Journal of Hospital Infection 102 (2019) 82–88

Available online at www.sciencedirect.com

Journal of Hospital Infection

journal homepage: www.elsevier.com/locate/jhin

Acetic acid as a decontamination method for ICU sink drains colonized by carbapenemase-producing Enterobacteriaceae and its effect on CPE infections

D. Smolders [a,*], B. Hendriks [b], P. Rogiers [b], M. Mul [a], B. Gordts [a]

[a] Department of Microbiology, Ziekenhuis Netwerk Antwerpen, Antwerp, Belgium
[b] Department of Intensive Care, Ziekenhuis Netwerk Antwerpen, Antwerp, Belgium

ARTICLE INFO

Article history:
Received 12 October 2018
Accepted 14 December 2018
Available online 21 December 2018

Keywords:
Acetic acid
Decontamination
Carbapenemase-producing Enterobacteriaceae
ICU

SUMMARY

Background: Carbapenemase-producing Enterobacteriaceae (CPE) are emerging pathogens representing a major concern for public health. In Belgium, the OXA-48 carbapenemase resistance gene is identified most frequently. Sink drains in intensive care units (ICUs) are known to be colonized by Gram-negative bacilli. A correlation between environmental contamination and CPE infections in ICUs has been established. A long-term CPE epidemic in a local ICU proved difficult to control.

Methods and results: A variety of CPE strains, all carrying the OXA-48 resistance gene, were isolated from almost all sinks in patient rooms in the ICU. Decontamination of the sinks with 250 mL 25% acetic acid three times weekly was implemented. Sink drain colonization was followed up for six months thereafter. Both the number of CPE-colonized sinks and the number of patients colonized or infected with CPE decreased drastically, to the extent that the epidemic was considered to be eradicated. In-vitro growth of all isolates was inhibited by a concentration of acetic acid equal to or smaller than that used for decontamination. Epidemiological analysis demonstrated a positive and significant relationship between contaminated sinks and CPE acquisition of patients admitted to ICU rooms, indicating the importance of contaminated sinks as the environmental reservoir of the epidemic.

Conclusion: Decontamination of sink drains with acetic acid is a valuable alternative to other methods, such as heated sinks and water-free care, especially when other options are not feasible in the short term. Acetic acid is cheap, widely available, effective and manageable from a safety and technical point of view.

Background

Carbapenemase-producing Enterobacteriaceae (CPE) have become a major source of concern for public health [1–5]. Before the 1990s, they were almost non-existent [6]. The first emerging CPE was a *Klebsiella pneumoniae* carbapenemase (KPC)-producing *K. pneumoniae*. In recent years, different types of CPE have been emerging globally. For example, in Europe, the epidemiological situation of CPE worsened from 2013 to 2015 [7]. In 2013, six of 38 countries reported inter-regional spread or an endemic situation of CPE. In 2015, this number more than doubled to 13 of 38 countries. The most

* Corresponding author. Address: Lindendreef 1, 2020 Antwerpen, Belgium. Tel.: +32 476 47 95 31.
E-mail address: smolders.david@gmail.com (D. Smolders).

