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Toilet drain water as a potential source of hospital room-to-room transmission of carbapenemase-producing *Klebsiella pneumoniae*

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SUMMARY

Background: Carbapenemase-producing Enterobacterales (CPE) have rapidly emerged in Europe, being responsible for nosocomial outbreaks.
Aim: Following an outbreak in the burn unit of Ghent University Hospital, we investigated whether CPE can spread between toilets through drain water and therefrom be transmitted to patients.
Methods: In 2017, the burn centre of our hospital experienced an outbreak of OXA-48-producing *Klebsiella pneumoniae* that affected five patients staying in three different rooms. Environmental samples were collected from the sink, shower, shower stretcher, hand rail of the bed, nursing carts, toilets, and drain water to explore a common source. Whole-genome sequencing and phylogenetic analysis was performed on *K. pneumoniae* outbreak isolates and two random *K. pneumoniae* isolates.
Findings: OXA-48-producing *K. pneumoniae* was detected in toilet water in four out of six rooms and drain water between two rooms. The strain persisted in two out of six rooms after two months of daily disinfection with bleach. All outbreak isolates belonged to sequence type (ST) 15 and showed isogenicity (<15 allele differences). This suggests that the strain may have spread between rooms by drain water. Unexpectedly, one random isolate obtained from a patient who became colonized while residing at the geriatric ward clustered with the outbreak isolates, suggesting the outbreak to be larger than expected. Daily application of bleach tended to be superior to acetic acid to disinfect toilet water; however, disinfection did not completely prevent the presence of carbapenemase-producing *K. pneumoniae* in toilet water.

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Conclusion: Toilet drain water may be a potential source of hospital room-to-room transmission of carbapenemase-producing *K. pneumoniae*.

Introduction

Carbapenemase-producing Enterobacterales (CPE) are a major public health concern for infection control and individual therapeutic management, as treatment options against CPE infections are very limited [1,2]. Carbapenem resistance may result from the acquisition of carbapenemase enzymes that are capable of inactivating almost all β-lactam antibiotics [1,2]. CPE strains have been rapidly emerging since 2010 in Europe with a prevalence of 0.55% of Enterobacterales in hospitalized patients in Belgium in 2015 compared to 0.25% in 2012 [3,4]. In 2018, four additional European countries reported regional or inter-regional spread compared to 2015 resulting in 20 out of 37 European countries reporting at least regional spread of CPE (epidemiological stages 3–5) [5]. The first CPE type OXA-48 worldwide was reported in Turkey in 2001 [6]. OXA-48-producing CPE are now commonly found in Europe, being responsible for nosocomial outbreaks [3,4]. The nowadays most frequently detected CPE organism is *Klebsiella pneumoniae*, which predominantly causes healthcare-associated infections [1,7]. Carbapenemase-positive *K. pneumoniae* strains are associated with high transmissibility and epidemic potential [7]. The European epidemic of carbapenemase producing *K. pneumoniae* is driven by the expansion of a small number of clonal lineages, comprising sequence types (ST) 11, 15, 101, 258/512, and their derivatives [7]. These lineages are often associated with outbreaks and provide a greater opportunity for the acquisition of antibiotic resistance genes compared to other lineages [7]. ST258/512 has played a major role in the dissemination of KPC enzymes in the USA and has successively spread to southern European countries [7,8]. ST11, 15 and 101 are also international STs and mainly associated with OXA-48-like and NDM-like enzymes in Europe [7,9,10].

Since intestinal carriage serves as a reservoir of CPE, screening in healthcare settings and isolation of carriers is necessary to limit the spread of these pathogens [1]. Effective prevention and control of *K. pneumoniae* outbreaks require a detailed understanding of how transmission occurs by tracking infected and colonized patients [11,12]. Several phenotypic methods for pathogen characterization (antimicrobial susceptibility testing, mass spectrometry-based methods, serotyping) are used to monitor the spread of CPE in hospitals worldwide [11]. In recent years, pathogen characterization has moved to more sensitive molecular methods [11]. Whole-genome sequencing (WGS) provides a rapid tool in outbreak analysis with a higher resolution compared to conventional methods [11]. WGS has been applied to clinical isolates to monitor the evolution of a newly emerged extended-spectrum β-lactamase-producing *K. pneumoniae* in the Netherlands [13]. WGS on clinical isolates as well as metagenomic sequencing directly on faecal samples were also applied to track an outbreak of carbapenem-resistant *K. pneumoniae*, revealing unexpected patient-to-patient transmissions [12,14,15]. Next-generation sequencing is a promising tool to link patients directly to environmental isolates and to detect reservoirs.

