

Human surveillance for West Nile virus infection in Ontario in 2000

E. Lee Ford-Jones,* Margaret Fearon,† Chuck Leber,† Prabo Dwight,† Moira Myszak,*† Beverly Cole,*† Pam Baker Greene,*† Sheila Artes,† Allison McGeer,‡ Colin D’Cunha,† Monika Naus,† for the Ontario West Nile Virus Working Group§

Abstract

Background: The first reports of West Nile virus (WNV) infection in the United States in 1999 prompted Ontario to establish a surveillance protocol to monitor for the possible spread of the virus into the province. Surveillance components included evaluation of dead birds, sentinel chickens, mosquito pools and human disease. We report the results of human surveillance in 2000.

Methods: Between July 1 and Oct. 31, 2000, an active surveillance program was undertaken in which designated site coordinators in sentinel hospitals identified patients who met the suspect case definition (fever and fluctuating level of consciousness [encephalopathy], with or without muscle weakness). During the same period, following province-wide distribution of educational material, all other patients tested for WNV antibodies were identified through review of provincial laboratory reports (laboratory-based enhanced passive surveillance).

Results: Of the 60 hospitals contacted, 59 agreed to participate in the active surveillance program; 52 provided information on a regular (weekly) basis, and 7 submitted fewer than 8 reports. Thirty-six (61%) of the sentinel sites reported suspect cases. In total, 188 patients were tested (130 identified through active surveillance and 58 through enhanced passive surveillance). Patients identified through active surveillance were more likely than those identified through passive surveillance to meet the suspect case definition (43% [$n = 56$] v. 7% [$n = 4$]), to be admitted to hospital (75% [$n = 99$] v. 16% [$n = 9$]), to have a longer hospital stay (mean 25 v. 3 days), to have had a second (convalescent) serum sample collected (37% [$n = 48$] v. 31% [$n = 18$]), to have had a cerebrospinal fluid (CSF) sample banked (56% [$n = 73$] v. 14% [$n = 8$]) and to have had a discharge diagnosis reported (79% [$n = 103$] v. 28% [$n = 16$]). Of the 60 patients (32%) who met the suspect case definition, 34 (57% [31 active, 3 passive]) had a discharge diagnosis of encephalitis. Of these, 17 (50% [15 active, 2 passive]) had paired serum samples collected, and 18 (51% [all active]) had a CSF sample banked. The reported causal agents were herpes simplex virus ($n = 8$), varicella virus ($n = 2$), Powassan virus ($n = 1$), echovirus 30 ($n = 1$) and group B *Streptococcus* ($n = 1$); the cause was unknown in 18 cases. One patient died of encephalitis. The remaining 26 patients who met the suspect case definition were ultimately found to have nonencephalitic infections, vascular events or alcohol- or drug-related illness. The 128 (68%) tested for WNV who did not meet the suspect case definition included 9 patients ultimately discharged with a diagnosis of encephalitis. No cases of WNV infection were identified.

Interpretation: Only one-third of the tested patients met the suspect case definition of encephalopathy on admission, and nearly half of them were later found to have another diagnosis; others did not meet the case definition but were later discharged with a diagnosis of encephalitis. This affirms that identification of acute encephalitis on the basis of symptoms at the time of admission is often impossible.

Research

Recherche

From *the Hospital for Sick Children, University of Toronto, †the Ontario Ministry of Health and Long-term Care, and ‡Mount Sinai Hospital, Toronto, Ont.

§A list of the working group members appears at the end of the article.

