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Original Article

Sink-traps are a major source for carbapenemase-producing *Enterobacteriaceae* transmission

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Abstract

Objective: We studied the extent of carbapenemase-producing Enterobacteriaceae (CPE) sink contamination and transmission to patients in a nonoutbreak setting.

Methods: During 2017–2019, 592 patient-room sinks were sampled in 34 departments. Patient weekly rectal swab CPE surveillance was universally performed. Repeated sink sampling was conducted in 9 departments. Isolates from patients and sinks were characterized using pulsed-field gel electrophoresis (PFGE), and pairs of high resemblance were sequenced by Oxford Nanopore and Illumina. Hybrid assembly was used to fully assemble plasmids, which are shared between paired isolates.

Results: In total, 144 (24%) of 592 CPE-contaminated sinks were detected in 25 of 34 departments. Repeated sampling (n = 7,123) revealed that 52%–100% were contaminated at least once during the sampling period. Persistent contamination for >1 year by a dominant strain was common. During the study period, 318 patients acquired CPE. The most common species were *Klebsiella pneumoniae*, *Escherichia coli*, and *Enterobacter* spp. In 127 (40%) patients, a contaminated sink was the suspected source of CPE acquisition. For 20 cases with an identical sink-patient strain, temporal relation suggested sink-to-patient transmission. Hybrid assembly of specific sink-patient isolates revealed that shared plasmids were structurally identical, and SNP differences between shared pairs, along with signatures for potential recombination events, suggests recent sharing of the plasmids.

Conclusions: CPE-contaminated sinks are an important source of transmission to patients. Although traditionally person-to-person transmission has been considered the main route of CPE transmission, these data suggest a change in paradigm that may influence strategies of preventing CPE dissemination.

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Public health authorities including the US Centers for Disease Control and Prevention (CDC) and World Health Organization have defined carbapenemase-resistant Enterobacteriaceae (CRE) as one of the most urgent threats of emerging antibiotic resistance.[1] CRE produce a broad spectrum of infections associated with high mortality and very limited therapeutic options. A common mechanism of carbapenem resistance is production of carbapenemases (ie, carbapenemase-producing Enterobacteriaceae or CPE), which are β-lactamase enzymes (*bla*) that hydrolyze carbapenems. Bloodstream infections caused by CPEs carry mortality rate of ~50%.[2] Despite increased awareness and national and international interventions,[3–5] these genes and bacteria are spreading and becoming endemic worldwide, being highly mobile and able to transfer between different genomic backgrounds and species.[6]

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In Israel, a nationwide outbreak of CPE emerged in 2006, with nosocomial spread of carbapenemase-producing *Klebsiella pneumoniae* (KPC), predominantly strain type (ST) 258, which was presumably introduced to Israel in 2005.[7] The outbreak was partially contained following a national intervention in all acute-care hospitals and long-term facilities, including active surveillance of high-risk patients, carrier isolation and cohorting.[4] Yet, CPE remain widespread in most medical centers. In recent years, other types of CPE have emerged, including NDM-1, OXA-48, and VIM-producing Enterobacteriaceae.[8–10]

The major route of CPE transmission has been traditionally attributed to patient-to-patient cross transmission. Thus, colonized patients are considered the most important reservoir, and infection control breaks in hand hygiene via healthcare workers are considered the predominant transmission chain.[11] Recently, the role of environmental reservoirs, and particularly the hospital water environment, in the spread of multidrug-resistant organisms (MDRO) has been stressed.[12–14] Specifically, sink-traps and water

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drainage systems have been increasingly recognized as sources of gram-negative MDRO nosocomial outbreaks.[5,14–16] Although the mechanism of transmission from sink drains to patients has been partially understood,[17–19] the scale of the problem is unknown.

In this study, we assessed the extent and persistence of sink-drain and sink-outlet contamination and traced the possible transmission patterns between patients and hospital room sinks in our institution.

Methods

Setting

The Sheba Medical Center (SMC) is the largest tertiary-care academic medical center in Israel. SMC has a 1,600-bed capacity for both acute-care and rehabilitation patients, including 300 beds in 7 internal medicine departments, with overall 96,800 annual admissions to the acute-care hospital. Most hospital rooms accommodate 3 patients, and a few single bedrooms are available in each ward. ICUs include either semiclosed or closed single-bed units. Each room is provided with an in-room sink and a shower room with additional sink. Routine cleaning of sinks and showers in all hospital rooms takes place once to twice a day by pouring into the sink drain 500 mL NaDCC (ie, sodium dichloroisocyanurate) solution at a concentration of 1,000 ppm. Since 2007, a CPE-defined cohort area including 10–20 beds, has been allocated in a rotating system in one of the internal medicine wards. These accommodate mostly internal medicine patients but also include a few surgical patients. The CPE cohort space shifts from one ward to another every 6 months. Thus, since 2007, each of the internal medicine wards hosted the cohort area at least once. A similar, but smaller and permanent cohort area is allocated in the children's hospital (including 4–6 beds).

