REVIEWE ARTICLE

MICROBIAL LANDSCAPE AND ANTIBIOTIC RESISTANCE IN SOUTH BENIN'S AGRO-PASTORAL SECTOR: TOWARDS SUSTAINABLE DEVELOPMENT

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ABSTRACT

Introduction: Benin's agropastoral sector, encompassing poultry, cattle, and fish, faces significant challenges due to insufficient information on livestock management and the emergence of pathogenic resistant strains such as Salmonella spp and Escherichia coli, which could potentially transfer to humans. Breeders often lack adequate training and resort to self-administering animal treatments, leading to a rapid increase in foodborne illnesses. This poses a severe threat to public health, resulting in high rates of mortality and morbidity in both animals and humans from zoonotic bacteria resistant to antibiotics. **Objective**: Given the urgent need to address these challenges, this review aims to contribute to the documentation of agropastoral farms in southern Benin. Specifically, it focuses on elucidating the microbiological profile of predominant strains, assessing antibiotic resistance patterns, and identifying the constraints hindering the development of the agropastoral sector. **Methods**: To achieve these objectives, a comprehensive review of existing literature on Benin's agropastoral sector, microbiological profiles of pathogenic strains, and antibiotic resistance patterns was conducted. Relevant databases were searched, and studies addressing the specified objectives were critically analyzed to extract pertinent information. **Results**: The review revealed a concerning microbiological profile characterized by the prevalence of pathogenic strains such as Salmonella spp and Escherichia coli in Benin's agropastoral sector. Furthermore, antibiotic resistance among these strains was found to be widespread, posing significant challenges to both animal health and public safety. The excessive use of antibiotics in breeding practices was identified as a primary driver of antibiotic resistance, highlighting the urgent need for improved management practices and regulatory measures. **Conclusion**: In conclusion, this review underscores the critical need for enhanced documentation and understanding of Benin's agropastoral sector, particularly regarding microbiological profiles and antibiotic resistance patterns. Addressing the identified constraints, including inadequate training among breeders and the unregulated use of antibiotics, is essential to safeguarding both animal and human health in southern Benin. Efforts to promote sustainable livestock management practices and mitigate antibiotic resistance are imperative for the long-term viability and development of the agropastoral sector in the region.

Keywords: *Agropastoral farm, microbiological profile, antibiotic resistance, Benin.*

1.INTRODUCTION

Food safety is threatening nowadays from a public health point of view because foodborne illnesses continue to increase each year, particularly affecting children under five years old and the elderly (WHO, 2020). Bacterial agents are the majority cause of morbidity and mortality linked to foodborne infections (Ateba *et al.*, 2008). A certain number of pathogenic bacteria have been associated with food contamination of animal origin; these include, among others, *Salmonella spp*, *Campylobacter spp*, *Staphylococcus aureus*, *Listeria monocytogenes*, *Clostridium perfringens*, *Clostridium botulinum*, *E. coli* 0157: H7 and enterohemorrhagic *E. coli* (EHEC) (Akoachere *et al.*, 2009; Movassagh *et al.*, 2010; Ahouandjinou *et al.*, 2016). Livestock remains an essential resource for food, nutrition, income, and livelihoods for most people (FAO, 2016).

In Benin, livestock farming is the second largest agricultural activity after crop production and contributed to 13.44% of agricultural GDP in 2016 (DE, 2017; Dognon *et al.*, 2018). Traditional livestock production systems remain dominant. It is divided into the sedentary system and the transhumant system. The national livestock includes domestic animal species (cattle, sheep, goats, pigs, poultry, etc.) and unconventional species such as grass cutters (Youssao, 2015; DE, 2016; Dognon *et al.*, 2018). The increase in animal production in recent decades was possible thanks to the use of veterinary drugs in modern livestock farming (Moretain, 2005; Tatsadjieu *et al.*, 2009), either as a curative, preventive treatment or, in certain extreme cases, to compensate for inadequacies in terms of hygiene in livestock farming as food additives or growth promoters in animals (Sanders, 2005). Various antibiotics are produced worldwide for animals to cure or prevent disease or promote their growth to increase yield (Sanders *et al.*, 2017; Batie, 2018). The unreasonable use of antibiotics in veterinary and human medicine is considered the most critical factor for the appearance and spread of antibiotic-resistant microorganisms (Mousse, 2016; Ouedraogo *et al.*, 2017). Antibiotic-resistant pathogens are present in all microbial communities (Acar & Moulin, 2013), thus representing a serious threat to human and animal populations, given their potential transfer to humans through the food chain (WHO, 2014; Heuer *et al.*, 2006). The

problem of antibiotic resistance is increasingly becoming a global ecological difficulty where the commensal intestinal flora of animals is considered a reservoir of resistance genes potentially transmissible to humans (Bouvarel *et al.*, 2005). Faced with these challenges, it is vital to provide solutions to encourage the appropriate use of antibiotics by establishing a well-coordinated government regulation and surveillance system, coupled with the proper research support that will effectively aid resistance management antibiotics in common microorganisms such as *Escherichia coli* and species of the genus *Salmonella spp* responsible for zoonoses (WHO, 2011). Therefore, this review article was written to assess the development level of agropastoral farms in southern Benin. It also presents the microbiological profile of the main strains predominant on these farms and addresses the resistance of the pathogenic strains isolated from them to antibiotics.

2. METHODS

2.1 Literature Search Strategy

A comprehensive literature search was performed using various electronic databases, including PubMed, Google Scholar, Web of Science, and ScienceDirect. The search terms included "agropastoral farms", "Benin", "microbiological profile", "antibiotic resistance", "Salmonella", "Escherichia coli", and their combinations. The search was limited to articles published in English and French languages, with no restriction on the publication year.

2.2 Inclusion and Exclusion Criteria

Studies were included if they met the following criteria: (1) conducted in Benin, (2) focused on agropastoral farms (poultry, cattle, or fish farming), (3) reported microbiological profiles, including the presence of Salmonella spp. and Escherichia coli, (4) investigated antibiotic resistance patterns of isolated strains, and (5) addressed constraints or challenges faced by the agropastoral sector in Benin. Review articles, case reports, and studies conducted outside Benin were excluded.

2.3 Data Extraction and Analysis

Relevant data from the included studies were extracted and tabulated using a standardized data collection form. The extracted information included study characteristics (authors, year of publication, study design), agropastoral sector (poultry, cattle, or fish farming), microbiological profiles (prevalence of Salmonella spp. and Escherichia coli), antibiotic resistance patterns, and identified constraints or challenges. A narrative synthesis of the extracted data was performed, and findings were summarized and critically analyzed.

2.4 Quality Assessment

The methodological quality of the included studies was assessed using appropriate tools, such as the Joanna Briggs Institute (JBI) Critical Appraisal Checklists for different study designs. Studies with significant methodological limitations were considered cautiously during data synthesis and interpretation.

3. RESULTS

3. Overview of the livestock sector in Benin

3.1. Cattle sector in Benin

Cattle breeding is practiced in Benin in two central systems: the semi-improved system on state farms and the traditional or extensive system on private farms (Youssao, 2015). The national cattle herd is estimated at 1,773,157 head, the majority of which 76.79% are located in the departments of Alibori, Borgou, and Atacora (DSA, 2021). Most of the livestock is held by the extensive system. The breeds raised in this system are the Lagunaire, the Somba, the Borgou, and the zebus, mainly the Peulh, White Fulani, and M'Bororo Rouge zebus (Youssao, 2015; Kassa *et al.*, 2016). It should be noted that livestock production is mainly limited by food shortages, especially during the dry season (Musco *et al.*, 2016). That of cattle is mainly based on the extensive use of natural pastures, which are only available during the rainy season (Lesse *et al.*, 2016). One of the significant challenges facing the cattle industry is ensuring populations have sufficient meat and milk consumption in quantity and quality while developing exports (FAO and ECOWAS, 2017; Sounon *et al.*, 2019).

3.2. Poultry sector in Benin

The poultry sector in Benin contributes, among other things, to improving the livelihoods of rural households involved in traditional poultry farming. Two forms of poultry farming are practiced. This includes traditional and modern poultry farming. Traditional poultry farming plays a crucial role in food security and contributes to the livelihoods of rural populations on religious, social, and cultural levels (Youssao *et al.*, 2013). It constitutes a significant source of protein for farmers and urban populations in Benin. It concerns chickens, guinea fowl, ducks, turkeys, and geese (DE, 2016).

It also contributes to improving the income of small farmers in rural areas, particularly women, and serves as organic fertilizer for agriculture. Modern or commercial (intensive) breeding, for its part, allows the creation of jobs and income. The sector also contributes to food security and self-sufficiency (FAO, 2015). There were 10.250541 heads of local chickens, 1.348029 heads of guinea fowl, 356,098 heads of laying hens, and 117,750 heads of broiler chickens (DSA, 2021). However, despite the positive potential for its development, the poultry industry is not competitive enough with imports from the West and other regions of the world. In reality, the development of a domestic supply in Benin faces two significant challenges: The first is the increase in imports from the European Union and other countries, such as the United States of America or Brazil (Rudloff and Schmieg, 2016). The specific constraints of the poultry sector in Benin constitute the second.

3.3. Fish sector in Benin

The fish farming sector meets protein demands in Benin's rural and urban areas (Diogo *et al.*, 2018). It occupies 25% of the agricultural active population and 15% of the total active population (Baris et al., 2016). It also constitutes a significant source of income and protein for fishing communities (Rurangwa et al., 2014). Nationally, there are 10,593 fish holes, representing the most widespread infrastructure, far ahead of ponds (3,509) and basins (1,868). It should also be noted that some aqua-culturists operate "Acadja," which are installed on the waterways of the departments of Ouémé (877), Atlantique (363), Mono (73), and Littoral (7) (DSA, 2021). Several improved fish farming systems are being implemented, including monoculture and polyculture. Monoculture is the most common type of improved fish production system and consists of raising only one species of fish in the fish farming system. It has the advantage of preventing predation between species, allows the control of density in the device, and promotes the reduction of costs linked to food because food preference is limited to a single species. The fish species commonly farmed are Tilapias, Clarias, and Heterotis. Polyculture involves raising several fish species associated with the same fish farming system. The species generally associated are Tilapias and Clarias on the one hand and Tilapias and Heterotis on the other. The advantages recognized by fish farmers for this fish farming system are to take advantage of all the nutrients present in the system, to control the excessive reproduction of Tilapias, and to limit the risk of disease.