https://doi.org/10.1016/j.jhin.2018.12.009
0195-6701/

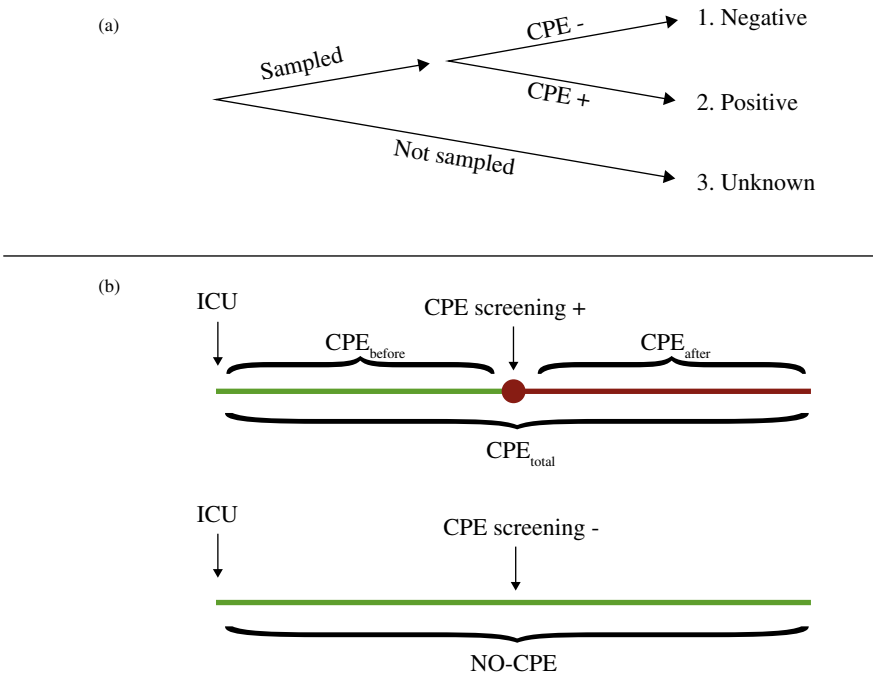

Figure 1. (a) Categorization of the different rooms based on cultures of the sink siphons. (b) Categorization of patients who screened positive (upper) and patients who screened negative (lower). CPE, carbapenemase-producing Enterobacteriaceae; CPE_{before}, patient-days in the intensive care unit (ICU) before the patient is CPE positive through screening or diagnostic sampling; CPE_{after}, patient-days following the day on which culture for CPE is positive; CPE_{total}, all patient-days of a patient who becomes CPE positive in the ICU, subdivided into CPE_{before} and CPE_{after}; NO-CPE, patients who do not become CPE positive during their ICU stay.

prevalent types of CPE in Europe are KPC, New Delhi metallo-beta-lactamase, carbapenem-hydrolysing oxacillinase (OXA-48) and Verona integron-encoded metallo-beta-lactamase. In Belgium, OXA-48 is the most prevalent carbapenemase among Enterobacteriaceae, followed by KPC. It is important to recognize, identify and act on this phenomenon as therapeutic options for patients infected with CPE are limited. A study from 2016 reported that mortality was three times higher in patients infected with carbapenem-resistant *K. pneumoniae* compared with carbapenem-sensitive *K. pneumoniae* [8]. CPE are part of a broader spectrum of multi-drug-resistant Gram-negative bacteria (MDR GNB).

Sinks have been associated with in-hospital infections since the 1970s [4]. Recent publications highlighted the role of in-patient room sinks in MDR GNB infections and outbreaks of CPE and extended-spectrum B-lactamase in hospital wards [9–15]. This was also experienced in the intensive care unit (ICU) at the study hospital. More infections with CPE have been emerging over the last few years. The contamination of bedside sinks was associated with an important risk of acquiring CPE. Based on these findings, the total burden of CPE infections in the ICU, the rate of colonization, and the potential use of acetic acid for sink decolonizion were investigated.

The main aim of this study was to investigate infection and colonization control of patients and the environment. Although it is well established that CPE infections are associated with higher mortality [8], the impact of the infection control intervention on the clinical outcome of patients was not evaluated as this was outside the scope of the study.

Methods

Setting

Ziekenhuis Netwerk Antwerpen is a hospital group in Antwerp, Belgium with 2200 beds over nine campuses. There are eight ICUs in total. The ICU studied in the present investigation is a general 14-bed adult ICU at a 400-bed hospital. All rooms have a sink. The distance between the sink and the patient's bed is often less than 1 m. There are also rooms with a front room, mainly used for patients in isolation.

In the study ICU, patient fluids are occasionally disposed of in the sinks. When washing patients, the skin is rubbed gently with a wash cloth, wetted with running tap water. This wash cloth is rinsed whenever dry or visibly stained. Tube feeding, aspirated stomach fluids, water for washing the patient, body hair and dialysate are all disposed of in the drain. Ideally, body fluids should be disposed elsewhere to avoid colonizing the sink with the patient's flora.

Recurrent epidemics of CPE infection occurred in the ICU between 2011 and 2017. In this study, 'epidemic' was defined as more than 1.5 new cases per 1000 patient-days per month.

Case definition

A case of CPE was defined as a patient admitted to the ICU between 1st January 2017 and 24th January 2018 for whom the laboratory identified a CPE in a screening or a diagnostic specimen.

