Hospital Tap Water

A Reservoir of Risk for Health Care–Associated Infection

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Abstract: Accepted as our most reliable weapon in the battle to reduce health care–associated infections, hospital tap water has also been recognized as "the most overlooked, important, and controllable source of HAI." Peer-reviewed literature has demonstrated that hospital tap water contains microbial pathogens and that biofilms in water systems resist disinfection and deliver pathogenic organisms to the health care environment. At-risk patients are susceptible to infection through direct contact, ingestion, and inhalation of waterborne pathogens. Systemic water treatment technologies reduce levels of recognized waterborne pathogens; however, they vary in initial and long-term maintenance costs, efficacy against specific organisms, and compatibility with facility plumbing system materials, and they cannot eradicate biofilms within health care facility plumbing. Existing point-of-use filtration technologies have been reported to interrupt clinical outbreaks of infection due to recognized waterborne pathogens in the health care environment and may offer a cost-effective complementary infection control strategy, particularly when targeted for patients at high risk.

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According to the US Centers for Disease Control and Prevention, health care–associated infections (HAIs) account for an estimated 1.7 million infections and 99,000 deaths annually in American hospitals.[1] Accepted as perhaps our most reliable weapon in the battle to reduce HAIs, hospital tap water has also been recognized paradoxically as a source of such infections. One investigation has estimated that 1400 deaths occur each year as a result of waterborne nosocomial pneumonias attributable to *Pseudomonas aeruginosa* alone.[2] However, despite concerns regarding the increasing incidence of serious HAIs due to multidrug resistant gram-negative pathogens, the risk of waterborne transmission of these microbes has received relatively little attention.

Regarding the risk of waterborne pathogens such as *Legionella*, Dr Bruce Dixon, director of the Allegheny County Health Department, has summed up the problem succinctly, "If you don't look for it, you won't find it. If you don't find it, you don't think you have a problem. If you don't think you have a problem, you don't do anything about it."[3] Indeed, an understanding of the ecology of waterborne pathogens in the health care environment is necessary to gain further insight into why this risk may go largely unrecognized. Waterborne microbes thrive to varying degrees in hot and cold water. Whereas cold water is delivered directly to the point of use, hot water is supplied via a recirculation loop, which contains nutrients to nourish waterborne microbes, maintains favorable temperatures for microbial growth, and promotes the formation of biofilm on internal surfaces of pipes and fixtures. Moreover, waterborne microbes, adapted to life in a relatively nutrient-poor environment, may be difficult to culture using nutrient-rich media for short incubation periods (eg, 24–48 hours at 37°C). Successful culturing may require special media (eg, R2A) and extended incubation periods at lower temperatures (eg, 25°C for 14–28 days).

BIOFILM

Ubiquitous in hospital plumbing as in nature, biofilm is a microbially derived sessile community characterized by cells that are irreversibly attached to a substratum or interface or to one another, are embedded in a matrix of extracellular polymeric substances that they have produced, and exhibit an altered phenotype with respect to growth rate and gene transcription.[4] Biofilm affords microbial pathogen protection from adverse environmental conditions outside the host,[5] and it has been established that biofilm bacteria display a higher level of resistance to antimicrobial agents[6–10] and environmental controls (eg, UV light, metals, and acid pH)[11–13] than do planktonic (free-floating) bacteria. Proposed mechanisms contributing to antimicrobial resistance of biofilm bacteria are many, including extracellular polymeric substance–antimicrobial interaction, altered bacterial cell surface properties, slower growth rates, enzyme production, plasmids, the contribution of phenotypically resistant microbes within the biofilm, and the surface topography of the material to which biofilm is adherent.[5] In addition, for clinically important organisms such as *P. aeruginosa*, a single genetic locus is associated with both the ability to form biofilm and antimicrobial resistance.[14]

