culture. Criteria for asymptomatic bacteriuria, as reported by the Centers for Disease Control and Prevention/ National Healthcare Safety Network,⁵ are primarily designed for surveillance purposes, and only patients with KPC-2-*Kp* bacteriuria who do not meet these criteria are thought to have urinary tract infection and receive treatment.

The results presented here show that the outcomes of CRE bacteriuria/bacteremia are influenced by both the choice of antimicrobial treatment and the CRE isolate type. The selective pressure imposed by antibiotic usage has been strongly associated with the emergence of resistance, as observed in this study and in previous reports regarding polymyxins, 6 tigecycline, 7 and fosfomycin, 4 which are considered "reappraised" therapeutic options to treat multidrug-resistant microorganisms.

Unequivocally, endemic KPC-2-*Kp* has become quite more competitive than multidrug-resistant noncarbapenemase isolates that proved to be self-limited, with neither bacteremia case nor development of resistance observed in this study (Table 1); KPC-2-*Kp* is probably favored by the presence of a more robust resistance mechanism, such as the production of carbapenemase, although *bla*_{KPC-2} gene has not been associated with virulence by itself.⁸

In conclusion, KPC-2-*Kp* isolates presented with recurrent/ subsequent bacteriuria as the main urinary outcome and as such developed cases of bacteremia with a high 30-day mortality rate being observed. Increase in resistance rates was observed for all agents evaluated, possibly driven by previous use similar to prior observations for KPC-2-*Kp* recovered from surveillance rectal swab samples. ⁹ These findings and the poor outcomes for KPC-2-*Kp* infection underscore the urgent need for better surveillance and stewardship programs to combat these antibiotic stains.

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The Chicken-Egg Dilemma: Legionnaires' Disease and Retrograde Contamination of Dental Unit Waterlines

To the Editor—On February 9, 2011, an 82-year-old Italian woman died from Legionnaires' disease (LD). Other than 2 appointments at a dental office, she had not been exposed to any obvious source of Legionella infection in the 2–10 days before symptom onset that occurred on February 7. On February 17, an epidemiologic field investigation in the dental office, performed by the regional healthcare agency, detected Legionella pneumophila serogroup 1 (sg1) in water samples from the cold-water tap (1500 CFU/L), the dental turbine (62000 CFU/L), and the cup filler (4000 CFU/L) of a dental unit, which had been routinely disinfected with H₂O₂. Strain typing revealed that the isolates from the environment and the patient's bronchial aspirate matched, suggesting that the dental unit waterlines (DUW) were the likely source of LD infection. 1

In line with the guidelines for epidemiologic field investigation, ² immediate control measures were taken.

TABLE 1. Results of the Epidemiologic Field Investigation in the Dental Office Attended by the Patient who died from Legionnaires' Disease on February 9, 2011^a

Treatment room	Cold-Water Tap	Hot-Water Tap	Cup Filler	Turbine	DUW-Turbine Connector
Inspection performed	d on February 17, 2011 (1	before shock chlorination	on)		
Room 1	1,500	•••	4,000	62,000	
Inspection performed	d on March 1, 2011				
Room 1		•••	•••		Not reported
Room 2		•••	•••		Not reported
Room 3	Not reported	Not reported	•••	Not reported	
Room 4	•••	•••	•••	Not reported	
Inspection performed	d on March 15, 2011 (aft	er shock chlorination) ^b			
Not reported	Undetected (unspeci	ified site)			

NOTE: DUW, dental unit waterline.

On March 1, the dental healthcare worker was warned to quit practicing until thorough DUW disinfection with chlorination was performed and to reculture the DUW for the presence of *L. pneumophila* after the disinfection process. On March 15, after the disinfection, a single water sample was collected by the agency from an indeterminant site, *Legionella* was not detected (<50 CFU/L) (Table 1) but *Pseudomonas aeruginosa* (400 CFU/L) was found. To date, this is considered the only confirmed LD case attributable to DUW.

This story, however, has an epilogue that was not reported and may subvert the alleged causal association between *L. pneumophila* in DUW and consequent LD development. Indeed, the dentist declared that the 2 appointments occurred on December 9 and January 24; during the latter appointment, the patient showed breathing difficulty, fatigue, and relayed a premonition of her imminent death. In addition, on March 1, the agency collected 6 more water samples in 4 treatment rooms of the dental office, and *L. pneumophila* was likely not detected because the results of these samples were not reported (Table 1).