2. General information on the *Salmonella* genus

2.1. History

Salmonella constitutes enterobacteria, so named to honor the American veterinarian Daniel Elmer Salmon (Bergeron, 2009). Indeed, it was in 1880 that Eberth observed the typhoid bacillus in sections of the spleen and lymph node, the culture of which was possible in 1884 by Gaffky. The genus Salmonella was used after the American bacteriologist Daniel Salmon and some colleagues isolated a bacterium from pigs in 1886, which was the cause of swine fever (swine cholera). Then, in 1896, Widal demonstrated the antigenic diversity of Salmonella strains using a new test called serodiagnosis. Since then, numerous serovars have been identified (Camart-perie, 2006).

2.2. Classification and taxonomy

Salmonella represents an enterobacterium and constitutes the most complex and large genus of the Enterobacteriaceae family. The nomenclature of the *Salmonella* genus is as follows:

Kingdom: Bacteria
Phylum: Proteobacteria
Class: Gamma proteobacteria
Order: Enterobacterial
Family: *Enterobacteriaceae*
Genus: *Salmonella*
Species: *Salmonella enterica*
Salmonella bongori
Salmonella subterranea.

The *Salmonella enterica* species is subdivided into six subspecies (I-VI) (Brenner *et al.*, 2000), taking into account their genomic linkage and their biochemical properties (Reeves *et al.*, 1989). Roman numerals designate the subspecies: I (*S. enterica* subsp. *enterica*); II (*S. enterica* subsp. *Salamae*); IIIa (*S. enterica* subsp. *Arizonae*); IIIb (*S. enterica* subsp. *Diarizonae*); IV (*S. enterica* subsp. *Houtenae*) and VI (*S. enterica* subsp. *Indica*). Members of *Salmonella enterica* subsp *enterica* (I) are found primarily in mammals and contribute to approximately 99% of Salmonella infections in humans and warm-blooded animals. The species S. bongori is found in the environment and cold-blooded animals and is rare in humans (Brenner *et al.*, 2000).

2.3. Epidemiology

2.3.1. Reservoir

Salmonella can be isolated from the intestines of many animal species. These are zoonotic agents for which animals constitute a reservoir, and dissemination in the environment comes mainly from fecal contamination (Hanes, 2003).

The more animals are concentrated in a particular area, the more difficult it is to control transmissions between them. Salmonella can also survive very long periods in the external environment (Gray & Fedorka-Cray, 2001). Some are exclusively adapted to humans, causing one or more severe pathologies. These are mainly Salmonella typhi and paratyphi, agents of typhoid and paratyphoid fevers (Hu & Kopecko, 2003). Others can be found in all vertebrates, and serotypes can be classified according to the target animal species. A whole series of serotypes can be of interest to animal species. Among these serotypes, we must mention S. dublin in cattle, Choleraesuis, Typhisuis in pigs, *Abortusequi* in horses, *Abortusovis* in sheep, and Specific *gallinarum* in poultry.

2.3.2. Dynamism and mode of contamination

Salmonella is present in a latent state or causing a subclinical disease and can reach humans either through food (the most common route) or direct or even indirect contact. Animals constitute a reservoir, and dissemination in the environment mainly comes from fecal contamination (Murray, 2000; Hanes, 2003). Human contamination by non-typhoid salmonella occurs mainly through consuming contaminated foods (eggs and egg-based preparations, poultry, cold meats, raw milk cheeses), consumed raw or insufficiently cooked (Weill, 2008). The mass production of food products and their large-scale distribution also favors the dissemination of salmonella and the occurrence of large-scale epidemics (Sánchez-Vargas *et al.*, 2011). Furthermore, within farms, the more animals are concentrated in a specific area, the more difficult it is to control transmissions between them (Korsak, 2004). Rodents and insects can also be an essential source of Salmonella on a farm (Yao *et al.*, 2019).

2.4. Virulence factors

Several *Salmonella* virulence factors must be distinguished, such as adhesion, invasion, and toxin genes. These factors are grouped in some regions of the chromosome called "*Salmonella* pathogenicity islands" (SPI) (Santos *et al.*, 2003), which can be located on the chromosome or a plasmid. Two characteristics of Salmonella pathogenesis, such as host invasion and intracellular proliferation, are directly linked to SPI genes. SPI-1 contains invasion genes, while SPI-2 is required for intracellular pathogenesis and plays a crucial role in systemic S. enterica infections (Hansen-Wester & Hensel, 2001).

2.5. Pathogenesis and pathogenicity of *Salmonella*

Most *Salmonella* strains can invade, multiply, and survive in host cells (Eng *et al.*, 2015). The purpose of *Salmonella* infection depends mainly on three factors: the infective dose, the virulence factor that influences the host cell, and the level of immunity (Venter *et al.*, 1994). According to Sheorey & Darby (2008), Salmonella strains are grouped into typhoid and non-typhoid *Salmonella* based on the clinical pattern of salmonellosis. The various manifestations noted during infection in humans are typhoid and paratyphoid fevers, gastroenteritis, septicemia, and extra-digestive complications. In animals, particularly in calves, the infection is acute and septicemic, marked by high fever, pneumonia, arthritis, enteritis, and lethality, whereas in adult cattle, salmonellosis is less frequent. It generally constitutes a secondary infection added to another disease or follows the appearance of clinical cases of salmonellosis on the same farm. The primary clinical symptoms are abundant diarrhea mixed with blood or intestinal epithelium, as well as violent abdominal pain. Pregnant cows can abort and shed the pathogen in large quantities.

3. General information on *Escherichia coli*

3.1. History

Escherichia coli was discovered by the Austrian German pediatrician Theodor Esherich from a human stool sample in 1885. It is a bacillus approximately 2-3 µm long and 0.6 to 0.7 µm in diameter, Gram-negative, non-sporulating, generally motile, family Enterobacteriaceae, typical host in the digestive tract of humans and animals (Hufnagel *et al.*, 2015). Its current name was given to it in 1919 by Castellani and Chambers. During the 1920s and 1930s, many researchers worked to identify specific types of *E. coli* causing enteropathy. However, no significant progress was made until Kauffmann's development in the 1940s of a precise serotype scheme (WHO, 1980). Since the 1950s, several strains of *E. coli* belonging to specific serotypes have been identified in humans and animals as being pathogenic strains responsible for various conditions such as diarrhea, severe systemic infections, and even fatal (Nataro & Kaper, 1998; Kaper *et al.*, 2004). It should also be noted that *E. coli* was responsible in 1982 for hemolytic uremic syndrome (HUS), leading to the death of around forty people in the United States. This situation illustrates the remarkable adaptability of *E. coli* to its environment thanks to genetic exchanges causing the appearance of pathogenic strains (Julien, 2004). Furthermore, E. coli is a central biology pillar (Milon, 1993). It was at the heart of the pioneering experiments of the 1950s-1970s, which laid the foundations of bacterial genetics and molecular biology (D'Ari & Sezonov, 2008).

3.2. Taxonomy and classification

The genus *Escherichia* belongs to the family *Enterobacteriaceae* in the kingdom of prokaryotes. It includes five species: *E. coli*, *E. albertii*, *E. furgusonii*, *E. hermannii* and *E. vulneris*. Each of its species has specific biochemical characteristics, allowing them to be distinguished from one another (Vimont, 2007). Furthermore, it should be clarified that the genera

Shigella and Escherichia are identical to modern criteria of bacterial taxonomy. The classification of the species is as follows:

Kingdom: Bacteria
Phylum: Proteobacteria
Class: Gammaproteobacteria
Order: Enterobacterial
Family: *Enterobacteriaceae*
Tribe: *Escherichiae*
Genus: *Escherichia*
Species: *Escherichia coli*,
Escherichia albertii,
Escherichia furgusonii,
Escherichia hermannii,
Escherichia vulneris.

3.3. Epidemiology

3.3.1. Habitat and host of *Escherichia coli*

The *E. coli* species belongs to the commensal microflora of humans and many animals. It is a colonizing bacterium in the digestive tract of warm-blooded animals (omnivores, carnivores, herbivores, and birds) and reptiles (Gordon & Cowling, 2003). *E. coli* nestles, more particularly in the mucus covering the epithelial cells of the digestive tract wall, which constitutes an ecological niche conducive to its development due to its conditions of temperature, humidity, and nutrient availability (Smati *et al.*, 2015).

✓ **Secondary habitat**

E. coli is released into the environment through feces and can also be found in environmental waters through effluents (sewage, slurry, or livestock manure) or animal droppings. Farmed or wild animals (Smati *et al.*, 2015). It is essential to specify that certain strains of commensal *E. coli* are very well adapted to coexistence with the host, but others are pathogenic for their hosts (Conrad *et al.*, 2016). Their presence in the environment is an indicator of fecal contamination. This is why it is necessary to systematically detect it in water used for drinking, food preparation, or swimming (Smati *et al.*, 2017). This naturalized population manages to survive as long in the environment outside their host, thus colonizing it (Walk *et al.*, 2007; Ishii *et al.*, 2006), illustrated by Figure 1. When this naturalized population establishes itself over time, it becomes a new autochthonous microbial community.

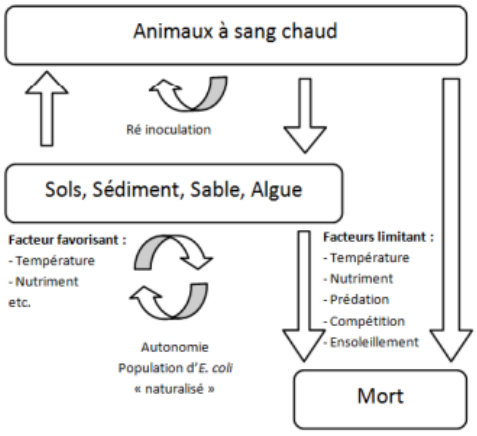

Figure 1: Life cycle of *E. coli* (after Ishii and Sadowski 2008).

