ISSN: 2322 - 0902 (P)
ISSN: 2322 - 0910 (O)

International Journal of Ayurveda and Pharma Research

Review Article

EVOLUTION AT THE HOST-PATHOGEN INTERFACE: MECHANISTIC INSIGHTS INTO ANTIMICROBIAL RESISTANCE

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Article info

Article History:

Received: 19-01-2026

Accepted: 23-02-2026

Published: 15-03-2026

KEYWORDS:

Antimicrobial resistance, Host-pathogen interactions, Resistance mechanisms, Horizontal gene transfer, Biofilm-mediated resistance.

ABSTRACT

The rapid global rise of antimicrobial resistance (AMR) represents a critical threat to modern medicine, undermining the effective treatment of infectious diseases and challenging long-standing therapeutic advances. This review adopts an integrative, mechanistic perspective to examine how microbial adaptation drives resistance development, persistence, and dissemination across clinical and ecological contexts. Core resistance mechanisms include target site modifications, enzymatic drug inactivation, altered membrane permeability, and activation of multidrug efflux systems, which are further amplified by horizontal gene transfer mediated through plasmids, transposons, and bacteriophages. Biofilm formation constitutes a critical phenotypic strategy that enhances antimicrobial tolerance by promoting metabolic heterogeneity, restricted drug penetration, and the survival of persister cells. In parallel, pathogens employ diverse immune evasion strategies, including antigenic variation, immune modulation, and intracellular persistence, prolonging infection and sustaining selective pressure for resistance evolution. Environmental reservoirs of antimicrobial residues and resistance genes originating from clinical, agricultural, and industrial sources further expand the global resistome, underscoring the interconnected nature of AMR across human, animal, and environmental domains. Recent advances in genomics, transcriptomics, proteomics, metabolomics, and systems biology have transformed understanding of resistance pathways and host–pathogen dynamics. Integrating these mechanistic insights within a One Health framework is essential for informing antimicrobial stewardship, guiding innovative therapeutic strategies, and preserving the long-term effectiveness of antimicrobial agents.

INTRODUCTION

Antimicrobial resistance (AMR) has emerged as one of the most formidable and multifaceted threats to global public health, compromising the effective prevention and treatment of infectious diseases and jeopardizing advances in modern medicine [1–4]. The progressive loss of antimicrobial efficacy has transformed previously manageable infections into persistent, recurrent, or life-threatening conditions, resulting in increased morbidity, mortality, and substantial economic burden [3–5]. Current global estimates attribute millions of deaths annually to antimicrobial-resistant infections, with projections indicating that AMR may surpass cancer as a leading cause of mortality in the coming decades if effective interventions are not implemented [3,4]. The emergence and propagation of antimicrobial resistance are fundamentally evolutionary processes driven by selective pressures acting on genetically diverse microbial populations [6–8]. While resistance can arise naturally through spontaneous genetic variation, anthropogenic factors have profoundly accelerated its evolution [6,9]. The widespread and often indiscriminate use of antimicrobial agents in human medicine, agriculture, veterinary practice, and aquaculture has intensified selective pressure, favoring the survival and clonal expansion of resistant variants while eliminating susceptible populations [9–11]. Repeated and sublethal antimicrobial exposure further promotes adaptive responses, enhancing resistance stability and