DETERMINANTS OF INFECTIVITY

Pathogen virulence and density clearly impact the likelihood of infection upon waterborne exposure. Some waterborne pathogens of recognized virulence include *P. aeruginosa*, *Stenotrophomonas maltophilia*, *Acinetobacter*

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species, and *Legionella* species. In addition, organisms such as atypical mycobacteria, *Aspergillus*, *Fusarium*, and *Cryptosporidium* are among other potential waterborne pathogens of substance. Some (eg, *Pseudomonas* species, *Legionella* species, and atypical mycobacteria) are also resistant to digestion by free-living waterborne amoebae that, upon phagocytosing, these bacteria may act as "Trojan horses" protecting the pathogens from disinfection by chlorination, acid pH, osmotic pressure, and temperature and transporting them to the point of use in the health care environment.[15]

Nevertheless, in the normal host, bacterial exposures from showers, faucets, and other aqueous sources (eg, by inhalation or ingestion) are typically cleared by innate defenses (eg, mucociliary escalator clearance of inhaled organisms).[16] Thus, immunocompromised hosts—recipients of bone marrow and solid organ transplants, individuals with congenital or acquired immunodeficiency syndromes, oncology and burn patients, critically ill patients in intensive care units, smokers, individuals with chronic cardiac and respiratory disorders, and residents of skilled nursing facilities—are likely to be at higher risk. It is precisely for such patients that appropriate environmental infection control methods are most important.

CONTROL MEASURES

Systemic Water Treatment

Methods used to disinfect water lines must address amoeba and biofilm to be effective. We have reviewed this subject[17] and provide a brief summary of that information, as well as focus on updates from the recent literature. Seven preventive water treatment strategies, excluding point-of-use (POU) water filters, have been used, usually in response to an outbreak. They include hot water flushing of the plumbing system, chlorination, chlorine dioxide, monochloramine (used exclusively at the municipal treatment level in the United States), copper-silver ionization, UV light, and ozonation. Each method has advantages and disadvantages related to ease of implementation, cost, maintenance issues, and short- and long-term effectiveness. All strategies, in contrast to POU filtration, are not completely effective in the long run because maintenance of systemic disinfection agents at levels that would prevent recolonization and biofilm elaboration is difficult and because biofilm is known to protect microbes against systemic disinfection strategies.

Hot water flushing is the easiest to implement but requires that all parts of the plumbing system be exposed to high-temperature water for a period during which use of water outlets is precluded. The potential for inadvertent scalding at the point of use is also an issue because water temperatures often exceed 65°C. Recent data corroborate earlier observations that hot water flushing is inadequate in eliminating *Legionella* from plumbing systems over the longer term,[18] even though temperatures above 59°C were associated with an inability to culture *Legionella*.[19,20] A single treatment has been shown to reduce the number of sample sites from which *Legionella* organisms are recovered to 45% and then to 9% after a second treatment. In another study, a system flush using hot water at 80°C was incapable of eradicating *Legionella* serogroup 5, and only 1 serogroup 6 strain was eradicated.[21] That this is not an isolated event is supported by the observation of a persistent strain of *Legionella* in a hospital during the course of 15 years.[22] Hyperchlorination was added to hot water flush, and *Legionella* organisms were still recovered from the showers, prompting the disconnection of central water supply lines and the use of electrical hot water heaters for showers. This resulted in a substantial reduction in recoverable *Legionella* without clinical incident.[23]

Chlorination is also simple to establish, but it can be challenging to maintain adequate levels of chlorine throughout the system, as may be inferred from its less than stellar performance in maintaining a microbially free environment. Electrolytic chlorine generation systems in large-scale studies seem to be no better than sodium hypochlorite.[24] However, chlorination is not free of potential by-product–associated genotoxicity, which is an emerging concern.[25] Bench-scale chlorination compared with UV irradiation showed that both methods were effective in reducing the bioburden of indicator organisms. However, pathogens of clinical concern were less affected by chlorination.[26] Data continue to accumulate, suggesting that waterborne pathogens are protected against chlorination by amoeba and biofilm.[27,28] These findings suggest that chlorination may be less effective than other alternatives, despite its relative cost efficiencies.

