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REVIEW ARTICLE

Are Sink Drainage Systems a Reservoir for Hospital-Acquired Gammaproteobacteria Colonization and Infection? A Systematic Review

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Increasing rates of antimicrobial-resistant organisms have focused attention on sink drainage systems as reservoirs for hospital-acquired Gammaproteobacteria colonization and infection. We aimed to assess the quality of evidence for transmission from this reservoir. We searched 8 databases and identified 52 studies implicating sink drainage systems in acute care hospitals as a reservoir for Gammaproteobacterial colonization/infection. We used a causality tool to summarize the quality of evidence. Included studies provided evidence of co-occurrence of contaminated sink drainage systems and colonization/infection, temporal sequencing compatible with sink drainage reservoirs, some steps in potential causal pathways, and relatedness between bacteria from sink drainage systems and patients. Some studies provided convincing evidence of reduced risk of organism acquisition following interventions. No single study provided convincing evidence across all causality domains, and the attributable fraction of infections related to sink drainage systems remains unknown. These results may help to guide conduct and reporting in future studies.

Keywords. Gammaproteobacteria; gram-negative bacteria; sink drains; waste water; infection control.

Gammaproteobacteria are members of a bacterial class that includes the *Enterobacteriaceae*, *Pseudomonas* species, and other nonfermenting gram-negative bacilli [1]. These organisms thrive in moist or wet environments and are an important cause of health care–associated infections [2–5].

Guidelines for health care facility design mandate that sinks be placed in acute care facilities to promote hand hygiene and protect patients from hospital-acquired infections [6–8].

It has become accepted that *Legionella* species in potable water and other Gammaproteobacteria contaminating sink faucets in hospitals may pose risk to patients [9–12]. There are also decades of studies that document an association between Gammaproteobacteria from sink drainage systems (including from sink drains, traps, drainpipes, and/or air samples above sink drainage outflow) and hospital-acquired colonization or infection of patients or health care workers [13–21]. The first suggestions to heat sink traps or modify sink construction to reduce splashing were made more than 40 years ago [22]. Despite this, it is only the increasing transmission of carbapenem-resistant organisms in hospitals that has refocused attention on the possible role of sink drainage systems as a reservoir [20, 21, 23, 24]. Some hospitals have become convinced of the importance of sinks as a reservoir for hospital-acquired Gammaproteobacterial infections and have removed them entirely from their intensive care units [25]. However, much of the evidence suggesting that sink drainage systems are reservoirs is circumstantial, and directionality of contamination of sinks and colonization or infection of patients is challenging to establish [25–28].

RATIONALE

The quality of evidence for causality of sink drainage systems as reservoirs for hospital-acquired Gammaproteobacterial colonization or infection has not been objectively evaluated.

OBJECTIVES

The objective of this study was to systematically assess the quality of evidence that Gammaproteobacteria in sink drainage systems represent a reservoir for colonization or infection of patients or health care workers in acute care hospitals.

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METHODS

Protocol and Registration

This systematic review was prospectively registered with PROSPERO (CRD42015027811). The original protocol can be accessed at https://www.crd.york.ac.uk/prospero/display_record.php?RecordID=27811.

Eligibility Criteria

Studies of any design were included if the authors presented data that led them to conclude that sink drainage systems in patient care areas of acute care hospitals were a reservoir for hospital-acquired Gammaproteobacteria colonization or infection of patients or health care workers. Studies that reported only on fungi, mycobacteria, or *Legionella* species were excluded, because the ecology of these organisms differs from that of other Gammaproteobacteria [27, 29]. Studies that focused only on water sources other than sink drainage systems were also excluded.

Search Strategies

A professional librarian (E.U.) ran searches in MEDLINE and EMBASE (OvidSP), CINAHL (EBSCOHost), Cochrane (Wiley), Aqualine (ProQuest), Scopus (ScienceDirect), BIOSIS, and Web of Science (Thomson Reuters) on October 6, 2016. Reference lists were reviewed to identify relevant citations. We used both subject headings and text words to search for articles on (bacteria or infection terms) AND (hospital departments) AND (sink or plumbing or hand hygiene terms). The results were not limited by language or publication year. Search strategies are listed in the Supplementary Data.

Study Selection

One reviewer assessed eligibility by title, and 2 independent reviewers screened English, French, German, and Spanish abstracts and full-text articles for eligibility (see the Supplementary Data for screening questions). Abstracts and full-text articles in other languages were read by 1 reviewer and discussed with another investigator (C.V.). A third reviewer was used if consensus was not achieved among the first 2 reviewers. Reviewers were not blinded to author, institution, or journal.

Data Collection Process

Data were extracted independently from included articles by 2 authors. For studies published between 2007 and 2016 where it was unclear which sink structure was cultured or implicated as causal in patient or health care worker colonization or infection, authors were contacted to try to obtain this information; studies were included if it was confirmed that criteria were met.

Data Items

Data were collected on study and population characteristics, sink design, sampling of sinks and other potential reservoirs, microbiologic methods, patient or health care worker colonization or infection, sink drainage system interventions and co-interventions, and potential causal pathways for transmission of Gammaproteobacteria from sink drainage systems to patients and/or health care workers.

Quality Assessment

During data extraction, 2 reviewers applied the Modified CADDIS Tool for Causality Assessment of Sink Drains as a Reservoir for Hospital-Acquired Gammaproteobacterial Infection or Colonization (Supplementary Table 1) to assess the quality of evidence for causality of sink drainage systems as reservoirs. This tool was modified from a US Environmental Protection Agency online causality application using a modified Delphi process with experts in infection control and hospital epidemiology [30, 31].

