Review Paper

Carcass *Salmonella* and Its Drug Resistance

ABSTRACT

Salmonella are the major pathogenic bacteria in humans as well as in animals. *Salmonella* species are leading causes of acute gastroenteritis in several countries and salmonellosis remains an important public health problem worldwide, particularly in the developing countries. Isolation of *Salmonella* from a wide range of sources suggests that *Salmonella* is widespread in food animals and meat products and underlines the necessity for a joint and coordinated surveillance and monitoring programs for salmonellosis and other major food borne zoonotic diseases. Food animals harbor a wide range of *Salmonella* and so act as sources of contamination, which is of paramount epidemiological importance in non-typhoid human salmonellosis. Salmonellosis is more aggravated by the ever increasing rate of antimicrobial resistance strains in food animals. The high prevalence and dissemination of multidrug resistant (MDR) *Salmonella* have become a growing public health concern. Multidrug resistant (MDR) strains of *Salmonella* are now encountered frequently and the rates of multidrug resistance have increased considerably in recent years. Food animal consumption is a potential cause for antimicrobial resistant *Salmonella* illnesses besides, the common factors such as overcrowding, poverty, inadequate sanitary conditions, and poor personal hygiene. Practicing good sanitary measures, extensive education programs for proper hygiene and improvement of managements are solutions to eliminate the high bacteriological load as well as prevalence of *Salmonella* in cattle carcass. Furthermore, restricting the use of antimicrobial agents in food animals, designation of multidrug-resistant *Salmonella* as an adulterant in ground beef, improving the mechanisms for product trace-back investigations and wise and discriminate use of antimicrobials should be practiced to combat the ever increasing situation of antimicrobial resistance. So, this review used for updating information on their prevalence and resistance patterns is very important to suggest the acceptance of the carcass in relation to the standards and for proper selection and use of antimicrobial agents in a setting.

Key words; Salmonella, Drug resistance, Food animal, Prevalence, Multi-drug resistance

1. INTRODUCTION

There have been heightened concerns about the safety of food animals, not only amongst scientists with an interest in food toxicology or microbiology but also economists and other social scientists that focus on the wider socio-economic issues associated with the safety of a country's food animal supply [1]. *Salmonella* are the major pathogenic bacteria in humans as well as in animals. *Salmonella* species are leading causes of acute gastroenteritis in several countries and salmonellosis remains an important public health problem worldwide, particularly in the developing countries [2].It is also one of the most common food borne zoonotic diseases. The presence of *Salmonella i*n food animals at slaughter and the consequent cross-contamination of edible carcass tissues present a significant food safety hazard [3; 4]. Non-typhoidal Salmonella represents an important human and animal pathogen worldwide [5]. Infection in animals is of importance because of the direct economic effect and even greater importance is that animals constitute a vast reservoir of these organisms for human infection [6].

Isolation of *Salmonella* from a wide range of sources suggests that *Salmonella* is widespread in food animals and meat products and underlines the necessity for a joint and coordinated surveillance and monitoring programs for salmonellosis and other major food borne zoonotic diseases. A periodic surveillance of the sources, distribution and prevalent *Salmonella* serotypes in slaughtered food animals, retail meat products and environment is necessary to control the spread of the pathogen and infection of man through contaminated animal products [7]. Often, infected animals shed *Salmonella* in feces without showing clinical signs. Various stress factors such as those associated with transport of animals from farm to slaughterhouse augments shedding of *Salmonella* from carrier animals. Food animals such as cattle

may carry *Salmonella* at slaughter and can serve as sources of contamination and provides an opportunity for entry of the pathogen into the food products [8; 9]. This implies that the presence of *Salmonella* in slaughter cattle and slaughterhouse environment and the potential cross-contamination of carcasses and edible organs can pose food safety hazards [9].

Food animals harbor a wide range of *Salmonella* and act as sources of contamination, which is of paramount epidemiological importance in non-typhoid human salmonellosis [10; 11]. More than 2,500 *Salmonella* serotypes have been identified, and the 20 most well-known human serotypes represented 78% of all human *Salmonella* isolates [114; 115].The process of removing the gastrointestinal tract during slaughtering of food animals is regarded as one of the most important sources of carcass and organ contamination with *Salmonella* at slaughterhouse. Moreover, contamination of meat by *Salmonella* may occur at slaughterhouse from the excretion of symptomless animals, contaminated slaughterhouse equipment, floors and personnel and the pathogen can gain access to meat at any stage during butchering. Cross contamination of carcasses and meat products could continue during subsequent handling, processing, preparation and distribution [10; 11].

Salmonellosis is more aggravated by the ever increasing rate of antimicrobial resistance strains in food animals [2]. The high prevalence and dissemination of multidrug resistant (MDR) *Salmonella* have become a growing public health concern. Of particular significance is the increasing number of *Salmonella* isolates that are resistant to clinically important antimicrobial agents such as fluoroquinolones and third-generation cephalosporins, which are used for the treatment of life threatening disease conditions in humans [12; 9]. Antimicrobial resistant *Salmonella* are increasing due to the use of antimicrobial agents in food animals at sub-therapeutic level or prophylactic doses which may promote on farm selection of antimicrobial resistant strains and markedly increase the human health risks associated with consumption of contaminated meat products [7; 13; 2]. Antimicrobial resistant *Salmonella* and other zoonotic bacterial pathogens can be transferred from animals to humans through consumption of contaminated food and food products and thus present a public health risk. The increase in *Salmonella* resistance to the commonly used antimicrobials both in the public health and veterinary sectors is one of the major threats of health care worldwide [7]. Cattle have been implicated as a source of human infection with antimicrobial resistant *Salmonella* through direct contact with livestock and through the isolation of antimicrobial resistant *Salmonella*from raw milk, cheddar cheese, and hamburger meat [2].

The emergence and spread of antimicrobial resistant *Salmonella* strains in food animals and humans may be associated with the use of medicated feeds in intensive animal husbandry systems, sub therapeutic doses and indiscriminate uses of antimicrobials both in animal and human treatments. Various antimicrobials in intensively managed food animals including chicken are often administered through the feed or drinking water either for therapy, prophylaxis or growth promotion. This enhances the risk of proliferation of resistant strains, which can have severe consequences on human health [7].

Multidrug resistant (MDR) strains of *Salmonella* are now encountered frequently and the rates of multidrug resistance have increased considerably in recent years. Even worse, some variants of *Salmonella* have developed multidrug resistance as an integral part of the genetic material of the organism, and are therefore likely to retain their drug resistant genes even when antimicrobial drugs are no longer used (14). Transfer of resistance genes can occur between *Salmonella* strains or from other bacterial species to *Salmonella.* These other species can be the basis of antibiotic resistance genes that might not be found in the *Salmonella* genetic pool at a given time. In *Salmonella,* plasmids and class I integrons are mainly responsible for such transfers. Genes conferring resistance to aminoglycosides [124, 115], beta-lactams [118, 120, 115], chloramphenicols [119, 120], tetracyclines [123], sulfonamides [117, 121], and trimethoprim [116] all have been found on numerous different plasmid types. Many of these plasmids carry multiple antibiotic resistance genes that are transferable to other *Salmonella* strains and other bacterial species [120, 115].

Most of the strains of *Salmonella* Typhimurium isolated in a study in western part on Nigeria were resistance to drugs like streptomycin, amoxicillin, tetracycline, ampicillin, kanamycin and chloramphenicol. This data is alarming since the isolates were already showing high resistance to drugs that are meant as alternate therapy to salmonellosis treatment; especially isolates from blood were resistance to the commonly used antibiotics [15]. Drug resistant *Salmonella* emerged in response to antimicrobial usage in food animals, which has also contributed or resulted in major outbreaks of salmonellosis. Selective pressure from the use of antimicrobials is a major driving force behind the emergence of resistance, but other factors also need to be taken into consideration [15].

Food animal consumption is a potential cause for antimicrobial resistant *Salmonella* illnesses besides, the common factors such as overcrowding, poverty, inadequate sanitary conditions, and poor personal hygiene [16]. *Salmonella* contamination was high in food items such as minced beef, mutton and pork samples obtained from retail supermarkets and slaughterhouses, that means *Salmonella* contamination is especially high in meat samples as compared to others food items. Supermarkets and slaughterhouses personnel are also a victim of *Salmonella* contamination and the magnitude of the problem represents a real public health hazard [17; 18].

Problems have their origin in the methods of farming of animal foods. Many farmers are illiterate and follow methods of production that are centuries old. And also raw meat coming from slaughterhouse is available in open air local retail shops without appropriate temperature control and this is purchased by households and also partially cooked minced meat (Kitfo) is served in restaurants. Meat processing at retail level is likely to contribute to the higher levels of contamination in minced carcasses [125]. However, considerable proportion of patients may not visit health centers unless symptoms are serious due to shortage of resources and lack of awareness. So, this review used for updating information on their

prevalence and resistance patterns which is very important to suggest the acceptance of the carcass in relation to the standards and for proper selection and use of antimicrobial agents.

2. SALMONELLOSIS AND ITS SOURCES

Food borne sources of *Salmonella* include a wide range of domestic and wild animals and a variety of food stuffs including food of both animal and plant origin [19]. *Salmonella* serotypes have a broad host range [20], prevalent in the warm blooded animal population [21], including rodents [22], snakes [23], and free living terrestrial and aquatic turtles [24]. *Salmonella* Typhimuriumand *Salmonella* Dublinappear to be the commonest serovars isolated from animal especially cattle, although the distribution of these two serovars may differ between countries, and *S.* Dublinis thought not to be present in some countries [25]. Infection by this *Salmonella* serovars occurs when susceptible animal ingest feed or water that has been contaminated with feces from animals shedding the organism. Some adult animal which recover from *Salmonella* infection, especially in the case of *S.* Dublin, may become active carriers and excrete the organism continuously or intermittently in their feces for years. Salmonellosis has a wide spectrum of manifestations in animals. Asymptomatic, mild clinical or fulminant bacteremia/septicemia and endotoxemic infections can occur. The number of *Salmonella* required to produce clinical disease is dependent on the virulence of the serotype, infectious dose and immunity of the host. Infection with a host adapted *Salmonella* strain (S. dublin in cattle) can result in a cyclic, endemic disease that is maintained on a farm by carrier animals shedding in the feces. The carriers can shed constantly or intermittently [26].

2.1. *Salmonella* in cattle

Different parts of animals such as cattle are one of the main source of cattle carcass. Hide has been familiar as the main source of foodborne pathogens, including *Salmonella*. *Salmonella* also has been found in cattle lymph nodes and immediately contaminate the carcass during slaughtering. Most of the lymph nodes located in fat tissues of beef carcasses are not removed during slaughtering. These lymph nodes are ground with lean and fat trimmings to produce ground beef, making lymph nodes a promising source of *Salmonella* in ground beef carcass. None of the published studies have included the simultaneous determination of *Salmonella* on the hides and in lymph nodes or sampling and tracking of all potential sources of *Salmonella* in ground beef [135].