CPE screening of hospitalized patients

Following a nationwide intervention program,[4] rectal screening of high-risk patients routinely takes place at SMC. High-risk patients are defined as anyone admitted with a history of hospitalization during the previous 6 months or admitted from a long-term care facility or another medical facility. High-risk patients are screened upon their admission to SMC and no later than 48 hours from admission. On average, 20%–25% of hospital admissions are defined as high-risk admissions, and adherence to the admission screening policy is ~70%. Moreover, ~2% of all high-risk patients screened on admission are positive for CPE.

Additionally, high-risk units, such as ICUs and hemato-oncology units, conduct a weekly or bi-weekly routine screening of all hospitalized patients. Since 2016, every patient exceeding 14 days of hospitalization has been screened weekly to further detect any acquisition of CPE. Once a patient is detected as a CPE carrier, all patients in that department are also screened to search for the index case. An index case is defined as a carrier possessing the earliest CPE isolate with >95% similarity to isolates from patients of the same department, determined using pulsed-field gel electrophoresis (PFGE). Any identified carrier is transferred to the designated cohort space, located within one of the internal medical departments, with dedicated nursing staff and equipment.

Sink-drain screening

In April 2017, we identified the sink traps as the source of a prolonged OXA-48–producing *Serratia marcescens* outbreak in our ICU.[5] Furthermore, most of the CPE acquisitions in all other departments could not be associated with an index-case CPE patient. This led us to screen all patient room sink-traps or sink-outlets in departments in which new CPE acquisitions were detected. Specifically, within a few months 7 internal medicine wards were screened, regardless of new CPE acquisitions because they all had hosted the CPE cohort space at some stage during the previous 4 years. One exception was a single newly renovated internal medicine ward (named IM-F).

Screening was performed by vigorously swabbing the sink-outlet surface with 4 sterile cotton swabs or inserting the 4 swabs as far as possible into the sink outlets and similarly swabbing the surface of the pipe leading to the sink-trap. To avoid enrichment of Enterobacteriacae over other bacterial species, swabs were placed in sterile saline and were transferred to the laboratory immediately. CPE isolates from sinks were compared using PFGE, to detect environmental and patients isolates from the same department, and the direction of transmission was determined according to sampling dates.

Longitudinal sink-outlet and drain screening was performed in departments where CPE acquisitions were most frequently detected. These were the 7 internal medicine wards, as well as in the general ICU and the hemato-oncology departments. The sink outlets in these departments were routinely screened on a weekly or monthly basis.

Study period

The study was conducted from April 2017 to March 2019. During this 2-year period, policy regarding active surveillance of patients did not change.

Microbiology methods

Surveillance rectal samples were obtained by Copan Amies sterile transport swabs (Copan Diagnostics, Murrieta, CA). Swabs were streaked in the classical method onto Chromagar KPC plates (Hy Laboratories, Rehovot) to achieve isolated colonies and samples were incubated overnight at 35°C in ambient air. Environmental samples were acquired as described above. The containers were vigorously spun in a vortexer prior to streaking the liquid onto Chromagar KPC plates with a new sterile swab. Suspicious colonies were identified using Maldi-TOF. Carbapenemase genes were identified by PCR using Xpert Carba-R cartridges (GeneXpert, Cepheid, Sunnyvale, CA).

Molecular characterization of isolates was performed using PFGE, where XbaI-digested bacterial DNA embedded in agarose plugs were subjected to PFGE analysis at 14°C using a CHEF-DR III system (Bio-Rad, Hercules, CA). Genetic similarity analysis was performed with BioNumerics version 7.6 software (Applied Maths, bioMérieux, Marcy-l'Étoile, France) using the dice coefficient and UPGMA clustering.

From the genetically similar pairs of sink-patient isolates, we randomly sampled 2 pairs for whole-genome sequencing. Sequencing methods are detailed in Supplement 1 (online).

