SOME ASPECTS OF THE EPIDEMIOLOGY AND DRUG SENSITIVITY OF BOVINE TRYPANOSOMOSIS IN KAURA LOCAL GOVERNMENT AREA OF KADUNA STATE

BY

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DEDICATION

This thesis is dedicated to my late father, Mr. Maikaje Shekarau, for laying the foundation of my academic carrier even though he did not live long enough to taste the fruits of his labour.

DIGITIZED

DECLARATION

I declare that the research published in this thesis was carried out by me in the Department of Parasitology and Entomology, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria.

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CERTIFICATION

This thesis, entittled **SOME ASPECT OF THE EPIDEMIOLOGY OF BOVINE TRYPANOSOMOSIS IN KAURA LOCAL GOVERNMENT AREA OF KADUNA STATE,** by **DOMINIC BAWA MAIKAJE,** meets the regulations governing the award of the degree of **DOCTOR OF PHILOSOPHY** of the Ahmadu Bello University, Zaria, and is approved for its contribution to knowledge and literary presentation.

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ABSTRACT

Outbreaks of tsetse fly infestation and bovine trypanosomosis in Kaura Local Government area (LGA) of Kaduna State, leading to high cattle mortality and mass exodus of settled Fulani pastoralists from the area were reported in the National Concord daily Newspaper of 6th November, 1990. Using the biconical and NiTse tsetse traps, Glossina palpalis palpalis and G. tachinoides were caught only along the banks of River Kajim in Kaura LGA. Stomoxys calcitrans. Haematopota spp, and Tabanus spp were detected in every part of the LGA using the same tsetse trapping equipment. Results of parasitological surveys conducted in this LGA revealed dry and rainy seasons bovine trypanosomosis prevalence rates of 17.26% and 53.04%, respectively. The antigen capture enzyme-linked immunosorbent assay (ELISA) revealed bovine typanosomosis prevalence rates of 49.40% and 63.48%, respectively during the two seasons. The principal etiologic agents of bovine trypanosomosis detected during these investigations were Trypanosoma brucei, followed by T. congolense and T. vivax and mixed infections with the three trypanosome species.

Drug sensitivity tests conducted in Red Sokoto goats revealed complete sensitivity of the X, brueei isolate to diminazene diaceturate (Berenil^{R}) and isometamidium chloride (Samorin), at doses of 7 mg/kg and 0.5 mg/kg body weight respectively. On the other hand, the T. congolense isolate in one of three experimentally infected goats relapsed to diminazenc at 7 mg/kg body weight three weeks post treatment. The remaining two, and three others from another group, infected with the same trypanosome isolate were cured with the 7.0 mg/kg diminazene and 0.5 mg/kg isometamidium respectively.

It was concluded that there were outbreaks of bovine trypanosomosis and its vectors which could hamper cattle breeding in Kaura LGA. The seemingly efficient trapping of the bovine trypanosomosis vectors using the biconical and NiTse tsetse traps observed during this study suggests that the large scale use of this locally fabricated equipment might effectively control these biting flies in Kaura LGA. Similarly, inspite of the rampant misuse of trypanocidal drugs in this LGA, many trypanosome isolates are still

apparently sensitive to the therapeutic doses of the commonly used trypanocides - diminazene diaceturate and isometamidium cloride. The chemothcrapcutic/chcinoprophylactic treatment of cattle may provide another viable option for the control of bovine trypanosomosis in Kama LGA.

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CHAPTER I

INTRODUCTION

CHAPTER I

1.0 INTRODUCTION

African trypanosomosis has long been recognised as the most debilitating livestock disease by owners of these animals in subsaharan Africa many years before the exact description of the vector, causative agent and mode of transmission (Ibrahim, 1986). The African livestock owner became increasingly aware with time, that certain zones were relatively free, others were endemic foci, while many were seasonally endemic for the disease (Omoogun, 1987). This knowledge partially influenced the evolution of the various age long traditional practices such as the treatment of infected cases with such herbs as Cochlospermum tinctorum (Ibrahim, 1986) and nomadic husbandry practices that characterise the livestock industry in Africa south of the Sahara (Aliu, 1975; Nuru, 1986). Despite these strategies adopted by the livestock owners to prevent the desease in their stock, the devastating effects of African animal trypanosomosis have continued to retard the development and growth of the livestock industry most especially in the subhumid zones of Africa (Nantulya and Moloo, 1989; Nantulya, 1990). The economic impact of trypanosomosis results from reduced resistance to other infectious diseases, low milk and meat yields, reduced work output by draught animals and those used as beasts of burden, stunted growth, infertility and high mortality rates (Finelle, 1974; Tacher, 1982; Hoste, 1987; Connor, 1989; ILRAD, 1990).

The endemicity and the socio-economic effects of African animal trypanosomosis have been reported in 37 African countries lying between latitudes 15^0 N and 30^0 S. It has been documented that this desease has rendered between 8 -15 million square kilometers (km^2) or 37% of potentially suitable land for livestock production unexploited (Finelle, 1974; Losos, 1986; Mortelmans, 1986; Nantulya and Moloo, 1989; Jabbar, 1994). Other reports in the literature indicate that cattle are the worst affected livestock species by this animal disease in the endemic areas of Africa (Barret, 1997) for the following obvious reasons:

1. Cattle are the fifth in population size in Nigeria (RIM, 1992) and the most exposed livestock to biting flies and thus to animal trypanosome challenges (Von Kaufmann; 1986). This is because raising these animals is still traditionally nomadic, which involves seasonal long distance and local migrations and extensive grazing through the endemic belts and foci of this disease (Aliu, 1975; Bourn et al., 1986)

2. Cattle are the most preferred hosts of tsetse and other fly vectors of animal trypanosomosis (Kalu et al., 1991) and only in their absence are other sources of blood meals such as small ruminants, pigs and reptiles, sought for (Omoogun, 1987; Laveissier et al., 1990).

Although the direct economic impact of bovine trypanosomosis has not been well evaluated in Nigeria, losses due to trypanosome-induced arbortion have been reported by many workers (Kirkby, 1963; Leeflang, 1975; Ogwu, 1983). Finelle (1974), the FAO (1976), Jemal et al. (1995) and Rowlands et al. (1996), estimated that the cattle population in sub-saharan Africa would rise several times if tsetse flies, the main vectors of bovine trypanosomosis, are controlled. It is estimated that 50 - 52 million out of approximately 167 - 173 million cattle in Africa are at risk of being infected by this disease (Nantulya and Moloo, 1989, Hursey and Slingenbergh, 1995; Shaib et al., 1997).

Although there are 11 species of tsetse flies in Nigeria, only 4 are actively involved in the cyclical transmission of bovine trypanosomosis where ever they are found (Omoogun, 1987). These include Glossina morsitans submorsitans G. palpalis palpalis, G. tachinoides and G. longipalpis. However, other biting flies belonging to the families Tabanidae, Muscidae and Hippoboscidae seem to be the primary mechanical vectors of bovine trypanosomosis outside the tsetse infested areas (Agu, 1981; Losos, 1986; Clair, 1987; Omoogun, 1987). The major pathogenic animal trypanosomes causing bovine trypanosomosis in the endemic areas of Africa include Trypanosoma congolense, T. brucei, and T. vivax (Ikede, 1986). The disease in cattle is characterized by fever, anaemia, progressive weakness, cachexia, abortion, still birth, infertility and death in untreated cases (Ikede et al., 1988; Nantulya and Moloo, 1989; Sekoni, 1994; Jeffcoate and Holmes, 1997).

The subhumid ecological zone of Nigeria occupies approximately 455,000km or 50% of the country. It is located between latitudes $8^0.30'$ and $10^0.35'$ north of the equator and has adequate water, good fodder, suitable vegetation and climate most conducive for increased livestock production (Waters-Bayer and Taylor-Powell, 1986; von Kaufmann, 1986; Omoogun, 1987). However, the same climatic conditions have made this area the most endemic for tsetse and African trypanosomosis, thus constituting an obstacle to livestock development and growth, (Mohammed - Saleems, 1986; Omoogun, 1987).

Duggan (1962) and von Kaufmann (1986) among others have reported that the subhumid zone of Nigeria as the most endemic ecological area for tsetse and African trypanosomosis. Consequently, various strategies aimed at controlling the disease and the vectors have been evolved and instituted over the years by various government agencies, international organisations, nomadic livestock owners and rural farmers (McMillan and Meltzer, 1996; Barret, 1997). Those aimed at eliminating the trypanosome parasites included case detections (Kalu et al., 1986; Maikaje et al.; 1989; Akinpelu and Oyejide, 1990) and trypanocide treatment (Leeflang, 1978; Aliu, 1981; 1997; Brun, 1993). On the other hand, those aimed at eradicating the tsetse vector were massive deforestation (Nash, 1941; 1948) glocinicide spraying (Bature, 1981; Dimloug, 1981), the sterile male insect technique (Sarmeintor and Jawonisi, 1981), and land utilization (Ologe et al.; 1981; Bourn et al.,1986; Omoogun et al., 1991). Consequently, approximately 210,000 square kilometers (kms) of tsetse infested land had been reclaimed by 1981 (Bature, 1981). Coupled with intensive land use for agricultural and other developmental projects, cases of bovine trypanosomosis declined and cattle population and densities in the subhumid zone of Nigeria increased (Bourn et al, 1986).

Unfortunately the severe national economic depression which began in 1983 resulted in the reduction of government funding for tsetse and trypanosomosis research and Control (ILRAD, 1987). The subsequent collapse of these projects, coupled with the growing cases of trypanosome drug resistance (ILRAD, 1987) have resulted in tsetse reinvasion and resurgence of animal trypanosomosis in areas previously reclaimed (Agu et al., 1989) and in others known to be free in the past (Edeghere et al., 1989; Joshua and

Shanthikutmar, 1989). Consequently the 9 to 13.9 million cattle population in Nigeria (von Kaufmann, 1986; Bincan, 1990; RIM, 1992) is now facing increased risk of infection. Nationwide surveys conducted by Ilemobade et al. (1991) indicate that approximately 1-10% bovine mortality and 10-50% mobidity are due to animal trypanosomosis. Similarly reported outbreaks of tsetse fly infestation and bovine trypanosomosis in Kaura LGA, leading to mass exodus of Fulanis and their cattle from this area compelled the Chief of Moroa to make a plea to the Kaduna State government for assistance to control their ravaging effects on cattle (National Concord, daily newspaper, 1990). Consequently epidemiological and drug sensitivity studies were designed to assess the incidence of, and the effects of trypanocides treatment on bovine trypanosomosis in Kaura LGA in order to evolve control measures that will permit the exploitation of the abundant resources (fodder and water) available for large scale cattle production.

CHAPTER II

LITERATURE REVIEW

CHAPTER II

2.0 LITERATURE REVIEW

2.1 CATTLE POPULATION, BREEDS AND DISTRIBUTION IN NIGERIA

2.1.1 CATTLE POPULATION IN NIGERIA

Lamorde and Franti (1975) once estimated that Nigeria had 9.3 million cattle within her borders. Eleven years later, von Kaufmann (1986) also reported that Nigeria had an estimated total population of 9.3million cattle. Within the same year Bourn et al., (1986) guessed that Nigeria had 10-15 million cattle residing within her territory. The most recently compilled figures documented by the National Livestock Projects Department (NLPD) in 1992 and the Resource Inventory and Management Limited (RIM) in 1992 showed that there are approximately 13.9 to 14 million cattle in Nigeria.

2.1.2 CATTLE BREEDS IN NIGERIA

There are probably no breeds of cattle that are strictly indigenous to Nigeria. The large humped Zebu cattle (Bos indicus) might have been introduced into the country from the present day Niger, Mauritania and Chad during the advent of the Islamic Jihad (holy war) in the 19th century, which was mainly carried out by the predominantly Nomadic Fulani cattle owners (Water-Bayer and Taylor-Powell, 1986). On the other hand the West African dwarf cattle breeds (Bos taurus) reportedly began to be imported into Nigeria from Ivory Coast, Zaire, Guinea and the Gambia about 7000 years ago (Ikomi, 1981; Meghe et al, 1994, Rege et al., 1994). The trypanosusceptible Zebu breeds which were introduced into Africa about 1400 years ago (Aliu, 1997) inhabit mainly the less tsetse infested Northern States of Nigeria. They include the Rahaji (red bororo) Wadara (shuwa), kuri, Adamawa and Sokoto gudali, while the trypanotolerant humpless taurine breeds-Ndama, muturu, and Keteku-inhabit the tsetse endemic Southern States (Ikomi, 1981; Bourn et al., 1986).

2.1.3 CATTLE DISTRIBUTION IN NIGERIA

The distribution of cattle in Nigeria is not restricted within confined borders, but is guided by such factors as the original breed habitat, availability and accessibility to all year round good forage, free grazing range, drinking water, land for the settlement of and crop cultivation by the pastoral Fulanis and freedom of the settlement and grazing land from tsetse and trypanosomosis (Bourn et al., 1986; Moloo, 1993). Most of the cattle are found in the Northern three - quarters of Nigeria (Figs. 1 & 2). The bulk of the population of these animals reside in the far North during the rainy season and in the subhumid zone during the dry season (Bourn et al., 1986). However, there seems to have been a marked southward drift in the distribution of cattle population since the 1950s (Fricke, 1979; Putt et al., 1980). The increasingly harsh climatic conditions of the semi-arid sahel zone of the north, the declining incidence of animal trypanosomosis, the abundant forage, water and land for free range grazing and crop cultivation in the subhumid zone have encouraged this southward drift of the Fulani herdsmen. This has led to the establishment of semi-nomadic or sedentary livestock settlements in the middle belt states of Nigeria (Waters-Bayer and Taylor-Powell, 1986). Van Ray (1975) had earlier observed that only 12% of the Nigerian Fulani cattle keepers were fully nomadic, while he considered 38% to be semi-settled and the remaining 50% to be permanently settled expecially in the subhumid zone of the country. Estimates of the State distribution of cattle population compiled by the National Livestock Projects Division (1992) shows that approximately 6,121,000 cattle inhabit the Semi-arid Northern States of Kano, Jigawa, Katsina, Sokoto, Kebbi, Borno and Yobe; the subhumid zone consisting of Kaduna, Bauchi, Plateau, Adamawa, Taraba, Kwara, Kogi, Benue and Niger States has a total cattle population of 7,015,000 while the Southern States of the humid zone consisting of Rivers, Akwa Ibom, Cross River, Imo, Abia, Enugu, Anambra, Oyo, Ondo, Lagos, Osun, Delta and Edo is occupied by 479,000 cattle. The large trypanosusceptible zebu cattle breeds made up of the Adamawa and Sokoto Gudali, Azawak, Bunaji (White Fulani), Rahaji (Red Bororo), Wadara (Shuwa), and the Kuri predominate the northern semi-arid and subhumid zones of Nigeria, while the Southern humid zone of the country is occupied by the dwarf trypanotolerant breeds, namely the Muturu, Keteku and the Ndama (RIM, 1992).

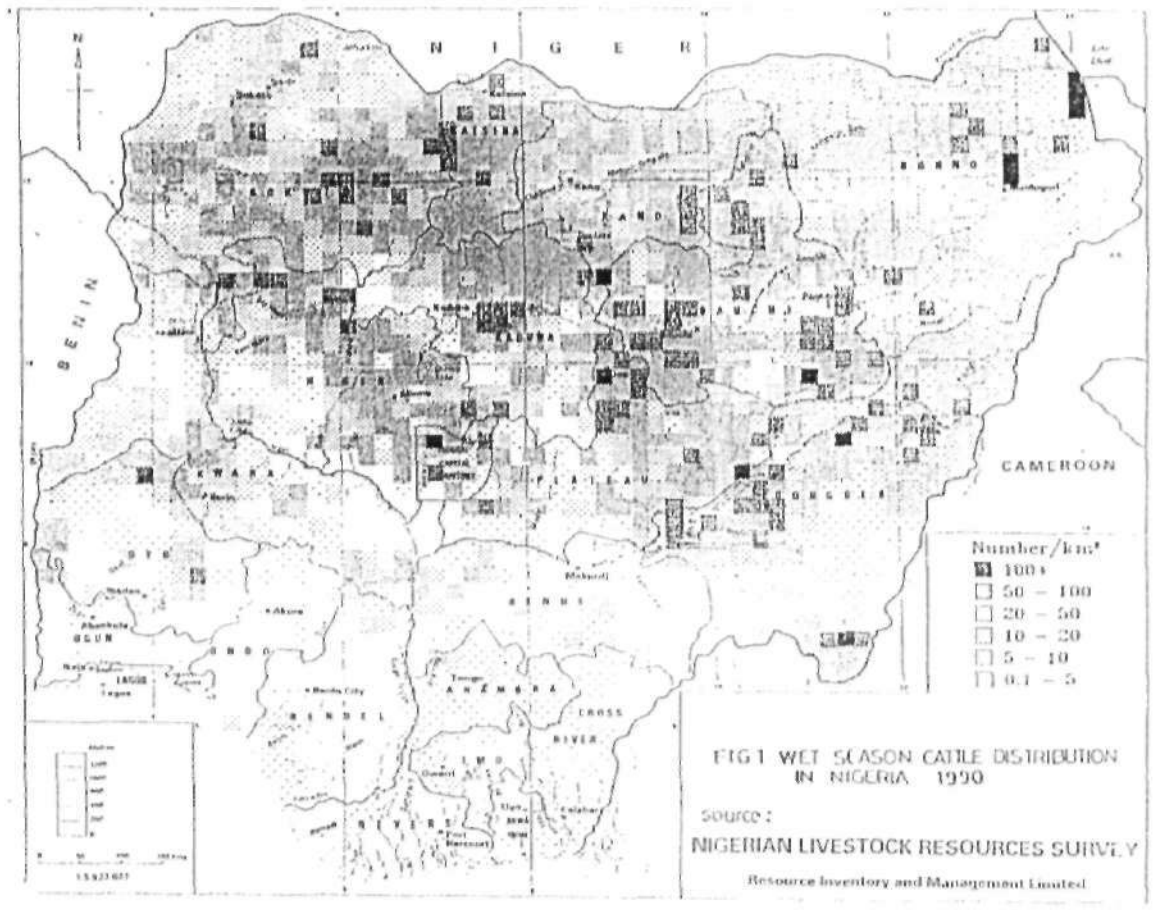


FIG 1 WET SEASON CATTLE DISTRIBUTION IN NIGERIA 1990

Source :

NIGERIAN LIVESTOCK RESOURCES SURVEY

Resource Inventory and Management Limited

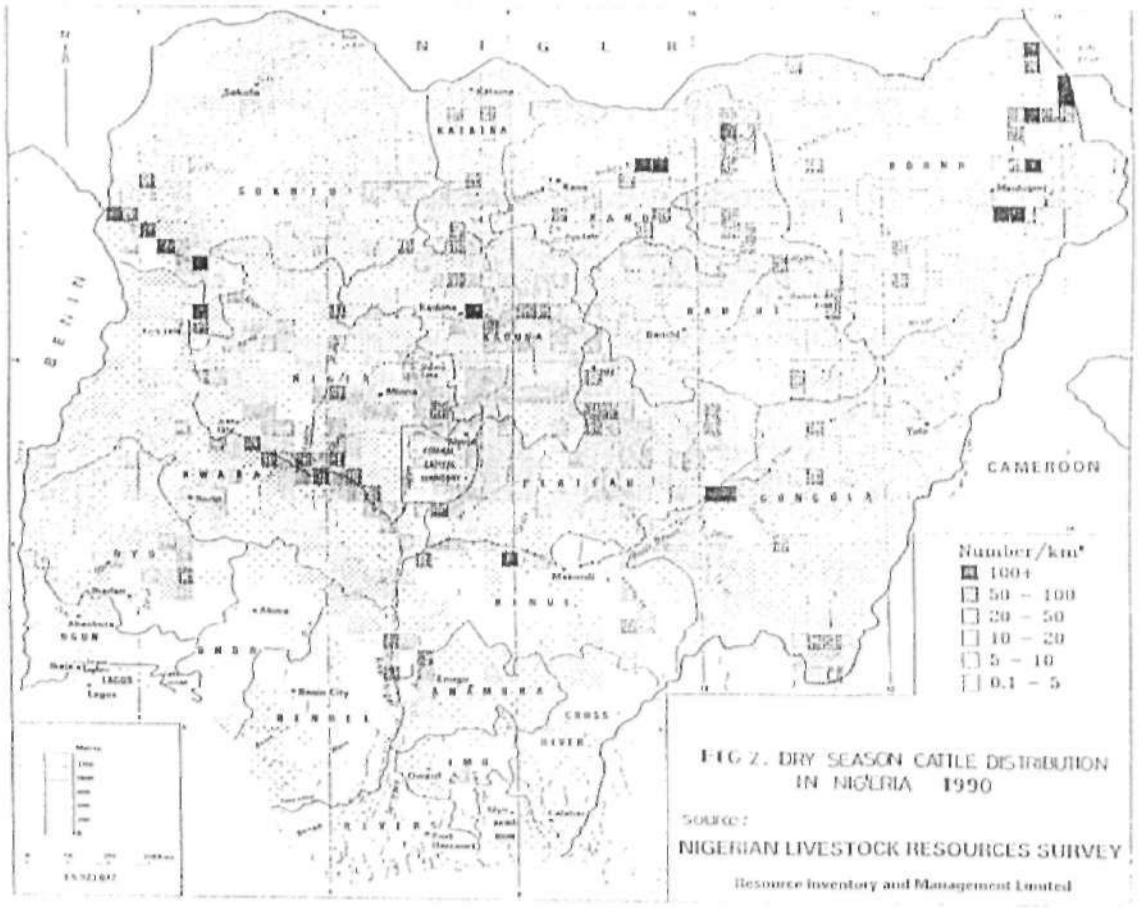


FIG 2. DRY SEASON CATTLE DISTRIBUTION IN NIGERIA 1990

SOURCE:
NIGERIAN LIVESTOCK RESOURCES SURVEY
Resource Inventory and Management Limited

2.2 CATTLE HUSBANDRY IN NIGERIA

The Funali pastoralists are traditionally the owners of approximately three quarters of the cattle resident in the northern States of Nigeria (Aliu, 1975). It is claimed that they once practised sedentary cattle husbandry system in the northern States as their home - base (Omoogun, 1987). However, increase in cattle numbers, decrease in grazing space and quality, long dry seasons and drought in the semi-arid zone, increased threats of tsetse and bovine trypanosomosis and other cattle diseases in the subhumid zone during the rainy season brought about the adoption of seasonal cattle migrations (Fig.3) between the North and the middle belt of Nigeria (Aliu, 1975; Bourn et al., 1986). The nomadic or transhumant cattle husbandry system had been practised until the 1950s when modifications began to be introduced (Fricke,1979; Putt et al., 1980) These changes may partly be associated with observed bovine trypanosomosis outbreaks in tsetse-free north following the introduction of infected animals from tsetse-fly infested south during the rainy season (Aliu, 1975). It is estimated that approximately 12-30% of the cattle ppulation in Nigeria are currently managed under the nomadic pastoral system (van Raay, 1975; Adamu, 1992).

Results of studies conducted by Fricke (1979), Putt et al. (1980); Hursey and Slingenbergh (1995) and Jabbah et al ; (1995) show that there have been marked Southward drift of cattle herds from the far North to the subhumid zone of Nigeria for permanent settlement. The increase in cattle population in this area from 2.8million (Jahnke, 1982) to 4.5milliom (Bourn and Milligan, 1983) within one year suggests the rapid rate at which Fulani pastoralists are abandoning the nomadic husbandry system. The choice of the subhumid zone for raising cattle has been encouraged partly by the declining endemicity of the morsitans tsetse and bovine trypanosomosis (Bourn et al., 1986; Omoogun et al., 1991; James, 1997). Other factors are the provision of grazing reserves and veterinary services by government (Buwai, 1992; Ojo, 1992) and the availability of farm land for the Fulani cattle herd owners provided by local communities (Bayer, 1986).

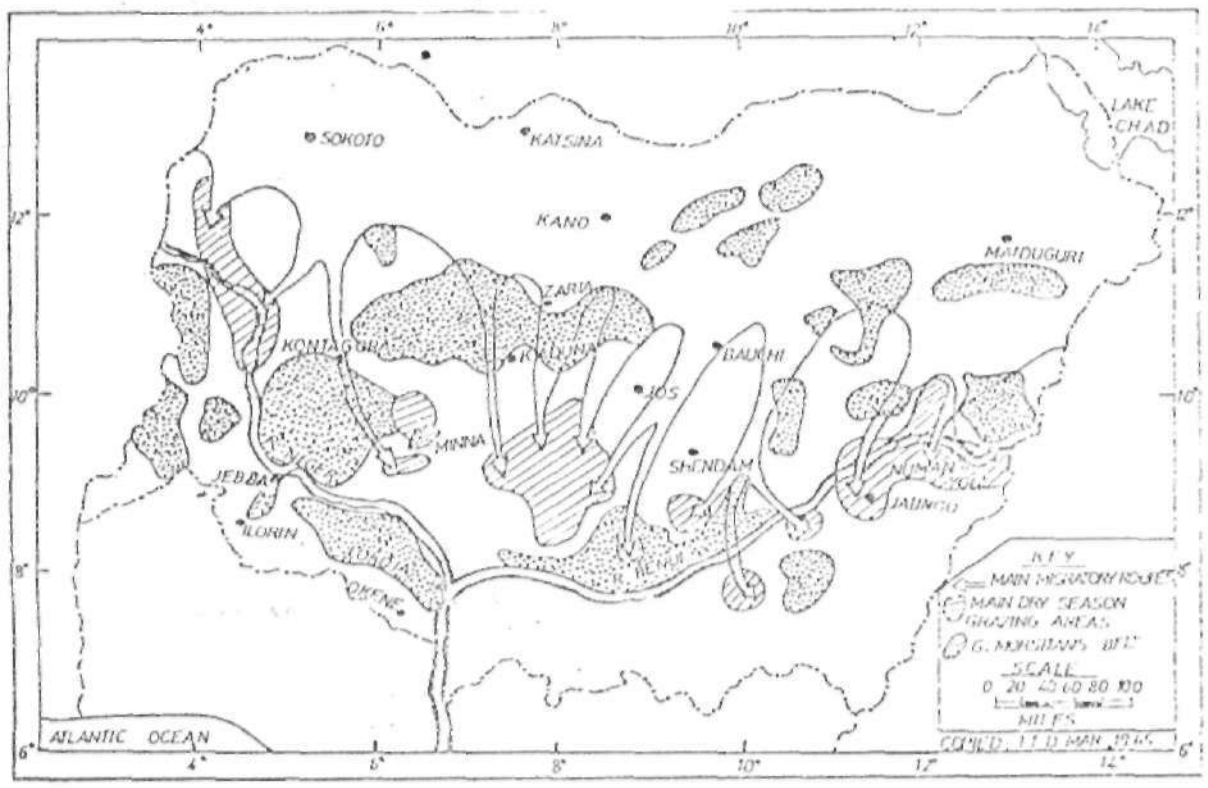


FIG 3 DRY SEASON FULANI TRANSHUMANCE IN THE NORTHERN STATES OF NIGERIA (source ALIU 1975)

2.3 AN OVERVIEW OF BOVINE TRYPANOSOMOSIS IN NIGERIA

Trypanosomosis has for long been identified as the main obstacle to the growth of the cattle population and productivities in Nigeria (Ikomi, 1981). Results of surveys in the recent past reveal that bovine trypanosomosis is still the most economically important and the most widespread cattle disease in Nigeria (Ilemobade et al., 1991; Anosa et al., 1992; 1995). Although annual losses due to cattle deaths and reduced animal products have not been accurately assessed in Nigeria, it has been generally believed that cattle populations and meat and milk products would double if tsetse and bovine trypanosmosis could be controlled (Griffin, 1978; Ikomi, 1981).

2.3.1 Vectors of bovine trypanosomosis in Nigeria.

The Glossina, commonly known as the tsetse fly, is the primary and the only biological vector of bovine trypanosomosis in Nigeria (Bourn et al., 1986). Survey results compiled by Bourn et al., (1986) show that two each of the Savannah, riverine and the forest tsetse species have been found in Nigeria. However, Omoogun (1987) stated that only two species each of the savannah and riverine tsetse are economically important vectors of bovine trypanosomosis in Nigeria. The distribution and abundance of tsetse flies in Nigeria are primarily determined by availability of suitable habitats and host (Omoogun et al.; 1991). Nord (1982) showed that the Savanna tsetse occurs in the northern, central and southern portions of the subhumid zone. This author, however, observed that the riverine tsetse species are distributed throughout the subhumid zone, while Omoogun (1987) reported their presence in the Southern (humid) parts of Nigeria also. Survey findings by Joshua (1986), Agu et al. (1989), Joshua and Shanthikutmar (1989) and Kalu et al. (1991) suggest that only the riverine tsetse can be detected in the subhumid zone of Nigeria currently.

