nos. MG808405–MG808410). BLAST (https://blast.ncbi.nlm.nih.gov/Blast.cgi) and phylogenetic analyses of the segment 2 sequences showed that the EHDV recently isolated in cattle in Israel belongs to serotype 1 and is closely related to the IbAr22619 strain from Nigeria, with which it shares 95.72%–95.76% identity (Figure).

Retrospective analysis of clinical signs in EHDV-1–infected cattle enabled us to conclude that in many farms EHDV infection was asymptomatic or subclinical; milk-yield reduction, fever, and recumbency were the only prominent clinical signs observed during the outbreak. However, animals with BTV and EHDV co-infections showed more severe clinical signs, including fever, abortion, lameness, subcutaneous emphysema, and death.

During recent years, several new arboviruses have been detected in Israel that were not identified previously. BTV serotype 3 was first identified in 2016 but probably was present in Israel since at least 2013 (*9*), EHDV serotype 6 was identified in 2015 (*6*), EHDV serotype 1 was found in 2016, and Shuni virus was detected in 2014 (*10*). These findings showed that new introductions of arthropodborne viral infections into the Middle East region had occurred. Molecular epidemiologic data indicate the viruses originated in Africa, as ours and other studies (*5*,*6*) have shown. Molecular diagnostics, vector-control strategies, and epidemiologic studies should be implemented in Israel to mitigate potential risk for future outbreaks.

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Ross River Virus Antibody Prevalence, Fiji Islands, 2013–2015

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DOI: https://doi.org/10.3201/eid2504.180694

A unique outbreak of Ross River virus (RRV) infection was reported in Fiji in 1979. In 2013, RRV seroprevalence among residents was 46.5% (362/778). Of the residents who were seronegative in 2013 and retested in 2015, 10.9% (21/192) had seroconverted to RRV, suggesting ongoing endemic circulation of RRV in Fiji.

Ross River virus (RRV) is an *Alphavirus* of the family *Togaviridae* and is transmitted to humans by *Aedes* and *Culex* mosquitoes (*1*). Marsupials are considered important reservoirs of RRV (*1*). Clinical manifestations of RRV infections include fever, rash, and arthralgia. RRV is endemic to Australia, where it causes ≈5,000 cases of epidemic polyarthritis annually (*1*). Outbreaks of RRV infection were reported during 1979–1980 in Fiji, the Cook Islands, American Samoa, New Caledonia, and Wallis and Futuna (*1–3*). Subsequently, RRV infections were detected in travelers returning from Fiji during 1997–2009 (*3*) and in patients with suspected dengue in Fiji in 2005 (*4*).

The Republic of the Fiji Islands comprises 322 islands distributed among 4 administrative divisions (Central, Western, Eastern, and Northern) and has a population of ≈830,000. Apart from RRV, the 4 serotypes of dengue virus were the only mosquitoborne viruses known to circulate in Fiji until the recent emergence of Zika and chikungunya viruses (*5*). We report evidence of endemic RRV circulation in Fiji on the basis of serologic analysis of blood samples collected in 2013 and 2015.

Our study included 778 participants recruited during September–November 2013 from the Central, Western, and Northern divisions for a community-based serosurvey for leptospirosis and typhoid (*6*). Among the residents from the Central division who had participated in the 2013 survey, 333 had blood drawn again during October–November 2015, including 311 whose serum sample collected in 2013 was available for testing. We tested all blood samples for RRV IgG by using a recombinant antigen-based microsphere immunoassay (*7*). We analyzed the data with GraphPad Prism 6.03 using the Fisher or χ^2 test. We considered p values <0.05 to be significant.

The prevalence of RRV antibodies among participants in 2013 was 46.5% and was lower in the Central (38.1%) than the Western (58.6%; p<0.0001) and Northern (55.9%; p = 0.0108) divisions (Table). The prevalence of RRV antibodies among the participants sampled in the Central division in 2015 (37.2%) was similar to results from this division in 2013 (38.1%). In 2013, a total of 37.4% of the participants born after 1982 (postoutbreak) had RRV antibodies, and this rate in 2015 (26.9%) was not significantly different (p = 0.0685). The prevalence of RRV antibodies increased with age (p<0.0001 in 2013, p = 0.0020 in 2015) and was higher in rural than in urban (p<0.0001 in 2013, p = 0.0197 in 2015) and periurban areas (p = 0.0060 in 2013). No difference by sex was observed. Among the 311 participants with available serum

Table. Prevalence of Ross River virus antibodies in a representative subset of the population of Fiji sampled during September–November 2013 (n = 778) and October–November 2015 (n = 333)*

