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ABSTRACT

Salmonellosis is caused by a family of bacteria that has thousands of members most of which can affect all species and which infected animals can eliminate in a period of weeks. The main member which occurs in cattle is *Salmonella typhimurium*, which can infect many species including man. *Salmonella* is an important bacteria that poses public health challenges worldwide, contributing to the economic problem of both developed and underdeveloped countries. A total of 131 blood and stool samples was collected from different cattle of various breeds and enrolled in this study including 54 (41.2%), male, 77 (58.8%) Female. Four breeds were identified in which white Fulani has the highest percentage. Crossbreed 10 (7.6%), Red Bororo 38 (29.0%), Sokoto Gudali 34 (26.0%), White Fulani 49 (37.4%). 101 (77.1%) were positive for Salmonella infections. The cross breed had the highest mean PCV value of (48.510) with lowest total protein mean value of (60.6680), Sokoto Gudali with the mean PCV value of (48.324) and Total protein of mean value of (70.4494), followed by Red Bororo with mean PCV value of (47.558) with highest Total protein mean value of (77.0274), White Fulani has the lowest PCV mean value of (44.051) with (67.2282) Total protein value. Further analysis was carried out to detect the effect of *Salmonella* on the different breeds. There was no statistically significant effect of Salmonella infection on the PCV and total protein content of the cattle serum tested in this study.

CHAPTER ONE

1.0 INTRODUCTION

There are several widely known diseases caused by Bacteria, Fungi, Viruses, and Parasites which can be found in cattle and can be transmitted to human. Disease of animal origin also known as Zoonosis can be defined as diseases transmitted between animals and humans through either direct or indirect contact, or through food (EFSA. 2016). Major Pathogens accountable for human diseases are of Animal origin (cattle, pigs, poultry), which can cause diseases such as Anthrax, *Samonellosis, Escherichia coli, Brucellosis,* Listeriosis, *Campylobacteriosis* (Karesh *et al.*,, 2012; Zhang *et al.*,, 2016). Based on research and 2017 report of the European Food Safety Authority and European Centre for Disease deterrence and control, it was recorded that the most common causes of Food-borne Zoonotic disease were *Campylobacter* and *Samonella* bacteria and most common parasite found in Ruminants include *Fasciola* and *Taenia* (EFSA. 2017). The word ruminant comes from a latin *ruminare*, which means "to chew over again". Ruminants are mammals with a specialized stomach by which they acquire nutrients from plant-based food by fermenting it before digestion (Foregut fermentation) through microbial activities. They have the ability to regurgitate food and chew again for further break down to aid digestion (rumination). Ruminant animals has been categorized to either domestic or wild species. The stomach of ruminants are divided into four compartments namely: Rumen, Reticulum, Omasum and Abomasum (Fernández. *et al* 2005). Organisms have the ability to co-exist in an environment. Likewise microorganisms such as bacteria, fungi, parasites and viruses can co-infect an animal. These complex relationships which involves the extrinsic factors e.g. nutrition, age, sex, heredities etc. and intrinsic factors such as reproductive state and environmental conditions.

Microorganisms can upturn their own chances of survival in an environment by modifying host immunity, which makes the host vulnerable to pathogenic infections, which often times leads to death of the animals. Most abattoirs lack proper sanitation system and friendly environment for the animals which can enhance vulnerability to infectious disease.

Salmonella infection is still a major public health challenge worldwide, contributing to the economic problem of both developed and underdeveloped countries (Crump *et al.,* 2004). *Salmonellosis* is a disease caused by a bacteria that affects the intestinal tract which belongs to the salmonella genus. *Salmonella* bacteria can be found in animal and human intestines and can be passed out mostly through feaces. Infection is usually spread by eating contaminated meat, eggs of milk. Symptoms mostly occur between 12 to 36 hours of exposure and may last from two to seven days (WHO, 2016). The bacteria has the tendency to remain in the gastrointestinal tract of the animal for periods between few months and a year. Symptoms may include diarrhea, anorexia, dehydration, low milk production and miscarriages. The economic importance of *Salmonellosis* in cattle expose producers to loss such as Mortality and weight loss, cost of medical care (Hoelzer, *et al.*, 2011).

Fasciolosis is a parasitic worm infection which is caused by common liver fluke (*Fasciola hepatica* and *Fasciola gigantic*). Both species are confined in the bile ducts of the liver or gall bladder. The disease main host is Ruminants such as cattle and sheep (which are considered the predominant animal reservoirs) but can as well affect humans. (Farrar *et al.,* 2013). The intermediate host of *F. hepatica* are fresh water snails that belongs to the family of Lymnaeidae (Torgerson., Claxton, 1999). In Ruminant animals such as cattle, sheep, goat, *Fasciolosis* results in great economic loss as a result of sudden death, weight loss as well as wool formation in the animals (Roseby, 1970). In cattle fasciolosis, in most cases, Adult cattle develop resistance to *F.*

hepatica infection while calves are susceptible to disease. Cattle *Fasciolosis* leads to damage of the livers and production loss due to weight loss by the animal (Phiri. *et al,* 2006). As a result of the impaired liver, the tissue may be infected with Bacteria such as *Clostridium novyi* type B. which leads to Black disease when Bacteria releases toxins into the bloodstream of the infected animal. These disease is found in an environment where there is high populations of Liver flukes and sheep (Merck veterinary Manual). Intravital diagnosis in animal is based on faeces analyses and immunological methods, the fluke eggs detectable in faeces 8- 12 weeks post infection. Particular antibodies in *F. hepatica* are considered using Western blot or Enzyme-linked immunosorbent assays (ELISAs) after 2- 4 weeks of post infection. This two method helps in early detection of the disease (Dumenigo *et al,* 2000). There are some bacterial that can be found in cattle which has the ability to co- infect the animal e.g. *Salmonella, Campylobacter, Listeria*.

Campylobacter is one of the bacteria which is responsible for animal disease. *Campylobacteriaceae* family is divided into four genera which are: *Campylobacter*, *Arcobacter*, *Dehalospirilum* and *Sulfurospirillum. Campylobacter* specie has been stated as most common foodborne bacterial zoonosis in the world, *campylobacteriosis* is majorly caused by the consumption of infected beef. Based on research it was recorded that 30 % of the infection was derived from consumption of poultry products (hens, turkey, ostriches and ducks). 20- 30% cases was derived from pathogens in cattle (Hald, *et al.,* 2016; Josefsen *et at*. 2015). Ruminant animals serves as a Reservoir for *campylobacter*, this Bacteria infect the alimentary canal of the cattle especially in the Gut, it can be found either in bristles or in lymphatic nodes and also on the hooves (Epps *et al.,* 2013). *Campylobacteriosis* does not just infect food of animal origin but it can also infect Vegetables which are the common vector of transmission through contacts (direct

or indirect) with animal faeces and use of contaminated irrigation water. *Listeria* is also one of the bacteria that can be transferred from animal to human.

Listeriosis is not a common disease but can cause havoc in the host because the disease is related with high mortality rate (Tahoun, *et al.,* 2017). *L. monocytogenes* are often found in the environment and isolated from the surface water, soil, sewage, faeces and agricultural vicinity. This specie of listeria is not defined because it depends on the host susceptibility and the virulence of a particular strain (Stea *et al.,* 2015). Pathogenic strain has the ability to infect and colonize ruminant and monogastric animals (sheep, cattle, goats, horses). And can be isolated from food products and vegetables of direct consumption (Mclntyre, *et al.,* 2015). Ruminants may be asymptomatic carriers of *L. monocytogenes.* Some of the symptoms of *Listeriosis* include articular pain, headaches, stomach disorder, vomiting, diarrhea, nausea and lack of appetite

1.1 JUSTIFICATION OF THE STUDY

Salmonella is one of the major pathogen associated with cattle. This microorganism may have an adverse effect on the quality of meat, milk and blood level. Which is of great economic importance (Crump, *et al*.,2004). This microbe causes morbidity and mortality worldwide both on animal and human; it has broad host ranges, the disease type differs in different host (thyphoid, enteric fever, bacteremia), and it has the ability to cause persistent infections if it is not properly treated; this microorganism is extremely resistant to many antibiotics drugs. Therefore, there is need for proper public health awareness on personal hygiene in order to reduce the risk of infection (Gillespie, *et al*.,2005).

1.2 AIM

The aim of the study is to determine the effect of *salmonella* infection on pack cell volume and Total protein level in different breeds of cattle.

