PREVALENCE AND RISK FACTORS FOR NEWCASTLE DISEASE AND GASTRO-INTESTINAL HELMINTHS OF DOMESTIC PIGEONS (*Columba liviadomestica*) IN KANO METROPOLIS, NIGERIA

BY

ABDULRASHID UMAR

P13VTVM8006

JUNE, 2017

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BY

ABDULRASHID UMAR

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A THESIS SUBMITTED TO THE SCHOOL OF POSTGRADUATE STUDIES, AHMADU BELLO UNIVERSITY ZARIA IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF MASTER OF SCIENCE IN AVIAN MEDICINE

DEPARTMENT OF VETERINARY MEDICINE
AHMADU BELLO UNIVRSITY
ZARIA, NIGERIA

JUNE, 2017

DECLARATION

I hereby declare that the work in this Dissertation entitled "**Prevalence and Risk Factors For Newcastle Disease and Gastro-intestinal Helminths of Domestic Pigeons (*Columba liviadomestica*) in Kano Metropolis Nigeri**a, has been carried out by me in the Department of Veterinary Medicine, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria under the supervision of Dr. A.M. Wakawa and Professor O.J. Ajanusi. The work of other investigators referred to in this study was duly acknowledged. No part of this Dissertation has been previously submitted for another degree, diploma or certificate at this or any other Institution.

Abdulrashid UMAR _____ _____

Signature Date

CERTIFICATION

This Dissertation titled "**PREVALENCE AND RISK FACTORS FOR NEWCASTLE DISEASE AND GASTRO-INTESTINAL HELMINTHS OF DOMESTIC PIGEONS (*Columba liviadomestica*) IN KANO METROPOLIS,NIGERIA**"by Abdulrashid UMAR meets the regulations governing the award of the degree of Master of Science of Ahmadu Bello University Zaria, and is approved for its contribution to scientific knowledge and literary presentations.

Dr A.M Wakawa Chairman, Supervisory Committee	_____ Signature	_____ Date
Prof, O.JAjanusi Member, Supervisory Committee	_____ Signature	_____ Date
Prof. L.B. Tekdek Head, Department of Veterinary Medicine	_____ Signature	_____ Date
Prof. S.Z. Abubakar Dean, School of Postgraduate Studies	_____ Signature	_____ Date

DEDICATION

This Dissertation is dedicated to my parentsHammaAdama and Maimunatu who always encouraged and supported me when Iam at crossroads, and my wife Rabi who partake in all part of the project.

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ABSTRACT

Newcastle disease (ND) and gastrointestinal (GIT) helminths remain constant threats to pigeon production in Nigeria and other parts of the world. It was against this background that this study was carried out, to determine the prevalence and risk factors for ND and GIT helminths of domestic pigeon (*Columba liviadomestica*) in Kano Metropolis, Nigeria. A total of 780 each of blood samples and GIT contents (comprising 260 each from households, live bird markets(LBM) and commercial farms) were collected from purposively sampled pigeons in the study area. Haemagglutination inhibition (HI) test was conducted according to standard protocols on serum samples to determine the presence and titre of ND antibodies. Parasitological examinations were performed on the GIT contents using gross examination for adult helminths and Simple flotation technique for helminth eggs. A structured questionnaire was also administered to owners of pigeons to evaluate the risk factors for occurrence and spread of ND and GIT helminths. Pearson Chi square was used to determine if the risk factors were significant, while Univariate Odd's Ration was used at 95% confidence interval (CI) to determine the strength of association between the factors and ND and GIT helminths. The overall seroprevalence of ND antibodies in domestic pigeons was 28.1%. The prevalence of ND in pigeons in households, LBMs and commercial farms were 26.2%, 16.5% and 41.5%, respectively. The overall mean ND antibody titre of the domestic pigeons was $2.13 \pm 0.65 \log_2$. The highest mean ND antibody titre was observed in pigeons in commercial farms ($2.38 \pm 0.34\log_2$) when compared to those in households ($1.90 \pm 0.20 \log_2$) and LBMs ($2.12 \pm 0.26 \log_2$). An overall prevalence rate of 46% was observed for GIT helminths, with the domestic pigeons predominantly infested with *Raillietinatetragona* (12.9%), *Raillietinaechinibothrida* (13.2%), *Railliettinacesticillus* (7.4%), *Ascaridiacolumbae* (6.2%)

and*Ascaridiagalli* (6.3%). Thehighest prevalence of intestinal helminths was observedin domestic pigeons in households (22.6%) in comparison to those in LBMs (12.2%) and commercial farms (11.3%). The study revealed that 12 (63.2%) of the 19 risk factors evaluated were significant ($p \leq 0.05$). The most significant risk factor was allowing pigeons to mix with other birds species ($p = 0.001$, OR = 11.58, 95%CI = 0.24-54.34) and allowing pigeons access to poultry house ($p = 0.002$, OR: 11.12, 95%CI: 0.80 – 6.36). The present study established that pigeons in the study area might have been exposed to ND virus or pigeon paramyxovirus type 1. The low ND HI titre implies that pigeons in the study area were not adequately protected against ND, and infestation of the domestic pigeons by GIT helminths was high. Majority of the risk factors for the occurrence and spread of ND and GIT helminths in domestic pigeons in Kano Metropolis were significant. Further studies should be conducted to isolate the ND virus and other parasites of domestic pigeons in the study area.

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LIST OF ABBREVIATIONS AND SYMBOLS

AB	Antibody
ABU	Ahmadu Bello University
Ag	Antigen
ANOVA	Analysis of Varaince
APMV	Avian paramyxovirus
^{o}C	Celsius
CI	Confidence Interval
CDC	Centre for Disease Control
Columbids	Members of the bird family Columbidae
e. g.	For example
ELISA	Enzyme Linked Immuno-sorbent Assay
et. al.	And others
FAO	Food and Agriculture Organization
FCT	Federal Capital Territory
FDL & PCS	Federal Department of Livestock and Pest Control Services
FGN	Federal Government of Nigeria
>	Greater than
GIT	Gastrointestinal tract
H	Haemagglutinin
HA	Haemagglutination
HAU	Haemagglutination Unit
HI	Haemagglutinin Inhibition
<	Less than

KSMI	Kano State Ministry of Information
LBMs	Live Bird Market (s)
LGAs	Local Government Areas
Log_2	Log to base 2
LPAI	Low Pathogenic Avian Influenza
LWBM	Live Wild Bird Market
µl	Microliters
MDT	mean death time
ml	Milliliters
NADIS	National Animal Disease Information System
ND	Newcastle Disease
NDV	Newcastle Disease Virus
NP	Nuclear protein
nm	Nanometer
NVRI	National Veterinary Research Institute
O.D	Optical Density
OIE	Office International des' Epizootics
OR	Odds Ration
%	Percentage
P	Probability
PBS	Phosphate Buffered Saline
PCR	Polymerase Chain Reaction
PPMV	Pigeons paramyxovirus

RBCs	Red Blood Cells
RNA	Ribonucleic Acid
S.E.	Standard Error
SP	Speckled Pigeon
S/P	Sample to Positive Ration
SPF	Specific Pathogen Free
Spp	Species
SPSS	Statistical Package for Social Science
UK	United Kingdom
UNDP	United Nation Development Programme
USA	United states of America
WB	Wild Bird
WHO	World Health Organization
X^2	Chi Square value

CHAPTER ONE

INTRODUCTION

1.1 Background of the Study

The poultry population in Nigeria was estimated at 104.3 million, comprising 72.4 million chickens, 11.8million ducks, 4.7million guinea fowls, 15.2 million pigeons and 0.2million turkeys (FDLPCS, 1992). Pigeons are one of the few domesticated birds, kept by humans for variety of purposes such as food, hobby (racing) and treatment of various diseases (Sari *et al*., 2008).The contributions of pigeons have not yet been defined in relation to the contribution of livestock sub-sector and whole poultry production, though the pigeons provide alternative source of animal protein. Comparatively low investment, less care, less feed and housing cost are involved, easy and economic husbandry practices, are required for pigeon farming. This is in addition to the short reproduction cycle and less disease occurrence (Barber *et al.*, 2010).

However, a variety of diseases affect pigeons, but viral diseases predominate (Opara *et al*., 2012; Chaka *et al*., 2013). Newcastle disease (ND) is among the most important viral diseases of poultry in Nigeria as it causes severe economic losses in bird populations, resulting from illness, reduced egg production, immunosuppression, and death (Abdu *et al*., 2006).Newcastle disease ranked as one of the chief constraints to the development of rural poultry production in Nigeria and in most developing countries, triggering serious dangers and concerns (Shamaki *et al.*, 1989; Oladele *et al*., 2003).

In pigeons, ND is caused by pigeon paramyxovirus serotype-1, which is closely related, but not identical to the paramyxovirus causing the disease in other avian species (Alexander, 2003). The

disease in pigeons is characterized mainly by sudden onset of listlessness, in-appetence, nervous manifestations and inability to fly (Jaganathan *et al*., 2015). Morbidity and mortality averages 100 and 80%, respectively (Otim *et al*., 2007). Several outbreaks of Newcastle disease have been reported in pigeon lofts throughout the world (Eisa and Omer,1984). The susceptibility of pigeons (*Columba livia domestica*) and other members of the *Columbidae* family to ND viruses has been related by several authors (Kapczynski *et al.*,2006) and it became clear that the disease in pigeons occurs as a result of the spread from diseased chicken flocks, as well as from domesticated or feral pigeons to poultry (Toro *et al.,* 2005).

Several health problems can affect pigeons, but parasite infections play a major role (Marcia et al., 2007). It is also important to note that pigeons feed on a wide range of food items, which include grains, slugs, earthworm and insects, and these in many instances may carry infective stages of helminths (Soulsby,1982; Marcia *et al*., 2007). Several helminths infections are considered as one of the greatest impediments to profitable pigeon production in Nigeria (Opara *et al*., 2012)

1.2 Statement of Research Problems

Several helminths have been implicated in causing morbidity and mortality in domestic pigeons (Soulsby,1982). Helminths infections may have particularly deleterious or debilitating effects on infected birds, especially the young ones (squabs), causing retarded growth by interfering with healthy development. In addition, infection makes older birds prone to secondary infections (Soulsby, 1982).

Generally, there is paucity of information on parasites of pigeons in Kano Metropolis. In Zaria, the only two reports on helminths of pigeons are those of Abdu *et al* (2004) and Adang *et al* (2008). Reports on studies done outside Zaria include those of Dede and Richards (1998) and Atsineka and Banke (2006) in the North Eastern and North Central (Makurdi) part of Nigeria, respectively.Several helminths have been implicated in causing morbidity and mortalityin domestic pigeons (Atsineka and Banke, 2006), and are considered as the greatest impediments to profitable pigeon production in Nigeria (Abdu *et al*., 2004).

Pigeon plays an important role in the epidemiology of several poultry diseases, for example, Geidam *et al*. (2006) reported that pigeons experimentally infected with chickens isolate of Infectious bursal disease virus, did not manifest any clinical signs throughout the period of the study, but demonstrated antibodies against the virus.In Nigeria, ND is endemic with the occurrence of outbreaks even after vaccination (Sa'idu et al.,2004; Nwanta et al., 2008; Sa'idu et al., 2008). The disease has being regarded as the most economically important disease that devastated village poultry in Nigeria (Abdu *et al*., 2005).

It was reported that the Newcastle epizootic in Europe during 20^{th} century were as a result of chickens fed feed contaminated with pigeon faeces (Eisa and Omer,1984).

1.3 Justification of the Research

Profitable pigeon farming may be an easy and reliable source of employment opportunity, way of family labour utilization and cash income. Sustainable and increased rate of pigeon farming

may also reduce the deficiency of animal protein in meals of the populace, reduce poverty and improve the socio-economic status of the poor rural communities (Bunbury *et al*., 2008; Catroxo et al., 2011). In the light of the aforementioned reasons, the demand for domesticated pigeons (*Columba livia domestica*) is of late on the increase as indicated by records in slaughter houses and sales at barbecue spots in many towns and villages of Northern Nigeria,thus making pigeons more expensive than beefand chicken sold at these barbecue spots. The report by Eisa and Omer (1984) which linked the 20th century ND epizootic in Europe to feeding of chickens with feed contaminated with faeces of ND-infected pigeons necessitate that the ND status in our local pigeons be determined using serology. Also, assessment of helminth burden in pigeons is needful, since infection may affect the overall wellbeing, and consequently, market value of the birds. The findings of this study may provide useful information to other researchers as well as farmers of pigeons in order to increase productivity in Kano State and other parts of Nigeria.

