MULTIPLE ANTIBIOTICS RESISTANT *STAPHYLOCOCCUS* SPP. FROM DOOR HANDLES OF OFFICES IN CALEB UNIVERSITY

BY

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DECLARATION

I, OLANIYAN ABOLADE MICHAEL, do hereby declare that this project is entirely my work and composition. The work embodied in this project has not been submitted in candidature for any degree and did not concurrently been submitted for any other degree. All references made to works of other persons have been duly acknowledged.

_____ _____

SIGNATURE DATE

CERTIFICATION

This is to certify that **OLANIYAN ABOLADE MICHAEL** with matriculation number 18/4575 carried out this research work titled **MULTIPLE ANTIBIOTICS RESISTANT STAPHYLOCOCCUS SPP. FROM DOOR HANDLES OF OFFICES IN CALEB UNIVERSITY** under my supervision in the Department of Biological Sciences and Industrial Biotechnology, College of Pure and Applied Sciences, Caleb University, Imota, Lagos State.

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DEDICATION

I dedicate this project to Almighty God, my creator, who kept me and gave me the heart to pursue my dreams and also to my wonderful parents.

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I value the people who were involved and helped me during this project work. I give thanks to Almighty God, for giving me strength and heart of patience all through my work.

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ABSTRACT

Studies have indicated *Staphylococcus* spp. as the leading cause of skin infections. The aim of this study was to determine the prevalence and antibiotic susceptibility of *Staphylococcus* spp. from Door Handles of Offices at Caleb University. Twenty samples were obtained from the admin building of the Caleb University, Lagos state. Sterile swab-sticks was used for the collection of specimens. The samples were directly plated on Mannitol Salt Agar and incubated at 37 °C for 18–24 h. An antibiotic susceptibility test was performed using the Clinical Laboratory Standard Institute's guidelines. Of all the isolation 60% were *S. aureus* and 40% *S. epidermidis* were isolated. *Staphylococcus* spp isolates were resistant to all the antibiotics used. The percentage of isolates resistant to Ofloxacin, Cefotaxime, Ceftriaxone, Cefixime, Levofloxacin, Ciprofloxacin, Azithromycin, Cefuroxime, Augmentin, Erythromycin, Imipenem, Gentamicin were 38.9%, 100.0%, 38.9%, 100%, 33.33%, 100%, 50%, 100.0%, 100.0%, 77.78%, 83.33%, 66.67% respectively. There were high levels of contamination of *Staphylococcus spp*. on door handles of the Offices at Caleb University. *Staphylococcus* species isolated had varied rates of resistance to the antibiotics used in this study. There is a need for periodic surveillance and monitoring of *Staphylococcus* spp. in the school environment as well as regular and effective cleaning of the offices door and contact surfaces in Caleb University. Further studies are needed, not only in Caleb University, but across Nigerian University offices, to understand the extent to which *Staphylococcus* spp. isolated from contact surfaces in the school environment.

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CHAPTER ONE

1.0 INTRODUCTION

1.1 Background to the Study

The human community is surrounded by the environment that harbours a wide range of both pathogenic and non-pathogenic microorganisms. There is no exception, if anything, it may harbour more pathogenic than non-pathogenic when infection control programs are not strengthened. Thanks to our bodies' immune system that controls and regulates the proliferation of these microbes. However, the school houses both immunocompetent and immunocompromised individuals: and the latter are prone to contracting nosocomial and opportunistic infections (Peton and Le Loir, 2014). In addition, microorganisms in school environment may be contracted from both wet and dry inanimate surfaces (Otto, 2014). For this reason, it is important to setup deliberate policies on microbial monitoring of the inanimate environment to detect the presence of specific nosocomial pathogens which can be used to evaluate the efficacy of routine cleaning and disinfection practices (Tong *et al*., 2015) Infectious diseases conveyed by hand contact have long been a major public health problem in many parts of the world. On touch surfaces such as doorknobs, tables, chairs, and windows, Gram positive and negative bacteria are regularly encountered (Saba *et al*., 2017). Infectious diseases are the biggest cause of death globally, and their contribution to morbidity and mortality is difficult to quantify due to a lack of statistics in most countries, therefore it continues to be a global concern (Grace *et al*., 2018). Contact with environmental surfaces can lead to common colds and sores, conjunctivitis, giardiasis, diarrhea, impetigo, meningitis, pneumonia, and other diseases and/or illnesses. Bacterial germs are responsible for a wide range of diseases (Kourtis *et al.*, 2019).

Human hands have been identified as a significant transmitter of microorganisms to environmental surfaces. According to Bouiller *et al*. (2020), hands regularly behave as vectors, transmitting disease-causing pathogens such as germs and viruses from one person to the next, either directly or indirectly via surfaces. Poor personal hygiene can transmit some of these harmful bacteria found in the environment to human hands (Abiose, 2019).

Environmental surfaces that are regularly touched with hands, such as toilet seats and restroom floors, have a greater bacterial burden, according to studies. This discovery could be linked to the contamination of door knobs caused by unhygienic conditions (Augustino *et al*., 2014). The first line of defense in avoiding illness spread was traditional hand washing; It has been overlooked, and families, schools, and healthcare providers must fully embrace it. Many people, on the other hand, appear to simply run water over their hands after using the restroom, and some do not wash their hands at all (Bouiller *et al*., 2020).

Staphylococcus spp. is a prevalent human and animal pathogens that causes both hospital- and community-acquired infections and diseases (Shen *et al*., 2013). Pimples, impetigo, boils, cellulitis, scalded skin syndrome (SSS), and abscesses; toxicosis, such as food poisoning and toxic shock syndrome (TSS); and systemic infections, such as endocarditis, brain abscesses, and meningitis, osteomyelitis, bacteremia, and sepsis are all infections caused by *Staphylococcus* spp. (Lee *et al*., 2018). *Staphylococcus spp.* has been revealed to carry antibiotic resistance genes, notably the mecA gene, which produces methicillin resistance (Flynt *et al.*, 2017). Resistance to penicillin was detected in *S. aureus* for the first time in 1947, four years after the antibiotic was put to the market. *Staphylococcus spp.* are also resistant to a number of medications, including beta lactam antibiotics like penicillin, amoxylin, and oxacillin. Treatment of *Staphylococcus spp.* infections has been

difficult due to the bacteria's ability to resist commonly used antibiotics in hospitals (Loubet *et al*., 2017).

Staphylococcus spp. are diseases of great concern because of their pathogenicity (Bouiller *et al*., 2016). Their ability to cause a wide range of life-threatening infections, as well as their ability to adapt to a variety of environmental situations (Davis *et al*., 2018). The most prevalent pathogen responsible for bloodstream infections, skin and soft tissue infections, bone and joint infections, urinary tract infections, and pneumonia has been identified as *Staphylococcus spp.* (Klein *et al*., 2013). Doorknobs are one of the most regularly identified potential sources of *Staphylococcus spp.* infections (Nworie *et al.*, 2012).

