Sahel J. Vet. Sci. Vol. 8, No. 2, pp. 57 - 68 (2009)

1605-8954/09/$25.00 + 00

Sahel Journal of Veterinary Science

Parasitic Gastroenteritis (PGE) Complex of Domestic Ruminants in Nigeria: A Review

A. W. Mbaya*[1], C. O. Nwosu[1] and U. I. Ibrahim[2]

[1]Department of Veterinary Microbiology and Parasitology; and [2]Department of Veterinary Medicine, University of Maiduguri, P. M. B. 1069, Maiduguri, Nigeria

ABSTRACT

Parasitic gastro-enteritis (PGE) is a major health problem and thus the main constraint on the profitable production of domestic ruminants in Nigeria. An annual loss of 60 million Naira due to the disease in domestic ruminants has been estimated. Parasitic gastro-enteritis is a complex of diseases involving several nematode species in these genera, *Haemonchus*, *Trichostrongylus*, *Oesophagostomum*, *Gaigeria*, *Cooperia* and *Strongyloides*. The disease has been shown to be a rainy season problem in the derived eastern savannah of Nigeria and the Sahel savannah or through out the year on the Jos plateau. Normal Nigerian temperature range of 13.8ºC (lower minimum for Obudu) to 40ºC (maximum for Maiduguri) are suitable for the development and survival of the eggs of the nematode species to infective larval stages of the parasites. During the dry season, however, when ambient temperature exceeds 40ºC in the arid-northeastern Nigeria, some degree of pasture sterilization occur with low parasitic load pertaining in the environment. The complex can either occur in a hyper acute, acute, or chronic form. The clinical signs include diarrhea, weight loss, submandibular oedema (bottle jaw), severe anaemia, dark-colored faeces and death depending on the stage. The anaemia is associated with either, intestinal haemorrhage or haemopoitic disturbances initiated by inapetence or loss of metabolites (due to impaired protein digestion) into the gut. The increased loss of plasma or serum proteins (protein-leaking gastro-enteropathy) occurs and is responsible for the hypoprotinaemia. Iron re-absorption in the gastrointestinal lumen is eventually reduced leading to low serum and bone marrow iron reserves leading to dyserythropoisis. Controls by grazing management, strategic anthelmintic medication have been used under varied conditions.

Key words: Parasitic gastroenteritis, domestic ruminants, Nigeria

INTRODUCTION

Nigeria has a large population of domestic ruminants where goats are the most numerous with an estimated population of 22.4 million followed by cattle and sheep at 11 and 7.6 million respectively (ILCA, 1985). Sheep and goats constitute the major source of animal protein in the country. However, in spite of the large population of small ruminants, animal protein consumption is far below national requirements. This short fall in animal protein availability is linked to the poor productivity of Nigeria's domestic stock (ILCA, 1985). Parasitic gastro-enteritis complex is one of the major limiting factors on the productivity of small ruminants (Schillhorn van veen, 1973; 1974; Akerejola *et al*., 1979; Pullan, 1980a; b; ILCA, 1985; Onugba and Agbede, 2006). Parasitic gastro-enteritis (PGE), results in enormous financial losses due to the associated morbidity, mortality and cost of treatment and control (Beaton, 1938; Schillhorn van veen, 1973). The losses associated with PGE in domestic ruminants in Nigeria have been enormous and increasing over the years. For instance, (Schillhorn van veen, 1973) estimated an annual loss of about 14 million naira. Much later, however, (Akerejola *et al*., 1979) estimated a national loss of over 60 million naira while (Ahmed *et al*., 1993) estimated the annual loss to a tune of 80 million naira due to the disease in small ruminants. These figures may represent and under estimation of the overall effect of PGE in domestic ruminants in Nigeria when losses due to unthriftiness cost of treatment and control measures are added (Nwosu, 1995; Nwosu et al., 1996).

PGE is a complex of diseases where several nematode parasites belonging to the genera *Haemonchus*, *Trichostrongylus*, *Oesophagostomum*, *Gaigeria* and *Strongyloides* contribute to the disease in domestic ruminants (Schillhorn van veen, 1973; 1978). The nematodes, which are parasitic in the gastro-intestinal tract of ruminants

*Author for correspondence

in Nigeria, include *Haemonchus contortus*; *H. placei* and *Trichostrongylus axei* in the abomasum; *T. colubriforms, Cooperia* species, *Bunostomum* spp., *Gaigeria pachyscelis* and *Strongyloides papillosus* in the small intestine and *Oesophagostomum columbianum*, *Trichuris* spp. and *Skrajabinema ovis* in the large intestine (reviewed in Chiejina, 1986). Among the various parasites, *H. contortus* is by far the most important and is responsible for most field outbreaks of acute and sub-acute PGE in ruminants (Schillhorn van veen, 1973; Anosa, 1977; Eysker and Ogunsusi, 1990; Nwosu *et al*., 2001; 2006; Mbaya and Aliyu, 2007).

Prevalence of PGE in domestic ruminants in Nigeria

Reports on various helminth parasites encountered among domestic ruminants in various geographical zones in Nigeria are presented in Table 1. Chiejina (1986) extensively reviewed the prevalence and seasonal fluctuations of nematode parasites of domestic ruminants in different geographical zones while Fagbemi and Dipeolu (1982) studied the incidence of *Strongyle* infections in the West African Dwarf (WAD) breed of sheep and goats in the rainforest and derived Savannah zones of Southwestern Nigeria. They reported that 29% of 829 goats and 36% of 335 sheep in the rainforest and 40.6% of 645 goats in the derived Savannah had *Strongyle* infections. Faecal cultures revealed mainly *H. contortus* and *T. colubriformis and O. columbianum*, the adults of which were similarly recovered during necropsy of some of the animals. Fabiyi (1970) and Okon (1975) gave the incidence of nematode parasites of goats located in the Northern Guinea Savannah zone. Out of 150 West African Dwarf goats examined in the Zaria area, Fabiyi (1970) reported that 89%, 56%, 51%, 40%, 25%, 3% and 1% were infected respectively with *Haemonchus, Trichuris, Trichostrongylus, Strongyloides, Gaigeria, Cooperia,* and *Skrajabinema* species. Among the various nematode species however, only *Haemonchus, Trichostrongylus, Gaigeria and Oesophagostomum* species occurred in numbers large enough to cause disease with the reported mean worm burdens for these helminths being 1,500, 1200, 12, 000, 10,000 respectively. With respect to *Haemonchus, Oesophagostomum and Gaigeria* species, Okon (1975) obtained essentially similar incidence rates from 295 goats examined in the same geographical zone.

Studies conducted in the derived Savanna of Eastern Nigeria, reported the occurrence of similar nematode species as recorded in other parts of the country with *Haemonchus, Trichostrongylus, Oesophagostomum and Gaigeria* as commonest (Adams and Mackay, 1966; Chiejina, 1984; Fakae, 1986; 1990; Fakae and Chiejina, 1993). However, in addition to these species, Fakae (1990) recorded *Bunostomum trigonocephalum* and *Capillaria* species in small ruminants maintained under the traditional husbandry system in the area. In the Sahel savannah however, *Haemonchus* (90.3%), *Trichostrongylus* (92.5%) and *Oesophagostomum* (67.5%) were the most predominant species encountered among small ruminants (Nwosu *et al*., 2001; 2006) and among semi-domesticated red fronted gazelles (*Gazella rufifrons*) and dorcas gazelles (*Gazella dorcas*) (Mbaya and Aliyu, 2007; Mbaya et al., 2008c).

