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Antibacterial Resistant Gram-Positive Bacteria in Oil Refinery Workers

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Abstract: ***Background*:** The genetic plasticity of gram-positive bacteria is exemplified by the rapid acquisition and dissemination of resistance genes. Mobile genetic elements (MGE), plasmids, and transposons, which promote horizontal gene transfer (HGT), help spread resistance-related traits quickly. ***Methodology*:** This study was carried out in the Northern Refinery Company (NRC), one of the biggest factories in the country, which is located in the north of Baiji city. 200 samples were collected from patients worked in the Northern Refineries Company in Baiji town, equal number of samples was collected from patients hospitalized in Tikrit teaching hospital as control. Swabs taken from upper respiratory tract and sputum samples taken from lower respiratory tract, as soon as possible taken to the laboratory, were cultured on four plates of culture media recommended for primary isolation of pathogens. Antibiotic resistance was also estimated using conventional methods. ***Results*:** The minimal inhibitory concentration (MIC) ranges of antibiotics for the *Staphylococcus aureus* that inhibited were carried out for the groups of both refinery and hospital isolates that showed 50% of resistance or more. Most of refinery strains coagulase negative staphylococci were inhibited by 4µg/ml, while most strains of hospital were inhibited by 16µg/ml. The majority of *Streptococcus pyogenes* strains show MICs values ranged between 1-32 µg/ml, rarely showed < 1 µg/ml. MIC for tetracycline ranged between ≤ 0.125 -64µg/ml among isolates of both refinery and hospital. Statistical analysis showed no significant differences in the MIC distributions of gentamicin or tetracycline between refinery and hospital isolates of *Streptococcus pneumoniae*. There were no statistically significant differences in the MIC distributions between refinery and hospital isolates of *Micrococcaceae* for amikacin, gentamicin, or tobramycin ($p > 0.05$ for all comparisons). ***Conclusions*:** Statistical analysis revealed that the differences in MIC distributions between R and H *Staphylococcus aureus* isolates were evaluated separately for each antimicrobial agent using an exact R × C contingency-table test with Monte-Carlo simulation (200,000 replicates), because of sparse cell frequencies. For coagulase negative staphylococci, Table 2 shows that MIC for amoxicillin/ clavulanic acid was ranged between 2 -64µg/ml among isolates of refinery and between 0.5 -64µg/ml among isolates of hospital. Most of refinery strains were inhibited by 4µg/ml, while most strains of hospital were inhibited by 16µg/ml. The results agreed to some extent. The majority of *Streptococcus pyogenes* strains show MICs values ranged between 1-32 µg/ml, rarely showed < 1 µg/ml. MIC for tetracycline ranged between ≤ 0.125 -64µg/ml among isolates of both refinery and hospital. The MIC distributions of gentamicin and tetracycline against *Streptococcus pyogenes* were compared between refinery and hospital isolates using an exact R × C contingency-table analysis with Monte-Carlo simulation

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(200,000 replicates). Statistical analysis showed no significant differences in the MIC distributions of gentamicin or tetracycline between refinery and hospital isolates of *Streptococcus pneumoniae*. There were no statistically significant differences in the MIC distributions between refinery and hospital isolates of *Micrococcaceae* for amikacin**,** gentamicin**,** or tobramycin **(**$p > 0.05$ for all comparisons). Although refinery isolates showed numerically higher median MIC values than hospital isolates for all three antibiotics, these differences did not reach statistical significance.

Keywords: Gram-Positive Bacteria, Antibiotic Resistance, Oil Refinery, Workers.

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INTRODUCTION

The genetic plasticity of gram-positive bacteria is exemplified by the rapid acquisition and dissemination of resistance genes. Mobile genetic elements (MGE), plasmids, and transposons, which promote horizontal gene transfer (HGT), help spread resistance-related traits quickly [1]. This is made more difficult by the joint localization of resistant gene loci on mobile genetic components, which results in a reservoir of flexible resistance potential [2, 3]. It was obvious from the study carried out by Boboye, *et al*., [4], that when the environment was contaminated with petroleum, the proportion of hydrocarbon-degrading microorganisms increased rapidly (Okerentugba and Ezeronye [5-8]. In particular, the proportion of bacterial populations with plasmids containing genes for utilization are increased [9]. Some of these organisms are true pathogens include; *Enterobacter aerogenes, Aerococcus viridans, Staphylococcus aureus, Micrococcus luteus,* and *Bacillus* spp. Resistance is defined as bacteria that are not inhibited by usually achievable systemic concentration of an agent with normal dosage schedule and/ or fall in the minimum inhibitory concentration ranges. Likewise the multiple drug resistance is defined as the resistance to two or more drugs or drug classes [10-13]. Acquisition of resistance to one antibiotic conferring resistance to another antibiotic, to which the organism has not been exposed, is called cross resistance [14-17]. Resistance to antimicrobial drugs by gram-positive bacteria is a complex phenomenon that is influenced by a number of different factors. These elements work together to amplify and spread resistance genes, making it more difficult to treat infections brought on by *Staphylococcus aureus*, *Enterococcus* species, and *Streptococcus pneumoniae* [18]. It is essential to have a thorough grasp of these contributing components to develop successful mitigation methods for the resistance propagation. Although the common causative pathogens for community-acquired bacterial RTIs are few, selection of antibiotic therapy is becoming more complicated by the increasing rate of bacterial resistance to many of the antibiotics commonly used to treat these RTIs. Antimicrobial agents are often categorized according to their principal mechanism of action [19-22]. Antibiotic resistance occurs when bacteria change in some way that reduces or eliminates the effectiveness of drugs, chemicals or other agents designed to cure or prevent the infection. Thus the bacteria survive and continue to multiply causing more harm. Widespread use of antibiotics promotes the spread of antibiotic resistance by induction. Healthcare-associated infections continue to be a predominant source of resistant gram-positive bacteria. Recent genomic studies have elucidated the complex dynamics of nosocomial transmission, revealing the part mobile genetic elements play in enabling the transfer of resistance genes between bacterial strains. Enhanced genomic surveillance has allowed for the tracing of transmission routes and identification of high-risk reservoirs within healthcare facilities [23]. Antibiotics are given to human for treatment and prophylaxis of infectious diseases, 80% to 90% of antibiotics are used in outpatients and the remainder in hospitals. Antibiotics appear to be used not only in excess but also inappropriately and this accounts for 20% to 50 % of all antibiotics used [24-28]. Resistance has been documented for all organisms associated with community-acquired RTIs. Current estimates indicate that 25% to more than 50% of *S. pneumoniae* strains are not completely susceptible to penicillin and that nearly one third of strains may be resistant to macrolides [29-32]. Penicillin-resistant *S. pneumoniae* also may have reduced susceptibility to other antibiotics, including tetracycline, erythromycin, azithromycin, cephalosporins, clindamycin, trimethoprim/sulfamethoxazole (TMP/ SMX), and chloramphenicol [33-36]. *Streptococcus pneumoniae* have developed resistance to fluoroquinolones as well. This is of particular concern to the Centers for Disease Control and Prevention (CDC), having influenced its recommendations regarding empiric treatment of pneumonia [37-40]. Macrolides have been mainstays in empiric therapy of Community Acquired Pneumonia because of their activity against both typical and atypical respiratory pathogens; however, their effectiveness has been compromised by a rapid rise in resistance by *S. pneumoniae*. An evaluation of *S. pneumoniae* isolates collected between 2000 and 2001 showed that resistance rates to erythromycin, clarithromycin, and azithromycin had increased to 31.0%, 30.7%, and 31.0%, respectively [41-44]. Further results from recent surveillance study