1.4 Aim of the Study

The aim of this study was to determine the prevalence of ND and GIT helminths in domestic pigeons, as well as the risk factorsassociated with acquisition of each of the two diseases.

1.5 Objective of the Study

Objectives of this study were to:

i. Determine the seroprevalence of Newcastle disease in domesticpigeons in Kano Metropolis, Nigeria.
ii. Identify and determine the prevalence of gastrointestinal helminths in domesticpigeons in Kano Metropolis, Nigeria.

iii. Evaluate the risk factors for occurrence and spread of Newcastle disease and gastrointestinal helminths in domesticpigeons in Kano Metropolis, Nigeria.

1.6 Research Questions

i. What is the seroprevalence of ND in domesticpigeons in Kano metropolis, Nigeria?

ii. What is the prevalence of GIT helminths of domestic pigeons in Kano metropolis, Nigeria?

iii. What are the risk factors that may contribute to occurrence and spread of ND and GIT helminths of domestic pigeons in Kano Metropolis, Nigeria?

CHAPTER TWO

LITERATURE REVIEW

2.1Introduction

Newcastle disease (ND) is an acute, rapidly spreading, contagious, nervous and respiratory disease of birds of all ages caused by the avian Paramyxovirus serotype 1 (Alexander, 2000). Currently identified as the single most dreaded disease in the Nigerian poultry industry (Abdu *et al*., 1985), Newcastle disease is ranked as one of the chief constraints to the development of rural poultry production in Nigeria and most developing countries, triggering serious dangers and concerns (Shamaki *et al.,* 1989; Oladele *et al*., 2003). Alexander (2000) reported that the spread of ND virus to chickens has occurred in several countries, including Great Britain, where 20 outbreaks in unvaccinated chickens occurred in 1984 through feed contaminated by faeces of infected pigeons. In rural Nigeria, it is common sight to find a combination of different poultry species and breeds together in the same compound, including chickens, turkeys, ducks and pigeons (Ibrahim and Abdu, 1992).

2.2 Historical Background of Newcastle Disease

The first outbreaks to be recognized and termed Newcastle disease (ND) occurred in chickens in 1926, in Java, Indonesia (Kraneveld, 1926), and in Newcastle-upon-Tyne, England (Cosgrove, 1962). However, there were earlier reports of similar disease outbreaks in Central Europe (Halasz, 1912). In particular, Macpherson (1956) attributed the death of all the chickens in the Western Isles of Scotland in 1956 as being due to Newcastle disease. It is possible therefore, that

ND did occur in poultry before 1926, but its recognition as a specifically defined disease of viral aetiology dates from the outbreaks during this year in Newcastle-upon-Tyne (Macpherson, 1956).

The first confirmed outbreak of ND in Nigeria was between 1952- 1953 in Ibadan,and is currently endemic in both local and commercial poultry with annual epidemics recorded in highly susceptible flocks (Abdu *et al*., 1985; Saidu *et al*., 1994).Serological evidence of ND infection in pigeons has also been reported in Nigeria (Oladele *et al.,* 1996).

2.3 Ae**tiology of Newcastle disease**

According to taxonomy of virus, NDV belongs to order Mononegavirales, family Paramyxoviridae and subfamily Paramyxovirinae (Cattoli *et al*., 2011). The subfamily is divided into five genera: *Mor-billivirus*, *Respirovirus*, *Henipavirus*, *Rubulavirus*, and *Avulavirus* (Miller *et al*., 2009); all the avian paramyxoviruses (APMVs) are part of genus *Avulavirus*. The virus exists in 10 serotypes; APMV-1 to APMV-10 (Waheed *et al.,* 2013), but all NDV isolates belong to serotype 1 (APMV-1). APMV-1 is synonymous with NDV (Cattoli *et al.,* 2011). Virions are roughly spherical; 150 nm or more in diameter and filamentous (Catroxo *et al.,* 2011). The genome is about 15.2 kb in length that codes for six structural and two non-structural proteins (Choi *et al.,* 2010). 'Rule of six' should be followed by genome because it should be of polyhexameric length to replicate rapidly. It encodes for six proteins in 3' to 5' direction; these are Nucleoprotein (NP), Large RNA polymerase (L), Fusion (F), Hemag-glutinin Neuraminidase (HN), Matrix (M) and phosphor-protein (P) (Al-habeeb *et al.,* 2013). The proteins W and V are additionally created within the P gene during transcription of mRNA at editing site by insertion

of guanines (Linde *et al*., 2011). In virus particles, NP is the most abundant protein which provides the NDVs core helical nucleocapsid structure. NP is the main regulator in replication of viral genome (Kho *et al.*, 2004). The genomic RNA is associated with NP, P and L proteins to form RNP complex, which serve as template for RNA synthesis (Kho *et al.*, 2004). NP is found to be highly immunogenic, as it induces antibody responses in chickens (Ahmad-Raus *et al.*, 2009). APMV-1 strains maintained in pigeon populations have some antigenic differences from other NDV isolates, and are sometimes called pigeon paramyxovirus type 1 (PPMV-1), (Alexander, 2000).

2.4 Epidemiology of Newcastle Disease

2.4.1 Host range

Although, Newcastle disease viruses (NDV) have been reported to infect animals ranging from reptiles to man (Beard and Hanson, 1984), the main host for the virus are birds. Alexander (1997) reported that NDV infections have been established in at least 241 species of birds representing 27 of the 50 Orders of the class.

2.4.2 Wild birds

Newcastle disease virus (NDV) isolates have been obtained frequently from migratory feral waterfowl and other aquatic birds(Alexander, 2001).Wild birds and semi-domestic birds are known to be susceptible and develop antibodies to ND hence, play major roles in the spread of ND viruses especially in Nigeria as well as other African countries with poor poultry husbandry practice (Ezeokoli *et al*., 1984; Oladele *et al*., 1996; Sa'idu *et al*., 2004). The circulation of ND

viruses in wild birds and maintenance in the environment were responsible for most outbreaks in backyard and commercial poultry even after vaccination (Sa'idu *et al*., 2008). Most of these isolates have been of low virulence for chickens and similar to viruses of the "asymptomatic enteric" pathotype. The most significant outbreaks of virulent NDV in feral birds have been those reported in double-crested cormorants (*Phalacrocorax auritus*) in North America during the 1990s (Alexander, 2001). Disease in double-crested cormorants was observed again in Canada in 1995 and in California in 1997 and in both instances NDV was isolated from dead birdsand the viruses appear to be closely related (Kuiken, 1998).

2.4.3 Caged "pet birds"

Virulent NDV isolates have often been obtained from captive caged birds (Senne *et al.*, 1983). Cattoli *et al* (2011) thought it unlikely that infections of recently imported caged birds resulted from enzootic infections in feral birds in the countries of origin. They considered that the infections probably originated at holding stations before export, either as a result of enzootic NDV at those stations or of spread from nearby poultry such as backyard chicken flocks.

2.4.4 Domestic poultry

Virulent NDV strains have been isolated from all types of commercially reared poultry, ranging from pigeons to ostriches (Alexander, 1997).

2.4.5 Racing and show pigeons

In the late 1970s, an NDV strain, PPMV-1, showing some antigenic differences from classical strains, appeared in pigeons. It probably arose in the Middle East and subsequently produced a true panzootic, spreading in racing and show pigeons to all parts of the world (Alexander, 1997). The effect of panzootics of ND have had on the poultry populations of different countries has not always been well recorded. Alexander (2001) documented the history of ND in Great Britain in detail and considered it a good example of the effect ND may have on the poultry industry in a developed Western country where eradication policies have been employed(Alexander, 1997).

2.5 Occurrence of Newcastle Disease

Velogenic NDV is endemic in areas of Mexico, Central and South America, widely spread in Asia, the Middle East and Africa, and in double-crested wild cormorants in the US and Canada. Lentogenic strains of NDV are worldwide in their distribution while mesogenic pathotypes are widespread with a special adaptation to pigeons (Alexander, 2001).

2.6 Sources of Newcastle Disease Virus

Some of the sources of infection of the NDV include respiratory secretions/discharges and faeces of infected birds and all parts of dead carcass (Abdu *et al.*, 2006). NDV is shed during the incubation period, during clinical stages and for a limited period during convalescence period. Wild birds and waterfowl may act as reservoir hosts for lentogenic pathotypes of ND; subsequently, these viruses could become virulent following mutation upon establishment in domestic poultry. Some psittacine birds have been demonstrated to shed ND virus intermittently for over 1 year and been associated with introduction into poultry (Alexander, 2000).

2.7 Transmission of Newcastle Disease Virus

Newcastle disease virus (NDV) can infect more than 240 species of birds and it spreads primarily through direct contact between healthy and infected birds (Alexander, 2001; Gilchrist, 2005). Several reviews have dealt with the way in which the ND virus may be introduced into a country or area, and subsequently spread from flock to flock. In summary, the main methods by which virus can be spread are:

2.7.1 Movement of live birds

Migratory feral birds may be responsible for the primary introduction of infection, but nearly all NDV isolates obtained from feral birds have been of low virulence(Alexander, 2001). World trade in captive caged birds is enormous and in many countries virulent NDV has been isolated frequently from such birds held in quarantined (Senne *et al*,. 1983). For example, 147 virulent NDV isolations were made from 2 274 lots of captive birds held in quarantine in the USA during 1974-1981 (Senne *et al*,. 1983). Some infected psittacines have been shown to excrete virulent virus intermittently for extremely long periods, in some cases for more than one year (Alexander, 1997). This further emphasises the potential role these birds may have in the introduction of NDV to a country or area. There is also considerable international trade in game birds, which are often imported for immediate release(Alexander, 1997).

The potential for racing pigeons to carry and introduce ND into a country or area has been highlighted by the panzootic in such birds over the last 10 years (Catroxo *et al*., 2011). Modern

methods of slaughter of commercial poultry, marketing of poultry meat and veterinary inspection, have reduced the movement of live commercial poultry (with the exception of day-old chicks) in many European and other developed countries(Alexander, 2001). However, in many countries, the normal method of trade is by live poultry markets. Such markets, where birds of many different species may be placed in close contact with each other, represent ideal opportunities for viruses to be disseminated. The movement of village chickens from one village to another, whether directly or through live bird markets, is the main method of spread of ND (Catroxo *et al*., 2011).

2.7.2 Movement of people and equipment

It has been reported that secondary spread during most epizootics of ND has been the result of the movements of personnel or equipment. Human beings may be infected with NDVs, but their most likely role is the transfer of infective poultry faeces from one site to another via clothing, footwear, crates, feed sacks, egg trays or vehicles (Senne *et al*., 1983; Alexander, 2003; Anosa and Adene, 2007).

2.7.3 Movement of poultry products

In the past, poultry meat has been seen as the main vehicle for the introduction and spread of NDV. Modern methods of poultry carcass preparation as well as legislation on the feeding of untreated swill to poultry have greatly diminished the risk from poultry products, but the possibility of spread in this way still remains (Alexander, 2003; Anosa and Adene, 2007)

2.7.4 Contaminated poultry food or water

In the British Isles, outbreaks of ND in commercial poultry have been associated with food contaminated with infective faeces from feral pigeons infected with the ND virus. Similarly, water contaminated with infective faeces may introduce NDV to a flock (Alexander *et al.*, 1985: Boostani *et al*., 2013).

2.7.5 Air-borne spread

In recent years, the significance of air-borne transfer of viruses has been the subject of some debate. There have been few attempts to assess the survival of airborne virus, but Hugh-Jones *et al*. (1973) were able to detect virus 64 metres but not 165 metres downwind of infected premises. These authors stressed the importance of environmental conditions, particularly relative humidity and wind speed with regard to the likelihood of air-borne spread. It is possible that when climatic conditions were right and poultry farms sufficiently concentrated, as in Northern Ireland in 1973 (Alexander, 1997), airborne spread may have played a significant role in epidemics of ND. But air-borne spread has not been an issue in reported outbreaks and there has nearly always been an alternative and more likely cause, particularly the movement of poultry and humans (Nwosu and Okeke, 1989).