Surveillance and isolation methods

All patients are screened for CPE colonization at admission and at discharge from the ICU. CPE colonization is investigated on rectal swabs, inoculated on a selective CPE culture medium (bioMérieux, Marcy l'Etoile, France). Bronchial aspirates and urine samples are sent to the microbiology laboratory for culture, identification and susceptibility testing of pathogens twice per week for all patients on invasive mechanical ventilation or with a urinary bladder catheter, respectively. Moreover, additional diagnostic samples are processed whenever clinical infection is suspected. Carbapenemase production in Enterobacteriaceae recovered from screening or diagnostic samples is excluded or confirmed using the Phoenix expert system (Becton Dickinson, Franklin Lakes, NJ, USA), and growth is tested using the selective CPE culture medium (bioMérieux). Screening for the OXA-48 resistance gene is tested using a rapid diagnostic test (Coris Bioconcept, Gembloers, Belgium), and susceptibility testing for meropenem is performed using an e-test (bioMérieux). The KPC resistance gene is screened for by polymerase chain reaction (LightCycler 480, Roche, Switzerland) of all OXA-48-negative isolates presenting with a minimum inhibitory concentration for meropenem >1 μg/mL. CPE-colonized or -infected patients are registered and nursed with barrier precautions.

Epidemiological investigation

The infection control electronic surveillance database tracks all patients admitted to the hospital, including all CPE patients. During the study, the room number and length of stay (LOS) were monitored continuously for every patient admitted to the ICU. A query was run for all new and existing CPE patients at the ICU between 1st January 2017 and 24th January 2018. The room they resided in and LOS were noted. All patients who were admitted to the ICU during the study period were included. Patients who had tested positive on screening for CPE before the study were excluded from the patient data for new CPE cases.

The relationship between space and time between CPE screenings from patients and the sinks was investigated in order to establish the role of environmental contamination in CPE colonization of patients and an eventual causal relationship.

Each room and patient were categorized as shown in Figure 1. Each room was categorized into one of three groups according to its CPE status: negative (i.e. environmental sample of siphon taken and not growing CPE); positive (i.e. environmental sample of siphon taken and growing CPE); or 3, unknown (i.e. no sample was taken). Periods in between sample days were categorized according to the previously established status (i.e. all days after a positive screening were considered positive until a negative screening result, and vice versa).

Patient-days were also divided into different groups: CPE_{total} (all patient-days of a patient who becomes CPE positive in the ICU, subdivided into CPE_{before} and CPE_{after}); CPE_{before} (patient-days in the ICU before the patient is CPE positive through screening or diagnostic sampling); CPE_{after} (patient-days following the day on which culture for CPE is positive); and NO-CPE (patients who do not become CPE positive during their ICU stay).

Environmental screening for CPE

The siphons of the sinks in all rooms of the ICU were sampled before decontamination. Samples were taken with cotton tips (Medical Industry, Tonbridge, UK). Sampling consisted of dipping the cotton tips in the water of the siphon. Samples were inoculated on a chromID Carba Smart selective, chromogenic bi-plate (bioMérieux) and incubated for 24 h at 37°C. Matrix-assisted laser desorption and ionization by time of flight mass spectrometry (Bruker Daltonics, Billerica, MA, USA) was used to identify all coloured colonies on the plates.

Sink decontamination protocol

A sink decontamination protocol (SDP) was developed in order to eradicate CPE from the sink environment of one ward of the ICU. Every Monday, Wednesday and Friday, 250 mL of 25% acetic acid was poured into the sink with a contact time of 30 min. Five of the 12 rooms were sampled one to three times per week just before decontamination took place. A two-week trial was set up on 16th October 2017 using 12% acetic acid instead of 25% in order to minimize the risk of toxicity for personnel. Some sinks quickly recontaminated, so the 25% concentration was re-introduced from 30th October 2017. The 25% concentration was chosen in analogy with a 2016 study from a Swedish hospital [16].

