TRYPANOSOMIASIS: A STUDY ON THE DEVELOPMENT AND CONTROL IN THREE BREEDS OF GOATS: ''

BY

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A THESIS

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DECLARATION:

I hereby declare that this thesis is my original work and has not been presented for a degree in any other University.

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SUMMARY

Trypanosomiasis is an important disease of tropical Africa which greatly limits livestock production. This disease has had extensive study in large ruminants while relatively little has been carried out in small ruminants. This study was carried out using three breeds of goats, namely, Toggenburg X East African, Galla X East African and the East African goats. Three aspects were considered; effect of chemotherapy on production parameters, comparative trypanotolerance among the East African goats and the relationship between tsetse fly numbers, rainfall, relative humidity and infection rates.

Each breed of goat was divided into an experimental and a control group. The experimental groups got a monthly injection of isometamedium chloride (Samorin$^{(R)}$) at the dose rate of 0.5 mg/kg body weight. The drug was administered intramuscularly at the side of the neck.

The production parameters considered in this study were packed cell volume and body weight. The packed cell volume was measured every two weeks while the body weight was taken once every month. The haematocrit centrifuge technique was used for trypanosome detection and the morphological

identification done after staining with Giemsa diluted 1:10 in neutral distilled water.

Among the control groups the body weights, the changes in packed cell volume, infection rates and mortality rates were compared.

Rainfall and relative humidity were measured on a daily basis while tsetse flies were caught by putting out Challier traps for four consecutive days every month. The flies were identified into species and sex and the number caught per trap per 24 hours worked out for every month.

Where a breed of goat was involved in mating, the fertility rates among an experimental or a control group was evaluated with regard to the proportion of animals kidding, the proportion of animals twinning and the mean birth weight of the kids.

The results indicate that, the groups of animals receiving samorin had higher mean packed cell volume values and higher weight gains than the controls that were not receiving any samorin. The results on fertility show that the animals in the treatment groups had higher proportions of animals kidding and twinning. The East African goats showed a higher degree of trypanotolerance than the other two breeds as manifested by the packed cell volume values and the mortality rates

due to trypanosomiasis. The Galla X East African goats had an intermediate degree of trypanotolerance between the East African and the Toggenburg X East African goats. The Toggenburg X East African goats showed an increase in infection rates following an increase in tsetse fly numbers. The East African goats also showed a similar increase but of a much lower magnitude. The Galla X East African goats showed changes in infection rates which were not concomitant with changes in tsetse fly numbers.

The cost of controlling trypanosomiasis using samorin on a monthly basis at the dose rate of 0.5 mg/kg body weight per tropical livestock unit was worked out. Some of the economic losses resulting from trypanosomiasis were also worked out. It was found that the cost/benefit ratio was highest among the Toggenburg X East African goats followed by the Galla X East African goats. It was lowest among the East African goats which manifested the highest degree of trypanotolerance.

TITLE: TRYPANOSOMIASIS: A STUDY ON THE DEVELOPMENT AND CONTROL IN THREE BREEDS OF GOATS:

INTRODUCTION

Trypanosomiasis is a Protozoal disease caused by various species of trypanosomes; Hornby (1949) defined it as a group of several allied diseases each of which is due to infection with a specific trypanosome.

The disease affects both man and domestic animals. Except in horses and dogs, animal trypanosomiasis is typically a chronic rather than a fulminating infection (Whiteside, 1958). The disease has for a long period of time continued to hamper animal production because it was overshadowed by other more clinically dramatic diseases such as rinderpest, East Coast fever, blackquarter, contagious bovine pleuropneumonia and anthrax. These diseases seemed economically more damaging and hence much of the then scanty veterinary attention was focussed on these diseases other than trypanosomiasis (Whiteside, 1958). Now that most of the above diseases are largely under control, it has become imperative to control trypanosomiasis (Kaaya, 1975) and hence increase the output of animal protein for the needy increasing human population. Daily per capita consumption of animal protein is 11 gms in developing countries compared to 49 gms in developed countries (Griffin, 1978).

The most important causes of trypanosomiasis

in livestock in Kenya are two species of trypanosomes, T. congolense and T. vivax (Whiteside, 1958; Omuse, 1973). These trypanosomes are cyclically transmitted by tsetse flies (Glossina species) and hence the distribution of the disease follows a pattern similar to that of Glossina distribution (Whiteside, 1958; Omuse, 1973). Within the African continent, the disease is spread between latitudes 14°N and 29°S of the equator. This area is almost one third of the total landmass of Africa (Kaaya, 1975) and its underutilized as far as the production of livestock is concerned (Finelle, 1974). In Kenya, the disease is enzootic in large areas of the Coast Province, Northern Province and Southern Province. In the Central and Nyanza Provinces, the disease is localised in certain districts (Whiteside, 1958; Omuse, 1973). The total Kenyan landmass occupied by tsetse flies is approximately 138,000Km^2 nearly a ¼ of the total landmass (Kaaya, 1975).

In 1933 cited by Whiteside,(1958), the annual reports from the Veterinary Department revealed that trypanosomiasis had by this time posed a serious menase to the grazing areas. Interest was thus taken into the study of the disease but most of the research carried out was in cattle and the small ruminants were ignored. Whiteside, (1958) states that sheep and goats are commonly resistant to the disease.

In view of this, the amount of literature available on trypanosomiasis in small ruminants is scanty, thus

most of the material on the literature review is based on the disease as it occurs in cattle. So far trypanosomiasis in goats has been explored where infection of the goats with the trypanosome species has been done experimentally. Thus Kaaya, (1975) delt with the pathogenesis of T. congolense. Isoun and Anosa, (1974) studied the effect of experimental infection of sheep and goats with T. vivax on the reproductive organs. Griffin and Allonby (1979a, b, c, d) worked on the disease in both sheep and goats investigating the epidemiology, trypanotolerance economic aspects and the clinical syndromes. The epidemiological and trypanotolerance investigation in goats revealed that exotic goat breeds were more susceptible to trypanosomiasis than the indigenous small East African goats while the crosses between the indigenous and the exotic breeds showed an intermediate susceptibility to the disease. The study also tried to reveal the correlation between tsetse fly numbers and rainfall. With the introduction of the milk goats such as Toggenburg, a similar study would be a necessity in multiplication centres such as Matuga (in the Coast Province) whose sole purpose is to upgrade the small East African goats and hence sell them to the farmers. The economic study in goats was carried out using two breeds of goats, the Saanen X Galla and the Galla goats.

This study left out the most common breed found in Kenya, the small East African goats and the crosses between the East African goats and the Gallas along with the crosses between the Toggenburgs and the East African goats. Since the upgrading process involves crossing the trypanotolerant East African goats with the exotic breeds of goats (Griffin and Allonby 1979b), then an economic study on Chemoprophylaxis would inevitably involve the indigenous goat breeds. Studies on trypanotolerance by Griffin and Allonby (1979b)in breeds of sheep and goats left out an important milk goat (Toggenburg X E. African), the animals numbers were few and may not have allowed a statistical analysis of the data obtained. The study was carried out by using an isolate strain of T. congolense, thus there was no allowance given for natural challenge to take place in every breed of goat except with the control mixed breed of goats. In view of the above a study with three breeds of goats under natural challenge was necessitated.

Infertility with regard to trypanosomiasis gets little mention in the literature. Isoun and Anosa (1974) reported the presence of T. vivax in the amniotic fluid of a pregnant ewe while, Paikne et al. (1972) reported abortion in a she buffalo due to T. evansi. In cattle Roberts and Gray, (1973) reported

that retarded growth with chronic trypanosomiasis is accompanied by underdevelopment of the reproductive system in both males and females. The males show testicular degeneration (Roberts and Gray, 1973; Kaaya, 1975). In view of the foregoing it was found necessarily to compare fertility rates wherever possible between groups of goats, one receiving samorin (R) (Isometamedium Chloride) and the other not getting any Samorin.

The aims of the research may be summarised thus:

(1) Determine the effects of Chemoprophylaxis using Isometamedium chloride (Samorin(R))on production parameters in the three breeds of goats.

(2) Assess the trypanotolerance among the East African goats raised in trypanosomiasis enzootic areas.

(3) Study the interrelationship that exists between rainfall, relative humidity, fly numbers and the infection rates among the goats.

2. LITERATURE REVIEW:

2.1. Disease distribution and epidemiology:

Generally, the disease is distributed mainly in the African continent between latitudes 14°N and 29°S of the equator (Ford, 1970). It is thus a disease of tropical Africa and its pattern of distribution closely follows the distribution of the insect vector, Glossina spp. The genus Glossina is divisible into three groups each with a somehow characteristic pattern of distribution.

The morsitans group dissappears 4°S of the equator while G. brevipalpis, G. austeni and G. pallidipes are found as far down as Mozambique and to the north of Mombasa where they are joined by G. longipennis. Here the conditions are arid and almost desert and these species have adapted to it. The same are found in West Africa alone with G. tachnoides which is found in both wet and dry conditions. The palpalis group is found along the river courses and in the Congo it is very heavily distributed. It is also found in the Cameroon and the Central African Republic. In general, this group is distributed in the Congo basin and neighbouring areas.

In Nigeria, G. tachnoides has a wide range of distribution with regard to the rainfall pattern of distribution. It is found in areas with as low

rainfall as 15" to areas with a rainfall of 130" per year; hence it has a remarkable ecological variability which is reflected in its feeding habits and hence influencing its role as a vector of trypanosomiasis. In the morsitans group, G. swynnertoni and G. morsitans centralis can complete their development in an atmosphere of 10% relative humidity (R.H.) while G. morsitans submorsitans needs a relative humidity of 30%. G. pallidipes, G. tachnoides G. fuscipes fuscipes and G. austeni have tolerance levels between 40 and 50% R.H. (Ford, 1970).

The palpalis group living in habitats associated with lake edges or rivers avoid the climate extremes of the areas in which they are found. G. austeni lives in the evergreen thickets along the East Coast of Africa often in company of G. brevipalpis. The bush along the Tana river or the arid Tsavo game park supports both G. brevipalpalis and G. longipennis as well as the intermediate species of G. pallidipes and G. austeni.

In general however, whatever might be the true phylogenetic relationships of the three groups of tsetse flies, it is certain today that the fusca group as a whole live in humid environment, the palpalis group live in waterside habitats and the morsitans group is associated with the more arid Savannah type of environment.

With regard to the vegetation, it is erroneous to think that an obligatory association exists between Glossina and the vegetation communities in which the various species are commonly found since tsetse-borne trypanosomes have succeeded in adopting themselves to a life which does not involve cyclical development in Glossina (T. viennei, a parasite of cattle imported to central America from Africa is transmitted by Tabanidae and it is a strain of T. vivax, Ford 1971). However, Boots Company Limited in the manual entitled 'The control of bovine trypanosomiasis' contends that the Glossina distribution is to some extent related to the preffered habitats of different species. Nevertheless, the climate, natural vegetation and the topography of Africa do provide the basis for geographical classification of these insects which is not without its practical uses.

The fusca group can be broadly subdivided into three categories depending on the preffered habitat;

(i). Tropical moist forest at low and medium altitudes e.g. G. tabaniformis and G. hanningtoni.

(i). Forest savannah mosaic or the edges of tropical moist forests e.g. G. fusca and G. medicorum.

(iii). Relict secondary forest patches or thickets in woodlands and savannah e.g. G. brevipalpis or in the wooded steppe G. longipennis. The morsitans group:

(i). G. morsitans morsitans - found in the woodlands of Mozambique, and Zimbambwe below 4,000 feet (1219.5 m) above sea level. It is also found in the East and Central highlands of Tanzania and as far north as the Kenya border just west of Mombasa and in the woodlands of Zambezi Valley.

(ii). G. morsitans centralis - is found in the Brachystergia fulbernardia woodlands west of Lusaka, Broken Hill, Mbeya, Iringa in Zambia, western Tanzania, Katanga and Angola. It is also found in Okavango Swamps in Botswana and in the north in Acacia gerardii wooded grassland west of Lake Victoria.

(iii). G. morsitans submorsitans is found in Northern Uganda, Southern Sudan, westwards to the republic of Guinea and Western Ethiopia.

(iv). G. pallidipes - Before its elimination by use of insecticides in Zululand, (Whiteside 1958), it used to extend further south than other species. It occurs in the North East of Congo basin, at 17°E and it has been replaced by a very similar species, G. longipalpis.

(v). G. swynnertoni - It occupies a relatively small area in the North Central Tanzania, East of Lake Victoria in Acacia cammiphora woodland steppe.

(vi). G. austeni is confined to the coastal plains of East Africa and being found only in a few places with less than 500 feet (152.4 M) above sea level or less than 150 miles (240 Km) inland.

The palpalis group:

With a few exceptions, it is confined to the shores and banks of lakes and rivers, that drain either to the Atlantic or to the Mediterranean Sea G. fuscipes infests the banks of Omo river draining to Lake Rudolf (now Lake Turkana) while G. tachnoides is found in Zimbambwe.

It seems that G. austeni and G. brevipalpis are showing a steady decline in the face of progressive habitat destruction (Snow, 1979) and significant populations only persist where areas of primary habitat remain, while G. pallidipes is still widely distributed and appears more adoptable in its habitat requirements utilizing primary, degraded and secondary habitat types, (Fig 1 and 2- maps on tsetse distribution in Africa and Kenya).

Other biting flies which have been found to transmit the disease other than tsetse flies at Mbarara in Uganda are Musca, Stomoxys, Haematopota and Tabanus. Other biting insects were mosquitoes,

fleas and midges. Ticks are also involved in the transmission of diseases but the relative role of each of the above was not defined (Soltys, 1953). Lucas (1955) successfully demonstrated the mechanical transmission of T. congolense by Tabanids and Stomoxys under field conditions. Kramer (1966) while investigating the incidence of trypanosomiasis in West African Dwarf sheep and goats also suggested that mechanical transmitters such as blood-feeding flies may play an active part in trypanosome transmission in both sheep and goats.

2.2. RESERVOIR HOSTS:

A table as rearranged and summarised by Ford (1971) reveals the results of blood meal analysis from Glossina species excluding those from domestic animals and man

	Host	% feeding on the Host
(i) Lacustrine and riverine feeders Palpalis group tsetse	Reptiles	55.6
	Bushbuck	22.3
	Remainder	22.1
(ii) Forest thicket or forest edge feeders		
Group (A) G.tabaniformis	Bushpig and	74.4
G. fuscipleuris	Forest hog	
G. austeni		
	Remainder	25.6
Group (B) G. fusca.		
G. Pallidipes	Bushbuck	61.1
G. longipalpalis	Bushpig	11.9
	Remainder	27.0
(iii) Savannah feeders	Warthog	57.2
G.m. morsitans,		
G.m. submorsitans	Buffalo	20.6
G. swynnertoni	Giraffe) Kundu Remainder)	22.2

RESERVOIR HOSTS (Contd.)

(iv) Specialised East African fusca group flies	Elephant	
	Rhinoceros	
G. brevipalpis	Hippopotamus	70.9
G. longipennis	Buffalo	
	Bushpig	17.6
	Remainder	11.5

The savannah dwelling Warthogs enabled Glossina to spread trypanosomiasis to the bovidae and giraffidae which are as yet well adjusted than the Suids. The main reservoirs of T. congolense are Giraffe, Waterbucks, Reedbucks, Gazelle, and Bushbucks. Lesser reservoirs include hartebeests and hippopotamus. The hosts of the most widely spread Glossina species (G. pallidipes), are bushbuck, bushpig (Potamocherus porcus) and warthog on the South Coast of Kenya. In widely cultivated areas, the wild pig and the warthog, the bushbuck and the smaller antelop persist (Snow, 1979) and may hence act like reservoirs.

T. congolense has been isolated from carnivores such as the hyaena and the lion but these possibly acquire the protozoa from their feeding habits-carnivorism (Ford, 1970).

T. vivax is commonly found in antelops in addition to several other hosts (McDiarmid 1962).

2.3. Disease Transmission: AGENT (VECTOR)

The true vector of trypanosomiasis is an invertebrate host belonging to the genus Glossina and has been classified by Ford (1971) in the following way:

CLASSIFATION OF GLOSSINA

Class: Insecta

Order: Diptera

Family: Muscidae

Subfamily: Stomoxydinae

Genus: Glossina Wiedemann 1830

Fusca group	Palpalis group	Morsitans grou
Glossina fusca fusca	Glossina palpalis	G. morsitans
Walker 1849	Palpalis Robineau-	Morsitans
G. fusca congolensis	Desvoidy 1830	Westwood 185(
Newstead and Evans 1921	G. palpalis gambiensis	G. morsitans
G. tabaniformis	Vanderplank 1949	submorsitans
Westwood 1850	G. fuscipes fuscipes	Newstead 191(
G. longipennis Corti 1895	Newstead 1910	G. morsitans
G. brevipalpis	G. fuscipes martinii	centralis
Newstead 1910	Zumpt 1933	Machado 1910
G. nigrofusca nigrofusca	G. fuscipes quanzensis	G. swynnertoni
Newstead 1910	Pires 1948	Austen 1923
G. nigrofusca hopkins	G. caliginea Austen 1911	G. longipalpis
Van Emden 1944	G. pallicera pallicera	Wiedeman 183(

G. fuscipleuris Austen. 1911

G. medicorum Austen. 1911

G. severin Newstead 1913

G. schwetzi Newstead and Evans 1921

G. hannington Newstead and Evans 1922

G. vanhoofi Henrard 1952

G. nashi Pottos 1955

Bigot 1891

G. pallicera newsteadi Austen 1929

G. tachnoides Westwood 1850

G. pallidipes Austen 1903

G. austeni Newstead 1912

Baker (1963) believes that trypanosomatidae begun as gut parasites of annelids and hence adapted themselves to vertebrate hosts - fishes, amphibians and reptiles via the leeches. In the latter, they developed in the anterior station. Insect trypanosomes originally developed in the posterior station and used the stercorarian mechanisms (as in G. palpalis) to transfer these parasites to the vertebrate host. The pathogenic African trypanosomes were thus first acquired by mammalian hosts from leeches and then found in Glossina already found carrying trypanosomes in the posterior station. This change in habitat could only have occured under humid

conditions where the leeches are common. Forest pigs seem to fit this requirement. Wild suidae among the susceptible animals are better adapted than are most other species save perhaps the buffalo to trypanosome infection. Experimental infection only causes transitory parasitaemia while antelops easily yield to the disease. Thus it is suggested that the Glossina acquired anterior station trypanosomes from forest pigs and from them turned their attention to warthogs while open country animals became secondary hosts of tsetse or the morsitans group after Glossina had invaded the savannahs in the wake of warthogs.

2.4. FATE OF TRYPANOSOMES IN THE INSECT VECTOR

Trypanosomes ingested with the blood meal pass to the gut of Glossina and are not digested, they undergo morphological changes and gain the crithidial form (Richardson, 1957). They now look like members of another genus belonging to the Kinetoplastida called CRITHIDIA. Here the Kinetoplast (the small deeply staining body near the root of the flagella) is placed infront of the nucleus whereas in all other stages of the cycle it lies behind the nucleus. Metamorphosis continues and the crithidial forms give rise to metacyclic trypanosomes which when injected into the vertebrate host blood stream (when Glossina is feeding) may establish themselves and complete the cycle by becoming blood forms.

The development of trypanosomes in Glossina differs with the different subgenera. Thus the development of T. congolense follows the foregoing pattern.

The blood forms begin their transformation in the gut after they have been ingested via a blood meal. Here they become elongated and move forward to the proventriculus and hence to the proboscis via the oesophagus. They then get to the hypopharynx where they change to metatrypanosomes (metacyclics) and are ready for innoculation into the vertebrate host. The development of Duttonella trypanosomes

is the simplest (Cmuse, 1973) as the ingested forms attach themselves to the proboscis wall and undergo metamorphosis into crithidial forms (epimastigotes, 16 - 35 mm in size). Here they multiply and give clusters of flagellates which finally detach themselves from the proboscis wall to invade the hypopharynx. They then become preinfective trypomastigote forms which later change to metatrypanosomes which resemble blood trypanosomes and are infective to the vertebrate hosts. This process takes 5 - 13 days but its partly dependent on temperature. Those forms of T. vivax that are taken beyond the proboscis into the digestive tract of the tsetse fly are digested. The trypanosome must undergo the whole lifecycle before it becomes infective except in cases of mechanical transmission. Female Glossina live longer than their male counterparts and work done by Harley (1967) showed that females had higher infection rates than males. Ford (1971) states that a greater number of infected flies (G. palpalis) was obtained when they (flies) were fed on various laboratory animals when the first meal was infected, hence suggesting that the younger the fly, the more easily it was infected. Work done by Snow and Tarimo (1981-personnal communication) showed that females had infection rates of 8.3% with the ratio of Trypanosoma congolense to Trypanosoma vivax

being 1:1. Males had infection rates of 6.3% and the ratio of the two trypanosomes being 5.5:1. These results agree with Harley's (1967)..

Work done by Omuse (1973) reveals that infection rates in Glossina pallidipes are consistently high in the Coast province of Kenya followed by G. longipennis, G. austeni and G. swynnertoni. Infection rates with T. vivax are higher than with T. congolense, this being attributed to the simpler life cycle of T. vivax. However, cattle are mostly found infected with T. congolense (Omuse, 1973). Kramer (1966) working in West Africa found a higher incidence of T. vivax in goats than that of T. congolense.

According to Hoare (1972), the systematic classification of the genus trypanosoma and its relation to the allied flagellates is as follows:

Phylum: Protozoa

Subphylum: Sarcomastigophora

Superclass: Mastigophora

Class: Zoomastigophorea

Order: Kinetoplastida

Suborder: Trypanosomatina

Family: Trypanosomatidae

Genus: Trypanosoma

The parasites in the genera of the family trypanosomatidae are digenetic in that their life cycle alternates between two hosts (Hoare,1972). The lifecycle is comprised of the following stages:

a) Amastigote

b) Sphaeromastigote

c) Promastigote

d) Epimastigote

and e) Trypomastigote. While trypomastigotes are typical forms occuring in the blood of the vertebrate host, trypanosomes may assume any of the above five stages which appear in different combinations at various stages of their digenetic life cycle, both in the vertebrate and in the invertebrate hosts.

The mammalian trypanosomes fall into two major sections characterised by the mode of their development, primarily in the vector and secondarily in mammalian host.

The sections are:

A) STERCORARIA:

This section comprises species, whose whole development in the host is in the fecal medium of the vector in the posterior station and transmission

is contaminative. In the trypanosomes of this section free flagellum is always present, the kinetoplast is large but not terminal and the posterior end of the body is pointed. Reproduction in the mammalian host takes place in amastigote or epimastigote stages and thus is discontinuous. Development in the vector is completed in the posterior station with formation of metatrypanosomes. The trypanosomes in this section are typically nonpathogenic except for T. cruzi.

According to Ford (1971) the subgenus in this group includes the following:

Subgenus	type - species
Megatrypanum	Trypanosoma theileri
Herpetosoma	T. lewisi
Schizotrypanum	T. cruzi
Endotrypanum	T. schaudinni

B) SALIVARIA:

The trypanosomes in this section complete their lifecycle in the insect-vector in a salivary medium and their transmission is inoculative. Their reproduction in the mammalian host is continuous taking place in the trypomastigote stage while the development in the vector is completed with formation of metatrypanosomes.

The subgenus in this section includes:

Subgenus	type - species:

Duttonella	T. vivax
Nannomonas	T. congolense
Trypanozoon	T. brucei (also includes T. evansi; T. equiperdum)
Pycnomonas	T. Suis.

A description of two of the above type species would be inorder in view of their importance and commonness in livestock. These are T. congolense and T. vivax (Omuse, 1973; Griffin, 1978; Snow, 1979).

The vivax group is presented by monomorphic forms in which a free flagellum is always present. Posteriorly the body is typically rounded. The kinetoplast is large and usually terminal. Development in the vector takes place in proboscis exclusively. The trypanosome is relatively large (20 - 26 mm in length) and in fresh blood films it swims with great rapidity across the microscope field. Hence its previous name T. cazalboui (Ford, 1971). It does not easily infect small laboratory animals but it infects all large domestic animals except dogs and pigs (Ford 1971). The same author contends that the disease in sheep and goats is mild as the organism in these animals shows low virulence.

Trypanosoma congolense is smaller than half the length of T. vivax (9 - 18 mm). It does not

have a free flagellum and possesses a medium sized kinetoplast. In wet blood preparation it does not move rapidly across the microscope field and it tends to be a focus of turbulence among the red blood corpuscles. The blood forms in this subgenus (Nannomonas) are pleiomorphic. They vary in form and size with complete continuity of intermediate forms but with extremes which are morphologically quite distinct. The kinetoplast is subterminal or terminal in position. The undulating membrane in most cases is inconspicous but the intracytoplasmic position of the flagellum typically arises somewhat behind the kinetoplast before emerging from the opposite side of the body. The posterior part of the body is either rounded or obtusely pointed in the longer forms.

The crithidial forms in the insect vector may be up to 40 um in length while the epimastigotes that develop into metatrypanosomes are 14.2 - 36 um long. In 1961 a worker in Nigeria with strains of T. congolense that occur there divided them into three main forms in correlation with their pathogenecity.

(1). Small forms of T. congolense with a mean length of 11.2 - 13.8 um characterised by low infectivity, virulence and parasitaemia

(ii). Intermediate type with a mean length of 13 - 15 um characterised by high infectivity parasitaemia and low virulence

(iii).Long dimorphon type with a mean length of 13.8 - 15.6 um, characterised by high infectivity, virulence and parasitaemia.

For the purpose of morphologic observations of the trypanosomes, the best stain is Giemsa (Mulligan, 1970) while Leishman's and Wrights stains may also be used. In natural infections, pathogenecity of the strain is not fixed and may undergo modification through passage via certain hosts or the vector.

Among the domestic animal species under field conditions cattle are perhaps the most seriously affected (Ford,1971). In West Africa, the Mturu and N'Dama breeds of cattle show marked resistance to infection especially when bred in areas endemic to trypanosomiasis (Weitz, 1970; Roberts and Gray,1973; Griffin and Allonby 1979b). This sort of innate resistance is termed as trypanotolerance. Various causes have been attributed to this form of resistance. Thus Parkin (1935) and Fiennes (1946) stated that calves were more resistant than adult cattle. Fiennes (1970) indicated that the ability of trypanotolerant breeds to withstand disease when they are adults depends on infections at calf-hood

and constant exposure thereafter, This suggests that, the tolerance is acquired and not innate. However, a different view which bases trypanotolerance as genetic has been expressed by several workers. Stewart (1951) while trying to relate dwarfism of the West African short horn cattle to trypanotolerance urgued that since dwarfism is readily inherited, then there must be genetic basis for trypanotolerance. Chandler (1952) while working on the comparative tolerance of West African N'Dama cattle to trypanosomiasis demonstrated that the tolerance of the N'Dama X Zebu was intermediate between the tolerance showed by the N'Dama and Zebu breeds of cattle. Stewart (1951) regarded trypanotolerance as resistance and not immunity as it could break down with such stress factors as constant bleeding; overwork, helminthiasis, Vitamin deficiency, and malnutrition. Similar views have been upheld by such other workers as Stephen, (1966); Kortelmans and Kageruka (1976).

Work done in small ruminants (Griffin and Allonby 1979b) also indicates that the small indigenous East African goats are more trypanotolerant than other breeds of goats which have not lived for a much longer time in evolutionary terms in trypanosomiasis endemic areas. The above workers were unable to demonstrate any correlation between the haemoglobin type and trypanotolerance. However, they agree that

there is a genetic basis for trypanotolerance since the intermediate breeds between the indigenous and the pure exotics showed an intermediate degree of trypanotolerance as indicated by such parameters as Packed cell volume (P.C.V.), mortality rates, and weight gain.

The disease syndrome in both cattle and goats infected with T. congolense seems to take similar courses. The disease can either take an acute, subacute or chronic course (Omuse, 1973; Anosa and Isoun , 1974; Griffin and Allonby, 1979a). When sheep and goats are herded together with cattle, they rarely get infected as tsetse flies seem to prefer cattle for their meals (Kramer, 1966). When goats are herded alone, they appear to come down with acute infection as has been observed at Kiboko in Machakos District of Kenya in the UNDP/FAO Sheep and Goat Project farm. Omuse (1973) reproduced the disease in 12 goats all of which died in 7 - 10 days post-infection after showing trypanosomes scantly in blood smears. In 1967, the Kenyan Department of Veterinary Services reported deaths (up to 10%) in goats due to trypanosomiasis. These deaths were due to T. congolense or T. vivax or both.

