Review Article

Acinetobacter: An Emerging Threat to Hospitalized Patients

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Abstract

Acinetobacter baumannii(A. baumannii) has emerged as one of the most important opportunistic pathogens associated with healthcare-associated infections worldwide. Its remarkable ability to survive in hospital environments, acquire antimicrobial resistance determinants, and cause outbreaks in intensive care units has made it a major public health concern. This review summarizes the taxonomy, epidemiology, virulence mechanisms, antimicrobial resistance patterns, clinical significance, current therapeutic approaches, and future treatment prospects of *Acinetobacter* species, with particular emphasis on multidrug-resistant (MDR) A. baumannii. A comprehensive review of published literature was conducted, focusing on global and regional epidemiology, molecular mechanisms of pathogenicity and resistance, diagnostic methods, and emerging therapeutic strategies against MDR and carbapenem-resistant *A. baumannii*. *A. baumannii* is a leading cause of ventilator-associated pneumonia, bloodstream infections, urinary tract infections, and wound infections, particularly among critically ill and immunocompromised patients. Its pathogenicity is mediated by multiple virulence factors, including outer membrane proteins, capsular polysaccharides, lipopolysaccharides, fimbriae, and biofilm formation. The organism demonstrates extraordinary genomic plasticity, enabling the acquisition and dissemination of resistance genes through plasmids, transposons, integrons, and insertion sequences. Consequently, MDR, extensively drug-resistant (XDR), and pan-drug-resistant (PDR) strains have become increasingly prevalent worldwide. Although carbapenems remain effective against susceptible isolates, treatment options for resistant strains are limited. Novel agents such as sulbactam-durlobactam, cefiderocol, abaucin, antimicrobial peptides, and phototherapy represent promising future interventions. MDR A. baumannii continues to pose a significant challenge to modern healthcare systems. Strengthened infection control practices, antimicrobial stewardship programs, continuous surveillance, and the development of innovative therapeutic strategies are essential to mitigate the growing threat of this highly adaptable pathogen.

Keywords: *Acinetobacter baumannii*; Multidrug Resistance; Carbapenem-Resistant *Acinetobacter baumannii (CRAB);* Antimicrobial Resistance; Healthcare-Associated Infections; Biofilm Formation; Virulence Factors.

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Introduction

Acinetobacter represents a heterogeneous group of free-living saprophytes that are widely distributed in nature and commonly present in diverse environmental niches.[1] Members of this genus are often part of the normal flora of the human skin[2], mucous membranes and pharyngeal and respiratory secretions.[2,3] They are opportunistic pathogens capable of causing a broad spectrum of infections, including pneumonia, septicemia and wound infections.[4]

In hospitalized individuals, this species primarily colonizes the skin, oropharynx and gastrointestinal tract.[5] These organisms have been recovered from various body sites of healthy individuals, including the forehead, nose, ear, throat, trachea, conjunctiva, hand, vagina and perineum-most frequently inhabiting moist areas such as the axillae, groin and web spaces.[6] *Acinetobacter* spp. have also been isolated from a wide range of animals, including birds, fish and rainbow trout, as well as from food sources such as raw vegetables, fruits, milk and dairy products.[7, 8]

One of the most remarkable features of *A. baumannii* is its ability to survive under desiccated conditions for prolonged periods. This resilience enables it to persist on reusable medical devices such as ventilator tubing, arterial pressure monitoring systems, humidifiers, washbasins, plastic urinals and respirometers.[8]

A. baumannii infections principally occur in healthcare settings, such as hospitals, long-term care facilities, and ICUs. It can cause a range of infections, including bloodstream infections, pneumonia, urinary tract infections, wound infections, and infections of surgical sites. They tend to affect individuals with compromised immune systems, such as individuals who are critically ill, have underlying medical conditions, or have undergone invasive procedures. These infections are troublesome because they can be difficult to treat, particularly when the bacteria are resistant to multiple antibiotics.[9]

It has been demonstrated to be a cause of community-acquired respiratory tract infections, including pneumonia, among immunocompetent individuals residing in tropical regions, in addition to intensive care unit patients. Furthermore, among the most frequent sources of infection among troops who suffered trauma during the Vietnam, Afghanistan, and Iraq conflicts is *Acinetobacter*. Notwithstanding these noteworthy correlations, the extent of the expanding worldwide pandemic of *Acinetobacter* ICU-acquired infections in critically ill patients is incomparable.[10] One of the most concerning aspects of *Acinetobacter* infections is that many strains are resistant to multiple antibiotics. This is due in part to the overuse of antibiotics, which has allowed the bacterium to develop resistance mechanisms. Because of its multidrug resistance, it has been termed an "ESKAPE" pathogen (*Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa, and Enterobacter sp*).[11]

