ResearchGate

See discussions, stats, and author profiles for this publication at: https://www.researchgate.net/publication/327119182

Sink-Related Outbreaks and Mitigation Strategies in Healthcare Facilities

Article *in* Current Infectious Disease Reports · October 2018

DOI: 10.1007/s11908-018-0648-3

CITATIONS
13

READS
841

2 authors:

Leighanne O. Parkes
McGill University
15 PUBLICATIONS **42** CITATIONS
SEE PROFILE

Susy Hota
University Health Network
43 PUBLICATIONS **678** CITATIONS
SEE PROFILE

Some of the authors of this publication are also working on these related projects:

Designing for Patient Safety View project

General infectious diseases View project

All content following this page was uploaded by Susy Hota on 28 August 2018.

The user has requested enhancement of the downloaded file.

Current Infectious Disease Reports (2018) 20:42
https://doi.org/10.1007/s11908-018-0648-3

HEALTHCARE ASSOCIATED INFECTIONS (G BEARMAN AND D MORGAN, SECTION EDITORS)

Sink-Related Outbreaks and Mitigation Strategies in Healthcare Facilities

Leighanne O. Parkes[1] · Susy S. Hota[2,3]

© Springer Science+Business Media, LLC, part of Springer Nature 2018

Abstract

Purpose of Review In this review, we summarize recent outbreaks attributed to hospital sinks and examine design features and behaviors that contributed to these outbreaks. The effectiveness of various risk mitigation strategies is presented. Finally, we examine investigational strategies targeted at reducing the risk of sink-related infections.

Recent Findings Outbreaks of hospital sink-related infections involve a diverse spectrum of microorganisms. They can be attributed to defects in sink design and hospital wastewater systems that promote the formation and dispersion of biofilm, as well as healthcare practitioner and patient behaviors. Risk mitigation strategies are often bundled; while they may reduce clinical cases, sink colonization may persist. Novel approaches targeting biofilms show promise but require more investigation.

Summary Emphasis should be placed on optimizing best practices in sink design and placement to prevent infections. Hospitals should consider developing a rational surveillance and prevention strategy based on the current design and state of their sinks.

Keywords Sink · Hand hygiene · Hospital-associated infections · Infection control · Biofilm · Multidrug-resistant organisms

Introduction

Since the mid-nineteenth century, when Semmelweis first proposed that simple handwashing could drastically reduce maternal mortality, hand hygiene has been a central tenet of infection prevention and control. As an important enabler of hand hygiene, hospital sinks play an important role in these efforts, and much emphasis has been placed on optimizing their accessibility in patient care settings. Ironically, hospital sinks are rich microbial breeding grounds and reservoirs for the transmission of nosocomial pathogens and resistance genes [1–3]. Accordingly, it behooves us to define effective infection control strategies to minimizing the contamination of hospital sinks and prevent microbial transmission to vulnerable patient populations.

Here, we review recent outbreaks related to contaminated hospital sinks, examining specifically design features and healthcare provider behaviors that contribute to the transmission of sink pathogens. We discuss various risk mitigation strategies that have been employed and their efficacy, with a focus on future directions.

Recent Outbreaks of Sink-Related Infections in Healthcare Facilities

The relationship between sinks and hospital-associated infection with hydrophilic organisms has long been described. Over 40 years ago, epidemiologic studies offered intriguing insights into the transmission of *Pseudomonas aeruginosa* from sinks to patients admitted to burn units, where culture-based environmental screening of sink drainage systems demonstrated high colonization rates of up to 70.2% [4, 5]. Since then, numerous outbreaks have been described, and advances in molecular techniques have strengthened the association between human infections and hospital sinks. Over the past 5 years, there has been an explosion of such outbreak reports, involving an ever-expanding patient population and pantheon of microorganisms.

This article is part of the Topical Collection on *Healthcare Associated Infections*

✉ Susy S. Hota
susy.hota@uhn.ca

[1] Department of Medicine, Division of Infectious Diseases, Jewish General Hospital, McGill University, Pavilion E-0054, 3755 Chemin de la Cote-Sainte-Catherine, Montreal, QC H3T 1E2, Canada

[2] Department of Medicine, Division of Infectious Diseases, University of Toronto, Toronto, ON, Canada

[3] Department of Infection Prevention and Control, University Health Network, 9th Floor – 8 PMB 102, 585 University Avenue, Toronto, ON M5G 2C4, Canada

Published online: 20 August 2018

While any water source within healthcare facilities may be susceptible to colonization with waterborne pathogens, contaminated hand hygiene sinks are commonly implicated as the source of outbreaks. These outbreaks most frequently occur in neonatal and adult intensive care units (ICUs) and burn units, as well as hematology-oncology and transplant wards (Table 1). Accordingly, the individuals most commonly affected by sink-related outbreaks are vulnerable patient populations, including neonates, the critically ill, and immunosuppressed.

Waterborne bacteria predominate in sink-related outbreaks, with *P. aeruginosa* being the most commonly identified organism. Other pathogens include Enterobacteriaceae, such as *Escherichia coli*, *Klebsiella pneumoniae*, *Klebsiella oxytoca*, *Serratia marcescens*, *Enterobacter* species, *Citrobacter* species, and *Pantoea agglomerans*. Non-fermenting organisms such as *Stenotrophomonas maltophilia*, *Acinetobacter baumanii*, *Elizabethkingia meningoseptica*, and *Burkholderia* species as well as *Fusarium* species and *Mycobacterium mucogenicum* have also been described (Table 1).

Multidrug-resistant (MDR) organisms are featured prominently in these reports, with carbapenemases most frequently identified. Enterobacteriaceae producing extended-spectrum beta-lactamases (ESBLs) as well as multidrug-resistant *P. aeruginosa* and *A. baumanii* are also commonly identified. The overrepresentation of MDR organisms in outbreak reports might simply reflect a failure to recognize sink-related transmission of more susceptible pathogens that are not commonly included in infection control surveillance. The true burden of sink-related infections is therefore likely underestimated as there is currently no widespread systematic surveillance strategy addressing this type of hospital-associated infection.

Design Features that Promote Sink-Related Infections

Sinks are complex items, with multiple fixtures that present a unique environment for microorganisms (Fig. 1). There are two main ways that sink design may facilitate the spread of pathogens: (1) by promoting formation of biofilm and (2) by encouraging disruption of established biofilm, resulting in aerosolization, splashing, or contamination of adjacent surfaces.

Promotion of Biofilm

Hospital water systems are rife with biofilm [1, 2, 49••]. Sinks, in particular, are susceptible to biofilm formation, as they are repositories of gray-water (wastewater without fecal contamination). Planktonic bacteria, when in the aqueous environment of a sink, form a biofilm by attaching to and colonizing solid surfaces. A multicellular and sessile bacterial community forms as the bacteria adopt a quorum-sensing phenotype. The bacterial community expands and matures, secreting extracellular polymeric substances, which encase and reinforce the growing colony, trap and concentrate nutrients, and protect against mechanical and chemical disinfection [50]. The polymicrobial constituents interact in complex cooperative and antagonistic ways, resulting in the emergence and transfer of resistance genes and virulence factors [51]. The horizontal transfer of GIM-1 [52], KPC, NDM [53], and MRSA [54] has been demonstrated in hospital sinks.

Sink biofilm formation is often enhanced by certain design features, leading to high microbial burden. These features include the use of plastic traps [36••]; faucets with aerators or other flow modulators [9, 10, 12, 13, 27, 46, 47, 55]; rimmed faucet spouts [7, 36••]; sink rubbers [56]; and overflow holes [31]. When biofilm and corrosion deposits are visibly noted on faucet aerators, *P. aeruginosa* load in water has been demonstrated to be on average 2-log higher compared to water from those faucets without aerators [13]. A number of outbreak investigations cite the presence of biofilm or "slime" coating these sink features, although the method of detection is most often visual [6, 13, 48, 57]. Other means of biofilm detection, such as biomass quantification (heterotropic plate counts, adenosine triphosphate measurement), visualization of structure (confocal laser scanning microscopy), and activity measurements, are more often employed in experimental models [58–61].