Much of the earlier characterization of chlorine dioxide is supported by more recent literature. The potentially corrosive properties of chlorine dioxide are not evident in the literature, and the maintenance of an effective concentration in hot water systems remains poorly characterized. Chlorine dioxide effectively reduces but may not eliminate *Legionella*.[28,29] Testing of multiple disinfection strategies has indicated that chlorine dioxide may be the most effective systemic disinfection regimen for the control of *Legionella*.[28] In simulated potable water system testing, chlorine dioxide was shown to be more effective in reducing heterotrophic bacterial counts, with reduced levels of some but not all organic halogenated by-products.[30] Relative to chlorine, it remains more costly to install.

Efficacy studies of chloramines alone or in combination with free chlorine attest to the fact that neither alone nor in combination is complete as a disinfectant.[31] In addition, the spectrum of potentially harmful halogenated by-products left by combination chlorination regimens[32] will take some time to assess. Chlorine and chloramines also differ in their spectrum of antimicrobial activity. For example, *Klebsiella pneumoniae* seems to be more sensitive to chloramines than to free chlorine under certain conditions.[33]

Copper-silver ionization has been recently reviewed when used either alone or in combination with other systemic disinfection strategies.[34] A review of 10 copper-silver ionization studies uniformly supported its effectiveness to varying degrees. However, this technology was also demonstrated to be more effective when used in combination with another disinfection technology. More importantly, none of these studies were able to demonstrate sustained eradication of *Legionella*. From a practical perspective, however, an in vitro study of copper and silver ions alone and in combination provided evidence to suggest bactericidal efficiencies

greater than 99.99% against the most significant clinical waterborne microbes, *P. aeruginosa*, *Acinetobacter baumannii*, and *S. maltophilia*, in addition to *Legionella*.[35,36]

As indicated previously,[17] UV light, which is rarely used in the hospital setting in the United States, has poor penetrating power, is only effective at the source of irradiation, and remains prone to fouling of the quartz sleeves surrounding the UV lamp.[37] The one notable advance is the use of light-emitting diodes to deliver UV-A radiation, which has been shown to be effective as a bactericidal systemic treatment. It must be noted, however, that this technology awaits further characterization.[38] Like other combinations of systemic disinfection strategies, it should not be surprising that UV and ozonation used in combination have been shown to be better than either used alone.[39] Although regulations governing the oxidative by-products of halogenated disinfectants exist, additional by-products continue to be identified. One obvious consideration is that any systemic disinfection strategy will always bear a level of uncertainty concerning toxic by-products that accrue from its use. In contrast, POU filtration lacks this drawback and offers the potential benefit of immediate and complete effectiveness against waterborne bacteria, fungi, and protozoa.

POU Water Filtration

Although the implementation of POU water filtration for at-risk patient populations in the health care setting is a relatively new phenomenon in the United States, this technology has been used extensively in Europe for the past 10 years. European health care institutions have long recognized the potential threat posed by waterborne bacteria, fungi, and protozoa to neonatal, elderly, and immunocompromised patient populations, as well as patients in intensive care units.

Point-of-use filtration studies have appeared extensively in the scientific literature and have repeatedly addressed the role of filtration technology in both reducing infections due to waterborne pathogens and saving money for the health care institution. These studies have focused primarily upon *Legionella* and *P. aeruginosa*, although studies in progress are beginning to investigate other common waterborne pathogens such as *Acinetobacter* species and *S. maltophilia*.

Sheffer et al[40] conducted a study during which it was demonstrated that POU filters labeled for a maximum use life of 7 days completely eliminated *Legionella pneumophila* and *Mycobacterium gordonae* from hot tap water during an 8-day period of use. Vonberg et al[41] contributed to the ever-growing body of evidence supporting the efficacy of POU filters through the observation that 99.6% (n = 256) of water samples obtained during their study were devoid of *Legionella* species. In the single sample that was positive, *Legionella* concentration was 1 colony-forming unit/mL.

