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Review Article

Review on Antibiotic Resistance in Veterinary Clinical Practice

Merete Mekuriya Detamo[1*] and Natinael Dawit Kalacho[2]

[1]*School of Veterinary Medicine, Wolaita Sodo University, Ethiopia*

[2]*Jinka University, Department of Veterinary Medicine, Ethiopia*

Abstract

Veterinary pharmaceuticals include a wide range of anti-infective and additives in the use for animal health, nutrition, reproduction, and productivity. Antimicrobials are among the most extensively used drugs in developing countries largely due to large population of livestock and the burden of infectious diseases. The introduction of penicillin in 1943 and other antibiotics thereafter provided remedies for many infections in humans and animals, reducing mortality and productivity losses. Since then, a repertoire of antibiotics and antimicrobials has been introduced as chemotherapeutics and/or prophylaxis. This success notwithstanding, many pathogens of consequences are no longer susceptible owing to emergence of antibiotic resistant (ABR) microorganisms. This has made treatment of infectious diseases less effective. Beside spontaneous emergence of mutant microorganisms, scientists are wary of ABR caused by intensive use of antibiotics in humans and animals, sometimes in sub therapeutic doses as preventive medicine. In developing countries, environmental exposure and persistent use of antibiotics in food animals may leave residues in the food chain. In addition to that, the consequences include development of antibiotic resistance are occurred. Alternatives to growth-promoting and prophylactic uses of antimicrobials in agriculture include improved management practices, wider use of vaccines, and introduction of probiotics. Monitoring programs, prudent use guidelines, and educational campaigns provide approaches to minimize the further development of antimicrobial resistance. In this seminar, antibiotic use in veterinary medicine and sequel in the emergence of ABR and alternative management have reviewed.

Keywords: Antibiotics; Antibiotic resistance; Alternatives; Diseases prevention

***Corresponding author:** Dr. Merete Mekuriya Detamo, School of Veterinary Medicine, Wolaita Sodo University, Ethiopia. E-mail: maremekuria2012@gmail.com

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Introduction

The therapeutic benefits of antibiotics to humans, livestock and companion animals are well recognized [1]. In livestock, antibiotics are essential in the therapeutic treatment of bacterial diseases that impact livestock health, production and welfare [2]. Antibiotics are also used for prophylaxis and metaphylaxis, to maintain animal health and increase productivity. In companion animals, antimicrobials are essential for therapeutic treatment of skin, wound, respiratory and urinary tract infections, as well as for reducing the frequency of sepsis and surgical site infections [3]. However, despite the huge successes recorded against bacteria, recent trends have shown a decline in the ability of antibiotics to control pathogenic bacteria [4].

Bacteria are becoming increasingly resistant to antibiotics, posing potential risks to veterinary health, welfare, food and feed production systems [5], and in humans, resulting in treatment failures and leading to increased morbidity, higher medical costs; prolonged hospital stays for human cases and increased mortality. Antibiotic resistance is accelerated by the overuse and misuse of antibiotics [6]. The use of antibiotics exerts selection pressure on microbes, allowing resistant pathogens to proliferate and leading to the emergence of antimicrobial resistance (AMR). The emergence of AMR in humans has been linked with AMR in animals and the environment [7-10].

Antibiotic resistance has been described as the ability of bacterial to survive and spread despite treatment with specific and combination therapy that are normally used against them [11]. The World Health Organization also emphasized that resistance happens when microorganisms change when they are exposed to antibiotics drugs. Some microorganisms that develop antibiotics resistance are sometimes referred to as “superbugs”. Antimicrobial resistance may be spontaneous and occur as a natural process, and resistance to antimicrobials dates back as far as when the first generations of antibiotics including penicillin were introduced in 1943/44 by Alexander Fleming [12,13]. Antibiotic resistance happens when germs like bacteria and fungi develop the ability to defeat the drugs designed to kill them. That means the germs are not killed and continue to grow. Infections caused by antibiotic-resistant germs are difficult, and sometimes impossible, to treat. Antibiotic resistance does not mean the body is becoming resistant to antibiotics; it is that bacteria have become resistant to the antibiotics designed to kill them [14].

The current study indicated that resistant pathogens in food-producing animals included both gram-positive and negative ones. The most common drug-resistant foodborne bacteria of relevance to human health were Salmonella, Campylobacter, and E. coli [15]. Salmonella bacteria are prevalent in food animals such as poultry, pigs, and cattle. Salmonellosis affects humans through the consumption of contaminated food of animal origin (mainly eggs, meat, poultry, and milk). A recent study in Brazil investigated the occurrence of resistance in Salmonella spp., isolated from products and raw material of animal origin (swine and poultry) to antimicrobials found that 51 (38%) out of 134 isolates were resistant to at least one of the eight antibiotics used, and 28 (55%) of resistant isolates were multi-resistant [16].

A recently published systematic review on the prevalence of antibiotic resistance in E. coli strains simultaneously isolated from humans, animals, food, and the environment indicated that colistin had the lowest prevalence and amoxicillin the highest in isolated human E. coli strains [17]. The systematic review also indicated that the prevalence of Extended-Spectrum Beta-Lactamase (ESBL)-producing E. coli was highest in animals compared to human or environmental/food isolates. A study of the global and regional burden of 22 foodborne diseases indicated that the leading cause of the foodborne illness was norovirus followed by campylobacter. The diarrheal and invasive infections caused by non-typhoidal Salmonella enterica infections caused the largest burden of disease. The authors found that the burden of foodborne illness was highest in WHO's African region [18].

