SURVEY OF SOME VIRAL, PARASITIC INFECTIONS AND HAEMATOBIOCHEMICAL PARAMETERS AT THE WILD-DOMESTIC ANIMALS' INTERFACE IN YANKARI AND SUMU WILDLIFE PARKS IN BAUCHI, NIGERIA

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A DISSERTATION SUBMITTED TO THE POSTGRADUATE SCHOOL AHMADU BELLO UNIVERSITY, ZARIA, IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE OF DOCTOR OF PHILOSOPHY IN FOOD ANIMAL MEDICINE

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FEBRUARY, 2018

DECLARATION

I declare that the work in the thesis entitled "Survey of some Viral, Parasitic Infections and Haematobiochemical Parameters at the Wild- domestic Animals' Interface in Yankari and Sumu Wildlife Parks in Bauchi, Nigeria" has been carried out by me in the Department of Veterinary Medicine, under the supervision of Profs. C.A. Kudi, P.A. Abdu, O.O. Okubanjo and CP Dr (Mrs) A. Abubakar-Baju. The information derived from the literature has been duly acknowledged in the text and a list of references provided. No part of this thesis was previously presented for another degree or diploma at any university.

Joel Yakubu ATUMAN	_____	_____
Name of Student	**Signature**	**Date**

CERTIFICATION

This Thesis entitled "SURVEY OF SOME VIRAL, PARASITIC INFECTIONS AND HAEMATOBIOCHEMICAL PARAMETERS AT THE WILD-DOMESTIC ANIMALS' INTERFACE IN YANKARI AND SUMU WILDLIFE PARKS IN BAUCHI, NIGERIA" by Joel Yakubu ATUMAN meets the regulations governing the award of the degree of Doctor of Philosophy of Food Animal Medicine of Ahmadu Bello University, Zaria and is approved for its contribution to knowledge and literary presentation.

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DEDICATION

I dedicate this work to God Almighty for the gift of wisdom and knowledge and to my wife Janet and son Nathaniel for their love, understanding and support.

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LIST OF ABBREVIATIONS

5'ND	5' Nucleotidase
AHS	African Horse Sickness
AHSV	African Horse Sickness Virus
ALP	Alkaline Phosphatase
ALT	Alanine Amino Transferase
AST	Aspartate Amino Transferase
BTV	Blue Tongue Virus
BUN	Blood Urea Nitrogen
BVD	Bovine Viral Diarrhoea
BVDV	Bovine Viral Diarrhoea Virus
CBC	Complete Blood Count
cELISA	Competitive Enzyme Link Immunosorbent Assay
CFT	Compliment Fixation Test
DLC	Direct Leukocyte Count
EDTA	Ethylene Diamine Tetraacetic Acid
ELISA	Enzyme Link Immunosorbent Assay
EM	Electron Microscope
EVF	Erythrocyte Volume Fraction

FCS	Foetal Calf Serum
FMD	Foot and Mouth Disease
FMDV	Foot and Mouth Disease Virus
GD	Dehydrogenase
GGT	Gama Glutamyl Transpeptidase
GIT	Gastro Intestinal Tract
GOT	Glutamate Oxalate
GPT	Glutamate Pyruvic Transaminase
HB	Haemoglobin Concentration
HCT	Haematocrit Centrifugation Test
IEP	Inter Epizootic Periods
IgG	Immunoglobulin G
IgM	Immunoglobulin M
IM	Intramuscular
LDH	Lactate Dehydrogenase
LGA	Local Government Area
Mab	Monoclonal Antibody
MCH	Mean Copuscular Haemoglobin
MCHC	Mean Corpuscular Haemoglobin Concentration

MCV	Mean Copuscular Volume
OCT	Ornithine Carbamyl Transferase
OD	Optical Density
PCR	Polymerase Chain Reaction
PCV	Packed Cell Volume
RBC	Red Blood Cell
RNA	Ribonucleic Acid
RT-PCR	Reverse Transcriptase Polymerase Chain Reaction
RVF	Rift Valley Fever
RVFV	Rift Valley Fever Virus
SAT 1	Sauth African Territory 1
SAT2	Sauth African Territory 2
SDH	Sorbitol Dehydrogenase
SWP	Sumu Wildlife Park
TLC	Total Leukocyte Count
VHF	Viral Haemorrhagic Fever
VNT	Viral Neutralization Test
WBC	White Blood Cell
YGR	Yankari Game Reserve

ABSTRACT

Wildlife play significant role in the transmission of several livestock diseases and pathogen spill-over may occur in complex socio-ecological systems at the wildlife-domestic animal interface which have been seldom studied. This study was carried out from October 2013 to October, 2016 during which the prevalence of some viral and parasitic infections and haematological and biochemical parameters in wildlife and domestic animals within and around Yankari game reserve (YGR) and Sumu wildlife park (SWP) in Bauchi State, Nigeria was detrmined. Blood, sera, faeces and ticks were collected from wildlife including 4 elephants (*Loxodonta Africana*), 11 waterbucks (*Kobus ellipsiprymus*), 1 hartbeest (*Alcelaphus baselaphus caama*), 24 elands (*Taurotragus oryx*), 53 zebras (*Equus quagga crawshayi*), 1 kudu (*Tragelaphus streptsiceros*) and 12 wildebeests (*Connochaetes taurinus*) and domestic animals including 300 cattle (*Bos indicus*), 200 donkeys (*Equus asinus*) and 200 horses (*Equus caballus*). Wild animals were immobilized using combination of etorphin Hcl (M 99) at 0.5-2 mg/kg and Azaperone at 0.1 mg/kg using a Dan-Inject rifle fitted with 3 ml dart syringe while domestic animals were restrained by their owners for samples collection. Antibodies to rift valley fever virus (RVFV), foot and mouth disease virus (FMDV) and African horse sickness virus (AHSV) were determined using Enzyme linked immunosorbent assay (ELISA) whereas Bovine viral diarrhoea virus (BVDV) and African horse sickness virus (AHSV) antigens were detected using conventional reverse transcriptase polymerase chain reaction (RT-PCR). Giemsa stained thin and thick blood smear and haematocrit centrifuge technique were used for haemoparasites identification. Gastrointestinal (GIT) parasites were identified using floatation and sedimentation techniques whereas ticks were identified morphologically using appropriate keys. Mean values for haematological and serum biochemical values for wildlife were determined using automated haematology analyzer (ABX ABC Vet,

Horiba ABX SAS Montpellier, France) and Ace chemistry analyzer (Alfa Wassermann, New Jersey, USA), respectively. Data generated were analysed using Graphpad Prism version 7 and levels of association between positivity and sex, breed, age and animal species were derived using Chi-square. Values of $P \leq 0.05$ were regarded as statistically significantly different. Seroprevalences in cattle and wildlife respectively were RVFV 11.3 % and 8.49 % ($P > 0.05$); FMDV 65.67 % and 24.53 % ($P < 0.05$) whereas AHSV in horses and zebras were 35.5 % and 20.8 % ($P < 0.05$). Antibodies to FMDV serotypes O, A, SAT 1 and SAT 2 were detected in elephants, waterbuck, eland and wildebeest. A BVDV detection rate of 9.67 % and 13.20 % were observed in cattle and wildlife respectively. AHSV antigen was detected from pooled tissues (spleen, liver and lymph nodes) of a zebra carcass. Overall prevalence of haemoparasites was higher in wildlife (56.6%) followed by cattle (42.5%) donkeys (31.5%) and horses (24.5%) whereas overall prevalence of GIT parasites was higher in donkeys 62.0 % followed by cattle 61.6 %, wildlife 59.4 %, and horses 27.0 %. Haemoparasites including *Babesia* spp, *Anaplasma marginale*, *Ehrlichia ruminantium* and *Trypanosoma* spp identified alone or in combination with others had significant ($P < 0.05$) effect on mean PCV of parasitized animals. *Fasciola* spp were the most prevalent helminths in cattle, elands, wildebeest and waterbucks whereas *Strongylus* spp were the most prevalent in zebras, donkeys and horses. *Amblyomma variegatum*, *Rhipicephalus* spp, *Boophilus* spp, and *Hyalomma* spp ticks were found infesting most of the wildlife species. The study provided a preliminary set of reference range values for haematogical and biochemical parameters of some wildlife in YGR and SWP in Bauchi State, Nigeria. Further study on molecular epidemiology of RVFV, FMDV, BVDV and AHSV and also on the identified parasites is advocated.

CHAPTER ONE

INTRODUCTION

1.1 Background of the Study

Disease outbreaks, epidemics or pandemics have been of importance for human and animal health worldwide and sparked enormous public interest (Diederich *et al.*, 2015). These outbreaks might be caused by known endemic pathogens or by emerging or re-emerging pathogens (Diederich *et al*., 2015). Wildlife are the major reservoirs and responsible for 70 % of these outbreaks (Kruse, 2004; Weiss *et al*., 2012; Pernet *et al*., 2014).

Wildlife play significant role in transmission of several livestock diseases and interspecific viral pathogen spill-over at the wildlife-livestock interface and have been of growing concern in the scientific community over the past years due to their impact on wildlife, livestock and human health (*Karesh et al*., 2005; Miguel *et al.*, 2013). Viral pathogens can be modified to cause severe out breaks in humans and animals due to gradual change in many related factors like environment and ecological factors (Martin *et al*., 2011; Oluwayelu, 2014). These factors include heavy rainfall, change in the onset of the rainy season (and thus an alteration of the interplay between rainfalls and reproduction of vectors and reservoir hosts) and changes in wind patterns (Sellers and Maarouf, 1991; Oluwayelu, 2014).

With the establishment of zoos and conservation areas (Odunlami and Lake, 2003; Messenger *et al*., 2014), wildlife-livestock interface are found in proximity to many protected areas in Africa (Daszak *et al*., 2000). Livestock is an important source of livelihood to people in Bauchi State, Nigeria. However, range land quality has been

dwindling particularly in grazing reserves due to droughts, desertification, global warming, floods and deforestation which have been identified as a major driver for emergence and re-emergence of viral infections (Jones *et al.*, 2008). Farmers respond to land grazing shortage by illegally grazing (poach grazing) livestock in the Yankari game reserve and Sumu wildlife park. Besides being illegal, the pattern of livestock movement in the reserve area can easily result in elevated probabilities of interspecies and cross species (spill-over) transmission of viral infections at the wildlife-livestock interface level (Shrivastav, 2001; Sarker, 2010). Moreover, extensive livestock production systems significantly influence the epidemiology of diseases in animals (Daszak *et al*., 2000).

Parasites regulate host populations in a natural environment by influencing the growth rate and reproductive potential of its host population and in some intances leading to host mortality (Van Wyk and Boomker, 2011). Knowledge about parasite infections in wildlife ecosystem therefore is essential. Parasitic infections transmitted between livestock and wildlife pose a significant risk to wildlife conservation efforts and constrain livestock productivity especially in tropical regions of the world (Vander Waal *et al.*, 2014). Ticks are ubiquitous blood feeding ectoparasites of animals and serve as vectors of a plethora of pathogens that cause severe infectious diseases in humans and livestock (Walker, 2003; Jongejan and Uilenberg, 2004). Tick infestation and the diseases they transmit result in huge economic losses in livestock production and continue to cripple the livestock industry, especially in sub- Saharan Africa (Jongejan and Uilenberg, 2004).

Haematological and biochemical studies are important tools for evaluation of health status of an animal (Douglas and Nelson, 1991). Examination of haematologic and serum biochemical parameters complement clinical assessments during health surveys of wild animal populations (Viggers *et al.,* 1993; Kock *et al.,* 2007). The physiological, nutritional and pathological conditions of animals are usually assessed using haematological and biochemical analyses (Awah and Nottidge, 1998; Opara *et al.,* 2006). Disease condition, nutrition, age, sex, genetics, reproduction, housing, starvation, environmental factors, stress and transportation are all known to affect haematological and biochemical parameters observed between tropical and temperate animals (Bush, 1991; Ogunsanmi *et al.,* 1993).

1.2 Statement of the Research Problems

The world is undergoing rapid ecological changes, so also the population of pathogenic organisms, their vectors and hosts which are capable of equally rapid change due to loss of biodiversity (Williams *et al*., 2002). This can lead to emergence and re-emergence of disease epidemics and is a threat to the health and survival of both wild and domestic animals (Brown and Slenning, 1996; Shrivastav and Singh, 2012).

Land use, human and animal movements and grazing by livestock have been considered as major factors in the spread of disease from domestic animals to wild populations and *vice versa* (Kock *et al*., 2002). The growth in human population has resulted in greater use of land thereby establishing human settlements around the game reserves and bringing livestock closer to wild populations (Kock *et al*., 2002; Jones *et al.*, 2008). Also, increase in the demand for protein has led to expansion of livestock farms and ranches thereby narrowing wildlife-livestock interface (Kock *et al*., 2002). Other risk

factors for pathogen spread at the wild-domestic animal interface include long distance live animal transportation, live animal markets, and building of dams, bush meat consumption and habitat destruction (MacLachlan and Dubovi, 2011). These have resulted in the emergence and spread of novel viral pathogens that are of economic and public health concern (Taylor *et al.,* 2001; Calisher *et al.,* 2006; Jones *et al.,* 2008; Kaur *et al*., 2008; Smith *et al.,* 2014). These viral pathogens have also been described as national security threats in the United States of America (Brower and Chalk, 2003).

With rapid human population growth on the African continent, wildlife is now more confined to restricted areas such as national parks and game reserves (Harmon and Brechin, 1994). In Nigeria and particularly in Bauchi State, livestock and farm lands and human settlements are located in the conservation area of Yankari Game Reserve (Omondi *et al.,* 2006; Nyanganji *et al.,* 2012), with conflicts and increasing potentials for pathogen spread at the level of wildlife and domestic animal interface (Atuman *et al*., 2014).

In Nigeria, a sharp rise in mean air temperature from 26.04 ℃ to 27.83 ℃ was reported between 1971 and 2005 (Odjugo, 2010). Elevation of ambient temperature might facilitate the proliferation of insects like mosquitoes (Oluwayelu, 2014) which may lead to increase in prevalence of some insect-borne livestock diseases such as Rift valley fever (RVF) and African horse sickness (AHS) (Gray, 2008; Oluwayelu, 2014). Most domesticated and wild animals perform optimally at temperatures between 10 and 30 □C (Das *et al*., 2016). At temperatures above 30 □C, most domestic and wild animals would reduce their feed intake by 3–5% for each unit temperature increase (Dittmar *et al*., 2014). Changes in temperature also trigger the secretion of stress hormones which

suppress immunological responses including the function of the white blood cells, tipping the host-pathogen interactions in favour of the pathogen (Dittmar *et al*., 2014).

Wildlife are capable of living with high parasite loads without any apparent ill-effect on their health (Malan *et al*., 1997). A number of studies have demonstrated that wildlife may be carriers of gastrointestinal parasites (Karere and Munene, 2002), ectoparasites (Kalema-Zikusoka *et al.,* 2002) and haemoparasites (Nizeyi *et al.,* 2001; Mutani *et al.,* 2003). The impact of spillover of human and livestock parasites to naïve species of wildlife and spill-back from wildlife is another emerging threat of potential public health and economic significance to humans, wildlife and livestock (Conradie, 2008). Ticks suck blood of their hosts resulting into severe anaemia, loss of production, weakness and immunosuppression (Agbede, 1981; Gwakisa *et al*., 2001) as well as damages to hides and skin leading to significant financial losses to livestock farmers (Biswas, 2003). Production losses due to ticks and tick-borne diseases around the globe were put at US$ 13.9 to US$ 18.7 billion annually (de Castro, 1997; Wall, 2000).

There are a number of bacterial, viral and parasitic diseases present at the wildlife-domestic animal interface and quite a few of these are zoonotic and emerging globally (Daszak *et al.,* 2000). These diseases can affect the haematological and biochemical normal values depending on the level of infections in wildlife (Rao and Acharyjo, 2002). Haematological studies are of ecological and physiological interest in helping to understand the relationship of blood characteristics of animals to the environment (Ovuru and Ekweozor, 2004) and so could be useful in the selection of animals that are genetically resistant to certain diseases and environmental conditions (Mmereole, 2008; Isaac *et al.,* 2013).

1.3 Justification of the Study

The outbreaks of diseases like ebola (WHO, 2014) and Monkey pox (WHO, 2017) and their spread around the world have illustrated the weakness of disease surveillance and early warning signs (Diederich *et al*., 2015). Minimizing the risk of disease transmission must be considered in wildlife conservation programs as disease epidemics have continued to be a threat to the health and survival of wildlife, humans and domestic animals (Shrivastav and Singh, 2012). Therefore, prompt and accurate diagnosis of conditions and diseases affecting wildlife and domestic animals is essential for facilitating timely treatment, reducing mortalities, and preventing the spread of disease with the overall goal of improving health, livelihood and conservation outcomes (Daszak *et al.*, 2000).

In Nigeria, reports on activities of RVFV, an arthropod-borne virus that infects a wide range of vertebrate hosts (humans and animals) date back to the 1970s and 1980s with the latest being in the mid-1990s (Olaleye *et al.*, 1996). Although, previous virological and serological surveys in the country revealed human and animal infections with RVFV (Fagbami *et al.*, 1973; Tomori, 1979;1980; Ezeifeka *et al.*,1982; Olaleye *et al.*, 1996). Little work has been done on this virus in recent years. However, a recent survey of human infections in Borno State indicated a seroprevalence of about 14.1% for RVFV-IgG (Bukbuk *et al.*, 2014).

Foot and Mouth Disease is one of the most economically important transboundary disease of animals in the world and it is extremely difficult to control (Brown and Slenning, 1996). In wildlife, pathogenesis of FMD varies from a completely inapparent to a rare acutely lethal infection. This makes diagnosis difficult either because the

variability in the severity of presenting clinical signs is greater than in domestic livestock or because it tends to be subclinical for the particular species (Thomson *et al.,* 2003; Arzt *et al.,* 2011; Miguel *et al.,* 2013). Presence of antibodies against the FMDV in several wildlife species has been documented in studies conducted in different countries of the African continent, but mainly within its eastern and southern regions (Williams *et al*., 2002; Bronsvoort *et al.,* 2008; Vosloo *et al.,* 2009). Such studies have not yet been conducted in Nigeria.

Bovine viral diarrhoea (BVD) is a disease of cattle but the virus can also be found in sheep, goats and wild ruminants, which may serve as reservoirs (Passler and Walz, 2010). The economic losses associated with BVDV are due to abortion, still births, congenital abnormalities, increased neonatal mortality, prenatal and postnatal growth retardation and persistently infected calves (Handel *et al.,* 2011). Various works have so far been carried on the prevalence of BVDV in both domestic and wild animals' worldwide (Aguirre *et al.,* 1995; Paton *et al.,* 1998; Ferreira *et al.,* 2000; Falcone *et al.,* 2001; Viltrop *et al.,* 2002; Handel *et al.,* 2011). In Nigeria, information on BVDV in domestic animals are few (Baba *et al*., 1994; Bello *et al*., 2016) and of immense concern is the paucity of information on BVDV in wildlife despite their abundance in zoos and game reserves across the Country.

The epizootiology of AHS in Nigeria has shown that the virus is circulating among horses with most of the cases associated with polo horses (Baba *et al.,* 1994; Kazeem *et al*., 2008; Fasina *et al.,* 2008; Ehizibolo *et al*., 2014). Local breed of horses are mostly owned and are used for social and economic value in Nigeria (Mshelia, 2010). There is paucity of information on AHS among the local breeds of horses despite the presence of

the vertebrate and invertebrate hosts and other ecological factors that favour the establishment of AHS in Bauchi State, Nigeria.

Wildlife tourism and ecotourism are becoming popular in Bauchi State due to the presence of YGR and SWP (Olokesusi, 1990). Biodiversity conservation programs are constrained due to increasing human activities (farming, grazing, hunting, felling of trees for firewood) at the YGR which could contribute to transmission of infectious agents through contaminated water, food or vectors (lice, fleas, ticks and mites) (van Vuuren and Penzhorn, 2015; Ernieenor *et al*., 2017). The presence of these pathogens may alter the normal physiology of the wildlife species (Shah, 1987). One certainty is that there is increasing need for veterinary input in the fields of wildlife disease in recognition of the growing intensity of wildlife, humans and domestic animal interactions and the emergence of diseases (Williams *et al.*, 2002).

Haematological and biochemical parameters have been reported in captive (Ogunsanmi *et al*., 2002) and wild grasscutters (Opara *et al*., 2006). However, these parameters in elephants, waterbucks, zebras, elands, and wildebeests in YGR and SWP in Buachi State, Nigeria have not been reported. There is the need to document these values considering the fact that wildlife capture and translocation to zoological gardens across the country is on the increase and the zoological parks in Bauchi State are known to be the major sources of wildlife.

To date, holistic approaches on identifying the diversity of putative reservoir species for RVFV, AHSV, BVDV, and FMDV are scarce. This study was planned to fill this gap

by specifically focusing on the interface of wildlife and domestic animals because this is where the risk of spillover infections is highest.

1.4 Aim of the Study

This study aimed to detect the presence of some viral and parasitic infections and determine haematological and biochemical parameters in some apparently healthy wildlife and domestic animals in Yankari game reserve (YGR) and Sumu wildlife park (SWP) in Bauchi State, Nigeria.

1.5 Objectives of the Study

1. Determine the sero-prevalence of RVFV, FMDV, and AHSV
2. Detect the presence of BVDV and AHSV antigens
3. Identify the haemo, endo and tick parasites
4. Determine the values of some haematological and serum biochemical parameters of some wildlife

1.6 Research Questions

1. What are the seroprevalences of RVFV, FMDV and AHSV?
2. What are the rates of BVDV and AHSV antigens?
3. What are the rates of haemo, endo and tick parasites?
4. What are the mean haematological and serum biochemical values in wildlife?

CHAPTER TWO

LITTERATURE REVIEW

2.1 Emerging and Re-emerging Viral Pathogens

2.1.1 Role of wildlife in disease outbreaks

The potential threat of emerging and re-emerging viral pathogens to the livestock, wildlife and human population in Africa and Europe has recently come to a broad scientific and public attention as they are of immense public health and economic concern (Johnson, 2013). The role of African wildlife in the occurrence of vector-borne infections in domestic animals has gained renewed interest as emerging and re-emerging infections occur worldwide at an increasing rate (Van Vuuren and Penzhorn, 2015). Large and devastating disease outbreaks and rapid epidemic or even pandemic pathogen spread have been of pivotal importance for human and animal health worldwide. These outbreaks or clusters of human or animal diseases might be caused by known endemic pathogens or alternatively by emerging or re-emerging pathogens (Diederich *et al.*, 2015). The emergence of novel pathogens is driven by multiple factors related to the pathogen itself, the reservoir, and livestock populations (Johnson, 2013). Changes in biodiversity and land use in particular have been identified as major drivers of disease emergence (Jones *et al.,* 2008). For some viral infections, wildlife may act as a reservoir through the inter-epidemic circulation of viruses with mild or subclinical manifestations. Wildlife may also act as introductory or transporting hosts when moved to new regions, e.g. Rift Valley fever virus and African horse sickness virus. Wildlife may also act as amplifying hosts when exposed to viruses in the early part of the warm season when vectors are active, with spill over to domestic animals later in the season, e.g. African horse sickness (Van Vuuren and Penzhorn, 2015).

2.1.2 Rift valley fever

2.1.2.1 Aetiology of rift valley fever

Rift Valley fever virus (RVFV) is an emerging zoonotic disease of public and animal health concern (Jori *et al.,* 2015). It is endemic in many countries in Sub-Saharan Africa and is responsible for severe outbreaks in livestock characterized by a sudden onset of abortions and high neonatal mortality which results in significant economic losses (Jori *et al.,* 2015). The virus was first identified in 1931 during an investigation into an epidemic among sheep on a farm in the Rift Valley of Kenya (Bishop *et al.,* 1980). RVF virus is a member of the Phlebovirus genus, one of the five genera in the family *Bunyaviridae*. It is an enveloped spherical virus with a diameter of 90–110nm. The envelope is composed of a lipid bilayer containing two glycoproteins. The genome consists of three single-stranded RNA segments designated large (L), medium (M) and small (S). The L and M segments are of negative polarity. The L segment encodes the viral RNA-dependent RNA polymerase (L protein). The M segment encodes the precursor to the glycoproteins Gn and Gc, and two non-structural proteins, designated NSm1 and NSm2. The S segment utilises an ambisense strategy to code for two proteins, the nucleoprotein N and a non-structural protein NS (Clements and Pfeiffer, 2009).

The RVF virus belongs to a group of agents causing viral haemorrhagic fevers (VHFs) namely, the taxonomically diverse RNA viruses of the *Arenaviridae, Bunyaviridae, Flaviviridae* and *Filoviridae* families. Outbreaks of VHFs have severe implications, such as public panic, disruption of social life and commerce, restrictions on the trade animals and animal products, and fear of wider and uncontrolled spread (Jori *et al.,* 2015). The capacity of the virus to spread into new territories and re-emerged in

endemic regions after long periods of silence to cause large outbreaks in human and animal populations constitutes a threat to public and veterinary health. The presence of mosquito vectors (*Aedes mcintoshi*) wide range of mammals susceptible to the virus, global changes in climate and increased animal trade and travel are some of the factors which might contribute to RVF wide spread globally (Paweska, 2015). In endemic areas, a number of rodent species have also been incriminated as putative reservoir species for RVFV (Olive *et al.,* 2012).

2.1.2.2 Host distribution of rift valley fever

RVF is able to infect many species of animals causing severe disease in domesticated animals including cattle, sheep, camels and goats (Madani *et al.,* 2003). Sheep appear to be more susceptible than cattle or camels (Jori *et al.,* 2015). Age has also been shown to be a significant factor in the animal's susceptibility to the severe form of the disease with mortality rates reaching 30% in adult animals and up to 100% in young animals (Madani *et al.,* 2003). An outbreak of RVF in animals frequently manifests itself as a wave of unexplained abortions among livestock and may signal the start of an epidemic. The rate of abortion among pregnant infected cattle, sheep and goats is almost 100% (King, 2004). Many African wildlife species have tested positive for antibody against RVFV, including: topi (*Damaliscus korrigum*); red-fronted gazelle (*Eudorcas rufifrons*); dama gazelle (*Nanger dama*); scimitar-horned oryx (*Oryx dammah*); common reedbuck (*Redunca redunca*); African buffalo (*Syncerus caffer*); Dorcas gazelle (*Gazella dorcas*); Thomson's gazelle (*Gazella thomsonii*); gerenuk (*Litocranius walleri*); lesser kudu (*Tragelaphus strepsiceros*); impala (*Aepyceros melampus*); sable antelope (*Hippotragus niger*); waterbuck (*Kobus ellipsiprymnus*); warthog (*Phacochoerus aethiopicus*); African bush elephant (*Loxodonta africana*); giraffe

(*Giraffa camelopardalis*); Burchell's zebra (*Equus burchellii*); and black rhinoceros (*Diceros bicornis*) (Olive *et al.,* 2012; Britch *et al.,* 2013). Although serological evidence suggests that a large number of African wildlife species might play a role in the epidemiology of RVF, their possible role in the cryptic maintenance of the virus is poorly understood (Olive *et al.,* 2012; Britch *et al.,* 2013; Paweska, 2015).

2.1.2.3 Mode of transmission and epidemiology of rift valley fever virus

Several different species of mosquito are able to act as vectors for transmission of the RVF virus. The dominant vector species varies between different regions and different species can play different roles in sustaining the transmission of the virus.Among animals, the RVF virus is spread primarily by the bite of infected mosquitoes of the subgenera *Neomelaniconion* (genus *Aedes*) and *Culex* (genus *Culex*) which can acquire the virus from feeding on infected animals (Rweyemamu *et al.,* 2012). The female mosquito is also capable of transmitting the virus directly to her offspring via eggs leading to new generations of infected mosquitoes hatching from eggs (Anyamba *et al.,* 2001). This accounts for the continued presence of the RVF virus in enzootic foci and provides the virus with a sustainable mechanism of existence as the eggs of these mosquitoes can survive for several years in dry conditions. During periods of heavy rainfall, larval habitats frequently become flooded enabling the eggs to hatch and the mosquito population to rapidly increase, spreading the virus to the animals on which they feed (Anyamba *et al.,* 2001). Biting flies such as midges, phlebotomids, stomoxids and simulids might serve as mechanical transmitters of infection.There is also a potential for epizootics and associated human epidemics to spread to areas that were previously unaffected. This has occurred when infected animals have introduced the virus into areas where vectors were present and it is of particular concern. When

uninfected Aedes and other species of mosquitoes feed on infected animals, a small outbreak can quickly be amplified through the transmission of the virus to other animals on which they subsequently feed on (Paweska, 2015).

In eastern and southern Africa, large outbreaks of RVF occur at irregular intervals of up to 15 years and are strongly associated with heavy rainfall and floods (Murithi *et al.*, 2011). The fate of the virus during long interepizootic periods (IEP) is, however, not well understood. Cryptic maintenance and transmission cycles have been postulated following isolation of RVFV from the floodwater *Ae.mcintoshi* mosquitoes collected during IEP in Kenya (Linthicum *et al.,* 1985). Based on these findings, *aedes* mosquitoes are generally accepted as being capable of maintaining the virus in nature by trans-ovarian transmission. It is believed that the virus can survive in dried aedine mosquito eggs for a long period of time. However, as yet, RVFV has never been isolated from the eggs of *aedes* mosquitoes and the mechanism through which the virus could survive for long periods is not understood (Linthicum *et al*., 1985). Of potential epidemiological importance is the finding that the larvae of *Cx. pipiens, Ae. mcintoshi* and *Ae. Circumluteolus* become infected with RVF after feeding on liver homogenates from experimentally infected animals (Turell *et al.,* 1990). Flooding is one of the major factors contributing to the emergence of RVF, after flooding of *aedes* mosquito breeding habitats (grasslands with shallow depressions called dambos or pens), the floodwater *aedes* mosquitoes are responsible for the primary cycle of RVFV transmission and the virus is then carried further afield in a secondary transmission cycle by *culicine* mosquitoes. While *aedes* mosquitoes tend to remain in the immediate vicinity of their breeding sites, *Culex* mosquitoes disperse widely to feed on vertebrate hosts (Linthicum *et al.,* 1985; Pepin *et al.,* 2010). Epidemics do not occur as a result of

lateral spread from a single source, but rather as a result of the intensification of vector activity following their massive and multiple emergences over large endemic areas. This would explain why localised heavy rainfall does not precipitate an epidemic (King, 2004). Flooding also increases the concentration of animals and humans on areas of dry land, thus further increasing the potential for virus transmission. The RVF outbreaks in North and West Africa are associated with mosquito vectors breeding in rivers and dams (Gerdes, 2004). The construction of large dam systems in North (Aswan dam) in Egypt and West Africa (Diama dam) in Senegal significantly increased mosquito breeding sites and also led to a high concentration of people and livestock in the proximity of the dams during severe drought conditions. In these regions, breeding conditions are more suited for flood-breeding *aedine* mosquitoes of the subgenera Aedimorphus, including *Ae. vexans, Ae. ochraceus, Ae. dalzieli,* and *Ae. Cumminsii* (Fontenille *et al.,* 1998). During the second large outbreak of RVF in this region in 1998–1999, there was apparently a shift in the dominant species towards *Mansonia uniformis* and *Cx. Poicilipes* due to their abundance, distribution and preference for humans and sheep (Jupp *et al*., 2002). Irrigation for agriculture in the Tihama regions of Yemen and Saudi Arabia and the proximity of the Jizan Dam in Saudi Arabia provide suitable breeding grounds for *Ae. Vexans* and *Cx. Tritaeniorhynchus* (Jupp *et al.,* 2002). Although mosquito bites are the principal infection mechanism of RVF in animals, during large epidemics other transmission mechanisms also play a role, including active vector dispersal, movements of infectious animals, and passive vector dispersal (Métras *et al.,* 2012).

2.1.2.4 Clinical manifestations of rift valley fever in animals

There are marked differences in the patterns of RVF in domestic animals, varying from moderate to serious clinical disease. New born lambs and kids are extremely susceptible to RVFV infection, with mortality exceeding 90% two days after the onset of illness in animals less than a week old. Lambs and kids older than two weeks and mature sheep and goats are less susceptible to the disease but some animals may die peracute (Swanepoel and Coetzer, 2004). Most of young develop an acute disease with high fever, anorexia, weakness, listlessness and hyperpnoea. Some animals may develop melaena or foetid diarrhoea and a blood-tinged, mucopurulent nasal discharge. Most adult sheep and goats undergo subclinical infection. The disease in calves resembles that in lambs and sheep, but a higher proportion of calves may develop icterus (Swanepoel and Coetzer, 2004). Death generally occurs two to eight days after infection, and mortality is usually less than 10%. Infection is frequently inapparent in adult cattle, but some animals develop acute disease (Swanepoel and Paweska, 2011). Abortion is an almost inevitable outcome of infection in pregnant sheep, goats and cattle. Animals may abort at any stage of gestation. However, abortion rates vary with epidemiological circumstances, and have ranged from 15% to 100% in different outbreaks, or in separate herds and flocks in a single outbreak. Frequently, abortion may be the only overt manifestation of disease in a herd or flock (Swanepoel and Paweska, 2011).

2.1.2.5 Diagnosis of rift valley fever

Acute RVF can be diagnosed using several different methods. Serological tests such as enzyme-linked immunoassay (the "ELISA" or "EIA" methods) may confirm the presence of specific IgM antibodies to the virus, both IgM and IgG are produced early

in RVF virus infection. The virus itself may be detected in blood during the early phase of illness or in post-mortem tissue using a variety of techniques including virus propagation (in cell cultures or inoculated animals), antigen detection tests, immunohistochemistry and RT-PCR (Pepin *et al.,* 2010). Single cases of RVF may be confused with many viral diseases of ruminants causing sudden death associated with generalised lymphadenopathy and haemorrhages. However, RFV should always be considered when there is a sudden onset of abortions and acute death in young animals, associated with necrotic lesions in all cases. Liver necrotic lesions are pathognomonic for RVF but laboratory confirmation is advisable (King, 2004).

2.1.2.6 Preventive control measures of rift valley fever in animals and humans

As most human cases of RVF are relatively mild and of short duration, no specific treatment is required for these patients. For the more severe cases, the predominant treatment is general supportive therapy (Gerdes, 2004). The most effective way of protecting animals and humans against RVF is by vaccination. The only vaccine cleared for human use is the killed product available only from the United States Army Medical Research and Material Command (USAMRMC). It is an initial three dose series for protective immunity with annual booster inoculations required to maintain that immunity and has been used to elicit immune response among laboratory staff in portharcourt, Nigeria (Frank-Peterside, 2000). A live-attenuated and killed vaccine is available for livestock. The live-attenuated Smithburn strain provides long-lasting immunity but it produces a teratogenic effect in sheep when administered in the first trimester and it is abortogenic in pregnant ewes (Frank-Peterside, 2000). Apart from these, the vaccine has been documented experimentally not to protect against the Nigeria and Lunyo strains of the virus in West African dwarf sheep (Tomori, 1979). The

live virus vaccines are recommended for use in endemic areas, while the killed vaccines are recommended for use outside endemic areas. Also, a formalin-inactivated vaccine is safe for pregnant ewes but provides only short-term immunity and hence requires a booster inoculation to maintain durable immunity (Swanepoel and Coetzer, 2004). An animal immunisation programme based on the use of a naturally occurring RVFV mutant (Clone-13) was recently introduced in South Africa (Gerdes, 2004). Immunisation of professionals at high risk of infection is implemented on a voluntary basis through the use of an experimental inactivated RVF vaccine (Gerdes, 2004). Outbreaks of RVF in animals can be prevented by a sustained programme of animal vaccination. Animal immunization must be implemented prior to an outbreak if an epizootic is to be prevented. Once an outbreak has occurred animal vaccination should NOT be implemented because there is a high risk of intensifying the outbreak (Gerdes, 2004). During mass animal vaccination campaigns, animal health workers may, inadvertently, transmit the virus through the use of multi-dose vials and the re-use of needles and syringes. If some of the animals in the herd are already infected and viraemic (although not yet displaying obvious signs of illness), the virus will be transmitted among the herd, and the outbreak will be amplified. Restricting or prohibiting the movement of livestock may be useful in slowing the spread of the virus from infected to uninfected areas (Gerdes, 2004).

Detecting animal cases during the early stages of an outbreak and sharing information about the cases with public health sectors is critical for implementation of timely control measures. Forecasting models and early warning systems using satellite images and weather/climate prediction data are useful for signalling increased risk of RVF outbreaks (Britch *et al*., 2013). Vector control of mosquito populations and restricted

movements during outbreaks can be effective measures since animals have high levels of viraemia and consequently amplify transmission. Educational campaigns to prevent human exposure to infected animal tissues or abortuses and exposure to mosquito vectors are effective. Vaccination of people in high-risk occupations is equally effective in combating an outbreak of RVF (Britch *et al*., 2013).

2.1.2.7 Public health education and risk reduction

During an outbreak of RVF, close contact with animals, particularly with their body fluids and tissues either directly or via aerosols, has been identified as the most significant risk factor for RVF virus infection. In the absence of specific treatment and an effective human vaccine, raising awareness of the risk factors of RVF infection as well as the protective measures individuals can take to prevent mosquito bites, is the only way to reduce human infection and deaths. Appropriate protective clothing should be worn and care taken when handling sick animals or their tissues or when slaughtering animals. In the epizootic regions, all animal products (blood, meat and milk) should be heat processed before eating (Swanepoel and Coetzer, 2004). The importance of personal and community protection against mosquito bites through the use of impregnated mosquito nets, personal insect repellent if available, by wearing light coloured clothing (long-sleeved shirts and trousers) and by avoiding outdoor activity at peak biting times of the vector species (Swanepoel and Coetzer, 2004). Larviciding measures at mosquito breeding sites are the most effective form of vector control if breeding sites can be clearly identified and are limited in size and extent. During periods of flooding, however, the number and extent of breeding sites is usually too high for larviciding measures to be feasible (Fontenille *et al*., 1998).

2.1.3 Foot and mouth disease

2.1.3.1 Aetiology of foot and mouth disease

Foot and mouth disease (FMD) is a highly contagious disease affecting artiodactylae, mostly cattle, swine, sheep, goats, and many species of wild ungulates (Brooksby, 1982). Foot-and-mouth disease virus (FMDV) was the first recognized viral pathogen and is the sole member of the genus Aphthovirus belonging to the Picornaviridae family. Seven immunologically different serotypes of the FMDV are known, namely, O, A, C, Asia-1, South-African Territories (SAT) -1, -2 and -3, which comprise more than 65 subtypes (Longjam *et al.,* 2011). FMD affects extensive areas worldwide and is included in the list of diseases notifiable to the World Organization for Animal Health. It is recognized as a significant epidemic disease threatening the cattle industry since the sixteenth century and till date it is a major global animal health problem. The history of FMD may be traced to era of Hieronymus Fracastorius, a monk who described a disease outbreak in 1546 A.D. that occurred in cattle near Verona, Italy (Longjam *et al.*, 2011). Almost 400 years later Loeffler and Frosch (1897) demonstrated that a filterable agent is responsible for FMD. This was the first demonstration that a disease of animal was caused by a filterable agent and ushered in the era of virology (Longjam *et al.,* 2011).

2.1.3.2 Molecular structure of foot and mouth disease virus

The FMD viral particle, or virion, contains a single-stranded RNA of positive polarity, approximately 8500 nucleotides long. It is an icosahedral particle with a smooth surface and a diameter of about 30 nm (Ryan *et al.,* 1989). The fine structure of the virus has been described by X-ray crystallography (Acharya *et al.,* 1989; Logan *et al.,* 1993). There are 60 copies of each of the structural protein VP1, VP2, VP3, and VP4. While the first three structural proteins (MW≈24 kDa) have surface components, the fourth

(MW≈8.5 kDa) is internal. The virion is also usually composed of one or two units of VP0, the precursor of VP2 and VP4 (Acharya *et al.,* 1989; Logan *et al.,* 1993). The structural proteins, VP1-3 fold into an eight-stranded wedge-shaped 13-barrel which fits together to form the majority of the capsid structure (Acharya *et al.,* 1989). The three-dimensional structure of FMDV has revealed a prominent surface feature formed by the loop between the G and H strands of VP1. The VP4 protein is located inside the capsid (Belsham *et al.,* 1991). The strands of the 13-barrel of VP1-3 are connected by loops which form the outer surface of the virion (Jackson *et al.,* 2002). Unlike other picornaviruses, FMDV lacks a surface canyon or pit which is the receptor binding site for entero- and cardioviruses (Belnap *et al.,* 2000). Another feature of the virion is the channel at the fivefold axis which permits the entry of small molecules such as Cscl into the capsid resulting in FMDV having the highest buoyant density of the picornaviruses (Jackson *et al.,* 2002).

The main cell attachment site and the immunodominant region of FMDV are both located on a solvent exposed region at the surface of the virion, namely, in trypsin-sensitive areas of VP1 (Bush, 1991). Earlier serological studies showed that different serotypes of FMDV shared a highly variable region of VP1, comprising residues 135 to 155 as one of the major antigenic sites of the virus (Strohmaier *et al.,* 1982).

2.1.3.3 Epidemiology of foot and mouth disease

The transmission dynamic of FMD in sub-Saharan Africa is mainly driven by two epidemiological cycles: one in which wildlife plays a significant role in maintaining and spreading the disease to other susceptible wild and/or domestic ruminants (Dawe *et al.,* 1994; Bastos *et al.,* 2000). Whilst with the second the virus is solely transmitted within

domestic populations and hence is independent of wildlife (Miguel *et al.,* 2013). FMD is a highly transmissible disease, and a limited number of infective particles can initiate host infection (Herold and Andino, 2001). Contaminated animal products, non-susceptible animals, agricultural tools, people, vehicles, and airborne transmission contribute to the mechanical dissemination of FMDV (Donaldson *et al.,* 1987). The epidemiology of FMD is complex, and it is affected by different viral, host, and environmental factors, among them, variations in virus virulence (severity of lesions, amount, and duration of virus release), particle stability in different microenvironments, and chances of long-term persistence. FMDV multiplication and spread also depend on the host species, nutritional and immunological status, population density, animal movements, and contacts between different domestic and wild host species and animals capable of mechanical dissemination of the virus (Nishiura and Omori, 2010). The environment can provide geographical barriers to virus dissemination or, alternatively, can promote virus transmission when appropriate atmospheric conditions prevail in this multifactorial scenario (Ferguson *et al.,* 2001; Sobrino and Domingo, 2001). The high potential for FMDV variation and adaptation has modelled complex evolutionary patterns that are being revealed by molecular epidemiology analyses, mostly based on nucleotide sequencing of capsid protein genes.

The agroclimatic and socioeconomic condition, mixed animal husbandry practices, unrestricted movement, and trade among animals and porous international border provide a conducive epidemiology niche for the FMDV to flourish, mutate, and persist over time and to affect the susceptible animal population (Rao, 1989; Mishra *et al.,* 1998). Losses from FMD are mainly due to reduction in milk yield, draught power, and breeding capabilities. In India where world's largest livestock populations exist, it is a

leading cause of loss of livestock economy (direct and indirect losses) due to its endemic nature (Longjam *et al.*, 2011).

2.1.3.4 History of foot and mouth disease in Nigeria

The earliest recorded history of FMD in Nigeria dated back to 1924 when the colonial government was setting up the Nigeria's embryonic veterinary service (Olabode *et al.*, 2013). Before that period, FMD probably existed in Nigeria but was not documented. FMD was detected and diagnosed clinically among trade cattle in Borno Province in 1929 (Olabode *et al.*, 2013). Subsequently, the disease spread widely throughout the country amongst trade cattle along several trade routes in Northern-Nigeria with little or no control measure being instituted (Olabode *et al.*, 2013). This resulted in sporadic outbreaks in young stocks especially during the dry season in the forties, (1946, 1947 and 1948) (Abegunde, 1987). In 1957, outbreak reports revealed serotype O was introduced by trade cattle from highland of Mambilla Plateau from the Cameroon spreading the disease to the Benue province (Olabode *et al.*, 2013). In 1960, the disease maintained mild epizootic status in the North-Eastern part of the Country amongst trade cattle spreading along cattle routes with serotype A causing the outbreaks and the introduction of SAT 2 from animals from Chad which was subsequently isolated from severe outbreaks in the region. Review of FMD situation in Africa reported outbreaks in Northern Nigeria and none in the Eastern and Western parts of the country (Libeau, 1960). During the early-mid sixties, as the collection and dispatch of field samples to World Reference Laboratory, Pirbright improved, outbreak viral isolation indicated serotype A in Adamawa Province, serotype O in Bauchi Province, SAT1 in Bauchi, Borno, and Zaria Provinces while SAT2 was isolated in Zaria Province (Owolodun, 1971; Nawathe and Goni, 1976; Asagba, 1982). These reviews tend to indicate that

FMD virus distribution and outbreak occurrence was limited to the Northern states of the country amongst trade cattle moving down south. However, in the early-70's and 80's investigations revealed outbreaks in the Western and South-Eastern Nigeria with Serotype A and SAT 2 documented to be active in the outbreaks (Nawathe and Goni, 1976; Durojaiye, 1981). The current trend of the disease occurrence is unknown but alarming, as there are regular outbreaks, no control measures, poor enforcement of legislation guiding disease reporting to veterinary authority as well as poor control of animal movements (Chuckwuedu *et al*., 2008; Lazarus *et al*., 2015). Cattle are most susceptible to infection by FMD followed by sheep while goats remain the least (Lazarus *et al*., 2015), however, pigs are equally highly susceptible (Kitching and Alexandersen, 2002). The semi-intensive system of animal management in developing countries encourages the spread of the disease (Williams, 2003). Questionnaire update in Nigeria confirmed serotype O, SAT1 and SAT2 while serological survey using Virus infection antigens (VIA) and 146S whole virion antigen and indirect ELISA revealed overall incidence and prevalence of 9.8% and 55% respectively (Abegunde, 1987). Update status of FMD by Nwanta and Ojemiren (1999), indicated serotypes O, A, SAT 1 and SAT 2 have been responsible for disease outbreaks in Northern Nigeria. Chukwuedo *et al.* (2008) demonstrated antibodies to SAT1 and SAT 2 serotypes, while Knowles *et al.* (2008) reported serotype O and SAT2 from outbreaks. Recent review confirmed serotypes, O, A, SAT 1 and SAT 2 have been circulating in the country in the last 54 years (1955-2009) (Olabode *et al.,* 2013). Serological surveys conducted between 2009 and 2011 in six Border States and two other states that host the major cattle trek routes showed increasing sero-prevalence in cattle sampled from Jos south LGA in Plateau state (56.3%) Ishola *et al.* (2011), Yobe State (82%), Plateau (80%), Ogun (77.77%), Taraba (73.50%), Adamawa (68%), Borno (67%), Sokoto (63%) cattle

and Bauchi 27.84% for only sheep and goat (Lazarus *et al*., 2012) as well as Kwara (75.11%) (Olabode *et al.*, 2013) as these reports confirmed increased disease endemicity with outbreaks been experienced annually due to absence of cross-immunity and heterogeneity amongst serotypes (Abdulkadir, 1989). In Nigeria, livestock population are from both within the country and neighbouring countries of west and central Africa, the animals are at high risk of infection of endemic strains as well as antigenic variants prevalent in neighbouring countries (Fakai *et al*., 2015). Pigs play a major role in the spread of FMD through their ability to emit airborne FMD virus (FMDV) and cattle are highly susceptible to infection by inhalation (Alexandersen and Donaldson, 2012; Fakai *et al.,* 2015). Once an animal is infected, the virus can be disseminated into the environment including pastures, water sources, and soil (Fakai *et al*., 2015). In spite of the annual disease burden, sero-typing and molecular studies for FMD infections are inadequate due to poor understanding of the epidemiology of FMD in Nigeria. Hence, occurrence and distribution pattern of the known serotypes still remains unexploited and poorly understood.

