PREVALENCE AND MOLECULAR CHARACTERISATION OF MULTIDRUG RESISTANT BACTERIA ISOLATED FROM PATIENTS WITH PELVIC INFLAMMATORY DISEASES ATTENDING SELECTED HOSPITALS IN NIGER STATE, NIGERIA

BY

OYEDUM, Uche Mary
PhD/SLS/2017/1011

DEPARTMENT OF MICROBIOLOGY
FEDERAL UNIVERSITY OF TECHNOLOGY MINNA

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ABSTRACT

This study determined the prevalence of multidrug resistant bacteria (MDR) isolated from patients with pelvic inflammatory disease (PID) attending nine hospitals in Niger State. Endocervical swabs and urine samples were collected from 1170 patients using sterile swab sticks and universal sample containers. The samples were transported on ice pack to the Microbiology laboratory, Federal University of Technology, Minna for further investigation.Screening for the presence of bacteria was done using streak method of inoculation on Nutrient agar, MacConkey agar, *Salmonella-Shigella* agar and blood agar respectively. The antibiotic susceptibility profile was determined using Kirby-Bauer disc diffusion technic on Mueller-Hinton agar. Molecular investigations of various multidrug resistant coding genes were done using specific primers**.** Seven hundred and twenty (720) bacterial isolates, comprising of 320 and 400 bacterial isolates from both endocervical swabs and urine samples were isolated and identified through Gram staining and other biochemical tests. Bacterial isolates identified in endocervical swabs include; *Klebsiella pneumoniae* (21.9%), *Escherichia coli* (22.5%), *Salmonella typhi* (21.6%), *Proteus vulgaris* (12.2%), *Streptococcus pyogenes* (10%) and *Staphylococcus aureus* (11.8%) while bacterial isolates identified in urine samples include; *Klebsiella pneumoniae* (21.2%), *Escherichia coli* (24.5%), *Salmonella typhi* (20.8%), *Proteus vulgaris* (13.5%), *Streptococcus pyogenes* (9.5%) and *Staphylococcus aureus* (10.5%). The rate of bacterial infection was observed more among patients who are rural dwellers (32.5%). Patients within the age of 25-29 years had more rate of infection (24.4%). 228 (31.7%) bacterial isolates, comprising of 90 (28.1%) and 138 (34.5%) bacterial isolates from both endocervical swabs and urine samples were confirmed as multidrug resistant (MDR) bacteria. The identified MDR bacterial isolates from endocervical swabs were: *Klebsiella pneumoniae* (50%), *Escherichia coli* (43.1%) and *Salmonella typhi* (34.8%) while the identified MDR bacterial isolates from urine samples were: *Klebsiella pneumoniae* (61.2%), *Escherichia coli* (49.0%), *Salmonella typhi* (39.8%) and *Proteus vulgaris* (9.3%). The antibiogram showed that 29(100%) and 20(100%) multidrug resistant bacteria from General Hospitals Agaie and Wushishi were resistant to Sulfamethoxazole trimethoprim (SXT) and Augmentin (Au) respectively, while 19(95%) MDR bacteria from General Hospital Kuta were resistant to Ampicillin, 18(94.7%) MDR bacteria from General Hospital Suleja were resistant to Ofloxacin and Nalidixic acid, 34(94.4%) MDR bacteria from Lapai were resistant to Cephalexin and 27(93.1%) MDR bacteria from General Hospital Agaie were resistant to Augmentin. Multidrug resistant bacteria resistant to 5 or more antibiotics, from both endocervical swabs (81.1%) and urine samples (79.7%) were mostly isolated. There was a significant difference ($P<0.05$) in the extended spectrum betalactamase ($28.00 \pm 3.03_b^b$, 49.00 ± 1.80^b_d) and cabarpenemase ($24.00 \pm 2.00_b^{ab}$, $44.00 \pm 0.00_d^b$) produced in *K. pneumoniae* isolated from endocervical swab and urine compared to the extended spectrum betalactamase and cabarpenemase produced by *Escherichia coli*, *Salmonella typhi* and *Proteus vulgaris* isolated from endocervical swab and urine. The PCR results of 12 MDR isolates that were found to be completely resistant to 10 different antibiotics used in this study revealed that multidrug resistant genes such as: *TEM* and *CTX-M* were contained in 91.7% of the isolates while *parC* and *OXA-48* were found in 83.3% of the isolates, *CTX-M2* and *aacC1* were contained in 58.3% of the isolates, *gyrA SHV* and *aacC2* were found in 50% of the isolates and *CTX-M1* was contained in 33.3% of the isolates. The results of this study confirmed the presence of multidrug resistant genes in MDR isolates in Niger State, hence there is the need for the intervention of Government and public health providers, to prevent treatment failure due to antibiotic resistance.

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CHAPTER ONE

1.0 INTRODUCTION

1.1 Background to the Study

Drug resistance is the ability of an organism to withstand the effect of a drug (particularly an antibiotic or a group of antibiotics). This condition basically occurs in various environments such as healthcare facilities and daycare centers, where microorganisms withstand the effects of antimicrobials. Drug resistance by microorganisms, particularly bacteria is on the increase and has been considered as a major health challenge worldwide (Mcintosh, 2018).

Emergence of drug resistance among the most important bacterial pathogens is recognized as a major public health threat affecting humans globally (Munita and Arias, 2016). Multidrug resistant organisms have emerged not only in the hospital environment but are now often identified in the community settings, suggesting that reservoirs of antibiotic resistant bacteria are present outside the hospital (Munita and Arias, 2016). The abilities of most bacteria to survive, when expose to antibiotics is due to mutational adaptation, acquisition of genetic materials or alteration of the binding or target sites in the microorganisms (Figure 1.1) and this condition, therefore results to the resistance of all antibiotics currently available and used in the clinical practice (Munita and Arias, 2016). However, most of these multidrug resistant bacteria are basically the main causative agents associated with most prevailing bacterial infections, such as pelvic inflammatory diseases (PID) prevalent among women folk in many developed and developing countries of the world (Nikaido, 2009; Adekunle, 2012).

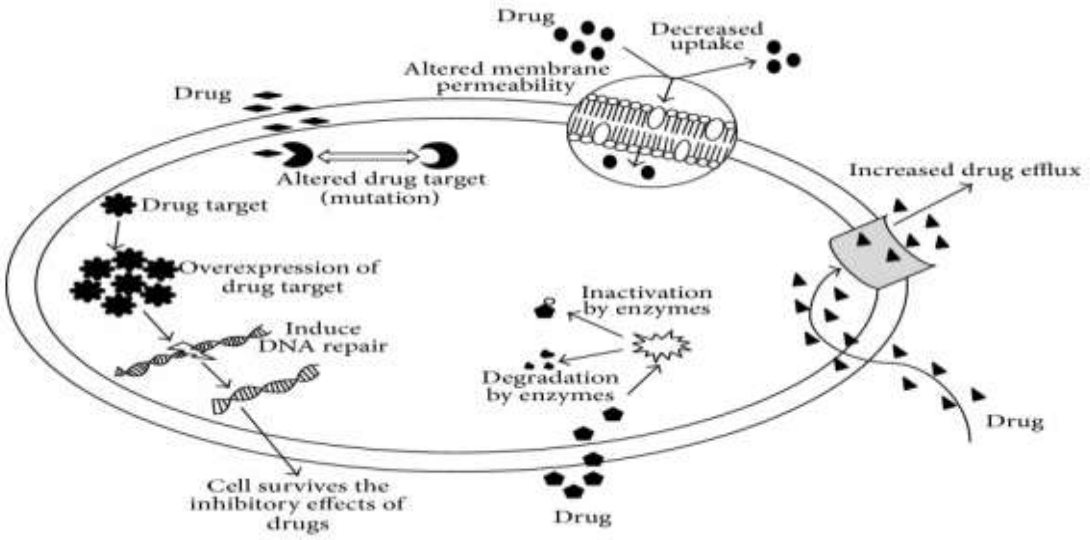

Figure 1.1: Mechanisms of Multidrug Resistance in Bacteria
Source: Tanwar *et al.* (2014)

Pelvic inflammatory disease, one of the global diseases among the female population has been previously attached to organisms associated with sexually transmitted diseases, but over time PID has been considered a polymicrobial infection (Meštrović, 2017). Bacterial species of *Staphylococcus*, *Streptococcus*, *Pseudomonas*, *Klebsiella* and *Proteus* have been implicated as major cause of pelvic inflammatory disease, which are mostly multidrug resistant.

Pelvic inflammatory disease has led to increased disease burden of infertility, ectopic pregnancy, chronic pelvic pain (American College of Obstetrician and Gynecologist, 2015) and cancer (Chan *et al.*, 1996; Chang and Parsonnet, 2010; Mitchell and Prabhu, 2013), due to treatment failures resulting from the association with resistant organisms.

1.2 Statement of the Research Problem

The emergence of multidrug resistant bacteria in many developing countries such as Nigeria has led to emergence of many life threatening diseases such as pelvic inflammatory disease, which in turn has resulted to, adverse complications (Figure 1.2) such as functional disability, emotional stress and reduced quality of life among the female populace (Vasque *et al.,* 1999).

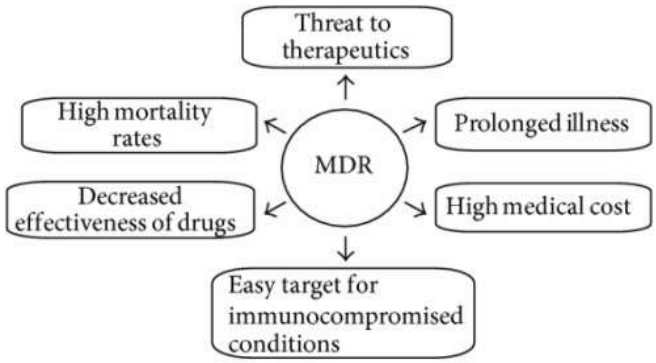

Figure 1.2: Problems Associated with Multidrug Resistant Bacteria
Source: Tanwar *et al.* (2014)

Pelvic inflammatory disease in Nigeria has been estimated to be one of the top three prevalent gynaecological problems (Usman, 2016). It is estimated that out of every 1 million women affected, 100, 000 women become infertile, while 150 women die as a result of the disease (Ahmed *et al.,* 2017). In Niger State, the percentage of women affected by PID has been estimated to be 61%. Out of the 61% affected with PID, majority of the women fall between ages 25-34years (productive age) (Usman, 2016).

1.3 Aim and Objectives of the Study

The study determined the prevalence and molecular identities of multi drug resistant bacteria isolated from patients with pelvic inflammatory diseases attending nine (9) General Hospitals in Niger State.

The objectives of the study were to:

i. Isolate and identify the urogenital bacteria associated with pelvic infection
ii. Determine the relationship between certain factors associated with the rate of bacterial infection and the occurrence of pelvic inflammatory disease
iii. Determine the multidrug resistant bacterial load among the isolated urogenital bacterial isolates
iv. Determine the prevalence of multi drug resistant bacteria in endocervical swab and urine of patients with pelvic inflammatory disease.
v. Screen for extended spectrum beta lactamase and carbapenemase production in the urogenital bacterial isolates.
vi. Characterise the multidrug resistant genes from the urogenital bacterial isolates.

1.4 Justification for the Study

Several studies Spencer *et al*. (2014); Usman, (2016); Oseni and Odewale, (2017) in Nigeria have reported prevalence of pelvic inflammatory disease. However, there is paucity of information about the molecular identities and antibiotics susceptibility or resistance profile of the bacterial isolates associated with pelvic inflammatory disease (PID) in Niger State. The results of the study have provided information on the molecular identities and antibiotics susceptibility or resistance profile of the bacteria associated with pelvic infections in Niger State and it has also enriched the national data base of the country on the burden of the disease.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Female's Genitourinary System

The genitourinary system of a female or a female urogenital tract is the system (as seen in Figure 2.1) is made up of all organs involved in the formation and release of urine (such as kidneys, ureters, bladder and urethra) and all organs involved in reproduction (such as uterus, ovaries, fallopian tubes and vagina).

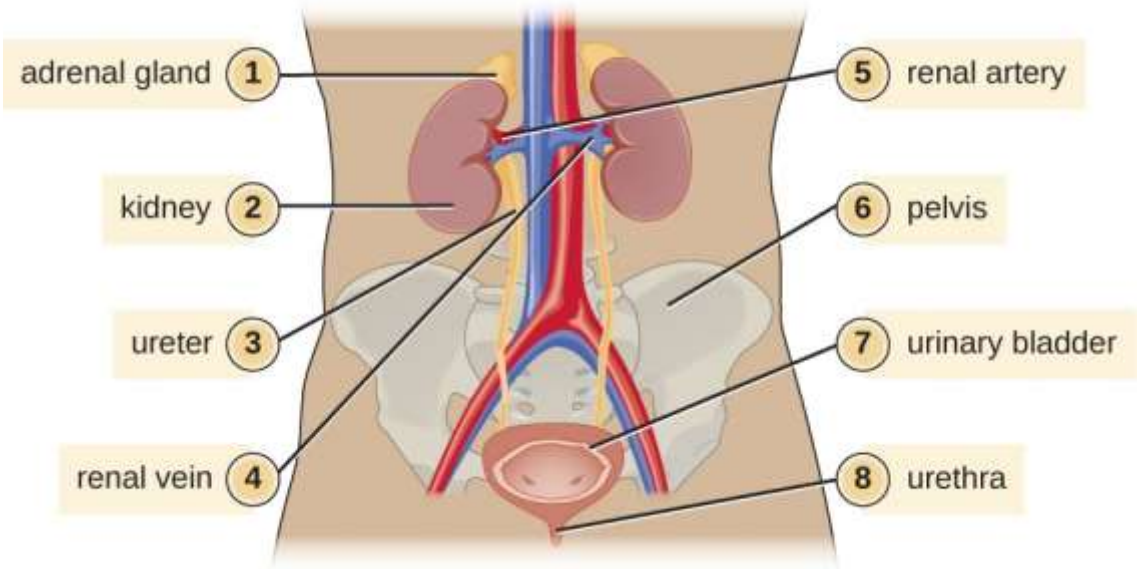

Figure 2.1: Anatomy of the Female's Genitourinary System
Source: WHO (2013)

The human pelvis or pelvic region, especially those found in females, is the lower part of the torso (that is trunk of the human body) (Schulman, 2018). It's located between the lower abdomen and the legs. This pelvic region provides support for the intestines and also contains the bladder and reproductive organs (as seen in Figure 2.2) (Irami *et al*., 2018). It is basically a bony structure formed in a ring and it encloses most female reproductive organs such as bladder, urethra, uterus, ovaries, cervix, vagina, endometrium, parametrium and rectum. The legs connect to the body at the pelvis (Schulman, 2018).

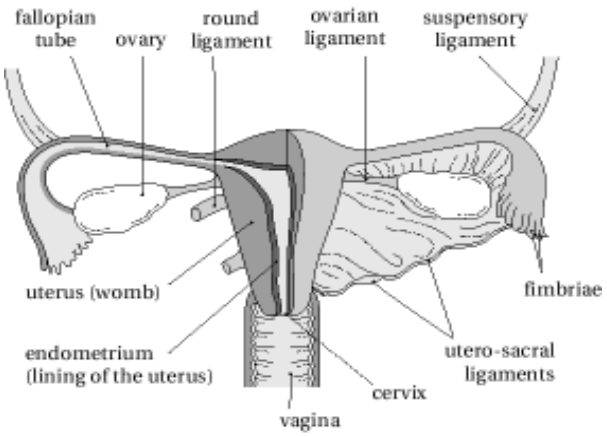

Figure 2.2: Structure of the Internal Female Reproductive Organs
Source: Brunham (2015)

They are all regarded as sterile internal female reproductive organs and are located in the pelvic area. However, most of these internal organs are usually infected by various microbes, thereby resulting into various infections, which lead to diseases such as pelvic inflammatory disease.

Pelvic inflammatory disease (PID) or pelvic inflammatory infection is referred to, as an infection in the uterus (womb), ovaries, fallopian tubes (tubes leading from the ovaries to the uterus), parametrium (connective tissue or ligaments near or around the uterus) or endometrium (lining in the womb). Basically, it is an infection of the female upper reproductive tract {the endometrium, fallopian tubes, ovaries, or pelvic peritoneum (tissue covering the pelvic region)}; it has a wide range of clinical manifestations. Pelvic inflammatory disease spreads from the vagina or cervix to the upper genital tract (Brunham *et al*., 2015; Irami *et al*., 2018).

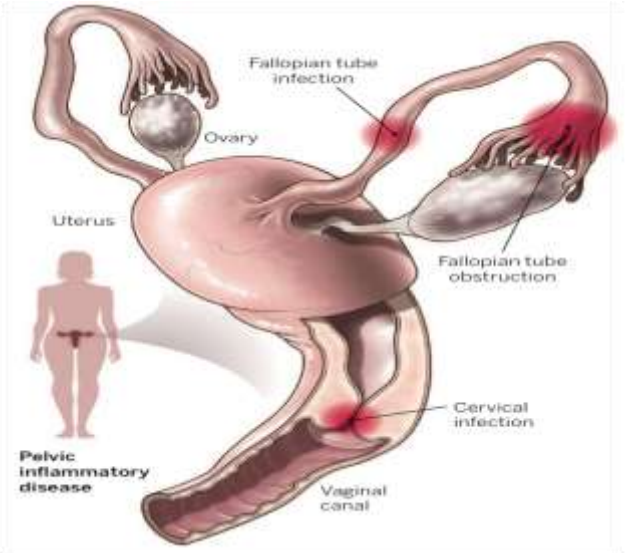

Figure 2.3: Various Organ Sites Associated with Pelvic Inflammatory Disease
Source: Hahn and Johnston (2017)

Basically, pelvic inflammatory disease, could either be acute pelvic inflammatory disease (which usually occurs when there is a short sudden inflammation or pain of the uterus, fallopian tubes or ovaries in the pelvic area due to infection); subclinical pelvic inflammatory disease (characterised by having no sign or symptoms); chronic pelvic inflammatory disease; which usually occurs when the severe inflammation, persist for a long time. Basically, acute pain usually last for 30 days or less, while chronic pelvic pain usually last for more than 30 days (American College of Obstetricians and Gynecologist, 2015). This disease is usually characterised as either being mild, severe, acute or chronic. In most women Pelvic Inflammatory Disease is asymptomatic, and up to 70% of women with PID have no clinical symptoms or signs (Ross, 2002; Nkwabong and Dingom, 2015), whereas women with symptomatic PID show symptoms such as; Abnormal vaginal discharge, pain in the lower abdomen (often a mild ache), pain in the upper right abdomen, abnormal menstrual bleeding, fever (more than 38 ^{o}C) and chills, painful urination, nausea and vomiting, painful sexual intercourse (American College of Obstetricians and Gynecologist, 2015).

Generally, PID is the clinical syndrome associated with the inflammation of the female upper genital tract, and it is mostly caused by the spread of micro-organisms from the lower genital tract such as the vagina and cervix (entrance of the uterus) to the upper genital tract such as the uterus (womb), fallopian tubes, ovaries (Simms *et al.*, 2006). If the PID is severe, the infection may result in an abscess (collection of pus) forming inside the pelvis. This is most commonly a tubo-ovarian abscess (an abscess affecting the tubes and ovaries) (Paavonen, 2008; Gradison, 2012; American College of Obstetrician and Gynecologist, 2015). Pelvic inflammatory disease (PID) is common and for every 10 visits of women under the age of 45 years, 4 to 5 cases of PID are usually accounted (Gradison, 2012).

Similarly, acquisition of these bacteria associated with PID is mainly via certain human activities such as; sexual intercourse, childbirth, miscarriage, vaginal douching or abortion; medical processes, such as; endometrial biopsy, hysterosalpingogram (HSG), hysteroscopy and artificial insemination (Irami *et al*., 2018). However, such processes are generally enhanced by several demographic factors (such as young age), behavioural factors (constant sex with multiple partners and frequent douching) and contraceptive factors (such as the use of intra uterine device), which have been identified as risk factors for PID acquisition (American College of Obstetrician and Gynecologist, 2015; Irami *et al*., 2018).

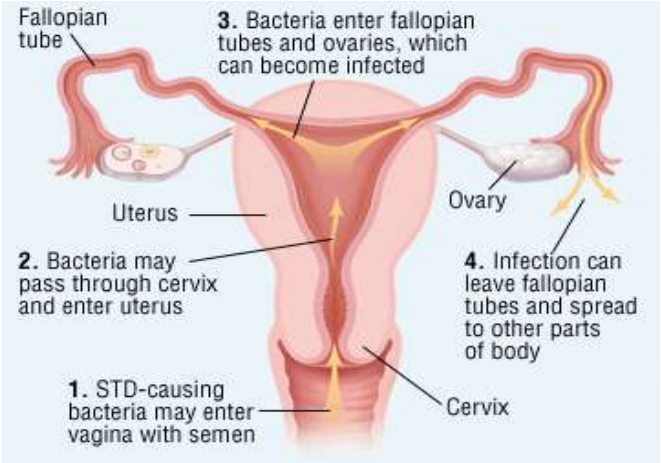

Figure 2.4: Target Sites of Infection in Female Reproductive Tract
Source: Ljubin-Sternak and Mestrovic, 2014

2.2 Microorganisms Associated with Pelvic Inflammatory Disease

Although *Chlamydia trachomatis* and *Neisseria gonorrhoeae* are said to be the main cause of PID, it is generally regarded to be polymicrobial (Ljubin-Sternak and Mestrovic, 2014) (that is, it is usually caused by many microbes) and these microorganisms are categorised into various groups opportunistic bacteria as follows; sexually transmitted bacteria (such as *Chlamydia trachomatis* and *Neisseria gonorrhoeae*, accounting for only 25 per cent of the cases in the developed countries (Bravender and Matson, 2012; Ross and Hughes, 2014), bacterial flora of the genitals or vagina (such as *Gardnerella vaginalis*, *Mycoplasma hominis*, *Mycoplasma genitalium* (Ross, 2005; Bjartling *et al.*, 2012), *Ureaplasma urealyticum*), enteric organisms (such as, *Escherichia coli* (Mitchell and Prabhu, 2013), *Enterococcus* (Bravender and Matson, 2012), and agents typically causing respiratory infections (*Haemophilus influenza, Streptococcus agalactiae*) and anaerobes (such as *Prevotella*, *Atopobium* and *Leptotrichia* species may also be implicated (Centers for Disease Control and Prevention, 2015), *Actinomyces*, *Campylobacter* and *Clostridia* species are rare causes of PID (Simms *et al.*, 2006).

The genital flora also referred to as the Endogenous" bacteria are commonly found in severe pelvic infections (such as turbo- ovarian abscess, TOA), in recurrent PID, and among intrauterine device (IUD) users and older women (Soper *et al.,* 1994). However, most of these bacteria associated with pelvic inflammatory disease are said to exhibit resistance not to one drug but in most cases many classes of antibiotics, there by resulting to multidrug resistance in them (Magiorakos *et al*., 2012).

Multi drug resistance (MDR) is a serious threat to public health. More than 70% of the existing cases of pelvic inflammatory disease are caused by organism resistant to at least one antibiotic (Centers for Disease Control and Prevention, 2015). In recent decades the incidence rate of pelvic inflammatory strains of *N. gonorrhoeae* or *C. trichomatis* has become of critical concern to clinicians worldwide and the World Health Organization, (WHO) prioritized cases of resistant strains of *N. gonorrhoeae* as one of the conditions that require high and effective attention (WHO, 2017). Multi drug resistance is a critical challenge for effective disease management. The percentages of antimicrobial resistance (AMR), especially multidrug resistance (MDR), continued to increase in developed and developing countries, leading to mounting healthcare costs, failed treatments, and deaths (Simms *et al*., 2006). However multidrug-resistance (MDR) is defined as the resistance of an organism to one agent in three or more antimicrobial classes (Oyedum, 2015), while extensively drug-resistance (XDR) is defined as resistance of an organism to one agent in all antimicrobial classes except two or fewer antimicrobial classes (that is organisms that are extensively drug-resistant are usually resistant to one agent in all antimicrobial classes but susceptible to one agent from two or less antimicrobial classes) and pan drug-resistance (PDR) is defined as resistance of an organism to all agents in all antimicrobial classes (Magiorakos *et al*., 2012).

Pelvic inflammatory disease just like many other available diseases is associated with high morbidity and mortality among the women populace (Usman, 2016**)**. This recent development is associated to the fact that most pathogenic bacteria associated with PID are of recent developing resistance to the regimen or antibiotics that were used in eradicating them. In addition, most physicians are faced with PID associated with resistant bacteria. Based on this, the world generally especially the developed and developing countries such as Nigeria are faced with PID of resistant bacteria origin (Spencer *et al*., 2014; Oseni *et al.*, 2017).

2.2.1 Resistant sexually transmitted pathogens associated with PID

Generally, about 40% of pelvic inflammatory disease is caused by sexually transmitted agents, namely; *N. gonorrhoeae* or *C. trichomatis* which are recently observed to possess resistant genes. The presence of these resistant genes in any of the sexually transmitted agents enhances and enables them to withstand the adverse effect of available drugs or antibiotics used as therapies for infections they cause (Dillon *et al.,* 2015).

2.2.2 Mechanisms of resistance in *N. gonorrhoeae*

Neisseria gonorrhoeae is a genetically diverse microorganism and it is able to take up DNA at all stages of its life cycle from other gonococci or related *Neisseria* species that are pathogenic or bacteria of other genera (Unemo and Nicholas, 2012). This ability of *N. gonorrhoeae* to acquire this external DNA has enabled *N. gonorrhoeae* develop efficient resistance mechanisms to various antimicrobial agents. These mechanisms have aided its survival among the human population and this in turn has made *N. gonorrhoeae* to be associated with high morbidity (Anschuetz *et al.,* 2012a; Unemo and Nicholas, 2012; Dillon *et al.,* 2015). *Neisseria gonorrhoeae* may become resistant to antimicrobial agents via mechanisms such as, enzymatic destruction of the antibiotic

(i.e. penicillin); target modification or protection (e.g. penicillin, tetracycline); efflux of antimicrobial agents (most classes of antibiotic) and, decreased influx of antimicrobial agents (e.g. penicillin, tetracycline) (Figure 2.5).

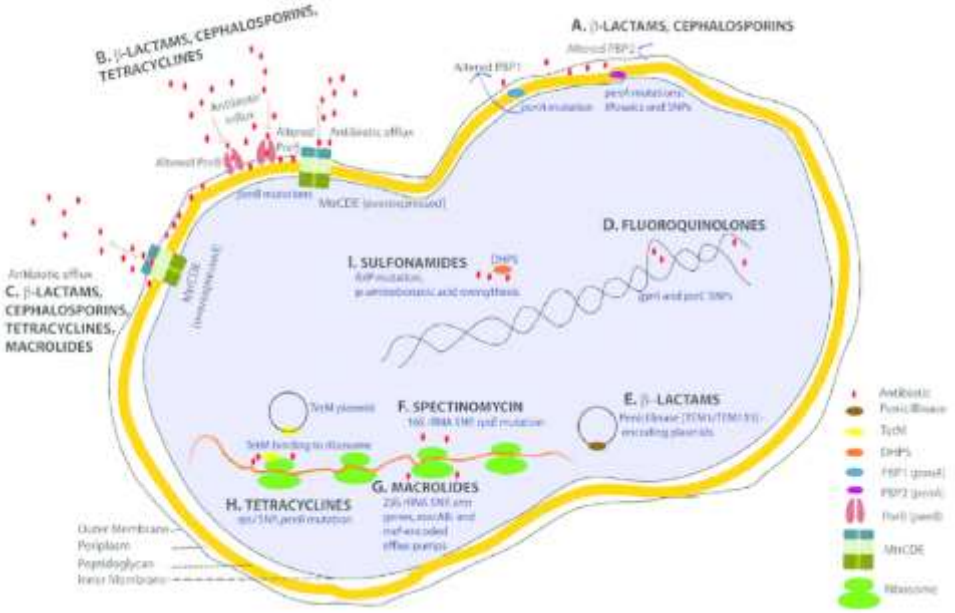

Figure 2.5: Resistance Mechanism in *N. gonorrhoeae*
Source: Dillon *et al.* (2015)

Resistance may arise either through spontaneous mutations in different chromosomal genes, through the uptake of mutated DNA acquired from transformation or through plasmids derived from conjugation (penicillin and tetracycline resistance only) (Unemo and Nicholas, 2012).

2.2.2.1 *Sulfonamides*

The emergence of resistance in *N. gonorrhoeae* isolates was observed with the first antimicrobial agents introduced to treat gonorrhea known as the Sulfonamides. Sulfonamides, discovered in 1935, were the first effective anti-gonococcal antimicrobial agents (Dillon *et al*., 2015). This antibiotic usually competes with p-aminobenzoic acid (PABA) to react with the enzyme known as, dihydropteroate synthase (DHPS), in order to prevent the formation of tetrahydrofolate (which is a major component of the

bacterial cell) needed for DNA synthesis. Resistance arose by chromosomal mutations emerging in the early 1940s, and in the early 1950s most *N. gonorrhoeae* isolates were resistant to various sulfonamides. However, Gonococci develop resistance to sulfonamides by hyperproducing (that is, over-producing) PABA, in order to avoid the binding of sulfonamides and the dihydropteroate synthase (DHPS), and this thereby prevents the inhibitory effect of sulfonamides. In addition, resistance to sulfonamides is also said to arise when there are mutations in *folP* which encodes DHPS. Such changes, brings about changes in the DHPS and it lowers the affinity of DHPS for sulfonamides (Lewis, 2014; Unemo and Shafer, 2014).

Similarly, ß-lactam antibiotics such as Penicillin was used to treat gonococcal infection in 1943 and then remained the antibiotic of choice for treating gonorrhea for many years (Goire *et al.*, 2014). Treatment failures of penicillin were reported in the early 1950s and this therefore caused the therapeutic dose of penicillin recommended for the treatment to rise, due to the fact that the susceptibility of the organism to penicillin decreased (Dillon and Pagotto, 1999). The major penicillin binding proteins (PBPs) of *N. gonorrhoeae* such as, PBP1 (coded by *ponA*) and PBP2 (coded by *penA*) are the targets of penicillin action. However, mutations in different genes, such as, *penA* (which codes for 70 different mutations), *ponA*, *mtrR* (which codes for multidrug resistance), *porB* (which regulate the activities of the outer membrane porin in the bacteria) and *pilQ* usually bring about chromosomally-mediated resistance of *N. gonorrhoeae* isolates to penicillin (Lewis, 2010; Goire *et al.*, 2014).

2.2.2.2 *Tetracyclines*

Tetracycline, discovered in 1945, was first used to treat gonorrhea primarily in patients allergic to penicillin. Chromosomal resistance to tetracycline was observed early (Dillon *et al.*, 2015; Adesoji *et al.*, 2016). Plasmid-mediated resistance to tetracycline was first

reported in United States of America, in 1985 and subsequently globally, and this led to a situation whereby the use of tetracycline for treating gonorrhea was avoided (Dillon and Pagotto, 1999). Tetracycline is still recommended in some countries for treating pelvic inflammatory disease (Lewis, 2010). Some resistance genes associated with resistance to penicillin also bring about resistance to tetracycline (Unemo and Shafer, 2014). A mutation in the 30S ribosomal protein, involved in the binding of *tRNA* to ribosomes, regulates the affinity of tetracycline to its rRNA binding site. In addition, mutations in bacterial genes such as *mtrR* and *porB* can also cause mutations and these mutations cause high-level chromosomal resistance to tetracycline (Unemo and Shafer, 2014).

2.2.2.3 *Macrolides*

Azithromycin, a macrolide that inhibits protein synthesis, is usually recommended with other antibiotic for the treatment of gonococcal infections due to the rapid development of resistance (Lewis, 2010; Lewis, 2014). Gonococcal resistance to azithromycin was first reported in 1990s, in Latin America and subsequently in North America and Europe (Dillon *et al*., 2006).

Resistance to macrolide antibiotics (azithromycin and erythromycin) in *N. gonorrhoeae* can arise by mutations in 23S rRNA – the binding site of macrolides. Such changes can cause both low and high-level resistance. Low level azithromycin resistance may be produced by the methylation of 23S rRNA by an enzyme known as, rRNA methylases (which is encoded by *ermB, ermC* and *ermF*). Also, this methylation, block the binding of macrolides to the ribosome (Roberts *et al.*, 1999).

Resistance to macrolides may also be conferred by mutations in *mtrR* that cause over expression of the efflux pump, which enhances efflux of the antibiotic. The over

expression of the efflux pump *MacAB* is also said to bring about decreased susceptibility of the organisms to macrolides (Unemo and Shafer, 2014).

2.2.2.4 *Fluoroquinolones*

Fluoroquinolones (for example ciprofloxacin, ofloxacin) were recommended to treat gonococcal infections (Lewis, 2010), when gonococci treated with previous antibiotics (such as ß-lactam antibiotics (e.g. penicillin and ampicillin), sulfonamides and tetracyclines) showed more than 5% resistant isolates (WHO, 2012). Gonococcal resistance to fluoroquinolones was first reported in the late 1980s from Asia-Pacific regions, North America and then internationally (Iverson *et al.*, 2004). Despite the high percentages of resistant isolates, fluoroquinolones are still recommended largely in some countries. The large recommendation is based on the fact that there is inadequate infrastructure of active surveillance of antimicrobial resistance and untimely modifications of treatment guidelines for gonococcal infections (Starnino and Galarza, 2012).

The quinolones block DNA replication by targeting the enzymes DNA gyrase (*gyrA*) and topoisomerase IV (*parC*). Quinolone resistance in *N. gonorrhoeae* is caused by point mutations arising in specific regions of *gyrA* (position S91 and D95) and *parC* (positions S88 and E91) (Unemo and Shafer, 2014). Mutations in DNA gyrase provides low levels of resistance to ciprofloxacin but mutations in topoisomerase IV provides high level of resistance (Unemo and Shafer, 2014; Dillon *et al.*, 2015).

2.2.2.5 *Aminoglycosides*

Aminoglycoside antibiotics, such as streptomycin, were used in the 1950s to treat gonococcal infections (Dillon *et al.*, 2015). However, their use was not widespread as *N. gonorrhoeae* isolates may become resistant to high levels of these antibiotics in a single mutational step. The aminoglycosides such as kanamycin and gentamicin have

been used in a few countries (Indonesia, Malawi) for the treatment of gonorrhea alone or in combination with other antibiotics (Lewis, 2014).

Extended-spectrum cephalosporins (ESCs), is currently the recommended primary treatment for gonococcal infections. They were first discovered in the late 1940s. They include both cefixime, an oral antibiotic, and ceftriaxone, which is administered intramuscularly; cefixime is the antibiotic of choice due to its ease of administration (Lewis, 2010). Reports from Europe, North America, Japan and South Africa have been documented on resistance of *N. gonorrhoeae* to cefixime and ceftriaxone (WHO, 2012). However, from all indications, ceftriaxone was meant to serve as the last choice of drug used as single treatment with regards to gonococcal infections but unfortunately, cases of confirmed treatment failure with ceftriaxone in Japan, Australia, Sweden and Slovenia have been reported (Lewis, 2014).

Cefixime resistance is mainly conferred by *penA*, but the occurrence of *mtrR* and *porB* in a bacterial cell confer little resistance to cefixime. However, the ceftriaxone resistance in a bacterial cell is mainly conferred by the presence of these three genes namely: *penA*, *mtrR* and *porB*. The exact mechanisms of resistance to ESCs are complex and involve different combinations of mutations, within multiple genes (Unemo and Shafer, 2014).

2.2.3 Resistant genital pathogens associated with PID

2.2.3.1 *Mechanisms of resistance in Mycoplasma and Ureaplasma*

Members of the genera *Mycoplasma* and *Ureaplasma* have no cell wall, they are insensitive to all types of β-lactam antibiotics (that is they are not affected by β-lactam antibiotics) and this is said to be a natural resistance (Unemo and Shafer, 2014). Sulfonamide also has no effect on these bacteria due to the absence of the metabolic pathway for the synthesis of folic acid. Most *Mycoplasma* and *Ureaplasma* spp have

been observed to exhibit high rate of resistance to macrolides (such as erythromycin and tetracycline) and moxifloxacin (Xie, 2011; Redelinghuys *et al.*, 2014; Waites and Xiao, 2015). Similarly strains of *M. genitalium* resistant to fluoroquinolone have been increased and reported in Japan (Kikuchi *et al.*, 2014).

The resistance of *Mycoplasma* and *Ureaplasma* to antibiotics is primarily associated with mutations in the 23S rRNA (which enhances resistance to macrolides) and *gyrA, gyrB, parC,* or *parE* gene (which enhances resistance to fluoroquinolones). The mutations responsible for macrolide, lincosamide, streptogramin, or ketolide group resistance occur in 23S rRNA in *M. hominis* and *M. genitalium*. The efflux genes and *erm* gene that contribute to the resistance of *Ureaplasma* to macrolides have been detected (Lu *et al.*, 2010).

2.2.3.2 *Mechanisms of resistance in Gardnerella vaginalis* (*G.vaginalis)*

Gardnerella vaginalis is treated with metronidazole (Nagaraja, 2008; Tomusiak *et al.*, 2011). Resistance to metronidazole has been found in some *G. vaginalis* strains. However, such resistance occurs due to certain mechanisms such as:

- A suppressed rate of activation of the drug inside the cell;
- Increased activity of DNA repair systems;
- Increased activity of enzymes that consume oxygen (i.e., catalase, peroxidase, and superoxide reductase);
- Removal of the drug from the cell by active efflux (Dhand and Snydman, 2009; Löfmark *et al.*, 2010).

2.2.3.3 *Mechanisms of resistance in Bacteroides fragilis (B. fragilis)*

The resistance of *B. fragilis* to different antimicrobial drugs has increased. Multidrug-resistant *B. fragilis* isolates resistant to imipenem, amoxicillin and metronidazole or

clindamycin were also found in Russia (Shilnikova and Dmitrieva, 2015). Antibiotic resistance is spread horizontally among the *B. fragilis* group of clinical isolates due to the antibiotic resistance genes carried on conjugative and mobilizable plasmids, conjugative transposons and integrated genetic elements (Eitel *et al.,* 2013).

The most important mechanism of resistance of *B. fragilis* to β-lactam antibiotics is the production of β-lactamases (Edwards, 1997). The *cepA* gene encodes β-lactamase, which is able to destroy penicillins and most cephalosporins (except cefoxitin). Resistance of cefoxitin-resistant strains was basically due to the presence of the *cfxA* gene located on the transposon carried on the conjugative elements of some of the *B. fragilis*. BexA pump (an example of efflux pump, also known as multi drug efflux pumps), is usually encoded by the *bexA* gene and it is responsible for the resistance of *B. fragilis* to fluoroquinolones and the elevated moxifloxacin (Unemo and Shafer, 2014).

2.2.4 Mechanisms of resistance in enteric pathogens associated with PID

The resistance of most enteric organisms, such as *E.coli, Klebsiella pneumoniae, Proteus sp, Pseudomonas sp* and *Salmonella sp,* occur when they possess, an outer membrane (which decreases the membrane permeability, thereby preventing the uptake and accumulation of antimicrobials), efflux pumping proteins (which pump out antimicrobials, thereby preventing the accumulation of antimicrobials) or enzymes (which inactivates antibiotics either by hydrolysis or by modification) (Wright, 2005; Yoneyama and Katsumata, 2006).

Generally, the cell wall of these enteric bacteria consists of a thin peptidoglycan and an outer membrane, which is also known as a lipid bilayer. This lipid bilayer usually acts as a barrier to the penetration of many antibiotics into the cell. The outer portion of this

lipid bilayer, is composed principally of lipopolysaccharide made up of tightly bound hydrocarbon molecules that prevents the entry of antibiotics (Labischinski *et al.,* 1985).

Efflux pumps are basically transporters that expel or export substances that are toxic to the bacterial cells such as antibiotics (Lin *et al.,* 2003). Basically most *E.coli* possess efflux pumps, which usually pumps antibiotics out, before they reach their targets. Most of these efflux pumps (widely spread in bacteria) (Langton *et al.,* 2005) expel many classes of drugs, and this in turn lead to resistance to varieties of drugs, thus encouraging the bacteria to be multidrug resistance (Yoneyama and Katsumata, 2006). Such efflux pumps are regarded as multidrug efflux pumps (Unemo and Shafer, 2014).

Some of these enteric bacteria, produce bacterial enzymes that reside within or near the cell surface, which selectively target and inactivate various drug used to treat them. Enzymatic inactivation can either be achieved by hydrolysis or by modification (group transfer and redox mechanisms). It is a major natural mechanism of resistance to antibiotics in pathogenic bacteria. Basically, enteric bacteria exhibit resistance to β-lactam antibiotics through the production of β-lactamases. Which are enzymes that inactivate these antibiotics by splitting the amide bond of the β-lactam ring (Yoneyama and Katsumata, 2006).

2.2.4.1 *Types of various betalactamases in enterobacteriaceae*

Beta-Lactamase is an enzyme produced by an organism that breaks down beta-lactams antibiotics, (which are antibiotics that consists of a chemical structure that includes a three-carbon, one-nitrogen cyclic amine structure known as the beta-lactam ring) such as; Penicillins (for example ampicillin and oxacillins); Cephalosporins (for example first, second and third generation cephasporins) as seen below in (Figure 2.6); Monobactams (for example aztreonam); Carbapenems (for example imipenems and monopenems) and Carbacephems (for example loracabef) (Unemo and Shafer, 2014).

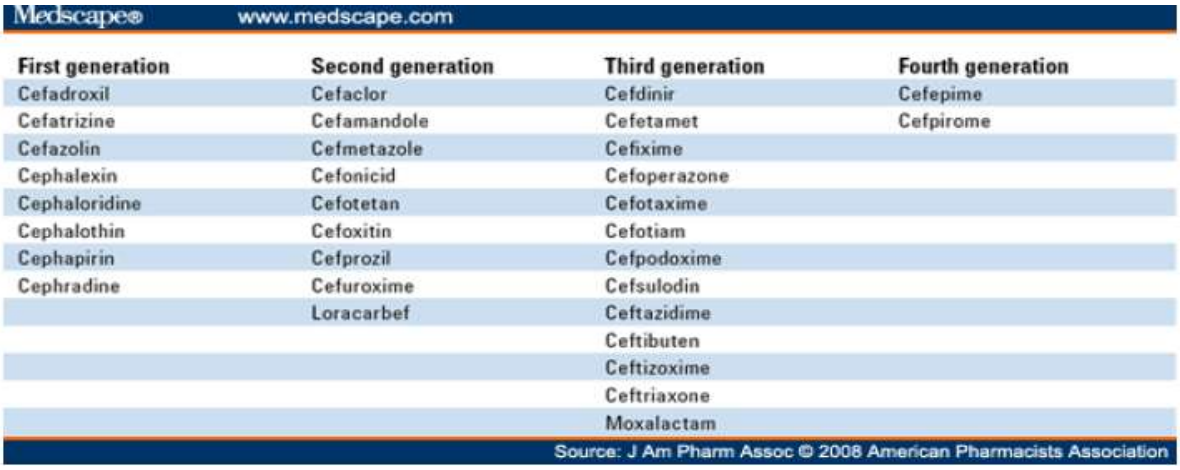

Figure 2.6: Various Types of First, Second, Third and Fourth Generation of Cephalosporins
Source: Thenmozhi *et al.* (2014)

However apart from conferring resistance to betalactam antibiotic which is a class of antibiotic, betalactamase also confer resistance other classes of antibiotics, such as Fluoroquinolones, Aminoglycosides, Trimethoprim and sulfamethoxazole. Beta-Lactamase is an enzyme produced by bacteria, that breaks down betalactam ring and this in turn ensures resistance to varous betalactam antibiotics used to treat infections when they occur (Unemo and Shafer, 2014).

The betalactamases have been classified according to their function or their structure (Schultsz and Geerlings, 2012). The functional classification is based on the substrate specificity of the enzymes and it categorizes betalactamase as narrow spectrum betalactamases, such as penicillinases and cephalosporinases (which usually occur when there is frequent exposure to the penicillins and the first and second generation cephalosporins); extended-spectrum betalactamases (ESBLs) (which arises there is frequent exposure to extended spectrum cephalosporins, which are mainly the third and fourth cephalosporins) and carbapenemases (which arises when there is frequent exposure to carbapenems).The structural classification of betalactamases categorizes betalactamases into four clases (namely: class A, B, C and D) based on the protein

similarities of the enzymes and also the substance the enzyme contains in their active site. Betalactamase class A, C and D are usually referred to as the serine betalactamases, based on the fact, that they contain a serine group at their active site, while betalactamase class B are called the metallo-betalactamases (MBL), based on the fact they require a zinc ion (in their active site) for hydrolysis of betalactam antibiotics (Schultsz and Geerlings, 2012; Sartelli *et al.,* 2016). The first discovered β-lactamase was Temoniera β-lactamase or TEM β-lactamase (which occur due to the presence of of the TEM- gene) was recovered from a clinical isolate of *E. coli*, which was recovered from a Greece patient named Temoniera, while sulfhydryl variable (SHV) β-lactamase (which occur due to the presence of the SHV- gene) was the second β-lactamase to be discovere and it was recovered from *Klebsiella pneumoniae* isolates (Shaikh *et al.,* 2015). Both TEM beta lactamases and SHV beta lactamases evolved from resistance to narrow or broad-spectrum penicillins such as ampicillin, tigecycline and piperacillin but not to the oxyimino substituted cephalosporins. Similarly, these enzymes are usually regarded as narrow spectrum β-lactamases base on their large potentials to hydrolyse penicillins and first generation cephalosporins; hence they are refered to as either penicillinase or cephalosporinase. Due to substitution of one or more amino acids of the active sites of TEM and SHV-β-lactamases, the spectrum of these two enzymes was extended to include 3rd generation cephalosporins and monobactams (Ahmed *et al.,* 2017) and hence, they are termed extended spectrum β-lactamases (ESBLs) (Sartelli *et al.,* 2016).

2.2.4.2 *Extended-spectrum β-lactamase (ESBL)*

Extended-spectrum betalactamases (ESBLs) are β-lactamases that posses the abilities to hydrolyze extended spectrum cephalosporins with an oxyimino side chain. These Cephalosporins include Cefotaxime, Ceftriaxone and Ceftazidime, as well as the

oxyimino-monobactam such as Aztreonam. Thus, ESBLs confer resistance to these antibiotics and related oxyimino-β lactams (Ahmed *et al.,* 2017). Basically, they are derived when genes of TEM or SHV undergo mutation, which is said to alter the amino acid configuration around the active site of this β-lactamases. Based on this mutation there is an extended spectrum of β-lactamase resistance; hence such enzymes are referred to as extended spectrum β-lactamases (ESBLs). The ESBLs are frequently plasmid encoded and as such can be exchanged between bacteria. Plasmids responsible for ESBL production frequently carry genes encoding resistance to other drug classes (for example, amino glycosides, Flouroquinolones and so on). Extended spectrum β-lactamases (ESBLs) can be inhibited by β-lactamases-inhibitors such as clavulanic acid, tazobactam or sulbactam (Shaikh *et al.,* 2015).

2.2.4.3 *Types of extended-spectrum β-lactamase*

(i) ***Temoniera β-lactamases (TEM β-lactamases)***

TEM-1 is the most commonly-encountered β-lactamase in Gram-negative bacteria. Up to 90% of ampicillin resistance in *E. coli* is due to the production of TEM. Although TEM-type β-lactamases are most often found in *E. coli* and *K. pneumoniae*, they are also found in other species of Gram-negative bacteria with increasing frequency. The amino acid substitutions responsible for the ESBL phenotype cluster around the active site of the enzyme and change its configuration, allowing access to oxyimino-β-lactam substrates. Single amino acid substitutions at positions of 104, 164, 238, and 240 produce the ESBL-potentials (Ahmed *et al.,* 2017). However, ESBLs with phenotypic characteristics of broadest spectrum is usually said to occur, due to more than a single amino acid substitution (Thenmozhi *et al.,* 2014; Shaikh *et al.,* 2015). However, based on the different combinations of changes (that is different amino acid substitutions), 140

TEM-type enzymes with broadest spectrum have been discovered. TEM-10, TEM-12, and TEM-26 are among the most common in the United States (Shaikh *et al.,* 2015).

(ii) ***Sulfhydryl variable β-lactamases (SHV β-lactamases)***

SHV-1 (that is the first isolated SHV) shares 68 percent of its amino acids with TEM-1 (that is the first isolated TEM) and has a similar structure with the TEM-enzymes. The SHV-1 β-lactamase is most commonly found in *K. pneumoniae* and is responsible for up to 20% of the plasmid mediated ampicillin resistance in this species. ESBLs in this family also have amino acid changes around the active site, most commonly at positions 238 or 238 and 240 and these mutations have resulted to more than 60 types of SHV enzymes with ESBL potentials (Thenmozhi *et al.,* 2014). They are the predominant ESBL type in Europe and the United States and are found worldwide (Ahmed *et al.,* 2017). SHV-5 and SHV-12 are among the most common (Shaikh *et al.,* 2015).

(iii) ***Cefotaximase Munich β-lactamases (CTX-M β-lactamases)***

These enzymes are said to have rapid hydrolysis for cefotaxime (one of the extended spectrum beta lactam antibiotics) or greater activity against cefotaxime than other oxyimino-β-lactam substrates (e.g., ceftazidime, ceftriaxone, or cefepime). In addition to this, another unique feature of these enzymes is that they are better inhibited by the beta-lactamase inhibitor tazobactam than by sulbactam and clavulanate (clavulanic acid). Rather than arising by mutation, they are regarded as β-lactamase genes normally found on the chromosome, that are acquired by the horizontal gene transfer from other bacteria using genetic materials such as conjugative plasmid or transposon (Shaikh *et al.,* 2015). However, despite their name, a few are more active on ceftazidime than cefotaxime. They have mainly been found in strains of *Salmonella* sp*, E. coli* and other species of Enterobacteriaceae. CTX-M-14, CTX-M-3, and CTX-M-2 are the most

widespread, while CTX-M-15 is currently the most widespread type in *E. coli* (Shaikh *et al.,* 2015).

(iv) *Oxacillinases β-lactamases (OXA β-lactamases)*

Oxacillinases are beta-lactamases that are resistant to ampicillin, cephalothin, oxacillin, cloxacillin and beta-lactamase inhibitor such as clavulanic acid. Based on their high hydrolytic activity against Oxacillin and Cloxacillin, they are referred to as Oxacillinases. They belong to the class D betalactamase. They are generally encoded by plasmids (Shaikh *et al.,* 2015). Similarly, just like in the betalactamases of TEM and SHV, substitutions in the amino acid in OXA enzymes can also give the OXA enzymes that posses phenotypic extended spectrum resistance (Ahmed *et al.,* 2017). While most ESBLs have been found in *E. coli, K. pneumoniae,* and other Enterobacteriaceae, the OXA type ESBLs (that is the oxacillinase enzymes that are extended spectrum betalactamase) have been found mainly in *P. aeruginosa* and other Enterobacteriaceae (Thenmozhi *et al.,* 2014). OXA beta-lactamases have resistance limited to the penicillins, but some became able to confer resistance to cephalosporins. OXA-1 and OXA-10 beta-lactamases have only a narrow hydrolytic spectrum on antibiotics such as, penicillin and first generation cephalosporins (Shaikh *et al.,* 2015). However, other OXA beta-lactamases including OXA-11, -14, -15, -16, -17, -28, -31, -35 and -45 confer resistance to cefotaxime, ceftazidime and aztreonam (Shaikh *et al.,* 2015; Ahmed *et al.,* 2017).