This study was planned to investigate the Multiple Antibiotics Resistant *Staphylococcus* Spp. on Door Handles of Offices at Caleb University. The site was targeted because these are frequently touched but most neglected from cleaning/disinfection procedures. Limited data is available regarding Multiple Antibiotics Resistant *Staphylococcus* Spp. on Door Handles of Offices at Caleb University. Identification of these sites and bacterial agents may help to reduce transmission of pathogens.

1.2 STATEMENT OF PROBLEM

Multiple Antibiotics Resistant *Staphylococcus* Spp. in schools poses a significant risk of infection to the general public, as well as the prospect of Multiple Antibiotics Resistant *Staphylococcus* Spp. infiltration into health-care facilities. Immunocompromised people are more likely to bring Multiple Antibiotics Resistant *Staphylococcus* Spp. home with them, as they are also at risk of infection if they come into contact with contagious pupils at school. All other family members who may spread the infection to other parts of the community congregate at home.

There have been few investigations into the occurrence of Multiple Antibiotics Resistant *Staphylococcus* Spp. in doorknobs. Multiple Antibiotics Resistant *Staphylococcus* Spp. spread poses a serious threat to humans and causes a significant financial strain on healthcare resources. Only a few studies have been done to see how often Multiple Antibiotics Resistant *Staphylococcus* Spp. isolates are in different door handles. Furthermore, the vast majority of studies focus primarily on specific surfaces and materials. As a result, this study was conducted at Caleb University to determine the level of Multiple Antibiotics Resistant *Staphylococcus* Spp. in practically all types of door handles.

1.3 AIM AND OBJECTIVES

This study is aimed at knowing the Multiple Antibiotics Resistant *Staphylococcus* Spp on Door Handles of Offices at Caleb University

The specific objectives are to:

i. isolate *Staphylococcus* spp. from Door Handles Of Offices In Caleb University
ii. determine the sensitivity pattern of isolated *Staphylococcus* spp. to multiple antibiotics

CHAPTER TWO

LITERATURE REVIEW

2.1 *Staphylococcus* spp.

Staphylococci are Gram-positive cocci with an average diameter of 0.9µm (Yusuf *et al*., 2020). This bacterium tends to be arranged most commonly in a group of irregular clusters or 'bunches of grapes' (Yokoe *et al*., 2014). Colonies of *staphylococci* are typically white with regular edges. *Staphylococci* are non-spore forming and non-motile, and most species are facultative anaerobes that display a fermentative metabolism (Yokoe *et al*., 2014). They are resistant to lysozyme, bacitracin, and to O/129, and are usually oxidase-negative and catalase positive (Yusuf *et al*., 2020).

Growth of *staphylococci* occurs on blood and nutrient agars, but not on MacConkey agar (Yokoe *et al*., 2014). Most strains of *staphylococci* do not have a capsule, but a limited number do. There are approximately 30 strands of *staphylococci* found among animals, but most are not pathogenic; *staphylococci* organisms are considered opportunistic pathogens (Yang *et al*., 2017).

Most infections that involve staphylococci are pyogenic and acute. The two major pathogenic species of *staphylococci* are *Staphylococcus intermedius* and *Staphylococcus aureus* (Yusuf *et al*., 2020). Recent studies have identified biofilms in wounds of infected persons and have begun to highlight how biofilms impede inflammatory responses and the efficacy of antimicrobial therapy (Yang *et al*., 2017).

2.2 *Staphylococcal* Classification

Both pathogenic strains of *staphylococcus*, *S. aureus*, and *S. intermedius*, are coagulase-positive, which typically correlates well with pathogenicity (Osiyemi *et al.*, 2018). Coagulase-negative forms of *staphylococci* mostly occur as commensal organisms within the environment (Omoshaba *et al*., 2020). *Staphylococci* are often hemolytic on blood agar and salt tolerant. Identification of *staphylococci* organisms requires biotype analysis for confirmation (Wisgrill *et al*., 2017).

2.3 *Staphylococci* Natural Habitat

Staphylococcal organisms are considered a major component of the normal microflora of humans and animals and only occasionally cause opportunistic infections (Usman *et al*., 2016). *Staphylococci* are widely common and present on animals; however, *staphylococci* can also be found and survive for long periods of time in the environment as well. *Staphylococci* are resistant to high salt concentrations and to dry conditions, making these organisms well-suited for the skin, which is considered their ecological niche (Sutter *et al*., 2016). This organism can be found as part of the normal flora of the upper respiratory tract (Stefani *et al*., 2012). *Staphylococci* are found commonly on the skin of healthy individuals. *Staphylococcus aureus* is even present in the nose of up to 30% of healthy people (Usman *et al*., 2016). However; *S. aureus* can cause infections where there is a lower host resistance, such as with damaged skin or an open wound

(Stefani *et al*., 2012).

2.4 Clinical Manifestations and Pathogenesis

Staphylococci are aerobic gram-positive bacteria that are either coagulase-negative or coagulasepositive. Gram staining is a staining technique used in microbiology to identify the cell wall structure of bacteria if a bacterium is gram-positive it has a cell wall higher in peptidoglycan and lower in lipid than gram-negative bacteria. The coagulase test is a method used for

differentiating between pathogenic and non-pathogenic strains of *Staphylococcus* (Okiki *et al*., 2020). Bacteria that produce coagulase use it as a defense mechanism by clotting the areas of plasma around them, thereby enabling themselves to resist phagocytosis by the host's immune system. Coagulase-negative *Staphylococci* are among the normal flora on the skin usually on the nasal mucosa, axilla, or groin and are generally nonpathogenic, but can also cause boils, abscesses, carbuncles, and serious upper respiratory infections. Coagulase positive staphylococci tend to be more virulent than coagulase-negative *Staphylococci* (Nworie *et al*., 2017). S. *aureus* is among the coagulase-positive form of *Staphylococci* and tends to form pus; coagulase-positive cause many types of infections. *Staphylococci* can cause a host of infections such as; *bacteremia*, *pneumonia*, *enterocolitis, osteomyelitis*, food poisoning, and skin infections. S. *aureus* expresses many potential virulence factors. One of the potential virulence factors are surface proteins that promote colonization of the host tissues. Other types of virulence factors inhibit phagocytosis such as the capsule and protein A. Lastly, some virulence factors are classified as toxins, which damage host tissues and cause disease symptoms (Mustapha *et al*., 2017).

S. *aureus* expresses many cell surface-associated and extracellular proteins that are potential virulence factors. For the majority of diseases caused by this organism, pathogenesis is multifactorial. Thus, it is difficult to determine precisely the role of any given factor. This also reflects the inadequacies of many animal models for *staphylococcal* diseases (Mkhize *et al*., 2021). However, there are correlations between strains isolated from particular diseases and expression of particular factors, which suggest their importance in pathogenesis. Before S. *aureus* can cause an infection, it must first enter the body. It can do this in several ways; through the nasal or mucosal opening, wounds, surgery, or IV drug use. Once in the body, S. *aureus* attaches to the host cells or tissues. Proteins are expressed on the surface of S. *aureus* cells that promote attachment to host proteins such as laminin and fibronectin that form part of the extracellular matrix. Fibronectin is

present on epithelial and endothelial surfaces, as well as being a component of blood clots. In addition, most strains express a fibrinogen/fibrin binding protein, which promotes attachment to blood clots and traumatized tissue (Foster, 2014).

