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Peste des Petits Ruminants* (PPR) — A Review

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Summary

Most papers dealing with Peste des petits ruminants (PPR) are headed by one or the other of the above synonyms and contain a paragraph or so referring to its close similarity to Rinderpest disease of cattle (RP). Some are in the form of preliminary reports (Whitney et al., 1967; Johnson and Ritchie, 1968; Isoun and Mann, 1972; Nduaka and Ihemelandu, 1973) and a few such Banazet, 1973; Dardiri, 1976 (notes on PPR) go into detail. Although none of these can claim comprehensiveness, they often have the advantage of being the authors’ own experiences in the field or experimental cases (Gilbert and Monnier, 1962; Whitney et al., 1967; Johnson and Ritchie, 1968; Rowland and Bourdin, 1970; Rowland et al., 1971; Isoun and Mann, 1972; Nduaka and Ihemelandu, 1973).

Peste des petits ruminants being a relatively new disease, known only in West Africa, lacks comprehensive review articles. This paper attempts a comprehensive review of the available literature on peste des petits ruminants.

Definition

Peste des petits ruminants (PPR) also known as pseudorinderpest of small domestic ruminants, stomatitis of sheep and goats, pneumonia-enteritis complex and kata, is a contagious virus disease of small domestic ruminants. The disease is characterised by pyrexia, catarrhal nasal and ocular discharge, necrotic stomatitis and an intestinal mucosal and lymphoid tissue reactional syndrome resembling rinderpest in cattle. The virus is antigenically and immunologically closely related but not identical to rinderpest virus (RV) but cattle are not clinically affected. Secondary bacterial and parasitic infections may exacerbate the disease.

History and Occurrence

PPR was first reported in sheep and goats in the Ivory Coast, a former French-occupied territory of West Africa, by Gargadennec and Lalanne (1942). In 1956, the etiological virus agent was isolated and identified in Senegal and Benin (Mornet et al., 1956) and was then considered a mutant of rinderpest virus. Later in 1967, outbreaks in goats were reported in the British-occupied Nigeria (Whitney et al., 1967). Johnson and Ritchie (1968) isolated a virus with the characteristics of PPRV. Later Bourdin (1973) reported the disease in Togoland.

The disease then called ‘pneumonia-diarrhoe complex’ had been reported in the Volta and Northern regions of British colonised Ghana (Nyarko and Taylor, unpublished data) and recently outbreaks in sheep and goats have been reported able to expect that the disease occurs in other countries particularly where rinderpest in cattle has been reported.

Properties of the Virus

Morphology

Examining PPR virus grown in embryonic lamb kindney (LEK) cells under the electron microscope, Laurent (1968) hinted on the structure of the virus. Using PPR strain G7F at the 7th
passage at 40°C also grown in LEK cells, Bourdin and Laurent-Vantier 1967 examined the virus at 30,000-40,000 micron diameter in an electron microscope (EM) and observed a more or less rounded virus particles varying in diameter between 150nm and 700nm. Most of them were about 500nm in diameter. There were no filamentous forms seen but each had an envelope 10nm wide with a fringe of projections 10nm high. On rupture, an internal component (nucleocapsid) consisting of a central canal about 18nm in diameter, helical in shape was found.

The structure thus is that of a typical paramyxovirus and it has been proposed that PPRV be classified as a fourth member of the genus Morbillivirus (Gibbs et al., 1979).

Physio-Chemical Properties

(a) Thermal Effect

Thermal inactivation studies have revealed that numbers of the family Paramyxoviridae eg. RV lose their infectivity at 50°C. PPR virus may be expected to conform to this, being a member of the group.

(b) Ether Sensitivity

$10^{5.3}$ TCID$_{50}$ of PPR virus is completely inactivated by 20 percent ether solution in 12hrs at +4°C (Laurent, 1968). This reveals the presence of an essential lipid component in the structure of the virus.

(c) Effect of pH

$10^{5.5}$ TCID$_{50}$ of PPR virus is inactivated at pH3 at room temperature (Laurent, 1968).

(d) Effect of Halogenated Pyrimidines

(5 Fluoro, 5 Bromo 5 deoxyuridine)

These substances inhibit the synthesis of deoxyribonucleic acid (DNA) but when Laurent (1968) infected a LEK cell culture with PPR virus viral repli-

cation was not inhibited after the addition of a solution of 5 iodo -2- deoxyuridine. This shows that the nucleic acid of PPR is not DNA.

(e) The Feulgen Reaction

Laurent (1968) did not find any evidence of the presence of DNA in PPR virus after Feulgen staining of intracytoplasmic and intranuclear inclusion. Instead he found that both inclusions were high in ribonucleic acid (RNA) using methylpyronin and ribonuclease.

Antigenic Properties

PPR virus appears to be antigenically stable because immunogenic types have not been reported. All the strains isolated, up till now, have identified antigenic properties. One major characteristic is its close antigenic relationship with RV. Hamdy et al., (1976) detected two lines in an agar gel precipitation test between PPR virus infected lymph nodes and RV immune serum.

Relationship with other Paramyxoviruses

Mornet et al., (1956) recognised the close relationship between PPR virus and rinderpest virus. Indeed so close was the relationship that for some years PPRV was considered a mutant of RV.

The neutralization of PPRV in vitro using rinderpest immune serum has been reported (Gilbert and Monner, 1962), Hamdy et al (1975, 1976). Hamdy and Dardiri (1976) further proved this relationship by neutralization tests but could not demonstrate cross neutralization. They reported that PPRV and RV were not identical though they were closely related. This was confirmed by Taylor and Abegunde (1979) who, however found a low level of cross neutralization.

Dardiri et al., (1976) showed that PPRV antiserum and PPRV, RV, measles virus (MV), canine distemper virus (CDV) would fix complement thus demonstrating the group relationship.
Cross Protection Between PPRV and RV

Reports on the cross protection antigenic relationship between PPRV and RV have been quite conclusive. Hamdy et al., (1975) concluded that goats immunised against PPR or RP developed complement fixing antibodies in their sera against both the homologous and heterologous virus and they resisted challenge with both virulent viruses.

Hamdy and Dardiri (1976) also reported that white-tailed deer that survived experimental infection with PPRV resisted challenge with virulent RV that was lethal to the control. Cattle immunised with PPRV resist challenge with RV (Dardiri et al., 1976). More recently, Taylor (1979b) protected goats against PPR using attenuated RV. He found that a single inoculation of RV conferred immunity to the goats for a 12 months period.

Host Range

Small Ruminants

*Peste des petits ruminants*, as the name implies, affects small ruminants particularly sheep and goats in West Africa where the disease was first described by Gargadennec and Lalanne (1942). Exposure of American goats to PPRV resulted in an acute to subacute infection with about 25% mortality (Dardiri et al., 1976).

Hamdy et al., (1975) used North American sheep and goats in a series of PPR tests and found them fully susceptible. British sheep and goats are also fully susceptible; the Dorset horn sheep are only mildly affected (Taylor, personal communication). Another small ruminant shown to be susceptible to PPRV is the American white-tailed deer (Odocoileus virginianus) Hamdy and Dardiri, (1976).

Large Ruminants

Reports have been centred on cattle and have been somewhat discrepant. Mornet et al., (1956) and Gilbert and Monnier (1962) found mild pyrexia and oral lesions in calves, but Dardiri et al., (1976) and Taylor and Abegunde (1979) could not produce a clinical disease in cattle. In each case, however, the PPRV stimulated the production of antibodies which rendered the cattle resistant to virulent RV. Dardiri et al., (1976) reported the transmission of subclinical PPR infection from sick goats to cattle. It will be interesting to know if such subclinical PPR infection in cattle occurs in the field.

Other Animals

The occurrence of clinical PPR infection in other animals or its adaptation to laboratory animals has not been reported.

Table 1: Growth in cell culture

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<tr>
<td>Embryo Kidney (LEK)</td>
<td>Bourdin and Laurent-Vantier (1967)</td>
</tr>
<tr>
<td>Primary Lamb</td>
<td>Taylor and Abegunde (1979)</td>
</tr>
<tr>
<td>Kidney (LK)</td>
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<td>Kidney (GK)</td>
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<td>Spleen (BES)</td>
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<tr>
<td>Bovine Embryo</td>
<td>Johnson and Ritchie (1968)</td>
</tr>
<tr>
<td>Kidney (BEK)</td>
<td></td>
</tr>
<tr>
<td>Calf Kidney</td>
<td>Taylor (1979a)</td>
</tr>
<tr>
<td>(BK)</td>
<td>Taylor and Abegunde (1979)</td>
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<td>VER0</td>
<td>Hamdy and Dardiri (1976)</td>
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PPRV produces cytopathic effects (CPE) in various cell cultures (Table 1). The CPE particularly in lamb and goat cells consist largely of small syncytia at the edges of outgrowing cells. These
syncytia enlarge gradually to form larger syncytia containing a ring of numerous nuclei described as "clock face" by Johnson and Ritchie (1968). The cytoplasm becomes increasingly eosinophilic. Large rose-red nuclear inclusions surrounded by a halo are found in association with the syncytia but unlike the CPE of RV described by Plowright and Ferris (1959) no rounded refractile cell clumps have been reported.

Inoculation of cell culture before monolayer formation, produces CPE quickly (Gilbert and Monnier 1962); but where the virus dose is small, the CPE may be small and positive identification may require examination for as long as 14 days.

The formation of CPE in Vero cells has been described by Hamdy et al., (1976). There is stellate syncytia formation slowly spreading from the periphery with cell rounding. Virus multiplication occurs by budding from cell membrane but stops with syncytia formation (Laurent, 1968).

**Epidemiology**

Detailed epidemiology of PPR has not been reported. The role of wild small ruminants in the epidemiology of the disease is unknown.

The disease has been reported, up till now only in West African countries (Gargadennec and Lalanne, 1942; Mornet et al 1956; Whitney et al 1967; Bourdin 1973; Taylor and Abegunde 1979). It is highly possible, however, that other areas may be included as the disease becomes more familiar.

**Transmission**

Infection may be transmitted to susceptible hosts by direct contact and indirectly from virus-laden secretions and excretions. The route of infection has not been determined but PPR can be transmitted experimentally by inoculation. The respiratory route of infection is thought to be a major route for the virus. Movement of infected animals among susceptible animals may be highly significant in the transmission of the disease.

Statistical data regarding morbidity and mortality rates have varied from 10% to 90% (Isoun and Mann, 1972) but outbreaks may range from an uneventful low level infection in enzootic areas to a total eradication of the sheep and goat population in a village (personal observation).

**Clinical Signs**

The clinical features of PPR have been reported by many workers (Gargadennec and Lalanne, 1942; Mornet et al., 1956; Whitney et al 1967; Isoun and Mann, 1972; Nduaka and Ihemelandu, 1973, Dardiri et al., 1976; Hamdy et al 1976).

The incubation period varies from 4 to 10 days, followed by a sudden onset of fever reaching peak values of 104 to 106°F by the third day of pyrexia and dropping to normal after about two days of peak temperature. The affected animals appear ill and are restless; anorexia may be pronounced. The mucous membranes are often congested and bilateral serous nasal discharge is found in most cases which later becomes catarrhal and drips from the nostril. This later dries up, forming crusts at the nostril. The nasal cavity may show small necrotic foci on the mucous membrane.

The conjunctiva is often congested; there may be profuse bilateral ocular discharge which becomes catarrhal, matting the eyelids together when dried. The oral mucosa is often congested with or without salivation but often shows areas of necrosis causing a foetid smell. The tongue and palates may be involved in this way. However, involvement may be limited to congestion of the mucous membranes.

In female animals, the disease may cause ulceration of the labial face of the vulva and there may be abortion in pregnant ones. Diarrhoea is almost constant, leading to dehydration, emaciation and finally prostration, the hind quarters and tail becoming characteristically soiled with faeces. Respiratory signs may include cough, sneezing and dyspnoea. Secondary bacterial complications
may occur causing broncho-pneumonia. Development of labial scabs in animals that survive the infection has been reported (Whitney et al., 1967; Johnson and Ritchie; 1968; Isoun and Mann 1972). This has been conspicuously absent in other reports and in all the outbreaks in Ghana (Personal observation) and may require reinvestigation.

Pathogenesis

A detailed systematic study of the development of PPR has not been reported. The route of infection is favoured to be respiratory, and Johnson and Ritchie (1968) failed to isolate any virus from pooled samples of spleen, liver and kidney within 30 days observation and a further 10 days post passage. On the contrary, they isolated virus from the mesenteric and mediastinal lymph nodes, 16 days after inoculation. Scrapings from illegal mucosa yielded virus 5 days post inoculation in unstained preparations.

Hamdy et al., (1976) recovered PPRV from blood samples taken 3 and 5 days post inoculation and from lymph nodes from 2 deer after autopsy. Isolation of PPRV from epithelial scrapings from the large intestine, lungs and to a lesser extent, the small intestines of dead sheep and goats has been reported (Taylor and Abegunde, 1979). Whether PPRV exhibits latency in some hosts is not known.

Pathology

PPRV causes a generalised systemic infection. The pathology of PPR has been described by a number of workers (Whitney et al., 1967; Rowland et al, 1969; Rowland and Bourdin, 1970; Rowland et al., 1971). Following an incubation period of 4 to 10 days early lesions consisting of a few white foci appear in the epithelium of the palate and the dorsum of the tongue. These develop to form extensive areas of superficial erosions and may involve the whole oral cavity, extending to cover the pharynx and down to the upper 3rd of the oesophagus. Histologically, the lesions are focal necrosis of the stratified squamous epithelium originating from the stratum granulosum. These extend rapidly and soon as mass of necrotic debris cover the intact stratum germinativum which itself is almost never affected. Eosinophilic intracytoplasmic inclusion bodies are often found in degenerating epithelial cells. Gross lesions in the large intestine are dramatic with “Zebra-striping” involving the caecum, colon and the rectum.

Histologically, there are focal areas of congestion and haemorrhage in the submucosa. The lymphoid follicles show areas of necrosis. Elsewhere in the carcass lesions are less dramatic. The payers patches in the small intestine may be grossly prominent with oedema and the lungs may be pneumatic from bacterial infection.

Immunization

It has been recognised that animals that recover from PPR develop an active resistance against a challenge that killed susceptible controls (Whitney et al, 1967; Hamdy and Dardiri, 1976). However, no specific vaccine has been developed against PPR.

Reports on attenuation of PPRV have been somewhat descriptant. Gilbert and Monnier (1962) passaging PPRV in LEK cells at 37°C and 40°C to look for attenuation reported that on the 12th passage at 37°C 2/2 goats had only low grade pyrexia and both survived. On the other hand, Benazet (1973) passaged PPRV at 32, 42 and 65 times in LEK cells and found considerable residual virulence at all levels.

Rinderpest vaccines have been examined by various workers for use against PPR due to the cross relationship between the two viruses (Bourdin et al., 1970; Bourdin, 1973; Taylor, 1979b), and the results have been encouraging.

Indeed, Taylor (1979b) reported that goats vaccinated with attenuated rinderpest vaccine were protected against PPR for 12 months and that although he neutralising antibodies developed were
at first directed against rinderpest virus, exposure to PPRV gave a secondary response resulting in high antibody levels against both RV and PPRV.

References


Received for publication on 11th August, 1981.
Characterization of Nigerian Strains of Infectious Bursal Disease Virus of Chickens

Histopathological Changes Occurring in Field Outbreaks

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Summary

Lesions in the bursa were characterized by massive destruction of lymphocytes, generalized edema, reticular cell hyperplasia and infiltration by heterophils. Sections of the bursa taken on the 6th and 8th week after the onset of the outbreak showed that some follicles were replaced by glandular tissue, some disintegrated, while the regenerating follicles manifested incomplete lymphocyte repopulation. Severe vascular degeneration and congestion were seen in the kidney. Lesions in the spleen and thymus were necrosis, loss of lymphocytes and hyperplasia of reticular cells. The proventriculus showed areas of congestion, necrosis and desquamation. In the muscles of the thigh and chest, haemorrhages, congestion, necrosis and edema were present. The liver, cecal tonsil and pancreas showed no lesions.

Introduction

Infectious bursal disease (IBD) was first described by Cosgrove (1962), and since then the disease has been reported in different parts of the world (Faragher, 1972). The first report of a confirmed outbreak of IBD in Nigeria was by Onunkwo (1975) in Plateau State. Since then, despite the fact that reports of field outbreaks of the disease have been frequent in this country, there has been no histopathological study of the disease (Onunkwo, 1978). However, Helmboldt and Garner (1964), Cheville (1967), Cho and Edgar (1972), and Henry et al., (1980) have described the histopathology of IBD in other parts of the world. This paper is an attempt to describe the histopathological characteristics of naturally occurring cases of IBD in Nigeria. Apart from its diagnostic significance, it provides a basis for comparing the lesions of IBD here with those occurring in other places.

Materials and Methods

In a project designed to investigate the existence of IBD in the eastern states of Nigeria, six outbreaks were confirmed in different farms by history of the disease in the flocks, clinical signs and mortality pattern, gross necropsy lesions, histopathology, virus isolation and identification, serology and chicken pathogenicity tests. The affected chicks were commercial birds imported into this country and reared on deep litter system. Their ages ranged between four and ten weeks at the time of the outbreaks. Samples of the bursa, kidney, spleen, thymus, cecal tonsil, pancreas, liver, proventriculus, and muscles of the chest and thigh were collected from chicks that died of the disease. The proventriculus and the muscle samples were collected from grossly congested or haemorrhagic areas. The bursa only was collected from some of the survivors that were sacrificed at the 6th and 8th week post infection (PI). These organs were fixed in 10% formol saline for at least twenty-four hours, processed and embedded in paraffin wax. Sections 5 um thick, were cut, stained with haematoxylin and eosin (H & E) and examined under the microscope.

Results

The lesions in the bursa were very severe. The internal epithelium of the plicae was hyperplastic, edematous and infiltrated by heterophils. Some areas
were either desquamated or convoluted. The spaces between the follicles and the epithelium and the interfollicular spaces showed numerous heterophils and some plasma cells, severe edema and reticular cell fibroplasia (Fig. 1). Marked congestion of the interfollicular and cortical blood vessels often occurred (Fig. 2). In the follicles some of the medulla contained cystic cavities containing eosinophilic fluid, cellular debris and necrotic lymphocytes, heterophils and plasma cells. The follicles were infiltrated by heterophils and plasma cells. There was marked destruction and loss of the lymphocytes, thereby exposing hyperplastic reticular cells (Fig. 3). Very few lymphocytes, most of which were necrotic remained in the cortex. The bursal muscle layer was edematous, necrotic and infiltrated by heterophils. In some sections, no heterophils were seen in the entire bursa. The follicular corticomедullary junction was often lined by hyperplastic bursal epithelial cells. Intrafollicular edema was also present.

Fig. 1: IBD affected bursa showing interfollicular edema (e), interfollicular fibroplasia (f), and cystic cavities (c). H&E, x 100.

Fig. 2: IBD affected bursa showing congestion of interfollicular blood vessels (k) and hyperplasia of the epithelial cells at the corticomeđullary junction (arrows). H&E, x 100
In sections of the bursa collected six weeks PI, the bursal epithelial cells at the corticomедullary junction had replaced the reticulum with glandular tissues in some follicles (Fig. 4). The regenerating follicles were reduced in size and the cortex contained a thin external rim of small lymphocytes. Plasmablasts, plasma cells and lymphoblasts were present inside the follicles. Some of the follicles had completely disintegrated and were being replaced by heavy interfollicular fibroplasia. The bursa collected at eight weeks PI contained both the glandular and disintegrated follicles. The regenerating follicles had many empty spaces and small lymphocytes were scattered in the cortex and the medulla. Hyperplasia and convolution of the internal bursal epithelium persisted.

Fig. 3: One of the follicles in Fig. 1 showing loss of lymphocytes and hyperplasia of the reticular cells (H), intrafollicular edema (arrows) and heterophilic infiltration (h). H.&E. x 400.

Fig. 4: IBD affected bursa six weeks PI showing glandular follicles (G), disintegrated follicles (D), and regenerating follicles (R). H.&E. x 100.
Fig. 5: IBD affected kidney showing congestion (K), vacuolar degeneration of the epithelia of the tubules and collecting ducts (V). H.&E. x 400.

Fig. 6: IBD affected spleen. Sheathed arteriole (A) showing hyperplasia of the reticular cells (arrows). H.&E. x 400.

Fig. 7: IBD affected spleen. Germinai Centre (GC) showing necrosis of lymphocytes (arrows) and the presence of lymphoblasts (L). H.&E. x 400.
The epithelia of the renal tubules and collecting ducts were detached from the basement membranes and showed severe vacuola degeneration (Fig. 5). Massive congestion, haemorrhage and heterophilic infiltration were also present in the kidney.

There was congestion of the sinuses in the spleen and hyperplasia of the reticular cells around the sheathed arterioles (Fig. 6). Most of the lymphocytes in the germinal centres were necrotic (Fig. 7). The centres contained some reticular cells in the middle and few lymphoblasts. The periarteriolar lymphoid sheaths (PLS) were depleted of lymphocytes and contained many lymphoblasts and reticular cells (Fig. 8). The diffuse lymphoid areas of the red pulp showed necrotic lymphocytes, some erythrocytes.

In the proventriculus, the mucosa, the glands, the muscle layer and the serosa were congested and often haemorrhagic. Some of the glandular cells were necrotic and desquamated into the lumina (Fig. 9). Slight heterophilic infiltration occurred in the interglandular connective tissues.

Sections of the muscles of the thigh and chest showed haemorrhages and severe congestion. The muscle fibers were necrotic and separated by edema spaces containing many heterophils (Fig. 10).

Lesions in the thymus were necrosis, loss of lymphocytes and hyperplasia of reticular cells.

The cecal tonsils, liver and pancreas had no lesions.

---

Fig. 8: IBD affected spleen, PLS (PS) showing loss of lymphocytes, the presence of lymphoblasts (arrows) and hyperplasia of reticular cells (H). H&E. x 400.

Fig. 9: IBD affected proventriculus showing congestion (arrows), necrosis and desquamation of the glandular cells (D). H&E. x 400.
**Discussion**

The lesions in the bursae were consistent, and diagnostic, and in agreement with the findings of previous workers (Helmholot and Garner, 1964; Cheville, 1967; Chuo and Edgar, 1972; and Henry et al., 1980). Failure to find heterophils in some sections might be due to their destruction by reticular macrophages. The persistence of the lesions for over six and eight weeks PI confirms the observations of Winterfield et al., (1972). The loss of follicles due to the formation of glandular tissues, obliterations and incomplete lymphocytic repopulation, constitute a permanent damage to the bursae. Such damage in chicks under three weeks of age has significant immunosuppressive effects on subsequent vaccinations and increase the susceptibility to infections (Giambrone, 1979). It is therefore possible that such early IBD attacks may be responsible for some cases of vaccine-break in this country. In investigating these cases, the persistence of the bursal lesions will make it possible to classify chicks that are serologically positive into those that have damaged bursae and those that have normal bursae. The former might have suffered clinical IBD infection while the latter would have acquired their immunity by subclinical infection or by vaccination.

Histopathological lesions in the proventriculus and the muscles of the thigh and chest, have not been described by the previous workers, although the gross lesions have been documented (Hitchner, 1978). The lesions in the proventriculus occurred mainly in chicks under six weeks old while those in the muscles were quite common. The changes in these organs may be significant in differential diagnosis.

In the spleen, no heterophils were present but reticular cell hyperplasia was common. This is contrary to the findings of Henry et al. (1980). The lesions described in this study are similar to the observations of Cheville (1967), Cho and Edgar (1972) and Helmboldt and Garner (1964). While the PLS had lost most of the lymphocytes and contained mainly reticular cells and lymphoblasts, the germinal centres contained many but necrotic lymphocytes. This is probably due to the differences in the origin and functions of the lymphocytes in these two areas of the white pulp.

The heterophilic infiltration present in the kidneys was also reported by Cho and Edgar (1972) and Helmboldt and Garner (1964), but not by Henry et al. (1980). While the consistent presence of hyperemia is in agreement with the observations of Cho and Edgar (1972) and Henry et al. (1980), no such lesion was reported by Helmboldt and Garner (1964). Cho and Edgar (1972), and Helmboldt and Garner (1964) recorded the presence of casts in the tubules. In
this study, similar to most of the findings of Henry et al. (1980), no casts were present. The severity of the kidney lesions associated with the high mortalities (11.5%–33.5%) recorded in these outbreaks tend to confirm an earlier observation that the kidney lesions may be the major cause of death in some cases of IBD (Onunkwo, 1978).