Ethical considerations

The study protocol was approved by the Institutional Review Board at Sheba Medical Center. All patients were sampled based on clinical needs and/or routine institutional screening policy. Accordingly, an exemption from informed consent was granted by the committee. All methods were performed in accordance with the relevant guidelines and regulations.

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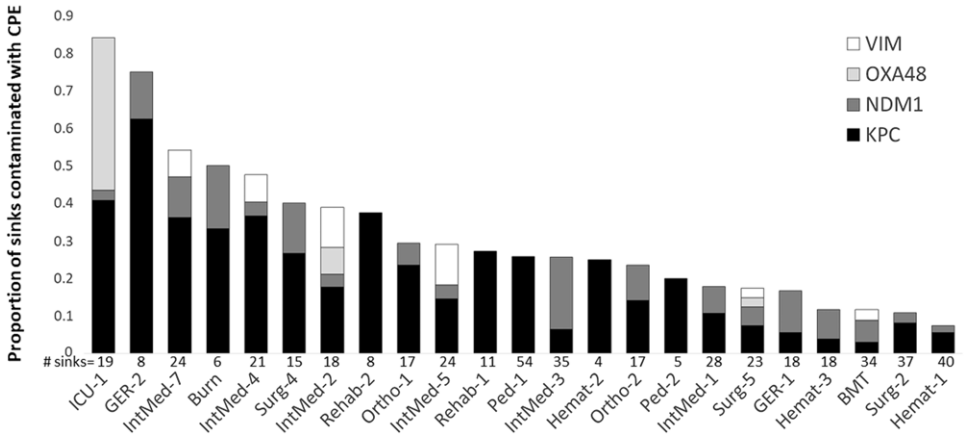


Figure 1. Initial point prevalence of CPE-contaminated sink-drains among departments in which CPE-contaminated sinks were identified.

Results

CPE-contaminated sinks

During the study period, 592 patient-room sinks in 34 departments were screened for CPE environmental contamination. In total, 144 CPE contaminated sinks (24% of all assessed sinks) were detected in 25 (73.5%) of the 34 departments. Of the 9 of 34 departments in which CPE was not recovered on initial screening, 7 were newly renovated departments in which the plumbing system had been fully replaced. The density of CPE contaminated sinks varied from CPE detection in only 1 or 2 sinks in some departments to nearly 90% of sinks in others. The leading CP-gene was bla_{KPC}, which accounted for >50% of contaminated sinks (Fig. 1). Longitudinal serial screening in medical wards, ICUs, and the hemato-oncology wards revealed that CPE-contaminated sinks continued to be contaminated with the same CP-producing bacteria throughout the screening period.

Patient acquisition of CPE

During the 2-year study period, 318 patients acquired CPE during their hospitalization. Intensive epidemiologic investigation of cases who acquired CPE were conducted, including screening of patients hospitalized in the same department during the 2-week preacquisition period and identifying potential interactions with other carriers with identical CPE strains in the other hospital wards. However, only 49 (15%) of 318 cases could the acquisition be attributed to another index-case patient (Fig. 2). In 127 patients (40%), no index case was detected, but a contaminated sink was identified with the same CPE strain (same species and same *bla* gene) as that acquired by the patient. In 57 additional cases, without an identifiable index case, we identified CPE-contaminated sinks with different bacterial species than that acquired by the patient but with the same *bla* gene, where the sink could be a possible source of transmission. In the remaining 85 patients (27%), the potential source could have been an unidentified index patient, an unrecognized community acquisition, or another environmental source.

The most commonly bacterial species acquired by patients was *K. pneumoniae* (46%), followed by *E. coli* and *Enterobacter* spp (23% each), whereas 2 or more different species were isolated in 11 cases (3%) (Fig. 3a). Each of the different bacterial species carried various CP-genes. We identified bla_{KPC} as the most common gene carried (44%) followed by bla_{NDM} (35%), $bla_{OXA\text{-}48}$ (13%) and bla_{VIM} (8%) (Fig. 3b). The daily prevalence of CPE carriers together with clinical cases was ~40 patients per day (~3%–4% of all hospitalized patients), and most but not all were hospitalized in the cohort area, as described above.

Although the distribution of CP genes detected in the sinks was very similar to that acquired by patients (Fig. 3b), the distribution of the various *Enterobacter* spp found in sinks differed significantly from that among patients. *Escherichia coli* was detected only in 2 sinks but was common in patients (<1% vs 23% accordingly), and *Enterobacter* spp was commonly detected in sinks but less so in patients (40% vs 23% accordingly) (Fig. 3a).

