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Assessment of the role of handwash basins in the airborne transmission of multi-drug-resistant organisms in clinical and non-clinical settings

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Summary

Background: While multidrug-resistant organisms (MDRO) transmission in clinical settings is traditionally attributed to contact with contaminated surfaces and medical devices, growing evidence suggests that other elements of the hospital environment, particularly handwash basins, may also act as MDRO reservoirs. However, airborne transmission of MDRO from handwash basins under real-world conditions remains poorly characterized.

Methods: Aerosol and surface sampling were conducted in five inpatient hospital rooms and three non-clinical restrooms during faucet operation and under control conditions. Aerosols were collected using a single-stage Andersen Cascade Impactor and a BioSpot-VIVAS sampler. Drain swab samples were also collected. All samples were cultured on selective and non-selective media, and isolates were identified using MALDI-TOF mass spectrometry, with a focus on carbapenem-resistant bacteria and *Candida* species.

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Conflict of interest statement

The authors declare no conflict of interest.

Results: We detected at least seven MDRO in aerosol samples during faucet operation in the inpatient rooms and restrooms. Types of microbes differed substantially between the two aerosol sampling methods. An overlap of organisms was detected in aerosol and drain swab samples in several rooms.

Conclusions: Handwash basin operation can contribute to aerosolization of viable MDRO, highlighting a potential pathway that necessitates consideration in infection prevention strategies. Moreover, it is necessary to consider multiple aerosol collection methods for a comprehensive assessment of airborne pathogens.

Introduction

Healthcare-associated infections (HAIs) are a major concern in the healthcare sector and are responsible for more than 100,000 deaths and \$45 billion in health-related costs annually in the US[1]. A large portion of HAIs are caused by multidrug-resistant organisms (MDRO), whose contribution is estimated to be >80%[2]. Consequently, extensive efforts have been undertaken to reduce the spread of MDRO in hospitals[3,4].

A key to success in HAI-mitigation efforts is understanding the reservoirs and transmission routes of MDROs. Traditionally, high-touch surfaces, such as bed rails, bedside tables[5], IV pumps[6], and monitor controls[7], as well as invasive medical devices, including catheters[8], feeding tubes[8], endotracheal tubes[9], and ventilators[10], have been considered MDRO reservoirs. They facilitate MDRO transmission through direct and indirect contact between patients and healthcare workers (HCWs), who interact with these reservoirs. Consequently, strategies have been developed and implemented to minimize these reservoirs and prevent the transmission of MDRO through these routes[11,12]. However, these strategies have not been universally effective in achieving their objectives; in many cases, such strategies have failed to reduce the number of HAI cases despite compliance with infection prevention measures by hospitals and HCWs[13–15].

One possible reason for the limited effectiveness of these strategies could be the presence of other major reservoirs and transmission pathways that current strategies do not address. In the past few years, besides high-touch surfaces and medical equipment, several new reservoirs of MDRO, including drains[16], sink traps[17], and drainage pipes[18], have been implicated in transmission. Moreover, the role of other transmission routes, especially airborne transmission, is being increasingly recognized. For example, MDRO have been found in ventilation grilles and HVAC ducts in hospitals[19,20], and aerosols generated by patients through sneezing, coughing, and speaking have been reported to carry pathogens[21–23]. Thus, MDRO may also originate from non-traditional sources and be transported across hospital environments *via* air movement, enabling a wider spread than is possible through direct or indirect contact alone. Moreover, the transmission potential of MDRO could also vary by organism type, as Gram-positive bacteria (GPB) generally tolerate desiccation better than most Gram-negative bacteria (GNB), whereas some Gram-negative organisms show similar environmental resilience[24–26].

Among several possible sources of bioaerosols in hospital rooms, handwash basins have garnered significant attention in recent years[27]. Handwash basins are connected to