2.5. CLINICAL SIGNS:

The pathology and pathogenesis of the disease as it occurs in cattle has been studied (Fiennes, 1954; Omuse, 1973; Losos et al. 1973). In sheep and goats three clinical syndromes have been recorded as the disease occurs with a natural infection by T. congolense (Griffin and Allonby, 1979a) Fiennes (1954) also recorded four disease syndromes in cattle but based them on the pathogenic processes taking place in the host system.

The clinical syndromes were described by Griffin and Allonby (1979a) as follows

(i). Acute - All the infections lasting up to six weeks from the first appearance of the parasites in a thick blood smear.

(a) Fatal - this was characterised by a rapid decrease in P.C.V. (to below 15% in some cases), fluctuent body temperature and little loss in body weight due to the rapidity with which death ensued.

(b) Self-cure - there was a slight elevation of the body temperature and a decrease in P.C.V. The parasites appeared in the peripheral circulation briefly but no deaths occured. There was little change in body condition and the outcome was recovery.

(ii). Sub-acute:- In this group, infections lasted between 6 weeks and 12 weeks after the initial appearance of parasites.

(a) Fatal - In such cases the P.C.V. showed a steady and marked decline remaining between 15 and 20%. The temperatures showed wide fluctuations from the onset of the disease but remained high prior to death. There was a marked weight loss at the beginning of the disease condition but remained steady latterly. There was accompanying emaciation and lethargy and the outcome was fatal.

(b) Self-cure - The animals which effected self-cure usually showed a decline in P.C.V. and body weight during the period of detectable parasitaemia. These parameters however reverted back to normal. Any loss of condition that occured was followed by improved condition and was however little. There was a brief period of elevated temperatures but was short-lived.

(iii).Chronic:- The animals in this group had a period of parasitaemia lasting over 12 weeks. There were wide temperature fluctuations with occasional peaks of up to 40.5^{o}C occuring every 5 or 6 weeks. The period of detectable parasitaemia was intermittent. The P.C.V. showed an initial decline but remained between 20 and 25%. The body weights showed an initial decline but became steady later on. The animals became emaciated with dry and starring hair coats and the final outcome was death.

In general, most workers have reported clinical observation of anaemia, decrease in body weight emaciation and fever (Stephen, 1970; Wellde et al. 1974; Griffin and Allonby, 1979a). Subnormal temperatures at or near the time of death have been recorded by Wellde et al. (1974) along with dyspnoea. Harsh coughing was reported in goats by Kaaya (1975) while Kramer (1966) reported that in his survey of trypanosomiasis incidence in small ruminants, pneumonia was prevalent concurrently with trypanosomiasis but it was not clear which one came first. Griffin and Allonby (1979a) recorded anorexia and lethargy in fatal cases of the disease in sheep and goats. Not many workers have reported the swelling of superficial lymph nodes but has been reported by Kaaya (1975) and van den Ingl et al. (1976) in cases of T. vivax infections in goats. In West Africa, Bungener and Mehlitz cited by Griffin, (1978) reported that T. brucei caused more severe disease in dwarf goats while T. vivax and T. congolense only caused mild infections with no clinical signs and only slight histological changes. This is not applicable in the East African situation as negated by the work of Omuse, (1973); Kaaya, (1975); Griffin and Allonby (1979a). Isoun and Anosa (1974) while working with T. vivax infection in sheep and goats showed that there was loss in weight and decrease in red

blood cell indices. Trypanosomiasis as well as causing severe lesions in small ruminants also caused considerably higher establishment of the worm burden and hence this makes the disease (trypanosomiasis) more important where both diseases are endemic, (Griffin, 1978).
van den Ingh et al. (1976) reported that T. vivax in goats caused haemorrhages, oedema and necrotic changes and attributed this to thrombi formation. A similar observation was made by Fiennes (1970) who attributed tissue degeneration to thrombosis in the small blood vessels. This may well agree with observation of thrombocytopenia made by Bruce et al. (1978) in cattle infected with T. congolense.

Little work has been done to determine the effect of trypanosomiasis on the reproductive performance in either large or small ruminants. Stephen (1966) while making observations on the resistance to trypanosomiasis of the N'Dama and Zebu cattle observed that infections with trypanosomiasis at an early age caused failure to reach sexual maturity. Paikne and Dhake (1972) reported abortion in a she buffalo due to T. evansi. Roberts and Gray (1973) reported that chronic trypanosomiasis in cattle was accompanied by underdevelopment of the reproductive system in both males and females. An experimental infection

with T. vivax in sheep and goats carried out by Isoun and Anosa (1974) showed that in males, there was an increase in production of abnormal spermatozoa from 5.2% before infection to 24.6% after two weeks, a rise of up to 28.3% after 4 weeks and 6 weeks latter the rise was 24%. Other changes included testicular atrophy, scrotal alopecia with areas of degeneration and calcification in the testicles. Histologically they reported degeneration and fibrosis of the seminiferous tubules. Similar observations were made by Kaaya (1975) and , Kaaya and Oduor (1980) while working with an experimental infection of T. congolense in goats. Isoun and Anosa (1974) demonstrated the presence of T. vivax in ovarian smears and in amniotic fluid of one pregnant ewe.

2.6. PATHOLOGY:

(i) HAEMATOLOGY

In both large and small ruminants, the most outstanding feature is the reduction in Packed Cell Volume (P.C.V.) in both acute and chronic cases of T. congolense infection. Leukopenia has also been recorded in T. congolense infection in cattle by Losos et al. (1973) and Wellde et al. (1974). While on the other hand Kaaya (1975) reported slight leukocytosis in goats. Wellde et al. (1974) also found many chromatophilic red blood cells and normoblasts in the blood early in the course of developing anaemia. Omuse (1973) observed morphological changes in the red blood cells which included anisocytosis, polychromasia and basophilic stippling. He also observed a decrease in the percentage of segmented neutrophils during the early period of infection. Erythrophagocytosis has been observed in cattle by Connal (1912), Fiennes (1954), Mackenzie et al. (1978), and Griffin (1978). The mechanism inducing phagocytosis of the red blood cells has been shown to be the coating of the red blood cells with trypanosome antigen so that they are recognised as foreign (Mackenzie et al. 1978). While erythrophagocytosis is accepted as one cause of anaemia, other causes have been agreed upon. Thus hemolysis also contributes to anaemia (Fiennes, 1954; Zuckerman, 1964; Kaaya et al. 1977; Mackenzie et al. 1978). Inhibition of reticulocyte maturation

from the bone marrow has been postulated as a cause of anaemia (Kaaya et al. 1977; Mackenzie et al. 1978). Haemodilution has been reported as a possible cause of anaemia in view of increased plasma volume due to a rise in gamma globulin levels exerting colloidal osmotic pressure (Clarkson, 1968; Anosa and Isoun, 1976).

Thrombocytopenia has been reported in cattle infected with T. congolense (Bruce et al. 1978). This phenomenon has not been reported in sheep and goats. Zuckerman (1964) suggested that antigen antibodies were mediators of thrombocyte destruction.. However, Davis et al. (1974) while working with T. rhodesiense in rats showed that thrombocyte aggregation occured in vitro when trypanosomes or supernatant containing lysed trypanosomes were added to thrombocyte suspension.

An indirect evidence for the participation of thrombocytes in the pathogenesis of trypanosomiasis is a decline in blood serotonin levels (Slots et al. 1977) as has been shown in goats infected with Trypanosoma vivax by van den Ingh et al. (1976). T. vivax complexes with the antibodies have been shown to cause an increased release of serotonin from prelabeled goat platelets (Slots et al. 1977).

(ii) SERUM BIOCHEMISTRY:

In cattle experimentally infected with T. congolense, there was a decrease in total serum proteins for the first 5 weeks (Fiennes, 1970; Wellde et al. 1974). Kaaya (1975) while working on experimental infection of goats with T. congolense did not observe any change in total serum proteins. The decrease in total serum proteins has been attributed to an increase in plasma volume resulting from an increase in gamma-globulins (Clarkson, 1968; Fiennes, 1970; Anosa and Isoun, 1976). Other causes of low serum proteins are an increased protein breakdown, loss by proteinuria due to glomerulonephritis (van den Ingh et al. 1976) and disturbances in metabolism or absorption (Wellde et al., 1974). Serum urea nitrogen was found to increase in both cattle and goats to small or moderate levels (Fiennes, 1970; Omuse 1973; Wellde et al., 1974; Kaaya, 1975). Infection with T. vivax in sheep showed an elevation in gammaglobulin levels (Clarkson et al., 1966). In cattle infected with T. congolense, Wellde et al., (1974) reported an early fall in transaminases but which remained stable afterwards except for one case. In goats with T. congolense infection, no significant change was found in transaminases which could be

attributed to the disease (Kaaya, 1975). In chronically infected cattle, serum creatinine levels tended to be lower while serum glucose levels remained constant except for terminal values which were reduced acutely in infected animals (Wellde et al., 1974).

Serum bilirubin values were raised in acutely infected cattle but not in chronically infected ones (Wellde et al., 1974). In goats infected with T. congolense, total serum bilirubin values were raised during the intermediate stages of the disease (Kaaya, 1975).

(7) POST-MORTEM LESIONS:

No pathognomonic lesions have been noted either in T. congolense or T. vivax infections. Losos et al. (1973) reported macroscopic evidence of increased activity of the bone marrow in the femur while Wellde et al. (1974) reported that the bone marrow from the ribs of cattle infected with T. congolense was red while that from the femur was yellow and fatty in consistency. In goats killed in the acute stage of infection with T. vivax, van den Ingh et al. (1976) reported hyperactivity of the bone marrow. Kaaya (1975) reported finding pink bone marrow in goats infected with T. congolense. Other gross findings include gelatinous fat atrophy at the auriculo-ventricular farrow and the perirenal area (Losos et al. 1973; Kaliner, 1974; Kaaya, 1975). The thoracic cavity contains straw-coloured fluid (Kaliner, 1974; Kaaya, 1975; van de Ing et al 1976). Haemorrhages in the lungs, brain and the heart surface have been reported (Kaliner, 1974) while van den Ingh et al.,(1976) also observed haemorrhages on the subpleural space.

Histologically, an abudance of normoblasts, leukocyte precussors and megakaryocytes were observed by Wellde et al. (1974) in the bone marrow. However, Losos et al. (1973) reported little evidence of leukopoiesis,

generalised hemosiderosis and absence of immature erythrocytes in the peripheral circulation hence suggesting extensive destruction of the erythrocytes without an adequate compensatory stimulation of the erythropoietic tissue. Kaaya (1975) made similar observations in goats where maturation arrest of the erythron cell series was at the prorubricyte/rubricyte stage. Yellow pigment (hemosiderin) has been reported in the macrophages of the spleen, bone marrow and lymph nodes (Wellde et al. 1974; Kaaya, 1975; van den Ingh, 1976; Mackenzie et al. 1978). The above phenomenon (erythrophagocytosis) has been held responsible for anaemia even in cases of human trypanosomiasis. Cells of the plasma series show proliferation in the spleen and haemolymphnodes (Losos et al. 1973) thus suggesting that immunological responses are primarily confined in the lymphoid tissues exposed directly to the parasites in the blood. Similar observations were made by Kaaya (1975). A detailed histological report was made by Kaliner (1974).

In the kidney, he reported invasion by various cells especially the cortex. These cells are histiocytes, lymphocytes and plasma cells. Most of these cells were opposite the vascular pole of the glomeruli but some were also present at the wide interstitium and juxtavascular. Some kidneys had hemosiderin in the tubular epithelium. Kaaya (1975) reported swollen glomeruli, hypercellularity and protein casts in the tubules. van den Ingh et al. (1976) reported the presence of proteinatious fluid in the kidney tubules along with hydronephrosis. Fiennes (1950) found T. congolense acting as a tissue parasite in the adrenal cortex and the anterior pituitary gland. Losos et al. (1973) also found aggregations of trypanosomes in the brain and skeletal muscles of 2 out of 5 cattle. However, observation of the trypanosome acting as a tissue parasite was not a constant finding in both cases, thus

any endocrine lesions were attributed to the direct action of the parasite on the gland. Kaaya (1975) observed T. congolense in the blood vessels of the myocardium, brain, kidney and a few in the liver but none were found outside the blood vessels.

In the lungs, Kaliner (1974) observed atelectatic and emphysematous areas. Non-inflammatory fluid was found in the alveoli while the alveolar septa was thickened with varying degrees of hemosiderin being found in the cells of the interalveolar and adventitial connective tissue of the lungs. The veins had accumulated macrophages with iron pigment while pathologic changes were also found in the walls of blood vessels. The intima was thickened and the tunica elastica was frequently divided, thickened and sometimes partially or totally lysed. The media appeared hypertrophic, swollen or necrotic with moderate to severe constriction of the lumen in some instances. The changes apparently began at the subendothelial zone, in some arteries there was partial loss of media. The nuclei of the muscle

cells of the media were often pyknotic and the adventitia was frequently loosened and thickened. However, van den Ingh et al. (1976) observed microthrombi consisting of thrombocytes, trypanosomes and monocytoid cells in T. vivax infections in goats. In the liver, ferriferous pigment was observed along with fatty infiltration of the hepatocytes consisting of fine to large droplets radiating from the central vein. Sinusoids in parts of the organ were dilated. Losos et al. (1973) and Kaaya.(1975) reported centrilobular necrosis. The latter also observed swollen Kpuffer cell nuclei and increased white blood cells in the sinusoids of goats experimentally infected with T. congolense.

In the brain Kaliner (1974) observed extravasation containing macrophages and leukocytes. The veins were dilated and filled with macrophages which had accumulated iron pigments. The perivascular infiltrations were composed of histiocytes, plasma cells, lymphocytes and occasional eosinophils. Single juxtravascular glial nodules were seen. The leptomeninges of some cattle were infiltrated with the same type of cells as the perivascular zone. No thrombi were seen here either, trypanosomes or segments were seen in the blood vessels but not in parenchyma.

Losos et al. (1973) observed focal encephalomalacia affecting the grey matter of the apex of gyri involving particularly the granular and pyramidal layers and not the underlying white matter.

In the spleen, hemosiderin but no hyperplasia was observed by Kaliner (1974). Kaaya (1975) however observed hyperplasia of the red and white pulp, increased plasma cells, macrophages and lymphocytes in the red pulp. Heavy deposits of haemosiderin were observed in the macrophages in the spleen in goats infected with T. congolense.

In the lymph nodes, slight to moderate fibrosis was observed while some animals showed sinus catarrh while in others lymphoid follicles were small, scanty and often the pale centre was missing indicating atrophy (Kaliner 1974).

2.8. DISEASE DIAGNOSIS:

The only certain way of diagnosis of trypanosomiasis is by microscopical demonstration of trypanosomes (Weitz, 1970). However, this is not always possible and hence reliance on other methods is advocated.

The situation in which the herd is found and herd history may lead one to suspect trypanosomiasis (Hornby et al. 1931). Clinical signs of chronic emaciation,

absence of anorexia and anaemia also offer a reliable guide to diagnosis. Giving the affected animal treatment with a drug that has specific action against the trypanosome organism with subsequent recovery of the animal confirms the disease. In such a case control animals would be necessarily.

Demonstration of the organisms by use of blood smears. There are three types of blood smears that can be used.
These are:

i). Wet films

These are quick and easy for early cases of T. vivax and T. congolense but T. brucei infection may be overlooked. It is however of little value in bovine trypanosomiasis in view of the many cases that would be missed. (Robson and Ashkar, 1972).

ii). Thick films

These are good for routine diagnosis of trypanosomiasis in the field but frequently, distortions are caused during preparation and staining and hence the species of the parasite cannot be determined very accurately. This method is more accurate than the thin film method as the blood may be 20 X in volume compared with thin blood film.

iii). Thin blood films are only good for identification but not for primary diagnosis.

Haematocrit centrifuge technique involves the observation of rippling movements from a fresh blood sample. The blood on centrifugation is divided into its three main components (red blood cells, white blood cells and plasma), the trypanosomes can be observed on a glass slide after breaking the capillary tube at the junction of the buffy coat and the red blood cells so that the contents of the buffy coat can be examined under the microscope after placing the coverslip on. When this method was used in sheep by Kobayashi et al., (1976) it could detect infections in the first weeks of innoculation with T. congolense. In the later stages, the parasites could be detected intermittently even with the mouse innoculation technique. However, it is good when the trypanosomes are in low numbers but requires good laboratory facilities such as centrifugation or ion-exchange absorption techniques. It is particularly good for flagellated trypanosomes such as T. vivax.

Animal innoculation method requires that susceptible laboratory animals such as laboratory-bred white rat be innoculated intraperitoneally with heparinised blood from the suscepted animal (Stephen, 1970). The organisms are then demonstrated from the blood of the laboratory animals. This method has been recommended for demonstration of T. congolense and T. brucei infections by Robson and Ashkar, (1972).

A Nigerian technical assistant cited by Stephen, (1970) has described a method by which the rats were innoculated intraperitoneally with infected blood and 10 - 15 minutes latter, a splenic puncture was made through the thin body wall and examined as a wet and stained film. This method demonstrated trypanosomes when other rapid tests were negative. In T. gambiense infection in man, lymph node puncture has been used as a method of diagnosis, but Stephen, (1970) doubts its value in the diagnosis of animal trypanosomiasis. However, it has been recommended in the diagnosis of T. vivax infections (Robson and Ashkar, 1972; Boots Company Ltd).

Xenodiagnosis is a diagnostic method which involves feeding clean laboratory-reared Glossina on suspected animals and then allowing them to feed on clean susceptible animals which are bound to come down with the disease or the Glossina are dissected after a period of cyclical development of the trypanosomes in the vector to demonstrate the organisms. This method has been used in South and Central America (Stephen, 1970). However, it is not a practicable method for routine diagnosis of animal trypanosomiasis in Africa. It is however a useful research tool for separating trypanosome species in mixed infections or establishing pure infections.

The serological tests available for diagnosis of trypanosome infections are many and include, red cell adhesion; agglutination test, trypanolytic reaction, precipitin test, immunoconglutinin level, immunodiffusion reaction, complement fixation test (CFT) and indirect florescent antibody test (IFAT). When the latter (IFA) test was used by Zwart et al. (1973) in Kiboko area (Machakos - Kenya) to diagnose trypanosomiasis in sheep, cattle and goats, it was found that 80% of the cattle were positive for T. vivax, T. congolense and T. brucei while the wet, thick, thin blood smears and mouse innoculation showed only 15% of the cattle being positive. The serological tests have their own limitations of not differentiating the infecting trypanosome species and not being able to detect early infections. The CFT has been used to detect dourine in horses where no other trypanosome infections except with T. equiperdum are present since other species will too give a positive reaction.

Chemical tests have been used in diagnosis of Surra in Camels but are not however specific for T. evansi since they simply indicate a rise in globulin levels.

Culture techniques are of little value in disease diagnosis in domestic animals since the African trypanosomes that are pathogenic to domestic animals have not been successfully propagated in artificial culture media. T. theileri has been successfully cultured but this is of little practical significance (Stephen, 1970).

Isoun and Isoun (1974) successfully cultured the West African isolates of T. vivax in mammalian tissue culture but attempts by Cawdery (1958) to culture T. vivax in embryonated eggs was impossible along with those of Baker (1958) to infect white rat with T. vivax. Hirumi et al., (1977) and Nyindo et al., (1978) have reported successful cultivation of Trypanosoma brucei in Roswell Park Memorial Institute (RPMI) 1640 medium.

At Post-mortem, examination of fresh and stained material and then innoculating suitable specimens into laboratory rats and mice are aids to diagnosis.

The filtration technique for diagnosis of trypanosomiasis:

This technique is a modification of the method of Lanham and Godfrey, (1970) for the separation of trypanosomes from host blood cells. It works on the principle that as the infected blood is passed through a DEAE-cellulose column, the negatively - charged blood cells will stick to the positively charged cellulose while the less negatively charged trypanosomes will pass straight through and can be collected as a pure suspension (Gibson - 1981, personal correspondence). For diagnostic purposes, a very small column is used and the suspension of trypanosomes collected is centrifuged so that any cells that pack at the bottom of a sealed pasteur pipette are then examined under a microscope for the demonstration of the parasites.

2.9. IMMUNITY:

There is no practical vaccination method which is as yet available. This is so because organisms such as T. vivax and T. congolense do not yield enough material even when in circulation which could be made into a vaccine. This is further hampered by the trypanosome ability to change the antigenic structure (Weitz, 1970). This phenomenon provokes the body to produce specific antibodies to each variant. Relapse variants of one strain have some antigens in common and hence partial suppression of the infection is brought about by an antibody response to those common antigens. However, no protective action by these antibodies has been demonstrated (Brown, 1966). The host immune responses involve a rise in the gamma-globulin content, (Brown, 1966; Clarkson et al. 1966; Clarkson, 1968; Kaaya, 1975 and Anosa and Isoun 1976), and an increase in the level of brandykinins (Brown, 1966; Boreham, 1966; Kaaya, 1975; van den Ingh et al., 1976).

Thus no evidence has as yet shown unequivocally an acquired protection after recovery from the disease either in animals or in man. The new possible approach to the protection of non-resistant animals in the field is based on chemoprophylaxis to induce infection - treatment resistance to trypanosome

infections (Stephen, 1966; Boots Company Ltd; Fiennes, 1970; Griffin and Allonby, 1979c).

2.10. CHEMOTHERAPY AND CHEMOPROPHYLAXIS OF TRYPANOSOMIASIS.

The need to control trypanosomes and the tsetse fly vector has been necessitated by population pressure which has forced people and cattle to move into fly belts not previously used (Boots Company Ltd.). Until 1930's the only drug available for treatment of trypanosomiasis was the inefficient tartar emetic. This drug is unreliable and only finds utility for its cheapness. However, the earliest drug to be used against trypanosomiasis was trypan red from which was developed trypan blue and hence afridol violet (Link, 1965).

As far back as 1857, David Livingstone records the use of arsenic for treatment of trypanosomiasis in horses. The mode of action is inhibition of enzymes such as hexokinase, adenosine triphosphatase and pyruvate kinase. Its toxic side effects are ocular lesions following treatment with pentavalent arsenicals, blindness resulting from degeneration of ganglionic cells of the retina and possibly due to vasoconstriction.

In monkeys and rabbits, the arsenic compounds have been shown to produce haemorrhagic and necrotic lesions at a number of sites in the

central nervous system. Further lesions produced in the cerebella and white matter involve considerable proliferation of glial cells. Arsenic compounds are no longer of any significance in trypanosomiasis control.

Antimony compounds act by inhibiting enzyme systems involved in respiration and glycolysis. These compounds are tedious to administer and have to be given intravenously as a 4% solution dissolved in physiological saline.
Tartar emetic can cause immediate violent reactions and can sometimes kill. However, it is scarcely or not used at all these days.

Naphthalene derivatives such as Suramin were developed from afridol violet and first came in to use in 1920. It's modes of action are inhibition of enzymes such as hyaluronidase, fumarase, urease and hexokinase, stimulation of RNA polymerase and AMP production, binding to cellular proteins such as protamine and inhibition of infectivity of trypanosomes. Suramin is administered subcutaneously. The toxic effects attributed to it include a tuberculosis promoting effect, toxic degeneration of the liver, kidneys and spleen. The toxic degeneration is most marked in the adrenal glands. Equidae are very susceptible to these effects. It is active against T. brucei, T. evansi and T. equinum

infections. T. vivax and T. congolense are resistant to suramin per se. However a compound derived from decamethylene bis (Iso-quinolinium) and called substance II by Austin et al., (1957) and which had prophylactic activity against T. congolense was potentiated when mixed with suramin. Similar potentiation with suramin has been observed by Williamson (1966) with a nucleoside antibiotic puromycin (stylomycin) and puromycin aminonucleoside. Potentiation was against a suramin - fast strain of T. rhodesiense which showed unexpected collateral hypersensitivity. Suramin acts as an anion and can form compounds (suraminates) with therapeutic or prophylactic agents acting as cations. Such agents are antrycide, ethidium, berenil (diminazene) and prothidium. Such compounds have greater prophylactic activity than suramin alone with the toxicity of the cation fairly reduced. However, suramin is still used in controlling T. evansi infections in camels.

The first phenanthridinium derivative (dimidium bromide) was discovered in 1938 and first came into use in Africa in 1940 (Boots Company Ltd; Link, 1965). Other phenanthridinium derivatives are ethidium (homidium bromide); Novidium (homidium chloride); Prothidium, metamedium and isometamedium chloride (samorin). Ethidium bromide acts by

binding to DNA and RNA and by inhibiting DNA and RNA polymerase. It acts against T. congolense and T. vivax infections but not or only little against T. brucei. It does not act against T. evansi or T. simiae.

The drug is administered subcutaneously as a one percent solution at the dose rate of 0.99 mg/kg body weight. This preparation will protect cattle against reinfection with the two species of trypanosomes for a period of four weeks. The drug causes painful swellings at the site of injection and a scar tissue may result. The drug causes death in cattle when administered at 10 times the therapeutic dose (TD). Death follows extensive liver damage. Cattle in poor condition will die when the drug is given at the dosage 5 X therapeutic dose. Ethidium Suraminate provides a protection lasting 6 - 12 months against T. congolense and T. vivax infections. Considerable local reaction can occur in case of subcutaneous injections. When ethidium bromide is given at a dosage lower than the therapeutic one, relapses may occur.

Novidium acts against T. congolense and T. vivax infections. This drug may cause photosensitization at its site of injection in light coloured animals along with local swellings. Prothidium (Phenanthridinium-Pyrimidinium) exerts its action

by stimulation of adenosine monophosphate (AMP) production and also causing cation loss. It is administered at the dosage rate of 2.0 mg/kg body weight subcutaneously at the dewlap or intramuscularly.

The delayed toxicity of dimidium and phenanthridinium was studied in cattle by Burdin (1953) and the changes observed were mainly lacrymation, hyperemia and serous exudation from the muzzle. There was dermatitis involving the face, ears, base of the horn and dewlap. Hardening and cracking of the skin at the thighs arms, vulva and udder. Dimidium bromide administered at the dose rate of 1 mg/kg body weight in Sotik area of Kenya gave a positive icterus test (-Van de Bergh test). At Post-mortem there was fatty degeneration and bile duct proliferation. Similar observations relating to photosensitization were made by Goodwin and Unsworth (1952) while studying the prophylactic and toxic actions of two phenanthridinium derivatives. Photosensitization following dimidium bromide administration may not be wholly attributed to dimidium but may also be aggrevated by a circulating photosensitive compound 'Phylloerythrin'

as in the case of 'Geel dik Kip', a South African disease or 'facial eczema' found in New-Zealand (Burdin, 1953).

Metamedium is a mixture of two isomers which is given intramuscularly at the dose rate of 1 - 4 mg/kg body weight to give protection against T. congolense and T. vivax. Prophylactic doses give protection for 2 - 4 months. Local swellings can result from subcutaneous injections. When metamedium and Suramin are mixed to give metamedium suraminate, the resultant local reaction on injection is minimal and the prophylactic activity is afforded (Bergs, et al., 1961; Gray and Stephen, 1962). Metamedium Suraminate (May and Baker Ltd.) protects cattle for 3 - 5 months against T. congolense and T. vivax infections.

Isometamedium chloride (Samorin) is soluble in cold water and is less toxic than metamedium. It acts against T. congolense and T. vivax infections. It can be used for both prophylactic and curative purposes giving protection for up to 12 weeks in cattle while work done on sheep and goats at Kiboko (Griffin and Allonby, 1979c)shows that the protective period afforded by this drug is 4 months.

Samorin is administered deep intramuscularly. For prophylaxis, the dosage is 0.5 - 1.0 mg/kg body weight but for curative purposes the dosage is 1.0 - 2.0 mg/kg body weight. The drug is made into

a 2 per cent solution and for the purposes of causing least damage to consumable meat injection at the side of the neck is recommended (Griffin and Allonby, 1979c).

