Adjacent to the screening, a prescribing and dispensing area was established for patient examination. Privacy screens were used to create examination areas, and personal protective equipment, patient gowns, and linens were provided. Physicians staffed the clinic to assess patients who were concerned that they might have scabies. Supplies for skin scrapings and slide preparation were available, and a lab technician was on-call to collect specimens for processing.

More than 1,000 doses of permethrin were distributed during the clinics. There was 1 confirmed case of scabies and 3 probable cases of secondary transmission as a result of this outbreak.

Establishing a centrally located clinic using the incident command system structure provided rapid and effective screening and prophylactic treatment of scabies to prevent a larger outbreak of scabies within the institution.

Acknowledgments. None.

Financial support. No financial support was provided relevant to this article.

Conflicts of interest. All authors report no conflicts of interest relevant to this article.

Low prevalence of the *mcr-1* gene among carbapenemase-producing clinical isolates of *Enterobacterales*

Tanise Vendruscolo Dalmolin[1,2], Priscila Lamb Wink[1], Daiana de Lima-Morales[1] and Afonso Luís Barth[1,2]

[1]Laboratório de Pesquisa em Resistência Bacteriana, LABRESIS, Centro de Pesquisa Experimental, Hospital de Clínicas de Porto Alegre, Porto Alegre, RS, Brazil and [2]Programa de Pós-Graduação em Ciências Farmacêuticas, Faculdade de Farmácia, Universidade Federal do Rio Grande do Sul, Porto Alegre, RS, Brazil

To the Editor—Polymyxins are the last resort for the treatment of infections caused by multidrug-resistant bacteria, in particular carbapenem-resistant *Enterobacterales* (CRE). Resistance to polymyxins used to be due only to chromosomal mutations, but in November 2015, Liu et al[1] described for the first time a colistin resistance mechanism mediated by a new gene (*mcr*-1) that was present in a transferable plasmid. The *mcr*-1 has already been described on most continents, being detected in different species and obtained from several sources, including carbapenemase-producing clinical isolates.[2,3] Infections due to clinical isolates harboring the *mcr*-1 and a carbapenem resistance gene is of particular concern because the treatment options would be seriously compromised.[4]

The aim of this study was to evaluate the prevalence of carbapenemase and *mcr*-1 genes co-occurring among *Enterobacterales* clinical isolates in southern Brazil between April 2013 and May 2018.

We evaluated the occurrence of the *mcr*-1 gene among 4,778 isolates of *Enterobacterales* with reduced susceptibility to carbapenems obtained from an epidemiologic study in several hospitals in southern Brazil. All isolates were submitted to multiplex real-time polymerase chain reaction with high-resolution melting (RT-PCR-HRM) analysis with primers for bla_{KPC}, bla_{NDM}, $bla_{OXA\text{-}48\text{-}like}$, bla_{GES}, bla_{IMP}, and bla_{VIM} and presented positive results for at least 1 of the carbapenemase gene(s) tested.

The presence of the *mcr*-1 gene was evaluated by pooling 10 isolates together and submitting them to DNA extraction and conventional PCR with specific primers for the *mcr*-1 gene.[1] All isolates from a pool with *mcr*-1 positive result were retested individually by the same conventional PCR to identify the isolate (s) that presented the gene. The amplicons from the individual isolates with positive result in the conventional PCR were submitted to Sanger sequencing and were confirmed as the *mcr*-1 variant. The minimal inhibitory concentrations (MICs) of several antibiotics were evaluated using broth microdilution method for the individual isolates positive for the *mcr*-1 gene, and the results were interpreted according to European Committee on Antimicrobial Susceptibility Testing (EUCAST) guidelines.[5]

We found only 5 isolates that presented the *mcr*-1 gene and a carbapenemase gene. All coharboring isolates presented the *mcr*-1 and the bla_{KPC} genes. We obtained 2 coharboring isolates (*Klebsiella pneumoniae* 3111F and *Escherichia coli* 3431F) in 2014, 1 coharboring isolate (*E. coli* 5798F) in 2016, and the other 2 coharboring isolates (*K. pneumoniae* 6701F and *E. coli* 6699F) in 2018. All 5 isolates were recovered from rectal swabs, with exception of *E. coli* 6699F, which was recovered from an ascites fluid.

