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Emergence and Clonal Dissemination of OXA-24- and OXA-58-Producing *Acinetobacter baumannii* Strains in Houston, Texas: Report from the SENTRY Antimicrobial Surveillance Program

We read with great interest the article by Shelburne et al. (4) describing two different clones of multidrug-resistant (MDR) *Acinetobacter baumannii* with distinct clinical outcomes. In that report, carried out in a university hospital in Texas, the authors detected the presence of two major clones (defined as A and B) that accounted for 61.7% of the *A. baumannii* isolates recovered during the study interval (4). Clone A was more likely to cause bacteremia, while clone B was related to peritonitis. Interestingly, these clones were recovered in different time periods.

We recently observed an increase in the carbapenem resistance among *A. baumannii* isolates from a medical center participating in the SENTRY Antimicrobial Surveillance Program, also located in the Houston, Texas, area. Since carbapenems are important antimicrobial agents for the treatment of infections caused by *A. baumannii*, we routinely screen for the production of acquired carbapenemases among carbapenem-resistant *A. baumannii* isolates collected and evaluated during 2007. These strains were recovered from patients admitted to the University of Texas Health Science Center (UTHSC), a component of the same medical center complex as the Ben Taub General Hospital (BTGH), from which the samples evaluated by Shelburne et al. (4) were collected.

Among 23 *A. baumannii* isolates from UTHSC, 13 (56.5%) strains showed nonsusceptibility against imipenem and meropenem (MIC, ≥8 μg/ml) according to the breakpoints defined by the Clinical Laboratory Standards Institute (CLSI, formerly the NCCLS) (1). These isolates, like the clusters evaluated by Shelburne et al., were MDR, showing resistance to all antimicrobial agents tested, with the exception of polymyxins (polymyxin B and colistin) and tigecycline (MIC_{50}, 2 μg/ml; MIC_{90}, 4 μg/ml). Carbapenem-resistant isolates were screened for the presence of metallo-β-lactamase production by using metallo-β-lactamase Etest strips (AB Biodisk, Solna, Sweden) according to the manufacturer's instructions. All isolates showed negative results.

Oxacillinases (OXA series enzymes) with carbapenemase activity-encoding genes were tested by multiplex PCR, as previously described (5). Eleven of the 13 carbapenem-resistant strains were found to carry genes encoding acquired oxacillinases. Six isolates possessed bla_{OXA-24}, and five strains carried bla_{OXA-58} (Table 1). All strains were positive for bla_{OXA-51}, the intrinsic enzyme-encoding gene characteristic of *A. baumannii*. We also evaluated the associations of bla_{OXA} and the insertion sequences (IS) IS*Aba-1*, *-2*, and *-3* by using custom primers anchoring in the oxacillinase-encoding genes and in the IS elements. These genetic structures have been associated with carbapenem-hydrolyzing oxacillinases and, when located upstream of bla_{OXA}, supply a transcriptional promoter that upregulates the expression of these β-lactamase genes (2, 3). All bla_{OXA-58} genes were located downstream of the IS*Aba-3* element; however, bla_{OXA-24} was not associated with any of the three types of IS tested. In the two isolates that showed negative amplification for acquired carbapenemases, bla_{OXA-51} was associated with IS*Aba-1*, which may have contributed to the increased carbapenem MICs.

A. baumannii strains with elevated carbapenem MIC results were evaluated for clonality by pulsed-field gel electrophoresis (PFGE). Isolates showing ≤3-band differences were considered identical or similar (subtypes). The *A. baumannii* isolates were clustered in three different molecular epidemiology patterns (A, B, and C), two of them showing subtypes (Table 1). Isolates belonging to clone A (A and A1) were generally found to produce OXA-24 and isolates from clone C were associated with OXA-58 production. The two isolates that were positive only for bla_{OXA-51} showed similar PFGE profiles but were distinct from the dominant clones (A and C).

Two *A. baumannii* isolates (one from each reported clone) from BTGH (4) were evaluated and compared with the oxacillinase-producing isolates from UTHSC detected in the present study. The isolates from the earlier study (4) were genetically different from these recent carbapenem-resistant strains in

TABLE 1. Characteristics of the 13 carbapenem-resistant *A. baumannii* isolates evaluated from a participant Texas medical center during 2007 in the SENTRY program

Isolate	Specimen type	Culture date (mo/day)	Service	PFGE pattern	Acquired bla_{OXA} gene[a]
86	Blood	01/02	Cardiothoracic/pulmonary	B	None
2539	Sputum	08/18	Surgery	B1	None
85	Blood	01/05	Orthopedics	C	bla_{OXA-58}
548	Blood	02/09	Family practice	C	bla_{OXA-58}
555	Blood	02/10	Family practice	C	bla_{OXA-58}
553	Blood	2/11	Family practice	C	bla_{OXA-58}
1323	Blood	03/05	Family practice	C	bla_{OXA-58}
546	Blood	02/10	Family practice	A	bla_{OXA-24}
550	Blood	02/09	Family practice	A1	bla_{OXA-24}
2545	Sputum	08/17	Internal medicine	A	bla_{OXA-24}
2522	Sputum	08/23	Internal medicine	A	bla_{OXA-24}
12533	Blood	11/14	Internal medicine	A	bla_{OXA-24}
1322	Blood	03/9	Family practice	C	bla_{OXA-24}

[a] All strains were positive for bla_{OXA-51}.

PFGE pattern (data not shown). However, PCR experiments for oxacillinase-encoding genes demonstrated that one of the isolates was positive for $bla_{\text{OXA-24}}$.

This complementary investigation demonstrates the recent dissemination of MDR *A. baumannii* strains clones carrying oxacillinases with carbapenemase activity in a Houston, T, medical center. These isolates were not genetically related to those evaluated in a collection from 1995 to 2004, illustrating the diversity and continued evolution of MDR *A. baumannii* strains through 2007. Among 10 strains collected from bloodstream infections, different clones and distinct bla_{OXA} genes were detected, showing little evidence of correlation to clinical outcomes as observed by Shelburne et al. (4). Also in contrast to the earlier sample (4), in our study, the clones were concomitantly recovered in the same time intervals during 2007.

The presence of $bla_{\text{OXA-24}}$ in one of the isolates evaluated in a prior time period (1995 to 2004) shows that this is a persistent resistance problem in this hospital complex and, more importantly, that these oxacillinases (OXA-24 and OXA-58) have rapidly become responsible for carbapenem and MDR patterns among *A. baumannii* isolates that currently have few treatment options.

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Mariana Castanheira*
JMI Laboratories
345 Beaver Kreek Centre, Suite A
North Liberty, Iowa 52317

Audrey Wanger
Mark Kruzel
University of Texas Health Science Center
Houston, Texas 77030

Lalitagauri M. Deshpande
Ronald N. Jones
JMI Laboratories
North Liberty, Iowa 52317

*Phone: (319) 665-3370
Fax: (319) 665-3371
E-mail: mariana-castanheira@jmilabs.com

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