# LETTERS

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-FED-ER-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).

#### References

- 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
- Lee SY, Park YJ, Yu JK, Jung S, Kim Y, Jeong SH, et al. Prevalence of acquired fosfomycin resistance among extendedspectrum 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
- Ho PL, Chan J, Lo WU, Lai EL, 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
- 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
- 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 rmtB 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
- Ho PL, Chan J, Lo WU, Law PY, Li Z, Lai EL, 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
- Villa L, Guerra B, Schmoger S, Fischer J, Helmuth R, Zong Z, et al. IncA/C plasmid carrying *bla*NDM-1, *bla*CMY-16, and *fosA3* in a *Salmonella enterica* serovar Corvallis 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
- 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
- Rodrigues C, Machado E, Pires J, Ramos H, Novais Â, 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
- 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/10.3201/eid1402.070350

Address for correspondence: Ângela Novais, UCIBIO/REQUIMTE Researcher, Laboratory of Microbiology, Faculty of Pharmacy, University of Porto. Rua Jorge Viterbo Ferreira no. 228 4050-313, Porto, Portugal; email: angelasilvanovais@gmail.com

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

# Inna Edelstein, Svetlana Rachina, Arabella Touati, Roman Kozlov, Nadège Henin, Cécile Bébéar, Sabine Pereyre

Author affiliations: Smolensk State Medical University of Ministry of Health of Russian Federation, Smolensk, Russian Federation (I. Edelstein, R. Kozlov); Inter-regional Association for Clinical Microbiology & Antimicrobial Chemotherapy, Smolensk (S. Rachina); University of Bordeaux, Bordeaux, France (A. Touati, N. Henin, C. Bébéar, S. Pereyre); Institut National de la Recherche Agronomique, Bordeaux (A. Touati, N. Henin, C. Bébéar, S. Pereyre); Bordeaux University Hospital, Bordeaux (C. Bébéar, S. Pereyre)

#### 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 communityacquired 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 daycare center 1 km away. One adult patient was the first aid driver who transported the children to the hospital. The other adult patient was a community center worker who spent time with the children. In both cities, the symptomatic patients received  $\beta$ -lactams as initial therapy before testing.

All specimens were processed in the laboratory of molecular diagnostics of the Smolensk State Medical Academy (Smolensk Russia). Nucleic acids were extracted by using the DNA-sorb-AM nucleic-acid extraction kit (InterLabService, Moscow, Russia), and *M. pneumoniae* was subsequently detected by using the AmpliSens *Mycoplasma pneumoniae/Chlamydophila pneumoniae*-FRT PCR kit (InterLabService). Two *M. pneumoniae* molecular typing methods, adhesin P1 typing and multilocus variablenumber tandem-repeat analysis (MLVA), were performed as previously described (1,5,7). Macrolide resistance-associated mutations were searched using real-time PCR and melting curve analysis (1). The *M. pneumoniae* isolates from the specimens collected in Ozerniy were all adhesin P1 type 2c and belonged to 4 distinct MLVA types, 1 of which, MLVA type 73563, has not been previously reported (Table). Without including the instable MPN1 marker (8), we observed only 2 MLVA types. The 19 *M. pneumoniae* isolates from the specimens collected in Duchovshina also were all P1 type 2c, and all belonged to the same MLVA type, 43562 (type M). No macrolide resistance–associated mutation was observed in any city.

