the use of masks, considering compliance with this advice unlikely because many Muslims believe that covering the face during the Hajj is prohibited and because masks need to be of high quality and changed at least every 6 hours to remain effective (*7*). Recent studies demonstrated that surgical and N95 masks were equally effective in preventing spread of PCR-detectable influenza virus when used by infected patients. These masks also were potentially effective at preventing respiratory virus acquisition by household contacts of infected persons when worn by healthy persons. However, effectiveness depended largely on adherence to mask use (*8*,*9*).

Maintenance of good hand hygiene is also effective in reducing spread of respiratory infection. The World Muslim League has issued a *fatwa* allowing use of alcohol-based hand-rubs on skin as a disinfectant (*10*).

The demonstration of high acceptability of simple physical measures to prevent ARI encourages the education of pilgrims during the pretravel encounter. The results also support conclusion that masks, hand-rubs, and disposable handkerchiefs should be provided to pilgrims, along with strong advice about the risk for ARI, to increase adherence to prevention measures.

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Persistent Extended-Spectrum β-Lactamase Urinary Tract Infection

To the Editor: Uncomplicated urinary tract infections (UTIs) in otherwise healthy adults are usually treated empirically because the causative microbe is highly predictable: 80%–90% are caused by *Escherichia coli*. In addition, short courses of therapy (1 day or 3 days) are usually completed before laboratory results become available. In the past decade, reports of community-acquired, extended-spectrum β-lactamase (ESBL)–producing *E. coli* isolates have increased worldwide, but they are still uncommon in the United States (*1*), where reported cases are generally associated with hospitals. An early report of true community-acquired ESBL-producing *E. coli* infections in the United States was published in 2007 (*2*). We report a case of community-acquired lower UTI caused by ESBL-producing and multidrug resistant *E. coli* in an otherwise healthy college-aged woman who had no hospital exposure. Despite proper treatment, her infection persisted subclinically and symptoms recurred 2 months later.

The patient was an afebrile 24-year-old female college student who had visited her university health service, where she was recruited into a clinical trial investigating the effects of cranberry juice on UTIs. Inclusion in the study required that participants have UTI signs and symptoms, positive urine culture, and physician diagnosis. Participants provided self-collected vaginal, rectal, and midstream urine specimens at the time of enrollment and at 3- and 6-month follow-up or UTI recurrence. Study protocol was approved by the University of Michigan Institutional Review Board.

E. coli was isolated from all specimens collected from the patient

at the time of enrollment; urinalysis confirmed pyuria (>100 leukocytes/high power field). Also at the time of enrollment, the patient reported no antimicrobial drug treatment during the previous 4 weeks, no history of hospitalization, no urethral catheterization, and no sexually transmitted infection (confirmed by medical record review). A 7-day regimen of nitrofurantoin was prescribed.

After 53 days, the patient returned to the health service with recurring UTI symptoms and was treated with a 3-day regimen of trimethoprim–sulfamethoxazole; no urine specimen was submitted at that time. However, *E. coli* isolates were recovered from recurrence urine and rectal specimens collected within 48 hours according to the clinical trial protocol. All *E. coli* isolates collected at the time of enrollment (n = 3) and recurrence (n = 2) appeared morphologically and phenotypically identical (API Rapid 20E; bioMérieux, Durham, NC, USA). Genotyping using enterobacterial repetitive intergenic consensus (ERIC) PCR with an ERIC-2 primer showed a shared ERIC type, indicating identity (Figure). When tested for antimicrobial drug susceptibility (Vitek 2; bioMérieux), all 5 isolates were identified as ESBL-producers and were resistant to β-lactams: ampicillin, cefazolin, ceftriaxone (MIC >64 μg/mL), aztreonam, and piperacillin. After an ESBL confirmatory test, recommended by the Clinical and Laboratory Standards Institute (*3*), showed positive results, the isolates were also considered resistant to ceftazidime (MIC 1–4 μg/mL) and cefepime. Disk diffusion indicated susceptibility to cefoxitin. The isolates were also resistant to fluoroquinolones, tetracycline, and trimethoprim–sulfamethoxazole but susceptible to aminoglycosides, carbapenems, and nitrofurantoin. Isolates from the time of enrollment had intermediate susceptibility to amoxicillin–clavulanate (MIC 16 μg/mL), but isolates from the recurrence episode were resistant (MIC 32 μg/mL).