3.3.2. Reservoir and mode of transmission

Transmission is human-to-human when people are in close contact with patients and is more significant when general hygiene is poor and contacts are close (Sugiyama *et al.*, 2005). Waterborne transmission refers to epidemics generally associated with the consumption of untreated water, drinking, or accidental ingestion of water while swimming (Jackson *et al.*, 1998). Meat products, undercooked beef, other slaughtered animals, unpasteurized milk and dairy products, fruits, and raw products are sources of many Escherichia coli infections. (Vernozy-Rozand *et al.*, 2005; Paton, 2001; Allerberger, 2001; Baranyi & Roberts, 1994). The transmission mode through direct or indirect contact with farm animals or their droppings leads to a high rate of healthy carriers in the population living in permanent contact with animals (Evans *et al.*, 2002); certain strains of *E. coli* produce toxins that are pathogenic for animals and can cause diarrhea in calves. Domestic ruminants, particularly cattle, appear to be the main reservoirs of STEC strains pathogenic for humans, particularly EHEC O157 strains (Gyles, 2007). In these animals, the digestive carriage and excretion of EHEC strains are

most often asymptomatic, and direct or indirect contact with their feces constitutes the main route of contamination of humans. Figure 2 summarizes the routes of *Escherichia coli* contamination.

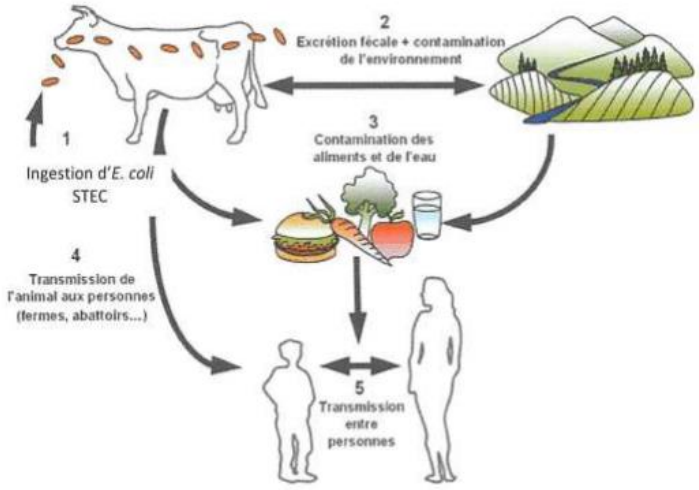

Figure 2: Routes of contamination by *Escherichia coli* (Marc, 2000)

3.4. Pathophysiology

3.4.1. Intestinal infections

Most clinical signs are due to the production of toxins (cytotoxins, enterotoxins). However, the infectious process is multifactorial and depends on bacterial and host factors (Paton & Paton, 1998). The critical steps in this process lie in the fact that strains of Pathogenic *E. coli* must resist stomach acidity after ingestion. Consequently, a stage of colonization of the digestive tract becomes essential. There are strains of *E. coli* that can produce attachment/effacement lesions. However, the toxins produced by the bacteria must subsequently cross the intestinal epithelium before joining the circulatory system and reaching specific receptors located on the surface of endothelial cells, mainly in the intestine. Toxins cause the death of target cells by stopping protein synthesis. The role of bacteria and toxins in activating the immune system is also suspected (Heyderman *et al.*, 2001).

3.4.2. Extra-intestinal infections

The strains causing extra-intestinal infections have virulence factors that contribute to the bacteria passing through the different stages of the physio-pathological process, such as adhesion, invasion, and multiplication (Johnson, 1991). During the infectious process, adhesion to epithelial cells represents a fundamental step. It allows bacteria to multiply and colonize the mucosa, stages followed by the invasive phase, the preamble to the infection. The fixation of *E. coli* on epithelial cells depends on the expression of adhesion proteins on its surface called adhesins, particularly fimbriae. The specific adhesion properties linked to fimbriae constitute one of the essential factors in the pathogenicity of uropathogenic *E. coli*. During the invasion phase, the bacteria can also secrete toxins responsible for significant tissue damage in the host, particularly a hemolysin, which causes the lysis of red blood cells and causes the formation of selective channels on membrane surfaces. If, at the human level, the virulence factors present in the strain are essential for the development of an extra-intestinal *E. coli* infection, the prediction of the initial severity of the infection and its evolution cannot be based on the sole study of the intrinsic virulence of the strain, and the taking into account of factors linked to the host (Maslow *et al.*, 1993; Martinez *et al.*, 2006).

3.5. Pathogenicity of *Escherichia coli*

Not all strains of avian *E. coli* are pathogenic. Strains pathogenic to poultry, called avian pathogenic *E. coli* (APEC), generally belong to specific serogroups, particularly serogroups 078, 01, and 02. Moreover, to a certain extent, they are found in serogroups 015 and 055 (Chart *et al.*, 2001). They are responsible for colibacillosis. These are undoubtedly the most frequent and critical bacterial infections in avian pathology, which most often affect broiler chickens, whose clinical signs (embryo mortality, respiratory problems, lameness, drop in laying) and lesions can vary. (Stordeur *et al.*, 2004). Colibacillosis is responsible for major economic losses in poultry farms and represents a significant cause of seizures at the slaughterhouse. It leads to numerous antibiotic treatments with the risk of emergence of resistance (Robineau *et al.*, 2010).

4. General information on antibiotics

4.1. History

Antibiotics represent one of the essential therapeutic classes that have revolutionized human medicine. Ernest Duchesne was the first in 1887 to notice the antibacterial power of molds, but his discovery did not undergo significant

development. In 1928, the official discovery of penicillin was made by Sir Alexander Fleming. The latter cultivated *Staphylococci* on Petri dishes and observed an inhibition of the growth of these bacteria on dishes contaminated by a fungus, Penicillium. He hypothesized that this fungus could synthesize a substance with antibacterial properties, which he calls "penicillin." He published his discovery in 1929. However, it was not until the Second World War that its therapeutic use began, following the isolation and purification of the molecule by the chemists Chain and Florey, which favored its clinical use (Muller, 2017). Since then, penicillin has been used extensively, saving millions of lives. Several other molecules were subsequently discovered and promote the treatment of many infections previously considered fatal. Figure 3 presents the timeline of the discovery of the main classes of antibiotics.

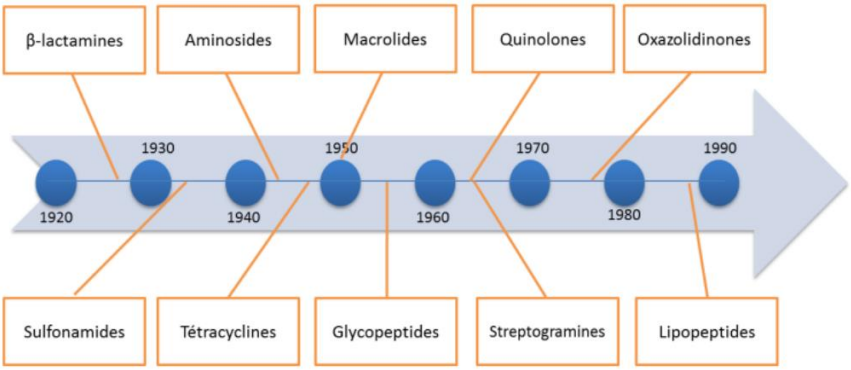

Figure 3: Timeline of the discovery of the main classes of antibiotics (Muller, 2017).

4.2. Definition

From the Greek anti, which means "against," and bios, which means "life," an antibiotic is a natural or synthetic substance that destroys or blocks the growth of bacteria (Vuillemin, 1890). According to Waksman (1945), antibiotic refers to a chemical substance of microbial origin with antimicrobial powers. This definition of antibiotic has been improved because molecules obtained by synthesis or by chemical modification of a natural molecule can be endowed with the same properties, and the appearance of synthetic antibiotics led to a new clarification stated in 1957 by Turpin and Velu. The latter defines the antibiotic as any chemical compound produced by a living organism or produced by synthesis with a high chemotherapeutic coefficient whose therapeutic activity is manifested at very low doses in a specific manner by inhibiting certain processes. Vital concerning viruses, microorganisms, or even certain multicellular beings (Cohen & Jacquot, 2008). It should be noted that the antibiotic refers to the name of all-natural substances produced by microorganisms and their synthetic analogs, capable of stopping the multiplication of bacteria (bacteriostatic) or destroying them (bactericidal) (Manvila *et al.*, 1995).

4.3. Classification

The classification of antibiotics is based on several criteria: the origin (biosynthesized), the chemical nature (amino acid derivatives, heterotic or polycyclic), the mechanism of action, and their spectrum of action. Among these classifications, the one generally most exploited concerns the family. Indeed, this criterion is based on common characteristics such as chemical composition or origin, similar or very close spectrum of action, identical bacterial targets, bacterial resistance, and closely related adverse effects (Yala *et al.*, 2001).

4.4. Mode of action

- ✓ **Inhibition of cell wall synthesis**

The wall is composed mainly of peptidoglycan (PG), or mucopeptide, a polysaccharide macromolecule formed by a regular succession of acetoglucosamine and N-acetylmuramic acid. These amino acids are formed into small peptides, and these are linked together by peptide bridges, giving excellent rigidity to the whole. This transpeptidation is the last step in synthesizing the bacterial cell wall and is carried out under the influence of an enzyme, transpeptidase. However, when bacteria are deprived of their wall, they become fragile and defenseless against mechanical attacks and osmotic disturbances (Bourin & Lindahl, 1993). Antibiotics such as penicillins, carbapenems, and cephalosporins can block the cross-linking of peptidoglycan units by inhibiting the formation of peptide bonds catalyzed by PLPs (penicillin-binding proteins) (Josephine *et al.*, 2004). Most antibiotics belonging to the glycopeptide class can inhibit bacterial growth by inhibiting PG synthesis. They inhibit PG synthesis by binding to PG units and blocking transglycosylase and transpeptidase activity (Kahne *et al.*, 2005).

