iResearch
2026, Vol. 1, No. 2, pp. 100–107
https://doi.org/10.11648/j.ir.20260102.14

Review Article

Review on Antibiotic Resistance in Veterinary Therapeutics: Mechanisms, Public Health Implications, and Alternative Approaches

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Abstract

Veterinary medicines include many antibiotics and additives used in animal management, breeding and food production. Antimicrobials are the most commonly used type of veterinary medicine in developing countries due to high livestock numbers and infectious diseases. The introduction of penicillin and antibiotics has had a huge impact on improving treatment options, decreasing livestock death and reducing losses. Antibiotic resistance occurs naturally but misuse of antibiotics can cause rapid development of resistant bacteria. Antibiotic resistance develops quickly when antibiotics are used excessively, irrationally, or as growth boosters. When antibiotics are overused in agriculture, toxins build up in food supplies and contaminate the environment. To overcome this problem we need to stop using antibiotics for growth promotion and instead implement sustainable solutions such as improving biosecurity, good animal husbandry, targeted vaccinations and probiotics. We also need to make sure we are using antibiotics responsibly by having monitoring systems and educating farmers. This review examines the processes underlying animal antibiotic resistance, the threats to public health, and alternate approaches to disease prevention.

Keywords

Antibiotics, Antibiotic Resistance, Alternatives, Disease Prevention, Public Health, One Health

1. Introduction

Antibiotics have been proven effective in human, veterinary and companion animals [1]. Antimicrobials play an essential role in managing bacterial pathogens within livestock production, ensuring animal health, welfare and economic viability [2]. Antimicrobials are frequently used to cure, prevent, and treat metaphylaxis. In order to lower the risk of post-operative sepsis, antimicrobials are crucial for treating complex infections in companion animals, such as skin, respiratory, urinary, and wound infections [3]. However, increasing evidence has shown that antibiotics are becoming less effective against major pathogens [4].

Bacterial drug resistance is causing problems for veterinary care, agriculture, and food security [5]. Human antibiotic resistance leads to treatment failure, increased patient morbidity and mortality, extended hospital stays, and increased medical costs. The primary cause of this is antibiotic abuse [6]. Antimicrobial use on a regular basis produces selection pressures that promote the development of resistance mutations. Restricting the use of medically important antimicrobials in

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Received: 20 October 2026; **Accepted:** 31 October 2026; **Published:** 13 November 2026

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food-producing animals is critical to mitigating these selection pressures and reducing resistant bacteria in livestock [7, 8, 10]. Agricultural runoff and extensive animal husbandry are two ways that multi-resistant zoonotic illnesses might spread. To lessen the spread of disease between sectors, a One Health approach that integrates veterinary, environmental, and human health should be employed [9].

Antibiotic resistance is operationally defined as the capacity of bacteria to survive and proliferate in the presence of antimicrobial concentrations that would normally prove bactericidal or bacteriostatic [11]. AMR is defined by the World Health Organization (WHO) as the structural, genetic, or physiological adaptations that microbes develop after being exposed to antimicrobial agents. Microorganisms exhibiting broad multidrug resistance profiles are colloquially designated as "superbugs." Although resistance can arise as a natural evolutionary phenomenon dating back to the initial clinical deployment of penicillin [12, 13], evolutionary selection is immensely accelerated by sub-optimal therapeutic regimens. Importantly, antibiotic resistance does not denote host physiological resistance to a drug; rather, it reflects acquired or intrinsic survival mechanisms within the pathogen population [14].

Surveillance studies demonstrate that major foodborne pathogens in animal production environments encompass both Gram-positive and Gram-negative lineages. Salmonella species, Campylobacter species, and Escherichia coli are some of the most serious drug-resistant bacteria endangering the human food supply chain [15]. Salmonella species are prevalent in networks that raise pigs, chickens, and cattle. Ingestion of infected animal-derived items, such as eggs, raw meat, and unpasteurized dairy, is the usual way for humans to get salmonellosis. Salmonella isolates that are resistant to both single drugs and multiple drugs are prevalent in swine and poultry supply chains, according to epidemiological studies [16].

