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The Epidemiology and Antimicrobial Resistance of *Salmonella*: A Review

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Abstract: *Antimicrobial resistance (AMR) remains an alarming issue with public-health concern and economic implications on human and animal populations worldwide. These antibiotics have so far been associated with high burden of diseases and the ramifications of veterinary antibiotic resistance of Salmonella spp., on the Sustainable Development Goals cannot be teased out. This review paper highlights the epidemiology of Salmonella spp., and antibiotic resistance in livestock productions systems.*

Keywords: *Epidemiology, Antimicrobial resistance; Salmonella, Livestock Systems.*

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Introduction

Globally, antibiotic use in livestock setting is accounted for approximately 80% of total consumption [1]. Veterinary antibiotics are used for livestock production, growth promotion, prevention and treatment of infections [2]. However, improper use of antibiotics to the livestock can lead to the development of antibiotic-resistant pathogens which can be exposed to the environment and pose a human health risk upon consumption [3]. The persistence and emergence, however, of antimicrobial resistance in bacterial communities in particular *Salmonella* spp., pose a sheer threat to treatment options of microbial infections in a cost effective manner solutions and thus place a burden on health services leading consequences in human and animal health settings [4].

Salmonellosis poses a major public health threat and represents significant economic implications to the poultry industry due to reduced production and mortality worldwide [5]. Moreover, *Salmonella* is a common intestinal inhabitant in a wide-range of animals, including mammals, reptiles, birds and insects [6]. Most cases of human infection are associated with the consumption of contaminated food products such as beef [7], pork [8], poultry, animal products [9], and vegetables [10]. Infections may also be associated with the contact between humans and infected animals [11]. The epidemiology and extent of antimicrobial resistance of *Salmonella* spp., in livestock systems has not been comprehensively reviewed. Thus, it is this concern that motivates our review and our approach to this question involves a rapid a review. In this paper, we have conducted overview of the epidemiology and antimicrobial resistance of *Salmonella* spp., in the wider context and close specific cases pertaining to the themes of interest in Malaysia.

Overview of *Salmonella*

Historical background

In 1884, Theobald Smith first recognized the organism *Salmonella*. A year later, Daniel Elmer Salmon, an American veterinarian and Smith, discovered and isolated the first organism, *Salmonella choleraesuis* from the intestines of pigs infected with classical swine fever (hog cholera) [12]. Although the organism was initially named *Bacillus choleraesuis*, it was later changed to *Salmonella choleraesuis* by Lignieres in 1900 [13-15].

The antigenic classification of *Salmonella* is a result of interactions between the antibody with the surface bacterial antigens in the 1920s to 1940s [16]. According to the Kauffmann-White scheme, each *Salmonella* serotype is classified by its possession of a particular lipopolysaccharide (LPS) or O antigen and a flagellar or H antigen and contains more than 2500 serotypes [14, 17-19].

Taxonomy

Historically *Salmonella* has been named based on original isolation places such as *Salmonella indiana* and *Salmonella london*. *Salmonella* belongs to the family Enterobacteriaceae, Phylum *Protobacteria* and Class Gamma Protobacteria. It is a member of the Order Enterobacteriales belonging to the Family *Enterobacteriaceae*. The Genus *Salmonella* comprised two species; *Salmonella enterica* and *Salmonella bongori. Salmonella enterica* is comprised of six subspecies namely; *S. enterica subsp. enterica, S. enterica subsp. salamae, S. enterica subsp. arizonae, S. enterica subsp. diarizonae, S. enterica subsp. houtenae and S. enterica subsp. Indica. Salmonella enterica* subspecies I is isolated mainly from warm-blooded animals and accounts for more than 99% of clinical isolates, whereas the remaining subspecies and *S. bongori* are mainly isolated from cold-blooded animals and account for less than 1% of clinical isolates [20-22]. Epidemiological classification of *Salmonella* is based on the host preference, which includes host-restricted serotypes that infect only humans such as *S. typhi*. Furthermore, the other classification comprises host-adapted serotypes which are associated with one host species but can cause disease in other hosts such as *S. Pullorum* in avian. The broad host range serotypes include *Salmonella enteritidis*, *Salmonella typhimurium* and *Salmonella heidelberg* are the most frequent serotypes recovered from human each year [23-27].

