COMPARATIVE STUDIES ON THE SUSCEPTIBILITY OF RED SOKOTO, SAHEL AND WEST AFRICAN DWARF GOATS TO EXPERIMENTAL TRYPANOSOMA VIVAX INFECTION

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JULY, 1998.

DECLARATION

I HEREBY DECLARE THAT THIS THESIS IS A RECORD OF MY RESEARCH WORK. IT HAS NOT BEEN PRESENTED BY ME IN ANY PREVIOUS APPLICATION FOR A HIGHER DEGREE.

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My sincere gratitude goes to my husband, Squadron Leader EA Akinwale, for his prayers, love, care, assistance and encouragement that saw me through this work. I also thank my children, Ayobami, Ifeoluwa and Oluwadamilola, my God's love gifts, whose prayers and love also worked tremendously to see me through. Lastly I thank my parents - in - law, Mr. and Mrs. G. O. Akinwale and my parents, Chief and Mrs. T. K. Ogunleye for their love and prayers for me.

DEDICATION

This work is dedicated to the glory of God Almighty, the God of all flesh, to whom be glory for ever and ever, amen.

TABLE OF CONTENTS

		Page
TITLE PAGE		i
DECLARATION		ii
CERTIFICATION		iii
ACKNOWLEDGMENTS		v
DEDICATION		viii
ABSTRACT		ix
TABLE OF CONTENTS		xii
LIST OF FIGURES		xvii
LIST OF PLATES		xviii
LIST OF APPENDICES		xix
ABBREVIATIONS		xx
CHAPTER 1		
1.0	Introduction	1
1.1	History of the research problems	1
1.2	Economic importance of African Animal Trypanosomiasis	3
1.3	Aims	6
1.4	Objective	7
CHAPTER 2		
2.0	Literature Review	8
2.1	Trypanosoma vivax infection in ruminants	8

2.1.1 Trypanosomes and their regional or geographical significance..... 8

2.1.2 Vector..... 11

2.1.3 Host..... 13

2.1.4 Wild life as reservoirs..... 15

2.1.5 The infection..... 15

2.1.6 The incubation period..... 17

2.2 Factors affecting T. vivax infection in ruminants..... 18

2.3 Clinical, pathological and histological features of T. vivax infection in ruminants

2.3.1 Clinico-pathological features..... 19

2.3.2 Histo-pathological features..... 21

2.4 Terminal changes in animals infected with T. vivax 25

2.5 Anaemia..... 27

2.5.1 Mechanisms of anaemia in animal trypanosomiasis..... 28

2.6 Imunosuppression in trypanosomiasis..... 40

2.7 Diagnosis..... 43

2.8 Control..... 44

2.9 Importance and characteristics of small ruminants..... 45

2.10 Geographical distribution of goats..... 47

2.11 The three main breeds of goats in Nigeria..... 48

2.11.1 Red Sokoto 49

2.11.2 Sahel 49

2.11.3 West African Dwarf 49

2.12 Reasons for promoting goats production..... 54

CHAPTER 3

3.0 Materials and Methods..... 56

3.1 Experimental animals..... 56

3.2 Trypanosomes..... 56

3.3 *Acclimatization*..... 56

3.4 Animal inoculation..... 57

3.5 Clinical examinations..... 57

3.6 Temperature and body weight..... 57

3.7 Parasite enumeration..... 57

3.8 Haematological parameters..... 58

3.9 Free serum sialic acid assay..... 58

3.10 Bone marrow biopsy and examination..... 59

3.11 Postmortem..... 60

3.12 Treatment..... 60

3.13 Statistical analysis..... 60

CHAPTER 4

4.0 Results..... 61

4.1 The clinical and parasitological features of T. vivax infection

in Red Sokoto, Sahel and West African dwarf goats..... 61

4.1.1 Discharges from external orifices..... 61

4.1.2 Pale mucous membrane..... 61

4.1.3 Appetite..... 62

4.1.4 Enlargement of lymph nodes..... 63

4.1.5 Deppression..... 63

4.1.6 Rectal temperature..... 64

4.1.7 Body weight..... 69

4.1.8 Parasitaemia..... 69

4.2 The haematological response to T. vivax infection

by Red Sokoto, Sahel and West African Dwarf goats..... 74

4.2.1 The erythrocyte counts..... 74

4.2.2 The packed cell volume..... 75

4.2.3 The platelet counts..... 79

4.2.4 The total leucocyte counts..... 80

Differential leucocyte counts..... 82

4.2.5 Neutrophil counts 82

4.2.6 Lymphocyte counts 86

4.2.7 Eosinophil counts..... 86

4.2.8 Monocyte counts..... 87

4.3 Free serum sialic acid concentration..... 88

4.4 Bone marrow cytology 89

4.5 The gross pathology of T. vivax infection in

Red Sokoto, Sahel and West African dwarf goats..... 96

KASHIM IBRAHIM LIBRARY

5. DISSCUSION..... 99

SUMMARY AND CONCLUSION..... 106

REFERENCES..... 110

APPENDICES..... 133

LIST OF FIGURES

Figure	Title	Page
1.	Tsetse distribution in Nigeria	12
2.	Nigeria natural vegetation	51
3.	Rectal temperature (°C) of infected and control Red Sokoto, Sahel and West African dwarf goats	68
4.	Body weight (Kg) of infected and control Red Sokoto, Sahel and West African dwarf goats.....	71
5.	The parasitaemia (LEV) of infected and control Red Sokoto, Sahel and West African dwarf goats	73
6.	The erythrocyte counts (n x 10^6 / µl) of infected and control Red Sokoto, Sahel and West African dwarf goats	77
7.	The packed cell volume (%) of infected and control Red Sokoto, Sahel and West African dwarf goats	78
8.	The platelet counts (n x 10^5 / µ) of infected and control Red Sokoto, Sahel and West African dwarf goats	81
9.	The total and differential leucocyte counts (n x 10^3 / µl) of infected and control Red Sokoto goats	83
10.	The total and differential leucocyte counts (n x 10^3 / µl) of infected and control Sahel goats	84
11.	The total and differential leucocyte counts (n x 10^3 / µl) of infected and control West African dwarf goats	85
12.	The free serum sialic acid concentration (mg / dl) of infected and control Red Sokoto, Sahel and West African dwarf goats	91

LIST OF PLATES

Plate	Title	Page
ia.	An uninfected Red Sokoto goat	52
ib.	An uninfected Sahel goat	52
ic.	An uninfected West African dwarf goat	53
iia.	An infected Red Sokoto goat four weeks post-infection showing weakness and recumbency	65
iib.	An infected Sahel goat four weeks post-infection showing weakness and soiled hindquarter	65
iic.	An infected West African dwarf goat four weeks post-infection showing weakness and rough hair coat	66
iiia.	The pre-infection bone marrow smear of a Red Sokoto goat showing myeloid cells (arrow head) and erythroid cells (arrow)	92
iiib.	The pre-infection bone marrow smear of a Sahel goat showing myeloid cells (arrow head) and erythroid cells (arrow)	92
iiic.	The pre-infection bone marrow smear of a West African dwarf goat showing myeloid cells (arrow head) and erythroid cells (arrow)	93
iva.	The post-infection bone marrow smear of a Red Sokoto goat three weeks post-infection showing proleferation of erythroid cells	94
ivb.	The post-infection bone marrow smear of a Sahel goat three weeks post-infection showing proleferation of erythroid cells	94
ivc.	The post-infection bone marrow smear of a West African dwarf goat three weeks post-infection showing proleferation of erythroid cells	95

LIST OF APPENDICES

Appendix	Title	Page
1.	The pre-infection bone marrow cytology (%) of Red Sokoto goats	133
2.	The pre-infection bone marrow cytology (%) of Sahel goats	134
3.	The pre-infection bone marrow cytology (%) of West African dwarf goats	135
4.	The post-infection bone marrow cytology (%) of Red Sokoto goats	136
5.	The post-infection bone marrow cytology (%) of Sahel goats	137
6.	The post-infection bone marrow cytology (%) of West African dwarf goats	138
7.	The chemical names of drugs used	139

ABBREVIATIONS

°C	Degree centigrade
±	Plus or minus
Kg	Kilogramme
%	Percentage
µl	Microlitre
mg / dl	Miligramme per decilitre
ml / Kg	Mililitre per kilogramme
mg / Kg	Miligramme per kilogramme
ad libitum	At will
pH	Hydrogen ion
ml	Mililitre

The most important trypanosome species affecting domestic livestock in Africa are Trypanosoma congolense, Trypanosoma vivax, Trypanosoma brucei brucei in cattle, sheep and goats, Trypanosoma simiae in pigs and Trypanosoma evansi in camels (Molyneux and Ashford, 1983; Trail et al., 1989). Each of these species has its own behavioural pattern and each consists of an undefined number of different strains, all capable of elaborating their own characteristic repertoires of variable antigen types (Murray, 1989).

Animal trypanosomiasis causes symptoms manifested by increase in heat production and body temperature, intermittent fever, anaemia, lymphatic enlargement with hepatosplenomegaly and a progressive cachexia (Dam et al., 1996). The animals become lethargic and develop dull starry coat, pale mucous membrane, subcutaneous oedema, ascites, ocular discharge and emaciation (Losos et al., 1973). Omotainse and Anosa (1995) and Onah et al. (1996) also observed some haematological changes in animals infected with trypanosomes. Sekoni (1994) reported that reproductive disorder caused in male animals included delayed puberty, loss of libido and severe degenerative changes of the genitalia manifested by the production of very poor quality semen or the cessation of semen production. In female animals, he noted that animal trypanosomiasis causes severe genital lesions, temporary or permanent anestrous and abnormal oestrous cycles. Akinbamijo and Reynolds (1994) reported low maternal weights and poor body condition at parturition associated with low lamb birth weights and survival. They also reported increased

Chapter 1

INTRODUCTION

1.1 History of the Research Problems

One of the two most important protozoan diseases of livestock on the African continent is trypanosomiasis, the other being East coast fever, theileriosis (ILRAD, 1984). Scientists from many countries have been working for decades to develop effective methods to control these diseases, but this task has proven very difficult due to the complex nature of the diseases and their vectors. Ford (1971) observed that trypanosomiasis is a serious, often fatal parasitic disease which occurs in large areas of Africa, Latin America, the middle East and Asia. It affects man, most species of domestic livestock and many types of wild animals. In Africa, trypanosomiasis is found in approximately one-third of the continent.

In tropical Africa, protozoan parasites cause several diseases of social and economic importance. One of the most devastating, which is trypanosomiasis, is caused by infection with trypanosomes, which are transmitted by tsetse flies to man, domestic and wild animals. Trypanosomiasis constitutes one of the most important problems to which man and his domestic livestock are exposed. It is a debilitating disease which is endemic in a large part of Africa, and some parts of South America. Man is infected by Trypanosoma brucei gambiense and Trypanosoma brucei rhodesiense causing sleeping sickness. While African animal trypanosomiasis is a debilitating disease called nagana", which constrains on agricultural development of more than a third of the African continent by causing livestock production losses (ILRAD, 1990).

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abortion and lamb mortality rates with less milk production during late lactation and loss of weight in infected lactating ewes.

1.2 Economic Importance of African Animal Trypanosomiasis

Sekoni (1994) stated that pathogenic trypanosomes are the causative agent of the most common livestock diseases and trypanosomal-induced reproductive disorders in animals are of significant economic importance, especially in sub-Saharan Africa, where tsetse-transmitted trypanosomiases are endemic and causes losses of pasture. Wilson et al. (1963) estimated that 10.4 million square kilometres of grazeable land in Africa is devoid of cattle due to trypanosomiasis thereby making some pasture unavailable for livestock production by trypanosomiasis. This has caused serious overgrazing of tsetse free areas with damage to the vegetation and consequent southward advancement of the Sahara desert.

Losses in livestock production, associated with deaths, particularly during migration between tsetse free and tsetse - infested pasture during the dry and rainy seasons also occur. Animals also lose weight due to infections by trypanosomes; decreased milk production (Agu, 1984), abortions and stillbirths (Elhassan, 1987; Okech et al ., 1996) and infertility due to testicular and cystic ovarian degeneration (Anosa and Isoun, 1980a). Sekoni, (1994) in his review stated that animal trypanosomiasis causes a wide range of reproductive disorders resulting in disruptions of the secretions and plasma concentrations of the hormones necessary for normal reproductive processes in both male and female animals.

Anosa (1983) observed that the presence of trypanosomiasis has militated against settled livestock farming in most parts of the guinea-savanna and forest belts of West Africa thereby causing precipitation and perpetuation of nomadism. Cattle migrations satisfy the necessity to move cattle northwards to tsetse-free area in the wet season and southwards to scanty pastures in tsetse-infested areas during the dry season. Migrations are also necessary to supply cattle to densely populated areas of the guinea-savanna and forest zones which cannot support intensive cattle-raising because of trypanosomiasis. These migrations often lead to serious weight losses and even deaths of livestock, and other varied effects on the nomads, such as denying their children of the benefits of formal education and modern agricultural technology.

Three approaches to the control of trypanosomiasis are chemotherapy, chemoprophylaxis and tsetse fly control. Eisler et al. (1996) reported that isometamedium chloride protected cattle against trypanosome infections for about 18 weeks under a chemoprophylactic regime in a tsetse-infested area of Zimbabwe. Fox et al. (1993) reported that animal trypanosomiasis was controlled for about 30 years by prophylactic drugs in a heavily tsetse-infested part of north-east Tanzania after which drug resistance developed. They observed that when cattle were dipped in synthetic pyrethroid deltamethrin the tsetse population decreased by more than 90%. Bauer et al. (1995) also used pyrethroid deltamethrin in a campaign against tsetse flies in the pastoral zone of Samorogouan, Burkina Faso and recorded a decrease

in the fly population. Offori (1993) reported the use of Sterile Insect Technique (SIT) as a control measure against tsetse flies in some countries in both West and East Africa by some workers but complete eradication was not achieved. Other chemicals like DDT, triflumuron, cypermethrin have also been used as a control measure against tsetse flies. Leak et al. (1996) stated that while effective tsetse control methods can be used to reduce the transmission of trypanosomes, their long-term impact is dependent on their sustainability.

However, trypanosomes present a major control problem due to their ability to avoid total destruction by the host's immune system by sequentially changing the protein antigens displayed on their surface membrane (ILRAD, 1989). This ability of the parasite to change the antigenic composition of its surface coat repeatedly is the primary mechanism that prevents most domestic animals from developing effective immunity against trypanosomiasis. In the laboratory, a variety of immunization procedures have been used which produce a strong resistance in animals to challenging trypanosome infection (Wellde et al., 1979). None of these procedures, however, has been shown to be effective against the disease in nature. This has been due partly to the variant specific nature of the protective immune response and to the relatively obscure antigenic structure of the naturally transmitted metacyclic trypanosomes.

Griffin and Allonby, (1979), Kanyari et al. (1986) and Mutayoba et al. (1989) studied trypanosomiasis in the small East African breed of goats and observed some degree of tolerance among the dwarf breeds. The West African dwarf sheep and goats have also been shown to posses a significant degree of tolerance to trypanosome infection (Opasina and Onyeka, 1982). It has therefore become necessary that more precise research information be made available on the susceptibility of all goat breeds in Nigeria : namely the Red Sokoto, Sahel and West African dwarf goats to trypanosome infection if these animals are to play their part in meeting people's large and growing demand for meat and milk.

1.3 Aims

1. To examine the effects of animal trypanosomiasis on Red Sokoto, Sahel and West African dwarf goat breeds by challenging them with a standard number of Trypanosoma vivax .
2. To compare the relative susceptibility of these goat breeds to experimental Trypanosoma vivax infection.
3. To monitor their subsequent patterns of parasitaemia, and relate the findings to the accompanying anaemia.

1.4 Objective

The objective of this work is to gain a clearer understanding of the factors that influence the susceptibility of these breeds of goats to trypanosomiasis. This can be

achieved by examining and comparing in the three breeds of goats, the following parameters:-

1. The pattern of parasitaemia after experimental infection.
2. The degree of anaemia through the estimation of their PCV, bone marrow responses and concentrations of the free serum sialic acid, to some extent.

Chapter 2

LITERATURE REVIEW

2.1 Trypanosoma vivax Infection in Ruminants

In Africa, south of the Sahara, an area 1.5 times the size of the United State of America, although suitable, cannot be fully used for livestock production, due to the presence of tsetse flies and the debilitating effect of trypanosomiases that they transmit (Murray et. al., 1982).

2.1.1 Trypanosomes and their Regional or Geographical Significance

Trypanosoma vivax is a parasite of major importance in West and East Africa, particularly in the former and is about the most important cause of animal trypanosomiasis by virtue of its predominance, compared to T. congolense and T. brucei . T. vivax is also able to survive in places devoid of its traditional vector, Glossina species, such as in parts of the Africa mainland, the island of Mauritius and several South American countries (Wells et al., 1980). T. vivax has remained the most predominant species, although there are reports of surveys in which T. congolense was predominant but such cases were attributed to the greater susceptibility of T. vivax to trypanosoma drugs with which the herds studied had been treated previously (Folkers and Jones - Davies, 1966). Esuruoso (1974), Losos (1986), Daniel et al. (1994), Anosa et al. (1995), and Kalejaiye et al. (1995) observed that T. vivax is the predominant trypanosome species of ruminants in the field.

Esuruoso (1974) stated that the reason for this may be due to mechanical transmission or the shorter development cycle of the trypanosome in the anterior station of the tsetse fly. Anosa (1983) reported that a survey of cattle slaughtered at the Nsukka abattoir between August 1979 and April 1980 showed that T. vivax was the only trypanosome that infected all the animals that were found to be suffering from trypanosomiasis. In East Africa, T. vivax is thought to be less important and less virulent than T. congolense in cattle (Willet, 1970). In Nigeria, T.vivax is observed to be the predominant cause of bovine trypanosomiasis and probably the most virulent (Ogwu and Njoku, 1987) making it to be about the most important cause of animal trypanosomiasis. It has also been observed that T. vivax is better adapted to development in tsetse flies than other trypanosomes (Hoare, 1972). Leeflang (1975) also reported that as one moves outside a focus of Glossina, the percentage incidence of T. vivax in cattle increases compared to T. b. brucei and T. congolense.

Mechanical transmission by biting flies such as Tabanids and Stomoxys has been recognized as an important factor in the transmission of T. vivax outside the Glossina zone in some parts of Africa (Mwongela et al., 1981). Kalu (1996) reported an outbreak of acute trypanosomiasis in a sedentary herd on the tsetse-free Jos Plateau, Nigeria where Tabanids and Stomoxys are prevalent. Ikede and Losos (1972) have also reported transplacental transmission of T. vivax as indicated by the presence of the parasites in a lamb at birth. T. brucei brucei is a tissue invading trypanosome which affects all domestic and wild ruminants, horses, dogs, cats, monkeys and

rodents (Molyneux and Ashford, 1983). T. congolense also has a wide range of hosts and some devastating effects on livestock. Natural infections of T vivax have been described in sheep and goats by Stephen (1970) and Dipeolu (1975). They observed that although natural infections of T.vivax in sheep and goats are relatively uncommon, they may be found in areas where it is impossible to keep cattle.

Bruce et al. (1911) demonstrated that T. vivax was transmitted by tsetse - flies. It has subsequently been stated that it is better adapted to development in these flies than other trypanosomes as shown by the dissection of tsetse - flies caught in the wild (Roberts and Gray, 1972). Mwongela et al. (1981) reported that mechanical transmission by bitting flies such as tabanids and stomoxys is an important factor in the transmission of T. vivax, and is responsible for the existence of its infections outside the Glossina zone in parts of Africa, the island of Mauritius, and the New World.

African trypanosomes exhibit a wide range of virulence in vertebrate hosts. Virulence is a variable characteristic of disease producing micro-organisms. Murray and Morrison (1978) observed that virulence is related to a host's capacity to limit the level of parasitaemia and the ability to control, or even eliminate the trypanosomes. They also attributed virulence to the capacity of different strains of the parasite to generate biologically active molecules, to activate complements and to undergo antigenic variation.

2.1.2 Vector

Glossina species are the insect vectors of the pathogenic salivarian trypanosomes. They inhabit over a third of the African continent (ILRAD, 1989) and expose about 30% of the estimated 160 million ruminants to the risk of infection. In Nigeria, they are found in the Guinea savanna down through the rain forest to the mangroove swamp forest (Fig.1). Animals in tsetse-infected areas are under constant challenge by tsetse flies carrying various trypanosome species or serodemes. In these situations, super-infections of animals are known to occur frequently (Masake et al., 1987). Page (1972) observed that the natural infection rates of Glossina species with trypanosomes of the T.brucei group are generally low. Several species of trypanosomes can be transmitted by more than twenty species of tsetse to many different species of domestic animals (Murray, 1989). He also noted that one infective trypanosome is sufficient to establish infection as is one infective tsetse. He however emphasised that an infected tsetse is not necessarily an infective one, and different species of tsetse have different capacities to transmit infection.

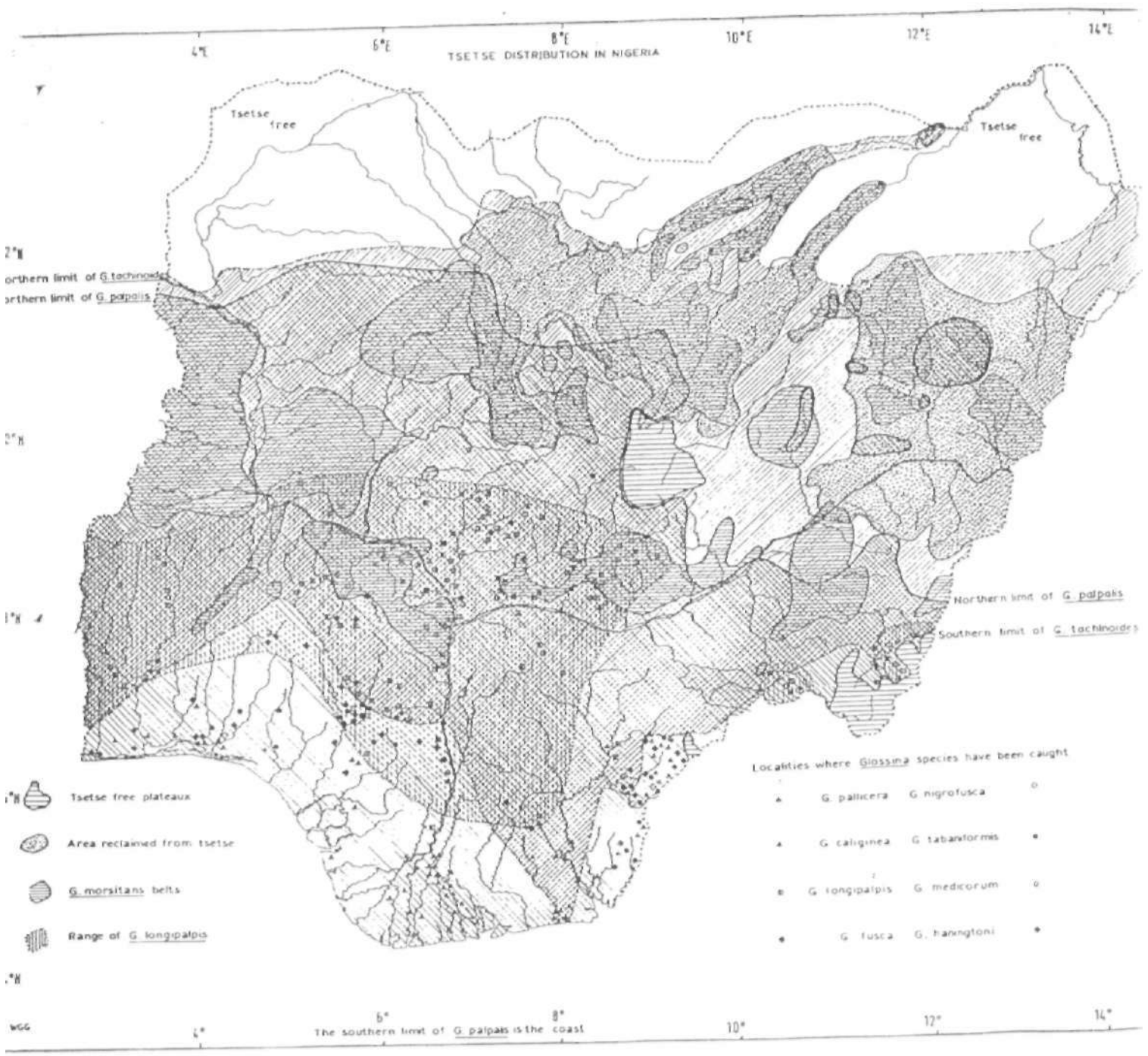


Fig. 1: TSETSE DISTRIBUTION IN NIGERIA

trypanosome serodemes. Esievo et al. (1986) also suggested that the N'dama may achieve its trypanotolerance through a more slowly released erythrocyte sialic acid during trypanosome infection. They noted that due to the much higher sialic acid concentrations in N'dama, its erythrocyte surfaces are much stronger than those of the Zebu, or the higher erythrocyte sialic acids on N'dama red cells will require much higher trypanosome concentrations (numbers), hence, higher sialidase activities than Zebu to achieve the same degree of anaemia. Esievo et al. (1990) found two types of sialic acid on N'dama erythrocytes as opposed to one type on zebu erythrocytes.

