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Modeling the dynamics, control and economic loss of newcastle disease in village chicken: a case of Pwani region in Tanzania

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MODELING THE DYNAMICS, CONTROL AND ECONOMIC LOSS OF NEWCASTLE DISEASE IN VILLAGE CHICKEN: A CASE OF PWANI REGION IN TANZANIA

Furaha Chuma

A Dissertation Submitted in Partial Fulfillment of the Requirements for the Degree of Doctor of Philosophy in Mathematical and Computer Sciences and Engineering of the Nelson Mandela African Institution of Science and Technology

Arusha, Tanzania

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ABSTRACT

Newcastle disease (ND) is a highly contagious viral bird disease affecting the domestic and other wild birds. The disease is a major threat to the farming of village chicken by small, medium, and large scale farmers.

In this dissertation, a non-linear deterministic mathematical model of ND to study the dynamics, control and the economic loss of the village poultry with village chicken population, wild birds population of virus in the environment is formulated and analyzed.

The basic reproduction number($\mathcal{R}_0$) which represents the number of secondary cases where one case would produce in a completely susceptible population is derived using the Next Generation Matrix technique. The bifurcation analysis of the equilibrium points shows that a model exhibits the forward bifurcation meaning that the $\mathcal{R}_0$ less than a unit is a sufficient condition to reduce the transmission of ND in village chicken population. The sensitivity analysis of the parameters in $\mathcal{R}_0$ were computed using a normalized forward sensitivity analysis, results show that the transmission coefficient of the Newcastle disease virus between the hosts and the environment is found to be the most positive sensitive parameter in the model.

A model is then extended to include three time dependent variables: vaccination, culling and the environmental hygiene and sanitation control strategies. To determine the best control strategy to mitigate the ND burden, the optimal control techniques are applied. The existence of the optimal control problem is proved with the necessary conditions for optimality determined using the Pontryagin's Maximum Principle. Numerical simulations were performed using the forward-backward sweep iterative scheme of Runge-Kutta method of order four.

Finally, a cost-effectiveness analysis is performed using the Incremental Cost-Effective Ratio (ICER). The results showed that the vaccination control strategy indicates the lowest cost compared to other control measures. The economic burden of the ND to chicken farmers, is considered as the total annual expenditure that a chicken farmer can incur to rescue the at risk chicken population from the ND is also investigated. The economic data of the model were collected from ten villages of Bagamoyo and Kibaha, Tanzania. Results from this study indicate that the recurrence of the ND in the village chicken population could lead to a serious economic loss at family level in this already financially constrained environment where small and medium farmers operate. The results obtained shows that there was 22.5% loss from their expected profit post Newcastle outbreaks in 2017. Also the results show that the occurrence of

the ND leads to an average range of $482.89 - 541.30\$$ economic loss at family in 2017. Therefore, for the effective control of NDV and its transmission we recommend vaccination to be paired with regular cleaning of chicken yards.

DECLARATION

I, Furaha Chuma do hereby declare to the Senate of Nelson Mandela African Institution of Science and Technology that this dissertation is my own original work and that it has neither been submitted nor being concurrently submitted for degree award in any other institution.

Furaha Chuma
(Candidate)

19/03/2019
Date

The above declaration is confirmed

Dr. Gasper Godson Mwanga
(Supervisor 1)

March 18, 2019
Date

Prof. Verdiana Masanja
(Supervisor 2)

21 March 2019
Date

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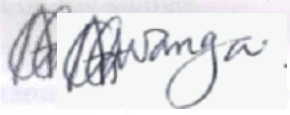
CERTIFICATION

The undersigned certify that they have read and hereby recommend for acceptance by the Nelson Mandela African Institution of Science and Technology the dissertation entitled: Modeling the Dynamics, Control and economic loss of Newcastle Disease in village chicken: A case of Pwani Region in Tanzania, in fulfillment of the requirements for the degree of Doctor of Philosophy in Mathematical and Computer Sciences and Engineering of the Nelson Mandela African Institution of Science and Technology.



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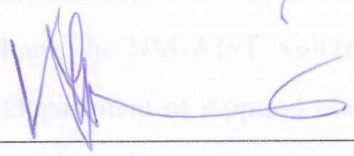

Prof. Verdiana Grace Masanja, Internal Supervisor — **Date** 21 March 2019

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DEDICATION

This work is dedicated to my lovely wife Aikaely Burton Mwanga, who has been a constant source of support and encouragement during the challenges of my studies and life in general. This work is also dedicated to our children Mary, Mark and Martin for their constant obedience to their lovely mother on my absence at home.

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LIST OF ABBREVIATIONS, ACRONYMS AND SYMBOLS

AIDS	Acquired Immune Deficiency Syndrome
AMCSE	Applied Mathematics and Computational Sciences and Engineering
APMV	Avians Paramyxovirus
BuSH	School of Business Studies and Humanity
CoCSE	College of Computational Sciences and Engineering
CBSD	Cassava Brown Streak Disease
DFEP	Disease Free Equilibrium Point
DUCE	Dar es Salaam University College of Education
EEP	Endemic Equilibrium Point
FBSM	Forward-Backward Sweep Method
GAS	Globally Asymptotically Stable
HIV	Human Immunodeficiency Virus
IAR	Infection Averted Ratio
ICER	Incremental Cost Effective Ratio
IRS	In-Door Residue Splaying
ITNs	Insecticide Treated Bed Nets
IPTp	Intermittent Preventive Treatment for Pregnant Women
LAS	Locally Asymptotically Stable
LP	Loss of Production
MCER	Marginal Cost Effective Ratio
MVL	Monentary Value Loss
ND	Newcastle Disease
NDV	Newcastle Disease Virus
NM-AIST	Nelson Mandela-African Institution of Science and Technology
ODE	Ordinary Differential Equations
PMP	Pontryagin's Maximum Principle
PC	Loss due to Prevention Costs
RK4	Runge-Kutta Method of order four
$\mathbb{N}$	A Set of Natural numbers

$\mathcal{R}_0$ Basic Reproduction Number

$\mathbb{R}^m$ A subset of Real numbers

$\mathbb{R}^n$ A field space of Real Numbers

u_1 Vaccination control variable

u_2 Culling Control variable

u_3 Environmental Hygiene and Sanitation Control Variable

CHAPTER ONE
INTRODUCTION

1.1 Background of the Study

Newcastle disease (ND) is a highly contagious viral disease affecting many domestic and wild avian species (Gilchrist, 2005; Ashraf and Shah, 2014; Brown and Bevins, 2017). The susceptibility of Newcastle disease virus (NDV) to the host depends on the isolates of the poultry groups among the avian species. The first isolate includes a group of chicken whilst the second isolate comprises of the group of other domestic and wild birds (Munir *et al.*, 2016). The effects of ND are more notable to chicken due to their high susceptibility than to other avian species (Alexander *et al.*, 2004).

The disease is caused by Avian Paramyxovirus Serotype 1 (APMV-1) virus in paramoxyviridae family (Yongolo *et al.*, 2002; Munir *et al.*, 2016) and it is a major constraint to the development of village chicken industry particularly in Africa and Asia (Otte *et al.*, 2004; Ashraf and Shah, 2014). High mortality rate of up to 90% have been documented with sometimes devastation of whole flocks during an outbreak (Yongolo *et al.*, 2002; Hugo *et al.*, 2017). ND is characterized by: coughing, head twisting, paralyzed legs and wings,greenish diarrhea, and other nervous symptoms that follow in one or two weeks (Alexander *et al.*, 2004; Oluwayelu *et al.*, 2014).

Figure 1: The clinical signs of ND (Source:https:www.agricpays.com)

However, these signs and symptoms are not pathognomonic thus it becomes hard to distinguish

the disease from other avian paramyxovirus diseases (Alexander *et al.*, 2004). The rates normally vary depending on the age of the host, virulence and the strains of the pathotypes (velogenic, mesogenic and lentogenic), susceptibility of the host, other diseases in the flock, environmental influences, and the vaccination history of the birds (Brown and Bevins, 2017). Though chickens among other domestic birds are mostly affected by the disease, young birds in a flock are extremely susceptible to disease where death rate reaches the peak of 100% (Knueppel *et al.*, 2009). Though ND is not common to human and other animals, the disease is transmissible to humans, with conjunctivitis, influenza-like symptoms being the most common clinical signs (Spradbrow, 2001; Ibitoye *et al.*, 2013).

The disease under consideration is of global importance as it could affects both poultry and humans. It is primarily posing a potential threat to village poultry farming by small and medium farmers leading to serious economic losses to an already financially constrained environment.

1.1.1 History of Newcastle Disease

The first documented outbreaks were in Java, Indonesia (1926) and in Newcastle-upon-Tyne, England in 1927 (Alexander, 2001; Kapczynski *et al.*, 2013). However; there were earlier reports of similar disease outbreaks in Central Europe before this date that wiped out all the domestic fowls in the North-West Isles of Scotland in 1896 (Macpherson, 1956). The disease is now endemic in Asia, the Middle East, Africa, Central and South America (Alexander *et al.*, 2004). Its history, origin and spread to Tanzania have not been reported but it is documented in some countries of Africa and the rest of the world (Awan *et al.*, 1994; Yongolo *et al.*, 2011).