Quinoline derivatives.

These derivatives manufactured by Imperial Chemical Industries Ltd. (ICI) include drugs such as, antrycide (Quinapyramine) which has activity against T. vivax, T. brucei, T. evansi, T. equinum, T. simiae and T. equiperdum. It has also activity against T. cruzi in rats. Antrycide methyl sulphate - a yellow powder up to 33% soluble in water is administered subcutaneously, and an overdosage can cause curare-like effects. Antrycide chloride has a very low solubility but a high margin of safety (up to three times the prophylactic dose) and it is therefore a safe prophylactic agent.

Antrycide prosalt is a mixture of antrycide methylsulphate and antrycide chloride and has both curative and prophylactic activities. The combination protects cattle for 2 - 4 months against T. vivax and T. congolense while the mixture protects against T. evansi for up to 10 months.

Antrycide suraminate has a good curative and prophylactic action against T. simiae infection in swine.

Antrycide when used in sheep and goats at the dose rate of 7.4 mg/kg body weight given subcutaneously affords a protective period of 8 months inspite of an increase in fly (tsetse) numbers in an area of medium challenge (Griffin and Allonby, 1979d). The quinoline derivatives exert their action by binding and activating cytoplasmic ribosomes and also inhibiting growth of protozoal organisms.

The toxicity resulting from quinoline derivative administration may be seen within 15 minutes to 2 hours of treatment. The signs are salivation, tremors of the neck and shoulder muscles, incoordination, accelerated respiration and heart rate, collapse and death in a few hours. Chronic drug toxicity is characterised by bloody diarrhoea, progressive weight loss leading in some cases to death in six to twenty days. At post-mortem nephrosis is a constant finding (Link, 1965). The nephrosis results from the fact that antrycide leaves the blood system quickly following absorption and is then found in highest concentrations in the liver and the kidneys. It is present in these organs for several weeks following injection and is excreted via urine.

Aromatic diamidine derivatives are berenil and pentamidine. They were introduced into chemotherapy in 1939. Berenil came to general use in 1956 (Boots Company Ltd.). They act by causing loss of potassium and magnesium ions, causing fragmentation of the kinetoplast, binding to kinetoplast DNA and causing denaturation of trypanosomal protein. The diamidine derivatives are administered intramuscularly or subcutaneously for curative purposes only. Berenil is given at the dose rate of 3.5 mg/kg body weight while pentamidine is given at the rate of 3 - 4 mg base/ kg body weight on alternate or consecutive days to a total of 25 - 30 mg base/kg body weight. Berenil acts against T. congolense, T. vivax, T. brucei, T. equinum and T. evansi infections. It is metabolised in the body and thus remains for a short time in the body giving a short protection against trypanosomiasis. Pentamidine has been found to inhibit the net synthesis of protein, R.N.A., D.N.A. and phospholipid when added to cultures containing Crithidia fasciculata in the exponential phase of growth (Gutteridge, 1966).

Oppong, (1969) reported toxicity of berenil in dogs at a dosage rate of 3.5 mg/kg body weight given subcutaneously. The signs of toxicity were

ataxia, weakness and dyspnoea due to histamine release. At post-mortem, the findings were swollen kidneys, toxic liver and jaundice. Berenil thus attacks the central nervous system causing tremors, nystagmus, ataxia, shaking, cramps, vomiting and even death. Nervous signs have been observed in cattle at the dosage rate of 14 mg/kg body weight. Blood pressure reducing effects are only slight and temporary and the drug shows no local toxicity.

In humans, albinurea has been observed while nephritis has been observed in ponies. Pathomorphology of side-effects of trypanocidic diamidines has been observed by Schmidt, et al., (1978). At high dosage, lesions were caused in the heart, liver, kidneys and small arteries of the brain. Histological findings were cloudy swelling, fatty infiltration and parenchymatous necrosis. The vascular lesions were acute and chronic arteritis which in the brain were associated with plasmatic and haemorrhagic necrosis.

In addition, pentamidine causes hypoglycaemia due to degeneration of alfa-cells of the pancreatic islets.

Berenil could be used with other resistance generating chemotherapeutic agents such as ethidium (a sanative pair). Some resistance to berenil has been known time and again (Jones, Davies, 1960;

Bhathacharjee and Sinha 1971); some of which could be overcome with higher dosages.

Apart from the forementioned trypanocidal agents, other drugs have been used for treatment of trypanosomiasis either in man or laboratory animals but not on a really wide scale.

These drugs include antibiotics such as:-

Puromycin or Stylomycin (Willet 1955/56)

Amphotericin - B (Actor and Rugano, 1962)

Nitrofurans (Robertson, 1959, 1961).

3. MATERIALS AND METHODS:

3.1. PROJECT AREA:

The research was carried out in Matuga sheep and goat multiplication centre which is located off the Coastal Strip South of Mombasa. The area is roughly 4^{o} South of the equator in the hinterland of Kwale District, 200 - 500 metres above sea level. The area has an annual rainfall of 1015 - 1270 mm. The location is classified into ecological zone II whose main vegetation is Acacia cammiphora with forests and derived grasslands and bushes. In the low lying areas the main grass type is Pennisetum purpureum.

The locality was chosen for having suitable goat breeds which could be used for research purposes and also because the area is endemic for trypanosomiasis (Omuse, 1973; Snow, 1979).

3.2. RESEARCH ANIMALS:

The station had three goat breeds, namely Toggenburg X East African, Galla X East African and the East African. These goats had been bought from the sorrounding areas and brought to the centre for multiplication purposes. Their numbers varied with their availability and as such the largest numbers were the East African goats followed by the Galla X East African and the fewest were Toggenburg X East African. The animals did not have a history

of trypanosomiasis prophylactic regime in the past. As the goats were meant for multiplication purposes, the majority were female goats and the few males of each breed that were present were for breeding purposes only.

Before the animals were divided into experimental (treatment) and control groups, the causes of variation in the two main parameters body weight and packed cell volume (P.C.V.) had to be considered. The causes of variation (Carles - 1980 personnal communication) are: Genotype (breed), age of the animal, sex, state of nutrition (dependent on the season), helminthiasis, blood parasites and state of hydration.

Hence, each breed was divided into an experimental and control group, where animals were of varying ages, of one sex (females) and were grazed together. Variation that could have resulted from helminthiasis, blood parasites and state of hydration was ruled out by the management practices.

At the beginning of the experiment, the animal numbers in each breed and group were divided as shown below:

EXPERIMENTAL	CONTROL
50 East African	108 East African
20 Galla X East African	19 Galla X East African
19 Toggenburg X East African	19 Toggenburg X East African

3.3. MANAGEMENT OF EXPERIMENTAL ANIMALS:

At the beginning of the experiment, the centre had three bomas and all were situated far from one another (more than 4 Km apart). Two of the bomas were holding female breeding goats while the third held male weaner goats and sheep. During the day time adult goats were accompanied by herdsmen when they went grazing while young kids were left in the bomas. The goats fed on ordinary grass and browse composed of Acacia cammiphora, Lantana camara and Pennisetum purpureum in the low-lying areas, without any supplimentation. In the evenings, the goats were housed in concrete floored bomas, with timber and wiremesh at the sides. The roof was covered by corrugated iron sheets.

The goats had plenty of treated clean water around the bomas which constantly filled the concrete water troughs. The grazing bushes had artificial dams which could suppliment the pure piped water in case of any problem in the pumping system. In order to control the worm burden (helminthiasis), the goats were drenched on a monthly basis using Ranide (Merke, Sharpe and Dohme Ltd.). This anthelmintic was used as a 2.5%($^{w}/_{v}$)suspension of Rafoxanide at the recommended dose of 2.0 mg/kg body weight. In order to control tick - borne diseases, the goats were dipped once every week

in quintiofos (Bacdip$^{(R)}$) diluted at 1:4000.

Other routine management procedures involved hoof-trimming, daily cleaning of the bomas and water-troughs. Disease conditions that affected the goats were treated accordingly. However, animals in the control groups were never treated for trypanosomiasis.

3.4. COLLECTION OF BLOOD SAMPLES:

From the first day of the experiment, blood samples were taken from both groups of animals and on a two weekly basis thereafter until the end of the experiment. However, as indicated later in the respective graphical results, unavoidable circumstances sometimes reduced sampling to once a month. Sampling was done from the ear vein during the early morning hours before the animals could be released to go and graze. The blood was obtained by pricking the ear vein with a sterile lancet.

(i) PREPARATION OF THICK BLOOD SMEARS:

In the initial stages of the experiment, shortage of essential equipment made it necessary to detect the presence of trypanosomes in the goats by making thick blood smears. Therefore it was impossible to measure the packed cell volume at that stage. A drop of blood from the earprick was put on a clean microscope slide, spread out evenly and then air dried. The red blood cells were lysed by putting the slide in a staining jar containing distilled water in a vertical position for 5 minutes. The slide was then air dried and fixed in absolute methyl alcohol for 3 - 5 minutes. The slide was then rinsed with distilled water, air dried and then stained with Giemsa diluted 1:10 (Medway, 1969; Mulligan, 1970) for 30 minutes. The slide was then rinsed with distilled water, air dried and examined under a compound microscope with the Oil-immersion lens.

When a microhaematocrit centrifuge was secured after the experiment had started, the thick blood smear method of diagnosis was abandoned in favour of the haematocrit centrifuge technique which simultaneously allows the measurement of Packed cell volume. The ear vein was pricked with a sterile lancet as before and the blood was filled

into heparinised capillary tubes by capillary action. On filling, the tube was covered on the upper end with the finger tip and the lower end dipped into plasticine. The finger tip was released and with the capillary tube in a verticle position it was gently pushed into plasticine so that the lower end could be sealed by about 2 mm column of plasticine. The blood was now ready for centrifugation.

The samples were centrifuged using Hawksley's micro-haematocrit centrifuge as recommended by Schalm (1965). After placing the capillary tubes in position, the centrifuge was set to run for 5 minutes at 12,000 r.p.m. At the end of this time, the capillary tubes were removed and the P.C.V. read off using the Haematocrit reader. The capillary tube was placed vertically and the upper plasticine level aligned with the baseline of the Haematocrit reader. The moveable arm rested on the borderline of the red blood cells and the buffy coat (white blood cells). The P.C.V. was read off the margin of the Haematocrit reader.

During the later part of the experiment, P.C.V. was read (measured) using the Hematocrit Reading Chart designed by Dr. George.M. Guest and prepared by Arthur H. Thomas Company.

The P.C.V. was measured by holding the chart at the bottom with one hand, placing the hematocrit tube on the chart with the upper end held lightly between the thumb and the forefinger of the other hand arched over the top of the chart. The tube was aligned as nearly parallel as possible with the vertical lines of the ruled area. The meniscus of the plasma was adjusted to the 100 per cent line at the top. The tube was moved across the face of the chart so as to bring the bottom of the cells (red blood cells) to the zero (0) line. The plasma meniscus was rechecked at the 100% line and the height of the column of the red blood cells read as per cent volume of packed cells in whole blood under a strong light.

(ii) EXAMINATION FOR TRYPANOSOMES:

After reading off the P.C.V., the capillary tube was placed horizontally on a flat surface. The junction of the red blood cells and the buffy coat was sawed using a diamond pencil or a glass slide edge so that breaking at that point became easier. After breaking the tube, the top was covered lightly using the forefinger and a drop of the buffy coat placed on a clean microscope slide. A coverslip was placed on top of the drop. The drop was examined under a compound microscope and observed under X 40 for rippling movements

among the blood cells being made by the trypanosomes. Tentatively T. vivax could be diagnosed by its fast speed across the microscope field as opposed to T. congolense which makes sluggish movements and hardly moves out of the microscope field (Richardson and Kendall, 1957; Ford, 1971).

All slides that did not show any rippling movements after thorough examination were discarded while the positive ones were stained for morphological identification of the trypanosomes.

(iii) STAINING FOR MORPHOLOGICAL IDENTIFICATION OF THE TRYPANOSOME.

The coverslip was removed from the positive smears and the smears spread out on the slide and air dried. They were fixed in absolute methyl alcohol and staining carried out as described for the thick blood smears. Examination was done at X 100 oil-immersion under a compound microscope. Morphological identification was done by using the criteria described by Hoare, (1972); the criteria was size of the trypanosome in relative terms, presence or absence of the free flagellum, degree of development of the undulating membrane, size and position of the kinetoplast, the shape of the anterior end and the position of the nucleus.

3.5. DRUG ADMINISTRATION:

Following the work done by Griffin and Allonby (1979c) on the economic aspects of trypanosomiasis in sheep and goats, samorin was shown to produce the best results with a regard to weight gain. It is also easier to make a solution of it than that of diminazene aceturate. Thus in considering the ease with which T. congolense and T. vivax develop resistance to quinapyramine (Lester, 1950), then isometamedium was selected for the present study.

Since samorin (May and Baker, Ltd. Dagerham) is supplied in powder form, it was made into a 2 per cent solution by dissolving 2 gm in 100 mls of sterile distilled water (supplied as water for injection U.S.P., Mac's Pharmaceuticals Ltd. Nairobi, and preserved in phenol 5%). Injection area was swabbed with 70% alcohol before injection. It was given at the dose rate of 0.5 mg/kg body weight intramuscularly at the side of the neck where least damage could be made to consumable meat (Griffin and Allonby 1979c). The drug was given to all the goats in the experimental group on a monthly basis.

3.6. WEIGHING EXPERIMENTAL ANIMALS:

All the goats in the station were weighed on a monthly basis. They were weighed using the standard sheep and goat weigh bridge (Animatics). Only one weigh bridge was available to serve all the three bomas. The weigh bridge is bulky and therefore requires special transportation facilities. Due to lack of such facilities which could move the bridge from boma to boma, in some months, it was impossible to weigh the goats and their weights for the purposes of drug administrations had to be extrapolated from those of the previous months.

Whenever kidding was taking place, a smaller balance (Bucket - balance) was available for taking the weights of one day old kids.

3.7. METEOROLOGICAL OBSERVATIONS:

These observations included measurement of rainfall and relative humidity. This was started on 6th August 1980. Rainfall was measured using a standard Marquis raingauge (Commonwealth Moulding Pty. Ltd.) and was done everyday at 9.00 a.m. The total rainfall in a month was worked out in mm by addition of daily rainfall amounts for all the days of a month.

The relative humidity (R.H.) was measured using a wet and dry bulb thermometer and was also measured on a daily basis at regular times of the day. In this case the readings were taken at 1.00 P.M. The mean relative humidity in a month was worked out by adding up the mean relative humidity values in a month and dividing the total by the number of days in the month.

The raingauge was positioned away from the buildings or trees which could otherwise interfere with daily rainfall readings.

3.8. FLY TRAPPING (GLOSSINA SPECIES):

The flies were caught by use of Challier traps. According to FAO'S tsetse and trypanosomiasis information quarterly (1978) these traps have two advantages over the Moloo and Langridge box Screen (LBS), they are less bulky and when compared to the rest of the animal model traps, they are two or three times as efficient for catching of G. morsitans and G. pallidipes. They are also good for the catching of palpalis group and G. longipennis (FAO, 1978 - Ed. Pollock).

The trap consists of two fabric cones, approximately equal in size, joined base to base and supported by a vertical pole passing through the middle. The bottom cone of white or blue cotton fabric is divided into four segments by

dark cloth partitions visible through the slits. The top cone of mosquito netting contains on top a wire frame supporting a collecting cage. Flies attracted to the trap enter through the dark slits at the base and move up to the collecting cage.

The whole cage was fixed firmly in the ground by the vertical metal pole within about 3 meters of the preferred Glossina habitat which is normally thick clumps of bushes and shrubs (Snow-personnal communication). The pole was smeared well with grease, 4 - 5 cm from the ground to prevent any termites, from climbing up to the cage and eat the trapped Glossina.

These traps were put up four days consecutively every month. Every day for those four days, the trapped flies were collected, identified into sex and species. At the end of four days, the total number of flies was calculated. This number was then divided by the number of traps multiplied by the number of days (4 days) to work out the number of flies caught per trap per day (24 hours), i.e.

$$\text{flies/trap/day} = \frac{\text{Total number of flies}}{\text{Number of traps} \times 4}.$$

Species identification was done as described in EATTRO'S "Notes on field studies of tsetse flies in East Africa" and by Snow (personnal communication).

3.9. FERTILITY RATES:

At approximately one year of age, the nannies were put together with the billy goats so that mating could be effected. The mating period was one and a half months to two months. After this period the billy goats were separated from nannies. The mating nannies were selected according to the management requirements and so not all nannies involved in the experiment were mated.

Any abortions that occured were noted with special reference as to whether the animal was in the control or experimental group. At kidding, the type of birth (single, twin or triplet) was noted and the body weight of the kids taken using a bucket-balance.

3.10. NECROPSY PROCEDURE:

All animals involved in the project were post-mortemed as soon as possible after death. In cases where the animals were found to be continously wasting, having pale mucous membranes and a progressively low packed cell volume, they were killed and post-mortemed. Necropsy procedures were carried out as described by Jones and Gleiser (1954). Samples were taken whenever there was need to confirm the causeof death. The samples included, kidney, lymph nodes, lungs, liver and brain. These were preserved in 10% formalin and

and later sectioned and routinely stained with Hematoxylin and Eosin (H&E). The histologic and staining procedures followed were according to the Mannual of Histologic and Staining Methods (3rd edition) of the Armed Forces Institute of Pathology (1968).

The working formulae for:

A. Instantaneous Point Prevalence and Incidence Rate (I.R).

This was done by the method described by Le-Riche (1971).

Thus

(i) Point Prevalence or (I.P.R.) = $\frac{\text{No. of cases of a disease existing in a population at a point in time}}{\text{No. of animals in that population at that same point in time}}$

(ii) Incidence Rate

$$\text{I.R.} = \frac{\text{No. of new cases of a disease which occur in a population during a stated period of time}}{\text{Average no. of animals in that population during the same period of time}} \times \text{Length of time period}$$

The incidence rate is expressed on an yearly basis, thus the length of time period is a fraction of an year (8/12).

B. CALCULATING FORMULA FOR VARIANCE AND HENCE STANDARD DEVIATION FOR THE PACKED CELL VOLUME (P.C.V.).

With real data, the mean is unlikely to be a whole number and hence taking each observation as a deviation from the mean and squaring the deviation $(X-\bar{X})^2$ becomes tedious. With the help of some algebra a "Calculating formula" is reached at.

$V = \frac{\Sigma x^2}{n}$ where x^2 = deviation from the Mean

n = total number of measurements

V = Variance

and $\Sigma x^2 = \Sigma(X-\bar{X})^2$ where X = observation

$\bar{X}$ = Mean of observations

When the above is expanded it becomes

$$= \Sigma(X^2 - 2X\bar{X} + \bar{X}^2)$$

In the above case $\bar{X}$ is a constant (k), then

$\Sigma k = nk$ and $\Sigma kX = k\Sigma X$.

Thus $\Sigma(X^2-2X\bar{X} + \bar{X}^2) = \Sigma X^2 - 2\bar{X}\Sigma X + n\bar{X}^2$

$$= \Sigma X^2 - \frac{2\Sigma X}{n}\Sigma X + \frac{n\Sigma X}{n}\frac{\Sigma X}{n}$$

as $\bar{X} = \frac{\Sigma X}{n}$

Cancelling the ns and subtracting gives

$$\Sigma x^2 = \Sigma X^2 - \frac{(\Sigma X)^2}{n} \quad \text{and} \quad V = \frac{\Sigma x^2}{n}$$

The Standard Deviation (σ) or S.D. $= \sqrt{V}$

C. TESTING THE DIFFERENCE IN P.C.V. BETWEEN THE EXPERIMENTAL AND THE CONTROL GROUPS:

For each sampling the t-test was used to find out whether the observed differencesin P.C.V. means were statistically significant.

The formula used to get the value of t was calculated as shown below (Kempthorne, 1969)

$$t_{n_1+n_2-2} = \frac{\bar{X}_T - \bar{X}_c}{\sqrt{\frac{\Sigma x^2 + \Sigma y^2}{n_1 + n_2 - 2}\left(\frac{1}{n_1} + \frac{1}{n_2}\right)}}$$

where n_1 = Total number of observation for the treatment group

n_2 = Total observations for the control group

$\bar{X}_T$ = Mean P.C.V. for treatment group

$\bar{X}c$ = Mean P.C.V. for control group

$\Sigma x^2 = \Sigma X^2 - \frac{(\Sigma X)^2}{n}$ for the treatment group i.e. The sum of individual observations squared minus the square of the pooled observations divided by the total number of observations.

Σy^2 as Σx^2 above for the control group.

(n_1+n_2-2) = Degrees of freedom.

The t values were obtained from the tables of Fisher and Yates (1963).

D. ECONOMIC EFFECTS OF TRYPANOSOMIASIS IN THREE BREEDS OF GOATS AT THE COAST PROVINCE OF KENYA:

I: Parasite determinants

1. Parasite attack rate
2. Mortality
3. Weight loss

II: Host determinants

1. A uniform total population at risk of 200 animals of each breed was used in the economic analysis.
2. Herd structure - 100% breeding females.
3. Mean weight

East African goats - 27 Kg

Galla X East African- 27 Kg

Toggenburg X East African- 30 Kg

III: Official price for:-

1. Milk = KSh. 1.85 per litre (official price for Kenya Cooperative Creameries).
2. Meat = KSh. 5 per Kg live weight.

IV: One Tropical Livestock Unit (TLSU) = 250Kg.

4. RESULTS:

Animal numbers.

The animals decreased gradually in number as the experiment continued. The main cause of this decrease in number is death from diseases. The animal numbers that managed to survive through to the end of the experiment in the various respective groups and breeds is shown in Table 1.

TABLE 1: Number of goats surviving by March 1981.

EXPERIMENTAL GROUP		CONTROL	
Number that survived	Breed	Number that survived	Breed
$47/_{50}$	East African	$91/_{108}$	East African
$20/_{20}$	Galla X East African	$10/_{19}$	Galla X East African
$18/_{19}$	Toggenburg X East African	$9/_{19}$	Toggenburg X East African

The numbers show that, the animals in the control group suffered the heaviest mortality. The mortality rates in various breeds and groups and the proportion dying out of trypanosomiasis is shown in Table II.

TABLE II: MORTALITY RATES AMONG THE VARIOUS BREEDS OF GOATS USED AND THE RELATIVE PROPORTIONS DYING OUT OF TRYPANOSOMIASIS:

BREED	GROUP	MORTALITY RATES (%)	PERCENTAGE DYING FROM TRYPANOSOMIASIS
1. East African	Control	16%	10.2%
	Experimental*	5%	0%
2. Galla X East African	Control	47%	36.8%
	Experimental*	0%	0%
3. Toggenburg X East African	Control	53%	42.0%
	Experimental*	6%	0%

* Samorin treated animals.

The mortality rates were highest in the control groups. The proportion of animals dying out of trypanosomiasis was from the control group of goats that were not getting any prophylactic treatment while none of the experimental animals died out of trypanosomiasis.

The breed of animals that had the highest mortality rate was the Toggenburg X East African goats followed by the Galla X East African goats. The lowest mortality was suffered by the small East African goats.

Trypanosomiasis as a cause of death rated highest among the Toggenburg X E. African goats while the East African goats suffered a 10.2% mortality from the disease. The Galla X East African goats suffered an intermediate degree of mortality 36.8% falling in between the mortality rates of the other breeds.

Packed Cell Volume (P.C.V.)

The mean P.C.V's of both the experimental and the control groups of Toggenburg X East African goats, and the standard deviations of the means are graphically presented in figure 3 and 4 respectively.

The experimental group in this breed showed a relatively stable P.C.V. which ranged from 24% to a maximum of 29%. The lowest P.C.V. was

recorded towards the end of October when it fell to 24% but from the beginning of November, the P.C.V. rose to 28% and from this time on to the end of the experiment, the P.C.V. stabilised such that the fluctuation was between 27% and 29%. The P.C.V's of the control group fluctuated greately compared to the experimental group. The highest P.C.V. recorded in this group was 24% in the month of September. There was a marked progressive decrease in P.C.V. The lowest P.C.V. was recorded towards the end of January when it was 16%, after this value was recorded the P.C.V. increased to 18% by the end of February. By the time the experiment ended there was a downward trend in P.C.V. among the control group and an upward trend among the experimental group.

After plotting the standard deviation of the means in Fig. 4 the results indicate that, in the first four months of the experiment, the standard deviations of the means were such that there was an interaction of the standard deviations of the experimental and control groups. At the fifth month the difference is clear but again it cannot be picked out for the next month. From the beginning of the sixth month, there was no interaction

of the standard deviations of the two means, this trend continued to the end of the experiment. The spread of the P.C.V's from the mean was greater in the control group than in the experimental group.

Fig. 5 and 6 represent the mean P.C.V. values and the standard deviations respectively of the Galla X East African experimental and control groups. The mean P.C.V.s of the experimental group started at 27% and maintained at this value to the end of September. In a months time (end of September to the end of October) the mean P.C.V. dropped from 27% to 23%, a value far much lower than that of the control group. The next sampling indicated a rise in P.C.V. values from 23% to 28% which henceforth stabilised at this value such that fluctuations were never greater than one percent to the end of the experiment. This phenomenon was observed with the Toggenburg X East African goats in the experimental group. Fluctuations in the mean P.C.V. values in the control group (as with the Toggenburg X East African control group) were greater than those of the experimental group. At the beginning of the experiment when the P.C.V. values were measured, the controls in this breed had a mean P.C.V. of 25%.

A value higher than this was recorded two months later and was 26%. Such a value was no longer recorded to the end of the experiment and a progressive lowering of the P.C.V. was noted towards the end of the experiment. The lowest mean P.C.V. was recorded towards the end of January at 18%. This is the same time when the lowest mean P.C.V. in the Toggenburg X East African controls was recorded at 16%.

The spread in mean P.C.V. as shown by plotting of the standard deviations only show a statistical significance in the month of January and at the beginning of March. During the rest of the time, the spread is so great that no difference can be identified. However, the standard deviations of the control group are larger than those of the experimental group thus indicating the spread in mean P.C.V. At the end of the experiment, the mean P.C.V. of the control appeared to have stabilised at a value of 21%, a value higher than that of the Toggenburg X East African controls whose last value was 17%.

Among the East African goats, the mean P.C.V. values and the standard deviation of the mean are presented in figure 7 and 8 respectively. In both the experimental and control groups, the lowest P.C.V. values were recorded in the early

part of October. The lowest value recorded among the experimental group was 27% while that recorded in the control group was 22%. In both groups the P.C.V. values stabilised by the fourth month of the experiment. The experimental goats showed a fluctuation from 27 to 31%. The highest values were recorded in the months of November and December. In the control group, the P.C.V. fluctuated from 22 to 25%. After the fourth month, the mean P.C.V. was maintained at constant level of 24% until the final sample was taken in March which showed a slight drop of 1%. Analysis of the mean P.C.V. by standard deviation indicate that no statistical significance is identifiable except on one single instance. Here again the spread in the control group is greater than in the experimental group.

TESTING THE DIFFERENCE BETWEEN THE MEAN P.C.V. OF THE EXPERIMENTAL AND CONTROL GROUPS BY MEANS OF A t-TEST.

The results of this test are represented in Table IIIa for the Toggenburg X East African goats. The difference in mean P.C.V. observed between the experimental andthe control group was statistically significant at 1 percent level after the second month of treatment. However, by the end of the third month this level of significance was not maintained but statistical significance was there at 5% level. From the third month henceforth, the differences between the two means was statistically significant throughout at 99% confidence limits. After the fifth month of samorin administration to the experimental group, the level of significance rose to 0.001, this level was maintained through to the end of the experiment.

TableIIIb represents the results of the t-test for the Galla X East African goats. Over the first four months of samorin administration, the mean P.C.V. values obtained for the control and experimental groups were not significantly different statistically at 5 percent level. After the fourth month, the difference observed was statistically significant at 5 per cent level. After 5 months of samorin administration the values maintained

a statistically significant difference at 5% level to the end of the experiment. Only in two instances were the differences statistically significant at 0.1% level. This was in the month of January.