Efforts to better understand the reservoirs and transmission routes of CPE are essential. Within hospitals, aqueous environments prone to biofilm formation such as sink drains, sink surfaces and faucets were most frequently colonized with carbapenem-resistant organisms and have been associated with CPE outbreaks [16–21]. This may be partly due to difficulties in eradicating CPE from sanitary systems [21]. Transmission may result from direct or indirect contact with water, or droplets created during water activities [17]. Contaminated toilets have been shown to produce potentially infectious aerosols in substantial quantities during flushing [22,23]. These aerosols may remain suspended in the air for longer than 30 min post flush and are mostly 0.3 μm in diameter [23]. Subsequent toilet users can be exposed to this toilet plume which may potentially result in the transmission of infectious microorganisms shed in faeces [22]. In this study, we investigated the hypothesis that CPE can spread between toilets in different hospital rooms with common waste plumbing through drain water and thereby transmitted to patients.

Methods

Outbreak setting

From December 2016 to March 2017, an OXA-48-producing *K. pneumoniae* outbreak took place at the burn centre of the Ghent University Hospital affecting five patients staying in three different rooms. The Ghent University Hospital is a tertiary care centre in Belgium with about 1000 beds. The burn centre consists of six linearly structured rooms (A–F) with open bathrooms, reserved for severely burned patients who need intensive care. All rooms share common waste plumbing. Hand hygiene is performed before touching a patient, before aseptic procedures, after body fluid exposure, after touching a patient, and after touching patient surroundings with an overall compliance of 80%. Before entering the room, the healthcare worker puts on gloves and a long-sleeve gown. In addition, a mask is worn during wound care procedures. Personal protective equipment is removed after leaving the room. Medical materials and cleaning equipment are dedicated to a single patient. Toilet brushes are replaced after patient discharge. Rectal and wound screening for multidrug-resistant Gram-negative bacteria is performed at the time of admission and twice weekly therafter.

Ethical approval

Ethical approval for this study was obtained from the local Ethics Committee (Ghent University Hospital, 2020-030).

Outbreak investigation

Given the standard use of maximal barrier precautions, an environmental source was suspected. Therefore, environmental screening was performed to explore a potential

common source. ESwab® (Copan, Brescia, Italy) samples were collected from the sink, shower, shower stretcher, and hand rail of the bed of rooms 3 and 6 in the burn centre (rooms that CPE-positive patients had occupied). Environmental objects were not screened in room 4 since surfaces and devices were already airborne-disinfected with acetic acid and hydrogen peroxide. In addition, two nursing carts used in the burn centre were sampled. Also, toilet water from all rooms was collected before disinfection and after two months of daily disinfection of toilet water with bleach. Drain water was tapped from a drain pipe located between rooms D and E of the burn centre.

