

- Shultz TR, Tapsall JW, White PA, Newton PJ. An invasive isolate of *Neisseria meningitidis* showing decreased susceptibility to quinolones. *Antimicrob Agents Chemother.* 2000;44:1116. DOI: 10.1128/AAC.44.4.1116-1116.2000
- Alcalá B, Salcedo C, de la Fuente L, Arreaza L, Uria MJ, Abad R, et al. *Neisseria meningitidis* showing decreased susceptibility to ciprofloxacin: first report in Spain. *J Antimicrob Chemother.* 2004;53:409. DOI: 10.1093/jac/dkh075
- Corso A, Faccone D, Miranda M, Rodriguez M, Regueira M, Carranza C, et al. Emergence of *Neisseria meningitidis* with decreased susceptibility to ciprofloxacin in Argentina. *J Antimicrob Chemother.* 2005;55:596–7. DOI: 10.1093/jac/dki048
- Singhal S, Purnapatre KP, Kalia V, Dube S, Nair D, Deb M, et al. Ciprofloxacin-resistant *Neisseria meningitidis*, Delhi, India. *Emerg Infect Dis.* 2007;13:1614–6.
- Neisseria multilocus* sequence typing [cited 2009 Jul 31]. Available from <http://neisseria.org/nm/typing/mlstodb>
- Hedberg ST, Fredlund H, Nicolas P, Caugant DA, Olcén P, Unemo M. Antibiotic susceptibility and characteristics of *Neisseria meningitidis* isolates from the African meningitis belt 2000–2006: phenotypic and genotypic perspectives. *Antimicrob Agents Chemother.* 2009;53:1561–6. DOI: 10.1128/AAC.00994-08

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Letters

Letters commenting on recent articles as well as letters reporting cases, outbreaks, or original research are welcome. Letters commenting on articles should contain no more than 300 words and 5 references; they are more likely to be published if submitted within 4 weeks of the original article's publication. Letters reporting cases, outbreaks, or original research should contain no more than 800 words and 10 references. They may have 1 Figure or Table and should not be divided into sections. All letters should contain material not previously published and include a word count.

Imported Chikungunya Virus Strains, Taiwan, 2006–2009

To the Editor: Chikungunya is a reemerging infectious disease that is endemic to Africa and Asia and caused by a mosquito-borne alphavirus in the family *Togaviridae*. Previous phylogenetic studies showed that chikungunya virus (CHIKV) strains were clustered into 3 distinct genotypes separated primarily by location into West African, Central/East/South African, and Asian genotypes (1,2).

Earlier outbreaks in Thailand, Cambodia, Vietnam, Myanmar, the Philippines, Malaysia, Indonesia, Pakistan, and India during 1960–1999 were caused by strains of the Asian genotype (2). However, explosive epidemics in Indian Ocean islands and India since 2005 and the worldwide increase in travel have changed the distribution of CHIKV genotypes. Recent studies have shown that different lineages of CHIKV strains of the Central/East/South African genotype have expanded locally and spread to new areas in Africa, Europe, and Asia and caused epidemics (2–7).

Imported chikungunya cases were identified at airports by active surveillance (fever screening) in Taiwan (3). Among 14,289 febrile patients arriving at Taiwan Taoyuan International Airport from January 2006 through February 2009, a total of 13 were confirmed to have CHIKV infections. One additional chikungunya case was detected at Kaohsiung International Airport among 801 febrile patients from February 2008 through February 2009. These imported cases were introduced from Indonesia (7 cases), Malaysia (4 cases), Singapore (1 case), Bangladesh (1 case), and India (1 case). Real-time quantitative reverse transcription–PCR showed virus titers ranged from $10^{3.6}$ PFU/mL to $10^{6.4}$ PFU/mL for day 1–3 acute-phase

serum samples from these patients. CHIKV strains were successfully isolated by using a cell culture (C6/36) method (online Technical Appendix, available from www.cdc.gov/EID/content/15/11/1854-Techapp.pdf).

