PATHOLOGY OF THE GENITALIA AND ENDOCRINE GLANDS OF FEMALE GOATS INFECTED WITH TRYPANOSOMA VIVAX

BY

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A THESIS

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CERTIFICATION

This thesis by Felix Apeh G. Lawani meets with the regulations governing the award of the degree of Master of Science of Ahmadu Bello University, and is approved for its contribution to knowledge and literary presentation.

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I hereby declare that this Thesis has been written by me and that it is a record of my own research work. It has not been presented in any previous application for a higher degree. All quotations are specifically acknowledged by means of references.

Felix APEH G. LAWANI

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DEDICATION

This thesis is dedicated to my children ONYI and MIRIAM-OWOJE and to ALMIGHTY GOD the giver of knowledge.

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ABSTRACT

Nineteen pubertous, non-kidded healthy red Sokoto does were experimentally infected with Trypanosoma vivax, while 5 others served as uninfected controls. Four infected does each were euthanised at weeks 4, 6, 8, and 10, and 2 at the 13th week post infection (PI), respectively. The infected does developed fulminating parasitaemia after a mean prepatent period of 4 days. This was accompanied by pyrexia of up to 40.1°C and a loss of 18.37% of pre-infection body weight. Anaemia reflected by a 40% drop in PCV, and leucopenia due to neutropenia and lymphocytosis occurred. Infected animals showed dullness, anorexia, rough haircoat, generalized lymphadenopathy, subcutaneous oedema, epistaxis, palor of the mucous membranes and bilateral ocular and nasal discharges. Gross pathological changes included slight enlargement of the adrenal glands, cystic degeneration of ovarian follicles and lack of corpora lutea (CL) formation, segmental aplasia of the uterine tubes and oedema of the vulva. Histological changes consisted of lymphocytic infiltration of the pituitary capsule, patchy necrosis, cyst formation and thrombosis of blood vessels in the adenohypophysis. There was hypertrophy and necrosis of the adrenal cortex. The ovaries were characterized by a relative decrease in number or complete absence of primordial to secondary follicles and by polycystic formation, fibrosis and infiltration of the ovarian parenchyma by mononuclear cells.

The ovaries of all but one infected animal had no functional CL. Degenerative changes along the tubular genitalia consisted of desquamation of the superficial and glandular epithelium of the uterus, endometritis/metritis, cervicitis and marked vaginitis and vulvitis.

This study provides further histological evidence that reproductive dysfunctions in does during trypanosomiasis could be due to degenerative changes in the pituitary, adrenals, ovaries and the tubular genitalia. It also shows that the severity of these lesions are independent of the duration of infection.

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CHAPTER ONE

LITERATURE REVIEW

1.0 GENERAL INTRODUCTION

There is an acute and growing shortage of calorie and protein for human consumption in almost all developing countries. In order to alleviate this widespread phenomenon, the quantity and quality of protein available to the people has to be increased. The average daily per capita consumption of animal protein in the developing countries has been put at between 11-13g, compared with 49-54.1g in the developed countries. Food and Agriculture Organization (FAO) (1970), Jasiorowski, 1972; FAO (1983) records show that between 1978 to 1980, only 20% of human protein consumption was provided by livestock in these countries, an indication that the situation has not changed.

According to the FAO (1976), 62.5% of the world's livestock resources are located in developing countries. However, the animal protein production from livestock farming per 1000 hectres in Africa is only 542kg compared with the 4,113kg/1000 hectres in Latin America and 38,085kg/1000 hectres in Europe (FAO, 1975). It is evident therefore, that the low level of animal protein production in the developing countries is not as a result of the low number of animals, but their poor productivity. One of the greatest hinderances to livestock productivity in Africa is the problem of diseases. Trypanosomiasis is regarded as one of the most important of all livestock diseases endemic in the African continent (Urquhart, 1974). It is transmitted by tsetse flies of the genus Glossina.

About 37% of Africa comprising of 10 million KM^2 of land cutting across 38 countries is infested with tsetse flies (FAO/WHO/OIE, 1982). In Nigeria, 6 to 7 x 10^5 sq. km or 75% of the entire country is infested with tsetse flies (Federal Ministry of Agriculture, NLA, 1981). Approximately 30% of susceptible livestock in the affected areas are exposed to infection with trypanosomiasis (FAO/WHO/OIE, 1982). The group of monomorphic salivarian trypanosomes is by far the most important to the livestock industry (Leeflang, 1978). This group includes two species Trypanosoma vivax and Trypanosoma congolense. A third important specie is T.brucei. These organisms have differing affinity for host tissues. Trypanosomes of the T.brucei group are capable of traversing capillary endothelium and multiplying outside the blood vascular system. T.vivax and to a lesser extent T.congolense, on the other hand, are intravascular parasites (Losos and Ikede, 1972; Bungener and Muller, 1976; Tizard et al., 1978a). These three parasites cause intrauterine infections, abortion, prolonged anaestrus and testicular degeneration leading to subfertility and infertility in both sexes of livestock (Ikede, 1979; Ogwu and Nuru, 1981; Ogwu, 1983; Ogwu et al., 1984, 1986).

In man, the population at risk from infection with trypanosomiasis or sleeping sickness is said to be over 50 million (De Raadt, 1984; Murray and Gray, 1984),

and this is caused by T.brucei gambiense and/or T.b. rhodesiense. In males, impotence, gynaecomastia and feminine distribution of fat (Apted, 1970; Ikede, 1974) have been observed. In females, the disease is characterized by menstrual disorders, abortion, stillbirths, and neonatal deaths (Apted, 1970; Olowe, 1975). Apart from death, human trypanosomiasis decreases the potential of the rural agricultural populace in terms of human farm labour. This serves to further compound shortages in food production. It has been estimated that if trypanosomiasis is eradicated, Africa could generate US $50 billion annually in livestock and agricultural development in tsetse infested areas (Trail et al., 1985).

Control measures of trypanosomiasis in use are, bush clearing, elimination of wildlife, chemotherapy and chemoprophylaxis, fly vector eradication by the use of insecticides, the sterile insect technique, and the rearing of trypanotolerant breeds of animals. These measures have not succeeded in eradicating the disease, while hopes of vaccination appear hampered by the antigenic variability of trypanosomes.

Although there has been intensive investigation into the pathology and pathogenesis of trypanosomiasis in cattle, reports of the disease in sheep and goats have either been considered unimportant, or of such low incidence as to warrant similar studies (Krammer, 1966; Stephen, 1970; Maclennan 1970).

This is as a result of reports that small ruminants kept in tsetse infested areas seldom suffer from trypanosomiasis (Ilemobade, 1982), and this appears to be due to the fact that sheep and goats are seldom fed upon by tsetse flies (Stephen, 1970; Davies, 1977). Furthermore, there are suggestions that several indigenous breeds of goats, especially those found in tsetse infested areas, are trypanotolerant (Griffin and Allonby, 1979; ILCA, 1979). Inspite of the foregoing, however, evidence abounds indicating that the disease does not only occur naturally in goats, but that high mortality, reproductive dysfunctions and consequent economic losses could result (Edwards et al., 1956a, b,; Anosa and Isoun, 1980a; Kanyari et al., 1983; Kalu and Edeghere, 1985). Not much is known of the pathogenetic mechanisms of reproductive abnormalities observed in infected females. This work was undertaken to study the probable pathogenetic mechanisms associated with reproductive disorders in female animals with special reference to gross and histopathology of the tubular genitalia, the ovary, pituitary and adrenal glands. The female Red Sokoto goat infected with T.vivax was the experimental model.

2.0. ECONOMIC IMPORTANCE OF GOATS

Goats form a part of the livestock industry, and in Nigeria, they represent a very important national resource.

In the face of a rapid rise in human population with no comparable increases in animal protein production, small ruminants, particularly goats can provide some of the much needed animal protein requirements within a relatively short time (Ilemobade, 1982).

Available statistics indicate that about 60% of rural households keep small ruminants, which contribute about 35% of the total national meat supply (David-West, 1985). Federal Ministry of Agriculture (FMA, 1981) estimated that goats contributed about 17% (65,000 tonnes) of the total meat supply. In some parts of the country, e.g. Kaduna, Kano and Gongola States, goats are milked (Ngere et al., 1984). The Nigerian leather derived from the skin of the Red Sokoto goat still enjoys a place of pride in the international market (David-West, 1985). In rural communities, goats serve as insurance against socio-economic and sociocultural demands such as: quick source of cash, preparation of special dishes, in sacrifices and other traditional ceremonies (French, 1970; Sandford, 1972; Ngere et al., 1984).

3.0. POPULATION AND DISTRIBUTION OF GOATS IN NIGERIA

There is a dearth of information regarding the population of goats in Nigeria, as they are almost entirely produced under traditional systems. Finelle (1974), FAO (1976) and Griffin (1978) put the population of goats in tsetse - infested Africa at about 50 million.

Of this number, Nigeria is said to have 33% (16.5 million) of all the goats in Africa (Ngere et al., 1984). However, a current figure of 26 million has been estimated as the population of goats in Nigeria (FAO/WHO/OIE, 1985).

There are 3 main breeds of Nigerian goats. These are the Sahel or 'desert' goat, the Red Sokoto or 'Maradi', and the West African Dwarf (WAD). The Sahel breed is found in the Sahel region of the country, where they are adapted to nomadic life and grazing in Semi-desert environments. Recorded mature weight ranges between 25 and 35 kgs. The Red Sokoto is found mostly in Sokoto state, but has spread eastwards and southwards throughout the Savanna belts and up to the humid forest zones of Nigeria. Mature animals of this breed weigh averagely between 23 and 30 kgs. The WAD is found throughout the humid forest belts of Nigeria. It is variable in size and many transitional types have been recognised as a result of unrestricted crossbreeding. This breed is noted for its hardiness and because it thrives in tsetse-infested humid forest and guinea savanna zones, it is reputed to be trypanotolerant. The mature weight of the commonest type is 20 - 25kg (Ngere et al., 1984).

4.0 REPRODUCTIVE PERFORMANCE OF GOATS

There is inadequate concise information on the reproductive performance of the Nigerian indigenous breeds of goats. This is because most of the population of goats in this country are kept under local traditional systems with no written records. The lack of good records and properly defined conditions at the local level makes it difficult to assess the productivity of the goats in the local village units. Consequently, most of the information available on the productivity of goats are those obtained from government, institutional or university farms.

4.1 Age at Puberty/First Kidding

Puberty is defined as that period of adolescence when a male or female is first able to release gametes (Hafez, 1975). For example, first ovulation or oestrus in the female indicates attainment of puberty. It is much more difficult to determine the exact time of puberty in male because the first differentiation of spermatogonia precedes the release of the spermatozoa from the seminiferous tubules by a month or more, and the sperm transport from testes to vas deferens needs at least 2 weeks (Thibault and Levasseur, 1975). However at puberty, irrespective of sex, secondary sexual characteristics become conspicuous and the essential organs of reproduction begin to undergo great increases in size (Hafez, 1975).

The onset of puberty is related to breed (Wiltbank, 1978) and body weight which depends on the level of nutrition, age, climatic and social factors (Devendra and Burns, 1970; Gill and Dev, 1972; Ali et al., 1973; Oyedipe, 1981) and involves changes at the pituitary level (Hammond and Marshall, 1952).

Age at first kidding is a fertility index and is closely related to the age at puberty. However, there are several factors affecting the doe-kid and causing the age at first kidding to be quite unrelated to the age at puberty. Such factors include early embryonic death, anaestrus and conception failures of various aetiology (Ogwu, 1983).

Available reports indicate that most breeds are puberal between 5 and 10 months of age (Riera, 1982), while the age at first kidding varies between 8 - 29 months with an average of 14.5 months for the Red Sokoto (Ngere et al., 1984), 416 ± 86 days for the Sokoto brown (Molokwu and Igono, 1978) and 10 - 21 months with an average of 18.5 months for the WAD breed (Ngere et al., 1984).

4.2. Services Per Conception

Information in goats related to the number of services per pregnancy is scanty. The few reports found in literature are those obtained from foreign breeds. The Black Bengal goats had 1.12 - 1.14 service per conception compared with 2.3 for the East African goat, while in Brazil, reports on native and exotic breeds showed a variation from 1.40 to 2.06 services

per pregnancy with the exotic Anglo-nubian and Bhoj being the highest with an index of 2.08 (Riera, 1982). Potential differences in management and environmental conditions among studies reported must be considered in comparing these data.

4.3 Kidding Rate

The kidding rate or prolificacy refers to the number of kids per conception and/or parturition. Prolificacy of goats has a very significant influence on reproductive efficiency. This factor and the birth weight together as an index will furnish an estimate of the potential kid production of the female. It is breed related, and except for the tropical and subtropical genotypes, may be influenced by season (Amble et al., 1964; Haumesser, 1975). There is a uniformity of opinion that feed availability is of great significance in determining the kidding rate. Breed related kidding rate varies from 1.01 for the Seanen, to 2.05 for the Black Bengal (Riera, 1982), for the Red Sokoto varies between 1.45 - 1.47 (Haumesser, 1975; Molokwu and Igono, 1978), while that for the WAD is between 1.70 and 1.90 (Kirkpatrick and Akindele, 1974). Although prolificacy is a useful measure of reproductive performance, the number of goats reared to weaning is of greater practical importance in measuring productive or reproductive efficiency.

It has been reported that the live body weight of the dam influences litter size in the goats as it does in sheep, cattle and other species. Does which were heavier at kidding gave higher kidding rates (Adu et al., 1979; Riera, 1982).

4.4. Kidding Interval

This is defined as the period between 2 consecutive kiddings and is composed of the service period (period from kidding to conception) and gestation period (Devendra and Burns, 1970). Kidding interval is an important production factor in goats as well as in most domestic animals because of its effect on efficiency of production and reproduction and on the genetic improvement because of faster population turnover and increased selection pressure. This interval varies with age, breed of doe, parity, level of lactation, kidding rate, season of the year and the level of nutrition (Riera, 1982). Extended kidding intervals may be caused by all major forms of infertility including anaestrus, conception failures and prenatal death (Osori, 1976). It has been demonstrated that does that lost their kids during neonatal period had an average kidding interval of 180 days, while does that raised a kid to at least one month of age had an average interval of 223 days (Riera, 1982). The kidding intervals for the Red Sokoto goat is between 237 $\pm$ 47 and 269 days (Haumesser, 1975; Ngere et al., 1984)

while that for the WAD ranges between 252 - 258 days (Riera, 1982; Ngere et al., 1984). The possibility of 2 kiddings within a period of 12 months has been demonstrated in Barbari does (Riera, 1982), while it has shown that with a kidding interval of 8 months, and with year round breedings, 3 kiddings in 2 years are possible in Nigerian breeds of goats (Ngere et al., 1984). Thus the potential for increasing livestock productivity with Nigerian goats is high, if trypanosomiasis and other factors that affect their productivity are controlled.

4.5 Postpartum Interval to Oestrus

The interval between parturition and the first postpartum oestrus and service period are also important traits which contribute to reproductive efficiency in goats. The earlier postpartum that does return to oestrus the earlier conception can occur, the shorter the kidding interval, and the more the reproductive efficiency. This interval varies among breeds, period of lactation and nutritional regimen (Riera, 1982). The interval for the WAD goat is reported to be between 13 - 62 days with a mean of 37 days (Kirkpatrick and Akindele, 1974; Ngere et al., 1984). This interval may be prolonged by conception failures, still birth and/or abortion, delayed uterine involution and resumption of ovarian cyclical activities, and disease among other factors.