both the water supply and wastewater drainage system, which can serve as reservoirs and dissemination pathways for microorganisms[28,29]. Microorganisms associated with handwash basins may originate from incoming water supply, from patient care and staff activities that introduce biological material and nutrients into the sink drain[30], and from retrograde contamination arising elsewhere within the wastewater plumbing network[31]. Such continuous exposure to moisture and nutrient-rich waste streams also promotes persistent biofilm formation within sink drains and traps. Several studies have investigated mechanisms that may contribute to microbial dispersal from handwash basins, including the influence of water flow characteristics on splash and aerosol generation[27], as well as positive air-pressure transients originating from the main body of the wastewater plumbing system that may promote the release of microorganisms from sink traps into the surrounding environment[32]. While at least one study has argued that transmission from contaminated sink drains may occur predominantly through droplet-mediated splash contamination rather than true aerosolization[33], both waterborne and wastewater-associated dispersal events are increasingly recognized as important contributors to healthcare-associated pathogen transmission[34]. Importantly, these transmission pathways are often not readily mitigated by standard infection prevention and control precautions[35], which has prompted some healthcare facilities to adopt “water-free” patient care strategies during persistent or otherwise intractable outbreaks linked to sinks and wastewater systems[36]. Overall, all these findings emphasize the importance of understanding the mechanisms and transmission routes of microbial dispersal from handwash basins to inform effective infection prevention and control strategies in healthcare settings.

However, several questions remain regarding the role of handwash basins in the airborne transmission of MDRO. Most of the current studies investigating this issue have relied on laboratory model handwash basins, with relatively few studies evaluating aerosolization processes in real clinical or non-clinical environments such as hospital patient rooms or public restrooms[37]. Finally, the role of handwash basin design features, including faucet characteristics (e.g., height and flow rate) as well as handwash basin and drain configuration, position, and state of drainage, on splashing intensity and hence bioaerosol generation rate[38–40] has not been thoroughly studied. As a result, although surface-based surveys have extensively characterized MDRO species in hospital environments, significant knowledge gaps remain regarding their prevalence in aerosol form and the contribution of handwash basins to MDRO transmission. Identifying which species are prevalent in aerosols and understanding how they differ from those in handwash basin drains can help determine which MDRO are more likely to spread *via* the aerosol route. Such insights can strengthen existing HAI prevention strategies and inform policies designed to reduce MDRO transmission from diverse sources and reservoirs. Moreover, this knowledge can be applied to disease transmission and health-risk prediction models to better estimate the contribution of handwash basins to airborne diseases in both clinical and non-clinical settings.

Therefore, in this study, we conducted sampling in five inpatient rooms in a hospital and three restrooms in a university academic building to assess the relevance of aerosol-mediated transmission in real-world settings. Overall, we aimed to (i) verify the presence of MDRO in the air of inpatient hospital rooms, and (ii) assess whether water splashing during faucet operation contributes to airborne MDRO dissemination.

Methods

Sampling sites and handwash basin characteristics

Aerosol sampling was conducted between June and September 2025, in three men's public restrooms (denoted as Rooms R01, R02, and R03) in an academic building on the University of Miami campus, and five inpatient rooms (denoted as Rooms A, B, C, D, and E) within an 560-bed tertiary-care academic facility in Miami, Florida. Further details on the sampling methodology are provided in Section S1 of the Supplementary Materials (SM).

In both restrooms and inpatient rooms, the handwash basins were located at an average height of 3 ft above the floor. The handwash basins in the public restrooms differed in design from those in the inpatient rooms, with the public restrooms featuring undermount basins made of ceramic and the inpatient rooms having wall-mount type basins made primarily of ceramic, except for two room in which the basin was made of stainless steel. The photos of the different handwash basin designs used in the various rooms is provided in Figure S2 of the SM. The spout of the faucets in the public restrooms was approximately 0.5 ft from the drain of the basin, whereas in the inpatient rooms, the distance between the spout and the drain was approximately 1 ft. To further characterize and differentiate the basins of the two types of rooms, we measured the faucet discharge rate by turning the faucet on to full capacity and using a stopwatch to record the time required to collect a fixed volume (~150 mL) of water. For each faucet, three replicate measurements were taken.

Sampling conditions

In each room, aerosol sampling was conducted using two instruments: SACI (Aerotech 6, EMLab P&K, USA) operating at a flow rate of 28 L/min and VIVAS (Model 300, Aerosol Devices Inc., USA) operating at a flow rate of 8 L/min. Further details on the principle of operation of the two instruments are provided in Section S2 of SM. Note, in this study, aerosols were defined according to the aerosol science convention as any solid or liquid particle suspended in air[41]. Because both samplers used in this study actively draw air into their inlets, they preferentially sample particles that remain suspended in the airflow. Larger splash droplets typically follow ballistic trajectories and deposit rapidly on nearby surfaces and therefore have lower aspiration efficiency than smaller airborne particles[42–44]. Therefore, the aerosols sampled in our study could be of a wide size range.