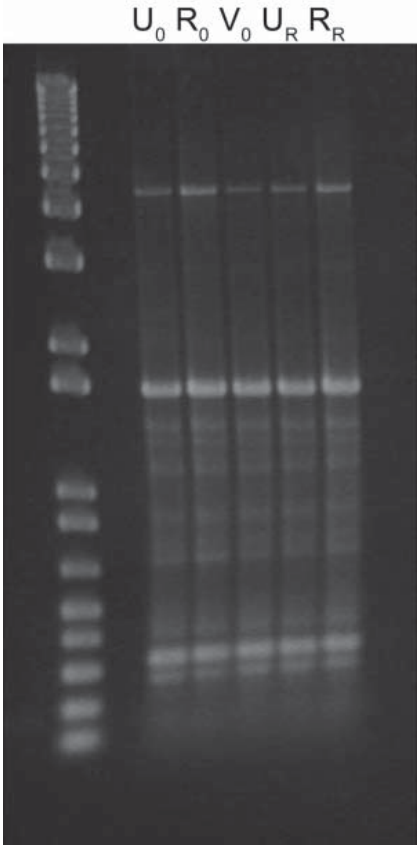

Figure. Enterobacterial repetitive intergenic consensus typing of extended-spectrum β-lactamase–producing *Escherichia coli* isolated from index ($_0$) and recurring ($_R$) urine (U), rectal (R), and vaginal (V) samples from a nonpregnant young woman.

Although the patient's initial UTI was treated adequately with nitrofurantoin, the infection recurred, implying that it remained in a reservoir, not uncommon for uncomplicated UTIs (*4*,*5*). Alternative antimicrobial drug treatment for outpatients with ESBL-producing *Enterobacteriaceae* is limited. Carbapenems remain the most effective drugs (*6*) but must be administered intravenously or intramuscularly (*3*). The reported efficacy of fosfomycin (*7*) suggests an option, but because agar dilution is the only recommended testing method, use of this drug in the United States is hindered. Use of antimicrobial drugs that concentrate in urine remains controversial as long as resistance is interpreted by MIC (blood-level resistance).

PCR detected β-lactamase resistance genes in all isolates, identifying them as ESBL positive when CTX-M consensus primer PCR was used but negative with TEM and SHV. Sequence analysis of the amplified gene showed that it encoded a CTX-M-15–like ESBL.

This isolate's increasing resistance to a β-lactamase–inhibitor combination, amoxicillin–clavulanate, suggested the possibility of inducible AmpC β-lactamase production. A negative AmpC disk test (with Tris/EDTA, cefoxitin, and *E. coli* ATCC 25922) refuted a plasmid-mediated AmpC β-lactamase (*6*); the remaining possible resistance mechanisms were hyperproduction of β-lactamase or an inhibitor-resistant penicillinase.

For the patient reported here, the multiple drug–resistant strain persisted for at least 53 days despite appropriate treatment with antimicrobial drugs. Furthermore, medical record review

found an additional UTI caused by *E. coli* 12 weeks later. Thus, because of the long duration of carriage of this highly resistant strain, potential for transmission to others is high.

The low number of previous reports of community-acquired ESBL in the United States does not necessarily suggest low community prevalence. Reports of ESBL-producer bacteremia in patients visiting emergency rooms suggests earlier and wider incidence (*8*). Returning to the practice of regularly culturing urine samples is difficult to justify; however, without ongoing surveillance to detect and control ESBL resistance, prevalence can only be expected to rise.

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Leishmania killicki Imported from Tunisian Desert

To the Editor: In North Africa, cutaneous leishmaniasis (CL) is a widespread zoonosis transmitted by sandflies. In Tunisia, 3 *Leishmania* species are responsible for CL: *L. major, L. infantum*, and *L. killicki. L. major* causes 2,000–4,000 zoonotic CL infections each year. *L. infantum*, the usual agent of visceral leishmaniasis, may be implicated in sporadic CL in northern Tunisia (dermotropic strains coexist with viscerotropic strains of *L. infantum* in these areas). *L. killicki* was first described in the desert region of Tataouine, Tunisia, in 1986 (*1*) and is the agent of chronic CL. We report a case of chronic CL caused by *L. killicki,* imported to Europe by a woman who had traveled to Tunisia.

A 76-year-old woman, with no relevant medical history, sought treatment from a dermatologist in Grenoble, France, for a cutaneous lesion on her right arm. This lesion had appeared 2 months after she returned from a July 2007 trip to Tunisia, where she spent 2 weeks in the desert riding camels and sleeping under a tent. The cutaneous lesion was isolated, round, 10 mm in diameter, ulcerative, surrounded by inflammation, and painless; no lymphadenopathy was found. The patient had no lesions on her mucous membranes and no concomitant general signs or symptoms. Given the absence of substantial signs or symptoms, the patient had paid no particular attention to this lesion until it became secondarily infected with bacteria. The secondary infection resolved after treatment with antimicrobial drugs, but the lesion persisted and a diagnosis of CL, presumably caused by *L. major*, was suggested.

Histologic investigation of a skin scraping showed amastigotes of *Leishmania* spp., but no further identification was done at that time. No treatment was given because the lesion was isolated and on the arm and because *L. major* lesions frequently heal spontaneously. After 2 months, the lesion had not healed, and *Leishmania* amastigotes were still found in scrapings. After 8 months, the lesion became inflamed, and a skin scraping sample was sent to the National Reference Center of *Leishmania* in Montpellier, France. DNA was extracted, and *L. killicki* was identified by genotyping. Various therapeutic options were considered, but no clear treatment recommendations were found. Parenteral therapy (pentavalent antimonials, pentamidine isethionate, or amphotericin B) was