revealed that examined macrolide resistance among patients with community-acquired RTIs being treated by primary care physicians indicated that 23% to 33% of *S. pneumoniae* isolates were resistant to macrolides [45-48]. Resistance also is becoming prevalent among other pathogens associated with community-acquired RTIs, including *H. influenzae* and *M. catarrhalis* [49-52]. This may complicate management of patients with community-acquired RTIs; infection with a treatment-resistant pathogen can increase risk for morbidity and mortality if treatment fails to eradicate the bacteria [53-56]. However, the clinical impact of resistance remains controversial. Resistance may be overcome with the use of an antibiotic with pharmacokinetic properties or a dosing regimen that achieves very high drug concentrations at sites of infection. Host defense mechanisms also may contribute to eradication of organisms with in vitro resistance to an antibiotic [57-61]. Further, since many infections are caused by viruses with high-resolution rates and since some bacterial infections resolve due to host response, the in vivo efficacy of an antibiotic may be much higher than its in vitro sensitivity [62].The present study is an attempt to assess the difference between incidence of antibiotic resistance detected among patients of oil refinery compared to community individuals complaining of respiratory infections in hospital. Within this context, this review aims to examine the rising incidence of antimicrobial resistance in bacterial isolates with gram-positive genes, with a particular focus on well-known pathogens including *Staphylococcus aureus*, *Enterococcus species*, and *Streptococcus pneumoniae*. Understanding the mechanics of how antibiotic resistance is changing and developing is crucial for developing measures to stop its spread, hence this topic must be thoroughly explored.

MATERIALS AND METHODS

Description of the Refinery

This study was carried out in the Northern Refinery Company (NRC), one of the biggest factories in the country, which is located in the north of Baiji city. This factory was established in 1980, and considered as one of the major oil companies in middle-east which depends on for bridging the needs of local consumption of petroleum products and export the surplus outside. The number of workers at this refinery is about 8000 workers, distributed on six main sections, these sections are divided into major sub-sections, and the section of production is one of those where the workers exposed to the direct and indirect risks of pollution.

Patients

From October 2008 to September 2009, samples were collected from patients with upper and lower respiratory tract infections, 200 samples were collected from patients worked in the Northern Refineries Company in Baiji town, equal number of samples was collected from patients hospitalized in Tikrit teaching hospital as control samples for comparison.

Samples

Swabs from upper respiratory tract infections: (Tonsillitis, laryngitis, sore throat and Pharyngitis) were transported to the laboratory using transport medium swabs and sputa from lower respiratory tract infections: (Bronchitis and Pneumonia) were transported using Cary-Bliar transport medium in screw capped vials [62]. Samples were taken according to the diagnosis of specialist physicians after washing the mouth three times to eliminate much amounts of mouth normal flora [63, 64].

Isolation of Bacteria

Swabs taken from upper respiratory tract and sputum samples taken from lower respiratory tract, as soon as possible taken to the laboratory, were cultured on four plates of culture media recommended for primary isolation of pathogens. These media included two plates of blood agar, Chocolate agar and MacConkey agar. One plate of Blood agar and Chocolate agar were incubated in a candle jar to enhance growth, while the other two plates were incubated aerobically. All the four plates were incubated at 37 º C for 18-24 hours. Culture negative plates were re-incubated for 48 hours with daily checking before discarding. Small isolated colonies grown on blood and chocolate media were purified and cultivated by streaking on plain blood agar plates with regard of gas requirement, whereas large colonies were purified by the same method on available ordinary media such as nutrient or brain heart infusion agar to be further investigated [65, 66].

Preservation of Purified Isolates

Directly, after purification, overnight culture broth at 37 º C for 18-24 hours was prepared for each stock cultures from purified isolates were made by cultivating bacteria in brain heart infusion broth and adding glycerol to obtain 20% as final concentration, then stored at - 20 º C [67, 68].

Identification Methods

All the identification tests were done according to the conventional methods described by Cruickshank [69], Collee [70], Al-Jebouri and Al-Bayati [68, 69].