Aerosol samples were collected using SACI and VIVAS simultaneously, under two conditions: first with the faucet off (control) and next with the faucet turned on to full capacity (F_{on}). During the collection of the F_{on} samples, the faucet was operated continuously at its maximum flow rate for the entire sampling duration. The control samples were used to represent the background airborne microbes and were collected before the faucet was turned on, while the F_{on} samples collected background aerosols and aerosols generated due to the water splashing in the handwash basin during faucet operation. In the public restrooms, VIVAS samples were collected into 3 mL Tryptic Soy Broth (TSB), and SACI samples were collected on REMEL™ Contact I Blood Agar plates (BAP; Thermo Scientific, Cat. No. R111007, USA). For each restroom, one control and one F_{on} sample were collected using the VIVAS with a sampling duration of 30 minutes,

while two control and two consecutive F_{on} samples of 15 minutes each were collected using the SACI. In the inpatient rooms, VIVAS samples were similarly collected using 3 mL TSB; however, the SACI samples were collected using BAP, BBL™ CHROMagar™ *Candida* (Beckton Dickinson, Cat. no. B4354093, Maryland, USA), and HardyCHROM™ CRE (Hardy Diagnostics, Cat. No. G323, California, USA) agar plates. Unlike in the public restrooms, the sampling duration in inpatient rooms was 1 hour for VIVAS and 20 minutes for SACI, with three consecutive SACI samples collected per room. In all rooms, the swab samples were collected from the drain mouth grill of each handwash basin using sterile nylon swabs (Nasal Flocked Swab, part no. 96000, Miraclean Technology Co, Ltd., China) after collecting the control samples and prior to collecting F_{on} samples. The swabs were then immersed in 9 mL of Tryptic Soy broth in a glass vial. A detailed flow chart showing the sequence of sampling in public restrooms and inpatient rooms used in our study is provided in Figure S1 of SM.

Sample analyses

Following sampling, all agar plates used with the SACI were incubated at 37 °C for 48 hours. Samples collected using VIVAS and swab samples suspended in TSB were vortexed for 5 minutes and incubated at 37 °C for 24 hours. After incubation, the broth was vortexed again for 5 minutes and sub-cultured onto BAP, MacConkey agar, Sabouraud dextrose agar, HardyCHROM™ CRE, and BBL™ CHROMagar™ *Candida* agar plates, followed by an additional incubation for 48 hours at 37 °C. After 48 hours, presumptive identification was performed according to the manufacturer's recommendations based on colony color and growth characteristics.

HardyCHROM™ CRE was used to screen for Carbapenem-resistant organisms. Unlike inpatient rooms where SACI samples were collected on multiple agar media plates, restroom SACI samples were collected only on BAP to recover gram-negative bacilli (GNB) and mold. Therefore, presumptive colonies from these BAP samples were further isolated onto MacConkey agar, Sabouraud dextrose agar for confirmation and characterization. Preliminary assessment of SACI, VIVAS, or swab samples collected in the public restrooms did not indicate any presence of *Candida* species, so no further subculturing on BBL™ CHROMagar™ *Candida* agar plates was performed for these samples.

After the preliminary identification and confirmation of various groups of microbes for all samples, suspected carbapenem-resistant bacteria and *Candida* species were subsequently identified to the species level using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF VITEK® MS PRIME). Overall, our study focused on the identification of two groups of MDRO: *C. auris* and carbapenem-resistant GNB. Although Gram-positive bacteria (GPB) and mold were isolated, they were not further speciated in this study. Note, no separate microbiological analysis of faucet water was performed as part of this study because routine monitoring of the hospital water supply to handwash basins used in our study indicated very low microbial loads, including negligible levels of *Pseudomonas aeruginosa* (Figure S3 in the SM).

Results

Faucet discharge rate

The faucet discharge rates measured in different rooms where sampling was conducted are summarized in Figure 1. The discharge rates varied across the rooms, with the average discharge rate in inpatient rooms substantially higher than that of the public restrooms. Discharge rates in inpatient rooms ranged from 74 to 135 mL/s, whereas restroom discharge rates ranged from 19 to 45 mL/s, indicating that restroom flow rates were approximately 3.5 times lower than those of inpatient rooms.

