Letters to the Editor

Survival of *Acinetobacter* on Three Clinically Related Inanimate Surfaces

To the Editor:

Acinetobacter species are increasingly recognized as nosocomial pathogens that cause outbreaks, especially in the intensive care unit (ICU). Survival of these bacteria on the skin and persistence in the hospital environment are believed to be important factors in the development and continuation of outbreaks. In Nottingham, we have experienced outbreaks in our adult ICU, and, during the most recent, patient and environmental isolates were indistinguishable.[1] This therefore prompted us to determine whether a local outbreak strain was capable of surviving for prolonged periods on commonly used clinical surfaces.

An enamel-coated drip stand and two horizontal surfaces, a Formica shelf and a stainless steel treatment trolley, were first decontaminated with a 2% (vol/vol) phenolic disinfectant (Hycolin, Adams Healthcare, Leeds, UK) and then rinsed with sterile water before sampling to confirm the absence of other bacteria. *Staphylococcus aureus* National Collection of Type Cultures (NCTC) 6571, *Pseudomonas aeruginosa* NCTC 10662, *Acinetobacter baumannii* isolated during the most recent outbreak,[1] and *Acinetobacter* species were assessed for their ability to survive drying. The *Acinetobacter* species was identified by tDNA fingerprinting[2] but did not match any of the 17 defined genospecies (having 50% similarity with *Acinetobacter johnsonii)* and therefore is referred to as *Acinetobacter* species throughout. Following contamination of Formica shelving with a range of inocula, an inoculum of 10^6/mL was chosen for all subsequent experiments. This inoculum facilitated the assessment of contamination and, based on isolation rates from the skin of infected patients, probably also reflects the level of surface contamination that occurs during outbreaks. Individual areas of the Formica shelves and the stainless steel surface were each inoculated with 0.25/mL of an overnight broth diluted to 10^6 organisms per mL and spread over 8 cm^2 with a sterile glass spreader. The enamel-coated drip stand was similarly inoculated, but with a sterile sponge to ensure an even spread. Experiments were carried out at ambient temperature and humidity in a non-artificially ventilated room, ie, 20° to 22°C and 60% to 70% humidity. Sampling was carried out daily, in triplicate from individual areas, with contact plates containing cystine lactose electrolyte-deficient (CLED) agar. The entire process was repeated three times. The drip stand was sampled with a swab moistened in sterile saline and inoculated on to CLED agar at similar intervals. All plates were incubated in air at 30°C (*A baumannii* and *Acinetobacter* species) or 37°C (*S aureus* and *P aeruginosa)* for 48 hours. The number of colonies observed on the Formica shelving and treatment trolley was counted, and, for the drip stand, growth was assessed as either present or absent.

A baumannii and *S aureus* persisted for mean durations of 11 and 9 days on the Formica shelf, 12 and 10 days on the stainless steel trolley, and 6 and 3 days, respectively, on the drip stand. *Acinetobacter* species and *P aeruginosa* survived for 6 and 4 days on the Formica shelf, 6 and 5 days on the stainless steel trolley, and 3 and 1 days, respectively, on the drip stand. The number of colonies ranged from over 200 to 1 per plate, and the number usually decreased with time.

These results confirm that a local strain of *Acinetobacter* species may persist on common clinical surfaces for relatively long periods compared with other bacteria, and this may partly explain our recent finding that a Nottingham outbreak strain of *A baumannii,* recovered during 1985 to 1986 and 1992 to 1993, continues to be isolated from both patients and the environment.[3] Further experiments are required, however, to determine whether other nosocomial isolates of *A baumannii* behave in a manner similar to this local strain.

Wendt and colleagues have demonstrated that survival of *Acinetobacter* species in the environment is significantly associated with the strain and its source, with strains recovered from dry sources or during outbreaks surviving for longer periods.[4] We only compared one local isolate with other nosocomial pathogens, *S aureus* and *P aeruginosa,* and experiments involving a larger number of *A baumannii* strains suspended in a variety of other menstrua are required to confirm this. We were mainly interested in determining whether the bacteria were present or not, but low numbers, which might not have been detected by our method, are probably less significant in the continuation of an outbreak. At the molecular level, mechanisms may have evolved to protect certain species from desiccation. For example, *Acinetobacter radioresistens* is a species with high radiation resistance that can persist in the environment for prolonged periods; mutant strains that lack the ability to repair DNA are sensitive to the lethal effects of both types of stress.[5] Continued vigilance of adaptable bacteria, such as *Acinetobacter,* and effective cleaning regimens remain of paramount importance in controlling and preventing outbreaks.

REFERENCES

1. Crowe M, Towner KJ, Humphreys H. Clinical and epidemiological features of an outbreak of *Acinetobacter* infection in an intensive therapy unit. *J Med Microbiol* 1995;43:55-62.
2. Ehrenstein B, Bernards AT, Dijkshoorn L, Gerner-Smidt P, Towner KJ, Bouvet PJM, et al. *Acinetobacter* species identification by using tRNA spacer fingerprinting. *J Clin Microbiol* 1996;34:2414-2420.
3. Webster CA, Crowe M, Humphreys H, Towner KJ. Surveillance of an adult intensive care unit for long-term persistence of a multiresistant strain of *Acinetobacter baumannii. Eur J Clin Microbiol Infect Dis* 1998;17:171-176.
4. Wendt C, Ditetze B, Dietz E, Rüden H. Survival of *Acinetobacter baumannii* on dry surfaces. *J Clin Microbiol* 1997;35:1394-1397.
5. Jawad A, Snelling AM, Heritage J, Hawkey PM. Exceptional dessication tolerance of *Acinetobacter radioresistens. J Hosp Infect* 1998;39:235-240.

Carol Webster, PhD
Kevin J. Towner, PhD
Hilary Humphreys, MD
University Hospital
Queen's Medical Centre
Nottingham, United Kingdom

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