To determine the prevalence and patterns of antibiotic resistance of *Acinetobacter* in critically ill patients throughout the globe, surveillance data were also reviewed along with other prospective and retrospective investigations of ICU-acquired infections. *Acinetobacter* infections are most common in hospitalized patients, especially those who are critically ill or who have weakened immune systems. They are often referred to as nosocomial or hospital-acquired infections. *A. baumannii* can survive on surfaces and medical equipment, allowing it to persist and spread within healthcare environments. Prolonged stays in the intensive care unit are closely linked to the development of infections acquired there in, which is associated to worse outcomes such as higher rates of morbidity and mortality.

Current Taxonomy: *Acinetobacter* species are short, plump, Gram-negative coccobacilli, measuring 1.0–1.5 μm in width and 1.5–2.5 μm in length during exponential growth; they adopt a coccoid form in the stationary phase and may form pairs or elongated chains.[12] The taxonomic classification is as follows:

- Domain: Bacteria
- Phylum: Proteobacteria
- Class: Gammaproteobacteria
- Order: Pseudomonadales
- Family: Moraxellaceae
- Genus: *Acinetobacter*

The genus is highly diverse and its taxonomy has been greatly refined through genomic methods.

Epidemiology

A. baumannii is primarily a healthcare-associated pathogen responsible for a variety of nosocomial infections, including septicaemia, bacteraemia, ventilator- associated pneumonia, wound infections, endocarditis, meningitis and urinary tract infections.[13] Outbreaks are commonly reported in intensive care and burn units, particularly among patients requiring mechanical ventilation.[8]

Global Epidemiology of *Acinetobacter species*

A. baumannii is widely distributed in water and soil, as well as in numerous healthcare settings, where it frequently colonizes hospitalized patients.[8,14] Epidemiological studies have documented the presence of multidrug-resistant *A. baumannii* across multiple regions worldwide, including Europe, North America, Argentina, Brazil, China, Taiwan, Hong Kong, Japan and Korea, often in association with nosocomial infections.[8] Community-acquired pneumonia caused by *A. baumannii* has been reported primarily in tropical areas, particularly during warm and humid months.[8,15]

In addition to *A. baumannii*, *Acinetobacter courvalinii (A. courvalinii)* has been identified as an opportunistic human pathogen. The first clinical isolation of *A. courvalinii* was reported from the urine of an eight-year-old boy with a neurogenic bladder in Russia.[16] More recently, a strain of *A. courvalinii* was isolated in a hospital in Ottawa, Canada, marking the first clinical report of this species in the country.[17]

South Asia Epidemiology of *Acinetobacter spp.* Carbapenem-resistant Gram-negative bacteria, particularly the *Acinetobacter baumannii–calcoaceticus* complex, are highly prevalent in South and Southeast Asia. These regions, which account for over one-third of the global population and host several major medical tourism destinations, face considerable challenges due to the high burden of antimicrobial resistance. In India, carbapenem resistance among *A. baumannii* isolates typically exceeds 40%, with OXA-type carbapenemases being the dominant resistance mechanism.[18,19]

In Pakistan, carbapenem resistance rates in *A. baumannii* range from 62% to 100% among clinical isolates 20. In Bangladesh, a study conducted in a university hospital ICU found that *A. baumannii* was isolated from approximately one-third of clinical samples, and two-thirds of these isolates were resistant to carbapenems.[21]

These data underscore the critical need for continuous regional surveillance, molecular detection of resistance genes, and stringent infection control practices to limit the spread of carbapenem-resistant *A. baumannii* in healthcare settings. [25]

Epidemiology of Outbreaks

A notable characteristic of *Acinetobacter baumannii* is its propensity to cause hospital outbreaks, driven by its multidrug resistance and remarkable ability to survive desiccation on surfaces.[26] Specific resistant clones are often responsible for these outbreaks. In Europe, three major clones, designated as I, II and III, have been identified and have caused geographically distinct outbreaks within healthcare institutions. In most outbreak settings, the majority of *A. baumannii* isolates typically belong to a single clone.[27,28]

