ASSESSMENT OF HEALTH PROBLEMS IN INDIGENOUS CHICKEN FLOCKS OWNED BY LOCAL WOMEN IN ZARIA, KADUNA STATE

BY

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DECLARATION

The work presented in this thesis is original and was carried out by me in the Department of Veterinary Public Health and Preventive Medicine, Ahmadu Bello University, Zaria, under the supervision of Professor J.U. Umoh, Dr. I. Ajogi, and Dr. P.O. Okaiyeto.

Reference made to the work of other Investigators is duly acknowledged. No part of this thesis has previously been submitted for a degree or diploma.

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CERTIFICATION

This thesis title "ASSESSMENT OF HEALTH PROBLEMS IN INDIGENOUS CHICKEN FLOCKS OWNED BY LOCAL WOMEN IN ZARIA" by WASH, YOP CHOJI GYANG meets the regulations governing the award of the Degree of Masters of Science of Ahmadu Bello University, Zaria and is approved for its Scientific contribution to knowledge and literacy presentation.

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DEDICATION

This work is dedicated to my dad Joshua Choji Gyang Wash (Tela) praying and hoping that he will come to the fullness/conformation of the measure of Christ and mother Marya Choji Gyang Wash, her influence in my life is a priceless and endless love and blessing.

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I want to thank Ahmadu Bello University for the sponsorship.

Above all, I dedicate my whole service, life, household to the Almighty God for being faithful. It has been this Jesus Christ that had started the work in my life and has brought everything to a complete end I will truely serve you Jesus Christ until Christ comes back again.

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ABSTRACT

A survey was carried out to assess the health status of indigenous chickens kept by rural women around Zaria. Women in households in 10 villages around Zaria were interviewed to obtain demographic data, information on chicken ownership, health status of the chicken and utilization of veterinary services by the women.

Of the 504 women interviewed, 65.7% owned local chicken. There was a statistically significant variation ($P< 0.05$) in the distribution of indigenous chickens among villages, with Zango having 98% women owning chickens and Giwa 19.6%. There were statistically significant relationships ($P< 0.05$) between ethnicity, marital status or religion and ownership status. Determinants of ownership of local fowls are trading (79.6%), non attendance of secondary school(78.5%); long experience of the owners in local poultry management (58.7%) and ownership of other domestic livestock such as goats (94.4%).

Level of significance ($P< 0.05$) between owners and non owners of local fowls were obtained for married women, and educational status (those not educated were more likely to have chicken). Only 17 of the 504 women were solely house wives, others were involved in some kind of occupation (trading, Islamic teaching, etc.). Of the 226 women involved in trading 79.6% owned local chicken. Majority (94.4%) of the women that owned local chicken also owned goats.

The birds were either purchased or obtained as gifts from relations and/or friends. The flock size ranged from 1 to 100 with a mean of 11.25+16.99 (+SD). The ages of birds range from below 1 to 156 weeks. Most (90%) of the owners kept hens. About 62.3% of the respondent reported that they housed the birds only at night.

There were no uniform feeding regimens. However, majority of the owners provided feed for the chicken only in the morning. None of the owners provided water for the chicken. The birds were reported to drink water from any available source around the house.

Out of the 331 women that owned local fowls, 56.3% raised birds for both sale and consumption purposes, while 26.5% for sale only and 17.5% for consumption only. Birds lost during the period 1 to all the flock with a mean 10.42. Reported mortality ranged from 1 to all the birds in the flock.

At the time of the survey, 104 (31.4%) of the owners reported health problems in their flocks. Only 19 (5.7%) of the owners reported ever vaccinating their birds. The physical examination of the sick birds revealed ectoparasites such as lice, mites, and fleas. From the clinical signs during the survey, there were suspected cases of Newcastle disease (NCD), poisoning killed (100%) of the flocks affected and fowl pox (100%)of the affected flock during harmattan period. Intestinal parasites seen were *Coccidia* sp (6.9%), *Acaridia* sp (12.1%), *Gongylonema* sp (0.5%), *Cyathostoma* sp (1%) *Hymenolepis* sp

(0.5%), and *Heterakis* (0.5%) Haemoparasites identified were *Leucocytozoon* sp (0.5%), *Plasmodium* sp (25.7%), *Aegyptianella* sp (8.3%), and mixed infections of *Leucocytozoon* and *Plasmodium* sp (0.5%), *Plasmodium* sp and *Borrelia anserina* (0.5%). About 70% of the mortalities in local birds were due to IBD and NCD, while 60% of mortalities in the young chicks were due to fowl pox.

Samples were screened for antibodies to infectious bursal disease (IBD) virus by agar gel precipitation and Newcastle disease by haemagglutination inhibition tests. The result indicates that 53.3% and 47% were positive for IBD and NCD viruses respectively.

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CHAPTER ONE

INTRODUCTION

In Nigeria, the poultry industry is an important component of agricultural sector of the economy comprising mainly of about 124 million local and 10 million exotic chickens and 1 million guinea fowls and constitutes the largest source of animal protein in the country (Abdu and Saidu, 1995).

About 70% of Nigerians live in rural areas. Rural household as in other parts of Africa, invariably keep indigenous poultry. The range of local flock size is 14-23 and rarely exceed 60 birds (Abdu and Saidu, 1995)

The indigenous birds are called different names at different places. In the United States of America (USA) and the United Kingdom (UK) the indigenous poultry is termed "cage" and "aviary" birds or "game" birds. These game birds are closer to the natural environment (Petrak, 1969; Arnall and Keymes, 1975; Aini *et al.*, 1990).

Poultry contributes protein to the economy. In India, the native layers contributed protein 3% in 1961, 18% in 1971, 58% in 1983 and 67% in the 1986 (Singh, 1990). But there has been 272.2% increase in the population of the native layers while there has been a drop in egg production to minus 42.9% the same year (Singh, 1990). In Nigeria, there has been about 70% of the local birds contributing protein to the economy (Abdu and Saidu, 1995).

Local chickens are believed to be uncared for and unvaccinated which make them susceptible to diseases. Some local birds are carriers of Newcastle disease (NCD) virus, and they are sources of infection to the exotic breeds (Saidu *et al.*, 1994). The free range system of management is responsible for low worm burden and pathogenicity (Saidu *et al.*, 1994). The local chicken is believed to be resistant to common diseases of poultry in our environment but may be carriers of certain diseases of exotic breeds (Agbede, 1981).

The commonest types of diseases in local chickens include viral, bacterial, and fungal diseases, and endo-, ecto- and haemoparasitisms, (Abdu and Saidu, 1995). Diseases have been the commonest and important cause of death, low production and high losses in the local birds (Agbede, 1981; Saidu *et al.*, 1994). Diseases cause poor brooding of hatchable eggs of local hen, and high mortality and high morbidity in local chickens.

Poor housing, nutrition and management, predators, lack of knowledge on modern poultry production, inability of farmers to invest labour and money into production, the presence of unorganised marketing system, and diseases are constraints to local chicken production in Nigeria (Abdu and Saidu, 1995).

Local poultry contribute immensely to the socio economic well being of rural communities in Nigeria but diseases has been a major hindrance to production of indigenous fowls within the rural community (Ibrahim *et al.*; 1992; Saidu *et al.*, 1994). Although modern technologies for the control of poultry diseases exist, they are not readily available in the

rural areas. For long the rural women have shown non-challant attitude towards investment in poultry production (Abdu and Saidu, 1995). The local women use medicinal herbs ("Mamulu", "Madaci", "gautan kaji", "pepper" and different unidentified powder) to manage the local poultry diseases (Nwude and Ibrahim, 1980).

In Ethiopia, indigenous fowls have direct consequences on a particular community which the fowls serve as a source of protein (Tegene, 1993). In Nigeria, also, fowls have direct consequences on the rural communities (Ibrahim and Abdu, 1992; Gadzama and Srivastava, 1986). Some of these consequences are (i) Diseases of local birds causing economic loss (Agbede, 1981). (ii) High mortality rates due to acute highly infectious and contagious diseases resulting in reduction of protein production.

Diseases being the constraint to production in these birds are an issue of concern (Agbede, 1981; Agbede, 1985). Because of the free ranging and unconfined types of management of local birds diseases in local birds are difficult to control. Control measures for diseases of local chickens are rarely practised by the villagers (Abdu and Saidu, 1995; Aini *et al.*, 1990).

Information on reportable diseases of local poultry in the country is based on postmortem examination of sick and dead chickens *(Saidu et al; 1994)*. It is important to check the diseases of local chicken so as to be able to improve their health status and thus the standard of living of the rural women who owned these fowls.

1.1 AIMS AND OBJECTIVES

The aims and objectives of this study are to.-

1.1.1 Survey women owning indigenous fowls, their attitude and limitations towards poultry keeping in villages around Zaria.

1.1.2 Obtain information on management of indigenous birds and associated factors of diseases.

1.1.3 Infer on the socio-economic contributions of indigenous poultry production to rural households.

1.1.4 Obtain a baseline information on the diseases and causative agents of the diseases in flocks of indigenous chickens.

1.1.5 Emphasize on the public health significance of the diseases to the owners.

1.1.6 Advise on prevention and control of diseases in the indigenous birds.

CHAPTER TWO

2.0 LITERATURE REVIEW

Rural women are the major owners of local fowls (Akinwumi *et al.*, 1979; Abdu and Saidu, 1995). Over 80% of the local poultry population in Nigeria is on free range. They occur in almost every village and town and are traditionally uncared for and unvaccinated (Akinwumi *et al.*, 1979).

Local poultry is the most numerous type (Olabode *et al.*, 1992) contributing immensely to the socio-economic well being of local Nigerian (Ibrahim and Abdu, 1992).

Diseases have been the commonest causes of sickness and death of local chickens (Abdu *et al.*, 1984). The commonest causes of sickness and death of local chickens are Newcastle disease (NCD), Gumboro (GD) and Fowl pox (FP), which are responsible for 76 percent (%) of all the mortalities recorded in local fowls (Abdu *et al.*, 1992, Abdu and Saidu, 1995; Agbede, 1985). A good number, 57% of these local fowls have been shown to be infested by helminths or ectoparasites (Abdu *et al.*, 1992, Abdu and Saidu, 1995; Agbede, 1985). Young birds accounted for 90% of all mortalities recorded in local chickens (Abdu *et al.*, 1984).

Viral diseases are responsible for 76% of all the mortalities recorded in local chickens. Birds that died of NCD, IBD, FP, were also infested by helminths or ectoparasites (Saidu *et al.*, 1994). Collibacilosis, pullorum disease, fowl typoid and

coccidiosis occurred only occasionaly (Saidu *et al.*, 1994). About 60% of the fowl pox outbreaks occurred in chickens (Saidu *et al.*, 1994).

There are also nutritional and metabolic disorders found in the indigenous poultry (Szubartowska and Grounysz-Kalwovska, 1992). Diseases such as ectoparasites and endoparasites and predators are presumed to be responsible for poor production and loss of chickens (Tegene, 1993). Generally these birds are subceptible to many diseases (Abdu *et al.*, 1985, Ezeokoli *et al.*, 1984). Thus actual losses due to these causes must be justified and feasible control must be instituted to rectify the losses (Tegene, 1993).

Diseases such as ecto- and endoparasites and predators are presumed to be responsible for poor production and loss of chickens (Tegene, 1993).

Some diseases of poultry show abnormal variation. There were three peaks of outbreaks of endoparasitism (January, June and December), and five peaks of NCD outbreaks (January, February, March, April, November) and the highest number of outbreaks were recorded in December (Saidu *et al.*, 1994; Abdu and Saidu, 1995). The third peak was recorded during the end of the rainy season during the months of August, September and October (Saidu *et al.*, 1994; Abdu and Saidu, 1995). Ectoparasitism is more prevalent in the rainy season (April-September) (Saidu *et al.*, 1994).

2.1 DISEASES AND PARASITES OF LOCAL BIRDS

2.1.1 ENDOPARASITISM

Several species of nematodes, cestodes and trematodes have been identified in local chickens (Agbede, 1981). The nematodes are *Syngamus trachea*, *Capilleria species*, *Gongylonema* species, *Dispharynx spiralis*, *Tetrameres* species, *Ascaridia* species, *Cheolospirura hamulosa*, *Ascaridia* species, *Heterakis* species, *Subulura* species, and *Strongyloides avium.* The trematodes are *Amoebotaenia* species, *Choanotaenia infundibulum*, *Davainea proglottina*, *Hymenolopsis* species, *Raillietina* species and *Prostharmostomum* species (Abdu and Saidu, 1995). The commonest helminths of local chickens are, however, *Syngamus trachea*. While the cestodes are *Tetrameres* species, and *Ascaridia galli* (Adene and Fatumbi, 1979; Agbede, 1981). Over one-third of chickens are infected with helminths (Agbede, 1981).

2.1.2 ECTOPARASITISM

Various species of ectoparasites have been found in local chickens (Agbede, 1981, 1985; Abdu and Saidu, 1995). The lice found include *Menacantus* species, Menopon *gallinae*, *Lipeureus caponis*, *Amyrsidea powelli*, *Gonoides gigas*, and *Numidilipeirius tropicalis* (Agbede, 1981, George *et al.*, 1990; Abdu and Saidu, 1995). The mites normally found are *Bdeltonomyssus bursa*, *Dermanyssus gallinae*, *Meganinia cubutales*, *Cnemidocoptes* species, *Syringophilis* species, and *Gytolochus nudus* (Agbede, 1981; George *et al.*, 1990). Ticks, fleas and lice are Argus persicus, *Haemaphysalis hoodi hoodi*,

Amblyomma variegatum, *Hyalomma* species, *Echinostophaga gallinacea*, and *Pseudolynchea canariensis* (Agbede, 1981; George *et al.*, 1990, Abdu and Saidu, 1995). The incidence of helminthiasis and ectoparasitism are highest in young birds (Saidu *et al.*, 1994).

2.1.3 HAEMOPARASITISM

Plasmodium gallinaceum, *Aegyptianella pullorum*, *Leucocytozoon* species and *Borrelia anserina* are the blood parasites of local birds (Abdu *et al.*, 1995).

2.1.4 BACTERIA, FUNGI AND DISEASES OF OTHER ORIGINS

Some important bacteria and fungi of poultry have been isolated from local chickens (Bagaiwa, 1986; Adekeye *et al.*, 1989; Abdullahi *et al.*, 1992). The bacteria are *Staphyloccus aureus*, *Streptococcus faecalis*, *Salmonella gallinarum*, *Camphylobacter fetus* sub-species *jejuni*. The fungi include *Mycoplasma gallisepticum*, *Mycoplasma synoviae*, *Trichophyton gallinae*, *Aspergillus fumigatus* and *Candida albicans* (Abdullahi *et al.*, 1992).