On the other hand, worldwide analyzes of E. Coli isolates recovered simultaneously from animals, human patients, animal-origin food, and agricultural runoff reveal high rates of resistance to base-line agents like amoxicil-lin and persistent reservoirs of strains that produce Extended-Spectrum Beta-Lactamase (ESBL) in livestock environments [17]. Additionally, the worldwide burden of enteric diseases is largely caused by non-typhoidal Salmonella enterica and Campylobacter species, with the greatest proportional effect concentrated in low- and middle-income nations [18].

Similar clinical issues are raised by gram-positive bacteria. Key repositories for transferable resistance characteristics include enterococcal species, such as Enterococcus faecalis and Enterococcus faecium [19]. Vancomycin-Resistant Enterococci (VRE) have emerged as a serious nosocomial infection on a global scale [20]. The extensive use of glycopeptide growth promoters such avoparcin in agriculture in the late 20th century was a major factor in the historical establishment of VRE in food-producing animals in some areas [21]. In parallel, *Staphylococcus aureus*—particularly Methicillin-Resistant *S. aureus* (MRSA)—continues to represent a major zoonotic and opportunistic pathogen causing complex therapeutic failures in both domestic animals and human populations [22].

Antimicrobial classes such as β-lactams, aminoglycosides and fluoroquinolones are amongst first-line treatment options used in human medicine. They are classed by the WHO as Highest Priority Critically Important Antimicrobials (HPCIAs) [19]. The long-term efficacy of these vital medications may be adversely affected by poor antimicrobial stewardship and abuse. Strict control, including a total prohibition on the use of some antimicrobials, was formerly necessary due to the rapid development of resistance in some antimicrobials, such as widespread resistance to chloramphenicol [14].

In recent years there has been increasing prevalence of multi-drug resistant bacteria (MDRB), making it difficult to treat community and hospital acquired infections. This has caused concern on a global scale that we are entering an antibiotic apocalypse [23]. From an evolutionary standpoint, bacteria with resistance genes are continuously selected for for a variety of reasons, such as:

1) High antimicrobial administration volume and frequency
2) Extended duration of sub-inhibitory treatment regimens
3) Widespread prophylactic and metaphylactic under-dosing; and
4) Unintended environmental exposure resulting from pharmaceutical residues in animal manure, wastewater, and food products [24, 25].

Strict mandated withdrawal periods, which are defined as the necessary amount of time that must pass between the last administration of a veterinary medication and the legal harvesting of meat, milk, or eggs for human use, are enforced by regulatory bodies to safeguard human consumers from chemical exposure [26]. When withdrawal periods are not adhered to, violative pharmaceutical residues enter the human food chain, continuously exerting low-dose selective pressure on the human gut microbiota and promoting AMR selection [2]. Furthermore, agricultural runoff and municipal sewage systems can act as bidirectional vectors, recycling resistant bacteria and active drug residues between human populations, livestock, and surrounding ecosystems [23].

Objectives of this Review Paper:

1) To assess the incidence, molecular processes, and implications for public health of antibiotic resistance in veterinary treatments.
2) To critically examine non-antibiotic alternative strategies for sustainable animal disease prevention and health management.

2. Antimicrobial Resistance in Veterinary Medicine

2.1. Brief History and Overview of Antibiotics in Veterinary Medicine

Veterinary medications are used to maintain animal health, cure or prevent disease, and increase the productivity of animal output. Anti-infectives, antiparasitics, anti-inflammatories, hormones, anesthetics, and feed additives are all included in this category of bioactive substances [27, 28].

It is also possible to employ drinking water for mass medicine. Antibiotic resistance has been evolving with naturally occurring antibiotics for thousands of years, according to analysis of ancient permafrost samples [27]. Microorganisms have developed and been shown to be resistant to every new type of antimicrobials (Table 1) [11, 13, 26]. Treatment options are restricted because microorganisms have evolved effective ways to mutate quickly.