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The first known North American outbreak of West Nile virus (WNV) infection occurred in New York City in 1999.¹ Sixty-two cases were reported, including 7 deaths involving people over 65 years old.²⁻⁵ The virus may have been introduced through infected birds, legally or illegally entering the United States,⁶ although other possible routes of transmission through humans or mosquitoes cannot be ruled out. Most cases are asymptomatic, but WNV can cause high fever with encephalitis, profound muscle weakness⁷ or meningitis in 1%–10% of those infected (mostly older adults)⁸⁻¹⁰ and occasionally other findings.¹¹⁻¹³

Despite a vector control program in New York State in September 1999, overwintering mosquitoes¹⁴ and a red-tailed hawk¹⁵ were found to have detectable virus in the late winter of 2000. To ensure the identification of WNV infection in Canada during the mosquito season of 2000, Health Canada organized a surveillance program to be adopted in provinces east of Alberta (a) to educate hospital staff about the virus and clinical findings, (b) to report all suspect cases of meningitis, encephalitis and profound muscle weakness, (c) to ensure that paired serum samples were sent to the provincial public health laboratories for testing and that a sample of cerebrospinal fluid was banked and (d) to initiate a public awareness campaign and take further preventive action to decrease the likelihood of human infection. We report the Ontario experience.

Methods

Between July 1 and Oct. 31, 2000, active human surveillance for WNV infection was undertaken at selected sentinel hospitals in Ontario, and enhanced passive surveillance was conducted through education and review of laboratory reports of specimens submitted from other hospitals and physicians' offices in the province. Complete surveillance data were disseminated to the public biweekly on the provincial government's Web site (www.gov.on.ca/health).

For the active surveillance, the Ontario WNV Working Group selected 60 sentinel hospitals in key geographic locations, including the metropolitan centres of Windsor, London, Hamilton and Toronto, and regions such as Haliburton-Kawartha, Niagara, Haldimand and Leeds. Each hospital was contacted, and a local site coordinator was identified and sent a weekly fax summarizing local and North American surveillance data. Designated hospital staff, with their ward-based medical and nursing colleagues and laboratory staff, identified patients who met the suspect case definition: fever and fluctuating level of consciousness, with or without muscle weakness, or infectious encephalopathy, meningitis, meningoencephalitis, transverse myelitis or Guillain-Barré syndrome of unknown causes. These symptoms were purposefully broad to ensure timely identification of a WNV case while excluding more trivial, nonspecific disease. The designated hospital staff ensured WNV testing and provided weekly reports to the site coordinator. The site coordinator then sent weekly faxes to the surveillance coordinator indicating whether or not suspect cases had been identified and, if present, provided demographic and clinical information. Sentinel hospitals failing to provide a weekly fax were contacted by telephone.

For the enhanced passive surveillance, the chief medical officer of health contacted all hospitals and appropriate specialists in the hospital and advised them of the importance of recognizing, test-

ing and reporting cases of encephalitis, meningoencephalitis and adult viral meningitis that may be caused by WNV. Reports were reviewed of WNV testing of specimens submitted to the provincial laboratory from hospitals other than the sentinel hospitals and from physicians' offices.

Patients identified through active or passive surveillance were classified into 5 categories — category 1: the suspect case definition was met, and the discharge diagnosis was encephalitis; category 2: the discharge diagnosis was encephalitis, but the suspect case definition was not met; category 3: the suspect case definition was met, but another cause was found (e.g., vascular, alcohol related); category 4: the discharge diagnosis was meningitis, and the suspect case definition may or may not have been met; and category 5: the suspect case definition was not met, and the patient had a diagnosis other than encephalitis or meningitis.

For testing, paired serum samples were collected: one at presentation (acute sample) and one at 10 to 14 days after the onset of symptoms (convalescent sample). Both serum samples were tested for antibodies to WNV and to eastern equine encephalitis, western equine encephalitis, dengue, Powassan and St. Louis encephalitis viruses with the use of a hemagglutination inhibition assay. Acute arboviral infection was defined as an antibody titre that was at least 4-fold higher or lower in the acute sample than in the convalescent sample. Specimens positive for arboviral infection were forwarded to the National Microbiology Laboratory in Winnipeg for confirmatory testing using the plaque reduction neutralization test in Vero cells,¹⁶ enzyme-linked immunosorbent assay or polymerase chain reaction. Where available, a sample of 1–2 mL of cerebrospinal fluid (CSF) was collected, frozen at –70°C and transported to the provincial laboratory for banking until the serologic test results were known.