Other haemophagous flies incriminated in the mechanical transmission of nagana and sura among cattle and camels respectively have been mostly observed in the semi-arid zone of Nigeria (Nawathe et al., 1988; Falope, 1991; Nawathe et al 1994). These flies belong to the genera Stomoxys (stable flies), Tabanus and Haemotopota (both commonly called horse flies) and Hippobosca (louse flies) (Itard, 1989)

2.3.1.1 The Savanna tsetse

Glossina m. submorsitans and G. longipalpis, are adapted to the climatic conditions of the Savanna woodland. This is why they inhabit the Guinea Savanna, referred to as the subhumid zone (Ford 1971). Although this ecological zone is the most suitable for livestock development, it was previously occupied by these Glossina species (Nash, 1969; Ford, 1971). Previously considered the most abundant and the most efficient in trypanosomosis transmission (Jordan, 1986: 1987), G.m. submorsitans, and G.longipalpis have been associated with major human and animal trypanosomosis outbreaks, thus making livestock development virtually impossible in the subhumid zone of Nigeria (Nash, 1969; Duggan,1970; Ford, 1971; Jordan, 1971). Davies (1977) observed that G.m. submorsitans is the greatest transmiter of animal trypanosomosis in Nigeria because of its high infection rates and abundance. Even when present in few or undetectable numbers, its transmission activities can give rise to severe trypanosomosis among susceptible animals (Jordan, 1961; Leeflang, 1975). However, the abundance and distribution of the Savanna tsetse began to be localized in the early 1980s into series of discontinuous belts and foci in the course of time (Nord, 1982). This is due to increase in human and animal population density in the subhumid zone in the last one and half decades resulting in accelerated clearance of the Savanna woodland which served as tsetse habitat. These cleared areas were used for agricultural activities, road and housing estate developments (Hadis et al., 1995). Similarly the combined effects of large scale ground and aerial glocinicide sprayings (Bature, 1981), and the mass destruction of their main feeding hosts, namely wildlife by hunters, and cattle by rinderpest (Bourn et al., 1986; Jordan, 1987; Omoogun et al. 1991) led to the decline in the population of Savanna tsetse (Bourn, 1983) and their complete extinction in some localities (Putt et al., 1980, Jordan, 1989). This probably explains why recent surveys carried out by Agu et al. (1989) in Zango Kataf and Jema'a LGAs of Kaduna State and on the Jos Plateau, by Kalu et al. (1991) in Gboko of Benue State, by Omoogun et al. (1991) in Egbe of Kwara State, and by Kalu (1995, 1996a, 1996b) on the Jos Plateau, all of which are within the subhumid zone of Nigeria, could not locate any Savanna tsetse.

2.3.1.2 The riverine tsetse

Glossina p. palpalis and G. tachinoides are the two most important riverine tsetse species and the most widespread throughout Nigeria (Bourn et al., 1986; Omoogun, 1989). Previous reports by Nash and Page (1953), Page and McDonald (1959) and Page (1959) suggest that the river banks are the main habitats of G.p. palpalis in the northern Nigerian subhumid zone, where the human hosts regularly visit for water. However, Killick - Kendrick and Godfrey (1963) observed that the habitat of G. tachinoides in the southern states or the humid zone is peridomestic since its feeding preference is for domestic and for wild animals. Also, recent observations by Bourn et al. (1986) and Laveissier et al. (1990) indicate that the habitat of the riverine tsetse is not strictly riverine. These authors reported that the typical habitat of these flies is any gallery forest with a high canopy cover, well sheltered thickets and semi-deciduous woodlands, while man, and his domestic animals provide the main sources of blood meals to these insects. While human activities resulting in the modification of the vegetation cover of the habitats of the woodland tsetse have virtually exterminated these insects, the riverine tsetse flies have continued to persist because their habitats have not been significantly so affected. (Bourn et al., 1986). Though they may be less efficient in transmitting animal trypanosomes than the morsitans subspecies, riverine tsetse are the main vectors of human and bovine trypanosomosis, especially in the subhumid zone, and in the absence of the savanna woodland tsetse (Bourn et al., 1986; Omoogun, 1987).

Various population patterns for G.p. palpalis and G. tachinoides have been reported by many workers in many parts of subhumid zone of Nigeria. Davies (1958) and Omoogun (1987) observed that the population of these flies were greatest in the early to mid dry season and least in the late rainy season in Gboko and Egbe respectively. On the other hand Nash and Page (1953) and Kernaghan (1962) observed an increase followed by a decrease in the populations of these flies during the raining and dry seasons respectively in Kaduna and in Niger states, all of which are locations in the subhumid zone of Nigeria (Fig.4). However, the populations of these flies in the humid rain forest have been reported to be lower than those in the subhumid zone throughout the year, with no significant difference between seasons (Page, 1959) due to deforestation (Ekejindu et al.,

1992). The population dynamics of the riverine tsetse are said to be related principally to climatic and breeding sites factors such as atmospheric temperature and humidity, volume of rainfall and the moisture content of the development soils for the fly larvae and pupae (Omoogun, 1987).

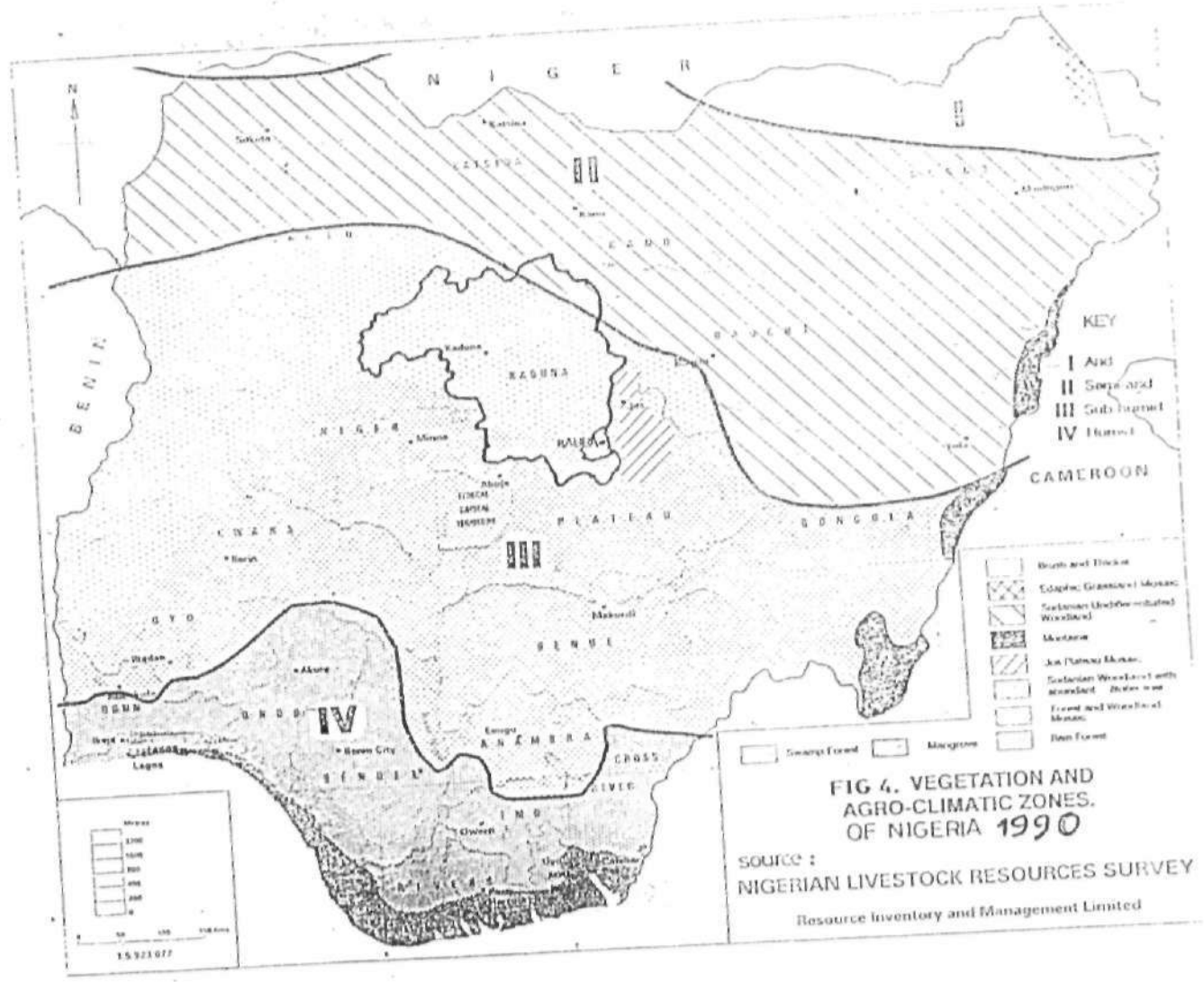


FIG 4. VEGETATION AND AGRO-CLIMATIC ZONES. OF NIGERIA 1990

SOURCE :
NIGERIAN LIVESTOCK RESOURCES SURVEY

Resource Inventory and Management Limited

2.3.1.3 The forest tsetse

Although twelve species of the forest tsetse are known, only seven are found in the West African forests. These are Glossina tabaniformis, G. haningtoni. G. nashi, G. nigrofusca, G.fusca, G.medicorum. and G. schwetsi (Langride et al., 1963: Ford, 1971). Out of these seven species only G. nashi and G. schwetsi have not been observed in the Nigerian forest zone (Moloo, 1985).

Most forest tsetse hardly have feeding contacts with domestic animals due to the inaccessibility of their habitat, while others may not even be attracted by the presence of mammalian hosts in their vicinity (Maclennan, 1981). Glossina fusca and G. medicorum have been reported to transmit trypanosomosis to cattle, causing serious disease (Jordan, 1962). A few other fusca tsetse species are known to be poor transmitters of trypanosomes, while some have been exterminated through the destruction of their habitats by human activities such as rapid deforestation resulting from logging and land cultivation (Wilson et al., 1972). Jordan (1987) therefore concluded that the forest tsetse may not be very important in animal trypanosomosis.

2.3.1.4 Mechanical Vectors

Mechanical vectors of bovine trypanosomosis are mainly dipterous haematophagous flies belonging to the families Tabanidae, Stomoxyidae and Hippoboscidae (Itard, 1989). The important Tabanidae involved in the mecahnical transmission of trypanosomosis belong to the genera Chrysops, Haematopota and Tabanus. The Stomoxyidae and Hippoboscidae acting as mechanical vectors of trypanosomosis are represented by the genera Stomoxys and Hoppibosca respectively (Wilson and Steven, 1989;Mattausch, 1990). These flies, which mostly inhabit the subhumid zone of Africa (Fassi-Febri, 1987), are ubiquitous during the rainy season while their numbers, which are smaller during the dry season, are restricted to river and stream valleys, and cattle, camel and horse sheds (Diall et al., 1988; Itard, 1989).

While only the females of the tabanid flies are haematophagous, both sexes among the stomoxids and the hippoboscids are blood suckers and can serve as mechanical transmitters (Itard, 1989). Although Trypanosoma evansi and T vivax are most adapted

to and are most commonly transmitted mechanically (Wilson and Stevenson, 1989), mechanical transmission of T.congolense and T. brucei by tabanids, stomoxids and hippoboscids have also been reported in cattle and sheep (Omara - Opyere, 1986: Mattausch, 1990). These flies may either spread trypanosomes in herds where infection has been established in a few animals by tsetse (Diall et al., 1988, Itard. 1989) or may be the sole transmitters of trypanosomes in areas where tsetse flies have been eradicated (Omara - Opyere 1986:, Dirie et al., 1989; Mattaussch, 1990). The frequent cases of T. evansi and T.vivax infections, inspite of the virtual eradication of savanna tsetse in Northern Nigeria (Bourn et al., 1986: Omoogun et al., 1991), may suggest that these biting flies are assuming a dominant role in the transmission of animal trypanosomosis (Jawonisi,1986; Nawathe et al., 1988; Opasina and Ekwuruke. 1988; Ilemobade et al., 1991).

2.3.2 Life cycle of tsetse flies.

The general life cycle of tsetse flies has been described in details by Chandler and Read (1961), Omoogun (1987) and Itard (1989) among others. In summary, reproduction of tsetse is dependent on availability of blood meal mainly required by the female, and atmospheric temperature. High atmospheric temperatures reduce interlarval periods while low temperatures increase them. Females usually mate at the age of 2-3 days while the males mate at the age of 7-8 days.

Following mating the female tsetse may produce larvae for the rest of her life. A third stage larva is laid by the female every 8-12 days. The mature, whitish, mobile maggot deposited in a shady spot by the female moves on the soil surface for up to one to two hours. Then it buries itself 1-2 centimeters below the surface of the soil that is dry, loose, rich in food and has a temperature range of 75^0 to 85^0 F to pupate immediately The development of an adult tsetse fly within the puparium to its emergence may take 17 days to 3 months depending on the temperature of the soil. High soil temperatures shorten the pupal stage while low soil temperatures lengthen it. It is also 2-3 days shorter for females than for males.

2.3.3 Life cycles of mechanical vectors of bovine trypanosomosis

The life cycles of all the mechanical vectors of bovine trypanosomosis described by Chandler and Read (1961) and Itard (1989) are similar except that of Hippobosca. These flies usually begin their life cycles with the laying of numerous eggs in batches on damp soil, rotting organic matter, rotting wood in tree or log holes, on leaves of marsh or water plants and in crevices of rocks along sides of streams. Stomoxys calcitrans lays batches of one hundred eggs at a time, while Tabanidae (Tabannus spp and Haematopota spp) lay several hundreds in shaped masses. Larvae are released from the hatched eggs of Stomoxys calcitrans after 2 to 3 days, while those of the Tabanus spp and Haematopota spp are normally hatched out of their eggs after 4 to 7 days.

The life cycle of Hippobosca is similar to that of tsetse flies. It begins with the laying of mature and well developed larvae on or near their hosts after every 7 or 8 days until a total of 12 to 15 have been born by each female fly. The larvae of Stomoxys calcitrans pupate after 15 to 18 days and adult flies emerge from these pupae after another period of 15 days. On the other hand, the larvae of the tabanids may develop into nymphs during a period of one year in tropical environments. These nymphs metamorphose into adult male and female flies after several days. Pupae which rapidly develop from the larvae laid by Hippobosca females metamorphose into adult flies after about 3 weeks.

2.3.4 Etiology of bovine trypanosomosis in Nigeria.

Survey results have revealed that Trypanosoma vivax is the most important etiologic agent of bovine trypanosomosis in the Sahel and the Sudan Savanna zones of Nigeria (Leeflang, 975; 1978; Magaji, 1981; Nawathe et al.; 1988; Opasina and Ekwuruke, 1988; Nawathe et al., 1994; Basu et al., 1995). On the other hand T vivax, T. brucei and. T. congolense are widespread in the Guinea and derived Savanna and forest zones of this country (Godfrey and Killick - Kendrick, 1961;1963; Godfrey et al.; 1964; Folkers and Jones-Davies, 1966; Omeke, 1994; Anene and Ezekwe, 1995; Kalejaiye et al., 1995; Kalu 1975). However, the predominance of T.vivax occassionally observed during recent bovine trypanosomosis surveys in Nsukka in the derived Savanna zone (Bourn et al.; 1986) and in parts of the Guinea Savanna like the Jos Plateau (Joshua 1986; Joshua and Shanthikutmar, 1989; Kalu

1995) in Kaduna and in Plateau States (Agu et al., 1989) and in Benue State (Kalu et al., 1991; Kalu, 1995) may support earlier assertions that this parasite is still the most important etiologic agent of bovine trypanosomosis in West Africa (Griffin, 1978; Losos, 1986). Although T. brucei is observed in cattle and small ruminants, its infection is reportedly characterized by low morbidity (Losos, 1986). Tsetse transmitted trypanosomosis of cattle in the field is frequently caused by multiple infections of T. congolense, T. vivax and T. brucei (Murray et al., 1981)

2.3.5 Nigerian Cattle breed susceptibility to animal trypanosomiasis.

Results of experimental and field studies have revealed that the pathogenic effects of bovine trypanosomosis are more severe in the infected Zebu cattle breeds (Bos indicus) than in the humpless West African dwarf (Bos taurus) cattle breeds (Roberts and Gray, 1973; Murray et al., 1981; Leperre Claxton,1994; Rege et al., 1994; Baker, 1995). However, even the trypanotolerant cattle breeds (Ndama, Muturu and Keteku) have not been spared from the severe pathogenic effects of trypanosomosis (Murray et al., 1983; Kalu, 1995; Taylor et al.,1995; Williams et al., 1996). Large numbers of supposedly trypanotolerant Ndama Cattle (Bos taurus) imported from the republic of Guinea acquired severe trypanosomosis and died when they were exposed to primary tsetse challenge in endemic areas of Benue, Edo and Delta States of Nigeria (Godfrey et al., 1964; Ikomi, 1981). It has also been reported that nutritional stress predisposes even trypanotolerant cattle to severe trypanosomes infection (Igbokwe, 1975; Murray et al., 1983;). However, Zebu cattle and calves, especially those resident in tsetse enzootic areas may tolerate low to moderate tsetse and trypanosomosis challenge (Losos, 1986; Uzoigwe, 1986; Leak et al., 1994 Mwangi et al., 1995; Rowlands et al., 1995)

2.3.6 Trypanosome species virulence in cattle.

It is well known that Trypanosoma congolense, T.vivax and T. brucei are the most economically important etiological agents of bovine trypanosomosis (Losos 1986). Although T. congolense is morphologically the smallest, it is the most virulent animal trypanosome to susceptible cattle (Boyt, 1986a), causing acute, subacute and chronic

disease syndromes (Itard, 1989). Losos (1986) reported more severe bovine trypanosomosis syndromes in East than in West Africa, due to T. congolense infections, terminating in cattle deaths four to six weeks post infection (p.i.).

On the other hand, T.vivax being the largest and the most active of the three economically important agents of bovine trypanosomosis has for long been considered to be more virulent to susceptible cattle in West Africa than in East Africa (Itard,1989). Generally, the course of uncomplicated T.vivax infections in cattle is longer and milder than that of T. congolense, terminating in death in untreated cases after many months (Boyt, 1986a) Although fulminating form of bovine trypanosomosis caused by T.vivax infection has been reported in East Africa (Hudson, 1944; Boyt and Makenzie, 1970), it is less frequent than that of T. congolense in this region and in Central Africa (Boyt, 1986a). It is commonly believed that T.brucei does not express severe clinical manifestations in cattle and therefore not economically important in bovine trypanosomosis (Boyt, 1986a; Itard, 1989).

2.3.7 *Life cycle of T.vivax, T. congolense and T.brucei.*

2.3.7.1 Vectoral development.

The life cycles of the three important trypanosome species (T.vivax, T.congolense and T.brucei) in bovine trypanosomosis have been described by Boyt (1986a) Itard (1989)and Vickerman (1997). In summary, when ingested with a blood meal by a tsetse fly, Trypanosoma vivax begins and completes the vectoral part of its cyclical development in the fly's proboscis. The life cycle of this animal trypanosome species is of short duration and depends on the atmospheric temperature. For example at 29^0c. T.vivax completes its development in the proboscis of Glossina palpalis after five days. However, the infective metatrypanosomes of T.vivax are fully developed after 13 days at 22^0c. On the other hand, T. congolense ingested with a mammalian blood meal by tsetse begins its vectoral development in the endoperitrophic space of the midgut. In this location T.congolense transforms into elongated trypomastigotes which multiply to considerable numbers 10 days after its ingestion with the blood meal by tsetse. This developmental form of T. congolense gradually moves towards the posterior of the peritrophic membrane

and invades the ectoperitrophic space of the midgut. From here these organisms move anteriorly and invade the proventriculus around the 20th day after the ingestion of the T. congolense infective form with a blood meal by tsetse. The trypomastigotes of this trypanosome species become thinner and elongated in the proventriculus and proceed to the oesophagus and the proboscis where they attach to the walls of the labium. Here they transform into epimastigotes, penetrate the anterior end of the hypopharynx and finally become infective metatrypanosomes, resembling blood stream forms of T. congolense. The cyclical development of T. congolense in the tsetse vector takes between 7-53 days depending on the atmospheric temperatures.

Out of the three T.brucei developmental stages, only the short stumpy form undergoes complex development in the tsetse vector, lasting 12 to 60 days, depending on the atmospheric temperatures. For example at 30^0c, the development of T.brucei takes 12 days; at 15.5^0c it takes two months (60 days) for this trypanosome species to complete its vectoral development. The long slender form of T.brucei is not adapted to survival and therefore cannot develop in the tsetse vector. The intermediate form of T.brucei is a transforming stage from the long slender to the short stumpy form in the mammalian host (Vickerman, 1985). The short stumpy forms of T. brucei ingested by tsetse transform into procyclic forms in the fly's crop. These organisms move into the endoperitrophic space of the midgut and become trypomastigote after 48 hours. The trypomastigotes multiply actively and invade the ectoperitrophic space of the tsetse midgut after 10 days. They then move anteriorly and reach the proventriculus within 10 to 20 days, where they change into slender forms. These organisms penetrate the peritrophic membrane to reach the salivary glands through routes not fully understood. In the salivary glands the trypomastigotes of T.brucei transform into epimastigotes which multiply and change into short metatrypanosomes infective to susceptible mammalian hosts.

2.3.7.2 Development in the mammalian host.

When deposited at the site of the tsetse bite the metatrypanosomes undergo another period of multiplication and development in the dermal connective tissue of the skin (Vickerman, 1985, 1997; Boyt, 1986a). Subsequently the long slender infective

trypanosomes invade the draining lymphatics and then the blood stream (Vickerman, 1985; 1997). T. congolense and T.vivax have been described as a haematic group of trypanosomes (Losos and Ikede, 1972). However, the former gets attached to the endothelial cells of microvasculature of especially the brain and the heart (Losos, 1986), usually producing low parasitaemia in the jugular vein and other vessels (Kalu, 1983). On the other hand T.vivax is capable of leaving the circulation and invading solid tissues, especially the heart (Van den Ingh and De Neljs - Bakker, 1979). In addition to the plasma, T. brucei is principally a solid tissue parasite, especially the brain (Rachel et al., 1984; Whitelaw et al.; 1985). In the plasma T. brucei is found in long slender intermediate and short stumpy forms (Vickerman, 1986). Tsetse flies are infected when the infective forms of T.congolense, T.vivax and the short stumpy forms of T.brucei are ingested with a blood meal and the vectoral phase of the trypanosomes' life cycle are initiated again.

2.3.8. Pathogenesis of bovine trypanosomosis.

The course of uncomplicated bovine trypanosomosis can be acute, terminating in either death or self-cure, while the chronic syndrome either leads to asymptomatic carrier or death (Boyt, 1986; Losos, 1986). Trypanosoma congolense causes more severe and acute disease in cattle than T.vivax, while T.brucei infections are reportedly mild and chronic leading, in most cases, to asymptomatic carrier state (Ikede, 1981; Boyt, 1986; Losos, 1986).

2.3.8.1 Prepatent period.

A prepatent period of 7-21 days for tsetse transmitted bovine trypanosomosis has been reported by Boyt (1986a). Wilson et al. (1975b) observed a mean prepatent period of 15.4 days for 61 East African Zebu heifers exposed to tsetse transmitted single and mixed Trypanosoma vivax, T congolense and T. brucei infections. Similarly Murray et al.(1981) reported prepatent periods of 10-63 days for 10 Zebu cattle and 9-63 days for nine Ndama cattle with single and mixed infections of T.vivax, T. brucei and T.congolense transmitted through tsetse bites. However, results of experimental studies carried out by Saror et al. (1979) and Uzoigwe (1986) showed prepatent periods of 4-6 and 2-4 days,

respectively in cattle intravenously infected with T. vivax using needle and syringe. On the other hand, Kalu and Aina (1984) recorded a prepatent period of 7-9 days in T. congolense infected cattle which were intravenously injected with these parasites using needles and syringes also.

2.3.8.2 Parasitaemia

Fulminating parasitaemia is associated with acute and severe disease while low levels of parasitaemia characterize chronic bovine trypanosomosis (Losos, 1979; Murray and Morrison, 1979). Trypanosoma vivax infections usually produce high (approximately 10-100 fold) parasitaemia peaks than those of T. congolense while T. brucei rarely causes heavy parasitaemia (Maxie and Losos, 1979; Ikede, 1981; Boyt, 1986; Losos, 1986). Each parasitaemic peak observed during bovine trypanosomosis is always associated with fever, while body temperature decline is observed following the fall in trypanosome numbers in circulation (Ikede, 1986; Losos, 1986).

2.3.8.3 Acute bovine trypanosomosis

Clinical signs of this disease syndrome are usually fulminating. Bovine trypanosomosis caused by both Trypanosoma vivax and T congolense is characterized by high parasitaemic peak, fever, rapid loss of body weight and appetite, severe anaemia, recumbency, circulatory failure, pale mucous membranes and enlarged peripheral lymph nodes (Ikede, 1981). Extensive haemorrhages on the mucous membranes, bloody nasal and faecal discharges have been reported in T. vivax infections (Maxie et al., 1979; Losos, 1986). In T. brucei infections, acute bovine trypanosomosis is also characterized by subcutaneous oedema, ocular discharge and nervous signs (Ikede, 1981; Losos, 1986). The acute disease syndrome usually lasts for days or a few weeks and either terminates in death or development of chronic bovine trypanosomosis (Boyt, 1986a).

2.3.8.4 Chronic bovine trypanosomosis.

Chronic bovine trypanosomosis with or without the initial acute syndrome lasts for months and in some cases for years, terminating in either death, self cure or asymptomatic carrier state (Losos, 1986). Most of the clinical signs of the chronic syndrome are milder than those of the acute phase with severe emaciation as a distinctive characteristic (Ikede, 1981, Boyt, 1986a). The course of chronic bovine trypanosomosis in susceptible cattle described for T. congolense infection by Boyt (1986a) provides a suitable model for the study and understanding of the pathogenesis and pathology of this disease. Ten days following infection, there is initial rise in body temperature, heart and respiratory rates. Continuous deterioration in health, steady loss of body condition, a gradual withdrawal of the infected animal from the grazing herd, preferring to stand idly under shade with drooping ears progressively develop. Its hair coat gradually becomes dull, and the underlying bones become visible as the body water, fat and muscles under the skin become gradually depleted. Diarrhoea may develop, the eyes become sunken and the visible mucous membranes become pale. The advance loss of condition is accompanied by increasing body weakness, staggering movements and sternal recumbency. This terminal stage of the disease may last for weeks or months before the infected animal dies or becomes an asymptomatic carrier.

2.3.8.5 The immunologic response to bovine trypanosomosis.

The immune response of cattle to trypanosome infection is dependent on the species of trypanosome and the cattle breed involved (Losos, 1986). Trypanosoma vivax and T congolense infections which are mainly intravascular seem to provoke principally the humoral immune response involving more of IgM than IgG (Mwambu and Losos, 1978, Losos, 1986). On the other hand T brucei which is mostly found in mammalian fixed tissues seems to provoke greater cellular immune response than humoral (Morrison et al., 1978). Results of previous studies reveal that trypanotolerant Ndama Cattle breeds produce higher IgG levels which control trypanosome parasitaemia better than the IgM prodominantly produced by the trypanosusceptible Zebu breeds (Desowitz, 1959; Murray et al., 1983; Authie et al., 1993; Andrianavo et al., 1995). The IgG predominantly ilicited

by the trypanotolerant (Bos taurus) cattle is directed against diversities of trypanosome antigens (33KD), while the IGM predominantly produced by the trypanosusceptible (Bos indicus) cattle is directed only at the invariant trypanosome antigen (69KD) (Authie et al., 1993; Taylor et al., 1996; Williams et al., 1996)

2.39 Pathology of bovine trypanosomosis

2.3.9.1 The chancre

The chancre is an inflammatory skin reaction to the multiplication of metatrypanosomes at the site of their introduction during tsetse fly bite. It covers an area with a diameter of 3-5 centimeters (cm) of thickened skin (Boyt, 1986a). It develops best with T brucei and least with T vivax infections and it's more prominent in susceptible than in trypanotolerant cattle (Ikede, 1986).

2.3.9.2 Fever

Except in T. brucei infections, (Losos, 1986), bovine trypanosomosis caused by T vivax and T. congolense is usually characterized by a positive correlation between fever and parasitaemic peaks (Ikede, 1986; Losos, 1986). Pyrexia is probably induced by pharmacologically active substances from damaged tissues, trypanosome metabolites and breakdown products released into circulation during the infection (Ikede, 1986).