After an observation period of 11 months, during which a high incidence of *P. aeruginosa* bacteremia was observed in a hematology unit with severely neutropenic patients, Vianelli et al[42] performed extensive sampling in an attempt to trace the environmental source of the isolates that were appearing in patient blood cultures. Upon identifying faucets and showers in the unit as the primary environmental sources of those isolates, POU filters were installed on all hematology unit water outlets. Highly statistically significant reductions in bloodstream infections were subsequently observed over the next 2 years.

In a study spanning a period of 2 years, Trautmann et al[43] documented a decrease in the monthly rate of *P. aeruginosa* infections in a surgical intensive care unit (SICU) from 2.5 per month before POU filter installation to 0.8 per month after POU filter installation. Of particular interest in this study was the fact that when implementation of POU filters was initiated during month 13, only 3 of a total of 9 faucets were fitted with filters. When the infection rate began to trend downward, POU filters were installed on the remaining 6 faucets at the beginning of month 17. During the final 8 months of the study (months 17–24), representing the entire time when all 9 SICU faucets were equipped with filters, not a single *P. aeruginosa* infection was observed for 6 of the 8 months.

Van der Mee-Marquet et al[44] surveyed pseudomonal infections of blood, urological, and pulmonary origin encompassing 23,611 patient-days in the intensive care environment during a period of 7.5 years (90 months). During a time frame of 2.5 years (30 months) before the use of POU filtration, 8.7 infections per 1000 patient-days were observed, whereas in the 5 years (60 months) after installation of POU filters, only 3.2 infections per 1000 patient-days were recorded.

In the neonatal intensive care unit, LaFerriere[45] used a variety of infection control interventions, including POU filtration, to effect a dramatic decline in HAIs attributable to *P. aeruginosa*.

The establishment of a genetic link between pathogenic bacterial strains isolated from tap water and strains isolated from patients has been extensively detailed.[2,46–55] These studies substantiate that the transfer of waterborne bacteria from unfiltered tap water sources in the health care environment to at-risk patients via inhalation of water vapor, ingestion (eg, drinking and ingestion of ice), and direct contact (eg, showering, bathing, wet hands of a health care provider, and contact with medical devices rinsed with tap water) is indeed a source of concern.

A potential limitation to the effectiveness of POU filtration and indeed to all efforts at water decontamination is the fact that waterborne pathogens, such as *P. aeruginosa*, may enter the health care environment endogenously via patient colonization. They may also enter via other contaminated fluids and instruments, such as endoscopes, bronchoscopes, artificial saliva, and even mouth swabs.[56] Thus, within the broader context of infection control monitoring, adequate staff and patient training with respect to the appropriate use, maintenance, and replacement of POU filters is important to reduce the risk of possible retrograde contamination of incoming tap water.[57] Another potential limiting factor is the additional cost of POU filters and other associated methods of risk reduction.

The added cost incurred for HAIs in US hospitals has been estimated at $15,275 to $38,656 per infection.[58,59] Although scientific studies have supported the use of POU water filters to reduce at-risk patient exposure to waterborne

pathogens, economic benefits can also be realized by health care institutions that adopt this technology. Hall et al[60] demonstrated that costs associated with filtered drinking water supplied to immunocompromised patients were drastically lower than those for both bottled sterile water and commercially available bottled water. Trautmann et al[51] recounted savings realized on the cost of antibiotics used to treat *P. aeruginosa* infections in a SICU during implementation of POU water filters on faucets. Finally, imminent changes to be implemented by the US Centers for Medicare and Medicaid Services to its reimbursement policy, namely, nonreimbursement of health care facilities for specific HAI, may encourage efforts to engineer a safer patient care environment by means such as implementation of POU filtration technology, not only to protect patients but also to avoid bearing the full cost of HAI treatment.