5.0 FACTORS AFFECTING REPRODUCTION IN GOATS

The levels of reproductive efficiency in goats is determined and regulated by different processes which results from interactions between genetic and environmental factors such as climate, nutrition and disease etc.

5.1 Climatic Factors

Different climatic factors such as, temperature and photoperiod regulate physiologic response. Under high temperature conditions, there is a change in the metabolism, which is reflected in prolonged parturition intervals and delayed puberty (Devendra and Burns, 1970). The fluctations of environment are related to the season of the year, and influence food availability and therefore cause a variation in reproduction (Riera, 1982). Reduced variation in photoperiod and perhaps other climatic factors in the equatorial,tropics and subtropics results in extended periods of breeding, allowing continuous, year round sexual activity in the goat (Devendra, 1962; Haumesser, 1975).

. In temperate zones, the goat behaves as a seasonal breeder showing clear anaestrus period due to the influence of changes in photoperiod (Shetlon, 1978).

5.2. Nutrition

Adequate and balanced nutrition is an important prerequisite for the healthy development of both the dam and the foetus. Records at Ibadan and Shika show that, though Nigerian goats breed throughout the year, more conceptions were recorded during the rains when feed is more abundant (Ngere and Mbap, 1982). Recently, a 62.5% abortion rate was observed in a flock of WAD goats in the University of Ibadan farm following the withdrawal of concentrate supplementation to the animals (Osuagwuh and Akpokodje, 1985). Literatures abound relating most reproduction efficiency factors to the availability and level of nutrition (Riera, 1982).

5.3. Diseases

Disease in animals is considered one of the greatest hinderances to increased livestock productivity in Africa (Griffin, 1978). Of all the livestock diseases endemic to the African continent, trypanosomiasis has been regarded as the biggest single factor which limits the numbers and productivity of cattle, sheep and goats (Urquhart, 1974).

6.0 AFRICAN TRYPANOSOMIASES

This is a disease complex caused by parasitic protozoa transmitted by different species of tsetse flies of the genus Glossina. Eleven of these species are known to occur in Nigeria (Davies, 1977; Onyiah, 1980). Each of the species is adapted to different climatic and ecological conditions (Ford, 1971).

They constitute an immense problem because of their ability to remain infective for long periods once they are infected; moreso, all the 11 species are capable of being vectors of trypanosome species infective to human beings and domestic animals.

The most important trypanosomes that cause disease in livestock are T.vivax, T.congolense and T.brucei (Ikede, 1981). The latter is less pathogenic for ruminants than the other 2 species. In pigs, T.simiae is the most virulent (Leach and Roberts, 1965). T.vivax is considered to be the most pathogenic and important trypanosome of ruminants (Glover, 1961; Leach et al., 1965; Agu, 1983) in West Africa, while in East Africa it is T.congolense (Griffin, 1978; Mwambu, 1983). T.evansi and T.equiperdum infect mainly camels and horses respectively and are of economic importance in North Africa. These groups of trypanosomes are non-cyclically transmitted by some biting flies other than Glossina (Scot, 1973)

In man, sleeping sickness is caused by T.brucei gambiense in West and Central Africa and by T.brucei rhodesiense in East Africa.

The severity of the disease in a host may depend on the species and strains of infecting trypanosome (Leach et al., 1981; Morrison et al., 1981).

There are large numbers of different strains in each species of trypanosomes, each capable of elucidating different stocks of variable antigens (Van Meirvenne et al., 1977). The success of the different strains of the parasites is due to the phenomenon of antigenic variation (Cross, 1975). Other factors which may affect the severity of the disease in a host are the breed and/or the genetic make-up of the host, state of nutrition, stress factors such as pregnancy, parturition and inter-current infections (Leach et al., 1965; Morrison et al., 1981).

6.1 Diseases Caused by T.vivax in Ruminants

The pathology and pathogenesis of trypanosomiasis is the subject of many comprehensive monographs and reviews (Losos and Ikede, 1972; Losos and Chouinard, 1979; Ikede, 1982, 1986; Anosa, 1983; Losos, 1986). These authors reported that the course of the disease in ruminants is highly variable, both in severity and duration.

The course of the disease may be acute, subacute or chronic. Animals become infected following a fly-bite with the development of a localized tissue reaction called chancre. A prepatent period, ranging from a few days to weeks occurs before overt signs of illhealth are observed. With a needle challenge, this period is considerably reduced.

The onset of parasitaemia is characterized by pyrexia, anorexia and dullness. The acute disease usually lasts a few days or a few weeks, and terminates either in death or the chronic form of the disease. The fever and parasitaemia then becomes irregular and intermittent. The major feature of the disease is anaemia, manifested by a decrease in packed cell volume (PCV), which may be precipitous or gradual. The decrease in PCV may progress terminally, or become stabilized at a low level for a variable period of time. Enlargement of superficial lymph nodes, emaciation, subcutaneous oedema, rough hair coat, lacrimation and nasal discharges have been observed.

Host and parasite related factors determine the course of the disease (Masake, 1980; Ikede 1981; 1986). The parasite factors include strain variation in virulence. For example, East African strains of T.congolense are regarded as more virulent than those of West Africa. The host factors include variations due to age, sex, breed, nutritional status, degree of challenge, health status, and chemotherapeutic measures.

6.1.1. T.vivax in Cattle

The effect of T.vivax has been extensively reviewed by Losos and Ikede (1972), Anosa (1983) and Losos (1986). The disease in cattle may be acute, subacute or chronic (Stephen, 1970).

At post mortem, the most consistent gross lesions observed are haemorrhages of the gastrointestinal tract, heart, lymph nodes, adipose tissue and the skeletal muscles.

In addition, the bone marrows of euthanized cattle have been found to be icteric (Fiennes, 1953; Masake, 1980).

6.1.2. T.vivax in Sheep and Goats

The disease in these breeds may be subacute, acute or chronic (Losos and Ikede, 1972; Losos, 1986). Anaemia is the main feature of both the natural and experimentally induced infection (Saror, 1980). The degree of anaemia is dependent on the severity of the infection. It is accompanied by pyrexia, palor of the mucous membranes, enlarged lymph nodes, rapid loss of weight and appetite, starring hair coat and nasal discharges (Anosa and Isoun, 1974; Masake, 1980; Anosa, 1983; Losos, 1986).

Gross changes observed at post mortem are oedema of the lymph nodes and haemorrhages of the heart, lungs and the gastro-intestinal tract. Microscopically, there is hemosiderosis in the lymph nodes, spleen, and the liver. Congested capillaries and distension of hepatic sinusoids also do occur (Anosa and Isoun, 1974, 1980b).

7.0. TRYPANOSOMIASIS AND LIVESTOCK REPRODUCTION

Trypanosomiasis in animals cause severe reproductive disorders. Recent reviews by Ogwu and Nuru (1981), Anosa (1983), Ikede et al., (1988) indicate that in female animals, irregular cyclicity, intrauterine infections, foetal death, stillbirth and abortions do occur following infection with trypanosomes, while varying degrees of testicular and epididymal degenerations

leading to poor semen quality and quantity have been described in the male (Isoun and Anosa, 1974; Ikede, 1979; Kaaya and Odour-Okelo, 1980; Agu et al., 1986; Sekoni, 1987).

7.1. Lesions of the Reproductive System of Female Animals:

a. The vulva

Reports of lesions of the vulva following infection with trypanosomes are limited. Transient oedema of the vulva has been observed in heifers 56 days post infection with T.vivax (Ogwu, 1983). Similarly, rabbits infected with T.evansi developed oedema of the vulva which was later followed by severe inflammatory reactions (Patel et al., 1983). Inflammation of the vulva of rabbits infected with T.brucei was also reported by Van den Ingh and Van Dijk (1975).

b. The Vagina

Vaginal changes during trypanosomiasis has received very little attention. Petechial and ecchymotic haemorrhages have been described in the mucosae of the vagina of T.vivax-infected cattle (Vohradsky, 1971; Mwongela et al., 1981).

c. The Uterus

Uterine changes following T.vivax infection in animals have not been reported. However, desquamation of the superficial and glandular epithelium of the uterus of some mice, rats and rabbits at 9, 15 and 45 days post-infection has been observed in T.evansi infections (Patel et al., 1983).

In T.brucei infected cattle, the uterus was found to be oedematous. In addition, there was perivascular cuffing with lymphocytes, plasma cells and eosinophils in the endometrium, and between the circular and longitudinal muscle bundles. Solitary lymph follicles with active germinal centres coupled with diffuse infiltrations by mononuclear cells were also seen beneath the endometrium (Moulton and Solled, 1976).

The pathogenetic mechanisms for these observed lesions were not elucidated. However, the inflammatory reaction could probably be an attempt by the immune system to combat trypanosome antigens as was the case in T.brucei infected rabbits (Van den Ingh and Van Dijk 1975). Consequent upon these lesions, the inflammed uterus may become hostile to the embryo or developing foetus with attempts to expel it (Hafez, 1968).

d. The Uterine Tubes

There is scanty literature on the effects of infection on the uterine tubes. Congestion with focal areas of haemorrhage have been described in the uterine tubes of some rats, mice and rabbits infected with T.evansi (Patel et al., 1983).

Acute or profiferative swellings could result in occlusion of the narrow lumen of the uterine tubes, thereby leading to conception failures. Portions of the ciliated epithelium and/or contractile muscle which propel the ovum to the uterus may be destroyed.

Also, spermatozoa could be destroyed by toxins from the exudate in the uterine tubes (Elhassan, 1987). Sterility may occur in the animal with these lesions (Jones and Hunt, 1983).

e. The Ovaries

The few reports available on the ovaries of infected animals show that cystic degeneration is the most consistent feature. Vohradsky (1971) reported the presence of cystic ovaries in cattle infected with T.vivax. Similarly, numerous cysts were observed on the ovaries of 2 sheep infected with T.vivax. Smears from these cysts were positive for trypanosomes (Isoun and Anosa, 1974). Ogwu (1983) observed the absence of functional structures in the ovaries of all T.vivax infected heifers. More recently, the mean ovarian weights of goats infected with T.congolense were found to be significantly lower than those of uninfected goats and this was associated with quiesence and atrophy. Histologically, there was extensive fibrosis of the ovarian stroma, a decrease in the number of primordial to secondary follicles and absence of corpus luteum. These were in addition to pyknosis of follicular cells, infiltration of ovarian stroma with mononuclear cells, and polycystic formation (Mutayoba et al., 1988a). Disruption of cyclical ovarian activity and persistent luteal tissue within the ovaries of 3 out of 4 goats, and 4 out of 12 cows infected with T.congolense have also been reported (Luckins et al., 1986).

The mechanisms of the trypanosome induced ovarian lesions are poorly understood (Mutayoba et al., 1988a). However, cystic ovaries are usually due to prolonged luteinizing hormone and follicle stimutating hormone secretion which maintain luteal structures and stimulate the follicles respectively (Jones and Hunt, 1983). The persistent luteal structures in infected animals could be either due to a failure in the synthesis and transport of PGF_2oC, or to a lack of responsiveness of the corpus luteum (Luckins et al., 1986).

f. The Pituitary

Varying reactions by the pituitary to infections have been described. Lesions were not seen in sheep and goats infected with T.vivax except for the presence of a few morula cells (Anosa and Isoun, 1983). In contrast, goats infected with T.congolense showed slight atrophy of the pituitary accompanied by partly degranulated basophilic cells with fewer secretory acini, and slight hypertrophy of the acidophils in the cortical zones of the adenophypophysis (Mutayoba et al., 1988a). T.brucei produced severe lesions in infected sheep, goat and horses (Ikede and Losos, 1975). This was manifested by focal to diffuse gliosis varying degrees of infiltration of the perivascular spaces and meninges by lymphocytes, macrophages, plasma cells and occasionally, morula cells in the neurohypophysis.

In addition, oedema, hyperemia, thrombosis of sinuoids, acute and focal coagulative necrosis of the adenohypophysis were also observed. Trypanosomes were found in sizeable numbers in intervascular spaces associated with fibrosis in sheep and goats (Losos and Ikede, 1972; Ikede and Losos, 1972; Ikede, Hill and Akpokodje, 1973; Ikede and Losos, 1975).

The pathogenesis of the observed lesions in the hypophysis is not clear. Jubb and Kennedy (1970) suggested that necrosis may accompany anaemia. Anaemia is known to be a consistent feature of trypanosomiasis in animals (Saror, 1980). In addition, there was thrombosis of the sinusoids in the areas of necrosis, indicating that necrosis could also have resulted from ischaemia (Ikede and Losos, 1975). Necrosis of the hypophysis might also have been due to the invasion of the interstitial tissue by trypanosomes, a process which has been shown to result in extensive tissue damage in the ear of rabbits infected with T.brucei (Goodwin, 1970). The direct effect of trypanosomes on tissues is probably due to their possession of haemotoxic factors (Mutayoba et al., 1988a). Trypanosome - borne serum factors which inhibited myeloid colony formation in calves and lysed red cells have been described by Tizard et al. (1978a) and Kaaya et al. (1980) respectively.

Severe damage of the pituitary might result in decreased production of gonadotropins, and this could lead to gonadal dysfunctions.

g. The Adrenals

There are very few reports on the reaction of adrenal glands in animals infected with T.vivax. Elhassan (1987) observed enlarged adrenals in 1 out of 5 T.vivax-infected pregnant ewes. Marked increases in mean weights have been described in T.brucei infected sheep, and this was associated with marked thickening of the cortices, vacuolation and fatty degeneration of the cells of the zona glomerulosa and fasciculata. In addition, there were foci of subcapsular mononuclear cell reaction (Ikede and Losos, 1975). In T.congolense infection, the cortices in some goats were hypertrophic, while some were atrophic. Other changes observed were focal necrosis of the zona fasciculata accompanied by infiltration with mononuclear cells (Mutayoba et al., 1988a).

Hypertrophy of the adrenal cortex is a common phenomenon in conditions of stress (Ikede and Losos, 1975) such as occurs in infections. This may cause the elaboration of excessive amounts of both mineralocorticoids and glucocorticoids, and may result in upsets in sodium/potassium balances and in the metabolism of protein, fat and carbohydrates.
In pregnant animals, excessive amounts of steroid from the fetal adrenals may precipitate expulsion of the fetus.

7.2 Intrauterine Infection in Ruminants

Transplacental infection has been recorded in different species of animals and man. This has been comprehensively reviewed by Ogwu and Nuru (1981). Pigneur (1932) detected T.vivax in the blood of a prematurely born calf of an infected cow. Inoculation of blood from the calf into a pregnant goat resulted in the birth of an infected premature kid. Subsequently, Dikmans et al. (1957) demonstrated the presence of T.theileri in the stomach contents of an aborted calf. Ikede and Losos (1972) inoculated sheep intravenously with T.vivax and reported the birth of a week lamb 33 days later that was positive for T.vivax. Although Isoun and Anosa (1974) did not report a similar finding, they were able to demonstrate the presence of T.vivax in the amniotic fluid of a pregnant sheep that had been experimentally infected. Termination of pregnancy in T.vivax-infected cows was reported by Nuru (1974); a cow inoculated with blood from the fetuses was parasitaemic. More recently, Ogwu (1983) described the occurrence of intrauterine infection in 1 out of 12 heifers experimentally infected with T.vivax. Similarly, Elhassan (1987) demonstrated infection in 2 aborted fetuses of ewes experimentally infected with T.vivax in the first trimester, and in a lamb born at term from an ewe that was infected in the third trimester of pregnancy.