Sensitivity testing of Gram-negative isolates to acetic acid

The first cultured bacteria, before decontamination, were stored in TSA tubes. From 20th December 2017, all cultured CPE from the sinks were stored in TSA tubes. Antibiotic and acetic acid resistance were tested for all stored species. Antibiotic resistance testing was performed using a Phoenix (Becton Dickinson) automated antimicrobial susceptibility testing instrument. Acetic acid resistance was evaluated based on a method described by Halstaed *et al.* [17]. A dilution series of 125 μL, ranging from 8% to 0.25%, and a control with tap water were used. Acetic acid was diluted with tap water. Dilution broths of the bacteria were made of 0.5 McFarland. Twenty microlitres of the broth was inoculated in the diluted acetic acid. Actual acetic acid concentrations after 20 μL inoculation were: 6.9%, 3.4%, 1.7%, 0.86%, 0.43%, 0.22% and 0%. After 5 and 30 min, 1 μL was inoculated on a blood agar plate. The plate was then incubated for a period of 14–72 h at 37°C with 5% CO_2.

Statistics

Chi-squared test was used to compare rates and proportions for significance.

Results

CPE endemic and epidemiology

There were three active outbreaks of CPE between August 2011 and March 2017. CPE was detected in 103 patients hospitalized or previously admitted to the ICU. The mean age of these patients was 73 years (range 23–92 years) and 73.8% (N=76 patients) were male. The average LOS was 15 days

Table I
All patient-days in the intensive care unit (ICU)

	Room	NO-CPE	CPE_{after}	CPE_{before}
Total follow-up	N	1043	10	0
	P	1322	120	65
	U	1802	88	38
Pre-intervention	N	233	3	0
	P	1241	120	65
	U	663	14	10
Post-intervention	N	810	7	0
	P	81	0	0
	U	1139	74	28

CPE, carbapenemase-producing Enterobacteriaceae; CPE_{before}, patient-days in the ICU before the patient is CPE positive through screening or diagnostic sampling; CPE_{after}, patient-days following the day on which culture for CPE is positive; NO-CPE, patients who do not become CPE positive during their ICU stay.
The total follow-up during ICU stay for the three patient groups was sorted by room siphon status: N (CPE negative), P (CPE positive) and U (unknown CPE status).

(range 1–55 days). Fifty-nine percent of these isolates were *Klebsiella pneumoniae*, 19% were *Enterobacter cloacae*, 9% were *Escherichia coli*, 7% were *Klebsiella oxytoca*, 5% were *Citrobacter freundii* and 1% were *Enterobacter aerogenes*. All of the isolates presented with the OXA-48 resistance gene.

Before the SDP was introduced, 14 new cases of CPE were detected in 2017 (2.15 per month). Following introduction of the SDP, the average rate of CPE acquisition decreased to 0.67 per month (four cases).

Table I shows all patient-days in the ICU. Only five of the 14 ICU rooms were sampled consistently throughout the study period, thus the CPE-environmental status of a proportion of rooms remains unknown.

The difference between the proportion of unknown siphons between the CPE_{after} group (CPE-positive patient-days) and the NO-CPE and CPE_{before} groups is not significant (P=0.4271). Therefore, further analysis only considers room-days with known status of the siphons. All 65 (100%) patients in the CPE_{before} group stayed in a CPE-positive room. CPE_{after} patients resided in CPE-positive rooms for 120 of 130 (92.3%) days. NO-CPE patients were admitted to CPE-positive rooms for 1322 of 2365 (55.9%) days. Patients who acquired CPE were nursed in a CPE-positive room at the time of acquisition significantly more often than patients who did not acquire CPE (P<0.0001). When looking at CPE-positive rooms, 120 of 1507 (7.96%) admittance days were CPE-positive patients. In CPE-negative rooms, only 10 of 1053 (0.95%) admittance days were CPE-positive patients, which was less than in the CPE-positive rooms (P<0.0001).

Before introduction of the SDP, 13 patients acquired CPE in a total of 2349 patient-days in the ICU. After the SDP was introduced, only four new cases of CPE were detected from a total of 2139 admittance days (P=0.0015). The average LOS in the ICU of CPE-negative patients was six days. The average LOS of CPE-positive patients was 14 days (4.5 days before acquiring CPE and 9.5 days after acquiring CPE).

CPE isolation in sink drains

Initially, before introduction of the SDP, CPE was isolated from nearly all sinks. All CPE were OXA-48 positive. When the SDP was introduced on 15th July 2017, recovery of CPE from sinks declined rapidly. Figure 2 shows the number of CPE-positive sinks chronologically. After introduction of the SDP, CPE-free sinks often recolonized with CPE, but only for a short period of time.

