has been identified in a patient with no recent history of travel. As in this last case, the description here of bla_{NDM-1}-carrying *A. pittii* in France is made in a patient with no recent history of travel. The diagnosis was made fortuitously from a rectal swab sample. The context of this diagnosis suggests that the circulation of ACB species carrying bla_{NDM} may be underestimated in France.

This case raises questions about the management of patients with carbapenemase-producing *A. pittii* carriage in hospitals. Here, all the patients hospitalized in the same ward and screened for carbapenemase carriage were negative. Dissemination of NDM-1-producing *A. pittii* has been noted in an ICU.[7] NDM-producing *A. pittii* had then been isolated in the air within the ICU, being suspected to contribute to the dissemination of the bacterium. Therefore, early detection of carbapenemases in *Acinetobacter* species seems critical to control the dissemination of carbapenemase-producing isolates.

Here, this first French case of NDM-producing *A. pittii* in a patient with no history of travel enhances the problem of carbapenemase-producing ACB species and their management in hospitals.

Nucleotide sequence accession number

The sequence of the *A. pittii* G867 isolate has been deposited in GenBank (FKLP01000001–FKLP01000207).

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Transparency declarations

None to declare.

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Recurrent detection of VIM-1-producing *Escherichia coli* clone in German pig production

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Sir,

Carbapenems are declared as 'critically important' antibiotics by the WHO.[1] Recently, carbapenemase-producing Enterobacteriaceae (CPE) arose as a major concern in human medicine, where they are

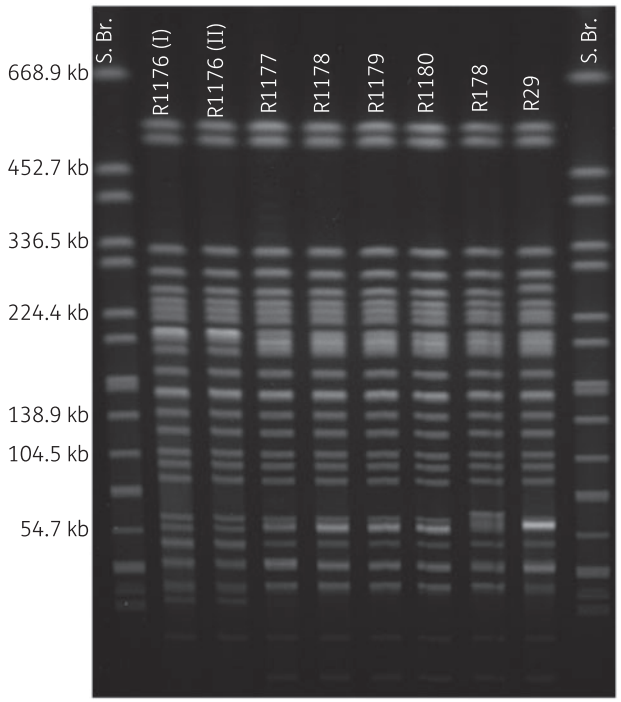

Figure 1. XbaI PFGE analysis. PFGE plugs of R1176 I and II were cast independently to verify reproducibility. R1176 was isolated in December 2015, R1177–R1180 were isolated in March 2016, and R178 and R29 are representatives of *E. coli* isolated in 2011–12. S. Br., *Salmonella* Braenderup (H9812) as size marker.

increasingly isolated from patients in hospitals. However, they are only sporadically reported from non-human sources.[2,3] In Europe, carbapenems are not licensed for veterinary use and no maximum residue levels are defined. Therefore, the isolation of VIM-1-producing *Escherichia coli* and *Salmonella* spp. in 2011–12 from German swine and poultry farms raised concerns that livestock might emerge as a reservoir for CPE and that pan-resistant isolates could be transmitted from this reservoir to humans.[4] In fact, spread and persistence of the β-lactamase gene (*bla*) VIM-1 over a whole fattening period was demonstrated in the VIM-1-positive swine farm. Here we describe the presence of VIM-1-positive *E. coli* isolates that appear closely related to the isolates from 2011, isolated in December 2015 from a different German swine farm.[4,5]

In Germany, monitoring of antimicrobial resistance (AMR) in commensal *E. coli* is linked to the national monitoring of zoonotic agents.[6] According to the Commission Implementing Decision 2013/652/EU, susceptibility is determined using the microdilution method following CLSI guidelines (CLSI M07-A9). Isolates with an ESBL/AmpC phenotype or phenotypic resistance to carbapenems are further characterized by PCR and subsequent sequencing of the PCR products.[5,7]

