

The LOUISIANA ANTIBIOGRAM

Louisiana Antibiotic Resistance 2015

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This report covers bacteria 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 for lack of systematic reporting and collection of comprehensive data.

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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 progresses 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 based on 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 resistant isolates for each one of those micro-organisms. 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 became in 2005 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 (inpatient, ICU, or nursing home),
- Specimen source (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.

Most hospitals issue once a year, 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 resistant 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	CTRK	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. It is no longer useful once the specific bacteria have been identified and an antibiotic resistance established for the patient’s specific infection.

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, so that resistant strains that tend to be cultured more often, this artificially inflate the proportion of resistance.

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. Hospitals for which a specific antibiotic sensitivity is an outlier should investigate the reason for the discrepancy.

2.3-Analysis

The purpose of this analysis is to determine if there is a significant trend in the rates of antibiotic resistance for these microorganisms from 2000 to 2015, and to present the resistance data for the most recent period 2015.

2.3.1-Trend:

For micro-organisms of interest, a trend table is presented with the first column containing the number of resistant isolates, the second column with the number of isolates tested during the year and the third column with the percentage of resistant strains. Statistical tests presented are:

- The Cochran-Armitage test for linear trend (CoArm) with χ^2 -square, degrees of freedom=1, and p-value (Abramson, J.H. Winpepi (Pepi-for-Windows[®]): computer programs for epidemiologists. Epidemiologic Perspectives & Innovations 2004, 1: 6)

- The simple linear regression analysis equation with rate per 100 = $ax + b$, a representing the slope of the linear trend line.

2.3.2-Recent data on resistance:

Recent data on resistance show resistance for 2015 with 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).

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 an antibiogram proves methicillin sensitivity.

2015 Methicillin Sensitive *Staph aureus* MSSA

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Cefazolin	Cephalosporin	258	0%	0%	0%
Ceftriaxone	Cephalosporin	299	0%	0%	0%
Ciprofloxacin	Quinolone	788	27%	13%	38%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	205	0%	0%	0%
Clindamycin	Lincosamides	3888	19%	5%	67%
Daptomycin	Lipopeptide	712	0%	0%	0%
Doxycycline	Cyclines	70	11%	11%	11%
Erythromycin	Macrolides	3894	46%	29%	64%
Gentamicin	Aminoglycosides	952	2%	0%	10%
Levofloxacin	Quinolone	1225	22%	9%	37%
Linezolid	Oxazolidinone	777	0%	0%	0%
Moxifloxacin	Quinolone	2650	13%	0%	34%
Nitrofurantoin	Quinolone	369	0%	0%	0%
Oxacillin	PenicillinRb-lactamase	3894	0%	0%	2%
Penicillin G	Penicillin	2505	78%	70%	84%
Quinu/Dalfopristin	Streptogramin	215	0%	0%	0%
Rifampin	Rifamycin	952	0%	0%	1%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	299	0%	0%	0%
Tetracycline	Cyclines	3387	8%	1%	22%
Trimethoprim-sulfa	Sulfonamide	3894	1%	0%	3%
Vancomycin	Glycopolyptide	3894	0%	0%	0%

3.2-*Staphylococcus aureus* resistance to methicillin (oxacillin)

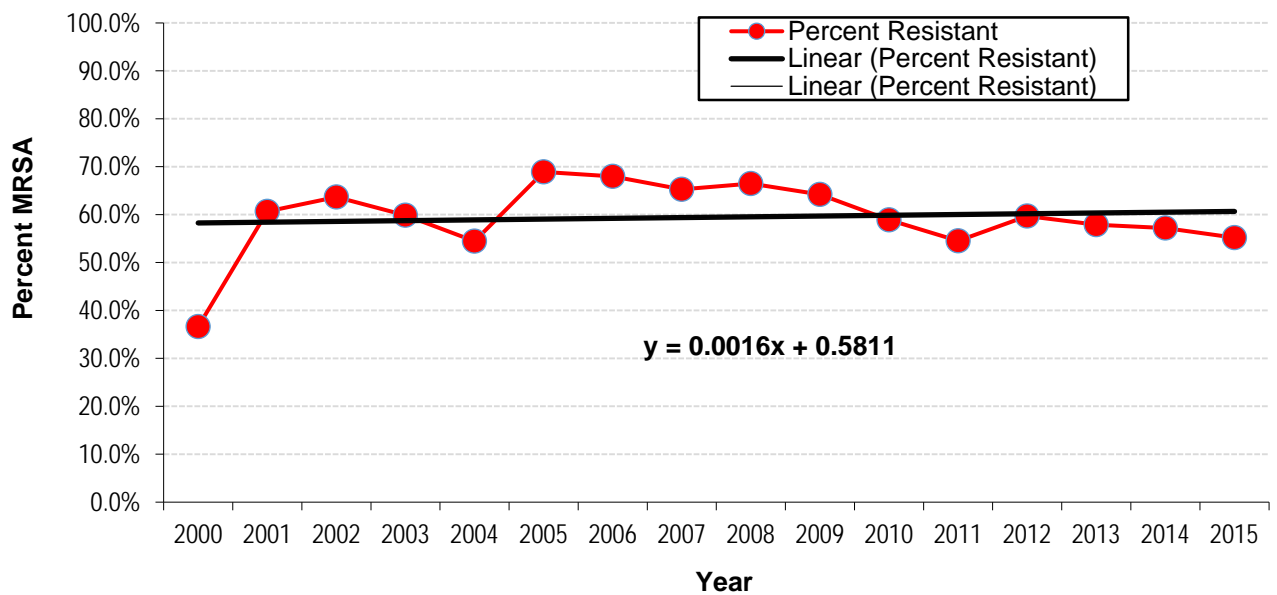
Methicillin Resistant *Staphylococcus aureus* (MRSA) is a growing problem both in the hospital and in the community. Resistance to methicillin is due to altered penicillin binding proteins.

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 Appendix [Table 4.1](#), the rates of methicillin-resistant *S. aureus* have increased from 2000 to 2005 from 38% to over 67%, and seemed to be stabilizing around 60% for a few years with a slight decline beginning in 2010. The graph below displays the trend seen in the table.



3.3-History of MRSA: Health care-associated (HA-MRSA) and community-associated MRSA (CA-MRSA).

MRSA infections that are reported in this report have not been differentiated into community-associated (CA) MRSA (or SCC mec Type IV or V PVL positive), and hospital-associated (HA) MRSA (or SCC mec Type II/III). Most Type IV MRSA 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 first appeared in hospitals, mostly as a nosocomial infection. MRSA was first recognized in 1961; one year after introduction of methicillin, resistant strains started to appear. The first documented MRSA outbreak in the U.S. was described at a Boston hospital in 1968. During the 1970s to the 1990s, most MRSA infections occurred in persons who had contact with hospitals or other health care facilities (HCF), hence the term healthcare-acquired or associated HA-MRSA. In the 1990s and 2000s, MRSA infections became more frequent among previously healthy individuals with no association with HCF. The acquisition of infections seems to have been from 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 (EMRSA). Most MRSA were simple colonizers. HA-MRSA were not more virulent than other SA: there was no difference in animal lethality, production of enzymes or production of toxins associated with invasiveness. However this strain was resistant to most antibiotics except vancomycin and a few newer antibiotics.

CA-MRSA started to spread in the late 1990s and 2000s and soon was taking over HA-MRSA. CA-MRSA is known to be more virulent, causing frequent skin and soft tissue infections as well as invasive infections (septicemia and pneumonias). Experiments showed that CA-MRSA produces toxins more frequently than its counterpart. CA-MRSA became the dominant MRSA clone in the USA.

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 be carrying 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; hence the importance of monitoring the sensitivity of MRSA.

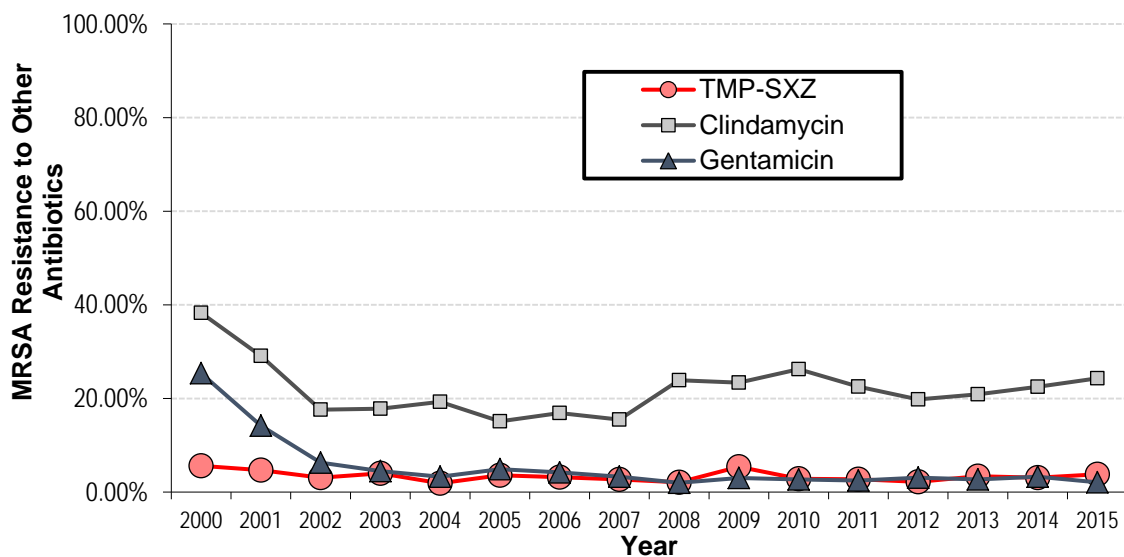
3.4-Other Antibiotics to which MRSA is resistant

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.



In Louisiana, TMP-SMX retains a relatively high sensitivity for some MRSA, illustrating the pattern seen in community-acquired organisms. The trend line is displayed in the graph above and in [Table 4.2](#). 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 (60% to 80%), and clindamycin (20% to 40%). They are less resistant to aminoglycosides (5% to 6% in recent years) and trimethoprim-sulfamethoxazole (2% to 5%).