CURRENT APPROACHES

The control of waterborne pathogens in US health care institutions is at best a fragmented work in progress. As has been previously stated, the United States lags far behind Europe in recognition of tap water as an important source of HAIs. Furthermore, the approaches taken by many US health care institutions to control waterborne pathogens vary greatly and generally fall into 1 of 4 categories: nonexistent, sporadic, incomplete, and enlightened. The *nonexistent* approach is self-explanatory. These institutions either lack awareness or actively choose not to address the problem at all. The *sporadic* approach is characterized by responding to an outbreak by culturing water and temporarily installing some preventive measures such as POU filters. When the outbreak ends, POU filters are removed, leaving the facility unprotected as the clock ticks toward the inevitable next outbreak. The *incomplete* approach involves an undisciplined and haphazard approach to water culturing, installation of a systemic disinfection technology, and failure to implement POU filtration. This approach leaves the facility continually vulnerable to biofilm in the plumbing system, changes in water pressure, and seasonal variations in water quality. It also ignores studies conducted indicating that electronic (nontouch) faucets can harbor and encourage the proliferation of waterborne pathogens due to the fact that their electrical solenoid valves remain warm at all times, thereby providing an incubated environment for planktonic and biofilm-based bacteria, fungi, and protozoa.[61–63] Finally, the *enlightened*, thought-leading facilities have recognized the need to perform routine microbial analyses of tap water in at-risk patient areas, to install an appropriate systemic disinfection technology, and to continually use POU filters to protect their most vulnerable patients.

SUMMARY

It has been suggested that hospital water distribution systems are among "the most overlooked, important, and controllable sources of HAI."[2,5] Available evidence in the peer-reviewed literature has demonstrated that hospital tap water contains microbial pathogens and that biofilms in water delivery systems resist disinfection and deliver pathogenic organisms to the health care environment. At-risk patients are susceptible to infection through direct contact, ingestion, and inhalation of waterborne pathogens, as numerous clinical reports attest. Systemic water treatment technologies reduce levels of recognized waterborne pathogens; however, they vary in initial and long-term maintenance costs, efficacy against specific organisms, and compatibility with facility plumbing system materials. Moreover, they do not permanently and completely eradicate biofilms within health care facility plumbing. Finally, existing POU filtration technologies have been reported to interrupt clinical outbreaks of infection due to recognized waterborne pathogens in the health care environment and may offer a cost-effective complementary infection control strategy, particularly when targeted for patients at high risk.