(v) *Other extended-spectrum β-lactamase*

Other plasmid-mediated ESBLs, such as PER (*Pseudomomnas* extended resistance), VEB (Vietnam extended spectrum β-lactamases), GES (Guyana ESBLs), and IBC (Integron-borne cephalosporinase) β-lactamases, have been described but are

uncommon and have been found mainly in *P. aeruginosa* and at a limited number of geographic sites. *Pseudomomnas* extended resistance-1 (PER-1) in isolates in Turkey, Korea, France, and Italy; VEB-1 and VEB-2 in strains from Southeast Asia; and GES-1, GES-2, and IBC-2 in isolates from South Africa, France, and Greece (Thenmozhi *et al.,* 2014; Shaikh *et al.,* 2015). Some of these enzymes are found in Enterobacteriaceae as well, whereas other uncommon ESBLs (such as BES-1 (Brazil extended spectrum-1), IBC-1 (Integron borne cephalosporins), SFO-1 (*Serratia fonticola* betalactamase) and TLA-1(Tlahuicas indianas betalactamase) have also been found only in Enterobacteriaceae (Thenmozhi *et al.,* 2014)

2.2.4.4 *AmpC-type β-lactamases*

AmpC type β-lactamases are β-lactamases that are commonly isolated from Gram negative bacteria that are resistant to extended-spectrum cephalosporin. Basically, unlike ESBLs, AmpC β-lactamases, hydrolyse broad and extended-spectrum Cephalosporins (Cephamycins as well as to oxyimino-β lactams) and they are not inhibited by β-lactamase inhibitors such as clavulanic acid (Thenmozhi *et al.,* 2014). AmpC β-lactamases are typically encoded on the chromosome of many Gram-negative bacteria including *Citrobacter*, *Serratia* and *Enterobacter* species where its expression is usually inducible (noticeable); it may also occur on *Escherichia coli* but is not usually inducible. AmpC type β-lactamases may also be carried on plasmids (Thenmozhi *et al.,* 2014).

2.2.4.5 *Carbapenemases*

Carbapenemases are a diverse group of β-lactamases that are active not only against the oxyimino cephalosporins and Cephamycins but also against the carbapenems. Based on their high hydrolytic activity on carbapenems (such as imipenems, meropenems, ertapenems and doripenems), they regarded as Carbapenemases. Basically, most

metallo betalactamases (such as NDM-1 (new Delhi metallo betalactamase-1), VIM or IMP) are regarded as carbapenemase producers. However, most of these metallo betalactamases exhibit poor hydrolytic activity on monobactams (such as Aztreonam), except IMP and VIM producers, that are resistant to monobactams due to other mechanisms. They are usually not inhibited by clavulanic acid or tazobactam (Thenmozhi *et al.,* 2014; Shaikh *et al.,* 2015). There are various types of carbapenemases, and they are:

(i) ***Imipenem-encoded metallo-β-lactamases (IMP-type carbapenemases)***

This is a Carbapenemase that is regarded as one of the metallo betalactamases. It is usually carried on the plasmids. Basically 17 varieties of these carbapenemase have been found in *Pseudomonas, Acinetobacter* species and also other enteric Gram-negative organisms (Thenmozhi *et al.,* 2014).

(ii) ***Verona integron-encoded metallo-β-lactamase (VIM)***

The VIM- *β-lactamase* is the second growing family of carbapenemases. It consists of 10 members with a wide geographical distribution in Europe, South America and United states. VIM enzymes occur mostly in *P. aeruginosa*, also very rarely in other members of Enterobacteriaceae. Amino acid sequence diversity is up to 10% in the VIM family, 15% in the IMP family, and 70% between VIM and IMP. Basically, enzymes of both VIM and IMP are usually integron-associated and sometimes they are carried within the plasmids. Both hydrolyse all β-lactams except monobactams, and they are inhibited by all β-lactamase inhibitors (Thenmozhi *et al.,* 2014; Shaikh *et al.,* 2015).

(iii) ***Oxacillinase (OXA) group of β-lactamases***

The OXA β-lactamases is also another type of carbapenemase which mainly occurs in Acinetobacter species and other Enterobacteriaceae. In most cases, the hydrolytic

activity of OXA carbapenemases is usually augmented by additional resistance mechanisms, such as impermeability or efflux. OXA carbapenemases also tend to have a reduced hydrolytic efficiency towards penicillins and cephalosporins (Shaikh *et al.,* 2015). OXA-23 and OXA-48 are classes of carbapenemases that belong to OXA-type beta-lactamases with carbapenem-hydrolyzing activities. While OXA-23 appears most frequently in *Acinetobacter baumannii*, OXA-48 enzymes have now become widespread in the Enterobacteriaceae (Sartelli *et al.,* 2016).

(iv) ***KPC (Klebsiella pneumoniae Carbapenemase)***

Klebsiella pneumoniae carbapenemases (KPCs) are beta-lactamases produced by Gram-negative bacteria. It was first isolated from *Klebsiella pneumoniae* but has recently been isolated from other members of Enterobacteriaceae (Shaikh *et al.,* 2015; Sartelli *et al.,* 2016). They efficiently hydrolyse penicillins, all cephalosporins, monobactams, beta-lactamase inhibitors, and even carbapenems. They are becoming an increasingly significant problem worldwide. They are also carried on the plasmids (Thenmozhi *et al.,* 2014). Ten variants, KPC-2 through KPC-11 are known, and they are distinguished by one or two amino-acid substitutions. *Klebsiella pneumoniae* carbapenemase (KPC) is currently the most common carbapenemase, which was first detected in North Carolina, US, in 1996 and has since spread among members of the Enterobacteriaceae worldwide (Thenmozhi *et al.,* 2014).

(v) ***CMY- Carbapenemase***

This is also another carbapenemase which was first isolated from a virulent strain of *Enterobacter aerogenes*. It is carried on a plasmid, pYMG-1, and is therefore transmissible to other bacterial strains (Thenmozhi *et al.,* 2014).

2.2.5 Mechanisms of Resistance in Respiratory Pathogens Associated with PID

Respiratory pathogens such as *S. pneumoniae* exhibit resistance to macrolide and penicillin by altering antibiotic's target site and their penicillin binding proteins (PBPs) on their cell wall, hence a decreased affinity for penicillin, is said to occur (Cornick and Bentley, 2012). PBPs are membrane-bound proteins or enzymes found on the cell walls of pneumococci. The PBPs usually enhances the attachment of the pneumococci to any surface, but once antibiotics are involved, the pneumococci are said to alternate its binding sites susceptible to the available antibiotic. The alteration of this target site is usually achieved by methylation of the 23S ribosomal target site (that is, the introduction of a methyl group to the 23S ribosomal target site) (Song, 2013). Methylation of the 23S ribosomal target site, is usually achieved by regulatory the activity of *ermB* gene. Similarly, resistance of macrolide can also be achieved via efflux pump, which is usually encoded by the *mef* genes (namely; *mefA* and *mefE*) (Wierzbowski *et al.,* 2005).

Also, the resistance of respiratory organisms to fluoroquinolones is usually mediated by (spontaneous point) mutations in the quinolone resistance determinant region of *gyrA* and/or *parC* (Cornick and Bentley, 2012). The target site, for most fluoroquinolones such as ciprofloxacin and levofloxacin in most respiratory organisms is the gene *parC,* while the target site for fluoroquinolones such as moxifloxacin (Li *et al.,* 2002) is the gene known as *gyrA,* which inhibits DNA gyrase.

2.3 Microbiology and Pathogenesis of Pelvic Inflammatory Diseases

The intermittent ascension of microorganisms from the lower genitourinary tract into the endometrial cavity and fallopian tubes likely occurs as a normal physiological phenomenon. The ability of these organisms to initiate PID depends on their viability,

number, pathogenicity, and immune defense mechanisms of the host (Hahn and Johnson, 2017).

Many different organisms can cause PID, but in most cases two common bacteria associated with PID are gonococcus (*Neisseria gonorrhea*) and *Chlamydia trichomatis*. In addition, their virulent nature subjects them to be very significant in causing PID. Basically, the virulent nature of these two common bacteria associated with PID occurs, when the *Chlamydia triichomatis* possess chlamydial heat shock protein 60 (CHSP60) (Kinnunen *et al.*, 2002) and *N gonorrhoeae* possess P9Opa(b) protein (Avan *et al.*, 2001; Makepeace *et al.*, 2001).

Basically, organisms associated with PID usually infect the cervix. Once they are in the cervix, as they grow, they alter the pH of the vaginal environment and increase microbiologic waste products (often regarded as nutrients for the growth of other endogenous and anaerobic flora). However, this leads to a condition referred to as bacterial vaginosis (abnormal growth of microbes around the vagina). The occurrence of bacterial vaginosis, brings about complex disruption of the vaginal flora that leads to the loss of lactobacilli that normally produce hydrogen peroxide, and the overgrowth of *Gardnerella*, *Mobiluncus*, *Prevotella*, alpha-hemolytic streptococci and black-pigmented anaerobic rods (Meštrović, 2017). These bacteria produce enzymes that break up the cervical canal or the physical barrier of the cervix (Hahn and Johnson, 2017). The distortion of this protective barrier, enables these organisms ascend to the various organs in the upper genital tracts, thereby resulting to acute PID.

The ascension of these organisms into various female upper reproductive tract cause either infection of the fallopian tubes, endometrium or the pelvic peritoneum, which trigger an inflammatory response. In the fallopian tubes, it causes sloughing (casting

off) of some cells and invades others. Thereafter, the bacteria are said to multiply within and beneath the cells of the fallopian tube (Meštrović, 2017). The infection then spreads to other organs such as the ovaries, uterus, endometrium e.t.c. resulting in more inflammation and scarring. Although, the tissue of normal fallopian tube has millions of tiny hair-like cilia that beat in waves in order to assist the transportation of the egg through the tube to the uterine cavity, presence of bacteria in the fallopian tube result to inflammation and tissue destruction of the fallopian tube. In addition, the tissues of the fallopian tube lose cilia, and this lead to dysregulation of egg transport and increased risk of ectopic pregnancy. Generally, the damage and scarring caused by PID may lead to the described sequelae of infertility, ectopic pregnancy, and chronic pelvic pain (Brunham, 2015)

The presence of a cervical mucus plug normally helps prevent the spread of microorganisms to the upper genital tract, but it is less effective during ovulation (growing of egg) and menses (flow of blood). Based on this, the bacteria associated with PID gain access easily during menses to the upper genital tract organs from the lower genital organs, especially if menstrual blood flows backward from the uterus into the fallopian tubes, carrying the organisms with it (Meštrović, 2017). In most cases, these natural protective mechanisms are impaired during menstruation. Also, after delivery or abortion, the cervical canal becomes dilated (that is expanded), the vaginal pH increases, and the protective epithelial lining of the endometrium is shed. All of that renders the genital tract more vulnerable and prone to infection (Meštrović, 2017)

The vector theory suggests that pathogens present in the lower genital tract are transported in a piggyback fashion by organisms possessing greater powers of locomotion. Both *Trichomonas vaginalis* and spermatozoa have been shown in vitro to

be capable of transporting potential pathogens that adhere to their surfaces and have been nominated as possible vectors (Hahn and Johnson, 2017).

Clinical factors associated with the ascension of microbes from the lower reproductive tract include frequency of intercourse, bacteriospermia (bacteria in semen), menstrual timing, diagnostic and therapeutic surgical procedures that disrupt the normal cervical barrier (for example, abortion, intrauterine device [IUD] insertion, hysterosalpingogram), non-use of hormonal contraceptives, hygiene practices (e.g., douching), and disturbance of normal vaginal flora from bacterial vaginosis (Trent, 2013).

2.4 Pathophysiology of Pelvic Inflammatory Diseases

Most cases of PID are presumed to occur in 2 stages. The first stage is acquisition of a vaginal or cervical infection. This infection is often sexually transmitted and may be asymptomatic. The second stage is direct ascent of microorganisms from the vagina or cervix to the upper genital tract, with infection and inflammation of these structures (Hahn and Johnson, 2017).

The mechanism (or mechanisms) by which microorganisms ascend from the lower genital tract is unclear. Studies suggest that multiple factors may be involved. Although cervical mucus provides a functional barrier against upward spread, the efficacy of this barrier may be decreased by vaginal inflammation and by hormonal changes that occur during ovulation and menstruation (Workowski and Bolan, 2015). In addition, antibiotic treatment of sexually transmitted infections can disrupt the balance of endogenous flora in the lower genital tract, causing normally nonpathogenic organisms to overgrow and ascend. Opening of the cervix during menstruation, along with retrograde menstrual flow, may also facilitate ascent of microorganisms (Meštrović, 2017).

Intercourse may contribute to the ascent of infection through rhythmic uterine contractions occurring during orgasm. Bacteria may also be carried along with sperm into the uterus and fallopian tubes (Patton *et al.,* 1993). In the upper tract, a number of microbial and host factors appear to influence the degree of inflammation that occurs and, thus, the amount of subsequent scarring that develops. Infection of the fallopian tubes initially affects the mucosa, but inflammation may rapidly become transmural. This inflammation, which appears to be mediated by complement, may increase in intensity with subsequent infections. Inflammation may extend to uninfected parametrial structures, including the bowel. Infection may extend via spillage of purulent materials from the fallopian tubes or via lymphatic spread beyond the pelvis to produce acute peritonitis and acute perihepatitis (Fitz-Hugh−Curtis syndrome) (Meštrović, 2017).

2.5 Clinical Manifestations

Women with PID present with a wide array of clinical manifestations that range from virtually asymptomatic to severe and debilitating symptoms. Women with acute PID may experience subtle, nonspecific symptoms such as dyspareunia, dysuria, or gastrointestinal symptoms, which they may not attribute to pelvic infection (Eschenbach *et al.,* 1997). This leads to a failure to seek care for many patients. When mild to moderate symptoms of PID do occur, women may complain of lower abdominal or pelvic pain, cramping, or dysuria. They may also exhibit signs such as intermittent or post-coital vaginal bleeding, vaginal discharge, or fever. Systemic signs, such as fever, chills, nausea, and vomiting are often absent in mild to moderate cases. On physical examination, there may be no external evidence of infection, but uterine tenderness, cervical motion pain, or adnexal tenderness is most often present (Hahn and Johnson, 2017; Meštrović, 2017). In severe PID, women appear very ill with fever, chills,

purulent vaginal discharge, nausea, vomiting, and elevated white blood cell count (WBC). Other laboratory indicators, such as erythrocyte sedimentation rate (ESR), may also be elevated. As seen with mild and moderate disease, uterine tenderness, cervical motion pain, with or without adnexal tenderness are expected. Available data suggest that some women develop subclinical upper genital tract infection that can nevertheless result in long-term sequelae, including infertility (Wiesenfeld *et al.,* 2012). The development of "silent PID" poses a major diagnostic and treatment challenge (Workowski and Bolan, 2015).

Women with acute PID can develop a range of inflammatory complications, including local tissue damage, fallopian tube swelling, tubal occlusion, and development of adhesions (Figure 2.7) (Rosen *et al.,* 2009). This may be accompanied by fallopian adhesions, tube obstruction and the development of a tubo-ovarian abscess (Hahn and Johnson, 2017).

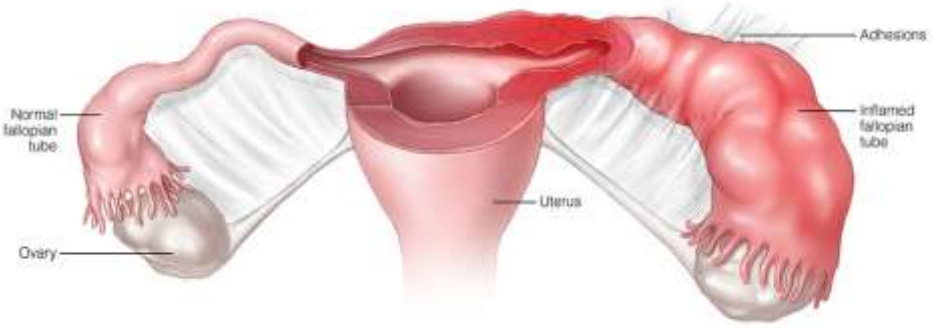

Figure 2.7: Acute Salpingitis with Pelvic Inflammatory Disease
Source: Hahn and Johnson (2017)

Although uncommon, the adhesion formation can involve the liver capsule and cause a perihepatitis referred to as the Fitz-Hugh Curtis Syndrome (Peter *et al.,* 2004; Eschenbach, 2008). The development of a tubo-ovarian abscess can occur as a subacute

complication of acute PID and some women will have a tubo-ovarian abscess at the time they present with acute PID (Chappell and Wiesenfeld, 2012).

2.6 Chronic Sequelae Associated with PID

The sequelae of PID, including ectopic pregnancy, infertility, or chronic pelvic pain may occur after a single episode of symptomatic PID. One recent retrospective cohort study of women admitted with PID or tubo-ovarian abscess (TOA) found that, in follow-up, 25.5% of the women met the criteria of infertility, 16.0% had recurrent PID, and 13.8% reported chronic pelvic pain (Chayachinda and Rekhawasin, 2016). Several studies have demonstrated that multiple episodes or more severe cases dramatically increase women's risk for infertility as well as for ectopic pregnancy (Weström *et al.,* 1992). The risk of ectopic pregnancy is increased 6- to 10-fold after PID. Tubal infertility occurs in 8% of women after one episode of PID, in 20% of women after two episodes of PID, and in 50% of women after three episodes of PID (Chayachinda and Rekhawasin, 2016). In addition to this, high burden of sequale arising from the manifestations of pelvic inflammatory disease is usually said to occur, when pathogens responsible for the occurring PID is resistant to one or more existing antibiotics (Workowski and Bolan, 2015).

2.7 Types of Pelvic Inflammatory Diseases

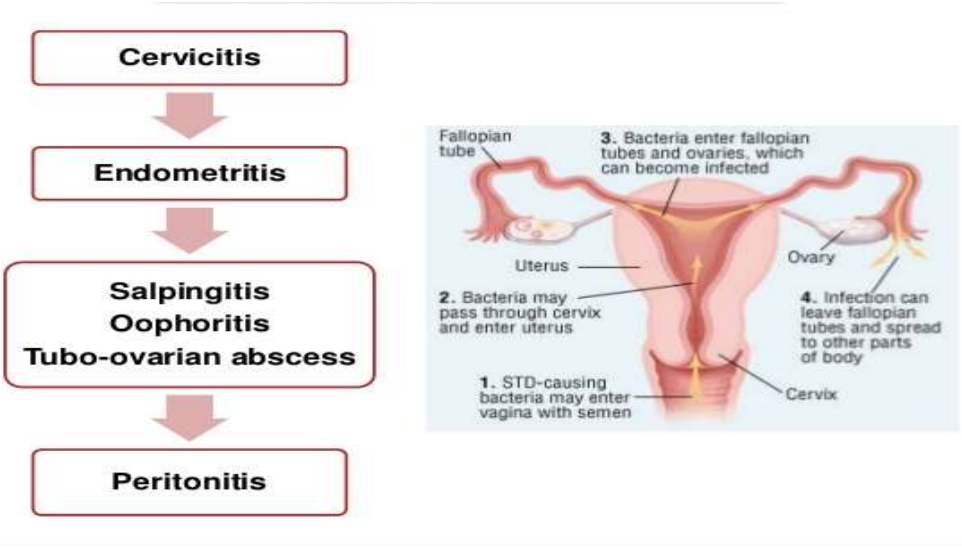

Figure 2.8: Acute PID: Pathology
Source: Bansal and Gupta (2015)

2.7.1 Cervicitis

Cervicitis is an inflammation (irritation) of the lining of the cervix. The cervix is the tip of the uterus (womb), and extends down into the vagina. Cervicitis is inflammation of the cervix, which can be due to infection, irritation or injury of cells that surrounds cervix (Bansal and Gupta, 2015). Cervicitis generally caused by infections that are passed during sexual activity. Sexually transmitted diseases that may cause cervicitis include, Gonorrhea, Chlamydia, Genital herpes, Trichomoniasis, *Mycoplasma* and *Ureaplasma*. Symptoms of cervicitis include Pain during sex, difficult, painful and frequent urination, Pale yellow vaginal discharge and Abnormal vaginal bleeding (Rivlin, 2018).

2.7.2 Endometritis

Endometritis is inflammation of the endometrial lining of the uterus. In addition to the endometrium, inflammation may involve the myometrium and, occasionally, the parametrium. Endometritis can be divided into pregnancy-related endometritis and endometritis unrelated to pregnancy. When the condition is unrelated to pregnancy, it is

referred to as pelvic inflammatory disease (PID). Endometritis is often associated with inflammation of the fallopian tubes (salpingitis), ovaries (oophoritis), and pelvic peritoneum (pelvic peritonitis) (Rivlin, 2018). Endometritis can be classified as acute versus chronic. Acute endometritis is characterized by the presence of neutrophils within the endometrial glands. Chronic endometritis is characterized by the presence of plasma cells and lymphocytes within the endometrium (Bansal and Gupta, 2015).

Endometritis is a polymicrobial disease, involving on average 2-3 organisms. In most cases, it arises from an ascending infection from organisms found in the normal indigenous vaginal flora. Commonly isolated organisms include *Ureaplasma urealyticum, Peptostreptococcus, Gardnerella vaginalis, Bacteroides bivius,* and group B *Streptococcus*, *Chlamydia* or *Enterococcus* (Rivlin, 2018).

2.7.3 Salpingitis

Salpingitis is an acute inflammation of the fallopian tubes. Although it is most commonly caused by sexually transmitted micro-organisms such as *N. gonorrhoeae, Chlamydia trachomatis* in adolescent and adult women, other bacteria mostly associated with salpingitis are, *Mycoplasma* sp, *Staphylococcus* sp and *Streptococcus* sp. Salpingitis occurs in an estimated 15% of reproductive-age women, and 2.5% of all women become infertile as a result of salpingitis by age 35 (Fertilitypedia, 2018a). It is very uncommon in premenarchal or sexually inactive girls. The infection usually has its origin in the vagina, and ascends to the fallopian tube from there. Based on the fact that, the infection can spread via the lymph vessels, infection in one fallopian tube usually leads to infection of the other (Bansal and Gupta, 2015).

There are two types of salpingitis: acute salpingitis and chronic salpingitis. In acute salpingitis, the fallopian tubes are extensively swollen. The main symptom of acute

salpingitis is pelvic pain. The cause of acute salpingitis is vaginal infection. Other causes are for example a surgery or a procedure (such as insertion of an IUD). There is a leakage of fluid that can cause tubal walls sticking to each other or to other organs. Acute salpingitis poses a risk of rupturing the wall of the fallopian tube and subsequent infection of the abdominal cavity. Acute salpingitis and swelling fallopian tube are conditions that arise due to pelvic inflammatory disease (PID) (Hahn and Johnson, 2017).

After acute salpingitis, chronic salpingitis may follow. Chronic salpingitis is usually milder, longer and is not as pronounced symptoms. Salpingitis may be diagnosed by pelvic examination, blood tests, and/or a vaginal or cervical swab (Rivlin, 2018).

2.7.4 Oophoritis

An inflammation of single or pair of ovaries is called oophoritis. Sometimes this term is used to describe the inflammation of pelvis. Very often, the inflammation spreads from ovaries to the fallopian tubes, which is called salpingo-oophoritis (Fertilitypedia, 2018b). On the other hand, when there is inflammatory process in Fallopian tubes it can affects surrounding pelvic organs including ovaries. Oophoritis can be caused by bacterial infection. However, pathological bacteria such as Gonorrhea and Chlamydia can infect the cervix and help other less invasive microorganisms to invade the fallopian tubes and reach the ovaries where they cause inflammation. Gonorrhea and Chlamydia are very common in young, sexually active women and the most common age of oophoritis is 25 years (Fertilitypedia, 2018b).

2.7.5 Tubo-ovarian abscess (TOA)

This is one of the late complications of pelvic inflammatory disease (PID) and can be life-threatening if the abscess ruptures and results in sepsis. It consists of an encapsulated or confined 'pocket of pus' with defined boundaries that forms during an

infection of a fallopian tube and ovary. These abscesses are found most commonly in reproductive age women and typically result from upper genital tract infection (Beigi, 2016).

A tubo-ovarian abscess (TOA) is an inflammatory mass involving the fallopian tube, ovary and, occasionally, other adjacent pelvic organs (for example, bowel, bladder) (Granberg *et al.*, 2009). These abscesses are found most commonly in reproductive age women and typically result from upper genital tract infection. Tubo-ovarian abscess (TOA) is a serious and potentially life-threatening condition. Aggressive medical and/or surgical therapy is required and rupture of an abscess may result in sepsis. Prior to the advent of broad-spectrum antibiotics and modern surgical practice, the mortality rate associated with TOA was approximately 50 percent or higher (Vermeeren and Te Linde, 1954). The mortality rate approaches zero for abscesses that are not ruptured. For patients with ruptured abscesses, current mortality rates have not been reported, but data from the 1960s suggest the rate may be as high as 1.7 to 3.7 percent (Rosen *et al.*, 2009).

2.7.6 Peritonitis

Peritonitis is inflammation of the peritoneum, the lining of the inner wall of the abdomen and cover of the abdominal organs (Ferri, 2017). Symptoms may include severe pain, swelling of the abdomen, fever, or weight loss (Gradison, 2012). One part or the entire abdomen may be tender. Complications may include shock and acute respiratory distress syndrome (Gradison, 2012). Causes of peritonitis include perforation of the intestinal tract, pancreatitis, pelvic inflammatory disease, stomach ulcer, cirrhosis, or a ruptured appendix (Ferri, 2017).

2.8 Epidemiology

Pelvic inflammatory disease (PID) affects 1 million American women annually in most developed countries such as the United States and affects more population of females in developing countries such as Nigeria. Similarly, in most developing countries, PID is basically polymicrobial and it is said to be on the increase (Molander, 2003; Okon *et al.*, 2008). Basically, sexually active adolescents usually make up 20% to 30% of PID cases and in most cases may have higher risk of PID compared to the adult females. Pelvic inflammatory disease (PID) results in more than 600,000 hospital admissions yearly and leads to serious long-term complications in 25% of women who are infected. The sequelae is particularly high among adolescents because of late presentation, delayed diagnosis, and inadequate treatment. The increased risk of PID during adolescence reflects both the biologic susceptibility of the immature cervix and the high prevalence of sexual behaviors that are risky.

PID cases are caused by *C. trachomatis* (11%-42%), although the rates vary over time and between countries (Molander, 2003). This wide range in rates reflects the background prevalence of these pathogens among different populations (Paavonen, 1998). Estimates are that, 10% to 40% of women with chlamydial cervicitis manifest PID (Stamm *et al*., 1984), while approximately 10 to 19% of women with *N. gonorrhoeae* manifest PID (Molander, 2003).

Pachori and Kulkarni (2016) determined that the incidence of PID was 36.7%, in a study that was carried out, however the antibiogram, as well as the molecular characterization of the isolates were not determined. Similarly, Oseni and Odewale (2017), also deduced that the prevalence rate of PID among the Female undergraduates in Irrua Specialist Teaching Hospital, Edo state, Nigeria is high (54%) and this could result to complications such as infertility, ectopic pregnancy and chronic pelvic pain. In

addition, Spencer *et al.* (2014) in a study carried out in Abuja revealed high (61%) prevalence rate of PID and various bacterial isolates. Although he reported the susceptibility pattern of these isolated bacteria, there was no report with regards to the molecular identity of the multi drug resistant strains associated with PID.

2.9 Diagnosis of Pelvic Inflammatory Disease

The diagnosis of pelvic inflammatory disease, especially acute pelvic inflammatory disease is usually difficult to determine based on the fact that most signs and symptoms are varied among different infected female individuals. Most PID cases have been mistaken for appendicitis, endometriosis and ectopic pregnancy. However, before the diagnosis of PID is said to commence, physicians or health care workers are expected to examine and determine if patients show certain signs and symptoms (such as pelvic pain, which is one of the commonest signs and symptoms detected in PID cases). However, in most cases the pelvic pain may be mild or absent in proven cases of PID (Eckert *et al.*, 2003). In general, due to the nature of PID (which may be asymptomatic in some cases), diagnoses are based on the following steps below;

2.9.1 Physical examination

A physical examination is basically referred to as the clinical examination. It is basically of two types namely; the general examination and the pelvic examination and in most cases, clinical examination begins with a general examination (Workowski and Bolan, 2015).

The general examination involves identifying the patient's temperature (which should exceed >100°F [>38°C]) light and deep palpation (touching) of the abdomen and blood and urine test. Light and deep palpation (touching) of the abdomen would elicit any lower abdominal tenderness, which is usually bilateral (that is affecting the left and

right sides of the abdomen). Blood test is also one of the general examinations carried out on a patient suspected to have PID. It is basically carried out to determine the presence of abundant numbers of white blood cells (WBCs) on saline microscopy of vaginal fluid; elevated erythrocyte sedimentation rate (ESR) and wet mount polymorphonuclear leukocytes (Peipert *et al.*, 2004; Crossman, 2006).

The pelvic examination involves the inspection of the external genitalia looking for any obvious vaginal discharge. This is then followed by a speculum examination to expose the vagina and cervix and look for any mucopurulent or purulent exudate at the endocervix. A bimanual examination is then performed to reveal one or more of the following minimum criteria (Workowski and Bolan, 2015): cervical motion tenderness, uterine tenderness and adnexal tenderness, which have been discovered to indicate high sensitivity cases of PID at 92%, 94% and 96% respectively (Haggerty and Ness, 2008). The clinical diagnosis is used for detecting cerviticitis, endometritis and both cervicitis and endometritis together (Rivlin, 2018).

2.9.2 Confirmation examination

More elaborate diagnostic evaluation is frequently needed when the diagnosis is questionable or the patient is not responding to therapy after physical examination. Confirmation examination involves further investigation such as imaging and invasive tests (Vandermeer and Wong-You-Cheong, 2009).

2.9.2.1 *Invasive Tests*

Basically there are two types of invasive tests namely;

i. *Laparoscopy*

Laparoscopy is a minor operation where two small cuts are made in a female's abdomen inorder to insert a thin camera into the body to view the internal pelvic organs (Rivlin, 2018). However, this diagnostic tool enables specimens to be taken from the fallopian tubes, and is particularly useful when there is diagnostic doubt. Laparoscopy will not detect endometritis or subtle inflammation of the fallopian tubes, but will be used to detect salpingitis. It should not be used as a routine diagnostic tool, especially when symptoms are mild or vague (Workowski and Bolan, 2015).

ii. *Endometrial biopsy*

Endometrial biopsy is usually carried out when women undergoing laparoscopy who do not have visual evidence of salpingitis (Workowski and Bolan, 2015). The presence of neutrophils and plasma cells in the endometrium is indicative of endometritis and may be used to diagnose PID (Cohen *et al.,* 2005).

2.9.2.2 *Imaging tests*

Imaging tests are reserved for patients who are severely ill or unresponsive to initial therapy. Imaging tests are most helpful when ruling out competing differential diagnoses, for example, the use of pelvic ultrasonography is used to rule out symptomatic ovarian cysts and computed tomography is used to rule out appendicitis. Basically pelvic ultrasonography is used to detect fluid-filled tubes and this is an indication of pelvic inflammatory disease or upper genital tract inflammation (Timor-Tritsch and Rottem, 1987). Pelvic ultrasonography should be ordered in patients

requiring hospitalization or those with a chronic or severe pelvic pain. There are various types of imaging tests namely; transabdominal ultrasound, transvaginal ultrasound, Computer tomography (CT), and Magnetic resonance imaging (MRI) (Workowski and Bolan, 2015).

i. ***Pelvic ultrasound or ultrasonography test***

This is a test whereby sound waves are used to create images of various female reproductive organs. This test uses color or power Doppler to detect abnormalities of endometritis, salpingitis, and oophoritis (Horrow, 2004). Basically, there are two types of pelvic ultrasound, they are the trans-abdominal ultrasound and transvaginal ultrasound. Transvaginal ultrasonography is preferred to transabdominal approach and it is helpful in guiding needles to drain abscesses (Horrow, 2004).

ii. ***Pelvic magnetic resonance imaging***

This test is considered superior to ultrasound at diagnosing PID when there is a tubo-ovarian abscess, fluid-filled tube, and/or enlarged polycystic ovaries with free intra-pelvic fluid. However, both ultrasound and CT are more cost effective than MRI (that is MRI is expensive but more sensitive). Therefore, MRI is rarely used and plays only a complementary problem-solving role (Vandermeer and Wong-You-Cheong, 2009). MRI is more accurate than transvaginal ultrasound and provides information about the differential diagnosis of PID (Workowski and Bolan, 2015).

iii. ***Pelvic computer tomography***

Pelvic Computer Tomography is indicated in patients with diffuse pelvic pain, peritonitis, or difficult ultrasound. It should be performed with both oral and intravenous therapy (Vandermeer and Wong-You-Cheong, 2009). Computed

tomography (CT) is reserved for evaluation of the extent of PID within the abdomen (Workowski and Bolan, 2015).

Basically, when imaging tests are employed, the first imaging test to consider is transvaginal ultrasound, and if PID are detected on ultrasound (Timor-Tritsch *et al.*, 1987) no further imaging test is required. However, if additional imaging test is required, Magnetic Resonance Imaging (MRI) would be recommended over CT because its overall accuracy is greater than 93% and does not carry the additional risk of ionizing radiation (Workowski and Bolan, 2015).

2.10 Treatment of Pelvic Inflammatory Disease

The treatment of PID can be divided into inpatient and outpatient treatment strategies as shown in respectively (Sweet, 2011). Basically, for females with mild-to-moderate disease, outpatient treatment such as oral therapies and parenteral or intravenous regimens are employed. Both oral therapies (such as cefoxitin or cefotetan plus deoxycycline and clindamycin plus gentamicin) and parenteral or intravenous regimens (such as ampicillin plus deoxycycline and erythromycin-based medications) appear to be equally effective (Workowski and Bolan, 2015). Patients who fail outpatient therapy are categorized to have severe or complicated pelvic inflammatory disease (such as, tubo-ovarian abscess, pregnancy, unable to take oral medications) or patients who are deemed for surgical emergencies are usually considered for inpatient therapy (Workowski and Bolan, 2015).

Several antimicrobial regimens have demonstrated efficacy in achieving clinical and microbiologic cure in randomized clinical trials (Sweet, 2011). Most regimens contain at least one agent with activity against anaerobic organisms which are said to be associated with PID. Metronidazole is usually added to the treatment of PID episodes,

based on the fact that most PID episodes occur due to concurrent bacterial vaginosis (BV) (Mesopan *et al.,* 2016). Due to the emergence of most treatment failure, fluoroquinolone therapy is utilized when parenteral (that is the injectable) therapy is not feasible or patient allergies prevent the selection of certain therapies. Doxycycline, a common component of both inpatient and outpatient regimens, should be administered orally based on the fact that it is caustic to veins when administered intravenously (Workowski and Bolan, 2015).

2.11 Prevention of Pelvic Inflammatory Disease

2.11.1 Practice safe sex

The very best way to prevent PID is to abstain from sex, including oral, vaginal and anal sex with multiple partners. Sex should be encouraged with a mutually monogamous sexual partner (such as one's spouse) to avoid the spread of sexually transmitted resistant bacteria that are potential cause of PID (Mesopan *et al.,* 2016; Rivlin, 2018).

2.11.2 Screen early for bacteria associated with sexually transmitted disease and treat PID

Physicians should encourage that people under the ages of 25 who are sexually active be screened or tested for bacteria associated with sexually transmitted disease, which are potential sources of PID annually (Rivlin, 2018). Women who have more than one sexual partner throughout the year should visit a gynecologist for a pap smear in order to catch any STDs in their early stages. Diagnosis with PID should be treated immediately with antibiotics along with their male partners to lower their risk of future re-encounter of PID and long-term complications which could lead to permanent damage to the reproductive system. However, pregnant and nursing women need to be especially cautious when treating early cases of PID and should always seek the help of

a physician to avoid complications in the foetus and babies dependent on their mother's breast milk (Mesopan *et al.,* 2016).

2.11.3 Antibiotics should be taken well to avoid resistance

Physicians should prescribe appropriate antibiotic to PID patients and educate them on the misuse of antibiotics and importance of complete dosage of antibiotics, to ensure that the causative bacteria of PID is completely eradicated and the development of resistant bacteria avoided (Mesopan *et al.,* 2016).

2.11.4 Prevent vaginosis and other common infections

Bacterial vaginosis (infection that occurs due to abnormal growth/overgrowth of bacteria in the vagina) a potential cause of PID can recur within three to 12 months if one has had it before. Some of the ways to prevent pelvic infections from developing or recurring include: (Rivlin, 2018):

a. Avoid using mild soap and detergent: Washing the vagina with commercial (usually alkaline) soaps can cause skin irritation, imbalance in pH and microflora, and increased vaginal discharge. A safer option is to use clean water to cleanse the vagina, which is naturally a self-cleaning organ (Rivlin, 2018).
b. Avoid using feminine deodorant sprays, perfumed or dyed products near the vagina (such as lubricants or scented pads).
c. Pads should be changed at least three times daily (at least every 6–8 hours) to avoid bacterial overgrowth.
d. Avoid sharing under wears and washing them with strong detergents containing perfumes and other chemicals that can rub off onto your skin.
e. Boost your overall immunity: A strong immune system won't protect one from acquiring a PID. But it may help prevent recurring infections like vaginosis and decrease risk for complications. Various ways of boosting immunity against

infections include: eating a healthy diet; taking probiotics and eating probiotic foods (probiotics including *Lactobacillus* species increase the number of "good bacteria" in the vagina and re-establish a balanced microflora); addressing allergies, nutrient deficiencies, diabetes and digestive problems; exercising, sleeping enough and avoiding medications that may contribute to infections (Mesopan *et al.,* 2016; Rivlin, 2018).

2.11.5 Avoid douching

Douching disrupts the normal bacterial balance inside the vagina, it's a risk factor for introducing bacteria, especially resistant bacteria associated with PID. Douching does not actually help to cleanse the vagina, but actually makes an infection worse by removing beneficial bacteria that are there to protect against harmful bacteria. Physicians should encourage that cleaning the vagina should be done with clean water (Mesopan *et al.,* 2016).

2.11.6 Ensure good hygiene habits

Be sure to wipe from front to back after a bowel movement to avoid spreading bacteria, especially resistant bacteria which could be potential sources of PID from the rectal area to the vagina (Rivlin, 2018).

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 Description of the Study Area

The study was conducted in Niger State (Figure 3.1), Nigeria. The State is located in the middle belt zone of the country. It lies between latitude 8˚20′N and 11˚30′N and longitudes 3˚30′E and 7˚20′E. It shares common boundaries with other States namely: Zamfara State to the north; Kaduna State to the north- east; Kebbi State to the north-west, Kogi State to the south; Federal Capital Territory (FCT) and Kwara State to the south-east and south-west respectively (Usman, 2016). The State covers a land area of about (76,363km^2), (29,484 square miles). About 85% of the populace in the State are involved in agriculture, particularly farming and they are majorly rural dwellers, while about 15% are urban dwellers, involved in white collar jobs, businesses, crafts and arts (Usman, 2016).

Key

: Nine (9) General Hospitals that were studied

Figure 3.1: Twenty-five (25) Local Government Areas in Niger State
Source: Usman (2016)

The State has three geopolitical zones, each zone with a distinct climate pattern and a defined agricultural system. Zone A is found in the Southern part of the State and it comprises Agaie, Bida, Edati, Katcha, Gbako, Lapai, Lavun and Mokwa Local Government Areas; while zone B comprises of Bosso, Chanchaga, Gurara, Kuta, Paikoro Rafi, Shiroro, Suleja and Tafa Local Government Areas and zone C comprises of Agwara, Borgu, Kontagora, Magama, Mariga, Mashegu, Rijau and Wushishi Local Government Areas (Figure 3.1). Nine (9) Local Government Areas (comprising 3 Local Government Areas from each zone) were randomly selected for this study.

3.2 Sample Size

The sample size was determined using the equation below (Idakwo, 2015).

$$\boldsymbol{n} = \frac{\mathrm{T}^2 \ \mathbf{x} \ \ \mathbf{P}(\mathbf{1}-\mathbf{P})}{\mathrm{m}^2} \_ \_ \_ \_ \_ \_ \_ \_ \_ \_ \_ \_ \_ \qquad (3.1)$$

Where

n= required sample size

T= Confidence level at 95% (standard value of 1.96)

P= Prevalence rate of bacterial infection in Niger State (9.3%) (Usman,2016)

m= Margin of error at 5% (Standard value of 0.05)

n= $(1.96^2 \text{x } 0.093 \text{ x } 0.907)/0.05^2 = 129.6 \cong 130$

The sample size for each L.G.A was (n) =130

Total samples for 9 local government areas =130 x 9= 1170 samples.

A total of 1170 samples were collected from 9 general hospitals located in 9 Local Government Areas of Niger State (Lapai, Bida, Agaie, Minna, Suleja, Kuta, Kontagora, Wushishi and Nasko).

3.3 Inclusion and Exclusion Criteria

Female patients within the age of 15-54 years diagnosed of PID and are attendees of the selected hospitals were recruited for this study. Female patients above 54 years and less than 15 years not diagnosed of PID and who are not attendees of the selected hospitals were excluded from this study.

3.4 Ethical Clearance

Ethical clearance for this study was sought from the Niger State Hospital Management board, Research and Ethics Committee.

3.5 Collection of Demographic and Clinical Data

A structured questionnaire was administered to obtain patient's demographic data (such as patient's location, age and awareness of the disease); patient's previous medical history; patient's socio-economic factors (such as patient's family status, occupation and education) and risk factors (such as douching frequency, source of water and type of toilet facility) as described by Kolo (2016) and Oseni and Odewale (2017).

3.6 Collection and Transportation of Samples

3.6.1 Endocervical samples

Sterile flexible swab sticks were used for the collection of swabs from the endocervical region of patients enrolled for the study (Einwalter *et al.,* 2005; Pachori and Kulkarni, 2016; Oseni and Odewale, 2017).

The samples were removed and submerged into normal saline and were taken to the Microbiology Laboratory of Federal University of Technology, Minna for further analysis (Enwa *et al.,* 2015).

3.6.2 Urine samples

Five millilitres (5 ml) of fresh urine were collected from each female patient diagnosed of PID into a universal bottle. The urine samples were transported to the Microbiology Laboratory of Federal University of Technology, Minna under cold condition (Hunter *et al*., 2013). The urine samples were stored at 4^{o}C for 24 hours for further analysis (Hunter *et al*., 2013).

3.7 Direct Examination

Saline wet preparation was carried out in order to rule out the presence of *Trichomonas vaginalis* which is characteristically associated with a yellow-green discharge, itching, redness and swelling (Spencer *et al.*, 2014).

3.8 Culture of Bacteria

The endocervical swabs and the serial dilution 10^{-4} of each urine samples were cultured and incubated on the following media such as Nutrient agar, MacConkey agar and *Salmonella- Shigella* agar at 37^{o}C for 24 hours for the isolation of Gram-negative and Gram-positive bacteria. Pure culture of each isolate was obtained by repeated sub-culturing using the streak method. The pure isolates were stored on a nutrient agar slant for further identification and characterization (Kolo, 2016).

3.9 Gram Staining Technique

Smear of the isolates were prepared with a wire loop by emulsifying a colony of the isolates with a drop of distilled water on a clean glass slide free of grease and was used to make a thin smear. The smear was air – dried and was heat fixed. The smear was flooded with crystal violet stain for sixty seconds and was washed with clean running tap water (Cheesbrough, 2010). The slide was tipped off and flooded with lugol's iodine for sixty seconds and washed with clean water. The smear was decolourized rapidly

using alcohol and washed immediately with clean water. The smear was flooded with neutral red stain (safranin) for one minute, washed with clean water and the back was wiped with clean cotton wool and the smear was allowed to air dry. The dried slides were examined microscopically under oil immersion, using x100 objective lens and the results recorded (Cheesbrough, 2010; Kolo, 2016).

3.10 Biochemical Tests

The bacterial isolates were identified based on the following conventional biochemical tests such as; Coagulase, Oxidase, Catalase, Citrate, Urease, Indole and Triple sugar agar test (Cheesbrough, 2010).

3.10.1 Coagulase test

A drop of distilled water was placed on two separate slides and a colony of the test isolate was emulsified on two slides to make a thick suspension. A loopful of plasma was added to one of the suspensions. Observation for agglutination reaction was done within 10 seconds of adding plasma cells (Cheesbrough, 2010).

3.10.2 Catalase test

Three milliliters of 3% hydrogen peroxide solution was added into a sterile test tube. A sterile wire loop was used to pick colonies of the test isolates and was immersed in the hydrogen peroxide solution. Observation for bubbles was done immediately and results recorded (Cheesbrough, 2010).

3.10.3 Triple sugar iron agar test

The test was performed to determine the ability of bacteria to ferment various carbohydrates such as glucose, lactose and sucrose. Inoculation with the test organism was done by stabbing through the centre of the medium to the bottom of the tube and the test organism was streaked on the surface of the agar slants. The agar slants were

incubated at 37ºC for 24 hours. Observations for colour change of the phenol red indicator to yellow both at the butt and slants (due to the fermentation of either glucose, lactose or sucrose), gas production indicated by bubbles or cracks on the medium and hydrogen sulphide (H_2S) production indicated by black pigment coloration was done and recorded (Cheesbrough, 2010).

3.10.4 Citrate test

Simmon citrate agar slants were prepared and the surfaces streaked with isolates and incubated at 37ºC for 24 hours. Observation for colour change was done and the results recorded (Cheesbrough, 2010).

3.10.5 Urease test

Urea agar slants were prepared and inoculated with test organisms and incubated at 37ºC for 24 hours. Observation for colour change was made and results recorded (Cheesbrough, 2010).

3.10.6 Indole test

A wire loop of the test organisms was inoculated in the test tubes containing peptone broth at 37ºC for 24 hours. After 24 hours, 0.5 ml of Kovac reagents was added into the test tubes and the solution was thoroughly mixed. Observations for colour change was made and results recorded (Cheesbrough, 2010).

3.10.7 Voges Proskauer test

A wire loop of the test organisms was inoculated in the test tubes containing peptone broth at 37ºC for 24 hours. One millilitre (1ml) of 40% potassium hydroxide (KOH) and 3 ml of 5% alcoholic alphanaphthol was added to the broth medium and then mixed

properly. Observation for colour change was made and results recorded (Cheesbrough, 2010).

3.10.8 Oxidase test

A piece of filter paper was placed in a sterile Petri dish and two drops of freshly prepared oxidase reagent (referred to as tetra-p-diaminechloride) was applied onto the piece of filter paper. A colony of the test organisms was introduced onto the soaked filtered paper. Observation for blue-purple colour within few seconds was done and result recorded (Cheesbrough, 2010).

3.11 Antimicrobial Susceptibility Testing of Isolates

3.11.1 Preparation of turbidity standard for the inoculums

The McFarland standard was employed in the standardization of the test organisms. Morphologically similar colonies of each test organism were transferred aseptically from an agar plate culture into a tube containing 4 to 5 ml of nutrient broth. The broth was subjected to agitation and was incubated at 37°C until it achieved or exceeded the turbidity of the 0.5 McFarland standard. The turbidity of the actively growing culture in the broth was adjusted with sterile saline or broth to obtain turbidity that was optically comparable to that of the 0.5 McFarland standard (Lalitha, 2004).

3.11.2 Inoculation of plates

Susceptibility test of the isolates was carried out using Kirby- Bauer disc diffusion method on Mueller-Hinton agar (Clinical and Laboratory Standards Institute, 2014). A sterile cotton swab stick was dipped into the adjusted suspension. The swab stick was rotated several times by pressing it firmly on the inside wall of the tube above the fluid level to remove excess inoculums from the swab stick (Kolo, 2016). The surface of the

sterile agar was inoculated by streaking the swab over the entire sterile agar surface. This procedure was repeated by streaking two or more times, rotating the plates approximately 60^{o} each time, to ensure uniform distribution of bacteria on the plates. The inoculated plates were left for 10 minutes to ensure pre-diffussion of the organisms and to allow excess surface moisture to be absorbed, before the agar plates are impregnated with discs. Each disc was pressed down to ensure complete contact with the agar surface. The plates were inverted and incubated at 37^{o}C for 24 hours (Clinical and Laboratory Standards Institute, 2014).

3.11.3 Reading plates and interpreting results

After 24 hours of incubation, each plate was examined and the diameters of the zones of inhibition were measured, including the diameter of the disc. Zones of inhibition were measured to the nearest whole millimeter using a meter rule (Kolo, 2016).

3.11.4 Screening for antibiotics resistant bacterial isolates

Antibiotics commonly available in the study areas were used for this study. Antibiotics such as penicillin G, Augmentin, cefotaxime, ceftriaxone, tetracycline, trimethoprim/sulfamethoxazole, gentamycin, ofloxacin and chloramphenicol test strips (AB BIODISK, Sweden) were employed (Spencer *et al.,* 2014).

3.11.4.1 *Screening for multi drug resistance bacteria*

Bacteria isolates resistant to three or more classes of antibiotics according to the Clinical Laboratory Standard Institute (CLSI, 2016) guidelines were termed multi-drug resistant (MDR) bacteria (Magiorakos *et al.,* 2012; Iliyasu *et al.,* 2015).

Bacterial isolates denoted as multidrug resistant were further secreened for the presence of two types of enzymes namely:

3.11.4.2 *Screening for extended spectrum beta-lactamase production*

The screening for extended spectrum β-lactamase (ESBL) producing bacteria was performed according to the double disc synergy test described by Sarojamma and Ramakrishna (2011) as follows. MacFarland turbidity standard suspension of the test organisms was inoculated onto the surface of the Mueller-Hinton agar plate using sterile swab stick. Augmentin disc (AMC 30 µg) was placed at the center of Mueller Hinton agar plate. Around the three sides of AMC (30 µg) disc, a disc of Ceftazidime CAZ (30 µg), Cefotaxime CTX (30 µg) and Ceftriaxone CRO (30 µg) was placed with distance of fifteen (15) mm from center to center of AMC (30 µg) disc. Then the plate was incubated overnight at 37°C. The result was considered as positive results for production of ESBL if the inhibition zone increases towards the AMC (30 µg) disc.

3.11.4.3 *Phenotypic detection of carbapenemase production*

The modified carbapenem inactivation method (mCIM) was performed according to CLSI Guidelines (2016). Sterile inoculating loop was used to take and suspend 1µl of test organism in an Eppendorf tube containing 400µl (0.4 ml) of sterile water. The bacterial suspension was thoroughly homogenized for 5 minutes (Hamed and Hasoon, 2019). Then, a 10-µg imipenem disc was immersed into the bacterial suspension. Subsequently, the bacterial suspension containing the imipenem disc was incubated for 2 hours at 37°C. A sterile swab stick was used to inoculate prepared 0.5 McFarland suspension of *E. coli* ATCC 25922 (a carbapenem-susceptible strain) on Muellar-Hinton agar (MHA) plates. After the incubation, the disk was removed using a sterile inoculating loop; the loop was dragged along the edge of the tube during removal to remove excess liquid, and the disk was placed onto the inoculated MHA plate, which was then incubated in for 24 hours at 37°C. Following the incubation, diameter of the inhibition zone around the disc was measured, a zone diameter of 6-10 mm or presence

of colonies within a 16–18 mm zone was considered to be a positive result, 16–18 mm an indeterminate result, and 19 mm a negative result (Hamed and Hasoon, 2019).Only bacterial isolates that were resistant to 3, 4, 5 or more classes of antibiotics isolated in this research, were regarded as multidrug resistant bacteria isolates and bacterial isolates completely resistant to all classes of antibiotics used in this study were subjected to molecular characterisation.