S. *aureus* expresses a number of factors that have the potential to interfere with host defense mechanisms. However, strong evidence for a role in virulence of these factors is lacking. The majority of clinical isolates of S. *aureus* express a surface polysaccharide of either serotype 5 or 8 (Loubet *et al*., 2018). This has been called a microcapsule because it can be visualized only by electron microscopy after antibody labeling, unlike the copious capsules of other bacteria, which visualize by light microscopy. The function of the microcapsule is unclear but it may interfere with the process of phagocytosis, but in in-vitro studies, this was only demonstrated in the absence of complement. Protein A is a surface protein of S. aureus, which binds immunoglobulin G molecules by the Fc region. In serum, bacteria will bind IgG molecules the wrong way round by this mechanism and in principle will disrupt opsonization and phagocytosis (Foster, 2014).

S. *aureus* expresses several different toxins that are potentially responsible for symptoms during infections. Some damage the membranes of erythrocytes, causing hemolysis. One toxin, leukocidin, causes membrane damage to leukocytes; another toxin alpha-toxin (α-toxin) causes septic shock; enterotoxins and toxic shock syndrome toxin (TSST-1) cause toxic shock (Lee *et*

al., 2018).

The most potent membrane-damaging toxin of S. *aureus* is α-toxin. It is expressed as a monomer that binds to the membrane of susceptible cells. Subunits then oligomerize to form hexameric rings with a central pore through which cellular contents leak. Susceptible cells have a receptor for α-toxin, which allows low concentrations of toxin to bind, causing small pores through which

monovalent cations can pass (Kwoji *et al.*, 2018). At high concentrations, the toxin reacts nonspecifically with membrane lipids, causing larger pores through which divalent cations and small molecules can pass. Platelets and monocytes are particularly sensitive to αtoxin. They carry high affinity sites that allow toxin to bind. A complex series of secondary reactions ensue, causing release of eicosanoids and cytokines, which trigger production of inflammatory mediators. These events can cause the symptoms of septic shock that occur during severe infections caused by S. aureus. Gamma-toxin (γ-toxin) and leukocidin are two-component protein toxins that damage membranes of susceptible cells. The proteins are expressed separately but act together to damage membranes. The γ-toxin locus expresses three proteins (Kourtis *et al.*, 2019). The B and C components of γ-toxin form a leukotoxin which has poor hemolytic activity, whereas the A and

B components of γ-toxin are hemolytic and weakly leukotoxic. The classical Panton and Valentine (PV) leukocidin is distinct from leukotoxin expressed by the γ-toxin locus. It has potent leukotoxicity and, in contrast to γ-toxin, is non-hemolytic. PV leukocidin causes dermonecrosis when injected subcutaneously in rabbits. Furthermore, at a concentration below that causing membrane damage, the toxin releases inflammatory mediators from human neutrophils, leading to degranulation. This could account for the histology of dermonecrotic infections (vasodilation, infiltration and central necrosis) (Foster, 2014).

S. *aureus* can express two different types of toxin with super-antigen activity, enterotoxins, of which there are six serotypes (A, B, C, D, E, and G) and toxic shock syndrome toxin (TSST-1). Enterotoxins cause diarrhea and vomiting when ingested and are responsible for staphylococcal food poisoning (Klibi *et al.*, 2018). When expressed systemically, enterotoxins can cause toxic shock syndrome (TSS) – indeed enterotoxin B and C cause 50% of non-menstrual TSS. TSST-1 is very weakly related to enterotoxins and does not have emetic activity. TSST-1 is also responsible

for 75% of TSS, including all menstrual cases. TSS can occur as a sequel to any staphylococcal infection if an enterotoxin or TSST-1 is released systemically and the host lacks appropriate neutralizing antibodies. Super-antigens stimulate T cells non-specifically without normal antigenic recognition. Up to one in five T cells may be activated, whereas only 1 in 10,000 are stimulated during antigen presentation. Cytokines are released in large amounts, causing the symptoms of TSS (Ike *et al*., 2016). Super-antigens bind directly to class II major histocompatibility complexes of antigen-presenting cells outside the conventional antigenbinding groove. This complex recognizes only the Vb element of the T cell receptor. Thus, any T cell with the appropriate Vb element can be stimulated, whereas normally, antigen specificity is also required in binding (Foster, 2014).

2.5 Emergence of Antimicrobial Resistant Bacteria

Since their discovery, antimicrobial drugs have proven remarkably effective for the control of bacterial infections (Gold, 2016). However, soon after the discovery of antimicrobial drugs, bacteria began to emerge that were resistant to antimicrobial agents and as time passed new "super" bacteria emerged that were resistant to all known antimicrobial agents, such as methicillin-resistant *Staphylococcus aureus* (MRSA). With the emergence of Vancomycin resistant S. *aureus* in patients with MRSA, the only remaining uniformly effective treatment of staphylococcal infection has been rendered ineffective (Smith, 2019). In light of the antibiotic resistance developed by S. *aureus*, it is self-evident that new technologies are needed to treat these common bacterial infections (Davis *et al*., 2017).

It's vital to understand how antimicrobials operate before delving into the processes of antimicrobial resistance. Antimicrobials work by interfering with critical bacterial activities as cell wall building, protein synthesis, nucleic acid synthesis, and metabolic pathways to prevent bacterial infection

(Mkhize *et al*., 2021). If an antibiotic can block, stop, or modify any of these crucial activities, the bacterium will be crippled or die.

The first antibiotic resistant microbes were found shortly after the therapeutic introduction of penicillin (Yusuf *et al*., 2020). Researchers didn't know much about resistance mechanisms at the time, but scientific knowledge has dramatically advanced in the subsequent 50 years. Bacteria can develop antibiotic resistance through two mechanisms: (1) selection of spontaneous mutations in their DNA through natural selection and (2) the incorporation of genetic material from other bacteria into their DNA (Abubakar and Sulaiman, 2018).

Bacteria regularly undergo spontaneous mutations, and the new mutation may lead to antibiotic resistance development. Penicillin-resistant pneumococci are a good example of this strategy, as they developed resistance due to a mutation in the penicillin binding site, rendering it ineffective (Abdullahi and Iregbu, 2018).

Bacteria can develop resistance by receiving genetic material from bacteria of the same or different species that encodes the resistance trait. This genetic material can be obtained through transformation, conjugation, or transduction (Osman, 2016; Yang *et al*., 2017). Transformation happens when bacteria absorb genetic material released from cells, usually by cell lysis, and it is present in the environment. The direct transfer of plasmids, or mobile genetic elements, between two species is known as conjugation. This can happen as a result of mechanisms such as a pilus, which can unite two cells, or when cells conjoin during sexual reproduction. Transduction occurs when a bacteriophage, a virus that infects bacteria and can occasionally transmit genetic material, transfers genes between bacteria. Antibiotic abuse and overuse have encouraged both sources of resistance (Macori *et al*., 2017). Antimicrobials stimulate the development of resistance strains of

bacteria since they destroy all typically vulnerable microorganisms. As a result, only bacteria that have developed resistance can survive and proliferate.