- ✓ **Disorganization of the structure or function of the cell membrane**

The classes of antibiotics that cause damage to bacterial cell membranes are linked to each microbial group based on differences in the types of lipids in their cell membranes. As an illustration, Daptomycin depolarizes the calcium-dependent membrane, which leads to the cessation of macromolecular synthesis and the disruption of the cell membrane in bacteria (Alborn *et al.*, 1991). Polymyxins cause the disintegration of the bacterial cell membrane by efficiently binding to the lipid moiety of the lipopolysaccharide in the bacterial cell (Falagas *et al.*, 2010).

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✓ **Inhibition of nucleic acid synthesis**

Antibiotics interfere with nucleic acid synthesis by blocking replication or stopping transcription. DNA replication results in unwinding the traditional double helix structure, a process facilitated by a helicase (Gale *et al.*, 1981). For example, quinolone antibiotics interfere with the functionality of the helicase enzyme, thus preventing the enzyme from playing its role in unwinding DNA. This action alters the DNA replication and repair process of bacteria sensitive to quinolones (Chen *et al.*, 1996). It should be noted that antibiotics whose mode of action is inhibiting nucleic acid synthesis also target the bacteria's topoisomerase II and topoisomerase IV. Disruption of the activities of these enzymes in bacteria negatively influences RNA polymerase, preventing RNA synthesis. Quinolones that inhibit bacterial nucleic acid synthesis in this manner do not interact with mammalian RNA polymerase, making them antagonistic to Gram-positive and some Gram-negative bacteria.

✓ **Inhibition of protein synthesis**

Antibiotics that inhibit protein synthesis are among the broadest classes of antibiotics and are divided into two subclasses: 50S inhibitors and 30S inhibitors. Erythromycin, clindamycin, lincomycin, chloramphenicol, linezolid, etc., constitute antibiotics that are part of the inhibitors of the 50S ribosome (Katz & Ashley, 2005). Generally speaking, antibiotics that inhibit the 50S ribosome do so by physically blocking either the initiation phase of protein translation or the elongation phase of protein synthesis, where the incoming amino acid is linked to the nascent peptide chain. growing (Patel et al., 2001). Oxazolidinone members are examples of antibiotics that block the initiation of protein translation (Patel et al., 2001), whereas macrolides such as lincosamide and streptogramin block protein synthesis by inhibiting the phase elongation of mRNA translation (Vannuffel & Cocito, 1996). Therefore, these latter groups of antibiotics would be ineffective when the elongation exceeded a critical length (Tenson *et al.*, 2003). 30S ribosome inhibitors act mainly by preventing access of aminoacyl-tRNAs to the ribosome. Examples of antibiotics that possess this type of functioning include tetracycline, streptomycin, spectinomycin, etc. (Hong et al., 2014). It is important to note that tetracycline inhibits specific proteins at the level of 50S ribosomes (Epe & Woolley, 1984). Regarding ribosome inhibitors, the subclass of aminoglycosides, of natural origin, is the only one to be largely bactericidal. Macrolides, streptogramins, spectinomycin, tetracyclines, and chloramphenicol are generally bacteriostatic. On the other hand, some of these ribosome-inhibiting antibiotics, which generally have a bacteriostatic action, could be bactericidal under certain conditions linked to a mode specific to the species or the treatment. This is the case of chloramphenicol, known to be typically bacteriostatic, which effectively kills *S. pneumoniae* and *Neisseria meningitidis* (Rahal & Simberkoff, 1979), as well as *H. influenza* (Goldstein *et al.*, 1990).

✓ **Blockage of main metabolic pathways**

Regarding blocking the main metabolic pathways, it should be noted that certain antibiotics, namely sulfonamides and trimethoprim, imitate a substrate necessary for the cellular metabolism of bacteria. This deception causes the bacterial enzymes to attach to the antibiotic rather than the normal substrate. Specifically, sulfonamides are the tetrahydrofolate necessary for synthesizing folic acid in bacterial cells (Talaro & Chess, 2012). Folic acid is essential in the metabolism of nucleic acids and amino acids.

Table 1: Mode of action of the main classes and molecules of antibiotics (UE, 2010).

Classes	Molecules	Antibacterial mode of action	Activity spectrum
Sulfamidae	All substances belonging to the sulphonamide group	They inhibit folate synthesis through the action of competitive inhibitors of dihydropteroate synthetase.	Gram-positive cocci
Quinolones	Oxolinic acid, ofloxacin, norfloxacin, norfloxacin, enrofloxacin, flumequine, marbofloxacin	They inhibit bacterial DNA gyrase or topoisomerase IV and, therefore, inhibit DNA replication and transcription.	Broad spectrum on *Mycobacterium tuberculosis* (fluoroquinolones, in combination with other antimycobacterial)
Bêta-lactams	Amoxicillin, ampicillin, benzylpenicillin, cefalexin, cefacetrile, cefalonium, cefapirin, cefaperadone, cefquinone, ceftiofur, cefazolin, cloxacillin, cefoperazone, penethamate, dicloxacillin, nafcillin, oxacillin	They disrupt the synthesis of the peptidoglycan layer of bacterial cell walls by binding to proteins that contribute to this synthesis.	Gram-positive cocci Gram-positive and Gram-negative bacteria, *Treponema pallidum*, *Borrelia*
Tetracyclines	Chlortetracycline, doxycycline, oxytetracycline, tetracycline	They bind to 30S ribosomal subunits, inhibiting aminoacyl-tRNA binding to the mRNA-ribosome complex.	*Treponema pallidum, Chlamydia, Borrelia, Rickettsia, Plasmodium falciparum*
Aminoglycosides	Dihydrostreptomycin, gentamicin, kanamycin, neomycin, streptomycin, paromomycin, apramycin, spectinomycin	They bind to the 30S subunit of the bacterial ribosome (some bind to the 50S subunit), inhibiting the translocation of peptidyl-tRNA from the A site to the P site and causing misreading of the mRNA.	Gram-positive and Gram-negative bacteria (including *Pseudomonas aeruginosa*), *Mycobacterium tuberculosis*
Phenols	Thiamphenicol, florfenicol	They bind to the 50S subunit of the ribosome, preventing the formation of peptide bonds.	*Neisseria meningitidis, Salmonella Typhi*
Macrolides	Erythromycin, spiramycin, tylosin, tilmicosin, azithromycin, tulathromycin, tylosin, tildipirosin	They bind reversibly to the 50S subunit of the bacterial ribosome by inhibiting peptidyl-tRNA translocation.	Gram-positive cocci, Treponema pallidum, intracellular pathogens, *Mycoplasma, Plasmodium falciparum*

Lincosamides	Lincomycin, pirlimycin	They bind to the 50S subunit of the ribosome, inhibiting transpeptidation/translocation.	Gram-positive cocci, anaerobic (clindamycin), *Plasmodium falciparum* (clindamycin)
Polypeptides	Bacitracin, colistin, tyrothricin	They react strongly with membrane phospholipids and disrupt the functioning and permeability of these membranes.	Gram-positive and Gram-negative bacteria, Bacillus polymyxa, Bacillus subtilis
Orthosomycins	Avilamycin		Gram-positive bacteria
Rifamycins	Rifamycin SV, rifaximin, rifampicin	They block the synthesis of messenger RNA.	Gram-positive and Gram-negative cocci, broad-spectrum Gram-positive bacilli
Ionophores	Salinomycin, monensin		Gram-positive, coccidiostats bacteria
Novobiocin	Novobiocin	They inhibit DNA replication.	Gram-positive and Gram-negative cocci, Gram-positive bacilli, *Haemophilus, Pasteurella*
Pleuromutilins	Tiamulin, valnemulin	They inhibit protein synthesis at the 50S unit of ribosomes.	Broad spectrum

4.5. Use of antibiotics in breeding

4.5.1. Objective of the use of antibiotics in breeding

Four objectives are fundamentally targeted for the use of antibiotics in livestock farming. These are the curative purpose, the prophylactic purpose, the metaphylactic use, and the zootechnical use. Antibiotics are all used for curative purposes in order to eradicate a present infection (Corpet, 1987). This treatment method aims to reduce bacterial excretion and contributes, in some instances, to obtaining a bacteriological cure and, during zoonotic infections, can help avoid human contamination. For prophylactic purposes, antibiotics are used to prevent infection. In this case, they make avoiding a potential risk situation possible. This anticipatory approach can prevent symptoms and avoid a drop in production. In addition, the metaphylactic use of antibiotics and adaptation to group medicine makes it possible to administer the same remedy to several individuals subjected to the same contaminating agent, whether or not they present symptoms (Labro, 2012). The zootechnical use of antibiotics aims to use them as additives to the ration to improve growth. This method of use has been banned in the European Union (EU) since 2006 because it poses enormous risks, such as the selection and spread of resistant bacteria.

4.5.2. Different ways of using antibiotics in breeding

In the livestock sector, veterinarians control the use of antibiotics. The use of antibiotics in breeding is under the control of veterinarians. Antibiotics are available in various galenic formulations to meet the requirements of different routes of administration. Some antibiotics can be administered intravenously, intramuscularly, or subcutaneously. Local (cutaneous) treatments use ointments and solutions (Sanders *et al.*, 2017). Apart from these routes, other antibiotics are incorporated into animal feed during manufacture and present in powders and solutions administered through drinking water. The most common formulations for individual treatments are tablets, boluses, solutions, or suspensions.

4.6. Main antibiotics used in breeding

It is essential to point out that only a few molecules are available as veterinary antibiotic drugs, although the prominent families of antibiotics are represented. Table 2 presents the list of antibiotics used in veterinary medicine.

Table 2: List of significant antimicrobial agents used in veterinary medicine (OIE, 2015).

