THE ROLE OF BABESIOSIS AND TRYPANOSOMOSIS ON THE POOR PERFORMANCE OF POLO HORSES IN TWO LOCAL GOVERNMENT AREAS OF KADUNA STATE, NIGERIA

BY

SAMDI, MUSA SAMSON
MSc/VET-MED/48550/2005-2006

A THESIS SUBMITTED TO THE POSTGRADUATE SCHOOL AHMADU BELLO UNIVERSITY IN PARTIAL FULFILLMENT OF THE REQUIMENTS FOR THE AWARD OF THE DEGRE OF MASTER OF SCIENCE IN VETERINARY MEDICINE

DEPARTMENT OF VETERINARY SURGERY AND MEDICINE,
FACULTY OF VETERINARY MEDICINE,
AHMADU BELLO UNIVERSITY,
ZARIA, NIGERIA

FEBRUARY, 2011

DECLARATION

I declare that the work in the thesis entitled: **"The Role of Babesiosis and Trypanosomosis on the Poor Performance of Polo Horses in Two Local Goverment Areas of Kaduna state, Nigeria"** has been performed by me in the Department of Veterinary Surgery and Medicine under the supervision of Prof. U.S. Abullahi, Prof. A.K.B. Sackey and Prof. K.A.N. Esievo.

The information derived from the literature has been duly acknowledged in the text and a list of references provided. No part of thesis was previously presented for another degree or diploma at any university.

_____ _____

Samdi, Musa Samson **Date**

CERTIFICATION

This thesis entitled **"THE ROLE OF BABESIOSIS AND TRYPANOSOMOSIS ON THE POOR PERFORMANCE OF POLO HORSES IN TWO LOCAL GOVERMENT AREAS OF KADUNA STATE, NIGERIA"** by Samdi, Musa Samson, meets the regulation governing the award of the degree of Master of Science of Ahmadu Bello University and is approved for its contribution to knowledge and literary presentation.

_____ _____

Prof. U.S. Abdullahi Date
Chairman, Supervisory Committee

_____ _____

Prof. A.K.B. Sackey Date
Member, Supervisory Committee

_____ _____

Prof. K.A.N. Esievo Date
Member, Supervisory Committee

_____ _____

Prof. A.K.B. Sackey Date
Head, Department, of Veterinary Surgery & Medicine
Ahmadu Bello University, Zaria

_____ _____

Prof A.A. Joshua Date
Dean Postgraduate School
Ahamdu Bello University, Zaria

DEDICATION

This work is dedicated to the Samdi family for their prayers and financial support to me.

ACKNOWLEDGEMENT

My unquantifiable gratitude goes to Almighty God who has given me life and resources to do this work. I also sincerely appreciate the contributions of my team of supervisors viz: Prof. U.S. Abdullahi (Chairman) Prof. A.K.B. Sackey. (Member) and Prof. K.A.N. Esievo (Member) who were dedicated in giving me the required supervision, guidance and moral encouragement through the various stages of this research work.

I am grateful to the Director General of Nigerian Institute of Trypanosomiasis Research and my Head of Department, for providing space and equipment in the department's laboratories for me to conduct this research work. I also thank the technical staff of Nigerian Institute of Trypanosomiasis Research consultancy services unit for their guidance during the laboratory procedures.

Also appreciated is the contribution of Mr. Tiam Emmanuel of Chemical Pathology laboratory, Ahmadu Belo University Teaching Hospital Shika, Zaria, for helping in running the Enzyme Immunosorbent Assay cortisol. My profound gratitude also goes to the Data processing Unit, Institute of Agricultural Research (IAR), A.B.U. Zaria, for statistical analysis of my data. Also acknowledged are Fift Chucker and Kaduna Polo Clubs for granting me permission to sample in Kaduna North and Igabi local goverment areas of Kaduna State. The participation and linkage with the authorities by Drs Kwanashie, Garba Musa and Mustapha is higly appreciated. I seek God's blessings for those who contributed in various ways to making this study successful, Amen.

ABSTRACT

A total of 192 polo horses from the polo club, private stables and public stables were sampled from the two local government areas of Kaduna state.The parasiteamia was ganerally low in all the cases of Babesiosis and Trypanosomosis recorded. The clinical signs observed in Babesia infected polo horses from two local government areas included fever (36%), congested mucous membrane (61%), emaciation (63%), weakness(47%), colic (11%), ocular discharge (69%), pale mucous membrane (27%), oedema of the ventral region and extremeties (58%), nasal discharge (80%) and Icterus (44%) for Babesiosis. The clinical signs observed in Trypanosome infected polo horses from the two local government areas included fever (66.7%), congested mucous membrane (83.3%), emaciation(50%), weakness (33%), oedema of the prepuce/scrotal region and extremeties (50%). The overall prevalence of *Babesia* infections in polo horses was 18.75%. Two species of *Babesia* parasites were identified in single infections namely *Babesia equi* (66.67%) and *B. caballi* (33.33%). The *Babesia* infection rate in Igabi 15.62%, (15 cases) was lower than the *Babesia* infection rate in Kaduna north 21.87%, (21cases). The overall prevalence of *Trypanosome* infected polo horses was 3.13%. Two species of *Trypanosoma* parasites were identified in single infections namely *Trypanosoma evansi,* (83.33%) and *T. congolense,* (16.67%). The t*rypanosome* infection rate in Igabi (2.08%) was lower than the *Trypanosoma* infection rate in Kaduna north (6.25%). Overall the mean live weight, body condition score, packed cell volume, red cell count, hemoglobin concentration, white blood cell count, total protein, cortisol and differencial leucocytes counts of *babesia* and *trypanosome* positive groups differ significantly ($P<0.05$) from their negative groups.

TABLE OF CONTENTS

CHAPTER 3: MATERIALS AND METHODS

CHAPTER 4: RESULTS

LIST OF TABLES

LIST OF FIGURES

LIST OF PLATES

LIST OF APPENDICES

ABBREVIATIONS

AAT	:	African Animal Trypanosomosis
BC	:	Before Christ
BCM	:	Buffy Coat Method
^{o}C	:	Degrees Celsius
CFT	:	Complement Fixation Test
DIC	:	Disseminated Intravascular Coagulation
ELISA	:	Enzyme Linked Immunosorbent Assay
EIA	:	Equine infectious anemia
EDTA-K:		Ethylene di-amine tetra-acetate-k
GFR	:	Glomerular Filtration Rate
HCT	:	Hematocrit Centrifugation Technique
IFAT	:	Indirect Fluorescent Antibody Test
IP	:	Incubation Period
LGAs	:	Local Government Areas
LD50	:	Lethal dose
NITR	:	Nigerian Institute for Trypanosomiasis Research

NPA : Nigerian Polo Association

PCV : Packed cell volume

PCR : Polymerase Chain Reaction

RWAFF: Royal West African Frontier Force

Spp : Species

TP : Total protein

VF : Very Few

WBA : Western Blot Analysis

CHAPTER 1

INTRODUCTION

1.1 POLO

Polo is thought to have originated from China and Persia about 2000 years ago and was only recently introduced into Nigeria.The International Olympic Committee has recognised it as a form of sport (Ascort, 2008). Several states in Nigeria have public and privately owned polo clubs or resorts that are responsible for the organisation of polo tournaments under the supervision of the National body, the Nigerian Polo Association (NPA).

1.2 HORSES IN NORTHERN STATES OF NIGERIA

Horses are kept by the traditional councils, polo patrons/players, herdsmen, riding clubs, the military and the police. The royalists and economically privileged, keep horses more than the lower social classes because of the high costs of acquisition and maintenance. Uses of the horse by most owners include durbar (ceremonial carnival of horses), defense and security, for work (draught animals), leisure sport (polo and racing), hunting, livestock herd control and as a symbol of wealth for the political class (Garba, 2006). Maximum performance is expected from these horses irrespective of the reason for which they are kept. However, diseases are known to hamper their maximum performance and productivity. The horse has gained additional importance due to an increase in local and international trade value. Several breeds of horses are now found in Nigeria; from the exotic breeds (Argentine, South African), to indigenous (Arewa, Sudanese) breeds. The world population of horses is aproximated at 58 million (OIE, 1997) with 950,000 horses in Nigeria (ILCA,1993) while the equine population in Kaduna state is estimated at 4,000 horses.

1.3 HEALTH PROBLEMS OF THE HORSE

Horses are known to suffer from infectious diseases, among which are babesiosis and trypanosomosis. Babesiosis of horses is reported to be the third most important health problem of horses around Zaria (prevalence of 10.6%) after tick infestation and lameness (Abdulkadir, 1994). Equine trypanosomosis and other equine diseases have not been fully documented and even though Hill and Akpokodje (1971) and Dipeolu and Oduye (1977) reported the presence of *Trypanosoma vivax, T. congolense* and *T. brucei* in flies feeding on equine spacies and equine waste in Ibadan Nigeria.

Babesiosis is an acute, sub acute or chronic hemolytic disease caused by intra erythrocytic protozoans, *Babesia equi* and *Babesia caballi* (Friedhoff *et al*., 1990). The disease is enzootic in most tropical and sub tropical regions of the world (Uilenberg, 1995). Infection occurs in horses, donkeys, zebras, and mules. The incidence of the disease has also been closely tied to the geographical distribution and seasonal activity of its biological vector, ticks (Russel *et al*., 2005).

Equine trypanosomosis is a complex, acute, subacute or chronic, hemolytic disease caused by hemoflagellated protozoans, *Trypanosoma vivax, T.congolense, T. brucei brucei* and *T. evansi.* It is also known as nagana and surra, dourine (Radostits *et al*., 2000; Gina *et al*., 2008). The infection occurs in horses, donkeys, zebras and mules (Dethie *et al*., 2001), and the incidence of the disease is closely related with the distribution of tsetseflies and mechanical transmitters (Seifert, 1996).

1.4 ECONOMIC IMPORTANCE OF BABESIOSIS AND TRYPANOSOMOSIS

Babesiosis, Theileriosis and Trypanosomosis cause major losses in the livestock industry in Nigeria (Preston, 2001; Uilenberg, 2001; Abenga *et al*., 2002). Equine Piroplasmosis is a disease of serious concern militating against increased international trade of horses. Infected foals could die within 24 hours (Metcalf, 2001) and the development of carrier status has

caused the disease to persist in equine populations (Radostits *et al.*, 2000). Trypanosomosis is threatening to health and productivity of cattle and horses through reduced productivity, reduced fertility, abortion and deaths in sub-Saharan Africa (Young *et al.*, 1988; Radostits *et al*., 2000).

1.5 AIMS OF THE STUDY

The aims of the study is to investigate the role of babesiosis and trypanosomosis on the poor performance of polo horses in Kaduna state.

1.6 OBJECTIVES OF THE STUDY

The objectives of the study were to determine:

1. The prevalence of babesiosis and trypanosomosis and the of *Babesia* and *Trypanosoma* species involved in causing these diseases.
2. The weight loss, physical condition and clinical signs commonly associated with babesiosis and trypanosomosis infection in polo horses.
3. The effect of babesiosis and trypanosomosis infections on the level of serum cortisol and the hematological parameter in Polo horses in Kaduna north and Igabi local goverment areas of Kaduna State.

1.7 JUSTIFICATION OF THE STUDY

Horses are historically and functionally important animals in Nigeria especially in the traditional institutions of the northern states. Horses also play important roles in animal traction for increase crop production and transportation in several parts of the northern states. The army and the police have mounted troops which are use during parades. Horses are also involved in sporting activities such as polo and racing. Polo, for example, has been

recognised by the International Olympic Committee as a form of important sport thus justifying an increase in the rate of horse importation in Nigeria.

However,equine babesiosis and trypanosomosis constitute a serious health problem in horses, causing reduced performance of horses (Annebearson *et al.*, 1996) in Nigeria. The current scanty information on the effect of these diseases on the horse industry is an important obstacle to instituting an effective control. The result of investigation on the effect of babesiosis and trypanosomosis on polo horses in the two local government areas of Kaduna State will therefore provide the much needed information on the impact of these diseases on breed of horses in Nigeria.

CHAPTER 2

LITERATURE REVIEW

2.1 THE HORSE

2.1.1 Taxonomy

The horse *(Equus caballus or Equus ferus caballus)* is a sizeable ungulate mammal, one of ten modern species of the genus *Equus.* Biologically, the horse belongs to the kingdom:

Animalia, Phylum: *Chordata*. Class: *Mammalia*, Order: *Perrissodactyla*, Family: *Equidae*, Genus: *Equus*, Subgenus: *Equus*, Species: *Equus caballus* (Naviaux, 1978).

The early horse, Eohippus (dawn horse), was a multi-toed animal measuring 12 to 14 inches in height. It lived in swamps and gradually became adapted to a new environment, became larger, higher and single-toed with added speed and ability to go further away from the water source. The survival of the horse, can be attributed to its ability to run away from danger, and kick suddenly at unwanted guests. This forms the basis for safety rules in horse handling (Naviaus, 1978).

2.1.2 Breeds of Horses

Breeds are groups of animals with common ancestry and certain distinguishable traits as a result of selective mating usually sufficiently fixed to pass progeny to successive generations. Such traits may be size, color or temperament. All may not be present in one breed but usually, there is something unique in a breed commonly distinguishable (Elwyn, 2000).Modern breeds of horses are categorized on the basis of their size, geographical location and a third category that do not fit into breed is the category of 'Type' of horses (Elwyn, 2000).

2.1.2.1 Foundation breeds

There are three known original natural breeds (foundation breeds) before human intervention in selective breeding to meet his desire for specific traits in horses, namely.

2.1.2.2 The Arabian Horse

Has the widest spread worldwide. It Originated from the Middle East (the Arabian peninsula) where it existed at least 2,000 years BC. It is fiery and courageous, innately gentle, has powerful limbs and endures long distance rides. Possesses slim body with long

head, concave lower neck region and small muzzle. Unique in its anatomy from other breeds that have 18 ribs, 6 lumbar vertebrae and 18 cocceygeal vertebrae; the Arabian horse has 17 ribs. 6 Lumbar and 16 Coccygeal vertebrae and high tail carriage. It measures between 14.2 to 15 hands (14.2 ─ 15hh) in height (Elwyn, 2000).

2.1.2.3 The Barb breed

From Morocco in North Africa is found a genetically powerful breed called the Barb breed. It is said to be as old as the Arabian if not older in existence. Its conformation is inferior to the Arab ideal. Phenotypically, it is not an impressive horse. It has sloping quarters, low-set tail, plain head with skull formation verging on that of primitive horse types. However, its profile is straight. It endures with unlimited stamina. It is exceptionally agile and can travel fast over short distances and its progeny provided basis for many of the foremost breeds of horses in Europe and America such as the Thoroughbred. It measures about 15.2hh in height (Elwyn, 2000).

2.1.2.4 Spanish breed

"Third man" as it is referred to, is a descendant of the barb breed. It is the premier horse of Europe for over two centuries and favorite mount of kings and captains. The present day breed of horses all over the world are products of selective cross breeding of its foundation breeds (Elwyn, 2000). Most present day European horse breeds have Spanish blood. It is not known for speed but has great strength, courage and stamina (Elwyn, 2000).

2.1.3 Breeds Based on Size

2.1.3.1 Ponies

Are breeds that have small size and usually measure between 10 and 15 hands tall when measured from ground to the point of withers. A hand (hh) in measurement is the approximate breadth of a man's hand and is largely a British and American convention, but

it is the traditional form of measurement. One (1) hand is accepted as being 4 inches (10 cm). The pony has a short back. The length of the head is usually equal to the length of measurement from withers to the point of shoulders and equals measurement from withers to the croup (Elwyn, 2000). Arewa breed of horse in Nigeria have height and size about that of a pony, therefore, could serve as a good example of a pony.

2.1.3.2 Light Horses

Height measurement is between 15 – 17.2 hh. It has a back that allows good fitting of saddles within the flat true ribs (the first eight) so that the saddle lies behind the trapezium muscle. The ten false ribs are rounded and well – sprung. The withers are clearly defined as having concave profile at the angle between neck and head ventrally as found in Arabian and Sudanese horses. They are good in racing and polo (Elwyn, 2000).

2.1.3.3 Heavy Horses

They stand between 16 and 18hh. They combine strength and weight. They have wide body, broad back and rounded withers to give them increased ruling power as draught horses. The body is heavily muscled, especially over the loins and quarters, shoulders are relatively upright to accommodate collars. The limbs are thick, short, and typically feathered (long tuft of hairs along the coronary boarders, fanning downward to partially cover the hooves) (Michael, 2003; Elwyn, 2000).

2.1.4 Breeds Based on Geographical Location

The locations of present day horses are used to describe the breeds. In Nigeria there are breeds such as: (1) local breed ("Arewa" breed), (2) Exotic breeds such as i. Argentine, ii. Arabian Cross, iii. Albino, iv. Sudanese, v. Algerian cross and vi. South African breeds (Lawal *et al.,* 1999).

2.1.4.1 Arewa breed

They are small sized horses that weigh averagely about 300kg and can be described as ponies. Confident with slim bodies, they are Arabian cross-breeds used mostly by royal fathers for ceremonial riding and the durbar.

2.1.4.2 Argentine breeds

Are heavy and tall, they are used for polo and racing in Nigeria.

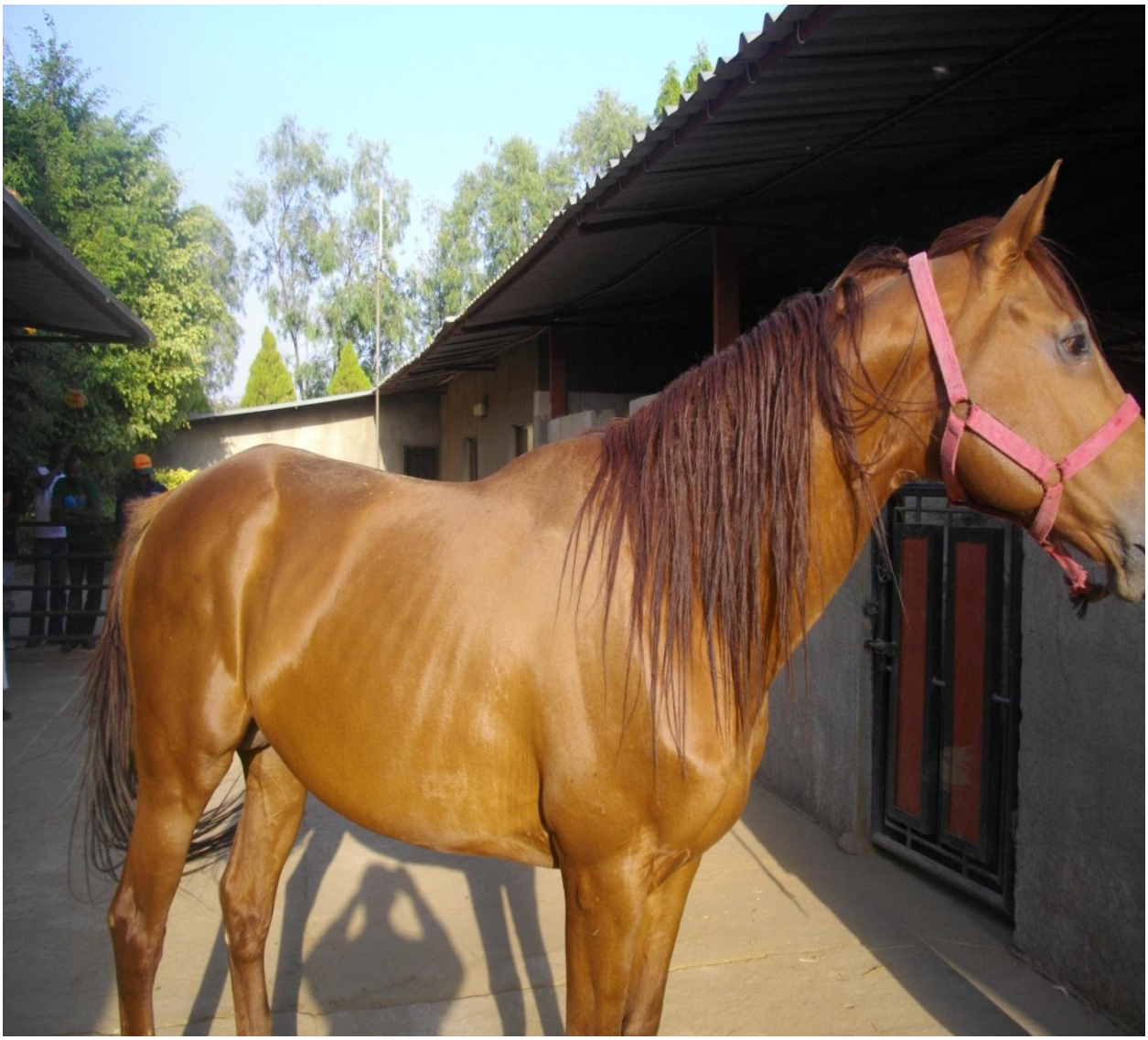


Plate I: An Argentine stallion in good health. Courtesy Fifth Chukker Stable Kaduna

2.1.4.3 South African breeds

Are heavy breads of horses and derived by cross-breeding of British Thoroughbred with South African indigenous horses. They weigh between 500-600kg

2.1.5 Types of Horses

Categorization of horses as type is based on usage. A type category may consist of different breeds that possess similar qualities that make them suitable for a particular purpose. These uses of horses include;

2.1.5.1 Defense and security

Is one of the uses of horses as far back as the Second World War (Elwyn, 2000) during which it was an important logistic machinery for moving officers and men to and from battle fields as well as in launching attacks on enemies. In 1896, the Nupe Empire, which was a big and strong ally of the Sokoto Caliphate, used traditional cavalry soldiers to launch offensives against a unit of 45 armed men of the British colonial Royal West African Frontier Force (RWAFF) entering Bida territory and caused the RWAFF to surrender their riffles and Gardner Machine gun (Jimada, 2005). These were achieved using horses. Today the use of horses in defense and security is limited to crowd control, patrol in busy city

streets, parks and rural anti-robbery patrol by the police in semi-arid and arid regions of northern Nigeria (Elwyn, 2000). The light breeds are good for this purpose. Many nations use cavalry for ceremonial purpose and no state occasion are considered complete without the pageantry of mounted horsemen (Elwyn, 2000).

2.1.5.2 Working Horse

Also known as draught horses, are used in Agriculture to plough or till soil, turn mills and machines in local sugar factories, draw buses or caravans in city centers. The heavy horse is larger and well-muscled to be used for pulling objects (Elwyn, 2000).

2.1.5.3 Sports

Because of the joy man derives in competition, horses are used in events like (i) Horse racing (ii) Show Jumping (iii) Dressage, (iv) Game of polo among the royals and affluent in the society, (v) Leisure ride by young men learning how to ride horses during festivities has resulted in the creation of more riding schools and recruiting instructors thereby creating more job opportunities. Most of the sports emerged from fitness tests for officers, men and their horses at the time horses were used for cavalry (Elwyn, 2000).

2.1.5.4 Hunting and livestock herd control

Horses are used in hunting and in control of movement of sheep and cattle herds during grazing as done by the Cow-boys of the Americas.

2.1.5.5 Durbar

Is a ceremonial horse riding carnival especially among traditional royal councils in northern states of Nigeria. It is organized during festivals like Sallah and any other occasion such as

victory in war, installation of a new ruler and to honour statesmen. State durbars were first organized in 1911 in Kano to honour Sir Lord Lugard, the governor of northern Nigeria. In 1959 it was organized for attainment of self-rule in northern Nigeria, in 1977 during FESTAC '77 and in 2004 to mark the closing ceremony of NAFEST 2004 in Kaduna to mention but a few major ones. Although the durbar has been described as a viable product of cultural tourism (Yahuza, 2005), the efficient use of horses for the mentioned purposes is greatly hampered by health problems that reduce performance.

2.2 BABESIA INFECTION IN HORSES

2.2.1 Etiology

Babesia is a pear-shaped, intra-erythrocytic protozoan parasite belonging to the Subclass - *Piroplasma*, Family-*Babesidae* and Genus - *Babesia* (McCosker, 1981). Different species of Babesia cause disease in domestic animals (horses, cattle, sheep/goat, pigs, dogs and cats). The disease is distributed world wide and known as tick fever, red water fever, texas fever, splenic fever and piroplasmosis in different species of animals (Mc Cosker, 1981). It has also been reported to be zoonotic (Barbara *et al*., 1996). In horses, *Babesia* infection is caused by *Babesia equi* which has recently been reported as *Theileria equi* because the two parasites are phylogenetically identical (Mehlhorn and Schein, 1998) and *Babesia caballi* (Friedhoff *et al.,* 1990*). Babesia (Theileria) equi* is more important as the causative agent of Babesiosis in horses world wide (Uilenberg, 1995) and is transmitted by the tick vector (Uilenberg, 1995; Knowles *et al*., 1992).

2.3 TRYPANOSOME INFECTION IN HORSES

2.3.1 Etiology

Trypanosome is an enlongated and flattened shaped protozoan cell with an undulating membrane wrapped around the body. It belongs to the Order *Kinetoplastida,* Suborder *Trypanosomidae,* Farmily *Trypanosomatidae,* Genus *Trypanosoma* (Maudlin *et al*.,2004).

The important disease producing parasites in cold blooded animals is known as Trypanosomes which was later found in horses sufering from surra in India. The nagana and Mal de cadera of South American horses is also caused by trypanosomes (Frans and Josef, 1926).

Different species cause diseases called trypanosomosis in man and domestic animals. Trypanosomosis is a complex deblitating, zoonotic hemoprotozoan disease of man and his domestic animals. This disease stretches across over 40 countries in sub-saharan Africa with 60million people and 45 million cattle at risk from the 23 species and 33 subspecies of tsetse flies infesting about 10million km^2 of the land mass resulting in annual deaths of 55,000 humans and 3 million cattle (Abenga *et al*., 2002; Kamuaga, 2003). African Animal Trypanosomosis (AAT) is a disease caused by tsetse-transmitted *Trypanosoma congolense*, *T. vivax*, *T. brucei brucei*, *T. simiae* (Itard,1989*)* or simultaneous infection with one or more of these trypanosomes. The tsetse-fly-infested areas of Africa extends from the southern edge of the Sahara desert (lat. 15° N.) to Angola, Zimbabwe, and Mozambique (lat. 20° S) (WHO, 1998). Of the three African animal trypanosomes, only *T. vivax* occurs in the Western hemisphere in at least 10 countries in the Caribbean and South and Central America (Mare, 2008). AAT is most important in cattle but can cause serious losses in horses which are susceptible to *Trypanosoma congolense*, *T. vivax*, *T. brucei brucei*, *T. evansi* and *T. equiperdum,* (Radostits *et al*., 2000; Taylor and Authei, 2004; Gina *et al*., 2008). Infection of horses by one or more of these trypanosomes has been reported by Gina *et al.* (2008) and may result in subacute, acute, or chronic disease. In Southern Africa, the

disease is widely known as nagana, which is derived from a Zulu term meaning "to be in low or depressed spirits"— a very apt description of the disease.

2.3.1.1 Subgenus Nannomonas

A group of small trypanosomes with medium-sized marginal kinetoplasts, no free flagella, and poorly developed undulating membranes. *T. congolense* resides in this group (Mare, 2008).

2.3.1.2 Subgenus Duttonella

A group of trypanosomes with large terminal kinetoplasts, distinct free flagella, and inconspicuous undulating membranes. *T. vivax* is a large (18-26 µm long) monomorphic organism (Maudlin *et al.*, 2004) that is very active in wet-mount blood smears.

2.3.1.3 Subgenus Trypanozoon

T. b. brucei is an extremely polymorphic trypanosome occurring as short, stumpy organisms without flagella, long slender organisms with distinct flagella, and intermediate forms that are usually flagellated. *T. evansi* and *T.equiperdum* cannot be distinguished morphologically from one another or from *T. brucei brucei* .

2.4 THE VECTORS OF BABESIOSIS AND TRYPANOSOMOSIS

2.4.1 Ticks as a Vector of Babesiosis

Ticks are the most important ectoparasites of horses in Brazil, causing serious damages, through their feeding habit on host animal predisposing horses to secondary bacterial skin infections until they adapt well to climate and vegetation of an area (Klompen *et al.*, 1996). Ticks cause great economic losses in livestock production in Nigeria (Mohammed and Agbede, 1980) and are said to be responsible for the greatest economic setback to livestock

production worldwide (Snelson, 1975; Griffiths, 1977). The genera incriminated in the transmission of *Babesia* parasite in horses are: *Rhipicehalus, Dermacentor, Boophilus, Hyalomma* and *Otobius* species (Uilenberg, 1967; Knowles *et al.*, 1992; Unilenberg, 1995). *Boophilus* sp. is a one-host tick with a life cycle of 3-4 weeks. It is found in the tropics and subtropics of Africa. *Boophilus microplus* is the only important specie responsible for the spread of Babesia in horses (Susan and Asa,1997). *Dermacentor* sp is a one or three host tick found in temperate climates. Only *D.nitens* is of veterinary importance, being a one host tick in the temperate climate. It is also found in tropical America (Susan and Asa, 1977). *Hyalomma* sp is a two or three host tick found in the tropical areas such as India, Pakistan, Ethiopia, and Saudi Arabia. Rhipicephalus sp is found in Africa, Asia and Europe. It is typically a three-host tick but some species in Africa are two-host ticks. *Rhipicephalus sanguineus* (three-host) is the common brown dog tick, which is active throughout the year in the tropics and subtropics. Another two–host tick specie which infects the horse is *R. evertsi* (Susan and Asa, 1997). *Otobius sp* are *Argasid* ticks found in low rainfall areas of USA, Canada and Mexico. It was introduced into Southeastern Africa through livestock trade. The nymph is the parasitic stage while adults have non-functional mouthparts and can survive on the ground without feeding for up to 2 years (Susan and Asa, 1997).

2.4.2 Tsetsefly (*Glossina* spp) as a Vector of Trypanosomosis

Tsetseflies are the most important vectors of trypanosomosis in subsaharan Africa because their feeding habits predispose their host to cyclical transmission of trypanosomosis (Seifert, 1996). Twentytwo species of Glossina are known in Africa with eleven of these species infesting 75% of the land mass in Nigeria. Tsetse and trypanosomosis has been discribed as the single major barrier to the development of the African continent. Africa has the potential to generate up to 50million US dollars from livestock and agriculture if tsetse infested areas

are eradicated (Abenga *et al.*, 2002). Gina *et al*. (2008) reported preference of horses over donkeys by tsetseflies during feeding.

2.4.3 Mechanical Transmitters as Vectors of Trypanosomosis

Trypanosomiasis is also mechanically transmitted by tsetse and other biting flies through the transfer of blood from one animal to another. The most important mechanical vectors are flies of the genus *Tabanus*. But *Haematopota, Liperosia, Stomoxys*, and *Chrysops* flies have also been implicated (Seifert,1996). In Africa, both *T. vivax* and *T. b. brucei* have spread beyond the "tsetse fly belts" (Roder *et al*., 1984) where transmission is principally by Tabanid and Hippoboscid flies (Seifert, 1996; Desquesnes and Dia, 2003; Mare, 2008).

2.5 TRANSMISSION

2.5.1 Mode of Transmission of *Babesia* Parasites

The infective stage of the parasite (Sporozoite) is injected into the host along with saliva from the tick (Smyth, 1996). *B. equi* transmission is trans-stadial only (Friedhoff, 1982; Young and Mozana, 1986). The following ticks have been reported to act as the vector of the disease; *R. saguineus, R. turanicus, R. bursa, D. reticularis, D. marginatus* and *H. excavatum* transmits *B. equi* while *R. sanguineus, R. bursa, D. nitens, D. salvarum, D marginatus D. reticularis* and *H. excavatum* species of ticks transmit *B. caballi*.

2.5.2 Mode of Transmission of *Trypanosoma* Species

The initial replication of trypanosomes is at the site of inoculation on the skin; this causes a swelling and a sore (chancre). Trypanosomes then spread to the lymph nodes and blood and continue to replicate (Maudlin *et al.*, 2004). In Africa, the primary vector for *T. congolense*, *T. vivax*, and *T. b. brucei* is the tsetse fly (Mare, 2008). These trypanosomes replicate in the tsetse fly and are transmitted through tsetse fly saliva when the fly feeds on a host. The three main species of tsetse flies responsible for transmission of trypanosomes are *Glossina*

morsitans, which favors the open woodland of the savanna; *G. palpalis*, which prefers the shaded habitat immediately adjacent to rivers and lakes; and *G. fusca*, which favors the high, dense forest areas (Seifert, 1996). Trypanosomes can be transmiited intrauterine or congenitally (Woo and Limebeer, 1971; WHO, 1998), through coitus in *T .evansi* (Radostits *et al.*, 2000) and through contamination in *T. cruzi* (WHO, 1998).