2015 Methicillin *S. aureus* (MRSA)

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Azithromycin	Macrolides	140	89%	89%	89%
Chloramphenicol	Chloramphenicol	140	2%	2%	2%
Ciprofloxacin	Quinolone	1922	70%	60%	79%
Clindamycin	Lincosamides	5139	25%	10%	61%
Daptomycin	Lipopeptide	1433	1%	0%	3%
Doxycycline	Cyclines	284	4%	0%	6%
Erythromycin	Macrolides	4261	84%	76%	91%
Gentamicin	Aminoglycosides	2178	3%	0%	16%

Levofloxacin	Quinolone	2410	65%	40%	79%
Linezolid	Oxazolidinone	2168	0%	0%	3%
Moxifloxacin	Quinolone	2966	26%	5%	46%
Nitrofurantoin	Quinolone	860	0%	0%	1%
Quinu/Dalfopristin	Streptogramin	221	0%	0%	1%
Rifampin	Rifamycin	2590	1%	0%	9%
Tetracycline	Cyclines	5125	9%	0%	86%
Tigecycline	Glycylcycline	334	0%	0%	0%
Trimethoprim-sulfa	Sulfonamide	5269	4%	0%	16%
Vancomycin	Glycopolypeptide	5269	0%	0%	0%

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 requirements of two blood cultures to define a coagulase-negative staphylococcal blood stream 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 from 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 in vitro include vancomycin, minocycline, linezolid, the combination streptogramin, quinupristin/dalfopristin, and daptomycin

Antibiotics to which most coagulase-negative staphylococci are susceptible in vitro include vancomycin, minocycline, linezolid, the combination streptogramin, quinupristin/dalfopristin, and daptomycin.

2015 *Staphylococcus*-Coagulase negative

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Ampicillin	Penicillin Amino	159	89%	89%	89%
Azithromycin	Macrolides	159	61%	61%	61%
Cefazolin	Cephalosporin 1	308	55%	46%	69%
Cefepime	Cephalosporin 4	159	46%	46%	46%
Cefotaxime	Cephalosporin 3	159	47%	47%	47%
Ceftriaxone	Cephalosporin 3	159	47%	47%	47%
Cephalothin	Cephalosporin 1	159	46%	46%	46%
Chloramphenicol	Chloramphenicol	159	1%	1%	1%
Ciprofloxacin	Quinolone	2153	43%	0%	57%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	159	46%	46%	46%
Clindamycin	Lincosamides	5401	45%	0%	69%
Daptomycin	Lipopeptide	1479	1%	0%	2%
Doxycycline	Cyclines	11	18%	18%	18%
Erythromycin	Macrolides	5136	69%	49%	84%
Gentamicin	Aminoglycosides	3363	12%	0%	27
Imipenem	Carbapenem	159	46%	46%	46%
Levofloxacin	Quinolone	3612	49%	28%	72%
Linezolid	Oxazolidinone	2159	1%	0%	3%
Moxifloxacin	Quinolone	2458	33%	21%	42%
Nitrofurantoin	Quinolone	1525	1%	0%	4%
Norfloxacin	Quinolone	159	57%	57%	57%
Oxacillin	PenicillinRb-lactamase	5440	57%	36%	78%
Penicillin G	Penicillin	1883	87%	86%	88%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	159	1%	1%	1%
Quinu/Dalfopristin	Streptogramin	271	2%	1%	4%
Rifampin	Rifamycin	3374	3%	0%	9%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	159	46%	46%	46%
Tetracycline	Cyclines	5642	20%	9%	48%
Tigecycline	Glycylcycline	1266	1%	0%	2%
Trimethoprim-sulfa	Sulfonamide	4484	38%	2%	55%
Vancomycin	Glycopolypeptide	5517	0%	0%	2%

2015 Methicillin Resistant *S. epidermidis* Coagulase-negative (MRSE)

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Ampicillin	Penicillin Amino	159	89%	89%	89%
Azithromycin	Macrolides	159	61%	61%	61%
Cefazolin	Cephalosporin 1	308	55%	46%	69%
Cefepime	Cephalosporin 4	159	46%	46%	46%
Cefotaxime	Cephalosporin 3	159	47%	47%	47%
Ceftriaxone	Cephalosporin 3	159	47%	47%	47%
Cephalothin	Cephalosporin 1	159	46%	46%	46%
Chloramphenicol	Chloramphenicol	159	1%	1%	1%
Ciprofloxacin	Quinolone	2153	43%	0%	57%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	159	46%	46%	46%
Clindamycin	Lincosamides	5401	45%	0%	69%
Daptomycin	Lipopeptide	1479	1%	0%	2%
Doxycycline	Cyclines	11	18%	18%	18%

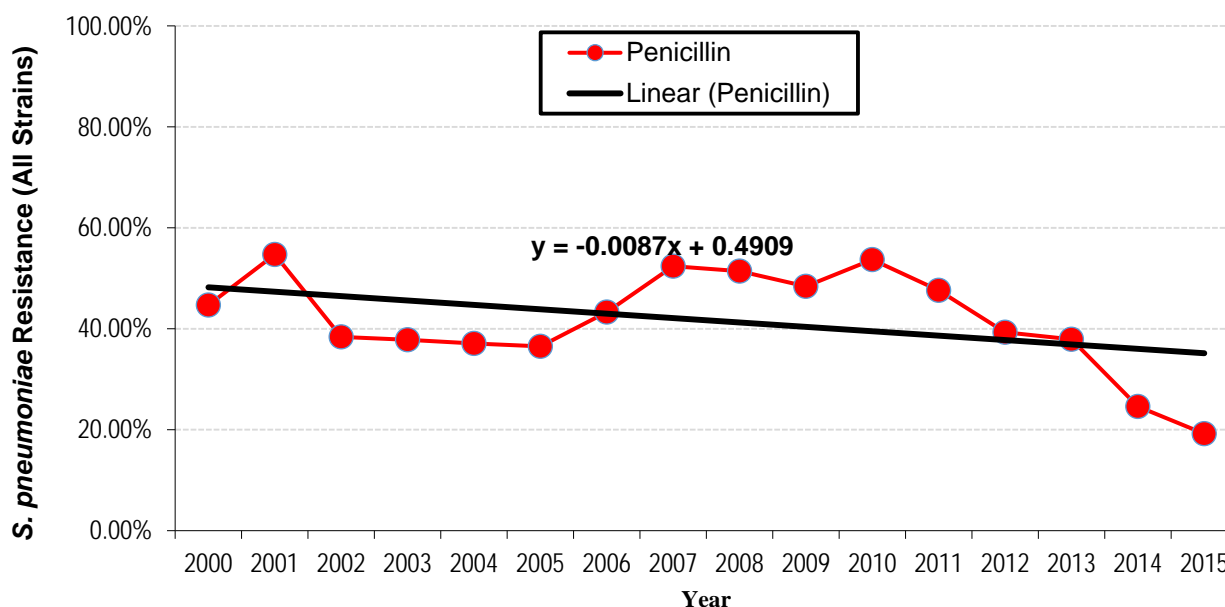
Erythromycin	Macrolides	5136	69%	49%	84%
Gentamicin	Aminoglycosides	3363	12%	0%	27%
Imipenem	Carbapenem	159	46%	46%	46%
Levofloxacin	Quinolone	3612	49%	28%	72%
Linezolid	Oxazolidinone	2159	1%	0%	3%
Moxifloxacin	Quinolone	2458	33%	21%	42%
Nitrofurantoin	Quinolone	1525	1%	0%	4%
Norfloxacin	Quinolone	159	57%	57%	57%
Oxacillin	PenicillinRb-lactamase	5440	57%	36%	78%
Penicillin G	Penicillin	1883	87%	86%	88%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	159	1%	1%	1%
Quinu/Dalfopristin	Streptogramin	271	2%	1%	4%
Rifampin	Rifamycin	3374	3%	0%	9%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	159	46%	46%	46%
Tetracycline	Cyclines	5642	20%	9%	48%
Tigecycline	Glycylcycline	1266	1%	0%	2%
Trimethoprim-sulfa	Sulfonamide	4484	38%	2%	55%
Vancomycin	Glycopolypeptide	5517	0%	0%	2%

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 10 leading causes of death in many countries, with a case fatality rate of 5% for pneumonia, 20% for bacteremia and 30% for meningitis.

Many antibiograms do not specify the criteria used to differentiate between intermediate and full resistance and do not specify what is included in their tables. So the percent resistance should be assumed to be a compilation of intermediate and fully resistant.

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 among countries, states, counties, and within populations in particular 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 for penicillin-binding proteins present in the bacterial cell wall. Penicillin resistance is thought to be due to horizontal transfer of genes of altered penicillin-binding proteins with 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 0.1–2.0 μ g/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 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 $\geq 2.0 \mu\text{g/ml}$). 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. The trend of penicillin resistance is displayed in the graph above and in [Table 4.3](#).

The susceptibility of *S. pneumoniae* to penicillin is currently defined by the NCCLS as follows: Susceptible isolates are inhibited by $0.06 \mu\text{g/mL}$ (i.e., minimal inhibitory concentration [MIC] $\leq 0.06 \mu\text{g/mL}$). Isolates with reduced susceptibility (also known as intermediate resistance) are inhibited by 0.1 to $1.0 \mu\text{g/mL}$, and resistant isolates are inhibited by $2.0 \mu\text{g/mL}$ 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 $1.0 \mu\text{g/mL}$) behaves as a susceptible organism when it causes pneumonia, but may not when it causes otitis, and does not when it causes meningitis. The recently revised definition of amoxicillin resistance (susceptible, MIC $\mu\text{g/mL}$; intermediately resistant, MIC 4 g/mL , resistant, MIC $>8 \text{ g/mL}$) 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 naso-pharynx of healthy carriers. They may cause pharyngitis, the most common clinical expression. The drug of choice in the treatment of streptococcal infection is penicillin, because of its efficacy in the prevention of rheumatic fever, safety, narrow spectrum, and low cost. Oral cephalosporins are 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.