Antibiotic Susceptibility Testing

A loopful growth from isolates of each bacteria were inoculated into nutrient broth and incubated at 37°C for 18 hours. The bacterial suspensions were diluted with ringer solution. The proportion of dilution was 1:1000 [34]. Diluted bacterial suspension were poured onto the surface of the Muller-Hinton agar plates. The excess of bacterial suspensions were discarded using Pasteur pipette and plates were left for one hour at room temperature to dry. The antibiotic discs which are shown in Table 1 were selectively applied by using sterile forceps which was flamed after being cleansed with

alcohol. The plates were incubated at 37 0C for over-night. The size of zones of inhibition were measured from edge of disc to the edge of inhibition of growth and the result was compared with standard diameter of inhibition zones for each antibiotic utilizing the method of Bauer *et al*., [35-72].

Statistical Analyses

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics such as means, standard deviations, and frequency distributions were computed to summarize the data. A paired sample t test correlation coefficient (R) and coefficient of determination (R2) were calculated [73-76].

RESULTS

Staphylococcus Aureus

The minimal inhibitory concentration (MIC) ranges of antibiotics for the bacterial groups that inhibited were carried out for the groups of both refinery and hospital isolates that showed 50% of resistance or more (Table 1, Figure 1). Statistical analysis revealed that the differences in MIC distributions between R and H *Staphylococcus aureus* isolates were evaluated separately for each antimicrobial agent using an exact R × C contingency-table test with Monte-Carlo simulation (200,000 replicates), because of sparse cell frequencies. Effect size was estimated using Cramer's V. Significant differences in MIC distribution were observed for gentamicin (CN; $\chi^2 = 21.10$, Monte-Carlo $p < 0.001$, Cramer's V = 0.631) and tobramycin (TOB; $\chi^2 = 13.36$, $p = 0.026$, V = 0.502). No significant differences were detected for amoxicillin/clavulanic acid (AMC; $p = 0.081$) or chloramphenicol (C; $p = 0.423$).

Table 1.Minimal Inhibitory Concentration (MIC) of antibiotics for *Staphylococcus aureus.*

CONC. µg/ml	AMC		C		CN		TOB	
	R No. (%)	H No. (%)	R No. (%)	H No. (%)	R No. (%)	H No. (%)	R No. (%)	H No. (%)
≤0.125		1 (2.8)			2 (11.8)			3 (8.3)
0.25	1 (5.9)		2 (11.8)	2 (5.6)	6 (35.3)	5 (13.9)	2 (11.8)	5 (13.9)
0.5	4 (23.5)	20 (55.6)	4 (23.5)	9 (25)		22 (61.1)	4 (23.5)	17 (47.2)
1	1 (5.9)	4 (11.1)	3 (17.6)	1 (2.7)		1(2.8)		
2		1 (2.8)					4 (23.5)	9 (25)
4	3 (17.6)	3 (8.3)		2 (5.6)	1 (5.9)	1(2.8)	2 (11.8)	
8	6 (35.3)	7 (19.4)					2 (11.8)	
16	1 (5.9)				3 (17.6)	3 (8.3)	3(17.6)	2 (5.6)
32	1 (5.9)				5 (29.4)	4 (11.1)		
64			7 (41.2)	16 (44.4)				
128			1 (5.9)	4 (11.1)				
256				2 (5.6)				
TOTAL	17 (100)	36 (100)	17 (100)	36 (100)	17 (100)	36 (100)	17 (100)	36 (100)

AMC, amoxicillin/clavulanic acid; C, chloramphenicol; CN, gentamicin; TOB, tobramycin.
R, refinery cases; H, hospital cases.

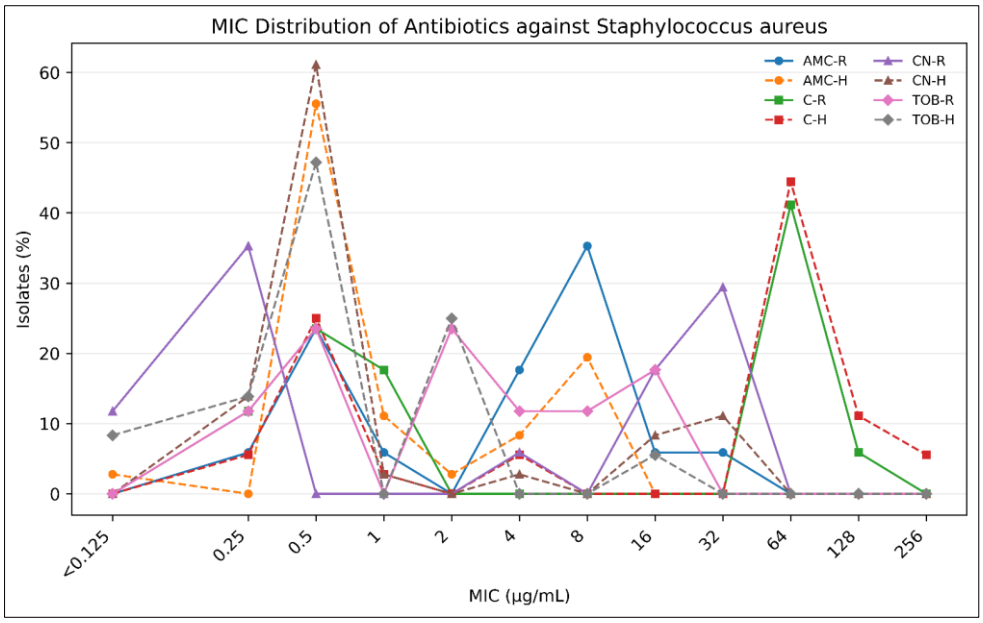


Figure 1: Distribution of minimum inhibitory concentrations (MICs) of amoxicillin/clavulanic acid, chloramphenicol, gentamicin, and tobramycin against *Staphylococcus aureus* isolates from R and H locations.

