. The young woman recovered uneventfully and denied history of travel outside of LAC, blood transfusions, or a history of organ transplantation. However, in 2002, there was no environmental evidence documenting the presence of WNV within LAC such as WNV-infected dead birds, mosquito pools or sentinel chickens with WNV sero-conversion. The first environmental evidence that WNV had arrived to LAC occurred in the summer of 2003 with the presence of WNV-infected dead birds, mosquito pools, and sero-positive sentinel chickens. In 2003, one human case of WNF, with symptom onset in mid-October, was laboratory confirmed, and was most likely infected by local WNV-infected mosquitoes. In June 2004, the LAC public health department documented the first of many human cases.

DISEASE ABSTRACT

- The overall incidence of reported WNV infections including asymptomatic blood donors in 2004 was 3.2 cases per 100,000 population (Figure 1).
- WNF was the most common WNV-associated clinical condition, 149 cases or 48.2 % of reported cases in 2004.
- There were 14 case fatalities, including 10 with encephalitis, 2 with meningitis, and 2 WNF cases. The mean age was 76.4 years (range 60-94).
- NID and deaths were each associated with older age.
- The lowest incidence of WNV infection was found in children; there was a dramatic increase in WNV



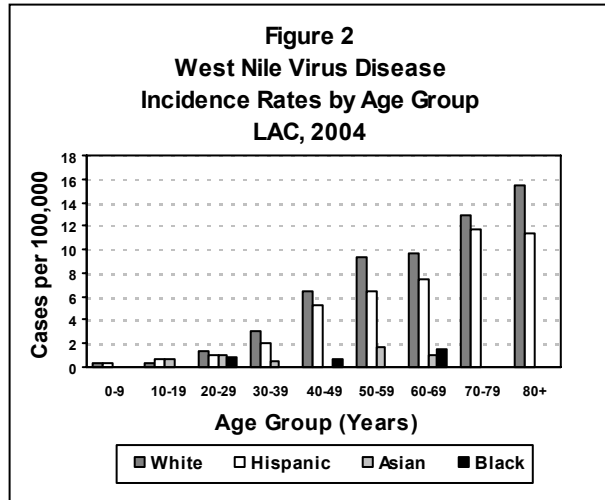
incidence after age 40 years (Figure 2).

- Non-Hispanic Whites had the highest proportion of symptomatic infections (57%) followed by Hispanics (39%).
- Significantly more male cases (65%, n=200) were reported than female (35%, n=109) in 2004.
- Most WNV infections occurred in persons residing in suburban valleys, areas close to the San Gabriel River and hillside communities.

STRATIFIED DATA

Seasonality: Cases were reported from late spring (first week of June) through fall (second week of October). The peak onset of cases was the 2nd week in August (week 33) when 41 cases were recorded (Figure 1).

Age: The median age for all reported WNV cases including asymptomatic blood donors was 54 years old (range: 5–94 years). Of the 149 WNF cases, the median age was 51 years (range: 6–91). The median age for the 82 meningitis cases was 53 years (range: 5–90). The median age for the 47 encephalitis cases was 70 years old (range: 28–94). The median age for the 7 reported AFP was 45 years (range: 34–78). The median age for the 14 fatal cases was 77.0 years (range: 60–94). The incidence of WNV infection increased steadily with age in Whites and Hispanics (Figure 2). The lowest incidence rates occurred in children under 10, 0.3 cases per 100,000, whereas the greatest incidence occurred in those > 80 years (11.6 cases per 100,000).



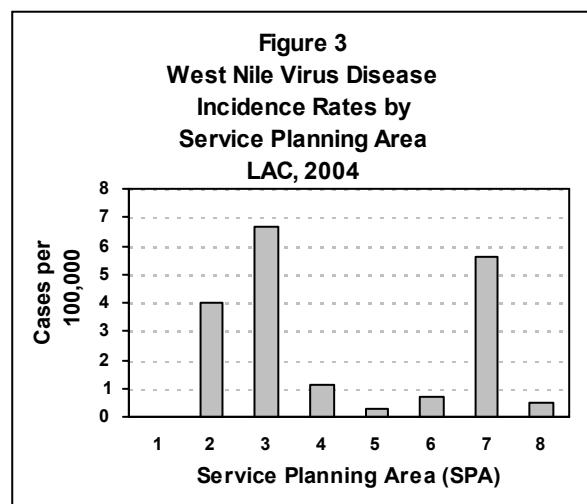
Sex: Males were almost twice as likely to present with WNV infection compared to females. The overall LAC WNV incidence rate was 4.2 cases per 100,000 male population versus 2.3 cases per 100,000 female population.

Race/Ethnicity: Whites had the both the greatest proportion of reported cases and overall highest incidence rates of infection, 57% of cases (n=170, 5.9 per 100,000), followed by Hispanics, 39% of cases (n= 117, 2.6 per 100,000). When incidence rates were reviewed by age group and race/ethnicity, whites had the highest incidence of infection in individuals age 30 and above, whereas, Hispanics had the highest incidence in age groups under age 20 years (Figure 2). Only 3% of reported cases occurred among Asian Pacific Islanders (n=8, 0.6 per 100,000) and 1% among Blacks (n=3, 0.3 per 100,000).