The Modified CADDIS tool includes 6 domains of evidence to evaluate the likelihood of a causal relationship: Spatial/Temporal Co-occurrence, Temporal Sequence, Stressor–Response Relationship, Causal Pathway, Evidence of Exposure and Biological Specificity, and Manipulation of Exposure. Within each domain, the scoring system contains phrasing that describes how evidence/findings influence the likelihood of causality of sink drainage systems in hospital-acquired infection or colonization (organism acquisition), with corresponding scores rated as (+++)/convincingly supports, (++)/strongly supports, (+)/somewhat supports, (0)/neither supports nor weakens, (-)/somewhat weakens, (--)/strongly weakens, (---)/convincingly weakens, or (R)/refutes causality.

Synthesis of Results

Characteristics of studies were entered in duplicate, cleaned, and analyzed in Microsoft Excel using descriptive statistics. The quality of evidence to determine causality was summarized using the Modified CADDIS tool scores and narrative description. Investigators' hypotheses regarding potential causal pathways and the success of attempts to manipulate exposure to sink drainage systems and reduce organism acquisition were summarized.

RESULTS

We screened 39511 records and identified 52 studies that met the inclusion criteria (Figure 1) [13–23, 32–72]. The characteristics of these studies are summarized in Table 1. All studies were conducted in acute care hospitals; 35/48 (73%) [13, 15, 17–23, 33, 36–39, 41, 43, 45, 48, 49, 51, 52, 54–58, 60–62, 64, 65, 69–72] were identified as tertiary care or university/teaching hospitals. Over half of studies (35/52, 67%) included intensive care units (ICUs) [14–19, 21–23, 32–34, 36, 38, 39, 41, 43, 45, 48, 49, 51, 52, 54, 56–58, 60, 63, 64, 67–72].

The most common organisms involved were *Pseudomonas aeruginosa* (*pyocyanea*; 31/52, 60%) [14–18, 22, 34–36, 38,

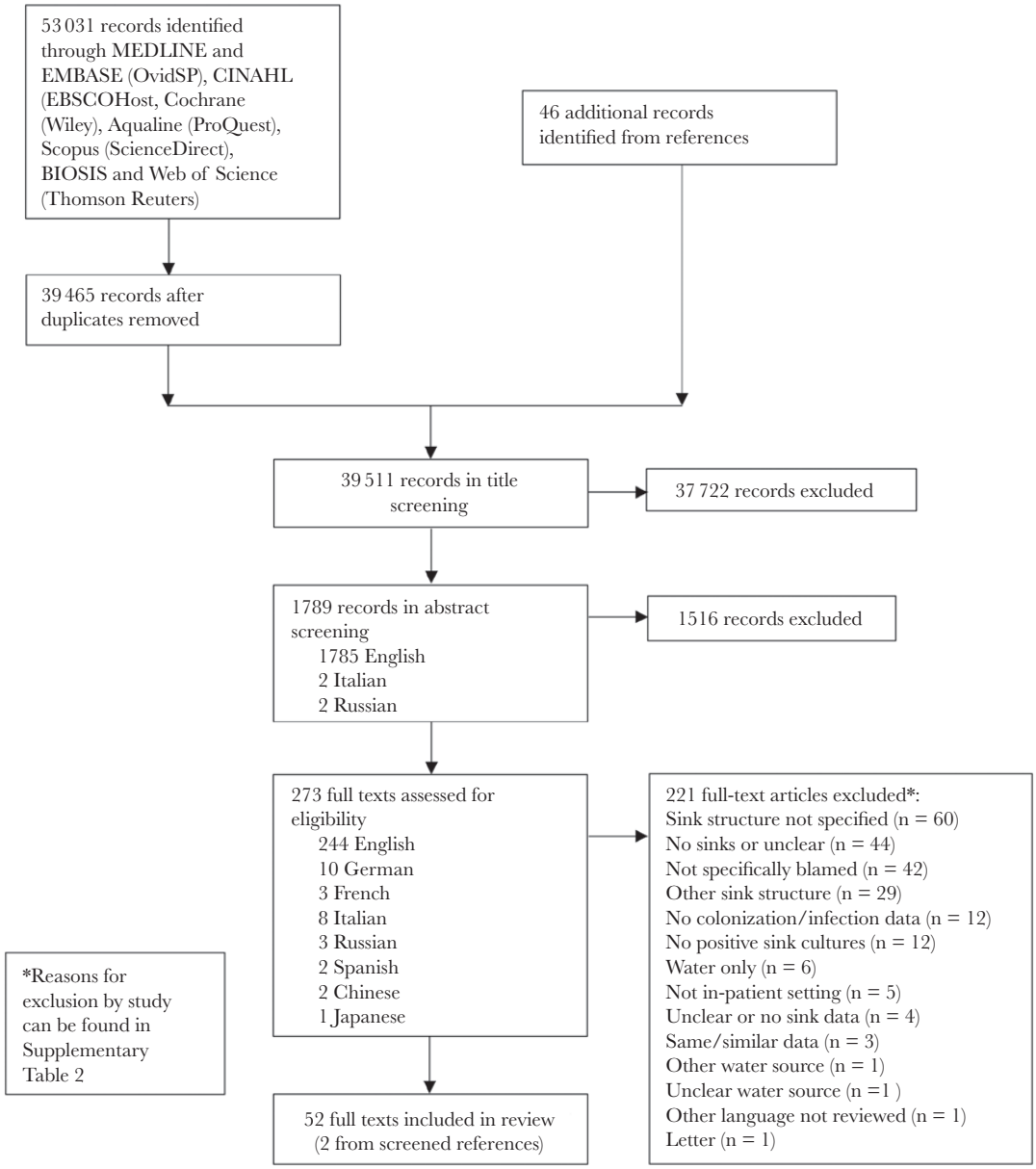

Figure 1. Flow diagram results of literature search.