Sinks as the source of patients' CPE acquisition

Of 132 cases with concordant sink-patient CPE, bacterial strains were available for further genotypic characterization in 107 cases. Among them, 39 (36.4%) had identical PFGE patterns, including different bacterial species harboring different genes (Table 1 and Supplementary Fig. S1 online). The bacterial species and genes of these identical pairs are reported in Supplementary Table S1 (online). In 20 cases, a sink-to-patient route of transmission was assumed due to the temporal relation, in which the sink contamination was documented before the patient was hospitalized and the patient initial screening was negative. Yet, in most cases, sinks were screened only after identifying the patient as a CPE carrier, and no assumption could be made regarding the route of transition (ie, sink-to-patient vs patient-to-sink).

The 2 sink-patient pairs randomly sampled for genome sequencing included *Klebsiella pneumoniae* bearing $bla_{\text{NDM-1}}$ and *Enterobacter cloacae* (appeared to be *Enterobacter hormaechei*) bearing bla_{VIM}. Using hybrid genome assembly, we resolved the chromosome and plasmids in each of the 4 isolates. Also, 3 isolates harbored 3 plasmids, and 1 isolate harbored only 2 plasmids. In each sink-patient pair of isolates, we identified 2 plasmids that were nearly identical with no major structural changes. The plasmids shared by the 2 *E. hormaechei* isolates and 1 of the plasmids shared by the 2 *K. pneumoniae* isolates harbored genes conferring antimicrobial resistance (Fig. 4).

Contamination persistence of sink-traps and pipes

During 2 years of follow-up, repeated sink sampling was performed in 9 departments where CPE carriers were frequently hospitalized. In total, 7,123 samples were collected from 166 repeatedly sampled sinks; 16 sinks of the ICU were sampled weekly for 110 weeks and sinks of the other 8 departments were sampled

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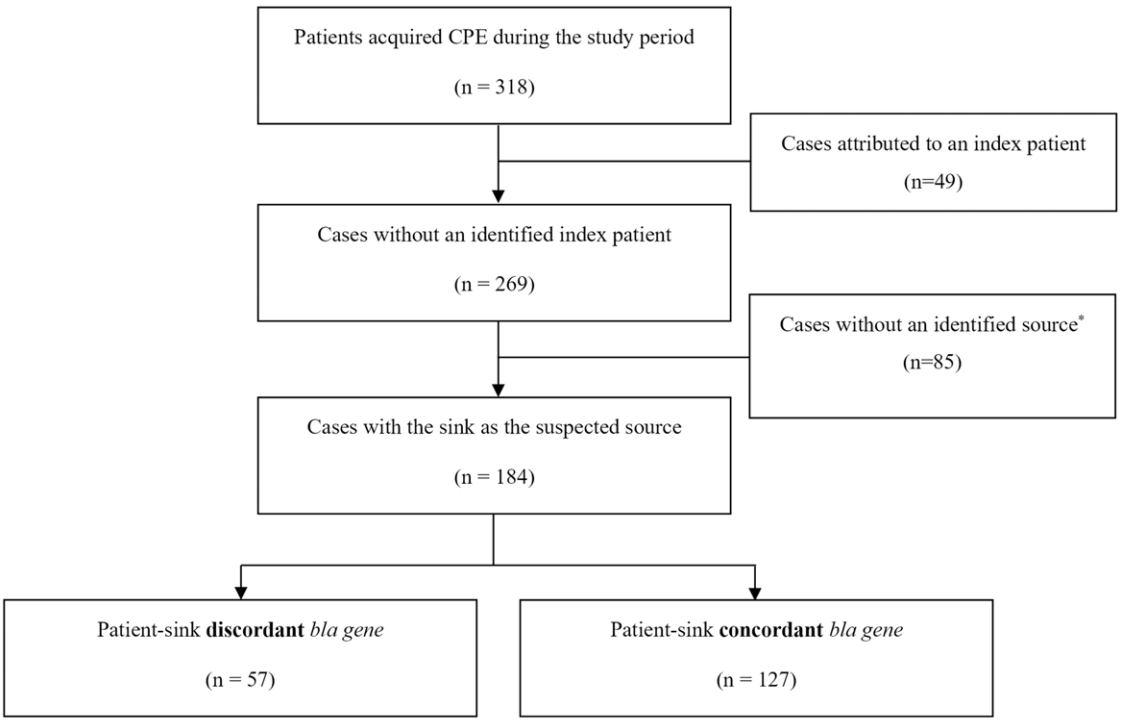


Figure 2. Results of the epidemiological investigation of the 318 CPE acquisition cases during the study period.