In this country, most of the chicks in the commercial farms are imported from the Western countries. However, whether the IBD virus was imported or is indigenous, the fact that the results of this study do not differ significantly from those carried out in other places, indicates that our local strains of IBD virus may closely resemble other strains elsewhere. It is noteworthy that no significant differences have been described among the various strains of IBD virus known to date.

Acknowledgement

We are grateful to the Senate Research Grand Committee of the University of Nigeria, Nsukka, for sponsoring this investigation. We thank Dr. D.N. Ezeasor of this Faculty for his assistance in microphotography.

References


Received for publication on 6th April 1981.
Characterization of Nigerian Strains of Infectious Bursal Disease Virus of Chickens

Clinicopathological Manifestations of Naturally Occurring Field Outbreaks

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Summary
Infectious bursal disease (IBD) was diagnosed and confirmed in ten different outbreaks in Nigeria. The major clinical signs were sleepiness, drooping of the head and wings, and diarrhoea. Affected chicks were 4-10 weeks old and the most consistent gross necropsy lesions were enlargement of the bursa of Fabricius and kidney, and hemorrhages in the muscles of the thigh and chest, and in the duodenum. Morbidity and mortality ranged between 70% to 95% and 11.5% to 33.5% respectively. All the sera collected from the surviving chicks two weeks after the disease was manifested showed precipitation lines with known IBD virus (IBDV) antigen in agar-gel diffusion precipitation test (AGDT). Bursal suspensions were positive in AGDT using known IBDV antiserum. Chicken pathogenicity tests reproduced typical IBD in susceptible chickens. The clinicopathological properties of the disease in Nigeria are compared with those of other places.

Introduction
Infectious bursal disease (IBD) is a highly contagious viral disease of chicks which was first described by Cosgrove (1962) in Gumboro area of Delaware, U.S.A. Since then, IBD has been described in many parts of the world including some African countries like Chad (Provost et al., 1972), Zambia (Sharma, 1978), Senegal (Sagna, 1977), South Africa (Coetzee, 1970), and Mauritania (Ba and Chamoiseau, 1977). In Nigeria, Ojo et al. (1973) first described an IBD-like disease but it was not until two years later that the first confirmed outbreak of IBD was reported by Onunkwo (1975). Since then, reports of severe outbreaks of unconfirmed IBD have been so frequent among our poultry farmers and field veterinarians that it has become very necessary to conduct a detailed study of the clinicopathological characteristics of confirmed natural outbreaks of this disease. Apart from its immense diagnostic importance, it forms a basis for comparing the clinicopathological features of IBD here with those of other places.

Materials and Methods

Flock History: The ten affected poultry farms were located in different parts of the eastern states of Nigeria. All the chickens were reared in deep litter houses and unvaccinated against IBD. Some were pullets and others were cockerels or broilers. The breeds affected were Harco, Babcock, Gold Link, Shaver and Yaffer.

Clinical Signs: The disease appeared suddenly in the flocks and spread rapidly. The main clinical signs were droopiness and watery diarrhoea. Infected chicks frequently had ruffled feathers and remained in one place with drooping head and wings, and eyes closed. The feces was watery, whitish, yellowish, greenish or a combination of any two of the colours. There was a drop in feed and water consumption and, in severe cases, prostration was followed by death. Mortality was higher initially and the course of the disease was 6-10 days. Morbidity was 70% to 95% while mortality rates were 11.5%-33.5%. Reoccurrence of the disease was recorded in three of the in-
fected farms and in one case the disease re-occurred twice in subsequent batches. Spread of the disease to other pens in the same house was common.

**Histopathology:** The bursa, kidney, spleen, thymus, cecal tonsil, pancreas, liver, proventriculus and muscles of the chest and thigh were collected and processed for histopathology as already described (Okoye and Uzoukwu, 1981).

**Bacteriological and Parasitological Examination:** Intestines, spleen, bursa of Fabricius and kidney were collected for routine bacteriological or parasitological examinations.

**Agar-gel Diffusion Precipitation Tests:** About 3ml of blood was collected from each of ten surviving chickens in each of the affected farms two weeks after the onset of the disease. The serum was collected and inactivated at 56°C for 30 mins. and used for AGDT.

In each outbreak, the bursa of Fabricius and the spleen of dead chickens were collected separately, weighed and homogenized with equivalent volumes of phosphate buffered saline (PBS). They were centrifuged at 3000rpm for 30 mins. The supernatant fluid was collected and used for virus identification in AGDT.

The agar used was a modification of the one used by Ley et al. (1979). It consisted of 1% lonaar No.2 in 8% NaCl, and 0.01% Sodium azide at pH 7.9. Six wells, each 6mm. in diameter and arranged 6mm. apart and equidistant from a central well, were used in the test which was carried out in a humid chamber at room temperature.

Reproduction of the Disease in Susceptible Chicks: In each outbreak, samples of freshly collected bursa of Fabricius of dead chicks were homogenized in PBS-antibiotic solution as a 20% suspension and centrifuged at 3,000rpm for 30 min. The supernatant fluid was administered intraocularly at the rate of 0.05ml per bird to twenty 4-week old susceptible chickens (test group). Another group of ten susceptible chickens of the same age were used as untreated control. The two groups were housed separately and observed every 24 hours for ten days. Two chicks from the test group and one from the untreated control were sacrificed every day for necropsy, histopathology, virus isolation and identification, and serology for ten days post infection (PI).

**Results**

The ages, mortality rates, breeds and types of the affected chicks are shown in Table 1.

**Necropsy Lesions:** The bursa was frequently enlarged (Fig. 1) with pro-

---

**Table 1: Showing breed, type, age and mortality in infected flocks.**

<table>
<thead>
<tr>
<th>Breed</th>
<th>Type</th>
<th>Age in Weeks</th>
<th>Mortality Rate %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Babcock</td>
<td>Pullet</td>
<td>4</td>
<td>23</td>
</tr>
<tr>
<td>Yaffer</td>
<td>Pullet</td>
<td>4</td>
<td>20</td>
</tr>
<tr>
<td>Harco</td>
<td>Broiler</td>
<td>7</td>
<td>33</td>
</tr>
<tr>
<td>Shaver</td>
<td>Pullet</td>
<td>10</td>
<td>30</td>
</tr>
<tr>
<td>Harco</td>
<td>Cockerels</td>
<td>9</td>
<td>11.5</td>
</tr>
<tr>
<td>Gold Link</td>
<td>Broiler</td>
<td>8</td>
<td>33.5</td>
</tr>
<tr>
<td>Babcock</td>
<td>Pullet</td>
<td>5</td>
<td>26</td>
</tr>
<tr>
<td>Harco</td>
<td>Broiler</td>
<td>6</td>
<td>32</td>
</tr>
<tr>
<td>Yaffer</td>
<td>Broiler</td>
<td>8</td>
<td>29.5</td>
</tr>
<tr>
<td>Shaver</td>
<td>Pullet</td>
<td>6</td>
<td>24</td>
</tr>
</tbody>
</table>
minent plicae that appeared creamy, congested or hemorrhagic. The bursal cavity contained yellowish gelatinous exudate, bloody fluid or caseous plugs. Different degrees of atrophy were seen in some bursa. The kidneys were enlarged, congested or hemorrhagic and the tubules were conspicuously distended. Lesions in the spleen were tiny grey spots on the serosal surface. Hemorrhages were observed on the mucosal surface of the proventriculus-gizzard junction. Hemorrhagic enteritis was common especially at the duodenum. The carcasses were dehydrated but otherwise in good body condition. Hemorrhages in the muscles of the chest and thigh were consistent (Fig. 1).

Histopathology: The histopathological lesions have been described and discussed (Okoye and Uzoukwu, 1981).

Agar-gel Precipitation Tests: Results appeared in 36-48 hours. All the serum samples collected from the outbreaks were positive in the test done with known IBDV antigen, positive control (known) antiserum and negative control antiserum (normal serum). Using the same design, the virus was identified in the bursal suspensions where they gave precipitation lines with known IBDV antiserum. The splenic suspensions gave no precipitation lines.

Chicken Pathogenicity Tests: All the chicks that were given drops of the 20% bursal suspensions in the eyes showed typical clinical and necropsy signs of IBD within three days post infection. Bursal lesions were characteristic histopathologically. IBDV was isolated and identified by AGDT and by serology. All the control chicks were normal.

Bacteriological and parasitological examinations were negative.

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Fig. 1: Post-mortem lesions of a 4-week old pullet that died in one of the field outbreaks of IBD. Note the swollen bursa (A) and the hemorrhages (B) in the muscles of the thigh and breast.
Discussion

The confirmation of these cases was based on clinical signs, necropsy lesions including the histopathology, isolation and identification of IBDV, serology and reproduction of the disease in susceptible chicks. These are probably the first confirmed cases of IBD in the eastern states of Nigeria.

Many of the clinicopathological properties observed in this study are similar to those reported by earlier workers (Cosgrove, 1962; Cho and Edgar, 1972; Hirai et al., 1973; Onunkwo, 1975; Luthgen, 1969; and Hitchner, 1978). But contrary to the findings of Luthgen (1969) and Hitchner (1978) and in agreement with the observations of Onunkwo (1975), picking of the vent was not seen in this study. Similar to the report of Onunkwo (1975) but contrary to the finding of Cho and Edgar (1972), Luthgen (1969), Hirai et al. (1973) and Hitchner (1978), no trembling or tremour was noticed in our affected chicks.

Hemorrhages on the mucosal surface of the proventriculus-gizzard junction were not consistent and appeared more in chicks of 4-6 weeks old. Failure to identify the virus in the splenic suspensions is in agreement with a previous finding that the virus is present in the spleen only at the initial viremic stage of the disease (Ide, 1975 and Vindevogel et al. 1976). Contrary to the observations of Onunkwo (1975), hemorrhages in the thighs and chest were consistent lesions.

The mortality rates recorded in these outbreaks are comparatively high. This may be due to the fact that IBD was appearing in the farms for the first time. It may be also that the Nigerian strains of IBDV are comparatively more virulent than many other strains. Onunkwo (1975) recorded a mortality rate of 43.8%.

The reoccurrence of IBD in infected farms has been reported by previous workers (Hitchner, 1978). This persistence is mainly because IBDV is resistant to many chemical antimicrobial agents and to adverse weather conditions and therefore survives for a long time in infected litter and premises. In Nigeria, the situation is complicated by the scarcity and inhibitory prizes of IBD vaccine. The disease is new and it is difficult to convince some farmers to budget for IBD inoculation until they experience the outbreak in their farms. On the basis of the data available here, and based on the design of the present experiment, it is difficult to suggest that either breed or type of chicken has any influence on the susceptibility and severity of IBD, because of the differences in management in the various farms. IBD has not been diagnosed in our indigenous chicks but the nature of the resistance has not been determined especially since IBD is a new disease in this environment.

Acknowledgement

The authors are grateful to Dr. O. Onunkwo of National Veterinary Research Institute, Vom, for supply of literature. We are also grateful to the Senate Research Grant Committee of the University of Nigeria, Nsukka, for sponsoring this investigation.

References


Received for publication on 1st September 1981.

Table 1: Prevalence of Gumboro Disease around Zaria, Nigeria

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Prevalence</th>
</tr>
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<tbody>
<tr>
<td>Number of chicken in the flocks</td>
<td>27,546</td>
</tr>
<tr>
<td>Number of flocks visited</td>
<td>1,116</td>
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<tr>
<td>Number of flocks affected</td>
<td>240</td>
</tr>
<tr>
<td>Number of flocks with Gumboro</td>
<td>229</td>
</tr>
<tr>
<td>Disease in affected flocks</td>
<td>237</td>
</tr>
<tr>
<td>Number of chicken affected</td>
<td>179</td>
</tr>
<tr>
<td>Number of chickens affected</td>
<td>137</td>
</tr>
<tr>
<td>Number of dead</td>
<td>85</td>
</tr>
<tr>
<td>Age of affected chickens</td>
<td>3-12 weeks</td>
</tr>
<tr>
<td>Morbidity rate</td>
<td>1.49%</td>
</tr>
<tr>
<td>Mortality rate</td>
<td>14.7%</td>
</tr>
</tbody>
</table>
The outbreak of IBV in chicken farms has been reported in many countries. IBV is a highly contagious virus that can be transmitted to other chickens. The virus can be spread through contact with infected feces, respiratory secretions, or contaminated feed and water.

Acknowledgments

The work described in this paper was supported by the National Institutes of Health (NIH) and the Department of Agriculture (USDA). We would like to thank Dr. X.Y. Zhang for his assistance in data collection and analysis.
Infectious Bursal Disease (Gumboro Disease) in Poultry: Case Report

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Summary

Infectious bursal disease was observed in 3-8 weeks old chicks in two hundred and eighty flocks containing over 27,000 chickens in Zaria, Nigeria, between October, 1976 to March, 1977. The mortality and morbidity rates were 14.7% and 31.4% respectively.

Incidence of infectious bursal disease was found to be increasing through the years. Most of the cases reported were in imported breeds from different sources. Economic loss is now assuming significant role on poultry industry in Nigeria, and such losses may extend to other developing countries that are dependent on imported breeds of chicken.

Introduction

Poultry population in Nigeria is estimated to be 83 million (FAO Animal Health Year Book, 1971). This constitutes the largest animal protein source in this country.

The industry has contributed immensely to the human society not only as a source of farm income but also as a source of highly needed animal protein in form of eggs and meat.

Infectious poultry diseases like Newcastle disease, fowl typhoid, fowl cholera, fowl pox, have been recognised for several years in Nigeria and in other countries. Other emerging diseases like infectious bursal disease and Marek’s Disease previously unnotice have recently started to produce high losses in poultry industry.

Gumboro disease was first described by Cosgrove in 1962. The disease was first reported in Nigeria by Ojo and others and was subsequently confirmed by Onunkwo. This paper reports the incidence of the disease in northern area where the greatest potential for livestock production lies.

Prevalence of the Disease Around Zaria Area

Between October 1st 1976 and March 20th 1977, 280 flocks with 27,546 chicken were visited during the avian ambulatory clinic in Zaria, Nigeria (Table 1). These were mainly back-yard poultry type farming with few large commercial poultry farms. Field diagnosis based on clinical signs and post mortem lesion were carried out on the farm.

Table 1: Prevalence of Gumboro Disease around Zaria, Nigeria

<table>
<thead>
<tr>
<th></th>
<th>Percentage</th>
<th>Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of chicken in the flocks visited</td>
<td>27,546</td>
<td></td>
</tr>
<tr>
<td>Number of flocks visited</td>
<td>280</td>
<td></td>
</tr>
<tr>
<td>Number of flocks affected</td>
<td>17</td>
<td>6.07</td>
</tr>
<tr>
<td>Number of chicken in affected flocks</td>
<td>2,378</td>
<td>8.63</td>
</tr>
<tr>
<td>Number of chicken affected</td>
<td>747</td>
<td>2.71</td>
</tr>
<tr>
<td>Number dead</td>
<td>351</td>
<td>1.27</td>
</tr>
<tr>
<td>Age of affected chicken</td>
<td>3–8 wks</td>
<td></td>
</tr>
<tr>
<td>Morbidity rate</td>
<td>31.4%</td>
<td></td>
</tr>
<tr>
<td>Mortality rate</td>
<td>14.7%</td>
<td></td>
</tr>
</tbody>
</table>
The disease occurred predominantly in 3-8 weeks old chicks; Two outbreaks of the disease occurred at ages between 10-12 weeks. There was no vaccination history against the disease in these farms.

Affected chicks showed typical clinical signs. The disease started suddenly, the affected chicks were depressed, anorexic, reluctant to move, ruffled feathered, with droopy appearance and pale yellow diarrhea.

The disease usually spread rapidly within the flocks and there was an average mortality and morbidity rate of 14.7% and 31.4% respectively. Mortality increased rapidly, peaked on the 3rd or 4th days of occurrence and then started declining.

The most common gross lesion was massive haemorrhage and darkened discoloration of the thigh and breast muscle. The kidney was enlarged, the tubules were prominent and the bursa of fabricius was oedematous enlarged haemorrhagic. Bacterial examination of kidney and bursa of fabricius in all cases were negative.

Discussion

Gumboro disease as described showed evidence of contagion. It probably was spread through fomites and from bird to bird. During all the reported outbreaks broad spectrum antibiotics, vitamins and mineral mixtures were not effective. No cases were found in indigenous breed. This supports the possibility of impor-

tation of the disease in day old birds. However, poultry industry in Northern part of Nigeria is getting increasingly dependent on rapidly growing and high egg producing imported birds. Considering the mortality rate and the proportions of imported versus indigenous birds. Gumboro disease should be considered as one of the major poultry diseases in this country and possibly in other countries dependent on imported breeds of chicken. This is particularly so since the mortality rate obtained was up to 14.7% whereas Edger et al. reported losses as being less than 0.7% in vaccinated birds. This also calls for increase in vaccine production on a scale similar to what obtains in other infectious poultry diseases.

Awareness by farmers of the prevalence of the disease in the country and the need for vaccination, adequate sanitation and restriction of movement of people, animals and fomites will also help to curb the disease.

References


Received for publication on 1st September, 1981.
Assessing Animal Health and Disease Trends through Meat Inspection in Nigeria

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and

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Abstract

The general picture of food animal health and disease in Nigeria was estimated by analyzing data on meat inspection from 1971 to 1979.


Though the prevalence rates calculated on the basis of figures from the various abattoirs over the years are very much lower than those which have been obtained in specific abattoir surveys and also from those obtained from the few abattoirs where conditions have permitted efficient meat inspection and recording of pertinent information, the data indicate that the livestock industry is plagued by a host of infectious diseases and parasites. The study revealed that many food animals, particularly indigenous cattle, are extremely lean or emaciated at slaughter. This is indicative of poor health and tends to explain previous observations on the very low dressing percentage of local breeds of cattle, which is generally below 50%.

In the absence of sufficient and reliable data from a variety of possible sources of information and despite their inadequacy, abattoir returns may still be the best available indicator of the magnitude of animal health and disease in Nigeria.

Introduction

Sound policy decisions and planning for the promotion of animal health and production depend largely on the availability of accurate and adequate information on animal diseases. The sources of data for animal disease assessment are many and depending upon the situation may include some or all of these: animal owners or farmers, veterinary practitioners or clinicians, large and small animal clinics, veterinary diagnostic laboratories, disease control agencies, dairies, sentinel farm systems, national surveys, field studies, and abattoirs.

Reports of disease outbreaks in the field are irregular, often inadequate and unsuitable for use in ascertaining the picture of animal health problems. Abattoir antemortem and postmortem returns have been reported routinely, quite extensively and on a regular basis since meat inspection became the responsibility of the Federal Ministry of Agriculture and Natural Resources in 1968.

This paper is an attempt to make
use of accumulated abattoir data in assessing the general trend of animal health and disease in Nigeria.

Materials and Methods

The monthly and annual reports on meat inspection of the various abattoirs and slaughter slabs for the fiscal years 1971 to 1979 were compiled and tabulated at the Epidemiological Unit of the Federal Livestock Department, Kaduna, Nigeria. The prevalence rates of diseases/conditions were expressed in percent but for the purpose of this report the rates have been stated in cases per 1000 or 10,000 animals slaughtered. Tests for independence of the prevalence rates over the years and among the various abattoirs were calculated by chi-square.

Results and Discussion

The diseases and conditions observed during ante and post mortem inspection of food animals in Nigeria from 1971 to 1979 are tabulated in Tables 1-3 and in Figure 1. The total number of food animals slaughtered are presented in Table 4.

As indicated in Tables 1-3 the prevalence rates vary over the years. The chi-square values were significant at the $x = 0.005$ level. However, the prevalence rates do not show a gradual increase or decrease over time except for the abrupt drop in the prevalence of bovine cysticercosis in 1974/75 (Table 2 and Figure 1) and the sudden upburst of hydatidosis in camels in 1976/77 and of fascioliasis in 1975/76 (see Table 2 and Figure 3). Significant variations were also noted in the prevalence rates from one abattoir to the other. The observed variations could not be attributed to any real increases or decreases of the diseases on the basis of time and place but to the efficiency of meat inspection and recording. Thus in those years when meat inspection was given greater attention, the recorded prevalence of certain diseases was higher in comparison to those obtained in times when meat inspection was somewhat lax. This observation is exemplified by the comparatively higher reported prevalence rates of cysticercosis bovis in the years 1971 to 1974; of cysticercosis cellulosa in 1978, of hydatidosis of camels in 1976/77, and of fascioliasis of cattle in 1975/76 (see Tables 1-2 and Figures 2-3). During these years, abattoir surveys were conducted on the prevalence of the particular diseases.

The prevalence rates are generally higher in abattoirs where conditions permit efficient meat inspection and recording of data and lower in others particularly small abattoirs and slaughter slabs, where meat inspection facilities and trained meat inspectors are wanting. The prevalence of the diseases and conditions reported for one abattoir with rather strong reporting as shown in Figure 1 illustrate this finding. The number of cases is definitely higher than the average derived from the various abattoirs which includes those which are operating under substandard conditions. The reason for the greater number of cases recorded is plainly due to the better conditions obtaining in the abattoirs which allow more efficient meat inspection and recording of pertinent data.

The influence of efficiency of meat inspection on detecting diseases is confirmed by the higher prevalence rates reported in specific abattoir surveys. Thus the prevalence of cysticercosis bovis was reported as ranging between 5.3% and 18.4% at Bauchi abattoir in Northern Nigeria (Babalola, 1976); between 3 to 14.6% in Nigerian cattle (Osiyemi, 1976); and 19.23% in 1975 over Nigeria (Belino, 1975). In other studies, the prevalence of hydatidosis in camels was 57.5% in 1979 (Dada and Belino, 1979) and the prevalence of fascioliasis in cattle was 11.03% in 1976 and 20.75% in 1977 at the Animal Products Laboratory Abattoir of the Ahmadu Bello University (Lombin & Belino — unpublished data).

