LOUISIANA DEPARTMENT OF HEALTH
Public Health

The LOUISIANA ANTIBIOGRAM Louisiana Antibiotic Resistance 2016

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This report examines bacteria capable of causing severe human infections and the antibiotics used to treat those infections. Resistance to other antimicrobials (antivirals, antifungals and anti-parasitic drugs) are not included due to a lack of systematic reporting resulting in limited data.

Contents

1-Introduction

1.1-Bacterial resistance to antibiotics is a major threat to human health

Bacterial resistance to antibiotics is becoming a major threat to human health. Bacteria become resistant to antibiotics through mutation or acquisition of genes from other bacteria. Antibiotics work by affecting the cell wall, distorting the cell surface, inhibiting bacterial protein synthesis, or preventing DNA formation. Some bacteria have been able to adopt ways to become resistant to the actions of antibiotics; some have become resistant to several classes of antibiotics. Resistance often emerges first in hospitals because of selective pressure.

As antibiotic resistance was developing, medical science made advances in treating illnesses that were fatal in older times. Therefore, there are now an increasing number of vulnerable patients with limited ability to fight infections (e.g. patients undergoing chemotherapy for cancer, dialysis for renal failure, and surgery, especially organ transplantation).

1.2-Tracking resistance patterns is a major action in the fight against antibiotic resistance

Most of the data published in the scientific literature on bacterial resistance is heavily influenced by limited surveys, case series and individual case reports. The data presented often comes from research institutions, tertiary care hospitals and other sources that are not representative of the "bacterial universe." These sources are biased toward reporting the unusual and more severe patterns. A report using population-based data sets provides a more representative picture of drug resistance patterns.

The Louisiana Antibiotic Resistance Surveillance System was started in 1998 to track the emergence of antibiotic resistant organisms. The goal of the program is to estimate the proportion of selected bacteria in the state that are resistant to antibiotics.

2-Methods

2.1-Active surveillance

In the early period of resistance monitoring, an active surveillance system was implemented. A select group of hospitals were called each month to provide information on a brief reporting form. The reports included (1) the number isolates from selected species from their lab for each month, (2) the number of drug-resistant or drug-intermediate isolates for each one of those microorganisms. Duplicates were not to be counted. Each report was entered into a Microsoft® Access database and from this annual summary, reports were generated for the participating hospitals. This type of surveillance was cumbersome, therefore limited to a few microorganisms. It was abandoned for the antibiogram collection approach.

2.2-Antibiogram collection

In 2001, a NCCLS (National Committee for Clinical Laboratory Standards, which in 2005 became the Clinical and Laboratory Standard Institute CLSI) subcommittee issued guidelines to use in analyzing and presenting cumulative antimicrobial susceptibility test data. They established standardized means of data extraction for all drugs tested and outlined how the data should be presented:

- Percent susceptibility for the first isolate from a patient within an analysis period (generally one year)

- Population tested (e.g., inpatient, ICU, nursing home)
- Specimen source (e.g., blood, sputum, urine)
- Number of isolates tested (minimum 10 for each organism)
- Separate data for Gram-negative, Gram-positive, aerobic and anaerobic organisms
- List drugs alphabetically or by class
- Avoid selective reporting (cascading): secondary agents reported only if isolate is resistant to the primary drug class.

Once a year, most hospitals issue an "antibiogram," which is a summary of the most important antibiotic resistance patterns for their hospital for the year. The antibiogram is a table listing the microorganisms in the left-most column and antibiotics in the remaining columns. The percent of organisms found to be susceptible to each antibiotic is recorded in the table's cells. Some hospitals generate reports every three, six or 12 months. These frequent reports result in small numbers of isolates, and sometimes result in large variations in percentage from one quarter to the next. These variations are usually not sustained and are not significant.

Example of an Antibiogram:

Organism	Total Isolates	PIP	CZOL	CTRX	CTAZ	CFPM	GEN	TOB	T/S	CIP	P/T	IMI
Acinetobacter baumannii	51	10	N/A	4	8	16	26	88	28	12	14	98
Citrobacter freundii	40	39	N/A	65	60	100	95	95	78	98	68	100
Enterobacter aerogenes	28	68	N/A	68	61	100	100	100	100	93	68	100
Enterobacter cloacae	98	42	N/A	43	45	89	84	84	69	85	60	100
Escherichia coli*	418	36	81	95	96	97	87	88	66	76	92	100
Klebsiella oxytoca	33	79	58	97	97	97	91	97	70	97	91	100
Klebsiella pneumoniae	146	76	90	94	93	95	93	93	85	88	92	98
Proteus mirabilis	40	91	85	100	98	100	83	88	73	75	100	100
Pseudomonas aeruginosa**	185	77	N/A	N/A	72	75	87	90	N/A	56	79	76
Serratia marcescens	26	100	N/A	96	96	100	100	96	96	92	100	100

The antibiogram shows the spectrum of sensitivity/resistance among the most common microorganisms detected by the microbiology laboratory. It provides useful information for the selection of an empiric antibiotic treatment when a presumptive diagnosis of infection with a specific bacteria is made. The antibiogram is no longer useful once the specific bacteria causing an infection have been identified and the antibiotic resistance patterns are known.

There are some limitations when using a hospital antibiogram:

1-Most hospital laboratories do not sort out community-acquired infections from hospital-acquired. The antibiotic resistance patterns for both groups may be substantially different. Gram-negative rods tend to be more prevalent in hospital infections and more resistant if they originate from a hospital source.

2-Some laboratories do not thoroughly eliminate duplicate cultures from the same patients. This can lead to an artificial inflation of the proportion of resistance, as resistant strains tend to be cultured more often.

3- Antibiograms only report the percent of isolates susceptible to an antibiotic. For the purposes of this report, it is assumed that isolates not susceptible to an antibiotic are resistant to it, though they may actually be intermediately susceptible. Therefore, the "percent resistance" presented in

the following tables and figures should be assumed to include a combination of intermediate and resistant isolates.

If constructed carefully and interpreted with caution, a hospital antibiogram is a useful tool.

The Statewide Louisiana Antibiogram

The Louisiana Antibiogram is not as useful as the individual hospital antibiogram for making empiric treatment decisions. However, it is useful to compare one individual hospital antibiogram to the rest of the state using the Louisiana Antibiogram. If any large differences in antibiotic susceptibilities are detected between a hospital and the Louisiana Antibiogram, hospitals should investigate the reason for the discrepancy.

2.3-Analysis

The purpose of this analysis is to examine trends in antibiotic resistance rates for the most medically important bacteria from 2000 to 2016 and to present the resistance data for the most recent period, 2016.

2.3.1-Trend tables:

For microorganisms of interest, a trend table is presented. The first column contains the number of resistant isolates, the second column contains the number of isolates tested during the year, and the third column lists the percentage of resistant strains.

2.3.2-Recent data on resistance:

Recent 2016 data on resistance are displayed in tables throughout the report. Data include the total number of isolates tested, the average resistance in percentage and the range of resistance percentages observed (lowest and highest resistance observed in any hospital antibiogram). Graphs are also used for some bacteria to visualize resistance trends over time.

3-Trends and recent situation

3.1-Methicillin-Susceptible *Staphylococcus aureus* (MSSA)

Staphylococcus aureus (SA), is a Gram-positive catalase-positive cocci typically seen in clusters on Gram stain. *Staphylococcus aureus* is the most important human pathogen of the Staphylococcal group. Its golden yellow pigment gives the species its name, though some isolates are non-pigmented. *S. aureus* is widespread in the population; about 30% are carriers, particularly in the nasal cavity, but also in the perineum, anal area and finger tips, among other areas. The most common infections include carbuncles, furuncles, cellulitis and wound infections. Food poisoning, toxic shock syndrome, acute endocarditis, septic arthritis, meningitis, osteomyelitis, pneumonia and septicemia are also seen. It is often isolated from nosocomial infections (10% to 20% of nosocomial infections), especially bacteremias, skin infections and surgical site infections.

Resistance due to penicillinase (an enzyme of the β-lactamase group) produced by *S. aureus*, developed as soon as penicillin was introduced for clinical use. This enzyme allows staphylococci to cleave the β-lactam ring of penicillin and neutralize its effectiveness. Nowadays, most *S. aureus* isolates are resistant to penicillin. The aminopenicillins (ampicillin, amoxicillin), carboxypenicillins (carbenicillin, ticarcillin), and ureidopenicillins (mezlocillin, piperacillin) are susceptible to neutralization

by penicillinase-producing *S. aureus*. The preferred antibiotics for the treatment of MSSA are penicillinase-resistant penicillins. These antibiotics include nafcillin, oxacillin, methicillin, cloxacillin, and dicloxacillin.

Alternative drugs used in the treatment of methicillin-sensitive *S. aureus* include:

- Amoxicillin-clavulanate
- Clindamycin if D test negative
- Doxycycline or minocycline plus Rifampin
- Moxifloxacin
- Trimethoprim-sulfamethoxazole (TMP-SMX) plus rifampin
- Vancomycin, linezolid or daptomycin

Practically any infection caused by *Staphylococcus aureus* is presumed to be resistant to methicillin unless laboratory testing proves methicillin sensitivity.

Table 1. 2016 Methicillin-sensitive *Staphylococcus aureus* resistance to specific antibiotics. For each antibiotic, data include the total number of isolates, the average observed resistance, and the lowest and highest resistance values reported.

2016 Methicillin-Sensitive *Staphylococcus aureus* (MSSA)					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Ampicillin	Penicillin Amino	307	100%	100%	100%
Cefazolin	Cephalosporin 1	731	25%	0%	100%
Cefepime	Cephalosporin 4	102	100%	100%	100%
Cefoxitin	Cephalosporin 2	102	100%	100%	100%
Ceftazidime	Cephalosporin 3	102	100%	100%	100%
Ceftriaxone	Cephalosporin 3	442	33%	0%	100%
Ciprofloxacin	Quinolone	998	16%	5%	26%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	531	0%	0%	0%
Clindamycin	Lincosamides	4422	21%	0%	37%
Clindamycin-Inducible Resistance		459	72%	62%	83%
Daptomycin	Lipopeptide	675	0%	0%	0%
Dicloxacillin	PenicillinRb-lactamase	86	0%	0%	0%
Doxycycline	Cyclines	188	6%	1%	12%
Erythromycin	Macrolides	4105	45%	0%	70%
Gentamicin	Aminoglycosides	1490	1%	0%	3%
Levofloxacin	Quinolone	1436	13%	5%	24%
Lincomycin	Lincosamides	27	0%	0%	0%
Linezolid	Oxazolidinone	1057	0%	0%	0%
Meropenem	Carbapenem	102	100%	100%	100%
Moxifloxacin	Quinolone	1722	5%	0%	17%
Nitrofurantoin	Quinolone	1252	1%	0%	4%
Oxacillin	PenicillinRb-lactamase	4348	1%	0%	7%
Penicillin G	Penicillin	2521	81%	64%	100%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	102	100%	100%	100%
Quinu/Dalfopristin	Streptogramin	145	0%	0%	0%
Rifampin	Rifamycin	1432	0%	0%	2%

Sulbactam-Ampicillin	Penicillin&b-lactamInhib	442	34%	0%	100%
Tetracycline	Cyclines	3829	8%	0%	16%
Tobramycin	Aminoglycosides	102	100%	100%	100%
Trimethoprim-sulfa	Sulfonamide	4467	1%	0%	5%
Vancomycin	Glycopolypeptide	4413	0%	0%	0%

3.2-Methicillin (oxacillin)-Resistant *Staphylococcus aureus* (MRSA)

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a growing problem both in the hospital and in the community. Resistance to methicillin is due to altered penicillin binding proteins.

S. aureus methicillin resistance resulted from a different mechanism. To overcome simple penicillin resistance, *S. aureus* was able to modify the site to which methicillin attaches (Penicillin Binding Protein), and thus became resistant to methicillin.

Methicillin results from the addition of large radicals (chemical chains) around the penicillin ring to provide protection against penicillinase. Methicillin is effective on *S. aureus* resistant to penicillin.

Acquisition of MRSA infections was a common concern among both patients and staff in acute and long-term care facilities, and now has become a concern for the general population.

As seen in Figure 1 below, the rates of methicillin-resistant *S. aureus* increased from 2000 to 2005 from 38% to over 68%, and now seem to be stabilizing just below 60%. Data indicate that methicillin resistance rates began a slight decline from 60% in 2012, which has continued through 2016.

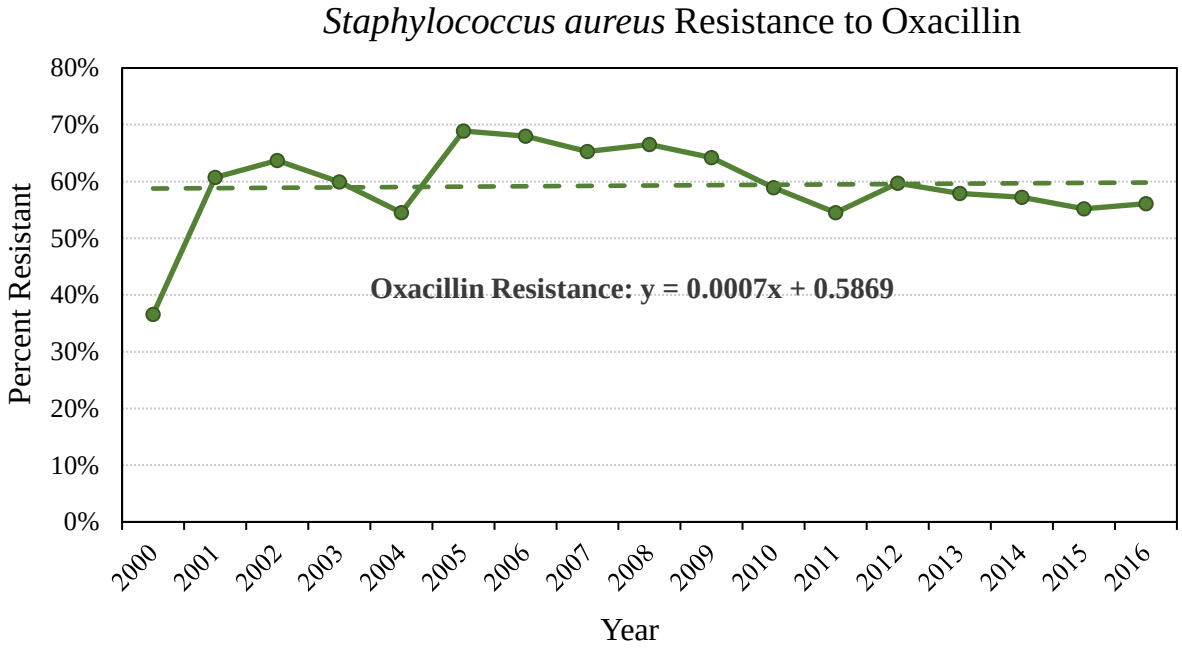

Figure 1. Rates of oxacillin resistance among *S. aureus* isolates in Louisiana from 2000 – 2016.

3.3-History of MRSA: Healthcare-associated (HA-MRSA) and Community-associated MRSA (CA-MRSA).

MRSA infections that are described in this report have not been differentiated into community-associated (CA) MRSA (or SCCmec Type IV or V PVL positive), and hospital-associated (HA) MRSA (or SCCmec Type II/III). Most Type IV MRSA isolates remains sensitive to TMP-SMX, clindamycin and fluoroquinolones, though some of these antibiotics may not be effective in vivo. Type II/III organisms tend to be sensitive only to vancomycin and newer agents like linezolid.

MRSA was first recognized in 1961, one year after introduction of methicillin. These resistant strains first appeared in hospitals, mostly as nosocomial infections. The first documented MRSA outbreak in the U.S. occurred in a Boston hospital in 1968. From the 1970s through the 1990s, most MRSA infections occurred in persons who had contact with hospitals or other healthcare facilities (HCF), hence the term healthcare-associated MRSA. In the 1990s and 2000s, MRSA infections became more frequent among previously healthy individuals with no association with HCFs. This suggested that infections were originating within the community, hence the term community-acquired MRSA or CA-MRSA.

HA-MRSA causes mostly sporadic cases with the exception of a few strains causing epidemics in hospitals. Most MRSA are simple colonizers. HA-MRSA are not more virulent than other *S. aureus* strains. There is no difference in animal lethality, production of enzymes or production of toxins associated with invasiveness. However this strain is resistant to most antibiotics other than vancomycin and a few newer antibiotics.

CA-MRSA started to spread in the late 1990s and 2000s and soon after became the dominant MRSA clone in the U.S. CA-MRSA is known to be more virulent than HA-MRSA, causing frequent skin and soft tissue infections as well as invasive infections (septicemia and pneumonias). Experiments have shown that CA-MRSA produces toxins more frequently than HA-MRSA.

MRSA resistance results from four mec genes (named I to IV), consisting in chromosomal elements of 30 to 50-kilobase coding penicillin-binding proteins. The mecA gene encodes a PBP with low affinity for β-lactam antibiotics. The mecA gene complex is carried on specific integrative genetic element (staphylococcal cassette chromosome - SCC). This cassette includes mec complex + cassette recombinase, which integrate and excise SCCmec element on staphylococcal chromosome. Molecular strain typing is done by Pulse Field Gel Electrophoresis (PFGE), arbitrarily primed PCR, randomly amplified polymorphic DNA, plasmid fingerprinting and multilocus sequence typing (MLST).

The difference between CA-MRSA isolates and HA-MRSA isolates is the type of SCCmec. The SCCmec is a cluster of chromosomes in which the mecA gene is carried. Typical CA-MRSA has SCCmec type IV while typical HA-MRSA carries SCCmec types I and II. I and II are larger genes, which may carry resistance for trimethoprim-sulfa, clindamycin, and some other antibiotics.

The PFGE classification is widely used. It includes USA 100 and 200 (old CA-MRSA), and strains 300 to 1100. The USA 300 strain has spread into healthcare settings to become the dominant strain. In 2005, 22% community-associated MRSA diagnosed in HCF and 16% hospital-onset invasive MRSA were caused by USA 300 (Klevens RM, Morrison MA, Nadle J, et al. Invasive methicillin-resistant. *Staphylococcus aureus* infections in the United States. JAMA 2007; 298: 1763-1771).

