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Research Paper

The Epidemic of West Nile Virus in the United States, 2002

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ABSTRACT

Since 1999, health officials have documented the spread of West Nile virus across the eastern and southern states and into the central United States. In 2002, a large, multi-state, epidemic of neuroinvasive West Nile illness occurred. Using standardized guidelines, health departments conducted surveillance for West Nile virus illness in humans, and West Nile virus infection and illness in non-human species. Illnesses were reported to the Centers for Disease Control and Prevention (CDC) through the ArboNET system. In 2002, 39 states and the District of Columbia reported 4,156 human West Nile virus illness cases. Of these, 2,942 (71%) were neuroinvasive illnesses (i.e., meningitis, encephalitis, or meningoencephalitis) with onset dates from May 19 through December 14; 1,157 (28%) were uncomplicated West Nile fever cases, and 47 (1%) were clinically unspecified. Over 80% of neuroinvasive illnesses occurred in the central United States. Among meningitis cases, median age was 46 years (range, 3 months to 91 years), and the fatality-to-case ratio was 2%; for encephalitis cases (with or without meningitis), median age was 64 years (range, 1 month to 99 years) and the fatality-to-case ratio was 12%. Neuroinvasive illness incidence and mortality, respectively, were significantly associated with advanced age ($p = 0.02$; $p = 0.01$) and being male ($p < 0.001$; $p = 0.002$). In 89% of counties reporting neuroinvasive human illnesses, West Nile virus infections were first noted in non-human species, but no human illnesses were reported from 77% of counties in which non-human infections were detected. In 2002, West Nile virus caused the largest recognized epidemic of neuroinvasive arboviral illness in the Western Hemisphere and the largest epidemic of neuroinvasive West Nile virus ever recorded. It is unknown why males appeared to have higher risk of severe illness and death, but possibilities include higher prevalence of co-morbid conditions or behavioral factors leading to increased infection rates. Several observations, including major, multi-state West Nile virus epidemics in 2002 and 2003, suggest that major epidemics may annually reoccur in the United States. Non-human surveillance can warn of early West Nile virus activity and needs continued emphasis, along with control of *Culex* mosquitoes. Key Words: West Nile virus—Epidemic—2002. Vector-Borne Zoonotic Dis. 4, 61–70.

INTRODUCTION

West Nile virus, which is indigenous to the eastern hemisphere (Campbell et al. 2002), was first recognized in the United States in 1999 during an outbreak in New York City (Nash et al. 2001). Through 2001, 10 states reported a total of 149 human West Nile virus illness cases and 18 deaths, while thousands of animal infections were reported from 27 states and the District of Columbia (Nash et al. 2001, Marfin et al. 2000, Centers for Disease Control

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and Prevention 2002a). In 2002, a multi-state West Nile virus epidemic resulted in an unprecedented number of neuroinvasive human illnesses and avian and equine infections that often preceded the identification of human illnesses (Centers for Disease Control and Prevention 2002b). The annual re-emergence of the virus in counties and states that were first affected in 1999, its transcontinental spread in only 3 years, many infected bird and mosquito species (Centers for Disease Control and Prevention 2002b), and continued, intense epidemic transmission in 2003 (Centers for Disease Control and Prevention 2003b), suggest that West Nile virus may establish a significantly different epidemiologic pattern than St. Louis encephalitis virus, a closely related flavivirus indigenous to the western hemisphere.

Using data collected through ArboNET, a cooperative surveillance system maintained by the Centers for Disease Control and Prevention (CDC) and 54 state and local health departments, this paper will describe the 2002 West Nile virus epidemic, including demographic factors associated with neuroinvasive West Nile virus illness and death, and the value of West Nile virus surveillance data for predicting human illness.

MATERIALS AND METHODS

Surveillance methods

The ArboNET surveillance system was established in 2000 to monitor the spread of West Nile virus in the United States. Through ArboNET, cooperating health agencies conducted surveillance using published guidelines (Centers for Disease Control and Prevention 2001a) and reported human cases of West Nile virus illness and West Nile virus-infected birds (through the collection and testing of dead birds; serial bleeding of sentinel birds; and/or avian seroprevalence studies), non-human mammals, and mosquitoes.