Infection in cattle may also occur via other routes, including the respiratory tract, by inhalation of aerosol [34]. The genus *Salmonella* pose a serious threat to the domestic food animal including cattle. All animals including cattle are at increased risk of developing disease if their normal flora is disrupted (stress, antibiotics). These circumstances render cattle susceptible to exogenous exposure or activation of silent infections. Poor sanitation, overcrowding, unfavorable weather, stress and surgery, parturition, parasitism, transportation, and concurrent viral infections are all factors which predispose cattle to clinical salmonellosis. In the subclinical form, cattle and other animal may have a latent infection and harbor the pathogen in its lymph nodes, or it may be a carrier and eliminate the agent in its fecal material briefly, intermittently, or persistently [27]. These organisms are responsible for significant morbidity and mortality in their respective hosts, as well as causing substantial disease to humans consuming processed meats derived from the infected cattle. What may be considered now is the emergence of different food vehicles, such as meat, as the source of these infections, forcing industry to examine its management practices and incorporate new procedures to reduce the incidence and severity of the problem [17]. The infectious dose for healthy adult cattle by *Salmonella* high in number [5]. In adult cattle salmonellosis commonly occurs close to parturition and may be associated with inter-current disease. The growth of *Salmonella* in the rumen following ingestion is influenced by dietary intake before and after the *Salmonella* is ingested. *Salmonella* disappear rapidly from the rumen of regularly fed cattle, but maintain or increase their numbers when feed intake is decreased or interrupted for one or more days. Feeding after a period of starvation is associated with multiplication of *Salmonella*. Disruption of normal fermentation with production of lactate favors the less fastidious *Salmonella*, which multiplies rapidly using the available substrate. Qualitative dietary stress and dietary changes have been implicated as a predisposing risk factor in *Salmonella* outbreaks in dairy cattle. Reduction in the prevalence of *Salmonella* may be observed following manipulation of the ration formulation and adjustment of feeding practices [28].

Salmonella within and between herd prevalence estimates vary considerably, with between herd point prevalence estimates for cattle operations ranging from 2-42% and within herd estimates for these operations ranging from 0-37% [29; 30]. In addition, herds with clinically sick animals are generally characterized by higher within herd prevalence than herds where clinical salmonellosis is absent, and *Salmonella* distribution may differ between herds with and without clinical cases. Large herd size represents an important risk factor for salmonellosis, and the risk of *Salmonella* shedding seems to vary by production system, housing type, general hygiene level, management type and animal age, although the results reported in the literature have been somewhat contradictory [31]. Calves, heifers, and parturient cows generally appear to be at a particular risk of infection, and one study found heifers and parturient cows to be the most likely cattle to become asymptomatic carriers. The distribution of *Salmonella* among cattle varies greatly over time, and differs among geographic regions, age groups, clinical manifestation, and production systems (32; 30]. Illness from salmonellosis in the cattle is seen predominantly in young calves, although occasionally it is seen in adult cattle as well. *Salmonella* have been isolated from the feces of healthy cattle, where the pathogen may exist as a normal member of the gastrointestinal population or as a transient member of the gastrointestinal microbial population. Researchers have shown that as herd size increased, fecal shedding of *Salmonella* increased. However, other studies have found that herd size did not play a

role in *Salmonella* shedding [33]. Genetics plays a very important role in the relationship between *Salmonella* and its potential host animal including cattle. Some *Salmonella* isolates display a very narrow host specificity, while many of the remaining members of this genus express a wider ranging host infectivity. Furthermore, members of a particular *Salmonella* isolate express differential capabilities for infecting a particular host. Conversely, genetics plays an important role in enabling the host to resist infection by a *Salmonella* pathogen [25].

2.2. *Salmonella* in other animals

As many as 90% of reptiles may be *Salmonella* carriers. Between 3% and 5% of all cases of salmonellosis in humans have been associated with exposure to exotic pets, especially reptiles (including pet turtles, iguanas, lizards, and snakes) [39]. The United States banned the sale of small turtles (carapace < 4 in. [10.2 cm]), and reissued a warning because of a resurgence of turtle sales and subsequent outbreaks [40]. Pet rodents probably represent an under-recognized source of *Salmonella* infection. In 2007, these animals were responsible for an outbreak of multidrug-resistant *Salmonella* in several states. Of 22 patients interviewed, 13 (59%) in 10 states reported exposure to pet hamsters, rats, or mice, and 2 (9%) had secondary infections [41].Animals in petting zoos may also serve as sources of infection, as also certain other animals, such as baby poultry and livestock [37]. After that, *Salmonella* is transmitted to vectors such as rats, flies and birds where *Salmonella* can shed in their feces for weeks and even months. Following the direct transmission, moving animals such as swine, cattle and chickens act as the important risk factor for infection [37]. The main reservoirs for non-typhoidal *Salmonella* are animals including; poultry, livestock and pets [36]. Historically, the major mode of transmission for non-typhoidal *Salmonella* was consumption of inadequately cooked or pasteurized foods of animal origin, such as poultry, beef (including ground beef), fish, eggs, and dairy products (including ice cream) [37]. Once carried by vectors or transferred to food, consumption by human can result in the risk of salmonellosis [34].

Pork has been identified as a repeated source for salmonellosis in various studies [138]. About 15 to 20% of all animal and human salmonellosis cases in Denmark, Netherlands, and Germany were associated with the eating of pork [137]. However, not only pork but also poultry have been associated with the transmission of *Salmonella* [136]. *Salmonella* is known to inhabit the gastrointestinal tract of animals without producing any clinical or pathologic-anatomic signs [137]. Therefore, carcasses can become contaminated with *Salmonella* at the time of slaughter. Contaminated raw or undercooked red meats are the main routes of transmission for this foodborne pathogen.

2.3. *Salmonella* in fomites

Animal reservoirs are infected orally because *Salmonella* normally originates from the contaminated environment and also contaminated feed. Other modes of transmission include ingestion of contaminated water and contact with contaminated dyes and medical instruments [37].The environment contaminated with *Salmonella* serves as the infection source for cattle carcass because *Salmonella* can survive in the environment for a long time. So, transmission of *Salmonella* from the food processing plants and equipment during food preparation are also of great importance to infect carcass. The *Salmonella* cells can attach to food contact surfaces such as plastic cutting board which may develop into biofilm once attached and hence cause cross-contamination. Consequently, *Salmonella* can enter the food chain at any point from livestock feed, through food manufacturing, processing and retailing as well as catering and food preparation in the home [34].

2.4. *Salmonella* in food handlers

Infected food handlers have been shown to transmit *Salmonella* and have been responsible for outbreaks. Workers who have been ill can shed *Salmonella* for a median of 30 days (range, 2 days to 280 days). Therefore, assessment of food-worker infection is essential for controlling outbreaks traced to restaurants [38]. For example, *Salmonella* Typhi and *Salmonella* Paratyphi A do not have animal reservoir, therefore infection can occur by eating the improperly handled and under cooked food by infected individuals [35; 24].

3. CONTAMINATION AND PREDISPOSING RISK FACTORS

3.1. Contamination and microbial load of carcass

The hygiene conditions at the production line for slaughter animals are one of the critical factors influencing both, the level of carcass microbial contamination and the type of determined microorganisms. Experimental studies have shown that total aerobic bacterial contamination depends on a slaughter site and may range in bovine carcasses from $10^2/cm^2$ up to $10^6/cm^2$ [42]. Effective intervention to reduce contamination of beef carcasses begins with determining potential sources of contamination. Tissues under the hide of healthy cattle are usually sterile [43]. Consequently, tissues become contaminated during the slaughtering process. Sources of meat contamination during slaughter may be classified as handling practices of slaughter man and cross-contamination. The extent to which potential contamination sources become hazardous to public health depends on management and unpredictable events or factors. Even in the best managed slaughter facilities, contamination may still occur. Fortunately, most bacterial colonies which have been isolated from beef carcasses have been non-pathogenic, although human pathogens such as *Salmonella* have been isolated also [44]. Surface contamination of carcasses during slaughter and processing can be reduced by ensuring good

manufacturing practices such as hygiene and sanitation of the floor, equipment, and carcasses, with suitable disinfectants and sanitizers [45]. Meat has a microbial flora from different sources. Also, several methods have been proposed for decreasing the microbial flora to a standard allowance for increasing the shelf-life and decontamination of microbial pathogens including cooking, freezing, fermenting, salting, smoking, drying, and pickling [46].

A cross-sectional study was conducted in cattle and pig carcasses showed that mean total viable counts ranged from 2.4 to 4.2 $\log_{10}$cfu/cm^2 on pig carcasses and from 2.7 to 3.8 $\log_{10}$cfu/cm^2 on cattle carcasses. Amongst sites, the back (pigs) and neck (cattle) tended to yield higher total viable counts [47]. Similarly in calf carcasses results have shown that the total aerobic bacteria count in each slaughter stage ranged from 3.5 x 10^3cfu/cm^2 up to 7.0 x 10^3cfu/cm^2. In most cases, no significant differences of total bacterial contamination of carcasses in each slaughter stage were obtained. At stage II, a significantly higher total aerobic bacteria count (10^4cfu/cm^2) was observed, when compared to stage I where 2.3 x 10^3cfu/cm^2 was reached [42]. In Khartoum State the study was conducted to evaluate the bacteriological contamination in indigenous cattle carcasses in slaughterhouse, during April 2008- June 2008. The mean total viable count of bacteria after skinning, evisceration and washing operations at shoulder site were, 3.03 ± 0.15, 2.73 ± 0.02 and 2.79 ± 0.10 $\log_{10}$cfu/cm^2, in the neck site were 3.65 ± 0.02, 3.42 ± 0.02 and 3.72 ± 0.02 $\log_{10}$cfu/cm^2 and in brisket site were 3.1 ± 0.14, 3.71 ± 0.04 and 3.65 ± 0.02, respectively. In addition, in the rump site, the total viable counts in these operations were 3.24 ± 0.02, 2.88 ± 0.02, and 3.18 ± 0.03 $\log_{10}$cfu/cm^2 in three points of operation [48]. Another study in China showed that beef samples from Sakasaka had the highest mean total bacterial count of 1.67×10^6 cfu/cm^2, followed by Aboabo (5.75×10^5cfu/cm^2), Central Market (internal) (4.325×10^5cfu/cm^2), Nyohini (3.875×10^5cfu/cm^2) and Central Market (external) (4.325×10^5cfu/cm^2). While their mean log counts were 6.22, 5.76, 5.64, 5.59 and 5.57 for Sakasaka,Aboabo, Central Market (internal), Nyohini and Central Market (external), respectively [49]. In Mumbai, a total of 54 swab samples were from the abattoir, while 81 swab samples were from three meat shops reported that the average total viable count (TVC) for all environmental contamination points in the abattoir was 5.80 ± 0.17, where as in the shops it was 6.05 ± 0.25 $\log_{10}$cfu/cm^2 indicating higher microbial load in traditional meat shops [50].