1.3 OBJECTIVES OF THE STUDY

The specific objectives of this study are to:

1. Detect the presence of *Salmonella* and in the stool samples of cattle
2. Determine the pack cell volume of cattle of different breeds
3. Evaluate the total protein levels of cattle of different breeds
4. Estimate the effect of *Salmonella* on the pack cell volume and total protein levels of different breeds of cattle
5. Ascertain the possible role of *Salmonella* on meat quality

CHAPTER TWO

2.0 LITERATURE REVIEW

Salmonella infection is still a major public health challenge all over the world, which contributes to the economic problem of both developed and underdeveloped countries (Crump *et al.,* 2004). It affects the gastrointestinal tracts of mammals and also the blood of an infected animal or person and is capable of causing disease known as Bacteraemia and enteric fever (Majowicz *et al.,* 2010). *Salmonella* is one the most commonly isolated foodborne pathogens, and is principally found in cattle, dairy product, fresh fruits, vegetables, poultry and eggs (Silva *et al.,* 2011). The major propagation channel of the pathogen to an individual is through improper washing or uncooked animal food products. Most abattoirs lacks proper hygiene which can be associated to the slaughtering process of cattle, this can be measured as one of the vital sources of contamination with *Salmonella* (Gillespie *et al.,* 2005). It has been reported that *Salmonella spp*. are the source of over 90 million of diarrhea-related diseases per year in the world, and 85% of such cases was associated with food (Hung, *et al.,*2017). Children below the age of 4 years are often infected with serotypes Enteritis or Typhimurium (Evangelopoulou, *et al.,* 2015; De Jong, *et al* 2012). A drastic reduction has been observed concerning incidence of salmonellosis along the year as a result of Global monitoring. Thus, in year 2015 and 2016 there was an indication of slightly increased in the number of incidence.

Year	**Total number of confirmed cases**
2005	176,395
2008	131,468
2010	99,020

2012	91,034
2015	94,625
2016	94,530

(Evangelopoulou, *et al.* 2015; EFSA, 2006., EFSA, 2017).

Salmonella is Gram-negative bacteria which belong to the family *Enterobacteriaceae*. It has two main species which are *Salmonella enterica* and *Salmonella bongori*. *S enterica* specie is further divided into six subspecies (Su & Chiu, 2007). The subspecies comprises of over 2,600 serotypes (Gal-Mor, *et al.,* 2014). It has a rod shape known as bacillus, the rod shape is conserved as an outcome of the bacterial cytoskeleton made of an actin-resembling protein (Khan. 2014). Salmonella rods are able to grow at temperatures between 8 and 45℃ with an optimum temperature of 37℃ (Li, *et al.,* 2013). Recommended pH for the growth of *Salmonella* is "between" 4.0 to 9.5 with an optimum pH of 6.5 to7.0, and in an environments of low water activity of 0.94 (Rönnqvist, *et al.,* 2018). *Salmonella* species move with the aid of peritrichous flagella (all around the cell body), with cell diameters between 0.7 and 1.5 µm, lengths from 2 to 5 µm (Fabrega. 2013). They are chemotrophs, with the use of organic source, they can acquire their energy through the process of oxidation and reduction. They are comparatively anaerobic, non-sporulating bacteria, intracellular facultative pathogens (Jantsch, *et al.,* 2011). *Salmonella spp* may be identified based on biochemical tests. And also their ability to grow when citrate is used as sole of carbon source. According to biochemical tests, they are oxidase-negative and catalase-positive (Razzuoli, *et al.,* 2017; Andino & Hanning. 2015).

2.1 CLASSIFICATION AND NOMENCLATURE OF SALMONELLA

The nomenclature of the *Salmonella* genus is complex and erratic in the area of dividing this bacteria genus into species, subspecies, subgenera, groups, subgroups, and serotypes with the use of the standard Kauffman– White scheme about 2600 serotypes have been identified and most of these serotypes have the ability to adapt within a mammals (hosts) including human (Allerberger *et al.,* 2003).

Domain:	**Bacteria**
Phylum:	*Proteobacteria*
Class:	*Gammaproteobacteria*
Order:	*Enterobacteriales*
Family:	*Enterobacteriaceae*
Genus:	*Salmonella*

Subspecies of Salmonella enterica
Salmonella enterica subsp. Arizonae
Salmonella enterica subsp. Diarizonae
Salmonella enterica subsp. Enterica
Salmonella enterica subsp. Houtenae
Salmonella enterica subsp. Indica
Salmonella enterica subsp. Salamae

Source: National center for Biotechnology information. Retrieved 2019

The *Salmonella* genus is alienated into two species: *S. bongori* and *S. enterica*, based on genomic empathy and biochemical responses. Likewise, *Salmonella* genus is categorized into six subspecies (Brenner, *et al.,* 2000). Namely: *entrica* (serotype I) , *salamae (*II), *arizonae* (IIIa), *diarizonae* (IIIb), *houtenae* (IV), and *indica* (VI). (Foley, *et al.,* 2013). Most serotypes are classified as *S. enterica subsp*. 99% of *salmonellos*is outbreak in human and warm-blooded animals was caused by these species (Bugarel, *et al.,* 2017). Clinically, *salmonellae* has been categorized as Invasive (typhoidal) or non-invasive (non-typhoidal samonellae). According to host preference and disease manifestation in humans (Okoro, *et al.*,2012). However, nontyphoidal salmonella can be invasive and has the ability to cause paratyphoid fever, which needs an urgent and immediate treatment with antibiotics which can otherwise lead to death. While Typhoidal serotypes can only be transferred between humans and has the potential to cause food-borne infection, (typhoid and paratyphoid fever). Infants and young children are more prone to infection (Ryan & Ray. 2004).

In association with Phylogeny, standard Kauffman– White scheme *Salmonella* species are further classify by serotype based on some major antigenic determinants which include somatic (O), capsular (K) and flagella (H) (Brenner *et al.*, 2000). The heat-stable somatic O antigen is the oligosaccharide component of lipopolysaccharide suituated at the outer membrane of bacterial. A specific serotype of *Salmonella* express more than one O antigen on its surface (Hu & Kopecko 2003). The heat-labile H antigens that are responsible in the activation of host immune responses are located in the bacterial flagella. Majority of Salmonella spp. Possess two distinct genes that encode for the flagella proteins; these bacteria possess the unique ability of expressing only one protein at a time which is known as diphasic (phase I and II). All serotype expresses specific phase I H antigens which are accountable for its immunological identity,

whereas phase II antigens are non-specific antigens that can be shared by many serotypes (Quiston *et al.,* 2008).

2.2 PATHOGENESIS

The effects of *Salmonella* infections varies depending on the serotype involved and the health status of the human host. Children below the age of 5 years, elderly people and patients with immunosuppression are more prone to *Salmonella* infection than healthy individuals.

Salmonella species are facultative intracellular microorganism which can be transmitted to the host cell (Jantsch et *al* 2011).Due to ingestion of contaminated food or by human feces. *Salmonella* serotypes has been divided into two main groups which are typhoidal and nontyphoidal. Nontyphoidal serotypes are the most common ones and usually cause gastrointestinal disease. They are zoonotic in nature meaning they can be transferred between humans and other animals. Typhoidal serotypes include *Salmonella* Typhi and *Salmonella* Paratyphi A, (HansenWester *et al.,* 2002). Which are modified to humans (non-zoonotic). *Salmonella* are pathogenic microbe as they have the ability to invade, replicate and survive in host cells through phagocytosis (Takaya *et al.,* 2003). The ability of the bacteria to survive within macrophages enables them to be carried in the reticuloendothelial system. (Monack *et al.,* 2004).

2.3 NONTYPHOIDAL AND TYPHOIDAL SALMONELLA

This type of serotype are divided into two invasive and non-invasive. S. Typhi and S. Paratyphi are known as typhoid *Salmonella* (Connor & Schwartz 2005). Nontyphoidal serotypes of *Salmonella* is associated with food poisoning these Infection mostly occurs when a person ingests foods that is contaminated with bacteria. Susceptibility of the infection varies in host, Children below the age of 5 years, the route of transmission in infants can be through inhalation

of bacteria-laden dust. Elderly people and patients with immunosuppression are more prone to *Salmonella* infection than healthy individual (Thielman & Guerrant 2004). When the microbe gets to the gastrointestinal tract, some of the microorganisms are killed in the stomach as a result of gastric acidity, while the surviving ones enter the small intestine and multiply in tissues but *Salmonella* has developed a degree of tolerance to acidic environments that allows a division of ingested bacteria to survive (Garcia-del Portillo *et al.,* 1993). *Salmonella* resistance to most antibiotics makes its treatment difficult leading to high risk of bloodstream infections, myalgia, bradycardia, hepatomegaly (enlarged liver), splenomegaly (enlarged spleen), and rose spots on the chest and abdomen and increase the rate of hospitalization (Kuvandik *et al.,* 2009). In rural regions where infection is dominant, about 15% of the infected individuals develop gastrointestinal complications which consist of pancreatitis, hepatitis and cholecystitis, Haemorrhage, lymphatic nodule (Parry *et al.,* 2002).

2.4 EPIDEMIOLOGY

Salmonellosis has been public health problem over time and occur worldwide, which leads to high morbidity and mortality rate and take place mostly in underdeveloped countries (The incidence and mortality rate of enteric fever vary from region to region) (Hardy 2004). In 2000, the incidence of enteric fever was estimated to be 22 million cases causing 200,000 deaths worldwide, majorly in underdeveloped countries (Crump *et al.,* 2004). Enteric fever is endemic in many regions of the African, Asian continents, Europe, South and Central America, and the Middle East (Cooke *et al.,* 2007). In endemic regions, enteric fever occurs more often in children (0-5 years) Epidemiological studies for the past few years show that the annual incidence of enteric fever among children below 5 years old was roughly 25 per 100,000 population in China and Vietnam, while the incidence in India and Pakistan reached up to 450 per 100,000 annually

(Mweu & English 2008). Recent research shows that Out of a total of 168 isolates 55.4% were S. Typhi and 44.6% S. Paratyphi A. Most of the isolates, 92.9%, were from children aged 6–18 years and adult population.