Seasonal occurrence of PGE in domestic ruminants

There appears to be seasonality in the occurrence and relative abundance of the different nematode species parasitic in small ruminants in Nigeria. Chiejina (1986) reported that worm burdens are generally higher during the rainy season than in the dry season but that significant sequence occurred in the succession and relative abundance of the various species. Fabiyi (1973) and Nwosu *et al*. (1996) examined 134 West African Dwarf Goats and 960 Red Sokoto Goats respectively in the rain forest region of Nigeria. They reported that *Haemonchus, Trichostrongylus, Strongyloides* and *Cooperia* species predominant during the rainy season from June to October with a peak count in July. Furthermore, they reported that *Haemonchus* and *Trichostrongylus* species generally dominate during the first two months (early) of the rainy season. Based on the above observations, Nwosu (1995) concluded that outbreaks of caprine haemonchosis would naturally occur from about two months into the rainy season to the end of the rains provided the rains were frequent.

Fagbemi and Dipeolu (1982), however, noted that in the rainforest and derived Savanna zones of Southwestern Nigeria, *T. colubriformis* dominate during the dry season while *H. contortus* and *O. columbianum* occurred during the early and late rainy season. They however, reported that immature (4^{th} larval) stages of *Haemonchus* occurred in the abomasums even during the dry season when few or no adults occurred.

PGE has also been shown to be a rainy season problem in the derived Savanna of Eastern Nigeria as higher worm burdens were recorded in domestic ruminants during this period than in the dry season (Fakae and Chiejina, 1988). The authors further, reported that up to four generations of larvae were available on pastures during a favourable season and that *Trichostrongylus* species dominated the first (May) peak while *Cooperia* and *Haemonchus* species dominated the later months.

On the Jos Plateau, Onyali *et al*. (1990) reported that the development and survival of infective larvae of *Strongyle* nematodes occur throughout the year except during the dry months of December to April. According to them, more infective larvae were recovered from herbage in June, July and August than in any other month. According to Eysker and Ogunsusi (1990), three or four generations of *Trichostrongylus* species develop in the Nigerian Savanna. They further observed that *Trichostrongylus* species had a lower turn over rate than *Haemonchus* species and that this resulted in a gradual build up of their populations during the rains.

Table 1. Report on various helminth parasites encountered in parasitic gastroenteritis (PGE) complex of domestic and semi-domestic ruminants and in pasture according to geographical locations in Nigeria

Geographic locations	Species of ruminants	Parasites encountered	References
Northeastern Nigeria (Sahel savannah)	Sheep, goats, cattle, semi-domesticated gazelles	i. *H. contortus* ii. *T. colubriformis* iii. *O. columbianum*	Ahmed *et al.* (1993) Mbaya *et al.* (2006, 2007)
North Central Nigeria (Northern guinea savannah)	Sheep, goats	i. *T. colubriformis* ii. *T. axei* *iii. H. contortus* iv. *O. columbianum* v. *Gaigeria* vi. *Cooperia* vii. *Skrajabinema*	Beaton (1938), Okon (1970, 1975), Schillhorn van veen (1973, 1978), Fabiyi (1970), Fagbemi and Dipeolu (1982), Ogunsusi (1978), Eysker and Ogunsusi (1990), Makinde and Lasisi (2004), Onugba and Agbede (2006)
Northwestern Nigeria (Sahel savannah)	Cattle, sheep, goats	i. *Strongyles* ii. *Strongyloides*	Salihu *et al.* (2005)
Jos Plateau (Montane region)	Pasture	i. *H. contortus*	Onyali *et al.* (1990)
Eastern Nigeria (Derived savannah)	Cattle, sheep, goats, pasture	i. *H. contortus* ii. *T. colubriformis* iii. *O. columbianum* i v. *Gaigeria*	Adams and Mackay (1966), Chiejina (1984, 1986), Chiejina and Emehelu (1986), Fakae and Chiejina (1988, 1993)
Western Nigeria (Derived savannah zone of southwestern Nigeria)	Cattle, sheep, goats	i. *H. contortus* ii. *T. colubriformis* iii. *S. papillosus* iv. *Cooperia* v. *O. columbianum*	Anosa (1977), Akerejola *et al.* (1979),Fagbemi and Dipeolu (1982), Nwosu (1995), Nwosu *et al.* (2001, 2006)

Life cycle and transmission of etiological agents of PGE

Most Strongyline nematodes, responsible for PGE of ruminants have simple life cycles (Soulsby, 1982; Hansen and Perry, 1994). Their life cycle in general, involves a free-living pre-parasitic phase and a parasitic phase of development. However, the life cycle of *Haemonchus contortus* is the most studied (Rogers and Somerville, 1963; Michel, 1976; Armour, 1982; Chiejina, 1986).

The free-living pre-parasitic phase of *H. contortus* starts in the abomasums with egg laid by the sexually mature female, which is voided out with faeces of the ruminants. The eggs develop and hatch into first larval stage (L_1) within 24 hours under favourable conditions of oxygen, moisture and temperature (Soulsby, 1982). These larvae feed on bacteria contained in the faecal pad, develop and ex-sheath to the second larval stage (L_2), which feed in a similar fashion and develop into the third larval stage (L_3), the infective stage. The L_3 which is enclosed in the sheath of the L_2, does not feed but migrates out of the faecal pad onto grass blades in the pasture through positive hydrotropism to the tip of grass blades. The developmental period from egg to the infective larval stage usually takes 4 - 6 days. Transmission to ruminants occurs when they ingest the infective larval stage (L_3) while grazing. At this point, the parasitic phase starts (Urquhart, et al., 1977; Soulsby, 1982; Nwosu, 1995). Ex-sheathment of the L_3 occurs, and on arrival in the abomasums, the larvae penetrate the gastric crypts from where they emerge later as the fourth stage larvae (L_4). The (L_4), thereafter, moults into the fifth (L_5) larval stage or young adult and undergoes sexual maturity and starts laying eggs. For *H. contortus*, the period of development from the ingestion of the L_3 to the commencement of egg laying in the abomasums takes about 14 – 18 days (Blood et al., 1979; Soulsby, 1982; Radostitis et al., 1997). This period may however, be extended in animals with acquired immunity (Soulsby, 1982). Some (L4) may become arrested (hypobiotic) after ingestion and do not emerge from the mucosal crypts until 4 -5 months later (Murray et al., 1974 Armour, 1982). Abbott et al. (1985a) and Rahman and Collins (1990a) reported that following patency, egg production increases tremendously until maximum out put is reached in 25 – 49 days period. It was also observed in sheep, that each adult female *H. contortus* produces, 1352 – 1730 eggs per gram of faeces (Cvetkovic and Lepovev, 1975). Dinnik and Dinnik (1955), however, reported that up to 10, 000 eggs are produced daily by female *H. contortus*.