2.7.6 Vaccines

Good manufacturing practices should ensure that vaccines are not carriers of virulent ND virus (OIE, 2012). However, in the past, birds have become infected by vaccines for other diseases being contaminated with ND and also as a result of failure to properly inactivate vaccines prepared from virulent ND virus. In 1996-1997, a series of ND isolates of low virulence were obtained from poultry flocks in Denmark, a country which pursues a non-vaccination policy for ND(Jorgensen *et al*., 2000). It was demonstrated that these viruses were the result of

contamination of avian virus vaccines with vaccinal ND viruses. This episode further emphasized the potential of spread of ND in this way(Jorgensen *et al*., 2000).

2.7.7 Non-avian hosts

This is likely to be by mechanical transfer of infective faeces, for example, by insects, rodents or scavenging animals. In some countries, reptiles may enter poultry houses and should not be ignored as potential spreaders of NDV, as their susceptibility to infection has been reported (Kouwenhoven, 1993).

2.8Pathogenesis of Newcastle Disease

Newcastle disease virus usually enters the body via the respiratory and/or the intestinal tract. Following multiplication of the virus at the site of introduction, it spreads rapidly to the bloodstream where the virus is carried to different organs (Alexander, 2003). The spread of the virus largely depends on the virulence of the strain. While lentogenic strains are present only at low titers in the circulation, mesogenic strains spread to the kidneys, lungs, bursa, and spleen. Virulent NDV can be found within 22-44 hours in nearly all tissues, with the highest titers in the thymus and lowest titers in the muscles and brain (Linde *et al*., 2011). During the second replication, the virus titer increases again and the 'virus is released into the bloodstream. This is associated with the appearance of general disease signs and virus excretion into the environment via feces and respiratory secretions. Virulent neurotropic viruses infect the central nervous system at the same time as the respiratory and intestinal tract. Viruses pass the blood-brain barrier at an accelerated rate, causing severe damage to this barrier (Kouwenhoven,1993; Snoeck *et a*l., 2013).

2.9 Clinical Signs of Newcastle Disease

Newcastle disease in pigeon is characterized by sudden onset of restlessness, inappetence, nervous manifestation and inability to fly. Morbidity and mortality can be 100% and 80%, respectively (Abdu, 2007). This virus is currently endemic in the pigeon population throughout the world. The PPMV-1 infection should always be kept in mind when high mortality or mild to severe illness affects pigeon flocks. Juvenile pigeons are very susceptible to infection; at times, the morbidity and mortality in young bird flocks may reach 100%, with nervous signs being dominant. In contrast, affected adults may completely recover after 10-14 days of illness (Cattoli *et al.,* 2011). In adult birds morbidity is variable, but often below 10%, with death occurring as a result of chronic disease and emaciation. In addition, subclinical infection seems to be common, contributing to the spread of the virus (Cattoli *et al.,* 2011). The incubation period is 7-14 days, with virus being shed in the faeces as early as 2 days post-infection. Infection may spread directly during the incubation period or indirectly through contaminated fomites (cages, transport vehicles, exhibitions) (Alexander *et al.,* 2003).

Clinical signs in naïve birds resemble those of the neurotropic form of ND in chickens. Initially, affected birds exhibit general signs, such as poor body condition, reduced feed intake, increased drinking (polydipsia), increased excretion of urea (polyuria), lethargy, inability to fly and ruffled feathers. During the next few days, clinical signs of neuronal disorders appear, such as incoordination, abnormal gait, tremors, paresis of the legs and/or wings, head tilting, torticollis and greenish diarrhoea (Cattoli *et al.,* 2011).

2.10 Pathology of Newcastle Disease

2.10.1 Gross lesions

The tissues or organs where gross lesionsappear usually depends on the tropism of the NDV strain causing the infection(Alexander, 2003). The virulent NDV causes haemorrhagic lesions of the intestinal and respiratory tracts, although there are no pathognomonic gross lesions (Boostani *et al*., 2013).

2.10.2 Histopathology

Histopathology of NDV depends on the viral strain and route of infection. In the nervous system, there are foci of glia cells, perivascular infiltration of lymphocytes and proliferation of endothelial cells in the cerebellum, medulla and spinal cord (Alexander, 2001). However there is hydropic degeneration, hyalinization of capillaries and arterioles. Similarly, there is thrombosis in small vessels and necrosis of endothelial cells of the vessels. There is also change in the intestinal, respiratory and reproductive system (Alexander, 2003).

In pigeons, histopathological changes seen in the central nervous system include non-supperative inflammation in the brain and spinal cord, usually involving the meninges with cuffs of lymphocyte around the blood vessels and necrosis of neurons and oedema of endothelial cells. However, non-suppurative nephritis comprising multiple small areas of lymphocytes and plasma cells infiltration of renal parenchyma were common ND lesions in pigeons. Focal non-suppurative pancretitis has been reported in infected Rock pigeon (Alexander, 1997).

2.11 Pathogenicity

In some circumstances infection with the extremely virulent viruses may result in sudden, high mortality with comparatively few clinical signs. Although none of the variable clinical signs can be regarded as pathognomonic, certain signs do appear to be associated with particular viruses. This has resulted in the grouping of viruses into five "pathotypes" on the basis of the predominant signs in affected chickens (Beard and Hanson, 1984; Abdu *et al*., 2002; Anosa and Adene, 2007) as follows:

2.11.1 Viscerotropic velogenic

These are viruses responsible for ND, characterised by acute lethal infections, usually with haemorrhagic lesions in the intestines of dead birds.

2.11.2 Neurotropic velogenic

These are viruses causing ND, characterised by high mortality which follows respiratory and neurological signs, without signs of GIT involvement.

2.11.3 Mesogenic

These are ND viruses causing clinical signs consisting of respiratory and neurological signs, with low mortality.

2.11.4 Lentogenic

These areND viruses causing mild infections of the respiratory tract.

2.11.5 Asymptomatic enteric

These viruses causing avirulent infections in which replication appears to be primarily in the guts.

2.12 Molecular Basis of Pathogenicity

The genome of NDV encodes for six major structural proteins. Viral replication, transcription and translation occur in the cytoplasm of the host cell, while virus particles are assembled in plasma membrane by budding (Choi *et al*., 2010; Madadgar *et al.,* 2013). Important pathogenic marker of NDV exists in F protein (Madadgar *et al.,* 2013). Disulphide linkage is present between F1 and F2. These proteins enable the virus to attach to the host cell membrane (Wen et al., 2007). At cleavage site, F0 protein has two pairs of basic amino acids that can be cleaved by the host proteases (Gould *et al*., 2001; Wen *et al*., 2007). Highly virulent NDV has three or more basic amino acids, which are lysine (K) or arginine (R) present at 113 - 116 residues and phenylalanine (F) at position 117 (Choi *et al*., 2010; OIE, 2012). Cleavage of F0 protein is due to the presence of these basic amino acids in virulent NDV (Boostani *et al.,* 2013). It has been found that avirulent viruses have 112G/E-K/R-Q-G/E-R-L117 and virulent viruses have 112R/K-R-Q-K/R-R-F117 amino acid sequence at cleavage site. Most of the pathogenic APMV-1 viruses for chicken have sequence 112R/K-R-Q/K/R-K/R-R116 (Gould *et al*., 2001). Office of International Epizootics (OIE) accepts F cleavage sequence as determinant of primary virulence (Boostani *et al.,* 2013). However, if this cleavage sequence is not found, then an Intra Cerebral Pathogenicity Index (ICPI) is required for determination of the virulence (Choi *et al*., 2010; Boostani *et al.,* 2013).

2.13 Differential Diagnosis of Newcastle Disease

The differential diagnoses for ND include fowl cholera, highly pathogenic avian influenza, Infectious laryngotracheitis, fowl pox (diphtheritic form), psittacosis (psittacine birds), mycoplasmosis, Infectious bronchitis, Aspergillosis (Alexander, 1997).ND can only be differentiated from other acutepoultry respiratory diseases such as AI byserological tests (Agar gel immuno-diffusiontest) and virus isolation. Samples for virusisolation should be taken from the trachea,lungs, airsacs, cloacal swabs and faeces.

2.14 Diagnosis of Newcastle Disease

2.14.1Clinical signs and lesions

Rapid and accurate diagnosis of ND outbreak is important because it clinically resembles highly pathogenic avian influenza (AI) (Khan *et al.*, 2010). For a definitive diagnosis of ND, both virus isolation and laboratory characterisation are necessary. Nevertheless, if the disease is known to be present in a given area, signs and lesions may be considered highly suggestive, especially for village chickens. Typical clinical signs are: a state of prostration and depression in the birds, with ruffled feathers; greenish white diarrhoea; and, in survivors, torticollis (turning of the head to one side) is very often seen, as are paralysis of the legs, wings or other neurological signs (Cattoli *et al*., 2011).

Other typical characteristics of the disease include: an incubation period of 2-3 days or in rare cases, 2 -15 days ,rapid spread; death within 2-3 days; a mortality rate of over 50 percent in naïve populations (Beard and Hanson, 1984).At necropsy, the typical lesions are mucus in the trachea, and usually haemorrhages in the intestine, particularly in the proventriculus. It should be borne in

mind that all the preceding signs and lesions can be caused by other diseases (Cattoli *et al*., 2011).

2.14.2 Serological diagnosis

In the absence of vaccination, the presence of specific antibodies against the ND virus indicates that the bird has been infected by the virus at some time, but not necessarily that it was suffering from the disease at the time of sampling. In practice, a high antibody titre is indicative of a recent infection (Docherty and Friend, 2009; OIE, 2012). Two methods are used to measure antibody titres: the haemagglutination inhibition (HI) test, and the enzyme-linked immunosorbent assay (ELISA). For both, it is necessary to collect blood samples from the birds. Blood can be drawn directly into a syringe, or collected into a tube after piercing the vein with a needle. In both cases, the sample is subsequently placed nearly horizontally to allow clotting and to permit separation of the serum sample, which should be straw-coloured. The serum sample should be kept cool until it can be frozen in the laboratory (OIE, 2012).

2.14.2.1 Haemagglutination inhibition test

The haemagglutination inhibition (HI) test is based on the principle that the haemagglutinin on the viral envelope can bring about the agglutination of chicken red blood cells and that this can be inhibited by specific antibodies. V-bottomed microtitre plates are used (OIE, 2012). The serum samples are diluted in serial two-fold dilutions in phosphate buffered saline and then a fixed quantity of viral antigen is added to each well. Usually 4 Haemagglutination Units are used (OIE, 2012). Following incubation, a suspension of red blood cells is added to each well and the

plate is incubated again. In the absence of any antibody against the virus, haemagglutination occurs, appearing as a diffuse red colour at the bottom of the well. In the wells where the antibody against the virus is of a sufficient level, haemagglutination is inhibited and the red blood cells sediment and appear as a small pellet at the bottom of the well. The presence or absence of agglutination is accurately assessed by tilting the plates. Only those wells in which the RBCs stream at the same rate as the control wells (containing RBCs and PBS only) are considered to show inhibition.

The HI titre is the reciprocal of the highest dilution of serum which completely inhibits haemagglutination and is usually and most conveniently expressed as the logarithm to the base 2 (OIE, 2012). Although the test is difficult to standardise between laboratories, the HI titre gives an indication of the immune status of the bird. A titre of $\log_2 3$ is indicative of protection and a titre of $\log_2 6$ or more suggests a recent infection by the virus. If no vaccination has taken place, diagnosis of the infection can be made on this basis, although it cannot be determined exactly when it took place (Maas, *et al*. 1998). Sequential samples taken at different times can indicate whether the titre is rising - indicative of a recent infection - or declining.When the HI titre is used as a measure of immunity (for example, when testing flock immunity following vaccination), it is recommended that an avirulent strain such as V4 or Ulster 2C be used as the viral antigen. The La Sota antigen has been found to be unsuitable for this purpose when vaccination is done by the same strain as it results in an overestimation of protective serum antibody titres (Maas, *et al*. 1998).

2.14.2.2 Enzyme-linked immunosorbent assay

Enzyme-linked immunosorbent assay (ELISA) works on the principle of recognition of anti-NDV antibodies, attached to a plate coated with viral antigen, by antibodies produced in another species against chicken antibodies (Bell*et al*., 1991). This anti-chicken antibody is conjugated to an enzyme that catalyses a reaction, causing a change of colour which can then be read quantitatively on a photo spectrometer designed to read microtitration plates. The Animal Production and Health Section of the Joint FAO/IAEA Division has produced an ELISA kit for antibodies against NDV which is designed to be easily transportable and give uniform results under widely varying ambient temperatures. HI and ELISA titres show a good degree of correlation and ELISA titres can be interpreted in reciprocal values to HI titres (Bell*et al*., 1991).

