for 2014. This prevalence is increasing compared to that described in France in 2012 (14%). We found 35 $A\rightarrow G$ substitutions at position 2058 or 2059, two A2062T mutations and one A2059C mutation (Table) (1,9). Notably, in patients 15 and 33, who were infected with strains with macrolide resistance—associated mutations, M. genitalium infection was unsuccessfully treated with azithromycin, with treatment failures after azithromycin (1 g) and extended azithromycin (1.5 g for 5 d), but moxifloxacin treatment was effective. Patient 15 had been treated 1 year earlier with azithromycin (1 g) for nongonococcal urethritis.

Among the 168 patients whose isolates were examined for the 23S rRNA, gyrA, and parC genes, strains from 2 patients (patients 3 and 6) had both macrolide and fluoroquinolone-associated mutations (1.2%; 95% CI 0.33%–4.24%). Both patients received azithromycin (1 g), and patient 6 received additional azithromycin (1.5 g) after failure of azithromycin (1 g). Patient 6 experienced azithromycin failure again after the extended regimen. M. genitalium multidrug resistance is described in France at a prevalence of 1.2%, lower than prevalence described in Australia (7.5%) (7) and Japan (30.8%) (10).

In conclusion, *M. genitalium* fluoroquinolone resistance is emerging in France, with a prevalence of 6% in 2013–2014. Further, macrolide resistance also increased during this period, to a rate of 17.2%. Patients infected with *M. genitalium* strains containing both macrolide and fluoroquinolone resistance mutations associated with therapeutic failure raise concerns about untreatable *M. genitalium* infections.

Acknowledgments

We thank Manon Zerbib and Manon Passard for technical assistance.

References

- Touati A, Peuchant O, Jensen JS, Bébéar C, Pereyre S. Direct detection of macrolide resistance in *Mycoplasma genitalium* isolates from clinical specimens from France by use of real-time PCR and melting curve analysis. J Clin Microbiol. 2014;52:1549– 55. http://dx.doi.org/10.1128/JCM.03318-13
- Couldwell DL, Tagg KA, Jeoffreys NJ, Gilbert GL. Failure of moxifloxacin treatment in *Mycoplasma genitalium* infections due to macrolide and fluoroquinolone resistance. Int J STD AIDS. 2013;24:822–8. http://dx.doi.org/10.1177/0956462413502008
- Pond MJ, Nori AV, Witney AA, Lopeman RC, Butcher PD, Sadiq ST. High prevalence of antibiotic-resistant *Mycoplasma* genitalium in nongonococcal urethritis: the need for routine testing and the inadequacy of current treatment options. Clin Infect Dis. 2014;58:631–7. http://dx.doi.org/10.1093/cid/cit752
- Shimada Y, Deguchi T, Nakane K, Masue T, Yasuda M, Yokoi S, et al. Emergence of clinical strains of *Mycoplasma genitalium* harbouring alterations in ParC associated with fluoroquinolone resistance. Int J Antimicrob Agents. 2010;36:255–8. http://dx.doi.org/10.1016/j.ijantimicag.2010.05.011
- 5. Deguchi T, Yasuda M, Horie K, Seike K, Kikuchi M, Mizutani K, et al. Drug resistance-associated mutations in *Mycoplasma*

- genitalium in female sex workers, Japan. Emerg Infect Dis. 2015;21:1062–4. http://dx.doi.org/10.3201/eid2106.142013
- Bissessor M, Tabrizi SN, Twin J, Abdo H, Fairley CK, Chen MY, et al. Macrolide resistance and azithromycin failure in a *Mycoplasma genitalium*—infected cohort and response of azithromycin failures to alternative antibiotic regimens. Clin Infect Dis. 2015;60:1228–36. http://dx.doi.org/10.1093/cid/ciu1162
- Tagg KA, Jeoffreys NJ, Couldwell DL, Donald JA, Gilbert GL.
 Fluoroquinolone and macrolide resistance-associated mutations in
 Mycoplasma genitalium. J Clin Microbiol. 2013;51:2245–9.
 http://dx.doi.org/10.1128/JCM.00495-13
- Kikuchi M, Ito S, Yasuda M, Tsuchiya T, Hatazaki K, Takanashi M, et al. Remarkable increase in fluoroquinolone-resistant Mycoplasma genitalium in Japan. J Antimicrob Chemother. 2014;69:2376–82. http://dx.doi.org/10.1093/jac/dku164
- Jensen JS, Bradshaw CS, Tabrizi SN, Fairley CK, Hamasuna R. Azithromycin treatment failure in *Mycoplasma genitalium*-positive patients with nongonococcal urethritis is associated with induced macrolide resistance. Clin Infect Dis. 2008;47:1546–53. http://dx.doi.org/10.1086/593188
- Deguchi T, Kikuchi M, Yasuda M, Ito S. Multidrug-resistant *Mycoplasma genitalium* is increasing. Clin Infect Dis. 2016;62:405–6. http://dx.doi.org/10.1093/cid/civ898