1.1.2 Transmission Dynamics of Newcastle Disease

Although the ND is endemic in rural poultry, many aspects of its epidemiology have not fully understood. In rural environment, poultry are managed in semi-free range and/or free range system where chickens are left freely searching for food themselves. Under the free range system village chickens get the ND and primarily spreads through direct contacts of the susceptible birds with the contaminated water, food, droppings or discharges of the infected birds and other

farm utensils (Dortmans *et al.*, 2011) of the infected birds or carrier birds and other un-infected birds in their flocks. Furthermore, the carrier birds may shade NDV in their discharges and contaminate the environment. Depending on the season, the virus can survive for days in the environment, forage, water and in the bird's feathers. The . Interactions of the wild birds and the chickens when searching for food is another way that virus passes to village chicken (Alexander *et al.*, 2004; Gilchrist, 2005).

1.1.3 Poultry Industry in Tanzania

The poultry industry in Tanzania is greatly dominated by local chickens and exotic birds (breed chickens, turkeys, guinea fowl, geese, parrots, pigeons and ducks). This industry though it may contribute very little to the growth domestic product (GDP), it is possibly the most important socio-economic factor of the rural population along with subsistence agriculture (Yongolo *et al.*, 2002). It is a good enterprise for less privileged groups in villages especially women and youth who are left behind economically (Alders *et al.*, 2009). The enterprise provides them with employment, nutritious food and income depending on the number of chicken available per household (Alexander *et al.*, 2004; Alders *et al.*, 2009). In 2011, Tanzania had an estimate of 56 millions chicken, where 80% of chicken were local breeds reared traditionally by the free range system and the rest (that is, 20%) were exotic breeds (Swai *et al.*, 2011).

1.1.4 The Study Area

Pwani Region is one among the administrative Regions in Tanzania Mainland. It is located between latitude 6^0 and 8^0 South of Equator and longitude 37^030 and 40^0 East Greenwich. It borders the Indian ocean and Dar es salaam Region in East, Morogoro Region in West, Tanga Region in North and Lindi Region in South. The Region has five Districts namely: Kisarawe, Mkuranga, Rufiji, Bagamoyo and Kibaha. Our study is focusing in two Districts of Kibaha and Bagamoyo which have a total estimate of $1,817,200$ village chickens. In Bagamoyo district four villages namely: Dunda, Kibindu, Lugoba and Zinga villages were considered for the study. However, six villages namely: Gwata, Soga, Janga, Kilangala, Ruvu and Mlandizi from

Kibaha district were selected for data collection.

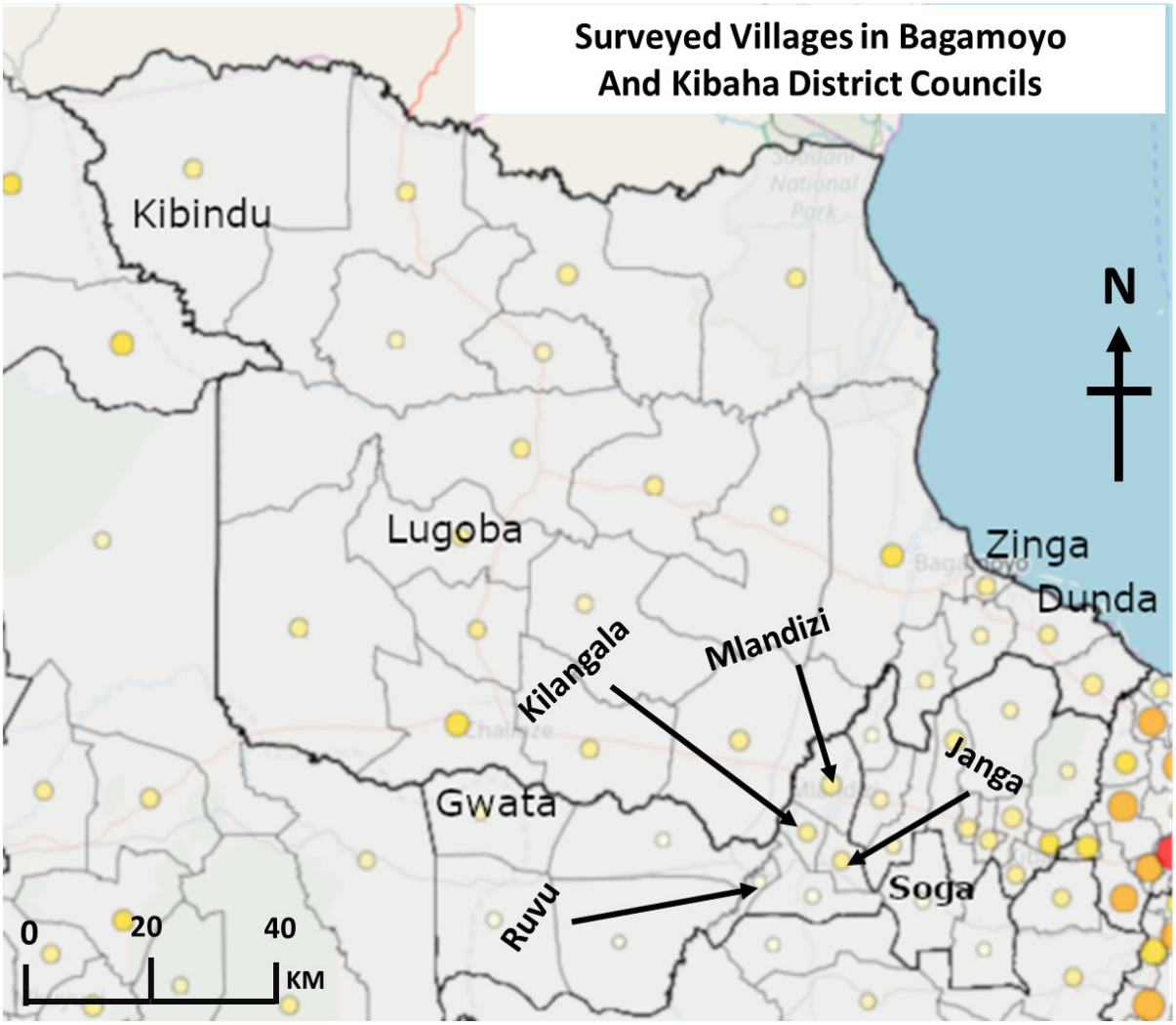


Figure 2: A map of Bagamoyo and Kibaha showing four surveyed villages in Bagamoyo and six surveyed villages in Kibaha Districts, Tanzania

1.2 Statement of the Research Problem

Different studies have been conducted on the village chicken looking at different aspects for the transmission dynamics of ND (Alexander *et al.*, 2004; Yongolo *et al.*, 2011; Rist *et al.*, 2015). Those literature have not adequately studied the transmission dynamics and control of ND with environment and wild birds reservoirs. However, interventions to reduce the spread of ND have been proposed but no study has considered the optimal control of the ND and other

poultry diseases that hinders the village poultry farming. The aim of this study is to formulate and analyze mathematical model of the ND transmission and its control in village chicken population.

1.3 Research Objectives

1.3.1 General Objective

The general objective of this study is to develop a mathematical model for the transmission, control and economic loss of ND in the village chicken.

1.3.2 Specific Objectives

The specific objectives of this study are:

(i) To formulate and analyze a basic mathematical model for ND.

(ii) To formulate and analyze a mathematical model of ND with vaccination, culling and environmental hygiene and sanitation control strategies.

(iii) To evaluate the cost-effectiveness in the control of ND.

(iv) To analyze the economic loss of ND at the family level.

1.4 Research Questions

(i) How to formulate a model for the transmission dynamics of ND with environment and wild birds reservoir?

(ii) How to formulate a transmission dynamics model of ND with an optimal control?

(iii) What is the Cost-Effectiveness in the control of ND?

(iv) To what extent does the recurrence of ND affect the economy of people at family level?

1.5 Justification of the Research Problem

The significance of this study are:

(i) Provision of mathematical framework for determination of the control strategies of the ND among the village chicken population.

(ii) Provision of understanding on the socio-economic importance of the ND as it affects both human and village chicken industry.

(iii) Provision of a platform for future researches on the transmission of ND among the village chicken.

1.6 Rationale of the Study

Understanding the transmission dynamics of the ND will help farmers, Veterinary officers and the policy makers to plan the best time for different interventions so as to reduce the possibilities for the spread of the Newcastle disease. The selected research topic aims to fill the gap that has been left behind by other theoretical and empirical studies by developing a mathematical model that shows the epidemiology of ND in the village chicken population. Identifying optimal controls of the ND will help poultry keepers and policy makers to plan for the best time and the control measures for reducing the spread of NDV. Furthermore, this study will help farmers to improve their economy by optimizing the number of poultry that can be reared per households.

1.7 Basic Mathematical Concepts

1.7.1 Dynamical System

A dynamical system $\dot{X} = \mathcal{G}(X, t)$ is a function which describes the time dependence of a point, $X \in \mathbb{R}^n$, in a geometrical space.

