PREVALENCE OF DIARRHOEA AND SOME OF THE CAUSES IN DOGS IN ZARIA AREA

BY

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D E C L A R A T I O N

The work presented in this thesis is original and carried out under the supervision of Drs. S. U. Abdullahi, E. O. Gyang and A. A. Adesiyun in the Small Animal Clinic of the Veterinary Teaching Hospital and the laboratory of the Department of Veterinary Public Health and Preventive Medicine, Ahmadu Bello University, Zaria.

The work of other investigators was referred to and acknowledged. No part of this thesis has previously been submitted for a degree or diploma.

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C E R T I F I C A T I O N

This Thesis entitled "Prevalence of Diarrhoea and some of the Causes in Dogs in Zaria Area" by A. R. Trimnell meets the regulations governing the award of the degree of Master of Science of Ahmadu Bello University, Zaria, and is approved for its contribution to scientific knowledge and literary presentation.

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D E D I C A T I O N

This work is dedicated to my wonderful and considerate parents, Mr. John Nino Lazzari and Mrs. Rosealine Ladi Lazzari, husband, Mr. Celestine Trimnell, daughter, Anne-Marie, other members of my family and friends for all their love and moral support.

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ABSTRACT

In an attempt to determine the epidemiology of diarrhoea in dogs of Zaria area, a two-year retrospective and an 18-month clinical studies were conducted. In a two-year study (January, 1984 to December, 1985), case records of 1,876 dogs that attended the Ahmadu Bello University, Veterinary Teaching Hospital, Zaria were evaluated for diarrhoea. The prevalence of diarrhoea was 6.72%. The incidence of diarrhoea in the dogs was highest in the month of March. Occurrence of diarrhoea in young dogs of 1-6 months old (16.10%) was statistically significantly different ($p<0.05$, x^2) from other age groups. The incidence of diarrhoea was higher in the Nigerian local breed of dogs (7.94%) and Alsatians (7.71%) when compared to the crossbreeds (2.96%); this difference was statistically significant ($p<0.05$, x^2). There was no significant difference in the incidence of diarrhoea between male (6.67%) and female (6.7%) dogs.

A total of 155 household dogs were surveyed during the 18-month-period of study (March, 1986 to August, 1987) of which 83 were diarrhoeic and 72 were apparently healthy dogs. The incidence of diarrhoea in the dogs was highest in the month of

March. Occurrence of diarrhoea in young dogs of 1-6 months old (66.3%) was highly significant ($p<0.001$) and was more common in Nigerian local breed of dogs (62.7%) when compared to Alsatians (28.9%) and crossbreeds (8.4%); this was statistically significant ($P<0.001$). The occurrence of diarrhoea was more common in male dogs (56.6%) than in females (43.4%) but this difference was not statistically significant ($P>0.05$). Majority of the dogs, both diarrhoeic and apparently healthy, had combined intestinal bacterial and parasitic infections; coccidiosis occurred alone in two diarrhoeic cases (2.4%) and Escherichia coli alone was the predominant bacterium in diarrhoeic (51.8%) and apparently healthy (41.7%) dogs. Bacterial isolation was higher in diarrhoeic dogs (83.1%) and was statistically significant ($P<0.05$).

Frequency of isolation of E. coli was 69 (83.1%) in diarrhoeic dogs and 53 (73.6%) in apparently healthy dogs; this difference was not statistically significant ($P>0.05$). There was a higher isolation rate from young diarrhoeic dogs of 1-6 months old (66.7%) than in older dogs of greater than 36 months (14.5%). This age effect was statistically significant ($P<0.001$). Of the 49 E. coli isolates serotyped, 26 were from diarrhoeic

dogs and 10 from apparently healthy dogs, of the 26 strains, a total of 25 different serotypes were obtained. Ten of the 20 E. coli strains from diarrhoeic dogs were positive for the invasive test and negative for enterotoxin production (both heat-labile (LT) and heat-stable (ST)). No significant statistical difference was observed between the heat-labile (LT) and heat-stable (ST) enterotoxin production by the E. coli ($P < 0.05$) in diarrhoeic and apparently healthy dogs even though LT enterotoxin production in diarrhoeic dogs was slightly higher (means, LT = 16.65, and ST = 12.25). Several of the E. coli isolated were resistant to common antibiotics but the majority of them were sensitive to gentamicin.

From the total dogs sampled, only one Yersinia enterocolitica (serogroup O:8) was isolated from an apparently healthy dog. This isolate was negative for heat stable (ST) enterotoxin, calcium dependency (37 °C), invasiveness and autoagglutination. It was highly susceptible to gentamicin, tetracycline and neomycin but resistant to erythromycin and streptomycin.

Salmonella isolation from diarrhoeic dogs was more frequently done in younger dogs of 1-6 months old (88.9%) than in older dogs of greater than 36

months (11.1%), while inapparent infections were more common in older dogs (60%); this age distribution was statistically significant ($P<0.001$). The prevalence of Salmonella spp. was 9.6%.

The Salmonella spp. isolated were from rectum (11), beddings (1), mesenteric lymph node (1) and liver (2). Of the nine isolates obtained from diarrhoeic dogs, serotypes S. schwarzengrund, S. hull, S. colindale, S. tabligo, S. poona and S. stanleyville were found. The five isolates from apparently healthy dogs included serotypes S. schwarzengrund, S. koketime, S. omifisan, S. whilhemsburg and S. rubislaw. Two dogs had more than one serotype.

Several antimicrobial agents were effective in preventing the growth of Salmonella spp. in vitro which included gentamicin, tetracycline, ampicillin and neomycin. Resistance was found to at least five out of the 11 antimicrobial agents which included chloramphenicol, erythromycin, sulphadiazine, lincomycin and nalidixic acid.

Salmonella infection in apparently healthy dogs was statistically ($P<0.05$) dependent on season, with an increased occurrence in the wet season (April to September).

Of the helminths detected from both diarrhoeic (25.3%) and apparently healthy dogs (29.2%), Ancylostoma spp. and Taenia spp. were the most common. Nine diarrhoeic dogs and 2 apparently healthy dogs were infected with Isospora spp. The oocyst count for the diarrhoeic dogs was from ++ (i.e. 3-5 oocyst per field of microscope) and for apparently healthy dogs from + (i.e. 1 oocyst per field of microscope). The predominant helminth counts for Ancylostoma spp. and Toxocara spp. in diarrhoeic dogs ranged from ++ to +++ and + to ++ in apparently healthy dogs. Taenia spp. had the same level of infection for both diarrhoeic and apparently healthy dogs. Spirocerca spp. and Trichuris spp. had a low count of +, each occurring in a diarrhoeic dog only. Dipylidium spp. was detected in an apparently healthy dog (++).

The total combined prevalence of helminth and enteric protozoal infection was 34.2%, 34.9% in diarrhoeic dogs and 33.3% in apparently healthy dogs. The frequency of isolation of ova and oocysts in the faeces of both diarrhoeic and apparently healthy dogs was highly statistically ($P<0.001$) dependent on age group, particularly in young dogs of 1-6 months old and also in dogs older than 36 months.

Although it was not possible to categorically incriminate particular agents as the causes of diarrhoea in this study, the agents were encountered in both diarrhoeic and non-diarrhoeic dogs, it is reasonable to conclude that E. coli and Salmonella spp. were responsible for causing diarrhoea in young dogs.

The prevalence of diarrhoea in both studies was similar. However, local dogs had higher prevalence of diarrhoea than Alsatians in the clinical study when compared to the retrospective study.

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Chapter 1

DIARRHOEA AND ITS CAUSES IN DOGS: A REVIEW OF THE LITERATURE

1.1 Introduction

Diarrhoea is an alteration in the normal pattern of defaecation that usually results in the increased frequency, volume and consistency of the faeces excreted. It is not a diagnosis, but a clinical sign of an underlying disease process or disorder (Strombeck, 1979). Numerous enteric diseases or conditions result in diarrhoea with the major consequences as dehydration, acidosis, impaired growth, or even death. It is the most consistent sign indicating problems in the small and large intestines. Other changes often seen include the presence of blood, mucus, undigested and unabsorbed nutrients and melena. Colonic and rectal problems leading to diarrhoea are usually associated with tenesmus or dyschezia, and increased frequency of defaecation with small volume of faeces. Gas production and accumulation may occur with diarrhoea leading to abdominal pain or discomfort, borborgymus eructation, flatus and pain. Halitosis or an offensive flatus or faecal odour is common.

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Defaecation frequency is usually increased when there are larger volumes of faeces and this is due to a greater content of faecal water which could result from greater amounts of undigested or unabsorbed nutrients and/or increased fibre in the diet. Large faecal volume can also result from small or large intestinal problem leading to hypersecretion or a failure of fluid absorption respectively. Diarrhoea could also occur due to inflammatory processes affecting intestinal motility and osmotic factors due to unabsorbed particles that could result from over-eating, pancreatic exocrine deficiency, brush-border disaccharidase deficiency, bile acid deficiency and the ingestion of excessive amounts of poorly absorbed ions (e.g. phosphates found in all meat diets) (Phillips, 1972; Anderson, 1975; Strombeck, 1979).

Diarrhoea could occur with infections that have a viremic, bacteremic or septicemic phase (e.g. canine distemper, canine parvoviral enteritis, infectious canine hepatitis, leptospirosis and bacterial diseases of the neo-natal puppy and kitten) (Murdoch, 1986).

Diarrhoea may be acute or chronic. It is one of the most common problems clinicians often have to treat. With chronic diarrhoea the signs are of

lesser intensity and it would occur over a period of time and may be intermittent (Strombeck, 1979; Murdoch, 1986).

1.2 Mechanisms of Diarrhoea

Diarrhoea is known to be produced by five basic mechanisms and successful understanding, diagnosis, treatment and management of cases would depend on understanding these mechanisms. The five basic mechanisms include intestinal motility disorders, hypersecretion, osmotic factors, malabsorption ard increased intestinal mucosal permeability (Strombeck, 1979; Murdoch, 1986).

1.2.1 Intestinal Motility Disorders

Increased intensity, frequency, or rate of progression of peristalsis would accelerate the transit of ingesta and alimentary secretions through the intestine. If this transit is so rapid that mucosal contact time for digestion and absorption is inadequate, diarrhoea would occur. However, there is no evidence to indicate that hypermotility is the primary functional derangement that leads to diarrhoea in any disease. Thus, other mechanisms would have to be considered as well when attempting

to understand and manage diarrhoeal diseases (Strombeck, 1979). Also, motility is probably decreased in some diarrhoeal diseases and decreased motility may predispose to the proliferation of intestinal bacteria in others.

Increased intestinal fluid caused by a primary mucosal derangement could also secondarily stimulate increased peristalsis. The enterotoxin that causes diarrhoea in human cholera also stimulated propulsive muscular contractions in rabbit small intestine (Mathias et al., 1976). This stimulation was independent of fluid accumulation in the intestinal lumen. Thus, hypermotility may contribute to diarrhoea in cholera. It is suspected that Escherichia coli enterotoxins in colibacillosis may have a similar effect (Strombeck, 1979), but it is most likely that hypersecretion is more significant in such diarrhoea. Salmonella species and invasive strains of E. coli can also cause hypermotility (Anderson, 1975).

Murdoch (1986) described two distinct patterns of peristalsis in the small intestine: (a) the 'segmental' peristaltic waves which are the primary intestinal movements, are associated with mixing and absorption from chyle, and (b) secondary 'propulsive'

peristaltic waves which cause mass movements along the alimentary tract. Hypermotility causes increased segmentation resulting in a net increase in total transit time and increased water absorption whereas hypomotility decreases segmentation leading to osmotic diarrhœa via reduction in water and particle absorption with retention of chyme in the terminal ileum. The most common etiology of diarrhoa in the dog and cat is a flaccid, hypomotile bowel with secondary peristaltic wave 'rushes' (Murdoch, 1986).

Increased transit rate leading to diarrhoea could be caused by: 1) Osmotic factors including, nutritional intestinal overload, malabsorption, impaired digestion, mucosal damage, enzyme deficiencies (e.g. lactose) and intramural disease; 2) hypersecretion due to bacterial endotoxins, hydroxylated bile salts, mucosal damage from viruses, bacteria or parasites, and 3) increased intestinal permeability due to lymphangiectasia, mucosal damage or protein-losing enteropathy. Reduced transit rate resulting in diarrhœa could be caused by a low fibre diet and partial obstruction in the intestine (Stromback, 1979; Murdoch, 1986).

1.2.2 Hypersecretion

This is defined as a net intestinal efflux of fluid and electrolytes, occurring independent of changes in permeability, absorptive capacity, or exogenously generated osmotic gradients. Hypersecretion is thought to result from an exaggeration of the normal secretory component of intestinal fluid and electrolyte transport which involves flux from blood to intestinal lumen (Moon, 1979).

The mechanism of intestinal secretion is not completely understood and these present concepts may change with time as research in the area progresses. The secretory and absorptive capabilities of the mucosa vary from crypt to villous (Moon, 1979).

The young, immature, or undifferentiated epithelial cells of the crypt lack digestive and absorptive enzymes. Thus, although the absorptive capacity of the crypts is minimal, they appear to be the principal site of intestinal secretions (Bellamy et al., 1973; Nasset and Ju, 1973). These undifferentiated epithelial cells also proliferate and produce a continuous supply of new epithelial cells, which then migrate out to the villi where they differentiate to mature villous epithelial cells,

losing their proliferative and some secretory capacities. These cells carry out the majority of digestive and absorptive functions of the intestines. Normally, villous absorption exceeds crypt secretion and the result is net absorption (Moon, 1979).

Enterotoxins produced by E. coli, Clostridium perfringes, Yersinia enterocolitica and some Salmonella species cause diarrhoeal diseases by inducing intestinal hypersecretion (Banwell and Sheer, 1973; Strombeck, 1979). In this case, the villi remain intact along with their digestive and absorptive capacities. The crypts also remain intact but their secretions are apparently increased beyond the absorptive capacity of the intestines resulting in diarrhoea (Koupal and Deibel, 1975; Sack et al., 1976; Pai and Mors, 1978; Moon, 1979).

Hypersecretion probably occurs in the crypts and it is thought to be mediated by increased cellular cyclic adenosine monophosphate (cAMP) (Murdoch, 1986) and the mucosal cAMP level is in turn probably regulated by hormones (Schwartz et al., 1974). In colibacillosis, the enterotoxin produced by E. coli is thought to act like a hormone which increases mucosal cAMP, therefore exaggerating intestinal secretion. This mode of hypersecretion by the crypts

may account for the diarrhoea observed in other diseases like human cholera (toxin), human pancreatic cholera (endocrine) and Salmonella infection (toxin) (Field, 1974; Giannella et al., 1975). The mechanism has not been well documented in the dog and cat (Murdoch, 1986).

Experimentally, it has been shown that the integrity of the mucosal structure and the resultant secreted fluid (that is, isotonic, electrolyte rich, alkaline and free of exudates) are diagnostically useful in enterotoxic colibacillosis (Moon et al., 1966). Also, the diagnosis of hypersecretion would lead to a therapeutic principle which has been applied successfully in some secretory diarrhoeal diseases of human beings and calves (Pierce and Hirschhorn, 1977). This principle involves exploiting the intact absorptive capacity of the mucosa (Whipp and Moon, 1973) by the oral administration of a fluid containing glucose, amino acids and sodium. Glucose and amino acid absorption would enhance the absorption of sodium and water, replacing fluid and electrolyte losses. But the underlying enterotoxin-induced abnormality would remain (Moon, 1979).