From the above, and reviews by Nuru (1974), and Ogwu and Nuru (1981), evidence abounds that transplacental transmission of trypanosomes occurs in animals and man. For the fetus to be infected from maternal circulation, trypanosomes must be capable or have a means of passing through tissue barriers (Ogwu and Nuru, 1981). This may possibly involve placental damage, which has been demonstrated by Ogwu (1983) who found histological lesions in the placenta of T.vivax-infected heifers in the third trimester of pregnancy . . . . Trypanosomes are known to possess biologically active substances such as the enzymes - proteases, phospholipases and vascular permeability factors (Tizard et al., 1978b). These enzymes may act together with other proteases on a number of biologically significant substrates such as plasminogen and fibrinogen (Boreham and Facer, 1973). Also, phospholipases, which are products of trypanosomal autolysis, are capable of destroying cell membrane phospholipids. T.vivax and T.congolense lodge preferentially within the microcirculation by attaching to the vascular endothelium (Bungener and Muller, 1976; Banks, 1978). It is therefore easy for them to exert some placental damage in infected pregnant animals.

7.3. Abortion

Abortion is the expulsion before term of a conceptus which is incapable of independent existence (Hubbert, 1971). Kirkby (1963) in reviewing the occurrence and epizootiology of bovine trypanosomiasis on the Shika Agricultural Research Station from 1929, stated that the high rate of abortion observed in 1948 was as a result of the 16% infection rate of the overall survey of the blood of all the animals. During 1958 - 59, 131 (31%) cattle at Shika were infected with T.congolense, and 41 (9.7%) abortions occurred (Kirkby, 1963; Leeflang, 1974). No other abortive agents were found to be associated with the abortions. Leeflang (1974), stated that in 1972, 8 out of 10 cows that aborted and 2 out of 3 cows that aborted in 1973 had trypanosomiasis.

Krampitz (1970) had suggested that cows infected with T.congolense are particularly endangered during pregnancy and a few days before delivery if the infection is acute. In 8 Zebu cows that were infected with mixed T.vivax, T.congolense and T.brucei organisms, abortion was demonstrated in 2 out of 5 cows in the first trimester, and in 3 cows in the third trimester. All the 5 cows died approximately 16 days post-abortion (Murray et al., 1981). Ogwu (1983) similarly observed abortion during the first trimester in one out of 4 heifers infected

with T.vivax, and 3 premature deliveries out of 4 heifers infected in the third trimester. All those infected in the second trimester, had normal deliveries. Earlier, Ikede and Losos (1972) had reported 3 cases of abortion and one case of fetal mumification in ewes infected with T.brucei. Abortions have also been reported in second (11.40%) and third (48.4%) trimester pregnancy in T.evansi-infected buffaloes (Lohr et al., 1986).

The mechanisms underlying trypanosomal abortions may be multifactorial. High temperature, resulting from infections, may cause abortions. Hyperthermia causes morphological abnormalities in eggs, e.g. shrunken cytoplasm, ruptured vitelline membrane or zona pellucida, or it may act directly on the millieu interieur of the uterus (Hafez, 1968). Zakari et al. (1981) have also shown that hyperthermia is detrimental to conception. Another factor may be the effect of the enzyme neuraminidase, which has been shown to be produced and released into the tissues of T.vivax-infected animals (Esievo, 1980). This enzyme has been reported to cause abortion by altering the antigenic makeup of the trophoblast (Muller and Saathoff, 1972) or by interfering with implantation (Gasic and Gasic, 1970). Severe maternal stress factors such as in late stage pregnancy or

during infection may activate the foetal hypothalamo-pituitary-adrenal axis to produce corticosteroids from the foetal adrenal glands (Tilton et al., 1972). These corticosteroids stimulate the production of prostaglandins and oestrogen from the placenta. The prostaglandins initiate abortion by reducing myometrial activity threshold through reduction in concentration of progesterone while oestrogen causes uterine contraction and foetal expulsion (Osburn and Kennedy, 1971; Liggins et al; 1972; Hafez, 1975). T.congolense preferentially attach to the vascular endothelium. Autolysis of these in pregnant animals may cause the elaboration of pharmacologically active materials into, and causing damage to the placenta, which may then result in abortion.

7.4. Pathogenesis

Infection of a host by all species begins with the injection or penetration of metacyclic forms of trypanosomes into the subcutaneous or submucosal tissue. At the site, multiplication may occur and may be associated with a local inflammatory reaction the chancre. This skin reaction occurs in T.congolense, T.vivax, and T.brucei infections, but may be different in their histological appearance, severity, and time of response (Emery et al., 1980;

Emery and Moloo, 1981). Trypanosomes enter the blood from the site of infection either directly or through the lymphatics, and are disseminated throughout the body by the circulating blood, thereby causing parasitaemia. The level of parasitaemia may be a reflection of the virulence of the organism (Losos, 1986).

Pathological lesions produced in a host could be determined by the tissue micro-environment in which the trypanosomes preferentially colonize; thus the 3 principal trypanosomes of ruminants have been classified into haematic and tissue - invading groups (Losos and Ikede, 1972; Banks, 1978). The T.brucei subgroup localizes in the solid tissues, particularly the connective tissue stroma (Losos, 1978) causing extensive degenerative changes with anaemia or blood changes being of secondary importance (Griffin, 1978). T.vivax and T.congolense are classified as being primarily parasites of the capillary, and that they do not usually leave the blood stream (Ssenyonga and Adam, 1975). The latter attaches to the endothelial cells (Bungener and Muller, 1976; Banks, 1978) with resultant vascular changes. However, later reports have shown that T.vivax may invade solid tissues, particularly the heart, causing extensive inflammatory response (Bungener and Mehlitz, 1977), thus raising doubts about the

traditional classification of this organism as a strict plasma parasite. The severity of lesions or disease induced can be influenced by nutritional state, intercurrent infections, and stress (Stephen, 1970; Ikede and Losos, 1975). It may also depend on the species and strain of the parasite, as well as the age, sex and breed of the host (Losos, 1978; Ikede, 1986).

Urquhart (1980) has classified the different hypotheses advanced on the pathogenesis of trypanosomiasis on 3 main factors: anaemia, tissue lesions and immunosuppression.

7.4.1. Anaemia

Anaemia associated with African trypanosomiasis is a consistent and significant feature in humans (Woodruff et al., 1973) and animals (Sadun et al., 1973; Saror, 1980). The exact mechanism of anaemia is however, an area of considerable controversy. Whatever the pathway, the genesis of trypanosome anaemia is multifactorial and includes:

7.4.1.A. Haemolysis:

i. Fever

Karle (1969) observed that red blood cells exposed to temperatures a few degrees above normal for a few hours show increases in osmotic fragility, which enhances haemolysis and induces a shortened lifespan in vivo. A common feature of African

trypanosomiasis is the occurrence of fever, often coinciding with peaks of parasitaemia. It can therefore be deduced that fever in trypanosomiasis contributes to red cell damage and destruction.

ii. Haemolytic Factor

Huan et al. (1975) reported that T.brucei produces a protein of molecular weight 10,000 daltons capable of lysing erythrocytes. During trypanosome lysis, there is the generation of phospholipase-A activity and free fatty acids which can also destroy erythrocytes (Tizard and Holmes, 1976; Tizard et al., 1978a); although these agents cause haemolysis in vitro, they are thought to be unlikely to do so in vivo because serum albumin block their activity. However, Rees (1969) and Anosa and Isoun (1976), have shown that levels of serum albumin decrease as infection progresses in cattle and sheep. It is probable that the low-levels of serum albumin during trypanosomiases may not effectively inhibit the haemolyzing effect of free fatty acids.

iii. Disseminated Intravascular Coagulation

Widespread occurrence of fibrin deposits in the microvasculature of various body organs of trypanosome infected sheep and goats has been observed (Anosa and Isoun, 1983). These fibrin deposits have been shown to distort the shape of erythrocytes producing

shistocytes, which are then liable to lysis or phagocytosis (Wintrobe, 1975). This may account for the observations of marked alterations in erythrocyte morphology, including erythrocyte fragments in T.brucei-infected rabbits and mice, (Jenkins et al., 1980; and Anosa and Kaneko, 1983) and cattle infected with T.congolense (Valli et al., 1978).

iv. Role of mononuclear phagocytic system (MPS)

African animal trypanosomiasis is characterized by an expanded and active MPS, which develops soon after infection and continues throughout the disease (Murray, 1974). It has been suggested that splenomegaly may result in erythrocyte stasis, and cause prolonged contact with the expanded and active MPS, hence increased lysis and erythrophagocytosis (Murray, 1979).

v. Immunologic mechanisms

Amole et al. (1982) demonstrated the presence of immune complexes on the surface of erythrocytes of T.brucei-infected mice. Similar observations have been made in T.congolense-infected calves (Kobayashi et al., 1976), and in T.vivax-infected cattle (Facer et al., 1982). These complexes attach to erythrocyte membranes, render the erythrocytes more susceptible to phagocytosis by macrophages.

vi. Direct trauma to erythrocytes

Trypanosomes may inflict injuries on erythrocytes when parasitaemia is high (Wintrobe, 1975). The coating of erythrocyte surface by trypanosome antigens following autolysis in T.congolense-infected sheep may also reduce erythrocyte lifespan (Mackenzie and GCruikshank, 1973). More recently, Anosa and Kaneko (1983) suggested that the many tiny foci of discontinuity on the surface of erythrocyte membrane at the points of contact between trypanosomes and erythrocytes may render the cells more susceptible to phagocytosis by macrophages. Esievo (1980) demonstrated increased production of neuraminidase in cattle infected with T.vivax, and suggested that this enzyme cleaves away sialic acid from erythrocyte surface, and renders them more susceptible to phagocytosis.

7.4.1. B. Dyshaemopoiesis

In acute infections, there are various degrees of conversion of yellow marrow to red marrow (Anosa and Isoun, 1983). The marrow of goats infected with T.vivax have been shown to have numerous clusters of developing erythroid cells, whereas in control uninfected goats, such cells were less densely packed (Saror, 1980).

However, in subacute to chronic infections, there is depletion of erythroid cells (Losos and Ikede, 1972). Microcytosis (Fiennes, 1970), hypoferremia (Tartour and Idris, 1973) and low plasma-iron turnover rates (Dargie et al., 1979) observed during chronic trypanosomiasis are suggestive of impaired erythropoiesis. It has been estimated that T.congolense-infected cattle lose between 40-55% of their circulatory iron (Dargie et al., 1979). This finding coupled with the build-up of hemosiderin deposits in the spleen and lymphnodes (Saror, 1980; Anosa and Isoun, 1983) suggests defective iron reutilization, and raises the possibility that in long standing infections, the marrow is effectively starved of iron and anaemia may be complicated by dyshaemopoiesis.

7.4.1. C. Haemodilution

Haemodilution has been suggested by many authors as contributing to the pathogenesis of anaemia. Naylor (1971) described a 30% increase in the plasma volumes of cattle infected with T.congolense. Likewise, Anosa and Isoun (1976) concluded that hypervolaemia associated with increased plasma and reduced red cell volumes was a feature of T.vivax infections in sheep and goats.

Earlier, Clarkson (1968) showed that plasma volumes were expanded, but not total blood volumes in T.vivax infection of sheep. There has been no satisfactory explanation as to the exact mechanism of haemodilution, and its role has been questioned by several workers (Jennings et al., 1974; Dargie, 1978; Maxie and Valli, 1978; Dargie et al., 1979). However, haemodilution could contribute to the pathogenesis of anaemia only if the plasma volume increases out of tune with the attendant drop in erythrocyte volume (Maxie and Valli, 1978).

7.4.2. Tissues lesions

The mechanisms involved in tissue injury are open to speculation, but may possibly include direct trauma due to the presence of trypanosomes, effects of by-products of trypanosome metabolism, circulatory disturbances and immunologic reactions.

A. Direct trauma

T.congolense is known to attach to the endothelium of the microvasculature (Bungener and Muller, 1976; Banks, 1978). In highly parasitaemic animals, this may lead to injury of vessel wall, with attendant aggregation of platelets vascular stasis and thus, thrombus formation may occur. Tissue anoxia leading to ischaemic necrosis of dependent parts may be a sequella.

This has been observed in the lungs and spleen (Losos, 1986). Similarly, Ikede and Losos (1975) had earlier described ocular lesions in sheep infected with T.brucei and attributed these lesions to the presence of the parasites in the eyefluids and interstitial tissue. The direct effect of trypanosomes on tissues is probably due to their possession of cytotoxic factors (Mutayoba et al., 1988a).

B. Trypanosome metabolism

The metabolic activity of a parasite might affect its host in 2 ways: by depleting essential nutrients or producing toxic metabolites. In highly parasitaemic animals, especially in T.vivax infections, the carbohydrate turnover is massive. Voorheis (1969) suggested that as a result of the continuous demand for glucose by trypanosomes, animals may exhibit signs resembling diabetes because of exhaustion of its carbohydrate reserve, with the resultant breakdown of liver function.

It has also been observed that T.brucei in mammalian tissues can on occasions, produce more pyruvate than the host's tissues can keep pace with and that the concentration increases with parasitaemia (Goodwin and Guy, 1973). These authors showed that fibroblasts become laden with lipid droplets and cease to produce collagen in infected rabbits.

Tissues in such animals assume a shaggy, rough structure compared with normal animals, and this has been attributed to the high pyruvate concentration in the tissue fluids of the infected animals. It is probable that the same mechanism may come into play during trypanosomiasis, resulting in the chronic wasting of muscles that is often observed in infected animals.

Tyrosine, an important precursor of catecholamines, has been shown to be reduced in trypanosome-infected animals (Stibbs and Seed, 1976), while Goodwin (1970) described the alleviation of shock in protozoal infections by the use of norepinephrine. It is probable that the low level of tyrosine, and thus catecholamines, in the brain of animals suffering from trypanosomiasis, may account for some of the observed neurological syndromes that accompany such infections.

C. Circulatory Disturbances

i. Disseminated intravascular coagulation (DIC):

This is the widespread occurrence of microthrombi in blood vessels. These thrombi are made up of platelets or fibrin or both. Its occurrence has been reported in sheep and goats infected with T.vivax (Anosa and Isoun, 1983). Organization of these thrombi may occur leading to vascular stasis, tissue anoxia, and ischaemic necrosis in such organs as the lung, spleen and testis (Losos, 1986) and the pituitary Ikede and Losos, 1975).

ii. Haemorrhage:

This refers to the extravasation of blood into tissues. It is common in T.vivax infections and has been reported in mice, sheep, goats and cattle (Anosa, 1983; Losos, 1986). This may be consequent upon various vasculopathies, and the thrombocytopaenia associated with disseminated intravascular coagulation.

iii. Oedema:

Oedema of subcutaneous tissues and serous effusions into body cavities has been observed in T.vivax infections (Anosa, 1983; Losos, 1986). This has been associated with the presence of the parasites in the blood vessels (Ikede and Losos, 1975) or to vascular damage due to pharmacologically active substances released by trypanosomes.

D. Immunologic reactions

The presence of a foreign antigen in an immunocompetent host elicits an immunologic response. This reaction which at the onset is beneficial to the host, may have adverse effects when it is sustained and intensified as is the case in trypanosomiasis due to the antigenic variation shown by trypanosomes. Antigen - antibody reactions cause the release of kinins, histamines and other pharmacologically active substances (Goodwin, 1974) which results in increased vascular permeability, swollen blood vessels etc.