Salmonella spp. are serologically classified using the Kauffmann-White scheme and contains more than 2500 serotypes [14, 17-19], based on the three antigens: H (flagellar antigen); O (somatic antigen); and Vi (capsular antigen). H antigen is a heat-labile that may occur in either or both forms, called phase 1 and phase 2. O antigens are heat stable and occur on the membrane of *Salmonella* and are determined by oligosaccharide component of lipopolysaccharide. Vi antigen is a heat sensitive antigen that is composed of polysaccharides and is a superficial antigen overlying the O antigen; it is present in a few serovars, the most important being *S typhi* [28-31].

Phenotypic and biochemical properties

Salmonella are Gram-negative, rod-shaped (bacillus), non-spore-forming, facultative aerobic bacteria and size ranging from 0.4-0.6×2-3μm. They are motile by means of peritrichous flagella, except *Salmonella pullorum* and *Salmonella gallinarum* (non-motile) [19, 32]. Furthermore, *Salmonella* spp multiply optimally at a temperature of 35◦C to 37◦C, pH about 6.5-7.5 and water activity between 0.94-0.84, also are sensitive to heat and often killed 70°C or above; therefore, it is sensitive to pasteurization. Complete growth inhibition occurs at temperatures less than 7°C, pH less than 3.8 or water activity less than 0.94, but resist to drying even for years [17, 33]. *Salmonella* produces acid on glucose fermentation; reduce nitrates to nitrite, and do not produce cytochrome oxidase. In addition, *S. typhi* produce gas (H2S) on sugar fermentation [32]. *Salmonella* are non-capsulated except *S. typhi*, *S. paratyphi* C and some strain of *S. dublin* [32].

Epidemiology of *Salmonella*

Salmonella is one of the leading causes of bacterial foodborne disease and the third cause of human death among diarrheal diseases worldwide, even though the occurrence varies between countries [34]. *Salmonella* serotypes are not evenly distributed around the world, for example, non-typhoidal *Salmonella* (NTS) is more common in Africa, while typhoidal *Salmonella* (e.g. serotypes typhi and paratyphi A) are more common in Southeast Asia [35]. In 2000, the most frequently isolated serotypes from human sources were *S. enterica* serotype *typhimurium* and *S. enterica* serotype *enteritidis*. Salmonellosis is a major foodborne disease in developing and industrialized countries, although incidence rates vary [36, 37].

The epidemiology of *Salmonella* associated infections depends on the serotypes of the *Salmonella* spp, which it has more than 2,500 serotypes with different reservoirs. Changes in food consumption, culture, production practices, economic impact, and distribution have led to increased frequency of various multistate outbreaks related to freshly produced and processed foods [38, 39]. *Salmonella* spp. are generally disseminated in domestic and wild animals and they are common intestinal inhabitant in many animals, including mammals, reptiles, birds, and insects [6, 40]. The animals are the primary source of *Salmonella*, and food-producing animals are the main transmission route to humans.

The risk of public health differs depending on risk factors as animal species, age group, husbandry practice and health status, and certain human communities are at a high risk of infection due to biological or behavioral factors [27]. Most cases of human infection are associated with the consumption of contaminated food products such as beef [7], pork [8], poultry and poultry products [9], vegetables [10]. Infections may also be associated with the contact between humans and infected animals such as reptiles and amphibian [11, 41]. The prevalence of *Salmonella* spp. from chicken carcass was 62.5% in Senegal [42]. A study from Vietnam

reported a high prevalence of *Salmonella* spp. in raw meat and poultry, which of 64% pork, 62% beef, and 53.3% of chicken samples[43].

