Narrative Review Article

Therapeutic Challenges and Opportunities in Treating *Acinetobacter baumannii*: A Review of AI Tools, Peptide-Based Approaches, and Oxidative Stress Mechanisms

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ABSTRACT

Antimicrobial resistance in *Acinetobacter baumannii* has underscored the need for treatments beyond single-drug regimens, given high mortality rates associated with carbapenem-resistant strains. This review explores combination therapy as a framework for addressing the diverse resistance mechanisms presented by *A. baumannii*, including membrane modifications, efflux regulation, biofilm formation, and target-site alteration. This manuscript compares the potential integration of therapeutic pillars, including macrocyclic peptides, AI-driven drug discovery, and oxidative stress, to understand their underlying mechanisms, current applications, and translational limitations as multimodal treatment strategies. Understanding oxidative stress mechanisms is paramount for addressing persistent Antimicrobial resistance (AMR) challenges, revealing why broad Reactive oxygen species (ROS) flooding is ineffective and how localized oxidants can bypass bacterial defenses. Through case studies such as Zosurabalpin, a macrocyclic peptide with selective inhibition, and AI susceptibility models, this review highlights how an interdisciplinary approach can advance AMR therapies. Zosurabalpin's specific mechanism of action demonstrates the value of narrow-spectrum molecules that disrupt essential structural pathways. At the same time, deep antimicrobial susceptibility phenotyping enables rapid phenotypic classification and virtual screening, significantly shortening discovery timelines. Finally, emerging compounds—including synthetic nanoparticles and antimicrobial peptides—are discussed that enhance membrane disruption and potentiate ROS-based killing. This review highlights that no single modality can overcome *A. baumannii*'s adaptability. Instead, the most promising and cost-effective approach is combination therapy, strategically pairing existing drugs to reduce the likelihood of resistance and improve clinical outcomes.

Keywords: *Acinetobacter baumannii*; antimicrobial resistance; combination therapy; macrocyclic peptides; AI drug discovery; oxidative stress; nanoparticles; Zosurabalpin

INTRODUCTION

Antimicrobial resistance (AMR) can be attributed to a variety of factors, including, but not limited to, the misuse of antibiotics and selection of the most virulent bacterial strains that survive through mutations acquired through DNA replication errors or horizontal gene transfer. Mortality rates for invasive carbapenem-resistant infections are incredibly high, ranging from

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40% to 60%, underscoring the urgency of effective bacterial treatments (1). This paper explores combination therapy, the strategic use of multiple antimicrobial approaches, as an essential solution to AMR, driven by the increasing diversity of bacterial strains, genetic and demographic differences, resistance mechanisms, and host responses that make single-drug treatments increasingly ineffective (2). To support this argument, existing research was reviewed to identify what strategies work, what limitations persist, and where innovation is most promising in combating antibiotic resistance. The following sections provide in-depth coverage of tools and methodologies for combating AMR, with a particular focus on *A. baumannii*, a priority pathogen classified by the World Health Organization.

Section I delves into the novelty of Zosurabalpin, a macrocyclic peptide, its mechanism of action, and surveys AI-driven methods that could expedite drug discovery. Section II reviews the advantages of oxidative stress and how this natural mechanism can be used to destroy bacteria, as well as the limitations posed by bacterial mutation. Finally, section III explores additional antimicrobial compounds, including synthetic nanoparticles and peptides.

Acinetobacter baumannii complex has multiple mechanisms of resistance, including alterations in the cell membrane, efflux pumps that affect permeability, and changes in target sites for several small-molecule antimicrobials. This strain of bacteria produces biofilms and thrives in them, causing syndromes such as ventilator-associated pneumonia (VAP) and catheter-associated bloodstream infection, contributing to high mortality (3). Acinetobacter baumannii complexes encased in biofilms are minimally active, so drugs targeting protein synthesis are less effective. Taken together, these demonstrate the clinical importance of investigating *Acinetobacter baumannii* due to its causation of severe hospital-acquired infections, limited therapeutic options, and persistently high morbidity and mortality.