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Quick Response Code

https://doi.org/10.47070/ijapr.v14i2.3984

persistence [7,22]. Central to resistance development is the dynamic and reciprocal interaction between hosts and pathogens. Host–pathogen interactions govern critical aspects of microbial fitness, including colonization, immune evasion, persistence, and transmission. Pathogens adapt to hostile host environments through a combination of genetic mutations, regulatory reprogramming, phenotypic plasticity, and acquisition of resistance determinants via horizontal gene transfer. Concurrently, host immune defenses exert selective pressure that shapes pathogen evolution, often driving the emergence of immune-evasive and drug-resistant phenotypes. Resistance evolution is further compounded by microbial community-level strategies such as biofilm formation and immune modulation. Biofilms provide a structurally and metabolically heterogeneous niche that restricts antimicrobial penetration, promotes cellular dormancy, and facilitates the survival of persister cells, thereby markedly reducing therapeutic efficacy [18-20]. In parallel, environmental reservoirs of resistance genes, sustained by antimicrobial contamination and ecological dissemination, serve as critical sources for resistance emergence across clinical and non-clinical settings [11,16,17]. Recent advances in molecular biology and omics-based technologies including genomics, transcriptomics, proteomics, metabolomics, and systems biology have revolutionized the investigation of antimicrobial resistance. These approaches enable high-resolution dissection of resistance mechanisms, evolutionary trajectories, and host–pathogen dynamics [21,23]. This review systematically examines the mechanistic basis of resistance development within host–pathogen interactions, integrating genetic, biological, environmental, and technological perspectives to inform the development of innovative therapeutic strategies and sustain the long-term effectiveness of antimicrobial agents [24,25]. Unlike previous reviews that predominantly examine antimicrobial resistance mechanisms in isolation, this review presents an integrated, evolution-informed framework that connects genetic, immunological, ecological, and systems-level processes shaping resistance at the host–pathogen interface [26–30].

METHODOLOGY

Study Design

The present study was conducted as a comprehensive integrative narrative review aimed at critically examining host–pathogen interactions and the molecular, immunological, and ecological mechanisms underlying AMR. An integrative narrative review approach was selected because it allows the synthesis of evidence across diverse disciplines and methodological traditions, enabling conceptual integration beyond the scope of conventional systematic reviews [31]. A multidisciplinary framework was adopted to synthesize evidence from microbiology, immunology, molecular and cellular biology, pharmacology, environmental sciences, and systems biology. This approach enabled a holistic and evolution-informed interpretation of resistance development, persistence, and dissemination across clinical and environmental contexts, rather than restricting the analysis to isolated molecular mechanisms [32].

Literature Search Strategy

A comprehensive and integrative literature review was conducted using major scientific databases, including PubMed and ScienceDirect, to identify peer-reviewed research articles, systematic reviews, and authoritative reports on antimicrobial resistance. The search covered publications from year 1999 to 2026**,** encompassing foundational studies on the origins, evolution, and molecular mechanisms of resistance, ecological and environmental drivers, clinical and public health impacts, and emerging One Health approaches to understanding and mitigating AMR. Search terms included combinations of controlled vocabulary and free-text keywords such as antimicrobial resistance*,* host–pathogen interactions*,* genetic mutations*,* horizontal gene transfer*,* biofilm formation*,* immune evasion*,* environmental resistome*, and* omics technologies*.* Boolean operators and database-specific filters were applied to refine results and prioritize studies with mechanistic and conceptual relevance, consistent with established approaches for AMR literature synthesis [33].

Inclusion and Exclusion Criteria

Publications were selected based on predefined inclusion and exclusion criteria to ensure relevance, scientific rigor, and conceptual depth. Included studies comprised peer-reviewed articles published in English that provided mechanistic insights into antimicrobial resistance, addressed host–pathogen dynamics, or examined genetic, immunological, environmental, or systems-level determinants of resistance. Landmark and historically significant publications were included where they contributed foundational concepts or shaped current understanding of AMR evolution. Studies lacking methodological transparency, non-peer-reviewed reports, and redundant publications were excluded unless they held clear conceptual or historical significance.

Data Extraction and Synthesis

Titles and abstracts were initially screened for relevance, followed by full-text evaluation of selected articles. Approximately 210 articles were screened, of which 78 publications were included based on relevance, mechanistic depth, and contribution to the thematic objectives of the review. Data from the

selected studies were systematically extracted and organized into major mechanistic domains, including genetic determinants of resistance, horizontal gene transfer, biofilm-associated tolerance, host immune evasion strategies, environmental reservoirs, and emerging omics-based analytical approaches [33,34]. A qualitative synthesis methodology was employed to integrate findings across disciplines, identify recurring evolutionary patterns, delineate conceptual linkages between host–pathogen interactions and resistance development, and highlight critical gaps in current knowledge.