2.6 LIFE CYCLE

2.6.1 Life Cycle of *Babesia* Species

There appears to be a pausity of reports in the transmission of equine babesiosis by other insects, however, iatrogenic transmission occurs during surgical procedures like castration and needle injection during vaccination (Russel *et al.*, 2005). The infective stage of the parasite, the *sporozoite,* is deposited into the bite site by an infected tick along with saliva from the tick. The parasite advances into the blood steam to infect erythrocytes, multiply by binary fission (asexual) to produce more parasites, the merozoites, most of which will reinvade more erythrocytes (Mehlhorn and Schein, 1984; Restic, 1988). Some of the merozoites will form ovoid, free living forms called the gamonts. During another blood meal by the tick, the infected erythrocytes are ingested and digested by the tick gut to release the *Babesia* cells. The gamonts that survive differentiate into 'ray bodies' within the hemolymph and fuse in pairs to form zygote giving rise to the motile kinete (Mehlhorn and Schein, 1984; Ristic, 1988). Some of the kinetes move to the ovaries and invade the developing ova, thus the eggs to be laid by this adult are already infected transtadially within the developing larvae. Some kinetes infect salivary gland acini cells, where many thousand of *sporozoites* are formed. When the infected tick nymph feeds on another susceptible host, the *sporozoites* are injected along with the saliva and the cycle begins again. This model of life cycle is best understood in *Boophilus sp* in the transmission of *B. canis* (Melhorn and Schein, 1984; Ristic, 1988).

2.6.2 Life Cycle of *Trypanosoma* Species

The life cycle of trypanosomes determines the mode of transmission which is either cyclically (Anterior station pattern) or by contamination (posterior station pattern) (Itard,1989). In the anterior station pattern during a blood meal on the mammalian host, an infected tsetse fly (genus *Glossina*) injects metacyclic trypomastigotes into skin tissue. The parasites enter the lymphatic system and pass into the bloodstream. Inside the host, they transform into bloodstream trypomastigotes and are carried to other sites throughout the body, with replication by binary fission (Soulby,1982). The entire life cycle of African Trypanosomes is represented by extracellular stages. The tsetse fly becomes infected with bloodstream trypomastigotes when taking a blood meal on an infected mammalian host (Mare, 2008). In the fly's midgut, the parasites transform into procyclic trypomastigotes, multiply by binary fission, leave the midgut, and transform into epimastigotes. The epimastigotes reach the fly's salivary glands and continue multiplication by binary fission and the cycle in the fly takes 3 weeks (Maudlin *et al*., 2004; Richard, 2004). In the posterior pattern, an infected triatomine insect vector (or "kissing" bug) takes a blood meal and releases trypomastigotes in its faeces near the site of the bite wound. Trypomastigotes enter the host through the wound or through intact mucosal membranes, such as the conjunctiva inside the host. The trypomastigotes invade cells, where they differentiate into intracellular amastigotes. The amastigotes multiply by binaryfission and differentiate into trypomastigotes. Trypomastigotes infect cells from a variety of tissues and transform into intracellular amastigotes in new infection sites. The bloodstream trypomastigotes do not replicate (different from the African trypanosomes). Replication resumes only when the parasites are ingested by the vector. The "kissing" bug becomes infected by feeding on human or animal blood that contains circulating parasites. The ingested trypomastigotes transform into epimastigotes in the vector's midgut. The parasites multiply and differentiate

in the midgut and differentiate into infective metacyclic trypomastigotes in the hindgut (Maudlin *et al*., 2004; Richard, 2006).

2.7 EPIDEMIOLOGY

2.7.1 Epidemiology of Equine Babesiosis in Nigeria

Equine babesiosis is widely distributed in the tropics and subtropics where the disease is endemic, and to a lesser extent in the temperate regions (Uilenberg, 1995). It is an enzootic disease around Zaria and has a slightly high prevalence in exotic breeds (35%) than in local breed of horses (33%) (Lawal *et al.,* 1999). However, *B. equi* infection predominates with 90% of the positive samples while *B.cabali,* is scanty with only 2% prevalence as observed around Zaria. The disease has seasonal variation with *B. equi* occurring with higher incidence in wet and early dry season because this is the period of higher activity of ticks (such as *R. Sanguineus*) is associated with equine babesiosis (Lawal *et al*.,1999). Abdulkadir (1994) had earlier reported a prevalence of 10.6% for equin babesiosis around the Country,making it the third most important health problem in horses after tick infestation with 17.8%.

2.7.2 Epidemiology of Equine Trypanosomosis in Nigeria

Trypanosomosis due to *Trypanosoma brucei brucei , T .vivax , T. congolense , T. evansi and T . equiperdum* has been reported in horses (Sarah *et al*., 2006; Gina *et al*., 2008). In Gambia, using polymarase chain reaction, an overall trypanosome prevalence in horses was 91% with an infection rate of 31% for *Trypanosoma congolense*, 87% for *Trypanosoma vivax* and 18% for *Trypanosoma brucei* sp. Infection with multiple species accounting for 43% (Gina *et al.*, 2008). Dethie *et al*. (2001) reported an average monthly incidence of trypanosome infection rate in horses at 45.5 % in Gambia. Sura has a world wide distribution in areas of Africa North of the tsetse belt, in the middle east, Asia, Central and

South America (Radostits *et al*., 2000). In some Countries the incidence increases within rainy season when there are large numbers and high activity of biting flies. Dourine is enzootic in northern and southern Africa, Asia, Southern Europe and South America. It was eradicated from Canada, greater part of the United States and is rarely reported in Africa.

2.8 CLINICAL SIGNS

2.8.1 Incubation Period for Babesiosis

The incubation period (IP) of the disease in horses is generally 8 to 10 days (Radostits *et al.*, 2000). In natural transmission, the IP is 10-21 days while in experimental infection it is 5-9 days (Zavagli, 1970).

2.8.2 Incubation Period for Trypanosomosis

The incubation period for *T. congolense* varies from 4 to 24 days; for *T. vivax*, from 4 to 40 days; and for *T. b. brucei*, from 5 to 10 days. For *T. evansi*, from 5 to 60 days (Sarah *et al.*, 2006; Mare, 2008).

2.8.3 Clinical Forms of Babesiosis

Babesiosis at times can be acute and sudden in onset, or chronic and sub-clinical (latent infection). The chronic form aids in the spread of the disease (Hailat *et al*., 1997). The acute form is characterized by sudden onset, reluctance to move, lateral recumbency and inability to respond to stimuli (Radostits *et al*., 2000). There is complete anorexia, fever of up to 40^{o}C (104 F) and after 24 hours of infection, the fever becomes intermittent.

Other signs include oedema of the fetlock, head, ventral abdomen, thick mucus on fecal balls, constipation, colic and often no hemoglobinuria and bronchitis may be present. Signs

are more severe in the younger horses and those brought into enzootic areas without pre-immunition. Infected foals may die within 24 hours and newborns infected intrauterine may develop severe jaundice and prostration. Sometimes signs are delayed 2-3 days after birth (Radostits *et al.,* 2000). Icterus, hemorrhagic mucous membranes of the vagina and conjunctiva. Profuse lacrimation may also be found in clinical cases (Zavagli, 1970). The Chronic form develops when the disease persists as a latent infection and horses recovering from the disease may not show any sign of disease despite presence of infection (Malhitra *et al*., 1978). At this stage parasitemia is low and microscopic technique may not be sufficient to demonstrate the parastite in the blood smear (Malhitra *et al*., 1978; David, 2001). The chronically affected are carriers and could maintain infection for up to 5 years (Holman, 1997). Clinical Babesiosis may be triggered off in carriers when subjected to stress factors or in immunosuppressive condition like splenectomy (Kyewalabye and Lawal, 1989). Super-imposed infection of other parasites such as *Trypanosoma evansi* can also precipitate disease (Ilemobade, 1971).

2.8.4 Clinical Forms of Trypanosomosis

Because simultaneous infections with more than one trypanosome species are very common (Nyeko *et al*., 1990) as well as simultaneous infection with trypanosomes and other hemoparasites (*Babesia* spp., *Theileria* spp., *Anaplasma* spp., and *Ehrlichia* spp.). It is difficult to conclude which clinical signs are attributable to a given parasite. Few adequately controlled studies have been made, and thus a "typical" clinical response to each trypanosome is difficult to reconstruct. What follows is a summation of the syndromes observed in field and experimental cases of trypanosomosis caused by each of the African animal trypanosomes.The cardinal clinical sign observed in AAT is anemia.Within a week of infection with the hematic trypanosomes (*T. congolense* and *T. vivax*), there is usually a pronounced decrease in packed cell volume, hemoglobin, red blood cell, and white blood

cell levels, and within 2 months these may drop to below 50 percent of their preinfection values (Mare, 2008). Also invariably present are intermittent fever, oedema and loss of condition. Abortion may be seen and infertility of males and females may be a sequel (Sarah *et al*., 2006). The severity of the clinical response is dependent on the species and the breed of affected animal and the dose and virulence of the infecting trypanosome. Stress, such as poor nutrition or concurrent disease, plays a prominent role in the disease process, and under experimental conditions, where stress may be markedly reduced, it is difficult to elicit clinical disease (Gina *et al*., 2008; Mare, 2008). *Trypanosoma. congolense* is a hematic trypanosome found only in the blood vessels of the animals it infects. Infection with *T. congolense* may result in peracute, acute, or chronic disease in cattle, sheep, goats, horses, and camels. Pigs often develop a milder disease. The incubation period is followed by intermittent febrile episodes, depression, lethargy, weakness, loss of condition, anemia, salivation, lacrimation, and nasal discharge. As the disease progresses, loss of condition and hair color changes from black to metallic brown are seen. The back is often arched and the abdomen "tucked up". Accelerated pulse and jugular pulsation occur and breathing is difficult (Seifert, 1996).

T. vivax has a variable incubation period, and, although it is considered to be less virulent for cattle than *T. congolense*, mortality rates of over 50 percent can occur (Mare, 2008). There seems to be a marked variation in the virulence of different strains of *T. vivax*, but it remains the most important cause of trypanosomosis of cattle, sheep, and goats in West Africa (Radostits *et al*., 2000). It causes mild disease in horses and chronic disease in dogs. *T. vivax* is often difficult to find in blood smears and can also be demonstrated in lymph node smears.

T. brucei has a relatively short incubation period and causes severe to fatal infection in horses, camels, dogs, and cats. It usually causes mild, chronic, or subclinical disease in

cattle, sheep, goats, and pigs. A febrile response occurs in the horse 4-14 days after infection. This is followed by recurrent febrile reactions. The heartbeat and respiration may be accelerated, and loss of condition and weakness are seen, dog sitting position, headtilt, circling, staggering , prostration, ataxia and death. Progressive anemia, icterus, and oedema of the ventral regions, especially the male genitalia, are characteristic (Seifert, 1996; Auty *et al.*, 2008).

2.9 PATHOGENESIS AND PATHOLOGY

The pathogenesis and pathology of both diseases has been reviewed by several workers; Fiennes (1970); Anosa and Keneko (1984 (a,c)); Esievo and Saror (1983) reviewed the pathogenesis and pathology for trypanosomosis and McCosker (1981); Uilenberg (1995); Barbara *et al* (1996) reviewed the pathogenesis and pathology for babesiosis.

2.9.1 Pathogenesis of Fever and Colic in Babesiosis

Parasitemia will trigger the host immune response causing cytokines and interleukin – 6 to stimulate the pre-optic area of the hypothalamus through the vascular organ of lamina terminalis to secrete prostaglandins which further raises body temperature (Smyth, 1996). Oedema may result from obstruction of peripheral venous return by slugging of lysed red blood cell (RBC) culminating in escape of plasma into the interstitial space or decrease in circulating proteins. Constipation seen in equine piroplasmosis causes abdominal distension and may give rise to physical colic (Radostits *et al.*, 2000).

2.9.2 Pathogenesis of Fever in Trypanosomiasis

Trypanosomes will trigger host immune response causing cytokines and interleukin–6 to stimulate the pre-optic area of the hypothalamus through the vascular organ of lamina terminalis to secret prostaglandins which further raises body temperature. The toxic products and byproducts of trypanosomes contributes to increase in tempereture (Franz and

Josef, 1926). The undulating fever associated with trypanosomosis may also contribute to erythrocyte distruction because, above normal body tempereture their osomotic fragility, membrane permeability increases and membrane plasticity decreases.

2.9.3 Clinical, Hematological and Biochemical Findings in Babesiosis

The pathogenesis of equine babesiosis is not adequately understood. Stress, placed on the parasitized erythrocyte and uptake of phosphorus to generate energy for the babesia parasite by the red blood cell may cause hypophosphatemia (Camacho *et al.,* 2005) and weakening of erythrocyte cell membrane causing hemolysis. Parasitized red blood cells lysed intravascularly producing hemoglobinemia during the acute phase of the disease, cause a marked hyperbilirubinemia and icterus which can be pronounced in many cases (Radostits *et al*., 2000). Hemoglobinuria seems to occur more frequently and severely with *B. equi* infection but can be seen in horses infected with the other species of babesia. In addition to regenerative hemolytic anemia, a significant monocytosis, eosinopenia can be seen in horses with babesiosis (Radostits *et al.*, 2000). In severe disease, the intravascular hemolysis may disturb capillary blood flow enough to cause disseminated intravascular coagulation (DIC), resulting in signs of coagulopathy (Radostits *et al*., 2000). The rates of urea and creatinine clearnance are primarily functions of glomerular filtration rate (GFR). These biochemical substances are endogenously produced at a constant rate and cleared steadily thereby maintaining a stable plasma level. However, in kidney disease of humans,the plasma levels are raised (Thomas and Ellis, 1974). A similar phenomenon has been reported in equine piroplasmosis (Camacho *et al*., 2005). This forms the basis for the use of plasma levels of these substances in assessing renal function (Thomas and Ellis, 1974). Hypophosphatemia due to uptake of phosphorus by infected erythrocytes to generate energy for the *Babesia*

organism and hypocalcaemia may be seen following equine piroplasmosis (Camacho *et al*., 2005).

2.9.4 Clinical Hematological and Biochemical Findings in Trypanosomosis

The anemic blood changes are anisocytosis, poikilocytosis, polychromasia, and punctate basophilia. All, some, or none of the above may be seen. The lesions caused by the trypanosomes in susceptible host species vary considerably, depending on the species and strain of trypanosome and the species and breed of host animals affected. The hematic trypanosomes (*T. congolense* and *T. vivax*) cause injury to the host mainly by the production of severe anemia (50%) reduction in PCV (Mare, 2008) which is accompanied in the early stages of the disease by leukopenia (30% to 50% reduction in total leucocytes) (M-audlin *et al*., 2004) and thrombocytopenia. In the terminal stages of the disease caused by the hematic trypanosomes, focal polioencephalomalacia probably results from ischemia due to massive accumulation of the parasites in the terminal capillaries of the brain. The lesions resulting from *T. b. brucei* (a tissue parasite) are remarkably different from those seen with the hematic trypanosomes. Anemia is an important lesion, but much more dramatic are the inflammation, degeneration, and necrosis resulting from cellular invasion of various organs. Marked proliferative changes reflecting immunologic response are observed in most body tissues. Biryomumaisho *et al*. (2003) reported that challanges with either *Trypanosoma congolense* or *T.brucei* results into cholestrolaemia, hypoproteinaemia, hypoalbuminaemia, hypocholestronaemia, low and high density hypolipidaemia.

2.10 GROSS FINDINGS

2.10.1 Gross Lesions in Babesiosis

Horse's carcass at necropsy shows lesions indicative of anemia and icterus. Petechial hemorrhages are found in the intestinal mucosa, on the heart along the cardiac vessels, in the

kidneys and commonly in the visceral peritoneum over the small intestine. Hepatic enlargement and degeneration are common findings. Chronic nephritis can also be seen. Carcasses from chronic cases may be emaciated (Zavagli, 1970). In addition to the above, thin watery blood, effusion into body cavities and of the pericardium are also seen (Radostits *et al*., 2000).

2.10.2 Gross Lesions in Trypanosomosis

No pathognomonic change is seen in African animal trypanosomosis. Anemia, eodema, and serous atrophy of fat are commonly observed. Subcutaneous oedema is particularly prominent and is usually accompanied by ascites, hydropericardium and hydrothorax (Radostits *et al*., 2000). The liver may be enlarged, and edema of lymph nodes is often seen in the acute disease, but they may be reduced in size in the chronic disease. The spleen and lymph nodes may be swollen, normal, or atrophic (Seifert ,1996). Necrosis of the kidneys and heart muscle and subserous petechial hemorrhages commonly occur. Gastroenteritis is common, and focal polioencephalomalacia may be seen.

2.11 DIAGNOSIS

2.11.1 Diagnosis of Babesiosis

2.11.1.1 History of the disease in the area

The associated clinical signs and presence of the tick vector on the body of suspected horses are important in making a tentative diagnosis.Because ticks are the most important vectors that transmit Babesiosis in horses (Uilenberg, 1981; Noval *et al*., 1992; Klompen *et al*., 1996; Radostits *et al*., 2000). Although the vectors of *B. equi* have not been confirmed in Nigeria, several tick species (such as *R. sanguineus*) incriminated in the transmission are prevalent (Neitz,1956). But it should be noted that *Rhipicephalus sp.* of ticks being difficult to identify to specie level, should draw the attention of experts (Walker and Koney, 1999).

2.11.1.2 Light microscopy

Light microscopy is a morphological technique which involves making a thin blood smear on glass slide. Fixed with alcohol and stained with Giemsa reagent. This will reveal the presence of paired piriform or pear-like bodies joined at an acute angle in the infected erythrocyte in the case of *Babesia equi* (small Babesia) and 4 piriform bodies that form a structure known as 'maltese cross' in the case of *Babesia caballi* (large Babesia) when examined at 100 x bright-field light microscopy (Benjamin, 1978). The technique of thin blood smear may not detect infection in the carrier animal where presence of parasite in blood is scanty and in acute cases at the onset of disease before parasitemia high but is a highly specific technique (Bruning, 1996; Papadopoulos *et al*., 1996).

2.11.2 Diagnosis of Trypanosomosis

Trypanosomosis should be suspected when an animal in an endemic area is anemic and in poor condition. Confirmation depends on the demonstration of the organism in blood or lymph node smears. Detailed description of parasitological techniques for diagnosis of trypanosomosis is reviewed by Murray *et al* (1983); Ikede (1991).

2.11.2.1 Parasitological method

This involves the demonstration of trypanosomes either directly by wet film, thick/thin film or after concentration by heamatocrit centrifugation and buffy coat methods as described by Woo (1970) or inoculation into laboratory animals. In the early phases of infection, especially with *T. vivax* and *T. congolense*, the parasite can readily be observed by microscopic examination of a wet-mount of blood slides. Thick blood films stained with Giemsa are also a good technique. But in thin fixed blood films, which are favored for species identification based on morphology, the parasites may be hard to demonstrate. When parasitemia is low smears of buffy coat (obtained by microhematocrit centrifugation) can be

useful for demonstration of the parasites. Because *T. congolense* tends to associate with the erythrocytes, it is essential that buffy coat and adjacent erythrocytes be included in the smear to ensure demonstration of the parasite (Itard, 1989).

2.12 SEROLOGY

2.12.1 Serological Methods for Babesiosis

Serological methods, like the Complement Fixation Test (CFT) and the Indirect Fluorescent Antibody Test (IFAT) are useful in detection of past infections but cross reactivity between species have been reported which confuses species differentiation (Schein, 1988; Bruning, 1996; Papadopoulos *et al.*, 1996). Western Blot Analysis (WBA) has also proved satisfactory in detecting the disease in carrier horses. Both IFAT and WBA have detected babesiosis in a horse with a 5 years old latent infection (Holman *et al*., 1997). Enzyme Linked Immunosorbent Assay (ELISA) is also a sensitive serological method (Bahiruddin *et al*., 1999) using recombinant antigen, was developed to replace CFT and IFAT for the diagnosis of *B. equi* in order to eliminate the problems of antibody detection and cross-reactivity thus ELISA has advantages over CFT and IFAT (Xuan *et al*., 2001).

2.12.2 Serological Method for Trypanosomosis

The conventional techniques of microscopic examination for the presence of trypanosomes are still widely used but newer and far more sensitive methods are beginning to supplant them. The antigen-detecting enzyme-linked immunosorbent assay is extremely sensitive for the detection of trypanosomosis (Mare, 2008), while others like indirect fluorescent antibody test, compliment fixation test, agglutination test and passive heamagglutination test have been reviewed by Murray *et al* (1983); Nantulya *et al* (1987).

2.13 MOLECULAR METHODS

2.13.1 Molecular Methods for Diagnosis of Babesiosis

Molecular techniques such as the Polymerase Chain Reaction (PCR) have proved very useful for detection and identification of many hemoparasiste species like the Theileria/Babesia group (Bahiruddin *et al.*, 1999; Caccio *et al*., 2000). The methods are based on specie specific PCR assay which mainly targets the 18s and RNA genes (Sparagano, 1999; Caccio *et al*., 2000; Birkenheuer *et al*., 2003; Criado-Fornelio *et al*., 2003a: Rampersad *et al*., 2003) but specific amplification protocols are needed for each specie. Nicoloiewsky *et al* (2001) and Joanne *et al* (2003) reported higher sensitivity of detection using nested-PCR. Although PCR do not generally detect mixed infections, there are some amplification protocols that can detect mixed piroplasmids (Birkenheuer *et al*., 2003: Criado-Fornelio *et al.*, 2003a). In order to over come the problem of mixed infection detection, different reverse line blot (RLB) assays were developed for simultaneous detection and identification of reckettsial and protozoan species infecting cattle and small ruminants (Bekker *et al*., 2002). RLB assay is a highly sensitive method of detect and identify simultaneously different *Theileria* and *Babesia* species in horses (Schnittger *et al* ., 2004; Daniel *et al*., 2004). The RLB based assay detect and identify *Babesia equi* and *B. caballi* at once even in mixed infections and apparently healthy horses having sub-clinical infection with higher efficiency than the CFT and IFAT. This technique is now used in diagnosis and epidemiological surveys (Daniel *et al*., 2004).

2.13.2 Molecular Methods for Diagnosis of Trypanosomosis

Molecular techniques such as PCR (polymarase chain reaction) have proved very useful for detection of infection and identification of trypanosomes involved (Herder, 2002; Willoughby, 2003). The technique as described by Geysen *et al* (2003) and Njiru *et al* (2005) is based on specie specific PCR RFLP using Ssu-rDNA amplification which mainly targets the genes, but specific amplification protocols are needed for each specie. Species-

specific DNA probes have been shown to detect simultaneous infection of cattle with *T. vivax*, *T. b. brucei*, and *T. congolense* when conventional methods revealed only single infections (Nyeko *et al.*,1990).

2.14 DIFFERENTIAL DIAGNOSIS

The clinical features of trypanosomosis and piroplasmosis in horses closely resemble those of Equine infectious anemia (EIA). There is lowering of P.C.V. in all the diseases but EIA is a viral disease. African Horse Sickness (AHS) on the other hand, shows severe emaciation and intermittent recumbency. Plant Poisoning such as with *Crotalaria* is associated with severe prostration and anemia at the terminal stages of the disease. Trypanosmosis resemble chronic piroplasmosis because of its severe weight loss but pica appetite is characteristic of trypanosomosis. Leptospirosis, though bacterial in etiology, shows severe icterus as in acute piroplasmosis (Zavagli, 1970).

2.15 CHEMOTHERAPY AND CHEMOPROPHYLAXIS

2.15.1 Chemotherapy and Chemoprophylaxis of Babesiosis

Both *B. equi* and *B. caballi* respond to the commonly used babesiocides in horses but *B. equi* is more refractory than the *B. caballi* infection. Imidocarb dipropionate (Imizol®) is the drug of choice in the treatment of carrier status. Kuttler (1981) recommended that in *B. caballi* infection, Imidocarb dipropionate can be given at 2 mg/kg twice at 24 hours interval while in *T. equi* infection, 4 mg/kg four times at 72 hours interval. Adams (1981), observed that the later amount of the drug approaches the lethal dose [LD50] at 32 mg/kg given in two divided 16 mg/kg doses at 24 hours interval. Following treatment with imidocarb at higher dose level. The common side effects which include restlessness, abdominal pain (Colic), sweating, rolling and heavy breathing are common occurrences. The use of diminazene aceturate at the dose rate of 8 mg/kg or imidocarb at the rate of 20 mg/kg three times at 7 days interval to treat acute cases has been reported in Zaria (Lawal *et al.*, 1999).

In new drug trials, in-vitro growth inhibition of *B. equi* and *B. caballi* was observed using pyrimethamine, artesunate and parvaquone (all anti-malarials) showing various sensitivities but pyrimethamine promises to be more effective when tried in vivo (Akiko *et al*., 2003).

2.15.2 Chemotherapy and Chemoprophylaxis of Trypanosomosis

The use of drugs for the prevention and treatment of trypanosomosis has been important for more than 35 years, and is estimated that about 35 million doses per year are currently used in Africa, but the rapidity with which the trypanosomes have developed resistance to each drug introduced has been reported in 13 countries in sub-saharan Africa in addition to the 11 countries mentioned earlier (Geerts and Holmes, 1998). In spite of this, some of the older chemoprophylactic drugs such as the quinapyramine derivatives Antrycide and Antrycide Prosalt are still used and they give effective protection against *T. b. brucei* infection in horses, camels and cattle for up to 3 months. The drug pyrithidium bromide (Prothidium and AD2801) is useful in the prophylaxis of *T. vivax* and *T. congolense* infections in cattle, sheep, and goats and can give protection for up to 6 months. The most widely used of the newer chemoprophylactics is isometamidium chloride. This drug is effective at a dose rate of 0.25-1mg/kg body weight, and has been in use for over 20 years and sold under the trade names Samorin, Trypamidium, and M&B 4180A. It is excellent for the prophylaxis of all three African animal trypanosomes, and gives protection for 3-6 months. The development of resistance to this drug has been reported in both east and west Africa. Homidium bromide has also been found to be an effective chemoprophylactic drug in Kenya at a dose rate of 1mg/kg body wieght, and the newly introduced arsenical Cymelarsan is effective in treatment of *T. b. brucei* infection.A very widely used chemotherapeutic drug is diminazine aceturate (Berenil), which is effective against all three African animal trypanosomes at a dose rate of 3.5-7 mg/kg body weight (Radostits *et al.,* 2000).

2.16 PREVENTION AND CONTROL

2.16.1 Vaccination

There are insufficient reports on vaccination against *B. equi*. No effective vaccine is available for equine babesiosis at the moment (Kuttler, 1981). No vaccine is currently available for AAT (Maudlin *et al*., 2004).

2.16.2 Trypanotolerance

It has long been recognized that certain breeds of African cattle are considerably more resistant to African trypanosomiasis than others. This is especially true of the West African short-horned cattle (Muturu, Baoule, Laguna, Samba, in Dahomey) and the N'Dama, which is also of West African origin. These cattle have existed in the region for over 5,000 years. Susceptibility studies have shown the N'Dama to be the most resistant breed followed by the smaller West African short-horned cattle, but the large and more recently introduced Zebu is the most susceptible. The mechanisms of trypanotolerance have been extensively studied, and it is now well established that trypanotolerance has a genetic basis. Trypanotolerance in sheep and goats has also been described, but the mechanisms of the tolerance phenomenon have not been defined. The small Logone horse or Kirdi pony of Chad is also trypanotolerant (FOA, 1991).

2.17 CONTROL OF THE VECTORS

2.17.1 Ticks

Cultural and Biological method – Is used for both free-living and parasitic stages of ticks especially the one-host. Destruction of microhabitats such as pasture rotation, among paddocks. Strict trans-border survey (quarantine), prevent entry of livestock into the country with new strains of the parasite or ticks, is the technique used in non enzootic areas (Susan

et al., 1997). However in the endemic areas like Nigeria, strict tick control and recovery of horses will be sufficient to reduce the disease incidence. Careful routine surgery and injections with sterilized equipment will prevent inoculation of Babesia into previously clean hosts (Russel *et al.*, 2005).

2.17.2 Tsetseflies and Mechanical Transmitters

Fly eradication and drug prophylaxis are the only effective trypanosomosis control methods now available. Several approaches to fly control have been used with varying degrees of success. Discriminative bush clearing, extensively used in early tsetse fly eradication campaigns, has been locally useful because it eliminates the breeding places of the tsetse. But, to be completely effective, bush clearing requires ecologically unacceptable destruction of vast areas of brush and forest. It is still a useful procedure when used locally in conjunction with other control methods. Game elimination, and thus elimination of the main source of bloodmeals for the tsetse, was used in early eradication campaigns. This was an ineffective and wasteful procedure. Application of the sterile male technique (as used in screwworm eradication in the United States) received considerable attention in the 1980's. Early problems with breeding of the male flies have been overcome, and field trials have been done in Platuea state Nigeria to determine the effectiveness of this approach in vector control. In limited trials, this procedure has reduced fly populations. Ground and aerial spraying with insecticides and the use of synthetic pyrethroids on cattle have lowered fly densities in some areas, but widespread use would require considerable international cooperation and expense. Widespread application of insecticide has the tremendous disadvantage of also eradicating many other arthropods, several of which are desirable. The recent introduction of odor-baited targets impregnated with insecticides is proving promising as a means of reducing the tsetse fly.

2.18 ZOONOSIS

2.18.1 Babesiosis

Human babesiosis is relatively rare and is acquired from cattle infected with *B. bovis,* which may be fatal and *B. microti* of rodents which is an emerging important tick-born disease of man in the temperate regions (Piesman,1987). World Health Organization (1987) reported 7 human cases of babesiosis in Europe and 200 cases in United States of America. The disease was formally thought to be more common in immuno-suppressed persons such as splenectomised individuals (Brocklesby, 1979). *Babesia divergens* has also been reported to infect cattle and man in USA, however no case of piroplasmids of equids infecting man has been reported (Russel *et al*., 2005).

2.18.2 Trypanosomosis

The three African Animal Trypanosomes are considered to be nonpathogenic for humans. *T. b. brucei,* although not causing human disease, is closely related to *T. b. gambiense* and *T.b. rhodesiense*. The latter is the cause of human sleeping sickness, a very debilitating and often fatal disease considered to be of major public health significance in 36 sub-Saharan countries of west, central, and east Africa with 50 million people at risk. In west and central Africa, a chronic form of human sleeping sickness is caused by *T. b. gambiense,* which uses humans as its major host but also infects pigs. In east and southern Africa, *T. b. rhodensiense* is the cause of a much more acute form of human sleeping sickness. This trypanosome also infects cattle, bushbuck (*Tragelaphus scriptus*), and probably many other wild animals that may serve as reservoirs of the parasite. The first case of animal

trypanosome (*Trypanosoma evansi)* infection in humans was reported by Prashant *et al* (2005) although Truc *et al* (1998) had earlier reported mixed *Trypanosoma brucei* and *T. congolense* infection in human.

CHAPTER 3

MATERIALS AND METHODS

3.1 EXPERIMENTAL DESIGN

3.1.1 Study Area

The study was undertaken in Igabi and Kaduna North local goverment areas of Kaduna State, Nigeria. The state is located between latitudes 09'00 and 11'30 north of the equator and Longitude 30^{o} 00' east of greenwich meridian. The state has a landmass of about 48,473.2 square Kilometers, a population of 6million people and shares borders with the following tsetse flies endemic states of Bauchi, Plateau, Niger and Abuja federal capital territory and tsetse flies free states of Kano, Zamfara and Kastina. The climate of the state consists of distinct wet and dry seasons within the guinea savanna zone of Nigeria (KGIS, 2008).

3.1.2 Period of Sampling

Sampling was done over a period of 4 months. It began in february, 2007 with Kaduna north and ended in may, 2007. Blood samples were collected from horses between 8:00 am and 10: 00 am because of the diurnal variation in cortisol. The time was considered appropriate to get adequate assistance from grooms to assist in restrainting of the horses for physical examination and subsequent blood collection.

3.1.3 Sampling Method

An initial survey to estimate horse population showed that the state had 4000 horses with Igabi having a population of 250 horses and Kaduna North, 300 horses. Sample survey, using simple random ballot technique (Donald *et al.*, 2007) was used in this research. Resident polo horses in Kaduna state are owned by polo patron/players, and these were our targeted units for sampling. Two local government areas (LGAs), Kaduna North and Igabi were sampled at random.

3.1.4 Sample Collection

3.1.4.1 Identification of horses and samples

All horses sampled were identified with a combination of indices (a) local goverment area, (b) stable and (c) sample number. This represented initials of stable number (1), the local government area as source (Kn or Ig) and small serial number was used to identify all sampled materials and clinical findings from any horse for the purpose of reference to that horse. In this regard, Kaduna north is represented by Kn and Igabi by Ig. Thus, horses and materials samples from them had numbers ranging from $1Kn_1$,$2Kn_2$ .....(Kaduna North) and $1Ig_1$, $2IG_2$ .....(Igabi). Permanent marker pen was used to inscribe numbers on masking tape applied to the container of the sampled material

3.1.4.2 History, clinical and physical examination

The history of horses and management practice were duelly noted.The horses were examined at a distance and at close range while an assistant held the examined horse by the halter and in some cases had one of the fore limbs raised. Any sign of ill health and presence of external parasites were recorded on an examination data sheet.