The distinction between these two types of MRSA is becoming increasingly blurry. CA-MRSA, particularly USA 300, is emerging as the dominant MRSA strain in the community and in health care settings, underscoring the importance of monitoring changing sensitivities.

3.4-Other Antibiotics to which MRSA is Resistant

Many cutaneous abscesses respond to drainage alone, and most of the remaining Type IV MRSA infections can be treated with trimethoprim–sulfamethoxazole or a tetracycline, such as doxycycline or minocycline. For serious infections, other antibiotics may be required for treatment. Options include vancomycin, fluoroquinolones, daptomycin, quinupristin–dalfopristin, newer-generation carbapenems, and linezolid.

Quinolones, such as levofloxacin or moxifloxacin, are effective orally and generally provide adequate coverage for CA-MRSA. Unfortunately, resistance is emerging among both MSSA and MRSA isolates. Data suggest that overuse of quinolones promotes emergence of MRSA strains in the community.

Linezolid, an oxazolidinone, is useful for severe refractory MRSA infections and can also be administered orally. In some severely ill patients, linezolid therapy has proved to be more effective than vancomycin, but resistance is emerging and the drug should be reserved for serious infections.

The possibility of inducible clindamycin resistance has discouraged some physicians from prescribing clindamycin. The inducible macrolide-lincosamide-streptogramin B phenotype is related to the erm gene. Strains with inducible resistance will test clindamycin-susceptible in vitro, but are erythromycin-resistant. If inducible resistance is present, there is a potential for treatment failure with clindamycin, despite the culture and sensitivity report indicating susceptibility. Some laboratories issue a report stating that macrolide resistance may be a marker for inducible lincosamide resistance. If the clinician is considering clindamycin, an erythromycin-clindamycin "D-zone" test is prudent. To perform a D-test, clindamycin and erythromycin disks are placed close together on a culture plate. If inducible lincosamide resistance is present, the zone of inhibition around the clindamycin disk is flattened on the side toward the erythromycin disk. This results in a zone of inhibition resembling a capital letter D instead of an O.

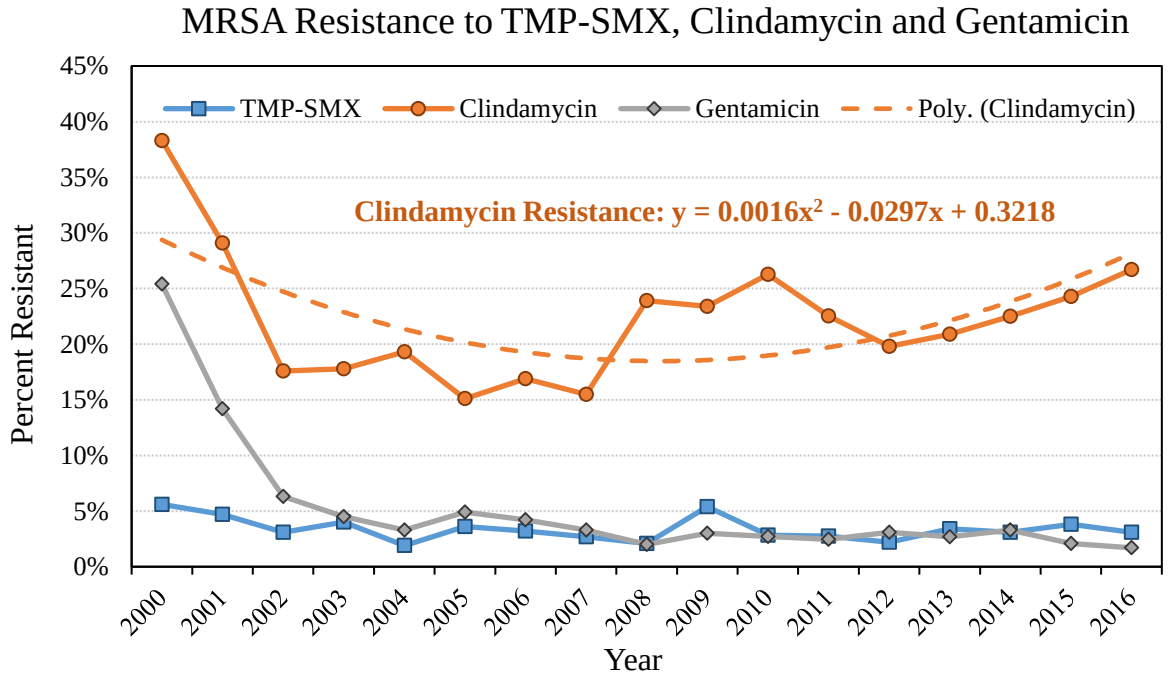

Figure 2. MRSA resistance rates to trimethoprim-sulfamethoxazole (TMP-SMX), clindamycin, and gentamicin in Louisiana from 2000-2016.

As seen in Figure 2 above, in Louisiana, TMP-SMX retains a relatively high sensitivity for some MRSA, illustrating the pattern seen in community-acquired organisms. Vancomycin remains effective and is still the first-line drug in the treatment of life-threatening infections caused by MRSA or *S. aureus* of unknown sensitivity.

MRSA strains are consistently sensitive to vancomycin, linezolid and daptomycin. They are resistant to macrolides (75% to 100%), fluoroquinolones (49% to 100%), and clindamycin (3% to 43%). They are less resistant to aminoglycosides (1% to 6% in recent years) and trimethoprim-sulfamethoxazole (0% to 13%).

Table 2. 2016 Methicillin-resistant *Staphylococcus aureus* resistance to antibiotics in 2016.

2016 Methicillin-Resistant *S. aureus* (MRSA)					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Ampicillin	Penicillin Amino	509	100%	100%	100%
Azithromycin	Macrolides	118	88%	88%	88%
Cefazolin	Cephalosporin 1	521	100%	100%	100%
Cefepime	Cephalosporin 4	124	100%	100%	100%
Cefoxitin	Cephalosporin 2	124	100%	100%	100%
Ceftazidime	Cephalosporin 3	124	100%	100%	100%
Ceftriaxone	Cephalosporin 3	509	100%	100%	100%
Chloramphenicol	Chloramphenicol	118	2%	2%	2%
Ciprofloxacin	Quinolone	2058	63%	43%	76%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	782	100%	100%	100%
Clindamycin	Lincosamides	6161	25%	3%	43%
Clindamycin-Inducible Resistance		442	80%	70%	90%
Daptomycin	Lipopeptide	1892	0%	0%	2%
Doxycycline	Cyclines	271	5%	1%	10%
Erythromycin	Macrolides	5189	84%	43%	91%
Gentamicin	Aminoglycosides	2691	1%	0%	6%
Levofloxacin	Quinolone	2564	64%	49%	100%
Linezolid	Oxazolidinone	2841	1%	0%	11%
Meropenem	Carbapenem	124	100%	100%	100%
Moxifloxacin	Quinolone	2405	26%	0%	54%
Nitrofurantoin	Quinolone	2117	0%	0%	3%
Norfloxacin	Quinolone	118	53%	53%	53%
Oxacillin	PenicillinRb-lactamase	2007	100%	100%	100%
Penicillin G	Penicillin	1503	100%	100%	100%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	124	100%	100%	100%
Quinu/Dalfopristin	Streptogramin	198	0%	0%	0%
Rifampin	Rifamycin	4056	2%	0%	11%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	509	100%	100%	100%
Tetracycline	Cyclines	6104	6%	0%	12%
Tigecycline	Glycylcycline	210	0%	0%	0%
Tobramycin	Aminoglycosides	124	100%	100%	100%
Trimethoprim-sulfa	Sulfonamide	6714	3%	0%	13%

Vancomycin	Glycopolypeptide	6570	0%	0%	1%

3.5-Coagulase-negative *Staphylococcus* (CONS)

CONS are habitual inhabitants of the skin with very low pathogenic potential. The group includes *S. epidermidis* and *S. saprophyticus*. They are commonly isolated as contaminants, especially in blood cultures, hence the requirement for two blood cultures to define a coagulase-negative staphylococcal bloodstream infection. They may cause nosocomial infections in patients with severe underlying medical problems or indwelling prosthetic devices (due to its polysaccharide capsule causing adherence to devices). The great majority of coagulase-negative Staphylococcal nosocomial infections are septicemias in immunocompromised neonates (*S. epidermidis*), followed by conjunctivitis, urinary tract (*S. saprophyticus*), and skin infections. The treatment of coagulase-negative staphylococci depends on the organism and the type of infection. Treatment must ultimately be decided based on susceptibility testing of the isolate.

Coagulase-negative staphylococci that cause nosocomial infections, particularly *S. epidermidis* and *S. hemolyticus*, are usually resistant to multiple antibiotics, with more than 80% resistant to methicillin. The methicillin-resistance gene (mecA) is identical in *S. aureus* and *S. epidermidis*. Antibiotics to which most coagulase-negative staphylococci are susceptible to in vitro include vancomycin, minocycline, linezolid, the combination streptogramin, quinupristin/dalfopristin, and daptomycin.

Table 3. 2016 Coagulase-negative *Staphylococcus* resistance to antibiotics in 2016.

2016 Coagulase-negative *Staphylococcus*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Ampicillin	Penicillin Amino	133	86%	86%	86%
Azithromycin	Macrolides	133	75%	75%	75%
Cefazolin	Cephalosporin 1	530	61%	56%	65%
Cefepime	Cephalosporin 4	133	56%	56%	56%
Cefotaxime	Cephalosporin 3	133	56%	56%	56%
Ceftriaxone	Cephalosporin 3	133	57%	57%	57%
Cephalothin	Cephalosporin 1	133	56%	56%	56%
Chloramphenicol	Chloramphenicol	133	3%	3%	3%
Ciprofloxacin	Quinolone	1867	44%	29%	54%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	385	62%	56%	68%
Clindamycin	Lincosamides	4164	45%	26%	62%
Daptomycin	Lipopeptide	1774	2%	0%	9%
Erythromycin	Macrolides	4428	71%	59%	79%
Gentamicin	Aminoglycosides	3703	15%	2%	33%
Imipenem	Carbapenem	133	57%	57%	57%
Levofloxacin	Quinolone	3872	45%	9%	67%
Linezolid	Oxazolidinone	2179	1%	0%	7%
Moxifloxacin	Quinolone	1176	25%	0%	42%
Nitrofurantoin	Quinolone	1404	1%	0%	8%
Norfloxacin	Quinolone	133	53%	53%	53%
Oxacillin	PenicillinRb-lactamase	4640	56%	38%	77%
Penicillin G	Penicillin	1213	94%	88%	100%

Piperacillin/Tazobactam	Penicillin&b-lactamInhib	323	4%	3%	4%
Quinu/Dalfopristin	Streptogramin	405	1%	0%	2%
Rifampin	Rifamycin	3278	3%	0%	6%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	133	56%	56%	56%
Tetracycline	Cyclines	4712	17%	7%	24%
Tigecycline	Glycylcycline	272	0%	0%	0%
Tobramycin	Aminoglycosides	190	0%	0%	0%
Trimethoprim-sulfa	Sulfonamide	3326	38%	9%	54%
Vancomycin	Glycopolypeptide	4496	0%	0%	3%

Table 4. 2016 Coagulase-negative methicillin-resistant *Staphylococcus epidermidis* resistance to antibiotics in 2016.

2016 Methicillin-resistant *S. epidermidis* Coagulase-negative (MRSE)					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Ciprofloxacin	Quinolone	24	75%	75%	75%
Clindamycin	Lincosamides	24	63%	63%	63%
Daptomycin	Lipopeptide	24	0%	0%	0%
Doxycycline	Cyclines	24	25%	25%	25%
Erythromycin	Macrolides	80	86%	84%	88%
Gentamicin	Aminoglycosides	80	26%	21%	30%
Levofloxacin	Quinolone	80	73%	71%	75%
Linezolid	Oxazolidinone	24	0%	0%	0%
Moxifloxacin	Quinolone	24	46%	46%	46%
Nitrofurantoin	Quinolone	80	0%	0%	0%
Rifampin	Rifamycin	80	5%	2%	8%
Tetracycline	Cyclines	80	18%	11%	25%
Tigecycline	Glycylcycline	24	0%	0%	0%
Trimethoprim-sulfa	Sulfonamide	80	62%	57%	67%
Vancomycin	Glycopolypeptide	80	0%	0%	0%

3.6-*Streptococcus pneumoniae*

Streptococcus pneumoniae (Pneumococcus) is the most common cause of community-acquired pneumonia both in children and adults. It causes about half of all otitis media cases and it is a frequent cause of meningitis and sepsis. Mortality resulting from pneumococcal infections is high. Pneumococcal pneumonia ranks among the top ten leading causes of death in many countries, with a case fatality rate of 5% for pneumonia, 20% for bacteremia and 30% for meningitis.

Because sensitive and rapid diagnostic tests are not available, most pneumococcal infections are treated empirically at first. Until the 1970s, all pneumococcal isolates were sensitive to easily-achievable levels of most commonly-used antibiotics, including penicillins, macrolides, clindamycin, cephalosporins, rifampin, vancomycin, and trimethoprim-sulfamethoxazole. Beginning in the 1990s, many pneumococcal isolates in the US showed decreased susceptibility to penicillin and other commonly-used antibiotics. In 2010, only 10.6% of all isolates obtained showed intermediate or resistant susceptibility patterns to penicillin (down from 24.8% in 2008; 25.6% in 2007). The prevalence of resistance varies greatly within countries, states, counties, and even cities, and may be as high as 30%-40% in some locations. In

Louisiana, rates of resistance have been consistently high. Resistance to penicillin is associated with a decreased affinity of the antibiotic to penicillin-binding proteins present in the bacterial cell wall. Penicillin resistance is thought to occur through horizontal transfer of genes associated with altered penicillin-binding proteins and lowered affinity to penicillin and other β-lactams. Pneumococci have become resistant by acquiring genetic material from other bacteria with which they coexist in close proximity - presumably viridans streptococci in the nasopharynx. At least 30% of the pneumococcal strains in the U.S. show intermediate resistance to penicillin (MIC 0.1–2.0µg/ml). This type of resistance can be overcome if the antibiotic concentration at the site of infection exceeds the MIC of the organism for 40%-50% of the dosing interval. Except for meningitis patients, these types of infections are readily treatable with increased doses of penicillin.

Of more concern is the appearance of pneumococcal isolates that are regarded as highly resistant to penicillin (MIC ≥2.0µg/ml) (Fig. 3). It is suggested that the extended consumption of oral cephalosporins contributes to pneumococcal resistance to penicillin. If these strains are circulating, it might be more reliable to treat severe pneumococcal infections with vancomycin. However, the rate of resistance to other commonly-used antibiotics such as erythromycin, tetracycline and trimethoprim-sulfamethoxazole is much greater in penicillin-resistant strains than in penicillin-sensitive strains.

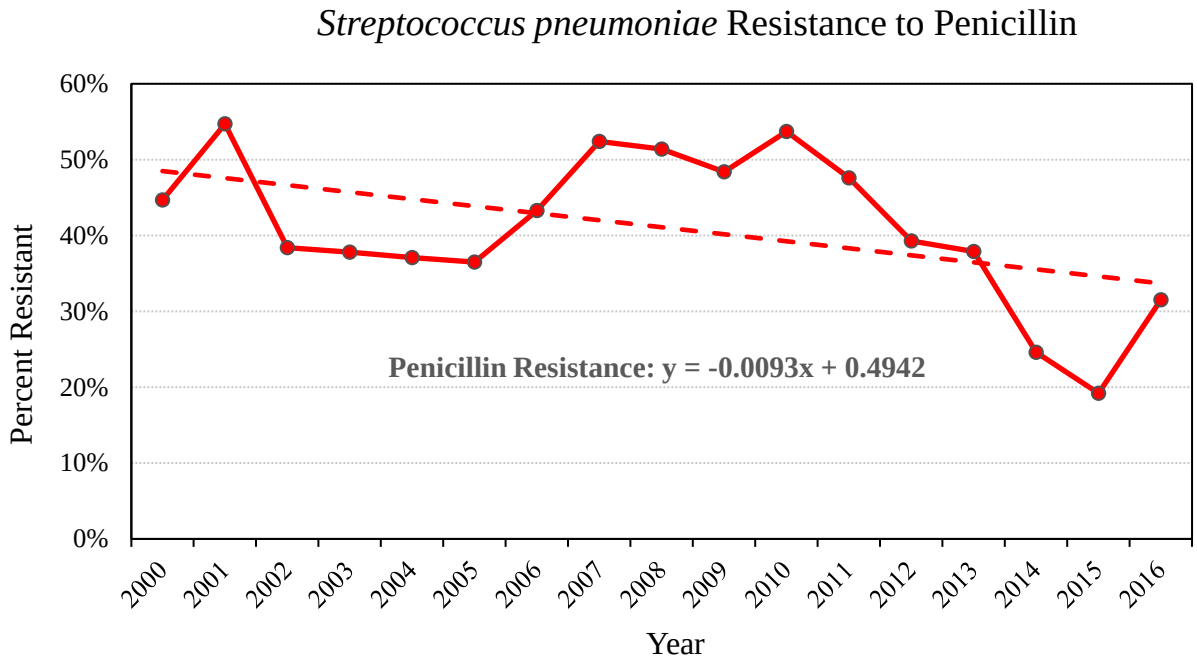

Figure 3. *S. pneumoniae* resistance rates to penicillin in Louisiana from 2000-2016.

The susceptibility of *S. pneumoniae* to penicillin is currently defined by the NCCLS as follows: susceptible isolates are inhibited by 0.06 µg/mL (i.e., minimal inhibitory concentration [MIC] ≤0.06 µg/mL); isolates with reduced susceptibility (also known as intermediate resistance) are inhibited by 0.1 to 1.0 µg/mL, and resistant isolates are inhibited by 2.0 µg/mL or more. This definition was derived based on achievable concentrations of penicillin in CSF during treatment of children for meningitis. From a clinical point of view, the meaning of the MIC depends on the infection being treated. A strain with reduced susceptibility (e.g., MIC of 1.0 µg/mL) may behave as a susceptible organism when it causes pneumonia, but not when it causes otitis or meningitis. The recently revised definition of amoxicillin resistance (susceptible, MIC µg/mL; intermediately resistant, MIC 4 g/mL, resistant, MIC >8 g/mL) is

based on serum levels, assuming that no physician would knowingly treat meningitis with this oral medication.