Surveillance guidelines recommended passive surveillance for probable or confirmed cases of West Nile meningitis and/or encephalitis meeting a standardized, clinical and laboratory-based case definition (Centers for Disease Control and Prevention 2001b) In addition, many jurisdictions voluntarily reported uncomplicated West Nile fever cases to CDC. Patients for whom clinical information could not be reviewed or for whom a clinical syndrome could not be determined were reported as having unspecified West Nile viral illnesses. Adherence to recommended guidelines was not evaluated. After the first case occurred within a jurisdiction, active case surveillance (Centers for Disease Control and Prevention 2001a) was recommended, including laboratory-based surveillance that involved testing of cerebrospinal fluid with pleocytosis. Based on evidence of increasingly prolonged seasonal West Nile virus transmission since 1999 (unpublished data), it was recommended that health departments conduct human disease surveillance from early spring through late fall 2002 in northern states, and through the entire year in southern states.

Laboratory methods

Acute-phase serum and cerebrospinal fluid samples and appropriately timed (Centers for Disease Control and Prevention 2001a), convalescent-phase serum samples were collected from suspected case-patients. In general, specimens were first tested in either state public health laboratories or commercial laboratories; results from commercial laboratories were confirmed in public health reference laboratories. Since 2000, public health laboratory personnel had received standardized protocols, reagents, and training from CDC. The following testing algorithms were recommended by CDC (Centers for Disease Control and Prevention 2001a). Serum and cerebrospinal fluid samples were tested for IgM antibodies to West Nile virus by enzyme-linked immunosorbent assay (ELISA; Martin et al. 2000, 2002) similar tests for IgM antibodies to St. Louis encephalitis virus and other closely related flaviviruses were conducted as geographically appropriate. Samples containing apparently cross-reactive IgM antibody to St. Louis encephalitis virus or another flavivirus were tested by plaque-reduction neutralization assay. Selected specimens of cerebrospinal fluid and/or brain tissue were tested by reverse-transcriptase polymerase

chain reaction (RT-PCR; Lanciotti et al. 2000) and virus isolation (Beaty et al. 1995).

Laboratory-confirmed evidence of a recent West Nile virus infection included the presence of West Nile virus-specific IgM antibody in cerebrospinal fluid; a four-fold or greater change in plaque-reduction neutralizing antibody titer against West Nile virus in acute- and convalescent-phase serum samples; the presence of West Nile virus-specific IgM and neutralizing antibodies in a single serum sample; the isolation of West Nile virus in culture; or the demonstration of West Nile virus genomic sequences in serum, cerebrospinal fluid, other bodily fluids, or tissues. Laboratory-probable evidence of West Nile virus infection included the presence of either IgM or neutralizing antibodies against West Nile virus in a single serum sample.

Data collection and analysis

Human cases were reported to ArboNET by the health department in the patient's state of residence via telephone or fax only. For each case, the date of illness onset, county and state of residence was reported and, when available, the patient's age, sex, clinical syndrome, and vital status was also reported. Cases with illness onset during 2002 and reported to ArboNET between January 1, 2002 and April 15, 2003 were included in this analysis. The reported cases of encephalitis, meningitis, and meningoencephalitis were combined as neuroinvasive West Nile virus illness, and the incidence was calculated as cases per million population using 2000 U.S. Census data. Incidence data were mapped by county and state using ArcGIS software, version 8.1 (ESRI). Crude relative risks and 95% confidence intervals (Miettinen and Nurminen 1985), continuity-corrected chi-squared test, Spearman's rank correlation (Hollander and Wolfe 1999), and Mantel-Haenzel chi-squared statistics were calculated using S-Plus Professional software, version 6.1 (Insightful Corp). All *p*-values presented are two-sided unless stated otherwise. West Nile virus infections of animals were continuously reported to ArboNET by health departments via the Internet using a secured data network.

