COMMENTARY

Waterborne Pathogen Detection: More than Just "Location, Location, Location..."

Brooke K. Decker, MD;[1] Tara N. Palmore, MD[2]

(See the article by Cristina et al, on pages 122–129.)

The complexity of modern hospitals and increasing proportion of immunologically vulnerable patients make healthcare facility safety a top priority for hospital epidemiologists. Contamination of hospital water systems and point-of-use outlets is a widespread, tenacious problem for which a lasting solution remains elusive. Even when municipal water is maintained within accepted standards of chlorination, structural features of hospital plumbing can lead to contamination of water as it flows distally toward the points of use. Hospitals tend to have large, complex waterworks with low-flow areas that produce stagnation and biofilm formation; hot and cold water temperatures that are not well regulated may be ideal for bacterial growth. Although these conditions occur in other facilities, the susceptibility of hospitalized patients and the presence of invasive devices put them at high risk for infection with organisms that contaminate hospital water. *Legionella* species are important waterborne pathogens; Enterobacteriaceae, *Pseudomonas* species, *Burkholderia* species, *Aeromonas* species, *Stenotrophomonas* species, and *Acinetobacter* species are among the other organisms that are frequently identified in healthcare facility water and cause nosocomial infection in immunocompromised patients.

In this issue, Cristina et al[1] report the findings of a study to determine the extent and narrow down the site of hospital water contamination. The investigators cultured water at several point-of-use outlets (faucets) in 2 Italian hospitals, then disinfected the faucets by an unspecified method and flame sterilization to isolate water from deeper in the plumbing system. Their results demonstrated significantly greater contamination of cold water from points of use than from the plumbing system with *Aeromonas* species (9.2% vs 1.3%; $P < .05$) and all gram-negative organisms (31.65 vs 6.6%; $P < .001$). Hot water from outlets and deeper in the plumbing system both grew *Legionella* in high proportions (47.4% vs 42.1%, respectively), and bacterial colony counts were far higher at the points of use than from deeper within the plumbing system. Serial measurements demonstrated that chlorine levels and hot water temperatures were inappropriately low, which are conditions that promote growth of *Legionella* species, nontuberculous *Mycobacteria* (not explored in this study), and other waterborne pathogens.

Contamination of aerators and other sink components has been demonstrated previously and, in some cases, implicated in nosocomial outbreaks of both *Legionella* and other gram-negative bacteria. Plumbing features that promote stagnation of water and growth of biofilm have been associated with nosocomial acquisition of infection, including Legionnaire's disease.[2] Touchless faucets may provide a particularly hospitable habitat,[3-5] and faucet aerators, as mentioned in this study, have been found to be contaminated with *Legionella* species[6-9] and other gram-negative pathogens.[10,11] Medical equipment that is rinsed in tap water, such as endoscopes, undergoes subsequent disinfection that should be adequate to eliminate the risk from waterborne bacteria. However, contaminated water could splash directly onto patients or patient care equipment, or potentially transfer to the hands of healthcare personnel who are washing hands in the sink. The irony of the latter scenario runs deep, but the possibility remains hypothetical because of a lack of convincing evidence of waterborne transmission to patients, even in the best-documented reports.[12,13]

Although water sources are implicated in the nosocomial acquisition of *Legionella* species, their role in transmission of nonfastidious gram-negative bacteria in comparison to other infection risk factors (such as breaches in hand hygiene) is not clearly understood. Pathogens abound in the patient environment, but relatively few are proven to make the transition to causing colonization or disease. Sites that are especially vulnerable to microbial acquisition (such as mucous membranes, surgical wounds, and catheters) are much less likely to come directly in contact with the environment than with the hands of healthcare providers.

Affiliations: 1. Critical Care Medicine Department, National Institutes of Health Clinical Center, Bethesda, Maryland; 2. National Institute of Allergy and Infectious Diseases and National Institutes of Health Clinical Center, National Institutes of Health, Bethesda, Maryland.

Received November 15, 2013; accepted November 19, 2013; electronically published January 13, 2014.

 0899-823X/2014/3502-0003. DOI: 10.1086/675067

Culturing hospital water, high-touch surfaces, or healthcare personnel attire will frequently demonstrate the presence of potential pathogens. We know patients acquire these pathogens in healthcare settings. What is missing is hard evidence of cross-transmission from these environmental sources and fomites to patients. The paradox of the chicken and egg often applies to environmental isolates: a patient colonized by a multidrug-resistant organism is reasonably likely to contaminate his or her environment, including the room sink, with that organism. Strain typing matches of isolates from sinks and patients provide circumstantial evidence, but they do not demonstrate where the organism was first or whether the sink isolate came from the patient or the patient acquired the sink isolate.