Surveillance coordinators liaised weekly with the provincial laboratory to determine the status of specimen collection. When necessary, staff at the laboratory requested follow-up specimens from the originating site. Surveillance coordinators contacted the site to obtain information on the patient's length of stay and discharge diagnosis.

Results

Of the 60 sentinel hospitals contacted for active surveillance, 59 agreed to participate in full or in part (i.e., reported only if any patients met the suspect case definition). Weekly faxed reports were provided by the site coordinators at 52 (88%) of the hospitals. The overwhelming majority of cases came from the densely populated Toronto area, reflecting the surveillance base, whereas passively reported cases came from all regions (Fig. 1).

Overall, 188 patients (100 men) were tested for WNV infection: 130 (69%) were identified through active surveillance and 58 (31%) through enhanced passive surveillance (Table 1). The majority of patients ($n = 119$) had discharge diagnoses provided (Table 1). Nine patients had a hospital discharge diagnosis of encephalitis but did not meet the suspect case definition (category 2). Nearly half of all reported cases (46% [87/188]) did not meet the case definition and had a diagnosis other than encephalitis or meningitis (category 5); proportionally more were identified through passive surveillance than through active surveillance (55% v. 45%)

in this group. Five patients (3%) died; the causes were encephalitis, suspected Creutzfeld–Jakob disease, sudden death of unknown cause, ischemic bowel and respiratory failure.

Patients reported through active surveillance were more likely than those identified through passive surveillance to meet the suspect case definition (43% [$n = 56$] v. 7% [$n = 4$]), to be admitted to hospital (75% [$n = 99$] v. 16% [$n = 9$]), to have a longer mean length of stay in hospital (25 v. 3 days), to have had a second (convalescent) serum sample collected (37% [$n = 48$] v. 31% [$n = 18$]), to have had a CSF sample banked (56% [$n = 73$] v. 14% [$n = 8$]) and to have had a discharge diagnosis provided (79% [$n = 103$] v. 28% [$n = 16$]).

Active human surveillance

Of the 130 cases reported by the sentinel hospitals, 56 (43%) met the suspect case definition (categories 1 and 3). Of the 128 patients for whom age was known, most (67%) were 18–65 years old; 13% were older than 65, and 20% were younger than 18.

In category 1, there were 31 patients with encephalopathy (Table 1), of whom 30 (97%) were admitted to hospital (mean stay 25 days). Serologic testing of acute samples was performed for 28 (90%) of the patients; 3 (11%) were found to have arboviral antibodies. Serological testing of convalescent samples was performed for 15 patients (48%). CSF samples were banked for 18 patients (58%). CSF test results were available for 28 patients, and 16 (57%) were found to have pleocytosis (white blood cell count $> 5 \times 10^6/L$). One patient, a 25-year-old woman in the Toronto area, died of encephalitis of unknown cause.

In category 2, 5 patients were reported by sentinel hospitals with fever or fluctuating level of consciousness, not both, and had a discharge diagnosis of encephalitis. All 5

were admitted to hospital (mean stay 18 days) and had serologic testing of acute samples; serologic testing of convalescent samples was performed in 2 cases, and CSF samples were banked in 3 cases. One patient was found to have arboviral antibodies (Table 1).

There were 25 patients in category 3; all met the suspect case definition, but encephalitis was ruled out in favour of another diagnosis (Table 1). Twenty-three (92%) were admitted to hospital (mean stay 18 days).

Twenty-nine patients were reported to have meningitis (category 4); in 3 the cause was bacterial. Four patients were less than 18 years of age. Most (97% [28/29]) were admitted to hospital (mean stay 8 days). Serologic testing of acute samples was undertaken in 27 (93%) of cases; convalescent samples were tested in 16 cases (55%), and 26 (90%) had a CSF sample banked. Three patients were found to have arboviral antibodies (Table 1).

