PREVALENCE OF ANTIBIOTIC RESISTANT BACTERIA ISOLATED FROM PHONES

BY

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DECLARATION

I, SHORUMU OPEYEMI SERAH, hereby declare that the project work titled **PREVALENCE OF ANTIBIOTIC RESISTANT BACTERIA ISOLATED FROM PHONES** is a record of an original work done by me, as a result of my research effort carried out in the Departments of Biological Sciences and Biotechnology, Caleb University Imota, Lagos.

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Student's Signature & Date

CERTIFICATION

This is to certify that the research project titled "Prevalence of Antibiotic Resistant Bacteria Isolated from Phones" was carried out by Shorumu Opeyemi Serah with matric number 19/6039 in the Department of Biological Sciences and Biotechnology, College of Pure and Applied Sciences, Caleb University, Imota, Lagos, Nigeria.

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EXTERNAL EXAMINER DATE

DEDICATION

I dedicate this project to the Almighty God for His grace in my life and for making this a success, and also to my irreplaceable parents, Mr. and Mrs. Shorumu for their ceaseless care and support throughout this journey.

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I show my sincere gratitude to the Almighty God for his sustenance, protection and favor throughout this programme. I also want to appreciate my parents Mr and Mrs. Shorumu, my aunty Adetoun and sibling Oluwaseun for their relentless efforts, financial support, guidance and encouragement throughout these years. I appreciate my supervisor Dr. C.C. Ezeanya-Bakpa for her utmost support, guidance and contribution to make me a better researcher and gave my work more innovation, for her ideas, efforts, time, discussions and solutions. I appreciate Mr Ayedun for his relentless efforts and support during this research and I am grateful for the support, attention, love and care my friends and roommates (Damilohun, Gbolahan, Naomi, Tomiwa, Samuel, Favor, Ayomide, Tayo, Timilehin) have shown to me throughout my academic years. I also want to appreciate my course mates who have contributed and given me moral support throughout this research work.

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ABSTRACT

Mobile Phones serve as a means of communication. They can also harbor bacteria, most especially drug-resistant bacteria. The objective of this study is to isolate, identify and determine the antibiotic susceptibility profile of the bacteria isolated from mobile phone samples. A total of 100 mobile phone swab samples from both female and male students of Caleb University was collected. The samples were collected using a sterile swab stick and were cultured on the MacConkey, Mannitol Salt agar, Nutrient agar and Eosin Methylene Blue agar. Pure colonies were further examined using Gram staining and Biochemical testing for identification of the bacterial isolates. Antibiotic susceptibility was done with the disc diffusion method. The questionnaires answered by the students showed 40% of male and 44% of females used hand sanitizer, 64% of male and 68% of females kept long nails, 60% of male and 44% of females had previously used antibiotics. Bacteria was isolated from all phone swabs (100%). The most prevalent bacteria isolates was *Klebsiella pneumoniae* (26%), *Staphylococcus aureus* (18%), *K. oxytoca* (10%), *S. epidermidis* (6%) and *Escherichia coli* (2%) among male samples and *K. pneumoniae* (32%), *S. aureus* (18%), *K. oxytoca* (12%), *Proteus spp.* (10%) and *Micrococcus sp.* (8%) among female samples. The isolates had multidrug resistance with resistance to Erythromycin, Clotrimazole, Ceftazidime, Tetracycline, Cefoxitin, Ceftriaxone and Imipenem. Mobile phones of students are colonized by multidrug resistant bacteria and thus serves as a means of transmitting these bacteria. Good hygiene habits, use of hand sanitizer are preventive methods that can be employed.

CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND OF STUDY

Mobile Phones were established to provide a better communication network and it has also become a part of daily life and are used for a variety of purposes, including conducting business, keeping in touch with family members, listening to music and playing video games. Some individuals carry more than one mobile phone for different purposes.

Drug-Resistant bacteria are bacteria that are resistant to several different antibiotics. Antibiotics are used to treat and prevent further spread of bacterial infection (Centers for Disease Control and Prevention [CDC], 2021). Antimicrobial resistance is increasing at an alarming rate and is due to the inappropriate use of antimicrobials, misuse of antimicrobials contributes to the development of resistance (James *et al.,* 2017). Sometimes the development of antimicrobial resistance may be due to the drug quality i.e the pharmacological quality of the drug, or might be due to prescription of the wrong drug or the dose of the drug (James *et al.,* 2017).

Several researchers have reported the colonization of bacteria on phone surfaces, Some bacteria are pathogenic and are also mesophilic in nature, they grow at the temperature of 20ºC-45ºC, and studies shows that the temperature of the mobile phone is between 25ºC-32ºC on a normal condition but may increase during use to 37ºC-43ºC, thereby making the phone a favourable environment for the bacteria (Savio *et al.,* 2020). More females are comfortable carrying their phones in their bags and purses and the heat generated in these bags also provide support to the growth of bacteria (Savio *et al.*, 2020). These bacteria have developed resistance to antibiotics that were commonly used to treat them and these bacteria can cause serious disease (Martina, Martinez *et al.*, 2019). Examples of drug resistant bacteria that have been isolated from phones

are *Staphylococcus aureus* showing resistant to Benzyl penicillin, *Pseudomonas stutzeri* resistant to Cephalothin, Cefuroxime and Cefoxitin (Vinod, Yahya *et al.,* 2014). *Escherichia Coli*, *Streptococcus pyogenes*, *Salmonella* spp., *Pseudomonas aeruginosa* and *Shigella* spp were 100% resistant to Ampicillin and Cefuroxime. (AL-Harmoosh *et al.,* 2018*)*.

Studies show that overcrowding, failures in cleanliness and inadequate infection control methods can contribute to the development of drug-resistant bacteria between students and in the community. Whereas, some bacteria are spread via student to student contact. Risk factors that promote the contamination of drug-resistant bacteria include; the excessive use of broad spectrum antibiotics, presence of Decubitus ulcers, contaminated humidifiers, delayed diagnosis or treatment, inadequate ventilation (Gopal, 1998). The hygiene habit between teenagers or students is also a risk factor that spreads the contamination of pathogenic bacteria, the contamination of mobile phone surfaces with bacteria is largely due to the microbes on the students' hands (Siiri *et al.,* 2017).

In Caleb University, the use of mobile phones is rapid among students as they use it for various activities; for communication, e-learning and recreationally and it is used in certain areas where bacterial population is high i.e. Clinics, Toilets, Cafeteria. Therefore, the contamination of mobile phones by drug-resistant bacteria within the school environment is expected.

1.2 STATEMENT OF PROBLEM

Mobile phones contaminated with nosocomial pathogens in hospitals have been recognized as potential vectors for transmitting nosocomial germs within the hospital community, but the contamination of students mobile phones with bacteria and the possible occurrence of antibiotic resistant bacteria have not been adequately studied yet (Siiri *et al.,* 2017). Mobile Phones have become a part of students' necessity and are used for various purposes such as entertainment or

educational purposes, despite the positive use of cell phones, they have become a source of addiction and undue reliance on technology among students. Students are becoming addicted to their mobile phones, causing them to use them in unsanitary environments such as the toilets and washrooms. Studies have shown that mobile phones are being used by owners in the toilets, washrooms, kitchen and hospitals which are usually loaded with microorganisms and germs (Al-Asmari *et al.*, 2015). The mobile phone can harbor various potential pathogens and become an exogenous source of infection for the students as well as a health risk for them and their friends or colleagues. The mobile phone can serve as a source of transmission of pathogenic bacteria in a population and subsequently, drug-resistant bacteria which are a threat to human health could emerge (Muktar *et al.,* 2014). In Nigeria, studies have been carried out in different universities on students' mobile phones and at least 62% of the mobile phones are contaminated with drug-resistant bacteria (Akinyemi *et al.,* 2009). Consequently; the prevalence of drug-resistant bacteria isolated from mobile phones of student's in Nigeria is increasing.

1.3 JUSTIFICATION OF THE STUDY

This present study was done to determine the prevalence of drug-resistant bacteria on mobile phones of the students. Although some bacteria found on phones are normal flora of the skin and they may not be harmful, however, there are some harmful bacteria that are also resistant to antibiotics that contaminate mobile phones. Such bacteria can cause both minor and severe diseases ranging from skin infections to diarrhea, pneumonia and meningitis (Al-Abdalall, 2010). Currently, antibiotic resistance is a continued global health concern, resulting in increased morbidity and mortality, longer hospital stays and higher treatment costs (Wanda, 2018).

1.4 AIMS AND OBJECTIVES

AIM: To determine the prevalence of drug-resistant bacteria isolated from mobile phones owned by Caleb University Students.

OBJECTIVES OF PROJECT RESEARCH WORK.

I. To isolate the bacteria from the phones.

II. To characterize and identify the isolates using standard microbiological methods.

III. To determine the antibiotic susceptibility test of the bacteria isolates.

IV. To determine the possible risk factors that promote drug-resistant bacteria on phones'.

CHAPTER TWO

LITERATURE REVIEW

2.1 MOBILE PHONES

Mobile phone is a device that is used to receive and make calls across a radio frequency link while the user is within a telephone service area, Mobile phones are the most frequently used and owned electronic devices worldwide, (Rando *et al.,* 2017). Mobile phones have evolved into one of the most important accessories in both professional and social life. Mobile Phones are frequently handled and held close to the face, despite the fact that they are usually stored in bags or pockets (Akinyemi *et al.*, 2009), phones can also serve as a means of transmitting bacteria pathogens to the owner and to others.

Cell phones are now used almost everywhere, including the dining table, the kitchen, a restaurant, the gym and even the toilet, exposing them to a variety of microorganisms, many microbes are resistant to desiccation and can survive for weeks on phone surfaces. Daily contact with the face, ears and hands may pose a direct health risk of infection from microbe infested cellular phones (Chaibenjawong and Foster, 2011).

2.2 HISTORY OF MOBILE PHONE

Mobile phones were invented in 1973, by John F. Mitchell and Martin Cooper, a senior engineer from Motorola. Cooper believed that a mobile phone is meant to belong to one individual and should be assigned a number, after a span of 10 years, it was brought into market in 1983 and was named Motorola DynaTAC 8000x. The first mobile phones were only allowed to make calls and nothing else. In 1987, the second generation of phone was invented, it was initially named Groupe Special Mobile before it was changed to Global System for Mobile Communications

(GSM). Unlike the first generation phones that used 1G network, GSM made use of 2G network (Jade, 2021).

Different types of phones were invented over the years and today there are different brands of smartphones and digital phones and they run using 4G or 5G networks.

2.3 PREVALENCE OF DRUG-RESISTANT BACTERIA ISOLATED FROM PHONES'

A Study was carried out between February-March 2012 to identify the bacteria associated with mobile phones in Bayero University, Kano, Nigeria, A total of 35 mobile phones were sampled and 28 bacterial isolates of *Escherichia coli*, *Staphylococcus aureus*, *Salmonella species*, and *Klebsiella* species were identified, Antibiotic susceptibility test of the isolates was done and *S. aureus* had the highest Susceptibility to Ciprofloxacin, Salmonella to Augmentin and *Klebsiella* was sensitive against Gentamicin and Streptomycin. *Salmonella species* were also resistant against Gentamicin, Perfloxacin and Streptomycin (Kawo and Musa, 2013).

150 mobile phones were randomly selected from 150 healthcare workers in three teaching hospitals in Kerman, Iran, 50 from each hospitals, the swabs were cultured in the laboratory using Blood agar and Eosin methylene blue (EMB) agar, incubated aerobically at 37ºC for 48 hours, then the samples were examined for antimicrobial activity for commonly used antimicrobials using disc diffusion method, *Staphylococcus epidermidis* was the most commonly cultured organism from all sites and the organism had resistance of 6.7% for Cephalothin and 25% for Amoxicillin, therefore mobile phones could be a source of nosocomial infection in these healthcare and also spread drug resistance bacteria in the settings (Gholamreza, Nooshin *et al.,* 2009).

In the University of Ghana, Korle-Bu Campus, Accra, 120 swab samples were collected from the surfaces of mobile phones of Healthcare students. The swabs were cultured using MacConkey agar, Blood agar and Mannitol salt agar, Bacteria identification was done and the bacterial isolates were tested against 9 commonly used antibiotics by the kirby-Bauer disc method. The bacteria isolated included *Staphylococcus epidermidis* (40.1%)*, Klebsiella species* (20.4%)*, Staphylococcus aureus* (14.5)*, Escherichia coli* (10.5%)*, Pseudomonas spp.* (8.6%) and *Enterobacter species* (2.6%). The bacterial isolates were highly resistant to ampicillin and tetracycline, moderately resistant to chloramphenicol and lower resistant to cefotaxime, ceftazidime, ciprofloxacin and gentamicin (Michael, Christian *et al.*, 2021).