As seen in [Table 4.4](#), there was no resistance to penicillin and a low resistance to erythromycin reported in 2008-2014.

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 infection in the newborn. It is a cause of urinary tract infections (UTI) and IV line infections, especially in diabetics or 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 (58%) and clindamycin (41%) restrict their use as empiric treatment for invasive infection or for intrapartum prophylaxis. Tetracycline resistance has increased to nearly 80%. This trend is shown in [Table 4.5](#).

2015 *Streptococcus group B, agalactiae*

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Ampicillin	Penicillin Amino	1301	0%	0%	1%
BetaLactamase	BetaLactamase	39	0%	0%	0%
Cefotaxime	Cephalosporin 3	107	0%	0%	0%
Ceftriaxone	Cephalosporin 3	331	2%	0%	6%
Chloramphenicol	Chloramphenicol	650	2%	2%	2%
Clindamycin	Lincosamides	1532	44%	35%	62%
Daptomycin	Lipopeptide	47	0%	0%	0%
Erythromycin	Macrolides	1286	57%	8%	90%
Levofloxacin	Quinolone	1621	2%	0%	12%
Linezolid	Oxazolidinone	295	0%	0%	0%
Oxacillin	PenicillinRb-lactamase	107	0%	0%	0%
Penicillin G	Penicillin	1685	0%	0%	0%
Quinu/Dalfopristin	Streptogramin	191	2%	2%	3%
Tetracycline	Cyclines	801	80%	52%	90%
Tigecycline	Glycylcycline	289	0%	0%	1%
Vancomycin	Glycopolyptide	2016	0%	0%	1%

3.9-Streptococcus viridans group

Streptococcus viridans is a group of streptococci which possesses no Lancefield antigens. They are most abundant in the mouth. *S. mutans*, is the etiologic agent of dental caries. They 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 1980s 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.

2015 *Enterococcus faecalis*

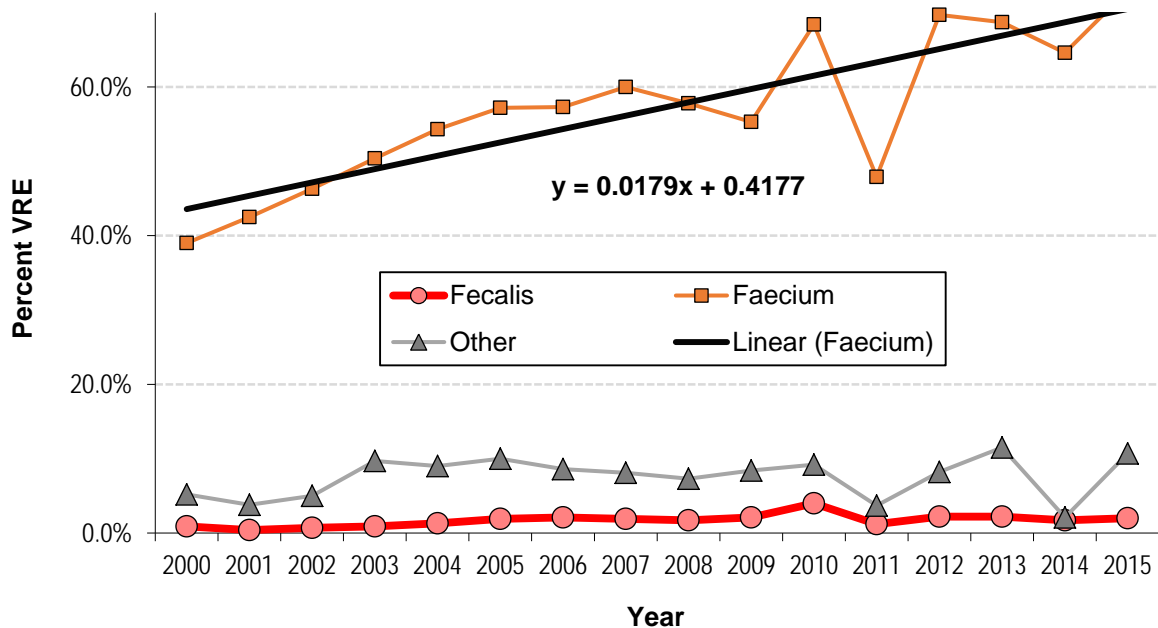
Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Ampicillin	Penicillin Amino	10904	3%	0%	42%
BetaLactamase	BetaLactamase	488	18%	1%	36%
Chloramphenicol	Chloramphenicol	186	10%	10%	10%
Ciprofloxacin	Quinolone	7480	31%	0%	60%
Clindamycin	Lincosamides	151	41%	41%	41%
Daptomycin	Lipopeptide	3261	0%	0%	1%
Doxycycline	Cyclines	334	70%	65%	76%
Erythromycin	Macrolides	1161	81%	69%	90%
Gentamicin	Aminoglycosides	1624	29%	15%	37%
Levofloxacin	Quinolone	5472	32%	0%	58%
Linezolid	Oxazolidinone	5575	2%	0%	13%
Nitrofurantoin	Quinolone	6831	2%	0%	18%
Norfloxacin	Quinolone	186	53%	53%	53%
Penicillin G	Penicillin	4102	3%	0%	18%

Rifampin	Rifamycin	778	28%	0%	41%
Streptomycin	Aminoglycosides	1028	21%	16%	27%
Tetracycline	Cyclines	8703	76%	57%	88%
Tigecycline	Glycylcycline	3317	0%	0%	1%
Vancomycin	Glycopolypeptide	11494	2%	0%	21%

2015 *Enterococcus faecium*

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Ampicillin	Penicillin Amino	1059	82%	63%	89%
BetaLactamase	BetaLactamase	93	0%	0%	0%
Ciprofloxacin	Quinolone	288	88%	86%	90%
Daptomycin	Lipopeptide	481	9%	0%	26%
Doxycycline	Cyclines	52	89%	87%	91%
Gentamicin	Aminoglycosides	211	5%	0%	9%
Levofloxacin	Quinolone	629	89%	84%	91%
Linezolid	Oxazolidinone	1003	4%	0%	19%
Nitrofurantoin	Quinolone	935	75%	50%	90%
Penicillin G	Penicillin	238	82%	69%	90%
Quinu/Dalfopristin	Streptogramin	168	15%	5%	36%
Rifampin	Rifamycin	27	89%	89%	89%
Streptomycin	Aminoglycosides	103	61%	58%	65%
Tetracycline	Cyclines	1173	81%	72%	89%
Tigecycline	Glycylcycline	552	2%	0%	10%
Vancomycin	Glycopolypeptide	1756	70%	36%	88%

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). The difference in the rate of resistance of *E. faecalis* and *E. faecium* to vancomycin is displayed in the graph below and in [Table 4.6](#). *E. faecalis* has a lower rate of resistance while *E. faecium* has a higher rate of resistance to vancomycin.



Overall rates of Vancomycin Resistant Enterococcus showed a significant increase over the years.

3.11-*Neisseria meningitidis*

Neisseria meningitidis is a colonizer of a few percent of the population and 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* 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 or cefotaxime) 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 6 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 (eg, ceftriaxone, ceftazidime, cefotaxime, ampicillin-sulbactam, fluoroquinolones,) to patients with uncomplicated meningitis for 7-14 days. The resistance to macrolides is high. Cefotaxime and ceftriaxone are the initial drugs of choice for suspected Hib meningitis.

Ceftiaxone and Fluoroquinolones have seen a slight increase in resistance to *H. influenzae*. The trend is shown in [Table 4.7](#).

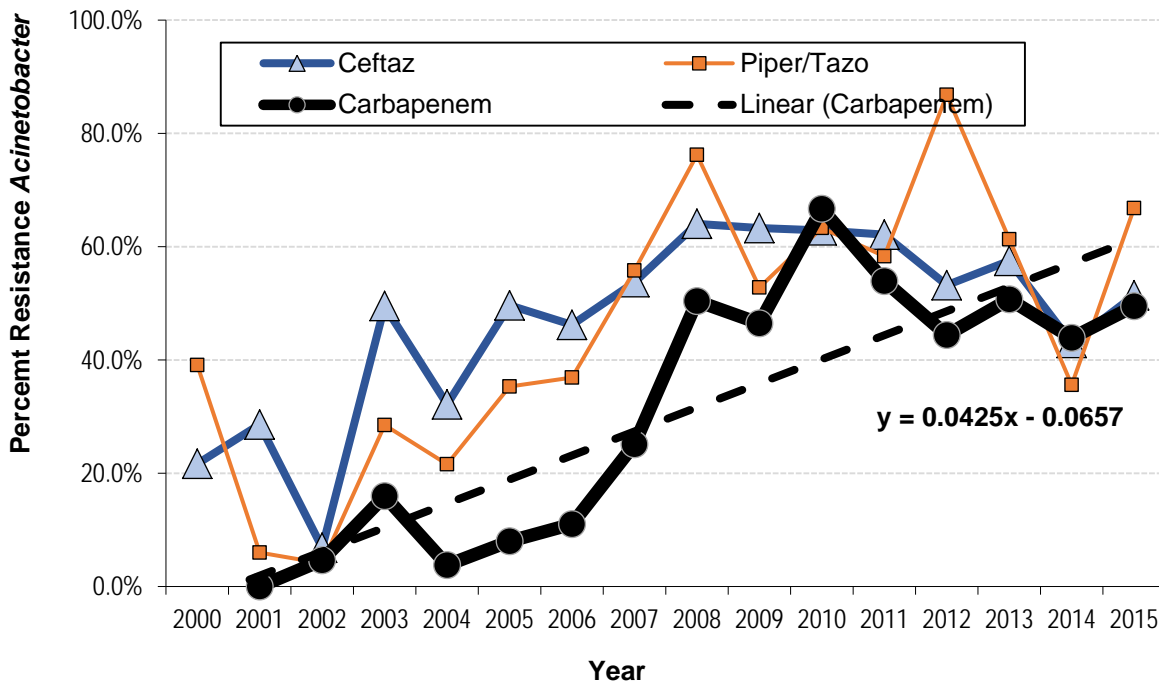
2015 *Haemophilus influenzae*

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Ampicillin	Penicillin Amino	141	36%	0%	81%
Cefepime	Cephalosporin 4	19	0%	0%	0%
Cefotaxime	Cephalosporin 3	37	0%	0%	0%
Ceftazidime	Cephalosporin 3	17	18%	18%	18%
Ceftriaxone	Cephalosporin 3	94	11%	0%	38%
Cefuroxime	Cephalosporin 2	37	3%	3%	3%
Chloramphenicol	Chloramphenicol	17	24%	24%	24%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	17	18%	18%	18%
Levofloxacin	Quinolone	40	10%	0%	19%
Rifampin	Rifamycin	37	0%	0%	0%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	57	8%	0%	18%
Tetracycline	Cyclines	40	0%	0%	0%
Trimethoprim-sulfa	Sulfonamide	94	26%	19%	35%

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. They are becoming a more common cause of nosocomial infections, usually ventilator-associated pneumonia, line sepsis or burn wound sepsis.