Conventional microbiological analyses

Samples (100 mL) of water obtained from toilet bowls and a drain pipe were concentrated using the membrane filter technique. The filter was submerged in 5 mL physiologic water with glass beads and vortexed. Subsequently, 100 μL of this solution and the eSwabs were cultured on a chrom-ID® ESBL (BioMérieux, Marcy l'Etoile, France) and chrom-ID CARBA SMART (BioMérieux) medium. The plates were incubated overnight aerobically at 35°C. All grown colonies were identified at species level using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (Bruker Daltonik GmbH, Bremen, Germany). Phenotypic susceptibility testing was performed using the Kirby–Bauer disc diffusion test (inoculation of a 0.5 McFarland solution on Mueller–Hinton II media and overnight incubation at 35°C). Susceptibility for the antibiotics ampicillin (10 μg), amoxicillin–clavulanate (30 μg), cefuroxime (5 μg), ceftazidime (10 μg), cefotaxime (5 μg), temocillin (30 μg), piperacillin–tazobactam (36 μg), meropenem (10 μg), amikacin (30 μg), tobramycin (10 μg), levofloxacin (5 μg), trimethoprim/sulfamethoxazole (25 μg) and colomycin (10 μg) were tested. Breakpoints were interpreted according to the EUCAST guidelines v10.0. Enterobacterales with a suspicious antimicrobial susceptibility profile were tested for bla_{OXA-48}, bla_{NDM}, bla_{KPC}, bla_{GES}, bla_{IMI}, bla_{VIM}, and bla_{IMP} resistance genes using an in-house polymerase chain reaction based on the method of Monteiro *et al.* [24].

Whole-genome sequencing

DNA was extracted from a liquid suspension of the purified OXA-48-producing *K. pneumoniae* cultures using the QIAamp DNA Mini Kit (Qiagen, Hilden, Germany). For each sample, 312 ng DNA (50 μL) was mechanically sheared with Covaris LE220-plus Focused-Ultrasonicator (Covaris, Inc., Woburn, MA, USA) to a target fragment size of 200 bp. The samples were purified with AMPure XP beads (Beckman Coulter, Indianapolis, IN, USA) using an automated MicroLab STAR® system (Hamilton Company, UT, USA). A barcode of 11 bp was attached. Cluster generation of pooled libraries (1.2% phiX spike-in) was performed with the cBot 2 cluster generation system (Illumina, San Diego, CA, USA). The library preps were sequenced on a HiSeq 3000 instrument (Illumina) in single-end mode generating 50 bp reads. Resulting FASTQ files were quality-trimmed and de-novo-assembled in contigs using CLC Genomics Workbench version 12 (CLC Bio, Aarhus, Denmark). In order to detect closely linked clusters of outbreak strains and determine phylogeny, whole-genome multi-locus sequence typing (wgMLST) was performed on the assemblies with Ridom SeqSphere+ version 05 (Ridom GmbH, Münster, Germany). The wgMLST analysis was performed at the University Medical Center of Groningen with an in-house wgMLST scheme for *K. pneumoniae* containing 4891 different loci. The wgMLST scheme was developed using the seed genome *K. pneumoniae* subsp. *pneumoniae* NTUH-K2044 (GenBank accession number NC_012731.1; 15-JUN-2016). A cluster alert quality of at least 90% good MLST isolates and cluster alert distance of 15 allele differences was used to detect closely related OXA-48-producing *K. pneumoniae* isolates [25]. These criteria are based on retrospective analysis of well-defined outbreaks and out-group OXA-48-producing *K. pneumoniae* isolates with the same MLST profile [25]. ST of WGS data was performed using the '*Klebsiella pneumoniae*' database from PubMLST.

Results

Description of the outbreak

In November 2016, patient 1 (index patient) was admitted to the emergency department and subsequently hospitalized in the burn centre in room B. At the time of admission and two days later, rectal swabs (Eswab) tested negative for CPE. However, three days later in early December 2016, the rectal swab yielded *K. pneumoniae* producing OXA-48. Contact isolation precautions were taken until the patient was discharged at the end of December 2016. Three weeks later, in January 2017, patient 2 was hospitalized in the same room as patient 1. The initial rectal screening was negative, but turned positive for OXA-48-producing *K. pneumoniae* three days later. Patient 2 was discharged in February 2017. These findings led to additional disinfection of the room involved with atomized hydrogen peroxide and peracetic acid (Aseptanios™; Laboratoires Anios, Lille, France) following the manual cleaning and disinfection procedure (on February 3rd, 2017). However, in the following weeks, three new cases of colonization with OXA-48-producing *K. pneumoniae* were detected among patients in the burn centre. All patients had negative rectal screening results initially and they stayed in rooms other than room B. Patients 3 and 5 were residing consecutively in room F and patient 4 was residing in room C. Patients 3, 4, and 5 tested positive for OXA-48-producing *K. pneumoniae* at the end of February to the beginning of March 2017. An overview of colonized patients 1–5 residing in rooms A–F of the burn centre as a function of time is shown in Figure 1.