Disruption and Dispersion of Biofilm

Cells within the biofilm can actively detach, reverting back to their planktonic phenotype or, passively slough as a consequence of changes in nutrient availability, chemical disruption, or aberrations in fluid dynamics [50, 60]. These sloughed aggregates may then transfer to the hands of healthcare workers and adjacent patient care items such as medications, medical supplies, or devices or make direct contact with patients, causing infection [61]. Two key sink design features that facilitate such aerosolization, splashing, and/or surface contamination are the depth of the sink basin, and the faucet positioning relative to sink drain.

Basin Depth

Shallow basins are thought to cause cross-contamination of hands during handwashing [11] and promote splashing [32, 38, 43] with subsequent contamination of the faucet, sink collar, and adjacent surfaces. In their investigation of several clusters of IMP-4 carbapenemase-producing Enterobacteriaceae (CPE) infections in the ICU setting, Kotsanas et al. identified shallow basins in addition to deteriorating porcelain, and a tap with water flowing directly into the drain, as all contributing to significant and visible water spray [38]. De Gyeter et al. performed air sampling, culturing

such Gram-negatives as *S. maltophilia*, *S. marcescens*, and *Pseudomonas* species from bioaerosols generated when the faucets in their ICU were running. As a consequence of the short vertical distance (20 cm) between the faucet and the drain, contamination of the faucet with bioaerosol was postulated as contributing to their facility's outbreak [32].

Faucet Positioning

Water flowing directly into the drain can disrupt established biofilm in sink traps, causing pathogens to disperse via the Venturi-effect. Numerous studies have identified or postulated this mechanism as contributing to outbreaks [6, 9, 14••, 15, 19, 28, 31, 32, 36••, 44, 55, 62]. Jencson and colleagues used culture-based methods to demonstrate the dissemination of *Candida* species from the sink drain onto the basin and surrounding countertops upon coincident water flow [63]. Hota et al. injected fluorescein into sink traps demonstrating spray of drain and trap contents up to 1 m from the sink during use [64]. Similarly, Starlander et al. injected safranin into their sink drains to reveal visible contamination of the basin rim with water running [29]. Kotay et al. employed an experimental design, using green-fluorescent protein-expressing *E. coli* to elucidate the mechanisms of bacterial dispersal from sinks. They demonstrated, over the course of 7 days, the extension of biofilm from colonized P-traps up into the drain at an astonishing rate of 2.5 cm per day and subsequent dispersal of *E. coli* to the surrounding areas (< 76 cm) with faucet use. They also described retrograde colonization of the P-trap from a common pipe, suggesting that biofilm creep might extend beyond an individual contaminated sink and into horizontal piping [65••].

Factors Beyond the Sink

The observation that sinks in patient care areas might be colonized by retrograde biofilm creep has implicated the greater network of hospital wastewater systems as sources of sink-related infection [6, 13, 28]. Materials used in hospital piping should be taken into consideration, given that plastic has been shown to encourage biofilm formation more than copper or stainless steel [66]. Shaw et al. implicated plastic P-traps as one of a number of sink design features that might have contributed to a high incidence of MDR Gram-negative bacilli (GNB) infections in their ICU. Given the perceived extent of contamination through their water system, they adopted a "water-safe" program involving the complete removal of hand hygiene sinks from their ICU to terminate the spread of these organisms [36••]. Defective conditions in water systems, such as underuse, high temperatures, excessive pressure fluctuations, and alterations in flow can lead to trap seal depletion, and shearing, thereby magnifying the problem. In a root cause analysis, Yablon et al. identified substantial dead-end water piping as resulting in inadequate chlorine residuals and subsequent colonization of multiple sinks with *P. agglomerans*, precipitating an outbreak on a hematology-oncology ward [39]. Gormley et al., using a full-scale test rig, modeled the aerosolization of *Pseudomonas putida* through a building as the consequence of empty traps, a defect not uncommon in many buildings [67••]. More recently, Mair-Jenkins and colleagues attributed a sustained restaurant *Salmonella enteric ser. Typhimurium* outbreak to bioaerosol contamination of a kitchen as the consequence of ineffective traps as well as wastewater pooling and biofilm formation [68••]. Although outside of a hospital environment, this example demonstrates that ineffective trap seals may lead to sink contamination.

Healthcare Provider Behaviors that Contribute to Infection Transmission from Sinks

Healthcare providers can contribute to the colonization of hand hygiene sinks and the transmission of nosocomial pathogens through two means: (1) the misuse of sinks and (2) the placement of patient care materials proximal to sinks.

Misuse of Sinks

The disposal of patient wastewater into hand hygiene sinks may directly introduce pathogens into sink plumbing and onto sink fixtures, resulting in colonization. Moreover, antibiotic run off and the organic materials in patient wastewater promote resistance in and provide nutrients to existing biofilms. A recent study has demonstrated that the upward growth of biofilm from colonized traps into drains was accelerated by the addition of nutrient-rich items similar to those frequently disposed down sinks [65••]. Balm and colleagues performed a root cause analysis in their investigation of a protracted *E. meningoseptica* outbreak. They describe the disposal of patient secretions and the cleaning of re-useable patient care items in hand hygiene sinks as significant contributors [46]. Sinks subject to such misuse were found to be more likely contaminated with *E. meningoseptica* (odds ratio 4.38, 95% CI 1.68–11.39, $p = 0.004$). These behaviors persisted despite directed interventions due to nursing time constraints, as well as the distance between patient rooms and the unit dirty utility room, which was perceived as interfering with workflow [46].

Placement of Patient Care Materials Adjacent to Sinks

The use of surfaces adjacent to hand hygiene sinks for preparation of patient care items or the storage of clean supplies has also been identified during outbreak investigations as contributing to transmission. During their evaluation of a prolonged clonal MDR *P. aeruginosa* outbreak, Salm et al. identified that

Table 1 Relevant sink-related outbreaks and associated risk mitigation strategies since 2012

Reference	Organism	Sink source	Molecular method used	Setting	Intervention									
					Complete replacement of sink	Replacement of sink components	Installation self-cleaning trap	Disinfection with chlorine solution	Disinfection with other	Pressurized steam	Enhanced cleaning	Descaling	Point-of-use filters	Eliminate storage of clean items near sink
Breathnach et al. [6]	*P. aeruginosa* (VIM)	Drain; T-pip-e	PFGE; VNTR	W	√	√								√
Garvey et al. [7]	*P. aeruginosa*	Drain	PFGE	BICU					U		√	√		
Garvey et al. [8]	*P. aeruginosa*	Faucet	PFGE	HO									√	
Davis et al. [9]	*P. aeruginosa*	Drain	WGS	NICU	√	√		√						
Mayes et al. [10]	*P. aeruginosa*	Aerator	N/A	NICU		√					√			
Ambrogi et al. [11]	*P. aeruginosa* (VIM)	Drain	PFGE	ICU		√			U					
Knoester et al. [12]	*P. aeruginosa* (MDR)	Drain; aera-tor	AFLP	ICU		√		√						
Bedard et al. [13]	*P. aeruginosa*	Drain; aera-tor	qPCR	NICU		√							√	√
Aspelund et al. [14••]	*P. aeruginosa* (MBL)	Drain; pipes	PFGE	W	√				√	√				
Wendel et al. [15]	*P. aeruginosa* (GIM-1)	Drain	PFGE; MLST	ICU W		√								√
Salm et al. [16]	*P. aeruginosa* (MDR)	Drain	Rep-PCR	ICU		√								
Johansson et al. [17]	*P. aeruginosa*	Drain	MLVA; PFGE	TW										
Vos et al. [18]	*P. aeruginosa* (MDR)	Drain	N/A	U					U					√
Schneider et al. [19]	*P. aeruginosa*	Trap	RAPD-PCR; microarray	HO		√	√						√	
Brandt et al. [20]	*P. aeruginosa* (CRO)	Drain	N/A	HO			√							
Diederen et al. [21]	*P. aeruginosa* (VIM-2)	Drain	N/A	ICU		√			U					
Guleri et al. [22]	*P. aeruginosa* (MDR)	Pipes; trap	VNTR	ICU	√			√						
Kossow et al. [23••]	*P. aeruginosa* (MDR)	Trap	N/A	HO			√		U		√		√	
Leistner et al. [24]	*P. aeruginosa* (XDR)	Sink	Rep-PCR	ICU		√								
Liese et al. [25]		Trap	N/A	HO					U					