The resultant oedema and extravasation of parasites and leucocytes may among other effects, cause the separation of myocardial fibres. This affects the conduction of impulses within the heart. Blindness may also result from too much pressure due to fluid accumulation in the chambers of the eye (Ikede and Losos, 1975). Damage to blood vessels may lead to fibrinous exudation into such organs as the lungs. This has been observed in T.vivax-infected animals (Masake, 1980; Anosa, 1983).

Immune complex deposits have been demonstrated in the renal glomeruli of infected animals (Nagle et al., 1974; Murray, 1974). These deposits have been associated with renal insufficiency in T.brucei infected rabbits (Goodwin and Guy, 1973). Immune complex deposits are liable to phagocytosis (Goodwin, 1971; Losos and Ikede, 1972). During these processes, there may be leakages of enzymes from the macrophages into the surrounding which might be cytolytic to tissue cells (Morrison et al., 1978).

7.4.3. Immunosuppression

African trypanosomiasis is associated with the depression of immune response to homologous or heterologous antigens.

Urquhart et al. (1973) and Corsini et al. (1977) hypothesized that in infected hosts, trypanosomal mitogen causes unregulated proliferation and IgM synthesis of B-cell clones, and that clonal exhaustion eventually results in the suppression of B-cell function. Mansfield (1978) and Jayawardena and Waksman (1977) suggested that immunosuppresion is mediated by the suppression or loss of suppressor T-helper cell function, with B-cell clones failing to switch from IgM to IgG synthesis. This implies that B-cell clones would be polyclonally stimulated to produce IgM to whatever antigenic determinants the clones were genetically committed to respond to.

Soluble suppressor substances produced by splenic macrophages may suppress other macrophage activity. As a result, the macrophages cannot adequately process and present antigens to lymphocytes and cannot adequately phagocytose in cell-mediated immunity (Wellhausen and Mansfield, 1979).

Complement is an important plasma constituent which effects the destruction of pathogens in combination with antigen-antibody complex. Its low level depresses the capacity of animal in warding-off pathogens, and also impairs some physiological functions such as changes in vascular permeability and smooth muscle activity (Elhassan, 1987).

Hypocomplementaemia has been reported in trypanosomiasis and this has been attributed to consumption by immune complexes (Murray et al., 1974; Nagle et al., 1974), and activation by trypanosomal glycoproteins (Musoke and Barbet, 1977) and proteases (Nielsen and Sheppard, 1977; Nielsen et al., 1977).

Immunosuppression has also been associated with the phenomena of antigenic variation and diversity in trypanosomiasis (Doyle, 1977). These mechanisms enable the parasites to evade the immune system of the host. The infected host continues to produce antibodies to the different trypanosome antigens. When this is sustained the capacity of the immune system wanes with the animal being unable to cope with other infections.

CHAPTER TWO

MATERIALS AND METHODS

1.0 Experimental Animals:

Twenty four (24) non-kidded Red Sokoto does aged between 8 and 24 months were purchased from Yan-awaki market in Kano State. The animals, which weighed between 10-17 kilograms at the time of purchase, were housed in flyproof pens with concrete floors. They were fed on rice hay, supplemented with commercial sheep and goat ration (Livestock Feeds Ltd, Kaduna) and given commercial mineralized salt licks and water ad libitum. During an acclimatization period of 12 weeks, they were allowed 2 - 3 hours daily exercise for 5 days in a week. All the animals were washed with the acaricide - Asuntol(R) (Bayer, Leverkusen, Germany) monthly, and treated with 2 doses each of Terramycin long-acting (Pfizer-Ikeja, Lagos) injection at a dose rate of 1.0ml per 10kg body weight at one week interval. They were also dewormed at 21 days intervals with Panacur(R) (Hoechst), and prophylactically treated with a dose each of a 7% solution of Berenil(R) (Hoechst) at 3.5mg/kg body weight.

Prior to these treatment, the animals were bled and their blood examined microscopically using a combination of haematocrit centrifuge technique (HCT) (Woo, 1970), the buffy coat method (BCM) (Murray, 1977) for trypanosomes, and giemsa-stained thin blood smears for other haemoprotozoan parasites.

Faecal examinations were also carried out to determine gastrointestinal parasitism. None of the animals was positive for trypanosomes. Daily rectal temperatures and weekly body weights were also taken. A weekly baseline haemogram was established for the animals during the acclimatization period.

2.0. Trypanosomes:

Trypanosoma vivax used to infect the animals was isolated from cattle in Fadan-Kamantan in Kachia Local Government Area of Kaduna State in 1984 by inoculation into a goat. Six intergoat passages through tsetse flies were carried out between 1985 and 1987. Thereafter, the parasite was frozen in liquid nitrogen. A vial of this stabilate was thawed and the parasites examined microscopically for viability, and inoculated into 2 goats which developed clinical trypanosomiasis with fulminating parasitaemia. Blood from these goats was used to inoculate the experimental goats.

3.0. Temperature and Body Weights:

Rectal temperatures were taken daily throughout the duration of the experiment between 8.00am and 9.30 a.m. using a centigrade thermometer.

A Salter$^{(R)}$ model-250 top-loading weighing scale was used for recording weekly body weight during the pre-infection period, and twice weekly post-infection (PI).

4.0. Haematology:

(i) Blood collection: The animals were bled weekly by jugular venipuncture using a 20 guage needle attached to a 5ml syringe. 2ml of blood was collected into labelled commercial tubes with ethylene diamine tetracetate (EDTA) as anticoagulant for haematological studies.

(ii) Haematological indices: The haematological indices monitored were packed cell volume (PCV), red blood cell counts (RBC), white blood cell total counts (WBC) and WBC differentials. PCV was estimated by the microhaematocrit method using the Hawksley microhaematocrit centrifuge at 1200g for 5 minutes. RBC and WBC totals were estimated using the improved Neubauer haemocytometer. WBC differential counts were done by the examination of Giemsa-stained thin peripheral blood smears and differentiating 100 leucocytes.

5.0. Infection of Does with Trypanosoma vivax:

19 does were each inoculated with 2.5×10^6 parasites intravenously (IV) using the jugular vein, while 5 served as uninfected controls.

6.0. Estimation of Parasitaemia:

Parasitaemia was estimated daily by the buffy coat technique (Murray, 1977).

The degree of parasitaemia was classified as follows:

Number of Trypanosomes	Score
1 - 10 per field	1+
11 - 50 per field	2+
51 - 100 per field	3+
> 100 per field	4+

The scores were then converted to logarithm equivalent values (LEV) as described by Walker (1969) and all the values for the animals were pooled together every week.

7.0. Clinical Examination:

All does were observed daily, and thorough clinical examination carried out weekly for signs of the trypanosomiasis.

8.0. Pathology:

Four infected does each were euthanized at the 4th, 6th, 8th and 10th weeks post-infection (PI). However, animals that were recumbent for more than 48 hours were also euthanized. The remaining 2 infected does were euthanized at the termination of the experiment 13 weeks PI. Animals in the control group were also euthanized at varying intervals for comparative purpose. Each of the euthanized does was subjected to thorough necropsy examination for gross and microscopic lesions. Tissue sections measuring 2.5. - 3.0 mm in thickness were taken from the vulva, vagina, cervix,uterus and the uterine tubes, while the ovaries, adrenals and the pituitary glands were preserved whole in freshly prepared 10% buffered formol saline solution in plastic containers.

After 48 hours, the tissues were cut into sections not more than 2.0 mm thick, properly labelled, and processed by a modification of the method described by Drury and Wallington, (1967) using an automatic (Shandon 2L Processor MK II) tissue processor thus:

1. 50% alcohol - 1 hour
2. 70% alcohol - 1 hour
3. 90% alcohol 1½ hours
4. Absolute alcohol I & II - 40 minutes each
5. Absolute alcohol III - 2 hours
6. Xylene I - 40 minutes
7. Xylene II - 40 minutes
8. Wax bath I - 1½ hours
9. Wax bath II - 1½ hours
10. Wax bath III in vacuum oven for 1 hour.
11. The blocks were then embedded in molten paraffin wax of melting point 54 - 56°C. These were then allowed to harden at room temperature, and then refridgerated until sectioning. Sections 4-6 um thick were cut using a Leitz rotary microtome, and stained with haematoxylin and eosin (HE). Prepared slides were examined using a Leitz Laborux-K research microscope.

9.0. Statistical Analysis:

The students t test was used to analyse the data obtained, and values of P less than 0.05 were considered significant. The relationship between parasitaemia and temperature and haematological indices were analysed with the coefficient of correlation.

CHAPTER THREE

RESULTS

1.0 Temperature:

Following the inoculation of the does with T.vivax, all the infected animals had pyrexia of 39.03°C - 40.1°C with a mean of 39.5°C being observed within the first week. Thereafter, the temperatures fell within normal range, but rose again at the 6th, 8th and 12th weeks PI (Table 1, figure 1). The control animals maintained normal body temperatures(38.60 ±0.32)throughout the duration of the infection. The mean weekly temperature values of the infected animals were significantly higher ($P < 0.05$) than those of the control group. There was no significant relationship ($P > 0.05$) between body temperature increase and the duration of infection.

2.0 Parasitaemia:

Parasites were detectable in the blood in one to 7 days after inoculation in the animals with a mean prepatent period of 4 days. The highest mean level of parasitaemia occurred in the 2nd and 3rd weeks PI (Table 2). Thereafter, there was a slight fall with the mean weekly levels remaining fairly high throughout the duration of the experiment (figure 1). Analysis of the coeffiient of correlation did not show any significant

TABLE 1: Mean temperature (^{o}C) values of Red Sokoto does infected with T.vivax and their controls.

Duration of Infection (weeks)	Infected (± SD)	Control (± SD)
-3	39.13 ± 0.34	39.14 ± 0.34
-2	38.89 ± 0.34	38.87 ± 0.33
-1	38.91 ± 0.41	38.85 ± 0.40
0	39.12 ± 0.67	39.07 ± 0.62
1	39.53 ± 0.28	38.39 ± 0.27
2	38.87 ± 0.22	38.35 ± 0.22
3	38.56 ± 0.36	38.17 ± 0.29
4	38.70 ± 0.37	38.14 ± 0.21
5	38.65 ± 0.27	38.12 ± 0.21
6	39.00 ± 0.28	38.36 ± 0.32
7	39.07 ± 0.35	38.90 ± 0.65
8	39.07 ± 0.56	38.80 ± 0.61
9	38.81 ± 0.27	38.52 ± 0.10
10	38.80 ± 0.14	38.34 ± 0.39
11	38.86 ± 0.26	38.68 ± 0.13
12	39.14 ± 0.10	38.54 ± 0.10
13	38.67 ± 0.04	38.51 ± 0.33

TABLE 2: Mean parasitaemia (LEV) values of Red Sokoto does infected with T.vivax.

Duration of Infection (weeks)	Parasitaemia (± SD) (LEV)
-3	-
-2	-
-1	-
0	-
1	1.36 ± 0.70
2	3.25 ± 0.71
3	3.02 ± 0.41
4	2.07 ± 0.70
5	1.51 ± 0.60
6	2.12 ± 0.36
7	1.96 ± 0.52
8	2.15 ± 0.87
9	1.64 ± 0.56
10	1.22 ± 0.59
11	1.45 ± 0.76
12	1.01 ± 0.41
13	1.78 ± 0.0

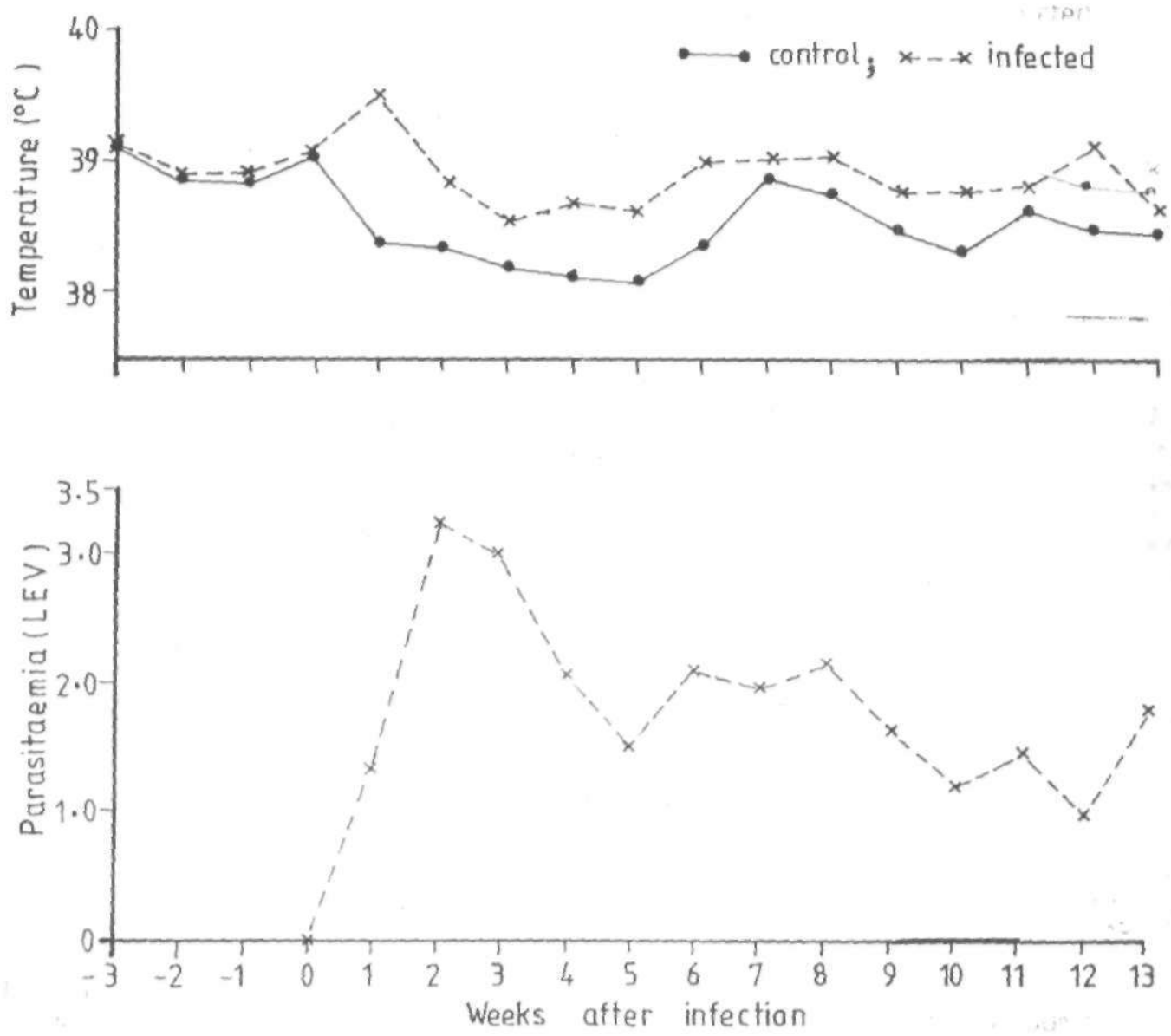


FIG. 1. TEMPERATURE AND PARASITAEMIA CURVES OF RED SOKOTO DOES INFECTED WITH *T. vivax* AND THEIR CONTROLS

relationship ($P > 0.05$) between parasitaemia and temperature, or the haematological indices.