Class	Subclass	Antimicrobial agents
Aminocoumarin		Novobiocin
Aminoglycosides	Aminocyclitol	Spectinomycin, Streptomycin, Dihydrostreptomycin
	Aminoglycosides + 2 Deoxystreptamine	Kanamycin, Neomycin, Framycetin, Paromomycin, Apramycin*, Fortimycin, Gentamicin, Tobramycin, Amikacin
Phenicol	Amphenicols	Florphenicol, Thiamphenicol
Ansamycin Rifamycins		Rifampicin, Rifaximin
Bicyclomycin		Bicozamycin
Cephalosporins	First generation cephalosporins	Cefacetrile, Cefalexin, Cefalotin, Cefapyrin, Cefazolin, Cefalonium
	Second generation cephalosporins	Cefuroxime
	Third generation cephalosporins	Cefoperazone Ceftiofur, Ceftriaxone
	Fourth generation cephalosporins	Cefquinome
Lincosamides		Pirlimycin, Lincomycin
Macrolides (C refers to Chemical structure)	Macrolides C14	Erythromycin, Oleandomycin
	Macrolides C15	Gamithromycin, Tulathromycin
	Macrolides C16	Carbomycin, Josamycin, Kismcin, Spiramycin, Tilmicsin, Tylosin, Mirosamycin, Tedecamycin, Tildipirosin, Tylvalosin
	Macrolides C17	Sedecamycin

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Orthosomycins		Avilamycin*
Penicillins	Natural penicillins (including esters and salts)	Benethamine, Penicillin, Benzylpenicillin, Penethamate* (hydriodide), Benzylpenicillin procaïne/ Benzathine penicillin
	Amdinopenicillins	Mecillinam
	Aminopenicillins	Amoxicillin, Ampicillin, Hetacillin
	Aminopenicillin + beta-lactamase inhibitor	Amoxicillin + clavulanic acid, Ampicillin Sulbactam
	Carboxypenicillins	Ticarcillin, Tobicillin
	Ureidopenicillin	Aspoxicillin
	Phenoxypenicillins	Phenoxymethylpenicillin, Phenethicillin
	Antistaphylococcal penicillins	Cloxacillin, Dicloxacillin, Nafcillin, Oxacillin
Phosphonic acid		Fosfomycin
Pleuromutilin		Tiamulin, Valnemulin
Polypeptides		Enramycin, Gramicidin, Bacitracin
	Cyclic polypeptides	Colistin, Polymixin
Quinolones	First generation quinolones	Flumequine, Miloxacin, Nalidixic acid, Oxolinic acid
	Second generation quinolones (fluoroquinolones)	Ciprofloxacin, Danofloxacin, Difloxacin, Enrofloxacin, Marbofloxacin, Norfloxacin, Ofloxacin, Orbifloxacin, Sarafloxacin
	Quinoxalines*	Carbadox, Olaquindox
Sulfamidae	Sulfonamides	Sulfachlorpyridazine, Sulfadiazine, Sulfadimethoxine, Sulfadimidine (Sulfamethazine, Sulfadimerazine), Sulfadoxine, Sulfafurazole, Sulfaguanidine, Sulfamerazine, Sulfadimethoxazole, Sulfamthoxine, Sulfamonomethoxine, Sulfanilamide, Sulfapyridine, Phthalylsulfathiazole, Sulfaquinoxaline
	Sulfonamides + diaminopyrimidines	Sulfamethoxypyridazine, Ormetoprime+ Sulfadimethoxine, Trimethoprime+ Sulfonamide
	Diaminopyrimidines	Baquiloprim, Trimethoprim, Ormetoprim
Streptogramins		Virginiamycin
Tetracyclines		Chlortetracycline, Doxycycline, Oxytetracycline, Tetracycline
Thiostrepton		Nosiheptide

4.7. Risks associated with the use of antibiotics

The significant risks relating to the use of antibiotics lie in the fact that their administration to farm animals can lead to antibiotic residues in foodstuffs from these animals and to the selection of resistant bacteria (Sanders *et al.*, 2017).

4.7.1. Risks related to antibiotic residues

Antibiotic residues cause two significant risks. These are toxicological risks and microbiological risks. On a toxicological level, the effects tested are effects on reproduction and development, mutagenic effects, and allergenic risks. As for the microbiological level, antibiotic residues can modify the resistance to colonization of the intestinal microbiota and the distribution of the main species composing it and contribute to the selection of resistant bacteria and the transfer of resistance genes within the microbiota (Cerniglia & Kotarski, 2005).

4.7.2. Selection of resistant bacteria

At the animal level, antibiotic resistance is interpreted as the consequence of selection pressure induced by antibiotics for veterinary use (Schwarz *et al.*, 2001). The effect of this pressure exerted by the antibiotic is the selection and maintenance of resistant organisms of the bacterial flora. Two factors determine the level of selection pressure: the antibiotic type and the antibiotic's method (dose and duration of use, route of administration) (McEwen & Fedorka, 2002).

5. Bacterial resistance

5.1. Definition

A bacterium is resistant when it can withstand a concentration of antibiotics much higher than that which inhibits the development of the majority of other strains of the same species (Pistes, 2002). Resistance results from the ability of certain bacteria to withstand attack from antimicrobial drugs such as antibiotics, so traditional treatments become ineffective, and infections persist, increasing the risk of spread (WHO, 2015).

5.2. Different types of resistance

5.2.1. Natural resistance

Natural resistance or intrinsic resistance is a characteristic specific to a bacterial species and shared by all strains of this species. It may be due to a chromosomal gene common to all bacteria of the species. For each class of antibiotic, there are bacterial species for which the antibiotic is inactive due to lack of target or access to the target. Consequently, the

absence of a wall in mycoplasmas makes β-lactams inactive towards these bacteria (Mehdi, 2008). It constitutes a criterion for stable species identification (Sabtu *et al.*, 2015).

5.2.2. Acquired resistance

Acquired bacterial resistance to an antibiotic is a phenomenon that appears at the level of strains of a given species, usually sensitive to this antibiotic. The acquisition of a genetic factor reduces sensitivity to the molecule, which is fatal to it. It can, therefore, be done either by chromosomal mutation or by acquisition of genes transferred from another microorganism (Mehdi, 2008). Two significant phenomena underlying the acquisition of resistance through modifications of the bacterial genome should be noted. These are the mutations responsible for endogenous resistance and the horizontal acquisition of foreign genetic material responsible for exogenous resistance. In addition, specific resistances result from the association of a mutation and a horizontal transfer of genes (Guardabassi & Courvalin, 2006).

5.3. Resistance mechanism

5.3.1. Reduced permeability

Gram-negative bacteria have a more complex envelope that is more difficult to pass through, unlike Gram-positive bacteria. Therefore, in Gram-negative bacteria, hydrophilic antibiotics enter the bacteria via transmembrane proteins called porins, while hydrophobic molecules diffuse through the phospholipid layer. Mutations in the genes that code for porins and lead to their loss, the reduction of their size, or even a reduction in their expression will result in the acquisition of low levels of resistance to many antibiotics. For example, we should cite the reduction in the expression of the OmpF porin in E. coli, which reduces sensitivity to quinolones, beta-lactams, tetracyclines, and chloramphenicol. Therefore, the reduction in permeability is a clinically crucial resistance mechanism in Gram-negative bacteria, more precisely in Enterobacteriaceae (Muylaert & Mainil, 2012).

5.3.2. Enzymatic inactivation of the antibiotic

Enzymatic inactivation of the antibiotic is one of the most widespread and effective mechanisms for bacteria. It consists of secreting an enzyme capable of inactivating the antibiotic before it has penetrated the bacteria. The antibiotics are β-lactams, MLS, aminoglycosides, and phenols (Mangin, 2016).

5.3.3. Modification or replacement of the target of the antibiotic

The modification of the target of the antibiotic is described for almost all antibiotics but more significantly in penicillins, glycopeptides, and MLS for Gram-positives and quinolones, whatever the type of Gram. When the antibiotic target is modified or replaced, the antibacterial agent loses its affinity for it and can no longer exert its activity at the level of the bacteria. The modification can occur by acquiring new genetic material coding for an enzyme altering the target or by a mutation within the nucleotide sequence of the target itself (Mangin, 2016).

5.3.4. Efflux pumps

Bacteria are equipped with systems that expel foreign metabolites or toxic compounds, such as antibiotics, into the external environment. This active efflux requires energy in the form of ATP (Adenosine Tri Phosphate) or a transmembrane electrochemical gradient, used by efflux pumps or active transporters, which reduce the antibiotic's intracellular concentration, limiting access to its target (Mangin, 2018). These efflux pumps often have a reasonably broad substrate specificity, and only some confer antibiotic resistance (Muylaert & Mainil, 2012). Resistance comes from the reduction in the antibiotic concentration in the cytoplasm of the bacteria, which prevents and limits the access of the antibiotic to its target. Some active transporters are particular, and we call them SDR (specific-drug-resistance) pumps, while others act on many molecules, and we call them MDR (multiple-drug-resistance) pumps.

5.3.5. Protection of the antibiotic target

Protection of the antibiotic target is a well-known mode of resistance for the tetracycline family and has more recently been described for quinolones and fluoroquinolones. It occurs thanks to steric hindrance of the ribosome by the production of Tet(M) and Tet(O) proteins, which dislodge tetracyclines from their target, or by synthesis of qnr (Quinolone Resistance) proteins which bind to topoisomerase, the target of fluoroquinolones, reducing their affinity for it (Demoré *et al.*, 2012).

5.3.6. Antibiotic trapping

When inactivation of the antibiotic or reduction of affinity for the target is impossible, the bacteria may be forced to sequester the antibacterial agent. Overproducing or synthesizing another target with an affinity for the antibiotic makes it possible to reduce its free concentration on the target (Demoré *et al.*, 2012). In other words, bacteria can trap an antibiotic by increasing the production of its target or by producing another molecule with an affinity for it. This reduces the antibiotic in the free state at the target level (Guardabassi & Courvalin, 2006). Figure 4 illustrates the synthesis of the different mechanisms of antibiotic resistance in a Gram-negative bacterium.

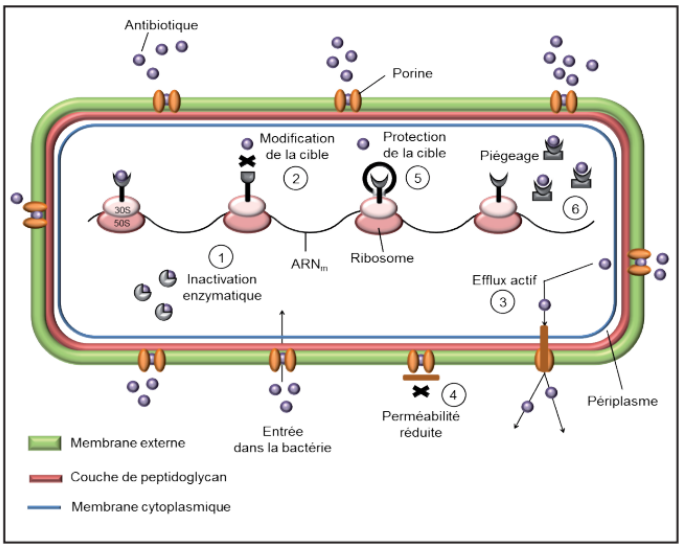

Figure 4: Summary of the different mechanisms of antibiotic resistance in a Gram-negative bacterium (Muylaert & Mainil, 2012).

