Acinetobacter baumannii is a ubiquitous pathogen capable of causing both community and health care–associated infections (HAIs), although HAIs are the most common form. This organism has emerged recently as a major cause of HAI because of the extent of its antimicrobial resistance and its propensity to cause large, often multifacility, nosocomial outbreaks. The occurrence of outbreak is facilitated by both tolerance to desiccation and multidrug resistance, contributing to the maintenance of these organisms in the hospital environment. In addition, the epidemiology of A. baumannii infection is often complex, with the coexistence of epidemic and endemic infections, the latter of which often is favored by the selection pressure of antimicrobials. The only good news is that potentially severe A. baumannii infection, such as bacteremia or pneumonia in patients in the intensive care unit who are undergoing intubation, do not seem to be associated with a higher attributable mortality rate or an increased length of hospital stay.

Acinetobacter baumannii is a ubiquitous pathogen capable of causing both community and health care–associated infections (HAIs) that has recently emerged as a major cause of HAI, because of its propensity to accumulate mechanisms of antimicrobial resistance that lead to pan-drug resistance and cause large HAI outbreaks that often involve multiple facilities. A. baumannii mainly causes pulmonary, urinary tract, bloodstream or surgical wound infections. Major risk factors include invasive procedures, such as the use of mechanical ventilation, central venous or urinary catheters, and broad-spectrum antimicrobials.

However, knowledge about A. baumannii is much less developed than knowledge about other opportunistic pathogens, such as Pseudomonas aeruginosa, as demonstrated by a search of the Medline database performed on 1 April 2005, which revealed 30,247 citations for P. aeruginosa versus only 703 for A. baumannii. Another important limitation is the difficulty in correctly identifying this organism, leading to the publication of data that are of questionable value. However, available data suggest that A. baumannii is a remarkable microorganism because of the diversity of its habitat, the way it accumulates mechanisms of antimicrobial resistance, its resistance to desiccation, its propensity to cause outbreaks of infection, and the complexity of its epidemiology. Therefore, in this review, we will focus on these unusual features and on unresolved issues.

**TAXONOMY**

The genus Acinetobacter consists of strictly aerobic, gram-negative coccobacillary rods that grow at 20°–30°C on usual laboratory media. Bacteria classified as members of the genus Acinetobacter have a long history of taxonomic changes, moving from the family Neisseriaceae to the family Moraxellaceae. Within the genus Acinetobacter, studies based on DNA/DNA hybridization have resulted in the description of 25 “genomic species” that fulfilled the criteria of 25 “genomic species” that fulfilled the criteria to be considered distinct species, 17 of which have officially been validated to date (http://www.bacterio.cict.fr/a/acinetobacter.html). Only 10 species have been given names, but differential biochemical and growth tests have been published for 19 species.

Schreckenberger and colleagues, authors of The Manual of Clinical Microbiology [1], believe that the majority of the genomic species cannot be reliably separated by phenotypic tests. However, because it is difficult to differentiate the isolates according to their phenotypic characteristics, the term “A. calcoaceticus–A. baumannii” complex is often used.
**HABITAT**

*A. baumannii* is ubiquitous in nature and has been recovered from soil, water, animals, and humans. *Acinetobacter* species are normal inhabitants of human skin and are frequently isolated from the throat and respiratory tract of hospitalized patients. For this reason, it has been suggested that human skin could be the source of severe infections, such as bacteremia. However, this hypothesis has not been confirmed in healthy humans. Berlau et al. [2] have studied the distribution of *Acinetobacter* species on the skin of 192 healthy volunteers and found that if 40% of volunteers carried *A. baumannii* numbers and that *A. baumannii*–*A. calcoaceticus* complex accounted for 56% of all strains isolated. In contrast, recent studies have shown that *A. baumannii* can be found in unsuspected reservoirs, such as food or arthropods, and additional reservoirs remain to be discovered.

From 1 January 2002 to 31 August 2004, *A. baumannii* grew from blood samples obtained from 102 patients hospitalized at military medical facilities treating service members injured in Afghanistan and the Iraq-Kuwait region. The sources of *A. baumannii* that led to the infections are still unknown, but during the Vietnam War, *A. baumannii* was the most common gram-negative bacillus recovered from traumatic injuries to extremities. Recent reports [3–7] also have identified *A. baumannii* infections in patients who suffered traumatic injuries, suggesting environmental contamination of wounds as a potential source, even if such a source is still unknown.