Other conditions encountered in the local chickens are omphalitis, bumble foot, subcutaneous emphysema, chemical poisoning, prolapsed oviduct, and rectum, fracture of the appendicular skeleton, and snake bites (Saidu *et al.*, 1995). Snake bites occur more frequently in the rainy season and hens sitting on eggs or those with chicks are more

vulnerable to attack by snakes (Saidu *et al.*, 1995).

2.1.5 VIRAL DISEASES

Viral diseases have been the outstanding cause of death in local poultry. These diseases are NCD, IBD and FP. Seventy percent (70%) of mortalities are caused by NCD, IBD, FP. Mortality was high in young chickens *(Saidu et al 1994)*. About 60% of the fowl pox outbreaks occured in chicks (*Saidu et al, 1994*)

Viral diseases have been recorded as the most important diseases of local fowls, young birds being more susceptible (Saidu *et al.*, 1994). There appeared to be seasonality in the prevalence of these diseases (Tegene, 1993). Concurrent infections, age and season are also important factor associated with the frequency of viral diseases (Saidu *et al.*, 1994).

The owners and local birds are susceptible to Newcastle disease (NCD) and the local chickens to Infectious bursal disease (IBD).

2.1.5.1 EPIDEMIOLOGY OF NEWCASTLE DISEASE

NCD has epizootiologic as well as pathologic significance (Upton *et al.*, 1953; Jordan, 1990). It is an infectious highly contagious and destructive malady. NCD attacks chiefly chickens and turkeys (Jordan, 1990) and the disease causes death of chickens irrespective of their ages (Hanson and Brandly, 1955). Various other birds and

occasionally human beings may also be infected with the virus (Hanson, 1964). The epizootology of Newcastle disease, particularly the pigeon associated epizootics, which appeared throughout the world (Doyle, 1927; Lancaster, 1966; Beaudette and Black, 1946). There are four strains of NCD virus isolated on sick birds. In 1927 the first strians isolated was called Doyle strain of Newcastle disease virus strain, causing a disease called the Doyle form of Newcastle disease (Doyle, 1927). The second strain isolated caused fever in chickens and respiratory signs and is called the Beach strain (Beach, 1942; Beach, 1946). The third isolate caused distinct signs of central nervous system disorders due to Beaudette strain of Newcastle disease virus. The central nervous system disorder is called Beaudette form of Newcastle disease (Beaudette and Black,2 1946; Jordan, 1990). The fourth isolate caused incidences of mixed clinical signs (respiratory disorders and neural disorders). The Strain is called the Hitchner Strain and the form of the disease was called Hitchner form or pneumoencephalitis (Hitchner and Johnson, 1948; Hitchner *et al.*, 1950).

The history of NCD is marked by panzootics. The history of NCD was traced to USA (Beaudette and Black, 1946; Doyle, 1927; Beaudette and Black, 1946; Lancaster, 1966). The first study began with the emergence of the disease in fowls in the 1920's during which the virus of the Newcastle was isolated in Queensland and the virulence of the isolates were determined in chicken embryo. All isolates produced diseases irrespective

of the routes of passage (Simmon, 1967; Hanson and Brandly, 1955). In addition the virus produces plaques on chicken embryo fibroblast (CEF) monolayers (Hanson and Brabdly, 1955). Chickens were reported as the hosts (Taylor, 1964).

2.1.5.2 AETIOLOGY OF NCD

NCD is caused by a group of closely related viruses which form the avian paramyxovirus type 1 (Pmv-1), (Bankowski and Costervet, 1960; Jordan, 1990). The recorded size of the ultrastructure was 80-120 µm. NCD virus has affinity for red blood cells (rbc's) (Burnet, 1942), is capable of using RBCS for hemagglutination (Kilman, 1949, Burnet, 1950; Burnet and Lind, 1950), resistant to chemical agents, ultraviolet rays and pH change (Jordan, 1990). The virus is stable to thermal treatment (Hanson, 1949).

Laboratory host system has shown that there are antigenic variations existing between different strains of NCD (Burnet, 1942; Upton *et al.*, 1953; Sinha *et al.*, 1952, 1957). The ability to cause the disease in chicken embryo and laboratory animals were dependent on the virulence of the strain of the virus, dose and route of administration, environmental conditions, temperature, susceptibility of the host (Sinha *et al.*, 1952, 1957). Emergence of serotypes led to the evolvement of strain classification of the virus (Hanson, 1949; Jordan, 1990). The strains are classified as below:

a) Viscerotropic Velogenic NCD virus cause a highly virulent form of a disease in

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which haemorrhagic lesions are characteristically present in the intestinal tract (Doyle, 1927, Jordan, 1990).

b) Neurotropic velogenic NCD virus cause high mortality following respiratory and nervous signs (Beach, 1942, 1946; Beaudette and Black, 1946, Jordan 1990)

c) Lentogenic respiratory NCD virus causes mild or inapparent respiratory infection (Jordan, 1990). All mesogenic and lentogenic strains produce sickness and death irrespective of the routes of infection (Hanson and Brandly, 1955).

d) Mesogenic NCD virus causes respiratory and sometimes nervous signs with low mortality (Beaudette and Black, 1946, Jordan, 1990).

e) Asymptomatic enteric NCD virus causes inapparent enteric infection (Jordan, 1990). There is always some degree of overlap and some virus strains are not easily placed in a specific pathotype (Jordan, 1990).

All Strains of NCDV induce infection of chicken embryos which lead to death but lentogenic strains do not cause death in embryos when antibodies are present (Hanson and Brandly, 1955; Report of Poultry Diseases Subcommittee, 1963).

Over 200 species of birds have been reported to be susceptible to the different pathotypes of NCDV in natural and experimental infections (Jordan, 1990). Chickens have been reported as the natural host (Hanson *et al.*, 1952) but other domestic fowls may serve as carriers (Gale, 1964).

2.1.5.3 TRANSMISSION AND CARRIERS OF NCD

The disease is transmitted via the aerosol (Sinha *et al.*, 1954). Birds showing the respiratory disease presumably shed virus in mucus which may be inhaled by susceptible birds (Jordan, 1990).

Viruses that are mainly restricted to intestinal replication may be transmitted by ingestion of contaminated faeces, either directly or indirectly by eating contaminated food or dried faeces (Hanson, 1967; Jordan, 1990). Susceptible birds can contact the disease from infected birds that the chicken handlers placed in poorly environmental situation (Sinha *et al.*, 1954, Jordan, 1990).

2.1.5.4 CLINICAL SIGNS AND LESIONS OF NCD

The Doyle form of the Newcastle disease produces clinical signs without causing death of birds (Iyer, 1943). Nervous system were recorded by Resang (1961). All the strains have high morbidity (100%) (Jordan, 1990). The highly virulent virus may produce peracute infections in fully susceptible chickens which the first indications of a disease being sudden death (Jordan, 1990). The mortality is usually 90% (Dobson, 1939). The clinical signs of depression, prostration, diarrhea and oedema. Appearance of shell-less or soft shelled eggs are typical of the disease followed by complete cessation of egg laying and marks an early sign in the adult chicken (Iyer, 1943, Bird, 1964, Jordan, 1990).

The mesogenic viruses cause severe respiratory disease, followed by nervous

signs, with mortality up to 50% or more (Resang, 1961; Report of Poultry Diseases Subcommittee, 1963; Jordan, 1990). The viruses of low virulence may cause mild respiratory distress for short time in chickens (Hanson *et al.*, 1952). No gross lesions are pathognomonic of NCD (Iyer, 1943; Jordan, 1990). Viruses causing respiratory diseases induced inflammation of the trachea (Beard *et al.*, 1967) often with hemorrhages present (Jordan, 1990). The air sacs may also be inflammed and appear cloudy and congested (Beard and Easterday, 1976; Jordan, 1990). Damages of the respiratory centres have been reported (Beard and Easterday, 1967).

The virulent viscerotropic strains, usually cause hemorrhagic lesions of the intestinal tract particularly the proventriculus which vary in size and severity (Jordan, 1990). The highly virulent viruses caused hemolysis of red blood cells (Kilman, 1949; Burnet, 1950; Burnet and Lind, 1950). Histopathological lesions observed in diseased (NCD) chickens were hyperemia, edema, and hemorrhages (Beard and Easterday, 1967). When the respiratory tract is involved, inflammation, cellular infiltration and hemorrhages of the trachea may be seen; sometimes with proliferation and exudative lung lesions (Upton *et al.*, 1953). In milder forms, lymphocyte infiltration in the air sac membrane, lungs, and trachea may be seen (Auer, 1952; Jordan, 1990). Examination of the central nervous system (CNS) has revealed encephalitis, general infiltration of nervous system by lymphocytes (Auer, 1952; Jordan 1990).

There were damages of uterus, and oviduct, and atresia of follicles (Bisural and Morill, 1954). Early maturity, increased egg production and loss of broodiness were reported in some infected birds (Bird, 1964). Other systems were affected such as the ear (internal ear exudation) (Upton *et al.*, 1953) and infiltration of the eyes (Upton *et al.*, 1953).

2.1.5.5 DIAGNOSIS OF NCD

There are several ways of diagnosing Newscatle disease. Clinical signs of disease are not pathognomic (Jordan, 1990). The diognostic methods are:

(a) Isolation and characterization, and

(b) serology are frequently used for diagnosis (Jordan, 1990).

Several serological methods have been used (Report of Poultry Diseases Subcommittee, 1963; Jordan 1990), enzyme-linked immunosorbent assay (ELISA; Jordan, 1990), and agglutination (Burnet, 1942). The most widely used of the serological methods is hemagglutination (HA) (Jordan, 1990).

2.1.5.6 PREVENTION AND CONTROL OF NCD

There are legislations as a tool for control and vaccination programmes applied in countries that import and export the indigenous birds (Jordan, 1990). In panzootic areas of the western hemisphere and with the transportation of exotic birds, quarantine is imposed followed by elimination of birds infected with Newcastle disease (Jordan, 1990).

Restrictions and banning of people (races) from specific countries and compulsory vaccination is imposed (Jordan, 1990). Many countries have slaughter policy for birds with NCD. Restrictions can be imposed by law, ring vaccination may be obligatory following an outbreak. In some countries prophylactic vaccination of poultry is required by law (Jordan 1990).

In control programmes effective diagnosis, monitoring and enforcement of control measures are important (Jordan, 1990). Basically three vaccines are available for NCD: The live lentogenic (Hitchner *et al.*, 1950, Jordan, 1990), the live mesogenic (Hitchner *et al.*, 1950) and the inactivated (Lancaster, 1966; Jordan, 1990).

Good management systems, and hygiene assist in disease control. There should be security to man domestic fowls under field conditions [Jordan, 1990]. Vaccinations only is insufficient to bring about effective control of Newcastle disease,and that it must be *accompanied by good hygiene. Good hygiene complemented by good management* is therefore of critical importance at all times and not merely during an epizootic of the disease (Jordan, 1990). He further suggested that depopulation of all poultry and products, including faeces, should be strictly adhered to. Burial or incineration after mass slaughter and a thorough, disinfection, preferably fumigation of premises are also recommended (Jordan, 1990; Saidu *et al.*, 1993).

2.1.5.7 EPIDEMIOLOGY OF INFECTIOUS BURSAL DISEASE (IBD)

After the first reported outbreak of IBD in the United States of America (Cosgrove, 1962) the disease has now been reported in virtually all countries of the world (Winterfield and Hitcher, 1962; Winterfield, 1969). The disease has been recognised in Nigeria since 1969 . It was first reported from the Western States in 1973 (Nawathe et al, 1978) and further outbreaks were described.

2.1.5.8 DISTRIBUTION AND INCIDENCE OF IBD

It is said to be more prevalent in commercial towns (Okeke, 1984) and appears with equal frequency throughout the year (Cosgrove, 1962). It reoccurs several times on many farms but with a decrease in the mortality rate during subsequent outbreaks (Cosgrove, 1962; Meroz 1966). The prevalence of clinical IBD appears to be low, but serological evidence indicates that subclinical infection is widespread (Faragher, 1972). There is serological evidence of high IBD prevalence in free range chickens in which clinical disease is rarely observed (Nawathe *et al.*, 1978 Umoh *et al.*, 1982; Abdu *et al.*, 1984, 1986).

2.1.5.9 AETIOLOGY OF IBD

a) Infectious bursal disease virus (IBDV) has been classified as a Birnavirus (Jordan, 1990). It replicates in the cytoplasm of infected cells and four structural units are present

along the edges of some viral particles (Kaufer and Weiss, 1976; McFerran *et al.*, 1980). It also differs from reoviruses in that its virion has a single instead of a double capsid and the RNA is single stranded (Kaufer and Weiss, 1976; Nick *et al.*, 1976; McFerran *et al.*, 1980; Jordan; 1990). The virion is non-enveloped, icosahedrally shaped with diameter between 55 and 65 mm (Jordan, 1990). The capsomers are also smaller than those of reoviruses and have been observed to be more than 32 in number (Cho and McDonald, 1980).

The buoyant densities of IBDV as determined in continuous sucrose gradient was found to be 1.178 g/cm^3 (Cho and McDonald, 1980) and 1.33g/ml by rate-zonal and density-equilibrium method (Fahey *et al.*, 1985).

There are two populations of morphologically - different but antigenically-related virions with mean diameters of 20mm and 60mm respectively have been detected in virus preparations (Almeida and Morris, 1973; Pattison *et al.*, 1975). Two major polypeptides with approximate molecular weights of 32,000 and 37,000 have also been found to be present in virus preparations (Fahey *et al.*, 1985). IBDV was found to be resistant to heat and ultraviolet irradiation (Benton *et al.*, 1967; Peter *et al.*, 1973; Cernik, 1983, Abdu et al; 1986; Jordan, 1990). The virus has also been found to be infective for chickens after exposure at 60°C for 90 minutes, at 25°C for 21 days and after storage for 3 years at -20°C (Yungcho, 1967). It can remain infectious in the environment for at least 4 months and it

can survive temperature of 60°C (but not 70°C). It was found to be resistant to ether, chlorofom, merthiolate, trypsin, phenol, and hyamine (Benton *et al.*, 1967a, 1967b). The virus has been reported to be acid and alkaline stable surviving at a range of 2 to 13 (Benton *et al.*, 1967a). It is also stable at pH2 but not at PH4 12 and is resistant to organic solvents (Jordan, 1990).

2.1.5.10 TRANSMISSION OF IBD

Infectious bursar disease is contagious (Benton *et al.*, 1967a) and also another scientist found IBDV as highly contagious (Jordan, 1990). They found houses that had housed infected birds were infective for other birds 122 day after removal of the infected birds and that water, feed and droppings taken from infected pens remain infectious for 52 weeks. A ground suspension of lesser meal worm, *Alphibotus disperinus*, taken from a house eight weeks after an outbreak of IBD, was successfully used to infect susceptible chickens (Snedeker *et al.*, 1967). Mites may play an important role in the transmission of IBDV (Brady, 1970; Jordan, 1990). A strain of IBDV isolated from mosquitoes have been identified (Howie and Thorsen, 1981). Experimental infections can be achieved by oral, intramuscular, intrabursal and subconjunctival routes (Abdu *et al.*, 1986). There is no evidence of older birds acting as carriers (Jordan, 1990).