Pathogenesis of PGE in ruminants

The pathogenesis of PGE is primarily associated with the haematophagus activity of *H. contortus* (Martin and

Clunies-Ross, 1934; Clark, et al., 1962; Soulsby, 1982; Hansen and Perry, 1994; Chiejina, 1987; Nwosu, 2001; 2006; Mbaya and Aliyu, 2007; Mbaya et al., 2008b, c). The authors reported that, the anaemia, commonly encountered in PGE is attributable, to the loss of whole blood because of the blood sucking activities of the 4th larval stage and subsequent haemorrhage into the abomasal lumen from points of attachment of the nematodes. Dargie (1975) and Dargie and Allonby, (1975), described 3 phases in the pathogenesis of the anaemia in PGE. They observed that the first phase, which occurs within the first 3 weeks of infection, is the acute phase. In this case, fatal blood loss occurs before erythropoitic response ensues. During the second phase, which occurs from the 3rd – 8th week of infection, an unabated loss of red blood cells and iron occurs, which is adequately compensated by erythropoitic response. Soulsby (1982), however, reported that iron re-absorption in the gastrointestinal lumen is eventually reduced leading to low serum and bone marrow iron reserves leading to erythropoitic exhaustion (dyserythropoisis). Dyserythropoisis occurs during the 3rd phase because of iron deficiency and possibly, amino acids (Nwosu, 1995). Meanwhile, anaemia is rarely encountered when *Oesophagostomum, Cooperia* and *Trichostrongylus* are involved. If they do occur, however, the anaemia is associated with either, intestinal haemorrhage or haemopoitic disturbances initiated by inapetence or loss of metabolites (due to impaired protein digestion) into the gut (Symons and Steel, 1978).

It was also observed that increased loss of plasma or serum proteins (protein-leaking gastro-enteropathy) occurs in PGE and is responsible for the hypoprotinaemia observed in this condition (Armstrong, et al., 1960; Soulsby, 1982). Hypoprotinaemia and a high fractional catabolic rate (turnover) of albumin have been reported in ostertagiasis, trichostrongylosis and haemonchosis (Kutler and Marble, 1960; Brenner, 1969; Dargie, 1975; Abbott *et al.*, 1985a; b; 1986a; b; Ahmed *et al*., 1990; Nwosu, 1995; Nwosu *et al*., 2006; Mbaya and Aliyu, 2007). Ahmed *et al*. (1990) noted that hypoprotinaemia was associated with a decline in total proteins and albumin/globulin ration in sheep infected with *H. contortus*. Among the globulin fractions, they also noted that the alpha, beta and gamma globulins increased at the peak of the infection and persisted after treatment when all other fractions returned to normal values. Ahmed *et al*. (1990) observed that the hypoprotinaemia, in general, contributes to the increased water retention in the dependent parts of the body, leading to bottle jaw, ascitis and general oedema. Diarrhoea is an integral part of the pathogenesis of *Strongyloides*, *Trichostrongylus*, *Cooperia*, *Nematodirus* and *Bunostomum* infections (Soulsby, 1982; Hansen and Perry, 1994) but not a common feature of *Haemonchus* infection (Soulsby, 1982). The pathogenesis of diarrhoea in the PGE complex of ruminants has been associated with the morphological and functional gastric changes in the host. The damage to the gut mucosa because of the infection, often lead to an increased permeability to plasma proteins and other macromolecules. This subsequently leads to the accumulation of fluid in the gastrointestinal tract due to increased hydrostatic pressure (Brenner, 1969; Castro *et al*., 1969; Holmes and MacLean, 1971; Barker, 1973; Abbott et al., 1985a, b; 1986a, b; Nwosu et al., 2001; 2006; Mbaya and Aliyu, 2007). The above situation coupled with reduced rate of absorption of water and electrolytes because of damage to the intestinal mucosae could lead to the development of diarrhoea (Symons and Steel, 1978).

Anorexia, a common feature of PGE, associated with a marked drop in food consumption was reported in animals infected with *Ostertagia*, *Nematodirus*, *Oesophagostomum*, and *Trichostrongylus* species (Gordon, 1964; Horak and Clark, 1964; 1966; Rowland and Robert, 1972). However, anorexia may not occur in haemonchosis (Soulsby, 1982; Abbott et al., 1986a, b). Several factors contribute to the development of anorexia in PGE of ruminants, where intestinal parasitism stimulates increased secretion of cholecystokinin (CCK) by mucosal cells of the small intestine, increased plasma levels of which acts on the appropriate centre in the brain to depress appetite (Symons and Hennessey, 1981).

Epizootiology of PGE in domestic ruminants

The acquisition of heavy nematode burdens by domestic ruminants is dependent on the availability of large numbers of infective larvae on pasture and their subsequent transmission to the animals. The latter, was influenced by a variety of factors such as biotic potential and generation interval of the nematodes, host resistance and environmental factors leading to spring rise and arrested development of larvae (Soulsby, 1982; Radostitis *et al*., 1997). Host factors such as nutrition, intercurrent diseases, and age and husbandry practices such as overcrowding and poor hygienic measures also influence the epizootiology of parasitic-gastroenteritis in domestic ruminants (Schillhorn van veen, 1973; Anosa, 1977; Chiejina, 1986; Eysker and Ogunsusi, 1990; Nwosu *et al*., 2001; 2006).

Effect of host age on PGE

Generally, young animals show clinical disease during their first challenge while adults exposed to repeated challenge show higher degree of resistance to PGE (Soulsby, 1982). However, adult domestic ruminants (Schillhorn van veen 1974; 1978; Allonby and Urquhart, 1975; Nwosu, 1995) may suffer clinical disease when their resistance is compromised by factors such as poor nutrition, intercurrent diseases, immunosuppression, and physical stress or under certain types of intensive husbandry systems.

Anosa (1977) reported a natural outbreak of ovine haemonchosis in which the most severely affected groups were lambs above weaning age (3 months) followed by older ones whereas lambs below 2 months were either free

of infection or mildly affected. Fagbemi and Dipeolu (1982) made the same observations where the lowest and highest rates of infection were encountered among kids aged 3 – 6 months and 7 – 12 months respectively.

Effects of concurrent diseases on PGE

The influences of concurrent disease on the severity of PGE in small domestic (Christensen *et al*., 1987; Nwosu *et al*, 2006) and semi domestic ruminants (Mbaya et al., 2008a; b; c) have been extensively studied. Such concurrent infections, which could occur between different species of helminths and protozoans frequently, result in heterelogous synergistic interactions involving two or more helminth species. The cumulative effect result in the enhancement of their pathogenesity as reflected by increased growth, fecundity and initial establishment as well as delayed expulsion of the parasites leading to self-cure. For instance, Kates (1943) reported that concurrent infection of sheep with *H. contortus*, and *O. circumcincta* enhanced the pathogenecity of *T. axei*. In addition, the author reported that simultaneous infection with *F. hepatica* inhibited the development of acquired resistance after primary infection of *H. contortus* unlike in animals, which recovered naturally from the infection.

Immunosuppression is one of the most important consequences of protozoan disease such as trypanosomosis in ruminants (Mackenzie *et al*., 1975; Nwosu *et al*., 2001; 2006) and in semi-domesticated ruminants (Mbaya *et al*., 2008a; b). Immunosuppression has been shown to be responsible for the synergistic effects of protozoans on helminths during concurrent infections (Nwosu, *et al*., 2006; Mbaya *et al*., 2008a; b). For instance, Urquhart *et al.* (1973) showed that immunosuppression due to *T. brucei* enhanced pathogenecity and prevented mounting of secondary immune responses against *Nippostrongylus braziliensis* in rats. Jenkins *et al*. (1974) and Murray *et al*. (1974) also reported that the immune responses against the nematode challenge were restored following trypanocidal therapy.