The environmental factors: These include age, nutrition, sex of the host, stress and previous exposures to infection. Wellde et al. (1981) noted that calves of less than one year are more resistant than adult. Work at the International Trypanotolerance Centre in the Gambia has demonstrated remarkable rates of growth and significant reduction in the severity of disease in N'dama cattle on a supplemented diet (Murray, 1989). There are reports that the stress of pregnancy, parturition, lactation and suckling can cause recrudescence and exacerbate infections (Ogwu et al., 1984; Elhassan, 1987; Edeghere et al., 1992). In addition to the effect of the reproductive cycle, other stress factors incriminated in exacerbing infection are overwork, repeated bleeding and intercurrent infection (Murray, 1989).

2.1.4 Wild Life as Reservoirs

The role of wild life as reservoirs of trypanosomes has been studied in East Africa. A review by Anosa (1983) of the incidence of trypanosomes in game animals in East Africa showed that 70 out of 1,242 (6.3%) of various game animals were positive for T. vivax. Ashcroft, (1959) observed that the wild animals most commonly infected by this organism were the waterbucks (33%), reedbuck (26%), giraffe (16%) with 12% accounting for bushpigs.

2.1.5 The Infection

The period immediately after tsetse fly bites is important both in establishing infection and expression of individual metacyclic variable antigen types (M-VATS) as well as the induction of immunity in the host (Taiwo et al., 1990). The earliest sign of trypanosomiasis following infection is an inflammatory reaction caused by the multiplication of trypanosomes in the skin at the site of the tsetse fly bite. There is a slight increase in lymph flow and lymphocyte output, and a dramatic rise in the relating number of lymphoblasts coincide with the formation of chancre. This is a localised reaction in the skin of a susceptible host to the presence of proliferating trypanosomes (Barry and Emery, 1984). Morrison et al. (1985) observed that the development of chancre occurs within a few days after the susceptible host has been bitten by infected tsetse flies. They noted that the reaction reaches a peak during the second week before declining to undetectable levels during the third week. These sites contain foci of trypanosomes in the dermal collagenous connective tissue which persist for 1 to 3 weeks after infection.

The development of local skin reactions (chancres) at the site of inoculation of metacyclic trypanosomes by the flies is an established part of the pathology of trypanosomiasis (Taiwo et al., 1990). The chancre serves as the site for establishing infection and also as the focus for multiplication and persistence of trypanosomes before their dissemination into the blood streams. It also provides a meeting place for the first contact between the trypanosome antigens and the host immune system (Akol and Murray, 1982 ; 1983).

Barry and Emery (1984) observed that the reaction is least severe in T. vivax infections. They reported that the chancre could serve as a site for sensitization of the host's immune system and this is indicated by the presence of large numbers of lymphoblasts and plasma cells, some of which contain Russell bodies in the later stages of chancre development. They also demonstrated that the development of protective immunity to an homologous tsetse - transmitted challenge depended on the timing of treatment in relation to the development of the chancre. They observed that animals treated 15 days or more after infection displayed complete acquired immunity, while those treated earlier than day 15 displayed partial or no immunity to homologous challenge. Taiwo et al. (1990) gave a possible explanation for the above observation; they said that the dividing trypanosomes in the chancre might retain the protective surface antigen epitopes of the corresponding metacyclics, so that the longer the treatment is delayed, the better the priming of the immune response by the antigens of the dividing trypanosomes in the chancre.

2.1.6 The Incubation Period

The incubation period of T. vivax infections of ruminants varies considerably, and is influenced by the route of infection (Anosa, 1983). This period in cattle, sheep and goats is discussed as follows:

Cattle: Intravenous infection of cattle is observed by Esievo and Saror (1983), Ogwu et al. (1984) and Sekoni et al. (1988 ; 1991), to produce the shortest incubation period, ranging from 3 to 8 days, while Masake (1980) recorded 4 to 12 days, but usually below 10 days in subcutaneously infected cattle. Cattle exposed to infected Glossina species had an incubation period of 8 to 20 days (Anosa et al., 1992).

Sheep: In sheep, the incubation period with intravenous infection with T. vivax is between 2 to 4 days (Agu et al., 1986; Sekoni, 1992 ; 1993). Anosa and Isoun (1983) observed an incubation period of 8 to 10 days in sheep infected intraperitonealy with T. vivax and an incubation period of 4 to 6 days in sheep exposed to laboratory bred G. palpalis .

Goats: Saror (1980) reported an incubation period of 4 to 7 days in goats infected intravenously, Masake (1980) and Kalu et al. (1987) recorded an incubation period of 5 to 7 days in goats subcutaneously inoculated with T. vivax, while Anosa (1983) reported an incubation period of 10 to 12 days in goats exposed to infected G. morsitans. Typically, T. vivax appears in the blood after the incubation period and the infected animals develop several waves of parasitaemia which are separated by

aparasitaemic or low parasitaemic periods (Anosa, 1983). The levels of parasitaemia are usually highest in the early phase of the infection while lower levels become manifest later in infection. Intravenous infection has been shown to produce the shortest incubation period.

2.2 Factors Affecting T. vivax Infection in Ruminants

Several factors influence the nature of clinical disease in ruminants. These are :- the species and strain of parasite, species, breed and age of the animal. Secondary infections also constitute a major factor influencing T.vivax infection in ruminants (Anosa, 1983). Since infected animals are immunodepressed, Rurangirwa et al. (1978) noted that secondary infections are common findings in infected animals. These are haemonchosis, haemoprotozoon diseases such as anaplasmosis, babesiosis, theileriosis and bacterial infections like salmonellosis, subcutaneous abscesses, purulent cellulitis and pneumonia. These secondary infections usually exacerbate the disease and cause early death. Malnutrition and other stress factors can predispose animals to or exacerbate clinical manifestations of trypanosomiasis.

infections in cattle, sheep and goats are characterised by fever; Anosa and Isoun (1980b ; 1983) and Saror (1980) observed that ruminants infected with T. vivax either fail to gain weight, lose weight or have a reduced growth rate compared to control animals. Pale mucous membranes, diarrhoea, sometimes with voidance of blood - stained faeces, haemorrhages in the gastro-intestinal tract, depressed rumina motility and loss of appetite in cattle, abortion and still births with congestion and oedema of the placenta, and blood - tinged allantoic and amniotic fluids have also been reported in ruminants (Masake, 1980 ; Anosa, 1983). Okech et al. (1996) also reported premature calving, perinatal loss, retained placenta, low birth weights and a prolonged period to the onset of postpartum ovarian activity in cattle infected with T. vivax. Nevertheless, with poor nutrition or secondary infections which are quite common in trypanosome - infected areas, animals may continue to lose weight or show growth retardation despite the abatement of parasitaemia.

Depressed milk yield and weight loss during late lactation were observed in infected West African dwarf ewes by Akinbamijo and Reynolds (1994). Accelerated heart rate has been observed in infected cattle with staring and roughened hair coat ,hair loss, swollen superficial lymph nodes, haemorrhages and epistaxis (Hill and Esuruoso; 1976) , Esuruoso (1977), Obi and Anosa (1980) and Anosa (1983). Losos (1978) and Masake (1980) observed bottle jaw, swollen head, corneal opacity, lacrimation, nasal discharge, dehydration, grinding of teeth and lowered heads also in sheep and goats infected with T. vivax. Anosa (1983) observed 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 terminally. Other gross lesions observed include emaciation, dehydration, oedema and enlargement of the lymph nodes, splenomegaly, hepatomegaly, gelatinous atrophy of body fat, pallor of the mucous membrane, enlargement of haemolymph nodes and accumulation of straw-coloured fluid in the serosal cavities (Saror, 1980; Anosa, 1983; Anosa and Isoun, 1983; Anosa et al., 1992). The symptoms observed by Opasina and Onyeka (1982) in trypanosome infection of goats include segregation from the rest of the groups in the shelters, pyrexia, pale mucous membranes of the eyes and mouth, dullness, weakness, drooping of the head, anorexia, emaciation, loss in weight, dyspnoea and anaemia.

2.3.2 Histo-Pathological Features.

Hyperplasia of the Mononuclear Phagocyte System (MPS): This occurs in the spleen, liver, lymph nodes, haemolymph nodes and lungs (Anosa and Isoun,1983). This change, they noted is consistent with the increased need to phagocytize particulate materials in the form of trypanosomes and damaged red blood and tissue cells. Exposure of the MPS to damaged red cells leads to marked proliferation of the phagocytes. The evidence of increased red cell destruction by the MPS is erythrophagocytosis and the presence of haemosiderin in the macrophages of various organs according to Anosa and Isoun (1980b).

Hyperplasia of the Lymphoid System: This is apparently a reaction to antigenic stimulation by trypanosomes. In animals with acute or subacute disease, there is depopulation of the lymphoid nodules (hypoplasia) in the spleen, lymph nodes and haemolymph nodes. This change, according to Naylor (1971) may represent an exhaustion phenomenon in the face of continuous intense antigenic stimulation. Anosa and Isoun (1983) observed that the severity of the disease influences the nature of the lymphoid tissue reaction.

Disseminated Intra- Vascular Coagulation (DIC) in the Blood Vessels: The most significant evidence of disseminated intravascular coagulation (DIC) is fibrin clots in the blood vessels, particularly of the brain and kidney. DIC is most common in sheep and goats which die of acute infection (Ingh et al., 1976). Another evidence of DIC is the rapid clotting of blood during bone marrow biopsy of some animals. Anosa and Isoun (1983) suggested that DIC might be due to vascular damage, or be precipitated by trypanosome antigen - antibody complexes.

Thickening of the Alveolar Septa of the Lungs: Edwards et al. (1959), described oedema, congestion, mononuclear cells infiltration and haemosiderosis of the lungs in *T. vivax* infected goats. Thickening of the alveolar epithelium of the lungs by oedema fluid, mononuclear cell infiltration, collagen deposition and sometimes congestion, without involvement of the alveolar lumen, had also been observed in animals without complicating pneumonia (Anosa and Isoun, 1983). They also

observed that the main pathological changes predisposing to death, which are anaemia, disseminated intravascular coagulation and hypoplasia of the lymphoid system are more marked in animals that died of acute or subacute infection. They suggested that the ability of an infected animal to withstand the disease may be associated with milder anaemia and an almost intact or even hyperplastic lymphoid system.

Reproductive Abnormalities: Trypanosomiasis caused by T. vivax infection is an important causative agent of infertility in ruminants, in areas where pathogenic trypanosomes and their vectors are prevalent (Sekoni et al., 1988; Sekoni, 1992; 1993). Recent studies have concentrated on the pathology of trypanosoma infections on the reproductive organs of some domestic animals (Anosa and Isoun, 1980a, 1983). Agu et al. (1986) studied the effect of T. vivax infection on semen quality of rams and reported very adverse effects of T. vivax infection on the semen quality. They observed that the sperm count, the percentage of live sperms and the percentage with progressive motility were markedly reduced. Also they stated that the percentage of sperm with morphological abnormality was very high. A number of testicular pathological changes have been described in animals experimentally infected with trypanosomes (Anosa and Isoun, 1980a; Ikede and Akpavie, 1982). Such lesions include mild to severe testicular degeneration, scrotal oedema, non-suppurative granulomatous periorchitis, atrophy, calcification and sclerosis. They observed that in some cases, these inflammatory and degenerative changes in the

genital organs might lead to total aspermatogenesis. Akpavie et al. (1986) studied the ejaculate characteristics of sheep infected with T.brucei and T.vivax. They noted that the changes in ejaculate of sheep infected with T.brucei were characterised by watery semen, a reduction in both sperm motility and concentration, an increase in the percentage of dead spermatozoa and an increase in sperm abnormalities. They observed that T.vivax infection was the same as above except that no appreciable effect on sperm concentration was observed.

Ogwu et al. (1984) and Elhassan (1987) reported intra-uterine infections resulting in still birth or neonatal mortality in T.vivax infections, while Edeghere et al. (1992) reported that T. b. brucei caused abortion, neonatal death and also death of infected dams irrespective of the trimester of gestation and the high dam mortality rate of 100% in all trimesters in T.b.brucei increases the constraint of livestock productivity in Africa. Agyemang et al. (1990) reported a substantial decline in milk production due to trypanosome infection in N'dama cattle in the Gambia. The same researchers demonstrated from carefully recorded data that trypanosome infection impairs reproductive performance post-partum in cows (Agyemang et al., 1991).

Ikede and Akpavie (1982) reported genital lesions in sheep infected with T. vivax. Ogwu et al. (1984) reported that experimental T.vivax infection caused the suspension of cyclic activities in infected heifers, while Sekoni et al. (1988) observed that T.vivax infection affect the reaction time of semen collection, with progressive

difficulty of obtaining ejaculates due to the stress of infection and damage to the genitalia. Anosa and Isoun (1980a) have associated trypanosome infection in sheep and goats with variable degrees of testicular degeneration and resultant disappearance of spermatozoa population in the epididymis. Sekoni (1993) reported significant deterioration in semen characteristics of sheep infected with T. vivax. He observed that this is accompanied by decreased mean volume, increased sperm morphological abnormalities and decreased sperm concentration per ejaculate, probably due to testicular degeneration associated with bouts of hyperthermia. Akpavie et al. (1986) found that T. vivax infection severely affected the quality of the ejaculate with a reduction in sperm motility and concentration, an increase in the percentage of dead spermatozoa and an abnormal sperm morphology. Sekoni et al. (1991) observed that infertility problems as a result of elevated sperm morphological abnormalities do persist in cattle, several weeks after physical recovery following chemotherapy. Ogwu (1983) reported abortions in zebu cattle. The effects of these parasites on pregnancy and reproductive organs are quite devastating, hence animal productivity will be reduced greatly.

2.4 Terminal Changes in Animals Infected with T. vivax

Ruminants in the terminal stages of infection are usually comatose and show paddling of feet. Terminal temperatures are either elevated or depressed (Anosa, 1977) while pulse and respiratory rates are depressed and anaemia is reported to be at its peak (Anosa and Isoun 1983). Serum biochemical changes associated with the

terminal phase include hypoglycaemia, hypophosphataemia, increased non-protein nitrogen and blood urea, decrease in sodium and alkaline phosphatase with severe proteinuria (Antia, 1981). Losos (1986) observed in cattle infected with T. vivax at post-mortem, gross lesions such as extensive haemorrhages on the serosal and mucosal surfaces of the buccal cavity, larynx and pharynx. The abomasum, small and large intestines, caecum, parietal and visceral peritoneum, pleural, epicardium, endocardium, lymph nodes, adipose tissue and skeletal muscles are also affected.

Masake (1980) reported that a consistent feature seen at necropsy in infected cattle is the marked generalised lymph node enlargement and splenomegaly. He attributed the enlargement largely to a marked lymphoid proliferative response and to an increase in the constituent cells of the mononuclear phagocytic system. He also observed some marked changes in the cardiovascular system in infected cattle. For instance, in severely affected animals, hydropericardium occurred and there was evidence of congestive heart failure (dilated decompensated heart accompanied with pulmonary oedema) and extensive subcutaneous oedema. He also noted a severe myocardial fibre degeneration and occasional necrosis, infiltration of lymphoid cells, macrophages, plasma cells and marked interstitial oedema. Masake (1980) also reported another finding of major potential significance, this is the occurrence of orchitis in T. vivax infected cattle. He observed that this is likely to cause infertility as it involves abnormal sperm formation in a trypanosome - infected bull adding further to the economic effects of trypanosomiasis.

2.5 Anaemia

Anaemia is one of the most consistent features of trypanosomiasis and it is the most common and significant pathological feature of natural and experimental T. vivax infections of ruminants (Murray, 1978; Anosa, 1983). The anaemia is due to reduced red blood cells, their haemoglobin content and packed cell volume. Murray (1978) observed that the cardinal sign and major disease promoting factor of both human and animal trypanosomiases is anaemia. It sets in more or less acutely with the first appearance of trypanosomes in the blood, depending on the species of trypanosome, the relative resistance of the host animal and its nutritional status. The importance of anaemia in the pathogenesis of trypanosomiasis varies in its degree with the species of the organism, the isolate of that species, the species of the infected host and the acuteness of the infection (Jenkins, 1980).

Anosa and Isoun (1976; 1980b) showed that anaemia in sheep and goats arose from accelerated extravascular destruction of red blood cells in the spleen, liver and lungs which is associated with decreased red cell survival. Esievo and Saror (1991) reviewed and noted that the anaemia in African animal trypanosomiasis is haemolytic in origin, occurring intra vascularly early in the acute phase, and also extra vascularly in the subacute and chronic course of the disease. They stated that haemodilution and bone-marrow depression are not significant reactions in the development of the anaemia, but erythrophagocytosis, haemolysis of erythrocytes either by haemolytic factors or by immunological means, and reduced erythrocyte life-span were advanced as being responsible.

Dargie et al. (1979a ; b) and Anosa (1983) observed that the onset of anaemia, determined by a drop in the PCV is closely correlated with the appearance, intensity and duration of parasitaemia. He further observed that by the second to third week of trypanosome infection, sharp drops in the red blood cell count and haemoglobin level develop, accompanied by an increase in the circulation of immature red blood cells, a few reticulocytes along with many macrocytes. He also stated that the number of white blood cells is reduced to about half the normal number due to reduction in the numbers of neutrophils and lymphocytes and that the number of circulating blood platelets also decreases early in the infection due to a shortened platelet life span.

2.5.1 Mechanisms of Anaemia in Animal Trypanosomiasis

Throughout the course of trypanosomiasis, anaemia persists as the cardinal sign, but its mechanisms appear to vary, depending on the presence or absence of trypanosomes. Although, most studies on the anaemia of African trypanosomiasis have considered the disease a static rather than a dynamic condition, the factors involved probably vary during the disease, which frequently can be protracted (Dargie, 1978). Dargie and D'Alesandro (1978) stated that there are three basic, but overlapping stages in the anaemia of African animal trypanosomiasis. The initial phase corresponds to the first parasitaemic peak, packed cell volume (PCV) values drop markedly, splenomegaly occurs, erythrocytes are destroyed rapidly, and there is an active, though limited erythropoietic response. They also observed that phase

two is the chronic phase of the anaemia, with little or no further drop in PCV values, but infected animals are stunted, the spleen size is variable, and there is marked hemosiderosis, particularly of the spleen, liver and bone marrow. They reported that the final phase of the anaemia is usually associated with complete or partial recovery.

Immunological explanations for anaemia also suggest that the immune system of infected host is also responsible for red cell destruction, either because the surface of the red blood cells is altered or the host produces antibodies to its own blood cells (ILRAD, 1984). In contrast, the close correlation of changes in circulating red cell volume, plasma volume, erythropoiesis and red cell removal from the circulation with the presence of parasitaemia, and the rapidity with which these parameters return to normal following treatment in the studies carried out by Holmes and Jennings (1983), clearly suggest that the anaemia is not immunologic in origin, but is as a result of the damage caused either directly by living trypanosomes or substances released by them.

In the review of the immunochemistry and immunopathology of animal trypanosomiasis by Esievo and Saror (1991), were highlighted some current concepts of the mechanisms of anaemia. They listed the causes of anaemia as haemodilution, haematopoietic response, erythrophagocytosis, immunoglobulin and complement, haemolysis, reduced erythrocyte life - span and sialic acid.

Haemodilution: The occurrence of haemodilution in animal trypanosomiasis remains to be proved, yet anaemia may be a reflection of haemodilution. Haemodilution is the process by which the blood volume is expanded, usually through increased plasma volume, without increasing red cell mass causing dilution of red cells (Maxie and Valli, 1978). The blood volume is maintained within a narrow range even when there is a wide fluctuation in PCV, because the plasma volume rapidly compensates for changes in red cell mass (Maxie and Valli, 1978). They also reported that plasma volume expansion occurred in T. congolense - infected calves and that haemodilution contributed to the development of anaemia. Anosa and Isoun (1976) also reported haemodilution in T. vivax infections of sheep and goats. Saror (1980) observed haemodilution in Red Sokoto goats infected with T. vivax as a significant contributing factor to the early development of anaemia. He also noted that in animals that developed acute trypanosomiasis, the rate of development of anaemia was too rapid for the host's haematopoietic system to be fully mobilised to produce sufficient erythrocytes to meet the blood's requirements for delivery of oxygen to vital tissues or organs, and this resulted in anoxia and sometimes death. Amole et al. (1982), also described haemodilution as a feature of the chronic phase in trypanosomiasis which serves as a compensation to the decreased red cell volume and is seen as a terminal phenomenon in trypanosomiasis. Anosa (1980), Whitelaw et al. (1980) and Amole et al.(1982) all reported increases in plasma volume in trypanosomiasis and this is usually accompanied by low levels of erythrocyte.

The contributory role of haemodilution in the anaemia of animal trypanosomiasis has been challenged by Jennings et al. (1974), Ingh et al. (1976) and Dargie et al. (1979a,b). The latter workers noted that 59Fe used to measure plasma volume by other workers did not equilibrate with the plasma space and seriously over-estimated the plasma volume of anaemic animals.