Outbreak investigation and conventional testing

All environmental samples collected from the sink, shower, shower stretcher, and hand rail of the bed (rooms 3 and 6, on March 6th, 2017), as well as from the nursing carts (March 13th, 2017), tested negative for CPE. OXA-48-producing *K. pneumoniae* was detected in toilet water of four of the six rooms of the burn centre (rooms B, C, D, and F, on March 22nd, 2017). In two of those six rooms (rooms C and D), OXA-48-producing *K. pneumoniae* was still detected in toilet water (May 15th, 2017) after two months of daily disinfection with bleach (from March to May 2017). Finally, we also isolated OXA-48-producing *K. pneumoniae* from the drain pipe between rooms D and E (July 4th, 2017). An overview of toilet/drain water screening results as a function of time is shown in Figure 1. All 12 isolates involved in the outbreak were resistant

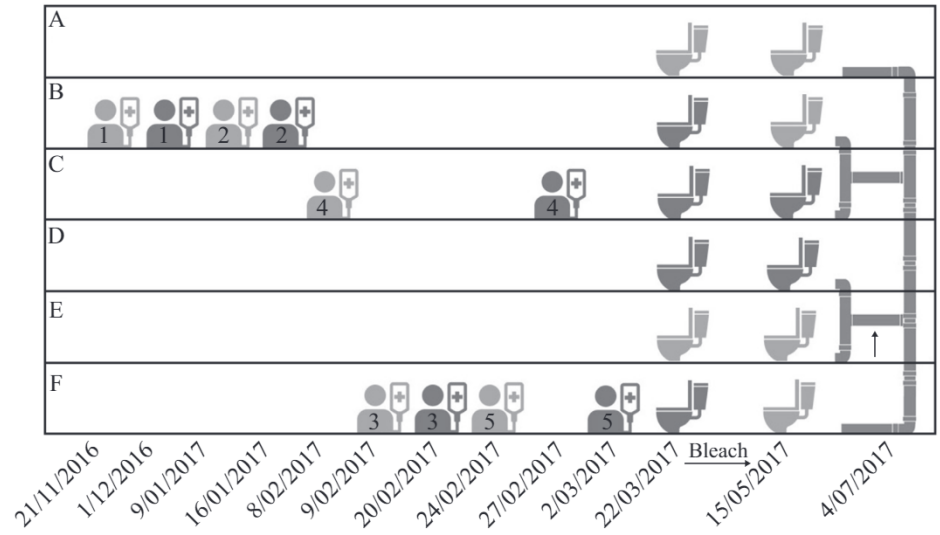

Figure 1. Overview of colonized patients 1–5 residing in rooms A–F of the burn centre and toilet/drain water results as a function of time. Patients are represented in light grey at the time of admission and in dark grey when the first positive rectal screening for OXA-48-producing *Klebsiella pneumoniae* was obtained. Non-contaminated sanitary facilities are displayed in light grey, facilities contaminated with OXA-48-producing *K. pneumoniae* in dark grey. The location where the drain pipe was sampled is indicated with an arrow. As schematically represented, the drain pipes of rooms B and C and of rooms D and E share a proximal connection.

to ampicillin, amoxicillin–clavulanate, cefuroxime, ceftazidime, cefotaxime, temocillin, piperacillin–tazobactam, tobramycin, levofloxacin, and trimethoprim/sulfamethoxazole. All isolates showed susceptibility or were intermediate resistant to meropenem, amikacin, and colimycin.