Furthermore, Griffin and Allonby (1979) recorded higher mortality rates among 44 goats and 29 sheep with concurrent mixed *Strongyle* nematode and *T. congolense* infections. In latter studies, Griffin and Preston (1981a; b) recorded increased establishment of the initial infection of *H. contortus* in *T. congolense* infected goats.
On the other hand, cross-resistance between nematode spp. or between nematodes and protozoans has been demonstrated (Griffin and Preston, 1981a; b; Nwosu, 1995). Such heterologous antagonistic interactions are thought to be mediated either by specific immunological cross-reactivity between closely related parasite species exhibiting a marked immunological similarity or by immunologically non-specific factors (Griffin and Preston, 1981a; b; Soulsby, 1994). This mechanism is possible through macrophage activation and inflammatory reactions commonly demonstrated by reduced establishment, survival as well as enhanced expulsion of the primary infection as a result of lack of specificity of the self-cure mechanism (Griffin and Preston (1981a; b). Holmes (1973), however, suggested that some of the antagonistic interactions between helminths are brought about by competition for nutrients and or direct mechanical interference.

Pathogenic effects of PGE

The pathogenesis and pathology of haemonchosis in domestic animals have been described (Anosa, 1977; Adams, 1981; Abbott *et al*., 1985a; b; 1986a; b; Rahman and Collins, 1990; Nwosu *et al*., 2001; 2006). According to the authors, the main features of haemonchosis in domestic ruminants are generally hypoalbuminaemia, anaemia, poor weight gains and sometimes sudden death. Soulsby (1982) and Chiejina (1987) reported that the onset and severity of the pathology depend largely on the species and population of nematodes involved, their stages and site of development within the gut, the larvae and factors as age, plane of nutrition, resistance and the presence or absence of intercurrent diseases.

Anaemia, hypo albuminaemia, haemorrhagic gastritis, pallor of skin and mucous membranes, watery blood, hydrothorax, hydropericardium, ascitis, cachexia, fragile and fatty liver, abomasitis have been reported in haemonchosis (Brunsden, 1970; Anosa, 1977; Nwosu, 1995; Nwosu et al., 2001; 2006). The authors reported that in addition to these changes, frank or coagulated blood, fibrin clots, mucous and desquamated cells also occur in PGE when other haematophagus species such as *Gaigeria* and *Bunostomum* are present. According to Chiejina (1987), non-blood suckers such as *Trichostrongylus axei* in the abomasum and *T. colubriformis* and *Cooperia* species in the small intestine cause proliferative gastropathy, accompanied with catarrhal inflammation and epithelial ulceration. Meanwhile, *Oesophagostomum* species produces nodular (pimply) lesions of various sizes (0.5 – 1.5 cm) on the serosal surface of the large intestine (Mbaya and Aliyu, 2007). The pimply lesions usually lead to ulceration with a leakage of plasma proteins with varying degrees of peritonitis as sequeale.

Clinical manifestation of PGE in domestic ruminants

Generally, PGE is a disease of young ruminants (Turner et al., 1962; Soulsby, 1982; Schillhorn van veen 1974; 1978; Allonby and Urquhart, 1975; Chiejina, 1986; Fakae and Chiejina, 1988; Nwosu, *et al*. 2001; 2006). The disease in either case is manifested in either hyper acute, acute, sub-acute or chronic form (Soulsby, 1982; 1994). According to the authors, the acute and sub-acute forms are rainy season problems predominantly due to *Haemonchus* and to lesser extent *Trichostrongylus* species and characterized by high worm burden of 5,000 –

25,000 eggs per gram of faeces, anaemia and sometimes sudden deaths. However, the chronic form, which occur throughout the year especially at the end of the rains, is associated with mixed infections of *Oesophagostomum, Trichostrongylus, Gaigeria,* cestodes and sometimes *Haemonchus* species which is manifested by low faecal egg output of 500 -5,000 (epg), diarrhea, weight loss and submandibular oedema. Similarly, Ogunsusi (1978) reported a chronic form of ovine PGE of about 18 weeks duration caused by mixed infections of *Trichostrongyle* species, which was associated with anaemia and high faecal egg counts.

The hyper acute form has been reported in small ruminants (Allonby, 1973) and in a herd of semi-domesticated ruminants in the arid zone of northeastern Nigeria (Mbaya and Aliyu, 2007). The outbreak in either case occurred following a sudden massive intake of extremely large numbers of infective larvae, which was associated with rapidly developing fatal severe anaemia, dark-coloured faeces and sudden death within 1 - 6 days. However, in the acute form, which lasts 1 – 6 weeks, the anaemia is usually associated with marked erythropoietic response, hypoproteinaemia, and generalized oedema especially bottle jaw and very high faecal egg counts of up to 100,000 eggs per gram of faeces (Schillhorn van veen, 1973).

On the other hand, however, the chronic form in red Sokoto (Maradi) goats was associated with the presence of very low numbers of adult worms in the abomasums manifested by low faecal egg counts and low mortality (Nwosu, *et al*., 2006). This form lasts for about 2 – 6 months with the severity of the anaemia depending on the erythropoietic response and the iron and nutritional reserve of the animal host (Allonby, 1973; Nwosu *et al*., 2001; 2006). These observations were more common during the rainy season but many deaths occur because of chronic haemonchosis during the dry season especially where the pasture is of poor quality (Cheijina, 1986).

Pathology of PGE in domestic ruminants

The pathological changes, which occur in small ruminants suffering from PGE, have been described by several workers (Soulsby, 1982; Chiejina, 1987; Nwosu *et al*., 2001; 2006) and among semi-domesticated ruminants (Mbaya and Aliyu 2007; Mbaya *et al*., 2006; Mbaya *et al*., 2008a; b). Depending on their site of localization within the gut, pathogenic nematodes generally cause morphological and functional changes in the intestine, which usually result in either direct or indirect production losses (Soulsby, 1982). The pathogenesis and pathology of haemonchosis have previously been studied (Dargie, 1973; Adams, 1981; Abott *et al*., 1985a; b; 1986a; b; Rahman and Collins, 1990; Ahmed *et al*., 1990). According to Soulsby (1982), both the adult and fourth larval stages (L_4) of *Haemonchus* species are pathogenic in the abomasum. The morphological changes noted during haemonchosis in domestic ruminants (Dargie, 1973; Adams, 1981; Abott *et al*., 1985a; b; 1986a; b; Rahman and Collins, 1990; Ahmed *et al*., 1990) involved damage to the integrity of the abomasal mucosa which show hemorrhages at points of attachment of the worms. However, the functional changes occur due to haematophagic activity of the adult and 4^{th} larval stages and through blood loss from hemorrhagic lesions resulting from their attachment onto the mucosa. In sheep infected with *H. contortus*, Adams (1981) reported that the parasite produced a blood loss anaemia, leucopenia, mild lymphopaenia, thymus atrophy, decreased size of the spleen, enlargement of the adrenal glands and a four-fold increase in the erythroid series cells of the bone marrow. The postmortem findings of haemonchosis may vary according to the clinical syndrome of the disease. According to Allonby (1973) and Dargie (1973), the primary lesions in hyper acute haemonchosis are hemorrhagic gastritis with signs of anaemia and a large number of juvenile worms in the abomasums enmeshed in red ingesta. The authors also observed that the acute disease is associated with generalized oedema, signs of anaemia and a large number of adult worms (1,000 – 10,000) in the abomasums while the chronic disease showed mainly hyperplastic gastritis, expanded bone marrow, low faecal egg and abomasal worm counts. However, Brunsden (1970) and Anosa (1977) have reported that individual variability influences the severity of the anaemia and abomasal parasite counts. Similar clinical and postmortem signs as those due to haemonchosis are produced by *Gaigeria* and *Bunostomum* spp. which are also blood suckers and equally parasitic in the gut of small ruminants (Brunsden, 1970; 1972).