A baumannii is intrinsically multidrug resistant. Relatively few antibiotics are active against this organism. The increasing resistance of antibiotics to *A. baumannii* is shown in [Table 4.8](#) and in the graph below.



2015 Acinetobacter

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Cefepime	Cephalosporin 4	53	36%	36%	36%
Ciprofloxacin	Quinolone	53	34%	34%	34%
Gentamicin	Aminoglycosides	53	6%	6%	6%
Imipenem	Carbapenem	53	30%	30%	30%
Tobramycin	Aminoglycosides	53	6%	6%	6%
Trimethoprim-sulfa	Sulfonamide	53	23%	23%	23%

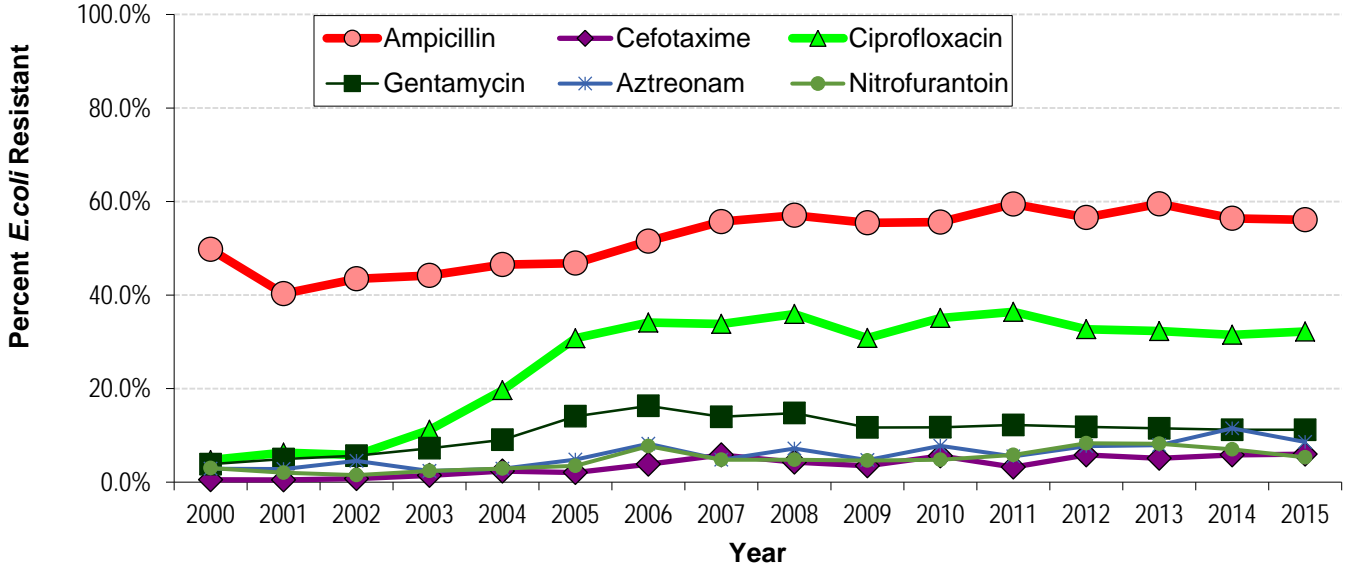
There is a huge increase in resistance to imipenem which went from 0% in 2001 to 67% in 2010.

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. A few isolates are not part of the human flora and when introduced in humans cause gastroenteritis (entero-toxigenic, entero-invasive and enterohemorrhagic *E. coli*).



2015 *E. coli*

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Amikacin	Aminoglycosides	51855	1%	0%	2%
Ampicillin	Penicillin Amino	50255	57%	39%	75%
Aztreonam	Monobactam	40440	6%	0%	22%
Carbapenem	Carbapenem	921	0%	0%	0%
Cefazolin	Cephalosporin 1	52682	14%	6%	38%
Cefepime	Cephalosporin 4	53791	5%	0%	19%
Cefotaxime	Cephalosporin 3	4616	6%	0%	11%
Cefotetan	Cephalosporin 2	423	1%	1%	2%
Cefoxitin	Cephalosporin 2	31352	8%	2%	20%
Cefpodoxime	Cephalosporin 3	363	11%	11%	11%
Ceftazidime	Cephalosporin 3	44747	6%	0%	17%
Ceftizoxime	Cephalosporin 3	363	8%	8%	8%
Ceftriaxone	Cephalosporin 3	58073	7%	0%	19%
Cefuroxime	Cephalosporin 2	10772	15%	2%	36%
Cephalothin	Cephalosporin 1	2732	58%	53%	63%
Ciprofloxacin	Quinolone	58189	33%	10%	63%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	34587	21%	10%	45%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	2400	15%	13%	18%
Doripenem	Carbapenem	363	0%	0%	0%
Doxycycline	Cyclines	447	19%	19%	19%
Ertapenem	Carbapenem	39562	0%	0%	3%
Gentamicin	Aminoglycosides	59457	11%	0%	26%
Imipenem	Carbapenem	25374	1%	0%	6%

Imipenem/Cilastatin	Carbapenem	3857	1%	0%	2%
Levofloxacin	Quinolone	32563	35%	10%	63%
Meropenem	Carbapenem	35795	0%	0%	3%
Moxifloxacin	Quinolone	2338	34%	22%	43%
Nalidixic	Quinolone	363	41%	41%	41%
Nitrofurantoin	Quinolone	43119	6%	0%	21%
Norfloxacin	Quinolone	363	34%	34%	34%
Piperacillin	Penicillin Ureido	2754	54%	50%	59%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	56103	6%	1%	32%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	48413	52%	36%	81%
Tetracycline	Cyclines	29885	29%	20%	44%
Ticarcillin	Penicillin Carboxy	363	53%	53%	53%
Tigecycline	Glycylcycline	6648	0%	0%	0%
Tobramycin	Aminoglycosides	56423	11%	5%	25%
Trimethoprim-sulfa	Sulfonamide	59452	36%	18%	59%

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* has become very resistant to ciprofloxacin in the early 2000s
- Resistance to aminoglycosides has also been increasing around 2004
- Although not as sharply, resistance to aztreonam is also increasing.

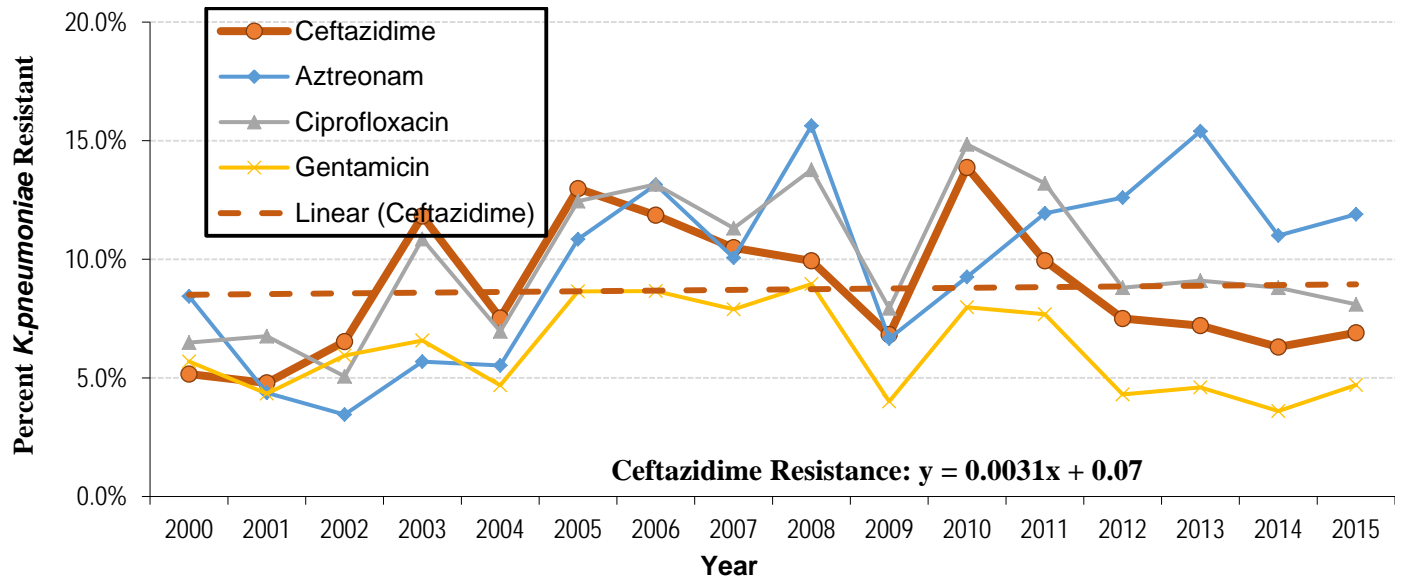
This trend is shown in [Table 4.9](#) and in the graph pictured above.

Antibiotics of choice 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. It is the most common pathogen in ICUs.

