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Survival of *Acinetobacter baumannii* on Dry Surfaces: Comparison of Outbreak and Sporadic Isolates

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***Acinetobacter* spp. are important nosocomial pathogens reported with increasing frequency in outbreaks of cross-infection during the past 2 decades. The majority of such outbreaks are caused by *Acinetobacter baumannii*. To investigate whether desiccation tolerance may be involved in the ability of certain strains of *A. baumannii* to cause hospital outbreaks, a blind study was carried out with 39 epidemiologically well-characterized clinical isolates of *A. baumannii* for which survival times were determined under simulated hospital conditions. The survival times on glass coverslips of 22 strains isolated from eight well-defined hospital outbreaks in a German metropolitan area were compared with the survival times of 17 sporadic strains not involved in outbreaks but rather isolated from inpatients in the same geographic area. All sporadic isolates have been shown by pulsed-field gel electrophoresis to represent different strain types. There was no statistically significant difference between the survival times of sporadic strains of *A. baumannii* and outbreak strains (27.2 versus 26.5 days, respectively; $P \leq 0.44$) by the Wilcoxon-Mann-Whitney test. All investigated *A. baumannii* strains, irrespective of their areas of endemicity or epidemic occurrence, have the ability to survive for a long time on dry surfaces. Antimicrobial susceptibility testing showed that *A. baumannii* outbreak strains were significantly more resistant to various broad-spectrum antimicrobial agents than sporadic strains. Both desiccation tolerance and multidrug resistance may contribute to their maintenance in the hospital setting and may explain in part their propensity to cause prolonged outbreaks of nosocomial infection.**

Acinetobacter spp. are ubiquitous gram-negative bacteria that can be isolated from soil, water, human skin, and the environment. Incomplete taxonomic nomenclature has long prevented a detailed investigation of the epidemiology of the various genospecies that constitute the genus *Acinetobacter* (14). Currently, at least 19 genomic species are recognized as members of the genus (3, 11). *Acinetobacter* spp. may cause a wide range of opportunistic infections, principally in elderly patients, infants, and patients with severe underlying disease. Those in intensive care units (ICU) with assisted mechanical ventilation and urinary or intravascular catheters are at particular risk. In an international multicenter study of blood culture isolates, *Acinetobacter* spp. were ranked among the top ten bacteria causing septicemia in 18 of 44 large European hospitals (31). It has been shown that most clinical isolates are strains of *Acinetobacter baumannii* (23) and that this species together with *Acinetobacter* DNA group 13TU is involved in the majority of *Acinetobacter* hospital outbreaks (3), whereas species such as *Acinetobacter* DNA group 3 and *Acinetobacter junii* have only occasionally been implicated in outbreaks of nosocomial infection (4, 17). *A. baumannii* isolates are resistant to many of the currently used antibiotics, and resulting infections are often difficult to treat. Imipenem remains the most active drug, but unfortunately the emergence of imipenem-resistant strains has been documented in recent hospital outbreaks (5, 16).

Compared with other genera of gram-negative bacilli, *Acinetobacter* is found to survive much better on fingertips or on dry surfaces when tested under simulated hospital environmental conditions (19, 20). The skin of patients and medical personnel is thought to be involved in the transmission of strains, and in some outbreaks, molecular typing has identified the epidemic strain on the skin of patients (12, 13, 22). Allen and Green (1) were the first to report that airborne spread may also serve as a mode of transmission. Contaminated reusable medical equipment, such as ventilator tubing, respirometers, and arterial pressure monitoring devices, used for the management of severely ill patients has also been implicated as a route of transmission to patients (2, 6, 8, 17). In addition, a wide variety of dry environmental objects such as bed mattresses (29), pillows (33), a tape recorder, a television set, and a fan (18) have been found to be contaminated with *Acinetobacter* and may serve as reservoirs during nosocomial outbreaks.