2.5 TRANSMISSION OF SALMONELLA

There are several factors responsible for the virulence of *Salmonella* which involves four steps the I. adherence to host's cell, II. Invasion and replication inside host's cells, III. Polysaccharide coating and IV. Production of toxins (Figueiredo *et al* 2015). The adherence to the host cell is modulated by fimbriae, adhesins and flagella mobility of the cell may indirectly facilitate adhesion (Wiedemann.*et al.*,2015). After the pathogen penetrates the host's cells; it invade and replicate inside the cell (Sun. *et al.*, 2016). (Secretion system coding genes are localized on SPIs) (Ramos-Morales 2012). Effector proteins accountable for invasion and replication of Salmonella spp. affect the survival and stimulates production of pro-inflammatory cytokines which leads to the development of infection (Bierschenk, *et al* 2017). The surface part of the membrane bilayer of Gram-negative bacteria is composed majorly of lipopolysaccharides; Lipid A—the lipid part of the external lipopolysaccharide layer, causes various immunological responses of the host cell, which leads to manifestation of pro-inflammatory molecules or adhesion proteins (Chessa, *et al.*, 2014; Van Asten, 2005). The bacteria then Produces toxins which could either be endotoxins (lipid A) or exotoxins (cytotoxins and enterotoxins) (Van Asten, *et al.*, 2005).

2.6 CLINICAL MANIFESTATION OF SALMONELOSIS

Salmonella genus cause three types of *salmonellosis* in humans: noninvasive and nontyphoid, invasive and nontyphoid, and typhoid fever caused by the serotype S. typhi, as well as paratyphoid fever caused by two serotypes S. paratyphi A, B, and C (Kurtz, *et al.*, 2017). There are four different unique clinical manifestations associated with human infection which are

enteric fever, gastroenteritis, bacteraemia and other extraintestinal complications (Sheorey & Darby 2008). *Salmonella Typhi* is the causative agent of typhoid fever, while paratyphoid fever is caused by *S. Paratyphi* A, B and C. "enteric fever" is used collectively for both fevers, and both *S. Typhi* and *S. Paratyphi* are referred as typhoid *Salmonella* due to indistinct features between paratyphoid and typoid fever (Connor & Schwartz 2005). Enteric fever is categorized by an incubation period of one week or more, with prodomal symptoms such as headache, abdominal pain and diarrhea followed by the onset of fever (Bhan *et al.,* 2005). Diarrhoea is more commonly observed in children, while immuncompromise patients are more likely to develop constipation (Thielman & Guerrant 2004). enteric fever show a specific fever pattern, if not treated, it develops from a low-grade fever (> 37.5°C to 38.2°C) to high-grade fever (> 38.2°C to 41.5°C) in space of two weeks and can persist for months (Patel *et al.,* 2010). Gastroenteritis an inflammatory condition of the gastrointestinal tract known as Gastroenteritis or stomach flu which is go along with symptoms such as non-bloody diarrhoea, vomiting, nausea, headache, abdominal cramps and myalgias. Apart from hepatomegaly and splenomegaly which are less commonly observed in patients infected with NTS (Hohmann 2001). Compared to typhoid infections, Non Typhoidal *Salmonella* infections have a shorter incubation period (6–12 hours) and the symptoms are usually self-limiting and last only for 10 days or less (Crump *et al.,* 2008). Gastrointestinal complications of NTS infections include cholecystitis, pancreatitis and appendicitis, while the perforation of the terminal ileum has no association with NTS infections (Hohmann 2001). Infants, young children, elderly people and immunocompromised patients are highly susceptible to NTS infections and develop more severe symptoms than normal individuals (Scallan *et al.,* 2011). *Salmonella* bacteraemia is state whereby the bacteria penetrates the host cell and migrates into the bloodstream after invading the intestinal barrier. S. Dublin and S.

Cholearaesuis are two invasive strains that are highly associated with the manifestations of bacteraemia (Woods *et al.,* 2008). In severe circumstances, the immune response triggered by bacteraemia can lead to septic shock, with a high mortality rate. The clinical manifestation of bacteraemia is mostly seen in NTS infections than in typhoid *Salmonella* infections. It was stated that salmonella plasmid virulence gene in NTS is associated with difference in clinical manifestation which causes non- typhoidal bacteremia (Guiney & Fierer 2011). About 4-5% of patients with NTS develop bacteremia likewise extra-intestinal complications may occur include cellulitis, urinary tract infections, pneumonia, endocarditis and meningitis (Shimoni *et al.,* 1999). Effect of *salmonella* on pack cell volume and Total protein of a cattle is not really significant. The packed cell volume (PCV) is a dimension of the proportion of blood that is made up of cells. It is the ratio of the volume occupied by the red cells to the volume of whole blood in a sample of capillary, venous, or arterial blood- (Brian *et al* 2017*).* Direct measurement of the PCV may be done by centrifugation using a hematocrit centrifuge. The hematocrit is a blood test that measures the volume percentage of red blood cells (RBC) in blood. While serum total protein is a biochemical test for measuring the total amount of protein in serum which is made up of albumin and globulin. Bacterial coinfection in cattle may have the ability to reduce the blood level of a cattle. Due to inflammatory process, Salmonellosis leads to changes in serum concentrations of accurate phase proteins (Heinrich et al. 1990, Kent 1992). Since acute phase protein increase more rapidly after the onset of inflammation, in response to inflammatory cytokines, the identification of changes in serum concentrations of these proteins might be useful to detect the early stage of infection and to monitor the progression of s*almonellosis.*

CHAPTER THREE

3.0 MATERIAL AND METHODS

3.0.1 STUDY SITE

The samples for the study was obtained from cattle slaughtered at abattoir in Kara-Isheri Lagos Ibadan Expressway. Isheri Local Government Ogun state. The area is surrounded by ogun River which serves as a source of drinking water for cattle about to be slaughtered, the consumption of the meat products from the abattoir could pose a great health risk. It is also used for domestic activities by residents along its bank. Blood, feaces and other cattle wastes are flushed down to the river which could be life threatening if such water is consumed.

3.0.2 STUDY POPULATION

Great number of cattle are transported into Lagos for consumption from the Kara abattoir. Stratified random sampling technique was used to collect one hundred and thirty one(131) stool and blood samples from cattle of different breeds, sex, color and age (6 years and above). Types of breeds include Sokoto Gudali, Red Bororo, White Fulani and cross breeds (between Sokoto Gudali and White Fulani, Red Bororo and White Fulani). Samples were collected from different breeds of slaughtered Cattle at Abattoir, Kara- Isheri, Lagos- Ibadan Expressway, Ogun State. The blood samples were collected in both EDTA bottles and plain bottles. EDTA bottles contain anti-coagulants that prevent blood clothing. The stool samples were collected in universal bottles and the breed, sex and colour for every cattle was documented.

3.0.3 ETHICAL CONSIDERATION

Approval to carry out this study was obtained from the Veterinary Department of the Ministry of Agriculture, Ogun State with Reference Number VET. 956/15.

3.0.4 BLOOD SAMPLE COLLECTION AND ANALYSIS

The Blood samples were collected into the EDTA and plain bottles at point of slaughter, bottles were placed at the jugular veins of the cattle and the blood flew into the tubes, the EDTA bottles were rocked to ensure the anti-coagulant mixes with the blood to prevent clothing and plain. It was then transported to the Laboratory in ice packed containers and was processed immediately in the lab, the blood samples in the plain bottles were centrifuged at 3000rpm for 10 minutes for separation of the serum from red blood cell. The supernatant which is the serum appears at the top of the EDTA bottle while the whole blood (palette) settles at the bottom of the tube. The

serum was then siphoned into a cryogenic vial using a micropipette and stored immediately in a freezer. The blood in the EDTA bottles was used to determine the Pack Cell Volume.

3.0.5 PACK CELL VOLUME DETERMINATION

The packed cell volume (PCV) is a dimension of the proportion of blood that is made up of cells. It is the ratio of the volume occupied by the red cells to the volume of whole blood in a sample of capillary, venous, or arterial blood- (Brian *et al* 2017*).* Direct measurement of the PCV may be done by centrifugation using a hematocrit centrifuge. The hematocrit is a blood test that measures the volume percentage of red blood cells (RBC) in blood.

MATERIALS

Capillary tubes (75± 0.5mm in length, 1.155± 0.085mm in bore), Critoseal, Hematocrit Centrifuge**,** Hematocrit Reader.

PROCEDURES

The capillary tube is placed into the blood in the EDTA bottle and slanted to allow free flow of blood into the capillary tube it was then sealed with plasticin to prevent the blood from flowing out when centrifuging. The capillary tube is carefully placed in the hematocrit centrifuge and spin at 1300 rpm for 5minutes. After the separation, the packed red blood cells and the serum was measured using the hematocrit reader. To get the PCV, the volume of packed red blood cells was divided by the total volume of the blood sample.

3.0.6 TOTAL PROTEIN

Serum total protein is a biochemical test for measuring the total amount of protein in serum which is made up of albumin and globulin. The traditional method for measuring total protein uses the biuret reagent, but other chemical methods such as Kjeldahl method, dye- binding and refractometry are now available**.**

MATERIALS AND REAGENTS

Standard, Biuret reagent, Test tubes, Controls, Distilled Water, Pipettes, samples, Spectrophotometers, 3% NAOH, Normal Saline TS (Total Solids) Meter.