41–50, 53, 54, 57, 58, 60, 61, 65, 66, 69, 70, 72] and *Klebsiella pneumoniae* (7/52, 13%) [21, 23, 39, 55, 62, 64, 67]. Reduced susceptibility or resistance of implicated bacteria to at least 1 carbapenem antibiotic was reported in 21/52 (40%) studies [18, 20, 21, 23, 33, 34, 39, 48, 51–53, 55, 58, 60, 62, 65, 67–70, 72], and carbapenemase production was reported in 12/52 (23%) [20, 21, 23, 33, 39, 55, 62, 65, 67–70].

The number of patients colonized or infected with Gammaproteobacteria that were also cultured from sink drainage systems was not consistently reported. Patients underwent some screening for carriage, most commonly from rectal (19/52 studies, 37%) [15, 19, 21, 23, 36, 38, 39, 43–45, 50, 52, 55, 56, 62, 68, 70–72], throat (14/52, 27%) [13, 15, 17, 22, 36, 37, 43, 44, 50, 52, 56, 68, 70, 71], feces (8/52, 15%) [17, 35, 37, 40, 42, 46, 67, 70], nasal (8/52, 15%) [13, 38, 39, 41, 42, 44, 50, 72], wound (7/52, 13%) [14, 36, 43, 47, 55, 60, 65], or sputum (7/52, 13%) [17, 22, 37, 44, 49, 65, 71] specimens. In the 16 studies that reported numerator and denominator data for screening, 411/5932 (6.9%) [14, 15, 17, 22, 23, 35, 37, 38, 40, 42–44, 55, 67, 68, 72] patients were colonized with the same Gammaproteobacterial species found in sink drainage systems, with a median (range) of 13.5% (0.01%–68.8%). However, not all cases were attributed to sink drainage systems. The mean or median time from admission or exposure to colonization or infection was reported by only 8 studies and ranged from 8 to 53 days (median, 17 days) [14, 23, 34, 50, 53, 55, 62, 68].

Table 1. Characteristics of Included Studies (n = 52)

Author, Country	Study Dates	Hospital Unit(s)	Population Characteristics	Bacteria	Typing Methods[a]
Cabrera [13], USA	1959–1960	Ward	Neonatal	*F. meningosepticum*	Biotyping, serotyping
Kohn [50], England	1963–1964	Ward	Medical-surgical, burn	*P. pyocyanea*	Serotyping, pyocin typing
Thomas [66], England	1970	OR	Surgical	*P. aeruginosa, other* gram-negative bacteria	Serotyping, phage typing
Teres [22], USA	1970–1972	ICU	Medical-surgical	*P. aeruginosa*	Pyocin typing
Edmonds [14], USA	1971-1971	ICU	Medical-surgical, burn	*P. aeruginosa*	Serotyping, pyocin typing, phage typing
Riser [59], England	1971–1976	Ward	Medical-surgical	*K. aerogenes*	Serotyping
Breitfellner [35], Austria	1973–1974	Ward	Neonatal, obstetric	*E. coli, P. aeruginosa*	Antibiotic susceptibility, serotyping
Brown [36], USA	NS (pre-1977)	ICU	Neonatal	*P. aeruginosa*	Pyocin typing
Cooke [40], England	NS (pre-1979)	Ward	Medical-surgical, neonatal	*K. aerogenes*	Serotyping
Gunther [47], Germany	NS (pre-1980)	Ward	Medical, pediatric	*P. aeruginosa*	Pyocin typing
Levin [15], USA	NS (pre-1984)	ICU	Medical-surgical	*P. aeruginosa*	Serotyping
Döring [43], Germany	1988–1989	ICU	Medical-surgical	*P. aeruginosa*	Exotoxin A probing
Döring [42], Germany	1989–1989	Ward	Medical, pediatric, immunocompromised	*P. aeruginosa*	Exotoxin A probing
Döring [44], Germany	1992–1992	Ward	Medical	*P. aeruginosa, B. cepacia*	PFGE
Kerr [16], Ireland	1993-1993	ICU	Medical	*P. aeruginosa*	RAPD PCR
Bert [34], France	1995–1997	ICU	Surgical	*P. aeruginosa*	PFGE
Berthelot [17], France	1995–1996	ICU	Medical-surgical, mechanically ventilated	*P. aeruginosa*	PFGE
Pitten [58], Germany	1997–1998	ICU, ward	Medical-surgical	*P. aeruginosa*	PFGE
Gillespie [46], Scotland	1997–1998	Ward	Medical, immunocompromised	*P. aeruginosa*	PFGE
Lowe [19], Canada	1997–2011	ICU, ward	Medical-surgical	*K. oxytoca*	PFGE
Orrett [57], Trinadad	1998–1998	ICU	Surgical, neonatal	*P. aeruginosa*	Antibiotic susceptibility
Ahmad [32], Pakistan	1998–2002	ICU, ward	Medical, pediatric	*B. cepacia*	Antibiotic susceptibility
Sissoko [63], Germany	2002–2004	ICU	Medical	Gram-negative bacteria	Typing not reported
Hota [18], Canada	2004–2006	ICU	Medical-surgical, immunocompromised	*P. aeruginosa*	PFGE
La Forgia [51], USA	2004–2008	ICU	Medical-surgical	*A. baumanii*	Restriction endonuclease of genomic DNA
Johansson [49], Sweden	2004–2009	ICU, ward	Medical-surgical, immunocompromised	*P. aeruginosa*	PFGE, MLVA
Schneider [61], Germany	2004–2010	Ward	Medical, pediatric, immunocompromised	*P. aeruginosa*	RAPD PCR
Cholley [38], France	2006–2006	ICU	Medical-surgical	*P. aeruginosa*	PFGE
Longtin [54], Switzerland	2006–2008	ICU	Medical-surgical	*P. aeruginosa*	PFGE
Inglis [48], Australia	2006–2008	ICU, ward	Medical-surgical	*P. aeruginosa*	PFGE
Tofteland [67], Norway	2007–2010	ICU	Medical-surgical	*K. pneumoniae*	PFGE
Maltezou [56], Greece	2007–2010	ICU	Neonatal	*S. marscecens*	PFGE
Salimi [60], Iran	2008–2008	ICU	Medical-surgical, burn	*P. aeruginosa*	PFGE
Landelle [52], France	2008–2009	ICU, ward	Medical-surgical	*A. baumanii*	PFGE
Stjarne Aspelund [65], Sweden	2008–2015	Ward	Medical	*P. aeruginosa*	PFGE
Ling [53], Singapore	2009–2009	Ward	Medical, immunocompromised	*P. aeruginosa*	PFGE
Vergara Lopez [68], Spain	2009–2011	ICU	Medical-surgical	*K. oxytoca*	PFGE
Kotsanas [23], Australia	2009–2012	ICU	Medical-surgical	*S. marscecens, K. pneumoniae, E.cloacae, E. coli*	PFGE
Willmann [70], Germany	2009–2013	ICU, ward	Medical, immunocompromised	*P. aeruginosa*	WGS
Starlander [64], Sweden	2010	ICU	Surgical	*K. pneumoniae*	PFGE
Zhou [72], China	2011–2011	ICU	Surgical	*P. aeruginosa*	PFGE
Amoureux [33], France	2011–2012	ICU, ward	Medical-surgical, pediatric	*A. xylosoxidans*	PFGE
Wolf [71], the Netherlands	2011–2012	ICU	NS	ESBL-producing gram-negative bacilli	AFLP
Lowe [55], Canada	2011–2012	Ward	Medical-surgical	*K. pneumoniae*	PFGE
Chapuis [37], France	2011–2013	Ward	Medical, immunocompromised	*E. cloacae*	PFGE
Leitner [20], Austria	2011–2013	Ward	Medical	*K. oxytoca*	MLST, rep-PCR
Fusch [45], Canada	2011–2013	ICU	Neonatal	*P. aeruginosa*	Typing not reported