1.7.2 Basic Reproduction Number

The basic reproduction number $(\mathcal{R}_0)$ is defined as the average number of secondary cases caused by one infectious individual introduced in a population that consisting of entirely susceptibles (Foppa, 2005; Hartemink *et al.*, 2008; Mwanga *et al.*, 2014). This number tells and quantifies the ability of an infectious disease to invade a purely susceptible population and persist (Foppa, 2005; Hartemink *et al.*, 2008) and is measured as the spectral radius of the next generation matrix $i.e; \mathcal{R}_0 = \rho(FV^{-1})$.

1.7.3 Next Generation Method (NGM)

It is a method developed by Van den Driessche and Watmough (2002) that give brief descriptions on how to calculate the basic reproduction number $\mathcal{R}_0$. This method is applied as follows; Given a dynamical system

$$\frac{dX_i}{dt} = \mathcal{G}_i\left(X, t\right), \quad for \;\; i = 1, 2, ..., n \in \mathbb{N} \tag{1}$$

where X_i be the status of the disease in the compartment i and suppose V_i^+ and V_i^- be the rate of transfer in and out of the compartment i, respectively. It also assumes that the disease free equilibrium point of the dynamical; system is given by ϕ_0. Therefore,

$$\frac{dX_i}{dt} = F_i\left(t\right) - V_i\left(t\right), \qquad \text{where,} \;\; V_i = V_i^- - V_i^+ \tag{2}$$

Then

$$F = \frac{\partial F_i\left(\phi_0\right)}{\partial t}, \quad V = \frac{\partial V_i\left(\phi_0\right)}{\partial t} \tag{3}$$

and lastly the basic reproduction number is found as the spectral radius of the Next Generation Matrix (NGM) written as;

$$\mathcal{R}_0 = \rho\left(FV^{-1}\right) \tag{4}$$

1.7.4 Metzler Matrix

The real square matrix $M = [m_{ij}] \in R^{n\times n}$ is called the Metzler matrix if its all off-diagonal entries are nonnegative, i.e. $m_{ij} \geq 0, i \neq j$.

1.7.5 Lipschitz condition

Let $(\mathcal{Z}, \|.\|)$ be a normed linear space, A dynamical system $\mathcal{G}(t, X(t)) : \mathcal{Z} \to \mathcal{Z}$ is said to be Lipschitz if $\exists \mathcal{K} \leq 0$ for which the Lipschitz condition $\frac{\|\mathcal{G}(X_1)-\mathcal{G}(X_2)\|}{X_1-X_2} \leq \mathcal{K}$ is satisfied for all pairs $X_1, X_2 \in \mathcal{Z}$, $X_1 \neq X_2$. The bound $\mathcal{K}$ is called a Lipschitz constant for $\mathcal{G}$.

1.7.6 The Optimal Control Theory

The optimal control theory is a mathematical tool that helps the designing of the optimization systems which are influenced by external factors to be controlled (Sadiq *et al.*, 2014; Kahuru *et al.*, 2017*b*; Hugo *et al.*, 2017). The theory helps to describe different external factors of complex models and provide control measures by analyzing the necessary conditions of optimal control using the Pontryagin's maximum principle (Lenhart and Workman, 2007; Kahuru *et al.*, 2017*b*). The theory was developed by Lev S. Pontryagin $(1968 - 1988)$ and his co-workers and over decades has been used for the analysis of the optimality of the solutions in different complex mathematical models from biological sciences (Lenhart and Workman, 2007; Schńttler and Ledzewicz, 2012; Kahuru *et al.*, 2017*b*).

Optimal Control theory as a mathematical tool has different procedures and/or ways of reaching the optimality of the desired problem. Lets consider the controlled dynamical system:

$$\begin{cases} \dot{X}(t) = \mathcal{G}(t, X(t), u(t)); \quad t > 0 \\ X(0) = X_0, \quad X(T) = X_T \end{cases} \tag{5}$$

According to the dynamical system (5), $X(t)$ refers to the state variable in a specified time t, X_0 is the initial condition of the state variables, X_T is the final condition of the state variable and $u(t)$ refers to time dependent control parameter. This model system is a continuous dynamical system and is governed by the set of non-linear ordinary differential equations (ODEs) under a fixed and/or free time interval. The state variable $X(t)$ is enclosed in the Euclidean space $\{X(t) \in \mathbb{R}^n : n \in \mathbb{N}\}$ and the control $u(t)$ variable is Lebesgue measurable $i.e$ $\{u(t) \in \mathcal{U} \in \mathbb{R}^m : 0 \leq u(t) \leq T\}$. The controls affects the dynamical system with the main purpose of minimizing or maximizing the cost functional. We minimize the cost function, $\mathcal{J}(u(t))$ by finding the primal control variable u^* such that,

$$\mathcal{J}\left(u_i^*\right) = \min_{u_i \in \mathcal{U}} \left\{\mathcal{J}\left(u_i\right)\right\}; for \;\; i = 1, 2, .., n \in \mathbb{N} \tag{6}$$

1.7.7 Optimal Problem

A controlled system is an optimal problem $\dot{X}\left(t\right) = \left(X\left(t\right), \mathcal{U}, \mathcal{G}\right)$ consisting of a state space $X\left(t\right)$, a control set $\mathcal{U}$, and the dynamics $\mathcal{G}$ (Schńttler and Ledzewicz, 2012). Throughout the dissertation we use the following notations for the data defining the optimal problem (5);

(i) The state space $X\left(t\right)$ is an open and connected subset of $\mathbb{R}^n$.

(ii) The control set $\mathcal{U}$ is a subset of $\mathbb{R}^m$

1.7.8 The Cost Function

The cost or objective function is a mathematical equation describing the production output that corresponds to the maximization or minimization of the target with respect to the optimal problem and the initial condition such that;

$$Maximize/Minimize \;\; \mathcal{J}\left(t, X, u\right) = \int_{t_0}^{t_f} \left\{\mathcal{G}\left(t, X\left(t\right), u\left(t\right)\right)\right\} dt \tag{7}$$

subject to the state equation;

$$\dot{X}\left(t\right) = \mathcal{G}\left(X\left(t\right), u\left(t\right)\right) \tag{8}$$

and the initial and terminal conditions in (5)

$$X \in \mathbb{R}^n : X\left(t_0\right) = X_0, \;\; X\left(t_f\right) = X_T; \;\; t \in \left[t_0, t_f\right] \tag{9}$$

where $u\left(t\right)$ is the control variable and t_f stands for the final time on the control trajectory.

A state variable $X\left(t\right)$ is an open and connected subset of the Euclidean space $\mathbb{R}^n$ that characterize the behavior of the dynamical system at an instantly time t. A control set is a set of points characterized by $u\left(t\right) \in \mathcal{U} \in \mathbb{R}^m$, $m \in \mathbb{N}$. A control variable $u(t)$ is said to be an admissible control if it is piecewise continuous defined on some time interval $t_0 \leq t \leq t_f$ with range in the control region $\mathcal{U}$, $u(t) \in \mathcal{U}$, $\forall t \in t_f$.

1.7.9 Equilibrium Point

Let $\mathcal{D} \in \mathbb{R}^n$ and $f : \mathcal{D} \mapsto \mathbb{R}^n$ be a nonlinear vector field. Then any point $\bar{X}$ that satisfies the condition $f\left(\bar{X}, t\right) = 0, \forall t > 0$ is an equilibrium point of the system f (Hunter, 2011; Selemani *et al.*, 2016; Olaniyi *et al.*, 2016).

1.7.10 Positive Invariant Solution

$\mathcal{D}$ is a positively invariant set for a dynamic system $\dot{X} = \mathcal{G}(t, X(t))$ if every trajectory $X(t)$ which starts from a point $X(0) \in \mathcal{D}$ remains in $\mathcal{D}$, $\forall t > 0$.

1.7.11 Optimal Trajectory

An optimal trajectory (X^*) refers to the set of constraints which its performance satisfies the condition of minimizing or maximizing the cost function $\mathcal{J}\,(t, X, u)$.

1.7.12 Optimal Solution

An optimal solution is a feasible solution of the optimal problem where the cost function reaches its minimum or maximum value. For the case of the minimization problem, a solution (t^*, X^*, u^*) is optimal if $\mathcal{J}\,(t^*, X^*, u^*) \leq \mathcal{J}\,(t, X, u)\ for\ all\ admissible\ \ (t, X, u)$. u^* is the optimal control variable which gives an optimal trajectory X^* of the system (5).

1.7.13 Hamiltonian Function

According to Poggiolini and Spadini (2011), Schńttler and Ledzewicz (2012) and Mwanga *et al.* (2014), the Hamiltonian function $\mathcal{H}$ of the optimal control problem is defined as

$$\mathcal{H} : \mathbb{R} \times [0, \infty) \times \mathbb{R}^n \times \mathbb{R}^m \to \mathbb{R} \tag{10}$$

with

$$\mathcal{H}(t, X, u, \lambda) = L(t, X, u) + \lambda f(t, X, u). \tag{11}$$

where $L(t, X, u)$ is the Lagrangian function and $\lambda = \lambda(t)$ stands for the adjoint or co-state variable of the function. X and u are real-valued functions on $[t_0, t_f]$ with values in $\mathbb{R}^n$ and $\mathbb{R}^m$ respectively. The adjoint or Co-state variable $\lambda(t)$ is a variable in the Hamiltonian function which is used for optimizing the solution of the controlled problem.