Coagulase-Negative Staphylococci

For coagulase negative staphylococci, Table 2 shows that MIC for amoxicillin/ clavulanic acid was ranged between 2 -64µg/ml among isolates of refinery and between 0.5 -64µg/ml among isolates of hospital. Most of refinery strains were inhibited by 4µg/ml, while most strains of hospital were inhibited by 16µg/ml. The distributions of minimum inhibitory concentrations (MICs) were compared between refinery and hospital isolates of coagulase-negative staphylococci using an exact R × C contingency-table analysis with Monte-Carlo simulation (200,000 replicates) because of sparse cell frequencies. A significant difference between locations was observed for tobramycin ($\chi^2 = 20.07$, $p = 0.003$, Cramer's $V = 0.675$), indicating a strong association between isolate source and tobramycin MIC distribution (Figure 2). No significant differences were detected for amoxicillin/clavulanic acid (AMC; $p = 0.565$) or gentamicin (CN; $p = 0.064$).

Table 2. Minimal Inhibitory Concentration (MIC) of antibiotics for Coagulase Negative Staphylococci

CONC. µg/ml	AMC		CN		TOB	
	R No. (%)	H No. (%)	R No. (%)	H No. (%)	R No. (%)	H No. (%)
≤0.125			3 (23.1)	2 (6.5)	3 (23.1)	10 (32.3)
0.25			1 (7.7)	11 (35.5)		3 (9.7)
0.5		2 (6.5)		2 (6.5)		
1					2 (15.4)	
2	2 (15.3)	1 (3.2)				
4	4 (30.8)	3 (9.7)				
8				2 (6.5)	1 (7.7)	
16	3 (23.1)	15 (48.4)		2 (6.5)	1 (7.7)	1 (3.2)
32	3 (23.1)	7 (22.6)	3 (23.1)	5 (16.1)		
64	1 (7.7)	3 (9.7)	5 (38.4)	7 (22.6)	1 (7.7)	13 (41.9)
128					5 (38.4)	
256						1 (3.2)
512			1 (7.7)			3 (9.7)
TOTAL	13 (100)	31 (100)	13 (100)	31 (100)	13 (100)	31 (100)

AMC, amoxicillin/clavulanic acid; CN, gentamicin; TOB, tobramycin.
R, refinery cases; H, hospital cases.

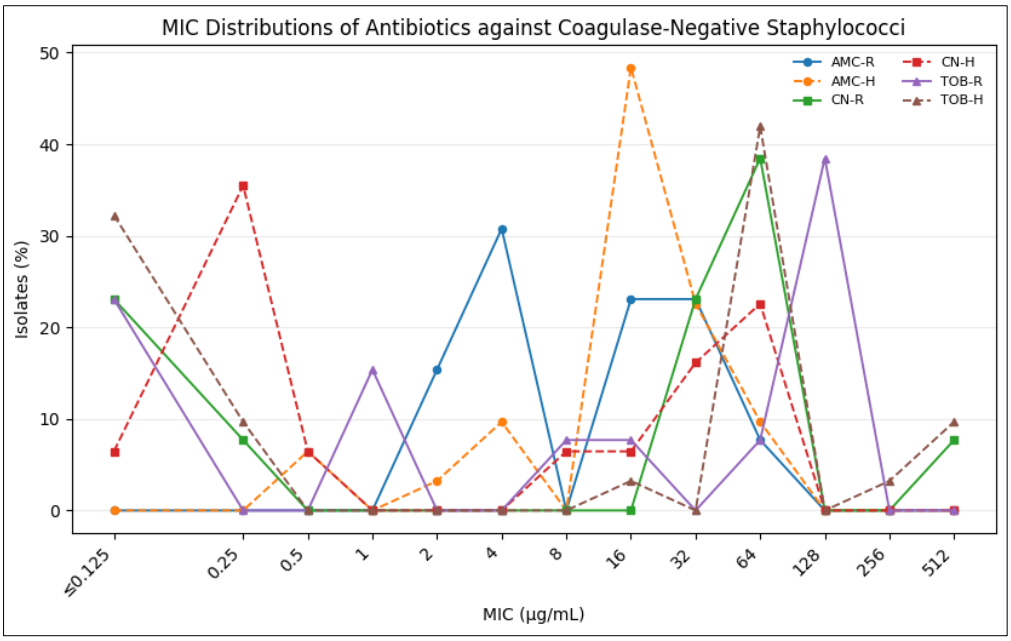


Figure 2: Distribution of minimum inhibitory concentrations (MICs) of amoxicillin/clavulanic acid, gentamicin, and tobramycin against coagulase-negative staphylococci isolated from refinery and hospital cases.

Streptococci

Table 3 shows that MIC for gentamicin used for *Streptococcus pyogenes* ranged between 0.5 -16µg/ml among isolates of refinery and between 2 -16µg/ml among isolates of hospital but the most of strains were inhibited at 8µg/ml. The results agreed to some extent. The majority of *Streptococcus pyogenes* strains show MICs values ranged between 1-32 µg/ml, rarely showed < 1 µg/ml. MIC for tetracycline ranged between ≤ 0.125 -64µg/ml among isolates of both refinery and hospital. The MIC distributions of gentamicin and tetracycline against *Streptococcus pyogenes* were compared between refinery and hospital isolates using an exact R × C contingency-table analysis with Monte-Carlo simulation (200,000 replicates). No statistically significant differences were observed between refinery and hospital isolates for gentamicin ($\chi^2 = 6.67$, $p = 0.135$, Cramer's V = 0.242) or tetracycline ($\chi^2 = 11.98$, $p = 0.095$, Cramer's V = 0.324). These findings indicate broadly comparable MIC distributions between the two sources(Figure 3). Statistical analysis showed no significant differences in the MIC distributions of gentamicin or tetracycline between refinery and hospital isolates of *Streptococcus pneumoniae*. For gentamicin, the difference between the two groups was not statistically significant (Mann–Whitney U = 159.5, p = 0.301). Similarly, tetracycline MIC values did not differ significantly between refinery and hospital isolates (Mann–Whitney U = 114.0, p = 0.508). Although refinery isolates tended to show higher gentamicin MIC values and tetracycline MICs displayed a wider distribution between the two groups, these variations did not reach statistical significance (p > 0.05)(Table 4,Figure 4).