PROCEDURES;

1.0 ml of biuret reagent was siphoned into a test tube using a micro pipette, 20µl of standard was added across all test tubes, 20µl of distilled water was added to reagent 1 (biuret reagent) in a test tube for reagent blanking, Buiret reagent was added to 20µl of serum in the test tubes for sample reading. Serum and reagent blank were mixed and kept to incubate for 30 minutes at room temperature (20°C -25°C). After the incubation the reagent blank was used for blanking and samples were placed into a cuvette and the absorbance was then read at wavelength of Hg 546nm. Calculations were done to determine the total protein concentration.

3.1 SALMONELLA ISOLATION

3.1.1 Faecal Sample Collection and analysis

The stool samples were collected from the cattle at the point of slaughter into universal bottles, the universal bottles were labeled in accordance to the documented information on each cattle

breed. Each labeled universal bottle was placed at the anus of the cattle that passed stool during the slaughter process, the samples were conveyed in sealed packs from the abattoir to the Laboratory where the bacterial culture procedure and were carried out on each of the stool samples. Isolation of *Salmonella* from faeces can be achieved with the following materials and procedures.

3.1.2 MATERIALS:

Inoculation loops, Straight wire, Petri dishes, Test tubes, Sterile Universal Sample bottles, Plain Sample bottle, Bunsen burner, Incubators at 37 ^{o}C.

3.1.3 MEDIA AND REAGENT:

Selenite F broth, Rappaport Vassiliadis soy peptone (RVS) broth, Buffered peptone water, *Salmonella Shigella* Agar (SSA), TSI agar, Urea broth,

3.1.4 PROCEDURE

Pre-enrichment: 25g of faeces was inoculated onto 10% buffered peptone water and incubated for 24 hours at 37^{o}C (Non-selective enrichment).

Enrichment: 1 ml of pre-enriched sample was inoculated onto 10 ml of Selenite F broth and incubated for 24 hours at 37^{o}C (Selective enrichment)

Culture: A loop full inoculum of the enriched sample was further inoculated onto a *Salmonella Shigella* Agar plate and was incubated at 37oC for 24 hours. Colonies with black pigmentation was selected and purified on nutrient agar.

CHAPTER FOUR

4.0 RESULTS

Using stool culture technique all positive plates produced pinkish mucoid colonies with black centers on *Salmonella/Shigella* Agar. The colonies were large with smooth and perfect margin and circular. The biochemical tests shown the isolates to be Catalase positive, Oxidase negative, Urease negative, Starch negative, Glucose positive, MR positive, Citrate negative, Maltose positive and Indole negative. The Gram reaction and microscopy showed the isolates to be Gram negative rods with polar flagellation. A total of 131 samples was collected from different cattle of various breeds and was enrolled in this study including 54 (41.2%), male, 77 (58.8%) Female. Four breeds were identified in which white Fulani has the highest percentage. Crossbreed 10 (7.6%), Red Bororo 38 (29.0%), Sokoto Gudali 34 (26.0%), White Fulani 49 (37.4%). As shown in figure 4.1

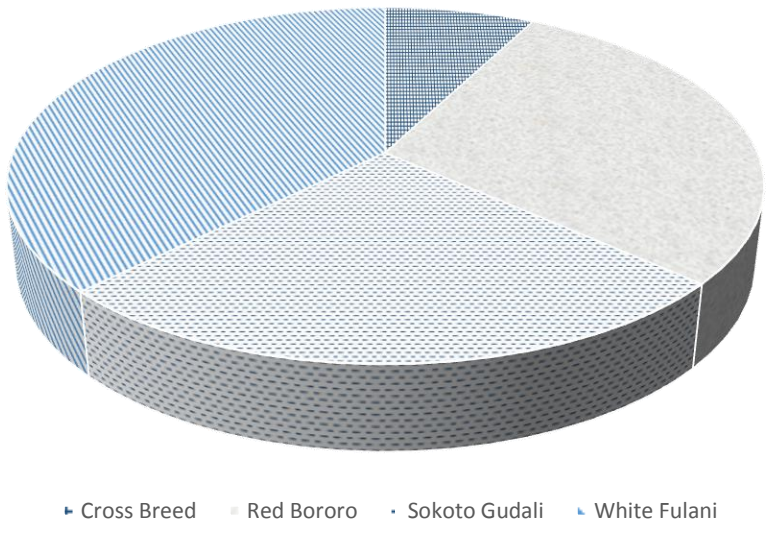

Figure 4.1: Distribution of the cattle breeds tested

In course of this study, colors of the cattle slaughtered were considered of which white cattle are of higher percentage and there percentage are as followed Black (7.6%), Black and white (3.8%), Brown (41.2%), Brown and white (2.3%) and white (45%). As shown in figure 4.2

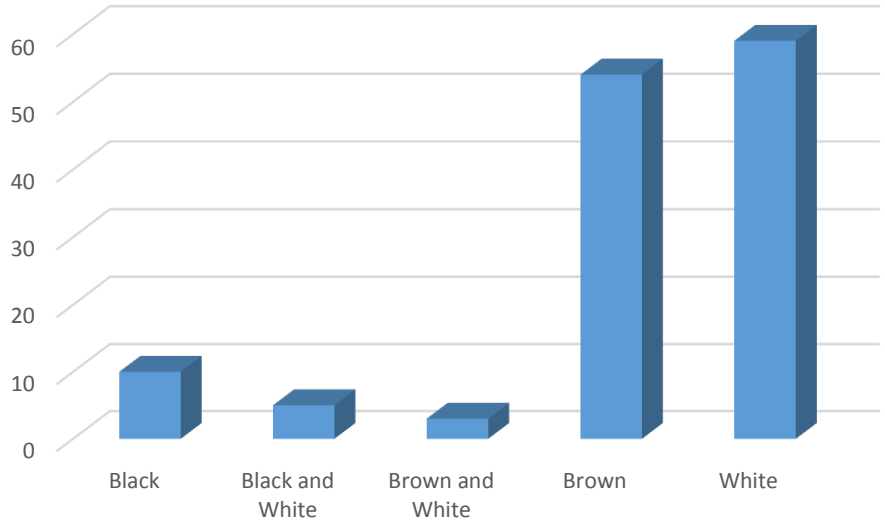

Figure 4.2: Colors of cattle tested

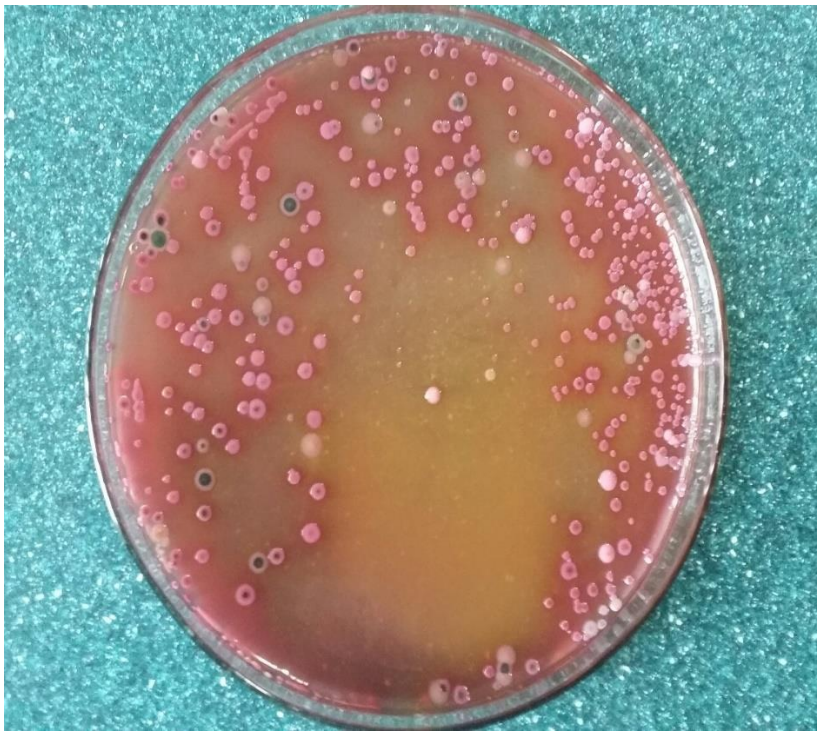

Figure 4.3: Salmonella colonies on SS Agar plate

4.0.1 EFFECTS OF SALMONELLA ON THE DIFFERENT BREEDS

4.0.2 Pattern of % Packed Cell Volume according to infection status

The pack cell volume among those infected was highest in the Sokoto Gudali breed (51.69± 9.98%). For those uninfected with *Salmonella,* the lowest PCV was found in white Fulani breed (44.25 ± 16.08%). However, the PCV was not significantly different among those infected and uninfected in Red Bororo (P=0.284), Sokoto Gudali (P=0.259), White Fulani (P=0.895). Analysis was not done for Cross breed due to low sample size. As shown in figure 4.4

