Multidrug-resistant bacteria isolated from cell phones in five intensive care units: Exploratory dispersion analysis

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Abstract

Introduction **Cell phones are susceptible to bacterial contamination. The aim of this study was to characterize the bacterial isolates and to explore their dispersion in five Intensive Care Units (ICUs) over the time.**

Methods **We performed a secondary analysis of non-fermenting Gram-negative bacteria and Gram-positive cocci isolated from a 5-month observational cohort study developed among health care workers' cell phones in five ICUs. Cell phones were sampled using a swab every 15 days. Antimicrobial resistance was determined by the minimum inhibitory concentration method. We constructed resistance phenotypes to group the isolates according to species and antimicrobial resistance pattern to explore dispersion through time.**

Results **A total of 35 *P. aeruginosa*, 16 *Acinetobacter* spp., 30 *S. aureus* and 26 *Enterococcus* spp. were isolated from 491 phone samples. Multidrug resistance was 2.9% for *P. aeruginosa*, 31.3% for *Acinetobacter* spp., 46.7% for *S. aureus* and 80.8% for *Enterococcus* spp. The resistance to methicillin in *S. aureus* and to vancomycin in *Enterococcus* spp. was 26.7% and 42.3%, respectively. We did not observe distribution patterns or clusters over the time for *P. aeruginosa*, *Acinetobacter* spp. and *Enterococcus* spp. isolates. All the *S. aureus* isolates grouped into eight phenotypes. Interestingly, we observed *S. aureus* isolates with the same phenotype in consecutive and separate sampling dates in the same cell phone.**

Conclusion **Cell phones are contaminated with highly harmful bacteria and potentially can maintain them for prolonged periods of time. These devices could be considered as a potential source of nosocomial infections in ICUs.**

Keywords **Fomites, cell phones, drug resistance, bacterial, intensive care units**

Introduction

Non-fermenting Gram-negative bacteria (NFGNB) such as *Pseudomonas aeruginosa* and *Acinetobacter* species, as well as Gram-positive cocci (GPC) like *Staphylococcus aureus* and *Enterococcus* species, are human pathogens that frequently cause nosocomial infections in developing countries.[1-4] These pathogens have been isolated from the environment and fomites in critical care settings.[5-11]

Cell phones are frequently used by health care workers during working hours and are not properly disinfected.[6,12] Cell phones are susceptible to contamination by bacterial pathogens and could be involved in nosocomial transmission.[10,13,14] Therefore, cell phones probably represent a constant and mobile source

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of infection, for hospitalized patients in critical care settings due to their portability and frequent use by providers.

From February to June 2012, we enrolled 114 health care workers' cell phones in three Pediatric and two Neonatology Intensive Care Units (ICUs) at three Peruvian Hospitals to investigate bacterial contamination.[12] We isolated multiple NFGNB and GPC during the study period. Due to high levels of antimicrobial resistance observed in the screening method based on the Kirby-Bauer disk diffusion susceptibility test, we characterized the NFGNB and GPC using the minimum inhibitory concentration (MIC) method. The objective of this study was to describe the antimicrobial resistance profiles of NFGNB and GPC isolated from cell phones and to explore their dispersion through time.

Methods

Bacterial isolation and identification

The trained study personnel collected the samples by swabbing the phone every 15 days as previously described.[12] Each swab was placed in a tube with 3 mL of trypticase soy broth and incubated aerobically for 18-24 hours at 35 °C. Then, swabs were plated to MacConkey, Mannitol salt and Blood agar. NFGNB were characterized using standard biochemical and enzymatic microbiologic procedures. The *S. aureus* isolates were characterized using the coagulase test and confirmed by a genus-specific PCR assay that targets a fragment of the 16S rRNA gene.[15] *Enterococcus* spp. isolates were classified using the catalase test and the esculin degradation on Bile esculin agar.