According to Chiejina (1987), non-blood suckers such as *Trichostrongylus axei* in the small intestine caused proliferative gastropathy, accompanied with catarrhal inflammation and epithelial ulceration. In such cases, the postmortem lesions seen included epithelial shedding and villous atrophy, cellular hyperplasia and metaplasia with the replacement of normal intestinal absorptive cells (enterocytes) by immature and non-functional mucus-producing cells. These morphological changes result in increased mucosal permeability and leakage of plasma proteins (protein-leaking-gastroenteropathy) and impaired digestion and absorption of nutrients. Ferguson and Jarret (1975) reported that the development of morphological and functional changes seen during PGE is contributed to by both direct physical damage by the nematode and immunological mechanisms of the host.

Oesophagostomum species which are parasitic in small domestic (Nwosu *et al*., 2001; 2006) and semi-domesticated ruminants (Mbaya and Aliyu 2007) often, produce nodular lesions on the serosal surface of the large intestine.

Diagnosis of PGE in domestic ruminants

The methods for the diagnosis of the disease are the same in domestic ruminants (Makinde and Lasisi, 2004;

Salihu *et al*., 2005; Onugba and Agbede, 2006). These include, history taking, clinical examination and appropriate coprological examination. Faecal culture, larval recovery and identification of infective larvae, postmortem examination for pathological lesions and total differential worm counts are very effective (Blood *et al*., 1979; Soulsby, 1982; Radostitis *et al.*, 1997; Nwosu, 1995; Mbaya *et al*., 2006).

Coprological examination for nematode ova is the oldest, simplest and most widely used laboratory technique presently available for the diagnosis of PGE (Soulsby, 1982). However, the method is messy and lacks adequate specificity, as the eggs of most of the nematodes responsible for PGE are similar (Anon, 1977). Faecal egg counts as a measure of worm burden to establish the severity of infection especially in young animals is advised under any circumstance (Soulsby, 1982). In such cases, treatment in small ruminants is absolutely necessary when the faecal egg count is in excess of 1000 epg while counts of 2,000 – 6,000 epg in small ruminants and over 10,000 epg in all species of domestic ruminants is associated with a severe clinical infection.

In addition, faecal culture technique to determine the specific nematode species requires a high degree of skill and experience to identify the infective larvae (Soulsby, 1982). For this method, a minimum of about 7 – 10 days is required before an accurate diagnosis is obtained (Nwosu, 1995). Moreover, Chiejina (1987) reported that both coprological examination and faecal culture might be associated with false negative results in pre-patent and even some patent infections.

Total and differential postmortem worm counts, which are accurate and simple field techniques, are superior to faecal egg counts as a measure of the severity of infection (Nwosu *et al*., 2001; 2006). However, it is often, limited by the unavailability of enough carcasses to examine. However, Blood *et al*. (1979) and Radostitis *et al*. (1997) recommended that at least two animals should be examined before arriving at a confirmatory diagnosis. According to them, in a mixed *Strongyle* infection, counts less than 2,000 adult worms in small/large ruminants are severe and pathogenic depending on the dominating species.

For *H. contortus,* they recommended that total counts of more than 1,000 worms in sheep might be considered a heavy infection while counts of 3,000 in lambs and 9,000 in adult sheep usually lead to heavy mortalities. In view of the obvious limitations of the traditional diagnostic methods, several workers have employed other methods. These include haematological examinations (Brunsden, 1972; Thomas and Waller, 1975), estimation of PCV, Hb, and RBC values (Chiejina, 1987; Nwosu, *et al*., 2001; 2006; Mbaya and Aliyu, 2007; Mbaya *et al*., 2008a) as well as serum gastrin and pepsinogen levels (Brunsden, 1972; Thomas and Waller, 1975) in infected animals. However, on their own, these methods have only been partially successful and at best complementary to other diagnostic methods.

Immunodiagnostic techniques may be more rapid and sensitive than the traditional diagnostic methods especially during pre-patent (acute) haemonchosis when eggs may be absent in feaces. Serodiagnostic methods based on antibody assay may be employed. However, antibody assay methods show limited specificity due to extensive cross-reactions between various nematode parasites and do not with certainty distinguish between previous and extent infections (Nwosu *et al*., 2001; 2006). For instance, Adams and Beh (1981) demonstrated that a rise in haemaglutinating antibody specific for *H. contortus* in serum of sheep during the course of a primary infection did not correlate with faecal egg counts. Furthermore, they observed that re-infections of the animal with the parasites did not stimulate a rise in titre. There is therefore, need for a more sensitive and specific immunodiagnostic method which may not only detect parasite species but the developmental stage or age of a given parasite, be able to detect both active and cured infections as well as correlate diagnosis with actual worm burdens or severity of the disease (Nwosu, 1995).

Serodiagnostic techniques based on the detection of circulating and urinary parasite antigens may fulfill these needs. Although attempts have been made by Ozerol and Silverman (1960; 1970) and Cox *et al.* (1989) to isolate, purify and characterize the antigens of the different developmental stages of the various nematodes involved in PGE for immunization purposes, not much has been done on the detection of these antigens in the circulation or urine of infected animals. Therefore, there is need for further studies to detect and further purify these antigens in order to determine those that could be useful in specific immunodiagnostics in PGE.

Control of PGE in domestic ruminants

Domestic ruminants are usually exposed to nematode infections as long as they graze on pastures and they continually contaminate such pastures with nematode ova. Therefore, the goal of control is to maintain the nematode populations at levels, which preclude outbreaks of clinical disease while allowing the animals to cope and develop some measures of acquired resistance (Nwosu, 1995). Generally, control measures are aimed primarily at protecting the most susceptible groups, which are the young ones exposed to infection for the first time.

Several methods have therefore, been proposed for the control of PGE in grazing animals (reviewed in Bourgton, 1955; Michael, 1976; Brunsden, 1972; 1980; Morley and Donald, 1980; Nwosu, *et al*., 2001; 2006). However, several factors including the concentration of animals, their nutritional status and the epidemiology of the nematodes in that environment influence one or a combination of methods, which may be adopted for use in any particular circumstance. According to Chiejina (1986) and Nwosu *et al*. (1996; 2006), the most commonly used

methods, which are of proven practical value in Nigeria, are grazing management, hygiene and strategic anthelmintic medication.

Control by grazing management

Theoretically, it is possible to raise worm-free animals if they are started on worm-free pastures and continually rotated on new worm-free pastures within the minimal period liable to elapse between the passages of worm eggs on pasture (Nwosu, 1995). According to Chiejina (1986), general hygienic practices and adequate knowledge of the epizootiology and bionomics of the nematodes will contribute to the success of grazing management as a control method. However, since the use of the method entails the possession of limitless land, which is not possible under the predominantly nomadic and traditional system of livestock management practiced in Nigeria, Chiejina (1986) considers the method inapplicable under such circumstance.

Control by strategic anthelmintic medication

This method describes a system whereby anthelmintics are administered at carefully selected periods in a grazing year. The existing knowledge of the epizootiology of PGE in an area indicates that heard dosing followed by their movement to new safe pastures would remove existing burdens thereby preventing pasture contamination and outbreak of clinical disease (Chiejina, 1986; Nwosu, 1995; Nwosu, *et al*., 2006). The effective use of this method entails that the epizootiology of the worms involved in PGE in an area must have been previously established.

