Sirs,

MRSA carrying the mec gene have recently been reported in cattle and humans.1 The discovery of this mecA variant raised the question of the prevalence of this gene, because mecA was not amplified by the mecA-specific primers routinely used to detect methicillin resistance. Thus, numerous retrospective and prospective studies were performed using new primers to look for mecC-positive MRSA, which were further detected in a variety of companion, food-producing and wild animals worldwide. These studies also highlighted geographical clusters of mecC-positive clones, zoonotic transmissions and even the potential role of water in mecC-positive MRSA dissemination.2-4

Horses have been reported as infected and/or colonized mainly by the mecA-carrying clonal complex (CC) 8 and CC398 so far. Here, we describe mecC-positive MRSA for the first time in horses. This finding should lead to the addition of equines to the list of animals potentially infected and/or colonized by mecC-positive MRSA. This development is of interest since the risk of zoonotic transmissions is likely to be increased due to close contact between horses and humans (veterinarians, riders or other people in contact with equines).

Antibiogram data from 936 non-duplicate coagulase-positive staphylococci isolated from horses were collected through the Resopath network (www.resopath.anses.fr) between January 2011 and December 2014. All presumptive MRSA detected by disc diffusion, i.e. cefoxitin-non-susceptible isolates (diameter <27 mm, according to the French Society of Microbiology Antibiogram Committee recommendations (http://www.sfm-microbiologie.org)), were sent to the National Reference Laboratory for Antimicrobial Resistance (Anses Lyon) for further analysis. Four of the isolates presented the mecC gene as detected by specific PCR.1 All isolates were characterized by spa typing (www.spaserver.ridom.de), MLST (http://saureus.mlst.net/) and microarray-based assay analysis (Staphylococcus aureus Genotyping, Identibac–Alere), allowing the detection of virulence and resistance genes and assignment to clones and/or CCs. No isolate presented associated resistance genes as shown by microarrays and disc diffusion susceptibility testing.

The first isolate was collected in the eastern region of France (Ain district) in October 2012 from a mare with a recurrent respiratory tract infection. The animal was first treated with penicillin, with no improvement. Eventually, an additional treatment with a sulphonamide/trimethoprim combination proved efficient. The MRSA isolate was assigned to the spa type t6220 (allelic profile 04-82-17-25-17-25-17-25) and ST1245 (CC130). The second MRSA isolate was sampled south-east of Paris in July 2013 from a horse with extensive recurrent skin infections. It was treated with penicillin and then with cefquinome plus non-antibiotic compounds, including corticosteroids, and its wounds slowly improved to complete healing. The strain additionally presented the sec and sel enterotoxin genes, harboured the t208 spa type (04-20-17-17-31-24-17-17-17-25) and belonged to ST49 (CC49). ST49 has recently been reported not only as a mecA-positive clone spreading among pigs in Switzerland, but also as a mecC-positive clone among patients in Belgium.5-6

The third isolate was collected in north-west France (Seine-Maritime district) in October 2013 from a 16-year-old gelding with a long-lasting pododermatitis. It was treated with injections of penicillin plus gentamicin followed by topical applications of cefquinome in addition to frequent and thorough washing with soap. The wound healed within 3 months. The MRSA strain was assigned to the spa type t11015 (04-17-25-17-16-17) and ST130 (CC130). The fourth isolate was sampled in the central area of France (Loiret district) in September 2014 from a 21-year-old mare. The animal had a large oozing foot wound that could not be cured with penicillin. A further treatment with cefquinome combined with topical non-antibiotic care (chlorhexidine, soap, fatty substances) slowly allowed the wound to heal. The strain harboured the t843 spa type (04-82-17-25-17-25-16-17-16) and belonged to ST130 (CC130).

These four cases represent the first report of horse-related mecC-positive MRSA. Three of them were sampled from recurrent local infections successfully treated with penicillin and/or with a broad-spectrum cephalosporin (cefquinome). Such an efficacy may be in accordance with data from a recent mouse model of endocarditis showing that some infections due to mecC-positive MRSA could be cured using B-lactams, especially when the antibiotic is massively administered directly at the site of infection.6 Off-label topical use of cefquinome in horses is still to be strongly discouraged since such a practice does not comply with the current national and international antibiotic-resistance control...
Clonal transmission of a colistin-resistant *Escherichia coli* from a domesticated pig to a human in Laos

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Sir,

Colistin, recently reintroduced in human medicine, is one of the most important antibiotics currently used to treat severe Gram-negative bacterial infections in humans. Unfortunately, it is also extensively used in animal production, including in swine and poultry farming against Gram-negative bacterial pathogens.1,2 However, the extensive use of antibiotics in food-animal production has been shown to increase the risk of transferring resistant bacteria to humans.3 In this study, we investigated the possible link between colistin-resistant *Escherichia coli* isolated from domesticated pigs and humans in a rural area in Laos.

In 2012, faecal samples were collected from 190 healthy individuals and 62 domesticated animals (44 free-range goats and 18 semi-free-range pigs) in Laos (GPS coordinates of latitude: 19°50’55.615”; longitude: 102°10′4.266”). Approval was obtained from the Ministry of Health Council of Medical Sciences, National Ethics Committee for Health Research Laos, number S1/NECHR. The samples were screened for colistin-resistant *E. coli* using Cepacia Medium (Becton Dickinson, Heidelberg, Germany) and isolates identified using MALDI-TOF MS. Colistin and polymyxin B MICs were determined by Etest on Mueller–Hinton II agar (bioMérieux, France) and microdilution with cation-adjusted Mueller–Hinton II broth (Becton Dickinson, Le Pont de Claix, France) and interpreted using EUCAST® and CLSI 2014 guidelines (M100-S24), respectively. Conjugation experiments were conducted as previously described using kanamycin.5 MLST was performed as described at http://mlst.warwick.ac.uk/mlst/dbs/Ecoli/documents/primerColi_html. Virulence typing was performed by targeting 10 virulence genes (*cpsMTII, papC, sfa/foc, afa/dra, iutA, iss, tsh, sitAP, ompT* and *hlyD*) and isolates classified as extraintestinal pathogenic *E. coli* (ExPEC) according to the criteria of Johnson et al.6 PFGE typing was performed as described previously7 using XbaI (Invitrogen, France) and 1.2% PFGE agarose gel (Sigma). Lastly, PCR and sequencing of the *mprAB, pbp2Q* and *mrgB* genes of colistin-resistant *E. coli* were carried out.8

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None to declare.

**References**


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