Whole-genome sequencing and phylogenetic analysis

Whole-genome sequencing was performed on 14 OXA-48-producing *K. pneumoniae* isolates collected over four months, including 12 isolates from the burn centre during the outbreak and two random strains (cultured from colonized patients on different departments in other hospital buildings in the same period). Five outbreak isolates were obtained from rectal swabs (patients residing in rooms B, C, and F), four from toilet water (rooms B, C, D, and F), two from toilet water after two months of daily disinfection with bleach (rooms C and D), and one from drain water (tapped from a drain pipe between rooms D and E). Only the first isolated OXA-48-producing *K. pneumoniae* strain from each patient involved in the outbreak was sequenced by WGS because strain isogenicity is expected between isolates obtained from the same patient. We chose to sequence all isolates detected in toilet water during the first screening in March 2017 to explore the link of toilet water to the outbreak and during the second screening in May 2017 to evaluate the effect of decontamination. Finally, drain water was screened only once on July 4th, 2017, resulting in one isolated OXA-48-producing *K. pneumoniae* strain that we sequenced to explore the potential role of drain water in the outbreak.

All isolates met the criteria of a minimum average base quality score of 30. After de-novo assembly, 196 to 260 contigs per isolate were obtained with an average N50 contig size of 65,778 bp. The assembled genome had an average coverage of 262× to 485× per isolate. More than 99% of the reads were used in the assembly. The MLST typing results showed that all outbreak isolates and the second control isolate were assigned to ST15 (MLST profile: 1-1-1-1-1-1-1); the first control isolate belonged to ST188 (MLST profile: 3-1-1-3-4-28-39). The data revealed isogenicity (<15 allele differences) of all isolates obtained at the burn centre (Figure 2). This suggests that the isolates may have spread between different rooms via the common wastewater drainage system and persisted despite disinfection. The first randomly selected control isolate used was obtained from a patient who was colonized in the dermatology department (different building than burn centre). This strain was not derived from the outbreak clone (3728 allele differences between the isolate and drain water). The second random control isolate was obtained from a patient who became colonized while residing at the geriatric ward, which is situated in a different building than the burn centre. Surprisingly, the strain belonged to the same genetic cluster, suggesting the outbreak to be larger than previously expected.

Patient movements (ward transfers and examinations) of the geriatric patient and other patients colonized with OXA-48-producing *K. pneumoniae* staying in the hospital at the same time were retrospectively assessed. However, no overlap in time nor space was identified between these patients. Due to the extensive CPE screening of burn centre patients, we do not expect to have missed any positive patients in this department. Moreover, the burn centre and geriatric department are located in different hospital buildings. In September 2017, we tested the toilet water of all hospital rooms that were recently (within three months) occupied with CPE-positive patients (data not shown). Of note, all toilets were disinfected with bleach after discharge of CPE-positive patients. In the geriatric department, toilet water of three rooms was tested. Two rooms tested positive for OXA-48-producing *K. pneumoniae*; however, the room where the control patient had been staying tested negative. The discovery that a patient from the geriatric department was also involved in the outbreak led to additional screening of toilet water in this department in December 2019.