Table 1. Continued

Author, Country	Study Dates	Hospital Unit(s)	Population Characteristics	Bacteria	Typing Methods[a]
Wendel [69], Germany	2011–2014	ICU, ward	Medical-surgical	*P. aeruginosa*	PFGE
Clarivet [39], France	2012–2014	ICU	Medical-surgical	*K. pneumoniae*	PFGE, rep-PCR
Pantel [21], France	2012–2014	ICU, OR	NS	*K. pneumoniae*	MLST, rep-PCR
Seara [62], Spain	2013–2014	NS	Medical-surgical	*K. pneumoniae*	PFGE
Davis [41], Australia	2013–2014	ICU	Neonatal	*P. aeruginosa*	WGS

Abbreviations: ESBL, extended-spectrum beta lactamase; ICU, intensive care unit; NS, not specified; OR, operating room.

[a]Typing method examples and their relative discriminatory power based on consensus evaluation by microbiologists and infection control practitioners: Adequately discriminatory: PFGE, pulsed field gel electrophoresis; RAPD PCR, random amplified polymorphic DNA polymerase chain reaction; RFLP, restriction fragment length polymorphism; WGS, whole-genome sequencing. Less discriminatory: AFLP, amplified fragment length polymorphism; ERIC PCR, enterobacterial repetitive intergenic consensus polymerase chain reaction; MLEE, multilocus enzyme electrophoresis; MLST, multilocus sequence typing; MLVA, multiple locus variable number tandem repeat analysis; rep-PCR, repetitive element palindromic polymerase chain reaction; VNTR, variable number tandem repeat. Inadequate: phage typing, biotyping, antibiotic susceptibility pattern, pyocin typing.

Health care workers were screened for carriage of Gammaproteobacteria from hands in 11 (21%) [13, 14, 22, 36, 40, 42, 44, 48, 57, 58, 72], throat in 5 (10%) [13, 50, 56, 66, 68], nose in 4 (8%) [13, 42, 50, 66], rectum in 2 (4%) [56, 68], feces in 2 (4%) [35, 42], hair in 1 (2%) [14], and nasopharynx in 1 (2%) [14] of 52 studies. Where the anatomical site was specified, cultures were positive for the same Gammaproteobacteria species found in sink drainage systems from hands in 5/11 (45%) [22, 36, 40, 42, 44], stool in 1/2 (50%) [42], and 1 nasopharyngeal swab in a single study [14]. However, methods of sampling and timing relative to handwashing varied, and only 1 study used adequately discriminatory typing (pulsed field gel electrophoresis) to determine that the strain of *P. aeruginosa* cultured from health care worker hands matched that sampled from sink drainage systems [44]. These cultures were obtained after hand disinfection and then washing in sinks known to be contaminated [44].