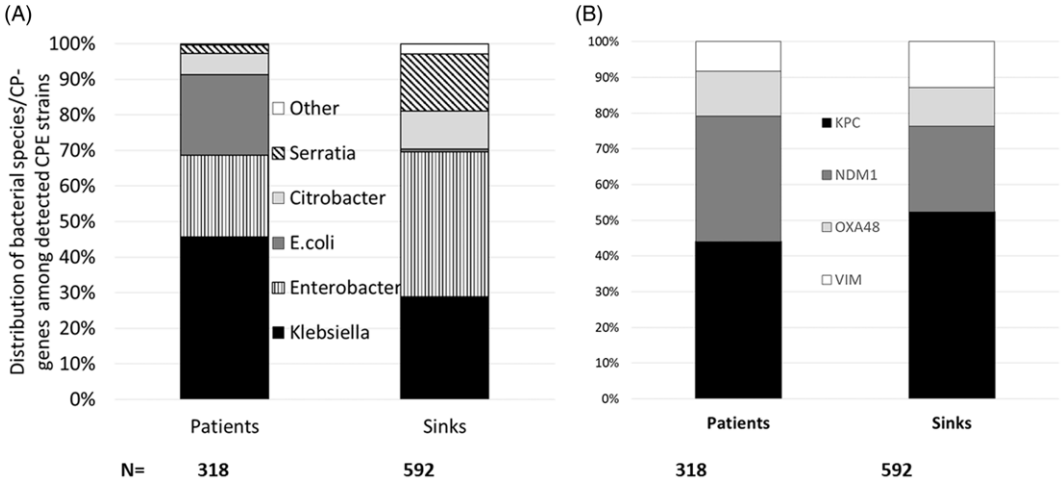


Figure 3. Distribution of the different bacterial species as defined by (A) matrix-assisted laser desorption/ionization time-of-flight mass spectroscopy (MALDI-TOF MS) and CPE genes, as defined by (B) polymerase chain reaction (PCR) among patients and sinks.

for at least 1 year, with 10–75 sampling times during this period. Repeated sampling of the ICU sinks rapidly revealed that 100% of sink traps were contaminated with CPE. The proportion of contaminated sinks in repeated sampling of the other departments revealed that sinks of most of the departments were heavily contaminated with CPE (52%–90% of sinks). However, 2 departments were exceptions: hemato-oncology, which was renovated 2 months after starting the environmental sampling, and the IM-F unit, which moved to a newly renovated department and did not host the CPE cohort area during the study period (Supplementary Fig. S2 online).

Although different CPE bacterial species with different CP genes were detected in different sinks, most commonly, a single dominant CPE strain was repeatedly isolated from a single sink for many weeks. Occasionally, a new strain replaced the dominant one, or 2 dominant strains were concurrently isolated. Yet, in 50 (21%) of the 240 repeatedly sampled sinks, we detected a single dominant strain (defined by PFGE). Different bacterial species carrying different CP genes persisted in the various sink-drains for over a year. Most common were KPC-producing *Enterobacter* spp (n = 13), followed by OXA-48–producing *S. marcescens* (n = 11) (Supplementary Fig. S3 online). Moreover, all of these sink traps

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Table 1. CPE Isolates Acquired by 318 Patients According to CP Genes and the Attributed Factor

Acquisition of CPE Genes	No.	Attributed to Another Index Case, No. (%)	Possibly Attributed to a Contaminated Sink, No. (%)[a]	Probably Attributed to a Contaminated Sink, No. (%)[b]	Genetically Identical Sink-Patient Isolates, n/N[c]
KPC	144	20 (13.8)	24 (16.7)	69 (47.9)	12/60
NDM-1	115	19 (16.5)	31 (26.9)	42 (36.5)	16/34
OXA-48	41	5 (12.2)	2 (4.9)	10 (24.4)	7/7
VIM	27	6 (22.2)	1 (1.8)	11 (40.7)	4/6

Note. CPE, carbapenemase-producing Enterobacteriaceae; CP, carbapenemase producing; KPC, carbapenemase-producing *Klebsiella pneumoniae*; NDM-1, New Delhi metallo β-lactamase-1; VIM, Verona integron-encoded; PFGE, pulsed-field gel electrophoresis.
[a]Sink contaminated by a different bacterial specie but with an identical CP gene and no index case suggested.
[b]Sink contaminated by an identical bacterial specie with an identical CP gene and no index case suggested.
[c]No. of patient-sink identical isolates per no. of pairs assessed by PFGE.