In a Specialized Hospital in Addis Ababa, Ethiopia. 27.1% *Klebsiella pneumoniae,* 14.6% *E. coli* and 14.6% *Acinetobacter spp* was isolated from 454 out of 572 mobile phones and they were highly resistant to ampicillin (95.8%), piperacillin (83.3%), cotrimoxazole (70.8%) and chloramphenicol (54.2%) and greatly sensitive to meropenem (87.5), amikacin (85.4%) (Shambel, Kassu *et al.,* 2021).

106 samples were collected from the mobile phones of patients in different hospitals of Jazan province of Saudi Arabia and 89 phones were contaminated with bacteria. 49.0% coagulase-negative *Staphylococcus*, 11.3% *Staphylococcus aureus*, 6.6% *Enterobacter cloacae,* 2.83% *Pseudomonas stutzeri* and 1.8% *Enterococcus faecalis* were isolated from the samples. Coagulase-negative *Staphylococcus* strains were resistant to benzylpenicillin, rifampicin, erythromycin and gentamicin, *S. aureus* strains were resistant to benzylpenicillin and erythromycin, *E. cloacae* complex strains were resistant to ampicillin, cefoxitin and cefuroxime, *P. stutzeri* strains were resistant to cephalothin, cefotaxime, cefoxitin and trimethoprim (Vinod *et al.,* 2014).

In a tertiary care hospital in Saudi Arabia, 38.7% *Staphylococcus aureus*, 10.4% *Klebsiella pneumonia,* 8.6% *Acinetobacter baumannii*, 5.5% *Escherichia coli* and 5.5% *Enterobacter cloacae* were isolated from mobile phone, high resistant rate was observed and 88.9% *E. coli* isolates were resistant to ampicillin, 55.6% to ciprofloxacin, ceftazidime and 44.4% were resistant to cefotaxime, cefoxitin. *K. pneumoniae* was 94.1% resistant to ampicillin, 52.9% to ceftazidime, 58.8% to ciprofloxacin, 47.1% to tetracycline and 52.9 % to cefoxitin. *E. cloacae* was 100% resistant to ceftazidime, 33.3 % was resistant to imipenem and 66.7% to ciprofloxacin. 35.7% *A. baumannii* isolates were resistant to gentamicin, 57.1% to imipenem (Asmari *et al.,* 2015).

2.4 PREVALENCE OF BACTERIA FROM STUDENT'S PHONES

Several studies have been done around the world by different researchers to see the prevalence of bacteria isolated from student's phones and if these bacteria are resistant to antibiotics.

In the University of Lafia, Nigeria, 70 mobile phones belonging to students were swabbed and cultured to detect microbial contamination, there was 51.4% bacterial contamination amongst female and 48.6% among male, the organisms that were isolated are *Escherichia specie, Salmonella specie, Bacillus sp., Pseudomonas sp., Staphylococcus sp., Klebsiella sp., Micrococcus sp.* and *Enterobacter sp.* (Ya'aba *et al.,* 2020).

In India, 4 mobile phones were randomly selected from college students in Government College for Women, Trivandrum. There was a bacteria growth in all four samples that were cultured, three samples belonged to Bacillus genus and one belonged to Pseudomonas genus. Antibiotic susceptibility test was carried out and multidrug resistance bacteria was not found among the pathogens (Praveen, Aswathy, 2014).

In the University of Baghdad, 20 samples (10 from males and 10 from females) were collected from students in the Biology department, College of Science. There was 100% growth in all samples, and six genera bacteria were identified from the cultures, Gram positive bacteria; *Staphylococcus epidermidis, Streptococcus pyogenes, Staphylococcus aureus, Bacillus spp* and Gram negative bacteria: *Pseudomonas aeruginosa, Escherichia coli.* The gram positive bacteria had 82.77% and gram negative bacteria had 17.33%.

40 mobile phones were collected in Alexandria University students' in Egypt and tested for bacterial contamination, 53% methicillin-resistant *Staphylococcus aureus,* 43% Bacillus, 18% methicillin-susceptible *Staphylococcus aureus* (MSSA), 13% *E. coli,* 8% *Klebsiella pneumoniae* and 3% *Acinetobacter baumannii* were isolated from the samples.

In the University of Cape Coast, Ghana, 100 samples were randomly collected from students and bacteria isolates including 10% *Klebsiella pneumonia,* 4% *Staphylococcus aureus,* 4% *Pseudomonas aeruginosa*, 3% *Salmonella spp.,* 2% *Shigella spp.* and 8% *Escherichia coli*.

2.5 ANTIMICROBIAL RESISTANCE

Antimicrobial Resistance occurs when bacteria, viruses, parasites and fungi evolve over time and lose their ability to respond to antibiotics, making infections more difficult to treat and increasing the risk of disease transmission, severe illness and death (World Health Organization, 2021).

Antimicrobial resistant pathogens that have developed new resistance mechanisms, leading to antimicrobial resistance continue to pose a threat to our ability to treat common infections. The rapid global spread of multi and pan resistant bacteria, which cause infections that are resistant to existing antimicrobial medicines such as antibiotics is particularly concerning.

Without effective tools for preventing and treating drug resistant infections, as well as improved access to existing and new quality-assured antimicrobials, the number of people who fail to respond to treatment or die from infections will rise.

2.6 ANTIBIOTIC RESISTANCE

Antibiotic resistance is the ability of bacteria to change or protect themselves in such a way antibiotics cannot kill them. Antibiotic resistance in bacteria is frequently developed as a result of unnecessary and inappropriate antibiotic use (Salih and Ali, 2013)

2.6.1 TYPES OF ANTIBIOTIC RESISTANCE

1. Natural (Intrinsic) resistance: this type of resistance is caused by bacteria's structural characteristics and is unrelated to antibiotic use, it does not have a hereditary property and it develops as a result of natural resistance, microorganisms lacking the target antibiotic's structure or antibiotics failing to reach their target due to their characteristics. For example, Gram negative bacteria are naturally resistant to vancomycin because vancomycin does not penetrate through the outer membrane, *Klebsiella* spp. Which is intrinsically resistant to Ampicillin because it produces β-lactamase enzymes that destroy the drug.
2. Acquired resistance: This type of resistance is caused by chromosomal or extrachromosomal structures (plasmid, transposon), because it is no longer affected by the antibiotics it was susceptible to before, Acquired resistance develops as a result of changes in the genetic characteristics of bacteria and it is found only in some strains of a bacterial species and it required laboratory methods for detection.
    a. Chromosomal Resistance: A mutation in chromosomal DNA causes a microorganism's drug resistance (Gooch, 2011). Physical (e.g. Ultraviolet etc.)

and chemical factors can cause such mutations, this could be the result of bacterial cell structural changes.

b. Extrachromosomal Resistance: swapping genetic material between neighboring bacteria. Plasmids, transposons and integro act as vectors for transferring antibiotic resistance genes in a variety of ways to other similar bacterial species. The genes are transferred via three ways: Transformation, Transduction and Conjugation (Clewell, 2014).

3. Cross resistance: is when bacteria develops resistance to an antibiotic that have a similar mechanism of action and resistance to other drugs, it is most common in antibiotics with similar structures such as resistance between cephalosporins and penicillins (Saliu and Ali, 2013), nalidixic acid and ciprofloxacin.
4. Multi-drug resistance: They are usually bacteria that have developed resistance to the antibiotics used to treat them. This indicates that a specific drug is no longer effective in killing or controlling the bacteria. Antibiotics were used inappropriately for treatment resulting in the selection of pathogenic bacteria that were resistant to multiple drugs.

2.7 ANTIBIOTIC RESISTANT BACTERIA

They are those bacteria that cannot be controlled or killed by antibiotics. In the presence of an antibiotic, they can survive and even multiply. Most bacteria that cause infections can develop resistance to at least some antibiotics. Antimicrobial resistance in bacterial pathogens is a global problem with high morbidity and mortality rates. Multidrug resistance patterns in Gram-positive and Gram-negative bacteria have resulted in infections that are difficult to treat, if not impossible to treat with traditional antimicrobials (Marianne *et al.,* 2016). The bacteria may have developed an ability to stop the medicine's effect, developed an ability to pump the drug out of the cell or

mutate. Bacteria resistance is accelerated when bacteria are forced to adapt due to the presence of too much antibiotics. Examples of bacteria resistant to antibiotics include methicillin-resistant *Staphylococcus aureus* (MRSA), penicillin-resistant *Enterococcus,* vancomycin-resistant *Enterococcus* (VRE), multi-drug resistant *Mycobacterium* tuberculosis (MDR-TB).

2.8 MECHANISM OF RESISTANCE TO ANTIBIOTICS

Antibiotic resistance can develop in bacteria through a number of genetic mechanisms. These mechanisms result in biochemical changes that alter certain bacterial cell properties that normally make the cell susceptible to antibiotics, resulting in resistance.

1. Antibiotic Sequestration: Sequestration is caused by the action of drug-binding proteins which prevents the antibiotic from reaching its target. The primary mechanism of resistance in producers of antibiotics in the bleomycin family is sequestration of the metal-bound or metal-free antibiotic (Peterson and Kaur, 2018) by binding proteins BlmA and ZbmA in , *Streptomyces verticillus* and *S. flavoviridis.* Each bleomycin family member has one or more genes related to ATP-binding cassette (ABC) transporters in their biosynthesis clusters which may be used to remove the antibiotics bound to binding proteins.
2. Antibiotic Modification or Inactivation: Antibiotic Modification is a common method of rendering antibiotics ineffective, Resistance genes may encode enzymes that chemically modify an antimicrobial, rendering it inactive, or hydrolyse an antimicrobial to destroy it. This mechanism leads to resistance to a wide range of antimicrobials, particularly in aminoglycoside antibiotics (such as kanamycin, gentamicin and streptomycin), chloramphenicol and β-lactams.

3. Target Modification: Antimicrobial medications have extremely specific targets, and structural alterations to those targets can prevent the drug from attaching to the cell, rendering it ineffective. Bacteria have an advantage that allows them to develop resistance to drugs through spontaneous mutations in the genes encoding antibacterial drug targets. Target modification acts as a self-resistance mechanism to antibiotics such as β-lactams, glycopeptides, macrolides, lincosamides and streptogramins (MLS), as well as aminoglycosides.
4. Prevention of Cellular Uptake or Antibiotic Efflux: It is another mechanism of self-resistance, although it usually occurs together with other mechanisms such as modification of the antibiotic. Microbes can develop resistance mechanisms that prevent antimicrobial drugs from reaching their cellular targets by preventing them from accumulating. Changes in outer membrane lipid composition, porin channel selectivity and porin channel concentrations are all typical among gram-negative pathogens in this technique. Furthermore, many gram positive and gram negative pathogenic bacteria produce efflux pumps, which actively transport an antimicrobial medication out of the cell and prevent the chemical from building up to an antibacterial level.
5. Limiting drug uptake: The bacteria has the capacity to limit the uptake of antibiotics, the structure of the lipopolysaccharide layer of gram negative bacteria helps provide an obstacle for the bacteria against some molecule, this makes the bacteria resistant to some certain antibiotics. Bacteria such as mycobacteria have a high lipid outer membrane that gives antimicrobial agents like hydrophobic drugs easier access to enter into the cell while hydrophilic drugs have limited access. Gram positive bacteria do not possess a cell wall, therefore restrictions to drugs are not common (Wanda, 2018).

2.9 MECHANISM OF BACTERIA RESISTANCE ISOLATED FROM PHONES

Escherichia coli is resistance to Ciprofloxacin. *Escherichia coli* is a pathogenic organism that often causes sepsis and diarrhoea (Mengchen *et al.,* 2019).

Resistance to Ciprofloxacin by Chromosomal Target Site Mutations

In *E. coli*, the major target of ciprofloxacin is the gyrase, which is made up of two GyrA subunits and two GyrB subunits. In Gram positive bacteria, topoisomerase IV is a secondary target and two ParC and two ParE subunits make up this enzyme (Laurent *et al.,* 2018). The first ciprofloxacin resistance markers to be discovered were mutations in GyrA, most mutations that reduce ciprofloxacin susceptibility were found within the quinolone resistance-determining region, which is located in GyrA between Ala67 and Gln107 and in ParC it is from Ala64 to Gln103. As mutations in GryA and ParC accumulate, the minimum inhibitory concentration of ciprofloxacin increases simultaneously. These mutations cause little or no ciprofloxacin susceptibility (Boas, Daniel *et al.,* 2019).