The t-test analysis for the East African goats as presented in Table IIIcshows a high level of statistical significance of the differences observed in mean P.C.V. The differences are significant at 99.9% confidence limits throughout the experiment except on one instance early in the experiment when the difference was statistically significant at 99% confidence limits.

TABLE III: t - test for the differences in mean P.C.V. between experimental and control groups.

BREED: (a) TOGGENBURG X EAST AFRICAN

GROUP MEANS($\bar{X}$)		Degree of	Value of	Probability
EXPERIMENTAL	CONTROL	freedom(df)	t	(P).
26.1	24.1	30	0.98	<0.400
27.6	21.7	26	2.830	<0.010
25.2	17.8	25	3.964	<0.001
22.9	18.2	26	2.505	<0.050
28.5	18.1	18	5.724	<0.001
29.2	21.9	20	3.674	<0.010
26.9	18.8	15	2.989	<0.010
27.5	19.6	21	5.143	<0.001
26.8	16.3	22	5.747	<0.001
28.3	16.6	19	6.652	<0.001
29.0	18.3	19	5.225	<0.001
29.2	16.7	20	6.197	<0.001

TABLE III (b)

BREED: GALLA X EAST AFRICAN

GROUP MEANS ($\bar{X}$)		Degree of	Value of	Probability
EXPERIMENTAL	CONTROL	freedom (df)	t	(P)
27.3	25.8	36	0.704	<0.500
27.2	23.6	17	1.592	<0.200
25	22.9	24	0.985	<0.400
23.1	25.8	27	-	-*
28.4	21.3	15	2.875	<0.050
29	24.4	24	2.309	<0.050
27.9	21	17	1.451	<0.200
28.3	21.2	25	3.979	<0.001
28.6	20.5	25	5.179	<0.001
28.4	17.7	21	3.789	<0.010
29.1	21.2	20	2.407	<0.050
29.5	21	20	3.771	<0.010

* - Not tested as the mean value of the control group was higher than that of the experimental group.

TABLE III (c)

BREED: EAST AFRICAN

GROUP MEANS ($\bar{X}$)		Degree of	Value of	Probability
EXPERIMENTAL	CONTROL	freedom (df)	t	(P)
27.9	22.7	98	4.924	<0.001
26.9	22.0	62	3.117	<0.010
28.9	23.1	107	5.477	<0.001
31.0	25.4	110	5.752	<0.001
30.7	24.5	108	5.546	<0.001
29.4	24.1	105	11.269	<0.001
29.6	24.1	105	5.607	<0.001
28.7	23.9	110	5.234	<0.001
30.1	24.0	105	5.782	<0.001
28.4	22.5	97	5.082	<0.001

TRYPANOSOME DETECTION AND IDENTIFICATION:

The trypanosome species identified from the ear ve in among the various breeds are presented in tables VI - VIII.

Table VI represents the Toggenburg X East African goats. There were only two species of trypanosomes that were detected and these are T. vivax and T. congolense. T. vivax was diagnosed when the experiment started by use of thick blood smears. It was identified from groups of animals that were grouped for samorin administration. T. congolense was diagnosed in far greater frequency than T. vivax. After samorin administration started, only in one instance was an experimental animal found infected with T. congolense. All the other positive samples were from the control group of goats. In this group of animals the ratio of T. vivax to T. congolense was 1:6.

The table shows that parasitaemia was not detected every time the samples were taken. In 6 out of 14 instances when the samples were obtained no trypanosomes were detected from the samples.

From the Galla X E. African goats, the trypanosome species diagnosed are presented in table VII. In this group of animals, T. congolense was again diagnosed in far greater frequency than T. vivax. In two instances, trypanosomes

were diagnosed from experimental goats. In one instance the infecting trypanosome species was T. congolense while in the second instance the trypanosome involved was T. vivax.

The ratio of T. vivax to T. congolense was 1:4.5. As in the case of Toggenburg X East African goats, parasitaemia was not detected with every sampling and thus in 8 out of 14 instances, no trypanosomes were detected.

With the East African goats, the data on table VIII shows that with commencement of samorin administration, none of the goats in the experimental group was ever positive for trypanosomiasis. The positive one detected by use of thick blood smear was bled on the first day of samorin administration and hence it had not received any samorin prior to the time of positive diagnosis.

Most of the trypanosomes detected were T. congolense and were from the control group. The ratio of T. vivax to T. congolense was 1:6.5. With the East African goats, in 4 out of 14 instances, no positive diagnosis was made.

The overall ratio of T. vivax to T. congolense was 1:6.1.

TABLE IV.

TRYPANOSOME SPECIES IDENTIFIED FROM EARVEIN BLOOD

a) TOGGENBURG X EAST AFRICAN GOATS:

Sampling (Weeks)	Method of Detection	Trypanosome number and species	Animal group
Week 0*	Thick blood smear	2 T. vivax	Experimental
		1 T. congolense	Control
2	Buffy coat	1 T. congolense	Experimental
4	"	- **	-
6	"	1 T. congolense	Control
8	"	1 T. congolense	Control
12	"	3 T. congolense	Control
14	"	-	-
16	"	1 T. congolense	Control
18	"	-	-
20	"	-	-
22	"	1 T. congolense	Control
24	"	1 T. congolense	Control
26	"	-	-
28	"	-	-

* : These samples were taken on the day the experiments began and no animal had received any samorin.

** : No trypanosomes identified from either control or experimental groups.

TABLE V.

b) GALLA X EAST AFRICAN GOATS:

Sampling (Weeks)	Method of Detection	Trypanosome number and species	Animal group
Week 0	Thick blood smear	-	-*
2	Buffy coat	1 T. vivax	Control
4	"	-	-
6	"	-	-
8	"	-	-
12	"	-	-
14	"	1 T. congolense	Experimental
	"	1 T. vivax	Control
16	"	1 T. congolense	Control
18	"	-	-
20	"	-	-
22	"	1 T. congolense	Control
	"	1 T. vivax	Experimental
24	"	2 T. congolense	Control
26	"	1 T. congolense	Control
28	"	-	-

-* : No trypanosomes identified either from control or experimental groups.

TABLE VI.

c) EAST AFRICAN GOATS:

Sampling (Weeks)	Method of detection	Trypanosome number and species	Animal group
Week 0*	Thick blood smear	2 T. congolense 1 T. vivax	Control
		1 T. vivax	Experimental
2	"	1 T. congolense	Control
4	"	-	-**
6	Buffy coat	1 T. congolense	Control
8	"	1 T. congolense	Control
10	"	2 T. congolense	Control
12	"	-	-
16	"	1 T. congolense	Control
18	"	2 T. congolense	Control
20	"	-	-
22	"	1 T. congolense	Control
24	"	1 T. congolense	Control
26	"	1 T. congolense	Control

* : These samples were taken on the day the experiments began and no animals had received any samorin.

** : No trypanosomes identified either from control or experimental groups.

DISEASE INCIDENCE:

The disease incidence among the three breeds of goats used in the experiment worked over a one year period for all trypanosome species is represented by the figures shown below:-

East African goats	12.2%
Galla X East African goats	41.6%
Toggenburg X East African goats	42%

As the figures indicate, the incidence is highest among the Toggenburg X East African goats followed by the Galla X East African goats and lowest among the East African goats.

DRUG ADMINISTRATION:

When samorin was administered intramuscularly at the side of the neck, only one case of reaction to the drug was noticed in a Galla X East African goat. The goat reacted by being drowsy and unstable in gait. However, this reaction lasted only for 10 - 15 minutes. Local swellings were recorded in a few of the animals that were under samorin (R) regime, these swellings were small, never more than 3 cm. in diameter and would only have been well appreciated on palpatation. The swellings never developed into wounds or abscesses.

GROWTH RATE:

The body weight changes are shown graphically in figures 15, 16 and 17 for the Toggenburg X East African, Galla X East African and the East African goats respectively. The Toggenburg X East African goats in the experimental group started with a lower body weight than the controls of the same breed. The first month of the experiment showed a weight gain in both groups. This trend continued for the goats in the experimental group but the control group showed a sharp decrease in weight. The highest mean weight among the experimental group recorded was 33.5 Kg in the month of January compared to the mean weight of 24.8 Kg at the beginning of the study. From January to April, the weight decreases recorded were 3.9 Kg for the experimental group and 2.0 Kg for the control group, respectively.

The Galla X East African goats had starting weights of 27.2 and 24 Kg for the experimental and control groups respectively. The body weights for the two groups showed little fluctuation. The experimental group showed a maximum weight of 30.2 Kg in September and 30.1 in January and decreased to 29.4 in April. The control group

showed a gradual weight gain from 24 Kg at the beginning of the study to 26.4 Kg in the month of April.

The East African goats showed mean weights of 27.6 and 24.6 Kg for the experimental and control groups respectively at the start. The experimental group showed a gradual weight gain recording a peak of 33.3 Kgs in the month of January but the weights decreased to 28.9 Kgs by April. The control group also showed a progressive weight gain and by January a maximum mean of 28.6 Kgs was attained and like the experimental group the mean weight decreased to 25.4 Kgs by April. Within the first six months of the study the gain in weight among the experimental goats was 5.7 Kgs and 4 Kgs for the control group.

FERTILITY:

The results on fertility of the two breeds of goats which were put into mating are shown below:

East African Goats:

(i) Percentage of goats that kidded:

	Experimental	Control
Number mated	40	71
Number that kidded	25	30
Percentage that kidded (%)	62.5	42.9
(ii) Percentage that twinned		
Number that kidded	25	30
Number that twinned	5	4
Percentage that twinned (%)	20	13.3
(iii) Mean weight of twins (Kg)		
Total weight of twins	14.5	7.0
Total number of kids	10	8
Mean weight per kid	1.5	0.9
(iv) Mean weight of singles (Kg)		
Total weight of singles	25.4	33.5
Total number of kids	20	26
Mean weight per kid	1.27	1.25

Galla X East African Goats:

(i) Percentage of goats that kidded

	Experimental	Control
Number mated	8	10
Number that kidded	4	2
Percentage that kidded (%)	50	20

(ii) Mean weight of kids (Kg)

Total weight of kids	8	2.5
Total number of kids	4	2
Mean weight per kid	2	1.3

The groups of goats that were receiving a monthly dose of samorin showed higher fertility rates. The percentage of goats that kidded among the East African experimental group was 62.5% compared to 42.9% among the control group. The experimental group of Galla X East African showed a value of 50% as giving birth to kids while the controls of the same breed showed a value of 20%. Among the Galla X East African goats, no animal gave birth to twins while among the experimental East African goats, 20% of the animals that kidded gave birth to twins. The controls showed a lower twinning ability since only 13.3% gave birth to twins. The mean weight per kid born as twins among the East African experimental group was 1.5 Kg compared to 0.9 Kg, being the weight per kid born twin among the control group.

The mean weights for kids born as singles had only a slight difference of 0.02 Kg and therefore, this difference may not be significant. The Galla X East African goats showed differences in the mean weights of kids born of nannies from different groups. Thus the kids born of experimental nannies had a mean weight of 2.0 Kg while those born of control goats had a mean weight of 1.3 Kgs.

Among the East African goats abortion occured in six of the goats. All these goats belonged to the control group. The cause of abortions could not be established due to unfavourable circumstances.

METEOROLOGICAL OBSERVATIONS:

The total monthly rainfall and relative humidity are presented in figures 12 and 13 respectively.

In the month of August when the experiment started, the total rainfall recorded was 42.5 mm. The two subsequent months were drier as the total rainfall recorded for each month was less than 24 mm. The fourth month (November) was marked by a heavy rainfall reaching a total of 192.6 mm. The months of December and January had total rainfall amounts of 42.7 and 22.0 mm respectively. The month of February was the driest as no rain fell at all. The last month (March) was marked by heavy rains totalling up to 188.0 mm.

The highest relative humidity was 83 per cent recorded in the month of August. It then decreased in the two subsequent months to 69 per cent in the month of October. In the following month (November) which was the wettest of all the months during the experimental phase, the relative humidity increased to 75%. Since this month, the relative humidity values decreased progressively and during February which was the driest month, the relative humidity was 66 per cent. In March, the relative humidity increased to 74 per cent, a value still lower than the value recorded for August.

INSTANTANEOUS POINT PREVALENCE RATES (I.P.R.):

Figure 9 represents the I.P.R. for the Toggenburg X East African goats. At the beginning of the experiment, the I.P.R. was 5.3% and then dropped to 0% by late August and early September. The rates then gradually increased to reach a maximum of 25% by late October; this value was not recorded again. Between late January and early February, the infection rate was maintained between 12.5 and 14.4%. During the last two samplings no infections were detected. The Galla X East African goats (fig. 10) did not show any infections during the first sampling but by the end of August an infection rate of 6.6% was recorded. This group of goats did not show any trypanosomes again in the peripheral circulation until the month of November when an infection rate of 9.1% was recorded for two consecutive samplings. The maximum infection rate was recorded at 28.6% during early February, then dropped to 16.7% by late February and by early March, the infection rate was 0%.

The East African goats (fig 11) showed an infection rate of 0% only in three instances, that is early September, November and late January. The infection rates fluctuated between 1% and 3.5%. This breed of goats showed three peaks (above 3%) during late October, early January and March. Among Galla X East African goats and the Toggenburg X East African goats, whenever infections were detected

FLY TRAPPING:

The flies caught over the experimental period were very fluctuent in number (fig. 14). In the month of September only 7 flies were caught per trap per 24 hours. The number increased to 18 and then dropped to 5 in November. From November to February, the flies caught ranged between 4 and 6. The number increased again to 17 in the month of March.

In the whole experimental period, the only species of flies caught was Glossina pallidipes. The female numbers were higher than the males. The overall ratio of males to females was 1:2.

CLINICAL SIGNS:

Animals with trypanosome infection showed chronic emaciation, progressive weight loss and anaemia which was clinically manifested by pale mucous membranes. The superficial lymph nodes were visibly swollen and this was especially so with the prescapular and submandibular lymph nodes. The animals dying of trypanosmiasis showed a progressive decrease in P.C.V. and anorexia was only noted terminally when the animals got too weak. In one case of a Toggenburg X East African goat, dyspnoea due to intercurrent pneumonia was noted with accompanying central nervous signs. These included unstable gait and torticollis. It was possible that pneumonia was a sequel of trypanosomiasis as the animal had been diagnosed positive for trypanosomiasis six months earlier than pneumonic signs were noted, but there was no proof.

POST-MORTEM FINDINGS:

On carrying out post-mortem on infected animals the main outstanding lesions were gelatinous fat atrophy at the auriculo-ventricular farrow and the perirenal area as well as mesenchymal fat tissue. Some animals showed the presence of straw coloured fluid in the pleural and peritoneal cavities. Haemorrhages

were noted in one case of the goat (above) which showed pneumonic signs. The haemorrhages were on the subpleural surface and were petechial in nature. The lymph nodes were oedematous. Splenomegally was not noted. Most of the animals dying out of trypanosomiasis showed a relatively high worm burden in the abomasum. The infecting helminth parasite was Haemonchus contortus.

HISTOPATHOLOGY:

Some of these results are presented in figures 18 to 23.

The histopathologic lesions manifested by the kidney consisted of degeneration of tubular epithelial tissue, mononuclear cell infiltration into the interstitium of the kidney and the glomeruli. Some glomeruli were swollen. The mononuclear cells infiltrating the kidney parenchyma and other tissues were mainly plasma cells and lymphocytes. In some cases, a low degree of haemosiderosis was present.

In the liver the main histologic features were centri-lobular degeneration and mononuclear cell infiltration.

The lymph nodes showed extensive hemosiderosis while in one case, mononuclear cell infiltration into the muscular tissue was noticed.

The lungs showed infiltration by the mononuclear cells, emphysema, atelectasis,

congestion of the blood vessels and hemosiderosis. The case of the goat with intercurrent pneumonia also showed haemorrhages and fibrin exudation into the lung tissue.

In the brain, the only lesion that was diagnosed was mononuclear cell infiltration.

ECONOMIC EFFECTS OF TRYPANOSOMIASIS IN THREE GOAT BREEDS WORKED OUT ON AN ANNUAL BASIS:

	East African	Galla X East African	Toggenburg X East African
No of animals at risk	200	200	200
Herd structure	100%	breeding	females
% Refractory to infection	87.8	58.4	58
A: Mortality % due to			
Trypanosomiasis	10.2	36.8	42
Numbers	20	74	84
Mean weight (Kg)	27	27	30
Total loss (Kg)	540	1998	2520
Loss in KSh.	2700	9990	12600
B: Loss of weight			
Numbers	180	126	116
Estimated Annual Loss	3.4	2.8	21.4
Total loss (Kg)	612	352.8	2482.4
Loss in KSh.	3060	2822	12412
C: Milk loss			
Mortality numbers	20	74	84
Lactation (Litres)	60	90	120
Milk loss/Goat (Aver. 2 lactations)Annually	120	180	240
Total milk loss (Litres)	2400	13320	20160
Loss in KSh	4440	24642	37296
TOTAL LOSS KSh.	10200	37454	62308
STOCK UNITS	21.6	21.6	24.0
LOSS/TLSU	472	1734	2596

COST OF CONTROLLING TRYPANOSOMIASIS PER TROPICAL LIVESTOCK UNIT USING ISOMETAMEDIUM CHLORIDE AT THE DOSAGE RATE OF 0.5 Mg/KG BODY WEIGHT MONTHLY FOR ONE YEAR.

GOAT BREED	COST OF DRUG	EQUIPMENT	LABOUR AND TRANSPORT	MUSTERING	COST/ TLSU
East African	34.60	1.20	4.00	15.	54.8
Galla X East African	34.60	1.20	4.00	15.	54.8
Toggenburg X East African	34.60	1.20	4.00	15.	54.8

COST/BENEFIT RATIO USING ISOMETAMEDIUM CHLORIDE

BREED OF GOAT	COST/TLSU	LOSS FROM TRYPS/TLSU	COST/BENEFIT RATIO
East African	54.8	472	1:8.6
Galla X East African	54.8	1,734	1:31.6
Toggenburg X East African	54.8	2,596	1:47.4

NB: Costs are in KSh.

5. DISCUSSION:

One of the aims of this study was to determine the effect of Isometamedium chloride (Samorin) on the production parameters in three breeds of goats.

This study has showed that goats kept under samorin regime can thrive better in trypanosomiasis endemic areas than unprotected goats. The goats kept under samorin prophylactic regime had higher body weights, higher P.C.V. values and none of them died out of trypanosomiasis.

These results are in agreement with previous studies carried out by various workers. Griffin and Allonby (1979c) showed that the mean increase in weight of goats protected against trypanosomiasis with samorin was 9.1 Kg, the goats used in this work were Galla and Saanen X Galla crosses and ranged in age between 9 and 12 months. These were young growing animals and with Isometamedium chloride they may have shown a higher weight gain than the goats used in the present study which had ages ranging from 1 year to 5 years and hence could not have shown dramatic weight gains.

During the first six months of the experiment, the Toggenburg X East African goats getting samorin showed a weight gain of 8.7 Kg while the control goats of the same breed showed

a mean weight loss of 2 Kg. The body weights by the month of April had decreased by 3.9 and 2.0 Kg for the treatment and control groups respectively. This period before March was a dry period and since the goats were not being feed supplimented there was undernourishment and increased stress.

The weight gain among the Galla X East African experimental goats between August and January was 2.9 Kg while the controls showed a continous weight gain from the start to the end, the gain in weight between August and January was 1.5 Kg. The experimental East African goats showed a weight gain of 5.7 Kg during the initial six months while the controls gained 4 Kgs over the same period. By April, both groups had lower mean body weight than the month of January. This decrease can be attributed to kidding andthe stress therein.

Samorin per se in absence of trypanosomiasis does not affect the growth rate or milk production (Lindau and Spielberger, 1973). Therefore the increase in weight and the high P.C.V. values observed in the treatment group of animals as compared to the controls was due to the effect of trypanosomiasis. Chemoprophylaxis as a means of protecting animals against trypanosomiasis in endemic areas has been recognised for a long period of time as the drugs used render premunition to

the animals (Stephen, 1966; Fiennes, 1970; Finelle, 1974; Griffin and Allonby, 1979c). Fiennes (1970) while reviewing the work done in cattle using curative and subtherapeutic antrycide doses concluded that the effectiveness of the drug depended mainly on its ability to suppress infection to a point where the animal's own defense mechanisms were able to gain control.

Subsequently, the control goats that were not protected against trypanosomiasis in this study showed mortality rates ranging from 10.2% for the East African goats to 42% for the Togenburg X East African goats resulting from trypanosomiasis. The clinical signs manifested by the infected goats included chronic emaciation, progressive weight loss, pale mucous membranes and a terminal anorexia. These findings are in agreement with those of earlier workers (Fiennes, 1954; Clarkson, 1968; Stephen 1970; Losos, et al., 1973; Omuse, 1973; Anosa and Isoun, 1976; Kaaya, 1975; Kaaya, et al., 1977). Superficial lymphadenomegally was also noted among the control goats, an observation that has also previously been made by Kaaya (1975) while working with an experimental infection of T. congolense in goats and van den Ingh, et al.(1976)while working with T. vivax infection in goats.

One case was encountered in which pneumonia with central nervous signs occured concurrently with trypanosomiasis, the goat affected was a Toggenburg X East African. Pneumonia occuring concurrently with trypanosomiasis has been reported by Kramer (1966)and Kaaya, (1975). In his (Kramer) survey work, farmers gave a history to the effect that animals diagnosed positive for trypanosomiasis also frequently showed pneumonic signs. Since there is no unequivocal prove that the occurence of pneumonia is related to the trypanosome infection and also in view of the fact that only one animal in the whole control group in this study showed pneumonia and trypanosomiasis concurrently, then it may be concluded that the two occur indipendently until further prove.

The trypanosome species found from infected animals were T. congolense and T. vivax. These trypanosome species are commonest and most important as far as livestock production in Eastern Africa is concerned (Whiteside, 1958; Omuse, 1973; Kaaya, 1975 and Griffin, 1978). The frequency with which T. vivax was diagnosed was lower than that of T. congolense. In all the three breeds of goats, the overall ratio of T. vivax to T. congolense was 1:6.1. These findings correlate with the figures quoted by

Whiteside (1958) on the disease incidence due to T. vivax and T. congolense in Kenya. Thus between 1946 and 1956, the disease due to T. vivax had an incidence rate of 7.0% while that due to T. congolense had an incidence rate of 90.0%. In the year 1956 to 1957, the disease incidence due to T. vivax was 16% while that due to T. congolense had an incidence of 79%. Over the two periods, T. brucei showed an incidence of 1.3 and 1.5% respectively, a very low incidence.

The post mortem findings which included gelatinous fat atrophy at the auriculo-ventricular farrow and perirenal areas, presence of straw coloured fluid in the pleural and peritoneal cavities and oedematous lymph nodes agree with the findings of other workers (Fiennes, 1953; Losos et al. 1973; Kaliner, 1974; Kaaya, 1975; van den Ingh, et al. 1976). The haemorrhages observed at the subpleural surface are also in agreement with the findings of other workers, van den Ingh, et al.(1976).Fiennes (1953) reported occurence of haemorrhages of the medulla of the lymphatic glands in cattle protected against trypanosomiasis either by antrycide or dimidium. He also reported sub-epicardial and sub-endocardial haemorrhages in unprotected control cattle.

In this study, splenomegally was not recorded. Many workers studying experimental trypanosomiasis have reported this condition. Fiennes (1953) did not observe enlarged spleens in cattle exposed to trypanosomiasis without any drug protection. His findings are in agreement with the fact that in this study too no splenomegally was observed.

The histopathological lesions are in agreement with other workers who have shown that, in the kidney, there is tubular epithelial degeneration with mononuclear cell infiltration consisting of lymphocytes, macrophages and plasma cells (Omuse, 1973; Kaliner 1974; Kaaya, 1975). In addition, Kaaya (1975) reported protein casts in the tubules while Fiennes (1953) reported the occurence of T. congolense in the myocardium (acting as a tissue parasite). In all the tissue sections examined, no trypanosomes were found in tissues. Further observations include the presence of hemosiderin in the kidneys, lungs and lymph nodes. The liver showed centrilobular degeneration with slight mononuclear cell infiltration. The lungs were atelectatic and emphysematous with mononuclear cell infiltration. These observations have been made earlier by Kaaya (1975), van den Ingh et al (1976).

Mononuclear cell infiltration into the brain has been reported in trypanosome infections by the above mentioned authors and was also observed in this study.

On three instances, trypanosomes were detected from the blood of animals in the treatment groups. Three factors may have contributed to this positive diagnosis. In one instance involving a Galla X East African goat, it was found that the goat had not received samorin injections for the previous two months. Also during the month prior to this, the fly numbers also registered a peak. The other two goats involved were a Toggenburg X East African and a Galla X East African. In both cases the goats were found to have heavy pregnancy or recently kidded. Stress is documented as a cause of breakdown in premunity resulting from chemotherapy (Stewart, 1951; Stephen, 1966; Mortelmans and Kageruka, 1976) and this may again explain the occurence of trypanosomes in the peripheral circulation of an animal under chemotherapy in this study.

EAST AFRICAN GOATS AND TRYPANOTOLERANCE:

Trypanotolerance among the East African goats as compared to the other two breeds used in this study is evident when the various parameters are taken into consideration. The mortality rates resulting from trypanosomiasis were lowest among the East African goats and highest among the Toggenburg X East African goats. The Galla X East African goats had a mortality rate intermediate between the other two groups. The respective mortality rates were 10.2%, 42% and 36.8%.

The disease incidence worked out over a one year period shows the following values 12.2%, 41.6%, 42% for the East African, Galla X East African and the Toggenburg X East African goats respectively. This trend is similar to that of mortality rates with the Galla X East African goats having an intermediate value between the values of the other two goat breeds.

The instantaneous point prevalence rates (I.P.R) show relatively low values for the East African goats while the other two breeds of goat show relatively high values. The highest value recorded among the East African goats was never above 4% while infection rates as high as 28.6% were recorded among the Galla X East African goats.

The highest value recorded among the Toggenburg X East African goats was 25%. On average, the infection rates increased in the order of East African goats, Galla X East African and Toggenburg X East African goat. Work by Griffin and Allonby (1979b) showed a similar pattern of trypanotolerance among the Saanen X Galla, Saanen, Galla and East African goats. Their work was carried out by experimental infection of sheep and goats with 1 ml blood containing 10^5 trypanosomes (T. congolense). They were able to monitor parasitaemia throughout the period of the experiment from the infected goats. In this study, challenge was natural and therefore the level of parasitaemia was bound to vary.

With respect to the P.C.V. values, the results show that there was more or less steady values for the East African goats. The lowest mean P.C.V. value recorded among this breed was 22% while the highest mean value recorded was 25.4%. These ranges fall within the normal haematocrit values for goats as given by Schalm (1965), the normal range being 19.5 to 38.5 with average of 28.5%. Therefore, even in the presence of trypanosomes, the East African goats are able to minimise the pathologic effects of trypanosomes such as hemolysis. In comparison, the Galla X East African control goat showed markedly fluctuent P.C.V's ranging from 17.7 to 25.8%, a range of

8.1%. This group of goats however showed a progressive decrease in P.C.V. from the start of the study, a phenomenon which is not apparent among the East African goats. Generally, the Galla X East African goats maintained a P.C.V. value above 20% and only once was the mean P.C.V. recorded below this level (17.7%).

The Toggenburg X East African showed great fluctuations in their P.C.V. mean values. The highest mean P.C.V. among the control group recorded was 24.1% and the lowest recorded was 16.3%, a range of 7.8%. Only on three instances were the mean P.C.V. above the 20% value while on all other occasions, the P.C.V's were below 20%. Here again the trypanotolerant quality of the East African goats is reflected followed by the Galla X East African goats while the Toggenburg X East African goats reflect how poorly they can withstand trypanosomiasis in absence of chemoprophylaxis. The latter group of goats also show a progressively decreasing mean P.C.V. from the start to the end of the study period. The above findings are in agreement with those of Griffin and Allonby (1979b),who also showed an intermediate degree of trypanotolerance in goat crosses between the indigenous and the exotic breeds. In the work of Griffin and Allonby (1979b),the Saanen X Galla crossbreed was intermediate in trypanotolerance between the Saanen (a newly introduced breed into

Africa - Griffin and Allonby 1979b) and the Galla goat. In this study, the Galla X East African goat was intermediate between a pure indigenous East African goat and the Toggenburg X East African goat. The Toggenburg like the Saanen is a new breed of goat that has recently been introduced in Africa unlike the Galla which has been in Africa longer. The work in this study confirms the work of Chandler (1952) who demonstrated that the trypanotolerance of the N'Dama and Zebu cross-breeds was intermediate between that of the pure breeds and suggested a genetic basis for trypanotolerance, the view which is also upheld by Murray and Morrison (1978), who state that trypanotolerance is a characteristic which has come into being by natural selection and is unrelated to the blood type. Griffin and Allonby (1979b) were not able to show any relationship between the haemoglobin type and trypanotolerance among various breeds of sheep used.