Antimicrobial resistance and virulence genes

Antimicrobial resistance was determined by the MIC method using the VITEK 2 automated system (bioMérieux Inc., Durham, NC, USA). The antibiotic susceptibility testing was done using the AST-GP67 and AST-N249 VITEK cards (bioMérieux Inc.) for CGP and NFGNB, respectively. Results for penicillin, β-lactam inhibitor combination, glycopeptides, aminoglycoside, macrolides, fluoroquinolones, lincosamides, folate pathway inhibitors, phenicols, ansamycins, oxazolidinones, tetracycline, cephems, monobactams, carbapenems and lipopeptides were interpreted according to the breakpoints described in the Clinical Laboratory Standards Institute' guideline[16] and results emitted by the VITEK 2. ATCC strains were used for quality control for the susceptibility testing.

Methicillin-resistant *S. aureus* (MRSA) isolates were screened using cefoxitin, and confirmed by a PCR that targets the *mecA* gene.[15] Additionally, all *S. aureus* isolates were screened for the erythromycin-induced resistance to clindamycin and for the Panton-Valentine leukocidin (PVL) production.[15,17]

Statistical analysis

Antimicrobial resistance was analyzed as a binary categorical variable, considering the intermediate-level resistance as resistant. Multidrug resistance (MDR) was defined as resistance to ≥1 agent in ≥3 different antimicrobial categories.[18] Also, all the MRSA isolates were considered as MDR according to a proposed standard definition.[18] The Chi-square test or Fisher's exact test was used for the analysis of resistant status between described species of NFGNB and GPC, as well as among MRSA and non-MRSA, and vancomycin-resistant enterococci (VRE) and non-VRE. Resistance to at least one agent was sufficient to be resistant to the antimicrobial category. Then, the Mann-Whitney U test was used to evaluate the resistance to antimicrobial categories. The resistance phenotypes were constructed using aggregated resistance information according to MIC results. We explored the dispersion of phenotypes through time (sampling dates) according to study sites (ICUs and hospitals). Data analysis was performed in Stata v.14.0 (StataCorp, College Station, TX, USA) considering $p<0.05$ as significant.

Results

A total of 35 *P. aeruginosa*, 16 *Acinetobacter* spp., 30 *S. aureus* and 26 *Enterococcus* spp. were isolated from 491 phone samples of ICU health care workers.

Antimicrobial resistance of non-fermenting Gram-negative bacteria (NFGNB)

A percentage of 12.5% (2/16) of the *Acinetobacter* spp. isolates were resistant to trimethoprim-sulfamethoxazole. The observed resistance to piperacillin, piperacillin/tazobactam, netilmicin, fluoroquinolones, cephems, aztreonam and imipenem was more frequent and associated to *Acinetobacter* spp. isolates (Table 1), in comparison to *P. aeruginosa* isolates. The frequency of MDR *Acinetobacter* spp. isolates was higher than that of *P. aeruginosa* isolates (p=0.009, Table 1). Nearly all the *P. aeruginosa* isolates (33/35, 94.3%) were susceptible to all antimicrobials tested in this study. Additionally, all the NFGNB were susceptible to colistin and to the aminoglycosides gentamicin, tobramycin and amikacin.

Table 1. Antibiotic resistance rates of non-fermenting Gram-negative bacteria isolated from health care workers' cell phones

Resistance to agent		*Pseudomonas aeruginosa* (n=35)	*Acinetobacter* spp. (n=16)	p-value*
Piperacillin		5.7 (2/35)	31.3 (5/16)	0.025
Piperacillin-tazobactam		2.9 (1/35)	31.3 (5/16)	0.009
Ticarcillin-clavulanic acid		5.7 (2/35)	NT	-
Netilmicin		0.0 (0/35)	18.8 (3/16)	0.027
Ciprofloxacin		0.0 (0/35)	31.3 (5/16)	0.002
Levofloxacin		0.0 (0/35)	31.3 (5/16)	0.002
Ceftazidime		2.9 (1/35)	31.3 (5/16)	0.009
Cefepime		2.9 (1/35)	31.3 (5/16)	0.009
Aztreonam		2.9 (1/35)	75.0 (12/16)	<0.001
Imipenem		0.0 (0/35)	25.0 (4/16)	0.007
MDR	Yes	2.9 (1/35)	31.3 (5/16)	0.009
Resistance to categories	Median (min-max)	1 (1-4)	1 (0-8)[€]	0.767[¥]

MDR – multidrug resistance; **NA** – not applicable; **NT** – not tested.

