Appendix Table, [http://wwwnc.cdc.gov/EID/article/22/2/15-1292-Techapp1.pdf](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. inini* 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.

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References

1. Ardillon V, Carvalho L, Prince C, Abboud P, Djossou F. Bilans 2013 et 2014 de la situation du paludisme en Guyane. Bulletin de veille sanitaire Antilles–Guyane. 2015 [cited 2015 Jul 15]. p. 16–20. [http://www.invrsante.fr/fr/Publications-et-outils/Bulletin-de-veille-sanitaire/Tous-les-nombres/Antilles-Guyane/Bulletin-de-veille-sanitaire-Antilles-Guyane-n1-Janvier-2015](http://www.invrsante.fr/fr/Publications-et-outils/Bulletin-de-veille-sanitaire/Tous-les-nombres/Antilles-Guyane/Bulletin-de-veille-sanitaire-Antilles-Guyane-n1-Janvier-2015)

2. Musset L, Pelleau S, Girod R, Ardillon V, Carvalho L, Dusfour I, et al. Malaria on the Guiana Shield: a review of the situation in French Guiana. Mem Inst Oswaldo Cruz. 2014;109:525–33. [http://dx.doi.org/10.1590/0074-0276140031](http://dx.doi.org/10.1590/0074-0276140031)

3. Carme B. Substantial increase of malaria in inland areas of eastern French Guiana. Trop Med Int Health. 2005;10:154–9. [http://dx.doi.org/10.1111/j.1365-3156.2004.01365.x](http://dx.doi.org/10.1111/j.1365-3156.2004.01365.x)

4. Berger F, Flamand C, Musset L, Djossou F, Rosine J, Sanquer MA, et al. Investigation of a sudden malaria outbreak in the isolated Amazonian village of Saul, French Guiana, January–April 2009. Am J Trop Med Hyg. 2012;86:591–7. [http://dx.doi.org/10.4269/ajtmh.2012.11-0582](http://dx.doi.org/10.4269/ajtmh.2012.11-0582)

5. Migliani R, Pradines B, Michel R, Aoun O, Dia A, Deparis X, et al. Malaria control strategies in French armed forces. Travel Med Infect Dis. 2014;12:307–17. [http://dx.doi.org/10.1016/j.tmaid.2014.05.008](http://dx.doi.org/10.1016/j.tmaid.2014.05.008)

6. Queyriaux B, Texier G, Ollivier L, Galoisy-Guibal L, Michel R, Meynard JB, et al. Plasmodium vivax malaria among military personnel, French Guiana, 1998–2008. Emerg Infect Dis. 2011;17:1280–2. [http://dx.doi.org/10.3201/eid1707.100009](http://dx.doi.org/10.3201/eid1707.100009)

7. Floch H, Abonnenc E. Anophèles de la Guyane Française. Arch Inst Pasteur Guyane. 1951;2:1–92.

8. Beebe NW, Saul A. Discrimination of all members of the *Anopheles punctulatus* complex by polymerase chain reaction–restriction fragment length polymorphism analysis. Am J Trop Med Hyg. 1995;53:478–81.

9. Snounou G, Viriyakosol S, Zhu XP, Jarra W, Pinheiro L, do Rosario VE, et al. High sensitivity of detection of human malaria parasites by the use of nested polymerase chain reaction. Mol Biochem Parasitol. 1993;61:315–20. [http://dx.doi.org/10.1016/0166-6851(93)90077-B](http://dx.doi.org/10.1016/0166-6851(93)90077-B)

10. Vezenezho SB, Adde A, Gaborit P, Carinci R, Issaly J, Pommier de Santi V, et al. Mosquito magnet® liberty plus trap baited with octenol confirmed best candidate for *Anophèles* surveillance and proved promising in predicting risk of malaria transmission in French Guiana. Malar J. 2014;13:384. [http://dx.doi.org/10.1186/1475-2875-13-384](http://dx.doi.org/10.1186/1475-2875-13-384)

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**Importation of Fosfomycin Resistance fosA3 Gene to Europe**

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

1These authors contributed equally to this article.
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 (gltP 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, gltP, 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 blaSSR genes (blaCTX-M, blaTEM, blaSHV) by PCR and sequencing (9).

