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Antimicrobial Resistant Infections

Section 1: Introduction

Antimicrobial-resistant infections were a contributing factor to almost 5 million global deaths in 2019 (WHO, 2023). Antimicrobial-resistant infections occur when pathogens develop the ability to thrive despite using drugs that were once effective at killing them (CDC, 2024a). There are varying opinions regarding the cause of antimicrobial-resistant organisms. Still, this reality affects all countries in all regions and socioeconomic levels (WHO, 2023). Nurses are on the front line of this modern crisis and can positively impact health outcomes through increasing knowledge regarding antimicrobial-resistant infections. Nurses must be able to identify the most common types of antimicrobial infections, understand risk factors for these infections, recognize signs and symptoms, and be informed of diagnostic, treatment, and prevention methods regarding antimicrobial infections. Nurses can improve their practice by understanding how the COVID-19 pandemic affected antimicrobial resistance and the importance of their role in this rapidly changing area of healthcare.

Section 2: Antibiotics and the Rise of Resistance

The modern antibiotic era began in the early 20th century when Paul Ehrlich discovered antimicrobials capable of treating syphilis. This led to the development of sulfonamides, which are still used today. In 1928, Alexander Fleming discovered penicillin, and after a team at Oxford University developed a system for mass production and distribution in 1945, antibiotics changed the outlook of many infectious diseases and kicked off the golden age of antimicrobial drugs, including antibacterials, antivirals, antifungals, and antiparasitic drugs (Dutta, 2022).

The increasing use of antimicrobials has accelerated the naturally occurring genetic changes in pathogens. This has allowed many pathogens to develop antimicrobial traits, making treating diseases more difficult and increasing the risk of spreading disease among humans, animals, and plants (WHO, 2023).

Antimicrobial agents are meant to kill pathogens and improve the health of humans. However, the natural process of genetic mutation enables some pathogens to become resistant to treatment. The use of antimicrobial agents creates environmental selection pressure. This means that only the organisms able to resist treatment survive and reproduce amongst themselves, decreasing the likelihood of successful antimicrobial treatment (Salam et al., 2023).

Pathogens have specific defense strategies that allow them to become antimicrobial resistant. These strategies are called resistance mechanisms. One resistance mechanism that is used by pathogens is restricting access of the antibiotic to the pathogen cell. Bacteria cells may change the entryways or limit the number of entryways into the cell. For example, a bacterium with an outer membrane may begin to selectively block the antibiotic drug from entering. Another resistance mechanism may be to remove the antimicrobial agent. Bacteria or fungi may start producing efflux pumps that eliminate the drugs that enter the cells. Pathogens may destroy the antibiotic using enzymes. They may also change the target of the antimicrobial drug through gene mutation, causing the drug to be unable to bind to the pathogen. Microbes may bypass the effects of antibiotics by developing new cell processes within the cells that avoid using the antimicrobial's target, bypassing the mechanism of action used by the drug. Bacteria and fungi resistant to even one drug designed to treat them are dangerous (CDC, 2024a).

The unregulated use of antimicrobial medications in humans and agriculture has been a significant contributing factor in the increasing resistance of pathogens to currently available drugs (Salam et al., 2023). The Centers for Disease Control and Prevention (CDC) reports that more than 2.8 million antimicrobial-resistant infections occur annually in the United States, resulting in more than 35,000 deaths each year (Prevention, 2024a).

The complex problem of antimicrobial-resistant infections has widespread implications. Routine healthcare is at risk as the ability to treat infections

weakens. Antimicrobial pathogens also affect animals and plants, which can reduce the productivity of farms and create food shortages. This global problem worsens in some areas, as lack of access to clean water and insufficient hygiene practices increase infection risk (WHO, 2023). Antimicrobial-resistant infections increase the risk of severe and extended illnesses and death, contribute to severe medication side effects, cause more prolonged hospitalizations and more frequent medical appointments, and impact the cost of medical care (Cleveland Clinic, 2023a). As this global crisis evolves, nurses are on the frontline, caring for patients with antimicrobial-resistant infections and actively participating in the effort to reduce antimicrobial resistance among pathogens.

Section 2 Personal Reflection

Are you surprised by the evidence of a long history of antibiotic use before the modern age? Why do you think pathogens develop different mechanisms to resist the actions of antimicrobial agents? Why are antimicrobial-resistant drugs dangerous?

Section 2: Types of Antimicrobial Resistant Infections

Note: The COVID-19 pandemic contributed to a delay in data collection regarding antimicrobial-resistant infections. The most recent CDC report on antibiotic threats in the United States was published in 2019.

Antimicrobial resistance affects all types of microbes. The CDC categorizes treatment-resistant pathogens into three categories: urgent, serious, and concerning. The CDC also publishes a watch list of pathogens that are becoming increasingly resistant to treatments. In 2013, the CDC published its first Antibiotic Resistance Threat in the United States report. This report was updated and again published in 2019 (CDC, 2019). This information is essential for healthcare workers to understand which infections are becoming more resistant to treatment and

how patients may be affected. Since the 2013 report was published, deaths due to antimicrobial-resistant infections have decreased, but remain critically high. Early detection and prevention of these specific pathogens can improve outcomes (CDC, 2019).

Carbapenem-resistant Acinetobacter baumannii

Definition: Carbapenem-resistant *Acinetobacter baumannii* (commonly called CRAB) is a bacteria categorized as an urgent threat by the CDC, as it is known to be a highly antibiotic-resistant organism. There were approximately 8,500 patients hospitalized and 700 deaths due to this bacteria in 2017 in the United States (CDC, 2019). *Acinetobacter* is the cause of roughly 2% of nosocomial infections in the United States (Bartal et al., 2022). The associated healthcare cost due to carbapenem-resistant *Acinetobacter* in 2017 was $281 million. This bacteria can cause pneumonia, as well as wound, bloodstream, and urinary tract infections (CDC, 2019).

Mechanism of Resistance: These resistant species can easily share genetic code (CDC, 2019), and some can make carbapenemase, an enzyme that makes carbapenem antibiotics ineffective (MDH, 2024). This mechanism rapidly spreads resistance and neutralizes the drugs used to treat the infection. The CDC has found that some *Acinetobacter* are resistant to nearly all currently available antibiotics (CDC, 2019).

Risk Factors: Patients in intensive care units are at increased risk for carbapenem-resistant *Acinetobacter* infections (Prevention, 2019). Hospitalized patients, especially those hospitalized for extended periods, are particularly vulnerable to *Acinetobacter* infections. Patients who require invasive medical devices, such as endotracheal tubes, feeding tubes, and central venous catheters, and those with open surgical wounds, are at increased risk. The use of long-term antibiotics can

also increase the risk of CRAB infection (MDH, 2024). This bacteria can survive for long periods on surfaces, including shared medical equipment (CDC, 2019). Infections can also spread from patient to patient through contaminated hands of healthcare workers (MDH, 2024). Acinetobacter rarely causes infections within the healthy, non-hospitalized community (Bartal et al., 2022).

Signs and Symptoms: Colonization of *A. baumannii* may be asymptomatic and never lead to infection (MDH, 2024). Pneumonia is the typical manifestation of CRAB infection, with 55% of CRAB infections involving the respiratory system. It is most often associated with ventilator-associated pneumonia (VAP). Symptoms include those associated with pneumonia and sepsis, and meta-analysis suggests that carbapenem resistance is the major predictor of mortality among those with *Acinetobacter* infection (Bartal et al., 2022).

Diagnostics: It can be challenging to determine if a patient is experiencing a CRAB colonization or infection unless there are clear symptoms of illness, such as fever, increased white blood cell count, elevated inflammatory markers, and abnormal imaging. If CRAB is recovered from a non-sterile location, like a sputum culture, it may be a colonization. If it is isolated from a typically sterile location, such as spinal fluid, it is more likely due to infection. Clinical judgment is necessary when CRAB is found in non-sterile sources, especially for patients with chronic and complex medical needs (Bartal et al., 2022).

Treatment and Prevention: Treatment for CRAB is only indicated for an actual infection. When CRAB organisms are found in blood or cerebrospinal fluid, indwelling medical equipment, like catheters and stints, should be considered. If there are no indwelling devices, the infection should be treated. If indwelling devices are present but there are no symptoms of infection, the device should be replaced, if possible, but antibiotic treatment is unnecessary. If there are symptoms of infection in the presence of an indwelling device, removal and

treatment with antibiotics are recommended. High doses of ampicillin-sulbactam are typically used for initial therapy as it has the most substantial evidence for efficacy. Still, there is limited data on other treatment options that suggest one may be more successful in treating CRAB than others. Tigecycline may be used in high doses, and polymyxins are also a treatment option, but dosing is complex, and the side effects of these medications are significant. More research is needed to determine the clinical efficacy of newer treatments, like eravacycline and cefiderocol. Research suggests combination therapy may be most effective but should include high-dose ampicillin-sulbactam. It is recommended that infectious disease specialists manage CRAB infections. Future research focuses on improving diagnostic distinguishing characteristics between colonization and infections of CRAB (Bartal et al., 2022).

Rigorous infection control techniques must be used to prevent outbreaks of *Acinetobacter* infection. When an *Acinetobacter* infection is resistant to treatment with carbapenem, alternative treatment options are limited, as this bacterium is already resistant to many other antibiotics. The overall rate of carbapenem-resistant *Acinetobacter* infections has decreased, but its ability to produce resistant enzymes appears to be increasing. This could potentially cause these infection rates to increase (CDC, 2019).