Table 1 (continued)

	P. aeruginosa (MBL)															
Clarivet et al. [26]	*Klebsiella pneumoniae* (OXA-48)	Trap; aerator	PFGE	ICU W		√		√	U							
Maltezou et al. [27]	Serratia marcescens	Drain	PFGE	NICU	√			√								√
Chapuis et al. [28]	*Enterobacter cloacae* (ESBL)	Drain	PFGE; MLST	HO				√						√		
Starlander et al. [29]	*Klebsiella pneumoniae* (ESBL)	Drain	PFGE	ICU	√											
Tofteland et al. [30]	*Klebsiella pneumoniae* (KPC)	Drain	PFGE; MLST	ICU	√			√								
Lowe et al. [31]	*Klebsiella oxytoca* (ESBL)	Drain; aerator	PFGE	ICU		√			√		√					
De Geyter et al. [32]	Polymicrobial CPE	Drain; trap	PFGE	ICU	√	√			√							
De Jong et al. [33]	*Klebsiella pneumoniae* (ESBL)	Drain	PFGE	ICU		√		√								√
Leitner et al. [34]	*Klebsiella oxytoca* (KPC)	Drain; overflow	Rep-PCR; MLST	HO	√											
Van Oers et al. [35]	Polymicrobial MDR-GNB	Sink	PFGE	ICU	√											
Shaw et al. [36••]	Polymicrobial MDR-GNB	Trap	N/A	ICU		√		√	U		√		√			
Seara et al. [37]	*Klebsiella pneumoniae* (NDM)	Trap	PFGE; MLST	IH		√		√	√	√						
Kotsanas et al. [38]	Polymicrobial CPE (IMP-4)	Drain	PFGE	ICU				√		√						
Yablon et al. [39]	Pantoea agglomerans	Sink	PFGE	HO					U							√
Wolf et al. [40]	Polymicrobial ESBL	Drain	AFLP	ICU			√									
Vergara-Lopez et al. [41]	*Klebsiella oxytoca* (IMP-8)	Trap; pipes	PFGE	ICU	√	√			√							
Umezawa et al. [42]	Acinetobacter baumanii	Faucet	Rep-PCR; MLST	ICU	√				U							
Hong et al. [43]	Acinetobacter baumanii	Faucet	MLST	PICU	√			√								
Landelle et al. [44]	Acinetobacter baumanii	Trap	N/A	ICU				√	√							
Guyot et al. [45]	Stenotrophomonas maltophilia	Sink	PFGE	ICU	√											
Balm et al. [46]		Aerator	Rep-PCR	ICU		√		√								

Table 1 (continued)

Ashraf et al. [47]	Elizabethkingia meningoseptica Mycobacterium mucogenicum	Aerator	Rep-PCR	HO		√						√
Litvinov et al. [48]	*Fusarium* species	Faucet; drains	Phylogenic	HO				√			√	

Reference	Intervention	Outcome						Follow-up	
	Eliminate disposal patient waste	Reduction cases	No further cases	Reduction colonization	No further colonization	Ongoing cases	Ongoing colonization	Surveillance	Environmental screening
Breathnach et al. [6]		√						√	
Garvey et al. [7]	√						√		√
Garvey et al. [8]	√			√					√
Davis et al. [9]			√					√	
Mayes et al. [10]								√	
Ambrogi et al. [11]			√		√			√	√
Knoester et al. [12]		√					√	√	√
Bedard et al. [13]			√				√	√	√
Aspelund et al. [14••]					√		√		√
Wendel et al. [15]			√					√	
Salm et al. [16]	√	√						√	
Johansson et al. [17]			√					√	
Vos et al. [18]		√						√	
Schneider et al. [19]		√						√	
Brandt et al. [20]							√	√	√
Diederen et al. [21]			√					√	
Guleri et al. [22]					√				√
Kossow et al. [23••]		√		√				√	√
Leistner et al. [24]	√	√						√	
Liese et al. [25]			√	√				√	
Clarivet et al. [26]			√		√			√	√
		√			√			√	√

Table 1 (continued)

Maltezou et al. [27]								
Chapuis et al. [28]		√					√	
Starlander et al. [29]	√	√	√	√			√	√
Tofteland et al. [30]			√		√		√	√
Lowe et al. [31]			√	√			√	√
De Geyter et al. [32]	√		√	√			√	√
De Jong et al. [33]	√	√			√			√
Leitner et al. [34]		√					√	√
Van Oers et al. [35]	√		√	√			√	√
Shaw et al. [36••]	√	√					√	
Seara et al. [37]			√	√			√	√
Kotsanas et al. [38]					√			√
Yablon et al. [39]		√					√	
Wolf et al. [40]			√				√	√
Vergara-Lopez et al. [41]			√	√			√	√
Umezawa et al. [42]			√				√	
Hong et al. [43]			√	√			√	√
Landelle et al. [44]	√				√		√	√
Guyot et al. [45]		√					√	
Balm et al. [46]					√			√
Ashraf et al. [47]			√				√	
Litvinov et al. [48]			√		√		√	√

U, unidentified; *N/A*, not available; *MLST*, multilocus sequence typing; *Rep-PCR*, repetitive element palindromic PCR; *PFGE*, pulse-field gel electrophoresis; *AFLP*, amplified fragment length polymorphism; *RAPD-PCR*, random amplification of palindromic DNA; *VNTR*, variable number tandem repeat; *MLVA*, multiple locus variable number tandem repeat; *WGS*, whole genome sequencing; *qPCR*, quantitative PCR; *XDR*, extensively drug-resistant; *HO*, hematology-oncology; *W*, ward; *TW*, transplant ward; *NICU*, neonatal ICU; *PICU*, pediatric ICU; *IH*, inter-hospital

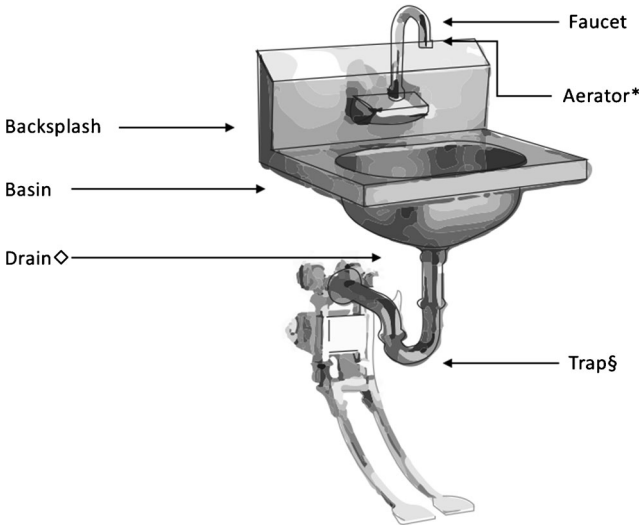

Fig. 1 Anatomy of a hospital sink and associated nomenclature. *Flow modulator; §U-bend/P-trap/S-trap/Siphon; ◊outlet/strainer; image courtesy of Bryan Graham Huck

admission to a room with a colonized sink and hemofiltration were independently associated with an elevated risk of acquiring the outbreak organism. A process audit revealed that during hemofiltration, dialysate bags were emptied in and prepared adjacent to the hand hygiene sinks. Droplet spray during sink use was thought to be contaminating the dialysate bags. After replacing sink traps, implementing single-use bags and restricting practices around sinks, the incidence of infections decreased [16]. Ashraf et al. report an outbreak of *M. mucogenicum* bloodstream infections originating from a hand hygiene sink with a colonized faucet aerator. An audit revealed that saline flushes were being prepared on the counter adjacent to the contaminated sink, using a saline bag that was hung over the sink basin, presumably leading to the contamination of the flushes [47].

