Anti-WNV IgG was more often detected in participants in the 20- to 29-year age group (odds ratio [OR] 4.23, 95% confidence interval [CI] 1.04-16.02, p = 0.03) and in persons who reported frequent mosquito bites (OR 8.62, 95% CI 0.44-169, p =0.08). IgG-positive persons were equally divided by sex. No significant differences were found between antibody-positive or antibody-negative persons with respect to their profession, place of occupation, current residence, time in current residence, outdoor activities, use of insecticides and repellents, or symptoms related to WNV infection.

No symptoms related to WNV infection were reported by the IgM/IgG-positive participant, who was 31 years of age, was born in the area, worked outdoors, and was frequently bitten by mosquitoes. He also reported travel to Cuba 1 year earlier, but he had not been vaccinated against flavivirus, and serologic test results for dengue were negative.

The other IgG- and neutralizing antibody-positive participant was 45 years of age and was born and works in the area. He had never traveled abroad or been vaccinated against flavivirus. He reported a 4-day fever of unknown origin during the summer 1 or 2 years before the study. He often fishes in the areas and is frequently bitten by mosquitoes.

In conclusion, the study found evidence of recent WNV infections in humans living in the Ebro delta, where previous flavivirus circulation has been suggested by Lozano and Filipe (6). IgG-positive results not confirmed by neutralization could be due to cross-reactive antibodies induced by other flavivirus infections or vaccinations (9,10). The probable WNV infection described was asymptomatic, as occurs in ≈20% of cases. Other WNV infections in the area may have remained undetected, including neuroinvasive cases. Intensified research and surveillance in this area will help determine and refine thresholds for public health interventions.

Acknowledgments

We thank Pedro Fernández-Viladrich for reviewing the manuscript and H. Zeller for providing the antigen used in this study.

Domingo Bofill,* Cristina Domingo,† Neus Cardeñosa,‡ Joan Zaragoza,* Fernando de Ory,† Sofia Minguell,‡ María Paz Sánchez-Seco,† Angela Domínguez,‡ and Antonio Tenorio†

*Hospital de Tortosa Verge de la Cinta, Tarragona, Spain; †Instituto de Salud Carlos III, Madrid, Spain; and ‡Catalan Health Department, Barcelona, Spain

References

- Briese T, Jia XY, Huang C, Grady LJ, Lipkin WI. Identification of a Kunjin/West Nile-like flavivirus in brains of patients with New York encephalitis. Lancet. 1999;354: 1261–2.
- Murgue B, Murri S, Triki H, Deubel V, Zeller HG. West Nile in the Mediterranean basin; 1950–2000. Ann N Y Acad Sci. 2001;951:117–26.
- Del Giudice PD, Schuffenecker I, Vandenbos F, Counillon E, Zeller H. Human West Nile Virus, France. Emerg Infect Dis. 2004;10:1885–6.
- Schuffenecker I, Peyrefitte CN, el Harrak M, Murri S, Leblond A, Zeller HG. West Nile virus in Morocco, 2003. Emerg Infect Dis. 2005;11:306–9.
- Connell J, McKeown P, Garvey P, Cotter S, Conway A, O'Flanagan D, et al. Two linked cases of West Nile virus (WNV) acquired by Irish tourists in the Algarve, Portugal. Eurosurveillance Weekly [serial on the Internet]. 2004 Aug 5 [cited 2006 May 12]. Available from www.eurosurveillance.org/ ew/2004/040805.asp
- Lozano A, Filipe AR. Antibodies against the West Nile virus and other arthropodtransmitted viruses in the Ebro delta region. Rev Esp Salud Publica. 1998;72:245–50.
- Gonzalez MT, Filipe AR. Antibodies to arboviruses in northwestern Spain. Am J Trop Med Hyg. 1977;26:792–7.
- 8. Murgue B, Murri S, Zientara S, Labie J, Durand B, Durand JP, et al. West Nile in France in 2000: the return 38 years later. Emerg Infect Dis. 2001;7:692–6.

- Hogrefe WR, Moore R, Lape-Nixon M, Wagner M, Prince HE. Performance of immunoglobulin G (IgG) and IgM enzymelinked immunosorbent assays using a West Nile virus recombinant antigen (preM/E) for detection of West Nile virus- and other flavivirus-specific antibodies. J Clin Microbiol. 2004;42:4641–8.
- Kuno G. Serodiagnosis of flaviviral infections and vaccinations in humans. Adv Virus Res. 2003;61:3–65.

Address for correspondence: Neus Cardeñosa Marín, Servei de Vigilància Epidemiològica, Departament de Salut, Trav de les Corts 131-159, Pavelló Ave Maria, 08028 Barcelona, Spain; email: neus.cardenosa@gencat.net

Shigellosis and Cryptosporidiosis, Baltimore, Maryland

To the Editor: Floret et al. argue convincingly that natural disasters, including severe floods and windstorms, tend not to result in epidemics of infectious disease (1). This conclusion is consistent with the lack of epidemics of shigellosis and cryptosporidiosis after hurricane rains in Baltimore, Maryland.