There were 39 patients who did not meet the suspect case definition and who had a diagnosis other than encephalitis or meningitis (category 5). Only one-third were admitted to hospital (mean stay 3.5 days). The other two-thirds were seen in emergency departments or in doctors' offices. Acute serum samples were tested in 34 cases (87%), convalescent samples were tested in 8 cases (21%), and 14 (36%) had a CSF sample banked. Two patients were found to have arboviral antibodies.

One patient, less than 18 years of age, was reported to have Guillain–Barré syndrome and was admitted to hospital for 19 days. Serologic testing showed no arboviral antibodies.

Enhanced passive surveillance

Of the 58 patients identified through the review of public health laboratory reports of WNV testing, 48 (83%) did

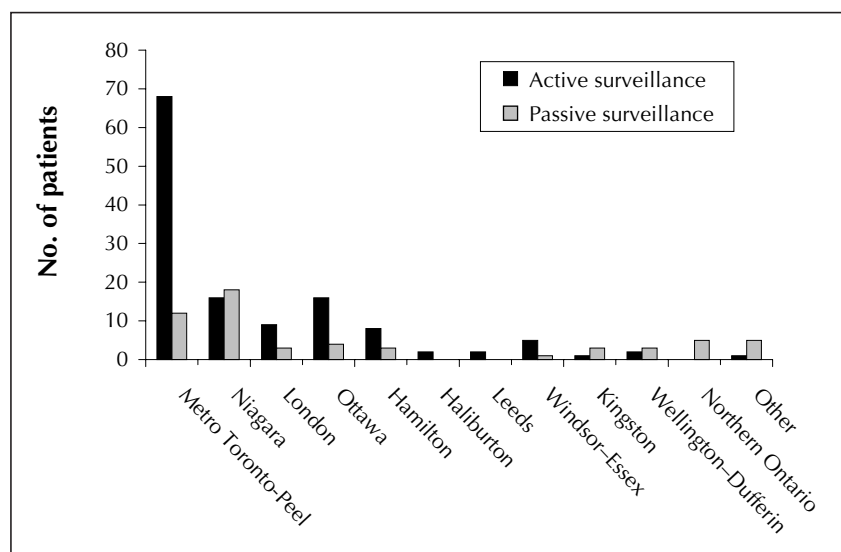


Fig. 1: Geographic distribution of patients identified through active ($n = 130$) and enhanced passive ($n = 58$) surveillance for West Nile virus testing in Ontario from July 1 to Oct. 31, 2000.

Table 1: Classification of patients identified through active and laboratory-based (enhanced passive) surveillance for West Nile virus infection in Ontario from July to October 2000

Characteristic	Category 1: Met case definition* and had discharge diagnosis of encephalitis n = 34		Category 2: Had discharge diagnosis of encephalitis but did not meet case definition n = 9		Category 3: Met case definition but another cause found n = 26		Category 4: Had discharge diagnosis of meningitis (may or may not have met case definition) n = 31		Category 5: Did not meet case definition and had non-CNS diagnosis n = 87	
	Active n = 31	Passive n = 3	Active n = 5	Passive n = 4	Active n = 25	Passive n = 1	Active n = 29	Passive n = 2	Active n = 39	Passive n = 48
Total no. of cases	31	3	5	4	25	1	29	2	39	48
Age, yr										
< 1	0	0	0	0	1	0	0	0	3	0
1-5	3	0	0	0	1	0	0	0	2	1
6-12	2	0	0	0	4	0	3	0	1	3
13-17	2	0	1	0	0	0	1	0	1	5
18-25	2	1	1	0	1	0	7	0	3	6
26-49	13	2	1	2	6	1	17	2	22	14
50-65	6	0	2	2	2	0	0	0	3	12
> 65	3	0	0	0	10	0	1	0	2	6
No. admitted to hospital	30	2	5	2	23	0	28	2	13	3
Mean LOS (and range), d	25 (1-192)	U	18 (4-46)	1 (0-3)	18 (0-95)	0 (n/a)	8 (0-40)	3 (2-4)	3.5 (0-21)	3 (0-7)
No. of deaths	1	0	0	0	1	0	0	0	3	0
Discharge diagnosis known	30†	3‡	5†	4‡	21†	1‡	29†	2‡	17†	6‡
No. with positive arbovirus serology result (HI antibody titre > 10)	3	0	1	0	2	0	3	0	2	1
	Acute††	Conv††	Acute	Conv	Acute	Conv	Acute	Conv	Acute	Conv
	1	2	3	1	2	3	1	2	1	1
	-	-	-	NA	-	NA	-	80	160	160
WNV	40	< 20	< 10	NA	NA	40	80	80	80	80
POW	-	20	-	NA	NA	NA	20	80	320	320
DEN	-	80	-	NA	NA	NA	40	20	640	640
SLE	-	-	20	NA	NA	NA	20	40	640	640