Enterococci such as Enterococcus faecalis and Enterococcus faecium were reported in the current study and had been reported to have intrinsic and acquired resistance to a wide range of antibiotics including vancomycin [19]. Currently, vancomycin-resistant enterococcus (VRE) is a challenge in clinical settings [20]. The emergence of VREs in food-producing animals was attributed to the widespread use of avoparcin in the 1990s in Europe for growth-promotion in animals [21]. In North America, the emergence of VRE in animals was not seen until 2008 and was attributed to the extensive use of vancomycin in clinical settings. Staphylococcus aureus is another gram-positive opportunistic pathogen in animals and harbors several AMR genes [22].

The current study listed B-lactams, aminoglycosides, and Quinolones/fluoroquinolones as the most commonly encountered antibiotics drug classes in the retrieved literature. These drug classes are important therapeutic choices in human health. These drugs were listed as critically important drugs in human medicine [19]. The misuse/overuse of these drug classes threatens the efficacy and safety of antibiotics in clinical use and governmental action is needed. The fast development of chloramphenicol resistance upon use in animals led the FDA to ban the use of chloramphenicol in food-producing animals [14].

In the last two decades, the growing problem of multidrug-resistant bacteria (MDRB) has made the routine therapy of some infections resulting from treatment in a hospital or healthcare unit, i.e. nosocomial infections, complicated and in few cases, impossible. The widespread nature of the problem has led some experts to speculate about a 'post antibiotic era' [23].

In evolution, selection pressure is bound to cause subpopulation of microorganism with resistance genes to emerge. This selective pressure has been ascribed to appropriate and inappropriate use of antibiotics but aggravated by (1) intensity of usage, (2) persistence of usage, (3) under usage and sub therapeutic doses that animals are exposed to in prophylactic treatment, and (4) unintended animals exposure through antimicrobials in food residues and the environment [24,25].

Veterinary practices use drugs for mitigating these diseases in animals, including food animals that have to be maintained in health and productivity (meat, egg, and milk). To prevent these drugs from getting into the food chain and being consumed by humans, "withdrawal time," which is the last time any drug may be administered before egg/milk and meat from such animals are collected and consumed is specified [26].

The withdrawal time for antimicrobials is intended to prevent harmful drug residues in meat, milk, and eggs. These waiting periods need to be observed from the time of treatment to when the animals are slaughtered for food. This is important because food products that contain antimicrobial residues not metabolized leaves residues beyond permissible limits at the end of the withdrawal period may be considered unwholesome for consumption and may contribute to antimicrobial resistance in humans [2].

Veterinary pharmaceuticals, therefore, contribute in many ways to the emergence of antimicrobial resistance either directly in suboptimal usage in animals or indirectly in human who consume sub therapeutic doses in animal products. When resistant organism emerges, it has also been argued that human sources also seed these resistant bacteria to animals and the environment through sewage [23].

Therefore, objectives of this senior seminar are:

- To review the antibiotic resistance in veterinary practice.
- To review the alternatives to antibiotics for diseases prevention.

Antibiotics Resistance

Brief history of antibiotics resistance

Veterinary pharmaceuticals include drugs, medications, and other substances in use to treat or prevent animal diseases for health, growth promotion, and productivity. These drugs can be broadly divided into categories according to the different pathogens or targeted infections. They include antibiotics, anti-parasitic drugs, anti-inflammatory, reproductive medication, surgical medications, anesthetics, nutritional drugs, and feed additives sometimes used as growth promoters. Among commonly used drug in veterinary medicine are antibiotics. These drugs and medicaments can be administered in form of injectable, tablet, bolus, drench, and bath/wash or added to feed and drinking water [27,28].

Antibiotic resistance is an ancient, biological phenomenon. A growing body of evidence shows that antibiotic resistance evolved alongside antibiotic production in the natural environment. Long before the introduction of modern antibiotics, bacteria developed mechanisms to survive the effects of natural antibiotics produced by bacteria and fungi in the environment. Resistance genes have been detected in 30,000-year-old ancient Alaskan soil sediment samples, which researchers say "firmly establishes that antibiotic resistance genes predate our use of antibiotics and offers the first direct evidence that antibiotic resistance is an ancient, naturally occurring phenomenon widespread in the environment" [27].

Alexander Fleming discovered penicillin, the first commercialized antibiotic, in 1928. Ever since, there has been discovery and acknowledgement of resistance alongside the discovery of new antibiotics. In fact, germs will always look for ways to survive and resist new drugs. More and more, germs are sharing their resistance with one another, making it harder for us to keep up [11-13].

Bacterial resistance is the capability of bacterial cells to prevent antibiotic bacteriostatic or bactericidal effects. The excessive and unintended usage of antibiotics contributes to resistance development in bacteria. Because of the extensive uptake, the evolvements of microorganisms resistant with the time and problems have arisen with these resistant microorganisms for the treatment of certain infections. Nowadays, resistance is determining as a big issue in the path of new drug

synthesis, developing antibiotic resistance is a major public health problem worldwide (Table 1) [26].