Table 3 and Figure 1 show a substantial number of degenerative, dropiscal, septic and inflammatory conditions.
<table>
<thead>
<tr>
<th>Year</th>
<th>Disease</th>
<th>Cases per 10,000 animals slaughtered</th>
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</thead>
<tbody>
<tr>
<td>1979</td>
<td>Foot and Mouth Disease</td>
<td>38</td>
</tr>
<tr>
<td>1978</td>
<td>Peste</td>
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</tr>
<tr>
<td>1977</td>
<td>Anthrax</td>
<td>22</td>
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<tr>
<td>1976</td>
<td>Avian Influenza</td>
<td>15</td>
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<tr>
<td>1975</td>
<td>Bluetongue</td>
<td>6</td>
</tr>
<tr>
<td>1974</td>
<td>Rabbit Babes</td>
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</tr>
<tr>
<td>1973</td>
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<td>4</td>
</tr>
<tr>
<td>1970</td>
<td>Rabbit Babes</td>
<td>15</td>
</tr>
</tbody>
</table>

Table 1: Infectious Diseases Observed on Meat Inspection in Nigeria, Fiscal Years 1971-79

Note: The table includes animal diseases observed in Nigeria during the fiscal years 1971-1979. The diseases listed are Bluetongue, Anthrax, Avian Influenza, Rabbit Babes, and Foot and Mouth Disease. The cases are recorded per 10,000 animals slaughtered.
<table>
<thead>
<tr>
<th>Animal</th>
<th>1971/72</th>
<th>72/73</th>
<th>73/74</th>
<th>74/75</th>
<th>75/76</th>
<th>76/77</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Carcine</td>
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<td>68</td>
<td>58</td>
<td>76</td>
<td>90</td>
<td>102</td>
<td>108</td>
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<td>Ovine</td>
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<td>110</td>
<td>100</td>
<td>90</td>
<td>80</td>
<td>70</td>
</tr>
<tr>
<td>Bovine</td>
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<td>200</td>
<td>200</td>
<td>200</td>
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<tr>
<td>Porcine</td>
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<td>130</td>
<td>130</td>
<td>130</td>
<td>130</td>
<td>130</td>
</tr>
</tbody>
</table>

Table 2: Periodic Diseases Observed on Meat Inspection in Nigeria, Fiscal Years 1971-79

*Note: The table shows the number of cases per 10,000 animals inspected.*
Table 3: Degenerative, Dyspneal, Septic and Inflammatory Conditions Observed on Meat Inspection in Nigeria, Fiscal Years 1975-1979

<table>
<thead>
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<td>67</td>
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<td>63</td>
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<tr>
<td></td>
<td>caprine</td>
<td>-</td>
<td>11</td>
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<td>Lung conditions:</td>
<td></td>
<td></td>
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<td>Liver conditions:</td>
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<td>caprine</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>6</td>
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<tr>
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<td>2</td>
<td>-</td>
<td>10</td>
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<td></td>
<td>ovine</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>8</td>
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<td>-</td>
<td>-</td>
<td>3</td>
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<tr>
<td>Carcass conditions:</td>
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<td>67</td>
<td>-</td>
<td>-</td>
<td>37</td>
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<tr>
<td>abscesses</td>
<td>ovine</td>
<td>60</td>
<td>23</td>
<td>-</td>
<td>-</td>
<td>42</td>
</tr>
<tr>
<td></td>
<td>caprine</td>
<td>35</td>
<td>13</td>
<td>-</td>
<td>-</td>
<td>24</td>
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</table>
Table 4: The number of food animals slaughtered throughout Nigeria, Fiscal Years 1971-79

<table>
<thead>
<tr>
<th>Years</th>
<th>Cattle</th>
<th>Sheep</th>
<th>Goat</th>
<th>Camel</th>
<th>Pig</th>
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</thead>
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<tr>
<td>1971-72</td>
<td>128,126</td>
<td>25,877</td>
<td>87,697</td>
<td>3,693</td>
<td>758</td>
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<tr>
<td>1972-73</td>
<td>215,280</td>
<td>46,248</td>
<td>194,569</td>
<td>8,371</td>
<td>1,363</td>
</tr>
<tr>
<td>1973-74</td>
<td>406,477</td>
<td>95,349</td>
<td>254,504</td>
<td>8,364</td>
<td>2,617</td>
</tr>
<tr>
<td>1974-75</td>
<td>369,554</td>
<td>138,510</td>
<td>282,855</td>
<td>11,354</td>
<td>6,038</td>
</tr>
<tr>
<td>1975-76</td>
<td>240,208</td>
<td>79,460</td>
<td>281,491</td>
<td>7,692</td>
<td>5,478</td>
</tr>
<tr>
<td>1976-77</td>
<td>229,602</td>
<td>73,159</td>
<td>315,980</td>
<td>9,746</td>
<td>7,576</td>
</tr>
<tr>
<td>1977-78</td>
<td>301,056</td>
<td>N.A.</td>
<td>N.A.</td>
<td>N.A.</td>
<td>N.A.</td>
</tr>
<tr>
<td>1978-79</td>
<td>312,980</td>
<td>9,105</td>
<td>36,265</td>
<td>192</td>
<td>1,726</td>
</tr>
<tr>
<td>Total</td>
<td>2,009,511</td>
<td>467,708</td>
<td>1,453,361</td>
<td>49,412</td>
<td>25,556</td>
</tr>
<tr>
<td>Mean</td>
<td>251,189</td>
<td>66,815</td>
<td>207,623</td>
<td>7,059</td>
<td>3,651</td>
</tr>
</tbody>
</table>

Many animals, particularly cattle, arrive at the slaughter house extremely lean or emaciated. These conditions would indicate that many Nigerian food animals are in the state of poor health. This observation is supported by the very low dressing percentage of indigenous cattle. The average dressing percentage of 273 White Fulani cattle with an average live weight of 324.5kg, was 47.0 percent (Abubakar, 1979) while the dressing percentage of White Fulani and Sokoto Gudali bulls fattened on feedlot rations and weighing from 250-350kgs at slaughter ranged from 47.6 to 53.4% (Umoh et al., 1980). These figures do not vary much from the dressing percentage recorded several years ago by Wehahn, et al., (1964) which was 45.2% for poorly conditioned White Fulani bulls and 54.4% for those in good condition.

The large number of bruises and carcass abscesses encountered denote that animals are roughly handled. Abattoir returns have some limitations as a basis for estimating animal disease prevalence. The results of abattoir findings apply only to those animals slaughtered and not necessarily those on farms or elsewhere. The data are limited to those conditions with gross pathological lesions and ignore other abnormalities. Many diseases do not show pathological symptoms or lesions observable at meat inspection. Finally, observations on meat inspection are subject to observer bias influenced by limited repertories of diagnoses, and the ability and integrity of the observers (Hugh-Jones, 1974). However, the advantages of abattoir surveillance data seem to outweigh the inherent disadvantages. Disease conditions encountered at meat inspection may be fairly representative of the general disease situation, particularly when meat inspection is conducted on a regular and country-wide basis and under efficient government supervision. Livestock consigned for slaughter generally include those animals which have high probability of having health problems. Accuracy of results is further enhanced when laboratory diagnosis supports postmortem findings as in the confirmation of tuberculosis in cattle granulomas. The sample size is large enough to make abattoir returns a useful source of data for assessing animal health in a given time and place (Hugh-Jones, 1974). Slaughter surveillance reduces the need for costly probability sample surveys for diseases of low prevalence. For instance when the reactor rate for bovine tuberculosis and the number of herds found infected for hog cholera gave poor estimates of the progress toward eradication, abattoir information was found to be very useful in assessing the trends of progress toward eradication.
Assessing Animal Health and Disease Trends through Meat Inspection in Nigeria

Fig. 1: Prevalence Rates of Diseases and Conditions per 1,000 Food Animals Slaughtered in one Abattoir in Nigeria, Fiscal Year 1978–1979.
Fig. 2: Prevalence Rates of Diseases per 10,000 Cattle Slaughtered in Nigeria, Fiscal Years 1971–1979.
Assessing Animal Health and Disease Trends through Meat Inspection in Nigeria

Fig. 3: Prevalence Rates of *Echinococcus granulosus* Hydatidosis per 10,000 Food Animals Slaughtered in Nigeria Fiscal Years 1971–1979
of the diseases (Beal, 1974). The diagnosis of tuberculosis in swine on the basis of lesions observed on meat inspection is, however, more than 93% efficient (Schilf 1974).

Conclusion

The data presented in this report cannot be taken as anything like an accurate reflection of the actual prevalence of animal diseases in Nigeria, the true figures being probably much higher. The problem of assessing the exact prevalence of animal diseases is largely a question of an accurate and adequate surveillance system.

At the present time when survey data from the various possible sources are inadequate, abattoir returns which are gathered routinely and extensively from all over Nigeria may still be the best available indicator of the general magnitude of food animal health and disease problems of socio-economic importance in Nigeria.

Acknowledgement

The authors would like to thank Professor G.W. Beran of the Department of Veterinary Public Health and Preventive Medicine, A.B.U., for helpful comments during the preparation of the paper and for reviewing the final manuscript; to Dr. K.S. Leow of the Department of Geography, A.B.U., for assistance in the statistical analysis of the data and drawing of the tables and figures; and to the Director, Federal Livestock Department for permission to write and publish this article.

References


Received for publication on 15th July, 1981.
A Serological Survey of Q Fever in Indigenous Domestic Birds of Nigeria

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Summary

The capillary agglutination test for antibodies to Coxiella burnetii in indigenous domestic birds in Nigeria was carried out. Birds examined were domestic fowls, ducks, guinea fowls and turkeys. Over 53 percent of the fowls had circulating antibodies. From the examinations of the small numbers of ducks, guinea fowls and turkeys, there was indication that antibodies were also prevalent in these birds in the proportion of 20.4 percent, 24.8 percent and 13.2 percent respectively. The role of these birds in the epidemiology of the disease in man is emphasized. Evidence of infection in the indigenous avian population adds new species in the list of animal reservoirs of C. burnetii in Nigeria.

Introduction

Q fever is an infectious diseases of man, first reported by Derrick (1937) who observed the disease among the abattoir workers in Brisbane Australia. Man is infected by contact with animals and/or their products or through inhalation of contaminated air. Infections in animals are generally asymptomatic. It is caused by an intracellular obligate rickettsia, Coxiella burnetii. Evidence for infection has been reported in cattle, sheep, goats, camels and man (Collard and Udoozo, 1959; Addo and Schnurrenberger, 1977; Addo and Schnurrenberger and Greenwood, 1977; Addo, 1980) but literature on the avian is rare. However, elsewhere Q fever is reportedly common in birds (Shesto-chenko 1960) and in fact, human infection by C. burnetii by eating raw eggs from infected hens have been reported (Zhmaeva and Pchelkina, 1957).

In view of the reports of Q fever in other indigenous domestic livestock and in man, it was desirable that a survey of indigenous domestic birds be carried out.

Materials and Methods

Sera were collected from local poultry markets in Sabon-Gari and Samaru both in Zaria area. Blood samples from fowl (Gallus domesticus), ducks (Cairina moschata) and guinea fowls (Numida meleagris) were collected as their necks were cut and allowed to bleed freely. The wing veins were used for collecting blood from the turkeys (Meleagris gallopavo). Blood was allowed to clot and serum was collected after centrifugation. It was observed that the proportion of serum obtained to the total amount of blood collected was small. Agitation of clots with applicator sticks caused hemolysis.

Refrigeration inhibited clotting but clotting was enhanced by placing samples in waterbath at 35°C. Only sera without hemolysis were considered suitable for testing. Serum samples were stored at about -20°C until they were tested. No storage lasted for more than a month.

All serum samples were tested by the capillary tube agglutination test (CAT) described by Luoto (1953). The CAT employs a stained suspension of C. burnetii grown in yolk sacs of embryonated eggs. Microhematocrit capillary tubes (Sherwood Medical Industries St. Louis, Missouri USA) were used for the tests. Approximately one third of the tube was filled with antigen and the remainder with serum by means of capillary action. The tubes were inverted and placed in a vertical position in a tube sealer and holder (Clay – Adams
Inc. New York N.Y., U.S.A.). Tests on undiluted sera were held at room temperature for four hours while tests for serially diluted sera were held for about 24h. before reading.

Positive sera showed blue-black agglomeration of *C. burnetii* whereas negative reaction did not show the agglomerates, rather, the antigen settled at the bottom of the tube. The antigen used was phase I antigen. A total of 1800 sera consisting of 1487 fowls, 103 ducks, 157 guinea fowls and 53 turkeys were tested. Ninety sera collected from 40 fowls, 20 ducks, 20 guinea fowls and 10 turkeys which were positive for antibodies using whole serum, were diluted serially and tested to determine their antibody titers.

**Results**

Sera from indigenous domestic birds were examined by the capillary agglutination test for Q fever rickettsial agglutinins. A total of 1800 sera consisting of 1487 fowls, 103 ducks, 157 guinea fowls and 53 turkeys were examined. The results of the tests are shown in Table 1. Seven hundred and ninety three sera (53.3 percent) collected from fowls, 21 (20.4 percent) from ducks, 39 (24.8 percent) from guinea fowls and seven (13.2 percent) from turkeys were positive.

The antibody titers of 90 sera (40 fowls, 20 ducks, 20 guinea fowls and 10 turkeys) randomly selected from among the positive sera are shown in Table 2. It was not possible to titrate all the positive sera because of the amount of antigen that would be involved. Seventeen positive sera (19 percent) among the 90 titrated had antibody titer 1:64 or above.

**Table 1: Results of Nigerian domestic birds’ sera tested for Q fever antibody**

<table>
<thead>
<tr>
<th>Biological name</th>
<th>Common name</th>
<th>Number tested</th>
<th>Number positive</th>
<th>Percent positive</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Gallus domesticus</em></td>
<td>Fowl</td>
<td>1487</td>
<td>793</td>
<td>53.3</td>
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<tr>
<td><em>Cairina moschata</em></td>
<td>Duck</td>
<td>103</td>
<td>21</td>
<td>20.4</td>
</tr>
<tr>
<td><em>Numida meleagris</em></td>
<td>Guinea Fowl</td>
<td>157</td>
<td>39</td>
<td>24.8</td>
</tr>
<tr>
<td><em>Meleagris gallopavo</em></td>
<td>Turkey</td>
<td>53</td>
<td>7</td>
<td>13.2</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td>1800</td>
<td>860</td>
<td>47.8</td>
</tr>
</tbody>
</table>

**Table 2. Antibody titer of ninety serially diluted sera**

<table>
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<tr>
<th>Biological name</th>
<th>Common name</th>
<th>Total</th>
<th>1:2</th>
<th>1:4</th>
<th>1:8</th>
<th>1:16</th>
<th>1:32</th>
<th>1:64</th>
<th>1:128</th>
<th>1:256</th>
<th>1:512</th>
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<tr>
<td><em>Gallus domesticus</em></td>
<td>Fowl</td>
<td>40</td>
<td>0</td>
<td>7</td>
<td>13</td>
<td>8</td>
<td>5</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><em>Cairina moschata</em></td>
<td>Duck</td>
<td>20</td>
<td>0</td>
<td>3</td>
<td>4</td>
<td>3</td>
<td>6</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td><em>Numida meleagris</em></td>
<td>Guinea fowl</td>
<td>20</td>
<td>1</td>
<td>4</td>
<td>3</td>
<td>5</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><em>Meleagris gallopavo</em></td>
<td>Turkey</td>
<td>10</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td>90</td>
<td>2</td>
<td>14</td>
<td>22</td>
<td>19</td>
<td>16</td>
<td>9</td>
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Discussion

A look at the literature on Q fever work carried out in the past in Nigeria shows that evidence of infections have been reported in most of the domestic animals (Addo and Schnurrenberger, 1977; Addo, 1980). In man infections have also been reported (Collard and Udeozo, 1959; Addo, Schnurrenberger and Greenwood, 1977). Information on birds is lacking. However, elsewhere Q fever is reportedly common in birds (Syrucek and Raska, 1956; Zhmaeva and Pchelkina, 1957; Shestochenko, 1960). This report shows a similar trend of evidence of infection in birds as exists in other parts of the world.

A serological prevalence of 13.4 percent in turkeys, 20.4 percent in ducks, 24.8 percent in guinea fowls and 53.3 percent in fowls appear high, especially in the fowls. All the sera tested were obtained from indigenous domestic birds raised on range system of husbandry. The husbandry practice will appear to account for the high number of positive sera. The free movements of the survey birds make them more likely to be exposed to C. burnetii infection. They are more likely to feed and roost near other livestock which may contaminate the air and soil of the area.

Human infection with C. burnetii is primarily by inhalation of contaminated air. Infected animals or their products and wastes act as source of such contamination. C. burnetii infection in man as a result of eating raw eggs has been reported (Zhmaeva and Pchelkina, 1957). In addition, infected poultry products and poultry wastes may contaminate the environment and so infect human.

Acknowledgements

Q fever antigen used was supplied freely from Rocky Mountain Laboratory, Hamilton, Montana, U.S.A. and the author is grateful. A large number of fowl sera tested were collected by Mr. J.O. Bale and Mr. Ojo of the National Animal Production and Research Institute, Ahmadu Bello University, Zaria, during examination for salmonellosis.

References


Received for publication on 28th July, 1981.
Table 1. Anthropoides velox, March 1961

<table>
<thead>
<tr>
<th>Biological stage</th>
<th>Count</th>
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<td>Stage 1</td>
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<tr>
<td>Stage 4</td>
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<td>Stage 5</td>
<td>5</td>
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<tr>
<td>Stage 6</td>
<td>6</td>
</tr>
<tr>
<td>Stage 7</td>
<td>7</td>
</tr>
</tbody>
</table>

Note: The table includes the count for each stage of the biological cycle of Anthropoides velox for March 1961.
Investigations of occurrence of \textit{Eimeria Necatix} (Johnson) in Zaria Northern Nigeria

G. BISHU, O.O. AKEREJOLA and A.I. ABDULKADIR

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Abstract

Studies were conducted on the existence of \textit{E. necatix} in Zaria, Nigeria, using criteria established to differentiate Eimeria species in chicken. Contents of 100 intestines were collected from a farm with an outbreak of coccidiosis.

Collected oocysts were purified and sporulated. Sporulated oocysts were fed to four-week-old coccidia-free chickens in graded quantities of 5,000, 50,000 and 500,000 per chick. Clinical signs in infected birds were found to be consistent with Eimeria infection. Intestines were collected from dead or sacrificed experimentally infected birds, and examined for gross and histopathological findings.

The small intestine, jejunum, and ileum were found to be distended, inflamed and had numerous pin point hemorrhages. The average dimension of oocysts was found to be 22.47 by 18.67 microns, which is within the range reported for \textit{E. necatix}.

The schizonts observed were deeply in the submucosal layer of small intestine and measured on the average 44.29 by 38.28 microns. The mature schizonts measured 58 microns in the largest diameter which is characteristic of \textit{E. necatix}.

These finding are confirmatory of \textit{E. necatix} although an additional involvement of ceca and the gross pathology observed is also indicative of the presence of \textit{E. tenella}.

Introduction

Coccidiosis is one of the commonest poultry diseases in Nigeria. The economic significance of the disease cannot be overemphasised. Eradication of the disease has been attempted but has been difficult to attain. However, it is important to know the types of species that afflict chicken in Nigeria in order to minimise losses.

Few surveys have been carried out to determine the prevalence in Nigeria. These were based mainly on clinical and postmortem findings (Hill \textit{et al} 1953; Ikeme 1961). Other unconfirmed outbreaks have also been reported (Anon 1954, 1956, 1960). However the exact information on the coccidial fauna in Nigerian poultry is lacking.

There are about 1,000 backyard poultry units in Zaria area, each with an average of 150 birds per unit. There are also about seven commercial units with above 5,000 birds per unit. Several clinical outbreak of coccidiosis have been recorded in many of these farms and have been confirmed by laboratory examinations. These diagnoses warrant a more detailed study of the \textit{Eimeria} species in poultry that occur in Nigeria.

This study reports the investigation of the types of Eimeria species present in Zaria area of Nigeria.

Materials and Methods

An outbreak of coccidiosis involving 3000 broilers was reported, 100 intestines were collected as soon as the birds were slaughtered. The contents of the intestines were evacuated into shallow sterile petridishes which contained 2.5% potassium dichromate and was allowed to sporulate. This formed the initial pool of oocysts. Fresh sporulated oocysts were made possible by reproducing oocysts in coccidia-free chickens.

Three houses were built and screened to achieve the recommendations of Tyzzer (1932). To avoid external coccidial contamination, day-old chicks on arrival were provided with sterilised equipment, cages, feeders and waterers. The feed used was commercial chick and growers mash without antibiotics or coccidiostats; each batch was autoclaved at 38-40°C with a relative humi-
dity of 40-70% to kill any contaminating oocysts (Warner 1933). Water was provided from hot water tap after cooling. Feed and water were supplied ad lib.

Chicken of the same age, sex and breed were used in all of the experiments. Specifications according to Ryley et al. (1976) was adhered to with regards to the age, inoculum, feeding status, collection medium, homogenisation, sieving, floatation, washing, sporulation and further purification of the oocysts.

Reproduction of oocysts was performed by placing metal trays containing 2.5% potassium dichromate under the cages for faecal collection. Faeces collected was broken with metal spatula and sieved using 8 inches diameter, 40 & 100-mesh sieves (aperture approximately 150 and 390 microns respectively). The sieved material was centrifuged to concentrate the oocyst at the sediment and the supernatant fluid discarded. Saturated salt solution was used to float oocysts which were removed from the surface, squirted into distilled water to wash free of salt. It was briefly centrifuged again in order to recover the oocysts and immediately put in 2.5% potassium dichromate solution for further sporulation at 29°C. Triplicate counts of oocysts per milliliter were made using haemocytometer and McMaster counters. The dichromate solution was washed off before storage at 4°C (Horton-Smith and Long 1965).

Four groups of ten coccidia-free 4 week-old warren breed cockrels were used. One group was used as control, while each bird in the remaining three groups received 5,000, 50,000 and 500,000 sporulated oocysts respectively. The oocysts were dropped into their crops directly using a pipette (Sharma 1964).

The intestine was removed from any chicken that died due to experimental infection. Two chicken were sacrificed at regular intervals of 24 hours for gross pathological and histological studies. Gross examination of the serosal and mucosal surfaces of the intestine was done. Mucosal scrappings of a portion of affected part was stained with Giemsa to check for developmental stages of coccidial organisms and the site of infection.

Sections of tissues, about 1mm thick and 2mm long, were cut, fixed in 10% formalin for seven days and processed in a Tissuematon*. Processed tissue were then embedded in paraffin, cut at 5-6μ, mounted and stained with Haematoxylin and Eosin (H & E). The sections were screened under low magnification (x100) and pathologic areas of the sections were examined under oil immersion. Locations of schizonts, gametocytes and oocysts were noted.

Degree of pathological changes were noted and measurements of the developmental stages were taken. The dimension (length x width) of oocysts was measured with Leitz micrometer.** The eye piece was calibrated against the micrometer slide (Reid 1968). The number of organisms observed were correlated with the degree and severity of infections.

The prepatent period was noted. Percentage sporulation and the shape index of oocysts were also determined.

Results

Table 1 shows the biometry of oocysts obtained in this study. Most of the oocysts were ovoidal in shape and had polar granules. The average oocyst dimensions was 22.47 by 18.67 microns.

Three percent sporulation was obtained eighteen hours and twenty eight percent recorded twenty hours after the passage of faeces. The minimum pre-patent period was 98 hours.

The group infected with 500,000 sporulated oocysts were on the third day seen to be sluggish, with ruffled feathers, loss of appetite, droopy with distinctly loose and blood-tinged faeces. By the seventh day two birds in the group were dead. Only loss of weight was observed in the group given 50,000 sporulated oocysts but they also had loose

*Tissue maton – Fischer Scientific Co., Chicago U.S.A.

**Leitz micrometer R. Leitz, Wetglar, West Germany.
and slightly blood tinged faeces. No significant clinical signs were observed in the group infected with 5,000 sporulated oocysts.

Table 1: Biometry of oocysts

<table>
<thead>
<tr>
<th>Length Range in Micron</th>
<th>% Oocyst</th>
<th>Width Range in Micron</th>
<th>% Oocyst</th>
</tr>
</thead>
<tbody>
<tr>
<td>25.00-27.30</td>
<td>9</td>
<td>21.00-23.21</td>
<td>12</td>
</tr>
<tr>
<td>23.00-24.99</td>
<td>32</td>
<td>20.00-20.99</td>
<td>5</td>
</tr>
<tr>
<td>21.00-22.99</td>
<td>37</td>
<td>19.00-19.99</td>
<td>42</td>
</tr>
<tr>
<td>19.00-20.99</td>
<td>20</td>
<td>17.00-18.99</td>
<td>23</td>
</tr>
<tr>
<td>17.75-18.99</td>
<td>2</td>
<td>16.00-17.99</td>
<td>18</td>
</tr>
</tbody>
</table>

Average Length: 22.47 microns
Length range: 17.75-27.30 microns
Average Width: 18.67 microns
Width range: 16.38-23.21 microns
Average shape index: 1.21
Shape Index range: 1.00-1.58

The small intestine particularly the jejunum and ileum was found to be distended, inflamed with numerous pin-point discrete haemorrhages. Histological examination of the jejunum and upper part of the ileum showed necrosis, sloughed epithelial cells, some inflammatory cells and damaged capillary vessels.

Table 2: Biometry of schizonts from jejunum

<table>
<thead>
<tr>
<th>Length range in micron</th>
<th>% schizonts</th>
<th>Width range in micron</th>
<th>% schizonts</th>
</tr>
</thead>
<tbody>
<tr>
<td>30.00-45.00</td>
<td>40</td>
<td>29.00-35.00</td>
<td>48</td>
</tr>
<tr>
<td>46.00-50.00</td>
<td>48</td>
<td>36.00-45.00</td>
<td>40</td>
</tr>
<tr>
<td>51.00-60.00</td>
<td>12</td>
<td>46.00-50.00</td>
<td>12</td>
</tr>
</tbody>
</table>

Average length 44.29 microns
Average width 38.28 microns.

The parasitised cells were enlarged. Parasitic granuloma were seen and focal type of reactions observed in lamina propria and adjacent muscular layer. Sloughed epithelium were being replaced by fibrin in certain areas. Typical second generation schizonts lying deep in the mucosa between the epithelial and muscular layers characteristic of *E. necatrix*

were seen. The average dimension of twenty five measured schizonts was 44.29 by 38.28 microns. Mature schizonts oval or spherical, packed with bundles of merozoites and above 58 microns in largest diameter were also observed, (Table 2).