RESULTS

Genetic Determinants of Antimicrobial Resistance

Analysis of the reviewed literature consistently identified genetic determinants as primary contributors to antimicrobial resistance across bacterial, fungal, and viral pathogens [35,36]. Resistance-associated genetic changes predominantly arose through spontaneous mutations, including point mutations, insertions, deletions, and gene amplifications. These alterations frequently affected antimicrobial target sites, resulting in reduced drug binding or diminished inhibitory activity [37]. Mutations in ribosomal RNA genes and ribosomal protein–encoding loci were commonly associated with resistance to macrolides, aminoglycosides, oxazolidinones, and tetracyclines [38,39]. Similarly, alterations within quinolone resistance–determining regions of *gyrA*, *gyrB*, *parC*, and *parE* genes were repeatedly linked to reduced fluoroquinolone susceptibility [40]. Resistance to β-lactam antibiotics was frequently associated with structural modifications of penicillin-binding proteins or acquisition of alternative binding proteins with lower affinity for β-lactams [41]. In addition, mutations affecting regulatory genes controlling efflux systems and outer membrane permeability were widely reported, contributing to increased antimicrobial efflux or decreased drug uptake [42]. Across multiple studies, compensatory mutations were observed to mitigate fitness costs associated with resistance, facilitating persistence of resistant strains [43].

Horizontal Gene Transfer and Resistance Dissemination

Horizontal gene transfer (HGT) was identified as a dominant mechanism facilitating the rapid acquisition and dissemination of antimicrobial resistance determinants across microbial populations [44]. Plasmid-mediated conjugation was the most frequently reported pathway, particularly in clinical and agricultural environments characterized by high antimicrobial exposure [45]. Conjugative plasmids commonly carried resistance genes encoding extended-spectrum β-lactamases, carbapenemases, aminoglycoside-modifying enzymes, and colistin resistance determinants [46,47]. These genes were often embedded within integrons and transposons, enabling coordinated gene capture and expression [48]. Natural transformation contributed to resistance acquisition in competent bacterial species through uptake of extracellular DNA, while bacteriophage-mediated transduction enabled transfer of resistance genes across taxonomic boundaries [49]. These mechanisms were shown to accelerate resistance dissemination at rates exceeding those achievable through de novo mutation alone [50].

Biofilm-associated Antimicrobial Tolerance

Biofilm formation was consistently associated with reduced antimicrobial susceptibility and treatment failure across a wide range of pathogens [51]. Biofilms were characterized as structured microbial communities embedded within an extracellular polymeric matrix composed of polysaccharides, proteins, lipids, and extracellular DNA [52]. Studies reported that this matrix impeded antimicrobial diffusion and altered local physicochemical conditions [53]. Within biofilms, spatial gradients of oxygen and nutrients generated heterogeneous microbial subpopulations with variable metabolic activity [54]. Cells located in nutrient-limited regions frequently exhibited reduced growth rates and decreased susceptibility to antimicrobials targeting active cellular processes [55]. Biofilm-associated cells also demonstrated altered gene expression profiles, including upregulation of stress-response pathways and efflux systems [56]. Additionally, persister cells, defined as phenotypically dormant subpopulations, were repeatedly detected within biofilms and were associated with survival under high antimicrobial concentrations [57].