3.7-*Streptococcus* group A

Streptococcus pyogenes, the group A Strep, are β-hemolytic and are found in the nasopharynx of healthy carriers. They may cause pharyngitis, which is the most common clinical expression. The drug of choice in the treatment of streptococcal infections is penicillin, because of its efficacy in the prevention of rheumatic fever, safety, narrow spectrum, and low cost. Oral cephalosporins are also highly effective in the treatment of streptococcal pharyngitis. First-generation oral cephalosporins are acceptable alternatives in the penicillin-allergic patient whose allergy is not of the immediate type.

In penicillin-allergic patients, erythromycin is the therapy of choice. The newer macrolides (azithromycin, clarithromycin) appear to be effective. There have been reports of resistance to macrolides and azalide antibiotics from several countries.

There has also been considerable recent interest in abbreviated courses of antimicrobial therapy. It has been reported that clarithromycin, cefuroxime, cefixime, ceftibuten, cefdinir, cefpodoxime and azithromycin are effective in eradication of group A streptococci from the pharynx when administered for five days or less.

3.8-*Streptococcus* group B

Streptococcus agalactiae, the Group B Strep, are partially β-hemolytic and can colonize the female genital tract which can lead to neonatal infections. It is a cause of urinary tract infections (UTI) and IV line infections, especially in diabetics and the elderly. It is also a rare cause of subacute bacterial endocarditis (SBE).

Group B streptococci remain uniformly susceptible to penicillins and cephalosporins in vitro, and penicillin G is the drug of choice once the diagnosis is established. They are also susceptible to ampicillin, vancomycin, and teicoplanin. Meropenem and imipenem also have good in vitro activity. Increasing resistance to erythromycin (62%) and clindamycin (50%) restrict their use as empiric treatment for invasive infection or for intrapartum prophylaxis. Tetracycline resistance has increased to nearly 83%.

Table 5. *Streptococcus agalactiae* (Group B Streptococcus) resistance to antibiotics in 2016.

2016 Streptococcus group B, agalactiae					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Ampicillin	Penicillin Amino	1458	0%	0%	4%
Azithromycin	Macrolides	139	87%	87%	87%
Cefepime	Cephalosporin 4	139	0%	0%	0%
Cefotaxime	Cephalosporin 3	363	0%	0%	1%
Ceftriaxone	Cephalosporin 3	942	1%	0%	2%
Ciprofloxacin	Quinolone	42	0%	0%	0%
Clindamycin	Lincosamides	1940	48%	20%	96%
Daptomycin	Lipopeptide	47	0%	0%	0%

Erythromycin	Macrolides	1404	62%	33%	80%
Levofloxacin	Quinolone	1632	2%	0%	7%
Linezolid	Oxazolidinone	670	0%	0%	0%
Moxifloxacin	Quinolone	249	2%	0%	6%
Penicillin G	Penicillin	1210	0%	0%	1%
Quinu/Dalfopristin	Streptogramin	279	0%	0%	1%
Tetracycline	Cyclines	678	82%	66%	88%
Tigecycline	Glycylcycline	304	0%	0%	0%
Vancomycin	Glycopolypeptide	1715	0%	0%	0%

3.9-Streptococcus Viridans Group

Streptococcus viridans is a group of streptococci which possess no Lancefield antigens. They are most abundant in the mouth. Among these streptococci is *S. mutans* - the etiologic agent of dental caries. Viridans streptococci may cause other mouth or gingival infections, and if they are introduced into the bloodstream may cause endocarditis. They are the most common causes of subacute bacterial endocarditis. For severe infections vancomycin and clindamycin remain the medication of choice.

3.10-Enterococci and Vancomycin-resistant Enterococci

Enterococci, formerly of the Streptococci are now part of the *Enterococcus* genus. These organisms grow under harsh conditions and are differentiated from the non-enterococcal group D streptococci in part by their ability to grow in 6.5% sodium chloride. Enterococci constitute a sizable portion of the normal flora of the gut. When there is disruption of mucosal or epithelial barriers, they can produce infection, including UTIs, endocarditis and intra-abdominal abscesses. *E. faecalis* is more common than *E. faecium* as a pathogen. Enterococci are difficult to treat because of extensive resistance to antibiotics used against Gram-positive cocci. They are intrinsically resistant to a large number of antibiotics, but can also easily acquire new mechanisms of resistance.

Enterococci are naturally fairly resistant to all β-lactam antibiotics because of the low affinity of their penicillin binding proteins. With the exception of cefoperazone, cephalosporins are not effective on them. They can also develop a more complete resistance to penicillin and ampicillin. Enterococci show a remarkable ability to acquire new mechanisms of resistance. As a result, susceptibility patterns vary considerably according to temporal and geographic variation. Aminoglycosides have difficulty penetrating through the outer envelope of the enterococci, but are used synergistically with penicillin or ampicillin in treatment. Enterococci have developed resistance to vancomycin (VRE) through a genetic mechanism which is also transferable within species, and possibly to other species.

Combinations of penicillin plus aminoglycosides produce bactericidal killing of enterococci. Unfortunately, enterococci can develop high-level resistance to streptomycin via chromosomal mutation. Strains of enterococci with high level resistance to streptomycin are not necessarily highly resistant to gentamicin and other aminoglycosides and, in recent years, penicillin (or ampicillin) plus gentamicin has become the standard of therapy for enterococcal endocarditis, meningitis, and other serious infections requiring bactericidal therapy. Unfortunately, the l980s and 1990s have seen a marked worldwide increase in strains of enterococci with genes that encode a bi-functional phosphor-transferase /acetyl-transferase enzyme that inactivates gentamicin and all other currently available aminoglycosides except

streptomycin. Such organisms are not killed synergistically by combinations of gentamicin plus cell-wall-active antibiotics.

Table 6. *Enterococcus faecalis* resistance to antibiotics in 2016.

2016 *Enterococcus faecalis*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Ampicillin	Penicillin Amino	9097	1%	0%	6%
Cefazolin	Cephalosporin 1	63	100%	100%	100%
Cefepime	Cephalosporin 4	326	69%	38%	100%
Cefoxitin	Cephalosporin 2	63	100%	100%	100%
Ceftazidime	Cephalosporin 3	63	100%	100%	100%
Ceftriaxone	Cephalosporin 3	63	100%	100%	100%
Chloramphenicol	Chloramphenicol	126	0%	0%	0%
Ciprofloxacin	Quinolone	5058	29%	10%	58%
Daptomycin	Lipopeptide	3445	9%	0%	97%
Doxycycline	Cyclines	759	74%	67%	80%
Erythromycin	Macrolides	1696	87%	57%	100%
Gentamicin	Aminoglycosides	1504	36%	15%	100%
Levofloxacin	Quinolone	5637	27%	0%	93%
Linezolid	Oxazolidinone	5003	1%	0%	8%
Meropenem	Carbapenem	63	100%	100%	100%
Moxifloxacin	Quinolone	95	1%	1%	1%
Nitrofurantoin	Quinolone	6181	1%	0%	7%
Norfloxacin	Quinolone	126	43%	43%	43%
Penicillin G	Penicillin	3384	2%	0%	13%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	63	100%	100%	100%
Quinu/Dalfopristin	Streptogramin	404	99%	99%	99%
Rifampin	Rifamycin	224	40%	37%	42%
Streptomycin	Aminoglycosides	564	15%	10%	26%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	63	100%	100%	100%
Tetracycline	Cyclines	6228	77%	62%	87%
Tigecycline	Glycylcycline	2719	1%	0%	6%
Tobramycin	Aminoglycosides	355	51%	2%	100%
Trimethoprim-sulfa	Sulfonamide	63	100%	100%	100%
Vancomycin	Glycopolypeptide	9320	3%	0%	20%

Table 7. *Enterococcus faecium* resistance to antibiotics in 2016.

2016 *Enterococcus faecium*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Ampicillin	Penicillin Amino	1236	72%	31%	100%
Ciprofloxacin	Quinolone	504	75%	0%	96%
Daptomycin	Lipopeptide	440	13%	3%	21%
Doxycycline	Cyclines	37	61%	33%	88%

Erythromycin	Macrolides	56	82%	67%	98%
Gentamicin	Aminoglycosides	288	10%	0%	21%
Levofloxacin	Quinolone	661	79%	33%	100%
Linezolid	Oxazolidinone	900	3%	0%	31%
Nitrofurantoin	Quinolone	675	76%	60%	88%
Penicillin G	Penicillin	326	69%	33%	90%
Quinu/Dalfopristin	Streptogramin	75	4%	0%	7%
Rifampin	Rifamycin	12	33%	33%	33%
Streptomycin	Aminoglycosides	68	55%	40%	70%
Tetracycline	Cyclines	944	79%	54%	92%
Tigecycline	Glycylcycline	455	0%	0%	1%
Tobramycin	Aminoglycosides	76	54%	54%	54%
Vancomycin	Glycopolypeptide	1243	55%	0%	83%

The emergence of vancomycin-resistant strains of enterococci (VRE) in the past 20 years has led to increased risks of invasive VRE infections with high lethality. Vancomycin resistant enterococcus is ubiquitous in the hospital environment, often found as a contaminant on medical equipment. Most patients are simply colonized and not infected (a ratio of 10:1). Persons at highest risk for VRE infections are those hospitalized with severe underlying or immunosuppressive conditions. These people may be affected by one of two mechanisms: drug resistance developed post-exposure to the antibiotic or via contact with the drug resistant pathogen (person-to-person or environmental transmission). The difference in the rate of resistance of *E. fecalis* and *E. faecium* to vancomycin is displayed in the graph below (Fig. 4). *E. fecalis* has a lower rate of resistance while *E. faecium* has a higher rate of resistance to vancomycin.

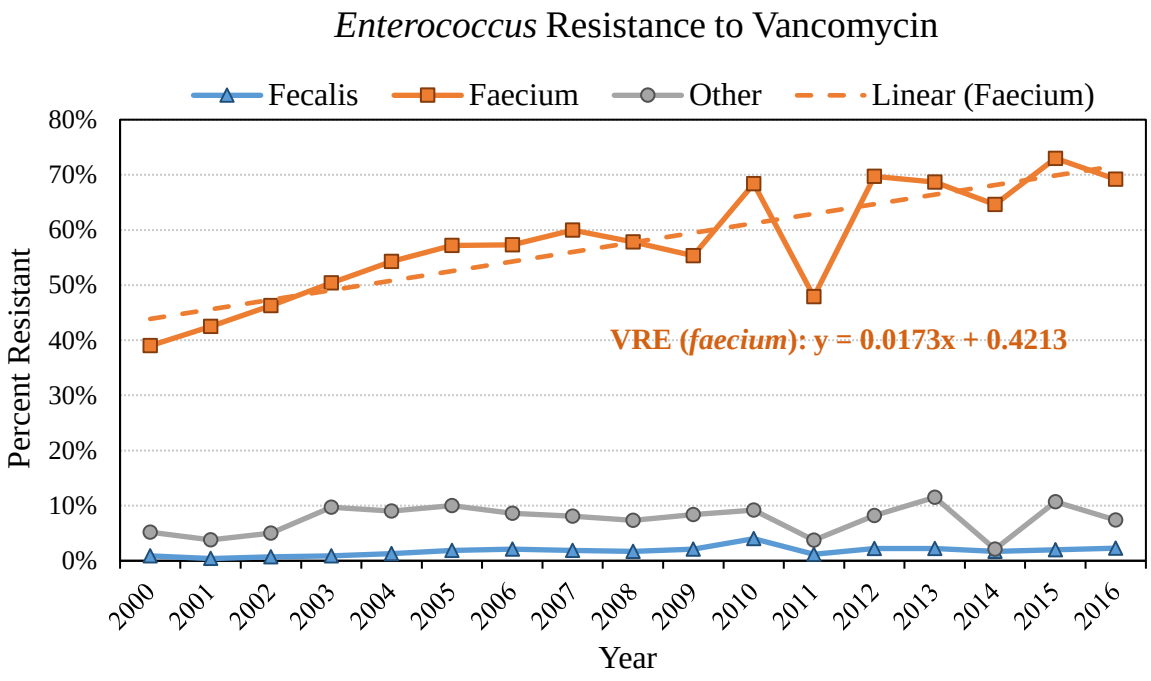

Figure 4. VRE resistance rates in Louisiana from 2000 – 2016.

3.11-*Neisseria meningitidis*

Neisseria meningitidis is a colonizer of a small percent of the population, and is also an important cause of septicemia and pyogenic meningitis. Reduced susceptibility to rifampin is of concern since this antibiotic is often used for prophylaxis of close contacts. The number of *Neisseria meningitidis* isolates tested for antibiotic sensitivity is very small (less than 20 per year). Sensitivity to cephalosporins and rifampin remain at 100%. Currently, a third-generation cephalosporin (ceftriaxone orcefotaxime) is the drug of choice for the treatment of meningococcal meningitis and septicemia. Penicillin G, ampicillin, chloramphenicol, fluoroquinolone, and aztreonam are alternative therapies (IDSA guidelines Jun 15, 2016).

3.12-*Haemophilus influenzae*

Haemophilus are Gram-negative bacilli specific to humans, normally colonizing the pharynx. They cause otitis media, sinusitis, conjunctivitis, bronchopneumonia, cellulitis, and invasive disease such as meningitis and septic arthritis. Some strains of *H. influenzae* possess a polysaccharide capsule, and these strains are serotyped into six different types (a-f) based on their biochemically-different capsules. The most virulent strain is *H. influenzae* type b (Hib). Some *H. influenzae* strains have no capsule and are termed nonencapsulated *H. influenzae* or nontypeable *H. influenzae* (NTHi) (Medscape website 2016).

Administer parenteral antibiotics (e.g., ceftriaxone, ceftazidime, cefotaxime, ampicillin-sulbactam, fluoroquinolones) to patients with uncomplicated meningitis for 7-14 days. Resistance to macrolides is high. *H. influenzae* has seen a slight increase in resistance to ceftriaxone and fluoroquinolones. Cefotaxime and ceftriaxone are the initial drugs of choice for suspected Hib meningitis.

Table 8. *Haemophilus influenzae* resistance to antibiotics in 2016.

2016 *Haemophilus influenzae*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Ampicillin	Penicillin Amino	118	40%	0%	80%
Azithromycin	Macrolides	72	4%	4%	4%
Cefixime	Cephalosporin 3	80	1%	0%	3%
Cefotaxime	Cephalosporin 3	109	1%	0%	1%
Ceftriaxone	Cephalosporin 3	49	8%	0%	33%
Cefuroxime	Cephalosporin 2	37	3%	3%	3%
Ciprofloxacin	Quinolone	12	0%	0%	0%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	76	9%	0%	18%
Levofloxacin	Quinolone	5	0%	0%	0%
Meropenem	Carbapenem	4	0%	0%	0%
Rifampin	Rifamycin	45	0%	0%	0%
Tetracycline	Cyclines	3	67%	67%	67%
Trimethoprim-sulfa	Sulfonamide	121	30%	0%	53%

3.13-*Acinetobacter*

Acinetobacter are small non-motile Gram-negative bacilli from the *Neisseriaceae* family. They have been designated *Mima*, *Herellea* and *Micrococcus* in the past. They are free-living organisms extremely common in food, water and on environmental surfaces. In humans, they are common in sputum, urine, feces and vaginal secretions. About 25% of adults are colonized. *Acinetobacter* are becoming a more common cause of nosocomial infections, usually causing ventilator-associated pneumonia, line sepsis or burn wound sepsis.

A. baumannii is intrinsically multidrug resistant. Relatively few antibiotics are active against this organism. There has been a large increase in resistance to imipenem, which went from 0% in 2001 to 67% in 2010. The increasing resistance of *A. baumannii* to antibiotics is shown in the graph below (Fig. 5).

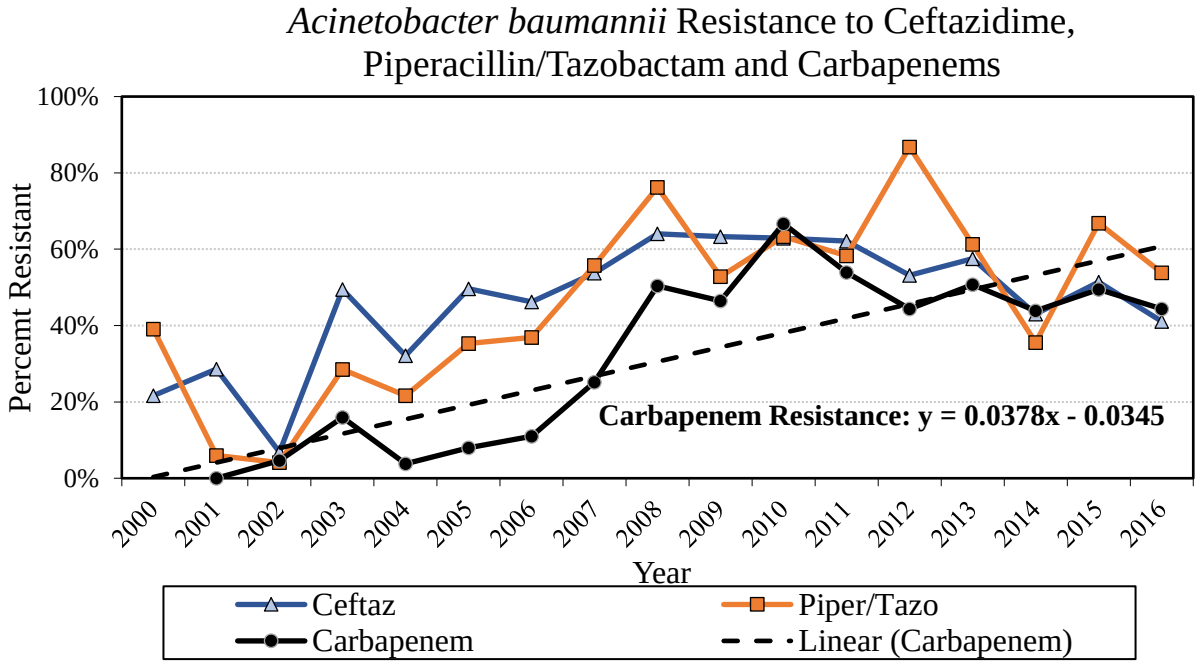

Figure 5. *Acinetobacter baumannii* resistance rates in Louisiana from 2000 – 2016.