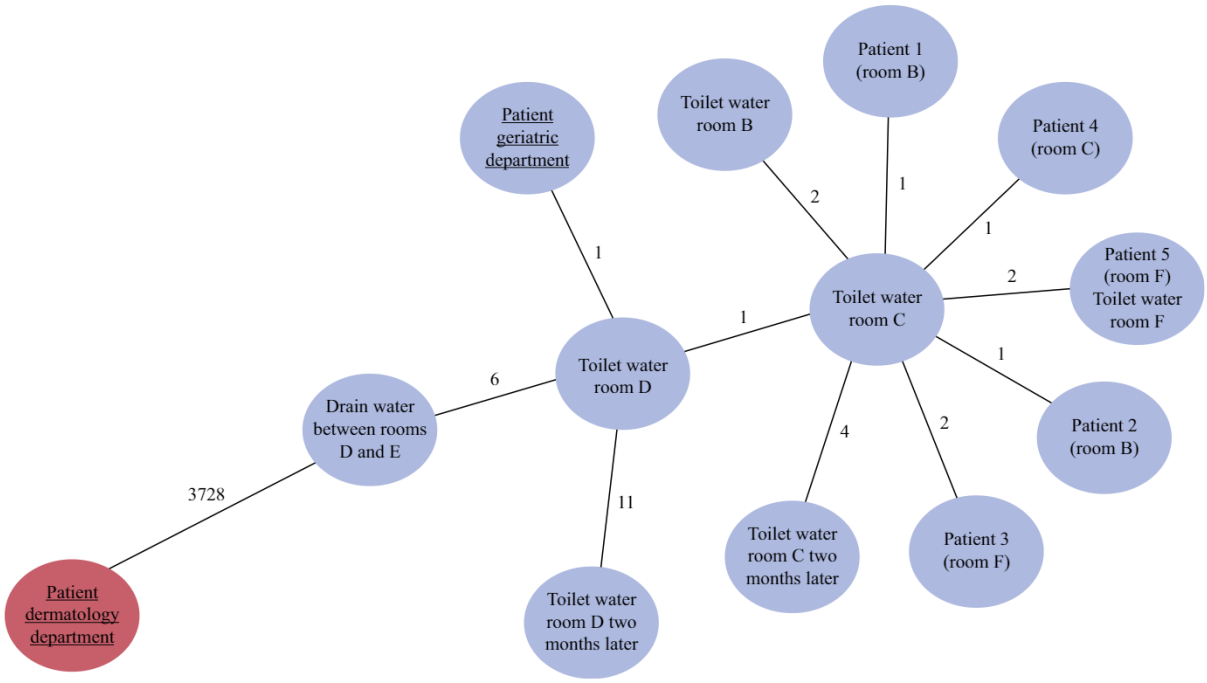

Figure 2. Phylogenetic tree (wgMLST analysis) of *Klebsiella pneumoniae* isolates with isogenic isolates indicated in blue, the non-isogenic isolate in red, control isolates underlined, and number of allele differences presented on the branches.

OXA-48-producing *K. pneumoniae* was found in toilet water of one out of 11 rooms of the geriatric department (data not shown). WGS was not performed to define whether the isolates detected in toilet water of rooms from the geriatric department were derived from the outbreak clone. It may be noted that we regularly observe new CPE colonization in the geriatric department of our hospital. In the period 2017–2019, 12 patients with initial negative rectal screening were colonized with *K. pneumoniae* OXA-48 during hospitalization in this department, starting with the control patient of this study (data not shown).

Infection control interventions

For two months (July 4th, 2017 to September 11th, 2017), 250 mL acetic acid (14°) was added daily to the toilet water of rooms A, B, and C and 250 mL bleach (15°) was added to the toilet water of rooms D, E, and F. Toilet water was sampled and cultured before the start of the experiment, once per week during the experiment, and one week after the experiment. Before the experiment, all three toilets to which bleach had been applied were contaminated, while only one out of three toilets to which acetic acid was added tested positive. Daily disinfection of toilet water with bleach resulted in three out of 24 positive test results of toilet water for carbapenemase-producing *K. pneumoniae* over two months, compared to 10 out of 24 positive test results when applying acetic acid (Figure 3). One week after the last application of acetic acid, the water of all three toilets screened positive for carbapenemase-producing *K. pneumoniae*. By contrast, all the toilets disinfected with bleach tested negative for carbapenemase-producing *K. pneumoniae*. Based on these results, the application of bleach as a disinfectant of toilet water tends to be superior to acetic acid; however, statistical analysis was not performed due to the limited dataset. Neither disinfectant prevented recolonization after discontinuation. No new nosocomial CPE acquisitions were observed in the burn centre after the outbreak while daily disinfecting the toilets with bleach (March 2017 to May 2017) and during the decontamination experiment with bleach versus acetic acid (July to September 2017).