2.3.9.3 Anaemia

Anaemia is one of the most prominent pathological features of bovine trypanosomiasis (Boyt, 1986a; Ikede, 1986; Antia et al., 1995; Onah et al., 1996). Each bout of decline in haematologic parameters (Pcv,Hb, Rbc) is associated with a rise in parasitaemic wave (Ikede, 1987). The trypanotolerant Ndama Cattle generally suppress the parasitaemia and so develop less severe anaemia than the trypanosusceptible Zebu (Murray et al., 1983). Anaemia develops gradually or rapidly and is usually very severe in T. vivax and T congolense infections, while T. brucei infections develop gradual and mild anaemia (Ikede, 1981).

The anaemia in bovine trypanosomosis has been variously classified as macrocytic normochromic in the acute stage and normocytic normochromic, in some cases tending towards microcytic normochromic in chronic stage (Valli et al. 1978a; Saror, 1979;Anosa and Isoun, 1980; Ikede, 1981; Losos, 1986). Factors responsible for the development of anaemia in bovine trypanosomosis reviewed by Ikede (1981, 1986), Assoku (1979), Boyt (1986a), Losos (1986) and Andrianarivo et al., (1995) include:

(a) extensive erythrophagocytosis especially in the spleen, liver and lymph nodes;

(b) occasional intravascular haemolysis;

(c) ineffective bone marrow response especially in the chronic stage;

(d) haemodilution due to increased plasma volume, and

(e) trypanosome - derived free fatty acids.

2.3.9.4 Emaciation

Emaciation is usually associated with chronic bovine trypanosomosis. In acute cases the infected animal dies or recovers before severe muscular wastage begins (Grriffin and Allonby, 1979). Ikede (1986) suggested that factors contributing to emaciation include reduced food intake, impaired efficiency of feed conversion and higher catabolic rates. However, Boyt (1986a) disagrees with the view that starvation is one of the contributing factors of muscular wastage since a trypanosome infected animal may eat regularly even during an ensuing recumbency. He therefore argued that emaciation in chronic bovine trypanosomosis is likely to be caused by a combination of catabolic and atrophic degenerative changes affecting the muscular proteins.

2.3.9.5 Reproductive disorders

Reproductive disorders associated with chronic bovine trypanosomosis have been reviewed by Sekoni (1994). Broadly, these reproductive disorders include degeneration of the hypothalamus, pituitary glands and gonads leading to the disruption in the secretions and plasma concentrations of the normal hormones necessary for reproductive processes in both sexes. In bulls, reproductive disorders associated with bovine trypanosomiasis include delayed puberty, loss of libido and severe degenerative changes of the genitalia,

manifested by the production of very poor semen quality or the cessation of its production leading to sterility. (Boly et al., 1993). On the other hand, bovine trypanosomosis in cows causes severe genital lesions, irregular oestrous cycles and temporary or permanent anoestrous. Transplacental foetal trypanosome infections in cows may be responsible for death during pregnancy, abnormal pregnancy, dystocia, abortion, premature, births, low birth weights, still births and neonatal deaths among other problems (Ogwu and Nuru, 1981; Ogwu, 1983; Ogwu et al., 1985; Akinbamijo and Reynolds, 1994; Okech et al., 1996a; 1996b; Jeffcoate and Holmes 1997).

2.3.9.6 Cardiovascular Changes

Nonsuppurative carditis usually occurs in T brucei infections (Ikede, 1981). However, in T.vivax and T.congolense infections varying degrees of oedema and muscular degeneration of the heart wall muscles occur (Valli et al., 1996b). Hyperpericardium and atrophy of the pericardial fat in subacute and chronic cases and petechial hemorrhages on the heart surfaces of acute cases were observed by Maikaje et al. (1991) during the postmortem of T. vivax infected udah rams. These pathological changes may be responsible for the enlarged and flabby appearance, weakened blood pumping actions and circulatory disturbances characteristic of the heart of trypanosomiasis infected ruminants earlier reported by Boyt (1986a).

2.3.9.7 Immunosupression in bovine trypanosomosis

An indication of immunosupression was first reported in Trypanosoma congolense infected cattle by Hornby (1921). He observed that these animals could not thrive in addition to losing condition and resistance to intercurrent infectious diseases. Fiennes (1954) later observed bronchopneumonia while complications of anaplasmosis, babesiosis and salmonellosis were subsequently reported in T.vivax and T. congolense infected cattle (Losos, 1975; Maxie et al., 1979). Immunosupression in bovine trypanosomosis is grossly evidenced by decreased secondary immune response to bacterial and viral vaccinations (Holmes et al., 1974; Scott et al., 1977; Rurangirwa et al., 1978; Whitelaw et al., 1979; Twinamasiko and Kakaire, 1995).

Studies on lymphoid changes associated with immunosupressions were carried out by many workers in order to elucidate the factors and the mechanisms involved. Morrison and Murray (1979) experimentally proved that there was no morphological evidence of immune system dysfunction during the first 3 - 4 months post infection in T congolense and T brucei infected cattle. However, examination of the lymphoid organs of cattle with chronic trypanoisomosis by these workers showed evidences of depletion. The lymphoides of these animals were small, fibrotic and relatively acellular. The paracortical areas were reduced and the lymphatic follicles were small and inactive.

A number of theories have been proposed to explain the mechanism of immunosupression in bovine trypanosomosis. These include:-

(i) Polyclonal B cell activation and exhaustion (Esuruoso, 1976; Assoku and Tizard. 1978).

(ii) Hypocomplementaemia and activation of both classical and alternate pathways of complement (Kobayashi and Tizard, 1976; Tabel et al., 1977; Nielsen et al., 1978; Rurangirwa et al., 1979).

(iii) Release of large quantities of free fatty acids by trypanosomes (Tizard et al., 1977; Assoku, 1979).

(iv) Functional defect in the macrophage population (Eardley and Jayawardena, 1977).

(v) Increased level or inhibition of suppressor T. cells (Jayawardena and Waksman, 1977).

(vi) production of soluble suppressor factor (Moulton and Coleman, 1979).

Recent investigations on the possible mechanism of immunosuppression in bovine trypanosomiasis carried out by Sileghem and Flynn (1992), Flynn and Sileghem (1993) and Flynn et al., (1994) revealed that the expression of the α-chain of interleukin 2 - receptor α- chain (IL2Rα) required to activate the proliferation of $\gamma\delta$T cells in the lymph nodes of T. congolense infected cattle was impaired by macrophage-like suppressor cells. The restoration of the immune system of an infected animal following successful trypanocide (Berenil®) treatment (Berger and Fairlamb, 1992) suggests that trypanosome derived factors identified to be cytokines (tumour necrosis factor and gamma interferon) produce the functional defects in some macrophage populations which are transformed

into suppressor-like cells as earlier postulated by Eardley and Jayawardena (1977), Lucas et al., (1993) and Darji et al., (1996).

2.3.10 The socio-economic importance of bovine trypanosomosis.

A comprehensive quantification of the economic importance of bovine trypanosomosis in Nigeria has not been accurately documented. This is mainly due to the paucity of reliable data on the accurate distribution of cattle and the insect vectors and insufficient knowledge of the effects of the disease on cattle production (ILRAD, 1993). These difficulties still exist mainly because approximately 90% of the cattle population in Nigeria are raised under the nomadic husbandry system which involves uncontrolled herd movements. However, the migrational cattle husbandry practices were evolved by the Fulani herders principally to avoid the devastating effects of bovine trypanosomosis, as well as seasonal lack of pasture and water. Because of the constant threats of trypanosomosis, cattle grazing especially during the rainy season, is restricted to areas of less tsetse and trypanosomosis challenges (Ikomi, 1981). Consequently, besides the pathological effects of the disease, the inability of the cattle populations in Nigeria to utilize the abundant fodder found in tsetse endemic foci has made it impossible to realize the full potentials of these animals. (Jabbah et al., 1995; Slingenbergh, 1995).

Finelle (1974) summarized the direct socio-economic consequences of bovine trypanosomosis. These include, cattle mortality which Ilemobade et al., (1991) estimated between 1-10%, emaciation, retarded growth, abortion, sterility, high cost of trypanosomosis diagnosis and trypanocides treatment. However, the indirect socioeconomic effects of bovine trypanosomosis documented by Connor (1994) include shortage of meat and milk for human consumption and lack of draught animals for increased agricultural output. Others are, the government limitation placed on the importation of highly productive exotic cattle breeds for upgrading the local ones through cross-breeding, overgrazing of tsetse free areas and the refusal of Fulani nomads to integrate themselves in national development due to their transhumant movements (Bourn et al., 1986).

2.3.11 Control of bovine trypanosomosis

2.3.11.1 Vector control

Since tsetse flies are the only biological and primary vectors of bovine trypanosomosis (Bourn et al., 1986; Itard, 1989; Odhiambo, 1995), control methods for these vectors are most abundantly documented in the literature.

2.3.11.1.1 Deforestation and land Utilization.

Tsetse flies concentrate in certain microclimates with characteristic temperatures and humidities determined by the respective vegetation covers (Finelle, 1974; Laveissiere et al., 1990). Nash (1941; 1948) and Bourn et al., (1986) reported various levels of success in controlling tsetse population in human and animal trypanosomosis endemic subhumid zone of Nigeria through selection or total deforestation, bush burning and clearing and land utilization (Ologe et al., 1981). Although the destruction of the Savannah woodland through this method seemed to have successfully eradicated the Morsitans tsetse (Omoogun, 1987) the Palpalis group of tsetse have persisted due to the inability of man to successfully modify their habitats (Laveissere et al., 1990). However, the main limitations of deforestation as a method of tsetse control are the high costs involved and the destruction of the environment leading to the erosion of exposed land surface (ILRAD, 1993). The method of tsetse control has therefore been abandoned.

2.3.11.1.2 Wild Life elimination

The control of mainly savannah tsetse in Eastern and Southern Africa (Finelle. 1974; Molyneux and Ashford, 1983) and in the subhumid zone of Nigeria (Bourn et al., 1986) has partly been attributed to the elimination of their wild life hosts such as warthogs (Phacochoerus porcus), bush pigs (Potamochoerus porcus), buffaloes (Syncerus caffer), and the spotted hyena (Crocuta Crocuta) among others (Takken, 1988; Groofenhius and Olubayo, 1993). On the other hand, the riverine tsetse have persisted due to their abilities to adapt to changes in the availability of their regular feeding hosts. For example in the absence of their regular mammalian hosts, G. tachinoides feeds on snakes and monitor lizards (Laveissire et el., 1990) while G. palpalis palpalis feeds on domestic pigs

(Molyneux and Ashford, 1983). However, the main difficulty with this method of tsetse control is the increasing concern for wild life preservation rather than destruction.

2.3.11.1.3 Glocinicides application

Direct control of tsetse flies in Nigeria has largely been achieved through the use of glocinicides which was started in 1955 (Davies 1964). By 1981 approximately 210,000km of tsetse infested land in Nigeria had been reclaimed through aerial and ground spraying of dieldrin, endosulfan and dicophane (DDT) (Bature, 1981; Dimlong, 1981; Omoogun, 1987). Chemical control of tsetse flies has largely been replaced by environmentally safer, simpler and economically cheaper methods including the use of tsetse traps and the sterile male insect technique.

2.3.11.1.4 Sterile male insect technique

The Biological control of tsetse (BICOT) was inaugurated in Nigeria as a joint project between the International Atomic Energy Agency (IAEA) and the Federal Ministry of Agriculture in 1980 (Samiento and Jawonisi, 1981). This was a supplementary control measure aimed at mopping up tsetse that survive chemical attack (Biwi, 1992). This control technique known as sterile male insect technique (SIT) involved the field release of large numbers of laboratory-reared tsetse fly males sterilized by irradiation (Offori, 1993). The life cycle of tsetse flies is disrupted through the inability of female tsetse to produce larvae after mating with the released sterile males.

2.3.11.1.5 Use of traps

Currently large scale control of tsetse flies is being effectively achieved in such endemic countries as Congo (Noireau et al., 1990) Cote d'Ivoire (Laveissire et al., 1990) Zaire (Mansinga et al., 1991) and Burkina Faso (Odhiambo, 1995) through the use of both attractant-impregnated and plain traps. The use of such traps in Nigeria is still at the experimental level.

2.3.12 Parasite Control

2.3.12.1 Trypanosome detection

One of the main aspects of African trypanosomosis control that has engaged research scientists is the evolution of accurate, simple and inexpensive trypanosome detection techniques in mammalian blood. Direct microscopic examination of blood either by the wet film method or as giemsa stained thick and thin smears described by Lumbsden et al. (1973) and Boyt (1986a) is the easiest (Nantulya, 1990). A drop of blood from a suspected animal placed on a microscopic slide with a covership placed on it is examined directly under the light microscope using the X40 objective. This method facilitates the rapid screening of large numbers of blood samples based based on the identification of trypanosome species using mainly motility characteristics.

On the other hand, a thin smear is prepared by drawing forward at an angle of 45^0 one edge of a clean slide touching a drop of blood on another clean slide. However, a thick smear is made by spreading a drop of blood on a clean slide in a circular area about 1.5cm in diameter, using one tip of the edge of another clean slide. Both blood preparations are air-dried, but only the thin blood smear is fixed with absolute methanol for 2 - 3 minutes. Finally both thin and thick blood films, are stained with giemsa for 30 minutes. After washing with water and drying, both stained blood smears are examined for trypanosomes under the microscope using the oil emersion objective (x 100 magnification). The morphology and degree of parasitaemia of trypanosomes can be determined using both thin and thick stained blood smears. The thick blood film is prepared for locating trypanosomes in subclinical cases with very low parasitaemia (Kalu et al., 1986). The direct microscopic examination of wet or giemsa stained thin and thick blood smears may not diagnose some subclinical cases with very low parasitaemia (Boyt, 1986a).

The sensitivity of the direct blood smear microscopy can be improved through the concentration of the trypanosomes by centrifugation as described by Woo (1967; 1970). Unclotted blood in a heparinized capillary tube with one end sealed with fire or cristoseal wax is spun in a haematocrit centrifuge (Hawksley Ltd., England) at 12,000g for 5 minutes. Trypanosomes concentrated at the buffy coat of the spun blood are either

extracted onto a slide or the buffy coat zone in the capillary tube is examined directly under the microscope using the X40 objective. This technique facilitates accurate determination of the presence and level of trypanosome parasitaemia even in an animal with subclinical infection (Murray et al., 1977). The limitation of the technique however, is that it requires the use of electricity which may not be available in the field. Alternatively, blood from suspected animals can be inoculated into susceptible animals, usually laboratory rodents. Blood from these inoculated rodents is microscopically examined regularly for not less than three weeks when trypanosomes are expected to be observed in positive cases. This technique is more sensitive than the direct blood microscopy (Paris et al., 1982; Kalu et al., 1986). However, diagnosis of trypanosomosis by this technique is not rapid and is expensive (Nantulya, 1990). Besides, Trypanosoma vivax cases may be missed since this trypanosome is not infective to laboratory rodents, except the rodent adapted strains of Y58, Y486, V953 (Leeflang et al., 1976).

Red blood cells can also be removed from the test blood sample by the diethyl amino-ethyl (DEAE) anion exchange, chromatography technique (MAECT) described by Lumbsden et al. (1979). However, the above techniques are too cumbersome and are therefore not used for routine parasitological diagnosis of animal trypanosomosis.

2.3.12.2 Antibody detection.

Although many antibody detection tests for the diagnosis of African animal trypanosomosis have been developed, only a few are simple, specific, sensitive, affordable and suitable for routine use (Nantulya, 1990). The introduction of the indirect flourescent antibody technique (FAT) by Gill (1964; 1966) for the diagnosis of Trypanosoma evansi infections marked significant improvements in the accurracy of immunodiagnosis of animal trypanosomosis especially in camels (Jaktar and Singh, 1971; Olaho and Wilson, 1981) and other raminants (Clarkson et al., 1971). The principle of this test described by Itard (1989) facilitates the in vitro contact between a characterized trypanosome fixed on a slide in a blood smear, serving as an antigen, and a test animal serum. If the animal donor of the test serum had recently been infected with a trypanosome sharing antiginic characteristics with one fixed on the slide, the elicited antibodies in the test serum bind to

the antigenic sites on the fixed trypanosomes. The anitbodies spcifically bound to the trypanosomes antigens on the slide are allowed to specifically react with fluorescein conjugated antigammaglobulins produced in the same animal specie as the donor of the test serum. This immune complex formed on the slide is observed in many fields under the ultraviolet (U.V) microscope as spots of yellow-green colour emited by the fluorescein conjugate. If the suspected animal is trypanosome-free no fluorescence will observed under the U.V microscope. This is because of the absence of trypanosome antibodies required to form the detectable immune complex. However, there are hindrances to the routine uses of this test for the diagnosis of animal trypanosomosis. These include the cumbersomeness of preparing large numbers of blood smears to provide a standardized antigen preparation, the stringent requirement of ultra-cold temperatures (-196^{0}F) for their storage and transportation and the substantial non-specific reactions provided by the prepared antigens (Nantulya, 1990). Some of these hurdles have been overcome to a large extent to facilitate the routine use of this test for accurate diagnosis of animal trypanosomosis. For example the trypanosome antigen in the blood smear is fixed in a mixture of acetone and formaldehyde solution and can be stored and transported conveniently (Katende et al., 1987). This improvement through antigen fixation facilitates a fairly accurate differentiation between infections due to different trypanosome species in ruminants.

A major breakthrough in the immunodiagnosis of animal trypanosomosis was achieved following the introduction of the enzyme - linked immunosorbent assay (ELISA) which was first used for the diagnosis of Trypanosoma rhodesiense in man by Voler et al. (1975). Subsequently Luckins et. al. (1978; 1979) used this test for diagnosing T. evansi infections in rabbits and camels. This test involves the in vitro reaction of antibodies against each trypamosome species in a test serum with the corresponding crude antigen lysate. The immune complex for each trypanosome species (T. vivax, T. congolense and T. brucei) is allowed to further react with its antiglobulin conjugated with the enzyme horse radish peroxidase (HRPO). The hydrolysis of hydrogen peroxide in a test solution for each trypamosome species indicated by a chromogen suggests a positive result.

Although the sensitivity and specificity of the antibody ELISA are comparable with those of the indirect fluorescent antibody technique (IFAT), the indices for the antibody ELISA cannot be standardized. This is because the quality of the crude trypanosome antigen lysate is ill defined (Nantulya, 1990). Recent improvements to the antibody ELISA for the diagnosis of animal trypanosomosis have been reported by Ijagbone et at. (1989). This observation indicates that a trypanosome antigen purified by fractionation can be used to improve the species specification of the antibody ELISA technique.
The general limitation of immunodiagnostic tests is their inability to indicate the stage of trypanosomosis (ILRAD, 1988).

2.3.12.3 Antigen detection

Unlike with immunodiagnostic tests, in vitro detection of trypanosome antigens in the serum of an animal indicates an active or current infection (Nantulya and Linqvist, 1989). Araujo (1982) made the first attempt to detect circulating antigens of Trypanosoma cruzi in order to diagnose chagas disease, using enzyme immunoassay. The low sensitivity observed by this author encouraged Rae and Luckins (1984) to develop a T. evansi antigen detection system, using polyclonal anti- T. evansi antibodies. The main limitation of T. evansi antigen dectection using polyclonal antibodies is its low specificity. To overcome the problem of cross-reactions with non-targeted trypanosome species antigen and possibly with the antigens of other parasites during screening with polyclonal antibodies, Nantulya et al. (1984; 1986; 1987) and Richardson et al. (1986) reported a breakthrough in the diagnosis of African trypanosomosis. They developed trypanosome species-specific monoclonal antibodies. These species-specific immunoglobulins were used to diagnose animal trypanosomosis using the sandwich enzyme-linked immunosorbent assay (ELISA). The principle of this assay technique is that the monoclonal antibodies against each trypanosome specie is used to capture its circulating antigen in a test serum. A second antibody which is enzyme - linked is introduced to bind to one of the many free combining sites on the captured trypanosome antigen. The immune complex formed from these reactions is revealed by the product of horse radish peroxidase hydrolysis of hydrogen peroxide detected by a chromogen. The same

monoclonal antibodies can be used for the diagnosis of animal trypanosomosis in several animal host species since host species specific anti-immunoglobulin reagents are not required (Nantulya et al., 1987). The antigen ELISA is highly sensitive, specific and economical. The best diagnostic effect of this test is most likely to be achieved through combining it with one of the most sensitive standard trypanosome detection methods (Nantulya, 1990).

2.3.13 Immunization

Research aimed at developing a vaccine to immunize animals against African trypanosomiasis has not yet yielded the desired results (Waghela et al., 1991; ILRI, 1996). This effort has been frustrated mainly by the phenomena of antigenic variation exhibited by the trypanosome parasite and immunosuppression induced in the mammalian host during infection (Holmes, 1980; Murray et al., 1991). The trypanosome surface coat which induces protective immunity in the mammalian host easily changes its antigenicity to frustrate recognition by the defence system of the infected animal (Doyle, 1977; Borst et al., 1996). It is estimated that successive generations of trypanosomes from a single organism may synthesize not less than 100,000 different surface coats (Van derPloeg et al., 1982 cited by Nantulya and Moloo, 1989). Consequently it is immpossible for a susceptible animal to acquire immunity to these variable antigenic types (VATS). Even if an infected animal spontaneousely recovers from trypanosomosis it develops immunity only to the trypanosome strain that infected it (Nantulya et al., 1984; 1986). Neither the nucleic acid hybridization studies carried out by Cross (1979) nor the immunological investigations results of Antia et al., (1996) and Miller et al., (1996) have fully explained the genetic basis or the possible mechanism of antigenic variation in trypanosomes. A further complication to the development of a vaccine against animal trypanosomosis is the occurrence of immunosuppression induced by the trypanosome. This phenomenon renders the infected susceptible animal unable to mount an immunological defence against trypanosomosis infection or to respond to vaccine challenge (Maxie et al., 1979; Rurangirwa et al., 1979; Twinamasiko and Kakaire, 1995). However, the

immunocompetence of trypanosome infected animals is restored after trypanocide treatment (Bevan, 1928; 1936; Wilson et al., 1975a; 1975b).

2.3.14 Trypanotolerance

Inspite of the problems of antigenic variation and immunosuppression, certain indigenous breeds of cattle (N'Dama, Muturu and Keteku) in West and Central Africa are able to survive and be productive without the aid of trypanocidal drug treatment in tsetse-fly infested areas (Murray et al., 1980; 1982; Agyemang et al., 1991; Dwinger et al., 1994; Baker, 1995,). Similarly, results of experimental studies carried out by Wellde et al. (1981), Uzoigwe (1986) and Trail et al. (1994) indicated that calves of trypanosusceptible Hereford and Zebu cattle between the ages of 4 and 12 months exhibited trypanotolerance. However, Desowitz (1959), Godfrey et al. (1964) Ikomi (1981) and Leak et al., (1994) reported that trypanotolerant Ndama and Muturu cattle breeds exposed to heavy primary trypanosome challenge died from as severe trypanosomosis as the trypanosusceptible Zebu breed. Therefore, it is evident that trypanotolerant cattle can only survive and remain productive in endemic areas under moderate levels of tsetse and trypanosomosis challenges (Barret, 1997). Results of experiemental studies reported by Murray et al. (1983) and Trail et al., (1991) also suggest that the resistance of trypanosusceptible cattle breeds to trypanosome infections can be improved through cross-breeding with trypanotolerant breeds (Willmut and Jere, 1991) and the continuos exposure of the former to low tsetse and trypanosome challenges.

2.3.15 Chemoprophylaxis and Chemotherapy

Chemoprophylaxis and chemotherapy have been widely used and heavily relied upon to minimize the effects of trypanosomosis in livestock in endemic areas (Aliu, 1981; Rushrigajiki et al., 1986; ILRAD, 1987; Peregrine, 1994; Aliu, 1997). This is because trypanosomosis vaccine development, complete vector eradication and the development of absolute trypanotolerance in cattle have been impossible inspite of many research efforts (Nantulya, 1990; Ilemobade et al., 1991; Anosa et al., 1992; ILRI, 1996). Unfortunately, only limited varieties of trypanocides have been in use for over 40 years during which only

melarsomine (cymelarsan®) was introduced into the market in 1985 (ILRAD, 1987;; 1990; Nantulya and Moloo, 1989; Raynaud et al., 1989; Murray et al., 1991; Aliu, 1997; Barret, 1997). These trypanocides include diminazene aceturate (Berenil ®; Hoechst AG. Frankfurt,Germany, homidium chloride (Novidium®; May and Baker, Degenham Ltd, England) and isometamidium chloride (Samorin ®; May and Baker, Degenham Ltd. England) (Ilemobade et al., 1975; Leeflang, 1978; Aliu, 1981: 1997; Arowolo, 1981; Ilemobade, et al., 1991). Approximately 25 million doses of these drugs are in use annually which cannot cover the estimated 50 million cattle, 30 million sheep and 40 million goats exposed to animal trypanosomiasis in the endemic region of Africa south of Sahara (Trail et al., 1985; ILRAD, 1987; Hursey and Slingenbergh, 1995; Shaib et al., 1997).

2.4 AN OVERVIEW OF COMMONLY USED TRYPANOCIDES IN NIGERIA

2..4.l Diminazene

This is an aromatic diamidine (diminazene diaceturate) manufactured and commonly marketed as Berenil® by HOECHST AG. Frankfurt, Germany. It is used for the treatment of Trypanosoma congolense. T.vivax. T.brucei. T.evansi. and T.simine infections in ruminants, dogs and pigs (Joshua and Kayit, 1984; Boyt, 1986b; Aliu, 1997). Diminazene binds to the trypanosome Kinetoplast DNA (kDNA) and inhibit its replication and thus inducing dyskinetoplasty (Newton, 1972: Newton and Le Page, 1967; Riou and Benard, 1980). The recommeded usage of Berenil® by the manufacturers (Hoechst, AG. Frankfurt, Germany) and by Boyt (1986b) is intramuscular injection of 3.5mg/kg or 7.0mg/kg of 7% freshly prepared solution in distilled water. Such doses as 5mg/kg and 7mg/kg are known to be curative for T. brucei and T. simiae in sheep respectively (Joshua and Kayit, 1984).

Results of recent studies done by Silayo and Marandu, (1991), Silayo et al. (1992) and Mamman et al., (1993) on the sensitivity of T.congolense to diminazene aceturate (Berenil®) showed that even strains of this parasite previously said to be resistant were eliminated in infected goats when treatment was administered at a dose of 7.2mg/kg 24 hours post infection and before the development of parasitemia. However, neither strain of T. congolense known to be sensitive or resistant to diaminazone®could be eliminated by this trypanocide in infected goats when treatment was administered after the appearance of parasitemia. It was concluded that the alteration in diminazene sensitivity by the strain of T. congolense used for this study could be associated with alteration in resistant phenotype of trypanosomes due to delay in treatment and increase in numbers of genetically distinct trypanosome phenotypes (Codjia et al., 1993).

2.4.2 Homidium

The phenanthridine trypanocides are manufactured as homidium bromide (Ethidium®) and homidium chloride (Novidium®) by FBC Limited, Cambridge, England, and May and Baker limited, Degenham, England, respectively. They are both used for

the treatment of T.vivax and T. congolense infections in ruminants, equidae and pigs (Mamman, 1993; Aliu, 1997).

These homidium salts effect their trypanocidal actions by intercalating between the adjacent base pairs of the double stranded helix of the nuclear DNA and inhibit its replication and the functioning of the single strands as templates for RNA synthesis (Newton, 1957; Aliu, 1981; Boyt, 1986b). Futhermore, these drugs bind to the kinetoplast DNA (kDNA) resulting in dyskinetoplastic trypanosomes (Steinert, 1969; Macadam and Williamson, 1974; Hajduk, 1978; Riou, et al., 1980). Homidium also selectively interact with ribosome membranes of trypansomes to prevent the reorganization of these organelles, necessary for cell division (Williamson et al., 1975). The normal architectural structure of the inner mitochondrial membrane is also modified by the the trypanocidal activities of homidium in the trypanosomes (Mahler et al., 1971; Azzim and Santato, 1971). Each of these phenanthridine salts is administered to trypanosome infected animals by intramuscular injection of 2.5% solution at a dosage of 1mg/kg (Boyt, 1986b). Although field and laboratory observations of Ethidium® and Novidium® resistance have been reported in the North-Central State (i.e. present Kaduna and Kastina States) where their use has been widespread (Ilemobade et al., 1975; Leeflang et al., 1978), the specific mechanism of resistance has not been documented in the literature.