Table 1. *HIstorical timeline of antimicrobial drug release and subsequent detection of clinical resistance in target pathogens.*

Antibiotic Class / Agent	Year Approved / Released	Resistant Pathogen Identified	Year Resistance Identified
Penicillin	1941	Penicillin-resistant Staphylococcus aureus	1942
		Penicillin-resistant *Streptococcus pneumoniae*	1967
		Penicillinase-producing *Neisseria gonorrhoeae*	1976
Vancomycin	1958	Plasmid-mediated vancomycin-resistant *Enterococcus faecium*	1988
		Vancomycin-resistant *Staphylococcus aureus* (VRSA)	2002
Amphotericin B	1959	Amphotericin B-resistant *Candida auris*	2016
Methicillin	1960	Methicillin-resistant *Staphylococcus aureus* (MRSA)	1960
Cephalosporins (3rd Gen)	1980	Extended-Spectrum β-Lactamase (ESBL) *Escherichia coli*	1983
Azithromycin	1980	Azithromycin-resistant *Neisseria gonorrhoeae*	2011
Imipenem	1985	*Klebsiella pneumoniae* carbapenemase (KPC) producer	1996
Ciprofloxacin	1987	Ciprofloxacin-resistant *Neisseria gonorrhoeae*	2007
Fluconazole	1990	Fluconazole-resistant *Candida* spp.	1988
Caspofungin	2001	Caspofungin-resistant *Candida* spp.	2004
Daptomycin	2003	Daptomycin-resistant MRSA	2004
Ceftazidime-avibactam	2015	Ceftazidime-avibactam-resistant KPC *K. pneumoniae*	2015

2.2. Principal Forms of Antibiotic Resistance

Bacterial resistance manifests through multiple evolutionary pathways, categorized into distinct physiological forms:

2.2.1. Natural (Intrinsic) Resistance

The bacterial species has this characteristic. This occurs either because the bacteria lack the treatment's biochemical target or because physical barriers prevent the medication from entering the bacterium. (23).

1) Physical barriers: The outer membrane of gram-negative bacteria acts as a built-in protection against big, hydrophobic substances. This suggests that these bacteria are inherently resistant to vancomycin and other glycopeptides. 17.
2) No target: Because they lack pep-tidoglycan cell walls, organisms without cell walls, such as Mycoplasma and Ureaplasma species, are inherently resistant to beta-lactams. (33).

2.2.2. Acquired Resistance

1) In susceptible bacteria, resistance develops as a result of either gene transfer between species or spontaneous

chromosomal change [23, 29].

2) Chromosomal MutationPoint mutations may occur which can result in changes in the target binding site, alterations to the porins of the outer membrane and up-regulation of efflux pumps. Mutant resistant cells will survive when resistance is chosen for, whereas susceptible cells would perish [30].
3) HGT, or horizontal gene transfer Resistant genes can be transferred from other bacteria to susceptible strains by conjugation (plasmid transfer), transduction (bac-teriophage), or transformation (naked DNA) [29, 30].

2.2.3. Extrachromosomal Resistance

Extrachromosomal resistance is mediated by mobile genetic elements (MGEs) that exist independently of the bacterial chromosome [31].

1) Plasmids: Circular double-stranded DNA molecules that replicate themselves and often include several drug resistance cassettes. Conjugative plasmids provide quick transmission both within and across species.
2) Transposons and Integrons: Transposons (sometimes called "jumping genes") facilitate the transfer of resistance determinants from plasmids to the host chromosome. Integrons are crucial platforms for multi-resistance assembly because they capture, express, and mobilize gene cassettes [31].

2.2.4. Cross-Resistance

Cross-resistance occurs when a single resistance mechanism confers tolerance to multiple antimicrobial agents sharing structural features, chemical classes, or target pathways [23, 29, 30]. Examples include cross-resistance between macrolides, lincosamides, and streptogramins (MLS group) via target methylation, or cross-tolerance across penicillins and cephalosporins via extended-spectrum beta-lactamase activity.

2.3. Multidrug and Advanced Resistance Profiles

Integrons play a crucial role in multi-resistance construction by capturing, expressing, and mobilizing gene cassettes [31].