2.1.5.11 SUSCEPTIBILITY

Natural infections with IBDV have been reported mainly in fowls (Faragher, 1972) and more rarely in turkeys (McNulty *et al.*, 1979; Sivanandan *et al.*, 1984; Chettle et al, 1985). However, village weavers (*Ploceus cucullatus*) have been found to be positive for IBDV antibody (Nawathe *et al.*, 1978).

IBD has been reported in nine-day to 20 week-old chicken (Cosgrove, 1962; Hanson, 1967; Luthgen, 1969, Onunkwo, 1975; Okoye and Uzoukwu, 1981; Abdu *et al.*, 1986). All breed of fowl may be infected but light breeds show more secure reaction to the virus than heavy breeds (Hitchner, 1978, Jordan, 1990). However, no significant difference in mortality rate between the breeds was found (Meroz, 1966).

2.1.5.12 DIAGNOSIS OF IBD

The history, clinical signs and gross lesions are adequate for recognition of the acute disease (Jordan, 1990). The signs observed included diarrhoea, soiled vent feathers, severe depression, anorexia, ruffled feathers, droppy appearance, trembling, prostration and death (Cosgrove, 1962; Giambrone *et al.*, 1977). Mortality may be nil but can reach levels of 10 to 30% (Hitchner, 1976; Winterfield, 1969). Morbidity usually begins on the third day post-infection and usually lasts for five to seven days (Cosgrove, 1962; Parkhurst, 1964; Hitchner, 1976).

Confirmatory diagnosis of IBD is based on the histopathological immunity of infected Bursa Fabricius (BF), serology, inoculation of susceptible chicks and viral isolation (*Cosgrove*, 1962; *Hemboldt and Garner*, 1964; *Asrubali and Gilletti*, 1971; *Weisman and* Hitchner, 1978; Cho *et al.*,1979; Ide, 1979; Abdu *et al.*, 1986, Jordan, 1990). Serological methods used to detect flocks exposed to IBDV are the agar gel precipitin test (Hirai *et al.*, 1972), serum neutralization test assayed in fully susceptible embryonated chickens eggs or susceptible chickens (Winterfield, 1969); plaque reduction test in chicken embryo kidney or fibroblast cell monolayers (Lukert and Davis, 1974., Cho *et al.*, 1979), immunofluorescent antibody test (Allan and Grough, 1974), indirect enzyme-linked immunosorbent assay, indirect immunoperoxidase antibody technique (Okeke and Lang, 1981), PCR and rapid counter immunoelectrophoresis (Berg, 1982).

2.1.5.13 CONTROL OF IBD

The highly infectious nature of IBD has made vaccination a necessity in major poultry producing areas throughout the world (Abdu *et al.*, 1986). There are at present live and inactivated IBDV vaccines (Jordan, 1990). The live vaccines are either midly or highly pathogenic Mild vaccines are low in their invasiveness of the BF and may easily be neutralized by Maternal antibodies (MA) levels (Winterfield and Thacker, 1978) while highly pathogenic vaccines are highly invasive and overcome the effect of MA (Fletcher *et al.*, 1978; Haffer, 1982). Chicks devoid of MA when given highly pathogenic vaccines

will be immunosuppressed and are capable of spreading the vaccine virus to susceptible chickens (Muskett *et al.*, 1979). There are two methods of controlling subclinical IBD namely, vaccination of breeders to produce high MA levels in the off-spring to prevent infection early during the first week of life and vaccination of progeny at day-old to prevent early infection (Giambrone et al, 1982).

Since MA declines steadily after hatch and is absent by three or four weeks of age, it cannot be depended on to protect against clinical IBD (Naqi *et al.*, 1983). The type of vaccine to use and the programme to follow will depend on the level and virulence of the field virus. Vaccination with a highly pathogenic IBDV vaccine at five to eight days in drinking water has been recommended (Giambrone, 1982). Day-old vaccination with mild vaccine is usually confined to progeny of parents that are not receiving killed vaccine or parents that are older than 45 weeks and have become seronegative to IBDV by agar-gel precipitin test or below 1:100 on the virus neutralization test (Giambrone, 1982; Abdu *et al.*, 1986).

2.2 CONTROL OF DISEASES IN INDIGENOUS BIRDS, ETHNOVETERINARY KNOWLEDGE AND PRACTICES

Vaccinations should be conducted along with regular deworming and ectoparasite control (Saidu *et al.*, 1994).

Abdu *et al.*, 1992) reported that in the rural Hausa/Fulani flocks disease occur all the year round but most mortalities are recorded when the weather is cold and dry. Several diseases had been given vernacular names as follows: fowl pox (Chubayi, Kisko, Junjeren Kaji), Newcastle disease (Birgirin kaji, mabori, Chiwon bori, Daniniya, Jinnu), coccidiosis (Atini), external parasite such as mites (kuma), Lice (kwarkwatan kaji) and soft ticks (tsutsan chiki), large round worms (Machigin chiki), darrhoea (Farin kashi), Coryza (pupu, mura, mashako) and gasping (Chiyon hamma) (Abdu *et al.*, 1992).

There are ways of reducing losses being adopted by the rural communities (Nwude and Ibrahim, 1980; Abdu *et al.*, 1992). The sick young birds are usually allowed to recover or die naturally to be subsequently thrown into pit. Sick adult birds are either sold cheaply to neighbours or dealers or slaughtered for consumption (Abdu *et al.*, 1992). The offals, feathers and body fluids of such adult sick birds) are buried deep or poured into pit as it is believed that walking across such materials by chicken result in sickness and death (Abdu *et al.*, 1985). In a number of cases, to avoid economic loss during cold dry weather, most of the farmers sell their birds *en-masse* prior to the commencement of the dry season (Abdu *et al.*, 1992).

The method of control is the use of modern drugs such as May and Baker (MB), paracetamol, aspirin, apc, tetracycline, and chloramphenicol for treatment of local birds (Nwude and Ibrahim, 1980). These drugs are used without due consideration to dosage

and duration of administration. Traditionally, fruits, leaves, barks, roots, lattice, juice and oils from plants and oils, and milk of animal origin are used for prevention and treatment of various ailments of chickens (Nwude and Ibrahim, 1980). Akut (1991) reported the use of these plants in the treatments of diseases. (a) Fowl pox (tsurup) treated with the fruit of *Solanum inconium* (Gashio Aboi) the fruit was sliced gently, rubbed topically on raw surfaces exposed after pustules are debrided; (b) Ectoparasites (tsutsa) treated with *Nicotiana tobaccum* ("taba") leaves which have been dried and grounded into powder, liberally dusted on the birds; *Ricinus communis* (Sakan) leaves were used as Nicotiana tobaccum leaves. Ashes (Ntan) obtained from firewood were also used for the treatment of ectoparasites. (c) Coccidiosis (Ashan biyun) was reported to be treated with *Capsicum annum* (shita) fruits, freshly sliced, marshed into paste and administered in drinking water (Akut, 1991). (d) IBD (Tsuwan) treated with *Parkia clappertoniana* bark, grounded and administered in drinking water and (e) NCD (Abuak) was treated with *Solanum inconum* (Grashioaboi) fruit, freshly sliced and placed in drinking water (Akut, 1991).

CHAPTER THREE

3.0 MATERIALS AND METHODS

All materials used are listed in Appendices A to C.

3.1 STUDY AREA

The study area included 10 villages around Zaria in Kaduna State. They have scattered settlements. The villages are Shika, Bomo, Giwa, Zango, Zaria City, Basawa, Palladan, Hayindogo Layin Zomo, and Samaru as shown in the map (Appendix L). The Veterinary Teaching Hospital (Ahmadu Bello University, Zaria) is centrally located in the study area. The village heads were informed and proper arrangements were made before each woman was interviewed.

The women were housewives, traders, farmers, with a few being involved in tailoring and weaving. Some of them kept domestic animals like cattle, goats, sheep, ducks, pigeons and indigenous poultry. The sources of drinking water for 2the households and domestic animals were wells, streams, rivers and taps.

The study area - geographical region has four seasons (a) dry and cool starts in mid-November and ends in February. (b) dry and hot is a short transitional period in between harmattan and wet seasons covering March and April. (c) hot season which starts in May and ends in mid-September and (d) dry and warm season begins at the end of the rains in October and ends in about mid-November (Olorin, 1987).

The local fowls serve as a source of revenue for the rural women who used the revenue to assist in household maintenance.

3.2 METHOD OF SURVEY

Women in the study area were interviewed using a structured questionnaire. Information was obtained from the women on ethnicity, religion, marital status, type of marriage, length of marriage, age last birthday, number of live children owned number of dead children, educational status, membership in women's club or associations, predominant occupation during the dry and rainy seasons, livestock ownership and type of livestock owned other than indigenous fowls.

Information on the management status of the local fowls included sources of birds, flock size, housing, feeding methods, type of feeding and feed supplements, feeding utensils, sources of water and frequency of giving water to the indigenous fowls.

Information on the health and disease conditions of the local fowls obtained included the number of sick birds, number of dead birds, treatment of birds in veterinary clinics, the season the disease occurred, vaccinations administered, number of lost birds, number of killed or sold birds and willingness of owners to vaccinate the birds.

Information was also obtained on losses due to dead fowls, prices of healthy and sick birds, the average revenue generated from the sells of the birds and losses to predators.

3.3 EXAMINATION OF SICK BIRDS IN THE VILLAGES DURING SURVEY

Sick birds seen during the survey were physically examined to identify ectoparasites (fleas, mites etc) and observed clinical signs of any disease.

3.4 MICROSCOPIC EXAMINATION OF FAECES AND INTESTINAL SCRAPINGS OF SLAUGHTERED INDIGENOUS BIRDS AT THE ZARIA ABATTOIR

Faeces and intestinal scrapings were collected from indigenous chickens slaughtered at the Zaria abattoir.

Using mortar and pestle, each sample collected was mashed and poured into centrifuge tubes. The centrifuge tubes were filled with flotation media until a meniscus was formed over the centrifuge tube. A cover slip was placed on each centrifuge tube and allowed to stand for 10 minutes. The cover slip was removed and placed on a labelled glass slide examined under microscope using x 10 eyepiece and X 40 objective lens. The microscopic examination was conducted as described by Baker and Silverton, (1985) .

3.5 IDENTIFICATION OF BLOOD PARASITES IN LOCAL BIRDS SLAUGHTERED AT THE ZARIA ABATTOIR USING THIN SMEAR TEST (GIEMSA STAINING)

The blood samples were collected into sterile bijou bottles and smears were prepared on glass slides and allowed to dry. The smears were fixed with methanol for 3 minutes, and stained with Giemsa stain for 30 minutes. The slides were air dried vertically in a wooden slide block and examined through a light microscope under oil immersion (X 100

objective).

3.6 SCREENING OF SLAUGHTERED INDIGENOUS BIRDS AT THE ZARIA ABATTOIR FOR NEWCASTLE AND INFECTIOUS BURSAL DISEASE

Two hundred samples of blood were collected from slaughtered indigenous birds at the Zaria abattoir in the labelled sterile tubes and allowed to clot at room temperature (25°C) for 6-12 hours.

The serum was obtained after centrigation at 3000 rpm for 3 minutes.

3.7 IDENTIFICATION OF NEWCASTLE DISEASE

The NCD virus (Lasota vaccine) titration (Hemagglutination test (Appendix E) was carried out to identify the working antigen. Hemagglutination inhibition test for Newcastle disease identification.

i. Collection and washing of erythrocytes:

Five millilitres (5mls) of blood was collected each time in ethyleneditetra-acetate (EDTA) solution and centrifuge at 3,000 rpm for 3 minutes to remove plasma. The supernatant was discarded. The red blood cells were washed 3 times in physiological saline solution (Appendix D). A 5% rbc concentration was prepared from the washed rbc as described by (Allan and Grough, 1974).

ii. Dilution of lasota used as antigens

Five millilitres of sterile water was added to the one 1000 dose ampoule of lyophilized lasota vaccine to dissolve these vaccine as described by (Allan and Grough, 1974).

3.7.1 TITRATION OF ANTIGEN (HAEMAGGLUTINATION TEST)

The stock antigen was titrated by hemagglutination test (Allan and Grough, 1974) and 4 hemagglutination unit (HAU) was determined and used for the hemagglutination inhibition tests (Appendix E).

3.7.2 HEMAGGLUTINATION INHIBITION TEST (H I)

This procedure is described by Allan and Grough (1974). Fifty microlitres of physiological saline (PSS) was added from wells 2 to 12 (using a microdiluter into a microtitre plate). Fifty microlitres of the test serum was added into well 1. The same 50ul was added to well 2 and shakened properly to dilute the mixture. Fifty microlitres of the mixture in well 2 was transferred to well 3 and was serially diluted in equal volumes from subsequent wells up to well 11.

Fifty microlitres of the working antigen (Appendix G) was added into wells 1 to 11 each and shakened properly to make a mixture. The mixture at this stage was incubated for 15 to 20 minutes.

Fifty microlitres of the 5% washed rbc was added from wells 1 to 12 and mixed. The mixture was incubated for 30-60 minutes. Readings were taken.

3.8 AGAR GEL PRECIPITATION TEST FOR INFECTIOUS BURSAL DISEASE IN INDIGENOUS BIRDS SLAUGHTERED AT THE ZARIA ABATTOIR

Antibody to IBDV was dictected in the serum samples from the local fowls by agar gel precipitation test according to Hirai *et al.* 1972 (Appendix H).

CHAPTER FOUR

4.0 RESULTS

4.1 OWNERSHIP OF LOCAL POULTRY BY OWNER IN THE VILLAGE

a) RURAL WOMEN IN HOUSEHOLDS

An overall survey of the 504 rural women in households showed that 331 (65.7%) owned local poultry and 173 (34.3%) did not.

b) STUDY AREA

Table 1 shows the distribution of ownership status by location of survey. The villages are Shika, Bomo, Giwa, Zango, Zaria City, Palladan, Hayin dogo, Layin Zomo and Samaru.

c) DISTRIBUTION

The distribution of ownership varied significantly from village to village ($P<0.05$).

4.2 FACTORS AFFECTING OWNERSHIP

The distribution of ownership by ethnicity indicated that most of the owners (82.1%) were hausas, 59.4% Fulani's, 76.4% were other tribes non specified and 35.1% owners did not respond, while non owners were 17.9% Hausa, 40.6% Fulani, 23.6%, others unspecified and 64.9% non owners did not respond.