Survival responses of different *Acinetobacter* spp. have been studied to some extent previously (15, 19, 34), but there are no reports comparing the desiccation tolerances of sporadic and outbreak strains of *A. baumannii*. To assess whether desiccation tolerance is a characteristic feature of epidemic *A. baumannii*, the survival times of 22 clinical *A. baumannii* strains isolated from eight hospital outbreaks were compared with those of 17 sporadic, non-outbreak-related strains. In addition, resistance to commonly used broad-spectrum antimicrobials was also compared between the epidemic and sporadic strains.

MATERIALS AND METHODS

Bacterial strains. Details of the strains used in this investigation are given in Tables 1 and 2. Twenty-two of these strains were isolated from eight well-defined hospital outbreaks in the Cologne metropolitan area in Germany and represented eight different clonal strains (two to four isolates from each outbreak). Details of these outbreaks have been described elsewhere (24, 25). Seventeen strains were sporadic isolates from hospitalized patients in the same geographic area but were epidemiologically unrelated and represented different strain types, as demonstrated previously by pulsed-field gel electrophoresis. All of these strains have been identified to the species level by current genotyping methods and have been well characterized in previous studies (26). *A. baumannii* ATCC 19606^T was also studied for comparison. Strains were grown on Iso-Sensitest agar

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TABLE 1. Survival times of sporadic strains of *A. baumannii* suspended in distilled water and kept at 31% RH and at a room temperature of 22 ± 3°C[a]

Strain no.	Site of isolation	Mean survival time (days) ± SD[b]
M 3317	Tracheal aspirate	23 ± 1.14
M 3789	Tracheal aspirate	32 ± 0.71
M 7360	Tracheal aspirate	25 ± 0.71
M 13546	Tracheal aspirate	25 ± 3.53
St-12084	Blood	21 ± 0.71
St-13641	Blood	29 ± 2.82
St-17306	Blood	26 ± 2.12
St-18748	Catheter	30 ± 2.82
St-20421	Blood	31 ± 0.71
U-9560	Urine	24 ± 2.12
V-4316	Wound swab	25 ± 1.14
V-12277	Wound swab	31 ± 0.71
V-12334	Wound swab	29 ± 2.82
V-12561	Abscess	30 ± 5.65
W-4329	Gastric juice	28 ± 2.82
W-8334	Wound swab	28 ± 1.14
W-8832	Wound swab	27 ± 5.53

[a] ATCC 19606^T was used for comparison.
[b] Overall mean, 27.29 days; range, 21 to 32 days.

(Oxoid Unipath, Hants, Basingstoke, United Kingdom) at 30°C. For long-term storage, strains were kept in 15% (vol/vol) glycerol broth at −70°C.

Desiccation survival assay. The procedure described by Jawad et al. (19) was followed to determine the length of time that strains could survive on glass coverslips when kept at 31% relative humidity (RH). A 1-ml aliquot of overnight nutrient broth culture was placed in a 1.5-ml Eppendorf tube and centrifuged for 5 min in a microcentrifuge (MSE Scientific Instruments, Crawley, Surrey, United Kingdom) at 11,600 × *g*. The supernatant fluid was discarded, and the cells were washed once with 1 ml of distilled water and then resuspended in another 1 ml. Twenty microliters of these suspensions (approximately 2×10^7 CFU) was deposited onto sterile 13-mm-diameter rounded glass coverslips, placed in uncovered petri dishes, and kept in airtight transparent plastic boxes (17 by 11 by 5.5 cm). The RH inside the plastic boxes was maintained at 31% ± 3% by the presence of a saturated salt solution of $CaCl_2 \cdot 6H_2O$ in an open 5-ml beaker. This RH was chosen as an approximation to conditions in United Kingdom hospitals. A digital thermohygrometer (Fison Scientific Equipment, Loughborough, Leicestershire, United Kingdom) was used to monitor RH and temperature.

Determination of viable counts. Viable counts were determined for the original nutrient broth cultures and the bacterial cell suspensions prior to inoculation onto the glass coverslips. Zero-time viable counts were determined by washing a glass coverslip in 2 ml of sterile distilled water, vortexing the fluid vigorously for 15 s, and inoculating 100-μl aliquots onto Iso-Sensitest agar plates by the spread plate method, after appropriate dilutions in distilled water had been made. The CFU appearing after overnight incubation at 30°C in air were counted. Viable counts were determined every 24 h and compared with the count of the original bacterial cell suspension. Three glass coverslips were used separately for every count, and three different dilutions were made for every coverslip. When the viable count decreased to ≤30 CFU on an agar plate after overnight incubation, an agar incorporation method was used to determine the viable count (19). Each assay was repeated three times, and the mean survival time was calculated.