For comparison purposes, because no previous data regarding *M. pneumoniae* molecular epidemiology in Russia were available, we retrospectively characterized 29 specimens, not from an outbreak, that were previously randomly collected for community-acquired pneumonia etiologic studies during October 2006–October 2007 and February– October 2010 by the laboratory of Smolensk State Medical

Table. Characteristics of 61 Mycobacterium pneumoniae-positive respiratory tract specimens collected in Ozerniy and Duchovshina	ł,
Russia*	

City or region				Respiratory			MLVA type	PCR-	Macrolide
and specimen	Sample	Patient	Hospitalization	clinical	Date of	MLVA	without	RFLP	resistance
designation	source	age, y	status	syndrome	collection	type†	MPN1‡	type	genotype
Ozerniy									
38795	Throat swab	12	Inpatient	Pneumonia	2013 Feb 15	53562 (S)	3562	2c	Wild type
38796	Throat swab	10	Inpatient	Pneumonia	2013 Feb 15	73562 (Y)	3562	2c	Wild type
38799	Throat swab	10	Inpatient	Pneumonia	2013 Feb 15	73563	3563	2c	Wild type
38812	Throat swab	9	Inpatient	Pneumonia	2013 Feb 15	73562 (Y)	3562	2c	Wild type
38814	Throat swab	11	Inpatient	Pneumonia	2013 Feb 15	73562 (Y)	3562	2c	Wild type
38941	Throat swab	33	Outpatient	Asymptomatic	2013 Feb 20	73563	3563	2c	No amp
38945	Throat swab	10	Inpatient	Pneumonia	2013 Feb 20	73563	3563	2c	No amp
38946	Throat swab	10	Inpatient	Pneumonia	2013 Feb 20	73563	3563	2c	Wild type
38960	Throat swab	33	Outpatient	Asymptomatic	2013 Feb 20	73563	3563	2c	Wild type
38962	Throat swab	51	Outpatient	Asymptomatic	2013 Feb 20	73562 (Y)	3562	2c	Wild type
39042	Throat swab	9	Inpatient	Pneumonia	2013 Feb 25	73562 (Y)	3562	2c	Wild type
39048	Throat swab	64	Outpatient	Asymptomatic	2013 Feb 25	63562 (V)	3562	2c	Wild type
39293	Throat swab	8	Inpatient	Pneumonia	2013 Mar 7	73562 (Y)	3562	2c	Wild type
Duchovshina									
43593	Throat swab	13	Inpatient	Pneumonia	2013 Oct 18	43562 (M)	3562	2c	Wild type
43596	Throat swab	15	Outpatient	Pneumonia	2013 Oct 18	43562 (M)	3562	2c	Wild type
43597	Throat swab	12	Inpatient	Pneumonia	2013 Oct 18	43562 (M)	3562	2c	Wild type
43692	Throat swab	5	Inpatient	Pneumonia	2013 Oct 22	43562 (M)	3562	2c	Wild type
43693	Throat swab	10	Inpatient	Pneumonia	2013 Oct 23	43562 (M)	3562	2c	Wild type
43694	Throat swab	9	Inpatient	Pneumonia	2013 Oct 22	43562 (M)	3562	2c	Wild type
43695	Throat swab	13	Inpatient	Pneumonia	2013 Oct 23	43562 (M)	3562	2c	Wild type
43804	Throat swab	9	Inpatient	Pneumonia	2013 Oct 27	43562 (M)	3562	2c	Wild type
43805	Throat swab	6	Inpatient	Pneumonia	2013 Oct 27	43562 (M)	3562	2c	Wild type
43806	Throat swab	14	Inpatient	Pneumonia	2013 Oct 27	43562 (M)	3562	2c	Wild type
43843	Throat swab	6	Inpatient	Pneumonia	2013 Oct 30	43562 (M)	3562	2c	Wild type
43888	Throat swab	58	Outpatient	Pneumonia	2013 Oct 31	43562 (M)	3562	2c	Wild type
43890	Throat swab	4	Inpatient	Pneumonia	2013 Oct 31	43562 (M)	3562	2c	Wild type
43919	Throat swab	41	Outpatient	Pneumonia	2013 Nov 1	43562 (M)	3562	2c	Wild type
43989	Throat swab	10	Inpatient	Pneumonia	2013 Nov 6	43562 (M)	3562	2c	Wild type
43990	Throat swab	13	Inpatient	Pneumonia	2013 Nov 6	43562 (M)	3562	2c	Wild type
43991	Throat swab	8	Inpatient	Pneumonia	2013 Nov 6	43562 (M)	3562	2c	Wild type
44174	Throat swab	10	Inpatient	Pneumonia	2013 Nov 14	43562 (M)	3562	2c	Wild type
44176	Throat swab	8	Inpatient	Pneumonia	2013 Nov 14	43562 (M)	3562	2c	No amp