Note: CNS = central nervous system, LOS = length of stay, WNV = West Nile virus, POW = Powassan virus, DEN = dengue, SLE = St. Louis encephalitis virus, U = unknown, - = negative, NA = serum sample not available.
 *Case definition = fever and fluctuating level of consciousness with or without muscle weakness.
 †Discharge diagnosis — Category 1: varicella encephalitis (2), group B streptococcal encephalitis (1), probable herpes simplex encephalitis (8), echovirus 30 infection (1), encephalitis of unspecified cause (18). Category 2: herpes simplex encephalitis (1), encephalitis of unspecified cause (4), alcohol- or drug-related illness (3), migraine (2), urinary tract infection (2), suspected Creutzfeldt-Jakob disease (1), sepsis (1), salmonellosis (1), viral hepatitis (1), streptococcal pneumonia (1), fever not yet diagnosed (1). Category 3: stroke (4), nonspecific viral illness (4), nonspecific viral illness (4), alcohol- or drug-related illness (3), meningitis (1), varicella meningitis (1), streptococcal meningitis (1), bacterial meningitis of unknown cause (2). Category 4: meningitis (12), viral meningitis of unknown cause (12), varicella meningitis (1), streptococcal meningitis (1), herpes meningitis (1), streptococcal meningitis (1), bacterial meningitis of unknown cause (2). Category 5: multiple sclerosis (1), nonspecific viral illness (3), hepatitis and alcohol-related illness (1), alcohol- and drug-related illness (2), fever not yet diagnosed (2), headache (2), psychiatric illness (1), death (3): sudden death, ischemic bowel and respiratory failure), other degenerative disorder (2).
 ‡Discharge diagnosis — Category 1: unspecified encephalopathic illness (3). Category 2: unspecified encephalopathic illness (3). Category 3: viral illness (1). Category 4: meningitis of unspecified cause (2). Category 5: viral illness (3), lymphadenopathy (1), pneumonia (1), *Campylobacter jejuni* infection (1).
 ††Acute = serum sample obtained during acute infection; conv = serum sample obtained approximately 10 to 14 days after presentation, during convalescent period.

not meet the suspect case definition. Of the remaining 10 patients, 3, 4, 1 and 2 were in categories 1 through 4 respectively (Table 1).

Arboviral testing

There were no patients for whom neutralizing antibodies to WNV were demonstrated. Twelve patients had evidence of previous exposure to an arbovirus, as determined by the presence of flavivirus antibodies using antigens to dengue, St. Louis encephalitis, Powassan and Japanese encephalitis viruses (Table 2). One patient had evidence of acute Powassan virus infection (Table 1: category 1, patient 3; Table 2: case 11). Of these 12 patients, 11 were identified through active surveillance and 1 through passive surveillance.