2.1.3.5 Host distribution of foot and mouth disease

Foot and mouth disease (FMD) is an acute systemic infection affecting even-toed ungulates, both domesticated and wild, including cattle, swine, sheep, and goats (Longjam *et al.,* 2011; Miguel *et al.,* 2013; Ehizibolo *et al.,* 2016). More than 70 wild animal species have been demonstrated to be susceptible to the FMD virus (FMDV) either by natural infection or by experimental challenge, and on several occasions the virus has been isolated from naturally infected animals (Weaver *et al.,* 2013). Some wildlife species affected with FMD includes elephant (Pyakural *et al.,* 1976; Dutta *et*

al., 1979; Verma and Sarma, 1997) yak, sambar, spotted deer, and wild buffalo (Barman *et al.,* 1999; Vosloo *et al.,* 2002).

2.1.3.6 Clinical signs of foot and mouth disease in animals

Foot and mouth disease virus (FMDV) produces an acute, systemic vesicular disease, which requires a differential diagnosis from other vesicular diseases blue tongue and mucosal disease (Sutmoller, 1992). In natural infection, the main route of virus entry is the respiratory tract. The initial virus multiplication usually takes place in the pharynx epithelium, producing primary vesicles, or "aphthae" (Burrows *et al.,* 1981). The vesicles produced by FMDV generally affect cells from the epithelial stratum espinosum. The clinical outcome of the disease may vary among the host species considered and the infecting virus strain. In cattle and pigs, fever and viraemia usually start within 24–48 h after epithelium infection, leading to viral spread into different organs and tissues and the production of secondary vesicles preferentially in the mouth and feet. The acute phase of disease lasts about 1 week and declines gradually coinciding with the emergence of a strong humoral response (Salt, 1993). The morbidity and mortality in FMD depends upon the breed and age of the animal where mortality in adult animals is very low (2 %) in comparison to 20 % in young stock (Knowles *et al.,* 2001). The calves show prominent signs of myocarditis, whereas piglets manifest gastroenteritis (Knowles *et al.,* 2001). In sheep and goats, symptoms are frequently less severe and may make the detection of the disease difficult (Knowles *et al.,* 2001). Cattle are also key hosts in the dynamics of FMD in domestic ungulate populations. Viruses can usually be isolated up to 28 days after infection (Charleston *et al.,* 2011) and the carrier state can last up to 3.5 years (Grubman and Baxt, 2004). The virus is highly

contagious in cattle populations and can be transmitted via direct contact between individuals, but also via environmental contamination (Backer *et al.,* 2012).

Asymptomatic, persistent infection can also be established in ruminants, during which infectious virus can be isolated from the oesophagus and throat fluids of the animals from a few weeks up to several years of the initial infection. There is epidemiological evidence to support the hypothesis that carrier animals may be the origin of outbreaks of acute disease when brought into contact with susceptible animals. This mode of transmission has been experimentally reproduced for serotype SAT isolates (Vosloo *et al.,* 2007).

2.1.3.7 Diagnosis of foot and mouth disease

The accurate diagnosis of infection with FMDV is of prime most importance for both control and eradication campaigns in FMD endemic areas and as a supportive measure to the stamping out policy in FMD-free areas (Rémond *et al.,* 2002). Early work by Hecke and the Maitlands in the early 1930s, followed by the crucial demonstration by Frenkel in 1947 that large amounts of the virus could be produced in surviving tongue epithelium, formed the basis for the vaccination programmes initiated in Europe in the 1950s (Brown, 2003). The subsequent development of cell lines has brought a remarkable degree of sophistication to the study of virus growth. The recognition of more than one serotype has led to the development of various techniques for serotyping of the virus.Two isolates of a serotype "A" virus could be differentiated by cross-neutralization tests (Rweyemamu, 1982). Earlier typing of FMDV was done by cross-immunity test in guinea pigs and less frequently in cattle (Longjam *et al.*, 2011). As this test was time consuming, expensive, and imprecise, different serological tests like

complement fixation test (CFT), virus neutralization test (VNT), and enzyme-linked immunosorbent assay (ELISA) were developed and the most recent is the development of molecular techniques, the polymerase chain reaction (PCR) method making diagnosis more rapid and precise. Longjam *et al.,* (2011) demonstrated that multiplex PCR could detect FMD virus in the highest number of samples (65.47 %) followed by sandwich ELISA (53.57 %) and virus isolation (42.85 %).

2.1.3.8 Control of foot and mouth disease

Current FMD control measures in southern Africa strongly rely on vaccination of livestock populations at risk of spill-over infection from wildlife populations, especially from buffalo (Miguel *et al.,* 2013). This prophylactic strategy at the wildlife livestock interface based on vaccination however, suffer from several technical and practical limitations. First, the current vaccine used in southern African region where FMD is endemic and not provide protection against strains that circulate in the buffalo populations. This would require frequent isolations of strains from wild and cattle populations, to adapt local vaccines, a strategy not currently implemented. Second, the immunity provided to vaccinated cattle only lasts for 3–6 months (Miguel *et al.,* 2013) and this may require increase in frequency of vaccination campaign. Developing countries such as Zimbabwe, which also faced severe economic difficulties during the past decade, lack the appropriate financial resources to purchase a sufficient number of vaccine doses to immunize a sufficient proportion of the cattle population at risk of infection from buffalo reservoirs to prevent outbreaks (Keeling *et al.,* 2003). Thus, the strategy of ‘‘ring vaccination’’ was adopted to contain outbreaks so that they do not spread to other areas of the country. Improving surveillance strategies, for instance through the use of NSP serological diagnostic on randomly selected animals, in all risky

areas (i.e., close to reservoir population) and during risky periods (i.e., dry season) could be an important step towards FMD control (Miguel *et al.,* 2013). The control of the transmission of FMD at the wildlife-livestock interface has only been successfully applied in South Africa at a considerable ecological and economic cost (Ferguson *et al*., 2013; Kock *et al*., 2014) by the means of herds separation (e.g. strict land-use policies, animal movement controls and fencing) and buffer vaccination of livestock population around the source of virus (Thomson, 1995). However, in countries where wildlife populations are integrated with extensive nomadic and semi-nomadic pastoralism the risk of FMD spread increases not only for the difficulty in applying effective control measures and movement of animals in an uncontrolled fashion, but also for the increase in the land-use pressure and conflict between pastoralists and wildlife competing for grazing and water resources (Sutmoller *et al*., 2000; Di Nardo *et al*., 2015).

2.1.4 Bovine viral diarrhoea

2.1.4.1 Aetiology of bovine viral diarrhoea

Bovine Viral Diarrhoea is a viral disease, which, although primarily affects cows, can also affect other ruminants (sheep, goats, wild ruminants). Animals affected show an array of clinical signs, however, with a majority of infected animals do not showing any clinical signs at all (Bovine viral diarrhea (BVD) is responsible for massive economic losses in livestock farming (Vanroose *et al.,* 1999). It is caused by the BVD virus (BVDV), an RNA-virus that belongs to the genus Pestivirus of the family *Flaviviridae*. This virus family also comprises the pathogens causing classical swine fever and border disease. BVDV is transmitted mainly horizontally from animal to animal via oral and nasopharyngeal secretions, or iatrogenically (rectal examinations, injections with contaminated needles, contaminated live vaccines (Couvillion *et al.,* 1980). An acute

infection usually remains inapparent or causes only mild disease and establishes a robust immunity. The importance of BVDV is due to the occurrence of persistently infected (PI) animals, resulting from infections of pregnant cows or heifers during gestation prior to the development of the fetus' immune system. In such a case, the virus is recognized as "self", and an animal born alive will spread BVDV lifelong. Persistently infected cattle may develop the fatal mucosal disease (Elazhary *et al.*, 1981). The isolation of BVDV and the occurrence of PI animals have been documented in various free-ranging and captive species (Fischer *et al.*, 1998) but the possible role of wild ruminants as reservoir remains unclear.

2.1.4.2 Clinical signs of bovine viral diarrhoea in animals

Infections with Bovine Viral Diarrhoea Virus (BVDV) are not limited to cattle, but may be detected in various species in the mammalian order Artiodactyla (Passler and Walz, 2010). The clinical picture is generally one of high morbidity and low mortality, though more severe disease is sometimes seen (Brownlie, 1990). Vertical transmission plays an important role in the epidemiology and pathogenesis of the disease (Bolin, 1990). Animals infected with BVDV may develop multiple and diverse clinical manifestations. The wide spectrum of disease associated with BVDV infection includes sub-clinical infections, BVD, congenital deformities in calves and acute and chronic mucosal disease (Blood and Radostits, 1989; Dehkordi, 2011). The majority of infections (70%-90%) in susceptible cattle are sub-clinical with the development of neutralising antibodies (Ames, 1986). However, infection with BVD is acute in seronegative, immunocompetent cattle (Dehkordi, 2011). Clinical signs may include pyrexia, depression, inappetence, oculonasal discharge and occasionally oral lesions characterised by erosions or shallow ulcerations and foul smelling watery diarrhoea;

high morbidity is experienced but mortality is low (Blood and Radostits, 1989; Dehkordi, 2011). The virus causes immunosuppression in the host and may potentiate or enhance the pathogenicity of co-infecting micro-organisms (e.g. infectious bovine rhino tracheitis, parainfluenza type-3, corona and rotaviruses, and *Pasteurella* and *Salmonella* spp.) (Dehkordi, 2011).

2.1.4.3 Epidemiology of bovine viral diarrhoea

The disease has a worldwide distribution and cattle and wild ruminants of all ages are susceptible (Houe, 1995; Passler and Walz, 2010). Various works have so far been carried on the prevalence of this virus in some parts of Nigeria (Baba *et al*., 1994; Bello *et al*., 2016) and Cameroons (Handel *et al.,* 2011) and the world at large (Soiné *et al.,* 1992; Paton *et al.,* 1998; Ferreira *et al.,* 2000; Falcone *et al.,* 2001; Viltrop *et al.,* 2002; Scott *et al.,* 2013). In North America, when positive titers are observed, antibody prevalence ranges from 4% in pronghorn (*Antilocapraamericana*) of Alberta, Canada to 70% in caribou (*Rangifertarandus*) of Quebec, Canada (Elazhary *et al.,* 1981). Other species with evidence of exposure include mule deer (*Odocoileushemionus*), elk (*Cervuselaphus*), moose (*Alcesalces*) (Aguirre *et al.,* 1995) and bison (*Bison bison*) (Thorsen and Henderson, 1971). The virus has been isolated from mule deer and white-tailed deer (Couvillion *et al.,* 1980). Several pestiviruses distinct from BVDV have been isolated from wildlife, including a pestivirus isolated from pronghorn antelope in Wyoming that was classified as a new pestivirus genotype (Vilcek *et al.,* 2005) Experimental infection of mule deer and white-tailed deer with BVDV revealed that they are susceptible to infection and can shed virus following inoculation without showing signs of clinical disease (Vilcek *et al.,* 2005). Likewise, infected elk showed no clinical signs, but all animals became infected and transmission to noninoculated, in-

contact animals was observed (Campen *et al.*, 1997). The occurrence of interactions between wild and domestic species has also been documented in other countries (Thorsen and Henderson, 1971; Campen *et al.,* 1997) and more attention has been directed toward the potential role of wildlife in the epidemiology of diseases that may be shared between wild and domestic species (Campen *et al.,* 1997). However, while the occurrence of such interactions is often reported in scientific articles, the information remains vague or not properly documented and there is a need for more systematically collected data.

2.1.4.4 Diagnosis and control of bovine viral diarrrhoea

Identification of persistently infected calves, in combination with a proper vaccination program, is essential for the successful control of BVDV (Ames and Baker, 1990). Various virus detection methods include isolation by cell culture, immunoassay, electron microscopy (EM), nucleic acid hybridization, and reverse transcription PCR. (RT-PCR) (Hamel *et al.,* 1995). Standard BVDV detection protocols, which use the combination of cell culture and fluorescein-labelled immunoassay, are hampered by the problem of false-positive results, because BVDV is a widespread contaminant of fetal calf serum (FCS) and cell culture (Bolin, 1990). In addition, cell culture assays are labor-intensive and lengthy, usually requiring several days for completion. These problems prevent many laboratories from simultaneously processing large numbers of samples. RT-PCR is becoming an increasingly popular procedure for detecting BVDV and other pestiviruses (Hamel *et al.,* 1995). Two limiting factors that govern the successful performance of RT-PCR are the dependability of the nucleic acid purification methods and the oligonucleotide-specific priming of the RT reaction. Other important

considerations include the thermocycling parameters and reagents used in the PCR (Hamel *et al.,* 1995).

2.1.5 African horse sickness

2.1.5.1 Aetiology of African horse sickness

African horse sickness (AHS) is a serious, often fatal, arthropod-borne (*Cuilicoides*) viral disease of horses and mules. The mortality rate can be as high as 95% in some forms of this disease (CFSPH, 2006). Asymptomatic or mild infections can occur in horses previously infected with a different serotype of the virus, as well as zebras and donkeys. Infected animals or vectors may carry the virus into AHS-free regions (Fasina *et al.,* 2008). Some authors speculate that global warming could increase the risk for spread of arthropod-borne diseases such as African horse sickness. *Culicoides imicola,* the principal vector for this disease, has made incursions into North Africa, southern Europe and the United States from east Africa (CFSPH, 2006).

African horse sickness virus (AHSV) is a member of the genus *Orbivirus* in the family Reoviridae. There are nine serotypes of AHSV. Serotype 9 is widespread in (sub-Saharan Africa) endemic regions, AHSVserotype 9 spread out of Africa to Saudi Arabia, Syria, Lebanon, Jordan, Iraq, Turkey, while serotypes 1 to 8 are found only in limited geographic areas of su-Saharan Africa (Mellor and Hamblin 2004). Serotype 9 has been responsible for the majority of African horse sickness outbreaks outside Africa. Serotype 4 caused one outbreak in Spain and Portugal between 1987 and 1990 (CFSPH, 2006). Fasina *et al.* (2008) reported the first outbreak of AHS caused by AHSV serotype 2 from Nigeria.

2.1.5.2 Host range for African horse sickness

The African horse sickness virus can infect horses, donkeys, mules, zebras, camels and dogs (Radostitis *et al.,* 2000). The most serious infections occur in horses and mules, which appear to be accidental hosts (Radostitis *et al.,* 2000). AHS virus is well adapted to Zebra, which is considered the natural host and also the main reservoir of the virus in Africa (Boinas *et al.,* 2009; Wilson *et al*., 2009). Other equine species and their crossbreeds are susceptible to AHSV infection, and with the exception of donkey, usually show clinical disease and high mortality. Other known susceptible species are dogs, wild carnivores (hyenas and jackals), elephants and camels (Mellor and Hamblin, 2004). Dogs get infection probably by the oral route, by consumption of meat from infected animals that died from the disease (Boinas *et al.,* 2009). However, the role of dogs in the epidemiology of AHS is considered to be irrelevant, as vectors do not appear to be attracted to them (Braverman and Chizov-Ginzburg, 1996). Serological surveys indicate that natural AHS infection is widespread among a diversity of African wild carnivore species (Alexander *et al.,* 1995). Similarly, to dog, it is believed that infection results from ingestion of meat and organs from AHS-infected prey species. The effect of AHSV on these species is largely unknown, as is their role in the maintenance cycle of the disease (Alexander *et al.,* 1995). Although as much as 10 of 104 (10.4%) camels (Hyalommadromedarii) were seropositive in a survey for AHS in Nigeria (Baba *et al.,* 1994), infection in camels is considered unapparent and rare. Level and duration of viraemia are unknown in this species (Boinas *et al.,* 2009). Altogether makes unlikely that camels play any role in the epidemiology of AHS and they are regarded as poorly susceptible and unlikely to be a reservoir of AHSV (Barnard *et al.,* 1994). In addition, antibodies have been found in sheep, goats and buffaloes in an Egyptian survey (Khan and Zafar, 2005).

2.1.5.3 Transmission of African horse sickness

African horse sickness is transmitted by midges in the genus *Culicoides*, and is not contagious by casual contact. Both *Culicoides imicola* and *C. bolitinos* are known to transmit AHSV in the field; *C. imicola* appears to be the principal vector (Wilson *et al.*, 2009). Other species of *Culicoides* may also be able to spread this virus, The North American species *C. variipennis*is an efficient vector in the laboratory (CFSPH, 2006). Wind has been implicated in the dispersal of infected *Culicoides* vectors during some epidemics. Transmission by insects other than midges is thought to be a minor source of infection; however, mosquitoes have been implicated as biological vectors, and biting flies in the genera *Stomoxys* and *Tabanus* may be able to transmit the virus mechanically (Mellor and Hamblin, 2004) It has been indicated that Culicoides have a widely varied distribution and biologic patterns (Mellor *et al.*, 2000; Paweska *et al.*, 2003; Mellor and Hamblin, 2004) and field observations in the different agro-ecological zones of Nigeria confirmed these reports (Fasina *et al*., 2008). The arthropods implicated in transmission of AHSV have been known to thrive well in damp and warm soil or bedding (Mellor and Hamblin, 2004). Previous studies conducted by Mellor *et al*. (2000) concluded that these vectors blood-feed more frequently and have increased virogenesis and consequently enhanced probability of transmission at warmer temperatures (25 □C and above) and relative humidity of 80% to 90%. Dogs can be infected experimentally, or if they eat contaminated horse meat (Khan and Zafar, 2005). Epidemics of African horse sickness tend to occur at cyclic intervals, and are associated with drought followed by heavy rain (Harvell *et al.*, 2002). Although zebras are thought to be the natural reservoir hosts, horses, mules and donkeys can also develop viremia sufficient to infect *Culicoides*. Most sources suggest that dogs

do not play a significant role in the maintenance or spread of AHSV (Kahn and Line, 2003). In experimental infections, the incubation period can range from 2 to 21 days. In natural infections, the incubation period appears to be approximately 3 to 5 days for the pulmonary form, 7 to 14 days for the cardiac form, 5 to 7 days for the mixed form and 5 to 14 days for horse sickness fever (Kahn and Line, 2003).

2.1.5.3 Epidemiology of African horse sickness

African horse sickness was confined to the African continent until the second world war when serious out breaks occurred in the middle east (Blood and Radostits, 1989) the disease came into prominence again in 1959 when it spread to Iran and Pakistan including Turkey to the eastern Mediterranean and Cyprus (Blood and Henderson, 1979). The disease continues as enzootic disease in Africa recurring annually in most of southern, Equatorial and Eastern Africa (Mellor *et al.,* 2000). The disease has been known to exist in Senegal and Sudan since the 19th century (Diouf *et al.,* 2013). The disease especially during rainy season was at that time responsible for reducing the strength of horses in the expeditionary corps (Diouf *et al.,* 2013). Several authors reporting in that era described the disease and identified it with a variety of names which include typhoid fever, pernicious fever and typhomalaria (Diouf *et al.,* 2013).

The epizootiology of AHS in Nigeria has been described by many workers (Baba *et al.,* 1994; Kazeem *et al*., 2008; Fasina *et al.,* 2008; Ehizibolo *et al*., 2014). Oladosu *et al.* (1993) isolated and identified AHSV using complement fixation test during an outbreak involving two horse stables in Lagos, Adeyefa and Hamblin (1994) reported on the continual prevalence of AHS in Nigeria when they tested for the presence of AHSV antibodies in horses and donkeys using Competitive ELISA technique, 79.8% of the

sera were positive confirming the continued prevalence of AHSV antibodies in Nigerian horses and donkeys. The presence of antibodies against the African horse sickness virus was investigated in 246 domestic animals (horses, donkeys, camels, dogs) in various regions of Nigeria by means of the complement-fixing rate (Baba *et al.,* 1994). About 34% of the sera tested were positive. Fasina *et al*. (2008) reported AHSV outbreaks among polo horses in Lagos, which spread to Kano, Kaduna, Yola, Katsina, and Ibadan, Nigeria and involved a large number of horses characterized by severe respiratory distress, fever, supraorbital edema, and death. It was eventually confirmed to be AHSV serotype 2 based on serology and PCR results from reference Laboratory in South Africa.

2.1.5.5 Pathogenesis of African horse sickness

Despite the distinct differences in the clinical severity of AHS infection in different equids, the typical pattern of pathogenesis is similar (Thompson *et al.*, 2012). Following exposure through the bite of an infected vector, the virus initially replicates in an adjacent lymph node before disseminating throughout the entire body via the circulatory system. This is the primary viraemia which leads to infection of the lungs and lymphoid tissues (Thompson *et al.*, 2012). The virus then undergoes a further replication cycle in these target organs which in turn gives rise to a secondary viraemia (Thompson *et al.*, 2012). The virus has a predilection for the vascular endothelial cells throughout the body, causing extensive damage including effusions into body cavities and tissues, and widespread haemorrhages (Coetzer *et al.,* 1994). The duration of this process from initial infection to secondary viraemia can vary between 2–21 days, although it usually takes less than nine (Mellor and Hamblin, 2004). In horses, the period of viraemia usually lasts between 4–8 days, whereas in donkeys, it may persist for up to four weeks. In zebra, this period may be extended to approximately 40 days' post-infection (Wilson

et al., 2009). In common with BTV, AHSV is often associated with red blood cells of the infected animal throughout both primary and secondary viraemia. In ruminants infected with BTV, this often leads to an extended period of viraemia, although this does not appear to be the case with AHS (Mellor and Hamblin, 2004).

2.1.5.6 Clinical signs of African horse sickness

African Horse Sickness displays itself in the form of four diseases: cardiac form, pulmonary form, mixed form, and horse sickness fever (Thompson *et al.,* 2012). The cardiac form of the disease is usually characterised by the development of a fever, oedema of the head, neck, chest, supraorbital fossae, petechial haemorrhages in the eyes, ecchymotic haemorrhages on the tongue, and colic. In these cases, mortality of infected animals may exceed 50% (Coetzer *et al.,* 1994). The pulmonary form of the disease is the most serious. It is typically associated with a rapid onset of symptoms which include the development of a fever, depression, severe respiratory distress, severe dyspnoea, coughing, and sweating. Typically, the mortality rates of this form of the disease exceed 95% (Coetzer *et al.,* 1994). The mixed form is often the most common form of the disease, it is a combination of the cardiac and the pulmonary form. Typically, the mortality rate may exceed 70%, and death can often occur within three to six days (Coetzer *et al.,* 1994). Horse sickness fever is the mildest form of the disease, during which, the animal will develop a moderate fever and some oedema of the supraorbital fossae (Boinas *et al.,* 2009). There is no mortality associated with this form of the disease (Coetzer *et al.,* 1994).

2.1.5.7 Gross lesions of African horse sickness in animals

Gross lesions at necropsy depend on the clinical form of the disease. In the pulmonary form, alveolar and interstitial oedema of the lungs and hydrothorax are the main findings. Oedema around the loose connective tissue of the trachea and of the aorta, and hyperaemia of the fundic portion of the stomach are also frequently observed. Petechial haemorrhages in the peritoneal, pleural and pericardial serosa may be seen, as well as hydropericardium. Many horses show frothy whitish or pink fluid arousing from the nares. In the cardiac form, oedematous infiltration of the head and neck musculature is the most prominent lesions, together with subcutaneous oedema of the head. Hydropericardium is consistently present, as well as haemorrhages in epicardium and endocardium. Pale areas of myocardial degeneration have been described. Lung oedema may be also present. In the mixed form, a combination of these lesions occurs. In fact, most of the fatal cases of AHS may be considered mixed forms, with lesions of the cardiac or the pulmonary form predominating (Coetzer *et al.*, 1994). Fatal cases in the dog usually resemble the pulmonary form of horses, with congestion and oedema of the lungs as main findings (Boinas *et al.*, 2009). Oedema in subcutaneous and intermuscular tissue and pulmonary oedema are the most striking findings in AHS. Haemorrhages are also a frequent finding in pleura, pericardium, and endocardium

2.1.5.8 Prevention and control measures of African horse sickness

Control and eradication of AHS has been achieved, so far, in Portugal, Morocco and Spain (Portas *et al.*, 1999; Sanchez-Vizcaino, 2003). The key to the success in the control of AHS in Spain was the use of a polyvalent attenuated vaccine produced by Ondersteport Veterinary Research Institute South Africa and attenuated monovalent type 4 vaccines from the same source, together with the establishment of protection

areas, control of animal movements, slaughter of affected animals, control of Culicoides and serological surveillance. Since AHS vaccines are sparse, a bank of vaccines is required in order to provide the required vaccination in areas in need (Boinas *et al.*, 2009). These measures should be accompanied by epidemiological studies, risk analysis of the spread and introduction of the disease in disease-free areas and follow-up studies on the efficacy of the control measures taken. Previous experience acquired in past outbreaks (like the one that took place in Spain from 1987 to 1991) should be taken into account for the design of control strategies in other countries (Boinas *et al.*, 2009).

2.2 Parasitic Infections in Wildlife and Domestic Animals

2.2.1. Role of wildlife in the epidemiology of haemoparasites

Parasitic infections transmitted between livestock and wildlife pose a significant risk to wildlife conservation efforts and constrain livestock productivity in tropical regions of the world (VanderWaal *et al.*, 2014). The emergence of infectious diseases with zoonotic potential has dominated investigations and commentary on wildlife pathogens (McCallum and Dobson, 1995; Holmes, 1996; Daszak *et al.*, 2000; Rhyan and Spraker, 2010). In terms of conservation, it is unfortunate that by doing so, not only have studies on the biodiversity and ecology of wildlife parasites been neglected, but control efforts have also been hampered (Thompson *et al.*, 2010). In Africa, biodiversity conservation and the expansion of livestock production have increased the risk of transmitting vector-borne infections between wildlife and livestock (Van Vuuren and Penzhorn, 2015).

There is abundant evidence of haemoparsitic infections in wildlife worldwide, in some circumstances displaying high prevalence with some of them serving as reservoirs for the haemoparasites (Alvarado-Rybak *et al*., 2016). There are wide ranges of potential

vectors that may allow these parasites to maintain endemic sylvatic life-cycles in their geographical distribution area (Shock *et al.,* 2011; Najm *et al.*, 2014; Alvarado-Rybak *et al.,* 2016). This could potentially lead to the transmission of infection to domestic species, especially in peri-urban and urban environments (Alvarado-Rybak *et al.,* 2016). Many wild reservoir hosts (e.g. red fox, golden jackal and raccoon, elephants, and waterbuck) are increasing in number and expanding their geographical ranges, thus increasing intra- and interspecies contact risk with domestic animals (Duscher *et al.,* 2014). However, a high prevalence of infection alone does not demonstrate that the species in question acts as a reservoir, some wildlife are not abundant, and probably unable to maintain a pathogen in the absence of domestic species reservoirs (Alvarado-Rybak *et al*., 2016).

2.2.2 Role of livestock in the epidemiology of haemoparasites

Haemoparasitic infections have a global distribution, stretching from the polar circle to the equator (Jonsson, 2006). This is due to the fact that their vectors; ticks and blood-sucking flies, also have a global distribution (Okorafor and Nzeako, 2014). The incidence of haemoparasitic infections in livestock have been reported by some workers (Bell-Sakyi *et al.,* 2004; Jonsson, 2006; Kamani *et al.,* 2010). Haemoparasitic infections in livestock in Africa are complex, with many ticks and tsetse fly species interacting with different hosts and transmitting a wide range of pathogenic and non-pathogenic organisms (Nwoha *et al.,* 2013). Haemoparasitic infections in livestock species cause substantial economic losses and the improvement of strategies to control the diseases in livestock requires more detailed knowledge of their prevalence and how they interact with each other (Uilenberg, 1995). Haemoparasites have generally shown to cause destruction of red blood cells resulting in anaemia, jaundice, anorexia, weight loss and

infertility (Jonsson, 2006). Some hemo- parasites species are only evident when the host is undergoing a clinical response to infection, while others may be easily seen in blood smears from apparently healthy animals (Kamani *et al.,* 2010). Trypanosomosis, babesiosis, heartwater, theleriosis and anaplasmosis are important haemoparasites of livestock in tropical countries (Bell-Sakyi *et al.,* 2004; Lako *et al.,* 2007; Kamani *et al.,* 2010). Particularly, trypanosomosis have remained a major concern in livestock production in Nigeria despite several therapeutic and control attempts (Onyiah, 1997; Abenga *et al.,* 2004; Samdi *et al.,* 2010). Prevalence rate of the disease in livestock are variable depending on the distribution of the vector (tse tse fly) in endemic areas (Nwoha *et al.,* 2013). The threat of African animal trypanosomosis on sustainable livestock production is compounded by failure of vector control as well as chemotherapy and chemoprophylaxis (Abenga *et al.,* 2004; Samdi *et al.,* 2010). These constitute a major threat to attaining food security in several parts of subsaharan Africa and Nigeria (Samdi *et al.,* 2010).

In some studies conducted on haemoparasites of cattle in Nigeria, Agu and Amadi (2001) reported a prevalence of 3.9% in Ebonyi State, Enwezor *et al.* (2009) in Kaduna State reported a prevalence rate of 13.5% and Agu and Amadi (2001) in a study also in Kaduna State reported a prevalence rate of 9.4%. In a study investigated among 637 cattle by Kamani *et al.* (2010) for haemoparasitic infections in North-Central, Nigeria, 25.7% prevalence rate was recorded. Okorafor and Nzeako (2014) reported a prevalence rate of 6.6% from 188 cattle sampled from three abattoirs (Bodija, Akinyele and Olorunsogo) in Ibadan, Oyo State, Nigeria.

Haemoparasites have severe and debilitating effects in the health and reproductive success of horses and donkeys (Swallow, 2000 Francisco *et al.,* 2009; Pam *et al.,* 2013). Many factors like management, climate, and parasite control program can influence the prevalence of parasites of domestic animals as infection rates can be associated with lack of immunity or lack of good management practice (Francisco *et al.,* 2009).

2.2.3 Life cycle of arthropod borne parasites

Piroplasmoses are among the most prevalent arthropod borne diseases of animals caused by haemoprotozoan parasites (Alvarado-Rybak *et al*., 2016). These parasites have a great economic, veterinary and medical impact worldwide. In fact, they are considered to be the second most commonly found parasites in the blood of mammals after trypanosomes (Schnittger *et al.,* 2012). They are frequently found infecting free-living animals worldwide (Alvarado-Rybak *et al*., 2016). Although piroplasmoses are among the most relevant diseases of wild and domestic animals (Yabsley and Shock, 2013; Alvarado-Rybak *et al.* 2016), many questions remain unsolved concerning their epidemiology and life-cycles. These include questions regarding their phases in the ixodid tick vector as well as the vertebrate host, especially with regard to wildlife (Zanet *et al.,* 2014). It is known that piroplasmids are maintained in a complex system of vectors and animal reservoirs, and infection of the mammalian host often takes place via the bite of the invertebrate vector, usually ticks (Mehlhorn and Schein, 1985; Chauvin *et al.,* 2009). While the tick is feeding, sporozoites are released from its salivary glands and enter the blood stream of the vertebrate host (Penzhorn, 2006). Parasites then attach to and are endocytosed by erythrocytes (*Babesia* spp.), or initially penetrate into lymphocytes or other leukocytes (*Theileria* spp.), or macrophages, histiocytes, reticuloendothelial cells and or endothelial cells (*Cytauxzoon* spp.) (Eiras *et al.,* 2014;

Mans *et al.,* 2015). This is followed by an intra erythrocytic cycle or intraleukocyte cycle, e.g. in some *Theileria* spp. (Eiras *et al.*, 2014). Once parasites are in the erythrocytes or leukocytes, they undergo asexual reproduction and merogony, and the daughter cells can infect new cells. A naïve tick then ingests infected erythrocytes. It is unclear whether the transformation from merozoite to gamete (gametocyte) begins in the vertebrate host or in the tick, however, in the tick midgut, the sexual phase of reproduction occurs when the gametes fuse to form a zygote (Soares *et al.,* 2014). The zygote invades the epithelial cell of the tick gut, and an asexual form of reproduction, sporogony, occurs. The resultant forms, ookinetes, leave the epithelial cell and invade either the salivary gland or the ovary of the tick, where they participate in transstadial and transovarial transmissions (Mehlhorn and Schein, 1985; Soares *et al.,* 2014; Alvarado-Rybak *et al.,* 2016).

2.2.4 Mode of transmission of arthropod borne parasites

Tick bites appear to be the primary manner of transmission for haemoparasites (Alvarado-Rybak *et al.*, 2016). However, other forms of transmission have been described for some piroplasmid species. For example, direct transmission for *Babesia gibsoni* is highly likely and may be the main mode of transmission in some geographical regions such as Australia (Birkenheuer *et al.,* 2005), North America (Jefferies *et al.,* 2007; Solano-Gallego and Baneth, 2011) and Europe (Solano-Gallego *et al.,* 2016). Vertical transmission is also possible by transplacental infection (Fukumoto *et al.,* 2005; Mierzejewska *et al.,* 2014). Another route of direct transmission in human is through blood transfusion in North America (Young *et al.,* 2012). On the other hand, experimental transmission of *Babesia* spp. from domestic to wild animals is

usually only successful in closely related species or after splenectomy (Evers *et al.*, 2003; Penzhorn, 2006).

2.2.5 Role of wildlife in the epidemiology of endo parasites

Wildlife appears to be capable of living with high parasite loads without any apparent ill-effect on their health (Erkenswick *et al.*, 2017). Helminths play an important role in the regulation of wildlife populations by having a density dependent harmful effect on their hosts (Henke *et al*., 2002). The density-dependent process is likely to be most apparent with helminths with a direct life cycle (Henke *et al.*, 2002). The regulatory role parasites play most often functions in combination with other biotic and abiotic factors (Van-Wyk and Boomker, 2011). For instance, during drought, dormant parasites may flare up to cause disease in an already compromised animal. Therefore, the prevalence of helminths is dependent on intrinsic factors related to the parasite itself, or extrinsic factors related to the host, both intermediate and definitive, or environmental factors (Van-Wyk and Boomker, 2011). Human intervention, such as translocation of livestock and fencing, can also influence the prevalence of helminths (Erkenswick *et al*., 2017).

The capacity of helminths to produce a high number of eggs is determined by many factors including biotic potential where a single host of nematode may harbour thousands of reproductively active female nematodes (Van-Wyk and Boomker, 2011). Viability of larval stages is determined by the availability of eggs on pasture or veld, host's defaecation habits and the feeding habits of the intermediate host (Boomker *et al.*, 2000). It is known that grazers usually harbour higher numbers of internal parasites than browsers (Horak, 1981). A definitive host that makes use of a latrine will play a lesser role in disseminating eggs compared to a host that defaecates indiscriminately

(Boomker *et al.*, 2000). Several agents have been suggested to play a role in the dispersal of helminth eggs, namely birds, the wind, rainfall, arthropods, earthworms, animal feet and blowflies (Raether and Hänel, 2003). Overgrazing, periodic droughts and the erosion give the parasites as hard a time as they do the livestock as such parasites themselves have several mechanisms to overcome and survive adverse environmental conditions through Hypobiosis which is the arrested development of the free-living stages of a nematode and is triggered by stimuli such as temperature and photoperiod which results in the inhibited development in a subsequent stage of the life cycle (Reinecke, 1983). Host specificity is a fundamental characteristic of parasites, yet it is seldom absolute and to a variable degree it is relative (Horak, 1981). Parasites can be considered as definitive, occasional or accidental parasites of their respective hosts. Definitive parasites have a high prevalence in the specific host population and the population has a high mean intensity of the respective parasite (Horak, 1981). The parasite can reproduce and survive for long periods in these hosts. Occasional parasites have a significant variance in the number of parasites per host. The parasite may be capable of reproduction, but survival is for only a limited period. Accidental parasites have a low prevalence in the host population (Van-Wyk and Boomker, 2011).

2.2.6 Role of livestock in the epidemiology of endo parasites

Livestock in Nigeria represent a valuable asset in both traditional and modern agriculture; in addition, they also provide meat, milk, skin, and draught power for farming (Tewe, 1997; Adedipe *et al.*, 2014). In some traditional settings, they also play an essential role in the socioeconomic system, representing family wealth or they can be regarded as a survival kit by nomadic people (Adedipe *et al.*, 2014). In Nigeria, the livestock sector contributes 5.2 % of the gross domestic products (GDP) while cattle

production solely contributes 50 % of the total meat (Adedipe *et al.,* 2014). Meat is one of the most important livestock products, although there could be losses due to various diseases including helminth infections. The quantity of meat and revenue obtained from domestic livestock is far below the national demand due to factors such as death and ill health with associated reduced productivity and increased cost of treatment (Bolajoko *et al.,* 2011; Adedipe *et al.,* 2014). Helminths are known to be a major constraint to domestic species well-being and productive performance (Keyyu *et al.,* 2005; Rajput *et al.,* 2006; Hesterberg *et al.,* 2007). Gastrointestinal helminths are ubiquitous parasitic agents of animals and are known to limit their production in many areas and countries (Hesterberg *et al.,* 2007; Adedipe *et al.,* 2014).

Mortality of animals due to parasitic diseases may not be alarming at times but their indirect effects on livestock productivity and their zoonotic impact on human health are considerably greater (Nahed-Toral *et al.,* 2003; Nwosu *et al.,* 2007; Ekong *et al.,* 2012). Indirect losses associated with helminth infections include the reduction in productive potential such as decreased growth rate, weight loss, diarrhea, anorexia, and sometimes anaemia (Swai *et al.,* 2006; Adedipe *et al.,* 2014).

Studies on prevalence of horse helminths in different parts of world have indicated varied prevalence under different management and parasite control systems (Chaudhry *et al.,* 1991; Chapman *et al.,* 2002; Boxell *et al.,* 2004; Capewell *et al.,* 2005). Strongylosis is the most common and economically devastating disease of horses; clinically infected horses exhibit signs of unthriftiness, anaemia, colic and diarrhoea (Urquhart *et al.,* 1996). Young horses may carry thousands of parasites and experience severe clinical symptoms with certain mortality if not treated (Herd, 1990).

Donkeys represent an important aspect of small holder farming system especially in rural communities and hamlets, where there exist poor road networks or even non-existing roads; they are used for conveying people, goods, and farm inputs and outputs to and from farms (Parsani *et al.,* 2013). Unfortunately, Donkeys are left malnourished and underfed, subjected to different conditions of hardships like maltreatment, poor management conditions, and over laden (Parsani *et al.,* 2013). Donkey still remains one of the most underappreciated animals and an important draught one, serving a key role in the agricultural economy of the developing world including Africa (Ahmed *et al.,* 2008). Donkeys are hosts to a variety of gastrointestinal tract parasites of the family Strongylidae, commonly called *Strongyle* nematodes or *Strongyles* (Ayele and Dinka, 2010). The most common gastrointestinal parasites of donkeys include large and small *Strongyles, Ascaris*, pinworms (*Oxyuris equi*), *Gasterophilus*, lung worms, and fluke and tape worms which are by far the commonly encountered parasites in most veterinary parasitological examinations of donkeys (Ibrahim *et al.*, 2011; Worku and Afera, 2012; Samuel *et al.*, 2015; Atawalna *et al.*, 2015; Jajere *et al.*, 2016). Some of these gastrointestinal tract parasites, especially *Strongyles*, are active bloodsuckers; where there is heavy parasitic burden, the commonly encountered manifestations are anaemia, weakness, emaciation, and sometimes colic and diarrhea (Burden *et al.*, 2010).

2.3 Wildlife-Livestock Interfaces

2.3.1 Types of wildlife-livestock interfaces

Wildlife and livestock contribute significantly to the economies of most sub-Saharan African countries. The wildlife sector in Africa is worth US $7 billion with an annual growth rate of 5%. It is thus a major contributor to the continental gross domestic

product (GDP) (Kock *et al.,* 2002). Whereas the livestock subsector contributes over 30% of the agricultural GDP and employs more than 50% of the agricultural labours force (Kock *et al.,* 2002). Dairy and livestock farming accounts for utilization of 30% of the high-to-medium-potential land and of 81% of the arid and semi-arid lands and is crucial for promoting rural development and reducing poverty (Kock *et al.,* 2002). The wildlife-livestock interface in Africa is largely influenced by the livestock production systems present in the country where the Nomadic and transhumance pastoralism is widely practiced compared to the ranching system which is rare (Kock *et al.,* 2002; Farnsworth *et al.,* 2005). The rapid growth in global human population has resulted in the expansion of human-dominated landscape and use of more land for agriculture and irrigation and developmental activities (Farnsworth *et al.,* 2005). This population growth has also led to the rise in demand for animal protein; thereby extensive livestock farming has taken place over the years and the altered agricultural practices results in the change in habitat thereby increasing competition for grazing and water resources with interactions between wildlife and livestock on a continuous or seasonal basis at the wildlife-livestock interface level (Farnsworth *et al.,* 2005).

Three main types of wildlife–livestock interfaces are present in Africa, each of them encompassing a range of different contexts (Kock *et al.,* 2014). Namely (1) a fenced (ranch) system, where land use is designated for wildlife or livestock or both, (2) an open rangeland with free mixing of wildlife and livestock, (3) protected area boundaries where livestock are excluded or access controlled on one side and wildlife unwanted and partially accepted on the other. In Africa, these different types of interface are not evenly distributed due to different conservation and natural resource policies (Kock *et al.,* 2014).

2.3.2 Disease transmission at the wildlife-livestock interface

Disease transmission is arguably one of the major obstacles to the coexistence of wildlife and livestock in sub-Saharan Africa (Bourn and Blench, 1999). Generally, in African countries pastoralists keep indigenous breeds of livestock that are more resistant to pathogens and that are well adapted to their environments (Kock *et al*., 2002). However, land-use pressure and conflict between pastoralists and wildlife have been increasing, resulting in a growing risk of disease transmission between livestock and wildlife and increasing competition for grazing and water resources (Kock *et al*., 2002; Atuman *et al*., 2014). Most pastoralists survive at subsistence level and have limited access to veterinary services. Disease control measures usually rely on ethno veterinary practices, based on traditional knowledge of livestock diseases (Bourn and Blench, 1999).

Movements of both humans and animals occur across the boundaries of game parks often driven by resource gradient which results in direct and indirect contact between wild and domestic animals, potentially leading to disease transmission (Murwira *et al.,* 2013). This is a major problem by people living in areas where wildlife and livestock frequently come into contact. Movements of livestock and wildlife across the edges of protected areas are an avenue for the transmission of pathogens in either direction: from wildlife to livestock and vice-versa. In some cases the direction of pathogen transmission has been documented in South Africa for Rabies (Sabeta *et al.*, 2003); Foot and Mouth Disease (Vosloo *et al.,* 2005) and Bovine tuberculosis (Michel *et al.*, 2006). The role as disease reservoir played by wildlife species is equally well established, for example, Buffalo act as reservoir of *Theileria parva lawrenci* responsible for ‘corridor’ disease. But in most of the cases a role is suspected but not sufficiently documented, as

the occurrence of the same disease is no proof of the transmission of pathogens between wildlife and livestock. Disease occurring in livestock and wildlife interface can be categorized into:

1. Wildlife-maintained diseases, to which wildlife are resistant or silent carriers of infection
2. Multi-species diseases, which have serious outcomes in both livestock and wildlife.

Important wildlife-maintained diseases are many including Foot and Mouth disease (FMD), malignant catarrhal fever, trypanosomosis, theileriosis (corridor disease), ehrlichiosis (heart water) lumpy skin disease and rift valley fever (Bengis *et al.*, 2002). Multi-species diseases, which include anthrax, rabies, brucellosis, and bovine tuberculosis, occur in both wildlife and domestic livestock, and transmission can occur in both directions. Uniquely these diseases generally have a fatal outcome in both wildlife and domestic livestock (Murwira *et al*., 2013).

Some wildlife species and cattle have similar ecological niches and tend to be attracted by the same type of food resources. Grazing and water are limiting factors in most semi-arid areas of Africa where wildlife/livestock live sympatrically. These resources potentially create opportunities for wildlife–livestock contacts and pathogen transmission (Sinclair, 1977), and seasonal profiles of contacts are usually observed, with higher rates of contact during the dry season when resources are depleted (Kock *et al*., 2006).