Religion did not seem to be an important factor in ownership of local chickens as the proportion of muslims and christians that owned chicken were 77% and 74.4%

respectively.

The proportion of married women that owned chicken was (86.3%). Only 16 single women were interviewed and 9 (56.3%) of them owned poultry (Table 2). Of 179 respondents involved in monogamous marriage, 80.5% were owners while of the 216 in polygamous marriage, 73.6% were owners. Table 3 indicates the relationship between length of marriage and ownership of local fowls. 119 of the women interviewed were in the first year of marriage and 79.8% of them own local chicken (Table 3). Length of marriage did not seem to affect ownership of chickens.

Also having a basic primary school education did not seem to affect chicken ownership (Tables 5) as the difference between proportion of owners among those who did not attend even primary school (78.5%) and those who attended (74.1%) was not significant. It was also observed that those who did not have secondary education were slightly more likely to own chicken than those who had secondary school education. Belonging to clubs/ associations did not significantly affect chicken ownership ($P > 0.05$). Majority 326 (65.7%) of women, however, did not belong to any club or association but owned local fowls and 170(34.3%) did not. A few 5(62.25%) that owned fowls were involved in associations.

The types of occupation of the women interviewed showed that most of them were traders. Only 17 of the respondents were full time housewives who also involved

themselves in gardening, farming, weaving, religoud instruction, fishing, and tailoring.

A large proportion (58.7%) of the respondents had up to 156 weeks of experience in poultry keeping. Of these, 229 (77.4%) were owners of local chicken (Table 6).

The most commonly kept livestock by respondents particularly local chicken owners was goat (Table 7).

Table 1: Pattern of distribution of indigenous fowls in ten villages in Zaria and its environs

VILLAGES	WOMEN INTERVIEWED	OWNERS	NON- OWNERS
Shika	49	36 (73.5)	13 (26.5)
Bomo	51	36 (74.6)	15 (29.4)
Giwa	51	10 (19.6)	41 (80.4)
Zango	49	48 (98.0)	1 (2.0)
Zaria city	50	33 (66.0)	17 (34.0)
Basawa	50	40 (80.0)	10 (20.0)
Palladan	51	48 (94.1)	3 (5.9)
Hayin Dogo	51	42 (82.4)	9 (17.65)
Layin Zomo	52	26 (50.0)	26 (50.0)
Samaru	50	33 (66.0)	17 (34.0)
TOTAL	504	331 (65.7)	173 (34.3)

Note: As shown in the map in appendix L.

Table 2: Ownership of Indigenous Chicken according to Marital Status of Local Women Around Zaria

MARITAL STATUS	WOMEN INTERVIEWED	OWNERS	NON-OWNERS
MARRIED	380	292 (76.3)	88 (23.2)
DIVORCED	13	11 (84.0)	2 (15.4)
WIDOW	18	14 (77.8)	4 (22.2)
SINGLE	16	9 (56.3)	7 (43.8)
NO RESPONSE	77	5 (6.5)	72 (93.5)
TOTAL	504	331 (65.7)	173 (34.3)

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Table 3: Distribution of Ownership of Indigenous Fowls Lengths of Marriage among Local Women in Zaria

LENGTHS OF MARRIAGE [YRS]	WOMEN INTERVIEWED	OWNERS	NON-OWNERS
1	119	95 (79.8)	24 (20.2)
2	56	41 (73.2)	15 (76.8)
3	64	41 (64.1)	23 (35.9)
24	44	32 (72.7)	12 (27.3)
5	33	27 (81.8)	6 (18.2)
6	19	15 (79.0)	4 (21)
7	25	22 (88.0)	3 (12)
8	6	4 (66.7)	2 (33.3)
9	46	37 (80.4)	9 (19.6)
10	4	4 (85.7)	0 (0)
11	7	6 (85.7)	1 (14.3)
12	9	6 (85.7)	3 (100)
13 and above	78	0 (0)	71 (91.0)
TOTAL	504	331 (65.7)	173 (34.3)

X^2 = 148.68 dF = 12 significant $P<0.05$

( ) = Specific rates of lengths of marriage in percent (%)

Table 4: Ownership of indigenous Fowls by Pattern of Education of the Local Women Around Zaria.

EDUCATION	WOMEN INTERVIEWED	OWNERS	NON-OWNERS	X^2	dF	P - Value
Can Read Hausa	249	181 (72.7)	68 (27.3)	10.75	1	Significant P<0.05
Can Read English	90	67 (74.4)	23 (25.6)	3.74	1	not significant P >0.05
Can Write English	87	65 (74.7)	22 (25.3)	19.6	1	not significant P >0.05
Can Speak English	102	73 (71.6)	29 (28.4)	1.97	1	not significant P >0.05

Table 5: Pattern of Ownership of Indigenous Fowls according to Years Spent in Primary, Secondary and Tertiary Schools of Local Women Around Zaria

YEARS SPENT PRIMARY SCHOOL	WOMEN INTERVIEWED	OWNERS	NON-OWNERS
DID NOT ATTEND PRIMARY SCHOOL	135	106 (78.5)	29 (21.5)
ATTEND PRIMARY SCHOOL	112	83 (74.1)	29 (24.9)
OTHER TRADITIONAL CLASSES	257	142 (55.3)	115 (44.8)
DID NOT ATTEND SECONDARY SCHOOL	149	118 (79.2)	31 (20.8)
ATTENDED SECONDARY SCHOOL	78	55 (70.5)	23 (29.5)
OTHER FORMS OF POST SECONDARY SCHOOL	277	157 (56.7)	120 (43.3)
TOTAL	504	331 (65.7)	173 (34.3)
MEAN TOTAL	504	331 (65.7)	173 (34.3)

Table 6: Distribution of Local Women's Experience in Keeping Indigenous Fowls Around Zaria

NUMBERS OF WEEKS OF EXPERIENCE	WOMEN INTERVIEWED	OWNERS	NON-OWNERS
1	16	9 (56.3)	7 (48.8)
8	92	68 (73.9)	24 (26.1)
156	296	229 (77.4)	67 (22.6)
157 AND ABOVE	100	25 (25.0)	75 (75.0)
TOTAL	504	331 (65.7)	173 (34.3)

X^2 = 97.73 dF = 3 significant $P < 0.05$

( ) = Specific rate (%) of experience of women keeping fowls.

Table 7: Local Women's Ownership of Domestic Livestock Distribution Around Zaria

NUMBER OF DOMESTIC LIVESTOCK	OWNERS				NON-OWNERS			
1	A	B	C	D	A	B	C	D
	17 (94.4)	-	-	1 (1)	1 (5.6)	1 (100)	1 (100)	8 (88.9)
2	16 (88.9)	5 (100)	1 (25)	1 (20)	1 (20.1)	- (0.0)	4 (75%)	4 (80)
3	8 (87.5)	-	-	-	1 (12.5)	-	2 (100)	2 (100)
4	3 (50)	-	-	1 (25)	4 (50)	-	4 (100)	3 (75)
5	4 (80)	1 (50)	1 (25)	2 (50)	1 (20)	1 (50)	3 (75)	2 (50)
6	2 (100)	1 (100)	2 (100)	-	-	-	2 (100)	3 (100)
7	-		2 (100)	-	-	1 (100)	-	2 (100)
8	1 (100)	-	-	-	-	1 (100)	-	-
9	-	-	-	-	1 (50)	-	-	-
10	1 (50)	-	3 (100)	1 (100)	1 (50)	-	-	-
11< 50 to >50	-	-	-	1(x13) 100(x13)	1 (100)	-	-	1(x13)
DOMESTIC LIVESTOCK KEPT IN THE PAST BUT NOT PRESENT	294 (62)	324 (67.8)	324 (66.8)	-	168 (38)	169 (34.3)	161 (33.2)	136 (98.9)
TOTAL	331 (65.7)	331 (65.7)	331 (65.7)	331 (65.7)	173 (34.3)	173 (34.3)	173 (34.3)	173 (34.3)

KEYS: (A) = Goats, (B) = Sheep, (C) = Cows, (D) = Others Unspecified, ( ) = Specific rates of types of Livestock (a,b,c,d) in percent (%), Sig. = Significant, Insig. = Insignificant.

4.3 SURVEY OF THE MANAGEMENT OF THE INDIGENOUS FOWLS

The women reported that they either bought their birds (9.1%) or were given by relatives (17.2%) or friends (15.4%). The ages of the birds as reported by respondents ranged from less than 1 week to 156 weeks (Table 8). The preferred sex of birds was female as reported by 32% of respondents. However, 192 of the 331 owners indicated that they never bothered about identifying the sex of their birds as they were still young. Most of the owners (62.3%) reported that they housed their chickens only at night; 22.7% housed during the day. About 42% of the owners indicated that they fed their birds only in the mornings and 12.4% did not provide any feed at all (Table 9). The feed consisted of rice bran, maize bran and grains (Table 10). Only very few owners gave protein supplements (12.7%) or vitamins (7.9%). Any container would be used as a feeder (Table 11) and some (21.8%) owners put the feed on the floor for the birds.

None of the owners deliberately gave water to the bird. The birds drank any water around the house even from gutters.

Table 8: Ages of Indigenous fowls Owned by Local Women Around Zaria

AGE RANGE IN WEEKS (WKS)	NUMBER OF WOMEN INTERVIEWED	PERCENT (%)
0 < 1	2	0.6
2 < 12	34	10.3
13 < 52	17	5.1
53 < 156	111	33.5
NO RESPONSE	167	50.5
TOTAL	331	100

Table 9: Feeding of Indigenous fowls owned by Local Women Around Zaria

FEEDING TIMES	NUMBER OF RESPONDENTS	PERCENT (%)
FEED PROVIDED IN THE MORNING ONLY	139	42.0
FEED PROVIDED IN THE EVENINGS ONLY	7	2.1
FEED PROVIDED AT ANYTIME OF THE DAY	61	18.4
FEED PROVIDED THROUGHOUT THE DAY	83	25.1
NO FEEDING	41	12.4
TOTAL	331	100

Table 10: Types of Feeds Provided to Indigenous fowls by Local Women Around Zaria

TYPE OF FEED	NUMBER OF RESPONDENT	PERCENTAGE (%)
RICE BRAN	25	7.6
MAIZE BRAN	139	42.0
WHEAT BRAN	17	5.1
NOT FED WITH BRAN	150	45.3
MILLET (GRAIN)	105	31.7
MAIZE (GRAIN)	3	0.9
GROUNDED MIXED GRAINS	17	5.1
GUINEA CORN (GRAIN)	40	12.1
BIRDS NOT FED GRAIN	166	50.2
MEAN TOTAL	331	100

Table 11: Feeding Utensils Used for feeding Indigenous fowls Around Zaria

TYPE OF FEEDING UTENSIL	NUMBER OF RESPONDENTS	PERCENTAGE (%)
ALUMINUM POT	13	3.9
BROKEN POTS	112	33.8
USED PLATES	95	28.9
BROKEN RUBBER PLATES	16	4.8
BROKEN BUCKETS	3	0.9
OLD PLATE (ANYTYPE)	3	0.9
EARTHEN WARE	1	0.3
CEMENTED HOLES	16	4.8
ON THE BARE FLOOR	72	21.8
TOTAL	331	100

4.4 ECONOMIC ASPECTS OF LOCAL CHICKEN OWNERSHIP

About 26.5% of the 331 owners reported that they raised the chicken for sale only but majority of the owners (56.3%) raised for both sale and home consumption. Sizes range from 1 to 100 with a mean of 11.25 birds. Table 12 shows the mean flock size fowls reported to be lost (10.41), fowls reported dead (13.08), birds that were sold (21.3.), and the mean revenue realized from sale of bird (₦1,466). Sale of birds were mainly in the market, although some owners sold at home at generally cheaper price than if the sale was in the market but birds are usually sold at home and any nearby market.

Table 12: Economic Analysis for Indigenous Fowls Kept by Local Women Around Zaria

ITEMS *	MEAN AT THE TIME OF SURVEY	STANDARD DEVIATION
FLOCK SIZE	11.25	16.99
LOST BIRDS	10.42	12.54
DEAD BIRDS	13.08	18.03
SOLD BIRDS	21.30	29.97
BIRDS EATEN BY HAWKS	10.39	12.33
REVENUE REALISED	N1,466	₦6,244

* Items are per the duration of poultry keeping per owner

4.5 SURVEY OF DISEASED, AND HEALTHY LOCAL FOWLS AND THEIR MANAGEMENT BY LOCAL WOMEN AROUND ZARIA

One hundred and four, 104 (31.4%), of the 504 local women reported they had sick birds at the time of the survey. Table 13 shows the various drugs repoted to be administered to the sick birds by their owners. Sick birds were never routinely taken to veterinary clinics for services of veterinarians.

Majority of the owners, (321(94.3%) (local women) did not vaccinate their fowls birds. Some (5.7%) of those who vacinated their birds did not indicate the type of vaccine that was used. However, 289 (87.3%) owners reported giving their birds some kind of prophylactic treatment consisting of local herbs and 7.0% did not give any prophylactic treatment or vaccine (Table 14). Also, 282 owners indicated their willingness to vaccinate their birds if the vaccines were made available. Level of education of the owners did not have any significant effect on whether they sought veterinary care for their birds or not ($P>0.05$).

Table 13: Treatment Administered to Sick Indigenous fowls by the Owners Around Zaria

DRUG/AGENTS	NUMBER OF RESPONDENTS	PERCENTAGE (%)
TERRAMYCIN "JADA YELLOW" HUMAN MIXTURE	33	31.7
PANADOL	12	11.5
"MADACI"	5	4.8
"GAUTAN KAJI" ALSO 10 OF THE WOMEN USED "JADA YELLOW" FOR TREATMENT MIXED WITH "GAUTAN KAJI".	31	29.8
"PEPPER"	23	22.1
"OIL"	1	1.0
TOTAL	104	100

NOTE: "Gawran Kaji" is the local guarden egg.

Table 14: Types of Vaccines Used to Vaccinate Indigenous fowls by the Owners Around Zaria

TYPE OF VACCINE	NUMBER OF RESPONDENTS	PERCENTAGE (%)
DON'T KNOW THE VACCINE USED	19	5.7
PROPHYLATIC AGENTS	289	87.3
NO PROPHYLATIC TREATMENT	23	7.0
TOTAL	331	100

NOTE: Prophylactic agents are the otal sum of materials used to prevent the local fowls from getting sick.

4.6 PHYSICAL EXAMINATION OF SICK BIRDS OWNED BY LOCAL WOMEN AROUND ZARIA

Table 15 shows the history, clinical signs and treatment of sick fowls. Many owners refused their sick birds being examined. From the history and discussion with the women, the most common diseases were Newcastle disease, fowl pox and ectoparasitic infestation.