Table 3. Minimal Inhibitory Concentration (MIC) of antibiotics for *Streptococcus pyogenes*

CONC. µg/ml	CN		TE	
	R No. (%)	H No. (%)	R No. (%)	H No. (%)
≤0.125			27 (43.6)	21 (40.4)
0.25			1 (1.6)	4 (7.6)
0.5	2 (3.2)			2 (3.9)
1				
2	11 (17.7)	14 (26.9)		2 (3.9)
4	5 (8.1)	1 (1.9)		
8	35 (56.5)	31 (59.7)		1 (1.9)
16	9 (14.5)	6 (11.5)	7 (11.3)	5 (9.6)
32			25 (40.3)	14 (26.9)
64			2 (3.2)	3 (5.8)
TOTAL	62 (100)	52 (100)	62 (100)	52 (100)

CN, gentamicin; TE, tetracycline. R, refinery cases; H, hospital cases.

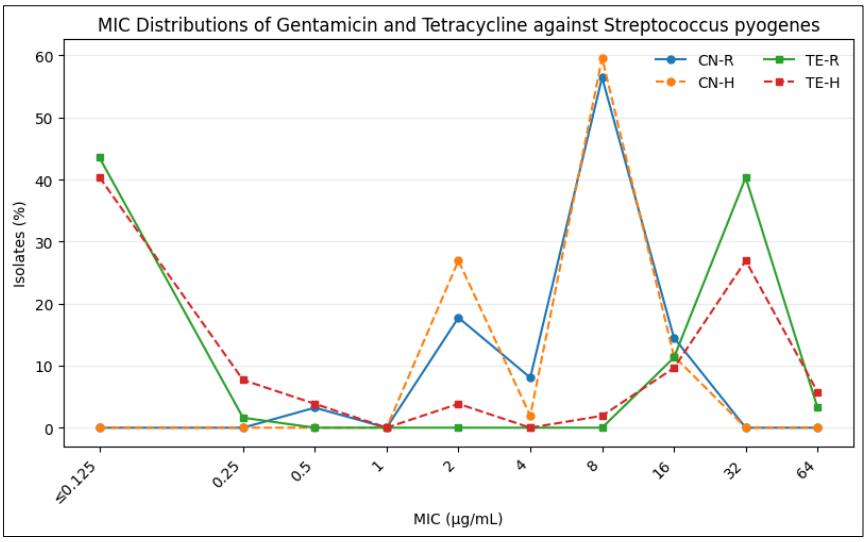


Figure 3: Distribution of minimum inhibitory concentrations (MICs) of gentamicin and tetracycline against *Streptococcus pyogenes* isolates from refinery and hospital cases.

Table 4. Minimal Inhibitory Concentration (MIC) of antibiotics for *Streptococcus pneumoniae*

CONC. µg/ml	CN		TE	
	R No. (%)	H No. (%)	R No. (%)	H No. (%)
≤0.125	2 (9.1)	2 (16.7)	6 (27.3)	5 (41.7)
0.25			2 (9.1)	
0.5	2 (9.1)		1 (4.5)	
2				1 (8.3)
8	1 (4.5)			
16		1 (8.3)	1 (4.5)	
32	6 (27.3)	6 (50)	12 (54.6)	1 (8.3)
64	11 (50)	3 (25)		4 (33.4)
128				1 (8.3)
TOTAL	22 (100)	12 (100)	22 (100)	12 (100)

CN, gentamicin; TE, tetracycline; TOB. R, refinery cases; H, hospital cases.

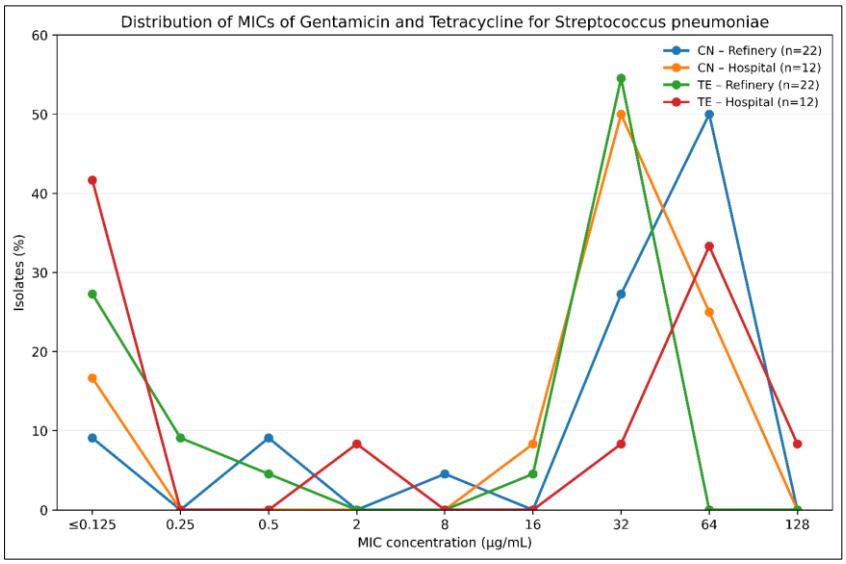


Figure 4: Distribution of Minimal Inhibitory Concentrations of Gentamicin and Tetracycline against *Streptococcus pneumoniae* Isolates from Refinery and Hospital Cases.