Discussion

In this study, we sequenced OXA-48-producing *K. pneumoniae* isolates obtained from patient rectal swabs as well as toilet and drain water during an outbreak at the burn centre of our hospital. The isogenicity between all isolates from the outbreak confirmed the single origin of the outbreak. Toilets can be contaminated with infectious organisms in an anterograde or retrograde manner. Anterograde contamination may result from rectally colonized patients who use the toilet, potentially leading to the formation of bacterial biofilms. Other ways of anterograde contamination are less likely to occur in the burn centre since strict measures are taken to prevent this. Every room has its own healthcare supplies as well as cleaning material and toilet brush (which is replaced after patient discharge). The medical and logistic staff use maximal barrier precautions by wearing personal protective equipment that is exchanged between the cleaning of different rooms and compliance to this protocol is high. The colonized patients have not been transferred from one room to another. Retrograde contamination may result from a retrograde flow of wastewater. During the outbreak period, several drain pipe obstructions were reported in the burn centre resulting in water reflux to the different toilets. Toilet water of room D tested positive for OXA-48-producing *K. pneumoniae* while no CPE colonized patients had been staying in this room. Since

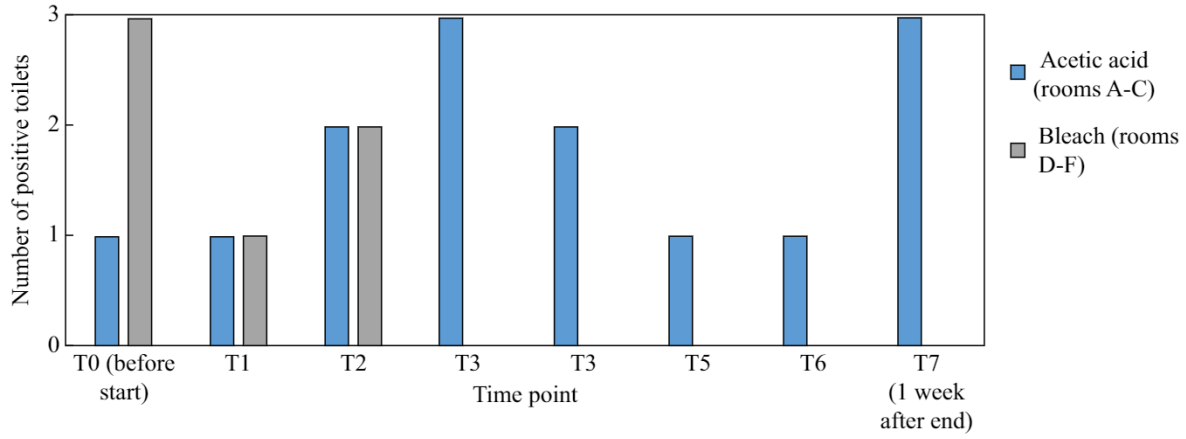

Figure 3. Number of toilets positive for carbapenemase-producing *Klebsiella pneumoniae* during two months' daily disinfection of toilet water with acetic acid versus bleach.

toilet water of the surrounding rooms as well as drain water between rooms D and E also tested positive for this strain, we suggest that toilet drain water may be the source of the outbreak. OXA-48-producing *K. pneumoniae* may have spread between different rooms via the common wastewater plumbing during the outbreak. The isogenicity of strains obtained from toilet water of two rooms before disinfection and after two months of daily disinfection with bleach indicates that this method is insufficient to eradicate the strain in toilet water. Unexpectedly, the data showed that the dimension of the outbreak may have been underestimated since an included isolate from a patient residing at a different ward belonged to the same genetic cluster as the burn centre isolates. There was no link between the patient who stayed at the geriatric department and outbreak isolates.

In our study, we used wgMLST which offers high-resolution pan-genome detection of closely linked outbreak strains. Other comparative genomics approaches for hospital outbreaks include core genome (cg) MLST and single nucleotide polymorphism (SNP) analysis [11,26]. SNP analysis has the highest discriminatory power since this method includes intergenic regions in addition to genes/loci, but is unsuitable for global data sharing and reanalysis [11,26]. However, Miro *et al.* observed an overall correlation between wgMLST, cgMLST, and cgSNP analysis to determine isogenicity of OXA-48-producing *K. pneumoniae* isolates [25]. The cluster allele distances observed for OXA-48-producing *K. pneumoniae* outbreak isolates in our study correspond to those reported by Zhou *et al.* (1–6 allelic differences), except for the isolate obtained from toilet water of room D after two months' disinfection [27].