Antibiotic Approved or Released	Year released	Resistant Germ Identified	Year Identified
Penicillin	1941	Penicillin-resistant Staphylococcusaureus	1942
		Penicillin-resistant Streptococcus neumonia	1967
		Penicillinase-producing Neisseria gonorrhoeae	1976
Vancomycin	1958	Plasmid-mediated vancomycin resistant Enterococcus faecium	1988
		Vancomycin-resistant Staph.aureus	2002
Amphotericin B	1959	Amphotericin B-resistant Candida auris	2016
Methicillin	1960	Methicillin-resistant Staphylococcus aureus	1960
Extendedspectrum	1980	Extended-spectrum beta-lactamase producing Escherichia coli	1983
Cephalosporins			
Azithromycin	1980	Azithromycin-resistant Neisseria gonorrhoeae	2011
Imipenem	1985	Klebsiella neumonia carbapenemase (KPC)-producing Klebsiella pneumoniae	1996
Ciprofloxacin	1987	Ciprofloxacin-resistant Neisseriagonorrhoeae	2007
Fluconazole	1990	Fluconazole-resistant Candida	1988
Caspofungin	2001	Caspofungin-resistant Candida	2004
Daptomycin	2003	Daptomycin-resistant methicillin resistant Staphylococcusaureus	2004
Ceftazidimeavibactam	2015	Ceftazidime-avibactam-resistant KPC-producing Klebsiella pneumoniae	2015

Table 1: Example of some pathogens showing resistance over time.

Principal forms of antibiotic resistance

The four principal forms of antibiotic resistance evolve as

Natural resistance (Intrinsic, Structural): In this type of resistance, the usage of antibiotics is not associated with the resistance but it caused by the bacteria's structural properties. This occurs as a result of intrinsic resistance, or microorganism which doesn't follow the target antibiotic structure, or antibiotics which due to its Characteristics do not encounter its target. Intrinsic resistance is the natural ability of bacteria to resist the effects of an antibiotic due to inherent structural or biochemical features of the bacteria e.g. if an antibiotic is too large to cross the cell wall of a bacteria and get access to its target within the cell [23]. Knowing which bacteria were never susceptible or have a natural resistance to particular antibiotics helps vets and clinicians to prescribe appropriate and effective treatments. Gram-negative bacteria and vancomycin, for example, vancomycin antibiotics do not move through the outer membrane so that these Gram-negative bacteria are naturally insusceptible to vancomycin. Likewise, L-form bacteria that are cell wall-less types of the bacteria, such a Urea plasma and Mycoplasma Mycoplasma that are naturally owning beta-lactam antibiotics resistance (Dar et al., 2016).

Acquired resistance: Resistance to antibiotics can also be acquired either from the result of mutation of bacterial genes involved in normal physiological processes and cellular structures. These genetic mutations are changes in the DNA sequence of bacteria that occur continuously, to varying degrees. Bacteria can also become resistant through the acquisition of foreign DNA originally from other bacteria, e.g. via plasmids (small circular DNA strands), in a process called Horizontal Gene Transfer (HGT) [23,29].

Sometimes these genetic mutations or the acquisition of foreign resistance genes can lead to the emergence of bacteria with an improved ability to survive treatment with particular antibiotics. If those bacteria are then exposed to these antibiotics, they increase in numbers, while the more susceptible bacteria are killed off. Bacterial population changes are inevitable without the careful management of antibiotics used against them and, as explained above, will occur more quickly if antibiotics are not taken in accordance with their prescribing instructions [23,29,30].

Regardless of resistance development due to alteration in the genetic features of bacteria, an acquired because it is not affected by the antibiotics it was previously susceptible to it. This form of resistance comes from the main chromosome or extra chromosome structures (plasmids, transposons, etc.). Chromosomal resistance results from mutations that change randomly bacterial chromosome, these mutations can occur by certain physical and chemical factors. This may be due to changes in the composition of bacterial cells, so that may be decreased bacterial drug permeability, or may be changes to the drug's target in the cell. Streptomycin, aminoglycosides, erythromycin, and lincomycin can develop resistance to these forms [30].

Extrachromosomal resistance relies on extrachromosomal genetic materials that can be transmitted via plasmids, transposons, and integrons. Plasmids are segments of DNA that a replication dependently of chromosomal DNA. A plasmid is typically responsible for the development of antibiotic inactive enzymes. There are main forms of holding genetic material (resistance genes and plasmids) from bacterial cells, this form are transduction, transformation, conjugation, and mechanism of transposition. The genes with antibiotic resistance on the chromosome or plasmid are intertwined and are situated at the beginning with different integration groups, or integrons. Recombination is very normal in integrons [31].

Cross-resistance: It is mean the resistance to a specific antibiotic by specific microorganisms, that work with the identical or related mechanisms and that are also resistant to other antibiotics. This is generally seen when antibiotics have common structures: such as resistance to erythromycin, neomycin kanamycin, or resistance to cephalosporins and penicillins. However, cross-resistance can be sometimes seen in acompletely distinct group of drugs as well, like a cross-resistance that exists amongst erythromycin-lincomycin, this resistance might be the chromosomal origin or not [23,29,30].