1.2.3 Osmotic Factors

If for any reason, there is a problem with the digestion and absorption of a dietary component ingested, then it would result in osmotically active particles entering the distal ileum and proximal colon with the following consequencies: (a) attraction of water into the lumen of the intestine; (b) fermentation of these particles by intestinal bacteria with the production of organic acids, ammonia, hydrogen sulphide, amines and bacterial toxins; (c) poorly-absorbed fatty acids become hydroxylated by intestinal bacteria resulting in compounds which reduce colonic water absorption and stimulate the secretion of fluids into the colonic lumen leading to osmotic diarrhoea (Murdoch, 1986).

Problems that could result in this kind of diarrhoea include nutritional intestinal over-load, malabsorption, impaired digestion (due to exocrine pancreatic insufficiency and bile salt deficiency), mucosal damage (caused by inflammation, neoplasia, gluten-enteropathy in dogs), enzyme deficiencies (lactase), intramural disease (lymphosarcoma), lymphangiectasia, chronic cellular infiltration (eosinophils, lymphocytes, plasma cells), amyloidosis,

and regional enteritis (Anderson, 1975; Strombeck, 1979; Murdoch, 1986).

1.2.4 Malabsorption

Malabsorption is a term used to characterise syndromes in which diarrhoea occurs either primarily due to digestive failure, or secondarily due to intestinal abnormalities resulting from defects in mucosal transport systems, reduced mucosal surface area, reduced brush-border enzymes, diseases involving the lamina propria and lymphatic drainage problems (Strombeck, 1979) and exocrine pancreatic insufficiency and bile salt deficiency (Murdoch, 1986). The ingesta is not absorbed leading to diarrhoea.

The virus causing transmissible gastroenteritis (TGE) in pigs multiplies selectively in villous absorptive cells and destroys them resulting in villous atrophy and impaired absorptive capacity. This marked loss of absorptive cells, surface area and enzymes lead to a generalised decrease in absorptive capacity. Intestinal absorption of fat, glucose, sodium ion and chlortetracycline has been shown to be impaired in pigs with TGE (McClung et al., 1976). Thus, the ingesta within the small intestine is poorly absorbed and passed on to the

colon. When the amount of ingesta exceeds the absorptive capacity of the colon, diarrhoea results (Moon, 1979).

This pathogenic sequence also occurs in several other epitheliotropic virus infections of the small intestine, which include rotavirus infections (Mebus et al., 1977), parvovirus infections (Kent and Moon, 1973) where the parvovirus has a predilection for immature proliferative crypt epithelium which it destroys without directly affecting absorptive cells. With time, the resultant aplastic crypts fail to replace the villous absorptive cells as they are shed and villous atrophy ensues leading to mucosal collapse. The major consequence is malabsorption (Moon, 1979). However, maldigestion, fermentation and secretion probably all complicate the process too.

The underlying pathogenic mechanisms in diarrhoea due to malabsorption have implications for diagnosis, treatment and recovery of the patient. For example, starvation with continued access to water resulted in cessation of diarrhoea (Hooper and Halterman, 1966). Apparently, the residual absorptive capacity adequately handles endogenous secretions when maldigestive fermentative components of the disease are removed. This restricted diet and feeding

of hypotonic solutions, or parenteral administration of fluids would be expected to be beneficial to the patient (Jaecker, 1973).

1.2.5 Increased Intestinal Mucosal Permeability

There are continuous fluxes of water and electrolytes across the intestinal mucosa - secretory fluxes (movement from blood to gut), as well as absorptive fluxes (movement from gut to blood) occur simultaneously. In a clinically normal animal, the absorptive fluxes exceed secretory fluxes resulting in net absorption. However, the secretory flux across the normal mucosa is substantial and the absorptive load presented to the intestine from endogenous secretions greatly exceeds that from ingested fluids. The flux in both directions occurs mainly via passive diffusion which takes place through microscopic pores in the junctions between the epithelial cells. Normally, the net result is to maintain an equilibrium or slight net absorption. very little of this flux requires active transport by and through the epithelial cells. If total secretion increases beyond the absorptive capacity, or if absorption is impaired, net secretion results and the excess would be expelled, resulting in diarrhoea (Moon, 1979).

Inflammation in the intestines could be accompanied by increased hydraulic pressures and increased 'pore size' in the mucous membranes, permitting increased flow throu h the membranes down the pressure gradient from blood to the intestinal lumen. When the amount of material exuded exceeds the absorptive capacity of the intestines, diarrhoea results (Moon, 1979). In contrast to exudation, permeability could be decreased in some diarrhoeal diseases (Fordtran et al., 1967). Such a decrease could be expected to reduce diffusion in both directions. This would lead to diarrhoea if the epithelium were presented with more material than it could absorb by active transport (Moon, 1979).

The size of material that would pass through the mucosa would vary depending on the magnitude of the increase in pore size. Small increases, resulting in pores that are still too small to be seen microscopically, could permit an increased diffusion of small molecules in solution. The resultant increase in permeability could be recognised by an increased movement of small, passively transported molecules such as creatinine, fructose, mannitol, erythritol or insulin across the mucosa, either from blood to intestinal lumen or from intestinal lumen to blood (Loehry et al., 1970).

Such a small increase in pore size could allow transudation of large amounts of small molecules in solution, and this could result in diarrhoea (Moon, 1979).

Diarrhoeal diseases associated with large increases in pore size sufficient to permit exudation of plasma protein are sometimes characterised as protein-losing enteropathies (Murdoch, 1986). Chronic enteritis of unknown cause, with lymphangiectasia, exudation of plasma protein to the intestinal lumen and hypoproteinemia is a well characterised example of such a syndrome in dogs (Finco et al., 1973). The process also occurs in some animals with paratuberculosis (Johne's disease) (Allen et al., 1974). Increased permeability in such syndromes probably occurs in part due to increased hydralulic pressure in the mucosa due to obstructed lymph flow. Such pressure could increase the size of existing pores or open new ones (Yablonski and Lifson, 1976). Also some chemical mediators of the inflammation process probably act directly to increase pore size (Merkal et al., 1970). Nematode infections could also lead to protein-losing enteropathy. The pores for protein movement from plasma to intestinal lumen in these infections are ultrastructurally recognisable

separations of the junctional complexes between epithelial and endothelial cells (Barker, 1975).

Haemorrhagic exudates require more than a ten-thousand-fold increase in membrane permeability, that is, the normal mean effective pore size of 0.5mm must increase to several microns or more. Exudation through the inflammed or necrotic small intestine in Clostridium perfringes type C enteritis in new-born pigs is an example of such a massive increase in membrane permeability. The presence of fibrin clots and whole blood in the intestinal lumen is evidence of defects (pores) in the membrane large enough to accommodate macro-molecular fibrinogen and erythrocytes. These large pores are found in areas where epithelial and endothelial cells have been lost or ruptured (Moon, 1979).

While increased permeability contributes to diarrhoea in some inflammatory lesions of the intestines, intestinal inflammation does not always result in increased intestinal permeability. For example, monkeys and rabbits with salmonellosis and acute enterocolitis showed normal intestinal permeabilities to albumin, mannitol and erythritol (Giannella et al., 1976) which led to the idea that active mucosal secretion rather than filtration

through a leaky epithelium accounted for the enteric fluid loss (Fromm et al., 1974).

1.3 Nature of Faeces in Diarrhoeic Dogs

The changes in daily volume, frequency of passage, colour, odour and consistency of faeces are useful indicators to diagnosing the problem area in the small intestine (Strombeck, 1979). The cardinal sign of acute inflammation of the small intestine is large-volume diarrhoea (Anderson, 1975; Murdoch, 1986). Red blood may be seen in the faeces (haematochezia) if the transit time is very short, or if bleeding occurs at the terminal part of the gastrointestinal tract (e.g. due to coccidiosis or foreign body) but if digestive action and bacterial degradation of haemoglobin have taken place, melena (faeces of dark colour) is seen. A rapid transit time also minimizes bacterial reduction of bilirubin and diarrhoeal faeces tend to be tan or even yellow in colour instead of the usual brown. The foul odour of diarrhoeal faeces is influenced by the degree of fluidity and the amount of fat, undigested protein and blood present. Undigested food components may be found in the faeces. T he rate of bowel movements in small intestinal disease is influenced by the degree of local irritation and the proportion of

water in the faeces, while colo-rectal disease is indicated by a greater frequency of small volume bowel movements which is related to the intensity of local irritation which induces repeated tenesmus (Anderson, 1975).

A great deal could be learned from observation of the faeces (Murdoch, 1986). Maldigestion and malabsorption often have a characteristic sour odour and sometimes fat droplets may be seen floating on the surface (Strombeck, 1979). Nutritional diarrhoea due to diet changes causing reduced digestion or absorption of nutrients is characterised by passage of watery, dark, evil-smelling faeces for several days (Murdoch, 1986). The use of diarrhoeal faecal appearance in localising GIT problems and its cause has also been reviewed by Murdoch (1986).

1.4 Causes of Diarrhoea in Dogs

The causes of diarrhoea are numerous and include, enteric bacteria, viruses, helminths, protozoa, endocrine disorders like pancreatitis, malabsorption syndromes, liver disorders and nutritonal diarrhoea (Anderson, 1975; Strombeck, 1979; Murdoch, 1986). For proper diagnosis and

treatment, it is important to know the causative agents and understand their pathogenesis in diarrhoea.

1.4.1 Dietary Factors

Nutritional diarrhoea could occur from various etiologies which include: (a) reduced digestion or absorption of nutrients due to a change in diet and problems with certain feeds like carbohydrates, milk and proteins, (b) passage of nutrients that encourage intestinal bacterial activity, (c) lack of dietary fibre and (d) dietary allergy (Ferguson, 1976; Murdoch, 1986).

Frequent changes in the diet of the dog may result in an increased incidence of diarrhoea (Breitschwerdt et al., 1982; Murdoch, 1986), especially when changing from a complete dry, cereal-based diet to canned or fresh meat, or from a diet of fresh meat to canned meat. The diarrhoea would be watery, dark and with a foul odour for several days; faecal consistency would return to normal after a couple of days, indicating an adaptation to the change in diet (Murdoch, 1986).

Diarrhoea due to intolerance to a high percentage of carbohydrates in the diet although uncommon, could occur in dogs. Such diets would include cereal-based biscuit, bread, milk, potatoes, and so on. Reduction in the amount of carbohydrates in the diet may improve the condition (Murdoch, 1986). The intolerance is more obvious when the grains or potatoes are raw (uncooked).

Milk intake can be a problem when the animal has not been used to a regular intake resulting in gastroenteritis in dogs and cats. It has been demonstrated that, if more than one gram of lactose per kilogram body-weight (one pint of milk to a twenty kilogram dog) is fed to a dog, it could lead to severe diarrhoea, as adult animals may be deficient in lactose enzyme (Murdoch, 1986).

Murdoch (1986) also explains dietary problems with proteins as being rare in the dog and cat, but when they do occur, it is either due to an all raw meat or all-meat diet, which would result in a high incidence of recurrent diarrhoea or frequent bouts of watery, foul-smelling diarrhoea respectively; also over-cooking of meat reduces its digestibility and could lead to diarrhoea. It was also observed that some dogs are intolerant to offal, particularly liver, kidney and heart.

Particular diets should be avoided if an animal is observed to have diarrhoea after being fed such a diet.

Sometimes, the passage of certain substrates encourages intestinal activity resulting in diarrhoea. For example, a diet rich in meat when fed to a dog would increase the activity and number of Clostridium perfringes and vice versa with predominantly cereal diet (Murdoch, 1986). Also, poorly digested nutrients or spoiled food could cause diarrhoea in a similar method. This could also occur in puppies from over-eating leading to intestinal overload and poor digestion.

Murdoch (1986) also described how the lack of dietary fibre could result in gastro-intestinal problems in the cat and dog. In the wild, these animals apart from consuming a diet of highly digestible proteins (skeletal muscle, liver and so on), also ingest material of low digestibility (skin, tendons, and so on) which aids in persistalsis and removal of residues. Diets of high digestibility and low fibre would lead to increased bacterial activity and diarrhoea (Murdoch, 1986).

According to Strombeck (1979), the gastro-intestinal tract or contents contain many different types of antigens most of which are dietary. With a constant diet, the same antigens enter the intestine at regular intervals; other transient antigens are consumed infrequently.

Another primary source of antigens includes that produced by the normal microflora of the intestine, which are located at specific sites along the intestinal tract and this is invariable daily in the normal animal. Bacterial antigens in the diet and those of intestinal parasites are included.

In allergic nutritonal diarrhoea, hypersensitivity to a particular antigen damages the intestinal mucosa and increases permeability to absorption of more antigens into the body, it is not possible to determine if these are a cause or an effect of an abnormal immune response, but it is believed to be a cell-mediated or delayed type of hypersensitivity. The response of a dog to a dietary allergen occurs soon after a meal and are mostly immediate reactions; some may be Arhtus-type reactions (Strombeck, 1979). Ferguson (1976) reported that this hypersensivity leads to a protein-losing enteropathy with a secretory and motility derangement resulting in vomiting and diarrhoea.

The incidence of true dietary allergy as a cause of diarrhoea in the dog and cat is unknown. Cases implicating milk, beef, horsemeat and wholemeat have been described (Murdoch, 1986). Gluten, a toxic protein found in wheat and many other cereals, could cause diarrhoea in dogs and cats (Strober, 1976), by having a cytotoxic effect on villi epithelial cells resulting in maldigestion and malabsorption with increased secretion and diarrhoea (Strombeck, 1979).

1.4.2 Bacteria

Diarrhoea due to bacteria can be caused by the disruption of the normal microflora due to transient intake and eventual colonisation of the gastrointestinal tract by pathogenic bacteria. This disruption could be caused by interference with the homeostasis-maintaining mechanisms of the tract. Most causes of diarrhoea would lead to this (e.g. dietary changes, malnutrition, stress leading to motility disorders and oral antibiotic) (Drachman, 1974). Once the bacteria colonise the gastro-intestinal tract, they multiply and produce diarrhoea by one of three means: (a) enterotoxin production; (b) mucosal invasion; and (c) sub-mucosal invasion (Strombeck, 1979).

Experimental work has proved that Salmonella species, Camphylobacter species and invasive E. coli strains penetrate the mucosal barrier of the gastro-intestinal tract, whereas, other bacteria like entero-toxigenic E. coli, Staphyloccoccus species and Klebsiella species produce diarrhoea via the production of enterotoxins (Banwell and Sheer, 1973). Apart from this experimental data, there are very few documented descriptions of bacterial diarrhoea in the dog and cat (Murdoch, 1986).

Salmonella spp:

These are enteric bacteria of the genus Salmonella. They have a worldwide distribution and an extensive host range consisting of wild and domestic animals. The main means of transmission is through the faecal-oral route, although it could occur from contaminated feeds and formites, water and drugs, and so on (Edwards and Galton, 1967; Cherubin, 1972; Silliker, 1980).