Moreover, 4 isolates presented low-level resistance to colistin (4 mg/L), and 1 isolate (*K. pneumoniae* 6701F) was susceptible to colistin (0.25 mg/L). All isolates were resistant to ertapenem, meropenem, imipenem, and ciprofloxacin and were susceptible to tigecycline. Susceptibility to aminoglycosides was variable, with most isolates susceptible to gentamicin and intermediate to amikacin (Table 1).

The prevalence of the *mcr*-1 gene was very low (0.1%) among carbapenemase-producing *Enterobacterales* (CPE) in our study. This rate is lower than that reported in Portugal, where 6.69% of the CPE isolates from colonized and infected patients were positive for the *mcr*-1 gene.[6] In Belgium, the prevalence reported was <1% among CRE of human origin.[7] These findings demonstrate that the prevalence of *mcr*-1 with carbapenemase genes is normally very low, although it can differ among countries.

The *mcr*-1 gene is usually evaluated only among colistin-resistant isolates (MIC >2 mg/L); however, the isolate *K. pneumoniae* 6701F was susceptible to colistin. Some isolates are *mcr*-1 positive; nonetheless they are colistin susceptible. One explanation for this is the assumption that the gene might be truncated in isolates positive for the *mcr*-1 but susceptible to

Author for correspondence: Afonso Luís Barth, LABRESIS, Hospital de Clínicas de Porto Alegre, Ramiro Barcelos 2350, Porto Alegre, Brazil. E-mail: albarth@hcpa.edu.br

Cite this article: Dalmolin TV, *et al.* (2019). Low prevalence of the *mcr-1* gene among carbapenemase-producing clinical isolates of *Enterobacterales*. *Infection Control & Hospital Epidemiology* 2019, 40, 263–264. doi: 10.1017/ice.2018.301

Table 1. Minimal Inhibitory Concentration (MIC)[a] of Several Antibiotics for *Escherichia coli* and *Klebsiella pneumoniae* Coharboring bla_{KPC}/*mcr*-1 Genes

Antibiotic	*E. coli* 3431F, MIC (mg/L)	*E. coli* 5798F, MIC (mg/L)	*E. coli* 6699F, MIC (mg/L)	*K. pneumoniae* 3111F, MIC (mg/L)	*K. pneumoniae* 6701F, MIC (mg/L)
Ertapenem	32	16	256	256	128
Meropenem	32	8	256	256	128
Imipenem	≥32	16	256	256	16
Ciprofloxacin	4	≥64	32	≥64	64
Amikacin	2	8	8	16	8
Gentamicin	1	2	4	32	0.5
Tigecycline	0.25	1	0.5	1	0.25
Colistin	4	4	4	4	0.25

[a]Performed by broth microdilution.

colistin, as already described in *Shigella sonnei*.[8] Interestingly, the truncated *mcr*-1 gene could be reactivated after conjugation experiments, resulting in a colistin resistant phenotype.[9] However, a truncated gene is not the only reason to justify the susceptibility; one study reported an *E. coli* colistin-susceptible *mcr*-1 positive isolate that presented an intact gene.[10] Therefore, further studies are needed to elucidate the reason why the *mcr*-1 gene of the *K. pneumoniae* 6701F in this study does not promote resistance to colistin. In fact, colistin-susceptible *mcr*-1 isolates may be contributing to a silent spread of the *mcr*-1 gene, which may be transferred to multidrug-resistant isolates such as the CPE described in this study.

In conclusion, the current prevalence of *mcr*-1 is very low, but the detection of 2 isolates in 2018 coharboring bla_{KPC}/*mcr*-1 genes is a warning for a possible increase in the prevalence of these isolates in the coming years. Considering the association of *mcr*-1 with the broad-spectrum resistance mechanisms (eg, carbapenemases), this emergence is of great concern.

Acknowledgments. The authors would like to thank Luiza Castro for technical support.

Financial support. This work was supported by Fundo de Incentivo a Pesquisa e Eventos do Hospital de Clínicas de Porto Alegre (FIPE/HCPA project no. 16-0559) and by a grant from the Conselho Nacional de Desenvolvimento Científico e Tecnológico—Brazil (CNPq).

Conflicts of interest. All authors report no conflicts of interest relevant to this article.

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https://doi.org/10.1017/ice.2018.301 Published online by Cambridge University Press