To identify genetic relationships among these 14 imported CHIKV isolates, complete structural polyprotein gene sequences of 10 isolates (GenBank accession nos. FJ807886–FJ807895) and full genome sequences of 4 isolates (Singapore/0611aTw, Indonesia/0706aTw, Bangladesh/0810aTw, and Malaysia/0810bTw strains) (GenBank accession nos. FJ807896–FJ807899) were determined. Nucleotide sequences of complete open reading frames of Singapore/0611aTw, Bangladesh/0810aTw, and Malaysia/0810bTw isolates were most closely related to the India IND-06-AP3 strain (99.95%, 99.84%, and 99.77% identities, respectively) and other India 2006 isolates, which suggests common genetic origins from India.

In comparison with other CHIKV strains, unique substitution K252Q in the envelope 2 (E2) protein was found in all 4 imported isolates from Malaysia, and 2 unique substitutions, V4A and N349D, in the envelope 1 (E1) protein were found in the imported Bangladesh/0810aTw isolate. The Indonesia/0706aTw isolate was most closely related to the Malaysia MY003IMR isolate (99.42% identity). A novel 4-aa deletion, corresponding to nonstructural protein 3 codons 379–382 (TTACCAACCATA coding for Leu-Pro-Thr-Ile in the Malaysia MY003IMR strain), was observed in the Indonesia/0706aTw strain when it was compared with other CHIKV sequences available in GenBank. Further sequence analysis showed that all 6 isolates from Indonesia had the same deletion in this region.

A phylogenetic tree based on 49 CHIKV partial E1 gene sequences was constructed to trace the origins of the 14 CHIKV strains reported in this study (Figure). Phylogenetic

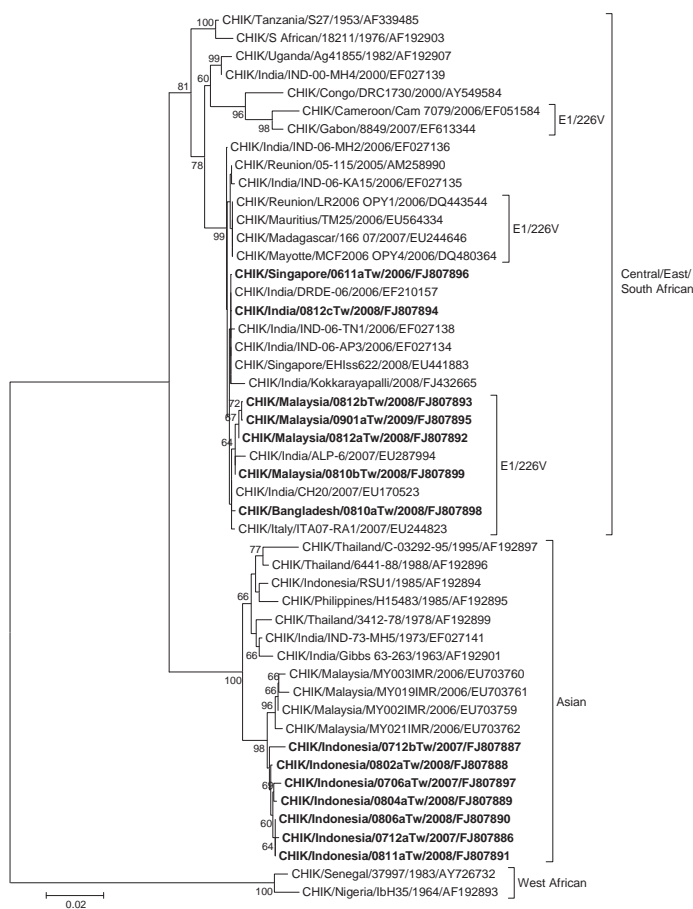


Figure. Phylogenetic relationships of chikungunya virus (CHIKV) isolates from 14 imported cases of chikungunya, Taiwan, 2006–2009. The tree was constructed on the basis of partial envelope 1 (E1) nucleotide sequences (836 bp, nt positions 10264–11099 of the prototype CHIKV S27 genomic sequence) of 49 CHIKV strains. Sequences obtained in this study are indicated in **boldface**. CHIKV strains with the E1-A226V mutation are indicated. Genotypes are indicated on the right. Viruses were identified by using the nomenclature of virus/country/strain/year of isolation/GenBank accession number. Analysis was performed by using MEGA 4 software and neighbor-joining (maximum composite likelihood) methods. Bootstrap support values >60 are shown (1,000 replicates). West African genotype Senegal strain 37997 sequence was used as the outgroup virus. Scale bar indicates nucleotide substitutions per site.