Clinical Significance

A. baumannii is a foremost cause of nosocomial infections, incorporating bloodstream infections, ventilator-associated pneumonia, surgical site infections, and urinary tract infections. It creates a significant challenge in healthcare surroundings due to its capability to acquire antibiotic resistance mechanisms.[29] The following is an overview of the clinical significance of Acinetobacter, supported by references:

- Healthcare-associated Infections (HAIs): *A. baumannii* is associated with to a range of healthcare-associated infections, including bloodstream infections, ventilator-associated pneumonia, urinary tract infections, surgical site infections, and catheter-related infections.[30,31] These infections primarily affect persons with compromised immune systems, such as acutely ill patients, those who go through invasive procedures, and individuals with prolonged hospital stays.[32]
- Antibiotic Resistance: *Acinetobacter sp.* has an increased unsavory reputation due to its capability to grow resistance to multiple antibiotics, including carbapenems, which are commonly used as a last resort for treating multidrug-resistant infections. The development of carbapenem-resistant *A. baumannii* (CRAB) has presented noteworthy trials in clinical settings, limiting treatment options, and contributing mortality rates.[33, 34]
- Outbreaks and Clonal Spread: *Acinetobacter* occurrences have been reported in healthcare facilities internationally, often concerning multidrug-resistant strains. These outbreaks are repeatedly accompanied by the clonal distribution of specific strains within a healthcare setting, indicating the significance of infection control measures to avoid transmission.[35, 36]
- Community-Acquired Infections: Although less common, *Acinetobacter* infections can also appear in the group, remarkably in individuals with underlying risk factors or preceding healthcare coverage. Community-acquired strains may exhibit antibiotic resistance, further complicating.[37]

- Wound Infections in Combat Casualties*: A. baumannii* has been an alarm in army healthcare settings due to its capability to be a source of severe wound infections, remarkably in combat fatalities. These infections have determined resistance to multiple antibiotics, posing significant challenges for treatment.[38, 39]

Virulence Factors of *Acinetobacter species*

Acinetobacter is considered to be an organism of low virulence.[8,40] Despite extensive research into the virulence potential of this emerging pathogen, little is still known about its true pathogenic capacity and virulence factors. Virulence factors help bacteria colonize epithelial surfaces, evade and inhibit the host immune response through biofilm formation and obtain nutrition from the host.[41,42] The major virulence factors of *Acinetobacter* include cell surface hydrophobicity, outer membrane proteins (OMPs), capsular polysaccharides and lipopolysaccharides, biofilm formation. Among these, OmpA has been determined to contribute significantly to the pathogenic potential of the organism.[43] OmpA binds to host epithelial cells and mitochondria, inducing mitochondrial dysfunction and swelling. This is followed by the release of cytochrome c, a heme protein, which leads to apoptosome formation and subsequent apoptosis of the host cell.[43] OmpA, being the most abundant surface protein of the pathogen, also contributes to resistance against complement-mediated killing and biofilm formation.[44, 45]

Acinetobacter species expresses surface capsular polysaccharides that serve as targets for protective antibody-based interventions. Antibiotic-induced overproduction of capsular polysaccharides increases resistance to complement-mediated killing. The lipopolysaccharides (endotoxins) are potent stimulators of circulating leukocytes inducing the release of pro-inflammatory mediators such as TNF-α and IL-8 from macrophages.[8,46] These endotoxins are toxic to neutrophils, inhibiting their migration and phagocytic activity. [8,15,40]

The organism also produces various extracellular enzymes, cytotoxins and vascular permeability factors that contribute to tissue damage, particularly in respiratory tract infections.[8,47] Bacterial attachment is believed to be an early and critical step in Gram-negative nosocomial pneumonia.[48] Type 1 fimbriae are adhesive virulence factors involved in adherence and colonization.[49,50]

One of the major factors contributing to the chronicity, persistence and antimicrobial resistance of *Acinetobacter* infections is its remarkable ability to colonize and form biofilms on both biotic and abiotic surfaces.[51] The biofilm formation rate in *A. baumannii* (80–91%) is significantly higher than that observed in other species (5–24%).[52] Biofilm development in *A. baumannii* involves multiple virulence determinants, including outer membrane protein A (OmpA), biofilm-associated protein (Bap), chaperone–usher pilus (Csu), extracellular exopolysaccharide (EPS),two-component regulatory system (BfmS/BfmR), poly-β-(1,6)-N-acetylglucosamine (PNAG) and quorum-sensing systems.[51,53] Other virulence-related enzymes secreted by the bacterium include esterases, aminopeptidases and acid phosphatases, which assist in host tissue invasion and nutrient acquisition.[8,15]