The percentage frequencies of respondents who had sick chickens at the time of survey are as below:

Zango (2%) fowl pox, Zago (2%) poisoning, Palladan (2%) mixed infection of cataract and Newcastle diseases, Palladan (2%) mixed infections of helminthiasis, collibacillosis and coccidiosis, Palladan (2%) suspected avian malaria, Palladan (6%) mixed infactions of newcastle disease, infactious bursal disease and ectoparaitism (Table 15).

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Table 15: History and Clinical Findings of Sick Indigenous Birds Owned Local Women of Fowls Around Zaria

VILLAGES	RURAL WOMEN WITH SICK BIRDS, WHO ALLOWED THEIR BIRDS BE EXAMINED	FLOCK SIZE	CHIEF COMPLAINT	HISTORY	CLINICAL SIGNS	TREATMENT	TENTATIVE DIAGNOSIS
ZANGO	1 (2%)	9	NODULES ON NEW BORN CHICKS ON COMBS, BODY EYES ETC	THE CHICKS WERE 1 WEEK, THE ONSET OF THE DISEASE IS ABOUT 5 DAYS, FREE RANGE FLOCKS, NOT HOUSED, SEXES UN-IDENTIFIED	SOFTHARD MASS NODULES ON THE 8 CHICKS AND 1 ADULT UNIDENTIFIED INSECTS WERE ON THE FLOCKS ("Kwari")	NO TREATMENT	FOWL P0X
ZANGO (SHE WAS TOLD BY HER HUSBAND	1 (2%)	100	DEAD OF 100 FLOCKS AFTER AFTER ADMINISTERING POWDER IN WATER TO HEALTHY BIRDS IN DRINKING WATER THAT THE CHICKENS GROW)	DEAD OF ALL BIRDS IN THE FLOCKS	SUDDEN DEATH AFTER ADMINIST-RATION OF POWDER	NO TREATMENT	POISONING
PALLADAN	1 (2%)	1	BLINDNESS OF THE CHICKEN	THE CHICKEN WAS BLIND	LEATHERY COVERING OF BOTH EYEBALLS (THE WOMEN SOLD THE BIRDS A DAY AFTER THE CHECKING)	NO TREATMENT	CATAR-ACT, NEW-CASTLE DISEASE

(%) = percentage frequency of respondents.

Table 15: Continued.....

PALLADAN	1 (2%)	1	WATERY FAECES IN A BROODING FEMALE CHICKEN	THE BIRDS WERE HOUSED IN A MUD BUILDING, CHICKEN WAS LAYING EGGS, FEED WAS PROVIDED IN THE MORNING ON THE GROUND, (THE CHICKEN WAS HOUSED WITH DUCKS.	SOILED VENT, WHITISH DIARRHEA ON THE GROUND, CONGESTED MUCUS MEMBRANE	NO TREATMENT	HEL-MINTHIASIS COLIBACI-LOSIS AND COCCIDIOSIS
PALLANDAN	1 (2%))	MANY	FEVER	HIGH TEMPERATURE ON THE BIRDS, CLUS-TERING OF BIRDS TOGETHER, BIRDS WERE HOUSED TOGE-THER WITH GOATS.	NO PHYSICAL EXAMINATION, SHE LAIMED BIRDS WERE HEALTHY THEN	"MADACI" IN WATER	SUSPECTED AVIAN MALARIA
PALLADAN	3 (6%)	MANY	"CHIWON BORI"	DEAD OF SOME BIRDS DURING HARMATTAN GREENISH DIARRHEA, CRAZINESS OF CHICKEN, "LOUSIZ-INESS" ON BIRDS, ANOREXIA, REST-LESSNESS, LOSS OF HARD FEATHERS	BIRDS HAD GONE ON RANCH (NO EXAMINATION WAS CONDUCTED	NO TREATMENT	NEW-CASTLE DISEASE, INFECT-IOUS BURSAL DISEASE AND ECT-OPARASI-TISM

4.7 IDENTIFICATION OF INTESTINAL PARASITES IN SLAUGHTERED LOCAL CHICKENS

Of the 203 birds examined for intestinal parasites, the following results were obtained: Coccidia oocyst [6.9%], Ascaridia eggs [12.8%], Gongylonema eggs (0.5%), Cyathostoma eggs (1.0%), Railleitina eggs (1.5%), Capillaria eggs (0.5%), Hymenolepis eggs (0.5%), Heterakis eggs (0.5%). No parasites were seen in 153 (75.5%) of the 203 samples examined (Table 16).

Table 16: Intestinal Parasites of Indigenous fowls slaughtered at the Zaria Abattoir

	INTESTINAL PARASITES									
	COCCIDIA OOCYST	ASCARIDIA EGGS	GONGYLONEMA EGGS	CYATHOSTOMA EGGS	RAILLIETINA EGGS	CAPILLERIA EGGS	HYMNOLEPIS EGGS	HETERAKIS EGGS	NPF	TOTAL
NUMBER OF TEST SAMPLES	14	26	1	2	3	1	1	1	153	203
PERCENT (%)	6..9	12.8	0.5	1	1.5	0.5	0.5	0.5	75.4	100

Total positive = 50 (24.6%)
Total negative = 153 (75.4%)
NPF = No parasite found

4.8 BLOOD PARASITES SEEN IN INDIGENOUS BIRDS SLAUGHTERED AT ZARIA ABATTOIR

The frequencies of blood parasites seen are shown in Table 17. Plasmodium sp (25.5%) was the most frequent blood parasite seen. About 62.1% of the 206 samples were negative for blood parasites.

Table 17: Blood Parasites Seen in Indigenous fowls Slaughtered at the Zaria Abattoir

	BLOOD PARASITES							
	LEUCO	**PLASMO**	**AEGYP**	**LEUCO + PLASMO ***	**B.ANSERINA+ PLASMO ***	**PLASMO + AEGYP**	**NPF**	**TOTAL**
NUMBER OF TEST SAMPLES	1	53	17	1	1	5	128	206
PERCENT (%)	0.5	25.7	8.3	0.5	0.5	2.4	62.1	100
TOTAL	0.5	25.7	8.3	0.5	0.5	2.4	62.1	100

Total positive = 78 (37.9%)
Total negatives = 128 (62.1%)
NPF = No parasite found.
** Mixed infection

KEYS:
LEUCO = *Leucocytozoon* sp
PLASMO= *Plasmoduim* Sp
AEGYP = *Aegyptianella pullorum*
B. ANSERINA = *Borelia anserina*

4.9 ANTIBODY TO NCD IN SLAUGHTERED BIRDS FROM ABATTOIR AROUND ZARIA

Table 18 shows the distribution of titres of antibody to Newcastle disease virus in the local chicken slaughtered in Zaria Abattoir. About 46.5% of the birds had antibody titres of 1:8 or more.

4.10 DISTRIBUTION OF ANTIBODIES TO IBDV IN THE SLAUGHTERED BIRDS

Of the 228 local chickens tested, 122 (53.5%) had precipitin lines indicating antibodies to IBDV.

Table 18: *Distribution of Newcastle Disease Antibody Titres in Sera of Slaughtered* Indigenous Chickens

ANTIBODY TITRES	0	4	8	16	32	64	128	256	512	1024	2058	
NUMBER OF SAMPLES TITRE	78	28	19	13	10	1	6	6	11	15	13	200
PERCENTAGE (%)	39	14	9.5	6.5	5.0	0.5	3.0	3.0	5.5	7.5	6.5	100
INTER-PRETATION		<----------> 53.5% -VE		46.5% Positive Ab titres <----------								

NB: Antibody titres of 1.8 and above were considered positive for unvaccinated indigenous birds only.

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CHAPTER FIVE

5.0 DISCUSSION

Two-thirds (331) of the 504 rural women interviewed owned local fowls. Akinwumi *et al.* (1979) and Abdu and Saidu (1995) stated that rural women are the sole owners of indigenous chickens. The reasons for this ownership by the local women may be to create job opportunities for the women to enhance their standard of living and to increase their protein availability.

The distribution of the indigenous fowls in the villages around Zaria owners shows almost the same pattern as obtained in a study by Akinwumi *et al* (1979) conducted in some Northern states, Kaduna, Kano, Katsina, Sokoto and Northern region as Zaria, Giwa, Funtua, etc.

Factors such as ethinicity, type of marriage, trading, educational status, ownership of goats influenced ownership of local fowls by the rural women.

Married women in polygamous setting, and older women and divorcees involve themselves in the production of local fowls. They also appear to be more experienced in raising chickens than the other marital groups. This is because majority of the married women hardly go out. It is possible that the women keep poultry to create job opportunity for them or create the feeing that they have something to show to the community i.e. it helps them to avoid being ridiculed by their neighbours. Some of the older women expressed that they have alot of

responsibilities in terms of family managememt, therefore, they raise local fowls as a means to an end.

Education was not a factor determining ownership. But the knowledge of western education will assist in improving on hygiene of the surrounding. Lack of western education could be the reason for poor hygiene of the environment in the rearing of fowls in Zaria. Many fowl owners leave faeces of the birds everywhere around the compound. This constitutes a public health problem and creates disease risk to man and other animals. Religion, customs and tradition have been a barrier for important poultry hygiene management because most women that keep the fowls stay indoors. The respondents explained that they don't cater for the fowls believing that every sickness is brought by god and also god will heal the chicken.

Majority of the rural women were traders. Trading could be another source of funding to the rural women and it is possible for them to find some source of finance to invest on chickens. Results of this study showed that the less educated women own more of local fowls.

The owners that are 2, 3, and 9 years (younger wives) in marriage that keep local birds are averagely 50 - 60% of the number that owns poultry. There is no reason for this distribution. This agrees with the work Abdu *et al.* conducted in 1995, which he stated that, the owner that keeo average local fowl rarely exceeds 60%.

Some rural women were involved in associations as other means of raising funds. Saidu *et al.* (1994) had earlier stated that the women are involved in associations called "Asusu" and "Adashe". The additional funds that the women generates from "Adashe" and "Asusu" are used to purchase domestic fowls and goats for keeping as seed ("irri").

There are other occupations the owners were involved in during the rainy season and dry season. This agrees with the work of Abdu and Saidu (1995). It is also a means of financing the needs of the rural owners.

Some of the women have 3 years (156 weeks) of consecutive experience of keeping poultry. Some did and later abandoned poultry keeping, some got frustrated as garthered from their complaints. Saidu *et al.* (1995) stated that, usually, the women are unwilling to invest in poultry production. The local women's efforts were frustrated because their fowls were dying due to sicknesses especially during the harmattan.

Statistical data established here that ownership is affected ($P > 0.05$) by rearing other domestic animals like goats; that is, that not much attention is directed to poultry keeping although the women rare the local fowls but instead they cater for the goats more because they feel it generates more money, goats are cheaper than sheep, can be bought for any occasion better than sheep (sheep may be only for Sallah).

There was price variation depending on where the fowls were sold. The *owners got their birds from nearby markets and some got as gift from relations.* This agrees with the study of Saidu *et al.* (1994) which he identified indigenous poultry marketing as a problem, with no definite markets for the indigenous fowls in the villages.

The indigenous fowls can survive up to 3 years. The birds must have developed resistance to disease causing agents over the years by constant exposure. Agbede (1981 and 1985) had discussed on diseases and control measures in local birds. *In his suggestions, he stated that resistance to diseases are developed by* contacting with the disease agent and developing immunities over that particular agent. It also agrees with the work of Adekeye *et al.* (1989). The local breed are resistant to some diseases (Akut, 1991 unpublished DVM project; Oluyemi and Roberts, 1985). The female fowls (hens) are more in number (58%) within the rural community, possibily because the hens need to produce chicks for future rearing.

The local fowls were not housed in the day time. They were mostly housed at night only. The local birds roam to feed all day (free range management system). Majority of owners did not provide feeds and water for the birds. Saidu *et al.* (1994) conducted studies on local birds and stated the birds were usually on free range. The free range system has public health significance to the owners. The birds also serve as carriers of disease agents (Abdu and Saidu, 1995). Some of the birds eat

in dirty places and drink water from gutters. The environment and the habit of the birds expose the owners the birds and other domestic animals to dangerous disease pathogens.

Majority of the owners reported that the whole flock died during the dry season which agrees with the work of Abdu *et al.* (1985). The dry harmattan being windy serves as vehicle for tranfer of airborne diseases. The birds ate food and drank water from dirty environment and gutters which expose them to parasites and other disease causing agents (Tegene, 1993). Diseases have been a major cause of losses of birds within the rural community (Saidu *et al.*, 1994).

The women prefer to use local herbs as a form of medication for their fowls. Nwude and Ibrahim (1980) studied on ethnoveterinary practices, and established that some of the herbs like "Taba" leaves have pharmacological importance. In this study, I will want to state that some of the herbs could cause death to the flocks since they are crude and it is possible to cause poisoning to the birds. There are no correct doses for the herbs administered to the local fowls. It is possible for the local fowls to die due to overdose.

Very few rural owners treated there chickens with the recommended drugs. Abdu and Saidu (1995) stated that the local owners are unwilling to care for these local fowls. Saidu *et al.* (1994) stated that birds are uncared for, and also unvaccinated in the rural areas. Therefore, lack of treatment, lack of vaccination

and poor management could result in massive deaths of birds.

The paliative drugs of choice for the owner has been the local herbs. One woman reported the powder she gave as treatment, killed all the her birds. This indicates that administration of local drugs which do not have proper information or appropriate doses could be dangerous to the birds.

Economically, losses value are as follows: lost birds due to missing birds (mean 16.42±SD.12.54; dead birds (mean 13.08±SD18.03); sold birds (mean 21.30±SD29.97) and birds eaten by hawks (mean 10.39±12.33) per duration of poultry keeping owners.

Losses were reported in this study through dissapearance of the birds, predators e.g. hawk eating some the birds and death. Saidu *et al.* (1994) stated also that predators eating the birds cause losses in local birds.

The mean revenue realised for the period of keeping birds per woman was ₦1,466.00. If the money is shared per year, it is very meagre. The funds raised is so small because these rural owners sell these birds just for little money.

Some of the reasons for low production and break down in production were as follows:-

(a) *Diseases of local birds frustrate the efforts of the local owners*

(b) Family tie (some do not slaughter the birds because of attachment to the birds), so they raise the birds to extend their relationships by giving as gifts

to relatives and friends (Akut, 1991).

(c) Mass sales before harmattan due to fear of sudden death of flock during the dry weather (Saidu *et al.*, 1994)

Keeping large number of the indigenous goats and poultry at the same time is a usual practise among the rural owners. These domestic livestock are slaughtered to serve guest, eaten up during ceremonies and also sold to raise funds in addition with the funds from the local fowls.

Majority of the owners kept the hens for breeding and production.