In healthcare facilities, antimicrobial-resistant bacteria such as *Staphylococci, Enterococci, Klebsiella pneumoniae,* and *Pseudomonas aeruginosa* have become common. In addition, some microorganisms have developed resistance to a range of treatments, including MRSA, vancomycin-resistant enterococci, and gram-negative bacilli. Because treatment options for multidrug-resistant diseases are extremely restricted, they are particularly worrying. It's especially troubling since some extended-range -lactamase-producing bacteria have been shown to be resistant to all currently available antibiotic therapies (Siegel *et al*., 2007).

2.6 Mechanism of Antibiotic Resistance

Development of antimicrobial resistance is of great concern to the medical community, since the rate of resistance is faster than that of new drug development. The mission to treat these infections becomes progressively more difficult. As we have learned more about the mechanisms and epidemiology of resistance to antimicrobial drugs, it has become clear that bacteria have a remarkable array of tools at their disposal to overcome antibiotics (Gold, 2005). A single genetic mutation may lead to resistance without altering the pathogenicity or viability of a bacterial strain (Gold, 2005). Theoretically, it should be possible to overcome mutational resistance by administering a combination of drugs in sufficient dosage for an appropriate amount of time to eradicate the infection, thus preventing person-to person dissemination of resistant bacteria (Gold, 2005). The worldwide emergence of multidrug-resistant Mycobacterium tuberculosis demonstrates that this goal may not be easy to achieve (Gold, 2005). Of potentially greater concern is the fact that bacteria may acquire exogenous genetic material that leads to antimicrobial resistance (Gold, 2005). Many of the genes that mediate resistance are found on transferable plasmids or on

transposons that can be disseminated among various bacteria by conjugation (Gold, 2005). Some transposons or plasmids have genetic elements termed integrons that enable them to capture exogenous genes (Gold, 2005). A number of genes may therefore be inserted into a given integron, resulting in resistance to multiple antimicrobial drugs or possibly allowing the accumulation of both regulatory and structural genes in the same transposon (Gold, 2005).

Since the introduction of the first antimicrobial drug, Penicillin in the 1940s S. *aureus* has evolved resistance to various drugs. Community and hospital-acquired MRSA evolve from prevalent methicillin-susceptible S. *aureus* (MSSA) upon acquisition of staphylococcal chromosomal cassette mec-like element (SCCmec) (Holden, 2014), which carries one or more genes for antibiotic resistance. Community-acquired MRSA has become increasingly endemic in many parts of the world. (Miller, 2015). The most common clinical syndrome has been skin and soft-tissue infections (Miller, 2015).

The evolutionary origin of methicillin-resistance strains of S. *aureus* is of immense importance to the medical community. Researchers have used a DNA microarray representing >90% of the S. *aureus* genome to characterize genomic diversity, evolutionary relationships, and virulence gene distribution among 36 strains of organisms causing toxic shock syndrome (Fitzgerald, 2011). It has been found that genetic variation in S. *aureus* is very extensive, with 22% of the genome comprised of dispensable genetic material (Fitzgerald, 2001). Some of these genes represent nonessential genetic information that is strain-specific. Some of these genes encode factors that facilitate colonization of specialized host or environmental niches (Fitzgerald, 2011). Many of the strain-variable open reading frames identified in the study done by Fitzgerald in 2011 form 18 discrete regions of difference, including pathogenicity island-, phage-, plasmid-, transposon-, and insertion sequence-related elements.

Two very different hypotheses have been put forth to explain the origin of MRSA strains (Fitzgerald, 2011). The association of the mec gene with genetically diverse lineages of S. *aureus*, together with data indicating that the mec gene was horizontally transferable in the laboratory, led to the hypothesis that MRSA strains had evolved many independent times by lateral gene transfer of the mec element into phylogenetically distinct methicillin susceptible precursor cell lines (Fitzgerald, 2011). In contrast, data obtained from study of MRSA by restriction fragment length polymorphism analysis with probes for mecA and Tn554 were interpreted to mean that MRSA organisms evolved from a susceptible clone that acquired the mec element and subsequently generated substantial chromosomal diversity (Fitzgerald, 2011). Fitzgerald (2011) discovered that MRSA strains were assigned to at least five distinct chromosomal genotypic groups that relative to one another, are highly divergent, in some cases differing by greater than several hundred genes. Hence, the only reasonable interpretation of the data is that the MRSA strains have arisen multiple independent times by lateral transfer of the mec element into methicillin-susceptible precursors (Fitzgerald, 2011).

2.7 Environmental Decontamination

The essential concept and practice of sanitizing patient-care materials and equipment were created over 40 years ago (Rutala and Weber, 2011). This technique employs three classes for patient-care items and equipment. The first are critical objects that represent a significant risk of illness if contaminated. Because they are used in sterile bodily regions, these things must be sterilized before use. When semi-critical items come into contact with mucous membranes or non-intact skin, they must be thoroughly cleaned. Non-critical goods, on the other hand, are items that may come into contact with healthy skin and only need to be disinfected at a low level.

Non-critical items, such as environmental surfaces, must be disinfected on a daily basis at a low level. The Centers for Disease Control and Prevention (CDC) has issued disinfection and sterilizing standards for medical facilities (Egege *et al*., 2020). Despite the fact that Multiple Antibiotics Resistant *Staphylococcus* Spp. has developed resistance to a variety of drugs, this paper emphasizes that it can be readily removed with the use of detergents and disinfectants. According to the authors, cleaning solutions must be used at the proper concentrations, applied to all surfaces, and left in contact with the surfaces for an acceptable amount of time in order to kill bacteria. A minimum of 10 minutes of contact time is required by most cleaners (Egege *et al.*,

2020). However, these procedures are not always followed, which could result in active Multiple Antibiotics Resistant *Staphylococcus* Spp. lingering on surfaces.

2.8 Multiple Antibiotics Resistant *Staphylococcus* Spp on Environmental Surfaces

Environmental surfaces can get contaminated with Multiple Antibiotics Resistant *Staphylococcus* Spp. when germs are discharged into the environment by sick or colonized people. Sneezing, coughing, talking, eating, or obtaining basic medical attention can all cause this. Infected or colonized patients have previously been demonstrated to shed their specific strain of Multiple Antibiotics Resistant *Staphylococcus* Spp. into the environment, resulting in high rates of contamination on surfaces and items near the patient (Igbinosa *et al.*, 2016; Aliyu *et al*., 2020; Ajoke *et al*., 2021). According to Igbinosa *et al*. (2016) Multiple Antibiotics Resistant *Staphylococcus* Spp. was found in more than half of the floor samples, bed linen samples, and patient gown samples. They also discovered that Multiple Antibiotics Resistant *Staphylococcus* Spp. contamination in the environment was found in 85 percent of patients with Multiple Antibiotics Resistant *Staphylococcus* Spp-infected wounds or urine, compared to only 36 percent of patients with Multiple Antibiotics Resistant *Staphylococcus* Spp. infection in the sputum, blood, or conjunctivae. Contamination rose

as the number of culture-positive body locations increased. Infected patient rooms also had 32 percent contaminated surfaces, compared to only 20% in colonized patient rooms.