2.4 Haematological and Biochemical Parameters in Wildlife

Haematological and biochemical studies are important tool for health evaluation and their interpretations to know the status of physiological functions of various organs. The

concentration of biochemical constituents in tissues as well as in body fluid is fixed and during adverse conditions, it may be elevated or decreased (Douglas and Nelson, 1991). Haematology refers to the study of the numbers and morphology of the cellular elements of the blood – the red cells (erythrocytes), white cells (leucocytes), and the platelets (thrombocytes) and the use of these results in the diagnosis and monitoring of disease (Douglas and Nelson, 1991). Haemtological studies are useful in the diagnosis of many diseases as well as investigation of the extent of damage to blood (Onyeyelli *et al.,* 1991; Togun *et al.,* 2007). Haematological studies are of ecological and physiological interest in helping to understand the relationship of blood characteristics to the environment (Ovuru and Ekweozor, 2004) and so could be useful in the selection of animals that are genetically resistant to certain diseases and environmental conditions (Mmereole, 2008; Isaac *et al.,* 2013). Haematological parameters are good indicators of the physiological status of animals (Khan and Zafar, 2005). Haematological parameters are those parameters that are related to the blood and blood forming organs (Bamishaiye *et al.,,* 2010; Waid *et al.,* 1985). Blood act as a pathological reflector of the status of exposed animals to toxicant and other conditions (Olafedehan *et al.,* 2010). As reported by Isaac *et al.,* (2013) animals with good blood composition are likely to show good performance. Laboratory tests on the blood are vital tools that help detect any deviation from normal in the animal or human body (Afolabi *et al*., 2011). The examination of blood gives the opportunity to investigate the presence of several metabolites and other constituents in the body of animals and it plays a vital role in the physiological, nutrition and pathological status of an organism (Aderemi, 2004; Doyle, 2006). According to Olafedehan *et al.* (2010) examining blood for their constituents can provide important information for the diagnosis and prognosis of diseases in animals. Blood constituents change in relation to the physiological conditions of health (Togun *et*

al., 2007). These changes are of value in assessing response of animals to various physiological situations (Khan and Zafar, 2005). According to Afolabi *et al.* (2011) changes in haematological parameters are often used to determine various status of the body and to determine stresses due to environmental, nutritional and/or pathological factors.

2.4.1 Haematological components and their functions

Blood which is a vital special circulatory tissue is composed of cells suspended in a fluid intercellular substance (plasma) with the major function of maintaining homeostasis (Isaac *et al.,* 2013). Haematological components, which consist of red blood cells, white blood cells or leucocytes, mean corpuscular volume, mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration are valuable in monitoring feed toxicity especially with feed constituents that affect the blood as well as the health status of farm animals (Oyewoye and Ogunkunle, 2004; Opara and Okoli, 2006)

2.4.1.1 Red blood cells

They serve as a carrier of haemoglobin. It is this haemoglobin that reacts with oxygen carried in the blood to form oxyhaemoglobin during respiration (Chineke *et al.,* 2006). According to Isaac *et al.* (2013) red blood cell is involved in the transport of oxygen and carbon dioxide in the body. Thus, a reduced red blood cell count implies a reduction in the level of oxygen that would be carried to the tissues as well as the level of carbon dioxide returned to the lungs (Ugwuene, 2011; Isaac *et al.,* 2013; Soetan *et al.,* 2013). In wild animals, the clinical signs and their magnitude depends on habitat and availability of nutritive materials. Prolonged nutritional deficiencies of protein vitamins and

minerals essential for erythrocytes production lead to anaemia (Shrivastav and Singh, 2012). A normocytic – nonchromatic, non-responsive anaemia is commonly found in association with chronic infections, chronic infectious inflammatory conditions and some type of malignancies though microcytic-hypochromic is the sign of iron deficiencies (Jain and Kono, 1975). Several blood sucking parasites produce blood loss anaemia in wildlife like *Haemonchus* spp, *Toxocara* spp that may cause haemolytic anaemia while *Trypanosomes, Babesia* and *Anaplasma* may alter the total blood as well as plasma volumes with acute blood loss (Shrivastav and Singh, 2012). Chronic blood loss may lead to gastrointestinal lesions, ulcers, heavy parasitism like coccidiosis, neoplasm with bleeding into body cavity, deficiency of Vitamin K and prothrombin (Shrivastav, 2001).

2.4.1.2 White blood cells

The major functions of the white blood cell and its differentials are to fight infections, defend the body by phagocytosis against invasion by foreign organism and to produce or at least transport and distribute antibodies in immune response (Esievo, 2017). Thus, animals with low white blood cells are exposed to high risk of disease infection, while those with high counts are capable of generating antibodies in the process of phagocytocis and have high degree of resistance to diseases (Soetan *et al.,* 2013) and enhance adaptability to local environmental and disease prevalent conditions (Isaac *et al.,* 2013; Kabir *et al.,* 2011; Okunlola *et al.,* 2012). Blood platelets are implicated in blood clotting. Low platelet concentration suggests that the process of clot-formation (blood clotting) will be prolonged resulting in excessive loss of blood in the case of injury (Soetan *et al.,* 2013).

2.4.1.3 Packed cell volume

Pack cell volume (PCV) which is also known as haematocrit (Ht or Hct) or erythrocyte volume fraction (EVF), is the percentage (%) of red blood cells in blood (Etim *et al.*, 2014). According to Isaac *et al.* (2013) PCV is involved in the transport of oxygen and absorbed nutrients. Increased PCV shows a better transportation and thus results in an increased primary and secondary polycythemia.

2.4.1.4 Haemoglobin concentration

The haemoglobin (Hb) is the iron -containing oxygen-transport metalloprotein in the red blood cells of all vertebrates (Acharya *et al.*, 2012) with the exception of the fish family (Sidell and O'Brien, 2006) as well as tissues of invertebrates. Haemoglobin has the physiological function of transporting oxygen to tissues of the animal for oxidation of ingested food so as to release energy for the other body functions as well as transport carbon dioxide out of the body of animals (Ugwuene, 2011; Omiyale *et al.*, 2012; Soetan *et al.*, 2013). According to Peters *et al.* (2011) PCV, Hb and MCH are major indices for evaluating circulatory erythrocytes, and are significant in the diagnosis of anaemia and also serve as useful indices of the bone marrow capacity to produce red blood cells as in mammals (Awodi *et al.*, 2005; Chineke *et al.*, 2006). Furthermore, Chineke *et al.* (2006) posited that high PCV reading indicated either an increase in number of Red blood cells (RBCs) or reduction in circulating plasma volume. Mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration indicate blood level conditions in which a low level is an indication of anaemia.

2.4.1.5 Leukocytes

The total leukocytes count (TLC) and differential leukocytes count are important parameters to judge the body response against diseases (Shrivastav and Singh, 2012).

Leukocytosis may be physiologically mediated by endogenous release of epinephrine or corticosteroids or it may be pathologic response to a diseases process (reactive leukocytosis) or a result of a neoplastic change in the haematopoiesis (proliferative leukocytosis) while leucopenia is always pathologic event (Shrivastav and Singh, 2012). Quantitative and qualitative changes in a particular type of leukocyte indirectly reflect the nature of disease process and the body response to it. Jain (1986) reported physiologic factors such as fright and "emotional" disturbances as an immediate effect on TLC and DLC and may confined to interpretation of conditions (Opara and Okoli, 2006). The normal response to the stress is decrease in lymphocytes and eosinophil numbers. In "emotional" leucocytosis, lymphocyte numbers are increased and equal or exceed neutrophil numbers while eosinophil commonly not affected (Shrivastav and Singh, 2012).

2.4.1.6. The neutrophils

Neutrophils considered as first line of defence against microbial infections and are important participants in inflammatory reactions (Shrivastav and Singh, 2012). A change in blood neutrophil differential count is associated with many consequences related to infectious diseases (Shrivastav and Singh, 2012). Several functions have been suggested for the contents of granules, as neutrophils are phagocytic cells and regulating adhesiveness and aggression hydroxyl radical formation and generation of compliment derived chemotactic factors while azurophilic granules are involved in modulation of inflammatory process (Galli, 1990). Condensation of nuclear chromatin leads to formation of darker-staining plaques separated by delicate, light-staining areas with slight brown colour cytoplasm.

2.4.1.7 The eosinophils

Eosinophils contained small, uniformly round bright eosinophilic granules almost occupying the entire and clear cytoplasm. The nuclei of the cells are generally less lobulated than those of the neutrophils. The eosinophils are slightly larger than neutrophils. The eosinophils are commonly seen in prolonged parasitic infections or allergic disorders (Shrivastav and Singh, 2012).

2.4.1.8 The basophiles

The basophile is a numerically insignificant but functionally important leukocyte that resembles mast cells and it believed to share similar function as it is associated with allergic reaction, inflammatory process and immunocompitancy to the body fluids. The phagocytosing ability of basophils is varied among species of wildlife depending on the ecology (Galli, 1990). The basophiles were rarely observed up to the size with 0 to 0.4 0.1 ± 1.21 5 % in free range wildlife (Shrivastav and Singh, 2012).

2.4.1.9 The lymphocytes

The lymphocytes are comparatively smaller than eosinophils. Small and large lymphocytes were also seen in the blood smear of wildlife species and some lymphocytes contained a few azurophilic granules in their cytoplasm (Shrivastav, 2001). Meyers-Wallen *et al.* (1984) attributed that wildlife species have high tendencies to develop lymphocytosis due to their wild habitat as escape behaviour.

2.4.1.10 The monocytes

The Monocytes are usually larger than lymphocytes with distinguishing feature of the reddish grey nucleus and well-defined vacuoles, the nucleus of the monocytes reported

amoeboid and sometime horseshoe shaped (Jain, 1986). The monocytes are associated with phagocytosis principally against intracellular bacteria, viruses, fungi and protozoa. The cells perform regulation of the immune response, phagocytic removal of tissue debris (affected cells, antibody coated cells and other foreign materials) as scavenger (Jain, 1986).

2.4.1.11 The platelets

Platelets are abundant in blood smear and usually distributed in small to large clumps. Shrivastav (2001) reported that individual platelets are pleomorphic with rounded to elongated shapes with a central cluster of azurophilic granules. Jain, (1986) observed the clumping of platelets in the blood and emphasized that the platelets of animal during excitement of 3 minutes and can cause a sudden increase in platelet counts. A slight decrease of platelet count occurred in sympathectomized animals and a somewhat greater decrease reported in splenectomized animals (Shrivastav, 2001).

2.4.2 Biochemical components and their function

The concentration of biochemical compounds in tissues and body fluid can be measured in a colorimetery, as it is capable of absorbing light of a particular wave length (Singh, 2004). Thus, the health status of animal can be assessed by evaluation of blood gases, acid base balances, electrolytes, metabolic intermediates, inorganic ions, enzymes and hormones (Shrivastav and Singh, 2012). Viral, bacterial and parasitic diseases are very common in wildlife which can affect the biochemical normal values (Jain, 1986; Rao and Acharyjo, 2002). Types of anaemia and significant blood loss may be estimated through complete blood count (CBC) and physiological function of different organs by biochemical parameters (Jain, 1986). Study of Biochemical components by blood

analysis of Albumin, Total protein, Total bilirubin, glucose, creatinine, Blood urea nitrogen (BUN), Glutamic oxalo-transaminase (GOT/AST), Glutamic pyruvic transaminase (GPT/ALT) by using an ERBA Chem-5 plus auto-analyzer (Transasia Bo-medicals Ltd.) with standard ERBA reagent kits for respective plasma timating the physiological function of different organs in an animal. (Shrivastav and Singh, 2012).

2.4.2.1 Icterus index

Icterus is known as jaundice or yellow jaundice. It refers to an excessive accumulation of a yellow pigment in the blood and tissues. When icterus has been present for any length of time, it will discolor many tissues and will become visible as jaundice on most body surfaces, including the skin (Esievo, 2017). It is most easily seen in the gingivae (gums), the sclerae (white part of the eyes), and the pinnae (ear flaps). The pathological causes of icterus in animals include destruction of red blood cells (haemolysis) which can occur within blood vessels or in the spleen or liver leading to haemolytic anaemia and disease conditions that damage liver and cause obstruction of bile duct (Shrivastav and Singh, 2012). Icterus index is a calculation indicating the level of bilirubin in serum or plasma determined by comparing the color of a sample of test serum with a set of color standards, it is the quantification of interference by bilirubin and can be measured in autoanalyzer without any reagent cost (Shrivastav and Singh, 2012). The icteric index is a semi quantitative test and can detect patients with total bilirubin values above the reference range. An increase in the values of Icterus index in plasma is an indicative of an absolute increase in bilirubin concentration due to removal of aged erythrocytes from the circulation by the reticuloendothelial and liver (Shrivastav and Singh, 2012).

2.4.2.2 Total plasma protein

Protein in plasma can provide information reflecting functional status of various organ and systems as blood is composed of approx 20 % of protein excluding haemoglobin (Richard, 1991). However, the total protein values give the information on nutritional consequences or severe organ diseases as they transported the carrier of most of the constituents of the plasma, maintains the colloid osmotic pressure, act as catalysts in biochemical reaction and play important role in formation of fibrin polymers during clot formation (Richard, 1991). The total plasma protein values are commonly increased in haemoconcentration and reduced in malnutrition, hepatopathy, less intake of protein and in neoplastic condition (Shrivastav and Singh, 2012).

2.4.2.3 Plasma albumin

The liver produces all the albumin and globulins while a small number of globulins is produced by reticuloendothelial tissue (Benjamin, 1978). Liver synthetic capacity or protein-losing nephropathy can be measured by albumin estimation in the blood plasma or serum. It also can interpret high or low calcium and magnesium level since albumin binds about one half of each of the ions (Richard, 1991). However, it appears to be a direct correlation between albumin turnover and body size because it is clinically significant. It is usually constituted with two third of total plasma protein and also serve as mobile amino acids for the liver (Shrivastav and Singh, 2012). Generally, hypoalbuminism is observed in malnutrition, increased protein catabolism, nephropathy and chronic enterophathy (Shrivastav, 2001). Reduction in total albumin values is observed in malnutrition, liver diseases, stress, kidney dysfunction (Shrivastav and Singh, 2012).

2.4.2.4 Total bilirubin

Bilirubin is a breakdown product of heme about 70 percent of which is derived from senescent red cells (Crawford *et al.,* 1988) however, 15 percent comes from hepatic cytoplasm and mitochondrial cytochromes and some from renal and other cytochromes, and some from defective red blood cell broken down in the bone marrow before release (Shrivastav, 2001). The yellow colour of serum or plasma is due chiefly to the pressure on bilirubin. Increased concentration of bilirubin is commonly seen in haemolysis, hepatocellular damage and biliary obstruction (Shrivastav, 2001).

2.4.2.5 Creatinine

Creatinine is important in muscles metabolism in that it provides storage of high energy phosphates through synthesis of phosphocreatine (Benjamin, 1978). Serum or plasma creatinine concentration and urinary creatine secretion are increased significantly by skeletal muscles necrosis or atrophy and defect in renal functions (Shrivastav and Singh, 2012).

2.4.2.6 Blood urea nitrogen

Urea is the end product of protein and amino acids and is generated in the liver through urea cycle (Woo and Cannon, 1991). Blood Urea Nitrogen is one of the important tools to know the renal function status. The decreased values of BUN are commonly seen in malnutrition and hepatic insufficiencies; however, increased BUN is generally associated with renal disease congestive heart failure, shock and hypertension. Shrivastav and Singh (2012) observed that the high rise of BUN in canine species might be also due to adlib intake of meat.

2.4.3 Hepatic enzyme

The serum enzymes used routinely in clinical diagnosis are synthesized in liver (Schaffner and Schaffner, 1991). In hepatocellular or in cholestatic forms of liver injury these hepatic enzymes are released in to the serum. The serum enzyme activities that are elevated in hepato cellular damage are Alanine Aminotransferase (ALT) Aspertate Aminotransferase (AST), Ornithine Carbamoyl transferase (OCT), Glutamic Dehydrogenase (GD), Sorbitol Dehydrogenase (SDH) and arginase. The elevated serum activities that suggest cholestasis (intra hepatic or extrahepatic) are Alkalinephosphotase (AP), Gamma glutamyltranspeptidase (GGT) and 5' nucleotidase (5'ND). The pathogenesis of the hepatic disease is associated with viral hepatitis, parasitic infections or mechanical injuries (Rao and Acharjyo, 2002). The liver has great functional reserves and signs of hepatic failure often do not develop until 70% or more of the functional capacity of the liver is lost (Schaffner and Schaffner, 1991).

2.4.3.1 Alanine aminotransferase

Alanine Aminotransferase (ALT) is an enzyme associated with liver parenchymal cells mainly in the liver, but also in smaller amounts in the kidneys, heart, muscles, and pancreas(Benjamin, 1978; Enwezor *et al.,* 2009). It is a more specific indicator of liver inflammation. ALT was formerly called serum glutamic pyruvic transaminase (SGPT). ALT is measured to see if the liver is damaged or diseased. Low levels of ALT are normally found in the blood. But when the liver is damaged or diseased, it releases ALT into the bloodstream, which makes ALT levels go up. Most increases in ALT levels are caused by liver damage (Esievo, 2017).

2.4.3.2 Aspertate aminotransferase

Aspertate aminotransferase (AST) just like ALT is an enzyme associated with liver parenchymal cells mainly in the liver (Esievo, 2017). It was formerly known as serum glutamic oxaloacetic transaminase (SGOT). Apart from liver, AST is also present in muscles and cardiac muscles. The higher value of AST though is not an organ specific but used as an indicator of liver dysfunctions (Shrivastav, 2001). AST may be elevated also in diseases affecting other organs, such as myocardial infarction, acute pancreatitis, acute hemolytic anemia, severe burns, acute renal disease, musculoskeletal diseases, and trauma. As a prototypical transaminase, AST relies on PLP (Vitamin B6) as a cofactor to transfer the amino group from aspartate or glutamate to the corresponding ketoacid. In the process, the cofactor shuttles between PLP and the pyridoxamine phosphate (PMP) form (Shrivastav, 2001). The amino group transfer catalyzed by this enzyme is crucial in both amino acid degradation and biosynthesis. In amino acid degradation, following the conversion of α-ketoglutarate to glutamate, glutamate subsequently undergoes oxidative deamination to form ammonium ions, which are excreted as urea. In the reverse reaction, aspartate may be synthesized from oxaloacetate, which is a key intermediate in the citric acid cycle (Esievo, 2017).

2.4.3.3 Alkaline phosphatase

Alkaline Phosphatase (ALP) is a homodimeric protein enzyme of 86 kilodaltons, containing two zinc atoms crucial to its catalytic function per monomer, and is optimally active at alkaline pH environments (Kim and Wyckoff, 1991). As its name indicates, ALP functions best under alkaline pH environments and has the physiological role of dephosphorylating compounds. The enzyme is found across a plethora of organisms, prokaryotes and eukaryotes alike, with the same general function but in

different structural forms suitable to the environment they function in (Kim and Wyckoff, 1991; Esievo, 2017). In humans for example, it is found in many forms depending on its origin within the body - it plays an integral role in metabolism within the liver and development within the skeleton. Due to its widespread prevalence in these areas, its concentration in the bloodstream is used by diagnosticians as a biomarker in diagnoses of conditions such as hepatitis or osteomalacia (Karaca and Ugar, 1999). Alkaline phosphatase is commonly used in the dairy industry as an indicator of successful pasteurization. This is because the most heat stable bacterium found in milk, *Mycobacterium paratuberculosis*, is destroyed by temperatures lower than those required to denature ALP. Therefore, ALP presence is ideal for indicating successful pasteurization (Karaca and Ugar, 1999).

2.4.3.4 Lactate dehydrogenase

Lactate dehydrogenase (LDH) is an enzyme that catalyzes the conversion of lactate to pyruvate the enzyme is required during the process of turning sugar into energy in cells (Madern, 2002). LDH is present in many kinds of organs and tissues throughout the body, including the liver, heart, pancreas, kidneys, skeletal muscles, lymph tissue, and blood cells. As cells die, their LDH is released and finds its way into the blood (Madern, 2002). Funtional LDH-1 (4H) are found in the heart and in RBC, LDH-2 (3H1M) in the reticuloendothelial system, LDH-3 (2H2M) in the lungs, LDH-4 (1H3M) in the kidneys, placenta, and pancreas and LDH-5 (4M) in the liver and striated muscle (Madern, 2002).

CHAPTER THREE

MATERIALS AND METHODS

3.1 Study Area

The study was conducted in Bauchi State, located between Longitude 9˚ 15□ E to 10˚ to 43□ E and Latitude 9˚ 55□ N to 12˚ 45□ N in the Northern Guinea/Sudan savannah zone of Nigeria. It has an annual rainfall of between 875 and 1075 mm. It shares borders with Plateau and Kaduna States to the west, Kano and Jigawa States to the north, and Yobe, Gombe, and Taraba States to the north-east (Olokesusi, 1990). The study locations were Yankari game reserve and Sumu wildlife park of Bauchi State and settlements surrounding them.

The Yankari game reserve (YGR) is located in the south-central part of Bauchi State. The reserve has an area of about 2,244 square kilometres and is surrounded by three districts Pali, Gwana and Duguri in Bauchi State (Omondi *et al.,* 2006). It lies between Latitude 9°34’ and 10°00’ N and Longitudes 10°17’ and 10°47’E. It is known to be an important refuge for over 50 species of mammals and over 350 species of birds and one of the few remaining areas where wild animals are protected in their natural habitat in Nigeria (Odunlami and Lake, 2003). Notable among the wildlife species are olive baboon (*Papio anubis*), Common warthog (*Phacochoerus africanus*), African buffalo (*Syncerus caffer*), Savanna elephant (*Loxodonta africana*), Western hartebeest (*Alcelaphus buselaphus*), roan antelope (*Hippotragusequinus)*, waterbuck (*Kobus defassa*), lion (*Pantheraleo*), hippopotamus (*Hippopotamus amphibius*), and bushbuck (*Tragelaphus scriptus*) (Omondi *et al.,* 2006). The assembly of these animals within the ‘Vegetation Island’ enhanced the tourist potential of the place and may have been one reason for the gazettement of the area as Bauchi Native Authority Yankari Forest

Reserve order, 1957 and was hence opened to tourists in December 1962 (Ezealor, 2002; Odunlami and Lake, 2003; Tende *et al.,* 2014). It was upgraded to a national park in 1992, although it was later handed over to the Bauchi State government in June 2006 (Tende *et al.,* 2014). The reserve boundaries continued to expand as a result of enactments of successive authorities and the animals stretched out to graze in areas hitherto not contained in the Reserve (Tende *et al.,* 2006).

The YGR is situated on sedimentary rocks with many rocky outcrops and consists of gently rolling hills. The altitude ranges between 200 m above sea level at the lowest point on the Gaji River at it exits from the YGR near Rimi village to 640m at the highest point east of the Kariyo Hill (Geerling, 1973). The largest river in the reserve is the perennial Gaji River which is joined in the north by Yashi River and in the south by Yuli River where it runs into the River Benue. The reserve has a high ground water level with five springs (Wikki, Dimel, Manulgo, Gwana and Tunganmaliki), which drains into the Gaji river (Geerling, 1973; Green and Amanche, 1987). Average rainfall in this region ranges from 900 to 1000 mm with August as the wettest month. Temperatures are moderate between 18°C and 33°C during the rainy season. During the dry season, between November and February, night temperatures are between 12°C and 18°C while day temperatures range from 30°C and 36°C. Between March and April, night temperatures range between 25°C and 30°C while day temperatures are between 38°C and 42°C (Geerling, 1973). The vegetation of the YGR is Sudan but numerous springs and the high ground water level of the Gaji river valley are responsible for more luxuriant vegetation than normally found in the Sudan zone. The vegetation of the YGR is made up of Savannah trees and shrubs with an open canopy and continuous layer of annual perennial grasses (Geerling, 1973).

The Sumu Wildlife Park (SWP) was established in the year 2006, when about 250 wildlife species - including, impala (*Aepyceros melampus*), springbok (*Antidorcas marsupialis*), oryx (*Orynx gazelle*), eland (*Taurotragus oryx*), zebra *(Equus quagga crawshayi*) kudu (*Tragelaphus strepsiceros*), blue wildebeest (*Connochaetes taurinus*), and giraffe (*Giraffa camelopardalis*) were imported from Namibia to the SWP to boost the tourist potentials of Bauchi State (Atuman, 2011). The Government of Bauchi State decided to develop the Sumu forest reserve and fenced about 40 square kilometres of it to develop the park which is located about 14 kilometres from Kafin-Madaki, the Local Government Headquarters of Ganjuwa Local Government (Atuman, 2011). Most of the wild animals were young when they were brought into the park. Presently, they have fully adapted to the Nigerian climate and vegetation and have reproduced in large numbers. However, though some of the wildlife species like springbok and orynx could not adapt to the new habitat and so all of them died (Atuman, 2011). The tourism potential of the SWP is great because of the variety of animal species that are unique and hardly found in the other parks and game reserves in Nigeria. This made it to be of particular interest to zoologists and has started to attract tourists (Atuman *et al.*, 2014). Although the populations of wildlife species in SWP have reproduced in large number over the years, however their subsequent growth will be influenced by a number of factors which include the suitability of their new environment and the impact of parasites and diseases (Anderson and Rowe, 1998).

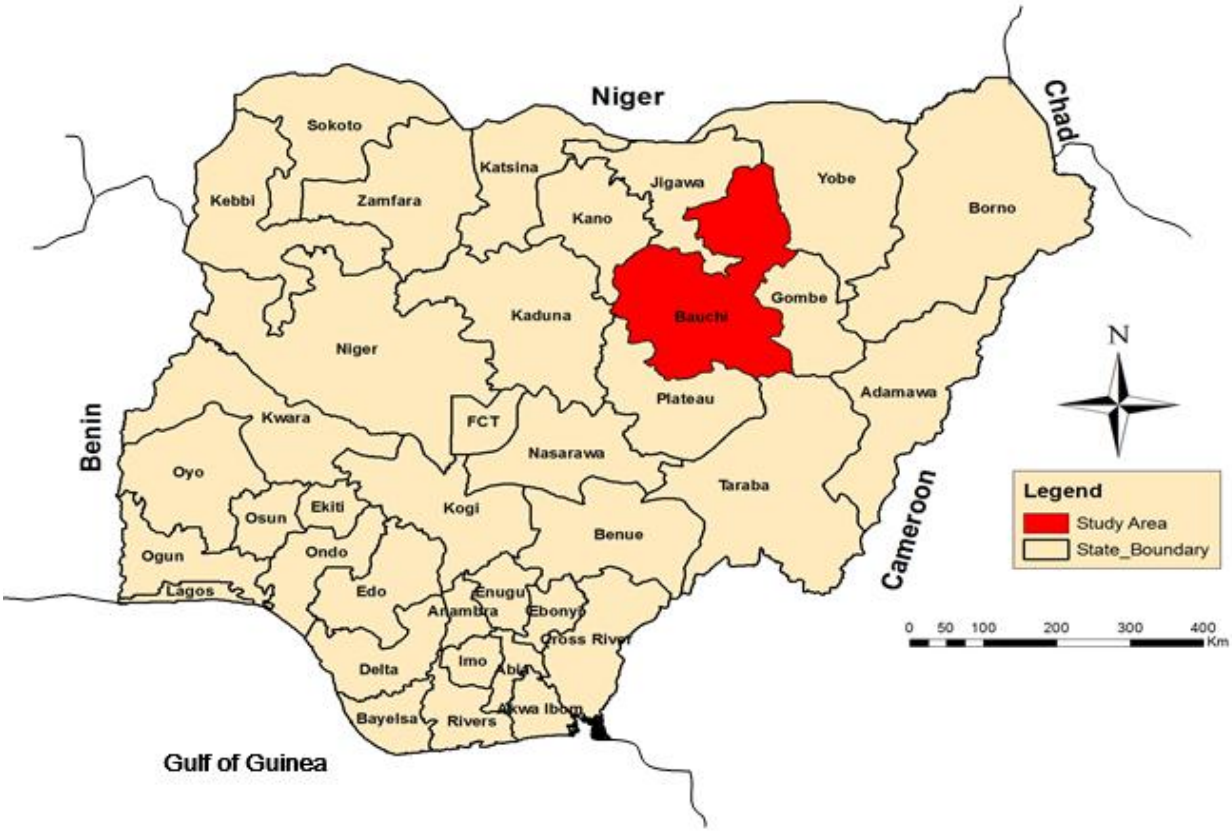

Figure 3.1 Map of Nigeria showing Bauchi State Source: Modified from the Administrative Map of Nigeria (http://www.theodora.com/maps)

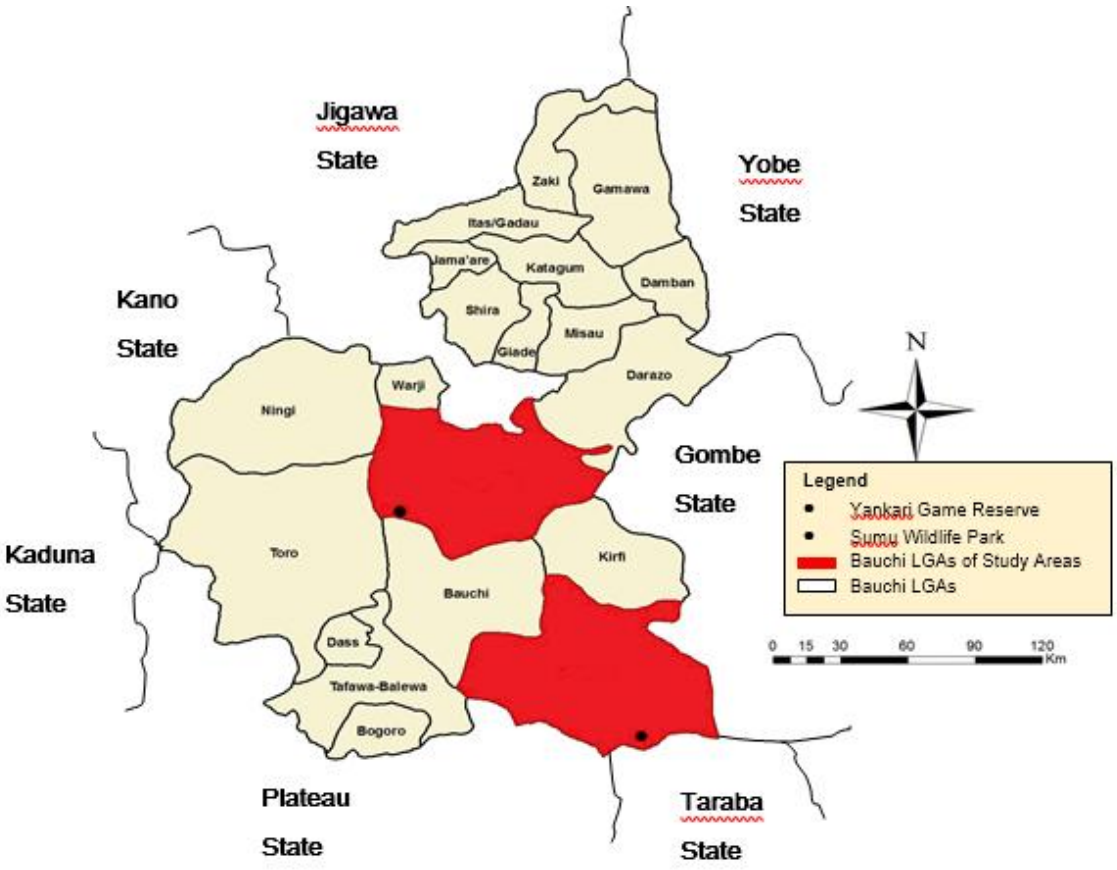

Figure 3.2: Map of Bauchi State showing study areas.Source: Modified from the Administrative Map of Nigeria (http://www.theodora.com/maps)

3.2 Sources of Samples

3.2.1 Wildlife sampled

Wildlife sampled included elephant (*Loxodonta Africana*), waterbuck (*Kobus ellipsiprymus*), Hartbeest (*Alcelaphus buselaphus caama*) from YGR and eland (*Taurotragus oryx*), zebra *(Equus quagga crawshayi*) kudu (*Tragelaphus strepsiceros*) and blue wildebeest (*Connochaetes taurinus*) from SWP.

3.2.2 Domestic animals sampled

Herds of cattle (*Bos indicus*) and donkeys (*Equus asinus*) owned by nomads who live in settlements at the fringes of the YGR and SWP with history of interacting with wildlife through grazing and sharing of water points were sampled. Horses (*Equus caballus*) used by rangers in SWP for patrol and those owned by villagers and nomads at the fringes of the YGR and SWP were also identified and samples collected from them.

3.3 Sampling Technique

Convenience sampling technique was used for both wildlife and domestic animals. Wildlife species were sampled during capture and translocation activities and also during field investigations for reported episodes of diseases and mortality in the YGR and SWP.

3.4 Sample Collection

Wildlife were immobilized using a combination of Etorphine Hydrochloride (M99® Krüger-Med South Africa) at 0.5-2 mg/kg combined with Azaperone (Stresnil®, Janssen Pharmaceuticals (Pty.) Ltd., South Africa) at 0.1mg/kg delivered intramuscularly (IM) from a distance of about 25 metres on ground in a 3mls dart syringe fitted with barbed needles using a Dan- Inject® rifle (Dan-Inject APS, Sellerup

Skovvej, Denmark). Immediately following immobilization, the animals were placed on lateral recumbency and the dart removed and blood samples collected. All vital parameters (temperature, pulse, respiratory rates and oxidation) were recorded accordingly every 10 min. For reversal, naltrexone (Trexonil® Kruger-Med South Africa) at 1.5 mg IM was used to ensure smooth and calm recovery of the wildlife (Cobus, 2012). Domestic animals were restrained by the owners who were identified and consulted for consent before sample collection.

Ten mililitre of blood samples were collected from the jugular vein of the wildlife and domestic animals and dispensed into an EDTA and non-EDTA vacuitainers. Faeces were collected per rectum into well-labelled sterile polythene bags and ticks were pulled off from the animals with forceps and placed in sterile loosely capped plastic universal sample bottles. All samples were transported in a cold box with ice packs to the National Veterinary Research Institute Laboratory Bauchi. Sera were collected from the blood in the non-EDTA vacutainers into cryovials after spinning for 10 min at 1200 g. All samples were divided into aliquots, labelled and kept at +4 □C for (blood and faeces) or (sera) at -20 □C until used.

3.5 Detection of Antibodies to Viral Infections

3.5.1 Rift valley fever virus antibody detection by enzyme linked immunosorbent assay

Sera samples from domestic and wildlife were evaluated at the Viral Research Division NVRI Vom, Nigeria for anti-RVFV IgM/IgG antibodies using ID screen® RVF competition multi-species ELISA kits (ID-Vet Innovative Diagnostics, Montpellier, France). Briefly described, 50µl each of ELISA buffer and test sera were added to each well containing the RVFV antigen coated test plates. Positive and negative control sera

were added to the designated wells on each test plate and were incubated for 45 min at 37^{o}C. The plates were emptied and washed three times with 300 µl of wash solution and 100 ml of diluted conjugate was added to all the plates and incubated for 30min at 21^{o}C. The plates were then emptied and washed 3 times with 300 µl washing solution and 100µl of the substrate solution was dispensed to all the wells and were incubated for 15 min at 21°C in the dark following which 100 µl of stop solution was added to all the wells. Readings were taken on a spectrophotometer Multiskan® ELISA reader (Thermo Scientific, USA) and the optical density (OD) determined at 450 nm. Positivity of each sample was determined by calculating its competition percentage (CP). Samples with CP = ≤40% were considered positive for RVFV antibodies, and those with CP = >50% were declared negative for RVFV antibodies while those with CP= >40%, ≤50 were declared doubtful.

3.5.2 Detection of foot and mouth disease virus antibody by enzyme linked immunosorbent assay

The ELISA was performed at the Viral Research Division NVRI Vom, Nigeria according to the manufacturer's instructions (PRIOCHECK® FMD-3ABC NS protein ELISA) for the detection of antibodies to the non-structural polypeptide 3 ABC of FMD virus in serum or plasma which detects infected animals regardless of their vaccination status and the FMDV serotype that caused the infection (Brocchi *et al*., 2006; Ularamu *et al*., 2017). Briefly described, 80 µl of the ELISA buffer and 20 µl of the test sera were added to the 3ABC-antigen coated test plates. Negative, weak positive and strong positive control sera were added to designated wells on each test plate, gently shaken and incubated overnight (18 h) at 22°C. The plates were then emptied and washed six times with 200µl of wash solution and 100 µl of diluted conjugate was added to all wells. The test plates were sealed and incubated for one hour at 22ºC. The plates were

then washed six times with 200 µl of wash solution and 100 µl of the chromogen (tetra-methyl benzidine) substrate was dispensed to all wells of the plates and incubated for 20 min at 22ºC following which 100 µl of stop solution was added to all the wells and mixed gently. Readings were taken on a spectrophotometer Multiskan® ELISA reader (Thermo Scientific, USA) at 450 nm and the OD 450 values of all samples was expressed as Percentage Inhibition (PI) relative to the OD 450 max using the following formula PI = 100 – [OD 450 test sample/OD450 max] × 100. Samples with PI = ≥ 50% were considered positive for FMD antibody while those with PI < 50% were declared negative for FMD antibody. Since the 3-ABC ELISA for FMD was = 100% specific and > 99% sensitive, the percentage prevalence was taken as true prevalence.

3.5.3 Detection of foot and mouth disease specific antibodies using solid-phase competitive enzyme linked immunosorbent assay

The 3ABC ELISA positive serum samples were analyzed for FMD-specific antibodies at the Viral Research division NVRI, Vom, Nigeria using a Solid-Phase Competitive ELISA (SPCE) for serotypes O, A, SAT 1 and SAT 2 (Chénard *et al*., 2003; Ularamu *et al*., 2017). The assays were performed using antibodies FMDV ELISA kits for serotypes O, A, SAT 1 and SAT 2 produced by IZSLER Biotechnology Laboratory (Italy). Briefly, 96 wells pre-coated with FMDV antigens captured by FMD serotypes O, A, SAT 1 and SAT 2 specific MAb flat-bottomed plates were used. Four dilutions of sera at 1/10, 1/30, 1/90 and 1/270 were made. Without washing, the conjugate (Horse-radish peroxidase) was added and incubated at room temperature for 1 h. The plate was washed and the substrate/chromogen solution (tetra-methyl-benzidine) was added and kept in the dark for 20 min. The reaction was stopped by the addition of a stop solution and the plates were read on a MultiSkan® spectrophotometer ELISA plate reader (Thermo Scientific, USA) at 450nm wavelength. Serum end-point titre was expressed as

the highest dilution producing 50% inhibition, with serum having end point titre ≥ 50% being classified as positive for the specific FMD antibody.

3.5.4 Detection of African horse sickness virus antibody by enzyme linked immunosorbent assay

Sera samples from horses and zebras were evaluated at the Viral Research Division NVRI Vom, Nigeria for total anti AHSV antibodies using INGEZIM AHSV COMPACT PLUS 14. AHSV.K3 as described by Maree and Paweska (2005). The principle of this test is to block the specific reaction between the recombinant VP7 protein absorbed on an ELISA plate and a conjugated MAb against VP7. The antibodies present in a suspicious serum will block this reaction and there will not be colour development which is evidence of presence of AHSV antibodies in the serum sample. Briefly, all reagents and samples were allowed to come to room temperature before use. About 100 µl of diluted samples, positive and negative controls were dispensed into the test plates in duplicate and were covered and incubated for 1 h at 37□C. They were washed 5 times and 100 µl of AHSV specific monoclonal antibody (Mab peroxidase conjugate) was added and incubated for 30 min at 37^{o}C. Presence or absence of labeled Mab can be detected by adding the substrate which in the presence of peroxidase will develop a colorimetric reaction. The reaction was stopped by the addition of a stop solution and the plates were read on a MultiSkan® spectrophotometer ELISA plate reader at 450 nm wavelength. Serum end-point titre was expressed as the highest dilution producing 50% inhibition, Percent Inhibition (PI) of ≥ 50% is classified as positive for AHSV antibodies, whereas PI = < 50% were considered negative for AHSV antibody.

3.6 Detection of Antigens to Viral Infections

3.6.1 Bovine viral diarrhoea virus detection by reverse transcriptase-polymerase chain reaction

The extraction of viral RNA was carried out at Viral Research Division, NVRI Vom, Nigeria using RNeasy kits (QIAGEN, West Sussex UK). Briefly, spin protocol for purification of viral RNA was employed. The extracted viral RNA was kept at -20˚C until use. PCR analysis was carried out by adding 5.0 µl of the template RNA to the 20.0 µl of the master mix in a one-step RT-PCR. The following thermal profile was used: 50°C µl for 30 min 95°C for 15 min; 35 cycles of 95°C for 30 sec; 55°C for 30 seconds; and 72°C for 1 min; followed by final extension of 72°C for 10 min and 4°C ∞. The PCR products were analysed by electrophoresis on a 1.5% agarose-Tris-borate-EDTA gel containing 0.5µg/ml ethidium bromide. DNA weight markers (GeneRuler 300 bp DNA Ladder plus, Ready-To-Use; Fermentas Inc., Hanover, MD, USA) were visualized alongside the samples to facilitate product identification and quantification.

3.6.2 African horse sickness virus detection by reverse transcriptase- polymerase chain reaction

Samples were processed and packaged according to international standard for the transportation of infectious materials affecting animals (UN 2900) and shipped to the Equine Research Centre, Ondersteeport Veterinary Research Institute South Africa. Real time Polymerase chain reaction (RT-PCR) was performed according to standard procedure (Maree and Paweska, 2005). Total RNA from blood and tissue samples were extracted from 140 µl of the supernatant using RNeasy kits (QIAGEN, West Sussex UK) according to manufacturer's instructions. Denaturation of extracted RNA was carried out prior to the RT-PCR procedure. The sequences of the PCR primers used were 5'-GTT-AAA-ATT-CGG-TTA-GGA-TG-3', which corresponds to the messenger

RNA polarity, and 5'-GTA-AGT-GTA-TTC-GGT-ATT-G-3', which is complementary to the messenger RNA polarity. All the components required for the reverse transcription and PCR are included in the reaction tube containing the denatured RNA. A one-step RT-PCR was carried out by incubating in a thermocycler as follows: 45 min to 1 h at 37–55°C, 5–10 minutes at 95°C, then 40 cycles of: 94–95°C for 1 min, 55°C for 1–1.5 minutes, 70–72°C for 2–2.5 minutes, followed by a final extension step of 7–8 minutes at 70–72°C. Analysis of the PCR products was carried out by agarose gel electrophoresis.

3.7 Detection of Haemo, Endo and Ecto Parasites

3.7.1 Haematocrit centrifugation techniques for detection of haemoparasites

Haematocrit centrifugation techniques (HCT) were performed as described by Soulsby (1982). Blood from each sample was introduced into a plain glass microhaematocrit tube, one end of the tube was sealed using molten candle wax or plasticin, and the tubes were spun for 5 min at 1200 g in a Microhaematocrit centrifuge (Hawksley, England). Packed Cell Volume was measured using a haematocrit reader (Hawksley, England) and the buffy coat examined for motile blood parasites on a Nikon microscope (low power objective (X10) eye piece.

3.7.2 Thin blood film staining technique for detection of haemoparasites

A thin film was prepared as described by Soulsby (1982). A drop of blood was placed in the centre of a grease-free microscope slide. The drop of blood was then quickly spread with a glass spreader held at an angle of 30□ to obtain a thin film with a smooth tail end. It was air dried, fixed in methanol for 2–3 min, stained in 5% Giemsa stain with added Azur II (2 g/l of undiluted stain) for 30 min, and rinsed in buffered water. The

smears were examined at ×1000 magnification (oil immersion) on a Nikon microscope. At least 50 fields were searched per slide. Presence of hemoparasites was recorded and identification was carried out to genus and, where possible, species level (Woo and Cannon, 1991).

3.7.3 Thick blood film staining technique for detection of haemoparasites

A thick film staining technique was performed as described by Soulsby (1982). A drop of blood was placed in the centre of grease-free microscope slide and spread with a glass spreader held at a steep angle to achieve a thick smear covering an area of 15 mm. This was allowed to air dry in a horizontal position. Then giemsa stain was applied on the thick film and allowed to stand for 30 min. The stain was washed off using distilled water and allowed to air-dry. The stained thick smear was viewed under a light microscope using 100x oil immersion objective lens.

3.8 Gastro Intestinal Tract Parasites Detection Methods

3.8.1 Floatation technique for faecal parasites examination

Floatation technique was performed to identify endo parasites as described by Soulsby (1982). Five grams of faecal sample was placed into a clean universal test tube and 5 ml of saturated salt solution (Industrial Nacl) added and emulsified with an applicator stick. The mixture was sieved and filtered using double layered cheesecloth into an empty chromersal tube. More saturated salt solution was added until a meniscus was obtained. A clean grease-free microscope slide was placed on top of the test tube containing the filtered faecal suspension for 10-15 min. The slide was then removed by inversion and covered with a clean cover-slip avoiding air bubbles and over flooding then examined under Nikon microscope with X10 and X40 objective lens (Soulsby, 1982).

3.8.2 Sedimentation technique for faecal parasite examination

Sedimentation method of faecal examination was performed to identify endo parasites as described by Soulsby (1982). Five grams (5.0 g) of faecal sample collected from each animal was poured into a clean test tube and 5 mls of normal saline added and emulsified using a glass rod, and the faecal sample suspension was filtered through a double-layer of cheesecloth into another clean test tube. More normal saline was added to the filtrate and stirred well. It was then centrifuged at 1, 200 g for 3 min and the supernatant discarded. The procedure was repeated until the supernatant was very clear. The upper layer was discarded and the sediments examined under the Nikon microscope for ova or cyst (Soulsby, 1982).