Multi-drug and other types of resistance: Multidrug-resistant species are typically pathogens that have been resistant to their antibiotics, this ensures that the bacteria will no longer be eliminated or regulated by a single drug [29]. Inappropriate utilization of antibiotics for treatment culminated in the introduction of multidrug resistant pathogenic bacteria. Either of the two mechanisms can induce multidrug resistance in bacteria. Firstly, these bacteria will acquire several genes; each coding for specific drug resistance, this

form of resistance usually exists on R-plasmids. Secondly, the form of multidrug resistance may also occurby enhanced gene expression encoding for efflux pumps, enzymatic inactivation for antibiotics, changes in target structure, and others [23,29].

If the bacterial strains are not susceptible to three or more antimicrobial types, they are called multidrug-resistant (MDR) bacteria. If the species resistant to all but one or two classes of antibiotics are deemed highly resistant to medicines, whether the species resistant to all usable antibiotics are known as pan-drug resistant. For example, Acinetobacter species with multidrug resistance (MDR) can be identified as the bacteria that having the resistant ability to at least three groups of antibiotics classes, for example for all penicillin and cephalosporin, aminoglycosides and quinolones groups. Extensive Acinetobacter spp., drug-resistant (XDR), isolate resistant to the three types of antibiotics classes mentioned above in (MDR), and even carbapenem-resistant, *Acinetobacter spp.*, Pandrug resistant, or pan-resistant (PDR), these bacteria can be going to be the XDR as well as polymyxin-resistant and tigecycline resistance [23,29,30].

Mechanisms of antibiotics resistance

Modifications drug target: Modifications that happen in the drug-related receptor and the location of the target regions of the relation with the antibiotics are distinct; these can be complex enzymes and ribosomes. The most frequently identified resistance consistent with variations in the ribosomal target is in macrolide antibiotics. The most popular examples here are the evolvement of penicillin resistance due to the mutations of penicillin-binding proteins beta-lactamase enzymes in Staphylococcus aureus, Streptococcus pneumoniae, Neisseria meningitides, and Enterococcus faecium strains [23].

Enzymatic inactivation of antibiotics: Most of the bacteria synthesize antibiotic degrading enzymes; the enzymatic inactivation mechanism is one ofthe most important antibiotics resistance mechanisms.In this group, beta-lactamases, aminoglycosidase, chloramphenicol, and erythromycin modifying enzymes arethe most popular examples [32].

Reduction of the inner and outer membrane permeability: This mechanism results from changes in the permeability of the internal and external membrane so that decreased drug uptake into the cell or rapidly ejected from the pump systems [23].

Due to a decrease in membrane, permeability because of porin mutations that may occur in proteins of resistant strains for example; a mutation in specific porins called OprD can cause resistance to carbapenem in Pseudomonas aeruginosa strain. Reduction in outer membrane permeability can play an important role in quinolone resistance and aminoglycoside resistance [27].

Active pumps system: Resistance develops most commonly in the tetracycline group of antibiotics via the active pump systems. With an energy-dependent active pumping system, tetracyclines are thrown out and cannot concentrate within the cell. This mechanism of resistance is in plasmid and chromosomal control. Active pumping systems for example are effective in resisting quinolones, 14-membered macrolides, chloramphenicol and beta-lactams [27,32].

Using an alternative metabolic pathway: Unlike some of the target alterations in bacteria, the latest drug-susceptible pathway eliminates the need for objective development. Bacteria can prepare folic acid from the environment, rather than synthesizing folic acid so that it becomes resistant among sulfonamide and trimethoprim [33].

Resistance by Antibiotics group Mechanisms

Beta-lactams resistance: Antibiotics of beta-lactam are a wide class of antibiotics, including penicillins, cephalosporins, monobactams, and carbapenems. Synthesis of beta-lactamase enzymes is the most common resistance mechanism here [31,32].

Beta-lactamase Enzymes: At the molecular level, there are 4 groups (A, B, C, D) of beta-lactamase enzymes. Beta-lactamases A, C, and D that deferent from B-class that function cool ester enzymes mediated, while the latest was need zinc ion as metallo enzyme [31,32].

i. **Beta-lactamases Class A:** these resistances occur in both Gram-positive and Gram-negative bacteria and mostly mediated by plasmid or transposon. This group includes the gram-negative bacteria primarily occurs in E. coli and Klebsiella pneumoniae [31,32].

ii. **Beta-lactamases Class B:** bacteroides fragilis, observable species of Aeromonas andLegionella, enzymes that hydrolyze carbapenems, penicillin, and cephalosporins [31,32].

iii. **Beta-lactamases Class C:** generally, seen in Gram-negative bacteria and chromoso melocalized (Group I, AmpC, etc.). This resistance mechanism is not inhibited by clavulanic acid and has aninducible characteristic so produced in high levels in thepresence of beta-lactam antibiotics. Often known as Inducible Beta-Lactamases (IBL), they found in Enterobactercloacae, Citrobacter freundii, Serratiamarcescens, and P. aeruginosa [31,32].

iv. **Beta-lactamases Class D:** these enzymes are induced by beta-lactamases antibioticsand pro duced in Gram-positive cocci such as Staphylococcus aureus so that degrade Oxacillin [31,32].