These organisms can produce diarrhoea by epithelial invasion, disruption of the brush-border resulting in acute inflammatory reaction. However, there is no evidence that permeability is altered

of plasma fluid (Giannella et al., 1976). Salmonella infection also leads to hypermotility resulting in diarrhoea (Anderson, 1975; Strombeck, 1979; Murdoch, 1986).

Certain species like S. typhimurium can elaborate an enterotoxin that could result in diarrhoea via intestinal hyper-secretion (Koupal and Deibel, 1975) thought to be mediated by increased cellular cyclic adenosine monophosphate (cAMP). This mechanism has not been properly documented in the dog and cat (Murdoch, 1986).

The clinical form and carrier states of salmonellosis are known to occur. In the adult dog, the carrier state occurs more commonly than clinical salmonellosis (Borland, 1975) and this could persist for some months with the organisms being excreted in the faeces (Buxton and Fraser, 1977). Clinical salmonellosis is more common in the young, debilitated and pregnant animals (Borland, 1975; Morse and Duncan, 1975).

Salmonellosis is enzootic in the canine population and occult carriers are probably present in any sizeable colony; source of infection is usually an infected dog (Morse and Duncan, 1975; Morse et al., 1978).

The clinical signs vary from a mild, chronic intermittent, sometimes bloody and fetid diarrhoea to an acute febrile illness in which diarrhoea, pneumonia, vomiting, abortion, still-birth and death could occur (Borland, 1975; Morse and Duncan, 1975). The disease is often complicated by simultaneous infections with two or more serotypes (Morse and Duncan, 1975) and household dogs yeild a wider variety than kennel or stray dogs (Shimi et al., 1976). According to Morse et al. (1976), re-infection with another serotype following elimination of the original pathogen is likely.

All salmonellae are potential pathogens for man and animals particularly as many are non-host adapted. Therefore, dogs discharging salmonellae are potential sources of infection for man especially in children (Ojo, 1974; Borland, 1975; Britt et al., 1978).

A variety of isolation rates for salmonellae have been reported from various countries ranging from 0.6% to over 30% of the dogs examined (Borland, 1975; Morse and Duncan, 1975). In Nigeria, Britt et al. (1978) had an isolation rate of 18% at Vom and Ojo (1974) had 8% at Ibadan. The wide variation in these isolation rates could be due to the organisms

lodging in the mesenteric lymph nodes where rates of isolation have been higher than from the intestinal contents (Khan, 1970). Furthermore, faecal excretion of the organisms is intermittent (Cruikshank, 1965; Burnie et al., 1983).

Animals continue to shed Salmonella organisms for longer periods if treated with antibiotics than if untreated; because these organisms when they invade the intestinal mucosa, localise in the macrophages making it impossible for drugs to destroy them. Therefore, treatment may lead to carrier states (Morse and Duncan, 1975) although treatment with chloramphenicol (50mg/kg) given three times a day) for clinical cases have been recommended (Anderson, 1975).

Escherichia coli:

This is an enteric bacterium of the family Enterobacteriaceae. This organism appears to be the common cause of acute diarrhoea in dogs resulting in a fluid diarrhoea with or without haematochezia (Banwell and Sheer, 1973); it causes colibacillosis, a high morbidity diarrhoeal disease of variable mortality rate in kittens and other newborn animals (Anderson, 1975). Dillon (1986) has advocated that

that the occurrence of this disease is related to host resistance rather than bacterial pathogenicity. The organism does not cause any problem until it colonises the intestine (Sack et al., 1976).

Different strains of E. coli cause diarrhoea basically by two different methods: (a) Invasiveness: invasive strains attach and penetrate the intestinal mucosal epithelium and result in septicemia (Strombeck, 1979). The incidence and characteristics of this aspect of colibacillosis in small animals is poorly documented. There is increased intestinal fluid due to the damaged mucosa which secondarily stimulates hypermotility (Anderson, 1975) resulting in diarrhoea, (b) Enterotoxigenicity: entero-toxigenic strains produce enterotoxins but do not cause disease as the enterotoxin is not effective in the colon unless it gets to the small intestine (Banwell and Sheer, 1973); thus, pathogenicity arises when the organisms colonises the small intestine.

Pathogenicity depends on: (a) Adherence of E. coli to the brush border of the intestine on specific receptors (i.e. presence of an adhesive antigen). For example, the adhesive factor or antigen is recognised as K88 or 987P antigen in pigs and K99 in the calf and lamb (Tzipori, 1985).

This factor has not been properly identified in small animals. The gastric acidity acts as a reasonably effective barrer to this adhesion factor. This is usually defective in neonates and lack of colostrum intake (Tzipori, 1985). (b) After adhension, the organism has to adapt itselt to the small intestine and proliferate; (c) It then produces enterotoxins which are also essential for further proliferation and establishment on the small intestine, by inducing changes in the lumen at the mucosal surface which enhances the establishment (Takeuchi et al., 1978).

Thus, enteropathogenic - enterotoxic E. coli colonise the mucosa, liberate enterotoxins which lead to diarrhoea. The organism elaborates two types of enterotoxins: (a) heat-labile (LT) enterotoxin (at 65°C) which is composed of two polypeptide subunits A (molecular weight of 25,000) and B (molecular weight of 11,500) (Tzipori, 1985). The sub-unit A stimulates adenylate cyclase activity leading to increased concentration of intracellular cyclic AMP in the intestinal epithelial cells which results in (i) a secretory effect by increased sodium ion secretion and loss of chloride ions and water in the villous cells; both effects lead to diarrhoea (Tzipori, 1985); (b) Heat stable (ST) enterotoxin - this bears no

antigenic cross-reactivity to the LT enterotoxin; there are two types: STa which is methanol-soluble, active in the neonatal piglet and detectable by the suckling mouse model and STb, a methanol-insoluble enterotoxin, active in the weaned piglet and rabbits and undetectable by the suckling mouse model (Tzipori, 1985). Escherichia coli may produce STa or STb or both (Tzipori, 1985). Furthermore, ST produced by strains isolated from various host species may differ both in molecular weight and amino acid sequence; STa is found in E. coli strains of human and animal origin while STb strains are usually of porcine origin (Tzipori, 1985).

The ST enterotoxin stimulates the activity of guanylate cyclase leading to an increase in cyclic GMP which takes place only in the intestinal epithelial cells (suggestive of a specific toxin receptor site); this action is mainly anti-absorptive to water and electrolytes resulting in diarrhoea. The mechanism is not well understood (Tzipori, 1985).

Thus, in terms of enteropathogenicity of E. coli the following would seem to prevail:-

i. Attachment (+) enterotoxin (+) = pathogenic

ii. Attachment (+) enterotoxin (-) = possibly pathogenic

iii. Attachment (-) enterotoxin (+) = not pathogenic

iv. Attachment (-) enterotoxin (-) = not pathogenic (Tzipori, 1985).

Even when classic enteropathogenic strains are absent, E. coli could cause diarrhoea if adhesion and/or other factors allow colonisation (Tzipori, 1985).

Previously, serotypes 086:B7, 026:B6 and 0126:b16 have been implicated in catarrhal and haemorrhagic enteritis of dogs (Fox et al., 1965) but recently, serotypes have been de-emphasised as many previously not associated with diarrhoea have now been documented to be pathogenic (Farmer et al., 1977). Also, Wilk (1981) did not associate enteritis with a higher frequency of specific serogroups. It is now apparent that many strains of E. coli (regardless of serotype) cause diarrhoea because they have acquired plasmids for enterotoxin production (Cross et al., 1976; Sack, 1976). There is still much to be learned about the role of E. coli in infant, adult and animal diarrhoea (Farmer et al., 1977).

A broad-spectrum antibiotic active against gram negative organisms is usually recommended for treatment (Anderson, 1975). Wilk (1981) found polymixin B, gentamicin, furazolidone and nitrofurantoin effective against all strains of E. coli isolated from both healthy and diarrhoeic dogs; they were resistant to penicillin, ertythromycin and sulphonamides. It was also found that a few strains were resistant to neomycin, kanamycin, chlortetracycline and streptomycin. All the E. coli strains isolated by Adetosoye and Ojo (1983) in Nigeria from diarrhoeic kids and mature goats were resistant to oxytetracycline, streptomycin, ampicillin and triple sulpha drugs.

Yersinia enterocolitica:

This is another enteric pathogen of the family Enterobacteriaceae that could cause an acute diarrhoeal disease (gastroenteritis syndrome) and septicemia in man. The organism is not known to cause disease in animals although some naturally occurring or experimentally induced yersiniosis have been reported in animals (Pederson, 1976; Une, et al., 1977).

The mechanism by which Yersinia enterocolitica produces diarrhoea is not well defined (Pai and Mors, 1978) but presently, there are virulence markers normally assayed for and used in determining pathogenicity: (a) Heat-stable (ST) enterotoxin - the exact role of this toxin is not known but it causes a watery diarrhoea similar to that caused by enterotoxigenic strains of E. coli (Banwell and Sherr, 1973). This has been found primarily in human strains followed by animal, water, raw milk and food strains (Pai and Mors, 1978). The toxin is detected by the infant mouse system (Pai and Mors, 1978) and the rabbit ileal loop test of pig gut (Mosimabale and Gyles, 1982); (b) calcium dependency at 37°C: This virulence marker is due to a plasmid coded for a calcium requirement for growth at 37°C, which is tested for by growing the organism on magnesium oxalate agar (Gemski et al., 1980). Those species of Yersinia that are positive for calcium dependency areconsidered to have some virulence; (c) Autoagglutination at 37°C - this test indicates potential virulence and negative results should be carefully interpreted, as this factor could be easily lost with sub-culturing (Schieman and Fleming, 1981). A positive result is seen as agglutinated cells concentrated along the

lower wall and bottom, with a nearly clear medium in the upper portion of the tube after 24 h incubation at 37°C and a nearly uniform turbid suspension without agglutination at 22°C (Laird and Cavanaugh, 1980); (d) Invasiveness-demonstrated by the sereny test, that is the ability to produce conjunctivitis in guinea-pig, indicates the invasiveness due to the presence of 41-megadalton plasmid and 42.2 + 1.1 megadalton plasmid (Gemski et al., 1980) and (e) Hela cell infectivity - this is the ability of pathogenic strains of Yersinia enterocolitica to actively penetrate Hela cells (not just adhere); and cell culture studies normally reveal damage and detachment of Hela cells (Schieman and Devenish, 1982). This relates to the invasive aspects of virulence rather than diarrhoeal aspects (Mosimabale and Gyles, 1982).

Recently ST enterotoxin production has been recommended to characterise Y. enterocolitica and is believed to play no role in pathogenicity (Aulisio et al., 1983). This is because some strains negative for this toxin still caused diarrhoea in mice indicating that other virulence factors are responsible for diarrhoea. Strains that are calcium dependent, autoagglutinate at 37°C and are invasive,

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are considered pathogenic (Mosimable and Gyles, 1982). These three factors have been demonstrated to be encoded by the same plasmid. Other pathogenic strains are known to survive phagocytosis and even multiply within the macrophages. The virulence of the organism is also enhanced at lower temperatures (less than 25°C) and this may be related to its resistance to phagocytosis; mechanism responsible is not known (Fukushima et al., 1984).

The disease yersiniosis is more important in human medicine than in veterinary medicine (Morris and Feely, 1976). In many cases, Yersinia enterocolitica has been isolated from clinically normal dogs (Toma and Deidrick, 1975; Soulias, 1976; Yanagawa et al., 1978; Szita et al., 1980). There are a few reports of canine enteritis associated with yersiniosis (Farstad et al., 1976; Papageorges and Gosselin, 1983). It would seem that isolation of the organism is not attempted in many clinical cases of canine enteritis (Langford, 1972).

The prevalance of yershiniosis in dogs was found to be low in Canada and the United States being 0.4% and 0.6% respectively (Pedersen, 1976; Staley et al., 1977) but in Japan and the Scandinavian countries, 6% of the dogs were found to be carriers

of the organisms (Soulias, 1976; Pedersen, 1976, Kaneko, 1976; Yanagawa et al., 1978). To date, there are no documented isolations of Y. enterocolitica from dogs in Nigeria.

The disease in dogs is a mucoid enterocolitis without fever or abdominal pain (Papageorges and Gosselin, 1983). Infection with the organism has been identified as a zoonosis; however, pigs are considered the major reservoir of the organism although, dogs are suspected to transmit the disease to man in certain instances (Toma and Deidrick, 1975; Wilson et al., 1976; Kaneko et al., 1977; Szita et al., 1980; Wooley et al., 1980). A definite mode of transmission from dog to man has not been confirmed although it has been suggested to be direct (Fukushima et al., 1984).

There were no significant differences in the monthly or seasonal incidence of the disease in dogs (Kaneko et al., 1977); but Fukushima et al. (1984) in Japan observed that variations in temperature (low temperatures) influenced the spread of Y. enterocolitica among dogs in Japan, with these dogs possibly becoming carriers in the cold months. This role of low temperatures in the pathogenicity is yet to be clarified.

Kaneko et al. (1977) found no significant difference in the distribution of the organism with the ages of dogs; also that, serotypes O:3 and O:9 strains may be harboured by dogs, and O:5 and O:6 could occur transiently; serotype O:3 has been frequently isolated from puppies (Fukushima et al., 1984) and it is known to be pathogenic for man (Kaneko et al., 1977).

The organism has been found to be sensitive to chloramphenicol, tetracycline, gentamicin, cephradine and trimethroprim - sulfamethoxazole and resistant to ampicillin (Toma and Lafleur, 1974; Van Noyen et al., 1979; Papageorges and Gosselin, 1983).

In Nigeria, two reports have been made on Y. enterocolitica gastroenterities in human beings and the serogroup O.3 was found to predominate (Anjorin et al., 1979; Agbonlahor et al., 1983). Agbonlahor et al. (1986) reported the prevelence of this infection in domestic animals. There is a report of the serogroup O.3 in a piglet by Adesiyun et al. (1984); also serological evidence of infection by this serogroup and others (O.3 and O:12,26) in dogs (Adesiyun et al., 1986).

Campylobacter Species:

Campylabacteriosis has been recognised as a problem in animals since 1909 (Fox et al., 1984).but was observed to affect man in 1972 (Murdoch, 1986). The isolation rate in man has increased drastically since then and in many countries exceeds that of Salmonella; this combined with the close association between pets and owners has revived interest in the organism (Nair et al., 1985; Fleming, 1983; Murdoch, 1986).

This organism is a gram-negative bacterium, cultured on selective media which inhibits the growth of competing organisms, under micro-aerophilic conditions. Campylobacter jejuni has not been conclusively proved to be a true enteric pathogen in the dog and cat (Murdoch, 1986); most surveys however indicate its isolation from faeces of about 10% of clinically normal dogs (Bruce and Fleming, 1983; Burnie et al., 1983). This isolation rate could be higher in the kenneled dogs because (a) they become infected after reaching kennels, or (b) may be carriers and only excrete the organism under stress or concomitant intestinal infection (e.g. parvovirus infection) (Murdoch, 1986).

The organism has been reported by Nair et al. (1985) to have an incidence of 1.7% in diarrhoeic dogs, 3.1% in clinically normal dogs and 6.7% in non-diarrhoeic but sick dogs, mainly in puppies under six months of age.