3.0 Body weight:

A gradual decrease in mean body weight was observed in the infected animals from the 1st to the 8th week PI (Table 3). This was reflected by a 9.4% drop from the mean pre-infection body weight value by the 8th week PI. There was some weight gain in the surviving animals between the 8th and 10th week PI. Thereafter, a sharp drop with a 18.37% loss of the mean pre-infection weight value occurred at the termination of the experiment at the 13th week PI. In contrast, the control animals gained weight until the 7th week PI, with a 11.37% increase in the mean pre-infection body weight between the 7th and 9th weeks PI, the remaining control animals lost weight up to 2.09% of their mean pre-infection weight value. Thereafter, they gradually regained weight, but this did not attain the mean pre-infection value before the termination of the experiment (figure 2). The mean weekly weight values of the control animals were significantly higher ($P < 0.05$) than those of the infected animals.

4.0 Haematology:

(a) PCV

Following the onset of parasitaemia, all the infected animals developed anaemia with a drop in the PCV values which reached the lowest level of 0.18 ± 0.06 litre per litre by the 7th week PI. This reflected a 40% drop from the mean pre-infection value (Table 4). The drop became less pronounced thereafter, but the PCV values was still far below the mean pre-infection values at the termination of the experiment (figure 2). The values for the control animals were within normal range (0.26 - 0.32) throughout the experiment, and these were significantly higher ($P < 0.05$) than those of the infected group.

(b) RBC

There was a decrease in the mean RBC values of the infected animals from the 2nd to the 8th week PI, with a 25.73% drop below the mean pre-infection value (Table 5). Recovery occurred between the 8th and 10th weeks PI, when values nearly attained the pre-infection levels. However the RBC values began to drop again until the termination of the experiment (Figure 2). The mean RBC values of the control animals remained within normal range, and were significantly higher ($P < 0.05$) than those of the infected.

(c) WBC

Total WBC fell in the infected animals between the 1st and 7th weeks PI. There was a marked fall

within the first 2 weeks with a 37.94% drop at week 1 PI, to 43.04% drop at week 2 PI, Leucocyte counts rose after the 7th week PI until the termination of the experiment; the highest increase (a 21.66% rise above the mean pre-infection values) was obtained at the 10th week PI (Table 6, figure 3). A significant ($P < 0.05$) difference in the mean total WBC between the infected and the control animals occurred between the 1st and 9th weeks PI, and between the 10th and 13th weeks, PI, respectively.

(d) Leucocyte Differential Counts:

(i) Neutrophil count:

Mean absolute neutrophil counts of the infected animals fell within the first 2 weeks of infection with a 36.9% and 36.6% drop below the mean pre-infection level at the 1st and 2nd weeks PI, respectively (Table 7). This was followed by a rise which attained the pre-infection mean at the 4th week PI. Thereafter, the values fell again below the pre-infection level until the termination of the experiment (Figure 3). The mean absolute neutrophil counts were significantly lower ($P < 0.001$) in the infected group compared with the controls.

(ii) Lymphocyte count:

A drastic fall in the mean absolute lymphocyte counts of the infected animals occurred within the first 2 weeks of infection with a 67.4% drop below the pre-infection mean at the 2nd week PI (Table 8). Thereafter, the values rose and surpassed the pre-infection mean at the 7th week PI, and continued until the experiment was terminated. Significant differences ($P < 0.05$) occurred between the values of the infected and control animals between the 1st and 3rd week PI, and between the 10th and 13th week PI respectively (Figure 3).

(iii) Monocyte, Eosinophil and Basophil counts:

These were no significant differences ($P > 0.05$) in the absolute values of these cells between the infected and control animals (Tables 9, 10, 11 and Figure 3).

TABLE 3: Mean Weight (kg) values of Red Sokoto does infected with T.vivax and their controls.

Duration of Infection (weeks)	Infected (± SD)	Control (± SD)
-3	13.38 ± 2.88	13.69 ± 2.82
-2	14.62 ± 2.77	14.65 ± 2.68
-1	14.24 ± 3.09	14.43 ± 2.97
0	14.35 ± 3.01	14.54 ± 2.90
1	14.05 ± 3.09	14.96 ± 2.71
2	13.88 ± 3.35	15.14 ± 2.92
3	13.92 ± 2.97	15.44 ± 3.11
4	13.26 ± 3.02	15.50 ± 2.91
5	13.07 ± 3.04	15.56 ± 2.93
6	13.03 ± 2.96	15.58 ± 2.93
7	12.98 ± 1.97	15.96 ± 2.75
8	12.82 ± 2.04	15.20 ± 3.60
9	13.35 ± 2.59	14.03 ± 2.80
10	13.29 ± 1.98	14.47 ± 2.87
11	12.85 ± 0.35	14.20 ± 2.26
12	11.95 ± 0.92	14.20 ± 2.26
13	11.55 ± 0.78	14.25 ± 2.05

TABLE 4: Mean PCV (Litre/litre) values of Red Sokoto does infected with T.vivax and their controls.

Duration of Infection (weeks)	Infected (± SD)	Control (± SD)
-3	0.29 ± 0.03	0.26 ± 0.04
-2	0.29 ± 0.04	0.28 ± 0.05
-1	0.29 ± 0.03	0.27 ± 0.04
0	0.32 ± 0.04	0.31 ± 0.05
1	0.29 ± 0.05	0.31 ± 0.04
2	0.24 ± 0.05	0.32 ± 0.03
3	0.21 ± 0.05	0.32 ± 0.04
4	0.20 ± 0.04	0.32 ± 0.03
5	0.20 ± 0.04	0.30 ± 0.03
6	0.20 ± 0.05	0.29 ± 0.03
7	0.18 ± 0.06	0.30 ± 0.03
8	0.21 ± 0.04	0.31 ± 0.04
9	0.22 ± 0.04	0.31 ± 0.05
10	0.22 ± 0.02	0.31 ± 0.03
11	0.27 ± 0.01	0.32 ± 0.06
12	0.22 ± 0.03	0.31 ± 0.00
13	0.22 ± 0.07	0.27 ± 0.06

TABLE 5: Mean RBC ($X10^6$/ul) values of Red Sokoto does infected with T.vivax and their controls.

Duration of Infection (weeks)	Infected (± SD)	Control (± SD)
-3	13.31 ± 2.10	13.37 ± 2.33
-2	12.91 ± 3.11	13.03 ± 3.14
-1	13.02 ± 2.31	12.97 ± 2.10
0	13.42 ± 2.19	13.43 ± 2.00
1	14.19 ± 1.56	13.42 ± 1.88
2	12.06 ± 2.40	15.30 ± 0.57
3	11.38 ± 2.88	16.52 ± 1.51
4	11.05 ± 21.86	15.64 ± 1.40
5	10.63 ± 3.22	15.04 ± 2.84
6	10.69 ± 2.67	15.60 ± 3.45
7	9.93 ± 2.30	15.94 ± 4.13
8	9.87 ± 2.18	16.90 ± 2.84
9	11.67 ± 1.59	15.83 ± 1.82
10	13.27 ± 2.58	15.35 ± 3.18
11	12.00 ± 3.11	14.70 ± 1.41
12	10.60 ± 0.57	15.60 ± 0.42
13	12.44 ± 1.13	15.80 ± 0.85

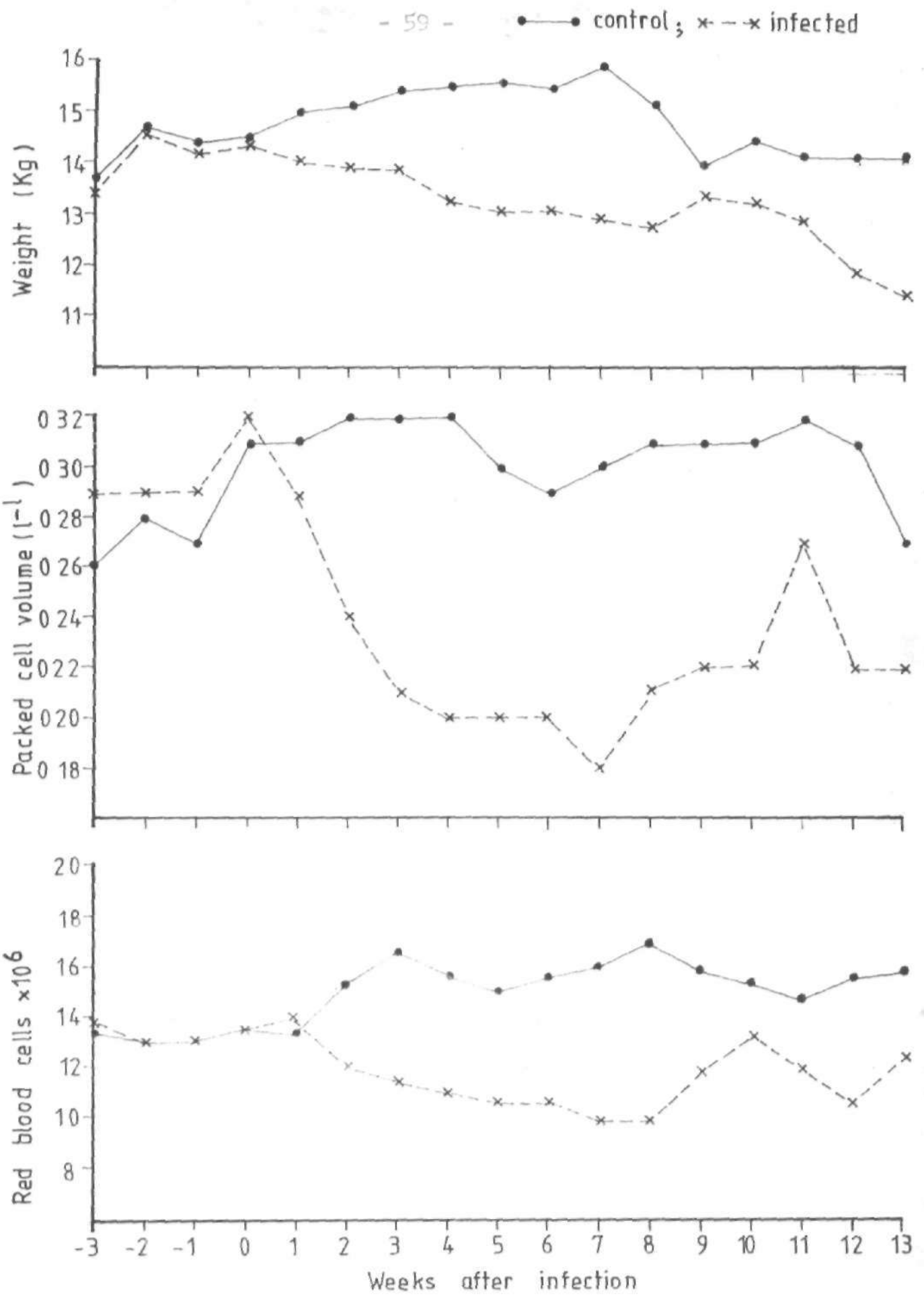


FIG. 2. BODY WEIGHT PACKED CELL VOLUME AND RED BLOOD CELL CURVES OF RED SOKOTO DOES INFECTED WITH *T. vivax* AND THEIR CONTROLS.

TABLE 6: Mean WBC total (X 10^3/ul) values of Red Sokoto does infected with T.vivax and their controls.

Duration of Infection (weeks)	Infected (± SD)	Control (± SD)
-3	10.29 ± 3.04	10.78 ± 3.54
-2	11.59 ± 2.50	11.66 ± 2.33
-1	11.43 ± 3.40	11.68 ± 3.60
0	10.63 ± 2.97	10.68 ± 2.92
1	6.82 ± 4.21	10.54 ± 2.59
2	6.26 ± 3.28	14.04 ± 3.69
3	10.06 ± 3.89	14.17 ± 3.64
4	9.95 ± 4.05	12.26 ± 3.41
5	9.97 ± 4.05	13.75 ± 3.82
6	10.01 ± 3.16	13.43 ± 4.26
7	10.33 ± 2.32	12.00 ± 4.11
8	11.86 ± 2.33	14.37 ± 1.10
9	11.01 ± 2.67	14.13 ± 0.78
10	13.37 ± 5.58	9.63 ± 4.09
11	12.40 ± 6.51	7.55 ± 1.40
12	12.40 ± 8.20	8.65 ± 5.30
13	13.00 ± 3.39	9.20 ± 0.85

TABLE 7: Mean neutrophil (x 10^3/ul) values of Red Sokoto does infected with T.vivax and their controls.

Duration of Infection (weeks)	Infected (± SD)	Control (± SD)
-3	3.76 ± 1.07	3.19 ± 0.69
-2	3.96 ± 1.07	4.17 ± 1.18
-1	3.87 ± 0.97	3.88 ± 1.05
0	3.47 ± 1.16	4.34 ± 1.97
1	1.39 ± 0.91	3.52 ± 0.35
2	1.38 ± 0.48	4.18 ± 1.97
3	3.21 ± 1.07	4.73 ± 1.98
4	3.97 ± 1.34	5.64 ± 0.59
5	3.43 ± 1.17	4.34 ± 1.62
6	2.87 ± 0.98	5.21 ± 1.15
7	2.04 ± 0.53	4.97 ± 1.06
8	2.27 ± 0.73	3.88 ± 1.12
9	2.33 ± 1.10	4.33 ± 0.83
10	2.18 ± 0.54	2.82 ± 0.28
11	3.02 ± 0.71	3.25 ± 0.13
12	1.92 ± 0.44	2.72 ± 0.06
13	2.73 ± 0	2.90 ± 0.07

TABLE 8: Mean lymphocyte ($X10^3$/ul) values of Red Sokoto does infected with T.vivax and their controls.

Duration of Infection (weeks)	Infected (± SD)	Control (± SD)
-3	6.13 ± 1.19	7.42 ± 0.65
-2	7.33 ± 1.08	6.97 ± 1.33
-1	7.19 ± 0.86	7.15 ± 1.27
0	6.77 ± 1.20	6.13 ± 1.81
1	5.32 ± 0.91	6.13 ± 0.28
2	4.62 ± 0.51	9.10 ± 2.00
3	6.66 ± 1.12	9.16 ± 2.12
4	5.57 ± 1.44	6.15 ± 0.76
5	6.34 ± 1.22	8.58 ± 1.72
6	6.74 ± 0.84	7.41 ± 0.77
7	7.92 ± 0.52	6.62 ± 0.99
8	8.99 ± 0.88	9.92 ± 1.18
9	8.37 ± 1.25	9.51 ± 0.86
10	10.85 ± 0.44	6.77 ± 0.22
11	8.87 ± 0.97	5.83 ± 0.22
12	9.86 ± 0.79	5.93 ± 0.06
13	10.21 ± 0.09	6.26 ± 0.13

TABLE 9: Mean monocyte ($X10^3$/ul) values of Red Sokoto does infected with T.vivax and their controls.

Duration of Infection (Weeks)	Infected (±SD)	Control (±SD)
-3	0.17 ± 0.53	0
-2	0	0
-1	0.01 ± 0.05	0
0	0	0
1	0	0
2	0.09 ± 0.21	0.14 ± 0.10
3	0.11 ± 0.16	0.11 ± 0.25
4	0.22 ± 0.22	0.29 ± 0.14
5	0.18 ± 0.22	0.66 ± 0.47
6	0.11 ± 0.13	0.30 ± 0.31
7	0.07 ± 0.08	0.21 ± 0.09
8	0.88 ± 0.10	0.34 ± 0.46
9	1.25 ± 0	0.10 ± 0.17
10	0.44 ± 0.20	0
11	0.97 ± 0.37	0.05 ± 0.07
12	0.79 ± 0.31	0
13	0.09 ± 0	0

TABLE 10 : Mean eosinophil ($X10^3$/ul) values of Red Sokoto does infected with T.vivax and their controls.