3.12 Molecular Characterisation of Multidrug Bacteria

3.12.1 DNA extraction

The DNA from the bacterial isolates was extracted using the protocol stated by (Trindade *et al.,* 2007). Single colonies grown on Nutrient broth was transferred to 1.5 ml of liquid medium and was transferred into a shaker for 48 hours at 28^0C. After this period, the culture was centrifuged at 4600 g for 5 minutes. The resulting pellets were resuspended into 520 µl of Tris Ethylene Diamine Tetra acetic Acid (TAE) buffer (10 mMTris-HCl, 1mM EDTA, pH 8.0). Fifteen microliters of 20% Sodium Dodecyl Sulfate (SDS) and 3 µl of Proteinase K (20 mg/ml) was added. The mixture was incubated for 1 hour at 37 ^{0}C, then 100 µl of 5 M NaCl and 80 µL of a 10% Cethyl Trimethyl Ammonium Bromide (CTAB) solution in 0.7 M NaCl was added and mixed. The suspension was incubated for 10 minutes at 65^0C. The suspension was kept on ice for 15 minutes. An equal volume of chloroform: isoamyl alcohol (24:1) was added, followed by incubation on ice for 5 minutes and centrifugation at 7200 g for 20 minutes. The aqueous phase was transferred into a new tube. Isopropanol (1: 0.6) was added and DNA precipitated at -20^0C for 16 hours. DNA was collected by centrifugation at 13000 g for 10 minutes, washed with 500 µl of 70% ethanol, air-dried at room temperature for three hours and finally dissolved in 50 µl of Tris Ethylene Diamine Tetra acetic Acid (TAE) buffer (Frank *et al.,* 2008).

3.12.2 Detection of 16S rRNA gene using polymerase chain reaction (PCR)

The polymerase chain reaction (PCR) cocktail consisted of ten micro litres (10 µl) of 5x GoTaq colourless reaction, 3 µl of 25mM $MgCl_2$, 1 µl of 10 mM of dNTPs mix, 1 µl of 10 pmol each 27F 5'- AGA GTT TGA TCM TGG CTC AG-3' and - 1525R, 5′-AAGGAGGTGATCCAGCC-3′ primers and 0.3units of Taq DNA polymerase (Promega, USA) made up to 42 µl with distilled water 8µl DNA template. The PCR was carried out in a GeneAmp 9700 PCR System Thermalcycler (Applied Biosystem Inc., USA) with a PCR profile consisting of an initial denaturation at 94°C for 5 minutes; followed by 30 cycles which will consist of 94^{o}C for 30s, 50^{o}C for 60s and 72^{o}C for 1 minute 30 seconds; and a final termination at 72^{o}C for 10 minutes, and cooled at 4^{o}C (Wawrik *et al*., 2005; Frank *et al*., 2008).

3.12.3 Agarose gel electrophoresis of PCR products

The integrity of the amplified product of about 1.5 Mb gene fragment was checked on a 1.5% Agarose gel to confirm the amplification. The buffer (1XTAE buffer) was prepared and subsequently used to prepare 1.5% agarose gel. The suspension was heated in a microwave for 5 minutes. The agarose gel was allowed to cool to 45^{o}C and stained with 3µl of 0.5 g/ml ethidium bromide. Gel comb was inserted into the slots of the casting tray and the agarose gel was poured into the tray. The gel was allowed to set for 20 minutes. The TAE buffer was poured into the gel tank to barely submerge the gel and the comb removed. Two microlitres (2µl) of 10X loading dye (which gives colour and density to the samples to make it easy to load into the wells and monitor the progress of the gel) was added to 10 µl of each of the PCR product and loaded into the wells. The 100 bp DNA ladder will be loaded into well 1. The gel was electrophoresed at 120 V for 45 minutes visualized by ultraviolet trans-illumination and photographed and the result was recorded (Frank *et al*., 2008). The sizes of the PCR products were

estimated by comparison with the mobility of a 100 bp molecular weight ladder that was ran alongside experimental samples in the gel.

3.12.4 Purification of amplified product

The amplified fragments were purified using ethanol in order to remove the PCR reagents. Briefly, 7.6 µl of sodium acetate 3M and 240 µl of 95% ethanol was added to 40µl PCR amplified product in a new sterile 1.5 µl Eppendorff tube and vortexed. The mixture of PCR product, ethanol and sodium acetate was kept at -20^{o}C for at least 30 minutes. It was centrifuged at 4^{o}C for 10 minutes at 13000 g then followed by removal of supernatant. This was followed by adding 150 µl of 70% ethanol to the pellets. Again, it was centrifuged at 4^{o}C for 15 minutes at 7500 g. The supernatants were removed and tube inverted on paper tissue and allowed to dry in the fume hood at room temperature (28^{o}C) for 15 minutes, then resuspended with 20 µl of sterile distilled water and kept at -20^{o}C prior to sequencing (Trindade *et al*., 2007).

3.12.5 Sequencing and blast

The purified amplified fragments were sequenced using a Genetic Analyzer 3130xl sequencer from Applied Biosystems using manufacturers' manual, while the sequencing kit used was that of Big Dye terminator v3.1 cycle sequencing kit. Bio- Edit software and MEGA 6 was used for all genetic analysis (Frank *et al*., 2008).

Each sequenced gene was uploaded in national center for biotechnology information (NCBI) - basic local alignment search tool (BLAST) (that the NCBI-gene bank) for sequence identification (Frank *et al*., 2008).

3.13 Molecular Detection of Specific ESBL and Other Multidrug Resistant Genes

Identified bacterial species was screened for ESBL-specific and other multidrug resistant genes. Samples were identified first via sequencing and blast of the 16S

ribosomal RNA. Molecular investigations on multidrug resistant and ESBL-coding gene in the DNA of identified bacterial strains was achieved through the use of region specific primers (such as β-lactamase genes (*blaTEM, blaSHV, blaCTX-M, blaCTX-M-1* group and *blaCTX-M-2*), flouroquinolone resistance-associated genes (*gyrA and ParC* and aminoglycoside resistance-associated genes (*aacC1* and *aacC2*). Reaction cocktail used for all PCR per primer set included (Reagent Volume µl) - 5X PCR SYBR green buffer (2.5), MgCl2 (0.75), 10pM DNTP (0.25), 10pM of each forward and reverse primer (0.25), 8000U of taq DNA polymerase (0.06) and made up to 10.5 with sterile distilled water to which 2µl template was added. Buffer control was added to eliminate any probability of false amplification. PCR was carried out in a GeneAmp 9700 PCR System Thermalcycler (Applied Biosystem Inc., USA) using the appropriate profile as designed for each primer pair (Frank *et al*., 2008).

3.14 Molecular Amplification of Resistant Genes in the Selected Multi Drug Resistant (MDR) Bacteria

The genetic amplification of various resistant genes such as β-lactamase genes (*blaTEM, blaSHV, blaCTX-M, blaCTX-M-1, blaCTX-M-2*and *OXA-48*), flouroquinolone resistance-associated genes (*gyrA and ParC*) and aminoglycoside resistance-associated genes (*aacC1* and *aacC2*) is shown in Table 3.1

Table 3.1: Primers used in this study

Genes	Oligosequences (3'-5')	Product size (bp)	Reference
β-lactamases resistance-associated gene			
blaTEM	F: TTTCGTGTCGCCCTTATTCC R:ATCGTTGTCAGAAGTAAGTTGG	237	Yuan *et al* 1998
blaCTX-M	F: CGCTTTGCGATGTGCAG R: ACCGGCGATATCGTTGGT	470	Yuan *et al* 1998
blaCTX-M-1	F: GACGATGTCACTGGCTGAGC R: AGCCGCCGACGCTAATACA	500	Pitout *et al* 2004
blaCTX-M-2	F: GCGACCTGGTTAACTACAATCC R: CGGTAGTATTGCCCTTAAGCC	351	,,
blaSHV	F: TCAGCG AAAAACACCTTG R: TCCCGCAGATAAATCACCA	470	Yuan *et al* 1998
blaOXA-48	F: AACGGGCGAACCAAGCATTTT R:TGAGCACTTCTTTTGTGATGGCT	590	Mir *et al* 2016
Aminoglycosides resistance-associated gene			
aacC1	F:ACCTACTCCCAACATCAGCC R: ATATAGATCTCACTACGCGC	169	Mir *et al* 2016
aacC2	F:ACTGTGATGGGATACGCGTC R: CTCCGTCAGCGTTTCAGCTA	400	,,
Fluoroquinolones resistance-associated genes			
gyrA	F: GTGTGCTTTATGCCATGAG R :GGTTTCCTTTTCCAGGTC	550	O'Regan *et al* 2009
ParC	F: CATCGTCTACGCCATGAG R: AGCAGCACCTCGGAATAG	420	,,

3.15 Phylogenetic tree showing the evolutionary relationship

Sequence analysis of DNA samples was carried out to confirm the genotype of various multidrug resistant (MDR) bacteria. The Finch TV (version 1.4.0) software was used to edit chromatograhs. All nucleotide sequences obtained were screened using the online BLAST (Basic Local Alignment Search Tool) http://blast.ncbi.nlm.nih.gov/Blast.cgi to search for similarity between sequences and previously reported sequenced in the

database that are closely related. The following are the accession numbers of different MDR bacteria reference sequence used in this study;

Table 3.2: Multi Drug Resistant Bacteria Reference Sequence

Accession numbers of different multi drug resistant bacteria reference sequence
CP054379.1, CP054556.1, CP046429.1, CP052767.1, CP046429.1, CP052295.1, KU936064.1

The evolutionary history was inferred using the Minimum Evolution method (Rzhetsky and Nei, 1992). The optimal tree with the sum of branch length = 0.28713192 is shown. The tree is drawn to scale, with branch lengths in the same units as those of the evolutionary distances used to infer the phylogenetic tree. The evolutionary distances were computed using the Maximum Composite Likelihood method (Tamura *et al.,* 2004) and are in the units of the number of base substitutions per site. The ME tree was searched using the Close-Neighbour-Interchange (CNI) algorithm (Nei and Kumar, 2000) at a search level of 1. The Neighbour-joining algorithm (Saitou and Nei, 1987) was used to generate the initial tree. This analysis involved 22 nucleotide sequences. Codon positions included were 1st+2nd+3rd+Noncoding. All ambiguous positions were removed for each sequence pair (pairwise deletion option). There were a total of 1554 positions in the final dataset. Evolutionary analyses were conducted in MEGA X (Kumar *et al.,* 2018).

3.14 Data Analysis

The data obtained was analyzed using package for Statistical Analysis System (SAS) version 9.4 and Chi-square test was used to determine the significant differences between the data obtained**.**

CHAPTER FOUR

4.0 RESULTS AND DISCUSSION

4.1 Results

Out of 1170 endocervical swabs and urine samples screened, only 720(62%) samples revealed the presence of bacteria (Table 4.1).

Table 4.1: Prevalence of Bacterial Infection in Nine General Hospitals

Samples	NSS	NPS	Prevalence (%)
Endocervical swab	1170	320	27.4
Urine	1170	400	34.2
Total	1170	720	62

Key: PID= Pelvic inflammatory disease; NSS=Number of samples screened; NPS= Number of positive samples

4.1.1 Frequency of occurrence of various bacteria

The bacteria isolated and identified are indicated in Table 4.2. It was observed that *E.coli* was more prominent followed by *Klebsiella pneumionae* in both endocervical swabs (ECS) and urine samples (Table 4.2).

Table 4.2: Frequency of Occurrence of Various Bacteria in Nine General Hospitals

	ECS		URINE		TOTAL	
	Number of isolates	Frequency of occurrence (%)	Number of isolates	Frequency of occurrence (%)	Number of isolates	Frequency of occurrence (%)
Klebsiella pneumoniae	70	21.9	85	21.2	155	21.5
Escherichia coli	72	22.5	98	24.5	170	23.6
Salmonella Typhi	69	21.6	83	20.8	152	21.1
*Proteus vulg*aris	39	12.2	54	13.5	93	12.9
Staphylococcus aureus	38	11.8	42	10.5	80	11.1
Streptococcus pyogenes	32	10	38	9.5	70	9.7
Total	320	100	400	100	720	100

Key: ECS=Endocervical swab

4.1.2 Factors associated with the rate of infection

The distribution of bacterial infection among patients according to location is shown in Table 4.3. Women in the rural areas had a higher prevalence of 32.5% than women in the urban areas.

The distribution of bacterial infection according to age is shown in Table 4.4. It was observed that patients within the ages of 25-29 years recorded more rate of infection (24.4%) while the least rate of infection (0.3%) was recorded among patients that were greater than 50 years of age (Table 4.4).

Table 4.3: Distribution of Bacterial Infection Among Patients According to Location of Residence

Settlement	NSS	NPS	Prevalence (%)	χ2(p-value)
Urban	723	340	29.1	
Rural	447	380	32.5	168.387(0.035)
Total	1170	720	62	

χ2=168.387, P=0.035, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples;
SD= Significant difference.

Table 4.4: Distribution of Bacterial Infection in Relation to Age of Patients

Age (years)	NSS	NPS	Prevalence (%)	χ^2(p-value)
<15	79	18	1.5	
15-19	187	118	10.1	
20-24	183	119	10.2	
25-29	330	285	24.4	
30-34	159	102	8.7	
35-39	96	40	3.4	212.396(0.012)
40-44	75	17	1.5	
45-49	43	18	1.5	
>50	18	3	0.3	
Total	1170	720	62	

χ2=212.396, P=0.012, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples;
SD= Significant difference.

The results in Table 4.5 and 4.6 revealed the distribution of bacterial infection according to history of pelvic inflammatory disease (PID) and sexually transmitted infection (STI). Patients with no history of PID (52%) and STI (47%) had highest prevalence of the bacterial infection compared to those that had history of PID (10%) and STI (15%).

The distribution of bacterial infection among patients based on history of abortion revealed higher prevalence among patients with no history of abortion (43.4%) while patients with history of abortion had a lower prevalence (18.1%) as seen in Table 4.7.

The result in Table 4.8 shows the distribution of bacterial infection according to history of urinary tract infection (UTI). Higher prevalence of bacterial infection was observed among patients with previous history of UTI (49.4%) while lower prevalence was observed among patients with no history UTI.

Table 4.5: Distribution of Bacterial Infection Among Patients According to History of PID

Previous PID	NSS	NPS	Prevalence (%)	χ^2(p-value)
Yes	478	115	9.8	
No	692	605	51.7	498.464(0.024)
Total	1170	720	62	

$\chi 2$=498.464, P=0.024, SD(P<0.05); X^2=335.084, P=0.00, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples;
SD= Significant difference.

Table 4.6: Distribution of Bacterial Infection Among Patients According to History of Sexually Transmitted Infections

Previous STI	NSS	NPS	Prevalence (%)	χ^2(p-value)
Yes	529	176	15.0	
No	641	544	46.5	335.084(0.015)
Total	1170	720	62	

$\chi 2$=335.084, P=0.015, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples;
SD= Significant difference.

Table 4.7: Distribution of Bacterial Infection Among Patients According to History of Abortion

Previous Abortion	NSS	NPS	Prevalence (%)	χ^2(p-value)
Yes	544	212	18.1	
No	626	508	43.4	233.163(0.031)
Total	1170	720	62	

$\chi 2$=233.163, P=0.031, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples; SD= Significant difference.

Table 4.8: Distribution of Bacterial Infection Among Patients According to History of Urinary Tract Infection

Previous UTI	NSS	NPS	Prevalence (%)	χ^2(p-value)
Yes	868	578	49.4	
No	302	142	12.1	157.706(0.025)
Total	1170	720	62	

$\chi 2$=157.706, P=0.025, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples; SD= Significant difference.

The distribution of bacterial infection among patients based on marital status revealed that married patients recorded higher prevalence of (51.5%) while single patients with (10.1%) as seen in Table 4.9.

The result in Table 4.10 shows the distribution of bacterial infection according to family status. Higher rate of infection was observed in patients who practiced polygamy (40.3%); followed by patients who practiced monogamy (11.1%) and the least rate of infection (10.1%) was recorded for patients who were unmarried.

Table 4.9: Distribution of Bacterial Infection Among Patients Based on Marital Status

Marital Status	NSS	NPS	Prevalence (%)	χ^2(p-value)
Single	492	118	10.1	
Married	678	602	51.5	212.151(0.013)
Total	1170	720	62	

$\chi 2$=212.151, P=0.013, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples; SD= Significant difference.

Table 4.10: Distribution of Bacterial Infection Among Patients According to Family Status

Family Status	NSS	NPS	Prevalence (%)	χ^2(p-value)
Unmarried	492	118	10.1	
Monogamy	190	130	11.1	161.929(0.019)
Polygamy	488	472	40.3	
Total	1170	720	62	

$\chi 2$=161.929, P=0.019, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples; SD= Significant difference.

The result in Table 4.11 shows the distribution of bacterial infection according to occupation, with unemployed having the highest prevalence (32.8%), followed by students with (19.0%) and the least prevalence was recorded among the employed patients (9.7%).

The distribution of bacterial infection on education status revealed that patients with uneducated status had the highest rate of infection (29.3%) while patients with tertiary educational status had the least rate of infection (4.5%) as seen in Table 4.12

Table 4.11: Distribution of Bacterial Infection Among Patients According to Occupation

Occupation	NSS	NPS	Prevalence (%)	χ^2(p-value)
Unemployed	448	384	32.8	
Student	350	222	19.0	254.454(0.023)
Employed	372	114	9.7	
Total	1170	720	62	

$\chi 2$=254.454, P=0.023, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples; SD= Significant difference.

Table 4.12: Distribution of Bacterial Infection among Patients According to Education

Education	NSS	NPS	Prevalence (%)	χ^2(p-value)
Uneducated	411	343	29.3	
Primary education	325	216	18.5	
Secondary education	244	108	9.2	206.041(0.017)
Tertiary education	190	53	4.5	
Total	1170	720	62	

$\chi 2$=206.041, P=0.017, SD(P<0.05)
Key: NSS=Number of samples screened; NPS= Number of positive samples; SD= Significant difference.

The result in Table 4.13 shows the distribution of bacterial infection according to douching frequency, with patients who practiced douching daily having the highest rate of infection of (44.4%) and those who practiced douching weekly having the least rate of infection of (17.2%).

The distribution of bacterial infection among patients based on douching products used revealed that patients who used water and soap recorded highest prevalence of (31.0%), followed by those who used only water, (16.5%) and the least prevalence was recorded among those who used other products (14.1%) for douching as seen in Table 4.14.

The result in Table 4.15 shows the distribution of bacterial infection according to water source. Patients who used river water had the highest rate of bacterial infection (27.7%) while patients who used borehole water had the lowest rate of bacterial infection (4.1%).

Table 4.13: Distribution of Bacterial Infection Among Patients Based on Douching Frequency

Douching Frequency	NSS	NPS	Prevalence (%)	χ^2(p-value)
Daily	588	519	44.4	
Weekly	534	201	17.2	
Monthly	37	0	0	
Yearly	11	0	0	383.112(0.021)
Not Practiced	0	0	0	
Total	1170	720	62	

χ2=383.112, P=0.021, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples; SD= Significant difference.

Table 4.14: Distribution of Bacterial Infection Among Patients Based on Douching Products

Douching Products	NSS	NPS	Prevalence (%)	χ^2(p-value)
Water Only	336	194	16.5	
Water and Toilet Soap	497	360	30.8	
Others	337	166	14.2	48.453(0.001)
Total	1170	720	62	

χ2=48.453, P=0.001, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples; SD= Significant difference.

Table 4.15: Distribution of Bacterial Infection Among Patients Based on Water Source

Water Source	NSS	NPS	Prevalence (%)	χ^2(p-value)
Borehole	216	48	4.1	
Tap	282	161	13.8	
Well	261	187	16.0	183.568(0.003)
River	411	324	27.7	
Total	1170	720	62	

χ2=183.568, P=0.003, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples; SD= Significant difference.

The result in Table 4.16 shows the distribution of bacterial infection according to various types of toilet facilities. Patients who defecate in the open environment recorded highest rate of infection (38.7%) and those who used water closet recorded the least rate of bacterial infection (7.4%).

Table 4.16: Distribution of Bacterial Infection Among Patients According to Toilet Facilities

Toilet Facility	NSS	NPS	Prevalence (%)	χ^2(p-value)
Water Closet	320	87	7.4	
Pit Latrine	331	180	15.4	280.270(0.004)
Open Environment	519	453	38.7	
Total	1170	720	62	

χ2=280.270, P=0.004, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples; SD= Significant difference.

The distribution of bacterial infection among patients based on birth control revealed patients who practiced no birth control recorded higher rate of bacterial infection (37.9%) while patients who practiced birth control recorded lower rate of bacterial infection (23.7%) as seen in Table 4.17.

The result in Table 4.18 shows the distribution of bacterial infection based on the type of birth control. Patients who used no birth control recorded the highest rate of bacterial infection (37.9%) while those who used pills recorded the least rate of bacterial infection (4.4%).

Table 4.17: Distribution of Bacterial Infection Among Patients According to Birth Control

Practice of Birth Control	NSS	NPS	Prevalence (%)	χ^2(p-value)
Yes	523	277	23.7	
No	647	443	37.9	28.986(0.002)
Total	1170	720	62	

χ2=28.986, P=0.002, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples; SD= Significant difference.

Table 4.18: Distribution of Bacterial Infection Among Patients According to Type of Birth Control

Types of Birth Control	NSS	NPS	Prevalence (%)	χ^2(p-value)
Intrauterine device	255	131	11.2	
Pills	131	51	4.4	
Condoms	137	95	8.1	150.603(0.015)
No type used	647	443	37.9	
Total	1170	720	62	

χ2=150.603, P=0.015, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples;

SD= Significant difference.

The result in Table 4.19 shows the distribution of bacterial infection based on self medication. Patients who practiced self medication had the highest prevalence (48.0) and those who do not practice self medication recorded the lowest prevalence (14.0%).

Table 4.19: Distribution of Bacterial Infection Among Patients Based on Self Medication

Self-Medication Practice	NSS	NPS	Prevalence (%)	χ^2(p-value)
Yes	625	556	47.5	
No	545	164	14.0	426.265(0.001)
Total	1170	720	62	

χ2=426.265, P=0.001, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples;

SD= Significant difference.

The result in Table 4.20 shows the distribution of bacterial infection based on drug dosage with patients who practiced incomplete drug dosage recording highest rate of bacterial infection (42.3%) and those who practiced complete drug dosage recording lowest rate of infection (19.2%).

Table 4.20: Distribution of Bacterial Infection Among Patients Based on Drug Dosage

Drug Dosage Practice	NSS	NPS	Prevalence (%)	χ^2(p-value)
Complete Dosage	601	225	19.2	
Incomplete Dosage	569	495	42.3	303.277(0.025)
Total	1170	720	62	

χ2=303.277, P=0.025, SD(P<0.05)

Key: NSS=Number of samples screened; NPS= Number of positive samples; SD= Significant difference.

4.1.3 Frequency of occurrence of various multidrug resistant bacteria in various samples of patients with pelvic inflammatory disease

A total of 228 (31.7%) multidrug resistant bacteria consisting of 90 (28.1%) and 138 (34.5%) were isolated from endocervical and urine samples respectively (as seen in Table 4.21). Tables 4.22, 4.23 and 4.24 show the percentage of occurrence of various bacterial isolates from endocervical swabs, urine samples and both samples respectively.

Table 4.21: Percentage Occurrence of Total Multi Drug Resistant (MDR) Bacteria in Various Samples of Patients with PID in Nine General Hospitals

Sample	Number of isolates	Total MDR Isolates	Total Non-MDR Isolates	Total Percentage
Endocervical swab	320	90	230	
% occurrence		28.1	71.9	100
Urine	400	138	262	
% occurrence		34.5	65.5	100
Total	720	228	492	
% occurrence		31.7	68.3	100

Table 4.22: Frequency of Occurrence of Various Multi Drug Resistant (MDR) Bacteria in Endocervical Swab of Patients with PID in Nine General Hospitals

	ECS			
Organisms	**Number of isolates**	**Number of MDR isolates**	**Number of Non-MDR isolates**	**Total percentage**
Klebsiella pneumoniae	70	35	35	
% Freq.		50	50	100
Escherichia coli	72	31	41	
% Freq.		43.1	56.9	100
Salmonella Typhi	69	24	45	
% Freq.		34.8	65.2	100
Proteus vulgaris	39	0	39	
% Freq.		0	100	100
Staphylococcus aureus	38	0	38	
% Freq.		0	100	100
Streptococcus pyogenes	32	0	32	
% Freq.		0	100	100
Total	320	90	230	
% Freq.		28.1	71.9	100

Table 4.23: Frequency of Occurrence of Various Multi Drug Resistant (MDR) Bacteria in Urine of Patients with PID in Nine General Hospitals

	Urine			
Organisms	**Number of isolates**	**Number of MDR isolates**	**Number of Non-MDR isolates**	**Total percentage**
Klebsiella pneumoniae	85	52	33	
% Freq.		61.2	38.8	100
Escherichia coli	98	48	50	
% Freq.		49.0	51.0	100
Salmonella Typhi	83	33	50	
% Freq.		39.8	60.2	100
Proteus vulgaris	54	5	49	
% Freq.		9.3	90.7	100
Staphylococcus aureus	42	0	42	
% Freq.		0	100	100
Streptococcus pyogenes	38	0	38	
% Freq.		0	100	100
Total	400	138	262	
% Freq.		34.5	65.5	100

Table 4.24: Frequency of Occurrence of Various Multi Drug Resistant (MDR) Bacteria in Both ECS and Urine of Patients with PID in Nine General Hospitals

ECS and Urine				
Organisms	**Number of isolates**	**Number of MDR isolates**	**Number of Non-MDR isolates**	**Total percentage**
Klebsiella pneumoniae	155	87	68	
% Freq.		56.1	43.9	100
Escherichia coli	170	79	91	
% Freq.		46.5	53.5	100
Salmonella Typhi	152	57	95	
% Freq.		37.5	62.5	100
Proteus vulgaris	93	5	88	
% Freq.		5.4	94.6	100
Staphylococcus aureus	80	0	80	
% Freq.		0	100	100
Streptococcus pyogenes	70	0	70	
% Freq.		0	100	100
Total	720	228	492	
% Freq.		31.7	68.3	100

4.1.4 Antibiotic susceptibility test of all multidrug resistant bacteria from both endocervical swabs and urine samples

Table 4.25 to 4.42 show antibiotics susceptibility tests of all multi drug resistant bacterial isolates from both endocervical swabs (ECS) and urine of patients attending nine general hospitals as seen below.

Table 4.25: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from ECS of Patients in General Hospital Nasko

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
2	$13.67\pm0.33^{c}_{b}$	$0.33\pm0.33^{a}_{a}$	$19.67\pm0.88^{d}_{c}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$19.00\pm0.58^{f}_{c}$	$24.67\pm0.90^{f}_{d}$	$19.00\pm1.15^{e}_{c}$	$22.67\pm0.80^{c}_{d}$	$23.00\pm0.57^{d}_{d}$
8	$0.00\pm0.00^{a}_{a}$	$11.00\pm0.58^{b}_{c}$	$23.00\pm0.60^{e}_{e}$	$11.00\pm0.50^{c}_{c}$	$10.00\pm0.59^{d}_{c}$	$8.00\pm0.56^{c}_{b}$	$19.00\pm0.50^{e}_{d}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$
12	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.33\pm0.33^{a}_{a}$	$0.33\pm0.30^{a}_{a}$	$0.33\pm0.31^{a}_{a}$	$0.33\pm0.35^{a}_{a}$	$0.33\pm0.32^{a}_{a}$	$0.33\pm0.36^{a}_{a}$
17	$11.00\pm0.57^{b}_{d}$	$0.00\pm0.00^{a}_{a}$	$16.00\pm0.58^{c}_{f}$	$6.00\pm0.60^{b}_{b}$	$8.33\pm0.33^{c}_{c}$	$13.00\pm059^{de}_{e}$	$0.00\pm0.00^{a}_{a}$	$11.00\pm0.50^{c}_{d}$	$16.00\pm0.59^{b}_{f}$	$0.00\pm0.00^{a}_{a}$
18	$14.00\pm0.57^{c}_{d}$	$0.33\pm0.33^{a}_{a}$	$0.33\pm0.31^{a}_{a}$	$21.00\pm0.50^{e}_{f}$	$19.00\pm0.59^{e}_{e}$	$4.67\pm0.30^{b}_{b}$	$8.33\pm0.33^{b}_{c}$	$4.33\pm0.35^{b}_{b}$	$0.33\pm031^{a}_{a}$	$0.33\pm0.33^{a}_{a}$
19	$13.00\pm0.57^{c}_{c}$	$0.00\pm0.00^{a}_{a}$	$7.33\pm0.33^{b}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$12.00\pm0.58^{d}_{c}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$12.00\pm0.50^{b}_{c}$
20	$14.00\pm057^{c}_{c}$	$0.33\pm0.33^{a}_{a}$	$0.33\pm0.306^{a}_{a}$	$13.66\pm0.3^{d}_{c}$	$5.33\pm0.33^{b}_{b}$	$14.00\pm0.58^{e}_{c}$	$17.00\pm0.50^{d}_{d}$	$17.00\pm0.59^{d}_{d}$	$0.33\pm0.37^{a}_{a}$	$20.00\pm0.60^{c}_{e}$
22	$21.00\pm0.58^{d}_{d}$	$0..33\pm0.33^{a}_{a}$	$18.66\pm0.67^{d}_{c}$	$0.33\pm0.30^{a}_{a}$	$18.33\pm0.88^{e}_{c}$	$0.67\pm0.67^{a}_{a}$	$15.00\pm0.57^{c}_{b}$	$0.33\pm0.30^{a}_{a}$	$0.33\pm0.31^{a}_{a}$	$20.00\pm0.60^{c}_{cd}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.26: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from ECS of Patients in General Hospital Kontagora

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
1	$21.00\pm1.00^{c}_{de}$	$17.00\pm1.05^{c}_{c}$	$25.00\pm1.73^{d}_{f}$	$0.00\pm0.00^{a}_{a}$	$19.00\pm1.70^{e}_{c}$	$0.00\pm0.00^{a}_{a}$	$24.00\pm1.20^{e}_{ef}$	$0.00\pm0.00^{a}_{a}$	$16.00\pm0.60^{c}_{c}$	$12.00\pm1.48^{b}_{b}$
2	$20.00\pm1.22^{c}_{e}$	$17.00\pm0.62^{c}_{de}$	$17.00\pm1.16^{b}_{de}$	$11.00\pm1.09^{c}_{b}$	$12.00\pm1.22^{d}_{bcd}$	$16.00\pm1.15^{d}_{cd}$	$5.00\pm1.00^{b}_{a}$	$6.00\pm0.71^{b}_{a}$	$13.00\pm1.20^{b}_{bc}$	$16.00\pm0.58^{c}_{cd}$
13	$20.00\pm0.60^{c}_{cd}$	$7.00\pm0.58^{b}_{a}$	$20.00\pm00^{c}_{cd}$	$7.00\pm1.16^{b}_{a}$	$18.00\pm0.60^{e}_{c}$	$13.00\pm1.20^{c}_{b}$	$23.00\pm1.16^{e}_{de}$	$22.00\pm0.59^{e}_{d}$	$25.00\pm1.10^{d}_{e}$	$18.00\pm1.17^{c}_{c}$
15	$24.00\pm0.58^{d}_{c}$	$18.00\pm1.20^{cd}_{b}$	$24.00\pm0.61^{d}_{c}$	$16.00\pm1.73^{d}_{b}$	$9.00\pm1.16^{bc}_{a}$	$11.00\pm0.58^{bc}_{a}$	$10.0\pm0.59^{c}_{a}$	$11.00\pm1.20^{c}_{a}$	$10.00\pm1.70^{b}_{a}$	$17.00\pm1.11^{c}_{b}$
27	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
31	$24.00\pm0.58^{d}_{e}$	$20.00\pm1.10^{d}_{d}$	$24.00\pm1.20^{d}_{e}$	$13.00\pm1.15^{cd}_{bc}$	$12.00\pm1.20^{cd}_{b}$	$16.00\pm1.16^{d}_{c}$	$8.00\pm1.16^{c}_{a}$	$13.00\pm1.70^{e}_{d}$	$12.00\pm1.15^{b}_{b}$	$29.00\pm1.10^{d}_{f}$
39	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
42	$12.00\pm1.15^{b}_{d}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$8.00\pm1.12^{bc}_{ab}$	$6.00\pm1.00^{b}_{b}$	$10.00\pm1.16^{b}_{cd}$	$15.00\pm1.15^{d}_{e}$	$15.00\pm1.14^{d}_{e}$	$0.00\pm0.00^{a}_{a}$	$28.00\pm1.30^{d}_{f}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.27: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from ECS of Patients in General Hospital Agaie

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
1	$13.00\pm0.58^{ab}{}_{c}$	$21.00\pm0.58^{c}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$9.00\pm1.16^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
3	$20.00\pm1.16^{c}{}_{d}$	$10.00\pm1.15^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm1.20^{c}{}_{b}$	$8.00\pm1.10^{c}{}_{b}$	$10.00\pm1.15^{d}{}_{b}$	$16.00\pm1.15^{de}{}_{c}$	$19.00\pm1.15^{e}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$7.00\pm0.60^{b}{}_{b}$
4	$13.00\pm0.57^{ab}{}_{d}$	$20.00\pm0.50^{c}{}_{g}$	$17.00\pm0.60^{e}{}_{f}$	$4.00\pm0.58^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm1.20^{d}{}_{c}$	$15.00\pm1.16^{cde}{}_{e}$	$22.00\pm0.58^{f}{}_{h}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
5	$15.00\pm1.5^{b}{}_{e}$	$0.00\pm0.00^{a}{}_{a}$	$2.00\pm0.58^{b}{}_{ab}$	$0.00\pm0.00^{a}{}_{a}$	$4.00\pm.60^{b}{}_{bc}$	$5.00\pm0.58^{b}{}_{c}$	$8.00\pm0.60^{b}{}_{d}$	$10.00\pm0.50^{b}{}_{d}$	$3.00\pm1.12^{b}{}_{b}$	$15.00\pm0.60^{c}{}_{e}$
7	$20.00\pm0.60^{c}{}_{f}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm1.15^{c}{}_{c}$	$0.00\pm0.00^{a}{}_{b}$	$11.00\pm0.57^{d}{}_{c}$	$16.00\pm0.58^{c}{}_{d}$	$18.00\pm0.60^{ef}{}_{e}$	$15.00\pm0.60^{d}{}_{d}$	$8.00\pm0.50^{c}{}_{b}$	$19.00\pm0.57^{d}{}_{ef}$
10	13.00 ± 0.61^{ab}	0.00 ± 0.00^{a}	10.00 ± 0.50^{c}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	16.00 ± 0.55^{c}
14	$11.00\pm0.61^{a}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$12.00\pm0.64^{d}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$11.00\pm0.58^{d}{}_{a}$	$7.00\pm0.51^{e}{}_{a}$	$12.00\pm1.16^{c}{}_{a}$	$12.00\pm0.63^{c}{}_{a}$	$10.00\pm0.58^{d}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
19	$25.00\pm0.66^{d}{}_{cd}$	$28.00\pm0.58^{c}{}_{a}$	$13.00\pm0.60^{d}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{cd}$	$0.00\pm0.00^{a}{}_{b}$	$20.00\pm1.13^{f}{}_{d}$	$0.00\pm0.00^{a}{}_{d}$	$0.00\pm0.00^{a}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$
20	$25.00\pm0.59^{d}{}_{d}$	$33.00\pm0.62^{d}{}_{e}$	$0.00\pm0.00^{a}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$13.00\pm1.21^{cd}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.28: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from ECS of Patients in General Hospital Suleja

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
1	$5.00\pm0.57^{b}_{b}$	$25.00\pm0.58^{d}_{f}$	$20.00\pm0.50^{cd}_{e}$	$5.00\pm1.15^{b}_{b}$	$12.00\pm0.56^{d}_{d}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$10.00\pm0.60^{c}_{c}$
2	$0.00\pm0.00^{a}_{a}$	$29.00\pm0.57^{e}_{e}$	$18.00\pm0.58^{c}_{d}$	$0.00\pm0.00^{a}_{a}$	$11.00\pm0.50^{cd}_{bc}$	$10.00\pm0.60^{c}_{b}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$12.00\pm0.59^{c}_{c}$	$0.00\pm0.00^{a}_{a}$
3	$0.00.\pm0.00^{a}_{a}$	$20.00\pm0.57^{c}_{d}$	$19.00\pm0.58^{cd}_{d}$	$6.00\pm1.15^{bc}_{b}$	$10.00\pm0.59^{c}_{c}$	$0.00\pm0.00^{a}_{a}$	$9.00\pm0.570^{c}_{c}$	$5.00\pm0.60^{b}_{b}$	$5.00\pm01.20^{b}_{b}$	$0.00\pm0.00^{a}_{a}$
4	$0.00\pm0.00^{a}_{a}$	$15.00\pm0.50^{b}_{d}$	$0.00\pm0.00^{a}_{a}$	$8.00\pm0.57^{c}_{b}$	$0.00\pm0.00^{a}_{a}$	$0..00\pm0.00^{a}_{a}$	$10.00\pm0.58^{c}_{c}$	$0..00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$
8	$0.00\pm0.00^{a}_{a}$	$22.00\pm1.15^{c}_{e}$	$21.00\pm1.20^{d}_{e}$	$7.00\pm1.10^{bc}_{bc}$	$0.00\pm0.00^{a}_{a}$	$5.00\pm0.00^{b}_{b}$	$10.00\pm0.58^{c}_{d}$	$8.00\pm0.60^{c}_{cd}$	$0.00\pm0.00^{a}_{a}$	$7.00\pm0.50^{bb}_{bc}$
9	$0.00\pm0.00^{a}_{a}$	$22.00\pm0.60^{c}_{e}$	$12.00\pm1.15^{b}_{d}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$5.00\pm0.00^{b}_{b}$	$5.00\pm0.58^{b}_{b}$	$0.00\pm0.00^{a}_{a}$	$6.00\pm0.59^{b}_{b}$	$8.00\pm0.50_{c}$
11	$0.00\pm0.00^{a}_{a}$	$13.00\pm0.57^{ab}_{d}$	$10.00\pm0.60^{b}_{c}$	$12.00\pm1.20^{d}_{cd}$	$0.00\pm0.00^{a}_{a}$	$6.00\pm1.10^{b}_{b}$	$5.00\pm1.16^{b}_{b}$	$5.00\pm0.50^{b}_{b}$	$6.00\pm1.00^{b}_{b}$	$7.00\pm0.59^{b}_{b}$
12	$0.00\pm0.00^{a}_{a}$	$12.00\pm0.57^{a}_{e}$	$10.00\pm1.10^{b}_{cde}$	$12.00\pm0.58^{d}_{e}$	$8.00\pm1.15^{b}_{bc}$	$11.00\pm1.00^{c}_{de}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$9.00\pm1.20^{c}_{bcd}$	$7.00\pm1.16^{b}_{b}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.29: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from ECS of Patients in General Hospital Kuta

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
2	$22.00\pm1.16^{c}_{d}$	$13.00\pm0.57^{a}_{c}$	$22.00\pm0.58^{de}_{d}$	$15.00\pm0.54^{c}_{c}$	$5.00\pm1.16^{b}_{a}$	$9.00\pm0.58^{c}_{b}$	$20.0\pm1.160^{f}_{d}$	$6.00\pm1.16^{bc}_{a}$	$5.00\pm1.17^{b}_{a}$	$10.00\pm1.16^{c}_{b}$
3	$0.00\pm0.00^{a}_{a}$	$25.00\pm1.15^{c}_{c}$	$25.00\pm1.10^{e}_{c}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$15.00\pm1.15^{d}_{b}$	$0.00\pm0.00^{a}_{a}$
4	$16.00\pm1.10^{b}_{c}$	$20.00\pm0.50^{b}_{d}$	$14.00\pm0.57^{a}_{c}$	$4.0\pm0.54^{b}_{b}$	$14.00\pm0.58^{d}_{c}$	$15.00\pm0.57^{d}_{c}$	$6.00\pm0.58^{bc}_{a}$	$15.00\pm0.57^{e}_{c}$	$19.66\pm0.88^{e}_{d}$	$9.66\pm0.89^{c}_{b}$
5	$15.00\pm1.15^{b}_{c}$	$14.00\pm0.57^{a}_{c}$	$20.00\pm1.17^{d}_{d}$	$10.00\pm1.17^{b}_{b}$	$10.00\pm1.15^{c}_{b}$	$9.00\pm0.58^{c}_{b}$	$8.00\pm0.57^{cd}_{b}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$
6	$0.00\pm0.00^{a}_{a}$	$15.00\pm0.58^{a}_{d}$	$25.00\pm1.16_{e}$	$17.00\pm1.16^{c}_{d}$	$5.00\pm0.57^{b}_{b}$	$5.00\pm1.17^{b}_{b}$	$10.00\pm1.73^{d}_{c}$	$15.00\pm0.58^{e}_{d}$	$10.00\pm1.74^{c}_{c}$	$15.00\pm1.15^{d}_{d}$
7	$3.00\pm0.56^{b}_{a}$	$25.00\pm0.55^{c}_{g}$	$15.00\pm1.15^{c}_{e}$	$25.00\pm1.17^{d}_{g}$	$13.00\pm1.73^{d}_{de}$	$20.00\pm0.58^{e}_{f}$	$15.00\pm0.57^{e}_{e}$	$7.00\pm1.17^{bc}_{bc}$	$10.00\pm1.18^{c}_{cd}$	$5.00\pm1.16^{b}_{ab}$
8	$2.00\pm0.57^{ab}_{a}$	$20.00\pm1.75^{b}_{e}$	$5.00\pm1.16^{a}_{b}$	$10.00\pm1.18^{b}_{c}$	$3.00\pm056^{b}_{ab}$	$8.00\pm0.57^{c}_{c}$	$5.00\pm0.58^{b}_{b}$	$8.00\pm0.56^{cd}_{c}$	$16.00\pm0.57^{d}_{d}$	$5.00\pm0.54^{b}_{b}$
10	$0.00\pm0.00^{a}_{a}$	$20.00\pm0.57^{b}_{e}$	$20.00\pm058^{d}_{e}$	$15.00\pm0.54^{c}_{d}$	$5.00\pm0.58^{b}_{b}$	$16.00\pm1.17^{d}_{d}$	$5.00\pm0.56^{b}_{b}$	$0.00\pm0.00^{a}_{b}$	$15.00\pm0.57^{d}_{d}$	$8.00\pm0.58^{c}_{c}$
11	$0.00\pm0.00^{a}_{a}$	$15.00\pm1.16^{a}_{de}$	$5.00\pm0.57^{a}_{b}$	$14.00\pm1.17^{c}_{d}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$17.00\pm1.17^{e}_{e}$	$10.00\pm1.18^{d}_{c}$	$20.00\pm1.16^{e}_{f}$	$9.66\pm0.88^{c}_{c}$
12	$0.00\pm0.00^{a}_{a}$	$20.00\pm1.16^{b}_{e}$	$10.00\pm0.56^{b}_{c}$	$0.00\pm0.00^{a}_{b}$	$15.00\pm0.58^{d}_{d}$	$15.00\pm1.16^{d}_{d}$	$9.66\pm0.88^{d}_{c}$	$5.00\pm0.58^{b}_{b}$	$15.00\pm1.18^{d}_{d}$	$10.00\pm1.16^{c}_{c}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.30: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from ECS of Patients in General Hospital Lapai

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
1	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm1.10^{de}{}_{d}$	$20.00\pm1.00^{fg}{}_{e}$	$7.00\pm0.50^{c}{}_{bc}$	$9.00\pm0.57^{c}{}_{c}$	$2.00\pm0.58^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$5.00\pm1.20^{bc}{}_{b}$
2	$12.00\pm0.50^{de}{}_{bc}$	$10.00\pm0.57^{c}{}_{ab}$	$18.00\pm0.58^{ef}{}_{d}$	$10.00\pm0.60^{de}{}_{ab}$	$12.00\pm0.56^{d}{}_{bc}$	$14.00\pm1.00_{ab}$	$10.00\pm1.20^{de}{}_{ab}$	$9.00\pm1.10^{d}{}_{a}$	$14.00\pm0.59^{e}{}_{c}$	$8.00\pm0.60^{de}{}_{a}$
3	$0.00\pm0.00^{a}{}_{a}$	$19.00\pm0.50^{f}{}_{e}$	$16.00\pm0.57^{de}{}_{d}$	$11.00\pm0.58^{e}{}_{c}$	$6.00\pm1.10^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{b}$	$6.00\pm1.20^{bc}{}_{b}$	$5.00\pm1.15^{b}{}_{b}$	$12.00\pm1.16^{de}{}_{c}$	$11.00\pm0.50^{fg}{}_{c}$
4	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm0.50^{c}{}_{cd}$	$8.00\pm0.57^{b}{}_{c}$	$10.00\pm1.15^{de}{}_{cd}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{b}$	$4.00\pm1.10^{b}{}_{b}$	$8.00\pm1.00^{cd}{}_{c}$	$12.00\pm0.59^{de}{}_{d}$	$5.00\pm1.11^{bc}{}_{b}$
5	$0.00\pm0.00^{a}{}_{a}$	$16.00\pm1.15^{e}{}_{d}$	$20.00\pm1.10^{fg}{}_{e}$	$13.00\pm1.00^{f}{}_{cd}$	$12.00\pm1.20^{d}{}_{c}$	$4.00\pm1.16^{b}{}_{cd}$	$14.00\pm1.15^{g}{}_{cd}$	$15.00\pm1.19^{g}{}_{cd}$	$0.00\pm0,00^{a}{}_{a}$	$4.00\pm1.00^{b}{}_{b}$
6	$15.00\pm0,50^{f}{}_{cd}$	$14.00\pm0.57^{de}{}_{c}$	$21.00\pm0.58^{g}{}_{e}$	$10.00\pm0.60^{de}{}_{b}$	$11.00\pm0.59^{cd}{}_{b}$	$15.00\pm50_{b}$	$10.00\pm0.58^{de}{}_{b}$	$10.00\pm1.00^{de}{}_{b}$	$17.00\pm1.10^{f}{}_{d}$	$7.00\pm0.59^{cd}{}_{a}$
7	$13.00\pm0.50^{e}{}_{b}$	$13.00\pm0.57^{d}{}_{b}$	$25.00\pm0.59^{i}{}_{e}$	$16.00\pm0.58^{g}{}_{cd}$	$17.00\pm0.60^{f}{}_{d}$	$10.00\pm0.57^{d}{}_{b}$	$13.00\pm0.54^{fg}{}_{b}$	$14.00\pm1.00^{fg}{}_{bc}$	$13.00\pm1.10^{de}{}_{b}$	$10.00\pm1.20^{efg}{}_{a}$
8	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00	0.00 ± 0.00^{a}
9	$0.00\pm0.00^{a}{}_{a}$	$22.00\pm0.50^{g}{}_{e}$	$17.00\pm0.57^{de}{}_{d}$	$10.00\pm.0.59^{de}{}_{c}$	$10.00\pm0.58^{cd}{}_{c}$	$10.00\pm0.60^{d}{}_{b}$	$4.00\pm1.00^{b}{}_{b}$	$10.00\pm1.20^{de}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm1.10^{h}{}_{d}$
10	$6.00\pm0.50^{b}{}_{a}$	$25.00\pm0.57^{h}{}_{f}$	$20.00\pm0.59^{fg}{}_{e}$	$20.00\pm0.58^{h}{}_{e}$	$10.00\pm0.60^{cd}{}_{c}$	$10.00\pm0.53^{d}{}_{d}$	$12.00\pm0.57^{efg}{}_{d}$	$9.00\pm0.50^{d}{}_{bc}$	$6.00\pm0.60^{b}{}_{a}$	$8.00\pm0.59^{de}{}_{b}$
11	$10.00\pm0.50^{c}{}_{a}$	$26.00\pm0.58^{h}{}_{d}$	$21.00\pm0.59^{g}{}_{c}$	$22.00\pm0.57^{i}{}_{c}$	$22.00\pm0.50^{g}{}_{c}$	$10.00\pm0.60^{d}{}_{a}$	$10.00\pm0.58^{de}{}_{a}$	$12.00\pm0.50^{ef}{}_{b}$	$13.00\pm0.59^{de}{}_{b}$	$12.00\pm0.60^{g}{}_{b}$
12	$5.00\pm0.50^{b}{}_{a}$	$13.00\pm0.57^{d}{}_{d}$	$23.00\pm0.58^{h}{}_{f}$	$16.00\pm0.50^{g}{}_{e}$	10.00 ± 0.59^{cd}	$15.00\pm0.60_{c}$	$11.00\pm0.57^{ef}{}_{c}$	$10.00\pm0.59^{de}{}_{c}$	$13.00\pm0.53^{de}{}_{d}$	$8.00\pm0.50^{de}{}_{b}$
13	0.00 ± 0.00^{a}	16.00 ± 0.50^{e}	16.00 ± 0.60^{de}	4.00 ± 0.58^{b}	10.00 ± 0.59^{cd}	6.00 ± 0.57^{c}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	9.00 ± 0.60^{def}
14	11.00 ± 0.50^{cd}	21.00 ± 0.57^{g}	20.00 ± 0.59^{fg}	11.00 ± 0.57^{e}	21.00 ± 0.60^{g}	15.00 ± 0.58	0.00 ± 0.00^{a}	12.00 ± 0.57^{ef}	16.00 ± 0.50^{f}	10.00 ± 0.60^{efg}
15	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	8.00 ± 0.50^{b}	9.00 ± 0.57^{cde}	11.00 ± 0.58^{cd}	10.00 ± 0.60^{d}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
16	0.00 ± 0.00^{a}	8.67 ± 0.88^{c}	11.00 ± 0.50^{c}	4.00 ± 0.57^{b}	10.00 ± 0.60^{cd}	0.00 ± 0.00^{a}	8.00 ± 0.59^{cd}	6.00 ± 0.50^{bc}	11.33 ± 0.33^{d}	0.00 ± 0.00^{a}
17	0.00 ± 0.00^{a}	16.00 ± 0.50^{e}	15.00 ± 0.57^{d}	9.00 ± 0.60^{cde}	15.00 ± 0.59^{e}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	8.00 ± 0.60^{c}	7.00 ± 0.59^{cd}
18	0.00 ± 0.00^{a}	5.67 ± 0.89^{b}	10.00 ± 0.50^{c}	8.00 ± 0.59^{cd}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	5.00 ± 0.59^{b}	0.00 ± 0.00^{a}	12.00 ± 0.60^{de}	0.00 ± 0.00^{a}

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.31: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from ECS of Patients in General Hospital Bida