Dogs and cats have been associated with less than 5% of human cases, but in the case of a positive isolation, the possibility of a zoonosis should be investigated and treatment to be continued until excretion has ceased (Fleming, 1983; Nair et al., 1985; Murdoch, 1986).

Staphylococcus aureus:

The enterotoxin is believed to cause diarrhoea in food poisoned dogs but it is not a major causative agent of diarrhoea in dogs (Anderson, 1975).

Clostridium perfringes:

The clinical importance of isolating this organism from diarrhoeic dogs and cats is uncertain (Anderson, 1975). Carman and Lewis (1983) reported chronic, recurrent diarrhoea in a dog caused by Cl. perfringes type A; this is the same causative agent for non-fatal food poisoning in man causing diarrhoea and oramps (Anderson, 1975).

1.4.3 Viruses

Although viruses have been isolated from the faeces of normal dogs (Binn et al., 1970; Strombeck, 1979), it is yet to be determined whether they are part of the normal microflora and contribute to diarrhoea under special circumstances or if they are pathogenic and foreign and consistently produce disease on entering the host (Strombeck, 1979). Diarrhoea due to viruses often responds to antibiotics, which implicates bacteria in the pathogenesis.

Canine Parvovirus:

The disease caused by this virus was observed to emerge between 1978 and 1981, with worldwide outbreaks (Kelly, 1978; Hayes et al., 1979; Hitchcock and Scarnell, 1979; Appel et al., 1979). According to Appel et al. (1979), the disease is similar to mink enteritis' (first observed in 1947) and the causal agent is a parvovirus serologically indistinguishable from feline panleucopenia virus. The virus is one of the smallest of the true viruses, contains DNA and can develop only in the nucleus of dividing cells; infections of rapidly growing cells are common and the constantly replicating epithelial cells are the favourite growth sites for some parvoviruses (Appel et al., 1979).

The epitheliotropic virus causes diarrhoea by selective destruction of immature, proliferative crypt epithelial cells, leading to varying degrees of villous atrophy, malabsorption and diarrhoea (Kent and Moon, 1973). The initial clinical signs (anorexia and dullness) develop four days after contact with the virus; the enteric signs develop a day later. Affected dogs deteriorate quickly and become recumbent within eight hours and death could occur within twenty-four hours from the onset of clinical signs (Murdoch, 1986). Faecal excretion starts on the third day of infection and is maximum between the fourth and seventh day, after which it drops rapidly.

Two forms of the disease have been reported: (a) enteritis with sometimes bloody diarrhoea in dogs of all ages, but more commonly in adults; and (b) myocarditis with sudden death seen in puppies less than eight weeks old (Appel et al., 1979; Hayes et al., 1979; Harcourt et al., 1980; Van Den Ingh et al., 1980; Prange et al., 1982). As maternally-drived antibody protects puppies and majority of the adult dogs are usually vaccinated or previously infected, most puppies are immune and the cardiac form is now uncommon (Murdoch, 1986). The disease

is transmissible from dog to dog via contact with contaminated environment (Eugster et al., 1978; Appel et al., 1979). Demonstration of the virus in faeces supports a diagnosis of canine parvoviral enteritis; conversely, failure to detect these viral particles rules out the disease in any acutely ill animal but is of no significance in the convalscent dog (Pollock et al., 1983). Detection could be by flourescent antibody technique or electron-microscopy (Appel et al., 1979).

Various serological methods are available for determining antibody titres in dogs, for example, haemagglutination-inhibition tests (HA-HI) used on faecal samples which is the most sensitive (Studdert et al., 1983). In most dogs the titres are detectable when clinical signs become obvious and rise rapidly during the course of the clinical illness; titres also vary markedly between individuals (Pollock et al., 1983).

In Nigeria the disease was first reported by Adeyanju et al. (1984) and then Ezeokoli et al. (1985) and it is believed to be a recent introduction into the country via importation of exotic breeds of dogs from the United States and Europe.

Treatment is specific using dichlorvos (31.5 mg/kg) or Disophenol (D.N.P. 2.2 mg/kg) (Anderson, 1975) blood transfusions are given if necessary and vitamins, intravenous fluids and antidiarrhoeals are also useful.

Roundworms:

Toxocara species are the most common and widely distributed nematodes of the dog and cat (Anderson, 1975), particularly in puppies (Malloy and Embil, 1979). Toxocara canis infection in the small intestine leads to a thickened intestinal wall, distension of the abdomen (pot-bellied appearance in puppies due to ascites), mucoid diarrhoea, stunted growth and unthrifty appearance; large numbers of worms could result in intestinal obstruction or intussusception and rarely, perforation (Anderson, 1975).

Else et al. (1977) reported an incidence of 16.2% for T. canis in the East Anglian Region of England which is high when compared to 2% of Cook (1965) in the same area. Reports confirm the predominance of this infection in young dogs (Holt, 1976; Else et al., 1977). Reports from other countries include those from Canada (Unruh, 1973) and Iraq (Al-Saffar et al., 1962). There are some reports

from Nigeria (Saror, 1979; Dada et al., 1979; Baba et al., 1983; Ezeokoli, personal communication).

The present prevalence of Toxocara infection in household dogs and its impact on sanitation requires that public health authorities, pet owners and veterinarians in crowded urban areas give more attention to this parasite. A programme of public health education on this aspect should be developed (Malloy and Embil, 1978).

Drugs like piperazine salts (100-200 mg/kg) are effective in getting rid of adult and immature worms in dogs. Dichlorvos (31.5 mg/kg) and pyrantel tartate are also effective against adult worms. Toxocarid pneumonia should also be treated with a broad-spectrum antibiotic. Proper hygiene is important for breeding kennels and environment (Anderson, 1975).

Tapeworms:

Cestodes of dogs and cats are known to cause flatulence and diarrhoea which alternate with formed faeces (Anderson, 1975). Vomiting may occur sometimes resulting in expulsion of the adult tapeworms (Soulsby, 1968). Experimental infection with tapeworms did not however produce a recognisable clinical syndrome.

Dipylidium caninum commonly infects dogs and rarely cats and is transmitted by the ingestion of infected Ctenocephalides canis and C. felis (dog and cat fleas respectively), the intermediate hosts. The gravid proglottids irritate the perineum as the migrate through the anus and affected dogs would be seen to rub the perineum on the floor or ground (Anderson, 1975). Else et al. (1977) reported an incidence of 10% in dogs in East Anglian Region of England, mainly in adult animals. A higher incidence (87.8%) was observed in Nigeria from stray dogs (Dada et al., 1979). Echinococcus granulosus had a very low incidence in Zaria, Nigeria (Dada et al., 1979).

Taenia infections are acquired by dogs from cysticerci-infected sheep, rabbits and other rodents and ungulates. Diphyllobothrium latum, a cestode infection of dogs is transmitted by the consumption of fresh-water fish like pike, trout, salmon, and others, which have acquired the plerocercoid of the helminth by ingestion of crustaceans (first intermediate host) (Anderson, 1975). Another cestode of the dog that is of public health importance is Echinococcus granulosus. Echinococcus multilocularis found in cats also causes alveolar hydatid disease in man (Wobeser, 1971).

Treatment is effective with drugs like bunamidine hydrochloride (25-50 mg/kg) for the tapeworms, by dissolution of the scolecies. Niclosamide (155 mg/kg) is also useful in removing the entire tapeworm; higher doses (300 mg/kg) are needed to remove E. granulosus (Anderson, 1975).

Whipworms:

Trichuriasis due to Trichuris vulpis has been reported to have a low incidence worldwide (Else et al., 1977; Dada et al., 1979; Hassan, 1982). Control of this infection is through the use of specific drugs like dichlorvos (31.5 mg/kg) and supportive therapy including vitamins and haematinics, and sanitary measures to get rid of the eggs and larvae (Anderson, 1975).

Strongyloides stercoralis:

This is a nematode infection which results in diarrhoea and cough in puppies, kittens and other mammals including man. Diarrhoea becomes obvious seven to ten days after the initial onset of anorexia, purulent occular discharge and a soft cough. The diarrhoea may be blood-stained. Clinically affected puppies usually die without fever. Stronglyloidiasis weakens puppies and predisposes them to other infections (Anderson, 1975).

Hassan (1982) found a very low incidence of the infection in the Western Area of Freetown (Sierra Leone). Dithiazine (22 mg/kg/day) for ten to twelve days is effective, with supportive therapy (Anderson, 1975).

Protozoa:

Coccidia are protozoan parasites of the intestine and usually more commonly of Isospora species in dogs and cats than Eimeria species. Isospora bigemina is the most pathogenic. The organism invades the subepithelial tissues of the intestine inducing an inflammatory response; others cause a more benign disease (Anderson, 1975). Malabsorption syndrome could occur if the disease is severe. The oocysts are excreted in the faeces.

The diarrhoae is usually catarrhal or when severe haemorrhagic (Anderson, 1975; Correa et al., 1983) and is associated with dehydration, anorexia, fever and vomiting. The incidence of coccidiosis is highest in puppies and kittens under conditions of high density housing (i.e. kennels, catteries and pet shops) and following weaning, shipping, exposure to cold and other stress factors. Adult dogs may become infected following high dose exposure (e.g. at a boarding kennel) (Ewing and Buckner, 1974).

Treatment of clinical coccidiosis is with sulphonamides - sulphamethoxine (27.5 mg/kg twice daily), or nitrofurazone (5.9 mg/kg thrice daily) for seven to ten days, or as long as oocysts appear in the faeces; supportive therapy with fluid and electrolytes, vitamins or blood transfusion may be necessary (Anderson, 1975). Treatment is not warranted in non-clinical cases, as this may just indicate resistance rather than an infection (Anderson, 1975). Amprolium, a coccidiostat has been useful in commercial kennels, given for seven to twelve days at a dose of 110 to 220 mg/kg/day.

Giardia canis is another protozoan parasite found in the faeces of normal and diarrhoeic animals and was formally regarded as non-pathogenic. In low numbers in the villous epithelial cells and intervillous spaces, it does not cause problems but when the numbers increase to cover the epithelial cells, diarrhoea ensues. It has not been proved to be invasive (Anderson, 1975).

It has been speculated that Giardia numbers are maintained at a low level by interactions with the normal microflora, and anything that changes the latter would cause the protozoa number to increase (Anderson, 1975).

Giardiasis has been considered to be a rare condition in dogs (Barlough, 1979) and infection could result in ulcerative colitis (Ewing and Aldrete, 1973; Watson, 1980), chronic diarrhoea (Shakrabati et al., 1982) and malabsorption syndrome (Merrit, 1980). The incidence in dogs varies considerably and has been reported to be as high as 30% in an American study (Wolf and Eckert, 1979). Stray dogs were more commonly affected than owned dogs (Burrows and Lillis, 1967). The incidence is higher in dogs six months old or younger than in older dogs (Pfeiffer and Supperer, 1976). However, Burnie et al. (1983) reported a low incidence with no significant difference between the age groups.

The Giardia species in small animals may be the same in humans and animals could be regarded as a reservoir of infection for humans (Raizman, 1976). Metronidazole (66 mg/kg/day for five days) is adequate treatement and a high-protein diet would effectively suppress the infection in puppies and adults (Anderson, 1975).

Entamoeba histolitica, the cause of amoebic dysentery in man may also cause dysentery in dogs. The organism localises in the caecum and colon of the dog causing dysentery (Anderson, 1975). The dog

is suspected to ingest the mature cysts in faeces from infected persons but the trophozoites from an infected dog could become established in the intestine of other dogs when given orally (Jordan, 1967).

There could be an interaction between Entamoeba and normal intestinal microflora that results in spontaneous entamoebiasis (Anderson, 1975). Jordan (1967) observed concurrent or pre-existing infections with Trichuris vulpis or Ancylostoma caninum to predispose the dog to severe entamebiasis. A systematic infection with the parasite has been reported in a puppy that had distemper with the organism being detected in the lungs, liver, kidney and spleen (Thorson et al., 1956). Treatment is with metronidazole (66 mg/kg/day) (Anderson, 1975).

1.4.5 Malabsorption Syndromes

This term includes all the chronic enteropathies that have persistent diarrhoea, nutrient loss in the faces leading to weight loss, chronic inflammation, mucosal-cell-enzymatic defect, villous atrophy, or thickened lamina propria. Thus, it is a provisional diagnosis and not definitive (Anderson, 1975). The condition has been observed to occur in some diarrhoeal disease of the small intestine of dogs and cats. In such cases, exocrine pancreatic function was found

to be normal (Pigeon, 1982).

Malabsorption could occur from: (a) a primary intestinal problem which is mainly in t he form of mucosal disease that affects its secretory and absorptive functions (Strombeck, 1979). Such problems include defects in mucosal transport systems, reduced mucosal surface area, reduced brush-border enzymes, disease involving the lamina propria and circulatory problems and (b) secondarily to the consequences of bacterial overgrowth of the small intestine produced by a loss of normal intestinal motility to the point of stasis; also develops when normal gastric secretion is lost (Strombeck, 1979). Malabsorption itself could result in bacterial overgrowth from the unabsorbed nutrients.

Villous atrophy, intestinal lymphangiectasia, and loss of intracellular mechanisms needed for lipid assimilation lead to diarrhoea and steatorr-hoea (Strombeck, 1979). Also, circulatory problems like hepatic cirrhosis or congestive heart failure could result in malabsorption of nutrients which are then lost via the intestine.

The clinical signs of malabsorption include weight loss and watery diarrhoea sometimes with undigested food and fat present; a sour odour may be obvious due to fermentation of carbohydrates in the colon (Strombeck, 1979).

Diagnosis of the condition is based on qualitative and quantitative faecal fat tests. The amount of faecal fat excreted should not exceed 50% of the dietary intake, depending on the type of diet fed (Strombeck, 1979). Absorptive qualitative tests reflect both digestion and absorption in the intestine. For example, the xylose-absorption test indicates if there is a mucosal disease, the pancreatic enzyme digestion and absorption test measures the action of chymotrypsin on a synthetic peptide (N-benzoyl-L-tyrosyl-P-aminobenzoic acid) which it splits into P-aminobenzoic acid (PABA) that is secreted in the urine. Dogs with pancreatic enzyme deficiency excrete less than 15% of PABA in the urine (Strombeck, 1979). Examinations for parasitic, microbial, dietary or management induced diseases should be done (Anderson, 1975).

For treatment, to reduce the work load on the intestine, small amounts of low-residue, highly nutritious and readily digestible diet should be fed one to three times daily. Increased food intake should be deferred until diarrhoea has subsided. Fat soluble vitamins should also be administered parenteraly (Anderson, 1975).

1.4.6 Endocrine Disorders

Chronic diarrhoea is a major problem in some dogs with hypothyroidism due to an alteration in gastro-intestinal motility (Kowalewski and Kolodej, 1977). This is due to a decreased frequency of electrically controlled activity so that smooth muscle contractile responses are reduced in the intestine (i.e. hypomotility leading to diarrhoea) (Strombeck, 1979).

Pancreatitis is a disease of middle-aged, obese dogs which often leads to diarrhoea due to altered motility of the intestine (Strombeck, 1979). Chronic pancreatitis and pancreatic atrophy are the most common causes of exocrine secretion deficiency due to destruction or loss of the acinar cells (Wormsley, 1972; Setari, 1975) and maldigestion - malabsorption syndrome (Wilson and Dietschy, 1971).