Duration of Infection (Weeks)	Infected (± SD)	Control (± SD)
-3	0.32 ± 0.32	0.47 ± 0.43
-2	0.28 ± 0.32	0.56 ± 0.41
-1	0.26 ± 0.03	0.59 ± 0.45
0	0.36 ± 0.47	0.19 ± 0.22
1	0.12 ± 0.11	0.21 ± 0.27
2	0.06 ± 0.11	0.31 ± 0.49
3	0.04 ± 0.07	0.14 ± 1.17
4	0.17 ± 0.25	0.17 ± 0.20
5	0.07 ± 0.13	0.06 ± 0.08
6	0.29 ± 0.30	0.51 ± 0.41
7	0.30 ± 0.34	0.29 ± 0.16
8	0.47 ± 0.53	0.24 ± 0.30
9	0.31 ± 0.29	0.19 ± 0.08
10	0.13 ± 0.16	0.03 ± 0.06
11	0.19 ± 0.09	0.43 ± 0.07
12	0.31 ± 0.44	0
13	0.07 ± 0.09	0.05 ± 0.07

TABLE 11 : Mean basophil ($X10^3$/ul) values of Red Sokoto does infected with T.vivax and their controls.

Duration of Infection (Weeks)	Infected (± SD)	Control (± SD)
-3	0.05 ± 0.02	0.13 ± 0.14
-2	0.01 ± 0.04	0
-1	0.02 ± 0.06	0.12 ± 0
0	0	0
1	0	0
2	0.01 ± 0.02	0.03 ± 0.07
3	0	0
4	0	0
5	0	0
6	0	0
7	0	0
8	0.02 ± 0.05	0
9	0	0
10	0	0
11	0	0
12	0	0
13	0	0

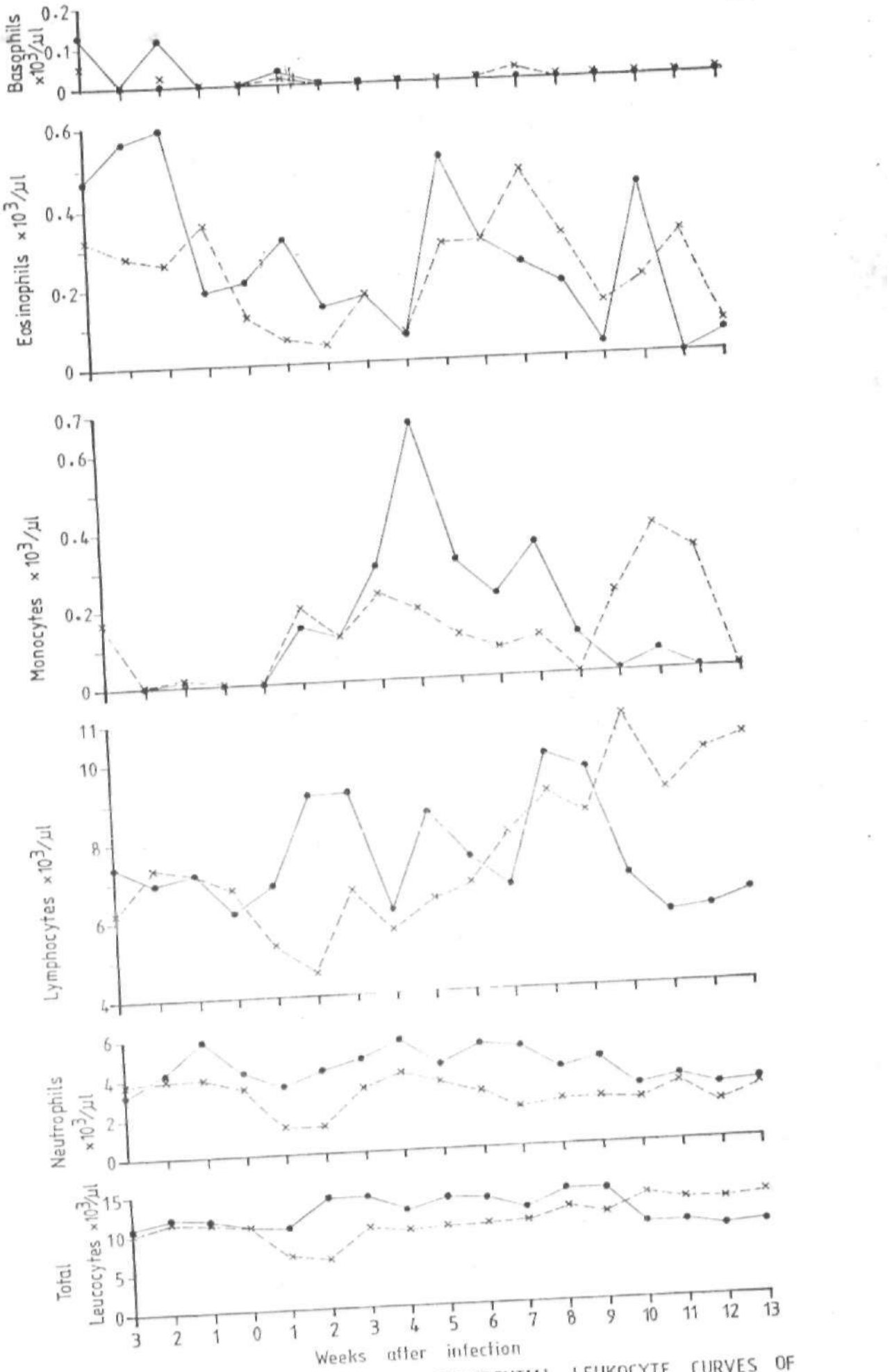


FIG. 3. TOTAL LEUKOCYTE AND DIFFERENTIAL LEUKOCYTE CURVES OF RED SOKOTO DOES INFECTED WITH *T. vivax* AND THEIR CONTROLS.

5.0. Clinical Signs:

The signs exhibited by the infected animals are summarized in Table 12. Following the onset of parasitaemia, the animals became dull, anorexic and had palor of the mucous membranes. Later, emaciation set in with the hair coats of most of the infected animals assuming a rough appearance. Enlargement of the peripheral lymph nodes, unilateral or bilateral ocular and nasal discharges which were catarrhal to mucoid in nature were also observed. One infected animal (No. 234) had epistaxis for over 72 hours, while 2 others (Nos. 202 & 216) had submandibular oedema. All the control animals were normal except for 2 which exhibited signs of "Kata" between the 7th and 9th weeks of the experiment.

6.0. Gross Pathology:

A summary of the necropsy findings of both infected and control animals are given in Table 13.

(a) The pituitary gland:

Gross lesions were not discernible in the pituitary gland of both the control and infected animals.

(b) The adrenal gland:

Enlargement of the adrenal glands of 2 infected animals slaughtered at the 6th and 13th week PI respectively were observed. The adrenals of the control animals appeared normal.

TABLE 12: Signs exhibited by Red Sokoto does infected with *T.vivax*.

	Signs	No. of Infected Animals = 19 No. Affected	No. of Control Animals = 5 No. Affected	Comments
1.	Pallor of the mucous membranes	19	1	*= Animals suspected to be suffering from "Kata"
2.	Emaciation/cachexia	17	2*	
3.	Anorexia	8	0	
4.	Starry hair coat	8	0	
5.	Ocular/nasal discharges	15	2*	
6.	Enlarged peripheral lymphnodes	17	0	
7.	Submandibular/subcutaneous oedema	2	0	
8.	Coughing	0	2*	
9.	Respiratory distress	1	2*	
10.	Diarrhoea	0	2*	
11.	Epistaxis	1	0	

(c) Ovaries:

The ovaries of 2 goats, slaughtered at the 10th and 13th week PI respectively appeared atrophied. In addition, cystic degeneration of the ovaries occurred in 5 of the infected animals including 2 slaughtered at the 4th week PI and one each at the 6th, 10th and 13th week PI respectively. Of all the infected animals, only one had a functional corpus luteum. All control animals had normal ovaries with variable numbers of follicles and apparently functional corpora lutea (CL).

(d) Tubular genitalia:

Two out of 4 goats slaughtered at the 10th week PI had segmental aplasia of the left uterine tube. The tubular genitalia of the rest of the infected animals and those of the control appeared normal.

TABLE 13: Summary of gross pathological findings at euthanasia in Red Sokoto does experimentally infected with T.vivax.

Weeks Post Infection	4	6	8	10	13	Total	Control
No. of Animals in the Group	4	5	4	4	2	19	
EXTERNAL APPEARANCE							
. Starry haircoat	2	3	1	0	0	6	0
. Emaciation/cachexia	2	4	1	2	0	9	1
. Pale mucous membrane	4	5	2	0	1	12	0
. Ocular/nasal discharge	0	1	0	0	0	1	1
. Soiled hindquarters	0	1	0	0	0	1	2
SUBCUTIS							
. Oedema							
. Blood-tinged	1	2	2	0	0	5	0
. Serous atrophy of fat	1	5	2	2	2	12	1
SEROUS CAVITIES							
. Hydrothorax (5.0mls)	0	2	0	0	0	2	0
. Hydropericardium	2	2	3	0	1	7	1
. Ascites	0	0	0	0	1	1	0
. Adhesions	0	1	2	0	0	3	0
GASTRO INTESTINAL TRACT							
. Ulcerations	0	1	0	1	0	2	2
. Haemorrhages	3	3	0	1	1	8	1
.Sloughing of mucosa	0	0	0	1	0	1	0

TABLE 13 (Continued)

LIVER							
. Enlarged	1	2	4	2	0	9	1
. Firm, friable or gritty	0	2	2	2	0	6	0
. Hyperaemia/congestion	1	2	2	1	0	6	0
RESPIRATORY SYSTEM							
A. TRACHEA/BRONCHI							
. Froth-filled	3	2	0	2	0	7	3
. Hyperaemia	1	1	0	1	0	3	1
B. LUNGS							
- Hyperaemia/congestion	0	1	1	0	0	2	1
- Oedema	0	1	1	0	0	2	0
- Red/Gray hepatization	0	2	0	2	0	4	4
- Emphysema	2	1	0	1	0	2	2
HEART							
. Haemorrhage	1	1	2	1	0	5	0
. Atrophy of coronary groove fat	1	2	0	0	1	4	1
. Mishapen/flabby	1	3	3	0	1	8	0
LYMPHATICS/SPLEEN							
. Atrophic	0	0	0	0	0	0	0
. Enlarged	4/3	3/1	23/3	0/2	0/2	9/11	1/0
. Haemorrhage	3/1	0	1/0	0	0	4/1	1/0
BRAIN							
. Hyperaemia/congestion	2	0	0	1	0	3	0
. Melanosis	0	0	0	1	0	1	0

TABLE 13 (Continued)

KIDNEYS							
. Mishapen	0	1	0	0	0	1	0
. Congestion	1	2	1	2	0	6	0
ENDOCRINE ORGANS							
A. PITUITARY							
. Atrophic	0	0	0	0	0	0	0
. Enlarged	0	0	0	0	0	0	0
. Congested	0	0	0	0	0	0	0
B. ADRENALS							
. Enlarged	0	2	0	0	0	2	0
.Hyperaemia/congestion	0	0	0	0	0	0	0
REPRODUCTIVE ORGANS							
A. OVARIES							
. Atrophic	1	0	0	1	0	2	0
. Fibrotic	1	0	0	1	0	2	0
. Follicular/luteal cyst	2	1	0	1	1	5	0
. Absence of corpus luteum	3	5	4	4	2	18	0
B. TUBULAR GENITALIA							
. Oedema	0	1	1	0	1	3	0
. Hyperaemia/congestion	0	0	0	0	0	0	0
. Segmental aplasia	0	0	0	2	0	2	0

7.0. Histopathology:

A summary of the histopathological findings of the pituitary, the adrenals, ovaries and the tubular genitalia are given in Table 14.

(a) The pituitary gland:

Lesions in 15 of the 19 infected goats and none in the controls (plate I) consisted of sparse lymphocytic infiltration of the connective tissue capsule and the subcapsular and perivascular spaces (plate II). Numerous foci of necrotic patches were observed in the pars distalis of one goat (No. 202) slaughtered at the 6th week PI (plate III). In addition, cyst-like bodies - some containing proteinaceous material in their lumen were also seen in the pars distalis of 3 goats, 2 slaughtered at the 6th week, and one at the 10th week PI (plate IV). A thrombosed vessel was observed in the pars distalis of the pituitary of animal No. 216. No lesions were seen in the pars nervosa of all the animals.

(b) The adrenal gland:

The adrenal gland of all control goats were normal (plate V). Melanin deposit was observed in the capsule of goat No. 234 slaughtered at the 4th week PI (plate VI). There was hypertrophy of the adrenal cortex of goats No. 202 and 239 slaughtered at the 6th and 13th weeks PI respectively.

This was characterized by a relatively thicker zonae fasciculata and reticularis when compared with the controls. In addition, a circumscribed foci of necrosis was observed in the zona glomerulosa of goat No. 216 slaughtered at the 10th week PI (plate VII).

(c) Ovaries

The ovaries of control goats were normal with variable numbers of primordial to tertiary follicles and most having apparently functional corpora lutea(CL) (plate VIII). In contrast, the ovaries of infected animals were characterised by a relative decrease in number, or complete absence of primordial to secondary follicles, while the tertiary follicles showed extensive degeneration of the granulosa cells, with the absence of CL (plate IX). These lesions were found in the ovaries of most of the animals irrespective of the duration of infection. Infiltration of the ovarian stroma by mononuclear cells mostly lymphocytes and a few plasma cells were noted in 5 goats, one each at the 4th and 6th week PI, and 3 at the 8th week PI. In addition, proliferation of fibrous connective tissue (plate X) was evident in half the number of animals except for those slaughtered at the 8th week PI. Two animals slaughtered at the 4th week PI, and one each at the 10th and 13th week PI had cystic degeneration of the follicles (plate XI) while one large corpus luteum cyst was seen in the ovary of goat No.211 slaughtered at the 6th week PI (plate XII).

(d) The uterine tubes:

No lesions were observed in the uterine tubes of both infected and control goats.

(e) The uterus:

The uteri of control animals were normal (plate XIII). Lesions observed in the infected animals consisted of areas of desquamation of the superficial and glandular epithelium (plate XIV). This was observed in 2 goats slaughtered at the 6th week PI. In addition, there were varying degrees of leucocytic infiltrations of the endometrium in 5 goats (plate XV), the predominant cell types being lymphocytes with admixtures of plasma cells, neutrophils and a few eosinophils. Extensions of these infiltrations into the myometrium and the serosa were observed in 2 of these animals.

(f) The cervix:

A focus of lymphocytic infiltration and oedema with disruption of the fibres of the longitudinal muscle layer was observed in the cervix of goat No.232 at the 10th week PI (plate XVI). The cervix of all other infected and control animals were normal (plate XVII).

(g) The vagina:

The vagina of all control animals appeared nrcmal (plate XVIII).

There was desquamation of the vaginal epithelium of 3 goats, one each slaughtered in the 6th, 8th and 13th week PI.

This was accompanied by mild to diffuse infiltration of the submucosa by lymphocytes, plasma cells neutrophils and occasionally eosinophils in all but one of the infected goats (plate XIX). There was concomittant but varying degrees of perivascular cuffing of most of the vessels in this area by lymphocytes (plate XIX). These lesions were most severe in the animals slaughtered at the 4th week PI. In addition, necrotic solitary lymphoid follicles were seen beneath the vaginal epithelium (plate XX) in 3 animals.