1) *Multidrug-Resistant (MDR):* Bacteria demonstrating non-susceptibility to at least one agent in three or more antimicrobial categories.
2) *Extensively Drug-Resistant (XDR):* Bacteria remaining susceptible to only one or two antimicrobial categories (non-susceptible to at least one agent in all but two or fewer categories).
3) *Pandrug-Resistant (PDR):* Bacteria exhibiting non-susceptibility to all clinically available agents across all antimicrobial classes.

3. Molecular Mechanisms of Antibiotic Resistance

Target site modification, enzymatic inactivation, membrane permeability reduction/active efflux, and metabolic pathway by-pass are the four main molecular mechanisms used by bacteria to counteract antimicrobial drugs (Figure 1).

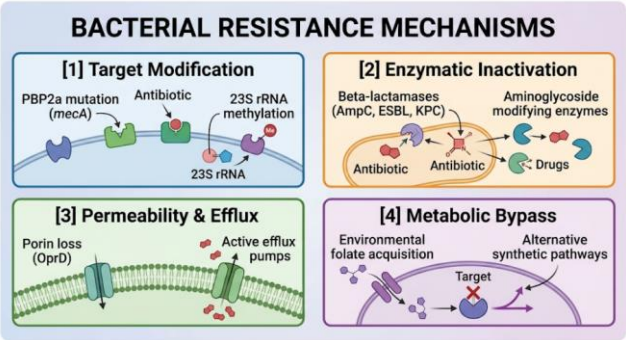


Figure 1. *SChematic overview of primary molecular resistance mechanisms in pathogenic bacteria.*

3.1. Modification of Target Sites

Conformational changes in target proteins or structures hinder drug binding without destroying essential physiological function [23].

1) *Ribosomal Mutations:* Alterations in ribosomal RNA or associated proteins impair macrolide, aminoglycoside, and tetracycline binding.
2) Penicillin-Binding Protein (PBP) Modifications: S encodes PBP2a, a low-affinity transpeptidase that provides resistance to almost all beta-lactam antibiotics. aureus acquires the mecA gene [31]. Penicillin insensitivity is also caused by PBP2b mutations in Streptococcus pneumoniae.

3.2. Enzymatic Inactivation and Degradation

To combat antibiotics, some resistance enzymes have developed in bacterial species [32].

1) *Beta-Lactamases:* enzymes that break down the amide link of the beta-lactam ring by hydrolysis.
2) *Aminoglycoside-modifying enzymes (AMEs):* Enzymes including acetyltransferases (AAC), phosphotransferases (APH) and nucleotidyltransferases (ANT) that chemically modify aminoglycosides making them unable to bind to ribosomes.
3) *Chloramphenicol acetyl transferases (CAT):* enzymes that help acetylate chloramphenicol, preventing it from attaching to the ribosome's 50S subunit.

3.3. Decreased Permeability and Efflux Pump Mechanisms

Antibiotic concentrations are kept sub-lethal by limiting their entrance into the cell [23, 27].

1) *Porin downregulation:* When outer membrane porin channels are lost or mutated, carbapenem entrance is prevented. For instance, Pseudomonas aeruginosa has lost its OprD channels.
2) *Transport proteins:* that actively remove different kinds of antibiotics from the cytoplasm are known as efflux pump systems. The Resistance-Nodulation-Division family (RND), key Facilitator Superfamily (MFS), and ATP-Binding Cassette (ABC) are some of the key families.

3.4. Utilization of Alternative Metabolic Pathways

Pathogens bypass antibiotic-inhibited pathways by acquiring alternative synthetic enzymes or utilizing exogenous metabolites [33]. For instance, resistance to sulfonamides and trimethoprim can occur when bacteria acquire mutated dihydropteroate synthase (DHPS) or dihydrofolate reductase (DHFR) enzymes, or absorb preformed folic acid directly from the environment.

3.5. Specific Resistance Mechanisms by Antibiotic Class

3.5.1. Beta-Lactam Class (Penicillins, Cephalosporins, Carbapenems)

Resistance is predominantly mediated by β-lactamase enzymes, divided structurally into Ambler Classes A through D [31, 32]:

1) *Class A:* Serine-based enzymes, including TEM, SHV, and KPC carbapenemas. Frequently plasmid-encoded.
2) *Class B:* Zinc-dependent Metallo-beta-lactamases (MBLs, e.g., NDM, VIM, IMP) capable of hydrolyzing all beta-lactams except monobactams.
3) *Class C:* Chromosomally encoded AmpC enzymes, resistant to clavulanic acid inhibition.
4) *Class D:* Oxacillinases (OXA-type) predominantly identified in *Staphylococcus* and *Acinetobacter* species.

