Appendix Table, http://wwwnc.cdc.gov/EID/article/22/2/15-1292-Techapp1.pdf).). *P. falciparum* infection was detected in 2 *Anopheles* species: 1 (12.5%) of 8 *An. ininii* and 1 (5.0%) of 19 *An. nuneztovari s.l.* mosquitoes collected; *P. vivax* infection was found in 1 (5.5%) of 19 *An. nuneztovari s.l.* mosquitoes.

In September 2013, another malaria outbreak occurred 3 weeks after the deployment of 15 soldiers in Dagobert (4.06028°N, -53.70667°E; Figure). The attack rate among these soldiers was 53.3% (8/15): 7 *P. vivax* infections and 1 co-infection with *P. vivax* and *P. falciparum*. Mosquitoes were collected 3 months later by using human landing catches during 5 consecutive days. The area had been free of illegal gold mining activities since the 15 soldiers were deployed. A total of 321 *Anopheles* mosquitoes were collected in this location; 95.6% were identified as the same 4 species as in the Eau Claire mosquito collection (online Technical Appendix Table). Only 1 specimen (0.4%, 1/282), *An. darlingi* mosquito, was infected with *P. vivax*.

These results suggest a high level of malaria transmission involving *An. darlingi* and other *Anopheles* species as primary vectors of malaria in the rainforest. The findings probably highlight malaria hyperendemicity in communities of undocumented gold miners, who are often mobile and pose a challenge for controlling malaria and other infectious diseases in the region. Indeed, these gold miners could reintroduce malaria in areas where competent vectors exist in the coastal part of French Guiana and in Surinam and Brazil, which border French Guiana. This potential for transmission could seriously threaten the success of malaria elimination programs in the Guiana Shield. Further studies are needed to better evaluate malaria epidemiology in these undocumented populations to determine how best to adapt strategies to control malaria transmission in this subregion of South America.

Acknowledgments

We thank military physicians who participated in malaria epidemiologic surveillance in French Guiana and France during 2008–2014, especially E. de Parseval, N. Barthes, J.-P. Boudsocq, C. Ilcinkas, P.-A. Poutou, G. Samy, E. Martinez, F.-X. Le Flem, and C. Marchand. We also thank P. Gaborit, R. Carinci, and J. Issaly for their support in the entomologic studies.

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Importation of Fosfomycin Resistance *fosA3* Gene to Europe

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DOI: http://dx.doi.org/10.3202/eid2202.151301

To the Editor: The wide spread of *Enterobacteriaceae* resistant to last-resource therapeutic options, including

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extended-spectrum β-lactams, fluoroquinolones, and aminoglycosides, has re-ignited interest in old antimicrobial drugs, such as fosfomycin (*1*). Fosfomycin resistance rates are generally low (<10%) but substantially higher when carbapenemase producers are considered (15%–34%) (*1*–*3*). Resistance phenotypes have been more thoroughly investigated in *Escherichia coli* and linked to chromosomal mutations in the target (*murA*) or transporter (*glpT* and *uhpT*) genes or less frequently to plasmid-mediated fosfomycin resistance genes (*fosA*, *fosB*, *fosC*) encoding glutathione S-transferases that inactivate the drug (*4*). *fosA3* is the most prevalent gene variant, disseminated mainly in *E. coli* isolates from clinical and nonclinical origins (healthy persons, companion and food animals) in countries in Asia (China, South Korea, and Japan) (*2*–*6*) and only recently in a migratory bird in Europe (*7*). We investigated the occurrence and molecular features of 43 fosfomycin-resistant *Enterobacteriaceae* isolates (21 *E. coli*, 21 *Klebsiella pneumoniae*, and 1 *Morganella morganii*). These isolates were identified among 461 third-generation cephalosporin-resistant *Enterobacteriaceae* isolates from a community laboratory in northern Portugal during a 13-month period (August 2012–August 2013).

We screened for carriage of plasmidborne fosfomycin resistance genes (*fosA*, *fosA3*, *fosB*, *fosC2*) by PCR and sequencing (*2*,*5*). Chromosomal mutations in *murA*, *glpT*, and *uhpT* were investigated for 9 representative *E. coli* isolates (*8*) and 7 representative *K. pneumoniae* isolates with variable MICs to fosfomycin (≥64 mg/L) by PCR and comparison of sequences with reference wild-type strains (*E. coli* ATCC25922 and *K. pneumoniae* type strain JCM1662) (*8*; this study). Fosfomycin-resistant isolates represented 9.3% (43/461) of the collection surveyed during the study period, which is in line with rates reported for clinical isolates from other countries (*2*,*3*). Bacterial identification and antimicrobial drug susceptibility testing to β-lactams and non–β-lactams were performed by automated methods and further confirmed by disk diffusion and agar dilution (for fosfomycin, MIC cutoff 32 mg/L) according to European Committee on Antimicrobial Susceptibility Testing guidelines (http://www.eucast.org). We screened bla_{ESBL} genes ($bla_{\text{CTX-M}}$, bla_{TEM}, bla_{SHV}) by PCR and sequencing (*9*).