RESULTS

In 2002, 4,156 human West Nile virus illness cases were reported to ArboNET from 739 counties in 39 states and the District of Columbia. Twenty-nine states and the District of Columbia reported their first human cases ever. In addition, 16,741 WNV-infected dead birds were reported from 42 states and the District of Columbia; 14,571 infected non-human mammals (14,539 equids) were reported from 41 states; and 6,604 infected pools of mosquitoes from 29 species were reported from 37 (81%) of 45 jurisdictions performing mosquito surveillance.

Patient demographic and clinical information

Of 4,156 reported human West Nile virus illness cases, 2,259 (54%) were classified as "confirmed" and 1,897 (46%) were "probable." Subsequently, confirmed and probable cases will be combined. Complete demographic and clinical data were available for 4,146 (99%) of the 4,156 total illness cases. Of these 4,146 illness cases, 2,942 (71%) were neuroinvasive (i.e., meningitis, encephalitis, or meningoencephalitis); 1,157 (28%) were uncomplicated West Nile fever; 47 (1%) were clinically unspecified; and 284 were fatal (Table 1). Among the fatal cases overall, the median age was 77.5 years (range: 19–99 years). Of the 2,942 total neuroinvasive illness cases reported from 36 states and the District of Columbia, 722 (25%) were meningitis; 918 (31%) were encephalitis; 1,302 (44%) were meningoencephalitis; and 276 (9%) were fatal. Of 2,220 encephalitis or meningoencephalitis cases, 1,239 (56%) were in males; the median age was 64 years (range: 1 month to 99 years) and 261 (12%) were fatal. Of 722 meningitis cases, 361 (50%) were in males; the median age was 46 years (range: 3 months to 91 years), and 15 (2%) were fatal. Of 1,157 West Nile fever cases reported from 30 states and the District of Columbia, 582 (50%) were in males and the median age was 49 years (range: 1 year to 97 years); of these, 7 (1%) were fatal. In the remainder of this paper, only the 2,942 neuroinvasive illness cases will be discussed.

Most neuroinvasive illness cases were reported from north-central and southern states

TABLE 1. DEMOGRAPHIC AND CLINICAL INFORMATION FOR 4,146 HUMAN WEST NILE VIRUS ILLNESS CASES REPORTED TO CENTERS FOR DISEASE CONTROL AND PREVENTION, UNITED STATES, 2002

	Clinical syndrome			
	Encephalitis or meningoencephalitis	*Meningitis*	*Fever*	*Unspecified*
No. cases (%)	2,220 (54)	722 (17)	1,157 (28)	47 (1)
No. males (%)	1,239 (56)	361 (50)	582 (50)	26 (55)
Age				
Median	64 years	46 years	49 years	43 years
Range	1 month to 99 years	3 months to 91 years	1–97 years	0–89 years
Group (%)				
0–39	359 (16)	253 (35)	306 (26)	21 (45)
40–69	974 (44)	367 (51)	678 (59)	21 (45)
70 and older	887 (40)	102 (14)	173 (15)	5 (10)
No. deaths (%)	261 (12)	15 (2)	7 (1)	1 (2)
No. males (%)	164 (63)	11 (73)	7 (100)	0 (0)
Age				
Median	78 years	74 years	72 years	89 years
Range	19–99 years	40–91 years	59–89 years	89 years

located along the Mississippi River or one of its major tributaries. Of the 2,942 reported cases, 2,478 (84%) were reported from only 11 states: Illinois, Indiana, Kentucky, Louisiana, Michigan, Mississippi, Missouri, Nebraska, Ohio, Tennessee, and Texas (Table 2). Mississippi had the highest incidence of neuroinvasive illness (57 cases per million persons). Several heavily populated counties in these states had reported incidences of 100 per million or greater (Fig. 1).