2.4.3 Isometamidium

Isometamidium chloride which is a phenanthridine aromatic amidine marketed as Samorin® by May and Baker, Degenham Ltd, England. This trypanocide is being used as a curative and prophylactic drug against T. brucei, T. congolense and T. vivax infections in domestic livestock (Hawkings, 1963; Williamson. 1970; Leach and Roberts; 1981). Plasma trypanocidal levels of this drug are known to last between 1-6 months (Boyt, 1986b). Isometadium chloride produces lethal effects in the trypanosomes by displacing magnesium ions and polyamines from ribosomes. It also modifies cytoplasmic membranes and ribsomes (Macadam and Williamson, 1972). All these changes inhibit phospholipid

synthesis (Gutteridge, 1969), basic amino acid transport (Gutheride, 1966) and oxygen uptake (Hill and Hultner, 1968).

Samorin® is usually administered to trypanosome infected animals by deep intramuscular (IM) injection of 1% or 2% solution at the dose rate of 0.25-1mg/kg (Aliu, 1981; ILRAD, 1987; Mamman, 1993. Some strains of T. congolense resistant to this drug has been reported by Pinder and Authie (1984), Schoenefeld et al. (1986, 1987) and Codjia et al. (1993).

Due to inadequate veterinary supervision, widespread misuse of these trypanocides without regard to dose, route and regime of administration is common in Nigeria. Also the nomadic and transhumant husbandry systems practised in Nigeria expose the cattle to infection by trypanosome strains with various levels of sensitivity and resistance to commonly used trypanocides. It has also been reported by Leeflang (1978) and Williamson (1979) that continuous inadequate treatment of bovine tryanosomosis with the same or different trypanocides of the same chemical type for long periods is conducive to the emergence of resistant trypanosome strains to such misused drugs.

2.5 FACTORS OF DRUG RESISTANCE

The main host and parasite-related factors in drug resistance in trypanosomosis have been documented by Leeflang (1978) Williamson (979), Kaminsky et al. (1980); Sones and Holmes, (1992); Hirumi et al., (1993) and ILRAD in annual reports 1990). The host-related factors of drug resistance in trypanosomosis include the concentration of the drug used; poor distribution of the trypanocide in infected tissues or intracellular sites inaccessible to the drug; variation in drug metabolism between individuals, and diminished activity of a drug in a host with suppressed immune system. On the other hand, the parasite-related drug resistance factors in this disease are the size of initial trypanosome inoculum, alterations at the primary site of drug uptake: reduced accumulation or accelerated disposal of the drug in the parasite, and a change in the drug's enzyme target through an increase in its production levels as in the case of dihydrofolate reductase thimydilate synthetase (DHFR-TS) in leishmania resistance to methotrexate. Other parasite-related factors in tryapanosome drug-resiatance phenomenon include changes in

enzyme afinity for drug due to a single amino acid substitution in the parasite, increased metabolite production or retention as reported in T. brucei, and the use of alternative metabolic pathways by trypanosome to bypass the site of drug inhibition. Cross-resistance between trypanocides has also been observed in field and laboratory studies. For example, resistance to tartar emetic was induced most especially among trypanosome strains already resistant to aromatic arsenicals (York et al., 1932 cited by Williamson in 1979). It has also been observed that resistance to quinapyramine and melarsomine apparently induces cross-resistance to diminazene (Osman et al.; 1992; Ndoutamia et al. 1993).

CHAPTER III

THE EPIZOOTIOLOGY OF BOVINE TRYPANOSOMOSIS IN KAURA LOCAL GOVERNMENT AREA OF KADUNA STATE

CHAPTER III

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3.1 INTRODUCTION

Results of the following previous surveys indicated that the LGA's of Zangon Kataf and Jema'a, and part of Plateau State adjoining Kaura LGA which were previously free of tsetse flies and trypanosomosis are now endemic for both the vector and the disease. Joshua (1986), Joshua and Shanthikutmar (1989) and Kalu (1991) reported bovine trypanosomosis prevalence rates of 9.7%, 9.6% and 38.6%, respectively in Plateau State. Similarly, Agu et al (1989) reported prevalence rates of 9.4% and 8.2% in Zangon kataf and Jema'a LGAs respectively. The biological vectors of bovine trypanosomosis observed during all the above studies were Glossina tachinoides and G. palpalis palpalis while T.vivax had the highest cattle infection rates followed by T. congolense and T. brucei, respectively.

An outbreak of tsetse infestation in Kaura LGA. leading to the mass exodus of settled Fulani pastoralists from this area was reported in the Concord daily newspaper of 6th November, 1990 (volume 2265, page 20). Consequently this study was designed to:-

1. identify and assess the types and seasonal population dynamics of vectors of bovine trypanosomosis vector in Kaura LGA.
2. determine the prevalence of bovine trypanosomosis, identify and assess cattle infection rates by the various animal trypanosome species in this study area

3.2 MATERIALS AND METHODS

3.2.1 Study area.

Kaura is one of the 23 Local Government Areas (LGAs) situated in the South Eastern part of Kaduna State (fig.5). Geographically it is located within the subhumid zone of Nigeria lying between latitudes 8^0 50′ and 10^0 5′ north of the equator (fig.4). Administratively this local government area made up of the Kagoro and Moroa chiefdoms is further subdivided into 6 districts namely: Zankan, Manchok, Fadan Kagoro, Kukum, Kadarko and Bondong (fig.6). The topography of the Local Government Area ranges from 2350 to 3880 meters above sea level, the latter being the peaks of the Kagoro and Atakar hills where some cattle emigrate to during the rainy season.

The climate of Kaura Local Government Area is typical of the subhumid zone of Nigeria which is characterized by a dry season period between the months of November and February, and a rainy season period between the months of March and October (Mohammed Saleem, 1986: Omoogun, 1989). Annual rainfall ranges between 2000 and 25000mm, being heaviest between the months of August and September. The dry season is characterized by temperature and atmospheric humidity ranges of 21 - 24^0C and 54-69%, respectively, with the driest and coldest harmattan weather experienced during the months of December to February. However, in the rainy season, temperature range increases to 22 - 32^0c while atmospheric humidity rises to 60-85%.

The vegetation types found in Kaura Local Government during this study were the same as those described for Egbe in Kwara State (Omoogun, 1989) also located in the subhumid zone of Nigeria. River Kajim Valley (fig.7) is the only location in Kaura Local Government Area which has the same perrenial vegetation types described for rivers Ofe and Ofili by Omoogun (1989), and for the forest region (Laveiseiera et al., 1990), considered suitable habitats for riverine tsetse flies. The green vegetation observed in the open bush during the rainy season surveys are completely dry during the dry season. It was also observed that water in almost all the streams in Kaura Local Government Area dried off during the dry season except in Rivers Kaduna, Gurgel and Kajim.

Kaura Local Government Area has three semi-urban human settlements that serve both as administrative and commercial centres.

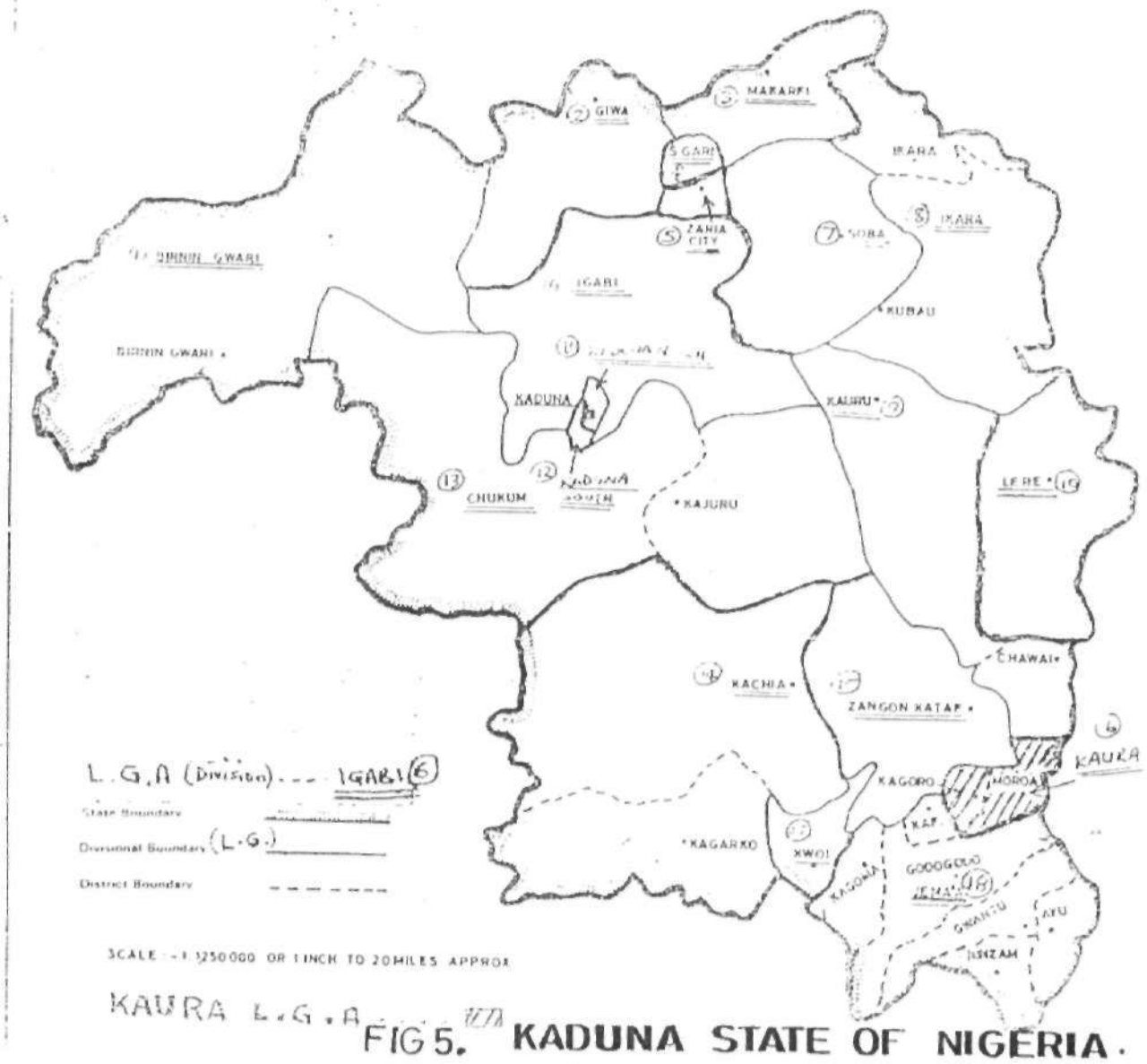
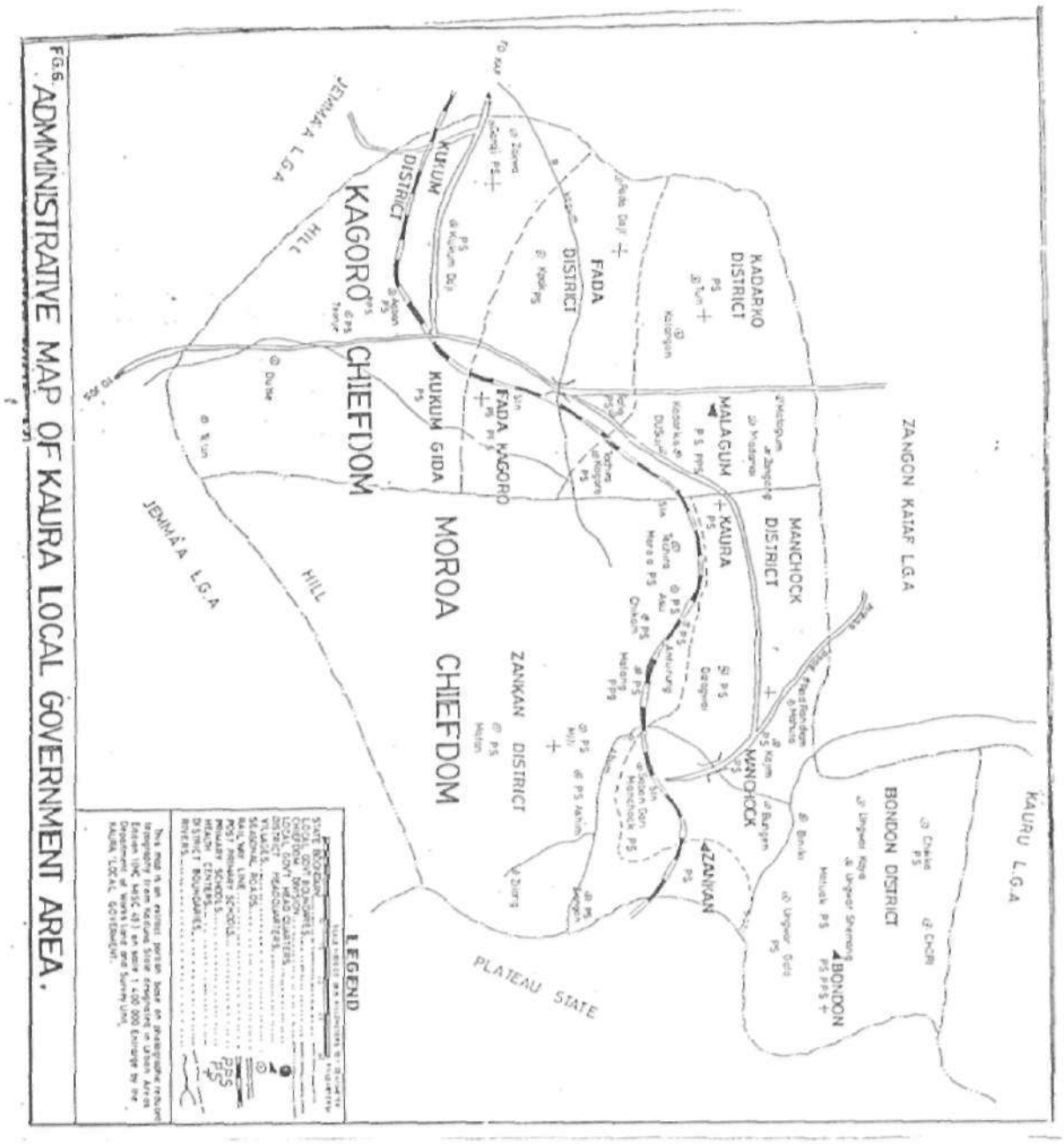


FIG 5. KADUNA STATE OF NIGERIA.



FG.6. ADMINISTRATIVE MAP OF KAURA LOCAL GOVERNMENT AREA.

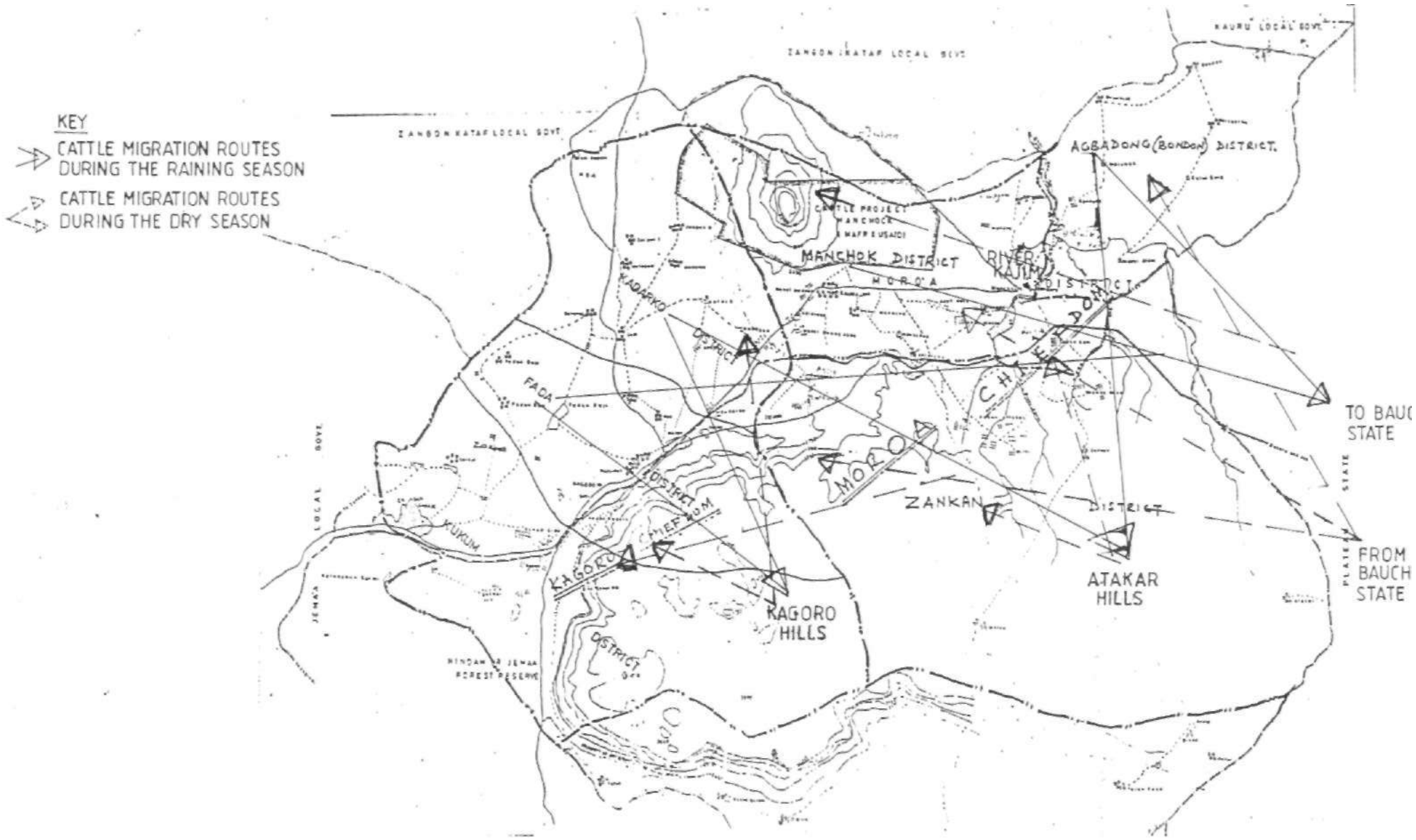


FIG 7 CATTLE SEASONAL MIGRATION PATTERNS IN KAURU LOCAL GOVERNMENT AREA

3.2.2 Experimental Procedure

Surveys for bovine trypanosomes and their vectors were done on district basis during the dry and rainy season months of February and March and June and July respectively in Kaura LGA. The districts in which these surveys were randomly done included Zankan, Manchok, Bondong, Kadarko, Fadan Kagoro and Kukum (fig.6).

3.2.2.1 Surveys for haemophagous flies.

Four each of non-impregnated biconical and NITSE traps were set about 100 meters apart on locations randomly selected in open bushes and along stream and river banks (Plates 1 & 2) as described by Cuissance et al., (1990). The biconical traps were modelled after Challier and Laveissiere (1973), while the NITSE traps were modelled by Dr. G.A Omoogun (personal communication, 1992).

The location of the tsetse traps was influenced by the season during which the surveys were done. During the dry season when most of the dry vegetation were burnt by fire the traps were set along banks of streams and rivers with riverine vetegations suspected to be infested by tsetse (Ford, 1971; Davies, 1977; Laveissiere et al., 1990). However, during the dry season, these traps were set in the open bushes with flourishing green shrubs and grasses suspected to be suitable habitants for tsetse flies (Laveissiere et al., 1990). These traps were removed after 24 hours and emptied. The biting flies caught in the cage of each trap were immobilized by placing these containers in cool boxes containing ice packs for one hour. These flies were discharged onto a table on arrival in the laboratory. All the flies harvested were identified by their generic and speciies names using external morphological descriptions (Itard, 1989) after which the numbers of flies in each trap were counted.

3.2.2.2 Physical examinations and bleeding of cattle.

Approximately 10% of each cattle herd visited during both dry and rainy season surveys in Kaura were randomly examined for trypanosomes. The name, age and sex of each animal examined was recorded. The physical examination of each animal for the appearance of the hair, body condition, tick infestation, the mucous membranes of the eyes

and mouth, its feeding rates and physical and mental alertness were done under the supervision of a veterinary surgeon.

Subsequently, $10cm^3$ of blood was aseptically withdrawn from the jugular vein using a needle and a $10cm^3$ syringe. Three cubic centimeters ($3cm^3$) of the blood withdrawn from each animal were discharged into labelled bijou bottle containing 0.5mg of ethylene diamine tetra-acetate (EDTA) powder as anticoagulant. The remaining $7cm^3$ blood was poured into a corespondingly labelled plain universal bottle. Similarly, blood was collected directly from the severed jugular veins of six slaughtered pigs in the Manchok abattoir using clean and labelled pairs of the same specimen bottle types used for collecting cattle blood during this study. Each specimen bottle containing each blood sample was tightly covered with a rubber cork. The daily blood samples collected in bijou bottles containing EDTA as anticoagulant were placed in unspecified postures while the blood samples collected in the plain universal bottles were placed in slanting positions in cool boxes to facilitate serum separation from the latter.

3.2.2.3 Parasitological screening of cattle blood for animal trypanosomes.

The uncoagulated blood sample from each animal contained in a bijou bottle was screened for trypanosomes employing parasitological techniques described by Woo (1969; 1970), Boyt (1986a) and Itard (1989). In summary, a wet blood film (WBF) was prepared by placing one drop of uncoagualted blood from each animal on the centre of a clean microscopic slide and carefully placing a clean cover slip on it to avoid the trapping of air bubbles. Similarly, a small drop of each blood sample obtained was placed on another clean microscopic slide about 3 centimeters (3cm) from one narrow edge. One narrow edge of a plastic spreader was placed on the microscopic slide, touching the drop of blood on it at an angle of 45^0. A thin blood smear was made by a gentle but steady forward movement of the spreader. After air-drying, the thin blood smears were fixed in methanol for two minutes. This was followed by staining with freshly prepared solution of Giemsa stain for 30 minutes. The excess stain on each smear was washed off by placing it under gently running water tap. The smears were then air-dried. Approximately 20 fields of

each of the wet and Giemsa stained blood films was examined under X40 magnification and the oil immersion (X100 magnification) objectives of the light microscope, respectively were examined.

In order to detect subclinical cases of bovine trypanosomosis and accurately determine the level of trypanosome parasitaemia, the haematocrit centrifugation technique (HCT), considered the most sensitive direct parasitologic method (Kalu et al., 1986; Boyt, 1986; Masake et al., 1995) was employed. Each uncongulated blood sample brought from the field in a bijou bottle was carefully mixed before aspirating same into three quarters of a heparinized capillary tube by capillary action. One end of each of the capillary tube was then carefully sealed with a hot flame from a bursen burner. The tubes were arranged in the grooves of the plate of a microhaematocrit centrifuge (Gelman Hawksley Ltd, England) with the sealed end outwards, and then contrifuged for five minutes at 12000g as previously prescribed by Dacie and Lewis (1975). The packed cell volume (Pcv) percentage of the spun blood in the capillary tube were read with the Hawksley microhaematocrit reader (Gelman Hawksley Ltd., England). The buffy coat/plasma junction of each spun blood in the capillary tube was examined directly for trypanosomes under the X40 magnification of light microscope. The trypanosome parasitaemia observed in the buffy coat of any blood sample was scored 1+, 2+, 3+ and 4+, equivalent to 1-5, 6-10, 11-20 and greater than 11-20 trypanosomes per microscopic field, respectively, as previously determined by Woo (1969; 1970). Blood samples that had massive parasitaemia of pure T.vivax, T. congolense and T. brucei isolates were separated and inoculated subcutaneously into a goat (for T.vivax) and a pair of rats (for T. congolense and T. brucei). The subinoculated animals were conveyed to the laboratory for further studies.

3.2.2.4 Serological screening of cattle blood for animal trypanosomes

Separated serum from each animal contained in a plain universal bottle was harvested into a pair of labbeled tubes using pasteur pippetes. Each pair of serum sample was screened for the antigens of Trypanosoma vivax, T. congolense and T. brucei using Enzyme-Linked Immunosorbent Assay (ELISA) previously described by Nantulya et al., (1987), Nantulya and Lindqvist (1989) and Masake et al., (1995). The ELISA KIT used for the diagnosis of single and mixed T. brucei, T. vivax and T. congolense infections during this study was prepared at the Food and Agricultural Organisation/International Atomic Energy Agency (FAO/IAEA) centre in Seiberdorf, Austria, in collaboration with

the International Livestock Research Institute (ILRI) in Nairobi, Kenya. All items used in this technique to detect circulating antigens of T. brucei, T. vivax and T. congolense in the test sera brought from the field were components of the ELISA KIT. Briefly, stocks of monoclonal antibodies (MABs) against T. brucei, T. vivax and T.congolense were diluted in a ratio of 1:5000 with a washing/serum diluting buffer made up of 0.5% tween - 80 in phosphate buffered saline (PBS) at pH 7.2. Each of 92 wells of a flat-bottom micro-ELISA plate was coated with 100µl of the diluted MAB against each of the trypanosome species and kept overnight 4^0c in a refrigerator at 4^0c. These plates were then flipped to discard unbound MABs, washed twice with the washing solution and blotted to dryness with tissue paper. Exactly 100ul of each test serum diluted with the buffer solution in the ratio of 1:10 for T. congolense or T.vivax and 1:20 for T. brucei were pipetted into the MAB coated of each plate. The plates containing the MAB against each species was labelled for easy idebtification. The plates were tapped gently with one finger to ensure proper mixing of the contents of each well. They were incubated at room temperature (25^0c) for 15 minutes to allow MAB/trypanosome antigen reaction. These plates were again flipped to discard excess antigen and then rinsed twice with the buffer solution. Horse radish peroxidase (HRPO) conjungated to homologous MAB against each trypanosome species FAO/Seiberdorf, Austria) was diluted with the buffer solution at the ratio of 1:500. Exactly 100ul of this was added to each well of the microtitre plate and incubated for one hour at 37^0C. Each plate was then washed by flooding with the buffer which was allowed to remain on the plate for 10 minutes. This washing was repeated thrice. This was followed by the addition of 100ul of a mixture of freshly prepared substrate, hydrogen peroxide and chromogen, (2, 2 - azinobis (3-ethyl) - benzithiazoline -6 - sulphonic acid) (ABTS, Sigma, St. Lous, U.S.A). These plates were incubated in the dark at room temperature (25^0C) for 30 minutes. Thereafter, the intensity of colour change in each well was visually scored as negative (–), and 1+, 2+, 3+ for slightly positive, moderately positive and strongly positive trypanosomosis cases respectively. The results of both the parasitological and serological (ELISA) screenings of cattle for bovine trypanosomosis were statistically analysed using the Chi square (X^2) statistical analysis.

3.3 RESULTS

3.3.1 Vectors of bovine trypanosomosis caught

The biological vectors of bovine trypanosomosis caught with both biconical and the NiTse tsetse traps in Kaura LGA during the dry and the rainy seasons were riverine testse flies, namely, Glossina palpalis palpalis and G. tachinoides. The mechanical vectors caught with these traps during the same survey periods were Stomoxys calcitrans, Tabanus spp and Haematopota spp. Greater numbers of the various biting flies were caught during the rainy season and with the NiTse traps than during the dry season and with the biconical traps (Table 1). Except for two G tachinocides caught in Kaura and Zankan villages during the rainy season, the riverine tsetse were caught along the banks of River Kajim (Plate 2) in Manchok district. The mechanical vectors of bovine trypanosomosis were, however, caught, throughout the entire Kaura LGA, during both the dry and rainy seasons. Greater numbers of G. p. palpalis were caught along River Kajim than G. tachinoides during both the dry and rainy seasons. The most commonly caught mechanical vector of trypanosomes in Kaura LGA during both the dry and rainy seasons were Stomoxys calcitrans followed by Haematopota spp, while Tabanus spp was the least encountered (Table 1)

3.3.2 Physical appearance of the Fulani cattle.

During the dry season surveys, the Fulani cattle in Kaura LGA appeared more cachectic, showing paler mucous membranes than the same animals examined during the rainy season. Ticks infestation limited to the soft skin surfaces of the groins, genito/anal, the tail and ear regions was scanty during the dry season and moderate during the rainy seasons. All trypanosome positive cattle given deep intramuscular injection of 7.0 mg/kg diminazene aceturate during this study were reported by the Fulani owners to have recovered.

3.3.3 Prevalence of bovine trypanosomosis

Higher overall bovine trypanosomosis of 53.4% (61) was observed in Kaura LGA, during the rainy season than the 17.26% (29) prevalence obtained during the dry season

(Fig.8, Tables 2 and 3), using the parasitological diagnostic methods. (x^2 = 40.31; P>0.05). The antigen, ELISA results of the same surveys similarly showed a higher prevalence of 63.48% (73) during the rainy season than the 47.40% (63) (x^2 =6.08; P>0.05) observed during the dry season for this disease among the same cattle population screened (fig. 8, Tables 2 and 3). More cases of bovine trypanosomosis were diagnosed using the antigen ELISA than those parasitologically diagnosed during the dry and rainy seasons surveys (fig. 8; Tables 2 & 3).