The use of Ampicillin as a course of treatment for *Klebsiella pneumoniae* ceased by 2012 because of its increasingly high resistance over the years. The other antibiotics displayed in the graph below and in [Table 4.10](#) have shown lower rates of resistance.



2015 *Klebsiella pneumoniae*

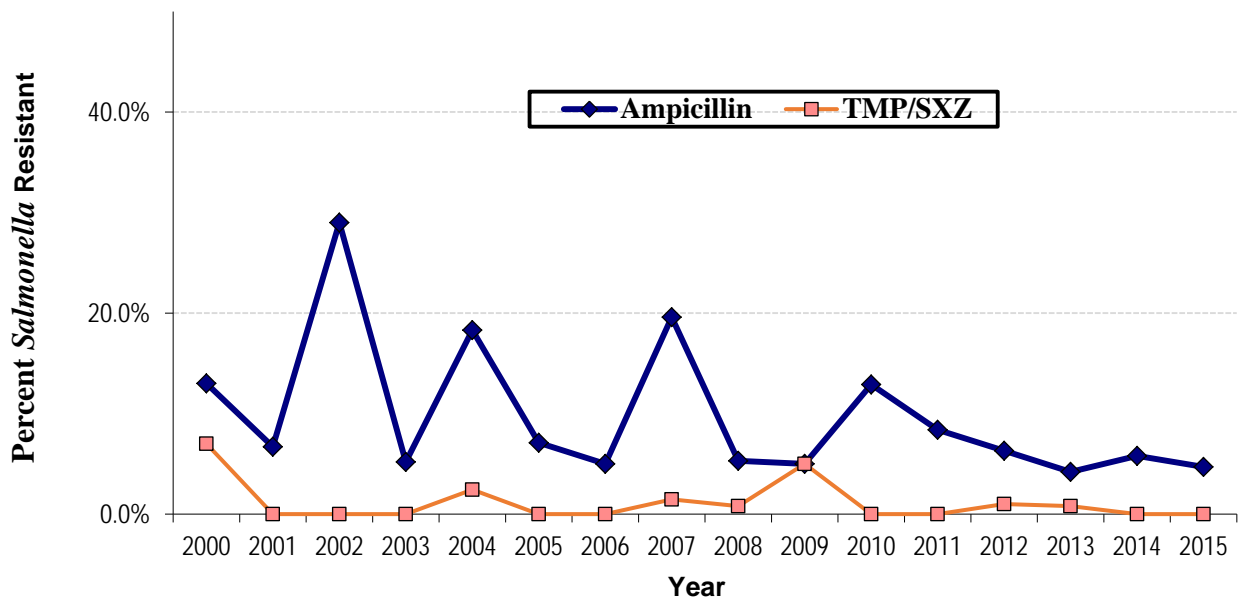
Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Amikacin	Aminoglycosides	11167	1%	0%	6%
Aztreonam	Monobactam	8633	9%	0%	80%
Carbapenem	Carbapenem	237	0%	0%	0%
Cefazolin	Cephalosporin 1	11306	14%	0%	80%
Cefepime	Cephalosporin 4	11169	10%	0%	79%
Cefotaxime	Cephalosporin 3	1081	5%	0%	12%
Cefotetan	Cephalosporin 2	123	2%	1%	4%
Cefoxitin	Cephalosporin 2	6202	8%	2%	25%
Cefpodoxime	Cephalosporin 3	96	8%	8%	8%
Ceftazidime	Cephalosporin 3	9563	7%	0%	25%
Ceftriaxone	Cephalosporin 3	12160	11%	0%	80%
Cefuroxime	Cephalosporin 2	2237	16%	1%	33%
Cephalothin	Cephalosporin 1	281	19%	15%	23%
Ciprofloxacin	Quinolone	12266	9%	0%	56%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	6659	6%	0%	25%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	578	8%	4%	15%
Doripenem	Carbapenem	98	0%	0%	0%
Doxycycline	Cyclines	52	19%	19%	19%
Ertapenem	Carbapenem	7883	1%	0%	4%
Gentamicin	Aminoglycosides	12524	5%	0%	22%
Imipenem	Carbapenem	5638	1%	0%	3%
Imipenem/Cilastatin	Carbapenem	1001	1%	0%	2%
Levofloxacin	Quinolone	7220	11%	0%	56%
Meropenem	Carbapenem	7434	0%	0%	2%
Moxifloxacin	Quinolone	628	5%	0%	11%
Nalidixic	Quinolone	98	14%	14%	14%
Nitrofurantoin	Quinolone	8632	60%	21%	81%
Norfloxacin	Quinolone	98	8%	8%	8%
Piperacillin	Penicillin Ureido	281	58%	58%	58%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	12083	7%	0%	38%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	9737	21%	4%	88%
Tetracycline	Cyclines	5639	15%	6%	28%

Tigecycline	Glycylcycline	1752	2%	0%	7%
Tobramycin	Aminoglycosides	11911	7%	0%	50%
Trimethoprim-sulfa	Sulfonamide	12266	12%	0%	56%

Antibiotics of choice 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. The human pathogens are 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.



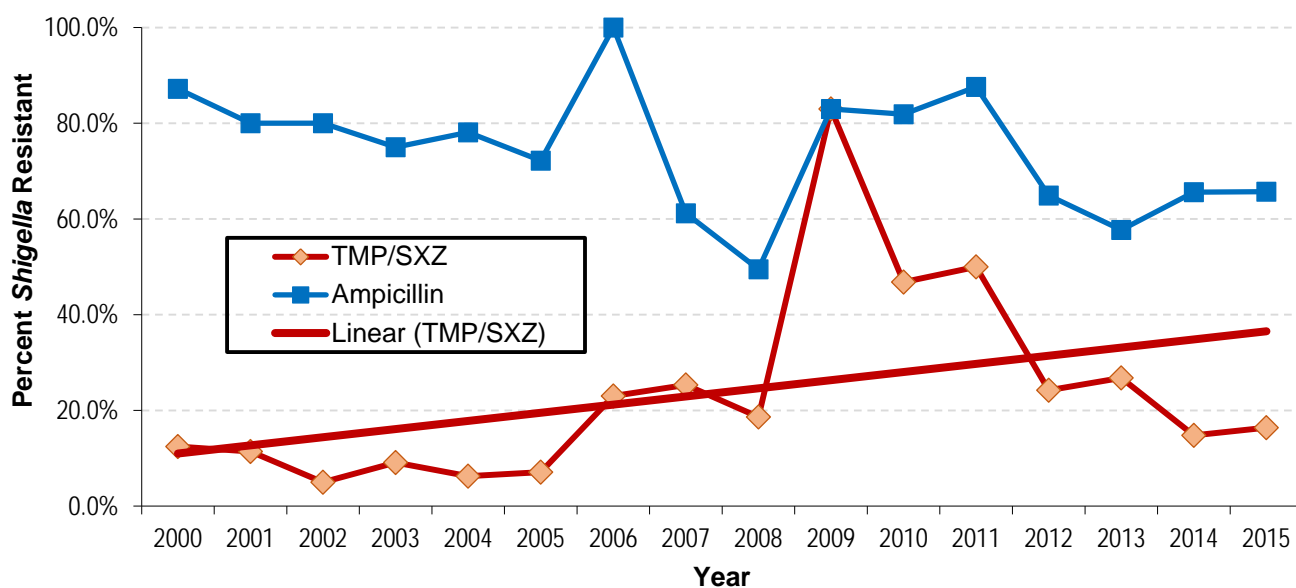
In most cases of simple enterocolitis due to *Salmonella*, no treatment is necessary. They do not appear to shorten the duration of symptoms and may prolong the carrier state. For severe enterocolitis and invasive disease (typhoid fever, paratyphoid fever) treatment is recommended. Antibiotics recommended include quinolone, macrolide, and third-generation cephalosporin pending sensitivities. The trend is shown in [Table 4.11](#) and in the graph above.

2015 Salmonella

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Ampicillin	Penicillin Amino	65	5%	5%	5%
Ceftriaxone	Cephalosporin 3	72	2%	2%	2%
Ciprofloxacin	Quinolone	75	0%	0%	0%
Trimethoprim-sulfa	Sulfonamide	76	0%	0%	0%

3.14.4-Shigella

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



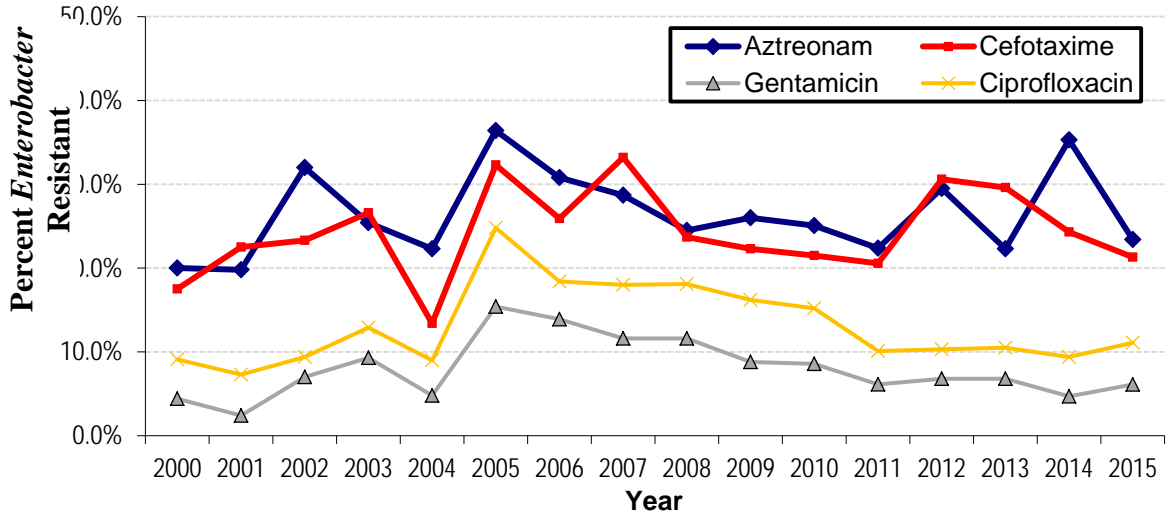
2015 Shigella

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Ampicillin	Penicillin Amino	67	66%	66%	66%
Ceftriaxone	Cephalosporin 3	67	0%	0%	0%
Ciprofloxacin	Quinolone	67	0%	0%	0%
Trimethoprim-sulfa	Sulfonamide	67	16%	16%	16%

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 [Table 4.12](#) and in the graph above, Ampicillin has a high rate of resistance along with TMP-SXZ, 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.