3.2. Predisposing risk factors for the prevalence of *Salmonella* species

There is a lack of studies of risk factors for *Salmonella* in cattle carcass [51]. The risk factor was divided into three sections: (i) slaughterhouse practices (cleaning and disinfection of pens, truck washing, frequency of knife disinfection, water treatment, etc.); (ii) information on the animal lots (time from farm to slaughter, cleanliness of the animals, tattoo number, and producer number); and (iii) any event during the slaughtering that may have affected the contamination of carcasses (mechanical problems, slaughter rate, stops, condemnation rate, contamination rate, gut ruptures, percentage of filled stomachs, and employee training) [52].

The probability that a live animal is contaminated (both internally and externally) and the extent of contamination (pathogen load) depend on factors which can be affected by management before transport to the abattoir (on-farm and market factors), during transport, and while the animals are being held at the abattoir before slaughter [44]. However, suggestions of factors of importance for *Salmonella* occurrence in cattle generally include hygienic factors in the herds e.g. flies in pens [51] contact with poultry manure or wild bird manure, outdoor calving, herd size and herd expansions [53]. Hygiene and contacts at markets and in vehicles are also likely to be important risk factors before slaughter. In Danish dairy herds, risk factors for becoming infected in 2003 included herd size, number of purchased cattle from test-positive herds and number of test-positive neighbor herds. Organic herds were less likely to recover than conventional herds indicating that different types of management can influence the occurrence of *Salmonella* in cattle herds [54].

Salmonella contaminated carcass could be from the actual infection of food animals at the farm. Off-farm rearing of heifers has been acknowledged as an important risk of infection with multi-drug-resistant *Salmonella* in US dairy herds [49]. One study also reported that for heifers and cows, recent antimicrobial treatment increased the probability of isolating *Salmonella* from fecal samples. Also, *Salmonella* has been associated with high calf mortality in dairy herds. This may be due to both direct effects of the infection and underlying management factors [32].

Transport factors such as the type and cleanliness of transport conveyance, distance travelled and duration of journey, harshness of ride, density of animals in the conveyance and frequency of stops, may affect the pathogen load including *Salmonella*. Interruption of feeding just before transport, during transport, and while being held at auction barns and abattoirs affect the growth of potential pathogens in the rumen and fecal shedding of bacteria. The number of calves which shed *Salmonella* has been found to increase after transportation [55]. The length of time animals are held at the abattoir before slaughter can affect the pathogen load by increasing the probability of exposure and infection. Sanitation of walkways, pen floors, railings, feed and water affect the pathogen load. Steep walkways with sharp turns increase the likelihood that animals will fall and become contaminated or injured. Excessive prodding of animals to move them bruises tissue [44].

Reptiles also the risk factors for cattle contamination by *Salmonella*. *S. enterica* subspecies *arizonae*is widely distributed in reptilian species. Reptiles, particularly snakes, are the natural reservoirs of *S. enterica* subspecies *arizonae*. This organism has also been responsible for severe outbreaks in turkeys, chickens and sheep and cattle [56]. Though the organism is rare, several studies suggest that snakes and reptiles harbor it and transmit it to humans and other mammals, resulting in gastroenteritis and systemic infections [57]. In particular, rattlesnake meat, capsules and powders have been

linked to infection with *S. arizonae,* although other animals or animal products have been implicated, including reptiles, poultry, sheep, rats, dogs, and cats [58].
With regard to risk factors at the slaughterhouse associated with the presence of *Salmonella* in the final product, study in Canada demonstrated the importance of the pre-slaughter and pre-evisceration environment on the final status of carcasses. Namely, the cleanliness of the hogs and the status of the scald water proved to be significant factors associated with the final bacteriological status of the carcasses. Results obtained by genetic characterization and serology indicated that particular attention should be paid to the herd contamination levels of incoming animals and the pre-evisceration environment to better control *Salmonella* at slaughter [52]. The feces and fecal contaminated products of animals can contain many enteric organisms including *Salmonella*. When the carcass is opened and the viscera removed, spillage of rumen and intestinal fluids may contaminate the carcass, workers, processing utensils and viscera tables or trucks [44]. People working in meat processing plants also can act as vector of many food borne pathogenic bacteria including *Salmonella* [59]. Microbial contamination of slaughtered cattle carcass results from starts during slaughter, processing and when the carcass becomes contaminated with microorganisms residing on external surfaces of the animal itself, the gastrointestinal tract, lymph nodes of the animal and in the plant environment [60; 61].
Furthermore, certain processing steps increase contamination by spreading the existing contaminants attached to the fresh meat surface to its entire mass or by introducing additional contaminants. For example, meat chopping or grinding results in greater microbial loads because of larger areas of exposed surface, more readily available water and nutrients, additional processing time, and contact with more sources of contamination such as equipment [62]. *Salmonella* contaminated carcass could be from cross-contamination during slaughtering, distribution and subsequent handling and processing. Cross-contamination may arise from knives and hand of the slaughter man. The other probable source of cross-contamination could be from *Salmonella* carrier slaughter house personnel [63]. The study conducted in Ethiopia also documented that level of carcass contamination was considered as an outcome variable taking skin swab, mesenteric lymph node, cecal content, evisceration's hand swab, eviscerating knife swab and water samples *Salmonella* status and total slaughter volume as risk factors for carcass contamination [11].
The study conducted in Canada showed that independent variables, when tested individually, indicated that *Salmonella* contamination of scalding tanks, knives, and boots, cleanliness of hogs, and the number of chain stops was associated with the prevalence of *Salmonella* in the lots. No difference was found between clean lots and relatively clean ones. There was a positive, but not significant, correlation between the prevalence of *Salmonella* on carcasses and chain speed and the frequency of knife washing. There was no correlation between prevalence of *Salmonella* on carcasses and cleaning product concentration used: chlorine or quaternary ammonium. *Salmonella* prevalence was similar for the two types of cleaning products and for the two types of rinsing [52].

4. PREVALENCE OF *SALMONELLA* SPECIES

Salmonella is a gram-negative bacteria that causes infections in a huge number of avian, mammalian, and reptilian species. The genus *Salmonella* includes the species *Salmonella bongori* and *Salmonella enterica,* which is divided into six subspecies: I, II, IIIa, IIIb, IV, and VI. Subspecies I signifies roughly 99% of all reported human isolates in the United States. *Salmonella* isolates are conventionally classified by serotype, which is based on the O and H antigen immune-reactivity of an isolate. More than 2,500 *Salmonella* serotypes have been identified, and the 20 most common human serotypes represented 78% of all human *Salmonella* isolates reported in the United States in 2003 [114,15].
Salmonella infections are typically contracted through the ingesting of contaminated food, water, or through contact with an infected host. *Salmonella* is one of the foremost causes of foodborne illness in the United States and the European Union (EU) [127,128], with estimated incidences of 15.1 cases [126] and 42.2 cases [127] respectively. Most *Salmonella* infections do not needed treatment and result in temporary gastroenteritis [129]. In invasive life-threatening infections, the use of antimicrobial drugs is needed [124], but the efficacy of many of these drugs is declining as antimicrobial-resistant *Salmonella* subtypes arise [130,115].

4.1. Global overview

In many countries incidence of human *Salmonella* infection has increased drastically over the years. The two most commonly isolated serotypes of concern and mostly implicated in disease outbreaks are *Salmonella enterica* serotype Typhimurium and *Salmonella enterica* serotype Enteritidis [64; 65; 66]. Besides the importance of this microorganism for public health, another aspect is the cost generated by human salmonellosis. During 1999, the cost linked to food borne salmonellosis ranged between 560 million and 2.8 billion € in Europe, where *Salmonella* was estimated to be responsible for nearly 166 000 cases [67].
It is reported that the rate of salmonellosis in the United States is between 15 to 20 cases per 100, 000 people [68]. The *Salmonella* species is one of the eight microorganisms in the European Union Zoonoses Monitoring Directive (EUZMD), which shows it is a disease considered to have a high impact on human and animal health in the Union [69]. The Enter-net surveillance program reported *Salmonella enterica* serotypes Enteritidis and Typhimurium, the most predominant organisms identified by the participating countries making up over 80% of all isolates during the period of 1998-2003. It also reported that for all *Salmonella* the general trend is declining with a reduction of 35.3% in 2003 over 1998 [70].

The burden of salmonellosis in Peshawar, Pakistan was estimated from published studies that Prevalence of *Salmonella* on cattle body coat, carcasses, slaughtering floor and tools of the butchers were investigated. The animals were divided into two groups i.e. washed and unwashed animals. *Salmonella* was found in 100% samples. Carcasses samples from unwashed animals had significantly higher log total viable count of *Salmonella* (24.45 ± 0.06) as compared with washed animals (21.77± 0.05) [71]. Studies in Spain showed that *Salmonella* was detected in 9 (17.3%) of the cattlesamples. All of the isolates werecharacterized as *Salmonella enterica* serotype Frintrop [72]. In the United Arab Emirates however, Wernery [73] found the prevalence of *Salmonella* in camels to be less than five percent. Another study in United Arab Emirates on fresh chicken meat samples 46.67% was positive for *Salmonella* of the total samples. Samples obtained from the supermarkets tested negative for *Salmonella* while chicken samples obtained from the butcheries tested positive [74]. In Australia, *Salmonella* was detected in 21 (6.8%) of the 310 cattle tested, 14 (9%) from lot-fed cattle feces and seven (4.5%) from grass-fed cattle feces. There was no significant difference in the prevalence of *Salmonella* between grass fed and lot-fed cattle [75]. In Lao People's Democratic Republic the reported organism were isolated from cecum samples of buffaloes and pigs. The organisms were a prevalence of 8% (4/50) from buffaloes and 76% (37/49) from pigs. In buffaloes, 1 animal harbored both *S.* Derby and *S.* Javiana. In pigs, the most predominant serotypes were *S.* Derby (51%) followed by *S.* Anatum (45%), *S.* Weltevreden (15%) and *S.* Stanley (5%) [76].
In Bhutan, the prevalence of *Salmonella* was 13% with *Salmonella* Enteritidis as the most frequently isolated serotype (84.62%), followed by *Salmonella* Typhimurium (15.38%). The isolation of *Salmonella* during winter and late spring was significantly different. Broiler carcasses were 10.62 times more likely to yield *Salmonella* in the hot season as compared to the winter season [3]. The reported prevalence of *Salmonella* in South Asian countries varies from country to country. Studies in northern Thailand revealed 57% prevalence during 2002-2003 [77], 14.5% prevalence in Kathmandu, Nepal [78], and 42.63% prevalence in Vietnam [79]. Sero-prevalence of *Salmonella* in Bangladesh has been reported to be 23.46% [80]. Not much literature has been available on the prevalence of *Salmonella* in carcasses, few researches reports negligible [81] to as low as 5% [82], to a prevalence of 69% [83]. The overall annual incidence of food borne salmonellosis in India is nearly 6 per 1000 inhabitants [1]. In Korea a total of 5.28% *Salmonella* species was isolated from fecal materials and organ samples. The predominant *Salmonella* is *Salmonella enterica* serotype and serovar was group B (69.8%) and *Salmonella* Typhimurium (47.6%), *S*. Derby (20.6%) and *S.* Heidelberg (1.6%) [84].

