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Zika Virus Disease in Travelers Returning to the United States, 2010–2014

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Abstract. Zika virus is an emerging mosquito-borne flavivirus that typically causes a mild febrile illness with rash, arthralgia, or conjunctivitis. Zika virus has recently caused large outbreaks of disease in southeast Asia, Pacific Ocean Islands, and the Americas. We identified all positive Zika virus test results performed at U.S. Centers for Disease Control and Prevention from 2010 to 2014. For persons with test results indicating a recent infection with Zika virus, we collected information on demographics, travel history, and clinical features. Eleven Zika virus disease cases were identified among travelers returning to the United States. The median age of cases was 50 years (range: 29–74 years) and six (55%) were male. Nine (82%) cases had their illness onset from January to April. All cases reported a travel history to islands in the Pacific Ocean during the days preceding illness onset, and all cases were potentially viremic while in the United States. Public health prevention messages about decreasing mosquito exposure, preventing sexual exposure, and preventing infection in pregnant women should be targeted to individuals traveling to or living in areas with Zika virus activity. Health-care providers and public health officials should be educated about the recognition, diagnosis, and prevention of Zika virus disease.

INTRODUCTION

Zika virus is an emerging mosquito-borne flavivirus that is transmitted to humans by the same *Aedes* species vectors that transmit dengue and chikungunya viruses.[1,2] During outbreaks, humans are the primary amplifying host for Zika virus.[3,4] An estimated 80% of people infected with Zika virus are asymptomatic.[5] Symptomatic disease is generally mild and characterized by acute onset of fever, arthralgia, rash, or conjunctival hyperemia.[5] Symptoms usually last from several days to 2 weeks. Mortality is rare, and severe disease is uncommon but includes temporally associated Guillain–Barré syndrome, as well as microcephaly and intracranial calcifications in infants born to mothers infected with Zika virus.[6–9] There is no vaccine to prevent Zika virus infection, and treatment consists of supportive care.

Zika virus RNA may be detected by reverse transcriptase polymerase chain reaction (RT-PCR) on serum collected within the first week after onset of symptoms.[10] Virus-specific immunoglobulin M (IgM) and neutralizing antibodies typically develop toward the end of the first week of illness; however, cross-reaction with related flaviviruses (e.g., dengue and yellow fever viruses) is common. Virus-specific cross-neutralization test can be used to discriminate between cross-reacting antibodies in primary flavivirus infections. However, neutralizing antibodies may still yield cross-reactive results in persons who were previously infected or vaccinated against a related flavivirus (i.e., secondary flavivirus infection).[10]

Zika virus was first identified in Uganda in 1947.[11] Prior to 2007, only sporadic human disease cases were reported from countries in Africa and Asia. In 2007, the first documented Zika virus outbreak was reported in Yap State, Federated States of Micronesia with an estimated 73% of the population being infected.[5] In subsequent years, outbreaks were identified in southeast Asia and the western Pacific.[3,12–14] In May 2015, Zika virus was identified for the first time in the Americas with large outbreaks reported in Brazil. By the end of 2015, local transmission had been identified in 12 other countries or territories in the region, including Puerto Rico.[15–17] The virus will likely spread to other areas in the Americas but, to date, local mosquito-borne transmission has not been identified in the continental United States. We reviewed the epidemiology and clinical features of travel-associated Zika virus disease cases identified in the United States from 2010 to 2014.

METHODS

Case finding. We identified Zika virus disease cases with laboratory testing performed at the U.S. Centers for Disease Control and Prevention (CDC) Arboviral Diagnostic Laboratory from 2010 to 2014. Since 2010, CDC has performed Zika virus testing on specimens received for arboviral disease testing from persons with recent travel to Africa and persons with a clinically compatible illness and travel to an area experiencing a Zika virus outbreak. In late 2013, routine Zika virus testing was expanded to include all persons with a sample submitted to CDC Arboviral Diagnostic Laboratory for arboviral disease testing and who had recent travel to Africa, southeast Asia, or Pacific Ocean Islands unless testing for a specific arboviral etiology other than Zika virus was requested by the submitter. During this period, approximately 160 specimens were submitted for arboviral disease testing and had Zika virus disease testing performed.