During the two-week period of decontamination with 12% acetic acid, Rooms 8 and 9 were regularly CPE positive. Concurrently with the re-introduction of 25% acetic acid, the worn-out and dirty siphon in Room 9 was replaced.

During the sampling period from April 2017 to January 2018, 51 species of Enterobacteriaceae were recovered and identified: *Citrobacter* spp. (45.1%), *Klebsiella* spp. (41.2%), *Enterobacter* spp. (7.8%) and *Escherichia coli* (5.9%). *C. freundii* and *K. pneumoniae* were isolated most frequently.

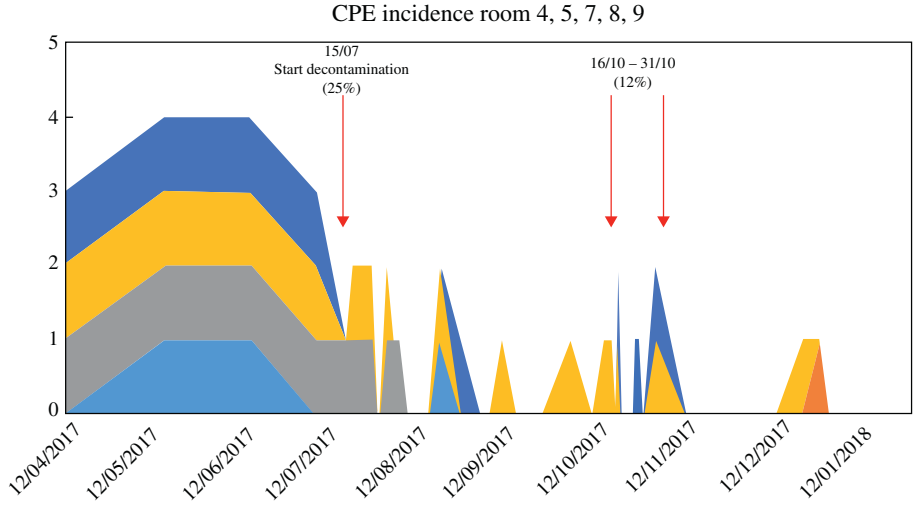

Figure 2. Timeline from 12th April 2017 when initial environment screening was undertaken. Decontamination with acetic acid 250 mL 25% started on 15th July 2017. Decontamination with acetic acid 250 mL 12% occurred between 16th and 31st October 2017. CPE, carbapenemase-producing Enterobacteriaceae. Room 4, light blue; Room 5, orange; Room 6, grey; Room 7, yellow; Room 8, dark blue.

Table II
Isolated pathogens tested for antibiotic resistance, minimal bactericidal concentration (MBC) of acetic acid and OXA-48

Isolate	Amoxicillin-clavulanic acid	Meropenem (MIC)	Piperacillin-tazobactam	Temocillin	Cefuroxime	Ceftriaxone	Ceftazidim	Cefepime	Amikacin	Ciprofloxacin	Sulfamethoxazole-trimethoprim	MBC acetic acid[a]	OXA-48
S.ma	-	-	-	-	-	-	-	-	-	-	S	0.43	nt
K.pn	R	S (1)	R	R	R	R	R	R	R	R	-	0.86	+
P.pu	-	I (4)	S	-	-	-	S	S	S	S	-	0.86	nt
P.pu	-	I (4)	S	-	-	-	S	S	S	S	-	0.86	nt
S.ma	-	-	-	-	-	-	-	-	-	-	S	0.86	nt
K.pn	R	S (1)	R	R	S	S	S	S	S	R	-	1.7	+
C.fr	R	S (0.5)	R	R	-	R	R	I	S	S	-	1.7	+
E.cl	R	S (2)	R	R	-	R	R	S	S	R	-	1.7	+
E.co	R	S (1)	R	R	R	R	R	I	S	S	-	3.4	+
C.fr	R	S (0.5)	R	R	-	S	S	S	S	S	-	0.86	+
K.pn	R	S (2)	R	R	R	R	S	R	S	S	-	3.4	+

S.ma, Stenotrophomonas maltophilia; K.pn, Klebsiella pneumoniae; P.pu, Pseudomonas putida; C.fr, Citrobacter freundii; E.cl, Enterobacter cloacae; E.co, Escherichia coli; nt, not tested; MIC, minimum inhibitory concentration; R, resistant; S, susceptible; I, intermediate.
[a] MBC acetic acid: see text for definition.