5. CONCLUSION

In Benin, livestock production is the second most important activity after plant production and contributed to 13.44% of agricultural GDP in 2016. The increase in animal production in recent decades was possible thanks to the use of veterinary drugs. Modern breeding uses it for curative and preventive purposes or to promote growth and increase yield. The excessive use of antibiotics in veterinary and human medicine is the most critical factor for the appearance and spread of antibiotic-resistant microorganisms, constituting a double risk for livestock (therapeutic failure) and humans. (direct transmission) leading to a high rate of mortality and morbidity. The predominant resistant bacteria in the agropastoral sector are generally found in the intestines of animals but can be disseminated in the environment through fecal contamination of animals. This contamination of the environment can cause the appearance of infectious diseases in humans via the food chain. The transmission and spread of resistant germs on farms can be controlled by biosecurity measures, training of breeders, and promoting the use of plants with inhibitory properties on these predominant bacteria to avoid bacterial resistance. Finally, veterinarians must control antibiotic use to limit resistance, which constitutes a constraint hindering this sector.

6. REFERENCES

1. Acar J.F., Moulin G., Page S.W., and Pastoret P.P. Antimicrobial resistance in animal and public health: Introduction and classification of antimicrobial agents. *Revue Scientifique et Technique de l'Office International des Epizooties*. 2012; 31(1):15-21.
2. Adesiji Y.O., Alli O.T., Adekanle M.A., and Jolayemi J.B. Prevalence of *Arcobacter*, *Escherichia coli*, *Staphylococcus aureus*, and Salmonella species in Retail Raw Chicken, Pork, Beef, and Goat meat in Osogbo, Nigeria. *Sierra Leone Journal of Biomedical Research*. 2011; 3(1):8-12. ISSN 2076-6270.
3. Ahouandjinou H., Baba-Moussa F., Gbaguidi B., Sina H., Adeoti K., Mousse W., Pouadjeu-Wouansi D., Toukourou F., Soumanou M. and Baba-Moussa L. Antibiotic Resistance and Virulence Factors of Escherichia Coli Strains Isolated from Bovine Carcasses from Benin. *European Scientific Journal*. 2016; 12(33). ISSN: 1857 – 7881.
4. Akoachere J.F., Bughe R.N., Oben B.O., Ndip L.M., and Ndip R.N. Phenotypic characterization of human pathogenic bacteria in fish from the coastal waters of South West Cameroon: public health implications. *Reviews on Environmental Health*. 2009; 24 (2):147-156.
5. Alborn W.E., Allen N.E., and Preston D.A. Daptomycin disrupts membrane potential in growing *Staphylococcus aureus*: *Antimicrobial Agents and Chemotherapy*. 1991; 35(11): 2282-2287.
6. Allerberger F., Wagner M., Schweiger P., Rammer H-P., Resch A., Dietrich M.P., Friedrich A.W., and Karch H. *Escherichia coli* O157 infections and unpasteurized milk. *Euro Surveillance*. 2001; 6(10):379. https://doi.org/10.2807/esm.06.10.00379-en.
7. Ateba C.N., and Bezuidenhout C.C. Characterization of *Escherichia coli* O157 strains from humans, cattle, and pigs in the North-West Province, South Africa. *International Journal of Food Microbiology*. 2008; 128(2):181-188.
8. Avril J.L., Dabernat H., Denis F., and Monteil H. Clinical bacteriology, 2nd ed. Paris: 2000. P.602
9. Baranyi J., and Roberts T.A. A dynamic approach to predicting bacterial growth in food. *International Journal of Food Microbiology*. 1994; 23:277–294.
10. Baris P., Lagandre D., Gogan A.C., Gandonou M., and Afomasse M. Study of the soya and fish sectors in Benin. Final report, as part of the feasibility study of the Protein Sector Development Support Project. 2016; p.147.
11. Bäumler A., Tsolis R., Ficht T., and Adams L. Evolution of host adaptation in *Salmonella enterica*. *Infection and Immunity*. 1998; 66:4579-4587.
12. Berends B., Urlings H., Snijders J., and Van Knapen F. Identifying and quantifying risk factors in animal management and transport regarding *Salmonella spp*. In pigs. *International Journal of Food Microbiology*. 1996; 30:37-53.
13. Bergeron P., andTremblay P.E. Spectroscopic analysis of da white dwarfs: stark broadening of hydrogen lines including nonideal effects. The American Astronomical Society. *The Astrophysical Journal*. 2009; 696(2).
14. Beutin L., Bulte M., Weber A., Zimmermann S., and Gleier K. Investigation of human infections with verocytotoxin-producing strains of *Escherichia coli* (VTEC) belonging to serogroup O118 with evidence for zoonotic transmission. *Epidemiology and Infection*. 2000; 125:47-54.
15. Bourin M.C., and Lindahl U. Glycosaminoglycans and the regulation of blood coagulation. *Biochemical Journal*. 1993; 289(2):313–330. doi: 10.1042/bj2890313
16. Brandl M.T. Fitness of human enteric pathogens on plants and implications for food safety. *Annual Review of Phytopathology*. 2006; 44:367-392.
17. Brenner F.W., Villar R.G., Angulo F.J., Tauxe R., and Swaminathan B. *Salmonella* nomenclature. *Journal of Clinical Microbiology*. 2000; 38:2465-2467.
18. Butt A.A., Aldridge K.E., and Sanders C.V. Infections related to the ingestion of seafood Part I: Viral and bacterial infections. *Lancet Infectious Diseases*. 2004; 4:201-212.
19. Camart-Perie A., and Perie P. Urinary disorders in adult cattle. *Point Vétérinaire*. 2006; 38(278):31-37.