Diagnostic testing included RT-PCR, IgM enzyme-linked immunosorbent assay (ELISA), and/or plaque-reduction neutralization test (PRNT) with a cutoff value of 90% for Zika, dengue 1–4, and chikungunya viruses. For patients with a history of yellow fever vaccination, yellow fever virus–specific IgM ELISA and PRNT were also performed.

Case definition. We defined a case of Zika virus disease as a patient with the following laboratory findings in serum: 1) Zika virus RNA detected by RT-PCR or 2) anti-Zika virus IgM antibodies detected by ELISA with ≥ 4-fold higher PRNT titer against Zika virus compared with dengue virus.[5,10,18,19]

Data collection and analysis. For all Zika virus disease cases, we collected information on sex, age, state of residence, date of illness onset, date of specimen collection, dates of

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travel, travel destination, and clinical symptoms and outcome. Data were obtained by the state health department of residence of Zika virus disease cases. We analyzed the data using Excel version 2010 (Microsoft, Redmond, WA). Continuous variables are presented as medians and ranges. Categorical and dichotomous variables are presented as counts and frequencies.

RESULTS

We identified 11 Zika virus disease cases in U.S. travelers from 2010 to 2014. All cases had their illness onset between December 2013 and April 2014 (Table 1). The median age of the cases was 50 years (range: 29–74 years), and six (55%) were male. Case patients were residents of seven states, including California ($N = 4$), Oregon ($N = 2$), and one each from Hawaii, Kentucky, Minnesota, New York, and Texas. All of the cases reported recent travel to islands in the Pacific Ocean, including French Polynesia ($N = 6$), the Cook Islands ($N = 4$), and Easter Island ($N = 1$). Case patients reported becoming ill a median of 0 days before returning home (range: 7 days before to 8 days after), including five cases who became ill while still at their travel destination, two with onset on the day of return, and four who became ill after returning to the United States.

Among cases with available clinical data, all (10 of 10) patients reported having a rash including maculopapular or diffuse in four and unknown type in six (Table 2). Other commonly reported symptoms included fever (9/10), myalgia (8/10), headache (6/9), arthralgia (7/11), and conjunctivitis (5/9). Gastrointestinal symptoms (e.g., nausea or diarrhea) were less commonly reported (3/10). Of nine patients with known hospitalization status, none were hospitalized. There were no deaths.

Zika virus RT-PCR was performed on acute serum specimens from six patients (Table 3). These specimens were collected at a median of 6 days (range: 3–7 days) after illness onset and were all negative for Zika virus RNA. All 11 patients had evidence of Zika virus IgM antibodies with ≥ 4-fold higher neutralizing antibody titer against Zika virus compared with dengue virus. In addition, four patients also had acute and convalescent serum specimens available for testing, and all showed a ≥ 4-fold rise in Zika virus neutralizing antibody titers; none of these case patients had a ≥ 4-fold rise in dengue virus neutralizing antibody titers.

There were 12 specimens submitted from 10 individuals that were tested for both dengue and Zika virus IgM antibody by ELISA (Table 3). Of these, six (50%) were positive for IgM antibodies against both Zika and dengue viruses, five (42%) were positive for Zika virus IgM antibodies and negative or equivocal for dengue virus IgM antibodies, and one (8%) was equivocal for Zika virus IgM antibodies and negative for IgM antibodies against dengue virus. Two case patients who were previously vaccinated against yellow fever had IgM antibodies against Zika, dengue, and yellow fever viruses; their Zika virus neutralizing antibody titers were ≥ 4-fold higher than the titers against dengue but not yellow fever virus. Ten cases had chikungunya virus RT-PCR or IgM antibody testing performed; all were negative.