In-vitro susceptibility of CPE to acetic acid and antibiotic sensitivity

In order to evaluate the sensitivity of the micro-organisms to acetic acid, the minimal bactericidal concentration (MBC) was defined as the lowest concentration of acetic acid in a two-fold dilution series for which no bacterial regrowth [0 colony-forming units (cfu)] could be detected after 30 min of exposure of a 0.5 McFarland suspension of micro-organisms. Table II shows the MBC and antimicrobial sensitivity patterns of all isolates tested.

As for other antiseptic compounds, the MBCs of acetic acid were found to vary significantly in relation to time of exposure, as shown in Table III. When time of exposure was reduced from 30 min to 5 min, the MBCs increased significantly for most of the strains tested: mean MBC for 30 min was 1.323% [standard deviation (SD) 0.856] and mean MBC for 5 min was 4.04% (SD 4.08).

Discussion

A strong correlation was found between CPE colonization/infection of patients admitted to the ICU and CPE contamination of sinks. It is hypothesized that the sink drain gets colonized with CPE from patients' body fluids. On initial screening, nearly all of the sinks were contaminated with CPE. It is assumed that colonized sinks represent an environmental CPE reservoir from which patients can acquire CPE. Only regular decontamination of the siphons can halt this 'cycling' of bacteria.

A recent study investigated sinks as a source of transmission of CPE in an ICU [5]. Different CPE were found in sink drains and identified as a potential route of transmission. Aerosol formation is a transmission route for bacteria in sink drains [18]. A study found that 42.5% of hospital personnel had hand cultures that were positive for *Pseudomonas* spp. while on duty. When entering the hospital, none of the personnel were positive for *Pseudomonas* spp. Turning on water taps was found to form aerosols, colonizing the hands of personnel in the short term ($t_{1/2}$ 3—76 min). Another study effectively removed the sinks from ICU patient rooms in an effort to reduce the rate of MDR

Table III
Difference in minimal bactericidal concentration of acetic acid for the isolated pathogens compared after 5 min and after 30 min of contact time

Isolate	5 min (%)	30 min (%)
S.ma	0.86	0.43
K.pn	1.7	0.86
P.pu	1.7	0.86
P.pu	1.7	0.86
S.ma	6.9	0.86
K.pn	6.9	1.7
C.fr	3.4	1.7
E.cl	1.7	1.7
E.co	13.8	3.4
C.fr	1.7	0.86

S.ma, Stenotrophomonas maltophilia; K.pn, Klebsiella pneumoniae; P.pu, Pseudomonas putida; C.fr, Citrobacter freundii; E.cl, Enterobacter cloacae; E.co, Escherichia coli.

GNB colonization in patients [4]. They replaced all activities involving water with a water-free patient care protocol. This study found a significant reduction in patient colonization, especially in patients with a longer ICU stay. Removal of all sinks in patient rooms was not feasible in the study ICU for the short term. Another study reviewed literature that used patient-level intervention to prevent resistant GNB infection in critically ill patients [19]. They found a positive effect, but concluded that more studies are needed to further investigate the effects of patient-level interventions on colonization and infection with MDR GNB.

The present study found a strong relationship between a CPE-positive room and CPE-positive patients. This finding implies that patients mainly get colonized or infected with CPE when admitted to CPE-contaminated rooms, indicating the sink as the reservoir from which the CPE is transmitted to the patient. Only a small proportion of CPE-positive patients resided in CPE-negative rooms, probably due to CPE acquisition prior to the ICU stay or breaches in hand hygiene. However, it is important to note that not all patients admitted to CPE-positive rooms acquired CPE.

The LOS of CPE-positive patients was longer than that of CPE-negative patients, suggesting that duration of exposure to CPE-contaminated sinks increases the risk for patient acquisition of CPE.

A puzzling and interesting finding is the fact that no CPE_{before} patients were identified following introduction of the intervention. This could mean that, following introduction of the SDP, no more patients acquired CPE during their ICU stay, and that all four positive patients were colonized before admission to the ICU. More data are needed to support this hypothesis.