Table 9. *Acinetobacter baumannii* resistance to antibiotics in 2016.

2016 *Acinetobacter baumannii*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Amikacin	Aminoglycosides	350	20%	0%	50%
Aztreonam	Monobactam	125	100%	100%	100%
Cefazolin	Cephalosporin 1	125	100%	100%	100%
Cefepime	Cephalosporin 4	424	44%	19%	71%
Cefotaxime	Cephalosporin 3	37	66%	45%	88%
Ceftazidime	Cephalosporin 3	531	37%	0%	72%
Ceftriaxone	Cephalosporin 3	326	73%	25%	100%
Ciprofloxacin	Quinolone	655	43%	13%	74%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	8	38%	38%	38%

Gentamicin	Aminoglycosides	603	22%	0%	45%
Imipenem	Carbapenem	197	43%	14%	67%
Imipenem/Cilastatin	Carbapenem	125	48%	48%	48%
Levofloxacin	Quinolone	394	45%	13%	71%
Meropenem	Carbapenem	304	36%	0%	68%
Minocycline	Cyclines	7	0%	0%	0%
Nitrofurantoin	Quinolone	125	100%	100%	100%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	359	54%	29%	100%
Polymyxin	Glycopolypeptide	7	0%	0%	0%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	522	34%	14%	58%
Tetracycline	Cyclines	103	35%	13%	44%
Tigecycline	Glycylcycline	7	0%	0%	0%
Tobramycin	Aminoglycosides	553	21%	0%	53%
Trimethoprim-sulfa	Sulfonamide	445	35%	14%	58%

3.14-*Enterobacteriaceae*

Enterobacteriaceae is a large group of Gram-negative organisms which are widely distributed in the soil and are normal colonizers of the intestinal tract of humans and animals. They are an important cause of infection when found outside the gastrointestinal tract. They account for 30% of all nosocomial infectious agents isolated (30% of septicemia isolates, 20% of surgical site infections, 55% of urinary tract isolates and 20% of pulmonary infections isolates). Among the *Enterobacteriaceae, Escherichia coli*, *Klebsiella, Proteus, Salmonella, Shigella* and *Enterobacter* are the most important pathogens.

3.14.1-*E. coli*

E. coli is a normal inhabitant of the human gastrointestinal tract. It produces disease when it is in other habitats such as the urinary tract, biliary tract, blood or meninges. However, a few types of *E. coli* are not part of the normal human flora. When introduced in humans, these strains cause gastroenteritis (entero-toxigenic, entero-invasive and entero-hemorrhagic *E. coli*). Antibiotic resistance rates are displayed in Figure 6 and Table 10 below.

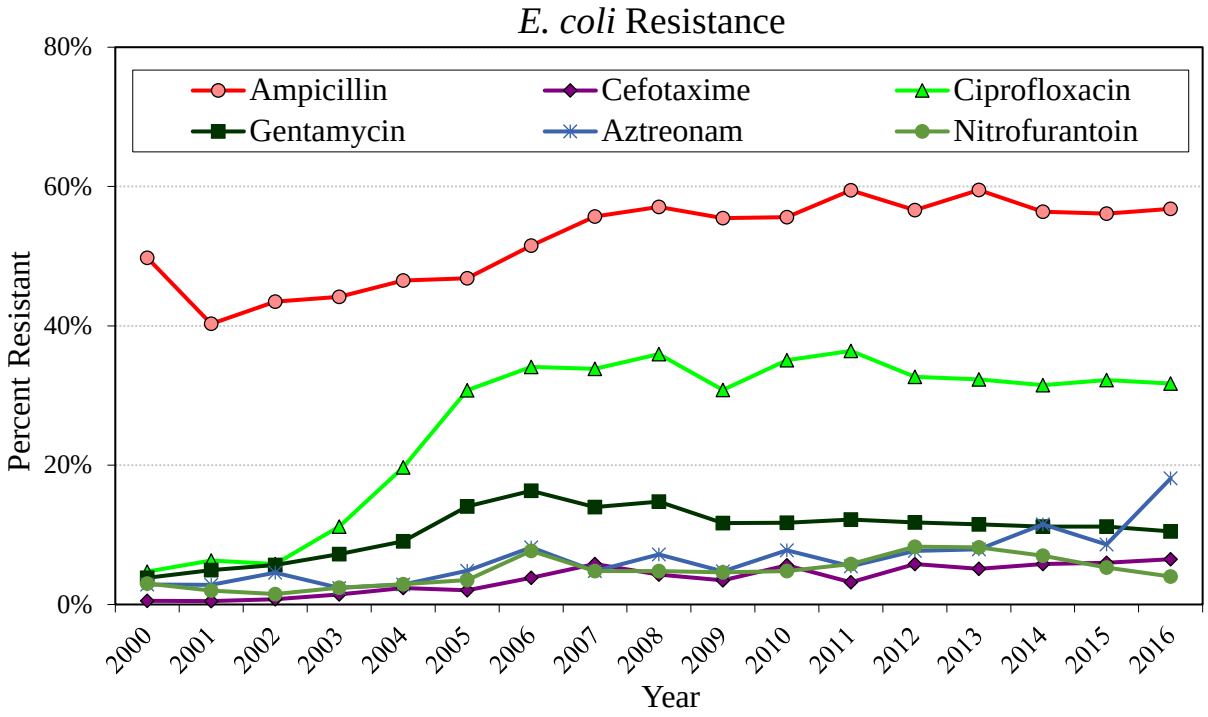

Figure 6. *E. coli* resistance rates in Louisiana from 2000 – 2016.

Table 10. *E coli* resistance to antibiotics in 2016.

2016 *E. coli*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Amikacin	Aminoglycosides	42116	3%	0%	83%
Amoxicillin	Penicillin Amino	281	22%	22%	22%
Ampicillin	Penicillin Amino	41647	57%	22%	76%
Azithromycin	Macrolides	301	2%	2%	2%
Aztreonam	Monobactam	33726	9%	0%	45%
Carbapenem	Carbapenem	1023	0%	0%	0%
Cefazolin	Cephalosporin 1	42571	18%	0%	100%
Cefepime	Cephalosporin 4	46035	8%	0%	35%
Cefixime	Cephalosporin 3	1095	3%	2%	4%
Cefotaxime	Cephalosporin 3	6544	6%	0%	12%
Cefoxitin	Cephalosporin 2	17298	8%	0%	21%
Cefpodoxime	Cephalosporin 3	1515	15%	15%	15%
Ceftazidime	Cephalosporin 3	39189	10%	0%	85%
Ceftriaxone	Cephalosporin 3	50531	10%	0%	35%
Cefuroxime	Cephalosporin 2	12411	18%	2%	42%
Cephalothin	Cephalosporin 1	3211	61%	59%	65%
Ciprofloxacin	Quinolone	49724	33%	11%	71%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	20670	21%	0%	65%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	2583	15%	13%	18%
Clindamycin	Lincosamides	539	100%	100%	100%

Doxycycline	Cyclines	2819	44%	19%	100%
Ertapenem	Carbapenem	21220	0%	0%	4%
Gentamicin	Aminoglycosides	52356	11%	4%	24%
Imipenem	Carbapenem	17074	1%	0%	7%
Imipenem/Cilastatin	Carbapenem	12988	0%	0%	1%
Levofloxacin	Quinolone	37294	33%	11%	71%
Linezolid	Oxazolidinone	539	100%	100%	100%
Meropenem	Carbapenem	28735	1%	0%	22%
Moxifloxacin	Quinolone	1578	29%	21%	34%
Nitrofurantoin	Quinolone	47964	4%	0%	12%
Oxacillin	PenicillinRb-lactamase	539	100%	100%	100%
Piperacillin	Penicillin Ureido	2435	58%	55%	68%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	50273	5%	0%	27%
Rifampin	Rifamycin	539	100%	100%	100%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	40578	51%	11%	74%
Tetracycline	Cyclines	14686	30%	25%	36%
Ticarcillin	Penicillin Carboxy	825	12%	12%	12%
Tigecycline	Glycylcycline	5142	0%	0%	0%
Tobramycin	Aminoglycosides	48597	11%	4%	26%
Trimethoprim-sulfa	Sulfonamide	52355	36%	15%	71%
Vancomycin	Glycopolypeptide	1635	65%	29%	100%

Ampicillin resistance is found in many *E.coli* strains due to their production of extended spectrum beta-lactamase (ESBL). Sensitivity to ampicillin has steadily increased to more than 55% overall in Louisiana. Resistance to cephalosporins is also increasing:

- *E.coli* became very resistant to ciprofloxacin in the early 2000s
- Resistance to aminoglycosides also began increasing around 2004, but has since plateaued
- Resistance to aztreonam is increasing

This trend is shown the graph (Fig. 6) pictured above.

Antibiotics of choice for treatment of *E. coli* infections include third generation cephalosporins, fluoroquinolones, trimethoprim/sulfamethoxazole, nitrofurantoin, piperacillin/tazobactam, imipenem/cilastin, and meropenem.

3.14.2-*Klebsiella pneumoniae*

Klebsiella pneumoniae may cause community-acquired lobar pneumonia in patients with severe underlying medical conditions. More importantly, these organisms have a predisposition to cause nosocomial infections such as ventilator-associated pneumonia, meningitis, cellulitis and UTIs. *K. pneumoniae* is the most common pathogen in ICUs.

The use of ampicillin as a course of treatment for *K. pneumoniae* ceased by 2012 because of its increasingly high resistance over the years. The other antibiotics displayed in the graph below (Fig. 7) have shown lower rates of resistance. In 2016, *K. pneumoniae* resistance to aztreonam increased from 12% in 2015 to 23%. This spike is largely driven by two facilities. Reasons as to why those facilities have such high rates of resistance relative to other facilities in the state are being investigated.

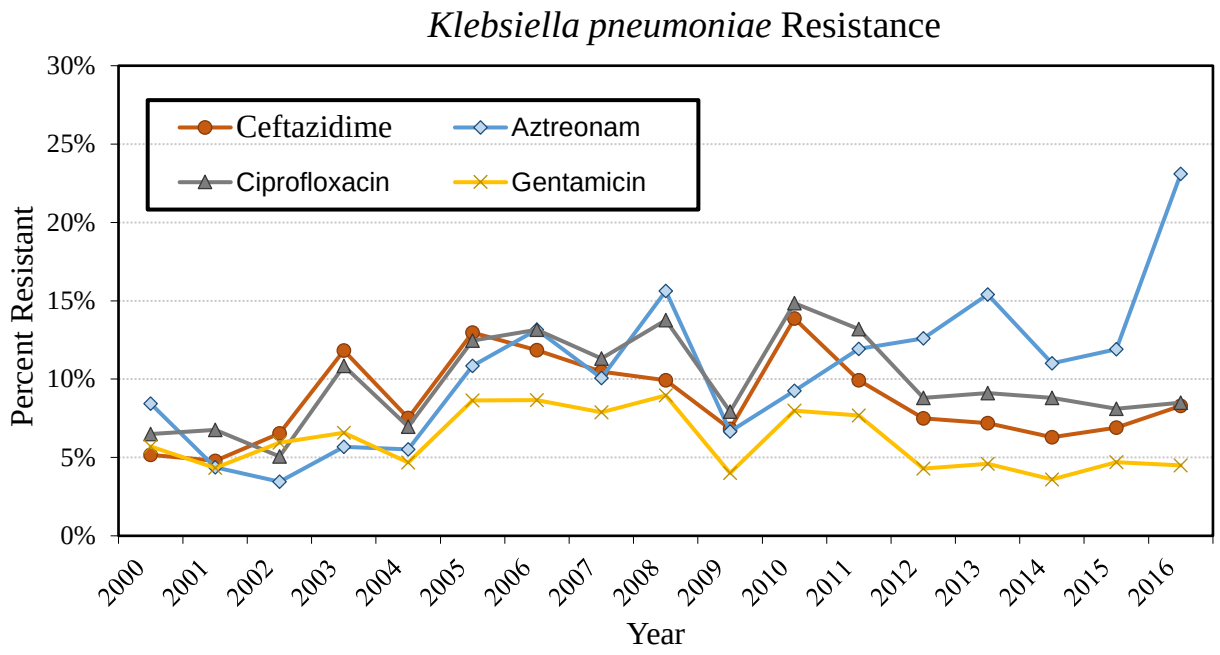

Figure 7. *K. pneumoniae* resistance rates in Louisiana from 2000 – 2016.

Table 11. *K. pneumoniae* resistance to antibiotics in 2016.

2016 *Klebsiella pneumoniae*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Amikacin	Aminoglycosides	9637	1%	0%	6%
Ampicillin	Penicillin Amino	429	100%	100%	100%
Azithromycin	Macrolides	105	2%	2%	2%
Aztreonam	Monobactam	7716	9%	0%	54%
Carbapenem	Carbapenem	262	0%	0%	0%
Cefazolin	Cephalosporin 1	9711	11%	0%	35%
Cefepime	Cephalosporin 4	10009	8%	0%	35%
Cefixime	Cephalosporin 3	250	0%	0%	0%
Cefotaxime	Cephalosporin 3	999	4%	0%	8%
Cefoxitin	Cephalosporin 2	4108	8%	3%	15%
Cefpodoxime	Cephalosporin 3	558	9%	9%	9%
Ceftazidime	Cephalosporin 3	8811	7%	0%	22%
Ceftriaxone	Cephalosporin 3	11650	8%	0%	35%
Cefuroxime	Cephalosporin 2	2009	10%	1%	22%
Cephalothin	Cephalosporin 1	169	12%	12%	12%
Ciprofloxacin	Quinolone	11189	9%	0%	35%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	4371	8%	2%	17%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	374	7%	7%	8%
Clindamycin	Lincosamides	118	100%	100%	100%

Doxycycline	Cyclines	552	35%	0%	100%
Ertapenem	Carbapenem	5115	2%	0%	12%
Gentamicin	Aminoglycosides	11715	4%	0%	11%
Imipenem	Carbapenem	3082	0%	0%	1%
Imipenem/Cilastatin	Carbapenem	2854	1%	0%	4%
Levofloxacin	Quinolone	7821	8%	0%	35%
Linezolid	Oxazolidinone	118	100%	100%	100%
Meropenem	Carbapenem	7093	1%	0%	8%
Moxifloxacin	Quinolone	408	10%	3%	20%
Nitrofurantoin	Quinolone	10213	61%	21%	84%
Oxacillin	PenicillinRb-lactamase	118	100%	100%	100%
Piperacillin	Penicillin Ureido	169	47%	47%	47%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	11675	6%	0%	30%
Rifampin	Rifamycin	118	100%	100%	100%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	9123	21%	4%	62%
Tetracycline	Cyclines	3174	18%	10%	28%
Ticarcillin	Penicillin Carboxy	169	15%	15%	15%
Tigecycline	Glycylcycline	963	1%	0%	4%
Tobramycin	Aminoglycosides	10533	6%	0%	24%
Trimethoprim-sulfa	Sulfonamide	11672	13%	0%	33%
Vancomycin	Glycopolypeptide	118	100%	100%	100%

Antibiotics of choice for *K. pneumoniae* infections include third-generation cephalosporins, carbapenems, aminoglycosides and quinolones. These antibiotics may be used as monotherapy or combination therapy. Other antibiotics that may be used are ampicillin/sulbactam, piperacillin/tazobactam, ticarcillin/clavulanate, and cefepime.

3.14.3-*Salmonella*

Salmonella is a group of organisms containing numerous serotypes, many of which are pathogenic for both animals and humans. Those that are pathogenic for humans fall within the species *S. enterica*. Ingestion of contaminated food is the main mode of transmission, with a few cases originating from contaminated water or from person-to-person transmission via the fecal-oral route. Gastroenteritis and enteric fever are the main clinical syndromes observed. *Salmonella* is periodically the source of foodborne outbreaks, usually arising from undercooked egg products, raw dairy, or contaminated meat.

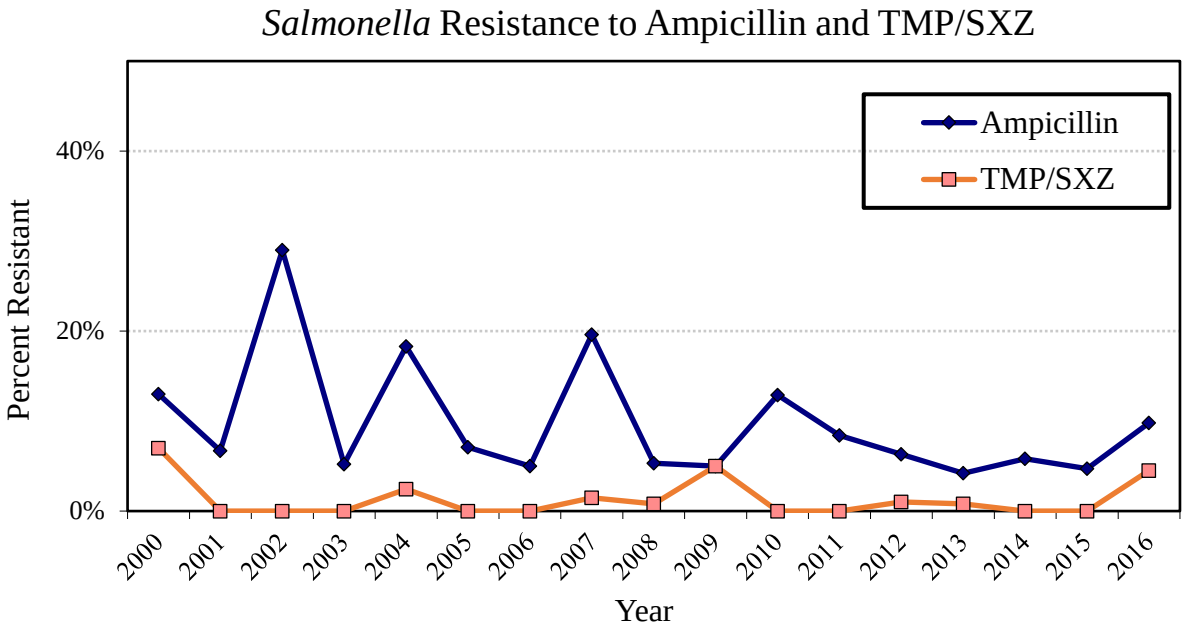

Figure 8. *Salmonella* resistance rates in Louisiana from 2000 – 2016.