Susceptibility testing. MICs were determined by a broth dilution method with factory-prepared antibiotic dilutions in microtiter trays (Micronaut-S; Merlin Diagnostics, Bornheim, Germany) in accordance with guidelines established by the National Committee for Clinical Laboratory Standards (NCCLS) (21). The following antimicrobials were included: gentamicin, amikacin, amoxicillin-clavulanate, mezlocillin, piperacillin-tazobactam, meropenem, imipenem, cefuroxime, cefepime, ceftriaxone, cefotaxime, and ciprofloxacin.

RESULTS

Survival time was defined as the length of time taken for the viable counts to reach a value of zero on three consecutive days, as determined by the agar incorporation assay, which was capable of detecting ≥1 CFU/coverslip. The mean survival times of the various *A. baumannii* isolates tested are shown in Tables 1 and 2. The mean survival time for sporadic strains was 27.29 days (range, 21 to 32 days), while the mean survival time for outbreak strains was 26.55 days (range, 21 to 33 days). No statistically significant difference between the survival times of outbreak and sporadic strains of *A. baumannii* ($P \leq 0.44$) was found when the Wilcoxon-Mann-Whitney test of significance was applied. The mean survival time for *A. baumannii* ATCC 19606^T was 6 days, which was significantly less time than that of the 39 clinical isolates of *A. baumannii* (mean survival time, 27 days). None of the strains exhibited mucoid colonies on the Iso-Sensitest agar. The mean survival times of wound swab isolates, catheter isolates, urine isolates, tracheal aspirate isolates, and blood isolates were 28, 27.4, 27, 26.8, and 26 days, respectively.

The in vitro activities of the various agents against sporadic and outbreak isolates of *A. baumannii* are shown in Table 3. All the strains tested were resistant to cefuroxime and sensitive to imipenem, meropenem, and piperacillin-tazobactam, with no difference between the geometric mean MICs for sporadic and outbreak isolates. *A. baumannii* outbreak strains, however, were significantly more resistant to aminoglycosides, amoxicillin-clavulanate, mezlocillin, cefepime, ceftriaxone, cefotaxime, and ciprofloxacin than sporadic isolates. This difference was most pronounced for gentamicin (geometric mean MICs, 12.1 and 5.0 mg/liter and percentages of isolates fully susceptible at NCCLS breakpoints, 25 and 67% for epidemic and sporadic isolates, respectively), amikacin (geometric mean MICs, 27.6 and 3.6 mg/liter and percentages of isolates fully susceptible, 63 and 100% for epidemic and sporadic isolates, respectively), and ciprofloxacin (geometric mean MICs, 10.6 and 1.3 mg/liter and percentages of isolates fully susceptible, 13 and 100% for epidemic and sporadic isolates, respectively).

DISCUSSION

There are reports of individual endemic strains of *A. baumannii* persisting in hospitals for up to 3 years (13, 24, 29, 32). It is currently unknown why some strains have a propensity for

TABLE 2. Survival times of outbreak strains of *A. baumannii* suspended in distilled water and kept at 31% RH and at a room temperature of 22 ± 3°C