Haematopoietic Response: These responses in trypanosomiasis are both complex and variable and are influenced by many factors such as breed, sex, age and nutritional status of the infected animal as well as by the number and species of the trypanosomes (Dargie and D'Alesandro, 1978). Several researchers have claimed that the red-cell synthetic machinery is impaired in trypanosome-infected animals, yet others have suggested that erythropoiesis is accelerated at some stages that the possible reasons are apparent in the iron metabolism. As a consequence of their anaemia, the infected cattle lost between 40 and 55% of their circulating iron. This loss, combined with the presence of massive haemosiderin deposits within the mononuclear phagocytic system (MPS), especially of the spleen, liver, bone marrow, and haemal nodes, indicates defective iron utilization and raises the possibility that in severe or long-standing infections, the marrow is effectively starved of iron and the anaemia complicated by dyshaemopoiesis. Dargie and D'Alesandro (1978) concluded that the erythropoietic response in the infected animals is completely inadequate to cope with the high rates of red-cell breakdown.

Saror (1980) observed marked erythropoiesis in the bone marrow of affected goats and extra medullary haematopoiesis in the spleen. Dargie et al. (1979a) also reported a progressive iron accumulation in the bone marrow, defective iron incorporation into developing red cells and reduced red cell release from the marrow. Postmortem findings by Murray (1978) gave indications of continued red cell destruction, iron trapping and dyshemopoiesis and the femoral marrow is found to be yellow, gelatinous and inactive. The failure of bone marrow has been incriminated as a major factor in the development of anaemia in trypanosomiasis (Elhassan, 1987). Although Anosa and Isoun (1980 b) reported increased erythropoiesis in sheep bone marrow and development of erythroblasts islands in mice spleen in trypanosomiasis, the regenerative response is not commensurate with the degree of erythrocyte destruction (Dargie, 1978).

Erythrophagocytosis: Murray (1978) observed that one of the most striking features of bovine African trypanosomiasis is the expanded and active mononuclear phagocytic system (MPS) that develops soon after infection and continues throughout the disease. He further suggested that splenomegaly leads to red cell stasis within the spleen, prolonged contact with expanded and active MPS and hence increased erythrophagocytosis. Splenomegaly is marked, partially as a result of massive red-cell sequestration and erythrophagocytosis as well as striking lymphoid hyperplasia. Erythrophagocytosis is not confined to the spleen and is widespread throughout the liver, lungs, haemal nodes, bone marrow and circulation. Dargie (1978) carried out

some erythrokinetic studies of infected cattle, and found out that the abnormally high rates of red-cell breakdown was due to haemolysis. He observed that the mononuclear phagocytic system (MPS) was expanded and active, and erythrophagocytosis was marked, particularly in the spleen which was consistently enlarged. Erythrophagocytosis is reportedly found throughout the body, widespread hemosiderosis in the spleen, lungs, liver and bone marrow. He further stated that during the early parasitaemic phases, red-cell destruction is triggered by the trypanosomes and that immunologic and physiologic factors render the cells more prone to erythrophagocytosis by an expanded MPS. The phenomenon of phagocytosis of erythrocytes in T. vivax infected goats has been observed by Saror (1980).

In a review, Esievo and Saror (1991) listed, among the causes of erythrophagocytosis, the attachment of trypanosome antigen to the surface of red cells, their subsequent combination with anti-trypanosome antibody in the plasma and eventual sequestration by erythrophagocytosis. Mackenzie et al. (1978) demonstrated trypanosomal material on sheep erythrocytes by the indirect florescent antibody test and concluded that the phagocytosis of erythrocytes in T. congolense was due to the coating of the surface of red blood cells with trypanosomal antigen. Esievo (1979;1983) and Esievo et al. (1982) reported the production of sialidase (neuraminidase) by T.vivax and also the cleavage of erythrocyte surface sialic (neuraminic) acid during bovine trypanosomiasis. They suggested that due to the

cleavage, new antigenic sites would be revealed on affected erythrocytes, and that with time, during the course of the disease, antibody would be produced against these new antigenic sites on the erythrocytes to render them suitable for erythrophagocytosis. Katz et al. (1977) have shown that anti-RBC antibodies are formed in human patients following removal of sialic acid from the surface of erythrocytes. They observed that though these anti-RBC antibodies were incomplete and unbound, they may serve as opsonin and promote phagocytosis and destruction of erythrocytes by macrophages as happens with the Rh-factor mechanisms (Eisen, 1974).

Immunoglobulin and Complement: In the review by Esievo and Saror (1991), it was noted that since restriction of parasite growth is primarily mediated by immunological mechanisms, it seems likely that the immune response elicited by trypanosomes plays an important role in the aetiology of the anaemia. Assoku (1975) suggested that the anaemia was due to sensitization of erythrocytes with immune complexes resulting in erythrophagocytosis. ILRAD (1984) stated that plasma cells produce anti-trypanosome antibody necessary to eliminate the parasites, which forms an antigen-antibody complex on the surface of the erythrocytes thereby exposing the cells to phagocytic actions by macrophages. Assoku and Gardiner (1989) reported antibodies to normal erythrocytes and platelets in the plasma of infected cattle using the enzyme - linked immunosorbent assay (ELISA). They suggested that the two most likely causes for the induction of the auto-antibodies are

trypanosome - induced alteration of the surface molecules of blood cells and defect in the regulation of the immune system.

In severe infections, the onset of anaemia is related to the decline of the first wave of parasitaemia and the rise of serum antibody (Table et al., 1978), hence they concluded that the attachment of immune complexes to erythrocytes contributes to the anaemia in trypanosomiasis. Facer et al. (1982) showed that immune reactions on the surface of erythrocytes contributed to the anaemia of T. vivax infections in calves. Amole et al. (1982) showed the presence of immune complexes on the surface of erythrocytes of T. brucei - infected mice. Elhassan (1987) explained that these complexes attach to erythrocyte membranes, render them more susceptible to phagocytosis by macrophages, while the complement is cytotoxic to erythrocytes. There is no doubt that immune complexes play an active role in inducing anaemia during trypanosoma infection from the above observations.

Haemolysis: One of the principal causes of anaemia in trypanosomiasis is haemolysis, due largely to immune sensitization of the red cells followed in the later stages by reduced bone marrow production. A significant drop in packed cell volume (PCV) usually occurs within a few days of infection, coinciding with the first parasitaemia and subsequent progress of the anaemia parallels waves of parasitaemia. Boreham and Cox (1978) stated that the manifestations of cellular injury in trypanosomiasis are many and varied and arise in a number of different

ways. They noted that for many years, toxins may be involved in the pathology of trypanosomiasis, and listed three possible causes which are, a haemolytic factor that may contribute to the anaemia, phospholipases that may cause vascular endothelial damage and immunodepression, and complement-activating factors that may be responsible for some cell damage. Fiennes (1954) suggested the existence of haemolytic factors when he observed that red cells from normal bovine blood were haemolysed when incubated with plasma from trypanosomes infected animals. These haemolytic factors include fever, disseminated Intra vascular coagulation, direct trauma to red cells and activation of the splenic reticuloendothelial tissues.

Haemolytic Factors: Huan et al. (1975) showed that T. brucei produces a protein of molecular weight 10,000 Daltons that is capable of lysing red blood cells, and Tizard et al. (1978) observed that T. congolense, during autolysis generates phospholipase A and free fatty acids that can destroy red blood cells. The haemolytic factor described by Murray (1978) is a heat - stable, trypsin - sensitive substance with a molecular weight just less than 12,000 Daltons and isoelectric points in different preparations ranging from pH 5.0 to 5.5.

Fever: The occurrence of fever, often coinciding with a trypanolytic crisis is a well recognised feature of African trypanosomiasis. Red blood cells exposed to temperatures of a few degrees above normal body temperature for a few hours have been shown to have increased osmotic fragility, undergo accelerated haemolysis, and

have a shortened life span in vivo, which then shows that fever also plays a role in red cell damage and destruction (Murray, 1978).

Disseminated Intra- Vascular Coagulation (DIC): This can also lead to haemolytic anaemia. This is termed microangiopathic haemolytic anaemia in which the red blood cells are damaged by widespread fibrin deposition in the microvasculature. The erythrocytes then appear as distorted cells or schisocytes which are liable to lysis or phagocytosis (Wintrobe, 1975). Jenkins et al. (1974) suggested DIC as a mechanism of anaemia in African trypanosomiasis when he observed marked alterations in red cell morphology, including red cell fragments in T. brucei infected rabbits.

Direct Trauma to Red Cells: Using a cine - microphotographic technique with nomarski light interference, Jenkins (1980) was able to film a process of "pinching out" of red cells by adherent trypanosomes in wet preparations of infected mouse blood. Hence, he concluded that when parasitaemia is severe, injury is inflicted on the cells by the lashing action of the trypanosomes. Goodwin (1970) described the changes occurring in small blood vessels which became grossly abnormal and distorted, a situation very likely to affect the integrity of circulating red cells. In such circumstances, disseminated intra vascular coagulation may occur resulting in thrombocytopenia (Jenkins, 1980).

Activation of the Splenic Reticuloendothelial Tissues: Holmes and Jennings (1976) stated possible effects of the spleen as non - specific red cells removal due to the extended splenic vasculature with prolonged exposure of the red cells to an intra-splenic alien biochemical environment. In the spleen there is extravascular haemolysis of red cells which have been sensitized with antibody, with or without complement. Red cell pooling or sequestration occurs in the enlarged spleen exchanging only slowly with the general circulation, thus depriving the vasculature of a significant part of the red cell volume.

Reduced Erythrocyte Life-span and Sialic Acid: Sialic acids, the acylated neuraminic acids occur in appreciable quantities on the erythrocyte surface in several animal species like cattle and camel (Esievo et al., 1981 ; 1982). The rapid clearance of 51Cr-labelled erythrocytes, which were desialylated with Vibrio cholera neuraminidase, from the circulation of rats and rabbits, with sequestration by the liver, led to the suggestion that reduction in erythrocyte sialic acids was a mechanism of erythrocyte senescence and destruction (Durocher et al., 1975). Banks (1979 ; 1980) showed that T. congolense bound to bovine erythrocytes in vitro through the neuraminic acid receptors on the erythrocytes, leading to erythrocyte damage. Magaji (1975) reported that serum sialic acid was elevated in cattle infected with T. brucei and T. vivax. Esievo et al. (1982) reported slight but obvious increase in the free serum sialic acid concentrations in infected calves on day 8 post - infection and this coincided with the period of greatest erythrocyte surface sialic acid loss and peak parasitaemia.

Durocher et al. (1975) observed a reduction in the surface sialic acid of the erythrocytes of humans and animals and hence reduced negative surface charge with consequent reduction in electrophoretic migration which led rapidly to death of such cells by sequestration. Esievo et al. (1982) stated that early anaemia observed in infected animals may be attributable to the ability of the circulating trypanosomes to produce sialidase, which in turn cleaves off erythrocyte surface sialic acids. This, they noted, results in early removal of the erythrocytes from the peripheral circulation and thus rendering them more prone to phagocytosis by the reticuloendothelial system and this will account for the reduction of erythrocyte life-span which is a characteristic feature of animal trypanosomiasis.

One would have expected that the decline in erythrocyte surface sialic acid would be associated with a concomitant increase in free serum sialic acid. However, it was suggested that sialic acid removed from the erythrocyte surface was being metabolised or detoxified just as soon as it was produced (Esievo et al.,1982). They then postulated that as trypanosomes increase in numbers they produce neuraminidase which cleave off erythrocyte surface sialic acid, thus causing physicochemical damage to the erythrocyte surface and rendering them more prone to the haemolysis by the reticuloendothelial system. Also they stated that the cleavage of sialic acid from erythrocyte surfaces created new antigenic sites on the surfaces of the affected erythrocytes, with time, antibodies could then be produced against these new antigenic sites and further render the erythrocytes to phagocytosis.

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Esievo (1980, 1983) observed a casual relationship between loss of erythrocyte sialic acid and significant development of anaemia.

The above findings and those of Banks (1979 ; 1980) that T. congolense bound in vitro to bovine erythrocytes through the sialic acid receptors on the erythrocytes, leading to injuries of such erythrocytes, are further evidences for the significant roles of erythrocytes sialic acid and trypanosome neuraminidase in the pathogenesis of the anaemia of African animal trypanosomiasis. Jancik et al. (1978) observed that desialylated erythrocytes are rapidly opsonized by immunoglobulin and complement molecules for degradation by phagocytic cells of the liver and spleen, and this accounts for the reduction of erythrocyte life-span and the loss of erythrocyte sialic acid in T. vivax infected cattle led to suggestion that such loss attributed for the reduced erythrocyte life-span, which is a characteristic feature of animal trypanosomiasis (Esievo et al., 1982).

2.6 Immunosuppression in Trypanosomiasis

Animals infected with African trypanosomes are often severely immunodepressed (Pearson et al., 1978). All the trypanosomes cause changes in the cells and structure of the lymphoid system, in the level of serum immunoglobulin, and in functional immunity, although the degree varies from one host species to another. Infection with trypanosomes predisposes animals to other infections and interfere with their response to vaccinations (Rurangirwa et al .,1978). Suppression of immune

responses to heterologous antigens introduced after established infections of pathogenic African trypanosomes in various animal species has been reported by Ilemobade et al., (1982).

Among the reasons advanced for immunosuppression in animal trypanosomiasis are a breakdown in the cooperation between the cellular components of the humoral response, non-specific activation of the immunoglobulin producing cells leading to an inhibition of new responses and some impairment in suppressor T-cell control resulting in hypergammaglobulinaemia M (Mansfield 1978). Nantulya et al., (1982) proposed that although the hyperglobulinaemia is due to the production of specific antibodies against the numerous variable antigen types (VATs) that arise during trypanosome infections in cattle, immunosuppression may be due to antigen competition between the trypanosome and the heterologous test antigens. Structural and functional changes in the lymphoid organs of infected cattle have been reported by Moulton (1988), and an effective immune response depends on the presence of healthy lymphoid tissue. Low levels of serum protein impair the function of lymphoid tissues and therefore, hypoproteinemia may account, in part, for the poor immune response of some cattle to trypanosome infection. The lymphatic system does not work in isolation, but is related to the other haematopoietic cells. Thus, the increased demand for stem cells of the erythroid and myeloid systems may result in stem cell competition with a reduction in lymphoid proliferation (Karstad and Valli, 1978).

In the course of an infection, successive generations of trypanosomes display different antigens on their surface coats and these antigens are called variable surface glycoproteins (VSGs). The process whereby trypanosomes sequentially develop different antigens on their surface, thereby evading the immune response of the host, is known as antigenic variation and involves switching on and off the expression of a large family of genes which code for different VSGs. The expression of different VSGs during the course of infection is associated with rearrangements in the parasite DNA. Trypanosomes with different VSGs are called variable antigen types (VATs). Trypanosomes in the tsetse fly are not coated with variable antigens until they develop into the metacyclic forms which are transmitted to mammals. Metacyclic trypanosomes are thought to display a limited number of VSGs, while the parasites which develop in the bloodstream may display very large numbers, thus evading the immune responses of the host.

Sometimes the expression of a VSG gene is accompanied by the appearance of a duplicated copy of the gene at a new location in the genome (ILRAD, 1984). The extra copy disappears when the gene is no longer expressed. This type of rearrangement has been termed "expression - linked duplication". The surface antigen coat is probably the only component found in common on all African trypanosomes. The integrity of this structure is crucially important to the survival of the parasites in the host bloodstream. A thorough knowledge of how the VSGs are synthesised in the parasite and exported to the cell surface may suggest some form

of interference which will disrupt the stability of the surface coat and render the trypanosomes susceptible to host defence mechanisms.

2.7 Diagnosis

The diagnosis of trypanosome infections under field conditions can be difficult ; early detection of the disease is very important and could contribute towards control programmes of the disease in any affected community, apart from treatment. A positive diagnosis can be established by demonstrating the presence of the trypanosomes themselves. They may be found in the initial chancre, blood, lymph glands, the cerebrospinal fluid or the bone marrow by microscopical examination of blood samples or body fluids. Trypanosomiasis is usually diagnosed by repeated microscopic examinations of blood, glandular and cerebrospinal fluids or through animal inoculation techniques. However, it is sometimes very difficult to diagnose the infection by only demonstrating the presence of the parasites. The most commonly used serological tests for the diagnosis of African trypanosomiasis are the indirect fluorescent antibody test (IFAT), the immunodifussion test, the capillary passive haemaglutination test and the enzyme-linked immunosorbent assay (ELISA). These serological techniques provide useful tools for indirect diagnosis of trypanosomiasis.

Some significant advances have been made in the diagnosis of trypanosomiasis recently. The techniques can be grouped into two:- parasitological and serological or immunodiagnosis. Various parasitological techniques have been developed and

adapted for diagnosis of trypanosomiasis and they involve concentration of the trypanosomes from blood and other biological materials. This could be done directly or after passage through anion-exchange medium or other substances through which red blood cells cannot permeate. Serological or immunodiagnosis depends on immunological changes that occur in the sera of infected animals as a result of the presence of the trypanosomes. Most of the techniques are based on either direct or indirect demonstration of antibodies produced by the host against the parasites or the trypanosome antigens. One of these techniques is the Micro-Enzyme-Linked Immunosorbent Assays (micro-ELISA). Engvall and Perlmann (1971) introduced the idea of using enzymes, conjugated with antigen or antibody to detect and measure antibodies or antigens in parasitic diseases. When purified antigens are used, the technique is sensitive and specific. Bocquentin and Durallet (1990) observed that simplicity and potential automation make the ELISA a universal tool for the detection of antibodies, and, more recently, of antigens.

2.8 Control

The search for a vaccine has proved more arduous than anticipated and the number of drugs available for therapy and prophylaxis is limited (Dolan, 1987). Due to the high cost and the variable results of conventional trypanosomiasis control methods, that is, chemotherapy and tsetse fly control, considerable attention has recently been focused on the exploitation of trypanotolerant livestock in tsetse-endemic areas of Africa. The concept of trypanotolerance was first used with special reference to the

breeds of cattle that showed a marked resistance to the ill effects of trypanosomiasis (Kanyari et al., 1986). D'ieteren (1994) observed that trypanotolerant livestock is a sustainable option for increasing livestock production in tsetse-infected areas.

2.9 Importance and Characteristics of Small Ruminants

In developing countries of the world, where protein - calorie malnutrition is a wide - spread phenomenon, the quantity and quality of protein available to the people has to be increased in order to meet the nutritional requirements of the people. Food and Agricultural Organisation (1983), observed that the projected protein consumption was 59.5g and out of this, 13.0g was of animal origin, and that livestock provided only 20% of human protein consumption from 1978 to 1980. The low production of animal protein is due, among other things, to animal diseases, of which animal trypanosomiasis is an important disease which is transmitted by tsetse flies of the genus Glossina. Livestock account for one third of Nigeria agricultural GDP, providing income, employment, food, farm energy, manure, fuel and transport. They are also a major source of government revenue.

Traditional livestock production in Nigeria is varied and complex. Livestock, especially ruminants, are the most efficient users of uncultivated land and can contribute substantially to crop production by providing manure through their waste. In Nigeria, small ruminants contribute an estimated 35% to the total meat supply

(Ngere et al ., 1984). Goats play an important role in many different agricultural systems in sub-Saharan Africa. They are particularly suited to traditional systems since their small size and lower feed requirements allow their use by poorer farming communities. They are also able to occupy a wide range of ecological habitats, including semi-arid, sub-humid and humid regions. They can make optimal use of relatively low quality vegetation and their habits are complementary to the raising of cattle (Luckins, 1992).

Small ruminants which include goats, sheep, llamas and alpacas are animals that eat low quality food, particularly fibrous vegetation, which cannot be eaten by humans and non - ruminants, such as pigs and poultry. They are kept as livestock because they produce meat, milk, wool or hair, skins and manure. Although goats and alpacas sometimes produce high - quality fibre, sheep are the only species of animals that produce wool, therefore these animals are capable of converting poor quality food into desirable products. They are also a form of investment, for example, in countries where there are no banking facilities and individual farmers do not own land, wealth is stored in the form of livestock. Investment in these animals avoids losses due to high inflation rates found in the unstable economies of many developing countries, and they can be sold quickly when cash is needed (Gatenby, 1991). There are several advantages of keeping small ruminants rather than large ruminants such as cows or buffalo. These advantages are low cost, little food needed, products in manageable quantities, low risk of total loss and high reproductive rate.

The cost of a goat is much less than that of a cow, and a small holder may be able to afford to have one or two goats but not a cow. A few goats can be kept on a limited area of land, and each animal needs only a small amount of food, hence they can be kept where there is not enough food for a large ruminant. They give outputs in amounts that are suitable for local consumption and the meat from a goat can be sold in one day by a village butcher. If a farmer keeps goats rather than large ruminants, he reduces the risk of total loss of his animals. Devendra and Mcleroy (1987) observed that goats are important animals in tropical livestock production system. In the subsistence sector, pastoralists and agriculturists often depend on them for much of their livelihood. Although goats are widely distributed in the tropics and subtropics, they noted that they remain neglected resources, and the important role of this specie in tropical agriculture they said, is inadequately understood.

According to Gatenby (1991), goats, as small ruminants have a high reproductive rate. This is important in an unfavourable environment where every now and then the numbers of animals are reduced by the drought ; when the environment becomes favourable again, the number of goats builds up quickly, but the number of large ruminants like cows stays for several years. Poor people as well as rich people own goats whereas it is usually the rich who have cattle. A programme to improve the productivity of cattle will therefore help only the rich, but a programme to improve goats will be of much wider benefit (Gatenby, 1991).

2.10 Geographical Distribution of Goats

Goats are widely distributed from arid semi -desert to humid rain - forest regions and represent 20.2 of the total populations of ruminant livestock in the tropics and sub-tropics (Luckins, 1992). The wide distribution of goats in the tropics and sub-tropics reflects their ability to adapt to a variety of environment. However, the preferred environment is on the lighter sandy soil, in the drier tropics, rather than in the wet humid tropics. In the dry tropics, they perform best and thrive in large numbers. The inherent characteristics of goats such as resistance to dehydration, preference for browse and wide - ranging feeding habits, enable them to thrive in regions that receive less than 750mm of rainfall (ILCA, 1989 ; 1990).

2.11 The Three Main Breeds of Goats in Nigeria

The goat is hollow-horned ruminant belonging to the mammalian order Artiodactyla, sub-order ruminantia, family Bocidae and either of the genera Capra or Hemitragus. The domestic goat belongs to the genus Capra (Devendra and Mcleroy, 1987). The three main breeds of goats in Nigeria are Savannah brown or Red Sokoto (RS), Sahel (Sh), and West African Dwarf (WAD). Nigeria has about 33% of all goats in Africa, and 52% of goats in the West Africa region (Ngere et al., 1984). Most households in the rural areas own a few goats. A survey by the federal office of statistics from 1965-1975 showed that the average number of goats per household owning stock in the southern parts of the country was then about four with a higher number for the northern savanna zones (Federal Ministry of Agriculture, 1981).

Except at government experimental farms, research institutes and universities, large herds of goats are rare. There are few commercial producers and communal ownership is equally rare. Ngere et al. (1984) observed that the commonest breed throughout the southern states is the West African Dwarf, while the Red Sokoto is the predominant and more widely distributed breed for the northern states.

2.11.1 Red Sokoto

This is also known as Maradi goat and is found mostly in Sokoto state but has spread Eastwards and Southwards through the Savanna belts of Nigeria (Fig.2). As they moved from the primary focus states, they became less uniform in size, colour and bone structure, lighter shades of red gave rise to the types described as the Kano brown, and further east, in Borno state, multicoloured and larger types are found (Ngere et al., 1979). Typically, this breed has a uniformly dark red - brown coat colour. The ears are short and horizontal. Horns are present in both sexes (Plate ia). Mature weight averages between 23 and 30kg.