Variable	No. seropositive/mo. tested (% [95% CI])	
	2013	2015
Birth year		
<1982	197/336 (58.6 [53.4–63.9])	68/144 (47.2 [39.6–55.7])
≥1982	165/441 (37.4 [32.9–42.1])	56/189 (29.6 [23.2–36.7])
1982–1990	58/117 (49.6 [41.1–58.9])	17/38 (44.7 [31.4–61.4)
1991–2000	66/146 (45.2 [37.7–53.6])	20/66 (30.3 [21.3–42.9])
2001–2010	40/170 (23.5 [18.1–30.7])	19/83 (22.9 [15.8–33.5])
>2011	1/8 (12.5 [0.3–52.7])	0/2 (0.0 [0.0–84.2])
Sex		
F	195/423 (46.1 [41.5–51.0])	73/190 (38.4 [32.1–45.8])
M	167/354 (47.5 [42.2–52.5])	51/143 (35.7 [28.6–44.1])
Division		
Central	172/451 (38.1 [33.9–42.8])	124/333 (37.2 [32.4–42.7])
Northern	33/59 (55.9 [44.1–68.7])	ND
Western	157/268 (58.6 [52.8–64.5])	ND
Zone		
Rural	189/344 (54.9 [49.8–60.3])	52/113 (46.0 [37.5–55.6])
Periurban	55/135 (40.7 [33.2–49.5])	27/77 (35.1 [26.0–46.8])
Urban	117/298 (39.3 [34.1–45.1])	45/143 (31.5 [24.8–39.8])
Total	362/778† (46.5 [43.1–50.1])	124/333 (37.2 [32.4–42.7])

*ND, no data (participants were not recruited from the Northern and Western divisions in 2015).
†For 1 participant, information about age and sex were not available; for another participant, information about the zone of residence was not available.

samples collected in both 2013 and 2015, a total of 21 (10.9%) of the 192 participants who had no detectable RRV antibodies in 2013 had seroconverted to RRV by 2015 (data not shown).

A serosurvey conducted after the RRV outbreak in Fiji in 1979 detected RRV antibodies in 92% of the participants from the Western division (*2*). In our study, which was conducted in 2013, RRV antibody prevalence in the Western, Central, and Northern divisions ranged from 38.1% to 58.6%, and 37.4% of persons born after 1982 had RRV antibodies, suggesting that RRV circulated in all 3 divisions after the 1979 outbreak. The report of RRV infection in travelers or inhabitants from Fiji during 1997–2009 (*3*,*4*), the observations that 10.9% of the seronegative participants in our study seroconverted to RRV during 2013–2015, and the increase in the prevalence of RRV antibodies with age, strongly suggest endemic RRV transmission in Fiji.

The finding that RRV seroprevalence was higher in rural than in urban and periurban environments suggests increased transmission risks in the rural areas, potentially because of higher-risk occupations of rural residents (including farming and outdoor work), greater exposure related to rural housing or other environmental factors, greater animal reservoir density, the possibility that nondomestic mosquito species in Fiji such as *Aedes vigilax*, *Ae. polynesiensis*, *Ae. pseudoscutellaris*, *Ae. albopictus*, and *Culex annulirostris* might be more competent vectors of RRV than peridomestic mosquito species such as *Ae. aegypti* (*8*).

Serosurveys conducted in American Samoa in 2010 (*1*), in French Polynesia during 2011–2013 (*9*) and 2014–2015 (*7*), and our study in Fiji during 2013–2015 suggest that endemic circulation of RRV in the Pacific region continued, or recommenced, after 1979–1980. These data provide further evidence for endemic transmission of RRV in areas where marsupials are absent (*10*). Because of extensive travel between Australia and the Pacific Islands, it is plausible that RRV is repeatedly seeded into the Pacific region. Whether this plays an important role in perpetuating local transmission in the Pacific Islands is unknown. As previously illustrated with Zika and chikungunya viruses, a risk exists for emerging arboviruses to be imported from the Pacific to other parts of the world, and RRV could be the next unexpected threat.

This work was part of ISID-Pacific and R-ZERO Pacific programs funded by the French Ministry for Europe and Foreign Affairs (Pacific Funds nos. 06314-09/04/14, 12115-02/09/15, 03016-20/05/16, and 04917-19/07/17). The study also received support from the Embassy of France in the Republic of the Fiji Islands. The study was supported by the French Government's Investissement d'Avenir Program (Labex IBEID no. ANR-10-LABX-62-IBEID). C.L.L. was supported by an Australian National Health and Medical Research Council Fellowship (no. 1109035). Fieldwork in 2013 was funded by the World Health Organization Western Pacific Region and by the Chadwick Trust. C.H.W. was supported by the UK Medical Research Council (grant no. MR/J003999/1). A.J.K. was supported by a Sir Henry Dale Fellowship, jointly funded by the Wellcome Trust and the Royal Society (grant no. 206250/Z/17/Z).