3.1.4.3 Weight measurement

The WikiHow (2006) used a tape to weigh a horse. The method where the girth and length are measured and multiplied, was applied **: girth $(cm)^2$ x length = weight(kg).**

3.1.4.4 Body condition score

The six parts (neck, withers, shoulder, ribs, loin and tailhead) method to classify and score horses as described by Henneke (1998) was adopted.

3.1.4.5 Rectal temperature

Rectal temperature was determined with the aid of mercury-in-glass clinical thermometer, placed in the rectum with the bulb tilted to the side to ensure direct contact with rectal mucosa.The thermometer was left in place for 60 seconds and reading was obtained in degrees Celsius, (^{o}C). Value of rectal temperature below normal range of 37^{o}-38^{o}C was considered hypothermia while above the range was regarded as hyperthermia, i.e. fever (Wosu, 2002).

Plate II: Taking rectal tempreture (normal 37.8^{o}C – 38.5 ^{o}C) in a mare at Yerima stables Kaduna North Local Government Area

3.2.1 Blood Sampling

A total of 192 polo horses predominantly Arewa and their crosses, Argentines and Sudanese breeds were sampled from two local government areas of Kaduna state. Ten milliliters (10 ml) of whole blood was collected from each horse through jugular venipuncture with a 10 ml sterile disposable syringe and 18G needle. The site of blood collection was first cleaned with methylated spirit to reduce skin contaminants. Four (4) ml of the blood was emptied into 5 ml plastic sample bottle containing 1mg of ethylene di-amine tetra-acetate-K (EDTA-

K) as anticoagulant. The collected blood was used for the following; thin blood smear, hematocrit centrifugation (HCT), buffy coat examination (BCM) (Kihurani *et al.*, 1994), hemogram and total protein determination.

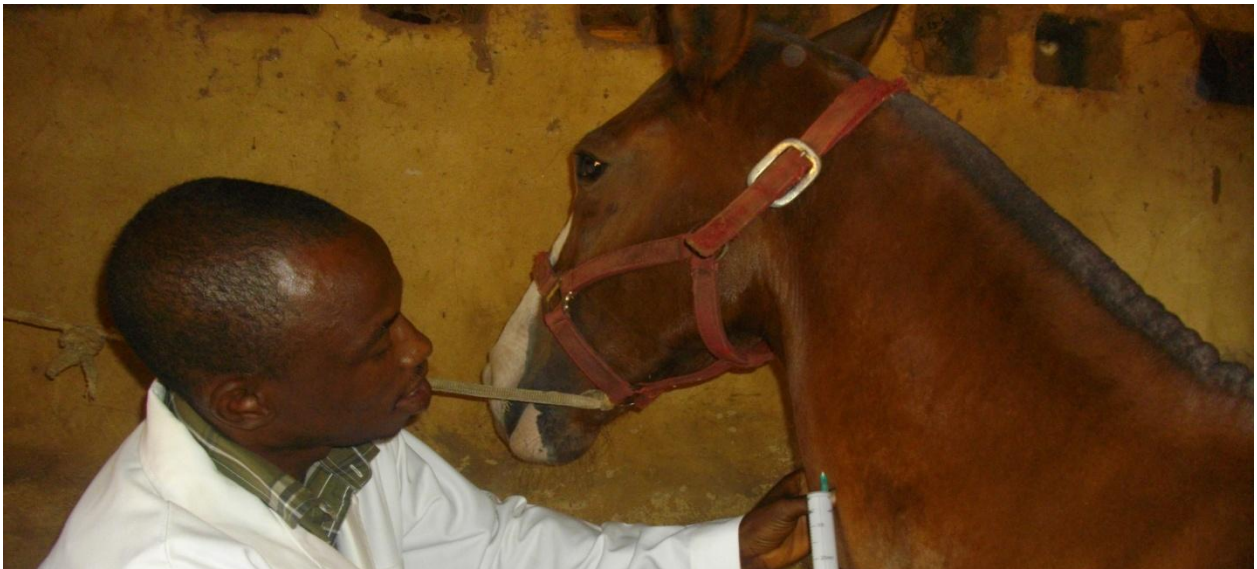


Plate III: Blood collection (5-10mls) in a mare at Yerima stables Kaduna North Local Government Area

3.2.2 Serum Preparation

The remaining 6ml of blood was emptied into sterile plain vacutainer bottle and then set on racks, arranged in a cold box for conveyance to the Nigerian Institute for Trypanosomiasis Research (NITR) laboratory in Kaduna, Kaduna where the samples were again kept at refrigeration temperature of 4 ^{o}C. The blood was allowed to clot and serum was decanted from the clotted blood, into clean serum-vials while poorly clotted samples were centrifuged in the Consultancy Services Unit laboratory at NITR. The serum samples were labeled and stored at –20^{o}C until used.

3.3 LABORATORY PROCEDURES

3.3.1 Hematological Analysis

Hematocrit centrifugation method, buffy coat method, wet film and thin blood film (Two-Slide Method) preparation, staining, examination and interpretation.

Thin blood films were made according to the method described by Israel and Douglas (1974). Hematocrit centrifugation method, buffy coat method and wet film as discribed by Woo (1960) were applied on blood samples. The hematocrit capillary tubes were read with hematocrit reader and observed under a microscope for parasites; smears were air-dried and fixed in absolute methanol. The fixed slides were immersed in a fresh prepared mixture of Giemsa stain and washed with 10ml of distilled water for parasite identification (Israel and Douglas, 1974). The stained slides were examined at x 1000 Bright-field light microscopy (Benjamin, 1978). The slides were examined systematically from one end of the film to another for blood picture including white blood cell differential counts, size, shape, pigmentation and blood parasites (Coles, 1974; Israel and Douglas, 1974).

Parasitaemia

Levels of parasitemia were scored as follows; where 1-5, 6-10,11-20 and >20 parasites are seen per microscopic field, such were represented by +, ++, +++ and ++++ respectively. If only an average of 1 parasite was seen per 3 microscope fields, it represented Very Few, VF (Lumsden *et al*., 1973).

3.3.2 Total Protein (TP g/dL)

This was determined with the aid of a refractometre in grammes per deciliter (g/dL) (Israel and Douglas, 1974).

3.3.3 Packed Cell Volume (PCV%)

This was determined using the Hematocrit Centrifuge Technique as described by Woo (1960).

3.3.4 Total and Differential Leucocytes Counts

These were determined using Haemocytometre and the stained thin blood film as described by Coles (1974).

3.3.5 Cortisol Assay

Cortisol values were determined by enzyme linked Immunosorbent Assay (ELISA) using BLK 96 ELISA kit for cortisol reference number BLK 1-1887 as discribed by users manual (2006).

3.4 DATA ANALYSIS

Prevalence = $\frac{\text{Total number of positive cases at a paticular time in an area}}{\text{Total number of population at risk at that paticular time in that area}}$ x100

The statistical package used was the statistical Package for social scientists (SPSS) version 16.0.

1. one simple t test: $Z = \frac{\sqrt{n}\bar{X}}{\sigma}$, where $\bar{X}$ is the sample mean of the data, *n* is the sample size, and σ is the population standard deviation of the data; *s* in the one-sample *t*-test is $\hat{\sigma}/\sigma$, where $\hat{\sigma}$ is the sample standard deviation.
2. Pearson Corellation (2-tail):- to find the relationship in the population

CHAPTER 4
RESULTS

4.1 PREVALENCE OF BABESIA AND TRYPANOSOME INFECTIONS IN POLO HORSES

Out of the one hundred and inity two Polo horses sampled, thirty six (18.75%) of these horses tested positive for babesia infection, one hundred and fifty six (81.25%) tested negative. The prevalence rate of babesia infection were (15) 15.60% in Igabi and (21) 21.90% in Kaduna North Local Government Areas (LGAs). Two species of *Babesia* parasites were identified: as a single infections namely *Babesia equi* 66.67% (24 cases) and *B. caballi* 33.33% (12 cases) in both LGAs.

Six (3.12%) of these horses tested positive for trypanosome infection and one hundred and eighty-six (96.83%) tested negative.The prevalence rate of trypanosome infection was (2) 2.80%% in Igabi and (4) 6.30% in Kaduna North LGAs. Two species of *Trypanosome*

parasites were identified: as a single infections namely *Trypanosoma evansi* (5 cases) 83.33% and *T. congolense* (1 case) 16.67% in both LGAs.

4.2 CLINICAL SIGNS

The clinical signs of babesia infected polo horses for stables 1 to 5 in Igabi and Kaduna North LGAs :

4.2.1 Fever

Overall, 36.00% (13 cases) of the babesia infected polo horses had fever out of which 13.90% (5 cases) occurred in Igabi and 19.40% (8 cases) in Kaduna North LGAs.

4.2.2 Congested Mucous Membrane

Overall, 61.00% (22 cases) of the babesia infected polo horses had Congested mucous membrane out of which 25.00% (9 cases) occurred in Igabi and 38.90% (14 cases) Kaduna North LGAs.

4.2.3 Emaciation

Overall, 63.90% (23 cases) of the babesia infected polo horses observed to be emaciated out of which 22.20% (8 cases) occurred in Igabi and 41.80% (15 cases) in Kaduna North LGAs.

4.2.4 Weakness

Overall, 47.20% (17 cases) of the babesia infected polo horses were Weak out of which 19.4%(7 cases) occurred in Igabi and 27.80%(10 cases) in Kaduna North LGAs.

4.2.4 Colic

Overall, 11.10% (4 cases) of the babesia infected polo horses had colic out of which 5.50% (2 cases) occurred in Igabi and 5.50% (2 cases) in Kaduna North LGAs.

4.2.5 Oedema

Overall, 58.30% (21 cases) of the babesia infected polo horses had oedema out of which 27.80% (10 cases) occurred in Igabi and 41.70% (15 cases) in Kaduna North LGAs.

4.2.6 Pale Mucous Membrane

Overall, 36.10% (13 cases) of the babesia infected polo horses had Pale mucous membrane out of which 13.90% (5 cases) occurred in Igabi and 22.20% (8 cases) in Kaduna North LGAs.

4.2.7 Ocular Discharge

Overall, 69.40% (25 cases) of the babesia infected polo horses had Ocular discharge out of which 27.80% (10 cases) occurred in Igabi and 41.70% (15 cases) in Kaduna North LGAs.

4.2.8 Nasal discharge

Overall, 80.50% (29 cases) of the babesia infected polo horses had nasal discharge out of which 33.30% (12 cases) occurred in Igabi and 47.20% (17 cases) in Kaduna North LGAs.

4.2.9 Icterus

Overall, 44.40% (16 cases) of babesia infected polo horses had icterus out of which 16.70% (6cases) occurred in Igabi and 27.80% (10 cases) in Kaduna North LGAs.

The clinical signs of trypanosome infected polo horses for stables 1 to 5 in Igabi and Kaduna North LGAs:

4.3 FEVER

Overall, 66.70% (4 cases) of trypanosome infected of polo horses had fever out of which 33.30% (2 cases) occurred in Igabi and 33.30% (2 cases) in Kaduna North LGAs.

4.3.1 Congested Mucous Membrane

Overall, 83.30% (5 cases) of trypanosome infected polo horses had congested mucous membrane out of which 16.60% (1 case) occurred in Igabi and 66.60% (4cases) in Kaduna North LGAs.

4.3.2 Emaciation

Overall, 50.00% (3 cases) of trypanosome infected polo horses were emaciated out of which 16.60% (1 case) occurred in Igabi and 33.30% (2 cases) in Kaduna North LGAs.

4.3.3 Weakness

Overall, 33.30% (2 cases) of trypanosome infected polo horses were weak out of which 0 % (no case) occurred in Igabi and 33.30% (2 cases) in Kaduna North LGAs.

4.3.4 Oedema

Overall 50.00% (3 cases) of trypanosome infected polo horses had oedema out of which 0 % (no case) occurred in Igabi and 50.00% (3 cases) in Kaduna North LGA.

4.4 PARASITAEMIA

The babesia and trypanosme parasitemias was generally low in both LGAs.The infection rates were higher in Kaduna North local government area than Igabi LGAs as represented in Table 1.

4.5 MEAN LIVE WEIGHT

4.5.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The overall mean live weight (Kg) ± standard error of mean (SEM) of babesia positive (476.92 ± 13.60Kg) was higher ($P<0.05$)than babesia negative (466.92 ± 9.18Kg) polo horses in Igabi and Kaduna North local Government Areas (LGAs) as presented in Table 2.

The mean live weight of babesia positive (495.50 ± 8.60Kg) polo horses in Igabi LGA is higher than the mean live weight of babesia positive (443.10 ± 6.20Kg) polo horses in Kaduna North LGA and the mean live weight of babesia negative (480.30 ± 5.40Kg) polo horses in Igabi LGA is higher than the mean live weight of babesia negative (443.10 ± 6.20Kg) polo horses in Kaduna North LGA. These shows that polo horses in stables 1 to 5 in Igabi LGA had higher mean live weight than polo horses in Kaduna North Local Government Areas.

The mean live weight of babesia positive and babesia negative polo horses in stables 1 to 5 in Igabi and Kaduna North LGAs where compared; in stables 1, 2, 3 and 5, the mean live weight of babesia positive groups for Igabi LGA was higher than babesia negative groups; only in stable 4, is the mean live weight of the babesia negative group higher than babesia positive group. In stables 2, 4 and 5, the mean live weight of the babesia negative groups in Kaduna North LGA is sligtly higher than babesia positive groups; in stables 1 and 3, the mean live weight of babesia positive groups is higher than babesia negative groups as presented in Figure 1.

4.5.2 Trypanosome Positive and Negative Groups in Igabi and Kaduna North LGAs

The overall mean live weight of trypanosome positive (451.10 ± 24.60Kg) was lower ($P \leq 0.05$) than trypanosome negative (461.90 ± 15.20Kg) polo horses in Igabi and Kaduna North LGAs as presented in Table 3.

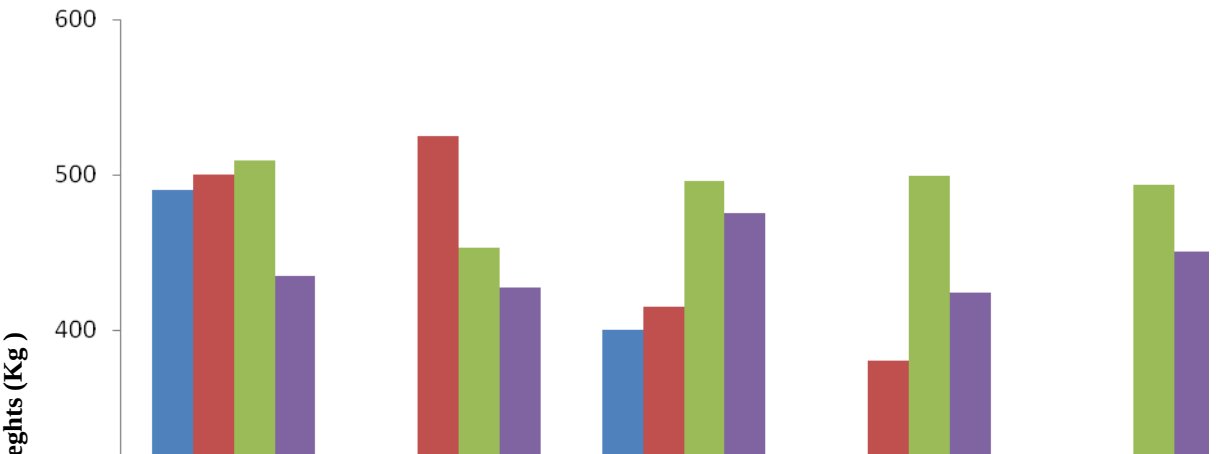
The mean live weight of trypanosome positive (460.00 ± 69.60Kg) polo horses in Kaduna North were higher than trypanosome positive (448.60 ± 48.30Kg) polo horses in Igabi LGA and the mean live weight of trypanosome negative (445.00 ± 63.60Kg) polo horses in Igabi LGA were higher than trypanosome negative (440.500Kg ± 50.3Kg) polo horses in

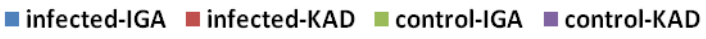
Kaduna North LGA. These show that polo horses in stables 1 to 5 in Igabi LGA had higher mean live weight than polo horses in Kaduna North LGA.

The mean live weight of trypanosome positive and trypanosome negative polo horses in stables 1 to 5 in Igabi and Kaduna North LGA were compared; in stables 1 and 3, the mean live weight of the trypanosome negative groups in Igabi LGA were higher than trypanosome positive groups; in stables 2, 4 and 5, no positive case was recorded only the mean live weight of the trypanosome negative groups was recorded. In stables 1 and 2, the mean live weight of trypanosome positive groups in Kaduna North LGA were higher than trypanosome negative groups; in stables 3 and 4, the mean live weight of trypanosome negative groups were higher than trypanosome infected groups; in stable 5, no positive case was recorded only the mean live weight of the trypanosoma negative group was recorded as presented in Figure 2.

The values in parenthesis represents the number of polo horses per stable

Figure 1: The mean live wieghts of babesia-positive and babesia-negative polo horses in Igabi and Kaduna North Local Government Areas





The values in parenthesis represents the number of polo horses per stable

Figure 2: The mean live wieghts of trypanosome positive and trypanosome negative polo horses in Igabi and Kaduna North Local Government Areas

4.6 MEAN BODY CONDITION SCORE

4.6.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The overall mean body condition score ± (SEM) of babesia positive (3.40 ± 0.20) were lower ($P \leq 0.05$) than the babesia negative (4.66 ± 0.10) polo horses in Igabi and Kaduna North LGAs as presented in Table 2.

The mean body condition score of babesia positive (3.80 ± 0.27) polo horses in Igabi LGA were higher than babesia positive (3.60 ± 0.32) polo horses in Kaduna North LGA and the mean body condition score of babesia negative (4.50 ± 0.25) polo horses in Igabi LGA were higher than babesia negative (4.30 ± 0.89) polo horses in Kaduna North LGA. These shows that polo horses in stables 1 to 5 in Igabi LGA had higher mean body condition score than polo horses in Kaduna North LGA.

The mean body condition score of babesia positive and babesia negative polo horses in stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stables 1, 2, 3, 4 and 5, the mean body condition score of babesia negative groups in Igabi LGA and Kaduna North LGA were generally higher than babesia positive groups as presented in Figure 3 .

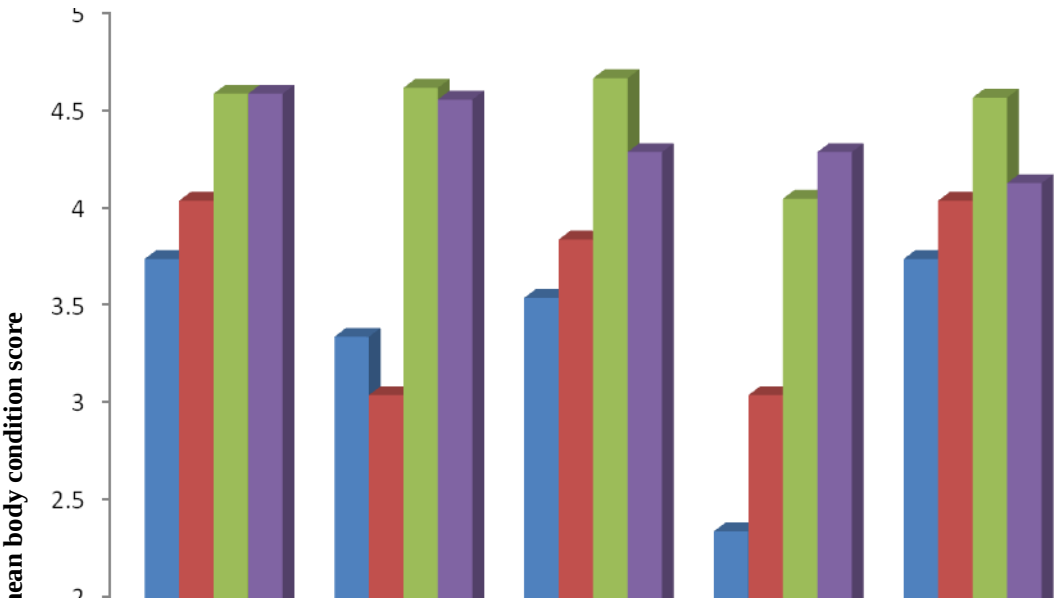
4.6.2 Trypanosome Positive and Negative Groups in Igabi and Kaduna North LGAs

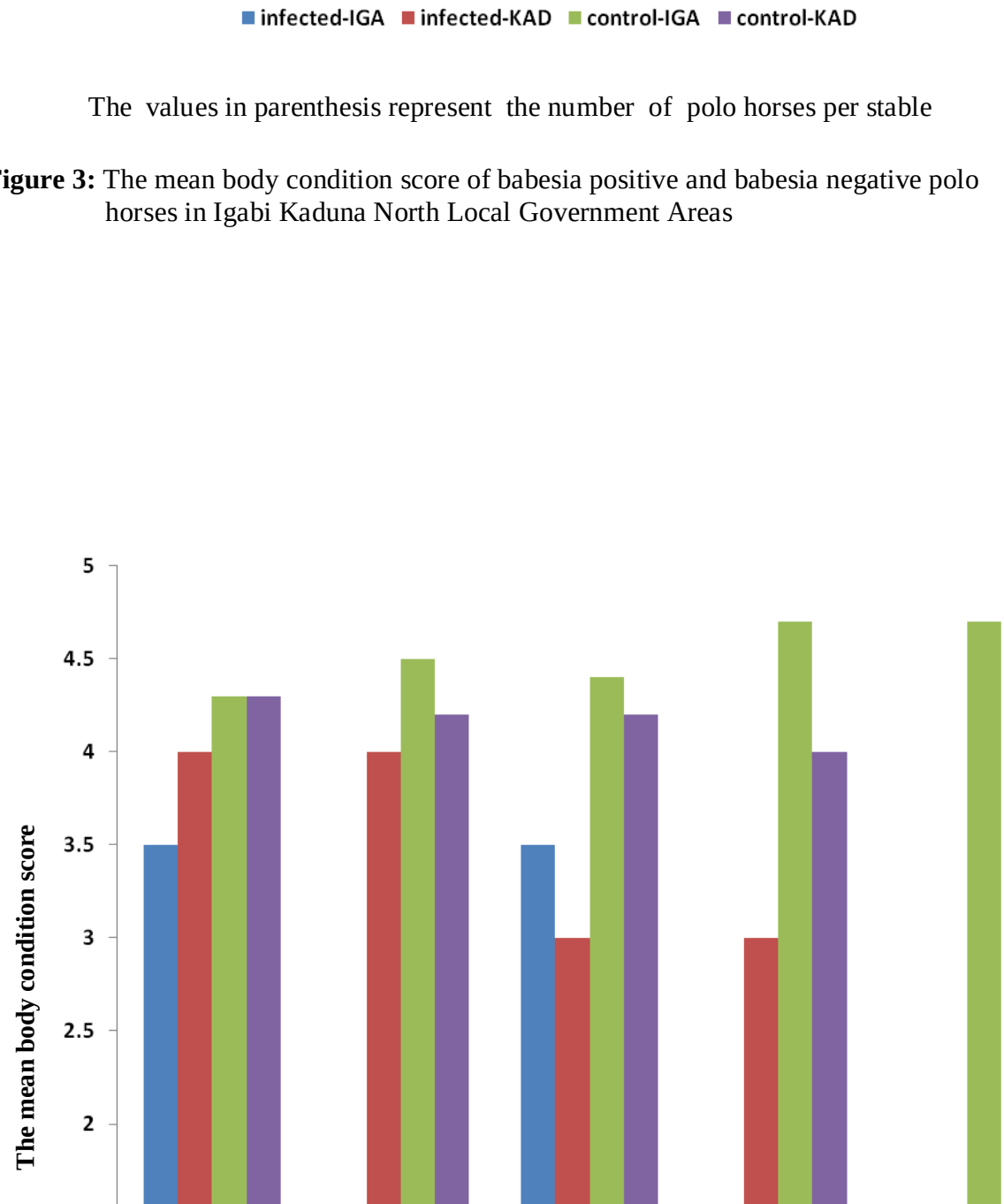
The overall mean body condition score of trypanosome positive (2.30 ± 0.70) polo horses were lower ($P<0.05$) than trypanosome negative (4.40 ± 0.07) polo horses in Igabi and Kaduna North LGAs as presented in Table 3.

The mean body condition score of trypanosome positive (2.80 ± 0.73) polo horses in Igabi LGA is slightly higher than trypanosoma positive (2.40 ± 0.82) polo horses in Kaduna North LGA and the mean body condition score of trypanosome negative (4.40 ± 0.83) polo horses in Igabi LGA was similar to trypanosome negative (4.50 ± 0.89) polo horses in Kaduna North LGA. These show that polo horses in stables 1 to 5 in Igabi LGA had higher mean body condition score than polo horses in Kaduna North LGA.

The mean body condition score of trypanosome positive and trypanosome negative polo horses for stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stables 1 and 3, the mean body condition score of trypanosome positive group in Igabi LGA was lower

than trypanosome negative groups; in stables 2, 4 and 5, no positive case was recored only the mean body condition score of the trypanosoma negative groups were recorded. In stables 1, 2, 3 and 4, the mean body condition score of trypanosome positive groups in Kaduna North LGA was lower than trypanosome negative groups; in stable 5, no positive case was recored only the mean body condition score of the trypanosome negative group was recorded as presented in Figure 4.





The values in parenthesis represent the number of polo horses per stable

Figure 3: The mean body condition score of babesia positive and babesia negative polo horses in Igabi Kaduna North Local Government Areas

The values in parenthesis represent the number of polo horses per stable

Figure 4: The mean body condition score of trypanosome positve and trypanosome negative polo horses in Igabi and Kaduna North Local Government Areas

4.7 MEAN PACKED CELL VOLUME

4.7.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The overall mean packed cell volumes of babesia positive (27.27 ± 0.85%) were lower ($P<0.05$) than babesia negative (34.4 ± 0.6%) polo horses in Igabi and Kaduna North LGAs as presented in Table 2.

The mean packed cell volumes of babesia infected (27.20 ± 1.85%) polo horses in Igabi LGA was higher than babesia infected (27.1 ± 0.8%) polo horses in Kaduna North LGA and the mean packed cell volumes of babesia negative (34.93 ± 4.61%) polo horses in Igabi LGA was higher than babesia negative (33.65 ± 0.55%) polo horses in Kaduna North LGA. These shows that polo horses in stables 1 to 5 in Igabi LGA had higher mean packed cell volumes than polo horses in Kaduna North LGA.

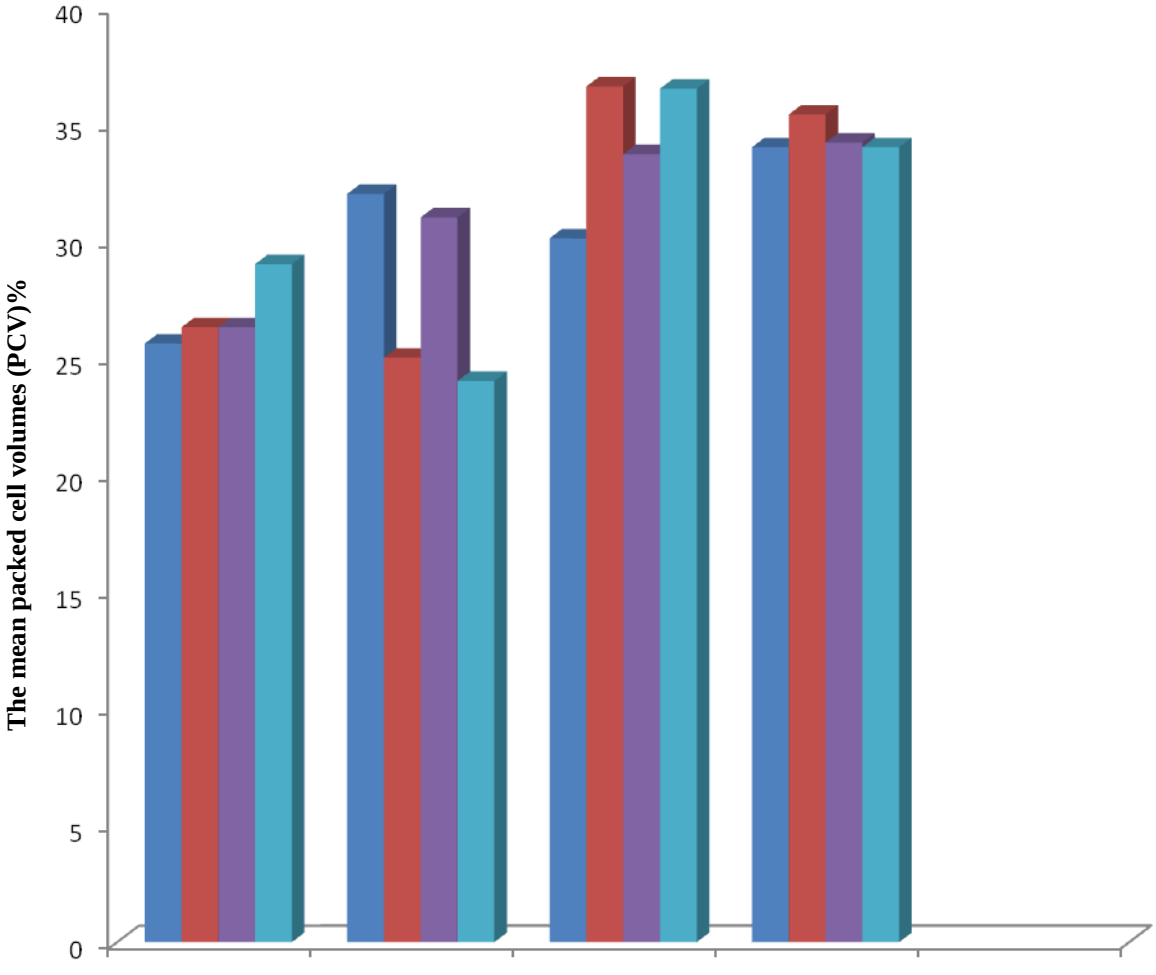
The mean packed cell volume of babesia positive and babesia negative polo horses in stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stables 1, 2, 3, 4 and 5, the mean packed cell volume of babesia positive group in Igabi and Kaduna North LGAs were generally lower than babesia negative groups as presented in Figure 5.

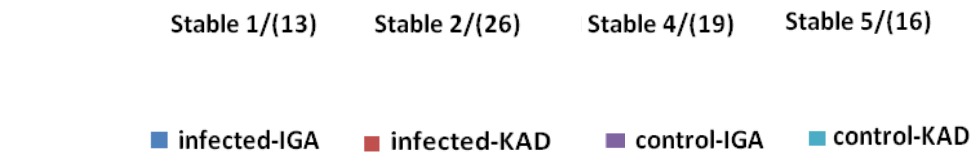
4.7.2 Trypanosome Positive and Negative Groups in Igabi and Kaduna North LGAs

The overall mean packed cell volumes of trypanosome positive (24.91 ± 0.85%) were lower ($P<0.05$)than trypanosoma negative (32.57 ± 0.73%) polo horses in Igabi and Kaduna North LGAs as presented in Table 3.

The mean packed cell volumes of trypanosome positive (29.50 ± 0.71%) in Igabi LGA was higher than trypanosome positive (25.16 ± 4.26 %) in Kaduna North LGA and mean packed cell volumes of trypanosome negative (33.80 ± 5.25%) in Igabi LGA were higher than trypanosome negative (32.71 ± 5.27%) in Kaduna North LGA. These show that polo horses in stables 1 to 5 in Igabi LGA had higher mean packed cell volumes than polo horses in Kaduna North LGA.