Over the period of this study the East African and the Galla X East African goats in the control groups showed increases in weight while the Toggenburg X East African goats in the control groups showed loss in weight. These results agree with those obtained by Griffin and Allonby (1979b). These workers showed a decrease in weight

among the exotic Saanen goats. The East African goats showed little change in weight but gained a weight of 2 Kg. The body weight changes of the Saanen X Galla goats was intermediate between those of Saanen and East African goats.

The results on fertility indicate that the animals under samorin regime had better fertility rates than those groups of goats not getting samorin. The percentage of the goats that kidded and twinned were higher for the experimental goats than for the controls.

The mean weights of kids born as twins was 50% higher for the kids born of experimental animals than kids born of control animals. Among the Galla X East African goats, no twins were recorded probably in view of the few numbers that were put into mating. However, the mean weight of the kids born as singles by experimental nannies were 54% higher than those of kids born of control nannies. Among East African goats, no differences were seen between singles born of experimental and control nannies with regard to their birth weight.

The above findings may be taken to mean that, with the trypanotolerant breeds, pregnancy of single kids has little stress effects but marked ones among less trypanotolerant goat breeds. This stress effect is then reflected in the birth

weight of kids. However, where twin pregnancy occurs among trypanotolerant breeds, the stress effects are reflected more in goats which are not under samorin prophylaxis and hence the birth weights of the resultant kids is less compared to twin kids born of samorin protected nannies.

The above observations are in agreement with other workers who have noted reduced fertility rates in trypanosomiasis infected animals (Stephen, 1966; Roberts and Gray, 1973; Finelle, 1974).

Abortion occured in six control goats. Although it cannot be definately stated that abortions were caused by trypanosomiasis in this study, the finding of T. vivax in the amniotic fluid of pregnant ewes (Isoun and Anosa, 1974) may have a bearing to the above findings. Abortion due to T. evansi was also recorded by Paikne and Dhake (1972) in a she buffalo.

Although the number of Galla X East African goats put into mating was rather low compared to that of the East African goats, the results obtained show that the Galla X East African goats that were in the control groups had lower percentage of nannies kidding (20%) compared to the same group of East African goats where 42.9% kidded.

Mortelmans and Kageruka (1976) while attempting to upgrade the Dahomey cattle by crossing them with trypanotolerant N'Dama cattle in Zaire, they noted improved fertility rates among the less trypanotolerant breeds.

The economic losses due to animal diseases have been classified into direct or immidiate losses and indirect losses, (FAO, 1962; Griffin and Allonby, 1979c). The direct effects are further subdivisible into visible losses which include mortality, abortions, condemnation at slaughter and damaged hides. All the above are directly measurable while the invisible losses include quantities of meat and milk that were not produced, reduced length of working life and infertility. Finelle (1974) estimated the value of additional meat that could be produced if trypanosomiasis were controlled while Griffin and Allonby (1979c) considered one aspect of "visible" losses (mortality) and another aspect of "invisible" losses (quantities of meat that were not produced). Their work reviews that considering only the two aspects, the net gain with samorin given on a four monthly basis is KSh. 264 per Tropical livestock unit (TLSU), assuming a body weight of 30 Kg per goat.

In this study the returns realised in using samorin on a monthly basis have been worked out as a cost/benefit ratio.

The cost/benefit ratio is highest with the least trypanotolerant breed of goat, the Toggenburg X East African. With these goats, the cost/benefit ratio is 1:47.4 while with the East African goats, the ratio is 1:8.6 and the Galla X East African goats have an intermediate value of 1:31.6. In view of the fact that such losses as may result from abortion, condemnation at meat inspection, infertility and reduced length of life were not considered during this study, the above ratios may have been higher. The values however indicate that losses due to trypanosomiasis are considerably higher than what previous studies have indicated.

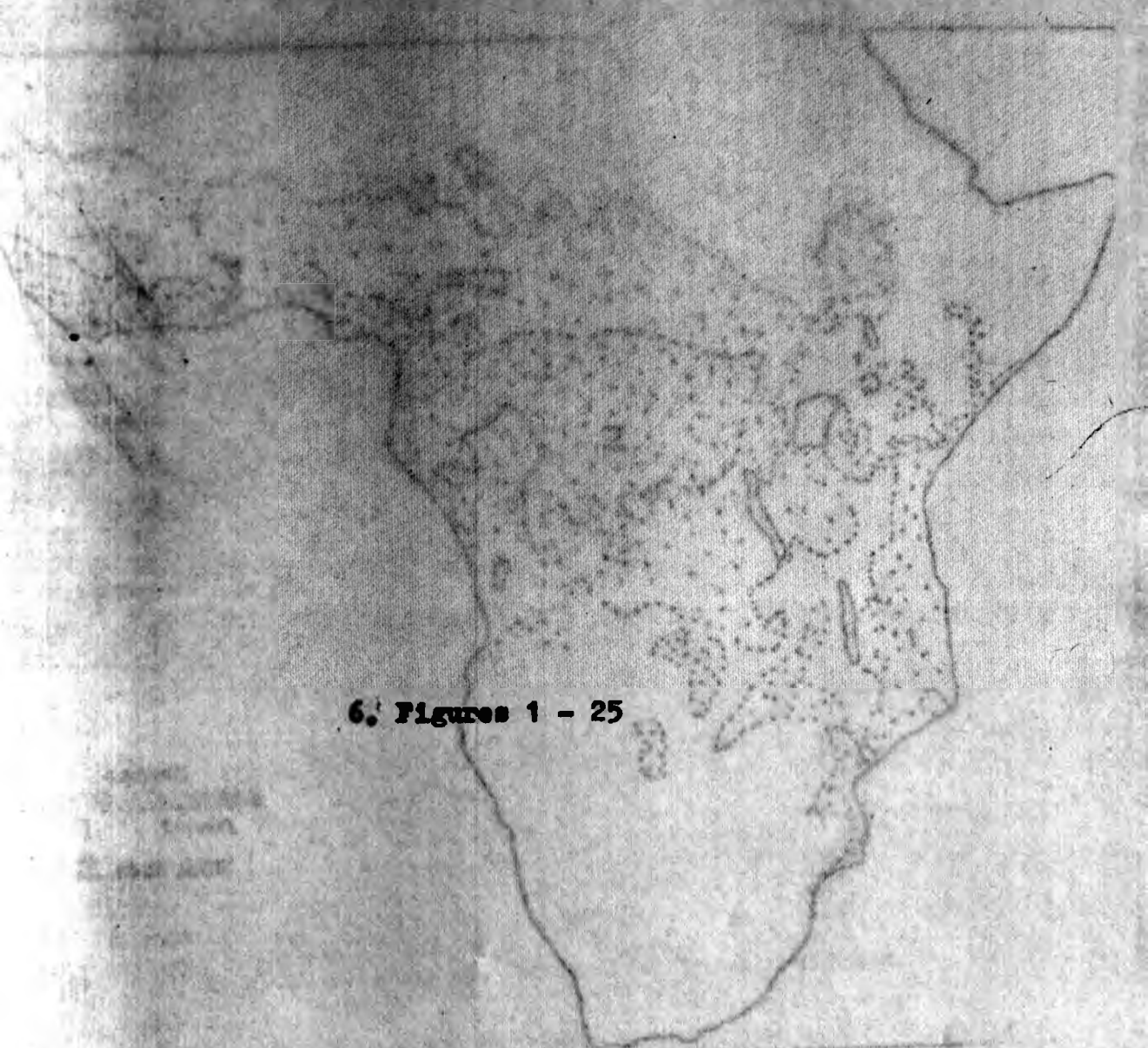
During the period of this study, rainfall and tsetse fly numbers registered two peaks almost at the same period of time. However, the first peak of fly number registered in October while the rainfall recorded its peak in the month of November. The lowest fly numbers were recorded in the months of January and February which were also the driest of the months. During the month of March, high peaks of rainfall and tsetse fly numbers were also recorded. The fly numbers and rainfall amounts tend to follow a similar pattern. Griffin and Allonby (1979d) found a highly significant degree of correlation between tsetse fly numbers and rainfall at Kiboko area in Machakos District over

a year when rainfall was very heavy but no correlation could be detected when the year was dry with intermittent rainfall. During the year with heavy rainfall, they noted a lag phase of one month between the falling of heavy rains and the appearance of tsetse flies in large numbers. They attributed the lag period to the length of time taken by the insect to pupate. In this study, no such marked increase in fly numbers was noted following heavy rains although in December, a slight peak was noted following the heavy rains in November. The relative humidity was never below 65% during the study period which would suit G. pallidipes (the only species caught) and this observation agrees with other reports (EATTRRO'S "Notes for field studies of tsetse flies in East Africa" 1955; FAO's "Training Manual for tsetse control personnel 1978"). With regard to the infection rates in the goats, during the month of October when the tsetse fly numbers recorded a peak, two breeds of goats also had peak infection rates. These goat breeds were the Toggenburg X East African and the East African goats. The Galla X East African goats on the other hand showed no infections at all. This latter breed of goats showed high infection rates during the month of January and February when fly numbers were relatively low.

Work done by Griffin and Allonby (1979d) in Kiboko range research station showed that concomitant with marked increase in fly numbers, the Karakul breed of sheep also showed a marked rise in infection while the Saanen X Galla goats showed a steady increase in infection rates which were not significantly affected by the changes in tsetse numbers. During that same year of study when the fly numbers were high, the purebred Galla goats showed low infection rates and only a moderate increase when the fly numbers also increased.

Over the second drier year of study when fly numbers were low a similar pattern emerged but the Galla and the East African goats remained free of infections. In this study, the infection pattern of the Galla X East African goat seems indipendent of the tsetse fly numbers. The Toggenburg X East African goats show marked increase in infection rates with the increase in fly numbers while in the East African goats, the increase is very small. This study has not been able to compare data over two years with varying rainfall amounts and possibly tsetse fly numbers, however it allows for the conclusion that infection rates in the Galla X East African goats are indipendent of tsetse fly numbers as shown in fig. 14b while among the East African and the Toggenburg X East African goats infection rates

are related to tsetse fly numbers. However, the increase in infection rates among the East African goats due to increase in tsetse fly numbers is relatively little compared to that of the exotic Toggenburg X East African goats.



6. Figures 1 - 25

Fig. 1. The distribution of Glossina spp. in Africa.
(Reproduced from FAO's Report of the second consultation of the programme for the control of African animal trypanosomiasis.)

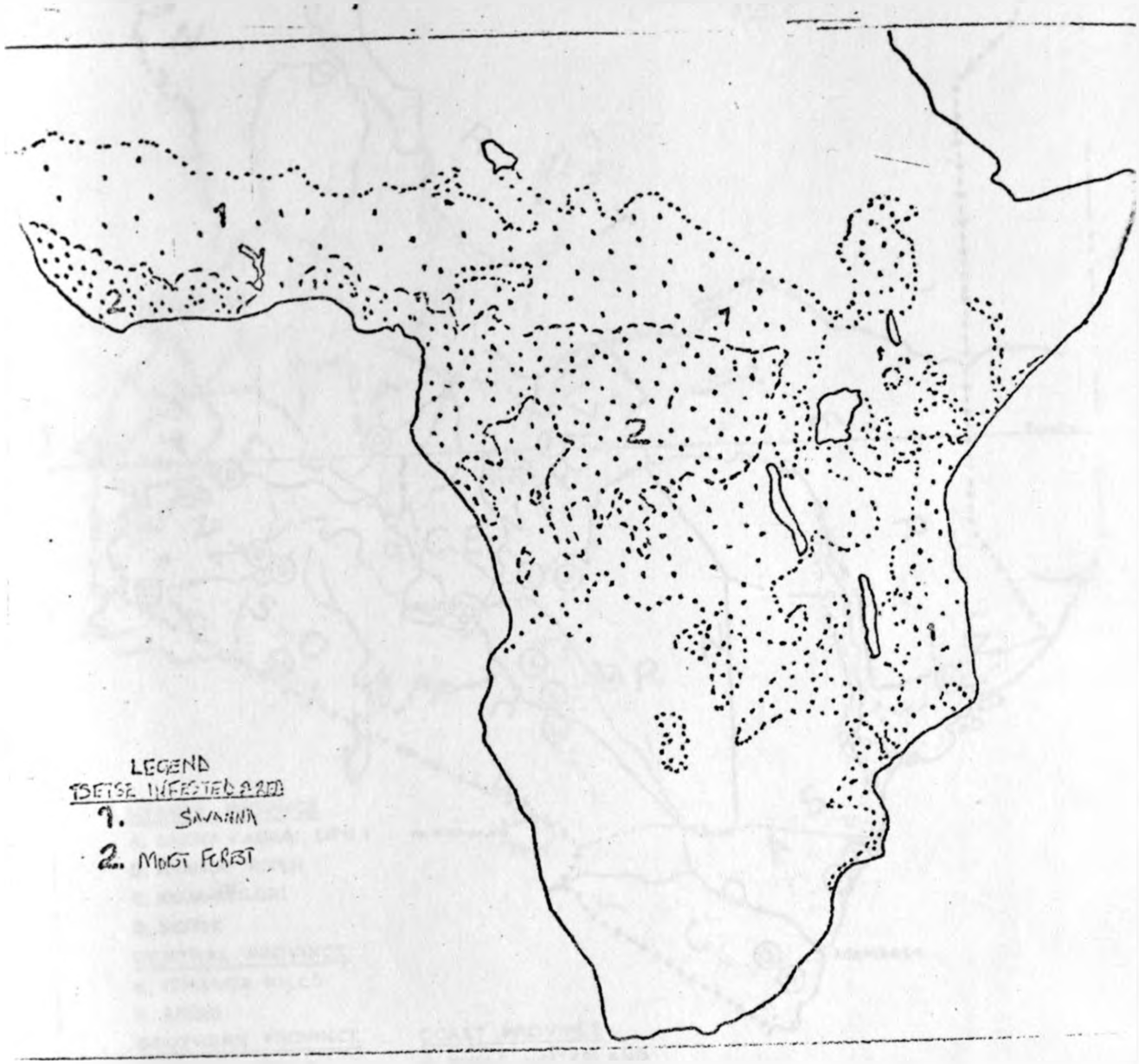
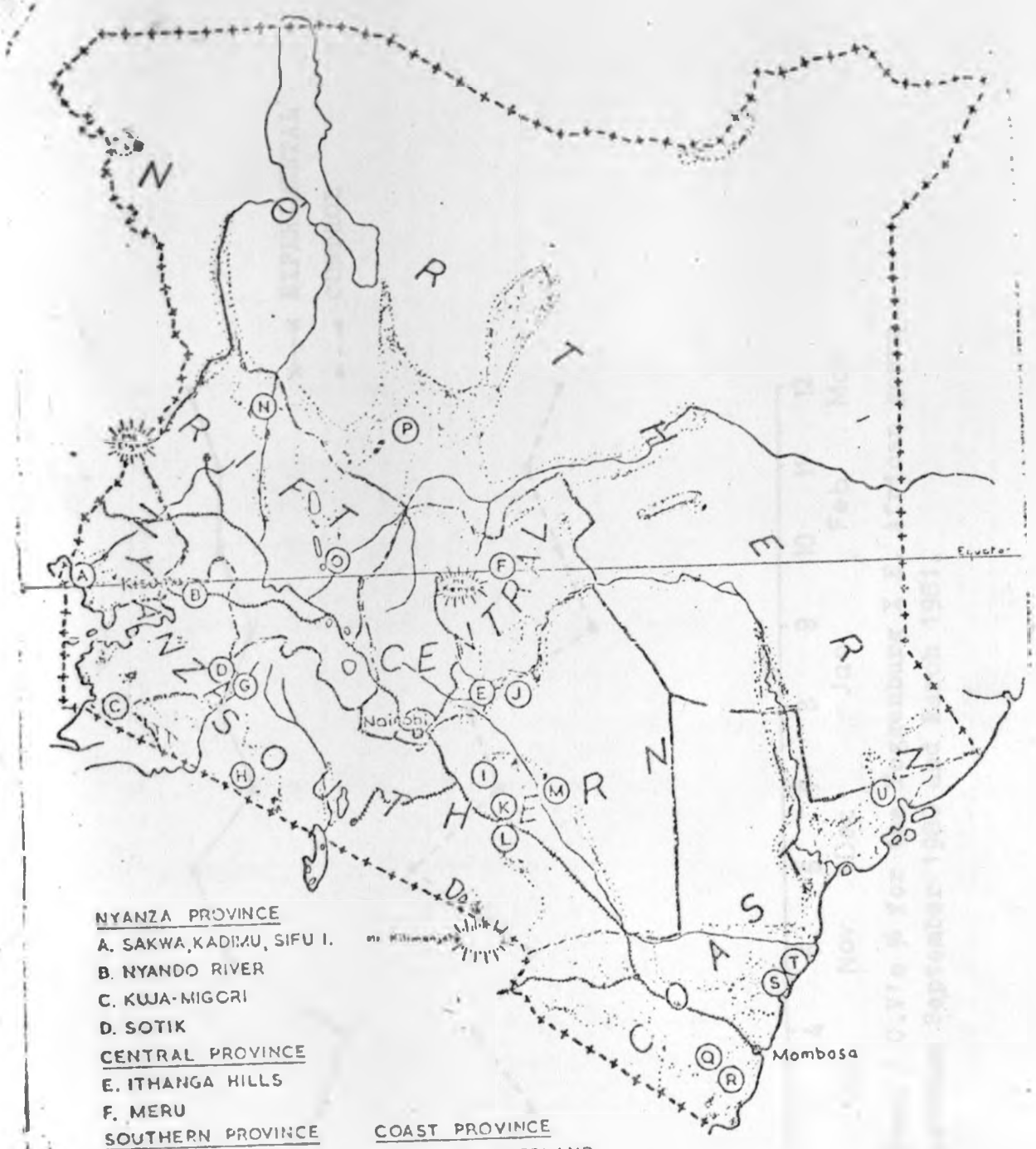


FIG. 1: The distribution of Glossina spp. in Africa. (Reproduced from FAO's Report of the second consultation of the programme for the control of African animal trypanosomiasis.)



NYANZA PROVINCE
A. SAKWA, KADIMU, SIFU I.
B. NYANDO RIVER
C. KUJA-MIGORI
D. SOTIK

CENTRAL PROVINCE
E. ITHANGA HILLS
F. MERU

SOUTHERN PROVINCE
G. MASAI-KIPSIGIS
H. TALEK
I. MAKUENI
J. YATTA B I
K. SIMBA
L. KIBOKO
M. ATHI-TIVA

RIFT VALLEY PROVINCE
N. WEST SUK
O. SOLAI-SUBUKIA

NORTHERN PROVINCE
P. MARALAL

COAST PROVINCE
Q. KWALE HINTERLAND
R. SHIMBA HILLS
S. KILIFI
T. GEDI
U. LAMU

KNOWN TSETSE FLY INFESTATION.....

...of Kenya showing areas infested by tsetse flies

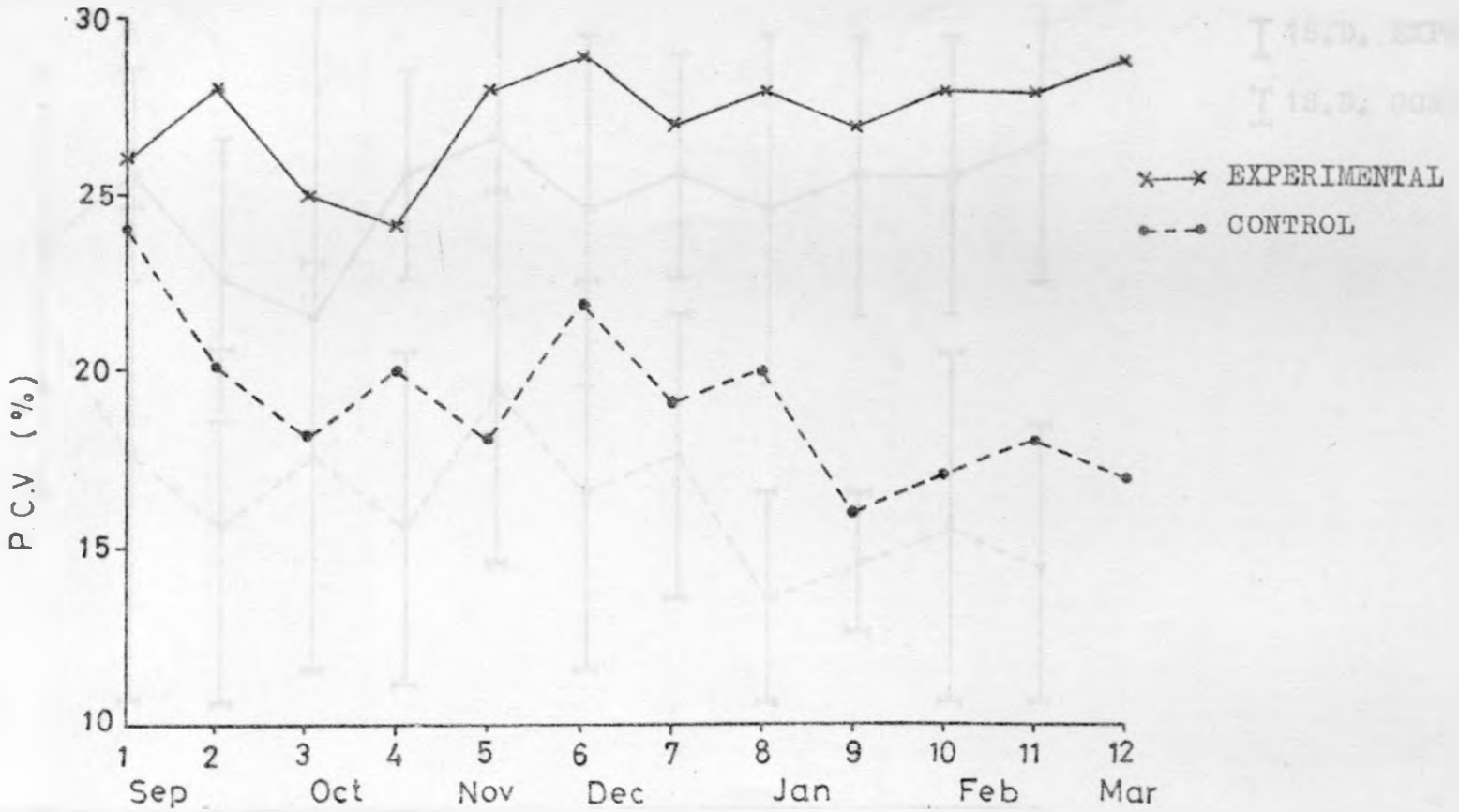


FIG. 3: Mean P.C.V's % for the Toggenburg X E. African goats between September 1980 and March 1981.

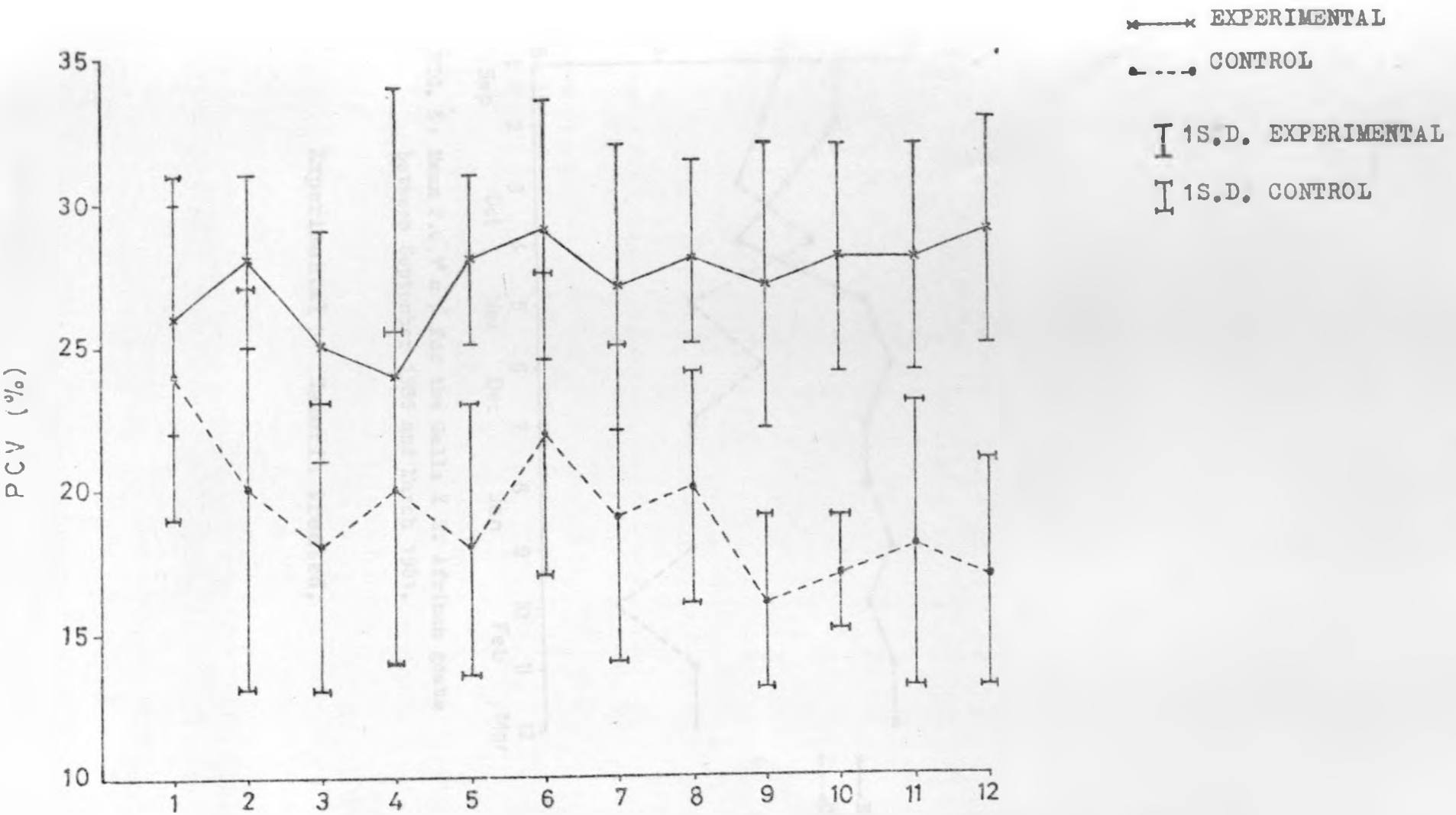


FIG. 4 : Standard deviations of the means P.C.V's at every sampling for the Toggenburg X East African goats, (S.D.= Standard deviation).