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
1	$18.00\pm1.10^{e}_{cd}$	$17.00\pm0.50^{de}_{cd}$	$12.00\pm0.88^{d}_{b}$	$20.00\pm1.00^{f}_{de}$	$18.00\pm1.10^{gh}_{cd}$	$15.00\pm0.57^{c}_{c}$	$8.00\pm1.20^{b}_{b}$	$0.00\pm0.00^{a}_{a}$	$20.00\pm1.15^{hi}_{de}$	$10.00\pm1.16^{c}_{b}$
2	$5.00\pm0.50^{b}_{a}$	$25.00\pm1.00^{h}_{f}$	$25.00\pm0.59^{f}_{f}$	$12.00\pm0.60^{bc}_{bc}$	$12.00\pm1.20^{cd}_{bc}$	$15.00\pm0.58^{c}_{de}$	$15.00\pm0.57^{ef}_{de}$	$17.00\pm0.58^{e}_{e}$	$13.00\pm0.60^{de}_{cd}$	$10.00\pm1.00^{c}_{b}$
3	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
4	$20.00\pm1.00^{e}_{d}$	$22.67\pm0.88^{gh}_{e}$	$24.00\pm0.59^{f}_{e}$	$0.00\pm0.00^{a}_{a}$	$16.00\pm0.60^{fg}_{c}$	$20.00\pm1.10^{de}_{d}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$15.00\pm0.59^{ef}_{c}$	$5.00\pm0.57^{b}_{b}$
5	$25.00\pm00^{f}_{f}$	$21.00\pm0.59^{fg}_{de}$	$25.00\pm0.50^{f}_{f}$	$20.00\pm1.00^{f}_{d}$	$23.00\pm0.59^{i}_{ef}$	$14.00\pm1.10^{c}_{c}$	$24.00\pm1.20^{h}_{f}$	$25.00\pm0.60^{g}_{f}$	$9.00\pm0.57^{b}_{b}$	$0.00\pm0.00^{a}_{a}$
10	$8.00\pm0.50^{c}_{a}$	$17.00\pm0.57^{de}_{ef}$	$11.00\pm0.60^{b}_{b}$	$14.00\pm1.00^{cd}_{cd}$	$12.00\pm1.10^{cd}_{bc}$	$11.00\pm0.60^{b}_{b}$	$15.00\pm0.58^{ef}_{de}$	$16.00\pm0.59^{e}_{def}$	$18.00\pm0.57^{gh}_{f}$	$13.00\pm0.0^{de}_{cd}$
11	$0.00\pm0.00^{ad}_{a}$	$20.00\pm1.00^{f}_{b}$	$19.66\pm0.88^{d}_{c}$	$16.00\pm1.10^{de}_{ab}$	$9.00\pm0.57^{b}_{ab}$	$15.00\pm0.50^{c}_{ab}$	$13.00\pm0.59^{de}_{ab}$	$0.00\pm0.00^{a}_{b}$	$14.00\pm0.58^{def}_{ab}$	$12.00\pm1.20^{cd}_{ab}$
12	$10.00\pm1.10^{c}_{b}$	$3.00\pm0.50^{b}_{a}$	$24.00\pm1.15^{f}_{e}$	$15.00\pm0.58^{d}_{c}$	$12.00\pm0.60^{cd}_{b}$	$16.00\pm0.59^{c}_{c}$	$21.00\pm0.57^{g}_{d}$	$3.00\pm0.53^{b}_{a}$	$10.00\pm1.10^{bc}_{b}$	$19.00\pm1.15^{g}_{d}$
13	$12.00\pm1.00^{d}_{b}$	$0.00\pm0.00^{a}_{a}$	$20.00\pm1.20^{de}_{c}$	$10.00\pm1.15^{b}_{b}$	$9.67\pm0.89^{bc}_{b}$	$0.00\pm0.00^{a}_{a}$	$12.00\pm1.20^{cd}_{b}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$
16	$12.00\pm1.00^{d}_{b}$	$16.00\pm0.50^{d}_{c}$	$20.00\pm1.20^{de}_{d}$	$20.00\pm1.15^{f}_{d}$	$15.00\pm0.58^{ef}_{c}$	$14.00\pm0.60^{c}_{bc}$	$21.00\pm1.20^{g}_{d}$	$16.00\pm0.57^{e}_{c}$	$22.00\pm1.10^{i}_{d}$	$0.00\pm0.00^{a}_{a}$
18	$12.00\pm1.00^{d}_{a}$	$19.00\pm1.10^{ef}_{cd}$	$13.00\pm1.15^{de}_{cd}$	$20.00\pm1.20^{f}_{d}$	$12.00\pm1.16^{cd}_{a}$	$15.00\pm0.50^{c}_{ab}$	$16.00\pm1.12^{f}_{bc}$	$19.00\pm0.60^{f}_{cd}$	$12.00\pm1.00^{cd}_{a}$	$16.00\pm0.58^{ef}_{bc}$
19	$0.00\pm0.00^{a}_{a}$	$5.00\pm1.00^{b}_{b}$	$16.00\pm1.20^{c}_{e}$	$15.00\pm0.60^{d}_{de}$	$12.00\pm0.59^{de}_{d}$	$19.00\pm0.57^{d}_{f}$	$10.00\pm1.15^{bc}_{c}$	$5.00\pm0.59^{c}_{b}$	$16.00\pm0.60^{fg}_{e}$	$15.00\pm0.57^{e}_{de}$
29	$9.00\pm0.57^{c}_{a}$	$10.00\pm1.00^{c}_{a}$	$12.00\pm1.20^{b}_{a}$	$18.00\pm1.10^{ef}_{b}$	$20.00\pm1.15^{h}_{bc}$	$22.00\pm1.00^{e}_{c}$	$17.00\pm0.50^{f}_{b}$	$10.00\pm0.60^{d}_{a}$	$20.00\pm1.15^{hi}_{bc}$	$18.00\pm0.60^{fg}_{b}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.32: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from ECS of Patients in General Hospital Wushishi

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
2	$11.00\pm0.50^{c}_{a}$	$15.00\pm0.60^{d}_{b}$	$18.00\pm1.00^{b}_{c}$	$11.00\pm0.59^{c}_{a}$	$12.33\pm0.88^{d}_{a}$	$12.00\pm0.58^{b}_{a}$	$15.00\pm0.60^{cd}_{b}$	$13.00\pm1.15^{d}_{ab}$	$20.00\pm0.58^{d}_{c}$	$11.00\pm0.57^{c}_{a}$
6	$0.00\pm0.00^{a}_{a}$	$15.00\pm0.57^{d}_{c}$	$18.00\pm2.00^{b}_{d}$	$6.00\pm1.20^{b}_{b}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$14.00\pm0.59^{c}_{c}$	$0.00\pm0.00^{a}_{a}$	$14.00\pm1.20^{b}_{c}$	$8.00\pm0.57^{b}_{b}$
10	$8.00\pm0.57^{b}_{bc}$	$20.00\pm0.50^{e}_{e}$	$22.00\pm1.10^{c}_{e}$	$6.00\pm0.60^{b}_{b}$	$9.00\pm1.00^{bc}_{c}$	$15.00\pm0.59^{c}_{d}$	$20.00\pm1.15_{e}$	$0.00\pm0.00^{a}_{a}$	$29.00\pm1.20^{f}_{f}$	$20.00\pm0.58^{f}_{e}$
12	$12.00\pm0.50^{c}_{cd}$	$10.00\pm1.00^{b}_{bc}$	$22.00\pm0.58^{c}_{g}$	$5.00\pm0.59^{b}_{a}$	$12.00\pm0.57^{ef}_{e}$	$18.00\pm0.60^{d}_{f}$	$14.00\pm1.10^{c}_{de}$	$8.00\pm0.59^{b}_{b}$	$25.00\pm0.57^{e}_{h}$	$16.00\pm0.60^{de}_{cd}$
15	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
18	$11.00\pm0.50^{c}_{b}$	$12.00\pm1.00^{bc}_{bc}$	$22.00\pm1.10^{c}_{f}$	$10.00\pm1.15^{c}_{b}$	$11.00\pm0.59^{cd}_{b}$	$16.00\pm1.20^{cd}_{de}$	$14.00\pm0.57^{c}_{cd}$	$7.00\pm0.60^{b}_{a}$	$17.00\pm0.58^{c}_{e}$	17.00 ± 0.50^{e}
22	$11.00\pm1.00^{c}_{a}$	$12.00\pm0.50^{bc}_{a}$	$20.00\pm1.20^{bc}_{d}$	$12.00\pm1.15^{c}_{a}$	$12.00\pm0.58^{de}_{ab}$	$17.00\pm1.10^{cd}_{c}$	$16.00\pm1.20^{cd}_{bc}$	$14.00\pm1.00^{d}_{abc}$	$12.00\pm1.30^{b}_{a}$	$11.00\pm0.59^{c}_{e}$
24	$17.00\pm1,00^{d}_{cd}$	$13.00\pm0.50^{cd}_{b}$	$19.00\pm0.57^{bc}_{de}$	$12.00\pm1.20^{c}_{b}$	$12.00\pm1.15^{f}_{cd}$	$16.00\pm0.58^{cd}_{ef}$	$17.00\pm0.57^{d}_{b}$	$10.00\pm0.60^{c}_{b}$	$21.00\pm0.54^{d}_{a}$	$11.00\pm0.59^{c}_{a}$
26	$13.00\pm0.50^{c}_{de}$	$10.00\pm0.57^{b}_{c}$	$17.00\pm0.60^{b}_{f}$	$6.00\pm1.00^{b}_{b}$	$11.00\pm0.59^{cd}_{cd}$	$15.00\pm0.58^{c}_{ef}$	$6.00\pm0.57^{b}_{b}$	$7.00\pm0.53^{b}_{b}$	$0.00\pm0.00^{a}_{a}$	$15.00\pm1.15^{d}_{ef}$
28	$16.00\pm1.00^{d}_{d}$	$0.00\pm0.00^{a}_{a}$	$17.00\pm1.10^{b}_{d}$	$13.00\pm1.20^{c}_{c}$	$8.00\pm1.15^{b}_{b}$	$0.00\pm0.00^{a}_{a}$	$16.00\pm0.60^{cd}_{d}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$13.00\pm0.58^{c}_{c}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.33: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from ECS of Patients in General Hospital Minna

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
2	$13.00\pm0.50^{bc}{}_{c}$	$10.00\pm0.59^{bc}{}_{b}$	$19.00\pm0.57^{b}{}_{e}$	$12.00\pm1.00^{b}{}_{c}$	$15.00\pm0.58^{c}{}_{d}$	$15.00\pm0.57^{d}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$12.00\pm0.60^{c}{}_{c}$	$13.00\pm0.59^{c}{}_{c}$
9	$14.00\pm0.50^{cd}{}_{bc}$	$10.00\pm0.59^{bc}{}_{a}$	$20.00\pm1.00^{bc}{}_{e}$	$14.00\pm1.20^{bc}{}_{bc}$	$12.00\pm1.15^{bc}{}_{ab}$	$12.00\pm1.20^{c}{}_{ab}$	$20.00\pm0.58^{d}{}_{e}$	$15.00\pm0.60^{c}{}_{cd}$	$17.00\pm0.57^{d}{}_{d}$	$16.00\pm1.10^{d}{}_{cd}$
14	$12.00\pm0.58^{b}{}_{c}$	$18.00\pm0.60^{d}{}_{d}$	$27.00\pm0.57^{d}{}_{f}$	$21.00\pm0.50^{d}{}_{e}$	$23.00\pm0.59^{d}{}_{e}$	$6.00\pm1.00^{bd}{}_{ab}$	$5.00\pm0.60^{b}{}_{a}$	$8.00\pm0.50^{b}{}_{b}$	$23.00\pm0.57^{b}{}_{e}$	$5.00\pm1.20^{b}{}_{a}$
15	$13.00\pm0.60^{bc}{}_{b}$	$9.00\pm0.57^{b}{}_{a}$	$20.00\pm1.20^{bc}{}_{d}$	$16.00\pm0.59^{c}{}_{c}$	$14.00\pm1.10^{b}{}_{bc}$	$15.00\pm0.58_{bc}$	$15.00\pm0.50^{c}{}_{bc}$	$14.00\pm1.15^{c}{}_{bc}$	$10.00\pm1.00^{c}{}_{a}$	$13.00\pm0.58^{c}{}_{b}$
16	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
17	$15.00\pm0.57^{d}{}_{b}$	$12.00\pm1.20^{c}{}_{a}$	$22.00\pm1.15^{c}{}_{cd}$	$13.00\pm0.60^{b}{}_{ab}$	$15.00\pm0.57^{c}{}_{b}$	$14.00\pm1.10^{cd}{}_{ab}$	$21.00\pm0.59^{d}{}_{cd}$	$23.00\pm0.57^{d}{}_{d}$	$20.00\pm1.00^{e}{}_{c}$	$29.00\pm0.50^{e}{}_{e}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.34: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from Urine of Patients in General Hospital Nasko

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
1	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
3	$16.00\pm1.00^{d}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$13.33\pm2.60^{a}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm1.70^{cd}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$24.00\pm1.15^{d}{}_{d}$
4	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm1.70^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm2.30^{c}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
6	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm1.70^{c}{}_{d}$	$6.00\pm0.50^{b}{}_{b}$	$10.00\pm2.00^{c}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
7	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
8	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm0.57^{c}{}_{c}$	$7.00\pm1.20^{b}{}_{b}$	$7.00\pm1.70^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$16.00\pm2.00^{c}{}_{c}$
9	$10.00\pm1.70^{b}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$3.00\pm1.15^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
10	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
12	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
14	$0.00\pm0.00^{a}{}_{a}$	$8.00\pm1.00^{b}{}_{bc}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$9.00\pm2.30^{b}{}_{cd}$	$16.00\pm1.70^{e}{}_{e}$	$12.00\pm1.15^{d}{}_{d}$	$5.00\pm1.20^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$
15	$15.00\pm1.00^{d}{}_{bc}$	$20.00\pm2.00^{d}{}_{c}$	$15.00\pm2.30^{c}{}_{bc}$	$10.00\pm1.10^{c}{}_{ab}$	$17.00\pm1.20^{d}{}_{c}$	$20.00\pm1.70^{d}{}_{c}$	$10.00\pm2.00^{cd}{}_{ab}$	$9.00\pm1.10^{bc}{}_{a}$	$10.00\pm1.90^{c}{}_{ab}$	$5.00\pm1.80^{b}{}_{a}$
16	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm0.50^{c}{}_{c}$	$12.00\pm1.15^{d}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$20.00\pm2.00^{d}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$
18	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
20	$29.00\pm1.00^{f}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm0.57^{c}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$16.00\pm1.15^{c}{}_{c}$	$9.00\pm1.20^{bc}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$11.00\pm1.10^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$
21	$21.00\pm1.00^{e}{}_{d}$	$15.00\pm2.30^{c}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm1.70^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm2.00^{cd}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
22	$3.00\pm1.00^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$5.00\pm1.15^{b}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.35: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from Urine of Patients in General Hospital Kontagora

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
27	$19.00\pm1.00^{e}{}_{d}$	$12.00\pm0.50^{de}{}_{ab}$	$9.00\pm1.15^{bc}{}_{a}$	$10.00\pm1.70^{c}{}_{a}$	$19.00\pm1.20^{f}{}_{d}$	$23.00\pm2.00^{g}{}_{e}$	$14.00\pm0.59^{c}{}_{bc}$	$17.00\pm1.10^{fg}{}_{cd}$	$10.00\pm1.15^{c}{}_{a}$	$18.00\pm1.00^{de}{}_{d}$
28	$19.00\pm1.00^{e}{}_{c}$	$10.00\pm1.10^{cd}{}_{b}$	$15.00\pm1.20^{de}{}_{bc}$	$10.00\pm2.30^{c}{}_{b}$	$0.00\pm0.00_{a}$	$15.00\pm3.00^{de}{}_{bc}$	$20.00\pm2.30^{de}{}_{c}$	$15.00\pm1.15^{def}{}_{bc}$	$0.00\pm0.00^{a}{}_{a}$	$20.00\pm1.10^{ef}{}_{c}$
29	$0.00\pm0.00^{a}{}_{a}$	$6.00\pm0.50^{b}{}_{b}$	$22.00\pm1.00^{fg}{}_{f}$	$20.00\pm1.15^{gh}{}_{ef}$	$12.00\pm1.10^{de}{}_{c}$	$19.00\pm1.20^{f}{}_{ef}$	$14.00\pm2.30^{c}{}_{cd}$	$6.00\pm0.58^{b}{}_{b}$	$20.00\pm0.60^{ef}{}_{ef}$	$17.00\pm1.10^{d}{}_{de}$
34	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm0.50^{cd}{}_{bc}$	$12.00\pm0.58^{cd}{}_{cde}$	$11.00\pm1.70^{c}{}_{bcd}$	$9.00\pm0.59^{c}{}_{b}$	$13.00\pm0.60^{cd}{}_{def}$	$15.00\pm1.20^{c}{}_{f}$	$14.00\pm0.57^{de}{}_{ef}$	$20.00\pm0.50^{ef}{}_{g}$	$15.00\pm0.53^{c}{}_{f}$
36	$20.00\pm1.00^{ef}{}_{d}$	$19.00\pm1.20^{hi}{}_{d}$	$25.00\pm1.15^{g}{}_{e}$	$13.00\pm0.50^{cde}{}_{c}$	$12.00\pm1.20^{de}{}_{bc}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$19.00\pm0.57^{gh}{}_{d}$	$10.00\pm0.60^{c}{}_{b}$	$10.00\pm0.59^{b}{}_{b}$
38	$6.00\pm1.00^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$35.00\pm3.00^{j}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{c}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
39	$25.00\pm1.00^{g}{}_{e}$	$20.00\pm1.70^{i}{}_{d}$	$23.00\pm2.00^{fg}{}_{de}$	$23.00\pm1.80^{hi}{}_{de}$	$18.67\pm1.50^{f}{}_{cd}$	$13.00\pm1.15^{cd}{}_{b}$	$15.00\pm1.10^{a}{}_{bc}$	$0.00\pm0.00^{a}{}_{a}$	$20.00\pm2.00^{ef}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$
40	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
42	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
43	$18.00\pm1.00^{de}{}_{bc}$	$18.00\pm1.15^{hi}{}_{bc}$	$15.00\pm1.20^{de}{}_{b}$	$15.67\pm0.89^{ef}{}_{b}$	$8.00\pm1.10^{bc}{}_{a}$	$11.00\pm1.15^{c}{}_{a}$	$25.00\pm1.00^{f}{}_{d}$	$20.00\pm2.90^{h}{}_{c}$	$10.00\pm0.59^{c}{}_{a}$	$25.00\pm0.50^{h}{}_{d}$
46	$16.00\pm0.50^{d}{}_{b}$	$14.00\pm0.57^{ef}{}_{b}$	$20.00\pm1.00^{f}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$19.00\pm1.10^{f}{}_{c}$	$14.00\pm0.58^{cd}{}_{b}$	$20.00\pm0.57^{de}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$19.00\pm1.15^{e}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$
47	$23.00\pm0.50^{fg}{}_{c}$	$24.00\pm1.15^{j}{}_{c}$	$30.00\pm2.30^{h}{}_{e}$	$25.00\pm1.20^{i}{}_{cd}$	$28.00\pm0.59^{g}{}_{de}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm2.30^{b}{}_{b}$	$13.00\pm0.60^{d}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
52	$24.00\pm2.30^{g}{}_{e}$	$9.00\pm0.57^{c}{}_{ab}$	$22.00\pm1.10^{fg}{}_{de}$	$11.00\pm00^{c}{}_{b}$	$21.00\pm1.20^{f}{}_{de}$	$7.00\pm1.10^{b}{}_{a}$	$10.00\pm1.00^{b}{}_{ab}$	$15.00\pm0.60^{def}{}_{c}$	$12.00\pm0.59^{c}{}_{bc}$	$20.00\pm0.57^{ef}{}_{d}$
56	$23.00\pm1.70^{fg}{}_{e}$	$17.00\pm1.20^{gh}{}_{cd}$	$14.00\pm0.60^{de}{}_{c}$	$5.00\pm0.58^{b}{}_{a}$	$14.00\pm0.59^{e}{}_{c}$	$19.00\pm0.50^{f}{}_{d}$	$10.00\pm1.00^{b}{}_{b}$	$20.00\pm0.57^{h}{}_{d}$	$6.00\pm1.20^{b}{}_{a}$	$25.00\pm1.15^{h}{}_{e}$
57	$0.00\pm0.00^{a}{}_{a}$	$13.00\pm1.00^{ef}{}_{b}$	$16.00\pm1.20^{e}{}_{c}$	$15.00\pm0.50^{def}{}_{bc}$	$14.00\pm0.58^{e}{}_{bc}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$16.00\pm1.10^{ef}{}_{c}$	$22.00\pm0.59^{f}{}_{d}$	$20.00\pm1.15^{ef}{}_{d}$
59	$13.00\pm1.00^{c}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$16.00\pm1.15^{e}{}_{c}$	$22.00\pm1.10^{hi}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm1.20^{de}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$9.00\pm1.70^{c}{}_{b}$	$10.00\pm1.20^{c}{}_{b}$	$21.00\pm0.59^{f}{}_{d}$
63	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
64	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$22.00\pm1.00^{fg}{}_{b}$
65	$20.00\pm1.00^{ef}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$25.00\pm1.10^{g}{}_{f}$	$17.00\pm1.15^{fg}{}_{c}$	$10.00\pm1.15^{cd}{}_{b}$	$21.00\pm0.60^{fg}{}_{de}$	$23.00\pm0.59^{ef}{}_{ef}$	$19.00\pm0.50^{gh}{}_{cd}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
66	$11.00\pm1.00^{c}{}_{b}$	$0.00\pm0.00_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$20.00\pm3.00^{fg}{}_{de}$	$23.00\pm0.50^{ef}{}_{e}$	$19.00\pm0.58^{gh}{}_{d}$	$15.00\pm0.60^{d}{}_{c}$	$29.00\pm0.57^{i}{}_{b}$
67	$20.00\pm1.10^{ef}{}_{ef}$	$15.00\pm0.50^{fg}{}_{cd}$	$8.00\pm2.30^{b}{}_{b}$	$12.00\pm1.20^{cd}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$18.00\pm2.30^{ef}{}_{de}$	$19.00\pm0.59^{d}{}_{e}$	$15.00\pm0.57^{def}{}_{cd}$	$0.00\pm0.00^{a}{}_{a}$	$23.00\pm0.57^{g}{}_{g}$
68	$20.00\pm0.50^{ef}{}_{e}$	$0.00\pm0.00^{a}{}_{a}$	$9.00\pm0.58^{bc}{}_{c}$	$10.00\pm0.60^{c}{}_{c}$	$6.00\pm2.00^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm0.57^{c}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$30.00\pm0.58^{i}{}_{f}$
71	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
73	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
75	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.36: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from Urine of Patients in General Hospital Agaie

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
1	$30.00\pm1.00^{h}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$20.00\pm0.57^{h}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm1.10^{de}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
2	$10.00\pm1.00^{d}{}_{ab}$	$0.00\pm0.00^{a}{}_{a}$	$7.33\pm7.33^{bc}{}_{ab}$	$7.00\pm1.15^{bc}{}_{ab}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$13.67\pm7.0^{c}{}_{b}$
3	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$25.00\pm0.57^{f}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$5.00\pm0.57^{ab}{}_{a}$
4	$13.00\pm1.00^{ef}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$6.00\pm0.57^{b}{}_{b}$	$10.33\pm0.88^{e}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm0.59^{c}{}_{e}$
5	$7.00\pm0.50^{c}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm0.60^{de}{}_{d}$	$10.00\pm1.10^{c}{}_{d}$	$10.00\pm0.57^{d}{}_{d}$	$17.00\pm1.20^{g}{}_{f}$	$4.00\pm1.15^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$14.00\pm1.00^{c}{}_{e}$
6	$10.00\pm0.50^{d}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm1.10^{bcd}{}_{b}$	$10.00\pm0.00^{de}{}_{b}$	$12.00\pm0.59^{cde}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
8	$12.00\pm0.57^{de}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$20.00\pm1.00^{e}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$13.00\pm1.10^{e}{}_{c}$	$6.00\pm0.58^{cd}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
9	$5.00\pm0.50^{bc}{}_{b}$	$10.00\pm1.00^{bc}{}_{c}$	$10.00\pm0.58^{bcd}{}_{c}$	$9.00\pm0.60^{cde}{}_{c}$	$14.00\pm1.15^{ef}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
10	$0.00\pm0.006^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$13.00\pm1.15^{def}{}_{c}$	$6.00\pm0.56^{b}{}_{b}$	$5.00\pm0.60^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
11	$15.00\pm0.57^{f}{}_{e}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$20.00\pm1.10^{g}{}_{f}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm1.20^{d}{}_{c}$	$13.00\pm0.58^{f}{}_{d}$	$5.00\pm0.50^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm0.57^{bc}{}_{c}$
12	$3.00\pm0.50^{b}{}_{b}$	$15.00\pm0.57^{cd}{}_{g}$	$13.00\pm0.60^{d}{}_{f}$	$8.00\pm1.00^{bcd}{}_{d}$	$4.00\pm0.59^{b}{}_{bc}$	$10.00\pm0.55^{d}{}_{e}$	$5.00\pm0.56^{b}{}_{c}$	$4.00\pm0.50^{b}{}_{bc}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
14	$14.00\pm0.50^{ef}{}_{e}$	$20.00\pm0.57^{d}{}_{f}$	$15.00\pm0.58^{de}{}_{e}$	$9.00\pm0.59^{cde}{}_{c}$	$12.00\pm0.60^{cde}{}_{d}$	$15.00\pm0.55^{f}{}_{e}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$5.00\pm0.57^{b}{}_{b}$	$6.00\pm0.60^{b}{}_{b}$
15	$15.00\pm0.50^{f}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm0.60^{de}{}_{d}$	$15.00\pm1.00^{f}{}_{d}$	$15.00\pm0.58^{fg}{}_{d}$	$8.00\pm1.20^{c}{}_{c}$	$10.00\pm0.59^{de}{}_{c}$	$5.00\pm0.57^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$8.00\pm0.55^{b}{}_{c}$
16	$10.00\pm1.00^{d}{}_{b}$	$10.00\pm0.50^{bc}{}_{b}$	$15.00\pm0.58^{de}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm1.70^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
17	$13.00\pm0.50^{ef}{}_{d}$	$20.00\pm0.57^{d}{}_{f}$	$6.00\pm0.60^{b}{}_{b}$	$11.00\pm1.10^{e}{}_{cd}$	$17.00\pm0.60^{g}{}_{e}$	$10.00\pm0.59^{d}{}_{c}$	$12.00\pm1.00^{ef}{}_{cd}$	$10.00\pm0.58^{c}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
18	$25.00\pm0.50^{g}{}_{b}$	$10.00\pm0.60^{bc}{}_{ab}$	$6.00\pm0.57^{b}{}_{b}$	$8.00\pm0.59^{bcd}{}_{ab}$	$5.00\pm0.58^{b}{}_{ab}$	$1.33\pm0.67^{a}{}_{a}$	$6.00\pm1.00^{cd}{}_{ab}$	$5.00\pm0.50^{b}{}_{ab}$	$5.00\pm0.50^{b}{}_{ab}$	$5.00\pm0.59^{ab}{}_{ab}$
19	$10.00\pm1.00^{d}{}_{bc}$	$7.00\pm7.00^{ab}{}_{ab}$	$19.00\pm0.59^{e}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$17.00\pm0.60^{g}{}_{cd}$	$12.00\pm0.58^{e}{}_{bcd}$	$13.00\pm0.58^{f}{}_{bcd}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm0.60^{c}{}_{bc}$	$0.00\pm0.00^{a}{}_{a}$
20	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$8.00\pm0.50^{cd}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$14.00\pm0.57^{c}{}_{c}$
21	$7.00\pm0.50^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$6.00\pm0.60^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm0.59^{f}{}_{d}$	$13.00\pm0.57^{f}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm0.58^{c}{}_{d}$
22	$10.00\pm1.00^{d}{}_{b}$	$6.66\pm6.66^{ab}{}_{ab}$	$12.00\pm0.50^{cd}{}_{b}$	$7.00\pm0.60^{bc}{}_{b}$	$11.00\pm0.57^{cd}{}_{b}$	$9.00\pm0.59^{cd}{}_{b}$	$10.00\pm0.58^{de}{}_{b}$	$10.00\pm0.50^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.37: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from Urine of Patients in General Hospital Suleja

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
1	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm1.00^{b}{}_{d}$	$23.00\pm0.50^{g}{}_{g}$	$15.00\pm0.57^{de}{}_{f}$	$10.00\pm0.58^{c}{}_{d}$	$5.00\pm0.59^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$13.00\pm0.60^{cd}{}_{e}$	$8.00\pm0.57^{c}{}_{c}$
2	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm0.50^{cd}{}_{d}$	$20.00\pm1.00^{f}{}_{e}$	$24.00\pm1.10^{g}{}_{f}$	$5.00\pm0.57^{b}{}_{b}$	$14.00\pm1.15^{d}{}_{d}$	$9.00\pm0.58^{b}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm0.59^{d}{}_{d}$	$13.00\pm0.60^{e}{}_{d}$
3	$5.00\pm0.50^{b}{}_{a}$	$13.00\pm0.6^{c}{}_{de}$	$18.00\pm0.57^{e}{}_{f}$	$14.00\pm1.00^{c}{}_{e}$	$9.00\pm0.58^{c}{}_{b}$	$10.00\pm1.15^{c}{}_{bc}$	$12.00\pm0.59^{c}{}_{cd}$	$14.00\pm0.60^{d}{}_{e}$	$18.00\pm0.57^{e}{}_{f}$	$11.00\pm0.55^{d}{}_{bcd}$
4	$20.00\pm1.00^{d}{}_{e}$	$15.00\pm0.50^{cd}{}_{d}$	$20.00\pm0.60^{f}{}_{e}$	$9.00\pm0.59^{b}{}_{c}$	$10.00\pm1.14^{c}{}_{c}$	$20.00\pm1.20^{e}{}_{e}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$5.00\pm0.00^{b}{}_{b}$
5	$0.00\pm0.00^{a}{}_{a}$	$16.00\pm1.00^{de}{}_{c}$	$15.00\pm0.50^{d}{}_{c}$	$15.00\pm0.60^{de}{}_{c}$	$14.00\pm1.10^{d}{}_{c}$	$5.00\pm0.57^{b}{}_{b}$	$14.00\pm1.20^{d}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$16.00\pm1.00^{f}{}_{c}$
6	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
7	$0.00\pm0.00^{a}{}_{a}$	$23.00\pm0.00^{g}{}_{e}$	$0.00\pm0.00^{a}{}_{a}$	$12.00\pm1.00^{cd}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$8.00\pm0.00^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$20.00\pm0.00^{f}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$
9	$9.00\pm0.50^{c}{}_{a}$	$18.00\pm0.60^{ef}{}_{bc}$	$26.00\pm0.59^{h}{}_{f}$	$11.00\pm0.57^{cd}{}_{c}$	$22.00\pm0.58^{e}{}_{de}$	$20.00\pm1.00^{e}{}_{cd}$	$20.00\pm0.57^{e}{}_{cd}$	$11.00\pm1.20^{c}{}_{a}$	$23.00\pm1.15^{g}{}_{e}$	$20.00\pm0.56^{g}{}_{cd}$
10	$0.00\pm0.00^{a}{}_{a}$	$20.00\pm1.00^{f}{}_{d}$	$8.67\pm0.88^{b}{}_{b}$	$25.00\pm0.57^{g}{}_{e}$	$11.00\pm0.58^{c}{}_{d}$	$20.00\pm1.20^{e}{}_{d}$	$20.00\pm0.60^{e}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$14.00\pm0.57^{c}{}_{c}$	$9.00\pm1.00^{c}{}_{b}$
11	$0.00\pm0.00^{a}{}_{a}$	$17.00\pm1.10^{de}{}_{d}$	$13.00\pm0.50^{c}{}_{c}$	$17.00\pm1.15^{e}{}_{d}$	$14.00\pm0.59^{d}{}_{c}$	$10.00\pm1.20^{c}{}_{b}$	$15.00\pm0.59^{d}{}_{cd}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm0.57^{d}{}_{cd}$	$0.00\pm0.00^{a}{}_{a}$
12	$0.00\pm0.00^{a}{}_{a}$	$20.00\pm0.50^{f}{}_{fg}$	$14.00\pm0.60^{cd}{}_{d}$	$21.00\pm0.57^{f}{}_{g}$	$11.00\pm0.58^{c}{}_{d}$	$19.00\pm0.57^{e}{}_{f}$	$23.00\pm1.00^{f}{}_{h}$	$5.00\pm0.57^{b}{}_{b}$	$11.00\pm0.59^{b}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.38: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from Urine of Patients in General Hospital Kuta

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
1	$0.00\pm0.00^{a}_{a}$	$10.00\pm1.00^{a}_{c}$	$20.00\pm1.10^{e}_{e}$	$10.00\pm1.15^{b}_{c}$	$0.00\pm0.00^{a}_{a}$	$6.00\pm1.20^{b}_{b}$	$17.00\pm1.10^{d}_{d}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$4.00\pm1.20^{b}_{b}$
2	$0.00\pm0.00^{a}_{a}$	$15.00\pm1.00^{bc}_{c}$	$13.00\pm0.50^{c}_{c}$	$10.00\pm1.10^{b}_{b}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$20.00\pm1.20^{e}_{d}$	$0.00\pm0.00^{a}_{a}$	$8.00\pm1.150^{bc}_{b}$	$0.00\pm0.00^{a}_{a}$
4	$0.00\pm0.00^{a}_{a}$	$17.00\pm1.00^{c}_{d}$	$16.00\pm1.10^{d}_{d}$	$15.00\pm0.58^{c}_{d}$	$0.00\pm0.00^{a}_{a}$	$4.00\pm1.20_{b}$	$5.00\pm0.60^{b}_{b}$	$0.00\pm0.00^{a}_{a}$	$10.00\pm1.15^{c}_{c}$	$0.00\pm0.00^{a}_{a}$
5	$0.00\pm0.00^{a}_{a}$	$13.00\pm0.50^{b}_{c}$	$17.00\pm1.00^{d}_{d}$	$0.00\pm0.00^{a}_{a}$	$12.00\pm0.57^{d}_{c}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$8.00\pm1.10^{c}_{b}$
6	$0.00\pm0.00^{a}_{a}$	$10.00\pm1.20^{a}_{c}$	$6.00\pm0.58^{a}_{b}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$6.00\pm1.20^{b}_{b}$	$0.00\pm0.00^{a}_{a}$
7	$3.00\pm0.50^{c}_{a}$	$25.00\pm0.57^{e}_{h}$	$16.00\pm0.58^{d}_{f}$	$24.00\pm0.59^{d}_{h}$	$13.00\pm0.60^{d}_{e}$	$21.00\pm0.55^{e}_{g}$	$15.00\pm0.56^{d}_{f}$	$7.00\pm0.50^{c}_{c}$	$10.00\pm0.57^{c}_{d}$	$5.00\pm0.58^{b}_{b}$
8	$2.00\pm0.50^{b}_{a}$	$20.00\pm0.60^{d}_{f}$	$5.00\pm0.57^{a}_{b}$	$10.00\pm0.59^{b}_{d}$	$3.00\pm0.58^{b}_{a}$	$8.00\pm0.55^{c}_{c}$	$5.00\pm0.56^{b}_{b}$	$8.00\pm0.60^{c}_{c}$	$16.00\pm0.57^{d}_{e}$	$5.00\pm0.60^{b}_{b}$
10	$0.00\pm0.00^{a}_{a}$	$20.00\pm0.50^{d}_{e}$	$20.00\pm0.57^{e}_{e}$	$15.00\pm0.58^{c}_{d}$	$5.00\pm0.56^{c}_{b}$	$16.00\pm0.59^{d}_{d}$	$5.00\pm0.60^{b}_{b}$	$0.00\pm0.00_{a}$	$15.00\pm0.58^{d}_{d}$	$8.00\pm0.59^{c}_{c}$
11	$0.00\pm0.00^{a}_{a}$	$15.00\pm0.57^{bc}_{d}$	$5.00\pm0.50^{a}_{b}$	$14.00\pm0.60^{c}_{d}$	$0.00\pm0.00^{a}_{a}$	$0.00\pm0.00^{a}_{a}$	$17.00\pm0.59^{d}_{e}$	$10.00\pm0.55^{d}_{c}$	$20.00\pm0.59^{e}_{f}$	$10.00\pm0.50^{c}_{c}$
12	$0.00\pm0.00^{a}_{a}$	$20.00\pm0.50^{d}_{e}$	$10.00\pm0.60^{b}_{c}$	$0.00\pm0.00^{a}_{a}$	$15.00\pm0.58^{e}_{d}$	$15.00\pm0.59^{d}_{d}$	$10.00\pm0.55^{c}_{c}$	$5.00\pm0.57^{b}_{b}$	$15.00\pm0.50^{d}_{d}$	$10.00\pm0.58^{c}_{c}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.39: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from Urine of Patients in General Hospital Bida

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
1	$9.00\pm0.50^{bc}{}_{b}$	$28.00\pm1.00^{ghi}{}_{f}$	$25.00\pm0.57^{fgh}{}_{e}$	$12.00\pm1.10^{bc}{}_{c}$	$15.00\pm0.59^{gh}{}_{d}$	$14.00\pm1.15^{c}{}_{cd}$	$13.00\pm0.60^{de}{}_{cd}$	$9.00\pm0.58^{ef}{}_{b}$	$15.00\pm0.55^{def}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$
2	$8.00\pm1.00^{b}{}_{b}$	$25.00\pm0.50^{def}{}_{g}$	$21.00\pm0.57^{d}{}_{f}$	$13.00\pm0.58^{cd}{}_{d}$	$10.00\pm1.10^{cde}{}_{bc}$	$13.00\pm1.15^{c}{}_{e}$	$11.00\pm0.59^{cd}{}_{cd}$	$17.00\pm0.60^{i}{}_{e}$	$19.00\pm1.20^{hi}{}_{ef}$	$0.00\pm0.00^{a}{}_{a}$
3	$0.00\pm0.00^{a}{}_{a}$	$24.00\pm1.00^{cde}{}_{d}$	$28.00\pm0.50^{ijk}{}_{e}$	$5.00\pm0.57^{b}{}_{b}$	$20.00\pm1.10^{j}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$4.00\pm1.20^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$6.00\pm1.15^{b}{}_{b}$
4	$0.00\pm0.00^{a}{}_{a}$	$24.00\pm0.50^{cde}{}_{g}$	$21.00\pm0.57^{d}{}_{f}$	$10.00\pm1.00^{b}{}_{c}$	$12.00\pm1.10^{ef}{}_{cd}$	$13.00\pm1.20^{c}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$6.00\pm0.58^{bc}{}_{b}$	$19.00\pm1.20^{hi}{}_{f}$	$16.00\pm1.15^{gh}{}_{e}$
5	$0.00\pm0.00^{a}{}_{a}$	$29.00\pm0.50^{hij}{}_{f}$	$15.00\pm1.00^{bc}{}_{de}$	$13.00\pm0.58^{cd}{}_{cd}$	$16.00\pm1.10^{hi}{}_{e}$	$12.00\pm0.60^{c}{}_{c}$	$11.00\pm0.59^{cd}{}_{c}$	$5.00\pm0.58^{b}{}_{b}$	$13.00\pm0.50^{cd}{}_{cd}$	$0.00\pm0.00^{a}{}_{a}$
6	$0.00\pm0.00^{a}{}_{a}$	$26.00\pm0.50^{efg}{}_{d}$	$27.00\pm1.00^{hij}{}_{d}$	$15.00\pm0.57^{ef}{}_{c}$	$8.00\pm0.58^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm0.59^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$16.00\pm0.60^{efg}{}_{c}$	$15.00\pm0.57^{fg}{}_{c}$
7	$12.00\pm0.50^{def}{}_{bc}$	$25.00\pm0.57^{def}{}_{f}$	$26.00\pm0.58^{ghi}{}_{f}$	$4.00\pm0.59^{b}{}_{a}$	$10.00\pm0.60^{cde}{}_{b}$	$16.00\pm0.55^{d}{}_{de}$	$17.00\pm0.50^{fg}{}_{e}$	$14.00\pm0.57^{h}{}_{cd}$	$16.00\pm1.10^{efg}{}_{de}$	$12.00\pm0.59^{e}{}_{bc}$
8	$10.00\pm0.50^{cd}{}_{bc}$	$30.00\pm0.55^{ij}{}_{h}$	$24.00\pm1.00^{efg}{}_{g}$	$12.00\pm0.57^{bc}{}_{cd}$	$9.00\pm1.10^{cd}{}_{b}$	$20.00\pm0.58^{ef}{}_{f}$	$15.00\pm1.15^{ef}{}_{e}$	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm1.20^{b}{}_{bc}$	$13.00\pm0.60^{ef}{}_{de}$
9	$14.00\pm0.50^{fg}{}_{abc}$	$25.67\pm0.89^{efg}{}_{e}$	$22.67\pm0.88^{def}{}_{d}$	$15.00\pm0.57^{ef}{}_{bc}$	$13.00\pm0.58^{fg}{}_{ab}$	$13.00\pm0.60^{c}{}_{ab}$	$13.00\pm0.59^{de}{}_{ab}$	$12.00\pm0.50^{g}{}_{a}$	$16.00\pm1.15^{efg}{}_{c}$	$13.00\pm0.57^{ef}{}_{ab}$
11	$24.00\pm0.50^{i}{}_{f}$	$20.00\pm0.57^{b}{}_{e}$	$30.00\pm1.00^{k}{}_{g}$	$14.00\pm0.58^{cd}{}_{d}$	$3.00\pm0.59^{b}{}_{b}$	$13.00\pm0.60^{c}{}_{d}$	$19.00\pm1.10^{g}{}_{e}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$9.00\pm0.58^{cd}{}_{c}$
13	$13.00\pm0.50^{ef}{}_{b}$	$31.00\pm0.57^{j}{}_{g}$	$29.00\pm0.58^{jk}{}_{f}$	$13.00\pm0.59^{cd}{}_{b}$	$12.00\pm0.50^{ef}{}_{b}$	$21.00\pm0.60^{ef}{}_{e}$	$15.00\pm0.55^{ef}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$16.00\pm0.56^{efg}{}_{c}$	$19.00\pm0.59^{i}{}_{d}$
14	$14.00\pm1.00^{fg}{}_{c}$	$27.00\pm1.10^{fgh}{}_{f}$	$27.00\pm1.70^{hij}{}_{f}$	$12.00\pm0.50^{bc}{}_{bc}$	$10.00\pm1.20^{cde}{}_{b}$	$19.00\pm1.15^{e}{}_{d}$	$25.00\pm0.59^{h}{}_{ef}$	$10.00\pm1.00^{f}{}_{b}$	$22.00\pm1.14^{j}{}_{de}$	$6.00\pm1.20^{b}{}_{a}$
15	$16.00\pm1.00^{h}{}_{a}$	$28.00\pm1.10^{ghi}{}_{e}$	$17.00\pm1.15^{c}{}_{c}$	$12.00\pm1.20^{bc}{}_{b}$	$11.00\pm0.50^{de}{}_{b}$	$21.00\pm0.60^{ef}{}_{d}$	$15.00\pm0.57^{ef}{}_{c}$	$6.00\pm0.59^{bc}{}_{a}$	$17.00\pm0.58^{fgh}{}_{c}$	$8.00\pm1.00^{bc}{}_{a}$
17	$12.00\pm0.50^{def}{}_{ab}$	$27.00\pm1.00^{fgh}{}_{f}$	$25.00\pm0.57^{fgh}{}_{f}$	$12.00\pm0.58_{ab}$	$10.00\pm0.59^{ab}{}_{b}$	$22.00\pm0.60^{f}{}_{e}$	$14.00\pm1.10^{cde}{}_{bc}$	$10.00\pm1.15^{f}{}_{a}$	$14.00\pm1.20^{cde}{}_{bc}$	$20.00\pm1.00^{ij}{}_{de}$
18	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{ag}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
19	$12.00\pm0.50^{def}{}_{d}$	$22.67\pm0.88^{cd}{}_{f}$	$26.00\pm0.58^{ghi}{}_{g}$	$20.00\pm0.57^{g}{}_{e}$	$5.00\pm0.59^{b}{}_{b}$	$6.00\pm0.60^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$8.00\pm0.57^{de}{}_{c}$	$21.00\pm0.56^{ij}{}_{e}$	$0.00\pm0.00^{a}{}_{a}$
20	$10.00\pm0.50^{cd}{}_{ab}$	$26.00\pm0.57^{efg}{}_{f}$	$26.00\pm0.58^{ghi}{}_{f}$	$15.00\pm0.59^{ef}{}_{c}$	$11.00\pm0.60^{def}{}_{b}$	$20.00\pm0.50^{ef}{}_{de}$	$11.00\pm0.59^{cd}{}_{b}$	$8.00\pm0.57^{de}{}_{a}$	$18.00\pm1.00^{gh}{}_{d}$	$22.00\pm1.15^{j}{}_{e}$
21	$11.00\pm0.50^{cde}{}_{c}$	$22.00\pm1.00^{bc}{}_{e}$	$25.00\pm0.57^{fgh}{}_{f}$	$4.00\pm0.58^{b}{}_{b}$	$18.00\pm1.10^{ij}{}_{d}$	$5.00\pm0.60^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$6.00\pm0.59^{bc}{}_{b}$	$10.00\pm0.57^{b}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$
22	$11.00\pm0.57^{cde}{}_{b}$	$25.00\pm1.00^{def}{}_{e}$	$22.00\pm0.58^{de}{}_{d}$	$19.00\pm0.59^{g}{}_{c}$	$10.00\pm0.50^{cd}{}_{b}$	$22.00\pm0.60^{f}{}_{d}$	$18.00\pm0.55^{g}{}_{c}$	$7.00\pm0.56^{cd}{}_{a}$	$29.00\pm0.55^{k}{}_{f}$	$11.00\pm0.57^{de}{}_{b}$
23	$15.00\pm0.50^{gh}{}_{bc}$	$25.00\pm0.57^{def}{}_{e}$	$13.00\pm0.58^{b}{}_{ab}$	$17.00\pm0.59^{f}{}_{cd}$	$13.00\pm0.60^{fg}{}_{ab}$	$12.00\pm0.55^{c}{}_{a}$	$17.00\pm0.50^{fg}{}_{cd}$	$14.00\pm0.59^{h}{}_{ab}$	$12.00\pm0.57^{bc}{}_{a}$	$8.00\pm1.70^{bc}{}_{a}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.40: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from Urine of Patients in General Hospital Wushishi

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
1	$11.00\pm0.50^{c}_{c}$	$13.00\pm0.57^{bc}_{d}$	$18.00\pm0.58^{b}_{f}$	$11.00\pm0.59^{c}_{c}$	$18.00\pm0.60^{e}_{f}$	$19.00\pm0.55^{g}_{f}$	$16.00\pm0.56^{de}_{e}$	$18.00\pm0.50^{f}_{f}$	$0.00\pm0.00^{a}_{a}$	$3.00\pm0.57^{b}_{b}$
7	$10.00\pm0.50^{c}_{b}$	$18.00\pm0.57^{e}_{f}$	$21.00\pm0.58^{c}_{g}$	$7.00\pm0.59^{b}_{a}$	$17.00\pm0.60^{de}_{de}$	$13.00\pm0.50^{d}_{c}$	$15.00\pm0.55^{de}_{d}$	$11.00\pm0.56^{d}_{b}$	$16.00\pm0.58^{g}_{de}$	$10.00\pm0.59^{d}_{b}$
8	$0.00\pm0.00^{a}_{a}$	$15.00\pm0.50^{cd}_{e}$	$25.00\pm0.57^{d}_{g}$	$13.00\pm0.58^{d}_{d}$	$8.00\pm0.59^{b}_{c}$	$7.00\pm0.60^{c}_{c}$	$17.00\pm0.55_{f}$	$0.00\pm0.00^{a}_{a}$	$4.00\pm0.57^{b}_{c}$	$13.00\pm0.60^{e}_{d}$
11	$0.00\pm0.00^{a}_{a}$	$17.00\pm0.57^{de}_{f}$	$27.00\pm0.58_{g}$	$11.00\pm0.59^{c}_{d}$	$6.00\pm0.60^{b}_{c}$	$18.00\pm0.55^{f}_{f}$	$4.00\pm0.56^{b}_{b}$	$4.00\pm0.59^{b}_{b}$	$6.00\pm0.57^{c}_{b}$	$13.00\pm0.60^{e}_{e}$
13	0.00 ± 0.00^{ac}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}
15	$11.00\pm0.40_{b}$	$11.00\pm0.60^{b}_{b}$	$19.00\pm0.48^{b}_{d}$	$11.00\pm0.57^{c}_{b}$	$13.00\pm0.59^{c}_{ab}$	$17.00\pm0.51^{ef}_{d}$	$14.00\pm1.00^{d}_{c}$	$11.00\pm0.56^{d}_{b}$	$20.00\pm0.62^{h}_{e}$	$6.00\pm0.50^{c}_{a}$
16	$11.00\pm0.51^{c}_{b}$	$16.00\pm1.20^{de}_{cb}$	$25.00\pm0.57^{d}_{e}$	$10.00\pm1.10^{c}_{ab}$	$18.00\pm0.59^{e}_{d}$	$8.00\pm0.53^{c}_{a}$	$16.00\pm1.15^{de}_{cd}$	$16.00\pm1.20^{e}_{cd}$	$14.00\pm0.37^{f}_{c}$	$17.00\pm0.51^{f}_{d}$
21	$15.00\pm0.50^{d}_{e}$	$15.00\pm1.00^{cd}_{e}$	$23.00\pm0.57^{d}_{f}$	$10.00\pm0.58^{c}_{c}$	$15.00\pm0.59^{cd}_{e}$	$16.00\pm0.60^{e}_{e}$	$16.00\pm0.55^{de}_{e}$	$7.00\pm0.56^{c}_{b}$	$12.00\pm0.51^{e}_{d}$	$0.00\pm0.00^{a}_{a}$
22	$5.00\pm0.50^{b}_{b}$	$18.00\pm0.58^{e}_{e}$	$24.00\pm1.00^{d}_{f}$	$0.00\pm0.00^{a}_{a}$	$14.00\pm1.10^{c}_{d}$	$5.00\pm0.57^{b}_{b}$	$10.00\pm1.15^{c}_{c}$	$0.00\pm0.00^{a}_{a}$	$8.00\pm0.55^{d}_{c}$	$22.00\pm0.60^{g}_{f}$
28	$11.00\pm0.50^{c}_{cd}$	$13.00\pm0.57^{bc}_{de}$	$21.00\pm0.58^{c}_{g}$	$10.00\pm0.59^{c}_{bc}$	$18.00\pm1.20^{e}_{f}$	$17.00\pm0.50^{ef}_{f}$	$14.00\pm1.15^{d}_{e}$	$8.00\pm1.10^{c}_{ab}$	$11.00\pm0.57^{e}_{cd}$	$6.00\pm0.60^{c}_{a}$

Table 4.41: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from Urine of Patients in General Hospital Minna

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
1	$22.00\pm1.00^{c}{}_{h}$	$18.00\pm1.10^{bc}{}_{ef}$	$16.00\pm1.15^{b}{}_{e}$	$5.00\pm0.50^{a}{}_{a}$	$12.00\pm0.57^{b}{}_{d}$	$19.00\pm0.58^{d}{}_{fg}$	$21.00\pm0.59^{e}{}_{gh}$	$11.00\pm0.60^{c}{}_{cd}$	$8.00\pm0.55^{a}{}_{b}$	$9.00\pm0.51^{b}{}_{bc}$
2	$12.00\pm1.00^{ab}{}_{b}$	$16.00\pm1.10^{b}{}_{cd}$	$25.00\pm1.15^{d}{}_{f}$	$8.00\pm0.50^{b}{}_{a}$	$11.00\pm0.60^{a}{}_{bc}$	$19.00\pm1.20^{d}{}_{de}$	$20.00\pm1.00^{e}{}_{e}$	$13.00\pm0.58^{c}{}_{bc}$	$20.00\pm1.10^{d}{}_{e}$	$16.00\pm1.20^{d}{}_{cd}$
3	$26.00\pm1.00^{d}{}_{e}$	$21.00\pm0.50^{de}{}_{de}$	$26.00\pm2.00^{d}{}_{e}$	$18.00\pm0.57^{d}{}_{cd}$	$12.00\pm1.00^{c}{}_{b}$	$15.33\pm6.00^{cd}{}_{bcd}$	$9.00\pm0.59^{a}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$11.00\pm0.55^{b}{}_{b}$	$12.00\pm0.60^{c}{}_{bc}$
5	$20.00\pm1.00^{c}{}_{e}$	$19.00\pm0.50^{cd}{}_{de}$	$17.00\pm0.60^{bc}{}_{cd}$	$17.00\pm0.57^{d}{}_{cd}$	$18.00\pm1.10^{de}{}_{cde}$	$10.00\pm1.15^{bc}{}_{b}$	$7.00\pm0.58^{ab}{}_{a}$	$5.00\pm0.59^{b}{}_{a}$	$16.00\pm1.20^{c}{}_{c}$	$12.00\pm1.00^{c}{}_{b}$
8	$15.00\pm0.50^{b}{}_{e}$	$13.00\pm1.00^{a}{}_{cde}$	$14.00\pm1.10^{ab}{}_{de}$	$10.00\pm0.57^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$11.00\pm0.58^{bc}{}_{bc}$	$13.00\pm1.10^{c}{}_{cde}$	$12.00\pm1.15^{b}{}_{bcd}$	$25.00\pm0.60^{e}{}_{f}$
9	$10.00\pm1.00^{a}{}_{b}$	$13.00\pm0.50^{a}{}_{c}$	$11.00\pm0.60^{a}{}_{bc}$	$20.00\pm0.57^{e}{}_{e}$	$15.00\pm0.58^{c}{}_{d}$	$7.00\pm0.59^{b}{}_{a}$	$12.00\pm0.60^{c}{}_{bc}$	$11.00\pm0.55^{c}{}_{bc}$	$19.00\pm0.51^{d}{}_{e}$	$18.00\pm0.56^{d}{}_{e}$
12	$26.00\pm1.00^{d}{}_{e}$	$23.00\pm0.50^{e}{}_{d}$	$26.00\pm0.60^{d}{}_{e}$	$7.00\pm0.57^{b}{}_{a}$	$16.00\pm0.56^{cd}{}_{b}$	$17.00\pm0.59^{d}{}_{b}$	$16.00\pm0.58^{d}{}_{b}$	$4.00\pm0.51^{b}{}_{a}$	$21.00\pm0.56^{d}{}_{c}$	$5.00\pm0.60^{a}{}_{a}$
16	$21.00\pm0.50^{c}{}_{e}$	$18.00\pm0.59^{bc}{}_{cd}$	$20.00\pm0.57^{c}{}_{de}$	$5.00\pm0.60^{a}{}_{a}$	$20.00\pm1.00^{e}{}_{de}$	$12.00\pm1.15^{bcd}{}_{b}$	$12.00\pm0.58^{c}{}_{b}$	$13.00\pm0.56^{c}{}_{b}$	$16.00\pm0.50^{c}{}_{c}$	$25.00\pm0.59^{e}{}_{f}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.42: Antibiotics Susceptibility Test of Multi Drug Resistant Bacterial Isolates from Urine of Patients in General Hospital Lapai