2.11.2 Sahel

It is called the "desert goat" and is found in the Sahel region in the north of the country (Fig. 2). It is adapted to nomadic life and grazing in semi-desert environments. A large, long-legged breed with white, short and fine hair coat. The ears are long and both sexes have horns (Plate ib). Recorded mature weight ranges between 25 and 35Kg (Ngere et al .,1984).

2.11.3 West African Dwarf

This breed is found throughout the humid forest belts of Nigeria (Fig.2). It is variable in size and many local types have been noted, observed Ngere et al. (1984). When compared with the other two breeds, the most common type of this breed is thicker boned and has shorter legs. Black is the most common coat colour followed by white, brown and mixtures of all three. Some types have wattles while some do not. The ears are of medium length and horizontal. Horns are found on both sexes (Plate ic). The breed is noted for its hardiness. As one moves northwards from the forest belts, one encounters many transitional types which are characterised by their bigger size and varied coat colour. The typical mature weight of the commonest type according to Mack et al. (1985) is 20 - 25kg. Certain WAD breeds of goats and sheep, such as the Djallonke, show a degree of trypanotolerance comparable to that of the N'dama cattle of West Africa (Murray et al., 1984). These small ruminants are sometimes found in tsetse infested areas in which other domesticated livestock are unable to thrive and in spite of the occurrence of trypanosomiasis, fertility is unaffected (Mack et al., 1985).

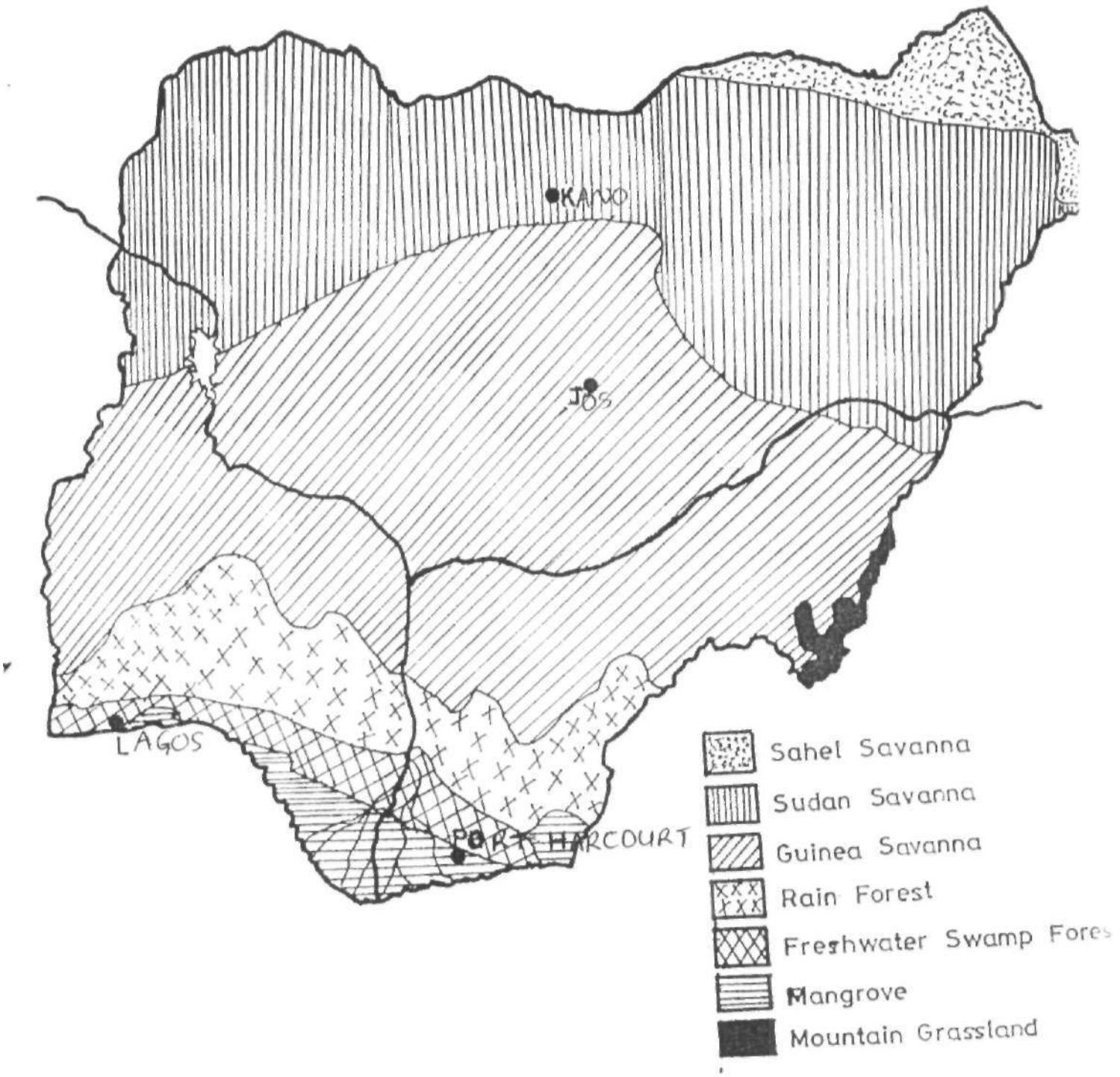


FIG. 2: NIGERIA NATURAL VEGETATION



PLATE ia: AN UNINFECTED RED SOKOTO GOAT



PLATE ib: AN UNINFECTED SAHEL GOAT



PLATE ic: AN UNINFECTED WEST AFRICAN DWARF GOAT

2.12 Reasons for Promoting Goat Production

The economic importance of goats depends on the value of their produce or services which include meat, milk, manures, fibres and skins among others. They may be kept as a source of investment and as an insurance against disaster. They are also used in ceremonial feasting and for the payment of social dues. They have a special and extremely important function as sacrificial animals amongst the followers of traditionalists in Africa, Western Asia and the Indian sub-continent, and in this context, the live animal has an intrinsic value far exceeding its value as a meat producer. Goats are also important as small experimental animals.

The rearing of goats provides a small, but nevertheless significant supply of animal protein in the form of milk and meat. This is particularly useful for the families of low-income farmers and landless labourers who cannot afford to purchase meat and milk (Devendra and Mcleroy, 1987). Federal Ministry of Agriculture (1981) estimated that goats contributed about 27% (65,000 tonnes) of the total meat supply. The skin of the RS is famous for its good quality leather which has excellent domestic and export value. In some parts of the country, for example, Kaduna, Kano and Adamawa states, goats are milked. In rural communities goats also serve a variety of socio-cultural purposes, quick source of cash, preparation of special dishes, sacrifices and other traditional ceremonies.

Human populations are growing very rapidly, creating a significant and increasing demand for additional animal protein foods. This demand can be met most easily by rapidly increasing the ruminant livestock population. Thus roughage feeds and crop by - products unsuitable for human consumption can be processed by the animals into desirable human foods. It is easier to increase the population of small ruminants such as goats than large ruminants such as cattle and buffaloes. This is because the capital investment for the farm is relatively low, average land holdings are usually small, the reproductive turnover of goats is high and they can be managed easily by family labour. The genetic variability within and between tropical goat breeds is so great that selection for improved animal productivity is possible and if this is combined with improved feeding and health, overall production gains could be considerable (Williamson and Payne, 1978).

There appears to be no doubt that goats will continue to play a very prominent role in the supply of animal protein to the rural communities in Nigeria in the foreseeable future, because of their hardiness, prolificacy and extensive distribution. In spite of their importance in the livestock economy of Nigeria, information on their performance and productivity remains scanty. Better knowledge of the factors that favour their production can greatly enhance their contribution towards alleviating protein intake deficit in the country. Comparative studies on host response to trypanosomiasis comprise an important research area. There is, however, little doubt that observations on laboratory - infected animal models which are carefully carried out, do provide valuable clues and collaborative evidence for the more difficult work pursued in field studies on larger animals and man.

Chapter 3

MATERIALS AND METHODS

3.1 Experimental Animals

Twenty four bucks made up of eight each of West African Dwarf, Red Sokoto and Sahel breeds were used. They were purchased from some villages that are known to be trypanosomiasis free, in Kujama local government area of Kaduna state.

3.2 Trypanosomes

The Trypanosoma vivax used was originally isolated from bovine and obtained from the Nigerian Institute for Trypanosomiasis Research (NITR). It was passaged twice in donor goats before inoculating the experimental animals with it.

3.3 Acclimatization

All the bucks were allowed to acclimatize for twelve weeks. During this period, their blood was routinely examined weekly for haemoparasites. However, they were treated with Terramycin LA (Pfizer, Nigeria) 1ml / 10kg body weight for bacterial infection, Berenil (Bayer, Germany) 3.5mg / kg body weight, for haemoparasites, sprayed with Azuntol (Bayer, Germany), for ectoparasites, and dewormed with Panacur (Hoechst, Nigeria) 0.75ml / kg body weight. They were housed in fly-proof pens, and fed on groundnut hay, commercial goat feeds (Livestock Feeds Limited, Kaduna), supplemented with maize bran and spent grains (Nigerian Breweries, Kaduna). They were also given cobalt-containing salt licks (Maclick, Wellcome, Nigeria) and water *ad libitum*.

3.4 Animal Inoculation

At the end of the acclimatization period, the twenty four bucks were divided into 3 groups of 8 Red Sokoto, 8 Sahel and 8 West African dwarf. Each group had 2 uninfected animals as control. Infected blood was taken from the donor goat with fulminating parasitaemia and was diluted with phosphate buffered saline glucose (PBSG : pH 8.0). The trypanosomes were counted and 1ml of blood containing approximately 2 x 10^6 trypanosomes was inoculated into each of the six animals in each group through the jugular vein.

3.5 Clinical Examinations

Both the control and infected animals were examined daily for the signs of the disease after inoculation.

3.6 Temperature and Body Weight

Rectal temperatures were taken daily with centigrade thermometer throughout the period of the experiment between 7.00 and 8.00 a.m. Body weights were determined weekly using salter (R) model-250 top loading weighing scale.

3.7 Parasite Enumeration

After inoculation, trypanosomes were detected in the blood by the buffy coat / dark ground method (Murray et al ., 1977) and the number of trypanosomes evaluated using the improved Neubauer haemocytometer (Hawksley, England), and were then converted to the Logarithm Equivalent Values (LEV).

3.8 Haematological Parameters

Blood was collected from the jugular vein of all the goats using a 21 gauge needle with 5ml syringe. One ml of blood was collected weekly into labelled vacutainers containing Ethylene Diamine Tetracetate (EDTA) as anticoagulant four weeks before the animals were inoculated with T. vivax for the determination of these haematological parameters which are the erythrocyte counts, total leucocytes counts, differential leucocytes, platelet counts(PT). For the estimation of packed cell volume(PCV), peripheral blood was collected from the ears of all the goats using heparinised capillary tubes weekly, four weeks before the animals were inoculated with the parasite. The RBC and WBC counts were done by the standard method using an improved Neubauer haemocytometer (Hawksley, England) while differential white blood cell counts were done by the examination of giemsa - stained blood smears and differentiating 100 leukocytes (Schalm et al., 1975).

Following inoculation, PCV was estimated daily for two weeks and every other day thereafter and was determined by the micro haematocrit centrifugation technique. The other parameters were measured weekly post-infection. Three ml of blood was collected into clean tubes without anticoagulant every other day for serum, two weeks pre- and post-inoculation for free serum sialic acid assay.

3.9 Free Serum Sialic Acid Assay

Free serum sialic acid was assayed by the method of Aminoff (1961). Half one mililitre of serum was added into Gibco tubes with screw cap. 0.5ml distilled

water was added into a separate Gibco tube with cap to serve as blank and 0.5ml of the standard was added into another tube with cap. 0.25ml of periodate reagent was then added to all the tubes and they were shaken. All the tubes were incubated in a water bath at 37°C for 30 minutes. They were removed from the water bath and 0.2ml Sodium arsenite solution was added to all the tubes and allowed to stand for two minutes for the mixture to turn colourless,(the sodium arsenite was to remove excess periodate),and were all shaken. Two ml of thiobarbituric acid was added to all the tubes and were covered. The tubes were then put in boiling water for 7.5 minutes, after which the mixture turned pink. The tubes were removed and put into iced water to cool. The colour (chromophore) was extracted with acid butanol by adding 5ml acid butanol to all the tubes and shaken vigorously. Very quick centrifugation was done at 3,100 revolution per minute (rpm) for 3 minutes at 25^0C. The top layer was aspirated with pasteur pipettes into glass cuvette and the absorbances of the samples and standard were read at 549nm against blank with the aid of a spectrophotometer.

3.10 Bone Marrow Biopsy and Examination

One week before infection, and after establishing infection between 21 and 30 days when the PCV was appreciably low in the infected goats , shortly before treatment, bone marrow aspirates were obtained from all infected and control goats through the femoral head by using sterilised 14 gauge bone marrow needles. The head of the femur was located by palpation and the skin over the site was shaved, disinfected with alcohol and the site injected with lignocaine so as to minimize the amount of stress to which the animals were subjected. The

femur was immobilised, with the stylate in place, the bone marrow needle was passed through the disinfected skin to the head of the femur. With constant pressure and gentle twisting motion, the needle was introduced into the marrow cavity. A 5ml syringe was then attached after the stylet had been removed and the marrow was aspirated and immediately transferred to clean glass slides for squash smears. The aspirated marrow was air dried, fixed and stained with Wright-Leishman's stain (Jain,1986). A differential marrow cell count based on 500 nucleated cells was made. The myeloid : erythroid ratio was then calculated.

3.11 Postmortem

The recumbent animals were euthanised and post-mortem examinations carried out.

3.12 Treatment

The infected animals were treated with Berenil (Hoechst, Germany) 3.5mg/kg body weight, when the PCV became very low and recumbency was observed.

3.13 Statistical Analysis of the Data Collected

Inter-group comparison of post-infection response of the three breeds of goats was carried out using analysis of variance (ANOVA).

Chapter 4

RESULTS

4.1 The Clinical and Parasitological Features of T. vivax Infection in Red Sokoto (RS), Sahel (Sh) And West African Dwarf (WAD) Goats

4.1.1 Discharges from External Orifices

Red Sokoto : During the second week of infection, goats numbers 759, 258 and 234 had mild bilateral watery ocular discharge. They also had mild watery nasal discharge including goat number 266. By the third week of infection, goat number 759 was the only one with mild mucoid ocular and nasal discharges, with persistent cough and at the end of the third week, goats numbers 234 and 258 had mild watery nasal discharge which soon disappeared after treatment.

Sahel: From the end of the second week to the middle of the fourth week, goats numbers 727 and 731 had mild mucoid ocular and nasal discharge. Goat number 726 had watery ocular discharge by the fourth week of infection.

West African Dwarf: No discharge from the external orifices was seen in any of the infected animals throughout the period of infection.

4.1.2 Pale Mucous Membrane

Red Sokoto: Three weeks into the infection, all the goats became generally dull with pale ocular mucous membrane. Following treatment, the ocular mucous membrane gradually assumed a pink colour, only to become pale from the fourth week post - treatment.

Sahel: By the end of the second week, all the goats became generally dull with pale ocular mucous membrane. The ocular mucous membrane was pale post-infection in goats numbers 726, 729 and 734 that were treated four weeks post-infection. Goats numbers 726 and 733 had sunken eyes.

West African Dwarf: At the end of the third week, all the infected goats had slightly pale mucous membrane. By the fourth week of infection, goats numbers 736, 737 and 740 had very pale mucous membrane. Towards the end of the fifth week, shortly before they were treated all the infected goats had very pale mucous membrane, were weak and dull. Two weeks post-treatment, the mucous membrane was pink in all the goats except goats numbers 736 and 737.

4.1.3 Appetite

Red Sokoto: They all maintained a steady appetite during the first 3 weeks of the infection, except goat number 759 which was off feed for 2 days and became recumbent three weeks post-infection. Goat number 234 was off feed, became emaciated and recumbent seven weeks post-treatment.

Sahel: The infected goats maintained almost a steady appetite during the period of the experiment except goats numbers 727 and 731 which were totally off feed by the fourth week post-infection and became recumbent.

West African Dwarf: The infected and control WAD goats maintained a steady appetite throughout the period of the observation.

4.1.4 Enlargement of Lymph Nodes

Red Sokoto: The superficial lymph nodes (prefemoral and prescapular) were enlarged in goats number 234, 4 weeks post - infection, while it is only the prescapular lymph nodes that were enlarged in goats numbers 759 and 250. Goats numbers 274, 266 and 258 had enlarged superficial lymph nodes fourth week post-treatment.

Sahel: The superficial lymph nodes (prefemoral and prescapular) were grossly enlarged in goats numbers 726, 729 and 733 four weeks post - infection.

West African Dwarf: The superficial lymph nodes (prefemoral and prescapular) were not palpable in the infected goats until the fourth week post-infection when they were observed to be enlarged in goats numbers 736, 737, 738 and 740. Third week post-infection, goats numbers 736 and 738 had slightly enlarged prescapular and prefemoral lymph nodes. Towards the end of the fifth week, all the infected animals had enlarged lymph nodes.

4.1.5 Depression

Red Sokoto: Four weeks post-infection, goats numbers 759, 234 and 258 were depressed, had wobbly gait, somnolent and stood with drooped heads. They later had difficulty in standing and braced themselves against the wall of the pen. Goat

number 759 (Plate iia) rose with difficulty after being prodded and was euthanised following 2 days of recumbency.

Sahel: Goat number 726 was very dull, depressed and stood with a drooped head (Plate iib) four weeks post-infection. Before it became recumbent, it suffered muscular tremor, and had mild watery ocular discharge.

West African Dwarf: Goat number 738 was weak and dull with slightly rough coat four weeks post-infection (Plate iic).

4.1.6 Rectal Temperature

Red Sokoto: The onset of parasitaemia was associated with increase in rectal temperature in all the infected goats. The temperature peak almost coincided with the first peak of parasitaemia in all the infected goats. As the parasitaemia waned, the temperature also dropped (Fig. 3). The highest temperature reached before death was 40.3° C by goat number 234 on week three post - infection. The infected goats had a mean temperature of 39.31 ± 0.03°C by the third week of infection. Thereafter, the temperature fluctuated until second week post-treatment, when the mean temperature fell to 38.13 ± 0.09°C. There was however a gradual increase in temperature from week five post - treatment, during which trypanosomes were found in the blood of the infected goats. The control goats had a mean rectal temperature of 38.62 ± 0.03°C.



PLATE iia: AN INFECTED RED SOKOTO GOAT FOUR WEEKS POST-INFECTION SHOWING WEAKNESS AND RECUMBENCY



PLATE iib: AN INFECTED SAHEL GOAT FOUR WEEKS POST-INFECTION SHOWING WEAKNESS AND SOILED HINDQUARTER



PLATE iic: AN INFECTED WEST AFRICAN DWARF GOAT FOUR WEEKS POST-INFECTION SHOWING WEAKNESS AND ROUGH HAIR COAT

Sahel: There was an increase in rectal temperature in all the infected goats as soon as parasites were detected in their blood. The temperature peak coincided with the only parasitaemic peak second week post-infection (Fig. 3). The mean rectal temperature had gone up to 39.15 ± 0.14°C from 38.54 ± 0.01°C which was the mean pre-infection rectal temperature. The mean rectal temperature for the control was 38.54 ± 0.02°C.

West African Dwarf: There was a gradual increase in the rectal temperature of the infected goats within the first three weeks of infection followed by a sharp increase with the peak coinciding with the second parasitaemic peak fifth week post-infection (Fig. 3). The mean rectal temperature being 40.30 ± 0.37°C fifth week post-infection from a mean pre-infection value of 38.49 ± 0.03°C. The control goats had a mean rectal temperature of 38.46 ± 0.02°C. However, there was a rise in the rectal temperature of the infected goats by the fourth week post-treatment, during which parasites were detected in the blood of the infected goats.

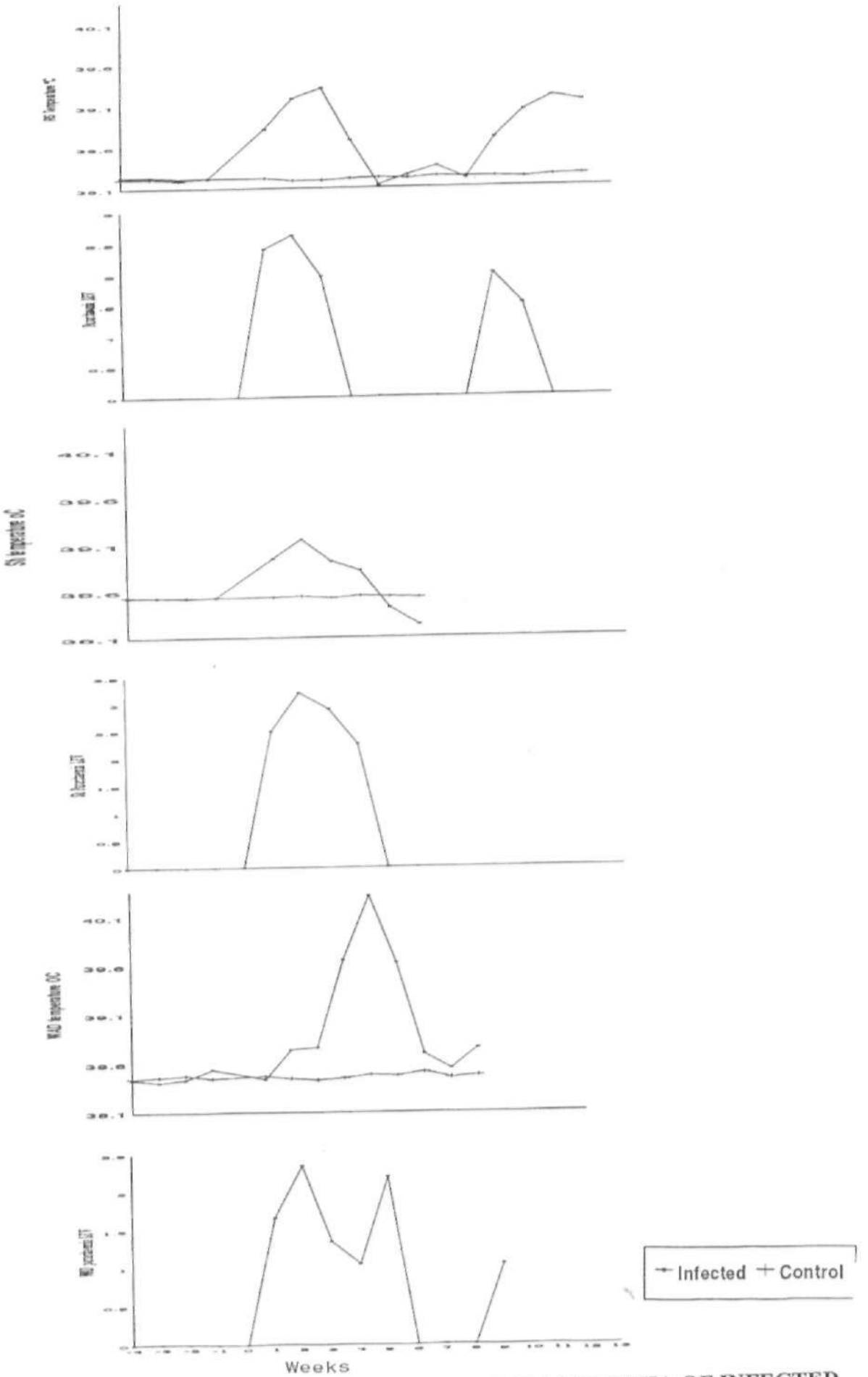


FIG. 3: RECTAL TEMPERATURE AND PARASITAEMIA OF INFECTED RED SOKOTO, SAHEL AND WEST AFRICAN DWARF GOATS

4.1.7 Body Weight

Red Sokoto: Loss in body weight was more noticeable after the third week post-infection in the infected goats. The weight dropped from the mean pre-infection value of 15.43 ± 0.02 kg to 14.44 ± 0.03 kg, 3 weeks post-infection (Fig. 4). The difference between the pre and post-infection values was not statistically significant ($p > 0.05$). The control goats had a mean body weight of 15.46 ± 0.02 kg.