S. aureus resistance to Ciprofloxacin.

Resistance arises from spontaneous amino acid changes in one or both of the enzymes required for DNA replication, DNA gyrase and Topoisomerase IV. High level fluoroquinolone resistance is conferred by mutations in a short section of these enzymes called the quinolone-resistance determining region (QRDR). In *S. aureus*, resistance is conferred by point mutations in the topoisomerase IV subunit ParC and the DNA gyrase subunit GyrB. The majority of MRSA strains have two mutations, one in ParC and the other in GyrA. Over-expression of an efflux pump known as NorA also contributes to the resistance trait in some strains (Wanda, 2013).

Klebsiella pneumoniae resistance to ceftazidime

The organism produces β-lactamase genes, especially the extended spectrum β-lactamase. The organism is resistant to broad spectrum cephalosporins.

2.10 SUSCEPTIBILITY OF DRUG RESISTANT BACTERIA ISOLATED FROM PHONES

From the studies observed, these drug resistant bacteria that were isolated from the samples are sensitive to some antibiotics:

Streptococcus epidermidis was sensitive against Streptomycin, Kanamycin, Amoxicillin (Enass, 2018), *Escherichia coli* was sensitive to Cetriazone, Ofloxacin, Pefloxacin, Ciprofloxacin (Enass, 2018). *Staphylococcus aureus* was sensitive against Pefloxacin, Ciprofloxacin, Ofloxacin, Streptomycin (Kawo and Musa, 2013). *Klebsiella pneumoniae* was sensitive against Cetriazone, Gentamicin, Ofloxacin, and Streptomycin. *Pseudomonas aeruginosa* was sensitive to Ciprofloxacin, Cetriazone, Ofloxacin, and Amoxicillin. *Salmonella* species were sensitive against Augmentin, Amoxicillin, and Ciprofloxacin. *Streptococcus pyogenes* was sensitive against Gentamicin, Ciprofloxacin, Kanamycin (Enass, 2018).

2.11 PATHOGENESIS AND CLINICAL MANIFESTATION OF BACTERIAL INFECTION

Staphylococcus aureus: is often considered to be an opportunistic pathogen and it is responsible for a wide range of human infections and disorders. Abscesses are a common symptom of *S. aureus* skin and tissue infections, and they form partly to contain the infections' nidus (Scott *et al,* 2015). *S aureus* expresses many cell surface-associated and extracellular proteins that are potential virulence factors (Timothy, 1996). They invade the defense mechanisms of the host skin, mucosal or tissue surfaces and cause tissue damage, for the majority of diseases caused by

this organism, pathogenesis is multifactorial. *S. aureus* produces disease by multiplying in tissues, Stimulating inflammation and liberating toxins (Timothy, 1996).

Staphylococcus aureus can cause superficial skin lesions like boils, styes, serious infections like endocarditis, pneumonia, meningitis and osteomyelitis in immunosuppressed patients. It can also cause food poisoning by producing enterotoxins into the food (Foster, 1996).

Escherichia coli: the pathogenesis of *E. coli* in host tissue includes colonization of the mucosal site, evasion of host defence, multiplication and host damage (James *et al.,* 2004). It causes infections such as; pyogenic infections, Intra-Abdominal Infection, peritonitis, Septicemias, Gastroenteritis (Kaper *et al.,* 2004).

Streptococcus pyogenes: It is a member of the normal flora. Pharyngitis, Scarlet fever (rash), Impetigo, Cellulitis and erysipelas are all symptoms of *Streptococcus pyogenes* infections. Necrotizing fasciitis, myositis and streptococcal toxic shock syndrome are among possible outcomes of invasive infections. Immune mediated complications such as acute rheumatic fever and acute glomerulonephritis can also occur (Patterson, 1996). M protein for attachment to the host cell, a hyaluronic acid capsule that prevents phagocytosis, and other extracellular products (Patterson, 1996).

Pseudomonas aeruginosa: Multiple bacterial virulence factors that enhance adhesion and disrupt host cell signaling pathways while targeting the extracellular matrix are involved in pathogenesis in P. aeruginosa. Various adhesions and secreted toxins, proteases, effector proteins, and pigments that facilitate adhesion, modify or disrupt host cell pathways, and target the extracellular matrix are involved in P. aeruginosa pathogenesis. Infections caused by Pseudomonas include pneumonia, endocarditis, infections of the eyes, ears and skin (Alhazmi, 2015).

Klebsiella pneumoniae: It is an opportunistic pathogen responsible for a wide range of infections (Guentzel, 1996), the bacteria spreads in the hosts' respiratory tract and cause pneumonia or in the host's blood causing an infection in the bloodstream such as meningitis, it also spreads from person-to-person through contact with contaminated hands. Symptoms include high fever and jelly-like sputum, shortness of breath and coughing, nausea and dizziness.

Streptococcus pneumoniae: It is a normal member of the respiratory tract flora, it is usually in the host mucosal surface of the nasal cavity, and from there it migrates to the lungs, where it causes pneumonia. It also causes meningitis and occult bacteremia (Lavida, 2018; Patterson, 1996). It remains the major cause for serious focal and systemic infections, it also causes sinusitis and conjunctivitis beyond childhood.

Salmonella: Salmonella ingested through food survive the gastric acid barrier and invade the mucous membrane of the small and large intestine, where they produce toxins. The invasion of epithelial cells triggers the release of proinflammatory cytokines, resulting in an inflammatory response. The bacteria can spread from the intestines to the rest of the body, resulting in systemic diseases. It causes infections like diarrhea, abdominal cramp, enteric fever, septicemia and gastroenteritis (Giannella, 1996).

Shigella: the ingestion of shigella causes infection via faecal-oral contamination, as it passes through the small intestine, the enterotoxins produced there may cause diarrhea. Inflammatory colitis and bacterial invasion of colonic epithelium are the hallmarks of shigellosis. Symptoms of shigellosis are abdominal pain, tenesmus, watery diarrhea, fever, vomiting and dysentery (Hale, 1996).

2.12 VIRULENCE FACTORS

Virulence factors are bacteria associated molecules that are required for a bacteria to cause disease while infecting eukaryotic hosts such as humans. It is the ability of an organism to cause disease to its host tissues (Aditya *et al.,* 2017).

2.12.1 *Staphylococcus aureus:*

The number of virulence genes are in the *S. aureus* genome; it has many cell-surface proteins that promote the colonization of host tissues. *S. aureus* produces many virulence factors such as toxins, capsule, Adhesins, Nucleases, Coagulase (Mandala, 2019), exoenzymes, hemolysins, leukocidins, proteases, lipase, phospholipase and enterotoxins which enables it to be a pathogen (Yuichi *et al.,* 2011).

2.12.2 *Escherichia coli:*

Virulence factor produced by *E. coli* is divided into two; Bacterial cell surface and secreted virulence factors. The bacterial cell surface virulence factor includes fimbriae, which helps the bacteria attach to the host cell surface, invade the tissue, biofilm formation and cytokine induction, the cell surface virulence factors also includes flagellum, capsular lipopolysaccharide and the secreted virulence factors include Siderophores, Exotoxins, Hemolysins, Enterotoxins (causes Diarrheas), Vero Cytotoxins (Chhaya *et al.,* 2019).

2.12.3 *Klebsiella pneumoniae:*

Capsular Antigen, Lipopolysaccharide, Capsular polysaccharide which creates resistance to phagocytosis and Fimbriae are the major recognised virulence factors of *K. pneumoniae* (Duyen *et al.,* 2017).

2.12.4 *Streptococcus pyogenes:*

Virulence factors include surface protein M, streptolysins, streptodornase, streptokinase, hyaluronidase, streptolysin, peptidoglycan (Golinska *et al*, 2016).

2.12.5 *Pseudomonas aeruginosa:*

Strains of *Pseudomonas* all have endotoxins, which is a major virulence factor in bacteremia and septic shock (Iglewski, 1996). Lipopolysaccharide, flagellum, Type IV Pili, Exotoxin A, Proteases, Biofilm production and Quorum Sensing are also virulence factors of *P. aeruginosa* (Alhazmi, 2015).

2.12.6 *Streptococcus pneumoniae:*

The most important virulence factor is the polysaccharide capsule, which protects the bacteria from phagocytosis (Patterson, 1996). Pneumolysin, is a toxin that binds to the host cells with cholesterol and forms pores in the cell membrane which leads to cell lysis. Autolysin, an enzyme that promotes the colonization of bacteria due to the release of toxins such as pneumolysin. Other virulence factors include pneumococcal surface protein (Proteins A, C), Pili, Hydrogen Peroxide and Biofilms (Lavida, 2018).

2.12.7 *Salmonella*

They must have the ability to invade the host's cells, replicate intracellularly and production of toxins (endotoxin, enterotoxin, cytotoxin), biofilm, fimbriae and pili for adhesion, virulence plasmid (Noor, 2021).

2.12.8 *Shigella*

Ipa proteins are the important virulence factors in shigella, which consists of IpaA, IpaB, IpaC and Ipa6D. IpaB is an essential factor for phagosome escape and macrophage apoptosis. *Shigella* also produces toxins which includes; *Shigella* enterotoxin 1, *Shigella* enterotoxin 2 and Shiga

toxin. *Shigella* enterotoxin 1 and *Shigella* enterotoxin 2 have the ability to secrete fluid into the intestine and therefore causing watery diarrhea. Shiga toxin is an exotoxin that is responsible for the development of vascular wounds in the kidney and central nervous system (Shih-Chun, Chi-Feng *et al.,* 2015).

2.13 RISK FACTORS

The Risk factors include excessive use of broad spectrum antibiotics, presence of Decubitus ulcers, contaminated humidifiers, delayed diagnosis or treatment, inadequate ventilation (Gopal, 1998). The hygiene habit between teenagers or students is also a risk factor that spreads the contamination of pathogenic bacteria, the contamination of mobile phone surfaces with bacteria is largely due to the microbes on the students' hands (Siiri *et al.,* 2017). Hand Hygiene prior treatment with antibiotics, prolonged hospitalisation, prolonged ICU stay and mechanical ventilation renal dysfunction, older age, surgical procedures and ICU admission (Rebekah *et al*., 2018) are also risk factors to antibiotic resistant bacteria. Other causes of antibiotic resistant bacteria include when an antibiotic is over prescribed or used, when people do not take antibiotics as directed, poor hygiene and lack of prevention and control and when bacteria receives resistant genes from other bacteria.

2.14 MODE OF TRANSMISSION

Bacteria can be transmitted from one place to another, from the human skin to the phone, the inanimate objects or surfaces the phones touch or through hand to hand contact,

S. epidermidis is a normal flora of the human skin, *Staphylococci* are also found on clothes and bed linen (Al-Abdalall, 2010).

S. aureus is found mostly on the skin and in the nose, it can also be transferred from the hand into food during preparation (Centers for Disease Control and Prevention [CDC], 2021). Some

enteric organisms are found on the toilet seat in which they can also be transferred to the phones when taken to the toilet. The hand serves a major role in the transmission of pathogens.

2.14.1 NASAL, ORAL OR OCULAR TRANSMISSION

The colonization of bacteria in students' nasal nares can be transmitted to mobile phones by hands (Chang *et al.,* 2017). Bacteria can spread to the hands through rubbing the eyes, sneezing or coughing.

2.14.2 FOOD TRANSMISSION

Bacteria can be transmitted from raw food like Chicken, Beef, Turkey, Pork, Potato and Vegetables to the hands, skin or mouth. The Bacteria on the hands are then transferred to other uncooked foods like salad, fresh fruits (Jana *et al.,* 2018).

2.14.3 HAND TRANSMISSION

Infection transmission through contaminated hands of students is a common means of spreading infection. Foods can be contaminated with bacteria through unclean hands which could be ingested (Al-Abdalall, 2010). Bacteria can also be spread via contact with infected animals or animal contaminated surfaces which could be transmitted to phones (Doron *et al.,* 2008).

2.14.4 TOILET TRANSMISSION

Phone users somewhat use their phone in the toilet. This likely gets more germs on the surfaces through contact with the toilet seat, paper roll or door knob. Pathogenic enteric bacteria are mostly transferred to the phones by the users via their hands, skin and other parts of the body (Al-Abdalall, 2010).

2.15 IDENTIFICATION OF BACTERIA ISOLATED FROM PHONES

2.15.1 Morphology:

The morphology of bacteria is incredibly diverse. Adaptive pressures that optimize bacterial fitness result in certain morphologies (Muriel *et al*, 2017). Morphology of bacteria varies in terms of shapes, sizes and structures. The size of a bacteria ranges from 0.3μm to 0.7mm. Bacteria have three major shapes; rod shape (bacillus), spherical shape (coccus), and spiral shape (vibrio). The structure of bacteria is based on the arrangement of cells, it may be single, paired (diplo), grape-like clusters (staphylo) or in chains (strepto) (Moshtaq, 2016)

Staphylococcus aureus: They are Gram positive bacteria and appear in a spherical shape, are in grape-like clusters and they range from 0.5-1.0 μm in size (Arumugam *et al.,* 2017).