It is not entirely clear how to use data like the findings of Cristina et al[1] to improve patient safety. There are divergent opinions on the value of water surveillance and disinfection in the absence of an outbreak. Some European guidelines and some US experts recommend prospective monitoring for *Legionella* species and other gram-negative colony counts, with thresholds that trigger action.[6,11] Others, including the US Centers for Disease Control and Prevention, recommend that approach when a water source of patient infection is suspected or confirmed, as in a cluster of cases of *Legionella* infection.[14,15] Although some have recommended routine cleaning of aerators or sink drains,[7] preemptively removing all potentially pathogenic organisms from biofilms within the hospital water system is a Sisyphean task with unclear payoff for the effort. Such cleaning and disinfection should certainly be done to remediate a situation in which transmission from sinks is known or strongly suspected.[9]

The work of Cristina et al[1] is valuable in quantifying the frequency, magnitude, and location of the potential hazard to patients from hospital water in their facilities. There is still a significant gap in our understanding of how and when such risk translates to patient infections.

ACKNOWLEDGMENTS

Potential conflicts of interest. All authors report no conflicts of interest relevant to this article. All authors submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest, and the conflicts that the editors consider relevant to this article are disclosed here.

Financial support. This research was supported by the National Institutes of Health Clinical Center.

Address correspondence to Tara N. Palmore, MD, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD (tpalmore@mail.nih.gov).

REFERENCES

1. Cristina ML, Spagnolo AM, Casini B, et al. The impact of aerators on water contamination by emerging gram-negative opportunists in at-risk hospital departments. *Infect Control Hosp Epidemiol* 2014;35(2):122–129(in this issue).
2. Heffelfinger JD, Kool JL, Fridkin S, et al. Risk of hospital-acquired legionnaires' disease in cities using monochloramine versus other water disinfectants. *Infect Control Hosp Epidemiol* 2003;24(8):569–574.
3. Best M, et al. Legionellaceae in the hospital water-supply: epidemiological link with disease and evaluation of a method for control of nosocomial legionnaires' disease and Pittsburgh pneumonia. *Lancet* 1983;2(8345):307–310.
4. Halabi M, et al. Non-touch fittings in hospitals: a possible source of *Pseudomonas aeruginosa* and *Legionella* spp. *J Hosp Infect* 2001;49(2):117–121.
5. van der Mee-Marquet N, et al. Non-touch fittings in hospitals: a procedure to eradicate *Pseudomonas aeruginosa* contamination. *J Hosp Infect* 2005;60(3):235–239.
6. Stout JE, et al. Role of environmental surveillance in determining the risk of hospital-acquired legionellosis: a national surveillance study with clinical correlations. *Infect Control Hosp Epidemiol* 2007;28(7):818–824.
7. Cross DF, Benchimol A, Dimond E.G. The faucet aerator: a source of pseudomonas infection. *N Engl J Med* 1966;274(25): 1430–1431.
8. Kappstein I, et al. Aerators as a reservoir of *Acinetobacter junii*: an outbreak of bacteraemia in paediatric oncology patients. *J Hosp Infect* 2000;44(1):27–30.
9. Weber DJ, et al. Faucet aerators: a source of patient colonization with *Stenotrophomonas maltophilia*. *Am J Infect Control* 1999; 27(1):59–63.
10. Muyldermans G, et al. Neonatal infections with *Pseudomonas aeruginosa* associated with a water-bath used to thaw fresh frozen plasma. *J Hosp Infect* 1998;39(4)309–314.
11. L'eau dans les etablissements de sante. Paris: Ministere de la Sante et des Solidarites, 2005.
12. Hota S, et al. Outbreak of multidrug-resistant *Pseudomonas aeruginosa* colonization and infection secondary to imperfect intensive care unit room design. *Infect Control Hosp Epidemiol* 2009;30(1):25–33.
13. Lowe C, et al. Outbreak of extended-spectrum β-lactamase-producing *Klebsiella oxytoca* infections associated with contaminated handwashing sinks. *Emerg Infect Dis* 2012;18(8):1242–1247.
14. Kool JL, et al. More than 10 years of unrecognized nosocomial transmission of Legionnaires' disease among transplant patients. *Infect Control Hosp Epidemiol* 1998;19(12):898–904.
15. Sehulster L, Chinn RYW. Guidelines for environmental infection control in health-care facilities. Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). *MMWR Recomm Rep* 2003;52(RR-10):1–42.