2015 *Enterobacter cloacae*

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Amikacin	Aminoglycosides	2068	0%	0%	3%
Ampicillin	Penicillin Amino	143	53%	19%	87%
Aztreonam	Monobactam	1329	22%	2%	50%
Cefazolin	Cephalosporin 1	127	48%	5%	90%
Cefepime	Cephalosporin 4	1965	6%	0%	24%
Cefotaxime	Cephalosporin 3	175	21%	12%	26%
Cefoxitin	Cephalosporin 2	59	20%	20%	20%
Cefpodoxime	Cephalosporin 3	19	90%	90%	90%
Ceftazidime	Cephalosporin 3	1815	19%	4%	39%
Ceftizoxime	Cephalosporin 3	25	16%	16%	16%
Ceftriaxone	Cephalosporin 3	2220	21%	4%	58%
Cefuroxime	Cephalosporin 2	252	61%	54%	74%
Ciprofloxacin	Quinolone	2253	10%	0%	36%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	84	90%	90%	90%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	73	29%	27%	31%
Doripenem	Carbapenem	16	0%	0%	0%
Ertapenem	Carbapenem	1473	3%	0%	14%
Gentamicin	Aminoglycosides	2298	4%	0%	14%
Imipenem	Carbapenem	1104	3%	0%	13%
Imipenem/Cilastatin	Carbapenem	159	4%	0%	6%
Levofloxacin	Quinolone	1062	8%	0%	18%
Meropenem	Carbapenem	1306	2%	0%	12%
Moxifloxacin	Quinolone	130	12%	5%	19%

Nalidixic	Quinolone	19	16%	16%	16%
Nitrofurantoin	Quinolone	1439	64%	25%	83%
Norfloxacin	Quinolone	19	5%	5%	5%
Piperacillin	Penicillin Ureido	19	42%	42%	42%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	2016	15%	0%	29%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	84	66%	63%	69%
Tetracycline	Cyclines	1018	13%	3%	27%
Ticarcillin	Penicillin Carboxy	19	47%	47%	47%
Tigecycline	Glycylcycline	174	0%	0%	0%
Tobramycin	Aminoglycosides	1963	5%	0%	15%
Trimethoprim-sulfa	Sulfonamide	2125	11%	0%	32%

These "ICU bugs" 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 [Table 4.13](#) and in the graph above.

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. They are implicated as serious causes of infections in humans. *Proteus* 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 (eg, *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.

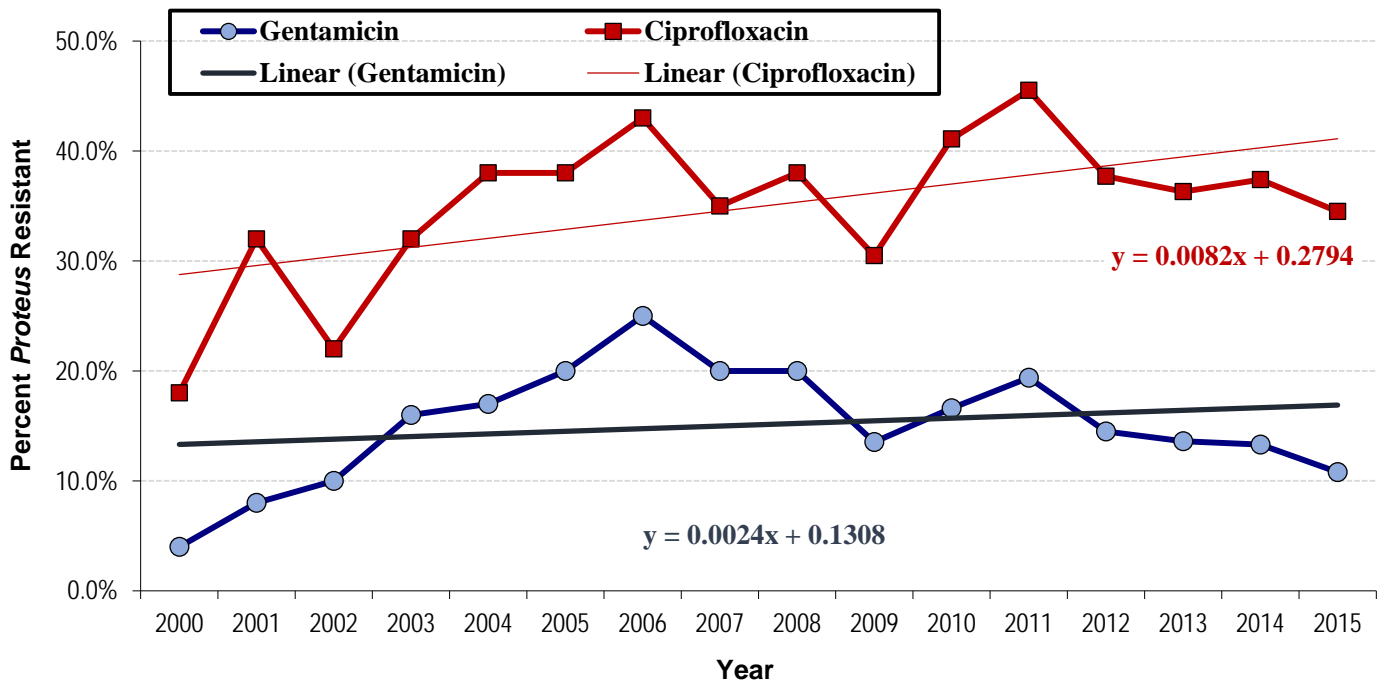
Proteus vulgaris is indole-positive and has more antibiotic resistance. *Proteus mirabilis*, which is indole-negative, is the most common species encountered in humans (90%).

P mirabilis remains susceptible to nearly many antimicrobials except cyclines. 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*. This trend is shown in [Table 4.14](#) and in the graph below.

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/SMZ, and quinolones have excellent activity (90%-100%).

2015 *Proteus mirabilis*

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Amikacin	Aminoglycosides	7147	1%	0%	7%
Ampicillin	Penicillin Amino	7089	25%	0%	49%
Aztreonam	Monobactam	5297	9%	0%	49%
Carbapenem	Carbapenem	185	0%	0%	0%
Cefazolin	Cephalosporin 1	7418	13%	0%	46%
Cefepime	Cephalosporin 4	7276	5%	0%	43%
Cefotetan	Cephalosporin 2	61	0%	0%	0%
Cefoxitin	Cephalosporin 2	3877	6%	0%	37%
Cefpodoxime	Cephalosporin 3	51	10%	10%	10%
Ceftazidime	Cephalosporin 3	6034	4%	0%	18%
Ceftizoxime	Cephalosporin 3	51	8%	8%	8%
Ceftriaxone	Cephalosporin 3	7895	6%	0%	43%
Cefuroxime	Cephalosporin 2	1503	7%	0%	17%
Cephalothin	Cephalosporin 1	244	26%	24%	28%
Ciprofloxacin	Quinolone	7902	35%	0%	86%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	4074	5%	0%	23%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	418	0%	0%	2%
Doxycycline	Cyclines	23	96%	96%	96%
Ertapenem	Carbapenem	5526	1%	0%	3%
Gentamicin	Aminoglycosides	7942	12%	0%	52%
Imipenem	Carbapenem	1673	8%	0%	52%
Imipenem/Cilastatin	Carbapenem	23	0%	0%	0%
Levofloxacin	Quinolone	4841	36%	0%	86%
Meropenem	Carbapenem	4656	1%	0%	8%
Moxifloxacin	Quinolone	461	44%	20%	86%
Nalidixic	Quinolone	51	43%	43%	43%
Norfloxacin	Quinolone	51	35%	35%	35%
Piperacillin	Penicillin Ureido	244	30%	22%	38%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	7826	1%	0%	8%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	6356	16%	0%	44%
Tetracycline	Cyclines	93	63%	0%	99%
Ticarcillin	Penicillin Carboxy	51	14%	14%	14%
Tigecycline	Glycylcycline	289	91%	84%	98%
Tobramycin	Aminoglycosides	7663	9%	0%	25%
Trimethoprim-sulfa	Sulfonamide	8078	30%	4%	91%



3.14.7-*Serratia marcescens*

Members of this genus produce characteristic red pigment, prodigiosin. *S. marcescens*, was formerly known as *Bacillus prodigiosus* because of its causing 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. marcescans 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.

2015 *Serratia marcescens*

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Amikacin	Aminoglycosides	701	1%	0%	8%
Ampicillin	Penicillin Amino	38	90%	90%	90%
Aztreonam	Monobactam	416	10%	0%	26%
Cefepime	Cephalosporin 4	742	1%	0%	9%
Cefotaxime	Cephalosporin 3	15	7%	7%	7%
Cefotetan	Cephalosporin 2	15	0%	0%	0%
Cefoxitin	Cephalosporin 2	77	76%	73%	79%
Ceftazidime	Cephalosporin 3	551	10%	0%	24%
Ceftizoxime	Cephalosporin 3	15	7%	7%	7%
Ceftriaxone	Cephalosporin 3	810	7%	0%	31%
Ciprofloxacin	Quinolone	749	11%	0%	24%
Doripenem	Carbapenem	15	0%	0%	0%
Ertapenem	Carbapenem	457	2%	0%	15%
Gentamicin	Aminoglycosides	810	3%	0%	10%
Imipenem	Carbapenem	261	3%	0%	16%
Imipenem/Cilastatin	Carbapenem	30	0%	0%	0%
Levofloxacin	Quinolone	460	8%	0%	18%
Meropenem	Carbapenem	450	1%	0%	7%
Moxifloxacin	Quinolone	53	12%	11%	13%
Nalidixic	Quinolone	15	13%	13%	13%
Norfloxacin	Quinolone	15	0%	0%	0%
Piperacillin	Penicillin Ureido	15	7%	7%	7%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	471	12%	0%	36%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	16	81%	81%	81%
Tetracycline	Cyclines	247	85%	71%	93%
Ticarcillin	Penicillin Carboxy	15	47%	47%	47%
Tigecycline	Glycylcycline	68	2%	0%	7%
Tobramycin	Aminoglycosides	620	17%	7%	28%
Trimethoprim-sulfa	Sulfonamide	681	6%	0%	31%

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*.