Food is known to be a source for gram-negative rods, such as *Escherichia coli*, but if *Acinetobacter* species isolates have been found in a variety of food items, including raw vegetables, few data exist about the presence of *A. baumannii* in the food chain. Therefore, Berlau and colleagues have sought to assess the distribution and frequency of *Acinetobacter* genospecies in a variety of purchased or harvested fresh fruits and vegetables [8]. Results showed that 17% of vegetables grew *Acinetobacter* in small numbers and that *A. baumannii* and genospecies 11 were the species most frequently isolated. The *A. baumannii*–*A. calcoaceticus* complex accounted for 56% of all strains isolated from fruits and vegetables and were found in apple, melon, bean, cabbage, cauliflower, carrot, potato, radish, lettuce, cucumber, pepper, mushroom, and sweet corn. According to Berlau et al. [8], hospital food could be a potential source for *A. baumannii* acquisition. In addition, studies have shown that digestive-tract colonization may be common in the hospital environment, with colonization rates as high as 41% in patients in the intensive care unit [9].

In a study seeking to isolate *Bartonella quintana* from body lice collected from homeless persons in Marseille, France, La Scola et al. [10] isolated and genotyped 40 *A. baumannii* strains, of which 21 were surprisingly susceptible to ampicillin. The genotypes of those strains were limited to 2 clones, including 1 that was susceptible to ampicillin. *A. baumannii* DNA was later detected in 21% of 622 lice collected worldwide, leading to the conclusion that there is an epidemic of *A. baumannii* infection among human body lice, which may be a source of human *A. baumannii* infection [10].

**DESSICATION TOLERANCE**

Several investigators have suspected that the survival of *A. baumannii* in the environment could contribute to the transmission of the organism during outbreaks. This hypothesis has been confirmed by several studies. By replicating hospital conditions, Jawad et al. [11] have shown that *A. baumannii* organisms were able to survive for an average of 20 days at a relative humidity of 31%. Other investigators showed that *A. baumannii* strains could be isolated from a hospital bed rail 9 days after the discharge of an infected patient from the hospital [12]. In another study, Wendt et al. [13] have shown that the ability of *A. baumannii* strains to survive under dry conditions varied greatly and was correlated with the source of the strains. Strains isolated from dry sources had better survival rates than strains isolated from wet sources. Of interest, Houang et al. [14] have shown that *A. baumannii* underwent morphological changes when desiccated with cells that had significantly thicker and more electron-dense cell walls and nucleic acid than those of control specimens. Even if such an environmental source does not explain per se the transmission of an epidemic strain, it suggests that hospital equipment could serve as a secondary reservoir for *A. baumannii* [15]. The potential role played by the capacity of *A. baumannii* to survive in the hospital environment in the spread of epidemic strains is suggested a posteriori by the success of control measures, including environmental decontamination with hypochlorite solutions. However, it is obvious that environmental decontamination is only one measure among many and that the independent role in infection control of this approach has never been evaluated [16].

**ANTIMICROBIAL RESISTANCE AND GENETICS**

More than in its virulence characteristics, the main danger associated with *A. baumannii* resides in its capability to acquire antimicrobial-resistance genes extremely rapidly, leading to multidrug resistance [17]. As a result, the management of *A. baumannii* infection has become a public health problem in many countries [18]. Of the 102 *A. baumannii* strains isolated from infections in service members injured in Afghanistan and the Iraq-Kuwait region, a high degree of antimicrobial resistance was discovered, including resistance to all drugs found in 4% of isolates [3]. In France, an outbreak of nosocomial infections occurred from 1 April 2003 to 31 May 2004 [19] and was caused by a multidrug-resistant *A. baumannii* isolate,
named AYE, which expressed the VEB-1 extended-spectrum β-lactamase and was susceptible only to imipenem, ticarcillin-clavulante, and piperacillin-tazobactam.

It is estimated that the acquisition of resistance mechanisms by *A. baumannii* strains is a recent phenomenon that started in the 1970s [17]. This extremely rapid development of antimicrobial resistance is likely to result from the ability of *A. baumannii* to respond rapidly to challenges issued by antimicrobials, coupled with the widespread use of antimicrobials in the hospital environment. In particular, the influence of the wide use of extended-spectrum cephalosporins and quinolones has been demonstrated [20–21].