Management of the local birds is poor due to:

(a) Lack of knowledge in poultry hygiene.

(b) No knowledge of poultry diseases by majority of the local owners.

(c) Lack of understanding that women/children and members of the households can contact diseases through the local birds.

(d) The owners easily adopt to seasonal keeping of local birds.

(e) Agile nature of the local birds makes drug administration and vacination difficult.

The poor attitude of the owners countributes to the diseases of the birds and eventually the death of the birds (Saidu *et al.*, 1994, Abdu and Saidu 1995).

Management factors such as, the women not feeding the birds or not providing water for all the birds affect the performance of the birds. The free range system exposes the birds to disease pathogens. The birds drink water from the surrounding where they get contact with disease pathogens. Diseases are

dangerous to the health of the owners and also the local birds.

Some how, the free range system can also be profitable to the birds, in the sense that they are exposed to sunlight necessary for vitamin D synthesis and they also get nutrients from the ground.

There are environmental factors such as, predators, dirty feeding environment, dise6ase pathogens and squatting of livestock in the same room with chickens which to discourage ownership of chickens. Predators, hawks, cats and dogs eat the chicks. Disease pathogens causes poor breeding, poor production and death of birds. The goats also match on the chicks and kill them. Saidu *et al.* (1994) stated that squatting of local birds with domestic animals causes overcrowding and poor production due to stress and even death of local birds and due to matching of birds by larger domestic livestock.

Economically, the poultry keeping adds to the revenue of the owners after sales. The birds also serve as an immediate source of protein. Serving the meat to guest, build up existing social bond and attachment in relationship among the villagers Akut (1991).

There are several disease causing agents detected in the local birds managed by women in Zaria. The public health significance is that the owners and birds are exposed to diseases and parasites.

The women use a lot of local "herbs" for prevention and this agrees with the work of the Abdu and Saidu (1995). Some of these herbs like "Taba" has been proven pharmacologically as stated in 1980 (Nwude and Ibrahim, 1980) to be effective. But some of the drugs can be dangerous due to having crude elements, overdosing and underdosing of the birds. In addition, 2poor production could be due to the adverse effects of the herbs used on the local birds.

85% of the women interviewed said they will give vaccines if available and 15% will not give caccines even if available.

Lack of modern drugs and the unwilling attitude of the owners make prevention and control of the diseases difficult. Poor hygiene, poor management by the owners, owners attitude and also the agile nature of the birds make handling difficult during treament and vaccination.

5.1 CONCLUSION

There are factors that determine ownership of local poultry by the rural women such as trading and ownership of other domestic livestock.

The women have poor attitude towards management of birds. No water was provided for the birds. There were poor housing, poor feeding and little feed supplements which exposed the birds to predators and disease pathogens.

There were problems of overcrowding and stress.

Low production is as a result of poor management and hygiene which expose the

birds to diseases and stress.

In terms of economy, the birds provide immediate protein to the rural women and their households. However, but in terms of financial contribution, the poor marketing system does not give room for raising large funds to meet their immediate needs.

Many disease causing agents were detected showing that diseases are a major constraint to production of the birds in large size. There are lack of drug to the local birds due to the owners' behaviours, unwillingness of the women to invest on production due to their efforts being frustrated. Sudden death of birds occur due to marching of birds by squatted goats, cows etc in one place with local birds. They have some knowledge of prevention and control of diseases since the local owners use herbs (ethnoveterinary practices) and few used unknown vaccines. They were not aware that diseases can be transfered from the birds to humans.

5.2 RECOMMENDATIONS

(1) The women should provide good housing, water, feeds and feed supplements for the fowls.

(2) Those factors such as good hygiene of the environment, vaccination of the chickens against diseases etc that enhance ownership should be encouraged.

(3) The factors such as squatting of livestock in the same place with chickens, chickens eating from gutters etc that could result in diseases should be prevented.

(4) Housing and squatting of domestic livestock with local birds should be avoided.

(5) The birds should be vaccinated against common diseases such as NCD, IBD, FP, fowl cholera etc for against which vaccines are available such as Newcastle disease and Gumboro disease.

(6) Treatment against parasites should be administered regularly.

(7) Cleaning of the environmnet where poultry are kept enhances the health of the birds and the owners. These help in preventing transfer of diseases. When the health of the poultry is enhanced, the livability of the local flock is increased when the local birds are healthy, production is enhanced so that when the birds are sold, the economic status of the women is improved.

REFERENCES

1. Abdu, P.A. Abdullahi, S.U. and Adesiyun, A.A. (1984): Infectious bursal disease virus antibody in chickens from Fulani nomads around Zaria. *Nigerian Veterinary Journal*, 13:2-3.

2. Abdu, P.A., George, J.B. and Umoh J.U. (1985): A study of poultry diseases diagnosed at Zaria from (1980 - 1984). *Nigerian Veterinary Journal* 14: 63 - 65.

3. Abdu, P.A., Abdullahi, S.U. Adesiyun A.A and Ezeokoli C.D. (1986): Infectious bursal disease. *World Poultry Science Journal* 42:219-230.

4. Abdu, P.A., Mera, U.A. and Saidu, L. (1992): A study on chicken mostly in Zaria, Nigeria. *Proceedings of the National Workshop on Livestock and Veterinary Services* held at the National Veterinary Research Institute, Vom. (August, 11th - 14th, 1992) (An abstract paper).

6. Abdu, P.A. and Saidu, L. (1995): Diseases of Local chickens and possible methods of their control. An absract of the *Workshop on Small Animal and Poultry Production*. National Veterinary Research Institute, Vom. 11th - 13th October, 1995.

7. Abdullahi, S.U., Abdu, P.A.; Ibrahim, M.A.; George, J.B.D.; Saidu, L; Adekeye, J.O. and Kazeem, H.M. (1992): Incidence of diseases of Poultry caused by non viral infectious agents in Zaria, Nigeria. The proceedings of the *World Poultry Congress Armsterdam, The Netherlands, 20th -24th September*, 1992. pp-159.

8. Adekeye, J.O; Abdu, P.A, Bawa E.K. (1989): Camphylobacter fetus Subspp jejuni in Poultry reared under different management systems in Nigeria. *Avian Diseases* 33 : 801 - 803.

9. Adene, D.F.; and Fatumbi, O. (1979): Diseases of poultry in Indigenous poultry. *Nigerian Veterinary Journal* 8: 230 - 232.

10. Agbede, R.I.S. (1981): A survey of ectoparasites and endoparasites of indigenous fowls; *Journal of Animal Production* 1: 179.

11. Agbede, R.I.S. (1985): The Fowl tick. Its importance and control. *Veterinary Speculum*, 1: 21.

12. Aini, I., Ibrahim, A.L., Favziah O. and Hussein A.A. (1990): Field Trials with an oral Newcastle disease vaccine. Indigenous poultry disease. *Proceedings of the Fifth International Conference on Livestock, Kuala Lumpur Journal* 24:70 (Reviewed pp 127-129).

13. Akinwumi J.A.; Adekeye, A.J., Ikpi, A.E. and Olayide, S.O. (1979): Economic analysis of Poultry Industry in *Livestock Production Plan for Nigeria*, 1981, Pg 65.

14. Akut, A.J. (1991): A study of indigenous poultry health, husbandry and preventive medicine among Moroa community. An *unpublished DVM PROJECT* submitted to the Faculty of Veterinary Medicine, Dept. of Surgery and Medicine: Pg 22.

15. Allan, W.H. and Grough, R.E. (1974): A standard Hemagglutination Inhibition (HI) test for Newcastle disease. A Comparism of microtitre method. *Veterinary record* 95 : 120 - 124.

16. Almassy, K. and Kakuk, T. (1976): Immnuosuppressive effect of a naturally acquired subclinical bursal agent infection on vaccination against Newcastle disease. *Veterinary record* 99: 435 - 437.

17. Almeida, J.O. and Morris, R. (1973): Antigenically related viruses associated with infections bursal disease. *Journal of General Virology* 20: 369 - 375.

18. Arnall, L. and Keymes, I.F. (1975): An abstraction on bird diseases Baillere Tindall, London, United Kingdom. Book society/Baillere Tindall).

19. Asrubali, G. and Gilletti, L. (1971): Diagnosis and Pathogenesis of Gumboro disease studied by Means of immunoflorescence, *Nvova Veterinarian* 47: 223-229. (English summary).

20. Auer, J. (1952): Functional localisation of lesions in Newcastle disease. I. General Survey. *Canadian Journal of Comprehensive Medicine Veterinary Science* 16: 277 - 284.

21. Bagaiwa, U.L.M. (1986): Isolation of Yersinia Pseudotuberculosis from chicken slaughtered at Sabon-Gari Market, Zaria. An unpublished DVM PROJECT, submitted to the Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, Nigeria.

22. Baker, E.J. and Silverton, R.E (1985): Introduction to Medical Laboratory Technology (6th Edition), pp 13-42. Butterworth and Co (Publishers) Limited (1985).

23. Bankowski, R.A., and Costervet, R. (1960): Immunity and the reproductive tract of laying hens vaccinated with the tissue culture. Newcastle disease virus. *American Journal of Veterinary Research 21*: 610 - 617. (published PH.D thesis by Wiscousin Vniversity Press).

24. Beach J.R. (1942): Avian Pneumoencephalitis *Proceedings of Annual Meeting of United States on Livestock Sanitation Association* 48: 203 - 223.

25. Beach J.R. (1946): The status of Avian Pneumoencephalitis and Newcastle disease in the United States. *Journal of American Veterinary Medical Association* 108: 372 - 376.

26. Beard, C.W. and Easterday, B.C. (1967): The influence of the route of administration of Newcastle disease virus on host response I. Serological and virus isolation studies, II studies on artificial passive immunity III. Immunofluorescent and histopathological studies. *Journal of Infectious diseases* 117: 55-61, 62, 65, 66 - 70.

27. Beaudette, F.R. and J.J. Black (1946): Newcastle disease in the New Jersey. *Proceedings of Annual Meeting of United States on Livestock Sanitation Association* 49: 49 - 58.

28. Benton, W.J., Cover M.S. and Rosenberger J.K. (1967a): Studies on 3the transmission of Infectious Bursal Agent (IBA) of chickens. *Avian diseases* 11: 430 - 438.

29. Benton, W.J., Cover M.S. and Rosenberger J.K., and Lake R.S. (1967b): Physico - chemical properties of the Infectious Bursal Agent (IBA). *Avian diseases* 11: 438 - 445.

30. Berg N.W. (1982): Rapid detection of infectious bursal disease antibodies

by counter immunoelectrophoresis *Avian Pathology* 11: 611 - 614.

31. Bird, H.R. (1964): The changing environment of chicken, Pp. 35 - 46. In Hauson, R.P. (Ed), Newcastle disease virus. An evolving pathogen. *University of Wisconsin Press, Madison*

32. Bisural, G.; and Morill, C.C. (1954): The pathology of the reproductive track of laying pullets affected with Newcastle disease. *Poultry Science Journal* 33: 880 - 897.

33. Brady J. (1970): Infectious bursal disease of poultry. *World's Poultry Science Journal* 26: 658.

34. Burnet F.M. (1942): The affinity of Newcastle disease virus to the infuenza virus group. *Australian Journal of Experimental Biology Medical Science* 20: 81 - 88.

35. Burnet, F.M. (1950): The hemolytic action of Newcastle disease virus I, Two types of interaction between virus and red cells. *Australian Journal of Experimental Biology Medical Science* 28: 299 - 309.

36. Burnet, F.M.; and Lind, P.E. (1950): Hemolysis by Newcastle disease virus II. General character of the hemolytic action. *Australian Journal of Experimental Biology Medical Science* 28: 129 - 150.

37. Cernik, J. (1983): Thermostability of a Vaccine strain of avian infectious bursitis virus. *Veterinary Medicina* 28: 81 - 88. (English summary).

38. Chettle, N.J., Eddy R.K, and Wyeth P.J. (1985): The isolation of infectious bursal disease virus from Turkeys in England. *British Veterinary Journal* 141: 141 - 145.

39. Chineme, C.N. and Cho Y. (1984): Clinico-pathological and morphological changes in chickens experimentally infected with infectious bursal (Gumboro) disease virus. *Tropical Veterinarian* 2: 218 - 224.

40. Cho, B.R. and McDonald, T.L. (1980): Infectious bursal disease virus. Further characterisation with evidence for a single stranded RNA virus. *Avian diseases* 23: 423 - 434.

41. Cho, B.R.; Raymon, R.G and Hill R.W (1979): Growth of infectious bursal disease virus in plaque formation in chickse embryo fibroblast cell culture. *Avian diseases* 23: 209 - 218.

42. Cosgrove, A.S. (1962): An apparently new disease of chicken - Avian nephrosis. *Avian diseases* 6: 385 - 384.

43. Dobson, N. (1939): Newcastle disease *Proceedings of the Seventh World's Poultry Congress* 7: 250 - 253.

44. Doyle, T.M. (1927): A hitterto unrecorded diseases of fowls due to a filter passing virus. *Journal of Comprehensive Pathology and Therapeutics* 40 : 144 - 169.

45. Ezeokoli, C.D.; Umoh, J.U.; Adesiyun, A.A. and Abdu, P. (1984): Prevalence of Newcastle disease virus antibodies in local and exotic chickens in Nigeria. *Bulletin of Animal Health Production in Africa,* 32: 253 - 257.

46. Fahey K.J, O'Ponnell, I.J. and Azad, A.A. (1985): Characterization by Western blotting of the immunogens of infectious bursal disease virus. *Journal of General Virology* 66: 1479 - 1488.

47. Faragher, J.T. (1972): Infectious bursal disease of chickens. *Veterinary bulletin* 42: 361 - 369.

48. Fletcher, O.J.; Pag E.R.K.; Lwkert, P.D; Gelb, J. and Kleven, S.H. (1978): Responses of progeny to infectious bursal disease virus vaccination when obtained from breeder flock vaccinated with either an inactivated or live infectinus bursal disease virus. *Proceedings of the One Hundred and fifteenth Annual Meeting of the American Veterinary Medical Association, Dallas.* pp. 156.

49. Gadzama, E.N., and Srivastava, G.C. (1986): Prevalence of intestinal parasites of market chickens in Borno State. *Zariya Veterinarian* 1: 126 - 127.

50. Gale, G. (1964): Recognition of Newcastle disease, as a new disease. Discussion, P.99. In Hanson, R.P. (Ed), Newcastle disease virus. An evolving pathogen, *University of Winsconsin Press, Madison*.

51. George, B.D.J.; Abdul, P.A. and Saidu L. (1990): An abstarct of the *14th Annual Conference of the Nigerian Society for Parasitology* September 23rd - 26th, 1990. Bayero University, Kano.