TABLE 1
Characteristics of Zika virus disease cases identified in travelers returning to the United States, 2010–2014

	Zika virus disease cases ($N = 11$)
	n (%)
Male sex	6 (55)
Age group	
< 20	0 (0)
20–39	3 (27)
40–59	5 (45)
≥ 60	3 (27)
Country where infection acquired	
French Polynesia	6 (55)
Cook Islands	4 (36)
Easter Island	1 (9)
Month of illness onset	
January–April	9 (82)
May–August	0 (0)
September–December	2 (18)

TABLE 2
Clinical symptoms of Zika virus disease cases identified in travelers returning to the United States, 2010–2014

Symptom	*n*/*N* (%)
Rash	10/10 (100)
Fever	9/10 (90)
Myalgia	8/10 (80)
Headache	6/9 (67)
Arthralgia	7/11 (64)
Conjunctivitis	5/9 (55)
Gastrointestinal symptoms	3/10 (30)

DISCUSSION

We identified 11 cases of travel-associated Zika virus disease in the United States occurring from 2010 to 2014 when samples submitted to CDC from travelers for arboviral disease testing have been more routinely tested for Zika virus infections. All cases occurred in 2013–2014 and were identified among travelers returning from areas experiencing known outbreaks of the disease. Many of the travelers were ill and potentially viremic after they had returned to the United States, representing a risk for introduction and possible local mosquito-borne transmission of Zika virus.

Prior to 2007, travel-related cases of Zika virus disease had not been readily recognized or reported. This was likely due to a combination of limited testing capacity and infrequent virus activity in remote locations. One of the first documented travel-related case of Zika virus disease was in a traveler visiting Yap during the 2007 outbreak. This case was diagnosed by IgM neutralizing antibody in the serum after arrival in the United States.[5] Since 2007, at least 15 travel-related cases have been reported, including four cases reported among U.S. travelers.[20–30] Three of the previous cases reported in the United States occurred during 2008. All three cases had ≥ 4-fold increase in Zika virus titers between their acute and convalescent samples tested with PRNT and ≥ 4-fold higher titer against Zika virus compared with dengue virus. However, two of the cases who had recently traveled to Senegal and had previous dose of yellow fever vaccine also had an increase in yellow fever virus titers by PRNT, which were not ≥ 4-fold different from their Zika virus titers. One case had not traveled and was hypothesized to have become infected from one of the two other cases

TABLE 3
Select diagnostic test results for Zika virus disease cases identified in travelers returning to the United States, 2010–2014

	Initial serum specimen						Second serum specimen				
Case no.	Days from illness onset to specimen one	Zika RT-PCR	Zika IgM	Zika PRNT	Dengue IgM	Dengue PRNT*	Days from illness onset to specimen two	Zika IgM	Zika PRNT	Dengue IgM	Dengue PRNT*
1	30	–†	Positive	5,120	Negative	80	NA	–	–	–	–
2	1	–	Positive	20	Equivocal	20	18	Positive	2,560	Positive	40
3	3	Negative	Equivocal	< 10	Negative	< 10	24	Positive	2,560	–	< 10
4	7	–	Positive	80	Positive	< 10	NA	–	–	–	–
5	5	Negative	Positive	10	Negative	–	NA	–	–	–	–
6‡	6	Negative	Positive	640	Positive	40	NA	–	–	–	–
7	7	Negative	Positive	160	Equivocal	20	NA	–	–	–	–
8‡	11	–	Positive	80	Positive	80	32	Positive	2,560	Positive	160
9	7	Negative	Positive	80	–	< 10	NA	–	–	–	–
10	11	–	Positive	320	Negative	< 10	NA	–	–	–	–
11	4	Negative	–	–	Positive	< 10	25	Positive	1,280	Positive	< 10

IgM = immunoglobulin M; NA = not applicable; PRNT = plaque-reduction neutralization test; RT-PCR = reverse transcriptase polymerase chain reaction.
*Represents the highest titer from PRNTs performed for each of the four serotypes of dengue virus.
†Testing not performed, typically due to limited sample quantity.
‡Cases reported a history of prior yellow fever vaccination.

through sexual contact. These three cases are not included in this article, as they have been previously described.[28] A fourth is a single case description from New York in December 2013 and is included here among the 11 cases.[29]

The cases presented here have similar clinical findings to previously reported travel-associated cases of Zika virus disease from other countries.[20–27,30] Most had fever and rash, and about half had conjunctivitis. The most frequently reported symptoms in our cohort were also similar to those reported for locally transmitted cases identified in Yap, though our cases had a higher proportion of those reporting myalgia and headache.[5] Differences in the rates of symptoms, however, might be due to low number of disease cases. The low numbers also limited our ability to detect any cases with rare manifestations such as Guillain–Barré syndrome.