Furthermore, traditional methods of drug development are tedious and time-consuming, such as susceptibility testing, which can take up to a few days. AI-based predictions can detect resistance from genomic data and analyze bacterial motion to determine whether a drug works within 2–4 hours (4). AI tools can also access electronic health records to guide better antibiotic choices. However, while these technologies accelerate detection and decision-making, they do not fully address the biological complexity and adaptability of AMR. This review aims to cover the methodologies explored to combat antimicrobial resistance across different solutions, while emphasizing combination therapy, given that diverse bacterial strains exhibit a variety of resistance mechanisms, making it challenging to discover a novel compound with high and sustained efficacy against multiple bacterial infections.

REVIEW OF AI APPLICATIONS AND MACROCYCLIC PEPTIDES

To contextualize the role of combination therapy in combating resistant bacteria, this section will explore two key pillars—macrocyclic peptides and AI-assisted drug discovery—that support complementary therapeutic strategies for AMR. Together, they provide both novel molecules and discovery workflows, helping researchers design combinations that are synergistic rather than empirically through trial and error.

Macrocyclic Peptides and Zosurbalpin

Macrocyclic peptides are small, linear amino acid chains that are cyclized, thereby limiting conformational flexibility and improving binding affinity for selective targets. Macrocyclic peptides generally exhibit unique, narrow-spectrum antibacterial activity, making them highly effective at destroying bacteria while increasing the difficulty of resistance development, and thus a promising antibiotic class. These peptides undergo macrocyclization—the process of chemically linking the ends or side chains of a linear peptide to form a large ring—which confers protease resistance and improves binding by lowering the entropic cost. The unique structure of macrocyclic peptides provides greater stability within the body and allows for oral consumption. Many clinically essential antibiotics are macrocyclic peptides, including polymyxin, bacitracin, and daptomycin (1).

Roche recently discovered an MCP, Zosurabaloin (ZAB, RO7075573), which was initially classified as RO7036668. The scientists who discovered Zosurabalapin conducted traditional whole-cell phenotypic screening of ~45,000 macrocyclic peptides and physically tested chemical compounds in the lab to identify which compounds effectively killed Acinetobacter baumannii, taking several years after placing a hit before proceeding to clinical trials and treatments (5).

Zosurablapin targets the LPTB2FGC complex in *A. baumannii*, which is vital in transferring lipopolysaccharides (LPS) from the inner membrane to the outer membrane, providing bacterial structural

integrity. Specifically, ZAB blocks where Lpt C is supposed to bind and forms hydrogen bonds (Thr321) with Lpt F through electrostatic interactions between ZAB's lysine (L-lys) and the negatively charged parts in Lpt F (Glu58 & Glu 249). The interrupted protein complex is part of *A. baumannii's* lipopolysaccharide transport system. Inner membrane components (LptB2FGC) hydrolyze ATP for LptB2FGCADE to transfer LPS. A simplified summary of the mechanism for the lipopolysaccharide transport system begins with LptF and LptG binding LPS, which is then passed to LptC and LptA in the periplasmic space between the inner & outer membranes, and finally inserted into the outer membrane via LptDE. When ZAB blocks this pathway, LPS accumulates in the inner membrane instead of reaching its destination. This buildup becomes toxic to the bacterial cell, compromising membrane integrity and ultimately leading to cell death. Testing has shown that the minimum inhibitory concentration (MIC) of Zosurabalpin against *A. baumannii* is 4 μg/mL, but it has limited activity against other Gram-negative and Gram-positive bacteria (1). Although Zosurabalpin's potency has a narrow spectrum, allowing it to kill *A. Baumannii* effectively; its use can be quickly expanded further with AI predictive tools to test for efficacy.