Note: Values are % (n/N). *p-values calculated using the Fisher's exact test. [€]One *Acinetobacter* spp. was resistant to all the antimicrobial categories listed plus the folate pathway inhibitor, resulting in resistance to 8 categories. [¥]p-value calculated using the Mann Whitney test, effect size = 0.041

Antimicrobial resistance of Gram-positive cocci (GPC)

The high-level aminoglycoside resistance in *Enterococcus* spp. was 30.8% (8/26) to gentamicin and 3.9% (1/26) to streptomycin. The resistance to tetracycline was 73.1% (19/26), and 30.8% (8/26) for linezolid. The resistance to vancomycin, erythromycin, chloramphenicol, rifampicin and linezolid was higher in *Enterococcus* spp. than in *S. aureus* (Table 2). Also, the frequency of MDR *Enterococcus* spp. was statistically higher compared to that of MDR *S. aureus* (p=0.009, Table 2). The resistance to vancomycin in *Enterococcus* spp. was 42.3% (11/26) and also associated with greater resistance to ciprofloxacin (p=0.045), rifampicin (p=0.010) and tetracycline (p=0.010).

The resistance to gentamicin in *S. aureus* was 10.0% (3/30) and 46.7% (14/30) to clindamycin. Of the 30 *S. aureus* isolates, 8 (26.7%) were classified as MRSA. The methicillin-resistant status was associated to an increased resistance to erythromycin (p=0.001), ciprofloxacin (p<0.001), clindamycin (p<0.001) and rifampicin (p=0.019). Nearly half of the *S. aureus* isolates encoded erythromycin-induced resistance to clindamycin (12/30, 40.0%). Additionally, all *S. aureus* were susceptible to trimethoprim-sulfamethoxazole. No PVL-producers were found.

Resistance phenotypes distribution

The 35 *P. aeruginosa*, 16 *Acinetobacter* spp. and 26 *Enterococcus* spp. were grouped into 3, 13 and 23 phenotypes, respectively. Neither clusters nor consecutive bacterial isolations with the same phenotype were observed in the same cell phone during the follow-up for those bacteria.

All *S. aureus* were grouped into 8 phenotypes (Table 3) and isolated through the study period (Table 4). The R1 phenotype was isolated in multiple sampling dates (Table 4), from all hospitals (Table 3) and even at consecutive dates in the same cell phone (code: AN015, Table 4). Phenotypes R2b, R4 and R5 were MRSA strains and were exclusively isolated at hospital A (Table 3). The R4 phenotype was isolated four times

Table 2. Antibiotic resistance rates of Gram-positive cocci isolated from health care workers' cell phones

Resistance to category/agent		*Staphylococcus aureus* (n=30)	*Enterococcus* spp. (n=26)	χ^2	p-value*
Penicillin		93.3 (28/30)	30.8 (8/26)	23.8	<0.001
Vancomycin		0.0 (0/30)	42.3 (11/26)	15.8	<0.001
Erythromycin		40.0 (12/30)	92.3 (24/26)	16.6	<0.001
Ciprofloxacin		23.3 (7/30)	46.2 (12/26)	3.24	0.072
Chloramphenicol		0.0 (0/30)	15.4 (4/26)	4.97	0.026
Rifampicin		13.3 (4/30)	73.1 (19/26)	20.5	<0.001
Linezolid		0.0 (0/30)	30.8 (8/26)	10.8	0.001
MDR	Yes	46.7 (14/30)[€]	80.8 (21/26)	6.91	0.009
Resistance to categories	median (min-max)	2 (0-5)[¥]	5 (1-7)	–	<0.001[£]

MDR – multidrug resistance
Note: Values are % (n/N).
*All p-values were calculated using the Chi^2 test.
[€]8 MRSA isolates + 6 non-MRSA that were MDR.
[¥]Includes all the antimicrobial categories listed plus aminoglycoside (gentamicin) and lincosamides (clindamycin).
[£]p-value calculated using the Mann Whitney test, effect size = 0.526.