Interpretation

The goal of the active surveillance was the early detection of WNV infection in patients admitted to hospital with encephalopathy. The criteria for the suspect case definition were deliberately broad to ensure collection of the first serum sample early in the course of illness. The insidious onset and nonspecific findings of encephalitis¹⁷ meant that more serum samples were tested at the acute stage of illness than was ultimately necessary. Many of the patients ultimately had alternative diagnoses (category 3). The frequency of findings suggestive of encephalitis has been reported to be lower at admission than later in the hospital stay: pleocytosis (59% v. 63%), electroencephalogram abnormalities (87% v. 96%) and neuroimaging abnormalities (37% v. 69%).¹⁸ This presents a challenge to any surveil-

Table 2: Classification of patients with positive arboviral serology results

Case*	Hospital LOS, d	Age, yr	Sex	Antibody titre				CSF sample banked?	Met case definition?	Travel history	Diagnosis	Vaccinated against yellow fever or Japanese encephalitis B?
				Acute sample	Convalescent sample							
1	7	45	F	SLE DEN POW	80 80 10	NA		Yes	Yes	None	No encephalopathy	U
2	U	34	M	POW	40	NA		Yes	Yes	Camping in Ontario	Encephalomyelitis	No
3	3	34	F	SLE POW DEN WNV	40 10 20 10	SLE POW DEN WNV	40 10 20 10	Yes	No	New York, July 2000	Aseptic meningitis	U
4	27	80	M	SLE DEN POW	80 80 20	NA		No	Yes	From Jamaica	Encephalitis with GBS	U
5	6	30	F	SLE POW DEN	80 40 80	NA		Yes	Yes	From India	Aseptic meningitis	U
6	5	25	M	SLE POW WNV	20 10 80	SLE POW WNV	20 10 80	Yes	U	From Iran	Meningitis	U
7	7	22	M	SLE POW DEN WNV	640 80 320 160	SLE POW DEN WNV	640 80 320 160	Yes	No	Jamaica, May 2000	U	U
8	10	15	F	SLE WNV	20 10	NA		Yes	No	None	Encephalitis	U
9	U	71	F	WNV	160	NA		Yes	Yes	None	Encephalitis	U
10	4	29	F	SLE POW DEN WNV	320 80 320 160	NA		No	No	From Pakistan	U	"Likely"
11	U	39	M	POW	< 10	POW	40	No	No	None	Meningoencephalomyelitis	U
12§	NA	30	F	SLE POW WNV	80 20 20	SLE POW WNV	80 20 20	No	No	None	Lymphadenopathy	U

Note: LOS = length of stay, CSF = cerebrospinal fluid, SLE = St. Louis encephalitis, DEN = Dengue, POW = Powassan encephalitis, WNV = West Nile virus encephalitis, NA = not applicable, U = unknown, GBS = group B *Streptococcus*.

*Identified through enhanced passive surveillance unless otherwise noted.

†Identified through active surveillance.

lance system for encephalitis and underscores the importance of an accurate discharge diagnosis.

Serum samples were often not obtained during the convalescent phase. Serologic testing of such samples is vital for the confirmation of a WNV diagnosis. Evaluation of any patient with encephalitic symptoms should always include the collection of paired serum samples as well as an attempt to detect a microbial organism through culture or molecular techniques. Such systematic investigation may yield the definite or probable microbial cause in 40% of cases and a possible cause in another 26%.¹⁸ The strength of purported etiologic associations in our study (category 1) is unknown; that is, a presumptive diagnosis of herpes simplex virus encephalitis may actually be caused by enterovirus 71, *Mycoplasma pneumoniae*, Epstein-Barr virus or other agents.¹⁸

The overwhelming majority of the sentinel hospitals complied with the active surveillance protocol. Many cases that did not meet our suspect case definition and were not admitted to hospital were identified passively through the laboratory from the Niagara region following findings of WNV in birds in adjacent counties of New York State. Our surveillance program complemented other surveillance efforts for WNV infection in Ontario in 2000, including investigation of dead birds, sentinel chickens and mosquitoes¹⁹ (C.L.: unpublished data). No WNV activity was demonstrated by any of these surveillance methods.