Micrococci

Table 5 shows that for Micrococcaceae isolated from refinery (represented for *Stomatococcus mucilagenosus* and *Micrococcus leuteus*), MICs of amikacin and gentamicin ranged between ≤ 0.125-16µg/ml and MICs of tobramycin ranged between 1-8µg/ml, while MICs values for strains isolated from hospital (represented for *Micrococcus leuteus* and *Micrococcus nishinomiyaensis*) were 0.5µg/ml, ≤ 0.125µg/ml and 2µg/ml of amikacin, gentamicin and tobramycin, respectively. There were no statistically significant differences in the MIC distributions between refinery and hospital isolates of *Micrococcaceae* for amikacin**,** gentamicin**,** or tobramycin **(**$p > 0.05$ for all comparisons). Although refinery isolates showed numerically higher median MIC values than hospital isolates for all three antibiotics, these differences did not reach statistical significance. This result should be interpreted cautiously because the number of hospital isolates was very small (**n = 2**)(Figure 5).

Table 5: Minimal Inhibitory Concentration (MIC) of antibiotics for Micrococcaceae

CONC. µg/ml	AK		CN		TOB	
	R No. (%)	H No. (%)	R No. (%)	H No. (%)	R No. (%)	H No. (%)
≤0.125	2 (22.2)		3 (33.3)	2 (100)		
0.5	2 (22.2)	2 (100)				
1			1 (11.1)		1 (11.1)	
2			1 (11.1)		1 (11.1)	2 (100)
8	2 (22.2)				7 (77.7)	
16	3 (33.3)		4 (44.4)			
TOTAL	9 (100)	2 (100)	9 (100)	2 (100)	9 (100)	2 (100)
AK, amikacin; CN, gentamicin; TOB, tobramycin. R, refinery cases; H, hospital cases.						

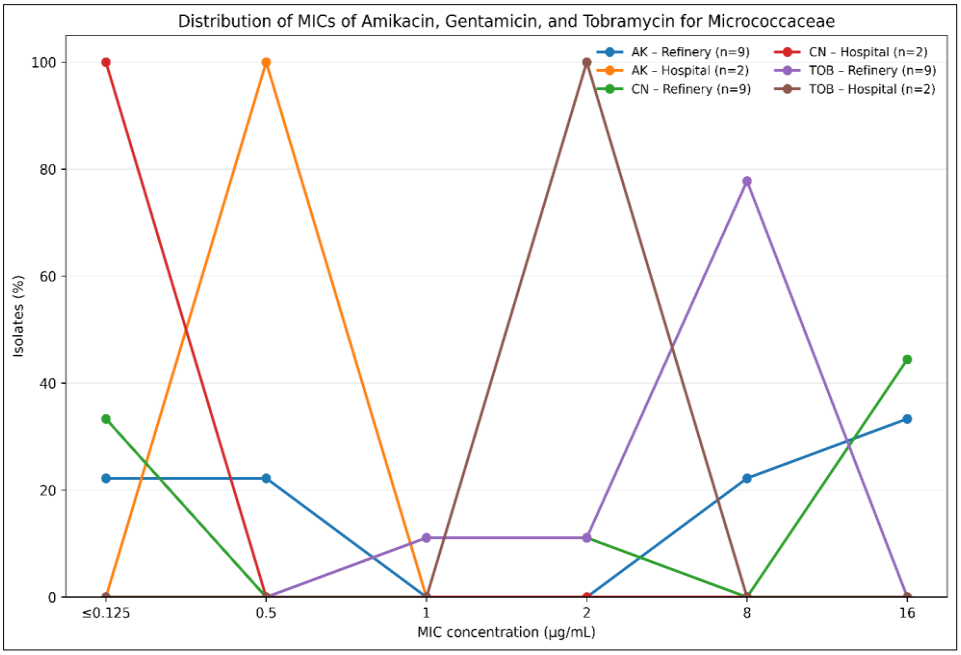


Figure 5: Distribution of Minimal Inhibitory Concentrations of Amikacin, Gentamicin, and Tobramycin against *Micrococcaceae* Isolates from Refinery and Hospital Cases

DISCUSSION

The present study revealed that the MIC for amoxicillin/ clavulanic acid, chloramphenicol, gentamicin and tobramycin was determined in all of the 17 and the 36 strains of *Staphylococcus aureus* isolated from both refinery and hospital, respectively. It was found that MICs of amoxicillin/clavulanic acid ranged between 0.25 and 32 µg/ml and ≤0.125-8 µg/ml and the largest number of isolates was inhibited by 8µg/ml and 0.5µg/ml for refinery and hospital isolates, respectively. This finding approximately disagreed with other results [77], who found that concentrations of 0.01 to 0.08 µg /ml inactivates 50% of ß-lactamases released by *S. aureus* and and agreed with the study was carried out by other workers [78], who found that 50% of *S. aureus* was inhibited by ≥ 64 µg /ml. MICs for chloramphenicol ranged between 0.25-128µg/ml for strains of refinery and between 0.25-256µg/ml for strains of hospital, the most strains were inhibited by 64µg/ml. Other workers [79], found thirteen chloramphenicol resistant *S. aureus* with MICs ranged between 16- 256µg/ml. MIC for