Candida auris

Definition: This emerging multidrug-resistant yeast, a type of fungus, is categorized as an urgent threat. First noted in the United States in 2015, this species rapidly spreads in US healthcare facilities. In 2023, 4,514 clinical cases of *Candida auris* were reported. Some strains are resistant to all antifungal medications, and some commonly used healthcare disinfectants are less effective in killing these pathogens. In 2018, 323 cases were documented in the United

States, and 90% of these fungi were resistant to at least one antifungal drug. Thirty percent were resistant to at least two antifungals. Individuals can be carriers of *Candida auris* without having an actual infection, which increases the spread of the fungus (CDC, 2019). As a result of increased resistance, *Candida auris* is associated with high mortality rates of 30-60% (Borton, 2024).

Mechanism of Resistance: *Candida auris* can survive on environmental surfaces. It can live on plastic devices for up to 14 days and moist surfaces for up to 7 days. It can form a biofilm that protects itself from antifungal agents. This biofilm protects *Candida auris* from many disinfectant products. It also can pump out antifungal agents that could harm the cell using efflux pumps. This pathogen is capable of genetic mutations that allow for survival when the fungi are present in a hostile environment. Even after treatment, the patient may remain colonized with the fungus (Borton, 2024).

Risk Factors: Individuals with underlying conditions are at the highest risk for acquiring a *Candida auris* infection. Immunocompromised patients and those with diabetes, stroke, or the inability to perform activities of daily living are among those at higher risk for *Candida auris*. Outbreaks have been reported in high-acuity healthcare settings, such as intensive care units, acute inpatient rehabilitation settings, and post-acute skilled nursing facilities with mechanical ventilator care. Mechanical ventilation and invasive devices, such as intravascular devices, urinary catheters, and feeding tubes, increase the risk of infection. Patients undergoing treatment with broad-spectrum antibiotics, carbapenem antibiotics, or antifungals within the previous 90 days are also at increased risk. Increased hospital stays and acute or post-acute care hospitalizations within the previous six months also increase risk. Patients who undergo a surgical procedure are more likely to acquire an infection of *Candida auris*. Healthcare workers and healthy household members of patients with *Candida auris* are at low risk for infection but may be colonized with the fungus (Borton, 2024).

Signs and Symptoms: *Candida auris* can colonize the skin without causing symptoms (Borton, 2024). Symptoms of infection may be similar to bacterial infections, including fever and chills (CDC, 2024b). Patients with a *Candida auris* infection may have mild symptoms or be critically ill due to sepsis and organ failure. *Candida auris* is most likely to cause bloodstream and wound infections, skin abscesses, and external ear infections. Approximately half of cases in the United States are attributed to bloodstream infections (Borton, 2024).

Diagnostics: There are four different clades, or strains, of *Candida auris* that are categorized based on geographical location. They have different characteristics and carry differing antifungal resistance profiles. A potential fifth clade has been identified. The regions associated with individual clades are South Asia, East Asia, Africa, South America, and Iran. Since this microbe is capable of colonization, distinguishing between a colonized occurrence and infection can be difficult. Species-level identification can be helpful when there has been a facility or unit outbreak of *Candida auris*. Matrix-assisted laser desorption/ionization time-of-flight (MADLI-TOF) can be used to differentiate *Candida auris* from other types of *Candida* (Borton, 2024).

Treatment and Prevention: Treatment with antifungals is indicated for cases where clinical symptoms of infection are present but is not recommended for cases of colonization. Supportive care is also recommended for severe symptoms. It is recommended that providers consult with an infectious disease specialist when treating *Candida auris*. The first-line antifungal class used to treat this pathogen in the United States are Echinocandins, including anidulafungin, caspofungin, and micafungin. Another class is Triazoles, which includes fluconazole, voriconazole, and other second-generation drugs of the same class. Polyenes, like Amphotericin B, may also be used to treat a *Candida auris* infection. Echinocandin-resistant and pan-resistant strains are increasing in the United States (Borton, 2024). For strains

resistant to treatment, providers may prescribe a combination of antifungals or a newer pre-approved antifungal medication (CDC, 2024b).

Multiple strategies are being implemented to combat the spread of *Candida auris*. Tests capable of rapidly identifying the fungus have been created, which can help healthcare facilities reduce the spread (CDC, 2019). Communication between diagnostic labs and clinicians is necessary to alert the hands-on care providers of the presence of *Candida auris* so that infection prevention and control measures may be implemented (Borton, 2024). Another strategy to reduce spread is learning more about this species. At the time *Candida auris emerged*, four different strains were noted at the same time. Researchers are unsure why this occurred, but all four strains have been noted in the United States, likely due to international travel (CDC, 2019). High levels of topical chlorhexidine can reduce the number of pathogens on the skin, but this method has not been systematically studied to provide clinical evidence for treatment. Facilities implementing chlorhexidine bathing practices continue to observe outbreaks and transmission of *Candida auris*. Standard precautions, meticulous cleaning and disinfecting, monitoring, and antimicrobial stewardship can all contribute to preventing further spread and resistance of *Candida auris* (Borton, 2024).

Clostridioides difficile

Definition: *Clostridioides difficile*, commonly known as *C. diff*, is a gram-positive, spore-forming, anaerobic bacteria that affects approximately 500,000 people and results in an estimated 30,000 deaths in the United States annually (Markovska et al., 2023). Healthcare-associated cases of *C. diff* have declined in recent years, while community-acquired cases have increased (Feuerstadt et al., 2023). It is the most common healthcare-associated infection. The healthcare cost of this bacteria is estimated to be $1 billion (CDC, 2019). *C. diff* can produce exotoxins.

These toxins increase proinflammatory cytokine production, leading to leukocyte infiltration, inflammation, and fluid secretion. They also damage the gut epithelium. In severe cases of *C. diff* infection, these toxins cause a thick yellow fibrous exudate, called pseudomembranes, to form and are found in pseudomembranous colitis. Recently emerged strains, categorized as hypervirulent, cause severe *C. diff infections* even without antibiotic use. *C. diff* spores can survive for long periods and in hostile environments. They are highly resistant to drying, temperature, and many disinfectants (Markovska et al., 2023).

Mechanism of Resistance: *C. diff* has developed resistance to metronidazole and vancomycin through plasmids, small DNA molecules in bacteria that replicate independently of chromosomal DNA. Metronidazole typically damages the DNA and proteins of the bacteria through free radicals and depletes cellular thiols. Specific plasmids disrupt this process, some through altering protein binding. Vancomycin resistance, though uncommon, occurs through plasmid-directed effects that preserve the cell wall integrity of the bacteria when vancomycin typically inhibits cell wall synthesis. Fidaxomicin works by inhibiting transcription by binding to RNA polymerase, but mutations in the RNA of the bacteria impede the action of fidaxomicin (Dureja et al., 2022).

Risk Factors: Individuals taking antibiotics are at increased risk for *C. diff* infections, as these medications disrupt the microbiome of the digestive tract, providing an environment where *C. difficile* can thrive (CDC, 2019). It is estimated that 60% of individuals diagnosed with a *C. diff* diagnosis used antibiotics within four months prior to infection (Feuerstadt et al., 2023). Using clindamycin, fluoroquinolones, cephalosporins, and penicillins creates the highest risk for *C. diff* infection, but any antibiotic can disrupt the gut microbiome and predispose a patient to infection. The dose and duration of an antibiotic, as well as combined therapies, are also a factor in the risk for infection. Patients with inflammatory

bowel disease are also at increased risk due to the lack of diversity of essential bacteria in the gut (Markovska et al., 2023).

Hospitalization is a significant risk factor for infection. Prolonged or frequent hospitalization presents even more of a risk (Markovska et al., 2023). Fluoroquinolones, especially, have been associated with serious *C. difficile* infections (CDC, 2019). In long-term care facilities, it is most noted in recently hospitalized residents. However, the number of individuals affected in long-term care facilities has decreased for patients ages 65 and older. This decrease correlates with the reduction in use of fluoroquinolones in this population. Since 2015, cases of *C. difficile* in hospitalized patients have decreased, but community-associated cases have not (CDC, 2019). *C. difficile* infections are associated with high rates of recurrence and mortality (Markovska et al., 2023). Older adults are ten times more likely to experience a *C. diff* infection. The use of proton pump inhibitors, immunodeficiency, and chronic disease increases the risk of infection. Data supports that *C. diff* spores persist in the food chain, which affects all humans (Markovska et al., 2023).

Signs and Symptoms: *C. difficile* can be asymptomatic, cause mild self-limited diarrhea, or can cause life-threatening diarrhea related to pseudomembranous colitis (Markovska et al., 2023) and tends to create more severe symptoms in older adults (CDC, 2019). Small children are generally asymptomatic due to a lack of toxin receptors and protective antibodies in human breast milk. At any given time, it is estimated that 2.4-17.5% of healthy adults are asymptomatic carriers of *C. diff*, but this number is known to be higher in hospital environments (Markovska et al., 2023).

Watery diarrhea is the hallmark symptom of *C. diff* infections, but symptoms may also include mild abdominal pain and cramping, low-grade fever, and nausea. Fever is rare and occurs in less than 15% of mild cases of *C. diff* infection. Mild

cases can cause moderate leukocytosis. These mild cases typically resolve 5-10 days after the antibiotic therapy that disrupted the gut microbiome is finished (Markovska et al., 2023).

Severe cases of *C.* diff infection may cause patients to experience hemorrhagic colitis with bloody diarrhea episodes 10-15 times per day, severe abdominal pain, and fever. These patients often have elevated creatinine concentration and leukocytosis. Patients who undergo endoscopic examination may have changes in the colon epithelium, including pseudomembranes and ulcers. This can lead to hypotension, renal insufficiency, and multisystem organ failure. Extracolonic symptoms are rare but include arthritis and bacteremia (Markovska et al., 2023). The likelihood of complications associated with *C. diff* infections is high (Feuerstadt et al., 2023).