Organisms in Control samples

The microorganisms detected in the control aerosol samples collected using SACI and VIVAS are presented in Figure 2. The microbial composition of the control samples varied by room. In the public restrooms, control samples consisted primarily of mold and GPB, and no MDRO were detected. Similarly, no MDRO were identified in most inpatient room controls, with the exception of Room C, where *Burkholderia contaminans*, a carbapenem-resistant GNB[45], was detected along with *Candida sp* (Not *C.auris*). *Penicillium sp* was also detected in Rooms D and E, while mixed GPB were observed in Rooms A and B. Notably, despite SACI and VIVAS collecting samples simultaneously and at similar locations in the room, the two instruments recovered different microbial profiles. For instance, in Room C, unidentified *Candida* spp were detected in SACI samples but not in VIVAS samples, whereas *Burkholderia contaminans* was recovered from VIVAS samples but not from SACI samples. Overall, the control samples chiefly consisted of mold and GPB.

Contribution of handwash basin to bioaerosols

The overlap of organisms detected in samples collected during faucet operation (F_{on}) using different sampling methods across various rooms is shown in Figure 3. In the public restrooms, swab samples revealed the presence of carbapenem-resistant *Aeromonas* spp.(in R01, R02, R03). There were also other organisms present in the swab samples from the public restrooms, including *Enterococcus* spp. and mold. Among inpatient rooms, swab samples from handwash basin drains showed substantial variability and significant presence of MDRO including carbapenem-resistant *Enterobacter cloacae* complex, a well-known MDRO and a cause of HAIs[46], carbapenem-resistant *Klebsiella pneumoniae*, carbapenem-resistant *Pseudomonas aeruginosa* (an emerging MDRO[47]), and *Stenotrophomonas maltophilia*.

Similar to swab samples, aerosol samples collected using SACI and VIVAS with the faucet turned on to full capacity (F_{on}), demonstrated the presence of MDRO frequently matching those from the drain swab, although they differed markedly in microbial composition, both within and across rooms. In restrooms, SACI samples largely contained mixed GPB, with mold and one MDRO, *Aeromonas hydrophila*, detected in R02. Unlike SACI samples, mixed GPB were not detected in any VIVAS samples collected from restrooms, and no MDROs were detected in VIVAS samples from R03. Instead, *Pantoea agglomerans*, a GNB with strains known to be carbapenem-resistant[48] was detected in R01. Unlike SACI

samples, no mold was detected in any of the VIVAS samples collected in the restrooms. In inpatient rooms, filamentous mold was detected in room B, and mixed GPB were detected in room C in SACI samples. Only one MDRO, *Stenotrophomonas maltophilia*, was detected in SACI samples in room B. In contrast, VIVAS samples captured a broader range of MDROs during faucet operation compared to SACI samples. We detected organisms such as *Pantoea dispersa* in room A, *Leclercia adecarboxylata*, and *Klebsiella pneumoniae* in room B, *Acinetobacter radioresistens* and *Pseudomonas aeruginosa* in Room C, and *Stenotrophomonas maltophilia* in Room D. No MDRO was detected in VIVAS samples collected in room E. Notably, across the study, overlap between MDRO detected in swab samples and those recovered in aerosol samples during faucet operation was observed in three of the five inpatient rooms (*Klebsiella pneumoniae* in room B, *Pseudomonas aeruginosa* in room C, and *Stenotrophomonas maltophilia* in room D) primarily in VIVAS samples. In contrast, among the restrooms, clear overlap of one MDRO was observed between swab samples and SACI samples in one of the three rooms (*Aeromonas hydrophila* in R02). Importantly, these overlapping organisms were not detected in corresponding VIVAS and SACI control samples, suggesting that faucet-induced water splashing may disrupt biofilms and facilitate the aerosolization of pathogenic microorganisms in both clinical and non-clinical settings.