Sahel: The mean body weight of the infected goats dropped slightly from the pre-infection value of 11.72 ± 0.02 kg to 11.68 ± 0.01 kg four weeks post-infection (Fig. 4). The difference between the pre and post-infection values was not statistically significant ($p > 0.05$). The control goats had a mean body weight of 11.74 ± 0.02 kg.

West African Dwarf: The mean body weight of the infected goats dropped from the mean pre-infection value of 10.14 ± 0.01 kg to 10.11 ± 0.01 kg by the fifth week post-infection (Fig. 4). The difference between the pre and post-infection values was not statistically significant ($p > 0.05$). The control animals had a mean body weight of 10.15 ± 0.02 kg.

4.1.8 Parasitaemia

Red Sokoto: All the infected goats showed parasites in the blood 3 to 5 days after intravenous inoculation with 2×10^6 Trypanosoma vivax. Parasites were first

recorded on day 3 post-infection in goat number 759 and on day 4 in goats numbers 286, 234 and 274 and on day 5 in goats numbers 266 and 258. The establishment of infection was followed by increasing parasitaemia. The first parasitaemic peak was very high and occurred second week post-infection (Fig. 5) which was followed by a gradual decline over the next two weeks. They were treated between the third and fourth week post-infection and the parasites disappeared from the blood until four weeks after.

Sahel: All the infected Sh goats showed parasites in the peripheral blood 4 to 5 days after intravenous inoculation with 2 x 10^6 T. vivax. Parasites were first recorded in goats nos 731 and 733 on day 4 post-infection. By day 6 post-infection all the other infected goats were positive for T. vivax. Soon after parasites were detected in their blood, the parasitaemia continued to rise and the only parasitaemic peak was reached on week two post-infection (Fig. 5). The two surviving animals were treated after four weeks of infection and this was followed by a gradual decline until the parasites could not be detected again.

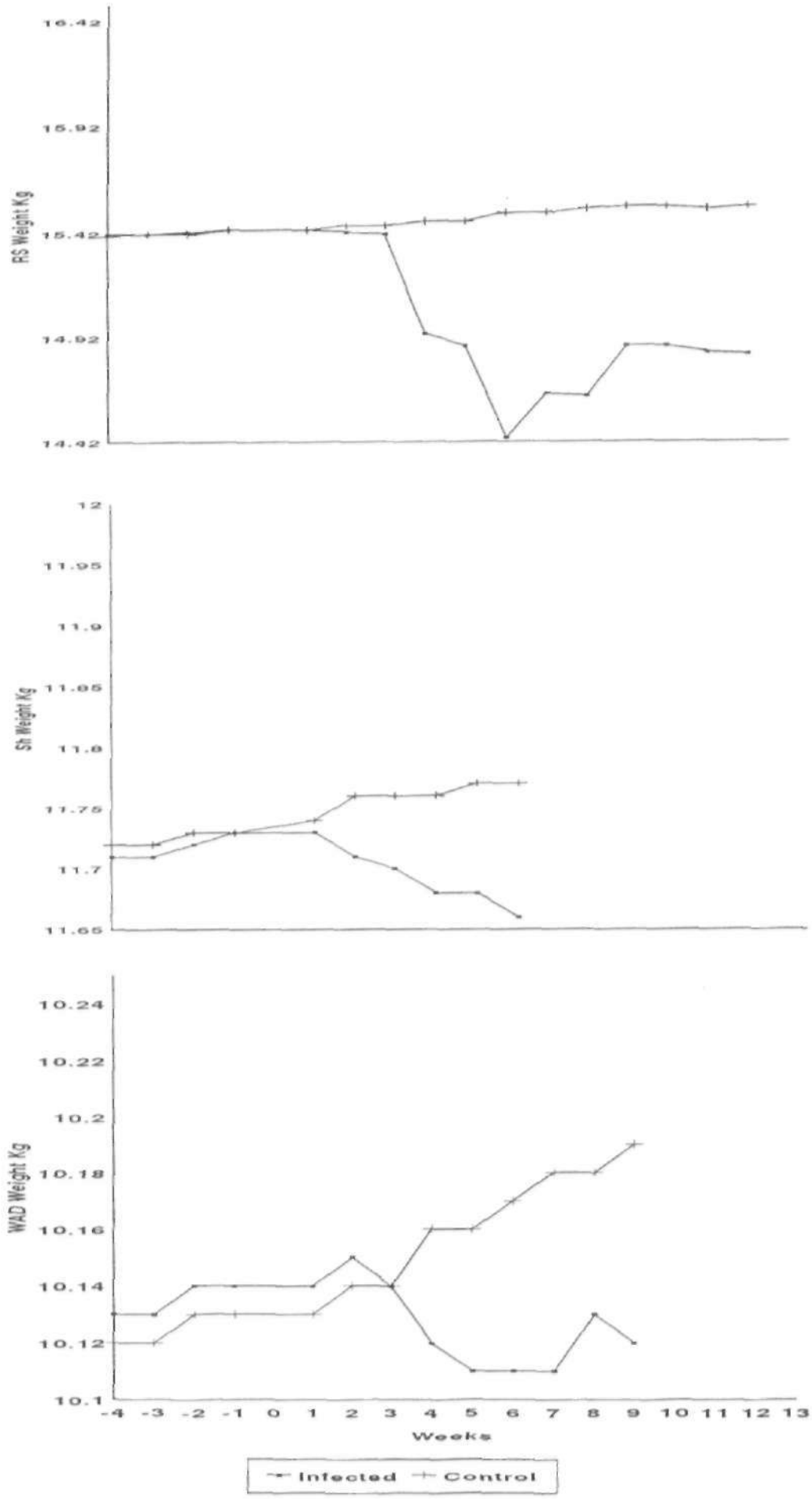


FIG. 4: BODY WEIGHT OF INFECTED RED SOKOTO, SAHEL AND WEST AFRICAN DWARF GOATS

West African Dwarf: All the infected goats showed parasites in the peripheral blood 3 to 5 days after intravenous inoculation with 2 x 10^6 Trypanosoma vivax. On day 3 post-infection, parasites were first recorded in goats numbers 739 and 742, followed by goats numbers 736 and 738 on day 4, while parasites were recorded in goats numbers 737 and 740 on day 5. After the parasites were detected in the blood of the infected animals, parasitaemia started to increase till the first parasitaemic peak was observed on week two post -infection. A gradual decline in the parasitaemia was observed till fourth week post-infection. Thereafter, parasitaemia started to build up and the second peak followed one week after, which was five weeks post-infection (Fig. 5). Following treatment after five weeks of infection, parasites were not detected in the blood. However, parasites were again detected in the blood of the infected goats between the third and fourth week post-treatment.

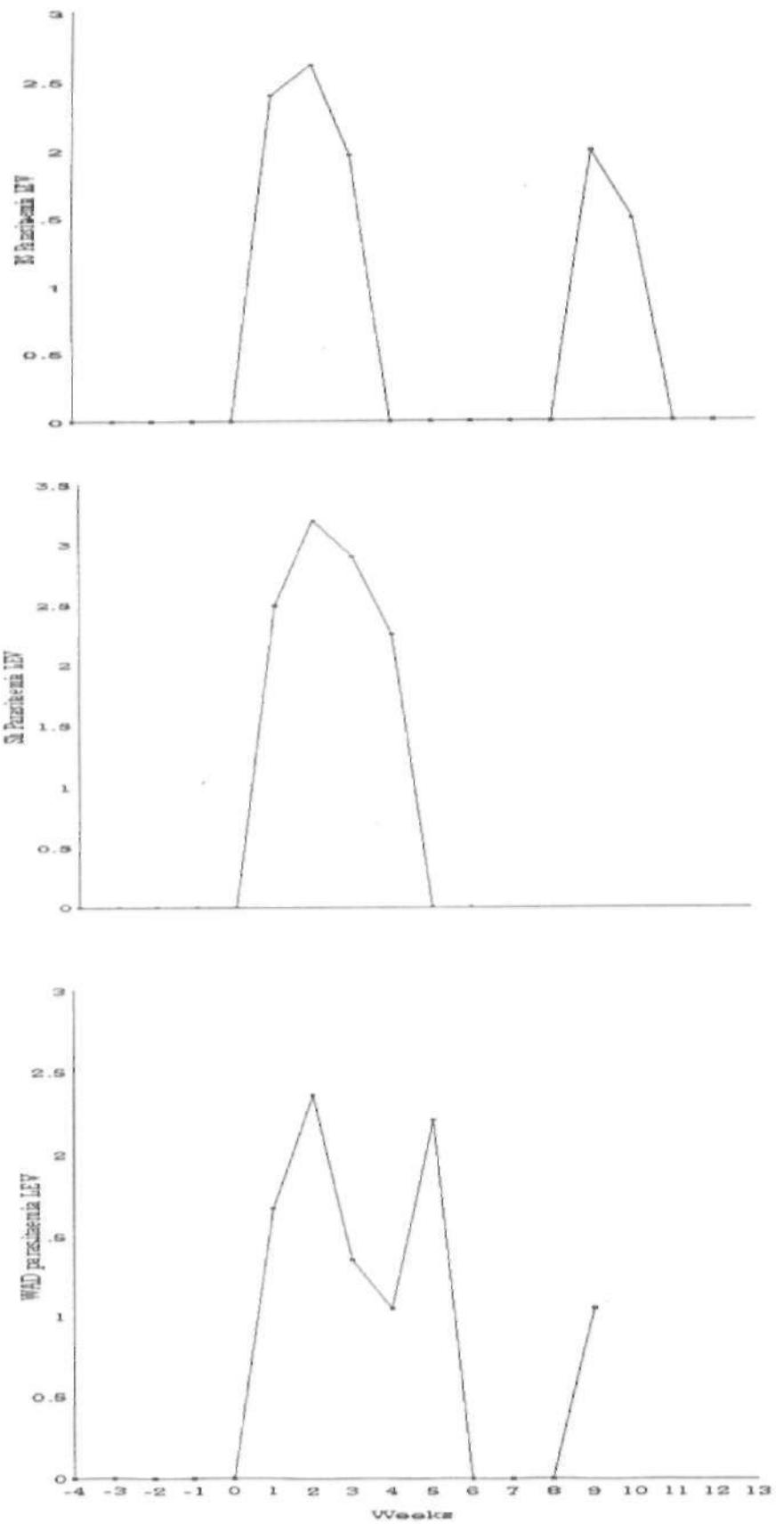


FIG. 5: THE PARASITAEMIA (LEV) OF INFECTED RED SOKOTO, SAHEL AND WEST AFRICAN DWARF GOATS

4.2 The Haematological Response to T. vivax Infection by Red Sokoto (RS), Sahel (Sh) and West African Dwarf (WAD) Goats

4.2.1 The Erythrocyte Counts

Red Sokoto: The mean erythrocyte count of the infected animals dropped after infection. The mean pre-infection value of the infected goats was 5.44 ± 0.01 x 10^6 / µl. By the fourth week post-infection, the mean erythrocyte count dropped to 1.52 ± 0.02 x 10^6 / µl. A 72.06% drop below the mean pre-infection value was recorded at the end of the fourth week post-infection (Fig. 6). Between the fourth and sixth week post-treatment, the erythrocyte count increased to 3.28 ± 0.11 x 10^6 / µl, though this was below the mean pre-infection value. There was, however a further drop in the values until the end of the experiment while the mean erythrocyte count of the control animals was 5.75 ± 0.02 x 10^6 / µl.

Sahel: The mean erythrocyte count of the infected animals dropped from the mean pre-infection value of 5.28 ± 0.02 x 10^6 / µl to 1.72 ± 0.05 x 10^6 / µl second week post-treatment (Fig. 6). This resulted in 67.42% drop. The mean erythrocyte count values of the control animals was 5.31 ± 0.02 x 10^6 / µl.

West African Dwarf: The mean erythrocyte count of the infected animals fell from pre-infection value of 5.24 ± 0.03 x 10^6 / µl to 2.2 ± 0.01 x 10^6 / µl fifth week post-infection. A 58.01% drop below the mean pre-infection value was recorded. There was a recovery period of the values after treatment, but could not reach the

mean pre-infection value (Fig. 6). The mean erythrocyte count of the control animals was 5.29 ± 0.03 x 10^6 / µl.

4.2.2 The Packed Cell Volume (PCV)

Red Sokoto: At the onset of parasitaemia, all the infected animals had a drop in their PCV values, thereby bringing the mean PCV from the mean pre-infection value of 30.76 ± 0.32 % to the lowest mean PCV value of 14.25 ± 1.82% at the end of the fourth week post-infection. This showed a 53.63% drop. There was a gradual but unsuccessful attempt at recovery in the PCV from the first week till fourth week post-treatment (Fig. 7). The recovery in the values fourth week post-treatment to 22.25 ± 0.13%, was still below the mean pre-infection value of 30.76 ± 0.32%. This was followed by a gradual drop in the PCV value from the fifth week post-treatment, which continues till the end of the experiment. The mean PCV for the control animals was 31.54 ± 0.06% at the end of the observation.

Sahel: At the onset of parasitaemia, all the infected animals had a drop in their PCV values which began within the first week of infection (Fig. 7). The mean PCV value was 13.21 ± 0.02% by the second week post-treatment, giving a 57.06% drop from the mean pre-infection value of 30.76 ± 0.32%. The terminal PCV values of the two surviving goats two weeks post-treatment were 13 and 12% respectively. The mean PCV value for the control animals was 31.62 ± 0.51.

West African Dwarf: There was a gradual fall in the values of the PCV following the appearance of parasites in the blood of the infected animals (Fig. 7). The

lowest mean PCV value was 16.73 ± 0.11% and coincided with the second peak of parasitaemia which occurred on the fifth week post-infection. However, there was a gradual increase in the mean PCV values which was still below the mean pre-infection value of 31.65 ± 0.05 %. The mean PCV of the control animals was 31.84 ± 0.42%.

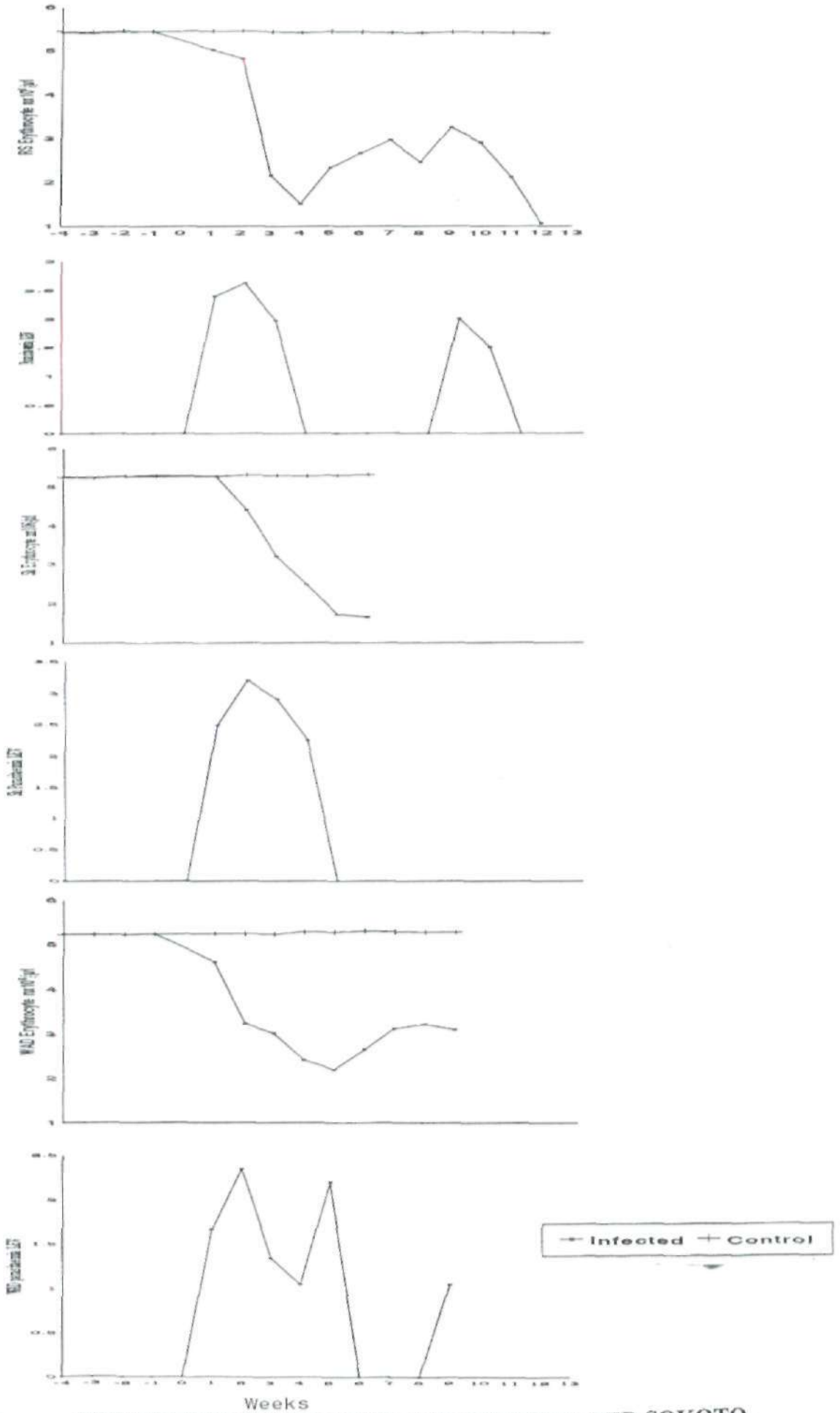


FIG. 6: THE ERYTHROCYTE COUNTS OF INFECTED RED SOKOTO, SAHEL AND WEST AFRICAN DWARF GOATS

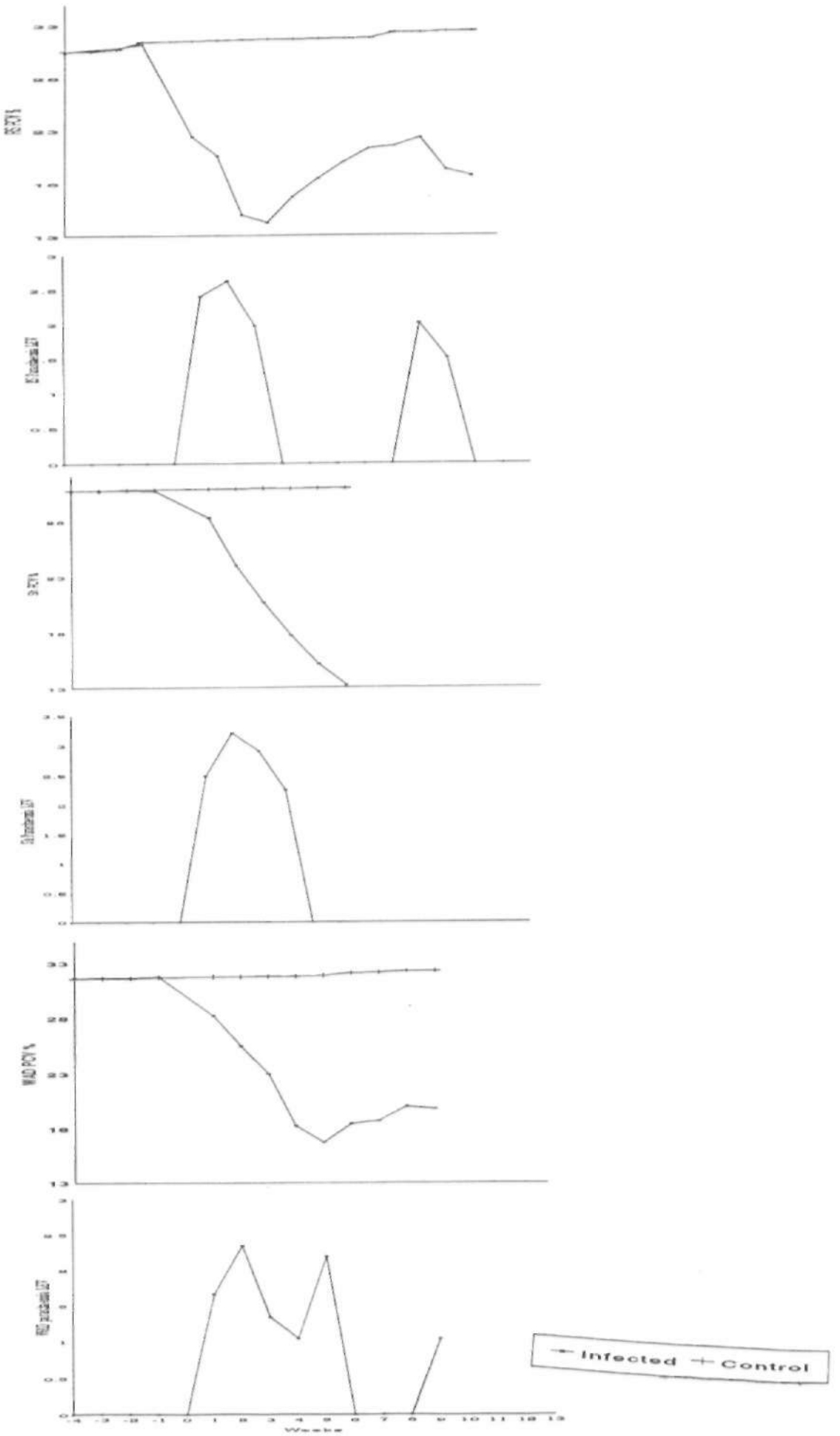


FIG. 7: THE PACKED CELL VOLUME OF INFECTED RED SOKOTO, SAHEL AND WEST AFRICAN DWARF GOATS

4.2.3 The Platelet Counts

Red Sokoto: The platelet counts of the infected animals fell from the mean pre-infection value of 2.13± 0.01x10^5 / µl to 1.07± 0.02 x 10^5 / µl three weeks post-infection(Fig. 8). This was followed by a mild recovery during the second week of treatment, in which the mean platelet counts rose to 1.7 ± 0.02 x 10^5 / µl. Thereafter there occurred a rise and fall in the values but did not reach the pre-infection level at the termination of the experiment. The control animals had a mean platelet value of 2.12 ± 0.01 x 10^5 µl.

Sahel : A fall in the platelet values for the infected animals began from the first week post-infection and reached the lowest value by the second week post-infection(Fig. 8). The drop was from the mean pre-infection value of 2.12 ± 0.01 x 10^5 / µl, to 1.86 ± 0.37 x 10^5 / µl. The control animals had a mean platelet counts of 2.09 ± 0.02 x 10^5 / µl.