Infection Control Strategies Used During Sink-Related Outbreaks

Infection control strategies are often bundled together during outbreaks, with an emphasis placed upon (1) cleaning and disinfection; (2) biofilm disruption; (3) installation of point-of-use filters; and (4) the replacement of sink plumbing and/or fixtures. It is thus difficult to determine whether a single given intervention was responsible for successfully interrupting the outbreak, or if success is predicated upon a variety of interventions.

Cleaning and Disinfection

Although many studies fail to describe what products and/or processes were employed, based upon reported outcomes, it appears that cleaning and disinfection alone is rarely effective in eliminating sink colonization. A variety of disinfectants, administered with varying frequencies, have been evaluated. Chlorine-containing solutions have been used in concentrations ranging from 250 [33] to 1000 ppm [36••, 37, 69] and administered multiple times per day [27, 43], daily [9, 28, 33, 36••, 37, 39, 55, 69, 70], and thrice weekly [12], either alone or in combination with other biocides [37]. Other chemical disinfectants have been applied with limited efficacy, including hydrogen peroxide [31], glucoprotamin [32], acetic acid [14••], amphoteric and cationic surfactants including quaternary ammonium compounds (QACs) [37, 70], sodium hydroxide [37], and polyhexamethylene biguanide hydrochloride [70]. When successful, the effect of disinfectants appears to be temporary or solely interrupts the outbreak without completely decolonizing the sinks. Garvey et al. report that "descaling and disinfection" with enhanced routine cleaning was insufficient to rid hand hygiene sinks of *P. aeruginosa* in their burn ICU, noting recolonization following 102 days [7]. Two other studies highlighted a similar pattern of clearance and subsequent re-emergence [38, 46]. Bacteria living in biofilm are able to survive in the presence of 100 to 1000 times higher concentrations of disinfectants than their planktonic counterparts. They may also limit the penetration of disinfectants, sequestering and expelling these agents [71]. The use of disinfectants in the presence of biofilm can select for resistance, and reduced susceptibility to chlorine and QACs has been reported [72]. Moreover, the presence of organic residue as well as inadequate contact time within the sink environment might contribute to the observed reduced efficacy of these agents.

Biofilm Disruption

Pressurized steam has been employed as an adjunct to chemical disinfection, capitalizing on the thermotoxic effect of high temperatures while also disrupting biofilm. Herruzo et al. achieved clearance of OXA-48 *K. pneumoniae* from their sink drains following the application of pressurized steam with a chlorine-containing solution. However, the effect of their intervention was short-lived, with 50% of sinks recolonized within 9 months [61]. A similar pattern of temporary clearance followed by re-emergence was noted in a similar study following a very brief 3 days [38].

Self-disinfecting traps have shown more promise in disrupting biofilm. These units use vibration, bundled with heat or ultraviolet radiation to remove existing biofilm, reduce microbial burden, and prevent further biofilm formation. In practice, these units may successfully reduce rates of sink-related hospital-associated infection and/or sink pathogen colonization [19, 20, 23••, 40, 72, 73••]. However, the implementation of self-disinfection traps is often bundled with other interventions, such as point-of-use filters, plumbing or fixture replacement, and chemical disinfection. When evaluated alone, Wolf et al. noted complete interruption of ESBL

transmission events as well as sustained negative environmental cultures at 20 weeks [40]. However, although Fusch et al. found a reduction in *P. aeruginosa* clinical cases and sustained lower sink aerosol contamination rates associated with the implementation of self-disinfecting traps, their sinks remained contaminated. These units were installed after replacement of the sinks alone failed to achieve a sustained reduction in aerosol contamination, raising the possibility of a deeper reservoir [72]. Although promising, self-disinfecting traps incur substantial cost, and they require further evaluation in healthcare settings.

Point-of-Use Filters

The resiliency of pathogens in established biofilms has prompted alternative risk mitigation strategies, including the installation of point-of-use filters when enhanced cleaning and disinfection have failed [13, 36••, 48, 74]. Filters are susceptible to leaking, saturation requiring frequent changes, and microbial contamination [7] and may have other disadvantages such as reduced water pressure [75]. When used, they require a rigorous maintenance program.

Replacement of Sink Plumbing and/or Fixtures

The replacement of sinks and/or sink components has been employed most successfully as an outbreak mitigation strategy [9, 11, 12, 19, 21, 22, 26, 27, 30–32, 36••, 37, 42, 43, 69]. However, replacement of individual sink components has not been universally effective, or has produced only a temporary effect, suggesting a persistent reservoir in the retained sink fittings. Despite the replacement of drainpipes, Bedard et al. noted ongoing *Pseudomonas* colonization of the drain and faucet on environmental screening [13]. Similarly, even with replacement of faucet aerators, two other studies reported ongoing microbial colonization of their sinks [12, 46].

Complete sink replacement has been reported as effective, however, like other risk mitigation strategies, may not always successfully result in decolonization. Hong et al. attempted disinfection of MDR *Acinetobacter* colonized sinks and faucets with sodium hypochlorite five times daily. In the face of ongoing environmental colonization and clinical cases, they proceeded with complete replacement of affected sinks, effectively halting the outbreak and rendering the sinks culture negative for *A. baumanii* [43]. De Geyter and colleagues eventually proceeded to replacing their sinks after targeted replacement of traps and pipes was unsuccessful, demonstrating complete clearance of CPE from their ICU sinks. However, environmental screening continued to demonstrate the presence of hydrophilic GNB, including MDR *Pseudomonas* and *Stenotrophomonas* species [32]. Similarly, Aspelund et al., who seemed to have successfully decolonized their sink drains of *P. aeruginosa* using weekly 24% acetic acid and hot water flushing, discovered that at 13 weeks following sink replacement, the drains once again cultured positive. The organism was detected in multiple wall drainpipes suggesting a deeper reservoir in the horizontal wastewater system [14••].

In a recent 6-year quasi-experimental study, the investigators explored the effect of the removal of all hand hygiene sinks from ICU patient rooms on rates of MDR-GNB. Although lacking molecular analysis, their intervention was associated with a significant decline in their baseline MDR-GNB rates [36••]. A water-free patient care environment was also explored in an ICU where hand hygiene sinks were removed from patient care areas. Following their intervention, they observed a significant reduction in GNB colonization in their patients, an effect which was most pronounced in those with long ICU-stays [76••]. Such an intervention could only be considered in a setting with high hand hygiene compliance rates using alcohol-based hand rub as well as low endemic *Clostridioides difficile* rates.

Despite attempts at disinfection, the implementation of biofilm disruption strategies, and complete replacement of affected sinks, bacterial pathogens often persist as colonizers of hospital premise plumbing and fixtures [49••, 77]. This said, a number of these interventions are successful in terminating transmission of pathogens from sinks to patients and therefore should not be dismissed as ineffective.

Investigational Strategies to Mitigate the Risk of Sink-Related Infections

A number of additional risk mitigation strategies are under investigation but have not yet been adequately tested in clinical environments. These include newer methods of biofilm disruption and prevention, and resistome modulation.

Enzymes, such as proteases, DNAses, and polysaccharide depolymerases, function by dismantling biofilm matrix; when used in conjunction with chemical disinfectants, they may enhance the biocidal effect [78]. However, enzyme efficacy is predicated upon the appropriate selection of a mixture of agents targeting the unique and heterogeneous composition of the biofilm matrix being treated. As of yet, enzymes have been used effectively only in laboratory based-experimental models and the food industry [79–81]. Similarly, bacteriophage therapy is conceptually intriguing given that phages are able to easily penetrate biofilm matrix, targeting and eradicating the constituent microorganisms [50]. Bacteriophages are species-specific, which hinders their use in multispecies biofilms like those in a hospital environment.