In most cases of simple enterocolitis due to *Salmonella,* no treatment is necessary. They do not appear to shorten the duration of symptoms and may prolong the carrier state. For severe enterocolitis and invasive disease (typhoid fever, paratyphoid fever) treatment is recommended. Antibiotics recommended include quinolone, macrolide, and third-generation cephalosporin pending sensitivities. Antibiotic resistance trends are shown in Figure 8 above and in Table 12 below.

Table 12. *Salmonella* resistance to antibiotics in 2016.

2016 *Salmonella*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Ampicillin	Penicillin Amino	132	10%	9%	11%
Cefotaxime	Cephalosporin 3	57	2%	2%	2%
Ceftriaxone	Cephalosporin 3	10	0%	0%	0%
Ciprofloxacin	Quinolone	67	0%	0%	0%
Levofloxacin	Quinolone	10	0%	0%	0%
Trimethoprim-sulfa	Sulfonamide	67	3%	0%	5%

3.14.4-*Shigella*

Shigella are responsible for acute gastroenteritis and bacillary dysentery transmitted by the fecal-oral route. They are a frequent cause of community outbreaks, particularly among daycare centers, homosexual men, and in overcrowded or unsanitary conditions.

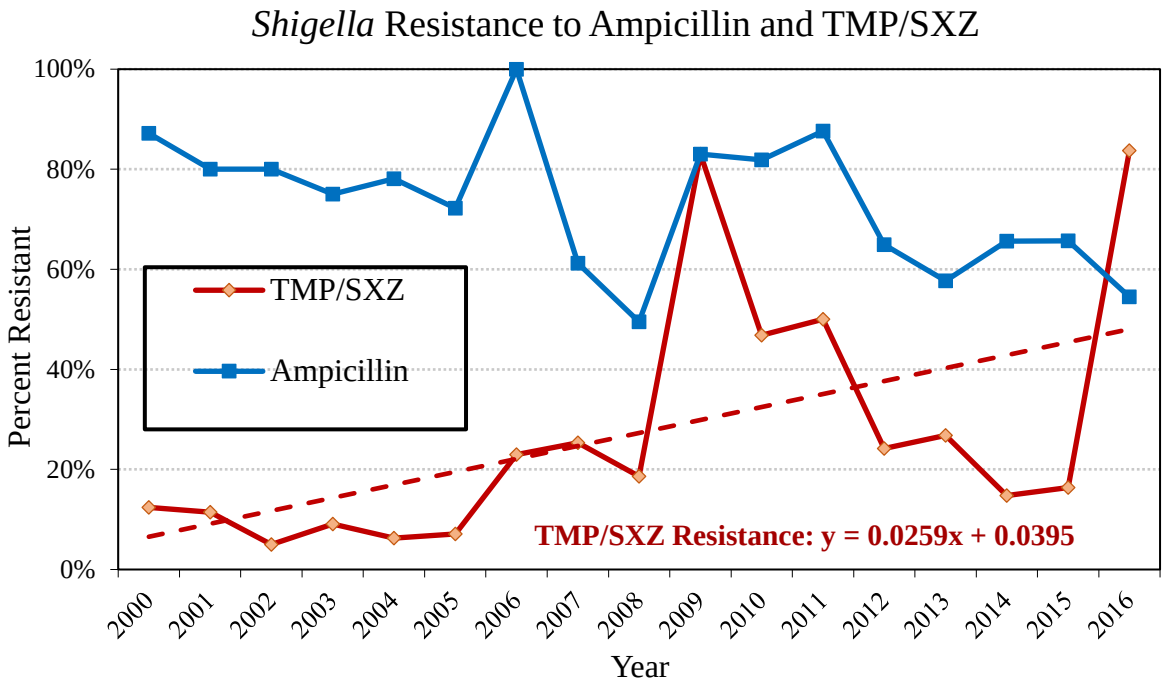

Figure 9. *Shigella* resistance rates in Louisiana from 2000 – 2016.

Table 13. *Shigella* resistance to antibiotics in 2016.

2016 *Shigella*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Ampicillin	Penicillin Amino	44	77%	44%	100%
Cefotaxime	Cephalosporin 3	34	0%	0%	0%
Ceftriaxone	Cephalosporin 3	2	0%	0%	0%
Ciprofloxacin	Quinolone	34	0%	0%	0%
Levofloxacin	Quinolone	8	0%	0%	0%
Trimethoprim-sulfa	Sulfonamide	43	77%	50%	100%

Antibiotics may not be required in individuals who are otherwise healthy. If antibiotic therapy is needed, the antibiotic susceptibility testing is essential before giving treatment. There is widespread resistance to ciprofloxacin, trimethoprim/sulfamethoxazole, and azithromycin. A third-generation cephalosporin or quinolone are the antibiotics of choice. As seen in Figure 9 and Table 13 above, TMP-SMX has a high rate of resistance along with ampicillin, which has seen some decline in resistance in recent years.

3.14.5-*Enterobacter cloacae*

Enterobacter species, particularly *Enterobacter cloacae* and *Enterobacter aerogenes*, are important nosocomial pathogens responsible for various infections, including bacteremia, lower respiratory tract infections, skin and soft-tissue infection, urinary tract infections (UTI), endocarditis, intra-abdominal infections septic arthritis, osteomyelitis and ophthalmic infections. *Enterobacter* species can also cause

various community-acquired infections, including UTIs, skin and soft-tissue infections, and wound infections among others.

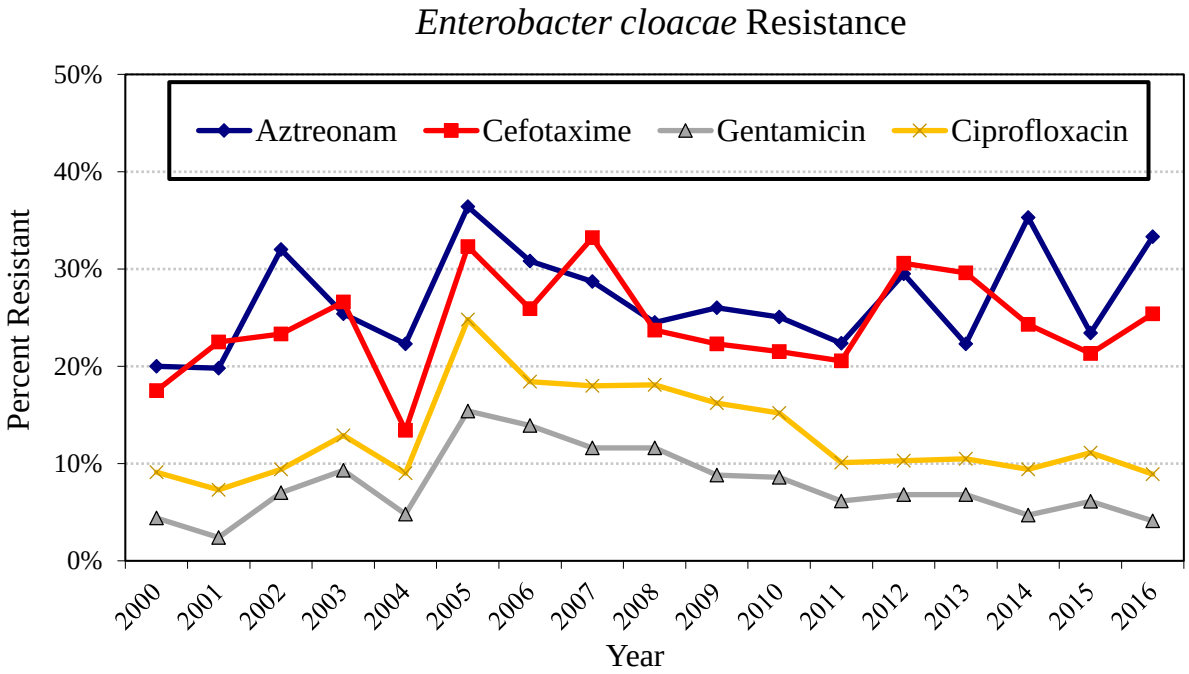

Figure 10. *Enterobacter cloacae* resistance rates in Louisiana from 2000 – 2016.

Table 14. *Enterobacter cloacae* resistance to antibiotics in 2016.

2016 *Enterobacter cloacae*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Amikacin	Aminoglycosides	1896	0%	0%	3%
Ampicillin	Penicillin Amino	234	79%	12%	100%
Azithromycin	Macrolides	33	39%	39%	39%
Aztreonam	Monobactam	1482	22%	7%	76%
Cefazolin	Cephalosporin 1	734	87%	0%	100%
Cefepime	Cephalosporin 4	1876	7%	0%	27%
Cefotaxime	Cephalosporin 3	256	22%	8%	37%
Cefotetan	Cephalosporin 2	26	100%	100%	100%
Cefoxitin	Cephalosporin 2	60	100%	100%	100%
Cefpodoxime	Cephalosporin 3	69	35%	35%	35%
Cefprozil	Cephalosporin 2	37	0%	0%	0%
Ceftazidime	Cephalosporin 3	1691	19%	0%	37%
Ceftriaxone	Cephalosporin 3	2096	22%	7%	51%
Cefuroxime	Cephalosporin 2	278	87%	61%	100%
Cephalothin	Cephalosporin 1	26	100%	100%	100%
Ciprofloxacin	Quinolone	1995	9%	0%	26%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	186	99%	97%	100%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	92	59%	31%	100%
Doxycycline	Cyclines	70	7%	7%	7%

Ertapenem	Carbapenem	994	3%	0%	10%
Gentamicin	Aminoglycosides	2216	4%	0%	18%
Imipenem	Carbapenem	692	4%	0%	15%
Imipenem/Cilastatin	Carbapenem	432	1%	0%	2%
Levofloxacin	Quinolone	1295	7%	0%	18%
Meropenem	Carbapenem	1389	2%	0%	10%
Moxifloxacin	Quinolone	60	13%	10%	16%
Nitrofurantoin	Quinolone	1667	65%	26%	92%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	2004	15%	0%	33%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	362	100%	100%	100%
Tetracycline	Cyclines	624	15%	0%	28%
Tigecycline	Glycylcycline	163	0%	0%	0%
Tobramycin	Aminoglycosides	2109	6%	0%	18%
Trimethoprim-sulfa	Sulfonamide	2212	12%	0%	29%

E. cloacae cause significant morbidity and mortality. Infection management is complicated by resistance to multiple antibiotics. *Enterobacter* species possess inducible β-lactamases, which are undetectable in vitro but are responsible for resistance during treatment.

For severe *Enterobacter* infections, carbapenems are the most reliable drug of choice and fourth-generation cephalosporins are a distant second choice. Other antibiotics of choice include aminoglycosides, fluoroquinolones, and trimethoprim-sulfamethoxazole. The rate of resistance to some of these antibiotics can be found in the graph above (Fig. 10).

3.14.6-*Proteus mirabilis*

Proteus organisms are most commonly found in the human intestinal tract as part of normal human intestinal flora, along with *Escherichia coli* and *Klebsiella* species, of which *E coli* is the predominant resident. However, they are often implicated as serious causes of infections in humans. *Proteu*s are prone to colonize and infect the urinary tract. Iatrogenic hematologic dissemination can occur after urologic procedures. Patients with recurrent infections, those with structural abnormalities of the urinary tract, those who have had urethral instrumentation, and those whose infections were acquired in the hospital have an increased frequency of infection caused by *Proteus* and other organisms (e.g., *Klebsiella, Enterobacter*, *Pseudomonas*, enterococci, staphylococci). *Proteus* are found in multiple environmental habitats, including long-term care facilities and hospitals.

Proteus mirabilis causes 90% of *Proteus* infections and can be considered a community-acquired infection. *Proteus vulgaris* and *Proteus penneri* are easily isolated from individuals in long-term care facilities and hospitals, and from patients with underlying diseases or compromised immune systems.

Proteus vulgaris is indole-positive and has more antibiotic resistance. *Proteus mirabilis*, which is indole-negative, is the most common species encountered in humans (90%).

P mirabilis remains susceptible to many antimicrobials except cyclines (Table 15). Resistance does not appear to be a significant clinical factor, but 10% to 30% of strains have acquired resistance to ampicillin and some cephalosporins. Acquisition of resistance to extended-spectrum alpha-lactamases

remains uncommon in *Proteus*. The trends in resistance to gentamicin and ciprofloxacin are shown in the graph below (Fig. 11).

P vulgaris and *P penneri* show higher resistance to ampicillin and first-generation cephalosporins. Activation of an inducible chromosomal beta-lactamase (not found in *P mirabilis*) occurs in up to 30% of these strains. Imipenem, fourth-generation cephalosporins, aminoglycosides, TMP-SMX, and quinolones have excellent activity (90%-100%).

Table 15. *Proteus mirabilis* resistance to antibiotics in 2016.

2016 *Proteus mirabilis*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Amikacin	Aminoglycosides	5750	1%	0%	6%
Ampicillin	Penicillin Amino	5333	23%	1%	83%
Azithromycin	Macrolides	60	0%	0%	0%
Aztreonam	Monobactam	4371	10%	0%	46%
Carbapenem	Carbapenem	183	0%	0%	0%
Cefazolin	Cephalosporin 1	5870	15%	0%	100%
Cefepime	Cephalosporin 4	5847	5%	0%	34%
Cefixime	Cephalosporin 3	142	0%	0%	0%
Cefotaxime	Cephalosporin 3	756	4%	0%	9%
Cefotetan	Cephalosporin 2	102	1%	1%	1%
Cefoxitin	Cephalosporin 2	2292	6%	0%	36%
Cefpodoxime	Cephalosporin 3	333	6%	6%	6%
Ceftazidime	Cephalosporin 3	5323	5%	0%	36%
Ceftriaxone	Cephalosporin 3	6887	5%	0%	33%
Cefuroxime	Cephalosporin 2	1246	6%	0%	22%
Cephalothin	Cephalosporin 1	266	14%	11%	16%
Ciprofloxacin	Quinolone	6675	31%	0%	75%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	2366	6%	0%	20%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	285	1%	0%	2%
Clindamycin	Lincosamides	47	100%	100%	100%
Doxycycline	Cyclines	245	100%	99%	100%
Ertapenem	Carbapenem	3114	1%	0%	4%
Gentamicin	Aminoglycosides	6872	12%	0%	50%
Imipenem	Carbapenem	862	12%	0%	29%
Imipenem/Cilastatin	Carbapenem	1059	23%	23%	23%
Levofloxacin	Quinolone	4930	35%	0%	75%
Linezolid	Oxazolidinone	47	100%	100%	100%
Meropenem	Carbapenem	3832	1%	0%	23%
Moxifloxacin	Quinolone	207	40%	35%	51%
Nitrofurantoin	Quinolone	1817	100%	99%	100%
Oxacillin	PenicillinRb-lactamase	47	100%	100%	100%
Piperacillin	Penicillin Ureido	164	21%	20%	21%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	6812	2%	0%	23%
Rifampin	Rifamycin	47	100%	100%	100%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	5417	15%	0%	39%
Tetracycline	Cyclines	359	100%	99%	100%

Ticarcillin	Penicillin Carboxy	89	0%	0%	0%
Tigecycline	Glycylcycline	102	100%	100%	100%
Tobramycin	Aminoglycosides	6211	9%	0%	31%
Trimethoprim-sulfa	Sulfonamide	6855	27%	4%	66%
Vancomycin	Glycopolypeptide	47	100%	100%	100%

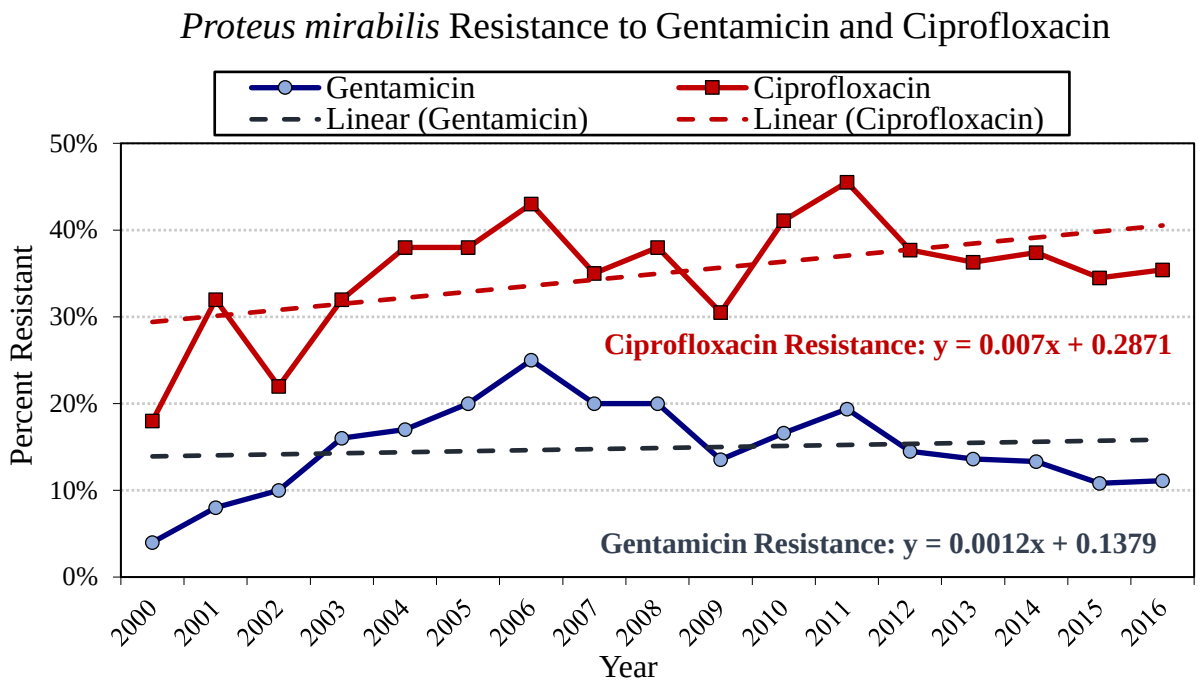

Figure 11. *Proteus mirabilis* resistance rates in Louisiana from 2000 – 2016.