The control of PGE based on principle of strategic anthelmintic medication and stock movement to safe pastures have been found to be very effective (Chiejina and Emehelu, 1986). At Nsukka, Nigeria, the authors obtained a significantly improved weight gain with a considerable reduction in pasture contamination following strategic dosing of three groups of intensively managed N'Dama cattle in April, July, August or September after they were moved to a safe pasture. In a similar, but previous study, Schillhorn van veen and Brinckman (1975) reported that monthly dosing of intensively managed lambs with thiabendazole led to an average weight gain of 17g/day over less frequently treated ones.

The method of anthelmintic administration, which makes the strategic movement of infected flock to the new pastures unnecessary, has been developed and found to be highly effective in controlling PGE of cattle in several countries in the temperate region (Weatherley *et al*., 1989). The device involves an intra-ruminal implantation, which slowly releases the anthelmintic Morantel over a period of 90 days. The efficacy of the Morantel Sustained Released Trilaminate (MSRT) bolus in the prevention of PGE in calves at pasture is not yet assessed in small ruminants nor has it achieved worldwide acceptance. Chiejina (1986) suggested its possible usefulness in controlling PGE of livestock in Nigeria Savannah.

ACKNOWLEDGEMENTS

The authors wish to thank all authors that provided their reprints for this work.

REFERENCES

Abbott, E.M., Parkins, J.J. and Holmes, P.H. (1985a). Influence of dietary protein on parasite establishment and pathogenesis in Finn Desert and Scottish black face Lambs given a single Moderate infection of *Haemonchus contortus*. *Res. Vet. Sci.* 38: 6-13.

Abbott, E.M., Parkins, and Holmes, P.H. (1985b). Influence of dietary protein on the pathophysiology of ovine haemonchosis in Finn Desert and Scottish black face Lambs given a single Moderate infection. *Res. Vet. Sci.*38: 54 -60.

Abbott, E.M., Parkins, J.J. and Holmes, P.H. (1986a). The effect of dietary protein on the pathogenesis of acute ovine haemonchosis. *Vet. Parasitol.* 20: 275-289.

Abbott, E.M., Parkins, J.J. and Holmes, P.H. (1986b). The effect of dietary protein on the pathophysiology of acute ovine haemonchosis. *Vet. Parasitol.* 20: 291-306.

Adams, D.B. (1981). Changes in blood leucocytes, bone marrow and lymphoid organ in sheep infected with *H. contortus*. *Intnl. J. Parasitol.* 11: 309 – 317.

Adams, D.B. and Beh, K.J. (1981). Immunity acquired by sheep from experimental infection with *H. contortus*. *Intnl. J. Parasitol.* 11: 381-386.

Adams, J.W. and Mackay, J. (1966). Gastrointestinal parasites of farm animals in Eastern Nigeria. *Vet. Rec*. 212: 216-217.

Ahmed, A., Anwar-ul-Hassan, C.H., Anwar-ul-Haq, A. and Majeed, M.A. (1990). Serum proteinogram of lambs in experimentally induced *H. contortus*. *Vet. Arhiv* 60: 195-200.

Ahmed, M.I., Nwosu, C.O. and Srivastava, G.C. (1993). Economic importance of helminth diseases of livestock in Maiduguri, Nigeria. *Nig. J. Anim. Prod*. 21: 152-155.

Akerejola, O.O., Schillhorn van veen, T.W. and Njoku, C.O. (1979). Ovine and caprine diseases in Nigeria: A

review of economic losses. *Bull. Anim. Hlth. Prod. Afr.* 27: 65 -70.
Allonby, E.W. (1973). Ovine haemonchosis: epidemiology, clinical signs and diagnosis. In: G.M. Urquhart and J. Armour (Editors), *Helminth Diseases of Cattle, Sheep and Horses in Europe*. Robert Maclehose and Co. Ltd., Glasgow, pp. 59-63.
Allonby, E.W. and Urquhart, G.M. (1975). The epidemiology and pathogenic significance of haemonchosis in Merino lock in East Africa. *Vet. Parasitol.* 1: 129-143.
Anononymous (1977). Manual of Veterinary Parasitological Laboratory Techniques, Technical Bulletin No. 18, Ministry of Agriculture, Fisheries and Food, London, pp. 129-132.
Anosa, V.O. (1977). Haematological observation on helminthiasis caused by *H. contortus* in Nigeria Dwarf sheep. *Trop. Anim. Hlth. Prod.* 9: 11-17.
Armour, J.D. (1982). The epidemiology of helminth disease in farm animals. *Vet. Parasitol.* 6: 7- 46.
Armstrong, F.B., Morgan, P.S. and Tarver, H. (1960). Plasma proteins: Site of degradation of serum albumin. *Proc. Roy. Soc. Exp.. Biol. Med.* 103: 592-596.
Barker, I.A. (1973). A study of the pathogenesis of *T. colubrioformis* infection in lambs with observations on the contributions of gastrointestinal plasma loss. *Int. J. Parasitol.* 3: 743-757.
Beaton, W.G. (1938). Goat husbandry in Northern Nigeria including a description of the cause of mortality in Nigeria, Federal Dept. Vet. Res. Annual Report. Appendix II, (1937), Govt. Printer, Kaduna, pp.471-478.
Blood, D.C., Henderson, J.A. and Radostitis, O.M. (1979). *Veterinary Medicine*. Bailiere Tindal, London, pp. 1135-1138.
Bourghton, D.C. (1955). Effective control of internal parasites. *Adv. Vet. Sci.* 2: 380-410.
Brenner, K.C. (1969). Pathogenic factors in experimental bovine eosophagostomiasis. III. Demonstration of protein-losing enteropathy with 51Cr-albumin. *Expl. Parasitol*. 24: 362-374.
Brunsden, R.V. (1970). Within flock variations in *Strongyle,* worm infections in sheep: the need for adequate diagnostic samples. *Nig. Vet. J.* 18: -188.
Brunsden, R.V. (1972). The potential role of pasture management in the control of *Trichostrongyle* worm infection in calves with observations on the diagnostic value of plasma pepsinogen determinations. *N.Z. Vet. J.* 18: 185-188.
Brunsden, R.V. (1980). Principles of helminth control. *Vet. Parasitol*. 6: 185-215.
Castro, G.A., Hassel, J.J. and Whalen, G. (1969). Altered intestinal fluid movement in response to *Trichinella spiralis* in immunized rats. *Parasite Immunol*. 1: 259 -266**.**
Chiejina, S.N. (1984.) Bovine parasitic gastroenteritis in Nsukka: Worm egg out put and herbage infestation following dry season calving and the performance of calves during their first grazing season. *Trop. Vet*. 2: 128 -132.
Chiejina, S.N. (1986). The epizootiology and control of parasitic gastro-enteritis of domesticated ruminants in Nigeria. *Helminthol. Abst*. 55: 413-429.
Chiejina, S.N. (1987). Parasitic gastro-enteritis in cattle and small ruminants: Pathogenesis, diagnosis and treatment. *Zariya Vet.* 2: 45-64.
Chiejina, S.N. and Emehelu, C.O. (1986.) Evaluation of three strategic anthelmintic programmes for the prophylaxis of parasitic gastroenteritis in cattle in Nigeria. *Trop. Anim. Hlth. Prod*. 18: 55-63.
Christensen, N.O., Nansen, P., Fagbemi, B.O. and Monrad, J. (1987). Heterologous antagonistic and synergistic reactions between helminthes and between helminthes and protozoans in concurrent experimental infections of mammalian hosts. *Parasitol. Res*. 73: 387-410.
Clark, C.H., Kiesel, G.H. and Goby, C.H. (1962). Measurement of blood loss caused by *H. contortus* infection in sheep. *Am. J. Vet. Res*. 23: 977-980
Cox, S.N., Shamansky, L.M. and Boisvenue, R.J. (1989). Identification and preliminary characterization of cuticular surface proteins of *H. contortus*. *Mol. Biochem. Parasitol.* 36: 233-242.
Cvetkovic, L. and Lepojev, O.(1975). Production of eggs by *H. contortus* in sheep under pasture conditions of infection. *Acta Veterinaria*, *Yugoslavia*. 25: 83-89.
Dargie, J.D. (1973). Ovine haemonchosis: Pathogenesis In: G.M.Urquhart and J. Armour (Editors), Helminth Diseases of Cattle, Sheep and Horses in Europe. Robert Maclehose and co. Ltd, Glasgow, pp. 63-71.
Dargie, J.D. and Allonby, E.W. (1975). Pathophysiology of single and challenge infections of H. contortus in Merino sheep: studies of red cell kinetics and the 'self cure' phenomenon. *Int. J. Parasit*. 5: 147-157.
Dinnik, J.A. and Dinnik, N.N. (1955). The development and survival of H. contortus larvae on pasture under the local conditions of the highlands of Kenya. *Helminthol. Abs.* 24: 631-632.
Eysker, M. and Ogunsusi, R.A. (1990). Observation on the epidemiology and clinical aspect of gastro-intestinal helminthes in sheep in Northern Nigeria during rainy season. *Res. Vet. Sci.* 28: 58-62.
Fabiyi, J.P. (1970). An investigation into the incidence of goat helminth parasites in the Zaria area of Nigeria. *Bull. Epiz. Dis. Afr.* 18: 29-34.
Fabiyi, J.P. (1973). Seasonal fluctuations of nematode infestations in goats in the savanna belt of Nigeria. *Bull.*