REFERENCES

1. Centers for Disease Control and Prevention. Estimates of Healthcare-Associated Infections. Available at: http://www.cdc.gov/ncidod/dhqp/hai.html. Accessed January 14, 2008.
2. Anaissie EJ, Penzak SR, Dignani C. The hospital water supply as a source of nosocomial infections - a plea for action. *Arch Int Med*. 2002; 162:1483–1492.
3. Squier CL, Stout JE, Krsytofiak S, et al. A proactive approach to prevention of health care-acquired Legionnaires' disease: the Allegheny County (Pittsburgh) experience. *Am J Infect Control*. 2005;33(6):360–367.
4. Donlan RM, Costerton JW. Biofilms: survival mechanisms of clinically relevant microorganisms. *Clin Microbiol Rev*. 2002;15(2):167–193.
5. Lindsay D, von Holy A. Bacterial biofilms within the clinical setting: what healthcare professionals should know. *J Hosp Infect*. 2006;64: 313–325.
6. Costerton JW, Lewandowski Z, Caldwell DE, et al. Microbial biofilms. *Annu Rev Microbiol*. 1995;49:711–745.
7. Donlan RM. Biofilms: microbial life on surfaces. *Emerg Infect Dis*. 2002;8:881–890.
8. Lindsay D, von Holy A. Different responses of planktonic and attached *Bacillus subtilis* and *Pseudomonas fluorescens* to sanitizer treatment. *J Food Prot*. 1999;62:368–379.
9. Dodds MG, Grobe J, Stewart PS. Modeling biofilm antimicrobial resistance. *Biotechnol Bioeng*. 2000;68:456–465.
10. Meyer B. Approaches to prevention, removal and killing of biofilms. *Int Biodeterior Biodegrad*. 2003;51:249–253.
11. Hall-Stoodley L, Costerton JW, Stoodley P. Bacterial biofilms: from the natural environment to infectious diseases. *Nat Rev Microbiol*. 2004;2: 95–108.
12. Hall-Stoodley L, Stoodley P. Biofilm formation and dispersal and the transmission of human pathogens. *Trends Microbiol*. 2005;13:7–10.
13. Jefferson KK. What drives bacteria to produce a biofilm? *FEMS Microbiol Lett*. 2004;236:163–173.
14. Drenkard E, Ausubel FM. Pseudomonas biofilm formation and antimicrobial resistance are linked to phenotypic variation. *Nature*. 2002; 416:740–743.
15. Greub G, Raoult D. Microorganisms resistant to free-living amoebae. *Clin Microbiol Rev*. 2004;17:413–433.
16. Prince A. Biofilms, antimicrobial resistance, and airway infection. *N Engl J Med*. 2002;347:1110–1111.
17. Ortolano GA, McAlister MB, Angelbeck JA, et al. Hospital water point-of-use filtration: a complementary strategy to reduce the risk of nosocomial infection. *Am J Infect Control*. 2005;33(5 suppl 1):S1–S19.
18. Mouchtouri V, Velonakis E, Hadjichristodoulou C. Thermal disinfection of hotels, hospitals, and athletic venues hot water distribution systems contaminated by *Legionella* species. *Am J Infect Control*. 2007;35(9): 623–627.
19. Lasheras A, Boulestreau H, Rogues AM, et al. Influence of amoebae and physical and chemical characteristics of water on presence and proliferation of *Legionella* species in hospital water systems. *Am J Infect Control*. 2006;34(8):520–525.