Looking at alternative methods to contain a CPE endemic, one hospital in Sweden used acetic acid (vinegar) as a decontamination method [16]. They used 24% acetic acid once weekly to treat sinks infected with metallo-beta-lactamase-producing *Pseudomonas aeruginosa* (Pae-MBL). This method resulted in negative cultures of the sinks and termination of transmission in the ICU. They tested the in-vitro susceptibility of Pae-MBL biofilms to acetic acid, and found that 0.75% acetic acid was sufficient to eradicate the biofilm. The present results of a mean concentration of 1.32% is higher. However, the present study pooled susceptibility of Enterobacteriaceae, *Pseudomonas* and *Stenotrophomonas*. ***Pseudomonas* spp. tested 0.86%, which is in line with previous results.**

It is important to note the dilution of acid in the sink. No isolate was resistant to more than 3.4% acetic acid. The maximum volume of the siphon needed to avoid diluting the acetic acid to less than 3.4% is 1.67 L. None of the sinks tested in this study had a siphon with such a large volume.

Decontamination with acetic acid has been studied since 1969 [20]. A 1% acetic acid solution was used and compared with 0.5% Amphyll and 0.1% Betadine. A 1-h incubation of a broth of *Pseudomonas* spp. was sufficient to reduce the initial colony count of 10^4–10^5 to 0 cfu. Shorter incubation times (1 min and 15 min) were insufficient to decrease the bacterial concentration in the broth below 1 cfu/μL. In both cases, >100 cfu were still present. In 2014, acetic acid at household concentration (6%) proved effective as disinfectant against different species of mycobacteria [21]. This study stressed the broadly effective, available and economic properties of acetic acid. A more recent study used a low concentration of acetic acid (0.16–0.31%) to treat biofilm formation in burn patients [17]. Evidence was found that acetic acid can inhibit growth of burn wound pathogens at a low concentration.

In 2013, antibacterial activity of acetic acid was tested on Gram-negative and Gram-positive bacteria, and MBCs for *P. aeruginosa* and *Acinetobacter baumanii* of 0.166% and 0.312%, respectively, were established [22]. Acetic acid 5% is not considered toxic for treating (*Pseudomonas* spp.) wound infections [23], and is degraded easily in the human body and in the environment.

Another method for the decontamination of sinks is the use of heated siphons (70°C) [18], but this is expensive and requires technical interventions.

Despite sink decontamination three times per week, CPE were occasionally isolated from some sinks, possibly due to frequent disposal of body fluids from CPE-positive patients in the sinks, or from persistent colonization with CPE further down the drain. A study from 2017 indicated that recontamination with *Pseudomonas* spp., *Stenotrophomonas maltophilia* and different Enterobacteriaceae takes several months after complete replacement of all siphons [24]. The present study continued to recover CPE from most sinks, but two sinks grew *S. maltophilia* repeatedly, which could not be linked to infected or colonized patients.

This study did not investigate clonal relations between the CPE strains recovered from sinks and patients. Although several articles have described a relationship between sink and outbreak isolates [10,14], the present authors concluded that the evaluation of clonality between all of the strains from sinks and patients would not contribute to the hypotheses for the mechanism of transmission. In the study setting, most sinks proved to be contaminated concomitantly with a rich mixture of different species of CPE with the same resistance plasmid.

In conclusion, initial colonization of sinks probably occurs when body fluids are disposed in the drains. The data suggest that CPE-positive siphons in the ICU constitute the environmental reservoir from which patients acquire CPE. Transmission from sink to patient could occur by the formation of aerosols and droplets when water drains from the tap into the siphon. Water-free care in the ICU would be the ideal situation, because room sinks would become obsolete, but this was not acceptable to the nursing staff. The decontamination method used for sink drains in this study (250 mL 25% acetic acid, three times weekly) is a valuable protocol. However, the volume of the siphon must be considered. Acetic acid is cheap, widely available and effective for the decontamination of siphons. Corrosiveness and the smell of acetic acid constitute minor inconveniences that can be overcome with proper drainage and the use of appropriate protective wear when handling.

Acknowledgements

The authors wish to thank the Department of Hospital Hygiene and the hospital technical staff for their help.

Conflict of interest statement
None declared.

Funding sources
None.