TABLE 2. NUMBER OF REPORTED CASES AND INCIDENCE OF NEUROINVASIVE HUMAN WEST NILE VIRUS ILLNESS FOR STATES REPORTING AT LEAST 40 CASES, UNITED STATES, 2002

State	*No. cases*	*Population*[a]	*Incidence*[b]
Mississippi	162	2,844,658	56.9
Michigan	557	9,938,444	56.0
Nebraska	84	1,711,263	49.1
Louisiana	204	4,468,976	45.6
Illinois	553	12,419,293	44.5
Indiana	180	6,080,485	29.6
Ohio	310	11,353,140	27.3
Missouri	126	5,595,211	22.5
Kentucky	53	4,041,769	13.1
Texas	202	20,851,820	9.7
Tennessee	47	5,689,283	8.3
New York	68	18,976,457	3.6
Pennsylvania	42	12,281,054	3.4

[a]2000 U.S. Census data.
[b]Per million population.

Several sparsely populated midwestern counties reported few cases but also had incidences exceeding 100 cases per million persons. Los Angeles County, California was the only county west of the Rocky Mountains to report a human case, but no other West Nile virus infections among non-human species were reported statewide.

Reported illness onset dates ranged over a 30-week period from May 19 (reported from the District of Columbia) to December 14 (reported from Mississippi) (Fig. 2), but 2,178 cases (74%) had onsets during a 6-week period from August 11 through September 21. The epidemic peak occurred during the week ending August 24, when 451 neuroinvasive illnesses occurred. The duration of the epidemic was longer in the southern United States, where neuroinvasive illnesses were reported weekly from mid-June through mid-December.

Age-specific neuroinvasive illness incidence and mortality

Neuroinvasive illness incidence (one-sided $p = 0.02$) and fatality-to-case ratios (one-sided $p = 0.01$), respectively, increased significantly with age by Spearman's rank correlation (Table 3). Compared to an incidence of four cases per million in persons aged <40 years (reference