![Fig. 1: A "colony" of matured schizont (second generation) of *E. necatrix* in sub epithelial tissue of the jejunum.](image)

Discussion

Findings in these studies show similarities to the survey reported by Gill in India (1961) and Long & Taneilian in Lebanon (1965). *Eimeria necatrix* was involved in the study in India while *E. mivati* was investigated in Lebanon. The extension of lesions to the caecum might also indicate the presence of *E. tenella*. The chickens that died during this study and those that were sacrificed at regular intervals showed specific pathological changes that indicated infection of *E. necatrix*.

The specific diagnosis of *E. necatrix* were based on gross pathological findings, and the part of intestine parasitised. The finding of blood-filled, massively
swollen intestine which was inflamed and beset with whitish pin-point opacities visible on serosal surface and containing second generation schizonts further indicates *E. necatrix*. The second feature confirming *E. necatrix* infection was the specific endogenous development. These being the large second generation schizonts which measured 58 micron at largest diameter, were found to have developed in the submucosa between the epithelial layer and muscular layer. Some of the mature schizonts were found to have ruptured the covering epithelial surface of small intestine freeing blood into the lumen, (Gill 1961; Reid 1968; Long *et al.* 1976).

The minimum prepatent period, minimum sporulation time and oocyst morphology found in this study are within the range reported for *E. necatrix* by (Gill 1961; Becker *et al.* 1956; and Reid 1968). This work undoubtedly establishes the presence of *E. necatrix* in Northern Nigeria, as the incidence has not been previously reported.

*Received for publication on 24th August, 1981.*

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The Pattern of Infectious Canine Distemper in Nigerian Dogs (Mongrel Breed)

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Summary
Record for the period of four years (January, 1976–December, 1979) from the Small Animal Clinic, Ahmadu Bello University, Zaria, Nigeria, were used to study the effect of season and age on, the clinical signs and pathological changes in canine distemper (C.D). The clinic recorded a total of 14712 canine cases and of these a total of 116 were C.D. The frequency of canine distemper was highest in March and lowest in August. The 6-0 month age group had the highest frequency (49.1%) of cases while the clinical signs indicating the involvement of the respiratory system (76.7%), integument (65.8%), gastrointestinal tract (56.8%) and the central nervous system (54.3%) were observed. Sixty two of the 116 cases died or were euthanised.

Pathological changes observed were variable, including pulmonary congestion and edema, areas of necrosis in the kidney especially in the cortex, hemorrhagic gastroenteritis, cystitis, hard foot pads, pustular dermatitis and conjunctivitis. Histopathological changes included widespread perivascular mononuclear infiltrations in the cerebellum and intranuclear and cytoplasmic inclusions in the gemastocytes and in epithelial cells of the urinary bladder. The clinical signs and pathological changes were similar to those which appear in the literature.

Introduction
Canine distemper (C.D) is a highly contagious viral disease of dogs enzootic in most parts of the world (Brian and Daria, 1975 and Siegmund, 1979). It is characterised by diphasic temperature, leukopenia, gastrointestinal and respiratory cattarrh frequently with pneumonia and neurological involvement (Brian and Daria, 1975 and Siegmund, 1979). The virus is classified under paramyxovirus, a large ribonucleic (RNA) virus, closely related to measles and rinderpest viruses (Siegmund, 1979).

Canine distemper was first recognised in Britain in the 18th century (Lauder et al., 1954). About twenty years ago comprehensive work was done on clinical and pathological features of C.D. viral infection at Glasgow University’s Veterinary School Clinic and the work showed almost no difference from earlier observations (Lauder et al., 1954).

In Nigeria, C.D. has been reported in four Nigerian dogs which showed nervous signs similar to those of rabies (Abdullahi, 1979).

The purpose of this report is to evaluate the effect of season and age on, and the common clinical signs and pathological changes of C.D. in indigenous dogs at the Small Animal Clinic, Ahmadu Bello University, Zaria, Nigeria and to compare them with those reported in the literature.

Materials and Methods
The information in this work was obtained from records for the period of four years (January, 1976–December, 1979) at the Small Animal Clinic, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, Nigeria.

Diagnosis of C.D. in this study was based on a combination of history, clinical signs and pathological changes. Blood samples were examined for total and differential white blood cell count and blood parasites. Swabs from mucopurulent ocular and nasal discharges were cultured for bacteria. Buffy coat smears were made, stained with Giemsa and examined for blood parasites and intranuclear and intracytoplasmic inclusion bodies. Post-mortem examinations
were performed on those sixty two (62) which died or were euthanised. Swabs from lesions in the lungs were cultured for bacteria. No virus isolation was attempted. Histopathological studies were done on Haematoxylin and Eosin (H & E) stained sections of the lung, intestine, urinary bladder, cerebellum, kidney, and stomach.

Age specific morbidity rates were calculated for the cases. Kolmogorov-Smirnov type statistics (Freedman, 1979) was used to test for seasonal (monthly) variation in the hospital frequency of C.D. (Appendix 1). Tables and graphs were used to reduce data where appropriate.

Appendix 1: The difference between the cumulative relative frequency of the observed number of cases and the cumulative relative frequency of the expected number of cases under the hypothesis of no seasonal variation is plotted against time.

<table>
<thead>
<tr>
<th>Mon.</th>
<th>Freq. (from T.1) (a)</th>
<th>Cumu. Freq. (b)</th>
<th>Fn</th>
<th>F</th>
<th>Fn-F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jan.</td>
<td>14</td>
<td>14</td>
<td>120.7 x 10^3</td>
<td>84.9 x 10^3</td>
<td>85.8 x 10^3</td>
</tr>
<tr>
<td>Feb.</td>
<td>5</td>
<td>19</td>
<td>163.8 x 10^3</td>
<td>162.2 x 10^3</td>
<td>1.6 x 10^3</td>
</tr>
<tr>
<td>Mar.</td>
<td>17</td>
<td>36</td>
<td>310.3 x 10^3</td>
<td>247.1 x 10^3</td>
<td>63.2 x 10^3</td>
</tr>
<tr>
<td>Apr.</td>
<td>12</td>
<td>48</td>
<td>413.8 x 10^3</td>
<td>329.2 x 10^3</td>
<td>84.6 x 10^3</td>
</tr>
<tr>
<td>May</td>
<td>7</td>
<td>55</td>
<td>474.1 x 10^3</td>
<td>414.1 x 10^3</td>
<td>60.0 x 10^3</td>
</tr>
<tr>
<td>Jun.</td>
<td>8</td>
<td>63</td>
<td>543.1 x 10^3</td>
<td>496.2 x 10^3</td>
<td>46.9 x 10^3</td>
</tr>
<tr>
<td>Jul.</td>
<td>8</td>
<td>71</td>
<td>612.1 x 10^3</td>
<td>518.1 x 10^3</td>
<td>31.0 x 10^3</td>
</tr>
<tr>
<td>Aug.</td>
<td>2</td>
<td>73</td>
<td>629.3 x 10^3</td>
<td>666.0 x 10^3</td>
<td>36.7 x 10^3</td>
</tr>
<tr>
<td>Sept.</td>
<td>4</td>
<td>77</td>
<td>663.8 x 10^3</td>
<td>748.1 x 10^3</td>
<td>84.3 x 10^3</td>
</tr>
<tr>
<td>Oct.</td>
<td>11</td>
<td>88</td>
<td>758.6 x 10^3</td>
<td>833.0 x 10^3</td>
<td>74.4 x 10^3</td>
</tr>
<tr>
<td>Nov.</td>
<td>15</td>
<td>103</td>
<td>887.8 x 10^3</td>
<td>915.1 x 10^3</td>
<td>27.2 x 10^3</td>
</tr>
<tr>
<td>Dec.</td>
<td>13</td>
<td>116</td>
<td>1.0000</td>
<td>1.0000</td>
<td>0.0000</td>
</tr>
</tbody>
</table>

\[
Fn = \frac{b}{116} \quad F = \frac{a}{365}\]
Table 1: Yearly and monthly variation of C.D. in mongrel breeds at small animal clinic, Ahmadu Bello University, Zaria, Nigeria.

<table>
<thead>
<tr>
<th>Month</th>
<th>1976</th>
<th>1977</th>
<th>1978</th>
<th>1979</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>1</td>
<td>1</td>
<td>5</td>
<td>7</td>
<td>14</td>
</tr>
<tr>
<td>February</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>March</td>
<td>1</td>
<td>8</td>
<td>5</td>
<td>3</td>
<td>17</td>
</tr>
<tr>
<td>April</td>
<td>0</td>
<td>4</td>
<td>3</td>
<td>5</td>
<td>12</td>
</tr>
<tr>
<td>May</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>June</td>
<td>0</td>
<td>6</td>
<td>1</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>July</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>8</td>
<td>11</td>
</tr>
<tr>
<td>August</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>September</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>October</td>
<td>1</td>
<td>0</td>
<td>4</td>
<td>6</td>
<td>11</td>
</tr>
<tr>
<td>November</td>
<td>0</td>
<td>1</td>
<td>7</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>December</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Total (a)</td>
<td>8</td>
<td>24</td>
<td>35</td>
<td>49</td>
<td>116</td>
</tr>
</tbody>
</table>

Total No. of Canine Cases seen in the clinic

Total (b) 2878 3966 4006 3862 14712

*Annual % of C.D. Cases (c) 0.28 0.61 0.87 1.3 0.79

*Annual % of C.D. Cases = $\frac{a}{b} \times 100\%$

\[ F_n = \text{Cumulative relative frequency of observed number of cases.} \]
\[ F = \text{Cumulative relative frequency of the expected number of cases.} \]

Fig. 1: Monthly variation in cases of canine distemper seen in Ahmadu Bello University Veterinary Clinic, Zaria (1976–1979).
Table 2: Age variation of the 116 cases of C.D. in mongrel breed at small animal clinic, Ahmadu Bello University, Zaria (1976-1980)

<table>
<thead>
<tr>
<th>Age Group in months</th>
<th>No. of C.D. Cases (a)</th>
<th>% of C.D. Cases (b)</th>
<th>Total No. of cases (c)</th>
<th>Age Specific % rate**</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–6</td>
<td>57</td>
<td>49.1</td>
<td>1112</td>
<td>5.13</td>
</tr>
<tr>
<td>7–13</td>
<td>23</td>
<td>19.3</td>
<td>1132</td>
<td>2.03</td>
</tr>
<tr>
<td>14–20</td>
<td>9</td>
<td>7.8</td>
<td>1632</td>
<td>0.55</td>
</tr>
<tr>
<td>21–27</td>
<td>6</td>
<td>5.2</td>
<td>2012</td>
<td>0.30</td>
</tr>
<tr>
<td>28–34</td>
<td>5</td>
<td>4.3</td>
<td>2816</td>
<td>0.16</td>
</tr>
<tr>
<td>35–41</td>
<td>4</td>
<td>3.4</td>
<td>2720</td>
<td>0.15</td>
</tr>
<tr>
<td>42–48</td>
<td>4</td>
<td>3.4</td>
<td>1519</td>
<td>0.26</td>
</tr>
<tr>
<td>49–72</td>
<td>4</td>
<td>3.4</td>
<td>1150</td>
<td>0.35</td>
</tr>
<tr>
<td>Unknown</td>
<td>4</td>
<td>3.4</td>
<td>619</td>
<td>0.65</td>
</tr>
</tbody>
</table>

* % of C.D. cases = \( \frac{a}{116} \times 100\% 

** Age specific % rate = \( \frac{a}{c} \times 100\% \)

The highest age specific morbidity rate, 5.13% cases was observed for the 0-6 month age group. This was followed by 7-13 month age group with age specific morbidity rate of 2.03% cases (Table 2). Cases within 0-6-month age group accounted for 49.1% of all the cases while cases within the 7-13 month age group accounted for 19.3% of all the cases of C.D. (Table 2). The lowest age specific morbidity rate 0.15% cases was observed for 35-41-month age which accounted for 3.4% of all the cases of C.D.

The clinical signs observed were those involving gastrointestinal tract (56.9%), respiratory system (76.74%), central nervous system (54.3%), integument (63.81%) and other (73.04%) (Table 3).

The common clinical signs observed in these systems were anorexia (31.03%), mucopurulent nasal discharge (34.5%), coughing (24.14%), dry rales (18%), depression (31.03%), dry and scaly muzzle (29.28%, Fig. 2), pyrexia (19.59%) and mucopurulent ocular discharge (30.17%) (Table 3). Others include emesis (13.80%), hypersalivation (4.31%), diarrhoea (7.76%), convulsion (7.76%), paraplegia (5.17%), chorea (10.34%), pustular dermatitis (12.93%, Fig. 3), hard foot pad (13.80%), dehydration (13.80%), congested mucous membrane (6.90%), tachycardia (6.90%), tonsillitis (5.17%), distemper breath (2.59%) and urinary incontinence (1.72%) (Table 3).

Leukocytosis followed by leukopenia was recorded for 110 (94.8%) cases. Ten cases (8.6%) had few eosinophils and polychromasia and hypoproteinaemia was observed in three cases (2.6%).

Different organisms were cultured from lung lesions of five fatal cases. These were Bordetella bronchiseptica from 2 fatal cases, Staphylococcus aureus from 2 fatal cases, and a combination of Nocardia spp. and Bordetella bronchiseptica from a fatal case.
### Table 3: Clinical signs of C.D. and their frequencies in mongrel breed at small animal clinic, Ahmadu Bello University, Zaria (Jan., '76-Dec., '79)

<table>
<thead>
<tr>
<th>Clinical Signs</th>
<th>Frequency of clinical Sign</th>
<th>% of frequency*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Gastrointestinal Problems:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anorexia</td>
<td>36</td>
<td>31.03</td>
</tr>
<tr>
<td>Emesis</td>
<td>16</td>
<td>13.80</td>
</tr>
<tr>
<td>Hypersalivation</td>
<td>5</td>
<td>4.31</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>9</td>
<td>7.76</td>
</tr>
<tr>
<td><strong>2. Respiratory Problems:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mucopurulent Nasal discharge</td>
<td>40</td>
<td>34.50</td>
</tr>
<tr>
<td>Coughing</td>
<td>28</td>
<td>24.14</td>
</tr>
<tr>
<td>Dry rales</td>
<td>21</td>
<td>18.10</td>
</tr>
<tr>
<td><strong>3. Central Nervous System Problems:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>36</td>
<td>31.03</td>
</tr>
<tr>
<td>Convulsion</td>
<td>9</td>
<td>7.76</td>
</tr>
<tr>
<td>Paraplegia</td>
<td>6</td>
<td>5.17</td>
</tr>
<tr>
<td>Chorea</td>
<td>12</td>
<td>10.34</td>
</tr>
<tr>
<td><strong>4. Intergument:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pustular Dermatitis</td>
<td>15</td>
<td>12.93</td>
</tr>
<tr>
<td>Hyperkeratosis of foot pads</td>
<td>16</td>
<td>13.80</td>
</tr>
<tr>
<td>Dehydration</td>
<td>16</td>
<td>18.80</td>
</tr>
<tr>
<td>Dry Scaly Muzzle</td>
<td>27</td>
<td>23.28</td>
</tr>
<tr>
<td><strong>5. Others:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mucopurulent ocular discharge</td>
<td>35</td>
<td>30.17</td>
</tr>
<tr>
<td>Tonsilitis</td>
<td>6</td>
<td>5.17</td>
</tr>
<tr>
<td>Distemper breath</td>
<td>3</td>
<td>2.59</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>8</td>
<td>6.90</td>
</tr>
<tr>
<td>Pyrexia</td>
<td>23</td>
<td>19.59</td>
</tr>
<tr>
<td>Congested mucous membrane</td>
<td>8</td>
<td>6.90</td>
</tr>
<tr>
<td>Urinary incontinence</td>
<td>2</td>
<td>1.72</td>
</tr>
</tbody>
</table>

*% of frequency = \( \frac{\text{frequency of clinical sign}}{\text{Total No. of Cases of C.D. (116)}} \times 100\% \)
Fig. 3: Pustular dermatitis in the inguinal area of a distemper case.

Fig. 4: Section of the cerebellum showing spongy medullary part of the folium. Note the large vacuoles (x 100).
Fig. 5: Section of lung showing interstitial pneumonia characterized by thickened interalveolar wall with infiltration of neutrophils and edema fluid. (x 200).

Fig. 6: Section of the urinary bladder showing areas of necrosis of the mucosal epithelial cells (x 200).
Gross lesions in the lungs were consolidation, edema and congestion especially of the anterior lobes. Purulent bronchopneumonia was observed when there was bacterial complication. The intestine and stomach showed, in some cases, enteritis and gastritis respectively. The liver was enlarged, congested and of nutmeg appearance. Splenomegaly was observed in few cases. The most significant change seen in the cerebellum was the spongy appearance of medullary part of the folium characterized by large irregular vacuoles (Fig. 4) and perivascular mononuclear infiltrations. Intranuclear inclusions were seen in some gemastocytes.

In the lung section was interstitial pneumonia characterized by thickened interalveolar wall which was infiltrated with neutrophils and macrophages (Fig. 5). Inclusion bodies were scanty in the lining epithelial cells of the bronchioles.

Extensive ulceration was seen in the epithelial layer of the urinary bladder. There was diffuse edema and neutrophil infiltration in the submucosa. Subepithelial hemorrhage was seen especially in the ulcerated areas. Some areas of degeneration and necrosis were seen in the mucosal epithelial cells and some epithelial cellular nuclei were highly enlarged and vacuolated while others had undergone pyknosis or karyolysis (Fig. 6).

The submucosa of the stomach was congested and dilated glands and some necrotic cellular debris were present.

In the kidneys there were areas of necrosis, especially in the cortical tubules.

Discussion

There is seasonal variation in C.D. in Nigerian dogs (as shown by the graph, Fig. 1). Dry seasons favours the spread of the disease as transmission is mostly by infective aerosols (Brian and Daria, 1975 and Wright et al., 1974). The dry season enhances the chance of the disease spread by direct contact. This together with the gregarious nature of dogs and their special behaviour of licking each other may account for the prevalence of the disease in most areas of the world (Brian and Daria, 1975).

In this study, young dogs between 0-13 months of age seem to suffer from the disease more than older dogs. This suggests that the physiological immune system is not mature and thus results in the increase in susceptibility of young dogs to C.D. infection (Steven and Adalbert, 1976a and Steven and Osburn, 1976b). Brown et al., (1972) reported that puppies acquire passive immunity through the maternal antibodies which wears out as they get older and that in endemic areas older dogs develop immunity from low grade infections. It was, therefore, suggested that in fear of maternal antibody interference by distemper vaccine, measles virus vaccine, could be administered to maintain puppies through the 8-9 weeks of age at which C.D. vaccine is given (Brown et al., 1972; Garber and Marrow, 1976 and Dubley et al., 1978).

The clinical signs observed in this study were similar to those reported by Lauder et al., (1954) who worked with 50 dogs most of which (82%) showed respiratory, gastrointestinal, ocular catarrhal and mucopurulent discharges and neurological signs. Diphasic febrile periods (Brian and Daria, 1975) were not recorded in this work. This could be due to lack of prompt action by dog owners bringing their dogs at the febrile periods. Urinary incontinence was seen in overiohysterectomised dogs. It is noted in this report that not all cases of C.D. manifested nervous signs. General nervous manifestations may be delayed for months or years or absent after the general episode of the disease (Wright et al., 1974). In some cases only the nervous signs were seen. This could be due to faster dissemination of the viral antigen in the nervous system than in other systems before death. Chorea (repetitive myoclonic twitching of the muscle groups of mastication) was the second most observed clinical signs of the CNS.

Widespread perivascular accumulation of mononuclear cells is a characteristic finding of encephalitis in C.D. (Lincoln
et al., 1971). The respiratory system was the most affected. This may suggest that the major route of infection was the upper respiratory tract. In general, the pattern of C.D. in Nigerian dogs (Mongrel breed) was not different from those found in other countries of the world.

References


Received for publication on 14th September 1981.
The report of preliminary observations on the occurrence of respiratory disease in the domestic fowl is presented. The disease is characterized by a variety of clinical signs, including fever, coughing, and respiratory distress. The examination of affected birds revealed the presence of characteristic lesions in the respiratory tract. The disease is thought to be transmitted through the respiratory route, and the identification of the etiological agent is under investigation. The implications of these observations for poultry health and production are discussed.
An Epizootic of Bovine Malignant Catarrhal Fever in Northern Tanzania in 1976

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Summary
An epizootic of African malignant catarrhal fever (MCF) occurred in Northern Tanzania in 1976 and killed several animals including 200 cattle on Manyara ranch alone. The disease was initially diagnosed in sick cattle on clinicopathological findings and was later confirmed by histological findings and virus isolation from wildebeest and sick cattle. Neutralizing antibodies to MCF virus were detected in all wildebeest tested but not in any cattle or sheep from the same ranch. An investigation conducted during this epizootic showed that MCF is an animal disease of economic importance in Tanzania because it kills 3 to 12% of the cattle in Masailand annually.

Introduction
Malignant catarrhal fever is a disease of cattle characterized by high fever, profuse, nasal and ocular discharge, hyperemia and necrosis of mucosae, corneal opacity and enlargement of lymph nodes. This disease kills all infected cattle. The disease observed in Africa is caused by a herpesvirus derived from the wildebeest (Mettam, 1923; Daubney and Hudson, 1936; Plowright et al. 1965). This disease usually affects one or a few animals in a herd because it is not contagious in cattle (Plowright, 1968). However, outbreaks can occur when many cattle are exposed to infected wildebeests (Mettam, 1923). Severe outbreaks of MCF have also been observed for the sheep-derived MCF (Pierson et al. 1973, 1974; James et al. 1975).

The purpose of this paper is to report an outbreak of the wildebeest-derived MCF that killed hundreds of cattle in Northern Tanzania in 1976. This outbreak was studied on Manyara ranch where at least 200 cattle died. We report also our discussions with Masai elders on the economic importance of this disease in Masailand in Tanzania.

Background of Manyara Ranch
Manyara ranch is found about 100 kilometers (km) South-West of Arusha town in Northern Tanzania. The ranch is approximately 17,600 hectares (44,000 acres) and carries about 7,000 Boran cattle and 3,000 local sheep. These animals are scattered on the ranch in groups of 500 to 1,000 each. They graze during the day and are kept in enclosures at night.

Several species of game animals are found on this ranch. These include wildebeest, gazelle (Thomson & Grants), zebra, buffalo, eland, warthog and giraffe. Jackals, hyenas and rodents are also present. Most wild animals

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on this ranch originate from Lake Manyara Park situated 10km. away. Domestic and wild animals graze and drink together freely except that wildebeest are chased away during their calving season.

The Outbreak

This outbreak started on Manyara ranch in early May when a few steers were found sick. Those diagnosed as cases of MCF were slaughtered. But when the number of sick animals increased beyond the expected few cases, the authorities became alarmed and sought assistance from the Veterinary Department in Arusha.

Subsequently, sick animals were examined and specimens taken from virus isolation, serological tests and histopathological examination (Table 1). Apart from the 14 sick animals examined, over 150 other cattle were recorded as having been diagnosed as MCF by the ranch’s personnel and then slaughtered between May and July, 1976.

### Materials and Methods

**Collection and Treatment of Specimens**

Samples of blood for virus isolation were collected from 8 wildebeest, 12 sheep and 14 sick cattle. A sample of 14 ml of blood was collected into a vial containing 5 ml of 1.5% ethylenediamine-tetraacetic acid (EDTA) suspended in 0.85% NaCl. These specimens were immediately placed in thermos flasks containing wet ice (4°C) and then sent by road to the laboratory in Kenya for testing 2 to 4 days later. Prescapular lymph nodes and spleens from 3 sheep, 8 wildebeest and 2 sick cattle were also collected for virus isolation. These tissues were placed in jars containing phosphate buffered saline (PBS) with antibiotics and transported to Muguga as described for blood samples.