\*MLVA, multilocus variable-number tandem-repeat; no amp, no amplification with the real-time PCR used to detect 23S rRNA mutations associated with macrolide resistance (1); RFLP, restriction fragment length polymorphism. An expanded version of this table is available online at http://wwwnc.cdc.gov/EID/article/22/2/15-1349-T1.htm.

†The profiles are named according to a string of allele numbers in order of MPN1, MPN13, MPN14, MPN15, and MPN16 markers showing the number of repeats at each locus. When available, the naming according to Degrange et al. is shown in parentheses (1).

The profiles are named according to a string of allele numbers in order of MPN13, MPN14, MPN15 and MPN16 markers showing the number of repeats at each locus. The instable MPN1 marker (8) was removed.

### LETTERS

Academy (Table). Of these specimens, 12 (41%) were P1 type 1, 15 (52%) were P1 type 2a, and only 2 (7%) were P1 type 2c. A polyclonal distribution with 8 distinct MLVA types was observed, with the MLVA type M representing 11 (38%) of the identified MLVA types. Without the MPN1 marker, 3 MLVA types were observed. No macrolide resistance–associated mutation was detected, similar to what was observed in the 32 specimens collected in 2013. This finding is consistent with the low prevalence of macrolide resistance reported in northern Europe (6,7).

We report 2 outbreaks of M. pneumoniae infections that occurred in the first and last quarter of 2013 in western Russia (Smolensk region). Despite the high predominance of P1 type 1 strains reported in the recent literature (1,2,7), these 2 outbreaks, reported in semiclosed settings involved only the newly described P1 type 2c variant; 1 outbreak represented a monoclonal phenomenon. In the Smolensk region, the circulation of both type 1 and 2 strains was observed a few years before the outbreak; most of these strains were P1 type 2a variants, and only a minority were type 2c variants, suggesting that the new type 2c variant had spread throughout this region of Russia since at least 2006. In other parts of the world, a switch between type 1 and type 2 strains might be occurring. Indeed, in the United States, P1 type 1 isolates predominated before 2010 but dropped to 50% of isolates in 2013, and type 2 and type 2 variant strains increased (9). This cyclic pattern of type 1 or type 2 predominance in the population has previously been reported (10).

In conclusion, we detected no macrolide resistance in western Russia. The P1 type 2c variant spread throughout this region and can be responsible for monoclonal outbreaks. The epidemiologic monitoring of *M. pneumoniae* P1 types will assess the potential switch to P1 type 2 in the United States and other parts of the world and detect the possible emergence of the P1 type 2c variant. This study was supported by internal funding.

#### References

- Pereyre S, Touati A, Petitjean-Lecherbonnier J, Charron A, Vabret A, Bébéar C. The increased incidence of *Mycoplasma pneumoniae* in France in 2011 was polyclonal, mainly involving *M. pneumoniae* type 1 strains. Clin Microbiol Infect. 2013;19:E212–7. http://dx.doi.org/10.1111/1469-0691.12107
- Liu Y, Ye X, Zhang H, Xu X, Wang M. Multiclonal origin of macrolide-resistant *Mycoplasma pneumoniae* isolates as determined by multilocus variable-number tandem-repeat analysis. J Clin Microbiol. 2012;50:2793–5. http://dx.doi.org/10.1128/ JCM.00678-12
- Chalker V, Stocki T, Litt D, Bermingham A, Watson J, Fleming D, et al. Increased detection of *Mycoplasma pneumoniae* infection in children in England and Wales, October 2011 to January 2012. Euro Surveill. 2012;17:20081.
- Pereyre S, Renaudin H, Charron A, Bébéar C. Clonal spread of Mycoplasma pneumoniae in primary school, Bordeaux, France. Emerg Infect Dis. 2012;18:343–5.