Escherichia coli: are Gram negative bacteria, they are rod shape, are in singles or in pairs and are about 2.0- 6.0 μm in length and 1.1-1.5 μm wide (Steven and David, 2014).

Klebsiella pneumoniae: They are Gram negative, rod shaped bacteria and are about 3-4 mm in diameter (Liu *et al.,* 2019).

Streptococcus pyogenes: They are Gram positive, spherical bacteria, are in long chains and are about 0.6-1.0 μm in diameter (Patterson, 1996).

Shigella species: They are Gram negative, small rods of about 0.3-1 μm in diameter and 1-6 μm in length, non-motile and non-spore forming. They appear in singles, pairs and in chains.

Salmonella species: They are Gram negative, rod shaped bacteria, are about 1-3 μm in length and 0.5-0.6 μm in diameter. They are in singles or in pairs.

Pseudomonas aeruginosa: They are Gram negative, rod shaped bacteria. They are about 0.5- 0.8 μm in diameter and 1.5-3.0 μm in length.

2.15.2 General Cultural and Biochemical Characteristics:

Bacteria on a culture media grow as colonies. There are different colony morphologies, and the characteristics used to describe the morphology includes; size, shape, colour, texture and height, in terms of shape, the bacteria colony can be circular, irregular, punctiform or rhizoid. Size of a colony on an agar plate can be small, medium or large. Texture of a bacterial colony can be dry, mucoid or moist. In terms of color of the bacteria colony, some bacteria produce pigments when they grow in a medium e.g. *Staphylococcus aureus* produce a yellow pigment on Mannitol Salt agar. The height or elevation of a bacterial colony can be flat, raised, convex or unbonate (Chesebrough, 2000).

Staphylococcus aureus: is an aerobic organism, forms large yellow or white colonies on nutrient agar, haemolytic in blood agar, grows on mannitol salt agar producing a yellow pigment, is catalase positive, produces coagulase enzyme, produces citrate enzyme and is oxidase negative (Arumugam *et al.,* 2017).

Escherichia coli: is an aerobic organism, it grows on MacConkey agar producing a pink colony as it is a lactose fermenter, produces a green metallic sheen on EMB agar, it is catalase positive, oxidase negative, citrate negative and is urease negative (Holt *et al.*, 1994).

Klebsiella pneumoniae: is facultative anaerobic bacteria, produces mucoid, pinkish colonies on MacConkey agar i.e. Lactose fermenters, it is positive for glucose, mannitol, sucrose, catalase, citrate and urease. It is also Indole negative and oxidase negative (Chesebrough, 2000).

Streptococcus pyogenes: is facultative anaerobic bacteria, catalase negative, α-haemolytic cocci, it has complete haemolysis on blood agar, non-motile bacteria and urease negative (Chesebrough, 2000).

Shigella species: is a facultative anaerobic bacteria, catalase positive, urease negative, oxidase negative, on MacConkey agar the colonies are small, circular and colourless and on EMB agar

the colonies are colorless due to lack of lactose fermentation and on *Salmonella Shigella* agar the colonies are colorless (Chesebrough, 2000).

Salmonella species: is a facultative anaerobic bacteria, there is no haemolysis on blood agar, on MacConkey agar, the colonies are colorless, on EMB agar the colonies are colorless and on *Salmonella Shigella* agar the colonies are colorless with black centre. It is catalase positive, citrate negative, indole negative, urease negative and oxidase negative (Chesebrough, 2000).

Pseudomonas aeruginosa: is an obligate aerobic organism, producing a large, opaque, flat colony and also small round colonies. It is colorless on MacConkey agar, it is oxidase positive, catalase positive, indole negative (Chesebrough, 2000).

2.16 PREVENTION AND CONTROL

Usage of antibiotics only when prescribed by a health professional, always follow the doctor's advice while using the antibiotics, regular washing of hands and good personal hygiene. Prepare food hygienically, don't use leftover antibiotics, use alcohol-based hand sanitizer, complete the entire course of the prescribed antibiotic so that it can be fully effective and not produce resistance (World Health Organization, 2021).

CHAPTER THREE

MATERIALS AND METHODS

3.1 STUDY SETTING

Caleb University is a private University, located in Ikorodu-Itokin Road, Imota, Lagos and it has over 4,000 Students' Population. Caleb University has a peaceful environment that is ideal for learning and character development.

3.2 STUDY POPULATION

Fifty mobile phones belonging to 50 Students, were randomly selected from 25 male and 25 female Students.

3.3 SAMPLE COLLECTION

Two sets of swab samples were collected from the front and back of mobile phones belonging to 25 male and 25 female students respectively, across different Departments in the University. The swab sample was collected by rubbing a sterile swab stick against the front and back of the phones. The swab sticks were immersed into a sterile peptone water prior analysis.

A self-structured questionnaire was used to collect demographic data such as age, gender, family size and department. Socio-behavioural characteristics were also collected from the participants.

3.4 IDENTIFICATION AND ISOLATION OF BACTERIA

The Pure isolate colonies were identified using the Gram staining technique, Biochemical tests and Cultural methods.

3.4.1 Gram Staining technique

It is a technique done to characterize bacteria into Gram positive and Gram negative bacteria likewise their morphology.

This technique was done according to the methods of Chesebrough (2000) whose method involved a thin smear of the isolate smeared on a clean glass slide and fixed by passing it through the flame. The slide was overlaid with crystal violet solution for 1 minute and washed off with distilled water. Then the slide was overlaid with Gram's Iodine Solution for 1 minute and washed off with distilled water. Few drops of Acetone-Alcohol Decolourizer was added on the surface of the smear and washed off with running water, then the smear was further overlaid with Safranin Counterstain for 30 seconds; washed off with running water and allowed to air dry. The slide was then examined under oil immersion objective.

3.4.2 Biochemical test

It is a test used for identifying bacterial species based on differences in metabolic activity. The following are the biochemical tests that will be used:

3.4.2.1 Sugar Fermentation test

This technique was done according to the methods of Chesebrough (2000) whose method involved identifying microorganisms that are able to ferment sugars such as: glucose, fructose, galactose, lactose, mannitol and sucrose. It was used to differentiate organisms based on carbohydrate fermentation patterns. Using an Inoculating loop, a colony was picked from a pure culture, the colony was inoculated into the sugar by shaking the loop in it. The cap was closed and incubated for 18-24 hours at 35ºC-37ºC in atmospheric air.

3.4.2.2 Indole test

This test was carried out according to the technique of Chesebrough (2000) whose method involved characterising bacteria that can decompose amino acid tryptophan to create indole. This test is important in the identification of members of *Enterobacteriaceae.* Four (4ml) of tryptophan broth was used in a sterilised test tube, a small amount of the pure culture was

inoculated into the test tube containing tryptophan broth and incubated at 37ºC for 24 to 48 hours. Five drops of Kovac's reagent was added into the broth culture and observed for the presence or absence of a ring.

3.4.2.3 Simmon's citrate test

This test was carried out according to the technique of Chesebrough (2000) whose method involved determining if an organism has the ability to utilise citrate as a source of energy. Using Simmon's citrate agar that was already in a slanted position in a test tube, a single colony was picked from the pure culture using an inoculating needle. The inoculum was streaked back and forth on the slanted agar and incubated aerobically at 35-37ºC for 4-7 days.

3.4.2.4 Urease test

This technique was done according to the methods of Chesebrough (2000) whose method was used for determining organisms that can hydrolyse urea and produce ammonia and carbon dioxide. Decarboxylation of amino acids produces Urea, Urea is hydrolysed to produce ammonia and CO2. The formation of ammonia alkalizes the medium changing the colour from light orange to magenta pink, positive organisms turn the entire medium pink within 24 hours and negative organisms produce no colour change. An inoculating loop was used to pick an inoculum from the pure culture and streaked on the surface of the urea agar slant. The cap was left loosely and incubated at 35ºC-37ºC for 48 hours to 7 days.

3.4.2.5 Catalase test

This test was carried out to detect the presence of the enzyme Catalase. It was used to differentiate bacteria that produces the enzyme "Catalase" such as *Staphylococci*, *Salmonella, Cryptococcus, Micrococci, Klebsiella, Pseudomonas* from non-catalase producing bacteria such as *Streptococci* and *Enterococcus* species.

This technique was done according to the methods of Chesebrough (2000) whose method involved adding two drops of hydrogen peroxide on a clean microscopic slide. A sterile loop was used to pick a small amount of organism from the pure culture and emulsified in the hydrogen peroxide on the slide.

3.4.2.6 Motility test

This test was carried out to determine the ability of an organism to move by itself, this technique was done according to the methods of Chesebrough (2000) whose method involved using a semi solid agar (a half strength nutrient agar).

A sterile inoculating needle was used to pick a colony of bacteria and was stabbed at the centre of the tube containing the agar to over half the depth, it was then incubated at 35ºC-37ºC for 48 hours. The motile organisms grew out of the line of inoculation and the non-motile organisms grew only along the line of inoculation.

3.4.2.7 Hemolysis test

This test was carried out to determine the ability of an organism to produce hemolysins, an enzyme that damages red blood cells. This technique was done according to the methods of Chesebrough (2000) whose method involved an inoculating loop used to pick a colony of the organism and streaked on a blood agar plate, the plate was incubated at 35ºC-37ºC for 24 hours. The plate was then inspected for signs of beta, alpha and gamma hemolysis. Beta hemolysis indicated by a complete hemolysis, a clear zone surrounding the colonies, alpha hemolysis indicated by a green, opaque zone and gamma hemolysis indicated by no zone around the colonies.

3.4.3 Cultural methods

The growth media used were according to the methods of Ya'aba *et al.* (2020) and they are; Nutrient agar (HiMedia Laboratories pvt. Ltd, India), Mannitol Salt Agar (HiMedia Laboratories pvt. Ltd, India), MacConkey agar (HiMedia Laboratories pvt. Ltd, India), Blood agar and Eosin Methylene Blue (EMB) agar (Biomark lab, India). Immediately the swab was collected, it was inoculated into a sterilised peptone water and further plated on any of the mentioned media above. The plate was then incubated aerobically at 37ºC for 18-24 hours.

3.5 ANTIMICROBIAL SUSCEPTIBILITY TEST

Antimicrobial susceptibility testing was carried out for all bacterial isolates using the Disk Diffusion method on Muller Hinton agar (HiMedia Laboratories pvt. Ltd, India) with different antibiotic disc. The antibiotics used were; Ciprofloxacin (5 µg), Tetracycline (30 µg), Gentamicin (10 µg), Cefoxitin (30 µg), Ceftazidime (30 µg), Clotrimazole (50 µg), Erythromycin (15 µg), Amoxicillin-Clavulanic acid (30 µg), Ceftriaxone (30 µg), Cefotaxime (30 µg) and Imipenem (10 µg) (Liofilchem s.r.l., Italy). Results were interpreted using the Clinical and Laboratory Standard Institute (CLSI, 2020) standard inhibition zones after measuring the zones of inhibition.

0.5 McFarland standard was used to prepare the inoculum, a sterile swab stick was used to inoculate the inoculum on the Mueller Hinton agar plate vertically and horizontally, making sure it is inoculated everywhere on the plate. The antibiotics disc were aseptically mounted on the plates, the plates were incubated at 37ºC for 18-24 hours. After incubation the plates were read and the zone of inhibition was measured in millimeters.

3.5.1 Detection of methicillin resistant *Staphylococcus aureus*

Resistance of the isolates to the fourth generation cephalosporin, Cefoxitin, was used as a substitute marker for the detection of methicillin-resistant *Staphylococcus aureus* and this technique was done according to the methods of Clarence *et al.* (2005).

3.5.2 Double disk synergy test

This test was carried out to detect the production of ESBL by members of *Enterobacteriaceae*. This technique was done according to the methods of Ezeanya *et al*., (2017) whose method was carried out by using a disc of amoxicillin-clavulanate (30 µg) along with two third generation cephalosporins; ceftazidime (30 µg) and cefotaxime (30 µg). An amoxicillin-clavulanate disc was placed in the center of the plate and the other two discs were placed 15mm apart, in the center to that of amoxicillin-clavulanate disc.

3.6 STATISTICAL ANALYSIS

Descriptive statistics was used to represent number and percentage. Mean and standard deviation was used to represent the age of the subject. T-test was used to determine the association between the predisposing factors and drug-resistant bacteria isolated from the mobile phones.