20. Saby S, Vidal A, Suty H. Resistance of *Legionella* to disinfection in hot water distribution systems. *Water Sci Technol*. 2005;52(8):15–28.
21. Perola O, Kauppinen J, Kusnetsov J, et al. Persistent *Legionella pneumophila* colonization of a hospital water supply: efficacy of control methods and a molecular epidemiological analysis. *APMIS*. 2005;113(1): 45–53.
22. Scaturro M, Dell'eva I, Helfer F, et al. Persistence of the same strain of *Legionella pneumophila* in the water system of an Italian hospital for 15 years. *Infect Control Hosp Epidemiol*. 2007;28(9):1089–1092. Epub 2007 Jul 3.
23. Oliveira MS, Maximino FR, Lobo RD, et al. Disconnecting central hot water and using electric showers to avoid colonization of the water system by *Legionella pneumophila*: an 11-year study. *J Hosp Infect*. 2007;66(4):327–331.
24. Clevenger T, Wu Y, Degruson E, et al. Comparison of the inactivation of *Bacillus subtilis* spores and MS2 bacteriophage by MIOX, ClorTec and hypochlorite. *J Appl Microbiol*. 2007;103(6):2285–2290.
25. Richardson SD, Plewa MJ, Wagner ED, et al. Occurrence, genotoxicity, and carcinogenicity of regulated and emerging disinfection by-products in drinking water: a review and roadmap for research. *Mutat Res*. 2007;636(1–3):178–242.
26. Blatchley ER 3rd, Gong WL, Alleman JE, et al. Effects of wastewater disinfection on waterborne bacteria and viruses. *Water Environ Res*. 2007;79(1):81–92.
27. Berry D, Xi C, Raskin L. Microbial ecology of drinking water distribution systems. *Curr Opin Biotechnol*. 2006;17(3):297–302. Epub May 15, 2006.
28. Loret JF, Robert S, Thomas V, et al. Comparison of disinfectants for biofilm, protozoa and *Legionella* control. *J Water Health*. 2005;3(4): 423–433.
29. Zhang Z, McCann C, Stout JE, et al. Safety and efficacy of chlorine dioxide for *Legionella* control in a hospital water system. *Infect Control Hosp Epidemiol*. 2007;28(8):1009–1012.
30. Rand JL, Hofmann R, Alam MZ, et al. A field study evaluation for mitigating biofouling with chlorine dioxide or chlorine integrated with UV disinfection. *Water Res*. 2007;41(9):1939–1948.
31. Chen C, Zhang XJ, He WJ, et al. Simultaneous control of micro-organisms and disinfection by-products by sequential chlorination. *Biomed Environ Sci*. 2007;20(2):119–125.
32. Hua G, Reckhow DA. Characterization of disinfection byproduct precursors based on hydrophobicity and molecular size. *Environ Sci Technol*. 2007;41(9):3309–3315.
33. Goel S, Bouwer EJ. Factors influencing inactivation of *Klebsiella pneumoniae* by chlorine and chloramine. *Water Res*. 2004;38(2):301–308.
34. Cachafeiro SP, Naveira IM, García IG. Is copper-silver ionisation safe and effective in controlling *Legionella*? *J Hosp Infect.* 2007;67(3):209–216.
35. Huang HI, Shih HY, Lee CM, et al. In vitro efficacy of copper and silver ions in eradicating *Pseudomonas aeruginosa*, *Stenotrophomonas maltophilia* and *Acinetobacter baumannii*: implications for on-site disinfection for hospital infection control. *Water Res*. 2008;42(1–2):73–80. [Epub ahead of print].
36. Mòdol J, Sabrià M, Reynaga E, et al. Hospital-acquired legionnaires disease in a university hospital: impact of the copper-silver ionization system. *Clin Infect Dis*. 2007;44(2):263–265.
37. Nessim Y, Gehr R. Fouling mechanisms in a laboratory-scale UV disinfection system. *Water Environ Res*. 2006;78(12):2311–2323.
38. Mori M, Hamamoto A, Takahashi A, et al. Development of a new water sterilization device with a 365 nm UV-LED. *Med Biol Eng Comput*. 2007;45(12):1237–1241.
39. Oh BS, Park SJ, Jung YJ, et al. Disinfection and oxidation of sewage effluent water using ozone and UV technologies. *Water Sci Technol*. 2007;55(1–2):299–306.
40. Sheffer P, Stout J, Wagener M, et al. Efficacy of new point-of-use water filter for preventing exposure to *Legionella* and waterborne bacteria. *Am J Infect Cont*. 2005;33(5 suppl 1):S20–S25.
41. Vonberg RP, Eckmanns T, Bruderek J, et al. Use of terminal tap water filter systems for prevention of nosocomial legionellosis. *J Hosp Infect*. 2005;60:159–162.