Discussion

Our results demonstrate the potential for transmission of MDRO through aerosols generated from faucet-induced water splashing in handwash basins in both clinical and non-clinical settings. In inpatient rooms, we detected at least seven MDRO in aerosol samples, including carbapenem-resistant GNB (*Pseudomonas aeruginosa*, *Pantoea dispersa*, *Stenotrophomonas maltophilia*, *Burkholderia contaminans*, *Acinetobacter radioresistens*, *Leclercia adecarboxylata*, and *Klebsiella pneumoniae*). Among these, carbapenem-resistant *Klebsiella pneumoniae*, carbapenem-resistant *Pseudomonas aeruginosa*, and *Stenotrophomonas maltophilia*, are well-established causes of hospital- and community-acquired pneumonia[47,49,50], while *Burkholderia contaminans* has been associated with chronic pulmonary infections in patients with cystic fibrosis[51]. Although *Acinetobacter radioresistens*, *Leclercia adecarboxylata*, and *Pantoea dispersa* are less frequently implicated in clinical diseases, they are recognized opportunistic pathogens capable of causing infections in immunocompromised individuals[52–54]. Beyond respiratory illnesses, all of these organisms have the potential to contaminate the patient's environment, and be transferred to the patient colonizing or causing a range of other infections, including urinary tract, bloodstream, wound, and skin infections. Importantly, the overlap between organisms detected in swab samples and aerosol samples during faucet operation observed in our study, particularly in inpatient rooms, supports the hypothesis that water splashing in handwash basins can aerosolize clinically relevant microbes which might be found in the drains of handwash basins. Similar overlap observed in one restroom (R02) highlights that handwash basins as a bioaerosol source may also be relevant in non-clinical environments, as shown by the detection of *Aeromonas hydrophila*, a pathogen capable of infecting both healthy and immunocompromised individuals[55].

Significant differences were observed in the microbial communities recovered by the two aerosol collection methods, SACI and VIVAS, with no overlap in organisms detected by the two samplers in any room, except for a mixed population of GPB in inpatient room B. These differences likely reflect variations in biological collection efficiency and the influence of collection media (liquid versus agar) used by the two samplers. Our findings align with and reinforce prior evidence that captured airborne microbial communities can vary substantially depending on the aerosol sampling method employed[56]. Previous studies have shown that microbial viability can vary widely across aerosol sampling techniques[57–59]; because our analysis relied on culture-based methods for preliminary identification, viability and cultivability were critical determinants of recovery. VIVAS has been reported to better conserve microbial viability compared with the Andersen impactor[60]. However, its lower sampling flow rate (8 L/min vs. 28 L/min for SACI) may result in a lower number of microorganisms collected, especially at low airborne concentrations. Since both samplers were operated simultaneously, the combined effects of sampling volume, collection mechanism, and microbial viability influenced which organisms were recovered. Therefore, our results demonstrate the limitation of relying on a single aerosol sampling method to characterize airborne microbiota. Additional factors, including basin design, basin material, use of aerators, and faucet discharge rate, may further influence bioaerosol generation. Notably, faucet flow rates in inpatient rooms were substantially higher than those in restrooms (Figure 1), which may have contributed to differences in aerosolization potential of the organisms. Another key observation that requires further investigation is the detection of organisms exclusively in the F_{on} aerosol samples, despite both negative background aerosols and negative drain swabs. This could possibly be due to the presence of the primary reservoir of these organisms in other parts of the handwash basin, such as the faucet aerator or spout, internal fixture plumbing, wastewater system, or the incoming water pathway and the water exiting from the faucet itself, rather than the drain biofilm sampled in our study. Faucet operation can mobilize low-abundance organisms intermittently through biofilm detachment from some of these reservoirs and generate aerosols *via* splash mechanisms that are absent when the faucet is off. Future studies should investigate all these factors thoroughly for a better understanding of the various reservoirs of MDRO. One more important factor that needs to be considered is that previous studies have shown that splash-generated droplets can play a dominant role in microbial dispersal from contaminated sink traps. In this study, aerosols were defined according to the aerosol science convention as any solid or liquid particle suspended in air, which includes particles that may be classified as droplets in infection-control literature. Because size-segregated aerosol sampling was not performed, it is difficult to determine which particle size ranges contribute most to MDRO dispersal. Our findings, therefore, suggest that both droplet- and aerosol-mediated pathways may occur during faucet operation.