Modifications in Penicillin-Binding Proteins (PBP): Penicillin-binding proteins (PBP) in peptidoglycan synthesis in the bacteria responsible for the Antibiotic target of beta-lactam, Carboxypeptidase PBPs, and the enzymes of bacteria, due to changes in it, resistance results in. Methicillin-resistant S. aureus (MRSA) is willing to take responsibility for methicillin resistance in strains, mecA gene, this gene results in PBP-2a synthesis enhancing betalactam antibiotic resistance. The modifications in S. pneumoniae in PBP 2b are responsible for the resistance to penicillin and cephalosporin [31].

Modifications in proteins of the membrane: Change in the porin channels in gram-negative bacteria, for example, P. aeruginosa with a devoted channel protein registered in OprD may evolve carbapenem resistance. Antibiotic accumulation can be prevented in the active pump systems cell. Consequently, the group of beta-lactams, tetracyclines, chloramphenicol, and quinolones can lead to resistance [14,26].

Antibiotics resistance of aminoglycoside group

Aminoglycosides modifying enzymes: The most important mechanism for the emergence of resistance to aminoglycosides in aerobic gram-negative bacteria is enzymatic inactivation. Enzyme modifying has amajor role in resistance to aminoglycosides. These enzymes are often of plasmid or transposon origin, there are acetyl transferase and phosphotransferase in this group. Modified enzymes are responsible for the high extent of gentamicin resistance in enterococci [31, 32].

Ribosomal target modifications: This approach is crucial in Streptomycin resistance, the target of streptomycin is not connected to the ribosomal 30S subunit due to mutations in the ribosomal 30S, inenterococci, this kind of resistance to streptomycin isessential [31, 32].

Tetracyclines resistance

Prevention of the absorption of drugs into cells and active pump systems: Reduction of membrane permeability resulting from spontaneous chromosome mutations in bacteria because of resistance development to prevent drug uptake. The organisms also can develop tetracycline resistance depending on active pump systems [32].

Protection of ribosome: The second significant mechanism that leads totetracycline resistance, withtetM, tetO, tetQ, tetS genes inhibit drug activity by modifying a cytoplasmic ribosome that binds to the tetracycline. These genes have been found in many genera like Campylobacter, Mycoplasma, Urea plasma, and Bacteroides, for example. They are plasmid and chromosome origin [14,32].

Macrolide, lincosamide, streptogramins (MLS) groups resistance

Gram-negative bacteria are naturally resistant to MLS group antibiotics.

Ribosomal target modification: This mechanism is most common in Gram-positive bacteria, in the 50S ribosomal subunit, this is connected to the drug with the 23S of the ribosome in rRNA-specific methylation of an adenine molecule has a structural change and reduces the drug's binding to ribosomal RNA. The resistance is of a structural or inducible type [11,33].

Inactivation of drug by enzymatic activity: The bacterial cells having enzymes that play a critical role in resistance like Erythromycin and other Macrolides resistance.

Chloramphenicol resistance: The inactivation of the chloramphenicol acetyltransferase (CAT) by enzymes that acetylate the chloramphenicol antibiotic leads to resistance in bacteria produced by this enzyme. Reduced drug uptake in certain bacteria especially gram-negative can also be responsible for chloramphenicol resistance [11,31].

Quinolones resistance

There are different mechanisms for quinolone resistance that including;

Mutation modification of the target topoisomerase: Modifications in the target enzymes topoisomerases caused mainly by mutations that reduce the affinity of quinolones without compromising the enzyme function are the most common mechanism of acquired quinolone resistance and have already been reported in several bacterial species. Resistance-related mutations are clustered in discrete regions of the enzyme subunits, called regions determining quinolone resistance [11,31].

A decreased intake of drugs by reduced permeability or active efflux: Increased resistance to quinolones in gram-negative bacteria is due to variations in their outer membrane proteins.so that they reduce the intake of drugs [29].

The target protection of topoisomerase with specific proteins: A family of small penta-peptide-repeat proteins, called Qnr proteins, which bind to the targets for topoisomerase and protect them from quinolone interaction, provides target protection. A similar mechanism has developed in bacteria to protect topoisomerases from microcin, which are pentapeptide repeat family proteins that are produced as a mechanism of biological competition by certain bacteria and can kill susceptible bacteria by inhibiting their topoisomerases [34].

Inactivation of the drug: The most recently identified mechanism of resistance to quinolones was inactivation by drug modification. A plasmid-encoded AAC enzyme variant that has the ability to acetylate some quinolone molecules in addition to aminoglycosides and have unsubstituted secondary amines such as ciprofloxacin and norfloxacin performs acetylation [34,35].

Causes of antibiotic resistance

Although antibiotic resistance also occurs in nature and is an inevitable consequence of even prudent antibiotic use, it is clear that overuse and misuse of antibiotics is the main determinant for the increases in antibiotic resistance. Intense antibiotic therapy may benefit the individual patient but, unlike any other drug category, has environmental and ecological consequences [31]. Thus, selective and decreased antibiotic use, both in Veterinary clinic and field, is the key issue. Limitation of antibiotic prescriptions, selection of the most appropriate treatment, use of narrow-spectrum antibiotics as often as possible, optimization of therapy duration, rapid de-escalation of initial empiric therapy, and control of resistant strains are the cornerstones of causes of antibiotic resistance. Rational antibiotic use should slow the dissemination of MDRB and effectively extend the sustainability of today's antibiotics [23].