For histopathological examination, sections of the brain, liver, kidney, adrenal, trachea, lymph node and abomasum were taken. They were fixed in 10% formalin in neutral buffered saline (Monobasic sodium phos-

### Table 1: Field and Laboratory Findings on MCF Cattle from Manyara Ranch

<table>
<thead>
<tr>
<th>Date of sampling</th>
<th>Animal No.</th>
<th>Tissue Examined</th>
<th>Virus Isolation</th>
<th>Form of Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>6/6/76</td>
<td>1</td>
<td>BC*</td>
<td>—</td>
<td>Severe with swollen tongue</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>BC</td>
<td>—</td>
<td>Severe</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>BC</td>
<td>+</td>
<td>Severe</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>Spleen</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>LN</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>BC</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Spleen</td>
<td>+</td>
<td>Severe</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LN</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>18/6/76</td>
<td>5</td>
<td>BC</td>
<td>—</td>
<td>Severe</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>BC</td>
<td>—</td>
<td>Severe</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>BC</td>
<td>+</td>
<td>Severe</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>BC</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>23/6/76</td>
<td>9</td>
<td>BC</td>
<td>+</td>
<td>Severe</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>BC</td>
<td>+</td>
<td>Severe</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>BC</td>
<td>+</td>
<td>Severe</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>BC</td>
<td>—</td>
<td>Severe</td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>BC</td>
<td>—</td>
<td>Severe</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>BC</td>
<td>—</td>
<td>Severe</td>
</tr>
</tbody>
</table>

*BC = Buffy coat  
LN = Lymph node  
+ = Virus isolated  
— = Virus not isolated
phate 4 g, dibasic sodium phosphate-anhydrous 6.5 g in 900 ml distilled water and 100 ml of formalin) embedded in paraffin wax and sectioned. They were stained with haematoxylin and eosin, mounted and examined. Blood for serum was collected from 8 wildebeest, 50 sheep and 14 sick cattle. Serum was obtained from clotted blood by centrifugation at 1000 x g for 20 minutes and was kept at -20°C until tested.

**Isolation of Virus from Specimens**

The cell cultures used throughout this study were either primary or secondary bovine thyroid (BTh) cells prepared as described by Plowright and Ferris (1961). The cells were seeded in the test tubes or Falcon flasks and grown in Earle's minimum essential medium (MEM) containing per ml 10% bovine serum, penicillin (200 units), dihydrostreptomycin (200 μg) kanamycin (80 μg) and mycostatin (60 units). The maintenance medium containing 2% bovine serum was changed every 2 days.

Buffy coats were prepared and inoculated into BTh cultures and then examined as described by Kalunda et al. (1980) or as summarized below. Buffy coats were obtained by centrifugation of blood at 500 x g for 30 to 45 minutes. White blood cells were then collected, washed once in PBS and centrifuged for 10 to 15 minutes. The resulting buffy coat cells were collected, suspended in 2 ml of the tissue culture maintenance medium and immediately inoculated into replicated monolayers of BTh cells. On the next day, the inoculated cultures were washed clear with PBS and then fed with fresh medium. Inoculated culture were examined every one to two days for 11 days.

Two to three blind cell culture passages were made if characteristic cytopathic effect (CPE) was not observed in the initial passage. In this case monolayers of infected cells were detached with 0.25% trypsin in PBS, expanded twice, grown and examined as before.

Cell suspensions of lymph nodes and spleens were prepared in ten Broeck grinders. They were diluted and inoculated for examination as described for buffy coats.

**Recovery of Virus from Inoculated Cattle**

Ten grade steers 12 to 18 months old were each inoculated intravenously with 10^2 to 10^4 tissue culture infective dose (TCID_{50}) of the virus derived from either bovine or wildebeest on Manyara ranch. Inoculated animals were examined daily for signs of disease. Rectal temperature was taken every morning and any rise to 39.3°C and above was an indication of disease. Sick animals were bled for virus recovery and in three cases two further animal passages were conducted with 10 ml of infective blood in EDTA. Tissue specimens from three sick cattle were collected and processed for histological examination.

**Virus Neutralization Test**

Sera from wildebeest, sheep and cattle were examined for neutralizing antibodies to strain WC11 of MCFV (Plowright et al. 1965) as described by Kalunda et al. 1980 or as summarized below: Serum samples were heat-inactivated at 56°C for 30 minutes and then diluted in two-fold series. Beginning with a 1:2 dilution, 1.0 ml of each serum dilution was mixed with an equal volume of the cell-free virus containing about 1000 TCID_{50} per ml of final dilution. After the serum-virus mixtures were held for one hour at 37°C, five culture tubes were each inoculated with 0.2 ml of every serum-virus mixture. Inoculated cultures were examined for 14 days. The 50% virus neutralization titre was calculated by the Karber's method (Lennette and Schmidt 1969).

**Results**

**Virus Isolation**

MCFV was isolated by cell culture techniques from 6 of 14 (42.8%) sick
cattle and from 2 of 8 (25%) wildebeest but not from any of the 12 sheep that were tested (Table 1). The virus produced syncytia in BTH cells in 4 to 12 days after inoculation. The identity of these isolates was later confirmed by neutralization test using reference antiserum and by their ability to reproduce the disease in experimental cattle.

All five isolates that were used reproduced the severe form of MCF in 14 to 25 days post inoculation. This disease was similar to that observed on Manyara ranch. This disease was reproduced in two further animal passages that were conducted.

**Histopathology**

Apart from fibrinoid necrotizing arteritis with thrombi observed in blood vessels, all tissues from sick cattle were infiltrated with mononuclear cells. There were histological lesions in the adrenals, brains, kidneys and livers similar to those described by Jubb & Kennedy (1970).

Gross pathological lesions of MCF were not observed in any of the wildebeest shot. But microlesions similar to those described above for cattle were seen.

**Neutralizing Test**

Neutralizing antibodies to MCFV were not detected in serum samples of any of the sick cattle or the 50 sheep examined. In contrast, all the tested wildebeest contained neutralizing antibodies (Table 2). Two of the wildebeest calves containing relatively low levels of antibodies yielded MCFV (Table 2).

**The Prevalence of MCFV in Masailand**

After investigating this severe outbreak of MCF on Manyara ranch, we examined Annual Reports of the Tanzanian Veterinary Department to find out the Economic important of this disease in Tanzania. Sporadic cases and a few minor outbreaks were reported.

The comprehensive account of MCF in Masailand was obtained from Mr. Letema, the Chairman of the Masai Ranch Association in Tanzania. He informed us that the three most economic cattle diseases in Masailand were MCF (Ugonjwa wa nyumbu i.e. the disease of the wildebeest), East Coast fever (Ndigana) and trypanosomiasis.

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**Table 2: Virus isolation and antibody detection in wildebeest from Manyara Ranch**

<table>
<thead>
<tr>
<th>Animal No.</th>
<th>Age*</th>
<th>Sex</th>
<th>Specimen</th>
<th>Results</th>
<th>MCF Antibody Titre</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4–6 m</td>
<td>F</td>
<td>BC &amp; LN</td>
<td>—</td>
<td>2.8**</td>
</tr>
<tr>
<td>2</td>
<td>4–6 m</td>
<td>F</td>
<td>BC &amp; LN</td>
<td>—</td>
<td>3.3</td>
</tr>
<tr>
<td>3</td>
<td>4–6 m</td>
<td>M</td>
<td>BC</td>
<td>Hemolysed</td>
<td>1.8</td>
</tr>
<tr>
<td>4</td>
<td>4–6 m</td>
<td>F</td>
<td>LN</td>
<td>+</td>
<td>1.4</td>
</tr>
<tr>
<td>5</td>
<td>2 y</td>
<td>F</td>
<td>BC &amp; LN</td>
<td>—</td>
<td>4.2</td>
</tr>
<tr>
<td>6</td>
<td>2 y</td>
<td>M</td>
<td>BC &amp; LN</td>
<td>—</td>
<td>3.8</td>
</tr>
<tr>
<td>7</td>
<td>2 y</td>
<td>M</td>
<td>BC &amp; LN</td>
<td>—</td>
<td>3.8</td>
</tr>
<tr>
<td>8</td>
<td>6 y</td>
<td>F</td>
<td>BC &amp; LN</td>
<td>—</td>
<td>2.8</td>
</tr>
</tbody>
</table>

*Age was determined from dentition aided by the fact that calves are born between December and February every year.

**Log neutralization index

BC = Buffy coat

LN = Lymph node

m = months

y = years
(Ndolobo) arranged in that order of importance. To emphasize the importance of MCF, the Chairman gave an example of cattle kept at Olokuro location where 50 to 100 and sometimes 200 (3.3% to 13.3%) of 1500 cattle die annually of this disease. Another cattle owner who lives near Lake Manyara Park told us that MCF killed 6 of his 150 cattle (4%) in 1975 and by early June 1976 five other cattle had already died from the same disease. Other cattle owners gave similar reports.

Mr. Letema told us that the Masai protect their cattle against MCF by moving them to highlands shortly before the beginning of the wildebeest calving season and returning them after the wildebeest have calved (i.e. December to March). This preventive measure is taken because the Masai believe that their cattle contract MCF by drinking foetal fluids and eating grass contaminated with this fluid or foetal membranes of calving wildebeest. This control method has not been effective in the last 5 years because severe droughts during this period forced wildebeest to follow cattle migration in pursuit of drinking water.

Mr. Letema concluded by informing us that the Masai do not normally report cases of MCF to the Veterinary Departments because preventive and curative treatments they would have sought in reporting such cases are not available for this disease. They therefore diagnose and slaughter all infected cattle which would otherwise die of MCF. Hence, the Veterinary Department is unlikely to determine the true incidence of MCF in Masailand from reported cases.

Discussion

We confirmed by histopathology and virus isolation that the disease observed on Manyara ranch was the African MCF caused by a herpes virus. The source of this virus was the wildebeest calves found on the same ranch. Other wildebeest from which we failed to isolate this virus had specific neutralizing antibodies indicating that they too were infected. The role of other game animals was not investigated. But sheep from the same ranch were not infected as evidenced by our failure to isolate virus and to detect neutralizing antibodies.

Our failure to isolate MCFV from 8 of 14 clinical cases was attributed to the long delays between specimen collection and inoculation of BTH cell cultures. These delays were inevitable because the specimens were transported by road from Manyara ranch to Muguga in Kenya, 300 miles away.

Our investigations on Manyara and other ranches established that this outbreak was precipitated by severe drought which prevented the traditional isolation of cattle during the calving season of wildebeest. Hence, many cattle were exposed to infected wildebeest resulting in this epizootic.

There is therefore a need to develop a vaccine against MCF as traditional preventive measures are not always effective.

We found during the investigations that the Masai still believe that cattle contract MCF from wildebeest foetal membranes and fluids. These beliefs which were first reported over 40 years ago (Daubney and Hudson, 1936) have not yet been proven. Attempts to isolate MCFV from wildebeest foetal membranes or to reproduce the disease by feeding cattle with these materials proved negative (Mettam, 1929; Plowright, 1964). There are also minimal chances that cattle are ever exposed to foetal materials because pregnant wildebeest usually segregate themselves and live in obscure places until their offspring are a few weeks old. Hence, wildebeest foetal materials are not the major source of cattle infection.

We therefore propose that wildebeest calves be considered as the main source of MCF because of the following reasons: (a) MCF is readily reproducible by keeping cattle in close contact with wildebeest calves (Mettam, 1929; Daubney and Hudson, 1936 and Plowright, 1965a, 1965b). (b) We
isolated MCFV from young but not from adult wildebeest. Similarly, Plowright (1965a) isolated MCFV more readily from wildebeest calves than adults.

It is worth noting that we isolated MCFV from wildebeest calves having low neutralizing antibodies. Even then, the virus was obtained from lymph node cell suspensions but not from buffy coats. These findings suggest that high MCF neutralizing antibodies suppress the circulation of the virus and therefore accounts for its non-recovery from animals with high antibody titres. Hence, wildebeest calves lacking colostrum might have high titres of viraemia and therefore good candidates for the dissemination of this disease.

Acknowledgements

We thank the Director of the East African Veterinary Research Organisation, Muguga and the Director of the Livestock Development Division, Tanzania for their permission to publish this work. We thank also Messrs J. Mumira and J. Mollel for their technical assistance. The assistance of Mr. F. Masawe, Farm Manager of Manyara ranch during this study is acknowledged.

References


Received for publication on 26th August, 1981
Mortality in Sheep on the University of Ibadan Teaching and Research Farm, Ibadan, Nigeria

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Summary

The post-mortem diagnoses on 319 sheep performed between January 1974 and December 1977 were reviewed. Most deaths (55.49%) occurred in sheep under one year old. The most common pathological conditions were pneumonia (23.2%), haemonchosis (16.30%), Kata (Peste des petits ruminants) (12.23%) and enterotoxaemia (11.29%). The presenting clinical signs and/or history were summarized.

Introduction

While beef continues to be the most important source of animal protein in the humid forest zones of West Africa, the people of the region rely on their neighbours towards the north of Africa for the supply of cattle. As a result of the recent drought which decimated the livestock population of the latter region and also because of the increased affluence and hence higher animal protein consumption in Nigeria, the Federal and State Governments embarked on vigorous programmes aimed at boosting the national livestock population (Akerejola, 1980).

Since trypanosomiasis severely limits the prospects of cattle rearing in the Southern parts of the country, greater attention is being devoted to the rearing of sheep and goats which, compared with cattle, are less attractive to tsetse flies and hence to clinical trypanosomiasis (Stephen, 1970). Nonetheless, there are other diseases which if not prevented or checked early in their course may lead to death in these stock. This paper reviews such conditions encountered on the University of Ibadan Teaching and Research Farm and gives a summary of the clinical history of the more important conditions.

Materials and Methods

The 319 animal necropsied between January 1974 and December 1977 were part of the sheep flock of the University of Ibadan Teaching and Research Farm. Some of them had received medical attention prior to death while those found “dead overnight” were sent directly from the farm to the Department of Pathology. The post-mortem examinations were carried out by the Veterinary Pathology Department while the clinical aspect was performed by members of the then Veterinary Medicine and Surgery Department. As a routine, parasitological and bacteriological isolation and identifications were attempted.

Results

The conditions incriminated in the deaths of the 319 sheep are presented on Table 1. The most commonly diagnosed condition, pneumonia, was associated with 74 or 23.2% of all deaths. Lesions of bronchopneumonia were seen in 47 cases. Pasteurella multocida was most frequently isolated although Corynebacterium pyogenes, Escherichia coli and Staphylococcus sp. were also isolated from the lung tissues. Clinically, the presenting signs were mucous to mucopurulent nasal discharges and cough. However, in some cases, dyspnoea (marked tachypnoea and extension of the neck) were observed. 19 of the 47 cases died overnight. The other 27 cases were diagnosed as aspiration pneumonia which was often associated
Table 1: Post-mortem findings on 319 sheep at the University of Ibadan, Nigeria 1974–1977.

<table>
<thead>
<tr>
<th>Conditions diagnosed</th>
<th>Age in months and number of sheep affected</th>
<th>Sex</th>
<th>Total</th>
<th>% of Grand Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0–3</td>
<td>4–6</td>
<td>7–11</td>
<td>12–23</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>19</td>
<td>15</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>Haemonchosis</td>
<td>10</td>
<td>13</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>“Kata”</td>
<td>9</td>
<td>8</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>Enterotoxaemia</td>
<td>2</td>
<td>14</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Starvation</td>
<td>2</td>
<td>9</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Tachniaisis</td>
<td>5</td>
<td>7</td>
<td>3</td>
<td>–</td>
</tr>
<tr>
<td>Coccidiosis</td>
<td>5</td>
<td>3</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Septicaemia</td>
<td>2</td>
<td>3</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Pregnancy toxaemia</td>
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<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Tetanus</td>
<td>3</td>
<td>4</td>
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<td>–</td>
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<tr>
<td>Mycotic rumenitis</td>
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<td>1</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Pyometra</td>
<td>–</td>
<td>–</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Colibacillosis</td>
<td>2</td>
<td>2</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Bloat</td>
<td>–</td>
<td>1</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Heartwater</td>
<td>–</td>
<td>–</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Scabies</td>
<td>–</td>
<td>–</td>
<td>2</td>
<td>–</td>
</tr>
<tr>
<td>Trichuriasis</td>
<td>–</td>
<td>–</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Blue tongue*</td>
<td>–</td>
<td>–</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Foot rot</td>
<td>1</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Traumatic injury</td>
<td>–</td>
<td>–</td>
<td>1</td>
<td>–</td>
</tr>
<tr>
<td>Karatoconjunctivitis</td>
<td>–</td>
<td>2</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Meningitis</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Trichobezoar</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>1</td>
</tr>
<tr>
<td>Rabies</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>60</td>
<td>82</td>
<td>35</td>
<td>57</td>
</tr>
<tr>
<td>% of Grand Total</td>
<td>18.81</td>
<td>25.71</td>
<td>10.97</td>
<td>17.87</td>
</tr>
</tbody>
</table>

*No virus isolation done.
Fig. 1: Monthly distribution of the three most commonly diagnosed conditions in sheep at the University of Ibadan Farm, Ibadan 1974–1977.
with history of anthelmintic drenching or dipping with Lindane (benzene hexachloride).

52 deaths or 16.31% were associated with haemonchosis. The most common presenting clinical signs were diarrhoea and anaemia. Less commonly, veterinary aid was sought because animals were weak or anorexic. In 16 cases, the sheep were reported as having died overnight.

Kata was incriminated in 39 cases or 12.23%. The presenting clinical signs were diarrhoea and pneumonia (cough and nasal discharges). Mouth lesions were observed less frequently.

Other conditions diagnosed include enterotoxaemia (11.29%) starvation (5.96%) taeniasis (5.64%) and intestinal coccidiosis (4.08%).

As shown by the monthly distribution of the three most common causes of mortality (Figure 1) most deaths occurred during the rainy season months.

Discussion

It should be emphasised that the mortality figures presented on Table 1 do not necessarily reflect the relative economic losses due to the conditions listed. Losses due to ectoparasitism mainly flea and lice infestations, which were prevented by regular dipping during the period under consideration, became significant in later years (Obasaju and Otesile, 1980). The farm experienced several outbreaks of contagious pustular dermatitis (Obi and Gibbs, 1978: Iwuoha, 1978) which, although on its own did not cause deaths, necessitated broad-spectrum antibiotic cover to combat secondary bacterial infections. Also, lameness due to foot-rot is an important disease condition but seldom resulted in death on this farm probably because it is quickly diagnosed and readily responds to antibiotic therapy. Although pneumonia caused more deaths than haemonchosis, the latter received more attention in terms of bi-monthly prophylaxis with thiabendazole or pyrantel tartrate.

The common peracute nature of pneumonia under this geographical environment (Oppong, 1973) might have been responsible for the high number of overnight deaths recorded.

The high mortality figures recorded for haemonchosis on this and other (Oppong, 1973) in spite of routine farms prophylaxis might be due to inadequate frequency of dosing. Sheep are drenched bi-monthly on this farm. However, a manufacturer of Thiabendazole, (Merck Sharp and Dohme, London) recommends that where pastures are heavily contaminated with worms, sheep should be dosed as often as once every three weeks (or even more frequently if signs of infestation are seen). Undoubtedly, in parts of the humid tropics such as Southern Nigeria where the acquisition of large areas of land for farming is difficult and the environment favours rapid build up of the parasite (Robertson, 1976), the cost of an efficient prophylaxis regime will be prohibitive.

37 of 41 deaths associated with enterotoxaemia occurred during 1974 and 1975. The sharp decline in the succeeding two years is attributed to prophylaxis with a multivalent clostridial vaccine (Covexin-8, Burroughs-Wellcome, England).

The cases of malnutrition were diagnosed mainly during the dry season in newly-weaned (4-6 months old) kids. At post-mortem, the carcases were dehydrated with scanty ruminal content. Usually such deaths were accompanied by a history of lack of supply of concentrate rations from the farm's feedmill. Taeniasis (Monieza expansa infection) commonly caused diarrhoea and the whitish gravid segments were often voided out with faeces. At post mortem, a large number of worms was often seen blocking the intestines. All deaths due to tetanus were sequel to castration. The only case of rabies resulted from the bite of a 3-year old ewe by a stray rabid dog.

Acknowledgement

The authors are indebted to Professors D.H. Hill and T.T. Isoun for permission to publish the clinical and post mortem information respectively.
Mortality in Sheep on the University of Ibadan Teaching and Research Farm, Ibadan, Nigeria 239

References


Received for publication on 20th October 1981.

Introduction

The importance of copper, zinc, magnesium and calcium in animal and human nutrition has long since been established (Underwood, 1971). These essential elements play a very vital role in many biochemical reactions in the animal body. It is known that deficiencies of copper in pigs may result in anemia, depressed growth, bone disorders, disorientation of hair and improper reproduction. Zinc deficiency in pigs results in poor feed efficiency, reduced growth, inactivity and parakeratosis while magnesium deficiency results in impaired growth, skeletal abnormalities, depressed reproductive function and ataxia of the newborn. Primary deficiencies of these and many other essential elements have been described in many parts of the world (Pumphrey et al., 1986; Lewis et al., 1956, Rockstra et al., 1956, Lucke et al., 1957, Underwood, 1971; Milah and McCracken, 1977).

Symptoms of essential mineral deficiencies are encountered sporadically in pigs in Nigeria but laboratory documentation has been lacking. Although there are some reports on serum levels of essential minerals in some Nigerian livestock (Oduye and Adefaraw, 1976, Oyang and Sarms, 1976), there is a paucity of information on serum levels of these elements in pigs. With the increased demand for meat production in Nigeria and the sprouting up of many commercial pig production ventures under semi-intensive management systems, it seems appropriate to determine serum levels of essential mineral deficiencies in different areas. This paper reports on serum mineral levels in growing Large White pigs raised under semi-intensive management systems in Nigeria.

Materials and Methods

Pigs used in this study were clinically normal growing Large White pigs raised on an institutional farm – The Animal Bello University Farm – and ranged in age from 2 days to 12 weeks. The pigs were raised semi-intensively in pens with concrete floors equipped with concrete watering and feeding troughs. The floors were kept and watered on daily. Pigs were allowed ad libitum with the pens until weaned at 7 to 8 weeks of age. During this period they were provided with wooden boxes in present coverage to the necessary wind. They were fed commercial feed recipes in meal form. Male pigs were castrated at 6 to 8 weeks of age. No disease conditions were encountered in the pigs during the period of investigation.
Serum Levels of some essential elements in growing Large White Pigs in Nigeria

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Faculty of Agriculture, Ahmadu Bello University, Zaria, Nigeria.

Summary

Serum levels of copper, zinc, magnesium and calcium were determined in growing Large White pigs from birth to 30 weeks of age under semi-intensive management. It was observed that variations occurred in these minerals as the pigs advanced in age but became stabilized by the time the pigs were 20 to 30 weeks old. It was also observed that no sex differences existed in serum levels of these essential elements. It is suggested that the values reported herein could serve as baseline data for pigs in this area.

Introduction

The importance of copper, zinc, magnesium and calcium in animal and human nutrition has long since been established (Underwood, 1971). These essential elements play very vital roles in many biochemical reactions in the animal body. It is known that deficiencies of copper in pigs may result in anaemia, depressed growth, bone disorders, depigmentation of hair and impaired reproduction. Zinc deficiency in pigs results in poor feed efficiency, subnormal growth, inappetence and parakeratosis while magnesium deficiency results in impaired growth, skeletal abnormalities, depressed reproductive function and ataxia of the newborn. Primary deficiencies of these and many other essential elements have been described in many parts of the world (Plumlee et al., 1956; Lewis et al., 1956, Hoekstra et al., 1956, Luecke et al., 1957, Underwood, 1971; Imlah and McTaggart, 1977).

Symptoms of essential mineral deficiencies are encountered sporadically in pigs in Nigeria but laboratory documentation has been lacking. Although there are some reports on serum levels of essential minerals in some Nigerian livestock (Oduye and Adedavoh, 1976, Gyang and Saror, 1979), there is a paucity of information on serum levels of these elements in pigs. With the increased demand for meat production in Nigeria and the springing up of many commercial pig production ventures under semi-intensive management systems, it seems appropriate to determine serum levels of essential elements in clinically normal pigs. Such basic information would be valuable in recognizing mineral deficiencies in affected areas. Thus, this paper reports on serum mineral levels of growing Large White pigs raised under semi-intensive management system in Nigeria.