- Zhao F, Cao B, Li J, Song S, Tao X, Yin Y, et al. Sequence analysis of the P1 adhesin gene of *Mycoplasma pneumoniae* in clinical isolates collected in Beijing in 2008 to 2009. J Clin Microbiol. 2011;49:3000–3. http://dx.doi.org/10.1128/JCM.00105-11
- Spuesens EB, Hoogenboezem T, Sluijter M, Hartwig NG, van Rossum AM, Vink C. Macrolide resistance determination and molecular typing of *Mycoplasma pneumoniae* by pyrosequencing. J Microbiol Methods. 2010;82:214–22. http://dx.doi.org/10.1016/ j.mimet.2010.06.004
- Dumke R, Schnee C, Pletz MW, Rupp J, Jacobs E, Sachse K, et al. *Mycoplasma pneumoniae* and *Chlamydia* spp. infection in community-acquired pneumonia, Germany, 2011–2012. Emerg Infect Dis. 2015;21:426–34. http://dx.doi.org/10.3201/ eid2103.140927
- Chalker VJ, Pereyre S, Dumke R, Winchell J, Khosla P, Sun H, et al. International *Mycoplasma pneumoniae* typing study: interpretation of *M. pneumoniae* multilocus variable-number tandem-repeat analysis. New Microbes New Infect. 2015;7:37–40. http://dx.doi.org/10.1016/j.nmni.2015.05.005
- Diaz MH, Benitez AJ, Winchell JM. Investigations of *Mycoplasma* pneumoniae infections in the United States: trends in molecular typing and macrolide resistance from 2006 to 2013. J Clin Microbiol. 2015;53:124–30. http://dx.doi.org/10.1128/JCM.02597-14
- Kenri T, Okazaki N, Yamazaki T, Narita M, Izumikawa K, Matsuoka M, et al. Genotyping analysis of *Mycoplasma pneumoniae* clinical strains in Japan between 1995 and 2005: type shift phenomenon of *M. pneumoniae* clinical strains. J Med Microbiol. 2008;57:469–75. http://dx.doi.org/10.1099/jmm.0.47634-0

Address for correspondence: Sabine Pereyre, USC EA3671 Mycoplasmal and Chlamydial Infections in Humans, University of Bordeaux, Campus Bordeaux Carreire, 146 rue Léo Saignat, 33076 Bordeaux, France; email: sabine.pereyre@u-bordeaux.fr

# Initial Costs of Ebola Treatment Centers in the United States

## Jocelyn J. Herstein, Paul D. Biddinger, Colleen S. Kraft, Lisa Saiman, Shawn G. Gibbs, Philip W. Smith, Angela L. Hewlett, John J. Lowe

Author affiliations: University of Nebraska Medical Center College of Public Health, Omaha, Nebraska, USA (J.J. Herstein, J.J. Lowe); Harvard Medical School, Boston, Massachusetts, USA (P.D. Biddinger); Emory University, Atlanta, Georgia, USA (C.S. Kraft); Columbia University Medical Center, New York, New York, USA (L. Saiman); Indiana University School of Public Health, Bloomington, Indiana, USA (S.G. Gibbs); University of Nebraska Medical Center College of Medicine, Omaha (P.W. Smith, A.L. Hewlett)

#### DOI: http://dx.doi.org/10.3201/eid2202.151431

To the Editor: The 2014–2015 outbreak of Ebola virus disease (EVD) in West Africa was unprecedented in scale and scope. During the outbreak, 11 patients with