CHAPTER FOUR

RESULTS

One hundred swab samples were collected from the back and front of the mobile phones of students.

A total of 50 students (25 males and 25 females) mobile phones were screened for drug resistant bacteria. The characteristics of the study population shows that majority of the male students (68%) were between 20-24 years unlike the 36% (9) female students that were between 20-24 years as shown in Table 4.1 and majority of the female students (60%) were between 15-19 years unlike the 32% (8) male students that were between 15-19 years.

Activities of the students that exposes them into harboring bacteria on their phone, and in the study it was found that 44% of the female students sometimes use hand sanitizer, 68% of male students use their phones in the toilet, 100% of both male and female students touch their phone always, all students reported to clean their phones but we had 19 (76%) to clean their phones once a while and the other responses given by the participants will be summarized in Table 4.2.

TABLE 4.1: Age differences between the study populations

Parameters		**Male n (%)**	**Female n (%)**
Age	15-19	8 (32%)	15 (60%)
	20-24	17 (68%)	9 (36%)
	25-29	0 (0%)	1 (4%)
Family size	1-3	0 (0%)	5 (20%)
	4-6	21(84%)	16 (64%)
	7-9	4 (16%)	3 (12%)
	9-Above	0 (0%)	1 (4%)

Table 4.2: Data of activities derived from questionnaires answered by students

Parameters	**Male n (%)**	**Female n (%)**
Touching of phone	25 (100%)	25 (100%)
Keeping of long nails	16 (64%)	17 (68%)
Usage of phone in toilet	17 (68%)	15 (60%)
Washing of hands after toilet	21 (84%)	22 (88%)
Washing with soap and water	17 (68%)	16 (64%)
Usage of phone while eating	12 (48%)	16 (64%)
Sharing of phone	11 (44%)	10 (40%)
Use of alcohol based hand sanitizer	10 (40%)	11 (44%)
Mobile storing places	Pockets 23 (92%) Bags 1 (4%) Hands 1 (4%)	Pockets 0 (0%) Bags 17 (68%) Hands 8 (32%)
Cleaning of phone	Twice a week 6 (24%) Once a week 0 (0%) Once a while 19 (76%)	Twice a week 4 (16%) Once a week 4 (16%) Once a while 17 (68%)
Use of nose masks	24 (96%)	24 (96%)
Use of antibiotics	15 (60%)	11 (44%)
Touching of face and nose	24 (96%)	25 (100%)

Bacteria was isolated from the mobile phones of all 25 male and female students respectively. Isolates with blue stained bacterial cells and pinkish stained bacterial cells were reported Gram positive and Gram negative respectively (Table 4.3). For the biochemical identification of the isolates; Sugar fermentation test result indicated by change of color from red to yellow was interpreted as positive while no change was interpreted as negative. Other biochemical tests such as Motility test, Indole test, Citrate, Urease test had isolates positive and negative for them (4.4-4.5).

The cultural characteristics of the isolates shown in (Table 4.6). The percentage of isolated bacteria from mobile phones of students were *Klebsiella pneumoniae* (26%) being the most isolated organism in the male samples and 32% *Klebsiella pneumoniae* isolated from female samples, *Staphylococcus aureus* with the same number 9 (18%) in both gender and *E. coli* 1 (2%) was isolated in male sample and no isolate of the organism in female sample (Table 4.7).

The test demonstrated that isolated bacteria were 100% resistant to Erythromycin and Clotrimazole, isolates of *Staphylococcus aureus* were sensitive to Ceftazidime, Ceftriaxone, Gentamicin and Ciprofloxacin and resistant to Tetracycline, while *E. coli* was sensitive to Tetracycline, Ciprofloxacin and Gentamicin and resistant to the remaining antibiotics used (Table 4.8).

Table 4.3: Cellular morphology of Bacterial isolates

Samples	Gram	Shape	Samples	Gram	Shape
MP1F	-ve	Rod	FP1F	-ve	Rod
MP1B	+ve	Cocci	FP1B	-ve	Rod
MP2F	+ve	Cocci	FP2F	-ve	Rod
MP2B	+ve	Cocci	FP2B	-ve	Rod
MP3F	-ve	Rod	FP3F	-ve	Rod
MP3B	+ve	Cocci	FP3B	+ve	Cocci
MP4F	-ve	Rod	FP4F	+ve	Cocci
MP4B	-ve	Rod	FP4B	-ve	Rod
MP5F	-ve	Rod	FP5F	+ve	Cocci
MP5B	+ve	Rod	FP5B	-ve	Rod
MP6F	-ve	Rod	FP6F	-ve	Rod
MP6B	-ve	Cocci	FP6B	-ve	Rod
MP7F	+ve	Irregular	FP7F	+ve	Cocci
MP7B	+ve	Cocci	FP7B	+ve	Cocci
MP8F	-ve	Rod	FP8F	-ve	Rod
MP8B	-ve	Rod	FP8B	+ve	Cocci
MP9F	-ve	Rod	FP9F	+ve	Cocci
MP9B	-ve	Rod	FP9B	-ve	Rod
MP10F	+ve	Cocci	FP10F	-ve	Rod
MP10B	+ve	Cocci, cluster	FP10B	-ve	Rod
MP11F	+ve	Cocci	FP11F	-ve	Rod
MP11B	+ve	Cocci	FP11B	-ve	Rod
MP12F	-ve	Rod	FP12F	-ve	Rod
MP12B	+ve	Cocci	FP12B	-ve	Rod
MP13F	+ve	Cocci	FP13F	-ve	Rod
MP13B	-ve	Rod	FP13B	-ve	Rod

Samples	Gram	Shape	Samples	Gram	Shape
MP14F	-ve	Rod	FP14F	-ve	Rod
MP14B	+ve	Cocci	FP14B	-ve	Rod
MP15F	-ve	Rod	FP15F	+ve	Cocci
MP15B	+ve	Cocci	FP15B	+ve	Cocci
MP16F	+ve	Cocci	FP16F	-ve	Rod
MP16B	+ve	Cocci	FP16B	-ve	Rod
MP17F	-ve	Rod	FP17F	+ve	Cocci
MP17B	-ve	Rod	FP17B	-ve	Rod
MP18F	+ve	Cocci	FP18F	-ve	Rod
MP18B	+ve	Cocci	FP18B	-ve	Rod
MP19F	-ve	Rod	FP19F	-ve	Rod
MP19B	+ve	Cocci	FP19B	+ve	Cocci
MP20F	-ve	Rod	FP20F	+ve	Cocci
MP20B	-ve	Rod	FP20B	+ve	Cocci
MP21F	-ve	Rod	FP21F	-ve	Rod
MP21B	-ve	Rod	FP21B	+ve	Cocci
MP22F	-ve	Rod	FP22F	-ve	Rod
MP22B	+ve	Cocci	FP22B	-ve	Rod
MP23F	-ve	Rod	FP23F	-ve	Rod
MP23B	+ve	Cocci	FP23B	-ve	Rod
MP24F	+ve	Cocci	FP24F	-ve	Rod
MP24B	-ve	Rod	FP24B	+ve	Cocci
MP25F	-ve	Rod	FP25F	-ve	Rod
MP25B	-ve	Rod	FP25B	-ve	Rod

KEY: -ve=Negative, +ve= Positive

Table 4.4: Biochemical tests of isolates from male students' phone

Isolate code	Catalase	Hemolysis	Motility	Urease	Citrate	Indole	Lactose	Sucrose	Fructose	Glucose	Galactose	Mannitol	Suspected Organism
MP1F	+	γ	+	-	-	+	+	+	+	+	-	+	*Escherichia coli*
B	+	β	-	+	+	+	-	+	-	+	-	-	*Staphylococcus epidermidis*
MP2F	+	γ	-	+	+	+	+	+	+	+	+	+	*Staphylococcus aureus*
B	+	β	-	+	+	+	-	+	-	-	+	-	*Staphylococcus intermedius*
MP3F	+	α	-	+	+	-	-	+	+	+	-	+	*Klebsiella pneumoniae*
B	+	α	-	+	+	+	+	+	+	+	-	+	*Micrococcus luteus*
MP4F	+	γ	-	+	+	-	-	+	+	+	-	+	*Klebsiella pneumoniae*
B	+	γ	-	+	+	+	-	+	+	+	-	+	*Klebsiella oxytoca*
MP5F	+	γ	-	+	-	-	-	-	-	+	-	-	*Proteus mirabilis*
B	+	γ	-	+	+	-	-	-	+	+	-	-	*Aerobacter sp.*
MP6F	+	γ	+	+	+		+	-	+	+	-	-	*Proteus sp.*
B	+	β	-	-	+	ND	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
MP7F	+	α	+	+	+	-	-	+	+	+	-	-	*Micrococcus sp.*
B	+	γ	-	+	+	+	-	+	+	+	+	-	*Staphylococcus sp.*
MP8F	+	β	-	+	-	+	+	+	+	+	+	+	*Klebsiella oxytoca*
B	+	β	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
MP9F	+	α	-	+	+	+	+	+	+	+	-	+	*Klebsiella oxytoca*

Isolate code	Catalase	Hemolysis	Motility	Urease	Citrate	Indole	Lactose	Sucrose	Fructose	Glucose	Galactose	Mannitol	Suspected Organism
B	+	A	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Klebsiella sp.*
MP10F	+	β	-	+	+	-	-	+	+	+	+	-	*Micrococcus sp.*
B	+	γ	-	+	+	+	-	-	+	+	-	-	*Staphylococcus epidermidis*
MP11F	+	γ	-	+	+	-	-	+	+	+	+	+	*Staphylococcus aureus*
B	+	α	-	+	+	ND	-	-	-	+	-	-	*Staphylococcus aureus*
MP12F	+	α	-	-	+	-	-	+	-	+	-	+	*Klebsiella sp.*
B	+	α	-	+	+	+	-	+	+	+	-	-	*Staphylococcus aureus*
MP13F	+	α	-	+	+	-	-	-	-	+	+	-	*Enterobacter aerogenes*
B	+	β	-	+	-	-	-	+	-	+	+	-	*Klebsiella pneumoniae*
MP14F	+	α	+	+	+	-	-	+	+	+	-	-	*Klebsiella pneumoniae*
B	+	γ	-	-	+	+	+	+	-	+	-	-	*Enterobacter aerogenes*
MP15F	+	γ	-	+	-	-	+	+	+	+	-	+	*Klebsiella pneumoniae*
B	+	α	-	+	-	ND	+	+	+	+	-	+	*Enterobacter aerogenes*
MP16F	+	γ	+	+	+	+	+	-	+	+	-	-	*Staphylococcus epidermidis*
B	+	α	-	+	+	-	+	+	+	+	+	+	*Staphylococcus sp.*
MP17F	+	β	-	+	+	+	+	+	+	+	+	+	*Klebsiella oxytoca*
B	+	γ	+	+	+	+	+	+	+	+	+	+	*Klebsiella pneumoniae*
MP18F	+	α	-	+	+	+	-	+	+	+	-	+	*Staphylococcus aureus*

Isolate code	Catalase	Hemolysis	Motility	Urease	Citrate	Indole	Lactose	Sucrose	Fructose	Glucose	Galactose	Mannitol	Suspected Organism
B	+	α	+	+	+	ND	-	+	+	+	-	-	*Staphylococcus aureus*
MP19F	+	α	-	+	-	-	+	-	+	+	-	+	*Klebsiella pneumoniae*
B	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	*Staphylococcus aureus*
MP20F	+	β	-	+	-	ND	ND	ND	ND	ND	ND	ND	*Klebsiella oxytoca*
B	+	γ	-	+	-	ND	ND	ND	ND	ND	ND	ND	*Proteus sp.*
MP21F	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
B	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	*Klebsiella sp.*
MP22F	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	*Klebsiella sp.*
B	+	γ	-	+	-	ND	ND	ND	ND	ND	ND	ND	*Staphylococcus aureus*
MP23F	+	α	-	+	+	-	+	+	+	+	-	+	*Klebsiella pneumoniae*
B	+	γ	ND	+	+	ND	+	-	+	+	+	+	*Staphylococcus aureus*
MP24F	+	γ	-	+	+	ND	-	+	+	+	-	-	*Micrococcus sp.*
B	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
MP25F	+	β	-	+	+	-	-	+	+	+	+	+	*Klebsiella pneumoniae*
B	+	α	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Klebsiella sp.*

KEY: +=Positive, - = Negative, α= Alpha hemolysis, β= Beta hemolysis, γ= Gamma hemolysis,
ND= Not done