2.14.3 Virus isolation

The definitive diagnosis of ND is done through isolation and identification of the virus (Alexander, 1998). Tracheal and cloacal swabs are good sources for isolation from living birds without having to kill them. A cotton-covered stick is inserted into the trachea or cloaca, and then put into a vial containing phosphate buffered saline plus penicillin and streptomycin. It is important to ensure that cloacal swabs are coated with faeces. These samples must be kept cool during transport to the laboratory where they should be stored at 4°C if they are to be processed within 48 hours or frozen at least at -20°C until the isolation attempt. Although cloacal swabs or faeces should always be sampled, virus can also be isolated from homogenised organs from dead birds, chosen to reflect the clinical signs(Alexander, 1998). Nine-day-old embryonated fowls' eggs are injected with 0.1 mL of the suspension into the allantoic cavity and returned to incubation. The eggs are candled twice daily. As dead eggs occur, they are chilled, together with

all the eggs after 5-7 days incubation, are chilled at 4°C at which point the allantoic fluid is then harvested and tested for its ability to haemagglutinate chicken red blood cells. Diagnosis is based on the inhibition of haemagglutination by specific anti-NDV serum. This proves infection of the bird by the virus, but does not indicate whether the virus is a pathogenic or avirulent strain (Alexander, 1998).

2.14.4 Pathogenicity tests

The full diagnosis of ND requires an assessment of the virulence of the virus. The current definition allows either molecular characterisation by nucleotide sequencing and deduction of the F0 cleavage site amino acid sequence or an *in vivo* estimation of virulence. The recommended *in vivo* test is the intracerebral pathogenicity index (ICPI) test in day-old chicks (OIE 2012). This involves the inoculation of virus derived from fresh infective allantoic fluid into the brain of ten-day-old chicks from specific pathogen-free parents. Each bird is examined at 24-hour intervals for eight days and graded zero if normal, one if sick and two if dead. The index is the mean score per bird per observation over the 8-day period (OIE, 2012). The most virulent viruses give ICPI values approaching the maximum score of 2.0, while lentogenic viruses give values of, or close to, 0.0. Where day-old chicks are not available, the mean death time (MDT) in eggs (i.e. the mean time in hours for the minimum lethal dose to kill all the inoculated embryos), can be used as a guide to virulence. The MDT has been used to classify ND virus strains into velogenic(taking under 60 hours to kill); mesogenic(taking 60 to 90 hours to kill); and lentogenic(taking more than 90 hours to kill) (OIE, 2012).Intravenous pathogenicity index (IVPI) can also be used in which freshly collected infective allantoic fluid (which should be no older than 24-48 hours and should have tested negative for bacterial contamination) with a HA

titre of >24 (>1/16) is diluted 1/10 in sterile isotonic saline, 0.1 ml of the diluted virus is injected intravenously into each of ten 6-week-old SPF chickens (OIE, 2012). Birds are examined at 24-hour intervals for 10 days and scored at each observation: 0 if normal, 1 if sick, 2 if paralysed or showing other nervous signs, and 3 if dead(OIE, 2012).The intravenous pathogenicity index (IVPI) is the mean score per bird per observation over the 10-day period. Lentogenic strains and some mesogenic strains will have IVPI values of 0, whereas the indices for virulent strains will approach 3.0(OIE, 2012).

2.15 Public Health Importance of Newcastle Disease

Humans are among the many species that can be infected by NDV in addition to avian species. NDV may cause conjunctivitis in humans, when a person has been exposed to large quantities of the virus (Alexander, 2000). Mostly, Laboratory workers and vaccinators are affected. The use of personnel protective equipment and biological safety cabinet has reduced the exposure of laboratory workers. Infection is rarely seen in the workers of a farm; moreover persons handling or consuming poultry products do not appear to be at risk (Alexander, 2003).The conjunctivitis usually resolves rapidly, but the virus will be shed in the ocular discharges from 4 to 7 days. In some cases, mild, self-limiting influenza-like disease with fever and headache has also been reported in humans (Alexander, 2000). There is no evidence found to support human to human transmission but the potential for human to bird transmission exists (Alexander, 2003).

2.16 Economic importance of Newcastle disease

Proteins are a significant part of balanced human diet. There are mainly two proteins sources,animals and plants. In developing countries, human diet is deficient in the animal proteins; approximately 66% population has protein-deficient diet (Maqbool, 2002). A single person per day requires 102.7 g protein, while only 69.61 g protein is used by a person per day. The global economic impact of ND is enormous and it may represent a bigger drain on the world's economy than any other animal virus. In countries with intensive poultry production, ND claims millions of dollars every year due to extensive vaccination campaigns, culling of birds, outbreak containment measures, and trade restrictions. For instance, the latest U.S. outbreak occurred in California in 2002 with an overall cost of $160,000, mostly due to bird depopulations and the strict limitations imposed on movement of poultry and poultry products. ND and avian influenza (AI) are major concerns of animal husbandry due to hazardous infections (Ge *et al.,* 2012). All over the world, poultry industry is facing severe economic losses with every passing year (Khan *et al.,* 2010).

In countries with extensive poultry production and a subsistence economy,lost of bird due to ND decreases the availability of animal proteins (meat and eggs) for human consumption or resale. It is estimated that 70% of all poultry products and 20% of human protein intake consumed in Africa derives from poultry that typically scavenge for the majority of their diet and are raised under a wide range of production conditions (village poultry). In these settings, ND is a serious threat, being able to severely affect the economies of small farmers and entire communities (Ge *et al*., 2012).

Newcastle disease poses a serious threat as it has economic and ecological impact on pet, free-living, as well as domestic birds. An average of 200-250 outbreaks of the disease are reported in Nigeria annually (Okeke and Lamorde, 1988). The virus strains prevalent in nature are highly virulent (velogenic) and are kept in circulation by vast populations of wandering local chickens, apparently normal ducks and free-flying birds (Abdu *et al.*, 1985). The isolation of velogenic viscerotropic ND virus from wild birds during ND outbreaks in poultry has led to the suggestion that wild birds could be important vehicles for the spread of the disease in Nigeria (Haruna *et al*., 1993).

2.17 Prevention and Control of Newcastle disease

2.17.1 Biosecurity and hygiene in the control of Newcastle disease

In countries or areas that are free of virulent NDV, the primary aim should be to prevent the introduction of the virus. Because migratory and other feral birds frequently carry NDV strains of low virulence, which spread from time to time to domestic poultry, it is usual to exclude such viruses from control policies (Miller *et al*., 2009). Vaccinal viruses are somewhat different, as some are sufficiently virulent to cause disease in fully susceptible birds (Kapczynski *et al*., 2006).

In some countries there is legislation designed to reduce the likelihood of outbreaks from specific sources. For example, on the island of Ireland there has been a legal requirement to heat-treat poultry feed to reduce the possibility of introduction of NDV by this route (Radfar *et al*., 2012). On commercial farms, control measures should attempt to prevent viruses from infecting the flock. It is of paramount importance that good hygiene and biosecurity measures aimed at

preventing the introduction of viruses by the routes described above are practised at all times on poultry farms (Radfar *et al*., 2012).

Biosecurity aimed at preventing disease should begin at the planning stage of commercial poultry farms. Farms and flocks should be well separated, hatcheries should be isolated from poultry farms, different species should be reared on different sites, and there should be an adequate fresh water supply, preferably one that does not draw on surface water (Radfar *et al*., 2012; Shewantasew *et al*., 2012). Often, in developed countries such practices are difficult to impose as the poultry industry may already be established in areas with high concentrations of poultry flocks and with little opportunity to change due to limitations of available land. But in countries where the commercialisation of poultry farming is at a developmental stage, these points should be adopted (Radfar *et al*., 2012; Shewantasew *et al*., 2012).

On the farms the following points should be observed: houses, food stores and water tanks should be bird-proofed; movements on and off the farm should be kept to a minimum; all equipment, especially vehicles, should be disinfected before access to the site is permitted; movements between different farms for egg collection, carcass collection, food delivery and the like should be confined to a specified collection and delivery point away from the poultry flocks (Shewantasew *et al*., 2012). Unfortunately, as poultry farming has become industrialised and its profits marginalised, the move has been away from such precautions which are often considered an expensive luxury. Visits by personnel such as bleeding or vaccination teams, inseminators and veterinarians are the most likely method of introduction of ND and if such visits are unavoidable,

regimens of clothing change, equipment disinfection and other basic hygiene controls must be enforced (Radfar *et al*., 2012; Shewantasew *et al*., 2012).

Possibly, the greatest aid to implementing biosecurity and hygiene measures that will assist in the prevention or control of ND is the education of farmers and those working with poultry with respect to the spread of virus and measures to avoid it. The best tool for the control of ND at any level, international, national or local farm, may well be an efficient, well-manned poultry extension service (Radfar *et al*., 2012; Shewantasew *et al*., 2012).

2.17.2 Sanitary prophylaxis

The following preventive measures have been recommended: bird-proof houses, feed and water supplies; proper carcass disposal; pest control in flocks;; avoidance of contact with birds of unknown health status including newly acquired domesticated poultry, pet birds and wild or feral birds; control of human traffic; facility employees should not have contact with outside birds and consideration of a policy of shower-in with dedicated clothing; control of vehicular traffic; and strict disinfection of conveyances and equipment (Shewantasew *et al*., 2012; Jaganathan *et al*., 2015).

2.17.2 Immuno-prophylaxis

Vaccines are available for chickens, turkeys, and pigeons and are used to induce an antibody response, so vaccinated birds must be exposed to a larger dose of vNDV to be infected (Shamaki *et al*., 1989). Unfortunately, ND vaccines do not provide sterile immunity, and in many areas of the world vaccines are used to prevent losses from sickness and death. Live lentogenic vaccines,

chiefly B1 and LaSota strains, are widely used and typically administered to poultry by mass application in drinking water or by spray (Waheed *et al*., 2013). Mucosal immunity induced in birds vaccinated by live vaccines applied by these routes decreases the amount of vNDV the vaccinated birds will shed if infected with vNDV, compared with the immune response induced by an inactivated vaccine. Mass vaccination methods are less labour intensive but if not applied properly may lead to <85% of the flock being immunized. Alternatively, individual administration of live vaccines is via the nares or conjunctival sac. *Mycoplasma*, some other bacteria, and other viruses affecting the respiratory tract, if present, may act synergistically with some vaccines to aggravate the vaccine reaction after spray administration (Jaganathan *et al*., 2015).Inactivated oil adjuvant vaccines are also used after live vaccine in breeders and layers, and may be used alone in situations where use of live virus vaccine may be contraindicated (eg, in pigeons) (Waheed *et al*., 2013). In countries where vNDV is endemic, a combination of live virus and inactivated vaccine can be used; or alternatively, if permitted by law, a live mesogenic strain vaccine may be used in older birds. The frequency of revaccination to protect chickens throughout life largely depends on the risk of exposure and virulence of the field virus challenge(Jaganathan *et al*., 2015).

2.18 Gastrointestinal Helminths of Pigeons

Helminth parasites including nematode, cestodes and trematodes inhabit the gastrointestinal tract and other visceral organs of pigeons, thereby instigating huge morbidity (Forond*aet al*.,2004). Endoparasitic infection is characterized by anorexia, loss of general body condition and poor growth performance, ultimately predisposing the affected birds to viral and bacterial pathogen

(Foronda *et al.*, 2004). Among the helminth parasites infesting pigeons, roundworm are considered to be the most widespread. Furthermore, endoparasitic infestation can induce immunosuppression, subsequently predisposing the affected birds to bacterial and viral infection (Sari *et al*., 2008).

Pigeons can carry many parasites and pathogens to different flocks (Opara *et al.*, 2012). They can also serve as a source for different zoonotic diseases for humans (Karatepe *et al.*, 2011). A good knowledge about the parasitic disease of the pigeons would aid in the development of possible control measures, which may help in enhancing its survival and complement efforts towards public enlightenment. There is a huge literature on avian medicine including parasitic diseases. However, little has been documented about the parasites in pigeons world wide (Krone and Cooper, 2002). In Nigeria, investigations on the parasites of Columbids are scanty compared with the large volumes of information available on the parasites of other birds such as chicken, guinea fowl, turkey and duck (Abdu *et al.*, 2002).The prevalence and intensity of parasitic infestations may be influenced by a number of epidemiological factors including host factors such as age, sex and breed and environmental factors such as climatic conditions (Nadeem *et al*., 2007).