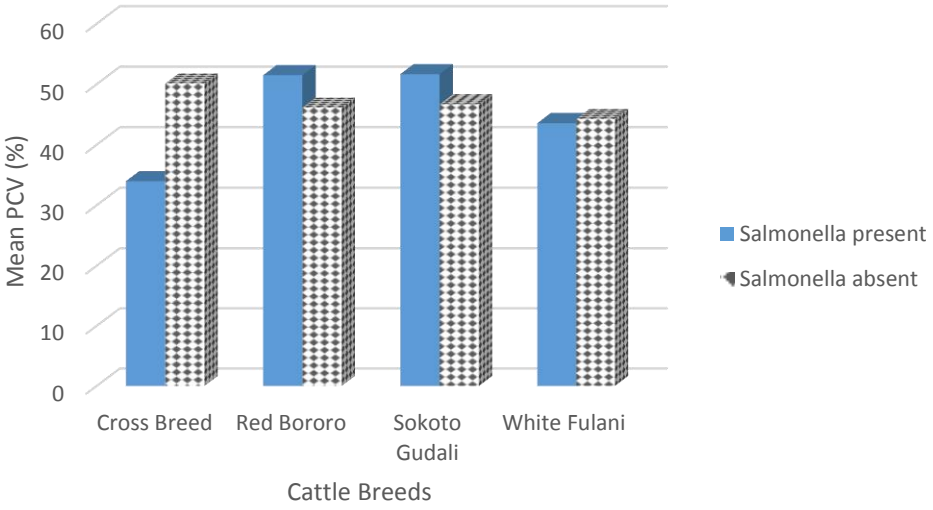

Figure 4.4: Distribution of Salmonella infection and mean PCV among cattle breeds

Pattern of % Total serum protein according to infection status

The Total protein among those infected was highest in the Red Bororo breed (85.887± 53.49%). For those uninfected with *Salmonella,* the lowest total protein was found in white Cross breed (63.378 ± 31.50%). However, the total protein was not significantly different among those infected and uninfected in Red Bororo (P=0.427), Sokoto Gudali (P=0.840), White Fulani (P=0.916). Analysis was not done for Cross breed due to low sample size. As shown in figure 4.5

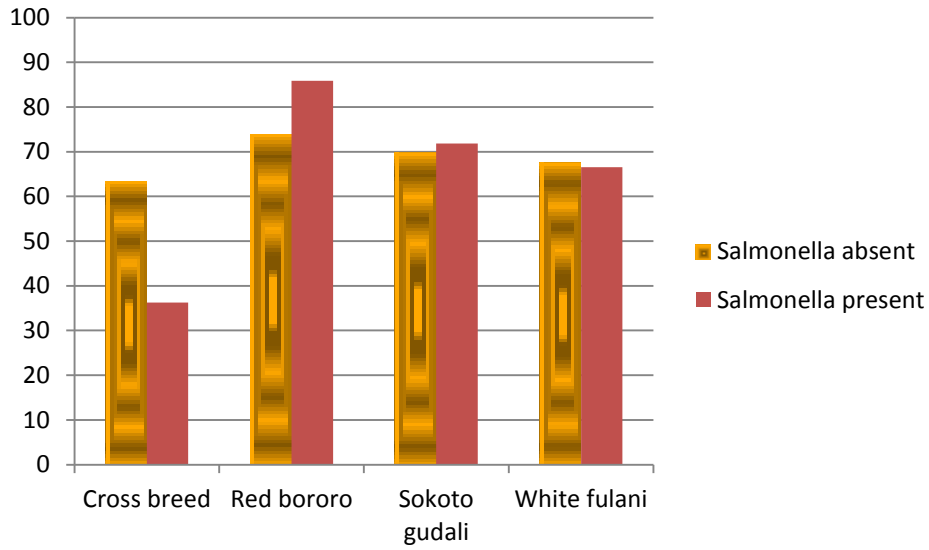

Figure 4.5: Distribution of Salmonella and mean total protein of different cattle breeds

CHAPTER FIVE

5.0 DISCUSSION

This study was carried out to detect the prevalence of *Salmonella* among cattle of different breeds in Kara- Isheri abattoir ogun state, and to determine the effect of the infection on their Pack cell volume and total protein. The prevalence of 101 (75.6%) positive samples were observed among 131 cattles. In the course of this study, presence of *Salmonella* did not have a statistically significant implication on the pack cell volume (PCV) and total protein of the different breeds. Further analysis was not conducted for cross breed due to low sample size. However, cross breed had the highest mean PCV value of (48.510) with lowest total protein mean value of (60.6680), Sokoto Gudali with the mean PCV value of (48.324) and Total protein of mean value of (70.4494), followed by Red Bororo with mean PCV value of (47.558) with highest Total protein mean value of (77.0274), White Fulani has the lowest PCV mean value of (44.051) with (67.2282) Total protein value. Further analysis was carried out to detect the effect of *Salmonella* on the different breeds as shown in the Appendix.

These results are slightly different from the study carried out in Adamawa state between four breeds of cattle in which the PCV for white Fulani was (6.6091), Red bororo (6.6423), Sokoto gudali (6.6908) and Adamawa gudali (6.6210). It is also somewhat different from the study carried out by Mirzadeh *et al.* (2012) who reported a mean PCV of (28.45%) for semental cattle in Iran. However, our cattle breeds were different. The differences in these studies may be as a result of several factors like geographical location of the study, environmental factors, and seasonal change in time of collection, nutritional factors and cattle hydration status.

5.1 CONCLUSION

Salmonellosis still remain a public health concern worldwide. The genetic composition of the Salmonella strains and its ability to resist most antimicrobials increases the difficulty in eliminating the bacteria. The prevalence of Salmonella infection in cattle at Kara- isheri abattoir ogun state Nigeria is high (75.6%). This situation calls for urgent control measures on the part of individual to practice absolute hygiene by cooking meat properly and proper processing of meat product. Although, the presence of salmonella in the cattles that was tested positive has no significant effect on the PCV and Total protein of different breeds. Those in living in the area are of high risk and can be infected through their water source because the cattle wastes before and after slaughter are flushed into the Ogun River. However, several preventive measures have been proposed to stop the widespread of infection but still the infection can be prevented.

5.2 RECOMMENDATION

The following recommendation can assist in control of widespread of infection to human:

1) Research on salmonellosis should be encouraged
2) There is need for multidimensional diagnostic approach in Salmonellosis clinical condition as a measure to avoid the widespread of the health threatening infection by employing effective treatment
3) Government should create a better channel where the cattle waste can be flushed instead of launching it into the ogun river
4) Emphasizing on the importance of proper hygiene and adequate cooking and processing of meat.

5) Public health awareness by educating the inhabitant of Kara- Isheri Ogun state Nigeria to pay close attention to their water source and treat their water before consumption.

REFERENCE

Allerberger, F., Liesegang, A., Grif, K., Khaschabi, D., Prager, R., Danzl, J., Hock, F., Ottl, J., Dierich, M.P., Berghold, C. (2003). "Occurrence of Salmonella enterica serovar Dublin in Austria". Wiener medizinische Wochenschrift. 153:148–152.

Andino, A. and Hanning, I. (2015). "*Salmonella enterica*: Survival, colonization, and virulence Differences among Serovars". *Sci. World J.*2015, 520179. [CrossRef] [PubMed].

Bhan, M.K., Bahl, R. and Bhatnagar, S. (2005). 'Typhoid and paratyphoid fever". *Lancet*. 366(2):749–762.

Bierschenk, D., Boucher, D. and Schroder, K. (2017). "Salmonella-induced inflammasome activation in humans". *Mol. Immunol*. 86, 38–43. [CrossRef] [PubMed].

Brenner, F.W., Villar, R.G., Angulo, F.J., Tauxe, R. and Swaminathan, B. (2000). "Salmonella nomenclature". *J Clin Microbiol*. 38:2465– 2467.

Brenner,F.W.,Villar,R.G.,Angulo,F.J.,Tauxe,R..Swaminathan,B.(2000)."Salmonella Nomenclature". *J.Clin. Microbiol.* 38, 2465–2467. [PubMed].

Bugarel, M., Tudor, A., Loneragan, G.H. and Nightingale, K.K. (2017). "Molecular detection assay of five Salmonella serotypes of public interest: Typhimurium, Enteritidis, Newport, Heidelberg, and Hadar" *J. Microbiol .Methods* 134, 14–20. [CrossRef] [PubMed].

Brian, S., Bull, M.D., John, A., Koepke, M.D., Elkin Simson, M.B., Onno, W., Van Assendelft, M.D.(2017). "Procedure for determining packed cell volume by the Microhematocrite method". Volume 20 Number 18

Centas, L. and Suer, K. (2014).Review: "The Important Bacterial Zoonoses in one health concept. *Front.publicHealth*.2014;2:144.doi:10.3389/fpubh.2014.00144.[PMCfree article] [PubMed] [CrossRef].

Chessa, D., Spiga, L., De Riu, N., Delaconi, P., Mazzarello, V., Ganau, G. and Rubino, S. (2014). "Lipopolysaccharides Belonging to Different Salmonella Serovars Are Differentially Capable of Activating Toll-Like Receptor 4". *Infect. Immun*. 82, 4553–4562. [CrossRef] [PubMed].

Chikeka, I., Dumler. J.S. (2015). "Neglected Bacterial Zoonoses".*Clin.Microbiol.Infect*.2015; 21:404-415.doi:10.1016/j.cmi.2015.04.022. [PMC free article] [PubMed] [CrossRef].