**Mechanisms of resistance.** Several mechanisms have been shown to play a role in the acquisition of a multidrug-resistance phenotype by *A. baumannii*, including the acquisition of mobile genetic elements, such as plasmids [22], transposons [23], integrons [19–24], and natural transformation [25]. The role of plasmids in the acquisition of antimicrobial resistance in *A. baumannii* is mostly related to the integron structures they carry. The presence of class I and class II integrons in *A. baumannii* has been significantly associated with multiple antibiotic resistance, and the nosocomial spread of isolates [19, 23, 24, 26] has been described in both clinical and environmental strains of Acinetobacter baumannii worldwide [27–32]. These integrons are mostly acquired by contact with bacteria present in similar environments, such as Enterobacteriaceae [28] or Pseudomonas species [19, 33, 34]. One of the most striking examples of integron transfer between *Pseudomonas* species and *A. baumannii* is the acquisition of the class I integron carrying the VEB-1 extended-spectrum β-lactamase and 6 other antimicrobial resistance genes, which were acquired from *P. aeruginosa* (figure 1) [19]. In addition to the acquisition of mobile genetic elements, *Acinetobacter* species show a high frequency of natural transformation [35–37]. Natural transformation in *A. baumannii* is made easier by homology-facilitated illegitimate recombination that enables stable integration within the chromosome of antimicrobial resistance markers carried by plasmids, followed by loss of plasmid markers [23, 25, 38–40]. The short-term accumulation and combination of several of these mechanisms in *A. baumannii* strains facilitate the coselective process under different antimicrobial selective pressures [41].

To date, *A. baumannii* has become resistant to almost all antimicrobial agents that are currently available [42, 43]. Resistance mechanisms involve antimicrobial-degrading enzymes, efflux pumps, target modification, and porin deficiency. One of the main concerns about antimicrobial resistance in *A. baumannii* has been the acquisition of carbapenem resistance, mainly through the acquisition of B and D class carbapenemases [18, 19, 26]. The latter resistance deprives physicians of one of the most active antimicrobials against *A. baumannii*.

Many reports have also described an increase in the heavy-metal resistance mechanisms in *A. baumannii* [23]. For example, mercury-resistance operons are widely distributed among both clinical and environmental bacteria, to which the operons are introduced by transposons [44]. As for arsenic-resistance operons, which have been described as encoding low or high levels of resistance to both arsenic and antimony, they are also widespread in environmental strains of *A. baumannii* [45].

Recently, the first genome-based study has been conducted to identify all resistance mechanisms accumulated by the AYE strain epidemic in France [46]. The authors identified an unexpected 86-kb resistance island, the largest in a bacterium to date, which contained 45 clustered resistance genes. In contrast, 3 plasmids also identified in the bacterium were free of any resistance marker.

**INFECTION**

**Types of infection.** Many *A. baumannii* organisms isolated from respiratory secretion and urine specimens obtained from hospitalized patients demonstrate colonization rather than infection. Most infections involve organ systems with a high fluid content, such as the respiratory tract, peritoneal fluid, and the urinary tract, and are associated with indwelling devices. Pathological changes that are observed depend on the organ involved and are not distinguishable from changes caused by other aerobic gram-negative bacilli.

The distribution of the different types of infection varies from one hospital to another and is probably related to the hospital population and the type of procedures and interventions performed. A prospective regional study performed in Spain of

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**Figure 1.** Schematic representation of class I integron found in Acinetobacter baumannii strain AYE. Gene cassettes are shown as boxes, with arrows indicating the orientation of transcription and black circles indicating the 5'9-base element. The *intI1* gene is an integrase. Filled and empty triangles indicate IS1999 inverted repeats. The resistance determinants carried by the integron are as follows: β-lactams, VEB-1 extended spectrum β-lactamase and OXA-10 (oxacillinase); aminoglycosides, aadB and aadA1 (adenyltransferases); rifampin, arr-2 (ADP-ribosylating transferase); chloramphenicol, cmI5 (major facilitator superfamily efflux pump); sulphonamides, sul1; and antisepsics, gacEΔ1 (small multidrug resistance family efflux pump). Adapted from [16] with permission.
240 A. baumannii infections showed that >90% of infections were nosocomially acquired and that only 4% were community acquired. In this study, respiratory tract infections were the most common (39%), followed by exudates and abscesses (24%) each and urinary tract infections (23%). Of interest is that bacteremia represented only 3% of all infections [47].