3.5.2. Aminoglycoside

ClassResistance is mainly due to plasmid borne aminoglycoside modifying enzymes (AMEs) however other mechanisms include expression of 16S rRNA methyltransferases leading to pan-aminoglycoside resistance and mutation in 30S ribosomal subunit genes [31, 32].

3.5.3. Tetracycline Class

Mechanisms of resistance include efflux pumps (encoded by 'tet(A)-tet(E)' genes) and ribosomal protection (by tet(M), tet(O) and tet(Q)) which removes tetracycline from 30S subunit [14, 32].

3.5.4. Macrolides, Lincosamide and Streptogramins (MLS Class)

Resistance occurs through methylation of the target by erm (Erythromycin Ribosome Methylation) genes, efflux pumps (msr) and mef genes) and enzymatic degradation by esterase [11, 33].

3.5.5. Quinolones and Fluoroquinolones:

Quinolone Resistant Determinant Regions (QRDR) are mutated at specific points within the target genes (gyrA encoding DNA gyrase and parC encoding topoisomerase IV) leading to resistance. Additional resistance can occur through the presence of Plasmid Mediated Quinolone Resistance genes (qnr) [11, 31].

4. Public Health Implications of Antimicrobial Resistance in Veterinary Medicine

4.1. Transmission of Zoonotic Pathogens

Resistant veterinary pathogens can be transmitted to humans in a number of ways (Figure 2):

1) Direct contact from farm workers, vets and slaughterhouse workers.
2) Ingestion of contaminated meat, milk or egg or through cross contamination when cooking.
3) Through agricultural wash off, manuring of crops and contamination of ground water.

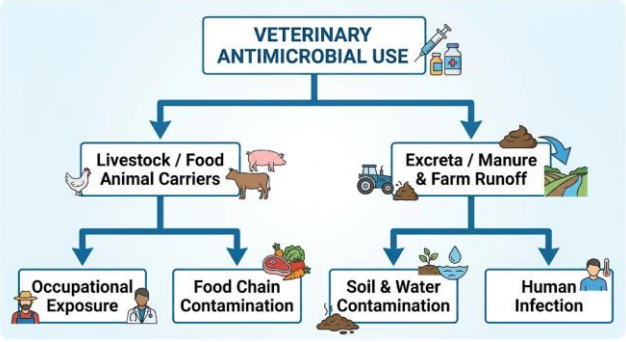


Figure 2. *PAthways of AMR dissemination from veterinary settings to public environments and human populations.*

4.2. Impact of Antibiotic Residues in the Food Chain

Failure to observe withdrawal periods will result in chemical residues. Constant exposure to sub-therapeutic levels can cause hypersensitivity reactions in susceptible individuals. This can cause changes to the microbial balance in the gut, and also cause selection pressures for resistant mutants in the gastrointestinal tract of humans [2, 26].

5. Alternative Solutions

To reduce veterinary AMR it is necessary to move away from using large amounts of therapeutics.

5.1. Vaccine/Immunisation

Immunisation programs are effective at reducing disease and thus removing the need for use of antimicrobials. Viral and bacterial diseases such as Newcastle disease, contagious bovine pleuropneumonia and salmonella can be vaccinated against en masse.

5.2. Probiotics, Prebiotics and Synbiotics

1) *Probiotics* - live microbes (Lactobacillus, Bifido-bacterium, Bacillus spp). These microbes will out-compete pathogens, produce bacteriocin and regulate mucosal immunity.
2) *Prebiotics* - non-digestible oligosaccharides which stimulate the growth of beneficial microbes within the gut.
3) *Synbiotics* - combination of probiotics and prebiotics. This will improve gut health and reduce reliance on prophylactic antibiotics within feeds.

5.3. Biosecurity and Farm Management

Implementing strict biosecurity measures on farms will significantly reduce the risk of pathogens entering and spreading.