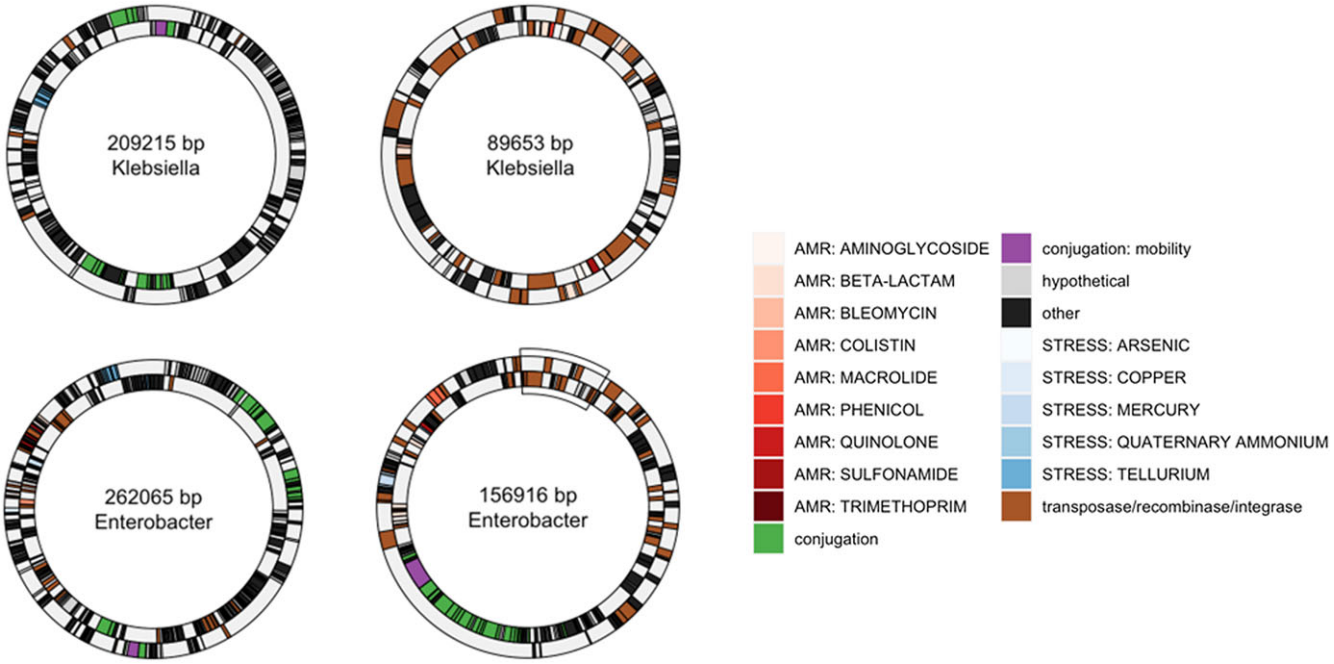


Figure 4. Circle plot representation of plasmids shared between patient and sink isolates. Coding domain sequences are colored based on annotations used in prokaryotic genome annotation pipeline (PGAP) with resistance cross annotated using AMRFinder software. The outer ring contains CDS on the positive strand and the inner ring contains CDS on the negative strand. The inversion between the smaller *Enterobacter* plasmid pair is denoted as a larger wedge.

were replaced at least once; however, recontamination with the same strain occurred rapidly. In several cases, a single strain persisted but carried different CP genes (Fig. 5a). In other cases, different species carrying the same resistance gene were detected in consecutive weeks (Fig. 5b).

Discussion

In this study, we have reported the frequency of CPE-contaminated sinks in an acute-care hospital, and we have demonstrated that they are frequently and persistently contaminated with a dominant clone. Most importantly, we have shown that CPE contaminated sinks are not only a source of outbreaks in high-risk units but that sink-traps serve as an important environmental niche of gram-negative MDRO (specifically CPE) that play a major role in CPE transmission in nonoutbreak settings.