Host Immune Evasion and Pathogen Persistence

The reviewed studies documented multiple immune evasion strategies that enable pathogens to persist within host environments despite immune surveillance and antimicrobial treatment [58]. Commonly reported mechanisms included antigenic variation, secretion of immune-modulatory factors, inhibition of complement activation, resistance to oxidative stress, and avoidance of phagocytic clearance [59]. Intracellular survival within host cells was frequently observed among bacterial and fungal pathogens, providing protection from both immune effector mechanisms and antimicrobials with limited intracellular penetration [60]. These immune evasion strategies were associated with prolonged infection duration and repeated exposure to antimicrobial agents across multiple treatment cycles [61].

Environmental Reservoirs and Ecological Amplification of Resistance

Environmental systems were consistently identified as significant reservoirs of antimicrobial

resistance genes and resistant microorganisms [62]. Antibiotic residues originating from hospital effluents, pharmaceutical manufacturing, agricultural runoff, and wastewater treatment facilities were frequently detected in aquatic and soil environments [63]. These residues were associated with selective enrichment of resistant microbial populations and increased prevalence of resistance genes [64]. Studies highlighted extensive horizontal gene transfer among environmental bacteria, contributing to the maintenance and expansion of environmental resistomes [65]. Aquatic and soil ecosystems were reported to act as interfaces linking human, animal, and environmental microbiota, facilitating resistance gene exchange across ecological boundaries [66]. Environmental dissemination was further influenced by factors such as urbanization, intensified agriculture, climate variability, and increased global mobility [67, 68]. A conceptual overview of the integrated mechanisms driving antimicrobial resistance at the host–pathogen interface. The schematic illustrates how genetic mutations, horizontal gene transfer, biofilm formation, host immune pressures, and environmental selective forces collectively shape resistance evolution and persistence. By visually integrating molecular, cellular, and ecological processes, the figure highlights the dynamic and interconnected nature of antimicrobial resistance development across clinical and non-clinical settings, as provided in Figure 1.

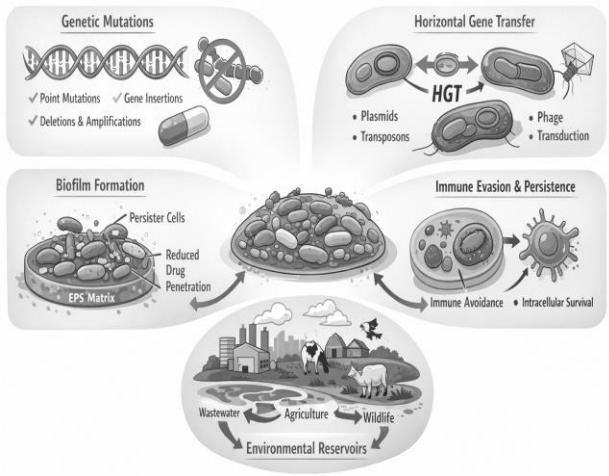

Figure: 1 Integrated mechanisms of antimicrobial resistance evolution at the host–pathogen interface