gentamicin ranged between ≤0.125- 32µg/ml and 0.5-32µg/ml for both, largest number inhibited by 0.25µg/m and 0.5µg/ml for both refinery and hospital isolates, respectively. It was found somewhere else [78], that 50% of *S. aureus* was inhibited by > 16µg/ml, while other [80], found that typical *S. aureus* MIC of gentamicin is 4 µg/ml. but strains of *S. aureus* that express multiple resistance mechanisms recorded MIC > 64µg/ml of gentamicin. MIC for tobramycin ranged between 0.5 - 16µg/ml, ≤0.125 -16µg/ml for both refinery and hospital isolates, respectively. Furthermore, resistance to antimicrobial drugs by gram-positive bacteria is a complex phenomenon that is influenced by a number of different factors. These elements work together to amplify and spread resistance genes, making it more difficult to treat infections brought on by *Staphylococcus aureus*, *Enterococcus* species, and *Streptococcus pneumoniae* [81]. However, it is essential to have a thorough grasp of these contributing components to develop successful mitigation methods for the resistance propagation. The clinical importance of the adaptive and versatile gram-positive bacteria *Staphylococcus aureus* has long been acknowledged. But the advent of methicillin-resistant *Staphylococcus aureus* (MRSA) has thrust this disease to the forefront of worries about antibiotic resistance. The implications of MRSA extend beyond its intrinsic β-lactam antimicrobial resistance, as it underscores the broader challenge of antibiotic resistance within the gram-positive bacterial landscape [82]. The expansion of MRSA's antibiotic resistance repertoire is facilitated by the remarkable adaptability conferred by horizontal gene transfer (HGT) and mobile genetic elements (MGEs). Genetic elements like plasmids, transposons, and integrons enable the transfer of resistance genes between bacterial species, contributing to the spread of antimicrobial resistance [83]. HGT and MGEs have enabled MRSA to acquire resistance determinants against a range of antibiotics, amplifying the clinical impact of this pathogen. Furthermore, The identification of *Diaphorobacter* sp. in our study is consistent with reports by others [84], who found that *Diaphorobacter* sp. strain MNS-0 could impressively degrade ampicillin and chloramphenicol [85]. This parallels our results where our *Diaphorobacter* isolates showed multidrug resistance. It is well known that heavy metal and antibiotic resistance genes often co-localize on plasmids, facilitating horizontal gene transfer and activating survival pathways in multi-pollutant environments. This co-resistance provides dual adaptive advantages that sustain bacterial viability in contaminated sites. Consequently, these plasmids drive efficient bioremediation through the simultaneous detoxification and catabolism of organic hydrocarbons and inorganic co-contaminants [86, 87]. phenanthrene while maintaining resistance to a wide spectrum of antibiotics, including most strains of refinery were inhibited by 0.5µg/ml and 2µg/ml, while most strains of hospital were inhibited by 0.5µg/ml. The statistical analyses using T- test showed there were no significance differences in the responses to different concentrations between both groups of *Staphylococcus aureus*. It was obvious from the study carried out elsewhere [4] that when the environment was contaminated with petroleum, the proportion of hydrocarbon-degrading microorganisms increased rapidly. In particular, the proportion of bacterial populations with plasmids containing genes for utilization are increased [88]. The bacterium with the highest degrading activities on kerosene was *S. aureus* that able to exist in the oil spilled environment. The genes pool of accessory genome of *Staph. aureus* is mainly composed of mobile genetic elements such as plasmids, transposons, phages and pathogenicity islands. For coagulase negative staphylococci, the present study shows that MIC for amoxicillin/ clavulanic acid was ranged between 2 -64µg/ml among isolates of refinery and between 0.5 -64µg/ml among isolates of hospital. Most of refinery strains were inhibited by 4µg/ml, while most strains of hospital were inhibited by 16µg/ml. Others [89] found that penicillin resistant coagulase negative Staphylococci were with MICs ranged between 0.12 - >16 µg/ml, while penicillin sensitive coagulase negative Staphylococci were with MICs ranged between 0.06-0.25µg/ml. MIC for gentamicin ranged between ≤0.125 -512 µg/ml among isolates of refinery and between <0.125-64 µg/ml among isolates of hospital but the most strains of both were inhibited at 64µg/ml and 0.25 µg/ml, respectively. Recently, it was found that coagulase negative staphylococci showing increased multidrug resistance have a MIC of gentamicin ranged between 0.5-64µg/ml, 50% and 90% of them having MIC values of 32µg/ml and 64µg/ml, respectively [90]. MIC for tobramycin ranged between ≤0.125 -128 µg/ml among isolates of refinery and between ≤0.125 - 512µg/ml among isolates of hospital. Most strains of refinery were inhibited at 128µg/ml while most of coagulase negative staphylococci isolated from hospital were inhibited at 64µg/ml. According to the EUCAST, (2009) MICs of tobramycin for pathogenic coagulase negative staphylococci range between 0.032 – 256µg/ml, most strains are inhibited by 0.25µg/ml. The present study shows that MIC for gentamicin used for *Streptococcus pyogenes* ranged between 0.5 -16µg/ml among isolates of refinery and between 2 -16µg/ml among isolates of hospital but the most of strains were inhibited at 8µg/ml when compared to other workers [91] the results agreed to some extent. The majority of *Streptococcus pyogenes* strains show MICs values ranged between 1-32 µg/ml, rarely showed < 1 µg/ml. MIC for tetracycline ranged between ≤ 0.125 -64µg/ml among isolates of both refinery and hospital. The most effect MIC is ≤ 0.125µg/ml. especially with respect to refinery strains, this was largely agreed with what found elsewhere [92], that found 39% of strains were with MICs ranged between 0.12 – 0.5 µg/ml and 59% of them had MICs ranged between 16- 64 µg/ml with 2 (out of 130) isolates showed MICs equal to 128 µg/ml and 256 µg/ml. The statistical analyses showed there were no significance differences. About MIC for *Streptococcus pneumoniae*, table 19, for gentamicin the MIC for strains