2.18.1 Overview of helminths of pigeons

Some common helminth species of poultry are presented in Table 2.1. Approximately 100 helminth species have been recognized in wild and domestic birds. Nematodes (roundworms) are the most significant, both in number of species and economic impact. Of helminth species found in commercial poultry, the common roundworm (*Ascaridia galli*) is by far the most common (Eslami *et al*., 2009; Macklin, 2013). Field studies show that poultry maintained under free-range conditions may be heavily parasitized; therefore, control measures such as preventing infections or chemotherapy are likely to improve weight gain and egg production. In surveys of poultry raised under non-confinement conditions throughout the world, an incidence of infection >80% is not uncommon (Eslami *et al*., 2009; Macklin, 2013).

Generally, nematodes have separate sexes that may have morphologic differences; e.g, males of *Tetrameres* spp are elongated and slender, whereas gravid females are globe-shaped. The size

Table 2.1 Common helminths of poultry

Parasite	Host(s)	Intermediate Host	Organ Infected	Pathogenicity
Nematodes				
Amidostomum anseri	Duck, goose, pigeon.	None	Gizzard	Severe
Capillaria caudinflata	Chicken, turkey, duck, game birds, pigeon	Earthworms	Small intestine	Moderate to severe
Capillaria obsignata	Chicken, turkey, goose, pigeon, quail	None	Small intestine, ceca	Severe
Dispharynx nasuta	Chicken, turkey, game birds, pigeon	Sowbugs	Proventriculus	Moderate to severe
Ornithostrongylus quadriradiatus	Pigeon, dove	None	Small intestine	Severe
Tetrameres americana	Chicken, turkey, duck, game birds, pigeon	Grass-hoppers, cockroaches	Proventriculus	Severe
Trichostrongylus tenuis	Chicken, turkey, duck, game birds, pigeon	None	Ceca	Severe
Cestodes				
Raillietina cesticillus	Chickens pigeon	Beetles	Duodenum, jejunum	Mild
Raillietina echinobothrida	Chickens pigeon	Ants	Lower intestine	Severe, nodules
Raillietina tetragona	Chickens Pigeon	Ants	Lower intestine	Moderate to Severe
Davainea proglottina	pigeons	Snails	Lower intestine	Moderate to Severe
Trematode				
Paratanaisia bragai	Pigeon	Snails	kidney	Moderate

Source: Macklin (2013)

and shape of nematode species vary widely; ascarids are sturdy and long (up to 4.5 in. [116 mm]); capillarids are more delicate, slender, and long (2.3 in. [60 mm]); and other nematodes are much shorter (0.08–0.48 in. [2–12 mm]) (Soulsby, 1982; Macklin, 2013).

Cestodes (tapeworms) also vary in size. *Raillietina* spp may be >12 in. (30 cm), whereas *Davainea proglottina* often is <0.16 in. (4 mm). The proglottids of individual tapeworms are hermaphroditic (Oniye *et al*.,2000). The invertebrate host and or vector for the cysticercoids of *Raillietina* sppare ants of the genera *Pheidole* and *Tretramorium* (Soulsby, 1982). Among the *Raillietina* spp of cestode found in pigeons, *echinobothrida* is the most pathogenic. Intestinal nodules at the sites of attachment of this parasite caused hyperplastic enteritis (Oniye *et al*.,2000). *Raillietina tetragona* which was the most prevalent cestode in pigeons and caused moderate to sevare pathogencity and obstruction of the intestine, weight loss and decrease egg production in laying pigeons (Soulsby, 1982).

Trematode infection in birds might be associated with feeding habits. In the area where the birds are looking for food, infected snails, such as *Subulina octona*, can act as an intermediate host for *P. bragai* may be ingested (Menezes *et al*., 2001). Mild to severe pathological lesions caused by species of the genus *Paratanaisia* have been identified in a number of wild avian species such as ruddy ground doves, *Columbina talpacoti* (Silva *et al*., 1990). This fluke has also been reported to infect domestic pigeons (*Columba livia*) (Silva *et al*., 1990). Parasitism by this renal flukes may result in a granulomatous interstitial nephritis, and ultimately lead to chronic renal failure and death (Bunbury*et al*., 2008).

2.18.2 Transmission of gastrointestinal helminths of pigeons

Modern confinement rearing of poultry has significantly reduced the frequency and variety of endoparasite infections, which are common in ranged birds and in backyard flocks. However, severe parasitism still may be seen in floor-reared layers, breeders, turkeys, or pen-reared game birds where management problems exist (Eslami *et al*., 2009). Contributing factors include the use of poorly managed built-up litter (which fosters the propagation of intermediate hosts and the accumulation of infective eggs) and resistance of the parasites to therapeutic drugs. Range infections of nematodes such as *Heterakis gallinarum* and *Syngamus trachea* may increase because of seasonal or climatic abundance of specific invertebrate transport hosts, such as earthworms and beetles (Eslami *et al*., 2009).

Nematodes have either a species-specific, direct life cycle with bird-to-bird transmission by ingestion of infective eggs or larvae, or an indirect cycle that requires an intermediate host (eg, insects, snails, or slugs). Eggs of many nematode species are resistant to low temperatures and disinfectants, but may be more susceptible to heat and desiccation (Sari *et al*., 2008; Macklin, 2013).Cestodes require an intermediate host (eg, insects, crustaceans, earthworms, or snails). Floor layers, breeders, and broilers are infected with *Raillietina cesticillus* by ingestion of the intermediate host, small beetles that breed in contaminated litter. Cage layers in unscreened houses may become infected with *Choanotaenia infundibulum* by eating its intermediate host, the house fly. Litter beetles in proximity may also serve as intermediate hosts (Macklin, 2013).

2.18.3 Pathogenesis and clinical findings

Ascaridia, *Heterakis*, and *Capillaria* spp are widely distributed and cause such nonspecific signs as general unthriftiness, inactivity, depressed appetite, and retarded growth; in severe cases,

death may result. A mere few ascarids may depress weight, and larger numbers may block the intestinal tract (Sari *et al*., 2008). *C contorta* in the mucosae of the crop and esophagus, and *C obsignata* in the wall of the small intestine, cause marked thickening and inflammation of the organs. Birds harboring large numbers of these thread-like worms become weak and emaciated, and may die (Macklin, 2013).Among other nematodes, *Amidostomum anseris* attacks the gizzard lining and causes dark discoloration, necrosis, and sloughing at the parasitic loci. *Dispharynx nasuta* causes ulceration, thickening, and maceration of the proventriculus; heavily infected birds may die. *Tetrameres americana*, a bright-red worm discernible through the proventricular wall, causes diarrhea, emaciation, and with heavy infection, death. *Trichostrongylus tenuis* causes inflamed ceca, weight loss, anemia, and death, especially in young birds. *Ornithostrongylus quadriradiatus*, a blood-sucking trichostrongyle, causes pigeons to regurgitate bile-stained fluid mixed with food; greenish mucoid diarrhea from hemorrhagic intestines, emaciation, and death may follow (Radfar *et al*., 2012; Macklin, 2013).

Most pathogenic tapeworms are found in the small intestine; the scolex, usually buried in the mucosa, generally causes mild lesions. *Davainea proglottina* may cause weight loss. *Raillietina tetragona* causes weight loss and decreased egg production; *R echinobothrida* produces granulomas at its attachment sites ("nodular disease") (Radfar *et al*., 2012).

2.18.4Diagnosis of gastrointestinal helminths of pigeons

A reliable diagnosis can be made only by accurate identification of the individually recovered parasites; careful and complete necropsy techniques are essential. Only by specific recognition of

the parasite can meaningful recommendations for flock therapy and management be made (Radfar *et al*., 2012; Macklin, 2013).

2.18.4Treatment and control of gastrointestinal helminths

Improvement of management and sanitation in confined operations will generally lower the parasite levels in the birds. In range birds, the only option is to move to new pastures, although the benefit that may result will be of short duration. Application of approved insecticides to soil and litter when premises are unoccupied may interrupt the life cycle of the parasite by destroying its intermediate host. When the premises are restocked, groups of birds of different species or ages should be widely separated to avoid spread of parasites. Migration of litter beetles or other insects may infect new or widely separated housing (Nnadi and George, 2010; Macklin, 2013).

CHAPTER THREE

MATERIALS AND METHODS

3.1 The Study Area

Kano Metropolis is located at the Central Western part of Kano State between latitude 11059´59.57 – 12002´39.570N of the equator and between longitudes 8033´19.69 – 8031´59.690E (Figure 1). It lies in the North Central boundary of Nigeria and is located some840 km away from the edge of the Sahara desert and 1,140km from the Atlantic Ocean (Oseiki, 2009).Kano State is located in the semi-arid zone of Northern Nigeria and enjoys the warm tropical climatic condition of Western Africa. Most parts of Kano State fall within the Sudan savannah vegetation zone, whereas the far southern area falls within the Guinea vegetation zone.

The two major types of zones identified, pockets of other vegetation also exists (Ahmed, 2008). Its metropolis population is the second largest in Nigeria, after Lagos. Kano state has a mean height of about 472.5m above sea level. Kano city has expanded over the years and has become the third largest in Nigeria.The Kano Urban area covers 137 km² and comprises eight Local Government Areas (LGAs)(KSMI, 2005).

The specific areas of this study included Municipal, Gwale, Dala, Tarauni, Nassarawa, Fagge, Ungogo and Kumbotso LGAs. The live bird markets (LBMs) enrolled for the study werethe ones atSabon Gari, Sharada, Kurmi, Rimi and Tarauni LBMs.

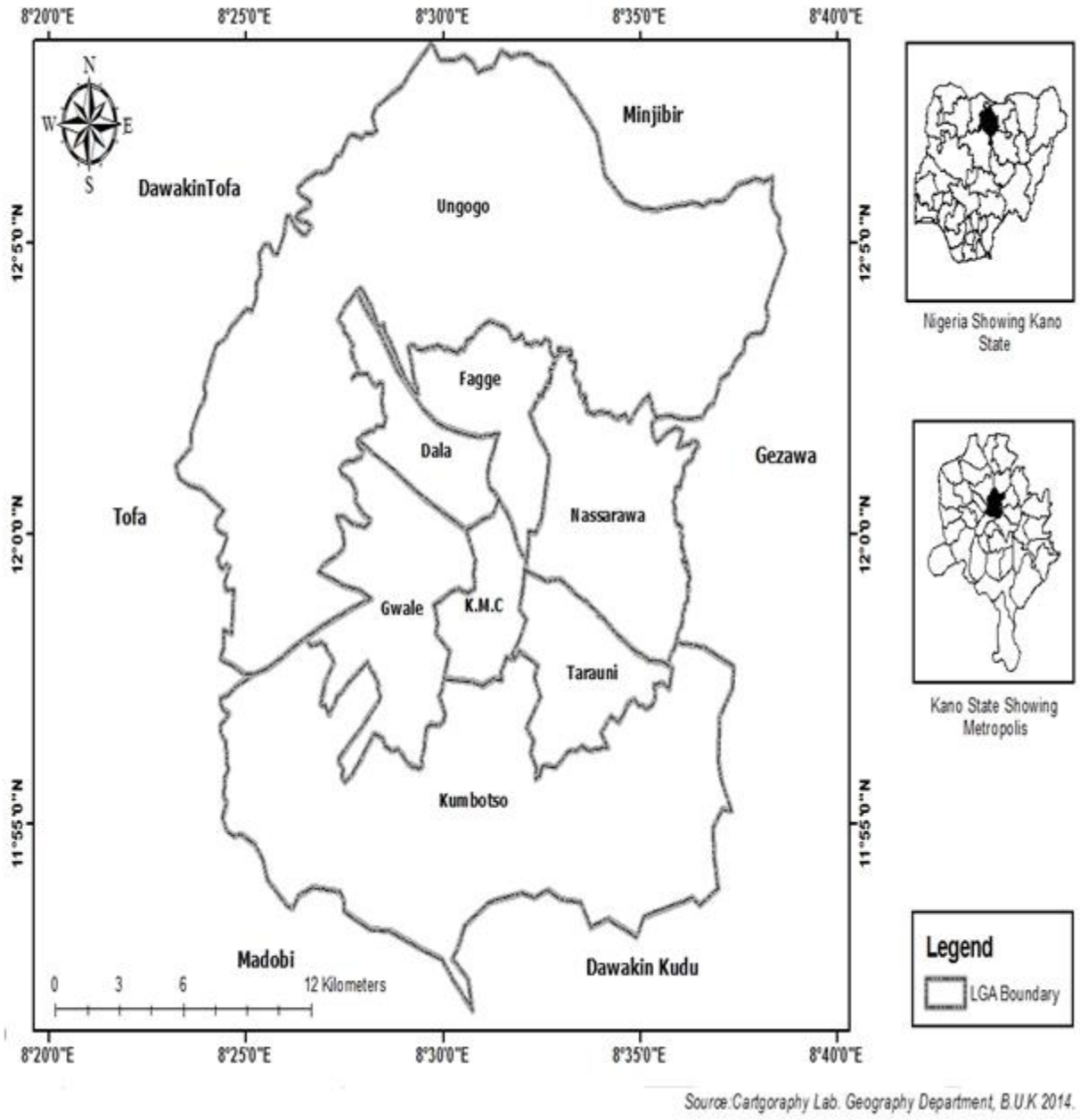


Figure 1: **Map of Kano Metropolis and its eight Local Governments Areas (KSMI, 2005).**

3.2 Sampling

3.2.1 Sample size

The sample size was determined using the formular describedby Thrustfied (2007) based on 95% confidence interval and prevalence of 50%.Thus:

$$N = \frac{z^2pq}{d^2}$$

Where N = Sample size, Z = Appropriate value for the standard normal deviation for the desired confidence interval (1.96), P = Prevalence = 50%, q = Complementary probability (1-p), d = Level of significance 5% (0.05).