1.7.14 Pontryagin's Maximum Principle (PMP)

The Pontryagin's Maximum Principle (PMP) states the necessary conditions that an optimal trajectory of the optimal control problem must hold (Evans, 1983; Anita *et al.*, 2011). The optimality of a solution is reached when all of the necessary conditions are fulfilled in a way that an optimal solution exists and is unique (Anita *et al.*, 2011; Schńttler and Ledzewicz, 2012). By considering a control system in equation (5), the PMP necessary conditions holds only if there exists an adjoint variable $\lambda(t)$ together with the state variables $X(t)$ and the optimal control $u(t)$ such that in terms of the Hamiltonian $\mathcal{H}$, the adjoint condition, transvesality condition, and the optimal condition holds.

Theorem 1.1

Pontryagin's Maximum Principle.

Let (t, X^*, u^*) be a controlled trajectory defined over the interval $[t_0, t_f]$ with the control u^* piecewise continuous. If (t, X^*, u^*) is optimal, then there exist an adjoint or a co-state variable $\lambda(t)$ such that the following conditions are satisfied:

(i) Non-triviality of the multipliers: $(\lambda(t)) \neq 0$ for all $t \in [t_0, t_f]$.

(ii) The adjoint variable $\lambda(t)$ is a solution to the time-varying linear differential equation

$$-\dot{\lambda}(t) = \mathcal{H}_X\left(X^*(t), \lambda(t), u^*\right) \tag{12}$$

where X stands for the state variables in the model.

(iii) The Transvesality condition: the final point of the controlled trajectory.

$$\lambda_i(t_f) = 0 \quad for \quad i = 1, 2, ..., r, \quad r \in \mathbb{N} \tag{13}$$

(iv) The Optimal condition

$$\dot{\mathcal{H}_{u_j}} = 0 \quad for \quad j = 1, 2, ..., n, \quad n \in \mathbb{N} \tag{14}$$

The Pontryagin's Maximum Principle is stated depending on the following:

(i) The time or source of the desired dependent time

(ii) Dimension and regularity of the source of the desired source

(iii) The cost containing only the final part, the running part or both

(iv) The time being fixed or free

After the formulation of the optimal cost function, then the existence of the control variable is proven. Thereafter the principle is used to characterize the control variables where an optimal solution of the model is obtained.

1.7.15 Forward-Backward Sweep Method (FBSM)

The Forward-Backward Sweep method (FBSM) is the indirect technique for solving numerically optimal control problems (McAsey *et al.*, 2012; Mwanga *et al.*, 2014). FBSM has the following successive steps;

(i) The total time is divided into N sub-intervals irrespectively to the state $\overrightarrow{X} = (X_1, X_2, ..., X_{N+1})$ and the Co-state variables as $\overrightarrow{\lambda} = (\lambda_1, \lambda_2, ..., \lambda_{N+1})$

(ii) The controls are assumed to take zero values for starting an iteration such that $\overrightarrow{\mathcal{U}} = [0, 0, .., 0]$.

(iii) With the initial condition $X(0) = X_0$, the state solutions in the ODE with the controls are solved forwardly by using the forward in time Runge-Kutta method of order four.

(iv) With the transvesality condition $\lambda_{N+1} = \lambda(t_f)$ where t_f is a final time, the values for $u(t)$ and $X(t)$ from the Co-state differential equation are solved by the backward in time Runge-Kutta method of order four.

(v) The update of the control is done by entering the new X and λ through the rule

$$u^* = \min\{u_{\max}, \max(u_{\text{sig}}, u_{\min})\} \tag{15}$$

where the boundedness of controls is defined as;

$$u^* = \begin{cases} u_{\min} & \text{if} \quad \frac{\partial \mathcal{H}}{\partial u} < 0 \\ u_{\min} < u_{\text{sig}} < u_{\max} & \text{if} \quad \frac{\partial \mathcal{H}}{\partial u} = 0 \\ u_{\max} & \text{if} \quad \frac{\partial \mathcal{H}}{\partial u} > 0 \end{cases} \tag{16}$$

(vi) If the last preceding iterations are negligible close such that $\frac{|X_{N+1} - X_{N-1}|}{|X_N|} < \epsilon$, then the last iteration is the complete solution otherwise return to step (iii) above.

1.7.16 Runge-Kutta Method (RK4)

The 4^{th} order Runge Kutta method is a built in MATLAB software used to approximate the solution to the first order differential equation (ODE's) of the form;

$$\frac{dX}{dt} = \mathcal{G}(X(t), t); \quad X(t_0) = X_0 \tag{17}$$

The 4^{th} order Runge Kutta scheme starts when an initial value of the function is given to start the algorithm. When $h > 0$ takes the algorithm to;

$$\begin{aligned} X_{n+1} &= X_n + \frac{h}{6}[k_1 + 2k_2 + 2k_3 + k_4], with \quad n = 1, 2, ... \\ k_1 &= \mathcal{G}(X(t_0), t_0) \\ k_2 &= \mathcal{G}\left(X(t_0), t_0 + k_1\frac{h}{2}, t_0 + \frac{h}{2}\right) \\ k_3 &= \mathcal{G}\left(X(t_0), t_0 + k_2\frac{h}{2}, t_0 + \frac{h}{2}\right) \\ k_4 &= \mathcal{G}(X(t_0), t_0 + k_3 h, t_0 + h) \end{aligned} \tag{18}$$

where as; k_1 describes the slope of the differential equation $\frac{dX}{dt}$ at the beginning of the first time $t = t_0$, k_2 at the mid-point at the time step $t = t_0 + h/2$ using the value of k_1, k_3 half way through the mid point at the time step $t = t_0 + h/2$ using the value of k_2 and k_4 estimates the slope of the function at the end point using time interval $t = t_0 + h$ and the value of k_3.

1.7.17 Convergent Criterion of Ordinary Differential Equation (ODE)

A steady state solution $X^*(t)$ of a dynamical system $\dot{X} = \mathcal{G}(X, t)$ is stable if, for any arbitrarily small $\epsilon > 0$, $\exists \delta > 0$ such that, for any trajectory $X(t)$ for which $\| X(0) - X^*(0) \| < \delta$, then the inequality $\| X(t) - X^*(t) \| < \epsilon$ is satisfied $\forall t > 0$ (Anishchenko *et al.*, 2014). According to Tumwiine *et al.* (2010); Selemani *et al.* (2016) and Wiggins (2003), a steady state $X^*(t)$ is stable iff all initial trajectories in an open set $X \in \mathbb{R}^n$ moves towards $X^*(t)$ and remain near it $\forall t \geq 0$ and is unstable if moves away from $X^*(t)$.

1.7.18 The Outline of the Dissertation

In this work, the review of the related literatures is done in Chapter two. The Chapter covers the review of previous works in the dynamics of ND, Optimal Control Theory as well as the cost effectiveness on various disease transmission models.

Chapter three of this work covers the formulation of the ND basic transmission model, its analysis on the basic properties of the model to include but not limited the invariant region, positivity and the equilibrium points of the model. However, the Chapter covers the area of computation of the basic reproduction number, the sensitivity of the model parameters, stability analysis of the equilibria of the model and last covers the simulation of the basic model.

Chapter three is then extended to include the control variables. In this part, vaccination, culling and the environmental hygiene and sanitation control variables are added to the basic model followed with its analysis to investigate the best countermeasure for limiting the spread of the ND among the village chicken. Then followed with the Cost-Effectiveness analysis which is done by using the Increment Cost-Effectiveness Ratio (ICER) method. The Chapter also covers

the analysis of the economic loss of the ND to village chicken farmers at a family level.

Chapter four of this work covers the methods and findings followed with the conclusion and recommendations for future works.

CHAPTER TWO
LITERATURE REVIEW

This section review and discuss literatures on ND ranging from clinical and theoretical studies. It also cover areas of mathematical modeling for poultry diseases, explore intervention strategies for control of different diseases, optimal control theory as well as the cost- effective techniques for the control of infectious disease.

Yongolo *et al.* (2002) conducted a study in Morogoro and Tabora Regions, involving ducks and village chicken. In each region, one district with five randomly selected villages was considered for the study. The aim was to study the ND and Infectious bursal disease (IBD) among free-range village chicken in Tanzania. Standardized questionnaires were used for data collections among chicken farmers. In the study, the confirmation of the NDV was achieved through isolation of the virus, the clinical and pathological signs and the characterization of the field virus strains. However, through the isolations of the virus and the serological survey, it was discovered that ND is a seasonal poultry disease that occurs between June and October. The APMV-1 (serotype-1) among other serotypes was identified as the main causative of the ND. Also the isolations of NDV among the domestic ducks revealed the role played by the ducks in the epidemiology of ND in the free-range system in Tanzania. The study recommended the need on the control of ND before the active period of NDV in June while taking considerations on age groups of the chicken. Also the study recommended the need on researching for other poultry diseases and risk factors for reducing high mortality rates in chicken.