Several studies showed the contamination of poultry samples with *Salmonella* across different countries; 23 to 29% in the United Kingdom [44, 45], 2.8 to 26.4% in Ireland [46, 47], 13.2% in The Netherlands [48], 35.8% in Spain [49], 36.5% in Belgium [50], and 36% in Korea [51], 60% in Portugal [52]. Furthermore, in Malaysia, *S. enteritidis* (28.1%), *S. weltevreden* (25.7%), *S. corvallis* (10.3%) and *S. typhimurium* (6.7%) were the most frequently non typhoidal *Salmonella* reported in 2003 – 2005.

Prevalence of Salmonellosis in Malaysia

In Malaysia, the prevalence of *Salmonella* from various food samples reported in different studies. For instance, [53] reported the prevalence of *S. enteritidis*, and *S. typhimurium* were 20.80%, 6.70%, and 2.50% , respectively. Additionally, [54], reported from Malaysia shows that a total of 27.6% collected samples were *Salmonella* spp., positive, and the chicken samples recorded higher rate 40.4% compared to beef 15.4% of *Salmonella* spp., isolates. This indicates the widespread of incidences and spread of *Salmonella* in retails level. Another study from Malaysia reported that was high prevalence of *Salmonella* was 88.46% in poultry, poultry processing environment [55]. [56] Reported that in Malaysia, the prevalence rate of *Salmonella* in poultry carcasses was 35.5% and poultry processing plants 50.0%. [57] Revealed that the prevalence of *Salmonella* in raw foods in chicken pieces 39%, livers 35%, and gizzards 44% samples in Malaysia. [30] Reported that prevalence of *Salmonella* was high with 27.2%% in beef and 72.7% in chicken meats at 2006 – 2009 in Kuala Lumpur, Malaysia. In Peninsular Malaysia, [58] reported the overall prevalence of *Salmonella* species was 29.1% from raw vegetables, chicken and processing environments. Another study showed that 22.0% retail meat and 22 7.5% street food samples were positive for *Salmonella* serovars [59]. The major variation between the prevalence of *Salmonella* in the different studies might be due to sample size, sample type, diagnostic techniques and geographical area.

Reservoir host and source of infection

Animals infected with salmonellosis and human's feacal wastes are the main sources of bacterial contamination to the environment and the food chain [26, 60]. *Salmonella enterica* subspecies *enterica* is widely distributed in the intestinal tracts of animals and the environment. Poor personal hygiene is the cause of human infection after direct contact with infected animals and humans. Environmental contamination, especially untreated water is also important. Consumption of contaminated food of animal origin is the source of most human infections [61-64]. Food of animal origin, meat, poultry, and, unpasteurized egg products are the primary sources of human salmonellosis [65, 66]. Reports show that 96% of *Salmonella* infections in humans are from livestock and their products [67]. Thus, the production, processing, transporting, and storage of the food in an unhygienic environment may become contaminated with *Salmonella*e and be responsible for diseases [68].

A less frequent source of non-typhoidal *Salmonella* infections is exposure to pets, especially reptiles [41]. Reptiles may have fecal carriage rates of up to 90%. It is estimated that approximately 74,000 infections with *Salmonella* result from exposure to reptiles and amphibians in the United States each year [69]. For instance, a study from Korea in health zoo animals revealed that *Salmonella* isolates was about 6% of animals, including 30% of reptiles, 7% of birds and 1% of mammals [70].

