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“Airborne Assault”: A New Dimension in *Acinetobacter baumannii* Transmission*

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Acinetobacter baumannii has emerged to become a predominant cause of nosocomial infections in the United States and across the globe. During the past decade, the remarkable increase in the proportion of *A. baumannii* strains that are carbapenem resistant has ushered in an era of far more lethal infections. In a recent study of 13,796 patients in 1,265 ICUs from 75 countries, *A. baumannii* was one of only two of 19 organisms studied that were strongly linked to increased hospital mortality by multivariate analysis (1). Patients with bacteremia or ventilator-associated pneumonia due to Carbapenem resistant have more than 50% to 60% mortality rates (2–5). These mortality rates result from the very high frequency with which empiric therapy (e.g., with carbapenems) is inactive against Carbapenem resistant (3–5) and the limited suboptimal definitive therapy (e.g., colistin and tigecycline) available.

Given their very poor outcomes and the lack of new drugs in development to treat these infections, understanding how *A. baumannii* is transmitted to patients is of critical importance to enable development of new prophylactic strategies. In the last few years, hospital environmental surfaces have been increasingly recognized as important reservoirs for pathogens (6, 7), and *A. baumannii* is a particularly hardy environmental organism. Indeed, when *A. baumannii* was inoculated into a plastic bottle with no added nutrients and the liquid was allowed to evaporate, the organism remained viable at detectable culture densities on the dry bottle surface for almost a year (8).

Thus, it generally has been assumed that *A. baumannii* transmission is due to interactions between healthcare providers, patients, and contaminated fomites in the environment. *A. baumannii* has not been considered an airborne droplet pathogen despite it causing outbreaks of community-acquired pneumonia in Australasia (9, 10) and an intriguing case report of a

*See also p. 1915.

nurse in whom inhalational *A. baumannii* pneumonia developed while caring for an infected patient (11). In this latter case, the airborne communication of *A. baumannii* from patient to healthcare worker was one of the first instances of an occupational transmission of a multidrug-resistant pathogen.

It is in this context that the report by Munoz-Price et al (12), in this issue of *Critical Care Medicine*, is so intriguing. The authors sought to determine if *A. baumannii* could be cultured in the air in patient rooms and if so, if the airborne isolates were clonally related to the clinical isolates cultured from patients. They sampled 53 rooms and found 12 air cultures positive for Carbapenem resistant, all of which came from rooms currently or previously housing patients colonized or infected with *A. baumannii*. Of the 21 patients known to be colonized or infected, the air in 11 (52%) of the patients' rooms contained Carbapenem resistant. In contrast, for rooms harboring patients not colonized by *A. baumannii* (as determined by active surveillance), none had positive air cultures. Thus, there was a clear association between the colonization status of the patient and air colonization with *A. baumannii* in the room. Because the air ducts in the room were not colonized (with one exception), it is highly likely that patients were the sources of the Carbapenem resistant strains found in the air.

These findings are alarming and suggest that environmental decontamination focusing on surfaces will not fully prevent *A. baumannii* transmission. "What goes up must come down," and it seems likely that *A. baumannii* in the air above and around patients has the potential to contribute to infection transmission in several ways: 1) by recontaminating surfaces in a room after environmental services personnel have disinfected those surfaces, but not the air in the room; 2) by spreading organisms from one colonization site on a patient to multiple sites on the same patient, as airborne bacteria settle out of the air; 3) by airborne contamination of healthcare providers' clothes and hands, even if adequate hand hygiene is done before entering the room; and 4) by airborne contamination of medical instruments (e.g., portable x-ray devices and wheelchairs) that travel from room to room and are not cleaned between rooms.

The primary limitation of the study is that molecular genotyping was only available from three of the 21 clinical strains (which did, however, match the corresponding airborne strains in the rooms for those three strains). Also, clinical data were not available to determine relationships between clinical and environmental factors that could be linked to airborne colonization. Such potentially important factors include, but are not limited to, whether the patient is infected or asymptomatically colonized, the patient site of infection/colonization (airway vs wound vs blood vs other), the impact of host immune deficiency or other comorbidities, and the impact of antibiotic therapy for infected patients. Environmental factors, such as humidity, temperature, number of air exchanges per hour in the room, and patient density, could also play a role in airborne transmission and merit investigation.

Ultimately the goal should be reduction of acquisition of *A. baumannii* infection. Thus, explicitly determining if airborne colonization increases the risk of acquisition of infection either by patient(s) currently in the room, patients subsequently admitted to the room, or even patients in neighboring rooms are critical questions that merit future study. As well, the current study highlights important and previously underrecognized potential limitations of current disinfection methods that focus on providers, patients, and environmental surfaces and not on potential airborne transmission. Novel disinfection methods (such as those that use hydrogen peroxide, microaerosolization of liquid disinfectants into the air, and ultraviolet lights) that can disinfect airborne bacteria should be studied to determine if they can reduce transmission of such infections (13). Finally, additional research is needed to determine how commonly airborne colonization occurs with other pathogens, and if so, what

the clinical impact is. Such research may move infection prevention from a 3D focus (providers, patients, environmental surfaces) to a scary new fourth dimension (air) of transmission of hospital-acquired infections.

Finally, the current study underscores that *A. baumannii* is a most elusive and misunderstood foe (14). The very name *Acinetobacter* is derived from the Greek term for lack of mobility (*a-kineto*); yet, we now know that it is motile not only on surfaces (15) but through the air as well.

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