Other causes of exocrine insufficiency include inflammatory processes or neoplasia that occlude the pancreatic ducts (Wormsley, 1972; Setari, 1975).

Treatment of these conditions require specific enzyme replacement therapy and special diets (Strombeck, 1979).

1.4.7 Liver Disorders

Intrahepatic and extrahepatic disease could reduce bile acid secretion which results in maldigestion and malabsorption of lipids leading to diarrhoea with or without steatorrhoea (Hofman, 1973); the liver loses its capacity to extract bile acids from the circulation and resecrete them for release into the intestine. Abnormal bile salt metabolism could result from: (a) Reduction of liver parenchymal cell mass; (b) Impaired hepatocyte function as a result of disease; and (c) Disturbance of enterohepatic circulation of bile acids (Javitt, 1975; Bjorkhem and Danielsson, 1976).

Chapter 2

EPIDEMIOLOGICAL SURVEY OF DIARRHOEA IN DOGS OF ZARIA AREA

2.1 Introduction

Diarrhoea has been known to be a common problem of dogs, particularly puppies (Strombeck, 1979; Murdoch, 1986). The causes of diarrhoea in dogs are many and include the following: nutritional problems (Ferguson, 1976; Strober, 1976; Murdoch, 1986); bacteria (Fox et al., 1965; Cherubim, 1972; Anderson, 1975; Borland, 1975; Fleming, 1983); viruses (Kelly, 1978; Appel, 1979; Hayes et al., 1979); intestinal parasites (Anderson, 1975; Castro et al., 1976; Holt, 1976; Soulsby, 1968); malabsorption disorders (Anderson, 1975; Strombeck, 1979); endocrine disorders (Strombeck, 1975); and liver diseases (Javitt, 1975; Strombeck, 1977).

There are only few documented reports on diarrhoea in dogs in Nigeria (Oduye and Olayemi, 1977; Oduye and Bobade, 1979) although there are several reports on infectious agents capable of causing diarrhoea in dogs (Ojo, 1974; Idowu et al., 1977; Britt et al., 1978; Abdullahi, 1979; Olufemi

and Bobade, 1979; Saror et al., 1979; Baba et al., 1983; Adeyanju et al., 1984; Ezeokoli, personal communication; Ezeokoli et al., 1985). Our clinical experience at the Ahmadu Bello University Veterinary Teaching Hospital (ABUVTH) seems to indicate that diarrhoea in dogs, particularly young puppies, has high morbidity and mortality. As there is no documented information on the prevalence and importance of diarrhoea in dogs at ABUVTH, this study was undertaken with the following objectives: (a) to determine the prevalence of diarrhoea in dogs presented at ABUVTH; (b) to identify the causes of diarrhoea in the dogs; (c) and to analyse data on diarrhoea and its causes and relate to ages, breeds and sexes of affected dogs.

2.2 Materials and Methods

2.2.1 Restrospective Study

Data were derived from the case records of Ahmadu Bello University, Veterinary Teaching Hospital (ABUVTH). All dogs that attended the Small Animal Clinic of ABUVTH between January, 1984 and December, 1985 were included in this study. The data were examined to determine the prevalence of diarrhoea

and its relationship to season of the year, age, breed and sex of the dogs.

2.2.2 Clinical Study

A clinical study was done as follows:

Collection of Samples:

Faecal swabs were obtained from all diarrhoeic dogs and matched with appropriate controls (matched for age, sex and breed) that were brought to the Small Animal Clinic of the ABUVTH over a period of 18 months (March, 1986 and August, 1987). This was done by inserting sterile swabs into the rectum beyond the anal sphincter and rotated gently several times to ensure collection of enough faeces. The swabs were then immersed in separate enrichment media specific for enrichment and isolation of Escherichia coli, Salmonella species and Yersinia enterocolitica. Swabs were also taken from the liver, intestine and mesenteric lymph nodes of diarrhoeic dogs that died and from the beddings of household dogs in outbreaks and treated as above. For dogs brought to the Small Animal Clinic of ABUVTH monthly faecal samples were also obtained from all the diarrhoeic dogs and control dogs during the 18-month-period of study using polythene bags. These samples were obtained per rectum.

Isolation and Characterisation of Escherichia coli:

The rectal swabs from each dog were enriched in 10 millilitres of brain-heart infusion (BHI) broth at 37°C overnight, then plated unto eosin methylene blue (EMB) agar (oxoid) and incubated at 37°C overnight. Colonies with the typical characteristic greenish-metallic sheen were picked and innoculated unto nutrient agar slants in bijou bottles which were also incubated at 37°C overnight and stored at 4°C till needed for biochemical test.

Isolates that produced acid/acid on triple sugar iron (TSI) agar, were indole and methyl-red positive, Voges-Proskauer and urease negative, citrate (Simmon's) negative and non-motile were subjected to confirmatory chemical reactions with mannitol, glucose and lactose as described by MacFaddin (1977). Those isolates that fermented these sugars with gas production were considered to be E. coli.

Serotyping of E. coli isolates was done by the International Escherichia and Klebsiella Centre, Statens Seruminstitute, Copenhagen Denmark and Rijkskinstitute, Volksgezondheiden Milieu, hygiene, Bilthoven, Netherlands.

To determine the enteropathogenicity of *E. coli* isolates, a modifed method of Moon and Whipp (1971) was used to detect heat-stable (ST) and heat-labile (LT) enterotoxins. Twenty millilitres of brain-heart-infusion (BHI) broth were each dispensed into 250 ml Erlenmeyer flasks and sterilised at 120°C for 15 minutes. Overnight growth of test cultures and controls in BHI broth at 37°C served as inocula. Flasks containing inoculated BHI broths were incubated at 37°C in a waterbath with a gyratory shaker (6.835 x g) for 18 h. Growth was then spun down in refrigerated centrifuge (4°C) for 20 minutes at 17,000xg and the supernatent millipore filtered (0.45u). For ST enterotoxin, half the quantity of each supernatent was dispensed into bijou bottles and subsequently heated at 65°C for 30 minutes in a water bath. The other half of the supernatent was unheated and used for enterotoxin assay. Both enterotoxins were frozen at -20°C until needed.

Adult rabbits were sedated with 10 mg of 2% xylazine intramuscularly and local anesthesia achieved with 3 ml of 2% xylocaine intramuscularly on the line of incision. An incision of 12 cm in length was made over the linea alba to expose most of the gastrointestinal tract. The ileum was

isolated and moistened with warm sterile physiologic saline solution. Starting at the ileo-caecal junction, ten loops were made measuring 5 cm in length by ligation, using surgical catgut. The first loop was inoculated with 1 ml of a positive control (No. 36004 - LT/ST), the subsequent loops each with 1 ml of the test culture filtrates (i.e. one culture filtrate per loop), using hypodermic needles (24 or 25 guage); the tenth loop was inoculated with 1 ml of a negative control (C972 - LT/ST).

Thereafter, the loops were carefully returned into the abdominal cavity and the line of incision sutured up using catgut for the muscle and subcutaneous tissue and silk for the skin.

For ST enterotoxin detection, the rabbits were kept at room temperature for 6 h then sacrificed and the gut removed. The length of each loop was measured and the volume of fluid which accumulated in each loop was accurately measured. Results were expressed as the ratio of volume (ml) to length (cm). A ratio of 1.0 or greater was considered as positive as suggested by Guerrant et al. (1978).

For LT enterotoxin detection, the rabbits were kept for 18 h also at room temperature before sacrificing them and the same procedure was followed as for ST detection.

The sereny test for invasiveness for the E. coli strains (i.e. the ability of the organisms to invade tissues) was determined using the method described by Mosimabale and Gyles (1982) for Yersinia enterocolitica strains with a slight modification. Each isolate was grown on tryptic soy broth (TSB) and incubated at 25°C for 24 h after which 0.3 ml of each was inoculated unto tryptic soy agar plates (TSA) in duplicate to achieve confluent growth. The plates were incubated at 37°C for 24 h and then harvested in sterile normal saline. The cell suspensions were washed twice in saline through centrifugation. The saline suspensions of the organisms were adjusted through dilution to contain 4.26×10^{11} c.f.u/ml. Drops of each of these suspensions were put under the upper and lower eyelids of an eye of each guinea-pig. One eye each of two different guinea-pigs were inoculated with each isolate. Normal saline and a strain of Shigella dysenterae served as negative and positive controls respectively. The guinea-pigs were examined daily

for five days for evidence of keratoconjunctivitis as shown by ocular discharges, matted hair around the eyelids or frank opacity of the cornea.

To determine the antibiograms of E. coli isolates using the disc diffusion method, antimicrobial discs (Difco and Oxoid) and their concentrations were used as follows: chloramphenicol (30 mcg), gentamicin (10 mcg), tetracycline (30 mcg), neomycin (30 mcg), kanamycin (30 mcg), ampicillin (20 mcg), streptomycin (10 mcg), erythromycin (15 mcg), sulphadiazine (30 mcg), penicillin (10 International Units), and methicillin (5 mcg).

A modification of the disc diffusion method of Bauer et al. (1966) was used. The test isolates of E. coli were plated on EMB for isolation from where colonies were picked and inoculated into sterile BHI broth and incubated at 37°C for 24 h. Sterile cotton swabs were dipped into the standardised suspension of the isolates and streaked evenly over the entire surface of Mueller-Hinton agar plates. The plates were allowed to dry and the antimicrobial discs placed on their surfaces with sterile applicators. Five discs were placed per plate and the plates were incubated at 37°C for 24 h. The diameter of the zones of inhibition

surrounding each disc to the nearest millimeter were measured with a ruler and the results recorded as sensitive or resistant by referring to the interpretative chart of zone size with the FDA standardised disc method (Wick et al., 1971).

Isolation and Characterisation of Yersinia enterocolitica:

The rectal swabs were placed in 5 ml of sterile m/15 phosphate buffered saline (PBS), pH 7.6 for cold enrichment at $4^{o}C$ for 3 weeks as described by Schiemanm and Devenish (1980). All enriched samples were plated on desoxycholate citrate agar (DCA) after one week and three weeks and incubated at room temperature for 24-48 h after which non-lactose fermenting colonies were carefully picked onto NA slants for further identification.

Thereafter all tiny, non-lactose fermenting colonies were inoculated unto triple sugar iron (TSI) slants. All isolates that produced acid/acid with or without gas but without hydrogen sulphide production on TSI were inoculated unto urea agar slants and those that were urease-positive were assayed for phenylalanine deaminase activity and motility at

25°C and 37°C. Isolates that were phenylalanine deaminase positive, motile at 25°C but not at 37°C were presumptively identified as Yersinia species. These isolates were further confirmed using the lysine decarboxylase (37°C), arginine dihydrolase (37°C), ornithine decarboxylase (37°C, indole (25°C and 37°C), citrate utilisation (25°C), (methyl-red) and Voges-Proskauer (25°C and 37°C) and hydrogen sulphide production in SIM, and fermentation of rhamnose, mannitol, lactose, dulcitol, glucose, raffinose and maltose sugars. All these tests were determined using the method described by Bercouvier et al. (1978).

Morphologically and biochemically identified suspects of Y. enterocolitica strains were kindly confirmed and serotyped by Prof. H. H. Mollaret of the Pasteur Institute, Paris.

To determine the antibiogram of Y. enterocolitica strains, the antibiograms were determined using the agar plate dilution procedure of Ericssen and Sherris (1971) which involved the use of two-fold serial dilutions, ranging in concentration from 0.25 to 128 mcg/ml for each of the following antimicrobial agents: chloramphenicol, gentamicin, togamycin

(spectinomycin), erythromycin, streptomycin, ampicillin, sulphadiazine, lincomycin, tetracycline, nalidixic acid and neomycin on Mueller-Hinton agar. The minimal inhibitory concentration (MIC), that is the highest dilution of each antimicrobial agent which prevented growth of the isolate in mcg/ml was observed. Reference strains of E. coli (K12) were used as controls. The procedure is shown in Table 2.1. The millilitres of the appropriate intermediate concentrations were added to 18 ml of molten Mueller-Hinton agar to obtain the final concentration in the plates. The isolates were streaked on DCA plates, then subcultured into sterile tryptic soy broth (TSB) and incubated for 24 h at 25°C. A 10^{-4} dilution of the growth of each isolate was made in sterile distilled water from which inocula consisting of 0.025 ml were pipetted, using Epperdoft pipette, onto the antimicrobial agent-containing plates. Mueller-Hinton agar plates without antimicrobial agents were inoculated with the test isolates and used as controls. All the plates were read after incubation and the minimum inhibitory concentration (MIC) was noted.

The same disc diffusion procedure used for Escherichia coli isolates was utilised except for the initial plating for isolation of colonies of Y. enterocolitica which was done on desoxycholate citrate agar at room temperature instead of EMB at 37°C. The results were noted and interpreted in a similar manner.

The pathogenicity-assay for Virulence markers of Y. enterocolitica was determined using a slight modification of the autoagglutination test (at 37°C) procedure described by Agbonlahor (1986). Isolated colonies of Y. enterocolitica cultures on DCA plates after overnight incubation at 25°C were inoculated into a pair of duplicate tubes each containing 5 ml of methyl-red- Voges-Proskauer (MR-VP) broth. A set of tubes were incubated at 37°C and the other pair of 25°C overnight. Un-inoculated sterile MR-VP broth served as a negative control. The organisms were considered autoagglutination positive when the tubes at 37°C showed aggregated cells along the lower wall and bottom of the tube with a nearly clear medium above whereas the tube at 25°C showed nearly uniform turbidity.

To determine calcium dependency for growth (at 37°C), a procedure similar to that described by Agbonlahor (1986) was used. Magnesium oxalate agar was prepared by adding 2.68 g of sodium oxalate and 4.067 g of magnesium chloride to 1l of typtic soy agar (TSA). The final pH was adjusted to 7.6.

Cultures of Y. enterocolitica were streaked on DCA plates and incubated for 24 h at room temperature (25°C) to obtain discrete colonies. Five individual colonies of each isolate were picked into 2 ml of phosphate buffered saline (PBS) and then diluted to 10^{-2}. The resulting cell suspensions served as inocula. Two plates comprising magnesium oxalate agar (MOA) and tryptic soy agar (TSA) were used for each isolate. Five drops of each of the cell suspensions were spotted onto five separate areas using sterile Pasteur pipettes and the plates were allowed to dry. The plates were then inverted and incubated at 37°C for 24 h.

Growth on TSA without growth on MOA or heavy growth on TSA and few colonies on MOA was taken as evidence that the particular colony selected from desoxycholate citrate agar plates was calcium-dependent for growth at 37°C.

The same sereny test for invasiveness performed for E. coli (modification of Mosimabale and Gyles, 1982 procedure) was utilised.

A slight modification of the heat stable (ST) enterotoxin procedure described by Mosimabale and Gyles (1982) was performed. Twenty millilitres of tryptic soy broth (TSB) were each dispensed into 250 ml Erlenmeyer flasks and sterilised at 121°C for 15 minutes. The broth was supplemented with millipore filtered 0.6% yeast extract. Overnight incubation of test cultures and controls in TSB at room temperature served as inocula. Flasks containing inoculated TSB were incubated on a gyratory shaker (6.835xg) for 18 h at 25°C in a chamber with temperature regulation. Growth was then spun down in a refrigerated centrifuge (4°C) for 20 minutes at 17,000 xg and the supernatent millipore-filtered (0.45u). The supernatent was subsequently heated for 30 minutes at 65°C in a water bath, cooled and frozen at -20°C until needed.