Table 4.5: Biochemical tests of isolates from female students' phone

Isolate code	Catalase	Hemolysis	Motility	Urease	Citrate	Indole	Lactose	Sucrose	Fructose	Glucose	Galactose	Mannitol	Suspected Organism
FP1F	+	β	-	+	-	-	+	+	+	-	-	+	*Klebsiella pneumoniae*
B	+	β	-	+	+	+	+	-	+	+	+	+	*Citrobacter diversus*
FP2F	+	β	-	+	-	+	+	+	+	+	-	+	*Klebsiella oxytoca*
B	+	β	-	+	-	-	-	+	+	+	+	+	*Klebsiella pneumoniae*
FP3F	+	β	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
B	+	α	-	+	+	+	-	+	+	-	-	+	*Micrococcus sp.*
FP4F	+	β	-	+	-	-	+	+	+	+	-	+	*Staphylococcus sp.*
B	+	β	-	+	+	+	+	+	+	+	-	+	*Klebsiella oxytoca*
FP5F	+	α	-	+	+	-	ND	ND	ND	ND	ND	ND	*Staphylococcus aureus*
B	+	β	-	+	+	+	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
FP6F	+	β	-	+	+	-	+	+	-		-	+	*Klebsiella pneumoniae*
B	+	α	-	+	+	+	ND	ND	ND	ND	ND	ND	*Klebsiella sp.*
FP7F	+	α	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Staphylococcus aureus*
B	+	α	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Staphylococcus aureus*
FP8F	+	β	-	+	+	-	-	-	+	+	-	-	*Proteus sp.*
B	+	γ	-	+	+	+	+	-	+	+	-	-	*Micrococcus sp.*
FP9F	+	β	-	+	+	ND	ND	ND	ND	+	ND	ND	*Staphylococcus aureus*
B	+	β	-	-	+	ND	+	+	-	+	+	+	*Klebsiella oxytoca*
FP10F	+	β	-	+	+	-	-	-	+	+	+	-	*Proteus sp.*

Isolate code	Catalase	Hemolysis	Motility	Urease	Citrate	Indole	Lactose	Sucrose	Fructose	Glucose	Galactose	Mannitol	Suspected Organism
B	+	γ	-	+	+	-	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
FP11F	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	*Klebsiella sp.*
B	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
FP12F	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	*Klebsiella sp.*
B	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	*Klebsiella sp.*
FP13F	+	γ	-	+	+	-	-	+	+	+	-	-	*Proteus sp.*
B	+	γ	-	+	-	ND	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
FP14F	+	γ	-	+	+	ND	-	ND	ND	ND	ND	ND	*Proteus sp.*
B	+	γ	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
FP15F	+	α	-	+	+	+	+	+	+	+	-	ND	*Staphylococcus aureus*
B	+	γ	-	+	-	-	+	-	+	+	-	+	*Staphylococcus sp.*
FP16F	+	α	-	+	+	ND	-	+	+	+	-	ND	*Klebsiella pneumoniae*
B	+	α	-	+	-	+	ND	ND	ND	ND	ND	ND	*Klebsiella oxytoca*
FP17F	+	β	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Staphylococcus aureus*
B	+	α	-	+	+	+	ND	ND	ND	ND	ND	ND	*Proteus sp.*
FP18F	+	γ	-	+	+	+	-	ND	ND	+	ND	ND	*Klebsiella pneumoniae*
B	+	γ	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
FP19F	+	β	-	+	+	+	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
B	+	γ	-	+	+	-	ND	ND	ND	ND	ND	ND	*Staphylococcus aureus*
FP20F	+	α	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Staphylococcus aureus*
B	+	α	-	+	-	ND	ND	ND	ND	ND	ND	ND	*Micrococcus sp.*
FP21F	+	γ	-	+	+	+	-	-	+	+	+	+	*Citrobacter diversus*

Isolate code	Catalase	Hemolysis	Motility	Urease	Citrate	Indole	Lactose	Sucrose	Fructose	Glucose	Galactose	Mannitol	Suspected Organism
B	+	γ	-	+	-	-	-	+	+	+	-	-	*Staphylococcus aureus*
FP22F	+	γ	+	+	+	ND	+	+	-	+	-	+	*Enterobacter cloacae*
B	+	α	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*
FP23F	+	β	-	+	-	ND	ND	ND	ND	ND	ND	ND	*Klebsiella oxytoca*
B	+	α	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Klebsiella sp.*
FP24F	+	α	-	+	-	+	ND	ND	ND	+	ND	ND	*Klebsiella oxytoca*
B	+	γ	-	+	+	ND	ND	ND	ND	ND	ND	ND	*Micrococcus sp.*
FP25F	+	αα	-	+	-	-	ND	ND	ND	+	ND	ND	*Klebsiella pneumoniae*
B	+		-	-	+	+	ND	ND	ND	ND	ND	ND	*Klebsiella pneumoniae*

KEY: +=Positive, -=Negative, α= Alpha hemolysis, β= Beta hemolysis, γ= Gamma hemolysis, ND= Not done

Table 4.6: Colonial Morphology of Bacteria Isolates of Male and Female Students

Isolate code	MacConkey Agar (MAC)	Mannitol Salt Agar (MSA)	Nutrient Agar (NA)	Eosin Methylene Blue Agar (EMB)	Suspected organisms
MP1F	pinkish, round	NG	whitish,mucoid, irregular	green shein, mucoid, pink	*Escherichia coli*
B	pinkish,mucoid, Irregular (60)	NU	Creamy, mucoid	pink, round, mucoid (3)	*Staphylococcus epidermidis*
MP2F	pink,round,mucoid(32)	Pink,yellow pigment	whitish, mucoid, irregular	pink, round, mucoid (22)	*Staphylococcus aureus*
B	pink,round,mucoid(14)	Pink, round	mucoid, creamy, irregular	pink, mucoid irregular (3)	*Staphylococcus intermedius*
MP3F	colorless,mucoid, irregular	NG	whitish, mucoid, irregular	NG	*Klebsiella pneumoniae*
B	round, pinkish, elevate,mucoid (1)	NG	creamy, mucoid, irregular	NG	*Micrococcus luteus*
MP4F	pink,yellow pigment (4) round,mucoid,elevated	NG	whitish, mucoid, irregular	Deep pink, mucoid,irregular (3)	*Klebsiella pneumoniae*
B	Pink, mucoid, round (5)	NG	creamy, mucoid, irregular, flat	pink,dark,flat,center, irregular, mucoid (4)	*Klebsiella oxytoca*
MP5F	pink,round,mucoid, elevated, yellow pigment (23)	NG	creamy, mucoid, irregular	deep pink, flat, mucoid (25)	*Proteus mirabilis*
B	pink,round,mucoid, elevated (8)		whitish, mucoid, irregular	pink, mucoid, irregular (1)	*Aerobacter sp.*
MP6F	pink (13x100)	creamy, mucoid	whitish, mucoid	light pink (25x100)	*Proteus sp.*
B	pink,mucoid (52x100)	pink, dry, flat	greyish, white	light pink, mucoid	*Klebsiella pneumoniae*

Isolate code	MacConkey Agar (MAC)	Mannitol Salt Agar (MSA)	Nutrient Agar (NA)	Eosin Methylene Blue Agar (EMB)	Suspected organisms
MP7F	pink (24x10)	NU	NU	pink, irregular	*Micrococcus sp.*
B	pink (30x30)	creamy, mucoid, yellow pigment	NU	pink, round	*Staphylococcus sp.*
MP8F	pink, mucoid, round (3)	NG	creamy,small (10) mucoid, irregular	NG	*Klebsiella oxytoca*
B	pink, mucoid, round (5)	NG	creamy, mucoid irregular,small (6)	NG	*Klebsiella pneumoniae*
MP9F	pink, mucoid,round (7)	NG	NU	light pink, moist	*Klebsiella oxytoca*
B	pink, round (4)	NG	NU	light pink, moist	*Klebsiella sp.*
MP10F	light pink, dry	white, mucoid, yellow pigment	creamy, mucoid (50x5)	light pink, dry (35x5)	*Micrococcus sp.*
B	light pink, dry	creamy, mucoid (25x10)	creamy, dry	light pink, dry	*Staphylococcus epidermidis*
MP11F	light pink, mucoid (27)	yellow	creamy, mucoid (24x5)	yellow, round	*Staphylococcus aureus*
B	light pink, mucoid (16x10)	yellow	creamy (15x3)	colorless	*Staphylococcus aureus*
MP12F	pink, dry (16x20)	NG	Creamy,yellow (22x10)	NG	*Klebsiella sp.*
B	pink, dry (32)	yellow pigment	creamy (20x5)	NG	*Staphylococcus aureus*
MP13F	light pink, dry (20x10)	NG	NU	light pink, dry darkcenter (30x5)	*Enterobacter aerogenes*
B	colorless (30x10)	NG	NU	pink, dry, dark center (70x10)	*Klebsiella pneumoniae*

Isolate code	MacConkey Agar (MAC)	Mannitol Salt Agar (MSA)	Nutrient Agar (NA)	Eosin Methylene Blue Agar (EMB)	Suspected organisms
MP14F	light pink, dry (21x10)	NG	NU	pink, dark center, (40x3)	*Klebsiella pneumoniae*
B	colorless, dry (37x10)	NG	NU	pink, dry, darkcenter (40x5)	*Enterobacter aerogenes*
MP15F	pink,round,raised mucoid (7x30)	mucoid, round, large,whitish (15)	round, creamy, flat, mucoid (60)	round, raised, pink, dark center, mucoid	*Klebsiella pneumoniae*
B	pink,round,raised mucoid (10x20)	creamy, round, mucoid (1)	round, creamy, flat, mucoid	pink, round,moist, dark center, flat	*Enterobacter aerogenes*
MP16F	pink, round, mucoid (6x21)	creamy, round (2) mucoid,yellow (1)	round, mucoid, creamy, flat	pink, round, raised, mucoid (20x6)	*Staphylococcus epidermidis*
B	pink, round, cluster, mucoid, flat (8x24)	mucoid, round, large, whitish (1)	creamy, flat, mucoid, round	pink, round, raised, mucoid (17x8)	*Staphylococcus sp.*
MP17F	light pink, round, elevated (17x9)	whitish, round,pink, mucoid, large	creamy, flat, mucoid, round	pink,mucoid,round, dark center (96)	*Klebsiella oxytoca*
B	pink, small, round, raised, mucoid	creamy, dry, round, mucoid	round, mucoid, creamy (85)	pink, round, small, dark center, mucoid	*Klebsiella pneumoniae*
MP18F	pink, small, round, elevated (59x2)	yellowish, mucoid (3)	mucoid, creamy elevated (20x7)	pink, large, mucoid, dark center, round	*Staphylococcus aureus*
B	pink, round, yellow pigment (80)	yellow, large, mucoid	creamy, mucoid, round (24x7)	pink, round, large, dark center (24x3)	*Staphylococcus aureus*
MP19F	pink, raised, mucoid (10x7)	creamy,mucoid, large,yellow pigment (3)	creamy, round, raised, mucoid	pink, mucoid, raised (60)	*Klebsiella pneumoniae*
B	pink, raised, mucoid (12x12)	mucoid, creamy, large, round	NU	round, raised, pink (4x12)	*Staphylococcus aureus*

Isolate code	MacConkey Agar (MAC)	Mannitol Salt Agar (MSA)	Nutrient Agar (NA)	Eosin Methylene Blue Agar (EMB)	Suspected organisms
MP20F	pink, round, raised, mucoid (22x6)	small, round, creamy, mucoid (2)	NU	pink,raised,mucoid, dark center, round	*Klebsiella oxytoca*
B	pink, round, raised, mucoid (24x6)	mucoid, creamy, irregular (5)	NU	pink,mucoid,round, dark center, raised	*Proteus sp.*
MP21F	pink,small, mucoid (21)	NG	NU	pink, large, mucoid (44)	*Klebsiella pneumoniae*
B	pink, mucoid, yellow pigment (52)	whitish, round, mucoid (13)	NU	pink, large, mucoid (53)	*Klebsiella sp.*
MP22F	irregular, mucoid, flat pink (37)	NG	NU	pink, round, flat, dark center	*Klebsiella sp.*
B	round, mucoid, pink (42)	creamy, round yellow pigment	NU	pink, round, flat, dark center	*Staphylococcus aureus*
MP23F	pink, mucoid, round, raised (24x8)	creamy,mucoid, small, (4) yellow pigment,	NU	pink, round, raised, mucoid	*Klebsiella pneumoniae*
B	pink, raised, mucoid (15x8)	creamy, mucoid round (2)	NU	pink, round, mucoid, raised	*Staphylococcus aureus*
MP24F	light pink, mucoid, irregular (55)	NG	NU	pink, mucoid, large, round	*Micrococcus sp.*
B	light pink, mucoid, round, small (40x3)	NG	NU	pink, mucoid, small, round	*Klebsiella pneumoniae*
MP25F	light pink,mucoid, irregular (20x2)	NG	NU	pink, mucoid, flat, large, irregular	*Klebsiella pneumoniae*
B	light pink, mucoid, round, small (50x3)	NG	NU	pink, mucoid, irregular	*Klebsiella sp.*
FP1F	pink, mucoid, (5)	NG	creamy, round, mucoid (3)	pink,mucoid, irregular,elevated (1)	*Klebsiella pneumoniae*