Location: The number of reported WNV cases was highest in SPA 3 (n=114, 6.7 per 100,000), SPA 2 (n=84, 4.0 per 100,000), and SPA 7 (n=77, 5.6 per 100,000) (Figure 3).

PREVENTION

Prevention and control of WNV and other arboviral diseases is most effectively accomplished through integrated vector management programs. These programs include surveillance for WNV activity in mosquito vectors, birds, horses, other animals, and humans, and implementation of appropriate mosquito control measures to reduce mosquito populations when necessary. Additionally, when virus activity is detected in an area, residents are alerted and advised to increase measures to reduce contact with





mosquitoes. Currently, there is no human vaccine available against WNV but several vaccines are under development. Important preventive measures against WNV include the following:

- Applying insect repellent to exposed skin. A higher percentage of DEET in a repellent will provide longer protection. DEET concentrations higher than 50% do not increase the length of protection.
- When possible, wear long-sleeved shirts and long pants when outdoors for long periods of time.
- Staying indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times.
- Help reduce the number of mosquitoes in areas outdoors by draining sources of standing water. This will reduce the number of places mosquitoes can lay their eggs and breed.

A wide variety of insect repellent products are available. CDC recommends the use of products containing active ingredients which have been registered with the U.S. Environmental Protection Agency (EPA) for use as repellents applied to skin and clothing. EPA registration of repellent active ingredients indicates the materials have been reviewed and approved for efficacy and human safety when applied according to the instructions on the label. Of the active ingredients registered with the EPA, two have demonstrated a higher degree of efficacy in the peer-reviewed, scientific literature. Products containing these active ingredients typically provide longer-lasting protection than others:

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

Picaridin is an EPA approved mosquito repellent that will be commercially available in 2005.

In 2002, WNV transmission was documented from all components of blood products including platelets, packed red blood cells, and plasma. Subsequently in 2003, all blood donors were screened for WNV infection utilizing PCR testing. Millions of units of blood were screened for WNV utilizing PCR based technology, testing donor mini-pools. Over 1000 donor units were held from distribution in 2003, and only 6 clinical WNV cases were documented to be associated with WNV infected transfusion. In 2004, WNV screening intensified and individual donors were screened in order to detect an even lower level of donor viremia than what could be detected from mini-pool screening.

COMMENTS

In 2004, 309 human WNV cases were confirmed in LAC among hundreds of tested patients. WNV is now considered endemic (enzootic) to LAC and Southern California. Sustained surveillance will be required in the coming years, including surveillance among humans, dead birds, mosquito pools, and sentinel chickens. These activities guide public health officials in providing targeted health education to communities at particularly high risk. In response to the 2004 WNV outbreak, LAC DHS specifically added WNV infection to its list of reportable diseases by authority of the Health Officer under California Code of Regulations, Title 17, Sections 2503 and 2505. Physicians and laboratories are required to report all positive laboratory findings of WNV to the Department of Health Services within one (1) working day.

Medical providers play a key role in providing WNV health education to their patients at high risk. A county-wide phone survey in September 2004 revealed that community knowledge of WNV as a potential health risk in LAC was high, 93%, and most people knew that WNV was transmitted by the bite of a mosquito, 97%. But the survey also showed the public's self-reported change in their behaviors to protect themselves against WNV was at about 50% of persons surveyed. Only 20% of respondents reported using repellent prior to 2004. Promotion of preventive measures can help to minimize the risk of being exposed to WNV is a critical part of the public health message.

VECTOR CONTROL

There are five local mosquito and vector control districts within LAC that provide mosquito abatement services to all areas of the county. They carry out mosquito and sentinel chicken surveillance, provide public information, and are critical to mosquito-borne disease control. They include:

- Greater Los Angeles County Vector Control District (GLACVCD)
- San Gabriel Valley Mosquito and Vector Control District (SGVVCD)
- Los Angeles County West Vector Control District (LACWVCD)



- Antelope Valley Mosquito and Vector Control District (AVMVCD)
- Compton Creek Mosquito Abatement District

These five local mosquito and vector control districts work closely with the ACDC to investigate confirmed and presumptive human cases of locally acquired vector-borne disease to determine the source and conditions of transmission.