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Possible Transmission of mcr-1-Harboring Escherichia coli between Companion Animals and Human

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DOI: http://dx.doi.org/10.3201/eid2209.160464

To the Editor: Plasmid-mediated, colistin-resistance mechanism gene *mcr-1* was first identified in *Escherichia coli* isolates from food, food animals, and human patients in November 2015 (1). Reports on detection of *mcr-1* in *Enterobacteriaceae* from humans and food animals

soon followed from ≈ 12 countries (2-5). Here we report detection of mcr-1 in colistin-resistant E. coli isolated from companion animals and the possible transmission of mcr-1-harboring E. coli between companion animals and a person.

Three *mcr-1*-harboring *E. coli* clinical isolates were identified from specimens of 3 patients admitted to a urology ward of a hospital in Guangzhou, China. *E. coli* isolate EC07 was identified in the urine of a 50-year-old male patient with glomerulonephritis in October 2015. Isolate EC08 was cultured from the urine of a 48-year-old male patient with prostatitis in December 2015. Isolate EC09 was identified in the blood of an 80-year-old male patient with bladder cancer 3 weeks after EC08 was identified.

Review of medical records identified the patient carrying *E. coli* isolate EC07 as a worker at a pet shop. In light of this finding, we collected a total of 53 fecal samples from 39 dogs and 14 cats in the pet shop where the man worked. We isolated and identified colonies consistent with *E. coli* from fecal samples on MacConkey agar plates (Thermo Fisher, Beijing, China) and API 20E system (bioMérieux,

Durham, NC, USA). We prepared crude DNA samples of isolates for PCR testing by boiling cells in water. Among them, 6 were positive for mcr-1 by PCR and sequencing (4 from dogs and 2 from cats). All 6 isolates were resistant to colistin, polymyxin B, cephalosporin, gentamicin, and ciprofloxacin by using the agar dilution method, in accordance with the European Committee on Antimicrobial Susceptibility Testing (http://www.eucast.org) for colistin and polymyxin B and Clinical and Laboratory Standards Institute guidelines (http://www.clsi.org) for the other antimicrobial drugs. We identified various resistance genes accounting for the multidrug resistance in these 9 mcr-1-positive isolates (6,7) (Table). We noted that *E. coli* isolate EC09 was also resistant to carbapenems and positive for bla_{IMP-4} . We observed co-production of mcr-1 and IMP-type metallo-βlactamase in E. coli.

We subjected all isolates to multilocus sequence typing, in accordance with the protocol described at http://mlst.warwick.ac.uk/mlst/dbs/Ecoli, and pulsed-field gel electrophoresis as described previously (8–10). We identified 5 *mcr-1*–positive isolates from 4 dogs (PET02–04 and PET06) and isolate EC07 as sequence