Most studies implicated more than 1 sink in transmission, though a single sink was deemed the culprit in 12 of 52 (23%) studies [13, 21, 32, 39, 41, 51, 55, 57, 60, 62, 64, 68]. Bacterial cultures were performed of drains in 37 (71%) [14–16, 18–21, 23, 32, 33, 36, 37, 40–44, 46–49, 51, 53–56, 58–61, 64–69, 71], traps in 21 (40%) [13, 17, 18, 22, 34, 35, 38, 39, 47, 48, 50–52, 57, 61–63, 66, 68, 70, 72], drainpipes in 2 (4%) [65, 68], and adjacent air in 4 (8%) [22, 43, 45, 63] of 52 studies. In 32 (62%) [14–20, 23, 32, 35, 36, 39–41, 46–48, 50–56, 58, 59, 65, 66, 68–70, 72] studies, at least 1 other sink structure was sampled, including (with number and percentage of studies with at least 1 positive culture) the adjacent rim or counter (3/3, 100%) [19, 59, 69], splash-backs (1/1, 100%) [41], sink basins (6/7, 86%) [14, 19, 35, 50, 51, 68, 70], overflows (3/4, 75%) [17, 20, 47, 68], faucets (6/20, 30%) [14, 18, 23, 32, 39, 46–48, 50–56, 58, 65, 66, 68, 72], and/or faucet aerators (2/6, 33%) [15, 19, 39, 41, 48, 65]. Water was sampled from the faucet in 19/52 (37%) studies and was culture positive in 26% (5/19) [17–19, 34, 37–39, 45, 49, 52, 54, 56–58, 60, 65, 68, 69, 72].

Environmental sampling of potential sources other than sinks was reported in 45/52 (87%) studies [13–23, 32–37, 39–44, 46, 47, 49–62, 66–70, 72], and 1 or more of these were found to be positive in 26 (58%) studies [13–16, 18, 20, 22, 33–35, 37, 40, 42, 46, 47, 49, 50, 52, 58, 59, 62, 66, 68–70, 72]. The following nonsink sites were sampled in at least 5 studies, and their percent positivity for implicated bacteria was as follows: showers (9/12, 75%) [18, 20, 33, 37, 42, 44, 46, 49, 53, 58, 62, 70], air or settle plates not described in relation to sinks (3/6, 50%) [13, 14, 36, 40, 50, 52], toilets (3/9, 33%) [22, 33, 35, 37, 42–44, 46, 70], trolleys/carts, table/countertops, or desks (3/10, 30%) [14, 17, 18, 21, 32, 37, 52, 55, 69, 72], disinfectants or their dispensers (2/8, 25%) [19, 32, 35, 54, 56, 60, 61, 66], ventilatory apparatus (3/11, 27%) [16, 18, 21, 22, 51, 52, 54, 55, 57, 58, 68], other respiratory equipment (3/15, 20%) [14–19, 32, 36, 40, 52, 54, 56–58, 68], beds or bedrails (2/10, 20%) [14, 15, 17, 21, 22, 32, 37, 52, 55, 72], and intravenous monitors or poles (0/5, 0%) [15, 18, 52, 55, 72].

There were limited data on sink design features considered to influence transmission of Gammaproteobacteria from sink drainage systems to patients. Six studies reported that water outflow from faucets was directed onto the drain [18, 20, 23, 37, 61, 65], 2 studies reported shallow sink basins [18, 23], and 6 studies described the use of aerators in faucets [15, 19, 39, 41, 48, 65].

Causality Assessment

Spatial/Temporal Co-occurrence

All 52 studies provided some evidence in support of a spatial or temporal co-occurrence of contaminated sink drainage systems and organism acquisition (Table 2). Thirty-six (69%) studies reported on specific sink locations [13, 16–21, 33–39, 42–47, 51, 53, 55, 58, 59, 61, 63–72], which in 32 (89%) included sinks located directly within the patient room [16–21, 33–39, 42–47, 51, 53, 55, 58, 63–65, 67–72].

Temporal Sequence

Twenty (38%) studies provided some evidence that patient or health care worker exposure to contaminated sink drainage systems was present before they were found to be colonized or infected with the implicated organism. Most studies did not collect or report adequate data to establish a temporal sequence.

Table 2. Summary Table of Scores for 2 Raters Applying the Modified CADDIS to 52 Articles Included in a Systematic Review

Study	Spatial/Temporal Co-occurrence	Temporal Sequence	Stressor–Response Relationship	Causal Pathway[a]	Evidence of Exposure and Biological Specificity[b]	Manipulation of Exposure
Hota [18]	+	0	0	+	++	+++
Bert [34]	+	0	0	+	++	+++
Schneider [61]	+	0	0	0	++	+++
Stjarne Aspelund [65]	+	0	0	0	++	+++
Tofteland [67]	+	0	0	0	++	+++
Vergara Lopez [68]	+	0	0	0	++	+++
Wolf [71]	+	++	0	0	0	+++
Sissoko [63]	+	0	0	+	0	+++
Cabrera [13]	+	++	0	+	0	+++
Chapuis [37]	+	++	0	0	++	+
Clarivet [39]	+	++	0	0	++	+
Longtin [54]	+	++	0	0	++	+
Lowe [19]	+	++	0	0	++	+
Lowe [55]	+	++	0	0	++	+
Davis [41]	+	0	0	+	++	+
Landelle [52]	+	0	0	+	++	+
Starlander [64]	+	0	0	+	++	+
Wendel [69]	+	0	0	+	++	+
Gillespie [46]	+	0	0	0	++	+
Johansson [49]	+	0	0	0	++	+
La Forgia [51]	+	0	0	0	++	+
Ling [53]	+	0	0	0	++	+
Maltezou [56]	+	0	0	0	++	+
Pitten [58]	+	0	0	0	++	+
Seara [62]	+	0	0	0	++	+
Willmann [70]	+	0	0	0	++	+
Leitner [20]	+	0	0	0	+	+
Pantel [21]	+	0	0	0	+	+
Riser [59]	+	0	0	+	0	+
Thomas [66]	+	0	0	+	0	+
Ahmad [32]	+	0	0	0	0	+
Breitfellner [35]	+	0	0	0	0	+
Fusch [45]	+	0	0	+	0	+
Orrett [57]	+	0	0	0	0	+
Döring [44]	+	++	0	+	++	0
Berthelot [17]	+	++	0	0	++	+
Zhou [72]	+	++	0	0	++	0
Cholley [38]	+	++	0	0	++	0
Amoureux [33]	+	0	0	0	++	0
Inglis [48]	+	0	0	0	++	0
Kerr [16]	+	0	0	0	++	0
Kotsanas [23]	+	0	0	0	++	0
Salimi [60]	+	0	0	0	++	0
Döring [42]	+	++	0	+	+	0
Brown [36]	+	++	0	+	0	0
Cooke [40]	+	++	0	+	0	0
Döring [43]	+	++	0	+	+	0
Teres [22]	+	++	0	+	0	0
Edmonds [14]	+	++	0	0	0	0
Gunther [47]	+	++	0	0	0	0
Levin [15]	+	++	0	0	0	0
Kohn [50]	+	++	0	0	0	0