Methods of isolation and identification of *Salmonella* pp,

Conventional culture techniques

In conventional culture techniques, pre-enrichment media is often necessary to permit the isolation and permit the detection of low numbers of *Salmonella* sub-lethally damaged. Pre-enrichment media including Buffered peptone water (BPW), and Enrichment media are liquid or semi-solid agar media which permit the growth of *Salmonella* and inhibiting the growth of other bacteria such as modified semisolid Rappaport-Vassiliadis (MSRV) or diagnostic semi-solid *Salmonella* medium (DIASALM), Rappaport- Vassiliadis (soya base) (RVS) [71, 72]. For selective plating for the bacterial culture media includes xylose lysine deoxycholate agar (XLD agar) and Brilliant green agar (BGA), bismuth sulfite agar and others could be used as the second plating-out medium as previously described [30, 58, 73]. Non- motile *Salmonella*e, including *S. Pullorum* and *S. Gallinarum*, and the *S. Enteritidis* strain do not grow in MSRV, thus it should be used different media to identified. For *S. Pullorum* and *S. Gallinarum* show a results obtained from direct enrichment in selenite cysteine and RVS [74]. *Salmonella* can be biochemically characterize using triple sugar iron agar (TSI) urea agar lysine iron agar (LIA), Voges Proskauer (VP), methyl red (MR) and Indole tests [19, 30, 73, 75].

Molecular and Serological confirmation

Various *Salmonella* detection methods are in use and are commercially available, these includes agglutination tests, immunomagnetic separation (IMS) the method described by [54], Enzyme-linked immunosorbent assay (ELISA), anti-globulin and compliment fixation tests (CFT) have been used to detect antibody responses to *Salmonella* infections. For DNA-based methods, gene probe PCR methods, real time PCR, as previously described[30, 76-79], quantitative PCR, pulsed-field gel electrophoresis (PFGE). PCR methods potentially detect faster and more accuracy compared with traditional culture but it offer high cost [80-82], and microarray analysis [83]. Rapid isolation methods may be linked with sophisticated detection systems, such as biosensors [84]. Slide agglutination used the detection of *Salmonella* O-; Vi- and H- antigens with the suited sera, from pure colonies and after auto-agglutinable strains have been eliminated. This method relies on the antibody/antigen reaction between cultured test and commercially prepared antiserum [73].

Antimicrobial susceptibility tests and resistance profile

Disk diffusion refers to the dissemination of an antimicrobial agent of an identified concentration from disks, tablets or strips, into the solid media that has been isolated in a pure culture. Disk diffusion is based on to determine the inhibition zone proportional to the susceptibility of the bacteria to the antimicrobial present in the disk. The diameter of the zone of inhibition around the antimicrobial disk is related to minimum inhibitory concentration (MIC) for the particular bacterium/antimicrobial combination; the zone of inhibition associates in reverse with the MIC of the tested bacteria. Generally, the larger zone of inhibition, the lower the concentration of antimicrobial required to inhibit the growth of the organisms. However, this depends on the concentration of antibiotic in the disk and its infusibility according to the guidelines of the [85]. Disk diffusion is a low-cost method that can be easily modified to test antimicrobial disks when needed. It used for screening tests against a large number of isolates and to identify a subset of isolates for further testing using other methods, such as MIC determination as previously applied in [53, 58, 77]. Broth and agar dilution methods also used to define the lowest concentration of the assayed antimicrobial that inhibits the visible growth of the bacterium being tested. Antimicrobial varieties should contain both the interpretive criteria (susceptible, intermediate and resistant) for specific bacterium/antibiotic combination and suitable quality control reference organisms. The selection of an AST methods based on the easy to perform, flexible, adaptable to automated or semi-automated systems, cost, reliable, and accurate.

Economic and public health significance of *Salmonella* infections

Over the last two decades, foodborne disease has emerged as a significant and increasing public health and economic impact in many countries. Frequent outbreaks caused by new pathogens, antibiotic use in farm animals, and the spread of antibiotic resistance to humans are just a few examples [86]. Thus, economic losses in livestock industry are generally due to increased mortality, performance losses, and costs associated with treatment and control of infections. Mortality rates characterized to *Salmonella* infection are particularly high in young animals, due to the required the large amount of treatment [27]. Salmonellosis is the most severe forms of enteric fever and food poisoning in humans and animals and remains a major threat to public health, contributing to the economic burden worldwide [55]. Even though, Salmonellosis have been associated with a wide ranges of food sources, in particular poultry, have been considered as the main cause of human salmonellosis [87].