Strain no.	Site of isolation	Outbreak[a]	Mean survival time (days) ± SD[b]
St-284	Blood	G-III	22 ± 4.42
St-14733	Blood	G-III	31 ± 0.71
St-1650	Blood	G-X	25 ± 1.14
St-1954	Blood	G-X	27 ± 0.71
St-2312	Blood	G-VIII	27 ± 1.14
St-8195	Catheter	G-VIII	21 ± 3.53
St-11681	Blood	G-VIII	21 ± 1.14
St-7961	Blood	G-VIII	23 ± 4.42
St-14970	Catheter	G-V	28 ± 4.42
St-15598	Catheter	G-V	26 ± 2.12
St-16706	Blood	G-IV	26 ± 1.14
St-20820	Blood	G-IV	30 ± 2.82
St-21359	Catheter	G-IV	32 ± 1.14
St-17093	Blood	G-VI	27 ± 2.12
V-7459	Tracheal aspirate	G-VI	26 ± 0.71
St-17108 I	Blood	G-VI	26 ± 1.14
St-17108 II	Blood	G-VI	25 ± 2.12
U-1901	Urine	G-I	24 ± 0.71
W-5420	Tracheal aspirate	G-I	30 ± 5.56
U-10247	Urine	G-II	24 ± 4.42
U-11177	Urine	G-II	33 ± 1.41
U-11432	Urine	G-II	30 ± 0.71

[a] Outbreak designations are as shown in reference 26.
[b] Overall mean, 26.54 days; range, 21 to 33 days.

TABLE 3. In vitro activities of various antimicrobial agents against epidemic and sporadic *A. baumannii* isolates

A. baumannii group (no. of strains tested)	Antibiotic	MIC (mg/liter)[a]				% Susceptible
		Range	50%	90%	Geometric mean	
Outbreak isolates (16)	Gentamicin	1–>8	>8	>8	12.1	25
	Amikacin	2–>32	8	>32	27.6	63
	Amoxicillin-clavulanate	4/2–>32/2	32/2	>32/2	37.5	25
	Mezlocillin	8–>32	>32	>32	45.5	31
	Piperacillin-tazobactam	≤1/4–32/4	4/4	16/4	7.0	94
	Meropenem	≤0.25–4	1	2	0.96	100
	Imipenem	≤0.25–1	≤0.25	0.5	0.25	100
	Cefuroxime	16–>32	32	>32	41.0	0
	Cefepime	2–64	8	16	11.8	81
	Ceftriaxone	4–>32	16	>32	33.5	44
	Cefotaxime	4–>16	16	>16	18.7	44
	Ciprofloxacin	0.5–>8	8	>8	10.6	13
Sporadic isolates (18)	Gentamicin	0.5–>8	1	>8	5.0	67
	Amikacin	1–16	4	8	3.6	100
	Amoxicillin-clavulanate	≤0.25/2–>32/2	4/2	>32/2	12.8	61
	Mezlocillin	8–>32	16	>32	23.6	61
	Piperacillin-tazobactam	≤1/4–32/4	≤1/4	16/4	5.3	100
	Meropenem	≤0.25–1	0.5	1	0.51	100
	Imipenem	≤0.25–0.5	≤0.25	≤0.25	0.14	100
	Cefuroxime	16–>32	32	>32	36.4	0
	Cefepime	1–8	2	8	3.3	100
	Ceftriaxone	4–>32	8	16	11.8	63
	Cefotaxime	4–>16	8	>16	11.8	63
	Ciprofloxacin	0.5–4	0.5	4	1.3	100

[a] 50% and 90%, MICs at which 50 and 90% of the isolates are inhibited. Values separated with a shill apply to the first- and second-listed antibiotics of the compound, respectively.

causing hospital outbreaks while others are only sporadically recovered from colonized or infected patients. Resistance to adverse environmental conditions such as desiccation is one property that might enhance transmissibility and might be characteristic of outbreak strains, distinguishing them from nonoutbreak-related strains. *Burkholderia cepacia* is another gram-negative pathogen responsible for an increasing number of nosocomial infections. Significant strain-to-strain differences in survival on dry surfaces have been reported, and Drabick et al. (10) have proposed that enhanced survival time may contribute to the apparent increased transmissibility of some strains of *B. cepacia*. A blind study was therefore carried out to compare survival times of clinical strains of *A. baumannii* representing eight well-described hospital outbreaks in the Cologne metropolitan area in Germany with survival times of epidemiologically unrelated sporadic strains isolated in the same geographic area. All isolates have been extensively characterized by modern molecular typing techniques. The response to drying, as measured by the survival time on glass coverslips, of *A. baumannii* strains was similar to that of *Staphylococcus aureus* tested previously in the same assay (19). The mean survival time of the German clinical isolates was 27 days and was as long as 33 days for some isolates. However, no statistically significant difference between the survival times of outbreak strains versus those of sporadic strains was found, suggesting that all strains may, when environmental conditions and opportunity permit, cause multiple infections. These conditions may include poor hygiene, improper disinfection measures, and probably the high degree of selective pressure associated with the extensive use of broad-spectrum antimicrobial agents. The last condition is supported by our observation that *A. baumannii* strains involved in hospital outbreaks are significantly more resistant to commonly used broad-spectrum antibiotics such as aminoglycosides, β-lactams, and fluoroquinolones than strains isolated only sporadically. This resistance may provide certain *A. baumannii* strains with a selective advantage in a setting where there is extensive exposure to antimicrobials, such as the modern ICU. Similar findings were also reported by Dijkshoorn et al. (9), who found that epidemic *A. baumannii* strains were more resistant to gentamicin and carbenicillin than sporadic isolates, without giving further details.