Note, our study also had some limitations that must be carefully considered when interpreting the results. First, we did not assess the full diversity of microorganisms present, potentially underestimating the overlap between surface and airborne samples. Our analyses were qualitative rather than quantitative, and microbial identification focused primarily on selected GNB, and GPB were not speciated. Therefore, there might be other pathogenic organisms in the swab samples that have also been aerosolized due to water splashing and

hence pose a potential risk. Moreover, all culture plates were incubated at 37 °C for 48 hours following CDC guidelines, which may not be optimal for all organisms[61]. As a result, some slow-growing bacteria, such as *Stenotrophomonas maltophilia*, that grow better at lower temperatures may have been under-detected[62]. Additionally, since our study focused on qualitative detection of microorganisms rather than quantitative measurement of airborne microbial concentrations, the airborne bioburden and the relative contribution of aerosol versus droplet-mediated transmission could not be assessed and should be addressed in future studies using quantitative and size-segregated aerosol sampling approaches. Second, the sampling flow rates and sampling duration of SACI and VIVAS differed, and thus, a direct comparison of sampler performance in capturing various airborne MDRO was not possible. Third, in our analyses, we used MALDI-ToF, which can provide a species-level identification of the microbes but requires advanced analyses to identify the strains. Moreover, MALDI-ToF cannot reliably discriminate between closely related species[63]. Therefore, although several MDROs were identified in our study, we cannot comment on their pathogenicity. A detailed assessment of the various strains in handwash basins and aerosol samples using advanced techniques such as whole genome sequencing should be conducted in future studies to identify the significance of handwash basins as a source of pathogenic MDRO in clinical and non-clinical settings. The fourth limitation of this study is that faucets were operated continuously at maximum flow for extended periods (30–60 minutes) to enable sufficient air sampling, which does not reflect typical clinical handwashing durations. Therefore, the aerosol generation observed in this study may not fully represent real-world conditions during routine handwash basin use. Fifth, the temporal resolution of sample collection is another major limitation. Using the current methods, it is unclear if the samples were mainly collected at the beginning of the sampling activity or throughout the sampling event. Time-resolved sampling is necessary to understand the kinetics of bioaerosol generation through handwash basin operation. Lastly, although this study focused on MDRO, antibiotic-susceptible microorganisms may also be transmitted through the same environmental pathways and should be considered in future investigations[64]. Future studies should therefore evaluate both resistant and susceptible microorganisms to better understand microbial dispersal from handwash basins and its implications for infection control. Despite these limitations, our findings provide evidence that handwash basin operation may serve as a source of airborne dissemination of opportunistic and antibiotic-resistant pathogens, and it is important to consider deploying more than one aerosol sampling method, with implications of airborne transmission in infection prevention and control.

Conclusion

In this study, we demonstrated that the operation of handwash basins can generate bioaerosols containing MDRO in both clinical and non-clinical settings. Through differences in microbial capture between two aerosol sampling methods, we also demonstrated that a single sampling method may not fully capture the entire airborne microbial spectrum. Therefore, aerosol sampling in hospitals cannot rely on a single method to monitor the airborne transmission of MDRO. Furthermore, our findings provide evidence that faucet flow rate could potentially influence bioaerosol generation. Collectively, our study shows the

need for further quantitative and mechanistic investigations to better understand handwash basin-associated bioaerosol formation and to inform infection prevention strategies aimed at reducing airborne spread of MDRO-driven HAIs in healthcare environments.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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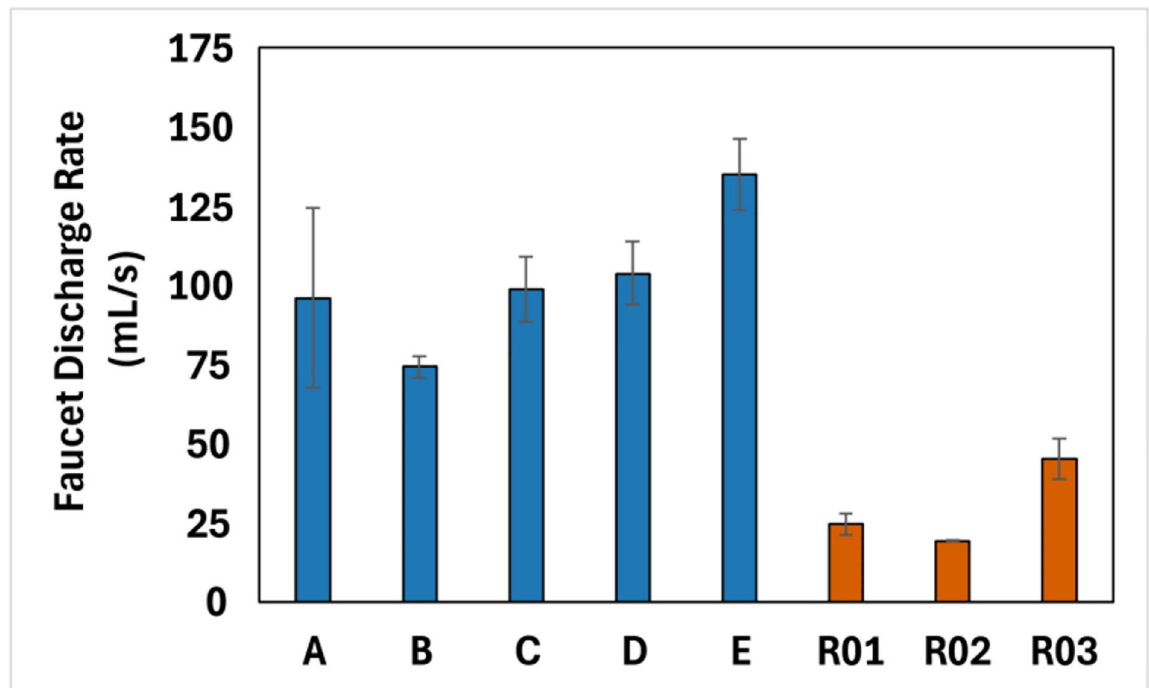