GLACVCD is the largest vector control district in LAC serving 4.5 million residents in a 1,330 square mile area covering cities from San Fernando Valley, Los Angeles River, the proximate cities of Maywood, Bell, Huntington Park and portions of LAC. The West Vector Control District covers approximately 600 square miles, contains 23 cities and unincorporated territories of the County of Los Angeles, and provides services for 2,866,000 people. The District includes the cities of Agoura Hills, Beverly Hills, Calabasas, Culver City, El Segundo, Hawthorne, Hermosa Beach, Hidden Hills, Inglewood, Lawndale, Lomita, the westerly portion of LAC, Malibu, Manhattan Beach, Palos Verdes Estates, Rancho Palos Verdes, Redondo Beach, Rolling Hills, Rolling Hills Estates, Santa Monica, Torrance, West Hollywood, Westlake Village, and unincorporated territory of the County of Los Angeles. The San Gabriel Valley Mosquito and Vector Control District covers many parts of the San Gabriel Valley, including cities of Alhambra to West Covina. This district had the most cases of WNV in 2004. The Antelope Valley Mosquito and Vector Control District offers programs that provide information and education for the Antelope Valley general public, schools and community organizations; propagated through brochures, pamphlets, seminars, speeches and presentations. The District encompasses an area of approximately 230 square miles and serves residents within District boundaries in Palmdale, Lancaster and Quartz Hill.

Since mosquitoes serve as vectors for disease transmission, WNV-positive mosquito pools are another critical environmental indicator; as such, mosquito pools are routinely tested for the presence of WNV. In 2004, 378 mosquito pools tested positive in LAC, nearly one-third of the identified positive mosquito pools in California (1,136 pools). The last positive mosquito pool in LAC was identified on October 21, 2004 from Harbor City.

In an effort to help protect the public health from the threat of WNV disease, the LAC DHS funded a one year agreement with local mosquito and vector control districts to provide mosquito abatement services to all areas of the county not currently within the jurisdiction of a control district. At least 186,000 persons reside in such regions. On July 2004, city managers in La Cañada-Flintridge, South Pasadena, and Baldwin Park—cities without mosquito control programs—were notified that the County would provide temporary mosquito abatement services by contracting with the San Gabriel Valley Mosquito and Vector Control District for fiscal year 2004-2005. Because portions of the cities of Palmdale and Lancaster as well as unincorporated Antelope Valley and Santa Clarita Valley regions are also without abatement services, we entered into a similar one-year agreement with the Antelope Valley Mosquito and Vector Control District to provided abatement and surveillance service, pending a permanent solution. The challenge of the coming months will be to follow-up with these cities to ensure that they have plans to continue funding of local mosquito abatement services when the county contracts expire in July 2005.

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1. Campbell GL, Marfin AA, Lanciotti RS, Gubler DJ. West Nile virus. *Lancet Infect Dis* 2002 ;519-29.
2. Watson JT, Pertel PE, Jones RC, et al. Clinical characteristics and functional outcomes of West Nile fever. *Ann Intern Med*. 2004;141:360-365.
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ADDITIONAL RESOURCES

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- Mosquito and Vector Control Association of California: www.mvacac.org/



WEST NILE VIRUS WITHIN LOS ANGELES COUNTY: FIRST AUTOCHTHONOUS HUMAN WEST NILE VIRUS INFECTION IN 2003

Since the introduction of West Nile virus (WNV) in the continental US in summer of 1999, WNV has become established in nearly all of the contiguous states causing nearly 10,000 cases and 262 deaths in 2003.

In 2002, the first locally acquired human case of WNV in California was identified in Los Angeles County (LAC). However, other forms of local WNV surveillance including dead birds, sentinel chickens, and mosquito pools did not reveal evidence of WNV. In 2003, one case of WNV fever acquired within LAC was documented and laboratory-confirmed.

The first case of locally acquired WNV fever in a LAC resident was laboratory-confirmed in late December 2003 by the California Department of Health Services Viral and Rickettsial Diseases Laboratory (VRDL). ACDC was notified by ARUP Laboratory of Utah of a positive WNV serum IgM test in late October 2003 in a LAC resident. The patient and his physician were contacted and interviewed by ACDC, and the patient provided another serum specimen in early December 2003 so that confirmatory testing could be completed at the LAC Public Health Laboratory.

The case, a 61 year-old Hispanic male, was admitted to an LAC hospital for complaints of fever, fatigue, nausea and diarrhea for 10 days in mid-October 2003. Serum WNV testing was ordered by the attending physician as part of a fever work-up during his hospital admission. The patient recovered uneventfully. He lived in Whittier and gave a history of mosquito bites two days prior to admission while sleeping in his living room with a broken screen door. He believes he was bitten in the early morning of the first week of October. He denied any travel outside of Whittier area 14 days before onset of symptoms. He received no blood products or organ donations within the month prior to symptom onset. In early December, the patient's serum tested weakly positive by CDC WNV ELISA testing in both the LAC Public Health Laboratory and the state VRDL. WNV confirmatory testing, plaque reduction neutralization test, performed at the VRDL confirmed the diagnosis of WNV fever in late December 2003.

Related environmental findings in the fall of 2003 include: 11 dead crows with WNV recovered in Whittier and 6 WNV-infected mosquito pools in two adjoining cities in late September to November 2003. In 2003, there were no sentinel chickens with WNV-positive blood tests from LAC. This is the third endemic case of WNV infection acquired in the state of California in 2003 with the first two human cases being reported from Imperial and San Bernardino counties respectively.