3.3.4 Trypanosome infection rates

Results of both parasitological and antigen ELISA diagnostic methods used during this study showed that pure T. brucei infections were the most commonly detected, followed in decreasing order, by pure T. conglolense and pure T. vivax infections among cattle during both dry and rainy seasons in Kaura LGA (Fig. 8 Tables 2 and 3). During the dry season bovine trypanosomosis cases due to mixed T. vivax/ T. congolense/ T. brucei infections were the most encountered (Table 2). The most commonly observed mixed typanosome infections among cattle during the rainy season were those of T. congolense/T. brucei (Table 3). Bovine trypanosomosis due to T. vivax/ T. congolense infection were the least observed during both dry and rainy seasons (Fig 8; Tables 2 and 3).

3.3.5 Apparent trypanosome species infection distribution

An apparent latitude-dependent distribution of trypanosomes species infecting cattle in Kaura LGA (Figs. 9a, 9b, 10a, and 10b). During the dry season the highest overall infection rate was recorded in Kukum district located 8^0 50′ north of the equator, while the lowest prevalence rate was recorded in Bondong at latitude 10^0 50'N (fig.9a). T. brucei showed a sudden rise from 12% in Manchok district located at latitude 9^0 50′N to 40% in Bondong located on latitude 10^0 50′N (fig.9a). On the other hand pure infection with T. congolense showed an increasing prevalence profile with increase in latitude, except for the fall from 30% observed in Manchok district located on latitude 9^0 50′N to 1% in Bondong district located on latitude 10^0 50′N (fig.9a). However, other pure or

mixed trypanosome infections (T.vivax, mixed T.vivax/T.congolense and T.vivax/T.congolense/T.brucei) maintained fluctuating prevalence rates ranging between 1% and 20% and unrelated to latitude profile in Kaura LGA (fig.9b)

Although only three districts were surveyed for bovine trypanosomosis during the rainy season in Kaura LGA, the overall prevalence range of between 65% and 50% was higher than that of the dry season which was between 49% and 18% (Fig.8). The overall infection trend for either pure T. congolense or mixed T. congolense/T. brucei was not related to the latitudinal location of any district (Figs 10a and 10b).



Plate 1: A NITSE trap set in the open bush during the rainy season in Zankan Village.

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Plate 2: Biconical and NITSE traps set along River Kajim Banks during the rainy season.

TABLE 1 SEASONAL AVERAGE TRAPPING RATES OF VECTORS OF BOVINE TRYPANOSOMOSIS VECTORS IN KAURA LOCAL GOVERNMENT AREA.

SEASON	NO. OF TSETSE TRAPS SET PER DAY	NO OF VECTOR TRAPPING CONSECUTIVE DAYS/WEEK	AVERAGE VECTOR TRAPPING RATE PER DAY (NO./TRAP/24 HOURS).				
			G. tachinoides	G (p) palpalis	Stomoxys calcitrans	Tabanus spp	Haematopota spp
DRY SEASON	4BICONICAL TRAPS	4	2(8.71%)	4(17.39%)	10(43.48%)	2(8.70%)	5(21.74%)
RAINY SEASON	4BICONICAL TRAPS	3	1(1.89%)	2(3.77%)	44(84.02%)	2(3.77%)	4(7.55%)
	4 NITSE TRAPS	3	2(2.38%)	7(8.33%)	65(77.38%)	2(2.33%%)	8(9.52%)

TABLE 2 PREVALENCE OF BOVINE TRYPANOSOMOSIS IN KAURA LGA DURING THE DRY SEASON

DISTRICT	NO. OF CATTLE SCREENED	OVERALL POSITIVITY				Tv			Tc			Tb			Tv/Tc			Tv/Tb			Tc/Tb			Tv/Tc/Tb		
		A	B	C	D	B	C	D	B	C	D	B	C	D	B	C	D	B	C	D	B	C	D	B	C	D
ZANKAN	36	8	0	0	14	0	0	0	0	0	1	0	0	7	0	0	0	0	0	2	0	0	1	0	0	3
MANCHOK	18	2	0	1	9	0	0	1	1	0	2	0	0	1	0	0	1	0	0	2	0	0	0	0	0	2
F/KAGORO	28	3	0	0	12	0	0	1	0	0	3	0	0	2	0	0	0	0	0	1	0	0	2	0	0	2
BONDONG	20	1	1	1	8	0	0	3	0	0	0	1	1	3	0	0	0	0	0	0	0	0	1	0	0	1
KADARKO	50	11	4	6	30	0	0	2	1	3	3	3	3	8	0	0	4	0	0	2	0	0	4	0	0	3
KUKUM	16	4	0	0	10	0	0	0	0	0	2	0	0	4	0	0	0	0	0	1	0	0	4	0	0	1
TOTAL	168	29	5	8	83	0	0	7	0	4	11	1	4	25	0	0	5	0	0	8	0	0	10	0	0	12

KEY:-

A = Haematocrit centrifugation technique (HCT)

B = Wet blood film (WBF)

C = Thin blood smear (TS)

D = Antigen Enzyme - linked immunosorbent Assay (ELISA)

Tv = Trypanosoma vivax

Tc = Trypanosoma congolense

Tb = Trypanosoma brucei

TABLE 3 PREVALENCE OF BOVINE TRYPANOSOMOSIS IN KAURA LGA DURING THE RAINY SEASON

DISTRICT	NO. OF CATTLE SCREENED	OVERALL POSITIVITY				Tv			Tc			Tb			Tv/Tc			Tv/Tb			Tc/Tb			Tv/Tv/Tb		
		A	B	C	D	B	C	D	B	C	D	B	C	D	B	C	D	B	C	D	B	C	D	B	C	D
ZANKAN	36	19	6	5	24	0	0	1	5	4	5	1	1	7	0	0	0	0	0	1	0	0	6	0	0	4
MANCHOK	35	13	1	0	23	0	0	2	1	1	3	0	0	4	0	0	2	0	0	2	0	0	6	0	0	4
F/KAGORO	44	29	15	12	26	1	3	4	5	1	2	9	6	8	0	1	1	0	0	2	0	1	3	0	0	6
TOTAL	115	61	22	17	73	1	3	7	11	6	10	10	7	19	0	1	3	0	0	5	0	1	15	0	0	14

KEY:-

A = Haematocrit centrifugation technique (HCT)

B = Wet blood film (WBF)

C = Thin blood smear (TS)

D = Antigen Enzyme - linked immunosorbent Assay (ELISA)

Tv = Trypanosoma vivax

Tc = Trypanosoma congolense

Tb = Trypanosoma brucei

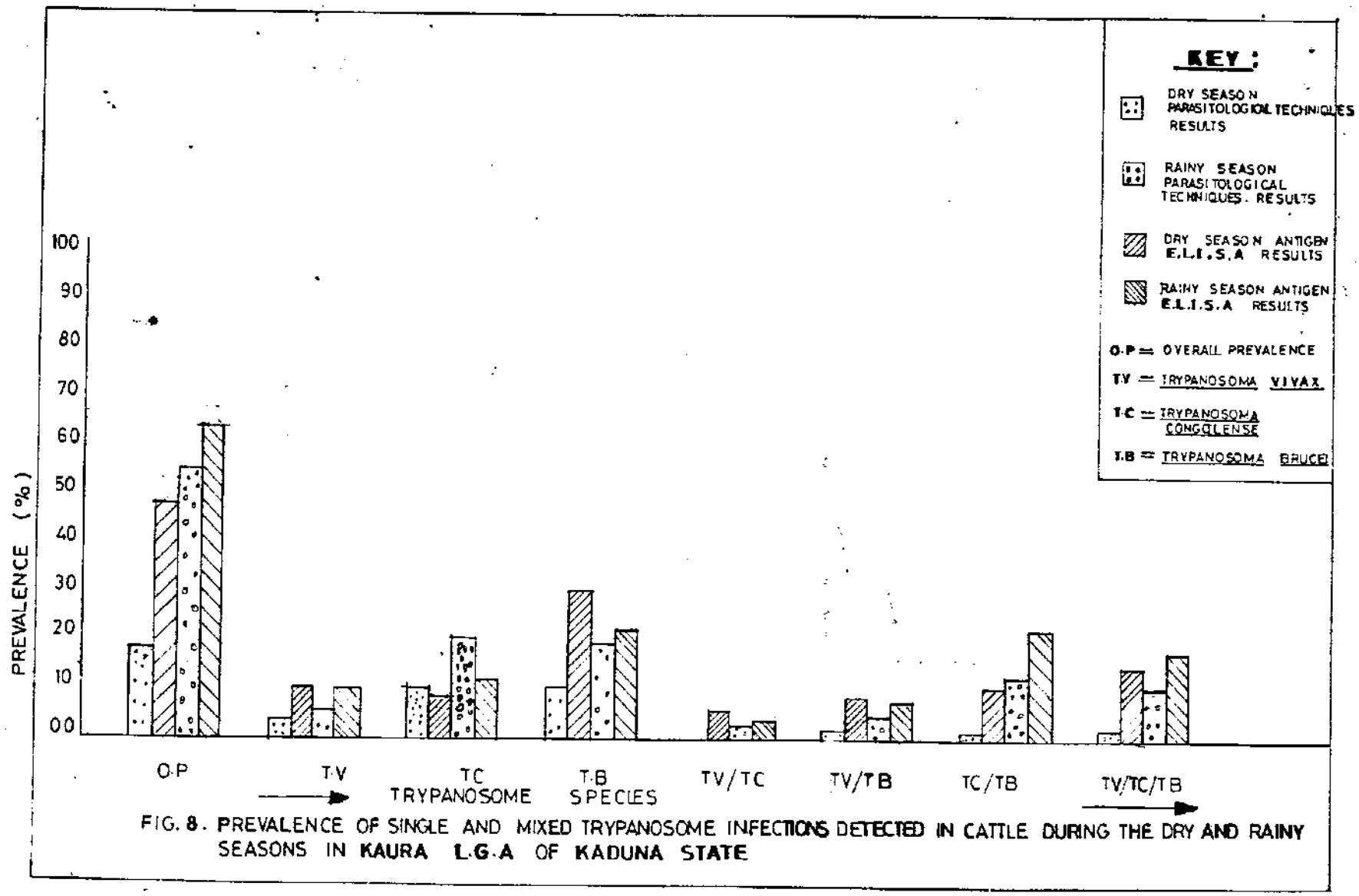


FIG. 8. PREVALENCE OF SINGLE AND MIXED TRYPANOSOME INFECTIONS DETECTED IN CATTLE DURING THE DRY AND RAINY SEASONS IN KAURA L.G.A OF KADUNA STATE

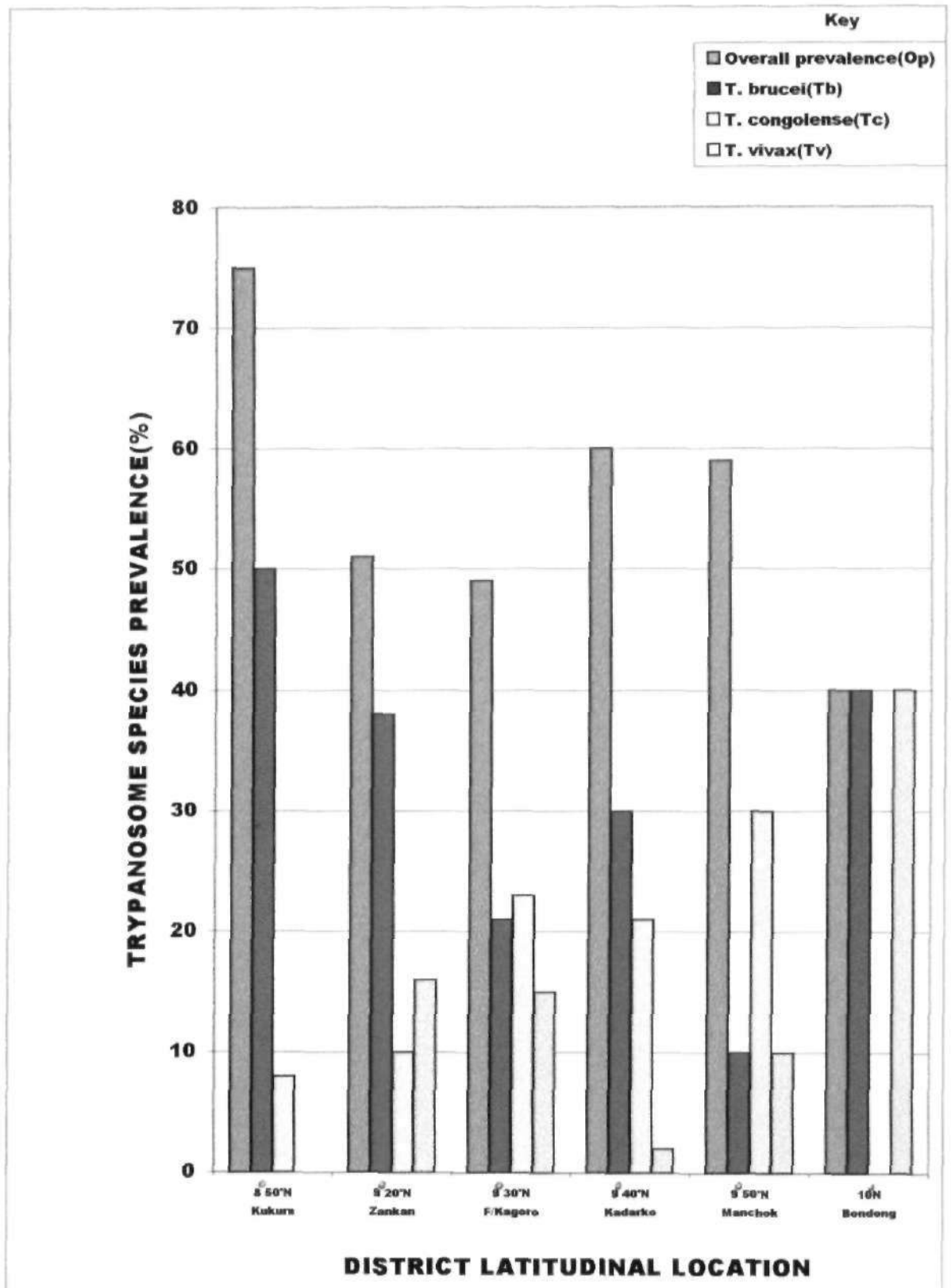


Fig. 9a. Apparent Latitude-Dependent Distribution Trend For The Overall Trypanosomosis and Single Trypanosome Species Prevalence in Cattle in Kaura LGA During the Dry Season.

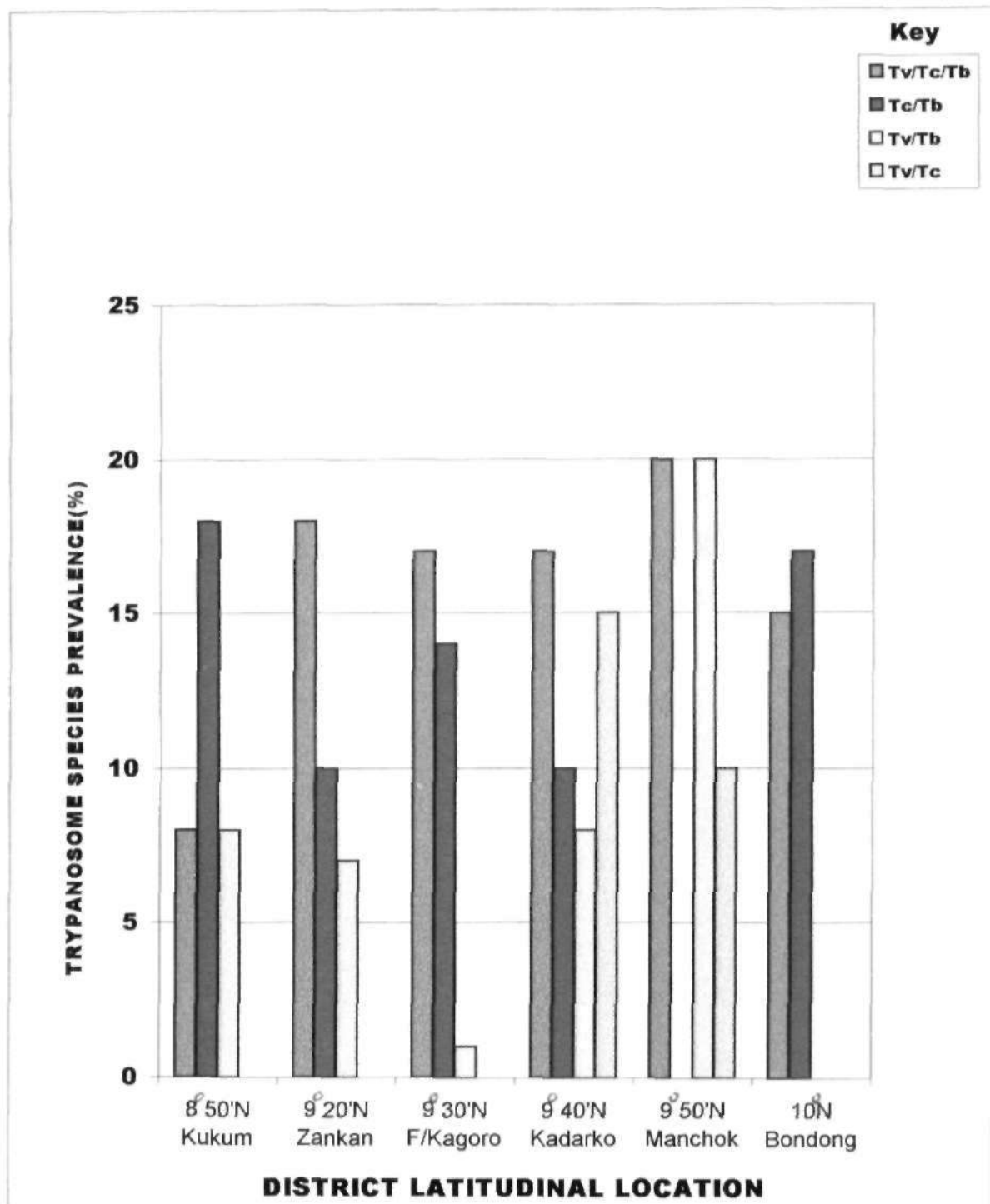


Fig. 9b Apparent Latitude-Dependent Distribution Trend For Mixed Trypanosome Species Prevalence In Cattle In Kaura LGA During The Dry Season.

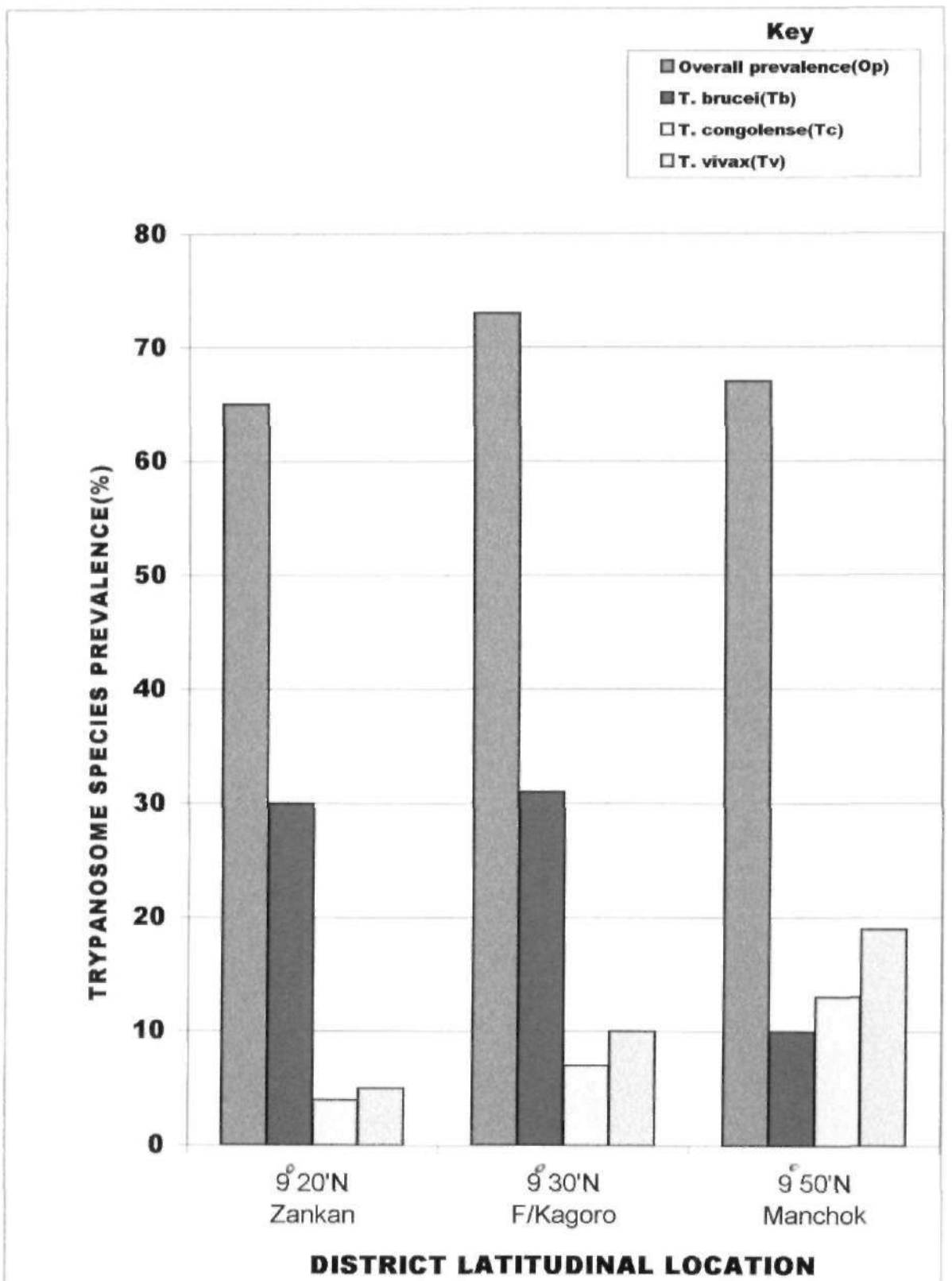


Fig. 10a. Apparent Latitude-Dependent Distribution Trend For the Overall Trypanosomosis and SingleTrypanosome Species Prevalence In Cattle In Kaura LGA During The Rainy Season.

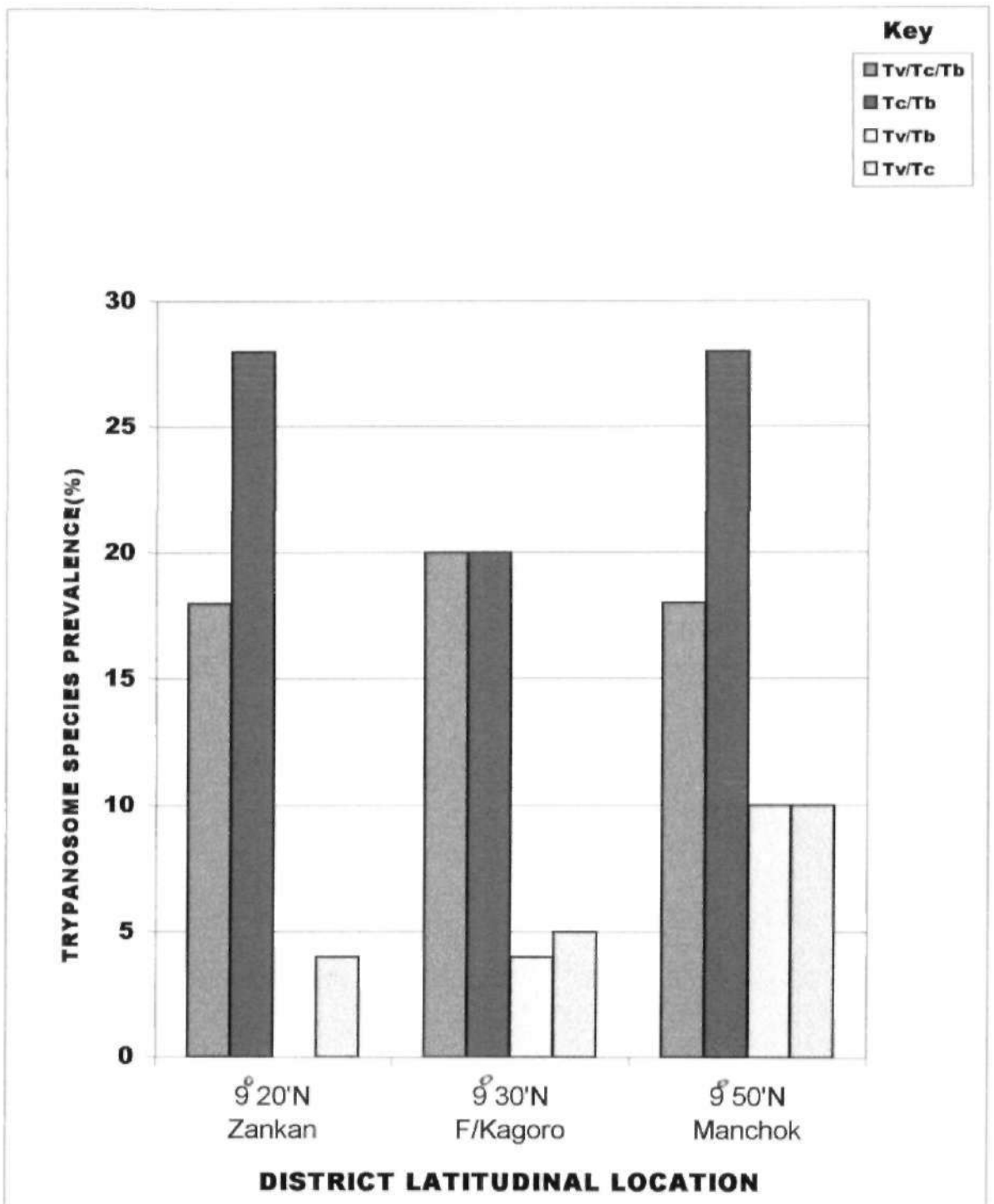


Fig. 10b Apparent Latitude-Dependent Distribution Trend For Mixed Trypanosome Species Prevalence In Cattle In Kaura LGA During The Rainy Season.

3.4 DISCUSSION

The high prevalence rates of bovine trypanosomosis observed during both the dry- and rainy-season surveys indicate the endemicity of the disease in Kaura LGA, constituting a serious obstacle to the growth of the cattle industry in the area. High bovine trypanosomosis prevalence rates, have been reported in supposedly tsetse-free Gongola (Daniel et al., 1993), Plateau (Kalu, 1996a; 1996b) and Borno (Nawathe et al., 1994) States and in tsetse endemic Benue (Kalu, 1995), Anambra (Ekejindu et al., 1992, Wosu and Nwanta, 1992; Anene and Ezekwe, 1995) and Oyo (Anosa et al., 1995) States. These observations indicate that bovine trypanosomosis is spreading steadily in Nigeria, reaching foci previously considered tsetse- and trypanosomosis- free. The ceasation of the tsetse control activities in Nigeria and the effects of global warming might account for the reinvasion of previously reclaimed areas and the infestation of new foci in previously free areas respectively by animal trypanosomosis vectors (Bature, 1981; Ilemobade et al., 1991; Cook, 1992).

The higher prevalence of bovine trypanosomosis during the rainy season may correlate with the corresponding increase in population and possible acceleration in transmission activities of the various vector species during this period in Kaura LGA. Results of previous epidemiological studies carried out in Kenya (Palling, 1989) and in the Ghibe Valley in Southwest Ethiopia (Leak et al., 1993; Rowlands et al., 1993) revealed similar correlation between seasonal tsetse densities and the prevalence of bovine trypanosomosis. This explains why most cattle in Kaura LGA are annually moved on the hoof to supposedly tsetse-free Bauchi State and Atakar/Kagoro hill top during the rainy season to minimise trypanosome infection rate and reduce mortality due to bovine trypanosomosis. Also, the higher infection rate detected using the antigen ELISA confirmed the previous observations of Nantulya and Linqvist (1989), Nantulya (1989, 1990), Akinpelu and Oyejide (1990), Waitumbi and Nantulya (1993) and Masake et al. (1995) and Mathioli and Faye (1996) that the technique is currently the most sensitive and specie-specific animal trypanosomosis diagnostic technique.