Our findings suggest a broader perspective of healthcare-associated infections, implying that sink traps and wastewater pipes may play a role in CPE transmission to patients. These findings contribute to the body of knowledge regarding transmission routes of nosocomial infection and may influence the types of interventions required to prevent CPE dissemination. At the SMC, as in most medical centers in Israel, rigorous nationwide infection control efforts have been undertaken since 2007. Yet these focused mainly on hand hygiene and strict isolation of patients and establishment of CPE-defined cohort areas. This paradigm, in which the role of patient-to-patient transmission was central and exclusive, led to the policy of a rotating CPE areas between the different hospital departments. Here, we have demonstrated how this has led, at least in our medical center, to gradual contamination of the wastewater pipes in most of the departments.

Sink contamination with CPE was detected in most of the hospital departments, with point prevalence of 24%. Nevertheless, repeated sampling revealed that the realistic proportion of CPE-contaminated sinks is much higher and depends on the age of the

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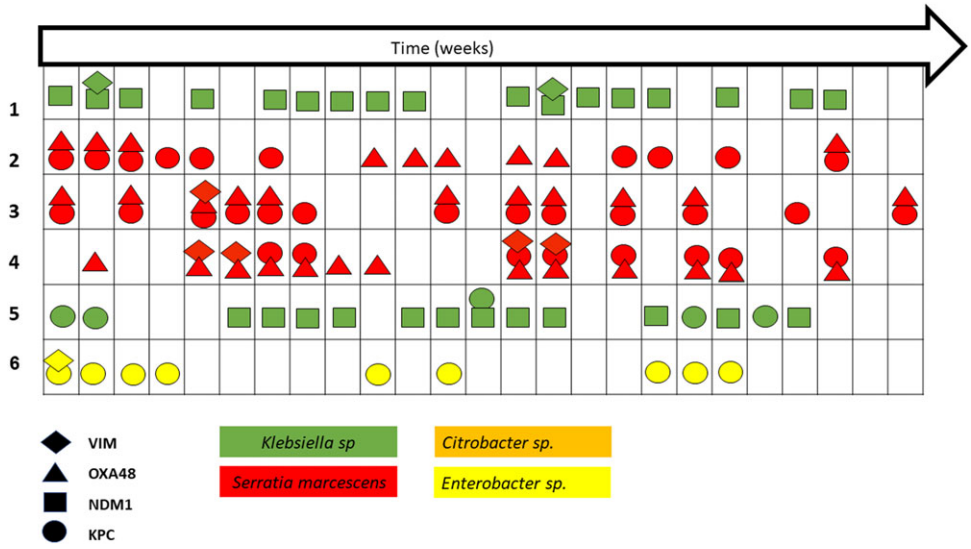


Figure 5a. Examples of patterns of prolonged CPE contamination in sinks in which a single specie was detected, but with various CP genes. Each CP gene is denoted in a different geometric shape: circle, *bla*KPC; square, *bla*NDM; triangle, *bla*OXA48; and rhombus, *bla*VIM). The colors of the shapes denote the bacterial species: yellow, *Enterobacter* spp; green, *Klebsiella* spp; red, *S. marcescens*; orange, *Citrobacter* spp.

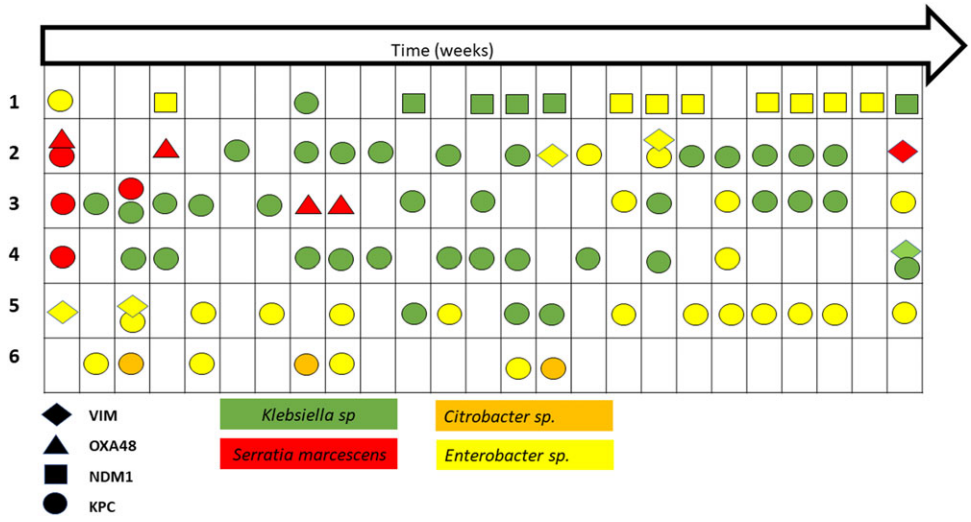


Figure 5b. Examples of patterns of prolonged CPE contamination in sinks in which different species were detected, but mostly with the same CP genes. Each CP gene is denoted in a different geometric shape: circle, *bla*KPC; square, *bla*NDM; triangle, *bla*OXA48; and rhombus, *bla*VIM. The colors of the shapes denote the bacterial species: yellow, *Enterobacter* spp; green, *Klebsiella* spp; red, *S. marcescens*; orange, *Citrobacter* spp.