Since the first report of OXA-48 carbapenemases in *K. pneumoniae* emerging in Turkey in 2001, the enzyme has been extensively reported as a source of nosocomial and community outbreaks worldwide, mainly in the Mediterranean area [6,28]. Within *K. pneumoniae*, *bla*OXA-48-like genes are found predominantly in ST101, ST147, ST395, ST405, ST11, ST14, and ST15 [10,28]. In our study, all OXA-48-producing *K. pneumoniae* outbreak isolates belonged to ST15. *K. pneumoniae* ST15 harbouring *bla*OXA-48 has been disseminated worldwide, notably in Europe, for example in Spain, Belgium, Germany, Finland, and Czech Republic [28]. ST15 *K. pneumoniae* is one of the four major lineages frequently associated with hospital outbreaks in Europe [7]. For example, a Dutch hospital experienced an outbreak of a CTX-M15-producing *K. pneumoniae* ST15 strain closely related to isolates previously found in the USA [13]. Furthermore, outbreaks of KPC-3-producing and VIM-1-producing ST15 *Klebsiella pneumoniae* have been reported in a Portuguese and Spanish hospital respectively [29,30]. By contrast, outbreaks with *K. pneumoniae* ST188, isolated from one control patient in our study, have not been described in the literature. Remarkably, OXA-48-producing *K. pneumoniae* ST15 has been isolated from Austrian wastewater between 2011 and 2012 [28]. The hospital wastewater environment, including toilets and drainage pipes, may be a reservoir for carbapenem-resistant organisms and has been associated with clinical outbreaks in the intensive care setting [17]. Smismans *et al.* were the first to describe the detection of CPE (*Citrobacter freundii* type OXA-48) in toilet bowls and traps in hospital rooms that shared common waste plumbing and subsequent transmission of CPE to four patients in one ward [31]. However, WGS was not performed to prove isogenicity of the detected isolates [31].

Determining effective infection control measures to decontaminate environmental reservoirs may minimize potentially lethal outbreaks [17]. A combination of reinforcement of general infection control measures and chemical disinfection appears to be a successful intervention, as described in the literature [17]. For example, disinfection with peracetic acid successfully eradicated OXA-48-producing *C. freundii* in toilet bowls and traps [31]. In sink drain water, some success with eradicating CPE was reported using bleach or hydrogen peroxide vapour [12,20]. However, decontamination of sink drains with acetic acid is a valuable alternative [32]. In our study, daily application of bleach tended to be superior to acetic acid to disinfect toilet water. However, bleach is not ecologically friendly and did not completely prevent the presence of carbapenemase-producing *K. pneumoniae* in toilet water. The effect of disinfectants is only temporary since biofilms are not disrupted [18]. Self-disinfecting traps appear to be more promising but include a substantial cost [18]. Replacement of colonized water reservoirs and design changes may be required for long-term clearance [17,21]. Further, the installation of covers on hoppers (waste disposal system) demonstrated a decrease in patient acquisitions of

carbapenemase-producing *K. pneumoniae* [33]. Importantly, the rooms of the burn centre have open bathrooms with toilets positioned at a distance of ~3 m from the bed, which may ease the transmission of CPE via toilet plume aerosols. Bioaerosol concentrations produced after toilet flushing have been shown not to differ across time (5 to 15 min) or distance (0.15 to 1 m) from the toilet [23]. Efforts are required to separate the toilet from the rest of the hospital room. In addition, redesign of toilets and wastewater drainage system should be initiated in our hospitals. The main limitation of this study is that WGS was not applied on OXA-48-producing *K. pneumoniae* isolates obtained from other patients or toilets of the geriatric department. Therefore, the dimension of the outbreak may have been underestimated.

In conclusion, the present study of WGS on OXA-48-producing *K. pneumoniae* gives support to the hypothesis of toilet drain water as a source of hospital room-to-room transmission of carbapenemase-producing *K. pneumoniae* and the reported nosocomial outbreak. Thus, WGS serves as an excellent tool to effect hospital infection control decisions and can be used for large-scale monitoring of bacterial outbreaks. Further research is needed to map the actual size of the outbreak in our hospital.

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Conflict of interest statement
None declared.

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