The procedure of Nunes and Ricciardi (1981) for the detection of ST enterotoxin using the infant mouse system was used. Heat-stable enterotoxin was detected in culture filtrates earlier prepared by

adding a drop of 1% Evans blue dye in normal saline to approximately 6 ml of supernatent. Each members of a group of 3 day-old suckling mice which were fed with milk (to make the stomach visible) was injected with 0.1 ml of the coloured supernantant of each isolate. The mice were held at room temperature ($25^{o}C$) for 4 h and then sacrificed using chloroform. The intestines of the three mice per group were removed, pooled and weighed after the body weight had been determined. The ratio of the gut weight values of 0.085 or greater were considered as positive as recommended by Gyles (1979).

A modified method of Schiemann (1981) for mouse diarrhoeal model was used. The test isolates and controls E. coli (K12) were grown on tryptic soy broth (TSB) overnight at room temperature after which the cultures were plated unto tryptic soy agar plates and incubated overnight at room temperature. The cells were harvested in 1 ml of saline and 10^{9} c.f.u. per mouse was given orally to 9 young mice and they were observed daily for 1 week for signs of diarrhoea. The presence of diarrhoea in the young mice is indicative of enteropathogenicity of the isolate.

Isolation and Characterisation of Salmonella spp:

The rectal swabs obtained were enriched in selenite - F broth (Oxoid) and incubated at 37°C for 24 h. Subcultures were made unto DCA plates and incubated at 37°C for 24 h before examination. Desoxycholate citrate agar plates showing no growth after 24 h were incubated for a further 24 h. Non-lactose fermenting colonies were inoculated unto tripe sugar iron (TSI) slants. Isolates that produced alkaline/acid, hydrogen sulphide and gas, or alkaline/acid alone were subjected to confirmatory tests with urea, Simmon's citrate, methyl-red, Voges-Proskauer, indole and motility media; fermentation tests with mannitol, dulcitol, maltose, rhamnose, raffinose, glucose and lactose sugars; nitrate reduction test, gelatin liquefaction test; reactions with ornithine, lysine, arginine and potassium cyanide (MacFaddin, 1977).

Isolates that were urease, indole and Voges-Proskauer negative and citrate, methyl-red, and nitrate reduction positive, fermented all sugars except raffinose, lactose and sucrose and were motile at 37°C, failed to grow in potassium cyanide, lysine decarboxylase positive and ornithine and arginine negative, were considered to belong to the genus Salmonella.

Serological tests were performed by the slide agglutination test, using Salmonella polyvalent antisera A-E. All the positive isolates were confirmed and their antigenic formulae determined by Prof. L. Le Minor of the Pasteur Institute, Paris.

To determine the antibiogram of Salmonella isolates, the same procedure was performed as was done for Escherichia coli isolates, using the disc diffusion method; same procedure was used as for Yersinia enterocolitica with the agar plate dilution method.

Faecal Examination for Helminth Ova and Larvae, and Protozoan Oocyst:

This was done according to Whitlock's method (1960). To do a direct smear examination, a small quantity of each faecal sample was added to a centrifuge tube containing a small amount of tap water. The faeces was thoroughly mixed with the water using a glass rod. A small amount of the suspension was pipetted unto a glass slide and smear made and examined under the microscope (x 10) for helminth ova, coccidia and Giardia canis oocysts.

To do the floatation technique (concentration method), a small quantity of each faecal sample was thoroughly mixed with a small amount of floatation fluid (150g sucrose plus 400g zinc sulphate per litre of tap water) in a centrifuge tube. The suspension was then filtered through layers of gauze into a plastic tube. More fluid was added until a meniscus was formed; a cover slide was then placed on the meniscus and left to stand for five minutes after which it was removed, placed on a slide and examined for helminth ova, coccidia and Giardia canis oocysts under the microscope. To quantify the ova or oocyst of each sample, the following graduations were used:

+	=	1 ova or oocyst/field of microscope (x10) objective
++	=	3-5 " " " " " " "
+++	=	15-25 " " " " " " "
++++	=	25 " " " " " " "

To do the Baerman's modified technique for detection of Living Larvae, the faecal samples were ground with a pestle and mortar to a moderately soft consistency, packed in a culture bottle and kept in a dark cupboard at room tempterature for 1 week. After 7 days, the culture bottle was taken out and

placed under a bright bulb (60 watts) for thirty minutes to stimulate the larvae (L_3). The bottle was then filled with water and a petri-dish placed on its top, then placed upside down. Water was introduced into the petri-dish and the larvae by gravitational pull sink into the petri-dish and were collected into centrifuge tubes via a Pasteur pipette and rubber teat. The larvae were then sedimented by centrifuging at 26.553 x g for 3-5 minutes. The sediments were examined under the microscope for larval identification.

Analysis of Data:

The data were analysed by the use of chi- quare tests with a computer at the data processing unit of the National Animal Production Research Institute, Shika. A p value of greater than 5% or more (i.e. $p>0.05$) was considered statistically non-significant.

2.3 Results

2.3.1 Retrospective Study

Of the 1,876 dogs evaluated during the 2-year-period of study only 126 were presented with diarrhoea. The results indicate that the prevalence of diarrhoea was 6.72%. The incidence was highest during the month of March (Table 2.2) but there was no significant seasonal variation ($P>0.05$).

Younger dogs (1-6 months old) were more commonly affected (Table 2.3) and age was a statistically significant factor ($P<0.05$). The incidence of diarrhoea was also higher in the Nigerian local breed of dogs (7.94%) and Alsatians (7.71%) when compared to the Cross breeds (2.96%) (Table 2.4); this difference was statistically significant ($P<0.05$). The results indicate no significant difference in the incidence of diarrhoea between male (6.67%) and female (6.68%) dogs (Table 2.5).

Table 2.2: Monthly Incidence of Diarrhoea in Dogs that Attended ABUVTH, Zaria

Month	Total No. of Dogs	No. of Dogs Diarrhoeic (%)
1984		
January	61	6 (9.84)
February	78	7 (8.97)
March	106	15 (14.15)
April	92	4 (4.35)
May	86	4 (4.65)
June	94	8 (8.51)
July	66	3 (4.55)
August	83	1 (1.20)
September	62	1 (1.61)
October	51	1 (1.96)
November	50	2 (0.04)
December	61	4 (6.56)
1985		
January	69	0 (0.00)
February	67	2 (2.98)
March	127	17 (13.38)
April	116	9 (7.76)
May	114	8 (7.02)
June	111	15 (13.51)
July	67	4 (5.97)
August	60	1 (1.66)
September	67	5 (7.46)
October	68	4 (5.88)
November	65	3 (4.61)
December	55	2 (3.64)
Total	1,876	126 (6.72)

Table 2.3: Age Distribution of Dogs with Diarrhoea that Attended ABUVTH, Zaria

Age Group* (Months)	Total No. of Dogs	No. of Dogs Diarrhoeic (%)
≤6 - 6	534	86 (16.10)
7 - 12	406	11 (2.71)
13 - 18	219	7 (3.20)
19 - 24	152	3 (1.97)
25 - 30	145	4 (2.76)
31 - 36	141	0 (0.00)
≥36	279	15 (5.38)
Total	1,876	126 (6.72)

* Dogs ≥6 months old were considered in the 7-12 months group; dogs ≥12 months old, in the 13-18 months groupl dogs ≥18 months old, in the 19-24 months group; dogs ≥24 months old, in the 25-30 months groups; and dogs ≥30 months old, in the 31-36 months group.

Table 2.4: Breed Distribution of Dogs with Diarrhoea that Attended ABUVTH, Zaria

	Breed			Total
	Local	Alsation	Cross breeds	
Total No. of Dogs	970	467	439	1,876
No. of Dogs Diarrhoeic	77	36	13	126
Percentage Diarrhoeic	7.94	7.71	2.96	6.72

Table 2.5: Sex Distribution of Dogs with Diarrhoea that Attended ABUVTH, Zaria

	Sex		
	Male	Female	Total
Total No. of Dogs	1139	737	1,876
No. of Dogs Diarrhoeic	76	50	126
Percentage Diarrhoeic	6.67	6.78	6.72

2.3.2 Clinical Study

Incidence of Diarrhoea:

From a total of 1,072 of dogs that attended the ABUVTH Small Animal Clinic during the 18-month-period of study, 83 dogs were presented with diarrhoea (Table 2.6). During the period, 72 apparently healthy dogs were sampled; these dogs served as controls for the diarrhoeic dogs. The results indicate that the prevalence of diarrhoea was 7.7% while the incidence was highest during the month of March. Dogs 1-6 months old were more commonly affected (Table 2.7) which was highly statistically significant ($P<0.001$). The incidence was also higher in Nigerian local dogs (62.7%) when compared to the Alsatians (28.9%) or crossbreeds (8.4%) (Table 2.8) and this was statistically significant ($P<0.001$). The occurrence of diarrhoea was more common in male dogs (56.6%) than in females (43.4%) but the difference was not statistically significant ($P>0.05$) (Table 2.8).

Table 2.6: Monthly Incidence of Diarrhoea in Dogs that Attended ABUVTH, Zaria

Month	Number of Dogs
1986	
March	12
April	8
May	4
June	10
July	3
August	0
September	4
October	3
November	3
December	2
1987	
January	4
February	6
March	14
April	2
May	3
June	2
July	3
August	0
Total	83

Table 2.7: Age Distribution of 83 Dogs with Diarrhoea that Attended ABUVTH, Zaria

Age Group* (Months)	Number of Dogs (%)
≤6 - 6	55 (66.3)
7 - 12	9 (10.8)
13 - 18	5 (6.0)
19 - 24	1 (1.2)
25 - 30	1 (1.2)
31 - 36	2 (2.4)
>36	10 (12.1)
Total	83 (100%)

* Dogs ≤6 months old were considered in the 7-12 months group; dogs >12 months old, in the 13-18 months group; dogs >18 months old, in the 19-24 months group; dogs >24 months old, in the 25-30 months group; and dogs >30 months old, in the 31-36 months group.

Table 2.8: Breed and Sex Distribution of 83 Dogs with Diarrhoea that Attended ABUVTH , Zaria

	Breed			Sex	
	Local	Alsatian	Mixed	Male	Female
No. of Dogs	52	24	7	47	36
(Percentage)	(62.7)	(28.9)	(8.4)	(56.6)	(43.4)
Total	83 (100)			83(100)	

Detection of Pathogens in Faeces:

A majority of the dogs (both diarrhoeic and apparently healthy) were positive for both bacterial and intestinal parasitic infections (Table 2.9). A few dogs had more than one bacterial or parasitic pathogen. Eschericia coli was the predominant bacterial pathogen isolated from both diarrhoeic and apparently healthy dogs (Table 2.10) while Ancylostoma was the most common intestinal parasite in both groups of dogs (Table 2.9). Bacterial isolation was significantly ($P<0.05$) more common in diarrhoeic than in apparently healthy dogs (Table 2.10).

Isolation of E. coli:

Escherichia coli was isolated from 69 (83.1%) diarrhoeic dogs and 53 (73.6%) apparently healthy dogs (Table 2.11) but the difference was not statistically significant ($P>0.05$). The isolation rate from diarrhoeic dogs was higher in younger dogs (66.7%) (66.7%) than in older dogs (14.5%) while inapparent infections were common in both young and older dogs (Table 2.12). The difference was statistically significant ($P<0.001$).

Table 2.9: Incidence of Bacterial and Parasitic Isolation from faeces of 83 Diarrhoeic and 72 Apparently. Healthy Dogs that Attended ABUVTH , Zaria

Parasitic - Bacterial Agents(s)	Number of Dogs (%) Diarrhoeic	Apparently Healthy
Ancylostoma spp. + Taenia spp. + E. coli	1(1.2)	1(1.4)
Ancylostoma spp. + Toxocara spp.	0(0.0)	2(2.8)
Ancylostoma spp. + E. coli	5(6.0)	7(9.7)
Toxocara spp. + E. coli	3(3.6)	1(1.4)
Taenia spp. + E. coli	6(7.3)	5(6.9)
Trichuris spp. + E. coli	1(1.2)	0(0.0)
Isospora spp. + E. coli	5(6.0)	0(0.0)
Ancylostoma spp. + E. coli + Salmonella spp.	2(2.4)	0(0.0)
Ancylostoma spp. + Toxocara spp. + E. coli	0(0.0)	3(4.2)
Isospora spp. alone	2(2.4)	0(0.0)
E. coli + Salmonella spp.	5(6.0)	2(2.8)
E. coli alone	41(47.5)	31(43.0)
Salmonella spp. alone	4(4.8)	3(4.2)
Toxocara spp. + E. coli + Yersinia spp.	0(0.0)	1(1.4)
Dipylidium spp. + E. coli	0(0.0)	1(1.4)
Ancylostoma spp. + Isospora	0(0.0)	1(1.4)
Strongyloides spp. + E. coli	0(0.0)	1(1.4)
No pathogen	8(9.6)	3(18.0)
Total	83(100)	72(100)

Table 2.10: Incidence of Bacterial Isolation from Faeces of 83 Diarrhoeic and 72 Apparently Healthly Dogs that Attended ABUVTH, Zaria

Bacterium	Number of Dogs (%)	
	Diarrhoeic	Apparently Healthly
Escherichia coli alone	63(75.9)	50(69.4)
Salmonella spp. alone	9(10.8)	5(7.0)
Escherichia coli+ *Salmonella* epp.	6(7.3)	2(2.8)
Yersinia spp. + *E. coli*	0(0.0)	1(1.4)
None[a]	5(6.0)	14(19.4)
Total	83(100)	72(100)

[a]None positive for *Salmonella* spp., *E. coli* and *Yersinia* spp.

Table 2.11: Monthly Incidence of Escherichia coli Isolation from 83 Diarrhoeic and 72 Apparently Healthy Dogs that Attended ABUVTH, Zaria

Month	Diarrhoeic Dogs No. of Samples	Diarrhoeic Dogs No. with E. coli (%)	Apparently Healthy Dogs No. of Samples	Apparently Healthy Dogs No. with E. coli (%)
1986				
March	12	11(91.7)	21	20(95.2)
April	8	6(75.0)	8	8(100.0)
May	4	4(100.0)	5	4(80.0)
June	10	7(70.0)	2	0(0.0)
July	3	3(100.0)	2	1(50.0)
August	0	0(0.0)	2	0(0.0)
September	4	4(100.0)	0	0(0.0)
October	3	1(33.3)	8	6(75.0)
November	3	3(100.0)	0	0(0.0)
December	2	2(100.0)	1	1(100.0)
1987				
January	4	4(100.0)	2	2(100.0)
February	6	6(100.0)	10	5(50.0)
March	14	10(71.4)	3	2(66.7)
April	2	2(100.0	2	0(0.0)
May	3	2(66.7)	1	1(100.0)
June	2	1(50.0)	4	2(50.0)
July	3	3(100.0)	0	0(0.0)
August	0	0(0.0)	1	1(100.0)
Total	83	69(83.1)	72	53(73.6)

Table 2.12: Age Distribution of 69 Diarrhoeic and 53 Apparently Healthy Dogs that Attended ABUVTH, Zaria from the Faeces of which *E. coli* was isolated

Age Group** (Months)	No. of Dogs with *E. coli* infection Diarrhoeic (%) (n = 83)*	Apparently Healthy (%) (n = 72)*
4.6 - 6	46(66.7)	17(32.1)
7 - 12	8(11.6)	7(13.2)
13 - 18	3(4.4)	4(7.5)
19 - 24	1(1.4)	3(5.7)
25 - 30	0(0.0)	3(5.7)
31 - 36	1(1.4)	0(0.0)
> 36	10(14.5)	19(35.8)
Total	69(100)	53(100)

*n = sample size

** = Dogs >6 months old were considered in the 7-12 months group; dogs >12 months old, in the 13-18 months group; dogs >18 months old, in the 19-24 months group; dogs >24 months old, in the 25-30 months group; and dogs >30 months old, in the 31-36 months group.