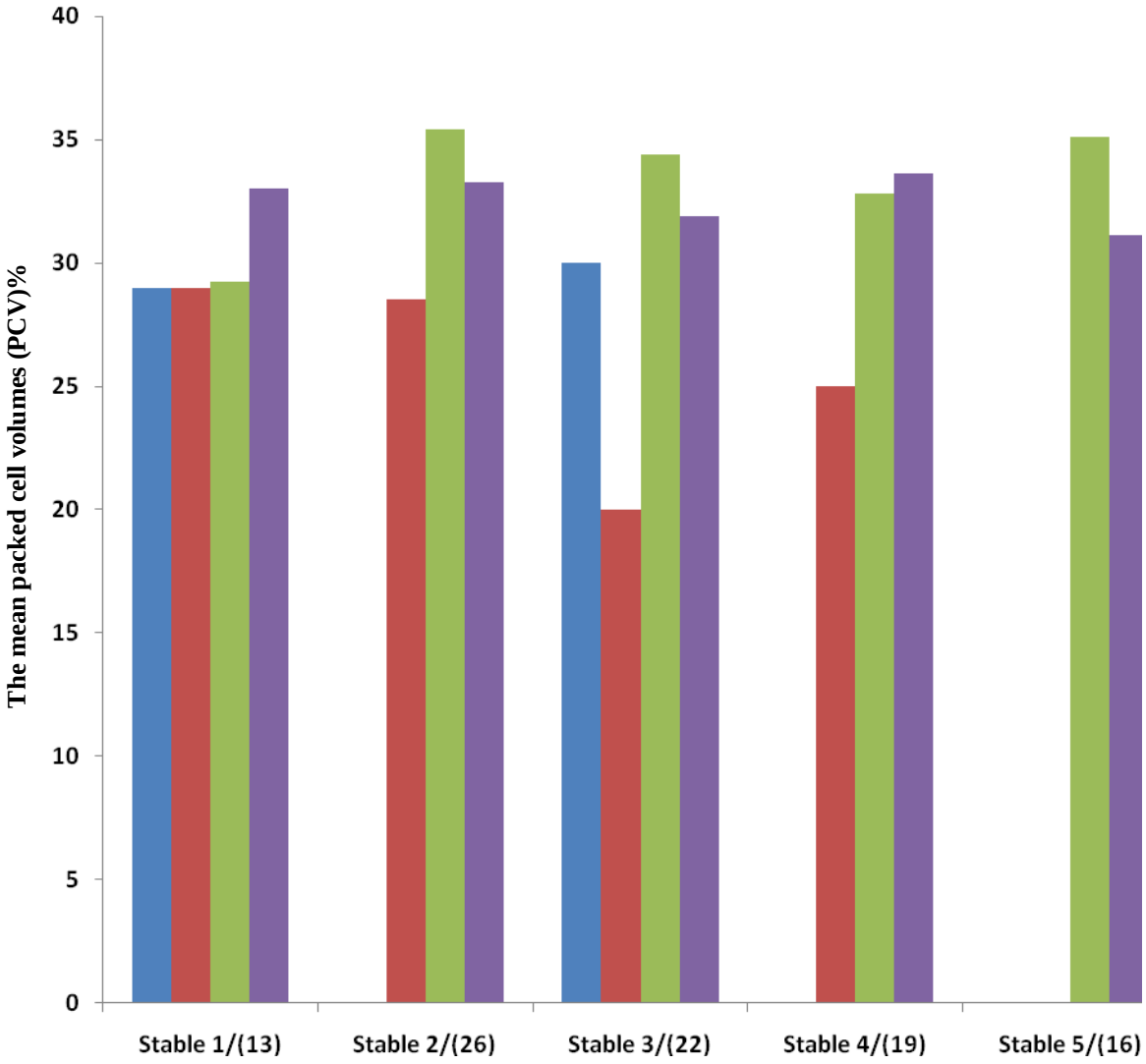
The mean packed cell volume of trypanosoma positive and trypanosome negative polo horses in stables 1 to 5 for Igabi and Kaduna North LGAs were compared; in stables 1 and 3, the mean packed cell volume of trypanosome positive groups in Igabi were lower than trypanosome negative groups;in stables 2, 4 and 5 no positive case was recored only the mean packed cell volume of trypanosome negative groups were recorded.In stables 1, 2, 3 and 4, in Kaduna North LGA the mean packed cell volume of trypanosome positive groups in Igabi were generally lower than trypanosome negative groups; in stable 5 no positive case was recored only the mean packed cell volume of trypanosome negative groups were recorded as presented in Figure 6.

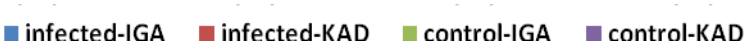

The mean packed cell volumes (PCV)%
40
35
30
25
20
15
10
5
0



The values in parenthesis represent the number of polo horses per stable

Figure 5: The mean packed cell volumes of babesia positive and babesia negative polo horses in Igabi and Kaduna North Local Government Areas





The values in parenthesis represent the number of polo horses per stable

Figure 6: The mean packed cell volumes of trypanosome positive and trypanosome negative polo horses in Igabi Kaduna North Local Government Areas

4.8 MEAN RED BLOOD CELL

4.8.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The overall mean red blood cell count of babesia positive (6.03 ± 0.16 $x10^9$ /L) were lower ($P<0.05$) than babesia negative (7.49 ± 2.12 $x10^9$ /L) polo horses in Igabi and Kaduna North LGAs as presented in Table 2.

The mean red blood cell count of babesia positive (6.46 ± 0.79 $x10^9$ /L) in Igabi LGA was higher than babesia positive (5.60 ± 1.03 $x10^9$ /L) polo horses in Kaduna North LGA and the mean red blood cell count of babesia negative (7.64 ± 0.90 $x10^9$ /L) polo horses in Igabi LGA was higher than babesia negative (7.17 ± 1.01 $x10^9$ /L) in Kaduna North LGA. These show that polo horses in stables 1 to 5 in Igabi LGA had higher mean red blood cell count than polo horses in Kaduna north LGA.

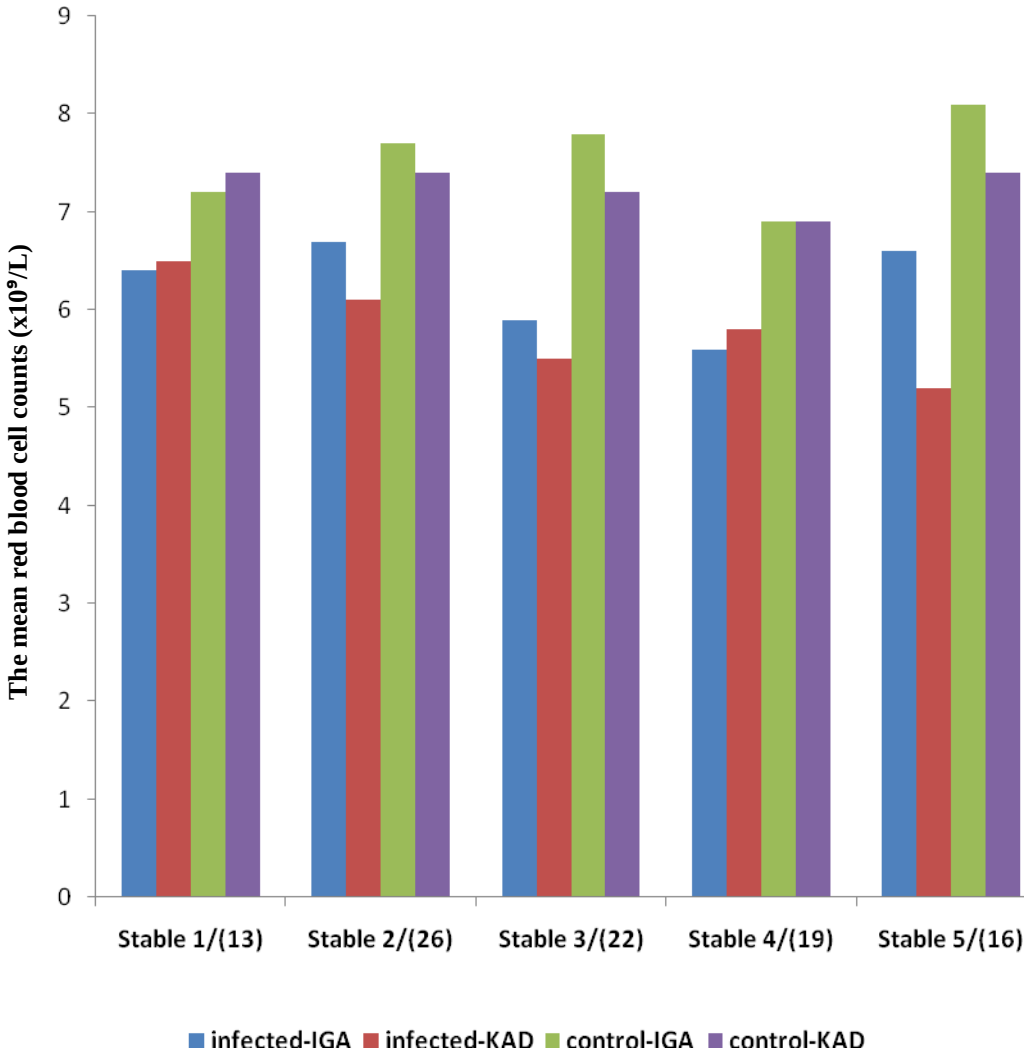
The mean red blood cell count of babesia positive and babesia negative polo horses for stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stables 1, 2, 3, 4, and 5, the mean red blood cell count of babesia positive groups in Igabi and Kaduna North LGAs were generally lower than babesia negative group in Igabi and Kaduna LGAs as presented in Figure 7.

4.8.2 Trypanosome Positive and Negative Groups in Igabi and Kaduna North LGAs

The overall mean red blood cell count of trypanosoma positive (6.02 ±0.27 $x10^9$ /L) were lower (P≤0.05) than trypanosoma negative (7.00 ± 0.14 $x10^9$ /L) polo horses in Igabi and Kaduna North LGAs.

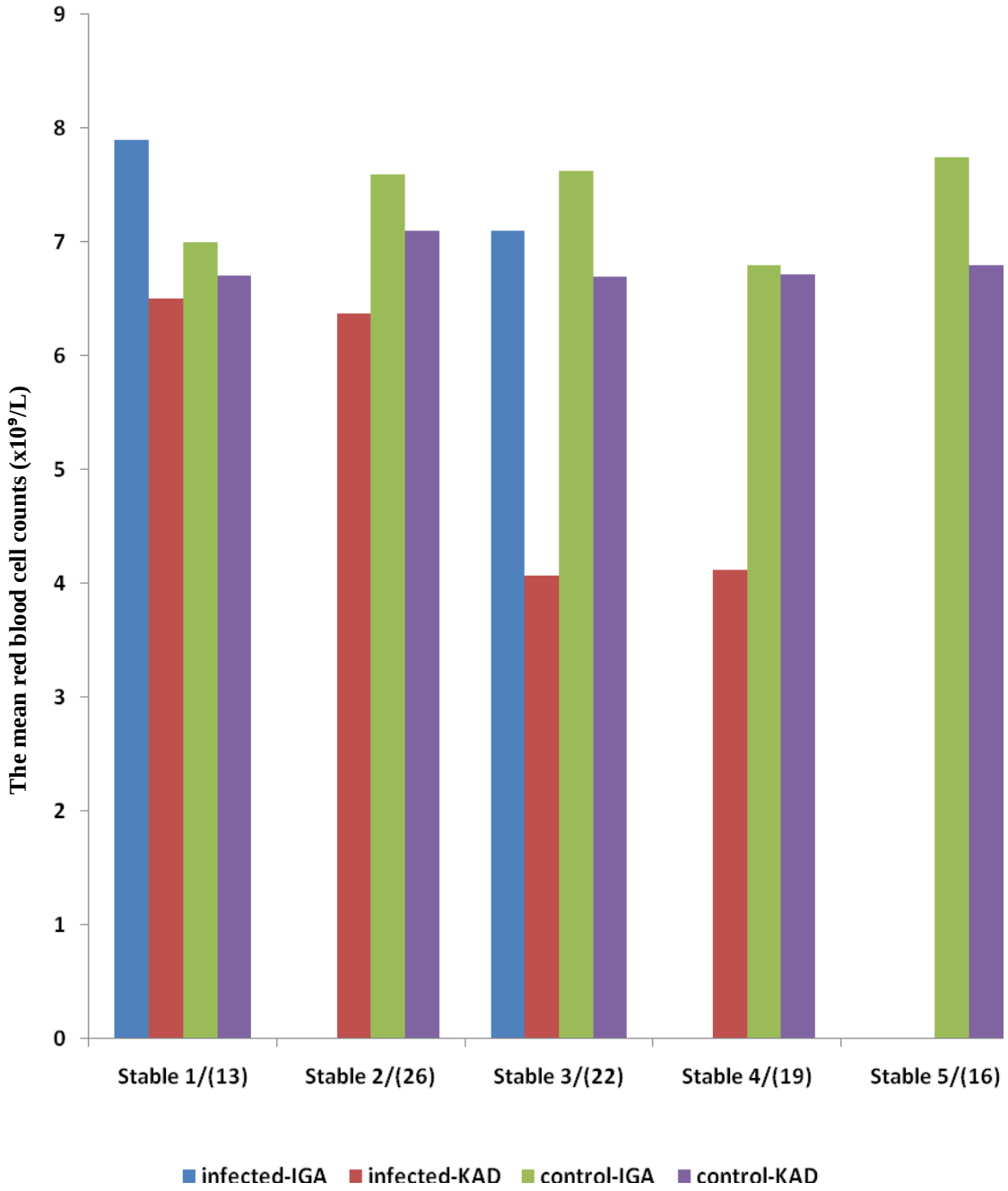
The mean red blood cell count of trypanosome positive (7.49 ± 0.51 $x10^9$ /L) polo horses in Igabi LGA were higher than trypanosoma positive (5.25 ± 1.28 $x10^9$ /L) polo horses in Kaduna North LGA and the mean red blood cell count of trypanosome negative (7.40±0.98 $x10^9$ /L) polo horses in Igabi LGA were higher than that of trypanosome negative (6.85 ± 1.16 $x10^9$ /L) polo horses in Kaduna North LGA. These show that polo horses in stables 1 to 5 in Igabi LGA had higher mean red blood cell count than polo horses in Kaduna North LGA.

The mean red blood cell count of trypanosome positive and trypanosome negative polo horses of stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stables 1 and 3, the mean red blood cell count of trypanosome positive groups in Igabi LGA was lower than trypanosome negative groups; in stables 2, 4 and 5, no positive case was recored only the mean red blood cell count of trypanosome negative groups were recorded. In stables 1, 2, 3 and 4, the mean red blood cell count of trypanosome positive groups in Kaduna north LGA were generally lower than trypanosome negative groups; in stable 5, no positive case was recored only the mean red blood cell count of trypanosome negative group was recorded as presented in Figure 8.



The values in parenthesis represent the number of polo horses per stable

Figure 7: The mean red blood cell counts of babesia positive and babesia negative polo horses in Igabi and Kaduna North Local Government Areas



The values in parenthesis represent the number of polo horses per stable

Figure 8: The mean red blood cell counts of trypanosome positive and trypanosome negative polo horses in Igabi and Kaduna North Local Government Areas

4.9 MEAN HAEMOGOLOBIN CONCENTRATION

4.9.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The overall mean haemoglobin concentration of babesia positive (9.02 ± 0.32 g/dL) were lower (P<0.05) than babesia negative (11.4 ±1.1 g/dL) horses in Igabi and Kaduna North LGAs.

The mean haemoglobin concentration of babesia positive (9.23 ±1.72 g/dL) polo horses in Igabi LGA were higher than babesia positive (8.73 ± 1.75 g/dL) polo horses in Kaduna North LGA and the mean haemoglobin concentration of babesia negative (11.58 ± 1.57g/dL) polo horses in Igabi LGA were higher than babesia negative (11.47 ± 1.35g/dL) polo horses in Kaduna North LGA. These show that polo horses in stables 1 to 5 in Igabi LGA had higher mean haemoglobin concentration than polo horses in Kaduna North LGA.

The mean hemoglobin concentration of babesia positive and babesia negative polo horses for stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stable 1, 2, 3, 4 and 5, the mean hemoglobin concentration of babesia positive groups in Igabi and Kaduna North LGAs were generally lower than babesia negative groups as presented in Figure 9.

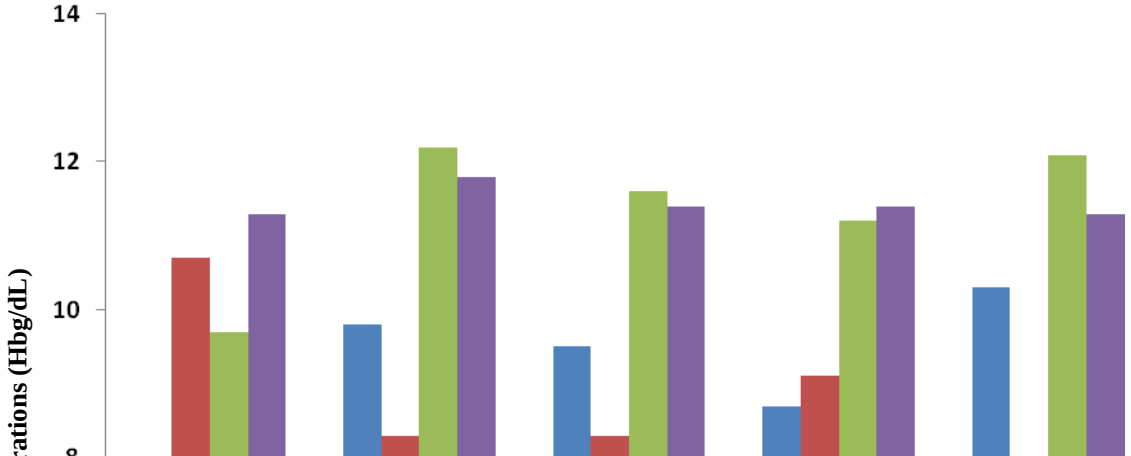
4.9.2 Trypanosome Positive and Negative Groups in Igabi and Kaduna North LGAs

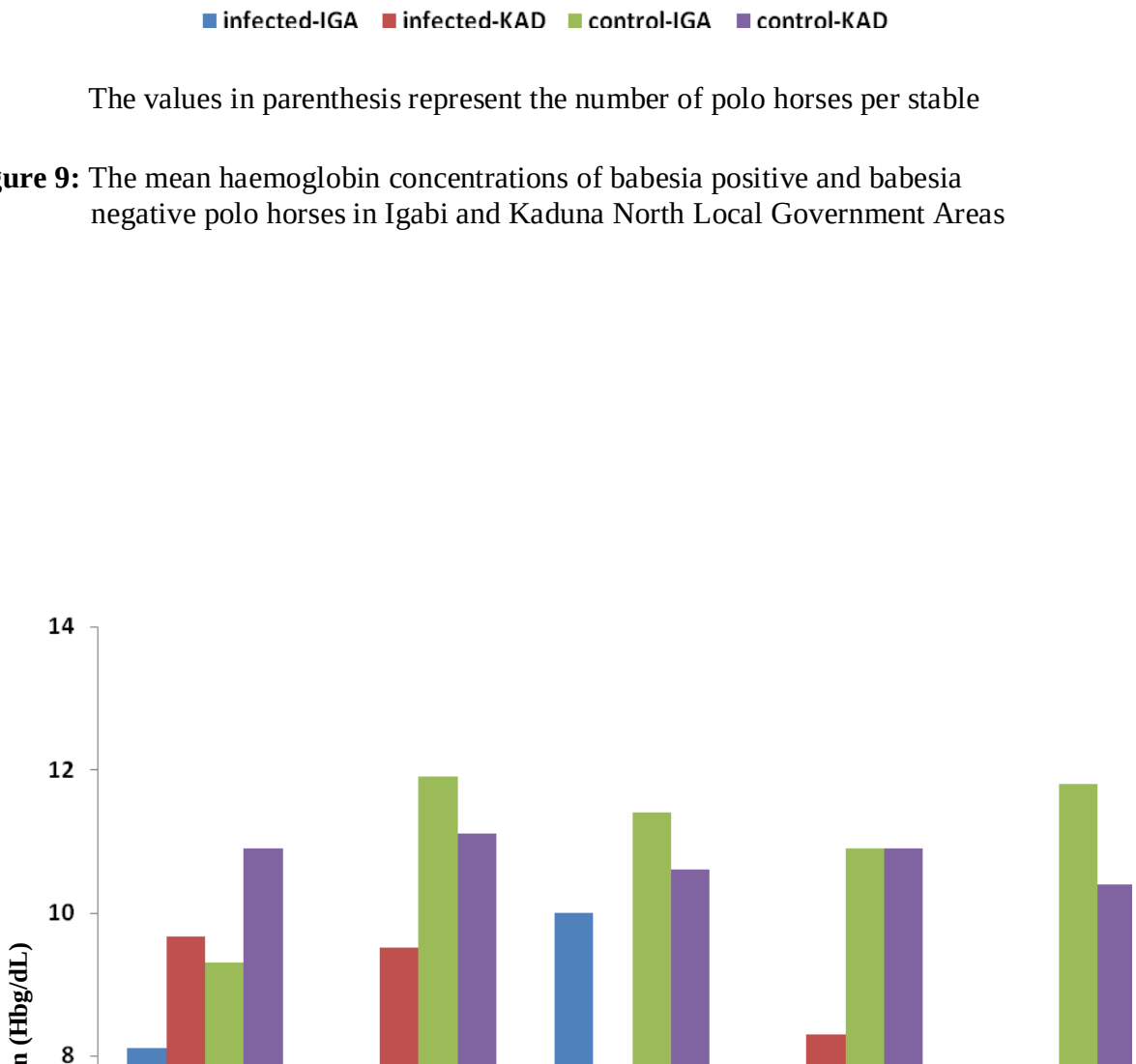
The overall mean haemoglobin concentration of trypanosoma positive (8.80 ± 0.52g/dL) polo horses was lower (P<0.05) than trypanosome negative (10.70 ± 0.30g/dL) polo horses in Igabi and Kaduna north LGAs as presented in Table 3.

The mean haemoglobin concentration of trypanosome positive (9.05 ± 1.34 g/dL) polo horses in Igabi LGA was slightly higher than trypanosome positive (8.39 ± 1.42 g/dL) polo

horses in Kaduna north LGA and the mean haemoglobin concentration of trypanosome negative (11.27 ± 1.83 g/dL) polo horses in Igabi LGA was slightly higher than trypanosome negative (10.85 ±1.85 g/dL) polo horses in Kaduna North LGA. These show that polo horses in stables 1 to 5 in Igabi LGA had higher mean haemoglobin concentration than polo horses in Kaduna North LGA.

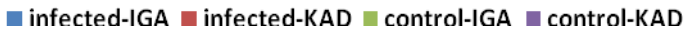
The mean haemoglobin concentration of trypanosoma positive and trypanosoma negative polo horses for stables 1 to 5 in Igabi and Kaduna North LGAs were compared;in stable 1 and 3, the mean haemoglobin concentration of trypanosome positive groups in Igabi was lower than trypanosome negetive groups;instables 2, 4 and 5, no positive case was recored only the mean haemoglobin concentration of trypanosome negetive groups were recorded.In stables 1, 2, 3 and 4, the mean haemoglobin concentration of trypanosome positive groups in Kaduna North LGA were generally lower than trypanosome negetive groups; instable 5, no positive case was recored only the mean haemoglobin concentration of trypanosome negetive groups were recorded as presented in Figure 10.





The values in parenthesis represent the number of polo horses per stable

Figure 9: The mean haemoglobin concentrations of babesia positive and babesia negative polo horses in Igabi and Kaduna North Local Government Areas



The values in parenthesis represent the number of polo horses per stable

Figure 10: The mean haemoglobin concentration of trypanosome positive and trypanosome negative polo horses in Igabi and Kaduna North Local Government Areas

4.10 WHITE BLOOD CELL COUNT

4.10.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The overall mean white cell count ($x10^9$ /L) ± (SEM) of babesia positive (6.11 ± 0.27$x10^9$ /L) polo horses were lower ($P<0.05$) than babesia negative (7.17 ± 2.15 $x10^9$ /L) polo horses in Igabi and Kaduna North LGAs as presented in Table2.

The mean white blood cell count of babesia positive (6.82 ± 1.03x10^9 /L) polo horses in Igabi was higher than babesia positive (5.41 ± 1.10x10^9 /L) polo horses in Kaduna North LGA and the mean white blood cell count of babesia negative (7.64 ± 1.20x10^9 /L) polo horses in Igabi LGA were higher than babesia negative (7.17 ± 0.01x10^9 /L) polo horses in Kaduna North LGA. These show that polo horses in stables 1 to 5 in Igabi LGA had higher mean white blood cell count than polo horses in Kaduna North LGA.

The mean White blood cell count of babesia positive and babesia negative Polo horses in stables 1 to 5 in Igabi and Kaduna North LGAs were compared;in stables I and 2, the mean White blood cell count of babesia infected groups for both LGAs were higher than babesia negative groups; in stables 3, 4, and 5, the mean White blood cell count of babesia positive groups in both LGAs were lower than babesia negative groups as presented in Figure 11.

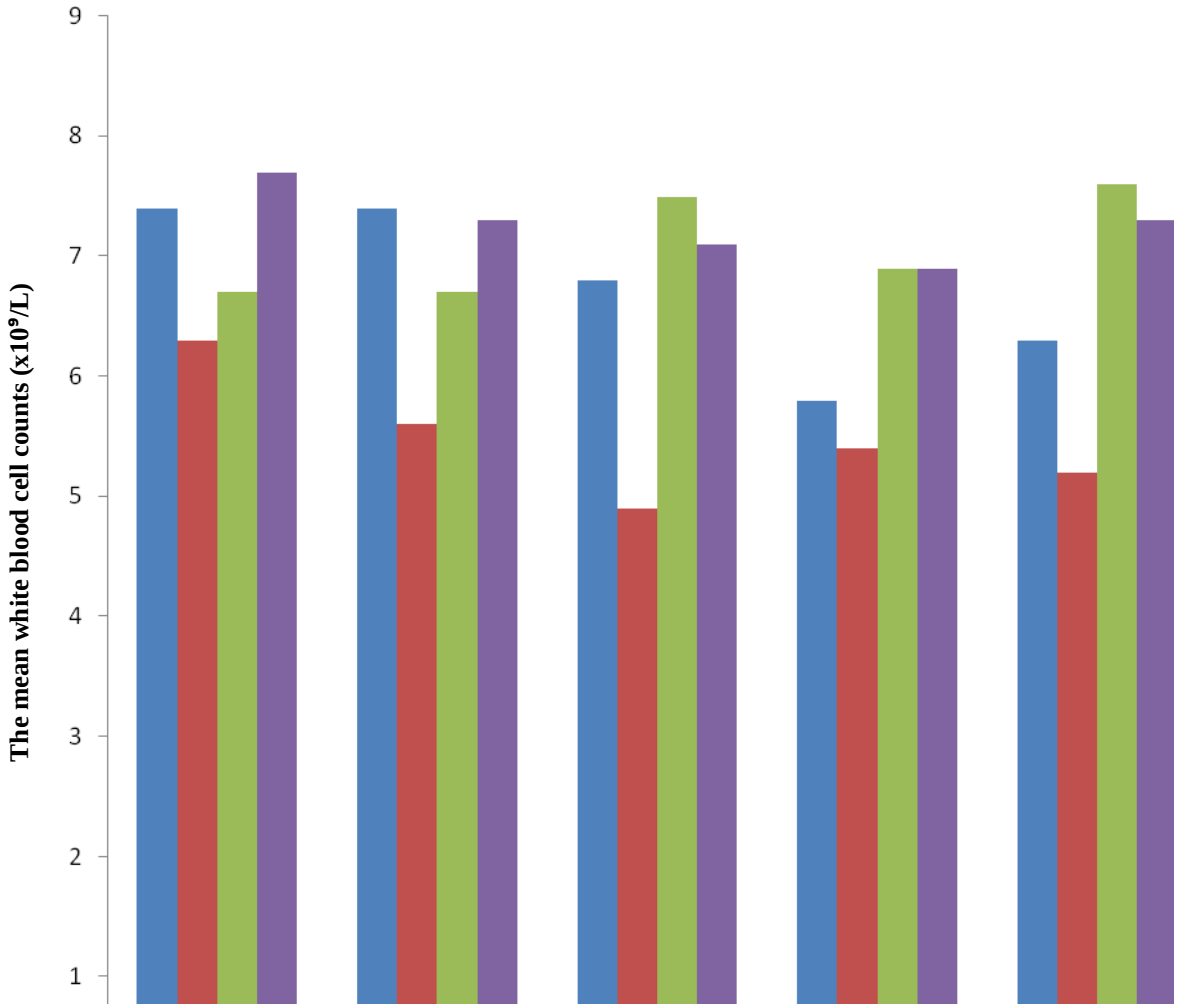
4.10.2 Trypanosome Positive and Negative Groups in Igabi and Kaduna North LGAs

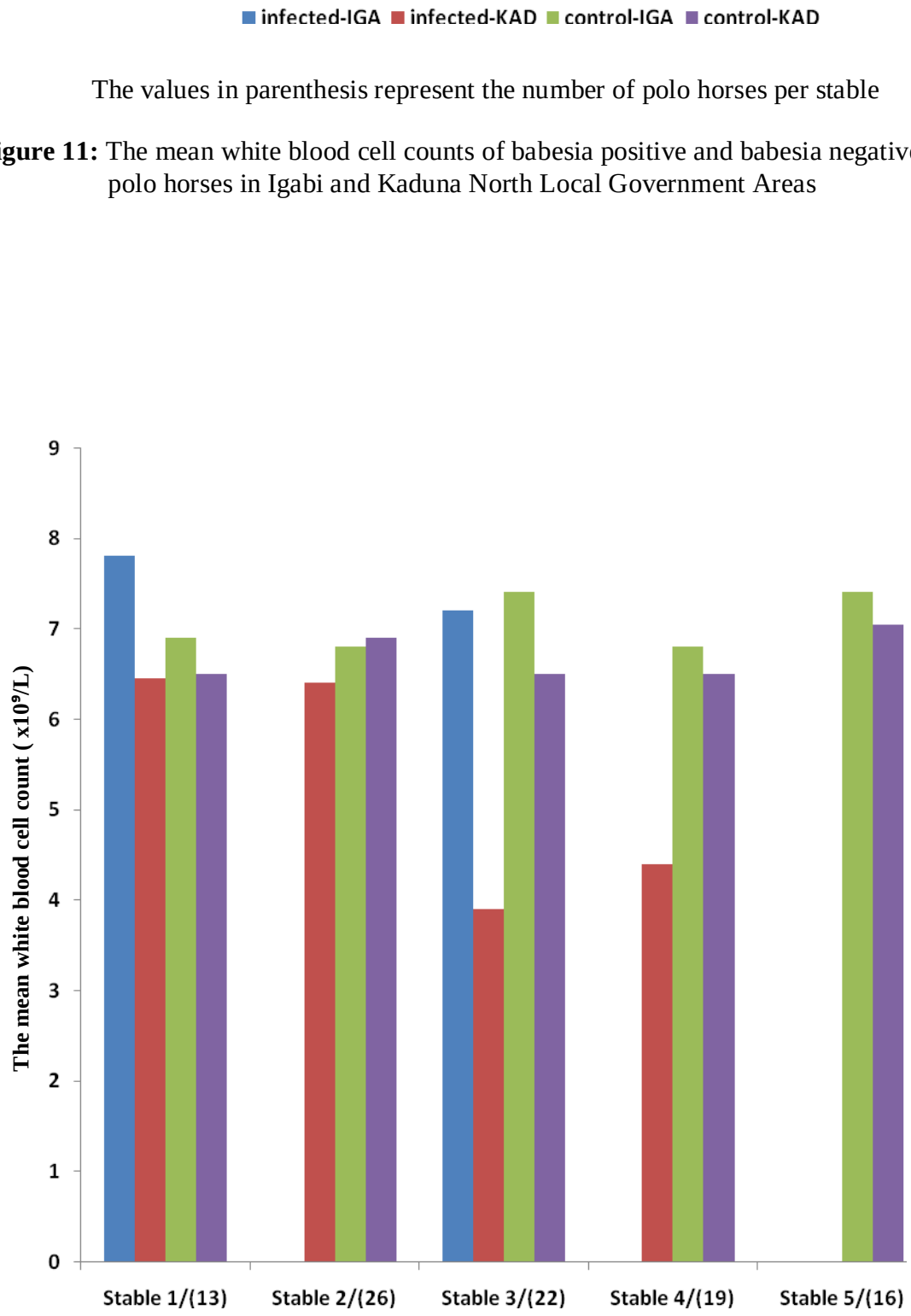
The overall mean white cell count of trypanosome (6.80 ± 0.14 x10^9 /L) polo horses was lower ($P<0.05$) than trypanosome negative (7.37 ± 1.11 x10^9 /L) polo horses in Igabi and Kaduna North LGA as represented in table 3.

The mean white cell count of trypanosome positive (7.49 ± 0.51 x10^9 /L) polo horses in Igabi LGA were higher than trypanosoma positive (5.25 ± 1.28 x10^9 /L) and the mean white blood cell count of trypanosome negative (7.09 ± 1.12 x10^9 /L) polo horses in Igabi LGA was lower than trypanosoma negative (8.56 ± 1.26 x10^9 /L) polo horses in Kaduna North LGA. These show that polo horses in stables 1 to 5 in Igabi LGA had higher mean white blood cell count than polo horses in Kaduna North LGA.

The mean white blood cell count of trypanosome positive and trypanosome negative polo horses in stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stable 1, the mean White blood cell count of trypanosoma positive group in Igabi was higher than

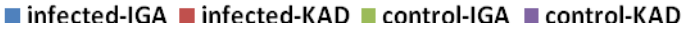
trypanosome negative group;instable 3 the mean White blood cell count of trypanosoma positive group lower than trypanosome negative group; in stables 2,4 and 5, no positive case was recored only the mean White blood cell count of the trypanosome negative group was recorded. In stable1, the mean White blood cell count of trypanosoma positive group inKaduna North LGA were the similer to trypanosome negative group;in stables 2, 3, and 4, the mean White blood cell count of trypanosome positive groups were lower than trypanosome negative groups; in stable 5, no positive case was recored only the mean White blood cell count of the trypanosome negative group was recorded as presented in Figure 12.