These germs will remain on environmental surfaces until they die or are wiped away. Multiple Antibiotics Resistant *Staphylococcus* Spp. has been discovered to live for up to 318 days on environmental surfaces if they are not cleaned or removed (Igbinosa *et al*., 2016). A variety of factors influence the persistence of Multiple Antibiotics Resistant *Staphylococcus* Spp. on environmental surfaces. According to recent studies, lower temperatures, lower humidity, and the presence of organic material, such as bovine serum albumin (used to mimic organic material), all increased the length of survival (Coughenour *et al*., 2011). In addition, the type of material utilized to make surfaces has an effect on longevity, with plastic and vinyl outlasting wood

(Coughenour *et al*., 2011). Copper inclusion in surfaces has the potential to reduce life spans (Nworie *et al*., 2017). When comparing Multiple Antibiotics Resistant *Staphylococcus* Spp. strains to non-resistant strains, survival times are comparable (Macori *et al*., 2017), while Multiple Antibiotics Resistant *Staphylococcus* Spp. strains that are more prone to cause an epidemic have been shown to survive longer than ordinary Multiple Antibiotics Resistant *Staphylococcus* Spp. strains (Ike *et al*., 2016). The importance of dirty ambient surfaces in the transmission of infections is still debated. Multiple Antibiotics Resistant *Staphylococcus* Spp concentrations on surfaces in the environment have supposedly been found to be high enough to allow transmission (Otter *et al*., 2011).

According to one study, the average concentration of Multiple Antibiotics Resistant *Staphylococcus* Spp. on surfaces was between 1 and 100 Colony Forming Units (CFU)/cm^2, which is higher than the infectious dose of less than 15 *S. aureus* cells that was shown to cause infection in experimental

lesions (Ajoke *et al*., 2021). Surfaces contaminated with Multiple Antibiotics Resistant *Staphylococcus* Spp. were also involved in the propagation of new infections during an MRSA outbreak in a London surgical facility (Aliyu *et al*., 2020). Furthermore, a study was able to link environmental sample strain types to patients, implying that the environment infected three of the 26 patients (Mustapha *et al*., 2016). Many examinations into the presence of Multiple Antibiotics Resistant *Staphylococcus* Spp. on various surfaces have been conducted, however the results have been inconsistent. According to Mustapha et al. (2016) Multiple Antibiotics Resistant *Staphylococcus* Spp. was found in 21.8 percent of hospital samples, with the area beneath the bed having the highest level of infection. Nworie *et al.* (2017) discovered that 27 percent of ambient samples in a hospital tested positive for Multiple Antibiotics Resistant *Staphylococcus* Spp, with the floor being the most contaminated. Igbinosa *et al*. (2016) discovered much higher rates of contamination at a 720-bed hospital with two intensive care units, with 56.3 percent of surface samples contaminated with Multiple Antibiotics Resistant *Staphylococcus* Spp. Door knobs were found to be contaminated in 19 percent of Multiple Antibiotics Resistant *Staphylococcus* Spp. patient rooms and 8.7% of all patient rooms (Sutter *et al.*, 2016).

CHAPTER THREE

MATERIALS AND METHOD

3.1 Materials

3.1.1 Equipment and Apparatus Used

The apparatus used included Microscope, Incubator, water bath, autoclave, refrigerator, weighing balance, antibiotic disc, forceps, inoculating loop, swab stick, petri dishes, foil paper, universal bottle, needle and syringe, cotton wool, gloves, nose mask, glass slide, matches, spirit lamp, test tube, conical flask, beaker, tube, Durham tubes, spatula, measuring cylinder, test tube rack, marker, masking tape, sanitizer, and McCartney bottle.

3.1.2 Reagents and Media used

Nutrient agar (HiMedia Laboratories Pvt Ltd, February 2025), MacConkey agar (HiMedia Laboratories Pvt Ltd, February 2025), Mueller Hinton Agar (HiMedia Laboratories Pvt Ltd, February 2025), Triple Sugar Iron Test, sucrose, maltose, fructose, lactose, glucose, Simmons Citrate agar (HiMedia Laboratories Pvt Ltd, February 2025), blood agar (HiMedia Laboratories Pvt Ltd, February 2025), peptone water, urea, urea agar base, starch soluble, potassium hydroxide pellet, hydrogen peroxide, Crystal violet, Iodine, decolourizer, Safranin, ethanol, Kovac's reagent.

3.2 Methods

3.2.1 Sample Collection

Twenty samples were obtained from the door handles of admin building of the Caleb University, Lagos state. Sterile swab-sticks were used for the collection of specimens. The swab sticks were briefly immersed in normal saline solution prior to swabbing of the door handle, and the specimen

were taken to the Caleb University Microbiology Laboratory immediately for analysis. The samples were properly labeled using reference numbers.

3.3 Laboratory Procedures

3.3.1 Isolation and Enumeration of Bacteria

The specimen were inoculated onto Nutrient agar for enumeration and Mannitol Salt Agar was used to isolate *Staphylococcus* spp. and the plates were incubated for 18 hours at 37 ^{o}C. Reference numbers or coding was used to appropriately label the samples. Standard laboratory methods such as colony morphology, Gram staining, and catalase test, and coagulase test was used to validate the isolates' identities.

3.4 Identification of the Isolates

To identify the isolates, biochemical tests such as Gram staining reaction, Catalase Test, Sugar Fermentation Test, Citrate Utilization Test, Motility test were carried out**.**

3.4.1 Gram Staining Reaction

A thin smear film of a 24-hours old culture of the microbe was prepared on a sterile clean grease free slide, air dried and heat fixed. The dried smear is then stained with crystal violet for a minute and rinsed off with water, the iodine is added after for one minute and rinsed off. Decolourizer (70% ethanol) was then added for 5 seconds and rinsed off. Safranin is finally added for a minute and then rinsed off. The slide is the allowed to dry and immersion oil was added to the slide and viewed under X40 objective lens and X100 oil immersion objective lens. The organisms that retained the crystal violet (purple colour), indicated Gram-positive organisms, while the organisms that appeared pink indicated Gram-negative organisms.

3.4.2 Catalase Test

Colonies of the isolates were aseptically picked with an inoculating loop and placed on a clean grease free slide. A drop of 6% hydrogen peroxide is dropped on the colonies. Evolution of gas bubbles caused by free oxygen indicated presence of catalase enzymes which shows positive result, while absence of bubbles indicates a negative reaction.

