Hospital-acquired infections with *Acinetobacter* spp. have been reported worldwide (13). Success of the organism is attributed to its ability for long-term survival in a hospital environment and its ability to rapidly acquire resistance to antimicrobials (2). Carbapenems have been the agents of choice for serious *Acinetobacter* sp. infections, but unfortunately the clinical utility of this class of antimicrobial is under threat with the emergence of acquired carbapenemases, particularly from Ambler classes B (metallo-β-lactamases) and D (oxacillinases) (10). Such mechanisms of resistance are being described in many geographic regions, including Australia (11). The class D carbapenemases found in *Acinetobacter* spp. have previously been divided into two distinct phylogenetic clusters, the OXA-23 cluster (OXA-23 and -27) and the OXA-24 cluster (OXA-24, -25, -26, and -40), with only 60% homology between them (1). More recently, a novel oxacillinase was described in France, OXA-58, in association with *Acinetobacter baumannii* infections in a burn unit (5, 12). To date, this enzyme has only been reported in Europe (8). A further novel oxacillinase has also been reported from Argentina, OXA-51 (3).

In March 2005, we identified a carbapenem-resistant *Acinetobacter junii* blood culture isolate. The organism was identified by using the VITEK GNI card (bioMérieux Vitek, Inc., Hazelwood, Mo.) and the result confirmed by the API 20NE system (bioMérieux, Inc., Hazelwood, Mo.). The isolate underwent susceptibility testing using broth microdilution according to CLSI standards (9, 4) and was sensitive to amikacin (MIC, 8 μg/ml), ciprofloxacin (MIC, 0.12 μg/ml), tetracycline (MIC, ≤2 μg/ml), and polymyxin B (MIC, ≤1 μg/ml). Intermediate resistance was observed for gentamicin (MIC, 8 μg/ml), piperacillin-tazobactam (MIC, 32 μg/ml), and ampicillin-sulbactam (MIC, 16 μg/ml). Resistance to imipenem, meropenem, ertapenem (all MICS, >8 μg/ml), cefazidime, cefepime (both MICS, >16 μg/ml), piperacillin (MIC, 128 μg/ml), ticarcillin-clavulanate (MIC, >128 μg/ml), and tobramycin (MIC, 16 μg/ml) was observed. Further testing, using a double-disk synergy test as described previously (11), identified that EDTA inhibited imipenemase activity, indicating the presence of a metallo-β-lactamase. To confirm the mechanism of carbapenem resistance, PCR was performed using primers specific for the *bla*<sub>IMP</sub> (14), *bla*<sub>VIM</sub> (14), and class D carbapenemase genes (OXA-23 and -24 cluster and OXA-58) (1, 12). Amplification products were identified for *bla*<sub>IMP</sub> and *blaoxa-58*. Nucleotide sequencing confirmed the presence of the *blaoxa-58* and *blaIMP-4* genes, with a complete sequence identified for each respective gene.

To our knowledge, this is the first report of both an OXA-58 outside of Europe and an *Acinetobacter* sp. carrying an OXA-58 and an IMP-4 gene. *A. junii* is a rare cause of disease, although documented cases of septicemia in adult and pediatric patients have been described (7). Also, *blaoxa-58* has been reported in a single isolate of *A. junii* (8). The *blaoxa-58* gene identified in France was found on a 30-kb plasmid, but in vitro conjugation experiments were unsuccessful using an *A. baumannii* recipient strain (5). Recently, it has been shown that OXA-58 activity contributes significantly to carbapenem resistance in *A. baumannii*, especially when additional efflux mechanisms are expressed (6). Our findings of multiple carbapenem-hydrolyzing β-lactamase genes in *Acinetobacter* spp. is worrying, the potential for spread and therapeutic failure being the primary concern. Further research is required to identify the genetic context of these genes and therefore understand the pathogenesis of spread. Incorporating strict infection control practices with controlled carbapenem use seems prudent at this stage.

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None of us has any conflict of interest.

**REFERENCES**


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