The values in parenthesis represent the number of polo horses per stable

Figure 11: The mean white blood cell counts of babesia positive and babesia negative polo horses in Igabi and Kaduna North Local Government Areas



The values in parenthesis represent the number of polo horses per stable

Figure 12: The mean white blood cell count of trypanosome positive and trypanosome negative polo horses in Igabi and Kaduna North Local Government Areas

4.11 MEAN TOTAL PROTEIN

4.11.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The overall mean total protein (g/dL) ± (SEM) of babesia positive (6.10 ± 0.30 g/dL) were slightly lower (P≥0.05) than babesia negative (6.50 ± 1.17 g/dL) horses in Igabi and Kaduna North LGAs as represented in Table 2.

The mean total protein of babesia positive (5.29 ± 0.44 g/dL) polo horses in Igabi LGA were slightly lower than babesia positive (5.6 ± 1.40 g/dL) polo horses in Kaduna North LGA and the mean total Protein of babesia negative (5.52 ± 1.99 g/dL) polo horses in Igabi LGA were slightly lower than babesia negative (6.46 ± 1.97 g/dL) polo horses in Kaduna North LGA. These shows that polo horses in stables 1 to 5 in Igabi LGA had higher mean total Protein than polo horses in Kaduna north LGA.

The mean total Protein of babesia positive and babesia negative polo horses for stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stables 1 and 5, the mean total Protein of babesia positive groups in Igabi LGA were higher than babesia negative groups ; in stables 2, 3 and 4, the mean total Protein of babesia positive groups were lower than

babesia negative groups. In stables 1, 2, 3 and 5 the mean total Protein of babesia positive groups in Kaduna North LGA were higher than babesia negative groups; in stable 4 the mean total Protein of babesia positive group were lower than babesia negative group as represented in Figure 13.

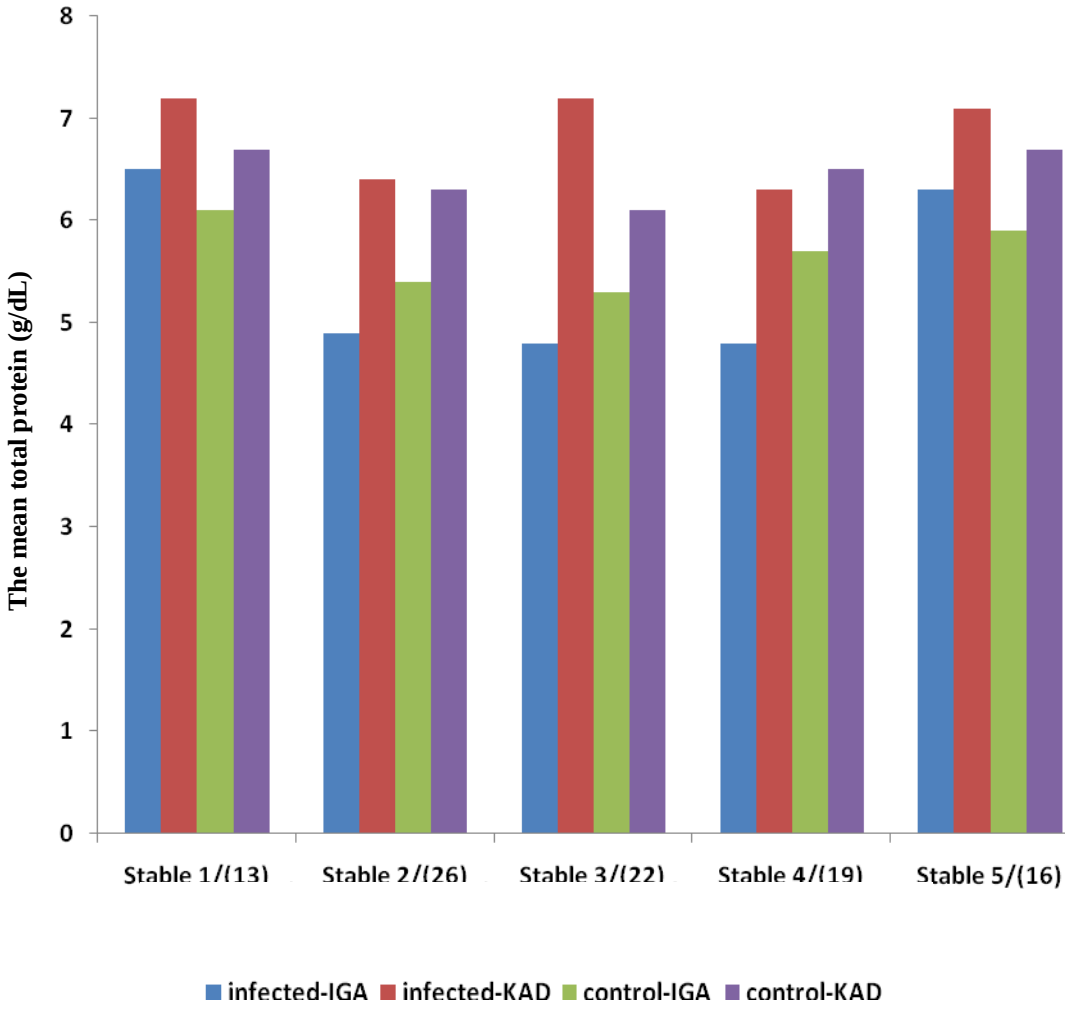
4.11.2 Trypanosome Positive and Negative Groups in Igabi and Kaduna North LGAs

The overall mean total protein of trypanosome positive (6.00 ±0.63 g/dL) polo horses was lower (P≥0.05)than trypanosome negative (6.10± 0.77g/dL) polo horses in Igabi and Kaduna North LGAs as represented in table 3.

The mean total protein of trypanosome positive (3.60 ± 1.04 g/dL) polo horses in Igabi LGA were lower than trypanosome positive (4.70 ± 1.20 g/dL) polo horses in Kaduna North LGA and the mean total protein of trypanosome negative (5.52 ± 0.39 g/dL) polo horses in Igabi LGA was lower than that of trypanosome negative (6.60 ± 0.10 g/dL) polo horses in Kaduna North LGA. These shows that polo horses in stables 1 to 5 in Igabi LGA had lower mean total protein than polo horses in Kaduna North LGA.

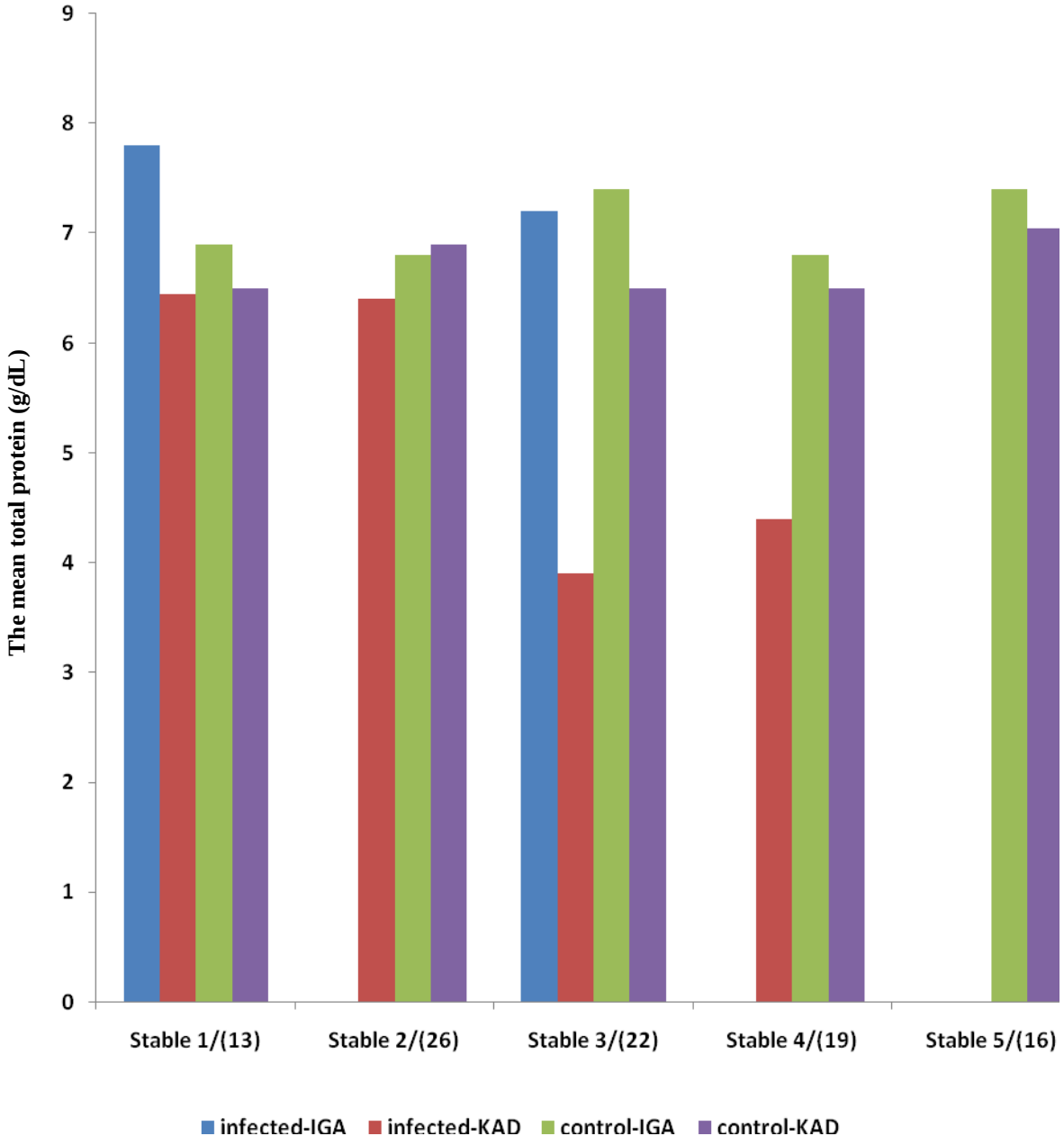
The the mean total Protein of trypanosome positive and trypanosome negative polo horses for stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stable 1, the mean total Protein of trypanosome positive group in Igabi LGA was higher than trypanosome negative group; in stables 3, the mean total Protein of trypanosome positive group was lower than trypanosome positive group; in stable 2, 4 and 5, no positive case was recored only the mean total Protein of trypanosome negative were recorded In stables 1, 2 and 3, the mean total Protein of trypanosome positive group in Kaduna North LGA was lower than

trypanosome negative group; in stable 4, the mean total Protein of trypanosome group was higher than trypanosome negative group; in stable 5, no positive case was recored only trypanosome negative group were recorded as represented in figure 14.



The values in parenthesis represent the number of polo horses per stable

Figure 13: The mean total protein values of babesia positive and babesia negative polo horses in Igabi and Kaduna North Local Government Areas



The values in parenthesis represent the number of polo horses per stable

Figure 14: The mean total protein of trypanosome positive and trypanosome negative polo horses in Igabi and Kaduna North Local Government Areas

4.12 MEAN SEGMENTED NEUTROPHILS

4.12.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The The overall mean segmented neutrophils count of babesia positive (7.54 ± 0.25 $x10^9$/L) polo horses were slightly higher (P≥0.05) than babesia negative (7.48 ± 0.66 $x10^9$/L) polo horses in Igabi and Kaduna North LGAs as represented in Table 4.

The mean segmented neutrophils count of babesia positive (7.54 ± 0.25 $x10^9$ /L) polo horses in Igabi LGA were slightly higher than babesia positive (7.20 ± 0.16 $x10^9$ /L) polo horses in Kaduna North LGA and the mean segmented neutrophils count babesia negative (7.48± 0.06 $x10^9$ /L) polo horses in Igabi LGA were slightly higher than babesia negative (7.20 ± 0.21 $x10^9$ /L) polo horses in Kaduna North LGA.

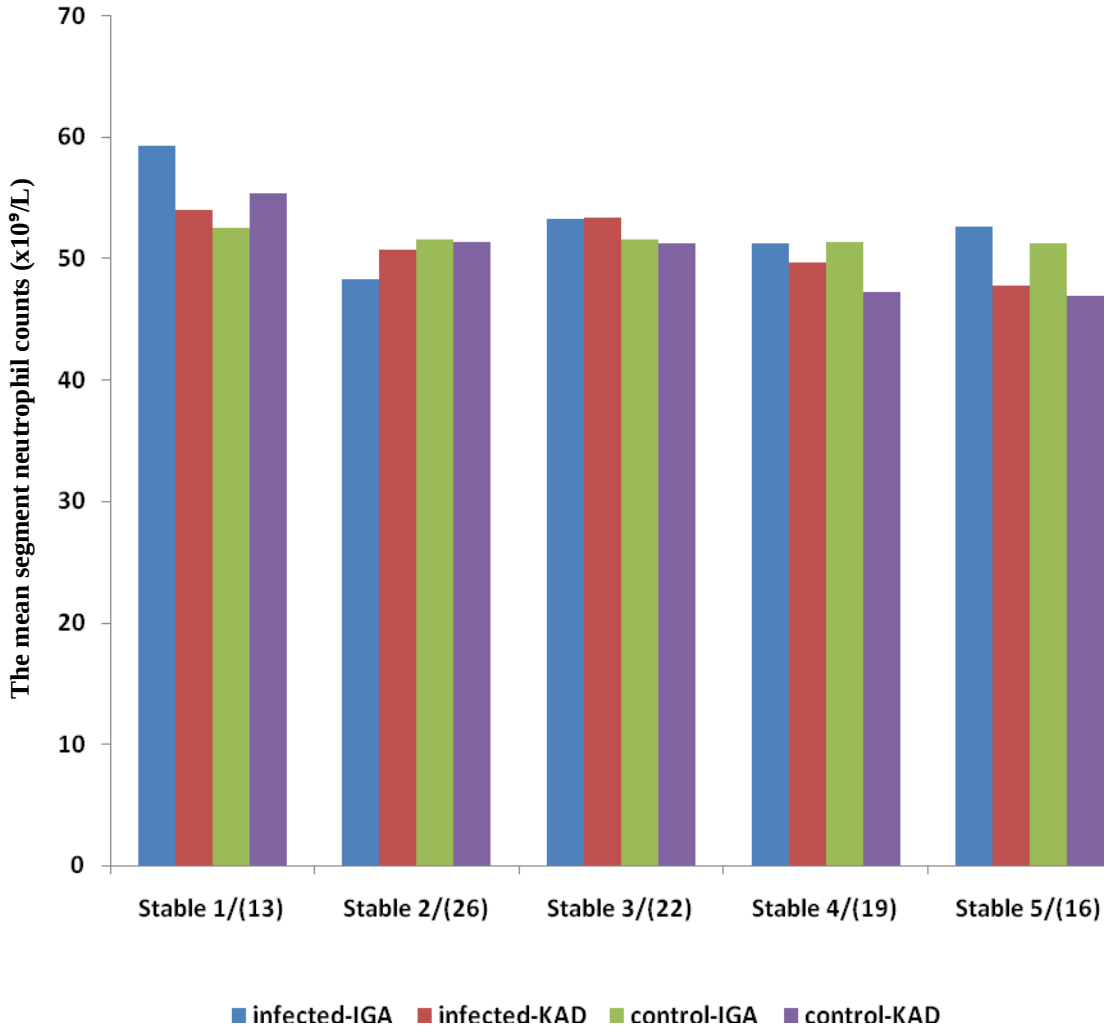
The mean segmented neutrophils of babesia positive and babesia negative Polo horses in stable 1 to 5 in Igabi and Kaduna LGAs were compared; in stables 1, 3 and 5, the mean segmented neutrophils count of babesia positive groups in Igabi LGA was higher than babesia negative groups; in stable 2, the mean segmented neutrophils count of babesia positive group was lower than babesia negative group; in stable 4 the mean segmented neutrophils count of both groups were similar. In stables 1 and 2, the mean segmented neutrophils of babesia positive groups in Kaduna North LGA were lower than babesia negative groups;in stables 3, 4 and 5, the mean segmented neutrophils count of babesia positive groups was higher than babesia negative groups as represented in figure15.

4.12.2 Trypanosome Positive and Negative Groups in Igabi and Kaduna North LGAs

The mean segmented neutrophils count of trypanosoma positive (5.60 ± 1.74 /L) polo horses was lower (P≤0.05) than trypanosome negative (7.36 ± 0.10 $x10^9$ /L) polo horses in Igabi and Kaduna North LGAs as represented in Table 5.

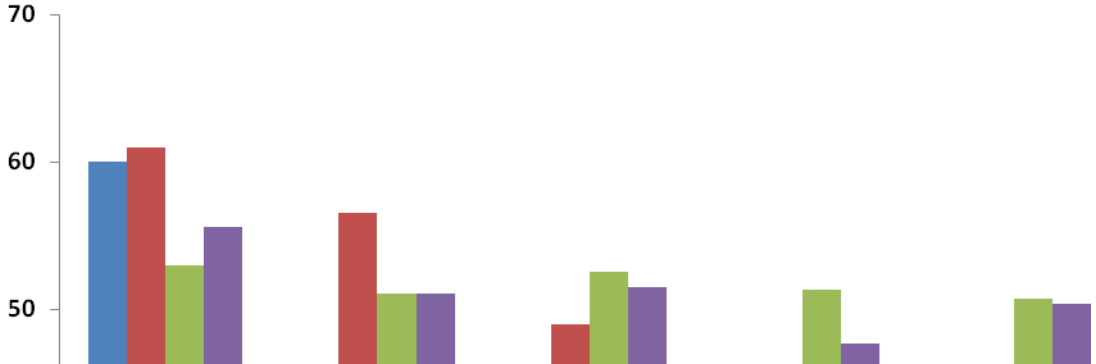
The mean segmented neutrophils of trypanosome positive (6.08 ± 1.56 $x10^9$ /L) polo horses in Igabi LGA was higher than trypanosoma infected (4.04 ±1.8 /L $x10^9$ /L) polo horses in Kaduna North LGA and the mean segmented neutrophils of trypanosome negative (7.30 ± 0.17 $x10^9$ /L) polo horses in Igabi LGA was higher than trypanosome negative (7.42 ± 0.0 4 $x10^9$ /L) polo horses in Kaduna North LGA.

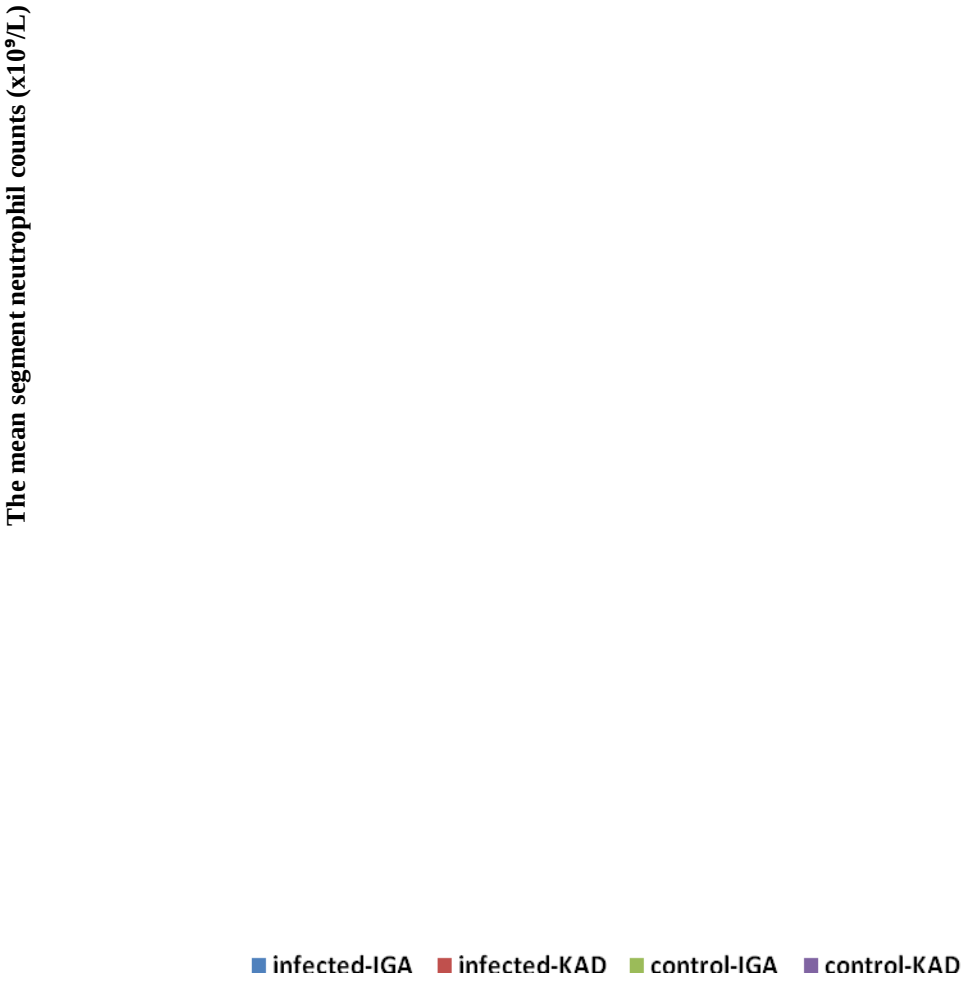
The mean segmented neutrophils of trypanosome positive and trypanosome negative polo horses in stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stable 1, the mean segmented neutrophils of trypanosome positive group in Igabi LGA was higher than trypanosome negative group; in stable 3, the mean segmented neutrophils of trypanosome positive group was lower than trypanosome negative group; in stables 2, 4 and 5, no positive case was recored only the mean segmented neutrophils of trypanosome negative groups were recorded.In stables 1 and 2, the mean segmented neutrophils of trypanosome positive groups in Kaduna North LGA were higher than the mean segmented neutrophils of trypanosome negative groups; in stables 3 and 4, the mean segmented neutrophils of trypanosome negative groups were higher than trypanosome positive groups; in stable 5, no positive case was recored only the mean segmented neutrophils of trypanosome negative was recorded as presented in figure 16.



The values in parenthesis represent the number of polo horses per stable

Figure 15: The mean segment neutrophil counts of babesia positive and babesia negative polo horses in Igabi and Kaduna North Local Government Areas





The values in parenthesis represent the number of polo horses per stable

Figure 16: The mean segment neutrophil counts of trypanosome positive and trypanosome negative polo horses in Igabi and Kaduna North Local Government Areas

4.13 MEAN MONOCYTE COUNT

4.13.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The mean Monocyte count of babesia positive ($0.06 \pm 0.02 \times 10^9$ /L) polo horses was higher ($P \geq 0.05$) than babesia negative ($0.04 \pm 0.01 \times 10^9$ /L) polo horses in Igabi and Kaduna North LGAs as represented in Table 4.

The mean Monocyte count of babesia positive ($0.06 \pm 0.03 \times 10^9$ /L) polo horses in Igabi LGA was similar to babesia positive ($0.06 \pm 0.02 \times 10^9$ /L) polo horses in Kaduna North LGA and the mean monocyte count of babesia negative ($0.02 \pm 0.01 \times 10^9$ /L) polo horses in Igabi LGA was similar to babesia negative ($0.06 \pm 0.01 \times 10^9$ /L) polo horses in Kaduna North LGA.

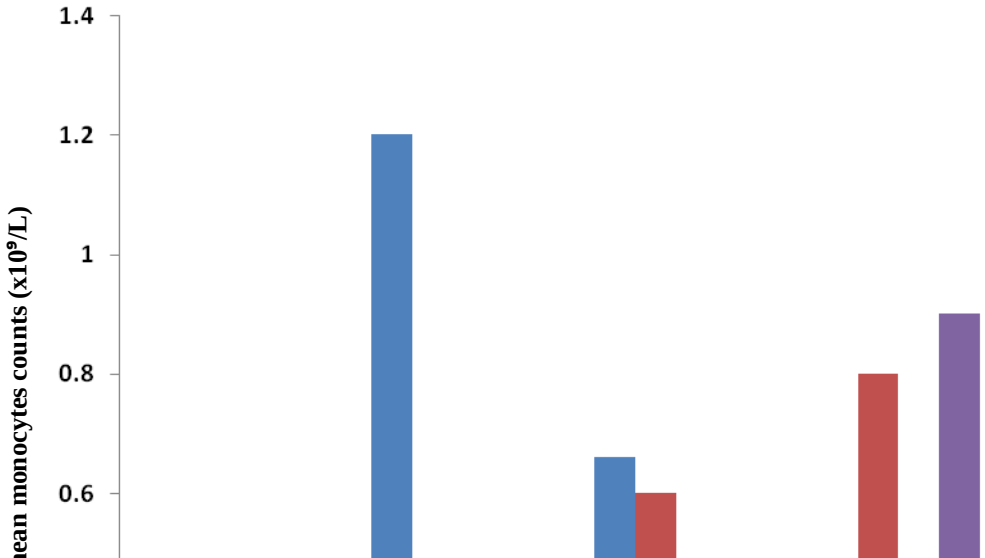
The mean monocytes count of babesia positive and babesia negative Polo horses for stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stable 1, the mean monocytes count of both positive and negative group in Igabi were not observed; in stables 2, 3 and 4, the mean monocytes count of babesia infected groups were higher than babesia negative groups; in stable 5, only the mean monocytes count of the babesia negative group was recorded the mean monocytes count of babesia positive group was not observed. In stable 1, only the mean monocytes count of the babesia negative group in Kaduna North LGA was recorded the mean monocytes count of babesia positive group was not recorded; in stables 2 and 4, the mean monocytes count of babesia positive groups were lower than babesia negative groups; in stables 3 and 5, the mean monocytes count babesia positive groups was higher than babesia negative groups as presented in Figure 17.

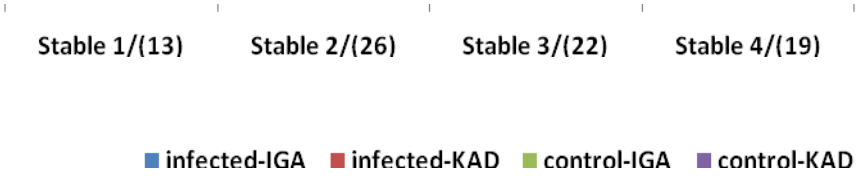
4.13.2 Trypanosome Positive and Negative Groups in Igabi and Kaduna North LGAs

The mean monocyte count of trypanosome positive ($0.03 \pm 0.03 \times 10^9$ /L) polo horses was lower ($P \leq 0.05$) than trypanosoma negative ($0.05 \pm 0.01 \times 10^9$ /L) polo horses in Igabi and Kaduna North LGAs as represented in table 5.

The mean monocyte count of trypanosome positive (0.06 ± 0.06 x10^9 /L) polo horses in Igabi LGA were higher than trypanosome positive (0.00 ±0.00 x10^9 /L) polo horses in Kaduna North LGA and the mean monocyte count of trypanosome negative (0.030 ± 0.01 x10^9 /L) polo horses in Igabi LGA was lower than trypanosome negative (0.06 ± 0.02 x10^9 /L) in Kaduna North LGA.

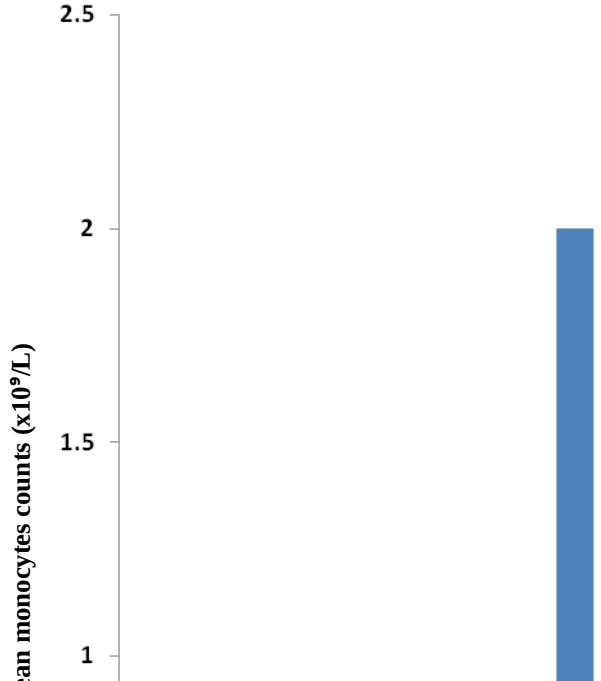
The mean monocytes count of trypanosome positive and trypanosome negative polo horses in Igabi and Kaduna North LGA were compared; in stable 1, the mean monocytes count of positive and negative groups in Igabi LGA were absent and only the mean monocytes count of negative group in Kaduna North LGA was recorded; in stable 2 ,only the mean monocytes count of negative groups was recorded in Igabi and Kaduna North LGAs; in stable 3, the mean monocytes count of trypanosome positive groups was higher than trypanosome negative groups in Igabi LGA and only the mean monocytes count of trypanosome negative group appeared the mean monocytes count of trypanosome positive groups in Kaduna North LGA was not observed; in stable 4 and 5, only the mean monocytes count of trypanosome negative groups appeared the mean monocytes count of trypanosome positive groups in Igabi and Kaduna North LGAs was not observed as presented in Figure 18.

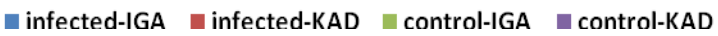




The values in parenthesis represent the number of polo horses per stable

Figure 17: The mean monocytes counts of babesia positive and babesia negative polo horses in Igabi and Kaduna North Local Government Areas





The values in parenthesis represent the number of polo horses per stable

Figure 18: The mean monocytes counts of trypanosome positive and trypanosome negative polo horses in Igabi and Kaduna North Local Government Areas.

4.14 MEAN EOSINOPHIL COUNT

4.14.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The mean eosinophil count of babesia positive ($0.08 \pm 0.03 \times 10^9$ /L) was higher ($P \geq 0.05$) than babesia negative ($0.06 \pm 0.01 \times 10^9$ /L) polo horses in Igabi and Kaduna North LGA as represented in Table 4.

The mean eosinophil count of babesia positive ($0.08 \pm 0.03 \times 10^9$ /L) polo horses in Igabi LGA was higher than babesia positive ($0.04 \pm 0.02 \times 10^9$ /L) in Kaduna North LGA and the mean eosinophil count of babesia negative ($0.06 \pm 0.01 \times 10^9$ /L) polo horses in Igabi LGA was lower than babesia negative ($0.8 \pm 0.2 \times 10^9$ /L) in Kaduna North LGA.

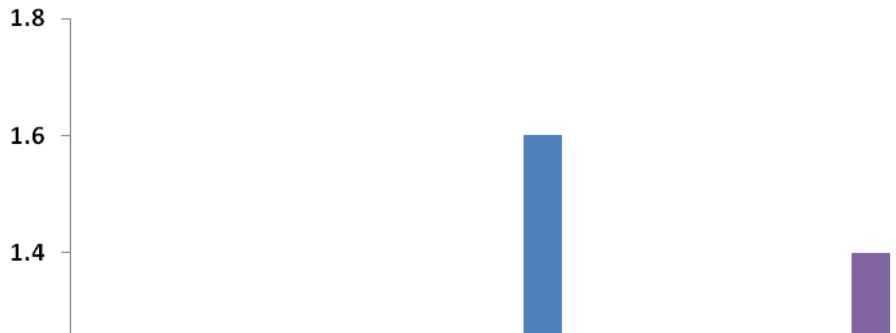
The mean eosinophils count of babesia positive and babesia negative polo horses of stables 1 to 5 in Igabi and Kaduna North were compared; in stables 1 and 2, the mean eosinophils count of babesia negative groups was higher than babesia positive groups for Igabi LGA; in stables 3 and 4, the mean eosinophils count of babesia positive groups in Igabi LGA were higher than babesia negative groups; in stable 5, only the mean eosinophils count of babesia negative was observed the mean eosinophils count of babesia positive was not observed. In stables 1 and 5, only the mean eosinophils count of the babesia negative group in Kaduna North LGA was observed the mean eosinophils count of babesia positive was not observed; In stables 2, 3 and 4, the mean eosinophils count of the babesia negative groups were higher than babesia positive groups as presented in Figure 19.