Epiz. Dis. Afr. 21: 277-286.
Fagbemi, B.O. and Dipeolu, O.O. (1982). *Strongyle* infections of small ruminants in Nigeria. *Vet. Parasitol.* 11: 347-353.
Fakae, B.B. (1986). Aspects of the epidemiology of bovine parasitic gastroenteritis in the derived savanna area of eastern Nigeria. M.Sc. thesis, University of Nigeria, Nsukka, pp. 234.
Fakae, B.B. (1990). The epidemiology of helminthiasis in small ruminants under the traditional husbandry system in eastern Nigeria. *Vet. Res. Comm*. 14: 381-391.
Fakae, B.B. and Chiejina, S.N. (1988). Further studies on the development and availability of infective larvae of bovine gastro-intestinal trichostrongylids on pasture in eastern Nigeria. *Vet. Parasitol*. 28: 143-152.
Fakae, B.B. and Chiejina, S.N. (1993). The prevalence of concurrent trypanosome and gastrointestinal nematode infections in West African Dwarf sheep and goats in Nsukka area of eastern Nigeria. *Vet. Parasitol*. 49: 313-318.
Ferguson, A. and Jarrett, E.E.E. (1975). Hypersensitivity reactions in the small intestine. 1. Thymus-dependence of experimental villous atrophy, *Gut*. 16: 114-117.
Gordon, H.M. (1964). Studies on resistance to *T. colubriformis*: influence of a qualitative reduction in the ration. *Aust. Vet. J*. 40: 55-61.
Griffin, L. and Allonby, E.W. (1979). Disease syndromes in sheep and goats infected with *T. congolense*. *J. Comp. Pathol.* 89: 457-464.
Griffin, L. and Preston, J.M. (1981a). The interaction of *T. congolense and H. contortus* infections in two breeds of goats. 1, Parasitology. *J. Comp. Pathol.* 91: 85–95.
Griffin, L. and Preston, J.M. (1981b). The interaction of *T. congolense and H. contortus* infections in two breeds of goats. 2, Haemotology. *J. Comp. Pathol.* 91: 97-103.
Hansen, J. and Perry, B. (1994). *The Epidemiology, Diagnosis and Control of Helminth Parasites of Ruminants,* International Laboratory for Research on Animal Diseases, Nairobi. pp. 80-92.
Holmes, J.C. (1973). Site selection by parasitic helminths: Interactions, site segregation and their importance to the development of helminth communities. *Can. J.* 51: 333-347.
Holmes, P.H. and MacLean, J.M. (1971). The pathophysiology of ovine ostertagiasis: a study of the changes in plasma protein metabolism following single infections. *Res. Vet. Sci*. 12: 265-271.
Horak, I.G. and Clark, R. (1964). A study of the blood picture of lambs suffering from parasitic gastritis. *Ondersteport J, Vet. Res.* 31: 163-176.
Horak, I.G. and Clark, R. (1966). The pathophysiology of helminth infestations. II. *Oesophagostomum columbianum*. *Ondersteport J. Vet. Res*. 33: 139-159.
International Livestock Center for Africa (ILCA). (1985). Research Report No. 9 (ILCA), Addis Ababa. pp. 45-46.
Jenkins, G.C., Fersberg, C.M., Brown, J.L. and Parr, C.W. (1974). Some haematological observations on experimental *T. brucei* infections in rabbits. *Trans. Roy. Soc. Trop. Med. Hyg.* 68: 154-156.
Kates, K.C. (1943). Over wintering survival on pasture of parasitic stages of some common nematodes parasitic in sheep. *Proc. Helminthol. Soc. Wash*. 10: 23-25.
Kutler, K.L. and Marble, D.W. (1960). Serum protein changes in lambs with naturally acquired nematode infections. *Am. J. Vet. Res.* 21: 445-448.
Mackenzie, P.K.J., Boyt, W.P., Emslie, V.W., Lander, K.P. and Swanepol, R. (1975). Immunosuppression in ovine trypanosomiasis. *Vet. Rec.* 97: 452-453.
Makinde, G.E.O. and Lasisi, O.T. (2004). Helminth and non-helminth causes of cattle, sheep and goat liver condemnations at Ibadan Municipal Abattoir, Nigeria. In: *Proceedings of the 41st Congress of the Nigerian Veterinary Medical Association*, 22nd – 26th November 2004, Vom, Nigeria, pp. 98 -99.
Martin, C.J. and Clunies-Ross, I. (1934). A minimum computation of the amount of blood removed daily by *H. contortus*. *J. Helminthol*. 12: 137-142.
Mbaya, A.W. and Aliyu M.M. (2007). An outbreak of parasitic gastroenteritis among captive gazelles in Maiduguri. In: *Proceedings of the 44th Congress of the Nigerian Veterinary Medical Association*, 22nd – 26th October 2007, Effurun- Delta, Nigeria, pp. 273-276.
Mbaya, A.W., Nwosu, C.O., Aliyu, M.M. and Ahmed, T.A. (2006). A comparative study of gastrointestinal parasites of captive and free-living wild animals in the semi-arid zone of northeastern Nigeria. *Nig. J. Exp. Appl. Biol*. 7: 185-193.
Mbaya, A.W., Aliyu, M.M., Nwosu, C.O., Egbe-Nwiyi, T.N.C. and Kumshe, H.A. (2008a). Studies on the leucocytic response to experimental infection with *Trypanosoma brucei* and *Haemonchus contortus* in red fronted gazelles (*Gazella rufifrons*). *Sahel J. Vet. Sci.* 7: 37-45.
Mbaya, A.W., Aliyu, M.M., Nwosu, C.O. and Egbe-Nwiyi, T.N.C. (2008b). The relationship between parasitaemia and anaemia in a concurrent *Trypanosoma brucei* and *Haemonchus contortus* infection in red fronted gazelles (*Gazella rufifrons*). *Vet. Arhiv.* 80: 46-59.
Mbaya, A.W., Aliyu, M.M., Nwosu, C.O. and Ibrahim, UJ. (2008c). Captive wild animals as potential reservoirs

of haemo and ectoparasitic infections of man and domestic animals in the arid-region of northeastern Nigeria. *Vet. Arhiv* 78 (5): 429-440.