Since the most common symptoms of Zika virus disease (e.g., fever, rash, arthralgia, and myalgia) are relatively nonspecific, the differential diagnosis is relatively broad and can include other mosquito-borne diseases such as chikungunya and dengue. Preliminary diagnosis should be based on clinical findings and epidemiologic characteristics, including travel history and other activities. Diagnostic testing includes RT-PCR and antibody detection. Among Zika virus disease cases identified in this study, 50% were also dengue IgM positive suggesting that cross-reactivity is relatively common. Therefore, Zika or dengue virus disease cases may be misdiagnosed or difficult to differentiate in areas where both viruses occur.

To date, none of the documented travel-related cases in the United States have led to subsequent local mosquito-borne transmission of the virus; however, one previously reported case from 2008 and six recently reported cases were related to possible sexual transmission.[28,31] All the cases identified in this study had illness onset from December through April. These months typically represent the times of lowest mosquito activity in the United States and thus likely decreased the risk of a travel-associated case leading to subsequent local mosquito-borne transmission. The accessibility of air travel, abundance of appropriate mosquito vectors in the Americas, including the United States, and the lack of population immunity in region have allowed for the spread of Zika virus throughout the Americas similar to what occurred with chikungunya virus starting in late 2013.[32] Chikungunya and Zika viruses have similar transmission cycles during outbreaks, using the same mosquito species to infect humans who amplify and typically spread the virus from one location to another. Within the first year of the introduction of chikungunya virus into the Americas, over 1.1 million suspected disease cases were reported, including more than 2,700 travel-associated confirmed cases and 11 local confirmed disease cases in the United States.[33,34]

Our study has several limitations. The 11 cases identified for this report all sought medical care and had a sample collected and sent to CDC for testing; they might not be representative and likely underestimate the number of travel-associated Zika virus disease cases. In 2013, CDC initiated more systematic Zika testing on specimens received from returning travelers. This might have influenced when cases were identified and their characteristics. Clinical data were not available on all cases; this and the low number of cases might further impact the representativeness of the data. Finally, we used 7 days after illness onset to denote a potential period of viremia and risk for local transmission for individuals who developed symptoms while abroad. This estimation is based on limited data, which suggest that Zika virus has been found in blood of infected individuals as late as 11 days after illness onset.[10] Further validation is needed to determine when an infected person is viremic and capable of leading to local transmission in at risk areas.

These travel-associated cases of Zika virus disease highlight the need for travelers to consult with travel health practitioners or country-specific travelers' health webpages (e.g., http://wwwnc.cdc.gov/travel/destinations/list/) prior to travel. Travelers should be educated about the symptoms of Zika virus and other mosquito-borne diseases and measures to prevent mosquito bites. Zika virus disease should be considered in the differential diagnosis in travelers with acute fever or rash after returning from areas where Zika virus is known to be circulating or persons with a direct epidemiologic link to a person with laboratory evidence of recent Zika virus infection (e.g., vertical transmission from other to baby or sexual contact). Clinicians are encouraged to report suspected Zika virus disease cases to state or local health departments to obtain testing and to reduce the risk of local transmission. Finally, additional data are needed to determine the rates of occurrence of rare or uncommon manifestations and death following Zika virus infection.