Of the 49 E. coli isolates serotyped, 26 were from diarrhoeic dogs and 10 were from apparently healthy dogs; 13 of these isolates were untypable. Of the 26 strains isolated from diarrhoeic dogs, a total of 25 different serotypes were obtained indicating diversity instead of predominance of serotypes (Table 2.13). Ten of the 20 E. coli strains from diarrhoeic dogs tested were positive for invasiveness (Table 2.14); all these strains were negative for enterotoxin production.

Of the 47 E. coli isolates from diarrhoeic dogs tested for heat labile (LT) enterotoxigenicity, 10 (14.5%) were positive and of the 22 isolates from apparently healthy dogs tested, 13(18.8%) were positive (Table 2.15). From the 13 E. coli isolates from diarrhoeic dogs tested for heat stable (ST) enterotoxin production, 2(10%) were positive and of the 7 isolates from apparently healthy dogs tested, 3(15%) were positive (Table 2.15). Overall, from the 69 strains tested for LT enterotoxin production 23(33.3%) were positive and of the 20 tested for ST enterotoxin production, 5(25%) were positive. No significant differences were found between LT and St enterotoxin production.

Table 2.13: Serotypes and Antigenic Formulae of 49 Escherichia coli strains isolated from the Rectum of dogs that Attended ABUVTH, Zaria

Diarrhoeic Dogs*		Apparently Healthy Dogs**	
Isolate No.	Antigenic Formula	Isolate No.	Antigenic Formula
D - 1	087:H-	H - 1(a)	0104:H7
D - 2	08:K87:H10	H - 1(b)	04:K?:H16
D - 3	04:K2:H5	H - 2	0132:H32
D - 4	07:K?:H4	H - 3	08:K40:H11
D - 5	08:K34:H2	H - 4	04:K7:H-
D - 6	079:H7	H - 5	018ac:K?H?
D - 7	0103:H11	H - 6	083:K24
D - 8	09:H9	H - 7	015:K?
D - 9	0146:K-	H - 8	08:K (A)?
D - 10	010:K?	H - 9	08:K46
D - 11	054:K?	H - 10	0125:K-
D - 12	08:K?		
D - 13	011:K?-		
D - 14	08:K-		
D - 15	083:K-		
D - 16	0116:K?		
D - 17	018:K?		
D - 18	0170:K?		
D - 19	0153:K-		
D - 20	09:K(A) 34		
D - 21	012:K?		
D - 22	086:K?		
D - 23	062:k		
D - 24	083:K-		
D - 25	070:K?		
D - 26	08:K49		

* Eight strains were untypable

** Five strains were untypable

Table 2.14: Frequency of Occurrence of Invasiveness in 20 Escherichia coli isolates from Diarrhoeic dogs that were heat-labile (LT) and heat-stable (ST) Enterotoxins Negative

Isolate Number	Serotype	Invasive Test Result
D - 36	N.D.	-
D - 29	N.D.	+
D - 37	N.D.	-
D - 30	N.D.	-
D - 6	079:H7	+
D - 1	087:H-	+
D - 32	N.D.	-
D - 13	011:k	+
D - 10	010:K?	-
D - 34	N.D.	-
D - 35	N.D.	-
D - 11	054:K?	-
D - 28	Untypable	+
D - 17	018:K?	+
D - 24	083:K-	+
D - 33	N.D.	+
D - 31	N.D.	+
D - 7	0103:H11	+
D - 27	Untypable	-
D - 22	086:K?	-

N.D. - Not serotyped

Table 2.15: Frequency of Occurrence of heat-labile (LT) and heat-stable (ST) Enterotoxin Production in Escherichia coli isolates from Dogs that Attended ABUVTH, Zaria

Status of Dogs	No. of Isolates	LT Production No. Tested	LT Production No. Positive (%)	ST Production No. Tested	ST Production No. Positive (%)
Diarrhoeic	69	47	10(14.5)	13	2(10.0)
Apparently Healthy	53	22	13(18.8)	7	3(15.0)
Total	122	69	23(33.3)	20	5(25.0)

LT - Heat-labile enterotoxin

ST - Heat-Stable enterotoxin

Of the 53 E. coli isolates tested from apparently healthy dogs and 69 isolates from diarrhoeic dogs, 53(100%) and 69(100%) respectively were resistant to 1 or more of the 11 antimicrobial agents (Table 2.16): The drug to which majority of the isolates were susceptible to was gentamicin (Table The most common resistance was associated with methicillin, penicillin, sulphadiazine and erythromycin (Table 2.17). A total of 43 resistance patterns were observed.

Isolation and Characterisation of Yersinia enterocolitica:

From the 83 diarrhoeic and 72 apparently healthy dogs sampled, Yersinia enterocolitica was isolated from only 1 apparently healthy dog. It was of the serotype O:8; it was negative for heat stable enterotoxin, calcium dependency at 37°C, invasiveness, and autoagglutination at 37°C. The isolate was highly susceptible to gentamicin, tetracycline and neomycin but resistant to erythromycin and streptomycin.

Table 2.16: The Incidence of Resistance of the Escherichia coli isolated from both Diarrhoeic and Apparently Healthy Dogs to the various antimicrobial agents

Antimicrobial Agent	Concentration	No. (%) of Resistant E. coli isolates	
		Diarrhoeic Dogs (n=69)*	Apparently Healthy Dogs (n=53)*
Methicillin	5 mcg	69(100)	53(100)
Penicillin	10 I.U.	69(100)	52(98.1)
Sulphadiazine	30 mcg	68(98.6)	48(90.6)
Erythromycin	15 mcg	65(94.2)	51(96.2)
Streptomycin	10 mcg	44(63.8)	32(60.4)
Tetracycline	30 mcg	36(52.2)	31(58.5)
Ampicillin	10 mcg	29(42.1)	20(37.7)
Kanamycin	30 mcg	26(37.7)	13(24.5)
Neomycin	30 mcg	14(26.4)	17(24.6)
Chloramphenicol	30 mcg	16(23.2)	16(30.2)
Gentamicin	10 mcg	15(21.7)	7(13.2)

I.U. = International Units

*n = Sample size

Table 2.17: Predominant Patterns of Antimicrobial Resistance of E. coli Isolates from the 155 dogs Sampled

Resistance Pattern [a,b]	No. of Isolates
M, P, SD, E	20
M, T, P, SD, S, E	9
M, T, P, S, E	8
M, P, SD, S, E	7
A, M, T, P, SD, S, E	6
M, N, T, P, SD, S, K, E, G	5
A, M, N, T, P, SD, S, K, E, C	4
A, M, N, T, P, SD, S, K, E, G, E	4
A, M, N, T, P, SD, S, K, E	4
A, M, T, P, SD, S, K, E, G	3
A, M, P, SD, S, E	3
M, P, SD,	3
M, T, P, SD, S, E, C	3
A, M, T, P, SD, S, K, E, C	3
A, M, T, P, SD, S, E, C	3
M, P, SD, K, E, G	2
M, P, SD, S, E, E,	2
A, M, P, SD, S, K, E	2
A, M, T, P, SD, E,	2
A, M, N, P, SD, S, K, E, C,	2
M, T, P, SD,	2
M, N, P, SD, S, E,	2
A, M, N, T, P, SD, S, E, C	2
M, P, E	2
None of the above [c]	19

[a] Overall, 43 antimicrobial resistance patterns were observed

[b] A = Ampicillin; M = Methicillin; N = Neomycin; T = Tetracycline; P = Penicillin; SD = Sulphadiazine; S = Streptomycin; K = Kanamycin; E = Erythromycin; G = Gentamicin; and C = Chloramphenicol

Isolation and Characterisation of Salmonella species:

Salmonella spp. were isolated from 9 diarrhoeic (10.8%) and 5 apparently healthy dogs (7%) (Table 2.18). The difference in the isolation rate between diarrhoeic and healthy dogs was not statistically significant ($P>0.05$). Ancylostoma caninum was also detected in 3 of the 9 diarrhoeic dogs and in 1 of the 5 apparently healthy dogs. Escherichia coli was isolated too from 6 of the 9 diarrhoeic dogs and from 2 of the 5 apparently healthy dogs. The prevelence of Salmonella spp. in the canine population was 9.6%.

Isolation from diarrhoeic dogs was more frequent in dogs 1-6 months old (88.9%) than in dogs 36 months old (0%) and above, while inapparent infections were more common in older dogs (Table 2.19); this age factor was highly statistically significant ($P<0.001$).

The Salmonella spp. isolated were from swabs from the rectum (11), beddings (1), mesenteric lymph nodes (1) and liver (2). Two dogs had more than one Salmonella serotype (Table 2.20).

Table 2.18: Incidence of Diarrhoea and *Salmonella* Isolation from Diarrhoeic and Apparently Healthy Dogs that Attended ABUVTH, Zaria

Month	Diarrhoeic dogs		Apparently Healthy dogs	
	No. of samples	No. with *Salmonella*	No. of Samples	No. with *Salmonella*
1986				
March	12	1	21	0
April	8	1	8	0
May	4	0	5	0
June	10	1	2	0
July	3	0	2	2
August	0	0	2	2
September	4	0	0	0
October	3	3	8	1
November	3	0	0	0
December	2	0	1	0
1987				
January	4	0	2	0
February	6	0	10	0
March	14	1	3	0
April	2	1	2	0
May	3	1	1	0
June	2	0	4	0
July	3	0	0	0
August	0	0	1	0
Total	83	9	72	5

Table 2.19: Age Distribution of 155 Dogs that Attended ABUVTH, Zaria from which *Salmonella* spp. were isolated

Age Group** (Months)	No. of dogs with Salmonella infection Diarrhoeic dogs (%) (n = 83)*	Apparently Healthy Dogs (%) (n = 72)*
<6 - 6	8(88.9)	0(0.0)
7 - 12	0(0.0)	1(20.0)
13 - 18	0(0.0)	0(0.0)
19 - 24	0(0.0)	1(2.0)
25 - 30	0(0.0)	0(0.0)
31 - 36	0(0.0)	0(0.0)
>36	1(11.1)	3(60.0)
Total	9(100)	5(100)

* n = sample size

** = Dogs >6 months old were considered in the 7-12 months group; dogs >12 months old, in the 13-18 months group; dogs >18 months old, in the 19-24 months groupl dogs >24 months old, in the 25-30 months group; and dogs >30 months old, in the 31-36 months group.

Table 2.20: Serotypes and Antigenic formulae of Salmonella strains isolated from Dogs and their Beddings that Attended ABUVTH, Zaria

Isolate No.	Source	Serotype	Antigenic Formulae
Diarrhoeic Dogs			
D - 1	Rectum	S. schwarzengrund	1:4, 12:d:1,7
D - 2	Rectum	S. hull	16, 6:1,2
D - 3	Rectum	S. colindale	6, 7:r:1,7
D - 4	Mesenteric lymph node**	S. tabligo	47:Z_4, Z_{23}:e,n,Z_{15}
D - 5	Liver**	S. ona*	28:g,s,t,
D - 6	Liver**	S. tabligo*	47:Z_4,Z_{23}:e,n,Z_{11}
D - 7	Rectum	S. hoghton	3,10:1,Z_{18},Z_{23}:-
D - 8	Rectum	S. poona	13,22:Z:1,6
D - 9	Rectum	S. stanleyville	1,4,5,12:Z_4,Z_{23}
Apparently Healthy Dogs			
H - 1	Rectum	S. schwarzengrund	1;4,12.1,7
H - 2	Rectum	S. schwarzengrund	1;4,12:d:1,7
H - 3	Beddings	S. koketime	1,44:Z_{38}:var 01+
H - 4*	Rectum	S. omifisan	40:Z_{29}:-
H - 5*	Rectum	S. wilhelmsburg	1,4,12:Z_{38}:-
H - 6	Rectum	S. rubislaw	11:r:e,n,x

*Isolates obtained from the same dog.

**Source of material was from necropsy.

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Table 2.21: Antibiograms of *Salmonella* isolates from dogs that Attended ABUVTH, Zaria

Isolate No.	Serotype	*Minimal Inhibitoy concentration (MIC, ug/ml) of Antimicrobial Agent											No of D to whi isolat resist
		Chlor.	Gent.	Tega.	Enyth.	Strept.	Amp.	Sulp	Linc.	Tetr.	NA	Neo.	
Diarrhoeic Dogs													
D - 1	S. schwarzengrund	32R	<0.25S	8S	>128R	8S	0.50S	>128R	>128R	2S	128R	2s	5
D - 2	S. hull	64R	<0.25S	8S	>128R	128R	0.25S	>128R	>128R	128R	128R	2S	7
D - 3	S. colindale	64R	0.25S	8S	>128R	8S	2.00S	>128R	>128R	128R	128R	2S	6
D - 4	S. tabligo	64R	<0.25S	16R	>128R	8S	2.00S	>128R	>128R	2S	128R	2S	6
D - 5	S. ona	64R	<0.25R	16R	>128R	8S	0.25S	>128R	>128R	2S	128R	2S	6
D - 6	S. tabligo	64R	<0.25R	16R	>128R	4S	0.25S	>128R	>128R	2S	128R	2S	6
D - 7	S. hoghton	128R	<0.25S	128R	>128R	32R	2.00S	>128R	>128R	2S	32R	16S	7
D - 8	S. poona	128R	<0.25S	128R	>128R	64R	2.00S	>128R	>128R	1S	8S	16S	6
D - 9	S. Stanleyville	128R	<0.25S	128R	>128R	32R	2.00S	>128R	>128R	1S	>128R	16S	7
Apparently Healthy Dogs													
H - 1	S. schwarzengrund	32R	<0.25S	8S	>128R	8S	0.50S	>128R	>128R	2S	128R	2S	5
H - 2	S. schwarzengrund	32R	<0.25S	8S	>128R	2S	0.50S	>128R	>128R	2S	128R	2S	5
H - 3	S. koketime	32R	<0.25S	4S	>128R	2S	128R	>128R	>128R	4S	128R	2S	6
H - 4	S. omifisan	32R	<0.25S	8S	>128R	8S	0.25S	>128R	>128R	4S	128R	4S	5
H - 5	S. wilhelmsburg	32R	<0.25S	8S	>128R	8S	0.25S	>128R	>128R	4S	128R	4S	5
H - 6	S. rubislaw	32R	<0.25S	8S	>128R	8S	0.50S	>128R	>128R	2S	128R	2S	5

Chlor. = Chloramphenicol, Gent. = Gentamicin, Toga. = Togamycin, Eryth. = Erythromycin, Strept. = Streptomycin, Amp. = Ampicillin, Sulph. = Sulphadimidine, Linc. = Lincomycin, Tetr. = Tetracycline, N.A. = Nalidixic Acid, Neo. = Neomycin.