3.8.3 Identification of ticks

The sampled ticks pulled off from each animal and stored in sterile loosely capped plastic universal sample bottles were washed with sterile water to remove excess particulate contamination from animal skin. They were mounted on slides and examined using a stereoscope microscope at a magnification of × 40, × 80 and × 100. Morphological identification of ticks was carried out as described by Walker, (2003).

3.9 Determination of Haematological and Biochemical Parameters

3.9.1 Determination of haematological parameters

Haematological parameters were measured using automated veterinary haematology analyzer (ABX ABC Vet, Horiba ABX SAS Montpellier, France). The output included total white blood cells counts, differential white blood cells counts (basophils,

eosinophil, neutrophils, lymphocytes and monocytes), total red blood cells counts, haemoglobin concentration and haematocrit values.

3.9.2 Determination of biochemical parameters

Biochemical parameters were measured using ace chemistry analyzer (Alfa Wassermann, New Jersey, USA) and these output included alanine amino transferase (ALT) aspartate amino transferase (AST), alkaline phosphotase (ALP), creatinine, glucose, albumin, total protein, urea, total bilirubin, direct bilirubin, sodium, and potassium.

3.10 Data Analyses

The obtained data were analysed using Graphpad Prism version 7. Results obtained from seroprevalence studies on RVFV, FMDV, AHSV and the detection of BVDV and AHSV antigens. Prevalence studies on haemo-, endo- and external parasites from both wildlife and domestic animals were summarized in tables and expressed as percentages. Also, values obtained from haematological and serum biochemical parameters were expressed as mean ± SEMs. Levels of association between positivity and sex, breed, age and animal species were derived using Chi-square. Values of $P \leq 0.05$ were regarded as statistically significantly different.

CHAPTER FOUR

RESULTS

4.1 Seroprevalence of Rift Valley Fever Virus in Wildlife and Domestic Animals in and around Yakari Game Reserve and Sumu Wildlife Park in Bauchi State, Nigeria

Total serum specimens from 300 cattle tested for RVFV neutralizing antibodies showed overall seroprevalence of 11.33% in cattle (Table 4.1) and classification of the cattle into sex and breed distribution showed that females had higher RVFV antibodies (13.6%) than males (6.38%). There was no significant difference between seropositivity rates among the different breeds of cattle ($P > 0.05$), however, White Fulani breed had the highest seroprevalence value of (12.6%) when compared to Red bororo (12.1 %) and Bokolo breeds (7.8 %) (Table 4.1). Sera from 106 wildlife tested for RVFV antibodies (Table 4.2) showed overall seroprevalence rate of 8.49%. Also, the results indicated that of the 7 wildlife species tested, detectable antibodies to RVFV were seen in Waterbuck, Wildebeest, Eland and Elephant. None of the 53 samples from Zebra and 1 sample each from Kudu and Hartbeest showed detectable antibodies to RVFV (Table 4.2). Comparison of the seroprevalence of RVFV at the wildlife-livestock interface level (Table 4.3) showed that seroprevalence of RVFV was higher in cattle than wildlife species tested, but with no significant difference ($P>0.05$). Risk factors for pathogen spill-over at the wild-domestic animal interface in YGR and SWP were observed (Appendix I)

Table 4.1: Seroprevalence of rift valley fever virus in cattle around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria

Variable	No. sampled (%)	No. positive (%)	X^2	P value	Odds ratio	CI at 95%
Breed						
Redbororo	58 (19.3)	7 (12.1)	0.745	0.677	0.339	0.624-0.730
Bokolo	51 (17)	4 (7.8)				
Whitefulani	191 (63.7)	23 (12.6)				
Overall	300 (100)	34 (11.3)				
Sex						
Male	94 (31.3)	6 (6.38)	3.196	0.076	0.441	0.071-0.082
Female	206 (68.7)	28 (13.6)				
Overall	300 (100)	34 (11.3)				

Table 4.2: Seroprevalence of rift valley fever virus in wildlife in Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Wildlife	No. Sampled (%)	No. positive (%)
Yankari game reserve		
Waterbuck	11 (10.4)	4 (36.0)
Elephant	4 (3.8)	1 (25.0)
Hartbeest	1 (0.9)	0
Sumu wildlife park		
Eland	24 (22.6)	3 (12.5)
Wildebeest	12 (11.3)	1 (8.3)
Zebra	53 (50.0)	0
Kudu	1 (0.9)	0
Overall	106 (100)	9 (8.5)

Table 4.3: Comparison of seroprevalence of rift valley fever virus in wildlife -cattle interface in Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Species	No sampled	No. positive	X^2	P value	Odds ratio	CI at 95%
Wildlife	106	9 (8.49)	0.668	0.414	0.726	0.336 - 1.569
Cattle	300	34 (11.33)				
Overall	406	43 (10.59)				

4.2 Seroprevalence of Foot and Mouth Disease Virus in Wildlife and Cattle in and around Yankari Game Reserve and Sumu Wildlife Park in Bauchi State, Nigeria

The result indicated that of the 300 sera collected from cattle, overall FMDV seroprevalence was 65.67 % (Table 4.4). Classification of the cattle into breed and sex (Table 4.4) showed that detectable antibodies to FMDV were significantly ($P < 0.05$) higher in white Fulani 157(82.2%) than in red bororo 23(39.7%) and bokolo 17(33.3%) respectively whereas in females 150(72.8%) out of the 206 had detectable antibodies to FMDV were significantly higher ($P <0.05$) than in males 47(50.0%).

Overall seroprevalence of FMDV in wildlife was 24.53% (Table 4.5). Detactable antibodies to FMDV were observed in waterbuck 2(28.6%), elephant 1(25.00 %), wildebeest 4(33.3%) and eland 6(25.0 %). None of the samples from Kudu and Hartbeest showed detectable antibodies to FMDV (Table 4.5).

Comparison of the overall seroprevalences of FMDV at the wildlife-cattle interface (Table 4.6) showed that detectable antibodies to FMDV were significantly higher ($P < 0.05$) in cattle 197(65.67%) than in wildlife 13(24%).

4.3 Serotypes of Foot and Mouth Disease Virus in some Wildlife in Yankari Game Reserve and Sumu Wildlife Park in Bauchi State, Nigeria

The results showed detectable antibodies to FMD serotype O, A, SAT 1 and SAT 2 in waterbuck, wildebeest and eland whereas antibodies to serotypes A and SAT 2 were dectected in elephant. Each of serotypes A and SAT 1 were shown to have highest reactors of 10(18.87%) whereas serotype O had the least reactors of 7(13.21%).

Table 4.4: Seroprevalence of foot and mouth disease virus in cattle around Yankari game reserve and sumu wildlife park in Bauchi State, Nigeria

Variable	No. sampled (%)	No +Ve (%)	X^2	P value	Odds ratio	CI at 95%
Breed						
Red Bororo	58 (19.3)	23 (39.7)	64.2	0.000		
Bokolo	51 (17.0)	17 (33.3)			0.544	0.241 - 1.225
White Fulani	191 (63.7)	157 (82.2)			5.019	2.550 -9.878
Overall	300 (100)	197 (65.7)				
Sex						
Male	94 (31.3)	47 (50)	14.9	0.000	0.373	0.225 -0.620
Female	206 (68.7)	150 (72.8)				
Overall	300 (100.0)	197 (65.)				

Table 4.5: Seroprevalence of foot and mouth disease virus in wildlife from Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Wildlife	No. sampled (%)	No. +ve (%)	X^2	P value	Odds ratio	CI at 95%
Yankari park						
Waterbuck	11 (10.4)	2 (18.2)	1.395	0.943	0.373	0.899-0.327
Elephant	4 (3.8)	1 (25.0)				
Hartbeest	1(0.9)	1 (0.0)				
Sumu park						
Eland	24 (22.6)	6 (25.0)				
Wildebeest	12 (11.3)	4 (33.3)				
Kudu	1 (0.9)	0 (0.0)				
Overall	53 (100)	13 (24.5)				

Table 4.6: Seroprevalence of foot and mouth disease at the wildlife-cattle interface in Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Species	No sampled	No +Ve (%)	X^2	P value	Odds ratio	CI at 95%
Wildlife	53	13 (24.53)	31.63	0.000	0.1699	0.087 - 0.332
Cattle	300	197 (65.67)				
Overall	353	210 (59.49)				

Table 4.7: Serotypes of foot and mouth disease virus in wildlife in Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Wildlife	No. tested	Foot and mouth disease virus serotypes			
		O	A	SAT 1	SAT 2
Waterbuck	11 (10.4)	2 (18.18)	2 (18.18)	3 (27.27)	1 (9.09)
Elephant	4 (3.8)	0	1 (25.0)	0	1 (25.0)
Hartbeest	1 (0.9)	0	0	0	0
Eland	24 (22.6)	3 (12.5)	6 (25.0)	4 (16.67)	4 (16.67)
Wildebeest	12 (11.3)	2 (16.67)	1 (8.33)	3 (25.0)	2 (16.67)
Kudu	1(0.9)	0	0	0	0
Overall	53 (100)	7 (13.21)	10 (18.87)	10 (18.87)	8 (15.09)

4.4 Prevalence of Bovine Viral Diarrhoea Virus in Cattle and Wildlife in YankariGame Reserve and Sumu Wildlife Park in Bauchi State, Nigeria

Results of BVDV antigen detection in cattle showed that out of the 300 cattle sampled, overall prevalence of 9.6% was observed (Table 4.8). Classification of cattle into breed and sex showed higher prevalence in whitefulani (10.47%) than red bororo (8.6%) and bokolo (7.84%) breeds respectively, whereas significantly ($P < 0.05$) higher prevalence was observed in females (12.13%) than males (4.25%)

Overall prevalence for BVDV in wildlife was 13.2% and distribution of the disease among them revealed prevalence rates of 18.2% in waterbuck, 8.3% in wildebeest, 12.5% eland and the only hartbeest examined was also positive. None of the sera samples collected from elephant and kudu were positive (Table 4.9). Comparison of prevalence of BVDV in wildlife and livestock (Table 4.10) showed higher prevalence in wildlife (13.2%) than in cattle (9.67%) ($P >0.05$). The Agarose gel electrophoresis of the PCR products from wildlife and cattle are presented in Appendix II.

Table 4.8: Prevalence of bovine viral diarhea virus in cattle around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria

Variable	No. Sampled (%)	No +Ve (%)	X^2	P value	Odds ratio	CI at 95%
Breed						
Red Bororo	58 (19.3)	5 (8.6)	0.409	Ref	0.432	0.799-0.881
Bokolo	51 (17.0)	4 (7.8)		0.902		0.229-3.559
White fulani	191 (63.7)	20 (10.5)		1.240		0.444- 3.464
Overall	300 (100)	29 (9.67)				
Sex						
Male	94 (31.3)	4 (4.3)	4.59	0.032	0.322	0.109 - 0.953
Female	206 (68.7)	25 (12.1)				
Overall	300 (100)	29 (9.67)				

Table 4.9: Prevalence of bovine viral diarrhoea virus in some wildlife from Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria

Wildlife	No. sampled	No. positive (%)
Yankari game reserve		
Waterbucks	11 (10.4)	2 (18.2)
Elephants	4 (3.8)	0 (0)
Hartbeest	1 (0.9)	1 (100)
Sumu wildlife park		
Elands	24 (22.6)	3 (12.5)
Wildebeests	12 (11.3)	1 (8.3)
Kudu	1 (0.9)	0 (0)
Overall	53 (100)	7 (13.2)

Table 4.10: Prevalence of bovine viral diarrhoea virus at the wildlife and cattle interface in Yankari game reserve and sumu wildlife park in Bauchi State, Nigeria.

Species	No. sampled	No. positive (%)	X^2	P value	Odds ratio	CI at 95%
Wildlife	53	7 (13.20)	0.616	0.432	1.422	0.588-3.438
Cattle	300	29 (9.67)				
Overall	353	36 (10.20)				

4.5 Seroprevalence of African Horse Sickness Virus in Wildlife and Horses in and around Yankari Game Reserve and Sumu Wildlife Park in Bauchi State, Nigeria

The results on detection AHSV antibodies indicated that of the 200 and 53 sera samples of horses and zebra screened for AHSV antibodies respectively, overall seroprevalence of 35.5% and 21.43% were observed in horses and zebra respectively (Table 4.11). Overall serprevalence of AHSV antibodies was significantly ($P < 0.05$) higher in horses than in zebra (Table 4.11). Classification of horses and zebras into sex for distribution of AHSV antibodies (Table 4.12) showed higher seroprevalences in both male horses (36.21%) and zebras (27.27%) than in female horses (26.67%) and zebras (10.0%). Also, classification of horses into age and breed distribution (Table 4.13) indicated that detectable antibodies to AHSV was higher in adult (36.67%) than the young (25.0%) horses examined and only the local breed and crossed breeds of horses showed detectable antibodies to AHSV at 37.23% and 9.0% respectively. No detectable antibody to AHSV was observed in the only one exotic breed of horse examined.

4.6 Molecular Detection of African Horse Sickness Virus Antigen

African horse sickness virus antigen was detected at RT-PCR from pooled tissues of lymphnodes, spleen and lungs of a zebra carcass following shipment of the samples to a reference laboratory at the Onderstepoort Veterinary Research Institute South Africa (Appendix III). No virus was isolated from the samples in cell culture as the samples were kept frozen at -80 ˚C for 7 months before shipment to the reference laboratory in South Africa.

Table 4.11: Seroprevalence of African horse sickness virus antibodies in horses and zebras from Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Animal	No. sampled	No. +Ve (%)	X2	P value	Odds ratio	CI at 95%
Horse	200	71 (35.5)	4.158	0.041	2.101	1.018- 4.337
Zebra	53	11 (20.8)				
Overall	253	82 (32.4)				

Table 4.12: Sex distribution of African horse sickness virus antibodies in horses and zebras in and around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Sex	No. sampled	No. postive (%)	X^2	P value	Odds ratio	CI at 95%
Horses						
Male	185	67 (36.2)	0.553	0.457	1.561	0.478- 5.098
Female	15	4 (26.7)				
Overall	200	71 (35.5)				
Zebras						
Male	33	9 (27.3)		0.175	3.375	0.648-17.57
Female	20	2 (10.0)				
Overall	53	11 (20.8)				

Table 4.13: Age and breed distribution of African horse sickness virus antibodies in horses around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Horses	No. sampled	No. postivie (%)	X^2	P value	Odds ratio	CI at 95%
Age						
Young	20	5 (25.0)	1.070	0.301	0.576	0.200- 1.657
Adult	180	66 (36.7)				
Overall	200	71 (35.5)				
Breed						
Local	188	70 (37.2)	1.070	0.301	0.576	0.200- 1.657
Cross	11	1 (9.09)				
Exotic	1	0 (0)				
Overall	200	71 (35.5)				

4.7 Haemoparasites and Gastrointestinal Parasites Identified in some Wildlife from Yankari Game Reserve and Sumu Wildlife Park in Bauchi State, Nigeria

Results on detection of haemoparasites in wildlife showed that in all, 106 wildlife were screened and 60(56.6%) were positive for haemoparasites. The identified haemoparasites were *Anaplasma marginale, Babesia bigemina, Babesia bovis, Babesia equi, Babesia cabali, Trypanosoma* spp and *Ehrlichia ruminantium* (Table 4.14). Morphological characteristics of some of these haemoparasites are presented in (Appendix IV). The result indicated that of the 4 wildlife species examined for haemoparasites in SWP, elands had the highest prevalence 19/24(79.2 %) followed by wildebeests 8/12 (66.7 %) and zebras 24/53(45.28 %), the only sample collected from kudu was also positive for *Babesia* spp (Table 4.15). Also, of the 3 wildlife species examined for haemoparasites in Yankari game reserve, waterbucks had the highest prevalence 5/11 (54.6%) followed by elephants 2/4 (50.0%) and the only hartbeest examined was also positive with *Ehrlichia ruminantium* (Table 4.15). The mean PCV for parasitized wildlife with one or more haemoparasites were shown to be significantly lower ($P < 0.05$) than non parasitized wildlife examined (Table 4.16). Samples parasitized by *Babesia* spp alone or in combination with *Anaplasma marginale* had the least PCV of 29.68 ± 1.06, followed by 30.56 ± 1.32 in samples parasitized by *Anaplasma marginale* alone or in combination with *Ehrlichia* spp, then 31.08±0.69 in samples parsitized by *Ehrlichia* spp alone or in combination with *Babesia* spp and 33.67 ± 8.01 in samples parasitized by *Trypanosoma* spp alone or in combination with *Anaplasma marginale* (Table 4.16). Results on gastrointestinal parasites of wildlife showed that of the 106 wildlife screened 63(59.4%) were positive with GIT parasites. A number of GIT parasites including two protozoans (*Balantidium coli* and *Eimeria* spp) and helminths from 18 genera including 11 nematodes (*Strongyle* type-egg, *Dictyocaulus, Cooperia, Strongyloides, Haemonchus, Trichuris, Trichomena,*

Oesophagostomum, Bunostomum, Ancylostoma and Triodontophorus), 4 Trematodes (*Fasciola, Schistosoma, Paramphistomum* and *Gastrodiscus*) and 3 Cestodes (*Anoplocephala, Taenia* and *Moniezie*) were identified from the faecal samples (Tables 4. 17). The results indicated that of the 4 wildlife from Sumu wildlife park examined for GIT parasites, prevalence was highest in wildebeests 8/12 (69.6%) followed by elands 14/24 (58.3%), zebras 29/53 (54.7%) and the only kudu examined was also positive with GIT parasite (Table 4.18). Also, of the 3 wildlife from Yankari game reserve examined for GIT parasites, prevalence was highest in waterbucks 7/11 (63.6%), followed by elephants 2/4 (50.0%) and the only hartbeest examined was also positive with GIT parasites (Table 4.18). Morphological characteristics of some of the GIT parasites are presented in Appendix VA and VB. During necropsy examinations on a waterbuck adult *Paramphistomum* spp and *Sarcocystic* cyst were identified in the rumen and muscle tissues respectively (Appendix VI).

4.8 Haemoparasite and Gastrointestinal Parasites of Cattle around Yankari Game Reserve and Sumu Wildlife Park in Bauchi State, Nigeria

Results of hamoparasites of cattle indicated that of the 300 cattle sampled, 127(42.33%) were positive for haemoparasites (Table 4.19). Classification of cattle into sex and breed showed that prevalence of haemoparasites was significantly ($P < 0.05$) higher in females (44.21%) than in males (33.04%) whereas higher prevalence was obtained in white fulani breed (44.21%) than red bororo (40.68%) and bokolo (37.25%) breeds of cattle ($P > 0.05$) (Table 4.19). Distribution of the identified haemoparasites in cattle (Table 4.20) showed high infection with *Anaplasma marginale* 59 (35.94%), followed by *Babesia bigemina* 42 (25.61%), *Trypanosoma* spp 33 (20.12%) and *Babesia bovis* 30 (18.29%). Also 18(6%) of the 300 cattle sampled had mixed infections with some of the haemoparasites (Table 4.20). Mean PCV of parasitized cattle with one or more

haemoparasites were shown to be significantly lower ($P < 0.05$) than non parasitized cattle (Table 4.21). Samples parasitized by *Trypanosoma* spp alone or in combination with *Babesia* spp had the lowest PCV of 25.78 ± 0.73, followed by 32.12 ± 0.57 in samples with *Babesia* spp alone or in combination *with Anaplasma marginale*, and 34.31 ± 0.85 in samples with *Anaplasma marginale* alone or in combination *Trypanosoma* spp (Table 4.21). Results on gastrointestinal parasites from cattle (Table 4.22) showed that 185 (61.67%) of the 300 cattle sampled were positive for gastrointestinal parasites. Classification of cattle into sex and breed (Table 4.22) showed that females had higher GIT parasites prevalence (62.62%) than males (59.57%) ($P > 0.05$), whereas white fulani breed had higher prevalence (63.68%) than bokolo 59.82% and red bororo (57.63%) breeds ($P > 0.05$). Distribution of the identified GIT parasites (Table 4.23) showed 2 protozoans (*Balantidium coli* and *Eimeria* spp) and helminths ova from 13 genera including seven nematodes (*Strongyle* type-egg, *Cooperia, Strongyloides, Haemonchus, Toxocara, Oesophagostomum* and *Bunostomum*), 3 trematodes (*Fasciola, Paramphistomum* and *Dicrocoelium*) and 2 cestodes (*Moniezia* and *Taenia* spp). Mixed infection was seen in 38 (12.67%) of the cattle population (Table 4.23).

Table 4.14: Distribution of types of haemoparasites identified in some wildlife from Yankari game reserve and Sumu wildlife park in Bauchi State Nigeria.

Haemoparasite	Waterbuck No. (%)	Elephant No. (%)	Hartbeest No. (%)	Zebra No. (%)	Wildebeest No (%)	Eland No. (%)	Kudu No. (%)
Anaplasma marginale	5 (45.5)	1 (25.0)	0 (0.0)	0 (0.0)	7 (58.3)	15 (62.5)	0 (0.0)
Babesia spp	2 (18.2)	0 (0.0)	0 (0.0)	0(0.0)	5 (41.7)	14 (58.3)	1 (100)
Trypanosoma spp	1 (9.1)	1 (25.0)	0 (0.0)	1 (1.9)	1 (8.3)	4 (16.7)	0 (0.0)
Babesia equi	0 (0.0)	0 (0.0)	0 (0.0)	14 (26.4)	0 (0.0)	0 (0.0)	0 (0.0)
Babesia cabale	0 (0.0)	0 (0.0)	0 (0.0)	9 (17.0)	0 (0.0)	0 (0.0)	0 (0.0)
Ehrlichia ruminantium	0(0.0)	0 (0.0)	1 (100.0)	0 (0.0)	5 (41.7)	7 (29.2)	0 (0.0)

Table 4.15: Prevalence of haemoparasites of some wildlife from the Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Wildlife	No. sampled	No. positive (%)
Sumu wildlife park		
Eland	24 (22.6)	19 (79.2)
Wildebeest	12 (11.3)	8 (66.7)
Zebra	53 (50.0)	24 (45.28)
Kudu	1 (0.9)	1 (100.0)
Yankari game reserve		
Waterbuck	11 (10.4)	5 (54.6)
Elephant	4 (3.8)	2 (50.0)
Hartbeest	1 (0.9)	1 (100.0)
Overall	106 (100)	60 (56.6)

Table 4.16: Haemoparasites status and significance on mean pack cell volume of wildlife in Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Haemoparasite status	No. animals (%)	Mean PCV±SEM	P-value	Mean difference	CI at 95%
No. parasites seen	47 (44.3)	38.68 ± 0.92			36.8-40.54
Ehrlichia spp alone or in combination with Babesia spp	13 (12.3)	31.08 ± 0.69	0.001	7.60	29.6-32.6
Babesia spp alone or in combination with *Anaplasma marginale*	25 (23.6)	29.68 ± 1.06	0.000	9.00	27.5- 31.9
Anaplasma marginale alone or in combination with *Ehrlichia* spp	18 (17.0)	30.56 ± 1.32	0.000	8.12	27.8-33.3
Trypanosoma spp alone or in combination with *Anaplasma marginale*	8 (7.6)	33.67 ± 8.01	0.611	5.01	27.8- 42.7

Table 4.17: Distribution of types of gastrointestinal parasites identified in some wildlife from Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Gastrointestinal parasites	Waterbuck No. (%)	Elephant No. (%)	Hartbeest No. (%)	Zebra No. (%)	Wildebeest No. (%)	Eland No. (%)	Kudu No. (%)
PROTOZOANS							
Balantidium spp	2 (18.2)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Eimeria spp	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	3 (12.5)	0 (0.0)
TREMATODES							
Fasciola spp	4 (36.4)	2 (50.0)	1(100.0)	9 (17.0)	7 (58.3	11 (45.8)	0 (0.0)
Paramphistomum spp	2 (18.2)	0 (0.0)	0 (0.0)	0 (0.0)	1 (8.3)	4 (16.2)	0 (0.0)
Schistosoma spp	2 (18.2)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Gastrodiscus spp	0 (0.0)	0 (0.0)	0 (0.0)	5 (9.4)	0 (0.0)	0 (0.0)	0 (0.0)
CESTODES							
Anoplacephala spp	0 (0.0)	0 (0.0)	0 (0.0)	4 (7.6)	0 (0.0)	0 (0.0)	0 (0.0)
Taenia spp	2(18.2)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Moniezia spp	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	2 (8.3)	0 (0.0)
NEMATODES							
Strongyle type egg	1 (9.1)	1 (25.0)	1 (100.0)	0 (0.0)	6 (50.0)	6 (25.0)	1 (100.0)
Strongylus spp	0 (0.0)	0 (0.0)	0 (0.0)	13 (24.5)	0 (0.0)	0 (0.0)	0 (0.0)
Ancylostoma spp	2 (18.2)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Haemonchus spp	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	2 (16.7)	3 (12.5)	0 (0.0)
Strongyloides spp	0 (0.0)	1 (25.0)	0 (0.0)	5 (9.4)	0 (0.0)	3 (12.5)	0 (0.0)
Dictyocaulus spp	1 (9.1)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Cooperia spp	1 (9.1)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Trichuris spp	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (4.1)	0 (0.0)
Oesophagostomum spp	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	2 (8.3)	0 (0.0)
Bunostomum spp	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	2 (8.3)	0 (0.0)
Trichomena spp	0 (0.0)	0 (0.0)	0 (0.0)	6 (11.3)	0 (0.0)	1 (4.1)	0 (0.0)

Table 4.18: Prevalence of gastrointestinal parasites of some wildlife from Sumu wildlife park and Yankari game reserve in Bauchi State, Nigeria.

Wildlife	No. sampled	No. positive (%)
Sumu wildlife park		
Zebra	53 (50.0)	29 (54.7)
Eland	24 (22.6)	14 (58.3)
Wildebeest	12 (11.3)	8 (66.7)
Kudu	1 (0.9)	1 (100.0)
Yankari game reserve		
Waterbuck	11 (10.4)	7 (63.6)
Elephant	4 (3.8)	3 (75.0)
Hartbeest	1(0.9)	1 (100.0)
Overall	106 (100)	63 (62.0)

Table 4.19: Prevalence of haemoparasites of cattle around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Variable	No. sampled	No. positive (%)	X^2	P value	Odds ratio	CI at 95%
Sex						
Male	94 (31.3)	32 (34.04)	3.855	0.049	0.603	0.363-1.001
Female	206 (68.7)	95 (46.12)				
Overall	300 (100.0)	127 (42.3)				
Breed						
White fulani	191 (63.7)	84 (44.21)	0.772	Ref		
Red Bororo	58 (19.3)	24 (40.68)		0.899		0.496-1.631
Bokolo	51 (17.0)	19 (37.25)		0.756		0.401-1.428
Overall	300 (100.0)	127 (42.3)				

Table 4.20: Distribution of types of haemoparasites identified in cattle, donkeys and horses around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Haemoparasites	Cattle. No. (%)	Donkeys. No. (%)	Horses. No. (%)
Anaplasma marginale	59 (19.67)	0 (0.0)	0 (0.0)
Babesia bigemina	42 (14.0)	0 (0.0)	0 (0.0)
Babesia bovis	30 (10.0)	0 (0.0)	0 (0.0)
Babesia caballi	0 (0.0)	36 (18.0)	35 (17.5)
Babesia equi	0 (0.0)	29 (14.5)	20 (10.0)
Trypanosoma spp	33 (11.0)	18 (9.0)	0 (0.0)
Mixed infections	18 (6.0)	9 (4.5)	5 (2.5)

Table 4.21: Haemoparasites status and significance on mean pack cell volume of cattle around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Haemoparasite status	No. of animals (%)	Mean PCV±SEM	P-value	Mean difference	CI at 95%
No. parasites seen	177 (59.0)	39.38 ± 0.19			39.0-39.8
Trypanosoma spp (alone or in combination with *Babesia* spp)	32 (10.7)	25.78 ± 0.73	0.000	13.6	24.3-27.9
Babesia spp (alone or in combination *with Anaplasma marginale*)	62 (20.7)	32.12 ± 0.57	0.000	7.25	31.0-33.3
Anaplasma marginale (alone or in combination *Trypanosoma* spp)	29 (9.7)	34.31 ± 0.85	0.000	5.07	32.6-36.1

Table 4.22: Prevalence of gastrointestinal parasites of cattle around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Variable	No. sampled	No. positive (%)	X^2	P value	Odds ratio	CI at 95%
Sex						
Male	94 (31.3)	56 (59.57)	0.254	0.615	0.880	0.534-1.450
Female	206 (68.7)	129 (62.6)				
Overall	300 (100.0)	185 (61.7)				
Breed						
White Fulani	191 (63.7)	121 (63.7)	0.631	0.729	Ref	
Red Bororo	58 (19.3)	34 (57.63)			0.820	0.450- 1.493
Bokolo	51 (17.0)	30 (58.82)			0.826	0.440- 1.553
Overall	300 (100.0)	185 (61.7)				

Table 4.23: Distribution of types of gastrointestinal parasites identified in cattle, donkeys and horses around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Gastrointestinal Parasites	Cattle No. (%)	Donkeys No. (%)	Horses No. (%)
PROTOZOANS			
Balantidium coli	5 (1.6)	0 (0.0)	0 (0.0)
Eimeria spp	25 (8.3)	0 (0.0)	0 (0.0)
TREMATODES			
Fasciola spp	118 (39.3)	29 (14.5)	6 (3.0)
Paramphistomum spp	17 (5.6)	0 (0.0)	0 (0.0)
Gastrodiscus spp	0	11 (5.5)	6 (3.0)
Dicrocoelium spp	7 (2.3)	0 (0.0)	0 (0.0)
CESTODES			
Taenia spp	16 (5.3)	0 (0.0)	0 (0.0)
Moniezia spp	14 (4.6)	0 (0.0)	0 (0.0)
Anoplacephala spp	0 (0.0)	9 (4.5)	5 (2.5)
Stelizia spp	0 (0.0)	7 (3.5)	0 (0.0)
NEMATODES			
Strongyle type egg	46 (15.3)	0 (0.0)	0 (0.0)
Strongylus spp	0 (0.0)	44 (22.0)	24 (12.0)
Haemonchus spp	10 (3.3)	0 (0.0)	0 (0.0)
Strongyloides spp	22 (7.3)	32 (16.0)	11 (5.5)
Cooperia spp	8 (2.6)	0 (0.0)	0 (0.0)
Toxocara spp	12 (4.0)	0 (0.0)	0 (0.0)
Bunostomum spp	23 (7.7)	0 (0.0)	0 (0.0)
Oesophagostomum spp	37 (12.3)	0 (0.0)	0 (0.0)
Trichomena spp	0 (0.0)	30 (15.0)	9 (4.5)
Triodontophorus spp	0 (0.0)	8 (4.0)	5 (2.5)
Gastrodiscus spp	0 (0.0)	11 (5.5)	6 (3.0)
Mixed infection	0 (0.0)	42 (21.0)	13 (6.5)

4.9 Haemoparasite and Gastrointestinal Parasites of Donkeys around Yankari Game Reserve and Sumu Wildlife Park in Bauchi State, Nigeria

The results on hamoparasites of donkeys (Table 4.24) indicated that 63 (31.5%) of the 200 donkeys sampled were positive for haemoparasites with females having higher prevalence of 36.96% than males with 29.87% ($P > 0.05$). Distribution of the identified haemoparasites (Table 4.20) showed high infection with *Babesia cabali* 41 (20.5%), *Babesia equi* 29 (14.5%) and *Trypanosoma* spp 13 (6.5%), mixed infections with haemoparasites were observed in 20 (10%) of the donkeys sampled. The mean PCV of parasitized donkeys with one or more haemoparasites were shown to be significantly lower ($P < 0.05$) than non parasitized donkeys (Table 4.25). Samples parasitized by *Babesia* spp alone or in combination with *Trypanosoma* spp had the lowest Mean PCV of 31.00 ± 0.81 than non parasitized donkeys 37.85 ± 0.25 (Table 4.25). Overall prevalence of GIT parasites of donkeys was 62% with females having the highest prevalence 36.96% than the males 29.87% (Table 4.26) ($P >0.05$). Distribution of the identified ova of GIT parasites (Table 4.23) showed that they were infected with helminths genera including 4 nematodes (*Strongyle* type eggs, *Strongyloides*, *Trichomena* and *Triodontophorus* species) and 2 Trematodes (*Fasciola* and *Gastrodiscus*) and 2 Cestodes (*Stilezia* and *Anoplocephala*).Mixed infections with the GIT parasites were seen in 42 (21%) of the donkeys sampled (Table 4.23).

Table 4.24: Prevalence of haemoparasites of donkeys around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Variable	No. sampled (%)	No.positive (%)	X^2	P value	Odds ratio	CI at 95%
Sex						
Male	154 (77.0)	46 (29.87)	0.824	0.364	0.727	0.364-1.450
Female	46 (23.0)	17 (36.96)				
Overall	200 (100.0)	63 (31.50)				

Table 4.25: Haemoparasites status and significance on mean pack cell volume of donkeys and horses around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Animal	Haemoparasite status	No. of animals (%)	Mean PCV±SEM	P-value	Mean difference	CI at 95%
Donkeys						
	No parasite seen	137 (65.5)	37.85 ± 0.25			37.4-38.4
	Babesia spp (alone or in combination with *Trypanosoma* spp)	63 (31.5)	31.00 ± 0.81	0.001	6.85	29.4-32.6
Horses						
	No parasite seen	151 (75.5)	37.72 ± 0.25			37.2-38.2
	Babesia equi (alone or in combination with *Babesia cabali*)	49 (24.5)	31.29 ± 0.65	0.000	6.43	30.0-32.6

Table 4.26: Prevalence of gastrointestinal parasites of donkeys around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Variable	No. sampled	No. positive (%)	X^2	P value	Odds ratio	CI at 95%
Sex						
Male	154	97 (63.0)	0.277	0.599	1.198	0.612-2.345
Female	46	27 (58.7)				
Overall	200	124 (62.0)				

4.10 Haemoparasites and Gastrointestinal Parasites of Horses around Yankari Game Reserve and Sumu Wildlife Park in Bauchi State, Nigeria

The results on hamoparasites of horses (Table 4.27) indicated that of the 200 blood samples collected overall prevalence of 24.5% was observed. Classification of the horses according to age, sex and breed (Table 4.27) showed higher prevalences of haemoparasites in the young (27.3%) than adult (24.2%), in males (25.4%) than females (13.3%) and also in the crossed breed 36.4% than local breed (25.5%), the only exotic breed examined was negative for haemoparasite. Distribution of the identified haemoparasites (Table 4.20) showed high infection with *Babesia cabali* 35 (17.5%), *Babesia equi* 20 (10.0%) and 5 (2.5%) of the horses had mixed infections with the identified haemoparasites. Mean PCV for parasitized horses with one or two haemoparasites were significantly lower ($P < 0.05$) than non parasitized horses (Table 4.25). Samples parasitized by *Babesia equi* alone or in combination with *Babesia cabali* had the least PCV of 31.29 ± 0.65 than in non parasitized horses 37.72 ± 0.25 (Table 4.25). Overall prevalence of 27.0% was observed for GIT parasites of horses (Table 4.28). Classification of horses according to age, sex and breed (Table 4.28) showed higher prevalence of GIT parasites in the young horses (36.4%) than adults (25.8%), in the males 51 (27.6%) than females 3 (20.0%) and in the local breed 51 (27.1%) than crossed breed 2 (18.1%). The only exotic breed examined was positive with GIT parasite. No significant difference ($P>0.05$) based on age, sex and breed distribution of GIT parasites was found in horses. On the distribution of the identified GIT parasites in horses (Table 4. 23) the results indicated that they were infected with various helminths genera including 4 nematodes (*Strongylus, Strongyloides, Trichomena* and *Triodontophorus*) and 2 Trematodes (*Fasciola* and *Gastrodiscus*) and 1 Cestode (*Anoplocephala*). Mixed infections with the GIT parasites were seen in 13 (6.5%) of the horses sampled (Table 4.23).

Table 4.27: Prevalence of haemoparasites of horses around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Variable	No. sampled (%)	No. positive (%)	X^2	P value	Odds ratio	CI at 95%
Age						
Young	22 (11)	6 (27.3)	0.103	0.749	1.177	0.433-3.198
Adult	178 (89)	43 (24.2)				
Overall	200 (100)	49 (24.5)				
Sex						
Male	185 (92.5)	47 (25.4)	5.85	0.368	2.214	0.482-10.18
Female	15 (7.5)	2 (13.3)				
Overall	200 (100)	49 (24.5)				
Breed						
Local	188 (94)	45 (23.9)	10.07	0.100	2.411	0.055-0.142
Cross	11 (5.5)	4 (36.4)				
Exotic	1 (0.5)	0 (0)				
Overall	200 (100)	49 (24.5)				

Table 4.28: Prevalence of gastrointestinal parasites of horses around Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

	No. sampled (%)	No. positive (%)	X^2	P value	Odds ratio	CI at 95%
Age						
Young	22 (11.0)	8 (36.4)	1.100	0.294	1.640	0.6460-4.162
Adult	178 (89.0)	46 (25.8)				
Total	200 (100.0)	54 (27.0)				
Sex						
Male	185 (92.5)	51 (27.6)	1.390	0.238	0.526	0.178- 1.554
Female	15 (7.5)	3 (20.0)				
Total	200 (100.0)	54 (27.0)				
Breed						
Local	188 (94.0)	51 (27.1)	4.810	0.0903	1.423	0.058-0.142
Cross	11 (5.5)	2 (18.2)				
Exotic	1 (0.5)	1 (100.0)				
Total	200 (100.0)	54 (27.0)				

4.11 Ticks Infestation of Some Wildlife in Yankari Game Reserve and Sumu Wildlife Park in Bauchi State, Nigeria

The results on ticks infestation showed that wildlife from Sumu wildlife park including zebras and elands were the most affected having 4 different genera of ticks including *Amblyomma, Boophilus, Hyalomma* and *Rhipiciphalus* followed by wildebeest and kudu with 3 genera each (*Amblyoma, Hyalomma* and *Rhipiciphalus*) and (*Amblyomma, Rhipiciphalus* and *Boophilus*) respectively, whereas wildlife from Yankari game reserve which included elephants, waterbuck and hartebeest were both infested with *Amblyomma* and *Boophilus* genera (Table 4.29). Tick infestations were shown to be very high especially in zebra, eland, kudu and wildebeest and mostly around the perineum, ear, vulva and anal region and in some cases generalized (Appendix VIII).

4.12 Mean Haematological Values of some Wildlife from Yankari Game Reserve and Sumu Wildlife Park in Bauchi State, Nigeria

The results on minimum and maximum values, arithmetic means and standard error of means of haematologic parameters (PCV, HB, WBC, RBC, MCV, MCH, MCHC, Neutrophil and Lymphocytes) for elephant, waterbuck, zebra, eland and wildebeest are presented in Tables (4.30- 4.34). The values vary among the wildlife species. Sex distribution of mean haematological values in wildlife (Table 4.35) showed that they are within same ranges with no signifant differences. However, in zebras mean RBC values were significantly higher ($P < 0.05$) in males than females; in elands, mean Hb and RBC values were significantly higher in females than males whereas mean WBC values were significantly higher ($P < 0.05$) in males than females; in wildebeest, mean WBC and Neutrophils values were significantly higher ($P < 0.05$) in males than females and in waterbucks and elephants no significant differences on haematological values were observed between sexes.

4.13: Mean Biochemical Values of some Wildlife from Yankari Game Reserve and Sumu Wildlife Park in Bauchi State, Nigeria

The results on minimum and maximum values, arithmetic means and standard error of means of biochemical values (ALT, AST, ALP, CREA,UREA, ALB, total protein, total bilirubin, direct biluribin, potassium, Sodium, Glucose and LDH) for some wildlife are as indicated in (Tables 4.36- 4.40). The values vary among the apparently wildlife species. Sex distribution of mean biochemical values in wildlife (Table 4.41- 4.43) showed that mean ALT, Creatinin, Albumin, Total protein and Direct bilirubin values were significantly higher ($P < 0.05$) in females than males whereas mean ALP, Urea, Potasium, Glucose and LDH values were significantly higher ($P < 0.05$) in males than females. No significant differences were observed between male and female elephants. In zebras, mean AST, ALT, Creatinin, Urea, Albumin and Potassium values were significantly higher ($P < 0.05$) in males than females whereas only mean ALT and LDH values were significantly higher ($P < 0.05$) in females than males. In elands, mean ALT, AST, ALP, Total protein, Total bilirubin and LDH values were significantly higher ($P < 0.05$) in males than females whereas mean Creatinin, Direct bilirubin, Potassium and Sodium values were significantly higher ($P < 0.05$) in females than males. In wildebeest, mean ALT, AST, ALP, Creatinin, Urea, Albumin, Total protein and LDH values were significantly higher in males than females wereas only Potassium, Sodium and Glucose values were significantly higher in females than males.

Table 4.29: Distribution of ticks collected from some wildlife in Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Wildlife	No. examined	Tick species identified
Zebra	53	*Amblyoma variegatum*
		Hyalomma spp
		Boophilus spp
		Rhipiciphalus spp
Eland	24	*Amblyoma variegatum*
		Hyaloma spp
		Boophilus spp
		Rhipiciphelus spp
Wildebeest	12	*Amblyoma variegatum*
		Hyaloma spp
		Rhipiciphalus spp
Kudu	1	*Amblyoma variegatum*
		Boophilus spp
		Rhipiciphalus spp
Elephant	4	*Amblyoma variegatum*
		Boophilus decolaratus
Waterbuck	11	*Amblyoma variegatum*
		Boophilus decolaratus
Heartbeest	1	*Amblyoma variegatum*

Table 4.30: Mean haematological values of elephants from Yankari game reserve in Bauchi State, Nigeria.

Parameter	No. examined	Mean	S.E.M	Min	Max
PCV (%)	4	52.50	4.291	42.00	60.00
Hb (g/dL)	4	17.68	1.274	14.00	19.70
RBC (x10^3/ul)	4	4.100	0.2041	3.800	4.700
WBC(x 10^3/ul)	4	6.125	0.8538	4.500	7.600
MCV (fl/cell)	4	126.6	9.152	105.3	150.00
MCH (pg/cell)	4	42.45	2.991	34.21	47.50
MCHC (g/dl))	4	33.58	1.112	31.67	36.74
Neutrophil (%)	4	56.75	6.498	42.00	70.00
Lymphocyte (%)	4	36.50	7.182	28.00	58.00

Table 4.31: Mean haematological values of waterbucks from Yankari game reserve in Bauchi State, Nigeria.

Parameter	No. examined	Mean	S.E.M	Min	Max
PCV (%)	8	38.38	1.782	30.00	45.00
Hb (g/dL)	8	12.51	0.5142	9.800	14.30
RBC ($x10^3$/ul)	8	8.188	0.5971	6.200	12.00
WBC(x 10^3/ul)	8	8.850	0.2478	8.000	9.800
MCV (fl/cell)	8	46.83	3.012	32.50	58.07
MCH (pg/cell)	8	15.66	0.9423	10.58	19.36
MCHC (g/dl))	8	32.66	0.2288	31.78	33.33
Neutrophil (%)	8	29.13	1.757	19.00	35.00
Lymphocyte (%)	8	54.75	2.671	43.00	63.00

Table 4.32: Mean haematological values of elands from Sumu wildlife park in Bauchi State, Nigeria.

Parameter	No. examined	Mean	S.E.M	Min	Max
PCV (%)	12	31.25	0.845	28.00	36.00
Hb (g/dL)	12	10.23	0.298	9.100	11.90
RBC (x10^3/ul)	12	4.575	0.096	3.900	5.100
WBC(x 10^3/ul)	12	3.825	0.071	3.500	4.200
MCV (fl/cell)	12	68.44	1.792	62.20	84.62
MCH (pg/cell)	12	22.40	0.603	20.22	27.69
MCHC (g/dl))	12	32.74	0.185	31.67	33.87
Neutrophil (%)	12	42.39	1.383	33.10	49.00
Lymphocyte (%)	12	60.00	2.041	43.00	69.00

Table 4.33: Mean haematological values of wildebeest from Sumu wildlife park in Bauchi State, Nigeria.

Parameter	No. examined	Mean	S.E.M	Min	Max
PCV (%)	8	36.38	1.558	33.00	45.00
Hb (g/dL)	8	11.85	0.668	10.20	15.00
RBC (x10^3/ul)	8	10.54	6.640	3.400	5.70
WBC(x 10^3/ul)	8	3.713	0.295	2.900	5.10
MCV (fl/cell)	8	89.67	4.444	78.26	113.9
MCH (pg/cell)	8	29.98	2.075	24.78	40.00
MCHC (g/dl))	8	32.17	0.673	30.90	35.29
Neutrophil (%)	8	34.13	2.133	28.00	42.00
Lymphocyte (%)	8	55.50	2.619	47.00	67.00

Table 4.34: Mean haematological values of zebras from Sumu wildlife park in Bauchi State, Nigeria.