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REFERENCES

1. Marchette N, Garcia R, Rudnick A, 1969. Isolation of Zika virus from *Aedes aegypti* mosquitoes in Malaysia. *Am J Trop Med Hyg 18:* 411–415.
2. Wong P, Li M, Chong C-S, Ng L-C, Tan C-H, 2013. *Aedes* (*Stegomyia*) *albopictus* (Skuse): a potential vector of Zika virus in Singapore. *PLoS Negl Trop Dis 7:* e2348.
3. Musso D, Nilles EJ, Cao-Lormeau VM, 2014. Rapid spread of emerging Zika virus in the Pacific area. *Clin Microbiol Infect 20:* O595–O596.
4. Hayes EB, 2009. Zika virus outside Africa. *Emerg Infect Dis 15:* 1347–1350.
5. Duffy MR, Chen T-H, Hancock WT, Powers AM, Kool JL, Lanciotti RS, Pretrick M, Marfel M, Holzbauer S, Dubray C, Guillaumot L, Griggs A, Bel M, Lambert AJ, Laven J, Kosoy O, Panella A, Biggerstaff BJ, Fischer M, Hayes EB, 2009. Zika virus outbreak on Yap Island, Federated States of Micronesia. *N Engl J Med 360:* 2536–2543.
6. European Centre for Disease Prevention and Control, 2015. *Rapid Risk Assessment: Microcephaly in Brazil Potentially Linked to the Zika Virus Epidemic*. Available at: http://ecdc.europa.eu/en/publications/Publications/zika-microcephaly-Brazil-rapid-risk-assessment-Nov-2015.pdf. Accessed December 15, 2015.
7. Oehler E, Watrin L, Larre P, Leparc-Goffart I, Lastere S, Valour F, Baudouin L, Mallet H, Musso D, Ghawche F, 2014. Zika virus infection complicated by Guillain-Barré syndrome—case report, French Polynesia, December 2013. *Euro Surveill 19:* pii 20720.
8. European Centre for Disease Prevention and Control, 2014. *Rapid Risk Assessment: Zika Virus Infection Outbreak, French Polynesia*. Available at: http://ecdc.europa.eu/en/publications/Publications/Zika-virus-French-Polynesia-rapid-risk-assessment.pdf. Accessed December 15, 2015.
9. Brasil P, Pereira JP Jr, Raja Gabaglia C, Damasceno L, Wakimoto M, Ribeiro Nogueira RM, Carvalho de Sequeira P, Machado Siqueira A, Abreu de Carvalho LM, Cotrim da Cunha D, Calvet GA, Neves ES, Moreira ME, Rodrigues Baião AE, Nassar de Carvalho PR, Janzen C, Valderramos SG, Cherry JD, Bispo de Filippis AM, Nielsen-Saines K, 2016. Zika virus infection in pregnant women in Rio de Janeiro—preliminary report. *N Engl J Med.* doi:10.1056/NEJMoa1602412.
10. Lanciotti RS, Kosoy OL, Laven JJ, Velez JO, Lambert AJ, Johnson AJ, Stanfield SM, Duffy MR, 2008. Genetic and serologic properties of Zika virus associated with an epidemic, Yap State, Micronesia, 2007. *Emerg Infect Dis 14:* 1232–1239.
11. Dick G, Ktichen SF, Haddow AJ, 1952. Zika virus (I). Isolations and serological specificity. *Trans R Soc Trop Med Hyg 46:* 509–520.
12. Heang V, Yasuda CY, Sovann L, Haddow AD, da Rosa APT, Tesh RB, Kasper MR, 2012. Zika virus infection, Cambodia, 2010. *Emerg Infect Dis 18:* 349–351.
13. Cao-Lormeau V-M, Roche C, Teissier A, Robin E, Berry A-L, Mallet H-P, Sall AA, Musso D, 2014. Zika virus, French Polynesia, South pacific, 2013. *Emerg Infect Dis 20:* 1085–1086.
14. Roth A, Mercier A, Lepers C, Hoy D, Duituturaga S, Benyon E, Guillaumot L, Souarès Y, 2014. Concurrent outbreaks of dengue, chikungunya and Zika virus infections—an unprecedented epidemic wave of mosquito-borne viruses in the Pacific 2012–2014. *Euro Surveill 19:* pii 20929.
15. Zanluca C, de Melo VCA, Mosimann ALP, dos Santos GIV, dos Santos CND, Luz K, 2015. First report of autochthonous transmission of Zika virus in Brazil. *Mem Inst Oswaldo Cruz 110:* 569–572.
16. Rodriguez-Morales AJ, 2015. Zika: the new arbovirus threat for Latin America. *J Infect Dev Ctries 9:* 684–685.
17. Centers for Disease Control and Prevention, 2015. *Zika Virus*. Available at: http://www.cdc.gov/zika/. Accessed December 24, 2015.