Materials and Methods

Pigs used in this study were clinically normal growing Large White pigs raised in an Institutional farm – The Ahmadu Bello University Farm – and ranged in age from 2 days to 30 weeks. The pigs were raised semi-intensively in pens with concrete floor equipped with concrete watering and feeding troughs. The floors were swept and cleaned out daily. Piglets were allowed to remain with the sows until weaned at 7 to 8 weeks of age. During this period they were provided with wooden boxes to prevent exposure to the harmattan cold. They were fed commercial feed rations in meal form. Male piglets were castrated at 6 to 8 weeks of age. No disease conditions were encountered in the pigs during the period of investigation.

*Faculty of Veterinary Medicine
Blood samples for haematological evaluation were obtained via the interior vena cava using 2 inch 18 or 19 gauge needles and placed into vacutainer tubes containing EDTA as anticoagulant. Blood samples for serum extraction were obtained in plain mineral-free vacutainer tubes and allowed to clot. Following clot formation, the tubes were centrifuged and serum decanted into plastic mineral-free tubes and frozen until analysed. Hemolysed samples were discarded. Serum levels of copper, zinc, magnesium and calcium were analysed using the Atomic Absorption Spectrophotometer*. The values obtained were recorded according to age and sex and statistically analysed by computer using analysis of Variance.

Results

The means and standard errors of copper, zinc, magnesium and calcium in growing Large White pigs are presented in Table 1. Serum copper levels of pigs from birth to 4 weeks of age were significantly higher (P<0.001) than in older pigs. The lowest copper values were recorded in 5 to 8 week old pigs. Thereafter, there was a significant increase (P<0.05) in copper values as the pigs advanced in age.

Serum zinc levels were significantly lower (P<0.05) in young pigs up to 4 weeks of age than in older pigs. Serum magnesium levels were significantly higher (P<0.001) in zero to 4 week old pigs but gradually decreased as the pigs advanced in age. Young pigs up to 4 weeks of age had significantly higher calcium (P<0.001) than older pigs. The values declined at weaning age and remained stable thereafter until the pigs were 20 to 30 weeks of age when a moderate but significant increase was observed (P<0.05).

The values for copper, zinc, calcium and magnesium in growing Large White pigs according to sex are presented in Table 2. No significant sex differences were observed (P<0.05).

<table>
<thead>
<tr>
<th>Age</th>
<th>Cu Umol/L</th>
<th>Zn Umol/L</th>
<th>Mg, mmol/L</th>
<th>Ca mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–4 weeks</td>
<td>38.68***</td>
<td>17.68*</td>
<td>1.18***</td>
<td>2.97***</td>
</tr>
<tr>
<td></td>
<td>±1.49</td>
<td>±1.65</td>
<td>±0.03</td>
<td>±0.04</td>
</tr>
<tr>
<td>5–8 weeks</td>
<td>27.97*</td>
<td>19.83</td>
<td>1.04</td>
<td>2.67*</td>
</tr>
<tr>
<td></td>
<td>±1.01</td>
<td>±0.96</td>
<td>±0.04</td>
<td>±0.04</td>
</tr>
<tr>
<td>10–13 weeks</td>
<td>29.36*</td>
<td>20.24*</td>
<td>0.96</td>
<td>2.55*</td>
</tr>
<tr>
<td></td>
<td>±1.92</td>
<td>±0.97</td>
<td>±0.02</td>
<td>±0.04</td>
</tr>
<tr>
<td>20–20 weeks</td>
<td>32.15*</td>
<td>16.79</td>
<td>0.95</td>
<td>2.87</td>
</tr>
<tr>
<td></td>
<td>±1.11</td>
<td>±0.61</td>
<td>±0.02</td>
<td>±0.03</td>
</tr>
</tbody>
</table>

* = P<0.05  *** = P<0.001

Table 2: Means and standard errors of selected serum elements of male and female Large White pigs.

<table>
<thead>
<tr>
<th>Sex</th>
<th>No</th>
<th>Cu Umol/L</th>
<th>Zn Umol/L</th>
<th>Mg mmol/L</th>
<th>Ca mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>36</td>
<td>32.82</td>
<td>18.72</td>
<td>1.05</td>
<td>2.76</td>
</tr>
<tr>
<td></td>
<td></td>
<td>±1.32</td>
<td>±0.84</td>
<td>±0.03</td>
<td>±0.04</td>
</tr>
<tr>
<td>Female</td>
<td>36</td>
<td>31.13</td>
<td>18.53</td>
<td>0.99</td>
<td>2.77</td>
</tr>
<tr>
<td></td>
<td></td>
<td>±1.02</td>
<td>±0.70</td>
<td>±0.02</td>
<td>±0.03</td>
</tr>
<tr>
<td>Total</td>
<td>72</td>
<td>31.97</td>
<td>18.53</td>
<td>1.02</td>
<td>2.76</td>
</tr>
</tbody>
</table>

*Pyec Unicam Ltd. Model SP192.
Discussion

The serum values for copper, magnesium and calcium in this study compare favourably with values reported in other parts of the world (Underwood, 1971; Imlah and McTaggart, 1977). Hoekstra et al., (1956) reported zinc serum levels of 8.8 umol/L in 16 week old pigs as opposed to 16.8 umol/L in 20 to 30 week old pigs in this study. The reasons for this wide difference is not apparent. We do not believe that contamination played a role. It is proposed therefore that the values for copper, zinc, magnesium and calcium reported herein may serve as guidelines in subsequent investigations of these mineral deficiencies in Large White pigs in Nigeria. It is evident in this study that sex differences do not influence mineral status of growing Large White pigs. Similar observations have been made (Underwood, 1971). The results of our study indicate that wide variations may occur in levels of essential elements in the sera of growing pigs.

Acknowledgements

We are indebted to Dr. O.A. Osinowo of the National Animal Production Research Institute for the computer analysis of data, and to Ahmadu Bello University Board of Research for the financial support for this investigation.

References


Received for publication on 17th July, 1981.
Antimicrobial activity of some Edible Plants

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Departments of Pharmacology and Veterinary Medicine, Faculty of Veterinary Medicine,
Cairo University, Egypt

Summary

The present work revealed that some of the investigated edible plants possess a powerful antimicrobial activity against many pathogenic micro-organisms. Allium sativum alcoholic extract in all concentrations used inhibited the growth of Brucella abortus, E. Coli, Salmonella typhimurium organisms, while Corynebacterium pseudotuberculosis was only inhibited with 20% extract.

Banana cavenideshi fruit extracts of peel and pulp has marked antibiotic effect against E. coli and Salmonella typhimurium but not against Brucella abortus or Corynebacterium pseudotuberculosis. It is proved definitely that the alcoholic extract of Hibiscus subdariffa flowers has a powerful antibacterial effect against Brucella abortus, E. coli, Salmonella typhimurium but not against Corynebacterium pseudotuberculosis which was only suppressed by 20% extract.

Portulaca oleracea proved to be of no antibacterial activity against the studied micro-organisms.

Introduction

Wide therapeutic applications of medicinal plants encouraged many workers to find out scientific basis which explain their uses. Therefore numerous studies were carried out for revealing their botanical, chemical, nutritive and pharmacological characteristics.

Investigation of antimicrobial activity in plants particularly those largely consumed by humans is highly indicated because it may be considered in the future an effective, available and inexpensive source of antimicrobial agent. Accordingly, this study is going to investigate antibacterial activity in Allium sativum, Banana Cavendeshi fruit (peel or pulp), Hibiscus subdariffa flowers and Portulaca oleracea as these plants are extensively cultivated and largely consumed in Egypt.

No available literature could be obtained on the antibacterial activity of these edible plants except those reported by Collier (1954) who concluded that saline extract of Hibiscus possesses an antibiotic action against E. Coli. Moreover Sharaf and Shalash (1960) demonstrated the antibacterial effects of Hibiscus on staphylococcus aureus microorganisms using the gutter techni-
The optimum growth of *Brucella abortus* was obtained at 37°C, pH 6.6 and atmospheric condition containing 5-10% CO₂, while that of *E. coli*, *Corynebacterium pseudotuberculosis* and *Salmonella typhimurium* was obtained at 37°C and pH 7.2.

Studying the antimicrobial effect of these extracts was continued for 3 successive days during which results were recorded at 24 hour intervals and then confirmed.

**Results**

The obtained results were recorded in Table 1 and revealed that *Allium sativum*, *Banana cavendeshii* fruit (peel or pulp) and *Hibiscus subdariffa* alcoholic extracts inhibited the growth of *E. coli* when they were tested in 5, 10 and 20 percent concentrations for 3 successive days. *Portulaca oleracea* alcoholic extract seemed to be of no effect on this organism.

Viability of *Salmonella typhimurium* in this study was inhibited by 5, 10 and 20 percent alcoholic extracts of *Allium sativum*, *Banana cavendeshii* fruit and *Hibiscus subdariffa* flowers, but that of *Portulaca oleracea* showed no effect on it.

*Corynebacterium pseudotuberculosis* was only suppressed by 20% alcoholic extracts of *Hibiscus subdariffa* flowers and *Allium sativum*. These extracts in 5 and 10% concentrations as well as *Banana cavendeshii* fruit (peel or pulp) and *Portulaca oleracea* extracts has no effect on *corynebacterium pseudotuberculosis*.

*Hibiscus subdariffa* flowers and *Allium sativum* fruit alcoholic extracts in all concentrations inhibited growth of *Brucella abortus* micro-organism for 3 successive days while that of *Banana Cavendeshii* fruit and *Portulaca oleracea* has no effect. The diameter of inhibitory zones increased with the increase in concentration of extract and period of incubation.
<table>
<thead>
<tr>
<th>Treatment</th>
<th>20</th>
<th>10</th>
<th>5</th>
<th>5</th>
<th>10</th>
<th>20</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>A: Blank</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>B: Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C: Test</td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

**Antimicrobial activity of some Edible Plants**

1. **E. coli**: The antimicrobial activity in edible plants was determined using the disk diffusion method.
Discussion

Investigation of antibacterial activity in edible plants seems to be highly significant as it may provide in future a valuable inexpensive and acceptable source of antimicrobial drugs.

Expensiveness and numerous side effects of sulphonamide and antibiotic therapy justifies the search for new antimicrobial drugs which will be cheaper, highly effective, of little side effect and available. These advantages may be found in those plants that were previously used in treatment of many diseases.

This study revealed that *Allium sativum* alcoholic extract in concentrations of 5, 10 and 20 percent markedly inhibited growth of *Brucella abortus*, *E. coli*, *Salmonella typhimurium* organisms. The inhibition observed may be attributed to its high volatile oil content.

The antibacterial effect of *Allium sativum* extract on *E. coli* and *Salmonella typhimurium* micro-organisms in this study explains and confirmed the fact indicated its row consumption in cases of dysentery in man. It is clear that *Corynebacterium pseudotuberculosis* was the highly resistant micro-organism to the extract as it was only inhibited by 20 percent of it.

*Banana cavendshii* fruit alcoholic extract either prepared from the peel or pulp prevented the growth of *E. coli* and *Salmonella typhimurium* but not that of *Brucella abortus* or *Corynebacterium pseudotuberculosis*.

The antibiotic property of banana extract may be attributed to its high content of tannins, (Ola, 1975) or iron (Patel and Shah 1955) or to its enzyme system content, (Deacon and Marsh, 1971). The antibacterial activity in banana fruit extracts is considered highly significant as it is a popular fruit in Egypt due to its nice palatable taste, high nutritive value and its availability all the year round (F.A.O. 1957). Moreover, intravenous injection of banana fruit extracts in doses up to 10 mg/Kg b.wt. had no effect on arterial blood pressure and respiration in anaesthetised dogs (Ola, 1975). The same author concluded that feeding of rats on diets containing 50% *Banana cavendshii* pulp or peel powder caused a significant increase in erythrocytic count but had no effect on haemoglobin percent.

This work noted that *Hibiscus subdariffa* alcoholic extract has a powerful antibiotic action as it prevents growth of *Burcella abortus*, *Corynebacterium pseudotuberculosis*, *E. coli* and *Salmonella typhimurium* in all examined concentrations. This may be and *Salmonella typhimurium* in all examined concentrations. This may be due to the colouring substances present in the extract e.g. anthocyanins as proved by Sharaf and Shalash (1960) and Sharaf and Sharaf and Gineidi (1961).

Presence of antibacterial activity in *Hibiscus subdariffa* flowers is considered to be of benefit, as it is commonly used as a cold or warm popular drink in Egypt. Accordingly it is advisable to be given to patients suffering from diseases caused by such pathogenic micro-organisms rather than its high nutritive value. Moreover, LD50 of *Hibiscus subdariffa* alcoholic extract after intraperitoneal injection in mice was 625 mg/Kg b.wt. (Mayah, 1978). He concluded also that intraperitoneal injection of the extracts in mice in a dose of 62.5 mg/Kg b.wt. for 6 weeks produced significant changes in blood criteria.

Concerning *Portulaca oleracea* (Regla) alcoholic extract, it was proved to be of no antibacterial effect against the studied organism and this may be due to the absence of any of the antimicrobial constituents commonly detected in plants having this property as mentioned by Sharaf and Shalash (1960) who attributed these to the presence of tannins, sulphur, volatile oils, anthocyanins etc.

Conclusively edible plants seems to be valuable and available inexpensive source of antimicrobial agents which needs further more investigations.
References


Received for publication 7th July, 1981
highly resistant to the extract as it was used in 10 thousand mg.

The antibacterial property was

not by attributed to contain of quercetin. Oda, 1970,

(Dacca and Shuk, 1952) or its in vivo and in vitro extracts. 1971). Antibacterial activity aspects of extracts are considered highly significant as it is a popular fruit in Egypt due to its high nutritional value and its availability the year-round (E.A.O., 1957). Montreal indicated injection of banana fruit extracts in doses up to 10 mg/kg b.w. to prevent its spread.
Studies of the Toxicity of Gnidia Latifolia (Meisn) in Cattle

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Faculty of Veterinary Medicine, University of Nairobi, P.O. Box 29053, Nairobi

Summary

An investigation was carried out on the toxicity of Gnidia latifolia in bull calves. Twelve calves were drenched with ground leaves and their responses studied. Signs of clinical intoxication were observed in all the treated animals and the intensity of the signs depended on the amount of material fed. Animals receiving high doses (1-2 gm/kg body weight) showed acute toxicity with signs of depression, inappetence, nasal and ocular discharges. Some animals developed diarrhoea. Acute abdominal pain, uneasiness, elevated pulse and respiratory rates were observed. Animals with chronic signs of poisoning had a marked loss of body condition, loss of hair, diarrhoea and submandibular oedema. Postmortem changes in the acutely intoxicated calves included petechial haemorrhages of the epicardium and submucosa of the rumen, abomasum and the intestines. The chronic cases showed a generalized emaciation with gelatinous atrophy of the body fat and accumulation of excess fluid in the body cavities. Haematological examinations revealed a significant drop in the white cell counts due to lymphocytic cell depletion.

Introduction

Many plants in the genus Gnidia or Lasiosiphon in the family Thymelaeaceae have been proved poisonous for livestock (Alexander, 1928; Terblanche, Pieterse, Adelaar and Smit, 1966; Nwude and Parsons 1978a). Several reports show that such plants are also used by some indigenous people in traditional medicine (Watt and Breyer-Brandwijk, 1962; Mugera, 1965; Verdcourt and Trump, 1969; Kokwaro, 1976). When G. latifolia is consumed by animals it is known to cause death with clinical and postmortem signs of acute haemorrhagic gastroenteritis. The pathology is accompanied by abdominal pain and rapid death.

There have been several reports of poisoning in livestock and people either because the plant material was ingested accidentally or because its dosage was not properly determined before use. The plant is known to be exceedingly poisonous to livestock especially on sprouting after fire outbreaks and armyworm invasions. In Ukambani the plant has been used as an abortifacient and deaths have been reported in humans following this application. The plant is also reported to have been used for homicidal purposes. A moderate amount of the root brew is drunk as a purgative and emetic for the treatment of other poisonings. The plant has even been employed as an antidote to snake bite poison.

We have been asked on several occasions to carry out postmortem examination of cattle allegedly poisoned maliciously with the plant leaves. Cases of human poisoning after improper therapeutic uses of the plant have been reported.

The aim of our experiment was to investigate and document the clinical signs and pathological lesions induced by this plant to help in future diagnosis of Gnidia toxicity in livestock and humans by studying the effect of the toxic component of the plant in different organs of the live animal. The study was also undertaken to determine whether these effects could explain the extensive usage of the plant as a herbal medicine.

This paper reports an investigation on the toxicity of G. latifolia in cattle.

Materials and Methods

Plants

G. latifolia was collected at Kilungu and Kilome hills of Machakos district.
The material consisted of the leaves, the flowers and the bark of young stems. The young branches and leaves were collected and transported to the Faculty of Veterinary Medicine, Kabete. They were then dried at shade temperature (22-25°C) for two to three days and the withered leaves separated. It was then possible to carry out separate experiments using the leaves and bark. The flowers were picked separately before drying. The leaves and flowers were further spread out and dried for two more days on gunny bags and packing paper sheets at the same temperature (22-25°C). Once dry, the leaves were ground separately into a fine powder using an electric grinding machine. The thinner young branches with the bark were ground separately using the same machine.

**Animals**

Sixteen high grade (Guernsey, Ayrshire and Friesian) bull calves aged 9-12 months old were used in this study. All animals were purchased locally and housed in pens at the Animal compound of the Faculty of Veterinary Medicine. They were examined clinically and treated for any parasites. They were kept for two to three weeks before the commencement of the dosing experiments. Only those animals which proved clinically sound were used.

**Dose and Feeding regimen**

The calf toxicity studies were carried out separately for each group of animals so as to observe daily clinical parameters in the treated groups. The dosage, feeding regimen and survival data on the calves are summarized in Table 1. Each animal was weighed and the dose rates determined. The appropriate weight of the Gnidia material was then weighed into a clean bottle, suspended in clean tap water and then used for drenching the calves. The drenching was carried out with either a long-necked bottle or a stomach tube. The dosages varied from 100 gm/kg 2 gm/kg body weight. Four control calves were fed dry rhodes grass hay and clean water only.

**Clinical examination**

Daily clinical examination was carried out on the treated and control calves. The parameters recorded were the appearance of the haircoat, the animal’s demeanour, the rectal temperature and the respiratory rates. The digestive system was also examined. Special emphasis was laid on the consistency of the faeces, the frequency and strength of the ruminal contractions and intestinal borborygmi. Clinical examination was carried out in the mid-morning before the drenching and the animals were bled at the same time. Routine clinical observations were also carried out in the mid-afternoon on the treated calves.

**Postmortem examination**

All animals which succumbed to the toxicity were subjected to a thorough postmortem examination. The ones that died during the night were autopsied at about 9 o’clock the next morning while those which died during the day the postmortem examinations were carried out between 3 and 4.30 of the same afternoon. Surviving animals were drenched with more Gnidia material. The amounts of material used were based on the animals’ original body weights. Tissues were obtained for histopathological examination. Routinely, the histological tissues were obtained from the liver, kidneys, heart, lungs, spleen, lymph nodes, intestines and the brain.

**Histopathology**

The histological tissues were fixed in 10 percent buffered neutral formalin for at least 48 hours. The fixed tissues were further trimmed and embedded in paraffin wax for a further 24 hours before being cut into thin sections.
which were mounted on glass slides and stained with haematoxylin and eosin.

**Haematology and Protein analysis**

Routine haematological examination was carried out to determine the packed cell volume (PCV), red blood cell (Rbc) counts, white blood cell (Wbc) counts and the total protein (TP) levels. Blood urea nitrogen (BUN) levels were also determined. The activity of the serum enzymes aspartate amino transferase (AST; formerly SGOT), lactate dehydrogenase (LDH) and Creatine phosphokinase (CPK) were determined.

**Results**

**Clinical Observations**

The feeding regimen, dose rates and the survival data of the calves are summarized in Table 1. Clinical changes were observed within a few hours in calves receiving high doses (1gm–2gm/kg).

<table>
<thead>
<tr>
<th>Calf No.</th>
<th>Body weight</th>
<th>Dosage Rate per Kg.</th>
<th>Survival time (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>74</td>
<td>100 mg leaves</td>
<td>142</td>
</tr>
<tr>
<td>10</td>
<td>116</td>
<td>&quot;</td>
<td>51</td>
</tr>
<tr>
<td>11</td>
<td>154</td>
<td>&quot;</td>
<td>36</td>
</tr>
<tr>
<td>12</td>
<td>82</td>
<td>&quot;</td>
<td>38</td>
</tr>
<tr>
<td>7</td>
<td>81</td>
<td>200 mg leaves</td>
<td>28</td>
</tr>
<tr>
<td>8</td>
<td>96</td>
<td>&quot;</td>
<td>31</td>
</tr>
<tr>
<td>5</td>
<td>98</td>
<td>500 mg leaves</td>
<td>28</td>
</tr>
<tr>
<td>6</td>
<td>112</td>
<td>&quot;</td>
<td>12</td>
</tr>
<tr>
<td>2</td>
<td>54</td>
<td>1 gm leaves</td>
<td>19</td>
</tr>
<tr>
<td>1</td>
<td>82</td>
<td>2 gm leaves</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>58</td>
<td>1 gm bark</td>
<td>16</td>
</tr>
<tr>
<td>3</td>
<td>81</td>
<td>2 gm bark</td>
<td>18</td>
</tr>
</tbody>
</table>

The signs observed included abdominal pain, depression, grinding of the teeth, grunting, excessive salivation, lacrimation and nasal discharges. The animals became dull on the second day with signs of hind leg weakness, inappetence, dry muzzles and muscular fasciculations.

The calves which received low doses were slightly depressed, had poor appetite and showed excessive thirst accompanied by grinding of the teeth. They then either developed diarrhoea or passed partially undigested loose faeces. Calf No. 1 which received 2 gm/kg leaf material died on the fourth day while No. 3 on 2 gm/kg bark material survived for 18 days. The calves which received lower doses survived for longer periods but they invariably developed signs of weak hind legs and muscular tremors and a tendency to lie down most of the time. The group of calves which received 200 mg/kg/day took much longer to develop similar clinical picture. The development of submandibular oedema was more pronounced in these chronic cases.

**Postmortem Findings**

The acute cases showed slight distension of the abdomen, eversion of the anus and slight rigor mortis. Gross gastrointestinal changes included haemorrhages of the submucosa of the rumen, abomasum and intestines. The abdominal and thoracic cavities of the acute cases showed moderate effusion of serosanguinous fluid. The more chronic cases showed marked ascites and hydrothorax with straw-coloured fluid which measured as much as two litres in the thorax of one calf. The pericardial sac of the chronically intoxicated animals contained 100-150ml. of similarly straw-coloured effusion. All the prominent joints, i.e. hips, stifles and shoulder had excessive serous yellowish fluid and all fatty tissue was degenerated into a gelatinous mass.

The livers in acute cases were congested, friable and showed subcapsular necrotic zones. Chronic cases had whitish subcapsular patches especially on the parietal surface. The liver capsule was firmly attached to the parenchyma in some of the white zones. Histologically, the whitish zones proved to be areas of fibrosis following parenchymal cell necrosis (Fig. 1). Some of the acute cases showed hyaline droplet formation and deposition in the sinusoidal spaces.
Fig. 1: Calf Liver: Extensive fibrosis in the portal triad of a hepatic lobule. The periportal zone shows increased fibroblastic cell formation in chronic *Gn. latifolia* poisoning. (H.&E., x 250)

Fig. 2: Calf Lymph Node: Marked lymphocytic cell degeneration in the lymph follicle of a markedly congested lymph node of a calf fed *Gn. latifolia* (H.&E. x 400)

Fig. 3: Calf Kidney: Increased fibroblastic proliferation and hyaline deposition in the proximal tubule of calf fed *Gn. latifolia* (H.&E. x 250).
The spleen and lymph nodes were grossly pale and on sectioning showed excessive gelatinous oedema with a marked reduction of cortical tissue. Histologically, these lymphoid organs showed lymphocytic degeneration with areas of cellular depletion in the lymph node and splenic follicles (Fig. 2). In chronic cases there was a relative increase in the amount of fibrous tissue in the lymph follicles, and an accompanying increase in the presence of mononuclear cell infiltration.

The kidneys were friable in the acute cases while they looked pale and oedematous in the chronic ones. Histological examination showed areas of slight interstitial fibrosis with hyaline deposition and albuminous casts in the descending tubules (Fig. 3). The lungs appeared grossly congested and slightly oedematous but the changes were not apparent in the histological sections. The heart also showed petechial and ecchymotic haemorrhages.