AI-Driven Drug Discovery Tools

In contrast to empirical screening used to identify Zosurabalpin, AI-driven approaches aim to accelerate and optimize antimicrobial discovery by extracting predictive insight from bacterial behavior itself. One such approach is deep antimicrobial susceptible phenotyping (DASP), a deep learning method for analyzing single-cell morphological phenotypes that provides results in as little as 30 minutes, compared with antimicrobial susceptible testing, which takes 18-24 hours (6). DASP examines individual bacterial cells using microscopy and a convolutional neural network (CNN), a type of artificial intelligence model that learns to recognize patterns in images. Specifically, DASP uses Mask R-CNN to segment individual cells from micrographs using the Nile Red channel. Followed by DenseNet121 to classify segmented cells into resistant or susceptible phenotypes using Nile Red and DAPI channels. Then DASP analyzes how cell structures (DNA & membranes) change when treated with antibiotics. This method uses single-cell phenotypes of the nucleoid and cell membrane, combined with CNNs, to classify antibiotic-treated cells as susceptible or resistant. This tool precisely detects bacterial evolution in behavior or exhibits phenotypic plasticity and proves reliable across clinical bacterial samples from patients, supporting the idea that it can capture diverse variations in bacterial behavior between the lab and real-world infections. DASP models have been built for four antibiotics, each targeting different cellular functions: Ciprofloxacin, which blocks DNA replication; Gentamicin, which blocks protein synthesis; Co-amoxiclav, which blocks cell wall synthesis; and Rifampicin, which blocks RNA synthesis. DASP can distinguish between bacteria that look or act differently after exposure to a specific amount of ciprofloxacin.

In the case of Zosurabalpin, the researchers could have used AI to predict active compounds from virtual libraries, to model drug-target interactions computationally, or to analyze bacterial genomes to identify resistance genes. This demonstrates that, instead of testing tens of thousands of compounds experimentally, as Roche did, AI can virtually screen millions of molecules using molecular docking simulations, thereby saving time and resources for clinical trials. Together, these developments illustrate how novel compounds can set the stage for mechanistic diversity to inform the design of combination therapies, guiding the next stage of this analysis, in which we explore additional strategies to combat AMR.

Although AI-designed macrocyclic peptides provide highly targeted antibacterial interventions, their clinical impact can be strengthened by pairing them with naturally occurring mechanisms that exert broad cellular pressure on resistant bacteria. Oxidative stress offers such a complementary pathway, inducing redox-driven damage that disrupts multiple bacterial processes simultaneously. Examining oxidative stress, therefore, reveals how mechanistic diversity—rather than single-agent specificity—forms the foundation of effective combination therapy strategies against antimicrobial resistance.

OXIDATIVE STRESS

This section reviews oxidative stress as a key antibacterial mechanism and examines the successes, failures, and limitations of redox-based approaches as strong candidates for combination therapy rather than stand-alone treatments. Oxidative stress is caused by highly reactive molecules like superoxide (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radicals (·OH). In the context of antimicrobial resistance, many antibiotics either induce oxidative stress or rely on redox-mediated damage to weaken resistant bacteria that evade direct

inhibition at their targets. Reactive oxygen species (ROS) are oxygen-containing molecules that readily react with biomolecules and are produced when oxygen (O_2) gains extra electrons during metabolism or immune responses, leading to oxidative stress that damages bacterial proteins and DNA (7). As resistance increasingly reduces the efficacy of single-target antibiotics, leveraging oxidative stress to destabilize bacterial defenses offers a complementary strategy that can resensitize resistant pathogens and enhance the effectiveness of existing antimicrobial therapies.

Studies in *E. coli* found that ROS, including O_2^- and H_2O_2, can damage proteins containing iron-sulfur (Fe-S) clusters, which are essential enzyme cofactors. The iron released from damaged clusters can react with H_2O_2 in a Fenton reaction, producing hydroxyl radicals (·OH) that disrupt metabolism and amino acid synthesis. Hydroxyl radicals damage DNA, proteins, and any nearby molecules. H_2O_2 can also oxidize cysteine residues in proteins, altering their function. Most importantly, superoxide in bacteria does not damage membranes as bacterial membranes lack polyunsaturated fatty acids, which are needed for lipid peroxidation. Superoxide cannot cross membranes, so its main damage is outside the cytoplasm (periplasm) (6).

Two of the best-known molecules that generate ROS are Menadione (MD) and Streptonigrin (SN); Figure 1 compares the pathways of each compound. MD is a redox-cycling compound that produces large amounts of O_2^- and H_2O_2, which are unlikely to be lethal to bacteria because bacteria can readily defend against ROS. Bacterial enzymes quickly clear up O_2^- and H_2O_2, so MD stresses cells rather than killing them. Alternatively, drugs like SN bind near DNA and generate oxidants locally, which avoids triggering defense systems that are more effective at killing bacteria. SN does not generate much free H_2O_2 or O_2^- inside the cell. Instead, it binds iron near DNA and generates ferryl radicals (Fe=O) that directly damage DNA, bypassing free ROS and causing bacterial lethality (8). Table 1 organizes and compares the mechanisms of action for each drug.