42. Vianelli N, Giannini MB, Quarti C, et al. Resolution of a *Pseudomonas aeruginosa* outbreak in a hematology unit with the use of disposable sterile water filters. *Haematologica*. 2006;91(7):983–985.
43. Trautmann M, Royer H, Helm E, et al. *Pseudomonas aeruginosa*: new insights into transmission pathways between hospital water and patients. *Filtration*. 2004;1(suppl 1):63–70.
44. Van der Mee-Marquet N, Bloc D, Brulard V, et al. Water microfiltration: a procedure to prevent *Pseudomonas aeruginosa* Infection. Abstract presented at the XVI Congress Nat. Soc. Franc. D'Hyg. Hospitaliere, Water and Hospital Symposium; June 4, 2005; Reims, France. Abstract S.137.
45. LaFerriere C. Infection control measures vs. waterborne microbes in the NICU. *SHEA Annual Meeting Poster Session*. Chicago, IL: Society for Healthcare Epidemiology of America; 2006.
46. Blanc DS, Nahimana I, Petiqnat C, et al. Faucets as a reservoir of endemic *Pseudomonas aeruginosa* colonization/infections in intensive care units. *Intensive Care Med*. 2004;30:1964–1968.
47. Kline S, Cameron S, Streifel A, et al. An outbreak of bacteremias associated with *Mycobacterium mucogenicum* in a hospital water supply. *Infect Control Hosp Epidemiol*. 2004;25(12):1042–1049.
48. Bert F, Maubec E, Bruneau B, et al. Multi-resistant *Pseudomonas aeruginosa* outbreak associated with contaminated tap water in a neurosurgery intensive care unit. *J Hosp Infect*. 1998;39(1):53–62.
49. Ferroni A, Nguyen L, Pron B, et al. Outbreak of nosocomial urinary tract infections due to *Pseudomonas aeruginosa* in a pediatric surgical unit associated with tap-water contamination. *J Hosp Infect*. 1998;39(4): 301–307.
50. Grundmann H, Kropec A, Hartung D, et al. *Pseudomonas aeruginosa* in a neonatal intensive care unit: reservoirs and ecology of the nosocomial pathogen. *J Infect Dis*. 1993;168(4):943–947.
51. Trautmann M, Michalsky T, Wiedeck H, et al. Tap water colonization with *Pseudomonas aeruginosa* in a surgical intensive care unit (ICU) and relation to *Pseudomonas* infections of ICU patients. *Infect Control Hosp Epidemiol*. 2001;22(1):49–52.
52. Weber DJ, Rutala WA, Blanchet CN, et al. Faucet aerators: a source of patient colonization with *Stenotrophomonas maltophilia*. *Am J Infect Control*. 1999;27:59–63.
53. Von Reyn C, Maslow JN, Barber TW, et al. Arbeit. Persistent colonization of potable water as a source of *Mycobacterium avium* infection in AIDS. *Lancet*. 1994;343:1137–1141.
54. Wallace RJ, Musser JM, Hull SI, et al. Diversity and sources of rapidly growing mycobacteria associated with infections following cardiac surgery. *J Infect Dis*. 1989;159:708–716.
55. Anaissie EG, Stratton SL, Dignani MC, et al. Pathogenic *Aspergillus* species recovered from a hospital water system: a 3-year prospective study. *Clin Infect Dis*. 2002;34:780–789.
56. Iversen BG, Jacobsen T, Eriksen H-M, et al. An outbreak of *Pseudomonas aeruginosa* infection caused by contaminated mouth swabs. *Clin Infect Dis*. 2007;44:795–801.
57. Rogues A-M, Boulestreau H, Lasheras A, et al. Contribution of tap water to patient colonization with *Pseudomonas aeruginosa* in a medical intensive care unit. *J Hosp Infect*. 2007;67:72–78.
58. Roberts RR, Scott RD II, Cordell R, et al. The use of economic modeling to determine the hospital costs associated with nosocomial infections. *Clin Infect Dis*. 2003;36(11):1424–1432.
59. Zahn C, Miller MR. Excess length of stay, charges, and mortality attributable to medical injuries during hospitalization. *JAMA*. 2003;290(14):1868–1874.
60. Hall J, Hodgson G, Kerr KG. Provision of safe potable water for immunocompromised patients in hospital. *J Hosp Infect.* 2004;58:155–158.
61. Halabi M, Weisholzer-Pittl M, Dchoberj J, et al. Non-touch fittings in hospitals: a possible source of *P. aeruginosa* and *Legionella* spp. *J Hosp Infect*. 2001;49(2):117–121.
62. Merrer J, Girou E, Ducellier D. Should electronic faucets be used in intensive care and hematology units? *Intensive Care Med*. 2005;31: 1715–1718.
63. Chaberny I, Gastmeier P. Should electronic faucets be recommended in hospitals? *Infect Cont Hosp Epidemiol*. 2004;25:997–1000.