A different distribution of infections was observed in a retrospective study performed in a 3956-bed teaching hospital in Marseille, France, during a 3-year period (1 January 2002–31 December 2004). In this study, 656 A. baumannii infections were identified, and exudates and abscesses were the most common infections (32%), followed by urinary tract infections (25%), respiratory tract infections (20%), and bacteremia (12%). This study also showed seasonality in the occurrence of A. baumannii infection during the 3-year period, with a significantly greater number of infections occurring from July through September (mean number of infections per month ± SD, 22 ± 6) than from January through March (mean number of infections per month ± SD, 13 ± 5; P = .005) (H. Richet, unpublished data). The reason for this seasonality is unknown, but it has also been observed by McDonald et al. [48], who, by analyzing the National Nosocomial Infections Surveillance System data for a 10-year period, showed that rates of Acinetobacter species infections were significantly higher during the July–October period and that this increase was mostly attributable to bloodstream infections and pneumonia and to infections occurring in the New England region.

**Outcome of A. baumannii infection.** Little is known about the genes and factors involved in virulence properties besides that siderophore activity and adherence properties play a role in the occurrence of colonization and infection [49]. Therefore, the only way to assess the virulence of A. baumannii is indirectly and consists of assessing the outcome of infections. Rates of mortality ranging from 5% in general wards to 54% in intensive care units have been associated with A. baumannii infections, suggesting the existence of a certain amount of virulence in this bacteria [50–51]. However, those are crude data, and few studies have sought to assess the attributable mortality and to evaluate the role of comorbidities in the outcome. Blot et al. [52] have sought to assess outcome and attributable mortality in critically ill patients with nosocomial A. baumannii bacteremia by performing a matched case-control study comparing patients with A. baumannii bacteremia in the intensive care unit with nonbacteremic patients in the intensive care unit. Matching criteria included having an equivalent APACHE II score, having the same principal diagnosis leading to the admission to the intensive care unit, and having the same length of stay in the intensive care unit. The crude mortality rate was 42.2% among case patients and 34.4% among control patients, resulting in an attributable mortality of 7.8% for A. baumannii bacteremia that was not statistically significant. In multivariate survival analysis, older age was the only variable significantly associated with in-hospital mortality, and A. baumannii bacteremia was not an independent predictor of mortality [52]. Another study seeking to assess the clinical impact of pneumonia caused by A. baumannii by using a matched case-control study approach showed the same type of results. Among patients in the intensive care unit undergoing intubation, pneumonia due to A. baumannii was not associated with significant differences in either attributable mortality rate or an increased length of stay in the intensive care unit [53].

**Frequency of nosocomial infections.** Data exist at the national, regional, or local level. At the national level, a prevalence survey performed in France in June 2001 in 1533 health care facilities containing 381,303 beds and including 305,656 patients showed a prevalence of A. baumannii infection of 0.075%. A. baumannii was the 15th most frequently isolated microorganism from nosocomial infections, representing only 1.2% of all microorganisms [54].

In a regional prospective study performed in a network of 28 health care facilities in Spain serving a population of 11,000,000 people, the overall incidence of A. baumannii infection and/or colonization was 0.39 cases per 1000 patient-days, ranging from 0 to 1.17 cases per 1000 patient-days, depending on the health care facility, and ranging from 0.14 to 4.55 cases per 1000 patient-days in intensive care units. Only 3 patients were pediatric. Among the 206 patients, 53% of cases were infection, 43.4% of cases were colonization, and 3.6% of cases were community acquired [47].

**Therapeutic challenges.** Currently, there has been renewed interest in 2 molecules for the treatment of infections caused by multidrug-resistant A. baumannii. Sulbactam, in combination with β-lactams, has been demonstrated to treat efficiently infections caused by A. baumannii moderately resistant to imipenem [55]. For pan-resistant strains, colistin remains the drug of choice. This antimicrobial has been used with success to treat severe infections, such as meningitis, bacteremia, or pneumonia [53, 56–61]. Deterioration of renal function was the main adverse effect, with a prevalence ranging from 19% to 27% [59–61].