Mcos	OFX	PEF	CPX	AU	CN	S	CEP	NA	SXT	PN
3	$17.00\pm0.50^{f}{}_{d}$	$31.00\pm0.60^{h}{}_{f}$	$25.00\pm0.57^{fg}{}_{e}$	$0.00\pm0.00^{a}{}_{a}$	$12.00\pm0.58^{bc}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$5.00\pm0.59^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$4.00\pm0.50^{b}{}_{b}$
6	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm1.00^{g}{}_{e}$	$11.00\pm0.60^{d}{}_{c}$	$13.00\pm0.57^{c}{}_{d}$	$7.00\pm0.58^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$16.00\pm0.59^{d}{}_{e}$
7	$18.00\pm0.50^{ef}{}_{b}$	$19.00\pm1.00^{g}{}_{b}$	$25.00\pm0.60^{g}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
8	$20.00\pm0.50^{g}{}_{c}$	$30.00\pm1.00^{gh}{}_{e}$	$25.00\pm1.10^{f}{}_{d}$	$5.00\pm0.60^{bc}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$20.00\pm0.58^{ef}{}_{c}$	$21.00\pm0.59^{g}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
10	$26.00\pm0.50^{g}{}_{c}$	$33.00\pm1.00^{i}{}_{d}$	$28.00\pm1.10^{g}{}_{c}$	$21.00\pm1.20^{g}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$13.00\pm1.15^{i}{}_{c}$	$22.00\pm1.00^{g}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$
14	$21.00\pm0.50^{g}{}_{c}$	$27.00\pm0.58^{g}{}_{d}$	$27.00\pm1.10^{g}{}_{d}$	$27.00\pm1.00^{gh}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$21.00\pm0.58^{g}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$14.00\pm0.60^{de}{}_{b}$
15	$20.00\pm0.50^{g}{}_{c}$	$17.00\pm0.60^{de}{}_{b}$	$22.00\pm1.00^{fg}{}_{cd}$	$20.00\pm1.10^{ef}{}_{c}$	$12.00\pm1.20^{f}{}_{cd}$	$21.00\pm0.58^{g}{}_{cd}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$23.00\pm1.15^{g}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$
17	$16.67\pm4.30^{de}{}_{b}$	$21.00\pm0.50^{fg}{}_{b}$	$20.00\pm1.00^{g}{}_{b}$	$16.00\pm1.10^{d}{}_{b}$	$19.00\pm1.20^{g}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$3.00\pm0.58^{ab}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
25	$0.00\pm0.00^{a}{}_{a}$	$10.00\pm0.50^{cd}{}_{e}$	$22.00\pm1.00^{i}{}_{g}$	$2.00\pm0.58^{ab}{}_{b}$	$3.00\pm0.57^{ab}{}_{bc}$	$0.00\pm0.00^{a}{}_{a}$	$7.33\pm0.88^{bc}{}_{d}$	$9.00\pm0.60^{d}{}_{de}$	$13.00\pm0.57^{d}{}_{f}$	$4.00\pm0.55^{b}{}_{c}$
28	$0.00\pm0.00^{a}{}_{a}$	$18.00\pm1.00^{ef}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$11.00\pm0.59^{d}{}_{c}$	$11.00\pm0.60^{cd}{}_{c}$	$5.00\pm0.58^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
29	$0.00\pm0.00^{a}{}_{a}$	$34.00\pm1.00^{ij}{}_{g}$	$23.00\pm0.58^{h}{}_{f}$	$15.00\pm0.57^{ef}{}_{d}$	$12.00\pm0.59^{cd}{}_{c}$	$18.00\pm1.70^{ef}{}_{e}$	$14.67\pm0.33^{de}{}_{d}$	$5.00\pm0.58^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$17.00\pm1.15^{f}{}_{de}$
32	$15.00\pm1.00^{de}{}_{b}$	$25.00\pm1.10^{ef}{}_{d}$	$29.00\pm1.20^{f}{}_{e}$	$15.00\pm0.50^{ef}{}_{b}$	$13.00\pm1.15^{c}{}_{ab}$	$15.00\pm1.13^{c}{}_{b}$	$10.00\pm1.16^{de}{}_{a}$	$10.00\pm1.10^{de}{}_{a}$	$13.00\pm0.59^{d}{}_{ab}$	$22.00\pm0.60^{g}{}_{c}$
34	$0.00\pm0.00^{a}{}_{a}$	$11.00\pm0.50^{c}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$13.00\pm1.00^{bc}{}_{e}$	$6.00\pm1.10^{c}{}_{b}$	$5.00\pm0.59^{b}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$8.00\pm0.60^{cd}{}_{c}$
36	$0.00\pm0.00^{a}{}_{a}$	$21.00\pm0.50^{ef}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$14.00\pm1.10^{cd}{}_{c}$	$11.00\pm0.57^{bc}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$13.00\pm0.58^{e}{}_{c}$
37	$0.00\pm0.00^{a}{}_{a}$	$32.00\pm1.00^{i}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$	$12.00\pm0.57^{bc}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$9.00\pm1.10^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$
38	$0.00\pm0.00^{a}{}_{a}$	$33.00\pm1.00^{j}{}_{e}$	$0.00\pm0.00^{a}{}_{a}$	$12.00\pm0.58^{bc}{}_{c}$	$4.00\pm0.59^{ab}{}_{b}$	$16.00\pm1.15^{cd}{}_{d}$	$3.00\pm0.58^{ab}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$4.00\pm0.60^{b}{}_{b}$
39	$0.00\pm0.00^{a}{}_{a}$	$32.00\pm1.00^{i}{}_{e}$	$0.00\pm0.00^{a}{}_{a}$	$15.00\pm0.50^{d}{}_{c}$	$0.00\pm0.00^{a}{}_{a}$	$5.00\pm1.20^{c}{}_{b}$	$0.00\pm0.00^{a}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$18.00\pm1.20^{ef}{}_{d}$	$0.00\pm0.00^{a}{}_{a}$
40	$10.00\pm1.15^{cd}{}_{a}$	$28.00\pm1.10^{f}{}_{a}$	$25.00\pm0.50^{ef}{}_{a}$	$12.00\pm0.59^{c}{}_{a}$	$4.67\pm0.89^{b}{}_{a}$	$11.00\pm0.58^{c}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$	$9.00\pm0.57^{bc}{}_{a}$	$19.00\pm0.55^{f}{}_{a}$	$0.00\pm0.00^{a}{}_{a}$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

4.1.5 Percentage Occurrence of Multidrug Resistant Bacterial Load in Each General Hospital

The load of multidrug resistant bacteria in nine general hospitals in Niger state, is shown in Figure 4.1. The result obtained revealed General Hospital Lapai (15.8%) had the highest followed by General Hospital Kontagora (14.5%) and General Hospital Bida (14.5%) which had equal load of multidrug resistant bacteria, while General Hospital Minna (6.1%) had the least load of multidrug resistant bacteria.

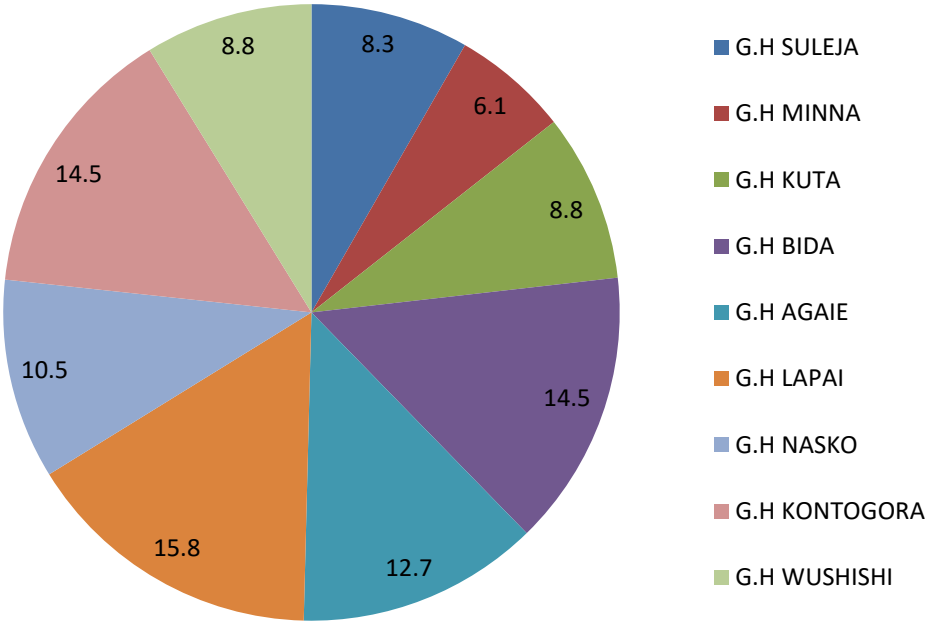

Figure 4.1: Percentage Occurrence of Multidrug Resistant Bacteria in Each General Hospital

4.1.6 Susceptibility pattern of multidrug resistant bacteria in patients with pelvic inflammatory disease (PID) from nine General Hospital

Table 4.43 show all multidrug resistant bacterial isolates obtained from Agaie and Wushishi were completely resistant (that is 100% resistant) to both Sulfamethoxazole and Augmentin.

Table 4.43: Susceptibility Pattern of Multidrug Resistant Bacteria in Patients with PID from Nine General Hospitals

	No of Isolates	Susceptibility pattern	OFX(%)	PEF(%)	CPX(%)	AU(%)	CN(%)	ST(%)	CEP(%)	NA(%)	SXT(%)	PN(%)
G.H.S	19	S	1(5.3)	5(26.3)	3(15.8)	3(15.8)	1(5.3)	0(0)	3(15.8)	0(0)	3(15.8)	1(5.3)
		I	0(0)	6(31.6)	6(31.6)	4(21.0)	2(10.5)	4(21.1)	1(5.3)	1(5.3)	6(31.6)	1(5.3)
		R	18(94.7)	8(42.1)	10(52.6)	12(63.2)	16(84.2)	15(78.9)	15(78.9)	18(94.7)	10(52.6)	17(89.4)
G.H.M	14	S	5(35.7)	2(14.3)	5(35.7)	3(21.4)	7(50)	0(0)	4(28.6)	1(7.1)	8(57.1)	4(28.6)
		I	5(35.7)	5(35.7)	6(42.9)	3(21.4)	1(7.1)	6(42.9)	2(14.3)	2(14.3)	3(21.4)	2(14.3)
		R	4(28.6)	7(50)	3(21.4)	8(57.1)	6(42.9)	8(57.1)	8(57.1)	11(78.6)	3(21.4)	8(57.1)
G.H.K	20	S	2(10)	3(15)	4(20)	2(10)	2(10)	1(5)	2(10)	0(0)	5(25)	0(0)
		I	1(5)	8(40)	7(35)	7(35)	3(15)	6(30)	5(25)	2(10)	5(25)	1(5)
		R	17(85)	9(45)	9(45)	11(55)	15(75)	13(65)	13(65)	18(90)	10(50)	19(95)
G.H.B	33	S	5(15.2)	21(63.6)	22(66.6)	7(21.2)	9(27.3)	5(15.2)	7(21.2)	2(6.0)	16(48.5)	5(15.2)
		I	4(12.1)	6(18.2)	6(18.2)	9(27.3)	2(6.1)	11(33.3)	8(24.2)	6(18.2)	8(24.2)	4(12.1)
		R	24(72.7)	6(18.2)	5(15.2)	17(51.5)	22(66.6)	17(51.5)	18(56)	25(75.8)	9(27.3)	24(72.2)
G.H.A	29	S	6(20.7)	3(10.3)	1(3.4)	1(3.4)	1(3.4)	0(0)	2(6.9)	2(6.9)	0(0)	1(3.4)
		I	9(31.0)	3(10.3)	3(10.3)	1(3.4)	5(17.2)	3(10.3)	3(10.3)	1(3.4)	0(0)	6(20.7)
		R	14(48.3)	23(79.3)	25(86.2)	27(93.1)	23(79.3)	26(89.7)	24(82.8)	26(89.7)	29(100)	22(75.9)
G.H.L	36	S	7(19.4)	16(44.4)	14(38.9)	5(13.9)	5(13.9)	2(5.5)	1(2.8)	1(2.8)	6 (16.7)	2(5.5)
		I	3(8.3)	7(19.4)	9(25)	7(19.4)	1(2.8)	6(16.7)	0(0)	2(5.5)	10(27.8)	3(8.3)
		R	26(72.2)	13(36.1)	13(36.1)	24(66.7)	30(83.3)	28(77.8)	35(97.2)	33(91.7)	20(55.5)	31(86.1)
G.H.KN	33	S	17(52)	1(3)	9(27.3)	5(15.2)	7(21.2)	2(6.1)	8(24.2)	6(18.2)	7(21.2)	16(48.5)
		I	1(3)	8(24.2)	5(15.2)	4(12.1)	2(6.1)	8(24.2)	4(12.1)	7(21.2)	4(12.1)	2(6.1)
		R	15(45)	24(72.7)	19(57.5)	24(72.7)	24(72.7)	23(69.7)	21(63.6)	20(60.6)	22(66.7)	15(45.4)
G.H.W	20	S	2(10)	0(0)	10(50)	0(0)	5(25)	0(0)	1(5)	0(0)	7(35)	4(20)
		I	2(10)	5(25)	8(40)	0(0)	2(10)	11(55)	9(45)	3(15)	5(25)	2(10)
		R	16(80)	15(75)	2(10)	20(100)	13(65)	9(45)	10(50)	17(85)	8(40)	14(70)

	No of Isolates	Susceptibility pattern	OFX(%)	PEF(%)	CPX(%)	AU(%)	CN(%)	ST(%)	CEP(%)	NA(%)	SXT(%)	PN(%)
G.H.N	24	S	4(16.7)	0(0)	1(4.2)	1(4.2)	3(12.5)	0(0)	2(8.3)	1(4.2)	3(12.5)	4(16.7)
		I	5(20.8)	1(4.2)	3(12.5)	0(0)	0(0)	6(25)	3(12.5)	1(4.2)	0(0)	1(4.1)
		R	15(62.5)	23(95.8)	20(83.3)	23(95.8)	21(87.5)	18(75)	19(79.2)	22(91.6)	21(87.5)	19(79.2)

Key:OFX:Ofloxacin;PEF:Perfloxacin;CPX:Ciprofloxacin;NA:Nalidixicacid;CN:Gentamicin;ST:Streptomycin;PN:Ampicillin;Cep:Cephalexin;AU:Augmentin;SXT:Sulfamethoxazole; S: Susceptible; I:Intermidiate; R:Resistance; G.H.S: General Hospital Suleja; G.H.M: General Hospital Minna; G.H.K: General Hospital Kuta; G.H.B:General Hospital Bida; G.H.A: General Hospital Agaie; G.H.L:General Hospital Lapai; G.H.KN: General Hospital Kontagora; G.H.W: General Hospital Wushishi; G.H.N: General Hospital Nasko

4.1.7 Multiple antibiotics resistance (MAR) indices of the isolated MDR bacteria

Table 4.44 show 35(15.4%) multidrug resistant bacteria out of 228 were completely resistant to all the antibiotics, while 14 (6.1%) were resistant to only 3 antibiotics as seen below.

Table 4.44: Multiple Antibiotics Resistance (MAR) Indices of the Isolated MDR Bacteria

MDR Bacterial isolates	Resistance pattern	Susceptibility pattern	Number of antibiotics that were resisted	MAR Index
H-1S	OFX, AUG, CN, S, CEP,NA,SXT,PN	PEF, CPX	8	0.8
H-2S	OFX, AUG, CN, S, CEP,NA,SXT,PN	PEF, CPX	8	0.8
H-3S	OFX, AUG, CN, S, CEP,NA,SXT,PN	PEF, CPX	8	0.8
H-4S	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-8S	OFX, AUG, CN, S, CEP,NA,SXT,PN	PEF, CPX	8	0.8
H-9S	OFX, CPX, AUG, CN, S,CEP,NA,SXT,PN	PEF	9	0.9
H-11S	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-12S	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-1S	OFX, PEF, CN, S,CEP,NA, PN	CPX, AUG, SXT	7	0.7
U-2S	OFX, PEF, CN, S,CEP,NA, PN	CPX, AUG, SXT	7	0.7
U-3S	OFX, PEF, CN, S,CEP, PN	CPX, AUG, NA, SXT	6	0.6
U-4S	PEF, AUG, CN, CEP,NA,SXT,PN	OFX, CPX, S	7	0.7
U-5S	OFX, CPX, S,CEP,NA,SXT	PEF, AUG, CN, PN	6	0.6
U-6S	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-7S	OFX, CPX, AUG, CN, S,CEP,NA, PN	PEF, SXT	8	0.8
U-9S	OFX, AUG, NA	PEF, CPX, CN, S,CEP, SXT,PN	3	0.3
U-10S	OFX, CPX, CN, NA, PN	PEF, AUG, S, CEP, SXT	5	0.5
U-11S	OFX, CPX, S, NA, PN	PEF, AUG, CN, CEP, SXT	5	0.5
U-12S	OFX, CPX, CN, NA, PN	PEF, AUG, S, CEP, SXT	5	0.5
H-2M	PEF, AUG, CEP,NA, PN	OFX, CPX, CN, S, SXT	5	0.5
H-9M	PEF, CN, S	OFX, CPX, AUG, CEP,NA,SXT,PN	3	0.3
H-14M	OFX, S,CEP,NA, PN	PEF, CPX, AUG, CN, SXT	5	0.5
H-15M	PEF, SXT,PN	OFX, CPX, AUG, CN, S,CEP,NA	3	0.3
H-16M	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-17M	PEF, AUG, S	OFX, CPX, CN, CEP,NA,SXT,PN	3	0.3
U-1M	AUG, CN, NA,SXT,PN	OFX, PEF, CPX, S,CEP	5	0.5

MDR Bacterial isolates	Resistance pattern	Susceptibility pattern	Number of antibiotics that were resisted	MAR Index
U-2M	OFX, AUG, CN, NA	PEF, CPX, S,CEP, SXT,PN	4	0.4
U-3M	CEP,NA, CN,PN	OFX, PEF, CPX, AUG, S, SXT	4	0.4
U-5M	S,CEP,NA, PN	OFX, PEF, CPX, AUG, CN, SXT	4	0.4
U-8M	PEF, CPX, AUG, CN, S,CEP,NA	OFX, SXT,PN	7	0.7
U-9M	OFX, PEF, CPX, S,CEP,NA	AUG, CN, SXT,PN	6	0.6
U-12M	AUG, NA, PN	OFX, PEF, CPX, CN, S,CEP, SXT	3	0.3
U-16M	AUG, S,CEP,NA	OFX, PEF, CPX, CN, SXT,PN	4	0.4
H-2KU	PEF, CN, S, NA,SXT,PN	OFX, CPX, AUG, CEP	6	0.6
H-3KU	OFX, AUG, CN, S,CEP,NA, PN	PEF, CPX, SXT	7	0.7
H-4KU	CPX, AUG, CEP, PN	OFX, PEF, CN, S, NA,SXT	4	0.4
H-5KU	PEF, AUG, CN, S,CEP,NA,SXT,PN	OFX, CPX	8	0.8
H-6KU	OFX, PEF, CN, S,CEP, SXT	CPX, AUG, NA, PN	6	0.6
H-7KU	OFX, CPX, NA,SXT,PN	PEF, AUG, CN, S,CEP	6	0.6
H-8KU	OFX, CPX, AUG, CN, S,CEP,NA, PN	PEF, SXT	8	0.8
H-10KU	OFX, CN, CEP,NA, PN	PEF, CPX, AUG, S, SXT	5	0.5
H-11KU	OFX, PEF, CPX, CN, S, NA, PN	AUG, CEP, SXT	7	0.7
H-12KU	OFX, CPX, AUG, CEP,NA, PN	PEF, CN, S, SXT	6	0.6
U-1KU	OFX, PEF, AUG, CN, S, NA,SXT,PN	CPX, CEP	8	0.8
U-2KU	OFX, PEF, CPX, AUG, CN, S, NA,SXT,PN	CEP	9	0.9
U-4KU	OFX, CN, S,CEP,NA,SXT,PN	PEF, CPX, AUG	7	0.7
U-5KU	OFX, PEF, AUG, CN, S,CEP,NA,SXT,PN	CPX	9	0.9
U-6KU	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-7KU	OFX, NA,SXT,PN	PEF, CPX, AUG, CN, S, CEP	4	0.4
U-8KU	OFX, CPX, AUG, CN, S,CEP,NA, PN	PEF, SXT	8	0.8
U-10KU	OFX, CN, CEP,NA, PN	PEF, CPX, AUG, S, SXT	5	0.5
U-11KU	OFX, PEF, CPX, CN, S, NA, PN	AUG, CEP, SXT	7	0.7
U-12KU	OFX, CPX, AUG, CEP,NA, PN	PEF, CN, S, SXT	6	0.6

MDR Bacterial isolates	Resistance pattern	Susceptibility pattern	Number of antibiotics that were resisted	MAR Index
H-1BD	CPX, CEP,NA, PN	OFX, PEF, AUG, CN, S, SXT	4	0.4
H-2BD	OFX, AUG, CN, PN	PEF, CPX, S, CEP, NA, SXT	4	0.4
H-3BD	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-4BD	AUG, CEP,NA, PN	OFX, PEF, CPX, CN, S, SXT	4	0.4
H-5BD	S, SXT,PN	OFX, PEF, CPX, AUG, CN, CEP,NA	3	0.3
H-10BD	OFX, CPX, CN, S,PN	PEF, AUG, CEP,NA,SXT	5	0.5
H-11BD	OFX, CN, CEP,NA, PN	PEF, CPX, AUG, S, SXT	5	0.5
H-12BD	OFX, PEF, CN, NA,SXT	CPX, AUG, S, CEP, PN	5	0.5
H-13BD	OFX, PEF, AUG, CN, S,CEP,NA,SXT,PN	CPX	9	0.9
H-16BD	OFX, S, PN	PEF, CPX, AUG, CN, CEP,NA,SXT	3	0.3
H-18BD	OFX, CPX, CN, SXT	PEF, AUG, S, CEP,NA, PN	4	0.4
H-19BD	OFX, PEF,CN, CEP,NA	CPX, AUG, S, SXT,PN	5	0.5
H-20BD	OFX, PEF, CPX, CEP, NA	AUG, CN, S, SXT,PN	4	0.4
U-1BD	OFX, AUG, S,CEP,NA, PN	PEF, CPX, CN, SXT	6	0.6
U-2BD	OFX, AUG, CN, S,CEP, PN	PEF, CPX, NA, SXT	6	0.6
U-3BD	OFX, AUG, S,CEP,NA,SXT,PN	PEF, CPX, CN	7	0.7
U-4BD	OFX, AUG, CN, S,CEP,NA	PEF, CPX, SXT,PN	6	0.6
U-5BD	OFX, CPX, AUG, S,CEP,NA, PN	PEF, CN, SXT	7	0.7
U-6BD	OFX, CN, S,CEP,NA	PEF, CPX, AUG, SXT,PN	5	0.5
U-7BD	OFX, AUG, CN, PN	PEF, CPX, S,CEP, NA,SXT	4	0.4
U-8BD	OFX, AUG, CN, NA,SXT,PN	PEF, CPX, S,CEP	6	0.6
U-9BD	CN, S,CEP,NA, PN	OFX, PEF, CPX, AUG, SXT	5	0.5
U-11BD	S, NA,SXT,PN	OFX, PEF, CPX, AUG, CN, CEP	4	0.4
U-13BD	AUG, CN, NA	OFX, PEF, CPX, S,CEP, SXT,PN	3	0.3
U-14BD	AUG, CN, NA, PN	OFX, PEF, CPX, S,CEP, SXT	4	0.4
U-15BD	AUG,CN, NA, PN	OFX, PEF, CPX, S,CEP, SXT	4	0.4
U-17BD	OFX, AUG, CN, CEP, NA	PEF, CPX, S, SXT,PN	5	0.5

MDR Bacterial isolates	Resistance pattern	Susceptibility pattern	Number of antibiotics that were resisted	MAR Index
U-18BD	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-19BD	OFX, CN, S,CEP,NA, PN	PEF, CPX, AUG, SXT	6	0.6
U-20BD	OFX, CN, CEP,NA	PEF, CPX, AUG, S, SXT,PN	4	0.4
U-21BD	OFX, AU,S,CEP,NA,SXT,PN	PEF,CPX,CN	7	0.7
U-22BD	OFX,CN,NA,PN	PEF,CPX,AU,S,CEP,SXT	4	0.4
U-23BD	CPX,S,PN	OFX,PEF,AU,CN,CEP,NA,SXT	3	0.3
H-1AG	CPX, AUG, CN, S,CEP,NA,SXT,PN	OFX, PEF	8	0.8
H-3AG	PEF, CPX, AUG, CN, S, SXT,PN	OFX, CEP,NA	7	0.7
H-4AG	AUG, CN, S, SXT,PN	OFX, PEF, CPX, CEP,NA	5	0.5
H-5AG	PEF, CPX, AUG, CN, S,CEP,NA,SXT	OFX, PN	8	0.8
H-7AG	PEF, CPX, AUG, CN, SXT	OFX, S,CEP,NA, PN	5	0.5
H-10AG	PEF, CPX, AUG, CN, S,CEP,NA,SXT	OFX, PN	8	0.8
H-14AG	PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	OFX	9	0.9
H-19AG	CPX, AUG, CN, S, NA,SXT,PN	OFX, PEF, CEP	7	0.7
H-20AG	CPX, AUG, CN, S,CEP,NA,SXT,PN	OFX, PEF	8	0.8
U-1AG	PEF, CPX, AUG, S,CEP,NA,SXT,PN	OFX, CN	8	0.8
U-2AG	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT	PN	9	0.9
U-3AG	OFX, PEF, AUG, CN, S,CEP,NA,SXT,PN	CPX	9	0.9
U-4AG	PEF, CPX, AUG, CN, S,CEP,NA,SXT	OFX, PN	8	0.8
U-5AG	OFX, PEF, CPX, AUG, CN, S, NA,SXT	CEP, PN	8	0.8
U-6AG	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-8AG	OFX, PEF, AUG, CN, S,CEP,NA,SXT,PN	CPX	9	0.9
U-9AG	OFX, PEF, CPX, AUG, S,CEP,NA,SXT,PN	CN	9	0.9
U-10AG	OFX, PEF, CPX, AUG, S,CEP,NA,SXT,PN	CN	9	0.9
U-11AG	PEF, CPX, CN, S,CEP,NA,SXT,PN	OFX, AUG	8	0.8
U-12AG	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-14AG	CPX, AUG, CN, CEP,NA,SXT,PN	OFX, PEF, S	7	0.7
U-15AG	PEF, CPX, S,CEP,NA,SXT,PN	OFX, AUG, CN	7	0.7
U-16AG	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-17AG	CPX, AUG, S,CEP,NA,SXT,PN	OFX, PEF, CN	7	0.7
U-18AG	PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	OFX	9	0.9

MDR Bacterial isolates	Resistance pattern	Susceptibility pattern	Number of antibiotics that were resisted	MAR Index
U-19AG	OFX, PEF, AUG, S,CEP,NA,SXT,PN	CPX, CN	8	0.8
U-20AG	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT	PN	9	0.9
U-21AG	OFX, PEF, CPX, AUG, CN, CEP,NA,SXT	S, PN	8	0.8
U-22AG	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-IL	OFX, PEF, AUG, CN, S,CEP,NA,SXT,PN	CPX	9	0.9
H-2L	OFX, PEF, AUG, CN, CEP,NA, PN	CPX, S, SXT	7	0.7
H-3L	OFX, AUG, CN, S,CEP,NA, PN	PEF, CPX, SXT	7	0.7
H-4L	OFX, PEF, CPX, AUG, CN, S,CEP,NA, PN	SXT	9	0.9
H-5L	OFX, AUG, CN, S,CEP, SXT,PN	PEF, CPX, NA	7	0.7
H-6L	PEF, AUG, CN, CEP,NA, PN	OFX, CPX, S, SXT	6	0.6
H-7L	PEF, S,CEP, PN	OFX, CPX, AUG, CN, NA,SXT	4	0.4
H-8L	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-9L	OFX, AUG, CN, S,CEP,NA,SXT	PEF, CPX, PN	7	0.7
H-10L	OFX, CN, S,CEP,NA,SXT,PN	PEF, CPX, AUG	7	0.7
H-11L	OFX, S,CEP,NA, PN	PEF, CPX, AUG, CN, SXT	5	0.5
H-12L	OFX, PEF, CN, CEP,NA, PN	CPX, AUG, S, SXT	6	0.6
H-I3L	OFX, AUG, CN, S,CEP,NA,SXT,PN	PEF, CPX	8	0.8
H-14L	OFX, AUG, S,CEP,NA, PN	PEF, CPX, CN, SXT	6	0.6
H-15L	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-16L	OFX, PEF, CPX, AUG, CN, S,CEP,NA, PN	SXT	9	0.9
H-17L	OFX, CPX, AUG, S,CEP,NA,SXT,PN	PEF, CN	8	0.8
H-18L	OFX, PEF, CPX, AUG, CN, S,CEP,NA, PN	SXT	9	0.9
U-3L	AUG, CN, S,CEP,NA,SXT,PN	OFX, PEF, CPX	7	0.7
U-6L	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT	PN	9	0.9
U-7L	AUG, CN, S,CEP,NA,SXT,PN	OFX, PEF, CPX	7	0.7
U-8L	AUG, CN, S, SXT,PN	OFX, PEF, CPX, CEP,NA	5	0.5
U-10L	CN, S,CEP,NA, PN	OFX, PEF, CPX, AUG, SXT	5	0.5
U-14L	CN, CEP,NA,SXT	OFX, PEF, CPX, AUG, S, PN	4	0.4
U-15L	CN,CEP,NA, PN	OFX, PEF, CPX, AUG, S, SXT	4	0.4
U-17L	S,CEP,NA,SXT,PN	OFX, PEF, CPX, AUG, CN	5	0.5
U-25L	OFX, PEF, AUG, CN, S,CEP,NA, PN	CPX, SXT	8	0.8

MDR Bacterial isolates	Resistance pattern	Susceptibility pattern	Number of antibiotics that were resisted	MAR Index
U-28L	OFX, CPX, AUG, CN, S,CEP,NA,SXT,PN	PEF	9	0.9
U-29L	OFX, CN, NA,SXT	PEF, CPX, AUG, S,CEP, PN	4	0.4
U-32L	CN, CEP,NA	OFX, PEF, CPX, AUG, S, SXT,PN	3	0.3
U-34L	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-36L	OFX, CPX, CN, S,CEP,NA,SXT,PN	PEF, AUG	8	0.8
U-37L	OFX, CPX, AUG, CN, S,CEP,NA,SXT,PN	PEF	9	0.9
U-38L	OFX, CPX, AUG, CN, CEP,NA,SXT,PN	PEF, S	8	0.8
U-39L	OFX, CPX, CN, S,CEP,NA, PN	PEF, AUG, SXT	7	0.7
U-40L	OFX, AUG, CN, S,CEP,NA, PN	PEF, CPX, SXT	7	0.7
H-2N	PEF, AUG, CN	OFX,CPX,S,CEP,NA,SXT,PN	3	0.3
H-8N	OFX, PEF, AUG, CN, S, NA,SXT,PN	CPX, CEP	8	0.8
H-12N	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-17N	OFX, PEF, AUG, CN, S,CEP,NA, PN	CPX, SXT	8	0.8
H-18N	PEF, CPX, S,CEP,NA,SXT,PN	OFX, AUG, CN	7	0.7
H-19N	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-20N	PEF, CPX, CN, S, SXT	OFX, AUG, CEP,NA, PN	5	0.5
H-22N	PEF, AUG, S, NA,SXT	OFX, CPX, CN, CEP, PN	5	0.5
U-1N	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-3N	PEF, CPX, AUG, CN, S,CEP,NA,SXT	OFX, PN	8	0.8
U-4N	OFX, PEF, CPX, AUG, CN, CEP,NA,SXT,PN	S	9	0.9
U-6N	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-7N	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-8N	OFX, PEF, CPX, AUG, CN, CEP,NA,SXT	S, PN	8	0.8
U-9N	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-10N	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-12N	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-14N	OFX, PEF, CPX, AUG, CN, S, NA,SXT,PN	CEP	9	0.9
U-15N	CPX, AUG, CEP,NA,SXT,PN	OFX, PEF, CN, S	6	0.6
U-16N	OFX, PEF, CPX, AUG, CN, CEP,NA, PN	S, SXT	8	0.8

MDR Bacterial isolates	Resistance pattern	Susceptibility pattern	Number of antibiotics that were resisted	MAR Index
U-18N	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-20N	PEF, CPX, AUG, CN, CEP,NA, PN	OFX, S, SXT	7	0.7
U-21N	PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	OFX	9	0.9
U-22N	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-1KN	AUG, S, NA, PN	OFX, PEF, CPX, CN, CEP, SXT	4	0.4
H-2KN	AUG, CN, CEP,NA	OFX, PEF, CPX, S, SXT,PN	4	0.4
H-3KN	PEF, AUG, S	OFX, CPX, CN, CEP,NA,SXT,PN	3	0.3
H-5KN	CN, S,CEP,NA,SXT	OFX, PEF, CPX, AUG, PN	5	0.5
H-27KN	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-31KN	AUG, CN, CEP,NA	OFX, PEF, CPX, S, SXT,PN	4	0.4
H-39KN	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-42KN	OFX, PEF, CPX, AUG, CN, S, SXT	CEP,NA, PN	7	0.7
U-27KN	PEF, CPX, AUG, CEP, SXT	OFX, CN, S, NA, PN	5	0.5
U-28KN	PEF, CPX, AUG, CN, SXT	OFX, S, CEP, NA, PN	5	0.5
U-29KN	OFX, PEF, CN, CEP,NA	CPX, AUG, S, SXT,PN	5	0.5
U-34KN	OFX, PEF, CPX, AUG, CN, S	CEP,NA,SXT,PN	6	0.6
U-36KN	AUG, CN, S,CEP, SXT,PN	OFX, PEF, CPX, NA	6	0.6
U-38KN	OFX, PEF, CPX, CN, S,CEP,NA,SXT,PN	AUG	9	0.9
U-39KN	S, NA, PN	OFX, PEF, CPX, AUG, CN, CEP, SXT	3	0.3
U-40KN	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-42KN	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-43KN	CPX, CN, S, SXT	OFX, PEF, AUG, CEP,NA,PN	4	0.4
U-46KN	PEF, AUG, S, NA, PN	OFX, CPX, CN, CEP, SXT	5	0.5
U-47KN	S,CEP,NA,SXT,PN	OFX, PEF, CPX, AUG, CN	5	0.5
U-52KN	PEF, AUG, S,CEP	OFX, CPX, CN, NA,SXT,PN	4	0.4
U-56KN	CPX, AUG, CEP, SXT	OFX, PEF, CN, S, NA, PN	4	0.4
U-57KN	OFX, PEF, S,CEP	CPX, AUG, CN, NA,SXT,PN	4	0.4

MDR Bacterial isolates	Resistance pattern	Susceptibility pattern	Number of antibiotics that were resisted	MAR Index
U-59KN	PEF, CN, CEP,NA,SXT	OFX, CPX, AUG, S, PN	5	0.5
U-63KN	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-64KN	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT	PN	9	0.9
U-65KN	PEF, CN, SXT,PN	OFX, CPX, AUG, S,CEP,NA	4	0.4
U-66KN	OFX, PEF, CPX, AUG, CN	S,CEP,NA,SXT,PN	5	0.5
U-67KN	CPX, AUG, CN, SXT	OFX, PEF, S,CEP,NA, PN	4	0.4
U-68KN	PEF, CPX, AUG, CN, S, NA,SXT	OFX, CEP, PN	7	0.7
U-71KN	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-73KN	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-75KN	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-1WU	OFX, AUG, SXT,PN	PEF, CPX, CN, S, CEP, NA	4	0.4
H-7WU	OFX, AUG, S, NA, PN	PEF, CPX, CN, CEP, SXT	5	0.5
H-8WU	OFX, PEF, AUG, CN, S, NA,SXT,PN	CPX,CEP	8	0.8
H-11WU	OFX, AUG, CN, CEP,NA,SXT,PN	PEF, CPX, S	7	0.7
H-13WU	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
H-15WU	OFX, PEF, AUG, CEP,NA, PN	CPX, CN, S, SXT	6	0.6
H-16WU	OFX, AUG, S	PEF, CPX, CN, CEP,NA,SXT,PN	3	
H-21WU	PEF, AUG, NA,SXT,PN	OFX, CPX, CN, S,CEP	5	0.5
H-22WU	OFX, AUG, S,CEP,NA,SXT	PEF, CPX, CN, PN	6	0.6
H-28WU	OFX, PEF, AUG, CEP,NA,SXT,PN	CPX, CN, S	7	0.7
U-2WU	OFX, PEF, AUG, S, NA, PN	CPX, CN, CEP, SXT	6	0.6
U-6WU	OFX, PEF, AUG, CN, S,CEP,NA, PN	CPX, SXT	8	0.8
U-10WU	OFX, AUG, CN, NA	PEF, CPX, S, CEP, SXT,PN	4	0.4
U-12WU	OFX, PEF, AUG,CN, CEP,NA	CPX, S, SXT,PN	5	0.5
U-15WU	OFX, PEF, CPX, AUG, CN, S,CEP,NA,SXT,PN	_	10	1
U-18WU	OFX, PEF, AUG, CN, CEP,NA	CPX, S, SXT,PN	6	0.6
U-22WU	OFX, PEF, AUG,CN, PN	CPX, S, CEP,NA,SXT	5	0.5
U-24WU	PEF, AUG,CN, NA, PN	OFX, CPX, S,CEP, SXT	5	0.5
U-26WU	OFX, PEF, AUG, CN, CEP,NA,SXT	CPX, S, PN	7	0.7
U-28WU	PEF, AUG, CN, S, NA,SXT,PN	OFX, CPX, CEP	7	0.7

Key: OFX:Ofloxacin;PEF:Perfloxacin;CPX:Ciprofloxacin;NA:Nalidixicacid;CN:Gentamicin;ST:Streptomycin;PN:Ampicillin;Cep:Cephalexin;AU:Augmentin;

SXT:Sulfamethoxazole

4.1.8 Resistance profile of multidrug resistant bacteria to various classes of antibiotics in each General Hospital

High rate of resistance was observed for Nalidixic acid in 5 hospitals (namely General Hospital Minna, Kuta, Bida, Agaie and Lapai) compared to other fluoroquinolones; High rate of resistance was observed for Gentamicin in 7 hospitals (namely General Hospital Suleja, Kuta, Bida, Lapai, Nasko, Kontagora and Wushishi) compared to other aminoglycosides and high rate of resistance was also observed for Ampicillin in 4 hospitals (namely General Hospital Suleja, Kuta, Bida, and Wushishi) compared to other betalactams (as seen in Table 4.45).

Table 4.45: Resistance Profile of Multidrug Resistant Bacteria to Various Classes of Antibiotics in each Hospital

Hospitals	MDR	OFX(%)	PEF(%)	CPX(%)	AU(%)	CN(%)	S(%)	CEP(%)	NA(%)	SXT(%)	PN(%)
G.H.S	19	18(94.7)	8(42.1)	10(52.6)	12(63.2)	16(84.2)	15(78.9)	15(78.9)	18(94.7)	11(57.9)	17(89.5)
G.H.M	14	4(28.6)	7(50)	3(21.4)	8(57.1)	6(42.9)	8(57.1)	8(57.1)	11(78.6)	3(21.4)	8(57.1)
G.H.KU	20	17(85)	9(45)	10(50)	11(55)	15(75)	13(65)	13(65)	18(90)	10(50)	19(95)
G.H.BD	33	24(72.7)	6(18.2)	8(24.2)	17(51.5)	21(63.6)	17(51.6)	19(57.6)	25(75.8)	10(30.3)	24(72.7)
G.H.AG	29	13(44.8)	23(79.3)	25(86.2)	27(93.1)	236(79.3)	26(89.7)	24(82.8)	26(89.7)	29(100)	21(72.4)
G.H.L	36	26(72.2)	13(36.1)	13(36.1)	24(66.7)	31(86.1)	28(77.8)	34(94.4)	33(91.7)	20(55.6)	31(86.1)
G.H.N	24	16(66.7)	23(95.8)	20(83.3)	22(91.7)	21(87.5)	18(75)	19(79.2)	22(91.7)	20(83.3)	19(79.2)
G.H.KN	33	15(45.5)	23(69.7)	19(57.6)	24(72.7)	24(72.7)	23(69.7)	21(63.6)	20(60.6)	22(66.7)	15(45.5)
G.H.WU	20	17(85)	14 (70)	2(10)	20(100)	12(60)	9(45)	10(50)	17(85)	10(50)	15(70)

Key: G.H.S (General hospital Suleja); G.H.M (General hospital Minna); G.H.KU (General hospital Kuta); G.H.B (General hospital Bida); G.H.AG (General hospital Agaie); G.H.L (General hospital Lapai); G.H.N (General hospital Nasko); G.H.KN (General hospital Kontagora); G.H.WU (General Hospital Wushishi)

4.1.9 Graphical representation of resistance profile of Multidrug Resistant Bacteria to various Classes of Antibiotics in each Hospital

The study revealed that all isolates from five (5) General Hospitals exhibited high resistance to Nalidixic acid, while isolates in two (2) General Hospitals exhibited high resistance to Perfloxacin as seen in Figure 4.2. High resistance to Gentamicin was observed in seven General Hospitals as seen in Figure 4.3.

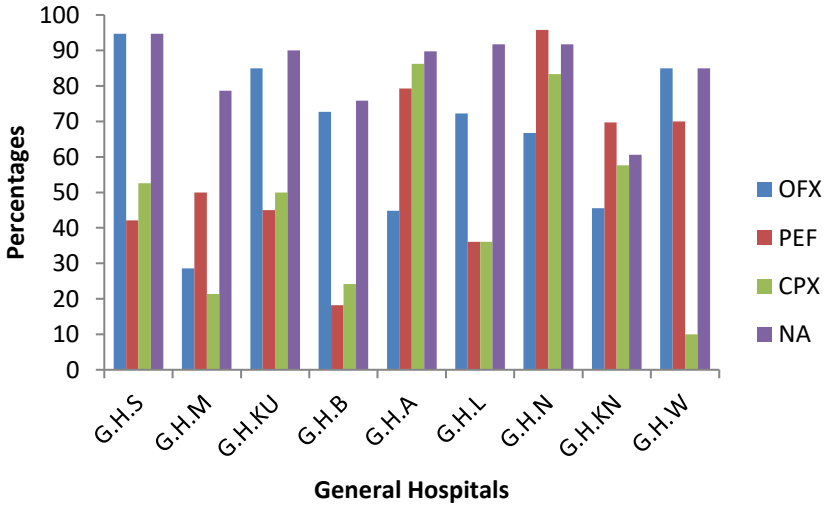

Figure 4.2: Antibiotic Resistance Profile Multidrug Resistant Bacteria to Quinolones in various General Hospitals

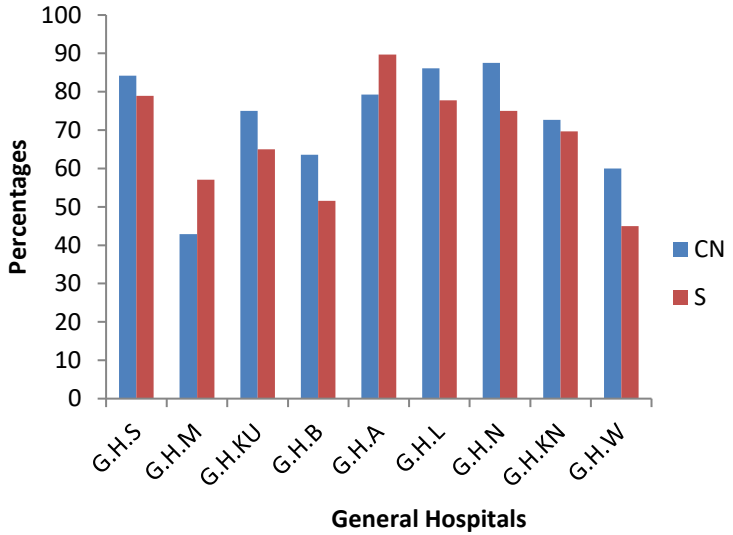

Figure 4.3: Antibiotic Resistance Profile of Multidrug Resistant Bacteria to Aminoglycosides in various General Hospitals

Key: OFX: Ofloxacin; PEF: Perfloxacin; CPX: Ciprofloxacin; NA: Nalidixic acid; CN: Gentamicin; S: Streptomycin; PN: Ampicillin; Cep: Cephalexin; AU: Augmentin; SXT: Sulfamethoxazole.

This study also revealed that urogenital bacterial pathogens were resistant to Ampicillin in four different General Hospitals (as seen in Figure 4.4). This study also revealed that bacterial uropathogens isolated from PID patients in eight General Hospitals were resistant to Augmentin as seen in Figure 4.5

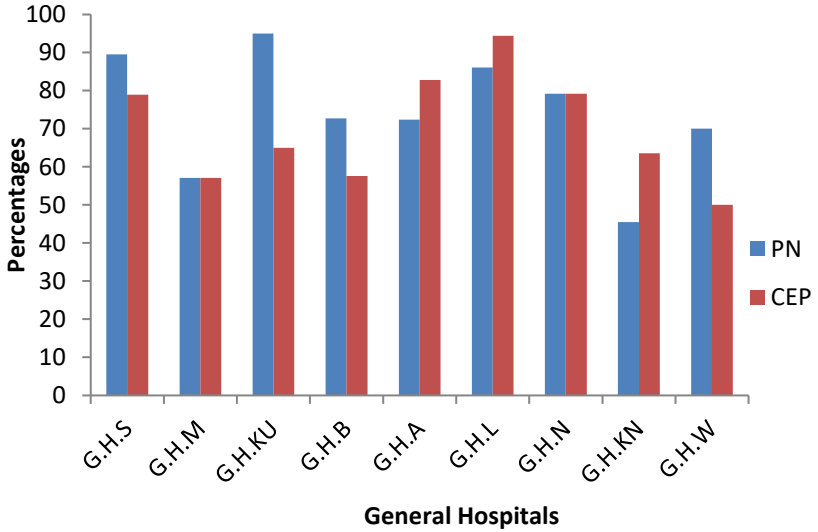

Figure 4.4: Antibiotic Resistance Profile Multidrug Resistant Bacteria to Betalactams in various General Hospitals

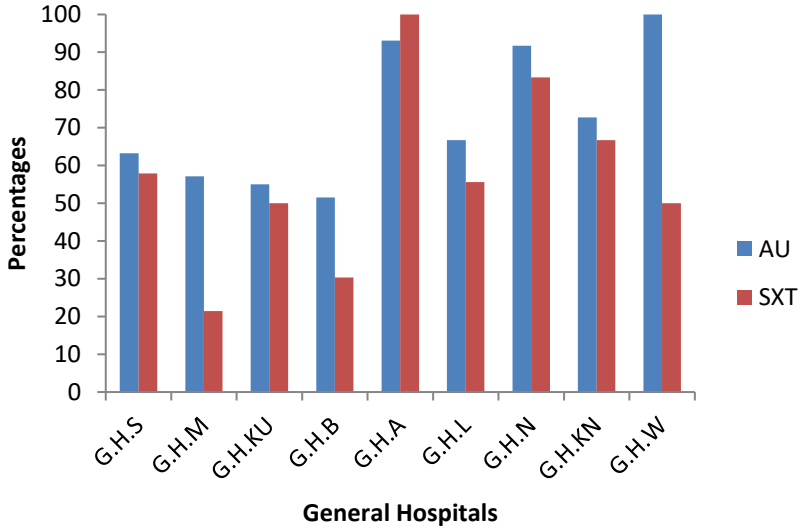

Figure 4.5: Antibiotic Resistance Profile of Multidrug Resistant Bacteria to other Antibiotics in various General Hospitals

Key : OFX: Ofloxacin; PEF : Perfloxacin; CPX: Ciprofloxacin; NA: Nalidixic acid; CN: Gentamicin; S: Streptomycin; PN: Ampicillin; Cep: Cephalexin; AU: Augmentin;SXT: Sulfamethoxazole; G.H.S (General hospital Suleja); G.H.M (General hospital Minna); G.H.KU (General hospital Kuta); G.H.B (General hospital Bida); G.H.AG (General hospital Agaie); G.H.L (General hospital Lapai); G.H.N (General hospital Nasko); G.H.KN (General hospital Kontagora); G.H.WU (General Hospital Wushishi)

4.1.10 Occurrence of multi drug resistant bacteria in ECS and urine samples in each General Hospital

The occurrence of multidrug resistant bacteria associated with various samples in each General Hospital in Niger State, is shown in Figure 4.6. The result revealed that in six general hospitals, there were more multidrug resistant bacteria in urine samples than in the endocervical swabs.