Cooke, F.J, Day, M., Wain, J., Ward, L.R. and Threlfall, E.J. (2007). "Cases of typhoid fever imported into England, Scotland and Wales (2000–2003)". Transactions of the Royal Society of Tropical Medicine and Hygiene. 101:398–404.

Crump, J. A., Luby, S. P., and Mintz, E. D. (2004). "The global burden of typhoid fever". Bull. W.HO. 82:346–353.

Crump, J.A, Kretsinger, K., Gay, K., Hoekstra, R.M., Vugia, D.J., Hurd, S., Segler, S.D., Megginson, M., Luedeman, L.J., Shiferaw, B., *et al.*, (2008). "Clinical response and outcome of infection with Salmonella enterica serotype Typhi with decreased susceptibility to fluoroquinolones: a United States foodnet multicenter retrospective cohort study". *Antimicrob. Agents Chemother*. 52:1278–1284.

Crump, J.A., Kretsinger, K., Gay, K., Hoekstra, R.M., Vugia, D.J., Hurd, S., Segler, S.D., Megginson, M., Luedeman, L.J., Shiferaw, B. *et al.*, (2008). "Clinical response and outcome of

infection with Salmonella enterica serotype Typhi with decreased susceptibility to fluoroquinolones: a United States foodnet multicenter retrospective cohort study". *Antimicrob*. Agents Chemother. 52:1278–1284.

Connor, B.A., Schwartz, E. (2005). "Typhoid & Paratyphoid fever in travelers". *Lancet infect Dis*.2005.Oct. 5(10):623-8.Doi:10.1016/s1473-3099(05)70239-5.PMID:16183516.

De Jong, H.K., Parry, C.M., van der Poll, T. and Wiersinga, W.J. (2012). "Host–Pathogen Interaction in Invasive Salmonellosis". *PLoS Pathog*. **2012**, 8, 1–9. [CrossRef] [PubMed].

Dumenigo, B.E., Epsino, A.M., Finlay, C.M. and Mezo, M. (2000). "Kinetics of antibody- based antigen detection in serum and feaces of sheep experimentally infected with fasciola hepatica". *Vet. Parasitol*. 89 (1-2): 153-61.doi:10.1016/S0304-4017(00) 00206-5. PMID 10729655.

Epps, S.V.R., Harvey, R.B., Hume, M.E., Phillips, T.D., Anderson, R.C. and Nisbet, D.J. (2013). "Foodborne Campylobacter: Infections, Metabolism, Pathogenesis and Reservoirs". *Int. J. Environ. Res. Public Health".* 2013, 10, 6292- 6304. [CrossRef] [PubMed].

European food safety Authority (EFSA) European centre for Disease prevention and control (ECDC) The European Union summary report on trends and sources of zoonoses, zoonotic agents and food borne outbreak in 2015. EFSA J.2016;14:e04634.doi:10.2903/*j.efsa*.2016.4634. [CrossRef].

European food safety Authority (EFSA) European centre for Disease prevention and control (ECDC) The European Union summary report on trends and sources of zoonoses, zoonotic agents and food borne outbreaks in 2016.*EFSA J*.2017,e5077.

European Food Safety Authority (EFSA); (2006). "European Centre for Disease Prevention and Control (ECDC). The Community Summary Report on Trends and Sources of Zoonoses, Zoonotic Agents, Antimicrobial Resistance and Foodborne Outbreaks in the European Union in 2005". *EFSA J.* **2006**, 4, 94. [CrossRef].

European Food Safety Authority (EFSA); (2017). "European Centre for Disease Prevention and Control (ECDC). The European Union summary report on trends and sources of zoonoses, zoonotic agents and food-borne outbreaks in 2016". *EFSA J.* **2017**, 15, e5077. [CrossRef].

Evangelopoulou, G., Kritas, S., Christodoulopoulos, G. and Burriel, A.R. (2015) "The commercial impact of pig Salmonella spp. infections in border-free markets during an economic recession". *Vet. World* **2015**, 8, 257–272. [CrossRef] [PubMed].

Fabrega, A. and Vila J. (2013). "Salmonella enterica serovar Typhimurium skills to succeed in the host: virulence and regulation". *Clinical microbiology Reviews*. 26(2):308-41.doi:10.1128/CMR.00066-12.PMC 3623383.PMID 23554419.

Fabrega, A. and Vila, J. (2013). "*Salmonella enteric* Serovar Typhimurium skills to succeed in the Host: virulence Regulation*". Clin. Microbial*. Rev. 2013,26,308-341. [CrossRef] [PubMed].

Farrar., Jeremy., Hotez., Peter., Junghanss., Thomas., Kang, Gagandeep., Lalloo, David., White. And Nicholas, J. (2013). *Manson's Tropical Diseases.* Elsevier Health sciences. ISBN 9780702053061.

Fernández, Manuel Hernández; Vrba, Elisabeth S. (2005). "A complete estimate of the phylogenetic relationships in Ruminantia: a dated species-level super tree of the extant

ruminants". Biological Reviews. **80** (2): 269–302. doi:10.1017/s1464793104006670. ISSN 1469-185X.

Figueiredo, R., Card, R., Nunes, C., AbuOun, M., Bagnall, M.C., Nunez, J., Mendonça, N., Anjum, M.F. and da Silva, G.J. (2015). "Virulence Characterization of Salmonella enterica by a New Microarray: Detection and Evaluation of the Cytolethal Distending Toxin Gene Activity in the Unusual Host S. Typhimurium". *PLoS* ONE, 10, e0135010. [CrossRef] [PubMed]

Foley, S.L., Johnson, T.J., Ricke, S.C., Nayak, R. and Danzeisen, J. (2013). "Salmonella Pathogenicity and Host Adaptation in Chicken-Associated Serovars". *Microbiol. Mol. Biol.* Rev. 2013, 77, 582–607. [CrossRef] [PubMed].

Gal-Mor, O., Boyle, E.C. and Grassl, G.A. (2014). "Same species, different diseases: how and why typhoidal and non- typhoidal *Salmonella enterica* serovars differs". *Frontiers in microbiology.* 5:391. doi:10.3389/fmicb.2014.00391. PMC 4120697. PMID 25136336.

Garcia-del Portillo, F., Foster, J.W and Finlay, B.B. (1993). "Role of acid tolerance response genes in Salmonella typhimurium virulence". *Infection and Immunity*. 61 (10): 4489–92. PMC 281185 PMID 8406841 (https://www.ncbi.nlm.nih.gov/pubmed/8406841).

GBD 2015. Mortality and causes of death, collaborators.(2016) "Global regional and national life expectancy, all- cause mortality and cause specific mortality for 249 causes of death,1980-2015: a systematic analysis for the global Burden of Disease study 2015". *Lancet.* 388(10053): 1459-1544. doi: 10.1016/s0140-6736(16)31012-1. PMC 5388903. PMID 27733281.

Gillespie, I.A., O'Brien, S.J., Adak, G.K., Ward, L.R. and Smith, H.R. (2005). "Foodborne general outbreaks of Salmonella Enteritidis phage type 4 infection", England and Wales, 1992–2002: where are the risks? Epidemiology and Infection. 133: 759–801.

Guiney, D.G. and Fierer, J. (2011). "The role of the spv genes in Salmonella pathogenesis". *Frontiers in Microbiology*, 2: 129.

Hald, B., Skov, M.N., Nielsen, E.M., Rahbek, C., Madsen, J.J., Waino, M., Chriel, M., Nordentoft, S., Baggesen, D.L. and Madsen, M. (2016)."*Campylobacter jejuni* and *campylobacter coli* in wild birds on Danish livestock farms. *Acta Vet. Scand*.2016, 58,11. [CrossRef] [PubMed].

Hansen-Wester, I., Stecher, B. and Hensel M. (2002). "Type III secretion of Salmonella enterica serovar Typhimurium translocated effectors and SseFG". *Infect Immun*. 70:1403–1409.

Hardy, A. (2004). "Salmonella: a continuing problem". Postgraduate *Medical Journal*. 80:541–545.

Hoelzer, K., Moreno, Switt, A.I. and Wiedmann, M. (2011). "Animal contact as a source of human Non- typhoidal salmonellosis. *Vet. Res*. 2011, 42, 34. [CrossRef] [PubMed].

Hohmann, E.L. (2001). "Nontyphoidal salmonellosis". *Clinical Infectious Disease,* 15(32):263–269.

Heinrich, P.C., Castell, J.V. & Andus T. (1990). "Interleukin-6 and the acute phase response". *Biochem. J*. 265:621-636.

Hu L, D.J. and Kopecko. (2003). "Typhoid Salmonella". In: M.D; Millotis and J.W; Bier. Editor. International handbook of foodborne pathogens. New York: Marcel Dekker, Inc; p. 151–165.

Hung, Y.T., Lay, C.J., Wang, C.L. and Koo, M. (2017). "Characteristics of nontyphoidal gastroenteritis in Taiwanese children": A 9-year period retrospective medical record review. *J. Infect. Public Health* **2017**, 10, 518–521. [CrossRef] [PubMed].