Alexander *et al.* (2004) reviewed the transmission dynamics of ND among local chicken as one of the constraints on increasing the small-scale poultry production. The review covered the origin and nature of ND, its characteristics, epidemiology, symptoms and its control. The study aimed on making a reference and platform for the control of ND in developed countries. However, a study pointed out the avian paramyxovirus 1 (APMV-1) virus as the main cause of ND in indigenous chicken. The study also revealed that the prevalence of ND in local chicken or backyard flocks in many countries are not well documented which sometimes become hard to trace the occurrence of the disease in those areas. Also the study shows the role of wild birds in the spread of the disease and its pathogenic varies widely depending on the virus,

age of the host, the host species and the immune state of the host. Also, the review revealed that the spread of the NDV was spotted to be caused by live birds for trading, movement of people and equipments, poultry products, contaminated food or water, air borne spread and non-avian hosts such as rodents, insects or scavenging animals. On the other hand, the biosecurity and hygiene in the control of the ND was highlighted since its occurrence for the first time.in Europe. The study revealed that live lentogenic, live mesogenic and inactivated vaccines have been developed and applied in some countries for controlling the spread of ND. However, the choice of vaccine in backyard chicken revealed to depend on their cost, the nature of service providers, past experience, the climatic condition and the population distribution of chicken. Lastly, the study pointed out the live vaccine to be less costly especially when produced locally than the inactive vaccines.

McDermott *et al.* (2001) studied the role of improving the control of ND in southern Africa. Both epidemiological and economic data were used to predict relative control of ND among local chicken. A mathematical model for the transmission of ND in local was developed. The developed model was the extension of the model for foot and mouth disease in Thailand (Perry *et al.*, 1999). The model was developed in the assumption that the transmission of ND to commercial sector has different roots and possibility of infections. Also a model assumed that no compartment of the recovered chicken after being attacked by the ND. Economic analysis of ND and its control was also carried out. The study revealed that in order for the vaccination of ND to be active it must be conducted frequently in a large population of the local chicken.

Daut *et al.* (2016) developed two mathematical models aimed on showing the influence of illegal harvest and effects of age structurer of the wild wingled-Parakeets on the dynamic of ND. Interactions of ND transmission and harvest were evaluated through their basic reproductive numbers and the population dynamics of the wingled-Parakeets in a short time. The findings showed the relationship between the introduction of ND in the Parakeets population with its mortality. The results show that the population decreases up to its total population in two years. However much harvest shows to reduce the spread of the ND in the Parakeets population. But the second case showed a slight difference that means the age of the Parakeets can influence the spread of ND though not in a very great extent.

Rist *et al.* (2015) established a mathematical model to show the effects of poultry diseases on the economy of people living in rural Madagascar. A proposed model have three sub models; the epidemiological model that consists of poultry compartments, the income generation model and the simple economic model. The coupled ecology-economic model was built in a sense that poultry diseases are economical drivers on the people in rural Madagascar. Data for parameterize the model were collected from 1520 households and from the demographical survey where 80 households pilot study was conducted on livestock health. Equilibrium monthly household income was used to estimate the mean burden of disease as the percent of income lost to disease for a range of potential transmission rates. The results from the sensitivity analyses were included in the burden estimation to account for the high uncertainty in model parameter values. Based on the Latin Hyper-cube sampling method, 1000 simulations were run, each with different combination of parameters, at transmission rates from 0 to 1. An exponential increase in the economic burden is observed at transmission rates below 0.4 while the burden approaches a fixed value at higher transmission rates. The majority of simulations with a transmission rate predicted a $10 - 25\%$ loss of monthly income. PRCC results suggested that in the presence of poultry disease, both economic and epidemiological parameters highly influenced the outcomes of the model.

Hugo *et al.* (2017) formulated a deterministic compartmental eco-epidemiological model with the optimal control of ND. The model has human and chicken as its populations. It incorporated three control strategies; vaccination, human education campaign and treatments of the infected human. Necessary conditions of optimal control were analyzed with the Pontryagin's maximum principle. The cost effectiveness analysis techniques were employed and found that the combinations of chicken vaccination and human education strategies are the best strategies to be applied in the scarcity of the resources.

Seidu and Makinde (2014) formulated an optimal control model with the efforts of reducing HIV/AIDS infections, irresponsibilities and non productivity in the work place. The model incorporate four interventions aimed at reducing the spread of the disease in the working places. The interventions include the efforts for reducing the infections of the susceptible individuals, efforts for treating the infected individuals, control efforts at changing the behavior of people around and the efforts aimed at reducing non productivity at the working places.

Kahuru *et al.* (2017*b*) applied an optimal control techniques to minimize the number of the infected human, animals and the sand flea population in the dynamics of Tungiasis. In this work, five control strategies are incorporated in the model as part of the efforts to reduce the spread of the disease and the cost of the control among the human and animals populations.

Blayneh *et al.* (2009) applied the optimal control theory to study the effects of prevention and treatment for the control of a malaria disease while reducing the cost of control. the results shows that there are cost effective control efforts for treatment of the infectives as well as the prevention of host-vector contacts.

Wang and Modnak (2011) developed a mathematical model of cholera dynamics. In this model, three control namely: vaccination, theraupetic treatment and the water sanitation were included. They applied the optimal control techniques aiming at minimizing the number of the infected people as well as the cost of controls over a short period of time. The analysis of their model showed that the combination of the multiple strategies is the the accurate measure to achieve the optima; control of cholera.

Kim *et al.* (2012) used a deterministic differential equations to develop a plasmodium vivax malaria model with the control terms. They performed the analysis and its numerical solutions. Finally they suggested that the use of mosquito reduction strategies is more effective than the personal protection.

Okosun *et al.* (2013) used a mathematical approach to study the cost-effectiveness on the controls towards the prevention of malaria. In their model; the use of insecticide sprays, treating of the infective human and the use of bed nets preventive measures were involved. They calculated the Infection Averted Ratio (IAR) and then used the Incremental Coast-effectiveness Ratio (ICER) to investigate the most cost-effective strategy for the control of malaria. In this work, the combination of the insecticides splay and treating of the infected human have found a cost-effective strategy above all.

Athithan and Ghosh (2015) formulated a non-liner mathematical model of malaria and extended it to an optimal problem. They used the Pontryagin's Maximum Principle to find the optimality of the control. Simulation of the extended model shows better results than the model without

control.

Kinene *et al.* (2015) developed an optimal model to study the control and the cost effective intervention of the cassava brown streak disease (CBSD). In this model, two time dependent intervention strategies were included. The Pontryagin's maximum principle was used to establish the necessary conditions for the control of the CBSD. They also used the Incremental cost-effectiveness Ratio (ICER) to analyze the cost effectiveness of the control strategies. They concluded that, uprooting and burning of the infected plants is more cost effective than the application of the combination of the chemical spray and uprooting of the infected plants.

Otieno *et al.* (2016) formulated a deterministic malaria transmission model which includes human and mosquito populations for controlling malaria disease in Kenya. Four time dependent control variables namely; the use of Insect treated bed nets (ITNs), treated of infective human, spray of insect sides and treated of pregnant women were included in the model. The aim of this model was to find which strategy is effective and cost benefit. The cost effective analysis was done using the Incremental cost effective ratio (ICER),The analysis showed that, in the endemic regions the combination of insect treated nets (ITNs), indoor residual sprays (IRS) and Intermittent Preventive Treatment for Pregnant Women (IPTp) is the most effective for malaria prevention and control.

To the best of my knowledge, there is no any study applied the optimal control Theory and the Incremental Cost-Effectiveness Ratio (ICER) to study the dynamics and control of ND by considering the local poultry farming. This study is therefore design and analyze a mathematical model to study the transmission dynamics and the control of ND among the local poultry farming. The economical burden of the ND at the household level is also studied.

CHAPTER THREE
MATERIALS AND METHODS

In this section, a basic model of ND transmission is formulated based on the idea that wild birds and the environment are primary reservoirs of NDV (Alexander *et al.*, 2004; Gilchrist, 2005; Nwanta *et al.*, 2008; Martin and row, 1992; Lawal *et al.*, 2015). A system of ODEs was considered to represent the parameters and change of state variables in the non-linear deterministic mathematical model. The model is formulated and analysed qualitatively and numerically. The basic model is then extended by incorporating vaccination, culling and the environmental hygiene and sanitation control strategies. The purpose is to study the dynamics of ND and investigating the impact of the controls for reducing its transmission.

3.1 The Basic Model of Newcastle Disease

The village chicken population $N_c(t)$ is divided into three subpopulations namely: $S_c(t)$ that represents a number of susceptible village chicken, $E_c(t)$ that represents a number of exposed chicken in the population, $I_c(t)$ that represents number of village chicken in the population which is severe infected from the infection. The total population size of village chicken is denoted by $N_c(t) = S_c(t) + E_c(t) + I_c(t)$.