Table. Character	istics of 9 mic	r-r-positive E	Scriencina co	on isolates iro		animais and	numan patiei	its, Guarigzi	iou, Crima
		5550	5550	D==0.4	Isolate	55700			=
Characteristic	PET01	PET02	PET03	PET04	PET05	PET06	EC07	EC08	EC09
Isolation date	2016 Jan 1	2016 Jan 1	2016 Jan 2	2016 Jan 2	2016 Jan 2	2016 Jan 4	2015 Oct 10	2015 Nov 2	2015 Nov 21
Specimen source	Cat	Dog	Dog	Dog	Cat	Dog	Human	Human	Human
Specimen type	Feces	Feces	Feces	Feces	Feces	Feces	Urine	Urine	Blood
Phylogenetic group	B2	D	D	D	B2	D	D	B1	B1
ST†	ST93	ST354	ST354	ST354	New	ST354	ST354	ST156	ST156
PFGE type	IV	ı			V	ı		11	III
Resistance	mcr-1,	mcr-1,	mcr-1,	mcr-1,	mcr-1,	mcr-1,	mcr-1,	mcr-1,	mcr-1,
genes	bla _{TEM-1} , qepA	bla _{TEM-1} , bla _{CTX-M-15} , fosA3, aac(6')-lb-	bla _{TEM-1} , bla _{CTX-M-15} , fosA3, aac(6')-lb-	bla _{TEM-1} , bla _{CTX-M-15} , fosA3, aac(6')-lb-	bla _{TEM-1} , bla _{SHV-12} , bla _{CTX-M-15} , fosA3, rmtB,	bla _{TEM-1} , bla _{CTX-M-15} , fosA3, aac(6')-lb-	bla _{TEM-1} , bla _{CTX-M-15} , fosA3, aac(6')-lb-	bla _{TEM-1} , bla _{CTX-M-55} , fosA3, rmtB,	bla _{IMP-4} , bla _{TEM-1} , bla _{CTX-M-55} , fosA3,
		cr	cr	cr	qnrS, aac(6')-Ib-cr	cr	cr	qepA1	rmtB, qepA1
MIC, μg/mL									
Colistin	16	8	8	16	16	8	8	8	64
Polymyxin B	16	16	16	32	16	16	8	8	64
Ampicillin	>256	>256	>256	>256	>256	>256	>256	>256	>256
AMX/CLV	16	32	32	32	256	16	32	16	16
Cefotaxime	64	>256	256	>256	256	>256	256	256	>256
Ceftazidime	16	256	128	256	64	256	128	32	>256
Cefepime	8	256	128	256	16	256	64	64	>256
Gentamicin	128	>256	>256	>256	256	>256	>256	>256	>256
Amikacin	4	>256	>256	>256	>256	>256	>256	>256	>256
Ertapenem	<0.25	1	0.5	0.25	<0.25	1	0.5	<0.25	>16
Imipenem	<0.25	<0.25	<0.25	< 0.25	<0.25	<0.25	<0.25	<0.25	>16
Meropenem	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	>16
Fosfomycin	32	>512	>512	>512	>512	>512	>512	>512	>512
Tigecycline	<2	<2	<2	<2	<2	<2	<2	<2	4
Nitrofurantoin	<16	32	32	32	128	32	64	64	32
Ciprofloxacin	256	128	128	128	64	128	128	256	256

^{*}AMX/CLV, amoxicillin clavulanic acid; PFGE, pulsed-field gel electrophoresis; ST, sequence type.

†By multilocus sequence typing.

type (ST) 354. Isolates PET01 and PET05, identified from cats, belonged to ST93 and a new ST strain, respectively. Isolates EC08 and EC09, from the patients who shared the same hospital room with the pet shop worker, were ST156 (Table). Results of pulsed-field gel electrophoresis were consistent with multilocus sequence typing results and showed that isolates consisted of 5 types (types I to V; online Technical Appendix, http://wwwnc. cdc.gov/EID/article/22/9/16-0464-Techapp1.pdf). Isolate EC07 was clonally related to 4 E. coli strains from dogs, according criteria described by Tenover et al. (10), suggesting possible transmission of mcr-1-harboring E. coli between dogs and the patient. Colistin resistance was successfully transferred to E. coli C600 through conjugation in all isolates, suggesting that mcr-1 was located on transferable plasmids.

These findings suggest that *mcr-1*–producing *E. coli* can colonize companion animals and be transferred between companion animals and humans. The findings also suggest that, in addition to food animals and humans, companion animals can serve as a reservoir of colistin-resistant *E. coli*, adding another layer of complexity to the rapidly evolving epidemiology of plasmid-mediated colistin resistance in the community.

Acknowledgments

We sincerely thank the patients and the owners of companion animals for giving written consent for publication.