Parameter	No. examined	Mean	S.E.M	Min	Max
PCV (%)	15	32.93	1.803	24.00	46.00
Hb (g/dL)	15	10.97	0.592	8.000	15.40
RBC (x10^3/ul)	15	3.820	0.149	2.400	4.900
WBC(x 10^3/ul)	15	4.333	0.270	3.200	6.000
MCV (fl/cell)	15	90.76	6.376	70.59	160.0
MCH (pg/cell)	15	28.79	1.383	23.33	42.78
MCHC (g/dl))	15	33.28	0.099	32.44	33.80
Neutrophil (%)	15	51.80	4.347	30.00	80.00
Lymphocyte (%)	15	46.67	5.085	20.00	90.00

Table 4.35: Sex distribution of mean haematological values of some wildlife from Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Wildlife	Sex	No. examined	PCV	HB	RBC	WBC	Neutrophil	Lymphocyte
Zebra	M	8	35.38 ± 2.112	11.71 ± 0.692	4.163 ± 0.154*	4.488 ± 0.396	47.50 ± 6.059	50.38 ± 8.479
	F	7	30.14 ± 2.807	10.11 ± 0.941	3.429 ± 0.177*	4.157 ± 0.380	56.71 ± 6.163	42.43 ± 5.273
Eland	M	7	29.83 ± 0.792	9.650 ± 0.249*	4.383 ± 0.101*	3.983 ± 0.095*	44.60 ± 1.738	60.17 ± 2.120
	F	5	32.67 ± 1.308	10.82 ± 0.440*	4.767 ± 0.126*	3.667 ± 0.056*	40.18 ± 1.857	59.83 ± 3.719
Wildebeest	M	4	39.50 ± 2.179*	13.05 ± 0.961	4.625 ± 0.429	4.325 ± 0.386*	39.00 ± 2.121*	57.00 ± 4.243
	F	4	33.25 ± 0.250*	10.65 ± 0.450	3.625 ± 0.103	3.100 ± 0.082*	29.25 ± 0.947*	54.00 ± 3.536
Waterbuck	M	4	41.50 ± 2.179	13.40 ± 0.558	7.725 ± 0.522	9.275 ± 0.377	29.75 ± 1.652	56.00 ± 4.601
	F	4	35.25 ± 1.887	11.63 ± 0.630	8.650 ± 1.118	8.425 ± 0.155	28.50 ± 3.379	53.50 ± 3.329
Elephant	M	3	50.33 ± 5.24	17.00 ± 1.53	3.900 ± 0.058	6.667 ± 0.933	61.67 ± 6.01	29.33 ± 0.67
	F	1	59	19.7	4.7	4.5	42	58

Keys - * Values Significantly different at P<0.05, PCV-Pack Cell Volume, Hb-Haemoglobin concentration, RBC-Red blood cell count, WBC-White blood cell cout.

Table 4.36: Mean biochemical values of elephants from Yankari game reserve in Bauchi State, Nigeria.

Parameter	No. examined	Mean	S.E.M	Min	Max
Alanine amino transferase (µ/L)	4	7.250	2.428	1.00	12.00
Aspartate amino transferase (µ /L)	4	24.25	8.440	10.0	47.00
Alkaline phosphatase (µ /L)	4	15.51	7.277	1.84	35.88
Lactate dehydrogenase (µ /L)	4	146.4	38.69	77.00	256.5
Creatinin (µMol/L)	4	7.585	1.814	3.20	12.04
Urea (mMol/L)	4	3.600	0.8997	1.07	5.330
Albumin (g/L)	4	36.51	5.016	27.15	50.00
Total protein (g/L)	4	93.83	4.814	86.00	106.4
Total bilirubin (mg/dl)	4	2.728	0.5950	1.63	4.420
Direct bilirubin (mg/dl)	4	3.023	0.6264	1.94	4.830
Potassium (MEq/L)	4	21.34	3.774	12.20	29.90
Sodium (MEq/L)	4	137.1	4.157	125.4	145.0
Glucose (mm/L)	4	1.505	0.5504	0.4200	2.910

Table 4.37: Mean biochemical values of waterbucks from Yankari game reserve in Bauchi State, Nigeria.

Parameter	No. examined	Mean	S.E.M	Min	Max
Alanine amino transferase (µ/L)	8	14.25	2.094	8.00	22.00
Aspartate amino transferase (µ/L)	8	56.63	5.913	34.0	79.00
Alkaline phosphatase (µ/L)	8	22.72	2.201	13.8	31.00
Lactate dehydrogenase (µ/L)	8	82.87	17.60	38.12	176.0
Creatinin (µMol/L)	8	39.52	11.16	8.00	88.10
Urea (mMol/L)	8	67.56	8.225	37.07	98.00
Albumin (g/L)	8	159.5	20.48	103.0	252.3
Total protein (g/L)	8	97.30	7.782	58.97	125.3
Total bilirubin (mg/dl)	8	8.855	0.8928	5.680	12.68
Direct bilirubin (mg/dl)	8	6.800	0.6608	4.080	9.660
Potassium (MEq/L)	8	146.9	19.60	98.44	221.0
Sodium (MEq/L)	8	136.3	4.651	106.4	149.8
Glucose (mm/L)	8	9.011	1.274	3.670	12.60

Table 4.38: Mean biochemical values of elands from Sumu wildlife park in Bauchi State, Nigeria.

Parameter	No. examined	Mean	S.E.M	Min	Max
Alanine amino transferase (µ/L)	12	24.50	2.407	11.00	38.00
Aspartate amino transferase (µ/L)	12	60.25	4.000	40.00	83.00
Alkaline phosphatase (µ/L)	12	148.6	26.38	46.00	339.5
Lactate dehydrogenase (µ/L)	12	195.8	21.49	101.2	296.7
Creatinin (µMol/L)	12	52.38	8.694	14.60	88.10
Urea (mMol/L)	12	19.87	3.084	2.130	33.83
Albumin (g/L)	12	16.64	2.722	4.930	30.18
Total protein (g/L)	12	94.52	11.09	36.80	138.0
Total bilirubin (mg/dl)	12	15.32	2.507	2.560	27.83
Direct bilirubin (mg/dl)	12	10.75	1.344	5.890	19.00
Potassium (MEq/L)	12	23.57	2.214	12.00	33.00
Sodium (MEq/L)	12	137.0	4.942	110.0	162.4
Glucose (mm/L)	12	5.668	0.812	1.170	11.10

Table 4.39: Mean biochemical values of wildebeests from Sumu wildlife park in Bauchi State, Nigeria.

Parameter	No. examined	Mean	S.E.M	Min	Max
Alanine amino transferase (µ/L)	8	12.40	0.814	9.000	15.50
Aspartate amino transferase (µ/L)	8	32.69	0.918	28.80	36.00
Alkaline phosphatase (µ/L)	8	8.939	1.202	4.140	13.82
Lactate dehydrogenase (µ/L)	8	31.06	2.404	20.95	40.06
Creatinin (µMol/L)	8	16.19	3.055	1.600	27.23
Urea (mMol/L)	8	75.62	4.968	59.00	98.00
Albumin (g/L)	8	210.6	9.369	179.8	246.5
Total protein (g/L)	8	65.95	5.210	49.90	88.00
Total bilirubin (mg/dl)	8	1.629	0.098	1.080	1.980
Direct bilirubin (mg/dl)	8	3.629	0.157	3.000	4.110
Potassium (MEq/L)	8	22.39	3.003	10.17	32.60
Sodium (MEq/L)	8	106.7	6.716	77.80	128.1
Glucose (mm/L)	8	4.525	0.655	2.980	8.400

Table 4.40: Mean biochemical values of zebras from Sumu wildlife park in Bauchi State, Nigeria.

Parameter	No. examined	Mean	S.E.M	Min	Max
Alanine amino transferase (µ/L)	15	5.867	0.435	4.000	8.00
Aspartate amino transferase (µ/L)	15	21.60	1.917	10.00	30.00
Alkaline phosphatase (µ/L)	15	28.14	3.725	6.900	59.04
Lactate dehydrogenase (µ/L)	15	106.3	11.84	36.18	192.4
Creatinin (µMol/L)	15	47.56	6.828	8.010	79.81
Urea (mMol/L)	15	40.92	5.785	15.47	77.33
Albumin (g/L)	15	32.96	2.883	20.00	48.42
Total protein (g/L)	15	95.38	5.273	72.79	134.0
Total bilirubin (mg/dl)	15	1.493	0.188	0.590	2.650
Direct bilirubin (mg/dl)	15	2.204	0.170	1.310	3.830
Potassium (MEq/L)	15	27.99	2.811	13.15	42.15
Sodium (MEq/L)	15	137.6	2.832	105.5	146.1
Glucose (mm/L)	15	1.673	0.093	0.930	2.000

Table 4.41: Sex distribution of mean biochemical enzymes values of some wildlife from Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Wildlife	Sex	No. examined	Alanine amino transferase (µ/L)	Aspartate amino transferase (µ/L)	Alkaline phosphatise (µ/L)	Lactate dehydrogenase (µ/L)
Zebra	M	8	4.667 ± 0.288*	25.56 ± 1.608*	30.48 ± 4.894*	77.33 ± 7.060*
	F	7	7.667 ± 0.211*	15.67 ± 2.789*	24.62 ± 5.959*	149.8 ± 15.23*
Eland	M	7	29.86 ± 2.324*	69.71 ± 3.372*	193.9 ± 34.39*	253.5 ± 10.49*
	F	5	17.00 ± 1.673*	47.00 ± 2.55*	85.24 ± 19.71*	114.9 ± 3.552*
Wildebeest	M	4	14.72 ± 0.602*	35.18 ± 0.8803*	11.98 ± 0.531*	37.06 ± 1.531*
	F	4	10.50 ± 0.597*	30.95 ± 0.754*	6.068 ± 0.802*	25.49 ± 1.838*
Waterbuck	M	4	8.33 ± 0.333*	45.0 ± 3.055	28.59 ± 1.527*	137.4 ± 19.33*
	F	4	17.80 ± 1.960*	63.60 ± 7.916	19.19 ± 2.143*	50.15 ± 6.40*
Elephant	M	3	5.667 ± 2.60	16.67 ± 5.24	8.720±3.70	109.8 ± 17.39
	F	1	12	47	35.88	256.48

Key - * Values Significantly different at $P < 0.05$

Table 4.42: Sex distribution of mean biochemical values of some wildlife from Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Wildlife	Sex	No. examined	Creatinin (µMol/L)	Urea (mMol/L)	Albumin (g/L)	Total protein (g/L)	Total bilurubin (mg/dl)	Direct bilurubin (mg/dl)
Zebras	M	8	65.83 ± 4.515*	55.34 ± 5.598*	40.37 ± 2.626*	87.81 ± 2.379	1.226 ± 0.184	2.143 ± 0.109
	F	7	20.16 ± 5.207*	19.31 ± 1.802*	21.84 ± 0.639*	106.7 ± 11.75	1.893 ± 0.339	2.295 ± 0.414
Elands	M	7	32.23 ± 7.701*	20.75 ± 4.774	12.62 ± 3.233	120.9 ± 6.307*	20.44 ± 2.702*	7.213 ± 0.427*
	F	5	80.60 ± 5.748*	18.65 ± 3.777	22.27 ± 3.656	57.66 ± 12.08*	8.164 ± 2.043*	15.69 ± 1.028*
Wildebeests	M	4	20.07 ± 3.389*	80.61 ± 5.272*	247.1 ± 9.191*	77.93 ± 2.828*	1.755 ± 0.0772	3.503 ± 0.163
	F	4	8.925 ± 2.491*	63.79 ± 2.216*	187.1 ± 2.529*	53.15 ± 1.883*	1.515 ± 0.152	3.788 ± 0.189
Waterbucks	M	4	10.93 ± 2.099*	93.13 ± 4.376*	104.9 ± 1.164*	74.32 ± 8.422*	8.85 ± 1.233	4.953 ± 0.533*
	F	4	56.67 ± 12.31*	52.21 ± 5.167*	192.3 ± 21.36*	111.1 ± 4.646*	8.858 ± 1.333	7.908 ± 0.565*
Elephants	M	3	7.447 ± 2.56	4.443±0.44	32.02 ± 3.15	93.01 ± 6.71	2.163 ± 2.267	2.420 ± 0.243
	F	1	8.0	1.07	50	96.30	4.42	4.83

Key - * Values Significantly different at $P < 0.05$

Table 4.43: Sex distribution of mean potassium, sodium and glucose values of some wildlife from Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria.

Wildlife	Sex	No. examined	Potassium (MEq/L)	Sodium ((MEq/L)	Glucose (mm/L)
Zebras	M	8	36.21 ± 1.184*	137.8 ± 4.662	1.592 ± 0.105
	F	7	15.66 ± 1.119*	137.3 ± 1.982	1.793 ± 0.173
Elands	M	7	19.30 ± 2.527*	126.5 ± 3.953*	6.391 ± 0.546
	F	5	29.56 ± 1.888*	151.7 ± 5.948*	4.654 ± 1.813
Wildebeests	M	4	14.26 ± 1.424*	90.39 ± 2.969*	4.013 ± 0.881*
	F	4	29.75 ± 1.429*	123.2 ± 2.595*	4.650 ± 0.463*
Waterbucks	M	4	213.3 ± 3.93*	126.0 ± 9.801	12.14 ± 0.26*
	F	4	107.0 ± 3.342*	142.5 ± 2.505	7.134 ± 1.474*
Elephants	M	3	24.39 ± 3.147	141.0±2.06	1.04 ± 0.409
	F	1	12.20	125.42	2.91

Key - * Values Significantly different at $P < 0.05$

CHAPTER FIVE

DISCUSSION

The finding of RVFV antibodies in both wildlife and livestock is of significance especially when the epidemiology of the disease is taken into consideration as it affects livestock productivity and is also of zoonotic importance (Swanepoel and Coetzer, 2004). The proximity of cattle-grazing to wildlife population makes interspecies disease transmission possible (Daszak *et al*., 2000). The zoonotic implication of detecting RVFV antibodies is that both the YGR and SWP are used for ecoutourism and grazing of livestock. Interaction between wildlife and livestock in the environment of wildlife could be a source of RVFV infection to the livestock which may inturn be a source of infection to susceptible livestock not in the vicinity of the game reserves. This statement is supported by other workers, who have reported that important diseases of economic and public health importance like RVF, brucellosis and tuberculosis are shared among species and easily transmitted at the wildlife-livestock interface (Daszak et al., 2000; Bengis *et al*., 2002; Evans *et al*., 2008; Gortazar *et al*., 2015; Jori *et al*., 2015). Game viewing, swimming and other activities like camping in the reserve and poaching may serve as potential source of RVFV infection to humans. These activities and other factors have been reported as potential means and sources for human infection with RVFV (Olaleye *et al*., 1996; Bengis *et al*., 2002; Metras *et al*., 2012; Bukbuk *et al*., 2014). Cattle herders around the reserves sell milk and meat for consumption by humans and this may lead to RVFV infection. An association between consumption of unprocessed milk and human infection with RVFV has been reported during RVF outbreak in South Africa (Swanepoel and Coetzer, 2004).

The presence of RVFV antibodies observed in waterbuck, elephant, eland and wildebeest is not suprising as there has been evidence of exposure in similar species in other countries (Olive *et al*., 2012; Britch *et al*., 2013; Paweska *et al*., 2015; Jori *et al*., 2015). Anderson and Rowe, (1998) demonstrated antibodies to RVFV in black and white rhinos, buffaloes and waterbucks in Zimbabwe, Evans *et al*. (2008) reported that during wildlife surveillance for RVFV in Kenya, antibodies were detected in buffaloes, black rhinos, kudu, impala, elephant, eland, gerenuk and waterbuck, Jori *et al*. (2015) reported circulation of RVFV antibodies in buffaloes in Botswana. It is indicative that wildlife could feature in the epidemiology of RVFV in Nigeria and could play a significant role in transmission at the wildlife-livestock interface.

The high RVFV prevalence in waterbuck, a water dependent wildlife, in YGR is of concern as the reserve has characteristic savannah vegetation, including swamps with river floodplains, grassland and thick bushes (Odunlami and Lake, 2003) which could support the proliferation of *Aedes* and *Culex* mosquito which are vectors for RVFV. These mosquitoes can aquire RVFV from feeding on infected animals (Rheyemamu *et al*., 2012). Female mosquitoes are capable of transmitting the RVFV directly to their offspring via eggs leading to new generation of infected mosquitoes (Anyamba *et al*., 2001). The availability of resources for the hatching, infection, proliferation and spread of vectors of RVF in YGR is a potential for possible epizootics and associated human epidemics occurring.

This is the first time antibody to RVFV is probably reported in wildlife in Bauchi State, Nigeria. Though is not suprising as antibodies to the infection have previously been reported in livestock and humans in Nigeria (Fagbemi *et al*., 1973; Olaleye *et al*., 1996;

Bukbuk *et al*., 2014) and neighboring Countries of Chad, Niger and Cameroun (WHO, 2016). The possible explanation for the presence of RVFV is that there is uncontrolled movement of livestock across international and state borders in Nigeria (Lazarus *et al*., 2015) and increasing interactions during grazing of cattle and other animals in the same environment with wildlife. Studies have shown that both mixing of wildlife with cattle herds and increasing cattle herd size were associated with higher seroprevalence of RVFV (Daszak *et al*., 2000; Madani *et al*., 2003; King, 2004; Evans *et al*., 2008; Gortazar, 2015; Jori *et al*., 2015).

Female cattle reacted to RVFV more positively than their male counterparts in this study, similar observation have also been reported for cattle (Olaleye *et al*., 1996). This could be due to high preference for female cattle by herders who keep them for longer period in the herd for reproductive purposes and are more prone to disease exposure during this period (Olaleye *et al*., 1996). Susceptibility of livestock to RVFV has been reported irrespective of breed of the animals (Olaleye *et al*., 1996; Evans *et al*., 2008).

The findings of this study have shown that antibodies FMDV are present in both cattle and wildlife. Previous survey for FMDV antibodies in Nigeria showed a seroprevalence of 75.1% in cattle in Kwara State (Olabode *et al*. 2013). Also, Ehizibolo *et al*. (2010) and Wungak *et al*. (2015) reported a seroprevalences of 64.3% and 71.0% in cattle respectively, in studies carried out in Plateau State. Serological studies conducted between 2009 and 2011 by Lazarus *et al*. (2012) in six border States and two other states that lie on the major cattle routes in Nigeria showed higher seroprevalence in cattle from Yobe State (82.0%), followed by those from Plateau (80%), Ogun (77.8%),

Taraba (73.5%), Adamawa (68.0%), Borno (67.0%), Sokoto (63.0%) and (27.8%) in sheep and goats from Bauchi State. The similarity of the findings of the spresent study with previous studies have shown that FMD is still an enzootic disease in Nigeria and this could be attributed to the lack of complementing vaccination campaign programs in Nigeria. There is unrestricted herds mobility, continuous contact and intermingling of different cattle herds at water points, communal grazing areas and porous borders (Lazarus *et al*., 2012; Wungak *et al*., 2016).

The higher FMDV seroprevalence in female cattle during this study was consistent with the findings of Olabode *et al*. (2013) and Wungak *et al*. (2016) who reported a risk difference in association with sex during FMD studies in Kwara and Plateau States, Nigeria respectively. Similarly, Mazengia *et al*. (2010) reported higher incidence of FMD in females in Northwest Ethiopia. However, most of the cattle sampled were females as opposed to males therefore; the significant association in sex could be attributed to the preference of females to males by the nomads for reproductive purposes and milk production and therefore females are kept for longer period thereby having higher exposure risks than males (Longjam *et al*., 2011; Olabode *et al*., 2013; Wungak *et al*., 2016).

This is the first report, to the best of my knowledge on serological status of FMD in wildlife in YGR and SWP in Bauchi State, Nigeria. The findings have shown that antibodies to FMDV were present in elands, wildebeests, waterbucks and elephants. This is not suprising as FMD is endemic in Nigerian livestock (Owolodun, 1973; Abegunde, 1987; Chukwuedu and Nimzing, 2012; Ehizibolo *et al*., 2017). The positivity at the interface could be through grazing of ruminants with other livestock

and interacting with wildlife in the same environment. Study in Ethiopia found significant association between cattle exposed to FMDV and their contact history with wildlife (Molla *et al*., 2010). Also, a study conducted at the periphery of protected areas in Zimbabwe indicated that interactions between livestock and buffalos can account for FMD primary outbreaks (Miguel *et al*., 2013). Also, in other Countries like South Africa, Zimbabwe, Zambia, Botswana, Namibia, India, and Chad FMD antibodies have been demonstrated in numerous wildlife species including those sampled in this study which were Eland (*Taurotragus oryx*), Waterbuck (*Kobuselli psiprymnus*), Kudu (*Tragelaphus imberbis*), Wildebeest (*Connachaetes gnou*), African Elephant (*Loxodonta africana*), and Hartebeest (*Alcelaphus buselaphus*) (Sutmoller *et al*., 2000; Thomson *et al*., 2003; Vosloo *et al*., 2005; Vosloo *et al*., 2007; Swai *et al*., 2009; Arzt *et al*., 2011; Weaver *et al*., 2013; Di-Nardo *et al*., 2015). Although in most parts of West and Central Africa, the role of wildlife in the epidemiology of FMD has not been fully studied (Sutmoller *et al*., 2000; Bengis *et al*., 2002; Alexandersen *et al*., 2002), the presence of antibodies to FMDV in wildlife species observed in this study is suggestive they are potential reservoirs for transmission to domestic animals.

The presence of FMD antibodies in both wildlife and cattle at the interface level observed in this study might be driven by direct contact between wildlife species and livestock. Direct contact at wildlife-livestock interface seems to be more common in open habitats and during the dry seasons, when water availability constrains animals to closely congregate thus increasing the transmission likelihood of water-related infections like FMD (Sutmoller *et al*., 2000; Bengis *et al*., 2002; Di Nardo *et al*., 2015). The occurrence of FMD antibodies during the study probably occurred through indirect contact (i.e. through soil, forage and water contaminated by bodily discharge of infected

animals), which might be regarded as the most likely factor to be associated with the risk of FMDV transfer from domestic to wildlife species (Di nardo *et al*., 2015).

The study confirms the presence antibodies to FMDV serotype O, A, SAT 1 and SAT 2 among eland, wildebeest, waterbuck and elephants with high levels of serotypes-specific antibodies types A and SAT 1 observed. This is the first serological evidence of FMD serotypes O, A, SAT 1 and SAT 2 circulating in wildlife species to the best of my knowledge in Nigeria. Olabode *et al*. (2013) confirmed that FMD serotypes O, A, SAT 1 and SAT 2 have been circulating in the country in the last 54 years (1955-2009). Also, Chukwuedo and Nimzing (2012) confirmed the circulation of serotypes SAT 1 and SAT 2 among cattle in northern Nigeria with the highest circulation of SAT 1 in Bauchi and Plateau States. Ehizibolo *et al*. (2017) reported that antibodies against FMDV serotypes O, A, SAT1, SAT2 and SAT3 were detected from FMD outbreaks in cattle in Kaduna, Kwara, Plateau and Bauchi States northern Nigeria. He also reported a novel FMDV SAT1 strain which differ from the previously known SAT 1 topotype in West Africa. The present study showed that some of the FMDV serotypes circulating in livestock in Nigeria are also present in wildlife in YGR and SWP in Bauchi State, Nigeria. The sharing of abundant grazing, water and 'salt lick' resources available in the wildlife environment by both wildlife and livestock species may serve as potential source of interspecies and cross species transmission of the FMDV serotypes. It has been shown that once an animal is infected with FMDV, it can be disseminated into the environment including pastures, water sources and soil (Fakai *et al*., 2015) and the disease can persist in wildlife for 24 years or longer (Condy, *et al*., 1985; Thomson *et al*., 1992; Vosloo *et al*., 2007; Weiss *et al*., 2012).

Bovine Viral Diarrhoea (BVD) is a disease of cattle, inflicting substantial losses to both beef and dairy enterprises worldwide (Vanroose *et al*., 1999). BVDV antigen was detected in cattle population in the study area. The prevalence observed from this study was similar to the 13.4% seroprevalence reported by Taylor *et al*. (1977) in Plateau State, but much lower than 36.8% prevalence reported by Ferreira *et al*. (2000) in South African dairy herds. The differences could however be attributed to the variations in the techniques employed by the researchers in the analysis of sample.

The distribution of BVDV infections in cattle based on sex in the study showed a significantly ($P < 0.05$) higher prevalence in females than in males. This agrees with the studies by Bello *et al*. (2016) who reported higher prevalence of BVDV in female cattle in Sokoto State, Nigeria. Also, Peter *et al*. (2015) recorded higher BVDV prevalence in female camel than males in Borno State, Nigeria. They associated their findings to the fact that most female cattle stay for longer period in the herd for reproductive purposes and are mostly presented to the abattoir for slaughter at advanced age during which their immunity against diseases are mostly weaken due to pregnancy and lactation. However, more of the animals sampled during this study were female as oppose to male cattle; therefore, the significant association in sex prevalence could be attributed to the small number of males sampled. However, occurrence of BVD persistently infected (PI) animals is associated with infections of pregnant cows or heifers during gestation prior to the development of the foetus immune systems and animals born alive will spread BVDV life long (Elazhary *et al*., 1981).

Bovine Viral Diarrhoea Viral antigens were detected in waterbuck, eland, hartbeest and wildebeest during this study. Presently no other study regarding molecular detection of

BVDV in wildlife in Nigeria has been performed as such this finding can be compared only with those from other countries (Hunter and Carmichael 1975; Paton *et al*., 1998; Viltrop *et al*., 2002; Handel *et al*., 2011). Some researchers tested pooled samples (pooled serum, milk and buffy coat samples or pooled ear notch phosphate buffered saline) by RT-PCR to screen many animals, if the pooled sample is positive by RT-PCR, samples are tested individually by PCR to identify the respective BVDV positive animal (Munoz-Zanzi *et al*., 2000; Kennedy *et al*., 2006; Khodakaram-Tafti *et al*., 2016). The pooling could provide an initial, rapid, cost-effective method of screening cattle herds for BVDV. Bovine viral diarrhea virus uses multiple strategies to ensure survival and successful propagation in mammalian hosts, which includes suppression of the host's immune system, transmission by various direct and indirect routes, and induction of persistently infected (PI) hosts that shed and transmit BVDV much more efficiently than non-PI animals (Walz *et al*., 2010). The sharing of common grazing and water points in the study areas can significantly influence the potential for interspecies and cross species transmission of BVDV. Infection with BVDV is associated with close contact with infected animals especially the PI animals and spread of the virus from these PI animals may be assisted by mechanical spread by biting flies (Tarry *et al*., 1991).

Even though, the role of cattle in BVDV maintenance is well established (Baba *et al*., 1994; Paton *et al*., 1998; Bello *et al*., 2016; Rodríguez-Prieto *et al*., 2016), the epidemiology of the disease in wild ruminants is not so clear and more difficult to assess. The findings of this study showed that BVDV is actively circulating in both domestic and wild animals, however, the finding cannot answer the question of how long BVD has existed in the indigenous cattle and wildlife population of Nigeria,

perhaps due to the fact that the disease has not been critically investigated as an important disease in Nigeria. However, the possibility of introduction of BVDV into the country through import of semen and persistently infected exotic cattle in the past few decades cannot be ruled out. Also, extensive and continuous use of BVDV contaminated semen, primary bovine cell culture vaccines and biological products may serve as potential sources of BVDV spread in the country.

The high seroprevalence of AHSV obtained from this study is consistent with previous serological studies in Nigeria (Nawathe *et al*., 1981; Oladosu *et al*., 1993; Adeyefa and Hamblin, 1994; Baba *et al*., 1994; Ehizibolo *et al*., 2014) and may be an indication of continual exposure of horses regardless of age, sex and breed. The study area Bauchi State extends from the tropical savannah of Guinea to the savannah of Sudan, with thick vegetation and hot and dry climatic conditions which favour vector propagation and virus transmission (Tende *et al*., 2006). The practice of keeping horses outside as observed during this study could increase their chances of coming in contact with insects, especially *Culicoides* spp that transmit AHSV, should these insects be harbouring the virus.The fact that there are water pools and such other areas favourable for the breeding of these insects in the study areas could mean that more infections could occur in these horses and this agrees with the observations of Mellor *et al*., (2000). Furthermore, Adeyefa and Dipeolu (1993) reported the presence of *Culicoides* breeding sites around horse stables in Nigeria. They also reported the presence of *Culicoides quinquelineatus, C. imicola and C. schulzei* as possible vectors of AHS in Nigeria. Also, Fasina *et al.* (2008) reported the presence of biting midges in stables during a field investigation in various stables across Nigeria. Findings from this study showed that both antibodies and antigen to AHSV were detected in zebras. Zebras are

known to be natural reservoirs of AHSV and in most instances they are the cause of sudden outbreaks in Africa (Barnard, 1998). Investigations during the study revealed that when Sumu Wildlife Park was established in the year 2006, some wildlife species were imported from Namibia to the park during which over 20 horses were brought into the game park for patrol activities. It was observed during the study that almost all the 20 patrol horses used by the game rangers died after clinical signs manifestations typical of AHS. Barnard, (1998) reported that the first cases of African horse sickness (AHS) then in South Africa occurred in the horses of hunters who entered zebra territory.

To date there has not been any report of AHSV in wild equidae in Nigeria; hence this is .the first report of AHSV in Horse-Zebra interface level. This calls to question the previous understanding of the epidemiology of the disease and open new areas of research in equidae viral infections

The prevalence for haemoparasite infections in the wildlife obtained from this study was high and it is interesting to note that major tick-borne parasitic diseases affecting livestock in Nigeria (Enwezor *et al*., 2009; Kamani *et al*., 2010; Agu and Amadi, 2001; Pam *et al*., 2013; Okarafor and Nzeako, 2014) were detected in the wildlife species. The identified haemoparasites were *Anaplasma marginale*, *Ehrlichia ruminantium*, *Trypanosoma* spp and *Babesia bovis, Babesia bigemina*, *Babesia equi*, and *Babesia cabale*. Elands, Wildebeests and Zebras from SWP were shown to have higher haemoparasite burden than Elephants, Waterbucks and Hartebeest from YGR. The probable reason could be tick infestation as most of the Wildlife species particularly from SWP had very high tick burden infestation Despite the presence of the haaemoparasites, clinical manifestations were not observed during the study, these

findings are consistent with other studies that have shown that detection of blood parasites in wildlife is often incidental, they are resistant to haemoparasite infections and that clinical disease is often stress related (Homer *et al*., 2000; Schuster, 2002; Munang'andu *et al*., 2009; Munang'andu *et al*., 2010; Duscher *et al*., 2014). Overall, these findings point to the fact that wildlife could play an important role in the epidemiology of haemoparasites in Bauchi, State, Nigeria. This implies that the enchroachement of livestock into wildlife habitat for grazing purposes could expand the reservoir host occupancy range of tick borne diseases whose spill-over at the interface level would impact negatively on livestock production. This poses a significant challenge for control of tick-borne diseases especially in the interface areas where expansion of wildlife and livestock is taking place.

The present study confirms the range of haemoparasites found in cattle in Nigeria (Enwezor *et al*., 2009; Kamani *et al*., 2010). The high infection rate suggests a continuous challenge by haemoparasites and possibility of existence of carrier state in most of the cattle sampled. *Babesia* spp (*B. bigemina* and *B. bovis*) alone or in combination with other parasites accounted for most of the parasites seen followed by *Anaplasma* and *Trypanosoma* spp respectively. This is in agreement with the work of Kamani *et al*. (2010) who conducted a survey in cattle in North-West Nigeria but in contrast with the work of Bell-Sakyi *et al*. (2004) who observed a reverse trend where *Typanosoma* spp and *Anaplasma marginale* were found to be most prevalent during survey conducted on livestock in Ghana. High prevalence of *Anaplasma marginale* and *Trypanosoma* spp were observed in this study compared to previous report in Nigeria (Enwezor *et al*., 2009; Kamani *et al*., 2010). The probable reason could be due to abundance of ticks in the environment and on cattle observed during sampling and that

YGR is a known tsetse fly infested area (Tende *et al*., 2006) probably resulting in high burden of haemoparasites on animals in and around the YGR and SWP.

The significantly higher haemoparasitemia observed in female than male cattle during this study could be attributed to accumulation of parasites by the females' due to extended breeding for economic reasons such as calving and milk production. This confirms previous reports of sex dimorphism in the incidence of haemoparasitism in Nigeria (Agu *et al*., 1989; Agu and Amadi, 2001; Enwezor *et al*., 2009; Kamani *et al*., 2010; Okorafor and Nzeako, 2014). Also, the susceptibility of cows might be attributed to reduced immunity as a result of stress due to pregnancy and lactation. The variability in breed specific parasitemia observed in this study was in line with observations made by Agu and Amadi (2001) that attributed this variability to host specific factors like physical features, disease resistance, climatic stress resistance and production traits peculiar to individual breeds.

The high prevalence of haemoparasites in donkeys' comapared to other livestock in this study could be due to the fact that donkeys still remain one of the most underappreciated animals and an important draught animal, serving a key role in the agricultural economy of the developing world including Africa (Ahmed *et al*., 2008). Unfortunately, these animals are left malnourished and underfed, and mostly subjected to different conditions of hardships like maltreatment, poor management conditions, and over laden. Javed *et al*. (2014) found higher prevalence of Babesiosis during a study on haemoparasites of donkeys in Pakistan where he linked it to the potential negative effect especially on their welfare and health status. The trend of female donkeys having higher haemoparsites infection than males was observed in this study. The phenomenon of

parasitism during pregnancy due to stress and decreased immune competence (Urquhart, 1996) in female animals may have contributed to that even though we did not identify the exact number of pregnant animals during the study.

The low prevalence of haemoparasites of horses observed during this study is congruent with the study conducted by Pam *et al*. (2013) on haemoparasites of horses in Plateau State, Nigeria. In contrast Garba *et al*. (2011) reported higher prevalence of 80.4 % during studies on haemoparasites of horses in Niger State, Nigeria. Also, Javed *et al*. (2014) reported prevalence of 74.45 % during studies on tick borne haemoparasites of horses in Pakistan. They associated their findings to lack of immunity in the young horses and lack of good management practices. The low infection with haemoparasites in horses observed in this study compared with that of wildlife, cattle and donkeys could be due to the relative care being given to horses in terms of welfare and health management compared to other species and they are mostly associated with royalty. The lower infection with haemoparasites observed in female horses could be due to lack of exposure to sources of infection. Königová *et al*. (2003) and Love (2003) showed that many intrinsic factors such as sex, age, and breed and extrinsic factors like management, climate and parasite control programs influence the prevalence of parasites of domestic animals.

Most hemoparasites reported in this study are known to replicate in the erythrocytes leading to hemolysis and anaemia. All the hemoparasites mentioned singly or in combination led to significant ($P < 0.05$) reduction in the Mean PCV. The ability of *Trypanosoma* spp alone or in combination with other parasites to cause a significant reduction of PCV of infected animals especially in cattle and donkeys

during this study lends credence to the fact that animal trypanosomosis is still a serious challenge to profitable livestock production in sub Saharan Africa (Enwezor *et al*., 2009; Swallow, 2000; Kamani *et al*., 2010). In contrast *Trypanosoma* spp alone or in combination with *Anaplasma marginale* did not cause significant reduction in PCV in wildlife species during this study. The mean PCV of non infected animals was significantly higher ($P < 0.05$) than in animals with one or more parasites which may be related to compounding factors like scarcity of good quality feeds and stress of trekking experienced by most pastoral animals in search of feeds (Kamani *et al*., 2010).

The present report suggest the presence of possible carrier populations of haemoparasites infections at the wildlife-livestock interface level which both serving as reservoirs of infection for tick-vectors and susceptible wildlife and domestic animals and with potential for clinical relapse under stressful conditions. The pastoral management system which subjects the livestock species to continuous challenge of vectors, high cost of acaricides and scarcities of feeds are compounding factors to efforts at controlling vector borne diseases (Kamani *et al*., 2010).

On the burden of GIT parasites, the study provided valuable information on GIT parasite infections at the wildlife and domestic animal interface level with donkeys having the highest prevalence followed by cattle, wildlife and the horses being the least.

Gastrointestinal parasite burden in wildlife species in this study indicated that waterbucks from YGR and elands from SWP had the highest burden of GIT parasites than the other wildlife species sampled. These animals could have been infected at

watering points and during grazing activities. It has been shown that GIT worm infection rates are dependent on a number of factors which include the number of infective larva ingested by the host which is in turn influenced by climatic factors, vegetation, and animal density (Maingi *et al*., 2004). Dry open areas prone to excessive heat are hostile to the survival of infective larva while moist areas near water sources are conducive for the survival of infective larva (Munang'andu *et al*., 2012). The climate of the study areas is favourable for the survival of infective larva even during dry season because of the rich source of moist areas, water points and thick vegetation that attract more animals to the area thereby increasing the animal population density in the area and these bring about the possibility of direct and or indirect transmission of GIT parasites at the pastures and water points. On the moist herbage, larva of different helminths migrates up and down the blades of grass which facilitate the uptake of infective larva by grazing animals (Munang'andu *et al*., 2012). This would account for the reason waterbuck being water dependent had high infection rate of different species of helinths during the study. Munang'andu *et al*. (2012) in Zambia also observed high infection rates of helminths species among water dependent wildlife species and associated his findings with continuous exposure to infective helminth larva at pastures.

Wildlife are likely to serve as critical determinants of infection to each other which in turn can equally serve as critical determinants of infection to livestock as they contaminate the pastures around water sources and also the water holes with faecal droppings containing infective larva (Munang'andu *et al*., 2012). Active transmission cycle can be maintained at the Wildlife-Livestock interface level so long as the livestock species continue to graze on those contaminated pastures and drink water from the water holes even in the event of routine deworming of the livestock by owners.

These observations indicate that reducing infection to the wildlife species at the water sources is likely to reduce the source of infection to other animals. It should be noted that even the wildlife can also be infected by livestock species during poach grazing activities in the reserve area. Hence the need for innovative disease controls strategies that would reduce the cycle of transmission within wildlife population and at the wildlife- livestock interface level.

The study indicated that *Fasciola* spp were the most prevalent among the helminths identified in both wildlife and domestic animals. Phiri *et al*., (2007) pointed out that snail intermediate of worm species like *Schistosom*a and *Fasciola* are often concentrated in marshy areas or marginal shallow water areas. This would account for the reasons why waterbuck were the only species infected by *Schistosoma* spp during this study as they are final hosts and predominantly occupy marshy areas. They are also likely to maintain a high transmission cycle of the *Schistosoma* spp in the study area which would be a threat to other animals and humans. Also, the availability of water holes and moist pastures in the study areas serves as a very good source of miracidia-snail contact especially during dry season which attracts both wild and domestic animals for grazing activities on those pastures thereby accounting for the reason behind high prevalence of *Fasciola* in the study area. Both Mir *et al*. (2013) and Parsani *et al*. (2013) reported that *Fasciola* spp is the most prevalent helminth in studies carried out in India and Ethiopia respectively and associated their findings to moist pastures close to water sources and availability of intermediate host. The presence of mature flukes of *Paramphistomum* spp in the rumen of waterbuck observed during this study is indicative of the availability of the intermediate hosts (snail) in the study area. As the immature Amphistomes excyst in the duodenum or mid-to-proximal jejunum, they

migrate and attach firmly to the mucosa and may penetrate as far as the muscularis mucosa. Damage is related to the numbers of migrating flukes and it increases in intensity from localized enteritis through patches of villous atrophy to severe destruction of the mucosa. Radostits *et al*. (2000) observed that clinical and production effects of Paramphistomiasis are dependent upon the extent of the lesions in the GIT as some compensation for functional deficiency can take place in the un-damaged lower small intestine. The presence of mature flukes in the rumen does not usually elicit any significant response, however in massive infections papillae are short and red, becoming fused into aggregations with ruminal contents adhering firmly to the surface (Radostitis *et al*., 2000). The waterbuck carcass observed during the study was found to be emaciated and anaemic with large quantity of adult flukes in its rumen. Diaz *et al*. (2006) observed that amphistomes infected cattle and buffaloes were shown to be emaciated due to reduction of feed digestibility and depression of appetite. Halium *et al*. (2014) showed that the blood sucking hematophagous activity especially of the immature stages of Amphistomes causes anaemia in cattle.

This study also found that nematode infections were particularly high in wildlife, cattle, horses and donkeys followed by trematodes, cestodes and protozoa. Worthy of note is the fact that none of donkeys and horses had infections with protozoan GIT parasites. High nematode infection has huge impact on livestock production since they result in reduced milk, meat, wool, hide products and stamina of working animals (Ekong *et al*., 2012). This can result in diminution of production potentials such as decreased growth rate, weight loss in the young and late maturity of the animals (Swai *et al*., 2006, Adedipe *et al*., 2014). GIT nematodes have negative effects on body condition, body

mass and fecundity in wildlife species where most of the effect is attributable to reduced appetite (Stien *et al*., 2002).

Of great concern is the zoonotic implication of some of the GIT parasites identified in both wildlife and domestic animals which include *Fasciola* spp, *Dicrocoelium dendriticum*, *Taenia* spp, *Schistosoma* spp, *Moniezia* spp *Ancylostoma* spp and *Sarcocystic* cyst. They are known to be zoonotic and have caused considerable economic losses and health problems (Elele *et al*., 2013; Mir *et al*., 2013). It is suggestive that humans that are directly or indirectly in contact with wildlife and domestic animals especially during herding, processing and consumption of meat (bush meat) and other by-products from livestock and wildlife, utilization of rich resources in the reserve area like water are at risk of zoonotic infections from the zoonotic GIT parasites.

Fasciola spp were identified in all the wildlife and domestic animals sampled, *Strongyle*-type eggs were identified in cattle, eland, wildebeest, kudu, elephant and waterbuck whereas *Strongylus* spp were identified only in the equidae (zebra, donkeys and horses). The other GIT parasites all occurred in three or fewer animals which classifies them as either occasional or accidental parasites (Horak 1981). The relative few GIT parasites identified in some of the wildlife species could also be due to smaller population size and number of the wildlife sampled. Where a large number of wildlife species are present, cross-transmission can take place more readily (Boomker *et al.* 2000). Cross-transmission of *Trichomena* spp known to be specific helminth for equids (zebra, donkeys, horses) (Soulby, 1982) was identified in eland during the study.

On the variation in sex prevalence among wildlife and domestic animals sampled females had higher infections with gastrointestinal helminths than males in cattle, this could be due to stress and decreased immune competence in female animals which is prominent during pregnancy and can increase the possibility and severity of parasitism in females than males (Urquhart *et al*., 1996). Male donkeys and horses had higher GIT parasites burden than females. Raza *et al*. (2013) pointed out that males were more likely to be infected with helminth than female, reason given was that male animals were more aggressive when feeding and thus likely to pick up more ova of helminths on the pasture.

The variation in breed prevalences in domestic animals could be attributed to the existence of favorable environmental factors necessary for the prolonged survival and development of infective larval stage of most helminths (Rossanigo and Gruner, 1995). Furthermore, management system of an animal and availability of good pastures for grazing could also be accountable for the difference in prevalences (Regassa *et al*., 2006). Some of the wildlife and livestock with good nutrition must have tolerated helminth infections better or that both host and parasites had reached a state of equilibrium and were asymptomatic at the point of faecal collection. In addition, mixed infections with GIT parasites were observed among the wildlife and domestic animals. Mixed infection was characterized by the presence of two or more helminthes or protozoan in the same animal. The phenomenon of mixed infection has been suggested to be an important cause of morbidity and reduced production in livestock (Jatau *et al*., 2011; Kumsa *et al*., 2011). Furthermore, the immunosuppression of the host immune system by mixed infections increases host susceptibility to other diseases or parasites (Wang and Macdonald, 2006).

Wildlife species were observed to be infested with *Amblyomma variegatum* and *Rhipiciphalus appendiculatus* as the most common tick-species infesting multiple host species both the bovids and equids. *Hyaloma* spp and Boophilus spp were collected from fewer animal hosts. It is not known whether this difference was based on host preference or the relative abundance of the different tick species on the game parks. Moreover, some animal species like waterbucks in YGR were mildly infested with *Amblyoma and Boophilus* ticks whereas zebra, eland, wildebeest, and kudu from SWP were highly infested with ticks' genera including *Amblyoma, Rhipiciphalus, Boophilus* and *Hyaloma*. Waterbuck are semi aquatic medium sized antelopes which often submerged up to the shoulders sometimes leaving only the nostrils when frightened (Nefdt and Thirgood, 1997). Ticks infesting this animal species are likely to drop-off when the animals are submerged in water thereby reducing the attachment time on the host. This could be the foreseeable reason for the low burden of ticks observed in them. However, there is need for detailed experimental studies to determine the host preference of tick infestations in wildlife and to establish reasons why some animal species are less infested than others. Information obtained from such studies would help in selecting wildlife species for culling especially in situation where population reduction of selected wildlife species is aimed at reducing the tick burden. The possible reasons for the high tick burdens observed in some of the wildlife during this study include increased host density, habitat changes, poor body condition of the hosts and parasite-defence grooming, but these factors are not independent (Cumming, 1982; Mooring *et al*., 2003 Muruthi *et al*., 2016). Increased host density increases the probability that ticks will find a suitable host, and may also lead to overgrazing. In savannahs overgrazing is often accompanied by bush encroachment (Cumming 1982)

which provides cover, and may actually improve the microclimate for the free-living stages of some tick species. Overgrazing can also cause a reduction in the available biomass of plant material and a shift towards less nutritious invader species in the plant community. This can cause nutritional stress which reduces host resistance (Muruthi *et al*., 2016). Tick infestation has been suggested as a cause of mortality in several ungulate species (Melton and Melton 1982; Gallivan *et al*., 1995; Muruthi *et al*., 2016). Wild mammals in Africa mostly have high levels of innate resistance to haemoparasites and the tick vector that transmit those haemoparasites (Fyumagwa *et al*., 2007). The increasing activities of domestic animals at the fringes and within YGR and SWP can facilitate cross-transmission of ticks from the wildlife to domestic animals. Ticks are important to the health of both domestic and wild animals in Africa, some of which are also a threat to human health, but in Africa it is the domestic animals that are severely affected by ticks and the pathogens they transmit (Makala *et al*., 2003). Various tick genera are associated with various diseases, for example *Amblyomma* species are associated with viruses noted to be mosquito borne. More importantly, the impact of ticks on human health is on the rise and more novels microbial associations have continued to be described (Jasinskas *et al*., 2007). In this study, ticks collected from the wildlife species in YGR and SWP were identified morphologically using Walker's identification keys, where *Amblyomma* spp were identified by ornamentation, long mouths and festoons and *Rhipiciphelus* spp identified by unique striped enamel on a brown-dark background over their conscutum (Walker, 2003). Other tick samples could not be identified due to lost body parts and physical damages. For example, in the genus *Amblyomma* some tick samples had partially lost the conscutum tampering with the ornamentation, making it difficult to identify, a problem observed by Abdigoudarzi *et*

al. (2011) who noted that morphological changes that occur on ticks' surface body may result to misleading identification

The study provided a preliminary data on haematology and biochemistry values of some wildlife species from YGR (elephants and waterbuck) and SWP (elands, wildebeests and zebras) in Bauchi State, Nigeria. Results of haematologic parameters including PCV, HB, RBC, WBC, MCV, MCH, MCHC, Neutrophils and lymphocytes indicated some differences in some of the values obtained for males and females wildlife species sampled. It was observed during the study that more males were sampled than females and the males had better body condition with bigger body mass and size than females where body condition are affected by preganancy and lactation (Dolka *et al*., 2014). Health status, body condition and habitat of wildlife species can influence their blood values to either increase or decrease (Peters *et al*., 2011; Dolka *et al*., 2014). Ticks were found on the wildlife species during sampling and most of the wildlife species were darted during dry season a period of limited pasture resources in YGR and SWP (Tende *et al*., 2006). According to Thrall *et al*. (2006) haematological values vary with age, sex, physiological state, temperature, time of the day and season and also, ticks and tick borne pathogens and nutritional stress could be the probable reason for the PCV, HB, RBC and WBC differences observed in the wildlife species sampled (Váhala *et al*., 1989; Silberová *et al*., 2010; Mohamed *et al.*, 2012; Ozkan *et al*., 2012; Chege *et al*., 2013). Changes in many of the haematological values could be due to excitability stress and fright syndrome (Schalm et al., 1975; Brown and Slenning, 1996; Mohammed *et al*., 2012). The impact of the dart syringe and needle and chemicals used for immobilization during the wildlife capture might have triggered the stress and fright syndrome in the wildlife and subsequent changes in haematological values (Thrall,

2006). It was observed during this study that some of the wildlife species were agitated and run before and after being darted while others did not. Also, some were darted in the morning while others darted in the hot afternoon. These could have contributed to the differences in haematological values observed during the study. Drevemo *et al*. (1974) observed that the blood of deer that did not run befeore and after immobilization had higher haematological values caused by mobilization of blood cell reservoirs such as spleen with an increase in PCV, Hb and RBC (Drevemo et al., 1974). On the contrary Rosen and Bischoff (1952) observed that the blood of deer that had run before and after darting had much lower PCV, HB, and RBC than animals that did not run and suggested the cause of this difference to haemodilusion or expansion of plasma volume. Futhermore, it was reported that low Hb content characterizes animals severely stressed before death (Devemo *et al*., 1974). On the use of drugs it is known that certain tranquilizers affect blood values (Váhala *et al*., 1989; Silberová *et al*., 2010). Etorphine and azaperone were used to immobilize the wildlife during the study. Silberová *et al*. (2010) observed that decrease PCV, Hb and RBC in tranquilized animals could be caused by decrease heart rate, low blood pressure and increase plasma volume. The differences in the haematological values in particular animal or in group of animals in this study could be caused by factors of excitement and the use of drugs. These factors must always be kept in mind when interpreting results.