Diagnostics: *C. difficile* infection has specific clinical criteria for diagnosis. Three or more episodes of diarrhea within 24 hours or toxic megacolon with positive laboratory tests for certain toxins are used to differentiate colonization versus infection of *C. difficile (Markovska et al., 2023)*.

Recurrent *C. diff* infections are diagnosed when symptoms reappear within 2-8 weeks after completing treatment. The cause of recurrence is not well understood. Recurrence is common, with 25-30% of patients experiencing a recurrent infection within 30 days of completing treatment. Once a patient has had a recurrent infection of *C. diff*, they are 40-65% more likely to experience further recurrences (Markovska et al., 2023).

Treatment and Prevention: There is a multimodal approach to reducing *C. difficile*, including improving antibiotic stewardship, infection control practices, and cleaning and disinfecting healthcare facilities (CDC, 2019). Treating this pathogen is difficult because antibiotics are considered both a treatment and a risk factor for *C. diff* infection. It is recommended that antibiotic therapy only be used for

patients who are symptomatic from infection. Clinical treatment recommends vancomycin and metronidazole, though research has found vancomycin to be more successful. Fidaxomicin may also treat *C. diff* infections and does not seem to impact the gut microbiota. This medication is also known to prevent recurrent infections. It is recommended that primary *C. diff* infections be treated with vancomycin or fidaxomicin, and metronidazole may be used, but only in conjunction with vancomycin. If *C. diff* occurs during antibiotic treatment for another condition, the antibiotic should be stopped immediately, if possible, or changed if it cannot be stopped. Antibiotics associated with lower risk for *C. diff* infection include macrolides, aminoglycosides, sulfonamides, vancomycin, or tetracyclines (Markovska et al., 2023).

Researchers continue to seek antibiotic alternatives. One possibility may be Bezlotoxumab, a monoclonal antibody that binds to *C. difficile* toxin B. The FDA has approved it to prevent recurrent *C. diff* for patients at high risk for recurrence. This medication is costly, though, and is also associated with heart failure. Fecal microbiota transplantation, using FDA-approved Rebyota, has been successful as a method to manipulate the gut microbiota. Fecal samples from healthy donors are introduced to patients infected with *C. diff* through capsules taken orally or through colonoscopy. When used together, stopping the antibiotic therapy and fecal microbiota transplantation has a 75-90% success rate and is also associated with a high rate of preventing recurrence. Risks associated with fecal microbiota transplantation include the transmission of infectious pathogens from the donor to the recipient and the development of autoimmune disorders. Calcium aluminosilicate and human serum albumin have shown the ability to bind to *C. diff* toxins in laboratory settings. Research regarding aspirin therapy is promising, and human alpha-defensins and ambroxol have been shown to have antitoxic activity. Ridinilazole, which inhibits cell division and toxin production, and Ibezapolstat, a DBA polymerase IIIC inhibitor, are under investigation for clinical use in treating *C.*

diff. Purified *Firmicutes* spores to modulate gut microbiota have been shown to reduce the risk of *C. diff* infection with no significant side effects, but research continues. Live biotherapeutics and quality-controlled stool production are also being investigated. There have been clinical studies of probiotics, including *Saccharomyces boulardii* and *Lactobacillus*, as a prophylactic treatment for *C. diff*. Still, results are controversial, and continued research is needed, so probiotic use is not currently included in treatment guidelines (Markovska et al., 2023).

Carbapenem-resistant Enterobacteriaceae (CRE)

Definition: Carbapenem-resistant Enterobacteriaceae (CRE) is an urgent threat to healthcare in the United States. Approximately 13,100 hospitalized patients in 2017 experienced a CRE infection. That same year, it was estimated that 1,100 deaths were associated with CRE, and the financial impact was $130 million in attributable healthcare costs (CDC, 2019).

This bacterium is among the normal gut flora of humans and animals but can cause serious infections. It includes species like *Escherichia coli* and *Klebsiella pneumoniae* that have become resistant to carbapenem. This pathogen is spread through person-to-person contact due to poor hand hygiene, wounds, or feces. It can also be spread through contaminated medical equipment or devices. There have been a few reports of infection acquisition from animals. Animals can get CRE, but the risk of transmission from pet to owner is very low. This bacteria can colonize the skin without causing symptoms, which causes the resistant pathogens to be spread easily and unknowingly (CDC, 2024c).

Mechanism of Resistance: Approximately 30% of CRE can produce carbapenemase, an enzyme that destroys carbapenem antibiotics, rendering them ineffective (CDC, 2019). These pathogens may have multiple mechanisms for resisting the actions of carbapenem antibiotics using efflux pumps and blocking

binding sites, but carbapenemase production is the primary mechanism of resistance (Smith et al., 2024).

Risk Factors: Hospitalized patients are at higher risk for CRE infections (CDC, 2024c), especially those who require the long-term use of invasive devices, like urinary catheters (CDC, 2019), mechanical ventilators, or central venous devices. Patients who are on long-term courses of antibiotics (CDC, 2024c), have recently taken multiple antibiotic therapies (Smith et al., 2024), or are immunocompromised are also at increased risk for CRE infections. CRE does not typically lead to an infection in healthy adults (CDC, 2024c).

Signs and Symptoms: CRE symptoms include those associated with pneumonia, bloodstream infections, urinary tract infections, wound infections, and meningitis (CDC, 2024c).

Diagnostics: The CDC has successfully developed a method to detect carbapenemase-producing CRE. Identifying individuals with a CRE infection helps slow or stop the spread of this pathogen (CDC, 2019). Polymerase chain reaction (PCR) testing has become the most effective way to test for and track CRE infections (Smith et al., 2024).

Treatment and Prevention: The treatment plan for CRE depends on the infection site. For uncomplicated cystitis, trimethoprim/sulfamethoxazole, nitrofurantoin, aminoglycoside, or fluoroquinolone may be used to treat the CRE infection. Complicated cystitis cases may require sensitivity testing to select the most appropriate antibiotic. Various antibiotics may be used for CRE outside the urinary tract, but the type depends on the specific carbapenemase present (Smith et al., 2024).

As a result of available detection methods, when a case is identified, healthcare personnel can implement contact precautions to control the spread of the

infection. Increased ability to identify CRE infections may contribute to increased prevalence statistics as it is suggested that many cases were undiagnosed prior to increased ability to identify CRE infections (CDC, 2019). Infection control protocols are necessary to prevent an outbreak of CRE once a case has been identified (Smith et al., 2024). Strategies to reduce the spread of CRE have been effective for some types of bacteria, but the overall rate of CRE infections in hospitalized patients has increased since 2014 (CDC, 2019).

Drug-resistant Neisseria gonorrhoeae

Definition: *Neisseria gonorrhoeae* is the bacteria that causes gonorrhea, a common sexually transmitted infection that can cause pelvic inflammatory disease in women and eye problems that lead to blindness in infants born to an infected mother (Cleveland Clinic, 2024b). Antimicrobial resistance of this pathogen has increased rapidly in recent years and has limited options for treatment. In 2020, there were more than 82 million new cases of gonorrhea worldwide (WHO, 2024). In the United States, this urgent threat-level pathogen causes approximately 1.14 million new infections each year, and of those, 550,000 are resistant to antibiotics. The annual discounted direct medical costs due to drug-resistant *N. gonorrhoeae* are estimated to be $133.4 million (CDC, 2019).

Mechanism of Resistance: This pathogen resists the action of antibiotics through the overproduction of efflux pumps, enzymes that destroy antibiotics, and altering binding sites (Derbie, et al., 2020). Currently, gonorrhea is resistant to all but one class of antibiotics and spreads easily CDC, 2019). *Neisseria gonorrhoeae* antimicrobial resistance has occurred over the last 80 years and has resulted in treatment resistance to tetracyclines, macrolides, sulfonamides/trimethoprim, and quinolones. In addition, ciprofloxacin resistance is very high in some regions, with increasing resistance developing to ceftriaxone and cefixime. *Neisseria*

gonorrhoeae that are also resistant to treatment with penicillin are referred to as gonorrhea superbugs or super gonorrhea (WHO, 2024).

Risk Factors: Most people affected by gonorrhea are 15-49 years old, and the disease is more prevalent in Africa and the Western Pacific (WHO, 2024). Individuals who have a history of sexually transmitted infections are at increased risk for a *Neiserria gonorrhoeae* infection. Sexual activity with multiple partners and not using protective barriers, like condoms and dental dams, consistently and correctly also increase risk. Men who have sex with men are at higher risk for this infection compared to other populations (Cleveland Clinic, 2024b).

Signs and Symptoms: Gonorrhea infections, very often, are asymptomatic. If symptoms do occur, it may take up to two weeks for them to emerge. For women, they can cause unusual white or yellow vaginal discharge, pain during sexual intercourse, bleeding between periods, pain or burning with urination, and lower abdominal pain. Men may experience white, yellow, or green discharge from the penis, testicular pain and swelling, or dysuria. Both men and women may experience an itchy and sore throat and have trouble swallowing. They may also experience anal itching and discharge or pain with bowel movements (Cleveland Clinic, 2024b). In severe cases, *N. gonorrhoeae* can lead to cardiovascular and neurological problems (CDC, 2019).

Diagnostics: *N. gonorrhoeae* is diagnosed through sexual history, pelvic exam, cervical fluid sampling, urine sample testing, throat or rectal swabs, or penile fluid (Cleveland Clinic, 2024b).

Treatment and Prevention: This often asymptomatic infection may go untreated for long periods, increasing the spread before treatment can be prescribed. The CDC recommends a specific treatment regimen to reduce the acquired drug resistance of *N. gonorrhoeae* (CDC, 2019). Intramuscular ceftriaxone is typically used to treat gonorrhea. Alternatively, gentamicin and azithromycin may be used

in combination for those who cannot take ceftriaxone. As *N. gonorrhoeae* becomes increasingly resistant to treatment, it becomes more critical for patients to follow prescription instructions and prevent reinfection (Cleveland Clinic, 2024b).