*R = Resistant to the antimicrobial agent

S = Sensitive to the antimicrobial agent

Table 2.22: Antibiogram of Salmonella Isolates from Dogs that attended ABUVTH, Zaria determined by the Disc Method

Isolate No.	Serotype	*Antimicrobial Agent and zone size (mm)										No. of Drugs to which isolate is resistant
		Chlor.	Pen.	Strept.	Amp.	Gent.	Eryth	Meth.	Kan.	Tetra.	Neo.	
Diarrhoeic dogs												
D - 1	S. schwarzengrund	28S	11R	15R	20S	23S	12R	0R	21s	16S	20S	3
D - 2	S. hull	29S	13S	0R	22S	23S	13S	0R	29S	0R	22s	3
D - 3	S. colindale	22S	11R	12S	19S	20S	10R	0R	21S	0R	18S	4
D - 4	S. tabligo	26S	12S	15S	21S	22S	13S	0R	21S	20S	19S	1
D - 5	S. ona	26S	12S	14S	21S	22S	11R	0R	22S	16S	19S	2
D - 6	S. tabligo	27S	13S	15S	21S	22S	13S	0R	22S	19S	21S	1
D - 7	S. houghton	25S	13S	17S	22S	18S	0R	0R	22S	20S	15S	2
D - 8	S. poona	25S	7R	10R	20S	20S	0R	0R	22S	25S	20S	4
D - 9	S. stanleyville	25S	9R	10R	22S	20S	0R	0R	20S	25S	22S	4
Apparently healthy dogs												
H - 1	S. schwarzengrund	21S	11R	16S	21S	21S	12R	0R	22S	19S	18S	3
H - 2	S. schwarzengrund	28S	13S	19S	21S	22S	11R	0R	23S	19S	22S	2
H - 3	S. koketime	26S	0R	20S	12S	22S	18S	0R	22S	20S	21S	2
H - 4	S. omifisan	24S	13S	15S	21S	21S	12R	0R	22S	17S	20S	1
H - 5	S. wilhelmsburg	24S	13S	15S	21S	21S	12R	0R	22S	17S	20S	2
H - 6	S. rubislaw	28S	14S	12S	24S	21S	14S	0R	21S	18S	19S	1

Chlor = Chloramphenicol, Gent. = Gentamicin, Pen. = Penicillin, Strept. = Streptomycin, Amp. = Ampicillin, Eryth. = Erythromycin, Meth. = Methicillin, Kan. = Kanamycin, Tetra. = Tetracycline, Neo. = Neomycin.

*R = Resistant to the antimicrobial agent
S = Sensitive to the antimicrobial agent

Table 2.23: Incidence of helminth ova in Faeces of Diarrhoeic and Apparently Healthy Dogs that Attend ed AB U V TH, Zaria

Helminth	Number of Dogs(%) Diarrhoeic (n = 83)*	Apparently Hea lthy (n = 72)*
Ancylostoma spp. alone	7(8.4)	8(11.1)
Taenia spp. alone	8(9.6)	5(6.9)
Toxocara spp. alone	3(3.6)	2(2.8)
Dipylidium spp. alone	0(0.0)	1(1.4)
Trichuris spp. alone	1(1.2)	0(0.0)
Spirocerca spp. alone	1(1.2)	0(0.0)
Strongyloides spp. alone	9(0.0)	1(1.4)
Ancylostoma spp. + Taenia spp.	1(1.2)	1(1.4)
Ancylostoma spp. + Toxocara spp.	0(0.0)	3(4.2)
Total	21(25.3)	21(29.2)

*n = Number of dogs examined

Table 2.24: Seasonal Incidence of Helminth ova Detection in Faeces of Diarrhoeic and Apparently Healthy Dogs that Attended ABUVTH, Zaria

	Diarrhoeic Dogs		Apparently Healthy Dogs	
	No. of samples	No. with helminths	No. of samples	No. with helminths
1986				
March	12	3	21	9
April	8	4	8	2
May	4	1	5	2
June	10	2	2	1
July	3	0	2	0
August	0	0	2	0
September	4	1	0	0
October	3	0	8	5
November	3	1	0	0
December	2	2	1	0
1987				
January	2	2	0	
February	6	0	10	2
March	14	2	3	0
April	2	2	2	0
May	3	1	1	1
June	2	0	4	1
July	3	0	0	0
August	0	0	1	1
Total	83	21	72	24

Table 2.25: Helminth ova Counts in Faeces of 21 Diarrhoeic and 24 Apparently Healthy Dogs that Attended ABUVTH, Zaria

Helminth	Ova Count*	
	Diarrhoeic Dogs (n = 21)**	Apparently Healthy Dogs (n = 24)**
Ancylostoma spp.	++ to +++	+ to ++
Toxocara spp.	+++	+ to ++
Taenia spp.	+ to ++	+ to ++
Trichuris spp.	+	0
Spirocerca spp.	+	0
Strongyloides spp.	0	+
Dipylidium spp.	0	++

* + = 1 ova/microscope field (x 10 objective)
++ = 3-5 ova/microscope field (x 10 objective)
+++ = 15-25 ova/microscope field (x 10 objective)
++++ = 25 ova/microscope field (x 10 objective)

** n = sample size

Nine diarrhoeic and 2 apparently healthy dogs had Isospora canis infection. The oocyst count for the diarrhoeic dogs ranged from ++ (i.e. 3 to 5 oocysts per microscope field) to +++ (i.e. 15 to 25 oocyst per microscope field) while the count for apparently healthy dogs was + (i.e. 1 oocyst per microscope field).

The frequency of occurrence of ova oocysts in the faeces of diarrhoeic and apparently healthy dogs was highly dependent on age group ($P<0.001$), particularly in dogs 1-6 months of age and in dogs 36 months of age or older (Table 2.26) the difference between diarrhoeic and apparently healthy dogs of the same age group was not statistically significant ($P>0.05$).

Table 2.26: Age Distribution of dogs that Attended ABUVTH, Zaria with Parasitic Infections

Age Group**	Number of Dogs % Diarrhoeic Dogs (n = 83)*	Apparently Healthy Dogs (n = 72)*
<6 - 6	22(26.5)	12(16.7)
7 - 12	2(2.4)	3(4.2)
13 - 18	1(1.2)	1(1.4)
19 - 24	0(0.0)	1(1.4)
25 - 30	0(0.0)	1(1.4)
31 - 36	0(0.0)	1(1.4)
> 36	4(4.8)	5(6.9)
Total	29(34.9)	

* n = sample size

** = Dogs 76 months old were considered in the 7-12 months group; dogs >12 months old, in the 13-18 months group; dogs >18 months old, in the 19-24 months group; dogs >24 months old, in the 25-30 months group; and dogs >30 months old, in the 31-36 months group.

2.4 Discussion

The observation that the results of both restrospective and clinical studies were similar was not unexpected. However, the low incidence of diarrhoea observed in both studies is unexpected when the number of potential causes of diarrhoea encountered in the dogs is considered. The relatively higher incidence in the month of March cannot easily be explained; diarrhoea is expected to be more common during the rainy season (April to September) when helminth and protozoal infections are more common, as observed by Oduye and Olayemi (1977). The observation that greater than 70% of the diarrhoeic dogs were one year old or younger agrees with the general observation that young dogs are more commonly affected by diarrhoea (Strombeck, 1979; Murdoch, 1986). The apparent higher incidence of diarrhoea in local Nigerian dogs as compared to exotic breeds or their crosses in the clinical study is probably due to the fact that local dogs lead a semi-stray way of life which results in higher exposure of the causative agents of diarrhoea. However, the observation that both local and Alsatian breed of dogs had a higher incidence as compared to their crosses in the restrospective study cannot be explained.

From the clinical study, several potential causes of diarrhoea were identified but these causative agents were encountered in both diarrhoeic and apparently healthy dogs. Thus, it was not possible to determine for sure the exact cause(s) of the diarrhoea in all the diarrhoeic dogs. Higher incidence of diarrhoea resulting from intestinal parasitism in local dogs has been observed by Oduye and Olayemi (1977).

It is disturbing to note that the most commonly used antimicrobial agents at ABUVTH (namely penicillin, streptomycin and tetracycline) are not effective against an appreciable number of the isolates; especially since E. coli also causes other infections (e.g. septicemia, urinary tract infections, etc). Gentamicin, an expensive drug, seems to be the most effective antibiotic against E. coli. The role of E. coli in puppies is not well documented as in other animals.

The observed high detection rate of E. coli in the faeces of younger dogs agrees with the observation of Dillon (1984). The results of this study indicate diversity of E. coli serotypes as earlier observed by Wilk (1981). The fact that a significant number of E. coli isolates were invasive and/or produced toxins

suggests that the isolates were probably responsible for the diarrhoea, particularly in the younger dogs where other pathogens assayed for were not detected. It is also possible that the diarrhoea may have been due at least in part to some dietary or other non-infectious factors.

The majority of the tested E. coli isolates were resistant to commonly available antimicrobial agents; this has similarly been observed elsewhere and is thought to be the result of increased miuse of the amtimicrobials (Jackson, 1981).

The isolation of Y. enterocolitica serogroup 0:8 from an apparently healthy dog in this study is of significance because, except for one report of isolation from a pig (Adesiyun et al., 1984), this serotype had never been documented outside the U.S.A. and Canada. Its detection in a healthy dog suggests the possibility that dogs may be reservoirs of human infection with Yersinia. This suggestion is supported by the fact that the serotype 0:8 infection has been serologically detected in human beings in Northern Nigeria (Awunor-renner and Lawande, 1982).

Serological evidence of infection by the serogroup in dogs in Zaria area have also been demonstrated (Adesiyun et al., 1986). It was of interest to observe that the isolates was negative for the two in vitro tests conducted to detect virulence (calcium dependency at 37°C and autoagglutination at 37°C) and also non-invasive and non-enterotoxigenic. Yersinia enterocolitica strains are however known to lose their virulence characteristics through plasmids (Gemski et al., 1980). It is also known that some serotypes include both virulent and avirulent strains which differ biochemically (Schiemann and Devenish, 1980). It is noteworthy that the isolate was sensitive to most of the readily available antimicrobial agents and the resistance pattern was similar to that reported by Papageorges (1983) in the United States.

The clinical significance of the strain Y.. enterocolitica isolated cannot be defined, but it may be necessary to determine the degree of involvement of this serogroup (O:8) in human diseases in Zaria area and how many of such cases are attributable to contact with dogs.

The rate of Salmonella isolation from both diarrhoeic and apparently healthy dogs was low (9.6%). This rate is lower than the 18% reported by Britt et al

(1978) from Vom and the 23.5% reported in dogs from Sudan (Khan, 1970) but higher than the 8% reported in dogs from Ibadan, (Ojo, 1974). The use of more than one enrichment and selective media has been known to increase isolation rate. That may also be responsible for the higher rates in other studies compared to this. Although the rate of Salmonella isolation in diarrhoeic dogs was higher than that in apparently healthy dogs, the difference was not statistically significant (P 0.05). Other enteric pathogens like Campylobacter spp., enteroviruses, e.t.c. and non-infectious agents may have been responsible for the diarrhoea seen. This finding indicates that there are carriers of salmonellae in the Zaria canine population. This carrier state in dogs has also been observed elsewhere in Nigeria (Ojo, 1974; Britt et al., 1978) and other parts of the world (Khan, 1970; Morse and Duncan, 1975; Borland, 1975; Shimi et al., 1976). Since all salmonellae are potential pathogens to man and animals (Morse and Duncan, 1975) and are of zoonotic importance (Morse et al., 1976; Morse et al., 1978), the finding that dogs carry the organisms in Zaria should be of concern to pet owners, dog meat consumers, public health workers and small animal practitioners.

The diversity of Salmonella serotypes isolated in this study is in agreement with findings in other animal species like cattle in the same environment (Kwaga, 1984). The isolation of more than one serotype in two dogs is not uncommon as observed by Morse and Duncan (1975). The results of the sensitivity testing on the isolated serotypes of Salmonella indicate that antibiogram patterns are similar to those reported elsewhere (Morse et al., 1978). It is expected that most isolates from dogs in Zaria area are likely to be sensitive to gentamicin, ampicillin and neomycin.

Intestinal helminths and protozoa had a high prevalence in both diarrhoeic and apparently healthy dogs. However, the infection load as indicated by egg counts was higher in diarrhoeic dogs. Unlike the report of Oduye and Olayemi (1977) where it was found as a major cause of diarrhoea. Ancylostoma spp. being one of the most common parasites observed has also been reported to be common by Dada et al. (1979) (65.5% in stray dogs) and Ezeokoli, personal communication (36.8% in household dogs).

Unlike the seasonal tendency observed by Ezeokoli, (personal communication) with a higher incidence during middle to late rainy season (June to November), this

study showed no statistical significance with seasonal variation. The higher rate of detection of ova and oocysts in the faeces of young dogs (1-6 months old) and older dogs (36 months old) may be an indication of decreased immuno-competency at these ages; for example, acute diarrhoea due to hookworm infections is common in young puppies (Anderson, 1975).

Strongyloides spp. and Trichuris spp. were not common infections observed in this study, probably because these parasites are common in dogs kept in kennels on heavily contaminated soil or grounds (Anderson, 1975), while majority of the dogs in this study are kept out-of-doors. Unlike the report of Dada et al. (1979) in which Dipylidium spp. was commonly detected in stray dogs, this was the opposite with the household dogs studies. Toxocara spp. was not a common parasite found in the household dogs.

Although it was not possible to categorically incriminate particular agents as the the causes of diarrhoea in this study as the same agents were detected in healthy dogs, it is reasonable to conclude that E. coli and Salmonella spp. were responsible for causing diarrhoea in young dogs as previously observed by other workers (Anderson, 1975; Borland, 1975; Morse and Duncan, 1975; Dillon, 1986).

CONCLUSIONS

From the results of both studies, the following can be concluded:

1. The results of the retrospective and clinical studies were similar;
2. Diarrhoea is not a common problem in the pet canine population of Zaria area;
3. Younger dogs and local breed of dogs are more commonly affected with diarrhoea compared to older dogs and their exotic breeds respectively.
4. Both enteric bacterial and parasitic infections are common in young dogs and also dogs older than 36 months;
5. Escherichia coli infections exist both as a cause of diarrhoea and inapparent infections, in the pet canine population amd may have clinical and zoonotic importance;
6. The Y. enterocolitica strain isolated (serogroup O:8) is a human serogroup, a possible indication that a few dogs may be a source of infection for human beings in Zaria;
7. Salmonella spp. exist in the pet canine population of Zaria area and both clinical salmonellosis and carrier forms exist;

8. Salmonella infection in apparently healthy dogs is common in the wet season;
9. Dogs in Zaria are potential sources of salmonellae for other animals and man and pet owners should be informed about this potential danger;
10. Drug resistance seems to be more of a problem with E. coli;
11. Ancylostoma spp. infection is an important consideration in dogs in Zaria environment. Although helminth infection was high in both diarrhoeic and apparently healthy dogs, the infection load was higher in the former.

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