Although WNV is suspected to have entered Ontario, owing to the discovery of WNV-positive birds found in neighbouring counties of New York State, it was not documented in 2000. WNV should now be considered as established in North America, if not yet endemic.²⁰ WNV infection should be included in the differential diagnosis of infectious encephalopathy originating within North America; the virus may have spread to Florida, eastern Canada, the Caribbean and Mexico with migrating wild bird populations. Should WNV become apparent in Canada, we will have to remain vigilant to reduce the likelihood of outbreaks and minimize human infection. The public has been advised to use judicious mosquito control measures, including limiting outdoor activities between dusk and dawn, wearing long pants and sleeves outdoors when mosquitoes are most active, eliminating sources of standing water on their properties, repairing screens on doors and windows and using insect repellents that contain DEET (*N*-diethyl-meta-toluamide). Nevertheless, even in regions where mosquitoes carry the virus, less than 1% of mosquitoes are infected and, of humans cases, less than 1% will become severely ill. Among those with severe illness, the case-fatality rate is 3%–15% and involves mostly elderly people.

Provincial initiatives in Ontario for the 2001 season included enhanced surveillance of dead birds (especially crows) and human surveillance coordinated through medical officers of health, emphasizing the vital need for the collection of a second serum at 10 to 14 days after presentation in patients with diagnoses of encephalitis. Of 19 patients admitted to hospital during the epidemiologic inves-

tigation of the New York outbreak, 79% were not reported to the department of health. Physician liaison with public health departments is vitally important for the early detection of outbreaks. Just as management of the US epidemic was a tribute to public health organizations and dedicated physicians,²¹ so, too, is the Canadian initiative.

Competing interests: None declared.

Contributors: Lee Ford-Jones prepared the study protocol, supervised the study and prepared the manuscript. Moira Myszak, Beverly Cole and Pam Baker Greene coordinated the study at all sentinel hospital and laboratory sites. Allison McGeer developed the model of sentinel hospital surveillance used in the study. Monika Naus (project director) initiated West Nile virus surveillance, including development of the study protocol, and directed human and other WNV surveillance. Chuck Leber coordinated all surveillance and educational activities and supervised nonhuman surveillance. Margaret Fearon provided laboratory testing and results of passive human surveillance. Prabo Dwight developed the database used during the study. Sheila Artes provided ongoing liaison with the Ministry of Health and Long-term Care during the study and evaluated the database. Colin D'Cunha directed West Nile virus management and all related educational initiatives across Ontario for the medical officers of health and medical specialists. All authors contributed to revising the manuscript.

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Correspondence to: Dr. E. Lee Ford-Jones, Division of Infectious Diseases, Hospital for Sick Children, 555 University Ave., Toronto ON M5G 1X8

Ontario West Nile Virus Working Group:

Members: Chuck Leber (chair), Ontario Ministry of Health and Long-Term Care, Toronto, Ont.; Pam Baker Greene, Beverly Cole, E. Lee Ford-Jones and Moira Myszak, Hospital for Sick Children, Toronto, Ont.; Allison McGeer, Mount Sinai Hospital, Toronto, Ont.; and Sheila Artes, Colin D'Cunha, Prabo Dwight, Margaret Fearon and Monika Naus (project director), Ontario Ministry of Health and Long-Term Care, Toronto, Ont. **Project Team (Advisory Group):** David Alves, Ontario Ministry of Agriculture, Food and Rural Affairs, Toronto, Ont.; Sharon Balsys, Bill Hunter and Kim Murray Perrault, Ontario Ministry of Health and Long-Term Care, Toronto, Ont.; Ian Barker, Doug Campbell and James Heal, University of Guelph, Guelph, Ont.; Howard Barton and Larry Clay, Ontario Ministry of Municipal Affairs and Housing, Toronto, Ont.; Geoff Cutten, Ontario Ministry of the Environment, Toronto, Ont.; Shelley Deeks, City of Toronto Public Health, Toronto, Ont.; Alex Hukowich, Haliburton, Kawartha and Pine Ridge District Health Unit, Port Hope, Ont.; Robbin Lindsay, Health Canada, Ottawa, Ont.; Rick Rosatte and Roxanne St. Martin, Ontario Ministry of Natural Resources, Toronto, Ont.; Nina Szpakowski, Canadian Food Inspection Agency; and Robert Thompson, Wellington, Dufferin and Guelph Health Unit, Guelph, Ont.



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