Therefore, $N = \frac{1.96^2 x 0.5\ (1-0.5)}{0.05^2} = 768$ (The sample size was rounded up to 780)

3.2.2 Sampling techniques and units

Between the months of May to September, 2016, purposive sampling method was used to collect a total of 780 blood samples and gastrointestinal contents from domestic pigeons (irrespective of age, size and sex) comprising 260 each from LBMs, commercial farms and households. A total of 16 households, 5 LBMs and 10 commercial farms were enrolled for this study.

3.2.3.1Sample collection and processing

One to two ml of blood sample was collected through jugular venipuncture at slaughter or using 23 Gauge needle under suitable restrain technique with the head and neck extended. The blood samples weretransferred into non-heparinized test tubes and labelled accordingly. The labelled test tubes were placed in a slanting position under shade to allow the blood sample to clot. The sample was then transported in a in a leak-proof, ice-packed containerto Nutrition

Laboratory,Department of Veterinary Medicine,Ahmadu Bello University, Zaria. The blood samples were centrifugedatabout 447.2 g for 5 minutes (Collee *et al*., 1982). The sera were then decanted into serum tubes which were properly labelled.

3.2.3.2Gastrointestinal track

Gastrointestinal tracts of pigeons (780 comprising 260 each from households, LBMs, and commercial farms) werealso collected in a clean labelled polythene bag and transported to Veterinary Helminthology Laboratory, Ahmadu Bello University, Zaria, for helminths identification.

3.3 Laboratory Analyses

3.3.1 Preparation of 1% red blood cells

Two ml of blood was collected from 5 day-old chicks and pooled in an equal volume of Alsever's anticoagulant solution. Cells were washed three times in Phosphate Buffered Saline (pH 7.2) by centrifuging at 447.2 g for 5 minutes (Collee *et al.*, 1982). One percent red blood cells (RBC) (packed cell v/v) suspension was made by adding 99 ml of phosphate buffered saline (PBS) to 1ml of washed RBC.

3.3.2 Source of Newcastle disease antigen

Newcastle disease vaccine La Sota strain was obtained from National Veterinary Research Institute, Vom, Nigeria, and used as antigen for the test. The titre of the antigen was determined using haemagglutination test (HA)as described by OIE (2012).

3.3.3 Determination of titre of Newcastle disease antigen

Haemagglutination test (HA) was carried out according to the method described by OIE (2012) in order to determine the titre of Newcastle disease antigen.

3.3.4 Determination of Newcastle disease virus antibody titre

Haemagglutination inhibition test (HI) was carried out on the sera according to the method described by OIE (2012) in order to determine theNewcastle disease virus antibody titre.

3.4 Identification of Helminthes in the Gastrointestinal Tract of Domestic Pigeons

3.4.1 Examination of gastrointestinal tract of domestic pigeons for helminths

The gastrointestinal tract (GIT) of each bird was cut out with various sections ligated then severed and placed in saline solution in Petri dishes for 20minutes to facilitate detachment of any attached worms. The contents of the proventriculus were washed into petri dishes and the mucosae were scrapped and examined for parasite against a black background with a desk lamp as sourceof light. Honey layer of gizzard was peeled to expose embedded parasites. Parasitic nodules were teased gently to dislodge embedded parasites. Helminths found were detached gently with a pair of forceps. Helminths recovered were treated with few drops of hot

lactophenol for rapid clearance of internal organs, while grossly visible helminths were mounted for examination of morphology under light microscope at x10 magnification, and counted. The recovered helminths were preserved in labeled universal bottles containing 5% formalin for further confirmatory identification. All parasites were identified using the helminthological key described by Soulsby (1982).

3.4.2 Simple flotation technique

This was carried out to detect and identify helminth eggs as described by Lynne and Garcia (1999). Four grams of faeces (intestinal content) was put into a universal bottle containing 5 ml of floatation medium (zinc sulphate solution). The faeces was broken using a glass rod to make a suspension.into the medium and sieved into a centrifuge tube or straight walled test tube. More of the medium wasadded until a convex meniscus was formed on which a cover slip was gently placed and left for 3-5 minutes. Finally, the cover slip was mounted on a glass slide and examined for eggsusing keys described by Soulsby (1982).

3.5 Questionnaire Design and Administration

A structured questionnaire was developed forthis study and pretested first at five households keeping pigeons. The questionnaire was then modified according to new factors encountered during the pre-testing. In the final questionnaire, 19 variables covering information about management practices, environment, physical structures, vaccination, deworming, feed and water sourceswere evaluated. The questionnaires were administered to owners of pigeons in households, live bird markets and commercial farms through interviews during on-site visits.

Personal observations were used to validate the responses of the respondents during the questionnaire administration.

3.6 Data Analyses

Data were reduced into Tables. Antibody titre was expressed as Mean ± standard error of mean. Pearson Chi square was used to determine if there was association between a risk factor and ND and GIT helminths. Odds ratio at 95% confidence interval was used to determine the strength of association between risk factors and ND or GIT helminths. Values of $P \leq 0.05$ were considered significant. Data obtained wascalculated using SPSS version 20.0 (SPSS Inc. Chicago, IL, USA, 20011).

CHAPTER FOUR

RESULTS

4.1 Seroprevalence of Newcastle Disease in Domestic Pigeons

The overall seroprevalence of ND antibodies in domestic pigeons in the study area was 28.1%, Theprevalence of pigeons in households, LBMs and commercial farms were 26.2%, 16.5% and 41.5%, respectively (Table 4.1). The overall mean ND antibody titre was 2.13 ± 0.65 $\log_2$. The highest mean ND antibody titre was observed in pigeons in commercial farms (2.38 ± 0.34) when compared to those in households (1.90 ± 0.20 $\log_2$) and LBMs (2.12 ± 0.26 $\log_2$) (Table 4.1). It was also observed that pigeons in Tarauni LBM had the highest mean ND antibody titre (2.60 ± 0.31) in comparison to pigeons in the other 4 LBMs (Table 4.2).

4.2 Prevalence of Intestinal Helminths in Domestic Pigeons

Helminths of the genera *Raillietina* and *Ascaridia* were found to be the major intestinal parasites of pigeons in the study area, with an overall prevalence rate of 46%. The pigeons were predominantly infected with *Raillietina tetragona* (12.9%) (Plate I and VI), *Raillietina echinibothrida* (13.2%) (Plate II), *Railliettina cesticillus* (7.4%) (Plate III), *Ascaridia columbae* (6.2%) (Plate IV) and *Ascaridia galli* (6.3%) (Plate V and VII) (Table 4.3). The highest prevalence of intestinal helminths was observed in pigeons in households (22.6%) in comparison to those in LBMs (12.2%) and commercial farms (11.3%) (Table 4.4). Of the five LBMs enrolled for this study, the highest prevalence for all the species of helminths recovered from pigoens were observed in Sharada (11.2%) and Tarauni (10.7%) LBMs had the highest prevalence for all the species (Table 4.5).

Table 4.1:Prevalence and mean Newcastle disease antibody titres of domestic pigeons (*Columba livia domestica*) in Kano Metropolis, Nigeria.

Category	No.of units	Sample size per unit	No.of samples positive	Prevalence (%)	Mean ± S.E	P value
Households	10	260	68	26.2	1.90 ± 0.20	0.013
LBMs	5	260	43	16.5	2.12 ± 0.26	
Commercial farms	10	260	108	41.5	2.38 ± 0.34	
Total	25	780	219	28.1	2.13 ± 0.65	

SE: Standard error of mean, LBM: live bird markets

Table 4.2:Prevalence and mean Newcastle disease antibody titres of domestic pigeons (*Columbalivia domestica*) in some live bird markets in Kano Metropolis, Nigeria.

	Location	sample size	Number positive (%)	Mean ± SEM Ab titre (log_2)
1.	Sabongari	52	7 (13.5)	2.20 ± 0.12
2.	Sharada	52	7 (13.5)	1.94 ± 0.18
3.	Kurmi	52	3 (5.8)	2.06 ± 0.09
4.	Rimi	52	18 (34.6)	1.82 ± 0.12
5.	Tarauni	52	8 (15.4)	2.60 ± 0.31
	Total	260	43 (16.5)	2.12 ± 0.26

P<0.05 significant

Table 4.3: Prevalence and predilection sites of intestinal helminths recovered from domestic pigeons (*Columba livia domestica*)in Kano metropolis, Nigeria.

S/n	Species of helminth recovered	Number of samples positive	Prevalence (%)	Site of recovery
1	*Raillietina tetragona*	101	12.9	Doudenum/ileum
2	*Raillietina echinobothrida*	103	13.2	Doudenum/ileum
3	*Raillietina cesticillus*	58	7.4	Ileum
4	*Ascaridia columbae*	48	6.2	Doudenum/ileum
5	*Ascaridia galli*	49	6.3	Doudenum/ileum
	Total	359	46.0	

N = 780

Table 4.4: **Prevalence of gastrointestinal helminths recovered from domestic pigeons (*Columbalivia domestica*)in Kano metropolis, Nigeria.**

Category	Species of helminths recovered (%)					Overall Prevalence (%)
	R. tetragona	*R. chinobothrida*	*R. cesticillus*	*A. columbae*	*A. galli*	
Households	52 (6.7)	61 (7.8)	37 (4.7)	15 (1.9)	11 (1.4)	176 (22.6)
Live bird markets	26 (3.3)	28 (3.6)	11 (1.4)	13 (1.7)	17 (2.2)	95 (12.2)
Commercial farms	23 (2.9)	14 (1.8)	10 (1.3)	20 (2.6)	21 (2.7)	88 (11.3)
Sub-Total	101 (12.9)	103 (13.2)	58 (7.4)	48 (6.2)	49 (6.3)	359 (46.0)
Total			359 (46.0)			

N = 780

Table 4.5: Prevalence of intestinal helminths recoverd from domestic pigeons (*Columba livia domestica*)in live bird markets in Kano metropolis, Nigeria.