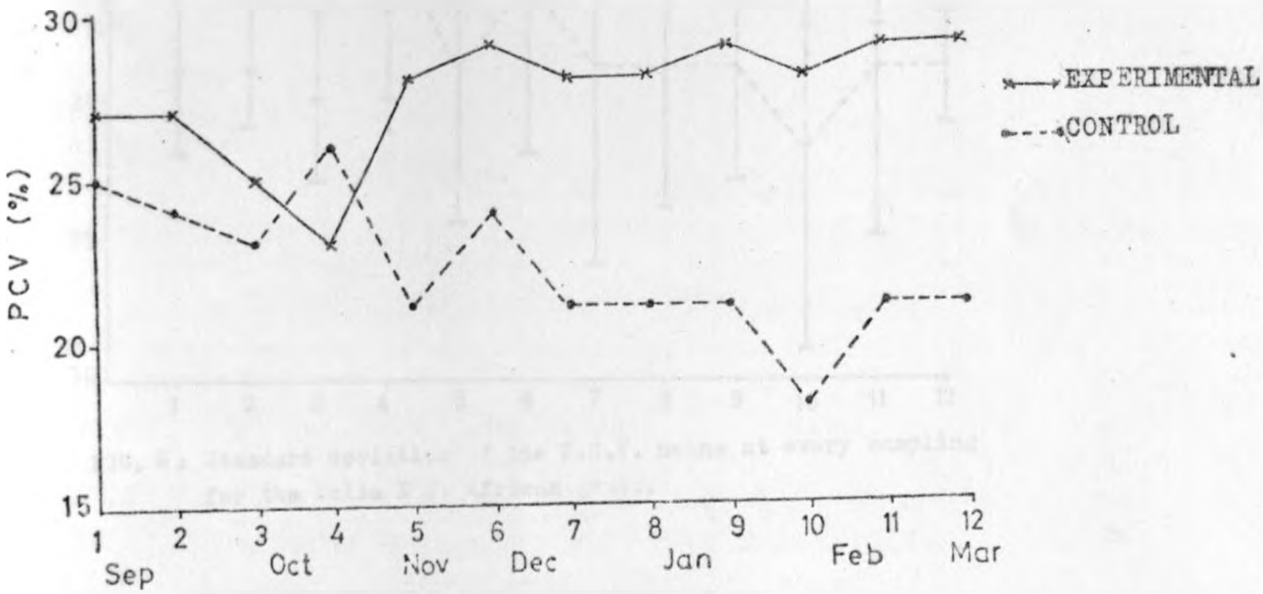


FIG. 5: Mean P.C.V's % for the Galla X E. African goats between September 1980 and March 1981.

Experimental :- Samorin treated.

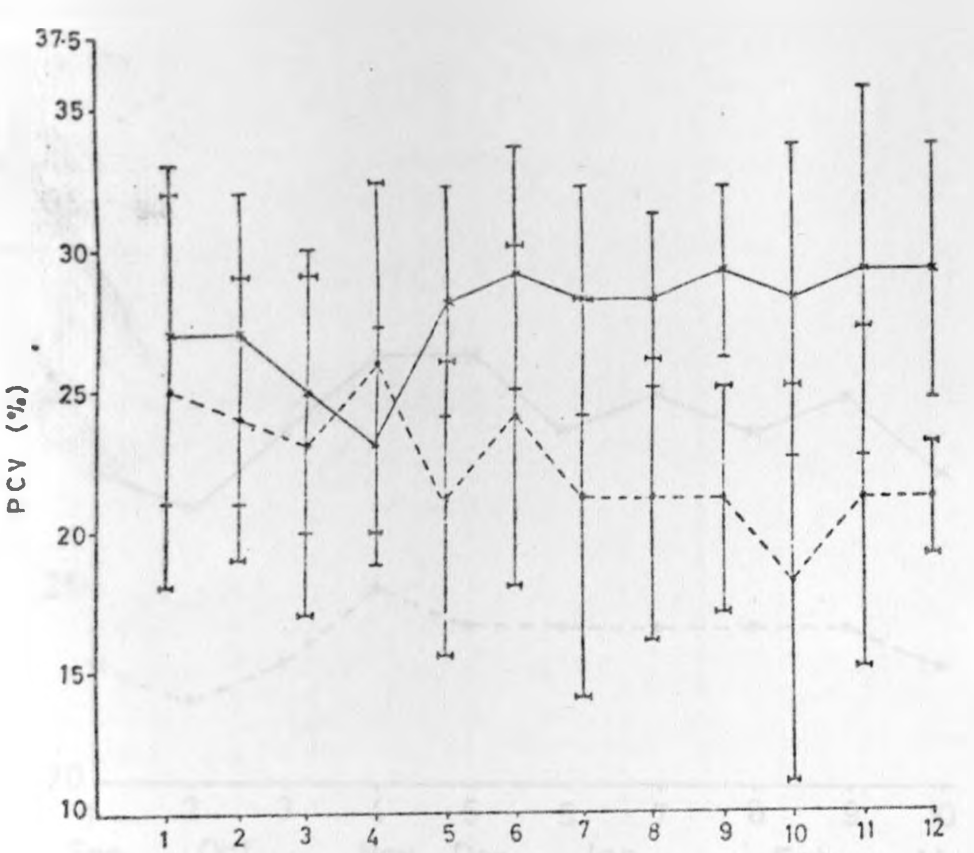


FIG. 6 : Standard deviation of the P.C.V. means at every sampling for the Galla X E. African goats.

Experimental :- Samorin treated.

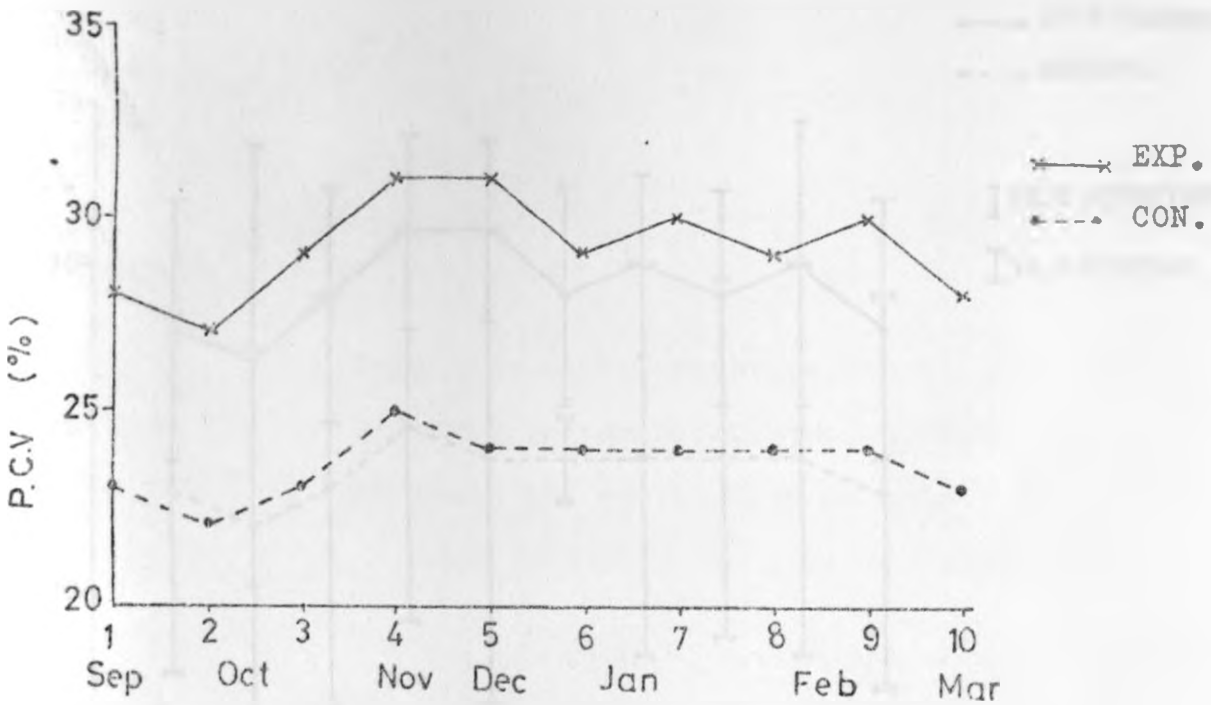


FIG. 7: Mean P.C.V.s % for the East African goats between September 1980 and March 1981. (EXP. = EXPERIMENTAL; CON. = CONTROL).

Experimental :- Samorin treated.

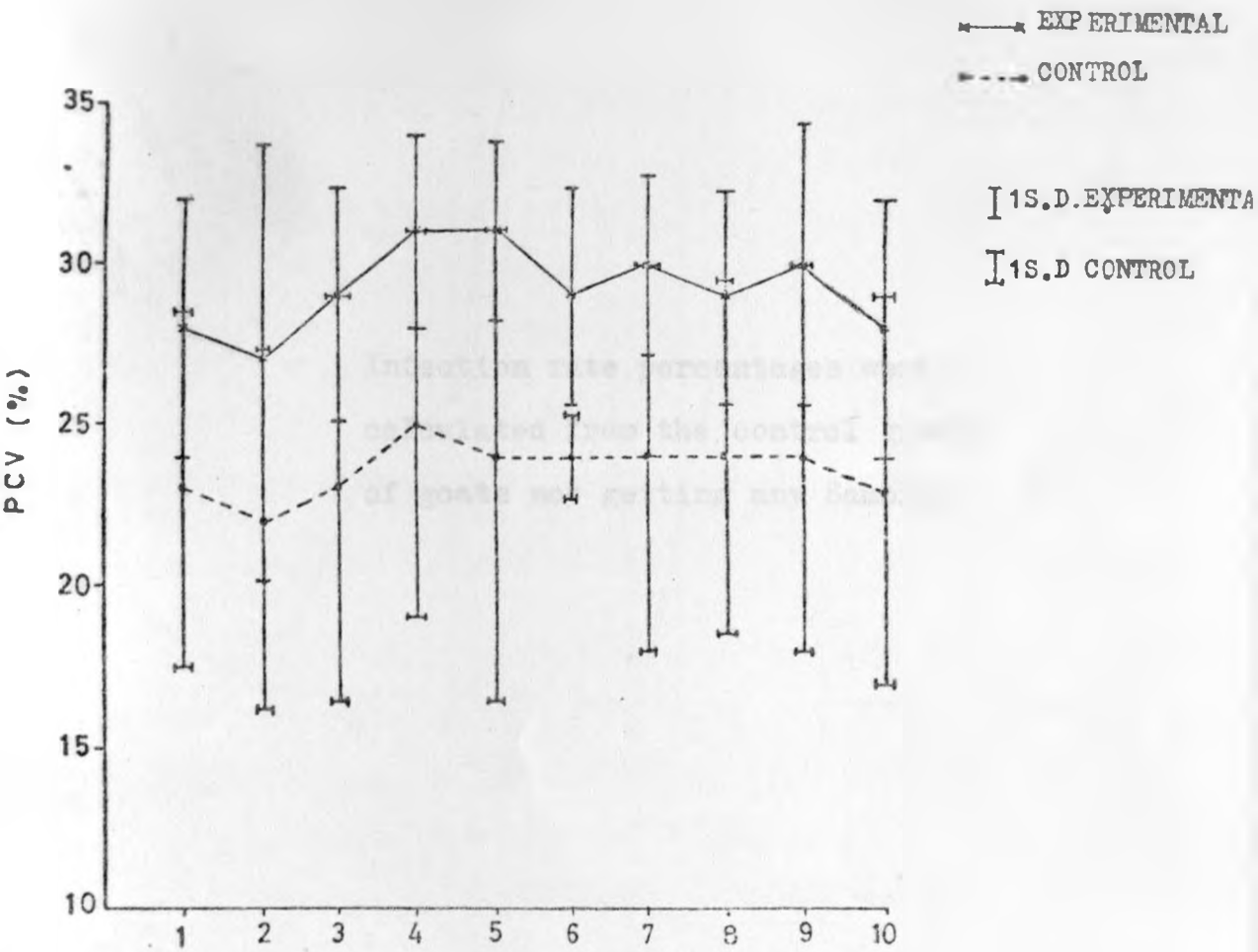


FIG. 8: Standard deviations of the mean P.C.V's at every sampling for the E. African goats.

Experimental - Samorin treated.

Infection rate percentages were calculated from the control groups of goats not getting any Samorin.

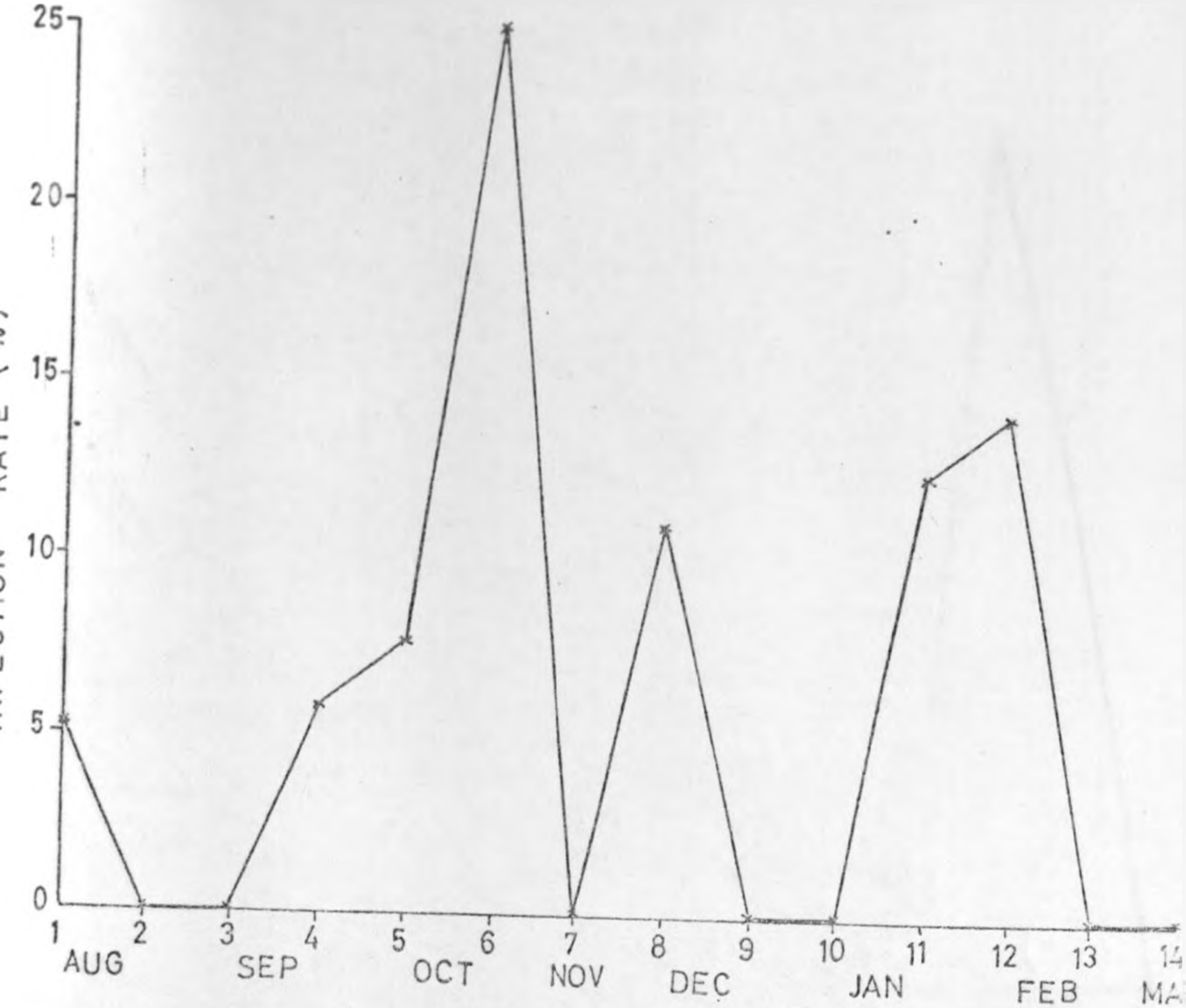


FIG: 9: Infection rate % for the Toggenburg X East African goats calculated at every sampling from August 1980 to March 1981.

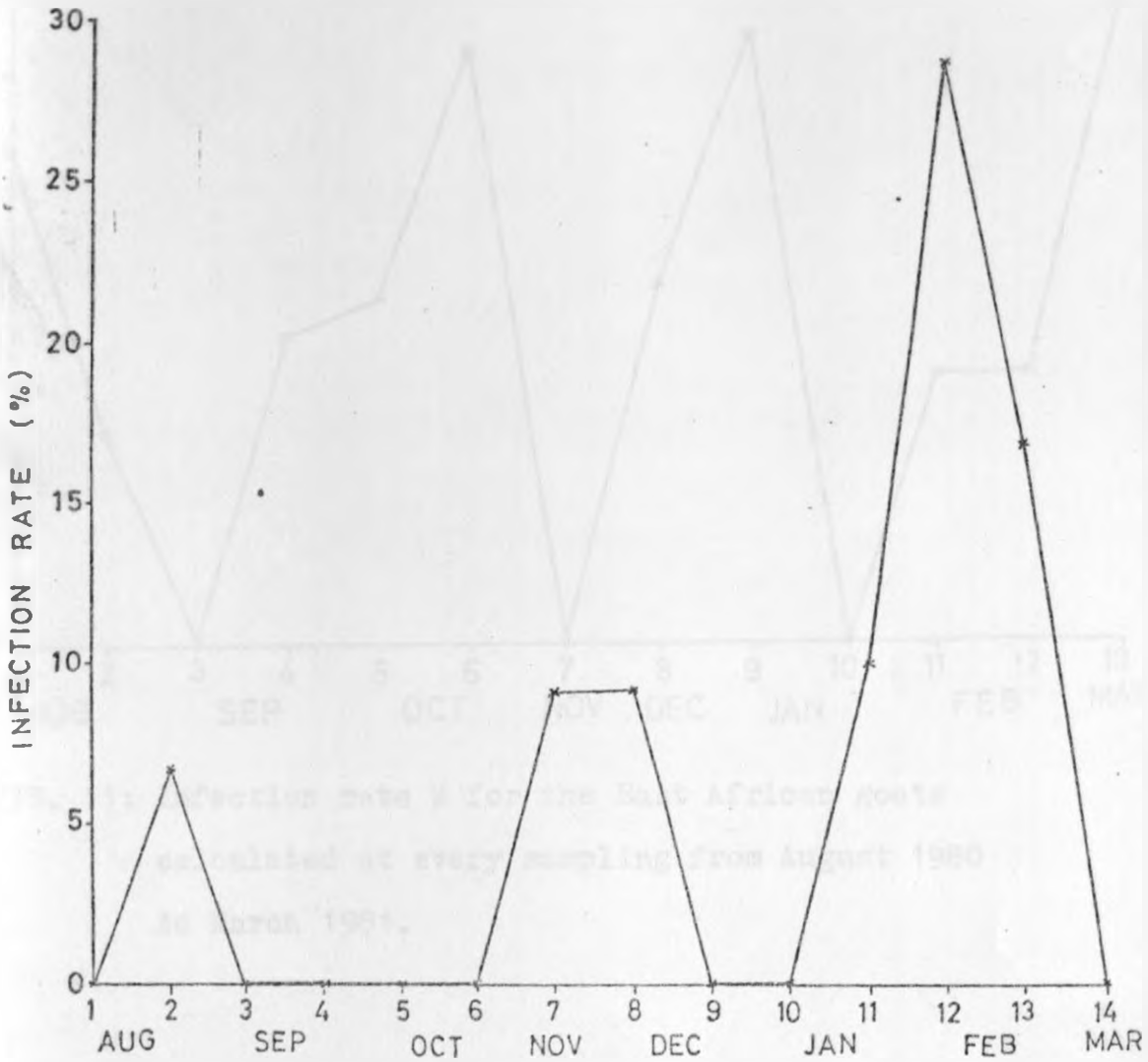


FIG. 10: Infection rate % for the Galla X East African goats calculated at every sampling from August 1980 to March 1981.

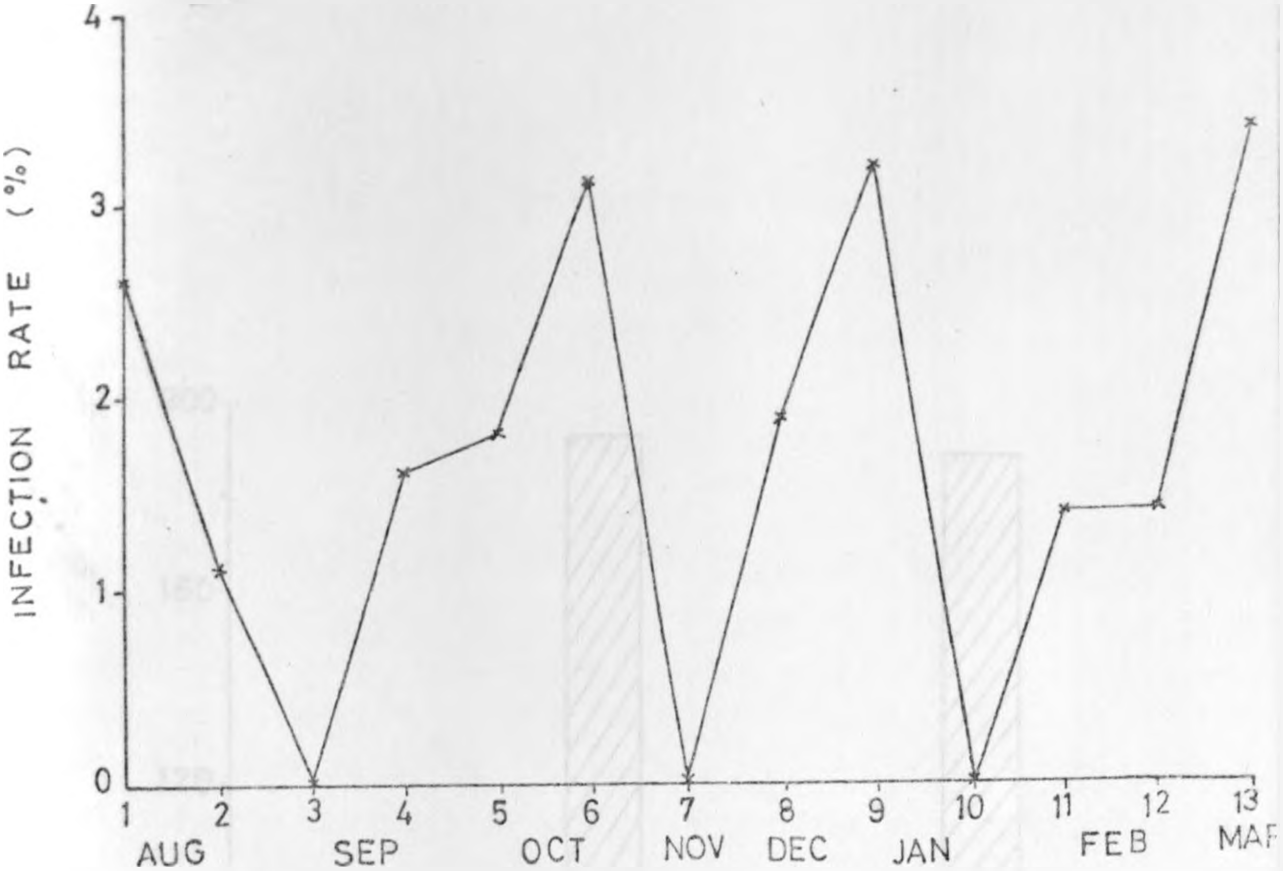


FIG. 11: Infection rate % for the East African goats calculated at every sampling from August 1980 to March 1981.

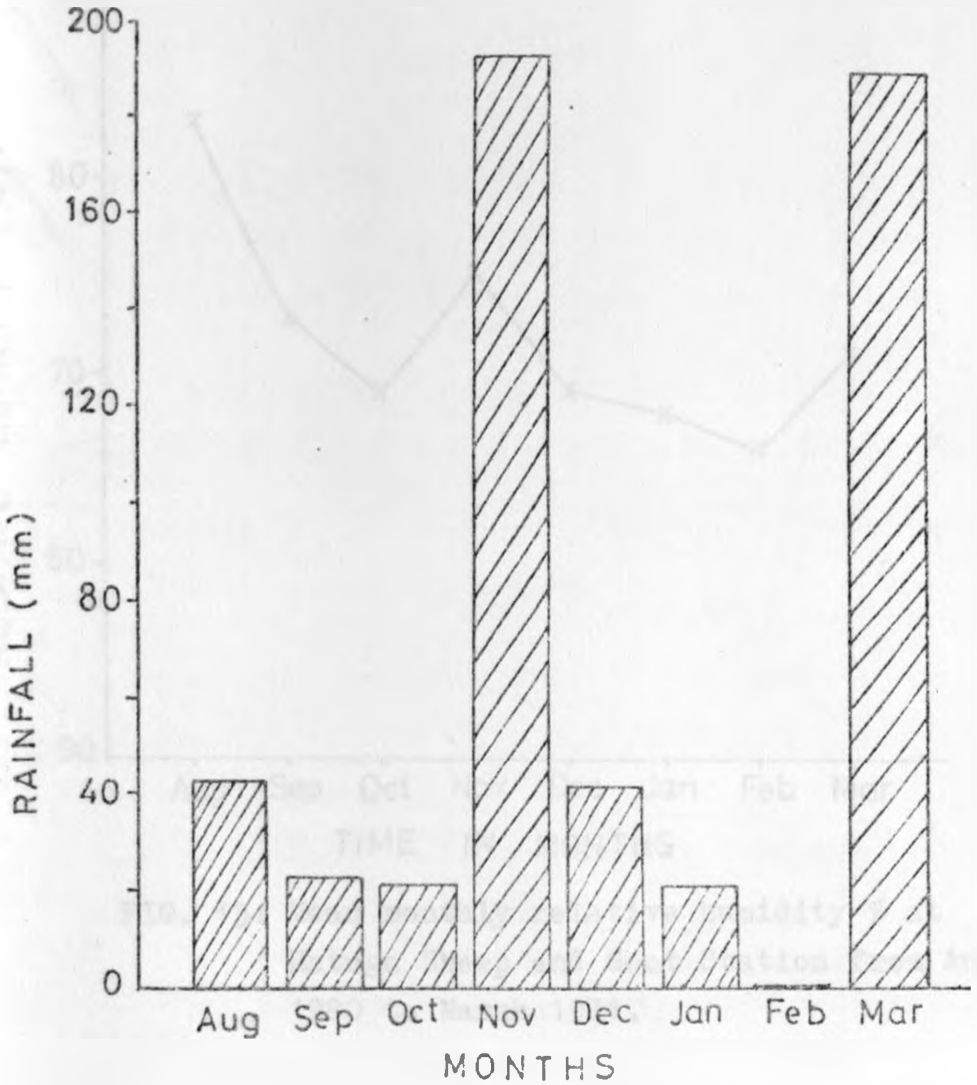


FIG. 12: Total monthly rainfall at Matuga Sheep and Goat Station from August 6th to March 31st 1981.

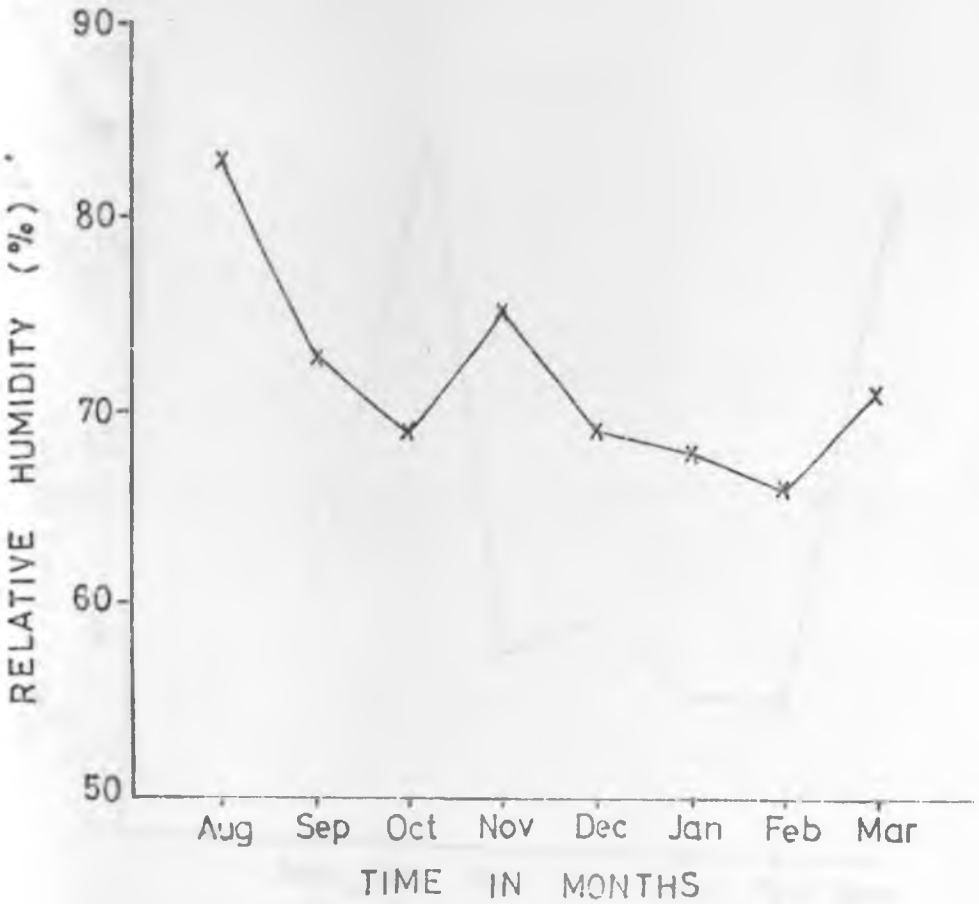


FIG. 13: Mean monthly relative humidity % at Matuga Sheep and Goat Station from August 1980 to March 1981.