West African Dwarf: There was a decrease in the platelet values of the infected animals from the mean pre-infection value of 2.03 ± 0.01x10^5 / µl to 1.6 ± 0.01 x 10^5 / µl three weeks post-infection(Fig. 8). This was followed by a sharp increase in the mean value of the infected goats which attained the pre-infection value by the third week post-treatment. The control animals maintained had a mean value of 2.03 ± 0.01 x 10^5 / µl.

4.2.4 The Total Leucocyte Counts

Red Sokoto: The mean total leucocyte counts of the infected animals fell gradually from the first week post-infection till the second week post-treatment. This was from the mean pre-infection value of 12.94 ± 0.02 x 10^3 / µl to 7.02 ± 0.03 x 10^3 / µl which was the lowest value reached during the infection period (Fig.9). Thereafter, there was a gradual increase to 10.93 ± 0.02 X 10^3 / µl by the end of the experiment. The control animals had a mean total leukocyte count of 12.86 ± 0.03.

Sahel: Total leukocyte counts fell in all the infected animals from the onset of infection coinciding with the rising wave of parasitaemia. There was a transient mild recovery at the end of the parasitaemic wave from the fourth week post-infection (Fig. 10). The mean pre-infection value was 12.37 ± 0.01 x 10^3 / µl, while by the end of the second week post-infection, it fell to 9.4 ± 0.03 x 10^3 / µl. The control animals had a mean total leukocyte count of 12.24 ± 0.02 x 10^3 / µl.

West African dwarf goats: There was a decrease in the mean total leukocyte count of the infected animals between the first and fourth weeks post-infection (Fig. 11). The mean pre-infection value was 12.59 ± 0.01 x 10^3 / µl. Four weeks post-infection, the mean value was 9.08 ± 0.02 x 10^3 / µl. Thereafter, the value began to increase but was still below the pre-infection value even at the end of the experiment while the mean total leukocyte count for the control animals was 12.48 ± 0.01 x 10^3 / µl.

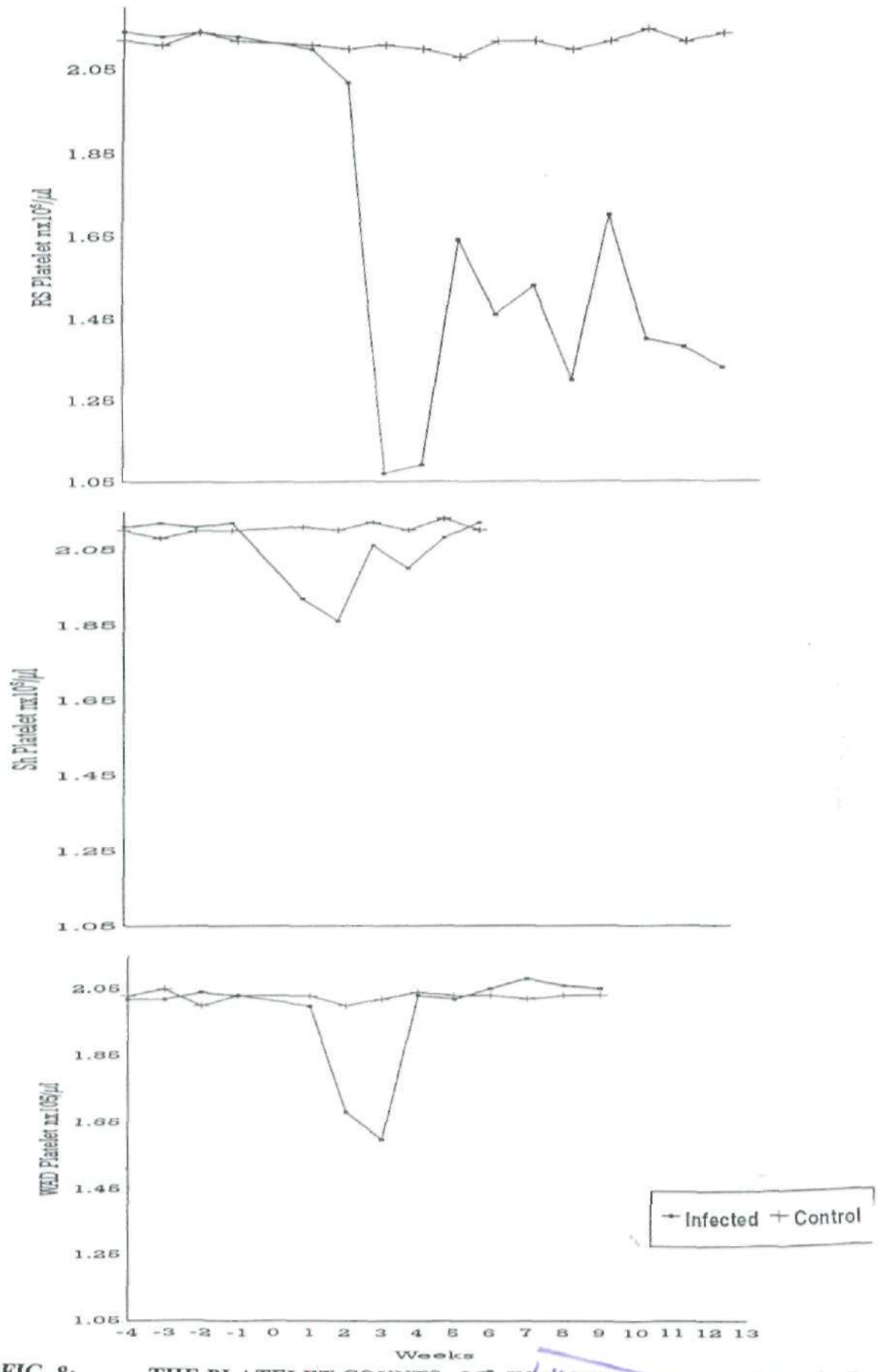


FIG. 8: THE PLATELET COUNTS OF INFECTED RED SOKOTO, SAHEL AND WEST AFRICAN DWARF GOATS

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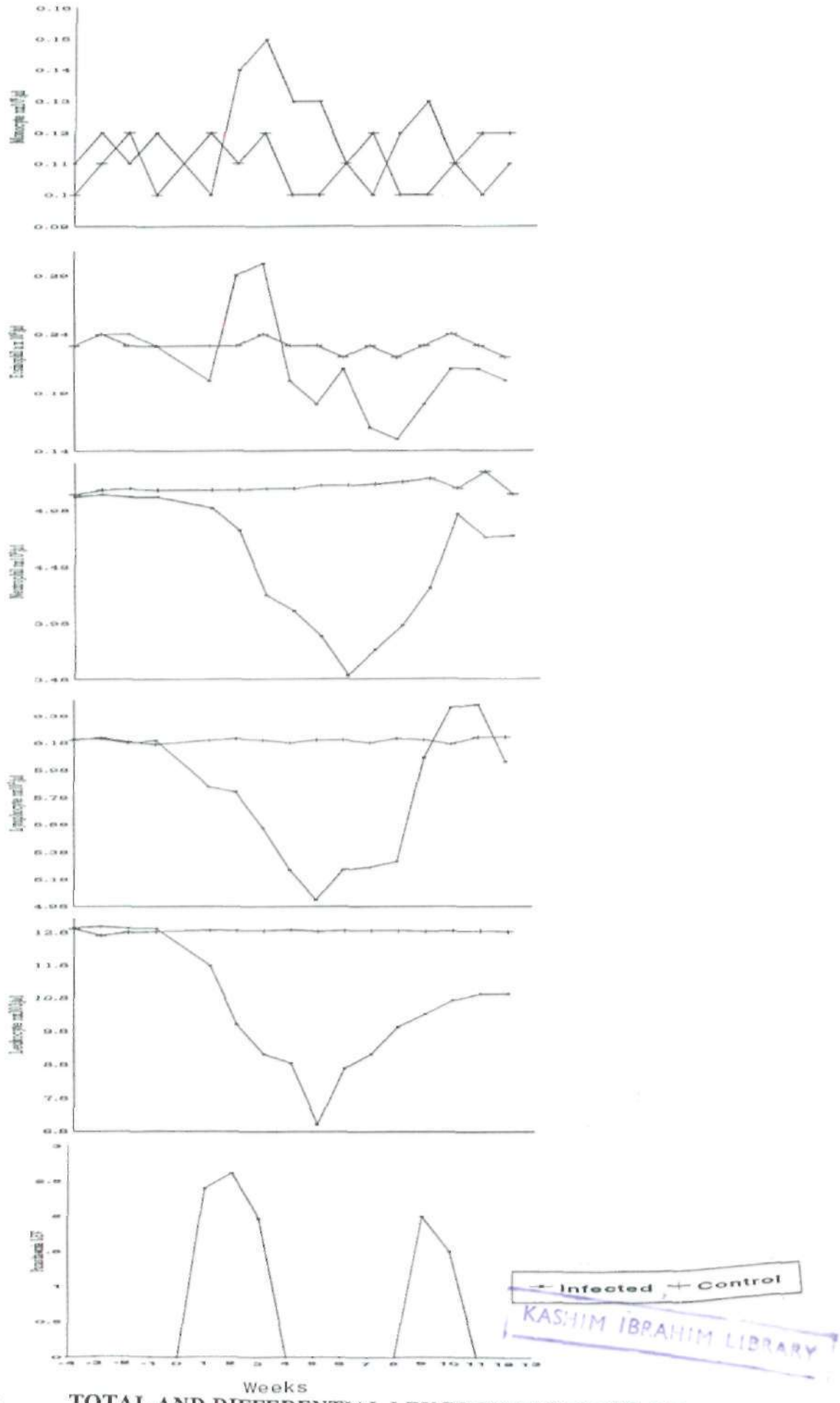
Differential Leucocyte Counts

4.2.5. Neutrophil counts

Red Sokoto: There was a drop in the mean absolute neutrophil counts in the infected animals which started by the second week of infection. The mean pre-infection value was 5.11 ± 0.01 x 10^3 / µl. The decrease was followed by a rise in the values but could not reach the pre-infection level till the end of the experiment (Fig. 9). There was a drop in the value by the third week post-treatment, thereby giving a 31.31% drop below the mean pre-infection value. The mean count for the control animals was 5.21 ± 0.03 x 10^3 / µl.

Sahel: There was a significant drop in the mean absolute neutrophil counts in the infected animals which occurred within the second and third weeks of infection (Fig. 10). The value dropped from the mean pre-infection value of 5.54 ± 0.02 x10^3 / µl to 3.33 ± 0.01 x 10^3 / µl three weeks post-infection. This gave a 39.89% drop below the mean pre-infection value. The control had a mean of 5.59 ± .05 x 10^3 / µl.

West African Dwarf: There was a drop in the mean absolute neutrophil counts of the infected animals which occurred within the first three weeks of infection rising thereafter till the end of the experiment. The mean pre-infection value was 5.13 ± 0.01 x 10^3 / µl, while by the fourth week post-infection, it fell to 3.0 ± 0.01 x 10^3 / µl. The mean value of the control was 5.12 ± 0.02 x 10^3 / µl (Fig. 11).



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FIG. 9: TOTAL AND DIFFERENTIAL LEUCOCYTE COUNTS OF INFECTED AND CONTROL RED SOKOTO GOATS

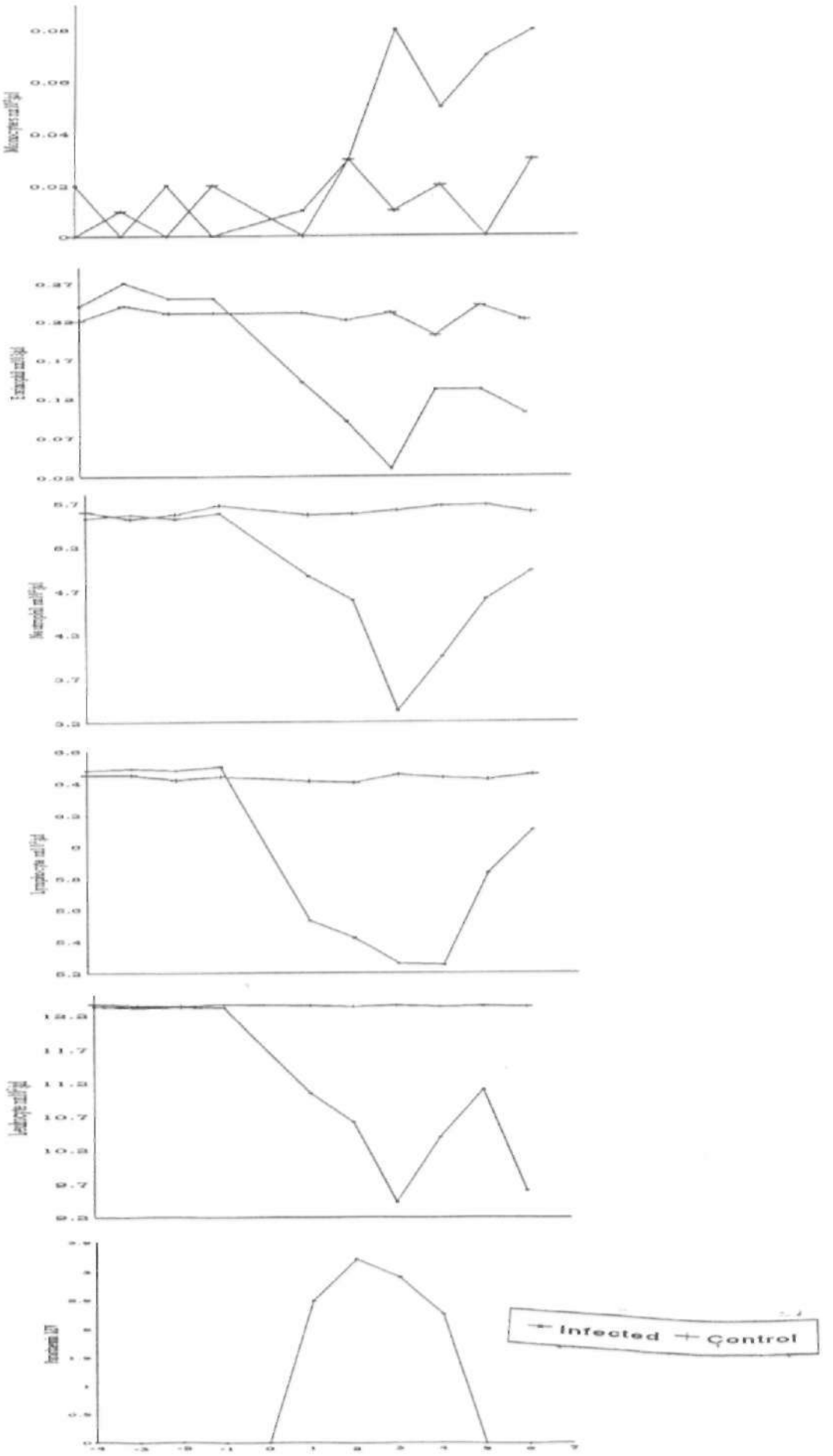


FIG. 10: TOTAL AND DIFFERENTIAL LEUCOCYTE COUNTS OF INFECTED AND CONTROL SAHEL GOATS

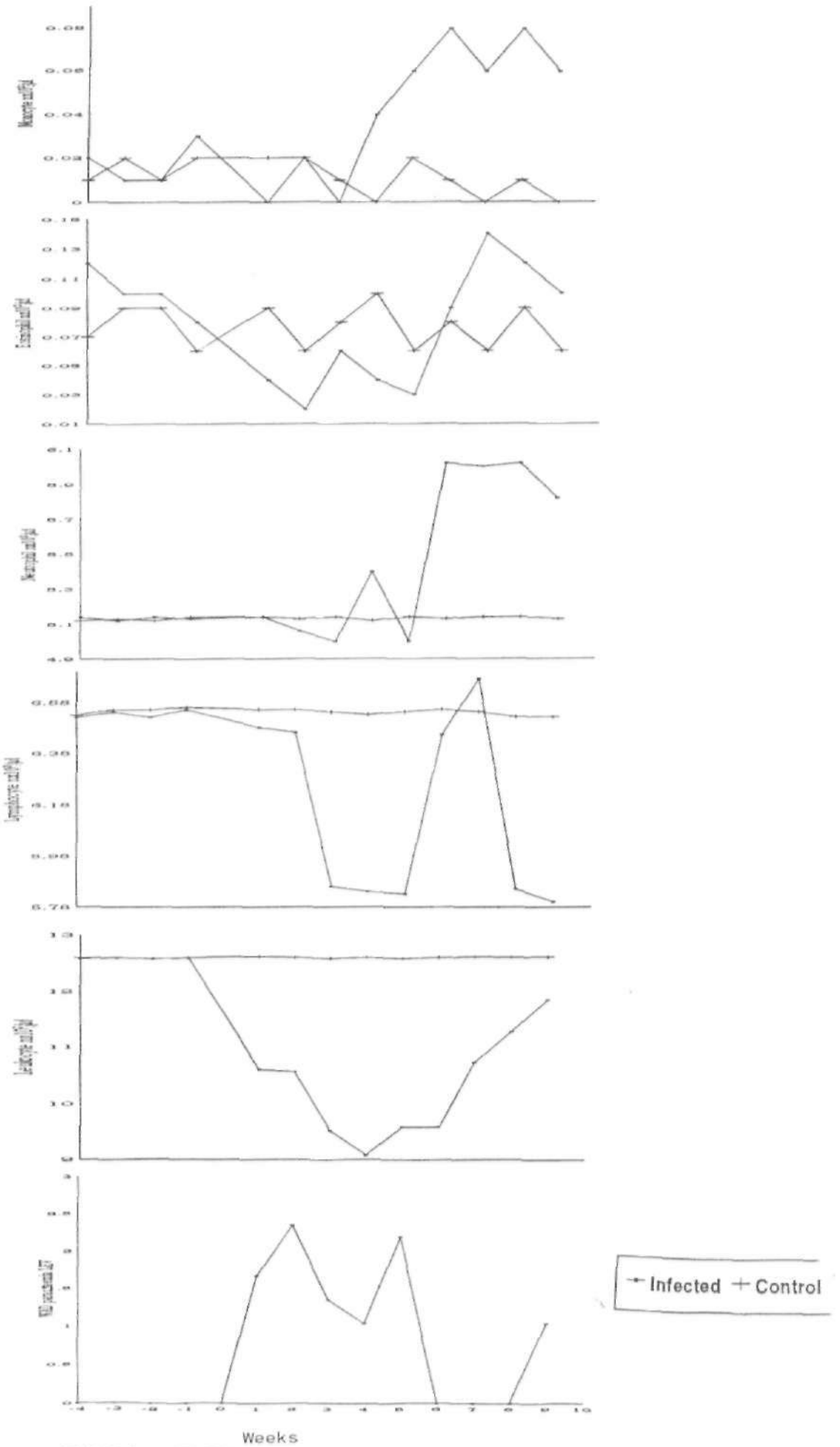


FIG. 11: TOTAL AND DIFFERENTIAL LEUCOCYTE COUNTS OF INFECTED AND CONTROL WEST AFRICAN DWARF GOATS

4.2.6 Lymphocyte Counts

Red Sokoto: There was a fall in the mean absolute lymphocyte count from the mean pre-infection value of 6.20 ± 0.01 x 10^3 / µl to 5.03 ± 0.02 x 10^3 / µl two weeks post-treatment (Fig. 9). Thereafter the value started to rise but could not reach the mean pre-infection value. The control animals had a mean value of 6.19 ± 0.03 x 10^3 / µl post-study.

Sahel: The mean absolute lymphocyte counts of the infected animals fell from the mean pre-infection value of 6.49 ± 0.01 x 10^3 / µl to 5.2 ± 0.02 x 10^3 / µl four weeks post-infection(Fig. 10). One week post treatment, it began to rise but could not rise to the pre-infection level before the experiment was terminated. The control animals had a mean value of 6.45 ± 0.02 x 10^3 / µl post-study.

West African Dwarf: The mean absolute lymphocyte counts of the infected animals fell from the pre-infection value of 6.53 ± 0.02 x 10^3 / µl to 5.46 ± 0.01 x 10^3 / µl second week post-infection. It started rising again by the third week and two weeks post -treatment, it rose above the pre-infection value to 6.67 ± 0.02 x 10^3 / µl (Fig. 11). The control had a mean value of 6.54 ± 0.02 x 10^3 /µl post-study.

4.2.7 Eosinophil Counts

Red Sokoto: The mean eosinophil count showed a slight fall within the first week of infection from the mean pre-infection value of 1.24 ± 0.01 x 10^3 / µl to 1.20 ±

0.01 x 10^3 / µl. There occurred a rise and fall in the value which continued till the end of the experiment. The control animals had a mean value of 1.34 ± 0.01 x 10^3 / µl (Fig. 9).

Sahel: There was a fall in the mean eosinophil count of the infected animals from the mean pre-infection value of 0.25 ± 0.01 x 10^3 / µl to 0.03 ± 0.02 x 10^3 /µl third week post-infection (Fig. 10). Thereafter, an intermittent rise and fall in the value was observed. The control had a mean value of 0.23 ± 0.01 x 10^3 / µl.

West African Dwarf: There was a drop in the mean values of the absolute eosinophil counts by the second week post-infection from 0.11 ± 0.01 x 10^3 / µl the mean pre-infection value, to 0.0 2 ± 0.01 x 10^3 / µl (Fig. 11). Thereafter, there was a gradual increase in the values which rose above the mean pre-infection value by the second week post-treatment. The control had a mean value of 0.08 ± 0.02 x 10^3 / µl.

4.2.8 Monocyte Counts

Red Sokoto: There was a fall in the mean post-infection values of the monocyte counts in the infected animals within the first week of infection. Then there occurred a rise in the mean value by the third week post-infection to 0.15 ± 0.01 x 10^3 / µl from the mean pre-infection value of 0.12 ± 0.01 x 10^3 /µl (Fig. 9). The mean monocyte count for the control was 1.02 ± 0.02 x 10^3 / µl

Sahel: There was a fall in the mean post-infection values of the absolute monocyte counts in the infected animals during the first week of infection (Fig. 10). The value then rose between the second and third week post-infection to 0.03 x 10^3 / µl from 0.00 x 10^3 / µl which was the mean pre-infection value. The control had a mean value of 0.01 ± 0.00 x 10^3 / µl.

West African Dwarf: There was a fall in the mean pre-infection values of the absolute monocyte counts of the infected animals by the third week of infection. This was from a pre-infection value of 0.02 ± 0.01 x 10^3 / µl to 0.0 ± 0.0 x 10^3 / µl three weeks post-infection. Thereafter there was a rise in the value above the pre-infection value (Fig. 11). The control had a mean value of 0.02 ± 0.00 x 10^3 / µl.

4.3 Free Serum Sialic Acid Concentration

Red Sokoto: There was an increase in the mean value of the free serum sialic acid of the infected animals within the first week of infection. The mean pre-infection value was 0.43 ± 0.01mg / dl (Fig. 12). By the fourth week post-infection, the value rose to 0.69 ± 0.01mg / dl. This resulted in an increase of 37.68% above the mean pre-infection value. However, this was followed by a drop in the mean value between the sixth and ninth week post-treatment which almost reached the pre-infection value. The mean values for the control was 0.43 ± 0.01mg / dl.

Sahel: There was a gradual increase in the mean value of the free serum sialic acid in the infected animals between the first and third weeks of infection (Fig. 12).