Quorum-sensing inhibitors or quorum quenching strategies have been suggested as effective means to prevent biofilm formation; however, this research remains very much in its infancy [82]. The use of surfaces with antimicrobial properties may prevent biofilm formation by limiting microbial burden. Metals with biocidal properties have been evaluated in healthcare settings [83]. Copper has been identified as being most effective at reducing overall bacterial burden, with studies examining sinks and sink drainage pipes [83, 84]. However, there is limited high-quality evidence to suggest the efficacy of copper alloy in reducing sink-associated infections. Moreover, the longevity of the antimicrobial effects of copper ions in the sink environment and its ability to generate resistance remains undefined. Silver and selenium nanoparticles have similarly been investigated but their efficacy in sinks has not been clearly demonstrated [85, 86].

More recently, ozone has been implemented in sink design as a biofilm prevention strategy. A recent study examined the efficacy of a stand-alone hand hygiene sink that features an ozonation cycle designed to prevent biofilm formation in the drain and trap. Following experimental inoculation of the sink with *Pseudomonas* species and *Candida auris*, complete eradication of the organisms from the trap was noted at 9 days. The study failed to demonstrate significant decolonization of either the strainer or the remainder of the sink [87]. It is unclear how this system would perform in a hospital setting where well-established multispecies biofilm might be present in the distal wastewater plumbing.

A *Bacillus*-based cleaning strategy has been used to modulate the hospital resistome by counteracting the growth of drug-resistant surface pathogens [88]. However, no research has evaluated the introduction of similar, non-pathogenic probiotic organisms in an aqueous environment. Moreover, a metagenomics study characterizing the microbiome of hospital shower hoses identified genes related to disinfectant tolerance and antimicrobial resistance amidst the largely non-pathogenic microbes identified [49••]. More research directed at understanding the complexities of biofilm communities is required before a similar risk mitigation approach is introduced.

Lessons Learned

These outbreaks have taught us that reactionary responses and mitigation strategies are woefully ineffective at eliminating sink colonization with clinically significant pathogens and, in some cases, their transmission. Furthermore, the implementation of policies restricting the use of hand hygiene sinks to handwashing is unlikely to be sufficient in isolation. With respect to hospital sink-related infections, there is a role for prevention through design. Despite the frequent identification of deficiencies in sink design as a primary driver behind sink-related infections, many of the sinks described in recent outbreak investigations do not adhere to current recommended standards. The reasons behind this are likely multifactorial and might simply reflect the age of the healthcare facilities and the relative expense of retrofitting. It may also reflect the often overlooked importance of infection prevention and control in healthcare design.

Currently, the Canadian Standards Association, in alignment with many other national facility design standards like the American Institute of Architects, stipulate that a hand hygiene sink be installed within each inpatient room and no more than 6 m distance from a given patient's bed [89, 90]. As the presence of hand hygiene sinks remains an essential component in infection prevention and control, our focus should be on optimizing sink design to prevent microbial transmission. The standards themselves have been informed by the literature and provide provisions for a design that discourages formation of biofilm, minimizes the aerosolization of water from the drain and/or trap, and dissuades high-risk behaviors. At minimum, new builds of healthcare facilities should adhere to these standards. Older facilities should take stock of their existing design and implement simple engineering controls that follow the same principles.

Beyond design-based prevention methods, infection control strategies should include "safe water practices." These should stipulate that hand hygiene sinks be dedicated to handwashing, and that the disposal of patient wastewater in sinks is prohibited. Moreover, until a safe distance is clearly defined, it is reasonable to suggest that the placement of clean supplies or clean work surfaces within 1 m of hand hygiene sinks be avoided or, alternatively, a barrier be installed to protect these vulnerable zones.

Finally, recognizing that sinks will always contain microorganisms, infection prevention and control programs should consider performing a facility-wide risk assessment to determine the hazard potential of their current sinks, based on design features. This will inform the need for enhanced surveillance and/or proactive risk mitigation to avoid sink-related outbreaks.

Conclusion

Hospital sinks provide a permissive environment for biofilm formation and microbial colonization and sink-related outbreaks are increasingly reported over time. The role of hospital sinks has become even more salient in the era of emerging antimicrobial resistance, given that CPEs and other MDR-GNBs have demonstrated an affinity for this environmental niche. Risk mitigation strategies such as cleaning and disinfection as well as sink replacement have been employed with variable success, often halting outbreaks or reducing clinical cases but failing to decolonize the sinks. It is neither reasonable nor feasible to expect sterility of hospital sinks. Emphasis should thus be placed on optimizing best practices in sink design and placement, as well as healthcare provider behaviors to prevent transmission of potentially dangerous pathogens from sinks.

Acknowledgements We would like to thank Infection Prevention and Control construction leads, Jessica Fullerton and Karl Zebarth, for the details they provided regarding the existing national facility engineering standards. We would also like to thank Ani Orchanian-Cheff for her assistance in performing the literature search and Bryan Graham Huck for his sink illustration.

Compliance with Ethical Standards

Conflict of Interest We declare that we have no conflicts of interest relevant to this manuscript. Full disclosures available upon request.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
•• Of major importance