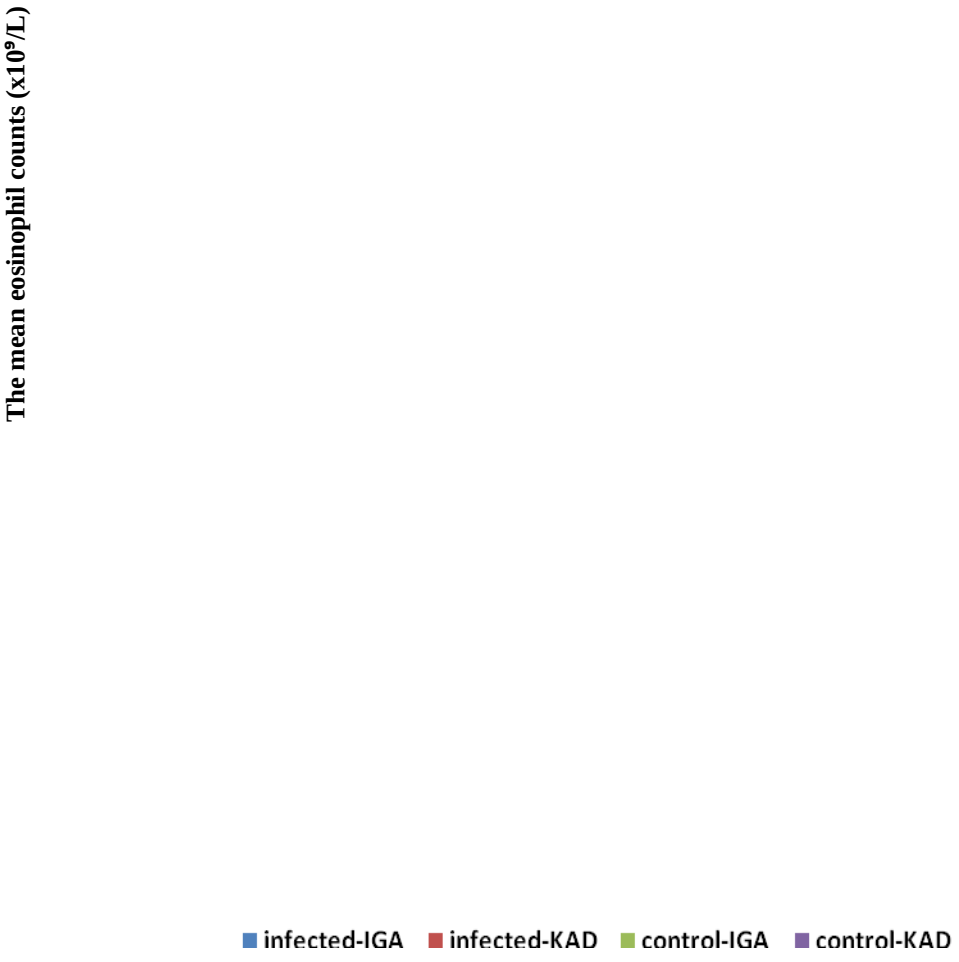
4.14.2 Trypanosome Positive and Negative Groups in Igabi and Kaduna North LGAs

The mean eosinophils count of trypanosome positive (0.03 ± 0.02 x10^9 /L) polo horses was lower (P≤ 0.05) than trypanosome negative (0.35 ± 0.05 x10^9 /L) polo horses in Igabi and Kaduna North LGAs as represented in Table 5.

The mean eosinophils count of trypanosome positive (0.05 ± 0.03 x10^9 /L) polo horses in Igabi LGA was higher than trypanosome positive (0.01 ±0.01 x10^9 /L) polo horses in Kaduna North LGA and mean eosinophils count of trypanosoma negative (0.60 ± 0.01 x10^9 /L) polo horses in Igabi LGA was higher than trypanosome negative (0.10 ± 0.03 x10^9 /L) polo horses in Kaduna North LGA.

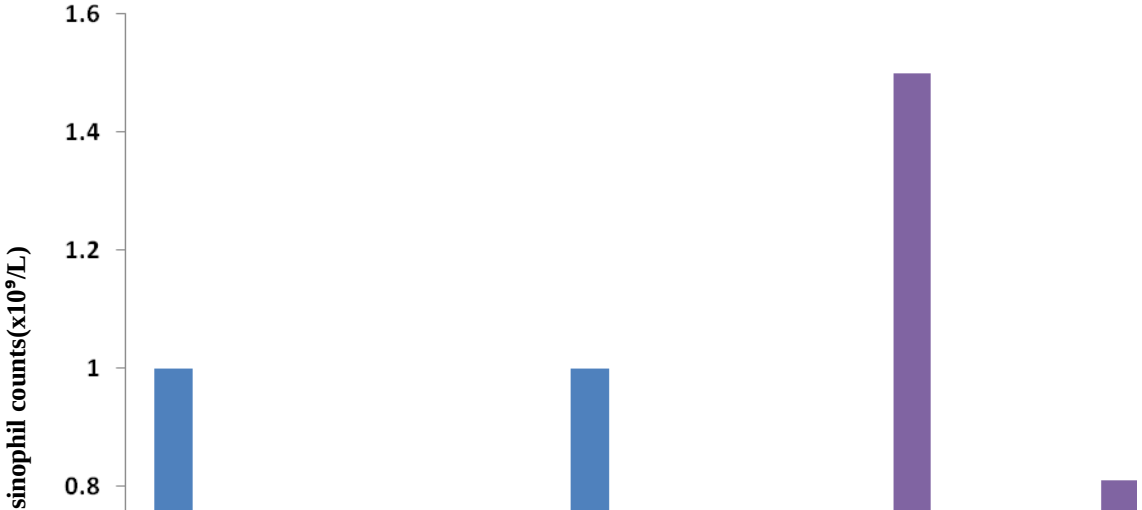
The mean eosinophils count of trypanosome positive groups and trypanosome negative groups in Igabi and Kaduna North LGAs were compared; in stables 1 and 3, the mean eosinophils count of trypanosome positive groups in Igabi LGA were higher than trypanosome negative groups; in stables 2, 4 and 5, the mean eosinophils count of trypanosome positive groups in Igabi and Kaduna North LGAs were not observed. In stable 3 the mean eosinophils count of the trypanosome negative group in Kaduna North LGA was higher than trypanosome positive groups; in stables 1, 2, 4 and 5 only the mean eosinophils count of the trypanosome negative group appeared the mean eosinophils count for trypanosome positive group in Kaduna North LGA was not observed as presented in Figure 20.





The values in parenthesis represent the number of polo horses per stable

Figure 19: The mean eosinophil counts of babesia positive and babesia negative polo horses in Igabi and Kaduna North Local Goverment Areas



The values in parenthesis represent the number of polo horses per stable

Figure 20: The mean eosinophil counts of trypanosome positive and trypanosome negative polo horses in Igabi and Kaduna North Local Government Areas

4.15 MEAN LYMPHOCYTES

4.15.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The mean lymphocyte count of babesia positive (6.65 ± 0.17 $x10^9$ /L) polo horses was similar (P≥0.05) to babesia negative (6.60 ± 0.24 $x10^9$ /L) polo horses in Igabi and Kaduna North LGAs as represented in Table 4.

The mean lymphocyte count of babesia positive (6.90 ± 0.14 $x10^9$ /L) polo horses in Igabi LGA were higher than babesia positive (6.40 ± 0.21 $x10^9$ /L) polo horses in Kaduna North LGA and the mean lymphocyte count of babesia negative (6.60 ± 0.10 $x10^9$ /L) in Igabi LGA was lower than mean lymphocyte count of babesia negative (6.60 ± 0.18 x 10^9 /L) polo horses in Kaduna North LGA.

The mean lymphocytes count of babesia positive and babesia negative in stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stables 1, 3, 4 and 5, the mean lymphocytes count of babesia positive groups were lower than babesia negative groups; in stables 2, the mean lymphocytes count of babesia positive group in Igabi LGA was higher than babesia negative group. The mean lymphocytes count of babesia positive and babesia negative groups in Kaduna North LGA were compared; in stables 1, 2 and 5, the mean lymphocytes count of babesia positive groups were higher than babesia negative groups; in stables 3 and 4, the mean lymphocytes count of babesia positive groups lower than babesia negative groups as presented in Figure 21.

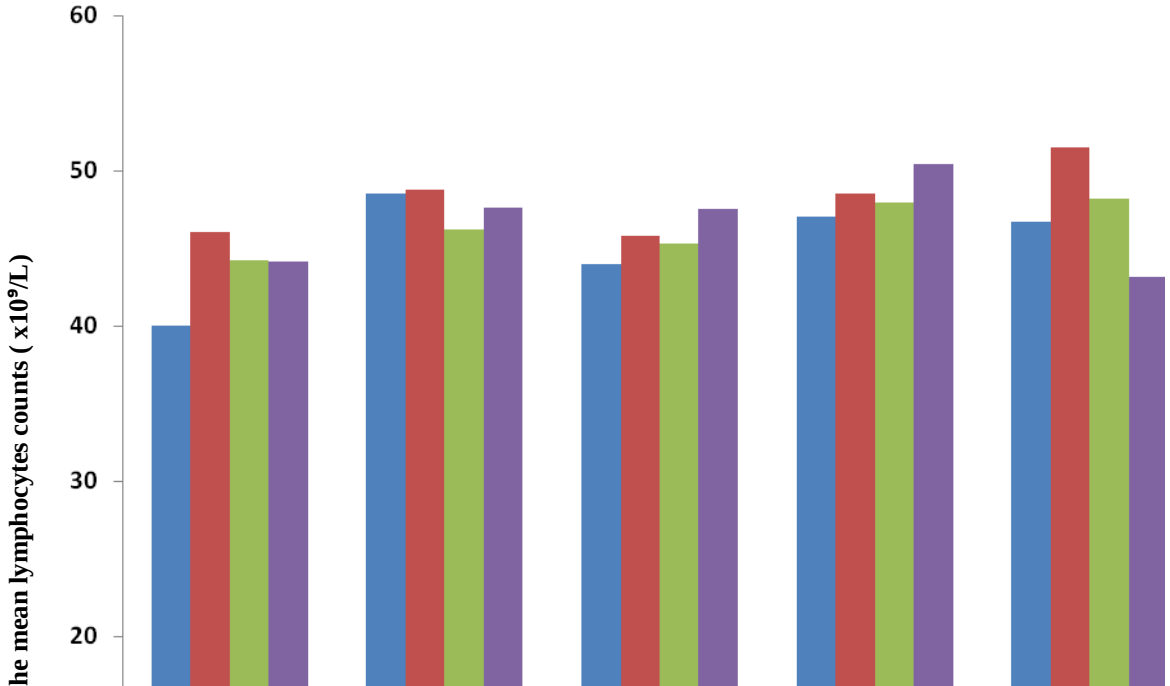
4.15.2 Trypanosome Positive and Negative Groups in Igabi and Kaduna North LGAs

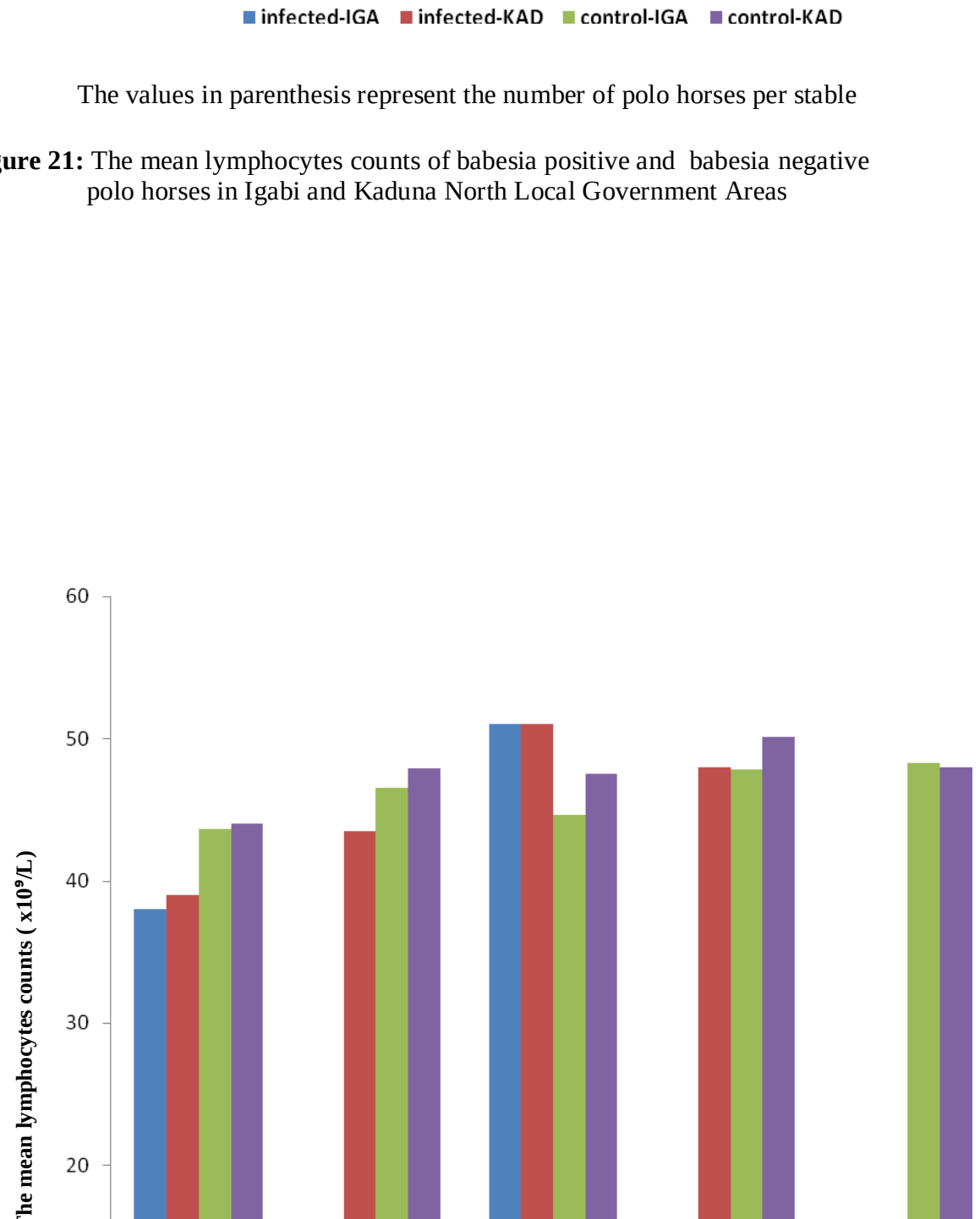
The mean lymphocyte count trypanosome positive (4.5 ± 1.4 $x10^9$ /L) was lower ($P \leq 0.05$) than trypanosome negative (6.5 ± 1.6) polo horses in Igabi and Kaduna North LGAs as represented in Table 5.

The mean lymphocyte count of trypanosome positive (5.1 ± 1.3 $x10^9$ /L) polo horses in Igabi LGA was higher than trypanosome positive (4.1 ±1.5 $x10^9$ /L) polo horses in Kaduna North

LGA and the mean lymphocyte count of trypanosome negative (6.3 ± 0.13 x10^9 /L) polo horses in Igabi LGA was lower than trypanosome negative (6.8 ± 1.5 x10^9 /L) polo horses in Kaduna North LGA.

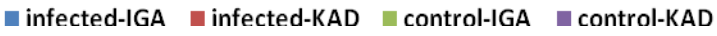
The mean lymphocytes count of trypanosome positive and trypanosome negative polo horses in stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stable 1, the mean lymphocytes count of trypanosome negative group was higher than trypanosome positive group in Igabi LGA; in stable 3, the mean lymphocytes count of trypanosome positive group was higher than trypanosome negative group in Igabi LGA; in stables 2, 4 and 5, only the mean lymphocytes count of trypanosome negative group was observed, the mean lymphocytes count of trypanosome positive group was absent. The mean lymphocytes count of trypanosome positive and trypanosome negative groups in Kaduna north LGA were compared; in stable 3, the mean lymphocytes count of trypanosome positive group was higher than trypanosome negative group in Kaduna North LGA; in stables 1, 2 and 4 the mean lymphocytes count of trypanosome negative groups was higher than trypanosome positive groups as presented in Figure 22.





The values in parenthesis represent the number of polo horses per stable

Figure 21: The mean lymphocytes counts of babesia positive and babesia negative polo horses in Igabi and Kaduna North Local Government Areas



The values in parenthesis represent the number of polo horses per stable

Figure 22: The mean lymphocytes counts of trypanosome positive and trypanosome negative polo horses in Igabi and Kaduna North Local Government Areas

4.16 MEAN BASOPHILS

4.16.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The mean Basophil count of babesia positive (0.36 ± 0.03 x10^9 /L) was lower ($P \leq 0.05$) than babesia negative (0.50 ± 0.01 x10^9/L) polo horses in Igabi and Kaduna North LGAs as represented in Table 4.

The mean Basophil count babesia positive (0.66 ± 0.00 x10^9 /L) polo horses in Igabi was higher than babesia positive (0.06 ± 0.01 x10^9 /L) polo horses in Kaduna North LGA and the mean basophil count of babesia negative (0.60 ± 0.01 x10^9 /L) polo horses in Igabi LGA was higher than babesia negative (0.04 ± 0.01 x10^9 /L) polo horses in Kaduna north LGA.

The mean basophil count of babesia positive and babesia negative in stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stables 1, 2, 3, 4, and 5, the mean basophil count of babesia positive groups in Igabi LGA was higher than the mean basophil count of babesia negative groups. In stables 1, 4 and 5 ,the mean basophil count of babesia positive groups for Kaduna North LGA was higher than babesia negative groups; in stables 2 and 3, the mean basophil count of babesia negative groups for Kaduna North LGA were higher than babesia groups as presented in Figure 23.

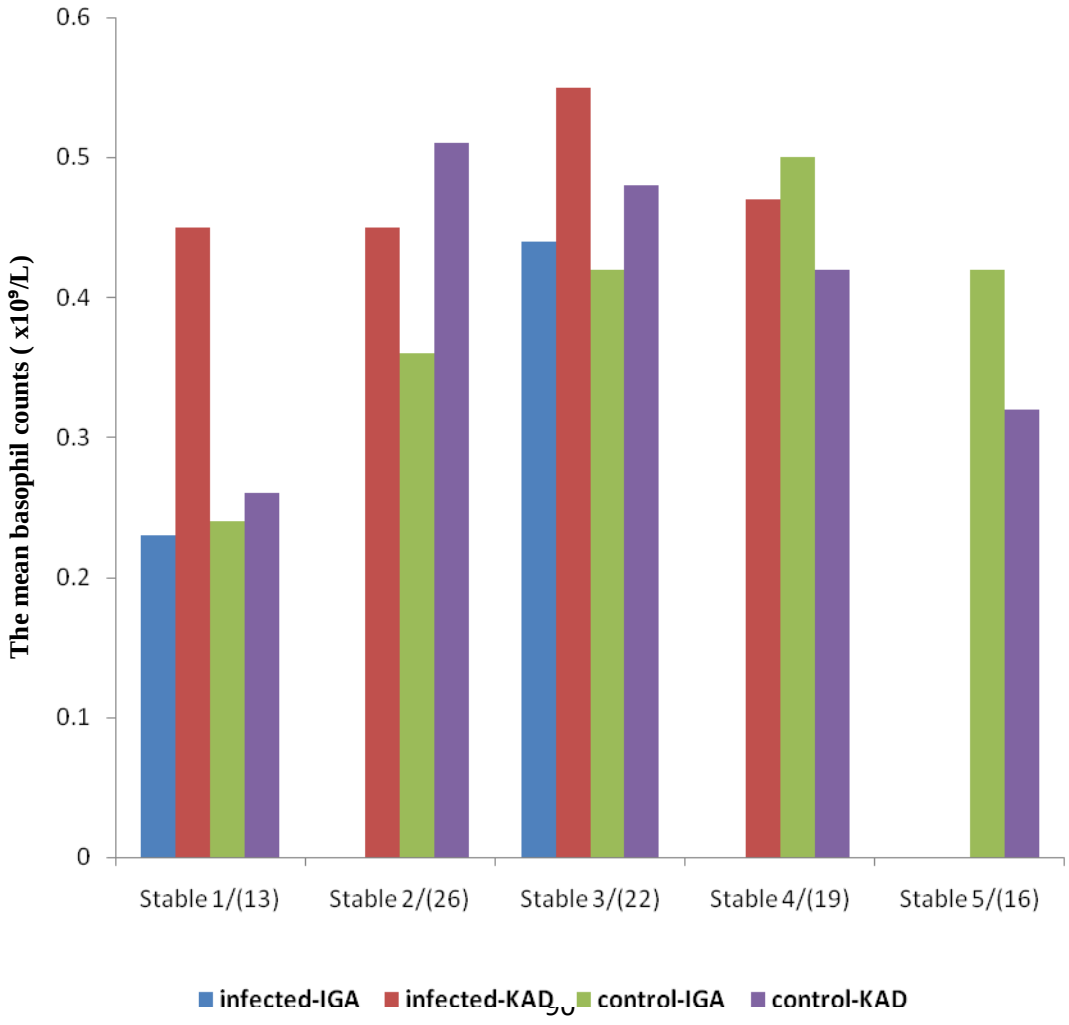
4.16.2 Trypanosoma Positive and Negative Groups in Igabi and Kaduna North LGAs

The mean Basophils count of trypanosoma positive ($0.10 \pm 0.01 \times 10^9$ /L) polo horses was higher ($P \leq 0.05$) than trypanosome negative ($0.05 \pm 0.01 \times 10^9$ /L) polo horses in Igabi LGA and Kaduna North LGA as represented in Table 5.

The mean basophils count trypanosome positive ($0.18 \pm 0.13 \times 10^9$ /L) polo horses in Igabi LGA was higher than trypanosome positive ($0.18 \pm 0.12 \times 10^9$ /L) polo horses in Kaduna North LGA and the mean basophils count trypanosome negative ($0.05 \pm 0.01 \times 10^9$ /L) polo horses in Igabi LGA was lower than trypanosome negative ($0.05 \pm 0.01 \times 10^9$ /L) polo horses in Kaduna North LGA.

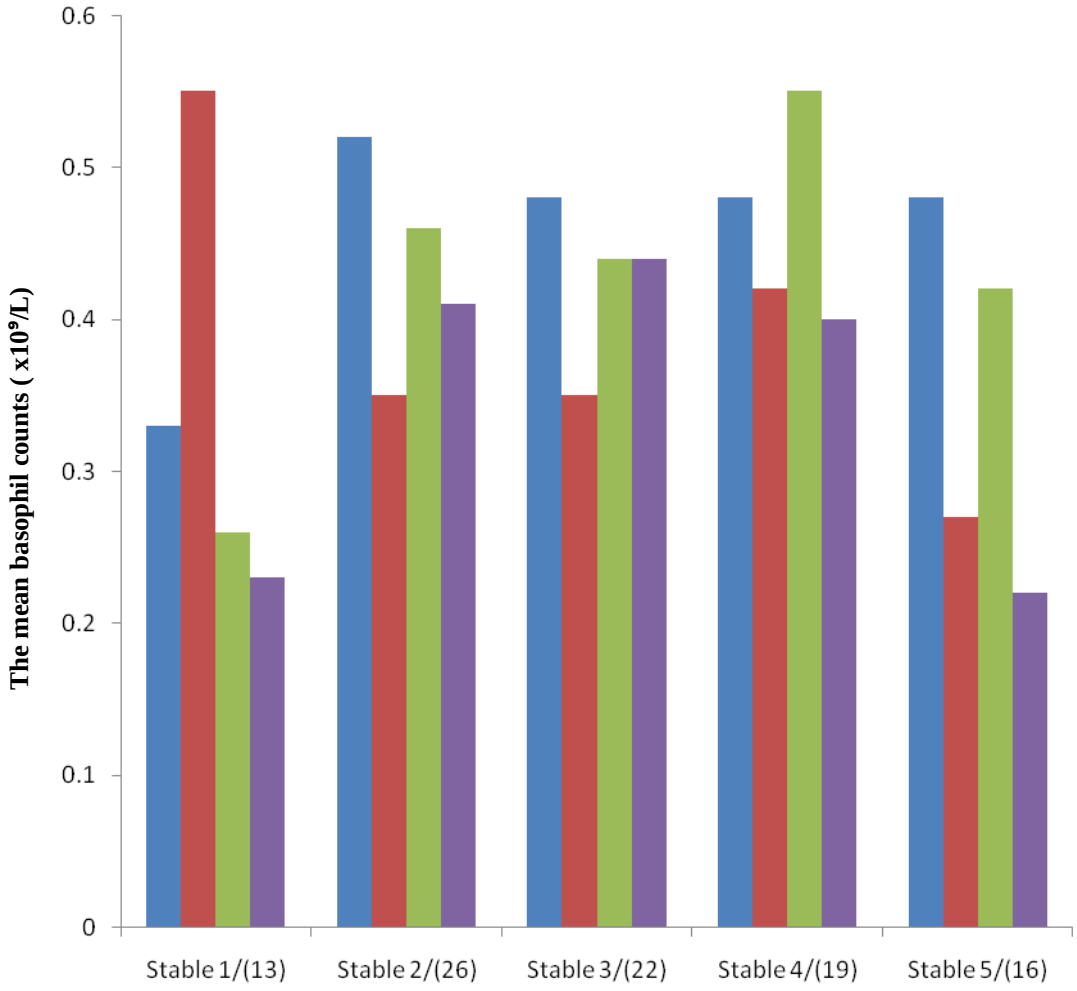
The mean basophil count of the trypanosome positive and trypanosome negative polo horses in stables 1 to 5 in Igabi and Kaduna North LGAs were compared; stable 1, the mean basophil count of trypanosome negative group in Igabi LGA was higher than trypanosome positive group; in stable 3 the mean basophil count of trypanosome positive group was higher than trypanosome negative group; in stables 2, 4 and 5, only the mean basophil count of trypanosome negative group appeared the mean basophil count of trypanosome positive group was not observed. In stables 1, 3 and 4, the mean basophil count of the trypanosome positive groups for Kaduna North LGA were higher than trypanosome negative groups; in

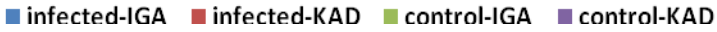
stable 2 the mean basophil count of trypanosome negative group was higher than trypanosome positive group as presented in Figure 24.



The values in parenthesis represent the number of polo horses per stable

Figure 23: The mean basophil counts of trypanosome positive and trypanosome negative polo horses in Igabi and kaduna North Local Government Areas





The values in parenthesis represent the number of polo horses per stable

Figure 24: The mean basophil counts of babesia positive and babesia negative polo horses in Igabi and Kaduna North Local Government Areas

4.17 MEAN CORTISOL VALUES

4.17.1 Babesia Positive and Negative Groups in Igabi and Kaduna North LGAs

The overall mean cortisol values of babesia positive (68.87 ± 8.70 ng/mL) were higher ($P<0.05$) than babesia negative (53.40 ± 1.30 ng/mL) polo horses in Igabi and Kaduna North LGAs.

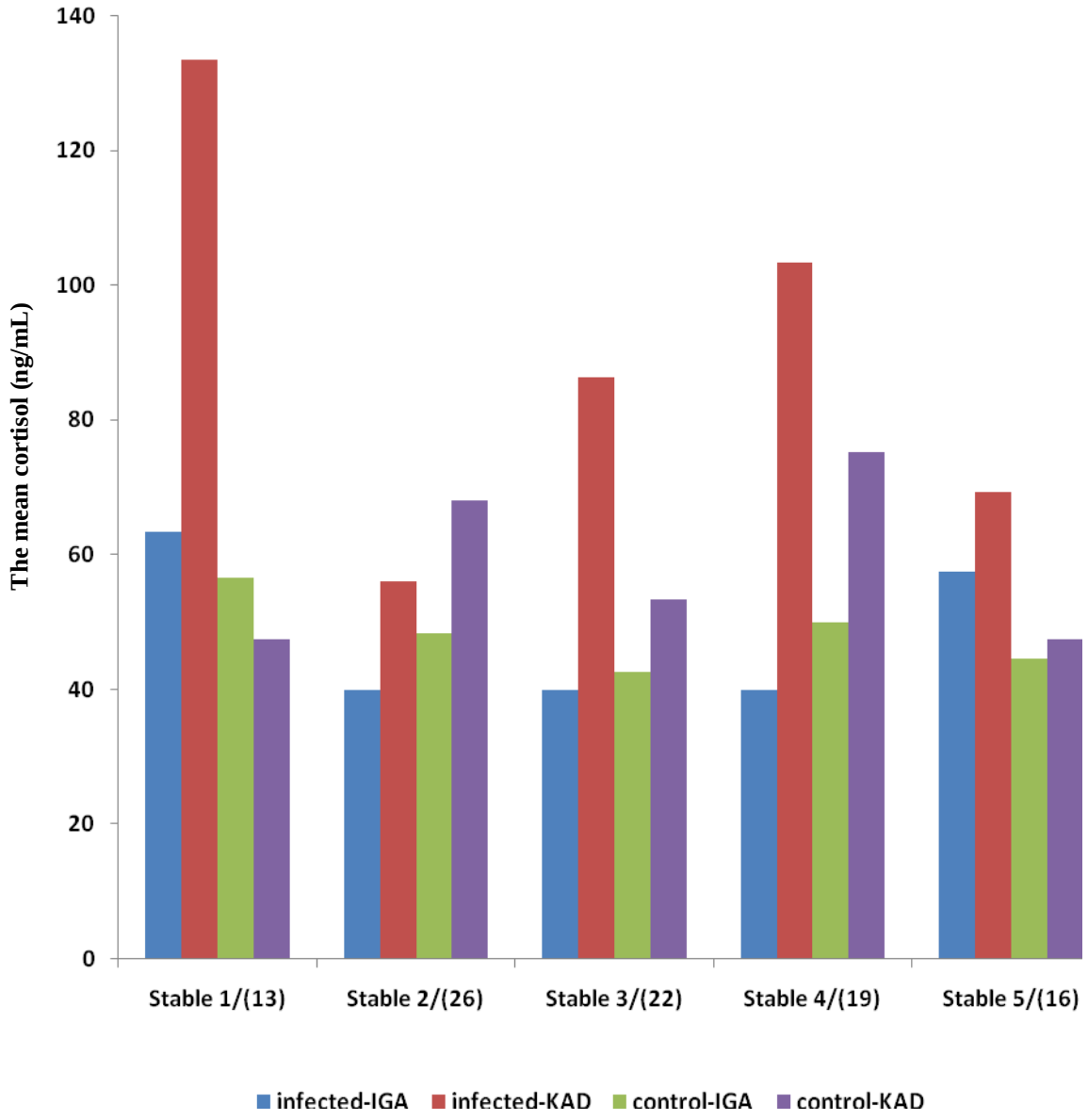
The mean cortisol of babesia positive (48.60 ± 5.08 ng/ml) polo horses in Igabi LGA was lower than babesia positive (89.60 ±13.50) polo horses in Kaduna North LGA and the mean cortisol of babesia negative (48.40 ± 2.40 ng/ml) polo horses in Igabi LGA was lower than babesia negative (58.30 ± 5.60 ng/ml) polo horses in Kaduna North LGA.

The mean cortisol values of babesia positive and babesia negative polo horses in stables 1 to 5 in Igabi and Kaduna North LGAs were compared; in stables 1 and 5, the mean cortisol of babesia positive groups in Igabi LGA was higher than babesia negative groups; in stables 2, 3 and 4, the mean cortisol values of the babesia positive group were lower than babesia negative groups. In stables 1, 3, 4 and 5, the mean cortisol of babesia positive groups in Kaduna North LGA were higher than babesia negative groups; in stable 2, the mean cortisol of babesia positive groups was lower than mean cortisol values of babesia negative groups as presented in Figure 26.