The mean pre-infection value was 0.39 ± 0.01mg / dl. Within two weeks of infection, the value rose to 0.48 ± 0.02mg / dl which was 23.08% above the mean pre-infection level. Thereafter, a gradual fall of the value was observed till the end of the experiment. The mean values for the control was 0.38 ± 0.02 mg / dl.

West African Dwarf: An increase in the value of free serum sialic acid in the infected animals was observed between the second and third week post-infection (Fig. 12). The mean pre-infection value was 0.32 ± 0.01 mg/dl. By the week post-infection, the value rose to 0.37 ± 0.01mg/dl thereby giving an increase of 5% above the mean pre-infection value. The mean values for the control animals was 0.32 ± 0.01 mg / dl.

4.4 Bone Marrow Cytology

Red Sokoto: The light microscopic examination of the pre-infection bone marrow smears of both the infected and control goats showed normal haematopoiesis with fewer number of normoblasts compared with the number of granulocytes(Plate iiia). The pre-infection myeloid : erythroid ratio of the infected goats was 1.63 : 1 (Appendix 1). Two weeks post-infection, there was a decrease in the granulocytic cells, mostly affected are the metamyelocytes, bands and segmenters, while there was a marked increase in the nucleated normoblasts (Plate iva) thereby giving a post-infection myeloid : erythroid ratio of 0.48 : 1 (Appendix 2).

Sahel: There was normal haematopoiesis in the animals before infection (Plate iiib). The pre-infection myeloid : erythroid ratio of the infected goats was 1.55 : 1 (Appendix 3). Following infection, there was a marked decrease in the granulocytes, especially the metamyelocytes and the bands within two weeks of infection (Plate ivb), while there was a marked increase in the basophilic normoblasts and the pronormoblasts of the erythrocytic cells giving a post-infection myeloid erythroid ratio of 0.52 : 1 (Appendix 4).

West African Dwarf: The pre-infection myeloid : erythroid ratio of the infected goats was 1.45 : 1 (Appendix 5) showing normal haematopoiesis (Plate iiic). By the end of the third week of infection, there was a marked decrease in the segmenters and neutrophilic myelocytes of the granulocytic cells with an increase in the nucleated normoblasts of the erythrocytic cells (Plate ivc) giving a post-infection ratio of 0.61 : 1 (Appendix 6).

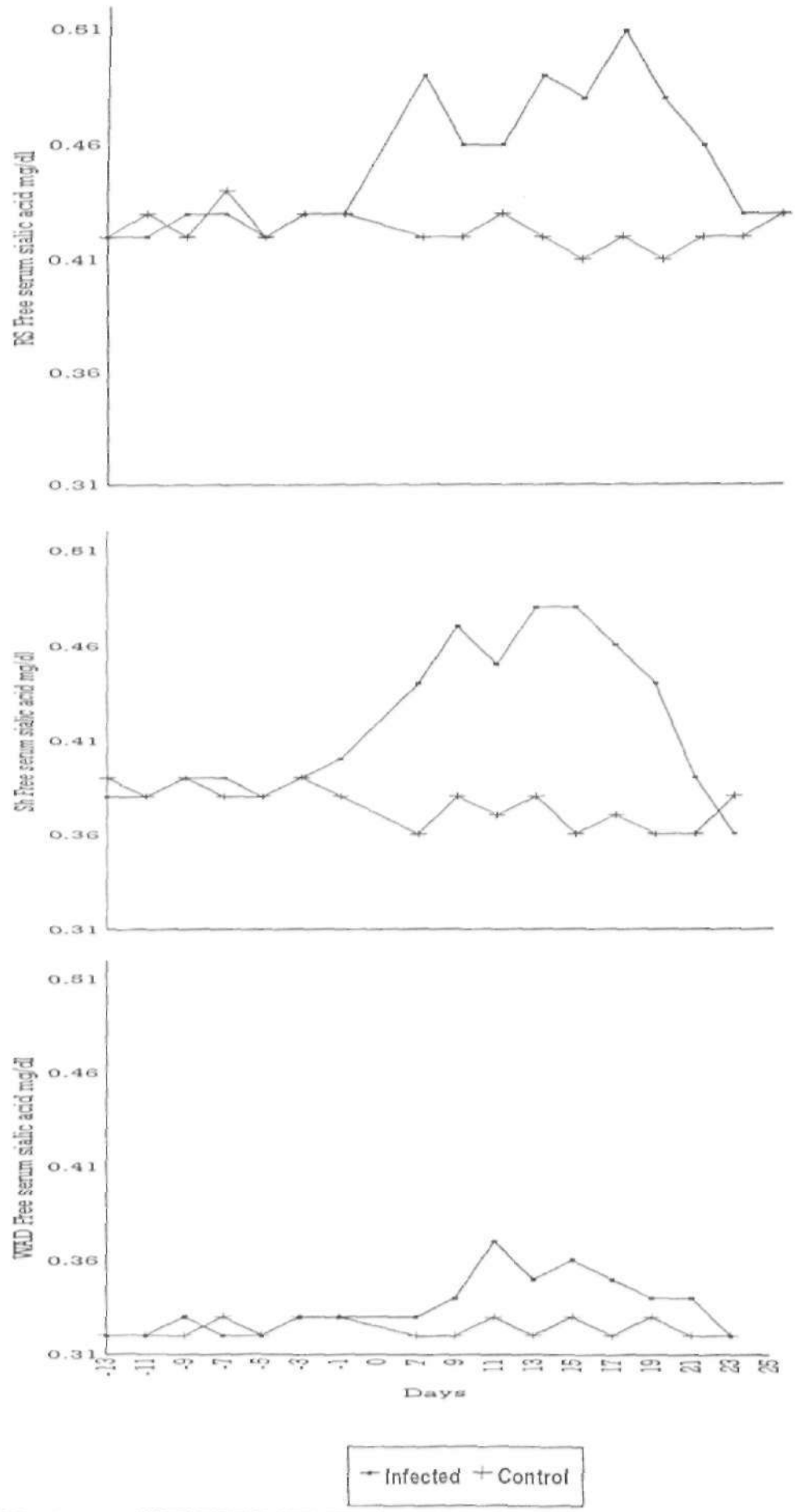


FIG. 12: THE FREE SERUM SIALIC ACID CONCENTRATIONS OF INFECTED RED SOKOTO, SAHEL AND WEST AFRICAN DWARF GOATS

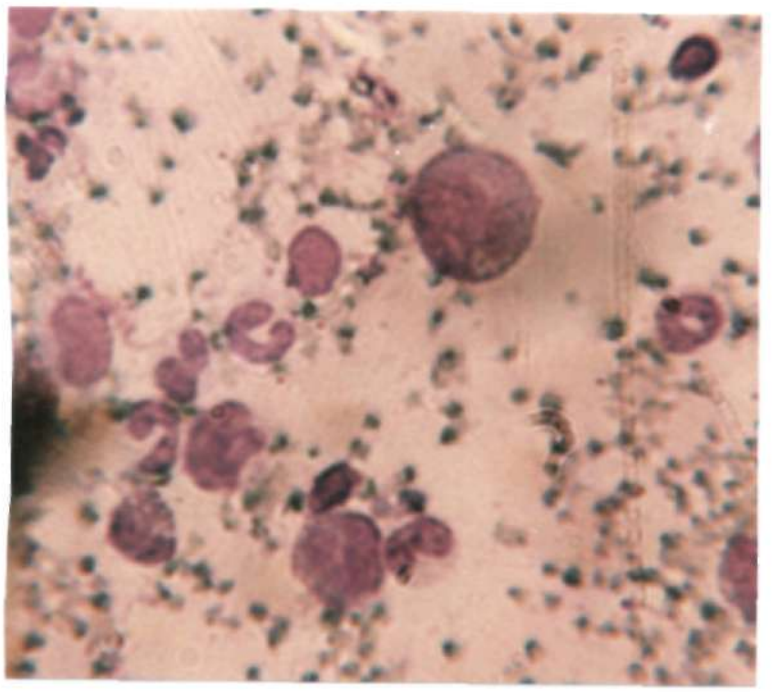


PLATE iiia: THE PRE-INFECTION BONE MARROW SMEAR OF A RED SOKOTO GOAT SHOWING MYELOID CELLS (ARROW HEAD) AND ERYTHROID CELLS (ARROW)

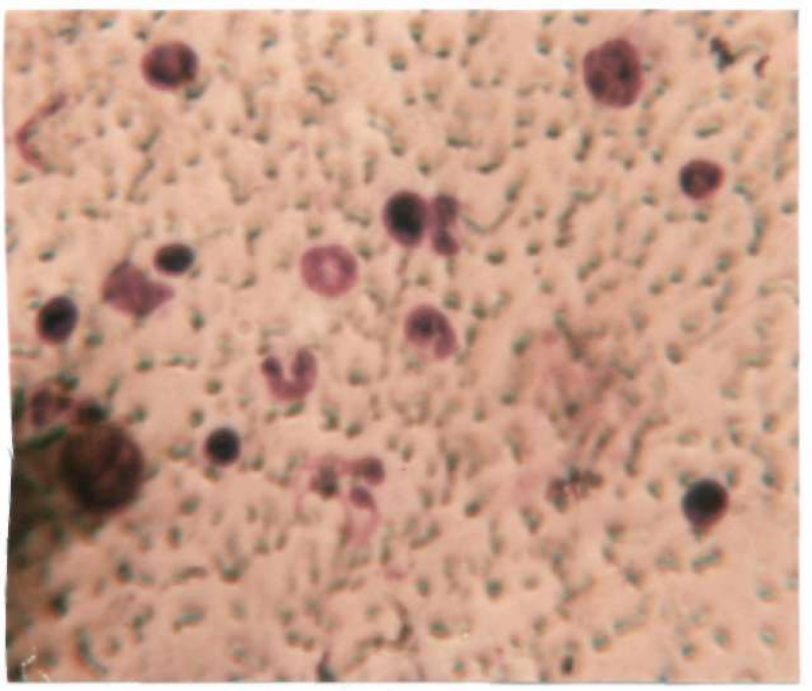


PLATE iiib: THE PRE-INFECTION BONE MARROW SMEAR OF A SAHEL GOAT SHOWING MYELOID CELLS (ARROW HEAD) AND ERYTHROID CELLS (ARROW)

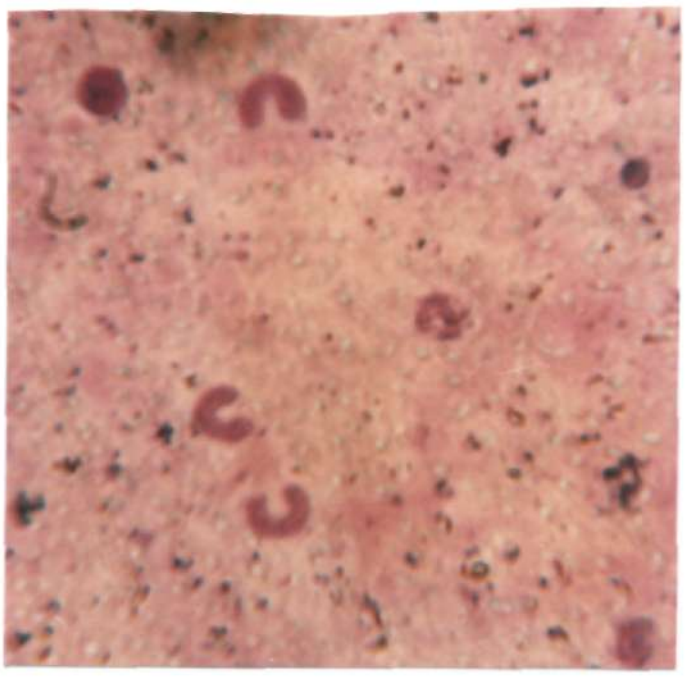


PLATE iiic: THE PRE-INFECTION BONE MARROW SMEAR OF A WEST AFRICAN DWARF GOAT SHOWING MYELOID CELLS (ARROW HEAD) AND ERYTHROID CELLS (ARROW)

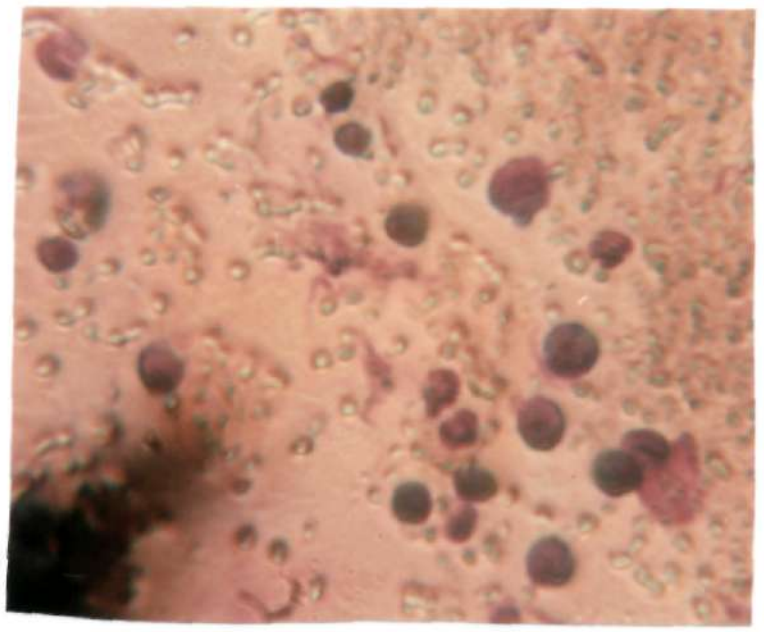


PLATE iva: THE P0ST-INFECTION BONE MARROW SMEAR OF A RED SOKOTO GOAT THREE WEEKS POST-INFECTION SHOWING PROLIFERATION OF ERYTHROID CELLS

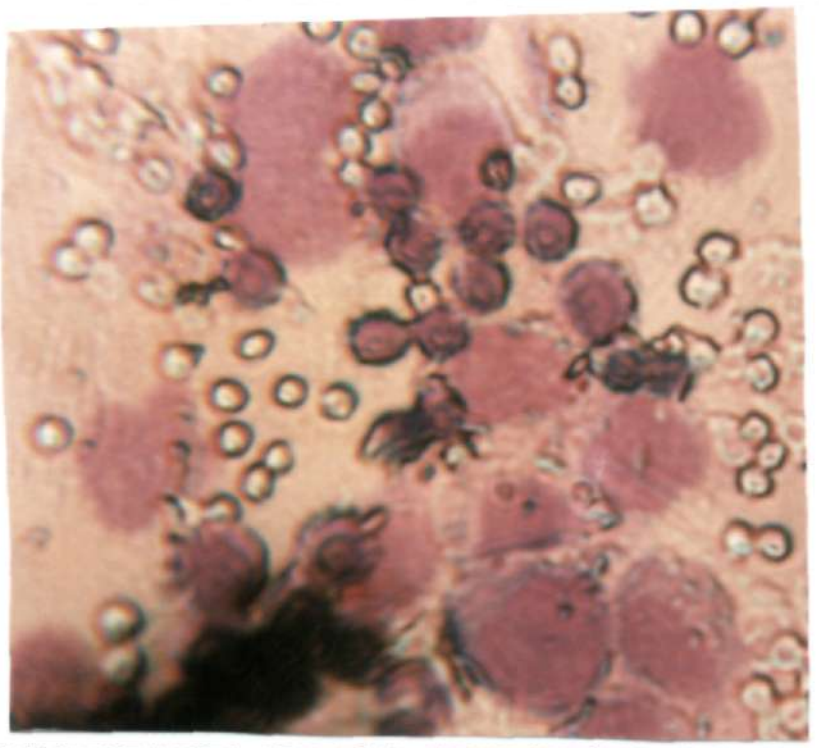


PLATE ivb: THE P0ST-INFECTION BONE MARROW SMEAR OF A SAHEL GOAT THREE WEEKS POST-INFECTION SHOWING PROLIFERATION OF ERYTHROID CELLS

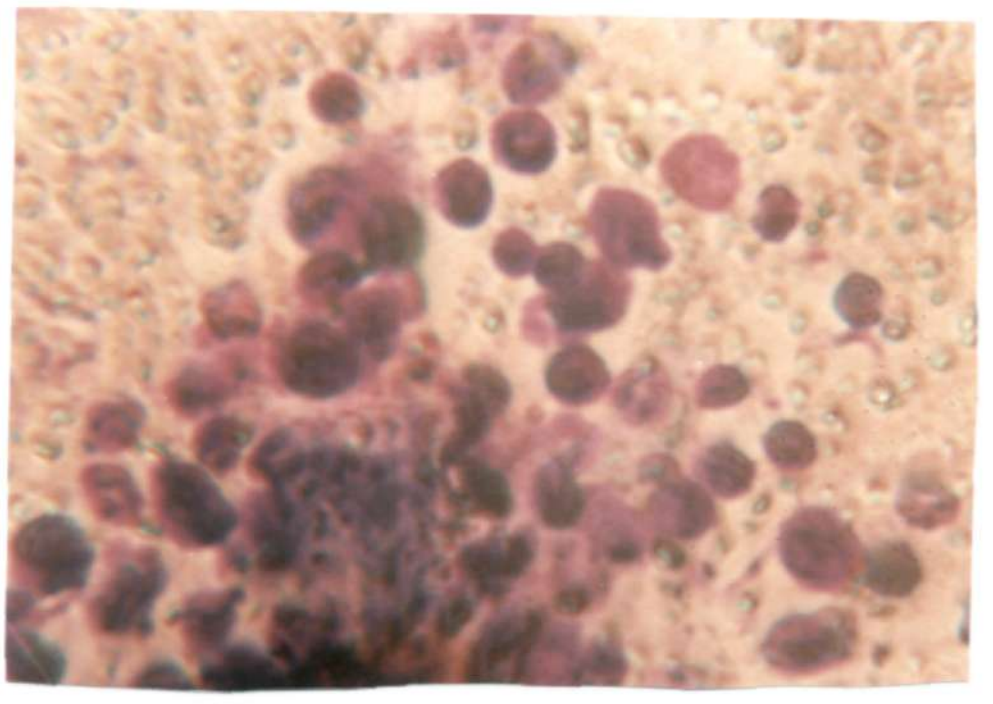


PLATE 1Vc: THE P0ST-INFECTION BONE MARROW SMEAR OF A WEST AFRICAN DWARF GOAT THREE WEEKS POST - INFECTION SHOWING PROLIFERATION OF ERYTHROID CELLS

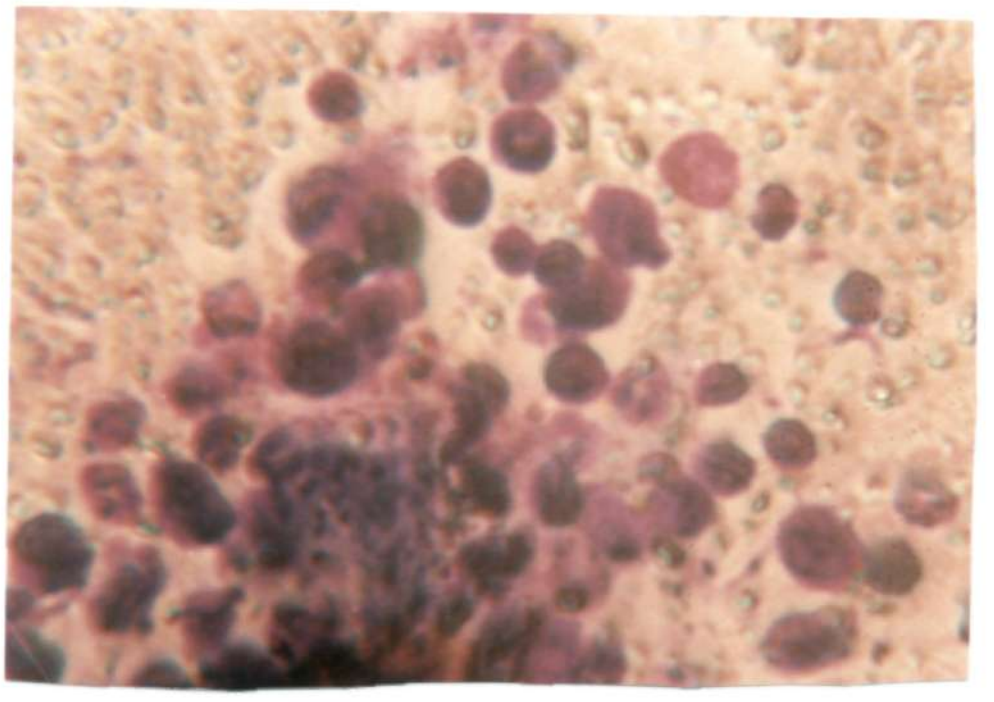


PLATE 1Vc: THE P0ST-INFECTION BONE MARROW SMEAR OF A WEST AFRICAN DWARF GOAT THREE WEEKS POST - INFECTION SHOWING PROLIFERATION OF ERYTHROID CELLS

4.5 The Gross Pathology of T. vivax Infection in Red Sokoto, Sahel and West African Dwarf Goats

Red Sokoto: Three weeks post-infection, goats number 759 was dehydrated and had yellow coloured peritoneal fluid (5.0 ml), with fibrinousadhesions of the visceral organs to the peritoneal wall. Likewise are goats numbers 258, three weeks post-treatment and goats numbers 266, 274 and 286 seven weeks post-treatment. All the infected goats had pale mucous membrane while goat number 286 had diarrhoea which soiled its hindquarter.

Goat number 258 had congested lungs with froth-filled bronchi and tracheae. Goat number 759 had slightly congested, pale and emphysematous lungs. The liver was firm and enlarged. Goat number 258 had necrotic and congested liver two weeks post-treatment. Both goats together with goat number 274 had enlarged bile ducts. Goat number 266, 286 and 234 had firm and slightly congested liver. In all the infected goats, the spleens were found to be firm, dark red and enlarged.

Goats numbers 759 and 258 had pallor of the heart and atrophy of the coronary fat, while goats numbers 258 and 266 have necrosis of the pericardiac fat and flabby hearts. There was hydropericardium with straw coloured pericardial fluid(~ 5.0ml) in goats numbers 759, 258, 266 and 234. The pituitary and adrenal glands of goats numbers 258 and 274 were enlarged while those of the other infected goats were not. The kidneys were pale and enlarged in goats numbers 258, 759, 266 and 286.

Sahel: All the infected goats that were necropsied were dehydrated and had yellow coloured peritoneal fluid (≈ 5.0 ml), with fibrinous adhesions of the visceral organs to the peritoneal wall. There was oedema of the subcutaneous tissues in goats numbers 727 and 734. All the infected goats had pale mucous membrane. Goat number 729 had slightly congested, pale and emphysematous lungs, while goats numbers 727 and 734 had highly congested lungs.

Goat number 729 had necrotic and congested liver 9 days post-treatment. The liver was firm and enlarged with enlarged bile ducts. In goats numbers 727 and 729 the spleens were found to be firm, dark red and enlarged. Goats numbers 727 and 734 had pallor of the heart and atrophy of the coronary fat, while the pericardial fat in goat number 729 was necrotic and the heart was flabby. There was hydropericardium with straw coloured pericardial fluid (≈ 5.0ml) in all the three goats. The adrenal and the pituitary glands of all the infected Sahel goats that were necropsied were not enlarged but the kidneys were pale and enlarged.