In the United States, the incident rate of non-typhoidal salmonellosis has doubled in the past two decades. CDC estimates *Salmonella* causes about 1.2 million illnesses, 23,000 hospitalizations, and 450 deaths in the United States every year. Food is the source for about 1 million of these illnesses [88, 89]. Sheep and goats can be reservoir in variety of *Salmonella* serovars, including the most common serovars for human infections, *Salmonella enterica* serovar Enteritidis and *Salmonella enterica* serovar Typhimurium [90]. Contact with small ruminant poses a health risk to occupationally exposed subpopulations as well as the general public, but the risk varies greatly depending on the serotype [27]. In 2004, 192,703 human cases of salmonellosis were reported in the European Union (EU). These and similar data from other countries almost certainly underestimate the scope of the problem, as many salmonellosis cases go unreported. In addition to human health implications, *Salmonella* may be a pathogen of great importance in worldwide animal production and therefore the emergence of antibiotic-resistant strains, due principally to the therapeutic use of antimicrobials in animals, could be a further threat to human and animal health [91]. Human salmonellosis is considered as an significant socioeconomic disease posing substantial economic burden globally [92]. It also generates negative economic impacts for surveillance investigation, and treatment and prevention measures [68]. EU Commissioner for Health and Consumer Protection, estimated the costs of food-borne *Salmonella* alone are up to € 2.8 billion annually in EU countries altogether [89, 93].

In Malaysia, food and waterborne diseases (e.g., typhoid, cholera, dysentery, hepatitis A, and food poisoning) have a prevalence of 60.28 cases per 100,000 individuals with the reference typhoid fever being the

most common and the majority of the other cases of food poisoning. According to the Ministry of Health Malaysia 2007, outbreaks of foodborne diseases were mostly due to unhygienic food handling, resulting in more than 50% of the food poisoning cases classified as the NTS serovars Typhimurium, Enteritidis, and Corvallis [94]. Typhoid fever is a common foodborne disease in Malaysia. In Klang Valley, typhoid cases were higher in men (n = 272) at 55.6% than in women (n = 217) at 44.4%. The mean age of typhoid fever cases in Klang Valley was 29.80 years (±17.44). Most of the typhoid cases were detected among patients aged 21 to 30 years [95]. Other studies in Malaysia have shown that children (0–4 years) and young adults (25–29 years) were more susceptible to typhoid than the older age population [96]. The overall trend of typhoid cases in Klang Valley had increased from 2011 (37 cases), 2012 (44 cases) and 2013 (50 cases). There was a sudden spike of cases in 2015 with 98 cases compared with 36 cases in 2014. This sudden increase was related with an outbreak that involved construction workers where 13.7% (n = 70) of cases from this study were contributed by foreigners [95]. Typhoid fever is endemic in Malaysia where the country still experiences periodic epidemic outbreaks. In Malaysia, typhoid is more common in Kelantan state where various outbreaks were recorded in Kelantan in 2001 until 2007 [96], with a major outbreak in 2005. A previous study from the outbreak of typhoid fever in Kelantan In 2005, State Health Department of Kelantan reported 517 confirmed typhoid reported cases. Included 2 deaths cases, Show that 19 (2.14%) food samples, 12 (2.9%) drinking water samples and 2 (0.2%) food handlers tested positive for *Salmonella* spp [97]. The annual incidence rate in Kelantan state (which has 10 districts) was 37 per 100,000 population were estimated [95]. Other reports in Malaysia revealed that the incidence rates of typhoid fever in the Federal Territory of Kuala Lumpur for 1996 and 1997 were 3.68 and 3.78 per 100,000 population respectively [98], but lower incidence rates were recorded in a study from Klang Valley with 0.58 and 1.42 per 100,000 population in 2011 and 2015, respectively. Another study from Perak state observed an outbreak in a residential school shows *Salmonella enteritidis* strain was isolated from the clinical samples from the infected patients, and food sample, microbiological analysis for the clinical samples(rectal swabs) from 26 of the samples (89.6%) out of 29 patients were positive to *Salmonella enteritidis* [99].