The observations that T. brucei and T.congolense caused the most frequent single infections, respectively in cattle during this study have also been reported in Eastern Nigeria (Fakae and Chiejina, 1993), Southern Nigeria (Omeke, 1994), some parts of Kaduna and Plateau States (Agu et al., 1989; Joshua and Shanthikutmar, 1989; Kalu 1991) as well as during nationwide bovine trypanosomosis surveys (Ilemobade et al., 1991; Anosa et al., 1992). These observations are in contrast with the report that T vivax had the highest prevalence in cattle in Shika (Leeflang, 1975), Jema'a and Zangon Kataf LGAs (Agu et al., 1989), some parts of Borno state (Nawathe et al., 1994; Basu et al., 1995) Gongola (Daniel et al., 1993), Benue (Kalu et al., 1991; Kalu, 1995) and Plateau State (Kalu, 1991, 1996a) States. It is possible that the high prevalence reported for T. vivax by these authors, especially in the subhumid zone of Nigeria, where Kaura LGA is also located was mostly derived from extrapolated results from point studies which did not use the highly sensitive and specific antigen ELISA technique. The dominance of single and mixed T. brucei and T. congolense infections observed in Kaura LGA in this study is introducing a new dimension in the epidemiology of bovine trypanosomosis in West Africa where these organisms are considered to be of lesser importance than T.vivax (Leeflang, 1975; Boyt, 1986a; Losos, 1986). Maybe T.vivax, which is predominantly intravascular in the mammalian host and therefore easily accessible, is being gradually suppresed with trypanocides which are used rather indiscriminately in Kaura LGA.

Since all cattle that parasitologically tested positive for bovine trypanosomosis during the rainy season survey were found on top of the Atakar hills, it is not clear whether they acquired the trypanosome infections there or on the lowlands just before they emigrated to the hill top at the onset of the rainy season. Since all the cattle in Kaura LGA move and graze freely during the dry season across the length and breadth of this area, the latitudinal trend of bovine trypanosomosis observed in this study can only be used as a model to determine the prevalence pattern of this disease and that of each of the causative trypanosome species across national and international boundries.

The absence of any Savannah tsetse in all the traps set during both dry and rainy seasons surveys in Kaura LGA confirms the earlier observations of Putt et al. (1980), Bourn (1983), Joshua and Shanthikutmar (1989), Kalu et al. (1991), Kalu (1991),

Omoogun et al (1991) and Odhiabo (1995) that these Glossina species (G.morsitans submorsitans and G. longipalpalis) have been eradicated from the subhumid zone of Nigeria. The possible additional transmission activities of the abundant mechanical vectors observed every where in Kaura LGA might be enhancing the spreading of all the trypanosome species among all the cattle there as previously reported by Diall et al., (1988), Mattausch (1990) Mihok et al., (1995) Solano and Amsler Delafosse (1995) and Egbe - Nwiyi and Chaudhrai (1996). The trapping of only one G. tachinoides each from Kaura and Zankan villages and one G.palpalis palpalis in Kaura village during the rainy season survey might suggest that the riverine tsetse inhabiting River Kajim have also adopted the host seeking migrations of the Savannah tsetse (Leeflang, 1976) as previously reported by Clair (1987) and by Laveissiere et al. (1990).

The concentration of all the riverine tsetse in River Kajim can only be attributed to the availability of the favourable vegetation covers, water and abundant pigs, human, cattle, wild animals and snakes as hosts for blood meals, as previously observed by Bourn et al. (1986), Clair (1987) and Leveissiere et al. (1990). Although the NITSE traps were more efficient in tsetse trapping than the biconical, the use of both might prove useful in controlling bovine trypanosomosis vectors in Kaura LGA in addition to chemotherapeutic and chemoprophylactic covers for the cattle. These control measures must be instituted if the maximum livestock raising potentials of Kaura LGA are to be realized.

CHAPTER IV

DRUG SENSITIVITY OF BOVINE TRYPANOSOMOSIS IN KAURA LOCAL GOVERNMENT AREA OF KADUNA STATE

CHAPTER IV

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4.1 INTRODUCTION

Due to the high morbidity and mortality of bovine trypanosomosis in Kaura Local Goverment Area, cattle owners in the area have come to rely mainly on the chemoprophylactic and chemotherapeutic use of trypanocides to control the disease. The most commonly used trypanocides in the area include diminazene aceturate (Berenil®; Hoechst, Frankfurt Germany) homidium chloride (Novidium®), homidium bromide (Ethidium®) and isometamidium chloride (Samorin®), May and Baker, Dagenham limited, England) (personal communication). However, due to inadequate veterinary supervision, widespread misuse of these trypanocides (without regard to weight of the animal, dose per unit body weight, and frequency of administration) is common. Similarly, uncontrolled cattle movements within, or in and out of the area expose these animals to infections by trypanosome strains with various levels of sensitivity/resistance to commonly used trypanocides. It has been reported by Leeflang (1978), Williamson (1979) and Barret (1997) that prolonged and inadequate invivo exposure of African trypanosomes to the same or different trypanocides of the same chemical type for long periods is conducive for the emergence of resistant strains to such misused drugs. Results of investigations on trypanosomal drug sensitivity have revealed that the combined effects of delayed treatment, rapid decline in plasma concentrations of diminazene aceturate (Aliu et al., 1984; Silayo et al., 1992; Mamman et al., 1993) and the sequestration of the infective forms of T. congolense and T. brucei in the brain which is inaccessible to drugs (Jennings et al., 1977; 1979, 1993; Masake et al., 1984; Whitelaw et al., 1985;) render trypanocides ineffective in the treatment of animal trypanosomosis.

It was revealed by animal health assistants and Fulani cattle owners that for over ten years, bovine trypanosomosis had continued to be responsible for high cattle deaths in Kaura LGA than any other animal disease in spite of the widespread chemoprophylactic

and chemotherapeutic use of trypanocides (personal communication). In spite of these findings there is no information in the literature on the trypanocidal sensitivity of trypanosome isolates from Kaura LGA. This study was therefore designed to investigate the sensitivites of Kaura isolates of T. brucei and T. congolense to therapeutic doses of diminazene aceturate and isometamidium chloride in Red Sokoto goats. Results obtained from this study would be used to design effective chemoprophylactic and chemotherapeutic treatment of bovine trypanosomosis in Kaura LGA.

4.2 MATERIALS AND METHODS

4.2.1 Trypanosome isolates

Isolates of T.congolense and T. brucei obtained from cattle in Kaura LGA during bovine trypanosomosis surveys were used for this study. Trypanosoma congolense and T. brucei isolates from Kaura were accurately identified prior to this study using their individual biological features as described by Boyt (1986a) and Itard (1989) and the antigen ELISA described by Nantulya et al. (1987), Nantulya and Lindqvist (1989) and Masake et al. (1995). Albino rats were inoculated with cattle isolates of T. congolense and T. brucei obtained from the field during the bovine trypanosomosis surveys in Kaura LGA. Wet blood film preparations from previously inoculated rats were examined under the X40 magnification of the light microscope. T. congolense and T. brucei were indentified using their movement characteristics as described by Kalu et al. (1986) and Boyt, (1986a). When the first peaks of trypanosome parasitaemia were dectected using the Giemsa stained and wet blood films (WBF) described by Boyt (1986a) and the haematocrit centrifugation techniques (HCT) described by Boyt (1986a) and Kalu et al. (1986), the rats were anaesthesized with chloroform soaked in cotton wool in large glass beakers for two to three minutes. Five cubic centimeters of blood was collected from each rat by cardiac puncture using the tuberculin syringe. The blood withdrawn from each rat was discharged into a labelled bijou bottle containing 0.5mg of ethylenediaminetetraacetate (EDTA) as anticoagulant. The trypanosomes contained in each blood sample were cryopreserved as stabilates in liquid nitrogen canisters.

4.2.2 Experimental animals

Red Sokoto goats, which had been described as being highly susceptible and therefore suitable for studying clinical trypanosomosis (Saror, 1980; Adah et al., 1993) were used for this study. The animals were purchased in the open market of Kafur village in Katsina State considered to be tsetse-free (Aliu, 1975). Their ages ranged from 10 to 12 months. These animals were confined in fly-proof pens in the Faculty of Veterinary Medicine, A.B.U, Zaria, throughout the study period and fed with ground maize, guinea corn husk (dusa), hay, groundnut and beans leaves, and water.

4.2.3 Preconditioning of experimental animals.

During the preconditioning period of two to three weeks, each of the goats was given one dose of long acting oxytetracyline (Terramycin-LA, Pfizer Nigeria Limited) by intramuscular injection (IM) forthnightly at a dosage of 10mg/kg body weight to protect them against cowdriosis. They were dewormed by the oral adminstration of albendazole (Sam Pharmaceuticals Nig. Ltd.) at a dose of 7.5mg/kg of body weight. Tick infestation was controlled with deltamethrin pour-on preparation applied with a syringe on a straight line from one earbase to another, and from the head through the backline to the tail of each animal as prescribed by the manufacturer (Zimbabwe Phosphate Industries Limited, Harare).

4.2.4 Grouping of experimetal animals

Six goats were randomly selected for Trypanosoma brucei studies. These goats were further divided into two groups of three each for post-infection studies on the effects of diminazene and isometamidium chemotherapy. Similarly, another set of six goats randomly selected were divided into two groups of three each for studying the chemotherapeutic effects of diminazene and isometamidium respectively, following T. congolense infections. However, one goat each used as T. brucei and T. congolense donor was left as untreated control. Two goats were preserved as uninfected controls.

4.2.5 Preinfection screenings of experimental animals

4.2.5.1 Body weights (kg)

During the preconditioning period, each goat was weighed weekly with a bathroom scale. The average weekly weight (in kilograms) for each group of goats was obtained by totalling the individual body weights and dividing the figure obtained by the number of animals in each group. The average values were plotted graphically.

4.2.5.2 Rectal temperature (^{0}C)

The morning (8.00 am) rectal temperature of each goat in degrees celcius (^{0}C) was obtained thrice weekly (on Mondays, Wednesdays and Fridays). The temperature was

determined by reading the mercury level of a clinical thermometer inserted into the rectum of each goat and left for one minute. The average weekly temperature values for each group of goats was obtained and plotted graphically.

4.2.5.3 Haematological parameters

The haematological parameters were determined thrice weekly for each goat using techniques described by Dacie and Lewis (1975) and Boyt (1986). The blood withdrawn from each goat by jugular venipuncture was discharged into a labelled bijou bottle containing 5mg of Ethylenediaminetetraacetate (EDTA) as anticoagulant. The packed cell volume (PCV) for each goat was determined as described previously.

The total red blood cell (RBC) count (10^6/mm^3 of blood) for each goat was determined with the Neubauer Counting Chamber following the dilution of its blood sample with formal-citrate solution in a ratio of 1:200, using an rbc dilution pipette. Similarly, the total white blood cell (WBC) count (10^3/mm^3 of blood) was determined for each animal using the Neubauer counting Chamber after diluting its blood sample with 3% acetic acid solution in a WBC pipette at a ratio of 1:20. The haemoglobin concentration (g/100cc of blood) for each goat blood sample was determined using the cyanmethaemoglobin method as follows. Approximately 0.02cc of each uncoagulated blood sample taken was pipetted into a tube containing 5cc of Drabkins cyanide ferricyanide solution (modified Drabkins reagent). After mixing thoroughly, the dilluted blood was allowed to stand at room temperature for about 3 minutes before the concentration of the cyanmethaemogobin (stable form of haemogobin) was read in a colorimeter at a wavelength of 540 nanometer (nm). The weekly average values of PCV, RBC, WBC and Hb determined for each group of goats were plotted graphically.

4.2.5.4 Screening for haemoprotozoans

The blood of each goat was microscopically examined for haemoparasites using Giemsa stained thin blood smear (Boyt, 1986; Itard, 1989).

4.2.2.5.5 Preinfection screening for trypanosome antigen

Serum separated from blood samples taken weekly from the jugular vein of each goat was screened for the antigen of T. brucei, T. congolense and T. vivax, using the antigen ELISA technique as described by Nantulya et al. (1987) Nantulya and Lindqvist (1989) and Masake et al (1995). This serological technique was used earlier to determine the prevalence of bovine trypanosomosis in Kaura LGA. The weekly pre-infection screening of these goats was done to determine whether these animals had been exposed to trypanosomosis prior to this study.

4.2.6 Experimental procedure

One donor goat (#287) was inoculated with T.brucei (Stabilate No.140) via the jugular vein using syringe and needle, while another (#272) was similarly inoculated with T. congolense (stabilate No. 136). Both stabilates were isolated from cattle in Kaura LGA. These donor goats also served as infected and untreated controls, while two other goats (#258 and #134) served as uninfected untreated controls. Six other goats (#1650, #1687, #1686, #286, #257, #289) were similarly inoculated with approximately $2x10^3$ trypanosomes harvested from the first parasitaemic peak observed in the T. brucei donor goat one week post-infection. Another set of six goats (#293, #287, #274, #261, #299, #255) were inoculated with approximately $1x10^3$ trypanosomes harvested from the first parasitaemic peak observed in the T. congolense donor goat three weeks post infection. Subsequently, changes in body weight (kg), rectal temperatures, (^{0}c) , packed cell volume (PCV), red and white blood cells counts (RBC and WBC), haemoglobin concentration (Hb) and the presence of circulating trypanosome antigens were monitored for each of the goats using the same techniques thrice weekly.

When trypanosome were detected microscopically for three consecutive days in the blood of both T. brucei and T. congolense infected goats as recommended by Jennings et al. (1977), three animals from each group were each given an intramuscular injection of 2% isometamidium chloride at a dosage of 0.5mg /kg body weight. The remaining three in each group were each given an intramuscular injection of 7% diminazene aceturate at a dosage of 7mg/kg body weight. As from twenty four hours after the trypanocidal

treatments haematological parameters, rectal temperature, trypanosome parasitaemia and antigenaemia and body weight were monitored daily until trypanosomes were detected in the blood of each goat and cleared from their circulation therapeutically seven days post-treatment. Subsequently these parameters were monitored for these animals thrice weekly. In the T. brucei group, the post-treatment observation was for ten weeks and eleven weeks for the T.congolense group. Serum extracted from the blood of all the goats weekly during this study were examined for both T. brucei and T. congolense antigenaemia using the antigen ELISA described by Nantulya et al (1987), and Lindqvist (1989), Masake et al (1995) and previously used to determine the prevalence of bovine trypanosomosis in Kaura LGA. The optical density of each serum assayed for trypanosome antigen using the protocol described by these authors was read using the ELISA plate reader fitted with a filter at a wavelength of 405 nanometers ($O.D_{405}$) and a negative $O.D_{405}$ cut-off point of 0.10.

Two cubic centimeters of blood was withdrawn weekly from each infected and treated goat and injected subcutaneously into a pair of mice. These mice were monitored for trypanosome parasitaemia daily for four weeks. Any animal that showed infection relapse post-treatment was treated with an alternative trypanocide and monitored until the study was terminated.

4.3 RESULTS

During the daily examination of the blood of each goat using the HCT it was observed that the Trypanosoma brucei donor goat (#287) became parasitaemic (1 - 5 trypanosomes per microscopic field at x40 magnification) four days post-infection (pi). Its first parasitaemic peak (11 - 20 trypanosomes per microscopic field at x40 magnification) appeared two days later and it died of trypanosomosis as an untreated control three weeks pi. On the other hand, all the six T. brucei infected goats in the treatment group, became parasitaemic (6 - 10 trypanosomes per microscopic field using the HCT) after a prepatent period of one day. The T. congolense donor goat (#272) became parasitaemic (1 - 5 trypanosme per microscopic field at x40 magnification using the HCT) 11 days pi. It exhibited the first parasitaemic peak (11 - 20 trypanosomes per microscopic field at x40 magnification) and died of trypanosomosis on that day as untreated control 14 days pi. All six goats in the treatment group infected with T. congolense became parataemic (1 - 5 trypanosomes per microscopic field at x40 magnification using the HCT) 7 days pi. One died of trypanosomosis two days post-patency.

Similarly, using the HCT, it was observed that the Trypanosoma brucei parasitaemia was cleared in six treated goats by both isometamidium chloride and diminazene aceturate two days post - treatment. No infection relapse was observed in any of these infected goats throughout the post-treatment period of eleven weeks. Parasitaemia was cleared in the six T. congolense infected goats one day post-treatment with either isometamidium chloride or diminazene aceturate. However, infection relapse was observed in one T. congolense infected and diminazene aceturate treated goat (#261) using the HCT 17 days post-treatment. It had a patency of 6-10 trypanosomes per microscopic field, a PCV of 16% and antigenaemia showing an optical density ($O.D_{405}$) of 0.215 (the negative cut-off point being 0.10). The T.congolense relapsed infection in this goat (#261) was cleared with one intramuscular injection of isometamidium chloride at a dosage of 0.5mg/kg body weight. Although T. congolense could not be detected in the peripheral blood of this goat (261) following the second treatment with isometamidium, a second fall in PCV (16%) and rise in antigenaemia ($O.D_{405}$ = 0.210 the negative cut-off point of 0.10) were observed three weeks later (Figs. 12 and 13). The rest of the T.

congolense infected goats, like the six others initially treated with samorin,® neither showed parasitaemia nor rise in trypanosome antigenaemia after both the initial diminazene aceturate and the subsequent isometamidium chloride treatments. Also, none of the mice inoculated with blood from all the T. congolense or all the T. brucei infected and trypanocide treated goats showed parasitaemia throughout the observation period of four weeks.

The change in rectal temparatures, body weights and haematological parameters (PCV, Hb, and RBC) for the T. brucei and T. congolense donor goats are shown in figures 11 and 12. While the total white blood cell count (WBC) for the T. brucei donor goat (#287) continued to decline, that of the T. congolense donor (272) continued to increase with time. The decline in all the clinical parameters (body weights, PCV, RBC, WBC and Hb) observed in all six goats at the onset of the T. brucei infection was reversed to normal values within three weeks of treatment with either trypanocide (isometamidium or diminazene) (Fig.11). Intervention with the same trypanocides at the same dosage did not reverse the decline in the clinical parameters induced by T. congolense infection (Fig. 12). The ELISA results showed that T. brucei liberated more antigens into the circulation of the donor goat than did T. congolense. (Fig. 13). However, ELISA tests in both the T. brucei and T. congolense infected goats did not detect antigenaemia relapse after isometamidium chloride or diminazene aceturate treatment. (Fig. 13). In spite of the absence of detectable antigenaemia, one T. congolense infected goats (#225)died three weeks post diminazene treatment. Another one (#293) treated with isometamidium chloride died four weeks post-treatment, while the third goat (#261) initially treated with diminazene and then with isometamidium following infection relapse died five weeks later.

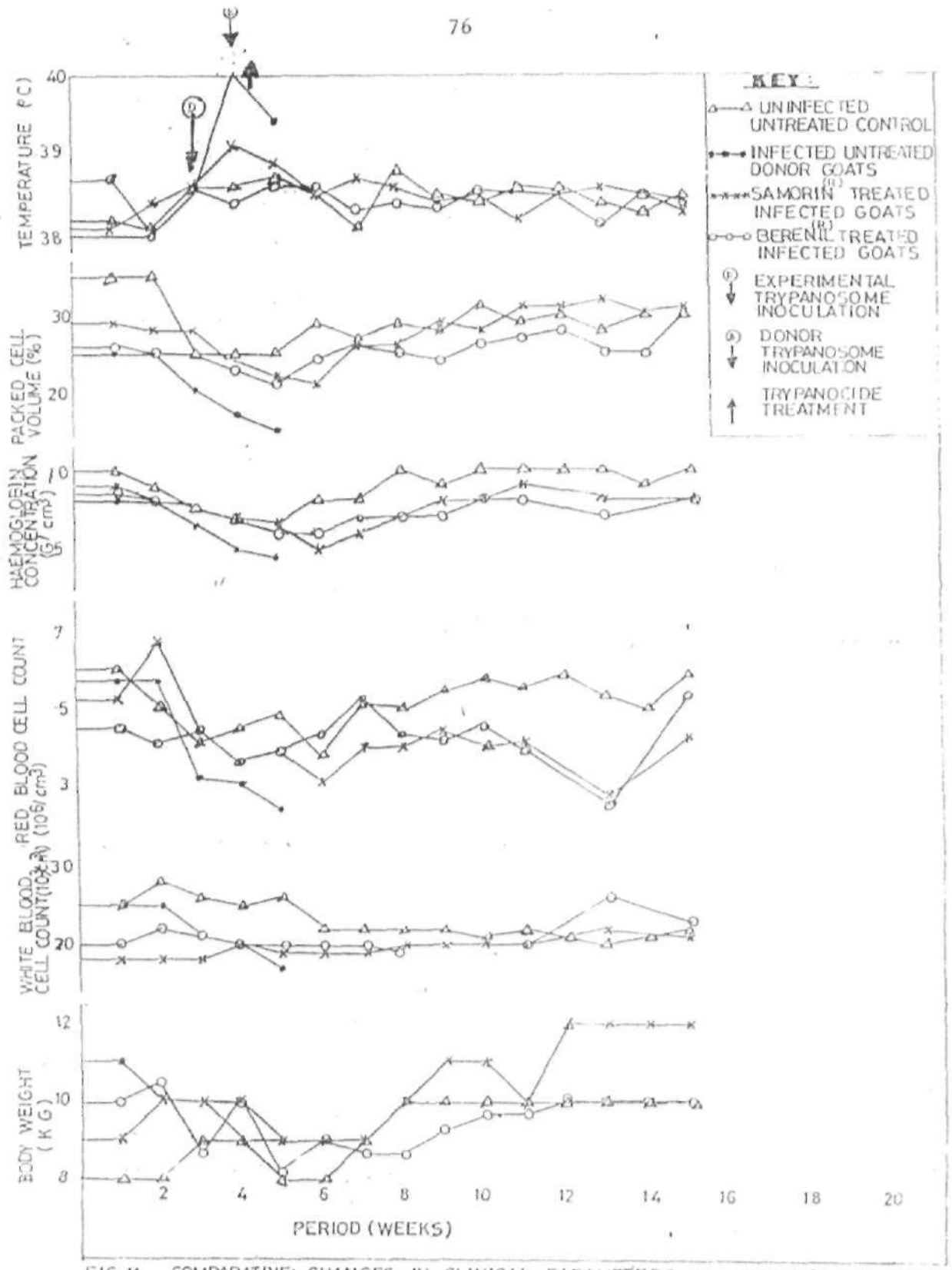


FIG 11. COMPARATIVE CHANGES IN CLINICAL PARAMETERS IN TRYPANOSOMA BRUCEI INFECTED AND TRYPANOCIDE TREATED GOATS

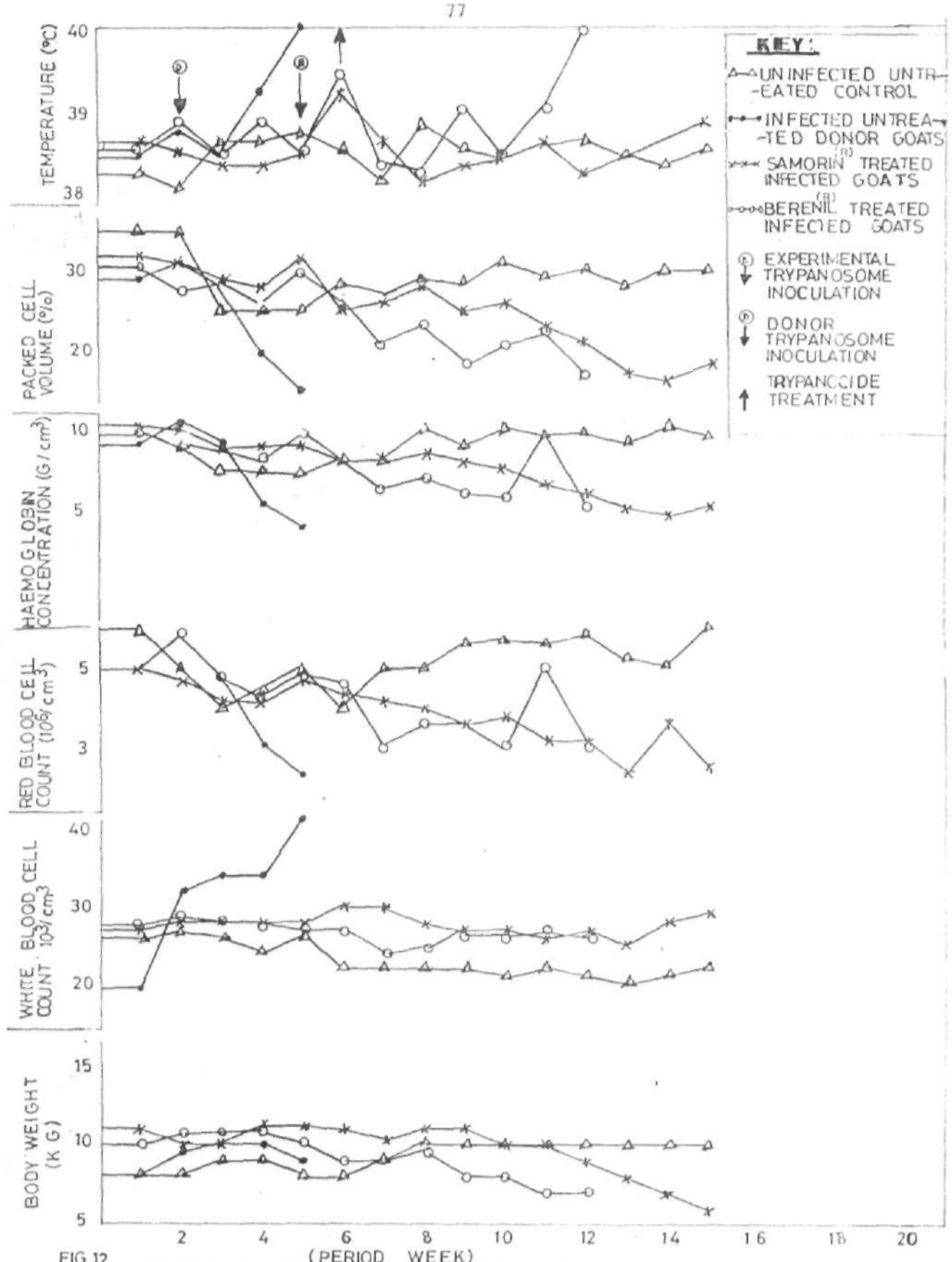


FIG 12 COMPARATIVE CHANGES IN CLINICAL PARAMETERS IN T. CONGOLENSE INFECTED AND TRYPANOCIDE TREATED GOATS

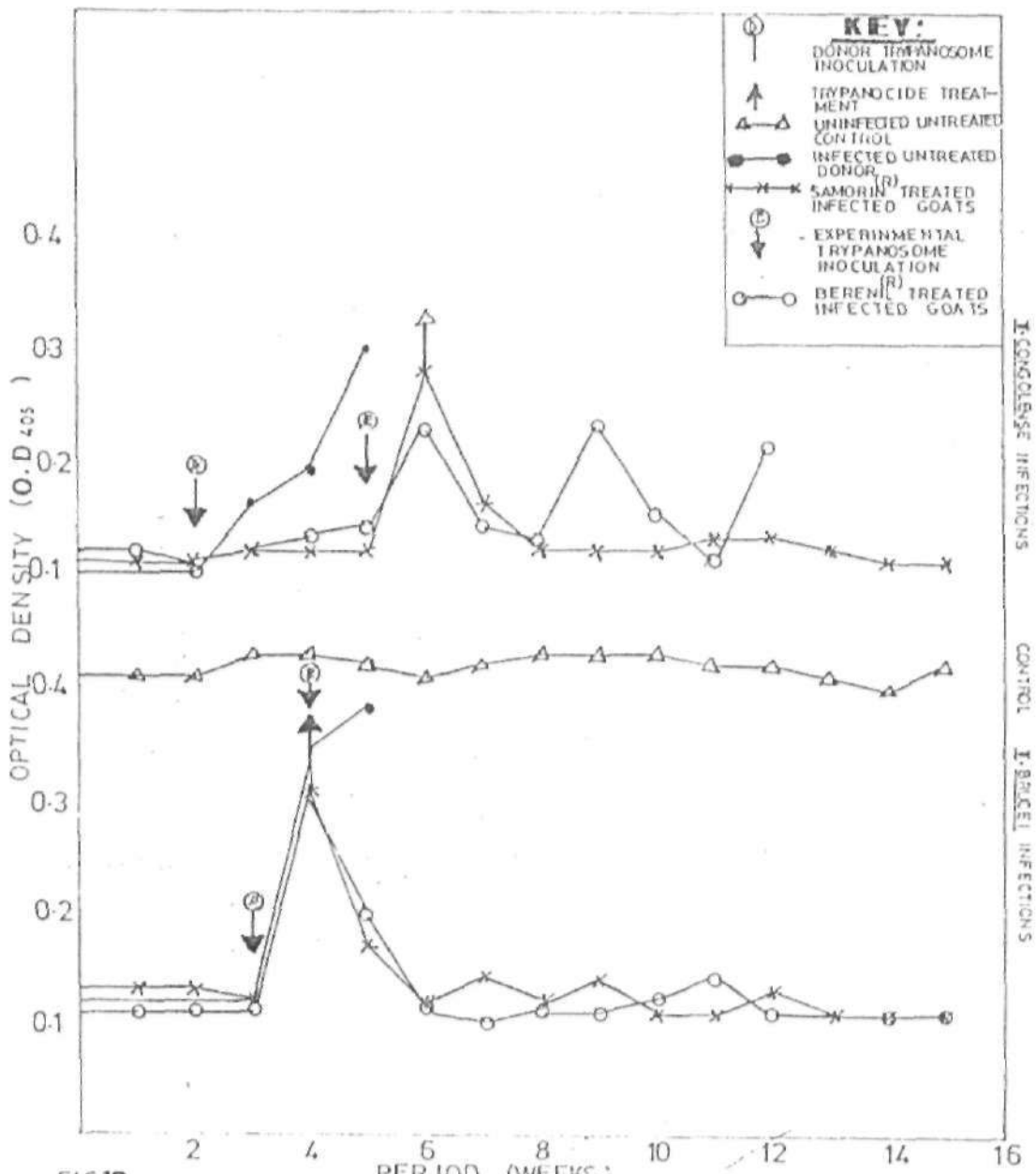


FIG.13 ANTIGEN ELISA ON SERA OF I. CONGOLENSE AND I. BRUCEL INFECTED AND TREATED GOATS

4.4 DISCUSSION

The observed pre-patent periods of one to four days, in T. brucei infected goats was consistent with the range previously reported by Kalu (1983), Kalu and Aina (1984), Boyt (1986), Moloo and Kutuza (1990) and Silayo et al. (1992). Similarly the seven to eleven days prepatent period range observed for T. congolense is within the range previously documented by Whitelaw et al (1985), Kagwa et al. (1988) and Anene et al. (1989) among others.