Jantsch, J., Chikkaballi, D. and Hensel, M. (2011). "Cellular aspects of immunity to intracellular Salmonella enterica". *Immunological Reviews.* 240(1):185-95.doi:10.1111/j.1600-065X.2010.00981.x.PMID 21349094.

Josefsen, M.H., Bhunia, A.K., Olsson Engvall, E., Fachmann, M.S.R. and Hoorfar, J. (2015). "Monitoring in the poultry production chain- from culture to genes and beyond". *J. Microbiol. Methods* 2015, 112, 118- 125. [CrossRef] [PubMed].

Karesh, W.B., Dobson, A., Lloyd-Smith, J.O., Lubroth, J., Dixon, M.A., Bennett, M., Aldrich, S., Harrington, T., Formenty, P., Loh, E.H., *et al.*, (2012). "Ecology of Zoonoses: Natural and unnatural histories".*Lancet*.2012;380:1936-1945.doi:10.1016/S014-6736(12)61678-X. [PubMed] [CrossRef].

Kent, J. (1992). "Acute phase proteins: Their use in veterinary diagnosis". *Brit. Vet. J*. 148: 279-282.

Khan, C.M.A. (2014). " The Dynamic Interactions between *Salmonella* and the Microbiota, within the Challenging Niche of the Gastrointestinal Tract" . *Int. Sch. Res. Not*. **2014**, *2014*, 846049. [CrossRef] [PubMed].

Kurtz, J.R., Goggins, J.A. and McLachlan, J.B. (2017). "Salmonella infection: Interplay between the bacteria and host immune system". *Immunol. Lett*. 190, 42–50. [CrossRef] [PubMed].

Kuvandik, C., Karaoglan, I., Namiduru, M. and Baydar, I. (2009). “Predictive value of clinical and laboratory findings in the diagnosis of the enteric fever”. The New *Microbiologica*. 32: 25–30.

Li, H.P., Wang, H., D’Aoust, J.Y. and Maurer, J. (2013). “ Food Microbiology”: *Fundamentals and Frontiers*, 4th ed.; *Salmonella species*; Doyle, M.P., Buchanan, R., Eds.; ASM Press: Washington, DC, USA, 2013; pp. 225–261, ISBN 9781555818463, 9781628702255.

Majowicz, S.E., Musto, J., Scallan, E., Angulo, F.J., Kirk, M., O’Brien, S.J., Jones, T.F., Fazil, A. and Hoekstra, R.M. (2010). “The global burden of nontyphoidal Salmonella gastroenteritis. Clinical Infectious Diseases”. 50(6):882–889.

Mas-Coma, S., Bergues, M. D., and Esteban, J. G. (1999). “Human Fasciolosis”. p. 411–434. In J. P. Dalton (ed.), Fasciolosis. CAB International, Wallingford, United Kingdom.

Merck Veterinary Manual – infection Necrotic Hepatitis.

Monack, D.M., Mueller, A. and Falkow, S. (2004). “Persistent bacterial infections: the interface of the pathogen and the host immune system”. Nat Rev *Microbiol*. 2:747–765.

Mweu E. and English, M. (2008). “Typhoid fever in children in Africa”. Tropical medicine & International Health. 13:532–540.

Okoro, C.K., Kingsley, R.A., Connor, T.R., Harris, S.R., Parry, C.M., Al-Mashhadani, M.N., Kariuki, S., Msefula, C.L., Gordon, M.A., de Pinna, E., Wain, J., Heyderman, R.S., Obaro, S., Alonso, P.L., Mandomando, I., MacLennan, C.A., Tapia, M.D., Levine, M.M., Tennant, S.M., Parkhill, J. and Dougan, G. (2012). "Intracontinental spread of human invasive Salmonella Typhimurium pathovariants in sub-Saharan Africa". Nature Genetics. 44 (11): 1215–21.

doi:10.1038/ng.2423. PMC 3491877 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3491877). PMID 23023330 (https://www.ncbi.nlm.nih.gov/pubmed/23023330).

Parry, C.M., Hien, T.T., Dougan, G., White, N.J. and Farrar, J.J. (2002). "Typhoid fever". The New England Journal of Medicine. 347:1770–1782.

Patel, T.A., Armstrong, M., Morris-Jones, S.D., Wright, S.G., Doherty, T. (2010). "Imported enteric fever: case series from the hospital for tropical diseases, London, United Kingdom". The American Journal of Tropical Medicine and Hygiene. 82:1121– 1126.

Phiri, I.K., Phiri, A.M. and Harrison, L.J. (2006). "Serum antibody isotype responses of Fasciola-infected sheep and cattleto excretory and secretory products of fasciola species". *Vet.Parasitol*.141 (3-4):234-42.doi:10.1016/j.vetpar.2006.05.019.PMID 16797844.

Ramos-Morales, F. (2012). "Impact of Salmonella enterica Type III Secretion System Effectors on the Eukaryotic Host Cell". ISRN *Cell Biol*. 2012, 787934. [CrossRef].

Razzuoli, E., Amadori, M., Lazzara, F., Bilato, D., Ferraris, M., Vito, G. and Ferrari, A. (2017). "Salmonella serovar-specific interaction with jejunal epithelial cells". *Vet. Microbiol*. 207, 219–225. [CrossRef] [PubMed].

Rönnqvist, M.; Valttila, V.; Ranta, J.; Tuominen, P. (2018). "Salmonella risk to consumers via pork is related to the Salmonella prevalence in pig feed". *Food Microbiol*. 2018, 71, 93–97. [CrossRef] [PubMed].

Roseby, F.B. (1970). "The effect of Fasciolosis on the wool production of Merino sheep". Aust. *Vet. J*. 46(8): 361- 365. doi:10.1111/j.1751-0813.1970.tb15573.x.PMID 5471269.

Ryan, I. K.J., Ray. and C.G, eds. (2004). *Sherris Medical Microbiology* (4th ed.). McGraw Hill. pp. 362–8. ISBN 978-0-8385-8529-0.

Salmonella(https://www.ncbi.nlm.nih.gov/Taxonomy/Browser/wwwtax.cgi?mode=Tree&id=590&lvl=3&keep=1&srchmode=1&unlock) (HTML). *NCBI* taxonomy. Bethesda, MD: National Center for Biotechnology Information. Retrieved 27 January 2019.

Scallan, E., Hoekstra, R.M., Angulo, F.J., Tauxe, R.V., Widdowson, M.A., Roy, S.L., Jones, J.L. and Griffin, P.M. (2011). "Foodborne illness acquired in the United States – major pathogens". Emerging Infectious Diseases. 17:7–15.

Sheorey, H. and Darby, J. (2008). "Searching for Salmonella". Australian Family Physician. 37:806–810.

Shimoni, Z., Pitlik, S., Leibovici, L., Samra, Z., Konigsberger, H., Drucker, M., Agmon, V., Ashkenazi, S. and Weinberger, M. (1999). "Nontyphoid Salmonella bacteremia: age-related differences in clinical presentation, bacteriology, and outcome". *Clinical Infectious Diseases,* 28:822–827.

Silva, J., Leite, D., Fernandes, M., Mena, C., Gibbs, P.A.and Teixeira, P. (2011). "Campylobacter spp. as a foodborne pathogen". A review. Frontiers in Microbiology. 2.

Sinclair, K.B. (1962). "Observations on the clinical pathology of Ovine Fasciolosis". *Brit. Vet. J.* 118:37-53.

Stea, E.C., Purdue, L.M., Jamieson, R.C.., Yosk, C.K., and Truelstrup Hansen, L. (2015). "Comparison of the prevalences and diversities of Listeria species and Listeria monocytogenes in

an Urban and a Rural Agricultural watershed". *Appl. Environ. Microbiol.*2015,81, 3812- 3822. [CrossRef] [PubMed].

Su, L.H. and Chiu, C.H. (2007). "Salmonella: clinical importance and evolution of nomenclature". *Chang Gung medical journal.* 30 (3): 210-9. PMID 17760271.

Sun, H., Kamanova, J., Lara-Tejero, M. and Galán, J.E. (2016). "A Family of Salmonella Type III Secretion Effector Proteins Selectively Targets the NF-κB Signaling Pathway to Preserve Host Homeostasis". *PLoS Pathog*. 12, 1–19. [CrossRef] [PubMed].

Tahoun, A.B.M.B., Abou Elez, R.M.M., Abdelfatah, E.N., Elsohaby, I., El-Gedawy, A.A. and Elmoslemany, A.M. (2017). "*Listeria monocytogenes* in raw milk, milking equipment and dairy Workers": Molecular characterization and antimicrobial resistance patterns. *J. Glob. Antimicrob. Resist.* 2017, 10, 264-270. [CrossRef] [PubMed].

Takaya, A., Suzuki, M., Matsui, H., Tomoyasu, T., Sashinami, H., Nakane, A. and Yamamoto T. (2003). "Lon, a stress-induced ATP dependent protease, is critically important for systemic Salmonella enterica serovar typhimurium infection of mice". *Infect Immun*. 71:690–696.

Thielman, N.M. and Guerrant, R.L. (2004). "Acute infectious diarrhea. The New England Journal of Medicine". 350:38–47.

Thielman, N.M., Guerrant, R.L. (2004). "Acute infiectious diarrhea". The New England Journal of Medicine. 350:38–47.