Drug-resistant Campylobacter

Definition: *Campylobacter* is a gram-negative bacterium that commonly causes gastroenteritis and enterocolitis in humans (Portes et al., 2023). Drug-resistant *Campylobacter* is classified as a significant threat by the CDC, with estimates of 448,400 infections and 70 deaths linked to this pathogen annually. The drug-resistant type of *Campylobacter* is responsible for almost one-third of all *Campylobacter* cases and resists treatment with fluoroquinolones and macrolides. Drug-resistant *Campylobacter* prevalence has nearly doubled in the last two decades, and these pathogens continue to develop increased treatment resistance. The direct medical costs of *Campylobacter* infections are approximately $270 million each year. It is spread through raw or undercooked chicken, unpasteurized milk, contaminated food and water, and direct contact with animals (CDC, 2019).

Mechanism of Resistance: Antimicrobial resistance of *Campylobacter* species is partly due to the use of antibiotics in feed additives for animals. Spontaneous mutations and the acquisition of resistant characteristics contribute to the increasing antimicrobial resistance of *Campylobacter* species. Resistance mechanisms vary among species and to which antibiotic resistance has been developed. Fluoroquinolone resistance occurs through a single-point mutation in the DNA, allowing the pathogen to become resistant. Tetracycline resistance is due to a gene that encodes a protein that inhibits the effect of the antibiotic on conjugation. While not common, *Campylobacter* resists erythromycin by

mutations that affect macrolide binding, efflux pumps, and changes to the membrane's permeability. Antibiotic resistance varies by region (Portes et al., 2023).

Risk Factors: Travelers are at increased risk for *Campylobacter* infections, as the drug-resistant strains are most often found in low- and middle-income countries (CDC, 2019). This pathogen is especially prevalent in South America and is most often related to food originating from animals or contact with farm animals. Immunocompromised individuals are at increased risk for serious illness (Portes et al., 2023).

Signs and Symptoms: *Campylobacter* usually causes diarrhea, which is often bloody, fever, and abdominal cramps. It is associated with complications such as irritable bowel syndrome, Guillain-Barré syndrome, and reactive arthritis (CDC, 2019).

Diagnostics: *Campylobacter* infections are diagnosed through microscopic examination of a stool sample. Rarely, a blood culture may be used to check for bacteremia (Cleveland Clinic, 2024a).

Treatment and Prevention: Most cases of *Campylobacter* infection self-resolve in 5-7 days, but some individuals may require antibiotic treatment (Cleveland Clinic, 2024a). For many years, fluoroquinolones were used to treat campylobacteriosis, but increased selective pressure has reduced the efficacy of this class of antibiotics. Currently, macrolides are being used for treatment, though resistance to erythromycin has risen in recent years. As a result, macrolides have been banned from food additives in many countries to reduce the resistance to erythromycin (Portes et al., 2023). Treatment also includes hydration and electrolyte supplementation. Prevention methods include following food safety guidelines to minimize the risk of foodborne pathogens, thoroughly cooking meat

and seafood, consuming only pasteurized dairy, drinking water from safe sources, and having good hand hygiene (Cleveland Clinic, 2024a).

Drug-resistant Candida

Definition: There are dozens of *Candida* species, a fungi group (CDC, 2019). *Candida auris*, previously mentioned, is especially resistant to antimicrobials, but other *Candida* species also show increased resistance to antimicrobial treatment (CDC, 2024l). These pathogens cause a wide variety of infections, including mild oral infections (thrush) and vaginal yeast infections to severe invasive infections. Many species are resistant to the drugs used to treat them. *Candida* is responsible for more than 3.6 million healthcare visits in the US each year, and the associated direct medical costs exceed $3 billion. The drug-resistant form of *Candida* is responsible for approximately 34,800 infections in hospitalized patients and 1,700 deaths in 2017. Most people are familiar with *Candida* albicans, which is typically easily treated. Other species, like *Candida* glabrata, are often resistant to available treatments and can have fatal consequences (CDC, 2019).

Mechanism of Resistance: Approximately 7% of *Candida*-infected blood samples resist fluconazole (CDC, 2024l). The mechanism of resistance depends on the type of *Candida*. *Candida* fungi can resist azole antifungals through increased expression of membrane transporters, efflux pumps, toxic sterol formation, changes in importing cholesterol and serum from the blood, and reproducible gene mutations (Bhattacharya et al., 2020).

Risk Factors: Immunocompromised patients, pregnant women, newborns, and hospitalized patients are at increased risk for drug-resistant *Candida* infections. Individuals who have recently taken antibiotics, steroids, or chemotherapy are also at increased risk. Patients diagnosed with HIV/AIDS, cancer, and diabetes are more likely to experience a *Candida* infection. Women who use hormonal birth

control are at increased risk for vaginal candidiasis. Individuals who use inhaled corticosteroids, medications that cause dry mouth, smoke, or who have dentures are at increased risk for candidiasis of the mouth or throat. Specific risk factors for invasive candidiasis include preterm birth, kidney failure, long periods in the ICU, central venous catheters, total parenteral nutrition, organ transplant, recent surgery, and hemodialysis (CDC, 2024l).

Signs and Symptoms: Symptoms include those associated with candidiasis, including vaginal yeast infections, thrush, candidiasis of the esophagus, and invasive candidiasis, which affects the internal organs and causes sepsis (CDC, 2024l).

Diagnostics: Invasive candidiasis is diagnosed through the patient's medical history, symptoms, physical exam, and laboratory tests, including blood culture (CDC, 2024l).

Treatment and Prevention: Currently, only three antifungal drugs are available to treat severe *Candida Infections*. They include azoles, echinocandins, and amphotericin B. Limited ability to test for drug resistance in *Candida* makes treatment guidance and tracking resistance difficult (CDC, 2019). Infections that are resistant to fluconazole and echinocandin are treated with amphotericin B, but this medication can be toxic for seriously ill patients (CDC, 2024l).

Extended-spectrum beta-lactamase (ESBL)-producing Enterobacteriaceae

Definition: ESBL-producing Enterobacteriaceae describe a family of different types of bacteria capable of producing enzymes that destroy commonly used antibiotics. Enterobacteriaceae species include *Escherichia coli (E. coli)*, which spreads rapidly in community and healthcare settings, causing severe infections that were previously common and easily treated (CDC, 2019). It also includes *Klebsiella*

pneumoniae (CDC, 2024d). Unlike most antimicrobial-resistant infections, almost half of ESBL-producing Enterobacteriaceae occur in individuals who have not experienced a recent hospitalization or invasive procedure. In 2017, it was estimated that there were 197,400 cases in the United States and 9,100 resulting deaths. The attributable healthcare costs for these infections in 2017 totaled approximately $1.2 billion. Cases of ESBL-producing Enterobacteriaceae continue to increase, and researchers are working to identify a cause (CDC, 2019).

Mechanism of Resistance: The enzyme responsible for drug resistance, extended-spectrum beta-lactamase, is spread through DNA. This enzyme can break down certain antibiotics. These bacteria are currently resistant to penicillins and cephalosporins (Prevention, 2024d). Increased reliance on carbapenem antibiotics increases the possibility for ESBL-Enterobacteriaceae to develop even more resistance (CDC, 2019).

Risk Factors: Patients in hospitals and nursing homes are at increased risk for ESBL-producing Enterobacterales infections, but they can also be found in healthy people. Individuals who travel internationally are at increased risk, as these bacteria can be spread through contaminated food and water (CDC, 2024d)

Signs and Symptoms: Symptoms of ESBL-producing Enterobacterales include those consistent with urinary tract and bloodstream infections. These bacteria commonly colonize within a host and remain asymptomatic (CDC, 2024d).

Diagnostics: Diagnosis is made through patient history information, symptom evaluation, and urine and blood culture testing (CDC, 2024d)

Treatment and Prevention: An infection may require hospitalization and IV carbapenem or other complex treatments to kill the bacteria (CDC, 2019). Prevention includes proper hand hygiene and food and water safety practices (CDC, 2024d).

Vancomycin-resistant Enterococci (VRE)

Definition: Vancomycin-resistant *Enterococci* (VRE) are anaerobic gram-positive cocci that live in the gastrointestinal tract (Levitus et al., 2023). The CDC categorizes VRE as a serious threat, with 54,500 estimated cases in 2017, which includes 5,400 deaths and approximately $539 million in associated healthcare costs. This type of bacteria causes serious bloodstream, surgical site, and urinary tract infections in healthcare settings. Approximately 30% of all healthcare-associated enterococcal infections are resistant to vancomycin, which reduces treatment options (CDC, 2019).

Mechanism of Resistance: *Enterococci* are resistant to several antibiotics using a variety of resistance mechanisms. Vancomycin resistance of these pathogens is caused by changes in the bacterial cell wall that affect the ability of vancomycin to bind to the bacteria. These changes are accomplished through changes in the bacteria's DNA. VRE has developed resistance to most aminoglycosides through the development of an inactivating enzyme (Levitus et al., 2023).

Risk Factors: Almost all VRE infections occur in patients with recent healthcare exposures. The need for complex healthcare and weakened immune systems are associated with VRE infections. Patients who are admitted to a long-term care facility or intensive care unit, undergoing an organ transplant, or receiving cancer treatment are at increased risk for acquiring a VRE infection (CDC, 2019). Patients who have previously taken antibiotics, especially vancomycin, have had surgery, or have an indwelling medical device are at increased risk for a VRE infection (CDC, 2024o).