In general, there are six main causes of antibiotic resistance such as, Over-prescription of antibiotics, patients not finishing the entire antibiotic course, overuse of antibiotics in livestock and fish farming, poor infection control in health care settings, poor hygiene and sanitation and absence of new antibiotics being discovered [14].

Some of antibiotic-resistant pathogens in different animals

These summaries identify bacteria of particular risk, how to manage infections caused by them in the context of antimicrobial resistant, and their overall implications for animal health and welfare.

In cattle, bacteria responsible for endemic cattle diseases in the United States, such as bovine keratoconjunctivitis (IBK or "pink eye") or bovine respiratory disease, are developing resistances to multiple available antibiotics. Difficult to treat infections have significant impacts on animal health and welfare, as well as long-lasting economic consequences for cattle producers [28]. A key component of effective antimicrobial uses for cattle is disease prevention, which includes implementing good management practices and regular use of vaccines. More research is needed around strategies for disease prevention, control, and treatment in individual animals, as well as herd outbreaks [36].

In horses, some pathogens affecting equine health, such as *Staphylocuccus spp* and *Pseudomonas aeruginosa*, appear to be resistant to multiple antimicrobials [12]. FDA-approved antimicrobials for horses are often not active against the resistant pathogens; legal, extra label use of human and animal antimicrobial drugs may be necessary for treatment. Comprehensive attention is important, including strategies for infection prevention and pathogen control; diagnostic testing to better informs therapeutic decisions, and prompt treatment and compliance with veterinarians' recommendations [38].

In sheep and goats, Mastitis and abortions in these small ruminants are greatly impacted by antibiotic-resistant pathogens. *Staphylococcus spp* and *Campylobacter jejuniare* the main pathogens of concern [28]. Antimicrobial-resistant infections can be more difficult to treat and may only be recognized as non-responsive infections, leading to additional rounds of therapy with alternative antimicrobial drugs. Poor health has negative effects on animals' welfare, and results in significant economic loss for producers by reducing both the quality and quantity of milk, meat, and fiber [27]. According to Daniel [39] report, out of 80 Salmonella positive sheep samples, all (100%) of them were resistant to amoxicillin and ampicillin while sixty-eight (85%), 68 (85%), and sixty (75%) isolates were susceptible to gentamicin, ciprofloxacin, and kanamycin, respectively. Thirty (37.5%) Salmonella isolates were resistant to both trimethoprim and tetracycline and 25% of the isolates were resistant to both doxycycline and chloramphenicol while 12.5% of the isolates were resistant to nalidixic acid.

In dogs and cats, the prevalence of resistant bacteria in dogs and cats is not known but those of concern include methicillin resistant Staphylococcus (MRSA), as well as Pseudomonas and Campylobacter jejuni. Prior antimicrobial use is one risk factor for reduced effectiveness of subsequent antimicrobial therapy. Comprehensive healthcare plans and veterinary oversight of antimicrobial drug use in dogs and cats may help reduce antimicrobial resistance and maintain the effectiveness of critical therapeutic agents [28].

In swine, resistance to antimicrobials has been identified for opportunistic, environmental, and commensal pathogens, such as Pasteurella, Streptococcus, E. coli, and Salmonella. Because these bacteria are ubiquitous in swine production, working with producers to consistently implement core principles of good swine health management is critical to prevent infections. These principles include not mixing pigs from different sources, adopting all-in/all-out management whenever practical, maintaining good hygiene, and minimizing environmental stresses due to temperature fluctuations and poor ventilation [32].

In poultry, the prevalence of *E. coli* and *Pasteurella multocida* infections increased substantively between 2018 and 2019. Identification of multi-drug resistance in these common pathogens calls for increased attention to biosecurity and environmental factors, such as water sanitation, rodent control, and ventilation [36].

In Fish, high mortality (up to 60% for Edwardsiellaspp in catfish), combined with multi-drug resistance to the few FDA approved antibiotics available to treat disease in fish and shrimp, harms animal health and welfare and can be economically devastating for aquaculture. Some antimicrobials used for aquatic species are sold over-the counter and online to the public, including many that are prohibited or restricted for use in these species. Removing products that are not FDA-approved from the market and requiring veterinary oversight for those products that are approved may help reduce the development and spread of antimicrobial resistance [32,37].

Alternatives to Antibiotics for Disease Prevention

Antibiotics and their alternatives can also be used to prevent diseases in healthy animals. Disease prevention uses are defined as the administration of a drug to healthy animals in a situation where a specific and increased disease risk is present. This use is distinct from situations where antibiotics are used to control the spread of diseases in a herd or flock when some animals already show clinical signs of disease [30]. Both uses, however, are aimed at protecting animals from disease during times of increased risk of infection and are grouped under disease prevention for the purpose of this analysis. Key similarities exist between growth promotion and disease prevention uses for drugs and alternatives, including the administration to healthy animals and potentially long durations of use. In many cases, it is likely that the growth-promoting effect is at least partially due to the product's ability to inhibit or kill bacteria. At the same time, preventing animals from becoming sick can prevent productivity losses due to illness, whether clinical or subclinical in nature [37].