One (2.3%) of 43 *E. coli* isolates carried *fosA3*, $bla_{\text{CTX-M-15}}$, and $bla_{\text{TEM-1}}$ and contained mutations in GlpT (L297F, T348N, Q443E, E444Q) and UhpT (E350Q) (GenBank accession nos. KT832798 and KT832797, respectively), most of which were previously associated with fosfomycin resistance (*8*). This isolate was detected in a urine sample from a 61-year-old man who had a clinical history of chronic prostatitis and was associated with a urinary tract infection (UTI) acquired after travel to Asia (China, Philippines). *aac-6'-lb-cr*, $bla_{\text{OXA-I}}$, and *rmtB* genes were negative by PCR. This isolate exhibited fosfomycin MIC ≥256 mg/L and was concomitantly resistant to cefotaxime, cefepime, aztreonam, ciprofloxacin, gentamicin, kanamycin, netilmycin, streptomycin, sulphonamide, tetracycline, tobramycin, and trimethoprim but not to carbapenems, amoxicillin/clavulanic acid, or cefoxitin. In other *E. coli* isolates, fosfomycin resistance phenotypes were linked to mutations in transporter proteins UhpT (8 isolates, E350Q) and GlpT (3 isolates, premature stop codons resulting in truncated proteins of 45, 134, or 442 aminoacids [GenBank accession nos. KT832799, KT832800, and KT832801, respectively]); however, no amino acid changes were detected in *K. pneumoniae* isolates. The detection of *fosA3* in a clinical *E. coli* isolate in Europe is alarming because of its association with $bla_{\text{CTX-M-15}}$, which is highly disseminated in Portugal and other European Union countries (*9*), whereas fosfomycin is increasingly being used to treat UTIs caused by extended-spectrum β-lactams–producing *E. coli* (*1*).

Strain typing (identification of *E. coli* phylogroups and multilocus sequence typing; http://mlst.warwick.ac.uk/mlst/) revealed that this isolate belonged to phylogenetic group D_1 and the sequence type 393 clone (*9*). This clone was not previously detected among *fosA3*-carrying isolates (*3*,*4*), but it is distributed worldwide (including Asia) linked to community-acquired UTI and multidrug resistance patterns (*9*).

Conjugative assays (solid/broth mating at 24°C/37°C using *E. coli* Hb101 azide and kanamycin resistant as recipient) and plasmid typing assessed by PCR-based replicon typing, IncFII typing formula (FAB), I-*Ceu*I pulsed-field gel electrophoresis, and hybridization (*5*) showed that both *fosA3* and $bla_{\text{CTX-M-15}}$ were co-located in a conjugative F2:A-:B- plasmid (transconjugant MIC to fosfomycin ≥256 mg/L). Moreover, the genetic environment of *fosA3* was assessed by PCR mapping and sequencing (*2*,*6*), showing a composite transposon containing an insertion sequence *26* 323 bp upstream *fosA3*; the *orf1*, *orf2*, and *orf3* genes (homologous to regulatory ones in *K. pneumoniae* 342); and an insertion sequence (IS) *26* downstream (GenBank accession no. KT734860). The genetic platform (IS*26* composite transposon) and the IncFII plasmid variant (F2:A-:B-) are main vehicles for disseminating *fosA3* among clinical isolates, companion and food animals in Asian countries (*3*,*5*,*6*), or $bla_{\text{CTX-M-15}}$ worldwide (*10*). Thus, epidemiologic and molecular data suggest that the detection of *fosA3* in a clinical isolate in Europe is associated with a travel-related infection acquired after international travel to Asia. The acquisition of *fosA3* by a successful clone and an efficient resistance plasmid, which might entail subsequent dissemination and alerts to the need of close monitoring of fosfomycin resistant isolates, is of particular concern.

This work received financial support from European Union FEDER (Fundo Europeu de Desenvolvimento Regional) funds through COMPETE (Programa Operacional Fatores de Competitividade), and National Funds (Fundação para a Ciência e Tecnologia) through project UID/Multi/04378/2013. The work also received financial support from the European Union (FEDER funds) under the framework of QREN (Quadro de Referência Estratégica Nacional through project NORTE-07-0124-FEDER-000066. C.R. and Â.N. were supported by fellowships from Fundação para a Ciência e Tecnologia (SFRH/BD/84341/2012 and SFRH/BPD/104927/2014, respectively).

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Mycoplasma pneumoniae Monoclonal P1 Type 2c Outbreak, Russia, 2013

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DOI: http://dx.doi.org/10.3201/eid2202.151349

To the Editor: *Mycoplasma pneumoniae* is a major cause of respiratory infections among children and young adults and is responsible for up to 40% of all community-acquired pneumonia. In 2011, an epidemic of *M. pneumoniae* infection was reported in several countries in Europe and Asia and in Israel that primarily involved adhesin P1 type 1 strains and only a few P1 type 2 strains (*1*,*2*). The spread of *M. pneumoniae* was polyclonal (*1*–*3*), except in a few semiclosed settings, such as schools (*4*). Recently, a new adhesin P1 type 2 variant, named 2c, was reported (*5*,*6*) and accounted for 8.3% of 96 *M. pneumoniae*–positive samples in Germany (*7*).

In 2013, an increase in the number of community-acquired pneumonia cases was reported in children and their adult contacts from 2 towns in Russia separated by 45 km, Ozerniy and Duchovshina, during January–March and October–November, respectively. To characterize the outbreak in Ozerniy, we collected 13 throat swabs from 9 symptomatic children and 4 asymptomatic adults who were the parents or grandparents of the affected children. All children attended the same school and were treated in the same district hospital as inpatients or outpatients. In Duchovshina, throat swab samples were collected from 17 children and 2 adults. The children attended the same school, and the preschool-aged children visited the same daycare center 1 km away. One adult patient was the first aid driver who