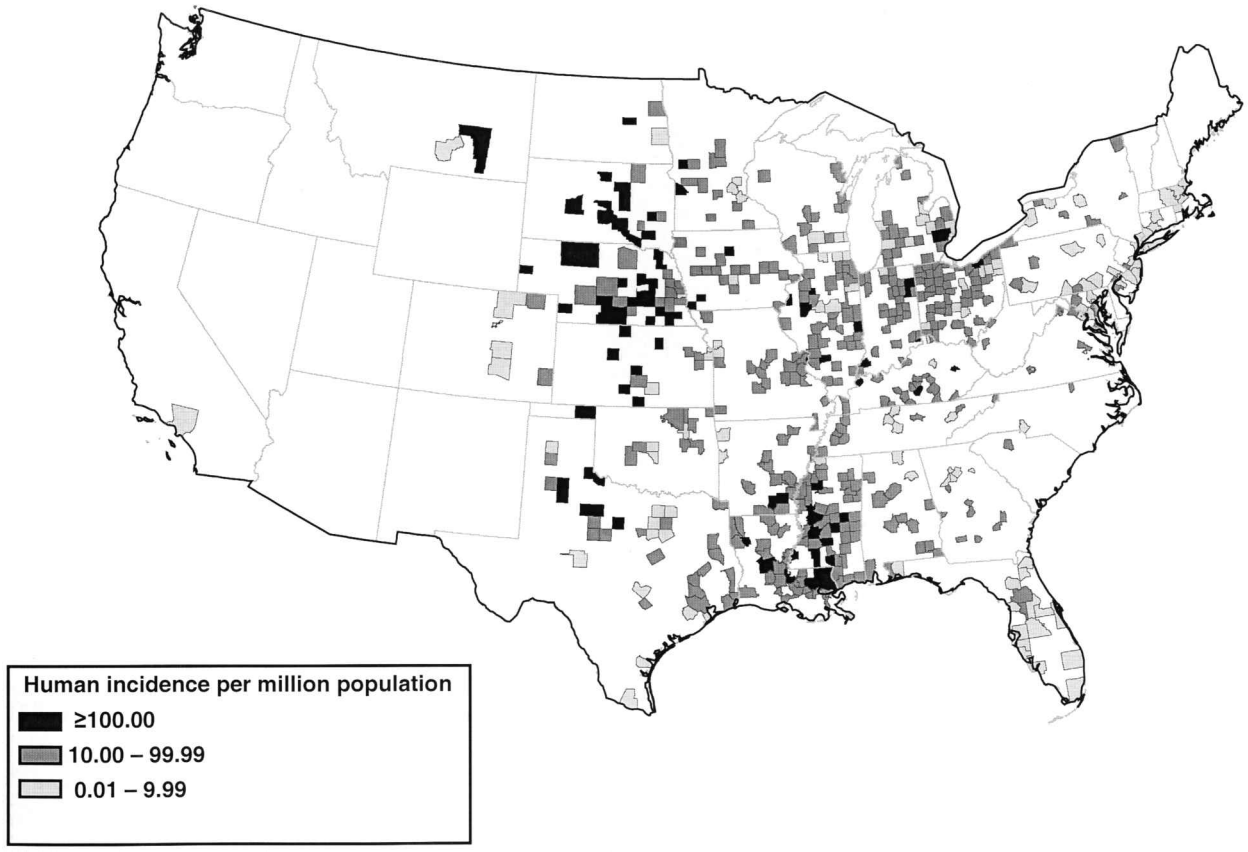

FIG. 1. Reported incidence of neuroinvasive human West Nile virus illness, by county and state, United States, 2002 (n = 589 counties).

population), the incidence was more than three times as high in persons aged 40–59 years; six times as high in those aged 60–69 years; 10 times as high in those aged 70–79 years; and nearly 12 times as high in those aged ≥80 years. Of 2,942 neuroinvasive illnesses, 276 (9%) were fatal (Table 3). Mortality was greatest among older persons. Of 989 persons aged ≥70 years, 205 (21%) died compared to 70 (4%) of 1,837 persons aged 20–69 years and only 1 (1%) of 116 persons aged ≤19 years.

Among middle-aged and older individuals, both neuroinvasive illness incidence ($p < 0.001$) and fatality-to-case ratios ($p = 0.002$) were significantly higher in males than in females (Table 3). Among persons aged 40–69 years, the incidence was 1.3 times as high and fatality-to-case ratio nearly twice as high for men than for women. Among persons aged ≥70 years, the incidence was nearly twice as high and fatality-to-case ratio 1.3 times as high for men as for women.

Surveillance events preceding neuroinvasive human illnesses

Of 589 counties reporting human neuroinvasive illnesses, 527 (89%) first detected West Nile virus transmission in non-human species, while 59 (10%) first detected transmission in humans. In three counties (1%), illness onset of the first reported human case and collection of the first infected animal occurred on the same day. Among the 527 counties where non-human infections were found before human cases, the detection of West Nile virus-infected dead birds was the first positive surveillance event in 378 counties (72%); infected non-human mammals in 95 (18%); infected mosquitoes in 34 (6%); and seroconversions among sentinel birds in 12 (2%). In eight counties (2%), multiple types of non-human surveillance events occurred on the same day. In 378 counties in which infected dead birds were the first posi-