*The original Causal Analysis/Diagnosis Decision Information System (CADDIS) Summary Table of Scores can be found at https://www.epa.gov/caddis-vol1/summary-tables-scores [31].

+++ convincingly supports, ++ strongly supports, + somewhat supports, 0 neither supports nor weakens, - somewhat weakens, -- strongly weakens, --- convincingly weakens, or (R)/ refutes causality.

[a]Causal pathway example: bacterial transmission from sinks splashing onto health care workers' hands, and then to patients. See Döring et al. [44].

[b]Evidence of Exposure and Biological Specificity is based on adequacy of typing methods employed (Supplementary Table 1).

Stressor–Response Relationship

No study presented clear evidence that the likelihood of organism acquisition differs in relation to duration of exposure or degree of sink drainage system contamination, with the exception of those where differences appeared to be due to a direct effect of manipulation of exposure (see below).

Causal Pathway

Hypothetical causal pathways were mentioned in 36 of 52 (69%) studies, most commonly involving splash-back of drain contents to health care worker hands, fomites, or surroundings (Table 3). Seventeen (47%) of these studies demonstrated 1 or more potential steps in corresponding causal pathway(s) [13, 18, 22, 34, 36, 40–45, 52, 59, 63, 64, 66, 69]. These included positive cultures from health care worker hands (n = 5) [36, 40, 42–44], positive water cultures from contaminated sinks used for patient care (n = 1) [34], or findings that suggest splash, aerosolization, or leak of bacteria from contaminated sink drainage systems onto taps, surroundings, or fomites (n = 12) [13, 18, 22, 40, 41, 45, 52, 59, 63, 64, 66, 69].

Evidence of Exposure and Biological Specificity

Thirty-two (62%) studies utilized adequately discriminatory typing methods (based on consensus evaluation by microbiologists and infection control practitioners) (Supplementary Table 1) to identify that the organisms cultured from sink drainage systems and colonized or infected patients or health care workers were the same or closely related [16–19, 23, 33, 34, 37–39, 41, 44, 46, 48, 49, 51–56, 58, 60–62, 64, 65, 67–70, 72]. Among these, 2 used whole-genome sequencing [41, 70]. None of the 10 studies published before 1990 used adequately discriminatory typing methods.

There were 319 colonized or infected patients with isolates reported as matching those recovered from sink drainage systems in the 35 (67%) studies that provided these data. In the 13 studies that used adequately discriminatory typing methods to determine the proportion of colonization or infection related to sink drainage systems, a median (range) of 75% (7%–100%) of colonized or infected patients had isolates matched to bacteria from contaminated sink drainage.

Manipulation of Exposure

Attempts to manipulate exposure to sink drainage systems were made in 40 (77%) studies (Table 4; Supplementary Table 3), most commonly by cleaning or replacing some or all parts of the sink or its drainage system [13, 17–23, 32, 34, 35, 37, 39, 41, 42, 45, 46, 48–59, 61–71]. In 9 (23%) of these studies, organism acquisition appeared to be affected by the manipulation, and there were no reported co-interventions ("+++" under Manipulation of Exposure in Table 2) [13, 18, 34, 61, 63, 65, 67, 68, 71]. In 26 (65%) of these studies, 1 or more sink drainage system interventions were reported to be successful, but there were co-interventions rendering assessment of effect challenging, or the evidence was not convincing because too few data were presented ("+" under Manipulation of Exposure in Table 2) [17, 19–21, 32, 35, 37, 39, 41, 45, 46, 49, 51–59, 62, 64, 66, 69, 70]. In 1 study, all trialed interventions were unsuccessful [23], and in another the effect of sink drainage system manipulation was unclear [48]. In 3 other studies the authors reported some success in reducing drain contamination, but data to support a reduction in organism acquisition were not provided [22, 42, 50]. In addition to frequent co-interventions, short or unclear duration of follow-up in many studies limited interpretation of findings.