Isolate code	MacConkey Agar (MAC)	Mannitol Salt Agar (MSA)	Nutrient Agar (NA)	Eosin Methylene Blue Agar (EMB)	Suspected organisms
B	round, light pink	NG	creamy, mucoid (3)	NG	*Citrobacter diversus*
FP2F	pink, mucoid, round (2)	NG	whitish, irregular,	pink, mucoid, irregular (2)	*Klebsiella oxytoca*
B	round, mucoid, pink, elevated (8)	NG	whitish, irregular,	NG	*Klebsiella pneumoniae*
FP3F	Pink, round, mucoid (5)	NG	whitish, flat, mucoid	NG	*Klebsiella pneumoniae*
B	pinkish,mucoid, irregular	NU	creamy, mucoid (20)	pink, dark center mucoid, irregular	*Micrococcus sp.*
FP4F	NG	yellow,mucoid, round	creamy, irregular	NG	*Staphylococcus sp.*
B	Round,mucoid, pink, yellow pigment (2)	NG	whitish,irregular	pinkish, mucoid round, elevate (5)	*Klebsiella oxytoca*
FP5F	pink, dry (65)	yellow, mucoid	NU	Transparent-colorless	*Staphylococcus aureus*
B	pink (54)	NG	NU	pink	*Klebsiella pneumoniae*
FP6F	pink, round (15)	NG	NU	light pink	*Klebsiella pneumoniae*
B	Pink, round	NG	NU	light pink	*Klebsiella sp.*

Isolate code	MacConkey Agar (MAC)	Mannitol Salt Agar (MSA)	Nutrient Agar (NA)	Eosin Methylene Blue Agar (EMB)	Suspected organisms
FP7F	pink, dry (36x5)	yellow pigment,mucoid	whitish, dry	pink, moist	*Staphylococcus aureus*
B	pink, dry	NG	whitish	colorless	*Staphylococcus aureus*
FP8F	pink, round, mucoid (16x7)	pink, flat, mucoid (11x12)	NU	pink, clusters, flat, dark center, mucoid	*Proteus sp.*
B	pink, clusters, round, mucoid	pink, flat, mucoid (8x15)	NU	pink, mucoid, flat, round, dark center	*Micrococcus sp.*
FP9F	pink, dry (10x10)	white, mucoid	creamy, dry	pink, moist	*Staphylococcus aureus*
B	pink, dry (31x5)	light pink, mucoid	yellow, mucoid	pink	*Klebsiella oxytoca*
FP10F	pink, round, mucoid, raised (7x20)	NG	NU	flat, pink, mucoid, round, dark center	*Proteus sp.*
B	pink, round, raised, mucoid	NG	NU	round, pink, flat dark center, mucoid	*Klebsiella pneumoniae*
FP11F	pink,irregular, yellow pigment, mucoid (34)	NG	whitish, mucoid, round	pink, elevated, round, large (38)	*Klebsiella sp.*
B	pink, irregular, dry, flat (140)	NG	mucoid, round, white, small	pink, small, round (25x4)	*Klebsiella pneumoniae*
FP12F	small, round, pink, yellow pigment (160)	NG	creamy, round, small, mucoid	pink, large, flat, dry dark center, round	*Klebsiella sp.*

Isolate code	MacConkey Agar (MAC)	Mannitol Salt Agar (MSA)	Nutrient Agar (NA)	Eosin Methylene Blue Agar (EMB)	Suspected organisms
B	yellow pigment,pink small, round (20x7)	NG	creamy, small, dry, irregular	small, mucoid, dark center, pink, flat (10x5)	*Klebsiella sp.*
FP13F	pink, yellow pigment (23x10)	NG	flat, mucoid, whitish	pink, dry, small, dark center (60)	*Proteus sp.*
B	pink, yellow pigment (80)	creamy, mucoid, round, yellow pigment (3)	mucoid, whitish, yellow	pink, dry, small, dark center (33x4)	*Klebsiella pneumoniae*
FP14F	flat, mucoid, light pink (90)	NG	creamy, small, mucoid	mucoid, round, pink, (37x4)	*Proteus sp.*
B	pink, mucoid yellow pigment (115)	NG	creamy, flat, small, mucoid	pink, round, small, dry (150)	*Klebsiella pneumoniae*
FP15F	light pink, mucoid, yellow pigment, small	yellowish (12)	creamy, round, small, mucoid	pink, large, round, moist, dark center (66)	*Staphylococcus aureus*
B	pink, large, mucoid, round (32)	whitish, round, mucoid (5)	creamy, small, round, mucoid	pink, round, mucoid, dark center, small	*Staphylococcus sp.*
FP16F	pink, flat, mucoid, round (27)	whitish, round, flat mucoid (10)	creamy, mucoid, round	pink, raised, round, mucoid, (35)	*Klebsiella pneumoniae*
B	pink, mucoid, flat, round (20)	whitish, mucoid, elevated, irregular (4)	creamy, round, raised	pink, round, large, mucoid, (55)	*Klebsiella oxytoca*
FP17F	pink, mucoid, large, round (5)	mucoid, round, creamy, yellow pigment (8)	NU	pink, round, elevated, mucoid, large	*Staphylococcus aureus*
B	pink, mucoid, round, yellow pigment (41)	round, moist (3)	NU	pink, round, mucoid, small (58)	*Proteus sp.*

Isolate code	MacConkey Agar (MAC)	Mannitol Salt Agar (MSA)	Nutrient Agar (NA)	Eosin Methylene Blue Agar (EMB)	Suspected organisms
FP18F	light pink, dry, small, round (48)	creamy, mucoid, round, small (1)	NU	pink, mucoid, round, large (26)	*Klebsiella pneumoniae*
B	pink, yellow pigment, small,round (57)	creamy, mucoid,round, yellow pigment,small (9)	NU	pink, round, dark center (66)	*Klebsiella pneumoniae*
FP19F	light pink, mucoid, (8) yellow pigment, round	creamy, moist, round (1)	NU	round, mucoid, pink, small (7)	*Klebsiella pneumoniae*
B	light pink, small, round, yellow pigment, mucoid (58)	creamy, small, mucoid, round (16)	NU	mucoid, round, pink, small (9)	*Staphylococcus aureus*
FP20F	light pink, small, round, yellow pigment, mucoid (46)	mucoid, round, small, moist (7)	NU	mucoid, pink, round, large (54)	*Staphylococcus aureus*
B	light pink, mucoid, round, small (17)	NG	NU	mucoid, small, pink, irregular (19)	*Micrococcus sp.*
FP21F	light pink, mucoid, round (5)	NG	NU	pink, mucoid, small, round (9)	*Citrobacter diversus*
B	light pink, small, mucoid (23)	yellow pigment, round, small, (27)	NU	pink, mucoid, small, round (35)	*Staphylococcus aureus*
FP22F	light pink, mucoid, flat (8x10)	NG	NU	pink, mucoid, flat	*Enterobacter cloacae*
B	light pink, mucoid, irregular, flat (30)	NG	NU	pink, mucoid, flat	*Klebsiella pneumoniae*

Isolate code	MacConkey Agar (MAC)	Mannitol Salt Agar (MSA)	Nutrient Agar (NA)	Eosin Methylene Blue Agar (EMB)	Suspected organisms
FP23F	light pink, mucoid	NG	NU	pink,mucoid, irregular	*Klebsiella oxytoca*
B	light pink, mucoid, irregular	NG	NU	pink,mucoid, irregular	*Klebsiella sp.*
FP24F	light pink, mucoid	NG	NU	pink,mucoid, round	*Klebsiella oxytoca*
B	light pink, mucoid	NG	NU	pink,mucoid, irregular	*Micrococcus sp.*
FP25F	pink, mucoid, round, flat	NG	NU	pink, mucoid, dark center	*Klebsiella pneumoniae*
B	pink, mucoid, flat	NG	NU	pink, mucoid, dark center	*Klebsiella pneumoniae*

KEY: NG= No growth, NU= Not used.

Table 4.7: Organisms suspected ratio and their frequency by gender

Suspected organisms	Male n (%)	Female n (%)
Escherichia coli	1 (2%)	0 (0%)
Staphylococcus epidermidis	3 (6%)	0 (0%)
Staphylococcus aureus	9 (18%)	9 (18%)
Staphylococcus intermedius	1 (2%)	0 (0%)
Klebsiella pneumoniae	13 (26%)	16 (32%)
Micrococcus luteus	1 (2%)	0 (0%)
Klebsiella oxytoca	5 (10%)	6 (12%)
Proteus mirabilis	1 (2%)	0 (0%)
Aerobacter sp.	1 (2%)	0 (0%)
Proteus sp.	2 (4%)	5 (10%)
Micrococcus sp.	3 (6%)	4 (8%)
Staphylococcus sp.	2 (4%)	2 (4%)
Klebsiella sp.	5 (10%)	5 (10%)
Enterobacter aerogenes	3 (6%)	0 (0%)
Citrobacter diversus	0 (0%)	2 (4%)
Enterobacter cloacae	0 (0%)	1 (2%)
Total	50 (100%)	50 (100%)

Table 4.8: Antimicrobial Susceptibility Test

SAMPLES	ORGANISM ISOLATED	ANTIBIOTICS ZONE OF INHIBITION (MM)										
		AUG (30µg)	CAZ (30µg)	CTX (30µg)	IMI (10µg)	CRO (30µg)	CLO (50µg)	FOX (30µg)	TE (30 g)	E (15µg)	CIP (5µg)	CN (10µg)
M1F	*E. coli*	0 (R)	14 (R)	20 (R)	17 (R)	13 (R)	0 (R)	0 (R)	15 (S)	0 (R)	32 (S)	22 (S)
M2F	*S. aureus*	0 (R)	20 (S)	19 (I)	0 (R)	18 (I)	0 (R)	0 (R)	14 (R)	0 (R)	0 (R)	0 (R)
M3F	*Klebsiella pneumoniae*	14 (I)	16 (R)	18 (R)	17 (R)	12 (R)	0 (R)	0 (R)	0 (R)	0 (R)	33 (S)	0 (R)
M3B	*Micrococcus luteus*	0 (R)	16 (I)	18 (I)	17 (S)	17 (I)	0 (R)	0 (R)	8 (R)	0 (R)	29 (S)	22 (R)
M4F	Klebsiella *pneumoniae*	0 (R)	18 (I)	22 (R)	19 (R)	20 (I)	0 (R)	10(R)	0 (R)	0 (R)	0 (R)	0 (R)
M4B	*Klebsiella oxytoca*	0 (R)	13 (R)	16 (R)	18(R)	0 (R)	0 (R)	0 (R)	8 (R)	0 (R)	30 (S)	0 (R)
M5F	*Proteus mirabilis*	0 (R)	12 (R)	17 (R)	0 (R)	18 (R)	0 (R)	9 (R)	9 (R)	0 (R)	0 (R)	0 (R)
M6F	*Proteus sp.*	0 (R)	0 (R)	14 (R)	0 (R)	15 (R)	0 (R)	0 (R)	8 (R)	0 (R)	0 (R)	0 (R)
M7F	*Micrococcus sp.*	0 (R)	0 (R)	11 (R)	14(I)	14 (R)	0 (R)	9 (R)	9 (R)	0 (R)	0 (R)	0 (R)
M8F	*Klebsiella oxytoca*	0 (R)	17 (R)	17 (R)	16 (R)	0 (R)	0 (R)	19 (S)	0 (R)	0 (R)	0 (R)	0 (R)
M9F	*Klebsiella oxytoca*	0 (R)	0 (R)	0 (R)	11 (R)	0 (R)	0 (R)	0 (R)	10 (R)	0 (R)	0 (R)	0 (R)
M10F	*Micrococcus sp.*	0 (R)	0 (R)	13 (R)	14 (I)	16 (R)	0 (R)	8 (R)	10 (R)	0 (R)	0 (R)	0 (R)
M11F	*S. aureus*	0 (R)	20 (S)	20 (I)	14 (I)	20 (I)	0 (R)	0 (R)	0 (R)	0 (R)	0 (R)	0 (R)
M12F	*Klebsiella sp.*	0 (R)	0 (R)	14 (R)	13 (R)	14 (R)	0 (R)	8 (R)	8 (R)	0 (R)	0 (R)	0 (R)
M13F	*Enterobacter aerogenes*	0 (R)	0 (R)	0 (R)	14 (R)	15 (R)	0 (R)	9 (R)	7 (R)	0 (R)	0 (R)	0 (R)
M14F	*Klebsiella pneumoniae*	34 (S)	17 (R)	23 (I)	0 (R)	0 (R)	0 (R)	0 (R)	13 (I)	0 (R)	0 (R)	24 (S)
M15F	*Klebsiella pneumoniae*	15 (R)	15 (R)	20 (R)	22 (R)	18 (R)	0 (R)	10 (R)	0 (R)	0 (R)	0 (R)	0 (R)