wastewater pipes and the burden of exposure of the sinks to CPE colonized patients. Thus, sinks in newly renovated departments were generally negative for CPE, whereas sinks in departments that hosted the CPE cohort area were mostly contaminated. Using sequencing methods, we have demonstrated that the sink isolates carried similar plasmids as those found among patients hospitalized in the same hospital room. Thus, sinks are not only a habitat for MDROs and a reservoir for CPE transmission but also potentially contribute to nosocomial transmission of antibiotic resistance.

We observed that although sink contamination was persistent, there was no specific bacterial specie or CP gene. Although *Serratia*, *Klebsiella*, and *Enterobacter* were the most prevalent contaminating species, other Enterobacteriaceae were also detected.

Interestingly, although the distribution of different CP genes among sink isolates was very similar to the distribution among patient isolates, the distribution of bacterial species differed. For instance, *E. coli* was the most common bacterial species detected among patients but was only rarely detected in sinks. This may suggest the transmission of CP genes, transposons, or plasmids between common sink bacterial strains to different bacterial species, which are more common in patients. The transmission of CP genes between different bacterial species has been previously reported in vitro and in vivo.[20–22] Thus, the sinks may serve as hubs for evolution and spread of diverse CPE strains.

Despite repeated replacements of sink traps, the new traps were rapidly recontaminated with the same strain. Because none of the hospitalized patients at that time carried these bacteria, the wastewater pipes were the likely source of this recontamination.

Particularly disturbing is the lack of reported successful interventions to eliminate sink-trap and wastewater contamination. In our experience, as in previous reports, sink-trap and even full pipe replacement resulted only in a temporary resolution.[5,23] Similarly poor results were reported with other interventions such as weekly cleaning with acetic acid, bleach, or hydrogen peroxide.[12,24–27] Expensive self-disinfecting siphons have also been suggested, but these do not always result in decontamination.[5,15] Several publications have suggested that foam disinfectants containing hydrogen peroxide may be more effective than bleach for sink drain disinfection.[28–30] Prospective evaluation is warranted to assess their effect on sink-to-patient transmission. Currently, the only effective means to prevent sink-to-patient MDRO transmission

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may be structural aspects (eg, eliminating sinks that are not necessary) together with education to address the infectious risks of sinks and prevention of their misuse (no flushing of any substance into the sink and sole use for hand washing). Additionally, instead of rotating the cohort site between different hospitalization wards, it is reasonable to place MDRO carriers in cohorts in a single designated site. It might also be helpful to delay the reuse of hospital sites that have accommodated CPE carriers. Because there is no evidence for the efficacy of these interventions, further investigation on the topic is warranted.

Our study had several limitations. First, not all bacterial isolates were genetically characterized. Furthermore, only in 20 cases we could define the temporal relation, providing direct evidence of sink-to-patient transmission. Frequently, the sinks were sampled only after a CPE carrier patient was already detected. Last, the genetic characterization of the isolates included only PFGE and further genomic investigation of the isolates may lead to further elucidation of the route of transmission between sinks and patients. Further genomic studies of real-life settings are required to understand the evolution of CPE in wastewater pipes and their transmission to patients.

In conclusion, the persistent and widespread sink-trap contamination with CPE in our medical center, despite multiple infection control measures, is worrisome. This report adds to the accumulating data indicating the significant role of sinks in CPE transmission and suggests a paradigm change, in which infection control interventions to prevent CPE dissemination should focus on environmental control and appropriate behavior regarding the sink and its surroundings. Innovative technologies to decontaminate sink traps and wastewater are urgently needed.

Supplementary material. To view supplementary material for this article, please visit https://doi.org/10.1017/ice.2023.270

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Competing interests. G.R.Y. served as a member of an advisory board for Moderna, and received speaking fees from MSD, Pfizer, AstraZeneca, and Medison. W.P.H. served as a scientific advisor to Biobot Analytics and have been a consultant for Merck Vaccines.

All other authors declare no competing interests.

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