Stated succinctly, oxidative stress is a key antibacterial mechanism, as many antibiotics induce or depend on redox-mediated damage when direct target inhibition fails. ROS disrupt bacterial metabolism by damaging enzymes and amplifying DNA damage. However, bacteria have evolved strong antioxidant defenses that neutralize diffusible ROS such as superoxide and hydrogen peroxide, reducing their lethality and contribution to resistance.

Bacterial Defense Systems and Limits of ROS

Beyond the mechanisms of action of MD and SN, some bacteria have developed mechanisms to mitigate oxidative stress. Unlike most bacteria (which get destroyed), *Salmonella* uses special proteins (via its SPI-2 secretion system) to alter the phagosome, creating a unique compartment called the *Salmonella*-containing vacuole (SCV). This compartment may reduce the intensity of certain antimicrobial attacks, but ROS remain present. Therefore, *Salmonella* must actively resist ROS to survive, and *Salmonella's* SPI-2 secretion system helps modify the SCV to reduce assembly of the Phox enzyme—the host enzyme responsible for producing superoxide—on the vacuole. But Phox activity isn't eliminated, because *Salmonella* still needs

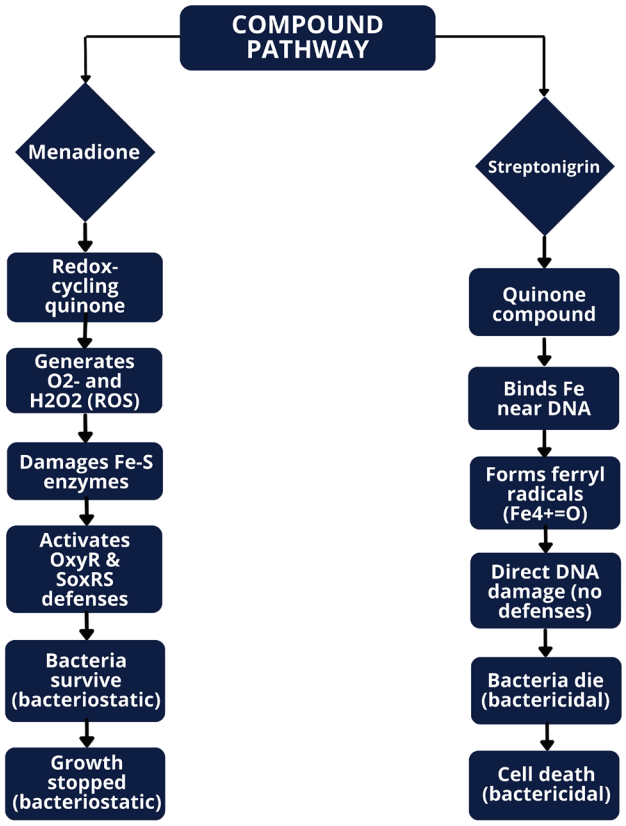
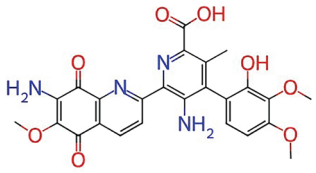
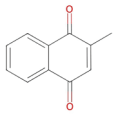


Figure 1. *Comparative Mechanistic Pathways of Menadione and Streptonigrin. Mechanistic comparison of menadione (MD) and streptonigrin (SN), illustrating MD's bacteriostatic effect via ROS generation and cellular stress response activation, versus SN's bactericidal action by iron-mediated ferryl radical formation and direct DNA damage.*

Table 1. *Comparison of the mechanisms between Streptonigrin and Menadione. Bactericidal activity refers to the ability to kill bacteria, whereas bacteriostatic activity refers to the inhibition of bacterial growth.*