The wild birds population $N_b(t)$ is divided into four sub-populations as follows: $S_b(t)$ are susceptible wild birds; $E_b(t)$ are exposed population of wild birds; $I_b(t)$ the severe infected wild birds population and $I_r(t)$ are the mild infected wild birds population. The total population size of wild birds is therefore denoted by $N_b(t)$ with $N_b(t) = S_b(t) + E_b(t) + I_b(t) + I_r(t)$ and the environment has only one compartment denoted by $H(t)$.

The village chicken population is recruited by the density dependent recruitment rate μN_c through birth. Initially, village chicken acquires NDV when a sick village chicken is introduced in a flock or environment and come into contact with other un-affected chicken. Village chicken can also acquire NDV when exposed into unhygienic environment as well as when interacting with other mild infected wild birds (Lawal *et al.*, 2015; Daut *et al.*, 2016). Village

chicken spread the NDV after developing the clinical signs within the incubation period of two to fifteen days (Perry *et al.*, 1999; Alexander *et al.*, 2004; Sharif *et al.*, 2014). Chicken acquire infections at a rate $\beta_c\left(I_c, I_r, H\right)$ which is defined by:

$$\beta_c\left(I_c, I_r, H\right) = \left(\psi\frac{I_c}{N_c} + b\frac{I_r}{N_b} + d\frac{H}{\kappa + H}\right) S_c \tag{19}$$

where ψ is the transmission coefficient between the infected village chicken and susceptible population of the village chicken, b is the transmission coefficient between the mild population of wild birds and the susceptible village chicken, d is the coefficient transmission constant rate of NDV with the hosts when come into contact with the unhygienic environment. The parameter $kappa$ is the saturation constant rate of NDV in the environment. The ratio

$$d\frac{H(t)}{\kappa + H(t)} \tag{20}$$

is the density of NDV in the environment which gives the great chance for the disease outbreak (Martin and row, 1992; Nwanta *et al.*, 2008). After few days, chicken starts to show aerosol signs and progress to chronic stage of the disease at the rate $\gamma E_c(t)$. We assume that village chicken cannot recover from disease but they die naturally at a rate μ and by the disease induced death rate δ_c.

Wild birds are assumed to be recruited by the density dependent recruitment rate μN_b through birth and migrations. Like the village chicken, the susceptible wild birds gets NDV from the contaminated environment as well as when interacts with the severe infected and mild infected wild birds population at the rate $\beta_b\left(I_b, I_r, H\right)$ defined by:

$$\beta_b\left(I_b, I_r, H\right) = \left(\frac{\varphi I_b + aI_r}{N_b} + d\frac{H}{\kappa + H}\right) S_b \tag{21}$$

where φ is the transmission coefficient between the chronically infected population of wild birds and the susceptible wild birds and a is the transmission coefficient between the mild infected wild birds and the susceptible wild birds. Wild birds are resistant to the ND which makes them to be carries of the virus (Awan *et al.*, 1994; Brown and Bevins, 2017). Therefore the progress of the ND in the wild birds leads to two infected subpopulations; severe infected, $I_b(t)$ and the mild population, $I_r(t)$ at the proportions of ρ and $1 - \rho$ respectively. It is assumed that wild birds cannot recover from the disease once affected but they are reduced by natural death μ and

others by the disease induced death at the rate δ_b. NDV are introduced in the environment by the severe infected village chicken, severe infected wild birds and the mild infected wild birds through shedding at the rate α_c and α_b respectively (Awan *et al.*, 1994; Nwanta *et al.*, 2008). The NDV can survive for some months at a temperature between $20 - 30^0C$ and much longer at cooler physical environments (Martin and row, 1992). The model is formulated with the following assumptions;

(i) The contaminated environment with NDV, the Infected Village chickens and the wild birds reservoirs are the primary sources of the NDV infections to the village chicken (Nwanta *et al.*, 2008; Lawal *et al.*, 2015; Brown and Bevins, 2017).

(ii) The environment is considered to carry only active viruses (Mesogenic, Lentogenic and the velogenic) during the outbreak of the ND.

(iii) Wild birds are reservoir of the ND strains and can be maintained for a long period.

(iii) Susceptible population of village chicken can get NDV by either through direct contact with an infected Village chicken or from mild and severe infected wild birds and the environment (forage and water).

(iv) Neither age structure nor vertical transmission is considered in building the model.

(v) severe infected wild birds, the mild infected wild birds and the infected village chicken contaminate the environment through shading of the NDV (Awan *et al.*, 1994; Nwanta *et al.*, 2008).

(vi) Both village chicken and wild birds cannot recover from the ND once infected.

(vii) All avians have the same shedding rate of virus into the environment.

The parameters and the model state variables used in the formulation of the ND model are summarized in tables below:

Table 1: Descriptions of the Model State Variables used in the formulation of the Model

Variable	**Description**
$S_c(t)$	Susceptible village chicken population
$E_c(t)$	Exposed village chicken population
$I_c(t)$	Infected village chicken population
$S_b(t)$	Susceptible wild birds population
$E_b(t)$	Exposed wild birds population
$I_b(t)$	Severely infected wild birds population
$I_r(t)$	Mildly infected wild birds population
$H(t)$	NDV population in the surroundings
$N_c(t)$,	Total population of village chicken
$N_b(t)$	Total population of wild birds

Table 2: Descriptions of Parameters used in the formulation of the Model

Parameter	Description
a	Transmission coefficient between the mild population of wild birds and the susceptible population of wild birds
b	Transmission coefficient between between mild population of wild birds and the susceptible population of the village chicken
ψ	Transmission coefficient between the severe infected and the susceptible population of village chicken
κ	Half saturation constant of NDV in the environment
d	Contact rate between susceptible populations of village chicken and wild birds with the environment
ρ	Proportion of the exposed wild birds which become chronically infected with NDV
α_b	Shading rate of NDV in the environment by chronically infected and the carrier wild birds
α_c	Shading rate of NDV in the environment by the chronically infected village chicken
β_c	Force of infection among the village chicken population
β_b	Force of infection among the wild birds population
φ	A transmission coefficient between severely infected and the susceptible wild birds population
μ	Natural mortality death of the host populations
μ_v	Clearance rate of the NDV from the environment
δ_b	Disease induced death rate in wild birds populations
δ_c	Disease induced death rate in the village chicken population
γ	Progression rate of the disease in the host populations

3.1.1 Model Flow Diagram

Based on the Transmission Dynamics of the ND, Model assumptions, definition of variables and parameters respectively, the dynamics of the ND is summarized in the flow diagram as follows:

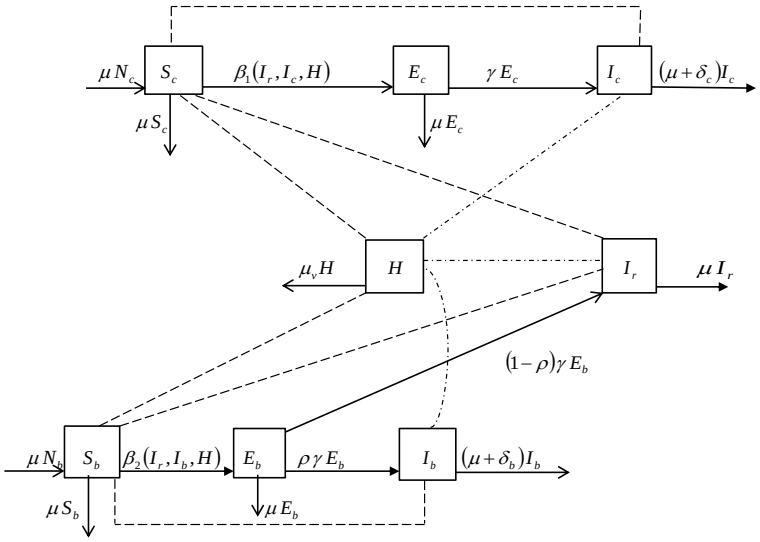


Figure 3: Compartment model diagram for the Transmission Dynamics of Newcastle disease in village chicken population. The sold lines show the constant transmission from one compartment to another, the dotted lines show the normal interactions between different compartments and a dash dot lines represent the shedding of NDV onto the environment

.

3.1.2 Equations of the Model

Now using model assumptions discussed before, the dynamics of ND is described by the following systems of nonlinear differential equations:

Chicken

$$\frac{dS_c(t)}{dt} = \mu N_c(t) - \left(\psi\frac{I_c(t)}{N_c(t)} + b\frac{I_r(t)}{N_b(t)} + \frac{dH(t)}{\kappa + H(t)} + \mu\right) S_c(t) \tag{22a}$$

$$\frac{dE_c(t)}{dt} = \left(\psi\frac{I_c(t)}{N_c(t)} + b\frac{I_r(t)}{N_b(t)} + \frac{dH(t)}{\kappa + H(t)}\right) S_c(t) - (\mu+\gamma)E_c(t) \tag{22b}$$

$$\frac{dI_c(t)}{dt} = \gamma E_c(t) - (\delta_c + \mu)I_c(t) \tag{22c}$$

Wild birds

$$\frac{dS_b(t)}{dt} = \mu N_b(t) - \left(\frac{\varphi I_b(t) + aI_r(t)}{N_b(t)} + \frac{dH(t)}{\kappa + H(t)} + \mu\right) S_b(t) \tag{23a}$$

$$\frac{dE_b(t)}{dt} = \left(\frac{\varphi I_b(t) + aI_r(t)}{N_b(t)} + \frac{dH(t)}{\kappa + H(t)}\right) S_b(t) - (\mu + \rho\gamma)E_b(t) \tag{23b}$$

$$\frac{dI_b(t)}{dt} = \rho\gamma E_b(t) - (\delta_b + \mu)I_b(t) \tag{23c}$$

$$\frac{dI_r(t)}{dt} = (1-\rho)\gamma E_b(t) - \mu I_r(t) \tag{23d}$$

Environment

$$\frac{dH(t)}{dt} = \alpha_c I_c(t) + \alpha_b \left(I_b(t) + I_r(t)\right) - \mu_v H(t) \tag{24}$$

With initial conditions,

$S_c(0) > 0$, $E_c(0) \geq 0$, $I_c(0) \geq 0$, $S_b(0) > 0$, $E_b(0) \geq 0$, $I_b(0) \geq 0$, $I_r(0) \geq 0$, $H(0) \geq 0$.

