tween these 2 groups, statistically different parameters from the univariate analysis included the number of days that a urinary catheter was in place, the number of days that an intravascular catheter was in place, the number of mechanical ventilation days, length of stay in an intensive care unit, transplantation status, and use of third-generation cephalosporins, trimethoprim, vancomycin, or fluoroquinolones during the 14 days before a clinical specimen culture was positive. Kaplan-Meier analysis showed a significantly increased mortality in the A. baumannii group, even after adjusting for stay in an intensive care unit (P = .0002).

With the above-mentioned limitations in mind, we believe that colonization or infection with MDR A. baumannii may be an independent risk factor for increased mortality.

Acknowledgments


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References


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Table 1. PCR primers and products used in sequence analysis of IS1016 insertion elements from Haemophilus influenzae type b strains 37, 149, and 205, compared with those from the reference sequence AF549213.

<table>
<thead>
<tr>
<th>Primer set</th>
<th>Nucleotide sequence (5′ to 3′)</th>
<th>Positiona</th>
<th>Region amplified</th>
<th>Size, bp</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rd sec97/leftr bexA</td>
<td>AGTTTCAGGGGTAGATCTCAGCG</td>
<td>34–57</td>
<td>IS1016-V5</td>
<td>477</td>
</tr>
<tr>
<td></td>
<td>CAATGATTCCGCTAATAATGT</td>
<td>510–489</td>
<td>IS1016-V5</td>
<td></td>
</tr>
<tr>
<td>Region III orf3 bexA</td>
<td>TGTTCCTCGTGCAACTTGG</td>
<td>16884–16903</td>
<td>IS1016-V2, in the bridge region</td>
<td>1724</td>
</tr>
<tr>
<td></td>
<td>CAATGATTCCGCTAATAATGT</td>
<td>18608–18587</td>
<td>IS1016-V2, in the bridge region</td>
<td></td>
</tr>
<tr>
<td>Region III orf3 bexA</td>
<td>TGTTCCTCGTGCAACTTGG</td>
<td>34981–35000</td>
<td>IS1016-V2, at the 3′ end of cap b locus</td>
<td>931</td>
</tr>
<tr>
<td>Rd sec97/right</td>
<td>AATTGGGGGGAAGGTAAGGA</td>
<td>35912–35893</td>
<td>IS1016-V2, at the 3′ end of cap b locus</td>
<td></td>
</tr>
</tbody>
</table>

a Position on AF549213.

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We read with interest the article by Kapogiannis et al. [1] describing 2 cases of invasive Haemophilus influenzae infection caused by serotype a isolates possessing the putative virulence-enhancing IS1016-bexA partial deletion. Such a deletion is usually observed in invasive H. influenzae type b (Hib) strains, in which the capsulation (cap) locus lies between direct repeats of the IS1016 insertion element and contains a duplication of the genes carrying a 1.2-kb deletion at the 5′ end of the cap repeat within the IS1016 element and the bexA gene [2, 3]. It has been hypothesized that the IS1016-bexA deletion stabilizes the gene duplication, resulting in increased capsule production and virulence [4]. It has also been suggested that direct repeats of IS1016 provide a molecular substrate for further cap gene amplification [2, 5]. We are particularly concerned with this issue, because strains harboring >2 copies of the cap b locus (multiple-copy strains) have been recently observed in Italy [6]. No data were available for the IS1016 sequence in amplified cap b loci.

Here, we report the detection of a new Hib clone exhibiting remarkable polymorphism within its IS1016 sequences, with further deletions and point mutations in addition to the usual IS1016-bexA deletion. This clone included 3 Hib isolates (strains 37, 149, and 205) recovered from children aged ≈24 months who had severe invasive disease and who had not received Hib conjugate vaccine. All 3 isolates were found to contain 4 copies of the cap b locus by Southern blotting [6]. In each isolate, the IS1016 element at the 5′ end (IS1016-V5), the “inner” IS1016-V2 elements, and the IS1016-V2 element at the 3′ end were separately amplified by PCR, employing 3 sets of primers complementary to the flanking genes [7] (table 1). Sequencing of the IS1016-V5 PCR products revealed 100% identity in nucleotide sequences among the 3 isolates. In comparison with the reference sequence (GenBank accession number AF549213), the IS1016-V5 sequences herein described (EMBL accession numbers AM268182–AM268184) exhibited 2 new deletions (a 40-bp deletion within a previously described open reading frame [2] plus a single nucleotide deletion within the 19-nucleotide inverted repeat bracketing the
open reading frame) in addition to the usual IS1016-bexA deletion, together with several point mutations (figure 1A). Sequencing of the IS1016-V2 PCR products (EMBL accession numbers AM268187–AM268189) indicated that (1) in each isolate, the inner IS1016 elements were identical in sequence to the one at the 3′ end, identical in sequence. Strain code numbers are indicated on the right. Underline, start codons; double underline, stop codons; arrows, the 19-nucleotide inverted repeats; gray areas, deletions.

Figure 1. A, Alignment of IS1016-V5 elements. B, Alignment of “inner” IS1016-V2 elements representative also of those at the 3′ end, identical in sequence. Strain code numbers are indicated on the right. Underline, start codons; double underline, stop codons; arrows, the 19-nucleotide inverted repeats; gray areas, deletions.

Prevalence of Influenza B during the 2004–2005 Season in Japan

To the Editor—we appreciate Dr. Baum’s comments [1] regarding our study [2] comparing the effectiveness of oseltamivir for the treatment of influenza A and influenza B. Baum mentions that “the first [factor]—unexplained—is the relatively high incidence of influenza B. It is not clear whether many more influenza A cases were in fact identified but not studied, or whether almost one-half of the cases of influenza identified were indeed influenza B. The latter would be very unusual” [1, p. 446]. We would like to comment about this. Via the internet, we collect almost all cases that are diagnosed

 References

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