52. Giambrone, J.J.; Ewest, D.L. and Eidson, C.S. (1977): Effect of infectious bursal disease virus on the immunological responsiveness of the chicken. *Poultry Science journal* 56: 1591 - 1594.

53. Giambrone, J.J.; Anderson, W.I.; Reid, W.M. and Eidson, C.S. (1982): Effect of infectious bursal disease virus on the severity of Eimeria tenella in broilers. *Poultry Science journal* 56: 243 - 245.

54. Haffer, K. (1982): Field test studies of the 2512 strain of infectious bursal disease *Avian diseases* 26 : 847 - 851.

55. Hanson, R.P. (1949): Characteristics of Certain Strains of Newcastle disease virus. Ph.D. Thesis pp 1-352. University Wisconsin, Madison.

56. Hanson, R.P. (Ed). (1964): Newscatle disease virus. An evolving pathogen, Pp 1 - 352. *University of Wisconsin Press, Madison*

57. Hanson, B.S. (1967): Post-mortem lesions diagnostic of certain poultry diseases. *Veterinary record* 8: 109 - 122.

58. Hanson, R.P. and Brandly, C.A. (1955): Identification of Vaccine Strains of Newcastle disease virus. *Science journal* 122: 156 - 157.

59. Hanson, R.P.; Sinha, S.K.; Brandly C.A. (1952): Comparison of the tropisms of six strains of NCD virus in chickens following aerosol infection. *Journal of Infectious diseases* 91: 276 - 282.

60. Hemboldt, C.F. and Garner, E. (1964): Experimentally Induced Gumboro Disease (IBD). *Avian diseases* 8: 561 - 575.

61. Hirai, K.; Shimakura, S. and Hirosa, M. (1972): Immunodiffiusion reaction of avian infectious bursal disease virus. *Avian diseases* 16: 961 - 964.

62. Hitchner, S.B.; and Johnson, E.P. (1948): A virus of low virulence for immunizing fowls against Newcastle disease (Avian Pneumoencephalitis) *Journal of Veterinary Medicine* 43: 525 - 530.

63. Hitchner, S.B.; Reisang, G. and Van Roekel, H. (1950): The intranasal vaccine. Its role in a newcastle disease control programme. *Proceeding of Annual Meeting of United States of America Livestock Sanitation Association* 54: 154 - 160.

64. Hitchner, S.B.B. (1976): Immunization of adult hens against infectious bursal disease virus. AVIAN DISEASES VOLUME 20: 611 - 613.65.Hitchner, S.B. (1978): Infectious bursal disease, In: Hofstad, M.S., Canek, B.W; Hemlboldt, C.F.; Reid, W.M.; Yoder H.M. (Ed.), *Diseases of Poultry*, Iowa State University Press, Annes, Iowa, U.S.A. Pp 647 - 654.

66. Howie, R.I. and Thorsen J. (1981): Identification of a strains of infectious bursal diseases virus isolated from mosquitoes. *Canadian Journal of Comparative Medicine* 45: 315 - 679.

67. Ibrahim, M.A. and Abdu, P.A. (1992): Ethno-agro-veterinary perspectives of Poultry Management, Health and Production among the Hausa and Fulani of rural Nigeria. *29th Annual General Meeting of the Nigerian Veterinary Medical Association* Pp 172 - 181.

68. Ide, P.R. (1979): Infectious bursal agent vaccination of chickens from infectious bursal agent vaccinated dams. *Canadian Veterinary journal* 21: 35 - 40.

69. Iyer, S.G. (1943): Studies on Newcastle (Ranikhet) disease virus. *Indian Journal of Veterinary Science* 13: 1 - 26.

70. Jordan, F.T.W. (1990): *Poultry diseases* fifth edition, Pg 177 - 181. English Language Book Society/76ailliere Tindall.

71. Kaufer I. and Weiss E. (1976): Electron microscope studies on the pathogenesis of infectious bursal disease after intrabursal application of the causal virus. *Avian diseases* 20: 483 - 496.

72. Kilman L. (1949): Newcastle Disease Virus (NDV) hemolysis. *Proceedings of Society of Experimental Biological Medicine* 71: 63 - 66.

73. Lancaster, J.E. (1966): Newcastle disease, An abstract review 1926 - 1966 Can Dep. Agri. Monograph 3, Queen's Printer, Ottawa.

74. Lukert, P.D. and Davis, R.B. (1974): Vaccination of Maternally immune chickens with an attenuated infectious bursal disease vaccine. *Journal of American Veterinary Medical Association* 199: 1141.

75. Luthgen, W. (1969): Gumboro disease. *Veterinary Medical Review* 1: 3 - 18.

76. McFerran, J.B.; McNulty, M.S.; McKillop, E.R.; Connor, J.J.; McCracken, R.W.; Collins, D.S. and Allan, G.M. (1980): Isolation and Serological Studies with bursal disease viruses from fowls, turkeys and ducks: Demonstration of a Second Serotype. *Avian Pathology* 9: 395 - 404.

77. McNulty, M.S; Allan, G.M. and McFerran, J.B. (1979): Isolation of infectious bursal disease virus from Turkeys. *Avian Pathology* 8: 205 - 212.

78. Meroz, M. (1966): A epidemiological Survey of Gumboro disease. *Refuah Veterinarith* 23: 235 - 237.

79. Muskett, J.C.; Hopkins, I.G.; Edwards, K.R. and Thornton, D.H. (1979): Comparison of two infectious bursal disease vaccine strains - Eficacy and potential hazards in susceptible and maternally immune birds. *Veterinary Record* 104: 332 - 334.

80. Naqi, S.A.; Marquez, B. and Sahin, N. (1983): Maternal antibodies its effect on infectious bursal disease immunization. *Avian diseases* 27: 623 - 631.

81. Nick, H.; Cursiefen, D. and Becht, H. (1976): Structural and growth characteristics of infectious bursal disease virus. *Journal of Virology* 18: 227 - 234.

82. Nawathe, D.R.; Onunkwo O.; Smith, I.M. (1978): Serological evidence of infection with the virus of infectious bursal disease in wild and domestic birds in Nigeria. *Veterinary Record* 102: 444.

83. Nwude, N. and Ibrahim, M.A. (1980): Plants used in traditional veterinary medical practice in Nigeria. *Journal of Veterinary Pharmacology and Therapeutics*. (An abstract).

84. Ojo, M.O.; Oduye, O.O.; Noibi, L.M. and Idowu, A.L. (1973): Gumboro -like disease in Nigeria. *Tropical Animal Health and Production* 5: 5256.

85. Onunkwo, O. (1975): An outbreak of Infectious Bursal Disease (IBD) of chickens in Nigeria. *Veterinary Record* 97: 433.

86. Okeke, E.N. and Lang, G. (1981): The application of the Immunoperoxidase (IP) antibody technique for the etiological diagnosis of Infectious Bursal Disease (IBD) in chickens. *Nigerian Journal of Animal Production* 8: 36 - 49.

87. Okeke, E.N. (1984): Infectious bursal (Gumboro) disease (IBD). *Proceedings of National Workshop on diseases of Livestock and Poultry,* National Veterinary Research Institute, Vom, Nigeria, Pg 76- 80.

88. Okoye, J.O.A. and Uzoukwu, M. (1981): An outbreak of Infectious Bursal Disease among chickens between 16 and 20 weeks old. *Avian Diseases* 25: 1034 - 1039.

89. Okoye J.O.A. and Uzoukwu, M. (1984): Histopathogenesis of infectious bursal disease in the bursal of Fabricius. Persistence of infectious bursal disease virus and the appearance of preoccupations in infected chickens. *Tropical Veterinarian* 2: 91 - 102.

90. Olabode, A.O.; Lamorde, A.G.; Shidali, N.N. and Chukweledo, A.A. (1992): Village chickens and Newcastle diseases in Nigeria. Newcastle disease in local chicken. *ACIAR Proceedings* No.39: Pg 159 - 160.

91. Olorin, E.A. (1987): Some aspects of the physical geography of the Zaria region related human responses. An unpublished Departmental Lecture Note No.1. k Geography Department, Bayero University, Kano, Nigeria.

92. Oluyemi, J.A. and Roberts, F.A. (1985): Poultry production in warm wet climate: Pg 16 - 28. (McMillan Press Publisher).

93. Paniscup, S.A; Verma, K.C. and Mohaty, G.C. (1984): Age resistance in chickens against infectious bursal disease. *ACTA Veterinaria* Scandinavica 28: 561 - 566.

94. P8rkhurst, R.T. (1964): Pattern of mortality in avian nephosis. *Poultry Science* 43: 788 - 789.

95. Pattison, M.; Alexander, D.J.; and Harkness, J.W. (1975): Purification and preliminary characterization of a pathogenic strain of infectious bursal disease virus. *Avian Pathology* 4: 175 - 187.

96. Peter, M.D.; Aprile, P.N. and Cancellotti, F. (1973): Biological and Physiochemical Properties of the infectious bursal disease virus (IBDV) *Avian Pathology* 2: 135 - 152.

97. Petrak, M.L (1969): Diseases of cage and aviary birds LEA and Feibiges, Philadelphia U.S.A. (cited in the proceedings of the fifth *International Conference on Livestock, Kuala Lumpur Journal* 24:127-129).

98. Report of Poultry diseases sub-committee on Animal Health, Agr. Bd., Div. Biol. Agri, Nat. Acad. Sci, Nat. Res. Council, Washington D.C. (1963). Methods for the examination of Poultry Biologics, 2nd Ed. (Rev.). Publ. 1038: Pg 1 - 158.

99. Ressang, A.A. (1961): Newcastle disease Indonesia Part II, its symptomatology gross and microscopic anatomy. *Communicable Veterinarian (Borgor Indonesia)* 5: 16 - 37.

100. Saidu, L.; Ogbogu, V.C. and Abdu, P.A. (1993): Retrospective study of helminthic infection of poultry in Zaria. *30th Annual Conference of the Nigerian Veterinary Medical Association,* Abeokuta, Ogun State, 20th - 27th October, 1993.

101. Saidu,L.; P.A. Abdu., Umoh, J.U. (1994): Diseases of Nigerian Indigenous Chicks. *Bulletin of Animal Production in Africa* 42:19- 23.

102. Saidu, L.; Agbede, R.I.S; and Abdu, P.A. (1995): Prevalence of Avian Spirochaetosis in Zaria (1980 - 1989) *Israel Journal of Veterinary Medicine* 50: 1.

103. Simmon, G.C. (1967): The isolation of Newcastle disease virus in Queen's Land. *Australian Veterinary Journal* 43: 29 - 30.

104. Sinha, S.K.; Hanson, R.P.; Brandly, C.A. (1952): Comparison of the tropisms of six strains of NCD virus in chickens following aerosol infection. *Journal of Infectious Disease* 91: 276 - 282.

105. Sinha, S.K.; Hanson, R.P. and Brandly, C.A. (1954): Aerosol transmission of Newcastle disease in chickens. *American Journal of Veterinary Research* 15: 287 - 292.

106. Sinha, S.K.; Hanson, R.P. and Brandly, C.A. (1957): Effect Environmental Facility of Aerosol Transmission of Infection and Severity of Newcastle Disease Among Chickens. *Journal of Infectious diseases* 100: 162 - 168.

107. Singh, R.A. (1990): An abstract of Poultry Production, submitted to Department of Livestock Production and Management, Aaryanck Agricultural University Hisar, Kalyau Publishers, New Delhi.

108. Sivanandan, V.; Limermpao, J.; Benson, H.J. and Newman J.A. (1984): Serological evidence of infectious bursal disease virus serotype II infection in Minnesota Turkeys. *Avian Diseases* 28: 765 - 769.

109. Snedeker, C.; Wills, F.K. and Moulthrop, I.M. (1967): Some studies on the infectious bursal agent. *Avian Diseases* 11: 519 - 529.

110. Szubartowska, E.G; Grounysz-Kalwovska, K. (1992): Blood Morphology in Poultry and Quails after poisoning with Feninothion. Comparative Biochemistry and Physiology (record cited in the review of PH.D thesis, University of Wisconsin Press, Madison pp. 519-529).

111. Taylor, L.W. (1964): The chicken as an evolving host, PhD Thesis, Pages 23 - 24. In Hanson R.P. (Ed); Newcastle disease virus. An evolving pathogen university of Wisconsin Press, Madison. pp 23-24.

112. Tegene N. (1993): Prevalence of diseases, predators of local chicken in Ieku, Southern Ethiopia *Bulletin of Animal Production* 41: 317 - 321.

113. Umoh J.U.; Ezeokoli, C.D.; Adesiyun, A.A.; Abdu, P. and Bishu, G. (1982): Surveillance of infectious bursal diseases by precipitating antibody survey in Zaria area, Nigeria. *Journal of Animal Production Research* 2: 153 - 162.

114. Upton, E.R.P.; Hanson, R.P.; Dow, D., and Brandly, C.A. (1953): Studies on intracerebral inoculation of Newcastle disease virus (NDV) into mice I. Response of Weaning Mice to 25 Strains of *NDV. Journal of infectious diseases 92: 175 - 185.*

115. Weismann, J. and Hitchner S.B. (1978): Virus - neutralization versus agar-gel precipitation tests for detecting serological response to infectious bursal disease virus. *Avian Diseases 22: 598 - 603.*

116. Winterfield, R.W and Hitchner, S.B. (1962): Etiology of infectious nephritis - nephrosis syndrome of chickens. *American Journal of veterinary residence 23: 1273 - 1279.*

117. Winterfield, R.W. (1969): Immunity response to the infectious bursal agent. *Avian Diseases* 13: 548 - 557.

118. Winterfield, R.W and Thacker, H.I. (1978): Immune-response and pathogenicity of different strains of infectious bursal disease virus applied as vaccines. *Avian Diseases* 22: 721 - 731.

119. Yung C. (1967): A study of infectious bursal disease and its control by immunization. Unpublished Ph.D. Thesis. Aubiry University, Alabama, U.S.A. Cited by Abdu (1986) in a reprinted paper on Infectious bursal Disease, *World Poultry Science Association Journal* 3:219-230.2

APPENDICES

A: MATERIALS USED FOR FAECAL/INTESTINAL SCRAPPINGS

The materials used were glass slide/cover slips, quaze/sieve, mortar/pestle, polythene tube, Rack, funnel, centrifuge tube, glass rod, microscope, flotation media (mixture of 72% sucrose solution and 42% sodium chloride) were used for this experiment.

B: MATERIALS USED FOR THIN BLOOD SMEAR TEST (GIEMSA STAINING TECHNIQUE)

The materials used for this practicals were; bijor bottles, glass slides, methyl alcohol, staining jar, stock Giemsastain, buffer solution (PH 7.0) distilled water, wooden slide block, microscope and immersion oil were used.