of both refinery and hospital ranged between ≤ 0.125 - 64µg/ml, the high effect was at 64µg/ml for refinery and 32µg/ml for hospital strains. This approximately agreed with what was found by others [91]. A Gram-positive bacterium called *Streptococcus pneumoniae* causes various infections, from minor respiratory disorders to fatal invasive diseases. The prevalence of *pneumococcal* infections has dramatically decreased due to vaccinations that target specific serotypes; the relentless evolution of antibiotic resistance threatens to undermine these gains. Recent findings shed light on the escalating prevalence of multidrug-resistant *Streptococcus pneumoniae* (MDRSP) strains, marking a critical juncture in the fight against bacterial infections [93, 94]. The escalation of antibiotic resistance in *Streptococcus pneumoniae* has amplified therapeutic challenges. The limited arsenal of effective antibiotics, coupled with the increasing prevalence of MDRSP strains, underscores the urgency of innovative approaches. Advances in genome editing technologies, the repurposing of existing drugs, and the creation of innovative treatments that tackle the causes of resistance offer glimpses of hope in the fight against *pneumococcal* infections [95]. MIC for tetracycline ranged between ≤ 0.125 -32µg/ml among isolates of refinery with high effect at 32µg/ml, while ranged between ≤ 0.125 -128µg/ml and most influential MIC was ≤ 0.125 µg/ml among isolates of hospital. This result was widely varied when compared to the result of others [96] who found that clinical isolates of *Streptococcus pneumoniae*, in Italy collected between 1999 and 2002 were resistant to tetracycline with MICs ≥8 µg/ml, it may be due the consequent increase in using of tetracycline since that time led to the development of resistance mechanisms in *Streptococcus pneumoniae.* With respect to the tetracycline, the statistical analyses showed there was a significant difference ($p < 0.05$) between both refinery and hospital strains. Table 24 shows that for Micrococcaceae isolated from refinery (represented for *Stomatococcus mucilagenosus* and *Micrococcus leuteus*), MICs of amikacin and gentamicin ranged between ≤ 0.125-16µg/ml and MICs of tobramycin ranged between 1-8µg/ml, while MICs values for strains isolated from hospital (represented for *Micrococcus leuteus* and *Micrococcus nishinomiyaensis*) were 0.5µg/ml, ≤ 0.125µg/ml and 2µg/ml of amikacin, gentamicin and tobramycin, respectively.

However, the small size of hospital strains samples almost eliminated the statistical difference. These findings were compared with the results of other investigators [97], who found that MIC of amikacin and gentamicin that inhibited 90% of the strains of *Stomatococcus mucilagenosus* ranged between ≤ 0.0.031->16 µg/ml for each one, respectively, while MIC of both that inhibited 90% of the strains of *Micrococcus spp.* ranged between 0.5>16µg/ml and 0.125-2µg/ml, respectively. In general, studies involving the resistance of *Stomatococcus mucilagenosus* to tobramycin are scarce, but other workers [98], found that typical MIC of tobramycin for *Micrococcus spp*. is equal to 20µg/ml. A stark reality in combating antibiotic resistance is the scarcity of new antibiotic agents entering the market. While research has identified potential drug candidates, challenges in drug discovery, development, and regulatory approvals have hindered progress. Recent advances in exploring non-traditional antimicrobial approaches, such as the repurposing of existing drugs and the discovery of novel compounds from natural sources, offer hope for revitalizing the antibiotic pipeline [99]. The petroleum product is a harmful compound that alters the diversity of the soil and aquatic microbial community, leading to bacteria that become “persisters” or “resisters.” When bacterial cells are exposed to petroleum pollutants, two possible scenarios can occur: First, there may be bacteria that develop resistance to the antimicrobial agent, while the non-resistant bacteria can be killed, leaving only the resistant cells. As the resistant bacteria grow back, all bacteria in the culture become resistant. The other possibility is that “persistent” bacteria (dormant, not resistant) may be present. The non-persistent bacteria are killed, leaving only the persistent bacteria. When the persistent cells grow back, the cells that are not in a quiescent state are still susceptible to the antimicrobial attack. This results in a less diverse microbial community dominated by a few resistant and persistent bacteria [100-104]. Molecular identifications have revealed that genera such as Acinetobacter, Alcaligenes, Arthrobacter, Bacillus, Corynebacterium, Micrococcus, Pseudomonas, Methylobacterium, and Rhodococcus are among the most common hydrocarbon degraders. Many of these bacteria also exhibit resistance to heavy metals and antibiotics, alongside biosurfactant producing properties [105-108]. Furthermore, PAH contamination in industrial areas is often coupled with the presence of heavy metals. Heavy metal contamination can negatively affect the bioremediation of hydrocarbons due to the synergistic cytotoxic effects of these dual contaminants. Therefore, searching for bacterial strains that can degrade PAHs while exhibiting high resistance to heavy metals in the soil has become a critical research priority [109-113]. Additionally, the biological degradation capacity of microorganisms is often restricted by the hydrophobic nature of petroleum. While chemical surfactants have been developed to enhance degradation, microbial-derived biosurfactants are more effective for decontaminating oil spills due to their superior biodegradability [114-117].

CONCLUSIONS

Statistical analysis revealed that the differences in MIC distributions between R and H *Staphylococcus aureus* isolates were evaluated separately for each antimicrobial agent using an exact R × C contingency-table test with Monte-Carlo simulation (200,000 replicates), because of sparse cell frequencies. For coagulase negative staphylococci, Table 2 shows that MIC for amoxicillin/ clavulanic acid was ranged

between 2 -64µg/ml among isolates of refinery and between 0.5 -64µg/ml among isolates of hospital. Most of refinery strains were inhibited by 4µg/ml, while most strains of hospital were inhibited by 16µg/ml. The results agreed to some extent. The majority of *Streptococcus pyogenes* strains show MICs values ranged between 1-32 µg/ml, rarely showed < 1 µg/ml. MIC for tetracycline ranged between ≤ 0.125 -64µg/ml among isolates of both refinery and hospital. The MIC distributions of gentamicin and tetracycline against *Streptococcus pyogenes* were compared between refinery and hospital isolates using an exact R × C contingency-table analysis with Monte-Carlo simulation (200,000 replicates). Statistical analysis showed no significant differences in the MIC distributions of gentamicin or tetracycline between refinery and hospital isolates of *Streptococcus pneumoniae*. There were no statistically significant differences in the MIC distributions between refinery and hospital isolates of *Micrococcaceae* for amikacin**,** gentamicin**,** or tobramycin **(**p > 0.05 for all comparisons). Although refinery isolates showed numerically higher median MIC values than hospital isolates for all three antibiotics, these differences did not reach statistical significance.

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