On the biochemical parameters, the mean values and minimum and maximum ranges for liver enzymes (ALT, AST, ALP, LDH) and creatinine, urea, albumin, total protein, total bilurubin, direct bilirubin, potassium, sodium and glucose were dertemined for apparently healthy elephant, waterbuck, eland, wildebeest and zebra from YGR and SWP in Bauchi State, Nigeria. Data on these parameters in wildlife are limited in Africa

(Drevemo *et al*., 1974; Mohammed *et al*., 2012). A period of poor quality habitat and low nutrient availabity in wildlife habitat is associated with dry season (Clarke *et al*., 2013). Most of the wildlife species were sampled for the study during this stressful period and may account for the differences observed in some of the biochemical parameters. Increased muscle enzymes are associated with stress and capture myopathy which may have a physical or psychological component in the wildlife (Kitchen, 1978; Chege *et al.*, 2013). Alteration of one or more of these biochemical values especially deficiencies affect fertility, immune response and survival of the young (Wilson and Grace, 2001; Jenkins, 2008; Kaneko *et al*., 2008; Özkan and Akgül, 2010). This may account for the cases of mortality observed in wildlife especially in SWP during dry season.

Also, increase in protein and glucose levels indicated impaired renal functions (Jenkins, 2008) and sometimes exercise and stress can cause such increase (Özkan and Akgül, 2010). In the current study the elevated levels of BUN, creatinine and glucose in some of the wildlife could be due to their vulnerability to perceived threat of capture (Chege *et al*., 2013).

CHAPTER SIX

CONCLUSION AND RECOMMENDATIONS

6.1 Conclusion

1. Overall seroprevalences were RVFV 11.3% and 8.49 %, FMDV 65.67 % and 24.53 % in cattle and wildlife respectively, whereas AHSV in horses and zebra were 35.5 % and 20.8 % respectively but zebras and horses were negative to RVFV antibodies.
2. Antibodies to FMDV serotypes O, A, SAT1 and SAT 2 were detected in elephants, waterbuck, eland and wildebeest.
3. Bovine viral diarrhoea virus antigens detection rates of 9.67% and 13.20% were observed in cattle and wildlife respectively.
4. African horse sickness virus was detected from pooled tissues (spleen, lung and lymph nodes) of a zebra carcass.
5. Overall prevalence of haemoparasites was higher in wildlife 56.6 % followed by cattle 42.5 %, donkeys 31.5 % and horses 24.5 % whereas overall prevalence of GIT parasites was higher in donkeys 62.0 % followed by cattle 61.6 %, wildlife 59.4%, and horses 27.0 %.
6. Haemoparasites identified included *Babesia* spp, *Anaplasma marginale*, *Ehrlichia ruminantium* and *Trypanosoma* spp.
7. *Fasciola* spp were the most prevalent helminths in cattle, elands, wildebeest and waterbucks whereas *Strongylus* spp were the most prevalent in zebras, donkeys and horses.
8. Parasites of zoonotic importance (*Fasciola, Ancylostoma, Schistosoma Balantidium and Taenia*) were identified.

9. *Amblyomma* spp and *Rhipicephalus* spp ticks were found infesting most of the wildlife species.
10. The study provided a prelimanary data on haematogical and biochemical values of some wildlife in YGR and SWP in Bauchi State, Nigeria

6.2 Recommendations

From this work it is recommended that:

1. Further studies should be carried out on molecular epidemiology of RVFV, FMDV, BVDV and AHSV in wildlife and livestock in and around YGR and SWP in Bauchi State
2. Further studies on the role of buffalos and warthogs as sources of FMDV and RVFV to other species should be investigated in YGR.
3. There should be enhanced public enlightenment to people within and at the fringes of YGR and SWP on the implications of encroachment and zoonoses.
4. The arthropod vectors of RVFV and AHSV should be investigated to determine their infection rate with RVFV and AHSV.
5. Effective ticks control methods in the wildlife ecosystem should be carried out.
6. Studies on haematobiochemical parameters involving more wildlife species within YGR and SWP should be carried out.

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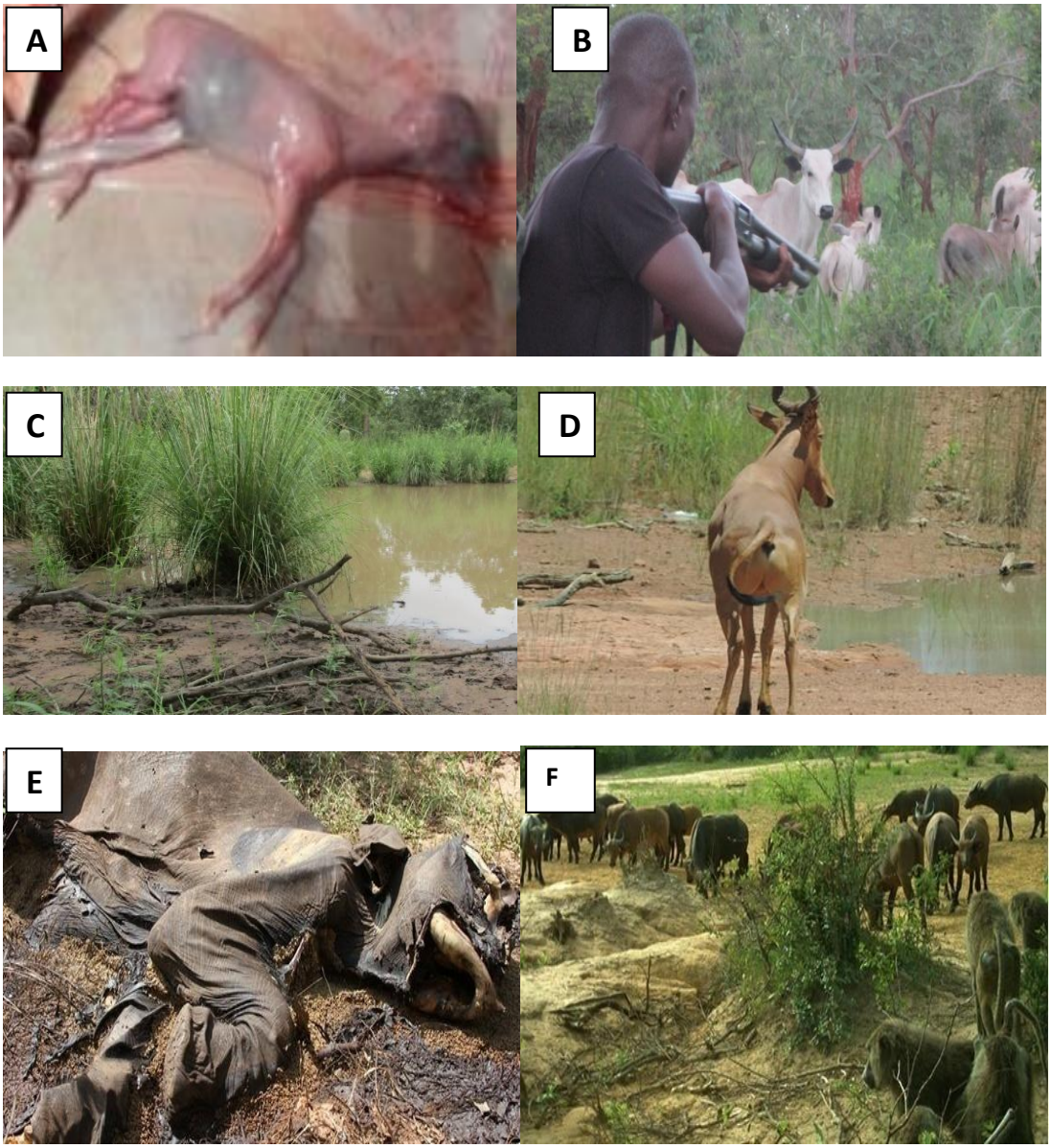
APPENDIX 1: Risk factors associated with pathogen transfer at the wildlife-domestic animals interface at Yankari and Sumu wildlife Parks in Bauchi State, Nigeria



Key: Aborted foetus of Eland (A), Shooting of Livestock by game warder in Yankari Game Reserve (B), Water holes in Yankari game Reserve used by wildlife and livestock (C), Hartbeest at a water hole in Yankari(D), Elephant carcass (E), Wildlife species at the Salt Lick in Yankari (F)

Appendix II: Agarose gel electrophoresis of PCR products from sera of wildlife and cattle.

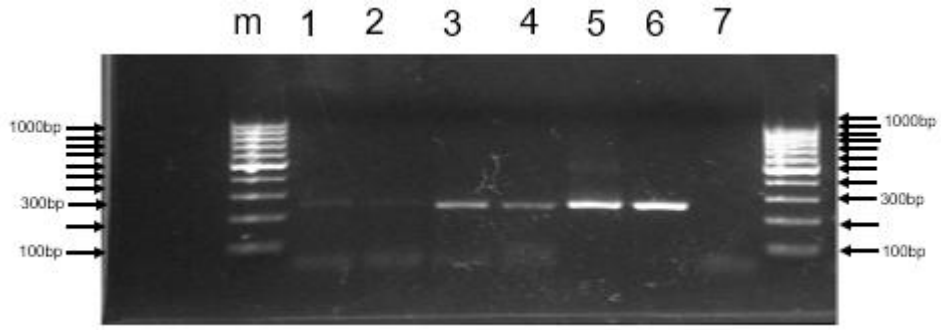


Plate II: Agorose gel electrophoresis of PCR products for BVDV in wildlife. m is the molecular weight marker and number 1-5 are samples. 6 is positive control and 7 is negative control

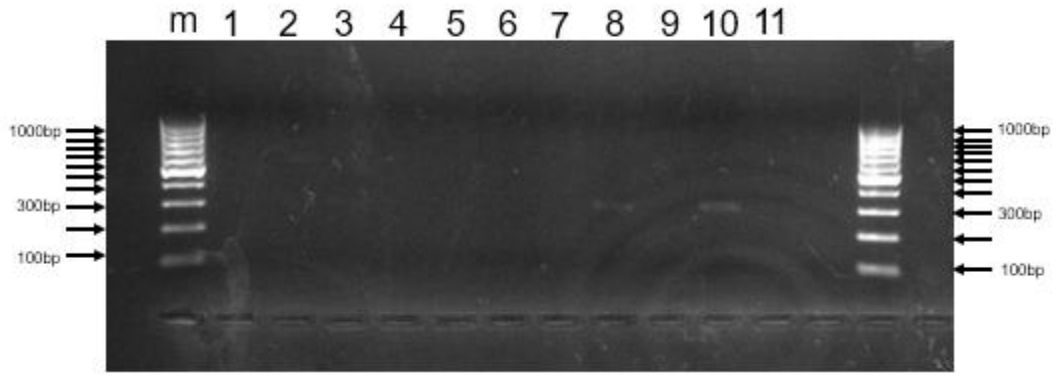


Plate III: Agorose gel electrophoresis of PCR products for BVDV in cattle. m is the molecular weight marker and number 1- 9 are samples. 10 is positive control and 11 is negetive control

Appendix III: Postmortem examination of Zebra for African Horse Sickness lesions in Sumu wildlife park Bauchi State, Nigeria

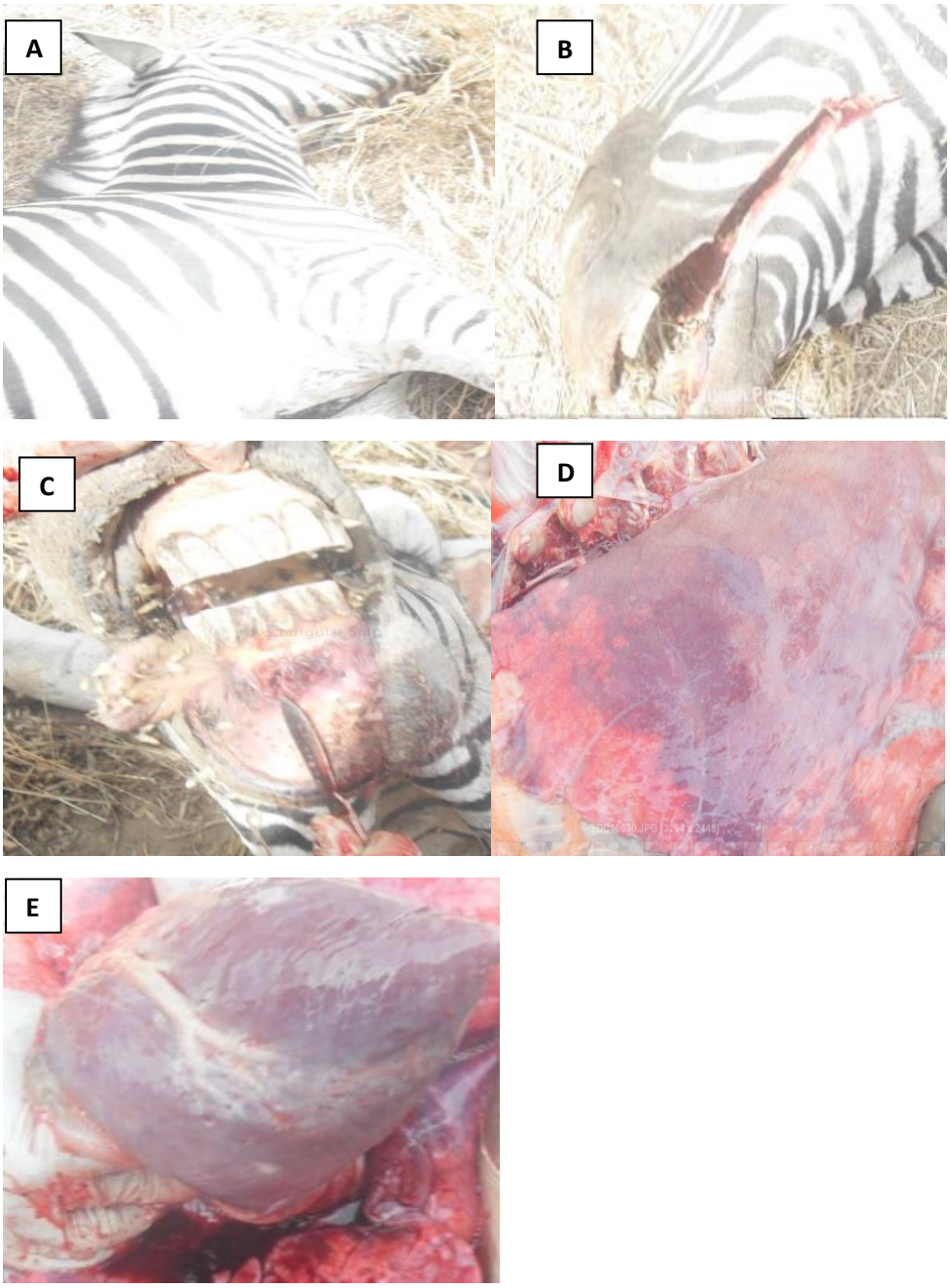
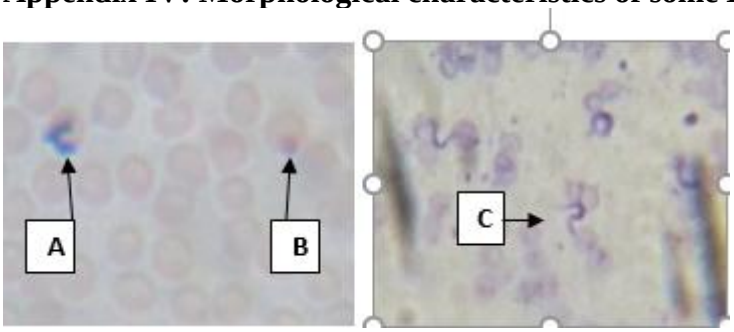


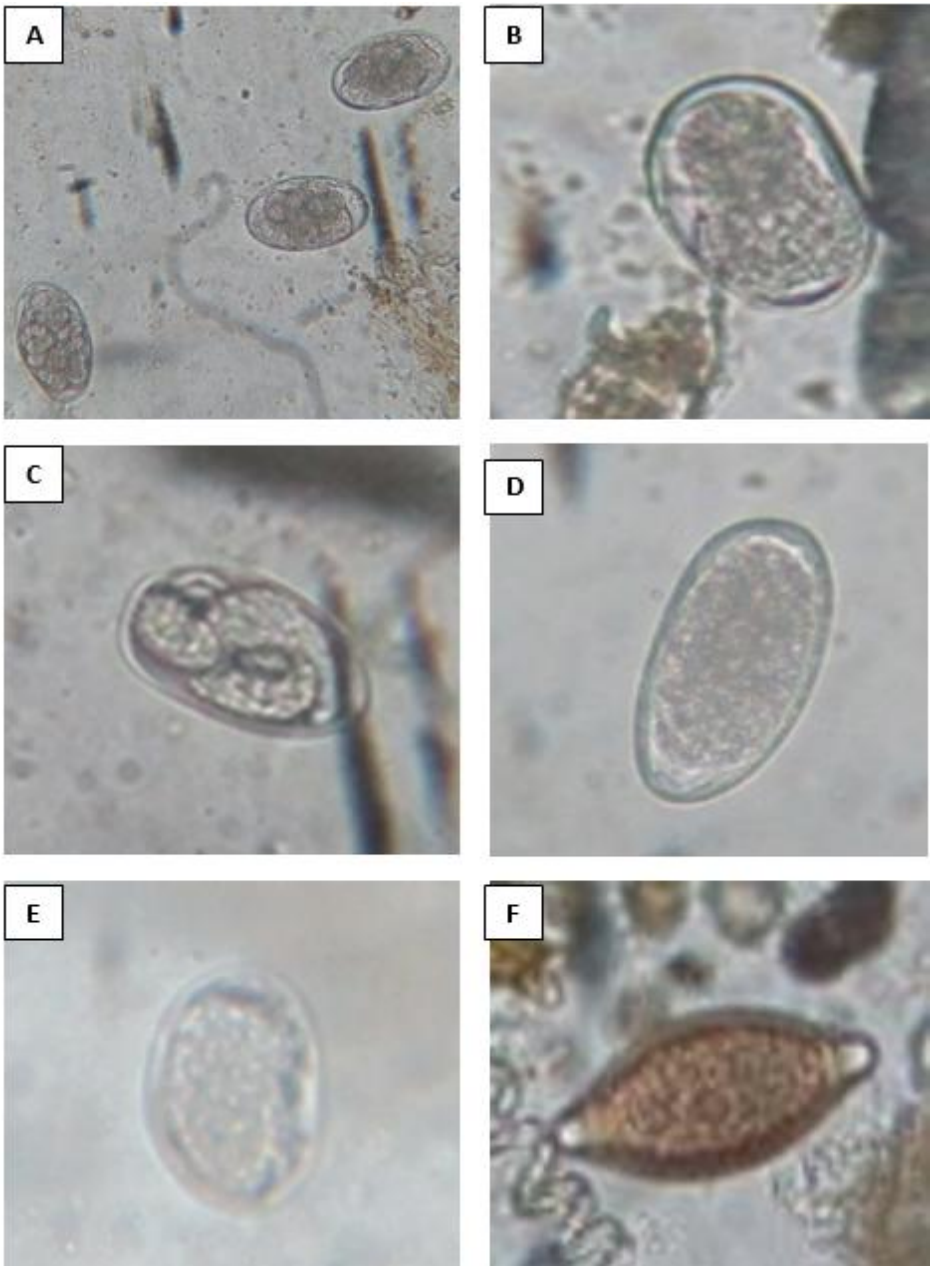
Plate II: Post-mortem examination of a zebra showing the whole carcass A, nasal discharge B, haemorrhagic gums C, Haemorrhagic lungs D, enlarged and haemorrhagic heart E.

Appendix IV: Morphological characteristics of some Haemoparasites



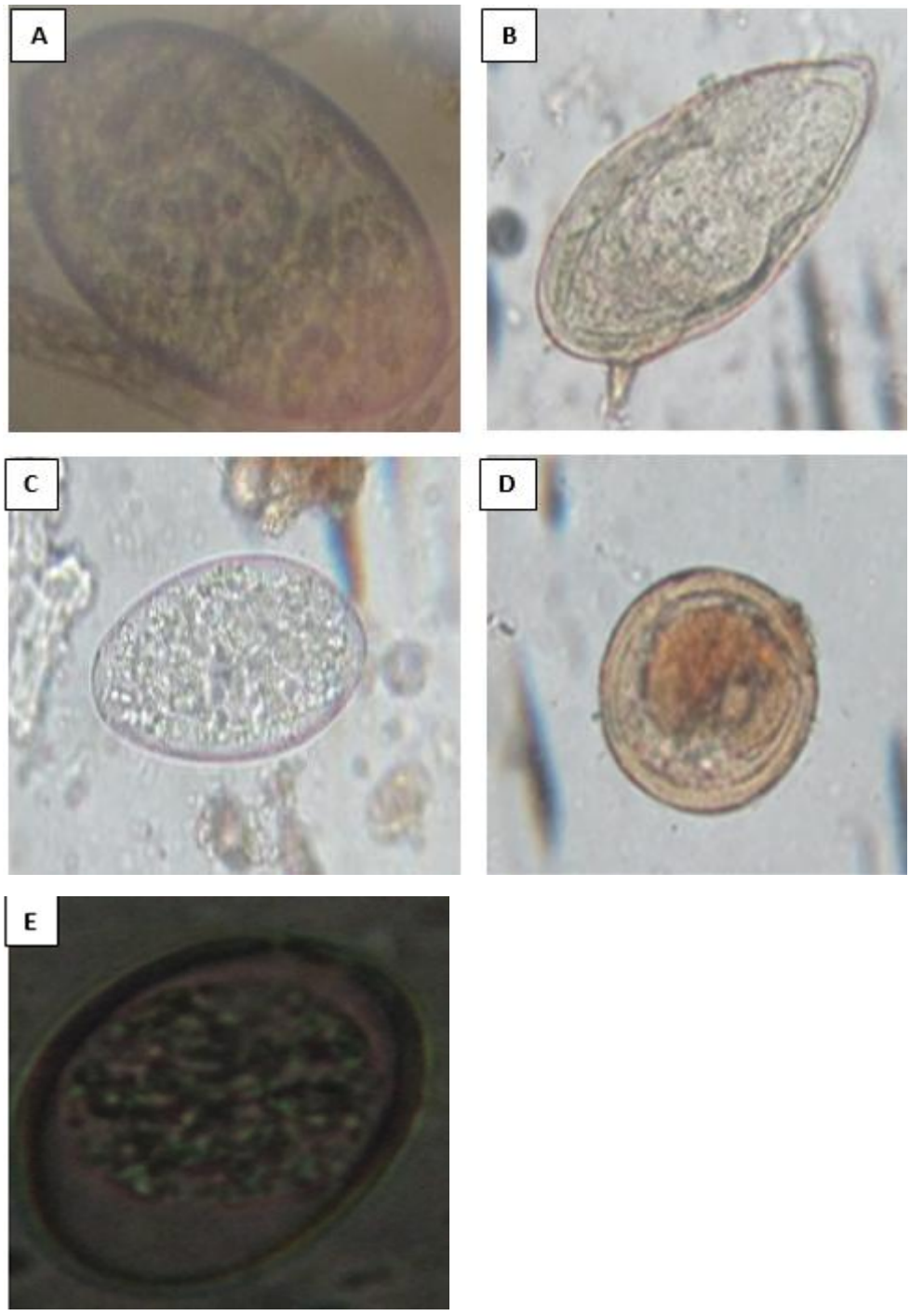
KEY: ***Babesia spp (A), Anaplasma marginale (B), and Trypanosome spp (C)***

Appendix V: Morphological characteristics of eggs of some gastrointestinal parasites from wild and domestic animals in and around the Yankari and Sumu wildlife parks in Bauchi State, Nigeria



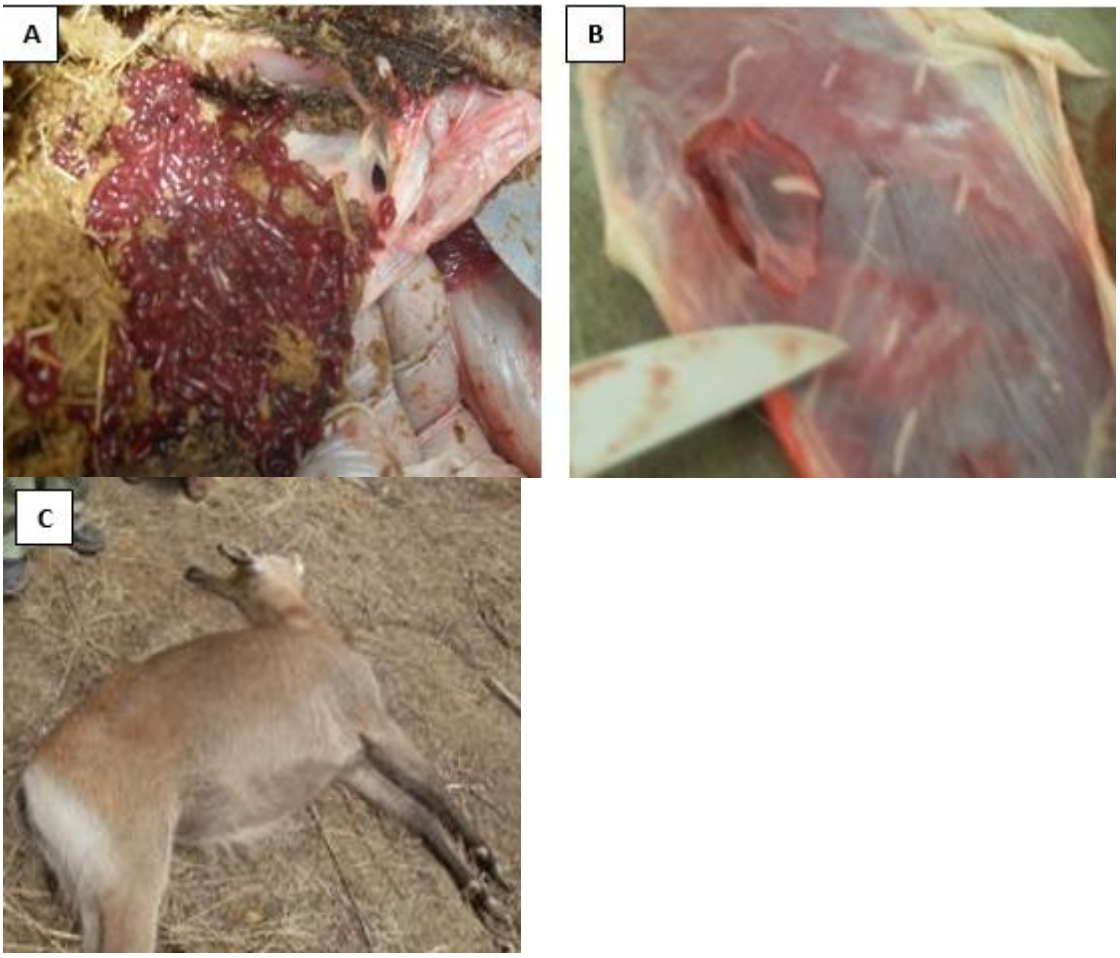
Key Eggs of some Helminths Parasites I: *Haemonchus contortus (A), Bunostomum spp (B), Strongyloides spp (C), Oesophagustomum spp (D), Ancylostoma spp (E), Trichuris trichuris (F).*

Appendix VI: Morphological characteristics of ova/oocyst of some gastrointestinal parasites from wild and domestic animals in and around the Yankari and Sumu wildlife Parks in Bauchi State, Nigeria



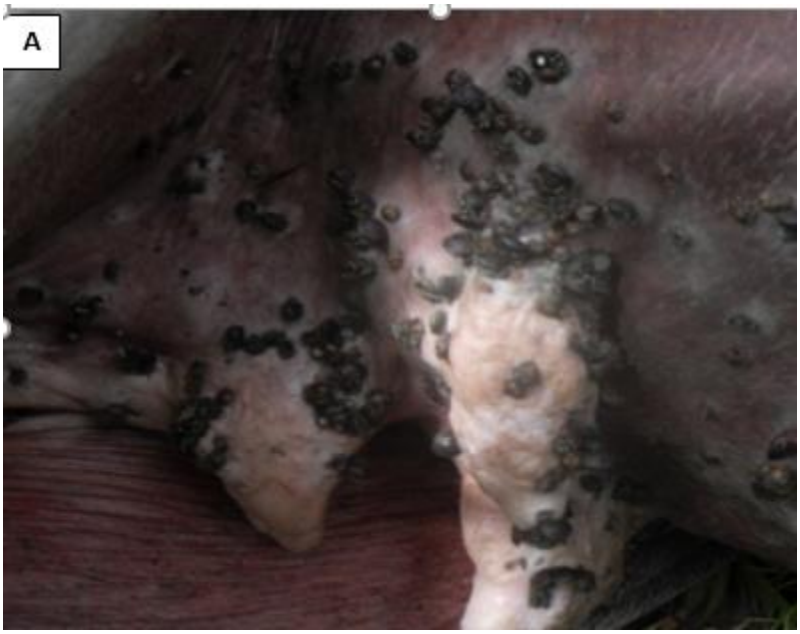
Key. *Paramphistomum cervi (A), Schistosoma spp (B), Fasciola* spp *(C), Taenia* spp *(D), Eimeria spp (E).*

Appendix VII: Morphology of adult worms in rumen and muscles of a waterbuck from Yankari game reserve in Bauchi State, Nigeria



Key Waterbuck at necropsy showing Adult *Paramphistomum* rumen flukes (A), Rice-grain sized cysts of *Sarcocystis* spp on skeletal muscle (B), Waterbuck carcass (C)

Appendix VIII: Ticks infestation on mammary gland of an Eland from Sumu Wildlife Park in Bauchi State, Nigeria



APPENDIX IX: Rift valley fever and Foot and mouth disease antibodies, Bovine viral diarrhoea antigen in cattle around Yankari and Sumu Wildlife Parks in Bauchi State, Nigeria

S/N	SEX	BREED	RVFV	FMDV	BVDV
1	M	WF	-Ve	-Ve	-VE
2	F	WF	-Ve	+Ve	-VE
3	M	WF	-Ve	-Ve	-VE
4	F	WF	-Ve	+Ve	+VE
5	F	WF	-Ve	+Ve	-VE
6	F	WF	+Ve	+Ve	-VE
7	F	WF	-Ve	+Ve	-VE
8	F	WF	-Ve	+Ve	+VE
9	M	WF	-Ve	+Ve	-VE
10	M	WF	-Ve	+Ve	-VE
11	M	WF	-Ve	+Ve	-VE
12	F	WF	-Ve	+Ve	-VE
13	F	WF	-Ve	+Ve	-VE
14	M	BK	-Ve	+Ve	-VE
15	F	BK	-Ve	+Ve	-VE
16	F	BK	+Ve	-Ve	-VE
17	M	BK	-Ve	-Ve	-VE
18	F	BK	-Ve	-Ve	-VE
19	F	WF	+Ve	+Ve	-VE
20	M	WF	-Ve	+Ve	-VE
21	M	WF	-Ve	-Ve	-VE
22	F	WF	-Ve	+Ve	+VE
23	M	WF	-Ve	-Ve	-VE
24	F	WF	-Ve	+Ve	+VE
25	F	WF	-Ve	+Ve	-VE
26	M	WF	-Ve	-Ve	-VE
27	F	RB	-Ve	+Ve	-VE
28	F	RB	-Ve	+Ve	-VE
29	M	RB	-Ve	-Ve	-VE
30	F	RB	-Ve	+Ve	-VE
31	F	RB	-Ve	-Ve	+VE
32	F	WF	-Ve	+Ve	-VE
33	M	WF	-Ve	+Ve	-VE
34	F	WF	-Ve	+Ve	-VE
35	M	RB	-Ve	+Ve	-VE
36	F	RB	-Ve	+Ve	-VE
37	F	RB	-Ve	+Ve	-VE
38	F	RB	-Ve	+Ve	-VE
39	M	RB	-Ve	+Ve	-VE
40	M	RB	-Ve	+Ve	-VE
41	F	RB	+Ve	+Ve	-VE
42	M	WF	-Ve	+Ve	-VE

43	F	WF	-Ve		-VE
44	F	WF	-Ve	+Ve	-VE
45	F	WF	-Ve	+Ve	-VE
46	F	WF	-Ve	+Ve	-VE
47	M	WF	-Ve	+Ve	+VE
48	F	WF	-Ve	+Ve	-VE
49	F	WF	-Ve	+Ve	-VE
50	M	WF	-Ve	+Ve	-VE
51	F	WF	+Ve	+Ve	-VE
52	M	WF	-Ve	-Ve	-VE
53	M	WF	-Ve	+Ve	-VE
54	F	WF	-Ve	+Ve	-VE
55	F	WF	-Ve	+Ve	-VE
56	M	RB	-Ve	+Ve	-VE
57	F	RB	-Ve	+Ve	-VE
58	F	RB	-Ve	-Ve	-VE
59	F	WF	+Ve	+Ve	-VE
60	M	WF	-Ve	+Ve	-VE
61	F	WF	-Ve	-Ve	-VE
62	M	WF	-Ve	-Ve	-VE
63	F	RB	-Ve	+Ve	-VE
64	F	RB	-Ve	-Ve	-VE
65	F	RB	-Ve	-Ve	-VE
66	F	WF	-Ve	+Ve	-VE
67	F	WF	-Ve	+Ve	-VE
68	F	WF	-Ve	+Ve	-VE
69	F	WF	-Ve	+Ve	+VE
70	M	WF	-Ve	-Ve	-VE
71	F	WF	-Ve	+Ve	-VE
72	M	WF	-Ve	+Ve	-VE
73	F	WF	-Ve	-Ve	-VE
74	F	WF	-Ve	+Ve	-VE
75	F	WF	-Ve	+Ve	-VE
76	F	BK	-Ve	-Ve	+VE
77	F	BK	-Ve	-Ve	-VE
78	F	BK	-Ve	-Ve	-VE
79	F	BK	-Ve	+Ve	-VE
80	F	BK	+Ve	+Ve	-VE
81	M	BK	-Ve	-Ve	-VE
82	F	BK	-Ve	+Ve	-VE
83	F	BK	-Ve	+Ve	-VE

Appendix X: Rift valley fever and Foot and mouth disease antibodies and Bovine viral diarrhoea antigen in Wildlife from Yankari and Sumu wildlife parks in Bauchi State, Nigeria.

S/N	WILDLIFE	SEX	RVFV	FMDV	BVDV
01	Waterbuck	F	+Ve	-Ve	-VE
02	Waterbuck	M	-Ve	-Ve	-VE
03	Waterbuck	M	+Ve	-Ve	-VE
04	Waterbuck	M	-Ve	+Ve	-VE
05	Waterbuck	F	-Ve	+Ve	Positive
06	Waterbuck	F	-Ve	-Ve	-VE
07	Waterbuck	M	+Ve	-Ve	-VE
08	Waterbuck	M	-Ve	-Ve	-VE
09	Waterbuck	F	-Ve	-Ve	Positive
10	Waterbuck	F	+Ve	-Ve	-VE
11	Waterbuck	M	-Ve	-Ve	-VE
12	Wildebeest	M	-Ve	+Ve	-VE
13	Wildebeest	F	-Ve	+Ve	-VE
14	Wildebeest	M	-Ve	-Ve	-VE
15	Wildebeest	F	+Ve	-Ve	Positive
16	Wildebeest	M	-Ve	+Ve	-VE
17	Wildebeest	M	-Ve	-VE	-VE
18	Wildebeest	M	-Ve	-VE	-VE
19	Wildebeest	F	-Ve	-Ve	-VE
20	Wildebeest	M	-Ve	-Ve	-VE
21	Wildebeest	M	-Ve	-Ve	-VE
22	Wildebeest	F	-Ve	+Ve	-VE
23	Wildebeest	M	-Ve	-Ve	-VE
24	Eland	M	-Ve	-Ve	-VE
25	Eland	F	-Ve	-Ve	-VE
26	Eland	F	+Ve	-Ve	-VE
27	Eland	M	-Ve	-Ve	-VE
28	Eland	M	-Ve	-Ve	-VE
29	Eland	F	-Ve	-Ve	Positive
30	Eland	M	-Ve	-Ve	-VE
31	Eland	M	-Ve	-Ve	-VE
32	Eland	F	-Ve	+Ve	-VE
33	Eland	F	+Ve	+Ve	Positive
34	Eland	M	-Ve	-Ve	-VE
35	Eland	M	-Ve	-Ve	-VE
36	Eland	F	-Ve	+Ve	-VE
37	Eland	F	-Ve	+Ve	-VE
38	Eland	F	-Ve	-Ve	-VE
39	Eland	M	-Ve	-Ve	Positive
40	Eland	M	-Ve	+Ve	-VE
41	Eland	M	-Ve	-Ve	-VE
42	Eland	M	+Ve	-Ve	-VE
43	Eland	M	-Ve	+Ve	-VE
44	Eland	M	-Ve	-Ve	-VE
45	Eland	F	-Ve	-Ve	-VE

46	Eland	F	-Ve	-Ve	-VE
47	Eland	M	-Ve	-Ve	-VE
48	Elephant	M	-Ve	-Ve	-VE
49	Elephant	M	-Ve	-Ve	-VE
50	Elephant	M	-Ve	+Ve	-VE
51	Elephant	F	+Ve	-Ve	-VE
52	W/ Heartbeest	M	-Ve	-Ve	Positive
53	Kudu	M	-Ve	-Ve	-VE
54	Zebra	M	-Ve		
55	Zebra	F	-Ve		
56	Zebra	M	-Ve		
57	Zebra	F	-Ve		
58	Zebra	M	-Ve		
59	Zebra	M	-Ve		
60	Zebra	M	-Ve		
61	Zebra	M	-Ve		
62	Zebra	F	-Ve		
63	Zebra	M	-Ve		
64	Zebra	M	-Ve		
65	Zebra	M	-Ve		
66	Zebra	M	-Ve		
67	Zebra	F	-Ve		
68	Zebra	F	-Ve		
69	Zebra	M	-Ve		
70	Zebra	F	-Ve		
71	Zebra	M	-Ve		
72	Zebra	M	-Ve		
73	Zebra	F	-Ve		
74	Zebra	M	-Ve		
75	Zebra	M	-Ve		
76	Zebra	M	-Ve		
77	Zebra	M	-Ve		
78	Zebra	F	-Ve		
79	Zebra	M	-Ve		
80	Zebra	F	-Ve		
81	Zebra	M	-Ve		
82	Zebra	M	-Ve		
83	Zebra	M	-Ve		
84	Zebra	M	-Ve		
85	Zebra	F	-Ve		
86	Zebra	M	-Ve		
87	Zebra	F	-Ve		
88	Zebra	F	-Ve		
89	Zebra	M	-Ve		
90	Zebra	M	-Ve		
91	Zebra	F	-Ve		
92	Zebra	F	-Ve		
93	Zebra	M	-Ve		

94	Zebra	F	-Ve		
95	Zebra	M	-Ve		
96	Zebra	F	-Ve		
97	Zebra	M	-Ve		
98	Zebra	M	-Ve		
99	Zebra	F	-Ve		
100	Zebra	M	-Ve		
101	Zebra	F	-Ve		
102	Zebra	M	-Ve		
103	Zebra	M	-Ve		
104	Zebra	F	-Ve		
105	Zebra	M	-Ve		
106	Zebra	F	-Ve		

Appendix XI: Serotypes of foot and mouth disease in wildlife from the Yankari and Sumu wildlife parks in Bauchi State, Nigeria

S/N	Sample No.	SPECIE	SEX	Serotype O	Serotype A	SAT 1	SAT 2
1	54	Waterbuck	F	-Ve	-Ve	-Ve	-Ve
2	55	Waterbuck	M	+Ve	-Ve	+Ve	-Ve
3	56	Waterbuck	M	+Ve	+Ve	+Ve	-Ve
4	57	Waterbuck	M	-Ve	-Ve	-Ve	-Ve
5	58	Waterbuck	F	-Ve	+Ve	+Ve	+Ve
6	59	Waterbuck	F	-Ve	-Ve	-Ve	-Ve
7	60	Waterbuck	M	-Ve	-Ve	-Ve	-Ve
8	65	Wildebeest	M	+Ve	-Ve	+Ve	-Ve
9	66	Wildebeest	F	-Ve	+Ve	+Ve	+Ve
10	67	Wildebeest	M	-Ve	-Ve	-Ve	-Ve
11	68	Wildebeest	F	-Ve	-Ve	-Ve	-Ve
12	69	Wildebeest	M	-Ve	-Ve	-Ve	-Ve
13	70	Wildebeest	M	+Ve	-Ve	+Ve	+Ve
14	71	Wildebeest	M	-Ve	-Ve	-Ve	-Ve
15	72	Wildebeest	F	-Ve	-Ve	-Ve	-Ve
16	73	Wildebeest	M	-Ve	-Ve	-Ve	-Ve
17	74	Wildebeest	M	-Ve	-Ve	-Ve	-Ve
18	75	Wildebeest	F	-Ve	-Ve	-Ve	-Ve
19	76	Wildebeest	M	-Ve	-Ve	-Ve	-Ve
20	77	Eland	M	-Ve	-Ve	-Ve	-Ve
21	78	Eland	F	-Ve	-Ve	-Ve	-Ve
22	79	Eland	F	+Ve	-Ve	-Ve	-Ve
23	80	Eland	M	-Ve	-Ve	-Ve	-Ve
24	81	Eland	M	-Ve	-Ve	-Ve	-Ve
25	82	Eland	F	-Ve	-Ve	-Ve	-Ve
26	83	Eland	M	-Ve	-Ve	-Ve	-Ve
27	84	Eland	M	-Ve	-Ve	-Ve	-Ve
28	85	Eland	F	+V	+Ve	+Ve	+Ve
29	86	Eland	F	-Ve	+Ve	-Ve	+Ve
30	87	Eland	M	-Ve	-Ve	-Ve	-Ve
31	88	Eland	M	-Ve	-Ve	-Ve	-Ve
32	89	Eland	F	-Ve	+Ve	-Ve	-Ve
33	90	Eland	F	-Ve	-Ve	-Ve	+Ve
34	91	Eland	F	-Ve	-Ve	-Ve	-Ve
35	92	Eland	M	-Ve	-Ve	-Ve	-Ve
36	93	Eland	M	-Ve	+Ve	+Ve	-Ve
37	94	Eland	M	+Ve	+Ve	+Ve	+Ve
38	95	Eland	M	-Ve	-Ve	-Ve	-Ve
39	96	Eland	M	+Ve	+Ve	+Ve	-Ve
40	97	Eland	M	-Ve	-Ve	-Ve	-Ve
41	98	Eland	F	-Ve	-Ve	-Ve	-Ve
42	99	Eland	F	-Ve	-Ve	-Ve	-Ve
43	100	Eland	M	-Ve	-Ve	-Ve	-Ve
44	101	Elephant	M	-Ve	-Ve	-Ve	-Ve
45	102	Elephant	M	-Ve	+Ve	-Ve	+Ve
46	103	Elephant	M	-Ve	-Ve	-Ve	-Ve
47	104	Elephant	F	-Ve	-Ve	-Ve	-Ve
48	105	Heartbeest	M	-Ve	-Ve	-Ve	-Ve
49	106	Kudu	M	-Ve	-Ve	-Ve	-Ve