3.14.7-*Serratia marcescens*

Members of this genus produce characteristic red pigment, prodigiosin. *S. marcescens*, was formerly known as *Bacillus prodigiosus* because it produced a bright red color on communion bread. It was also thought to be non-pathogenic and was used to study the dispersal of bacteria throughout the atmosphere (California coastal area 1950). In fact, *Serratia marcescens* is the only pathogen in this genus and usually causes nosocomial infections.

In the hospital, *Serratia* species tend to colonize the respiratory and urinary tracts, rather than the gastrointestinal tract in adults. *Serratia* infection is responsible for about 2% of nosocomial infections of the bloodstream, lower respiratory tract, urinary tract, surgical wounds, and skin and soft tissues in adult patients. Outbreaks of *S. marcescens* meningitis, wound infections, and arthritis have occurred in pediatric wards.

S. marcescens is naturally resistant to ampicillin, macrolides, and first generation cephalosporins. Antibiotics of choice in treatment of *Serratia* infections include aminoglycoside plus an antipseudomonal beta-lactam, amikacin, and quinolones. There are reports that indicate increasing resistance to gentamicin and tobramycin. Cefepime may be a treatment option for strains that produce AmpC β-lactamase.

Table 16. *Serratia marcescens* resistance to antibiotics in 2016.

2016 *Serratia marcescens*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Amikacin	Aminoglycosides	762	2%	0%	14%
Ampicillin	Penicillin Amino	95	95%	85%	100%
Aztreonam	Monobactam	436	9%	0%	23%
Cefazolin	Cephalosporin 1	236	100%	100%	100%
Cefepime	Cephalosporin 4	864	1%	0%	10%
Cefotaxime	Cephalosporin 3	83	22%	17%	27%
Cefoxitin	Cephalosporin 2	73	72%	64%	77%
Ceftazidime	Cephalosporin 3	647	17%	0%	52%
Ceftriaxone	Cephalosporin 3	930	10%	0%	40%
Cefuroxime	Cephalosporin 2	121	99%	99%	100%
Ciprofloxacin	Quinolone	815	14%	0%	64%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	46	100%	100%	100%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	37	5%	5%	5%
Doxycycline	Cyclines	20	90%	90%	90%
Ertapenem	Carbapenem	410	2%	0%	17%
Gentamicin	Aminoglycosides	976	2%	0%	10%
Imipenem	Carbapenem	181	11%	0%	33%
Imipenem/Cilastatin	Carbapenem	161	2%	2%	2%
Levofloxacin	Quinolone	527	4%	0%	11%
Meropenem	Carbapenem	628	1%	0%	5%
Nitrofurantoin	Quinolone	274	100%	100%	100%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	571	13%	0%	31%
Tetracycline	Cyclines	109	92%	89%	96%
Tigecycline	Glycylcycline	64	1%	0%	4%
Tobramycin	Aminoglycosides	622	21%	0%	64%
Trimethoprim-sulfa	Sulfonamide	862	4%	0%	33%

3.14.8-*Citrobacter freundii*

Citrobacter can be found almost everywhere in soil, water, wastewater, etc. It can also be found in the human intestine. They are rarely the source of illnesses, except for infections of the urinary tract and infant meningitis and sepsis.

C. freundii strains have inducible ampC genes encoding resistance to ampicillin and first-generation cephalosporins. In addition, isolates of *Citrobacter* may be resistant to multiple other antibiotics as a result of plasmid-encoded resistance genes.

Citrobacter infections follow the principles for treatment of other *Enterobacteriaceae* infections because there are no comparative studies of antibiotic therapy. The preferred treatment for *C. freundii* infections are based on an in vitro study done and include aminoglycosides, fluoroquinolones, carbapenems and fourth-generation cephalosporins. The first line drugs of treatment for *C. koseri* include third-generation cephalosporins, aztreonam and piperacillin. Alternative treatment choices include those also used in treatment for *C. freundii*.

Table 17. *Citrobacter freundii* resistance to antibiotics in 2016.

2016 *Citrobacter freundii*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Amikacin	Aminoglycosides	516	1%	0%	6%
Ampicillin	Penicillin Amino	64	81%	64%	100%
Aztreonam	Monobactam	287	15%	0%	27%
Cefazolin	Cephalosporin 1	217	50%	0%	100%
Cefepime	Cephalosporin 4	517	2%	0%	12%
Cefixime	Cephalosporin 3	18	0%	0%	0%
Cefotaxime	Cephalosporin 3	17	41%	41%	41%
Cefoxitin	Cephalosporin 2	5	80%	80%	80%
Cefpodoxime	Cephalosporin 3	52	19%	19%	19%
Ceftazidime	Cephalosporin 3	489	20%	0%	59%
Ceftriaxone	Cephalosporin 3	597	16%	0%	27%
Cefuroxime	Cephalosporin 2	50	24%	24%	24%
Ciprofloxacin	Quinolone	575	11%	0%	33%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	22	90%	80%	100%
Doxycycline	Cyclines	47	7%	0%	14%
Ertapenem	Carbapenem	193	1%	0%	3%
Gentamicin	Aminoglycosides	614	10%	0%	53%
Imipenem	Carbapenem	216	2%	0%	8%
Imipenem/Cilastatin	Carbapenem	148	1%	1%	1%
Levofloxacin	Quinolone	461	10%	0%	18%
Meropenem	Carbapenem	368	0%	0%	2%
Nitrofurantoin	Quinolone	532	9%	0%	40%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	518	10%	0%	24%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	5	60%	60%	60%
Tetracycline	Cyclines	103	18%	12%	21%
Tigecycline	Glycylcycline	30	0%	0%	0%
Tobramycin	Aminoglycosides	438	6%	0%	29%
Trimethoprim-sulfa	Sulfonamide	505	23%	0%	53%

3.14.9-*Morganella morganii*

Morganella morganii is a commensal Gram-negative bacillus of the intestinal tract of humans and other mammals and reptiles. Few reports exist in the literature regarding infections caused by this organism. It is an uncommon cause of community-acquired infections and nosocomial infections.

Antibiotic treatment should be initiated with an extended-spectrum antipseudomonal cephalosporin or penicillin combined with an aminoglycoside. Some preferred beta-lactam antibiotics include cefepime, ceftazidime, aztreonam, piperacillin and piperacillin-tazobactam. Carbapenems and intravenous fluoroquinolones should be reserved for resistant cases. With the widespread use of third-generation cephalosporins there has been an emergence of highly resistant *M. morganii*.

Table 18. *Morganella morganii* resistance to antibiotics in 2016.

2016 *Morganella morganii*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Amikacin	Aminoglycosides	494	1%	0%	5%
Ampicillin	Penicillin Amino	170	96%	83%	100%
Aztreonam	Monobactam	228	12%	0%	37%
Cefazolin	Cephalosporin 1	263	96%	83%	100%
Cefepime	Cephalosporin 4	533	3%	0%	10%
Cefixime	Suprax	12	50%	50%	50%
Cefotaxime	Cephalosporin 3	22	18%	18%	18%
Cefotetan	Cephalosporin 2	22	9%	9%	9%
Cefoxitin	Cephalosporin 2	92	42%	9%	75%
Ceftazidime	Cephalosporin 3	473	19%	0%	33%
Ceftriaxone	Cephalosporin 3	576	12%	0%	35%
Cefuroxime	Cephalosporin 2	65	89%	82%	95%
Cephalothin	Cephalosporin 1	22	100%	100%	100%
Ciprofloxacin	Quinolone	519	39%	0%	100%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	68	95%	83%	100%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	22	9%	9%	9%
Ertapenem	Carbapenem	194	0%	0%	3%
Gentamicin	Aminoglycosides	576	16%	0%	46%
Imipenem	Carbapenem	188	49%	5%	100%
Imipenem/Cilastatin	Carbapenem	169	12%	12%	12%
Levofloxacin	Quinolone	467	39%	0%	100%
Meropenem	Carbapenem	347	1%	0%	4%
Nitrofurantoin	Quinolone	221	99%	97%	100%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	576	4%	0%	22%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	257	84%	0%	100%
Tetracycline	Cyclines	81	74%	59%	92%
Tigecycline	Glycylcycline	22	50%	50%	50%
Tobramycin	Aminoglycosides	335	11%	0%	46%
Trimethoprim-sulfa	Sulfonamide	576	35%	0%	100%

3.14.10-*Providencia stuartii*

Providencia stuartii is an opportunistic pathogen seen in patients with severe burns or long-term indwelling urinary catheters. In animals, P. stuartii infections can cause neonatal diarrhea in dairy cows. In humans, P. stuartii can be isolated from urine (most common), stool and blood, as well as from sputum, skin and wound cultures. P. stuartii septicemia is primarily of urinary origin. It is the most common cause of purple urine bag syndrome.

Good first-line antibiotics for non-life-threatening infections include amikacin and beta-lactam/beta-lactamase inhibitors such as piperacillin/tazobactam. Carbapenems are the best choice for empirical therapy in life-threatening infections or nosocomial outbreaks. Once the susceptibility pattern is known, target therapy with the most narrow-spectrum agent to which the organism is susceptible.

Table 19. Providencia stuartii resistance to antibiotics in 2016.

2016 *Providencia stuartii*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Amikacin	Aminoglycosides	97	0%	0%	0%
Ampicillin	Penicillin Amino	24	96%	96%	96%
Aztreonam	Monobactam	24	0%	0%	0%
Cefazolin	Cephalosporin 1	24	0%	0%	0%
Cefepime	Cephalosporin 4	74	0%	0%	0%
Cefoxitin	Cephalosporin 2	7	0%	0%	0%
Ceftazidime	Cephalosporin 3	75	11%	0%	29%
Ceftriaxone	Cephalosporin 3	98	7%	0%	25%
Cefuroxime	Cephalosporin 2	7	57%	57%	57%
Ciprofloxacin	Quinolone	78	77%	70%	91%
Ertapenem	Carbapenem	44	2%	2%	2%
Gentamicin	Aminoglycosides	34	93%	90%	96%
Imipenem	Carbapenem	68	29%	0%	58%
Levofloxacin	Quinolone	91	70%	58%	91%
Meropenem	Carbapenem	18	0%	0%	0%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	98	1%	0%	5%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	56	95%	91%	100%
Tobramycin	Aminoglycosides	24	96%	96%	96%
Trimethoprim-sulfa	Sulfonamide	86	12%	0%	38%

3.15-*Pseudomonas aeruginosa*

Pseudomonas aeruginosa is a common bacterium which can cause infections in animals and humans. It is found in soil, water, and most man-made environments throughout the world. It thrives not only in normal atmospheres, but also with little oxygen, and has thus colonized many natural and artificial environments. It uses a wide range of organic material for food. In animals, this versatility enables the organism to infect damaged tissues or people with reduced immunity.

P. aeruginosa causes pneumonias (community-acquired but predominantly health care-associated), septicaemia, urinary tract infection, gastrointestinal infection (especially in premature infants and neutropenic cancer patients), and skin and soft tissue infections. It is often associated with diffuse bronchopneumonia, skin lesions of ecthyma gangerenosum, urinary tract catheterization, necrotizing enterocolitis (NEC), hemorrhage and necrosis.

Those at greatest risk of infection are cystic fibrosis patients, neutropenic patients, burn victims and patients with wound infections.

One of the most worrisome characteristics of *P. aeruginosa* is its low antibiotic susceptibility. This low susceptibility is attributable to a concerted action of multidrug efflux pumps with chromosomally-encoded antibiotic resistance genes (e.g. *mexAB*, *mexXY*) and the low permeability of the bacterial cellular envelopes. In addition to this intrinsic resistance, *P. aeruginosa* easily develops acquired resistance either by mutation in chromosomally-encoded genes or by the horizontal gene transfer of antibiotic resistance determinants. Development of multidrug resistance by *P. aeruginosa* isolates requires several different

genetic events, including acquisition of different mutations and/or horizontal transfer of antibiotic resistance genes. Hypermutation favors the selection of mutation-driven antibiotic resistance in *P. aeruginosa* strains producing chronic infections. Whereas the clustering of several different antibiotic resistance genes in integrons favors the concerted acquisition of antibiotic resistance determinants. Some recent studies have shown that phenotypic resistance associated to biofilm formation or to the emergence of small-colony variants may be important in the response of *P. aeruginosa* populations to antibiotic treatment. The resistance trends are shown in Figure 12 below.

Double drug therapy is recommended for serious infection, consisting of an anti-pseudomonal penicillin (piperacillin/tazobactam, ticarcillin/clavulanate), meropenem or cefepime, plus a fluoroquinolone or an aminoglycoside. Alternative antibiotics of choice include ceftazidime, other carbapenems, mezlocillin and ciprofloxacin.

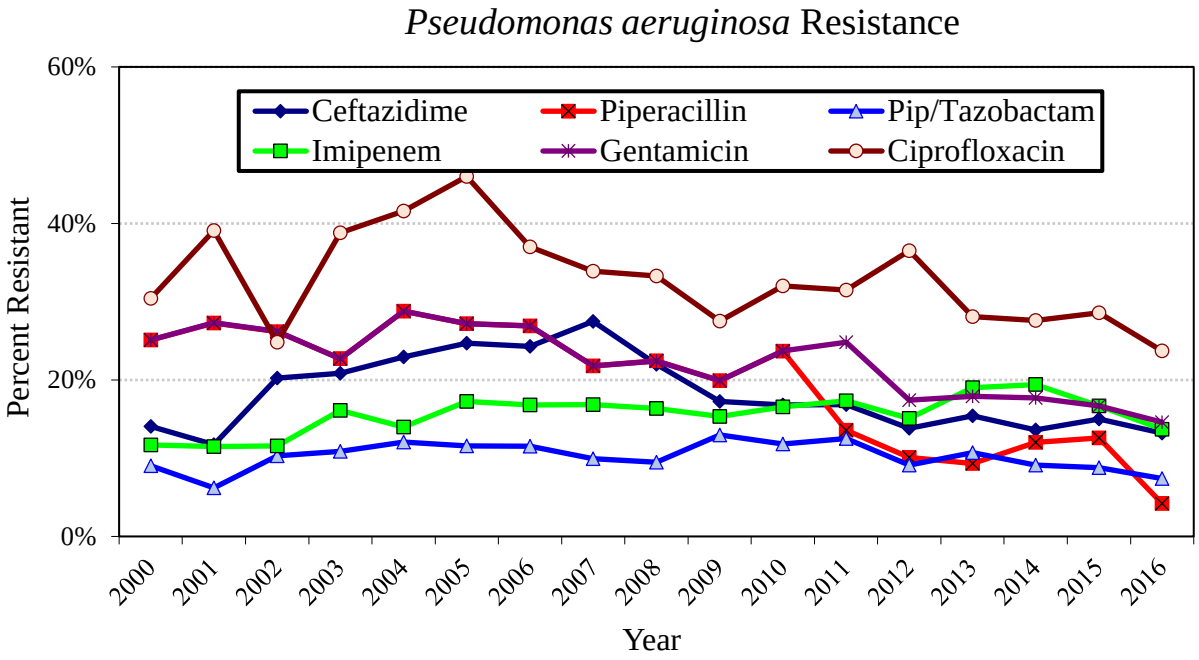

Figure 12. *Pseudomonas aeruginosa* resistance rates in Louisiana from 2000 – 2016.

Table 20. *Pseudomonas aeruginosa* resistance to antibiotics in 2016.

2016 *Pseudomonas aeruginosa*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Amikacin	Aminoglycosides	6815	5%	0%	27%
Ampicillin	Penicillin Amino	278	98%	98%	100%
Azithromycin	Macrolides	78	24%	24%	24%
Aztreonam	Monobactam	4286	25%	0%	45%
Cefazolin	Cephalosporin 1	464	99%	98%	100%
Cefepime	Cephalosporin 4	7678	14%	0%	44%
Cefotaxime	Cephalosporin 3	520	99%	97%	100%
Cefoxitin	Cephalosporin 2	313	99%	98%	100%
Ceftazidime	Cephalosporin 3	6694	14%	0%	72%

Ceftriaxone	Cephalosporin 3	579	98%	96%	100%
Ciprofloxacin	Quinolone	7787	25%	0%	59%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	130	98%	98%	98%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	544	26%	10%	67%
Clindamycin	Lincosamides	64	100%	100%	100%
Doxycycline	Cyclines	64	100%	100%	100%
Gentamicin	Aminoglycosides	7976	14%	0%	37%
Imipenem	Carbapenem	2241	11%	0%	20%
Imipenem/Cilastatin	Carbapenem	1946	18%	7%	42%
Levofloxacin	Quinolone	5459	29%	0%	54%
Linezolid	Oxazolidinone	64	100%	100%	100%
Meropenem	Carbapenem	5470	9%	0%	25%
Nitrofurantoin	Quinolone	194	99%	98%	100%
Oxacillin	PenicillinRb-lactamase	64	100%	100%	100%
Piperacillin	Penicillin Ureido	283	5%	0%	18%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	7697	7%	0%	27%
Rifampin	Rifamycin	64	100%	100%	100%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	508	99%	98%	100%
Tetracycline	Cyclines	234	99%	98%	100%
Ticarcillin	Penicillin Carboxy	266	17%	4%	25%
Tigecycline	Glycylcycline	130	98%	98%	98%
Tobramycin	Aminoglycosides	7369	5%	0%	22%
Trimethoprim-sulfa	Sulfonamide	480	75%	0%	100%
Vancomycin	Glycopolypeptide	64	100%	100%	100%

3.16-*Stenotrophomonas maltophilia*

Stenotrophomonas maltophilia is a Gram-negative rod which causes uncommon, but difficult to treat infections in humans. Initially classified as *Pseudomonas maltophilia*, *S. maltophilia* was also grouped in the genus *Xanthomonas* before eventually becoming the type species of the genus *Stenotrophomonas* in 1993.