2.11 Species Identification

In 1986, DNA-DNA hybridization was used to delineate species within the *Acinetobacter* genus.[54] Identification of *Acinetobacter* species is challenging due to the lack of standardized techniques. Initially, species were distinguished based on phenotypic characteristics such as growth temperature, colony morphology, growth medium and glucose non-fermentation. The *Acinetobacter calcoaceticus–baumannii*(ACB) complex encompasses closely related species, including *A. pittii*, *A. nosocomialis,* and *A. calcoaceticus* has been expanded to incorporate more recently described species such as *A. seifertii* and *A. lactucae*.[55] Members of this complex exhibit high phenotypic and genotypic similarity, sharing comparable colony morphology, biochemical characteristics and other laboratory traits.[56,57] Due to this similarity, accurate species-level identification within the genus requires molecular approaches.[58] Common genotypic techniques include amplified 16S rRNA gene restriction analysis, high-resolution fingerprinting via amplified fragment length polymorphism (AFLP), sequencing of the 16S–23S rRNA intergenic spacer region, rpoB gene sequencing and gyrB multiplex PCR.[7,33] The OXA-51-like gene serves as a species-specific genetic marker intrinsic to *A. baumannii*.[59]

One notable lineage, the haemolytic clade, is characterized by strong haemolytic activity on sheep erythrocytes and in some strains, proteolytic activity. [60]

Antimicrobial resistance

Over the past three decades, *Acinetobacter baumannii* has progressively acquired resistance to newly

developed antimicrobial agents, leading to the emergence of multidrug-resistant (MDR) strains. These MDR strains are now recognized as major nosocomial pathogens worldwide.[8,61]

Criteria for defining MDR, XDR and PDR in *Acinetobacter* spp. [62]

- MDR (Multidrug-resistant): Non-susceptible to at least one agent in three or more antimicrobial categories.
- XDR (Extensively drug-resistant): Non-susceptible to at least one agent in all but two or fewer antimicrobial categories (i.e., susceptible to only one or two categories).
- PDR (Pan-drug-resistant): Non-susceptible to all agents in all antimicrobial categories.
- This species exhibit remarkable genetic adaptability, being naturally transformable Gram-negative bacteria capable of acquiring foreign resistance genes through transformation, conjugation and transduction.[63] Mobile genetic elements such as plasmids, transposons and integrons play a central role in resistance dissemination.[26] Class I and II integrons are strongly associated with multidrug resistance and are frequently integrated into the chromosome, promoting clonal spread of resistance determinants. Insertion sequences (IS elements) also enhance β-lactamase expression and acquisition of additional resistance phenotypes. [31,63]

Mechanisms of Antibiotic Resistance in *A. baumannii*

The major mechanisms of antimicrobial resistance in *Acinetobacter baumannii* include the following:

1. Enzymatic inactivation of antibiotics
2. Reduced permeability or restricted entry of antibiotics into the bacterial cell
3. Alteration of target sites or cellular functions due to mutations.[64]

A. baumannii is gaining popularity due to a growth in antimicrobial resistance and the emergence of strains that are resistant to nearly all existing medicines.[26,65] This organism is naturally resistant to many antibiotics, including aminopenicillins, third generation cephalosporins (Ceftazidime), chloramphenicol, Imipenem, Chloramphenicol, Ciprofloxacin, Doripenem, and Tobramycin. Its resistance mechanisms involve β-lactamase production, efflux pumps, porin mutations, and aminoglycoside-modifying enzymes, making treatment options increasingly limited. The rapid evolution of MDR *A. baumannii* highlights the urgent need for novel therapeutic strategies and strict antibiotic stewardship to control its spread. A study was conducted on the isolation, characterization, and antibacterial susceptibility of *Acinetobacter* species obtained from a tertiary care hospital. The study found varying resistance levels across different antibiotics. Aminopenicillins showed the lowest resistance at 26.6%, while imipenem resistance was alarmingly high at 76.6%, indicating the widespread presence of carbapenemase enzymes. Similarly, ceftazidime resistance was recorded at 70%, suggesting the role of extended-spectrum β-lactamases. Other antibiotics, including chloramphenicol (63.3%), ciprofloxacin (60%), doripenem (60%), and tobramycin (66.6%), also exhibited high resistance rates. These findings highlight the increasing challenge of multidrug-resistant *Acinetobacter baumannii* infections and emphasize the urgent need for alternative treatment strategies and effective antibiotic stewardship program.[27.66] It also has a remarkable ability to develop resistance mechanisms to broad-spectrum-lactams, aminoglycosides, fluoroquinolones, and tetracyclines, Numerous investigations have found an increase in the number of *A. baumannii* strains that are resistant to these drugs. MDR isolates from geographically different locations have also been shown to be clonally related, whereas susceptible strains are genotypically heterogeneous, implying that the problem of resistance may be associated with a limited number of successful *A. baumannii* lineages. Resistance to carbapenems, which were introduced in 1985 and have been the most important drugs for the treatment of MDR *A. baumannii* infections for many years, is of special concern. Even though clinical *A. baumannii* were shown to be invariably susceptible to these drugs in early studies.[28,67] Hospital outbreaks caused by carbapenem-resistant strains had already been reported by the early 1990s.[29, 68] indicating that *A. baumannii* can cause infections that are completely resistant to the currently available antimicrobial arsenal.