1. Kizny Gordon AE, Mathers AJ, Cheong EYL, Gottlieb T, Kotay S, Walker AS, et al. The hospital water environment as a reservoir for carbapenem-resistant organisms causing hospital-acquired infections-a systematic review of the literature. Clin Infect Dis. 2017;64(10):1435–44.
2. Decker BK, Palmore TN. The role of water in healthcare-associated infections. Curr Opin Infect Dis. 2013;26(4):345–51.
3. Moffa M, Guo W, Li T, Cronk R, Abebe LS, Bartram J. A systematic review of nosocomial waterborne infections in neonates and mothers. Int J Hyg Environ Health. 2017;220(8):1199–206.
4. Ayliffe GAJ, Rabb JR, Collins BJ, Lowbury EJL, Newsom SWB. Pseudomonas aeruginosa in hospital sinks. Lancet. 1974;7:578–81.
5. Kohn J. A study of Ps. pyocyanea cross infection in a burns unit. In: Wallace AB, Wilkinson AW, editors. Research in burns. Edinburgh: E & S Livingstone LTD; 1966. p. 486–501.
6. Breathnach AS, Cubbon MD, Karunaharan RN, Pope CF, Planche TD. Multidrug-resistant Pseudomonas aeruginosa outbreaks in two hospitals: association with contaminated hospital waste-water systems. J Hosp Infect. 2012;82(1):19–24.
7. Garvey MI, Bradley CW, Tracey J, Oppenheim B. Continued transmission of Pseudomonas aeruginosa from a wash hand basin tap in a critical care unit. J Hosp Infect. 2016;94(1):8–12.
8. Garvey MI, Bradley CW, Holden E. Waterborne Pseudomonas aeruginosa transmission in a hematology unit? Am J Infect Control. 2017;28:28.
9. Davis RJ, Jensen SO, Van Hal S, Espedido B, Gordon A, Farhat R, et al. Whole genome sequencing in real-time investigation and management of a Pseudomonas aeruginosa outbreak on a neonatal intensive care unit. Infect Control Hosp Epidemiol. 2015;36(9):1058–64.
10. Mayes C, McCracken G, Rooney P. Minimising risk from Ps aeruginosa using tap design. Arch Dis Childhood: Fetal Neonatal Edition. 2014;1:A40.
11. Ambrogi V, Cavalie L, Mantion B, Ghiglia MJ, Cointault O, Dubois D, et al. Transmission of metallo-beta-lactamase-producing Pseudomonas aeruginosa in a nephrology-transplant intensive care unit with potential link to the environment. J Hosp Infect. 2016;92(1):27–9.
12. Knoester M, de Boer MG, Maarleveld JJ, Claas EC, Bernards AT, de Jonge E, et al. An integrated approach to control a prolonged outbreak of multidrug-resistant Pseudomonas aeruginosa in an intensive care unit. Clin Microbiol Infect. 2014;20(4):O207–15.
13. Bedard E, Laferriere C, Charron D, Lalancette C, Renaud C, Desmarais N, et al. Post-outbreak investigation of Pseudomonas aeruginosa faucet contamination by quantitative polymerase chain reaction and environmental factors affecting positivity. Infect Control Hosp Epidemiol. 2015;36(11):1337–43.
14.•• Aspelund AS, Sjostrom K, Olsson Liljequist B, Morgelin M, Melander E, Pahlman LI. Acetic acid as a decontamination method for sink drains in a nosocomial outbreak of metallo-beta-lactamase-producing Pseudomonas aeruginosa. J Hosp Infection. 2016;94(1):13–20. **This study describes a nosocomial outbreak of drug-resistant Pseudomonas aeruginosa associated with sink drains. As a mitigation strategy, simple and inexpensive weekly treatments of 24% acetic acid were employed, which resulted in negative environmental surveillance cultures and appeared to halt transmission.**
15. Wendel AF, Kolbe-Busch S, Ressina S, Schulze-Robbecke R, Kindgen-Milles D, Lorenz C, et al. Detection and termination of an extended low-frequency hospital outbreak of GIM-1-producing Pseudomonas aeruginosa ST111 in Germany. Am J Infect Control. 2015;43(6):635–9.
16. Salm F, Deja M, Gastmeier P, Kola A, Hansen S, Behnke M, et al. Prolonged outbreak of clonal MDR Pseudomonas aeruginosa on an intensive care unit: contaminated sinks and contamination of ultrafiltrate bags as possible route of transmission? Antimicrob Resistance Infect Control. 2016;5(53).
17. Johansson E, Welinder-Olsson C, Gilljam M. Genotyping of Pseudomonas aeruginosa isolates from lung transplant recipients and aquatic environment-detected in-hospital transmission. APMIS. 2014;122(2):85–91.
18. Vos MC, Voorintholt A, Gommers D, Severin J. Multi resistant VIM-positive Pseudomonas aeruginosa in the health care setting - lessons learned to combat transmission. Int J Infect Dis. 2016;1:288.
19. Schneider H, Geginat G, Hogardt M, Kramer A, Durken M, Schroten H, et al. Pseudomonas aeruginosa outbreak in a pediatric oncology care unit caused by an errant water jet into contaminated siphons. Pediatr Infect Dis J. 2012;31(6):648–50.
20. Brandt C, Vavrova A. Drains from patient's rooms sinks, showers and toilets as an environmental reservoir for carbapenem-resistant Pseudomonas aeruginosa on haematology-oncology wards. Int J Med Microbiol. 2015;1:154–5.
21. Diederen BMW, Hattink-Malipaard CJR, Vloemans AFPM, Hene I, Euser SM, Van Der Reijden WA, et al. A prolonged hospital outbreak with metallo-betalactamase producing Pseudomonas aeruginosa in a burn centre and intensive care unit linked to an environmental reservoir. Clin Microbiol Infect. 2012;3:14.
22. Guleri A, Palmer R, Jackson P, Przybylo M, Sharma R, Peel A, et al. Outbreak report of multidrug-resistant Pseudomonas aeruginosa [MDR-PA] in a cardiac intensive care unit at a Lancashire cardiac centre. Clin Microbiol Infect. 2012;3):13.
23.•• Kossow A, Kampmeier S, Willems S, Berdel WE, Groll AH, Burckhardt B, et al. Control of multidrug-resistant pseudomonas aeruginosa in allogeneic hematopoietic stem cell transplant recipients by a novel bundle including remodeling of sanitary and water supply systems. Clin Infect Dis. 2017;65(6):935–42. **This study demonstrates the efficacy of structural controls combined with a bundle of infection prevention measures at reducing MDR-Pseudomonas aeruginosa detection in both the environment and in patients. Self-disinfecting siphons were installed under every sink. These units utilize ultraviolet light, vibration, and heat in combination with an antibacterial coating to prevent biofilm formation. This was used in conjunction with a rimless**

toilet basin design, optimized shower head and drain design, daily room disinfection, improved hand hygiene practices, and active surveillance and isolation. The bundle succeeded in minimizing the risk of infection as well as environmental contamination.