Compound	Mechanisms
Streptonigrin ($C_{25}H_{22}N_4O_8$) 	• Avoids ROS flooding • Causes localized DNA damage via ferryl radicals • Defense activation delayed • Causes cell death • Bactericidal*
Menadione ($C_{11}H_8O_2$) 	• Fills cells with ROS • ROS in bacteria activate OxyR and SoxRS defense pathways • Cells survive after repairing damage • Bacteriostatic

SodCI, a protective enzyme that uses Copper and Zinc to neutralize O_2^-. Experiments in Phox-deficient mice show that *Salmonella* grows much better, indicating that ROS are important in controlling infection. However, ROS may not just directly kill bacteria; they may also signal and activate other antimicrobial pathways (9).

Another mechanism, the OxyR system, detects H_2O_2, a common ROS produced by gram-negative bacteria, to mitigate the adverse effects of ROS. When H_2O_2 rises, OxyR turns on genes for scavenging H_2O_2, shrinks the cell's free iron pool (less chance for DNA damage), and repairs oxidized DNA/proteins. MD weakly activates OxyR. Still, this protection matters, since OxyR-deficient bacteria (mutants) are susceptible to menadione. However, SN doesn't produce H_2O_2, and OxyR remains inactivated. Even OxyR mutants respond the same as wild-type cells, but if OxyR is artificially activated, cells become resistant to SN because OxyR depletes iron, which SN requires (8). These mechanisms reveal that bacteria can tolerate oxidative stress when it triggers defensive pathways such as OxyR or SodCI. In contrast, localized, iron-dependent oxidative damage circumvents these systems, highlighting the limits of ROS as a stand-alone antimicrobial strategy.

Though each method has its benefits, iron-dependent oxidative damage can bypass defenses and achieve bactericidal outcomes; diffusible ROS are often neutralized, limiting their reliability as a stand-alone treatment. These findings demonstrate that oxidative stress is constrained by bacterial localization and detoxification pathways. Thus, there is no single ideal solution that can address a broad range of bacteria; instead, a therapy that combines two or more treatments simultaneously could enable efficacy against bacteria that were once resistant to single treatments. A combination therapy of antimicrobial peptides (AMPs) and ROS has been shown to damage *Salmonella's* outer membrane, allowing ROS and proteases better access to targets. SodCI is unusually protease-resistant and tethered, which helps *Salmonella* survive this combined assault. There may be synergistic effects, meaning that ROS makes bacteria more vulnerable to peptides, or that peptides make them more vulnerable to ROS. These limitations highlight why oxidative stress alone is insufficient to overcome AMR yet supports its use as a combination-based therapeutic strategy.

ALTERNATIVE METHODOLOGIES REVIEW

Synthetic Peptides and Nanoparticles

Other promising compounds explored for combating antimicrobial resistance include synthetic nanoparticles (NPs) and peptides, which complement oxidative reactions. More specifically, metallic NPs like iron oxide, gold, zinc oxide, and silver are being studied as new weapons against resistant microbes (10). The hypothesized mechanism of action is that they first attack microbial cell membranes directly, releasing toxic metal ions that damage microbes. This damage then generates ROS, which in turn cause additional oxidative damage. The specialty of NPs is their small size, typically 1-100 nm, which allows them to enter microbial cells easily (11). NPs exhibit high reactivity, customizable surfaces, and a large surface-to-mass ratio. They can

act as antimicrobial agents or drug carriers to deliver antibiotics more effectively (12). NPs can be used in wound dressings, coatings on implants/devices, medical textiles, drug-delivery vehicles, inhalable or topical formulations (13). Because they can act via multiple mechanisms, bacteria may have a harder time developing resistance (requiring multiple simultaneous mutations) than traditional antibiotics. They also offer potential to reduce antibiotic doses, target the infection site, and improve efficacy in challenging contexts (biofilms, device-associated infections) where antibiotics alone fail. A setback of NPs is toxicity and biocompatibility. Since NPs are small and reactive, they may damage human cells or organs, generate ROS in non-target tissues, or release harmful ions.