Antimicrobial Therapy

Acinetobacter baumannii is classified by the Infectious Disease Society of America as a "red alert" pathogen due to its significant threat to the effectiveness of current antimicrobials.[7] With rising resistance, only a limited number of drugs can reliably treat MDR *Acinetobacter* infections, making them difficult to manage and often leading to adverse patient outcomes. Carbapenems,

such as imipenem/cilastatin and meropenem, remain the drugs of choice for severe infections caused by susceptible *Acinetobacter* isolates. However, infections with carbapenem-resistant strains are increasingly common. Treatment of these infections requires the use of older drugs like colistin, less commonly used agents such as sulbactam, or drugs with limited clinical experience such as tigecycline. Combination antimicrobial therapy is frequently employed to manage infections caused by multidrug-resistant strains. [69]

Future Treatment Options

New Antibiotics

The application of deep learning has enabled the discovery of *Abaucin*, a structurally novel, narrow-spectrum antibiotic with potent activity against *Acinetobacter baumannii*.[70] This molecule selectively disrupts lipoprotein trafficking by targeting the LolE-mediated pathway, exploiting a unique feature of the *A. baumannii* lipoprotein transport system. The study highlights the growing role of artificial intelligence in antibiotic discovery, providing a promising molecular scaffold for combating multidrug-resistant *A. baumannii* while minimizing off-target effects on commensal flora. [70]

The treatment arsenal for carbapenem-resistant *Acinetobacter baumannii–calcoaceticus* complex (ABC) has been transformed by the phase 3 ATTACK trial, which led to the approval of sulbactam-durlobactam (SUL-DUR).[71] This β-lactam/β-lactamase inhibitor combination demonstrated non-inferior 28-day all-cause mortality compared to colistin and a substantially lower incidence of nephrotoxicity (13% vs. 38%).[71] In a retrospective cohort study of 104 adults with carbapenem-resistant *Acinetobacter baumannii* bloodstream infections, cefiderocol was evaluated against colistin. Patients treated with cefiderocol showed higher clinical cure rates (66% vs. 44.4%) and fewer adverse events, primarily due to reduced acute kidney injury. The findings highlight cefiderocol as a valuable therapeutic option, particularly for frail patients or those at high risk of nephrotoxicity. [72]

Antimicrobial Peptides (AMPs)

The escalating threat of multidrug-resistant *Acinetobacter baumannii*, particularly carbapenem-resistant strains (CRAB), underscores a critical need for novel therapeutic agents beyond last-resort polymyxins.[73] Some AMPs, like the stapled peptide L8S1, function by potentiating conventional antibiotics such as rifampicin, enhancing their penetration by disrupting the outer membrane.[74] Other peptides, including the cyclic peptide B2, adopt a targeted approach by inhibiting essential outer membrane proteins like BamA, leading to bacterial death and showing efficacy in murine lung infection models.[75]

A significant challenge for AMPs is their susceptibility to proteolytic degradation, which can be mitigated through engineering strategies like hydrocarbon stapling to improve in vivo stability.[76] Recent discoveries continue to expand this arsenal; for instance, the peptide DvAMP exhibits potent activity against CRAB by disrupting membrane integrity, binding to lipopolysaccharides and phospholipids and inducing lethal oxidative stress, demonstrating significant efficacy in a mouse pneumonia model.[73] Similarly, Octopromycin, a peptide derived from Octopus minor, has been shown to inhibit biofilm formation and disrupt bacterial membranes in *A. baumannii*, proving effective in zebrafish infection models with low toxicity. [77]