These results are very important, because they suggest that *L. pneumophila* sg1 transmission from DUW to the patient cannot be confirmed and that an alternative hypothesis cannot be ruled out. Namely, the patient may have been infected before the dental treatment, which may have led to the retrograde contamination of room-1 water systems, including the DUW, through the outlet points. This hypothesis is plausible because the patient harbored *L. pneumophila* in her respiratory tract, ¹ and during dental treatments biological fluids and microorganisms from the respiratory tracts of dental patients are aspirated into the DUW through the outlet points. ³

Such an alternative hypothesis is corroborated by the consideration that the first appointment occurred 62 days before patient's death, an interval largely exceeding the LD incubation period, and the patient's condition during the second appointment suggests that she was already affected with LD and, therefore, was already colonized with *L. pneumophila*. Secondarily, legionellae were detected at a high level in the

room-1 turbine on February 17 and not reported in 4 treatment rooms on March 1. There could be two explanations for the negative findings: (1) water-system chlorination was performed by the dentist before receiving the official notification from the healthcare agency; (2) retrograde DUW contamination had occurred through the outlet points (ie, turbine, cup filler, and faucet) during treatment, which produced transient *L. pneumophila* colonization in room 1 and no colonization in rooms 2–4. The latter hypothesis was corroborated by the fact that, despite the high microbial load detected in the DUW during the first inspection, no other cases of LD or Pontiac fever among the dental staff and patients were reported.¹

It is not possible to exclude the possibility that the patient was exposed to other sources of *Legionella* infection in the days preceding the dental appointments because the patient's history was limited to 10 days before symptom onset and LD incubation is frequently longer.⁴

The present probable retrograde water system contamination from an infected patient, along with other episodes, suggests that it is time to reconsider the routes of Legionella transmission to patients and spread in the environment. Indeed, person-toperson LD transmission was recently demonstrated for the first time in Portugal where a patient developed LD taking care of her son previously infected 300 km away from home with L. pneumophila ST1905.⁵ In addition, the outlet points of 2 Italian hospitals were more frequently and heavily contaminated with L. pneumophila sg1 than the building water systems. This finding was explained by retrograde contamination.⁶ Finally, a dentist who died from LD had positive results for Legionella dumoffii, L. pneumophila, and L. longbeachae, and these microorganisms were detected in the DUW and in the domestic water system, implying that the infected dentist was associated with at least 1 retrograde contamination.⁷

Legionella pneumophila is detectable in the DUW,⁸ which suggests that patients and staff could be at risk for LD. However, no LD clusters and outbreaks have been associated with dental treatments, and there are only 2 suspected sporadic cases including this one.^{1,7} In contrast, the number of exposed

^aDetection of Legionella pneumophila serogroup 1: counts in CFU/L; lowest limit of detection, 50 CFU/L.

^bPseudomonas aeruginosa (400 CFU/L) was detected.

individuals is very high; 200 million people have dental visits in United States each year, ⁹ and the US dental workforce routinely and occupationally exposed to *L. pneumophila* comprises almost 200,000 dentists. ¹⁰ These data demonstrate that LD incidence, and therefore LD risk in dental healthcare settings, is limited. Such an inference, however, does not imply that infection control measures focusing on DUW are unnecessary, ¹¹ given the general high level of contamination, ⁸ but only that these measures are based on the Precautionary Principle. ¹²

In conclusion, the chicken–egg dilemma (ie, strain-typing matches of isolates from the environment and the patient do not demonstrate where the organism occurred first) regarding waterborne pathogens¹³ may also apply to the present report. In addition, the scientific evidence for an active role of human carriers in LD transmission and *L. pneumophila* spread is increasing. This hypothesis is even more convincing than the hypothesis of the atmospheric dispersion of contaminated aerosols for more than 10 Km, in explaining the long-distance LD outbreaks.¹⁴

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Antimicrobial Curtains: Are They as Clean as You Think?

To The Editor—Hospital-acquired infections have become an increasing public health concern in the last decade. Growing evidence suggests that healthcare textiles, including curtains in patient rooms, sheets and even apparel, are associated with a higher risk of transmission of hospital pathogens and, potentially, increased healthcare-associated infections. Multiple reports have linked textiles to horizontal transmission of pathogens since the first documented fabric-associated outbreak in the late 1970s.3 In recent years, technology and innovation have led to the use of antimicrobial fabrics, designed to decrease the spread of organisms through pretreated clothing, curtains, and sheets. In 2014, our institution decided to switch all curtains to antimicrobial fabric. Because of this change, facilities managers decided that it was no longer necessary to clean or exchange curtains between patient uses unless they were clearly soiled. We aimed to determine the degree of bacterial contamination of antimicrobial curtains in our medical intensive care unit (MICU).

This infection control project was performed at a 650-bed, academic, teaching hospital in the greater Milwaukee area. We sampled 20 curtains from 10 different patient rooms in the