Appendix XII: African horse sickness virus antibodies in horses around Yankari and Sumu wildlife parks in Bauchi State, Nigeria

S/N	AGE	SEX	BREED	AHSV RESULT
1	Adult	Male	Local	-Ve
2	Adult	Male	Local	-Ve
3	Adult	Male	Local	-Ve
4	Adult	Male	Local	-Ve
5	Adult	Male	Local	-Ve
6	Adult	Male	Local	-Ve
7	Adult	Female	Local	Positive
8	Adult	Male	Local	Positive
9	Adult	Male	Local	-Ve
10	Adult	Male	Local	Positive
11	Adult	Male	Local	-Ve
12	Adult	Male	Local	-Ve
13	Young	Male	Crossed	-Ve
14	Adult	Male	Crossed	-Ve
15	Adult	Male	Local	-Ve
16	Adult	Male	Local	-Ve
17	Adult	Male	Local	Positive
18	Adult	Male	Local	Positive
19	Adult	Male	Agentine	-Ve
20	Adult	Male	Local	Positive
21	Adult	Male	Local	-Ve
22	Adult	Male	Local	-Ve
23	Adult	Male	Local	-Ve
24	Adult	Male	Local	-Ve
25	Adult	Male	Crossed	-Ve
26	Adult	Male	Crossed	-Ve
27	Young	Male	Local	Positive
28	Young	Male	Local	Positive
29	Young	Male	Local	-Ve
30	Young	Male	Local	Positive
31	Young	Male	Local	-Ve
32	Young	Male	Local	-Ve
33	Adult	Male	Local	-Ve
34	Adult	Male	Local	-Ve
35	Young	Male	Local	-Ve
36	Adult	Male	Local	-Ve
37	Adult	Male	Local	Positive
38	Adult	Male	Local	Positive
39	Adult	Male	Crossed	-Ve
40	Adult	Male	Local	-Ve
41	Adult	Male	Local	Positive
42	Adult	Male	Local	-Ve
43	Adult	Male	Local	-Ve
44	Adult	Male	Local	-Ve
45	Adult	Male	Local	-Ve
46	Adult	Male	Local	-Ve
47	Adult	Male	Local	-Ve
48	Adult	Male	Local	Positive
49	Adult	Male	Local	Positive
50	Adult	Male	Local	-Ve

51	Adult	Female	Local	-Ve
52	Adult	Female	Local	Positive
53	Adult	Female	Local	-Ve
54	Adult	Female	Local	-Ve
55	Adult	Male	Local	-Ve
56	Adult	Male	Local	-Ve
57	Adult	Male	Local	-Ve
58	Adult	Male	Local	-Ve
59	Adult	Male	Local	Positive
60	Adult	Male	Local	Positive
61	Adult	Male	Local	-Ve
62	Adult	Male	Local	Positive
63	Adult	Male	Crossed	-Ve
64	Adult	Male	Crossed	-Ve
65	Adult	Male	Local	Positive
66	Adult	Male	Local	-Ve
67	Young	Male	Local	-Ve
68	Adult	Male	Local	-Ve
69	Adult	Male	Local	-Ve
70	Adult	Male	Local	-Ve
71	Adult	Male	Local	Positive
72	Adult	Male	Local	-Ve
73	Adult	Male	Local	Positive
74	Adult	Male	Local	-Ve
75	Adult	Male	Local	-Ve
76	Adult	Male	Local	Positive
77	Adult	Male	Local	-Ve
78	Adult	Male	Local	-Ve
79	Young	Male	Local	Positive
80	Young	Male	Local	-Ve
81	Adult	Male	Local	Positive
82	Adult	Male	Local	-Ve
83	Adult	Male	Local	-Ve
84	Adult	Male	Local	Positive
85	Young	Male	Local	-Ve
86	Young	Male	Local	-Ve
87	Adult	Male	Local	Positive
88	Adult	Male	Local	-Ve
89	Adult	Male	Local	-Ve
90	Adult	Male	Local	Positive
91	Adult	Male	Local	-Ve
92	Adult	Male	Local	Positive
93	Adult	Male	Local	-Ve
94	Adult	Male	Local	-Ve
95	Adult	Male	Local	Positive
96	Adult	Male	Local	Positive
97	Adult	Male	Local	-Ve
98	Adult	Male	Local	-Ve
99	Adult	Male	Local	Positive
100	Adult	Male	Local	-Ve
101	Adult	Male	Local	-Ve
102	Adult	Male	Local	-Ve
103	Adult	Male	Local	Positive

104	Adult	Male	Local	Positive
105	Adult	Male	Local	-Ve
106	Adult	Male	Local	-Ve
107	Adult	Male	Local	-Ve
108	Adult	Female	Local	Positive
109	Adult	Female	Local	-Ve
110	Adult	Female	Local	-Ve
111	Adult	Female	Local	Positive
112	Adult	Male	Local	Positive
113	Adult	Male	Crossed	-Ve
114	Adult	Male	Local	-Ve
115	Adult	Male	Local	-Ve
116	Adult	Male	Local	-Ve
117	Adult	Male	Local	Positive
118	Adult	Male	Local	Positive
119	Adult	Male	Local	-Ve
120	Adult	Male	Local	-Ve
121	Adult	Male	Local	Positive
122	Adult	Male	Local	-Ve
123	Adult	Male	Local	-Ve
124	Adult	Male	Local	-Ve
125	Adult	Male	Local	-Ve
126	Adult	Male	Local	Positive
127	Adult	Male	Local	-Ve
128	Adult	Male	Local	Positive
129	Adult	Male	Local	Positive
130	Adult	Male	Local	-Ve
131	Young	Male	Local	-Ve
132	Adult	Male	Local	Positive
133	Adult	Male	Local	-Ve
134	Adult	Male	Local	-Ve
135	Adult	Male	Local	-Ve
136	Adult	Male	Local	-Ve
137	Adult	Male	Crossed	-Ve
138	Adult	Male	Crossed	-Ve
139	Adult	Male	Local	Positive
140	Adult	Male	Local	-Ve
141	Adult	Male	Local	-Ve
142	Adult	Male	Local	Positive
143	Young	Male	Local	-Ve
144	Young	Female	Local	-Ve
145	Adult	Male	Local	Positive
146	Adult	Male	Local	Positive
147	Adult	Male	Local	-Ve
148	Adult	Male	Local	Positive
149	Young	Male	Local	-Ve
150	Young	Male	Local	-Ve
151	Young	Male	Local	Positive
152	Adult	Male	Local	-Ve
153	Adult	Male	Local	Positive
154	Adult	Male	Local	-Ve
155	Adult	Male	Local	Positive
156	Adult	Male	Local	-Ve

157	Adult	Male	Local	-Ve
158	Adult	Male	Local	Positive
159	Adult	Male	Local	Positive
160	Adult	Male	Local	-Ve
161	Adult	Female	Local	-Ve
162	Adult	Female	Local	-Ve
163	Adult	Male	Local	-Ve
164	Adult	Male	Local	-Ve
165	Adult	Male	Local	Positive
166	Adult	Male	Local	Positive
167	Adult	Male	Local	-Ve
168	Adult	Male	Local	-Ve
169	Adult	Male	Local	-Ve
170	Adult	Male	Local	Positive
171	Adult	Male	Local	-Ve
172	Adult	Male	Local	-Ve
173	Adult	Male	Local	Positive
174	Adult	Male	Local	-Ve
175	Adult	Male	Local	-Ve
176	Adult	Male	Local	Positive
177	Adult	Male	Local	Positive
178	Adult	Male	Local	-Ve
179	Adult	Male	Local	Positive
180	Adult	Male	Local	Positive
181	Adult	Male	Local	Positive
182	Adult	Male	Local	-Ve
183	Adult	Female	Local	-Ve
184	Adult	Female	Local	Positive
185	Adult	Male	Local	Positive
186	Adult	Male	Local	-Ve
187	Adult	Male	Local	-Ve
188	Adult	Male	Crossed	Positive
189	Adult	Male	Local	-Ve
190	Adult	Male	Local	-Ve
191	Adult	Male	Local	Positive
192	Adult	Male	Local	Positive
193	Adult	Male	Local	-Ve
194	Adult	Male	Local	Positive
195	Adult	Male	Local	Positive
196	Young	Male	Local	-Ve
197	Adult	Male	Local	-Ve
198	Adult	Male	Local	Positive
199	Adult	Male	Local	Positive
200	Adult	Female	Local	-Ve

Appendix XIII: African horse sickness antibodies in zebras from Sumu wildlife park in Bauchi State, Nigeria.

S/N	SPECIE	SEX	AHSV RESULTS
01	Zebra	M	-VE
02	Zebra	F	Positive
03	Zebra	M	-VE
04	Zebra	F	-VE
05	Zebra	M	-VE
06	Zebra	M	-VE
07	Zebra	M	Positive
08	Zebra	M	-VE
09	Zebra	F	-VE
10	Zebra	M	-VE
11	Zebra	M	-VE
12	Zebra	M	Positive
13	Zebra	M	-VE
14	Zebra	F	-VE
15	Zebra	F	-VE
16	Zebra	M	-VE
17	Zebra	F	-VE
18	Zebra	M	Positive
19	Zebra	M	Positive
20	Zebra	F	-VE
21	Zebra	M	-VE
22	Zebra	M	-VE
23	Zebra	M	-VE
24	Zebra	M	-VE
25	Zebra	F	-VE
26	Zebra	M	-VE
27	Zebra	F	-VE
28	Zebra	M	Positive
29	Zebra	M	-VE
30	Zebra	M	-VE
31	Zebra	M	-VE
32	Zebra	F	-VE
33	Zebra	M	-VE
34	Zebra	F	-VE
35	Zebra	F	-VE
36	Zebra	M	Positive
37	Zebra	M	Positive
38	Zebra	F	-VE
39	Zebra	F	Positive
40	Zebra	M	-VE
41	Zebra	F	-VE
42	Zebra	M	-VE
43	Zebra	F	-VE
44	Zebra	M	Positive
45	Zebra	M	-VE
46	Zebra	F	-VE
47	Zebra	M	-VE
48	Zebra	F	-VE
49	Zebra	M	Positive
50	Zebra	M	-VE
51	Zebra	F	-VE
52	Zebra	M	-VE
53	Zebra	F	-VE

Appendix XIV: Haemo and endo parasites of cattle around Yankari game reserve and Sumu Wildlife park in Bauchi State, Nigeria

S/N	SEX	BREED	HAEMOPARASITES	GIT PARASITES	PCV
01	M	WF	*Trypanosoma spp* + *Babesia bigemina* +	Ova of *Oesophagostomum spp* ++	22
02	F	WF	-VE	Ova of *Fasciola* spp+ Ova of *Strongyle* type-egg ++	45
03	M	WF	*Anaplasma marginale* ++	Ova of *Fasciola* spp+	43
04	F	WF	-VE	Ova of *Fasciola* spp+	42
05	F	WF	*Babesia bigemina* +	Ova of *Fasciola* spp+++ Oocyst of *Eimeria spp* ++	19
06	F	WF	-VE	Ova of *Oesophagostomum* spp+ Ova of *Strongyle* type-egg ++	40
07	F	WF	-VE	-VE	46
08	F	WF	-VE	Ova of *Strongyle* type-egg + Ova of *Bunostomum* spp +	42
09	M	WF	-VE	Ova of *Fasciola* spp+ Ova of *Strongyloides* spp++	43
10	M	WF	*Trypanosoma spp* + *Babesia bigemina* ++	Ova of *Fasciola* spp ++ Oocyst of *Eimeria* spp ++ Ova of *Strongyle* type-egg +	20
11	M	WF	-VE	-VE	38
12	F	WF	*Babesia bovis*+	Ova of *Fasciola* spp+ Ova of *Moniezia* spp+ Ova of *Strongyle* type-egg +	33
13	F	WF	*Anaplasma marginale* ++	Ova of *Cooperia spp* ++ Ova of *Bunostomum* spp+ Ova of *Eimeria* spp +	35
14	M	BK	-VE	-VE	40
15	F	BK	*Babesia bigemina* +	Ova of *Fasciola* spp+ Ova of *Strongyle* type-egg ++ Ova of *Moniezia* spp +	36
16	F	BK	-VE	Ova of *Fasciola* spp +++	42
17	M	BK	-VE	Ova of *Taena* spp ++ Ova of *Eimeria* spp +	40
18	F	BK	*Babesia bovis* +	-VE	30
19	F	WF	*Babesia bigemina* + *Anaplasma marginale* +	Ova of *Fasciola* spp + Oocyst of *Eimeria* spp +	24
20	M	WF	-VE	-VE	36
21	M	WF	-VE	Ova of *Fasciola* spp ++ Ova of *Strongyle* type-egg ++	40
22	F	WF	*Babesia bigemina* +	-VE	30
23	M	WF	*Babesia bigemina* +	Ova of *Fasciola* spp ++ Ova of *Taena* spp +	36
24	F	WF	*Babesia bovis* + *Anaplasma marginale* ++	-VE	24
25	F	WF	-VE	Ova of *Bunostomum* spp+ Ova of *Eimeria* spp +	39

26	M	WF	-VE	Ova of *Fasciola* spp + Ova of *Moniezia benedeni* + Ova of *Strongyle-type egg* +	36
27	F	RB	*Babesia bigemina* +++	Ova of *Fasciola* spp ++ Ova of *Paramphistomum* spp +	28
28	F	RB	*Babesia bigemina* +	Ova of *Oesophagostomum* spp ++	36
29	M	RB	-VE	-VE	40
30	F	RB	*Trypanosoma spp* +	Oocyst of *Eimeria spp* +++ Ova of *Strongyle* type-egg + Ova of *Paramphistomum* spp +	23
31	F	RB	-VE	-VE	48
32	F	WF	-VE	Ova of *Fasciola* spp ++ Ova of *Paramphistomum* spp +	40
33	M	WF	*Babesia bigemina* +	Ova of *Fasciola* spp + Ova of *Strongyle* type-egg++	34
34	F	WF	-VE	Ova of *Fasciola* spp + Oocyst of *Eimeria spp* +	39
35	M	RB	-VE	-VE	39
36	F	RB	-VE	Ova of *Fasciola* spp + Ova of *Bunostomum* spp +	40
37	F	RB	*Anaplasma marginale* ++	Ova of *Oesophagostomum* spp ++ Ova of *Strongyle-type egg* ++	27
38	F	RB	-VE	-VE	40
39	M	RB	*Babesia bovis* + *Anaplasma marginale* +	-VE	28
40	M	RB	-VE	Ova of *Fasciola* spp + Ova of *Oesophagostomum* spp ++	29
41	F	RB	-VE	-VE	40
42	M	WF	*Babesia bigemina* +	Ova of *Paramphistomum* spp +++	38
43	F	WF	-VE	Ova of *Fasciola* spp + Ova of *Haemonchus spp* ++	36
44	F	WF	-VE	Ova of *Fasciola spp*+ Ova of *Taena* spp +	38
45	F	WF	*Babesia bigemina* +	-VE	27
46	F	WF	-VE	Ova of *Fasciola* spp + Ova of *Bunostomum spp* + Ova of *Strongyle egg-type spp*+	39
47	M	WF	*Trypanosoma spp* ++	-VE	26
48	F	WF	-VE	-VE	38
49	F	WF	*Babesia bigemina* ++ *Anaplasma marginale* ++	Ova of *Oesophagostomum spp* + Ova of *Strongyle-type egg* + Ova of *Fasciola spp*+	23
50	M	WF	-VE	Ova of *Fasciola spp*+ Ova of *Bunostomum spp* + Ova of *Strongyloides spp* ++	38
51	F	WF	-VE	-VE	39
52	M	WF	*Trypanosoma spp* ++	Ova of *Strongyle-type egg* + Ova of *Bunostomum spp* +	26

53	M	WF	-VE	Ova of *Toxocara spp* + Ova of *Paramphistomum spp* ++	38
54	F	WF	-VE	-VE	38
55	F	WF	*Babesia bovis* +	Ova of *Strongyloides spp* ++	27
56	M	RB	-VE	Ova of *Fasciola spp*+ Ova of *Oesophagostomum spp* + Ova of *Toxocara spp* +	40
57	F	RB	*Babesia bovis* + *Anaplasma marginale* +	Ova of *Fasciola spp*+ Ova of *Strongyle egg-type*++	26
58	F	RB	-VE	-VE	39
59	F	WF	*Anaplasma marginale* + *Babesia bigemina* +	Ova of *Strongyle-type egg* + Ova of *Fasciola gigatntica* ++ Ova of *Eimeria* spp +	27
60	M	WF	-VE	Ova of *Fasciola spp* + Ova of *Haemonchus spp* +	39.5
61	F	WF	*Trypanosoma spp* ++	Ova of *Paramphistomum spp* +	24.5
62	M	WF	-VE	-VE	38
63	F	RB	-VE	Ova of *Fasciola spp*+ Ova of *Haemonchus spp* + Ova of *Strongyloides spp* +	38
64	F	RB	*Babesia bigemina* +	-VE	31
65	F	RB	-VE	-VE	40
66	F	WF	-VE	Ova of *Fasciola spp*+ Ova of *Strongyle-type egg* + Ova of *Eimeria* spp +	38
67	F	WF	*Babesia bigemina* +	Ova of *Fasciola* spp +++ Ova of *Oesophagostomum spp* +	29
68	F	WF	-VE	Ova of *Toxocara spp* + Ova of *Strongyloides spp* ++	31
69	F	WF	-VE	-VE	41
70	M	WF	*Babesia bigemina* +	-VE	32
71	F	WF	*Babesia bovis*++	Ova of *Fasciola spp* + ++ Ova of *Strongyle egg-type*+	30
72	M	WF	*Anaplasma marginale* ++	-VE	32
73	F	WF	-VE	-VE	42
74	F	WF	*Anaplasma marginale* +	Ova of *Fasciola spp* + Ova of *Strongyloides spp* +	38
75	F	WF	-VE	-VE	39
76	F	BK	*Babesia bovis* +	Ova of *Fasciola spp* +++ Ova of *Eimeria* spp + Ova of *Taenia* spp	28
77	F	BK	*Trypanosoma spp* + *Anaplasma marginale* +	Ova of *Fasciola spp* + Ova of *Haemonchus spp* +	19
78	F	BK	-VE	Ova of *Fasciola spp* ++ Ova of *Taenia* spp +	40
79	F	BK	*Babesia bigemina* + *Anaplasma marginale* +	-Ve	28
80	F	BK	-VE	Ova of *Fasciola spp* +	41

81	M	BK	-VE	-VE	42
82	F	BK	-VE	Ova of *Strongyloides spp* + Ova of *Paramphistomum spp* +	40
83	F	BK	*Anaplasma marginale* + *Trypanosoma spp* ++	Ova of *Oesophagostomum spp* ++	24
84	F	BK	-VE	Ova of *Fasciola spp*+ Ova of *Taenia spp* +	36
85	F	BK	-VE	-VE	38
86	F	BK	-VE	-VE	38
87	F	BK	*Anaplasma marginale* +	Ova of *Fasciola spp* + Ova of *Paramphistomum spp* +	34
88	F	WF	*Babesia bovis* ++ *Anaplasma marginale* +	Ova of *Fasciola spp* + Ova of *Strongyle* type-egg ++	24
89	F	WF	-VE	-VE	39
90	F	WF	-VE	Ova of *Fasciola spp*+ Ova of *Bunostomum* spp+ Ova of *Dicrocoelium dendriticum* +	38
91	M	WF	-VE	-VE	40
92	F	WF	*Trypanosoma spp* + Babesia bigemina +	-VE	22
93	F	WF	-VE	Ova of *Fasciola spp* + Ova of *Oesophagostomum spp* +	39
94	F	RB	*Anaplasma marginale* +	Ova of *Fasciola spp* +	33
95	F	RB	-VE	-VE	40
96	F	RB	*Babesia bigemina* +	Ova of *Fasciola spp*+ Ova of *Eimeria* spp ++	28.5
97	M	WF	-VE	Ova of *Bunostomum spp* + Ova of *Fasciola spp*+ Ova of *Strongyloides spp* ++	36.5
98	M	WF	*Anaplasma marginale* +	-VE	35
99	F	WF	-VE	-VE	40
100	F	WF	-VE	Trophozoites of *Balantidium coli* +	39
101	F	WF	*Trypanosoma spp* +	Ova of *Fasciola spp* +++ Ova of *Oesophagostomum spp* +	25
102	F	WF	-VE	-VE	40
103	F	WF	*Babesia bovis*++	Ova of *Fasciola spp* profused Ova of *Bunostomum spp* +	26
104	M	WF	-VE	-VE	40
105	F	WF	-VE	Ova of *Fasciola spp* ++ Ova of *Strongyle egg-type*++	39
106	F	WF	*Babesia bigemina* +	-VE	36
107	F	WF	-VE	Ova of *Fasciola spp*+ Ova of *Taenia spp* ++	37
108	M	WF		Ova of *Oesophagostomum spp* ++	36
109	F	WF	*Babesia bovis* +	-Ve	30
110	F	BK	*Babesia bigemina* +	-Ve	30

111	M	BK	-VE	Ova of *Fasciola spp*++ Ova of *Paramphistomum spp* + Ova of *Strongyle-type egg*	40
112	F	BK	-VE	Ova of *Toxocara spp* ++	40
113	F	BK	-VE	-VE	41
114	M	BK	-VE	Ova of *Fasciola spp* ++ Ova of *Dicrocoelium dendriticum* ++	39
115	F	WF	*Anaplasma marginale* + *Babesia bigemina* +	-Ve	30
116	M	WF	-VE	Ova of *Haemonchus spp* ++ Ova of *Paramphistomum spp* ++	39.5
117	F	WF	-VE	Ova of *Fasciola spp* profused Ova of *Strongyloides spp* +	38
118	F	WF	-VE	Ova of *Moniezia benedeni*++	38
119	F	RB	-VE	-VE	41
120	F	RB	*Trypanosoma spp* +	Ova of *Fasciola spp* +++ Ova of *Oesophagostomum spp*++	25
121	F	RB	*Anaplasma marginale* +	Ova of Fasciola spp + Ova of *Haemonchus spp* + Ova of *Strongyle-type egg* ++	26
122	F	RB	-VE	Ova of *Fasciola spp* + Ova of *Oesophagostomum spp* ++	39
123	F	BK	*Trypanosoma spp* +	Ova of *Fasciola spp* + Ova of *Strongyle-type egg* + Ova of *Toxocara spp* + Ova of *Bunostomum spp* +++	30
124	F	BK	-VE	Ova of *Bunostomum spp* +	39
125	F	BK	*Babesia bigemina* + *Anaplasma marginale* +	-VE	29
126	M	BK	-VE	Ova of *Fasciola spp* + + Ova of *Strongyle-type egg* +	36
127	F	BK	-VE	-VE	48
128	M	BK		-VE	38
129	F	BK	*Anaplasma marginale*+++	-VE	41
130	F	BK	-VE	Ova of *Fasciola spp* + Ova of *Strongyle egg-type*++ Ova of *Paramphistomum spp* +	42
131	F	WF	*Babesia bovis* ++	Ova of *Oesophagostomum spp* + Ova of *Fasciola spp* +	33
132	M	WF	-VE	-VE	39
133	M	WF	-VE	Ova of *Strongyle egg-type*++	39.5
134	F	WF	*Babesia bigemina* +	Ova of *Fasciola spp* ++ Trophozoites of *Balantidium coli* +	27
135	F	WF	*Babesia bigemina* ++	Ova of *Moniezie* spp +	33
136	F	WF	-VE	Ova of *Fasciola* spp++ Ova of *Moniezie* spp + Ova of *Strongyle-type egg* ++	35
137	F	WF	*Trypanosoma spp* ++	-VE	30

138	F	WF	-VE	Ova of *Fasciola* spp + Ova of *Cooperia* spp	38
139	F	WF	-VE	Ova of *Fasciola* spp+ Ova of *Oesophagostomum* spp+	39
140	F	WF	*Babesia bigemina* ++	-VE	31
141	M	WF	-VE	Ova of *Fasciola spp*++ Ova of *Oesophagostomum* spp +	39
142	F	WF	-VE	Ova of *Fasciola* spp + Ova of *Taenia spp* +	38
143	M	WF	-VE	*Ova of Strongyloides* spp ++	39
144	M	WF	*Anaplasma marginale* +	-VE	41
145	F	WF	*Babesia bovis* +	Ova of *Fasciola* spp + Ova of *Moniezia* spp+ Ova of *Strongyle-type egg* +++	27
146	F	WF	*Babesia bovis* + *Anaplasma marginale* +	Ova of *Fasciola spp* + Ova of *Oesophagostomum spp* +	30
147	M	WF	-VE	-VE	48
148	F	WF	-VE	Ova of *Fasciola spp* ++ Ova of *Moniezia benedeni* + Ova of *Dicrocoelium dendriticum* +	40
149	M	WF	*Babesia bigemina* +	Ova of *Fasciola spp* +	34
150	F	WF	-VE	-VE	42
151	M	WF	-VE	Ova of *Fasciola spp* ++ Ova of *Paramphistomum spp* +	39
152	F	WF	*Babesia bigemina* +	-VE	38
153	F	WF	-VE	-VE	46
154	M	WF	*Trypanosoma spp* ++	Ova of *Fasciola spp* + Ova of *Moniezia benedeni* +	26
155	F	WF	-VE	Ova of *Bunostomum spp* +	38
156	F	WF	*Babesia bigemina* +++	Ova of *Oesophagostomum spp* +	29
157	F	WF	-VE	Ova of *Eimeria* spp profused	38
158	M	WF	-VE	-VE	39
159	F	WF	*Trypanosoma spp* ++	Ova of *Fasciola spp* + Trophozoites of *Balantidium coli* +	24
160	F	WF	-VE	Ova of *Fasciola spp* + Ova of *Haemonchus spp* +	41
161	M	WF	-VE	Ova of *Oesophagostomum spp* + Oocyst of *Eimeria spp* ++ Ova of *Fasciola spp* ++	40
162	F	WF	*Babesia bovis* +	-VE	34
163	F	WF	-VE	Ova of *Cooperia* spp ++	39
164	F	WF	*Babesia bovis* +	-Ve	33
165	F	WF	-VE	Ova of *Fasciola spp* + Trophozoites of *Balantidium coli* + Ova of *Paramphistomum spp* ++	39
166	F	RB	*Anaplasma marginale* +	Ova of *Fasciola spp*+	27

			Babesia bigemina +	Ova of *Bunostomum spp* + Ova of *Strongyloides spp* +	
167	M	RB	-VE	-VE	39.5
168	M	RB	*Trypanosoma spp* ++	Ova of *Fasciola spp* + Ova of *Oesophagostomum spp* +	33.5
169	F	RB	-VE	-VE	38.6
170	M	RB	-VE	Ova of *Fasciola spp* ++ Ova of *Eimeria* spp +	39
171	F	RB	*Babesia bigemina* +	Ova of *Fasciola spp*+ ++ Ova of *Oesophagostomum spp* +	30
172	M	RB	-VE	Ova of *Fasciola spp* +++ Ova of *Dicrocoelium dendriticum* ++	33
173	F	WF	-VE	Ova of *Fasciola spp*+ Ova of *Oesophagostomum spp* +	39
174	M	WF	*Babesia bigemina* + *Anaplasma marginale* ++	Ova of *Fasciola spp* +++ Ova of *Strongyle egg-type*+	27
175	F	WF	-VE	-VE	39.5
176	M	RB	-VE	Ova of *Eimeria* spp +	40
177	F	RB	*Babesia bigemina* +	Ova of *Moniezia benedeni*++	38
178	F	RB	*Babesia bovis*++	Ova of *Fasciola spp* +++ Ova of *Dicrocoelium dendriticum* + Ova of *Cooperia* spp +	33
179	F	RB	-VE	Ova of *Toxocara spp* + Ova of *Strongyloides spp* +	35
180	F	RB	-VE	Ova of *Fasciola spp* ++ Ova of *Taena* spp + Ova of *Toxocara spp* +	36
181	F	RB	*Anaplasma marginale* +	Ova of *Fasciola spp* + Ova of *Bunostomum spp* +	37
182	F	RB	-VE	Ova *of Haemonchus spp* + + Ova of *Taenia* spp ++ Ova of *Eimeria* spp +	29
183	F	RB	*Babesia bovis* +	-VE	32
184	M	WF	*Trypanosoma spp* +	-VE	29
185	M	WF	-VE	Ova of *Dicrocoelium dendriticum* +	40
186	F	WF	*Babesia bigemina* +	Ova of *Fasciola spp* ++ Ova of *Strongyle-type egg* +	38
187	M	WF	-VE	-VE	40
188	F	WF	-VE	Ova of *Cooperia* spp ++	39
189	M	WF	-VE	*Ova of Strongyloides spp* ++ Ova of *Eimeria* spp +	38
190	F	WF	*Anaplasma marginale* + *Trypanosoma spp* ++	Ova of *Fasciola spp* ++ Ova of *Strongyloides spp* + Trophozoites of *Ballantidium coli* +	23
191	F	WF	-VE	Ova of *Strongyle-type egg* + Ova of *Bunostomum spp* +	36
192	M	WF	*Anaplasma marginale* ++	Ova of *Fasciola spp* profused Ova of *Strongyle egg-type*+ Ova of *Oesophagostomum spp* +	28

193	F	WF	-VE	-Ve	48
194	F	WF	*Babesia bovis* ++ *Anaplasma marginale* +	Ova of *Strongyle-type egg* +++ Ova of *Eimeria* spp +	30
195	M	WF	-VE	Ova of *Strongyle-type egg* ++ Ova of *Oesophagostomum spp* +	40
196	F	WF	-VE	-VE	40
197	F	WF	*Anaplasma marginale* +	Oocyst of *Eimeria spp* + Ova of *Fasciola spp* ++	39
198	F	WF	-VE	Ova of Strongyle-type egg Ova of *Oesophagostomum spp* +	39
199	M	WF	*Anaplasma marginale* +	-VE	33
200	F	WF	*Babesia bovis* +	Ova of *Bunostomum spp* + Ova of *Paramphistomum spp* ++	35
201	M	WF	-VE	-VE	40
202	M	WF	*Anaplasma marginale* +	*Ova of Strongyloides spp* ++	37
203	M	WF	-VE	Ova of *Paramphistomum spp* +	39
204	M	WF	*Babesia bovis* +	-VE	35
205	F	WF	*Babesia bigemina* +	Oocyst of *Eimeria* spp + Ova of *Strongyle-type egg* +	39
206	M	WF	*Babesia bovis*	-VE	35
207	F	WF	-VE	-VE	40
208	F	WF	-VE	-VE	41
209	F	WF	*Anaplasma marginale* +	*Taenia spp* +	35
210	M	WF	-VE	Ova of *Moniezia benedeni* + Ova of *Bunostomum spp* +	39
211	M	WF	*Babesia bovis* +	-VE	38
212	F	WF	-VE	Ova of *Fasciola spp* + Ova of *Strongyloides spp* +	40
213	F	WF	-VE	-VE	41
214	F	WF	-VE	Ova of *Strongyle egg-type* ++	40
215	F	WF	*Anaplasma marginale* ++	-VE	29
216	F	WF	*Anaplasma marginale* +	Ova of *Bunostomum spp* +	30
217	F	WF	-VE	-VE	40
218	F	WF	*Babesia bigemina* +	Ova of *Fasciola spp* ++ Ova of *Moniezia benedeni* ++	39
219	F	WF		Ova of *Fasciola* spp + Ova of *Strongyloides spp* +	38
220	F	WF	-VE	Ova of *Cooperia spp* + *Ova of Fasciola spp* +	40
221	F	BK	*Babesia bigemina* +	-VE	33
222	F	BK	*Babesia bovis* + *Anaplasma marginale* +	-VE	38
223	F	BK	-VE	Ova of *Bunostomum spp* ++ Ova of *Moniezia benedeni* +	39

				Ova of *Fasciola spp* ++	
224	M	BK	-VE	-VE	40
225	F	BK	*Anaplasma marginale* +	Ova of *Paramphistomum spp* ++	31
226	M	BK	-VE	-VE	40
227	F	BK	*Babesia bovis* ++	Ova of *Haemonchus spp*+ Ova of *Strongyloides spp* ++	36
228	F	BK	-VE	Ova of *Toxocara spp* + Ova of *Fasciola spp* +	40
229	F	BK	-VE	-VE	39.5
230	M	BK	*Anaplasma marginale* ++	Ova of *Paramphistomum spp* + Ova of *Bunostomum spp* +	38
231	F	BK	-VE	-VE	40
232	F	BK	*Babesia bovis* + *Anaplasma marginale* +	Oocyst of *Eimeria* spp profused Ova of *Haemonchus spp* +	23
233	F	BK	-VE	-VE	39.5
234	F	WF	-VE	-VE	46
235	F	WF	-VE	-VE	39.5
236	M	WF	-VE	*Taenia spp* + Ova of *Moniezia benedenni* +	39
237	M	WF	*Anaplasma marginale* ++	Ova of *Moniezia benedeni* +	35
238	M	BK	*Anaplasma marginale* +	Ova of *Oesophagostomum spp* ++	35
239	F	BK	*Babesia bigemina* ++	Ova of *Fasciola spp* + Ova of *Oesophagostomum spp* +	36
240	M	WF	-VE	-VE	40.5
241	F	WF	*Anaplasma marginale* +	Ova of *Strongyle egg-type* ++ Ova of *Bunostomum spp* +	34
242	F	WF	-VE	-VE	39
243	F	WF	*Babesia bovis* +	-VE	34
244	F	WF	*Trypanosoma* spp + *Anaplasma marginale* +	Ova of *Bunostomum spp* +	24
245	M	BK	-VE	Ova of *Fasciola spp* + Oocyst of *Eimeria spp* +	40
246	M	BK	-VE	Ova of *Fasciola spp* +++	39
247	M	BK	-VE	Ova of *Cooperia spp* + *Ova of Fasciola spp* +	38
248	F	BK	-VE	-VE	40.5
249	M	BK	-VE	Ova of *Fasciola spp* + Ova of *Strongyle-type egg* +	39.5
250	M	BK	-VE	-VE	40
251	F	WF	*Babesia bigemina* +	Ova of *Fasciola spp* + Ova of *Oesophagostomum spp* +	34
252	F	WF	-VE	-VE	39
253	M	WF	-VE	Ova of *Strongyle egg-type*+	39.5
254	F	WF	-VE	Ova of *Oesophagostomum spp* +	38.5

				Ova of *Fasciola spp* ++	
255	M	WF	-VE	-VE	40
256	M	WF	*Anaplasma marginale* +	-VE	40
257	F	WF	-VE	Ova of *Fasciola spp* ++ Ova of *Strongyle-type egg* ++	39
258	F	WF	*Babesia bovis* +	-VE	39.5
259	F	WF	-VE	Ova of *Fasciola spp* + Ova of *Strongyloides spp* ++	39
260	F	WF	-VE	Ova of *Toxocara spp* +	40
261	M	WF	-VE	-VE	41
262	F	WF	*Anaplasma marginale* ++	-VE	38
263	F	WF	-VE	-VE	40
264	F	WF	-VE	Ova of *Toxocara spp* +	39
265	F	WF	*Anaplasma marginale* +	Ova of *Taenia spp* + Ova of *Oesophagustomum spp* + Ova of *Toxocara spp* +	35
266	F	WF	-VE	Ova of *Strongyle egg-type*+	38
267	F	WF	*Babesia bovis* +	-VE	34
268	F	WF	-VE	Ova of *Fasciola spp* + Ova of *Oesophagostomum spp* +	39.5
269	F	RB	-VE	-VE	39
270	F	RB	*Anaplasma marginale* +	Ova of *Strongyle egg-type* ++	29
271	F	RB	-VE	-VE	38
272	M	RB	-VE	-VE	39
273	F	RB	-VE	-VE	39.5
274	M	WF	*Babesia bovis* +	Ova of *Fasciola* spp + Ova of *Oesophagostomum spp* +	30
275	F	WF	*Trypanosoma* spp +	Ova of *Fasciola* spp ++ + Ova of *Strongyle-type egg* +	24
276	M	WF	-VE	Ova of *Strongyloides spp* + *Ova of Fasciola spp* +	40.1
277	F	WF	-VE	-VE	39.5
278	F	RB	-VE	Ova of *Cooperia spp* + Ova of *Fasciola spp*++	40
279	F	RB	*Anaplasma marginale* ++	Ova of *Strongyle-type egg* ++	35
280	M	RB	-VE	-VE	38
281	F	RB	*Babesia bigemina* + *Anaplasma marginale* +	-VE	29
282	M	RB	*Trypanosoma* spp ++ *Anaplasma marginale* +	Ova of *Dicrocoelium dendriticum* ++	34
283	F	RB	-VE	Ova of *Fasciola spp*+ Ova of *Oesophagostomum spp* +	38
284	F	RB	-VE	-VE	39

285	F	WF	-VE	-VE	46
286	F	WF	-VE	Ova of *Taenia* spp Ova of *Fasciola spp* + Ova of *Oesophagostomum spp* +	40.5
287	F	WF	-VE	Ova of *Fasciola spp*+++	39.5
288	M	WF	*Anaplasma marginale* +	Ova of *Oesophagostomum spp* + Ova of *Fasciola spp* +++ Ova of *Taenia* spp +	24
289	F	WF	-VE	-VE	40
290	M	WF	-VE	-VE	39.5
291	F	WF	*Babesia bigemina* + *Anaplasma marginale* ++	Ova of *Fasciola spp*+ Ova of *Strongyloides spp* ++	20
292	F	RB	*Trypanosoma* spp + *Anaplasma marginale* +	-VE	24
293	F	RB	-VE	Ova of *Fasciola spp*+ Ova of *Taenia* spp +	36
294	F	RB	-VE	Ova of *Toxocara spp* + + Oocyst of *Eimeria spp* + Ova of *Fasciola spp* ++	37
295	F	RB	-VE	-VE	39
296	M	RB	-VE	Ova of *Strongyle egg-type*++	40
297	F	RB	*Anaplasma marginale* +	Ova of *Oesophagostomum spp* + + Ova of *Strongyle-type egg* profused	40
298	M	RB	-VE	-VE	39
299	F	WF	-VE	-VE	41
300	M	WF	*Babesia bigemina* + *Anaplasma marginale* ++	Oocyst of *Eimeria spp* +++ Ova of *Fasciola spp* ++ Ova of *Strongyloides spp* ++	38

Appendix XV: Haemo and endo parasites of wildlife in Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria

S/N	SPECIE	SEX	HAEMOPARASITE	FAECAL PARASITE	PCV
01	Zebra	M	*Babesia equi* +	Ova of *Trichomena* spp + Ova of *Triodontophorus* spp+	32
02	Zebra	F	-Ve	Ova of *Strongyloides* spp + *Ova of Fasciola* spp++	46
03	Zebra	M	*Babesia equi* ++	Ova of *Gastrodiscus* spp+	32
04	Zebra	F	*Babesia equi* +	Ova of *Triodontophorus* spp++	40
05	Zebra	M	-Ve	-Ve	40
06	Zebra	M	-Ve	Ova of *Trichomena* spp + Ova of *Gastrodiscus* spp+ Ova of *Strongylus* spp ++	36
07	Zebra	M	-Ve	-Ve	26
08	Zebra	M	-Ve	Ova of *Strongylus* spp ++	42
09	Zebra	F	*Babesia cabali* ++	-Ve	32
10	Zebra	M	*Babesia equi* ++	Ova of T*richomena* spp ++ Ova of *Triodontophorus* spp+	40
11	Zebra	M	-Ve	-Ve	33
12	Zebra	M	-Ve	*Ova of Anoplocephala* spp ++ Ova of *Strongylus* spp+	37
13	Zebra	M	*Babesia equi* ++	Ova of *Gastrodiscus* spp+	34
14	Zebra	F	-Ve	Ova *of Fasciola* spp++ Ova of *Strongyloides* spp	36
15	Zebra	F	-Ve	-Ve	36
16	Zebra	M	-Ve	-Ve	32
17	Zebra	F	*Babesia cabali* +	Ova of *Strongylus* spp + *Ova of Fasciola* spp+	32
18	Zebra	M	-Ve	Ova of *Triodontophorus* spp+ *Ova of Fasciola* spp++	38
19	Zebra	M	-Ve	-Ve	39
20	Zebra	F	*Babesia cabali*++	Ova of *Gastrodiscus* spp + *Ova of Fasciola* spp++	32
21	Zebra	M	-Ve	Ova of *Strongylus* spp +++ Ova of *Gastrodiscus* spp+	39
22	Zebra	M	*Babesia equi*+	-Ve	46
23	Zebra	M	*Babesia cabali* ++	-Ve	40
24	Zebra	M	*Babesia equi* ++	*Ova of Anoplocephala* spp+ Ova of *Strongylus* spp	44
25	Zebra	F	*Trypanasoma spp*+ *Babesia equi*+	-Ve	46
26	Zebra	M	-Ve	-Ve	37
27	Zebra	F	-Ve	-Ve	36
28	Zebra	M	-Ve	Ova of *Strongylus* spp+	40
29	Zebra	M	*Babesia equi*+	Ova of *Strongylus* spp++	45
30	Zebra	M	-Ve	-Ve	36
31	Zebra	M	*Babesia cabali*+	-Ve	40
32	Zebra	F	*Babesia equi*+	Ova of *Trichomena spp* +	36
33	Zebra	M	-Ve	-Ve	41
34	Zebra	F	-Ve	-Ve	45

35	Zebra	F	-Ve	-Ve	39
36	Zebra	M	-Ve	Ova of *Trichomena spp*+ Ova of *Strongylus* spp+	45
37	Zebra	M	*Babesia equi* ++	-Ve	38
38	Zebra	F	*Babesia equi*++	Ova of *Fasciola* spp+ Ova of *Strongyloides* spp+	40
39	Zebra	F	-Ve	*Ova of Anoplocephala* spp+ Ova of *Strongylus* spp ++	36
40	Zebra	M	*Babesia equi* +	Ova of *Fasciola* spp+ Ova of *Strongyloides* spp+	31
41	Zebra	F	*Babesia cabali*++	Ova of *Strongylus* spp+	34
42	Zebra	M	*Babesia cabali*++	-Ve	40
43	Zebra	F	-Ve	Ova of *Trichomena* spp ++ Ova of *Triodontophorus* spp++	38
44	Zebra	M	*Babesia equi* +	Ova of *Strongyloides* spp++	36
45	Zebra	M	-Ve	-Ve	36
46	Zebra	F	-Ve	-Ve	59
47	Zebra	M	-Ve	Ova of *Fasciola* spp + Ova of *Triodontophorus* spp +	60
48	Zebra	F	-Ve	-Ve	37
49	Zebra	M	*Babesia cabali*++	Ova of *Strongylus* spp ++	32
50	Zebra	M	-Ve	-Ve	30
51	Zebra	F	-Ve	*Ova of Anoplocephala* spp++	30
52	Zebra	M	*Babesia cabali*++	-Ve	30
53	Zebra	F	*Babesia equi* ++	Ova of *Fasciola* spp++ Ova of *Strongylus* spp +	30
54	Waterbuck	F	*Babesia* spp + *Anaplasma marginale*++	Ova of *Fasciola* spp + *Ova of Taenia* spp++ Ova of *Ancylostoma* spp	30
55	Waterbuck	M	-Ve	-VE	34
56	Waterbuck	M	-Ve	Ova of *Schistosoma* spp++ *Ova of Taenia* spp+	28
57	Waterbuck	M	-Ve	Ova of *Paramphistomum* spp+ Ova of *Schistosoma* spp+ Ova of *Fasciola* spp +	32
58	Waterbuck	F	*Anaplasma marginale*+	Ova of *Paramphistomum* spp++ Ova of *Strongyle* type-egg+	33
59	Waterbuck	F	*Babesia* spp++ *Anaplasma marginale*+	Ova of *Ancylostoma* spp++ Ova of *Dictyocaulus* spp+	28
60	Waterbuck	M	-Ve	-V	30
61	Waterbuck	M	-Ve	Ova of *Fasciola* spp + Ova of *Cooperia* spp +	34
62	Waterbuck	F	*Trypanosoma spp*+ *Anaplasma marginale*+	-Ve	34
63	Waterbuck	F	-Ve	-Ve	31
64	Waterbuck	M	*Anaplasma marginale*+	Trophozoites of *Ballantidium* spp+ Ova of *Cooperia spp*+ Ova of *Fasciola* spp ++	29
65	Wildebeest	M	-Ve	Ova of *Fasciola spp* ++	21