Signs and Symptoms: *Enterococcus* infection can cause many illnesses. The most common is a urinary tract infection, but many cases are due to colonization rather than an actual infection. It can also commonly cause bacteremia, endocarditis, and intra-abdominal and pelvic infections (Levitus et al., 2023).

Diagnostics: A diagnosis of VRE begins with the provider establishing the presence of an infectious disease. Potential sources of infection must be evaluated, and then an appropriate culture sample must be obtained before beginning empiric antibiotic therapy. Some sensitivity testing may not be standard in the laboratory but can be added as needed (Levitus et al., 2023).

Treatment and Prevention: Antibiotic treatment for VRE depends on the type of infection and the susceptibility determined through laboratory testing. It may be challenging to assess whether results are due to colonization in polymicrobial infections. Therefore, treatment should be based on the failed use of other antibiotics. *E. faecalis*, the most common cause of enterococcal infections, is usually susceptible to beta-lactams and aminoglycosides (Levitus et al., 2023). VRE is becoming increasingly resistant to more antibiotics, which could cause the drugs currently used to treat VRE to become less effective. Infection control practices have effectively reduced cases of VRE in hospitalized patients since 2012 (CDC, 2019). Cases of colonization do not require antibiotic treatment (CDC, 2024o).

Multidrug-resistant and Extensively drug-resistant Pseudomonas aeruginosa

Definition: *Pseudomonas aeruginosa* is a commonly occurring bacteria, often found in soil and water (CDC, 2024g), that causes healthcare-associated infections, including pneumonia, bloodstream infections, urinary tract infections, and surgical site infections. *Pseudomonas aeruginosa* is particularly dangerous for patients with chronic lung disease (CDC, 2019). Historically, beta-lactams were used to treat these pathogens, but due to the development of resistance, they are now typically ineffective. Multidrug-resistant species resist at least one antibiotic in at least three drug classes. Extensively drug-resistant strains are resistant to nearly all antibiotics (at least one agent in all but two or fewer antibiotic classes) (Kunz

Coyne et al., 2022), including carbapenems, which are typically used for difficult-to-treat infections (CDC, 2019). These pathogens are responsible for at least 32,600 infections, 2,700 deaths, and $767 million in healthcare costs each year (Kunz Coyne et al., 2022).

Mechanism of Resistance: *Pseudomonas aeruginosa* uses multiple mechanisms to resist the effects of antibiotics and often uses more than one mechanism at once. Outer membrane permeability may be restricted through altering binding sites, efflux systems that can pump out antibiotics may be present, and enzymes that can destroy antibiotics may all be used to resist treatment (Kunz Coyne et al., 2022). A small portion of *Pseudomonas aeruginosa* pathogens contain the genetic element that produces the carbapenemase enzyme. This genetic component is easily shared, and drug resistance among this species is rapidly increasing (CDC, 2019).

Risk Factors: *Pseudomonas aeruginosa* is considered a nosocomial infection; therefore, hospitalized individuals are at increased risk for infection (Kunz Coyne et al., 2022). Individuals who have been admitted to the intensive care unit in the past year, immunocompromised patients, patients with chronic lung disease, and those who have previously received antipseudomonal drugs like carbapenems and fluoroquinolones are at increased risk for multidrug-resistant and extensively drug-resistant *P. aeruginosa* (Kunz Coyne et al., 2022). Patients who are on mechanical ventilators, have indwelling medical devices, or have surgical wounds or burns are also at increased risk (CDC, 2024g).

Signs and Symptoms: Symptoms of *P. aeruginosa* infection vary depending on the type of infection. Blood infections may cause chills, fatigue, fever, joint pain, hypotension, and muscle pain. Ear infections may cause earache, discharge, itching, swelling, and hearing loss. GI tract infections may result in headaches, diarrhea, nausea, and vomiting. Eye infections may lead to inflammation, pain,

discharge, redness, swelling, and sudden vision loss. Lung infections may cause chills, cough, dyspnea, and fever. Skin infections may result in discolored bumps, foul-smelling exudate from wounds, itchiness, and abscesses. Urinary tract infection symptoms include urgency, frequency, incontinence, pelvic pain, and dysuria (Cleveland Clinic, 2023b).

Diagnostics: The diagnosis of *P. aeruginosa* is based on specimen culture and testing for antimicrobial susceptibility. This testing will also determine whether the particular pathogen is multi-drug-resistant or extensively drug-resistant (CDC, 2024g).

Treatment and Prevention: Infection prevention and appropriate antibiotic use have contributed to the decline in *Pseudomonas aeruginosa* infections since 2012 (CDC, 2019). Susceptibility testing in the lab can help to determine the most effective treatment (Prevention, 2024g). Combination therapy with a traditional antipseudomonal beta-lactam and an antibiotic of a different class is used to treat infections. However, this can lead to toxicity and secondary infections, particularly *C. difficile*. New antimicrobial agents that combine beta-lactams and beta-lactamase inhibitors have been introduced, but research regarding efficacy is ongoing. These agents may be used as monotherapy when other treatments have been unsuccessful. This method is preferred over a combination of traditionally used antibiotics (Kunz Coyne et al., 2022). Water management plans may be necessary in some regions to contain an outbreak (CDC, 2024g).

Drug-resistant nontyphoidal Salmonella

Definition: Nontyphoidal *Salmonella* is responsible for 1.35 million infections each year. Of those, 212,500 are attributed to drug-resistant strains of the pathogen. Of the 420 Nontyphoidal *Salmonella* related deaths each year, it is estimated that 70 are due to drug-resistant nontyphoidal *Salmonella*. This pathogen is acquired by

humans when they consume contaminated food products or encounter feces from infected people or animals. Drug-resistant nontyphoidal *Salmonella* is classified as a serious threat by the CDC, as the percentage of *Salmonella* strains resistant to ciprofloxacin, azithromycin, and ceftriaxone treatments is increasing (CDC, 2019).

Mechanism of Resistance: Nontyphoidal *Salmonella* has demonstrated resistance to treatment with ampicillin, trimethoprim-sulfamethoxazole, and chloramphenicol. Resistance to third-generation cephalosporins has also emerged in all sub-Saharan African regions. There has been some fluoroquinolone and azithromycin resistance (Crump et al., 2023). These pathogens utilize efflux pumps, antimicrobial inactivation through enzymes, changes in DNA that alter target sites, and changes in the cell membranes to resist the actions of antimicrobial agents (Alenazy, 2022).

Risk Factors: Children younger than five years of age, immunocompromised and malnourished individuals, those with HIV/AIDS, or a recent case of malaria are at increased risk for a nontyphoidal *Salmonella* infection. Living in areas of poor sanitation and hygiene challenges increases the risk of infection (Crump et al., 2023). International travelers who are adventurous eaters or pet/touch animals are at increased risk (Plumb et al., 2024).

Signs and Symptoms: This bacterium can cause bloody diarrhea, fever, and abdominal cramps. Severe cases can include bloodstream infections and lead to life-threatening complications (CDC, 2019).

Diagnostics: When available, clinical laboratory testing and cultures are used to diagnose nontyphoidal *Salmonella* (Plumb et al., 2024). In many regions, nontyphoidal *Salmonella* infection may be difficult to diagnose, as symptoms present similarly to other febrile illnesses, like malaria (Crump et al., 2023). As a result, in areas of the world where the source of infection may not be reliably

identified, all children with severe malaria are typically also treated with antibiotics. As this could contribute to antibiotic resistance, research is ongoing to determine a more accurate method to diagnose *Salmonella* infections (Crump et al., 2023).

Treatment and Prevention: Without treatment, nontyphoidal *Salmonella* infections typically resolve after 1-7 days. Oral rehydration therapy, in addition to other supportive therapies, is recommended (Plumb et al., 2024). Severe *Salmonella* infections are sometimes treated with ciprofloxacin, azithromycin, and ceftriaxone, but drug-resistant strains can be more severe and are more likely to lead to hospitalization (CDC, 2019). Susceptibility testing should be used to determine the appropriate treatment (Plumb et al., 2024). Oral antibiotics are used for uncomplicated cases. Research regarding prevention through vaccination is in progress (Crump et al., 2023). Safe food and water precautions should be utilized to prevent infection. Travelers to regions where nontyphoidal *Salmonella* is prevalent should avoid untreated water, undercooked meat, or other animal products. Good hand hygiene practices should be followed (Plumb et al., 2024).

Drug-resistant Salmonella serotype Typhi

Definition: Different from drug-resistant nontyphoidal *Salmonella*, the serotype Typhi version of the bacteria causes typhoid fever, which can be fatal. Therefore, the CDC categorized drug-resistant *Salmonella* serotype Typhi as a serious threat. This pathogen is responsible for approximately 4,100 infections and less than five deaths each year in the United States. Globally, *Salmonella* Typhi causes 11-21 million infections each year (CDC, 2019). Drug-resistant *Salmonella* Typhi is defined as strains that are resistant to treatment with antibiotics commonly used in the past, including chloramphenicol, ampicillin, and trimethoprim-sulfamethoxazole (Marchello et al., 2020).

Mechanism of Resistance: In 2017, data showed 74% of *Salmonella* Typhi infections were resistant to treatment with ciprofloxacin. One recently emerging strain is resistant to all but two antibiotic classes (CDC, 2019). These pathogens have developed resistance to antimicrobial therapy through genetic changes that allow the bacteria to inactivate antibiotic agents, alter drug targets, and through efflux pumps (Khan & Shamim, 2022).

Risk Factors: Most infections reported in the United States are due to international travel to countries where this pathogen is common due to poor sanitation and lack of safe drinking water (CDC, 2019). Those exposed to contaminated food and water sources are most at risk for a *Salmonella* Typhi infection (Khan & Shamim, 2022).