2015 *Citrobacter freundii*

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Amikacin	Aminoglycosides	775	0%	0%	0%
Ampicillin	Penicillin Amino	13	46%	46%	46%
Aztreonam	Monobactam	508	15%	0%	29%
Cefepime	Cephalosporin 4	731	1%	0%	13%
Cefotaxime	Cephalosporin 3	7	14%	14%	14%
Ceftazidime	Cephalosporin 3	621	16%	8%	29%
Ceftizoxime	Cephalosporin 3	7	14%	14%	14%
Ceftriaxone	Cephalosporin 3	718	14%	0%	33%
Cefuroxime	Cephalosporin 2	73	15%	15%	15%
Ciprofloxacin	Quinolone	789	11%	0%	39%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	20	81%	77%	86%
Doxycycline	Cyclines	13	8%	8%	8%
Ertapenem	Carbapenem	524	1%	0%	7%
Gentamicin	Aminoglycosides	810	8%	0%	33%
Imipenem	Carbapenem	329	2%	0%	8%
Imipenem/Cilastatin	Carbapenem	13	0%	0%	0%
Levofloxacin	Quinolone	425	12%	0%	39%
Meropenem	Carbapenem	551	1%	0%	7%
Nitrofurantoin	Quinolone	564	9%	0%	21%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	722	11%	0%	33%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	13	8%	8%	8%
Tetracycline	Cyclines	375	16%	0%	27%
Ticarcillin	Penicillin Carboxy	7	14%	14%	14%
Tigecycline	Glycylcycline	94	0%	0%	0%
Tobramycin	Aminoglycosides	677	6%	0%	14%
Trimethoprim-sulfa	Sulfonamide	709	18%	0%	39%

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 are reserved for resistant cases. With the widespread use of third-generation cephalosporins there has been an emergence of highly resistant *M. morganii*.

2015 *Morganella morganii*

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Amikacin	Aminoglycosides	538	0%	0%	2%
Ampicillin	Penicillin Amino	142	95%	90%	98%
Aztreonam	Monobactam	182	11%	0%	33%
Cefazolin	Cephalosporin 1	156	94%	90%	98%
Cefepime	Cephalosporin 4	527	2%	0%	10%
Cefotaxime	Cephalosporin 3	13	8%	8%	8%
Cefotetan	Cephalosporin 2	13	15%	15%	15%
Cefoxitin	Cephalosporin 2	132	66%	62%	73%
Ceftazidime	Cephalosporin 3	391	25%	18%	34%
Ceftizoxime	Cephalosporin 3	13	23%	23%	23%
Ceftriaxone	Cephalosporin 3	527	9%	0%	22%
Cefuroxime	Cephalosporin 2	57	92%	91%	92%
Ciprofloxacin	Quinolone	556	40%	20%	64%
Clavulanic-Amoxicillin	Penicillin&b-lactamInhib	98	93%	90%	96%
Ertapenem	Carbapenem	292	0%	0%	2%
Gentamicin	Aminoglycosides	587	22%	0%	55%
Imipenem	Carbapenem	141	27%	0%	82%
Levofloxacin	Quinolone	506	36%	15%	49%
Meropenem	Carbapenem	247	1%	0%	8%
Moxifloxacin	Quinolone	57	32%	31%	34%
Nalidixic	Quinolone	13	31%	31%	31%
Norfloxacin	Quinolone	13	23%	23%	23%
Piperacillin	Penicillin Ureido	13	39%	39%	39%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	540	4%	0%	21%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	116	95%	93%	98%
Tobramycin	Aminoglycosides	417	8%	0%	20%
Trimethoprim-sulfa	Sulfonamide	587	42%	25%	82%

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 due to *P. stuartii* infection 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.

2015 *Providencia stuartii*

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Amikacin	Aminoglycosides	191	0%	0%	0%
Aztreonam	Monobactam	21	0%	0%	0%
Cefepime	Cephalosporin 4	191	1%	0%	6%
Cefotaxime	Cephalosporin 3	8	0%	0%	0%
Cefotetan	Cephalosporin 2	8	0%	0%	0%
Cefoxitin	Cephalosporin 2	76	10%	0%	20%
Ceftazidime	Cephalosporin 3	108	12%	0%	18%
Ceftizoxime	Cephalosporin 3	8	0%	0%	0%
Ceftriaxone	Cephalosporin 3	191	10%	0%	50%
Cefuroxime	Cephalosporin 2	8	50%	50%	50%
Ciprofloxacin	Quinolone	171	84%	77%	91%
Ertapenem	Carbapenem	55	0%	0%	0%
Imipenem	Carbapenem	50	4%	4%	4%
Levofloxacin	Quinolone	170	78%	50%	91%
Meropenem	Carbapenem	39	0%	0%	0%
Piperacillin	Penicillin Ureido	8	13%	13%	13%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	191	2%	0%	10%
Ticarcillin	Penicillin Carboxy	8	13%	13%	13%
Trimethoprim-sulfa	Sulfonamide	73	20%	8%	44%

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, the versatility enables the organism to infect damaged tissues or people with reduced immunity.

It 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 to diffuse bronchopneumonia, skin lesions of ecthyma gangrenosum, urinary tract catheterization, necrotizing enterocolitis (NEC), hemorrhage and necrosis.

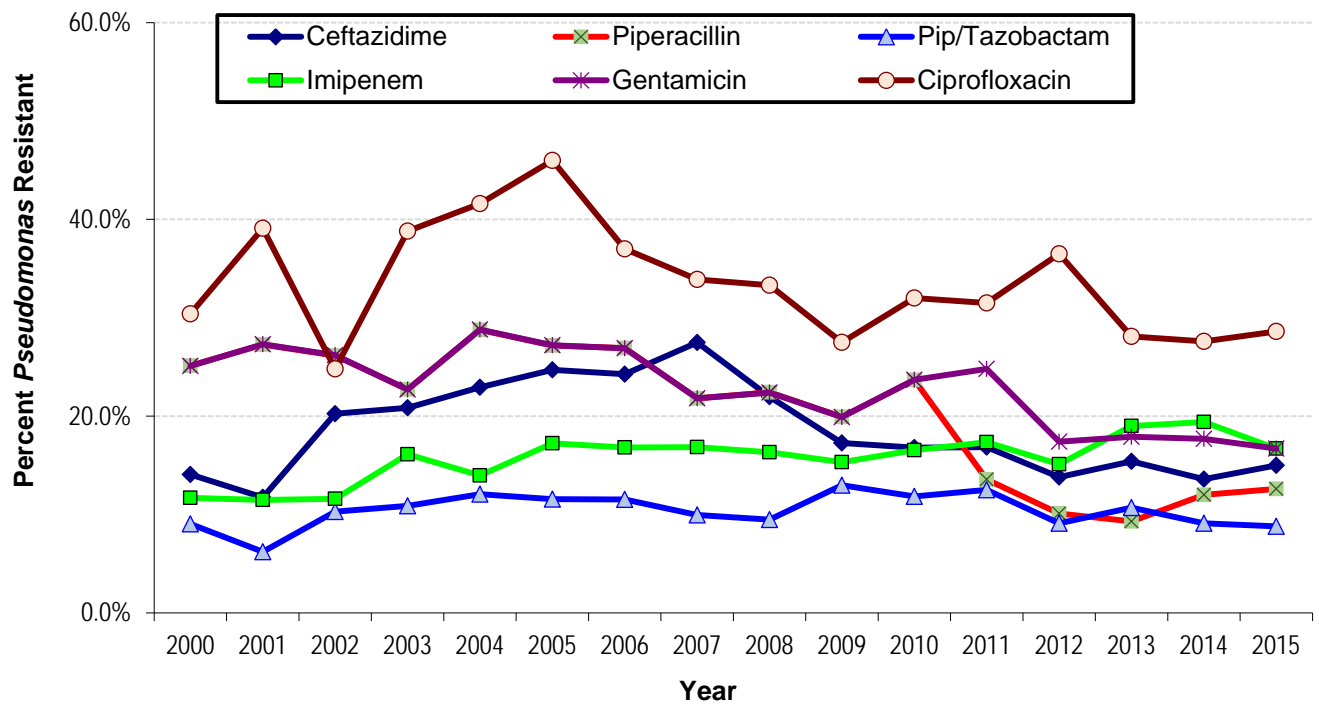
Those at greatest risk of infection are cystic fibrosis patients, neutropenic patients, burn victims and patients with wound infections.

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 trend is shown in [Table 4.15](#) and in the graph below.