	Live bird market	*Raillietina tetragona* (%)	*Raillietina echinobothrida* (%)	*Raillietina cesticillus* (%)	*Ascaridia columbae* (%)	*Ascaridia galli* (%)	Overall prevalence (%)
1	Sabon Gari	3 (1.2)	4 (1.5)	4 (1.5)	2 (0.8)	5 (1.9)	18 (6.9)
2	Rimi	2 (0.8)	0	3 (1.2)	0	3 (1.2)	8 (3.1)
3	Sharada	8 (3.1)	11 (4.2)	2 (0.8)	5 (1.9)	3 (1.2)	29 (11.2)
4	Tarauni	9 (3.5)	9 (3.5)	2 (0.8)	6 (2.3)	2 (0.8)	28 (10.7)
5	Kurmi	4 (1.5)	4 (1.5)	0	0	4 (1.5)	12 (4.6)
	Total	26 (5.9)	28 (7.7)	11 (4.0)	13 (4.8)	17 (6.2)	95 (12.2)

N = 260



PLATE I: Anterior end of *Raillietina tetragona* recovered from a domestic pigeon (*Columbae livia domestica*) showing two sucker (arrowed) in Kano Metropolis, Nigeria(X100)



PLATE II: Anterior end *Raillietina echinobothrida* recovered from a domestic pigeon (*Columbae livia domestica*) showing two suckers (arrowed) in Kano Metropolis, Nigeria(X100).



PLATE III: Anterior end of *Raillietina cesticillus* recovered from a domestic pigeon (*Columbae livia domestica*) showing two suckers (arrowed) in Kano Metropolis, Nigeria(X100).

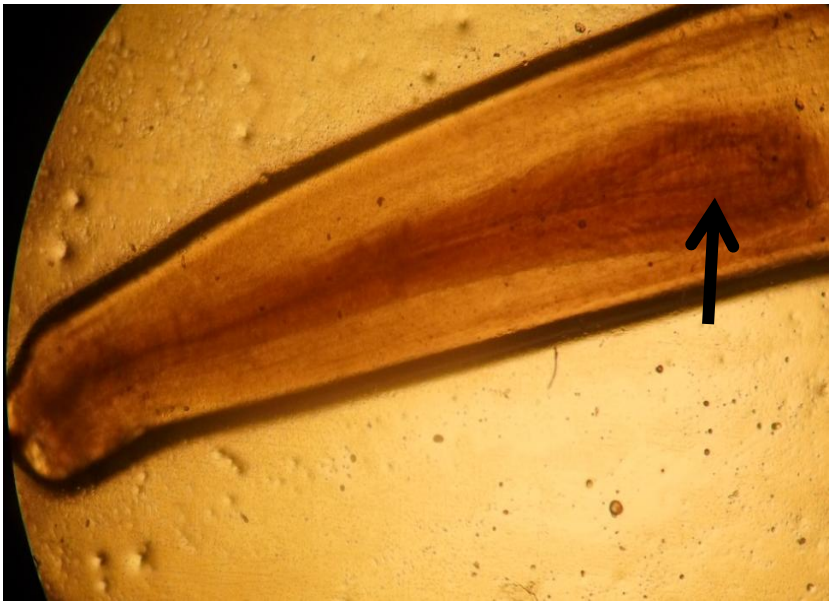


PLATE IV: Anterior end of *Ascridia columbae* recovered from a domestic pigeon (C*olumbae livia domestica*) in Kano Metropolis, Nigeria(X40). Note the characteristic bulb-shaped oesophagus (arrowed).

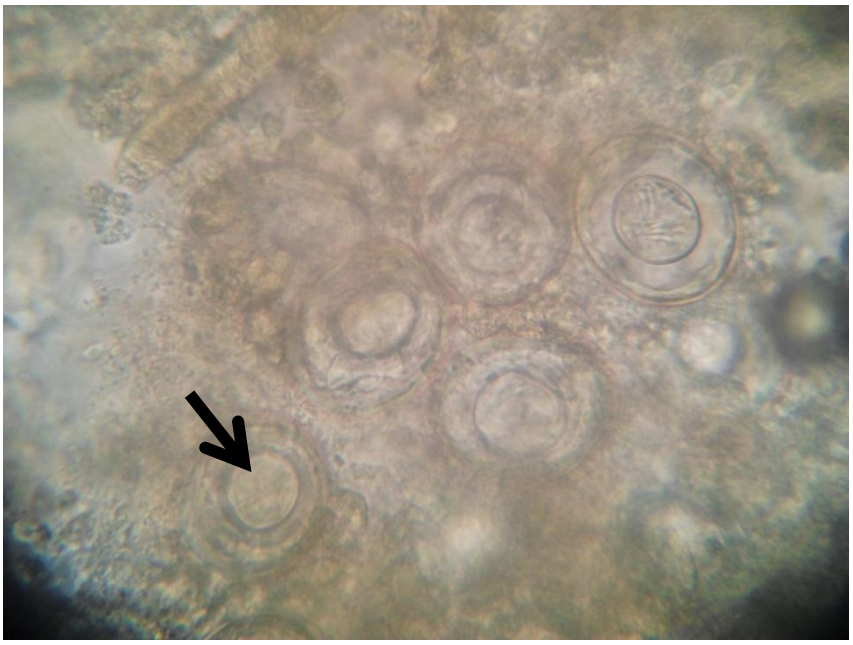


PLATE VI: *Raillietina* eggs (arrowed) recovered from faeces of a domestic pigeon (C*olumbae livia domestica*)in Kano Metropolis, Nigeria(X40).

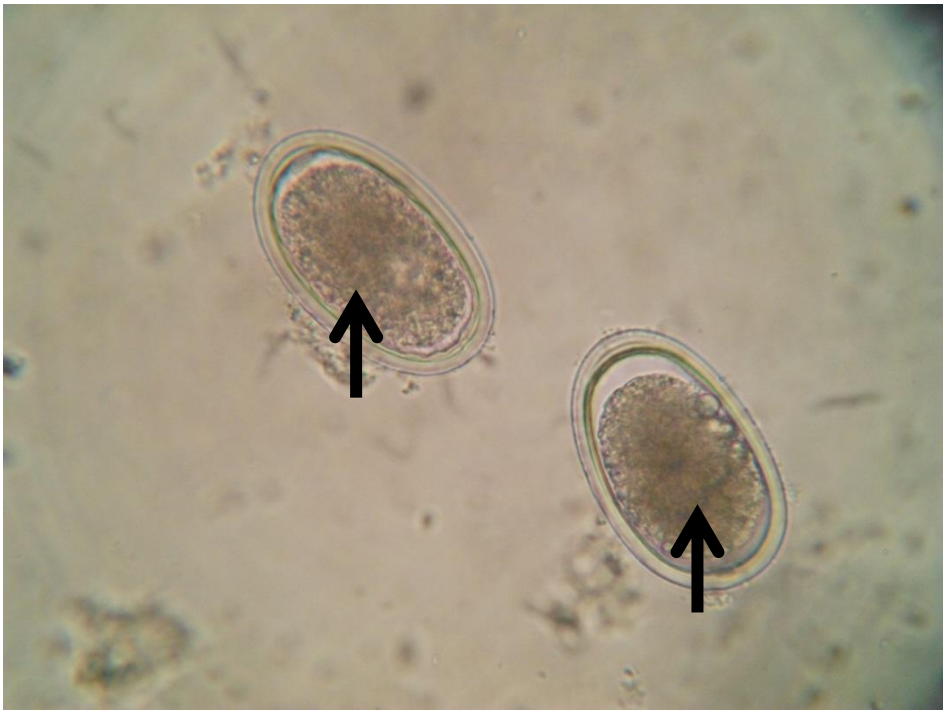


PLATE VII: *Ascaridia* eggs recovered from faeces of a domestic pigeon (C*olumbae livia domestica*)in Kano Metropolis, Nigeria(X40).

Table 4.6: Risk factors for the spread of Newcastle disease and gastro-intestinal helminths of domestic pigeons (*Columba livia domestica*) in Kano Metropolis, Nigeria.

s/n	Question		Response (%)		p value	OR	95% CI
			Yes	No			
1	Source of pigeons:	LBM	14 (60.9)	9 (39.1)	0.030	4.92	2.24 – 20.71
		Distributors	12 (62.2)	11 (47.8)	0.231	0.02	
		Hatcheries	0	23 (100%)			
2	Pigeons mix with other bird species		10 (43.5)	13 (56.5)	0.001	11.58	0.24 – 54.34
3	Pigeons feed together with other birds species		12 (54.5)	10 (45.5)	0.004	5.26	0.03 – 0.87
4	Presence of backyard poultry within premises		13 (56.5)	10 (43.5)	0.017	1.85	0.42 – 5.38
5	Good hygienic practices		5 (21.7)	18 (78.3)	0.349	0.08	0.64 – 18.65
6	Regular cleaning of pigeon house/cage		5 (21.7)	18 (78.3)	0.349	0.08	0.79 – 5.92
7	Routine washing and disinfection of pigeon nest		0	23 (100.0)	0	0	0.32 – 32.60
8	Pigeons access to poultry house		7 (30.4)	16 (69.6)	0.002	11.12	0.80 – 6.36
9	Free range chickens or ducks within the premises		13 (56.5)	10 (43.5)	0.025	3.67	0.63 – 11.59
10	Presence of pets (owls, parrots, doves)		5 (21.7)	18 (78.3)	0.759	0.99	0.23 – 1.87
11	Several pigeon flocks of different ages kept together		23 (100)	0	0	0	0.50 – 15.52
12	Pigeon owner works in poultry house		8 (34.8)	15 (65.2)	0.239	0.08	0.23 – 2.23

OR: Odds Ratio, CI: Confidence Interval, p: Probability, LBM: live bird markets

Table 4.7: Risk factors for the spread of Newcastle disease and gastro-intestinal helminths of domestic pigeons (*Columba livia domestica*) in Kano Metropolis, Nigeria (continued).

s/n	Question	Response (%)		P value	OR	95% CI
		Yes	No			
13	High poultry farm density in the area	2 (8.7)	21 (91.3)	0.608	0.57	0.70 – 5.62
14	Presence of dam/pond as open water source	8 (34.8)	15 (65.2)	0.026	3.80	2.24 – 4.34
15	Presence of roosting trees	11 (47.8)	12 (52.2)	0.053	1.43	0.06 – 0.68
16	Presence of untreated surface water	16 (69.6)	7 (30.4)	0.052	3.22	0.02 – 0.30
17	Presence of cockroaches/dung beetles around pigeon house or manure	16 (69.6)	7 (30.4)	0.051	3.22	0.33 – 2.37
18	Vaccinates pigeons against Newcastle disease	7 (30.4)	16 (69.6)	0.039	4.27	0.27 – 2.97
19	Deworm pigeons routinely	6 (26.1)	17 (73.9)	0.051	2.82	0.33 – 6.97

OR: Odds Ration, CI: Confidence Interval, p: Probability

4.3 Risk Factors for Newcastle Disease and Gastrointestinal Helminths in Domestic Pigeons

This study revealed that 12 (63.2%) of the 19 risk factors for ND and GIT helminths(table 4.6) evaluated in the study area were significant ($p \leq 0.05$). The most significant ($p \leq 0.001$) risk factor is allowing pigeons mix freely with other species of birds, which had the strongest point estimate of risk (odds ratio (OR) 11.58, at 95% confidence intervals (CI) of 0.24-54.34 (Table 4.6). Newcastle disease and GIT helminths are 11.58 times more likely to occur in pigeons that mix withother species of birds. Allowing pigeons access to poultry house is 11.12 times more likely to contribute to the occurrence and spread of ND and GIT helminths in pigeons,(OR: 11.12, CI: 0.80 – 6.36) (Table 4.6).

Othersignificant risk factors for occurrence and spread of ND and GIT helminths in pigeons were: sourcing of pigeons from LBMs (p= 0.03, OR = 4.92, CI= 2.24-20.71), pigeons fed together with other bird species (p = 0.004, OR = 5.26, CI = 0.03-0.87), presence of backyard poultry within premise (p = 0.017, OR = 1.85, CI = 0.42 – 5.38), presence of free range chickens or ducks within premises (p = 0.025, OR = 3.67, CI = 0.63 – 11.59) (Table 4.6).

It was also observed that the presence of dam/pond or open water body may be 3.8 times more likely to contribute to occurrence and spread of ND and GIT helminths in pigeons (p = 0.026, CI = 2.24 -4.34) (Table 4.7), Other significant risk factors associated with physical structures include: presence of trees as roosting places (p = 0.053, OR = 1.43, CI = 0.06 – 0.68) and presence of untreated surface water within premises (p = 0.052, OR = 3.22, CI = 0.02 – 0.30) (Table 4.7).