The study was approved by the Fiji National Health Research Ethics Review Committee (FNRER/no. 2015.114.NW and FNRER/no. 2015.45.MC), the University of the South Pacific (FSTER/2015/10/Research Proposal Approval), and the London School of Hygiene and Tropical Medicine Observational Research Ethics Committee (approval nos. 6344 and 10207).

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Malignant *Aspergillus flavus* Otitis Externa with Jugular Thrombosis

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DOI: https://doi.org/10.3201/eid2504.180710

We report a case of malignant otitis externa with jugular vein thrombosis caused by *Aspergillus flavus*. Magnetic resonance imaging revealed an unusual ink smudge pattern deep in a cervical abscess. The pattern was consistent with mycetoma and may be important for diagnosing these life-threatening infections.

A 73-year-old male patient sought care from the otorhinolaryngology department at University Hospital, Marseille, France. He had a 5-month history of malignant otitis externa (MOE), which was worsening despite 4 months of treatment with intravenous ceftazidime, oral ciprofloxacin, and topical neomycin, polymyxin B, dexamethasone, and thiomersal combination. The patient had a history of high blood pressure, treated with perindopril and nicardipine, and diabetes mellitus, inadequately controlled (hemoglobin A1c 7.7%) with metformin and sitagliptin.

The patient was admitted, and otoscopic examination found otorrhea, inflammation, and stenosis of the right external auditory canal; we could not see the tympanic membrane. Examination of the cranial nerve was normal. Pure-tone audiogram showed a right mixed hearing loss with air-bone gap at 15 dB and symmetric bone curve by presbycusis. Laboratory testing showed elevated erythrocyte sedimentation level (42 mm at 1 h, 82 mm at 2 h) and leukocytosis (11 g/L); C-reactive protein results were within reference range. A computed tomography (CT) scan of the head showed thickening of the ear skin; focal tympanal bone osteolysis; partial right mastoid air cells and middle-ear cavity opacification; and osteolysis of the occipital, styloid, and mastoid bones consistent with MOE (Appendix Figure, https://wwwnc.cdc.gov/EID/article/25/4/18-0710-App1.pdf). Magnetic resonance imaging (MRI) with contrast media confirmed skull base osteomyelitis, evidenced by bone lysis and marrow enhancement of the clivus (Figure, panels A–C). Both MRI and CT showed a right jugular vein thrombosis and cellulitis and abscess in the carotid and perivertebral spaces. Abscess content had an unusual aspect: T2-weighted imaging signal void foci surrounded by a hypersignal rim.

We treated the right jugular vein thrombosis with enoxaparin. The patient underwent surgical debridement with facial nerve monitoring; we collected transmastoid biopsy samples and pus for microbiological analysis and inserted a transtympanic aerator. Direct microscopic examination of the samples showed hyaline septate hyphae consistent with hyalohyphomycosis. Biopsy samples grew 2 bacteria, *Corynebacterium striatum* and *Enterococcus faecalis,* and 1 filamentous fungus, *Aspergillus flavus*, that we identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (Microflex LT, https://www.bruker.com) against an in-house database described by Normand et al. (*1*). Etest antifungal susceptibility testing (bioMérieux, https://www.biomerieux.com) showed that the *A. flavus* strain was sensitive to voriconazole (MIC 0.380 mg/L) and resistant to amphotericin B (MIC 12 mg/L). We stopped administration of auricular drops, continued intravenous ceftazidime (1.5 g/d) and oral ciprofloxacin (1.5 g/d), and started voriconazole therapy (6 mg/kg/12 h intravenously, followed by 400 mg/d orally). Otalgia, otorrhea, and inflammatory external auditory canal symptoms were relieved, and the patient recovered after 6 weeks. No further follow-up was available.

Fungi cause ≈10% of MOE (*2*). The 3 leading species, by decreasing frequency, are *A. fumigatus*, *A. flavus*, and *A. niger* (*3*). *A. flavus* is more frequently involved in MOE than is *A. niger* (*3*,*4*).

Jugular vein thrombosis (JVT) was previously reported in MOE (*5*) and other conditions such as Lemierre syndrome, invasive fungal infection, or any inflammatory process including otitis media. Various pathogens can cause JVT, especially *Fusobacterium necrophorum* and zygomycetes.