S. maltophilia is ubiquitous in aqueous environments, soil and plants, including water, urine, or respiratory secretions. In immunocompromised patients, *S. maltophilia* can lead to nosocomial infections. *S. maltophilia* frequently colonizes breathing tubes such as endotracheal or tracheostomy tubes, the respiratory tract and indwelling urinary catheters. Infection is usually facilitated by the presence of prosthetic material (plastic or metal), and the most effective treatment is removal of the prosthetic material (usually a central venous catheter or similar device). The growth of *S. maltophilia* in microbiological cultures of respiratory or urinary specimens is therefore sometimes difficult to interpret and not proof of infection. If, however, it is grown from sites which would be normally sterile (e.g., blood), then it usually represents true infection.

In immunocompetent individuals, *S. maltophilia* is a relatively unusual cause of pneumonia, urinary tract infection, or blood stream infection; in immunocompromised patients, however, *S. maltophilia* is a growing source of latent pulmonary infections. *S. maltophilia* colonization rates in individuals with cystic fibrosis have been increasing.

S. maltophilia is usually resistant to aminoglycosides, antipseudomonal penicillins, and antipseudomonal third-generation cephalosporins. It is consistently susceptible to trimethoprim-sulfamethoxazole (TMP-SMX). If TMP-SMX cannot be used, the organism is usually sensitive to minocycline, respiratory quinolones, or colistin/polymixin B.

Table 21. *Stenotrophomonas maltophilia* resistance to antibiotics in 2016.

2016 *Stenotrophomonas maltophilia*					
Antibiotic	**Group**	**Sum of Isolates**	**Average Resistance**	**Low Resistance**	**High Resistance**
Ceftazidime	Cephalosporin 3	301	64%	36%	82%
Levofloxacin	Quinolone	330	17%	0%	50%
Minocycline	Cyclines	16	0%	0%	0%
Trimethoprim-sulfa	Sulfonamide	384	8%	0%	19%

4- Appendix

Table 4.1 - *S. aureus* /Oxacillin (Methicillin) Trend

	***S. aureus* /Oxacillin (Methicillin)Trend**		
	# Res	**Total**	**% Res**
2000	1,391	3,798	36.6%
2001	645	1,064	60.7%
2002	3,076	4,831	63.7%
2003	12,025	20,090	59.9%
2004	3,830	7,032	54.5%
2005	6,047	8,776	68.9%
2006	12,594	18,528	68.0%
2007	11,480	17,582	65.3%
2008	10,790	16,231	66.5%
2009	11,328	17,642	64.2%
2010	6,589	11,190	58.9%
2011	8,310	15,085	55.1%
2012	9,060	15,478	58.5%
2013	7,869	13,595	57.9%
2014	7,869	13,595	57.9%
2015	7,006	12,701	55.2%
2016	6067	10,805	56.1%

Table 4.2 - MRSA Trend

	MRSA Trend								
	Azithromycin			**Levofloxacin**			**Clindamycin***		
	# Res	**Total**	**% Res**	**# Res**	**Total**	**% Res**	**# Res**	**Total**	**% Res**
2000	106	116	91.0%	335	401	83.5%	153	401	38.3%
2001	321	346	92.7%	404	591	68.4%	233	800	29.1%
2002	214	233	91.9%	359	797	45.0%	140	797	17.6%
2003	89	95	93.8%	454	1,224	37.1%	584	3,275	17.8%
2004	446	478	93.4%	649	1,943	33.4%	734	3,808	19.3%
2005	78	78	100.0%	809	1,882	43.0%	667	4,413	15.1%
2006	184	198	93.0%	1,127	2,730	41.3%	1,166	6,902	16.9%
2007	449	471	95.2%	1,302	2,924	44.5%	593	3,829	15.5%
2008	383	431	88.8%	3,044	6,594	46.2%	1,180	4,930	23.9%
2009	336	430	78.1%	1,571	3,233	48.6%	691	2,953	23.4%
2010	355	379	93.6%	2,319	3,843	60.3%	1,527	5,814	26.3%
2011	355	402	88.3%	2,062	3,759	54.9%	1,369	6,077	22.5%
2012	487	550	88.5%	4,000	7,076	56.5%	1,708	8,627	19.8%
2013	203	241	84.2%	2,482	4,012	61.9%	1,282	6,144	20.9%
2014	113	134	84.0%	1,216	3,276	37.1%	791	5,900	13.4%
2015	125	140	89.3%	1,527	2,410	63.4%	1,249	5,139	24.3%
2016	104	118	88.1%	1,603	2,564	62.5%	1,648	6,161	26.7%

*Reports made do not specify if D test was made.

	MRSA Trend (cont'd)								
	Gentamycin			Rifampin^			Trimethoprim/Sulfa		
	# Res	Total	% Res	# Res	Total	% Res	# Res	Total	% Res
2000	102	401	25.4%	20	401	5.0%	33	598	5.6%
2001	115	811	14.2%	27	739	3.7%	42	902	4.7%
2002	39	617	6.3%	5	539	1.0%	24	797	3.1%
2003	125	2,750	4.5%	8	550	1.4%	134	3,355	4.0%
2004	125	3,788	3.3%	40	2,438	1.6%	68	3,608	1.9%
2005	239	4,830	4.9%	7	692	1.0%	198	5,518	3.6%
2006	308	7,281	4.2%	50	3,067	1.6%	251	7,753	3.2%
2007	146	4,358	3.3%	57	3,579	1.6%	135	5,058	2.7%
2008	153	7,629	2.0%	97	6,634	1.5%	173	8,154	2.1%
2009	116	3,843	3.0%	56	3,355	1.7%	189	3,540	5.4%
2010	173	6,366	2.7%	117	5,031	2.3%	184	6,461	2.8%
2011	102	4,149	2.5%	78	5,439	1.4%	164	5,964	2.7%
2012	229	7,472	3.1%	103	6,376	1.6%	223	9,969	2.2%
2013	88	3,318	2.7%	68	3,827	1.8%	214	6,336	3.4%
2014	65	3,839	1.7%	44	3,875	1.1%	114	5,768	2.0%
2015	45	2,178	2.1%	33	2,590	1.3%	200	5,268	3.8%
2016	47	2,691	1.7%	50	4,056	1.2%	205	6,714	3.1%

^Always to be used in conjunction with another antibiotic

Table 4.3 - *S. pneumoniae* / Penicillin Trend

	***S. pneumoniae* / Peni Trend**		
	# Res	**Total**	**% Res**
2000	108	242	44.7%
2001	60	110	54.7%
2002	154	400	38.4%
2003	317	839	37.8%
2004	153	414	37.1%
2005	232	635	36.5%
2006	322	744	43.3%
2007	727	1,388	52.4%
2008	498	970	51.4%
2009	317	655	48.4%
2010	410	764	53.7%
2011	529	1,112	47.6%
2012	683	1,747	39.1%
2013	481	1,269	37.9%
2014	192	1,008	19.1%
2015	97	505	19.2%
2016	196	623	31.5%

Table 4.4 - *Streptococci* Group A Trend

	***Streptococci* Group A Trend**					
	Penicillin			**Erythromycin**		
	# Res	**Total**	**% Res**	**# Res**	**Total**	**% Res**
2008	0	588	0.0%	71	588	12.1%
2009	0	632	0.0%	94	632	14.9%
2010	0	608	0.0%	79	608	13.0%
2011	0	645	0.0%	90	645	14.0%
2012	0	529	0.0%	90	529	17.0%
2013	0	744	0.0%	112	744	15.1%
2014	0	716	0.0%	79	716	11.0%
2015	0	-	-	0	-	-
2016	0	568	0.0%	56	568	9.9%

Table 4.5 - *Streptococci* Group B Trend

	***Streptococci* Group B Trend**											
	Penicillin			**Erythromycin**			**Tetracycline**			**Clindamycin**		
	# Res	**Total**	**% Res**	**# Res**	**Total**	**% Res**	**# Res**	**Total**	**% Res**	**# Res**	**Total**	**% Res**
2000	1	102	1.2%	19	175	11.0%	54	62	87.0%	8	62	12.9%
2001	1	83	1.2%	1	83	1.2%	7	9	78.0%	1	9	11.1%
2002	11	1,047	1.0%	95	1,221	7.8%	1,011	1,130	89.5%	42	331	12.7%
2003	25	3,448	0.7%	996	2,585	38.5%	2,078	2,439	85.2%	485	2,758	17.6%
2004	10	854	1.1%	208	563	36.9%	207	218	95.0%	160	677	23.6%
2005	6	935	0.7%	336	824	40.8%	732	873	83.8%	155	902	17.2%
2006	5	2,331	0.2%	1,071	2,143	50.0%	1,677	1,960	85.5%	535	2,122	25.2%
2007	55	3,302	1.7%	400	798	50.2%	476	581	81.9%	818	2,929	27.9%
2008	2	1,458	0.1%	1,572	2,089	75.3%	1,694	2,032	83.4%	1,295	3,141	41.2%
2009	3	2,157	0.1%	1,983	2,709	73.2%	2,249	2,628	85.6%	1,973	2,999	65.8%
2010	4	3,360	0.1%	2,376	2,864	83.0%	2,396	2,749	87.1%	1,538	3,299	46.6%
2011	5	856	0.6%	447	814	54.9%	568	686	82.8%	539	1,216	44.3%
2012	7	1,188	0.6%	346	626	55.3%	532	641	83.0%	316	727	43.5%
2013	8	1,425	0.6%	470	853	55.1%	667	826	80.8%	390	896	43.5%
2014	3	805	0.3%	220	395	55.8%	657	809	81.2%	370	745	49.7%
2015	0	1,685	0.0%	740	1,283	57.7%	551	687	80.2%	635	1,532	41.4%
2016	2	1,210	0.2%	863	1,404	61.5%	563	678	83.0%	960	1,940	49.5%

Table 4.6 - *Enterococcus* Trend

	***Enterococcus* Trend**								
	***E. fecalis* / Vancomycin**			***E. faecium* / Vancomycin**			***Enterococcus* spp / Vancomycin**		
	# Res	**Total**	**% Res**	**# Res**	**Total**	**% Res**	**# Res**	**Total**	**% Res**
2000	56	6,187	0.9%	240	615	39.0%	63	1,223	5.2%
2001	33	7,381	0.4%	327	769	42.5%	42	1,118	3.8%
2002	59	7,867	0.7%	378	817	46.3%	54	1,079	5.0%
2003	72	8,024	0.9%	414	821	50.4%	139	1,428	9.7%
2004	85	6,414	1.3%	376	693	54.3%	112	1,239	9.0%
2005	72	3,737	1.9%	289	505	57.2%	104	1,040	10.0%
2006	73	3,491	2.1%	276	482	57.3%	111	1,295	8.6%
2007	88	4,581	1.9%	446	743	60.0%	118	1,458	8.1%
2008	112	6,455	1.7%	524	907	57.8%	93	1,276	7.3%
2009	147	6,898	2.1%	538	973	55.3%	107	1,278	8.4%
2010	422	10,585	4.0%	1,256	1,837	68.4%	127	1,381	9.2%
2011	123	10,505	1.2%	641	1,339	47.9%	43	1,153	3.7%
2012	295	13,383	2.2%	1,388	1,990	69.7%	162	1,976	8.2%
2013	267	11,933	2.2%	1,263	1,839	68.7%	182	1,576	11.5%
2014	103	10,989	0.9%	714	1,525	46.8%	15	601	2.6%
2015	230	11,494	2.0%	1,279	1,751	73.0%	157	1,468	10.7%
2016	213	9,320	2.3%	860	1,243	69.2%	72	972	7.4%

Table 4.7 - *Haemophilus influenza*e Trend

	***Haemophilus influenzae* Trend**											
	Ceftriaxone			**TMP-SMX**			**Macrolides**			**Quinolones**		
	# Res	**Total**	**% Res**	**# Res**	**Total**	**% Res**	**# Res**	**Total**	**% Res**	**# Res**	**Total**	**% Res**
2000	0	129	0.0%	25	94	26.7%	-	-	-	0	121	0.0%
2001	0	14	0.0%	21	87	24.1%	-	-	-	0	95	0.0%
2002	0	115	0.0%	27	127	21.6%	-	-	-	0	18	0.0%
2003	1	187	0.4%	25	186	13.4%	-	-	-	0	34	0.0%
2004	3	85	3.8%	27	85	31.6%	-	-	-	0	84	0.0%
2005	0	43	0.0%	10	43	23.1%	1	13	8.0%	0	43	0.0%
2006	0	38	0.0%	2	38	5.0%	4	28	14.0%	1	38	3.0%
2007	6	293	2.1%	80	320	25.2%	16	65	25.0%	1	126	0.9%
2008	0	138	0.0%	65	224	28.8%	19	46	41.0%	0	102	0.0%
2009	0	154	0.0%	71	294	24.0%	24	75	32.0%	0	60	0.0%
2010	0	108	0.0%	56	196	28.5%	28	88	32.0%	0	56	0.0%
2011	0	205	0.0%	65	237	27.4%	12	62	19.4%	0	30	0.0%
2012	0	279	0.0%	104	358	29.1%	27	92	29.3%	0	39	0.0%
2013	1	115	0.9%	66	196	33.7%	30	81	37.0%	0	24	0.0%
2014	0	50	0.0%	27	108	25.0%	12	58	20.7%	1	10	10.0%
2015	9	94	9.6%	23	94	24.5%	0	-	-	4	40	10.0%
2016	1	49	2.0%	49	121	40.5%	3	72	4.2%	0	17	0.0%

Table 4.8 - *Acinetobacter* Trend

	***Acinetobacter* Trend**								
	Ceftazidime			**Piperacillin/Tazobactam**			**Carbapenem (class)**		
	# Res	**Total**	**% Res**	**# Res**	**Total**	**% Res**	**# Res**	**Total**	**% Res**
2000	19	88	21.6%	84	215	39.1%	-	-	-
2001	21	75	28.0%	3	50	6.0%	0	11	0.0%
2002	4	59	6.8%	2	49	4.1%	2	44	4.6%
2003	343	692	49.6%	158	554	28.5%	67	419	16.0%
2004	42	131	32.1%	42	194	21.6%	7	173	3.8%
2005	93	187	49.7%	66	187	35.3%	2	30	8.0%
2006	152	329	46.2%	101	274	36.9%	8	75	11.0%
2007	347	646	53.7%	130	233	55.8%	148	589	25.1%
2008	701	1,095	64.0%	412	541	76.2%	655	1,300	50.4%
2009	389	614	63.4%	245	464	52.8%	421	906	46.5%
2010	455	724	62.8%	188	297	63.3%	738	1,107	66.7%
2011	595	958	62.1%	95	163	58.3%	556	1,031	53.9%
2012	589	1,107	53.2%	203	234	86.8%	648	1,459	44.4%
2013	412	716	57.5%	49	80	61.3%	514	1,013	50.7%
2014	222	516	43.0%	21	59	35.6%	298	679	43.9%
2015	291	566	51.4%	137	205	66.8%	364	735	49.5%
2016	218	531	41.1%	193	359	53.8%	278	626	44.4%

Table 4.9 - *E. coli* Trend

	***E. coli* Trend**								
	Ampicillin			**Cefotaxime**			**Ciprofloxacin**		
	# Res	**Total**	**% Res**	**# Res**	**Total**	**% Res**	**# Res**	**Total**	**% Res**
2000	3,205	6,441	49.8%	20	4,015	0.5%	176	3,731	4.7%
2001	1,116	2,770	40.3%	12	2,511	0.5%	152	2,402	6.3%
2002	4,015	9,235	43.5%	57	7,580	0.8%	303	5,232	5.8%
2003	13,900	31,459	44.2%	136	9,370	1.4%	2,732	24,430	11.2%
2004	7,588	16,310	46.5%	134	5,687	2.4%	2,549	12,961	19.7%
2005	4,853	10,364	46.8%	47	2,300	2.1%	3,256	10,587	30.8%
2006	10,411	20,207	51.5%	278	7,277	3.8%	5,732	16,804	34.1%
2007	13,910	24,970	55.7%	553	9,514	5.8%	6,651	19,660	33.8%
2008	18,414	32,275	57.1%	477	11,156	4.3%	8,690	24,179	35.9%
2009	20,920	37,724	55.5%	421	12,127	3.5%	8,723	28,317	30.8%
2010	13,455	23,757	56.6%	303	5,399	5.6%	5,720	16,303	35.1%
2011	15,099	25,375	59.5%	317	9,874	3.2%	11,929	32,765	36.4%
2012	26,344	46,197	57.0%	489	8,439	5.8%	17,069	52,142	32.7%
2013	25,780	45,059	57.2%	238	4,694	5.1%	15,838	49,110	32.3%
2014	26,254	46,577	56.4%	411	7,123	5.8%	15,941	50,612	31.5%
2015	28,174	50,253	56.1%	277	4,616	6.0%	18,735	58,189	32.2%
2016	23,636	41,647	56.8%	428	6,544	6.5%	15,787	49,724	31.7%

	E. coli Trend (cont'd)								
	Gentamycin			Aztreonam			Nitrofurantoin		
	# Res	Total	% Res	# Res	Total	% Res	# Res	Total	% Res
2000	268	6,996	3.8%	117	4,059	2.9%	187	6,243	3.0%
2001	136	2,770	4.9%	54	1,935	2.8%	24	1,207	2.0%
2002	522	9,235	5.6%	173	3,782	4.6%	118	7,766	1.5%
2003	2,367	32,685	7.2%	293	12,297	2.4%	507	21,499	2.4%
2004	1,507	16,644	9.1%	191	6,658	2.9%	333	11,340	2.9%
2005	2,240	15,894	14.1%	382	7,902	4.8%	345	9,744	3.5%
2006	4,395	26,941	16.3%	1,110	13,550	8.2%	1,296	16,856	7.7%
2007	3,550	25,406	14.0%	850	17,794	4.8%	947	19,784	4.8%
2008	5,020	33,981	14.8%	2,023	28,236	7.2%	1,537	31,894	4.8%
2009	4,407	37,724	11.7%	1,309	27,687	4.7%	1,008	21,774	4.6%
2010	2,834	24,163	11.7%	1,091	14,056	7.8%	971	20,182	4.8%
2011	4,331	35,428	12.2%	1,221	22,317	5.5%	1,787	30,579	5.8%
2012	6,678	56,784	11.8%	2,663	34,680	7.7%	3,048	36,919	8.3%
2013	6,098	53,174	11.5%	2,776	35,184	7.9%	2,979	36,246	8.2%
2014	5,935	52,980	11.2%	3,948	34,362	11.5%	2,411	34,360	7.0%
2015	6,675	59,455	11.2%	3,492	40,440	8.6%	2,265	43,119	5.3%
2016	5,523	52,356	10.5%	6,111	33,726	18.1%	1,929	47,964	4.0%