A highly effective and cost-effective NP is biosynthetic ZnO NPs. ZnO exerts antimicrobial activity through membrane disruption, localized ROS generation, and Zn^{2+} release, making it effective against multidrug-resistant (MDR) pathogens such as A. baumannii and complementary to combination therapy strategies, such as AMPs. ZnO electrostatically binds to the bacterial membrane, interfering with lipid biosynthesis and increasing membrane permeability, thereby allowing Zn^{2+} to enter the bacteria. Furthermore, it generates continuous ROS at the surface, overwhelming the bacteria's antioxidant defense system since many antioxidants operate in the cytoplasm. Its mechanism of action disrupts biofilm formation, a major defense mechanism of *A. baumannii* and Gram-negative bacteria (14).

While each method shows promise, challenges such as narrow activity spectra and potential toxicity underscore that there is no single approach to sufficiently combat MDR *A. baumannii*. Thus, combination therapy is a strategic approach to achieve broader efficacy and minimize the development of resistance. The conclusion will synthesize these insights, emphasizing how integrating empirical discoveries with AI-driven prediction can guide novel antimicrobial interventions.

CONCLUSION

There is no single broad compound that can be applied to all bacterial resistance mechanisms, since different strains exhibit distinct mechanisms. Since every method has its limitations, combining therapies helps mitigate shortcomings and fill gaps that a single approach may lack.

This review has covered the advantages of varying approaches: Zosurabalpin, AI tools, oxidative stress, nanoparticles, and antimicrobial synthetic peptides. Zosurabalpin and macrocyclic peptides demonstrate the potential of structure-based innovation in targeting previously inaccessible pathways. Zosurabalpin's precise inhibition of the $LptB_2FGC$ complex in *A. baumannii* shows that selective mechanisms can disrupt essential bacterial functions with minimal cross-resistance. However, its narrow spectrum and lengthy empirical testing highlight the limitations of relying solely on a single compound class. DASP drastically reduces testing time from over 18 hours to roughly 30 minutes by using machine learning to analyze bacterial morphology.

Oxidative stress–based therapies demonstrate that leveraging natural bacterial vulnerabilities, such as ROS-mediated damage, remains an effective but incomplete strategy. Compounds like menadione (MD) induce broad oxidative stress but fail to kill bacteria outright due to strong enzymatic defenses, such as OxyR and SoxRS. In contrast, streptonigrin (SN) causes lethal DNA damage by generating a localized ferryl radical. These findings emphasize the importance of targeted oxidative disruption over generalized ROS flooding.

While the reviewed studies underscore progress, several methodological and practical limitations persist. We should also acknowledge the lack of clinical validation. Most studies remain preclinical, with no large-scale human trials confirming safety or efficacy. Furthermore, there are accessibility and data limitations, as many AI models rely on private or incomplete genomic datasets, which limit reproducibility and transparency. Rates of AMR also differ geographically due to local prescribing habits, yet the underlying physiological mechanisms remain consistent across humans, suggesting that the findings have global relevance despite regional variability. Despite these limitations, the results collectively illustrate that a multimodal approach—uniting empirical discoveries, computational prediction, and biochemical synergy—holds the most significant promise for mitigating antimicrobial resistance on a global scale.

More importantly, many of these limitations are not inherent barriers; emerging AI-driven tools can enable rapid susceptibility profiling, virtual compound screening, and predictive modeling of resistance evolution, reducing reliance on slow empirical testing and incomplete datasets. Ideas for AI recommendations for research include improved surveillance systems, emphasizing responsible and prudent antimicrobial use, promoting antimicrobial stewardship programs, exploring the relationship between elements leading to

resistance, such as antimicrobial prescribing practices and horizontal gene transfer, fostering interdisciplinary cooperation, developing new antimicrobial drugs and alternative therapies, and enhancing research and development to uncover novel antibiotics.

AI tools such as Synthemol can generate synthetic molecules for drug discovery and antimicrobial resistance research. Synthemol exemplifies how generative models can design synthetic molecules for antimicrobial use, highlighting AI's role in accelerating speed, precision, and adaptability —crucial for keeping pace with bacterial evolution. In future work, integrating generative AI tools like Synthemol with experimental validation pipelines may enable the rational design of combination therapies that are both clinically scalable and resilient to resistance evolution.

CONFLICT OF INTEREST

The author declares that there are no conflicts of interest related to this work.

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