20. Cerniglia C.E., and Kotarski S. Approaches in the safety evaluations of veterinary antimicrobial agents in food to determine the effects on the human intestinal microflora. *J Vet Pharmacol Ther*. 2005; 28(1):3-20.
21. Chart H., Smith H.R., La Ragione R.M., and Woodward M.J. An investigation into the pathogenic properties of *Escherichia coli* strains BLR, BL21, DH5α, and EQ1. *J Appl Microbiol*. 2001; 89(6): 1048-58. Doi: 10.1046/j.1365-2672.2000.01211.x.
22. Chen C.R., Malik M., Snyder M., and Drlica K. DNA gyrase and topoisomerase IV on the bacterial chromosome: quinolone-induced DNA cleavage. *Journal of Molecular Biology*. 1996; 258:627-637.
23. Conrad C., Stanford K., McAllister T., Thomas J., and Reuter T. Shiga toxin-producing *Escherichia coli* and current trends in diagnostics. *Animal Frontiers*. 2016; 6(2):37–43. https://doi.org/10.2527/af.2016-0021.
24. Corpet D.E. Antibiotic residues and drug resistance in human intestinal flora. *Antimicrobial Agents and Chemotherapy*. 1987; 31:587-593.
25. Croxen M.A., and Finlay B.B. Molecular mechanisms of *Escherichia coli* pathogenicity. *Nature Reviews Microbiology*. 2010; 8:26-38.
26. DE (Direction de l'Élevage); Livestock department. Annual Report 2015. Republic of Benin. p106. 2016.
27. De Jong B., Andersson Y., and Ekdahl K. Effect of regulation and education on reptile-associated salmonellosis. *Emerging Infectious Diseases*. 2005; 11:398-403.
28. Demoré B., Grare M., and Duval R. Clinical and therapeutic pharmacy. 4th edition. Chapter 40: General information on systemic antibiotics and principles of use. Elsevier Masson. 2012.
29. Diogo V.C., Bébo D.B., and Elégbé H.A. Comparative study of the productivity of tilapia, Oreochromis niloticus (L.) in an integrated fish-chicken and non-integrated system in the commune of Aplahoué in southern Benin. *Afrique Science.* 2018; 14(2). 1 - 14 1 ISSN 1813-548X.
30. Direction de la Statistique Agricole (DSA), Department of Agricultural Statistics. (2021). National agricultural census. Main results of the core module, volume 2, 299p.
31. Dognon S.R., Salifou S.F.A., Dougnon J., Dahouda M., Scippo M-L., and Youssao A.K.I. Production, import, and quality of meat consumed in Benin. *Journal of Applied Biosciences*. 2018; 124:12476-12488. ISSN 1997-5902.
32. Ducluzeau R., and Raibaud P. Microbial ecology of the digestive system. Agressology: *International Journal of Physio-biology and Pharmacology Applied to the effects of Aggression*. 1985; 26:161- 163.
33. Eng S.K., Pusparajah P., Ab Mutalib N.S., Ser H.L., Chan K.G., and Lee L.H. *Salmonella*: A review on pathogenesis, epidemiology, and antibiotic resistance. *Frontiers in Life Science*. 2015; 8:284-293.
34. Epe B., and Woolley P. The binding of 6-demethylchlortetracycline to 70S, 50S, and 30S ribosomal particles: A quantitative study by fluorescence anisotropy. *EMBO Journal*. 1984; 3:121-126.
35. Falagas M.E., Rafailidis P.I., and Matthaiou D.K. Resistance to polymyxins: Mechanisms, frequency and treatment options. *Drug Resistance Updates*. 2010; 13:132-138.
36. FAO, CEDEAO. Review of the livestock/meat and milk Value Chains and Policies influencing them in West Africa. Rome/Italy: FAO/ECOWAS: 2017. p122. www.fao.org/3/a-i5275e.pdf
37. FAO. Poultry sector Benin. National Livestock Reviews of the FAO Animal Production and Health Division. No.10; 2014; p74 Rome.
38. Gale E., Cundliffe E., Reynolds P.E., Richmond M.H., and Waring M.J. The molecular basis of antibiotic action. New York, USA. 1981. p670.
39. Goldstein F.W., Emirian M.F., Coutrot A., Acar J.F. Bacteriostatic and bactericidal activity of azithromycin against Haemophilus influenzae. *Journal of Antimicrobial Chemotherapy*. 1990; 25:25-28.
40. Gordon D.M., and Cowling A. The distribution and genetic structure of *Escherichia coli* in Australian vertebrates: host and geographic effects. *Microbiology*. 2003; 149:3575-3586.
41. Grassl G.A., and Finlay B.B. 2008. Pathogenesis of enteric *Salmonella* infections. *Current Opinion in Gastroenterology*. 2008; 24:22–26.
42. Gray J.T., and Fedorka-Cray P.J. Survival and infectivity of *Salmonella choleraesuis* in swine feces. *Journal of Food Protection*. 2001; 64:945-949.
43. Guardabassi L., and Courvalin P. Modes of antimicrobial action and mechanisms of bacterial resistance. In Antimicrobial resistance in bacteria of animal origin. Aarestrup F.M. (ed.). Washington: ASM Press. 2006. p1-18.
44. Gyles C.L. Shiga toxin-producing *Escherichia coli*: an overview. *Journal of Animal Science*. 2007; 85:45-62.
45. Hanes D. Nontyphoid *Salmonella*. In International Handbook of Foodborne Pathogens. Miliotis N, Bier J. (Eds.). New York: Marcel Dekker. p137-149. 2003.
46. Hansen-Wester I., and Hesel M. *Salmonella* pathogenicity islands encoding type III secretion systems. *Microbes Infect.* 2001; 3:549–559.
47. Heyderman R.S., Soriani M., and Hirst T.R. Is immune cell activation the missing link in the pathogenesis of post-diarrhoeal HUS? *Trends in Microbiology.* 2001; 9:262- 266.
48. Hong W., Zeng J., and Xie J. Antibiotic drugs targeting bacterial RNAs. *Acta Pharmaceutica Sinica* B, 2014; 4 :258-265
49. Hu L., and Kopecko D.J. Typhoid *Salmonella*. International handbook of foodborne pathogens. Millotis M.D and Bier J.W, editor. New York: Marcel Dekker. 2003. p151-165.
50. Johnson J.R. Virulence factors in *Escherichia coli* urinary tract infection. *Clinical Microbiology Reviews*. 1991; 4:80-128.
51. Johnson T.J., Wannemuehler Y.M., Johnson S.J., Logue C.M., and White D.G. Plasmid replicon typing of commensal and pathogenic *Escherichia coli* isolates. *Applied and Environmental Microbiology*. 2007; 73:1976-1983.
52. Josephine H.R., Kumar I., and Pratt R.F. The Perfect Penicillin? Inhibition of a bacterial D-peptidase by peptidoglycan-mimetic beta-lactams. *Journal of the American Chemical Society*. 2004; 126:81222-81223
53. Kahne D., Leimkuhler C., Lu W., and Walsh C. Glycopeptide and lipoglycopeptide antibiotics. *Chemical Reviews*. 2005; 105:425-448.
54. Kaper J.B., Nataro J.P., and Mobley H.L.T. Pathogenic *Escherichia coli*. Nature *Reviews Microbiology Journal*. 2004; 2:123-140.
55. Kassa S.K., Ahounou G.S., Dayo G.K., Salifou C.F.A., Dotché O.I., Issifou T.M., Gandonou P., Kountinhouin G.B., Mensah G.A., Yapi-Gnaoré V., and Youssao A.K.I. Evaluation and modeling of milk production from Girolando, Borgou, Lagunaire, and Azawak × Lagunaire crossbred cows raised in the semi-improved system in Benin. *Journal of Applied Biosciences.* 2016; 103:9829 – 9840. ISSN 1997–5902.
56. Kassa S.K., Ahounou S., Dayo G., Salifou C., Issifou M.T., Dotché I., Gandonou P.S., Yapi-gnaoré V., Koutinhouin B., Mensah G.A., and Youssao A.K.I. Milk production performance of West African cattle breeds. *International Journal of Biological and Chemical Sciences*. 2016; 10(5):2316-2330.
57. Katz L., and Ashley G. W. Translation and protein synthesis: macrolides. *Chemical Reviews*. 2005; 105:499-528.
58. Kauffmann F. The serology of the *E. coli* group. *Journal of Immunology*. 1947; 57:71-100.
59. Kirk M.D., McKay I., Hall G.V., Dalton C.B., Stafford R., Unicomb L., and Gregory J. Food safety: Foodborne disease in Australia: The Oz Food Net experience. *Clinical Infectious Diseases*. 2008; 47:392-400.
60. Korsak N., Clinquart A., and Daube G. *Salmonella spp* in foodstuffs of animal origin: a real public health problem? *Annals of Veterinary Medicine.* 2004; 148:174- 193.
61. Kuhle V., Jäckel D., and Hensel M. Effector Proteins Encoded by *Salmonella* Pathogenicity Island 2 Interfere with the Microtubule Cytoskeleton after Translocation into Host Cells. *Traffic*. 2004; 5(5):356-370.
62. Labro M.T. Immunomodulatory effects of antimicrobial agents. Part I: Antibacterial and antiviral agents. *Expert Review of Anti-infective Therapy*. 2012; 10:319-340.
63. Lailler R., Moury F., Leclerc V., Bohnert M., Cadel-Six S., and Brisabois, A. Surveillance of Salmonella in the food chain to detect emergencies in France. *Epidemiological bulletin, animal health, and food*. 2015; 68: 11-16.
64. Le Minor C., and Richard C. Technical notes. Laboratory methods for the identification of enterobacteria. Ed. Pasteur Institute. Paris. 1993. 217p.
65. Le Minor L., and Veron M. Medical bacteriology. Edition 2, *Sciences Flammarion*. 1989; ISBN, 2257124189,9782257124180; p1107.
66. Lesse D.P.A.A. Management and modeling of the dynamics of transhumance routes in a context of climatic variability in the north-east of Benin. Thesis, The University of Abomey-Calavi. 2016.
67. Lesse P., Houinato M., Azihou F., Djenontin J., and Sinsin B. Typology, productivity, carrying capacity and pastoral value of pastures in transhumant rangelands in the North East of the Republic of Benin. *International Journal of Innovation and Applied Studies*. 2016; 14(1). ISSN 2028-9324.