The annual incidence rate in Malaysia is between 10.2 and 17.9 per 100,000 population [95]. The most common non typhoidal *Salmonella* Serotypes identified and reported to the Laboratory Based Surveillance database, 2003-2005 includes; serotypes Enteritidis, Weltevreden, Corvallis, Typhimurium, Stanley, Biegdam, Tshongwe, Albany, Braenderup, and Newport [97].

Antibiotic resistance of *Salmonella*

The development of antimicrobial resistance in *Salmonella* strains is a serious health concern globally [100]. In the early 1960s, the first incidence of *Salmonella* resistance to the antibiotics was discovered in particularly chloramphenicol [101]. Meanwhile, the frequencies of *Salmonella* isolation towards resistance to one or more antimicrobial agents have become increasingly prevalent in many countries around the world [102].The primary key factors contributing the development of resistance in LMIC countries include poor drug-resistant infections surveillance, quality of the available antibiotics are low, clinical misuse, and antibiotics accessibility. Whereas similar drivers leading to the developed countries include unregulated use of antibiotics and the weak of low on medication imports and rampant use of antibiotics in food-producing animals [103].
Livestock under intensive farm management generally use large amounts of antimicrobials for wellbeing farming conditions, therapeutic level to improve the health, productivity, and economic revenues by reduction of disease occurrence, morbidity and mortality of animals [104]. Misuse of these antimicrobials leading food contamination with resistant bacterial strains that can be transferred to other pathogens, potentially failure the treatment of severe bacterial infections, also from livestock into the environment and food chain to human consumption of contaminated food with resistant bacteria which cause a major burden for public health [43, 105].

Therefore, prevalence of resistant *Salmonella* towards antimicrobials has reported many countries from different types of food. Annual estimation of antimicrobial-resistant *Salmonella* spp., infections about 100,000 in United States[106]. In Vietnam study in raw meat and poultry samples was observed that approximately 50.5% of the *Salmonella* spp. isolates were resistant to at least one antibiotic; the highest prevalence rates of resistance was in pork 78.1% and chicken samples 88.9% [43]. Previous reviews from Asian countries, India, Pakistan and Vietnam and Malaysia shown higher rates of MDR isolates of *Salmonella* spp., than Indonesia and China [59, 107] another study revealed with a high rate of MDR isolates of *Salmonella* spp., in Pakistan, India, Nepal and Vietnam, while in China, Indonesia and Laos the incidence rate of MDR *Salmonella* spp., is relatively low [108]. Another study from Senegal shown that 78.9% *Salmonella* isolates were resistant to one or more antimicrobials [42]. In Malaysia study revealed that 55% of the *Salmonella* isolates were multidrug resistant[58]. Previously reported by [59], 67% of *Salmonella* was MDR.

Mechanisms of Antimicrobial Resistance

Resistance mechanism generally contains three sections; antimicrobial inactivation; decreased permeability; and antimicrobial agent alteration or replacement; [109-111]. Permeability changes in the bacterial cell wall which restricts antimicrobial access to the target sites, active efflux of the antibiotic from the microbial cell, enzymatic modification of the antibiotic, degradation of the antimicrobial agent, acquisition of alternative metabolic pathways to those inhibited by the drugs, modification of antibiotic targets and overproduction of target enzyme. Of particular concern is the development of resistance to key antibiotics such as the tetracyclines, Sulphonamides and Trimethoprim, quinolones and fluoroquinolones beta lactamas, aminoglycosides and chloramphenicol [112, 113]. Here we briefly review the above listed antibiotics in details:

Tetracyclines

Tetracyclines are broad spectrum antibiotics against wide range of gram negative and gram positive bacteria, atypical organisms such as chlamydiae, mycoplasmas and rickettsiae and protozoan parasites [114]. Tetracyclines are one of the widely used antibiotic in human, veterinary medicine and livestock farming as growth promoter in animal industry and for prophylaxis in plant agriculture and aquaculture as a result of their efficiency, low cost and safety [114, 115]. Tetracyclines are characterized as first-generation tetracycline, including tetracycline; chlortetracycline and oxytetracycline and second generation tetracyclines such as minocycline and doxycline [115]. Tetracycline inhibit proteins synthesis by preventing the binding of aminoacyl tRNA to the 30s ribosomal subunit acceptor site in the bacterial cell [114]. Resistance mechanisms to tetracycline include efflux pumps, ribosomal protection proteins (RPPs), and inactivation of the enzymes [110] . However, in *Salmonella* and *E. coli* efflux systems are more prevalent and the genes associated with an efflux mechanism, including *Salmonella tet*(A), *tet*(B), *tet*(G), and *tet*(R) and *E.coli tet*(A), *tet*(B), *tet*(C), *tet*(D), *tet*(E), *tet*(G), *tet*(J), *tet*(L), *and tet*(Y) [109, 115-117] . Tetracycline resistance has spread to most bacteria genera due to the consequence use over a long time in humans, in animals, and as growth promoters in animals [109, 114]. Tetracycline resistance in most bacteria is due to the acquisition of new genes, often associated with mobile elements. These genes are usually associated with plasmids and/or transposons and are often conjugative [117].

Sulphonamides and Trimethoprim

Trimethoprim and sulfonamides are synthetic antibacterial agents with a broad antibacterial spectrum for gram positive and gram negative bacteria used for treatment of respiratory system, skin and urinary tract infections [116]. Sulfonamides and trimethoprim work on the pathway of folic acid in bacteria by interfering the production of dihydrofolic acid. They have been used in food animals as growth promoters and for treatment of coccidiosis in poultry and colibacillosis in swine [109, 118] . Sulfonamides are bacteriostatic when used alone or bacteriocidal when used in combination (trimethoprim-sulfamethoxazole) [109, 119] . Sulfonamides compete with the structural analog p-amino- benzoic acid for binding to dihydropteroate synthase (DHPS), a catalytic enzyme in the folic acid biosynthesis pathway thus inhibiting the formation of dihydrofolic acid [120]. Resistance in gram-negative bacilli generally arises from the acquisition of dihydropteroate synthase (DHPS) genes in the integrons that are not inhibited by the drug [121]. Resistance mechanism of trimethoprime including: efflux pumps, mutation in the target enzymes impaired drug penetration, existence of naturally insensitive target dihydrofolate reductase enzymes, and the acquirement of drug-resistant target enzymes. Sulphonamide and trimethoprim is resistance is often encoded by *Sul1,Sul2* and *Sul3* genes and *dhfr* or *dfr* genes in *Salmonella* and *E.coli* animal isolates respectively [122-126].

Beta Lactams

Penicillin was the first Beta lactams antibiotic discovered and developed for clinical use in humans, and was one of the first antibiotics to which bacteria developed resistant [109, 110]. Beta lactams contain penicillin and cephalosporins which kill the bacteria by interfering with cell- wall biosynthesis. The β-lactam antibiotics are active inhibiting the cell wall synthesis by binding to the penicillin-binding proteins (PBPs) in bacteria and interfering the production of peptidoglycans in the bacterial cell wall resulting cytolysis due to osmotic pressure [110]. The most common and important mechanism resistance in enterobacteriacea to β- lactams is the production of β-lactamases including extended-spectrum β-lactamases (ESBLs),plasmid-mediated AmpC enzymes and carbapenem-hydrolyzing β-lactamases (carbapenemases) [110, 127, 128] which are encoded chromosomally or on plasmids and inactivate β -lactams by hydrolyzing the β-lactam ring [129]. The beta lactams used in veterinary medicine include, penicillins, ampicillin, amoxillin, benzyl penicillin, cloxacillin, hetacillin, nafcillin, penethamatehydroiodide [129];Penicillin beta lactamase inhibitor combination, amoxicillin/clavulanate first generation cephalosporins (cefadroxil, cefapiril, cephalexin), third generation cephalosporin (cefovecin, cefpodoxime, ceftiofur and fourth generation cephalosporins) [129].