(h) The vulva:

Except for goat No. 202 which had oedema of the vulva, no lesions were seen in the other infected and control animals.

TABLE 14: Summary of histopathological findings in Red Sokoto does experimentally infected with T.vivax

Weeks Post Infection	4	6	8	10	13	Total	Control
No. of Animals in the Group	4	5	4	4	2	19	5
PITUITARY GLAND							
- Infiltration of capsule and Perivascular Spaces by mononuclear cells	4	2	3	4	2	15	0
- Presence of cyst in the pars distalis	0	2	0	1	0	3	0
- Necrosis	0	1	0	0	0	1	0
ADRENAL GLAND							
- Melanin deposit (capsule)	1	0	0	0	0	1	0
- Hypertrophy of the Cortex	0	2	0	0	0	2	0
- Necrosis	0	0	0	1	0	1	0
OVARIES							
- Decreased No. of 1^{o} or 2^{o} follicles	3	3	2	3	1	12	0
- Infiltration of stroma by mononuclear cells	1	1	3	0	0	5	0
- Fibrosis	2	2	0	2	1	7	0
- Cystic degeneration							
- Absence of functional C.L.	4	2	4	2	1	11	0
UTERUS							
- Desquamation of superficial epithelium	0	2	0	0	0	2	0
- Endometritis/metritis	0	2	2	0	1	5	0
CERVIX							
- Cervicitis	0	1	0	1	0	2	0
VAGINA							
- Desquamation of superficial epithelium	0	1	1	0	1	3	0
- Vaginitis	4	4	4	4	2	18	0
- Necrosis of solitary lymph follicles	1	1	0	0	1	3	0

Plate I: Pituitary gland of a control goat showing a normal pars distalis.
H & E: x400

Plate II: Pituitary gland of an infected goat showing lymphocytic infiltration of the capsule (arrow).
H & E: x400

Plate III: Pituitary gland of an infected goat showing necrotic patches (arrows) in the pars distalis.
H & E: x450

Plate IV: Pituitary gland of an infected goat showing a large cyst containing a proteinaceous material (arrow).
H & E: 400

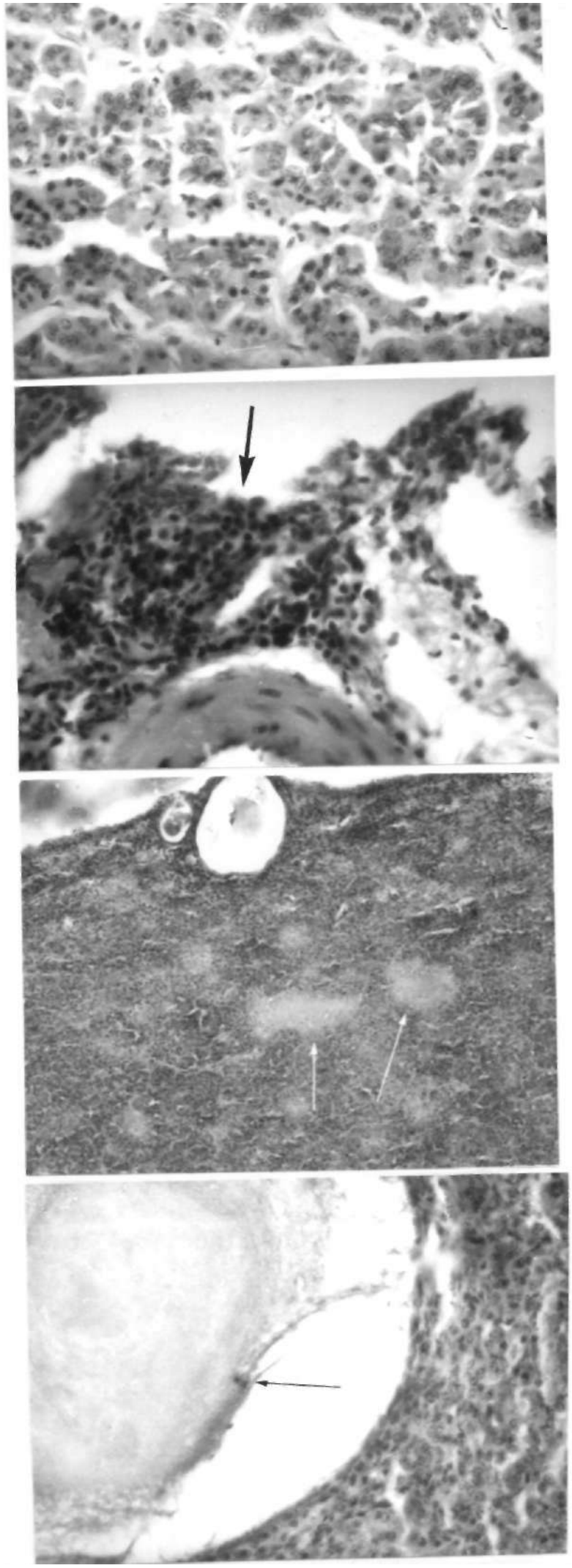


Plate V: Adrenal gland of a control goat showing a normal cortex:
H & E: x160

Plate VI: Adrenal gland of an infected goat showing melanin deposits in the capsule (arrow).
H & E: x160

Plate VII: Adrenal gland of an infected goat showing an area of necrosis (arrow) in the zona glomerulosa.
H & E: x160

Plate VIII: Ovary of a control goat showing normal primordial cells (a), Secondary (b) and tertiary follicles (c).
H & E: x160

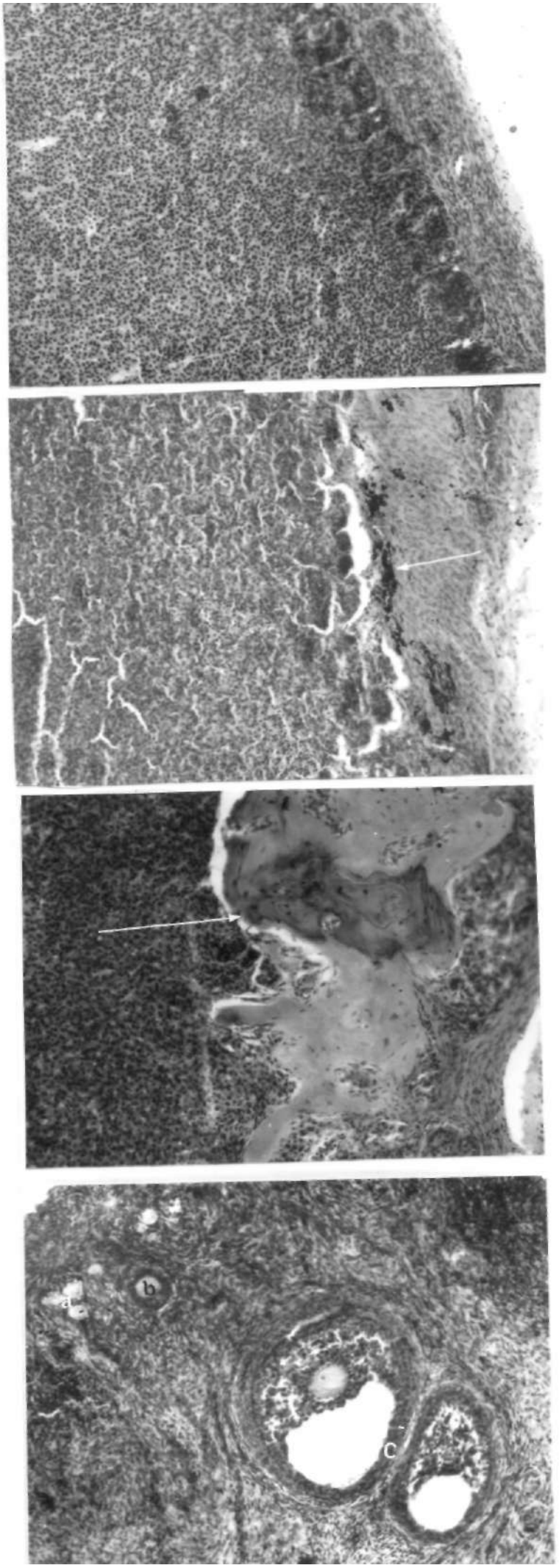
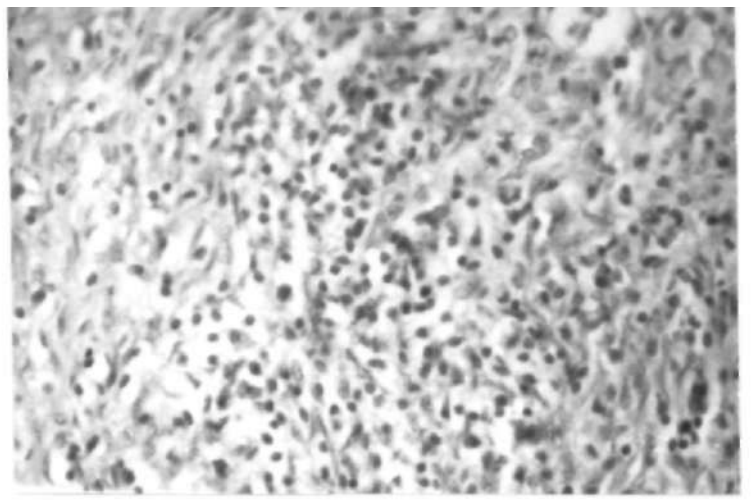

b
c

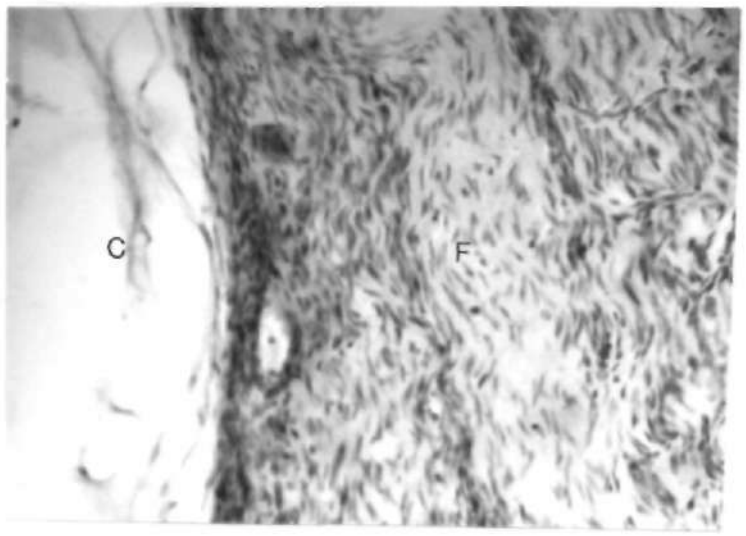
Plate IX: Ovary of an infected goat showing mononuclear cellular infiltrations.
H & E: x250

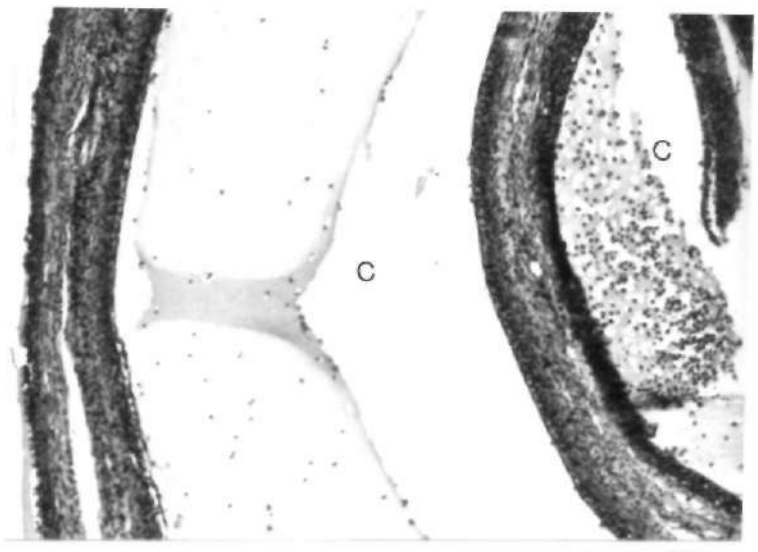
Plate X: Ovary of an infected goat showing follicular cyst (c) and fibrosis (f).
H & E: x250

Plate XI: Ovary of an infected goat showing sections of follicular cysts (c).
H & E: x150

Plate XII: Ovary of an infected goat showing a section through the lumen (l) and wall (w) of a corpus luteum cyst.
H & E: x150




C
F


C
C

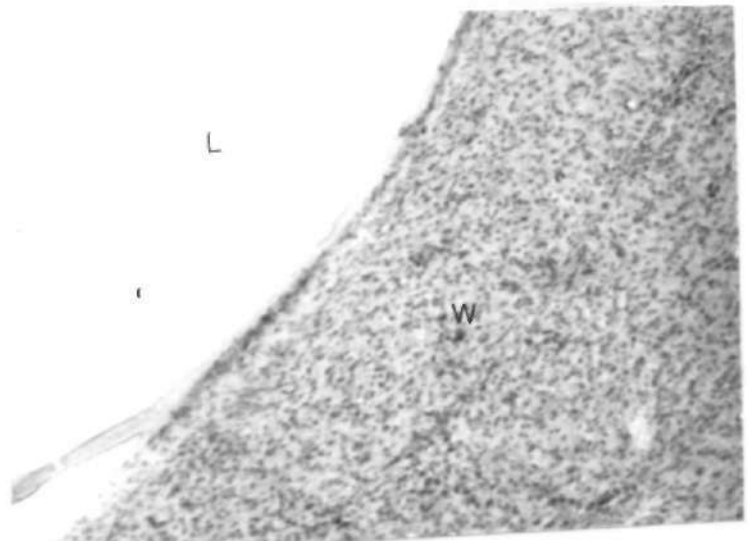

L
W

Plate XIII: Uterus of a control goat.

H & E: x250

Plate XIV: Uterus of an infected goat showing desquamation of the superficial epithelum of the endometrium (arrow).