So far, no carbapenemase-producing *E. coli* isolates had been recorded within the scope of the national monitoring programmes. This study describes the first detection of a commensal *E. coli* isolate showing resistance to meropenem (MIC ≥0.5 mg/L), ertapenem (MIC ≥0.12 mg/L) and imipenem (MIC ≥2 mg/L) (isolate R1176, isolated in December 2015 from the colon content of a slaughter pig) within the monitoring programme. PCR and subsequent sequencing analysis revealed the presence of a bla_{VIM-1} gene. XbaI PFGE revealed a highly similar restriction pattern of R1176 to *E. coli* isolates described by Fischer *et al.*[4,5] from samples collected in 2011 from a swine farm (Figure 1; R29 and R178). This indicates a clonal relationship of these VIM-1-positive *E. coli*, although the affected livestock farms are regionally clearly separated. In contrast to *E. coli* isolates R29 and R178, in R1176 neither the bla_{ACC-1} gene nor the typical 220 kb VIM-1 plasmid of the former isolates was detected through PCR and S1 nuclease PFGE (Figure S1, available as Supplementary data at *JAC* Online). Supported by the failure of bla_{VIM-1} hybridization experiments on S1 nuclease PFGE (Figure S2) and transformation experiments, a chromosomal location of the bla_{VIM-1} gene in R1176 is assumed. This might be driven by an association of the bla_{VIM-1} gene with mobile genetic elements, as described for R178 as well.

To verify a potential clonal persistence of VIM-1-positive *E. coli* within the farm that the pig originated from, in March 2016 colon content from five healthy animals in another slaughter batch from this farm was examined. This resulted in the isolation of four additional carbapenem-resistant *E. coli* (isolates R1177–R1180) from one of the samples. Again, XbaI PFGE patterns of these four isolates were very similar to those mentioned above (Figure 1). This hints at the presence of a specific clone on this farm and a link with isolates obtained from the farm investigated in 2011.[4] S1 nuclease PFGE with subsequent hybridization experiments, described by Rodriguez *et al.*[7] in 2009, revealed bla_{VIM-1} localization on 180–200 kb IncHI2 plasmids in all four isolates (Figure S1). And, indeed, the VIM-1-plasmid-harbouring isolates were also positive for the bla_{ACC-1} gene, further resistance genes *strA* and *strB* and class-I-integron-associated resistance genes *aadA1* and *aacA4* and *sul1*, as shown for pRH-178, assuming the presence of a highly similar plasmid in these isolates compared with the ones from 2011.[4,5]

All five isolates described in this study belonged to ST88 and phylogenetic group A and harboured the bla_{VIM-1} gene on a class 1 integron with gene cassettes that were identical to those described for R29 and R178, independently of its localization on the plasmid or the chromosome.[5]

In the above-mentioned national AMR monitoring programmes no carbapenemase-producing *E. coli* had been detected until the end of 2015, indicating a very low prevalence of such bacteria in the German livestock population. However, detection of highly related VIM-1-producing *E. coli* isolates from an additional swine farm in Germany in this study indicates persistence of a VIM-1-producing *E. coli* clone in the swine population for at least 4 years. Further investigations on the persistence of this clone are currently under way. Detailed genomic analysis will be carried out to reveal a potential reason for stable maintenance of this clone and to uncover potential transmission pathways of these isolates. The understanding of transmission pathways and the persistence of CPEs among different populations to limit the spread of CPEs in livestock is of major relevance for public health. Finally, results of this study underline the importance of the carbapenemase monitoring recommended by the European Food Safety Authority (EFSA) and the European Commission.

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Transparency declarations

None to declare.

Supplementary data

Figures S1 and S2 are available as Supplementary data at *JAC* Online (http://jac.oxfordjournals.org/).

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MCR-1 in ESBL-producing *Escherichia coli* responsible for human infections in New Caledonia

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Sir,

Following the report on the plasmid-mediated colistin resistance gene *mcr-1* in Enterobacteriaceae in China from multiple sources,[1] this gene has been observed in *Escherichia coli*, *Klebsiella* spp., *Salmonella* spp. and *Shigella* spp. isolates, mostly of animal origin.[1–6] It has also been detected in humans in Asia, South-East Asia, Europe, the Middle East, North Africa and North and South America.[6]

In order to study the plasmid-mediated colistin resistance gene *mcr-1* in Enterobacteriaceae isolated from clinical specimens in France, we performed retrospective screening in a database of 610 whole-genome sequences of ESBL-producing Enterobacteriaceae isolates collected in French hospitals.

The bacterial collection included consecutive and non-repetitive strains representative of ESBL-producing Enterobacteriaceae observed in clinical samples collected in French hospitals in different geographic areas (Metropolitan France $n = 210$, Guadeloupe $n = 100$, Guyana $n = 100$, Reunion Island $n = 100$ and New Caledonia $n = 100$). The whole-genome sequences were assembled *de novo* at a $\geq 60\times$ coverage level from 2×150 bp paired-end reads produced by the NextSeq facility (Illumina, San Diego, CA, USA). WGS data were used to characterize the isolates according to antibiotic resistance genes and plasmid incompatibility with the CARD resistance gene database and the website of the Center for Genomic Epidemiology, respectively. The strains were genotyped by MLST using the Warwick University scheme.

We detected gene *mcr-1* in only two *E. coli* isolates, designated NC68 and NC101. These isolates were further tested for