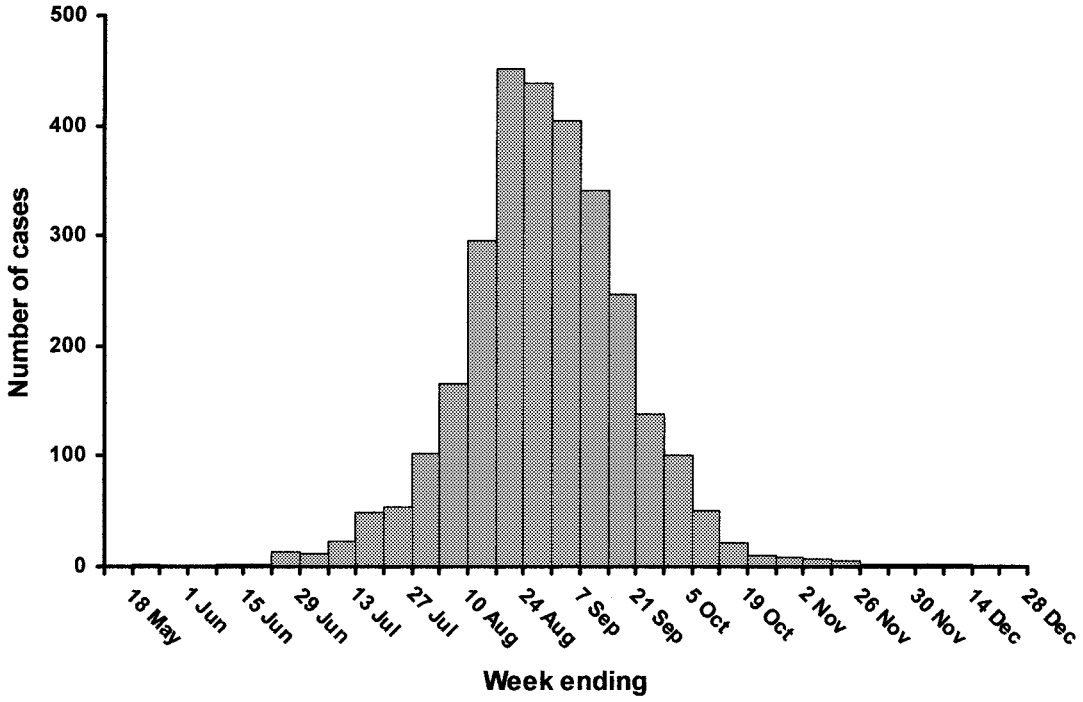

FIG. 2. Reported number of neuroinvasive human West Nile virus illness cases by week of illness onset, United States, 2002 (n = 2,942 cases).

tive surveillance event, the median lead time was 38.5 days (range: 2–252 days). In 320 (85%) of these counties, the lead time exceeded 14 days (the approximate upper limit of West Nile virus incubation in humans). Of 2,531 counties that reported any West Nile virus transmission in 2002, 1,939 (77%) reported infected non-human species but did not report any neuroinvasive human illnesses.

DISCUSSION

In 2002, West Nile virus caused the largest recognized epidemic of neuroinvasive arboviral illness in the western hemisphere (Creech 1977) and the largest epidemic of neuroinvasive West Nile virus illness ever recorded (Nash et al. 2001, Tsai et al. 1998, Platanov et al. 2001, Weinberger et al. 2001). In 2002, human West Nile virus illnesses were reported from each of 10 states that had reported cases in 2001 (Centers for Disease Control and Prevention 2002a). In addition, in 2002, human illnesses were reported for the first time from 29 states and the District of Columbia, including intense human transmission in the Midwest (Table 2) and marked westward geographic expansion of West Nile virus activity across the United States.

Based on the reports of roughly 3,000 neuroinvasive illnesses in 2002 and on previously derived serosurvey estimates that 60 to 400 human West Nile virus infections occur for each reported neuroinvasive illness (Tsai et al. 1998, Mostashari et al. 2001, Centers for Disease Control and Prevention 2001c), an estimated 180,000 to 1,200,000 infections occurred in the United States in 2002. Although persons with neuroinvasive illness have been shown with persistent West Nile virus-specific IgM in the following transmission season (Roehrig et al. 2003), the finding of acute central nervous system disease and West Nile virus-specific IgM is unlikely to be coincidental and to have caused significant overestimation of neuroinvasive illness incidence in 2002, given the low incidence of West Nile virus infections the United States (Mostashari et al. 2001, Centers for Disease Control and Prevention 2001c).

As reported in previous West Nile virus outbreaks (Nash et al. 2001, Tsai et al. 1998, Platanov et al. 2001, Weinberger et al. 2001), neuroinvasive illness incidence and mortality were

TABLE 3. RELATIVE RISK ESTIMATES FOR NEUROINVASIVE HUMAN WEST NILE VIRUS ILLNESS[a] AND FATALITY BY AGE GROUP AND SEX, UNITED STATES, 2002