References

[1] Nordmann P. Carbapenemase-producing Enterobacteriaceae: overview of a major public health challenge. Med Mal Infect 2014;44:51—6.

[2] Jean SS, Lee WS, Lam C, Hsu CW, Chen RJ, Hsueh PR. Carbapenemase-producing Gram-negative bacteria: current epidemics, antimicrobial susceptibility and treatment options. Future Microbiol 2015;10:407—25.

[3] Oteo J, Miro E, Perez-Vazquez M, Navarro F. Evolution of carbapenemase-producing Enterobacteriaceae at the global and national level: what should be expected in the future? Enferm Infecc Microbiol Clin 2014;32(Suppl. 4):17—23.

[4] 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.

[5] 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 Resist Infect Control 2017;6:24.

[6] Doi Y, Paterson DL. Carbapenemase-producing Enterobacteriaceae. Semin Respir Crit Care Med 2015;36:74—84.

[7] Albiger B, Glasner C, Struelens MJ, Grundmann H, Monnet DL, Group tESoC-PEw. Carbapenemase-producing Enterobacteriaceae in Europe: assessment by national experts from 38 countries, May 2015. Euro Surveill 2015;20:30062.

[8] Hoxha A, Karki T, Giambi C, Montano C, Sisto A, Bella A, et al. Attributable mortality of carbapenem-resistant *Klebsiella pneumoniae* infections in a prospective matched cohort study in Italy, 2012—2013. J Hosp Infect 2016;92:61—6.

[9] Costa Pde O, Atta EH, Silva AR. Infection with multidrug-resistant Gram-negative bacteria in a pediatric oncology intensive care unit: risk factors and outcomes. J Pediatr (Rio J) 2015;91:435—41.

[10] 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;59:714—6.

[11] 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. Emerg Infect Dis 2012;18:1242—7.

[12] Nseir S, Blazejewski C, Lubret R, Wallet F, Courcol R, Durocher A. Risk of acquiring multidrug-resistant Gram-negative bacilli from prior room occupants in the intensive care unit. Clin Microbiol Infect 2011;17:1201—8.

[13] Roux D, Aubier B, Cochard H, Quentin R, van der Mee-Marquet N, HAI Prevention Group of the Réseau des Hygiénistes du Centre. Contaminated sinks in intensive care units: an underestimated source of extended-spectrum beta-lactamase-producing Enterobacteriaceae in the patient environment. J Hosp Infect 2013;85:106—11.

[14] 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;82:122—4.

[15] Wolf I, Bergervoet PW, Sebens FW, van den Oever HL, Savelkoul PH, 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;87:126—30.

[16] Stjarne Aspelund A, 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 Infect 2016;94:13—20.

[17] Halstead FD, Rauf M, Moiemen NS, Bamford A, Wearn CM, Fraise AP, et al. The antibacterial activity of acetic acid against biofilm-producing pathogens of relevance to burns patients. PLoS One 2015;10:e0136190.

[18] Doring G, Ulrich M, Muller W, Bitzer J, Schmidt-Koenig L, Munst L, et al. Generation of *Pseudomonas aeruginosa* aerosols during handwashing from contaminated sink drains, transmission to hands of hospital personnel, and its prevention by use of a new heating device. Zentralbl Hyg Umweltmed 1991;191:494—505.

[19] Zaky A, Zeliadt SB, Treggiari MM. Patient-level interventions to prevent the acquisition of resistant Gram-negative bacteria in critically ill patients: a systematic review. Anaesth Intensive Care 2015;43:23—33.

[20] Hedberg M, Miller JK. Effectiveness of acetic acid, betadine, amphyll, polymyxin B, colistin, and gentamicin against *Pseudomonas aeruginosa*. Appl Microbiol 1969;18:854—5.

[21] Cortesia C, Vilcheze C, Bernut A, Contreras W, Gomez K, de Waard J, et al. Acetic acid, the active component of vinegar, is an effective tuberculocidal disinfectant. MBio 2014;5:e00013—4.

[22] Fraise AP, Wilkinson MA, Bradley CR, Oppenheim B, Moiemen N. The antibacterial activity and stability of acetic acid. J Hosp Infect 2013;84:329—31.

[23] McDonnell GE. Antisepsis, disinfection, and sterilization. Washington, DC: American Society of Microbiology; 2007.

[24] 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.