Figure 1.
Faucet discharge rate for handwash basins in different rooms where aerosol sampling was conducted in our study. Rooms A, B, C, D, and E, are inpatient rooms, R01, R02, and R03, are public restrooms. Error bar represents one standard deviation.

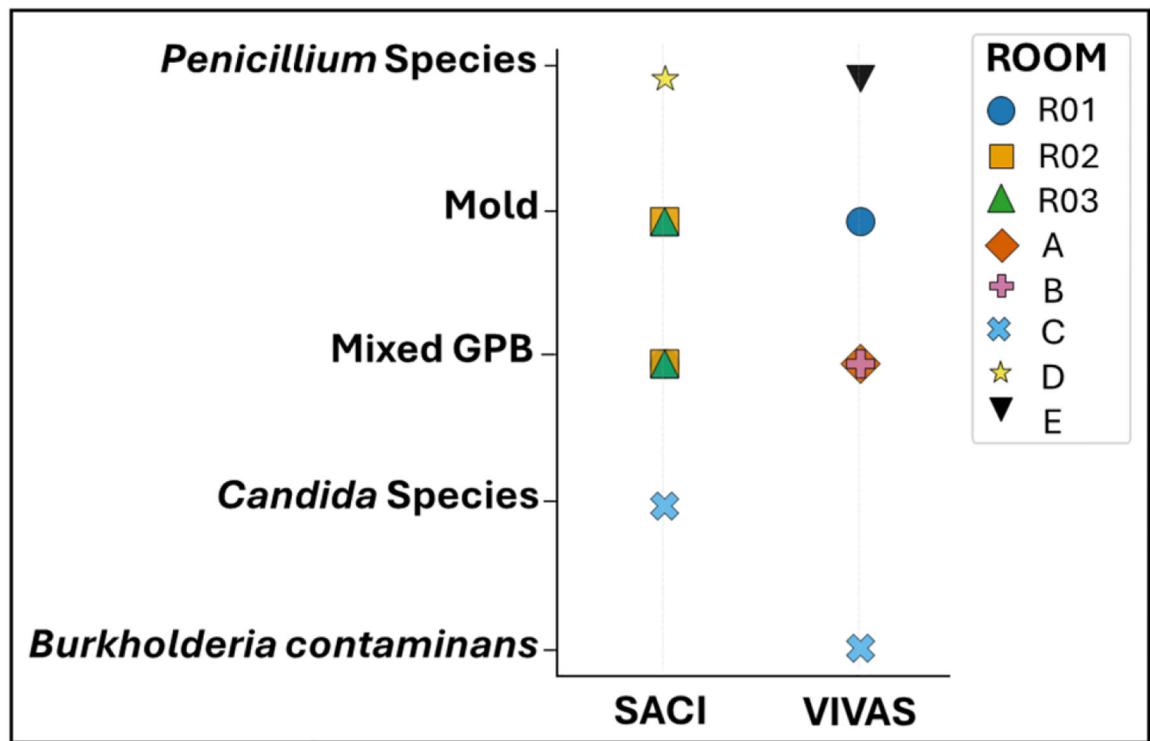


Figure 2.