	Neuroinvasive West Nile virus illness				*Fatal neuroinvasive West Nile virus illness*	
	Population at risk[b]	*Number (%)*	*Incidence per million population*[c]	*Relative risk (95% CI)*[d]	*Number (fatality-to-case ratio [%])*[c]	*Relative risk (95% CI)*[d]
All cases						
Age category (years)						
0–9[e]	35,677,630	42 (1)	1	Reference	0 (0)	Reference
10–19[e]	36,554,351	74 (3)	2	Reference	1 (1)	Reference
20–29[e]	34,481,647	177 (6)	5	Reference	2 (1)	Reference
30–39[e]	39,028,636	319 (11)	8	Reference	4 (1)	Reference
40–49	38,277,913	451 (15)	12	2.8 (2.5–3.2)	7 (2)	1.4 (0.5–3.7)
50–59	27,933,078	431 (15)	15	3.7 (3.2–4.2)	16 (4)	3.3 (1.4–7.8)
60–69	18,363,781	459 (16)	25	6.0 (5.3–6.7)	41 (9)	8.5 (3.9–18.4)
70–79	14,735,227	594 (20)	40	9.6 (8.6–10.7)	100 (17)	17.5 (8.4–36.8)
80–89	7,020,171	353 (12)	50	12.0 (10.5–13.6)	90 (25)	29.6 (14.2–62.0)
90 and older	1,323,508	42 (1)	33	7.6 (5.5–10.3)	15 (36)	48.0 (21.5–105.2)
Total	253,395,942	2942 (100)	12		276 (9)	
Male						
Age category (years)						
0–39	74,030,546	323 (20)	4	1.1 (0.9–1.3)	5 (2)	2.2 (0.5–10.0)
40–69	41,047,519	741 (46)	18	1.3 (1.2–1.5)	45 (6)	1.8 (1.1–3.2)
70 and older	9,015,501	536 (34)	59	1.8 (1.6–2.1)	125 (23)	1.3 (1.0–1.7)
Total	124,093,566	1,600 (100)	13	1.2 (1.1–1.3)	175 (11)	1.5 (1.2–1.9)
Female						
Age category (years)						
0–39[f]	71,711,718	289 (21)	4	Reference	2 (1)	Reference
40–69[f]	43,527,253	600 (45)	14	Reference	19 (3)	Reference
70 and older[f]	14,063,405	453 (34)	32	Reference	80 (18)	Reference
Total[f]	129,302,376	1,342 (100)	10	Reference	101 (8)	Reference

[a]Includes encephalitis, with or without meningeal signs, and meningitis.
[b]Population figures are from the 2000 U.S. Census and exclude the 14 states that did not report neuroinvasive human West Nile virus illnesses in 2002: AK, AZ, DE, HI, ID, ME, NV, NH, NM, OR, UT, VT, WA, WY.
[c]Neuroinvasive illness incidence and fatality rates, respectively, increased significantly with age (Spearman's rank correlation one-sided, $p = 0.02$; 0.01)
[d]CI denotes confidence interval.
[e]Persons in these four age groups were combined to serve as the reference population.
[f]Females in all age groups were combined to serve as the reference population.

strongly associated with advancing age. In addition, in 2002 neuroinvasive illness incidence and mortality were slightly higher for males than females. Although this sex-specific risk has not been reported during other outbreaks, it was seen in the United States during 1999–2001 (Campbell et al. 2002); during this period, 60% of the 142 nationally reported neuroinvasive illnesses were among males. No sex-related difference in mortality among these cases was noted. The reasons why males appear at higher risk than females for developing neuroinvasive illness are unknown but may include behavioral differences that result in different infection rates (e.g., occupational or recreational exposure), or co-morbid conditions such as cerebrovascular disease (Centers for Disease Control and Prevention 2003a) that more greatly affect males than females. In limited studies, sex-related differences in post-epidemic West Nile virus seroprevalence have not been reported (Tsai et al. 1998, Centers for Disease Control and Prevention 2001c). Similarly, the reasons why men with neuroinvasive illness appear at higher risk of death than women are unknown.

Alternatively, either or both of these apparent associations could be the result of surveillance artifacts and should be interpreted cautiously. The ArboNET surveillance system

relies on general guidelines developed in cooperation with participating public health departments to standardize data collection. However, types and duration of surveillance, clinical categorization, and decisions to report individual cases are left to participating agencies, and studies to validate these activities have not yet been conducted. Thus, the effect of misclassification and reporting biases on these results, if any, is unknown.