Table 3. Resistance phenotypes of *S. aureus* isolates

Phenotype*	n (%)	PEN	GEN	RIF	CIP	CLI	ERY	MRSA	D-test	Hospital A (n=18)	Hospital B (n=7)	Hospital C (n=5)
S	2 (6.7)	S	S	S	S	S	S	-	-	.	.	X
R1	11 (36.7)	R	S	S	S	S	S	-	-	X	X	X
R2a	3 (10.0)	R	R	S	S	S	S	-	-	X	X	.
R2b	1 (3.3)	R	S	S	S	R	S	+	-	X	.	
R3a	5 (16.7)	R	S	S	S	R	R	-	+	X	.	X
R3b	1 (3.3)	R	S	R	S	R	S	-	-	.	X	.
R4	4 (13.3)	R	S	S	R	R	R	+	+	X	.	.
R5	3 (10.0)	R	S	R	R	R	R	+	+	X	.	.

CIP – ciprofloxacin; **CLI** – clindamycin; **D-test** – erythromycin-induced resistance to clindamycin test; **ERY** – erythromycin; **GEN** – gentamicin; **MRSA** – methicillin-resistant *S. aureus*; **PEN** – penicillin; **RIF** – rifampicin; **R** – resistant; **S** – susceptible; **"-"** – negative; **"+"** – positive; **"."** – no *S. aureus* isolation; **"X"** – *S. aureus* isolation.
*Only antimicrobial agents to which *S. aureus* was resistant.

from two cell phones (codes: AP010 and AN003, Table 4) in two different sampling dates spaced at least 75 days.

Discussion

We isolated resistant *P. aeruginosa*, *Acinetobacter* spp., *S. aureus* and *Enterococcus* spp. from cell phones used by health care workers in ICUs, as previously described by other studies.[6,9-11,14,19] The MDR status varied from 2.9% to 80.8%, with *P. aeruginosa* being the least resistant and *Enterococcus* spp. the most resistant isolates of the four bacterial species described in this study. Also, we identified several MRSA, VRE, and imipenem-resistant *Acinetobacter* spp. strains. Those isolates represent a threat in ICUs and have been described as causative agents of outbreaks and nosocomial infections in developing countries,[3,4,20] although, there is not

Table 4. Distribution of *S. aureus* phenotypes according to sampling date, hospital and intensive care units

Code*	Sampling date									
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th
AP004	R1	.	.	-	.	.	-	.	-	-
AP007	.	R1	.	.	-	-	.	-	.	.
AP010	.	R4	.	.	.	.	.	.	R4	.
AP012	.	-	.	-	-	.	.	R3a	.	-
AP016	-	-	.	R3a	.	.	-	.	.	-
AN002	.	-	.	-	.	-	R1	.	-	.
AN003	.	R4	.	R5	.	.	R4	.	.	-
AN004	.	.	.	-	.	-	-	.	R1	.
AN005	.	-	.	.	-	-	.	.	R3a	.
AN009	.	.	-	.	R2b	.	-	.	.	.
AN011	.	-	.	-	.	-	.	.	.	R2a
AN015	-	R1	R1	.	.	-	.	-	.	-
AN017	-	R5	.	.	-	.	-	-	.	.
AN019	-	.	.	.	R5	.	-	.	-	.
BP002	.	.	-	-	.	-	R1	.	.	.
BP003	R3b	.	.	R1	.	-	.	.	-	.
BP011	.	-	-	.	.	.	R2a	.	-	-
BP016	.	.	-	.	R2a	.	-	.	.	-
BP018	R1	.	.	-	-	.	-	.	-	.
BP033	-	-	-	.	R1	.	.	.	-	.
CP010	.	.	-	-	.	-	.	.	.	S
CP011	.	.	.	-	.	-	.	-	R1	.
CN003	.	S	.	.	.	.	.	.	.	.
CN005	.	.	-	-	.	.	-	.	R3a	.
CN022	-	.	.	.	R3a	.	.	.	.	.