Microbes found in control samples collected by SACI and VIVAS in various rooms. Rooms A, B, C, D, and E, are inpatient rooms, R01, R02, and R03, are restrooms. Mold represents non-*Penicillium* mold genera. Key: GPB = Gram-positive bacteria

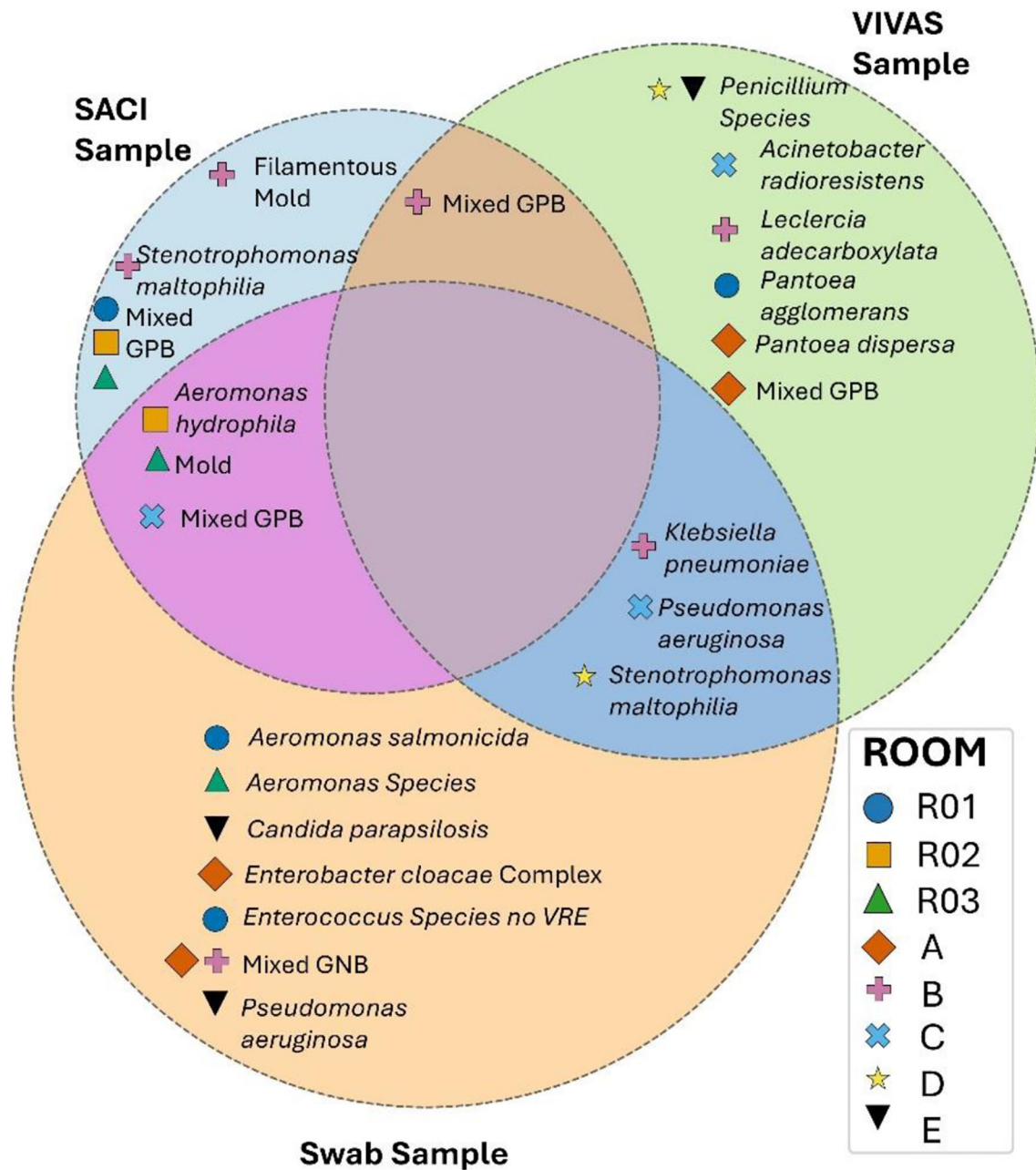


Figure 3.

Venn diagram showing the overlap of organisms among swab samples and aerosol samples collected during the operation of the faucet, using different sampling methods. Rooms A, B, C, D, and E, are inpatient rooms, R01, R02, and R03, are restrooms. Key: VRE = Vancomycin-Resistant *Enterococci*; GPB = Gram-positive bacteria; GNB = Gram-negative bacilli.