It is difficult to predict what long-term epidemiologic pattern West Nile virus will assume in the United States; this pattern may be similar to that of either St. Louis encephalitis or Japanese encephalitis virus, two related but epidemiologically distinct flaviviruses. St. Louis encephalitis virus transmission to humans has been recognized in most areas of the United States. Like West Nile virus, it is maintained and amplified in transmission cycles that involve passerine birds as amplifying hosts and culicine mosquitoes as vectors. Since 1933 when it was first recognized in the United States (United States Public Health Service 1935), St. Louis encephalitis virus has caused sporadic cases, case clusters or regional outbreaks resulting in dozens to hundreds of neuroinvasive illnesses. However, in 1975, a major epidemic occurred in the Mississippi River drainage with 2,131 neuroinvasive cases and 171 deaths (Creech 1977). In contrast, Japanese encephalitis virus occurs only in Asia where intense seasonal transmission can occur in rural transmission cycles involving culicine mosquitoes, aquatic birds, and pigs and where annual epidemics involving thousands of cases have been reported (Tsai et al. 1999).

Several epidemiologic characteristics of the 1975 St. Louis encephalitis epidemic, including thousands of human neuroinvasive cases, intense human transmission in Illinois, Indiana, Kentucky, Michigan, Mississippi, Ohio, and Tennessee, and an epidemic peak in late August and early September, bear similarity to the West Nile virus epidemic of 2002. However, other observations suggest that, at least initially, West Nile virus will establish a Japanese encephalitis-like epidemic pattern in the United States. These observations include perennial West Nile virus transmission to animals and humans in the eastern United States for the past 3–4 years; in 2002, far higher numbers of neuroinvasive human West Nile virus illnesses compared to St. Louis encephalitis illnesses in regions where both were reported (unpublished data); detection of West Nile virus in many mosquito species, including anthropophilic *Aedes*, *Culex* (i.e., *Cx. tarsalis*) and *Ochlerotatus* species (Marfin et al. 2000, Centers for Diease Control and Prevention 2002a,b), and in many avian species that are potential amplifying hosts (Komar et al. 2003); a prolonged transmission season; and, in 2003, a major, multi-state epidemic centered in the western Great Plains states (Centers for Disease Control and Prevention 2003b).

Since its first description in 1937 (Smithburn et al. 2003), large epidemics of uncomplicated West Nile fever with few neuroinvasive illnesses have been described (Marberg et al. 1956, McIntosh et al. 1976). Although a modest number of West Nile fever cases were reported in the United States during 1999–2001 (Campbell et al. 2002), the reporting of such cases increased greatly in 2002 and 2003 (unpublished data), with more than 1,000 annually reported cases nationwide. Nevertheless, West Nile fever was probably significantly underdiagnosed in the United States, as it has been estimated from a previous serosurvey that approximately 20 West Nile fever illnesses occur for every neuroinvasive illness (Mostashari et al. 2001). The value of increased testing and surveillance for West Nile fever cases is unknown.

In 2002, West Nile virus transmission in nonhuman species occurred before the first human case in nearly 90% of counties that reported neuroinvasive human illnesses. However, the short lead time between the first animal surveillance event and the illness onset of the first human cases, and delays in laboratory testing and reporting that often occur, present challenges for public health officials who are trying to use these data to intensify public health interventions and educational messages before humans are at significant risk. In addition, the specificity of non-human surveillance data as a predictor of subsequent human illness is apparently low.

In 2002, neuroinvasive human illnesses were reported in only 23% of 2,519 counties in which

animal-based surveillance detected the virus. However, because public health measures (e.g., intensified mosquito control and educational programs) taken following the detection of West Nile virus in non-human species may decrease human risk, and because such effects are difficult or impossible to quantify in a scientifically rigorous fashion, this observation should be interpreted with caution. For the foreseeable future, West Nile virus prevention and control strategies should be based on more "common sense" approaches—well-funded vector mosquito control programs that emphasize the control of *Culex* mosquitoes (i.e., utilizing larval source mapping, source reduction, and larviciding); high-quality non-human and human surveillance; and ongoing public education on the importance of personal protection measures and the elimination of periresidential mosquito habitat.

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