analysis shows that all 7 strains from Indonesia isolated during 2007–2008 are grouped into the Asian genotype and clustered in a distinct lineage. This lineage shows close relationship to the Malaysia/MY002IMR/2006 isolate. However, the 4 strains from Malaysia isolated during 2008–2009 belong to the Central/East/South African genotype and are clustered with CHIKV strains of India/ALP-6/2007, Italy/ITA07-RA1/2007, and Bangladesh/0810aTw/2008. These viruses also have the E1-A226V

mutation. The imported Singapore/0611aTw/2006 and India/0812cTw/2008 strains belong to the Central/East/South African genotype and are clustered with several India/2006 (IND-06-AP3, IND-06-TN1 and DRDE-06), India/Kokkarayapalli/2008, and Singapore/EHIss622/2008 strains, which have an alanine at the position E1-226.

Our results provide insights into the current distribution of different CHIKV genotypes and lineages. Phylogenetic analysis demonstrated

that CHIKV strains isolated from Indonesia during 2007–2008 remain stable and belong to the Asian genotype, whereas the other 7 isolates from Singapore, Bangladesh, Malaysia, and India belong to the Central/East/South African genotype. The Malaysia/2008–2009 and Bangladesh/2008 isolates have the E1-226(V) mutation similar to reported variants isolated in Cameroon, some Indian Ocean islands, India, Italy, and Gabon during 2006–2007 (4,6–9). These results show that different lineages of CHIKV strains from India with the Central/East/South African genotypes have been transmitted long distances by infected persons to various countries in Asia, including Singapore, Malaysia, and Bangladesh.

Although the urban mosquito *Aedes aegypti* is the primary vector for dengue and chikungunya transmission in Asia, the *Ae. albopictus* mosquito, a less efficient vector, was recently identified as the main or alternate vector in chikungunya outbreaks in central and East Africa (4,7,10), India (8), and Italy (6). Recent studies have suggested that the increased chikungunya outbreaks caused by CHIKV strains of the Central/East/South African genotype might be associated with a change in 1 nt, the A226V mutation, in the E1 protein during continuous epidemics (8,9). It is not known whether E1-A226V variants play a dominant role in urban or periurban areas of Asia and Africa where *Ae. aegypti* and *Ae. albopictus* mosquitoes are present.

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References

1. Powers AM, Brault AC, Tesh RB, Weaver SC. Re-emergence of chikungunya and o'nyong-nyong viruses: evidence for distinct geographical lineages and distant evolutionary relationships. *J Gen Virol*. 2000;81:471-9.
2. Powers AM, Logue CH. Changing patterns of chikungunya virus: re-emergence of a zoonotic arbovirus. *J Gen Virol*. 2007;88:2363-77. DOI: 10.1099/vir.0.82858-0
3. Shu PY, Yang CF, Su CL, Chen CY, Chang SF, Tsai KH, et al. Two imported chikungunya cases, Taiwan. *Emerg Infect Dis*. 2008;14:1326-7.
4. Peyrefitte CN, Rousset D, Pastorino BAM, Pouillot R, Bessaud M, Tock F, et al. Chikungunya virus, Cameroon, 2006. *Emerg Infect Dis*. 2007;13:768-71.
5. Yergolkar PN, Tandale BV, Arankalle VA, Sathe PS, Sudeep AB, Gandhe SS, et al. Chikungunya outbreaks caused by African genotype, India. *Emerg Infect Dis*. 2006;12:1580-3.
6. Bonilauri P, Bellini R, Calzolari M, Angelini R, Venturi L, Fallacara F, et al. Chikungunya virus in *Aedes albopictus*, Italy. *Emerg Infect Dis*. 2008;14:852-4. DOI: 10.3201/eid1405.071144
7. Pagès F, Peyrefitte CN, Mve MT, Jarjaval F, Brisse S, Itean I, et al. *Aedes albopictus* mosquito: the main vector of the 2007 chikungunya outbreak in Gabon. *PLoS One*. 2009;4:e4691. DOI: 10.1371/journal.pone.0004691
8. Santhosh SR, Dash PK, Parida MM, Khan M, Tiwari M, Lakshmana Rao PV. Comparative full genome analysis revealed E1: A226V shift in 2007 Indian chikungunya virus isolates. *Virus Res*. 2008;135:36-41. DOI: 10.1016/j.virusres.2008.02.004
9. de Lamballerie X, Leroy E, Charrel RN, Tsetsarkin K, Higgs S, Gould EA. Chikungunya virus adapts to tiger mosquito via evolutionary convergence: a sign of things to come? *Virology*. 2008;5:33. DOI: 10.1186/1743-422X-5-33
10. Schuffenecker I, Itean I, Michault A, Murri S, Frangeul L, Vaney MC, et al. Genome microevolution of chikungunya viruses causing the Indian Ocean outbreak. *PLoS Med*. 2006;3:e263. DOI: 10.1371/journal.pmed.0030263