4.2 Status in African countries

The Food and Agriculture Organization of the United Nations and the World Health Organization jointly state that "illness due to contaminated food was perhaps the most widespread health problem in the contemporary world," and "an important cause of reduced economic productivity" [85]. With the increasing population in the developing world, there is an increasing demand for meat and meat products which will force the present resource driven system of livestock production to a demand driven system [86] which will increase the disease transmission risks. There is a multi-factorial risk of food borne hazards in the developing countries due to poor sanitation and inadequate access to potable water [1].
Studies conducted in different regions of Africa including Namibia, Kenya and Nigeria have always topped the incidence of salmonellosis [87; 4; 88] and is the most seriously perceived food risks, even in the developed countries [89]. In Kenya sixteen (13.8%) of 116 samples were positive for *Salmonella*. Three *Salmonella enterica* subspeceis*enterica* serovars, namely Saintpaul, Braenderup, and Heidelberg were identified, *S*. Saintpaul being predominant [4]. Also in Namibia from a total samples examined, 10.9% were found to be positive for *Salmonella*. A total of 29 *Salmonella* serovars were identified from one or both sample types, with *S*. Chester being the most frequent isolated, followed by *S*. Schwarzengrund and *S*. Chartres [88]. In Nigeria Lagos among *Salmonella* species isolated from the stool samples collected from food handlers were *S.* Typhi, *S.* Enteritidis, *S.*Choleraesuis, *S.* ParatyphiA and *S.* Arizonawith prevalence of 6.8%, 5.3%, 2.9%, 1.5% and 0.5%, respectively. *S*. Enteritidisand *S*. Typhimuriumwere isolated from fecal cattle samples with prevalence of 12% and 3%, respectively [87].
Like other developing countries, in Ethiopia *Salmonella* species are the major cause of food born disease and it cause mortality and morbidity particularly in human and animal. A cross-sectional study was conducted in central region of Ethiopia to determine the prevalence and distribution of *Salmonella* serotypes in minced meat beef, mutton and pork from retail supermarkets reported that out of the total meat samples examined, 44 (14.7%) were *Salmonella* positive. *Salmonella* was detected in 14.4% (23/160) minced beef, 14.1% (12/85) mutton and 16.4% (9/55) pork samples analyzed. Of the total 44 *Salmonella* positive samples, nine different serotypes were identified. The dominant serotype identified was *S.* Infantis (36.4%) followed by *S.*Braenderup (29.5%), *S.* Anatum (9.1%) and *S.* Bovismorbificans (9.1%). Other *Salmonella* serotypes isolated include *S.* Vejle, *S.* Dublin, *S.*Saintpaul, *S.*Infantis and *S.* Braenderup were isolated from minced beef, mutton and pork samples whereas *S.* Dublin and *S.* Saintpaul were isolated only from minced beef samples [18].
In recent time a study by Fentabil Getnet [90] in the central part of Ethiopia (Addis Ababa) reported that eight *Salmonella* species were isolated among 233 food handlers giving an isolation rate of 3.4%, all were females. Of these; two *S.* Typhi, one *S.* ParatyphiA and five unidentified *Salmonella* species were isolated. A study conducted byBayleyegnMolla*et al*. [7] different serotypes were identified from slaughtered cattle (4.2%), camels (16.2%), chicken meat and giblets (23.6%). Among this predominant serovars were *S.* Braenderup, *S.* Dublin and *S.*Saintpaulfollowed by *S.* Typhimurium(including var. Copenhagen) and *S.* Anatum. Similarly, a study in Addis Ababa from September 2003 to February 2004 documented

that *Salmonella* species isolated from food items and stool samples, of which, 7.8% were positive for *Salmonella* from food samplesand of sixty-eight stool samples five gave positive result (7.4%). About 14% of chicken carcass, 11.3% of pork, 10.8% of mutton, 8.5% of minced beef, 2.1% of cottage cheese, 2.3% of fish and none of the ice cream yielded *Salmonella.* A total of 14 different serotypes out of 98 *Salmonella* isolates were identified. *Salmonella* Newport (41.8%) was the most prevalent serotype, followed by *S.* Braenderup (12.2%), *S.* Hadar (8.2%), *S.* Typhimurium (7.1%), *S.* Dublin (6.1%) and *S.* Haifa (6.1%). Less commonly isolated *Salmonella* serotypes included: *S.* Infantis, *S.* Kentucky, *S.* Bovismorbificans, *S.* Anatum, *S.* Zanzibar, *S.* Kottbus, *S.* Saintpaul and *Salmonella* Newport and *S.* Kentucky were reported for the first time in Ethiopia. *Salmonella* Newport was isolated from all sample types except ice cream, while *S.* Braenderup, *S.* Kottbus, *S.* Saintpaul were detected only from chicken carcass, pork and minced beef samples, respectively [17].

In Bahir Dar *Salmonella* isolates from cattle consisting of *Salmonella* Typhimurium, *Salmonella* Newport, *Salmonella* Haifa, *Salmonella* Heidelberg, *Salmonella*Infantis, and *Salmonella*Mishmarhaemek were identified. *Salmonella* Typhimurium and *Salmonella* Newport were most frequently isolated while *Salmonella* Heidelberg and *Salmonella*Mishmarhaemek were isolated least [91]. According to Sefinew Alemu and BayleyegnMolla [91] *Salmonella* was detected from liver, mesenteric lymph nodes, carcass swab, and intestinal content samples with prevalence of 1.1%, 3.2%, 4.8%, and 5.9%, respectively. According toBayehAbera*et al*. [92] 1.6% food handler were found positive for *S.* Typhi. Of these, 6.5% were suffering from diarrhea.

5. ANTIMICROBIAL SUSCEPTIBILITY OF *SALMONELLA*

5.1. Drug resistivity of *Salmonella* species

Until recently, *Salmonella* species were highly susceptible to the most commonly used antibiotics [93]. The resistance of *Salmonella* to a single antibiotic was first reported in the early 1960s [94; 34]. The most widely used antibiotics for treatment of salmonellosis in humans is a group of fluoroquinolones and third-generation cephalosporins. The earlier drugs chloramphenicol, ampicillin, amoxicillin and trimethoprim-sulfamethoxazole are occasionally used as alternatives [95]. Since then, the isolation frequency of *Salmonella* strains resistant to one or more antibiotics have increased in the Saudi Arabia, United States, United Kingdom and other countries of the world. This is due to the increased and uncontrolled use as well as easy accessibility to antibiotics in many countries of the world [96; 34]. Emerging resistance in *Salmonella* has been described especially in Africa and Asia and the appearance of *Salmonella* Typhimurium DT104 in the late 1980s raised main public health concern, thereby threatening the lives of infected individuals stated that multi-resistance occurred in *Salmonella* species (97; 94; 34].

Fluoroquinolones are the most commonly used antimicrobial agent for the treatment of invasive *Salmonella* infections in adults [98]. *Salmonella* isolates with decreased susceptibility to fluoroquinolones (but that are not resistant to fluoroquinolones) commonly have a single point mutation in a chromosomal gene [99]. Third-generation cephalosporins, such as ceftriaxone, are commonly used for treatment of invasive *Salmonella* infections in children because of their pharmacodynamic properties and low prevalence of resistance to these agents. Therefore, there is concern about the potential emergence of ceftriaxone-resistant *Salmonella*. The first reported case of domestically acquired ceftriaxone-resistant *Salmonella* was in a 12-year-old child in Nebraska [100].

High degree of multiple drug resistance was observed in *Salmonella* isolates from different food animal samples that indicate drug resistance of *Salmonella* is becoming a crucial health problem in this part of the world. The prevalence of *Salmonella* strains resistant to more than one antibiotic may be due to the comprehensive use of antibiotics included in feeds as growth promoters and due to the widespread use of antibiotics in food animal industries. Studies in Dubai, United Arab Emirates reported that all the isolates (100%) showed resistance to cephalexin and rifampicin. A high degree of resistance was also observed for ampicillin and tetracycline while 87.88% of these isolates were sensitive to ciprofloxacin and amikacin [74]. A cross-sectional study was conducted from November 2006 to April 2007 in Bhutan was reported the antimicrobial susceptibility of *Salmonella* isolates. Among seven antimicrobial tested, resistance was highest for nalidixic acid (96.15%) followed by amoxicillin (11.54%) and cephalexin (5.77%). Ciprofloxacin and sulpha-trimethoprim showed resistance of 1.92% each. While gentamicin was sensitive to all the isolates tested, chloramphenicol had a sensitivity of 98.08%. The isolates were resistant to a maximum of three antimicrobials. All eight *Salmonella* Typhimurium isolates were resistant to nalidixic acid with one isolate showing simultaneous resistance to cephalexin. *Salmonella* Enteritidis was resistant to five of the seven antimicrobials tested with simultaneous multidrug resistance in up to three antimicrobials [3].

In Korea antimicrobial susceptibility of the *Salmonella* isolated from pig varies as follows: norfloxacine (75%), ciprofloxacin (67.5%), amikacin (60%), colistin (60%), enrofloxacin (55%). All of isolates were resistant to erythromycin, penicillin, tetracycline and lincomycin [84].In Tehran, Iran, among the variety of antibiotics tested, the highest resistance was found with nalidixic acid followed by tetracycline, trimethoprim, and streptomycin. The percentages resistance of isolates from meat samples was 36.8%, 21%, 26.3%, and 5.3%, respectively. About 23.5% of the *Salmonella* strains were multi-resistant to two or more antibiotic families. In overall, the degree of resistance of *Salmonella* to nalidixic acid was greater than other tested antibiotics [101].

In USA study conducted on cattle carcass and feces showed that *Salmonella* isolates were tested for antimicrobial drug susceptibility. Among this 97% (n =101) of the isolates were resistant to at least one antimicrobial drug; however, only 4.0% were resistant to two or more. The most common resistance was to sulfamethoxazole. These results indicate that

the presence of microorganisms resistant to antimicrobial drugs is common in cattle and beef [102]. *Salmonella* isolates recovered from dairy cows had relatively little resistance to the antimicrobial agents; 83.0% of the isolates were susceptible to all antimicrobials tested [29]. Another study in USA indicated that of the 18 *Salmonella*-positive lymph node samples, 3 contained multidrug-resistant *Salmonella*. All three of these samples were from lymph nodes removed from the carcasses of cull cattle [103].