The presence of cockroaches/dung beetles around pigeon house or manure was also significant ($p = 0.051$, OR = 3.22, CI = 0.33 – 2.37). Non vaccination of pigeons against ND is 4.27 times more likely to contribute to outbreaks of ND in pigeons ($p = 0.039$, OR = 4.27, CI = 0.27 – 2.97). Lack of routine deworming of pigeons may 2.8 times more likely to contribute to infestation of the pigeons by GIT helminths ($p = 0.051$, OR = 2.8, CI = 0.33 – 6.97) (Table 4.7)

CHAPTER FIVE

DISCUSSION

The low overall prevalence rate for ND antibodies in domestic pigeons in the study area suggests that majority of the pigeon population in the study area were not protected against ND as reported by OIE(2012). The result implied that the pigeons might have been exposed to either ND and Pigeon Paramyxovirus type 1 (PPM-type 1). Pigeon Paramyxovirus type 1 (PPMV-1) is closely related to ND virus (NDV), as they are both classified in the same family of viruses, Paramyxoviridae, and are within the same serotype, referred to as Paramyxovirus type 1as reported bySanei *et al*., (2009).

The presence of ND antibodies in the sera of pigeons might be attributed to previous exposure of these birds to theND viruses,since these birds were not routinely vaccinated against ND. This findings is similar to the findingsof Tsai and Lee,(2006) in Taiwan. The low HI antibody titres recorded in pigeons may be a source of concern because they are reported to be among the species of bird that are highly susceptible to ND. On the other hand, it is important to note that PPMV-1 is typically very contagious and can spread from infected pigeons to other pigeons or commercial poultry flocks, because an ND epizootic was reported in Great Britain which is as a result of contamination of feed by faeces of infected pigeons by Alexander (1987).

Previous research suggested that the pigeons has potential of disseminating ND viruses continuously if they are infected, thereby infecting commercial exotic breed of birds reared in intensively managed farms (Sa'idu *et al*., 1994; Abraham-Oyiguh *et al*., 2014).

Previous research has suggested that the pigeons have been disseminating ND viruses continuously if they are infected, thereby infecting commercial poultry exotic breed of birds reared in intensively managed farms (Sa'idu*et al*., 1994; Abraham-Oyiguh *et al*., 2014).

The mean ND antibody titres of pigeons in all three epidemiologic units studied (household, LBM and commercial farms) were all below protection level when compared to the recommended minimum protection antibody titre against ND, i.e, 4.0 $\log_2$as recommended by (OIE (2012). Therefore, in the event of infection by ND virus, their antibodies may not be sufficient to protect the pigeons against infection. It is again important to note that owners of these multi-species backyard flocks rarely vaccinate their birds against ND as reported by previous research (Alexander, 2000; Musa *et al*., 2009).It is also important to note that Ibrahim and Abdu (1992) reported thatThese backyard poultry species are reared under relatively semi-intensive system of management. And their movement is mostly unrestricted and there is no vaccination against ND for these pigeons.

The finding that the highest prevalence for ND antibodies was observed in domestic pigeons in Rimi LBM implied that these birds may be of importance in the spread of ND viruses to other species of poultry, more importantly to the commercial chickens. It is important to note that, in 1984, a number of outbreaks in commercial poultry flocks in the UK, with clinical signs such as drop in egg production, nervous signs and increased mortality were attributed to infection with PPMV-1(Sanei *et al*., 2007). Contamination of feed with pigeon feces and carcasses was considered the main source of most of these outbreaks (Sanei *et al*., 2007).

In this study the overall prevalence rate of GIT helminths in the domestic pigeons is considerably high, which indicates that parasitic infection is a common problem in the study area. The relatively high prevalence of helminths in the gastrointestinal tracts of the birds might be due to continuous ingestion of infested droppings or infested intermediate hosts of organisms such as beetles, cockroaches, earthworm, flies and grasshoppers as reported by previous workers (Adang *et al*., 2008; Eslami *et al*., 2009; Radfar *et al*., 2012). The finding that the pigeons were infected with different species of helminths is not surprising. This is in view of the fact that multiple helminthosis was reported by Abdu *et al*. (2004) and Adang *et al*.(2008), to be common in mixed species of poultry that are kept under extensively and semi-intensively managed facility.

Raillietina species appeared to be one of the most common helminth species in pigeons in the study area, as shown by high prevalence rates in comparison to the other helminths species recovered, especially in household pigeons. Even though, the prevalence rates were lower in comparison to the work of Eshetu *et al* (2001), who reported prevalence rates for *Raillietinaechinobothrida* and *Raillietina tetragona* as 28.26 % and 26.08 %, respectively, in domestic pigeons in Ethiopia, this finding might have far-reaching implications because it may impact negatively on the productivity of pigeons in the study area. The high prevalence of *Raillietina* spp. could be attributed to possible abundance and accessibility of intermediate hosts (dung beetles, ants) to the pigeons as was reported by Dede and Richards, (1998) andRadfar *et al*., 2012). Dung beetles and ants may be very common in the study area. Although cestodes in poultry are known to cause retarded growth, enteritis, diarrhea, haemorrhages and hypovitaminosis B (Eslami *et al*., 2009), heavy infections with *Railiietina* species may also be

associated with mortality in young birds and loss of egg production in laying poultry (Mwale and Mosika, 2011). Furthermore, *Raillietina tetragona* is considered as an infectious agent in chickens (Soulsby, 1982).

It is also not surprising to observe that the highest prevalence rate for all helminths species recovered were in pigeons in households in comparison to LBM and commercial farms. This may be attributed to the type of management system in the study area, as birds of different species are kept and fed together in close contact. The possibility of pigeons picking up faecal droppings from a helminth-infested bird could be high in such situation as explained by Nnadi and George (2010) and Mwale and Masika (2011).

The study on Risk factors indicated that LBM may likely contribute to the spread of ND and GIT helminths in pigeons. Earlier reports indicated that marketing systems of poultry may play a considerable role in the dissemination of diseases over wide geographical areas in a relatively short period of time as reported by Shewantasew *et al*(2012). It is also important to note the role of poultry LBM in spread of disease is a major concern to expert in biosecurity in poultry.

Risk practices such as Mixing of pigeons with other species of bird, feeding of pigeons together with other species of bird and the presence of free range chickens or ducks within the same premises might also significantly contributed to the spread of ND and GIT helminthes to pigeons. The mixing of pigeons and other poultry species as indicated by most of the farmers in an attempt to diversify their production by keeping ducks, turkeys, and guinea fowls is a common practice in Kano. The implication is that, ducks and chickens which potentially act as

“reservoirs” for ND viruses and GIT helminthes, respectively, may spread the infection to pigeons, thereby facilitating the development of a possible outbreak. Infected ducks may exhibit no clinical signs, yet can excrete high concentrations of ND viruses that are pathogenic to other poultry species as was reported by previous workers (Echeonwu *et al*.,1993). It has been reported that close contact of pigeons with free range chickens, and aquatic birds, as well as neighboring poultry flocks, may pose a risk of transmission of ND virus (Alexander, 2000).

The presence of dam/pond and untreated surface water were shown to be significant risk factors likely to contribute to the occurrence of ND in pigeons. The roles of untreated surface water, which is mostly contaminated by faeces of both infected and non infected birds in thetransmission of diseases from infected to an uninfected, have been reported (Radfar *et al.*, 2012). The possible risks could be; run-off from infected premises that may enter either surface or ground water and might travel in ditches downstream to another uninfected premises, possibly carrying along ND viruses in washed-off manure that was spread onto agricultural fields. It is important to note that Radfar *et al*., (2012)suggested that Pigeons tend to congregate in large numbers around water bodies. As a consequence, they may drink water contaminated with ND viruses or helminth eggs.

It is important to note that vaccination of domestic pigeons against ND in the study area is not a common practice among pigeon owners.The observation that ND is more likely to spread in pigeon flocks that are not routinely vaccinated against ND have highlighted the importance of vaccination as a preventive measure. It was reported by Tsai and Lee (2006) that vaccinating pigeons with Newcastle disease vaccine may reduce the impact of infection, and may provide a

useful adjunct to biosecurity in minimizing the potential damage that will be caused by that disease.

On the other hand, unvaccinated pigeons may shed ND viruses if naturally infected (Alexander, 2000). The implication is that the ND virus may be spread. Secondly, the carrier status of the pigeons could be of importance considering the fact that pigeons fly long distances and are capable of scavenging around the environment spreading ND viruses to vaccinated and unvaccinated healthy exotic birds (Abraham-Oyiguh*et al.,* 2014).

It is expected that pigeons that are not routinely dewormed may likely suffer from helminthosis. However, there is also the possibility that these pigeons can be reservoirs for a number of parasitic infections and transmit their parasites to other birds. This may pose health and economic problems as well as sources of infection for commercial poultry, wild birds and man (Eslami *et al.*,2009).

CHAPTER SIX

CONCLUSION AND RECOMMENDATIONS

6.1 Conclusions

From the findings of this study, it can be concluded that:

i. The overall seroprevalence of ND antibodies in pigeons in the study area was low (28.1%), with prevalence of 26.2%, 16.5% and 41.5% fordomestic pigeons in households, LBMs and commercial farms accounting for 26.2%, 16.5% and 41.5%, respectively.

ii. The overall mean ND antibody titre (2.13 ± 0.65 $\log_2$) determined for domestic pigeons in Kano Metropolis were below minimum protective antibody level (4.0 $\log_2$) recommended by the OIE.

iii. The overall prevalence rate of GIT helminths in the domestic pigeons is considerably high (46%).

iv. The domestic pigeons were predominantly infested with *Raillietina tetragona* (12.9%), *Raillietina echinibothrida* (13.2%), *Railliettina cesticillus* (7.4%), *Ascaridia columbae* (6.2%) and *Ascaridia galli* (6.3%).

v. Domestic pigeons in Sharada and Tarauni LBMs may play major roles in the spread of GIT helminthes to other birds because the highest prevalence for all the species of helminths recovered from pigoens were observed in the two LBMs with respective prevalence of 11.2% and 10.7%.

vi. This study revealed that 12 (63.2%) of the 19 risk factors assessed for ND and GIT helminths were significant.

vii. The most significant risk factors which were more likely to contribute to the occurrence and spread of ND and GIT helminths in domesticpigeons were allowing pigeons to mix with other birds species, and allowing pigeons access to poultry house.

6.2 Recommendations

From this work itwas recommended that,

i. Since low prevalence rate of ND antibodies was observed, it is highly recommended that vaccination of domesticpigeons against ND, which is not a common practice, should be introduced and sustained in the study area.

ii. Vaccination programme against ND specifically for pigeons should be designed through research on antibody decay.

iii. There should be routine deworming of domestic pigeons and other birds species kept together in close contact or proximity.

iv. Further studies should be conducted to isolate and characterize the ND virus affecting domestic pigeons in the study area.

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APPENDIX I

Questionnairre for Evaluation of Risk Factors for Occurrence and Spread of Newcastle Diseases and Gastrointestinal Parasites in Pigeons in Kano Metropolis

Date:……

1. Code:	
2. Location/Village:	3. GPS coordinates:
4. Local Government Area:	

5. Flock size	
6. Type	
7. Breed	

Risk type	**Yes**	**No**	**Observation**
8. Source pigeons from live bird markets			
9. Source pigeons from Intermediaries (Distributors)			
10. Source pigeons from hatcheries			
11. Pigeons mixed with other bird species in the same compartment			
12. Pigeons fed together with other bird species			
13. High Poultry farm density in the area			
14. Presence of a backyard poultry within premise			
15. Presence of a Dam or pond (open water reservoir) within Premises or in close proximity.			
16. Premises in good maintenance (hygiene)?			
17. Regular cleaning of pigeon house/cage			
18. Regular disinfection of pigeon house/cage			
19. Presence of trees on site which are used for wild birds roost?			
20. Pigeons access poultry house			
21. Free range chickens or ducks within farm premise			
22. Presence of pets (owls, parrots, doves)			
23. Several pigeon flocks of different ages kept together			
24. Presence of untreated surface water (dam, lake, river, pond, gutter)			

25. Presence of cockroaches/dung beetles around pigeon house or manure			
26. Pigeon owner works in poultry farm			

Risk type	**Yes**	**No**	**Comments**
27. Well			
28. Tap			
29. Bore hole			
30. River/Stream			
31. Pigeon house accessible to rodents and wild birds			
32. No specific location for dead birds disposal by Several poultry farms			
33. Poultry manure piled within premises			
Vaccinations/Medications	**Yes**	**No**	**Comments**
34. Newcastle disease			
35. Avian influenza			
36. Deworm pigeons routinely			