One (2.3%) of 43 E. coli isolates carried fosA3, blaCTX-M-15, and blaTEM-1 and contained mutations in GltP (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’-Ib-cr, blaOXA-1, 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, netilmicin, 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 GltP (3 isolates, premature stop codons resulting in truncated proteins of 45, 134, or 442 amino acids [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 blaCTX-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 D1 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-CeuI pulsed-field gel electrophoresis, and hybridization (5) showed that both fosA3 and blaCTX-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 (IS26 composite transposon) and the IncFII plasmid variant (F2:A-::B-) are major vehicles for disseminating fosA3 among clinical isolates, companion and food animals in Asian countries (3,5,6), or blaCTX-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.
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References
1. Giske CG. Contemporary resistance trends and mechanisms for the old antibiotics colistin, temocillin, fosfomycin, mecillinam and nitrofurantoin. Clin Microbiol Infect. 2015;21:899–905. http://dx.doi.org/10.1016/j.cmi.2015.05.022
2. Lee SY, Park YJ, Yu JK, Jung S, Kim Y, Jeong SH, et al. Prevalence of acquired fosfomycin resistance among extended-spectrum beta-lactamase–producing Escherichia coli and Klebsiella pneumoniae clinical isolates in Korea and IS26-composite transposon surrounding fosA3. J Antimicrob Chemother. 2012;67:2843–7. http://dx.doi.org/10.1093/jac/dks319
3. Ho PL, Chan J, Lo WU, Lau PY, Cheung YY, Lau TC, et al. Prevalence and molecular epidemiology of plasmid-mediated fosfomycin resistance genes among blood and urinary Escherichia coli isolates. J Med Microbiol. 2013;62:1707–13. http://dx.doi.org/10.1099/jmm.0.062653-0
4. Sato N, Kawamura K, Nakane K, Wachino J, Arakawa Y. First detection of fosfomycin resistance gene fosA3 in CTX-M-producing Escherichia coli isolates from healthy individuals in Japan. Microb Drug Resist. 2013;19:477–82. http://dx.doi.org/10.1089/mdr.2013.0061
5. Hou J, Huang X, Deng Y, He L, Yang T, Zeng Z, et al. Dissemination of the fosfomycin resistance gene fosA3 with CTX-M beta-lactamase genes and mtrB carried on IncFII plasmids among Escherichia coli isolates from pets in China. Antimicrob Agents Chemother. 2012;56:2135–8. http://dx.doi.org/10.1128/AAC.05104-11
6. Ho PL, Chan J, Lo WU, Law PY, Li Z, Lau TC, et al. Dissemination of plasmid-mediated fosfomycin resistance fosA3 among multidrug-resistant Escherichia coli from livestock and other animals. J Appl Microbiol. 2013;114:695–702. http://dx.doi.org/10.1111/jam.12099
7. Villa L, Guerra B, Schmoger S, Fischer J, Helmuth R, Zong Z, et al. Inc/A/C plasmid carrying blaNDM-1, blaCMY-16, and fosA3 in a Salmonella enterica serovar Cervallis strain isolated from a migratory wild bird in Germany. Antimicrob Agents Chemother. 2015;59:6597–600. http://dx.doi.org/10.1128/AAC.00944-15
8. Takahata S, Ida T, Hiraishi T, Sakakibara S, Maebashi K, Terada S, et al. Molecular mechanisms of fosfomycin resistance in clinical isolates of Escherichia coli. Int J Antimicrob Agents. 2010;35:333–7. http://dx.doi.org/10.1016/j.ijantimicag.2009.11.011
9. Rodrigues C, Machado E, Pires J, Ramos H, Novais A, Peixe L. Increase of widespread A, B1 and D Escherichia coli clones producing a high-diversity of CTX-M-types in a Portuguese hospital. Future Microbiol. 2015;10:1125–31. http://dx.doi.org/10.2217/fmb.15.38
10. Coque TM, Novais A, Carattoli A, Poirel L, Pitout J, Peixe L, et al. Dissemination of clonally related Escherichia coli strains expressing extended-spectrum beta-lactamase CTX-M-15. Emerg Infect Dis. 2008;14:195–200. http://dx.doi.org/10.3202/cid1402.070350

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

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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 active samples in Germany (1). 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).