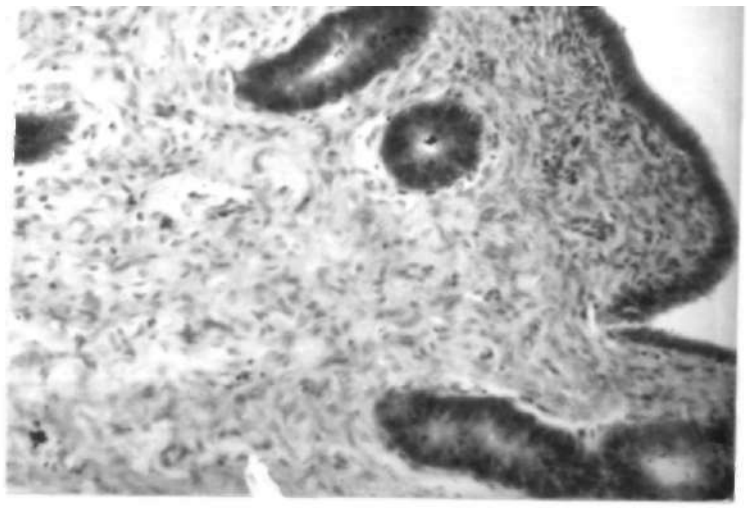
H & E: x250

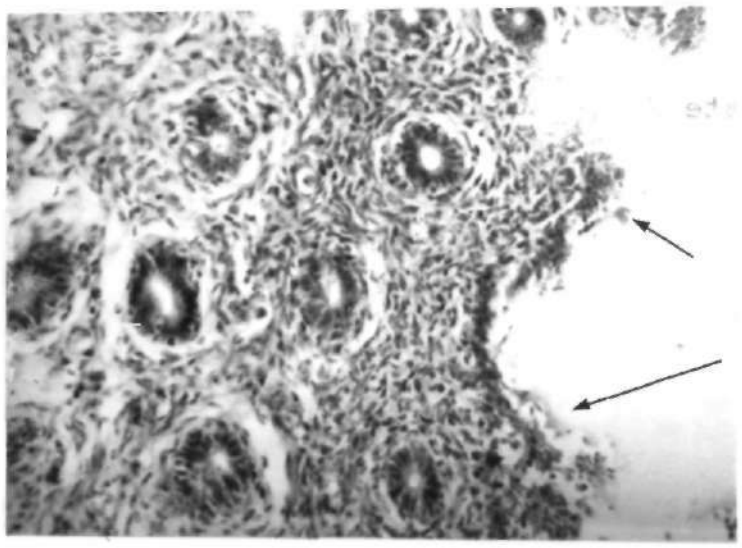
Plate XV: Uterus of an infected goat showing dense infiltration of the endometrium by inflammatory cells (arrow).

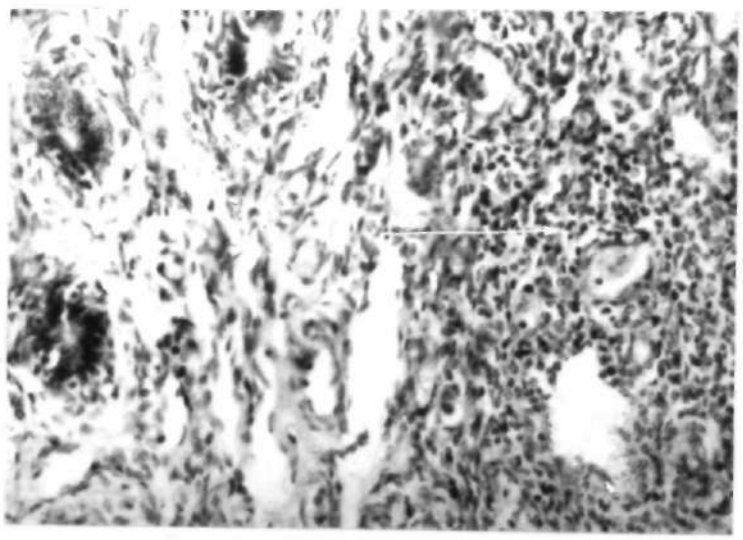
H & E: x250

Plate XVI: Cervix of a control goat showing normal serosa.

H & E: x250







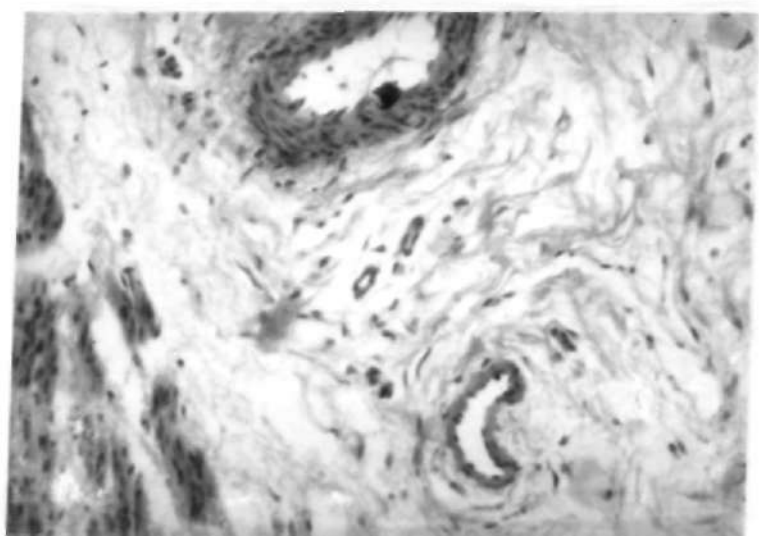
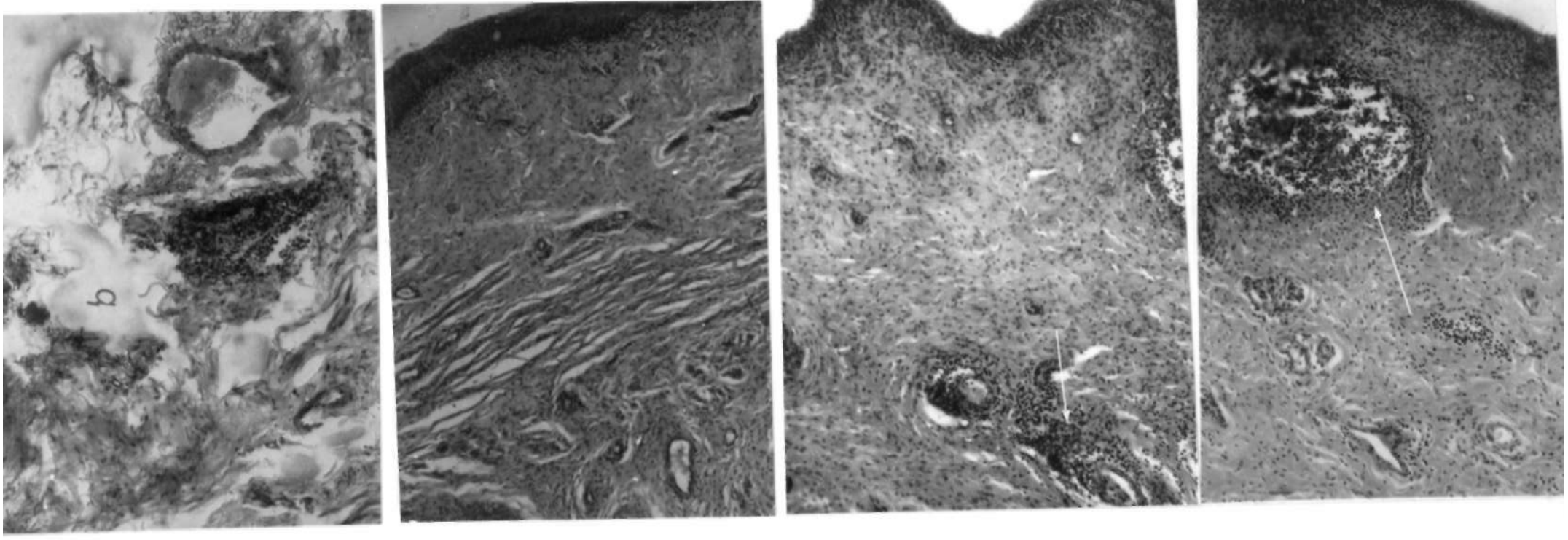


Plate XVII: Cervix of an infected goat showing a foci of mononuclear cellular infiltration (a) and edema (b) of the connective.
H & E: x160

Plate XVIII: Vagina of a control goat.
H & E: x160

Plate XIX: Vagina of an infected goat showing diffuse lymphocytic infiltration and perivascular cuffing (arrow) of the submucosa.
H & E: x160

Plate XX: Vagina of an infected goat showing a degenerating lymphoid follicle (arrow).
H & E: x160.


b

CHAPTER FOUR
DISCUSSION

The strain of T.vivax used in this study caused clinical trypanosomiasis in all the infected does. Following infection, a mean prepatent period of 4 days was obtained which is in agreement with the observations of Edwards et al. (1956a) and Saror (1980) for T.vivax infection in goats. The pattern of parasitaemia observed in the infected animals was usually cyclical with the highest levels occurring during the early phase of infection up to 4 weeks PI, while lower levels became manifest as the infection progressed. Similar observations have been made by Anosa and Isoun (1974; 1980b).

The infection was characterized by a highly fluctuating fever often coinciding with parasitaemic waves, although the degree of fever was not often proportional to the level of parasitaemia. This observation agrees with those earlier made by Anosa and Isoun (1974; 1980b), Veenendaal et al., (1976) and Saror (1980).

Ruminants infected with T.vivax have either failed to gain weight, lost weight or had reduced growth rate compared with control un-infected animals (Edwards et al., 1956a; Ogwu, 1983; Elhassan, 1987). A comparable finding was obtained in this study with the infected animals losing between 9.4 to 18.37% of their mean pre-infection body weights.

The changes in erythrocyte and leucocyte profiles might have been influenced by trypanosome antigens and plasma neuraminidase released by them (Esievo et al., 1982; Esievo and Saror 1983).

The gross pathological lesions observed in the infected animals are similar to those described elsewhere by various authors, and agree with the review of Anosa (1983) that: "the most striking feature of T.vivax infections in ruminants is the absence of any severe or pathognomonic lesions which may be expected to accompany the severe debilitation observed".

Inflammatory reactions of the capsule and patchy necrosis of the pars distalis observed in the pituitary are consistent with the findings of Ikede and Losos (1975), Masake (1980) and Mutayoba et al. (1988a). This is in addition to vascular thrombosis and the cysts seen in the pars distalis of some animals. Intravascular coagulation has been reported as a predominant feature of T.vivax infection in goats (Masake 1980; Anosa and Isoun 1983), but there has as yet been no reports of pituitary cysts in animals infected with trypanosomes. Jubb, et al. (1985) suggested that cysts may develop from the remnants of the distal craniopharyngeal duct, or may result from the failure of the differentiation of the oropharyngeal ectoderm of Rathke's pouch, and that except for brachycephalic dog breeds, the cysts normally disappear at birth in most animals species. Melanin deposits in the adrenal capsule is not significant and is unlikely to be due to the effect of trypanosome infection.

The adrenocortical hypertrophy observed in 2 of the infected animals compares favourably with earlier observations of Ikede and Losos (1975), Elhassan (1978) and Mutayoba *et al*. (1988a). This finding suggests an increase in adrenocortical activity.

Histopathological changes were most severe in the ovaries of infected animals. These included inhibition of follicular maturation, degeneration of the ovarian stroma and primordial germ cells, polycystic formation and lack of CL formation. Similar lesions have been described in the ovaries of female goats infected with *T.congolense* and these paralleled the cessation of oestrus cycles and significant decline in plasma progesterone and oestradiol levels (Luckins *et al*. 1986; Mutayoba *et al*. 1988a, b). These hormonal changes were not evaluated in this study. The occurrence of endometritis in cattle infected with *T.vivax* (Vohradsky, 1971) tallies with the findings in this study. In addition, there was metritis in 2 of the infected animals. Cervicitis was observed in only one of the infected animals, while severe vaginitis was a consistent finding in most of the infected animals. This is in consonance with the report of Mwongela *et al*. (1981).

The pathogenesis of the trypanosome-induced lesions in the pituitary, adrenals, ovaries and the tubular genitalia are not clear, and are poorly understood. Except for the adrenals, inflammatory reactions were the most consistent findings in these structures, and could possibly be due to the invasion of the these organs by trypanosomes - a process which has been shown to result in extensive damage in the ear of rabbits infected with T.brucei (Goodwin, 1970). Eventhough parasites were not demonstrated in the parenchymatous areas of these organs, the severe vasculitis observed in some of these organs could allow for extravasation of trypanosomes. This has been reported by Van den Ingh et al. (1976) and could account for the necrosis in the pituitary, or the necrosis might have accompanied anaemia and thrombosis (Jubb and Kennedy, 1970). Anaemia was marked in the infected animals, and is a consistent feature of animal trypanosomiasis (Saror, 1980). However, there was no evidence of ischaemia even though a thrombosed vessel was seen in the pituitary of one infected animal.

The significance of the cysts in the pituitary of infected animals in the present study are not known. However, Jubb et al. (1985) have suggestedd that it is possible that these cysts may become large enough to exert pressure, and causing adjacent structures to atrophy to varying degrees owing to compression and interference with blood supply.

The disruption of such cysts with the escape of the proteinaceous contents into the adjacent tissues may excite an intense local inflammation with subsequent fibrosis that may lead to pituitary dysfunction. The pituitary defects enunciated above, and those from other studies (Ikede and Losos, 1975; Masake, 1980; Mutayoba et al.,1988a, b) suggest that gonadal and adrenal anomalies could have occurred sequel to pituitary dysfunction. The suggested increase in adrenocortical activity in the infected goats in the face of pituitary damage appears contradictory. However, as Mutayoba et al. (1988a) suggested, it could be that adrenocortical hypertrophy might have occurred before the pituitary was sufficiantly damaged. Necrosis of the zona glomerulosa may cause inadequate secretion of mineralo corticoids leading to lethal retention of potassium and loss of sodium (Jubb et al. 1985).

Anosa and Isoun (1980a) had ascribed testicular degeneration in T.vivax infected sheep and goats to the thermal effects of pyrexia. Repeated bouts of pyrexia was observed in the infected goats used in this study, and it is possible that this might have contributed to the ovarian anomalies. The pathogenesis of ovarian cysts is not clear. However it is thought to be due to continuous excessive follicle stimulating action, presumable pituitary FSH

(Jones and Hunt, 1983), or due to a delayed or inadequate release of pituitary luteinizing hormone with the result that mature follicles are not exposed to the ovulating or luteinizing action (Jubb et al. 1985). In the light of pituitary lesions earlier mentioned, the latter explanation is the most probable.

The severe vaginitis observed coupled with cervicitis and endometritis/metritis would prevent conception. With vaginitis, copulation will definitely be painful and as such sexual receptivity will be difficult in affected animals. Moreso, inflammatory exudate in these structures could be toxic to the spermatozoa, and the ovum even if fertilized, would perish (Jones and Hunt, 1983). As a result of these lesions, the inflammed uterus may in pregnancy become hostile to any embryo or developing foetus with attempts to expel it (Hafez, 1968).

This study shows that trypanosomiasis adversely affects the reproductive function of female goats by causing degeneration of the pituitary with the attendant degeneration of primordial cells and inhibition of follicular maturation in addition to causing varying inflammatory lesions along the genital tract. However, the severity of these degenerative changes were not dependent on the duration of infection. The presence of necrotic areas in the pituitary of an infected animal is likely to affect its sensitivity to gonadotropin-releasing

hormone from the hypothalamus and thus, its capacity to adequately produce and store gonadrotropins. Even if these are produced in sufficient levels and ratios, the degenerate ovaries are unlikely to respond to them and in turn cause the reduced production and release of oestrogens and progesterones or viable ova. The interrelationship between reproductive hormones and the pathologic lesions observed in the endocrine glands and the tubular genitalia cannot be overlooked. A sequential measurement of plasma FSH, and LH, oestrogens, progesterones and corticotropins and relating these to tissue and organ lesions during the course of the disease in both non-pregnant and pregnant animals will possibly help explain the pathogenetic mechanisms of reproductive dysfunctions during trypanosomiasis.

CONCLUSION

1. This study provides further histopathological evidence that reproductive dysfunctions in the female animal may be due to degenerative changes in the pituitary, adrenal, ovary and the tubular genitalia.
2. The severity of the observed lesions are independent of the duration of infection.

3. There is the need for a concurrent investigation of the relationship between sequential pathological changes in the reproductive and endocrine organs, the level of reproductive hormones and the reproductive performance of female animals suffering from trypanosomiasis.

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