DISCUSSION

The findings synthesized in this review reinforce the concept that antimicrobial resistance is fundamentally an evolutionary process shaped at the host–pathogen interface, where microbial genetic plasticity, host immune pressures, and environmental selective forces converge to drive resistance emergence and persistence [69–72]. Rather than arising solely as a consequence of antimicrobial misuse, resistance reflects adaptive responses to multifactorial pressures operating across molecular, cellular, and ecological scales. Genetic mutations affecting antimicrobial targets, regulatory pathways, and membrane permeability remain central to resistance development; however, their long-term persistence is frequently enabled by compensatory adaptations that mitigate associated fitness costs, allowing resistant strains to remain competitive even in the absence of antimicrobial exposure[73]. These evolutionary dynamics underscore why resistance often persists despite reductions in antibiotic use. Horizontal gene transfer further amplifies resistance evolution by enabling rapid acquisition of pre-adapted resistance determinants across species and ecological boundaries. The dissemination of resistance genes via plasmids, integrons, and transposons highlights the collective nature of microbial adaptation and challenges traditional species-centered views of resistance evolution[74]. Such mechanisms allow resistance to spread at rates far exceeding those driven by spontaneous mutation alone. Biofilm-associated growth and phenotypic heterogeneity represent another critical dimension of resistance that extends beyond classical genetic mechanisms. The protective extracellular matrix, metabolic stratification, and enrichment of persister subpopulations collectively promote antimicrobial tolerance and treatment failure, particularly in chronic and device-associated infections [75]. These observations emphasize that resistance should be understood not only as a genetic trait but also as an emergent property of microbial community organization. Host immune pressures further shape resistance trajectories by selecting for pathogens capable of immune evasion, intracellular survival, and prolonged persistence. By enabling recurrent or chronic infections, immune evasion strategies indirectly increase antimicrobial exposure and create conditions favorable for resistance selection and fixation [76]. This interplay between immunity and resistance highlights the importance of integrating immunological context into AMR research and therapeutic strategies. Beyond the host, environmental reservoirs play a decisive role in maintaining and amplifying resistance genes. Aquatic and terrestrial ecosystems exposed to antibiotic residues from clinical, agricultural, and industrial sources act as hubs for resistance gene accumulation and exchange, facilitating transmission across human, animal, and environmental microbiomes [62–66,77]. These findings reinforce the necessity of a One Health framework to address resistance as a global ecological phenomenon rather than a purely clinical problem. The integrated mechanisms discussed in this review illustrate that antimicrobial resistance emerges from dynamic, interconnected processes spanning genetic innovation, ecological interaction, and evolutionary selection. Addressing AMR therefore requires systems-level interventions that extend beyond drug development to

include environmental stewardship, infection prevention, and global surveillance strategies informed by evolutionary principles [67,68,78].

CONCLUSION

In conclusion, AMR is not the result of isolated genetic events but an emergent evolutionary outcome of sustained interactions among microbial adaptation, host immune pressure, and environmental selective forces. This review demonstrates that resistance evolution is driven by the convergence of genetic mutations, horizontal gene transfer, biofilm-mediated tolerance, immune evasion, and ecological amplification, operating across clinical and non-clinical settings. Understanding AMR through this integrated host–pathogen interface provides a more accurate framework for interpreting why resistance persists, spreads, and repeatedly undermines therapeutic interventions. Three key conclusions emerge from this synthesis. First, resistance mechanisms function as interconnected networks rather than independent pathways, explaining the limited durability of therapies that target single molecular processes. Second, host immune pressure and chronic infection dynamics play an active role in shaping resistance trajectories, even in the absence of direct antimicrobial exposure. Third, environmental reservoirs act as persistent sources of resistance genes, reinforcing the transboundary and systems-level nature of AMR. Future efforts to combat antimicrobial resistance must move beyond conventional antimicrobial discovery strategies. Priority directions include the development of anti-virulence and host-directed therapies that attenuate pathogenicity without imposing strong selective pressure for resistance, and the design of evolution-aware antimicrobials that account for adaptive trade-offs and resistance stability. In parallel, routine integration of environmental surveillance into AMR monitoring frameworks is essential to identify emerging resistance threats before clinical establishment. Addressing antimicrobial resistance therefore requires coordinated, evolution-informed strategies that integrate molecular insight, host immunity, environmental stewardship, and global policy action. Only through such decisive and system-wide approaches can the long-term effectiveness of antimicrobial therapies be preserved.

Declaration statement

AI-assisted tools were used solely for language refinement and figure conceptualization; no scientific interpretation or data generation was performed by AI.

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Cite this article as:
Nitin Kumar Jumnani, Shafeequllah, Chandra Kumar. Evolution at the Host–Pathogen Interface: Mechanistic Insights into Antimicrobial Resistance. International Journal of Ayurveda and Pharma Research. 2026;14(2):175-181.
https://doi.org/10.47070/ijapr.v14i2.3984
Source of support: Nil, Conflict of interest: None Declared

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