This work was supported by research grants from the National Natural Science Foundation of China (no. 81471988), the 111 Project (nos. B13037 and B12003), the Guangdong Natural Science Foundation (no. S2013010015810), and the Program of Science and Technology New Star of Guangzhou (no. 2014J2200038).

References

- Liu Y-Y, Wang Y, Walsh TR, Yi L-X, Zhang R, Spencer J, et al. Emergence of plasmid-mediated colistin resistance mechanism MCR-1 in animals and human beings in China: a microbiological and molecular biological study. Lancet Infect Dis. 2016;16:161–8. http://dx.doi.org/10.1016/ S1473-3099(15)00424-7
- Nordmann P, Lienhard R, Kieffer N, Clerc O, Poirel L. Plasmid-mediated colistin-resistant *Escherichia coli* in bacteremia in Switzerland. Clin Infect Dis. 2016 Mar 1:ciw124. http://dx.doi.org/10.1093/cid/ciw124
- Falgenhauer L, Waezsada SE, Yao Y, Imirzalioglu C, Käsbohrer A, Roesler U, et al. Colistin resistance gene *mcr-1* in extendedspectrum β-lactamase–producing and carbapenemaseproducing Gram-negative bacteria in Germany. Lancet Infect Dis. 2016;16:282–3. http://dx.doi.org/10.1016/S1473-3099(16)00009-8
- Tse H, Yuen KY. Dissemination of the mcr-1 colistin resistance gene. Lancet Infect Dis. 2016;16:145–6. http://dx.doi.org/10.1016/ S1473-3099(15)00532-0
- 5. Malhotra-Kumar S, Xavier BB, Das AJ, Lammens C, Butaye P, Goossens H. Colistin resistance gene *mcr-1* harboured on a

- multidrug resistant plasmid. Lancet Infect Dis. 2016;16:283–4. http://dx.doi.org/10.1016/S1473-3099(16)00012-8
- Tian GB, Huang YM, Fang ZL, Qing Y, Zhang XF, Huang X. CTX-M-137, a hybrid of CTX-M-14-like and CTX-M-15-like β-lactamases identified in an *Escherichia coli* clinical isolate. J Antimicrob Chemother. 2014;69:2081–5. http://dx.doi.org/10.1093/jac/dku126
- Yong D, Toleman MA, Giske CG, Cho HS, Sundman K, Lee K, et al. Characterization of a new metallo-beta-lactamase gene, bla_{NDM-1}, and a novel erythromycin esterase gene carried on a unique genetic structure in *Klebsiella pneumoniae* sequence type 14 from India. Antimicrob Agents Chemother. 2009;53:5046–54. http://dx.doi.org/10.1128/AAC.00774-09
- Wirth T, Falush D, Lan R, Colles F, Mensa P, Wieler LH, et al. Sex and virulence in *Escherichia coli*: an evolutionary perspective. Mol Microbiol. 2006;60:1136–51. http://dx.doi.org/10.1111/j.1365-2958.2006.05172.x
- Tian GB, Wang HN, Zhang AY, Zhang Y, Fan WQ, Xu CW, et al. Detection of clinically important β-lactamases in commensal *Escherichia coli* of human and swine origin in western China. J Med Microbiol. 2012;61:233–8. http://dx.doi.org/10.1099/ jmm.0.036806-0
- Tenover FC, Arbeit RD, Goering RV, Mickelsen PA, Murray BE, Persing DH, et al. Interpreting chromosomal DNA restriction patterns produced by pulsed-field gel electrophoresis: criteria for bacterial strain typing. J Clin Microbiol. 1995;33:2233–9.

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Acetobacter indonesiensis Bacteremia in Child with Metachromatic Leukodystrophy

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DOI: http://dx.doi.org/10.3201/eid2209.160566

To the Editor: Acetobacter indonesiensis, first described in 2000 (1), belongs to the group of acetic acid bacteria (AAB), which includes the genera Acetobacter, Gluconobacter, Asaia, Granulibacter, and others in the family Acetobacteriaceae. AAB are of great industrial interest for use in vinegar fermentation processes because they oxidize alcohols or sugars incompletely, which leads