The death of the T. congolense (infected untreated control) goat (#272) during the first parasitaemic peak and another (#299) two days post T. congolense patency has lent support to the observations of Murray and Morrison (1979), Boyt (1986) and Akinpelu and Oyejide (1996) that T. congolense usually causes a severe form of animal trypanosomosis especially in domestic livestock. The failure of the monitored clinical parameters to reverse to normal values in T. congolense infected goats after trypanocide treatment may indicate the virulence of the isolate (Kaura) and possible resistance to the dose of the trypanocide. The reported cytotoxic effects of T. congolense derived free fatty acids (FFAs) phospholipases, proteases, neuraminidases, pyruvates, trypanosome autolysates and aromatic byproducts (Assoku, 1979; Ibokwae, 1994) released within the circulation before and after trypanocidal treatment might be responsible for the death of the goats. Tizard et al. (1979) reported the induction of irreversible anaemia by released FFAS and phospholipases during experimental T. congolense infection. Other effects of liberated FFAs and phospholipases during active trypanosomosis reported by Assoku (1979) and Tizzard et al., (1979) include the possible disruption of the spleen architecture leading to the tritriated uridine uptake depressions (Meade and Mertin, 1976,), thrombosis (Connor et al., 1963), thrombocytopenia (Shore and Alpers, 1963), hypoglycaemia resulting from accelerated insulin secretion (Losos and Ikede, 1972) and myocardial lesions (Willebrands et al., 1973).

Although serum albumin can block the haemolytic activity of FFAs in animals with low T. congolense parasitaemia (Tizard et al., 1979), larger doses of these and other biologically active substances might have been released from lysed T. congolense than the level of serum albumin in some of the infected goats could completely block from inducing

the pathogenic changes that led to their deaths. Adah et al., (1992) had observed that treatment of T. congolense infected goats with Berenil® nine days post infection did not lower siginificantly the elevated levels of glutamate oxalacetate transaminase (GOT) and glutamate pyruric transaminas (GPT). On the other hand, significant reductions in plasma albumin have been observed in T. congolense infected scottish Blackface sheep (katuyuka - Rwakishaya et al., 1995)

The T. congolense infection relapse observed three weeks post-treatment in one Berenil® treated goat (#261) could be attributed to delay in trypanocidal administration. For example, Saror et al. (1981) observed relapse in four groups of goats experimentally infected with a T.vivax strain reported to be resistant to homidium chloride and diminazene aceturate and when treatment with diminazene at a dosage of 3.5mg/kg body weight was delayed until four, seven, fourteen and twenty-one days post-patency. Results of experimental studies carried out by Jennings et al. (1979), Masake et al. (1984), Whitelaw et al. (1985) and Mamman et al. (1993; 1994), revealed that when trypanocidal treatment is delayed, beyond 24 hours post infection, T. brucei and T. congolense invade the central nervous system (CNS) , the microcirculation, lymph nodes, spleen, heart or born marrow of infected animals where most drugs cannot easily penetrate. Alternatively, the short half-life of diminazene aceturate marked by rapid decline of its plasma concentration over 14 - 21 hours (Aliu and Ødegaard., 1985; Aliu, 1989; Mamman et al,. 1995; 1996) might suggest that some T. congolense cells insufficiently exposed to this drug during this study might have developed resistance to it as previously observed by Kamisky et al. (1990).

The absence of parasitaemia and significant levels of antigenaemia (using ELISA) over a four week period in mice inoculated with blood of the three goats infected with T. congolense and treated with isometamidium chloride probably indicate that there was no infection relapse in these animals. From the results of this study it can be concluded that the T. brucei isolate used is susceptible to the curative doses of diminazene and isometamidium. However, since T. congolense is most likely to develop drug resistance (Boyt, 1986c), it is most probable that the isolate used during this study is resistant to Berenil at the therapeutic dose of 7.0 mg/kg. body weight.

CHAPTER V

CONCLUSION

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Considering the prevailing favourable epizootiological factors, the high prevalence rates of bovine trypanosomosis observed during this study, and the attendant socio-economic effects of the disease, Kaura LGA can be considered an endemic focus for bovine trypanosomosis. It was previously believed that T. brucei is not economically important in bovine trypanosomosis (Boyt, 1986a) and T. congolense is principally the important etiological agent of this disease in East Africa, while T. vivax predominates in West Africa (Leeflang, 1978; Boyt, 1986a). However, the results of this study have confirmed those of the other recent workers (Kalu, 1991; Ilemobade et al., 1991; Anosa et al., 1992) that T. brucei and T. congolense might be posing greater threats to livestock population than T. vivax in the West African subhumid zone. The restriction of tsetse flies to River Kajim and the wide distribution of mechanical vectors of tryapnosomes throughout Kaura LGA suggest that the biological vectors maintain the trypanosomes while the other biting flies spread them among cattle in locations where tsetse flies are not prevalent as previously observed by Mattausch (1990) in Burkina Faso. Consequently since River Kajim is very short (about three kilometers in length) it will be easier and more cost effective to eradicate tsetse flies in the area using tsetse traps which are affordable and can be operated and maintained by the farmers rather than employing insecticides. The use of tsetse traps has achieved much success in controlling tsetse and trypanosomosis in Northern Cote d'Ivoire (Douti et al., 1986), Yeremo grazing area of Central African Republic (Cuisance, 1988) and in Sideradougou in Burkina Faso (Cuisance et al., 1990).

Although the results of the drug sensitivity studies on cattle trypanosome isolates from Kaura LGA recorded an apparent infection relapse with T. congolense further drug trial need be done with diminazene and isometamidium using patency-dependent time interval treatment techniques of Silayo et al. (1992) and Mamman et al. (1993) to clearly

elucidate the trypanocide sensitivity of T. congolense. This is because these trypanocides are still the most effective for the treatment of animal trypanosomosis and cross resistance between them has not been reported (Schillinger, 1989).

Abundant forage, adequate water and great potentials for agricultural crops have encouraged many Fulani cattle owners to adopt agropastoral live styles in Kaura LGA. The control of tsetse and bovine trypanosomosis in his area will enhance cattle raising and meat production in Kaduna State. However, the present spreading rates of tsetse and bovine trypanosomosis which is enhanced by the current global warming effects (Cook, 1992; Rogers and Randolph, 1993) is likely to hinder the development of livestock in the subhumid zone if vigorous vector and parasite control measures are not pursued and sustained.

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APPENDICES

RESULTS OF DRY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN ZANKAN DISTRICT OF KAURA LOCAL GOVERNMENT AREA

ANIMAL'SNAME	AGE(YEARS /SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	E TS	L	I Tv	SA Tc
1. NORE	6/F	Emaciated, pale m/m	23	+	-ve	ntf		+	
2. KORO	4/F	Emaciated, pale m/m	25	-ve	-ve	"			
3. BOJO	6/F	Good appearance, normal m/m	30	+	-ve	"			
4. SODE	3/F	Good appearance Hyloma tick	27	+	-ve	"			
5. DINYAYE	3/F	Good appearance	28	-ve	-ve	"			+
6. YAKAMA	5/F	Good appearance	28	-ve	-ve	"			
7. NGWO	4.F	Good appearance	28	+	-ve	"		+	
8. DAUDE	6/F	Good appearance	28	+++	-ve	"		+	+
9. DEBOJO	2/M	Good appearance	26	-ve	-ve	"			
10. WULE	6/F	Emaciated Hyloma ticks	22 10	-ve	-ve	"		+++	++
11. NDOWA	6/F	Emaciated and weak, pale m/m	19	-ve	-ve	"			
12 DONA	10/F	Emaciated. pale m/m	26	-ve	-ve	"			+
13. GODEL	12/F	Emaciated, pale m/m	20	-ve	-ve	"			
14. JIDOMA	8/F	Emaciated, pale m/m	26	-ve	-ve	"			
15. HURE	6/F	Emaciated, Boophilus ticks	24	-ve	-ve	"			
16. DAKE	5/F	Emaciated, pale m/m	23	-ve	-ve	"			
17. HURE	6/F	Emaciated, pale m/m	30	-ve	-ve	"			
18. SAJE	7/F	Emaciated. pale m/m strunted gro.vth, poor calving	15	-ve	-ve	"			

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF DRY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN ZANKAN DISTRICT OF KAURA LOCAL GOVERNMENT AREA

ANIMAL'SNAME	AGE(YEARS/SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
19. JOMO	9/F	Emaciated. Boophilus ticks	15	-ve	-ve	ntf			+
20. FURE	8/F	Emaciated, had diarrhoa	14	-ve	-ve	"			
21. JOMO	7/F	Emaciated. Boophilus ticks	18	-ve	-ve	"			
22. SAJE	10/F	Emaciated, pale m/m	14	-ve	-ve				++
23. KABEL	10/F	Emaciated, pale m/m	17	++					
24. NYAYE	16/F	Emaciated, Hyaloma ticks. Pale m/m	21	-ve	-ve	ntf			
25 MALe	3/F	Good appearance, Amblyoma Variegatum	33	+++					
26. MBODERI	2/F	Emaciated	24	-ve	-ve	ntf			
27. MALERI	2/M	Good appearance	25	-ve	-ve	"			
28 DAUDE	3/M	Good appearance	30	-ve	-ve	"	+	+	+
29 MORCHI	4/M	Good appearance	25	-ve	-ve	"			+
30. NORE	5/F	Emaciated	26	-ve	-ve	"			
31. TURKEL	9/F	Good appearance	36	-ve	-ve	"			
32. HUPe	6/F	Monge, mitestrice	23	ve	-ve	"			
33. YELEI	3/F	Good appearance	25	+++					
34. NYAYE	4/F	Emaciated	18	-ve	-ve	ntf			
35. WULE	4/F	Emaciated	15	-ve	-ve	"			
36. Not named	5/F	Good appearance	27	-ve	-ve	"			

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF DRY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN MANCHOK DISTRICT OF KAURA LOCAL GOVERNMENT AREA

	ANIMAL'SNAME	AGE(YEARS/SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
1	REDU	7/F	Good appearance	28	-ve	-ve	ntf			
2.	AUTE	10/F	Good appearance	30	-ve	-ve	"			
3	WUNE	8/F	Emaciated	29	+	-ve	"	+		
4	SAIGE	7/F	Emaciated	23	-ve	-ve	"			+
5	KORO	3/F	Good appearance	29	-ve	-ve	"	+++	+	++
6	YAKANA	10/F	Emaciated	-	-ve	-ve	"			
7	BEGUWA	2/F	Good appearance	32	-ve	-ve	"			
8	WULEI	7/F	Emaciated	27	-ve	-ve	"	+		+
9	NORE I	6/F	Good appearance	29	-ve	-ve	"		++	
10	NORE II	9/F	Good appearance	34	-ve	-ve	TC(1/F)			
11	BEBUA	9/F	Good appearance	25	++	-ve	"		+	
12	NORE II	6/M	Emaciated	20	-ve	-ve	m.			
13	KORO I	5/F	Good appearance	34	-ve	-ve	"	+		++
14	BALE	5/F	Emaciated	28	-ve	-ve	"			
15	KORO II	3/F	Good appearance	28	-ve	-ve	"	+	+	
16	BIGA	7/F	Good appearance	28	-ve	-ve	"			
17	KOROBALE	8/F	Emaciated	22	-ve	-ve	"			
18	BADDA	5/F	Good appearance	30	-ve	-ve	"	++	++	++

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF DRY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN BONDONG DISTRICT OF KAURA LOCAL GOVERNMENT AREA

	ANIMAL'SNAME	AGE(YEARS/ SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
1	SHINE	7/F	Emaciated	25	-ve	-ve	ntf			
2	SAYE	5/F	Emaciated	-	-ve	-ve	"			
3	WULE	6/F	Emaciated, Boophilus ticks	22	-ve	-ve	"	+		
4	MBULA	8/F	Emaciated, pale m/m	23	-ve	-ve	"	++		
5	SAJE	2/F	Good appearance	28	-ve	-ve	"			
6	WULE I	9/F	Emaciated	26	-ve	-ve	"			
7	LELA	5/F	Good appearance	33	-ve	-ve	"			++
8	HURE	5/F	Good appearance	25	-ve	-ve	"	+	+	+
9	DULE	2/M	Good appearance	30	-ve	-ve	"			
10	SHINGE	3/M	Good appearance	25	-ve	-ve	"			

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
.ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF DRY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN BONDONG DISTRICT OF KAURA LOCAL GOVERNMENT AREA

	ANIMAL'SNAME	AGE(YEARS/ SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	T
11	ZANE II	10/F	Recumbent, pale m/m	14	+++	Tb(3-5/F)	Tb(1-2/F)		
12	ZANE II	6/F	Good appearance	28	-ve	-ve	ntf		
13	BUNA	10/F	Emaciated,	26	-ve	-ve	"		
14	KURUMBE	8/F	Emaciated pale m/m	18	-ve	-ve	"		
15	BULA	6/F	Good appearance	29	-ve	-ve	"		
16	WODE I	7/F	Emaciated	26	-ve	-ve	"		
17	WODE II	1/F	Emaciated	29	-ve	-ve	"		
18	SHODA	4/M	Good appearance	33	-ve	-ve	"		
19	JAME	9/F	Emaciated	27	-ve	-ve	"		
20	NYELLE	6/F	Emaciated	25	-ve	-ve	"	++	

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF DRY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN FADAN KAGORO DISTRICT OF KAURA LOCAL GOVERNMENT AREA

	ANIMAL'SNAME	AGE(YEARS / SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S / Tb
1	TOKWAIRU	10/F	Emaciated, pale m/m	23	-ve	-ve	nts			
2	JANEI I	9/F	Emaciated,	23	-ve	-ve	"	+		+
3	JANEI II	2/F2/F	Emaciated,, general Alopecia	18	+	-ve	"		+	
4	JANEI II	2/F	Emaciated, weak	28	-ve	-ve	"			
5	WULE	12/F	Emaciated, lactating	21	-ve	-ve	"		+	+
6	RANE I	11/F	Emaciated, lactating	22	-ve	-ve	"			+
7	KABA	3/F	Good appearance	26	-ve	-ve	"		+	
8	DAKIL	3/M	Good appearance	26	-ve	-ve	"			
9	KABA II	8	Emaciated,	27	-ve	-ve	"		+	++
10	DAKE II	7/F	Emaciated,	22	-ve	-ve	"			
11	KONWA	7/F	Emaciated, Bookphilus Hyaloma ticks	25	-ve	-ve	"			
12	KRUMBEL	6.F	Emaciated, diarrhoea	24	-ve	-ve	"	+	+	++
13	WUNE I	9/F	Emaciated,	29	-ve	-ve	"	+++		+
14	MAGGA I	10/F	Emaciated, pale m/m	20	-ve	-vc	"			

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF DRY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN FADAN KAGORO DISTRICT OF KAURA LOCAL GOVERNMENT AREA

	ANIMAL'SNAME	AGE(YEARS / SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
15	WUNE	2/F	Emaciated, pale m/m	18	-ve	-ve	"			
16	MAGGA II	2/F	Good appearance	23	-ve	-ve	"			
17	LOSHE	12/F	Good appearance	30	-ve	-ve	"			
18	SAIGE	9/F	Good appearance	24	-ve	-ve	"			
19	BIGEL	10/F	Emaciated, pale m/m	14	-ve	-ve	"			
20	WULE	8/F	Emaciated	24	-ve	-ve	"			
21	FELLE	9/F	Emaciated, pale m/m	26	+	-ve	"			+
22	JITOIN	5/F	Emaciated, pale m/m	21	-ve	-ve	"	+	++	+
23	FARFARE	15/F	Emaciated, pale m/m	20	-ve	-ve	"			
24	WULE	7/F	Good appearance	23	-ve	-ve	"		+	
25	SAJE	6/F	Emaciated, pale m/m	24	-ve	-ve	"			
26	DAKE	8/F	Emaciated, pale m/m	24	-ve	-ve	"			
27	SARGE	6/F	Emaciated, pale m/m	22	-ve	-ve	"			
28	KOBAI	6/F	Emaciated, pale m/m	24	+	-ve	"	+		

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF DRY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN KUKUM DISTRICT OF KAURA LOCAL GOVERNMENT AREA

	ANIMAL'SNAME	AGE(YEARS / SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
1	BODERI	3/F	Stainted growth	28	+	-ve	ntf	+++		++
2	WODEI	4/F	Emaciated, pale m/m	21	-ve	-ve	"		+	++
3	YAKANA	5/F	Emaciated Ambloma ticks	22	-ve	-ve	"			+
4	KOROA	4/F	Good appearance	24	+	-ve				
5	WULE II	5/F	Emaciated	15	-ve	-ve	"			
6	WULE I	8/F	Emaciated	27	++	-ve			++	
7	TADADE	10/F	Good appearance	17	-ve	-ve	"			++
8	KODEI	6/F	Good appearance	26	-ve	-ve	"			+
9	NOJA	6/F	Emaciated	24	-ve	-ve	"			
10	BOJO	4/F	Good appearance	28	-ve	-ve	"			
11	KOROA II	4/F	Good appearance	26	-ve	-ve	"			
12	BALEI	9/F	Emaciated	21	-ve	-ve	"		++	
13	KOROA III	4/F	Emaciated	22	-ve	-ve	"	+	+	+
14	BALEI	6/F	Good appearance	23	+	-ve	"			
15	NOFA	6/F	Emaciated	20	-ve	-ve	"	++		++
16	MALLERI	5/F	Good appearance	23	-ve	-ve	"			++

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF DRY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN KADARKO DISTRICT OF KAURA LOCAL GOVERNMENT AREA

	ANIMAL'SNAME	AGE(YEARS / SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
1	NORE I	7/F	Emaciated	23	-ve	-ve	ntf			++
2	JAJO	8/F	Emaciated, Boophilus ticks	25	-ve	-ve	"	+		+
3	LOSHIA	8/F	Emaciated	-	-ve	-ve	"			
4	MALEI	7/F	Emaciated	17	-ve	Tb(0-1/F)	Tb(0-1/F)			+
5	GIDOMA	9/F	Emaciated	25	-ve	-ve	ntf			+
6	BALEI	6/F	Good appearance	10	-ve	-ve	"		+	
7	NORE II	3/F	Emaciated	23	-ve	-ve	"		+++	
8	BIGA	3/F	Emaciated	21	-ve	-ve	Tc(5-6/F)		+++	
9	WULE	8/F	Emaciated	23	++	-ve	"	+	++	++
10	HODEI	12/F	Emaciated	20	-ve	-ve	"		+	
11	NKWE	8/F	Emaciated, pale m/m	24	-ve	-ve	"		++	+
12	SHOUGE	10/F	Emaciated, pale m/m	20	-ve	-ve	"		++	++
13	MAKORI	3/F	Emaciated, pale m/m	20	-ve	Tb(0-1/F)			+	+
14	NYAYE	3/F	Emaciated, pale m/m	23	-ve	-ve	ntf			+
15	DAKE	15/F	Emaciated, pale m/m	22	+++	-ve	Tb(1-2/F)			+
16	MIYEIPEI	10/F	Emaciated, pale m/m	16	++	-ve	"			
17	WAGUMBE	8/F	Emaciated, pale m/m	10	-ve	-ve	"			

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF DRY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN KADARKO DISTRICT OF KAURA LOCAL GOVERNMENT AREA

ANIMAL'SNAME		AGE(YEARS/ SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
18	SAIGE	10/F	Emaciated, pale m/m	25	-ve	-ve	ntf	+		+
19	SHONGEL	10/M	Emaciated Boophilus	21	-ve	Tb(1-2/F)	"	+		
20	Not named		Emaciated, pale m/m	19	++	-ve	Tb(1-2/F)			++
21	HURE	10/M	Good appearance	20	+	-ve	ntf			
22	BODEJO	8/M	Emaciated, pale m/m	15	-ve	-ve	"	+	+++	
23	DAKE	3/F	Emaciated stainted growth	23	-ve	-ve	"			
24	BOLA	9/M	Good appearance, Boophilus	22	-ve	-ve	"			
25	HODE	8/F	Emaciated, pale m/m	15	++	Tc(0-1/F)	Tc(0-1/F)			+
26	NISAIGE	5/F	Emaciated, pale m/m	20	-ve	-ve	"			
27	MORE	7/F	Emaciated, pale m/m	29	-ve	-ve	ntf			
28	NOURU	7/F	Emaciated, pale m/m	23	-ve	-ve	"		++	
29	KOMA	6/F	Emaciated, pale m/m	23	-ve	-ve	"			
30	DULE	4/F	Emaciated, pale m/m	22	-ve	-ve	"	+		
31	NYAYE	4/F	Emaciated, pale m/m	22	-ve	-ve	"		+	
32	BALERI	4/M	Emaciated, ents soil	22	-ve	-ve	"		+	++
33	BAKORI	5/M	Emaciated, pale m/m	23	-ve	-ve	"			
34	BALEI	6/F	Emaciated, pale m/m	23	-ve	-ve	"	+	++	++

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF DRY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN KADARKO DISTRICT OF KAURA LOCAL GOVERNMENT AREA

ANIMAL'SNAME		AGE(YEARS/ SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
35	KOLJOMO	2/F	Good appearance	23	-ve	-ve	ntf			
36	FURE I	3/F	Good appearance	26	-ve	-ve	"			
37	SAGE II	5/F	Good appearance	27	-ve	-ve	"	+	+	
38	FURE II	7/F	Good appearance	24	-ve	-ve	"	++	+	
39	AMARE	7/F	Good appearance	28	-ve	-ve	"	+	+	
40	SAIGE	8/F	Good appearance	28	-ve	-ve	"			
41	FURE II	7/F	Good appearance	28	-ve	-ve	"			
42	DEREL	7/F	Good appearance	34	-ve	-ve	"			
43	DULEI	8/F	Emaciated, pale m/m	20	+	-ve	"			
44	SANGE	6/F	Emaciated, pregnant	25	-ve	-ve	"			
45	MASARE	9/F	Emaciated	24	++	-ve	Tc(0-1/F)		++	
46	BALE	3/F	Emaciated, pale m/m	17	-ve	-ve	ntf	+	++	++
47	DAWADO	7/F	Good appearance	31	++	-ve	"			
48	SAJE	7/F	Good appearance	25	-ve	-ve	"			+
49	GEDAL	6/F	Good appearance	30	++	-ve	"			
50	SAI	8/F	Emaciated	25	+	-ve	"			

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF RAINY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN ZANKAN DISTRICT OF KAURA LOCAL GOVERNMENT AREA

	ANIMAL'S NAME	AGE(YEARS / SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
1	WAJE	7/F	Good appearance	26	+	Tc(1-2/F)	Tc(o-1/F)	++	++	+++
2	DULE	9/F	Good appearance	26	++	Tb(1-2/F)	Tb(o-1/F)			++
3	YAKAMA	8/F	Emaciated, pale m/m	21	+	-ve	ntf			+
4	LULE	4/F	Good appearance	29	+	Tc(2-4/F)	Tc(2-3/F)	++	++	++
5	BODE	8/F	Good appearance	28	+	-ve	ntf			+
6	DOSHIE	9/F	Good appearance	29	+	-ve	"	+	+	+
7	FEDELI	7/F	Good appearance	23	+	-ve	"		++	++
8	LOSHEI	7/F	Good appearance	23	-ve	-ve	""	+		
9	DODEI I	9/F	Good appearance	21	-ve	-ve	"		++	++
10	MINGEL	7/F	Good appearance	31	-ve	-ve	"			
11	DIDANDE	4/F	Good appearance	22	-ve	-ve	"			
12	DAKE	3/F	Good appearance	23	-ve	-ve	"			
13	MORE I	4/F	Good appearance	29	-ve	-ve	"			
14	KODA	10/F	Emaciated, (old age)	27	-ve	-ve	"	++		++
15	LOSHIA	8/F	Good appearance	26	-ve	-ve				
16	SHODEI	2/F	Good appearance	27	-ve	-ve				
17	NYAYE	9/F	Emaciated, pale m/m	18	++	Tc(2-3/F)	Tc(1-2/F)		+	
18	MORE II	5/F	Good appearance	23	-ve	-ve	"		++	

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF RAINY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN ZANKAN DISTRICT OF KAURA LOCAL GOVERNMENT AREA

	ANIMAL'SNAME	AGE(YEARS/ SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L A Tv	I Tc		S A Tb
19	KOKORI	5/F	Good appearance	20	-ve	-ve	nts				+
20	DANGOGO	4/F	cassal appearance	31	-ve	-ve	"			++	
21	NYAYE	7/F	Emaciated, pale m/m	15	+	-ve	"			+	++
22	GDAGA	3/F	Emaciated, pale m m	17	-ve	-ve	"				
23	DOSHEI II	7/F	Good appearance	25	-ve	-ve	"				
24	MORE III	7/F	Emaciated, pale m/m	15	++	-ve	"	+		+++	+++
25	MORDI	6/F	Emaciated, pale m/m	15	++	-ve	"				+
26	DAUDE	6/F	Emaciated, pale m/m	15	+		Tc(1-2/F)			+	+
27	BOJO	8/F	Good appearance	33	-ve	-ve	ntf				
28	MUGEL	3/M	Emaciated	20	++	-ve	"				+
29	NGO	3/M	Emaciated	20	+	Tc(1-6/F)	"			++	+
30	NYAYE III	5/F	Emaciated, pale m/m	15	++		"				
31	MINGEL	8/F	Good appearance	23	-ve	Tc(o-1/F)	"			++	
32	NBULDI	5/F	Emaciated, pale m/m	22	++		"			++	
33	TUNKEL	10/F	Emaciated, pale m/m	21	+	-ve	"				
34	WAGE	11/F	Emaciated, pale m/m	18	++	-ve	"				
35	BODERI	4/F	Emaciated, pale m/m	20	++	-ve	"				+
36	Not named	10/F	Emaciated, pale m/m	22	+	-ve	"			+	+

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF RAINY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN FADAN KAGORO DISTRICT OF KAURA LOCAL GOVERNMENT AREA

	ANIMAL'SNAME	AGE(YEARS / SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
1	KOROA I	12/F	Good appearnce	23	-ve	-ve	ntf		+	
2	KOROA II	8/F	Emaciated pale, m/m	11	+++	Tb(3-6/F)	Tb/Tc2-4/F)			
3	NYAYE I	6/F	Emaciated pale, m/m	17	+	-ve	ntf			
4	SAYE II	6/F	Emaciated pale, m/m	12	+	-ve	"			
5	NYAYE II	9/F	Emaciated pale, m/m	18	-ve	-ve	"			
6	SAYE II	2/F	Emaciated pale, m/m	15	+++	Tb(3-7/F)	Tb(1-3/F)			
7	WULE I	7/F	Emaciated pale, m/m	20	-ve	-ve	ntf			
8	DAKE I	7/F	Emaciated pale, m/m	19	+	Tc(1-2/F)	"			++
9	BALE I	8/F	Emaciated, rough hair	19	++	-ve	"			
10	WULE II	7/F	Good appearnce	18	+	Tc(1/F)	"			++
11	NORE	4/F	Emaciated, Diarrhoea Tears	19	-ve	-ve	"		++	++
12	NYAYE III	6/F	Good appearnce	18	+	-ve	"	+		
13	DULEI	5/F	Emaciated	20	-ve	-ve	"		++	+
14	WAGE I	3/F	Emaciated pale, m/m	12	-ve	-ve	"	+	++	++
15	NYAYE IV	2/F	Emaciated pale, m/m	14	-ve	-ve	"	++	+	++
16	WULE III	1/M	Emaciated pale, m/m	16	+++	-ve	"			