4.17.2 Trypanosome Positive and Negative Groups in Igabi and Kaduna North LGAs

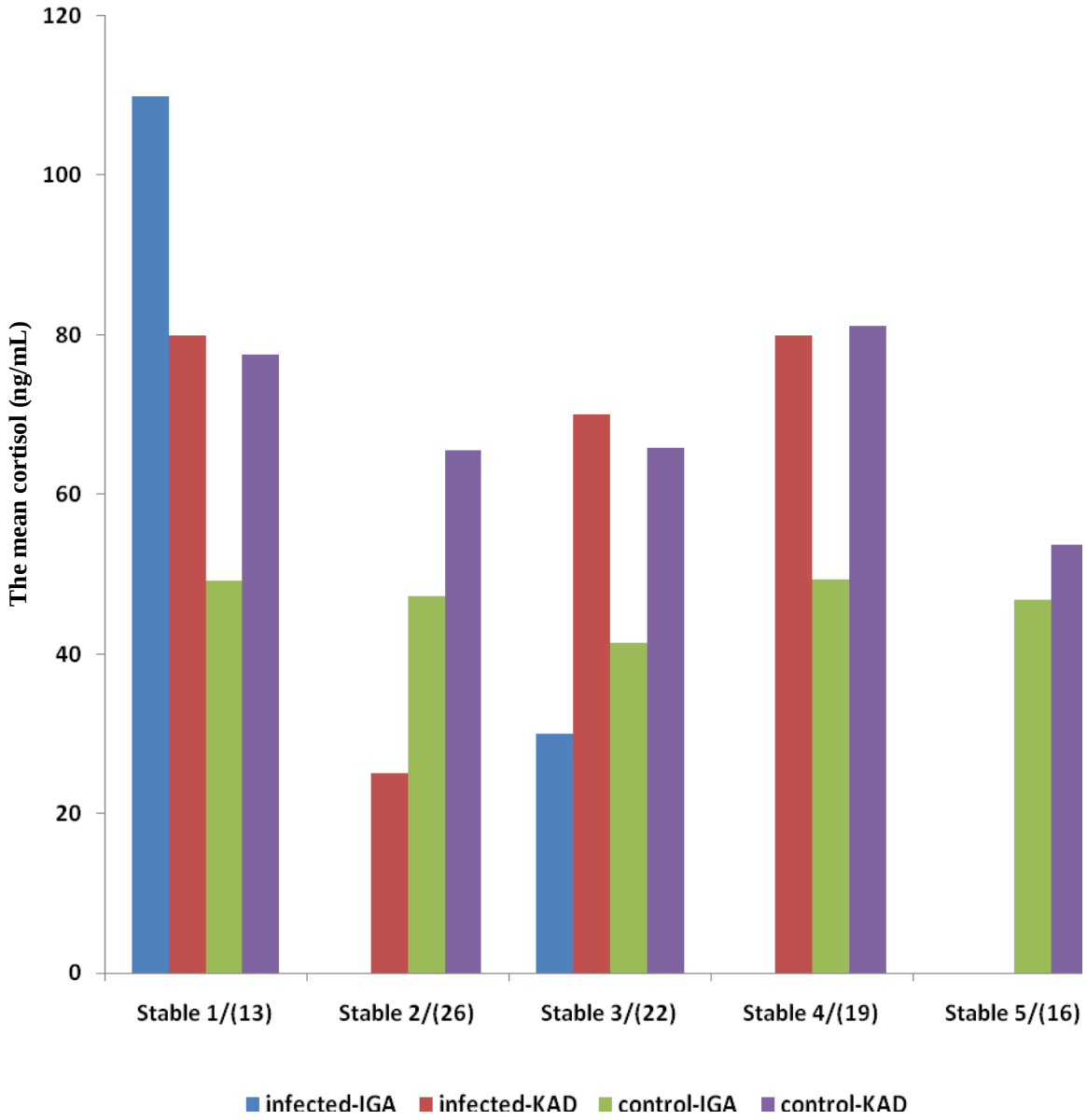
The overall mean cortisol values of trypanosome infected (65.80 ±10.70 ng/mL) polo horses were higher($P \geq 0.05$) than trypanosome negative (63.4 ± 12.3 ng/mL) polo horses in Igabi and Kaduna North LGA. The mean cortisol of trypanosome positive (28.60 ± 21.30ng/ml) polo horses in Igabi LGA was lower than trypanosome positive (51.00 ± 16.50 ng/ml) polo horses in Kaduna North LGA and the mean cortisol of trypanosoma negative (46.80 ± 1.40 ng/ml) polo horses in Igabi LGA was lower than trypanosome negative (68.30 ± 4.80 ng/ml) polo horses in Kaduna North LGA.

The mean cortisol of trypanosome positive and trypanosome negative polo horses for stables 1 to 5 in Igabi LGA and Kaduna North LGAs were compared; in stable 1, the mean cortisol of trypanosome positive group in Igabi LGA were higher than trypanosome negative group; in stable 3, the mean cortisol of trypanosome positive group in Igabi LGA was lower than trypanosoma negative group; in stables 2, 4 and 5, no positive case was recorded only the mean cortisol of trypanosome negative group in Igabi LGA was recorded. In stables 1 and 3, the mean cortisol of trypanosome positive groups in Kaduna North LGA were higher than trypanosome negative groups; in stables 2 and 4, the mean cortisol trypanosome positive groups were higher than trypanosome negative groups; in stable 5, no positive case was recorded only the mean cortisol of trypanosome negative group were recorded as presented in Figure 25.



The values in parenthesis represent the number of polo horses per stable

Figure 25: The mean cortisol of babesia infected and babesia polo horses in Igabi and Kaduna North Local Government Areas



The values in parenthesis represent the number of polo horses per stable

Figure 26: The mean cortisol of trypanosome positive and trypanosome negative polo horses in Igabi and Kaduna North Local Government Area

CHAPTER 5

DISCUSSION

Equine trypanasomosis and babesiosis is stil prevalent in Nigeria. In this study of polo horses in Igabi and Kaduna North local government areas of Kaduna state, the prevalence of babesiosis was 18.8% and trypanosomosis 3.1% in the two local government areas. The prevalence rate of *babesia* infection in polo horses from each local government area were: 15.6% in Igabi and 21.9% in Kaduna North LGA.The prevalence rate of *trypanosome* infection in polo horses from each local government area were: 2.1% in Igabi and 6.3% in Kaduna North LGA. Two species of *Babesia* and two species of *Trypanosome* parasites were identified: as a single infections namely *Babesia equi* 66.7% (24 cases), *B caballi* 33.3% (12 cases) in both LGAs, 83.3% (5 cases) of *Trypanosoma evansi* infection in both local government areas and 16.8% (1 case) of *T. congolense* infection in Igabi LGA only. *Babesia equi* is a more serious threat than *B caballi* in both LGAs. *Trypanosoma evansi* is a more serious threat than *Trypanosoma congolense* in Igabi and Kaduna North LGA. The infection rates were higher in Kaduna North LGA than Igabi LGA. The diference observed may be due good management practices of controlling the vectors by application topical fly repellent on horses during grooming and occasionally fumigating the stables and its environment.

The polo horses in stables 1 to 5 in Kaduna North LGA had higher *Trypanosoma* parasitemias than Igabi LGA. The clinical signs observed in infected polo horses are similar to those reported by Roberto *et al* (1995).

Babesia infection was reported to be endemic around Zaria with a higher prevalence in the wet season than dry (Lawal *et al.*, 1999) and had a prevalence of 10% in Nigeria

(Abdulkadir, 1994). The polo horses in stables 1 to 5 in Igabi and Kaduna North LGAs had babesia infection rate similar to that earlier reported by Garba, (2006). Kaduna state is in the guinea savanna zone of Nigerian with dense vegetation, high humidity, non availability of effective prevention/control measures favors vector survival and enhance transmission of parasites.

The parasitemia was generally low in both LGAs which may be due to the chronic nature of Babesiosis in horses as described by Malhitra *et al* (1978), the infection rates were higher in Kaduna North LGA than Igabi LGA. The higher *Babesia* parasitemia observed in Kaduna North LGA may be due poor managment practice while the low infection rate in Igabi may be due to good managment practices of controlling the vectors by application of acaricides (dust, bath or pour ons) and hand picking of ticks during grooming.

The mean live weight of babesia and trypanosome positive polo horses were higher than the mean live weight of babesia and trypanosoma negative polo horses these diference in weight observed may be due to size and breed differences.

The mean body condition score of babesia and trypanosome positive polo horses was lower than babesia and trypanosoma negative polo horses these difference may be due to decrease in water and feed intake resulting in emaciation in positive horses.

The mean packed cell volume of babesia and trypanosome positive polo horses were lower than babesia and trypanosoma negative polo horses these differences observed may be due to,dehydration, fear and excitement as a result of restrain (Transient polycythemia) excessive excercise (Lab tests online, 2001).

The mean red blood cell count of babesia and trypanosome positive positive polo horses was lower than babesia and trypanosoma negative polo horses; these may be due fear and

excitement as a result of restraint, excessive excercise, excessive sweating and dehydration (Blurtit, 1999).

The mean hemoglobin concentration of babesia and trypanosome positive polo horses was lower than mean hemoglobin concentration of babesia and trypanosoma negative polo horses these diference may be due to fear and excitement as a result of restraint, dehydration hence increases the hemoglobin concerntration (Medha , 2010).

The mean white blood cell count of babesia and trypanosome positive was lower than babesia and trypanosome negative polo horses these difference may be due to fear,stress or excitment stimulating secretion of adrenaline which inturn stimulate the release of white blood cell stored in the maginal pool into the circulation (Robert, 1999).

The mean total Protein (TPg/dL) of babesia and trypanosome positive polo horses was slightly higer than babesia and trypanosoma negative polo horses these difference may be due to dehydration which concentrate the total protein (Jeremy, 2010).

The mean cortisol values (ng/ml) of babesia and trypanosome positive polo horses was higher than mean cortisol values (ng/ml) of babesia and trypanosoma negative polo horses these diferences in mean cortisol values (ng/ml) could be due to the stress from fear/excitement and excercise which inturn stimulates increase in cortisol release (Valerie, 2005).

Changes involving leucocytes may be difcult to predict because initial leucopaenia during acute babesiosis may be followed by leucocytosis during the recovery period (Oladosu and Olufemi, 1990)

The mean segmented neutrophils of infected polo horses was higher than mean segmented neutrophils of negative polo horses; these increases may be due to fear/excitement or exercise hence return of neutrophils from marginal pool into circulation.

The mean monocytes count of infected polo horses was higher than mean monocytes count of negative polo horses; these diferences may be due to increased phagocytosis.

The mean eosinophils count of babesia infected polo horses was similar to babesia negative polo horses; may be due to systemic hypersensitivity reactions a form of inflammation caused by parasitic diseases.

The mean lymphocytes count of infected polo horses was higher than mean lymphocytes count of negative polo horses; these diferences may be due to fear/excitement or stress.

The mean basophil count of infected polo horses was higher than mean basophil count of negative polo horses.

CHAPTER 6

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

6.1 SUMMARY

Equine trypanasomosis and babesiosis is stil prevalent in Nigeria. In this study of polo horses in Igabi and Kaduna north local government areas of Kaduna state, the prevalence of babesiosis was 18.75% and trypanosomosis 3.12% in the two LGAs. The prevalence rate of *babesia* infected polo horses were: (15) 15.62% of the polo horses were from Igabi and the remaining (21) 21.87% of the polo horses are from Kaduna north local government

areas. *Babesia equi* 66.67% (24 cases) and *B. caballi* 33.33% (12cases) in both local government areas. The prevalence rate of *Trypanosoma* infected polo horses were: Igabi LGA (2 cases) 2.08% and Kaduna north (4 cases) 6.25% two species of *Trypanosoma* parasites were identified: as single infections namely *Trypanosoma evansi* (5 cases) 83.33% in the two local government areas and *T. congolense* (1 case) 16.67% in only Igabi local government area..

6.2 CONCLUSIONS

These study is intended as a practical contribution for Veterinary practitioners because it describes different clinical presentations and laboratory findings of equine piroplasms and trypanosomosis. *Babesia equi* is more a threat than *babesia caballi and Trypanosoma evansi* is a more serious threat than *Trypanosoma congolense* in Igabi and Kaduna North LGAs. The infection rates were higher in Kaduna North LGA than Igabi LGA. Infected horses presented low red cell count, packed cell volume, hemoglobin concentration indicating low oxygen carrying capacity of the blood in infected horses reducing their tolerance to exercise. Non infected horses submitted to training programs involving adquate component of exercise achieve a better degree of fitness and present marked increases in erythrocyte numbers, packed cell volume, haemoglobin concentration, total white cell count and lymphocytes. However stress increases adrenaline and cortisol which inturn contracts the spleen of most mammals releasing stored cell , parasites and depresses the immune system.

6.3 RECOMMENDATIONS

There should be a regular vector control by fumigation of stables and application of pour on. Infected horses should not be subjected to stress, from excitement, exercise, fear dehydration or transportation. Hematinics should be included in treatmemt while steroidal pain relievers should be applied with caution administered.

REFERENCES

Abenga, J.N., Enwezor, F.C.N., Lawani F.A.G., Ezebuiro. C., Sule J. and David, K. (2002). Prevalence of trypanosomiasis in trade cattle slaughter in Kaduna, *Journal of Parasitology*, 23:107-111.

Abdulkadir, A.L. (1994). *Infectious Diseases of Livestock in Nigeria; An Outline*. First Edition. Ahmadu Bello University Press, Zaria, Nigeria, pp 43.

Adams, L.G. (1981).Clinicopathological aspects of imidocarb dipropionat toxicity in horses, *Researches in Veterinary Science*, 31: 4 – 61.

Adeyefa, C.A.O. and Dipeolu, O.O. (1983). Parasites isolated from insects feeding on blood and secretion of horses in Oyo State Nigeria, *Nigeria Veterinary Journal*, 12(1):78– 81.

Adawa, Y.D.A., Abdullahi, S.U., Adeyanju, B.J. and Agbede, R.I.S. (1990). Efficacy of ivermectin against external and internal parasites of dogs, *Zariya Veterinarians*, 5(1 and 2): 37-44.

Akiko, N., Naoaki, Y., Tomohide, M., Sabine, B., Haruyuki, H., Xuenan X., Yinchnag, Z., Florencia, G., Kozo, C. and Ikuo, I. (2003). Growth-inhibitory effects of articulate, pyrimathamine and parvaquine against *Babesia equi* and *Babesia caballi* in in-vito

cultures antimicrobial agents and chemotherapy *American Society for Microbiology*, 47(2): 800-803.

Annebearson, R., Lawrence, P.R, and Smith, A.J. (1996). In the field of drought animals research, *Tropical Animal Health Production*, 28:49-59.

Anonymous (1970). Cattle Tick Control, *A cooper research organization publication*, Martins Press Limited, Woodbridge Street E.C.I. England, pp 65.

Anonymous (1977). *Manual of Veterinary Parasitological Laboratory Techniques*: Bulletin No. 18. Published for Her Majesty's Stationary Office by Albert Gait Limited, London, pp 58 – 112.

Anonymous (2000). A Comprehensive Investment and Tourist Guide to the Federal Republic of Nigeria, Executive Governors of the Fourth Republic, *Published by Ministry of Tourism*, pp 98 – 130.

Anonymous (2003). International Commission of Zoological Nomenclature Opinion 2027, (Case 3010) usage of 17 specific names based on wild species, which are pre-dated by or contemporary withthose based on domestic animals (*Lepidoptera, Osteichthye Mammalia*) conserved, *Bulletin of Zoological Nomenclature*, 60:81-84.

Anonymous (2002). Body condition score of horses and donkey, Annex C and D equine industry welfare guidline compoundium for horses ponies and donkeys.

Anosa, V.O. and Kenenko, J.J. (1983a). Pathogenesis of *Trypanosoma brucei* in a deer mice. hematologic, erythrocytic, biochemical and iron metabolic aspect, *American Jounal Of Veterinary Research*, 44: 639.

Anosa, V.O. and Kenenko J. J. (1983c). Pathogenesis of *Trypanosoma brucei* in a deer mice macrophage ultrastructure and function, *Veterinary Pathology*, 20:617-631.

Auty, H., Mundy, A., Fyumagna, R.D., Picozzi, K., Welburn, S. and Hoare, R. (2008). Health management of horses under high challange from trypanosomes: A case study from serengeti Tanzania, *Veterinary Parasitology*, 154(3 and 4): 233-241.

Ascort, P. (2008). A short history of polo, *Ascot Park Polo Club*, Chobham, Surrey, UK www.aspectgroup.co.uk.

Rebelo, E. (1999). Molecular detection of *B. Equi* and *B. caballi* in horse blood by P.C.R amplification of part of the16s RNA gene. *Veterinary Parasitology*, 84:75-83.

Barbara, L.H., David, H.P., Eric, A.P., Goff, W.L., Dane, A.M., Philip, W.T., Eberhard M.L., and Andre, F.G. (1996). A fatal case of babesiosis in Missouri: Identification of another piroplasm infect human, *Annals of Internal Medicine*, 129(7): 643-650.

Barth, D. and Perston, J.M. (1988). Efficacy of tropically administered ivermectin against chorioptic and sarcoptic mange in cattle, *Veterinary Record*, 123:101-104.

Bekker, C, de Vos, S, Taoufik, A., Sparaagano, O. and Jongejan, F. (2002). Simultaneous detection of *E. ruminantium in Amblyomma variegatum* ticks by reverse line blot hybridization, *Veterinary Microbiology*, 89:223-238.

Benjamin, M.M. (1978). *Outline of Veterinary Clinical Pathology*, Third edition Iowa State University Press Iowa, U.S.A pp 30.

Birkenheuer, A.J., Levy, M.G. and Breitch-Werdt, E.B. (2003). Development and evaluation of a semi-nested P.C.R for detection and differentiation of *Babesia gibsoni* (Asian Genotype) and *B. carius* DNA in canine blood samples, *Journal of Clinical Microbiology*, 41:4172-4177.

Blood, D.C., Henderson, J.A. and Radostits, O.M. (1979). Diseases of the stomach and intestine, In: *Veterinary Medicine*, Fifth Edition, Baillliere Tindal, London, pp 1114-1122.

Blurtit, O. (1999). What causes high red blood cells count? www.blurtit.com.

Brod, J. and Sirota, J.M. (1948). Determination of serum creatinine using the Jaffe reaction, *Journal of Clinical Investigation*, 27:645.

Brocklesby, D. (1979). Human Babesiosis, *Journal of South Africa Veterinary Association*, 50:302-307.

Bruning, A. (1996). Equine piroplasmosis, an update on diagnosis, treatment and prevention, *British Veterinary Journal*, 152:139-151.

Burger, H.J. and Bauer, C. (1987). Efficacy of four anthelmintics against benzimidazole-resistant *cyanthotmum* of horses, *Veterinary Records*, 120(19):293-296.

Biryomumaisho, S., Katunguka R.E. and Rubair-Akiiki, C.M. (2003). Serum biochemical changes in *Trypanosoma congolense and Trypanosoma brucei* infected goats, *Veterinarski Arhiv*, 73(3):167-180.

Caccio, S., Camma C., Onuma, M. and Severini, C. (2000). The Beta tubulin gene of *Babesia* and *Theileria* parasites in an informative marker for species discrimination, *International Journal of Parasitology*, 30:1181-1185.

Camacho, A.T., Guitian. F.J., Pala, E., Gestalt, J.J., Olmedo A.A., Habla, M.A., Telford III, S.R. and Spielman, A. (2005). *Theileria (Babesia) equi and Babesia caballi* infection in horses in Galicia Spain. *Tropical Animal Health and Production*, Published by Spring Netherlands, 37(4):293-302.

Cheesbrough, M. (1991). *Medical Laboratory Manual for Tropical Countries*, Second Edition, Cambridge University Press, London, 1:481-494.

Coles, E.H. (1974). *Veterinary Clinical Pathology*, Saunders company, London, pp 543-593.

Cristiane, D.B., Rosangela, Z.M., Tânia, F.R. and Aramis, A.P. (2007). Serodiagnosis of *Babesia equi* in horses submitted to exercise stress, *Pesquisa Veterinaria*

Brasileira, vol.27 no.4 Rio de Janeiro Apr. 2007. doi: 10.1590/S0100-736X2007000400009.

Crido-Fornelio, A., Gonzalez, D.R., Bulin-Sarana, A. and Barba-Carretero, J.C. (2003a). Molecular characterization of a *Babesia gibsoni*, isolated from a Spanish dog, *Veterinary Parasitology*, 13:83-92.

Criado–Fornelio, A., Martinez-Marcos, A., Buling–Sarana, A.A. and Barba-Carretero J.C. (2003b). Presence of *Mycoplasma hemofelis, M. hemominitum* and piroplasmid in cats from Southern Europe: A molecular study, *Veterinary Microbiology*, 93:307-317.

Daniel, N., Josune G., Ana, L.G., Ramon, A.J and Ana, H. (2004). Detection and identification of equnine *Theileria and Babesia* species by reverseline blotting: Epidemiology survey and phylogenetic analysis, *Veterinary Parasitology*, 123:41-54.

Donald, E. Stem, Jr. and Charles, W. Lamb, Jr. (2007). The marble-drop technique: Aprocedure for gathering sensitive information, *Decision Sciences*, 12(4):702-707.

David, A. (2001). Mechanism of immune evasion by pathogens, *Veterinary Biology*, htm//www.vetmed.ufl.edu/path/teach/vem5131/publish/ve2017.htm accessed on 25/06/2008.

Déthié, F., Paulo J.L., Pereira de A., Bart, G., Sabine, O., Momar, N., Dirk, B., Niko, S., Françoise, N. and Geerts, S. (2001). Prevalence and incidence of trypanosomiasis in horses and donkey in Gambia, *Veterinary parasitology,* 101(2):101-114.

Desquesnes, M. and Dia, M.L. (2003). *Trypanosoma vivax* machanical transmission by one common African tabanids, *Atlotus agrestis*, *Experimental Parasitology*, 103 (1 and 2):35-43.

Dipeolu, O.O. and Oduye, A.O. (1977). Survey of blood parasites of horses in Ibadan Western Nigeria, *Parazitology*, 22:155-159.

Duncan, J.R. and Press, K.W. (1986). *Veterinary Laboratory Medicine*, 2nd edition, Lowa State University Press, pp 22 – 25.

Elwyn, H.E. (2000). Horses, Dorling Kindersley handbooks. Dorling Kindersley Limited, London, pp 28-17.

Esievo, K.A.N. (1979). Invivo production of neuraminidess by *T. vivax*, proceeding of the 16 meeting of the international scientific council for trypanosomiasis, *Cameroun*, pp 205, publication no111.

Esievo, K.A.N. and Saror, D.I. (1983). Leucocyte response in *Trypanosoma vivax* infection in cattle, *Journal of Comparative Pathology*, 93:165-169.

Esievo, K.A.N. and Saror, D.I. (1991). Immunochemistry and immunopathology of animal trypanosomiasis, *Veterinary Bulletting*, 61:765-777.

Esterina, F., Pietro, M., Vincenzo, A., Loredana, G. and Adriana, F. (2008). Circulating β-endorphin, adrenocorticotrophic hormone and cortisol levels of stallions before and after short road transport: Stress effect of different distances, *Acta Veterinaria Scandinavica*, 2008, 50:doi:10.1186/1751-0147-50-6.

F.A.O. (1991). Proceedings of expert consultation on genetic aspect trypanotolerance extract from the report of the first FAO expert consultation on research on trypanotolerance and breeding of trypanotolerant animals, Animal production and health 94, *Program*.

Franz, H. and Josef, M. (1926). Special pathology and therapeutics of the disease of domestic animal, 3rd edition, 1:1010.

Fiennes, R.N.T.W. (1970). Pathogenesis and pathology of African animal trypanosomiasis: In the African trypanosomiasis, London,United Kingdom, pp729-773 (edited by Mulligan H W; Potts W.H).

Fissile, C.H. and Subbarow, Y. (1925). Determination of inorganic phosphate in serum and urine, *Journal of Biochemistry,* 66:375.

Friedhoff, K.T. (1982). Piroplasm of the equidae, significance for international commerce, *Berl. Munch. Tie rarztl. Wschr*, B 368-374.

Friedhoff, K.T., Tenter, A.M. and Muller, I. (1990). Hemoparasites of equines, impact on international trade of horses, *Reveue scientifique ettechnique*, 01E9, pp 187-194.

Garba, U.M. (2006). Babesia in royal horses in Niger state. M.Sc thesis presented to Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria.

Georges, K., Loria, G.R., Riili, S., Creco, A., Caracappa, S., Jongejan, F. and Sparagano, O. (200I). Detection of hemoparasites in cattle by reverseline blot hybridization with a note on distribution of ticks of sicily, *Veterinary Parasitology*, 99:273-286.

Geets, S. and Holmes, P.H. (1998). Drug management and parasite resistance in bovine trypanosomiasis in Africa, *PAAT Technical and Scientific Series*, pp1-28 FAO ROME, 1998.

Geysen, D., Delespaux, V. and Geerts, S. (2003). PCR-RFLP using Ssu-rDNA amplification as an easy method of specie specific diagnosis of *trypanosoma* species in cattle, *Veterinary Parasitology*, 110(3-4):171-180.

Gill, B.S., Singh, T. and Gill, J.S. (1987). *Trypanosoma evansi* in pigs in india, *Veterinary Records,* 120(4): 92.

Gitelman, H.J. (1967). Determination of calcium using O-ereosulphathalein complexome, *Anals of Biochemistry*, 18:521.

Gina, L.P., Liam, J.M., Andy, T., Joanna, L., Lucinda, M., Saloum, J., Jibril, J., Amadou, J. and Robert, M. (2008). Tyrpanosomiasis in Gambia: Prevalence in workinghorses and donkey detected by whole genome amplification and PCR, and evidence for interaction between trypanosome species, *B.M.C. Veterinary Research*, http//creativecomnions.org//licence/by/2.0.

Griffith, R.B. (1977). Symposium, British society for parasitology, *Blackwell Scientific Publication*, Oxford, London, 16L:41-66.

Gubbels, J.M., Devos, A.P., Van-der Weide, M., Viseras, J., SchouIs, LM., de Vries E. and Jongejan, O. (1982). Simultenous detection of bovine *Theileria* and *Babesia* species by reverse line blot hydridization, *Journal of Clinical Microbiology*, 37: 1782-1789.

Hailat, N.Q., Lafi, S.Q., Al-daragji, A.M. and Al-ani, F.K. (1997). Equine babesiosis associated with strenuous exercise, Clinical and pathological studies in Jordan, *Veterinary Parasitology*, 69:1-8.

Hashim, W.H. and Wasfi, I.A. (1986). Intermectiin treatment of camels naturally infected with sarcoptic mange, *World Animal Review*, 57:26-29.

Henneke, D. (1998). Body condition scoring system in habits for horses equine protection organisation, pp 1-3. www.habitatforhorses.org/bodyscoring.

Herder, S.S., Nkinin, S. and Njiokou, F. (2002). Identification of trypanosomes in wild animals from Sounthern Cameroun using polymerase chain reaction (PCR), *Parasites*, 9(4):345-349.

Hill, D.H. and Akpokoje, J.U. (1971). Trypanosomiasis in horses at Ibadan, Western Nigeria, *International Scientific Committee on Trypanosomiasis Research* 105:117-119.

Holman, P.J., Hietala, S.K., Kayashima, L.R., Oslon, D., Waghela, S.D. and Wagner, G.G. (1997). Case report: Field acquired subclinical *Babesia equi* infection confirmed by in vitro culture, *Journal of Clinical Microbiology*, 35(2): 474-476.

Igbokwe, I.O. (1993). Free radicals, lipids peroxidation and African trypanosomiasis submitted for publication.

Ikede, B.O. (1991). Diagnostic techniques for African trypanosomiasis. In: Control of trypanosomiasis in Nigeria as a strategy to improve livestiock production.

Ikeme, M.M. (1989). Ruminant helminths: Classification, economic importance and diagnosis, II workshop paper presented at the *National Veterinary Research Institute*, Vom, Nigeria, June 5-9, 1989.

ILCA (1993). A hand book of African livestock statistics *International Livestock Center for Africa*, Adis Ababa, Ethiopia, pp 9.

Ilemobade, A.A. (1971). Studies on the incidence and pathogencity of *T. evansi* in Nigeria, the pathogencity of *T. evansi* for bovine and equine species In: Proceedings of 13/11 meeting of the *International Scientific Council for Trypanosomiasis Research*, Lagos (C.C.T.A Publication, No. 105), 1971, pp 104-107.

Israel, D. and Douglas, A.N. (1974). The blood. In: Israel, D. and John, B.H., (eds). *Clinical Diagnosis by Laboratory Methods*, Fifteenth edition. W.B. Saunders Company, London, pp 100-200.

Itard, J. (1989). The Africa trypanosomiasis. *In: the Manual of Veterinary Tropical Parasitology*, pp 181 CAB Publication.

Jay, R.G. and Marion, E.G. (1990). Qualitative faecal examination, *Parasitology for Veterinarians*, Fifth edition, W.B. Saunders company, London, pp 267.

Jeremy, E.K. (2010). Serum proteins, http://www.drkaslow.com/html. 720 North Tustin Avenue Suite, 104 Santa Ana, CA 92705-3606.

Jimada, I.S. (2005). British imperial interest and Nupe resistance, C.1886-1903 In:Yakubu A.M., Jumare, I.M. and Saeed, A.G. (eds). Northern Nigeria, A century of transformation, 1903-2003, First edition. Published by Arewa house, *Centre for Documentation and Research*, Ahmadu Bello Univeristy, Zaria Kaduna State, Nigeria, pp 29-40.

Joanne, R., Ernest, C., Marvrin, D.C., Michael, S. and David, A. (2003). A field evaluation of PCR for the routine detection of *Babesia equi* in horses. *VeterinarvParasitology, 114: 81 - 87*.

Kamuaga, M. (2003). Socio economic and agricultural factors in research and control of trypanosomiasis, *PAAT Technical And Scientific Series*, 4. FAO ROME.

KGIS (2008). *Kaduna Graphic Information System*, Kaduna State Ministry of Land Survey and Country Planing, Kaduna state. www.KGIS.com.

Kihurani, D.O., Nantulya V.M., Mbuik S.M, Mogoa, E., Nquhimwanga, J. and Mbith, P.M.F. (1994). *Trypanosoma brucei, T. vivax, T. congolense* infection in horses on a farm in Kenya, *Tropical Animal Health Production*, 26(2):95-98.

Klaus, D.E. (1996). Immunology: *Understanding the Immune System*. First edition Wiley-liss Inc. U.S.A., pp 247-257.

Klompen, .J.S.H., Black, W.C.I.V., Keirans, .I.E. and Oliver, Jim, J.H. (1996). Evaluation of ticks, *Annual Revue Entomologyy*, 41:141-161.

Knowles, D.P., Kappmeyeyer, L.S., Stiller, D., Hennager, S.G. and Pennyman, L.E. (1992). Antibody to a recombinant merozoite protein epitopeidentifies horses infection with *Babesia equi*, *Journal of Clinical Microbiology*, 30:3122-3126.

Kuttler, K.L. (1981). Chemotheraphy of Babesiosis: A review In: Ristic, M., KrejcI' Jr. (cds) Babesiosis, New York, Academic press, pp 65-85.

Kuttler, K.L., Zaugg, J.L. and Gipson, C.A. (1987). Imidocarb and Parvaquone in the treatment of piroplasmosis (*B. equi*) in equids, *American Journal of Veterinary Research*, 48(1):1613-1616.

Kycwalabye, E. and Lawal, A.I. (1989). *Babesia equi* and *Tryponosoma vivax* infections in donkeys, *Revue Elevage Medicine Veterinarie pays Tropicans*, 42(2):205-210.

Lab tests online (2001). Hematocrit, www.labtestsonline.org

Lawal, A.I., Kwanashie, G., Okubanjo, O.O., Natala A.J. and Idoko R. (1999). The prevalence of piroplasmosis and microfilaria infection in local and exotic horses in Zaria, *Zariya Veterinarian*, 6(1):16-22.

Lewis, B.D., Penhorn, B.L. and Rebollar, LM.L. (1995). Immune response to South African *Babesia canis* and the development of a preliminary vaccine, *Journal of South African Veterinary Association*, 66:61-65.

Les, S. (2001). Its in the blood. *The Horse Magazine*, Article # 868. http//www.theHORSE.com..

Lumsden, W.H.R., Herbert,W.J. and McNeillage, G.J.C. (1973). *Techniques with trypanosomes*, Churchill Livingstone, Edinburgh, pp 167.

Medha, G. (2010). High hemoglobin and hematocrit. www.buzzle.com.

Malhitra, D.V., Banerjeen, D.P. and Gautam, O.P. (1978). Prevalence of latent cases of *B. equi* infection in some parts of Northwest India as measured by the capillary agglutination test. *Equine Veterinary Journal*, 10(1):24.

Malherbe, W.D. (2006). The manifestations and diagnosis of babesia infections. some protozoan diseases of man and animals: Anaplasmosis, babesiosis and toxoplasmosis, *Annals of the New York Academy of Sciences,* 64:128-146.

Margi, S. (1995). Blood chemistry, *Veterinary Clinical Laboratory Procedures*, published by Mosby, New York, pp 101-151.

Mare, C.J. (2008). African trypanosomiasis (Nagana) Part IV Foriegn animal dieases, *Veterinary Science/Microbiology*, University of Arizona, Tuson, Az.

Maudlin, I., Holmes P.H. and Miles, M.A. (2004). *The Trypanosomiases*, CABI publishing, a division of CAB international 875 Massachausetts avenue 7 floor Cambridge, USA, pp 595.

Mehlhorn, H. and Schein, E. (1984). The piroplasms: Life cycle and sexual stages, *Advances in Parasitology*, 23:37-103.

Mehlhorn, I.I. and Schein, E. (1998). Redescription of *Babesia equi* Leveran 1901 and eileriaequi, *Parasitology Research*, 84:467-475.

Metcalf, E.S. (2001). The role or international transport of equine semen on disease transmission, *Animal Reproduction Science,* 68:229-232.

Cosker, P.J. (1981). The global importance of bebesiosis. In: Ristic, M. and Kreier, I.P. (eds). *Babesiosis*, Academic press, New York, pp 1-124.

Michael, E. (2003). *Horses Photographs*, published by Alfred A.K., New York, pp 10-20.

Mohammed, A.N. (1974). The seasonal incidence of ixodid ticks of cattle in Northern Nigeria and the in the Netherlands with particular reference to their role in the transmission of bovine piroplasmosis Ph.D dissertation, pp 234.

Mohammed, A.N. and Agbede R.I.S. (1980). Control of ectoparasites of ruminants in Nigeria. In: Proceedings on control of problems facing the leather industry in Nigeria, *Lerin,* Zaria, pp 21-27.

Murray, M., Trail, J.C.M., Turner, D.A. and Wissocq, Y. (1983). Livestock production and trypanotolerance, *Training Manual*, ILCA, Adis Ababa, Ethiopia, pp 6-28.