In Brazil *Salmonella* isolates from broiler carcasses observed resistance to colistin, novobiocin, erythromycin and tetracycline in 100% isolates. Strains showed intermediate resistance at different levels to kanamycin (1.25%), enrofloxacin (3.75%), neomycin (3.75%), fosfomycin (20%), sulphonamides (86.25%) and nitrofurantion (90%). Resistance to ciprofloxacin, norfloxacin, gentamicin, polymyxin B, sulphametrim and sulphazotrim was not found [104]. In Canada a study conducted in swine farm reported that more than half of the isolates (53.4%) were susceptible to all of the 18 antimicrobials. No resistance was observed to amikacin, amoxicillin/clavulanic acid, cefoxitin, ceftiofur, ceftriaxone, cephalothin, ciprofloxacin, imipenem or nalidixic acid. Less than 1% of isolates were resistant to apramycin, gentamicin and trimethoprim/sulfamethoxazole. Higher frequencies of resistance were observed for chloramphenicol (4.7%), ampicillin (7.8%), kanamycin (11.8%), sulfamethoxazole (21.1%), streptomycin (25.5%) and tetracycline (38.8%) [105]. Study in Rockville, Maryland showed that of the 257 *Salmonella* isolates obtained, 54 isolates (21%) were resistant to at least one antimicrobial [106].

Studies in European countries indicated that *Salmonella* were isolated from food producing animals and tested for antimicrobial susceptibility. In Japan *Salmonella* isolates resistant to ampicillin, dihydrostreptomycin, kanamycin, oxytetracycline, chloramphenicol, bicozamycin, nalidixic acid, oxolinic acid and trimethoprim were obtained from healthy animals and diagnostic sample submissions. *Salmonella* Dublin was isolated only from cattle and showed resistance to older quinolones [107]. Another study in Japan on food producing animal was indicated that resistance was found for 8 of 11 antimicrobials tested, at the following rates: 46.4% for dihydrostreptomycin followed by ampicillin and oxytetracycline (both 8.9%) [108]. In Faisalabad, Pakistan also *Salmonella* isolates showed 100% resistance against bacitracin, erythromycin and novobiocin [109]. Similarly in Canary Islands, Spain all isolates were susceptible to all of the tested antimicrobial agents, which included ampicillin, amoxicillin/ clavulanic acid, tetracycline, enrofloxacin, chloramphenicol, nalidixic acid, piperacillin and trimethoprim-sulfamethoxazole [72]. Another study in Spain also showed that all isolates were multi-resistant. The average number of resistances per strain increased from 3.98 in 1993 to 5.00 in 2006. An increase in the incidence of resistance was observed between 1993 and 2006 for cephalothin, enrofloxacin and tetracycline [110].

Resistance in *Salmonella* has been described especially in Africa countries and the appearance of *Salmonella* Typhimurium DT104 in the late 1980s raised main public health concern [97; 94; 34]. In Namibia study conducted in food animals documented that from the *Salmonella* isolates, 19.7% were resistance to one or more of the antimicrobials whereas 80.3% were susceptible to all 16 antimicrobials tested. Resistance to sulfisoxazole and the trimethroprim-suflamethoxazole combination were the most common [88]. In Sudan, 46.8% of *Salmonella* serotypes isolated from animal were found to be resistant to at least one of the tested nine antimicrobial agents and 45 isolates (37.8%) were found to be multidrug-resistant. Ciprofloxacin and gentamicin were found to be highly active against the isolates. But the isolates showed high resistance to ampicillin, chloramphenicol, tetracycline and sulfamethoxazole-trimethoprim [111]. According to Fasure*et al*. [112] reported in Nigeria *Salmonella* isolates were 100% resistant to ampicillin, 90.6% to tetracycline and moderately sensitive to nalidixic acid (62.5%). Fluoroquinolone resistant *S.* Typhimuriumstrains from food animal were also observed. In Meknès, Morocco, 43 (75.43%) *Salmonella* isolates were resistant to one or more antimicrobials. Out of 43 resistant *Salmonella* isolates, 17 (39.5%) showed multiple resistance to two or more different antimicrobials. Resistance to tetracycline, sulfamides, trimethoprim and streptomycin was the most frequent [113]. According to Gideon *et al*. [4] in Kenya, antimicrobial resistance was found in 35.7% of the isolates. The *S*. Heidelberg isolates were susceptible to all the antimicrobials tested. Multidrug-resistance was found in 7.1% of the *Salmonella* isolates.

Salmonella isolates from Ethiopia at different times showed that *Salmonella* were susceptible to ciprofloxacin, cotrimoxazole, and chloramphenicol [2], resistance to streptomycin (24/32, 75%), ampicillin (19/32, 59.4%), tetracycline (15/32, 46.9%), spectinomycin (13/32, 40.6%) and sulfisoxazole (13/32, 40.6%) [17]. Studies conducted in Addis Ababa documented that the antimicrobial susceptibility profile showed all except one were resistant to ampicillin and all isolates were resistant at least to one of antimicrobials tested [90]. Antimicrobial resistance was most common among *Salmonella* isolated from carcass (18/29, 62.1%) followed by pork (5/22, 22.7%). Multiple antimicrobial drug resistance was observed in 23 *Salmonella* isolates (23.5 %) [17]. Eleven of the 28 (39.3%) *Salmonella* isolate from cattle were resistant to one or more of the antimicrobials tested. Resistance was shown to ampicillin, chloramphenicol, gentamycin, norfloxacin, polymyxin-B, streptomycin, tetracycline, and trimethoprim. Four of 11 (36.4%) were multiple antimicrobial resistant. All the isolates tested were susceptible to the antimicrobial effects of gentamycin, norfloxacin, and trimethoprim. Eleven, four, and two isolates of the 28 were resistant to streptomycin, tetracycline, and ampicillin, respectively [91]. According to BayleyegnMolla*et al*. [7], fifty-one (63.7%) of the 80 *Salmonella* strains were resistant to one or more antimicrobials of which 42 (52.5%) displayed multiple-drug resistance. Among the strains, 51.2% were resistant to sulfisoxazole, 46.2% to spectinomycin, 45% to amoxicillin-clavulanic acid and ampicillin, 41.2% to tetracycline and 30% to chloramphenicol.

5.2. Resistance mechanism of *Salmonella* species

Before, during and after infection non- resistant *salmonella* can be multi-drug resistant easily due to transfer of resistance genes from resistant bacteria or from the environment. The two mechanisms critical for the spread of antimicrobial resistance in *Salmonella* populations are (i) horizontal transfer of antibiotic resistance genes and (ii) clonal spread of antimicrobial drug-resistant *Salmonella* isolates [131]. Antimicrobial resistance genes are typically found on mobile genetic elements, such as integrons and plasmids, which are readily transferred among *Salmonella* strains and between other bacterial species and *Salmonella.* MDR *Salmonella* strains resulting from acquisition of these genetic elements have been found worldwide and are a growing concern for public health and food safety [114].

Salmonella resists the action of antibiotics by the following ways: (i) Inactivation of the antimicrobial agent**:** this is a communal cause of resistance that destroys or inactivates antimicrobial agents. The bacterial pathogens struggle attack by inactivating drugs through chemical modification. One enzyme of this type is ß- lactamase. Several ß- lactamase exist in numerous bacteria. The best known example is the hydrolysis of the ß-lactam ring of Penicillin by the enzyme penicillinase. They are skilled of breaking the ß- lactam ring of penicillin and some cephalosporin [134]. (ii) Efflux or transport of the antimicrobial**:** This resistance mechanism works by pumping the drug out of the cell after it has entered. Some pathogens have plasma membrane translocases, often called efflux pumps, that expels drugs. Because they are relatively nonspecific and can pump many different drugs including quinolones, these transport proteins often are called multi-drug resistance (MDR) pumps. Many are drug/proton antiporters i.e, proton enter the cell as the drug leaves [133]. (iii) Modification of the antimicrobial target site**:** Resistance arises when the target enzyme or cellular structure of the pathogen is changed so that it is no longer susceptible to the drug. This mechanism is found in *Salmonella* and other sulfonamide-resistant bacteria. These organisms have developed an enzyme that has a very high affinity for p-aminobenzoic acid and a very low affinity for sulfonamide. Therefore, even in the presence of sulfonamides, the enzymes work well enough to permit the bacterium to function [122]. (iv) Reduced permeability of the antimicrobial agent: Pathogens often become resistant simply by stopping entrance of the drug. The modification in membrane permeability occurs when new genetic information changes the nature of proteins in the membrane. Such amendments change a membrane transport system pores in the membrane, so an antimicrobial agent can no longer cross the membrane. In *Salmonella* species, resistance to tetracycline, quinolones, and some aminoglycosides have happened by this mechanism. A decrease in permeability can also lead to sulfonamide resistance [133]. Most drug resistance is due to a genetic alteration in the organism, either a chromosomal mutation or the acquisition of a plasmid or transposon [132].

6. KEY ACTIONS

The presence of even small numbers of pathogens in carcass meat may lead to heavy contamination of minced meat when it is cut into pieces; as more microorganisms are added to the surfaces of exposed tissue (Gebisa Ejeta *et al*., 2004). Proper cooking of meat before consumption and improving personal and meat hygiene in the line of meat production from farm to fork should be adopted to ensure the safety of meat and meat products for human consumption (Gebisa Ejeta *et al*., 2004). In addition to this, updated information on carcass is very important for knowing the prevalence of *Salmonella* species and microbial load for suggesting the acceptance of the carcass in relation to the standards and it would also contribute for the realization the current status of commonly used antibiotics and generating information on the possible antibiotics to treat salmonellosis.

7. CONCLUSION

Problems have their origin in the methods of farming of animal foods. Many farmers are illiterate and follow methods of production that are centuries old. They live in very close contact with their animals, often under poor hygienic conditions, thereby increasing the likelihood of food borne zoonoses. However, considerable proportion of patients may not visit health centers unless symptoms are serious due to shortage of resources and lack of awareness. Practicing good sanitary measures, extensive education programs for proper hygiene and improvement of managements are solutions to eliminate the high bacteriological load as well as prevalence of *Salmonella* in cattle carcass. In addition to this, laboratories should have the power and responsibility to investigate product trace-back bacteriological load and report to the stockholders before consumption about the quality and safety of cattle carcass. Both single and multiple antimicrobial resistance patterns to the commonly practiced antimicrobials in the veterinary and public health set up were observed, which is of special concern in developing countries here use of antimicrobials has problems. In animals, there is treatment restriction because of inadequate drug alternatives; therefore, limited drugs are frequently used for treatment; this practice leads resistance to limited antibiotics. In addition to this, multidrug resistance was observed due to lack of restricting, discriminate and appropriate use of antibiotics in the food animal industry. So, restricting the use of antimicrobial agents in food animals, designation of multidrug-resistant *Salmonella* as an adulterant in ground beef, improving the mechanisms for product trace-back investigations and wise and discriminate use of antimicrobials should be practiced to combat the ever increasing situation of antimicrobial resistance.

CONFLICT OF INTEREST: We declare that we have no conflict of interest.