24. Leistner R, Gastmeier P, Salm F. Prolonged outbreak of clonal MDR/XDR *P. aeruginosa* on an intensive care unit: ultra-filtrate bags as possible route of transmission? Int J Med Microbiol. 2016;306(8 Supplement 1):35.
25. Liese J, Grashorn S, Willmann M, Vogel W, Peter S. Control of multidrug resistant Pseudomonas aeruginosa by environmental disinfection and surveillance. Int J Med Microbiol. 2015;1:130–1.
26. Clarivet B, Grau D, Jumas-Bilak E, Jean-Pierre H, Pantel A, Parer S, et al. Persisting transmission of carbapenemase-producing Klebsiella pneumoniae due to an environmental reservoir in a university hospital, France, 2012 to 2014. Euro Surveill. 2016;21(17):28.
27. Maltezou HC, Tryfinopoulou K, Katerelos P, Ftika L, Pappa O, Tseroni M, et al. Consecutive Serratia marcescens multiclone outbreaks in a neonatal intensive care unit. Am J Infect Control. 2012;40(7):637–42.
28. Chapuis A, Amoureux L, Bador J, Gavalas A, Siebor E, Chretien ML, et al. Outbreak of extended-spectrum beta-lactamase producing Enterobacter cloacae with high MICs of quaternary ammonium compounds in a hematology ward associated with contaminated sinks. Front Microbiol. 2016;7:1070.
29. Starlander G, Melhus A. Minor outbreak of extended-spectrum beta-lactamase-producing Klebsiella pneumoniae in an intensive care unit due to a contaminated sink. J Hosp Infect. 2012;82(2):122–4.
30. Tofteland S, Naseer U, Lislevand JH, Sundsfjord A, Samuelsen O. A long-term low-frequency hospital outbreak of KPC-producing Klebsiella pneumoniae involving intergenus plasmid diffusion and a persisting environmental reservoir. PLoS One. 2013;8(3):e59015.
31. Lowe C, Willey B, O'Shaughnessy A, Lee W, Lum M, Pike K, et al. Outbreak of extended-spectrum beta-lactamase-producing Klebsiella oxytoca infections associated with contaminated handwashing sinks(1). Emerg Infect Dis. 2012;18(8):1242–7.
32. De Geyter D, Blommaert L, Verbraeken N, Sevenois M, Huyghens L, Martini H, et al. The sink as a potential source of transmission of carbapenemase-producing Enterobacteriaceae in the intensive care unit. Antimicrob. 2017;6:24.
33. De Jong E, Hopman J, Hilkens MGEC, Loeffen FLA, Van Leeuwen WB, Melchers WJ, et al. A prolonged outbreak of an extended-spectrum betalactamase producing Klebsiella pneumoniae (EKP) on an ICU due to contamination of sinks. Clin Microbiol Infect. 2012;3:14.
34. Leitner E, Zarfel G, Luxner J, Herzog K, Pekard-Amenitsch S, Hoenigl M, et al. Contaminated handwashing sinks as the source of a clonal outbreak of KPC-2-producing Klebsiella oxytoca on a hematology ward. Antimicrob Agents Chemother. 2015;59(1):714–6.
35. Van Oers J, Vos P, Harts A, Koevoets T, Van Beurden L, Beerens M, et al. Interventions to stop the transmission of highly resistant microorganisms in a Dutch intensive care unit. Int J Med Microbiol. 2015;1:153.
36.•• Shaw E, Gavalda L, Camara J, Gasull R, Gallego S, Tubau F, et al. Control of endemic multidrug-resistant Gram-negative bacteria after removal of sinks and implementing a new water-safe policy in an intensive care unit. J Hosp Infect. 2018;98:275–81. **Although lacking environmental testing, this study suggests that implementing a water-free environmental in critical care settings might reduce endemic rates of MDR-GNB infection, specifically Klebsiella pneumoniae. Their intervention included deep cleaning and disinfection of drains and valves; water filtering; replacement of siphons and tap aerators; and mandatory use of filtered water from central sinks for daily patient hygiene, or use of 2% chlorhexidine-impregnated wash cloths when water was not needed; as well as the removal of sinks from patient rooms. Their intervention yielded a RR of acquiring MDR-GNB of 0.24 (95% CI 0.17–0.34).**
37. Seara N, Oteo J, Carrillo R, Perez-Blanco V, Mingorance J, Gomez-Gil R, et al. Interhospital spread of NDM-7-producing Klebsiella pneumoniae belonging to ST437 in Spain. Int J Antimicrob Agents. 2015;46(2):169–73.
38. Kotsanas D, Wijesooriya WR, Korman TM, Gillespie EE, Wright L, Snook K, et al. "Down the drain": carbapenem-resistant bacteria in intensive care unit patients and handwashing sinks. Med J Aust. 2013;198(5):267–9.
39. Yablon BR, Dantes R, Tsai V, Lim R, Moulton-Meissner H, Arduino M, et al. Outbreak of Pantoea agglomerans bloodstream infections at an oncology clinic - Illinois, 2012-2013. Infect Control Hosp Epidemiol. 2017;38(3):314–9.
40. Wolf I, Bergervoet PWM, Sebens FW, Van den Oever HLA, Savelkoul PHM, Van der Zwet WC. The sink as a correctable source of extended-spectrum beta-lactamase contamination for patients in the intensive care unit. J Hosp Infect. 2014;87(2):126–30.
41. Vergara-Lopez S, Dominguez MC, Conejo MC, Pascual A, Rodriguez-Bano J. Wastewater drainage system as occult reservoir in a protracted clonal outbreak dur to metallo-β-lactamase-producing Klebsiella oxytoca. Clin Microbiol Infect 2013;19(11):E490–8.
42. Umezawa K, Asai S, Ohshima T, Iwashita H, Ohashi M, Sasaki M, et al. Outbreak of drug-resistant Acinetobacter baumannii ST219 caused by oral care using tap water from contaminated hand hygiene sinks as a reservoir. Am J Infect Control. 2015;43(11):1249–51.
43. Hong KB, Oh HS, Song JS, Lim JH, Kang DK, Son IS, et al. Investigation and control of an outbreak of imipenem-resistant Acinetobacter baumannii infection in a pediatric intensive care unit. Pediatr Infect Dis J. 2012;31(7):685–90.
44. Landelle C, Legrand P, Lesprit P, Cizeau F, Ducellier D, Gouot C, et al. Protracted outbreak of multidrug-resistant acinetobacter baumannii after intercontinental transfer of colonized patients. Infect Control Hosp Epidemiol. 2013;34(2):119–24.
45. Guyot A, Turton JF, Garner D. Outbreak of Stenotrophomonas maltophilia on an intensive care unit. J Hosp Infect. 2013;85(4):303–7.
46. Balm MND, Salmon S, Jureen R, Teo C, Mahdi R, Seetoh T, et al. Bad design, bad practices, bad bugs: frustrations in controlling an outbreak of Elizabethkingia meningoseptica in intensive care units. J Hosp Infect. 2013;85(2):134–40.
47. Ashraf MS, Swinker M, Augustino KL, Nobles D, Knupp C, Liles D, et al. Outbreak of Mycobacterium mucogenicum bloodstream infections among patients with sickle cell disease in an outpatient setting. Infect Control Hosp Epidemiol. 2012;33(11):1132–6.
48. Litvinov N, da Silva MT, van der Heijden IM, Graca MG, Marques de Oliveira L, Fu L, et al. An outbreak of invasive fusariosis in a children's cancer hospital. Clin Microbiol Infect. 2015;21(3):268.e1–7.
49.•• Soto-Giron MJ, Rodriguez-R LM, Luo C, Elk M, Ryu H, Hoelle J, et al. Biofilms on hospital shower hoses: characterization and implications for nosocomial infections. Appl Environ Microbiol. 2016;82(9):2872–83. **This is the first study to utilize metagenomics to characterize the composition of biofilm communities in hospital water pipelines. One significant finding was that the biofilm community harbored genes related to disinfectant tolerance in addition to genes conferring resistance to β-lactam, aminoglycosides, amphenicol and quinolone antibiotics.**