Nantulya, V.M., Bajyana O. and Holmes, E. (1987). Detection of circulating trypanosomal antigen in *T. evansi* infected animal using *T. brucei* group specific monoclonal antibody, *Tropical Medicine and Parasitology*, 40:263-266.

Natelsons, A.M. (1951). Determination of serum urea: Diacetyl monoxime method using Thiosemicarbazide, *Journal of Clinical pathologyy*, Dec. 219120:1153-1172.

Naviaux, L.J. (1978). *Horses in Health and Disease*, First edition. A.R.C. Book incorporated, New York, pp 12-30.

Neitz, W.O. (1956). Classification transmission and biology of piroplasms of domestic animals, *New York Academy of Science*, 64:56-111.

Nicolaiewsky, T.R., Richter, M.F., Lunge, V.R., Cunha, C.W., Delagostin, O., Ikuta, N., Fonseca, A.S., da silva, S.S. and Ozaki, L.S. (2001). Detection of *B. equi* La Veran, by nested polymerase chain reaction (n-P.C.R.), *Veterinary Parasitology*, 101:9-21.

Noval, R.A.I., Perry, B.D. and Young, A.S. (1992). *Epidemiology of theilleriosis in Africa*, Academic press, London, p 481.

Nyeko, J.H.P., Ole-Moiyoi, O.K., Majiwa, P.A.O., Otieno, L.H. and Ociba, P.M. (1990). Characterization of trypanosome isolates from cattle in Uganda using species-specific DNA probes reveals predominance of mixed infections, *Insect Sciences Applications,* 11:271-280.

Njiru, Z.K., Constantine C.C., Guya, S., Crowther, J., Kiragu, J.M., Thompson, R.C.A. and Davila, A.M.R. (2005). The use of ITSI rDNA PCR in detecting pathogenic African trypanosome. *Parasitology Research. J.* 95(3):186 -192.

OIE Press Release (1997). www.oie.int.eng/press/a-970530.

Orhue, N.E.J., Nwanze, E.A.C. and Okafor, A. (2005). Serum total protein, albumin and globulin levels in *Trypanosoma brucei* infected rabbits: Effect of orally administered *Scoparia* dulcis, *African Journal of Biotechnology*, 4(10):1152-1155 ISSN 1684–5315.

Oladosu, L.A. and Olufemi, B.E. (1990). Hematological parameters of Argentinian polo horses with acute babesiosis, *Tropical Veterinarian*, 8:163-169.

Papadopoulos, B., Brossard, M. and Perie, N.M. (1996). Piroplasmosis of domestic animals in the Macedonia region of Greece 3. Piroplasmosis of small ruminants, *Veterinary Parasitology*, 63:67-74.

Peter, J.I. (2009). Canine babesiosis: From molecular taxonomy to control, *Parasites and Vectors*, (2)1, 2009.

Penzhorn, B.L., Lewis.B.D., De Walls, D.T. and Rebollar, M.L. (1995). Sterilization of *B. canis* infection by imidocarb alone or in combination with diminazene, *Journal of South Africa Veterinary Association*, 66:157-159.

Philip, Y. and Doris, L.L. (2004). *The New Websters dictionary or the English language*: International edition lexicon publication incorporated, p 308.

Piesman, J. (1987). Emerging tick-borne diseases in temperate climates, *Parasitology Today*, 3:197-199.

Prashanp, J., Vijayr, S., Rajaram, P., Stephane, H.R., Harshar, S., Vibhawaris, D., Aradhana, B., Jean, J. and Philippe, T. (2005). Human trypanosomiasis caused by *Trypanosoma evansi* in India: the first case report, *Annual Journal of Tropical Medicine and Hygiene*, 73(3):491-495.

Preston, P.M. (2001). *Theileriosis*. In: Service, M.W. (cd). Encyclopedia of Arthropod - transmitted infections of man and domesticated animals. CABI Publishing, Walling Ford, pp. 487 - 502.

Purnell, R.E. (1981). Babesiosis in various hosts. In: Ristic, M. and Kreier, J.B., (eds). *Babesiosis*, New York, Academic press, pp 25-63.

Putt, S.N.H., Shaw, A.P.M., Wood, A.I., Tyler, L. and James, A.D. (1988). Veterinary epidemiology and economics in Africa. A manual for use in the design and appraisal of livestock health policy, 2nd edition, *International Livestock Center for Africa*, ILCA, pp 22-68.

Radostits, O. M., Gay, C.C., Blood, D.C., and Hinchchiff, K.W. (2000). *Veterinary Medicine*: A textbook of diseases of cattle, sheep, pig, goat and horses. Ninth edition, Harcourt publishers Ltd, London, pp 1289-1296.

Rampersad, I., Cesar. E., Campbell, M.D., Samlal, M. and Ammons, D. (2003). A field evaluation of P.C.R. for the routine detection of *B. equi* in horses, *Veterinary Parasitology*, 114:81-87.

Ristic, M. (1988). *Babesia* of domestic animals and man. R.R.C Press. Boca Raton Florida. Ronald, J.R. and Susan, E.(1996). *Illustrated Atlas of Equine Clinical Anatomy and Disorders of the Horses*, Equistar Publication Ltd, pp 10-20.

Roncalli, R.A. and Sutherland I.H. (1986). The use of ivermectin injectabe against *Psoroptes ovis (Acarine psoroptidae)* in sheep. Proceedings of the 3151 annual

meeting of the *American Association of Veterinary Parasitologists*, Atlanta Georgia, July 20-22.

Roberto, A.M.S., Heitor, M.H., Luzimari, B.D., Flora, A.X. and Alberto, M.D. (1995). Pathogenesis of *Trypanosoma evansi* infection in dogs and horses: *Hematological and Clinical Aspect, Sciences Rural*, 25(2) *Santa Maria*, ISSN0103-8478.

Robert, K. (1999). Causes for Low White Blood Cells. www.ehow.com.

Roder, P.L., Scott, J.M. and Pegram, R.G. (1984). Acute *Trypanosoma vivax* infection of Ethiopian cattle in the apparent absence of tsetse, *Tropical Animal Health Production*, 16:141-147.

Richard, H. (2004). Molecular parasitology trypanosomes a eucaryote with a different way of doing thing. Return to the parasitology section of microbiology and immunology, on-line. This page copyright 2004, the board of trustees of the university of south Carolina, www.universityofsouthcarolina.com.

Russell, Z.E., Holly, M., Bruce, E.L. and Latimer, A. (2005). *Equine Babesiosis* - A review. Department of Pathology, College of Veterinary Medicine, University of Georgia, Athens, GA 3062-7388.

Saidu, S.N.A. (I990). Control of internal parasites of livestock. Extension bulletin, published by *National Agricultural Extension and Research Liaison Services, Federal Ministry of Science and Technology*, Ahmadu Bello University, Zaria. No. 48, Series 10, pp 14-6.

Sarah, W., Heather, L., Tarpley., Susan, E. and Kenneth, S.L. (2006). *Trypanosoma evansi* in horses (Class of 2006 Womack), Department of Parasitology (Little), Department of Pathology (Tarpley, Latimer), College of Veterinary Medicine, University of Georgia, Athens, GA 30602-7388.

Sally, W. and Micheal A. (2000). Oxford Advanced Learner's Dictionary. Sixth edition Oxford University, pp 378.

Schein, E. (1988). *Equine babesiosis*, In: Ristic, M. (cd.), babesiosis of domestic animals and man, CRC Press Boca Raton, Fla, p. I97-208.

Schinittger, L., Yin, H., Qi, B., Gubbels, J.M., Beyer. D., Niemann, S., Jongejan, E. And Ahmed, I.S. (2004). Simultaneous detection and differentiation of *Theileria* and *Babesia* parasites infecting small ruminants by reverse line blotting, *Parasitology Research*, 92:189-196.

Siefert, H.S.H. (1996). Vector borne diseases caused by protozoans, *Tropical Animal Health*, CTA Kluwer acadamic publishers.

Smyth, I.O. (1996). *Animal Parasitology*, Low price edition, Cambridge University Press, pp 140-143.

Snelson, J.T. (1975). Plant protection bulletin, Published by Food and Agricultural Organisation (FAO), 13:103.

Soulby, E.J.L. (1982). Helminth, Arthropods and protozoan of domesticated animal, Bailliere Tindall, London, pp 360-380.

Sparagano, O. (1999). Molecular diagnosis of *Theileria and Babesia* species. *Journal of Veterinary Parasitology*, 13:92-93.

Susan, E.A., and Asa, M. (1997). Ticks, *Merck's Veterinary Manual*, Eighth edition Marks and Co. Inc. U.S.A. pp 670-686.

Taylor, K. and Authei, E.M.L. (2004). Pathogenesis of animal trypanosomiasis, *The Trypanosomiases*, CABI, pp 331-354.

Tekdek, I., Gyang, E.O., Agbede, R.I.S. and Sanni, B.D. (1988). Sarcoptic Mange infestation in goats, three case reports, *Zariya Veterinarian*, 3(2):93-96.

Thomas, S.S. and lIiss, B. (1971). Renal function and its evaluation. In: Israel, D. and John. B.H., *Clinical Diagnosis by Laboratory Methods*, Fifteenth edition, W.B. Sauders company, London, pp 84-90.

Tony, B. (2002). Mechanism of immune evasion by pathogens, *Veterinary molecular Biology*, http//www.vetmed.ufl.Edu/path/teach/vem5131/publish/ve02017.htm.

Truc, P., Jamonneau, V.N., Guessan, P., Ndri L., Diallo, P.B. and Cuny, G. (1998). *Trypanosoma brucei* spp and *Trypanosoma congolense*: Mixed human infection in Cote d ivoir, *Society for Tropical Medical Hygiene*, 92:537-538.

Uilenberg, G. (1967). Note sur la piroplasmose equine a Madagascar, *Revue dEle Vage Medicine Veterinaire pays Tropicaux*, 20:156.

Uilenberg, G. (1981). Theilerial species in domestic livestock. In: Irvin, A.D., Cunningham, M.P., and Young, A.S. (eds), *Advances in the Control of Theileriosis*, Martinus Nijhoff publishers, The Hague, pp 37-44.

Uilenberg, G. (1995). *International Collaborative Research*. Significance or Tick-borne hemoparasitic disease to world animal health, *Veterinary Parasitology*, 57:19-41.

Uilenberg, G. (2001). *Babesiosis*. In: Service, M.W. (ed). Encyclopedia of arthropod transmitted infections of man and domesticated animals, CABI Publishing. Walling Ford, pp 50-53.

Valerie, B. (2005). The-Effects-Stress-and-Cortisol-Have-on-the-Body . www.articledashboard.com

Walker, A.R. and Koney, E.M. (1999). Distribution of ticks (Acari: *Ixodida*), Infesting domestic ruminants in Ghana. *Bulletin of Entomology Research*, 89:473-480.

WIKIHOW (2006). How to measure the weight of horse. http://www.wikihow.com/Use-a-Tape-to-Weigh-a-Horse

Willoughby, K. (2003).The ABC of PCR . In *Practice*, 25(3):140-145.

World Health Organization (WHO) (1998). Control and surveillance of African trypanosomiasis, *Technical Series*, 871.

World Health Organization (WHO) (1987). Parasitic diseases, *Human Babesiosis Weekly Epidemiological Records*, 62(28): 22.

Woo, P.T.K. (1960). The hematocrit centrifugation technique for detection of trypanosomes in blood, *Canadian Journal of Zoology*, 47:921-923.

Woo, P.T.K. (1970). The heamatocrit centrifugation technique for diagnosis of African trypanosomiasis, *Acta Tropica*, 27:263-386.

Woo, P.T.K. and Limebeer, R. (1971). Evidence of intrauterine transmission of trypanosome in cattle, *Acta Tropica*, 28:61-63.

Wosu, L.O. (2002). The Veterinarian's handbook, First edition. Mike social press, 4 University Road, Nsukka, pp 268.

Xuan, X., Nagai, A., Battsetseg, B., Fukumoto, S., Makala, L.H., Inoue, N., Igarashi, I., Mikami, T. and Fujisaki, K. (2001). Diagnosis of equine piroplasmosis in Brazil by serodiagnostic methods with recombinant antigens, *Journal of Veterinary Medical Science*, 63: 1159-1160.

Yahuza, A.M. (2005). Diversifying Nigerian Economy: Durbar as a viable product of culturaltourism. A seminar paper presented at the *National Research Center for Durbar Tradition*, Kaduna zonal office, January, 2005. advahuz@yahoo.com.

Young, A.S. and Morzaria, T.P. (1986). Biology of *Babesia*, *Parasitology Today*, 2:211-219.

Young, A.S., Groocock, C.M. and Kariuki, D.P. (1988). Integrated control of ticks and tick-borne diseases of cattle in Africa, *Parasitology*, 96: 403-432.

Zavagli, V. (1970). *Equine piroplasmosis*. In: Proceedings of Second International Conference on equine injectious diseases, Paris, 1969, pp 244-248.

APPENDICES

APPENDIX I

Normal Blood Values of Equines-Hematology and Other Blood Constituents

white blood cell count in a horse is	**5.4 to 14.3**	**$x10^9$/L**
monocytes,	**0-1**	**%**
segmented or mature neutrophils about	**60**	**%**
band or immature neutrophils	**0**	**%**
basophils,	**0-1**	**%**
eosinophils,	**0-1**	**%**
lymphocytes	**40**	**%**
PCV	**31- 47**	**%**
red blood cells count in a horse is	**5.50 to 12.90**	**$x10^9$ /L**
hemoglobin concentration for horses is	**8.0 to 19.0**	**grams per deciliter**
Cortisol	**83–359**	**nmol/l**
Total protein	**5.4-7.9**	**g/dL**

coutesy of

Margi,Sirois, veterinary Clinical laboratory procedures,published by Mosby New York,1995.

Les Sellnow **(2001)Equine health care from, *The Horse* magazine.http.www.the HORSE.com .Article # 868 October 16 2001. *Michael A. Ball, and Christina S. Cable,(2003) The Horse* magazine.http.www.the HORSE.com Article # 4514 August 01 2003**

APPENDIX II

The levels of parasiteamia in Babesia infected and Trypanosome infected Polo horses in Igabi and Kaduna North Local Government Areas

	LEVEL OF PARASITAEMIA			
parasites loction	**1-5 parasites (+)**	**6-10 parasites (++)**	**11-20 parasites (+++)**	**>20 parasites (++++)**
Babesia infected Igabi LGA	8	5	2	1
Babesia infected Kaduna North LGA	6	1	3	2
Trypanosome infected Igabi LGA	1	1	0	0
Trypanosome infected Kaduna North LGA	1	3	0	0

Key: + Very-few, ++ Few, +++ Many, ++++ High

APPENDIX III

The Mean (±SEM) body condition score,live wieght and some hematological and biochemical parameters of bebesia infected and babesia negative polo horses in Igabi and Kaduna North local Government Areas

Parameter	Babesia Infected n=	Babesia Negative n=
Weight (Kg)	476.90 ± 13.60[a]	466.90 ± 9.20[a]
Body Condition	3.40 ± 0.20[a]	4.60 ± 0.10[b]
Packed cell volume (pcv)%	27.30 ± 0.80[a]	34.30 ± 0.60[b]
Red blood cell counts ($x10^9$/L)	6.00 ± 0.20[a]	7.40 ± 2.10[b]
Hemoglobin concentration (g/dL)	9.00 ± 0.3 0[a]	11.40 ± 1.20[b]
White cell counts ($x10^9$/L)	6.10 ± 0.30[a]	7.20 ± 2.2 0[b]
Total protein (g/dL)	6.10 ± 0.30[a]	6.50 ± 1.20[a]
Cortisol (ng/ml)	68.80 ± 8.70[a]	53.30 ±1.30[b]
Temperature ^oC	39.08 ± 1.10[a]	38.04 ± 0.10[b]

NOTE: n=number of animals examined. a, b values in rows or columns unit of different super script differ significantly (P≤0.05).

APPENDIX IV

The Mean (±SEM) body condition score,live weight and some hematological parameters of trypanosome infected and trypanosome negative polo horses in Igabi and Kaduna North Local Government Areas

Parameter	Trypanosoma Infected n=	Trypanosoma Negative n=
Weight(Kg)	451.10 ± 24.60[a]	461.92 ± 15.20[a]
Body Condition	2.30 [a] ± 0.70 [a]	4.40 ± 0.10[b]
Packed cell volum (pcv)%	24.91[a] ± 0.80 [a]	32.57 ± 0.70[b]
Red blood cell counts ($x10^9$/L)	6.02[a] ± 0.30[a]	7.00 ± 0.10[b]
Hemoglobin concentration (g/dL)	8.80[a] ± 0.50[a]	10.7 0 ± 0.30[b]
White cell count ($x10^9$/L)	6.80[a] ± 0.10[a]	7.37 ± 1.10[b]
Total protein (g/d)	6.00[a] ± 0.60[a]	6.10 ± 0.70 [a]
Cortisol (ng/ml)	65.80 ± 10.70 [a]	63.40 ± 12.30[a]
Temperature ^oC	38.50 ± 1.00[a]	38.01 ± 0.60[b]

NOTE: n=number of animals examined. a, b values in rows or columns unit of different super script differ significantly (P≤0.05).

APPENDIX V

The Mean (±SEM) absolute differential leucocyte of babesia infected and babesia negative polo horses in Igabi and Kaduna North Local Government Areas

Parameter	Babesia Infected	Babesia Negative

Segmented Neutrophils ($x10^9$/L)	**7.50 ± 0.20^a**	**7.40 ± 0.60^a**
Monocytes($x10^9$/L)	**0.10 ± 0.00^a**	**0.10 ± 0.00^a**
Eosinophils ($x10^9$/L)	**0.10 ± 0.00^b**	**0.10 ± 0.00^b**
Lyphocytes($x10^9$/L)	**6.70 ± 0.10^b**	**6.60 ± 0.20^b**
Basophils ($x10^9$/L)	**0.40 ± 0.00^a**	**0.50 ± 0.00^a**

NOTE: n=number of animals examined.a, b values in rows or columns unit of different super script differ significantly (P<0.05).

APPENDIX VI

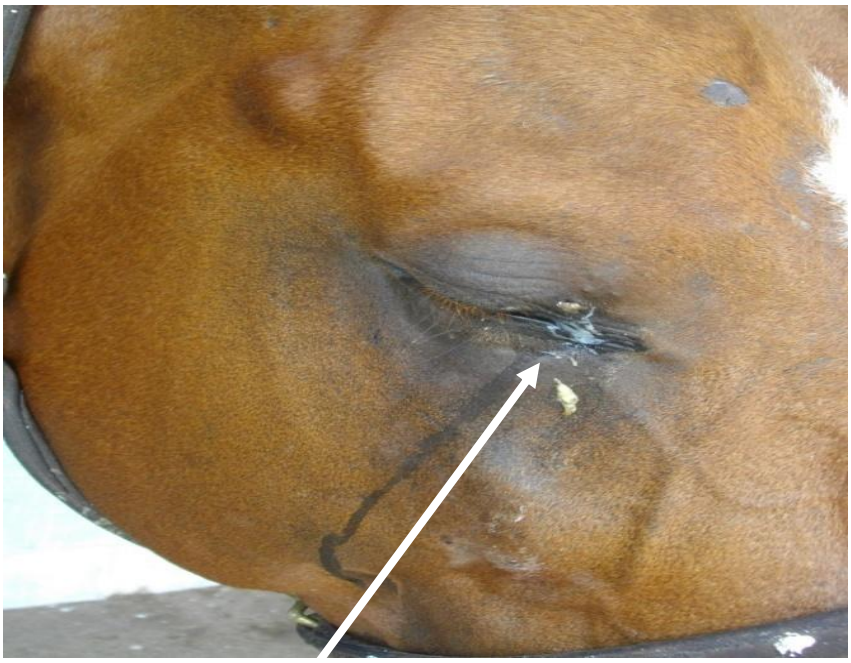
The (Mean ±SEM) absolute differentaial leucocyte of trypanosome infected and trypanosome negative polo horses in Igabi and Kaduna North local Government Areas

Parameters	**Trypanosome Infected** **n=**	**Trypanosome Negative** **n=**
Segmented Neutrophils ($x10^9$/L)	5.60 ± 1.70 [a]	7.40 ± 0.10[a]
Monocytes ($x10^9$/L)	0.00 ± 0.00[a]	0.10 ± 0.00[a]
Eosinophils ($x10^9$ /L)	0.00 ± 0.00 [a]	0.40 ± 0.10[a]
Lyphocytes ($x10^9$/L)	4.50 ± 1.40[a]	6.50 ± 1.60[b]
Basophils ($x10^9$/L)	0.10 ± 0.00 [a]	0.10 ± 0.00[a]

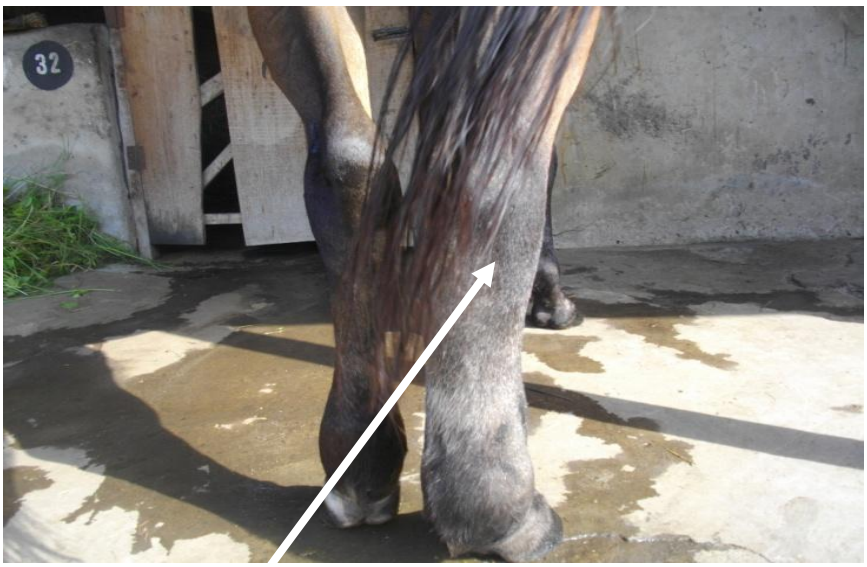
NOTE: n=number of animals examined.a, b values in rows or columns unit of different super script differ significantly (P<0.05).

APPENDIX VII

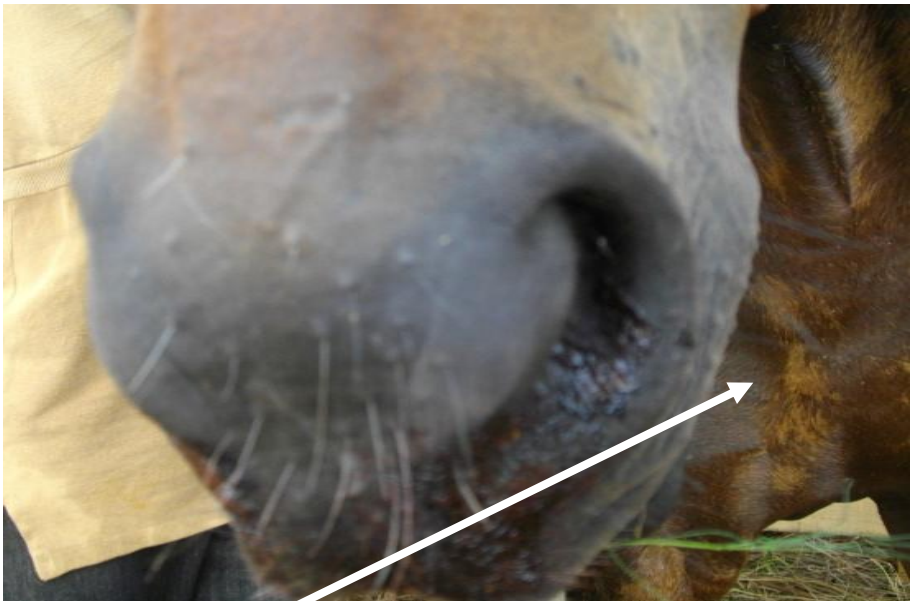
Clinical Signs Observed in:-



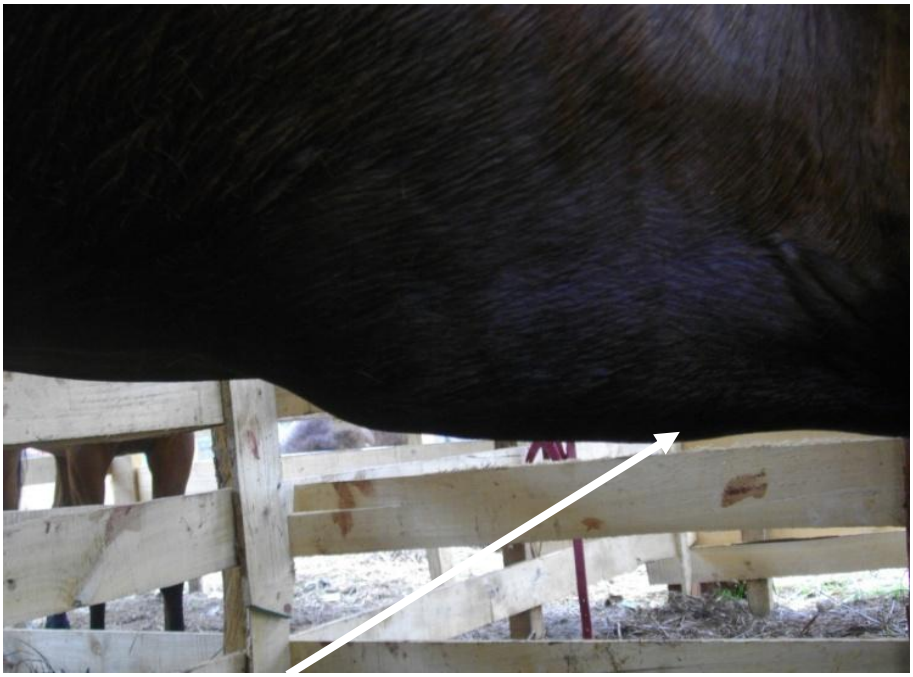
a: Occular discharge in babesiosis



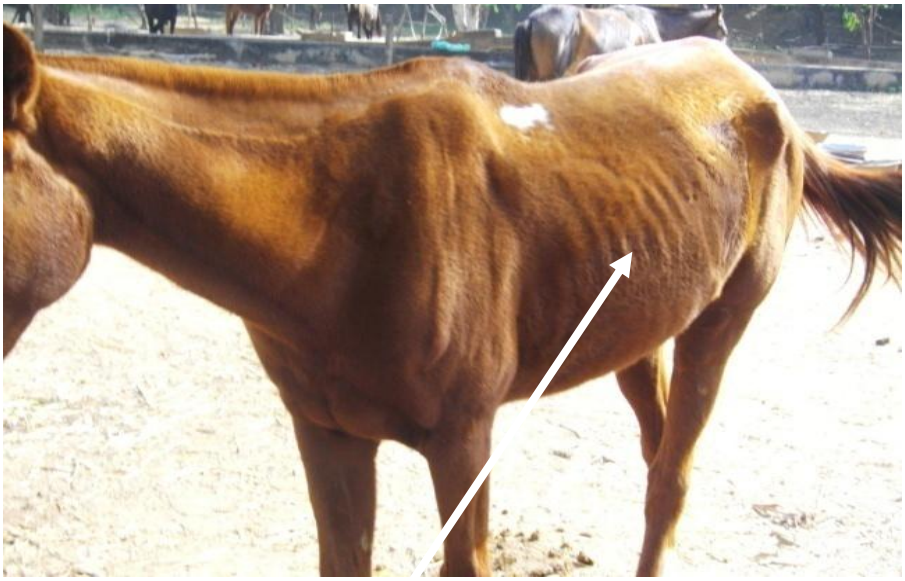
b: Oedema of the extrimities in babesiosis



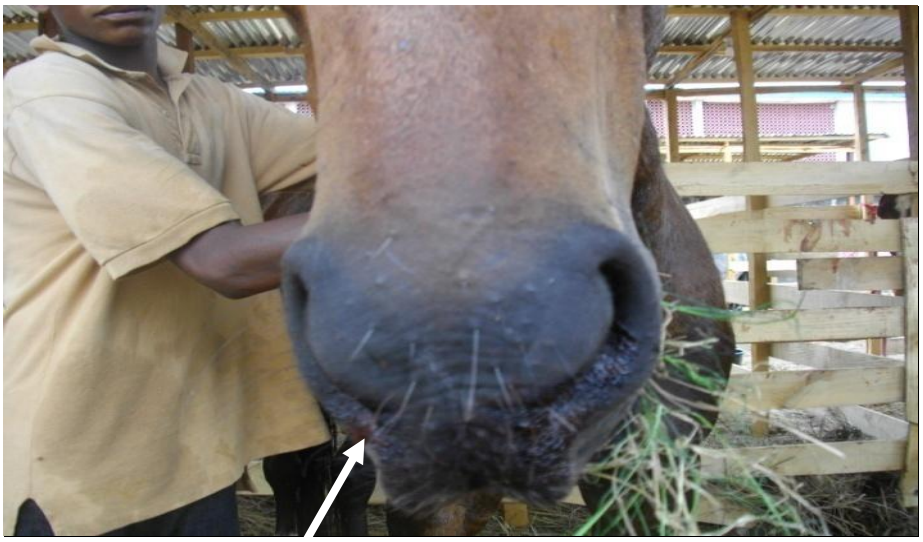
c: Profuse sweating in babesiosis



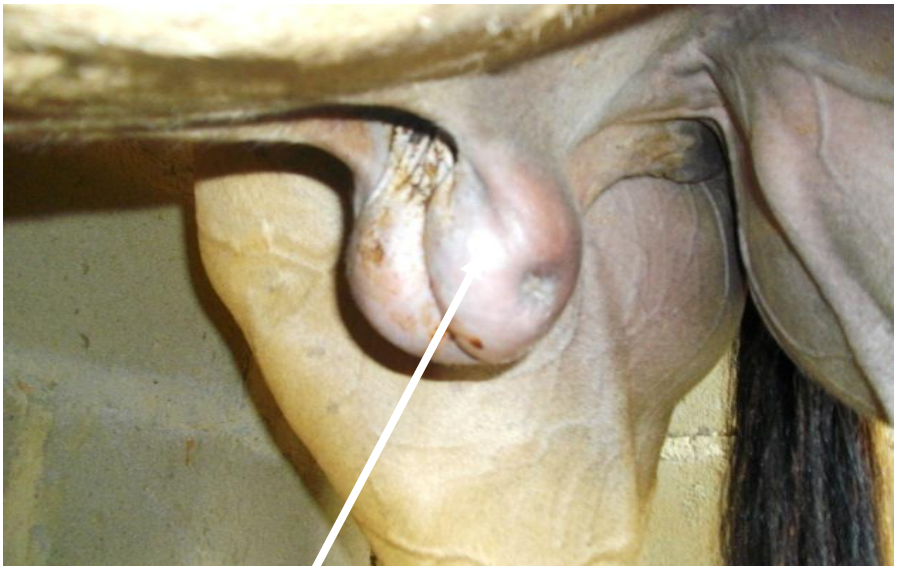
d: Oedema of the ventral region



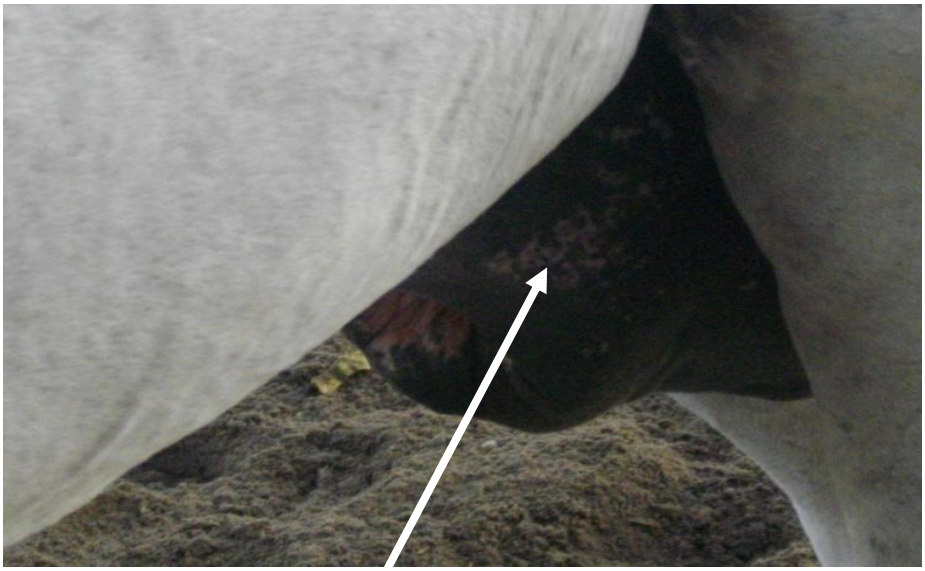
e: Emaciation in babesiosis



f: Bloody nasal discharge in babesiosis



g: Oedema of the prepuce in trypanosomosis in a geldin



h: Odema of the prepuce in trypanosomosis in a stallion