C: PREPARATION OF PHYSIOLOGICAL SALINE SOLUTION (PSS) USED DURING THE PRACTICALS

Eighty-five percent (0.85g) of Nacl was weighed on a weighing machine and poured inside a conical flask, 100 mls of distilled water was added and shakened properly to dissolved in salt particles and stored. Fresh physiological saline solution (PSS) was used through the practical were necessary.

D: WASHING OF RBC

Five millilitres (5ml) of Pss was added to the settled red blood cells (RBC). The mixture was shakened properly and centrifuged at 3,000 rpm for 3 minutes (washing of rbcs). From the process of 5 ml of pss to centrifugation was repeated 3 times to make sure the rbc's were properly washed. Fresh Rbc's were used through-out the preparation of 5% Rbc.

E: HEMAGGLUTINATION TEST

Fifty micrilitres (50µl) of Pss was added into each well of labelled microtitre plate using a microdiluter calibrated 50μ litre from wells 2 to 12.

Fifty microlitres (50µl) of the diluted Lyophilized antigen (Lasota vaccine) was introduced into wells 1. The same antigen (Ag) mixture was added to well 2 from diluted lyophilized antigen bottle, using a microdiluter and shaken properly to mix.

From the mixture from well 2, 50 µl was transferred to well three, shaken and 50µ litres from well 3 was transformed to well four. This procedure was continued subsequently to well 11. 50 µl of the mixture from well II was discarded.

Fifty microlitres of the 5% washed Rbc was added to each well (wells 1-12). The mixture was incubated for 30 to 60 minutes and readings were taken. The end *point titres were recorded for each sample. Wells 1 and 12 served as negative and* positive controls respectively. The whole2 procedure was repeated for all the

samples. (As indicated in the diagram in Table 19).

The end point titre for unvaccinated birds of up to $^1/_8$ dilution is considered positive (Jordan, 1990).

F: HEMAGGLUTINATION UNIT (HAU)

The significance of the end point titre in this test identifies the hemagglutination unit (or 1HAU) of the antigen. Two wells titre dilution just before the end point titre well (table 19, see appendix H) signifies the 4HAU which is the standard for calculating the quantity of the working antigen (Ag) to be used (Allan and Grough, 1974).

G: WORKING ANTIGEN DETERMINATION

Therefore, one volume of the Ag added to one less than the 4HAU titre volume of Pss to give a total volume of the 4HAU is considered as the working Ag is the calculated quantity of the working Antigen. The marking of the working antigen dilution at 4 HAU formed the basis for hemagglutination inhibition test (HI) as described by Allan and Grough (1974).

LIBRARY

H: Table 19: Serial Dilution of Antigen

1	2	3	4	5	6	7	8	9	10	11	12
⊙	⊙	⊙	⊙	⊙	⊙	⊙	⊙	⊙	⊙	⊙	⊙
	½	¼	1/8	1/16	1/32	1/64	1/128	1/256	1/512	1/1024	1/2040
50 µl PSS + 50 µl 5% Rbc served as negative control (-ve control)			4 HAU			1 HAU					50 µl PSS + 50 µl Ag + 50 µl 5% rbc
No agglutination of Rbc but there was no settling of RBCs			50 µl serially diluted + 50 µl PSS + 50 µl 5% Rbc								Serve as positive control. There is agglutination of Rbc

* 1/8 = Agglutination at 1/8 for unvaccinated birds is considered positive (Jordan, 1990)

Ag = Antigen PSS = Physiological Saline

Ab = Antibody RBC = Red blood cells

HAU = Hemagglutination Unit

I: Table 20: Serial Dilution of Antibody (Ab) Against Newcastle Disease Virus (Lasota Vaccine)

1	2	3	4	5	6	7	8	9	10	11	12
⊙	⊙	⊙	⊙	⊙	⊙	⊙	⊙	⊙	⊙	⊙	⊙
	½	¼	1/8	1/16	1/32	1/64	1/128	1/256	1/512	1/1024	1/4096
50 µl served Ab + 50 µl Ag +50 µl of 5% rbc (positive control	50 µl PSS + 50 µl serum Ab serially diluted + 50 µl Ag + 50 µl 5% Rbc										50 µl PSS + 50 µl 5% rbc + 50 µl 5% rbc (positive control)
Settling of rbc i.e no agglutination of rbcs											There is agglutination of rbc

J: AGAR GEL PRECIPITATION TEST FOR INFECTIOUS BURSAL DISEASE

(a) Materials

The materials used were; plates, 1.25% agar, 8.0% sodium choride 0.5% phenol, buffer solution PH. 7.0), cellulose wadding pad, steamer, universal bottles, plastic petridish, pen nib, positive antigen and 1 M 8sodium hydroxide. The media were 1.25% agar (Oxoid No.1 Oxoid, UK), 8.0% sodium chloride and 0.5% phenol.

(b) Methodology

Sodium chloride and phenol were dissolved in distilled water. PH was adjusted to 7.5 using one molar sodium hydroxide. The agar was added to the mixture and steamed to dissolve, while the mixture was still very hot, it was filtered through a pad of cellulose wadding covered with a few layers of muslin. The medium was dispensed, while still hot into 15 ml volumes in universal bottles and stored at 4°C until required for use.

Plates were poured also with the still hot mixture and allowed for 24 hours to solidify before use. Each time the stored media is going to be use the agar was dissolved in a steamer and poured (15 ml into 9 cm plastic petri dish or plate). The volume of the poured agar gave the agar depth of 3 mm. After 24 hours the agar was set, plates were stored at 4°c until required for use.

Plates were poured also with the still hot mixture allowed for 24 before use to

solidify. Each time the stored media is going to be use the agar was dissolved in a steamer and poured (15 ml into 9 cm plastic petridish or plate). the volume of the poured agar gave the agar depth of 3 mm. After 24 hours the agar was set, plates were stored at 4°c if not used immediately.

Three vertical rows of wells were cut, and the agar was removed using a pen and nib. A positive antigen was incorporated into the well centres which served as control (positive control) and positive antiserum (Ab) around the positive antigen. A reaction system of Antigen (Ag) to Antibody (Ab) reaction was observed called precipitin lines or precipitation reaction.

Ab 0 0Ab
0 + Ag
0 0Ab
Ab

The same procedure was repeated for two hundred and twenty eight (228) samples collected, the test sera (T) were poured around the antigen (fig) at centre well.

T_1 0^{T2}
O

0 T = test serum
Ag Ag = positive antigen

o OT_4
T_3

All the plates were incubated in a steamer at 22°C for 24-48 hours. Recidings were taken after 24 hours, and 48 hours if necessary. Precipition lines were observed (Hirai *et al.*, 1972).

K INVESTIGATION OF LOCAL POOR WOMEN KEEPING NIGERIAN INDIGENOUS FOWLS AND THEIR ATTITUDE

A) OWNERSHIP ADDRESS: .....

DATE: .....

1. Name of Village: .....
2. Name of District: .....
3. Name of L.G.A.: .....

B) SOCIO-ECONOMIC CHARATERISTICS OF RESPONDENT

4. Ethnicity: .....
5. Religion Tick ( ) 01 Moslem ( )

02 Christian ( )

03 Others (specify)

04 NR

6. If Christian (indicate) Denomination .....

NR .....

7. Marital status 01 Married ( )

02 Divorced ( )

03 Widow ( )

04 Single ( )

04 NR

8. Type of marriage 01 Monogamous ( )

02 Polygamous ( )

9. If Polygamous 01 Number of co-wives

02 Rank in marriage

10. Age of marriage 01 Below 15 years ( )

02 15-19 years ( )

03 20-24 years ( )

04 25-29 years ( )

05 30-34 years ( )

06 35-39 years ( )

07 40-44 years ( )

08 45-49 years ( )

09 50-54 years ( )

10 55-59 years ( )

11 60 and above ( )

12 Cannot/will not say ( )

11. Indicate age last birthday (as above) .....

12. Indicate number of live children .....

13. Indicate ages of live children .....

14. Number of dead children .....

15. Can you 01 read Hausa ( )

02 write Hausa ( )

03 read English ( )

04 write English ( )

05 speak English ( )

06 NR

16. Indicate number of years completed in primary school .....

17. Indicate number of years completed in secondary school .....

18. Did you attend 01 Adult education classes ( )

02 Koranic education classes ( )

03 Agricultural education classes( )

04 Others (specify) .....

05 NR

19. Are you a member of women's association clubs? (Yes) or (No)

20. Name association/clubs in which you are a member:

01 .....

02 .....

03 .....

04 NR

21. Mention 2 predominant occupation you are engaged in during the raining season:

01 .....

02 .....

03 .....

04 NR

22. Mention 2 predominant occupation you are engaged in during the dry season:

01 .....

02 .....

03 .....

04 NR

23. Experience in poultry keeping:

01 Weeks .....

02 Months .....

03 Years .....

24. Livestock ownership Yes ( ) No ( )

25. Type of livestock and number owned .....

(C) MANAGEMENT

26. Source of birds .....

27. Flock size .....

28. Age group .....

29. Sex of birds .....

30. Housing: 01 House throughtout the day ( )

02 House only in the night ( )

31. Feeding: 01 Feeding provided in the morning only ( )

02 Feeding provided in the evening only ( )

03 Feeding provided at any time ( )

04 Feed provided throught out the day ( )

32. Type of feed: 01 brain: rice ( )

maize ( )

wheat ( )

02 grain: millet ( )

grounded grain ( )

33. Feed supplement:

01 grounded cake ( )

02 vitamins ( )

34. Feeding utensils:

01 Brokenpots ( )

02 Used plates ( )

03 Broken rubber buckets ( )

04 Constructed feeders:

wooden ( )

cement ( )

alluminium ( )

05 Broken buckets ( )

06 Old plate ( )

07 Earthen ware ( )

35. Source of water:

01 well ( )

02 tap ( )

03 gutter ( )

04 borehole ( )

05 dam ( )

06 stream ( )

07 canal ( )

08 others ( )

36. How frequently do you give water to your chicks?

01 once a day

02 twice a day

03 all day

37. If you don't provide water for your birds, where do they take water?

.....

(D) HEALTH DISEASES

38.

39. Have you ever taken the sick birds to veterinary clinic for treatment?

Yes ( ) No ( )

40. If yes, can you remember the drugs that was administered and the advice given to you? .....

41. If no, what do you give? .....

42. Do the birds have disease? Yes ( ) No ( )

43. If they don't, do you sell them? ( )

OR kill them for consumption? ( )

44. Can you remember the number of chicken you lost during the past year? .....

45. Have you ever vaccinated your birds? Yes ( ) No ( )

46. If yes, which type of vaccine? .....

47. If no, are ready to give the birds any vacine? Yes ( ) No ( )

48. How many birds died during the period which you keep the range?

.....

49. How many birds were sold? .....

50. How many birds were eaten by hawks? .....

51. How much revenue was realized? .....

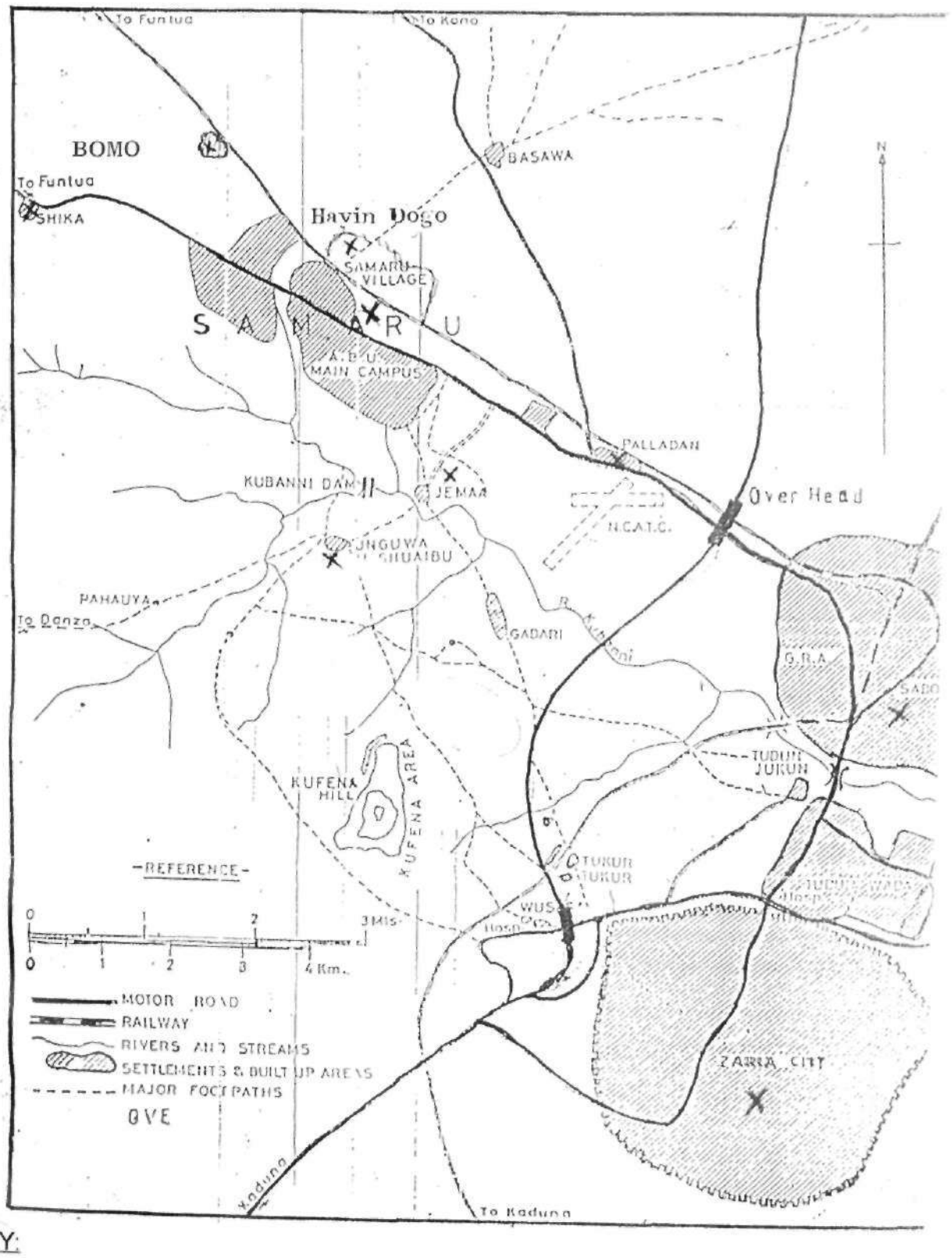
52. Is it cheaper to sell in market? ( )

OR sell at home ( )

53. Are the sick birds sold at the same price with the healthy birds?

Yes ( ) No ( )

L MAP OF NINE VILLAGES IN THE STUDY AREA IN ZARIA



KEY:

X: The villages where the interview were conducted.