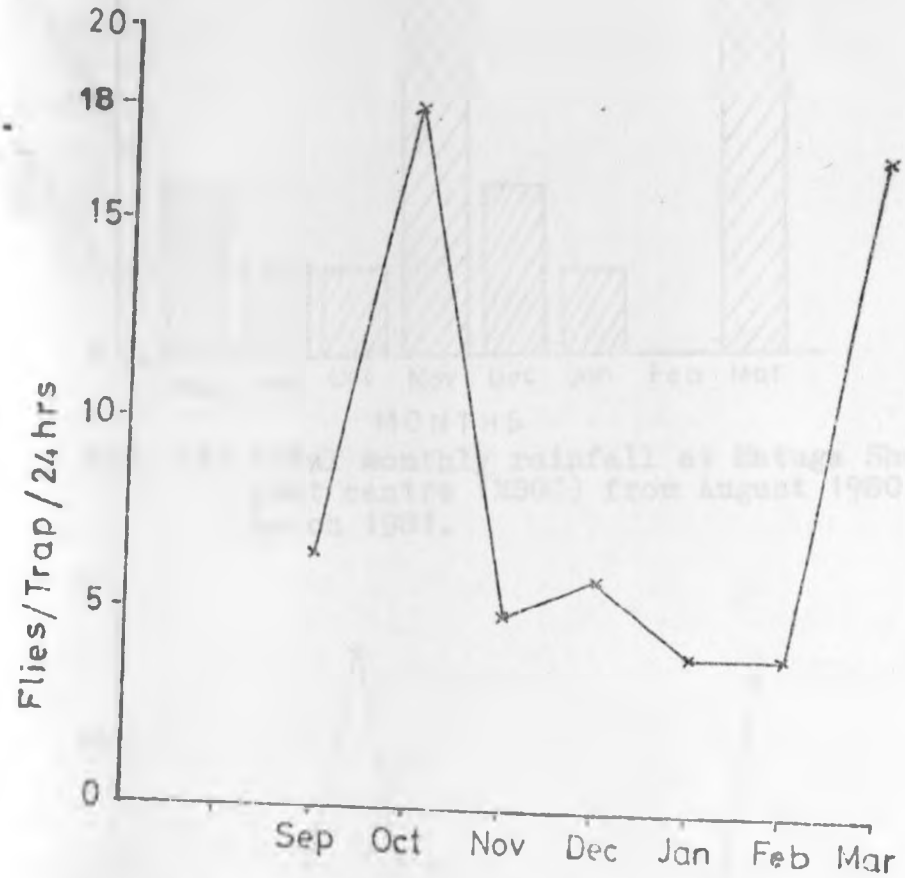


FIG. 14: Mean fly numbers (Glossina Spp) per trap per 24hrs at Matuga Sheep and Goat Centre from September 1980 - March 1981.

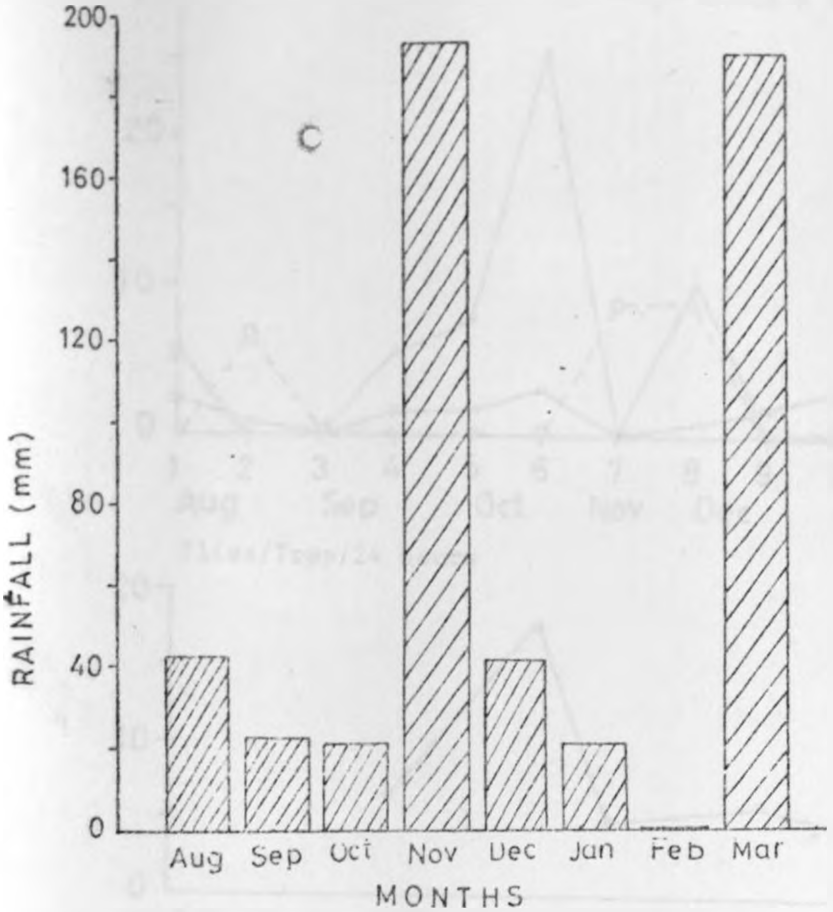


FIG. 12: Total monthly rainfall at Matuga Sheep and goat centre (MSGC) from August 1980 to March 1981.

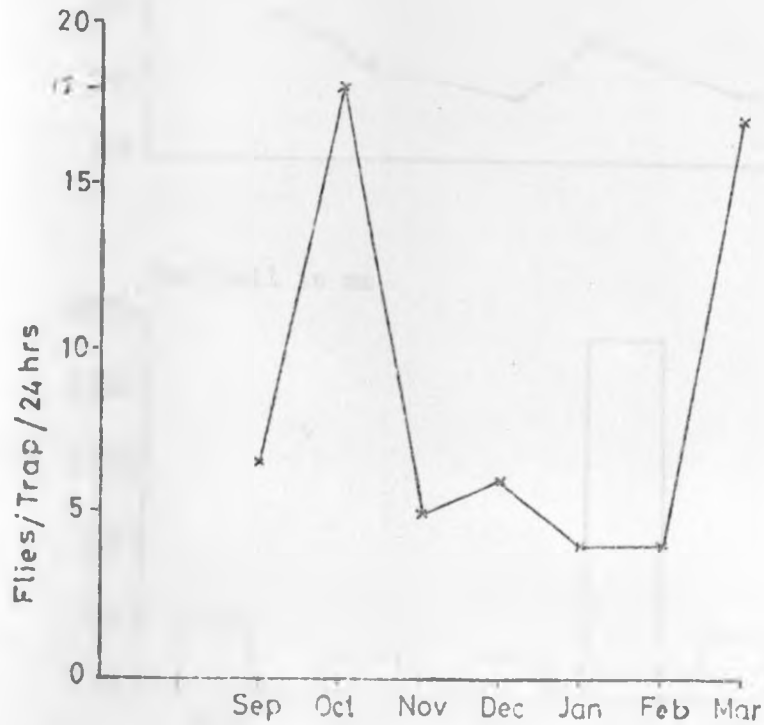


FIG. 14: Mean fly numbers (Glossina spp) per trap per 24 hours at MSGC from September 1980 - March 1981.

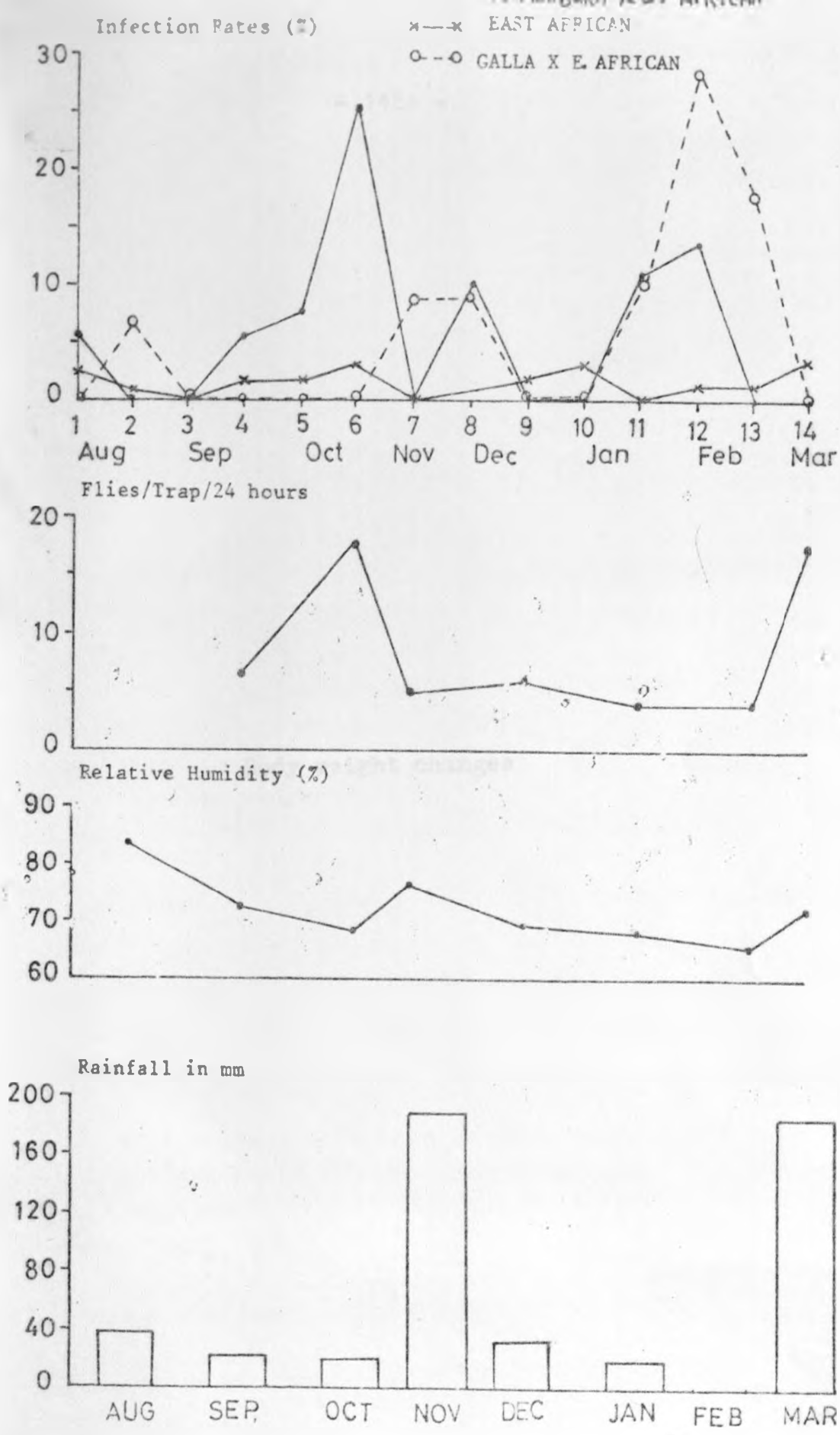


Fig. 14b: - Relationship between infection rates tsetse fly numbers, relative humidity and rainfall

Body weight changes

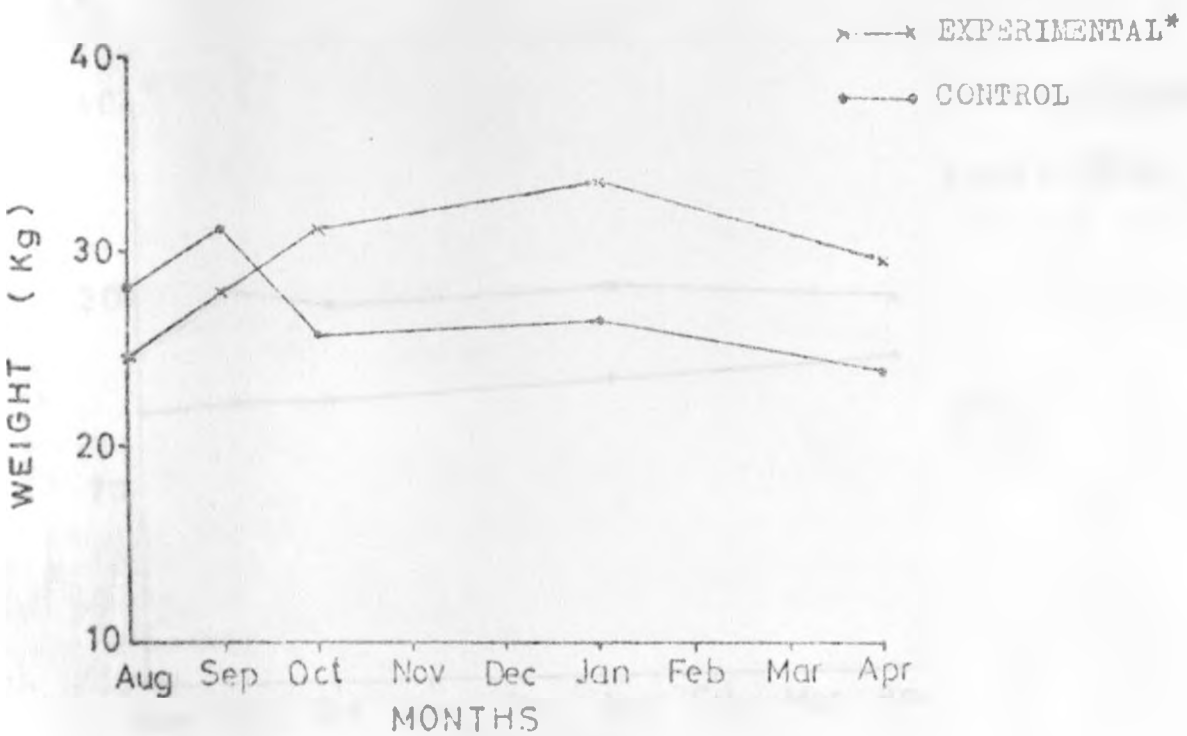


FIG. 15: Mean monthly body weights of Toggenburg X E. African goats between August 1980 and April 1981.

* Samorin treated.

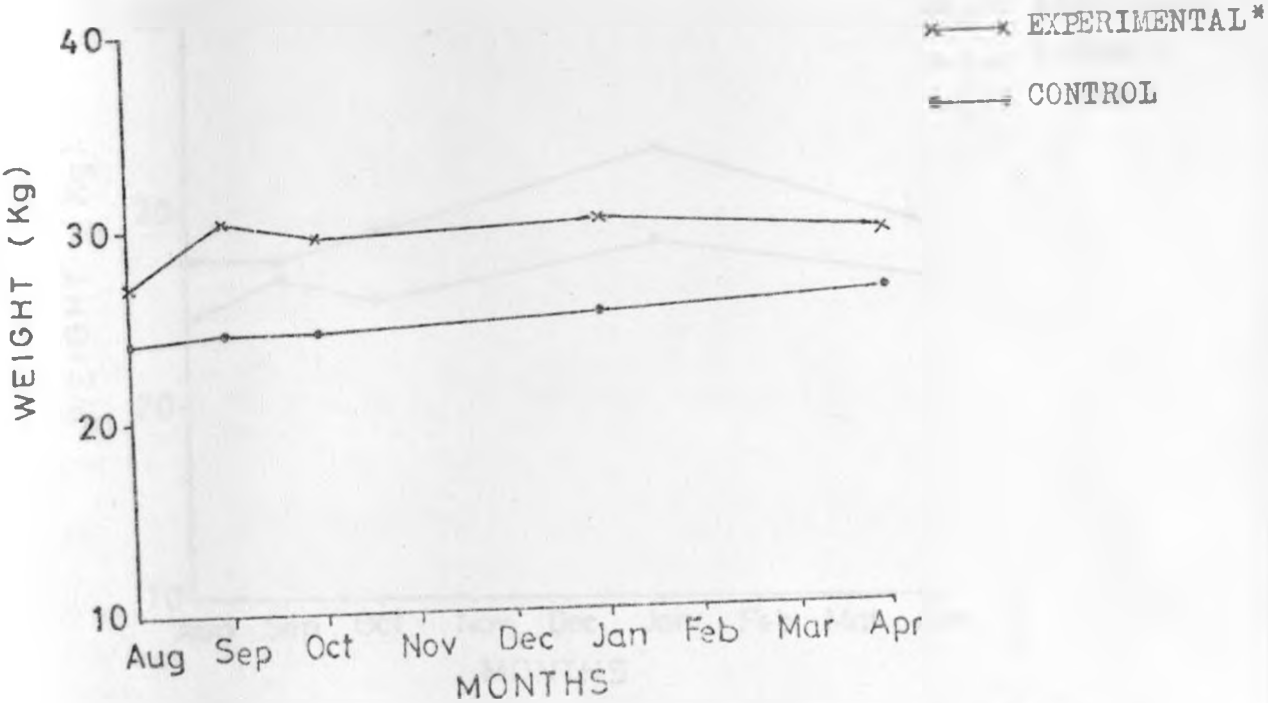


FIG. 16: Mean monthly body weights of Galla X E. African goats between August 1980 and April 1981.

* Samorin treated.

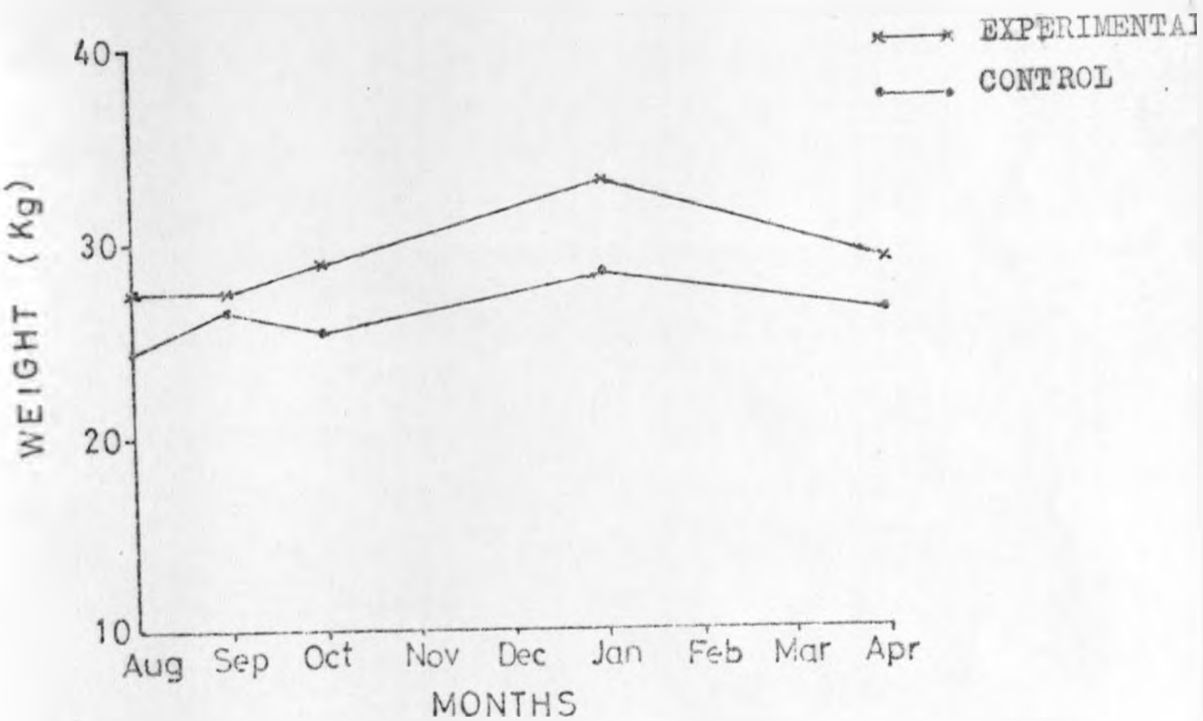


FIG. 17: Mean monthly body weights of East African goats between August 1980 and April 1981.

* Samorin treated.

HISTOPATHOLOGY

All the histopathologic sections shown on figures 18 to 23 are from the control group of goats.

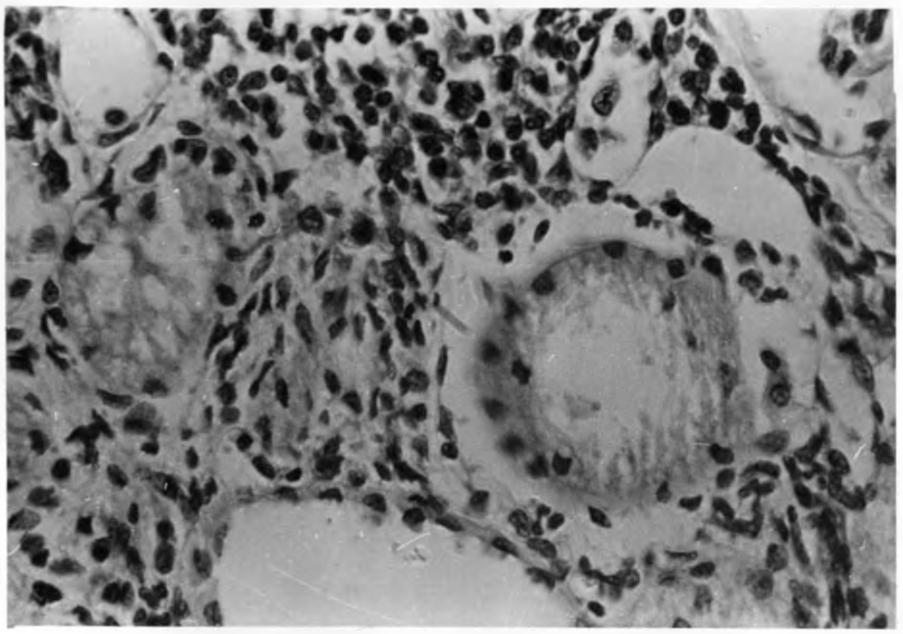


FIG. 18: KIDNEY: With mononuclear (Plasma) cell infiltration and tubular epithelial degeneration. (H & E, 1000)

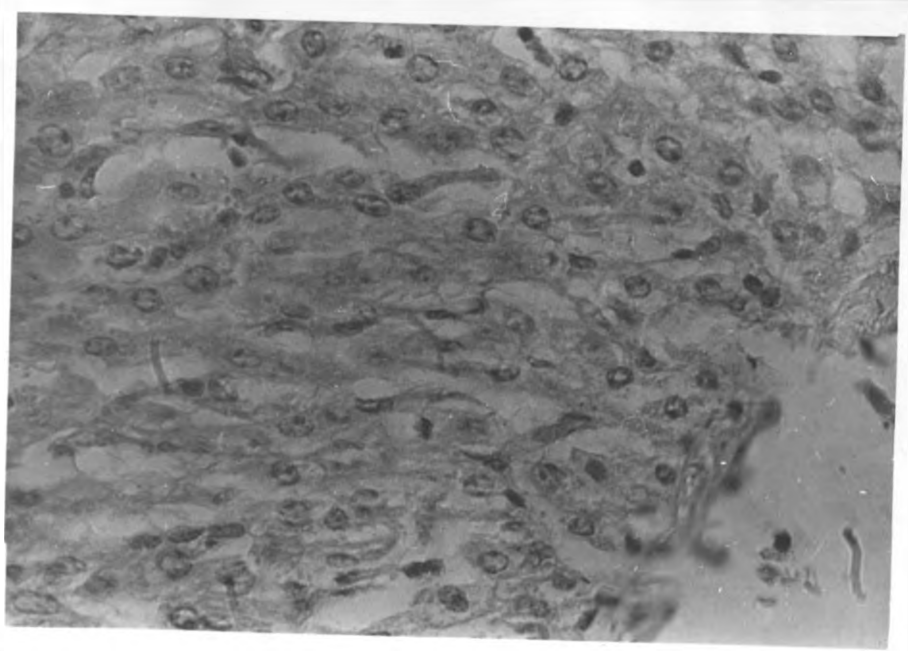


FIG. 19: LIVER: Showing centrilobular degeneration and a few mononuclear cells. (H & E, 1000) -

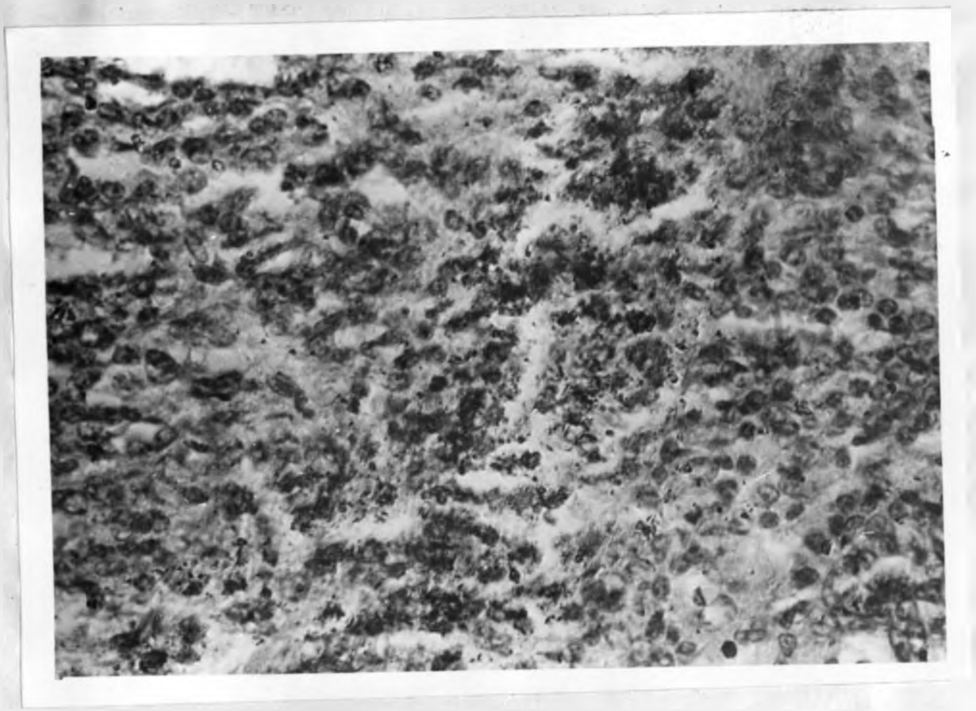


FIG. 20: Lymph node with hemosiderosis and oedema. (H & E, 1000)