DISCUSSION

Summary of Evidence

When applying the Modified CADDIS tool to the 52 included studies, for each domain other than the stressor response domain there was >1 study with evidence supporting causality of sink drainage systems in hospital-acquired Gammaproteobacterial colonization or infection. However, even after excluding the stressor response domain, no single study provided supporting evidence for all remaining Modified CADDIS domains. As each

Table 3. Hypothetical Causal Pathways Between Sink Drainage and Patient or Health Care Worker Colonization or Infection

Hypothetical Causal Pathways	Number of Studies That Mention This Causal Pathway (References)	Number of Studies That Demonstrate 1 or More Potential Steps in This Causal Pathway (References)
Direct patient use of sinks with contaminated drains	6 [20, 33, 37, 42, 53, 61]	
Water from sinks with contaminated drains used in relation to patient care activity	1 [34]	1 [34]
Contamination of health care personnel hands or gowns during use of sinks with contaminated drains, and subsequent transmission to patients	24 [13, 15, 17, 18, 22, 23, 32, 33, 35–37, 39, 40, 42–44, 50, 51, 63, 66, 67, 71, 72]	5 [36, 40, 42–44]
Splash or aerosolization of bacteria from contaminated sink drains into taps	1 [66]	1 [66]
Splash, aerosolization, or leak of bacteria from contaminated sink drains onto surroundings/fomites	19 [13, 14, 17, 18, 23, 32, 37, 40, 41, 45, 51, 52, 59, 62–64, 67–69, 71, 72]	11 [13, 18, 22, 40, 41, 45, 52, 59, 63, 64, 69]
Splash or aerosolization of bacteria from contaminated sink drains directly onto patients	3 [18, 35, 39]	

Table 4. Manipulations[a] of Exposure to Sink Drainage Systems in Effort to Reduce Patient or Health Care Worker Colonization or Infection

Intervention Type	No. of Studies	No. (%) of Studies With Elimination or Reduction in Organism Acquisition	Proportion (%) of Successful Studies Reporting Co-intervention(s)	Durability[b] of Success in Studies Reporting Elimination or Reduction in Organism Acquisition, Duration, No. (%) of Studies	No. (%) of Studies With Reduction in Sink Drain Contamination but no Data on Impact on Organism Acquisition Before Other Interventions	No. (%) of Studies Reporting Unsuccessful Attempts to Reduce Sink Drain Contamination or Organism Acquisition
Additional sink cleaning[c]	17	8 (47)[d]	6 (75)	>6 mo: 4 (50%) <6 mo: 1 (13%) Uncertain: 3 (38%)	3 (18)[e]	5 (29)[f]
Repair or replacement of all or part of sink drainage	10	8 (80)	7 (88)	<6 mo: 1 (13%) >6 mo: 2 (25%) Uncertain: 5 (63%)		2 (20)
Installation of heater–vibrator trap devices	6	4 (67)	1 (25)	>6 mo: 3 (75%) Uncertain: 1 (25%)	2 (33)	
Replacement, cleaning, or disinfection of taps thought contaminated from drain	1	1 (100)	1 (100)	>6 mo: 1 (100%)		
Sink removal, relocation, or closure of unit not otherwise detailed	3	3 (100)	2 (67)	>6 mo: 1 (33%) Uncertain: 2 (67%)		
Additional sink cleaning, repair, or replacement of all or part of sink drainage	8	8 (100)	5 (63)	<6 mo: 1 (13%) >6 mo: 3 38%) Uncertain: 4 (50%)		
Installation of heater–vibrator trap devices, taps redirected away from drain	1	1 (100)	0	>6 mo: 1 (100%)		
Replacement of traps, distance and barrier between drain *and* workspace or patient, taps redirected away from drain, water pressure reduced	1	1 (100)	0	>6 mo: 1 (100%)		
Exchange and disinfection of traps, use of sink for patient care or placement of materials adjacent to sink forbidden, use of reusable hair wash basins stopped	1	1 (100)	1 (100)	>6 mo: 1 (100%)		

[a]Additional details and references provided in Supplementary Table 3.

[b]Durability of successful reduction in organism acquisition by patients or health care workers determined based on reported periods with significantly reduced or no new cases of colonization or infection attributed to sinks, or time to manuscript submission/receipt in those who had reported sustained effect.

[c]Unclear effect of manipulation of exposure for 1 study with additional cleaning (disinfectant poured down drains).

[d]Additional sink drain cleaning that was reported to result in some success without structural sink intervention included the use of chlorine or bleach products (n = 4), formalin (n = 1), or acetic acid (n = 1) with or without dismantling and physical cleaning, sink disinfection or cleaning not further specified (n = 2).

[e]Additional sink drain cleaning that without structural sink intervention was reported to result in some reduction in drain contamination but not patient acquisition: accelerated H2O2 gel poured into drains and weekly cleaning with a sodium hypochlorite solution (n = 1), use of phenolics or paracetic acid (n = 1), use of bleach or chlorine and sodium hydroxide and alkyldimethyl amine oxide with steam cleaning (n = 1).

[f]Additional sink drain cleaning that without structural sink intervention was reported unsuccessful included the use of bleach products with mechanical brushing (n = 1) or with phenolic flushing (n = 1), or with steam cleaning (n = 1), or the use of hydrogen peroxide (n = 1), or sink cleaning not further specified (n = 1).

domain is necessary but not sufficient to assess causality, this limits the overall causality assessment.

It is not surprising that there was evidence of a spatial and/or temporal co-occurrence of contaminated sink drainage systems and acquisition of Gammaproteobacteria in studies that implicate sink drainage systems, although many studies did not report the specific sink location. While some studies have documented splash distance from sinks, the risk to patients in relation to sink proximity is not known and presumably depends on the causal pathway responsible for transmission. Among the other domains, changes in rates of organism acquisition with manipulation of sink drainage systems, typing to ensure organisms in sink drainage and patients are the same or closely related, and demonstration of a temporal sequence of exposure to contaminated sink drainage systems before organism acquisition would seem likely to provide the most support for causality when present. However, no single study provided evidence that strongly or convincingly supported causality of sink drainage systems across all of these important domains.