These include; Strict cleaning, sanitation and disinfecting practices, All-in all-out stocking, Correct stocking densities, ventilation and feeding, andStrict quarantine regimes.

5.4. Antimicrobial Stewardship

Implementation of good management will help to reduce AMR sustainably;

1) *Use only when necessary* - HPCIAs should not be used outside of clinical situations where testing has shown there are no alternatives.
2) *Remove growth promoters* - banning medically important antimicrobials.
3) *Monitoring* - constant monitoring of resistance and usage.
4) *Training* - training livestock producers and technicians to administer drugs sensibly observe withdrawal periods and manage diseases preventatively.

6. Conclusions and Recommendations

6.1. Conclusions

Antimicrobial resistance in veterinary medicine is a complex issue that can have implications for animal production, food security and human public health. Whilst antibiotic resistance develops naturally through evolution, human related factors such as misuse of antimicrobials, constant sub-therapeutic dosing, promotion of growth and not adhering to withdrawal periods have increased and spread this globally. Bacteria utilise various methods to overcome these drugs; including modifying the drug target, enzymatic breakdown, efflux and metabolic bypass. These often occur on mobile genetic elements which can be transferred between species. Overall it is important we move away from chemical prophylaxis and focus on management strategies, good biosecurity and surveillance.

6.2. Recommendations

1) Enforce strict regulatory controls on the over the counter sale of veterinary antimicro-bials. Prohibit the use of medically important antibiotics for growth promotion.
2) Encourage the use of alternative non-antibiotic methods including vaccines, probiotics, pre-biotics and phytogenic additives.
3) Promote one health surveillance systems to incorporate veterinary clinical data, food safety data, human health data and environmental data.
4) Improve biosecurity measures by supporting extension services to assist farmers in improving sanitation, stocking and nutrition.
5) Educate veterinary personnel, animal health technicians and livestock personnel on the importance of rational prescribing, evidence based treatment and adherence to withdrawal periods.

Abbreviations

AMR	Antimicrobial Resistance
AME	Aminoglycoside-Modifying Enzyme
AR	Antibiotic Resistance
ESBL	Extended-Spectrum Beta-Lactamase
FDA	Food and Drug Administration
HGT	Horizontal Gene Transfer
HPCIA	Highest Priority Critically Important Antimicrobial
MDR	Multidrug-Resistant
MDRB	Multidrug-Resistant Bacteria
MGE	Mobile Genetic Element

MRSA	Methicillin-Resistant Staphylococcus Aureus
PBP	Penicillin-Binding Protein
PDR	Pandrug-Resistant
QRDR	Quinolone Resistance-Determining Region
VRE	Vancomycin-Resistant Enterococci
WHO	World Health Organization
XDR	Extensively Drug-Resistant

Acknowledgments

The author acknowledges the North Shewa Zone Livestock Resource Office and Debre Berhan University for providing academic and institutional support during the preparation of this review.

Author Contributions

Kurabachew Gebrehiwot: Conceptualization, Data curation, Investigation, Methodology, Visualization, Writing – original draft, Writing – review & editing

Conflicts of Interest

The author declares no conflicts of interest.

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Biography



Kurabachew Gebrehiwot was born on 10 December 1977 in Menz Keya district of North Shoa, Amhara Region. He completed his elementary and junior education at Zemero Elementary and Junior School, and his secondary studies at Mehal-Meda Senior Comprehensive Secondary School. In 2002 he joined Alage ATVET College and graduated in 2005 with a diploma in Animal Health. Soon after, he began his professional career with the Amhara Region Bureau of Agriculture, serving from 2005 to 2009 as a field veterinarian in Asagirt District, North Shoa Administrative Zone. Committed to advancing his expertise; he enrolled at Wollo University in 2010 and earned a Bachelor of Veterinary Science (BVSc) degree in 2012. Since then, he has worked with the North Shoa Zone Agricultural Office and later the North Shoa Zone Livestock Office, taking on expert and supervisory roles and providing technical support across all districts of the zone. He enrolled at DebreBerhan University, College of Graduate Studies to pursue specialization in veterinary public health.