18. Beaty BJ, Calisher CH, Shope RE, 1995. Arboviruses. Lennette EH, Lennette DA, Lennette ET, eds. *Diagnostic Procedures for Viral, Rickettsial and Chlamydial Infections*. 7th edition. Washington, DC: American Public Health Association, 189–212.
19. Calisher CH, Karabatsos N, Dalrymple JM, Shope RE, Porterfield JS, Westaway EG, Brandt WE, 1989. Antigenic relationships between flaviviruses as determined by cross-neutralization tests with polyclonoal antisera. *J Gen Virol 70:* 37–43.
20. Zammarchi L, Stella G, Mantella A, Bartolozzi D, Tappe D, Günther S, Oestereich L, Cadar D, Muñoz-Fontela C, Bartoloni A, Schmidt-Chanasit J, 2015. Zika virus infections imported to Italy: clinical, immunological and virological findings, and public health implications. *J Clin Virol 63:* 32–35.
21. Zammarchi L, Tappe D, Fortuna C, Remoli M, Gunther S, Venturi G, Bartoloni A, Schmidt-Chanasit J, 2015. Zika virus infection in a traveller returning to Europe from Brazil, March 2015. *Euro Surveill 20:* pii 21153.
22. Dupont-Rouzeyrol M, O'Connor O, Calvez E, Daures M, John M, Grangeon J-P, Gourinat A-C, 2015. Co-infection with Zika and dengue viruses in 2 patients, New Caledonia, 2014. *Emerg Infect Dis 21:* 381–382.
23. Kwong JC, Druce JD, Leder K, 2013. Zika virus infection acquired during brief travel to Indonesia. *Am J Trop Med Hyg 89:* 516–517.
24. Kutsuna S, Kato Y, Takasaki T, Moi M, Kotaki A, Uemura H, Matono T, Fujiya Y, Mawatari M, Takeshita N, Hayakawa K, Kanagawa S, Ohmagari N, 2014. Two cases of Zika fever imported from French Polynesia to Japan, December 2013 to January 2014. *Euro Surveill 19:* 1–5.
25. Tappe D, Rissland J, Gabriel M, Emmerich P, Gunther S, Held G, Smola S, Schmidt-Chanasit J, 2014. First case of laboratory-confirmed Zika virus infection imported into Europe, November 2013. *Euro Surveill 19:* pii 20685.
26. Wæhre T, Maagard A, Tappe D, Cadar D, Schmidt-Chanasit J, 2014. Zika virus infection after travel to Tahiti, December 2013. *Emerg Infect Dis 20:* 1412–1414.
27. Fonseca K, Meatherall B, Zarra D, Drebot M, MacDonald J, Pabbaraju K, Wong S, Webster P, Lindsay R, Tellier R, 2014. First case of Zika virus infection in a returning Canadian traveler. *Am J Trop Med Hyg 91:* 1035–1038.
28. Foy BD, Kobylinski KC, Foy JLC, Blitvich BJ, da Rosa AT, Haddow AD, Lanciotti RS, Tesh RB, 2011. Probable non-vector-borne transmission of Zika virus, Colorado, USA. *Emerg Infect Dis 17:* 880–882.
29. Summers DJ, Acosta RW, Acosta AM, 2015. Zika virus in an American recreational traveler. *J Travel Med 22:* 338–340.
30. Korhonen E, Huhtamo E, Smura T, Kallio-Kokko H, Raassina M, Vapalahti O, 2015. Zika virus infection in a traveller returning from the Maldives. *Euro Surveill 21:* 30107.
31. Hills SL, Russell K, Hennessey M, Williams C, Oster AM, Fischer M, Mead P, 2016. Transmission of Zika virus through sexual contact with travelers to areas of ongoing transmission—continental United States, 2016. *MMWR 65:* 215–216.
32. Staples JE, Fischer M, 2014. Chikungunya virus in the Americas—what a vectorborne pathogen can do. *N Engl J Med 371:* 887–889.
33. Pan American Health Organization, 2015. *Chikungunya: PAHO/WHO Data, Maps and Statistics*. Available at: http://www.paho.org/hq/index.php?option=com_topics&view=rdmore&cid=7928&Itemid=40931&lang=en. Accessed September 29, 2015.
34. Centers for Disease Control and Prevention, 2015. *Chikungunya Virus: 2014 Final Data for the United States*. Available at: http://www.cdc.gov/chikungunya/geo/united-states-2014.html. Accessed September 29, 2015.