A common and effective mechanism among many of these peptides is the permeabilization of the bacterial membrane, as seen with B2 and DvAMP, which leads to a loss of membrane potential and cell death.[75,73] Crucially, these advanced AMPs are designed not only for high antimicrobial and antibiofilm activity but also for low hemolytic activity and high serum stability, addressing key translational barriers.[73,75] The success of these peptides in various animal models, from zebrafish to mice, confirms their therapeutic potential and in vivo safety.[73,74]

Phototherapy: Phototherapy involves the use of a photosensitizer in combination with light and oxygen to produce reactive oxygen species, which can damage bacterial DNA and disrupt membranes. Currently, this approach is largely restricted to topical applications due to potential local tissue injury. Among studied photosensitizers, tetra pyrrole porphyrins and phenothiazinium salts have demonstrated the most activity against Acinetobacter. [78]

Standard precautions

Includes hand hygiene, proper and consistent use of gloves and appropriate use of gowns and eye protection.

Contact precautions

Involves using dedicated patient care equipment and wearing gowns and gloves for healthcare personnel.

Cohorting patients

Grouping colonized and infected patients within a designated unit or section of a unit to prevent cross-transmission.

Antimicrobial stewardship

Programs designed to promote judicious use of antimicrobials and prevent the emergence of resistance.

Given the wide range of resistance associated with *Acinetobacter*, the function of avoiding the pathogen's spread to additional patients is critical. According to the Centers for Disease Control and Prevention (CDC) infection control recommendations, hospitals with high rates of multidrug-resistant *Acinetobacter* should implement more aggressive infection control measures to control and prevent further nosocomial transmission.[79] Although difficult, strong infection control techniques can effectively control *Acinetobacter* infection outbreaks. Increased staff education, single-use items for specific patients, hand hygiene, cohorting, and isolation are all more likely to be effective measures. Antibiotic control programs appear to be effective in modifying the development of antibiotic resistance under restriction. [80] Controlling the use of ciprofloxacin and ceftazidime, for example, has been linked to a rise in Acinetobacter-associated imipenem and amikacin resistance.[80] Antibiotic control strategies can also change (select for other) microorganisms that cause illnesses in hospitals. Because antibiograms cannot always predict the clonality of an epidemic strain, *Acinetobacter* isolates should be molecularly typed. *A. baumannii* has been known to propagate clonally (from one clone to another) in several hospitals in Europe and Asia. This strain was limited to colistin and tigecycline resistance. [81]

Future Challenges

The control of multidrug resistance and its spread in *A. baumannii* is a difficult task. While multiple drug resistance in this organism is developing and carbapenem resistance is rapidly expanding across the continent, there is a steep decline in the discovery of novel antimicrobial agents that can manage MDR *A. baumannii*. There is no new medicine in the pipeline, and none of the FDA approved antimicrobial agents evaluated had a significant effect on MDR *A. baumannii* control. Existing antibiotics have also failed to limit resistance development and effectively eliminate MDR forms. A logical synergistic approach to several combination medicines, while effective, requires greater in-depth understanding and rigorous trials to control likely outbreaks. The development of pan-drug resistance variations must be avoided, and efforts must be made to find new *anti-Acinetobacter* drugs.

Conclusions

Microbiological monitoring plays a crucial role in tracking bacterial trends and antibiotic resistance patterns in hospitals. It helps in identifying emerging resistance mechanisms, shaping antimicrobial stewardship policies, and contributing epidemiological data for infection control strategies. This review highlights that longer hospital stays, ICU admissions, total parenteral nutrition, invasive procedures such as endotracheal intubation and nasogastric tube insertion, and prior antibiotic use are significant risk factors for *Acinetobacter* infections. MDR *Acinetobacter* species, particularly in hospital ICUs, present a serious global public health challenge, leading to high mortality rates. The geographical variability in resistance patterns underscores the importance of local surveillance in guiding appropriate treatment strategies. With limited treatment options available for MDR organisms, further pharmacokinetic and pharmacodynamic studies are essential to optimize combination therapies until novel, more effective drugs are introduced into clinical practice. Strengthening infection control measures, enhancing antimicrobial stewardship, and fostering research on alternative therapeutic strategies will be key to combating the growing threat posed by MDR *Acinetobacter* infections.

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