68. Mangin L. Antibiotics and resistance: a survey of the knowledge and behaviour of the general public. *Pharmaceutical Sciences*. MSc. Thesis. University of Lorraine. France. 2016.
69. Manvila L., Manvila A., and Nicolin N. Antibiotic. In: Quevauvilles, Jacques, Pocket medical dictionary. Paris: Masson. 1995.
70. Martinez J.A., Soto S., Fabrega A., Almela M., Mensa J., Soriano A., Marco F., Jimenez de Anta M.T. & Vila J. Relationship of phylogenetic background, biofilm production, and time to detection of growth in blood culture vials with clinical variables and prognosis associated with *Escherichia col*i bacteria. *Journal of Clinical Microbiology*. 2006; 44:1468- 1474.
71. Maslow J.N., Mulligan M.E., Adams K.S., Justis J.C., and Arbeit R.D. Bacterial adhesins and host factors: role in the development and outcome of *Escherichia coli* bacteria. *Clinical Infectious Diseases*. 1993; 17:89-97.
72. Mattila A.L., Viinikka L., Saario I., and Perheentupa J. Human epidermal growth factor: renal production and absence from plasma. *Regulatory Peptides Journal*. 1988; 23(1):89-93. PMID: 3070644, DOI: 10.1016/0167-0115(88)90424-7.
73. McEwen S.A., Fedorka-Cray P. L. Antimicrobial use and resistance in animals. *Clinical Infectious Diseases.* 2002; 34:S93-S106.
74. Morisse J.-P., Cotte J.-P., and Huonnic D. 1994. Animal protection and intensive beef production. Animal Protection and Veterinary Services. *Scientific and technical review - Office International des epizooties (OIE)*. 1994; 13(1):79-88.
75. Mousse A. Toxinogenic profile and ESBL production of fluorescent strains of *Escherichia coli*, *Klebsiella pneumoniae*, and *Pseudomonas spp*., isolated from street foods and market garden products in southern Benin. Thesis in Biochemistry, Microbiology and Molecular Biology, University of Abomey-Calavi, Benin. 2016.
76. Movassagh M.H., Mohammad S., and Jalii Z. The Prevalence of *Salmonella spp*., in Bovine Carcass at Tabriz Slaughterhouse, Iran. *Global Veterinaria*. 2010; 5(2):146-149.
77. Muller A. Proper use of antibiotics: results of actions in different types of health establishments. University of Burgundy Franche-Comté. 2017. 2017UBFCE021. tel-01842246.
78. Murray C.J. Environmental aspects of *Salmonella*. *Salmonella* in Domestic Animals. Wray C., Wray A. (Eds.). Oxon: CABI Publishing. p265-283. 2000.
79. Musco N., Koura I.B., Tudisco R., Awadjihè G., Adjolohoun S., Cutrignelli M.I., Mollica M.P., Houinato M., Infascelli F., and Calabrò S. Nutritional characteristics of forage grown in the south of Benin. *Asian-Australasian journal of animal sciences*. 2016; 29(1):51-61].
80. Muylaert A., and Mainil J.G. Bacterial resistance to antibiotics: the mechanisms and their "contagiousness". *Annals of Veterinary Medicine.* 2012; 156:109-123.
81. Nataro J.P., and Kaper J.B. Diarrheagenic *Escherichia coli*. *Clinical Microbiology Reviews*. 1998; 11:142–201.
82. Ouedraogo A.S., Jean-Pierre H., Banuls Anne-Laure, Ouedraogo R., and Godreuil S. Emergence and spread of antibiotic resistance in West Africa: contributing factors and threat assessment. *Tropical Medicine and Health*. 2017; 27 (2):147-154. ISSN 2261-3684.
83. Patel U., Yan Y.P., Hobbs F.W.Jr., Kaczmarczyk J., Slee A.M., Pompliano D.L., Kurilla M.G., and Bobkova E.V. Oxazolidinones mechanism of action: Inhibition of the first peptide bond formation. *Journal of Biological Chemistry*. 2001; 276:37199-37205.
84. Paton J.C., and Paton A.W. Pathogenesis and diagnosis of Shigatoxin-producing *Escherichia coli* infections. *Clinical Microbiology Reviews.* 1998, 11:450–479.
85. Popoff M.Y., and Norel F. Molecular bases of *Salmonella* pathogenicity. *Medicine and infectious diseases.* 1992; 22:310-324.
86. Rahal J.J., and Simberkoff M.S. Bactericidal and bacteriostatic action of chloramphenicol against meningeal pathogens. *Antimicrobial Agents and Chemotherapy*. 1979; 16:13-18.
87. Reeves M.W., Evins G.M., Heiba, A.A., Plikaytis B.D., and Farmer J.J. The cloned nature of Salmonella Typhi and its genetic relatedness to other salmonellae are shown by multilocus enzyme electrophoresis and the proposal of the *Salmonella bongori* comb. November. *Journal of Clinical Microbiology*. 1989; 27:313-320.
88. Robineau B., and Moalic P-Y. A current disease in poultry production: colibacillosis. Bulletin of the French Veterinary Academy. Tome 163-3. 2010. p207-212.
89. Rudloff B., and Schmieg E. European disintegration: Too much to lose. No 21/2016, SWP Comments from Stiftung Wissenschaft und Politik (SWP), German Institute for International and Security Affairs. 2016.
90. Rurangwa E., Van Den Berg J., Layeye P.A., Duijn V., and Rothuis A. Fishing, fish farming, and aquaculture in Benin. A quick scan of the sector for intervention possibilities. IMARES report C072/14. p70. 2014.
91. Sabtu N., Enoch D.A., and Brown N.M. Antibiotic resistance: What, why, where, when, and how? *British Medical Bulletin.* 2015; 116:105-113.
92. Sahlström L., De Jong B., Aspan A. *Salmonella* isolated in sewage sludge traced back to human cases of salmonellosis. *Letters in Applied Microbiology*. 2006; 43(1):46-52.
93. Sánchez-Vargas F.M., Abu-El-Haija M.A., and Gómez-Duarte O.G. *Salmonella* infections: an update on epidemiology, management, and prevention. *Travel Medicine and Infectious Disease*. 2011; 9:263-277.
94. Sanders P., Perrin-Guyomard A., Moulin G. Evolution of the use of antibiotics in animal production. *Nutrition and Dietetics Notebooks*. 2017; 52:301-311.
95. Santos R.L., Reis B.P., Zhang S., Tsolis R.M., Bäumler A.J., and Adams L.G. The attenuated sop B mutant of *Salmonella enterica* serovar *Typhimurium* has the same tissue distribution and host chemokine response as the wild type in bovine Peyer's patches, *Veterinary Microbiology*. 2003; 97:269-277.
96. Scherer C.A., and Miller S.I. Molecular pathogenesis of *Salmonellae*. In Groisman. E. A. (Ed.). Principles of bacterial pathogenesis. United States of America: Academic Press. 2001. p265-316.
97. Schmidt H., Hensel M. Pathogenicity islands in bacterial pathogenesis. *Clinical Microbiology Reviews.* 2004; 17:14-56.
98. Schwarz S., Kehrenberg C., and Walsh T.R. Use of antimicrobial agents in veterinary medicine and food animal production. *International Journal* of *Antimicrobial Agents*. 2001; 17:431-437.
99. Scott S. The most probable number method and its use in Enumeration, Qualification, and validation. *Journal of Validation Technology*. 2010; 16:35-38.
100. Sheorey H., and Darby J. Searching for *Salmonella*. *Australian Family Physician*. 2008; 37:806- 810.
101. Smati M., Clermont O., Bleibtreu A., Fourreau F., David A., Daubie A.S., Hignard C., Loison O., Picard B., and Denamur E. Quantitative analysis of commensal Escherichia coli populations reveals host-specific enterotypes at the intra-species level. *Microbiology Open*. 2015; 4: 604-615.
102. Smati M., Magistro G., Adiba S., Wieser A., Picard B., Schubert S., and Denamur E. Strain-specific impact of the high-pathogenicity island on virulence in extra-intestinal pathogenic *Escherichia coli*. *International Journal of Medical Microbiology*. 2017; 307(1):44–56.
103. Sounon A.K.L.S., Aho E., Lesse P., Ickowicz A., Messad S., Lesnoff M., et Houinato M.R.B. Assessment of the direct economic value of cattle breeding in northern Benin. *International Journal Biological and Chemical Sciences*. 2019; 13(1):8-17. ISSN 1991-8631.
104. Sounon A.K.L.S., Lesse P., Ickowicz A., Messad S., Houinato M., and Mensah G.A. Developments in cattle breeding systems in north-west Benin. Bulletin of Agronomic Research of Benin. Interdisciplinary Special Issue. p22-29. 2019.
105. Swanson S.J., Snider C., Braden C.R., Boxrud D., Wünschmann A., Rudroff J.A., Lockett J., and Smith K.E. "Multidrug-resistant *Salmonella enterica* serotype *Typhimurium* associated with pet rodents. *New England Journal of Medicine.* 2007; 356(1):21-8.
106. Talaro K.P., Chess B. Foundations In microbiology. Microbiology Medical. New York, USA. 2012. p579.
107. Tenson T., Lovmar M., and Ehrenberg M. The mechanism of action of macrolides, lincosamides, and streptogramin B reveals the nascent peptide exit path in the ribosome. *Journal of Molecular Biology*. 2003; 330:1005- 1014.
108. Thorene G, Hurmonal immune responses to Shiga-like toxins and *Escherichia coli*; p43. 1994.
109. Tindall B. J., Grimont P. A., Garrity G. M. & Euzéby J. P. Nomenclature and taxonomy of the genus *Salmonella*. *International Journal of Systematic and Evolutionary Microbiology*. 2005; 55:521-524.
110. Tindall B.J., Grimont P.A., Garrity G.M., and Euzéby J.P. Nomenclature and taxonomy of the genus *Salmonella*. *International Journal of Systematic and Evolutionary Microbiology*. 2005; 55:521-524.
111. Todd E.C., Greig J.D., Bartleson C.A., and Michaels B.S. Outbreaks where food workers have been implicated in the spread of foodborne disease. Part5. Sources of contamination and pathogen excretion from infected persons. *Journal of food protection*. 2008; 71:2582-2595.
112. Vannuffel P., and Cocito C. Mechanism of action of streptogramins and macrolides. Drugs. 1996; 51:20-30.

113. Vernikos G.S., and Parkhill J. Interpolated variable order motifs to identify horizontally acquired DNA: Revisiting the *Salmonella* pathogenicity islands. *Bioinformatics*. 2006; 22:2196-2203.
114. Vimont A. Optimization of research into Shiga toxin-producing *Escherichia coli* (STEC). Life Sciences [q-bio]. Claude Bernard University - Lyon I. 2007. French. NNT: tel-00139179. Available: https://theses.hal.science/tel-00139179.
115. Vuillemin P. Antibiosis and symbiosis, French Association for the Advancement of Sciences, report of the 18th session, second part, Notes and memoirs. 1890; 11:525-543.
116. Wang Q., Ruan X., Wei D., Hu Z, Wu L., Yu T., Feng L., and Wang L. Developing a Serogroup-Specific Multiplex PCR Assay to Detect a Set of *Escherichia coli* Serogroups Based on Identifying Their O-antigen Gene Clusters. Molecular and Cellular Probes. 2010; 24:286-290.
117. Wattiau P., Boland C., and Bertrand S. Methodologies for *Salmonella enterica* subsp. *enterica* subtyping: gold standards and alternatives. *Applied and Environmental Microbiology*. 2011; 77:7877-7885.
118. Weill F-X. Non-typhoid *Salmonella* of animal origin and antibiotic resistance. *Bulletin of the French Veterinary Academy*. 2008; 1:221-234.
119. WHO: World Health Organization. Antimicrobial resistance: global report on surveillance. Geneva, Switzerland: World Health Organization. 2014. p232. ISBN 978 92 4 156474 8.
120. WHO: World Health Organization. *Escherichia coli* diarrhea. Bulletin of the World Health Organization. 1980; 58:23-36.
121. WHO: World Health Organization. Food safety. Annual Report. Join FAO/WHO expert meetings on microbiological risk assessment. 2020. Available: https://www.who.int/foodsafety/micro/jemra/zn/.
122. WHO: World Health Organization. Foodborne illnesses: Nearly a third of deaths occur in children under five years old. Geneva, Switzerland. 2015. URL: https://www.who.int/fr/news-room/detail/03-12-2015-who-s-first-ever-global-estimates-offoodborne-diseases-find-children-under-5-account-for-almost-one-third-of-deaths. Accessed September 30, 2016.
123. Yala. D, Merad A.S, Mohamedi. D, Ouar Korich M.N. Classification and mode of action of antibiotics, Médecine du Maghreb n°91. 2001. URL: http://www.sante.dz/aarn/classification.pdf.
124. Yao K.R., Coulibaly K.J., Cissé S., Tiécoura K.B., Goualié G.B., Gueu Kpon R., Yapi H.F., and Djaman A.J. Prevalence of *Salmonella* Strains in Cattle Breeding in the District of Abidjan (Côte d'Ivoire). *International Journal of Current Microbiology and Applied Sciences.* 2019; 6(3):1154-1162. ISSN: 2319-7706.
125. Youssao A.K.I. Dahouda M., Attakpa E .Y, Koutinhouin G.B., Ahounou G.S., Toleba S.S., and Balogoun B.S. Diversity of Borgou cattle breeding systems in the Sudanian zone of Benin. *International Journal of Biological and Chemical Sciences.* 2013; 7(1): 125-146. ISSN 1991-8631.
126. Youssao A.K.I. Programme National d'Amélioration Génétique. Projet d'Appui aux Filières Lait et Viande (PAFILAV) : National Genetic Improvement Program. Support Project for the Milk and Meat Sectors. Cotonou, Bénin. 2015. p344.

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