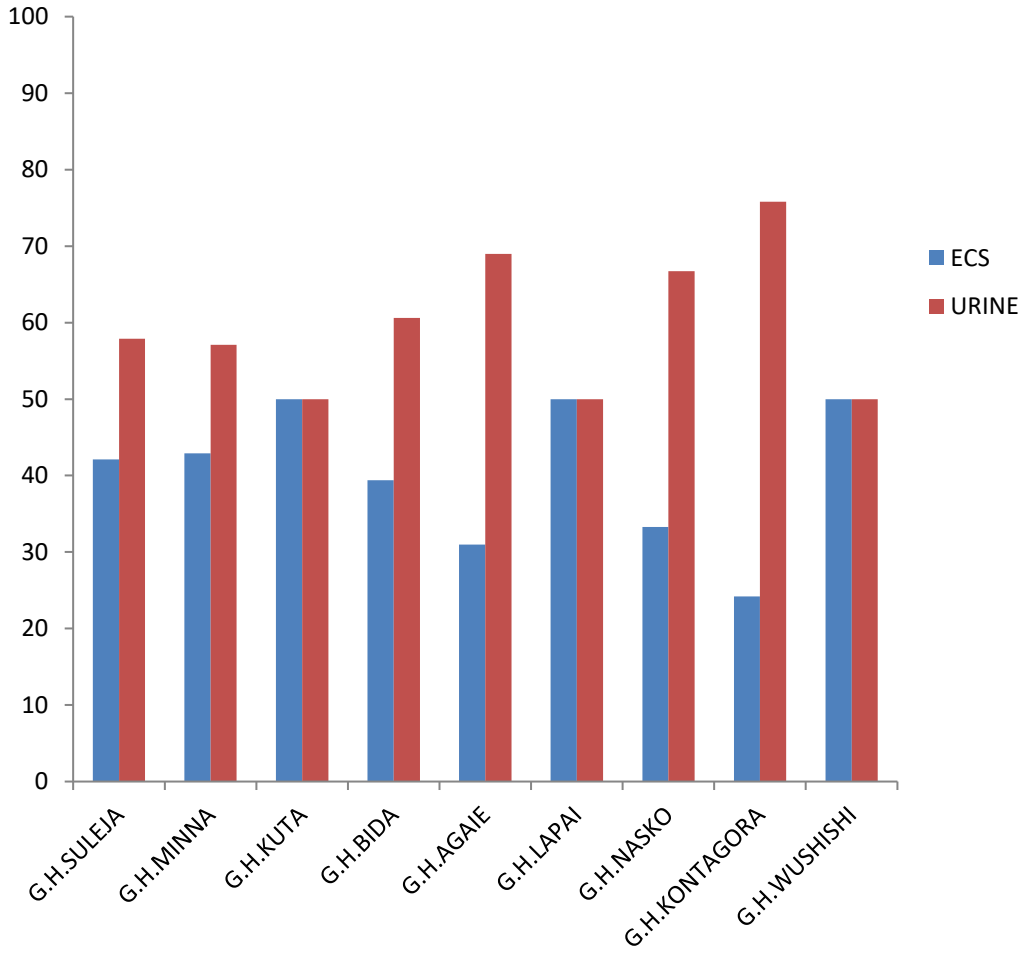

Figure 4.6: Percentages of Multidrug Resistant Isolates in ECS and Urine in each General Hospital

4.1.11 Prevalence of multidrug resistant bacteria in endocervical swabs and urine samples of patients with pelvic inflammatory disease

Out of the 228 multidrug resistant bacteria isolated, 7.8% and 5.1% of the isolated bacteria from ECS and Urine were resistant to three antibiotics, while 81.1% and 79.7% of the isolated bacteria from ECS and Urine were resistant to five or more antibiotics as presented in Table 4.46 and Table 4.47 respectively.

Table 4.46: Prevalence of Multidrug Resistant (MDR) Bacteria in Endocervical Swabs of Patients with Pelvic Inflammatory Disease

ECS	Total number of isolates	Number of MDR isolates	MDR isolates resistant to 3 antibiotics	MDR isolates resistant to 4 antibiotics	MDR isolates resistant to 5 or more antibiotics	Total % prev.
Klebsiella pneumoniae	70	35	3	2	30	
% Prev		50	8.6	5.7	85.7	100
Escherichia coli	72	31	1	3	27	
% Prev		43.1	3.2	9.7	87.1	100
Salmonella Typhi	69	24	3	5	16	
% Prev		34.8	12.5	20.8	66.7	100
Proteus vulgaris	39	0	0	0	0	
% Prev			0	0	0	0
Staphylococcus aureus	38	0	0	0	0	
% Prev			0	0	0	0
Streptococcus pyogenes	32	0	0	0	0	
% Prev			0	0	0	0
Total	320	90	7	10	73	
% Prev			7.8	11.1	81.1	100

Key: MDR: Multi drug resistant, ECS: Endocervical swab

Table 4.47: Prevalence of Multidrug Resistant (MDR) Bacteria in Urine of Patients with Pelvic Inflammatory Disease

Urine	Total number of isolates	Number of MDR isolates	MDR isolates resistant to 3 antibiotics	MDR isolates resistant to 4 antibiotics	MDR isolates resistant to 5 or more antibiotics	Total % prev.
Klebsiella pneumoniae	85	52	3	9	40	
% Prev		61.2	5.8	17.3	76.9	100
Escherichia coli	98	48	2	4	42	
% Prev		49.0	4.2	8.3	87.5	100
Salmonella Typhi	83	33	2	8	23	
% Prev		39.8	6.1	24.2	69.7	100
Proteus vulgaris	54	5	0	0	5	
% Prev		9.3	0	0	100	100
Staphylococcus aureus	42	0	0	0	0	
% Prev			0	0	0	0
Streptococcus pyogenes	38	0	0	0	0	
% Prev			0	0	0	0
Total	400	138	7	21	110	
% Prev			5.1	15.2	79.7	100

Key: MDR: Multi drug resistant

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Table 4.48: Prevalence of Multidrug Resistant (MDR) Bacteria in Both ECS and Urine of Patients with Pelvic Inflammatory Disease

ECS and Urine	Total number of isolates	Number of MDR isolates	MDR isolates resistant to 3 antibiotics	MDR isolates resistant to 4 antibiotics	MDR isolates resistant to 5 or more antibiotics	Total % prev.
Klebsiella pneumoniae	155	87	6	11	70	
% Prev		56.1	6.9	12.6	80.5	100
Escherichia coli	170	79	3	7	69	
% Prev		46.4	3.8	8.9	87.3	100
Salmonella Typhi	152	57	5	13	39	
% Prev		37.5	8.8	22.8	68.4	100
Proteus vulgaris	93	5	0	0	5	
% Prev		5.4	0	0	100	100
Staphylococcus aureus	80	0	0	0	0	
% Prev			0	0	0	0
Streptococcus pyogenes	70	0	0	0	0	
% Prev			0	0	0	0
Total	720	228	14	31	183	
% Prev			6.1	13.6	80.3	100

Key: MDR: Multi drug resistant, ECS: Endocervical swab

4.1.12 Frequency of occurrence of isolates associated with the production of different inactivating enzymes

The production of each enzyme among the resistant bacteria isolated from ECS and urine was significantly different, while there was no significant difference in the production of different enzymes by each resistant bacterium. The result revealed that *Klebsiella pneumoniae* produced more enzymes than other isolated bacteria as seen in Table 4.49 and Table 4.50 respectively.

Table 4.49: Frequency of Occurrence of Isolates Producing Different Inactivating Enzymes in Endocervical Swab

	Bacterial Isolates (Number of MDR Isolates)			
Enzymes	***Escherichia coli* (31)**	***Klebsiella pneumoniae* (35)**	***Salmonella* Typhi (24)**	***Proteus vulgaris* (0)**
ESBL	$25.00\pm3.00_b^a$	$28.00\pm3.03_b^b$	$22.00\pm1.23_b^a$	$0.00\pm0.00_a^a$
CPnase	$22.00\pm2.00_b^a$	$24.00\pm2.00_b^{ab}$	$16.00\pm1.80_b^a$	$0.00\pm0.00_a^a$
ESBL+CPnase	$16.00\pm0.00_b^a$	$17.00\pm1.75_b^a$	$13.00\pm1.00_b^a$	$0.00\pm0.00_a^a$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

Table 4.50: Frequency of Occurrence of Isolates Producing Different Inactivating Enzymes in Urine

	Bacterial Isolates (Number of MDR Isolates)			
Enzymes	***Escherichia coli* (48)**	***Klebsiella pneumoniae* (52)**	***Salmonella* Typhi (33)**	***Proteus vulgaris* (5)**
ESBL	$40.00\pm2.00_c^a$	$49.00\pm1.80_d^b$	$25.00\pm3.02_b^a$	$2.00\pm0.00_b^{ab}$
CPnase	$37.00\pm2.11_c^a$	$44.00\pm0.00_d^b$	$26.00\pm1.04_b^a$	$4.00\pm0.58_b^b$
ESBL+CPnase	$28.00\pm3.67_c^a$	$36.00\pm1.24_d^a$	$19.00\pm2.00_b^a$	$1.00\pm0.00_b^a$

Values are represented as Mean±Standard error of mean of triplicate determinations. Values with different superscript across the column are significantly different ($p<0.05$) while values with different subscripts along the row are significantly different ($p<0.05$)

KEY: ESBL : Extended specteum beta lactamase; CPnase : Carbapenemase; ESBL+ CPnase: Extended specteum beta lactamase and Carbapenemase

4.1.13 Enzymes production among isolates from different urogenital samples

The production of each enzyme among different urogenital samples was significantly different. The result revealed that resistant bacteria from urine produced more of the enzymes compared to resistant bacteria isolated from ECS, as seen in Table 4.51- 4.53

Table 4.51: Production of ESBL in Urine and ECS

Enzymes ESBL	Urine	ECS
E. coli	40.00±2.00[b]	25.00±3.00[a]
K.pneumoniae	49.00±1.80[b]	28.00±3.03[a]
S.typhi	25.00±3.02[a]	22.00±1.23[a]
P.vulgaris	2.00±0.00[a]	0.00±0.00[a]

Table 4.52: Production of CPnase in Urine and ECS

Enzymes CPnase	Urine	ECS
E. coli	37.00±2.11[b]	22.00±2.00[a]
K.pneumoniae	44.00±0.00[b]	23.00±2.00[a]
S.typhi	26.00±1.04[b]	16.00±1.80[a]
P.vulgaris	2.00±0.58[a]	0.00±0.00[a]

Table 4.53: Production of ESBL+ Cpnase in Urine and ECS

Enzymes ESBL+CPnase	Urine	ECS
E. coli	28.00±3.67[b]	16.00±0.00[a]
K.pneumoniae	36.00±1.24[b]	16.00±1.75[a]
S.typhi	19.00±2.00[b]	13.00±1.00[a]
P.vulgaris	1.00±0.00[a]	0.00±0.00[a]

KEY: ESBL : Extended specteum beta lactamase; CPnase : Carbapenemase; ESBL+ CPnase: Extended specteum beta lactamase and Carbapenemase; ECS: Endocervical swab

4.1.14 Bacterial isolates that completely resisted all antibiotics

Out of the 228 resistant bacteria isolated from both ECS and urine, 35 bacteria isolates were completely resistant to all antibiotics as seen in Table 4.54.

Table 4.54: Bacteria completely resistant to 10 different antibiotics

S/N	Isolate code	Name of bacterial isolates	S/N	Isolate code	Name of bacterial isolates
1	H-4S	*Escherichia coli*	30	U-63KN	*Klebsiella pneumoniae*
2	H-11S	*Klebsiella pneumoniae*	31	U-71KN	*Escherichia coli*
3	H-12S	*Klebsiella pneumoniae*	32	U-73KN	*Escherichia coli*
4	U-6S	*Escherichia coli*	33	U-75KN	*Klebsiella pneumoniae*
5	U-16M	*Escherichia coli*	34	H-15W	*Salmonella typhi*
6	U-6K	*Escherichia coli*	35	U-13W	*Escherichia coli*
7	H-3BD	*Escherichia coli*			
8	U-18BD	*Salmonella typhi*			
9	U-6AG	*Proteus vulgaris*			
10	U-12AG	*Escherichia coli*			
11	U-16AG	*Salmonella typhi*			
12	U-22AG	*Salmonella typhi*			
13	H-8L	*Klebsiella pneumoniae*			
14	H-15L	*Salmonella typhi*			
15	U-34L	*Escherichia coli*			
16	H-12N	*Salmonella typhi*			
17	H-19N	*Salmonella typhi*			
18	U-1N	*Klebsiella pneumoniae*			
19	U-6N	*Proteus vulgaris*			
20	U-7N	*Salmonella typhi*			
21	U-9N	*Proteus vulgaris*			
22	U-10N	*Proteus vulgaris*			
23	U-12N	*Salmonella typhi*			
24	U-18N	*Klebsiella pneumoniae*			
25	U-22N	*Escherichia coli*			
26	H-27KN	*Klebsiella pneumoniae*			
27	H-39KN	*Escherichia coli*			
28	U-40KN	*Klebsiella pneumoniae*			
29	U-42KN	*Salmonella typhi*			

4.1.15 Amplification of the 16s rRNA region in the selected multidrug resistant bacterial isolates

Plate I presents the electrograph of various multidrug resistant bacterial isolates amplified gene (lane A to L). The DNA of the isolates amplified at 1500bp indicated pure bacteria isolates. Lane MK represents the molecular marker (ladder) (Plate I).

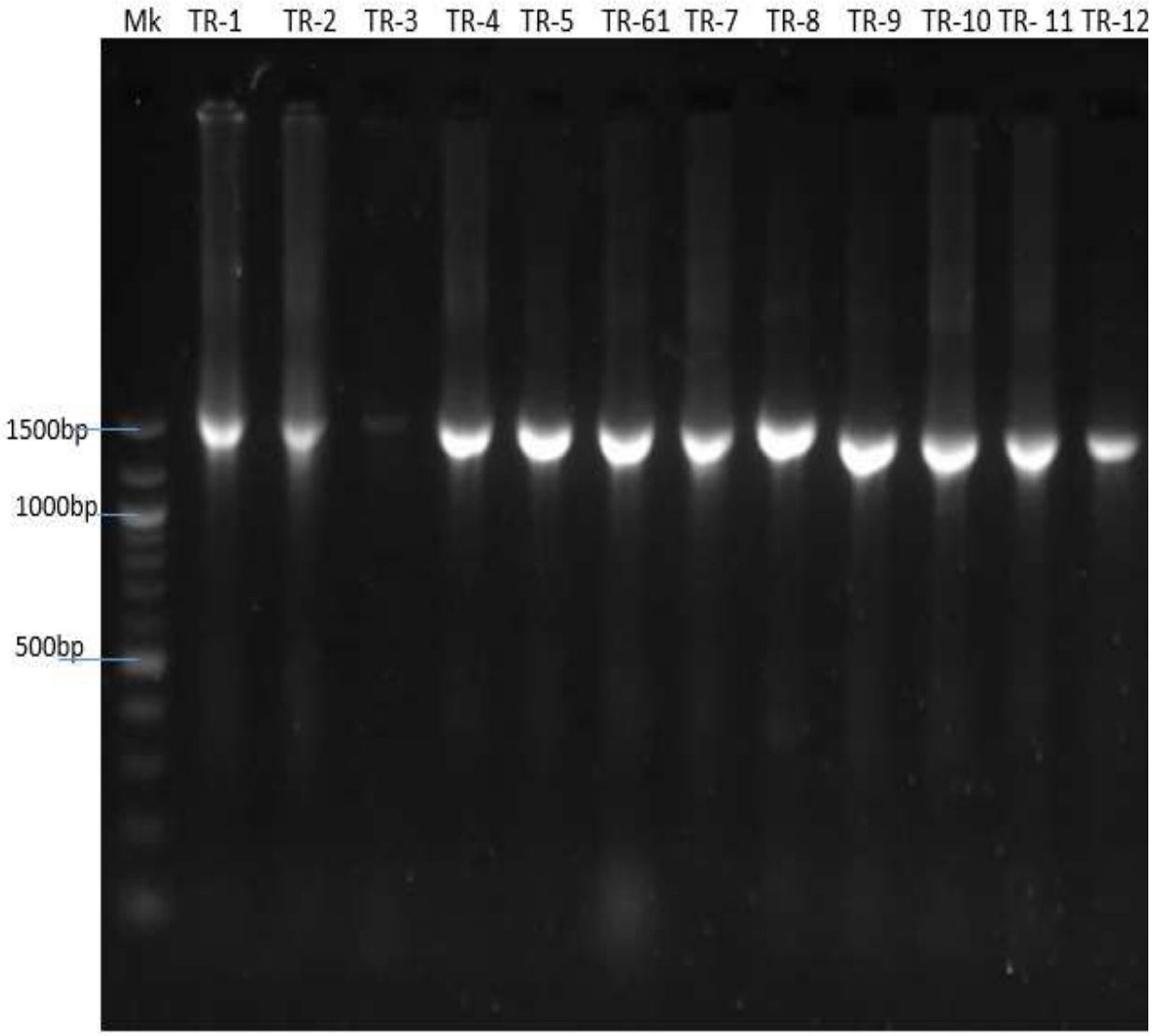

Plate I: Agarose Gel Electrophoresis Indicating the Positive Amplification of the 16s rRNA Gene Fragment used for Bacteria Identification

The presence of a 1500bp indicates positive amplification.

Key: TR1= H-4S; TR 2= U-6S; TR 3= H-11S; TR 4= U-75KN; TR 5= H-15N; TR 6= U-18BD; TR 7= U-6AG; TR 8= U-9N; TR 9= U-16M; TR 10= U-1N; TR 11= H-15L; TR 12= U-6N

Table 4.55: BLAST Pairwise Alignment of Twelve Amplicons Sequenced Against Reference Strains

Sample ID	Description	Max Score	Total Score	Query Cover	E value	Per. Ident	Accession
TR-1	*Escherichia coli* strain SCU-175 chromosome, complete genome	2643	15223	100%	0	99.75%	CP054379.1
TR-2	*Escherichia coli* strain SCU-175 chromosome, complete genome	2634	18374	100%	0	99.59%	CP054379.1
TR-9	*Escherichia coli* strain LWY24 chromosome, complete genome	2177	15214	99%	0	99.84%	CP054556.1
TR-5	*Salmonella enterica* strain R19_2839 chromosome, complete genome	2645	18515	99%	0	99.66%	CP046429.1
TR-6	*Salmonella enterica* subsp. enterica strain LHST_2018 chromosome, complete genome	2647	18534	99%	0	99.66%	CP052767.1
TR-11	*Salmonella enterica* strain R19_2839 chromosome, complete genome	2628	18395	99%	0	99.46%	CP046429.1
TR-3	*Klebsiella pneumoniae* strain E16KP0210 chromosome, complete genome	2567	20512	99%	0	99.25%	CP052295.1
TR-4	*Klebsiella pneumoniae* strain E16KP0210 chromosome, complete genome	2671	21342	99%	0	99.80%	CP052295.1
TR-10	*Klebsiella pneumoniae* strain GXNN3 16S ribosomal RNA gene, partial sequence	2590	2590	99%	0	99.66%	KU936064.1
TR-7	*Proteus vulgaris* strain FCC64 16S ribosomal RNA gene, partial sequence	2615	2615	99%	0	99.79%	JF772095.1
TR-8	*Proteus vulgaris* strain MAR 16S ribosomal RNA gene, partial sequence	2516	2516	99%	0	99.51%	MK572636.1
TR-12	*Proteus vulgaris* strain FDAARGOS_556 chromosome, complete genome	2719	18989	100%	0	99.61%	CP033736.1

4.1.16 Molecular detection of various genes coding for various bacterial resistance

The molecular analysis of various bacterial resistance genes in twelve (12) bacterial isolates is shown in Plate II – XI. These results revealed electrographs of PCR products of various resistant genes.

Molecular detection of *blaTEM*-coding Genes

The electrograph (plate II) shows that out of 12 resistant bacteria, 11(91.7%) of the bacterial isolates haboured *blaTEM* gene whereas 1(8.3%) were negative.

Plate II: Agarose Gel Electrophoresis of the PCR Products of *aacc1* Gene in Selected Bacteria Isolates

Lane 1: 1.5 Mb pair of the genomic DNA ladder

Lane 2: Isolate TR- 1shows a *blaTEM* band with a gene size of 237bp

Lane 3: Isolate TR-2 shows a *blaTEM* band with a gene size of 237bp

Lane 4: Isolate TR-9 shows a *blaTEM* band with a gene size of 237bp

Lane 5: Isolate TR-5 shows no a *blaTEM* band

Lane 6: Isolate TR-6 shows a *blaTEM* band with a gene size of 237bp

Lane 7: Isolate TR-11 shows a *blaTEM* band with a gene size of 237bp

Lane 8: Isolate TR-3 shows a *blaTEM* band with a gene size of 237bp

Lane 9: Isolate TR- 4 shows a *blaTEM* band with a gene size of 237bp

Lane 10: Isolate TR-10 shows a *blaTEM* band with a gene size of 237bp

Lane 11: Isolate TR-7 shows a *blaTEM* band with a gene size of 237bp

Lane 12: Isolate TR-8 shows a *blaTEM* band with a gene size of 237bp

Lane 13: Isolate TR-12 shows a *blaTEM* band with a gene size of 237bp

Lane 14: Buffer (Negative control)

Molecular detection of *bla SHV*-coding Genes

The electrograph (plate III) shows that out of 12 resistant bacteria, 6(50%) of the bacterial isolates haboured *blaSHV* gene whereas 6(50%) were negative.

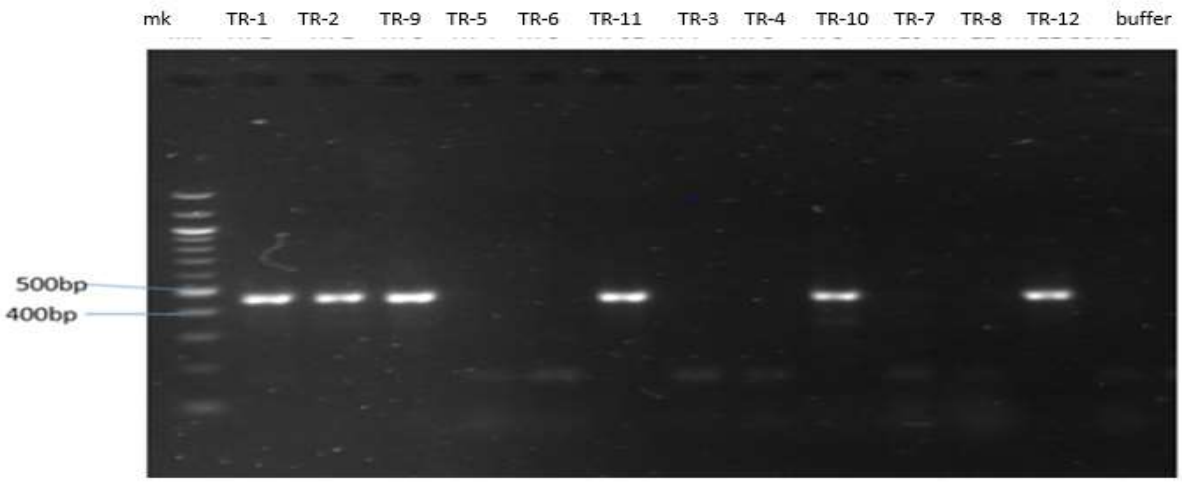

Plate III: Agarose Gel Electrophoresis of the PCR Products of *Blashv* Gene in Selected Bacteria Isolates

Lane 1: 1.5Mb pair of the genomic DNA ladder

Lane 2: Isolate TR-1 shows a *blaSHV* band with a gene size of 470bp

Lane 3: Isolate TR-2 shows a *blaSHV* band with a gene size of 470bp

Lane 4: Isolate TR-9 shows a *blaSHV* band with a gene size of 470bp

Lane 5: Isolate TR-5 shows no *blaSHV* band

Lane 6: Isolate TR-6 shows no *blaSHV* band

Lane 7: Isolate TR-11 shows a *blaTEM* band with a gene size of 470bp

Lane 8: Isolate TR-3 shows no *blaSHV* band

Lane 9: Isolate TR-4 shows no *blaSHV* band

Lane 10: Isolate TR-10 shows a *blaSHV* band with a gene size of 470bp

Lane 11: Isolate TR-7 shows no *blaSHV* band

Lane 12: Isolate TR-8 shows no *blaSHV* band

Lane 13: Isolate TR-12 shows a *blaSHV* band with a gene size of 470bp

Lane 14: Buffer (Negative control)

Molecular detection of *BlaCTX-M* coding Genes

The electrograph (plate IV) shows that out of 12 resistant bacteria, 11(91.7%) of the bacterial isolates haboured *BlaCTX-M* gene whereas 1(8.3%) were negative.

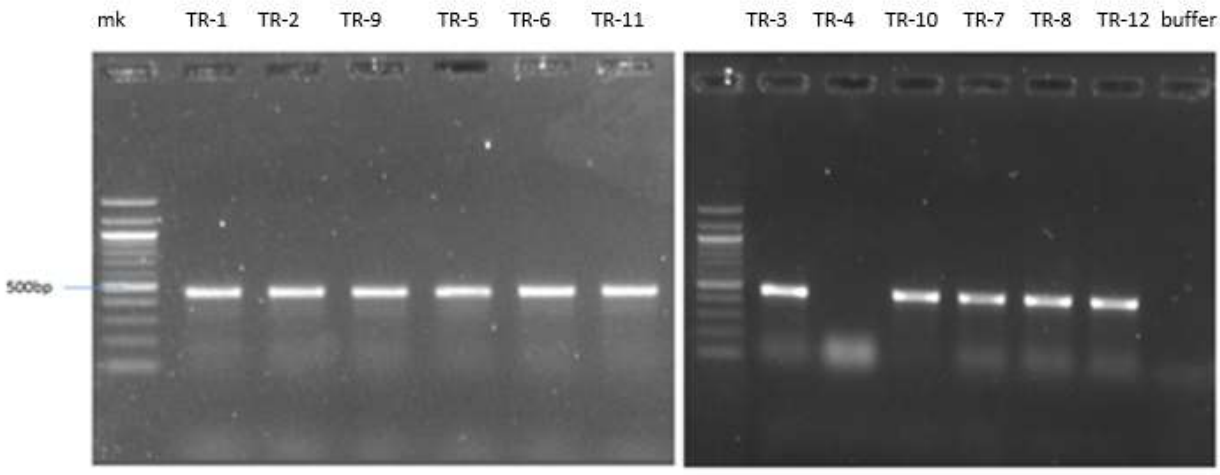

Plate IV: Agarose Gel Electrophoresis of the PCR Products of *BlaCTX-M* Gene in Selected Bacteria Isolates

Lane 1: 1.5 Mb pair of the genomic DNA ladder

Lane 2: Isolate TR- 1 shows a *BlaCTX-M* band with a gene size of 470bp

Lane 3: Isolate TR-2 shows a *BlaCTX-M* band with a gene size of 470bp

Lane 4: Isolate TR-9 shows a *BlaCTX-M* band with a gene size of 470bp

Lane 5: Isolate TR-5 shows a *BlaCTX-M* band with a gene size of 470bp

Lane 6: Isolate TR-6 shows a *BlaCTX-M* band with a gene size of 470bp

Lane 7: Isolate TR-11 shows a *BlaCTX-M* band with a gene size of 470bp

Lane 8: Isolate TR-3 shows a *BlaCTX-M* band with a gene size of 470bp

Lane 9: Isolate TR- 4 shows no *BlaCTX-M* band

Lane 10: Isolate TR-10 shows a *BlaCTX-M* band with a gene size of 470bp

Lane 11: Isolate TR-7 shows a *BlaCTX-M* band with a gene size of 470bp

Lane 12: Isolate TR-8 shows a *BlaCTX-M* band with a gene size of 470bp

Lane 13: Isolate TR-12 shows a *BlaCTX-M* band with a gene size of 470bp

Lane 14: Buffer (Negative control)

Molecular detection of *BlaCTX-M1* coding Genes

The electrograph (plateV) shows that out of 12 resistant bacteria, 4(33.3%) of the bacterial isolates haboured *BlaCTX-M1* gene whereas 8(66.7 %) were negative.

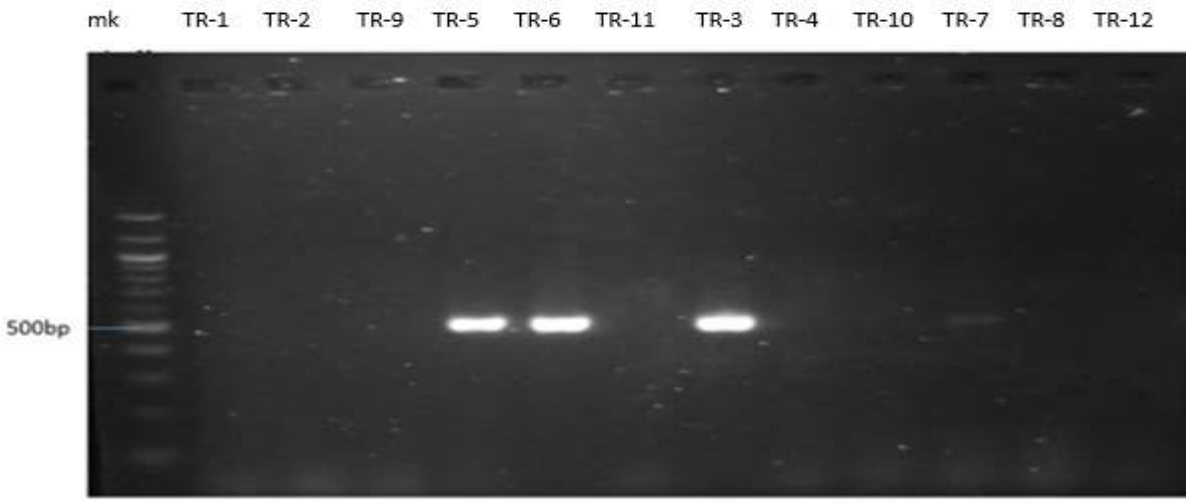

Plate V: Agarose Gel Electrophoresis of the PCR Products of *BlaCTX-M1*Gene in Selected Bacteria Isolates

Lane 1: 1.5 Mb pair of the genomic DNA ladder

Lane 2: Isolate TR-1shows no *BlaCTX-M1* band

Lane 3: Isolate TR-2 shows no *BlaCTX-M1* band

Lane 4: Isolate TR-9 shows no *BlaCTX-M1* band

Lane 5: Isolate TR-5 shows a *BlaCTX-M1* band with a gene size of 500bp

Lane 6: Isolate TR-6 shows a *BlaCTX-M1* band with a gene size of 500bp

Lane 7: Isolate TR-11 shows no *BlaCTX-M1* band

Lane 8: Isolate TR-3 shows a *BlaCTX-M1* band with a gene size of 500bp

Lane 9: Isolate TR-4 shows no *BlaCTX-M1* band

Lane 10: Isolate TR-10 shows no *BlaCTX-M1* band

Lane 11: Isolate TR-7 shows a *BlaCTX-M1* band with a gene size of 500bp

Lane 12: Isolate TR-8 shows no *BlaCTX-M1* band

Lane 13: Isolate TR-12 shows no *BlaCTX-M1* band

Lane 14: Buffer (Negative control)

Molecular detection of *BlaCTX-M2* coding Genes

The electrograph (plateVI) shows that out of 12 resistant bacteria, 7(58.3%) of the bacterial isolates haboured *BlaCTX-M2* gene whereas 5(41.7%) were negative.

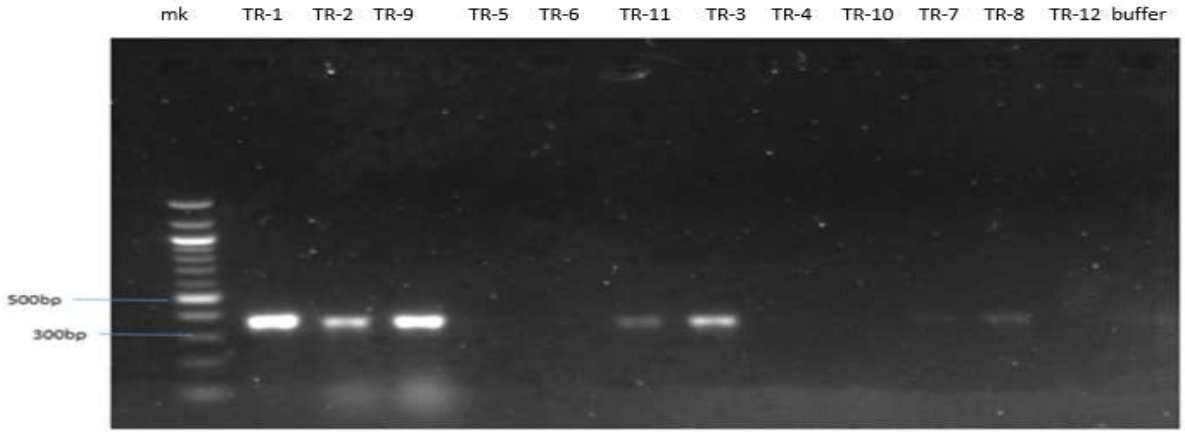

Plate VI: Agarose Gel Electrophoresis of the PCR Products of *BlaCTX-M2* Gene in Selected Bacteria Isolates

Lane 1: 1.5 Mb pair of the genomic DNA ladder

Lane 2: Isolate TR- 1 shows a *BlaCTX-M2* band with a gene size of 351bp

Lane 3: Isolate TR-2 shows a *BlaCTX-M2* band with a gene size of 351bp

Lane 4: Isolate TR-9 shows a *BlaCTX-M2* band with a gene size of 351bp

Lane 5: Isolate TR-5 shows no *BlaCTX-M2* band

Lane 6: Isolate TR-6 shows no *BlaCTX-M2* band

Lane 7: Isolate TR-11 shows a *BlaCTX-M2* band with a gene size of 351bp

Lane 8: Isolate TR-3 shows a *BlaCTX-M2* band with a gene size of 351bp

Lane 9: Isolate TR-4 shows no *BlaCTX-M2* band

Lane 10: Isolate TR-10 shows no *BlaCTX-M2* band

Lane 11: Isolate TR-7 shows a *BlaCTX-M2* band with a gene size of 351bp

Lane 12: Isolate TR-8 shows a *BlaCTX-M2* band with a gene size of 351bp

Lane 13: Isolate TR-12 shows no *BlaCTX-M2* band

Lane 14: Buffer (Negative control)

Molecular detection of *OXA-48* coding Genes

The electrograph (plate VII) shows that out of 12 resistant bacteria, 10(83.3%) of the bacterial isolates haboured *OXA-48* gene whereas 2(16.7%) were negative.

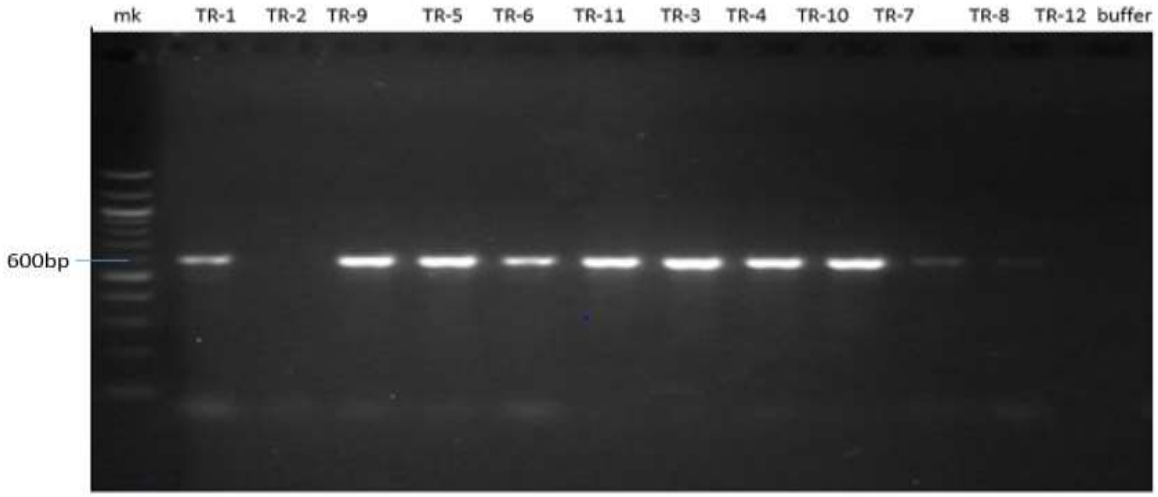

Plate VII: Agarose Gel Electrophoresis of the PCR Products of *OXA-48* Gene in Selected Bacteria Isolates

Lane 1: 1.5 Mb pair of the genomic DNA ladder

Lane 2: Isolate TR-1 shows a *OXA-48* band with a gene size of 590bp

Lane 3: Isolate TR-2 shows no *OXA-48* band

Lane 4: Isolate TR-9 shows a *OXA-48* band with a gene size of 590bp

Lane 5: Isolate TR-5 shows a *OXA-48* band with a gene size of 500bp

Lane 6: Isolate TR-6 shows a *OXA-48* band with a gene size of 590bp

Lane 7: Isolate TR-11 shows a *OXA-48* band with a gene size of 590bp

Lane 8: Isolate TR-3 shows a *OXA-48* band with a gene size of 590bp

Lane 9: Isolate TR-4 shows a *OXA-48* band with a gene size of 590bp

Lane 10: Isolate TR-10 shows a *OXA-48* band with a gene size of 590bp

Lane 11: Isolate TR-7 shows a *OXA-48* band with a gene size of 590bp

Lane 12: Isolate TR-8 shows a *OXA-48* band with a gene size of 590bp

Lane 13: Isolate TR-12 shows no *OXA-48* band

Lane 14: Buffer (Negative control)

Molecular detection of *gyrA* coding Genes

The electrograph (plate VIII) shows that out of 12 resistant bacteria, 6(50%) of the bacterial isolates haboured *gyrA* gene whereas 6(50%) were negative.

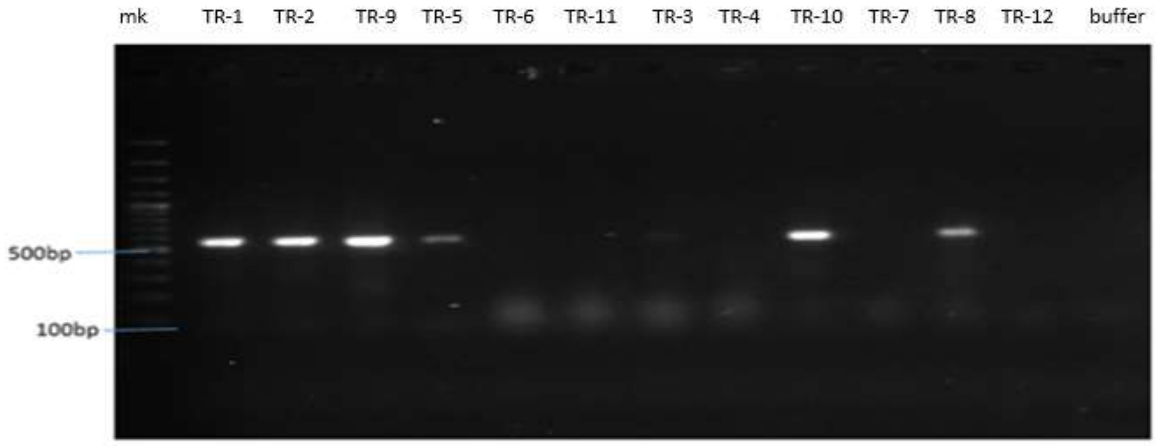

Plate VIII: Agarose Gel Electrophoresis of the PCR Products of *gyrA* Gene in Selected Bacteria Isolates

Lane 1: 1.5 Mb pair of the genomic DNA ladder

Lane 2: Isolate TR-1 shows a *gyrA* band with a gene size of 550bp

Lane 3: Isolate TR-2 shows a *gyrA* band with a gene size of 550bp

Lane 4: Isolate TR-9 shows a *gyrA* band with a gene size of 550bp

Lane 5: Isolate TR-5 shows a *gyrA* band with a gene size of 550bp

Lane 6: Isolate TR-6 shows no *gyrA* band

Lane 7: Isolate TR-11 shows no *gyrA* band

Lane 8: Isolate TR-3 shows no *gyrA* band

Lane 9: Isolate TR-4 shows no *gyrA* band

Lane 10: Isolate TR-10 shows a *gyrA* band with a gene size of 550bp

Lane 11: Isolate TR-7 shows no *gyrA* band

Lane 12: Isolate TR-8 shows a *gyrA* band with a gene size of 550bp

Lane 13: Isolate TR-12 shows no *gyrA* band

Lane 14: Buffer (Negative control)

Molecular detection of *ParC* coding Genes

The electrograph (plate IX) shows that out of 12 resistant bacteria, 10 (83.3%) of the bacterial isolates haboured *ParC* gene whereas 2(16.7%) were negative.

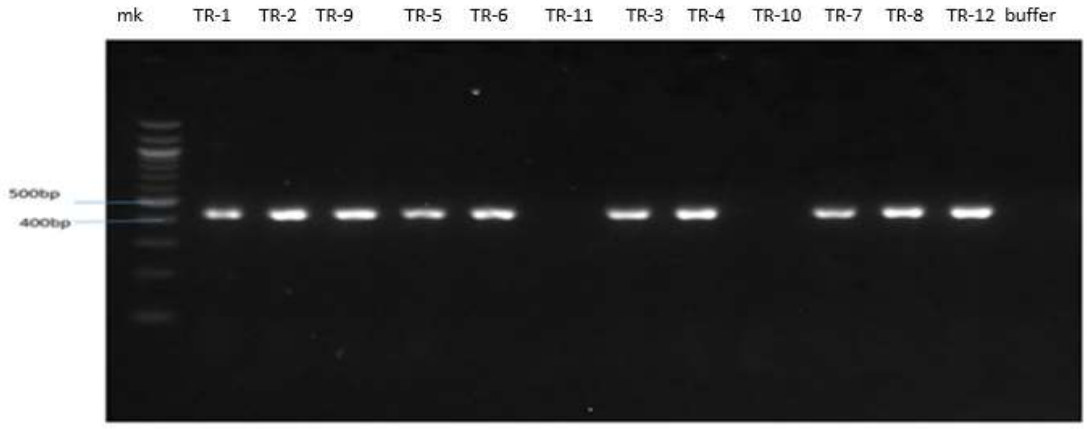

Plate IX: Agarose Gel Electrophoresis of the PCR Products of *ParC* Gene in Selected Bacteria Isolates

Lane 1: 1.5 Mb pair of the genomic DNA ladder

Lane 2: Isolate TR-1 shows a *ParC* band with a gene size of 420bp

Lane 3: Isolate TR-2 shows a *ParC* band with a gene size of 420bp

Lane 4: Isolate TR-9 shows a *ParC* band with a gene size of 420bp

Lane 5: Isolate TR-5 shows a *ParC* band with a gene size of 420bp

Lane 6: Isolate TR-6 shows a *ParC* band with a gene size of 420bp

Lane 7: Isolate TR-11 shows no *ParC* band

Lane 8: Isolate TR-3 shows a *ParC* band with a gene size of 420bp

Lane 9: Isolate TR- 4 shows a *ParC* band with a gene size of 420bp

Lane 10: Isolate TR-10 shows no *ParC* band

Lane 11: Isolate TR-7 shows a *ParC* band with a gene size of 420bp

Lane 12: Isolate TR-8 shows a *ParC* band with a gene size of 420bp

Lane 13: Isolate TR-12 shows a *ParC* band with a gene size of 420bp

Lane 14: Buffer (Negative control)

Molecular detection of *aacC1* coding Genes

The electrograph (plate X) shows that out of 12 resistant bacteria, 7(58.3%) of the bacterial isolates haboured *aacC1* gene whereas 5(41.7%) were negative.

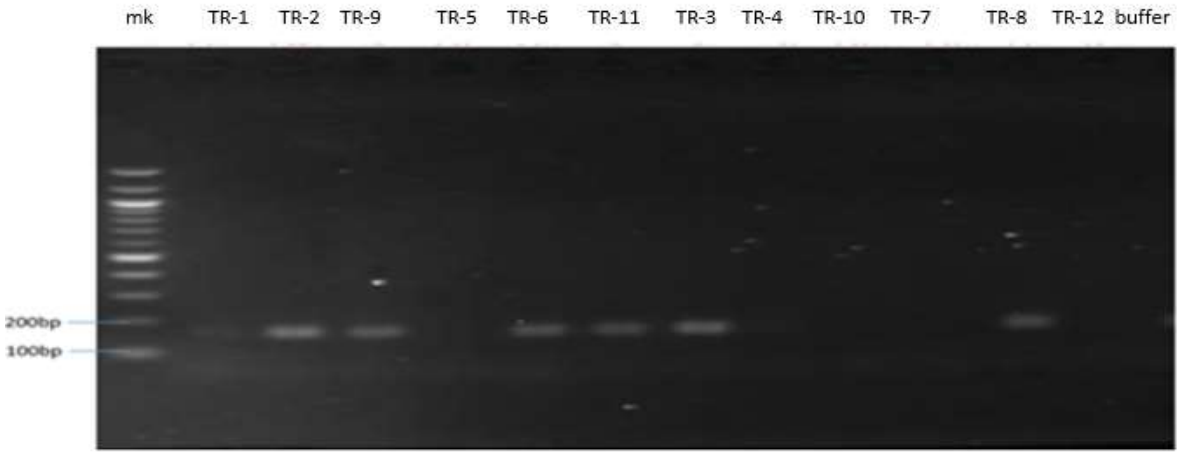

Plate X: Agarose Gel Electrophoresis of the PCR Products of *aacC1* Gene in Selected Bacteria Isolates

Lane 1: 1.5 Mb pair of the genomic DNA ladder

Lane 2: Isolate TR- 1shows an *aacC1* band with a gene size of 169bp

Lane 3: Isolate TR-2 shows an *aacC1* band with a gene size of 169bp

Lane 4: Isolate TR-9 shows an *aacC1* band with a gene size of 169bp

Lane 5: Isolate TR-5 shows no *aacC1* band

Lane 6: Isolate TR-6 shows an *aacC1* band with a gene size of 169bp

Lane 7: Isolate TR-11 shows an *aacC1* band with a gene size of 169bp

Lane 8: Isolate TR-3 shows an *aacC1* band with a gene size of 169bp

Lane 9: Isolate TR- 4 shows no *aacC1* band

Lane 10: Isolate TR-10 shows no *aacC1* band

Lane 11: Isolate TR-7 shows no *aacC1* band

Lane 12: Isolate TR-8 shows an *aacC1* band with a gene size of 169bp

Lane 13: Isolate TR-12 shows no *aacC1* band

Lane 14: Buffer (Negative control)

Molecular detection of *aacC2* coding Genes

The electrograph (plate XI) shows that out of 12 resistant bacteria, 6(50%) of the bacterial isolates haboured *aacC2* gene whereas 6(50%) were negative.

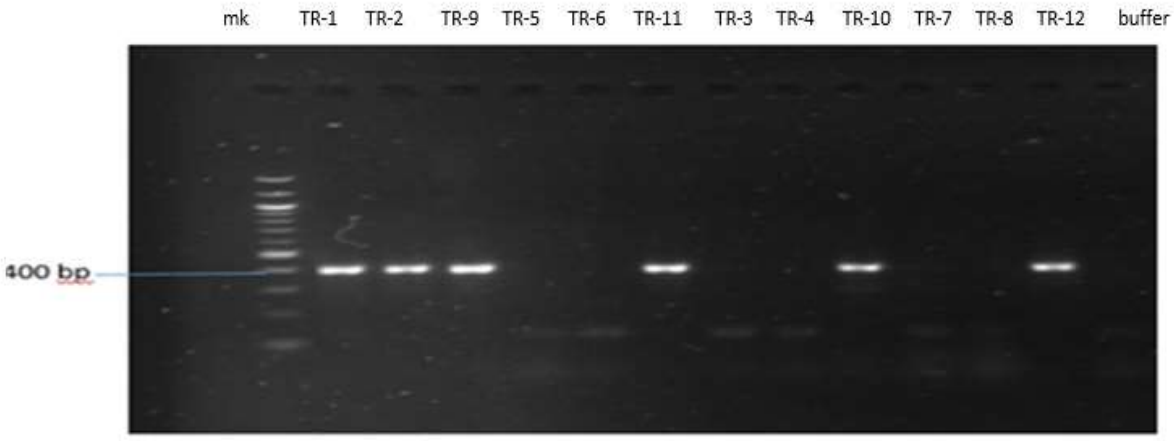

Plate XI: Agarose Gel Electrophoresis of the PCR Products of *aacC2* Gene in Selected Bacteria Isolates

Lane 1: 1.5 Mbase pair of the genomic DNA ladder

Lane 2: Isolate TR-1 shows an *aacC2* band with a gene size of 400bp

Lane 3: Isolate TR-2 shows an *aacC2* band with a gene size of 400bp

Lane 4: Isolate TR-9 shows an *aacC2* band with a gene size of 400bp

Lane 5: Isolate TR-5 shows no *aacC2* band

Lane 6: Isolate TR-6 shows no *aacC2* band

Lane 7: Isolate TR-11 shows an *aacC2* band with a gene size of 400bp

Lane 8: Isolate TR-3 shows no *aacC2* band

Lane 9: Isolate TR-4 shows no *aacC2* band

Lane 10: Isolate TR-10 shows an *aacC2* band with a gene size of 400bp

Lane 11: Isolate TR-7 shows no *aacC2* band

Lane 12: Isolate TR-8 shows no *aacC2* band

Lane 13: Isolate TR-12 shows an *aacC2* band with a gene size of 400bp

Lane 14: Buffer (Negative control)

4.1.17 Prevalence of resistant genes from resistant organisms

Out of the twelve isolates screened, eleven isolates were found to be positive for TEM (Temoneira); six isolates were found to be positive for SHV; eleven isolates were found to be positive for CTXM; four isolates were found to be positive for CTXM1; seven isolates were found to be positive for CTXM2; six isolates were found to be positive for gyrA; ten isolates were found to be positive for parC; seven isolates were found to be positive for aaC1; six isolates were found to be positive for aaC2; ten isolates were found to be positive for OXA-48 (Table 4.56).

Table 4.56: Frequency of Occurrence of Resistant Genes from Multidrug Resistant Organisms

Isolates	***TEM***	***SHV***	***CTXM***	***CTXM1***	***CTXM2***	***gyrA***	***parC***	***aacC1***	***aacC2***	*OXA-48*
TR-1	+	+	+	-	+	+	+	+	+	+
TR-2	+	+	+	-	+	+	+	+	+	-
TR-9	+	+	+	-	+	+	+	+	+	+
TR-5	-	-	+	+	-	+	+	-	-	+
TR-6	+	-	+	+	-	-	+	+	-	+
TR-11	+	+	+	-	+	-	-	+	+	+
TR-3	+	-	+	+	+	-	+	+	-	+
TR-4	+	-	-	-	-	-	+	-	-	+
TR-10	+	+	+	-	-	+	-	-	+	+
TR-7	+	-	+	+	+	-	+	-	-	+
TR-8	+	-	+	-	+	+	+	+	-	+
TR-12	+	+	+	-	-	-	+	-	+	-
	11(91.7%)	6(50%)	11(91.7%)	4(33.3%)	7(58.3%)	6(50%)	10(83.3%)	7(58.3%)	6(50%)	10(83.3%)

4.1.18 Profile of various resistant genes in multidrug resistant organisms

Out of the four different organisms screened, all isolates (that is 100% isolates) of *Klebsiella pneumoniae* were found to be both positive for TEM and OXA-48 (as seen in Table 4.57)

Table 4.57: Profile of Various Resistant Genes in Multidrug Resistant Organisms

Isolates	**Number of isolates**	***TEM* (%)**	***SHV* (%)**	***CTXM* (%)**	***CTXM1* (%)**	***CTXM2* (%)**	***gyrA* (%)**	***parC* (%)**	***aacC1* (%)**	***aacC2* (%)**	***OXA-48* (%)**
Escherichia coli	3	3(100)	3(100)	3(100)	0(0)	3(100)	3(100)	3(100)	3(100)	3(100)	2(66.7)
Klebsiella pneumoniae	3	3(100)	1(33.3)	2(66.7)	1(33.3)	1(33.3)	1(33.3)	2(66.7)	1(33.3)	1(33.3)	3(100)
Salmonella Typhi	3	2(66.7)	1(33.3)	3(100)	2(66.7)	1(33.3)	1(33.3)	2(66.7)	2(66.7)	1(33.3)	3(100)
Proteus vulgaris	3	3(100)	1(33.3)	3(100)	1(33.3)	2(66.7)	1(33.3)	3(100)	1(33.3)	1(33.3)	2(66.7)

4.1.19 Phylogenetic analysis result

The Phylogenetic tree was constructed using 12 isolates from this study and 8 reference strains selected from NCBI data base on percentage similarity, the tree had 3 clades. Clade 1 had most of the reference strains (as seen in Figure 4.4).