Torgerson, P. and Claxton, J. (1999). "Epidemiology and control". In Dalton, J.P. *Fasciolosis.* Wallingford, Oxon, UK:CABI pub.pp.113-49.ISBN 0-85199-260-9.

Tortora, G.A. (2008). *Microbiology: An introduction* (9th ed.). Pearson. Pp.323- 324. ISBN 978-8131722329.

Van Asten, A.J., A.M. and van Dijk, J.E. (2005). "Distribution of "classic" virulence factors among Salmonella spp". FEMS *Immunol.* Med. *Microbiol*. 44, 251–259. [CrossRef] [PubMed].

Wiedemann, A., Virlogeux-Payant, I., Chaussé, A.-M., Schikora, A. and Velge, P. (2015). "Interactions of Salmonella with animals and plants". *Front. Microbiol*. 2015, 5, 791. [CrossRef] [PubMed].

Woods, D.F., Reen, F.J., Gilroy, D., Buckley, J., Frye, J.G. and Boyd, E.F. (2008). "Rapid multiplex PCR and real-time TaqMan PCR assays for detection of Salmonella enterica and the highly virulent serovars Choleraesuis and Paratyphi C". *J Clin Microbiol*. 46:4018–4022.

Zhang, H.L., Mnzava, K.W., Mitchell, S.T., Melubo, M.L., Kibona, T.J., Cleaveland, S., Kazwala, R.R., Crump, J.A., Sharp, J.P. and Halliday, J.E.B.(2016) "Mixed methods Survey of Zoonotic Disease Awareness and Practice among Animal and Human Healthcare providers in Moshi, Tanzania". *PLos Negl.* Trop.Dis.2016;10: e0004476 doi:10.1371/journal.pntd.0004.476. [PMC free article] [PubMed] [CrossRef].

APPENDIX

Frequency distribution

SEX OF CATTLE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	F	54	41.2	41.2	41.2
	M	77	58.8	58.8	100.0
	Total	131	100.0	100.0	

BREED

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	C.B	10	7.6	7.6	7.6
	R.B	38	29.0	29.0	36.6
	S.G	34	26.0	26.0	62.6
	W.F	49	37.4	37.4	100.0
	Total	131	100.0	100.0	

COLOR

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Black	10	7.6	7.6	7.6
	Black an	2	1.5	1.5	9.2
	Brown	54	41.2	41.2	50.4
	Brown &	2	1.5	1.5	51.9
	Brown& W	1	.8	.8	52.7
	White	59	45.0	45.0	97.7
	White &	3	2.3	2.3	100.0
	Total	131	100.0	100.0	

Mean Values and T-test (without considering infection status)

Group Statistics

	Breed	N	Mean	Std. Deviation	Std. Error Mean
PCV	R.B	38	47.558	13.3743	2.1696
	S.G	34	48.324	11.8678	2.0353
Total Protein	R.B	38	77.0274	40.40495	6.55455
	S.G	34	70.4494	26.42229	4.53139

PCV is not significantly different between the two groups, p=0.799

Protein is not significantly different between the two groups, p=0.422

Group Statistics

	Breed	N	Mean	Std. Deviation	Std. Error Mean
PCV	R.B	38	47.558	13.3743	2.1696
	W.F	49	44.051	15.8240	2.2606
Total Protein	R.B	38	77.0274	40.40495	6.55455
	W.F	49	67.2282	28.78359	4.11194

PCV is not significantly different between the two groups, p=0.276

Protein is not significantly different between the two groups, p=0.190

Group Statistics

	Breed	N	Mean	Std. Deviation	Std. Error Mean
PCV	R.B	38	47.558	13.3743	2.1696
	C.B	10	48.510	12.2179	3.8636
Total Protein	R.B	38	77.0274	40.40495	6.55455
	C.B	10	60.6680	30.91428	9.77595

PCV is not significantly different between the two groups, p=0.840

Protein is not significantly different between the two groups, p=0.241

Group Statistics

	Breed	N	Mean	Std. Deviation	Std. Error Mean
PCV	S.G	34	48.324	11.8678	2.0353
	W.F	49	44.051	15.8240	2.2606
Total Protein	S.G	34	70.4494	26.42229	4.53139
	W.F	49	67.2282	28.78359	4.11194

PCV is not significantly different between the two groups, p=0.186

Protein is not significantly different between the two groups, p=0.606

Group Statistics

	Breed	N	Mean	Std. Deviation	Std. Error Mean
PCV	S.G	34	48.324	11.8678	2.0353
	C.B	10	48.510	12.2179	3.8636
Total Protein	S.G	34	70.4494	26.42229	4.53139
	C.B	10	60.6680	30.91428	9.77595

PCV is not significantly different between the two groups, p=0.966

Protein is not significantly different between the two groups, p=0.328

Group Statistics

	Breed	N	Mean	Std. Deviation	Std. Error Mean
PCV	W.F	49	44.051	15.8240	2.2606
	C.B	10	48.510	12.2179	3.8636
Total Protein	W.F	49	67.2282	28.78359	4.11194
	C.B	10	60.6680	30.91428	9.77595

PCV is not significantly different between the two groups, p=0.405

Protein is not significantly different between the two groups, p=0.519

EFFECTS OF SALMONELLA ON DIFFERENT BREEDS

Group Statistics

Breed		Sal	N	Mean	Std. Deviation	Std. Error Mean
C.B	PCV	A	9	50.122	11.7769	3.9256
		P	1	34.000	.	.
	Total Protein	A	9	63.3778	31.50471	10.50157
		P	1	36.2800	.	.

R.B	PCV	A	28	46.150	13.8490	2.6172
		P	10	51.500	11.6852	3.6952
	Total Protein	A	28	73.8632	35.27037	6.66547
		P	10	85.8870	53.49091	16.91531
S.G	PCV	A	23	46.713	12.5540	2.6177
		P	11	51.691	9.9812	3.0095
	Total Protein	A	23	69.8030	23.99035	5.00233
		P	11	71.8009	32.16679	9.69865
W.F	PCV	A	34	44.253	16.0755	2.7569
		P	15	43.593	15.7814	4.0747
	Total Protein	A	34	67.5200	31.09405	5.33258
		P	15	66.5667	23.68333	6.11501

Breed	**Parameters**	**P-value**
CB	PCV	
	Total Protein	
RB	PCV	0.284
	Total Protein	0.427
SG	PCV	0.259
	Total Protein	0.840
WF	PCV	0.895
	Total Protein	0.916

Presence of *Salmonella* did not have a significant implication on the PCV and Total protein of the different cattle breeds. Analysis was not conducted for CB due to the low sample size.

Table 4.1: Biochemical analysis for detection of *Salmonella*

Catalase	**Citrate**	**Glucose test**	**Maltose test**	**Gram stain**	**MR**	**Oxidase**	**Urease**	**Suspected Organism**
†	—	†	†	—	†	—	—	*Salmonella*
†	—	—	—	—	†	—	—	*Shigella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	—	—	—	†	—	—	*Shigella*
†	—	—	—	—	†	—	—	*Shigella*
†	—	—	—	—	†	—	—	*Shigella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	*NIL*

†	—	—	—	—	†	—	—	*Shigella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
Nil	Nil	Nil	†	—	†	—	—	*NIL*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	*NIL*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	—	—	—	†	—	—	*Shigella*
NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	*NIL*
NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	*NIL*
†	—	—	—	—	†	—	—	*Shigella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
Nil	Nil	Nil	NIL	NIL	NIL	NIL	NIL	*NIL*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*

†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
Catalase	**Citrate**	**Glucose test**	**Maltose test**	**Gram stain**	**MR**	**Oxidase**	**Urease**	**Suspected Organism**
NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	*NIL*
Nil	Nil	Nil	NIL	NIL	NIL	NIL	NIL	*NIL*
NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	*NIL*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	—	—	—	†	—	—	*Shigella*
†	—	—	—	—	†	—	—	*Shigella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
Nil	Nil	Nil	NIL	NIL	NIL	NIL	NIL	*NIL*

†	—	—	—	—	†	—	—	*Shigella*
—	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
—	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
Nil	Nil	Nil	NIL	NIL	NIL	NIL	NIL	*NIL*
Nil	Nil	Nil	NIL	NIL	NIL	NIL	NIL	*NIL*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
Nil	Nil	Nil	NIL	NIL	NIL	NIL	NIL	*NIL*
—	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
Nil	Nil	Nil	NIL	NIL	NIL	NIL	NIL	*NIL*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
Nil	Nil	Nil	NIL	NIL	NIL	NIL	NIL	*NIL*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*

Nil	Nil	Nil	NIL	NIL	NIL	NIL	NIL	*NIL*
Nil	Nil	Nil	NIL	NIL	NIL	NIL	NIL	*NIL*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
Nil	Nil	Nil	NIL	NIL	NIL	NIL	NIL	*NIL*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*

Catalase	**Citrate**	**Glucose test**	**Maltose test**	**Gram stain**	**MR**	**Oxidase**	**Urease**	**Suspected Organism**
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
Nil	Nil	Nil	NIL	NIL	NIL	NIL	NIL	*NIL*
Nil	Nil	Nil	†	NIL	NIL	NIL	NIL	*NIL*

†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	—	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
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†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*
†	—	†	†	—	†	—	—	*Salmonella*