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Cutaneous Larva Migrans Acquired in Brittany, France

To the Editor: Hookworm-related cutaneous larva migrans is a parasitic dermatosis caused by the penetration of larvae, mostly of a dog or cat hookworm, into the epidermis of humans (1,2). This eruption is most commonly found in tropical and subtropical areas but was recently reported from western Europe, including Germany (3,4), England (5,6), Scotland (7), and southern France (8). We report a patient from the Netherlands who acquired hookworm-related cutaneous larva migrans while on a holiday in Brittany, France.

A previously healthy 40-year-old man from the Netherlands traveled to Brittany, France, to visit from September 1 to September 15, 2008. He and his partner slept in tents, sometimes camping rough (not on designated camping sites or on private property), and they stayed in low-budget hotels. They spent a lot of time on several beaches along the Atlantic Ocean on the southern shore of Brittany ($\approx 48^\circ\text{N}$). The weather during their stay was variable. The patient was frequently bitten by mosquitoes, especially on his feet. He had not traveled to the tropics before and did not own any pets.

After his return to the Netherlands, the area around 2 presumed mosquito bites at the lateral side of his right foot became red, swollen, and itchy. This area evolved into a

1-cm pustule that later turned into a bulla. On November 10, he visited his general practitioner, who made a diagnosis of cellulitis and started the patient on amoxicillin/clavulanic acid 625 mg, 3 \times /day for 10 days. During antimicrobial drug treatment, skin inflammation improved, but after 2 days the patient noticed that an itching red streak had developed, extending from the lesions on the lateral side of the right foot to the whole width of the sole of the foot. The tip of the streak proceeded along the sole of the foot at the rate of 2 cm/day. On the fifth day, he was referred to our Tropical Diseases outpatient clinic.

Physical examination showed 2 elevated, ulcerative lesions on the lateral side of the right foot, and from each originated an elevated serpiginous lesion (Figure, panels B and C). These were typical tortuous lesions 2 cm in width. One of the lesions ran across the whole sole of the right foot and was 14 cm in length (Figure, panels A and C). The medial end of the lesion was fervently erythematous. Based on clinical signs, we diagnosed the skin lesion as hookworm-related cutaneous larva migrans with secondary impetiginization. The patient was subsequently treated with a single oral dose of 12 mg ivermectin. The itch and the progression of the lesion halted instantly and the lesion disappeared during the following weeks. The larva was not extirpated and thus not further identified.

Hookworm-related cutaneous larva migrans is usually caused by *Ancylostoma brasiliense*, *A. caninum* or, rarely, *Uncinaria stenocephala*. These zoonotic hookworms need a high temperature and a moist environment to develop from an embryo to filariforme larva (1,2). Hookworm-related cutaneous larva migrans is typically a disorder of tropical and subtropical zones and it is rather common among tourists who visit tropical beaches. This was the first patient we had seen with this disease who became infected in west-