Table 4.10 - *Klebsiella pneumoniae* Trend

	***Klebsiella pneumoniae* Trend**								
	Ampicillin			Ceftazidime			Aztreonam		
	# Res	Total	% Res	# Res	Total	% Res	# Res	Total	% Res
2000	1,531	1,721	89.0%	56	1,088	5.2%	93	1,097	8.4%
2001	541	578	93.7%	41	860	4.8%	32	737	4.4%
2002	2,209	2,245	98.4%	135	2,071	6.5%	34	984	3.5%
2003	6,496	6,652	97.7%	720	6,093	11.8%	186	3,281	5.7%
2004	3,438	3,557	96.7%	191	2,536	7.5%	122	2,212	5.5%
2005	2,259	2,316	97.6%	305	2,349	13.0%	279	2,571	10.8%
2006	4,269	4,473	95.4%	521	4,393	11.9%	433	3,290	13.1%
2007	4,764	4,824	98.7%	733	6,989	10.5%	606	6,016	10.1%
2008	4,327	4,631	93.4%	848	8,540	9.9%	1,188	7,598	15.6%
2009	378	385	98.1%	474	6,948	6.8%	1,001	15,036	6.7%
2010	389	479	81.2%	712	5,134	13.9%	187	2,019	9.3%
2011	323	341	94.7%	678	6,818	9.9%	724	6,062	11.9%
2012	-	-	-	713	8,491	8.4%	1,011	8,466	11.9%
2013	-	-	-	542	7,546	7.2%	1,167	7,556	15.4%
2014	-	-	-	562	8,986	6.3%	850	7,701	11.0%
2015	-	-	-	663	9,562	6.9%	1,029	8,633	11.9%
2016	429	429	100%	735	8,811	8.3%	1,779	7,716	23.1%

	Klebsiella pneumoniae Trend (cont'd)								
	Ciprofloxacin			Gentamicin			Carbapenem (class)		
	# Res	Total	% Res	# Res	Total	% Res	# Res	Total	% Res
2000	75	1,151	6.5%	112	1,969	5.7%	6	1848	0.3%
2001	58	854	6.8%	47	1,087	4.3%	8	909	0.8%
2002	68	1,340	5.1%	148	2,500	5.9%	6	2336	0.3%
2003	769	7,091	10.9%	615	9,353	6.6%	20	6021	0.3%
2004	228	3,272	7.0%	200	4,273	4.7%	34	3199	1.1%
2005	322	2,586	12.5%	349	4,039	8.6%	0	3064	0.0%
2006	487	3,704	13.2%	560	6,467	8.7%	186	5026	3.7%
2007	641	5,671	11.3%	627	7,952	7.9%	37	9616	0.4%
2008	877	6,365	13.8%	881	9,840	9.0%	95	13759	0.7%
2009	499	6,294	7.9%	344	8,591	4.0%	91	12945	0.7%
2010	635	4,276	14.8%	493	6,175	8.0%	42	6191	0.7%
2011	1,127	8,543	13.2%	691	8,995	7.7%	151	16270	0.9%
2012	1,073	11,447	9.4%	573	12,255	4.7%	230	21328	1.1%
2013	967	10,584	9.1%	513	11,132	4.6%	235	20311	1.2%
2014	927	10,476	8.8%	401	11,257	3.6%	398	20885	1.9%
2015	994	12,264	8.1%	586	12,522	4.7%	91	22283	0.4%
2016	948	11,189	8.5%	529	11,715	4.5%	158	18,144	0.9%

Table 4.11 - *Salmonella* spp. Trend

	Salmonella spp. Trend											
	Ampicillin			TMP-SMX			Cefotaxime			Ciprofloxacin		
	# Res	Total	% Res	# Res	Total	% Res	# Res	Total	% Res	# Res	Total	% Res
2000	2	16	13.0%	1	16	7.0%	0	16	0.0%	-	-	-
2001	1	15	6.7%	0	12	0.0%	0	15	0.0%	-	-	-
2002	2	7	29.0%	0	7	0.0%	0	12	0.0%	0	7	0.0%
2003	1	19	5.2%	0	19	0.0%	0	7	0.0%	0	18	0.0%
2004	7	38	18.3%	1	41	2.4%	0	7	0.0%	0	40	0.0%
2005	2	27	7.1%	0	27	0.0%	0	6	0.0%	0	27	0.0%
2006	6	113	5.0%	0	118	0.0%	0	118	0.0%	1	118	0.8%
2007	29	146	19.6%	2	142	1.5%	0	4	0.0%	0	142	0.0%
2008	7	127	5.3%	1	127	0.8%	0	22	0.0%	0	112	0.0%
2009	2	41	5.0%	2	41	5.0%	0	41	0.0%	0	41	0.0%
2010	18	136	12.9%	0	146	0.0%	0	61	0.0%	0	136	0.0%
2011	13	154	8.4%	0	154	0.0%	0	75	0.0%	0	154	0.0%
2012	12	191	6.3%	2	191	1.0%	0	83	0.0%	1	171	0.6%
2013	5	120	4.2%	1	132	0.8%	0	43	0.0%	1	132	0.8%
2014	8	138	5.8%	0	228	0.0%	0	72	0.0%	0	138	0.0%
2015	3	64	4.7%	0	64	0.0%	0	-	-	0	64	0.0%
2016	13	132	9.8%	3	67	4.5%	1	57	1.8%	0	67	0.0%

Table 4.12 - *Shigella* spp. Trend

	***Shigella* spp. Trend**											
	Ampicillin			**TMP-SMX**			**3rd Gen. Cephalosporin**			**Ciprofloxacin**		
	# Res	**Total**	**% Res**	**# Res**	**Total**	**% Res**	**# Res**	**Total**	**% Res**	**# Res**	**Total**	**% Res**
2000	41	47	87.2%	6	47	12.4%	0	12	0.0%	-	-	-
2001	4	5	80.0%	4	35	11.4%	0	10	0.0%	0	5	0.0%
2002	8	10	80.0%	2	41	5.0%	0	9	0.0%	0	1	0.0%
2003	9	12	75.0%	3	33	9.1%	0	7	0.0%	0	1	0.0%
2004	25	32	78.1%	2	32	6.3%	0	25	0.0%	0	31	0.0%
2005	26	36	72.2%	2	31	7.1%	0	1	0.0%	0	1	0.0%
2006	110	110	100.0%	25	110	23.0%	0	110	0.0%	1	110	0.9%
2007	97	158	61.2%	40	158	25.3%	0	52	0.0%	0	158	0.0%
2008	50	101	49.5%	19	102	18.6%	0	19	0.0%	0	104	0.0%
2009	5	6	83.0%	5	6	83.0%	0	15	0.0%	0	6	0.0%
2010	77	94	81.9%	44	94	46.8%	0	0	0.0%	0	94	0.0%
2011	113	129	87.6%	69	138	50.0%	0	75	0.0%	0	129	0.0%
2012	87	134	64.9%	32	132	24.2%	0	134	0.0%	0	134	0.0%
2013	41	71	57.7%	19	71	26.8%	0	71	0.0%	0	71	0.0%
2014	40	61	65.6%	9	61	14.8%	0	61	0.0%	0	61	0.0%
2015	44	67	65.7%	11	67	16.4%	0	67	0.0%	0	67	0.0%
2016	24	44	54.5%	36	43	83.7%	0	36	0.0%	0	34	0.0%

Table 4.13 - *Enterobacter cloacae* Trend

	***Enterobacter cloacae* Trend**								
	Aztreonam			**Cefotaxime**			**Gentamicin**		
	# Res	**Total**	**% Res**	**# Res**	**Total**	**% Res**	**# Res**	**Total**	**% Res**
2000	74	372	20.0%	66	378	17.5%	28	628	4.4%
2001	23	115	19.8%	31	140	22.5%	5	203	2.4%
2002	75	235	32.0%	117	504	23.3%	42	595	7.0%
2003	236	930	25.4%	223	840	26.6%	203	2,173	9.3%
2004	116	522	22.3%	35	261	13.4%	39	822	4.8%
2005	219	603	36.4%	33	101	32.3%	110	716	15.4%
2006	256	832	30.8%	72	278	25.9%	176	1,265	13.9%
2007	431	1,505	28.7%	300	901	33.2%	255	2,199	11.6%
2008	428	1,744	24.5%	130	551	23.7%	269	2,312	11.6%
2009	352	1,354	26.0%	153	685	22.3%	171	1,939	8.8%
2010	107	427	25.1%	44	205	21.5%	103	1,202	8.6%
2011	136	607	22.4%	111	540	20.6%	86	1,397	6.1%
2012	471	1,597	29.5%	111	363	30.6%	167	2,467	6.8%
2013	266	1,194	22.3%	107	361	29.6%	150	2,210	6.8%
2014	414	1,174	35.3%	84	346	24.3%	84	1,806	4.7%
2015	309	1,323	23.4%	36	169	21.3%	139	2,293	6.1%
2016	493	1,482	33.3%	65	256	25.4%	90	2,216	4.1%

	Enterobacter cloacae Trend (cont'd)								
	Ciprofloxacin			TMP/SMX			Carbapenems		
	# Res	Total	% Res	# Res	Total	% Res	# Res	Total	% Res
2000	37	348	10.6%	56	607	9.1%	2	598	0.3%
2001	20	169	11.6%	15	203	7.3%	2	180	1.1%
2002	26	402	6.4%	62	655	9.4%	3	593	0.5%
2003	243	1,748	13.9%	172	1331	12.9%	8	1,453	0.6%
2004	41	574	7.2%	62	686	9.0%	2	770	0.3%
2005	88	411	21.5%	146	587	24.8%	8	700	1.1%
2006	123	723	17.0%	192	1042	18.4%	28	1,211	2.3%
2007	250	1,518	16.5%	369	2053	18.0%	61	2,441	2.5%
2008	221	1,630	13.6%	394	2178	18.1%	94	3,098	3.0%
2009	193	1,493	12.9%	299	1848	16.2%	64	2,636	2.4%
2010	125	823	15.2%	225	1202	18.7%	46	1,658	2.8%
2011	134	1,329	10.1%	182	1397	13.1%	34	2,447	1.4%
2012	240	2,323	10.3%	331	2372	14.0%	84	4,032	2.1%
2013	221	2,099	10.5%	281	2033	13.8%	74	3,751	2.0%
2014	156	1,667	9.4%	216	1727	12.5%	92	2,985	3.1%
2015	249	2,253	11.1%	253	2120	11.9%	140	4,053	3.5%
2016	177	1,995	8.9%	266	2,212	12.0%	116	3,507	3.3%

Table 4.14 - *Proteus mirabilis* Trend

	Proteus mirabilis Trend								
	Ampicillin			Clavulanic-Ticarcillin			Ceftriaxone (3rd gen Ceph)		
	# Res	Total	% Res	# Res	Total	% Res	# Res	Total	% Res
2000	102	1,341	7.6%	7	1,261	0.5%	14	973	1.5%
2001	93	566	16.5%	7	606	1.1%	9	459	1.9%
2002	585	2,511	23.3%	26	1,063	2.4%	3	1,362	0.2%
2003	2,323	7,798	29.8%	26	2,135	1.2%	50	4,496	1.1%
2004	1,582	4,711	33.6%	10	1,380	0.7%	49	2,493	2.0%
2005	902	2,741	32.9%	2	682	0.3%	14	1,967	0.7%
2006	1,780	4,982	35.7%	123	1,489	8.3%	161	3,827	4.2%
2007	1,737	6,183	28.1%	165	2,653	6.2%	121	6,057	2.0%
2008	2,547	7,891	32.3%	16	2,217	0.7%	464	7,716	6.0%
2009	1,311	4,792	27.4%	21	2,584	0.8%	198	4,326	4.6%
2010	600	2,564	23.4%	2	875	0.2%	247	3,896	6.4%
2011	2,070	6,369	32.5%	4	1,265	0.3%	777	6,661	11.7%
2012	2,033	6,605	30.8%	19	2,076	0.9%	917	8,348	11.0%
2013	1,714	5,417	31.6%	17	1,812	0.9%	762	7,562	10.1%
2014	1,360	4,749	28.6%	15	1,938	0.8%	447	6,934	6.4%
2015	1,711	7,087	24.1%	4	418	1.0%	546	7,893	6.9%
2016	1,239	5,333	23.2%	3	285	1.1%	409	6,887	5.9%

	Proteus mirabilis Trend (cont'd)								
	Gentamicin			Ciprofloxacin			TMP/SMX		
	# Res	Total	% Res	# Res	Total	% Res	# Res	Total	% Res
2000	65	1,540	4.2%	167	925	18.1%	72	1,491	4.8%
2001	56	702	7.9%	164	516	31.7%	71	702	10.1%
2002	254	2,511	10.1%	323	1,487	21.7%	524	2,737	19.1%
2003	1,194	7,637	15.6%	2,020	6,307	32.0%	1,683	5,776	29.1%
2004	784	4,711	16.6%	1,341	3,554	37.7%	1,292	3,893	33.2%
2005	557	2,774	20.1%	589	1,567	37.6%	807	2,326	34.7%
2006	1,360	5,392	25.2%	1,112	2,589	42.9%	1,907	4,675	40.8%
2007	1,341	6,857	19.6%	1,742	4,959	35.1%	2,154	6,444	33.4%
2008	1,687	8,860	19.0%	2,209	5,693	38.8%	3,166	8,162	38.8%
2009	815	6,025	13.5%	1,314	4,309	30.5%	1,815	6,012	30.2%
2010	688	4,144	16.6%	1,293	3,146	41.1%	1,545	4,243	36.4%
2011	1,326	6,827	19.4%	3,007	6,650	45.2%	2,464	6,400	38.5%
2012	1,289	8,875	14.5%	3,105	8,240	37.7%	2,889	8,764	33.0%
2013	1,073	7,871	13.6%	2,638	7,273	36.3%	2,439	7,755	31.5%
2014	942	7,072	13.3%	2,525	6,746	37.4%	2,082	7,224	28.8%
2015	857	7,940	10.8%	2,729	7,902	34.5%	2,314	8,078	28.6%
2016	762	6,872	11.1%	2,365	6,675	35.4%	1,989	6,855	29.0%

Table 4.15 - *Pseudomonas aeruginosa* Trend

	Pseudomonas aeruginosa Trend								
	Ceftazidime			Piperacillin			Piperacillin / Tazobactam		
	# Res	Total	% Res	# Res	Total	% Res	# Res	Total	% Res
2000	260	1,852	14.1%	61	638	25.1%	135	1,491	9.0%
2001	158	1,350	11.7%	71	990	27.3%	64	1,025	6.2%
2002	535	2,643	20.2%	293	1,692	26.2%	291	2,823	10.3%
2003	1,700	8,152	20.8%	432	2,980	22.7%	685	6,302	10.9%
2004	780	3,402	22.9%	163	1,191	28.8%	400	3,314	12.1%
2005	679	2,748	24.7%	14	137	27.2%	430	3,717	11.6%
2006	1,262	5,201	24.3%	100	596	26.9%	692	6,001	11.5%
2007	2,088	7,597	27.5%	196	1,298	21.8%	711	7,151	9.9%
2008	2,105	9,580	22.0%	226	2,063	22.4%	1,011	10,658	9.5%
2009	1,188	6,882	17.3%	77	888	19.9%	950	7,322	13.0%
2010	840	4,999	16.8%	70	713	23.7%	623	5,267	11.8%
2011	1,248	7,423	16.8%	199	1,466	13.6%	775	6,203	12.5%
2012	1,194	8,647	13.8%	155	1,529	10.1%	650	7,126	9.1%
2013	1,013	6,584	15.4%	63	681	9.3%	837	7,825	10.7%
2014	821	6,029	13.6%	73	608	12.0%	589	6,504	9.1%
2015	953	6,371	15.0%	49	390	12.6%	662	7,528	8.8%
2016	902	6,814	13.2%	12	283	4.2%	570	7,697	7.4%

	Pseudomonas aeruginosa Trend (cont'd)								
	Imipenem			Gentamicin			Ciprofloxacin		
	# Res	Total	% Res	# Res	Total	% Res	# Res	Total	% Res
2000	224	1,917	11.7%	500	1,990	25.1%	527	1,749	30.1%
2001	155	1,350	11.5%	405	1,486	27.3%	525	1,344	39.1%
2002	318	2,748	11.6%	795	3,041	26.2%	729	2,096	34.8%
2003	1,131	7,015	16.1%	2,506	11,030	22.7%	3,460	8,912	38.8%
2004	528	3,781	14.0%	1,491	5,183	28.8%	1,657	3,988	41.6%
2005	660	3,826	17.2%	1,175	4,322	27.2%	968	2,104	46.0%
2006	911	5,419	16.8%	1,795	6,669	26.9%	1,111	3,000	37.0%
2007	1,361	8,075	16.9%	1,772	8,125	21.8%	1,959	5,780	33.9%
2008	1,620	9,916	16.3%	2,179	9,714	22.4%	2,161	6,488	33.3%
2009	1,013	6,613	15.3%	1,526	7,664	19.9%	1,572	5,725	27.5%
2010	808	4,882	16.5%	1,281	5,411	23.7%	1,315	4,106	32.0%
2011	1,344	7,758	17.3%	2,018	8,141	24.8%	2,481	7,878	31.5%
2012	1,012	6,712	15.1%	1,760	10,086	17.4%	2,515	6,883	36.5%
2013	1,040	5,486	19.0%	1,464	8,168	17.9%	2,214	7,889	28.1%
2014	839	4,322	19.4%	1,303	7,379	17.7%	1,854	6,712	27.6%
2015	651	3,908	16.7%	1,327	7,936	16.7%	2,183	7,630	28.6%
2016	307	2,241	13.7%	1,164	7,976	14.6%	1,849	7,787	23.7%