Vaccines

Vaccines have been widely used in veterinary medicine to prevent diseases caused by viruses or certain bacteria, and they are promising substitutes for some antibiotic uses. Notably, reducing viral infections may lead to decreased antibiotic use because of the risk of misdiagnosis and because antibiotics may be used to prevent or treat secondary bacterial infections [35]. Therefore, vaccines for both viral and bacterial infections are relevant to the discussion around alternatives to antibiotics. Evidence suggests that at least some vaccines may also have positive effects on growth rates and animal performance, even though external factors such as the need to handle animals for vaccine application can impede them [24].

Vaccines stimulate a protective immune response that is more or less comparable to the effects that follow a natural infection, but generally without the negative impacts caused by the clinical progression of the disease, and vaccines have a long history of successful use in animals [26]. Varieties of vaccines are commercially available and actually used on U.S. operations as a management option to prevent and reduce the spread of infectious diseases. For instance, according to recent NAHMS data, more than 70 percent of U.S. operations are estimated to vaccinate very young (i.e., nursery-age) pigs against Mycoplasma pneumonia; similarly nearly 60 percent of beef cow-calf operations vaccinate against clostridial diseases caused by *C. chauvoei*. By preventing infection, vaccination can reduce antibiotic use. For example, vaccination against Lawsonia intracellularis, a bacterium causing a severe intestinal disease called ileitis, has been shown to reduce the need for oxytetracycline in pigs in Denmark. In the U.S., an estimated 26 percent of breeding pig operations vaccinates against *L. intracellularis*. Therefore, vaccines may become better alternatives to antibiotics in the future [39].

Immune modulators

Immune modulators, which as defined here include the transfer of antibodies to elicit passive immune responses, are promising alternatives for disease prevention and potentially for treatment as well [12]. In contrast with vaccines, immune modulators stimulate the immune system in a way that is less dependent on the pathogen causing infection, which makes them effective against a broad range of pathogens. A very broad variety of immune stimulatory substances has been investigated as potential alternatives to antibiotics. These include cytokines (i.e., substances that are secreted by certain immune cells to regulate other parts of the immune system), lipopolysaccharides (i.e., large molecules that are present in the wall of certain bacterial cells and trigger innate immune responses), short segments of bacterial DNA that also stimulate innate immune responses, antibodies derived from egg yolk that provide short-term immunity, and certain plant materials [23].

In chickens, a meta-analysis showed that egg-yolk antibodies significantly reduce the risk of necrotic enteritis, and several studies have provided promising results for other types of immune modulators. For example, after day-old broiler chickens were intentionally infected with E. coli, significantly fewer clinical symptoms were reported in those animals treated with a CpG-based immune modulator than in the control chicks [32].

In swine, a meta-analysis demonstrated efficacy of egg-yolk antibodies in preventing diarrhea caused by a variety of bacterial and viral pathogens. A systematic review concluded that another type of immune modulator, in the glycan family, failed to demonstrate efficacy in pigs but that the data were scarce. However, individual scientific studies of challenges with bacterial toxins showed highly promising results for vitamin C and glycan's in young piglets. Feeding of antibodies derived from egg yolk has also shown promise for the prevention and treatment of diarrhea in young piglets, even though limited stability in the swine gut and narrow host spectrum pose potential challenges, and cost-effectiveness so far remains elusive due to high production costs [33].

In the U.S., two immune modulators have recently successfully demonstrated safety and efficacy and have been approved for use in cattle. One is for use in dairy cows to prevent udder infections after calving; it is based on a cytokine and recently received animal drug approval from the Food and Drug Administration [14].

The efficacy of immune-stimulants relies on a functioning immune system and therefore may not always be a feasible option; for instance, in very young animals, the immune system is not yet fully functional, and severe stress and disease can limit the functionality of the immune system. There are also safety concerns about using immune-stimulants before the immune system is fully formed because of the potential risk for adverse developmental effects [35].

Bacteriophages, endolysins, and hydrolases

A number of viruses and the enzymes they generate show promise as alternatives for antibiotics that may be used for disease prevention and potentially for treatment, thereby also potentially indirectly affecting production performance [25].

Bacteriophages: Bacteriophages are viruses that infect and kill bacteria. Most bacteriophages have a narrow range of bacterial strains they can infect, which in extreme cases can be restricted to a single strain of a bacterium. Therefore, Bacteriophages can be used in a highly targeted way with minimal unintended impacts on other bacteria and the host. In addition, antibiotic resistance typically does not interfere with the bacteriophage's ability to infect and kill the bacterium, which may make them one of few treatment options for infections with multidrug-resistant bacteria. In addition, because the bacteriophages multiply in the bacteria they infect, a reasonably broad dosage range can be effective [35]. However, bacteria can become resistant to bacteriophages; bacteriophages may rapidly degrade in the environment; and there is some risk that certain bacteriophages may have the ability to spread antibiotic resistance genes. Overall, bacteriophage therapy tends to be extremely time-sensitive. For example, phage therapy had limited efficacy when administered more than 16 hours after experimental infection. Notably, bacteriophages are actually naturally occurring and common in the environment [35].

Bacteriophages have been used for disease prevention and treatment, with promising results. For example, they have protected chickens from respiratory disease after experimental infection with E. coli. Similarly, Salmonella infection in day-old broiler chicks was successfully treated by a phage cocktail containing bacteriophages specific to Salmonella enteritidis. Bacteriophages have also been evaluated as treatments for colibacillosis in chickens, and mortality was comparable to the comparison group that received the antibiotic enrofloxacin [30].