However, the results of animal studies have a tendency to contradict the results of clinical studies regarding the role and place of colistin for the treatment of severe infections caused by multidrug-resistant A. baumannii [62, 63].

**OUTBREAKS**

One important feature of A. baumannii is its propensity to cause outbreaks, which is probably in relation to 2 important characteristics of the organism already described in this review: antimicrobial resistance and resistance to desiccation. The main results of 86 outbreaks investigations caused by A. baumannii, which have been published in the English language literature
from October 1990 to October 2004, are shown below. First, it is important to underline the fact that epidemiologic data were present in only 54% of the publications; the remaining articles included only microbiologic data.

The analysis of the locations where these outbreaks occurred shows that 2 outbreaks involved multiple health care facilities and that another 2 outbreaks involved multiple services and/or departments in the same health care facility. Regarding the specific services and/or departments involved, 26 (59%) of them were intensive care units (table 1). Many risk factors for epidemic infections have been identified, including host factors, procedures, treatments, or the circumstances of the hospitalization, and more specifically, admission to wards with a high density of infected and/or colonized patients (table 2). Environmental contamination of various hospital items has been often identified, ranging from suctioning equipment to pillows and mattresses (table 3). The role of hands has also been investigated, showing that hand-carriage rates among staff members (nurses and physicians) ranged from 3% to 23% and that the carriage was usually transient, except in the case of damaged skin [64, 65]. It is important to note that strict infection-control measures, such as contact isolation precautions, limit both staff colonization and environmental contamination. This was clearly demonstrated by Paavilainen et al. [66], who performed a prospective study to assess staff and environmental colonization following the transfer of a pediatric patient with severe burns who was colonized with multidrug-resistant *A. baumannii* from another hospital, where this strain had caused an outbreak. Of the 1907 postexposure cultures performed on specimens collected from the staff and 425 environmental samples, only 0.7% and 4%, respectively, grew this microorganism. However, it is important to emphasize that the patient was cared for in a private room, with contact isolation, by a hospital staff of 26 persons, of whom there were 7 main nurses who did not participate in the care of other patients in the ward [66].

**Multifacility outbreaks.** Recently, *A. baumannii* has been shown to cause multifacility outbreaks. Such outbreaks have occurred in France and in the United States. In France, from July 2003 to May 2004, 290 cases of infections and/or colonizations occurred in 55 health care facilities located in 55 different administrative departments. Thirty strains were typed by PFGE, were shown to share the same restriction profile, and were identical to the multidrug-resistant AYE strain described in the Antimicrobial Resistance Section of this article. Most patients were hospitalized in intensive care units. Of the 217 patients for whom clinical data were available, 73 (33%) suffered from infection and 144 (67%) were colonized. Among the 73 infected patients, the deaths of 19 (26%) were directly attributed to *A. baumannii* infection. It was suspected that the transfer of infected and/or colonized patients from one facility to another was responsible for the spread of the epidemic strain across France [67].

Another multifacility outbreak occurred in 15 health care facilities in Brooklyn, New York, from July to September 1999, during which 419 isolates were collected, with 62 of them belonging to a single clone that was present in all 15 health care facilities. Fifty-three percent of the isolates were resistant to imipenem, and 12% were resistant to all standard antimicrobials [18].

**Control of outbreaks.** When a source and/or reservoir were identified, the outbreak was controlled by the eradication of the source/reservoir. In other circumstances, various measures were used, including unit closure, cohorting of patients and staff, strict hand hygiene, contact or strict isolation, environmental disinfection, discharge of all colonized patients, con-

### Table 1. Locations of health care outbreaks caused by *Acinetobacter baumannii*.

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of outbreaks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple health care facilities</td>
<td>2</td>
</tr>
<tr>
<td>Multiple services and/or departments within the same health care facility</td>
<td>2</td>
</tr>
<tr>
<td>Adult intensive care unit</td>
<td>26</td>
</tr>
<tr>
<td>Neonatal intensive care unit</td>
<td>3</td>
</tr>
<tr>
<td>Burn unit</td>
<td>4</td>
</tr>
<tr>
<td>Neurosurgery unit</td>
<td>3</td>
</tr>
<tr>
<td>Surgery unit</td>
<td>2</td>
</tr>
<tr>
<td>Internal medicine unit</td>
<td>1</td>
</tr>
<tr>
<td>Oncology unit</td>
<td>1</td>
</tr>
</tbody>
</table>