3.4.3 Sugar Fermentation Test

Peptone water was prepared and divided into four beakers, each sugars: maltose, glucose, sucrose and galactose were added to each beaker and labeled accordingly. Phenol was added to each. They were put in test tubes, glucose which was put in test tubes with inverted Durham tubes placed in them. They were sterilized and inoculated with colonies of the isolates and were incubated at 37^0C for 48 hours. Colour change from red to yellow indicates positive result while negative result has no colour change. In glucose, colour change from red to yellow shows positive result for presence of acid

3.4.4 Citrate Utilization Test

The Citrate test was done in order to determine the ability of the isolates to utilize citrate as their sole source of carbon and ammonia as their sole source of nitrogen. Simmons citrate was prepared and homogenized in a water bath. It was then put in test tubes and autoclaved. After sterilization, it was slanted and allowed to solidify. It was then inoculated with the isolates and incubated at 37^0C for 48 hours. Colour change from green to deep blue indicated positive results while no colour change indicates negative result.

3.4.5 Motility test

Half strength of Nutrient agar was prepared and homogenized in a water bath. It was then put in test tubes and sterilized. The agar was allowed to partially solidify and the isolates were stabbed into the agar and incubated at 37^0C for 48 hours. Motility is indicated by the spreading of the organisms outside the line of stab.

3.4.6 Urease Test

Urea agar base and urea solution were prepared. They were then autoclaved in conical flasks together with empty test tubes. After sterilization, the urea agar base was mixed with the urea solution and dispensed into the test tubes and slanted. After it was solidified, the isolates were aseptically taken with inoculating loops and inoculated on the slants. The tubes where then incubated at 37^0C for 72hours. Colour change from yellow to pink indicated positive result while no colour change indicated negative result.

3.5 Antibiotic Susceptibility of the Isolates

The organisms were sub cultured from glycerol broth onto nutrient agar and incubated at a temperature of 37^0C for 24hours. After incubation, the organisms where inoculated on Mueller Hinton agar and the antibiotic disc was placed on each of them, depending on their gram. They were then incubated at 37^0C for 24hours. A ruler was used to measure the zone of inhibition and result were taken**.**

CHAPTER FOUR

RESULTS

The cultural and morphological characteristics of the isolates. The colours were either white or yellow, some isolates are convex while some are raised in elevation, some isolates are moist, some are mucoid or some are dry in texture, all the isolates are round in form. All the isolates are opaque. Some isolates are entire while some are curled in margin (Table4.1)

The results for biochemical tests. All the isolates are catalase positive i.e. produce the enzyme, catalase. The isolates are positive to urease test. The motility and citrate test are negative for all isolates. All the isolate are gram negative organisms. And all the isolates are positive to glucose, sucrose, maltose and galactose i.e. all the isolates can ferment sugar glucose (Table4.2) The sensitivity of the Isolate and their percentage. Some of the isolates are resistance to OFX, ZEM, LBC, CIP, AZN, AUG, and GN. While all the isolates are resistance to CXM, AUG, CTX and IMP.(Table4.3) The Percentage of the Isolates. *Staphylococcus epidermidis* (60%) and *Staphylococcus aureus* (40%) (Figure4.1) and the percentage of the sensitivity of the Isolate was well presented (Figure4.2)

TABLE 4.1: CULTURAL AND MORPHOLOGICAL CHARACTERISTICS OF THE ISOLATES

Sample	SIZE	COLOR	ELEVAT ION	TEXTURE	FORM	MARGIN	OPACIT Y
OFF 1 (A)	medium	yellow	convex	mucoid	round	entire	opaque
OFF1 (B)	medium	white	raised	smooth	round	curled	opaque
OFF 2 (A)	medium	yellow	convex	mucoid	round	entire	opaque
OFF2 (B)	medium	white	convex	moist	round	entire	opaque
OFF3 (A)	medium	yellow	convex	moist	round	entire	opaque
OFF 3 (B)	medium	white	raised	smooth	round	entire	opaque
OFF 4 (A)	medium	yellow	convex	moist	round	entire	opaque
OFF 4 (B)	medium	white	raised	dry	round	entire	opaque
OFF 5 (A)	medium	yellow	convex	moist	round	entire	opaque
OFF 5 (B)	medium	white	raised	moist	round	entire	opaque
OFF 6 (A)	medium	yellow	convex	moist	round	entire	opaque
OFF 6 (B)	medium	white	raised	moist	round	entire	opaque
OFF 7 (A)	medium	yellow	convex	moist	round	entire	opaque
OFF 7 (B)	small	white	raised	dry	round	entire	opaque
OFF 8 (A)	Medium	yellow	convex	moist	round	entire	opaque
OFF 8 (B)	medium	white	raised	moist	round	entire	opaque
0FF 9 (A)	medium	yellow	convex	moist	round	entire	opaque
OFF 9 (B)	medium	white	raised	dry	round	entire	opaque

TABLE 4.2 RESULTS FOR BIOCHEMICAL TESTS

ISOLATE CODE	CATALASE	UREASE TEST	MOTILITY	CITRATE TEST	GRAM STAINING STAINING	GLUCOSE	SUCROSE	MALTOSE	GALACTOSE	PROBABLE ORGANISMS
OFF 1 (A)	+	+	-	+	+	+	+	+	+	*Staphylococcus aureus*
OFF1 (B)	+	+	-	+	+	+	+	+	+	*Staphylococcus epidermidis*
OFF2 (A)	+	+	-	+	+	+	+	+	+	*Staphylococcus aureus*
OFF 2 (B)	+	+	-	+	+	+	+	+	+	*Staphylococcus epidermidis*
OFF3 (A)	+	+	-	+	+	+	+	+	+	*Staphylococcus aureus*
OFF3 (B)	+	+	-	+	+	+	+	+	+	*Staphylococcus epidermidis*
OFF 4 (A)	+	+	-	+	+	+	+	+	+	*Staphylococcus aureus*
OFF4 (B)	+	+	-	+	+	+	+	+	+	*Staphylococcus epidermidis*
OFF 5 (A)	+	+	-	+	+	+	+	+	+	*Staphylococcus aureus*
OFF 5 (B)	+	+	-	+	+	+	+	+	+	*Staphylococcus epidermidis*
OFF 6 (A)	+	+	-	+	+	+	+	+	+	*Staphylococcus aureus*
OFF 6 (B)	+	+	-	+	+	+	+	+	+	*Staphylococcus epidermidis*
OFF 7 (A)	+	+	-	+	+	+	+	+	+	*Staphylococcus aureus*
OFF 7 (B)	+	+	-	+	+	+	+	+	+	*Staphylococcus epidermidis*
OFF 8 (A)	+	+	-	+	+	+	+	+	+	*Staphylococcus aureus*

OFF 8 (B)	+	+	-	+	+	+	+	+	+	*Staphylococcus epidermidis*
OFF 9 (A)	+	+	-	+	+	+	+	+	+	*Staphylococcus aureus*
OFF 9 (B)	+	+	-	+	+	+	+	+	+	*Staphylococcus epidermidis*