The susceptible populations of the hosts S_c and S_b are positive they and cannot be zero at any how, but other infected populations can either be zero or greater than zero depending on the disease status in the population. They are zero if the the population is free from the disease and greater than zero if disease persists in the population.

The total population sizes of village chicken and the wild bird are given by

$N_c(t) = S_c(t) + E_c(t) + I_c(t)$ and $N_b(t) = S_b(t) + E_b(t) + I_r(t) + I_b(t)$ respectively.

3.2 Basic Properties of the Model

3.2.1 Invariant Region of the Solution

The ND model system $(22a) - (24)$ has three subpopulations where all parameters and variables are positive $\forall t \geq 0$.

Lemma 3.1

Given the model system $(22a) - (24)$ in $\mathbb{R}^8_+$ with the initial conditions $S_c(0) > 0$, $E_c(0) \geq 0, I_c(0) \geq 0$, $S_b(0) > 0$, $E_b(0) \geq 0$, $I_b(0) \geq 0$, $I_r(0) \geq 0$, $H(0) \geq 0$, its solution enters the

invariant region $\mathcal{D} = \mathcal{D}_1 \cup \mathcal{D}_2 \cup \mathcal{D}_3 = \mathbb{R}^3_+ \times \mathbb{R}^4_+ \times \mathbb{R}^1_+$ where;

$$\begin{aligned}
\mathcal{D}_1 &= \left\{(S_c(t), E_c(t), I_c(t)) \in \mathbb{R}^3_+ : S_c(t) + E_c(t) + I_c(t) = N_c\right\} \\
\mathcal{D}_2 &= \left\{(S_b(t), E_b(t), I_b(t), I_r(t)) \in \mathbb{R}^4_+ : S_b(t) + E_b(t) + I_b(t) + I_r(t) = N_b\right\} \\
\mathcal{D}_3 &= \left\{H(t) \in \mathbb{R}^1_+\right\}
\end{aligned} \tag{25}$$

Proof : to establish the feasible region of the ND model solution, we apply the box invariant method as used in (Abate *et al.*, 2009; Mpeshe *et al.*, 2014*b*; Kahuru *et al.*, 2017*a*). For our dynamical system $\dot{X} = \mathcal{G}\left(X, t\right), X \in \mathbb{R}^n$, we assume the continuity and the Lipschitz properties of its solution. The model system $(22a) - (24)$ is reduced to the form

$$\frac{dX}{dt} = Q\left(x\right) X + G \tag{26}$$

where $X = (S_c, E_c, I_c, S_b, E_b, I_b, I_r, H)^T$ and a column vector $G = (N_c, 0, 0, N_b, 0, 0, 0, 0)^T$.

$$Q\left(x\right) = \begin{pmatrix} Q_1\left(x\right) & 0 & 0 \\ 0 & Q_2\left(x\right) & 0 \\ 0 & 0 & Q_3\left(x\right) \end{pmatrix} \tag{27}$$

is a Metzler matrix for all $X \in \mathbb{R}^8_+$ with sub-matrix $Q_1\left(x\right)$, $Q_2\left(x\right)$ and $Q_3\left(x\right)$ from the village chicken, wild birds and environment respectively. We define the sub matrices from the system (27) as follows:

$$Q_1\left(x\right) = \begin{pmatrix} -\left(\beta_c(t, I_c, I_r, H) + \mu\right) & 0 & \frac{\psi}{N_c} \\ \beta_c(t, I_c, I_r, H) & -(\gamma + \mu) & 0 \\ 0 & \gamma & -(\delta_c + \mu) \end{pmatrix} \tag{28}$$

$$Q_2\left(x\right) = \begin{pmatrix} -\left(\beta_b(t, I_b, I_r, H) + \mu\right) & 0 & 0 & \frac{b}{N_b} \\ \beta_b(t, I_b, I_r, H) & -(\gamma + \mu) & 0 & \frac{a}{N_b} \\ 0 & \rho\gamma & -(\delta_b + \mu) & 0 \\ 0 & (1-\rho)\gamma & 0 & -\mu \end{pmatrix} \tag{29}$$

$$Q_3\left(x\right) = \begin{pmatrix} 0 & 0 & \alpha_c & 0 & 0 & \alpha_b & \alpha_b & -\mu_v \end{pmatrix} \tag{30}$$

By combining the matrices in equation (28) , (29) and (30), we get the matrix $Q\,(x)$ which is a Metzler matrix for all $X \in \mathbb{R}^8_+$ defined as:

$$Q\,(x) = \begin{pmatrix} -A_1 & 0 & \frac{\psi}{N_c} & 0 & 0 & 0 & 0 & 0 \\ A_2 & -(\gamma+\mu) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma & -(\delta_c+\mu) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_3 & 0 & 0 & \frac{b}{N_b} & 0 \\ 0 & 0 & 0 & A_4 & -(\gamma+\mu) & 0 & \frac{a}{N_b} & 0 \\ 0 & 0 & 0 & 0 & \rho\gamma & -(\delta_b+\mu) & 0 & 0 \\ 0 & 0 & 0 & 0 & (1-\rho)\gamma & 0 & -\mu & 0 \\ 0 & 0 & \alpha_c & 0 & 0 & \alpha_b & \alpha_b & -\mu_v \end{pmatrix} \tag{31}$$

where

$$A_1 = \beta_c(t, I_c, I_r, H) + \mu; \quad A_2 = \beta_c(t, I_c, I_r, H), \quad A_3 = \beta_b(t, I_b, I_r, H) + \mu$$
$$A_4 = \beta_b(t, I_b, I_r, H)$$

A reduced Metzler matrix $Q\,(x)$ in (31) has all negative values along its principle diagonal and the rest non-negative values in its off diagonal. Hence proves that all variables enter and remain in the invariant region $\mathcal{D}$. This shows that the ND model system $(22a) - (24)$ is epidemiologically meaningful and well posed in the invariant region $\mathcal{D}$.

3.2.2 Positivity of the Solution

Theorem 3.2

Let the initial set of variables of the model in the equation $(22a)-(24)$ be $S_c(0) > 0$, $E_c(0) \geq 0$, $I_c(0) \geq 0$, $S_b(0) > 0$, $E_b(0) \geq 0$, $I_r(0) \geq 0$, $I_b(0) \geq 0$ and $H(0) \geq 0$ then the solution set $\{(S_c(t) > 0, E_c(t) \geq 0, I_c(t) \geq 0, S_b(t) > 0, E_b(t) \geq 0, I_r(t) \geq 0, I_b(t) \geq 0, H(t) \geq 0\} \in \mathbb{R}^8_+$ is positive for all t.

Proof:

Lets consider the equation $(22a)$ of the model system $(22a) - (24)$

$$\frac{dS_c(t)}{dt} = \mu N_c - \beta_1(t, I_c, I_r, H)S_c(t) - \mu S_c(t) \tag{32}$$

$$\frac{dS_c(t)}{dt} \geq -\left(\beta_1(t, I_c, I_r, H) + \mu\right) S_c(t) \tag{33}$$

$$\int_0^t \frac{dS_c(t)}{S_c(t)} \geq -\int_0^t \left(\beta_1(t, I_c, I_r, H) + \mu\right) dt \tag{34}$$

$$S_c(t) \geq S_c(0) e^{-\mu t - \int_0^t (\beta_1(t, I_c, I_r, H) dt} \tag{35}$$

Thus as $t \to \infty$ then it follows that $S_c(t) \geq S_c(0) e^{-\mu t - \int_0^t (\beta_1(t, I_c, I_r, H) dt} \geq 0$. From equation $(22b)$ of the model system $(22a) - (24)$, we have

$$\frac{dE_c(t)}{dt} = \beta_c(t, I_c, I_r, H) S_c(t) - (\gamma + \mu) E_c\,(t) \tag{36}$$

$$\frac{dE_c(t)}{dt} \geq -(\gamma + \mu) E_c(t) \tag{37}$$

$$\frac{dE_c(t)}{E_c(t)} \geq -(\gamma + \mu) dt \tag{38}$$

Integrating both sides of equation (38) with respect to time we then have

$$\int_0^t \frac{dE_c(t)}{E_c(t)} \geq -\int_0^t (\gamma + \mu) dt \tag{39}$$

and finally we get

$$E_c(t) \geq E_c(0) e^{-(\gamma + \mu) t} \tag{40}$$

As $t \to \infty$, $E_c(t) \geq E_c(0) e^{-(\mu + \gamma) t} \geq 0$, we have $E_c(t) \geq 0$

Also from equation $(22c)$ of the model system $(22a) - (24)$ we have

$$\frac{dI_c(t)}{dt} = \gamma E_c(t) - (\delta_c + \mu) I_c(t) \tag{41}$$

$$\int_0^t \frac{dI_c(t)}{I_c} \geq -\int_0^t (\delta_c + \mu) dt \tag{42}$$

which gives $I_c(t) \geq I_c(0) e^{-(\delta_c + \mu) t} \geq 0$. Following the same procedures it follow that; $S_b(t) \geq 0$, $E_b(t) \geq 0$, $I_b(t) \geq 0$, $I_r(t) \geq 0$ and $H(t) \geq 0$ which proves that all state variables are positive $\forall t$.

