later this year with higher virulence or widespread antiviral drug resistance.

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Imported Ciprofloxacin-Resistant *Neisseria meningitidis*

To the Editor: Emergence and spread of antimicrobial drug resistance in community-acquired infections is a global threat. Resistance of *Neisseria meningitidis* raises concern because of severity of disease caused by this organism and the need for immediate treatment of infected patients.

We report an imported case of meningococcal disease caused by fluoroquinolone-resistant *N. meningitidis*. The patient, a previously healthy, unvaccinated 43-year-old man who had traveled internationally, was hospitalized because of high fever, neck stiffness, and a diffuse petechial rash Signs and symptoms were observed 24 hours after he had returned to Italy from a 10-day business trip during February–March 2009, to New Delhi and Chennai in India and a stopover of a few hours in Frankfurt, Germany.

Microscopic examination of cerebrospinal fluid showed gram-negative diplococci and culture documented *N. meningitidis* serogroup A. The strain was characterized as serotype 4,21 subtype P1.9 by using monoclonal antibodies. Multilocus sequence typing performed at the National Reference Laboratory for Invasive Meningococcal Diseases in Rome characterized the strain as sequence type (ST)-4789 and belonging to clonal complex ST-5/subgroup III.

Antimicrobial drug susceptibility was determined by using an agar dilution test, and MICs were determined by using an agar disk-diffusion test (Etest; AB Biodisk, Solna, Sweden) and standard techniques. The strain was resistant to ciprofloxacin, levofloxacin, and trimethoprim/sulfamethoxazole and susceptible to penicillin, ampicillin, ceftriaxone, chloramphenicol, rifampin, and azithromycin. MICs for ciprofloxacin, levofloxacin, penicillin, ampicillin, and ceftriaxone were 0.25, 0.25, 0.03, 0.12, and <0.016 mg/L, respectively (Figure). The patient recovered after treatment with ceftriaxone.

Before results of antimicrobial drug–susceptibility testing were available, 15 adult contacts of the patient received ciprofloxacin as chemoprophylaxis according to public health recommendations in Italy. After positive test results, all contacts were offered repeat chemoprophylaxis with rifampin; 13 of them accepted. A diagnosis of meningitis and results of antibiograms were sent to the patient's place of employment in India and to the airport manager in Frankfurt. However, we were not able to assess what chemoprophylaxis was given to the patient's fellow employees and air travel contacts. No secondary cases have been detected so far in Italy.

Sporadic cases of infection with *N. meningitidis* (mainly serogroup B) with reduced susceptibility to ciprofloxacin have been reported in Europe, North and South America, and Australia since 2000 (*1–4*). Ciprofloxacin-resistant *N. meningitidis* of serogroup A caused an outbreak of meningococcal meningitis in Delhi, India, in 2005 and a recurrence in 2006 (*5*). Although the patient reported in our study had no known contact in India with patients who had meningococcal disease, mul-

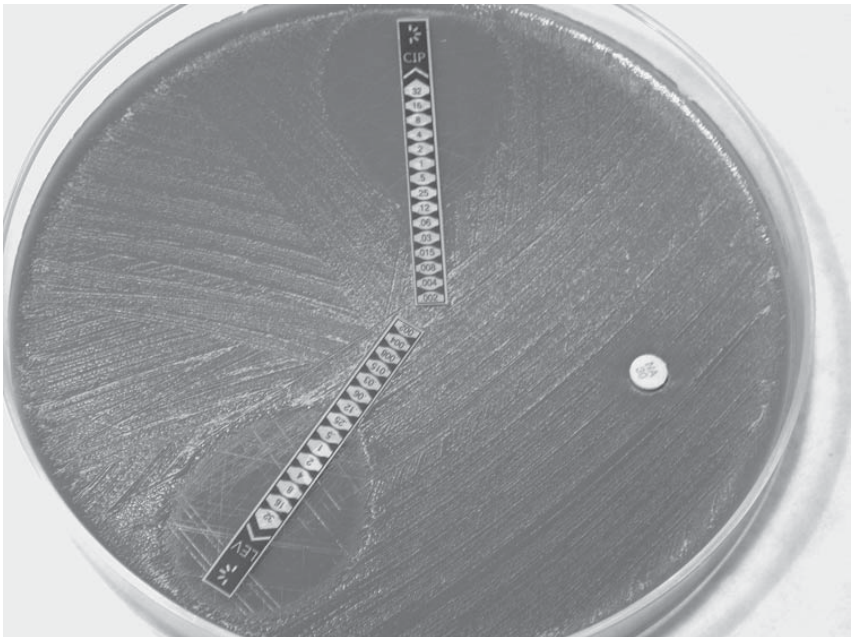

Figure. Antimicrobial drug–susceptibility test, showing resistance to levofloxacin (LEV, lower strip), ciprofloxacin (CIP, upper strip), and nalidixic acid (NA, disk) for the strain of *Neisseria meningitidis* isolated from the patient. A color version of this figure is available online (www.cdc.gov/EID/content/15/11/1852-F.htm).

tilocus sequencing typing analysis showed that the isolate had the same sequence type as isolates from the epidemic in India (*5*,*6*).

We report isolation of an imported, ciprofloxacin-resistant strain of *N. meningitidis* isolated from a patient with meningococcal disease. During the past 2 years, 182 strains of *N. meningitidis* have been sent to the Istituto Superiore di Sanità; all were susceptible to ciprofloxacin and MICs ranged from 0.002 mg/L to 0.006 mg/L (National Reference Laboratory for Invasive Meningococcal Diseases, pers. comm.) Serogroup A *N. meningitidis* accounted for only 1 of these strains; serogroups B and C are the most common groups in Italy. In contrast, group A meningococci are the major cause of meningitis outbreaks worldwide, especially in Africa and Asia. To date, spread of ciprofloxacin resistance in serogroup A appears to be limited to India because a recent report of antimicrobial drug susceptibility of *N. meningitidis* in the meningitis belt of Africa during 2000–2006 showed no evidence of ciprofloxacin resistance (*7*).

Temporal correlation and epidemiologic features strongly suggest that transmission of *N. meningitidis* to our patient occurred during his journey to India. Meningococcal disease is rarely imported because onset of symptoms is often rapid and severe. Nonetheless, the enormous increase in global trade and travel and shortening of international travel time may increase the risk for spread of infectious diseases and drug-resistant organisms. In addition, carriage of *N. meningitidis* in the nasopharynx of otherwise healthy persons can occur.

Emergence of fluoroquinolone resistance in some countries raises concerns about current chemoprophylaxis recommendations for meningococcal disease. Ciprofloxacin is widely used for postexposure prophylaxis of close contacts of infected persons because it is simple to use (single oral dose) and lacks toxicity. However, patients and their contacts should be questioned about possible recent travel. When transmission of *N. meningitidis* is suspected in regions where fluoroquinolone resistance has been found (New Delhi, India, and North Dakota and western Minnesota in the United States), alternative chemoprophylaxis such as rifampin or ceftriaxone should be used.

Emergence of autochthonous ciprofloxacin-resistant *N. meningitidis* is possible in countries where fluoroquinolones are widely used. In vitro drug susceptibility testing is not routinely and uniformly used in all settings because treatment or chemoprophylaxis are usually started before antibiogram results are available. Our case demonstrates that drug susceptibility testing should be encouraged and routinely performed for all isolates. Local and worldwide surveillance for antimicrobial drug–resistant *N. meningitidis* is crucial for determining antimicrobial drug resistance trends and future recommendations for chemoprophylaxis and treatment.

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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