### Table 2. Risk factors for epidemic *Acinetobacter baumannii* infections and/or colonizations.

<table>
<thead>
<tr>
<th>Risk Factor</th>
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<tbody>
<tr>
<td>High APACHE II score</td>
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<td>Prematurity</td>
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<tr>
<td>Procedure</td>
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<tr>
<td>Surgery</td>
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<tr>
<td>Catheterization</td>
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<tr>
<td>Mechanical ventilation and duration</td>
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<tr>
<td>Previous antimicrobial therapy</td>
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<tr>
<td>Carbapenems</td>
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<tr>
<td>Fluoroquinolones</td>
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<tr>
<td>Third generation cephalosporins</td>
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<tr>
<td>Aminoglycosides</td>
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<tr>
<td>Receipt of blood products</td>
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<tr>
<td>Contaminated parenteral solutions</td>
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<tr>
<td>Enteral feeding</td>
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<tr>
<td>Circumstances of hospitalization</td>
</tr>
<tr>
<td>Length of stay</td>
</tr>
<tr>
<td>High work load</td>
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<tr>
<td>Admission to wards with a high density of infected and/or colonized patients</td>
</tr>
</tbody>
</table>

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Table 3. *Acinetobacter baumannii* outbreaks and items involved in environmental contamination.

<table>
<thead>
<tr>
<th>Item</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suctioning equipment</td>
</tr>
<tr>
<td>Washbasin</td>
</tr>
<tr>
<td>Bedrail</td>
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<tr>
<td>Bedside</td>
</tr>
<tr>
<td>Table</td>
</tr>
<tr>
<td>Ventilator</td>
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<tr>
<td>Infusion pump</td>
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<tr>
<td>Sink</td>
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<tr>
<td>Hygroscopic bandage</td>
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<tr>
<td>Shower trolley</td>
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<tr>
<td>Pillow</td>
</tr>
<tr>
<td>Mattress</td>
</tr>
<tr>
<td>Resuscitation equipment</td>
</tr>
<tr>
<td>Stainless steel trolley</td>
</tr>
</tbody>
</table>

trolled antimicrobial use, and prompt identification of new cases.

**COMPLEXITY OF THE EPIDEMIOLOGY OF A. BAUMANNII INFECTION**

Prevention of infection requires knowledge about the epidemiology of infection. This epidemiology can be straightforward when sources and reservoirs have been identified and when it is easy to differentiate between sporadic and epidemic cases of infection. This is not the case with *A. baumannii*. However, the development of molecular typing methods has provided clinical microbiologists with powerful tools to explore the epidemiology of infections and to reveal the molecular heterogeneity of the isolates of *A. baumannii* within clinics or health care facilities. Rodriguez-Bano et al. [47] have shown in their multicenter study that the number of clones in an individual health care facility ranged from 1 to 4, except in 3 health care facilities in which 9–15 clones were identified. In addition, a predominant clone was observed in the 14 health care facilities where the greatest numbers of cases were observed [47]. In another study performed in a medical intensive care unit in France, molecular typing showed that, during an “outbreak,” epidemic cases coexisted with sporadic cases, and the epidemiologic investigation revealed that risk factors for epidemic cases included having undergone a surgical procedure in the emergency operating room, whereas the main risk factor for sporadic cases was previous receipt of fluoroquinolone. In addition, a parallelism was shown between the incidence of *A. baumannii* infections and the amount of intravenous fluoroquinolone used. In conclusion, the control of these infections was made possible by the implementation of control measures adapted to the risk factors identified [68].

**CONCLUSIONS**

In conclusion, *A. baumannii* is a fascinating microorganism because of the diversity of its reservoirs, its capacity to accumulate genes and/or mechanisms of antimicrobial resistance, its resistance to desiccation, and its propensity to cause outbreaks. However, several shadow areas persist. We know very little about the virulence of the microorganism, reservoirs remain to be discovered, and strategies to control the spread of multidrug-resistant strains have to be designed and evaluated. There is also another challenge: the treatment of infections caused by multidrug-resistant strains.

**Acknowledgments**

*Potential conflicts of interest.* P.E.F. and H.R.: no conflicts.

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