**Haematology and Protein findings**

The PCV showed a gradual drop which was more marked on the last two or three days before death. The Rbc counts showed no change although there were day to day variations. There were no changes observed in the total protein levels, even in the chronic cases that showed a generalized oedema and fluid effusion into the body cavities. The Wbc counts showed a marked drop which was due mainly to a lymphocytopenia. There was a drop in the counts from 8,000 cells per cubic millimetre (cu.mm.) at the beginning of the feeding to values less than 3,000 cells per cu.mm. before the calves died. The BUN levels initially remained low (5 to 15 mg/100 ml) but showed an increase to values higher than 50 mg per 100 ml before the calves died. The LDH activity increased from values of 220 to 260 Wacker units (W.u.) per 100 ml serum at the beginning of the drenching to values of 1,000 W.u. in the severely intoxicated animals. There were no changes in the activity of the AST (formerly SGOT) and CPK enzymes.

**Discussion**

The results of the calf feeding experiments confirm that *Gn. latifolia* (Mein) is toxic to animals. Animals receiving high doses (500 mg – 2 gm/kg) showed clinical disturbances. Nwude and Parsons (1978a) reported the intoxication of various species of domestic animals with *Lasiosiphon Kraussianus*. The changes they observed were similar to those earlier reported by Terblanche *et al.* (1966) from feeding *Gn. burchellii* to domestic animals. The nasal and ocular discharges in the intoxicated animals were due to the irritation caused by the toxic principle(s) of *Gn. latifolia*. Alexander (1928) reported that people employed in the preparation of *Gn. anthylloides* material experienced an intense irritation of the throat and nose. It would appear that the same irritant can be carried in the blood to the eyes and nose (Nwude and Parsons 1978a). This appears to be a common property of the *Gnidia* plants and the same irritant principle(s) could be responsible for the gastrointestinal lesions observed in the present work. Nwude and Parsons (1978a) further thought that the principle(s) was (were) a corrosive poison(s).

Marked serous fluid effusion into body cavities was observed in these experiments. This was accompanied by oedema of the connective and fatty tissues. There was, however, no change in the blood protein levels even though there were demonstrable renal injuries which could have lead to loss of blood proteins. Both the liver and kidneys are primary targets in many types of chemical poisonings. As yet the physiological and biochemical basis for drug interference in cellular function is poorly understood (Farnsworth, 1966; O’Gara *et al.*, 1971). Cellular damage in the intoxicated kidneys, liver and the adrenal bodies interfere with the vital functioning of these organs.

*Gn. latifolia* is extensively used in traditional medicine in the management of swelling and tense enlargements. It appears that it suppresses the
production of the mononuclear cell series. There was a marked lymphopaenia which was likely to have resulted from direct injury of the lymphoblastic cells in the lymph follicles. Nwude and parsons (1978a) reported submucosal infiltration of mononuclear cells in the L. Kraussianus poisoning. They also postulated that the active principle(s) of L. Kraussianus either caused the lysis of the lymphocytes or the release of adrenocorticotropic substances which caused lymphopaenia. The present experiment with Gn. latifolia has not shown any submucosal cellular infiltration. The injury demonstrated in the lymphocytic follicles of the spleen and lymph nodes would suggest the origin of the lymphopaenia. This would tend to support the hypothesis of lymphocytic lysis as the cause of lymphopaenia. This cytotoxicity seems to occur at the lymphoblastic stages of the maturation process. We could therefore postulate further that the immature stages of the lymphocytic cell series are more susceptible to the toxic principle(s) of Gnidia plants. It affects also the other cells of glandular and reticuloendothelial systems causing capillary damage and leading to haemorrhages.

Acknowledgements

We are indebted to Messrs A. Mangara, J. Muongi and J. Mungai for assistance in preparation of the Gnidia material and handling the experimental animals. We are also grateful to D. Sembe and Kahara D. Njau for laboratory assistance and to Miss C.W. Kariuki for typing the manuscript.

References


Received for publication on 11th August, 1981.
The Effects of Extracts of Fresh Leaves of *Vernonia Amygdalina* (DEL) in pregnant local Albino Mice

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and

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Summary
Differential solvent extracts of fresh leaves of *Vernonia amygdalina* (Del) were tested for their abortifacient properties in 12-13 days pregnant local albino mice. The mice treated with methyl alcohol extract (75mg and 150mg) aborted within twenty-four hours after the last dose.

The chloroform extract was found toxic at relatively low doses and the animals treated with the aqueous extracts littered normally.

Introduction
*Vernonia amygdalina* (Del) is a cultivated tropical shrub or small tree (1.8-4.5m high) that grows abundantly in parts of Africa, Australia and America (Hutchinson and Dalziel, 1963).

Various medicinal properties have been ascribed to the roots, bark and leaves of this plant. In Angola, the bark of both the root and stem is used as a tonic in fevers and is a remedy for stomach upsets. In West Africa where the shrub is grown in compounds, the young twig is chewed as a stomachic and the leaves are used in soup. The Zulus also use the fruit as food and as a remedy for schistosomiasis (Watt and Breyer-Brandwijk, 1962).

Puri and Talalaj (1964) in their survey of some plants used in native medicine in West Africa claimed that the root, bark and leaves of this plant are used as cure for gonorrhoea, skin diseases and parasitic infections in addition to being used as febrifuge, diuretic, antiscorbutic, purgative and tonic. Singha (1966) in his survey also made similar claims.

In the search for tumour inhibitors, Kupchan *et al.* (1969b) reported that a chloroform extract of *V. amygdalina* was found to show significant inhibitory activity *in vitro* against cells derived from human carcinoma of the naso-pharynx (KB) in tissue culture. They isolated two cytotoxic sesquiterpene lactones; vernodalin and vernomygdin from this extract. Similar compounds, vernolepin and vernomenin, have also been isolated from an alcoholic extract of *V. hymenolepis* (A. Rich), (Kupchan *et al.* 1969a).

In April 1978, a farmer reported that two of his three West African dwarf sheep had recurrent late abortions in their last three pregnancies. The third sheep, a primipara, had a premature lamb. The sheep which were zero grazed in a compound which contained many *V. amygdalina* plants, were known to have been eating the leaves of these plants any time they were short of cut forage. He was advised to transfer the sheep to an enclosure that had no *V. amygdalina* plants. This was done and subsequent pregnancies were carried to full term. No literature available to the authors mentioned the abortifacient property of this plant.

This work describes the screening of *V. amygdalina* leaves for abortifacient properties. It was motivated by the
suspicion that the leaves may have caused the abortions in the above sheep.

**Materials and Method**

A total of 66 local albino mice 12-13 days pregnant were used. They were caged in groups of six and given mice cubes and water ad-libitum.

Five extracts (methyl alcohol, acetic acid in water (ratio 1:9), alkaline water (10% Sod. bicarbonate), water puris and chloroform) of the fresh leaves of the plant were used. After evaporation of the solvent in vacuo, the crude extracts were used as 10% suspensions.

One percent Tween 60 in normal saline served as the vehicle. The method of extraction is similar to that used by Iwu and Ohiri (1980).

Two groups of mice were allotted to each extract and one group to the vehicle as control. One of the two groups assigned to each extract was given 75mg and the other 150mg of the extract per Os. The control group was given 1.5ml of the vehicle. This represents the volume of the reconstituted extracts which contained 150mg of the extracts.

Dosing was carried out in the morning and was done with a 20 gauge oral administration needle fixed to a 5ml syringe. The mice were dosed for three consecutive days except where death occurred before the third dose. After the abortions, the surviving (after chloroform euthanasia) and the dead mice were autopsied in order to find out the extent of abortion.

**Results**

Table 1 shows the effects of the five extracts of *V. amygdalina* and the vehicle on pregnant local albino mice.

Twenty-four hours after the third dose, 46 and 45 aborted foetuses were counted in the cages containing the mice which had 75mg and 150mg of the methyl alcohol extract respectively. One mouse in the 75mg group and two mice in the 150mg group were dead.

At autopsy (after euthanasia of the living mice) circumscribed haemorrhagic areas were observed in the endometrium of all the mice including the dead ones. These haemorrhagic areas were sites of foetal attachments. While the uteri of the euthanised mice were completely evacuated of foetuses, the 3 dead mice had 1, 2 and 2 foetuses respectively left in their uteri. Thus, the dead mice aborted some foetuses before death. All the aborted foetuses were encased in their placentae. Abortions did not occur

<table>
<thead>
<tr>
<th>Extracts</th>
<th>No. of mice</th>
<th>Death without abortion</th>
<th>Death after abortion</th>
<th>Normal littering</th>
<th>Death without abortion</th>
<th>Death after abortion</th>
<th>Normal littering</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control*</td>
<td>6</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Methyl alcohol</td>
<td>6</td>
<td>5</td>
<td>Nil</td>
<td>1</td>
<td>Nil</td>
<td>4</td>
<td>Nil</td>
</tr>
<tr>
<td>Alcohol</td>
<td>6</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>Acetic acid in water</td>
<td>6</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Alkaline water</td>
<td>6</td>
<td>Nil</td>
<td>2</td>
<td>Nil</td>
<td>4</td>
<td>Nil</td>
<td>3</td>
</tr>
<tr>
<td>Puris</td>
<td>6</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Chloroform</td>
<td>6</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*1.5ml 1% tween 60 in normal saline.
in the groups given the other extracts and in the control group. Deaths were recorded as shown in the table. Apart from the two groups which had the chloroform extract in which all the twelve mice died within 12 hours after the first dose, the surviving mice dosed with the other extracts and the vehicle littered normally 7-8 days from the start of the experiment.

Discussion

Puri and Talalaj (1964), and Singha (1966) ascribed some medicinal properties to the roots, bark and leaves of *V. amygdalina*. Cytotoxic compounds, vernodalin and vernomygdin, have also been isolated from the chloroform extract of the leaves of this plant (Kupchan et al. 1969b). The results of this screening exercise have added a new factor to the properties of *V. amygdalina*.

The results from this study indicate that the methyl alcohol extracts of the leaves of this plant caused abortion in the third trimester of gestation in all the twelve mice. The abortifacient factor(s) appeared very potent in the mice since the uteri were completely evacuated of foetuses except in those that died before the action was completed. The few foetuses left in the three dead mice would probably have been aborted if the mice had not died while aborting. The foetal placentae were expelled along with the foetuses. The deaths observed may indicate the toxicity of this extract to pregnant mice.

The chloroform extract was most toxic. All the mice dosed with this extract died within 12 hours after the first dose. The cytotoxic compounds isolated by Kupchan et al. (1969b) from a similar extract, may have been responsible for the extreme toxicity. The few deaths and the normal littering of the other mice in the groups dosed with the other extracts may indicate the absence of the abortifacient factor(s) in these extracts. The absence of deaths and abortions in the control group shows that the vehicle (1% tween 60 in normal saline) which was used to reconstitute all the extracts is not abortifacient per se nor is it toxic to pregnant mice.

The abortions which occurred with the methyl alcohol extract, have increased the suspicion of the authors that the leaves of *V. amygdalina* were responsible for the abortions observed in the sheep. If this suspicion is right, then the abortifacient principle(s) do not have permanent effects on sheep as to prevent future conceptions because the sheep not only were able to conceive after the abortions but were also able to lamb successfully when they were prevented from eating the leaves of the plant.

The results of this study are also important because the leaves of *V. amygdalina* are eaten by people in West Africa, especially the Igbo-speaking people of Nigeria. The leaves are however mashed and washed in several changes of water until the bitter taste is removed before being used for making soup. The washings and the subsequent cooking probably render this vegetable innocuous to pregnant women.

Work is continuing in our laboratories to isolate and characterise the major constituents of *V. amygdalina* and subject these compounds to the pharmacological tests described above.

Acknowledgement

The authors are grateful to Mr. C.C. Abana of the animal laboratory unit, Department of Veterinary Pathology and Microbiology, University of Nigeria, Nsukka, for his technical assistance.

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tory at Jamu (Kashmir) India on December 3-5, 1964.


Received for publication 23rd June, 1981
Bovine anaplasmosis and babesiosis are enzootic in Nigerian native cattle but outbreaks of clinical disease are relatively rare. The status of these diseases among exotic breeds in Nigeria is unknown. This paper reports severe losses among Friesian cattle in Vom apparently due to anaplasmosis and babesiosis occurring concurrently.

Eighty Friesians (78 heifers and 2 bulls) were recently purchased from the United Kingdom by the National Research Institute, Vom. They arrived in the country by air in the month of February 1980. All the heifers were in the latter half of pregnancy at the time they arrived in Vom. Within two weeks of their arrival in Nigeria, two had died of shipping fever and another of dystocia. Those remaining were immediately treated against shipping fever with chloramphenicol. No further deaths were reported until approximately three weeks later, when animals started to die one after the other. Death and abortions were recorded almost daily for two weeks before anaplasmosis and babesiosis were diagnosed and the outbreak was brought under control with the use of imidocarb dipropionate (IMIZOL, Wellcome)*. At the end of the outbreak, ten deaths and thirteen abortions had been recorded.

Clinical manifestations observed during the outbreak included high fever (body temperature 104°–107°F), anorexia, haemoglobinuria, low packed cell volume, anaemia and general weakness. Severely affected animals were found squatting and they found it difficult to stand.

Parasitological findings from blood smears made from dead and aborted animals and stained with Giemsa, revealed high parasitaemia with *Anaplasma marginale* and *Babesia bigemina*. *Theileria mutans* parasites were also found in the blood of these animals, but they are known to be non-pathogenic in cattle. The degree of parasitaemia in the remaining animals that survived and did not abort varied from low to very high. Smears from the liver, lung, kidney and spleen, stained in Giesma, were found to contain *Anaplasma* and *Babesia* infected erythrocytes.

Necropsy findings were marked jaundice, anaemic conditions of the mucous membranes and the skeletal muscles, blood stained urine, water blood and moderately enlarged livers.

Treatment of the remaining animals, some of which were also showing moderately serious symptoms, with imidocarb dipropionate using a single dose of 300mg/100kg bodyweight of a 12% solution administered intramuscularly was followed by rapid disappearance of the clinical symptoms and of *Anaplasma* and *Babesia* organisms in the blood smears.

Two main factors may have precipitated the present outbreak. The first is breed susceptibility to *Anaplasma marginale* and *Babesia bigemina* and the second is climatic and pregnancy stress. Exotic breeds, especially those originating from temperate climates, and in particular pure-breeds of high performance milking cows, are known to be very susceptible to anaplasmosis and babesiosis (Ristic 1968, Riek 1968).
Furthermore, it appears that these animals were relatively old at the time of first exposure which would have predisposed them to a more severe clinical infection. The Friesian cattle were flown from the United Kingdom in February and came from a cold and wet winter to the hot and dry climate of the Jos Plateau. The sharp change in weather conditions may be expected to have some effect on the ability of the animals to resist massive infections. In order to minimise losses in the future, these two factors — climatic and susceptibility should be borne in mind when importing exotic breeds into Nigeria.

Before the recent introduction of imidocarb (MIZOL) into the Nigerian market, tetracyclines and Berenil were the drugs of choice for the treatment of anaplasmosis and babesiosis respectively in Nigeria. Apart from its high effectiveness against both anaplasmosis and babesiosis as observed in the present instance, imidocarb is more convenient to use against mixed infections of this nature and also less expensive as it comprises a single dose treatment.

Acknowledgement

The authors wish to thank the Director, National Veterinary Research Institute, Vom, for his permission to publish this manuscript.

References


Received for publication on 5th August, 1981.

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The indigenous West African dwarf sheep and goats live under perennial conditions and varying degrees of tsetse challenge and are said to be trypanotolerant (ILCA, 1979). Kramer (1966) found 13.9% of goats and 11% of sheep infected with *Trypanosoma congoense* and *T. vivax* in a group of villages in the Nsukka area of eastern Nigeria. Clinical symptoms of trypanosomiasis in sheep and goats have been described (Losos and Ikede, 1972). Clinical trypanosomiasis is common in southern Nigeria (Akerejola et al., 1979). Trypanosome infection among tolerant animals may result in poor health and hence lowered resistance to other diseases (ILCA, 1979). Malnutrition and other stress factors can predispose animals to, or exacerbate clinical manifestations of trypanosomiasis.

In the southern derived savanna zone of Oyo State of Nigeria, 44 dwarf sheep and 38 goats were monitored for disease incidence at the Fashola ILCA experimental station between November 1979 and October 1980. Prophylactic measures included strategic drenching against helminthiasis, monthly dipping against ectoparasites, and annual vaccination against *Peste des petits ruminants* (PPR) and enterotoxaemia. Animals were examined routinely every week for subclinical and clinical incidence of trypanosomiasis. Thick and thin blood smears were made early in the morning and late in the evening from blood collected from jugular or ear veins. Slides were air-dried and fixed with methanol for 3 minutes. Thick smears were stained with Giemsa for 30 minutes for detection of trypanosomes, while thin smears were stained similarly for 45 minutes for identification of the trypanosome species and other blood protozoa under oil immersion objective. In addition, wet smears were made for detecting types of motility exhibited by the trypanosomes. Haemoglobin concentrations of clinical cases were also determined.

Of the total 44 sheep and 38 goats monitored, 18.1% and 34.3% respectively were positive for trypanosomiasis. In the sheep, *T. vivax* (75%), *T. congoense* (12.5%), and *T. brucei* (12.5%) and, in the goats, *T. vivax* (84.6%) and *T. congoense* (15.4%) were identified from positive cases.

In the 4.5% of the sheep with clinical infection, the symptoms observed included anaemia (with haemoglobin concentration of 5-6 gm %), intermittent fever, conjunctivitis, dullness, weakness, anorexia, dyspnoea, frequent urination, recumbency, incoordination, and oedema of the knees. Mortality was 4.5%. *T. vivax* and *T. congoense* were associated equally with the losses.

The symptoms observed in 13.2% of goats with clinical infection included segregation from the rest of the groups in the shelters, pyrexia (40.5° C—41.5° C), pale mucous membranes of the eyes and mouth, dullness, weakness, drooping of the head, anorexia, emaciation, loss in weight, dyspnoea and anaemia (with haemoglobin concentration of 4-5gm %). One abortion case was observed during the febrile period. Mortality was 10%, with 7.4% associated with *T. vivax* and 2.6% with mixed infection caused by *T. vivax* and *T. congoense*.

The incidence of trypanosomiasis in the sheep and goats combined was 3.7% in the dry season (November—February), and 19.5% in the rainy season (March—October). The incidence in the goats during the wet season was 31.6%. Star-
vation was one of the factors considered to predispose the goats to infection, as they habitually refused to graze or browse during wet periods. Other blood parasites observed were Babesia spp., (0.1%) in both sheep and goats combined.

There appeared to be little or no tsetse challenge at Fashola stock farm when the survey was carried out. The sheep and goats might therefore have already been in a state of premunition to the local strain of trypanosomiasis when purchased. In this case their resistance would have been broken down so that parasitaemias, with clinical and subclinical cases of trypanosomiasis, occurred. On the other hand, mechanical transmission by Stomoxys spp. from already infected to uninfected animals within the flock could also have occurred. *T. vivax* appeared to be the most pathogenic species encountered.

References


Received for publication on 9th June, 1981.
Skin Bacterial Flora of Friesian Cattle with an advanced infection of Cutaneous Streptothricosis in Nigeria

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Summary

150 skin swabs taken from diseased and non-diseased body sites of thirty Friesian Cows with advanced infection of cutaneous streptothricosis were cultured and examined bacteriologically with a view to identifying other micro-organisms on the skin surface. Apart from Dermatophilus congolensis (D. congolensis) the causative organism, coagulase-positive Staphylococcus pyogenes (S. pyogenes) and Pseudomonas aeruginosa (P. aeruginosa) were present on the skin surface beneath the scab tissue while α-haemolytic Streptococcus and Staphylococcus epidermidis (S. epidermidis) were seen on the scab surface. Both coagulase positive S. pyogenes and coagulase – negative S. epidermidis, β-haemolytic Streptococcus, Escherichia coli (E. coli) and a few Bacillus species were noted on non-diseased abdominal skin sites, but no Bacillus species were seen on the diseased sites of the skin.

Introduction

A study of the normal skin bacterial flora of some cattle breeds in Nigeria had earlier been undertaken and reported by Nwufoh and Amakiri (1981). The results show that coagulase-negative S. epidermidis, β-haemolytic Streptococcus, E. coli and Bacillus species organisms were consistently seen on clinically normal White Fulani, N'Dama and Friesian cattle. In the N'Dama, the Bacillus species were very prevalent with few Staphylococcus organisms while in the Friesian and White Fulani, the latter organisms were the most prevalent.

It was deemed necessary to investigate the skin bacterial flora of cattle with an established streptothricosis infection to elucidate the type of organisms present and their probable influences when D. congolensis, the main causative agent is established.

The present paper reports the bacterial flora identified on the skin of Friesian cows with advanced streptothricosis infection.

Materials and Methods

Thirty adult Friesian cows that had had localized chronic lesions of streptothricosis for 3 months and over were kept in a fly-proof house and sampled in this study. Duplicate skin swabs were taken from the scab surfaces, and skin surfaces after the removal of the scabs in the dorsal and neck region of these animals. Samples were also taken from apparently healthy lesion-free skin sites of the same animals from the ventral abdominal wall, ventral neck, dewlap and forehead.

Swab samples were placed in tryptose soya broth (TSB) for 24h and cultured on blood agar at 37°C for 24h and for 48h on blood agar under CO₂ enriched atmosphere for D. congolensis isolation. Discrete colonies were later subcultured on blood agar twice to obtain pure cultures of the organisms. The morphology of the various organisms was later examined microscopically after staining by Gram’s technique. Colonies resembling Staphylococcus species morphologically were further examined for coagulase production with human plasma. Various biochemical tests were also carried out to confirm the identification of the organisms using standard methods of

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**Department of Veterinary Microbiology and Parasitology, University of Ibadan, Ibadan, Nigeria.
Results

There were differences in the types of organisms found on the various skin surfaces sampled as shown below:

**Scab Surface** – Colonies of α-haemolytic *Streptococcus* and *S. epidermidis* were mainly isolated from the scab surface of the dorsum and neck regions. *D. congolensis* organisms were not isolated from these surfaces.

**Skin Surface underneath Scabs**

*D. congolensis* organisms were the most abundant on these sites and they grew best at 48h incubation. Other bacterial organisms identified on these areas included coagulase positive *S. pyogenes* and *P. aeruginosa*.

**Lesion-free skin sites of infected cattle**

On these locations, coagulase – positive *S. pyogenes*, *S. epidermidis*, β-haemolytic *Streptococcus*, *E. coli* and a few *Bacillus* species were the most predominant colonial types. *D. congolensis* organisms were also not isolated from swabs taken from these sites.

Discussion

The type of bacterial organisms found on lesion-free sites of infected cattle were similar to those identified in our earlier study on healthy cattle by Nwufoh and Amakiri (1981) except for the absence of coagulase positive *S. pyogenes* in the former. The lesion free skin sites of infected animals thus appear to harbour more bacteria than those of apparently healthy animals in agreement with the finding of Hallwell and Ihrke (1976) and Kristensen and Krogh (1978).

The bacterial species *S. pyogenes* and *P. aeruginosa* found on the skin underneath scab surface are probably secondary invaders which may be responsible for the extensive suppuration and severe toxæmia characteristic of chronic streptothricosis infection by Jubb and Kennedy (1970). Also, the necrotic or gangrenous foul-smelling dermatitis or the pyogenic infections and cellulitis that develop in streptothricosis infection have been attributed to secondary organisms by Jubb and Kennedy (1970) which may well be those identified in the present study.

The presence of some organisms and absence of others in and around lesions containing *D. congolensis* indicates that the micro-environment created by this infective organism is favourable to the growth of some organisms while unfavourable to others. Also significant is the finding that *Bacillus* species were few on lesion-free sites of infected cattle and completely absent on scab surfaces and the skin underneath scab lesion. This strengthens the suggestion by Nwufoh and Amakiri (1981) that the *Bacillus* species most predominant on the skin of the N’Dama, may be inhibitory to the growth of *D. congolensis in vivo*, and also lends weight to other findings by Ojo (1975) that *Bacillus pumilus* inhibited *D. congolensis in vitro*. Alternatively, the absence of this organism on infected skin locations could mean that once *D. congolensis* becomes established, the environment created became unfavourable for the growth of *Bacillus* species. The absence of *D. congolensis* organisms in normal skin sites of infected animals further indicates that the organism is not widespread on the skin surface and may therefore probably not be a normal skin resident.