Shigellosis and cryptosporidiosis are associated with waterborne and foodborne transmission (2,3). We examined Baltimore shigellosis and cryptosporidiosis incidence to assess whether disease risk was related to temperature or rainfall from January 1, 1998, to December 31, 2004. Maryland FoodNet supplied case data; population estimates were acquired from the Maryland Department of Planning State Data Center; and meteorologic data for Baltimore Washington International airport (≈10 miles from the city center) were obtained from the National

Atmospheric and Oceanic Administration (4).

During the study period, 38 cases of cryptosporidiosis and 943 cases of shigellosis were reported in Baltimore. Temperature was strongly seasonal; precipitation was not. A dry period during 1999 was observed. No seasonal cryptosporidiosis patterns were identifiable. Two outbreaks of shigellosis occurred; in 2000 (≈50 cases) and 2002–2004 (≈870 cases). Sporadic cases of shigellosis were not seasonal.

Two hurricanes resulted in heavy rainfall in Baltimore during the study period (5). Hurricane Floyd inundated the city with rain on September 16, 1999, and on September 19, 2003, Hurricane Isabel produced heavy rains and storm surge in Baltimore (which is located near the northern end of Chesapeake Bay). Approximately 4 other named tropical storms or depressions directly affected Baltimore rainfall during the study. However, collectively, none of these events had distinguishable signatures in the incidence of shigellosis or cryptosporidiosis in this urban environment.

The institutional review boards of the University of Maryland School of Medicine, The George Washington University Medical Center, and the Maryland Department of Health and Mental Hygiene approved this study. Dr Hartley is supported by a National Institutes of Health Career Development Award (K25 AI-58956).

David M. Hartley,* Karl C. Klontz,†‡ Patricia Ryan,§ and J. Glenn Morris Jr*

*University of Maryland School of Medicine, Baltimore, Maryland, USA; †The George Washington University School of Public Health and Health Services, Washington, DC, USA; ‡US Food and Drug Administration, College Park, Maryland, USA; and §Maryland Department of Health and Mental Hygiene, Baltimore, Maryland, USA

References

- Floret N, Viel JF, Mauny F, Hoen B, Piarroux R. Negligible risk for epidemics after geophysical disasters. Emerg Infect Dis. 2006;12:543–8.
- Centers for Disease Control and Prevention. FoodNet surveillance report for 2002. May 2004. [cited 2006 Apr 6]. Available from http://www.cdc.gov/food-net/reports.htm
- 3. Naumova EN, Christodouleas J, Hunter PR, Syed Q. Effect of precipitation on seasonal variability in cryptosporidiosis recorded by the north west England surveillance system in 1990–1999. J Water Health. 2005;3: 185–96.
- National Oceanic and Atmospheric Administration. Federal climate complex global surface summary of day data.
 Version 6. National Oceanic and Atmospheric Administration National Climatic Data Center. [cited 2006 Feb 18].
 Available from ftp://ftp.ncdc.noaa.gov/ pub/data/globalsod/readme.txt
- National Oceanic and Atmospheric Administration. Hurricane history. [cited 2006 Apr 6]. Available from http://www. nhc.noaa.gov/HAW2/english/history.shtml

Address for correspondence: David M. Hartley, Department of Epidemiology and Preventive Medicine, University of Maryland School of Medicine, 660 West Redwood St, Baltimore, MD 21201, USA; email: dhartley@epi.umaryland.edu

Human Hantavirus Infection, Brazilian Amazon

To the Editor: Since hantavirus pulmonary syndrome (HPS) caused by Sin Nombre virus (SNV) was identified in the southwestern United States in 1993, cases have been diagnosed in many Latin American countries, and an increasing number of hantaviruses and their rodent reservoirs have been reported (1). The first evidence of hantavirus circulation in the western Brazilian Amazon region

was documented in 1991 (2). Vasconcelos et al., by using antigens from the Old World hantavirus, found evidence of hantavirus antibodies in 45.2% of serum samples acquired from contacts of patients who died with undiagnosed hemorrhagic fever in Manaus.

The first human cases of symptomatic infection by hantaviruses were reported from Brazil in 1993, in Juquitiba (São Paulo State). HPS developed in 3 young brothers, who lived in a forested region along the Atlantic Coast, after they had cleared trees on their land, and 2 of them died. These patients were living in poor conditions, without appropriate storage spaces for human food or for animal feed, and their dwelling was constantly invaded by wild rodents who were looking for food (3). Since then, many other HPS cases have been reported, especially from the southern and southeastern regions of Brazil where agricultural activities are prominent; the mean case-fatality ratio is 48% (3). In the Brazilian Amazon, HPS has been frequently reported in Mato Grosso and sporadically in Maranhão and Pará states, which indicates an endemic circulation of hantaviruses (4,5)

We report here the first human cases of HPS in the state of Amazonas in the western part of the Brazilian Amazon. All 4 patients belonged to the same family cluster and came from a rural area near the town of Itacoatiara, on the edge of an important industrial waterway for soybean transport (the Itacoatiara soybean terminal). This family (patients 1, 2, and 3) had cleared a forested area on their farm and killed many rodents found in the bases of trees and near the house from May 25 to June 5, 2004. They also reported that wild rodents were inside their house.

All serologic tests were performed in the Arbovirology and Hemorrhagic Fever Department, at the Evandro Chagas Institute (Pará, Brazil), with