REFERENCES

1. Henson S. The economics of food safety in developing countries. Ecological Science of America Working Paper.2003:3-19.
2. Zellalem A, Nigatu K, Zufan W, Haile G, Alehegn Y, Tesfu K. Prevalence and Antimicrobial resistance of *Salmonella* isolated from lactating cows and contact humans in dairy farms of Addis Ababa: a cross sectional study. Infectious Diseases. 2011:11:1-3.
3. Narapati D. Prevalence and antimicrobial resistance of *Salmonella* in imported chicken carcasses in Bhutan. Master of veterinary public health. M.Sc. thesis, Chiang Mai University. 2007.
4. Gideon MK, Ombui JN, Mitema ES. Serotypes and antimicrobial resistance profiles of *Salmonella* isolates from pigs at slaughter in Kenya.Journal of Infectious Development Ctries. 2010:4:243-248.
5. Hoelzer K, Andrea I, Martin W.Animal contact as a source of human non-typhoidal salmonellosis. Veterinary Research. 2011: 42: 34-42.
6. Libby JS, Halsey AT, Altier C, Potter J, Gyles LC. Pathogenesis of bacterial infections in animals (3rd edition). United State of America. Blackwell Publishing. 2004:143–167.
7. Bayleyegn M, Arthuro M, Danial A. Multiple antimicrobial resistant *Salmonella* serotypes isolated from chicken carcass and giblets in Debre Zeit and Addis Ababa, Ethiopia. Ethiopian Journal of Health Development. 2003:17: 131–149.
8. Fegan N, Vanderlinde P,Higgs G, Desmarchelier P. A study of the prevalence and enumeration of *Salmonella enterica* in cattle and on carcasses during processing. Journal of Food Protection. 2005:68:1147–1153.
9. Sibhat B, Bayleyegn M, Zerihun A, Anne M, Cole L, Boerlin P, Wilkie E, Perets A, Mistry K, Wondosen A. Salmonella serovars and antimicrobial resistance profiles in beef cattle, slaughterhouse personnel and slaughterhouse environment in Ethiopia. Zoonoses and Public Health. 2011:58: 102-109.
10. Hjartardottir S, Gunnarsson E, Sigvaldadottir J. Salmonella in sheep in Iceland. Acta VeterinariaScandinavica. 2002:43: 43-48.
11. Akafete T, Haileleul N. Assessment of risk factors and prevalence of Salmonella in slaughtered small ruminants and environment in an export abattoir, Modjo, Ethiopia. American-Eurasian Journal of Agriculture and Environmental Science. 2011:10: 992-999.
12. Wondwossen A, Davies PR, Turkson P, Morgan MWE, Funk JA, Altier C, Thakur S. Characterization of antimicrobial resistant phenotypes and genotypes among *Salmonella enterica* recovered from pigs on farms, from transport trucks, and from pigs after slaughter. Journal of Food Protection. 2004:67*:* 698–705.
13. Endrias Z, Cornelius P. Resistance pattern of Salmonella serotypes isolated from food items and personnel in Addis Ababa, Ethiopia. Tropical Animal Health and production. 2009:41: 241-249.
14. Center for Diseases Control and Prevention. Coordinating centre for infectious diseases / Division of bacteria and mycotic diseases. Bulletin. 2006.
15. Olowe OA, Olayemi A, Eniola KIT, Adeyeba, AO. Etiological agents of diarrhea in children under 5 years of age in Osogbo. African Journal of Clinical andExperimental Microbiology. 2007: 4: 62–66.
16. Siddiqui FJ, Rabbani F, Hasan R, Nizami SQ, Bhutta ZA. Typhoid fever in children: some epidemiological considerations from Karachi, Pakistan. International Journal of Infection Disease. 2006: 10: 215–222.
17. Endrias Z.Prevalence, distribution and antimicrobial resistance Profile of *Salmonella* isolated from food items and Personnel in Addis Ababa, Ethiopia.M.Sc. Thesis, Addis Ababa University. 2004.
18. Gebisa E, Bayleyegn M, Daniel A, Anne M. Salmonella serotypes isolated from minced beef, mutton, and pork in Addis Ababa, Ethiopia. Revue Medical and Veterinary. 2004:155: 547-551.
19. European Food Safety Authority. A quantitative microbiological risk assessment on Salmonella in meat: source attribution for human salmonellosis from meat:Scientific opinion of the panel on Biological hazards. European Food Safety Authority. 2008:625: 1-32.
20. Santos RL, Tsolis RM, Baumler AJ, Adams LG. Pathogenesis of Salmonella-induced enteritis. Brazilian Journal of Medical and Biological Research. 2003:36: 3-12.
21. Jones BD. Salmonella gene invasion regulation: a story of environmental awareness. The Journal of Microbiology 2005:43: 110-117.
22. Porwollik S, Santiviago CA, Cheng P, Florea L, Jackson S, McClelland M. Differences in gene content between Salmonella enterica serovar Enteritidis isolates and comparison to closely related serovars Gallinarum and Dublin. Journal of Bacteriology. 2005:187: 6545-6555.
23. Solari CA, Mandarino JR, Panizzutti MHM, Farias RHG. A new serovar and a new serological variant belonging to Salmonella enterica subspecies arizonae. Memorias do Instituto Oswaldo Cruz 2003:98: 501-502.
24. Vila JH, Paniagua CD, Escobar CDF, Martinez CJ, Santigosa NP. *Salmonella*in free living terrestrial and aquatic turtles. Veterinary Microbiology 2006:119: 311-315.
25. Wray C, Davies RH. *Salmonella* infections in cattle. In: *Salmonella* in domestic animals, (Wray, C. and A. Wray). New York, Center for Agricultural and Bioscience International Publishing. 2000:169–190.
26. Sheila MM, Simon P. Salmonellosis in cattle; American association of bovine practitioners 36th Annual Conference, School of Veterinary Medicine, University of Wisconsin. 2003.

27. Acha PN, Szyfres B. Zoonoses and communicable diseases common to man and animals (3rd edition). Washington DC: Pan American Health Organization 2001:1: 233-246.
28. John K, Bradford PS. Profitable strategies to control salmonellosis in dairy cattle. Sydney University Veterinary Center Camden New South Wales, Australia.Published in International Veterinary Information Service with the Permission of the World Boxing Council. 2004.
29. Blau DM, MccluskeyBJ, LadelySR, DargatzDA, Fedorka-cray PJ, Ferris KE, Headrick ML.*Salmonella* in dairy operations in the United States: prevalence and antimicrobial drug susceptibility.Journal of Food Protection2005:68: 696–702.
30. Cobbold RN, Rice DH, Davis MA, Besser TE, Hancock DD. Long-term persistence of multi-drug-resistant *Salmonellaenterica* serovar Newport in two dairy herds. Journal of American Veterinary Medicine Association. 2006:228: 585-591.
31. Nollet N, Maes D, De Zutter L, Duchateau L, Houf K, Huysmans K, Imberechts H, Geers R., de Kruif A, Hoof J. Risk factors for the herd level bacteriologic prevalence of *Salmonella* in Belgian slaughter pigs. Preventive Veterinary Medicine 2004:65: 63-75.
32. Nielsen TD, Nielsen LR, Toft N, Houe H.Association between bulk-tank milk *Salmonella* antibody level and high calf mortality in Danish dairy herds. Journal of Dairy Science. 2010:93: 304-310.
33. Fossler CP, Wells SJ, Kaneene JB, Ruegg PL, Warnick LD, Bender JB, Eberly LE, Godden SM, Halbert LW, Campbell AM, Bolin CA, Zwald AM. Herd-level factors associated with isolation of Salmonella in a multi-state study of conventional and organic dairy farms. Salmonella shedding in calves. Preventive Veterinary Medicine. 2005:67*:* 39*–53.*
34. Pui CF, Wong WC, Chai LC, Tunung R, Jeyaletchumi P, Noor Hidayah MS, Ubong A, Farinazleen MG, Cheah YK, Son R. *Salmonella*: a food borne pathogen.International Food Research Journal 2011:18: 465-473.
35. Newell DG, Koopmans M, Verhoef L, Duizer E, Aidara-Kane A, Sprong H, Giessen J, Kruse H. Food borne diseases-the challenges of 20 years ago still persist while new ones continue to emerge. International Journal of Food Microbiology 2010:139: 3-15.
36. Pegues DA, Ohl ME, Mille SI. *Salmonella* species, including *Salmonella* Typhi. In: Principles and practice of infectious diseases, (Mandell, G.L., Bennett, J.E., Dolin, R., Mandell, D. and Bennett's). Elsevier Churchill Livingstone. 2005:2: 2636-2654.
37. American Academy of Pediatrics. *Salmonella* infections. Report of the committee on infectious diseases. American Academy of Pediatrics. 2006:27: 579-584.
38. Medus C, Smith KE. *Salmonella* outbreaks in restaurants in Minnesota, 1995 through 2003: evaluation of the role of infected food workers. Journal of Food Protection. 2006:69: 1870-1878.
39. Centers for Disease Control and Prevention. Preliminary food net data on the incidence of infection with pathogens transmitted commonly through food selected sites, United States. Morbidity and Mortality Weekly Report. 2004:53: 338-343.
40. Centers for Disease Control and Prevention. Multistate outbreak of human *Salmonella* infections associated with exposure to turtles, United States. Morbidity and Mortality Weekly Report. 2008:57: 69-72.
41. Swanson SJ, Snider C, Braden CR. Multidrug-resistant *Salmonella enterica* serotype Typhimurium associated with pet rodents. New England Journal of Medicine. 2007:356: 21-28.
42. Paszkiewicz W, Renatapyz L.Bacterial contamination of calf carcasses during production cycle.Bulletin Veterinary Institute Pulawy. 2012:56: 47-49.
43. Anderson ME, Marshall RT, Dickson JS. Estimating depths of bacterial penetration in to post-rigor carcass tissue during washing. Journal of Food Safety. 1992:12: 191-198.
44. Galland JC. Risks and prevention of contamination of beef carcasses during the slaughter process in the United States of America.Revue Science and Technology Office of International Epizootics. 1997:16: 395-404.
45. Food and Agriculture Organization. Principles of meat processing hygiene and regulatory practices. In meat processing technology for small to medium-scale producers. Regional Office for Asia and the Pacific Bangkok. *Animal Production and Health Paper* 2010:91:1-43.
46. Hussein MA, El-Ghareeb WR, Lotfy O. Shelf life improvement of camel meat treated with potassium sorbet 0.3%. Journal of American Science. 2012:8: 507-511.
47. Zweifel C, Fischer R, Stephan R. Microbiological contamination of pig and cattle carcasses in different small-scale Swiss abattoirs. Meat Science. 2005:78: 225-231.
48. Abdalla MA, Suliman SE, Ahmed DE, Bakhiet AO. Estimation of bacterial contamination of indigenous bovine carcasses in Khartoum (Sudan). African Journal of Microbiology Research. 2009:3: 882- 886.
49. Adzitey F, Teye GA, Kutah WN, Adday S. Microbial quality of beef sold on selected markets in the Tamale Metropolis in the Northern Region of Ghana. Livestock Research for Rural Development. 2011:23: 105-112.
50. Bhandare SG,Paturkar AM, Waskar VS, Zende RJ. Bacteriological screening of environmental sources of contamination in an abattoir and the meat shops in Mumbai, India. Asian Journal of Food and Agro-Industry. 2009:**2**: 280-290.