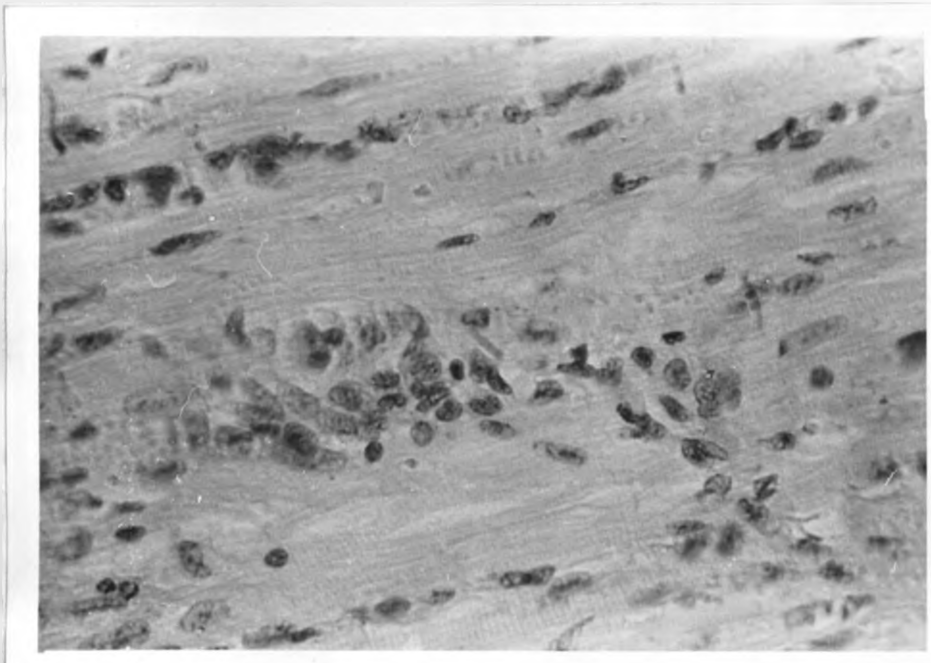


FIG. 21: Muscular tissue (Skeletal) with mononuclear cell infiltration. (H & E, 1000)

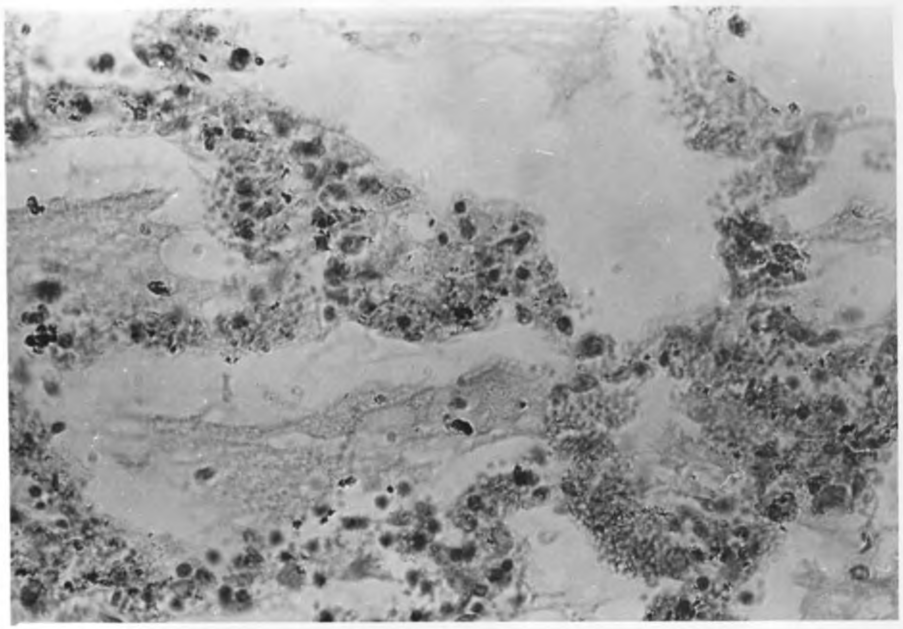


FIG. 22: Lung showing hemosiderosis, mononuclear cell infiltration and exudation.(H & E, 1000)

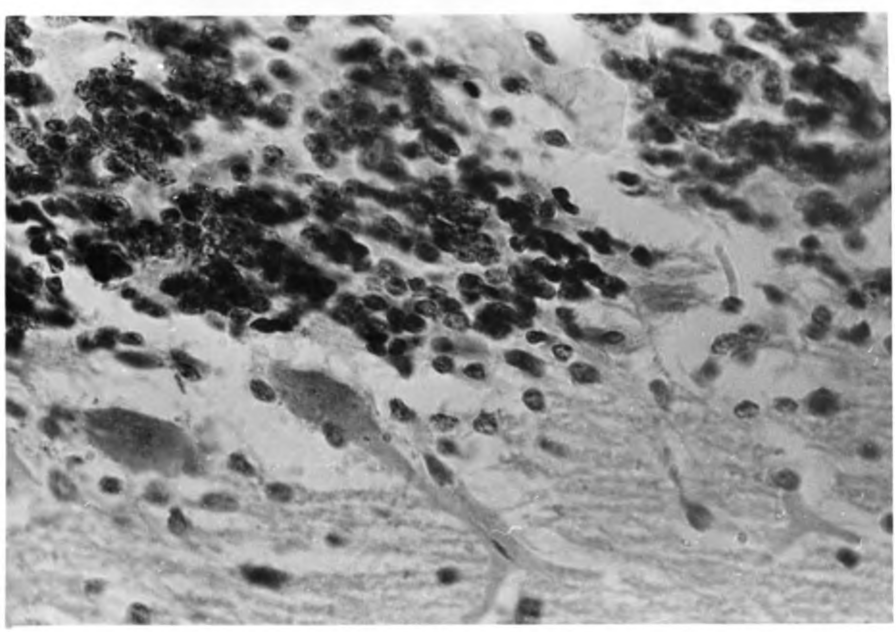


FIG. 23: Brain tissue showing mononuclear cell infiltration.(H & E, 1000)

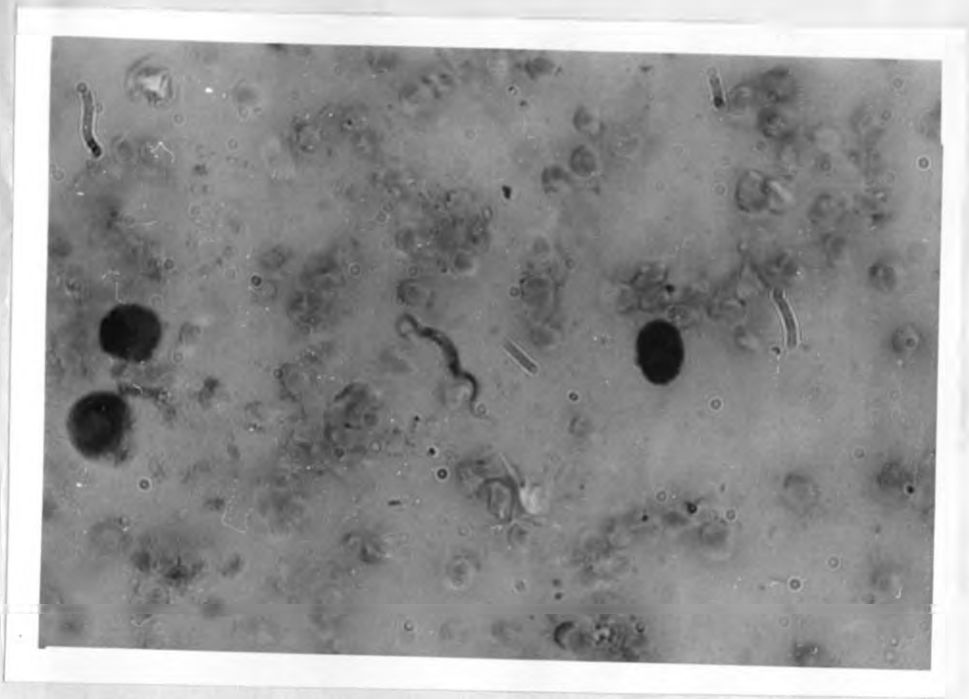


FIG. 24: Trypanosoma vivax isolated from a Galla X East African goat.(H & E, 1000)

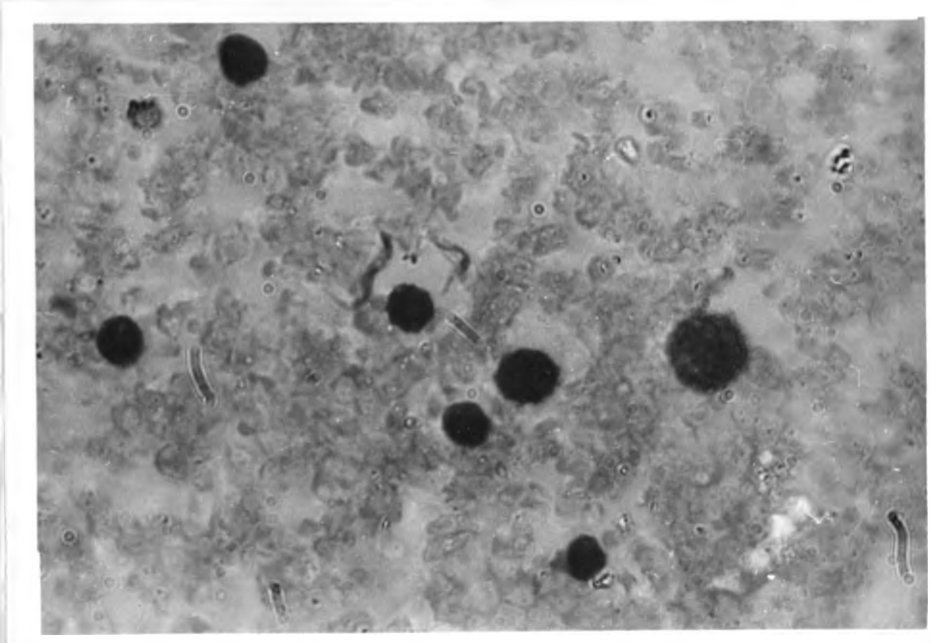


FIG. 25: Trypanosoma congolense from a Galla X East African goat. (H & E, 1000)

REFERENCES

ACTOR, P.W. & RAGANO, J.F; (1962): "Potentiation" of amphotericin B against T. congolense in mice" Proceedings of the society for experimental biology; New York; 110: 409 - 412.

ANOSA, V.O. & ISOUN, T.T. (1974): Experimental trypanosoma infection of sheep and goats: The relationship between parasitaemia, growth rate and anaemia. J. Nig. Vet. Med. Ass. 3: 102 - 108.

ANOSA, V.O. & ISOUN, T.T., (1976): "Serum proteins and blood plasma volumes in experimental Trypanosoma vivax infections of sheep and goats" Trop. Ani. Hlth. Prod. 8: 14 - 19.

Armed forces institute of pathology (1968): 'Manual of histology and staining methods' 3rd Edition, Washington D.C.

AUSTIN, W.C. COLLIER, H.O.J.; POTTER, M.D., SMITH, G.K.A. & TAYLOR, E.P. (1957): "New compound (Toxocide) active against T. congolense and T. vivax". Nature, London 179: 143 - 144.

BAKER, J.R., (1958): An attempt to infect white rats with T. vivax by giving them supplimentary injections of normal sheep serum" EATTRRO Annual Report P. 7.

BAKER, J.R. (1963): "Speculations on the evolution of the family Trypanosomatidae Doflein, 1901" Expl. Parasit. 13: 219 - 233.

BERGS, S; BROWN; K.N. LUCAS, J.M.S. (1961):
"The trypanocidal activity and local tolerance of sparingly soluble salts of metamedium"
Ann. trop. med. parasit, 55: 298 - 304.

BHATHACHARJEE, U.K. & SINHA, P.K. (1971): "Effect of berenil on T. evansi infection"
Indian vet. J. 48: 541 - 543.

BOOTS COMPANY LTD (N.D.) "The control of bovine trypanosomiasis"
Agrochemicals division, Nottingham, England.

BOREHAM, P.F.L., (1966): Sixth Seminar on Trypanosomiasis "Pharmacologically active peptides produced in tissues of the host during trypanosome infections"
Trans. R. Soc. trop. med. Hyg., 60: 124.

BROWN, K.N. (1966): Sixth seminar on trypanosomiasis: "Immunity in chronic trypanosomiasis and malaria"
Trans. R. Soc. trop. med. Hyg., 60: 124 - 125.

BRUCE, T; KOVATCH, R.M; CHUMO, D.A. & WYKOFF, D.E, (1978): "Trypanosoma congolense; thrombocytopenia in experimentally infected cattle"
Exp. Parasit; 45: 26 - 33.

BURDIN, M.L. (1953): "Delayed toxicity in cattle following the use of phenthridium coumpounds"
International veterinary congress Proceedings of the 15th. Meeting. Stockholm, pp 491 - 494.

CARLES, A.B. (1980): Department of Animal Production, University of Nairobi Kenya - Personnal communication.

CAWDERY, M.J.H. (1958): "Attempted cultivation of T. vivax in embryonated eggs" EATTRRO Annual Report, P. 7.

CHANDLER, R.L. (1952): "Comparative tolerance of West African N'Dama cattle to trypanosomiasis" Ann. : trop. med. Parasit., 46: 127 - 134.

CLARKSON, M.J., REES, J.M., CREWE, S. & AWAN, M.A.G. (1966): Sixth seminar on trypanosomiasis "trypanosomiasis in sheep" Trans. R. Soc. trop. med. Hyg., 60: 119.

CLARKSON, M.J. (1968): "Blood and plasma volumes in sheep infected with T. vivax" J. comp. path., 78: 189 - 193.

CONNAL, A. (1912): "Autoerythrophagocytosis in protozoal diseases" J. path. Bact. 16: 502 - 517.

DAVIS, C.E., ROBBINS, R.S., WELLER, R.D. & BRAUDE, A.I. (1974): "Thrombocytopenia in experimental trypanosomiasis" The Journal of Clinical Investigation, 53: 1359 - 1267.

EATTRO (1955): "Notes for field studies of tsetse flies in East Africa"
Edit. Potts and others, Nairobi.

FAO/WHO/10E (1962), Animal Health year book, 7th Ed.
Rome, 1963.

FAO (1978): Training Manual for tsetse control personnel vol. 2.
Edit. Pollock. P. 37.

FAO (1978): Tsetse and trypanosomiasis information quartery vol. 1.
Edit. Pollock, pp. 1 - 4.

FAO (1979): Report of the second consultation of programme for the control of African Animal Trypanosomiasis.
Rome, P. 23.

FIENNES, R.N.T. -W- (1946): "T. congolense (Broden) disease of cattle - The parenchymatous lesion and its relation to the cellular defences"
J. comp. path., 56: 28 - 37.

FIENNES, R.N.T. -W- (1950): "The cattle trypanosomiasis:- Cryptic Trypanosomiasis"
Ann. Trop. Med. Parasit., 44: 222.

FIENNES, R.N.T. -W- (1953): "The results of autopsies on trypanosome infected cattle".
Brit. Vet. J. 109: 511 - 520.

FIENNES, R.N.T. -W- (1954): "Haematological studies in trypanosomiasis of cattle"
Vet. Rec., 66: 423 - 434.

FIENNES, R.N.T. -W-, (1970): "Pathogenesis and Pathology of animal trypanosomiasis" In Mulligan's "The African Trypanosomiasis" George, Allen & Unwin, London 729 - 750.

FINELLE, P. (1974): "African Animal Trypanosomiasis" Part IV. Economic Problems. World Animal Review. 10: 15 - 18.

FISHER, R.A. & YATES, F. (1963): "Statistical tables for biological, Agricultural and Medical Research" Oliver & Boyd, Edinburg, Tweeddale Court; P. 46:

FORD, J. (1970): "Geographical distribution of Glossina" In Mulligan's 'The African trypanosomiasis' George, Allen & Unwin, London: 274 - 297.

FORD, J. (1971): 'The Role of Trypanosomiasis in African Ecology' CLAREDON PRESS, OXFORD: 54, 62.

GIBSON, W. (1981): KETRI - KIKUYU - KENYA Personnal correspondence.

GOODWIN, L.G. & UNSWORTH, K., (1952): "Effect of phenthridinium derivatives on T. vivax" Brit. J. Pharamacology: 7: 581 - 590.

GOODWIN, L.G. & UNSWORTH K., (1952): "Prophylactic and toxic actions of 150C47" Brit. J. Pharmacology; 7: 591 - 600.

GRAY, A.R. & STEPHEN, L.E. (1962): "A comparative trial of the local toxicity and prophylactic activity against (T. vivax and T. congolense) trypanosomiasis in West African Zebu cattle of Metamedium Chloride, Suramin Salt and embonate with antrycide prosalt". Vet. Rec. 74: 696 - 702.

GRIFFIN, L. (1978): "African trypanosomiasis in sheep and goats" Vet. Bull. 48: 819 - 824.

GRIFFIN, L. & ALLONBY, E.W. (1979a): "Disease syndromes in sheep and goats naturally infected with Trypanosoma congolense. J. comp. path. 89: 457 - 464.

GRIFFIN, L. & ALLONBY, E.W., (1979b): "Trypanotolerance in breeds of sheep and goats with an experimental infection of Trypanosoma congolense" Vet. Parasit. 5: 97 - 105.

GRIFFIN, L. & ALLONBY, E.W. (1979c): "The economic effects of Trypanosomiasis in sheep and goats at a range station in Kenya" Trop. Ani. Hlth. Prod. 11: 127 - 132.

GRIFFIN, L. & ALLONBY, E.W. (1979d): "Studies on epidemiology of trypanosomiasis in sheep and goats in Kenya" Trop. Ani. Hlth. Prod. 11: 133 - 142.

GUTTERIDGE, W.E. (1966): "Chemotherapy and drug sensitivity, further investigations on the mode of action of pentamidine"
Sixth Seminar on trypanosomiasis
Trans. R. Soc. Trop. Med. Hyg. 60: 120.

HARLEY, J.M.B. (1967): "Further studies on age and trypanosome infection rates in G. pallidipes Aust; G. palpalis fuscipes Newst; G. brevipalpalis, Newst. in Uganda".
Bull. ent. Res. 57: 459 - 476.

HIRUMI, H., DOYLE, J.J. & HIRUMI, K., (1977): "Cultivation of blood stream Trypanosoma brucei"
Bulletin of World Health Organisation 55: (2-3), 405 - 409.

HOARE, C.A. (1972): Trypanosomes of mammals
Blackwell Scientific Publications OXFORD & EDINBURG, Pg 57; 59; 60.

HORNBY, M.E. & BAILEY, H.W., (1931): "Diurnal variation in the concentration of Trypanosoma congolense in the blood vessels of the oxs ear"
Trans. Roy. Soc. Trop. Med. Hyg., 24: 557 - 564.

HORNBY, M.E. (1949): "Development of our knowledge on animal trypanosomiasis"
Vet. Rec. 61: 375 - 380.

ISOUN, T.T. & ANOSA, V.O. (1974): "The effect of experimental infection of Trypanosoma vivax on reproductive organs of sheep and goats" A preliminary report.
Nigerian J. of Animal production No. 2: 143 - 150.

ISOUN, T.T. & ISOUN, M.J. (1974): "In vitro cultivation of T. vivax isolated from cattle"
Nature 251: 513 - 514.

JONES - DAVIES, W.J. (1968): "Diminazene aceturate and homidium chloride: Resistance in tsetse fly transmitted trypanosomes of cattle in Northern Nigeria".
Vet. Rec. 83: 433 - 437.

JONES, T.C. & GLEISER, C.A., (1954): Veterinary necropsy procedures.
London, Sir Isaac Pitman & Sons Ltd; 23 - 37.

KAAYA, G.P. (1975): "Pathology and pathogenesis of Trypanosoma congolense in goats"
M.Sc. Thesis, University of Nairobi, Kenya.

KAAYA, G.P., WINQVIST, G. & JOHNSON, L.W. (1977): "Clinical pathological aspects of Trypanosoma congolense infection in goats"
Bull. Ani. Hlth. Prod. Afr. 25: 397 - 408.

KAAYA, G.P. & ODUOR-OKELLO, D. (1980): "The effect of Trypanosoma congolense infection on the tsetse and epididymis of the goat"
Bull. Ani. Hlth. Prod. Afr. 28: 1 - 5.

KALINER, G. (1974): Trypanosoma congolense.
II: Histologic findings in experimentally infected cattle.
Exp. parasit. 36: 20 - 26.

KEMPTHORNE O. . (1969): An introduction to genetic statistics, Iowa State University Press, Ames, Iowa, U.S.A. PP. 220.

KOBAYASHI, A., SOLTYS, M.A. & WOO, P.T.K. (1976): "Comparative studies on the laboratory diagnosis of experimental Trypanosoma congolense infection in sheep"
Ann. Trop. Med. Parasit. 70: 53 - 58.

KRAMER, J.W. (1966): "Incidence of trypanosomiasis in West African Dwarf sheep & goats in Nsukka, Eastern Nigeria".
Bull. Epizoo. Dis. Afri. 14: 423 - 428.

LANHAM, S.M. & GODFREY, D.G. (1970): "Isolation of salivarian trypanosomes from man and other mammals using DEAE-Cellulose".
Exp. Parasit. 28: 521 - 534.

LE-RICHE, W.H. (1971): "Epidemiology as medical ecology"
CHURCHILL, LIVINGSTONE, EDINBURG & LONDON,
Pg. 111 - 113.

LESTER, H.M.O. (1950): "Antrycide Research"
EATTRRO: Annual Reports Pg. 5

LINDAU, M. & SPIELBERGER, U. (1973): "Effect of Samorin (isometamedium) infections on growth and milk yield of West African Zebu of the Azaouak breed" Berliner und Munchener Tierarztliche wochenschrift 86: Heft 7, 129 - 130. Abstr. 4212.

LINK, R.P. (1965): "Drugs acting against special parasites" In Jones "Veterinary Pharmacology and Therapeutics" IOWA STATE UNIVERSITY PRESS, AMES, IOWA, U.S.A. Pp. 692 - 696.

LOSOS, G.J., PARIS, J., WILSON, A.J. & DAR, F.K. (1973): "Pathology of disease caused by Trypanosoma congolense in cattle" Bull. Epizoot. Dis. Afr. 21: 239 - 244.

LUCAS, J.M.S. (1955): "Transmission of Trypanosoma congolense in cattle under field conditions in absence of tsetse flies" Vet. Rec. 67: 403 - 406.

MACKENZIE, P.K.I., BOYT, W.P., NESHAM, V.W. & PIRIE, E. (1978): "Aetiology and significance of the phagocytosis of erythrocytes and leukocytes in sheep infected with Trypanosoma congolense (Broden, 1904)" Res. Vet. Sci. 24: 4 - 7.

McDIARMID, A. (1962): "Diseases of free-living wild animals" FAO of the United Nations, Rome P. 72.

MEDWAY, W. (1969): "Textbook of Veterinary Clinical Pathology"
BAILLIERE, TINDALL & CASSELL, LONDON, P. 230.

MORTELMANS, J. & KAGERUKA, I. (1976): "Trypanotolerant cattle breeds in Zaire"
WORLD ANIMAL REVIEW: 19: 14 - 17.

MULLIGAN, H.W. (1970): "The African trypanosomiasis"
George Allen & Unwin LTD., London P. 71.

MURRAY, M. & MORRISON, W.I. (1978): "Recent developments in trypanotolerance" FAO Report of the second consultations of the programme for the control of African animal trypanosomiasis. Pp. 66 - 69.

NYINDO, M., DARJI, N.: KAMINJOLO, J.S. Jr., CHIMTAWI, M, PATEL, N. & ONYANGO, P. (1978): "Trypanosoma brucei - Cultivation in Vitro of infective forms derived from the midgut of Glossina Morsitans' J. Parasit, 64: (3), 469 - 474.

OMUSE, J.K., (1973): "Pathogenesis of Trypanosoma congolense in cattle"
M.Sc. Thesis, University of Nairobi, KENYA.

OPPONG, E.N.W., (1969): "Toxicity of berenil in dog"
Vet. Rec. 85: 370.

PAIKNE, D.L. & DHAKE, P.R. (1972): "Abortion due to Trypanosoma evansi in a she buffalo"
Indian Vet. J., 49: 1091 - 1092.

PARKIN, B.S. (1935): "Symptomotology of some trypanosomiasis of domestic animals" Onderstpoort J. Vet. Sci. Ani. Ind., 4: 251 - 267.

RICHARDSON, U.F. & KENDALL, S.B., (1957): Veterinary Protozoology, 2nd Edit. Oliver & Boyd, Edinburg, Tweedale Court, London, 39A WELBECK STREET, W.I., Pp. 26 - 44.

ROBERTS, C.J. & GRAY, A.R., (1973): "Studies of trypanosoma resistant cattle (1) The breeding and performance of N'Dama, Muturu and Zebu cattle maintained under the same conditions of husbadry" Trop. Ani. Hlth. Prod., 5: 211 - 233.

ROBERTSON,D.H.H. (1959): "Nitrofurazone in T. rhodesiense Sleeping sickness" EATRO: Annual report, P. 41.

ROBERTSON, D.H.H. (1961): "Hemolytic anaemia in a woman treated with nitrofurazone" EATRO: Annual report P. 51.

ROBSON, J. & ASHKAR, T.S. (1972): "The efficiency of different diagnostic methods in animal trypanosomiasis - Based on surveys carried out in Nyanza Province of Kenya" Bull. Epizoot. Dis. Afri. 20: 303 - 306.

SCHALM, O.W., (1965): Veterinary Haematology LEA & FEBIGER, Philadephia; Pp. 85 & 247.

SCHIDT, H., KADUK, B., & FINK, E., (1978):
"Pathomorphology of side - effects of
Trypanosidic diamidines"
Vet. Path., 15: 574.

SLOTS, J.M.M.: Van MIERT, A.S.J.P.A.M., AKKERMAN,
J.W.N. & DE GEE, A.L.W., (1977):
"Trypanosoma brucei and Trypanosoma vivax:
Antigen-Antibody complexes as a cause of
platelet serotonin release in Vitro and in vivo
Exp. Parasit: 43: 211 - 219.

SNOW, W.F., (1979): "The ecology and Epidemiology
of Trypanosomiasis in the Gambia and on
the South Coast of Kenya", A paper
presented at the 16th Meeting of the International
Scientific council for trypanosomiasis research
and control YAOUNDE.

SNOW, W.F. & TARIMO, S.A. (1981) - ICIPE
Personal Communication.

SOLTYS, M.A., (1953): "Transmission of T. congolense
by Vectors other than tsetse".
EATTRRO: Annual Report. P. 30.

STEPHEN, L.E., (1966): "Observations on the
resistance of West African N'Dama and
Zebu cattle to trypanosomiasis following
challenge by wild Glossina morsitans
from an early age".
Ann. Trop. Med. Parasit., 60: 230 - 246.

STEPHEN, L.E. (1970): "Clinical manifestations of trypanosomiasis in livestock and other domestic animals". In Mulligans "The African trypanosomiasis".
George Allen & Unwin LTD; London, 775 - 792.

STEWART, J.L., (1951): "The West African Short horn cattle; their value to Africa as trypanosomiasis resistant animals"
Vet. Rec. 63: 454 - 457.

Van den INGH, G.A.M., SWART, D: SCHOTMAN, A.J.H, Van MIERT, A.S.J.P.A. & VEENENDAAL, G.H., (1976): "Pathology and Pathogenesis of Trypanosoma vivax in goats"
Res. Vet. Sci; 21: 264 - 270.

Veterinary Services department - Kenya, Annual Reports 1962 - 1979.

WEITZ, B.G.F. (1970): "Infection and Resistance; Practical significance of Immune responses"
In Mulligans; 'The African trypanosomiasis"
George Allen & Unwin Ltd; London: 118 - 124.

WELLDE, B., LÖTZSCH, R., DEINDL, G., SADUN, E., WILLIAMS, J. & WARUI, G. (1974): "Trypanosoma congolense; 1. Clinical observations of experimentally infected cattle".
Exp. Parasit. 36: 6 - 19

WHITESIDE, E.F. (1958): "The control of animal trypanosomiasis in Kenya".
Zoological Section, Department of Veterinary Services in Kenya.

WILLET, K.C., (1955 - 1956): "Chemotherapy"
EATRO: Annual Reports. P. 35.

WILLIAMSON, J., (1966): "Trypanosocidal drug action: Some observations on synergism". Sixth Seminar on Trypanosomiasis.
Trans. Soc. Trop. Med. Hyg. 60: 121.

ZUCKERMAN, A., (1964): "Autoimmunization and other types of indirect damage to host cells as factors in certain protozoan diseases"
Exp. Parasit. 15: 138 - 183.

ZWART, I; PERIE, N.M., KEEPPLER, A., & GOEDBLOED, E., (1973): "A comparison of methods of diagnosis of trypanosomiasis in East African domestic ruminants"
Trop. Ani. Hlth. Prod. Afr. 5: No. 2: 79 - 86.