*Code: Hospital, "P" for pediatric ICU or "N" for neonatology ICU, and cell phone ID

"." – no *S. aureus* isolation; **"-"** – no sample collected.

enough evidence to conclude direct transmission from cell phones to hospitalized patients.[6]

Several studies have highlighted the role of the cell phone as potential reservoir of resistant bacteria and its capacity to spread pathogens.[6,11,19] According to our previous results[12] and the findings reported here, the bacterial contamination in cell phones was frequent and distributed across the study time. This finding suggests that cell phones are devices susceptible to pathogen contamination. The maintenance of bacterial contamination over time was not assessed in this study due to the design, wide-ranging interval between sampling dates and incomplete sampling for each cell phone enrolled. Further studies with more continuous sampling are needed to determine if cell phones can maintain bacteria for prolonged periods of times.

The bacterial contamination in cell phones occurs in short periods of time generating a continuous turnover of bacterial populations.[21] Also, cell phones may be involved in cross-contamination between hospital wards.[11,19] We hypothesized that the continuous contamination from several sources, not specifically within the ICUs, might explain the high diversity of antimicrobial resistance patterns observed in *Enterococcus* spp. isolates. An extensive surveillance network conducted during 2008 to 2009 in Peru showed that MDR *P. aeruginosa* are frequently isolated from blood cultures.[22] In this study, the less variable phenotypes observed in non-resistant *P. aeruginosa* isolates could reflect a constant, unique and non-hospital source of contamination. Despite not having determined the contamination source in this study, we recommend that further studies explore the epidemiological role of cell phones within ICUs. In the meantime, the use of cell phones should be controlled or banned within the ICU to avoid bacterial threats.

The integration of epidemiological data in terms of time and the application of molecular tools allow testing transmission routes in clinical care settings.[20] Health care workers have been described as carriers of *S. aureus* in nares, hands, or both.[6-8] Recently, Chang et al.[8] found that 87.5% of *S. aureus* isolated from health care workers and their cell phones were clonally related. In this study, we observed phones that harbored bacteria with the same phenotype in consecutive sampling dates and also on separate sampling dates. We did not characterize the isolates at the molecular level to infer their clonal

relationship or to compare them to other strains like USA300, USA100 or other clonal clusters defined by multilocus sequence typing. Also, we did not collect samples from health care workers. However, because the phenotypes were constructed using information derived from MIC results for multiple antimicrobial agents, the MRSA status and one mechanism of antimicrobial resistance, the findings suggest that strains may be related to each other. Further comprehensive studies are needed to determine if clonal pathogens can remain over the time on the cell phone surface.

Conclusion

In summary, our data suggest that cell phones are susceptible to be contaminated with highly harmful pathogens such as methicillin-resistant *S. aureus*, vancomycin-resistant *Enterococcus*, and MDR *P. aeruginosa* and *Acinetobacter* spp. Additional studies are needed to evaluate the cell phone's capacity to preserve bacterial pathogens over time in order to elucidate its role as a reservoir.

Authors' contributions statement: SL, LG and JT designed and supervised the study. SL, LG, EA, NS and JT participated in sample and data collection, and carried out the laboratory work. SL and JT wrote the manuscript. All the authors read and approved the final version of the manuscript.

Conflicts of interest: All authors – none to disclose.

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