51. Vanselow BA, Hornitzky MA, Walker KH, Eamens GJ, Bailey GD, Gill PA, Coates K, Corney B, Cronin JP, Renilson S.*Salmonella* and on-farm risk factors in healthy slaughter-age cattle and sheep in eastern Australia. Australian VeterinaryJournal 2007:85: 498-502.
52. Letellier A, Beauchamp G, Vremont EG, Dallaire S, Hurnik D. sylvain QS. Risk factors at slaughter associated with presence of *Salmonella* on hog carcasses in Canada. Journal of Food Protection. 2009:72: 2326–2331.
53. Warnick LD, Crofton LM, Pelzer KD, Hawkins MJ. Risk factors for clinical salmonellosis in Virginia, United State of America cattle herds. Preventive Veterinary Medicine. 2001:49: 259-275.
54. Nielsen LR, Warnick LD. Greiner M. Risk factors for changing test classification in the Danish surveillance program for *Salmonella* in dairy herds. Journal of Dairy Science. 2007:90: 2815-2825.
55. Corrier DE, Purdy CW, DeLoach JR. Effects of marketing stress on fecal excretion of *Salmonella* spp. in feeder calves. American Journal of Veterinary Research 1990:51: 866-869.
56. Libby S, Lesnik P, Hasegawa M, Kurth C, Belcher J, Fierer J. Guiney G. Characterization of spv locus in *Salmonella enterica* serovar *arizonae*. Infection and Immunology 2002:70: 3290-3294.
57. Hall MLM, Rowe B.*Salmonella arizonae*in the United Kingdom from 1966–1990. Journal of Epidemiology and Infection 1992:108: 59–65.
58. Aoust JY, Daley E, Crozier M. Sewell AM. Pet turtles: a continuing international threat to public health. American Journal of Epidemiology 1990:132: 233-238.
59. Biswas AK, Kondaiah N, Anjaneyulu ASR, Mandal PK. Causes, concerns, consequences and control of microbial contaminants in meat-a review. International Journal of Meat Science 2011:1: 27-35.
60. Samelis J.Managing microbial spoilage in meat industry, in food spoilage microorganism. Edited by C. de W. Blackburn, Cambridge: Wood Head Publishing Limited. 2006:213-286.
61. Nychas GJ, Skandamis EPN, Tassou CC. Koutsoumanis K. Meat spoilage during distribution. Meat Science. 2008:78: 77-89.
62. Doulgeraki AI, Paramithiotis S, Kagkli DM. Nychas GJE. Lactic acid bacteria population dynamics during minced beef storage under aerobic or modified atmosphere packaging. Food Microbiology. 2010:27:1028–1034.
63. Frew T. Microbiological quality and safety of street vended raw meat in Jijiga town: South east Ethiopia. M.Sc. thesis, Addis Ababa University. 2011.
64. Buck JD, Immerseel FV, Haesebrouck F, Ducatelle R. (2004). Colonization of the chicken reproductive tract and egg contamination by *Salmonella*. Journal of Applied Microbiology. 2004:97: 233-245.
65. Chiu CH, Su LH, Chu C. *Salmonella enterica* serotype Choleraesuis: epidemiology, pathogenesis, clinical disease, and treatment. Clinical Microbiology Reviews. 2004:17: 311-322.
66. Sadeyen JR, Trotereau J, Velge P, Marly J, Beaumont C, Burrow PA, Bumstead N, Lalmanach AC. *Salmonella* carrier state in chicken: comparison of expression of immune response genes between susceptible and resistant animals. Microbes and Infection. 2004:6: 1278-1286.
67. Korsak N, Degeye JN, Etiene G, Beduin JM, China B, Ghafir Y, Daube G. Use of serological approach for prediction of *Salmonella* status in an integrated pig production system. International Journal of Food Microbiology. 2006:108: 246-254.
68. Oscar TP. A quantitative risk assessment model for *Salmonella* and whole chickens. International Journal of Food Microbiology. 2004:93: 231-247.
69. Jong BD, Ekdahl K. The comparative burden of salmonellosis in the European Union member states, associated and candidate countries. Biomedical and Central Public Health. 2006:6: 1-9.
70. Fisher IS. International trends in *Salmonella* serotypes 1998-2003-a surveillance report from the Enter-net International Network. Eurosurveillance. 2004:9: 45-47.
71. Aftab M, Rahman A, Qureshi MS, Akhter S, Sadique U, Sajid A, Zaman S. Level of *Salmonella* in beef of slaughtered cattle at Peshawar.The Journal of Animal and Plant Sciences. 2012:22: 24-27.
72. Tejedor MT, Gonzalez M, Rodríguez NF, Gutiérrez C. Prevalence, serotypes and antimicrobial resistance patterns of *Salmonella* isolates from apparently healthy camels in Canary Islands (Spain).Journal of Came lid Science. 2010:3: 44-48.
73. Wernery U. The prevalence of *Salmonella* infections in camels (Camelus dromedarius) in the United Arab Emirates. British Veterinary Journal. 1992:148: 445-450.
74. Khan MA, Suryanarayan P, Ahmed MM,Vaswani RB, Faheem SM.Antimicrobial susceptibility of *Salmonella* isolates from chicken meat samples in Dubai, United Arab Emirates.International Journal of Food, Nutrition and Public Health 2010:3: 149-159.
75. Fegan N, Vanderlinde P, Higgs G, Desmarchelier P. Quantification and prevalence of *Salmonella* in beef cattle presenting at slaughter. Journal of Applied Microbiology 2004:97: 892–898.
76. Boonmar S, Markvichitr K, Chaunchom S, Chanda C, Bangtrakulnonth A, Pornrunangwong S, Yamamoto S, Suzuki D, Kozawa K, Kimura H, Morita Y. *Salmonella* prevalence in slaughtered buffaloes and pigs and antimicrobial susceptibility of isolates in Vientiane, Lao People's Democratic Republic.Journal of Veterinary Medicine and Science 2008:70: 1345–1348.

77. Padungtod P, Kaneene JB. *Salmonella* in food animals and humans in northern Thailand. International Journal of Food Microbiology. 2006:108: 346-354.
78. Maharjan M, Joshi V, Joshi DD, Manandhar P. Prevalence of *Salmonella* species in various raw meat samples of a local market in Kathmandu. Part II. Trends in the Study of Disease Agents. 2006:1081: 249-256.
79. Bao VN. Prevalence of *Salmonella* and *Campylobacter* spp. from broiler meat in abattoirs at Ho Chi Minh city, Vietnam. Free University, Berlin dissertation. 2005.
80. Sikder AJ, Islam MA, Rahman MM, Rahman MB. Sero-prevalence of *Salmonella* and *Mycoplasma gallisepticum*infection in the six model breeder poultry farms at Patuakhali district in Bangladesh. International Journal of Poultry Science 2005:4: 905-910.
81. Vaidya VM, Paturkar AM, WaskerAS, Zende RJ, Rawool DB. Detection of indicator organisms in poultry carcass sites in an organized slaughterhouse. Journal of Muscle Foods. 2005:16: 289-297.
82. Rahman H, Bhattacharya DK, Murugkar HV. Prevalence of *Salmonella* in poultry in North Eastern India. Indian Journal of Veterinary Research. 2004:13: 1-7.
83. Bajaj BK, Sharma V, Thakur RL, Koul S. Incidence of *Salmonella* in poultry and meats and growth inhibition of *Salmonella* Enteritidis by organic acids. Journal of Food Science and Technology. 2003:40: 556-558.
84. Jung H, Lee S, Kim C, Sunwoo S, Lyoo TS. Serovars distribution and antimicrobial resistance patterns of *Salmonella* species isolated from the swine farms and slaughter houses in Korean. Journal of Veterinary Research. 2011:51: 123-128.
85. Kaferstein FK. Food safety in food security and food trade. Food safety as a public health issue for developing countries. 2020 vision for food, agriculture and the environment. Focus. 2003:10: 2-17.
86. Zessin KH. Emerging diseases; a global and Biological perspective. Journal of Veterinary Medicine.2006: 53: 7-10.
87. Smith SI, Bamidele M, Goodluck HA, Fowora MN, Omonigbehin EA, Opere BO, Aboaba OO.Antimicrobial susceptibilities of *Salmonella* isolated from food handlers and cattle in Lagos, Nigeria. International Journal of Health Research 2009:2: 189-193.
88. Renatus PS, Elisabetta DG, Percy MC, Godwin PK. Prevalence and antimicrobial resistance pattern of *Salmonella* in animal feed produced in Namibia. *VeterinariaItaliana*. 2012:**48**: 125-132.
89. Yeung RMW. Consumer perception of food risk in chicken meat. Nutrition and Food Science 2001:31: 270-279.
90. Fentabil G.Isolation of *Salmonella* species among apparently healthy food handlers of Addis Ababa University students' cafeteria, Ethiopia. M.Sc. thesis, Addis Ababa University.2011.
91. Sefinew A, Bayleyegn M. Prevalence and antimicrobial resistance profiles of *Salmonellaenterica* serovars isolated from slaughtered cattle in Bahir Dar, Ethiopia. Tropical Animal Health and Production. 2012:44: 595-600.
92. Bayeh A, Fantahun B, Belay B. (2010). Prevalence of *Salmonella* Typhi and intestinal parasites among food handlers in Bahir Dar Tawon, North West Ethiopia. Ethiopian Journal of Health Development. 2010:24: 46-50.
93. Mandomando I, Jaintilal D, Pons MJ, Valles X, Espasa M, Mensa L, Sigallque B, Alonso PL, Ruiz Z. Antimicrobial and mechanism of resistance in *Shigella* and *Salmonella* isolated from children under five years of age with diarrhea in rural Mozampique. American Society for Microbiology 2009:53: 2450-2454.
94. Montville TJ, Matthews KR. Food microbiology: an introduction (2nd edition). United States of America. *American Society of Medical Press*, Washington.2008.
95. World Health Organization. WHO media centre http://www.who.int/mediacentre/factsheets/fs139/en/print.html. 2005: (Accessed on 08/08/2007).
96. Yoke-Kqueen C, Learn-Han L, Noorzaleha AS, Son R, Sabrina S, Jiun-Horng S, Chai-Hoon K. Characterization of multiple antimicrobial resistant *Salmonellaenterica*subspeciesisolated from indigenous vegetables and poultry in Malaysia. Letters in Applied Microbiology. 2007:46: 318-324.
97. Van TTH, Moutafis G, Istivan T, Tran LT, Coloe PJ. Detection of *Salmonella* species in retail raw food samples from Vietnam and characterization of their antibiotic resistance. Applied and Environment Microbiology. 2007:73: 6885-6890.
98. Angulo FJ, Johnson KR, Tauxe RV, Cohen ML. Origins and consequences of antimicrobial-resistant non-typhoidal *Salmonella* implications for the use of fluoroquinolones in food animals. Journals of Microbial Drug Resistant. 2000:6: 77-83.
99. Crump J, Barrett TJ, Nelson JT, Angulo FJ. Re-evaluating fluoroquinolone break points for *Salmonella enterica* serotype Typhi and for non-Typhi salmonellae. Clinical Infectious Disease. 2003:37: 75–81.
100. Anderson AD, Nelson JM, Rossiter S, Angulo FJ. Public health consequences of use of antimicrobial agents in food animals in the United States.Microbial Drug Resistance 2003:9: 373-379.
101. Dallal SMM, Taremi M, Gachkar L,Modarressi S, Sanaei M, Bakhtiari R, Yazdi MKS, Zali MR. Characterization of antibiotic resistant patterns of *Salmonella* serotypes isolated from beef and chicken samples in Tehran.Jundishapur Journal of Microbiology. 2009: 2: 124-131.
102. Fluckey WM,LoneraganGH, Warner R,Brashears MM.Antimicrobial drug resistance of *Salmonella* and *Escherichia coli* isolates from cattle feces, hides, and carcasses.Journal of Food Protection. 2007:70: 551–556.