Signs and Symptoms: *Salmonella* Typhi leads to typhoid fever. Infections may initially be asymptomatic for 2-4 weeks, followed by fever, fatigue, loss of appetite, headache, myalgia, nausea, dry cough, and diarrhea. Bacteria can spread from an asymptomatic host. This illness can lead to perforated intestines, internal bleeding, and hemorrhage. In very severe cases, neurological symptoms may be observed (Khan & Shamim, 2022).

Diagnostics: It can be challenging to distinguish typhoid fever from other severe febrile illnesses. Laboratory testing is ideal but may not be available in low- and middle-income countries (Marchello et al., 2020). Though the standard procedure for diagnosing typhoid fever is bone marrow biopsy, it is not commonly performed, as this test is impractical in many settings. When testing is available, blood culture is more commonly used. More sensitive diagnostic methods continue to be investigated (Khan & Shamim, 2022).

Treatment and Prevention: Treatment requires antibiotics, though the bacteria is becoming increasingly resistant to treatment (CDC, 2019). Vaccination is available and recommended before travel to prevent typhoid fever (CDC, 2019). The

prevalence of typhoid fever can be controlled by increasing access to safe drinking water and food and improved sanitation (Marchello et al., 2020).

Drug-resistant Shigella

Definition: Drug-resistant *Shigella* bacteria are categorized as a serious threat. *Shigella* is present in feces and is spread through contact between people, including sexual contact or contaminated food, water, or surfaces. *Shigella* causes approximately 450,000 infections annually, and it is estimated that 77,000 of these are due to drug-resistant strains (CDC, 2019).

Mechanism of Resistance: *Shigella* resist the actions of antibiotics through various mechanisms, including target site modification, enzymatic inactivation, and efflux pumps (Asad et al., 2024)

Risk Factors: Specific populations are at increased risk for drug-resistant *Shigella* infections, including young children, immunocompromised individuals, men who have sex with men, and travelers to countries with inadequate sanitation (CDC, 2019). People experiencing homelessness are also at high risk for infection. Individuals who eat or drink contaminated food or water or who are in contact with someone who has recently had a *Shigella* infection are at increased risk for infection (CDC, 2024h).

Signs and Symptoms: Symptoms of *Shigella* infection typically start 1-2 days after infection and typically last about seven days. *Shigella* can cause bloody or prolonged diarrhea, fever, abdominal pain, and rectal pressure (CDC, 2024h).

Diagnostics: The diagnosis is based on a physical exam and laboratory testing of a stool sample for *Shigella* toxins (Mayo Clinic, 2024). Susceptibility testing with diagnosis is necessary to determine treatment (CDC Health Alert Network (CHAN), 2023).

Treatment and Prevention: Most *Shigella* infections do not require treatment, but severe infections are typically treated with antibiotics, including azithromycin and ciprofloxacin, especially in immunocompromised patients (CDC, 2019). Ampicillin or trimethoprim/sulfamethoxazole may be used for resistant strains (CHAN, 2023). This antibiotic treatment aims to reduce the number of days the patient experiences diarrhea, reducing the risk of spreading the pathogen. Since 2013, the number of drug-resistant *Shigella* bacteria has increased significantly. This pathogen spreads quickly, and researchers are exploring new prevention efforts. *Shigella* surveillance does not typically include sexual behavior information, which may be a missed opportunity for data collection and disease prevention since outbreaks are frequently documented among men who have sex with men. Strategies implemented to reduce the prevalence of sexually transmitted infections could help to decrease the incidence of drug-resistant *Shigella* infections (CDC, 2019). There are no guidelines to treat extensively drug-resistant *Shigella*, but recent research suggests oral pivmecillinam with fosfomycin, or IV carbapenems with colistin may be effective treatment strategies (CHAN, 2023).

Methicillin-resistant Staphylococcus aureus (MRSA)

Definition: MRSA is a commonly recognized strain of *Staphylococcus aureus*, with an estimated 323,700 cases leading to hospitalization in 2017. That same year, 10,600 deaths and $1.7 billion in healthcare spending were attributed to MRSA, leading the CDC to categorize it as a serious threat. *Staphylococcus aureus* spreads quickly and commonly causes infections in healthcare settings. MRSA can lead to infections that are difficult to treat due to antibiotic resistance (CDC, 2019). It is reported that as many as 90% of *Staphylococcus aureus* infections are due to MRSA (Abebe & Birhanu, 2023).

Mechanism of Resistance: MRSA is resistant to antibiotics through the expression of a penicillin-binding protein that blocks the action of beta-lactam antibiotics, including methicillin, and through beta-lactamase enzymes that destroy these antibiotics (Abebe & Birhanu, 2023).

Risk Factors: Individuals who use injected drugs are 16 times more likely to develop an invasive MRSA infection compared to individuals who do not (CDC, 2019). Individuals who are hospitalized or who have spent significant amounts of time in healthcare facilities are at increased risk for infection. Community-associated cases began to be noted in the 1980s (Abebe & Birhanu, 2023). High school wrestlers, childcare workers, and those living in crowded conditions are at increased risk for community-associated MRSA (Mayo Clinic, 2022b).

Signs and Symptoms: MRSA often begins as a painful abscess on the skin. These areas may feel warm to the touch, contain pus or other drainage, and may be accompanied by a fever (Mayo Clinic, 2022b). Severe cases of MRSA may cause life-threatening infections in the bones and surgical wounds, as well as bacteremia, endocarditis, and pneumonia (Cleveland Clinic, 2024c).

Diagnostics: MRSA is diagnosed by examining tissue samples and cultures. Since cultures typically take 48 hours or more to show results, new testing that detects staphylococcus DNA is becoming more commonly utilized (Mayo Clinic, 2022b).

Treatment and Prevention: Several therapies are available for MRSA infections, but this pathogen has become resistant to many commonly used antibiotics (CDC, 2019). Treatment of MRSA abscesses requires surgical draining (Mayo Clinic, 2022b). Research is focused on alternative therapies (Abebe & Birhanu, 2023).

MRSA infections can be prevented through various strategies. Screening all patients upon admission, tracking MRSA infections, using contact precautions when working with infected patients, and emphasizing effective hand hygiene

practices have significantly reduced MRSA infections. Overall, MRSA infections are steadily decreasing, but progress in preventing MRSA-related sepsis is slowing (CDC, 2019).

Drug-resistant Streptococcus pneumoniae

Definition: *Streptococcus pneumoniae*, or pneumococcus, is responsible for two of every five infections (CDC, 2024j). This pathogen is a leading cause of bacterial pneumonia and meningitis in the United States and also causes bloodstream, ear, and sinus infections. There are more than 2 million pneumococcal infections in the United States annually, which leads to more than 6,000 deaths and approximately $4 billion in healthcare spending. Pneumococcal pneumonia is the cause of approximately 150,000 hospitalizations each year. Research suggests that more than 30% of pneumococcal infections resist one or more antibiotics (CDC, 2019).

Mechanism of Resistance: Antibiotic resistance is caused by alteration in molecular cell wall targets, which inhibits the binding of some antibiotics, efflux pumps, and enzymes. These changes that increase antibiotic resistance are accomplished through genetic changes in the DNA (Li et al., 2023)

Risk Factors: Individuals who have recently used antibiotics are more likely to experience a resistant strain of *S. pneumoniae*. People who work at or attend childcare centers, those who have been recently hospitalized, or who are immunocompromised are also at increased risk for infection (CDC, 2024j).

Signs and Symptoms: Symptoms of *S. pneumoniae* include those consistent with otitis media, nasosinusitis, pneumonia, bacteremia, and meningitis (Li et al., 2023). These symptoms may appear 1-3 days after exposure (CDC, 2024n).

Diagnostics: Diagnosis may be made through various methods. If the provider suspects meningitis or bacteremia, samples for blood culture may be obtained.

Molecular diagnostic techniques may be used. Urine tests for diagnosis in adults are available (CDC, 2024f).

Treatment and Prevention: Unlike most drug-resistant pathogens, an effective vaccine is available to prevent *S. pneumoniae* infections (CDC, 2019). Penicillin continues to be the primary treatment for *S. pneumoniae*, followed by erythromycin (CDC, 2024j).

Drug-resistant Tuberculosis (TB)

Definition: Tuberculosis (TB) is a condition that results when *Mycobacterium tuberculosis* attacks the lungs, which causes serious illness and can lead to death. In 2017, the drug-resistant form was responsible for 847 documented cases of drug-resistant Tuberculosis, resulting in 62 deaths. TB spreads from host to host through droplets in the air and is one of the most deadly diseases in the world. Drug-resistant TB describes strains that are resistant to one antibiotic. Multidrug-resistant (MDR) cases of TB are resistant to two first-line antibiotics commonly used for TB treatment (CDC, 2019). In 2023, 1.4% of drug-resistant TB were categorized as multidrug-resistant strains (CDC, 2025b). Extensively drug-resistant (XDR) cases are resistant to some first- and second-line antibiotics. Treatment for drug-resistant TB is expensive. Treating an MDR case costs $164,000, and XDR cases cost $526,000 (CDC, 2019).

Mechanism of Resistance: Multiple genetic mutations can increase drug resistance of *M. tuberculosis* strains. One mechanism is that a genetic mutation causes the bacteria not to need the element the antibiotic targets to function. Efflux pumps and protein modifications are also resistance mechanisms found in *M. tuberculosis* (Dheda et al., 2024). Primary drug resistance is when a person has contracted drug-resistant TB from another person. Secondary drug resistance is when, during TB treatment, the bacteria become resistant. This may be due to

inappropriate treatment, noncompliance, an issue with the body's absorption of medications, or drug-drug interactions that cause low serum drug levels (CDC, 2025b).