Michael, J.F. (1976). The epidemiology and control of some nematode infections in grazing animals. *Adv. Parasitol.* 14: 355-398.

Morley, F.H.W. and Donald, A.D. (1980). Farm management and systems of helminth control. *Vet. Parasitol*. 6: 105-134.

Murray, P.K., Jennings, F.W., Murray, M. and Urquhart, G.M. (1974). Immunosuppression in trypanosomiasis. In: E.J.L. Soulsby (Editor), *Parasitic Zoonosis: Clinical and Experimental Studies*, Academic press, New Yorke, pp. 133-150.

Nwosu, C.O. (1995). Clinical and immunological changes in red Sokoto (Maradi) goats infected with *T. brucei* and *H. contortus*. (PhD. Thesis). University of Ibadan, Ibadan, Nigeria. 82pp.

Nwosu, C.O., Ogunrinade, A.F. and Fagbemi, B.O. (1996). Prevalence and seasonal changes in the gastrointestinal helminths of Nigerian goats. *J. Helminthol*. 70: 329-333.

Nwosu, C.O., Ogunrinade, A.F. and Fagbemi, B.O. (2001). Clinico-pathological studies of *Haemonchus contortus* infection in red Sokoto (Maradi) goats. *Nig. J. Exp. Appl. Biol.* 2: 157-164.

Nwosu, C.O., Ogunrinade, A.F. and Fagbemi, B.O. (2006). Effect of concurrent *Trypanosoma brucei* infection on haemonchosis in Red (Maradi) goats. *Sahel J. Vet. Sci.* 5: 45-54.

Ogunsusi, R.A. (1978). The epidemiology of trichostrongylid infections of sheep in the Northern guinea savanna of Nigeria (Ph.D Thesis), Ahmadu Bello University, Zaria, Nigeria. pp. 259-260.

Okon, E.D. (1975). Epizootiology of *Strongyle* infections of small ruminants in Nigeria (Ph.D Thesis), University of Ibadan, Ibadan, Nigeria. pp. 232-235.

Onugba, A.A. and Agbede, R.I.S. (2006). Parasites of sheep and goats in residential areas on Ahmadu Bello University Campus, off campus and the University Farm. In: *Proceedings of the 43rd Annual Congress of the Nigerian Veterinary Medical Association*, 6th - 10th November 2006, Minna, Nigeria. pp. 113-117.

Onyali, I.O., Onwuliri, C.O.E. and Ajayi, J.A. (1990). Development and survival of *H. contortus* larvae on pasture at Vom, Plateau State, Nigeria. *Vet. Res. Comm.* 14: 211-216.

Ozerol, N.H. and Silverman, P.H. (1960). Partial characterization of *H. contortus* excretory fluid. *J. Parasitol*. 55: 79-87.

Ozerol, N.H. and Silverman, P.H. (1970). Further characterization of active metabolites from histotrophic larvae of *H. contortus* cultured *in vitro*. *J. Parasitol*. 56: 1199-1205.

Pullan, N.B. (1980a). Productivity of white Fulani cattle on the Jos Plateau, Nigeria. III. Disease and management factors. *Trop. Anim. Hlth. Prod.* 12: 77-84.

Pullan, N.B. (1980b). II nutritional factors. *Trop. Anim. Hlth. Prod.* 12: 203-208.

Radostitis, O.M., Blood, D.C. and Gray, C.L. (1997). *Veterinary Medicine: A Textbook of Cattle, Sheep, Pigs, Goats and Horses*, 8th ed. Saunders, London, pp. 200-201.

Rahman, W.A. and Collin, G.H. (1990). Changes in live weight gain, blood constitutes and worm egg output in goats artificially infected with a sheep derived strain of *H. contortus*. *Brit. Vet. J.* 146: 543-550.

Rogers, W.P. and Somerville, R.I. (1963). The infective stage of nematode parasites and its significance in parasitism. *Adv. Parasitol*. 1: 109-177.

Rowland, B. and Robert, A.J. (1972). Some pathophysiological changes in young lambs experimentally infected with *N. battus*. *Re. Vet. Sci.* 13: 323-329.

Salihu, M.D., Abubakar, M.S., Umar, A. and Yahaya, H.S. (2005). A retrospective study of diseases if livestock at Sokoto, Nigeria. In: *Proceedings of the 42nd – Congress of Nigerian Veterinary Medical Association*. 14th – 18th November 2005, University of Maiduguri, Maiduguri, Nigeria. pp. 33-35.

Schillhorn van veen, T.W. (1973). Small ruminant health problems in northern Nigeria with emphasis on the helminthiasis. *Nig. Vet. J.* 2: 26 -31.

Schillhorn van veen, T.W. (1974). Drought, malnutrition and parasitism. *Nig. J. Anim. Prod*. 1: 231-136.

Schillhorn van veen, T.W. (1978). Haemonchosis in sheep during the dry season in the Nigerian savannah. *Vet. Rec*. 102: 364-365.

Schillhorn van veen, T.W. and Brinckman, W.L. (1975). An evaluation of anthelmintic drenching schemes for lambs on wet season grazing. *Samaru Agric Newslett.* 17: 70-74.

Symons, I.E.A. and Steel, J.W. (1978). Pathogenesis of the loss of production in gastrointestinal parasitism. In: A.D. Donald, W.H. Southcott and J.K. Dineen (eds.), *The Epidemiology and Control of Gastrointestinal Parasites of Sheep in Australia*, Commonwealth Scientific and Industrial Research Organization, Australia, pp. 9-22.

Symons, I.E.A. and Hennessey, D.R. (1981). Cholecystockinin and anorexia in sheep infected by intestinal nematode *T. colubriformis*. *Int. Parasitol*. 11: 55-58.

Soulsby, E.J.L. (1982). *Helminths, Arthropods and Protozoa of Domesticated Animals*, 7th ed. Baillere Tindall, London. pp. 809-810.

Soulsby, E.J.L. (1994). How parasites tolerate their host. *Brit. Vet. J*. 8: 150-153.

Thomas, R.J. and Waller, P.J. (1975). Significance of serum pepsinogen and abomasal pH levels in a field infection of *O. Circumcincta* in lambs. *Vet. Rec*. 97: 468-471.

Turner, J.H, Kates, K.C. and, Wilson, G.I. (1962). The interaction of concurrent infections of the abomasal nematodes, *H. contortus, O. circumcinata* and *T. axei* in lambs. *Proc. Helminth. Soc. Wash*. 29: 210-216.

Urquhart, G.M., Murray, M., Murray, P.K., Jennings F.W.A. and Bates, E. (1973). Immunosuppression in *T. brucei* infections in rats and mice. *Trans. Roy. Soc. Trop. Med. Hyg.* 67: 528-535.

Weatherley, A.J., Grimashau, W.T.R., Shanks, D.J., Charobbier, J.L. and Jones, R.M. (1989). Evaluation of a new Morantel bolus (Morantel Sustained Released Trilaminate) in the control of parasitic gastro-enteritis in first year grazing cattle. *Revue Med. Vet*. 140: 1023-1027.