TABLE 4.3 ANTIBIOTIC SUSCEPTIBILITY OF THE ISOLATE

ISOLATE	OFX	CTX	CRO	ZEM	LBC	CIP	AZN	CXM	AUG	ERY	IMP	GN
OFF 1 (A)	12 (R)	6 (R)	6 (R)	6(R)	23 (S)	6 (R)	6 (R)	6 (R)	6 (R)	12 (R)	6 (R)	12 (R)
OFF1 (B)	25 (S)	6(R)	28 (S)	6(R)	6 (R)	6 (R)	29 (S)	6 (R)	6 (R)	6 (R)	26 (S)	10 (R)
OFF2 (A)	23(S)	6 (R)	16 (R)	6(R)	25 (S)	6 (R)	22 (S)	6 (R)	6 (R)	10 (R)	6 (R)	26 (S)
OFF 2 (B)	20(S)	6 (R)	29 (S)	6(R)	28 (S)	6 (R)	14 (R)	6 (R)	6 (R)	10 (R)	22 (S)	22 (S)
OFF3 (A)	18(I)	6(R)	10 (R)	6(R)	23 (S)	6 (R)	20 (S)	6 (R)	6 (R)	6 (R)	6 (R)	14 (R)
OFF3 (B)	22(S)	6(R)	25 (S)	6(R)	27 (S)	6 (R)	14 (R)	6 (R)	6 (R)	16 (I)	6 (R)	6 (R)
OFF 4 (A)	13(R)	6(R)	30 (S)	6 (R)	26 (S)	6 (R)	20 (S)	6 (R)	6 (R)	6 (R)	6 (R)	16 (I)
OFF4 (B)	50(S)	6 (R)	22 (S)	6(R)	10 (R)	6 (R)	27 (S)	6 (R)	6 (R)	18 (I)	6 (R)	30 (S)
OFF 5 (A)	8(R)	6(R)	6 (R)	7(R)	23 (S)	6 (R)	6 (R)	6 (R)	6 (R)	6 (R)	6 (R)	6 (R)
OFF 5 (B)	10(R)	6(R)	28 (S)	9(R)	10 (R)	6 (R)	6 (R)	6 (R)	6 (R)	6 (R)	6(R)	6 (R)
OFF 6 (A)	20 (S)	6(R)	22(S)	6 (R)	6 (R)	6 (R)	26 (S)	6 (R)	6 (R)	6 (R)	6 (R)	6 (R)
OFF 6 (B)	24 (S)	6(R)	6 (R)	6(R)	30(S)	6 (R)	6 (R)	6 (R)	6 (R)	6 (R)	6(R)	6 (R)
OFF 7 (A)	6 (R)	6(R)	25 (S)	6(R)	27(S)	10 (R)	28 (S)	6 (R)	6 (R)	12 (R)	6 (R)	12 (R)
OFF 7 (B)	20 (S)	6(R)	6 (R)	6(R)	20 (S)	6 (R)	14 (R)	6 (R)	6 (R)	16 (I)	22 (S)	20 (S)
OFF 8 (A)	6 (R)	6 (R)	6 (R)	6(R)	6 (R)	6(R)	20 (S)	6 (R)	6 (R)	10 (R)	6 (R)	18 (I)
OFF 8 (B)	22 (S)	6(R)	29(S)	6 (R)	20 (S)	14 (R)	6 (R)	6 (R)	6 (R)	16 (I)	6(R)	6 (R)
OFF 9 (A)	11 (R)	6(R)	25(S)	13 (S)	28 (S)	6 (R)	22 (S)	6 (R)	6 (R)	6 (R)	6 (R)	6 (R)

OFF 9 (B)	26 (S)	6(R)	29 (S)	6 (R)	6 (R)	6 (R)	6 (R)	6 (R)	6 (R)	6 (R)	6(R)	6 (R)

KEY: R-resistance, S- susceptible, I- intermediate, OFX- Ofloxacin, CTX- Cefotaxime, CRO-Ceftriaxone, ZEM- cefixime, LBClevofloxacin, CIP- Ciprofloxacin, AZN- Azithromycin, CXM- cefuroxime, AUG- Augmentin, ERY- Erythromycin, IMPImipenem, GN- Gentamicin

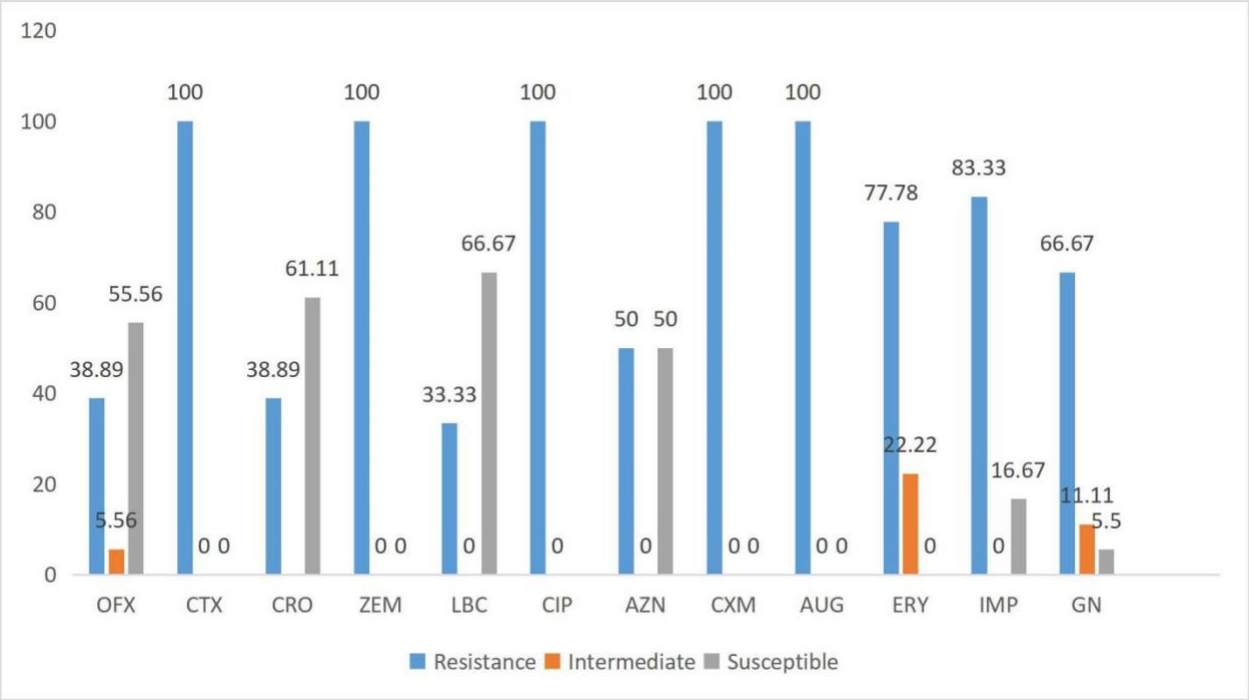

FIGURE 4.1: PERCENTAGE ANTIBIOTICS SUSCEPTIBILITY OF THE ISOLATE

KEY: R-resistance, S- susceptible, I- intermediate, OFX- Ofloxacin, CTX- Cefotaxime, CROCeftriaxone, ZEM- cefixime, LBC- levofloxacin, CIP- Ciprofloxacin, AZN- Azithromycin, CXM- cefuroxime, AUG- Augmentin, ERY- Erythromycin, IMP- Imipenem, GNGentamicin