Risk Factors: Drug-resistant TB is most commonly found in Central Asia and Eastern Europe. This disease remains associated with poverty, though malnutrition, overcrowding, and smoking are also risk factors. Individuals with HIV, diabetes mellitus, cancer, and chronic lung disease are at increased risk for TB infection (Dheda et al., 2024). People who have spent time with someone known to have TB are at increased risk. Drug-resistant TB is also found in individuals who have been treated for TB in the past. This risk is increased for people who do not take all their TB medications consistently and correctly (CDC, 2023). Traveling to areas with a high prevalence of drug-resistant TB increases risk (CDC, 2025b).

Signs and Symptoms: Initially, symptoms of TB include low-grade fever, fatigue, and cough during the primary phase of infection. This phase is followed by the latent phase, which is typically asymptomatic. The latent phase may last a few months or multiple years. Following the latent phase is active TB disease. Symptoms of active TB begin gradually and worsen over time. They include cough, coughing blood or mucus, chest pain, pain with breathing/coughing, fever, chills, night sweats, weight loss, poor appetite, fatigue, and malaise. TB can spread from the lungs to other body parts, and pain may occur near the infection site. Infants with TB may be lethargic, fussy, feed poorly, have bulging fontanel, and have poor reflexes (Mayo Clinic, 2025).

Diagnostics: Diagnosis is based on physical exam (Mayo Clinic, 2025) and laboratory testing (CDC, 2023). Drug susceptibility testing is necessary to determine if the patient has a drug-resistant strain of TB. Susceptibility testing should be repeated for patients who do not show clinical improvement after three months of antibiotic treatment (CDC, 2025b). There is a screening test available

for TB. A positive test indicates the individual likely has either latent or active TB disease. A positive test could also be a reaction to a TB vaccination. A chest x-ray is used to determine the presence of active TB (Mayo Clinic, 2025).

Treatment and Prevention: Most of the time, TB can be cured, but without adequate treatment, it is often fatal (CDC, 2019). TB treatment is complex. Drug-resistant TB can be prevented by taking all prescribed TB medications consistently and correctly (CDC, 2023). Due to effective control strategies, the number of drug-resistant TB cases is stable in the United States (CDC, 2019).

Erythromycin-resistant group A Streptococcus

Definition: Group A Streptococcus (GAS) bacteria is categorized as a concerning pathogen by the CDC as it causes 1-2.6 million cases of strep throat and 12,500-20,000 invasive infections each year. Annually, between 1,250 and 1,900 deaths each year can be attributed to GAS infections (CDC, 2019). Data from 2023 suggests that group A *Streptococcus* cases are at an all-time high (CDC, 2024m). GAS is the leading cause of sore throat symptoms, often called “strep throat.” This pathogen can also cause serious infections, like cellulitis, pneumonia, flesh-eating infections, and sepsis. This pathogen is becoming increasingly resistant to treatment with erythromycin and clindamycin. The percentage of group A *Streptococcus* infections resistant to erythromycin treatment has almost tripled in the last eight years. This complicates treatment and can lead to rheumatic fever, which can cause heart damage (CDC, 2019).

Mechanism of Resistance: One in three cases of group A *Streptococcus* is estimated to be resistant to erythromycin and clindamycin (CDC, 2024m). Efflux pumps and changes to drug-binding sites are mechanisms *Streptococcus* uses to resist treatment with erythromycin (Powell et al., 2023).

Risk Factors: Older adults, those with compromised skin integrity, and those with chronic medical conditions are at increased risk for serious GAS infections (CDC, 2019). People experiencing homelessness or who use injected drugs are at increased risk for group A *Streptococcus*. The rate of infection among children remains stable (CDC, 2024m).

Signs and Symptoms: Minor *Streptococcus* infections may cause symptoms consistent with impetigo, scarlet fever, and strep throat. Serious infections include symptoms consistent with cellulitis, necrotizing fasciitis, and streptococcal toxic shock syndrome. Infections can lead to long-term medical problems, including post-streptococcal glomerulonephritis and rheumatic fever (CDC, 2024e).

Diagnostics: To test for Streptococcus, a swab of the throat or suspected infected area is taken, and a rapid strep test or throat culture is used to determine if infection is present (CDC, 2024e).

Treatment and Prevention: GAS continues to be susceptible to penicillin and amoxicillin, but erythromycin and azithromycin may be used for patients who cannot take penicillin. Penicillin and clindamycin are often used in conjunction to treat severe GAS infections. As GAS becomes increasingly resistant to erythromycin and other macrolides, including clindamycin, the treatment of strep throat and invasive GAS infections becomes more complicated (CDC, 2019).

Clindamycin-resistant group B Streptococcus

Definition: Group B *Streptococcus* (GBS) is a bacterium that affects people of all ages. The CDC categorizes it as a concerning risk. In 2016, 31,000 severe GBS infections were documented, with an estimated 13,000 of those being attributed to clindamycin-resistant group B *Streptococcus*. In 2016, there were an estimated 720 deaths due to this drug-resistant strain (CDC, 2019).

Mechanism of Resistance: Group B *Streptococcus* uses multiple mechanisms to resist clindamycin treatment. This includes methylation modification of ribosomes and efflux pumps (Liu et al., 2023).

Risk Factors: GBS occurs in pregnant women, older adults, and individuals with complex medical conditions like diabetes. A GBS infection can be spread to an infant during labor and birth, causing a potentially life-threatening case of sepsis during the first week of life (CDC, 2019).

Signs and Symptoms: Pregnant women with group B *Streptococcus* infection may be asymptomatic or have symptoms consistent with sepsis, meningitis, osteomyelitis, and endocarditis. Non-pregnant adults may experience pneumonia, bacteremia, urinary tract infections, and skin/soft tissue infections (Liu et al., 2023).

Diagnostics: Diagnosis is conducted by collecting a specimen via swab and laboratory testing. Pregnant women are routinely screened in the urogenital area between 36 and 37 weeks of pregnancy (Cleveland Clinic, 2022).

Treatment and Prevention: Typically, penicillin is used to treat GBS, though those who cannot take penicillin may receive clindamycin. This makes clindamycin-resistant GBS particularly troublesome. It is estimated that clindamycin-resistant organisms cause 40% of all GBS infections. It is even more common for these pathogens to resist erythromycin treatment. This limits treatment options for GBS and places patients at risk (CDC, 2019).

Azole-resistant Aspergillus fumigatus

Definition: *Aspergillus fumigatus* is a common fungus that causes severe infections called aspergillosis in immunocompromised individuals. It is typically treated with triazole antifungal medications, commonly called azoles (CDC, 2024k). Azoles are often used in agricultural settings to prevent and treat fungal diseases in commercial crops. Combined with long-term use in humans, *Aspergillus fumigatus* has become increasingly resistant to azole treatment (CDC,

2019). It is estimated that 19% of *Aspergillus fumigatus* infections are resistant to treatment in some countries. Individuals infected with the azole-resistant form of this pathogen are 33% more likely to die as a result of their illness than patients who are not infected with the azole-resistant strain (CDC, 2024k). Azole-resistant *Aspergillus fumigatus* is not commonly identified in the United States but is more prevalent in other countries. Identifying respiratory illnesses due to *Aspergillus fumigatus* is difficult because the symptoms are similar to other respiratory diseases, and many US laboratories lack the necessary equipment to test for azole resistance (CDC, 2019). As a result, aspergillosis is one of the most prevalent missed diagnoses in intensive care units (CDC, 2024k). Azole-resistant *Aspergillus fumigatus* is on the CDC watch list, and research continues to determine this pathogen's threat to humans (CDC, 2019).

Mechanism of Resistance: Long-term use of azole antifungals can lead to azole-resistant *A. fumigatus* infection. The use of azoles in agriculture can also lead to resistance. Typically, azole antifungals target the fungi's cell membrane, but genetic changes in the cell membrane reduce their ability to disrupt it (Rivelli Zea & Toyotome, 2022).

Risk Factors: Immunocompromised individuals are at increased risk for infection (Rivelli Zea & Toyotome, 2022) Individuals with chronic lung disease or severe acute lung infection are also at risk (CDC, 2024k).

Signs and Symptoms: Chronic lung disease is a symptom of infection (Rivelli Zea & Toyotome, 2022). A case of aspergillosis can turn into aspergilloma, which are fungal masses in the lungs. Symptoms of aspergilloma may include hemoptysis, wheezing, shortness of breath, unintentional weight loss, and fatigue. Invasive aspergillosis can cause fever and chills, hemoptysis, chest or joint pain, headaches, eye symptoms, and skin lesions (Mayo Clinic, 2022a).

Diagnostics: Diagnosis may be difficult since *Aspergillus* is common in all environments. Providers may use chest X-rays, sputum tests, tissue or blood tests, and biopsies to direct an accurate diagnosis (Mayo Clinic, 2022a).

Treatment and Prevention: Uncomplicated single aspergillomas do not require treatment and often do not respond to medications. They are frequently monitored by X-rays, and antifungals may be used if the condition worsens. Allergic bronchopulmonary aspergillosis treatment focuses on preventing existing asthma or cystic fibrosis conditions from worsening. Oral corticosteroids may be used for these patients. The most effective antifungal is a new drug, voriconazole. Treatment may also include Amphotericin B. Surgery may be necessary to remove a fungal mass (Mayo Clinic, 2022a).

Drug-resistant Mycoplasma genitalium

Definition: *Mycoplasma genitalium* is a sexually transmitted bacteria that causes urethritis in men and cervicitis in women. If left untreated, it can lead to pelvic inflammatory disease in women. Drug-resistant *Mycoplasma genitalium* is challenging to treat, as it is becoming more resistant to azithromycin, the recommended antibiotic used for treatment globally. Research continues to establish the prevalence and the level of drug resistance among the *Mycoplasma genitalium* population (CDC, 2019).