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF RAINY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN FADAN KAGORO DISTRICT OF KAURA LOCAL GOVERNMENT AREA

ANIMAL'SNAME		AGE(YEARS/ SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
17	WAGE II	3/F	Emaciated pale, m/m	17	-ve	-ve	ntf			+
18	NYAURI I	3/F	Emaciated pale, m/m	15	+++	Tb(1-3/F)	Tb(o-2/F)		++	
19	DULE	7/F	Emaciated pale, m/m	20	++	Tb(o-1/F)	Tb(o-1/F)		+	++
20	WAGE	4/F	Emaciated pale, m/m	20	++	Tc/Tv(1-3)	Tc/Tv(o-2/F)	++	++	++
21	NYAYE V	7/F	Emaciated pale, m/m	11	-	clotted	ntf	+		+
22	YARE	7/F	Emaciated pale, m/m	16	+	-ve	"			++
23	DAKE	5/F	Emaciated pale, m/m	19	+	-ve	Tv(o-3/F)	++		
24	KOROA III	3/F	Emaciated pale, m/m	17	+	Tb(o-1/F)	ntf			++
25	MORE	1 /M	Emaciated pale, m/m	13	++	Tb(o-1/F)	Tv(o-3/F)	++	++	+++
26	SAYI I	4/F	Emaciated pale, m/m	20	+	-ve	ntf			+
27	BALE II	3/M	Emaciated pale, m/m	15	+++	Tb(2-3/F)	Tb(1-2/F)			
28	KOROA IV	7/F	Emaciated pale, m/m	19	++	Tv(2-3/F)	Tv(o-1/F)	+		
29	WAGE IV	6/M	Emaciated pale, m/m	16	-ve	-ve	ntf			
30	SAYI II	6month/m	Emaciated pale, m/m	18	-ve	-ve				
31	KOROA VI	6/m	Emaciated pale, m/m	20	++	Tb(1-2/F)	Tb(o-1/F)			++
32	BIDOMA	4/m	Emaciated pale, m/m	21	++	Tb(2-3/F)	Tb(o-1/F)			++

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF RAINY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN FADAN KAGORO DISTRICT OF KAURA LOCAL GOVERNMENT AREA

ANIMAL'SNAME		AGE(YEARS/ SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
33	NYAURI II	6/F	Emaciated, pale m/m	17	+	Tc(o-1/F)	ntf	+	++	++
34	YAREI	6/F	Good appearance	17	+	-ve	"			
35	DAKE	2/M	Emaciated, pale m/m	15	+++	Tc(1-2/F)	Tc(1-3/F)			
36	BALERI	5/M	Good appearance	19	-ve	-ve	ntf	+	+	
37	JEDDA	5/F	Emaciated, pale m/m	15	-ve	-ve	"			
38	DAKE	7/F	Good appearance	18	+	-ve	"			
39	YAREI	7/F	Emaciated	21	+	-ve	"	++	++	+++
40	MALERI	9/M	Emaciated, pale m/m	15	+	-ve	"			
41	MORDI	6 months/m	Good appearance	20	-ve	-ve	"			
42	KOROA VII	6/8	Emaciated, pale m/m	18	-ve	-ve	"			
43	NELLE	8/F	Good appearance	20	+	-ve	"	+		+
44	YEREI	6 months/F	Good appearance	21	+	-ve	"	+		

KEY:

M	=	Male	TC	=	Trypanosoma congolense
F	=	Female	TV	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

RESULTS OF RAINY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN MANCHOK DISTRICT OF KAURA LOCAL GOVERNMENT AREA

	ANIMAL'SNAME	AGE(YEARS/ SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
1	BALERI	15/M	Good appearance	17	-ve	-ve	ntf	+	+	+
2	NYAYE III	2F	Good appearance	20	-ve	-ve	"			
3	NYAYE II	8/M	Good appearance	18	+	-ve	"			
4	NOM	10/M	Emaciated, pale m/m	12	+++				+	++
5	NORE	7/M	Emaciated, pale m/m	13	-					++
6	NYAYE I	9/M	Good appearance	18	+				++	++
7	NORE II	2/F	Good appearance	22	++					+++
8	WULE	1/M	Emaciated, pale m/m	17	++					+++
9	BALE	7/F	Good appearance	20	-ve	-ve	ntf			
10	UBAYE	7 months/F	Emaciated, pale m/m	15	+++++			++		++
11	NORE	7 months/F	Emaciated, pale m/m	16	+++				++	
12	YAKAMA	5 months/F	Emaciated, pale m/m	26	-ve	-ve	ntf		++	
13	NARIA	5 months/M	Good appearance	28	-ve	-ve	"			
14	SEIGE	7 months/M	Good appearance	24	+	-ve	"			
15	WADE I	7 months/F	Emaciated, pale m/m	18	++++		"	+		++
16	WUNE	10/F	Good appearance	19	+	-ve	"	++	++	++
17	OBAE	8/F	Emaciated, pale m/m	20	-ve	-ve	"			
18	MALEI	10/F	Emaciated, pale m/m	21	-ve	-ve	"		+	+

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	TB	=	Trypanosoma brucei
ntf	=	no trypanosome found	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique.

RESULTS OF RAINY SEASON BOVINE TRYPANOSOMOSIS SURVEY IN MANCHOK DISTRICT OF KAURA LOCAL GOVERNMENT AREA

ANIMAL'S	NAME	AGE(YEARS/ SEX	PHYSICAL APPEARANCE	PCV	HCT	WBF	TS	E L Tv	I Tc	S A Tb
19	JIDOMA	8/F	Good appearance	21	-ve	-ve	ntf		++	++
20	BINDE	4/F	Good appearance	22	-ve	-ve	"			+
21	TUKUYE	10/F	Emaciated, pale m/m	16	-ve	-ve	"		++	++
22	WULE	9/F	Good appearance	28	-ve	-ve	"	++	+	++
23	BOJO	8/F	Emaciated, pale m/m	21	++	Tc(3-6/F)	Tc(1-3/F)		++	
24	BATURE	8/F	Emaciated. pale m/m	34	-ve	-ve	ntf			
25	MORE	10/F	Good appearance	30	-ve	-ve	"			
26	BOJO	2/F	Good appearance	31	-ve	-ve	"			
27	SAJE	3/F	Good appearance	25	+	-ve	"	+	++	
28	WULEI	11/M	Good appearance	30	-ve	-ve	"			
29	BALE	3/F	Good appearance	24	-ve	-ve	"	+++	++	+++
30	JEME	8/F	Good appearance	33	+	-ve	"			
31	MALLERI	8/M	Good appearance	25	-ve	-ve	"		++	++
32	BUNEIR	3/M	Good appearance	30	-ve	-ve	"			
33	WINE	6/F	Emaciated	24	-ve	-ve	"	+	+	
34	YAKAMA	6/F	Good appearance	23	-ve	-ve	"	++		
35	not named	7?F	Good appearance	29	-ve	-ve	"	+		

KEY:

M	=	Male	Tc	=	Trypanosoma congolense
F	=	Female	Tv	=	Trypanosoma vivax
mm	=	Mucous membrane	Tb	=	Trypanosoma brucei
ntf	=	no trypanosome foun :	Pcv	=	Packed cell volume
-ve	=	negative	WBF	=	wet blood film
+	=	positive	TS	=	Thin smear
HCT	=	Haematocrit centrifugation	ELISA	=	Enzyme - linked immunosorbent assay technique

GOAT NO 287

(T. brucei infected: Donor goat)

Week	HCT	BT(^{0}C)	PCV(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3/mm^3)	B/W(Kg)
(DONOR)							
1	NPF	38	25	8.0	5.73	24.8	13
2.	NPF	38	25	8.0	5.73	24.8	10.0
3.	+	38.5	19.7	6.5	3.2	20.6	10.0
4.	++++	40.1	16.5	5.3	3.1	20.3	9.0
5	++++	39.4	15.0	4.5	2.5	17.3	9.0
6.	Dead						

KEY:

NPF = NO PARASITE FOUND

= Trypanosome inoculation

BT(^{0}c) = Body temperature in degrees centrigrade

Pcv(%) = Packed cell volume in percentage

Hb(g/100cm^3) = Haemoglobing/concentration in grams per 100 cubic centimeter

RBC(10^6/mm^3) = Red blood count per cubic milimeter

WBC(10^3 /mm^3) = White blood count per cubic milimeter

B/W(kg) = Body Weight in Kilograms

\+ = degree of parasitaemia

GOAT NO. 286

(T. brucei infected: Berenil treated)

Week	HCT	BT(^{0}c)	PCV(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3/mm^3)	B/W(kg)
1.	NPF	37.8	28.5	9.7	4.8	18.5	11
2	NPF	37.9	26.5	9.8	4.5	17.9	10.0
3	NPF	38.6	25.5	7.7	3.7	17.9	9.0
4	++	38.7	19.0	7.5	2.9	19	9.0
5	NPF	39	21	5.2	3.0	21.4	8.5
6	NPF	38.8	21.0	5.0	2.7	21.3	9.0
7	NPF	38	21	6.3	4.1	20.3	9
8	NPF	38.2	24.3	7.0	4.3	19	9
9	NPF	38.5	22.6	7.1	3.7	19.7	10
10	NPF	38.3	25.0	7.5	4.0	19.0	10
11	NPF	38.2	29.7	8.0	5.3	19.0	10
12	NPF	38.3	28.5	ND	ND	ND	10
13	NPF	38	26	ND	ND	ND	10
14	NPF	38.4	24	ND	ND	ND	10
15	NPF	38.3	23	7.5	4.9	20.0	10

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
----------	=	Trypanosome inoculation
---------	=	Trypanocide treatment
BT(^{0}c)	=	Body temperature in degrees centigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm^3)	=	Haemoglobing in grams per 100 cubic centimeter
RBC(10^6/mm^3)	=	Red blood count per cubic milimeter
WBC(10^3 /mm^3)	=	White blood count per cubic milimeter
B/W(kg)	=	Body Weight in Kilograms
+	=	degree of parasitaemia

GOAT NO. 1687

(T. brucei infected: Berenil treated)

Week	HCT	BT(^{0}c)	Pcv(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3/mm^3)	B/W(kg)
1	NPF	37.3	21.5	6.6	3.9	20.9	9.0
2	NPF	38	21.0	6.3	3.2	16.9	11
3	NPF	38.5	23.7	8.0	4.8	25.9	7.5
4	++	38.6	20.6	6.0	3.8	22.3	9.0
5	NPF	38.2	17.3	5.7	3.7	20	8
6	NPF	38.6	22	5.3	2.9	21.1	9
7	NPF	38.5	25.7	5.7	3.6	19.7	8
8	NPF	38.5	23	7	4.3	20	8
9	NPF	38.4	23.7	8.1	4.0	20.5	8
10	NPF	38.8	28.3	9.0	5.0	21.0	9
11	NPF	38.9	26.7	8.5	4.5	20.6	9
12	NPF	38.5	30	ND	ND	ND	10
13	NPF	38.0	25.7	6.8	2.2	27.0	10
14	NPF	38.5	25	ND	ND	ND	10
15	NPF	38.4	35	10.1	6.7	24.0	10

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
----------	=	Trypanosome inoculation
---------	=	Trypanocide treatment
BT(^{0}c)	=	Body temperature in degrees centigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm^3)	=	Haemoglobing concentration in grams per 100 cubic centimeter
RBC(10^6/mm^3)	=	Red blood count per cubic milimeter
WBC(10^3 /mm^3)	=	White blood count per cubic milimeter
B/W(kg)	=	Body Weight in Kilograms
+	=	degree of parasitaemia

GOAT NO. 289

(T. brucei infected: Berenil treated)

Week	HCT	BT(^{0}c)	Pcv(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC10^3/mm^3)	B/W(kg)
1	NPF	38	27.5	9.2	4.8	29.3	10
2	NPF	37.8	27.0	9.8	4.6	31.8	10
3	NPF	38.6	24.7	7.5	4.6	20.4	9.5
4	++	38	30	7.5	4.0	19.8	12.0
5	NPF	38.7	26	8.3	4.9	18.4	8.50
6	NPF	35.5	27.7	7.3	4.0	18.1	9
7	NPF	38.3	30.7	8.0	4.0	19.8	9
8	NPF	38.5	27.7	7.3	4.3	19	9
9	NPF	38	26.3	8.6	4.9	20	10
10	NPF	38.4	25	8.2	4.5	20.1	10
11	NPF	38.5	24	7.8	4.2	20.0	10
12	NPF	38.6	24	ND	ND	ND	10
13	NPF	38.3	24.0	7.8	2.8	26.8	10
14	NPF	ND	ND	ND	ND	ND	ND
15	NPF	38.3	25	7.4	4.8	25.7	10

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
----------	=	Trypanosome inoculation
---------	=	Trypanocide treatment
BT(^{0}c)	=	Body temperature in degrees centrigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm^3)	=	Haemoglobing in grams per 100 cubic centimeter
RBC(10^6/mm^3)	=	Red blood count per square milimeter
WBC(10^3 /mm^3)	=	White blood count per square milimeter
B/W(kg)	=	Body Weight in Kilograms
+	=	degree of parasitaemia

GOAT NO. 1650

(T. brucei infected: Samorin treated)

Week	HCT	BT(^{0}c)	Pcv(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3/mm^3)	B/W(kg)
1	NPF	35.5	29	9.2	4.4	15.6	10
2	NPF	38.1	28	10	5.0	16.0	10
3	NPF	39.1	28	8.9	4.8	17.1	10
4	++	39.5	21	6.0	3.8	19.1	10
5	NPF	39.3	17.7	6	3.3	14.8	9
6	NPF	38.5	16.3	5	2.8	18	9
7	NPF	38.9	21	4.9	3.0	18.6	9
8	NPF	38.6	24	6	3	19.8	10
9	NPF	38.3	26	7.1	4.7	20.2	10
10	NPF	38.3	24.3	7.3	4.0	20.1	11
11	NPF	38.3	27	8.0	4.1	20.2	11
12	NPF	38	27.6	ND	ND	ND	11
13	NPF	38.2	26.3	ND	ND	ND	11
14	NPF	38.4	23	ND	ND	ND	11
15	NPF	38.3	25.0	6.5	4.0	26.0	11

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
----------	=	Trypanosome inoculation
---------	=	Trypanocide treatment
BT(^{0}c)	=	Body temperature in degrees centigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm^3)	=	Haemoglobing concentration in grams per 100 cubic centimeter
RBC(10^6/mm^3)	=	Red blood count per cubic milimeter
WBC(10^3 /mm^3)	=	White blood count per cubic milimeter
B/W(kg)	=	Body Weight in Kilograms
+	=	degree of parasitaemia

GOAT NO. 1686

(T. brucei infected: Samorin treated)

Week	HCT	BT(^{0}c)	Pcv(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3/mm^3)	B/W(kg)
1	NPF	38.9	27	7.9	6.0	20.8	9
2	NPF	38.6	29	6.3	3.8	17.8	10
3	NPF	38.3	28	5.6	4.3	17.6	9
4	++	39.3	21.4	5.5	3.2	70.1	10
5	NPF	38.9	20.6	6.4	3.9	23.6	9
6	NPF	38.3	21.7	6	3.6	20.3	9
7	NPF	38.1	25	5.6	3.2	19	9
8	NPF	38.4	23.3	6.3	3.8	20.1	10
9	NPF	38.2	23.7	7.7	4.2	20.2	10
10	NPF	38.7	22.7	6.8	3.5	20.2	11
11	NPF	38.2	26	6.9	3.6	20.1	11
12	NPF	39	24.5	ND	ND	ND	11
13	NPF	39	25.3	6.0	2.0	17.5	11
14	NPF	38.5	24.5	ND	ND	ND	11
15	NPF	38.0	25.0	7.0	3.5	28	11

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
----------	=	Trypanosome inoculation
---------	=	Trypanocide treatment
BT(^{0}c)	=	Body temperature in degrees centigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm^3)	=	Haemoglobing in grams per 100 cubic centimeter
RBC(10^6/mm^3)	=	Red blood count per cubic milimeter
WBC(10^3/mm^3)	=	White blood count per cubic milimeter
B/W(kg)	=	Body Weight in Kilograms
+	=	degree of parasitaemia

GOAT NO. 257

(T. brucei infected: Samorin treated)

Week	HCT	BT(^{0}c)	Pcv(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3/mm^3)	B/W(kg)
1	NPF	37.8	30	10.4	5.3	18.3	9
2	NPF	38.5	26	8.8	5.2	19.8	9
3	NPF	38.5	28.5	8.2	4.5	19	9.5
4	++	38.6	29.0	6.0	4.1	20.3	10
5	NPF	38.4	26.3	7.2	4.5	19.3	9.5
6	NPF	38.8	24.5	5	3	19.8	10
7	NPF	39	32	10	6.3	18.9	9
8	NPF	38.8	32	10	5.8	20	11
9	NPF	38.7	36	10.4	5.0	20.4	12
10	NPF	38.5	38	10.3	5.2	20.3	12
11	NPF	38	39.5	10.5	5.3	20.1	12
12	NPF	38.5	40.5	ND	ND	ND	13
13	NPF	38.6	43.3	10.4	3.5	26.1	13
14	NPF	38.7	41	ND	ND	ND	13
15	NPF	38.5	42.0	10.4	5.0	27	13

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
----------	=	Trypanosome inoculation
---------	=	Trypanocide treatment
BT(C)	=	Body temperature in degrees centigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm)	=	Haemoglobing in grams per 100 cubic centimeter
RBC(10/mm)	=	Red blood count per cubic milimeter
WBC(10 /mm)	=	White blood count per cubic milimeter
B/W(Kg)	=	Body Weight in Kilograms
+	=	degree of parasitaemia

GOAT NO. 272

(T. Congolense infected: Donor goat)

Week	HCT	BT(^{0}c)	Pcv(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3 /mm3)	B/W(kg)
1	NPF	38.4	29	9	5.0	19.8	8
2	NPF	38.7	31.3	10.5	5.8	32.2	9.5
3	NPF	38.4	28.0	9.3	4.8	34	10
4	+	39.2	20.0	5.4	3.1	38.9	10
5	+++	40	15	4.0	2.4	41	9
6	DEAD						

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
---------	=	Trypanosome inoculation
BT(^{0}c)	=	Body temperature in degrees centigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm^3)	=	Haemoglobing in grams per 100 cubic centimeter
RBC(10^6/mm^3)	=	Red blood count per cubic milimeter
WBC(10^3/mm^3)	=	White blood count per cubic milimeter
B/W(kg)	=	Body Weight in Kilograms
+	=	degree of parasitaemia

GOAT NO. 261

(T. congolense infected: Berenil treated)

Week	HCT	BT(^{0}c)	Pcv(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3/mm^3)	B/W(kg)
1	NPF	38.3	29	10	4.9	26.9	11
2	NPF	38.6	28.8	10	4.9	26.2	11
3	NPF	28.6	26.3	9.3	4.5	27	10
4	NPF	38.9	24	8.0	5.0	27.3	11
5	NPF	38.5	26.5	9.3	4.6	27.9	10
6	+++	38.9	26.3	9.2	5.3	27	9
7	NPF	38.5	20.8	6.5	3.4	27.5	8
8	NPF	38.0	25.7	7.3	4.1	273	9
9	++	39	18	6.2	3.5	26.8	8
10	NPF	38.4	20	5.6	3.1	26.3	8
11	NPF	39.0	22	9.5	5.1	26.4	7
12	++	40	17	5.0	3.1	25.9	7
13	NPF	DEAD					

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
----------	=	Trypanosome inoculation
---------	=	Trypanocide treatment
BT(^{0}c)	=	Body temperature in degrees centigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm^3)	=	Haemoglobing in grams per 100 cubic centimeter
RBC(10^6/mm^3)	=	Red blood count per cubic milimeter
WBC(10^3 /mm^3)	=	White blood count per cubic milimeter
B/W(kg)	=	Body Weight in Kilograms
+	=	degree of parasitaemia

GOAT NO. 255

(T. Congolense infected: Berenil treated)

Week	HCT	BT(^{0}c)	Pcv(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3 /mm^3)	B/W(kg)
1	NPF	38.4	29	8.5	4.7	20.8	10
2	NPF	38.7	22.2	7.1	4.3	22.3	11
3	NPF	38.1	25	7.3	4.6	23.5	11
4	NPF	38.7	24.5	7.1	4.0	23.5	11
5	NPF	38.5	29.5	10.7	5.0	19.9	11
6	++	39.5	24.7	7.2	3.8	20.5	10
7	NPF	38	20.5	6.3	2.5	21.0	10
8	NPF	38.4	20.7	6.5	3.1	21.7	10
9	NPF	DEAD					

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
----------	=	Trypanosome inoculation
---------	=	Trypanocide treatment
BT(^{0}c)	=	Body temperature in degrees centigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm^3)	=	Haemoglobing in grams per 100 cubic centimeter
RBC(10^6/mm^3)	=	Red blood count per cubic milimeter
WBC(10^3 /mm^3)	=	White blood count per cubic milimeter
B/W(kg)	=	Body Weight in Kilograms
+	=	degree of parasitaemia

GOAT NO. 299

(T. Congolense infected: Proposed for Berenil treated)

Week	HCT	BT(^{0}c)	Pcv(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3/mm^3)	B/W(kg)
1	NPF	38.8	33.7	11.3	5.5	35.9	9.5
2	NPF	39	31.3	10.7	5.5	37.3	10
3	NPF	38.6	34.3	9.6	5.3	34.3	11
4	NPF	38.7	30	9.3	5.2	33	10
5	NPF	38.4	34.0	9.8	5.1	33	10
6	+++	DEAD					

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
----------	=	Trypanosome inoculation
BT( )	=	Body temperature in degrees centigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm^3)	=	Haemoglobing in grams per 100 cubic centimeter
RBC(10^6/mm^3)	=	Red blood count per cubic milimeter
WBC(10^3/mm^3)	=	White blood count per cubic milimeter
B/W(kg)	=	Body Weight in Kilograms
+	=	degree of parasitaemia

GOAT NO. 287

(T. Congolense infected: Samorin(R) treated)

Week	HCT	BT(0c)	Pcv(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3 /mm^3)	B/W(kg)
1	NPF	37.8	32.7	10.5	5.3	34	11
2	NPF	38.5	30.3	10.3	5.1	34.8	0
3	NPF	38.3	23.3	9.5	4.4	32.7	10
4	NPF	38	23.0	9.5	4.4	32.7	10
5	NPF	38.4	24.0	9.6	4.9	32.9	10
6	++	39.4	19	8.9	3.4	36	9
7	NPF	39	19.3	8.5	4.1	35	10
8	NPF	38.2	24.3	9.3	4.5	32	11
9	NPF	38.2	24.7	9.7	4.4	30	11
10	NPF	38.5	24	8.9	4.2	31	9
11	NPF	38.6	23.5	7.5	3.6	30	9
12	NPF	38.1	22	7.0	3.7	30	9
13	NPF	ND	19	6.5	2.9	29	8
14	NPF	ND	15	5.3	2.5	29	7
15	NPF	38	20	6.0	2.7	30	6

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
---	=	Trypanosome inoculation
---	=	Trypanocide treatment
BT(^{0}c)	=	Body temperature in degrees centigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm^3)	=	Haemoglobing in grams per 100 cubic centimeter
RBC(10^6/mm^3)	=	Red blood count per cubic milimeter
WBC(10^3 /mm^3)	=	White blood count per cubic milimeter
B/W(kg)	=	Body Weight in Kilograms
+		degree of parasitaemia

GOAT NO. 294

(T. Congolense infected: Samorin treated)

Week	HCT	BT(^{0}c)	Pcv(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3/mm^3)	B/W(kg)
1	NPF	39.6	25.3	8.0	3.5	27.3	10
2	NPF	38.7	25	8.2	3.1	29.1	9
3	NPF	38.2	29.3	8.8	5.0	28.7	9
4	NPF	38.2	28.2	28.0	5.0	29.5	11
5	NPF	38.7	34.5	8.8	5.0	28	12
6	++	39	25	7.8	4.5	28	11
7	NPF	38.3	21.3	7.0	3.9	29	11
8	NPF	38.1	24.7	7.7	3.5	28.5	11
9	NPF	38.6	18.7	5.6	2.8	28.2	10
10	NPF	38.2	23.0	6.2	3.4	27.5	9
11	NPF	38.4	20.5	5.7	3.3	27.3	10
12	NPF	38.1	20.7	5.6	3.3	27.7	9
13	NPF	ND	17.5	5.0	3.1	27	8
14	NPF	ND	16.5	4.6	7.9	27.2	7
15	NPF	39	16	4.5	2.3	27.4	6

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
---------	=	Trypanosome inoculation
---------	=	Trypanocide treatment
BT(^{0}c)	=	Body temperature in degrees centigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm^3)	=	Haemoglobing in grams per 100 cubic centimeter
RBC(10^6/mm^3)	=	Red blood count per cubic milimeter
WBC(100^3 /mm^3)	=	White blood count per cubic milimeter
B/W(kg)	=	Body Weight in Kilograms
+	=	degree of parasitaemia

GOAT NO. 293

(T. Congolense infected: Samorin treated)

Week	HCT	BT(^{0}c)	Pcv(%)	Hb(g/100cm^3)	RBC(10^6/mm^3)	WBC(10^3/mm^3)	B/W(kg)
1	NPF	39.3	38.0	12.6	6.2	18.8	12
2	NPF	38.3	36.6	12.3	6.1	21	12
3	NPF	38.4	34.3	7.9	9.1	21	11
4	NPF	38.6	38	7.9	3.1	21	18
5	NPF	38.4	36.5	8.5	4.2	22	12
6	++	38.1	31.3	8.0	4.3	25	12
7	NPF	38.5	36.9	9.5	4.8	25	11
8	NPF	38	35	8.6	4.2	23	11
9	NPF	38.2	30	7.8	3.6	23	11
10	NPF	38.5	30.7	7.5	3.8	22	11
11	NPF	38.7	25.0	6.3	2.1	21	11
12	NPF	38.3	21.3	5.6	2.9	21	11
13	NPF	38.3	21.3	5.6	2.9	21	10

KEY:

NPF	=	NO PARASITE FOUND
ND	=	NOT DONE
---------	=	Trypanosome inoculation
---------	=	Trypanocide treatment
BT(^{0}c)	=	Body temperature in degrees centigrade
Pcv(%)	=	Packed cell volume in percentage
Hb(g/100cm^3)	=	Haemoglobing in grams per 100 cubic centimeter
RBC(10^6/mm^3)	=	Red blood count per cubic milimeter
WBC(10^3/mm^3)	=	White blood count per cubic milimeter
B/W(kg)	=	Body Weight in Kilograms
+	=	degree of parasitaemia

ANTIGEN E.L.I.S.A. OPTICAL DENSITIES (0. Ds) FOR SERA TRYPANOSOMA CONGOLENSE TREATED AND TRYPANOCIDE TREATED GOATS

	WEEK	1	↓ 2	3	4	5	6	7	8	9	10	11	12	13	13
DONOR GOAT Berenil Treated infected goats	++272	0.096	0.096	0.180	0.188	0.295 ↓	DEAD ↑								
	++299	0.117	0.115	0.112	0.114	0.115	0.234	DEAD							
	++261	0.159	0.110	0.130	0.134	0.183	0.215	0.146	0.128	0.229	0.146	0.106	0.205	DEAD	
	++255	0.118	0.117	0.116	0.140	0.138	0.225	0.135	0.126	DEAD					
AVERAGE 0.Ds		0.115	0.114	0.119	0.129	0.141	0.225	0.140	0.127	0.229	0.146	0.106	0.205		
Samorin® Treated Infected Goats	++233	0.099	0.118	0.098	0.122	0.119	0.292	0.155	0.127	0.120	0.135	0.130	0.125	0.128	DEAD
	++294	0.102	0.114	0.135	0.153	0.118	0.251	0.142	0.119	0.115	0.125	0.134	0.145	0.123	0.135
	++287	0.113	0.109	0.115	0.140	0.117	0.299	0.168	0.105	0.112	0.109	0.110	0.111	0.107	0.096
AVERAGE 0.Ds		0.105	0.114	0.116	0.118	0.118	0.277	0.155	0.117	0.116	0.123	0.125	0.127	0.119	0.113

↓ = TRYPANOSOME INOCULATION

↑ = TRYPANOSOCIDE TREATMENT