SAMPLES	ORGANISM ISOLATED	ANTIBIOTICS ZONE OF INHIBITION (MM) AUG (30µg)	CAZ (30µg)	CTX (30µg)	IMI (10µg)	CRO (30µg)	CLO (50µg)	FOX (30µg)	TE (30µg)	E (15µg)	CIP (5µg)	CN (10µg)
M16F	*S. epidermidis*	18 (R)	17 (I)	21 (I)	16 (S)	17 (I)	0 (R)	18 (S)	18 (I)	0 (R)	33 (S)	26 (S)
M17F	*Klebsiella oxytoca*	0 (R)	0 (R)	0 (R)	19 (R)	8 (R)	0 (R)	15 (I)	7 (R)	0 (R)	33 (S)	0 (R)
M18F	*S. aureus*	0 (R)	23 (S)	20 (I)	22 (S)	8 (R)	0 (R)	10 (R)	14 (R)	0 (R)	0 (R)	0 (R)
M19F	*Klebsiella pneumoniae*	24 (S)	19 (I)	26 (S)	17 (R)	21 (I)	0 (R)	23 (S)	0 (R)	0 (R)	26 (S)	19 (S)
M23F	*Klebsiella pneumoniae*	0 (R)	0 (R)	17 (R)	12 (R)	0 (R)	15 (R)	0 (R)	0 (R)	0 (R)	0 (R)	0 (R)
M24F	*Micrococcus sp.*	0 (R)	14 (R)	17 (I)	0 (R)	21 (S)	0 (R)	28 (S)	16 (I)	0 (R)	30 (S)	23 (S)
M25F	*Klebsiella pneumoniae*	7 (R)	18 (I)	23 (I)	0 (R)	24 (S)	0 (R)	30 (S)	12 (I)	0 (R)	32 (S)	18 (S)
F1F	*Klebsiella pneumoniae*	0 (R)	14 (R)	18 (R)	0 (R)	0 (R)	0 (R)	12 (R)	10 (R)	0 (R)	0 (R)	0 (R)
F2F	*Klebsiella oxytoca*	0 (R)	14 (R)	18 (R)	25 (S)	11 (R)	0 (R)	22 (S)	9 (R)	0 (R)	0 (R)	0 (R)
F2B	*Klebsiella pneumoniae*	0 (R)	12 (R)	22 (R)	0 (R)	21 (I)	0 (R)	10 (R)	16 (I)	0 (R)	30 (S)	20 (S)
F3B	*Micrococcus sp.*	0 (R)	10 (R)	15 (R)	20 (S)	14 (R)	0 (R)	13 (R)	13 (R)	0 (R)	0 (R)	0 (R)
F4F	*Staphylococcus sp.*	0 (R)	15 (I)	22 (I)	24 (S)	19 (I)	0 (R)	22 (S)	0 (R)	0 (R)	0 (R)	0 (R)
F5F	*S. aureus*	23 (S)	25 (S)	21 (I)	0 (R)	25 (S)	0 (R)	21 (S)	0 (R)	0 (R)	35 (S)	22 (S)
F6F	*Klebsiella pneumoniae*	22 (S)	23 (S)	27 (S)	21 (I)	22 (I)	0 (R)	17 (I)	0 (R)	0 (R)	29 (S)	19 (S)
F7B	*S. aureus*	0 (R)	8 (R)	15 (R)	0 (R)	15 (R)	0 (R)	13 (R)	12 (I)	0 (R)	22 (S)	0 (R)
F8F	*Proteus sp.*	12 (R)	13 (R)	16 (R)	20 (I)	22 (I)	0 (R)	20 (S)	22 (S)	0 (R)	18 (R)	23 (S)
F9F	*S. aureus*	31 (S)	27 (S)	28 (S)	0 (R)	29 (S)	0 (R)	26 (S)	16 (I)	0 (R)	32 (S)	22 (S)
F10F	*Proteus sp.*	0 (R)	12 (R)	18 (R)	15 (R)	20 (I)	0 (R)	11 (R)	13 (I)	0 (R)	0 (R)	0 (R)
F13F	*Proteus sp.*	0 (R)	0 (R)	0 (R)	0 (R)	16 (R)	0 (R)	12 (R)	16 (S)	0 (R)	17 (R)	26 (S)
F14F	*Proteus sp.*	12 (R)	14 (R)	18 (R)	14 (R)	21 (I)	0 (R)	9 (R)	18 (R)	0 (R)	8 (R)	22 (S)

SAMPLES	ORGANISM ISOLATED	ANTIBIOTICS ZONE OF INHIBITION (MM)										
		AUG (30µg)	CAZ (30µg)	CTX (30µg)	IMI (10µg)	CRO (30µg)	CLO (50µg)	FOX (30µg)	TE (30µg)	E (15µg)	CIP (5µg)	CN (10µg)
F15F	*S. aureus*	0 (R)	17 (I)	21 (I)	0 (R)	20 (I)	0 (R)	0 (R)	14 (R)	0 (R)	0 (R)	0 (R)
F16F	*Klebsiella pneumoniae*	0 (R)	0 (R)	17 (R)	9 (R)	19 (R)	0 (R)	11 (R)	0 (R)	0 (R)	33 (S)	0 (R)
F17B	*Proteus sp.*	0 (R)	19 (R)	30 (S)	0 (R)	32 (S)	0 (R)	31 (S)	22 (S)	0 (R)	27 (S)	23 (S)
F18F	*Klebsiella pneumoniae*	21 (S)	16 (R)	26 (S)	17 (R)	21 (I)	0 (R)	24 (S)	11(R)	0 (R)	20 (S)	17 (S)
F19F	*Klebsiella pneumoniae*	0 (R)	0 (R)	15 (R)	8 (R)	10 (R)	0 (R)	13 (R)	15 (S)	0 (R)	0 (R)	0 (R)
F20F	*S. aureus*	0 (R)	10 (R)	28 (S)	13 (R)	24 (S)	0 (R)	17 (R)	10 (R)	0 (R)	22(S)	18 (S)
F22F	*Enterobacter cloacae*	0 (R)	16 (R)	14 (R)	13 (R)	27 (R)	0 (R)	10 (R)	11 (R)	0 (R)	25 (S)	23 (S)
F23F	*Klebsiella oxytoca*	0 (R)	15 (R)	19 (R)	0 (R)	31 (S)	0 (R)	30 (S)	18 (S)	0 (R)	24 (S)	21 (S)
F23B	*Klebsiella sp.*	0 (R)	15 (R)	0 (R)	0 (R)	18 (R)	0 (R)	10 (R)	10 (R)	0 (R)	30 (S)	19 (S)
F24F	*Klebsiella oxytoca*	17 (I)	18 (I)	30 (S)	0 (R)	32 (S)	0 (R)	13 (R)	16 (S)	0 (R)	22 (S)	24 (S)
F25F	*Klebsiella pneumoniae*	0 (R)	0 (R)	23 (I)	0 (R)	20 (I)	0 (R)	8 (R)	14 (I)	0 (R)	20 (S)	22 (S)

KEY: AUG: Amoxicillin-Clavulanic acid, CAZ: Ceftazidime, CTX: Cefotaxime, IMI: Imipenem, CRO: Ceftriaxone, CLO: Clotrimazole, TE: Tetracycline, E: Erythromycin, CN: Gentamicin, CIP: Ciprofloxacin, FOX: Cefoxitin, R: Resistance, S: Sensitive, I: Intermediate

CHAPTER FIVE

DISCUSSION

5.1 DISCUSSION

During this research, the result showed 92% (46) of bacteria isolated from the mobile phones belonged to female students; whereas 84% (42) were obtained from mobile phones belonging to male students. This may be due to the fact that 34% (17) of the female students kept long nails which could harbor bacteria. This agrees with Ya'aba *et al.* (2020) who reported 69.4% of phone users of the University of Lafia, Nasarawa state had long nails with highly contaminated phones compared to 30.6% that did not.

The prevalence of bacteria isolated in this study, revealed *Staphylococcus aureus* (18%), *E. coli* (2%), *Staphylococcus epidermidis* (6%)*, Klebsiella pneumoniae* (26%) among mobile phones belonging to male students and *K. pneumoniae* (32%) for mobile phone belonging to female students. Interestingly, among the mobile phones belonging to the female students, the prevalence of *S. aureus* (18%) was the same for the male students, Similar findings have been recorded of 20.7% *S. aureus* isolates by Enass (2015) in University of Baghdad. 6% of *S. epidermidis* was isolated from male mobile phones in my study and this does not agree with study done by Micheal Olu-Taiwo (2021) done in University of Ghana, Accra and this can be due to difference in geographical area. *S. epidermidis* is a normal flora of the skin and high occurrence on mobile phones may be due to the presence of the bacteria on the hands and skin. 32% of the isolates were *K. pneumoniae* isolated from mobile phones belonging to female students and my result agrees with a study done by Ya'aba *et al.* (2020) in University of Lafia, Nasarawa state. 2% of *E. coli* was isolated from mobile phones belonging to male students in my

study and this does not agree with study done by Tagoe *et al.* (2011), this can be due to difference in methodology.

Some of the organisms isolated from the front of the phone are different from the ones isolated from the back of the phone. *E. coli* was isolated from the front of the phone sample but not isolated from the back. *S. epidermidis* was isolated more from the back of the phone sample than on the front of the phone and this may be due to the fact that the organism is found more on the hand and might have been transmitted to the phone through hand contact.

Antibiotic susceptibility test result showed that 16% of *S. aureus* was susceptible to Gentamicin, Ciprofloxacin, Ceftriaxone, Ceftazidime, 2% of *E. coli* was sensitive to Tetracycline, Gentamicin and Ciprofloxacin, 30% of *Klebsiella pneumoniae* was sensitive to Ciprofloxacin and Gentamicin, *S. epidermidis* was sensitive to Imipenem, Cefoxitin, Ciprofloxacin and Gentamicin and 8% *Proteus spp.* was sensitive to Gentamicin, Cefoxitin and Tetracycline.

The *S. aureus* isolated from the student's mobile phones showed a significant resistance to Cefoxitin. These isolates are described as Methicillin Resistant *Staphylococcus aureus* (CDC, 2021). Here 12% prevalence was found which agrees with a report by Aida *et al.* (2022) in a Mexican community.

Resistance to third generation cephalosporins (Ceftazidime and Cefotaxime) was 42% among the members of *Enterobacteriaceae; K. pneumoniae, E. coli, Proteus spp., K. oxytoca* isolated from mobile phones in this study. Multi-drug resistance was found among *Proteus, E. coli, K. pneumoniae, K. oxytoca, Enterobacter aerogenes* and *Citrobacter diversus* with prevalence of 10%, 2%, 20%, 12%, 2% and 2% respectively. The antibiotics with demonstrated multi-drug resistance were Erythromycin, Clotrimazole, Ceftazidime, Tetracycline, Cefoxitin, Ceftriaxone

and Imipenem. The high antibiotic resistance could be caused by the abuse of antibiotics by the students as observed in a study by Tagoe *et al.* (2010) in Cape Coast on antibiotic use.

5.2 CONCLUSION

In the current research, the surfaces of mobile phones of selected male and female students of Caleb University, Lagos were detected to be contaminated with different species of bacteria such as *Staphylococcus, Klebsiella, Enterobacter, Escherichia, Micrococcus* species etc. It was concluded that mobile phones can serve as a means of transmitting these bacteria because it serves as a depository for many microorganisms. All samples were contaminated with different types of bacteria with high resistance to commonly used antibiotics. I recommend periodic cleaning of the device with disinfectants to reduce or prevent bacterial contamination of mobile phone surfaces by drug-resistant bacteria.

Furthermore, it is advisable to observe good hygiene habits such as washing of hands and use of alcohol based hand sanitizer regularly to prevent germs.

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