In conclusion, this study establishes that there are other organisms present on infected skin of cattle either within or around lesions of Streptothricosis that could be actors or co-actors with *D. congolensis*, the main causative agent. These organisms may well be significant in determining the pathogenesis of the disease, especially in accounting for the suppurative inflammation associated with the infection.

Acknowledgements

The authors are grateful to Mr. M.A. Arasi for his technical assistance in the
bacteriological work and to Mrs. V.O. Ayanwale for typing the manuscript.

References


Received fro Publication on June 23, 1981.
Geographical Distribution of SALMONELLOSIS GALL in Africa

OAU/STRC
INTERAFRICAN BUREAU
FOR ANIMAL RESOURCES
MAP No. 456
1981

- Foci reported
- Widespread
- Enzootic/Sporadic but no Foci reported
- No official information available

Geographical Distribution of SHEEP POX in Africa

OAU/STRC
INTERAFRICAN BUREAU
FOR ANIMAL RESOURCES
MAP No. 457
1981

- Foci reported
- Widespread
- Enzootic: Sporadic but no Foci reported
- No official information available

Geographical Distribution of BOVINE TUBERCULOSIS in Africa

10° 15° 20° 25° 30° 35° 40° 45° 50° 35°

OAU/STRC
TERAFRICAN BUREAU
FOR ANIMAL RESOURCES
MAP No. 458

Faculty reported
Widespread
Enzootic/Sporadic but no Faculty reported
No official information available

Geographical Distribution of ANAPLASMOSIS in Africa

OAU/STRC
INTERAFRICAN BUREAU
FOR ANIMAL RESOURCES
MAP No. 459
1981

■ Foci reported
X Widespread
☑ Enzootic/Sporadic but no Foci reported
☐ No official Information available

Geographical Distribution of BOVINE PIROPLASMOSIS in Africa

OAU/STRC
INTERAFRICAN BUREAU
FOR ANIMAL RESOURCES
MAP No. 460
1981

- Foci reported
- Widespread
- Enzootic/Sporadic but no Foci reported
- No official information available

Geographical Distribution of BOVINE TRYPANOSOMIASIS in Africa

OAU/STRC
INTERAFRICAN BUREAU
FOR ANIMAL RESOURCES
MAP No. 481
1981

- Foci reported
- Widespread
- Enzootic/Sporadic but no Foci reported
- No official information available

ABSTRACTS
Vol. 30 No. 3 – Nos. 61–83

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75. Development and Distribution of Desophagostomum columbianum in Young Lambs after Oral or Intraruminal Infection.

76. Immunization With Irradiated Larvae Against Dictyocaulus filaria in Young Lambs.

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83. Effect of Feeding Digestible Crude Protein Levels and Urea on Rumen Liquor nitrogen Fractions and Volatile Fatty Acids.

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84. The Treatment of Coccidiosis: Studies on the Sensitivity of Recent Field Isolates of Eimeria acervulina Type to Anticoccidial Drugs Given in the Drinking Water.
IBAR/1982  J.W. SIMPSON 61  Bacterial Overgrowth causing Intestinal Malabsorption in a Dog.


AUTHORS' SUMMARY: The case history of a six-year-old female chow chow with intractable diarrhoea is described. The clinical history and symptoms, together with the methods of diagnosis, indicated that the cause was bacterial overgrowth of the intestine leading to a malabsorption syndrome. The method of treatment and the response is also recorded.


AUTHORS' SUMMARY: Evaluation of diagnostic sensitivity and specificity was based on test results of 346 sera from pigs known to be infected and 139 sera from pigs known not to be infected. All sera were tested with a monospecific antigen (serotype 1) and a polyspecific antigen (serotypes 1-5). The sensitivity of the polyspecific antigen was approximately 85% at serum dilution 1:2 and was significantly higher than the monospecific antigen at all serum dilution levels. The specificity of the two antigen preparations was not significantly different at any dilution and increased from approximately 78% at 1:2 to 100% at 1:128.

When pigs from herds with unknown incidence of infection were studied, it was found that a high proportion seroconverted, presumably as a response to subclinical infection. However, the antibody titres waned rapidly. This indicated that seroconversion expresses current or recent infection. Thus the complement fixation test provides a reliable means of diagnosing pleuropneumonia of pigs and might be useful as a tool to control this disease.

IBAR/1982  E.F. LOGAN, D.J. MEENEELY and D.P. MACKIE 63  Enzyme-linked immunosorbent Assay for *Streptococcus agalactiae* antibodies in bovine milk.


AUTHORS' SUMMARY: An enzyme-linked immunosorbent assay (ELISA) for the detection of *Streptococcus agalactiae* antibodies in bovine milk was developed using whole bacterial cells as antigen. Microtitre wells were coated overnight at room temperature with a 1:64 dilution of antigen in 0.05M carbonatebicarbonate buffer at pH 9.6. After washing, milk whey samples diluted 1:40 were added, incubated overnight and again washed. After incubation with rabbit antivovine serum, bound antibody was detected with alkaline phosphatase conjugated sheep antirabbit serum. Using the ELISA, the levels of *Str agalactiae* antibodies in the individual quarters of the mammary glands of cows in a severely infected dairy herd were measured. A high proportion of cows had specific antibody to *Str agalactiae* in one or more quarters. Using ELISA in association with electronic cell count and bacterial isolation, it was possible to identify latent and subclinical carriers of infection.

IBAR/1982  DEBRA A. DEEM and ROBERT H. WHITLOCK 64  Renal Cysts in a Cow with Anorexia Mypocalcemia and Abdominal Pain.


AUTHORS' SUMMARY: A ten-year-old Holstein cow developed anorexia, fever, and recumbency 2 weeks before calving. She was hypocalcemic with concurrent mild pneumonia and mastitis. Despite evidence of impending abortion, parturition was induced and the cow developed metritis. Despite post partum improvement of pneumonia, mastitis and metritis, the cow remained anorexic and had signs of abdominal pain. An exploratory laparotomy via the left flank revealed a greatly enlarged right kidney. On examination through the right flank, 2 large simple renal cysts were found and a nephrectomy was performed. Postoperatively, the cow's appetite gradually improved and she made an apparent clinical recovery.

IBAR/1982  ARELIDES MARTINEZ, ELISA AZNAR, C. VINA Y J.J. MARTINEZ 65  Experimental inoculation of *Mycoplasma bovis* in calves


AUTHORS' SUMMARY: Endobronchial inoculation in calves was performed with a *Mycoplasma bovis* strain isolated from a pneumonic calf in order to determine whether such strain is capable of producing
pulmonary changes. Nine calves were inoculated, 3 with 20 ml of the Dulbecco phosphate buffer (DBP) (modified) culture medium, and 6 with 20 ml of this culture medium containing $10^6$ CFU of M. bovis per ml, and nasal swabs were taken throughout the experiment until slaughter at 4 weeks postinoculation. At necropsy, samples of tissues and lung washings were collected for microbiological investigations. In the nasal swabs from the 6 calves inoculated with M. bovis, the organisms were found from the 11th till the 14th following inoculation. From the lung washings cultured, growths ranging from $1 \times 10^2$ to $5 \times 10^2$ CFU of M. bovis/ml of washing fluid were obtained in 4 out of the 6 calves inoculated with said organism. M. bovis was found to induce pulmonary changes in calves.

IBAR/1982 KNUD BORGE PEDERSEN and KRISTEN BARFORD
The Aetiological Significance of Bordetella bronchiseptica and Pasteurella multocida in Atrophic Rhinitis of Swine


AUTHORS' SUMMARY: Experimental infection with either pure culture of Bordetella bronchiseptica or B. bronchiseptica and a toxin-producing strain of Pasteurella multocida was studied in newborn piglets from 26 sows. Pigs inoculated with B. bronchiseptica alone got mild lesions, whereas pigs inoculated with both organisms showed clinical and pathological signs of atrophic rhinitis, corresponding to the progressive natural disease. The effect of vaccinating sows against B. bronchiseptica during pregnancy was studied.

IBAR/1982 K.T. CHONG and K. APOSTOLOV
The Pathogenesis of Nephritis in Chickens Induced by Infectious Bronchitis Virus.

J. Com. Path., 1982 92 (2) : 199-211

AUTHORS' SUMMARY: Nephritis in chickens caused by infectious bronchitis virus (IBV) was studied by virological, histological and electron microscopical methods. The T strain of the virus caused only mild respiratory signs in both Rhode Island Red (RIR) and White Leghorn (WL) breeds; the 50 per cent mortality induced was due to acute nephritis. All the infected birds developed high titres of antibody to IBV for up to 30 weeks. In spite of the persistence of antibody, about 35 per cent of the RIR developed chronic progressive nephritis. The histology showed varying degrees of pathological changes in the tubules, with relatively unaffected glomeruli. Foci of mononuclear cell infiltration were prominent in the cortex and medulla, particularly in chronic nephritis. Cytoplasmic IBV immunofluorescence was found in all segments of the tubules, but not in the glomeruli. There was no evidence of virus replication in the caecal tonsil and bursa of Fabricius. Evidence of extensive coronavirus replication was found in the cells of the tubules. A large number of viral inclusion bodies as seen containing dark smooth particles 120 nm diameter within a single membrane. Virus was readily recovered from the kidneys as well as faeces of birds with acute and chronic nephritis. It is concluded that direct virus-induced cell lysis is the primary cause of IBV nephritis. In addition, about 50 per cent of the chronically infected birds also developed brush-border auto-antibody.

IBAR/1982 F.A. ATA and K.V. SINGH
Rinderpest in Kuwait

Egyptian Veterinary Medical Association, 1982 42 (1) : 27

AUTHORS' SUMMARY: Rinderpest has been recognized in Kuwait since 1968. It occurred almost annually, causing considerable losses among dairy cattle. The disease mostly occurred during the rainy season especially in December and January of each year. The disease was diagnosed by the agar gel double diffusion test (AGDT) and by virus isolation and identification in bovine kidney (BK) cell culture. The cytopathic effect (CPE) was detected as early as the 4th day post inoculation (d pi). The plaques were small and attained a diameter of 1.5 mm in 10 days. The possible origin and maintenance of the disease were discussed.

IBAR/1982 F.A. ATA
Studies on the enzootic nature of Foot-and-Mouth Disease in Kuwait

Egyptian Veterinary Medical Association, 1982 42 (1) : 107

AUTHOR'S SUMMARY: Foot-and-Mouth disease has been recognized in Kuwait since long time. Type A appears to be the most widespread, while type O occurred sporadically but almost annually. In 1970, type SAT-1 was identified but was soon completely eradicated. Recently type Asia-1 was identified causing widespread infection among dairy cattle. The enzootic nature of the disease seems to be due to carrier or subclinically infected animals. Another possible reason could be a change in the antigenic properties of the vaccine strain due to extinctions and variability of subtypes.
Nomadism, open frontiers, and the importation into the country of slaughtered animals from infected countries were in favour of the spread and persistence of infection in Kuwait.


AUTHORS' SUMMARY: The ELISA and indirect immunofluorescence test were compared on 56 porcine sera which were tested for antibodies to porcine cytomegalovirus. Viral antigens were prepared in cells of a pig fallopian tube line. The ELISA was found to be a sensitive reproducible and practical test to measure specific antibodies to this infection.

IBAR/1982 J.A. HOUSE and R.J. YED-71 LOUTSCHING Sensitivity of Seven Different Types of Cell Cultures to Three Serotypes of Foot-and-Mouth Disease Virus


AUTHORS' SUMMARY: The ability of bovine tongue origin foot-and-mouth disease virus serotypes A, O and C to replicate in seven different types of cell cultures was studied. Primary and secondary calf cultures passed up to five times. Calf thyroid cells lost their susceptibility after two passages. Cryopreserved bovine kidney cell cultures passed three and four times were equivalent in susceptibility to sensitive calf thyroid and bovine kidney cells. Susceptibility to foot-and-mouth disease virus serotype C was most variable among the cells tested.

Lamb testicle and porcine kidney cells were susceptible to foot-and-mouth disease virus while goat and calf testicle and calf lung cells were refractory.

IBAR/1982 D.N. DHAR and R.L. 72 SHARMA Immunization with irradiated larvae against Dictyocaulus filaria in young lambs

Veterinary. Parasitology, 1981, 9 (2) : 125

AUTHORS' SUMMARY: In the Lungworth-endemic areas of Kashmir, 6-10 week old lambs of Karnah and Kashmir Merino breeds were vaccinated with two doses of 50 kr gamma-irradiated larvae of Dictyocaulus filaria, given a month apart. Assessed on the basis of reduced prevalence and significantly lower faecal larval output over an eight-month observation period, vaccinated lambs showed a high degree of resistance to naturally acquired D. filaria infection. The results also show that vaccination against D. filaria provided some degree of protection against infection with other lungworm species.

IBAR/1982 K.M. DASH 73 Development and distribution of Oesophagostomum columbianum in young lambs after oral or intraruminal infection

Veterinary Parasitology, 1981, 9 (2) : 145

AUTHORS' SUMMARY: There was no difference in the establishment or rate of development of Oesophagostomum columbianum in young lambs after infective larvae were administered either orally or by injection directly into the rumen. However, the linear distributions in the intestine of encysted third-stage larvae differed according to the route of infection. The distributions of fourth-stage larvae, after migration to the large intestine, did not differ. It is suggested that the distribution of parasitic third-stage O. columbianum is host-dominated, depending on the site of exsheathment and the rate of passage of ingesta, but that of fourth-stage and adult worms involves active site selection by the parasite.

IBAR/1982 KUNIO DOI, FUJIO FUJI-74 NAMI, AKIRA YASOSHI-MA, HITOSHI OKAWA & AZUSA OKANIWA Lesions in Pulmonary Artery in Feline Dirofilariaiopsis.


AUTHORS' SUMMARY: Pathological observation was carried out on 3 domestic cats spontaneously infected with Dirofilaria immits. Principal lesions were found in a small number of pulmonary arterial branches which corresponded roughly to the site of parasitic infection. Marked degenerative and exudative changes involving the whole layers of the vascular wall and perivascular loose connective tissues were conspicuous in large pulmonary arterial branches containing embolic dead parasites. A certain causal relation may present between disorganized dead parasites and severe necrotic changes.
IBAR/1982  D.E.  JACOBS,  M.T.  FOX,  
75  R.M.  JONES  &  D.H.  BLISS  
Control  of  Bovine  Parasitic  
Gastroenteritis  and  Parasitic  
Bronchitis  in  a  Rotational  
Grazing  System  Using  the  
Morantel  Sustained  Re- 
lease  Bolus.  

The  Vet.  Record,  1982,  110  (17) :  
599-402

AUTHORS’ SUMMARY: Sixty cattle (12 first season 
and 48 second season grazing animals) were allocated 
to three groups according to age and bodyweight. 
Each group was divided into ‘control’ and ‘treated’ 
subgroups. Before turnout, a morantel sustained 
release bolus (MSRB) was administered to each 
animal in the ‘treated’ category. The groups were 
moved from field to field according to the farmer’s 
normal rotational grazing policy. Each field was, 
however divided into two equal halves, one of 
which was reserved for the MSRB treated cattle, while 
the other was used exclusively for the control. Severe 
parasitic gastroenteritis occurred in the first season 
control during early September, while milder di- 
sese affected the untreated animals in the smaller 
of the second season groups. No gastrointestinal 
disease was apparent in the corresponding MSRB treated 
cattle. A mild outbreak of parasitic bronchitis occurred 
in the first year controls during October; there was 
evidence of less severe lungworm infection in the 
matching MSRB treated animals. The larger second 
season group showed no signs of parasitic disease.

IBAR/1982  F. HORCHNER,  H. SCHL- 
76  CHTING,  M.  MERKER,  G.  
WINKLER  &  I.  MULLER  
The  Occurrence  of  Hel- 
minths  of  Calves  in  Burundi.  

Ann.  Soc.  belge  Med.,  1981,  61  (5) :  
413-419

AUTHORS’ SUMMARY: During the dry season in 
autumn 1979 and rainy season in April and May, 
1980 fecal samples of 719 calves under 19 months of 
age from different provinces of Burundi were examin- 
ed on helminths and trematodes and after cultivating 
larvae 111 were differentiated. The prevalence of 
Strongyloides papillosus and Toxocara vitulorum rose 
up to 83% and 58% respectively from the first to the 
third month of life. Up to the 18th month the preva- 
ience of Strongyles and Trichostrongylus attained 
over 90% though the intensity of egg output was low. 
The prevalence of Fasciola gigantica (23%) and of 
Paramphistomum spp. (35%) were lower than among 
89 slaughtered cattle aged from 3 to 15 years.

IBAR/1982  I.M.  AL-SAQR,  K.I.  
77  ALTAIF  and  A.J.  AL- 
SUBAIYD  
Study  on  the  Pathogenicity  
of ostertagiosis  due  to  
Ostertagia  circumcincta  in 
sheep, in Iraq.  

Veterinary  Parasitology,  1981,  9  (2) :  
133

AUTHORS’ SUMMARY: The response of Awassi 
sheep to Ostertagia circumcincta, the most prevalent 
ovine strongylid species in Iraq, was studied. 
A dose of 100000 larvae of O. circumcincta (Iraqi 
strain) induced moderate clinical symptoms of ost- 
ertagiosis. These symptoms were correlated with elevat- 
ed pH of the abomasal fluid and increased plasma 
pepsinogen levels. There was no evidence of larval 
infestation since the majority of the fourth stage 
larvae (L4) continued their development. No appreci- 
able loss of worm population was observed and most 
of the parasites survived and exhibited a prolific 
egg-laying potential six months after initial infection. 
Assessments of the pathophysiological changes were 
performed and correlated with parasitological and 
clinical observations. 
The Awassi breed of sheep, the most prevalent in 
the Arab Middle East, seems to be more susceptible 
to ostertagiosis than other breeds and there was no 
spontaneous self cure in this breed.

IBAR/1982  Sugar  Cane  and  its  By-
78  products  for  Milk  and  Meat  
Production  

World  Review  Animal  Production  
1979,  15  (5) :  4

AUTHOR SUMMARY: The results of works carried 
out in Cuba with the aim of evaluating the possibility 
of utilizing sugar cane and its by-products as diets for 
bovine cattle in the tropics are analysed. 
The addition of urea and sulphur (ammonium 
sulphate) increases sugar cane consumption and milk 
production and substitutes natural protein (fish meal 
and torula yeast) in cows with an average potential of 
10 kg/day. With the supplementation of sugar cane 
fibre residues with a wet mixture containing 40% 
crude protein (75% of N in the form of NPN) 7.5 kg 
of milk can be obtained. 
The chemical treatment of bagasse pith increases 
its DM digestibility and augments the production of 
volatile fatty acids in the rumen. With this material 
8 kg of milk/day can be obtained. 
It is possible to obtain daily weight gains of 0.8 – 
1.0 kg in bulls fed high levels of molasses/urea supple- 
mented with small quantities of forage, natural pro- 
tein and minerals.
IBAR/1982 F.O. OLUBAJO  
79 The Feeding Value of Two Tropical Grass-and Grass-Pineapple Pulp Silage  
*Wid. Review of An. Prod.,* 1981 17 (4) : 3

**AUTHOR’S SUMMARY:** Silages made from guinea (*Panicum maximum*) or elephant (*Pennisetum purpureum*) grass with or without the addition of pineapple pulp at three levels of incorporation were fed to eight White Fulani (Zebu) steers ranging in liveweight from 125 kg to 173 kg in a 4 x 2 x 2 factorial design in two separate experiments.

Dry matter content of silages varied between 20.0% and 27.3% and 15.9% and 19.2% for guinea and elephant grass silage respectively.

The mean dry matter intakes of 42.1 g/Wkg\(^{0.75}\) and 33.7 g/Wkg\(^{0.75}\) for guinea and elephant grass silage respectively were low. This was due to the high moisture content of the silages. The low nitrogen intake was attributed not only to the low crude protein content but also to the low dry matter intake of the silages by the steers.

Digestible energy intake was influenced more by the digestibility of the dry matter than the silage crude protein content. Simple linear regression analysis showed that digestible dry matter intake, gross energy, digestible energy and metabolizable energy intakes can be predicted from dry matter.

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IBAR/1982 H. WILLEKE  
80 Comparison of Selection Schemes for Improving Litter Size in Pigs — Results of a Simulation Study.  
*Livestock Production Science,* 1982, 8 (6) : 535-540

**AUTHOR’S SUMMARY:** In a simulation study, the following four selection methods were compared: I selection of best 50% (parity ignored); II selection of best 50% within parity; III selection of all sows with litter size 8, excluding sows of 1st parity; IV selection of best 50% within full-sib family.

The simulation was based on 348 dam-daughter pairs (German Landrace sows) from one breeding farm. All daughters were born in 1976.

The selection differences for Methods I, II, III and IV were 2.27, 2.07, 1.67 and 1.50 piglets born per litter, respectively. The selection response was 0.10, 0.07, 0.17 and 0.32 piglets per litter. The realized heritability (twice the selection response, divided by the selection difference) was 0.09, 0.07, 0.20 and 0.43, respectively. The result of this study suggests that within full-sib family selection would be the most efficient method for increasing litter size genetically.

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IBAR/1982 D.A. WHITAKER  
82 A field trial to assess the effect of copper glycinate injections on fertility in dairy cows.  
*British Veterinary Journal,* 1982, 138 (1) : 40

**AUTHOR’S SUMMARY:** Two hundred and six blood serum copper analyses from representative groups of milking cows in four Friesian herds in New south Wales produced an average of 0.71 mg/l (SD 0.18, range 0.24 to 1.20). After a period of 12 to 18 months 400 mg of copper glycinate were injected into alternate cows in these herds within one week of calving. There were no consistent or significant differences between treated and untreated groups in the average interval between calving and...
first observed heat, services per conception or first service conception rate. It would appear to be inad-
visable to ascribe poor reproductive performance to subclinical hypocuprosis on the evidence of blood
copper analysis alone. Other factors such as manage-
tment, and energy and protein supply should be taken
into account.

IBAR/1982 M.B. PANDE and P.C. 
83 SHUKLA
Effect of Feeding Digestible 
Crude Protein Levels and 
Urea on Rumen Liquor 
Nitrogen Fractions and 
Volatile Fatty Acids.

*The Indian Veterinary Journal*, 1981, 
58 (11) : 894 and 900.

AUTHORS' SUMMARY: An experiment on growing 
buffalo calves was conducted to find out the changes 
in pH, TVFA, NH₃-N and total-N of SRL. The calves 
were fed two levels of digestible crude protein and 
urea. Time of sampling significantly affected the diffe-
rent parameters studied. pH of SRL was significantly 
higher in urea-fed group as compared to 100 per cent 
DCP fed group. Total volatile fatty acid production 
significantly reduced in urea group as compared to 
lower or higher level of DCP feeding. Ammonia pro-
duction was significantly higher in urea-fed group 
and total nitrogen content was lower in lower level 
of DCP feeding. SRL-strained Rumen liquor.

IBAR/1982 H.D. CHAPMAN

84 The Treatment of Coccidio-
sis: Studies on the Sen-
sitivity of Recent Field 
Isolates of *Eimeria Acervu-
lina* Type to Anticoc-
cidial Drugs Given in the 
Drinking Water

*J. Comp. Path.*, 1982 92 (2) : 213-
218

AUTHOR'S SUMMARY: Sulphaquinoxaline and sul-
phaquinoxaline plus pyrimethamine were effective 
against the Houghton strain of *E. acervulina*. Ampro-
lium plus ethopabate, alkomide plus sulphanitran 
and sulphaquinoxaline plus diaveridine were partially 
effective but a mixture of nitrofurazone plus fura-
zolidone had little effect.

Sulphaquinoxaline and sulphaquinoxaline plus 
pyrimethamine were only partially effective against 
field isolates obtained from broiler and breeder 
farms throughout the United Kingdom and it was 
concluded, therefore, that resistance had been ac-
brained to them. Amprolium plus ethopabate was also 
partially effective against field isolates but this was 
due to limited drug efficacy.

Differences were found between drugs and their 
effect upon broiler and breeder isolates. It is con-
cluded that sulphaquinoxaline and pyrimethamine 
may be the best treatment available for broiler chic-
kens and that this drug and amprolium plus etho-
pabate may be of use for breeder birds.
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