Phage therapy has also shown promising results in piglets and calves, where bacteriophages significantly reduced the prevalence of diarrhea caused by E. coli and successfully treated them in piglets. However, the major obstacles to using bacteriophages for disease treatment in animals include the lack of rapid and accurate diagnostics which are necessary because the phages typically are effective only against a very narrow range of bacterial strains the risk of phage inactivation via the host immune response, and rapid emergence of resistant bacterial strains. Phage cocktails that contain several different bacteriophage strains can help address these limitations, but to date, efficacy for treatment of pathogenic organisms has remained limited [2].

Endolysins and lysozymes: Endolysins and lysozymes are hydrolases. Hydrolases are enzymes that degrade peptidoglycans, the main building block of the bacterial cell wall, and thereby kill bacteria. The hydrolases can be derived from a number of different sources, including bacteriophages, as well as animals, plants, bacteria, and insects, with varying specificity for target bacteria [2].

Endolysins: Endolysins, also commonly referred to as virolysins, are generated by bacteriophages. Bacteriophages generate endolysins at specific stages of their life cycle, shortly before the virus destroys the bacterial cell. In that process, endolysins aid in the release of the newly generated bacteriophages. Endolysins tend to have a relatively narrow spectrum of bacteria against which they are effective and are highly thermo stable. In experiments at 100 degrees Celsius, some retained over 70 percent of their activity against Staphylococcus aureus. Such heat stability can be important to assure product integrity, as some feed is processed at high temperatures. The mechanism by which endolysins target and eliminate pathogenic bacteria has been fully described and depends on two distinct functions: binding to specific sites in the bacteria cell wall and cleaving the bonds between the peptidoglycans in the cell wall [2].

Endolysins are tentatively promising enzymes for the prevention and treatment of certain bacterial infections. In part, this is because it is believed to be more difficult for bacteria to develop resistance against them, and in part, because it may be possible to specifically engineer endolysins with the desired host spectrum. However, concerns about potential adverse immune responses and the downsides of a relatively narrow host spectrum have to be considered. Yet, although efficacy data specific for the use of endolysins in food-producing animals have so far remained scarce, endolysins have shown promising results against a relatively broad range of bacteria. It should be noted that endolysins are not effective against all bacteria. Because of differences in the bacterial cell wall, endolysins tend to have limited efficacy against Gram-negative bacteria [23].

Lysozymes and autolysins: Lysozymes and autolysins are hydrolases generated by eukaryotic organisms (i.e., animals and plants)

and bacteria, respectively. In humans, lysozymes are an important component of the innate immune system and naturally present in the skin and secreted into saliva, urine, milk, and other bodily fluids. Lysozymes in particular tend to have activity against a broad spectrum of bacteria and are known to effectively break down the carbohydrate component of peptidoglycan layer of bacteria. They are also known to be effective against viruses and other pathogens. Lysozymes and autolysins are promising alternatives to antibiotics, although they share many of the limitations discussed under endolysins [11].

Other disease prevention alternatives

A variety of other approaches for disease prevention has been proposed, including biofilm inhibitors and quorum-sensing inhibitors (i.e., substances that disrupt biofilm formation, a bacterial communication system that plays an important part in the infection process). While these approaches may offer innovative alternatives to antibiotics, data on safety and efficacy are to date largely lacking [13]. In addition, their impact on production performance for growth promotion purposes replacing antibiotics remains largely unknown. One class of specific and particularly promising products is virulence inhibitors: molecules that directly affect the harmful microbes and block key functions they need in order to survive and infect. For example, they may prevent bacteria from forming pili, structures that allow them to adhere to animal cells. Experimental data for inhibitors remain limited, so the safety and efficacy of these approaches are unclear; however, such novel approaches represent a new path, one that does not attempt to directly kill bacteria but rather tries to restrain some of their pathogenic activities. This approach may for instance be less likely to disrupt the healthy balance in the gut [36].

Farm management and biosecurity

Other alternatives including biosecurity and management practices are an important part of disease prevention that can improve overall animal health and significantly reduce the risk of pathogen introduction into the herd or flock [29]. Notably, a comprehensive approach that includes alternative products and improved management practices is likely to be more effective than relying on a single alternative product or approach to manage health and prevent disease. In fact, improvements in biosecurity have been widely accepted as an effective means of preventing the introduction of diseases into herds or flocks. This concept applies widely across species, production systems, and pathogens [12].

Conclusion and Recommendation

The current antibiotic resistance crisis is likely to be a permanent feature of animals and human society, causing increased animals suffering and social-economical costs. Managing this crisis to limit its effect upon animals will require a fundamental shift in the global perception of antibiotic usage. A variety of products and management practices may eventually be able to replace a substantive proportion of current antibiotic use for prevention and growth promotion purposes, but this effort will require a comprehensive approach that considers alternatives as one part of a herd health management program.

Therefore, the following points are recommended for the antibiotic resistance in veterinary medicine:-

Continuous awareness creation should be done to animal health professionals, animal producers and the general community for antibiotic resistance.

Implementable and inclusive antibiotic usage policy formulation and enforcement should be done.

Developing new antibiotics is important, but strategies to prevent infectious diseases by immunisation or other public health measures should be done.

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