103. Arthur TM, Brichta-harhay DM, Bosilevac JM, Guerini MN, kalchayanand N, Wells JE, Shackelford SD, Wheeler LT, koohmaraie M. Prevalence and characterization of *Salmonella* in bovine lymph nodes potentially destined for use in ground beef.Journal of Food Protection. 2008:71: 1685–1688.
104. Cardoso, MO, Ribeiro AR, Santos RL, Pilotto F, Moraes LS, Salle PT, Rocha SL, Nascimento PV. Antibiotic resistance in *Salmonella* Enteritidis isolated from broiler carcasses. Brazilian Journal of Microbiology. 2006:37: 368-371.
105. Rajic A, McFall ME, Deckert AE, Smith RR, Manninen K, Poppe C, Dewey CE, McEwen SA. Antimicrobial resistance of *Salmonella* isolated from finishing swine and the environment of 60 Alberta swine farms. Veterinary Microbiology. 2004:104: 189–196.
106. Li X, Bethune LA, Jia Y, Lovell RA, Proescholdt TA, Benz SA, Schell TC, Kaplan G, McChesney DG. Surveillance of *Salmonella* prevalence in animal feeds and characterization of the *Salmonella* isolates by serotyping and antimicrobial susceptibility. Food Borne Pathogens and Disease. 2012: 9: 692-698.
107. Esaki H, Morioka A, Ishihara K, Kojima A, Shiroki S, Tamura Y, Takahashi T. Antimicrobial susceptibility of *Salmonella* isolated from cattle, swine and poultry: report from the Japanese veterinary antimicrobial resistance monitoring program.Journal of Antimicrobial Chemotherapy. 2004:53: 266–270.
108. Asai T, Harada K, Kojima A, Sameshima T, Takahashi T, Akiba M, Nakazawa M, Izumiya H, Terajima J, Watanbe H.Phage type and antimicrobial susceptibility of *Salmonella enterica* serovar Enteritidis from food producing animals in Japan.National Institute of Animal Health. 2008:31: 555-559.
109. Akhtar F, Hussain I, Khan A, Rahman SU. Prevalence and antibiogram studies of *Salmonella* Enteritidisisolated from human and poultry sources. Pakistan Veterinary Journal. 2010:30: 25-28.
110. Fernandez EA, Calleja CA, Fernández CG, Capita R. Prevalence and antimicrobial resistance of *Salmonella* serotypes isolated from poultry in Spain. International Journal of Food Microbiology. 2006: 153: 281-287.
111. Fadlalla T, Mohamed E, Ahmed G, Mohamed T. Antimicrobial susceptibility of *Salmonella* serotypes isolated from human and animals in Sudan. Journal of Public Health and Epidemiology. 2012:4: 19-23.
112. Fasure AK, Deji-Agboola AM, Akinyemi KO. Antimicrobial resistance patterns and emerging fluoroquinolone resistant *Salmonella* isolates from poultry and asymptomatic poultry workers. African Journal of Microbiology Research. 2012:6: 2610-2615.
113. Abdellah C, Fouzia RF, Abdelkader C, Rachida SB, Mouloud Z. Prevalence and antimicrobial susceptibility of *Salmonella* isolates from chicken carcasses and giblets in Meknes, Morocco. African Journal of Microbiology Research. 2009:3: 215-219.
114. Centers for Disease Control and Prevention. *Salmonella* surveillance annual summary, 2003. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Atlanta.2004.
115. Samuel DA, Lorin DW, Martin W. Antimicrobial Resistance in Nontyphoidal *Salmonella.* Journal of Food Protection.2007:70(3):780–790.
116. Agodi A, Marranzano M, Jones CS, Threlfall EJ. Molecular characterization of trimethoprim resistance in *Salmonella* isolated in Sicily. Eur. J. Epidemiol. *1995:* 11:33–38.
117. Antunes P, Machado J, Sousa JC, Peixe L. 2005. Dissemination of sulfonamide resistance genes (*sul1, sul2,* and *sul3*) in Portuguese *Salmonella enterica* strains and relation with integrons. Antimicrob. Agents Chemother.2005: 49:836-839.
118. Bauernfeind A, Stemplinger I, Jungwirth R, Giamarellou H. Characterization of the plasmidic beta-lactamase CMY-2, which is responsible for cephamycin resistance. Antimicrob. Agents Chemother. *1996:*40:221-224.
119. Cloeckaert A, Baucheron S, Chaslus-Dancla E. 2001. Nonenzymatic chloramphenicol resistance mediated by IncC plasmid R55 is encoded by a *floR* gene variant. Antimicrob. Agents Chemother.2001: 45:2381-2382.
120. Doublet B, Carattoli A, Whichard JM, White DG, Baucheron S, Chaslus-Dancla E, Cloeckaert A. 2004. Plasmid-mediated florfenicol and ceftriaxone resistance encoded by the *floR* and *blaCMY-2* genes in *Salmonella enterica* serovars Typhimurium and Newport isolated in the United States. FEMS Microbiol. Lett. *2004:*233: 301-305.
121. Guerra B, Junker E, Helmuth R. Incidence of the recently described sulfonamide resistance gene *sul3* among German *Salmonella enterica* strains isolated from livestock and food. Antimicrob. Agents Chemother. *2004.*48:2712-2715.
122. Guerra B, Soto S, Helmuth R, Mendoza MC. Characterization of a self-transferable plasmid from *Salmonella enterica* serotype Typhimurium clinical isolates carrying two integron-borne gene cassettes together with virulence and drug resistance genes. Antimicrob. Agents Chemother. *2002:*46:2977–2981.
123. Pezzella C, Ricci A, DiGiannatale E, Luzzi I, Carattoli A. Tetracycline and streptomycin resistance genes, transposons, and plasmids in *Salmonella enterica* isolates from animals in Italy. Antimicrob. Agents Chemother. *2004:*48:903-908.
124. Shaw KJ, Rather PN, Hare RS, Miller GH. Molecular genetics of aminoglycoside resistance genes and familial relationships of the aminoglycoside modifying enzymes. Microbiol. Rev.*1993:* 57:138–163.
125. Mezgebu T, Mogessie A. Microbial load and incidence of *Salmonella* spp. in “kitfo” a traditional Ethiopian spiced, minced meat dish. Ethiopian Journal of Health Development. 1998:12: 135-140.

126. Anonymous. 2002. Preliminary FoodNet data on the incidence of foodborne illnesses—selected sites, United States, 2001. *Morb. Mortal. Wkly. Rep.* 51:325–329.
127. European Food Safety Authority. 2005. The community summary report on trends and sources of zoonoses, zoonotic agents and antimicrobial resistance in the European Union in 2004. European Food Safety Authority, Brussels.
128. Mead, P. S., L. Slutsker, V. Dietz, L. F. McCaig, J. S. Bresee, C. Shapiro, P. M. Griffin, and R. V. Tauxe. 1999. Food-related illness and death in the United States. *Emerg. Infect. Dis.* 5:607–625.
129. Nelson, J. D., H. Kusmiesz, L. H. Jackson, and E. Woodman. 1980. Treatment of *Salmonella* gastroenteritis with ampicillin, amoxicillin, or placebo. *Pediatrics* 65:1125–1130.
130. Winokur, P. L., A. Brueggemann, D. L. DeSalvo, L. Hoffmann, M. D. Apley, E. K. Uhlenhopp, M. A. Pfaller, and G. V. Doern. 2000. Animal and human multidrug-resistant, cephalosporin-resistant *Salmonella* isolates expressing a plasmid-mediated CMY-2 AmpC beta-lactamase. *Antimicrob. Agents Chemother.* 44:2777–2783.
131. Michael, G. B., P. Butaye, A. Cloeckaert, and S. Schwarz. 2006. Genes and mutations conferring antimicrobial resistance in *Salmonella*: an update. *Microbes Infect.* 8:1898–1914.
132. Denyer SP, Hodges NA, Gorman SP, Gilmore BF. Hugo and Russell s Pharmaceutical Microbiology (8th Edition). Wiley Blackwell Publishing House, New Delhi, India. 2011:200-229.
133. Willey J, Sherwood L, Wolverton C. Prescott s Microbiology. 9th Edition, McGraw-Hill, New York. 2013:51,191,377-400,840.
134. Zaki SA, Karande S. Multidrug-resistant typhoid fever: a review. The Journal of Infection in Developing Countries. 2011:5: 324-337.
135. Mohammad K, John AS, Michael JDLZ, Bijan K, Lourdes T, Vika B, Tam M, Kay G, Mansour S. Tracking the Sources of *Salmonella* in Ground Beef Produced from Non fed Cattle. Journal of Food Protection. 2012:75(8):1464–1468.
136. Bacon RT, Sofos JN, K. E. Belk KE, Hyatt DR, Smith GC. Prevalence and antibiotic susceptibility of Salmonella isolated from beef animal hides and carcasses. J. Food Prot. 2002:65:284–290.
137. Botteldoorn N, Heyndrickx M, Rijpens N, Grijspeerdt K, Herman L. *Salmonella* on pig carcasses: positive pigs and cross contamination in the slaughterhouse. J. Appl. Microbiol. 2003:95:891–903.
138. Murase T,Yamada M, MutoT, Matsushima A, Yamai S. Fecal excretion of Salmonella enterica serovar Typhimurium following a food-borne outbreak. J. Clin. Microbiol. 2000:38:3495–3497.