West African Dwarf: Goat number 740 was dehydrated with fibrinous adhesions of the visceral organs to the peritoneal wall. Goat number 736 had serous atrophy of fat and yellow coloured peritoneal fluid. Goats numbers 736 and 738 had slightly pale lungs.

The livers of the three goats (736, 738 and 740) that were necropsied were firm with goat number 736 having slightly enlarged liver and goat number 738 had

Chapter 5

DISCUSSION

All the infected goats in the three groups namely the Red Sokoto (RS), Sahel (Sh) and West African Dwarf (WAD) developed trypanosomiasis after inoculation with Trypanosoma vivax. In the RS and WAD groups, the prepatent period was 3 to 5 days. The Sh group developed trypanosomiasis 4 to 5 days post-infection. Saror (1980) reported a prepatent period of 5 days in RS goats intravenously infected with T. vivax while Masake (1980) and Kalu et al. (1987) reported a prepatent period of 5 to 7 days in RS goats subcutaneously infected with T. vivax. The above findings support the observation by Anosa (1983) that the incubation period of T. vivax infections of ruminants varies considerably and is influenced by the route of infection. This is also confirmed by Esievo and Saror (1983), Ogwu et al.(1984) and Sekoni et al. (1988 ; 1991).

In the three groups, the onset of parasitaemia was associated with increase in their rectal temperatures. The infected RS and Sh goats had their temperature peaks coinciding with their parasitaemic peaks while the WAD group had a more gradual increase in their temperature in which the peak only coincided with the second peak of parasitemia. The infected WAD goats recorded the highest mean rectal temperature of 40.30 ± 0.02°C five weeks post-infection. This agreed with the report of Opasina and Onyeka (1982) who reported a pyrexia of 40.5°C to 41.5°C in WAD goats infected with T. vivax . Zwart et al . (1990) also observed that T. vivax infection in WAD goats sets their body temperature at a higher level. Anosa and Isoun (1974)

and Saror (1980) also reported that goats infected with T. vivax suffer pyrexia. Weir (1981) stated that temperature is an important factor in determining the innate immunity of an animal to some infective agents and the pyrexia that follows so many different types of infections can function as a protective response against the infecting microorganisms although this may not be applicable to trypanosome infection.

The massive but uninterrupted parasitaemia with all the related combined pathogenic effects of the parasites at this period may account for the faster weight loss recorded by the infected Sh goats second week post-infection. While in the RS goats, weight loss did not occur until four weeks post-infection and there was an attempt by the infected RS goats to regain the lost weight after treatment. The infected WAD goats recorded a slight increase in their mean body weight between the first and second week of infection followed by a gradual loss of weight. This may be due to the steady appetite maintained by the WAD goats during the early period of the infection. Weight loss in goats infected with T. vivax has also been reported by Saror (1980), Opasina and Onyeka (1982), Ogwu (1983) and Zwart et al. (1991).

Soon after parasites were detected from the blood of the infected goats in the three groups, they developed several waves of parasitaemia. The peaks of parasitaemia was separated by a period of low parasitaemia in the infected WAD group. The highest parasitaemic peak amongst the three breeds occurred in the Sh group two

weeks post-infection. The infected RS and WAD goats also recorded their first parasitaemic peaks week two post-infection. The second peak was recorded sixth week post-treatment in the RS while that of the WAD was recorded fifth week post-infection. Anosa and Isoun (1974, 1980a), Masake (1980) and Anosa (1983) reported the first peak second week post-infection in RS goats infected with T. vivax, while Saror (1980) observed a time interval between subsequent peaks ranging from 4 to 7 days in RS goats.

Relapse occurred in the infected RS and WAD goats. Parasites were first seen in the blood of the infected RS goats between the fifth and sixth week post-treatment while it occurred in the infected WAD goats between the third and fourth week post-treatment. The red cell counts dropped in all the infected three breeds of goats. The Sh goats recorded the highest drop and this coincided with the high parasitaemia which may have a cumulative effect on their erythrocytes. This agrees with Anosa and Isoun (1980b) who observed that the red cell values of animals infected with T. vivax always drop precipitously.

The reduction in red blood cell values of the infected animals could also be due to the high temperature that accompanied the infection in the animals. Red blood cells exposed to temperatures of a few degrees above normal body temperature do have increased osmotic fragility, have a shortened life span and undergo accelerated haemolysis (Murray, 1978). It could also be due to immune sensitization of the red blood cells brought about by the trypanosome antigens which resulted in some

immunologic and physiologic reactions thereby rendering the cells more prone to erythrophagocytosis by the expanded mononuclear phagocytic system (MPS).

Saror (1980) stated that later in the course of the infection as the parasitaemia wanes, the red cell values begin to recover which sometimes attain the pre-infection levels. This was observed in the infected RS and WAD groups where there was a gradual increase in their red blood cell values as the parasitaemia waned, but the value remained well below the mean pre-infection values. The attachment of trypanosomal antigen which resulted in the release by the trypanosomes, enzyme sialidase, which cleaves off sialic acid from the erythrocyte surface also exposes the erythrocytes to phagocytes which could also account for the fall in the RBC value in the infected animals.

The infected RS goats had a sharp drop in their PCV. Their lowest mean PCV coincided with the first parasitaemic peak and following treatment, the PCV increased at almost the same rate at which it fell, but could not reach the pre-infection value. The infected Sh goats had the highest drop in their PCV and this supports the observation of uninterrupted parasitaemia observed in them. The infected WAD goats had a gradual fall in their PCV with the lowest value coinciding with the second peak of parasitaemia which occurred three weeks after the first peak. Anosa and Isoun (1974) stated that the low PCV observed in T. vivax infected animals indicates that trypanosomiasis is accompanied by anaemia. These observations agreed with

Anosa (1977) who noted that in ruminants, PCV drops during parasitaemic peaks and then rises slightly during the intervening periods, thereby suggesting that red cell destruction is closely related to the presence of parasites in the blood. Masake (1980) also reported a significant fall in PCV of goats infected with T. vivax which also coincided with the first wave of parasitaemia.

Thrombocytopenia occurred in the three groups early in infection and this agreed with Anosa and Isoun (1980a) who stated that this may be due to platelet phagocytosis or disseminated intra vascular coagulation. Between the second and fourth weeks of infection,the platelet values increased in the three groups and reached the pre-infection values in only the Sh and WAD groups.

Leucopenia characterized by neutropenia and lymphopenia was observed in the early stage of the infection in all the infected animals in the three groups. This corresponded with the peak, and duration of the parasitaemia. Kaaya et al. (1978) observed that the occurrence of leucopenia early in trypanosome infections could be due to massive leucophagocytosis in the liver, after the coating of leucocytes by trypanosomal antigen or to loss of stem cells caused by a circulating toxic factor from the trypanosomes. Esievo and Saror (1983) suggested that an increase in trypanosomal antigens and neuraminidase in the infected animals early in infection may result in leucopenia.

Eosinopenia was observed in all the infected goats within two weeks of infection and was followed immediately by eosinophilia. Eosinophilia also occurred later after treatment in the WAD group. The monocytopenia that occurred at the early stage of infection in the three breeds of goats was followed by monocytosis between the second and third weeks of infection with intermittent rise and fall in their values. This could be due to chronicity and the expanded and active mononuclear phagocytic system (MPS) which led to erythrophagocytosis and the clearing of the monocytes which might have engulfed some erythrocytes.

Marked erythropoiesis which was observed in the bone marrow of the infected goats following infection resulted in increase in the number of nucleated normoblasts and reduction in the number of granulocytic cells. This was in response to the erythrophagocytosis that occurred during the infection by phagocytes (monocytes). It was an attempt by the bone marrow to replace the red cells that were removed from circulation. The reduction in the number of luecocytic granulocytes could also be due to their clearance from circulation after phagocitising the red cells on which the trypanosomal antigen had attached and also after clearing from circulation, the macrophages and other phagocytes that could have engulfed the red cells. Saror (1980) also reported a marked erythropoiesis in the bone marrow of infected RS goats infected with T. vivax.

SUMMARY AND CONCLUSION

The three breeds of goats are all susceptible to the pathogenic effect of the Trypanosoma vivax used in this experiment. The Red Sokoto (RS) goats recorded the first death when one of the infected goats died three weeks post-infection. Others were treated and responded to treatment. Four of the infected Sahel (Sh) goats died within one week of treatment which was four weeks post-infection. The remaining two died two weeks post-treatment without responding to the treatment given. The infected West African dwarf (WAD) goats did not record any death throughout the period of infection. Five weeks post-infection they were treated when they were weak. They responded to treatment, however, three weeks post-treatment, relapse occurred. The Sh goats were the most susceptible because none of the infected ones responded to treatment . Although one of the RS goats died three weeks post-infection, others received and responded to treatment.

The infected WAD goats had the highest rectal temperature post-infection while the pattern was similar in both the infected RS and Sh goats. The infected WAD goats also showed an increase in their body weight within the first two weeks of infection as they maintained a steady appetite during the period. This was followed by a gradual loss in body weight. However, the pattern was similar in the infected RS and Sh goats.

There was a massive and uninterrupted parasitaemia in the infected Sh goats for four weeks. The parasitaemia reached its peak within two weeks of infection. The infected WAD goats had two weeks of low parasitaemia between the two parasitaemic peaks. The infected RS goats had a period of aparasitaemia three weeks post infection because they were treated at this time. Soon after parasites were detected in the blood of the infected goats in the three groups, they developed several waves of parasitaemia. The infected RS goats had a sharp drop in their PCV. Following treatment, the PCV increased gradually but could not reach the pre-infection value. The infected Sh goats had the highest drop in their PCV with no recovery in the value up till the end of the experiment. The infected WAD goats had a gradual fall in their PCV with the lowest value coinciding with the second peak of parasitaemia which occurred three weeks after the first peak. It has been shown that the low PCV observed in T. vivax infected animals indicates that trypanosomiasis is accompanied by anaemia (Anosa and Isoun, 1974). These observations agreed with Anosa (1977) who noted that in ruminants, PCV drops during parasitaemic peaks and then rises slightly during the intervening periods, thereby suggesting that red cell destruction is closely related to the presence of parasites in the blood. Masake (1980) also reported a significant fall in PCV of goats infected with *T. vivax* which coincided with the first wave of parasitaemia.

The erythrocyte counts dropped in all the infected goats after infection. The erythrocytes appear to be most vulnerable to the pathogenic effects of the circulating T. vivax in all the infected goats, with those of the RS and Sh being more vulnerable

than those of the WAD goats. The infected Sh goats recorded the highest drop in the erythrocyte counts which may be due to the massive uninterrupted parasitaemia. Leucopenia occurred in all the infected goats following infection. This was characterised by neutropenia and lymphopenia. However, following treatment, there occurred leucocytosis caused by lymphocytosis and neutrophilia in only the infected WAD goats. In the infected Sh goats, there was leucocytosis three weeks post-infection caused by neutrophilia and this was only followed by lymphocytosis four weeks post-infection. In the infected RS goats, the leucopenia characterised by lymphopenia and neutropenia continued right from the onset of infection till two weeks post-treatment. However, by the second week post infection, there was eosinophillia which in the infected RS goats continued till one week post-treatment. Eosinopenia occurred in both the infected Sh and WAD goats shortly after infection indicating systemic stress. One week post-treatment, there occurred eosinophilia in the WAD group which might be induced by the drug used for the treatment.

The bone marrow response in the infected goats showed that their bone marrow tried to replace the red cells which were being removed from the circulation through phagocytosis. This resulted in marked erythropoiesis two weeks post- infection in both the infected RS and Sh goats and three weeks post infection in the WAD goats.

The free serum sialic acid concentration increased in the infected goats two to three weeks post-infection. The increase was however lowest in the WAD group compared to the other two groups. This suggests breed influence on the attachment of

trypanosomal sialidase to the sialidase receptor on the membrane of the red blood cells and eventual cleaving off of sialic acid from the red blood cell membrane. The geographical distributions of these three breeds of goats and tsetse flies which are the vectors of the trypanosomes could have also played an important role in their susceptibility to the trypanosome infection. The Sh goats are naturally found in the Sahel savanna which is found to be tsetse free, while the other two breeds are found in the regions which are infested with tsetse flies. Trypanosoma vivax has been found to be virulent in the West African subregion (Sackey, 1998) and this may also be a major factor in the susceptibility of the three breeds of goats to the infection.

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APPENDICES

Appendix 1: Pre-infection Differential Femoral Bone Marrow Cytology (%) of Red Sokoto Goats

	Infected						Control	
	286	234	266	274	258	759	250	760
Erythrocytic cells:-Erythroblasts	1.4	1.6	1.4	3.2	1.0	2.8	0.5	2.0
Pronormoblasts	3.8	3.1	6.6	1.6	4.8	3.2	4.6	4.0
Normoblasts :- Basophilic	25.8	8.3	16.0	13.5	20.0	14.8	18.2	15.8
Polychromatophilic	5.8	2.8	6.4	7.7	5.4	4.8	5.8	7.2
Late normoblasts	4.0	7.6	8.0	8.2	4.8	6.0	6.2	3.4
Granulocytic cells:- Myeloblasts	1.0	4.8	6.4	1.4	3.4	0.6	1.2	10.8
Promyelocytes	1.4	2.2	4.8	3.2	1.6	2.2	2.8	3.6
Neutrophil :- Myelocytes	1.6	2.1	3.2	0.5	1.4	2.4	2.6	1.2
Metamyelocytes	0.2	12.4	11.8	26.4	17.6	15.6	10.0	10.4
Bands	3.6	2.2	4.0	5.2	2.8	7.0	7.2	6.2
Segmenters	28.0	15.6	23.4	13.4	26.8	20.2	30.8	18.2
Eosinophil :- Myelocytes	0.0	1.0	0.4	0.6	0.0	1.2	0.0	0.0
Metamyelocytes	1.4	0.0	0.6	1.4	2.0	1.6	0.0	1.0
Bands	0.0	1.3	0.2	0.6	0.0	0.0	0.0	0.0
Segmenters	11.2	0.0	12.8	8.3	9.0	1.0	6.8	0.4
Basophils	0.0	0.5	0.4	0.0	0.4	0.2	0.7	0.0
Myeloid : Erythroid ratio	1.28:1	1.78:1	1.77:1	1.49:1	1.81:1	1.65:1	1.76:1	1.61:1
Other cells :- Lymphocytes	8.0	4.6	11.4	10.0	2.6	10.0	10.2	9.2
Plasma cells	0.0	0.2	0.0	0.5	0.0	0.4	0.2	0.0
Monocytes / Monoblasts	0.6	1.0	0.4	0.8	2.0	1.2	0.0	2.0
Macrophages	0.6	2.6	0.6	2.3	0.6	4.8	1.0	2.4
Unclassified	0.2	0.0	1.2	0.0	0.4	0.6	0.5	0.4
Damaged	1.0	0.9	0.6	1.2	0.6	0.8	0.4	1.8
Mitotic figures	0.4	0.0	0.2	0.0	0.8	0.6	0.3	0.6
	10.8	9.3	14.4	14.8	7.0	18.4	12.6	16.4

Appendix 2: Pre-infection Differential Femoral Bone Marrow Cytology (%) of Sahel Goats

	Infected						Control	
	726	727	729	731	733	734	728	730
Erythrocytic cells:- Erythroblasts	1.2	2.6	1.0	2.8	0.4	1.0	1.4	0.4
Pronormoblasts	9.0	6.8	8.9	2.4	8.4	9.0	6.0	9.2
Normoblasts :- Basophilic	12.8	13.2	12.3	21.4	14.2	13.5	22.0	15.0
Polychromatophilic	4.0	4.2	8.0	4.4	6.5	3.4	5.0	7.6
Late normoblasts	3.2	3.6	1.8	1.6	2.6	6.0	3.0	2.2
Granulocytic cells:- Myeloblasts	2.4	0.0	0.5	0.2	0.0	1.6	1.4	0.8
Promyelocytes	3.4	2.2	0.6	1.4	2.6	0.4	1.0	3.6
Neutrophil :- Myelocytes	3.4	2.4	0.8	2.2	2.8	1.6	1.2	0.8
Metamyelocytes	14.2	6.8	12.6	20.4	15.3	7.4	10.6	13.0
Bands	4.8	14.0	10.2	4.8	8.5	10.4	7.0	2.0
Segmenters	15.6	20.0	18.0	18.2	17.0	14.2	21.0	20.6
Eosinophil :- Myelocytes	0.2	1.4	0.0	1.2	6.4	0.0	1.0	1.0
Metamyelocytes	0.2	1.2	0.4	1.4	0.0	2.8	0.4	1.2
Bands	0.4	0.0	0.2	0.0	0.6	0.0	0.0	0.4
Segmenters	6.0	0.0	0.8	0.4	0.0	3.6	1.0	0.0
Basophils	0.0	0.6	1.0	0.0	0.4	0.2	0.0	0.0
Myeloid : Erythroid ratio	1.68:1	1.71:1	1.41:1	1.54:1	1.67:1	1.27:1	1.13:1	1.26:1
Other cells :- Lymphocytes	14.0	15.2	17.4	10.4	13.4	12.4	10.0	16.0
Plasma cells	1.0	0.0	0.4	0.2	0.7	2.0	0.0	0.0
Monocytes / Monoblasts	2.2	1.6	2.3	1.6	0.8	2.4	1.0	0.6
Macrophages	0.6	2.6	2.0	2.8	0.8	3.4	1.0	1.0
Unclassified	0.2	0.2	0.5	0.8	0.0	1.6	0.0	0.2
Damaged	0.0	0.2	2.6	0.6	0.6	0.6	4.0	4.4
Mitotic figures	0.2	3.2	0.0	0.8	0.2	2.4	0.0	0.0
	18.2	23	25.2	17.2	16.5	24.8	16	23.2

Appendix 3: Pre-infection Differential Femoral Bone Marrow Cytology (%) of West African Dwarf Goats

	Infected						Control	
	736	737	738	739	740	742	735	741
Erythrocytic cells:- Erythroblasts	2.4	4.0	1.6	1.6	1.2	0.4	1.4	1.4
Pronormoblasts	7.2	2.0	6.0	6.2	8.0	10.0	8.6	2.4
Normoblasts :- Basophilic	11.0	13.0	15.2	12.6	17.2	8.2	13.0	15.8
Polychromatophilic	6.6	8.0	6.2	8.4	7.4	6.8	5.0	10.2
Late normoblasts	7.4	4.2	6.4	7.0	4.0	8.2	4.0	6.0
Granulocytic cells :- Myeloblasts	0.8	0.2	1.0	0.2	1.6	0.4	1.4	0.4
Promyelocytes	3.6	2.6	4.2	1.6	3.4	1.4	1.8	0.6
Neutrophil :- Myelocytes	10.8	12.4	10.4	4.4	4.0	10.6	11.2	10.2
Metamyelocytes	3.2	4.6	3.8	12.6	9.2	7.4	0.4	2.8
Bands	2.4	7.0	4.0	2.2	10.4	7.0	7.0	5.0
Segmenters	28.0	25.6	22.6	20.2	8.2	20.8	30.2	26.0
Eosinophil :- Myelocytes	1.0	1.6	1.8	0.2	0.6	0.4	1.2	0.4
Metamyelocytes	0.5	0.0	0.2	0.2	1.0	2.0	0.4	0.0
Bands	0.2	0.2	0.0	0.2	1.0	0.2	0.0	0.0
Segmenters	1.0	4.4	0.0	2.4	1.4	2.0	1.6	0.4
Basophils	0.0	0.0	0.6	0.0	2.4	0.0	0.2	0.0
Myeloid : Erythroid ratio	1.49:1	1.88:1	1.37:1	1.24:1	1.14:1	1.55:1	1.73:1	1.28:1
Other cells :- Lymphocytes	11.2	7.0	11.0	10.0	8.0	9.0	14.0	14.0
Plasma cells	0.2	0.6	0.4	2.0	1.2	0.0	0.0	0.0
Monocytes / Monoblasts	0.1	0.2	1.6	2.2	0.0	1.0	1.4	0.4
Macrophages	1.0	0.4	1.6	1.8	0.6	1.2	1.0	1.0
Unclassified	0.4	1.0	0.0	0.8	1.0	0.2	0.0	0.2
Damaged	1.0	0.6	0.6	1.8	0.0	2.2	0.6	1.4
Mitotic figures	0.2	0.6	0.2	1.4	0.0	0.6	0.4	1.4
	14.1	10.4	15.4	21	10.8	14.2	17.4	18

Appendix 4: Post-infection Differential Femoral Bone Marrow Cytology (%) of Red Sokoto Goats

	Infected						Control	
	286	234	266	274	258	759	250	760
Erythrocytic cells:- Erythroblasts	0.4	2.4	1.8	0.2	0.4	0.4	0.4	1.4
Pronormoblasts	2.6	14.8	10.4	1.0	2.0	3.0	4.0	2.8
Normoblasts :- Basophilic	60.0	29.8	30.4	51.2	53.4	47.4	14.6	18.0
Polychromatophilic	6.0	5.2	4.8	12.0	2.4	1.8	9.8	10.0
Late normoblasts	2.0	8.0	10.2	6.2	1.6	1.4	5.4	3.0
Granulocytic cells:-Myeloblasts	0.4	1.2	1.0	0.4	0.6	0.8	3.0	8.0
Promyelocytes	1.4	3.8	2.2	0.2	1.2	2.8	4.8	3.2
Neutrophil :- Myelocytes	0.6	4.4	6.8	0.6	1.0	4.2	2.2	5.8
Metamyelocytes	5.6	10.6	4.2	2.0	3.0	9.0	11.8	14.8
Bands	1.2	0.0	3.6	4.0	0.6	1.8	2.6	8.0
Segmenters	14.0	14.6	16.0	14.0	20.4	12.0	24.4	15.6
Eosinophil :- Myelocytes	0.0	0.0	0.4	0.0	0.0	0.0	0.2	0.8
Metamyelocytes	0.0	0.2	0.0	0.0	0.0	0.2	0.4	0.0
Bands	0.0	0.0	0.6	0.0	0.4	0.0	0.0	0.6
Segmenters	0.2	0.2	0.0	0.4	0.6	0.4	7.0	0.6
Basophils	0.2	0.0	0.2	0.0	0.0	0.0	0.0	0.2
Myeloid : Erythroid ratio	0.33:1	0.58:1	0.61:1	0.31:1	0.47:1	0.58:1	1.62:1	1.64:1
Other cells :- Lymphocytes	3.6	2.4	4.0	5.0	1.0	7.2	6.4	6.0
Plasma cells	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.2
Monocytes / Monoblasts	0.2	0.4	0.0	0.8	0.6	0.4	0.6	0.0
Macrophages	0.6	0.6	0.8	1.0	1.4	6.2	0.2	1.0
Unclassified	0.2	0.2	1.6	0.6	0.0	0.0	0.4	0.0
Damaged	0.6	0.2	0.0	0.2	1.8	0.6	1.0	0.0
Mitotic figures	0.6	0.2	0.0	0.2	1.8	0.6	1.0	0.0
	5.4	4.8	7.4	7.8	5.2	14.8	9.4	7.2

Appendix 7: **Chemical Names of Drugs Used**

Trade name	Chemical Name
Terramycin LA (Pfizer, Nigeria)	Neomycin oxytetracycline HCl
Berenil (Bayer, Germany)	Diminazene aceturate
Asuntol (Bayer, Germany)	Coumachlor - 3 - ∝ acetonyl - 4 - chlorobenzyl
Panacur (Hoechst, Nigeria)	Pyrantel tartarate