66	Wildebeest	F	*Babesia* spp ++ *Anaplasma marginale*++	Ova of *Strongyle* type-egg+ Ova of *Paramphistomum* spp+ Ova of *Fasciola* spp ++	45
67	Wildebeest	M	*Ehrlichia ruminantium* + *Babesia* spp++ *Anaplasma marginale*+	Ova of *Haemonchus* spp + Ova *of Fasciola* spp+	36
68	Wildebeest	F	-Ve	Ova of *Strongyle* type-egg +	30
69	Wildebeest	M	-Ve	Ova of *Strongyle* type-egg + Ova of *Fasciola* spp ++	30
70	Wildebeest	M	*Anaplasma marginale* ++ *Babesia* spp +	-VE	32
71	Wildebeest	M	*Ehrlichia ruminantium*+ *Anaplasma marginale*++	-VE	30
72	Wildebeest	F	-Ve	-Ve	29
73	Wildebeest	M	-Ve	Ova of *Haemonchus* spp++ Ova of *Strongyle* type-egg + Ova of *Fasciola* spp ++	38
74	Wildebeest	M	*Ehrlichia ruminantium*+ *Babesia* spp++ *Anaplasma marginale* ++	Ova of *Strongyle* type-egg ++ Ova of *Fasciola* spp ++	26
75	Wildebeest	F	*Anaplasma marginale*++ *Trypanosoma spp*+ *Ehrlichia ruminantium*+	-Ve	32
76	Wildebeest	M	*Babesia* spp +++ *Ehrlichia ruminantium*++ *Anaplasma marginale*++	*Ova of Strongyle* type-egg + Ova of *Fasciola* spp	29
77	Eland	M	*Ehrlichia ruminantium*++ *Anaplasma marginale*+	-Ve	24
78	Eland	F	*Babesia bovis*++ *Anaplasma marginale*+	Ova of *Fasciola* spp+ Ova of *Strongyle* type-egg ++	30
79	Eland	F	*Anaplasma marginale*+ *Babesia bigemina*++	Ova of *Trichomena* spp + Oocyst of *Eimeria* spp+ Ova of *Strongyle* type-egg ++	30
80	Eland	M	*Babesia bigemina*++ *Anaplasma marginale*+	Ova of *Trichuris* spp+ Ova of *Moniezie benedeni*+ Ova of *Fasciola* spp +	29
81	Eland	M	*Babesia bigemina*++	-Ve	29
82	Eland	F	*Ehrlichia ruminantium*++ *Anaplasma marginale*+	*Oesophagostomum* spp ++ Ova of *Strongyloides* spp ++ Ova of *Haemonchus* spp ++	40
83	Eland	M	-Ve	-Ve	26
84	Eland	M	*Babesia bovis*+ *Anaplasma marginale*+ *Trypanosoma spp* +	-Ve	33
85	Eland	F	*Ehrlichia ruminantium*++ *Babesia bigemina*++	Ova of *Paramphistomum* spp+ Ova of *Moniezie* spp + Ova of *Fasciola* spp +	33
86	Eland	F	-Ve	-Ve	19
87	Eland	M	-Ve	Ova of *Fasciola* spp+ *Ova of Bunostomum* spp ++ Ova of *Strongyle* type-egg +	33

88	Eland	M	*Anaplasma marginale*+ *Babesia bigemina*++	-Ve	28
89	Eland	F	*Babesia bigemina*+ *Anaplasma marginale*+	Ova of *Fasciola* spp ++ Ova of *Paramphistomum* spp+ Ova of *Strongyle* type-egg +	42
90	Eland	F	*Babesia bovis*+ *Anaplasma marginale*+ *Trypanosoma spp* ++	Ova of *Fasciola* spp + Ova of *Strongyloides* spp +	20
91	Eland	F	*Babesia bigemina*+ *Trypanosoma* spp++	Ova of *Paramphistomum* spp + Ova of *Fasciola* spp ++	24
92	Eland	M	*Ehrlichia ruminantium*+ *Anaplasma marginale*++	Ova of *Paramphistomum* spp+ *Ova of Oesophagostomum* spp+	30
93	Eland	M	-Ve	-Ve	25
94	Eland	M	*Ehrlichia ruminantium*++ *Babesia bigemina*+ *Anaplasma marginale*+	Ova of *Fasciola* spp + Oocyst of *Eimeria* spp Profused Ova of *Strongyloides* spp+	28
95	Eland	M	*Babesia bovis* + *Anaplasma marginale*+	-Ve	33
96	Eland	M	*Ehrlichia ruminantium*+ *Anaplasma marginale*+	Oocyst of *Eimeria* spp +++ Ova of *Haemonchus* spp+ *Ova of Bunostomum* spp++	21
97	Eland	M	*Trypanosoma spp* + *Anaplasma marginale*+	-Ve	24
98	Eland	F	*Ehrlichia ruminantium*++ *Babesia bigemina*++	Ova of *Haemonchus* spp+ Ova of *Strongyle* type-egg ++ Ova of *Fasciola* spp ++	30
99	Eland	F	*Babesia bovis*++ *Anaplasma marginale*+	-Ve	22
100	Eland	M	-Ve	Ova of *Strongyle* type-egg + Ova of *Fasciola* spp +	49
101	Elephant	M	*Anaplasma* spp++	Ova of *Fasciola* spp ++	30
102	Elephant	M	-Ve	Ova of *Strongyle* type-egg ++	30
103	Elephant	M	-Ve	-Ve	31
104	Elephant	F	*Trypanosoma spp*+	Ova of *Fasciola* spp+ Ova of *Strongyloides* spp+	31
105	Heartbeest	M	*Anaplasma spp*++ *Ehrlichia ruminantium*+	Ova of *Strongyle* type-egg +	33
106	Kudu	M	*Babesia spp*+	Ova of *Fasciola spp*++	30

Appendix XVI :Haemo and endo parasites of donkeys around Yankari game reserve and Sumu wildlife Park in Bauchi State, Nigeria

S/No	Sex	Faecal Parasites	Blood parasites	PCV
01	Male	Ova of *Gastrodiscus spp++*	-VE	38
02	Male	*Ova of Trichomena spp+*	-VE	39
03	Male	*-ve*	*Babesia caballi +*	37
04	Male	*-ve*	-VE	39
05	Male	*Ova of Anoplocephala spp+*	*Babesia caballi +*	27
06	Female	*-ve*	-VE	41
07	Male	*Ova of Trichomena spp +* Ova of *Fasciola* spp +	-VE	39
08	Male	*-ve*	-VE	38
09	Male	*-ve*	-VE	38
10	Male	*Ova of Strongylusspp++*	*Babesia caballi +* *Babesia caballi +*	24
11	Male	Ova of *Fasciola* spp ++	-VE	39
12	Male	*Ova of Trichomena spp++*	-VE	38
13	Male	*Ova of Anoplocephala spp+*	-VE	39
14	Male	*-ve*	*Babesia equi +*	40
15	Male	*Strongylus spp +* *Ova of Anoplocephala spp+*	-VE	27
16	Male	*-ve*	-VE	39
17	Male	*-ve*	*Trypanosoma* spp ++	27
18	Male	*Strongylusspp +*	-VE	39
19	Male	*-ve*	-VE	38
20	Male	*-ve*	*Babesia caballi +*	36
21	Male	Ova of *Gastrodiscus spp+*	*Babesia equi +*	32
22	Male	*-ve*	-VE	38
23	Male	*Ova of Trichomena spp++* Ova of *Fasciola* spp +	-VE	39
24	Male	*-ve*	*Trypanosoma* spp +	23
25	Female	-ve	-VE	38
26	Male	-ve	-VE	41

27	Male	Ova of *Fasciola* spp +	-VE	36
28	Male	-ve	*Babesia caballi* +	30
29	Male	*-ve*	-VE	39
30	Male	Ova of *Fasciola* spp ++	-VE	32
31	Male	*Ova of Trichomena spp* +	*Babesia equi*+	26
32	Male	*-ve*	-VE	38
33	Male	*Strongylus spp*++	-VE	44
34	Male	*-ve*	*Babesia caballi* +	32
35	Male	*Ova of Trichomena spp*++ Ova of *Fasciola* spp +	*Babesia caballi* + *Babesia caballi* +	30
36	Male	*-ve*	-VE	38
37	Female	*-ve*	-VE	40
38	Female	*Ova of Strongylusspp*++	*Babesia caballi* + *Babesia caballi* +	24
39	Male	*-ve*	-VE	39
40	Male	*Ova of Trichomena spp* + *Ova of Anoplocephala spp*+	*Babesia caballi* + *Babesia caballi* +	25
41	Male	*-ve*	-VE	39
42	Male	Ova of *Gastrodiscus spp*+	*Babesia caballi* + *Trypanosoma spp*++	22
43	Male	*-ve*	-VE	41
44	Male	-ve	-VE	40
45	Male	*Ova of Strongylusspp*++	-VE	36
46	Male	*Strongylus spp*+	-VE	38
47	Female	Ova of *Stelizia species* + Ova of *Gastrodiscus spp*++	*Trypanosoma spp*	30
48	Female	*Strongylus spp*+	-VE	36
49	Female	*Ova of Anoplocephala spp*+	-VE	37
50	Female	*-ve*	-VE	41
51	Male	*-ve*	-VE	38
52	Male	*Ova of Trichomena spp* + Ova of *Fasciola* spp ++	*Babesia caballi* + *Babesia caballi* +	24
53	Male	*Ova of Trichomena spp*+	-VE	39

54	Male	*-ve*	-VE	38
55	Male	Ova of *Fasciola* spp +	*Babesia equi*+	39
56	Male	*-ve*	-VE	38
57	Male	Ova of *Fasciola* spp ++	*Trypanosoma spp* +	36
58	Male	*-ve*	-VE	39
59	Male	Ova of *Stelizia species* + Ova of *Gastrodiscus spp*+	*Babesia caballi* + *Babesia caballi* +	22
60	Male	*Ova of Strongylus spp*++	-VE	36
61	Male	Ova of *Stelizia species* +	*Babesia equi* +	38
62	Male	*-ve*	-VE	42
63	Male	*Ova of Strongylus spp*++	-VE	46
64	Male	-ve	-VE	38
65	Male	Ova of *Gastrodiscus spp*+	-VE	37
66	Male	*-ve*	-VE	39
67	Male	*Ova of Trichomena spp* ++ *Ova of Anoplocephala spp*+	-VE	37
68	Male	*Ova of Trichomena spp*+	*Babesia caballi* +	30
69	Male	*-ve*	-VE	36
70	Male	Ova *of Strongylusspp*+ *Ova of Trichomena spp*+ Ova of *Fasciola* spp +	-VE	39
71	Male	*-ve*	-VE	41
72	Male	*-ve*	*Trypanosoma* spp ++	30
73	Male	*-ve*	*Babesia caballi* ++	41
74	Male	*-ve*	-VE	29
75	Male	Ova *of Trichomena spp* + Ova of *Fasciola* spp +	*Babesia equi*+	29
76	Male	*-ve*	-VE	39
77	Female	*Ova of Strongylus spp* ++ Ova of *Fasciola* spp +	*Babesia caballi* ++	28
78	Male	*-ve*	-VE	41
79	Male	*-ve*	-VE	39
80	Male	*-ve*	-VE	38

81	Male	Ova of *Strongylusspp* ++ Ova of *Gastrodiscus spp*+ Ova of *Fasciola* spp ++	*Babesia equi* + *Babesia caballi* +	25
82	Female	*-ve*	*Babesia caballi* +	39
83	Male	*-ve*	-VE	41
84	Male	*Ova of Anoplocephala spp*+ Ova of *Fasciola* spp +	-VE	36
85	Female	*-ve*	-VE	39
86	Male	*-ve*	-VE	42
87	Male	Ova of *Strongylusspp* ++ Ova of *Fasciola* spp +	-VE	36
88	Male	*-ve*	*Babesia caballi* + *Babesia caballi* +	34
89	Male	*-ve*	-VE	39
90	Male	Ova of *Stelizia species* + Ova of *Gastrodiscus spp*++	-VE	30
91	Male	*Ova of Trichomena spp*++	-VE	37
92	Male	*-Ve*	-VE	39
93	Male	*Ova of Trichomena spp* + Ova of *Fasciola* spp +	-VE	30
94	Female	*-ve*	-VE	41
95	Male	*Ova of Strongylus spp*+ Ova of *Fasciola* spp +	-VE	30
96	Female	*-ve*	*Babesia equi* ++	37
97	Female	*-ve*	*Babesia caballi* + *Babesia caballi* +	24
98	Male	*Ova of Trichomena spp*+ *Strongylus spp*+	-VE	32
99	Female	*-ve*	*Trypanosoma spp*	30
100	Female	*Ova of Strongylus spp*+ Ova of *Fasciola* spp +	*Babesia caballi* + *Babesia equi* +	28
101	Male	*Strongylus spp* + *Strongylusspp*++	-VE	30

102	Male	*Ova of Strongylus spp+*	-VE	39
103	Female	*Ova of Trichomena spp++* *Ova of Stelizia species+*	*Babesia caballi +*	30
104	Male	*Strongylus spp+* *Strongylusspp++*	*Babesia equi +* *Babesia equi +*	26
105	Female	*-ve*	-VE	41
106	Female	*Ova of Trichomena spp+*	-VE	39
107	Male	Ova of *Strongylus spp++*	-VE	38
108	Female	Ova of *Strongylusspp+*	-VE	39
109	Female	Ova of *Triodontophorus spp+*	-VE	41
110	Female	Ova of *Gastrodiscus spp+*	-VE	38
111	Male	*Strongylus spp++*	-VE	41
112	Male	*Triodontophorus spp+*	*Babesia equi +*	39
113	Male	*Strongylusspp+*	-VE	36
114	Male	*-ve*	-VE	39
115	Female	*Ova of Trichomena spp+* Ova of *Fasciola* spp +	*Babesia equi+*	30
116	Male	*Strongylusspp+*	-VE	38
117	Male	Ova of *Anaplocephala spp++*	-VE	37
118	Female	*Ova of Trichomena spp +* Ova of *Fasciola* spp +	*Babesia equi +* *Babesia equi +*	28
119	Male	Ova of *Strongylus spp +* Ova of *Fasciola* spp +	*Babesia equi+*	30
120	Male	*-ve*	-VE	36
121	Female	*Ova of Trichomena spp +* Ova of *Fasciola* spp +	*Babesia equi +*	30
122	Female	*-ve*	-VE	39
123	Male	Ova of *Strongyloides spp+*	*Trypanosoma* spp ++	29
124	Female	Ova of *Strongylus spp ++* Ova of *Anoplocephala spp++*	-VE	32
125	Female	Ova of *Triodontophorus spp+*	-VE	39
126	Female	*-ve*	-VE	38
127	Male	Ova of *Strongyloides spp+*	-VE	39

128	Male	-ve	*Babesia caballi* ++ *Trypanosoma spp* ++	23
129	Female	*Strongylus spp*++	-VE	38
130	Female	-ve	-VE	39
131	Female	*-ve*	-VE	38
132	Male	Ova of *Strongylus spp*++	*Trypanosoma spp* ++	29
133	Female	Ova of *Strongyloides spp*++	*Babesia caballi* +	30
134	Female	*Ova of Trichomena spp*+	-VE	36
135	Female	*-ve*	*Trypanosoma spp* ++	38
136	Male	Ova of *Strongylus spp*+	-VE	39
137	Male	Ova of *Strongyloides spp*+	-VE	38
138	Male	Ova of *Strongyloides spp* + Ova of *Strongylus spp*+	-VE	37
139	Male	Ova of *Strongyloides spp*++	*Babesia caballi* +	33
140	Female	*-ve*	-VE	41
141	Female	Ova of *Strongyloides spp*+	-VE	36
142	Male	Ova of *Strongyloides spp*+	-VE	38
143	Male	Ova of *Strongyloides spp*+	-VE	39
144	Male	Ova of *Triodontophorus spp*+	*Trypanosoma spp* ++	36
145	Male	Ova of *Strongylus spp*++	-VE	37
146	Male	Ova of *Strongyloides spp*+ Ova of *Triodontophorus spp*+	-VE	30
147	Male	Ova of *Strongylus spp*+	*Babesia caballi* + *Babesia equi* +	24
148	Male	Ova of *Strongyloides spp*+ Ova of *Strongylus spp*+	-VE	30
149	Male	Ova of *Strongyloides spp*++	*Babesia caballi* +	38
150	Male	Ova of *Strongyloides spp*+	-VE	39
151	Male	*Ova of Trichomena spp* ++ Ova of *Strongylus spp*+	*Babesia equi* +	41
152	Male	Ova of *Strongyloides spp*++	-VE	39
153	Male	*Ova of Trichomena spp*+	-VE	38
154	Male	*-ve*	-VE	41

155	Male	*Ova of Trichomena spp* + Ova of *Strongyloides spp*++ Ova of *Fasciola* spp +	*Babesia equi* + *Babesia caballi* ++	30
156	Male	Ova of *Triodontophorus spp*+ Ova of *Fasciola* spp +	-VE	32
157	Male	Ova of *Strongyloides spp* + Ova of *Fasciola* spp +	-VE	38
158	Male	-ve	*Babesia equi* + *Anaplasma centrale* +	26
159	Male	-ve	-VE	38
160	Male	Ova of *Strongyloides spp* ++ Ova of *Strongylus spp*++	-VE	39
161	Male	-ve	-VE	41
162	Female	*-ve*	*Babesia equi* +	38
163	Female	Ova of *Strongyloides spp*+	-VE	39
164	Male	Ova of *Strongyloides spp*+	-VE	38
165	Male	*-ve*	*Babesia caballi* +	38
166	Male	Ova of *Strongylus spp*+	-VE	39
167	Male	Ova of *Gastrodiscus spp*++	-VE	38
168	Male	Ova of *Strongyloides spp*+	-VE	39
169	Male	*-ve*	*Trypanosoma* spp ++	30
170	Male	Ova of *Strongyloides spp*++	-VE	38
171	Male	Ova of *Strongylus spp* + Ova of *Fasciola* spp +	-VE	36
172	Male	*Ova of Trichomena spp*++	-VE	36
173	Male	Ova of *Strongyloides spp*++	-VE	38
174	Male	Ova of *Stelizia species* +	*Babesia caballi* ++	36
175	Female	Ova of *Stelizia species* + Ova of *Gastrodiscus spp*++	-VE	30
176	Female	Ova of *Strongyloides spp*+	-VE	37
177	Male	Ova of *Strongylus spp* + Ova of *Fasciola* spp +	-VE	36
178	Male	*-ve*	-VE	41

179	Male	*Ova of Trichomena spp* + Ova of *Strongyloides spp*+	*Babesia caballi* +	39
180	Male	Ova of *Strongyloides spp*+	-VE	38
181	Male	Ova of *Strongyloides spp* Ova of *Triodontophorus spp*+	-VE	39
182	Male	Ova of *Strongyloides spp*+	*Babesia equi*+	39
183	Female	Ova of *Strongylus spp*+	-VE	41
184	Female	Ova of *Strongyloides spp*+	-VE	39
185	Male	*Ova of Trichomena spp*+	*Babesia caballi* +	38
186	Male	Ova of *Strongyloides spp*++	-VE	36
187	Male	Ova of *Strongylus spp* + Ova of *Fasciola* spp +	*Babesia equi* + *Babesia equi* +	24
188	Male	-ve	-VE	37
189	Male	*-ve*	-VE	39
190	Male	-ve	-VE	40
191	Female	Ova of *Trichomena spp* + Ova of *Fasciola* spp +	*Babesia equi* + *Babesia equi* +	27
192	Female	-ve	-VE	39
193	Male	Ova of Strongyloides spp +	-VE	38
194	Male	-ve	-VE	39
195	Male	Ova of *Trichomena* spp + Ova of *Fasciola* spp +	*Babesia caballi* ++	27
196	Male	-ve	-VE	39
197	Male	-ve	*Babesia caballi* + *Babesia equi* +	28
198	Female	Ova of *Strongylus* spp ++	-VE	38
199	Female	Ova of *Strongyloides spp* + Ova of *Triodontophorus spp* +	*Babesia equi* +	37
200	Male	Ova of *Strongyloides spp* ++	-VE	39

Appendix XVII Haemo and endo parasites of horses around the Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria

S/N	Age	Sex	Breed	Gastrointestinal Parasites	Haemoparasites	PCV
1	Adult	Male	Local	-VE	-VE	39
2	Adult	Male	Local	Ova *of Strongyloides spp+*	*Babesia caballi +*	30
3	Adult	Male	Local	-VE	-VE	40
4	Adult	Male	Local	-VE	-VE	38
5	Adult	Male	Local	-VE	*Babesia equi +*	27
6	Adult	Male	Local	Ova of *Triodontophorus spp+* *Ova of Strongylus spp+*	-VE	36
7	Adult	Female	Local	Ova *of Strongylus spp++*	-VE	38
8	Adult	Male	Local	-VE	-VE	39
9	Adult	Male	Local	-VE	-VE	38
10	Adult	Male	Local	-VE	-VE	38
11	Adult	Male	Local	-VE	-VE	37
12	Adult	Male	Local	-VE	-VE	40
13	Young	Male	Crossed	Ova of *Strongyloides spp +* Ova of *Gastrodiscus spp++*	*Babesia caballi +*	34
14	Adult	Male	Crossed	*Ova of Anoplocephala spp+*	-VE	35
15	Adult	Male	Local	-VE	*Babesia equi +*	32
16	Adult	Male	Local	-VE	-VE	39
17	Adult	Male	Local	-VE	-VE	40
18	Adult	Male	Local	-VE	*Babesia caballi ++*	33
19	Adult	Male	Agentine	Ova of *Strongyloides spp++*	-VE	39
20	Adult	Male	Local	-VE	-VE	40
21	Adult	Male	Local	-VE	-VE	39
22	Adult	Male	Local	-VE	*Babesia caballi +*	36
23	Adult	Male	Local	-VE	-VE	39
24	Adult	Male	Local	-VE	-VE	39
25	Adult	Male	Crossed	-VE	-VE	40
26	Adult	Male	Crossed	Ova of *Fasciola* spp+	*Babesia equi +*	25
27	Young	Male	Local	Ova of *Triodontophorus spp++* *Ova of Strongylus spp++*	-VE	33
28	Young	Male	Local	-VE	-VE	37
29	Young	Male	Local	-VE	-VE	38
30	Young	Male	Local	Ova of *Fasciola* spp+	-VE	30
31	Young	Male	Local	-VE	-VE	38
32	Young	Male	Local	-VE	-VE	39
33	Adult	Male	Local	-VE	-VE	39
34	Adult	Male	Local	Ova *of Strongylus spp++*	-VE	
35	Young	Male	Local	-VE	*Babesia caballi +*	34
36	Adult	Male	Local	-VE	-VE	39
37	Adult	Male	Local	-VE	-VE	40
38	Adult	Male	Local	-VE	-VE	40

39	Adult	Male	Crossed	-VE	*Babesia caballi* ++	36
40	Adult	Male	Local	Ova of *Strongyloides spp*+ Ova of *Gastrodiscus spp*+	-VE	38
41	Adult	Male	Local	-VE	-VE	41
42	Adult	Male	Local	-VE	-VE	39
43	Adult	Male	Local	-VE	-VE	40
44	Adult	Male	Local	Ova *of Strongylus spp*+	-VE	29
45	Adult	Male	Local	-VE	*Babesia caballi* +	
46	Adult	Male	Local	-VE	-VE	45
47	Adult	Male	Local	-VE	-VE	38
48	Adult	Male	Local	-VE	-VE	41
49	Adult	Male	Local	-VE	*Babesia equi* +	
50	Adult	Male	Local	-VE	-VE	38
51	Adult	Female	Local	Ova *of Strongylus spp*+++	-VE	36
52	Adult	Female	Local	-VE	-VE	38
53	Adult	Female	Local	-VE	-VE	40
54	Adult	Female	Local	-VE	-VE	39
55	Adult	Male	Local	-VE	-VE	41
56	Adult	Male	Local	-VE	-VE	39
57	Adult	Male	Local	-VE	*Babesia caballi* +	30
58	Adult	Male	Local	*Ova of Strongylus spp*+	-VE	32
59	Adult	Male	Local	*Ova of Strongylus spp*+	*Babesia caballi* + *Babesia equi*	24
60	Adult	Male	Local	-VE	-VE	36
61	Adult	Male	Local	-VE	-VE	37
62	Adult	Male	Local	-VE	-VE	41
63	Adult	Male	Crossed	-VE	-VE	34
64	Adult	Male	Crossed	-VE	-VE	38
65	Adult	Male	Local	-VE	*Babesia caballi* +	29
66	Adult	Male	Local	*Ova of Strongylus spp*+ Ova of *Fasciola* spp+	-VE	30
67	Young	Male	Local	-VE	-VE	38
68	Adult	Male	Local	-VE	-VE	36
69	Adult	Male	Local	Ova of *Strongylus spp*+	-VE	40
70	Adult	Male	Local	-VE	*Babesia caballi* ++	30
71	Adult	Male	Local	-VE	-VE	36
72	Adult	Male	Local	-VE	*Babesia caballi* + *Babesia equi*+ +	28
73	Adult	Male	Local	-VE	-VE	36
74	Adult	Male	Local	-VE	-VE	41
75	Adult	Male	Local	-VE	*Babesia equi* +	26
76	Adult	Male	Local	-VE	-VE	36
77	Adult	Male	Local	-VE	-VE	40
78	Adult	Male	Local	-VE	-VE	34
79	Young	Male	Local	Ova *of Strongylus spp*+	-VE	34
80	Young	Male	Local	-VE	-VE	35

81	Adult	Male	Local	-VE	*Babesia caballi* ++	29
82	Adult	Male	Local	-VE	-VE	38
83	Adult	Male	Local	Ova of *Triodontophorus spp*++	-VE	30
84	Adult	Male	Local	-VE	-VE	36
85	Young	Male	Local	-VE	-VE	38
86	Young	Male	Local	-VE	*Babesia caballi* +	27
87	Adult	Male	Local	Ova of *Triodontophorus spp*++	-VE	39
88	Adult	Male	Local	-VE	-VE	40
89	Adult	Male	Local	-VE	-VE	38
90	Adult	Male	Local	-VE	-VE	36
91	Adult	Male	Local	-VE	-VE	39
92	Adult	Male	Local	-VE	*Babesia equi* +	25
93	Adult	Male	Local	Ova *of Strongylus spp*+	-VE	38
94	Adult	Male	Local	-VE	-VE	36
95	Adult	Male	Local	-VE	-VE	39
96	Adult	Male	Local	-VE	*Babesia caballi* +	38
97	Adult	Male	Local	*Ova of Anoplocephala spp*+ Ova of *Strongyloides spp*+	-VE	36
98	Adult	Male	Local	-VE	-VE	40
99	Adult	Male	Local	-VE	-VE	38
100	Adult	Male	Local	Ova of *Triodontophorus spp*++ *Ova of Strongylus spp*++	-VE	34

101	Adult	Male	Local	-VE	*Babesia equi* ++	45
102	Adult	Male	Local	-VE	-VE	32
103	Adult	Male	Local	-Ve	*Babesia caballi* ++	40
104	Adult	Male	Local	-VE	-VE	30
105	Adult	Male	Local	*Ova of Anoplocephala spp*+ Ova of *Fasciola* spp+	-VE	38
106	Adult	Male	Local	-VE	-VE	30
107	Adult	Male	Local	-Ve	*Babesia caballi* +	40
108	Adult	Female	Local	-VE	-VE	40
109	Adult	Female	Local	-VE	-VE	38
110	Adult	Female	Local	-VE	-VE	25
111	Adult	Female	Local	*Ova of Anoplocephala spp*+ Ova of *Strongyloides spp*++	*Babesia equi* + *Babesia equi* +	29
112	Adult	Male	Local	*Ova of Strongylus spp*++	-VE	41
113	Adult	Male	Crossed	-VE	-VE	36
114	Adult	Male	Local	-Ve	-VE	37
115	Adult	Male	Local	-VE	-VE	27
116	Adult	Male	Local	-VE	*Babesia caballi* +	40
117	Adult	Male	Local	-Ve	-VE	38
118	Adult	Male	Local	*Ova of Strongylus spp*+	-VE	39
119	Adult	Male	Local	-VE	-VE	40
120	Adult	Male	Local	-VE	*Babesia caballi* +	41
121	Adult	Male	Local	-VE	-VE	39

122	Adult	Male	Local	-VE	-VE	38
123	Adult	Male	Local	-VE	-VE	34
124	Adult	Male	Local	-VE	*Babesia caballi* ++	32
125	Adult	Male	Local	*Ova of Strongylus spp*+	-VE	40
126	Adult	Male	Local	-VE	-VE	39
127	Adult	Male	Local	-Ve	-VE	31
128	Adult	Male	Local	Ova of *Triodontophorus spp*++	*Babesia equi* +	40
129	Adult	Male	Local	-Ve	-VE	31
130	Adult	Male	Local	-VE	*Babesia caballi* +	33
131	Young	Male	Local	*Ova of Strongylus spp*+ Ova of *Triodontophorus spp*++	-VE	40
132	Adult	Male	Local	-VE	-VE	30
133	Adult	Male	Local	Ova of *Fasciola* spp++	-VE	25
134	Adult	Male	Local	-VE	*Babesia equi* + *Babesia equi*+	41
135	Adult	Male	Local	-VE	-VE	36
136	Adult	Male	Local	-Ve	-VE	40
137	Adult	Male	Crossed	*-ve*	-VE	30
138	Adult	Male	Crossed	*Ova of Strongylus spp*+	-VE	29
139	Adult	Male	Local	-VE	*Babesia caballi* ++	31
140	Adult	Male	Local	-Ve	*Babesia caballi* +	41
141	Adult	Male	Local	-VE	-VE	32
142	Adult	Male	Local	*Ova of Strongylus spp*++	*Babesia caballi* +	39
143	Young	Male	Local	-VE	-VE	33
144	Young	Female	Local	-Ve	*Babesia caballi* +	40
145	Adult	Male	Local	-VE	-VE	37
146	Adult	Male	Local	-VE	-VE	29
147	Adult	Male	Local	-VE	*Anaplasma marginalle*	33
148	Adult	Male	Local	Ova of *Triodontophorus spp*+	-VE	34
149	Young	Male	Local	*Ova of Strongylus spp*+	-VE	37
150	Young	Male	Local	-VE	*Babesia caballi* +	41
151	Young	Male	Local	-VE	-VE	38
152	Adult	Male	Local	-VE	-VE	39
153	Adult	Male	Local	-VE	-VE	36
154	Adult	Male	Local	*Ova of Anoplocephala spp*+	-VE	38
155	Adult	Male	Local	Ova of Strongylus spp + Ova of *Triodontophorus spp*++	*Babesia caballi* ++	40
156	Adult	Male	Local	-VE	-VE	38
157	Adult	Male	Local	*Ova of Strongylus spp*+	-VE	40
158	Adult	Male	Local	-VE	-VE	39
159	Adult	Male	Local	-VE	*Babesia equi*	34
160	Adult	Male	Local	-VE	*Babesia caballi* +	36
161	Adult	Female	Local	*Ova of Strongylus spp*+	-VE	38
162	Adult	Female	Local	-VE	-VE	40
163	Adult	Male	Local	-VE	-VE	25

164	Adult	Male	Local	-VE	*Babesia caballi* + *Babesia equi*	39
165	Adult	Male	Local	-VE	-VE	30
166	Adult	Male	Local	*Ova of Strongylus spp*+	*Babesia caballi* ++	40
167	Adult	Male	Local	*Ova of Strongylus spp*+	-VE	38
168	Adult	Male	Local	-VE	-VE	41
169	Adult	Male	Local	-VE	-VE	37
170	Adult	Male	Local	-VE	-VE	40
171	Adult	Male	Local	-VE	-VE	36
172	Adult	Male	Local	*Ova of Strongylus spp*+	-VE	37
173	Adult	Male	Local	-VE	*Babesia equi*	30
174	Adult	Male	Local	Ova of *Strongyloides spp*+ Ova of *Gastrodiscus spp*+	-VE	32
175	Adult	Male	Local	-VE	*Babesia caballi* ++	40
176	Adult	Male	Local	-VE	-VE	38
177	Adult	Male	Local	-Ve	*Babesia caballi* +	39
178	Adult	Male	Local	Ova *of Strongylus spp*+	-VE	38
179	Adult	Male	Local	-VE	-VE	30
180	Adult	Male	Local	Ova of *Gastrodiscus spp*+	*Babesia caballi* +	39
181	Adult	Male	Local	-VE	-VE	38
182	Adult	Male	Local	-Ve	-VE	36
183	Adult	Female	Local	Ova of *Strongyloides spp* +	-VE	39
184	Adult	Female	Local	-VE	-VE	34
185	Adult	Male	Local	Ova *of Strongylus spp*++	*Babesia caballi* ++	37
186	Adult	Male	Local	-VE	-VE	39
187	Adult	Male	Local	-VE	-VE	29
188	Adult	Male	Crossed	Ova of *Strongyloides spp* ++	*Babesia equi* +	36
189	Adult	Male	Local	Ova of *Strongyloides spp* +	-VE	39
190	Adult	Male	Local	-VE	-VE	45
191	Adult	Male	Local	-VE	-VE	40
192	Adult	Male	Local	*Ova of Strongylus spp*+	-VE	31
193	Adult	Male	Local	*Ova of Strongylus spp*+ Ova of *Fasciola* spp+	-VE	
194	Adult	Male	Local	-VE	*Babesia caballi* ++	34
195	Adult	Male	Local	-VE	-VE	35
196	Young	Male	Local	Ova of *Gastrodiscus spp*+	-VE	36
197	Adult	Male	Local	-VE	*Babesia equi* +	34
198	Adult	Male	Local	Ova of *Strongyloides spp*+	-VE	39
199	Adult	Male	Local	-VE	-VE	45
200	Adult	Female	Local	Ova of *Gastrodiscus spp*+	-VE	30

Appendix XVIII: Haematological values in wildlife from Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria

S/N	Animal Spp	Sex	Age	PCV	HB	RBC	WBC	MCV	MCH	MCHC	N	L	M	E	B
1	Elephant	F	Adult	59	19.7	4.7	4.5	125.50	41.92	33.40	42	58	0	0	0
2	Elephant	M	Adult	60	19.0	4.0	7.6	150.00	47.50	31.67	50	30	0	0	0
3	Elephant	M	Juvenile	49	18.0	3.9	4.8	125.64	46.15	36.74	65	28	0	1	0
4	Elephant	M	Juvenile	42	14.0	3.8	7.6	105.26	34.21	32.50	70	30	0	2	0
5	Zebra	M	Adult	27	9.0	3.7	5.6	72.97	24.32	33.30	60	38	0	02	0
6	Zebra	F	Adult	24	8.0	3.4	3.4	70.59	23.53	33.30	43	56	0	01	0
7	Zebra	M	Adult	36	11.9	3.6	3.7	160.00	33.06	33.06	80	20	0	0	0
8	Zebra	M	Adult	39	13.0	4.9	5.5	79.59	26.53	33.33	32	65	0	03	0
9	Zebra	M	Adult	31	10.5	3.9	3.5	80.00	26.92	33.80	40	90	0	0	0
10	Zebra	M	Adult	45	15.0	4.4	5.4	102.27	34.10	33.33	30	70	0	0	0
11	Zebra	M	Adult	41	13.3	4.1	5.6	100.00	32.44	32.44	43	57	0	0	0
12	Zebra	F	Adult	46	15.4	3.6	5.1	128.00	42.78	33.48	70	38	0	0	0
13	Zebra	F	Adult	29	9.8	3.7	3.6	78.38	26.49	33.79	44	57	0	0	0
14	Zebra	M	Adult	32	10.5	4.2	3.4	76.19	25.00	32.81	58	35	0	0	0
15	Zebra	F	Adult	30	10.0	3.5	6.0	85.71	28.57	33.33	76	24	0	0	0
16	Zebra	M	Adult	32	10.5	4.5	3.2	71.11	23.33	32.81	37	28	0	0	0
17	Zebra	F	Adult	29	9.8	3.7	4.0	78.38	26.49	33.79	45	38	0	0	0
18	Zebra	F	Juvenile	24	8.0	2.4	3.4	100.0	33.33	33.33	43	56	0	1	0
19	Zebra	F	Adult	29	9.8	3.7	3.6	78.20	25.00	33.33	76	28	0	0	0
20	Eland	F	Adult	31	10.3	4.5	3.7	68.90	22.89	33.22	39	61	0	0	0
21	Eland	M	Adult	28	9.3	4.4	4.2	63.64	21.10	33.21	47	53	0	0	0
22	Eland	F	Adult	34	11.2	4.6	3.8	73.91	24.35	32.94	38	66	0	2	0
23	Eland	F	Adult	28	9.1	4.4	3.7	63.63	20.68	32.50	41	62	0	0	0
24	Eland	M	Adult	30	9.5	4.5	3.9	66.67	21.11	31.67	44	64	0	0	0
25	Eland	F	Adult	36	11.9	5.1	3.5	70.59	23.33	33.06	45	69	0	0	0
26	Eland	M	Adult	31	9.8	4.6	3.9	67.39	21.30	31.67	49	65	0	0	0
27	Eland	M	Adult	33	10.8	3.9	4.1	84.62	27.69	32.73	40.2	56	0	0	0
28	Eland	F	Adult	31	10.5	4.9	3.8	63.27	21.43	33.87	33.1	43	0	0	0
29	Eland	M	Adult	29	9.4	4.4	4.2	65.90	21.36	32.41	48.4	58	0	0	0

30	Eland	M	Adult	28	9.1	4.5	3.6	62.20	20.22	32.50	39	65	0	0	0
31	Eland	F	Adult	36	11.9	5.1	3.5	70.59	23.33	33.10	45	58	0	0	0
32	Wildebeest	M	Adult	36	11.4	4.6	4.0	78.26	24.78	31.67	42	61	0	0	0
33	Wildebeest	F	Adult	33	10.2	3.8	3.1	86.84	26.84	30.91	28	50	0	0	0
34	Wildebeest	M	Adult	41	14.4	3.6	3.4	113.89	40.00	35.12	39	49	0	0	0
35	Wildebeest	M	Adult	45	15	57	5.1	78.95	26.31	30.90	33	67	0	0	0
36	Wildebeest	F	Adult	34	12	3.4	2.9	100.00	35.29	35.29	32	56	0	0	0
37	Wildebeest	F	Adult	33	10.2	3.5	3.3	94.29	35.00	30.90	29	63	0	0	0
38	Wildebeest	M	Adult	36	11.4	4.6	4.8	78.26	24.78	31.67	42	51	0	0	0
39	Wildebeest	F	Adult	33	10.2	3.8	3.1	86.84	26.84	30.91	28	47	0	0	0
40	Waterbuck	F	Adult	30	9.8	7.4	8.6	40.54	13.24	32.67	35	57	1	0	0
41	Waterbuck	F	Adult	36	12.0	7.6	8.7	42.86	15.79	33.33	30	59	0	0	0
42	Waterbuck	M	Adult	45	14.3	8.4	9.3	53.57	17.02	31.78	32	63	0	3	0
43	Waterbuck	M	Adult	40	13	7.9	9.8	50.63	16.46	32.50	30	56	0	0	0
44	Waterbuck	F	Adult	39	12.7	12	8.0	32.50	10.58	32.56	19	54	1	1	0
45	Waterbuck	M	Adult	36	12	6.2	8.2	58.07	19.36	33.33	25	43	0	0	1
46	Waterbuck	F	Adult	36	12.0	7.6	8.4	42.86	15.79	33.33	30	44	0	0	0
47	Waterbuck	M	Adult	45	14.3	8.4	9.8	53.57	17.02	31.78	32	62	0	3	0

Appendix XIX: Biochemical values in wildlife from Yankari game reserve and Sumu wildlife park in Bauchi State, Nigeria

S/N	Animal Spp	Sex	ALT (U/L)	AST (U/L)	ALP (U/L)	CREAT (µMOL/L)	UREA (µMOL/L)	ALB (g/L)	T/P (g/L)	TBIL (mg/dl)	DIBIL (mg/dl)	POT (MEq/L	SOD (MEq/L)	GLU (Mm/L)	LDH (U/L)
1	Elephant	M	1.00	10.00	1.84	3.20	5.33	37.90	86.61	1.63	1.94	24.27	138.14	0.42	136.26
2	Elephant	F	12.0	47.0	35.88	8.0	1.07	50.00	96.30	4.42	4.83	12.20	125.42	2.91	256.48
3	Elephant	M	10.00	27.00	14.52	12.04	4.00	27.15	106.42	2.46	2.72	29.90	139.83	1.81	116.03
4	Elephant	M	6.00	13.00	9.80	7.10	4.00	31.00	86.00	2.40	2.60	19.00	145.00	0.88	77.00
5	Waterbuck	M	8.0	47.00	25.76	8.00	84.40	104.57	58.97	6.69	5.92	208.00	135.60	11.70	120.23
6	Waterbuck	F	16.00	34.00	26.68	24.03	44.93	252.31	125.31	5.68	7.36	118.44	134.75	3.67	40.08
7	Waterbuck	F	20.00	69.00	13.80	88.10	57.07	166.67	99.90	7.31	6.21	104.41	149.83	5.90	68.28
8	Waterbuck	M	9.00	39.00	29.00	15.00	98.00	103.0	88.00	10.96	4.86	221.0	136.00	12.60	176.00
9	Waterbuck	F	11.00	74.00	16.68	36.03	54.93	142.31	108.31	12.68	9.66	98.44	144.00	9.00	38.12
10	Waterbuck	F	22.00	62.00	19.80	55.10	67.07	233.67	117.90	11.31	8.10	109.41	144.03	5.20	63.00
11	Waterbuck	M	8.00	49.00	31.00	9.80	97.00	107.0	76.00	8.90	4.08	211.0	106.40	12.12	116.00
12	Waterbuck	F	20.00	79.00	19.00	80.10	37.07	166.67	104.00	7.31	8.21	104.41	139.83	11.90	41.28
13	Zebra	M	4.00	16.00	35.02	76.88	70.13	27.42	95.82	0.59	1.82	36.75	105.54	1.73	80.15
14	Zebra	F	8.00	28.00	6.90	8.01	15.47	20.43	124.85	2.65	3.08	16.27	134.75	1.93	192.36
15	Zebra	M	6.00	29.00	11.04	64.81	45.33	48.39	82.00	1.84	2.28	34.17	144.07	1.17	90.23
16	Zebra	F	7.0	10.00	42.32	12.42	24.80	22.09	72.79	0.87	1.31	13.15	133.90	2.00	178.02
17	Zebra	M	4.00	26.00	35.52	66.88	50.13	37.12	85.82	1.86	2.02	36.15	145.04	1.23	96.18
18	Zebra	F	8.00	17.00	16.88	28.01	15.98	24.43	134.00	1.95	3.83	19.97	134.00	0.93	104.24
19	Zebra	M	5.00	30.00	32.00	59.81	47.33	34.20	92.40	0.84	1.98	41.00	146.07	1.71	73.8
20	Zebra	M	4.00	19.00	59.04	34.81	25.33	38.19	89.00	1.04	2.89	33.67	124.12	2.00	49.23
21	Zebra	F	8.0	12.00	22.42	42.06	18.80	22.00	102.00	2.60	2.41	17.43	143.00	1.97	118.00
22	Zebra	M	5.00	26.00	33.12	78.00	66.13	47.00	75.00	1.59	2.02	36.85	145.94	1.83	36.18
23	Zebra	M	4.00	27.00	35.56	66.66	71.00	48.42	95.82	0.59	1.82	42.15	139.54	1.78	96.18
24	Zebra	F	8.00	17.00	16.88	18.01	15.98	20.00	134.00	2.42	1.83	13.97	144.00	1.93	128.24
25	Zebra	M	4.00	28.00	22.00	79.81	77.33	34.20	92.40	0.84	2.18	31.00	146.07	1.71	83.8
26	Zebra	M	6.00	29.00	11.04	64.81	45.33	48.39	82.00	1.84	2.28	34.17	144.07	1.17	90.23

27	Zebra	F	7.0	10.00	42.32	12.42	24.80	22.09	72.79	0.87	1.31	13.15	133.90	2.00	178.02
28	Eland	F	11.00	40.00	46.00	94.42	10.27	19.90	96.30	2.56	13.54	22.35	128.80	1.17	116.41
29	Eland	M	18.00	70.00	164.40	19.22	2.13	29.76	125.77	6.33	6.66	12.95	129.66	7.02	240.45
30	Eland	F	17.00	45.00	159.16	91.29	12.13	10.48	39.62	12.70	17.03	30.31	152.54	11.10	119.80
31	Eland	M	33.00	58.00	339.48	70.48	31.73	5.43	112.14	21.33	8.18	27.25	144.07	4.70	296.68
32	Eland	M	38.00	70.00	264.40	19.22	33.83	13.90	135.00	23.03	7.66	24.00	129.00	5.00	253.00
33	Eland	F	19.00	55.00	59.16	81.88	17.03	20.88	36.80	12.70	19.00	33.00	156.94	6.00	121.00
34	Eland	M	29.00	78.00	139.00	48.00	23.69	7.13	98.00	27.83	9.00	27.00	124.00	7.90	256.30
35	Eland	M	31.00	83.00	99.89	20.88	31.73	4.93	102.04	21.00	7.00	17.88	119.68	8.00	276.00
36	Eland	M	32.00	60.00	104.00	14.60	12.00	13.00	135.00	26.33	5.89	14.00	129.00	7.12	243.00
37	Eland	F	17.00	45.00	75.86	71.00	22.83	30.18	39.60	8.00	13.86	30.00	162.44	1.80	101.20
38	Eland	F	21.00	50.00	86.00	64.42	31.00	29.90	76.00	4.86	15.04	32.15	158.00	3.20	116.00
39	Eland	M	28.00	69.00	246,00	33.20	10.11	14.20	138.00	17.20	6.10	12.00	110.00	5.00	209.20
40	Wildebeest	M	13.00	31.00	10.12	27.23	78.00	217.75	70.03	1.63	3.79	10.17	97.46	8.40	32.06
41	Wildebeest	F	11.00	31.00	4.14	1.60	69.33	191.40	58.51	1.56	3.91	27.60	116.95	3.90	24.05
42	Wildebeest	M	15.00	36.00	12.0	22.30	88.00	237.80	88.00	1.88	4.00	13.49	88.28	3.02	40.06
43	Wildebeest	F	10.20	32.00	6.93	11.80	65.00	188.00	52.81	1.08	3.24	31.80	121.00	4.00	20.95
44	Wildebeest	M	13.70	35.00	11.30	24.00	98.00	234.06	77.00	1.98	3.09	18.66	77.80	3.20	38.00
45	Wildebeest	F	09.00	28.80	7.76	12.30	59.00	189.14	49.90	1.64	4.11	32.60	128.05	4.80	28.66
46	Wildebeest	M	15.50	35.70	13.82	20.30	85.80	246.45	80.00	1.48	3.00	17.77	97.00	2.98	36.42
47	Wildebeest	F	11.80	32.00	5.44	10.00	61.83	179.80	51.38	1.78	3.89	27.00	126.86	5.90	28.28