Mechanism of Resistance: Antimicrobial resistance in *Mycoplasma genitalium* is due to simple DNA mutations. This bacterium can mutate quickly, which facilitates the development of antibiotic resistance. Resistance is typically through mutation-based target modifications. Fluoroquinolones have two targets when treating bacteria, which can cause the pathogen to mutate even more quickly than usual. The use of fluoroquinolones in Japan has rapidly increased fluoroquinolone

resistance of *Mycoplasma genitalium* in that country and is not recommended for use (van Der Schalk et al., 2020).

Risk Factors: Individuals who have unprotected vaginal or anal sex with someone who is infected with *Mycoplasma genitalium* are at risk for infection (CDC, 2025a).

Signs and Symptoms: Symptoms include vaginal discharge, dysuria, and penile discharge (CDC, 2025a).

Diagnostics: Symptomatic patients may be tested for infection using a urine sample or a specimen swab of the vagina or cervix (CDC, 2025a).

Treatment and Prevention: Antimicrobial susceptibility testing before beginning antibiotic therapy is important. Cases are typically treated with macrolides, azithromycin, or josamycin, but increasing resistance may require the use of moxifloxacin or pristinamycin. There has been resistance noted to moxifloxacin. Doxycycline has proven to be effective in treating drug-resistant *Mycoplasma genitalium* (van Der Schalk et al., 2020). The only method to completely avoid a *Mycoplasma genitalium* infection is to abstain from vaginal and anal sex, but using condoms correctly and consistently, as well as a mutually monogamous long-term relationship, can significantly decrease the risk of acquiring a *Mycoplasma genitalium* infection (CDC, 2025a).

Drug-resistant Bordetella pertussis

Definition: *Bordetella pertussis* leads to a respiratory illness commonly known as whooping cough. It is incredibly contagious and can cause severe respiratory illness and lead to death, especially in infants (CDC, 2019). Toxins released by the bacteria cause damage to the cilia and airway swelling (CDC, 2024i). Resistant strains are not commonly found in the United States, but this pathogen is on the

CDC watch list as resistant *Bordetella pertussis* has been found in other countries (CDC, 2019).

Mechanism of Resistance: The primary resistance mechanism is genetic mutations that cause changes in the binding sites. It is suggested that resistance may also be accomplished through methyl group addition, which blocks binding sites, and through efflux pumps (Ivaska et al., 2022).

Risk Factors: Unvaccinated infants and those with underlying medical conditions are at risk for contracting *B. pertussis*. Infants under age one are at the most significant risk for infection and severe complications (CDC, 2024i).

Signs and Symptoms: Early symptoms include typical common cold symptoms, such as nasal congestion, low-grade fever, and mild cough. Infants with whooping cough may experience apnea and dyspnea. Severe cases may include symptoms of rapid, violent, and uncontrolled coughing fits that create a high-pitched “whoop” sound when they inhale. Individuals may vomit after these coughing fits, have difficulty sleeping, and may even fracture a rib due to the intensity of the cough (CDC, 2024i).

Diagnostics: Diagnosis of *Bordetella pertussis* infection is accomplished through culture, nucleic acid detection (PCR), and serology testing. The diagnostic method chosen may depend on the patient's age, vaccination history, and the onset of symptoms (Ivaska et al., 2022).

Treatment and Prevention: Vaccination can prevent infection with *Bordetella pertussis*. Azithromycin and erythromycin are typically used to treat whooping cough, but antibiotic resistance complicates treatment (CDC, 2019). Recently, azithromycin has replaced erythromycin as the primary method of treatment due to its increased efficacy and fewer side effects compared to erythromycin (Ivaska et al., 2022).

Section 2 Personal Reflection

What are commonalities you have observed in antimicrobial-resistant pathogens? Why do you think mechanisms of resistance tend to be similar? Why do you think there may be multiple resistance mechanisms within one type of pathogen? How do diagnostic procedures influence the ability to contain outbreaks of antimicrobial-resistant infections?

Section 3: Impact of COVID-19 on Antimicrobial Resistance

Antimicrobial stewardship, or utilizing antimicrobials responsibly to decrease resistance, was affected by the COVID-19 pandemic. Antibiotics were routinely prescribed for individuals with COVID-19 symptoms when they would not be effective at treating this viral illness. During the height of the pandemic, many bacterial infections were undiagnosed, as routine appointments and screening were not being done. Some rates of illness due to antimicrobial-resistant pathogens decreased during the pandemic, only to resurge once people began to interact on large scales again (CDC, 2022).

The challenges of the COVID-19 pandemic were not conducive to preventing the spread of antimicrobial-resistant organisms. Limited supplies made hand hygiene and cleaning equipment difficult. At times, personal protective equipment was unavailable or allocated to patients with symptoms of COVID-19. In many facilities, separating patients from one another was incredibly challenging due to the number of individuals requiring hospitalization. Indwelling medical devices, a known risk factor for many antimicrobial-resistant pathogens, were also in use longer during the pandemic than typical due to the severity of the illness (CDC, 2022).

Antimicrobial resistance data reporting, used for antimicrobial stewardship policies and epidemiological tracking, has been delayed since 2020. The focus on

COVID-19 management led to a backlog in testing pathogens, which also delayed information dissemination regarding changes in antimicrobial resistance. Rapid identification and treatment of infections are important to prevent severe illness but were challenging during the pandemic (CDC, 2022).

Moving forward, antibiotic stewardship can be improved. The Centers for Disease Control and Prevention are exploring options to better manage antimicrobial-resistant pathogens in the future, including access to wastewater facilities for data collection, studying antimicrobial resistance in wastewater globally and domestically, expanding the capacity to fight antimicrobial resistance in the environment, and mapping existing antimicrobial resistance ecology and its shifts over time. More strategies for infection prevention and diagnostics are also being researched. Public health and healthcare workers continue to impact antimicrobial-resistant pathogen outcomes using the skills and information they have gained from experiencing the COVID-19 pandemic (CDC, 2022).

Section 3 Personal Reflection

What challenges in your practice during the pandemic could have contributed to antimicrobial resistance? How have infection prevention policies in your workplace changed since the COVID-19 pandemic? Do you think these will decrease antimicrobial-resistant infections? How can what we have learned from the pandemic influence antimicrobial stewardship in the future?

Section 4: Nursing Implications

Nurses are critical in preventing increased resistance to antimicrobial agents. The World Health Assembly created a strategic plan that includes improving awareness and understanding of antimicrobial resistance, strengthening knowledge through data collection and research, implementing effective sanitation, hygiene, and infection prevention measures, optimizing the use of

antimicrobial drugs, and encouraging sustainable investment in new medications and vaccines (Salam et al., 2023). Nurses can provide effective patient education to help patients better understand what illnesses antibiotics are necessary for and illnesses they are ineffective in treating. When providing discharge information, nurses must educate their patients to correctly and consistently take their antimicrobial medication to avoid contributing to drug resistance. Nurses are vital in implementing and monitoring infection prevention strategies to improve outcomes and reduce antimicrobial resistance.

Nurses can lead initiatives to help reduce infections and prevent antimicrobial resistance. Healthcare facilities must have a multidisciplinary plan in place to manage these pathogens. Single-patient rooms are ideal, but if they are unavailable, cohorting, or placing patients with similar pathogens together, can be considered. Assigning healthcare personnel to consistent cohorts can help reduce the spread of infection. While cohorting may be a viable option, nurses considering this will need to think about the difficulty level in moving patients, as well as potential staffing challenges. When a patient is transferred to another facility, the receiving facility must be notified of the patient's infection or colonization status and any precautions in place (Borton, 2024).

Nurses use precautions to prevent the spread of infection, including antimicrobial-resistant pathogens. Standard precautions include routinely wearing gloves when needed, effective hand hygiene, and using a mask when fluid splashing could occur. Transmission-based precautions based on the pathogen should also be implemented. Environmental surfaces and patient-care equipment should be sanitized to prevent pathogen transmission. Patient rooms should be cleaned at least daily, and hospital-grade disinfectants should be used following the manufacturer's directions. Healthcare workers should be notified regarding which cleaning responsibilities they are accountable for to ensure the process is assigned and consistently completed (Borton, 2024).

Nurses are often instrumental in infection prevention management. Nurses may be responsible for regular audits of infection prevention practices. They may also participate in surveillance and screening, which usually involves collecting swabs of specimens and communicating with laboratory personnel. Strict adherence to infection prevention measures, removing indwelling medical devices as soon as possible, excellent hand hygiene, patient and caregiver education, and best practices are all ways nurses can improve outcomes and prevent antimicrobial-resistant infections (Borton, 2024).

Section 4 Personal Reflection

How does increased knowledge of antimicrobial-resistant infections impact your clinical practice? How can you ensure patients understand infection prevention teaching? How does cohorting patients reduce the spread of infection when single-patient rooms are unavailable? Why is staffing planning essential to minimize the spread of antimicrobial-resistant pathogens? How can nurses improve outcomes related to these pathogens?

Section 5: Conclusion

Antimicrobial-resistant infections are an ever-changing challenge for our healthcare system. Nurses who understand the most common and most critical resistant pathogens are better prepared to prevent the spread of infection and contribute to reduced resistance. This information is vital as pathogens develop increased resistance to multiple antimicrobial agents, making them difficult to treat, which increases mortality. The COVID-19 pandemic contributed to increased resistance to some pathogens and highlighted the need for improved healthcare processes to manage these infections. Information regarding particular pathogens, signs and symptoms, mechanisms of resistance, risk factors, diagnostic methods,

and treatment methods are all important in helping nurses care for patients with antimicrobial-resistant infections.

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