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



2015 *Pseudomonas aeruginosa*

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Amikacin	Aminoglycosides	7235	5%	0%	24%
Ampicillin	Penicillin Amino	190	99%	99%	99%
Aztreonam	Monobactam	4336	29%	12%	44%
Cefazolin	Cephalosporin 1	190	99%	99%	99%
Cefepime	Cephalosporin 4	7519	16%	0%	39%
Cefotaxime	Cephalosporin 3	167	91%	83%	97%
Ceftazidime	Cephalosporin 3	6375	15%	0%	62%
Ceftizoxime	Cephalosporin 3	73	99%	99%	99%
Ceftriaxone	Cephalosporin 3	607	99%	98%	99%
Cefuroxime	Cephalosporin 2	94	67%	67%	67%
Ciprofloxacin	Quinolone	7634	28%	0%	67%
Clavulanic-Ticarcillin	Penicillin&b-lactamInhib	585	26%	11%	70%

Doripenem	Carbapenem	56	0%	0%	0%
Gentamicin	Aminoglycosides	7940	17%	0%	56%
Imipenem	Carbapenem	3908	18%	0%	41%
Imipenem/Cilastatin	Carbapenem	607	13%	0%	19%
Levofloxacin	Quinolone	5081	31%	0%	75%
Meropenem	Carbapenem	5033	10%	0%	26%
Moxifloxacin	Quinolone	72	29%	29%	29%
Nitrofurantoin	Quinolone	190	99%	99%	99%
Norfloxacin	Quinolone	72	18%	18%	18%
Piperacillin	Penicillin Ureido	390	13%	6%	22%
Piperacillin/Tazobactam	Penicillin&b-lactamInhib	7532	8%	0%	26%
Sulbactam-Ampicillin	Penicillin&b-lactamInhib	263	98%	98%	99%
Tetracycline	Cyclines	116	97%	97%	97%
Ticarcillin	Penicillin Carboxy	296	25%	11%	38%
Tobramycin	Aminoglycosides	7557	7%	0%	69%
Trimethoprim-sulfa	Sulfonamide	426	98%	98%	99%

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 a 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-SMZ). If TMP-SMZ cannot be used, the organism is usually sensitive to minocycline, respiratory quinolones, or colistin/polymixin B.

2015 *Stenotrophomonas maltophilia*

Antibiotic	Group	Sum of Isolates	Average Res	Low Res	High Res
Ceftazidime	Cephalosporin 3	382	60%	36%	77%
Levofloxacin	Quinolone	317	17%	0%	31%
Moxifloxacin	Quinolone	141	26%	26%	26%
Trimethoprim-sulfa	Sulfonamide	506	8%	0%	33%

4- Appendix

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

<i>S.aureus</i> /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%
CoArm	X2 122.95	df 1	p 0.00

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%
CoArm	X2 28.38	df 1	p 0.00	X2 274.09	df 1	p 0.00	X2 10.25	df 1	p 0.001

*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	3355	4.0%
2004	125	3,788	3.3%	40	2,438	1.6%	68	3608	1.9%
2005	239	4,830	4.9%	7	692	1.0%	198	5518	3.6%
2006	308	7,281	4.2%	50	3,067	1.6%	251	7753	3.2%
2007	146	4,358	3.3%	57	3,579	1.6%	135	5058	2.7%
2008	153	7,629	2.0%	97	6,634	1.5%	173	8154	2.1%
2009	116	3,843	3.0%	56	3,355	1.7%	189	3540	5.4%
2010	173	6,366	2.7%	117	5,031	2.3%	184	6461	2.8%
2011	102	4,149	2.5%	78	5,439	1.4%	164	5964	2.7%
2012	229	7,472	3.1%	103	6,376	1.6%	223	9969	2.2%
2013	88	3,318	2.7%	68	3,827	1.8%	214	6336	3.4%
2014	65	3,839	1.7%	44	3,875	1.1%	114	5768	2.0%
2015	45	2,178	2.1%	33	2,590	1.3%	200	5268	3.8%
CoArm	X2 338.24	df 1	p 0.00	X2 10.02	df 1	p 0.002	X2 9.44	df 1	p 0.002

*Always to be used in conjunction with another antibiotic

Table 4.3-*S.pneumoniae* / Penicillin Trend

<i>S.pneumo</i> /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%
CoArm	X2 86.16	df 1	p 0.000

Table 4.4-*Streptococci* Group A Trend

<i>Streptococci</i> Group A Trend						
	Penicillin			Erythromycin		
	Res	Exam	%Res	Res	Exam	%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		

Table 4.5-*Streptococci* Group B Trend

<i>Streptococci</i> Group B Trend												
	Penicillin			Erythromycin			Tetracycline			Clindamycin		
	Res	Exam	%Res	Res	Exam	%Res	Res	Exam	%Res	Res	Exam	%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%
CoArm	X2 23.68	df 1	p 0.00	X2 1436.78	df 1	p 0.00	X2 29.25	df 1	p 0.00	X2 1025.27	df 1	p 0.00

Table 4.6-*Enterococcus* Trend

	<i>Enterococcus</i> Trend								
	<i>E.fecalis</i> /Vancomycin			<i>E.faecium</i> /Vancomycin			<i>E. spp</i> /Vancomycin		
	Res	Exam	% Res	Res	Exam	% Res	Res	Exam	% 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%
CoArm	X2 125.19	df 1	p 0.00	X2 321.81	df 1	p 0.00	X2 21.23	df 1	p 0.00

Table 4.7-*Haemophilus influenzae* Trend

	<i>Haemophilus influenzae</i> Trend											
	Ceftriaxone			TMP-SXZ			Macrolides			Fquinolones		
	Res	Exam	Res %	Res	Exam	Res %	Res	Exam	Res %	Res	Exam	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%
CoArm	X2 5.69	df 1	p 0.017	X2 9.53	df 1	p 0.002				X2 11.92	df 1	p 0.001

Table 4.8-*Acinetobacter* Trend

	<i>Acinetobacter</i> Trend								
	Ceftazidime			Pieracillin/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%
CoArm	X2 24.83	df 1	p 0.000	X2 329.24	df 1	p 0.000	X2 214.52	df 1	p 0.000

Table 4.9-*E. coli* Trend

	<i>E. coli</i> Trend								
	Ampicillin			Cefotaxime			Ciprofloxacin		
	Res	Exam	%R	Res	Exam	%R	Res	Exam	%R
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%
CoArm	X2 2259.69	df 1	p 0.00	X2 696.27	df 1	p 0.00	X2 4031.97	df 1	p 0.00

	<i>E. coli</i> Trend (cont'd)								
	Gentamycin			Aztreonam			Nitrofurantoin		
	Res	Exam	%R	Res	Exam	%R	Res	Exam	%R
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%
CoArm	X2 152.49	df 1	p 0.00	X2 1546.13	df 1	p 0.00	X2 1092.41	df 1	p 0.00

Table 4.10-*Klebsiella pneumoniae* Trend

	<i>Klebsiella pneumoniae</i> Trend								
	Ampicillin			Ceftazidime			Aztreonam		
	Res	Exam	Res %	Res	Exam	Res %	Res	Exam	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	0	0	0.0%	713	8,491	8.4%	1,011	8,466	11.9%
2013	0	0	0.0%	542	7,546	7.2%	1,167	7,556	15.4%
2014	0	0	0.0%	562	8,986	6.3%	850	7,701	11.0%
2015	0	0	0.0%	663	9,562	6.9%	1,029	8,633	11.9%
CoArm				X2 110.58	df 1	p 0.000	X2 203.81	df 1	p 0.00

<i>Klebsiella pneumoniae</i> Trend (cont'd)									
	Ciprofloxacin			Gentamicin			Carbapenem (class)		
	Res	Exam	Res %	Res	Exam	Res %	Res	Exam	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%
CoArm	X2 22.64	df 1	p 0.00	X2 172.65	df 1	p 0.000	X2 21.71	df 1	p 0.000

Table 4.11-Salmonella spp.Trend

<i>Salmonella</i> spp.												
	Ampicillin			TMP-SXZ			Cefotaxime			Ciprofloxacin		
	Res	Exam	Res %	Res	Exam	Res %	Res	Exam	Res %	Res	Exam	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%
CoArm	X2 9.52	df 1	p 0.002									

Table 4.12-*Shigella* spp.Trend

	<i>Shigella</i> spp.											
	Ampicillin			TMP-SXZ			Cephalo 3			Ciprofloxacin		
	Res	Exam	Res %	Res	Exam	Res %	Res	Exam	Res %	Res	Exam	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	
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%
CoArm	X2 11.58	df 1	p 0.001	X2 20.63	df 1	p 0.00						

Table 4.13-*Enterobacter cloacae* Trend

	<i>Enterobacter cloacae</i> Trend								
	Aztreonam			Cefotaxime			Gentamicin		
	Res	Exam	Res %	Res	Exam	Res %	Res	Exam	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%
CoArm	X2 0.42	df 1	p 0.519	X2 3.17	df 1	p 0.075	X2 45.36	df 1	p 0.000

	Enterobacter cloacae Trend (cont'd)								
	Ciprofloxacin			TMP/SXZ			Carbapenems		
	Res	Exam	Res %	Res	Exam	Res %	Res	Exam	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%
CoArm	X2 23.41	df 1	p 0.000	X2 1.22	df 1	p 0.269	X2 48.18	df 1	p 0.000

Table 4.14-*Proteus mirabilis* Trend

	Proteus mirabilis Trend								
	Ampicillin			Clav-Ticarcillin			Ceftriaxone (3rd gen Ceph)		
	Res	Exam	%R	Res	Exam	%R	Res	Exam	%R
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%
CoArm	X2 0.36	df 1	p 0.546	X2 25.19	df 1	p 0.000	X2 861.25	df 1	p 0.000

	<i>Proteus mirabilis</i> Trend (cont'd)								
	Gentamicin			Ciprofloxacin			TMP/SXZ		
	Res	Exam	%R	Res	Exam	%R	Res	Exam	%R
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%
CoArm	X2 65.35	df 1	p 0.000	X2 90.66	df 1	p 0.000	X2 56.33	df 1	p 0.000

Table 4.15-*Pseudomonas Aeruginosa* Trend

	<i>Pseudomonas Aeruginosa</i> Trend								
	Ceftazidime			Piperacillin			Pipe/Tazobactam		
	Res	Exam	Res %	Res	Exam	Res %	Res	Exam	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%
CoArm	X2 374.28	df 1	p 0.00	X2 13.28	df 1	p 0.000	X2 9.41	df 1	p 0.002

	<i>Pseudomonas Aeruginosa</i> Trend (cont'd)								
	Imipenem			Gentamicin			Ciprofloxacin		
	Res	Exam	Res %	Res	Exam	Res %	Res	Exam	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%
CoArm	X2 77.70	df 1	p 0.000	X2 501.99	df 1	p 0.000	X2 415.83	df 1	p 0.000