The second clade had three (3) strains of *Proteus vulgaris* namely TR-7, TR-12, TR-8 and reference strain CP033736 and MK572636. From the phylogenetic tree TR-7 was found to be closely related to reference strain CP033736 isolated from United States of America (USA) while both strains TR-12 and TR-8 had the same ancestral parents indicating high similarity between the two strains and they wereound to be closely related to the reference strain MK572636 isolated from Egypt (as seen in Figure 4.4)

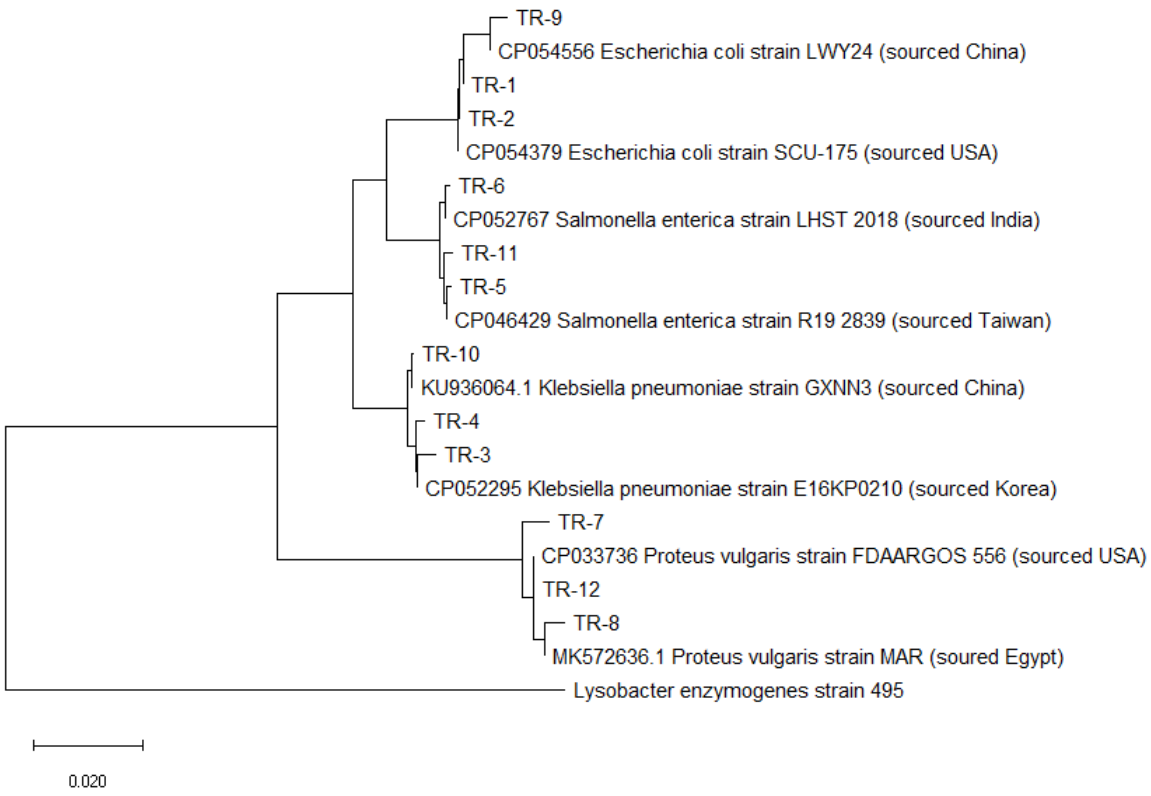

Figure 4.7: Evolutionary relationships of taxa

4.2 Discussion of Results

The study determined 720(62%) of the samples collected from PID patients were positive for bacterial growth. This is based on the silent spread of bacteria to the upper genital tract which results to high degree of damages such as miscarriage, preterm labor and ectopic pregnancy in the infected females (Ahmed 2017; Naaz *et al.,* 2016; Oseni and Odewale, 2017). These therefore lead to infertility among the female population. This is in agreement with the findings of (Pachori and Kulkarni, 2016 and Naaz *et al*., 2016) who reported that higher rates of bacterial infections such as 60%, 57% and 30% in Africa, Asia and Indian respectively. This result also agrees with the findings of Shinde *et al.* (2018), who observed that all 200 (100%) PID patients sampled had high rates of bacterial infection. However, this study disagrees with the findings of French *et al.* (2011) and Huang *et al.* (2019), who estimated the prevalence rate of PID infection to be 1.6% and 2% respectively. The observed variation may be due to different sample sizes sampled in these studies.

This study revealed the occurrence of different bacterial pathogens in the PID patients. This could be attributed to certain factors such as impairment of the natural protective layer during menstruation and the dilation of the cervical canal after abortion or delivery, which may render the genital tract highly vulnerable to pathogenic urogenital organisms. In addition, continuous use of intrauterine device and manual removal of placenta, also favor the entry and spread of these urogenital organisms as reported by (Fraimow and Abrutyn, 1995; Saini *et al.,* 2003; Padubidri and Daftary, 2010; Sharma *et al.,* 2013).

The highest occurrence of *E.coli* 170 (23.6%) revealed in this study could be based on the fact that *E.coli* predominantly colonize the gastrointestinal tract and is the main causative agent of urinary tract infection, and this frequently exposes the vagina to the

organism due to its proximity to periurethral openings and the perianal areas. This results is in agreement with the findings of Erdem *et al.* (2018), who revealed that majority of the organisms isolated from patients with urogenital infections are *E.coli*.

The higher rate of bacterial infection (32.5%) among women in the rural areas in this study, is due to the fact that most of these rural women are ignorant of symptoms associated with PID, due to lack of awareness of signs and symptoms of PID alongside inadequate standard health care facilities. This finding is in agreement with Dayal *et al.*, (2016), from a study conducted in a rural settlement in India, however, disagrees with Usman (2016), who reported high prevalence of bacterial infection (72.3%) in urban setting compared to (27.2 %) in the rural setting, and this could be based on the fact that the patients she studied, visited mainly General hospitals in the urban settlements.

The highest distribution of bacterial infection (24.4%) observed among patients within 25-29 years in this study could be attributed to the fact that these patients are sexually active and are also within their reproductive age. In addition to this, these young women posses cervical mucus which lacks antimicrobial properties and as such renders them highly vulnerable to urogenital pathogens. This result is also in agreement with Shinde *et al.* (2018) and Ahmed *et al*. (2017) who reported 26.5% and 54% infection rate in female participants between 25- 29 years and 26-35years respectively. This result is in disparity with the findings of Dayal *et al.* (2016), who reported the highest PID infection in patients within 31-40 years, based on the fact that most of these patients still engage in child bearing.

Furthermore, the reason for high prevalence of bacteria in 15-19 and 20-19 years is not clearly understood. However, majority of the participants in this age bracket are singles and are in the university or other form of learning. It is likely that these individuals

engage in frequent practice of sexual relationships and frequent abortion which exposes them to certain urogenital pathogens.

The high distribution of bacterial infection observed among patients with no previous episodes of PID (52.0%) and STI (47.0%) could be attributed to the fact that PID is a polymicrobial infection and can arise when pathogenic bacteria around the genital area resist the effect of lactic acid and as such alter the pH of the genital areas. This finding is in agreement with Simm (2006); Josey and Schwebke,2008 and Ahmed *et al.* (2017), who stated that all PID patients sampled had no PID and STI history.

The high rate of bacterial infection observed among patients who had never had abortion (43.4%) could be attributed to the fact that most of these women studied were from the rural settlement, and as such have depended and still depends primarily on local, less trained birth attendants and relatives for assistance during child birth, who conduct child delivery process in unhygienic environments (such as the patient's homes) with their bare hands. This gives an opportunity for potential pathogens to pass from the lower genital tract into the uterus. This finding is in disagreement with Ahmed *et al.* (2017), who stated that 50 (33.33%) out of 150 patients sampled practiced unsafe child delivery in their homes, which is a potential source for the entrance of pathogens.

Furthermore, patients who had previous episodes of urinary tract infections (UTI) and recorded high bacterial infection (49.4%) could be based on the fact that the urinary tract (which shows high proximity to the female genital organs) as well as the intestinal bowels is also a habitat for most opportunistic organisms such as the anaerobes which are the main aetiological agents for most PID (such as salpingitis). In addition, changes induced by pregnancy and delivery contribute to an easier access of bowel flora to the

vagina and then the upper genital tracts. This finding is in agreement with Ahmed *et al.* (2017), who stated that 60% PID cases sampled, all recorded urinary tract infections.

The highest rate of bacterial infection which occurred more among the married patients (51.5%), is due to the fact that most women studied are young and sexually active and as such involve most frequently in sexual activities, which enhances easier ascension of pathogenic bacteria from lower genitals to the upper genital tracts. This finding is in agreement with Ahmed *et al.* (2017), who stated 90% of women with PID cases are married and Shinde *et al.* (2018) who revealed that 79% of women with PID are married, but this finding was in disagreement with the study of Naaz *et al.* (2016), who stated that 58.6% of the patients that had percentages of PID were singles compared to 41.6% of the patients who were married.

Women who practiced polygamy, had the highest rate of bacterial infection (40.3%) based on the fact that most women studied, have partners who engage themselves in multiple sexual practices with other women, who could be potential sources of various pathogenic bacteria associated with PID. This finding is similar with the findings of Usman (2016), who stated that all the 66.7% of women studied were into polygamous family while 30.7% women were into monogamous family.

The highest rate of bacterial infection, observed among patients who are unemployed (32.8%) could be based on the fact that these women studied were mostly housewives with poor socio-economic status and as such lack adequate financial supports to enable them maintain certain hygienic standards around their genitals. This therefore leads to abnormal growth of causative agents of PID around the genitals. This finding is in agreement with Ahmed *et al.* (2017), who stated that out of all the PID cases sampled 90% were unemployed.

High rate of bacterial infection also observed in women who are uneducated (29.3%) could be based on the fact that most women studied, were not exposed to western education, which enlightens individuals on various prevailing diseases and adequate preventive measures needed to control this disease. This finding is in agreement with Naaz (2016) and Ahmed *et al.* (2017) who stated that women with PID infection revealed high percentages of illiterates such as 30% and 60% respectively.

The high rate of infection observed among patients who practiced douching daily (44.4%) could be attributed to the fact that the frequency of douching among such patients is high, and this alters the population of the patient's microflora around the genital area, thereby supporting the growth of pathogenic microbes especially anaerobes which are causative agents of most pelvic inflammatory diseases. This finding is in agreement with Usman (2016) and Short *et al.* (2015) who revealed that high percentages of women with PID practice douching.

Patients who used water and soap for douching had higher rate of bacterial infection (31.0%), based on the fact that consistent use of soap, which is a compound of various chemicals cause genetic mutation in some or all the microbes in a vaginal region. Such mutation therefore influences the virulence of these organisms, which causes the organisms to be pathogenic in their patients. In addition to this, patients who used river water to clean their genital areas possessed higher rate of bacterial infection (27.7%) (Table 4.15), based on the fact that river water habours numerous pathogenic bacteria and constant use of such river water exposes such women to numerous pathogens which are said to ascend from the lower genital tract to the upper genital tract. This finding is in agreement with the findings of Short *et al.* (2015).

The high bacterial infection observed among patients who pass out their waste products in the open environment (38.7%), could be based on the fact that most women studied lack adequate toilet facilities in their various residential communities and as such pass out their waste products on microbes contained environment (such as the soil), where they utilize various contaminated materials (such as paper, dry leaves or water) to clean themselves. This thereby enhances the spread of pathogens from the lower genital tract to the upper genital tract. This finding is in disagreement with Usman (2016), who stated that 64% of the women sampled who used water system, had high occurrence of PID while 24.3% and 11.7% of women sampled used pit latrine and open environment.

The high rate of bacterial infection observed in patients who are non users of birth controls (37.9%) could be based on the fact that the acceptance of family planning and adequate financial support to sustain family planning by such patients is low and, in most cases, such individuals engage in frequent child births with untrained health workers. This finding is in agreement with the findings of Ahmed *et al.* (2017). However, high rate of bacterial infection among women who use intrauterine device as birth control (11.2%), could be based on the fact that high pathogens usually accompany the unhygienic insertion of this device into the female urogenitals. This in turn, causes pelvic inflammatory disease (Table 4.18).

High rate of bacterial infection observed in patients who practiced self medication (48.0%) and incomplete dosage of drugs (42.3%) could be based on the fact that these patients practice irrational use of certain synthetic or herbal products which have therapeutic effect, without the prescription of a health care provider. This in turn lead to the development of resistance among pathogenic bacteria around the genital areas, and this therefore results to a type of PID that is usually difficult to control. This finding is in agreement with the findings of Dayal *et al.* (2016).

This study revealed that all multidrug-resistant (MDR) bacteria isolated from the endo cervical swabs (ECS) and urine samples were Gram-negative (as seen in Table 4.24). This could be based on the fact that Gram negative organisms posses various mechanisms of intrinsic resistance such as: the presence of lipopolysaccharides in the outer membrane or external layer (which prevent penetration of hydrophobic solutes such as antibiotics) (Ferreira *et al.,* 2019); the absence of two main porins in the outer membrane, which are encoded by *Omp*K35 and *Omp*K36 genes (which enhances reduced permeability of certain antibiotics in the outer membrane) (Effah *et al.,* 2020) and the presence of efflux pumps, especially the multidrug efflux pump system in the inner membrane, which are encoded by *Mex*AB-*OPr*M gene complexes or *Acr*AB and *mdtk* gene complexes (which pump out antibiotics such as fluoroquinolones, tetracyclines, phenicols, macrolides and betalactams) (Hujer *et al.,* 2006; Hirdon *et al.,* 2008; Ferreira *et al.,* 2019; Breijyeh *et al.,* 2020; Effah *et al.,* 2020) . However, these various intrinsic resistance, could either occur alone in bacterial cells or could occur syngergistically with one another in bacterial cells and this inturn, prevent easy penetration of antibiotics and chemicals into various Gram negative bacterial cells (Kudinha, 2017).

Similarly, the highest occurrence of multidrug resistant *K. pneumoniae* in this study 87(56.1%), could be attributed to the fact that multidrug resistant *K. pneumoniae* exhibit certain virulent factors such as: production of adhesin, encoded by genes such as *fim*H and *mrk*D (which enhances multidrug resistant *K. pneumoniae* to bind or adhere firmly to various host surfaces to ensure easy penetration); presence of capsule (which prevent phagocytosis and enhances multiplication of the multidrug resistant *K. pneumoniae* in the host) and production of siderophore (which are needed to chelate or remove iron from the host cell for their survival) (Kumar *et al.,* 2019; Effah *et al.,* 2020). This result

also conforms to the study of Metri *et al.* (2012), who revealed that 58 bacteria isolates associated with urogenital infections, were multi drug resistant *Klebsiella pneumoniae*.

The highest multidrug resistant bacteria observed in General hospital Lapai 36(15.8%) as seen in (Fig 4.1), could be based on the fact that, most women attending General hospital Lapai practice self medication/ irrational use of drugs, which is usually accompained with the partial elimination of these disease causing bacteria in most urogenital tracts of many female patients; thus enhancing the development of mutation in the bacterial genes as reported by Gorgani *et al.* (2009), Okonko *et al.* (2009) and Oyedum, (2015). This result disagrees with the findings of Alo and Dike, (2018), who stated that 28 (70%) out of 40 isolates, were multidrug resistant. This variation could be based on the differences in the, number of women in the various study populations, behaviours of various women in the various study populations and standard of personal hygiene of various women in the various study populations as reported by Shaifali *et al.* (2012).

The high resistance to Nalidixic acid and Ofloxacin in six (6) and three (3) general hospitals (as seen in Fig 4.2), could be based on the fact that the multidrug resistant bacteria in these hospitals exhibit alterations in their antibiotic target sites, which are the bacterial enzymes such as *gyrA* and *parC* needed for DNA replication. In addition, certain gene, referred to as *qnr*, which encodes for a protein that protects the DNA gyrase from the effect of various quinolones and fluoroquinolones could be contained in the plasmids present in these multidrug resistant bacteria, as reported by Mahaluca *et al.* (2019). This result agrees with the findings of Anuli *et al.* (2016) and Anejo-Okopi *et al.* (2015) who revealed that uropathogens isolated from females exhibited 100% and 83.3% resistance to Nalidixic acid and other fluoroquinolones. However, this result disagrees with the findings of Islam *et al.* (2013) who reported that the isolated

urogenital pathogens were 90% resistant to Amoxicillin and then 57% resistant to Nalidixic acid.

The high resistance of Gentamicin observed in seven (7) General hospitals (as seen in Figure 4.3) could be attributed to the alterations in the binding sites of the 30S subunit of these bacterial ribosomes, which is due to the illegal intake of this antibiotic intravenously or intramuscularly. Similarly, high resistance to Gentamicin could also be attributed to the acquisition of plasmids which contains genes that code for aminoglycoside- modifying enzymes which are narrow specific to a given aminoglycosides, as reported by Lotfollahi *et al.* (2015). This result agrees with the findings of Anejo-Okopi *et al.* (2017), who revealed 90% of uropathogens were resistant to Gentamicin.

The high resistance to ampicillin (as seen in Fig 4.4) could be atrnitributed to the fact that these bacterial isolates have plasmids which contain certain genes that code for the production of bacterial enzymes known as betalactamases, which inactivate betalactam drugs such as ampicillin. This result conforms to the findings of Anyadoh-Nwadike *et al.* (2015), Shaskolskiy *et al.* (2016), Saginela *et al.* (2017) and Waske *et al.* (2017), who revealed that bacterial urogenital pathogens isolated exhibited highest resistance of 88%, 94% and 100% to Ampicillin. However, this study disagrees with the findings of Saha and Kulkarni (2018), who revealed that bacterial urogenital pathogens isolated exhibited highest resistance of 57% to Cephalexin and the lowest resistance of 27% to Ampicillin. The observed variation may be based on the fact that Cephalexin was widely used in the study area.

The high resistance to Augmentin (as seen in Fig 4.5) in eight General hospitals could be attributed to the acquisition of plasmids, which contain genes that code for

Augmentin- modifying enzymes, which inactivates Augmentin. This result agrees with the findings of Chaudhary *et al.* (2016) and Kumar *et al.* (2019) who reported that all the bacterial isolates revealed high resistance of 69% and 64% to Augmentin compared to other antibiotics.

The study also revealed that patients from 6 general hospitals had higher multidrug resistant bacteria in their urine compared to their endocervical swabs (Fig 4.6). This could be based on the fact that most of these multi drug resistant pathogens posses certain unique factors such as; adhesins, bacterial toxins, host defense avoidance mechanisms and multiple iron acquisition systems (Sandoz and Rockey,2010; Scholes *et al.*, 2012; Hye *et al.,* 2019 and Sarowska *et al.,* 2019), which enhance their rapid attachment, invasion and multiplication in the urinary tract of the host as reported by Lavigne *et al.* (2011).

This study revealed that bacterial isolates from endocervical swabs and urine samples of patients, resistant to 5 or more antibiotics were the highest prevalent multidrug resistant bacteria (81.1%, 79.7%) (Table 4.46 and 4.47). This could be attributed to the fact that, these bacteria exhibited high horizontal gene transfer mechanisms via processes like conjugation, transformation and transduction, which are regarded as the basis of multidrug resistance (which could occur as antibiotics inactivation, alteration of target sites, alteration of semi permeable membrane, efflux pump or production of microbial modifying enzymes) among bacterial isolates (Nikaido, 2009; Muthoni, 2012; Iseghohi, 2016). In addition, the high prevalence of bacterial isolates resistant to 5 or more antibiotics, indicates high multiple antibiotic resistance index (MARI) among the multidrug resistant bacteria, thus implying that a large proportion of the bacterial isolates have been exposed to several antibiotics and as such are great threat to the health of the populace at large.

This study also revealed that *Klebsiella pneumoniae* is the most isolated multidrug bacteria in endocervical swab (50.0%) and urine (61.2%) (Table 4.46 and 4.47). This is based on the fact that multi drug resistant *Klebsiella pneumoniae* is one of the predominant pathogens that is commonly associated with the genitourinary or urogenital system. This result conforms with the findings of Woldu, (2015) and El-kady and Gouda, (2017), who stated that high percentages of multi drug resistant *Klebsiella pneumoniae* (71.2%) were associated with endocervical and urine infections. However, this finding disagrees with the findings of Anyadoh-Nwadike *et al.* (2018), who revealed that the most isolated multidrug resistant isolate from both endocervical swap and urine was *Staphylococcus aureus.*

In this study, the production of extended spectrum betalactamases (ESBL) and carbapenemase were observed more in *Klebsiella pneumoniae* compared to other multidrug resistant bacterial isolates (Table 4.49 and 4.50). This is attributed to the fact that these resistant *Klebsiella pneumoniae* that have been established to possess high level of genetic materials such as plasmids and transposons (such as, *Tn3* and *Tn 4401*), that contains genes responsible for producing, extended spectrum betalactamases and carbapenemase. This result conforms to the report of Ensor *et al.* (2009); Chander and Shrestha (2013); Ejikeugwu *et al.* (2013); Kavita *et al.* (2016); Ibrahimagic *et al.* (2017) and Tariq (2017). However, this finding disagrees with the findings of Indernath *et al.* (2018) and Onanuga *et al.* (2019), who stated that extended spectrum betalactamase (ESBL) and carbapenemase were expressed more in *Escherichia coli* than other multidrug resistant bacteria, and this is based on the fact that, all the multidrug resistant *Escherichia coli* in their studies possessed higher genetic materials coding for these enzymes production.

The high production of ESBL compared to other enzymes observed (in Tables 4.49 and 4.50) could be based on the misuse and inappropriate administration of third generation Cephalosporins in both hospital and community settings for the treatment of certain Gram-negative bacterial infections. This result agrees with the findings of Bora *et al.* (2016) and Mohammed *et al.* (2016), who revealed that ESBL were highly produced by various Gram-negative bacteria isolated from different clinical samples. However, the high occurrence of extended spectrum betalactamase and carbapenemase producing bacteria in urine compared to endocervical swab (as seen in Table 4.51- 4.53) is based on the fact that most patient with pelvic inflammatory disease also have asymptomatic urinary infections associated with resistant bacteria, and as these organisms proliferate in large numbers in the infected urinary sites, their resistant genes are also said to increase. This result agrees with the findings of Iqbal *et al.* (2014) and Kausar *et al.* (2014).

The highest occurrence of resistant genes such as *TEM* (91.7%) and *CTX-M* (91.7%) in this study, could be based on the fact that most urogenital pathogens within the study area, greatly habour large quantities of extended spectrum betalactames genes such as; *TEM* and *CTX-M* and these genes are mainly the basis of ESBL in these locations. This result agrees with the findings of Iseghohi (2016) and Seyedjavadi *et al.* (2016) who revealed that *TEM* (88%) and *CTX-M* (66.6%) were predominantly found in extended spectrum betalactamase-producing *E.coli* isolates from clinical samples. In addition, Bora *et al.* (2014) also revealed that high prevalence of *TEM* (88%) and *CTX-M* (77.6%) were the most common genotype detected from both *E.coli* and *Klebsiella pneumoniae* isolates obtained from various clinical samples. Sana *et al.* (2011) also reported high prevalence of *CTX-M* (79.5%) among urogenital pathogens isolated from various hospitals in Lebanon. Similarly, high prevalence rates of *CTX-M* (94.6%) and

TEM (56.8%) was reported by Moghaddan *et al.* (2012) while Dexheimer *et al.* (2015) reported high prevalence rate of *CTX-M* (90.32%) and *TEM* (70.69%). However this finding differs from the findings of Youssef and Al Subal (2015) who reported that higher prevalence of 100% and 76.1% of the *CTX-M-1* and *CTX-M-2* in ESBL-producing *Klebsiella pneumoniae* and *E.coli*. In the same vein, Shahid *et al.* (2011) also reported a high prevalence of 58.3% *CTX-M1* in bacterial isolates obtained from South-India.

The low prevalence of *blaSHV* (50%) in this study compared to other resistant gene could be attributed to the fact that, *blaSHV* is usually harboured by only a particular member of enterobacteriaceae, namely *Klebsiella sp*; hence its low prevalence among various members of the enterobacteriaceae. This result agrees with the findings of Ahmed *et al.* (2017) and Sana *et al.* (2011), who reported lower prevalence of 7.1% and 5.2% of *blaSHV* gene compared to the other ESBL-producing gene counterparts isolated from various bacteria from different clinical specimens. However, this result disagrees with the findings of Youssef and Al Subal, (2015), who revealed higher prevalence of *blaSHV* of 95% and 58% in *Klebsiella pneumoniae* and *Escherichia coli* compared to other genes responsible for ESBL. This change in the prevalence rate of *blaSHV* studied could be attributed to the different locations that were studied. Certain genes are more predominant in certain locations than others.

Similarly, the high prevalence of *OXA* (oxacillinase) gene (83.3%) among various urogenital pathogens in this study is based on the fact that, one of the betalactamase gene (particularly, the carbapenemase) capable of hydrolyzing carbapenems is also highly co-haboured with other genes of betalactamase. This result conforms to the findings of Mathlouthi *et al.* (2016), who reported high prevalence of *OXA-48* type of carbapenemase, among *Klebsiella pneumoniae* isolated from Libyan hospitals. In

addition to this, the highest prevalence rate (73.8%, 88% and 86%) of *OXA-48* among various members of the enterobacteriaceae was also reported by Hamed and Hasoon (2019); Dandachi *et al.* (2016) and Iraz *et al.* (2015) respectively.

The high prevalence of various betalactamases such as ESBL- producing genes (*TEM* and *CTX-M*) and carbapenemase-producing gene (*OXA-48*) observed in this study could be based on the fact that the inappropriate use of certain antibiotics such as Ceftazidime, Cefotaxime, Ceftriaxone and various carbapenems for the treatment of infections associated with urogenital pathogens is greatly practiced within the area of study. In addition to this, the dissemination of these resistant determinants coding for the production of extended spectrum betalactamase and carbapenemase among the same species or different species in this study area was high. This result agrees with the findings of Yusuf *et al*. (2012).

The high prevalence rate of fluoroquinolones-resistant genes {*gyrA* (50%) and *parC* (83.3%)} and aminoglycosides-resistant genes {*aaC1* (58%) and *aaC2* (50%)}, is based on the fact that the R-plasmids in most urogenital pathogens, also co-harboured genes that code for the resistance of other classes of antibiotics; hence the abilities of these urogenital pathogens in this study, to develop resistance to multiple classes of antibiotics. This result agrees with the findings of Peerayah *et al*. (2016), who reported that the high resistance to gentamicin could be based on the high occurrence of modifying enzymes such as AAC (aminoglycosides acetyltransferase); AHP (aminoglycosides phosphoryltransferase) and ANT (aminoglycosides nucleotidyltransferase) which inactivates aminoglycosides through various modification such as N- acetylation, O-phosphorylation and O-acetylation, while high resistance to ciprofloxacin could be based on the high alteration of fluoroquinolones's target sites (such as the *parC* and *gyrA* enzymes),which is said to occur due to mutations in the

parC and *gyrA* genes loctated in the chromosomes. Similarly, the prevalence rates of aaC6 (13.3%) and aaC3 (20%) were also observed alongside a high prevalence rate of ESBL-producing genes in various bacterial isolates obtained from various clinical samples as reported by Rizi *et al*. (2015).

The phylogenetic tree has shown that the various multi drug resistant strains were closely related to various reference strains (Figure 4.7). This similarity could be based on the fact that all strains (including both the identified and reference strains) were both isolated with same social behaviour of multiple partners throughout their marriage. This result agrees with the findings of Usman (2016).

The phylogenetic tree also revealed that reference strains and the various multi drug resistant bacteria strains in this study had high percentage similarity, same ancestral origin, a close branch distance but different accession numbers, thus indicating disparity in various nucleotide base pairs, in the multidrug resistant bacteria strains and their reference strains in this study. This disparity could be due to certain factors such as mutation (either by point mutation, deletion and insertion) and genetic makeup of the women from whom the bacteria were isolated. This result agrees with findings of Bruni *et al.* (2010) who reported that the genetic makeup of host causes disparity im various strains isolated.

CHAPTER FIVE

5.0 CONCLUSION AND RECOMMENDATIONS

5.1 Conclusion

The findings of this study revealed that high bacterial infection (62%) was observed among the females with pelvic inflammatory disease (PID) studied from various General hospitals in nine Local Government Areas in Niger State. This study also revealed various urogenital isolates, with *Escherichia coli* (22.5%, 24.5%), being the most predominant followed by *Klebsiella pneumoniae* (21.9%, 21.2%), *Salmonella* Typhi (21.6%, 20.8%), *Proteus vulgaris* (12.2%, 13.5%), *Staphylococcus aureus* (11.8%, 10.5%) and *Streptococcus pyogenes* (10%, 9.5%) in endocervical swab and urine respectively. Higher prevalence of bacterial infections in women residing in the rural areas (32.5%), between the ages of 25-29years (24.4%) and who had previous urinary tract infection (UTI) (49.4%) at P=0.00 and significant difference (P<0.05) were also revealed in this study.

Furthermore, the susceptibility study revealed a total of two hundred and twenty eight (228) multidrug resistant bacteria which consisted of 90(28.1%) and 138(34.5%) multidrug resistant bacteria isolated from endocervical and Urine samples respectively. This study revealed that 100% resistance to Sulfamethoxazole and Augmentin were observed in multidrug resistant bacterial isolates obtained from General Hospitals Agaie and Wushishi respectively. In addition, the resistance of 35(15.4%) multidrug resistant bacterial isolates completely resistant to all the antibiotics was also revealed in this study. This study also revealed that multidrug resistant bacteria, resistant to 5 or more antibiotics in both endocervical and urine samples (81.1% and 79.7%), were most prevalent, followed by those resistant to 4 antibiotics (11.1% and 15.2%) and those resistant to 3 antibiotics (7.8% and 5.1%) respectively. This study revealed that there

was a high significant difference between the enzymes, particularly extended spectrum betalactamase ($28.00 \pm 3.03_b^b$, $49.00 \pm 1.80^b{}_d$) and carbapenemase ($24.00 \pm 2.00_b{}^{ab}$, $44.00 \pm 0.00_d{}^b$) produced in *Klebsiella pneumoniae* in both endocervical swab and urine samples compared to other enzymes produced by other multidrug resistant organisms. Similarly, this study also revealed that extended spectrum betalactamase was the most produced enzyme in all the multidrug resistant organisms in this study. This study also revealed that two resistant genes such as *TEM* and *CTX-M* were highly prevalent (91.7%) among the urogenital bacteria.

5.2 Recommendations

It is recommended that:

1. Government health care providers should create awareness to rural women on certain life-threatening diseases.
2. Health care providers should diagnose and prescribe antibiotics properly to avoid re-infection.
3. Drug enforcement agencies should ensure appropriate use of antibiotics (such as third generation Cephalosporins) to avoid the development of resistant organisms.
4. Health care workers should encourage females to practice routine check-ups, to ensure that asymptomatic infections are completely eradicated.
5. New antibiotics and vaccines should be produced and administered properly to completely eradicate the existing resistant bacteria.
6. Further studies on variation analysis on both the identified genes sequences and reference sequences should be done to ascertain the percentage similarity or dissimilarity between them.

5.3 Contribution to Knowledge

This study provided information on the burden of pelvic inflammatory diseases among women (15-54 years) in Niger State. At the end of the study, we have identified the species of bacteria responsible for pelvic inflammatory disease. Similarly, the study has revealed that the identified multidrug resistant (MDR) bacterial isolates from endocervical swabs were: *Klebsiella pneumoniae* (50%), *Escherichia coli* (43.1%) and *Salmonella typhi* (34.8%) while the identified multidrug resistant bacterial isolates from urine samples were: *Klebsiella pneumoniae* (61.2%), *Escherichia coli* (49.0%), *Salmonella typhi* (39.8%) and *Proteus vulgaris* (9.3%). The study also provided that there was a significant difference ($P<0.05$) in the extended spectrum betalactamase ($28.00 \pm 3.03_{b}^{b}$, $49.00 \pm 1.80^{b}_{d}$) and cabarpenemase ($24.00 \pm 2.00_{b}^{ab}$, $44.00 \pm 0.00_{d}^{b}$) produced in *K. pneumoniae* isolated from endocervical swab and urine compared to the extended spectrum betalactamase and cabarpenemase produced by *Escherichia coli*, *Salmonella typhi* and *Proteus vulgaris* isolated from endocervical swab and urine. At the end of the study, we also identified that genotypic determinants such as *TEM* and *CTX-M* were contained in 91.7% of the isolates while *CTX-M1* was contained in 33.3% of the isolates. This study therefore implies that there was a high prevalence of multi drug resistant genes in the study area.

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APPENDICES

APPENDIX A

Table A.1: Cultural Characteristics of Isolates from endocervical swabs (ECS) and Urine from General Hospital Suleja

		Nutrient Agar			MacConkey Agar			Salmonella-Shigella Agar		
SAMPLE	Bacterial Count(cfu/cm^3/ml)	Colour	Texture	Shape	Colour	Texture	Shape	Colour	Texture	Shape
H-1SU	1.0 x10^{-3}	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-2SU	0.9 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-3SU	1.5 x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-4SU	0.4 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-8SU	1.1 x10^{-3}	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-9SU	0.5 x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-11SU	1.0 x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-12SU	1.1 x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-1SU	1.5 x10^{-3}	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-2SU	1.9 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-3SU	2.7 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-4SU	0.9 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-5SU	2.5 x10^{-3}	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-6SU	0.6 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-7SU	1.2 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-9SU	1.0 x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-10SU	2.3 x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-11SU	1.6 x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-12SU	2.0 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular

Table A.2: Biochemical Characteristics of Isolates from ECS and Urine from General Hospital Suleja

Nature of sample	Gram reaction	Cell shape	Motility	Catalase	Coagulase	Starch hydrolysis	Indole production	Urease	Citrate	Oxdase	Hydrogen sulphate	Methylred	Voges proskauer	D-Glucose	Sucrose	Mannitol	Fructose	Species of organism
H-1SU	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-2SU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-3SU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-4SU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-8SU	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-9SU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-11SU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-12SU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-1SU	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-2SU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-3SU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-4SU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-5SU	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-6SU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-7SU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-9SU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-10SU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-11SU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-12SU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*

Table A.3: Cultural Characteristics of Isolates from ECS and Urine from General Hospital Minna

		Nutrient Agar			MacConkey Agar			Salmonella-Shigella Agar		
SAMPLE	Bacterial Count(cfu/cm^3/ml)	Colour	Texture	Shape	Colour	Texture	Shape	Colour	Texture	Shape
H-7M	1.5 x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-9M	0.9 x10^{-3}	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-14M	0.5 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-15M	0.6 x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-16M	1.4 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-17M	0. 6x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-1M	1.9 x10^{-3}	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-2M	0.7 x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-3M	2.6 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-5M	1.8 x10^{-3}	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-8M	0.6 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-9M	1.4 x10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-12M	0.5 x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-16M	2.4 x10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular

Table A.4: Biochemical Characteristics of Isolates from ECS and Urine from General Hospital Minna

Nature of sample	Gram reaction	Cell shape	Motility	Catalase	Coagulase	Starch hydrolysis	Indole production	Urease	Citrate	Oxdase	Hydrogen sulphate	Methylred	Voges proskauer	D-Glucose	Sucrose	Mannitol	Fructose	Species of organism
H-7M	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-9M	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-14M	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-15M	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-16M	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-17M	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-1M	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-2M	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-3M	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-5M	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-8M	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-9M	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-12M	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-16M	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*

Table A.5: Cultural Characteristics of Isolates from ECS and Urine from General Hospital Kuta

		Nutrient Agar			MacConkey Agar			Salmonella-Shigella Agar		
SAMPLE	Bacterial count(cfu/cm^3/ml)	Colour	Texture	Shape	Colour	Texture	Shape	Colour	Texture	Shape
H-2KU	2.6×10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-3KU	2.8×10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-4KU	2.7×10^{-3}	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-5KU	1.9×10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-6KU	2.1×10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-7KU	2.2×10^{-3}	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-8KU	2.3×10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-10KU	1.8×10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-11KU	1.7×10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-12KU	2.1×10^{-3}	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-1KU	3.8×10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-2KU	3.5×10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-4KU	3.1×10^{-3}	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-5KU	2.4×10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-6KU	3.6×10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-7KU	2.5×10^{-3}	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-8KU	3.0×10^{-3}	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-10KU	2.3×10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-11KU	2.1×10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-12KU	2.6×10^{-3}	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular

Table A.6: Biochemical Characteristics of Isolates from ECS and Urine from General Hospital Kuta

Nature of sample	Gram reaction	Cell shape	Motility	Catalase	Coagulase	Starch hydrolysis	Indole production	Urease	Citrate	Oxdase	Hydrogen sulphate	Methylred	Voges proskauer	D-Glucose	Sucrose	Mannitol	Fructose	Species of organism
H-2KU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-3KU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-4KU	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-5KU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-6KU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-7KU	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-8KU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-10KU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-11KU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-12KU	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-1KU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-2KU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-4KU	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-5KU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-6KU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-7KU	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-8KU	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-10KU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-11KU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-12KU	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*

Table A.7: Cultural Characteristics of Isolates from ECS and Urine from General Hospital Bida

		Nutrient Agar			MacConkey Agar			Salmonella-Shigella Agar		
SAMPLE	Bacterial count(cfu/cm^3/ml)	Colour	Texture	Shape	Colour	Texture	Shape	Colour	Texture	Shape
H-1B	$2.8x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-2B	$2.7x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-3B	$2.4x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-4B	$2.1x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-5B	$2.2x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-10B	$2.5x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-11B	$1.9x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-12B	$2.3x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-13B	$2.4x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-16B	$2.7x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-18B	$3.2x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-19B	$3.1x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-20B	$2.7x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-1B	$3.5\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-2B	$3.7\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-3B	$2.9\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-4B	$2.8\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-5B	$3.1\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-6B	$3.5\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-7B	$4.1\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-8B	$3.3\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-9B	$2.8\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-11B	$2.5\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-13B	$3.0\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-14B	$3.4\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-15B	$3.6\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-17B	$2.9\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-18B	$4.2\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-19B	$4.3\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-20B	$3.6\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-21B	$2.9\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-22B	$3.7\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-23B	$3.2\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular

Table A.8: Biochemical Characteristics of Isolates from ECS and Urine from General Hospital Bida

Nature of sample	Gram reaction	Cell shape	Motility	Catalase	Coagulase	Starch hydrolysis	Indole production	Urease	Citrate	Oxdase	Hydrogen sulphate	Methylred	Voges proskauer	D-Glucose	Sucrose	Mannitol	Fructose	Species of organism
H-1B	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-2B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-3B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-4B	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-5B	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-10B	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-11B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-12B	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-13B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-16B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-18B	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-19B	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-20B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-1B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-2B	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-3B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-4B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-5B	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-6B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-7B	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-8B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-9B	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-11B	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-13B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-14B	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-15B	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-17B	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-18B	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-19B	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-20B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-21B	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-22B	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-23B	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*

Table A.9: Cultural Characteristics of Isolates from ECS and Urine from General Hospital Agaie

		Nutrient Agar			MacConkey Agar			Salmonella-Shigella Agar		
SAMPLE	Bacterial count(cfu/cm^3/ml)	Colour	Texture	Shape	Colour	Texture	Shape	Colour	Texture	Shape
H-1AG	$2.2x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-3AG	$2.4x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-4AG	$2.6x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-5AG	$2.0x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-7AG	$2.3x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-10AG	$2.9x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-14AG	$2.2x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-19AG	$3.0x10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-20AG	$2.4x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-1AG	$2.7\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-2AG	$2.9\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-3AG	$3.9\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-4AG	$3.2\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-5AG	$3.0\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-6AG	$2.8\ x10^{-3}$	White	Viscid	Irregular	Colourless	Viscid	Irregular	Black pigment	Viscid	Circular
U-8AG	$3.1\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-9AG	$2.9\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-10AG	$3.5\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-11AG	$3.7\ x10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-12AG	$3.3\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-14AG	$3.6\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-15AG	$3.5\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-16AG	$3.1\ x10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-17AG	$3.6\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-18AG	$3.7\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-19AG	$3.8\ x10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-20AG	$3.1\ x10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-21AG	$3.2\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-22AG	$3.0\ x10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular

Table A.10: Biochemical Characteristics of Isolates from ECS and Urine from General Hospital Agaie

Nature of sample	Gram reaction	Cell shape	Motility	Catalase	Coagulase	Starch hydrolysis	Indole	Urease	Citrate	Oxdas	Hydrogen sulphate	Methylred	Voges proskauer	D-Glucose	Sucrose	Mannitol	Fructose	Species of organism
H-1AG	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-3AG	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-4AG	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-5AG	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-7AG	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-10AG	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-14AG	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-19AG	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-20AG	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-1AG	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-2AG	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-3AG	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-4AG	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-5AG	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-6AG	-	Rod	+	+	-	-	+	+	+	-	+	+	-	+	+	+	+	*Proteus vulgaris*
U-8AG	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-9AG	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-10AG	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-11AG	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-12AG	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-14AG	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-15AG	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-16AG	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-17AG	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-18AG	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-19AG	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-20AG	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*

U-21AG	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-22AG	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*

Table A.11: Cultural Characteristics of Isolates from ECS and Urine from General Hospital Lapai

		Nutrient Agar			MacConkey Agar			Salmonella-Shigella Agar		
SAMPLE	Bacterial count(cfu/cm^3/ml)	Colour	Texture	Shape	Colour	Texture	Shape	Colour	Texture	Shape
H-1L	$3.4x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-2L	$3.3x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-3L	$3.2x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-4L	$3.1x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-5L	$2.9x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-6L	$3.0x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-7L	$2.8x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-8L	$3.0x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-9L	$2.5x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-10L	$2.8x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-11L	$2.9x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-12L	$3.2x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-13L	$3.0x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-14L	$3.3x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-15L	$3.1x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-16L	$3.0x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-17L	$2.8x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-18L	$3.2x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-3L	$4.1\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-6L	$2.1\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-7L	$3.0\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-8L	$2.0\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-10L	$1.8\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-14L	$1.9\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-15L	$2.4\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-17L	$3.3\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-25L	$3.9\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-28L	$3.4\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-29L	$4.2\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-32L	$3.6\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-34L	$3.5\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-36L	$4.1\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-37L	$4.0\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-38L	$3.5\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-39L	$3.6\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-40L	$2.9\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular

Table A.12: Biochemical Characteristics of Isolates from ECS and Urine from General Hospital Lapai

Nature of sample	Gram reaction	Cell shape	Motility	Catalase	Coagulase	Starch hydrolysis	Indole production	Urease	Citrate	Oxdase	Hydrogen sulphate	Methylred	Voges proskauer	D-Glucose	Sucrose	Mannitol	Fructose	Species of organism
H-1L	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-2L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-3L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-4L	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-5L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-6L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-7L	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-8L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-9L	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-10L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-11L	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-12L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-13L	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-14L	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-15L	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-16L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-17L	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-18L	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-3L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-6L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-7L	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-8L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-10L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-14L	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-15L	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-17L	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-25L	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-28L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-29L	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-32L	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-34L	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-36L	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-37L	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-38L	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-39L	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-40L	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*

Table A.13: Cultural Characteristics of Isolates from ECS and Urine from General Hospital Nasko

		Nutrient Agar			MacConkey Agar			Salmonella-Shigella Agar		
SAMPLE	Bacterial count(cfu/cm^3/ml)	Colour	Texture	Shape	Colour	Texture	Shape	Colour	Texture	Shape
H-2N	$3.2x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-8N	$3.0x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-12N	$2.9x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-17N	$2.7x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-18N	$2.9x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-19N	$3.0x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-20N	$2.5x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-22N	$2.8x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-1N	$3.4\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-3N	$2.5\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-4N	$3.0\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-6N	$3.1\ x10^{-3}$	White	Viscid	Irregular	colourless	Viscid	Irregular	Black pigment	Viscid	Circular
U-7N	$2.5\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-8N	$2.7\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-9N	$3.9\ x10^{-3}$	White	Viscid	Irregular	colourless	Viscid	Irregular	Black pigment	Viscid	Circular
U-10N	$4.0\ x10^{-3}$	White	Viscid	Irregular	colourless	Viscid	Irregular	Black pigment	Viscid	Circular
U-12N	$3.8\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-14N	$3.7\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-15N	$3.2\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-16N	$3.5\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-18N	$3.3\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-20N	$3.1\ x10^{-3}$	White	Viscid	Irregular	colourless	Viscid	Irregular	Black pigment	Viscid	Circular
U-21N	$3.8\ x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-22N	$3.5\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular

Table A.14: Biochemical Characteristics of Isolates from ECS and Urine from General Hospital Nasko

Nature of sample	Gram reaction	Cell shape	Motility	Catalase	Coagulase	Starch hydrolysis	Indole production	Urease	Citrate	Oxdase	Hydrogen sulphate	Methylred	Voges proskauer	D-Glucose	Sucrose	Mannitol	Fructose	Species of organism
H-2N	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	Salmonella spp
H-8N	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	Klebsiella spp
H-12N	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	Salmonella spp
H-17N	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	Klebsiella spp
H-18N	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	Klebsiella spp
H-19N	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	Salmonella spp
H-20N	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	Escherichia coli
H-22N	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	Escherichia coli
U-1N	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	Klebsiella spp
U-3N	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	Klebsiella spp
U-4N	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	Klebsiella spp
U-6N	-	Rod	+	+	-	-	+	+	+	-	+	+	-	+	+	+	+	Proteus vulgaris
U-7N	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	Salmonella spp
U-8N	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	Klebsiella spp
U-9N	-	Rod	+	+	-	-	+	+	+	-	+	+	-	+	+	+	+	Proteus vulgaris
U-10N	-	Rod	+	+	-	-	+	+	+	-	+	+	-	+	+	+	+	Proteus vulgaris
U-12N	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	Salmonella spp
U-14N	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	Escherichia coli
U-15N	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	Salmonella spp
U-16N	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	Escherichia coli
U-18N	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	Klebsiella spp
U-20N	-	Rod	+	+	-	-	+	+	+	-	+	+	-	+	+	+	+	Proteus vulgaris
U-21N	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	Salmonella spp
U-22N	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	Escherichia coli

Table A.15: Cultural Characteristics of Isolates from ECS and Urine from General Hospital Kontagora

		Nutrient Agar			MacConkey Agar			Salmonella-Shigella Agar		
SAMPLE	Bacterial count(cfu/ml)	Colour	Texture	Shape	Colour	Texture	Shape	Colour	Texture	Shape
H-1KN	$3.1x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-2KN	$3.2x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-13KN	$3.4x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-15KN	$3.1x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-27KN	$2.5x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-31KN	$3.3x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-39KN	$3.6x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-42KN	$3.2x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-27KN	$3.4 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-28KN	$4.1 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-29KN	$5.0 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-34KN	$5.5 x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-36KN	$4.5 x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-38KN	$3.9 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-39KN	$4.7 x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-40KN	$3.9 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-42KN	$6.0 x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-43KN	$4.7 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-46KN	$4.1 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-47KN	$3.4 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-52KN	$3.5 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-56KN	$4.1 x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-57KN	$3.2 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-59KN	$5.2 x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-63KN	$5.1 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-64KN	$5.0 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-65KN	$4.9 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-66KN	$4.7 x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-67KN	$4.6 x10^{-3}$	White	Viscid	Circular	colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-68KN	$3.5 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-71KN	$3.7 x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-73KN	$4.4 x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-75KN	$4.5 x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular

Table A.16: Biochemical Characteristics of Isolates from ECS and Urine from General Hospital Kontagora

Nature of sample	Gram reaction	Cell shape	Motility	Catalase	Coagulase	Starch hydrolysis	Indole production	Urease	Citrate	Oxdase	Hydrogen sulphate	Methylred	Voges	D-Glucose	Sucrose	Mannitol	Fructose	Species of organism
H-1KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-2KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-13KN	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-15KN	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-27KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-31KN	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-39KN	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-42KN	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-27KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-28KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-29KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-34KN	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-36KN	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-38KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-39KN	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-40KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-42KN	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-43KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-46KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-47KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-52KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-56KN	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-57KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-59KN	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-63KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*

U-64KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-65KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-66KN	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-67KN	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-68KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-71KN	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-73KN	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-75KN	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*

Table A.17: Cultural Characteristics of Isolates from ECS and Urine from General Hospital Wushishi

		Nutrient Agar			MacConkey Agar			Salmonella-Shigella Agar		
SAMPLE	Bacterial count(cfu/ml)	Colour	Texture	Shape	Colour	Texture	Shape	Colour	Texture	Shape
H-2W	$2.1x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-6W	$2.5x\ 10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-10W	$2.8x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-12W	$3.1x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-15W	$3.2x10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-18W	$3.0\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
H-22W	$2.8x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-24W	$2.5x10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
H-26W	$3.1x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
H-28W	3.3x10-3	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-1W	$4.5\ x10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-7W	$3.5\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-8W	$2.9\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-11W	$3.7\ x10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-13W	$3.0\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-15W	$4.1\ x10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-16W	$4.7\ x10^{-3}$	White	Viscid	Circular	Colourless	Viscid	Circular	Black pigment	Viscid	Circular
U-21W	$3.9\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular
U-22W	$4.2\ x10^{-3}$	Cream	Viscid	Circular	Pink	Viscid	Circular	Pink	Viscid	Circular
U-28W	$4.3\ x10^{-3}$	White	Mucoid	Irregular	Pink	Mucoid	Irregular	Pink	Mucoid	Irregular

Table A.18: Biochemical Characteristics of Isolates from ECS and Urine from General Hospital Wushishi

Nature of sample	Gram reaction	Cell shape	Motility	Catalase	Coagulase	Starch hydrolysis	Indole production	Urease	Citrate	Oxdase	Hydrogen sulphate	Methylred	Voges proskauer	D-Glucose	Sucrose	Mannitol	Fructose	Species of organism
H-2W	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-6W	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-10W	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-12W	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-15W	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-18W	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
H-22W	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-24W	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
H-26W	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
H-28W	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-1W	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-7W	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-8W	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-11W	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-13W	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-15W	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-16W	-	Rod	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	*Salmonella spp*
U-21W	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*
U-22W	-	Rod	+	+	-	-	+	-	-	-	-	+	-	+	+	+	+	*Escherichia coli*
U-28W	-	Rod	-	+	-	-	-	+	+	-	-	-	+	+	+	-	+	*Klebsiella spp*

APPENDIX B

Clinical laboratory standard institute interpretative index

S/N	Antibiotics	Susceptible	Intermediate	Resistance
1	Ofloxacin	≥ 16	13-15	≤ 12
2	Perfloxacin	≥ 21	16-20	≤ 15
3	Ciprofloxacin	≥ 21	16-20	≤ 15
4	Augumentin	≥ 18	14-17	≤ 13
5	Gentamicin	≥ 15	13-14	≤ 12
6	Streptomycin	≥ 21	15-20	≤ 14
7	Cephalexin	≥ 18	15-17	≤ 14
8	Nalidixic acid	≥ 19	14-18	≤ 13
9	Sulfamethoxazole	≥ 16	11-15	≤ 10
10	Ampicillin	≥ 17	14-16	≤ 13

APPENDIX C

Genetic identification of four multidrug resistant Isolates

Appendix C.1 to C.12 shows the sequences used to confirm the selected multidrug resistant bacteria as; *E.coli*, *Klebsiella pneumoniae*, *Salmonella typhi* and *Proteus vulgaris* (as seen below) after the amplification of the 16s rRNA region was carried out.