The previous observation that clinical *A. baumannii* strains are more resistant to desiccation than American Type Culture Collection *A. baumannii* strains (19, 34) was confirmed in the present study. There is currently no explanation for this finding, but it may be a consequence of the repeated subculturing that the type strain has undergone over the years. Greater variability in the ability to survive under dry conditions among different *A. baumannii* strains has been reported by Wendt et al. (34), who noted that strains isolated from dry sources survived better than those isolated from wet sources (34). However, this observation was based on a study of just six *A. baumannii* strains. Our findings, based on 25 distinct strains, including some recovered from urinary tract infections, do not support this observation.

It has been shown previously that *A. baumannii* strains survive desiccation far better than other *Acinetobacter* species such as *A. johnsonii*, *A. junii*, and *A. lwoffii* (19, 20). This may explain why strains belonging to these other species have only very rarely been implicated in hospital outbreaks (4, 17). *Acinetobacter* spp. form part of the bacterial flora of the skin of healthy subjects, patients, and hospital staff, particularly in moist regions such as axillae, the groin, and toe webs, and between 25 and 43% of healthy individuals carry *Acinetobacter* spp. on their skin (12, 28, 30). Strains of *Acinetobacter calcoaceticus* survived better on fingertips than strains of *A. lwoffii* (20). However, *A. johnsonii*, *A. lwoffii*, and *Acinetobacter radioresistens* predominate on human skin, whereas skin carriage of

A. baumannii, the species of *Acinetobacter* involved in the majority of nosocomial outbreaks, is very rare (1.5%) (28). Conversely, during outbreaks in the ICU setting, epidemic *A. baumannii* strains may be recovered with high frequency from skin and rectal samples (7, 27). Prior skin carriage of *A. baumannii* is an unlikely source of nosocomial outbreaks; rather, skin colonization is a result of multiple exposures to endemic *A. baumannii* in the ICU setting. Once colonized at one site, a bedridden patient may easily contaminate himself at any other body site. Increasing skin colonization was detected 1 or 2 weeks after admission in colonized or infected patients, respectively (14).

The skin of both patients and medical staff, as well as dry inanimate objects in hospital wards contaminated with *A. baumannii*, is able to facilitate transmission of strains and may serve as unrecognized reservoirs in prolonged nosocomial outbreaks. Desiccation tolerance and multidrug resistance, as shown here, may contribute to the ability of *A. baumannii* to cause hospital outbreaks and may explain why certain strains are able to establish themselves in the hospital environment while others are isolated only sporadically. Strain-to-strain variation in resistance to disinfectants and the ability to use a wide range of carbon sources for nutrition may be important additional factors. Proper disinfection of dry surfaces and medical equipment in the hospital ward, strict adherence to hand disinfection protocols, and prudent use of antimicrobials are crucial for the prevention of nosocomial transmission. The presence of capsules and fimbriae and the production of enzymes which may damage the infected host cells have all been implicated in the virulence of *Acinetobacter* spp. (3). It is not known whether expression of any of the postulated virulence factors is enhanced under drying conditions. There is now a need for a detailed investigtion of the pathogenicity factors of these bacteria, focusing on a comparison of outbreak and sporadic strains.

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