Aminoglycosides

Aminoglycosides are antimicrobials that inhibit protein synthesis and modify the integrity of bacterial cell membranes by binding to bacterial ribosome [130]. They have a broad antimicrobial spectrum for treating a broad range of life-threatening infections in humans, animals and for bacterial disease control in plants [131]. Most commonly used aminoglycosides in animal husbandry include gentamicin, neomycin, or streptomycin [132]. Aminoglycosides is an enzymatic modification of the compound; several aminoglycoside resistance mechanisms have been recognized such as active efflux; decreased permeability, ribosomal alteration; and enzymatic modification of the 16S rRNA to prevent the aminoglycoside from binding to its ribosomal target can lead to resistance. Enzymatic inactivation is normally due to acetyl transferases, nucleotidyltransferases and phosphotransferases [109, 110].

Quinolones and Floroquinolones

Quinolones are broad spectrum antimicrobials agents that have been used widely in human medicine and veterinary practice in treatment of infections caused by enteric bacteria such as *Salmonella* and *Escherichia coli* [109]. A number of fluoroquinolones have been used in food animals including ciprofloxacin, enrofloxacin, difloxacin, marbofloxacin, orbifloxacin, and sarafloxacin [133]. All fluoroquinolones has the same mode of action inhibition of the topoisomerase genes leading to inhibition of DNA replication for the use of humans or veterinary medicine [133]. The most common resistance mechanism to quinolones due to decreased permeability of the antimicrobial to the cell, efflux pumps, or alterations in the target enzymes DNA gyrase (the gyrA and gyrB genes) or topoisomerase genes (parC and par E genes). Most of these mutations occur in the quinolone resistance determining region (QRDR) which is a conserved site in these enzymes targeted by these antimicrobials [109, 133, 134]. For instance, *E. coli* resistance due to alterations in genes that encode subunits of the quinolone targets DNA gyrase (in the *gyrA* and *gyrB* genes) and topoisomerase IV (in *parC* and *parE* genes). Quinolones block the reaction and trap gyrase or topoisomerase IV as a drug- enzyme-DNA complex with subsequent release of lethal double stranded DNA breaks [134].

Chloramphenicol

The use of chloramphenicol in veterinary medicine has been banned from U.S and European Union (EU) for food animals due to its possible toxic effects on humans arising from chloramphenicol residues in carcasses of food animals, and limited uses to pets and non-food-producing animals [109, 135]. Chloramphenicol is a highly specific and potent inhibitor of protein biosynthesis of the bacteria due to inhibition of peptide chain elongation [110, 135]. The most frequently resistance mechanism of bacterial to chloramphenicol is enzymatic inactivation by chloramphenicol acetyltransferases (CATs) [109, 111], or non-enzymatic resistance by efflux pumps such as *floR* and *cmlA* [136]. In addition, the resistance gene *floR* is in the class I integron located in *Salmonella* Genomic Island1 (SGI-1) [124, 137, 138] . Florfenicol which is a fluorinated structural analog of thiamphenicol and chloramphenicol was approved for the treatment of bovine respiratory disease worldwide. Resistance florfenicol has been detected in clinically ill cattle and chickens [139, 140]. However, there are reports on other of chloramphenicol resistance mechanisms such as inactivation by phosphotransferases, mutation of the target site and permeability barriers [135].

Conflict of Interest

The authors declare that they have no any conflict of interest

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