**First Carbapenem-Resistant Isolates of *Acinetobacter soli* in Japan**

*Acinetobacter* sp. has emerged as a major hospital pathogen (8). The greatest concern has been the emergence of carbapenem resistance in *Acinetobacter baumannii* by the acquisition of OXA-type carbapenemase or metallo-β-lactamases, since few effective antimicrobial agents exist. Several mechanisms can underlie carbapenem resistance in *A. baumannii* (1), but less is known about carbapenem resistance in non-*A. baumannii* species (4). There have been only three reports about *Acinetobacter soli* (5, 6, 8), with no mention of carbapenem resistance. We isolated carbapenem-resistant *A. soli* from two Japanese patients with bloodstream infections.

In January and April 2011, carbapenem-resistant *A. soli* was isolated from blood cultures of two patients at the Tohoku University Hospital. A central venous catheter was in situ in both cases. The species was identified by partial sequencing of the RNA polymerase β-subunit (*rpoB*) gene (7). MICs were determined by the agar dilution method of the Clinical and Laboratory Standards Institute (2).

To detect OXA-51-like, OXA-23-like, OXA-24-like, and OXA-58-like carbapenemase genes and IMP-1-, IMP-2-, VIM-1-, VIM-2-, SIM-, and NDM-1-type metallo-β-lactamase genes, PCR was performed (3, 9). The proximity of IS*Ab1*, IS*Ab2*, IS*Ab3*, and IS18 to *bla*OXA-58-like gene (9) and the *carO* (outer membrane protein) gene (1) was investigated by PCR. The OXA-type carbapenemase and metallo-β-lactamase genes were sequenced. Pulsed-field gel electrophoresis (PFGE) was done with the *Smal* restriction enzyme (11). Isolates with >80% similarity were considered to be within the same cluster (10).

The MIC of imipenem was ≥16 μg/ml for both isolates (Table 1). PCR showed that one isolate possessed only the IMP-1 gene, while the other had both IMP-1 and OXA-58-like genes. No other carbapenem resistance genes were detected. The OXA-58-like carbapenemase gene was not linked to IS*Ab1*, IS*Ab2*, IS*Ab3*, or IS18. Sequencing of the *bla*OXA-58-like and *bla*IMP-1 genes yielded OXA-58 and IMP-1, respectively. Both isolates exhibited decreased expression of *carO*. Thus, the mechanism of resistance in one of these isolates could involve a synergistic interaction between IMP-1 expression and reduced expression of an outer membrane protein. The two isolates had different PFGE patterns (not shown).

Currently, 33 genomic species of the *Acinetobacter* genus have been identified by molecular methods (5). *A. baumannii* is generally the pathogen isolated most frequently in clinical settings, although it is difficult to perform accurate species identification at many institutions. Recently, sequencing has provided reliable identification of *Acinetobacter* isolates to the species level in laboratories (7), and severe infections caused by non-*A. baumannii* clinical isolates have been reported (5, 8). To our knowledge, however, carbapenem-resistant *A. soli* isolates have not been reported previously.

Three *Acinetobacter* isolates with imipenem MICs of ≥16 μg/ml were obtained from blood cultures at the Tohoku University Hospital over the past 5 years, and two of these isolates were identified as *A. soli* by partial *rpoB* gene sequencing. This indicates that carbapenem resistance is now present among clinical isolates of *A. soli*, and we should monitor its prevalence. The present findings emphasize the importance of performing accurate epidemiological investigation of non-*A. baumannii* species, including *A. soli*.

### Acknowledgment
We thank laboratory members for technical support.

### References

### Table 1 Antimicrobial susceptibilities of two *Acinetobacter soli* isolates from blood cultures

<table>
<thead>
<tr>
<th>Antibiotic agents</th>
<th>MIC (μg/ml)</th>
<th>Case 1</th>
<th>Case 2</th>
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<tr>
<td>Ampicillin</td>
<td>32</td>
<td>8</td>
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</tr>
<tr>
<td>Ampicillin-sulbactam</td>
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<td>8</td>
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<tr>
<td>Piperacillin</td>
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</tr>
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<tr>
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</tr>
<tr>
<td>Ceftazidine</td>
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<td>≥256</td>
<td>128</td>
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<tr>
<td>Cefepime</td>
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<td>≥256</td>
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<tr>
<td>Imipenem</td>
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<td>8</td>
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<tr>
<td>Meropenem</td>
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<tr>
<td>Amikacin</td>
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<td>128</td>
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<tr>
<td>Nalidixic acid</td>
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<td>Levofloxacin</td>
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<tr>
<td>Colistin</td>
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</tbody>
</table>

**LETTER TO THE EDITOR**

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