Table C.1: MT920648 *Escherichia coli* strain TR-1

AGTTTGATCATGGCTCAGATTGAACGCTGGCGGCAGGCCTAACACATGCAAGTCGA
ACGGTAACAGGAAGCAGCTTGCTGCTTTGCTGACGAGTGGCGGACGGGTGAGTAAT
GTCTGGGAAACTGCCTGATGGAGGGGGATAACTACTGGAAACGGTAGCTAATACCG
CATAACGTCGCAAGACCAAAGAGGGGGACCTTCGGGCCTCTTGCCATCGGATGTGC
CCAGATGGGATTAGCTAGTAGGTGGGGTAACGGCTCACCTAGGCGACGATCCCTAG
CTGGTCTGAGAGGATGACCAGCCACACTGGAACTGAGACACGGTCCAGACTCCTAC
GGGAGGCAGCAGTGGGGAATATTGCACAATGGGCGCAAGCCTGATGCAGCCATGCC
GCTGTATGAAGAAGGCCTTCGGGTTGTAAAGTACTTTCAGCGGGGAGGAAGGGAGT
AAAGTTAATACCTTTGCTCATTGACGTTACCCGCAGAAGAAGCACCGGCTAACTCCG
TGCCAGCAGCCGCGGTAATACGGAGGGTGCAAGCGTTAATCGGAATTACTGGGCGT
AAAGCGCACGCAGGCGGTTTGTTAAGTCAGATGTGAAATCCCCGGGCTCAACCTGG
GAACTGCATCTGATACTGGCAAGCTTGAGTCTCGTAGAGGGGGGTAGAATTCCAGT
GTAGCGGTGAAATGCGTAGAGATCTGGAGGAATACCGGTGGCGAAGGCGGCCCCCT
GGACGAAGACTGACGCTCAGGTGCGAAAGCGTGGGGAGCAAACAGGATTAGATAC
CCTGGTAGTCCACGCCGTAAACGATGTCGACTTGGAGGTTGTGCCCTTGAGGCGTGG
CTTCCGGAGCTAACGCGTTAAGTCGACCGCCTGGGGAGTACGGCCGCAAGGTTAAA
ACTCAAATGAATTGACGGGGGCCCGCACAAGCGGTGGAGCATGTGGTTTAATTCGA
TGCAACGCGAAGAACCTTACCTGGTCTTGACATCCACAGAACTTTCCAGAGATGGAT
TGGTGCCTTCGGGAACTGTAGACAGGTGCTGCATGGCTGTCGTCAGCTCGTGTTGTG
AAATGTTGGGTTAAGTCCCGCAACGAGCGCAACCCTTATCCTTTGTTGCCAGCGGTC
CGGCCGGGAACTCAAAGGAGACTGCCAGTGATAAACTGGAGGAAGGTGGGGATGA
CGTCAAGTCATCATGGCCCTTACGACCAGGGCTACACACGTGCTACAATGGCGCATA
CAAAGAGAAGCGACCTCGCGAGAGCAAGCGGACCTCATAAAGTGCGTCGTAGTCCG
GATTGGAGTCTGCAACTCGACTCCATGAAGTCGGAATCGCTAGTAATCGTGGATCAG
AATGCCACGGTGAATACGTTCCCGGGCCTTGTACACACCGCCCGTCACACCATGGGA
GTGGGTTGCAAAAGAAGTAGGTAGCTTAACTTCGG

Table C.2 B: MT920649 *Escherichia coli* strain TR-2

AGTTTGATCATGGCTCAGATTGAACGCTGGCGGCAGGCCTAACACATGCAAGTCGA
ACGGTAACAGGAAGCAGCTTGCTGCTTTGCTGACGAGTGGCGGACGGGTGAGTAAT
GCTGGGAAACTGCCTGATGGAGGGGGATAACTACTGGAAACGGTAGCTAATACCGC
ATAACGTCGCAAGACCAAAGAGGGGGACCTTCGGGCCTCTTGCCATCGGATGTGCC
CAGATGGGATTAGCTAGTAGGTGGGGTAACGGCTCACCTAGGCGACGATCCCTAGC
TGGTCTGAGAGGATGACCAGCCACACTGGAACTGAGACACGGTCCAGACTCCTACG
GGAGGCAGCAGTGGGGAATATTGCACAATGGGCGCAAGCCTGATGCAGCCATGCCG
CGTGTATGAAGAAGGCCTTCGGGTTGTAAAGTACTTTCAGCGGGGAGGAAGGGAGT
AAAGTTAATACCTTTGCTCATTGACGTTACCCGCAGAAGAAGCACCGGCTAACTCCG
TGCCAGCAGCCGCGGTAATACGGAGGGTGAAGCGTTAATCGGAATTACTGGGCGTA
AAGCGCACGCAGGCGGTTTGTTAAGTCAGATGTGAAATCCCCGGGCTCAACCTGGG
AACTGCATCTGATACTGGCAAGCTTGAGTCTCGTAGAGGGGGGTAGAATTCCAGGT
GTAGCGGTGAAATGCGTAGAGATCTGGAGGAATACCGGTGGCGAAGGCGGCCCCCT
GGACGAAGACTGACGCTCAGGTGCGAAAGCGTGGGGAGCAAACAGGATTAGATAC
CCTGGTAGTCCACGCCGTAAACGATGTCGACTTGGAGGTTGTGCCCTTGAGGCGTGG
CTTCCGGAGCTAACGCGTTAAGTCGACCGCCTGGGGAGTACGGCCGCAAGGTTAAA
ACTCAAATGAATTGACGGGGGCCCGCACAAGCGGTGGAGCATGTGGTTTAATTCGA
TGCAACGCGAAGAACCTTACCTGGTCTTGACATCCACAGAACTTTCCAGAGATGGAT
TGGTGCCTTCGGGAACTGTGAGACAGGTGCTGCATGGCTGTCTAGCAGCTCGTGTTG
TGAAATGTTGGGTTAAGTCCCGCAACGAGCGCAACCCTTATCCTTTGTTGCCAGCGG
TCCGGCCGGGAACTCAAAGGAGACTGCCAGTGATAAACTGGAGGAAGGTGGGGATG
ACGTCAAGTCATCATGGCCCTTACGACCAGGGCTACACACGTGCTACAATGGCGCAT
ACAAAGAGAAGCGACCTCGCGAGAGCAAGCGGACCTCATAAAGTGCGTCGTAGTCC
GGATTGGAGTCTGCAACTCGACTCCATGAAGTCGGAATCGCTAGTAATCGTGGATCA
GAATGCCACGGTGAATACGTTCCCGGGCCTTGTACACACCGCCCGTCACACCATGGG
AGTGGGTTGCAAAAGAAGTAGGTAGCTTAACTTCGG

Table C.3: MT920650 *Escherichia coli* strain TR-9

ACTCTTGCTGCTTTGCTGACGAGTGGCGGACGGGTGAGTAATGTCTGGGAAACTGCC
TGATGGAGGGGGATAACTACTGGAAACGGTAGCTAATACCGCATAACGTCGCAAGA
CCAAAGAGGGGGACCTTCGGGCCTCTTGCCATCGGATGTGCCCAGATGGGATTAGCT
AGTAGGTGGGGTAACGGCTCACCTAGGCGACGATCCCTAGCTGGTCTGAGAGGATG
ACCAGCCACACTGGAACTGAGACACGGTCCAGACTCCTACGGGAGGCAGCAGTGGG
GAATATTGCACAATGGGCGCAAGCCTGATGCAGCCATGCCGCGTGTATGAAGAAGG
CCTTCGGGTTGTAAAGTACTTTCAGCGGGGAGGAAGGGAGTAAAGTTAATACCTTTG
CTCATTGACGTACCCGCAGAAGAAGCACCGGCTAACTCCGTGCCAGCAGCCGCGGT
AATACGGAGGGTGCAAGCGTTAATCGGAATTACTGGGCGTAAAGCGCACGCAGGCG
GTTTGTTAAGTCAGATGTGAAATCCCCGGGCTCAACCTGGGAACTGCATCTGATACT
GGCAAGCTTGAGTCTCGTAGAGGGGGGTAGAATTCCAGGTGTAGCGGTGAAATGCG
TAGAGATCTGGAGGAATACCGGTGGCGAAGGCGGCCCCCTGGACGAAGACTGACGC

TCAGGTGCGAAAGCGTGGGGAGCAAACAGGATTAGATACCCTGGTAGTCCACGCCG
TAAACGATGTCGACTTGGAGGTTGTGCCCTTGAGGCGTGGCTTCCGGAGCTAACGCG
TTAAGTCGACCGCCTGGGGAGTACGGCCGCAAGGTTAAAACTCAAATGAATTGACG
GGGGCCCGCACAAGCGGTGGAGCATGTGGTTTAATTCGATGCAACGCGAAGAACCT
TACCTGGTCTTGACATCCACAGAACTTTCCAGAGATGGATTGGTGCCTTCGGGAACT
GTGAGACAGGTGCTGCATGGCTGTCGTCAGCTCGTGTTGTGAAATGTTGGGTTAAGT
CCCGCAACGAGCGCAACCCTTATCCTTTGTTGCCAGCGGTCCGGCCGGGAACTCAAA
GGAGACTGCCAGTGATAAACTGGAGGAAGGTGGGATGACGTCAAGTCATCATGGCC
CTTACGACCAGGGCTACACACGTGCTACAATGGCGCATACAAAGAGAAGCGACCTC
GCGAGAGCAAGCGGACCTCATAAAGTGCGTCGTCTAAT

Table C.4: MT920651 *Salmonella enterica* strain TR-5

AACGATTGAACGCTGGCGGCAGGCCTAACACATGCAAGTCGAACGGTAACAGGAAG
CAGCTTGCTGCTTTGCTGACGAGTGGCGGACGGGTGAGTAATGTCTGGGAAACTGCC
TGATGGAGGGGGATAACTACTGGAAACGGTGGCTAATACCGCATAACGTCGCAAGA
CCAAAGAGGGGGACCTTCGGGCCTCTTGCCATCAGATGTGCCCAGATGGGATTAGCT
TGTTGGTGAGGTAACGGCTCACCAAGGCGACGATCCCTAGCTGGTCTGAGAGGATG
ACCAGCCACACTGGAACTGAGACACGGTCCAGACTCCTACGGGAGGCAGCAGTGGG
GAATATTGCACAATGGGCGCAAGCCTGATGCAGCCATGCCGCGTGTATGAAGAAGG
CCTTCGGGTTGTAAAGTACTTTCAGCGGGGAGGAAGGTGTTGTGGTTAATAACCGCA
GCAATTGACGTTACCCGCGAAGAAGCACCGGCTAACTCCGTGCCAGCAGCCGCGGT
AATACGGAGGGTGCAAGCGTTAATCGGAATTACTGGGCGTAAAGCGCACGCAGGCG
GTCTGTCAAGTCGGATGTGAAATCCCCGGGCTCAACCTGGGAACTGCATTCGAAACT
GGCAGGCTTGAGTCTTGTAGAGGGGGGTAGAATTCCAGGTGTAGCGGTGAAATGCG
TAGAGATTGGAGGAATACCGGTGGCGAAGGCGGCCCCCTGGACAAAGACTGACGCT
CAGGTGCGAAAGCGTGGGGAGCAAACAGGATTAGATACCCTGGTAGTCCACGCCGT
AAACGATGTCTACTTGGAGGTTGTGCCCTTGAGGGTGGCTTCCGGAGCTAACGCGTT
AAGTAGACCGCCTGGGGAGTACGGCCGCAAGGTTAAAACTCAAATGCAATTGACGG
GGGCCCGCACAAGCGGTGGAGCATGTGGTTTAATTCGATGCAACGCGAAGAACCTT
ACCTGGTCTTGACATCCACAGAACTTTCCAGAGATGGATTGGTGCCTTCGGGAACTG
TGAGACAGGTGCTGCATGGCTGTCGTCAGCTCGTGTTGTGAAATGTTGGGTTAAGTC
CCGCAACGAGCGCAACCCTTATCCTTTGTTGCAGCGGTCCGGCCGGGAACTCAAAG
GAGACTGCCAGTGATAAACTGGAGGAAGGTGGGGATGACGTCAAGTCATCATGGCC
CTTACGACCAGGGCTACACACGTGCTACAATGGCGCATACAAAGAGAAGCGACCTC
GCGAGAGCAAGCGGACCTCATAAAGTGCGTCGTAGTCCGGATTGGAGTCTGCAACT
CGACTCCATGAAGTCGGAATCGCTAGTAATCGTGGATCAGAATGCCACGGTGAATA
CGTTCCCGGGCCTTGTACACACCGCCCGTCACACCATGGGAGTGGGTTGCAAAAGA
AGTAGGTAGCTTAACCTTCGGGGGGGCGCTTACCACTTTGTGATTCATGACTGGGGT
GAAGTCGTAACAAGGTAACCGTAGCAT

Table C.5: MT920652 *Salmonella enterica* strain TR-6

ATGGAGGCCTAACACATGCAAGTCGAACGGTAACAGGAAGCAGCTTGCTGCTTTGC
TGACGAGTGGCGGACGGGTGAGTAATGTCTGGGAAACTGCCTGATGGAGGGGGATA
ACTACTGGAAACGGTGGCTAATACCGCATAACGTCGCAAGACCAAAGAGGGGGACC
TTCGGGCCTCTTGCCATCAGATGTGCCCAGATGGGATTAGCTTGTTGGTGAGGTAAC
GGCTCACCAAGGCGACGATCCCTAGCTGGTCTGAGAGGATGACCAGCCACACTGGA
ACTGAGACACGGTCCAGACTCCTACGGGAGGCAGCAGTGGGGAATATTGCACAATG
GGCGCAAGCCTGATGCAGCCATGCCGCGTGTATGAAGAAGGCCTTCGGGTTGTAAA
GTACTTTCAGCGGGGAGGAAGGTGTTGTGGTTAATAACCGCAGCAATTGACGTTACC
CGCAGAAGAAGCACCGGCTAACTCCGTGCCAGCAGCCGCGGTAATACGGAGGGTGC
AAGCGTTAATCGGAATTACTGGGCGTAAAGCGCACGCAGGCGGTCTGTCAAGTCGG
ATGTGAAATCCCCGGGCTCAACCTGGGAACTGCATTCGAAACTGGCAGGCTTGAGTC
TTGTAGAGGGGGGTAGAATTCCAGGTGTAGCGGTGAAATGCGTGAGATCTGGAGGA
ATACCGGTGGCGAAGGCGGCCCCCTGGACAAAGACTGACGCTCAGGTGCGAAAGCG
TGGGGAGCAAACAGGATTAGATACCCTGGTAGTCCACGCCGTAAACGATGTCTACTT
GGAGGTTGTGCCCTTGAGGCGTGGCTTCCGGAGCTAACGCGTTAAGTAGACCGCCTG
GGGAGTACGGCCGCAAGGTTAAAACTCAAATGAATTGACGGGGGCCCGCACAAGCG
GTGGAGCATGTGGTTTAATTCGATGCAACGCGAAGAACCTTACCTGGTCTTGACATC
CACAGAACTTTCCAGAGATGGATTGGTGCCTTCGGGAACTGTGAGACAGGTGCGTG
CATGGCTGTCGTCAGCTCGTGTTGTGAAATGTTGGGTTAAGTCCCGCAACGAGCGCA
ACCCTTATCCTTGTTGCCAGCGGTCCGGCCGGGAACTCAAAGGAGACTGCCAGTGAT
AAACTGGAGGAAGGTGGGGATGAGTCAAGTCATCATGGCCCTTACGACCAGGGCTA
CACACGTGCTACAATGGCGCATACAAAGAGAAGCGACTCGCGAGAGCAAGCGGAC
CTCATAAAGTGCGTCGTAGTCCGGATTGGAGTCTGCAACTCGACTCCATGAAGTCGG
AATCGCTAGTAATCGTGGATCAGAATGCCACGGTGAATACGTTCCCGGGCCTTGTAC
ACACCGCCCGTCACACCATGGGAGTGGGTTGCAAAAGAAGTAGGTAGCTTAACCTT
CGGGAGGGCGCTTACCACTTTGTGATTCATGACTGGGGTGAAGTCGTAACAAGGTA
ACCGTAGGGGAACCTGCGGTTGGATAGTC

Table C.6: MT920653 *Salmonella enterica* strain TR-11

CCATTGAACGCTGGCGGCAGGCCTAACACATGCAAGTCGAACGGTAACAGGAAGCA
GCTTGCTGCTTTGCTGACGAGTGGCGGACGGGTGAGTAATGTCTGGGAAACTGCCTG
ATGGAGGGGGATAACTACTGGAAAAGGTGGCTAATACCGCATAACGTCGCAAGACC
AAAGAGGGGGACCTTCGGGCCTCTTGCCATCAGATGTGCCCAGATGGGATTAGCTTG
TTGGTGAGGTAACGGCTCACCAAGGCGACGATCCCTAGCTGGTCTGAGAGGATGAC
CAGCCACACTGGAACTGAGACACGGTCCAGACTCCTACGGGAGGCAGCAGTGGGGA
ATATTGCACAATGGGCGCAAGCCTGATGCAGCCATGCCGCGTGTATGAAGAAGGCC
TTCGGGTTGTAAAGTACTTTCAGCGGGGAGGAAGGTGTTGTGGTTAATAACCGCAGC
AATTGACGTTACCCGCGAAGAAGCACCGGCTAACTCCGTGCCAGCAGCCGCGGTAA
TACGGAGGGTGCAAGCGTTAATCGGAATTACTGGGCGTAAAGCGCACGCAGGCGGT
CTGTCAAGTCGGATGTGAAATCCCCGGGCTCAACCTGGGAACTGCATTCGAAACTGG

CAGGCTTGAGTCTTGTAGAGGGGGGTAGAATTCCAGGTGTAGCGGTGAAATGCGTA
GAGATTGGAGGAATACCGGTGGCGAAGGCGGCCCCCTGGACAAAGACTGACGCTCA
GGTGCGAAAGCGTGGGGAGCAAACAGGATTAGATACCCTGGTAGTCCACGCCGTAA
ACGATGTCTACTTGGAGGTTGTGCCCTTGAGGGTGGCTTCCGGAGCTAACGCGTTAA
GTAGACCGCCTGGGGAGTACGGCCGCAAGGTTAAAACTCAAATGCAATTGACGGGG
GCCCGCACAAGCGGTGGAGCATGTGGTTTAATTCGATGCAACGCGAAGAACCTTAC
CTGGTCTTGACATCCACAGAACTTTCCAGAGATGGATTGGTGCCTTCGGGAACTGTG
AGACAGGTGCTGCATGGCTGTCGTCAGCTCGTGTTGTGAAATGTTGGGTTAAGTCCC
GCAACGAGCGCAACCCTTATCCTTTGTTGCAGCGGTCCGGCCGGGAACTCAAAGGA
GACTGCCAGTGATAAACTGGAGGAAGGTGGGGATGACGTCAAGTCATCATGGCCCT
TACGACCAGGGCTACACACGTGCTACAATGGCGCATACAAAGAGAAGCGACCTCGC
GAGAGCAAGCGGACCTCATAAAGTGCGTCGTAGTCCGGATTGGAGTCTGCAACTCG
ACTCCAAGAAGTCGGAATCGCTAGTAATCGTGGATCAGAATGCCACGGTGAATACG
TTCCCGGGCCTTGTACACACCCCCGTCACACCATGGGAGTGGGTTGCAAAAGAAGT
AGGTAGCTTAACCTTCGGGGGGGCGCTTACCACTTTGTGATTCATGACTGGGGTGAA
GTCGTAACAAGGTAACCGTAGG

Table C.7: MT920654 *Klebsiella pneumoniae* strain TR-3

TATGGTAGCACAGAGAGCTTGCTCTCGGGTGACGAGCGGCGGACGGGTGAGTAATG
TCTGGGAAACTGCCTGATGGAGGGGGATAACTACTGGAAACGGTAGCTAATACCGC
ATAACGTCGCAAGACCAAAGTGGGGGACCTTCGGGCCTCATGCCATCAGATGTGCC
CAGATGGGATTAGCTAGTAGGTGGGGTAACGGCTCACCTAGGCGACGGTCCCTAGC
TGGTCTGAGGGATGACCAGCCACACTGGAACTGAGACACGGTCCAGACTCCTACGG
GAGGCAGCAGTGGGGAATATTGCACAATGGGCGCAAGCCTGATGCAGCCATGCCGC
GTGTGTGAAGAAGGCCTTGGGTTGTAAAGCACTTTCAGCGGGGAGGAAGGCGGTGA
GGTTAATAACCTCATCGATTGACGTTACCCGCAGAAGAAGCACCGGCTAACTCCGTG
CCAGCAGCCGCGGTAATACGGAGGGTGCAAGCGTTAATCGGAATTACTGGGCGTAA
AGCGCACGCAGGCGGTCTGTCAAGTCGGATGTGAAATCCCCGGGCTCAACCTGGGA
ACTGCATTCGAAACTGGCAGGCTAGAGTCTTGTAGAGGGGGGTAGAATTCCAGGTG
TAGCGGTGAAATGCGTAGAGATCTGGAGGAATACCGGTGGCGAAGGCGGCCCCCTG
GACAAAGACTGACGCTCAGGTGCGAAAGCGTGGGGAGCAAACAGGATTAGATACCC
TGGTAGTCCACGCCGTAAACGATGTCGATTTGGAGGTTGTGCCCTTGAGGCGTGGCT
TCCGGAGCTAACGCGTTAAATCGACCGCCTGGGGAGTACGGCCGCAAGGTTAAAAC
TCAAATGAATTGACGGGGGCCCGCACAAGCGGTGGAGCATGTGGTTTAATTCGATG
CAACGCGAAAACCACCTGGTCTTGACATCCACAGAACTTTCCAGAGATGGATTGGTG
CCTTCGGGAACTGTGAGACAGGTGCTCATGGCTGTCGTCAGCTCGTGTTGTGAAATG
TTGGGTTAAGTCCCGCAACGAGCGCAACCCTTATCCTTTGTTGCCAGCGGTTCGGCC
GGGAACTCAAAGGAGACTGCCAGTGATAAACTGGAGGAAGGTGGGGATGACGTCA
AGTCATCATGGCCCTTACGACCAGGGCTACACACGTGCTACAATGGCATATACAAA
GAGAAGCGACCTCGCGAGAGCAAGCGGACCTCATAAAGTATGTCGTAGTCCGGATT
GGAGTCTGCAACTCGACTCCATAACGGAATCGCTAGTAATCGTAGATCAGAATGCTA

CGGTGAATACGTTCCCGGGCCTTGTACACACCGCCCGTCACACCATGGGAGTGGGTT
GCAAAAGAAGTAGGTAGCTTAACCTTCGGGAGGGCGCTTACCACTTTGTGATTCATG
ACTGGGGTGAAGTCGTAACAAGGTAACCGTAGGGGAACCTGCGGTTGG

Table C.8: MT920655 *Klebsiella pneumoniae* strain TR-4

CTACCGCAGGTTCCCCTACGGTTACCTTGTTACGACTTCACCCCAGTCATGAATCAC
AAAGTGGTAAGCGCCCTCCCGAAGGTTAAGCTACCTACTTCTTTTGCAACCCACTCC
CATGGTGTGACGGGCGGTGTGTACAAGGCCCGGGAACGTATTCACCGTAGCATTCTG
ATCTACGATTACTAGCGATTCCGACTTCATGGAGTCGAGTTGCAGACTCCAATCCGG
ACTACGACATACTTTATGAGGTCCGCTTGCTCTCGCGAGTCGCTTCTCTTTGTATATG
CCATTGTAGCACGTGTGTAGCCCTGGTCGTAAGGGCCATGATGACTTGACGTCATCC
CCACCTTCCTCCAGTTTATCACTGGCAGTCTCCTTTGAGTTCCCGGCCGAACCGCTGG
CAACAAAGGATAAGGGTTGCGCTCGTTGCGGGACTTAACCCAACATTTCACAACAC
GAGCTGACGACAGCCATGCAGCACCTGTCTCACAGTTCCCGAAGGCACCAATCCAT
CTCTGGAAAGTTCTGTGGATGTCAAGACCAGGTAAGGTTCTTCGCGTTGCATCGAAT
TAAACCACATGCTCCACCGCTTGTGCGGGCCCCCGTCAATTCATTTGAGTTTTAACCT
TGCGGCCGTACTCCCCAGGCGGTCGATTTAACGCGTTAGCTCCGGAAGCCACGCCTC
AGGGCACAACCTCCAAATCGACATCGTTTACGGCGTGGACTACCAGGGTATCTAATC
CTGTTTGCTCCCCACGCTTTCGCACCTGAGCGTCAGTCTTTGTCCAGGGGGCCGCCTT
CGCCACCGGTATTCCTCCAGATCTCTACGCATTTCACCGCTACACCTGGAATTCTACC
CCCCTCTACAAGACTCTAGCCTGCCAGTTTCGAATGCAGTTCCCAGGTTGAGCCCGG
GGATTTCACATCCGACTTGACAGACCGCCTGCGTGCGCTTTACGCCCAGTAATTCCG
ATTAACGCTTGCACCCTCCGTATTACCGCGGCTGCTGGCACGGAGTTAGCCGGTGCT
TCTTCTGCGGGTAACGTCAATCGATGAGGTTATTAACCTCACCGCCTTCCTCCCCGCT
GAAAGTGCTTTACAACCCGCAAGGCCTTCTTCACACACGCGGCATGGCTGCATCAGG
CTTGCGCCCATTGTGCAATATTCCCCACTGCTGCCTCCCGTAGGAGTCTGGACCGTG
TCTCAGTTCCAGTGTGGCTGGTCATCCTCTCAGACCAGCTAGGGATCGTCGCCTAGG
TGAGCCGTTACCCCACCTACTAGCTAATCCCATCTGGGCACATCTGATGGCATGAGG
CCCGAAGGTCCCCCACTTTGGTCTTGCGACGTTATGCGGTATTAGCTACCGTTTCCAG
TAGTTATCCCCCTCCATCAGGCAGTTTCCCAGACATTACTCACCCGTCCGCCGCTCGT
CACCCGAGAGCAAGCTCTCTGTGCTACCGCTCGACTTGCATGTGTTAGGCCTGCCGC
CAGCGTACCTA

Table C.9: MT920656 *Klebsiella pneumoniae* strain TR-10

CGGCTCAGATTGAACGCTGGCGGCAGGCCTAACACATGCAAGTCGAGCGGTAGCAC
AGAGAGCTTGCTCTCGGGTGACGAGCGGCGGACGGGTGAGTAATGTCTGGGAAACT
GCCTGATGGAGGGGGATAACTACTGGAAACGGTAGCTAATACCGCATAACGTCGCA
AGACCAAAGTGGGGGACCTTCGGGCCTCATGCCATCAGATGTGCCCAGATGGGATT
AGCTAGTAGGTGGGGTAACGGCTCACCTAGGCGACGATCCCTAGCTGGTCTGAGAG
GATGACCAGCCACACTGGAACTGAGACACGGTCCAGACTCCTACGGGAGGCAGCAG
GGGGAATATTGCACAATGGGCGCAAGCCTGATGCAGCCATGCCGCGTGTGTGAAGA

AGGCCTTCGGGTTGTAAAGCACTTTCAGCGGGGAGGAAGGCGATGAGGTTAATAAC
CTCATCGATTGACGTTACCCTGCAGAAGAGCACCGGCTAACTCCGTGCCAGCAGCCG
CGGTAATACGGAGGGTGCAAGCGTTAATCGGAATTACTGGGCGTAAAGCGCACGCA
GGCGGTCTGTCAAGTCGGATGTGAAATCCCCGGGCTCAACCTGGGAACTGCATTCGA
AACTGGCAGGCTAGAGTCTTGTAGAGGGGGGTAGAATTCCAGGTGTAGCGGTGAAA
TGCGTAGAGATTGGAGGAATACCGGTGGCGAAGGCGGCCCCCTGGACAAAGACTGA
CGCTCAGGTGCGAAAGCGTGGGGAGCCAAACAGGATTAGATACCCTGGTAGTCCAC
GCCGTAAACGATGTCGATTTGGAGGTTGTGCCCTTGAGGCGTGGCTTCCGGAGCTAA
CGCGTTAAATCGACCGCCTGGGGAGTACGGCCGCAAGGTTAAAACTCAAATGATTG
ACGGGGGCCCGCACAAGCGGTGGAGCATGTGGTTTAATTCGATGCAACGCGAAGAA
CCTTACCTGGTCTTGACATCCACAGAACTTTCCAGAGATGGATTGGTGCCTTCGGGA
ACTGTGAGACAGGTGCTGCATGGCTGTCGTCAGCTCGTGTTGTGAAATGTTGGGTTA
AGTCCCGCAACGAGCGCAACCCTTATCCTTTGTTGCCAGCGGTTAGGCCGGGAACTC
AAAGGAGACTGCCAGTGATAAACTGGAGGAAGGTGGGGATGACGTCAAGTCATCAT
GGCCCTTACGACCAGGGCTACACACGTGCTACAATGGCATATACAAAGAGAAGCGA
CCTCGCGAGAGCAAGCGGACCTCATAAAGTATGTCGTAGTCCGGATTGGAGTCTGC
AACTCGACTCCATGAAGTCGGAATCGCTAGTAATCGTAGATCAGAATGCTACGGTG
AATACGTTCCCGGGCCTTGTACACACCGCCCGTCACACCATGGGAGTGGGTTGCAAA
AGAAGTAGGTAGCTTAACCTTCGGGAGGGCGCTTACCACTTTGTGATT

Table C.10: MT920657 *Proteus vulgaris* strain TR-7

CACCTGGCTCAGATTGAACGCTGGCGGCAGGCCTAACACATGCAAGTCGAGCGGTA
ACAGGAGGAAGCTTGCTTTCTTGCTGACGAGCGGCGGACGGGTGAGTAATGTATGG
GGATCTGCCCGATAGAGGGGGATAACTACTGGAAACGGTGGCTAATACCGCATGAC
GTCTACGGACCAAAGCAGGGGCTCTTCGGACCTTGCGCTATCGGATGAACCCATATG
GGATTAGCTAGTAGGTGAGGTAAAGGCTCACCTAGGCGACGATCTCTAGCTGGTCTG
AGAGGATGATCAGCCACACTGGGACTGAGACACGGCCCAGACTCCTACGGGAGGCA
GCAGTGGGGAATATTGCACAATGGGCGCAAGCCTGATGCAGCCATGCCGCGTGTAT
GAAGAAGGCCTTAGGGTTGTAAAGTACTTTCAGCGGGGAGGAAGGTGATAAAGTTA
ATACCTTTATCAATTGACGTTACCCGCAGAAGAAGCACCGGCTAACTCCGTGCCAGC
AGCCGCGGTAATACGGAGGGTGCAAGCGTTAATCGGAATTACTGGGCGTAAAGCGC
ACGCAGGCGGTCAATTAAGTCAGATGTGAAAGCCCCGAGCTTAACTTGGGAATTGC
ATCTGAAACTGGTTGGCTAGAGTCTTGTAGAGGGGGGTAGAATTCCACGTGTAGCG
GTGAAATGCGTAGAGATGTGGAGGAATACCGGTGGCGAAGGCGGCCCCCTGGACAA
GGACTGACGCTCAGGTGCGAAAGCGTGGGGAGCAAACAGGATTAGATACCCTGGTA
GTCCACGCTGTAAACGATGTCGATTTAGAGGTTGTGGTCTTGAACCGTGGCTTCTGG
AGCTAACGCGTTAAATCGACCGCCTGGGGAGTACGGCCGCAAGGTTAAAACTCAAA
TGAATTGACGGGGGCCCGCACAAGCGGTGGAGCATGTGGTTTAATTCGATGCAACG
CGAAGAACCTTACCTACTCTTGACATCCAGCGAATCCTTTAGAGATAGAGGAGTGCC
TTCGGGAACGCTGAGACAGGTGCTGCATGGCTGTCGTCAGCTCGTGTTGTGAAATGT
TGGGTTAAGTCCCGCAACGAGCGCAACCCTTATCCTTTGTTGTCAGCGCGTGATGGC

GGGAACCCAAAGGAGACTGCCGGTGATAAACCGGAGGAAGGTGGGGATGACGTCA
AGTCATCATGGCCCTTACGAGTAGGGCTACACACGTGCTACAATGGCAGATACAAA
GAGAAGCGACCTCGGGAGAGCAAGCGGAACTCATAAAGTCTGTCGTAGTCCGGATT
GGAGTCTGCAACTCGACTCCATGAAGTCAGGAATCGCTAGTAATCGTAGATCAGAA
TGCTACGGTGAATACGTTCCCGGGCCTTGTACACACCGCCCGCACACCATGGGAGTG
GGTTGCAAAAGAAGTAGGTAGCTTAACCTTCGGGAGGGCGCTTACCACTTTGTGA

Table C.11: MT920658 *Proteus vulgaris* strain TR-8

CCCTGGCGGCAGGCCTAACACATGCAAGTCGAGCGGTAACAGGAGAAAGCTTGCTT
TCTTGCTGACGAGCGGCGGACGGGTGAGTAATGTATGGGGATCTGCCCGATAGAGG
GGGATAACTACCTGGAAACGGTGGCTAATACCGCATGACGTCTACGGACCAAAGCA
GGGGCTCTTCGGACCTTGCGCTATGGATGAACCCATATGGGATTAGCTAGTAGGTGA
GGTAATGGCTCACCTAGGCAACGATCTCTAGCTGGTCCTGAGAGGATGATCAGCCAC
ACTGGGACTGAGACACGGCCCAGACTCTACGGGAGGCAGCAGTGGGGAATATTGCA
CAATGGGCGCAAGCCTGATGCAGCCATGCCGCGTGTATGAAGAAGGCCTTAGGGTT
GTAAAGTACTTTCAGCGGGGAGGAAGGTGATAAAGTTAATACCTTTATCAATTGACG
TTACCCGCAGAAGAAGCACCGGTAACTCCGTGCCAGCAGCCGCGGTAATACGGAGG
GTGCAAGCGTTAATCGGAATTACTGGGCGTAAAGCGCACGCAGGCGGTCAATTAAG
TCAGATGTGAAAGCCCCGAGCTTAACTTGGGAATTGCATCTGAAACTGGTTGGCTAG
AGTCTTGTAGAGGGGGGTAGAATTCCACGTGTAGCGGTGAAATGCGTAGAGATGTG
GAGGAAATCCGGTGGCGAAGGCGGCCCCCTGGACAAAGACTGACGCTCAGGTGCGA
AAGCGTGGGGAGCAAACAGGATTAGATACCCTGGTAGTCCACGCTGTAAACGATGT
CGATTTAGAGGTTGTGGTCTTGAACTGTGGCTTCTGGAGCTAACGCGTTAAATCGAC
CGCCTGGGGAGTACGGCCGCAAGGTTAAAACTCAAATGAATTGACGGGGGCCCGCA
CAAGCGGTGGAGCATGTGGTTTAATTCGATGCAACGCGAAGAACCTTACCTACTCTT
GACATCCAGCGAATCCTTTAGAGATAGAGGAGTGCCTTCGGGAACGCTGAGACAGG
TGCTGCATGGCTGTCGTCAGCTCGTGTTGTGAAATGTTGGGTTAAGTCCCGCAACGA
GCGCAACCCTTATCCTTTGTTGCCAGCGCGAATGGCGGGAACTCAAAGGAGACTGC
CGGTGATAAACCGGAGGAAGGTGGGGATGACGTCAAGTCATCATGGCCCTTACGAG
TAGGGCTACACACGTGCTACAATGGCAGATACAAAGAGAAGCGACCTCGCGAGAGC
AAGCGGAACTCATAAAGTCTGTCGTAGTCCGGATTGGAGTCTGCAACTCGACTCCAT
GAAGTCGGAATCGCTAGTAATCGTAGATCAGAATGCTACGGTGAATACGTTCCCGG
GCCTTGTACACACCGCCCGTCACACCATGGGAGTGGGTTGCAAAAGTAGTAGGTAG
CTTAACCTTCGGGCAGG

Table C.12: MT920659 *Proteus vulgaris* strain TR-12

TGGTCATGGCTCAGATTGAACGCTGGCGGCAGGCCTAACACATGCAAGTCGAGCGG
TAACAGAAGAAAGCTTGCTTTCTTGCTGACGAGCGGCGGACGGGTGAGTAATGTAT
GGGGATCTGCCCGATAGAGGGGGATAACTACTGGAAACGGTGGCTAATACCGCATG
ACGTCTACGGACCAAAGCAGGGGCTCTTGGACCTTGCGCTATCGGATGAACCCATAT
GGGATTAGCTAGTAGGTGAGGTAATGGCTCACCTAGGCAACGATCTCTAGCTGGTCT

GAGAGGATGATCAGCCACACTGGGACTGAGACACGGCCCAGACTCCTACGGGAGGC
AGCAGTGGGGAATATTGCACAATGGGCGCAAGCCTGATGCAGCCATGCCGCGTGTA
TGAAGAAGGCCTTAGGGTTGTAAAGTACTTTCAGCGGGGAGGAAGGTGATAAAGTT
AATACCTTTATCAATTGACGTTACCCGCAGAAGAAGCACCGGCTAACTCCGTGCCAG
CAGCCGCGGTAATACGGAGGGTGCAAGCGTTAACGGAATTACTGGGCGTAAAGCGC
ACGCAGGCGGTCAATTAAGTCAGATGTGAAAGCCCCGAGCTTAACTTGGGAATTGC
ATCTGAAACTGGTTGGCTAGAGTCTTGTAGAGGGGGGTAGAATTCCACGTGTAGCG
GTGAAATGCGTAGAGATGTGGAGGAATACCGGTGGCGAAGGCGGCCCCCTGGACAA
AGACTGACGCTCAGGTGCGAAAGGTGGGGAGCAAACAGGATTAGATACCCTGGTAG
TCCACGCTGTAAACGATGTCGATTTAGAGGTTGTGGTCTTGAACCGTGGCTTCTGGA
GCTAACGCGTTAAATCGACCGCCTGGGGAGTACGGCCGCAAGGTTAAAACTCAAAT
GAATTGACGGGGGCCCGCACAAGCGGTGGAGCATGTGGTTTAATTCGATGCAACGC
GAAGAACCTTACCTACTCTTGACATCCAGCGAATCCTTTAGAGATAGAGGAGTGCCT
TCGGGAACGCTGAGACAGGTGCTGCATGGCTGTCGTCAGCTCGTGTTGTGAAATGTT
GGGTTAAGTCGCAACGAGCGCAACCCTTATCCTTTGTTGCCAGCGCGTAATGGCGGG
AACTCAAAGGAGACTGCCGGTGATAAACCGGAGGAAGGTGGGGATGACGTCAAGTC
ATCATGGCCCTTACGAGTAGGGCTACACACGTGCTACAATGGCAGATACAAAGAGA
AGCGACCTCGCGAGAGCAAGCGGAACTCATAAAGTCTGTCGTAGTCCGGATTGGAG
TCTGCAACTCGACTCCATGAAGTCGGAATCGCTAGTAATCGTAGATCAGAATGCTAC
GGTGAATACGTTCCCGGGCCTTGTACACACCGCCCGTCACACCATGGGAGTGGGTTG
CAAAAGAAGTAGGTAGCTTAACCTTCGGGAGGGCGCTTACCACTTTGTGATTCATGA
CTGGGGTGAAGTCGTAACAAGGTAACCGTAGGGGAACCTGCGGTTGGATCACCTCC
TTA

APPENDIX D

Frequency of occurrence of multidrug resistance in the 9 General hospitals

Number of antibiotics	Number of resistance occurrence	Percentage (%)
3	23	10.1
4	32	14.0
5	29	12.7
6	22	10.0
7	32	14.0
8	31	13.6
9	24	10.5
10	35	15.4
Total	228	100

APPENDIX E

Screening of Isolates for ESBL and Carbapenemase Production

Table E.1: Screening of Isolates from Suleja for ESBL and Carbapenemase Production

Isolates	Amoxicillin-Clavulanic Acid	Ceftriaxone	Ceftoxime	Caftazidime	ESBL	Imipenem	C-Producer
H-1SU	24	9	15	17	+	16	+
H-2SU	14	8	9	8	+	14	+
H-3SU	18	12	14	10	+	16	+
H-4SU	0	0	0	0	-	19	+
H-8SU	26	16	18	19	+	29	-
H-9SU	27	11	17	18	+	15	+
H-11SU	19	12	19	15	+	27	-
H-12SU	23	11	17	19	+	16	+
U-1SU	24	16	16	16	+	13	+
U-2SU	2	3	4	3	-	13	+
U-3SU	29	15	20	22	+	28	-
U-4SU	24	17	18	19	+	14	+
U-5SU	24	15	17	16	+	13	+
U-6SU	24	10	16	19	+	24	-
U-7SU	34	27	11	14	+	16	+
U-9SU	28	13	11	14	+	14	+
U-10SU	34	16	14	16	+	17	+
U-11SU	33	16	16	15	+	24	-
U-12SU	49	28	33	21	+	19	+

(≥20)= Non- Carbapenemase producer; < 20= Carbapenemase producer

Table E.2: Screening of Isolates from Minna for ESBL and Carbapenemase Production

Isolates	Amoxicillin-Clavulanic Acid	Ceftriaxone	Cefotaxime	Ceftazidime	ESBL	Imipenem	C-Producer
H-2M	20	23	22	21	+	27	-
H-9M	26	24	26	22	+	19	+
H-14M	4	4	3	4	-	13	+
H-15M	24	18	21	20	+	24	-
H-16M	22	20	18	22	+	13	+
H-17M	23	24	26	20	+	11	+
U-1M	22	20	21	19	+	12	+
U-2M	26	24	22	20	+	16	+
U-3M	3	4	2	3	-	0	+
U-5M	2	3	3	2	-	16	+
U-8M	26	24	23	22	+	0	+
U-9M	22	23	22	24	+	19	+
U-12M	25	23	21	22	+	24	-
U-16M	26	21	19	20	+	0	+

Table E.3: Screening of Isolates from Kuta for ESBL and Carbapenemase Production

Isolates	Amoxicillin-Clavulanic Acid	Ceftriaxone	Cefotaxime	Ceftazidime	ESBL	Imipenem	C-Producer
H-2KU	17	14	17	19	+	14	+
H-3KU	27	15	19	17	+	25	-
H-4KU	3	4	4	2	-	19	+
H-5KU	24	17	16	17	+	24	-
H-6KU	29	15	20	16	+	13	+
H-7KU	22	17	14	15	+	12	+
H-8KU	29	24	20	11	+	23	-
H-10KU	28	22	15	15	+	12	+
H-11KU	4	3	3	2	-	15	+
H-12KU	26	14	10	15	+	10	+
U-1KU	26	19	20	21	+	19	+
U-2KU	15	12	14	13	+	12	+
U-4KU	17	16	19	18	+	12	+
U-5KU	2	2	4	3	-	14	+
U-6KU	24	19	21	18	+	23	-
U-7KU	4	4	2	1	-	17	+
U-8KU	22	24	20	23	+	10	+
U-10KU	21	22	21	20	+	12	+
U-11KU	2	3	3	2	-	17	+
U-12KU	23	17	18	19	+	14	+

Table E.4: Screening of Isolates from Bida for ESBL Production and Carbapenemase Production

Isolates	Amoxicillin-Clavulanic Acid	Ceftriaxone	Cefotaxime	Ceftazidime	ESBL	Imipenem	C-Producer
H-1B	24	22	19	20	+	15	+
H-2B	22	21	22	23	+	15	+
H-3B	23	23	24	22	+	25	-
H-4B	25	20	23	21	+	12	+
H-5B	3	2	3	3	-	15	+
H-10B	24	21	20	18	+	15	+
H-11B	22	18	20	21	+	0	+
H-12B	20	19	17	17	+	15	+
H-13B	2	4	2	3	-	17	+
H-16B	23	22	20	21	+	26	-
H-18B	19	20	20	19	+	14	+
H-19B	19	19	18	17	+	26	-
H-20B	24	21	22	20	+	25	-
U-1B	29	24	24	24	+	0	+
U-2B	14	17	15	15	+	0	+
U-3B	3	4	2	3	-	18	+
U-4B	16	18	19	17	+	0	+
U-5B	19	18	18	19	+	0	+
U-6B	15	11	16	15	+	0	+
U-7B	16	11	15	11	+	0	+
U-8B	31	29	30	28	+	0	+
U-9B	22	19	18	17	+	25	-
U-11B	16	11	15	14	+	14	+
U-13B	27	24	21	22	+	22	-

U-14B	22	19	16	14	+	24	-
U-15B	13	11	14	11	+	13	+
U-17B	11	11	10	11	+	24	-
U-18B	22	16	18	19	+	18	+
U-19B	23	20	19	21	+	24	-
U-20B	26	22	21	20	+	0	+
U-21B	22	20	22	21	+	13	+
U-22B	23	21	21	20	+	26	-
U-23B	23	20	20	22	+	13	+

Table E.5: Screening of Isolates from Agaie for ESBL Production and Carbapenemase Production

Isolates	Amoxicillin-Clavulanic Acid	Ceftriaxone	Cefotaxime	Ceftazidime	ESBL	Imipenem	C-Producer
H-1AG	24	23	11	13	+	17	+
H-3AG	36	20	12	19	+	19	+
H-4AG	28	23	15	17	+	19	+
H-5AG	20	18	17	19	+	18	+
H-7AG	24	20	15	19	+	17	+
H-10AG	24	16	10	15	+	21	-
H-14AG	28	14	13	10	+	20	-
H-19AG	18	16	10	18	+	18	+
H-20AG	24	22	11	14	+	21	-
U-1AG	32	21	19	20	+	14	+
U-2AG	21	14	15	13	+	0	+
U-3AG	27	16	14	12	+	14	+
U-4AG	19	16	18	12	+	17	+
U-5AG	29	22	17	10	+	10	+
U-6AG	28	23	21	20	+	24	-
U-8AG	30	25	21	20	+	9	+
U-9AG	24	23	21	20	+	0	+
U-10AG	23	21	15	16	+	14	+
U-11AG	52	27	32	29	+	13	+
U-12AG	26	18	17	15	+	14	+
U-14AG	25	21	19	20	+	19	+
U-15AG	30	18	13	13	+	22	-
U-16AG	3	3	4	2	-	15	+
U-17AG	26	14	15	18	+	19	+

U-18AG	25	20	19	22	+	10	+
U-19AG	24	16	20	19	+	0	+
U-20AG	27	17	20	21	+	19	+
U-21AG	17	13	12	10	+	24	-
U-22AG	27	21	20	22	+	26	-

Table E.6: Screening of Isolates from Lapai for ESBL Production and Carbapenemase Production

Isolates	Amoxicillin-Clavulanic Acid	Ceftriaxone	Cefotaxime	Ceftazidime	ESBL	Imipenem	C-Producer
H-1L	26	18	19	17	+	22	-
H-2L	20	14	15	12	+	19	+
H-3L	17	11	14	16	+	19	+
H-4L	10	12	11	10	+	21	-
H-5L	24	19	20	18	+	18	+
H-6L	4	3	3	2	-	18	+
H-7L	22	14	10	15	+	15	+
H-8L	4	2	1	2	-	17	+
H-9L	4	4	2	4	-	19	+
H-10L	26	14	11	19	+	22	-
H-11L	20	11	10	19	+	16	+
H-12L	20	11	11	17	+	20	-
H-13L	15	10	12	13	+	17	+
H-14L	19	12	11	17	+	21	-
H-15L	3	2	4	2	-	24	-
H-16L	16	15	18	19	+	14	+
H-17L	24	10	10	12	+	17	+
H-18L	30	18	12	16	+	21	-
U-3L	29	21	20	23	+	16	+
U-6L	49	33	29	28	+	24	-
U-7L	19	11	13	15	+	19	+
U-8L	34	14	14	16	+	21	-
U-10L	19	20	18	19	+	0	+
U-14L	23	17	17	16	+	17	+

U-15L	34	15	14	15	+	13	+
U-17L	29	16	15	15	+	15	+
U-24L	39	14	14	14	+	14	+
U-28L	29	14	14	14	+	13	+
U-29L	15	14	15	13	+	22	-
U-32L	29	16	15	15	+	14	+
U-34L	44	34	33	29	+	12	+
U-36L	4	4	3	2	-	15	+
U-37L	19	15	16	16	+	24	-
U-38L	20	15	17	19	+	25	-
U-39L	27	19	25	24	+	14	+
U-40L	29	39	23	21	+	26	-

Table E.7: Screening of Isolates from Wushishi for ESBL Production and Carbapenemase Production

Isolates	Amoxicillin-Clavulanic Acid	Ceftriaxone	Cefotaxime	Ceftazidime	ESBL	Imipenem	C-Producer
H-2W	19	18	19	17	+	18	+
H-6W	17	15	15	16	+	23	-
H-10W	31	20	22	26	+	17	+
H-12W	22	21	18	18	+	16	+
H-15W	16	14	17	15	+	21	-
H-18W	0	0	0	0	-	18	+
H-22W	24	20	17	19	+	14	+
H-24W	0	0	0	0	-	15	+
H-26W	19	20	21	18	+	0	+
H-28W	27	18	15	19	+	18	+
U-1W	29	20	18	19	+	21	-
U-7W	4	4	3	2	-	19	+
U-8W	25	19	20	21	+	24	-
U-11W	29	20	15	20	+	16	+
U-13W	27	22	20	21	+	16	+
U-15W	22	23	21	20	+	15	+
U-16W	24	19	16	19	+	18	+
U-21W	23	22	21	21	+	15	+
U-22W	27	21	23	22	+	17	+
U-28W	26	24	25	21	+	0	+

Table E.8: Screening of Isolates from Kontagora for ESBL Production and Carbapenemase Production

Isolates	Amoxicillin-Clavulanic Acid	Ceftriaxone	Cefotaxime	Ceftazidime	ESBL	Imipenem	C-Producer
H-1KN	0	0	0	0	-	19	+
H-2KN	23	23	22	20	+	18	+
H-13KN	0	0	0	0	-	17	+
H-15KN	0	0	0	0	-	19	+
H-27KN	20	15	15	15	+	20	-
H-31KN	22	16	15	16	+	21	-
H-39KN	23	18	18	17	+	19	+
H-42KN	19	14	14	13	+	22	-
U-27KN	23	22	21	20	+	19	+
U-28KN	0	0	0	0	-	19	+
U-29KN	16	15	16	17	+	20	-
U-34KN	19	14	14	13	+	16	+
U-36KN	20	15	15	10	+	22	-
U-38KN	12	11	13	14	+	18	+
U-39KN	20	15	14	15	+	0	+
U-40KN	15	10	10	10	+	19	+
U-42KN	12	14	13	11	+	0	+
U-43KN	15	10	10	10	+	0	+
U-46KN	0	0	0	0	-	18	+
U-47KN	22	21	24	23	+	17	+
U-52KN	12	11	11	10	+	15	+

U-56KN	18	20	19	17	+	16	+
U-57KN	0	0	0	0	-	14	+
U-59KN	24	24	22	21	+	18	+
U-63KN	20	17	19	15	+	23	-
U-64KN	25	26	19	20	+	15	+
U-65KN	24	19	19	19	+	14	+
U-66KN	0	0	0	0	-	18	+
U-67KN	21	23	20	22	+	19	+
U-68KN	0	0	0	0	-	16	+
U-71KN	19	22	23	20	+	15	+
U-73KN	20	15	15	15	+	18	+
U-75KN	22	19	15	20	+	18	+

Table E.9: Screening of Isolates from Nasko for ESBL Production and Carbapenemase Production

Isolates	Amoxicillin-Clavulanic Acid	Ceftriaxone	Cefotaxime	Ceftazidime	ESBL	Imipenem	C-Producer
H-2N	22	17	21	19	+	17	+
H-8N	24	19	20	18	+	17	+
H-12N	20	22	19	20	+	18	+
H-17N	24	21	18	16	+	20	-
H-18N	3	2	3	2	-	18	+
H-19N	19	15	17	15	+	21	-
H-20N	4	4	3	2	-	16	+
H-22N	25	15	17	16	+	18	+
U-1N	22	24	23	21	+	21	-
U-3N	17	17	20	19	+	20	-
U-4N	0	0	0	0	-	17	+
U-6N	0	0	0	0	-	17	+
U-7N	24	25	22	20	+	21	-
U-8N	18	19	17	18	+	0	+
U-9N	0	0	0	0	-	17	+
U-10N	23	25	22	19	+	18	+
U-12N	0	0	0	0	-	19	+
U-14N	21	20	19	18	+	0	+
U-15N	4	2	3	3	-	15	+
U-16N	22	21	20	21	+	16	+
U-18N	14	15	17	15	+	17	+
U-20N	2	4	4	2	-	19	+

U-21N	0	0	0	0	-	18	+
U-22N	14	17	16	15	+	21	-