Evidence for a temporal sequence of sink drainage system contamination before organism acquisition by a patient or health care worker may be lacking in many studies because most institutions do not perform routine screening for bacteria in sink drainage systems, and once they are a suspected reservoir, investigators are unwilling to allow persistent risk of transmission from sink drainage systems to patients while better evidence is collected. Stressor response data are difficult to collect because measures of increased exposure, which might for instance include concentration in drains and the number of drains that are contaminated, have not been established or validated. Measurement of causal pathways is fraught with challenges in measurement (eg, lack of standard methods to culture sink surroundings or fomites), an inherent temporal component, and the fact that more than 1 causal pathway for

transmission of Gammaproteobacteria between contaminated sink drainage systems and patients or health care workers may exist.

While more studies found support for the Evidence of Exposure and Biological Specificity domain than for other domains, early studies were limited by the lack of specificity in typing systems. When adequate typing methods are employed, directionality is difficult to determine, and few studies were able to convincingly document acquisition of organisms from sink drainage as compared with other sources. While some studies provided data on the proportion of cases of acquired Gammaproteobacteria that were matched, or in some cases attributed, to sink drainage system contamination, the methods used to ascertain this were inconsistent.

Evidence for successful interventions on contaminated sink drainage systems was often confounded by co-intervention and unclear or short duration of follow-up measurement of clinical cultures. Few studies reported a sustained decrease in organism acquisition attributed to additional cleaning of sink drainage systems, but heterogeneity of agents and regimens limits interpretation.

Installation of self-cleaning traps that use a combination of heat and vibration appeared to have a beneficial and durable effect, but the limited number and size of studies suggest that further data are needed before broad implementation of this intervention [45, 61, 63, 70, 71]. However, a recent 2-armed nonrandomized intervention trial of traps with similar properties compared with a new polyvinylchloride trap showed a significant reduction in sink drain and patient colonization with multidrug-resistant (MDR) *Pseudomonas aeruginosa* as compared with baseline rates [73].

It was noted during the review process that there was variation in sink structures sampled and in how authors determined the most likely source of acquired Gammaproteobacteria. Bert et al. blamed tap water from contaminated faucets for transmission of Gammaproteobacteria to patients, but they also implicated bacterial persistence in sink drains/traps, and an outbreak was only controlled after sinks were replaced and sink trap disinfection commenced [34]. An article we excluded identified faucet aerators as a common source reservoir in an outbreak of MDR *P. aeruginosa* [74]. However, in a recently published follow-up study, investigators identified the sink drainage system as the reservoir and reported a significant reduction in patient colonization with installation of self-disinfecting traps [73].

The data reported in studies included in this review were heterogenous, and we were unable to perform a meta-analysis. The strengths of this review lie in the broad search of 8 databases without date or language restrictions and in the use of a standardized causality tool. While there are no absolute criteria that can be used to determine causality, the Modified CADDIS is based on the fundamental principles of causal analysis, and use of the tool provided a more objective and transparent means to assess the quality of evidence for acquisition of Gammaproteobacteria from sink drainage systems in acute care settings.

Selection of only studies that implicate sink drainage systems in organism acquisition may be interpreted to introduce bias toward favoring these as causal. However, bacteria are acquired from multiple sources in hospitals. Inclusion of studies in which sink drainage systems were not blamed would be unlikely to change the assessment of the quality of published evidence for causality of sink drainage systems.

In summary, the studies included in this review provide evidence that sink drainage systems are a reservoir for hospital-acquired Gammaproteobacterial colonization or infection. However, these data do not assist in quantifying the attributable fraction of hospital-acquired Gammaproteobacterial infections acquired from sink drainage systems and are of limited value in understanding the causal pathways for infection or optimal mitigation strategies. Ideally more studies will be performed outside of outbreak settings and include prospective screening of patients and the environment with consideration of potential causal pathways, documentation of patient proximity and duration of stay relative to sinks with contaminated drainage systems, attempts to standardize methods and quantify environmental cultures, use of whole-genome sequencing, and before–after or cluster randomization studies of interventions.

Context

The literature regarding waste water drainage systems as reservoirs for Gammaproteobacterial infection is expanding rapidly. However, both individual publications and reviews have usually focused on outbreaks and carbapenem-resistant organisms and have taken at face value author conclusions about whether sinks and/or drains were the relevant reservoir for the outbreak [75–79]. In these reviews, the findings regarding organism and species distribution and difficulties in sustaining drain decolonization with different interventions were similar to ours. However, as might be expected, outbreaks of carbapenem-resistant organisms were found more commonly in ICUs and immunocompromised patients, while our review suggests that acquisition of infection from sink drains may be more widespread in in-patients [75, 77]. Other studies have either failed to detect or did not report health care worker hand colonization, which was identified in 10% of our reports, supporting the possibility of this route of sink-to-patient transmission in some cases. Our review was also able to describe presumed modes of sink-to-patient transmission, as well as time from patient admission to colonization/infection.

More recently, the introduction of whole-genome sequencing and a systematic approach to culturing and sequencing in waste water drainage systems resulted in the ability to define sink-to-patient directionality in a single infection due to *E. coli*, though even here the spatial association was not clear (the patient was housed in the same ward where the sink drain isolate was

recovered) [80]. More experimental work is also being done to investigate how bacteria from sink drainage systems may make their way into the environment where they pose risk to patients, as well as sink features that may be associated with this dispersal [81–85]. Further work is needed to define the burden of infection associated with endemic hospital-acquired infections from sink drain reservoirs.

Supplementary Data

Supplementary materials are available at *Open Forum Infectious Diseases* online. Consisting of data provided by the authors to benefit the reader, the posted materials are not copyedited and are the sole responsibility of the authors, so questions or comments should be addressed to the corresponding author.

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