50. Donlan RM. Biofilm formation: a clinically relevant microbiological process. Clin Infect Dis. 2001;33(8):1387–92.
51. Burmolle M, et al. Interactions in multispecies biofilms: do they actually matter? Trends Microbiol. 2014;22(2):84–91.
52. Wendel AF, Ressina S, Kolbe-Busch S, Pfeffer K, MacKenzie CR. Species diversity of environmental GIM-1-producing bacteria collected during a long-term outbreak. Appl Environ Microbiol. 2016;82(12):3605–10.
53. Weingarten RA, Johnson RC, Conlan S, Ramsburg AM, Dekker JP, Lau AF, et al. Genomic analysis of hospital plumbing reveals diverse reservoir of bacterial plasmids conferring carbapenem resistance. mBio. 2018;9(1):06.
54. Rahimzadeh G, Gill P, Rezai MS. Characterization and lytic activity of methicillin-resistant Staphylococcus aureus (MRSA) phages isolated from NICU. Australas Med J. 2016;9(6):169–75.
55. Roux D, Aubier B, Cochard H, Quentin R, van der Mee-Marquet N. Centre HAIPGotRdHd. Contaminated sinks in intensive care units: an underestimated source of extended-spectrum beta-lactamase-producing Enterobacteriaceae in the patient environment. J Hosp Infect. 2013;85(2):106–11.
56. Vickery K, Deva A, Jacombs A, Allan J, Valente P, Gosbell IB. Presence of biofilm containing viable multiresistant organisms despite terminal cleaning on clinical surfaces in an intensive care unit. J Hosp Infect. 2012;80(1):52–5.
57. Lalancette C, Charron D, Laferriere C, Dolce P, Deziel E, Prevost M, et al. Hospital drains as reservoirs of Pseudomonas aeruginosa: multiple-locus variable-number of tandem repeats analysis genotypes recovered from faucets. Sink Surfaces Patients Pathogens. 2017;6(3):09.
58. Winder EM, Bonheyo GT. DNA persistence in a sink drain environment. PLoS One. 2015;10(7):e0134798.
59. Howie RI, John M, Clark J. Assessing the level of bioburden and rate of contamination of high contact surfaces in private, semi-private and ward patient rooms. Am J Infect Control. 2014;1:S37.
60. Ghadakpour M, Bester E, Liss SN, Gardam M, Droppo I, Hota S, et al. Integration and proliferation of Pseudomonas aeruginosa PA01 in multispecies biofilms. Microb Ecol. 2014;68(1):121–31.
61. Herruzo R, Ruiz G, Vizcaino MJ, Rivas L, Perez-Blanco V, Sanchez M. Microbial competition in environmental nosocomial reservoirs and diffusion capacity of OXA48-Klebsiella pneumoniae: potential impact on patients and possible control methods. J. 2017;58(1):E34–41.
62. Zhou Z, Hu B, Gao X, Bao R, Chen M, Li H. Sources of sporadic Pseudomonas aeruginosa colonizations/infections in surgical ICUs: association with contaminated sink trap. J Infect Chemother. 2016;22(7):450–5.
63. Jencson AL, Cadnum JL, Piedrahita C, Donskey CJ. Hospital sinks are a potential nosocomial source of Candida infections. Clin Infect Dis. 2017;65(11):1954–5.
64. Hota S, Hirji Z, Stockton K, Lemieux C, Dedier H, Wolfaardt G, 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.
65.•• Kotay S, Chai W, Guilford W, Barry K, Mathers AJ. Spread from the sink to the patient: in situ study using green fluorescent protein (GFP)-expressing *Escherichia coli* to model bacterial dispersion from hand-washing sink-trap reservoirs. Appl Environ Microbiol. 2017;83(8):–15. **This study employs an experimental design to demonstrate the mechanism of biofilm growth from pipe to sink strainer and subsequent dispersal of bacteria from colonized hospital sink drains to patients. The study also demonstrates that bacterial contamination of sinks can occur via horizontal connections in wastewater plumbing. This work helps to inform risk mitigation strategies.**
66. Rogers J, Dowsett AB, Dennis PJ, Lee JV, Keevil CW. Influence of plumbing materials on biofilm formation and growth of *Legionella pneumophila* in potable water systems. App Environ Microbiol. 1994;60(6):1842–51.
67.•• Gormley M, Aspray TJ, Kelly DA, Rodriguez-Gil C. Pathogen cross-transmission via building sanitary plumbing systems in a full scale pilot test-rig. PLoS One. 2017;12(2):e0171556. **Pseudomonas putida is utilized in an experimental model to demonstrate how empty traps can permit aerosolization of pathogens which can be carried via plumbing system airflows between different floors of a building. Empty traps are prevalent in many buildings including hospitals, highlighting the importance of this mechanism of transmission.**
68.•• Mair-Jenkins J, Borges-Stewart R, Harbour C, Cox-Rogers J, Dallman T, Ashton P, et al. Investigation using whole genome sequencing of a prolonged restaurant outbreak of Salmonella Typhimurium linked to the building drainage system, England, February 2015 to March 2016. Euro Surveill. 2017;22(49). **This in vivo study investigating a Salmonella enterica serovar Typhimurium outbreak similarly demonstrates the role ineffective traps play in allowing the transmission of contaminated aerosols into the environment, further suggesting the importance of this sink feature in risk mitigation.**
69. Kong MY, Lai CKC, Lee SY, Tsang NC. A local experience sharing: hand wash basin as a potential source of carbapenemase-producing enterobacteriaceae transmission in hospital environments. Antimicrobial Resistance and Infection Control Conference: 8th International Congress of the Asia Pacific Society of Infection Control, APSIC. 2017;6(Supplement 2).
70. Varin A, Valot B, Cholley P, Morel C, Thouverez M, Hocquet D, et al. High prevalence and moderate diversity of Pseudomonas aeruginosa in the U-bends of high-risk units in hospital. Int J Hyg Environ Health. 2017;220(5):880–5.
71. Fux CA, Costerton JW, Stewart PS, Stoodley P. Survival strategies of infectious biofilms. Trends Microbiol. 2005;13:34–40.
72. Fusch C, Pogorzelski D, Main C, Meyer CL, El Helou S, Mertz D. Self-disinfecting sink drains reduce the Pseudomonas aeruginosa bioburden in a neonatal intensive care unit. Acta Paediatr. 2015;104(8):e344–9.
73.•• Mathers AJ, Vegesana K, German Mesner I, Barry KE, Pannone A, Baumann J, et al. Intensive care unit wastewater interventions to prevent transmission of multi-species *Klebsiella pneumoniae* carbapenemase (KPC) producing organisms. Clin Infect Dis. 2018;02:02. **This study bundled self-disinfecting siphons with the installation of covers on hoppers. The intervention was associated with a decrease in incidence of MDR-*Klebsiella pneumoniae*. Although the effect of the sink trap devices alone could not be determined, the proportion of colonized sink drains significantly decreased following the intervention (12/15 [80%] vs 40/840 [5%]; $p = 0.001$).**
74. Garvey MI, Bradley CW, Wilkinson MAC, Bradley CR, Holden E. Engineering waterborne Pseudomonas aeruginosa out of a critical care unit. Int J Hyg Environ Health. 2017;229:1014–9.
75. Baranovsky S, Jumas-Bilak E, Lotthe A, Marchandin H, Parer S, Hicheri Y, et al. Tracking the spread routes of opportunistic premise plumbing pathogens in a haematology unit with water points-of-use protected by antimicrobial filters. J Hosp Infect. 2018;98(1):53–9.
76.•• Hopman J, Tostmann A, Wertheim H, Bos M, Kolwijck E, Akkermans R, et al. Reduced rate of intensive care unit acquired gram-negative bacilli after removal of sinks and introduction of 'water-free' patient care. Antimicrob Resist Infect Control. 2017;6:59. **This study similarly demonstrates the effectiveness of removing sinks from patient rooms in the intensive care unit setting, and implementing a "water-free" patient care strategy. The strategy resulted in a significant and sustained reduction in patient colonization with GNB.**

77. Liu R, Yu Z, Guo H, Liu M, Zhang H, Yang M. Pyrosequencing analysis of eukaryotic and bacterial communities in faucet biofilms. Sci Total Environ. 2012;435–436:124–31.
78. Bridier A, Sanchew-Vizuete P, Guilbaud M, Piard JC, Naitali M, et al. Biofilm-associated persistence of food-borne pathogens. Food Microbiol. 2015;45:167–78.
79. Wang H, Wang H, Xing T, Wu N, Xu X, et al. Removal of Salmonella biofilm formed under meat processing environment by surfactant in combination with bio-enzymes. LWT Food Sci Technol. 2016;66:298–304.
80. Brown HL, Hanman K, Reuter M, Betts RP, Van Vliet AHM. Camplylobacter jejuni biofilms contain extracellular DNA and are sensitive to DNase I treatment. Front Microbiol. 2015;6:699.
81. Kim SH, Park C, Lee EJ, Bang WS, Kim YJ, Kim JS. Biofilm formation of campylobacter strains isolated from raw chickens and its reduction with DNase I treatment. Food Control. 2017;71: 94–100.
82. Coughlan LM, Cotter PD, Hill C, Alvarez-Ordonez A. New weapons to fight old enemies: novel strategies for the (bio) control of bacterial biofilms in the food industry. Front Microbiol. 2016;7: 1641.
83. Muller MP, MacDougall C, Lim M, Ontario Agency for Health P, Promotion Public Health O, Provincial Infectious Diseases Advisory Committee on Infection P, et al. Antimicrobial surfaces to prevent healthcare-associated infections: a systematic review. J Hosp Infect. 2016;92(1):7–13.
84. Soothill JS. Carbapenemase-bearing Klebsiella spp. in sink drains: investigation into the potential advantage of copper pipes. J Hosp Infect. 2016;93(2):152–4.
85. Wang Q, Larese-Casanova P, Webster TJ. Inhibition of various gram-positive and gram-negative bacteria growth on selenium nanoparticle coated paper towels. Int J Nanomedicine. 2015;10: 2885–94.
86. Orti-Lucas RM, Munoz-Miguel J. Effectiveness of surface coatings containing silver ions in bacterial decontamination in a recovery unit. Antimicrob. 2017;6:61.
87. Livingstone S, Cadnum JL, Jencson AL, Gestrich S, Donskey CJ. That sinking feeling: eradicating Pseudomonas and Candida auris from a sink drain system using ozonated water. Paper presented at The Society for Healthcare Epidemiology of America; Portland; 2018 Apr 18–20.
88. Caselli E, D'Accolti M, Vandini A, Lanzoni L, Camerada MT, et al. Impact of a probiotic-based cleaning intervention on the microbiota ecosystem of hospital surfaces: focus on the resistome remodulation. PLoS One. 2016;11(2):e10148857.
89. CSA Group. CSA Z8000-11 Canadian health care facilities – planning, design and construction. Mississauga; CSA Group. 2011;
90. American Institute of Architects Academy of Architecture for Health and The Facility Guidelines Institute. Guidelines for design and construction of hospital and health care facilities. Washington: The American Institute of Architects; 2014.

View publication stats