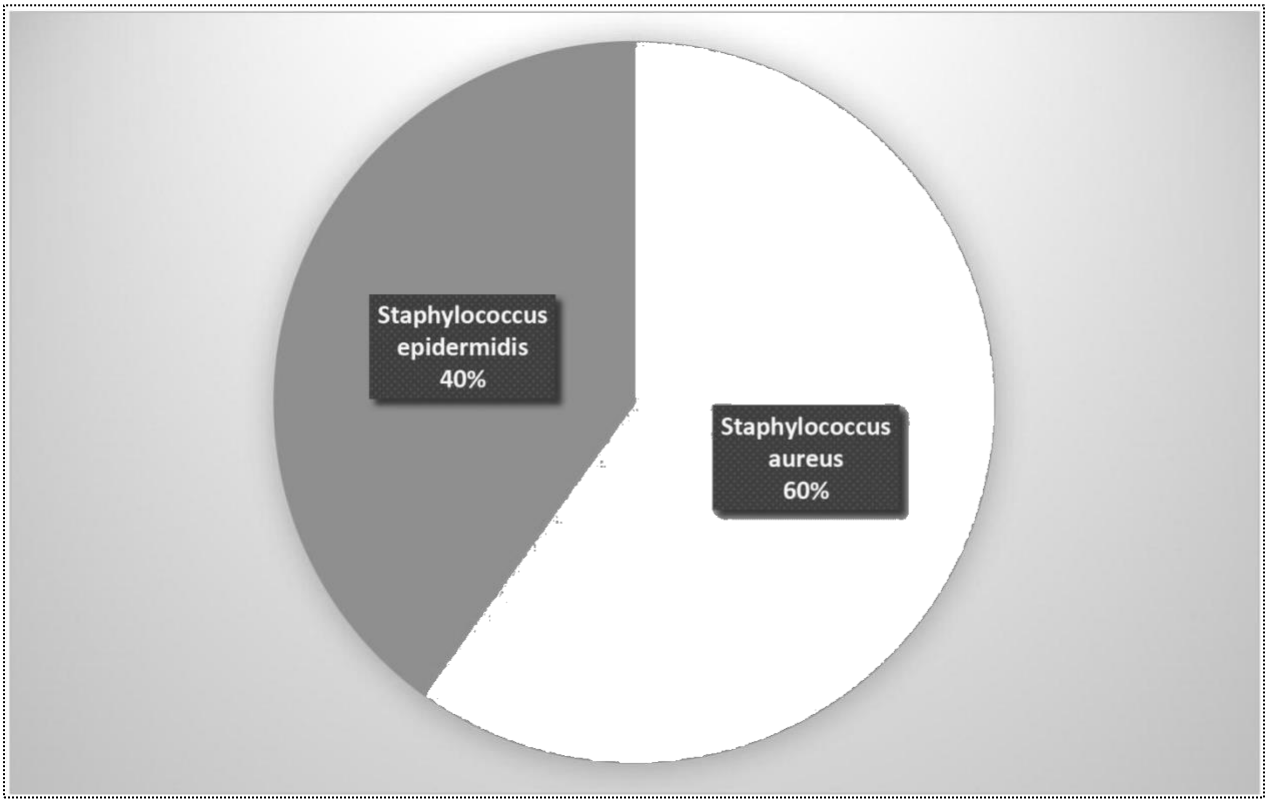

FIGURE 4.2: FREQUENCY OF ISOLATES

CHAPTER FIVE

DISCUSSION AND CONCLUSION

5.1 DISCUSSION

The dissemination of clinically significant microorganisms within our environment is fast growing through cross contamination of the surfaces due to poor hygiene. Reports have pinpointed doors handle, computer surfaces, phones etc. as sources of microbial transfer and could also be a route of infections in diseases outbreak (Boyce, 2016).

This study evaluates the prevalence of multiple antibiotics resistant *Staphylococcus* spp. on Door Handles of Offices at Caleb University. Of all the isolation 60% were *S. aureus* and 40% *S. epidermidis* were isolated. These findings concurred with the report of Ajayi and Ekozien (2014) in Ekpoma, Kawo *et al.,* (2012) in Kano and Itah and Ben (2004) in Akwa Ibom, Nigeria, who reported that *Staphylococcus aureus* is the most predominate surface microbe from door handles, tables, hand of students and computers. These surfaces especially the door handle are indeed a breeding ground for microbes as reported by Barker and Jones (2005), who noted that contamination of the environment via the surface-to-hand-to-mouth could be an avenue of contacting diseases and encourages disease wide spread. The results of this study are similar to those of Newman (2011) for a hospital in Accra where the *S. aureus* prevalence was 44% on contact surfaces at neonatal unit, and that of Hammuel *et al*. (2014) for a hospital in Zaria.

The antibiotic susceptibility profile of the *Staphylococcus aureus* isolated from the sampled door handles showed that the isolates were highly resistant (100%) to Cefotaxime, cefixime, Ciprofloxacin, cefuroxime and Augmentin. 33.3%, 50%, 77.8% 83.3%and 66.7% resistance to levofloxacin, Azithromycin, Erythromycin, Imipenem and Gentamicin respectively (Figure 4.1).

This result concurs with other studies that *Staphylococcus aureus* from surfaces have high percentages of antibiotic resistance even to methicillin (60.4%) (Adriano *et al*., 2011); Gentamicin, Amoxicillin-clavulanic acid and Cotrimoxazole (Ajayi and Ekozien, 2014). This suggests that in situation of such infections from door handle, these antibiotics should not be hoped on as the last resort for treatment. However, promising for therapeutics are Ofloxacin and Ceftriaxone as the isolates from this study showed high susceptibility to these antibiotics. This result is supported by the study of Ajayi and Ekozien (2014) and Ogbonna and Azuonwu (2019).

5.2 CONCLUSION

There were high levels of contamination of *Staphylococcus* spp. on door handles of the Offices at Caleb University. Isolates of *Staphylococcus* spp. have high rates of resistance to the antibiotics used in this study. This study provides preliminary information for the control of infections in the offices studied.

5.3 RECOMMENDATION

There is a need for periodic surveillance and monitoring of the prevalence of *Staphylococcus* Spp. in the school environment as well as regular and effective cleaning of the office doors and contact surfaces in Caleb University. Consumption of over the counter antibiotics without professional prescription should be discouraged. Further studies are needed, not only in Caleb University, but across Nigerian University offices as a means of surveillance.

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