3.2.3 Existence of the Steady States

The disease is endemic whenever persists in a population and the population is free iff no disease persists in it (Mwanga *et al.*, 2014; Selemani *et al.*, 2016). The steady state

$\phi^*\left(S_c^*, E_c^*, I_c^*, S_b^*, E_b^*, I_b^*, I_r^*, H^*\right)$ of the model system in equation $(22a) - (24)$ is obtained by setting the model system to zero and thus solving for the state variables. Therefore, we have the following system:

$$\begin{aligned}
\mu N_c - \left(\psi\frac{I_c(t)}{N_c} + b\frac{I_r(t)}{N_b} + \frac{dH(t)}{\kappa + H(t)} + \mu\right) S_c(t) = 0 \\
\left(\psi\frac{I_c(t)}{N_c} + b\frac{I_r(t)}{N_b} + \frac{dH(t)}{\kappa + H(t)}\right) S_c(t) - (\mu+\gamma)E_c(t) = 0 \\
\gamma E_c(t) - (\delta_c + \mu)I_c(t) = 0
\end{aligned} \tag{43}$$

$$\alpha_c I_c(t) + \alpha_b\left(I_b(t) + I_r(t)\right) - \mu_v H(t) = 0 \tag{44}$$

$$\begin{aligned}
\mu N_b - \left(\frac{\varphi I_b(t) + aI_r(t)}{N_b} + \frac{dH(t)}{\kappa + H(t)} + \mu\right) S_b(t) = 0 \\
\left(\frac{\varphi I_b(t) + aI_r(t)}{N_b} + \frac{dH(t)}{\kappa + H(t)}\right) S_b(t) - (\gamma+\mu)E_b(t) = 0 \\
\rho\gamma E_b(t) - (\delta_b + \mu)I_b(t) = 0 \\
(1-\rho)\gamma E_b(t) - \mu I_r(t) = 0
\end{aligned} \tag{45}$$

Compartment wise, we have the following steady states in village chicken population:

$$I_c^* = \frac{\gamma}{\delta_c + \mu}E_c^*, \;\; E_c^* = \frac{\beta_c S_c^*}{\mu + \gamma}, \;\; S_c^* = \frac{\mu N_c^*}{\beta_c + \mu} \tag{46}$$

Substituting S_c^* into E_c^* and E_c^* into I_c^* we get

$$E_c^* = \frac{\beta_c \mu N_c^*}{(\mu+\gamma)(\beta_c+\mu)} \tag{47}$$

$$I_c^* = \frac{\gamma}{(\delta_1+\mu)}\frac{\beta_c \mu N_c^*}{(\mu+\gamma)(\beta_c+\mu)} \tag{48}$$

By considering the force of infections in chicken, I_c is obtained by solving the equation

$$\mathcal{B}_2 I_c^* + \mathcal{B}_1 I_c^* + \mathcal{B}_0 = 0 \tag{49}$$

where:

$$\begin{aligned}
\mathcal{B}_2 &= \mu N_c(\delta_c+\mu)(\mu+\gamma)(\kappa+H) \\
\mathcal{B}_1 &= N_b N_c(\kappa+H) + bN_c(\delta_c+\mu)(\mu+\gamma)(\kappa+H)I_r + \mu N_b N_c(\kappa+H)(\delta_c+\mu)(\mu+\gamma) \\
\mathcal{B}_0 &= b\mu\gamma N_c^2(\kappa+H)I_r + d\mu\gamma N_b N_c^2(1-\mathcal{R}_0)
\end{aligned} \tag{50}$$

$$N_c^* = S_c^* + E_c^* + I_c^* = \left(1 + \mathcal{R}_{bch}\left(1 + \frac{\beta_1 \mu N_c^*}{(\mu+\gamma)(\beta_c+\mu)}\right)\right) S_c^* \tag{51}$$

where $\beta_c = \left(\psi \frac{I_c^*(t)}{N_c^*} + b\frac{I_r^*(t)}{N_b^*} + \frac{dH^*(t)}{\kappa + H^*(t)}\right)$, and $\mathcal{R}_{bch} = \frac{\beta_c \mu N_c^*}{(\mu+\gamma)(\beta_1+\mu)}$

and

$$I_c^* = \frac{-\mathcal{B}_1 \pm \sqrt{\mathcal{B}_1^2 - 4\mathcal{B}_2\mathcal{B}_0}}{2\mathcal{B}_0} \tag{52}$$

Also in the wild birds population we have the following steady states:

$$S_b^*(t) = \frac{\mu N_b^*}{\beta_b + \mu}, \;\; E_b^*(t) = \left(\frac{\beta_b S_b^*}{\mu+\gamma}\right) \tag{53}$$

$$I_b^* = \frac{\rho\gamma\mu\beta_b N_b^*}{(\delta_b+\mu)(\mu+\gamma)(\beta_b+\mu)} \tag{54}$$

$$I_r^*(t) = \left(\frac{(1-\rho)\gamma\mu\beta_b N_b^*}{\mu(\mu+\gamma)(\beta_b+\mu)}\right) \tag{55}$$

$$N_b^* = S_b^* + E_b^* + I_b^* + I_r^* = \left(1 + \mathcal{R}_{bch}\left(1 + \frac{\gamma\rho}{\mu} + \frac{\gamma\rho}{\delta_b+\mu}\right)\right) S_b^* \tag{56}$$

Where $\beta_b = \left(\frac{\varphi I_b^*(t) + a I_r^*(t)}{N_b^*} + \frac{dH^*(t)}{\kappa + H^*(t)}\right)$ and $\mathcal{R}_{cbh} = \frac{\beta_b \mu N_b^* \mu}{(\mu+\gamma)(\beta_b+\mu)}$

$$H^*(t) = \frac{\alpha_c\gamma\mu\beta_c N_c^*}{\mu_v(\mu+\gamma)(\beta_c+\mu)} + \frac{\alpha_b}{\mu_v}\left(\frac{\rho\gamma\mu\beta_b N_b^*}{(\mu+\gamma)(\beta_b+\mu)}\left(\frac{\mu}{\delta_b+\mu} - \frac{1}{\mu}\right)\right) + \frac{\gamma\beta_b N_b^*}{(\mu+\gamma)(\beta_b+\mu)} \tag{57}$$

From all these state variables, the solution $\beta_c \neq 0$ and $\beta_b \neq 0$ gives the endemic equilibrium points while $\beta_c = 0$ and $\beta_b = 0$ gives the disease free equilibrium point.

3.2.4 Existence of Disease Free Equilibrium Point

The model has a disease free equilibrium point (DFEP) which is obtained when all forces of infections in the steady states are set to zero $i.e$, $\beta_c(I_c, I_r, H) = \beta_b(I_b, I_r, H) = 0$. Therefore the disease free equilibrium point in their respective compartments are $\phi_c^0 = \{N_c, 0, 0\}$, $\phi_b^0 = \{N_b, 0, 0, 0\}$ and $\phi_H^0 = 0$ for village chicken,wild birds and the concentration of NDV in the the environment respectively. Generally, the disease free equilibrium point of a model is given by $\phi_0 = \{N_c, 0, 0, N_b, 0, 0, 0, 0\}$ where N_c and N_b represent the population size of village chicken and wild birds respectively.

3.2.5 Existence of Endemic Equilibrium Point

From the stead states in equation (46) to (57), the endemic equilibrium $\mathcal{D}^* = (S_c^*, E_c^*, I_c^*, S_b^*, E_b^*, I_b^*, I_r^r, H^*)$ is found iff the force of infections are not equal to zero $i.e$ $\beta_c\left(I_c, I_r, H\right) \neq 0 \quad \text{and} \quad \beta_b\left(I_b, I_r, H\right) \neq 0$. Therefore, the endemic equilibrium point $\mathcal{D}^*$ is a set of the steady states in a condition that $E_c^* \neq 0$, $I_c^* \neq 0$, $E_b^* \neq 0$, $I_b^* \neq 0$, $I_r^* \neq 0$ and $H^* \neq 0$.

3.2.6 Bifurcation Analysis for the Equilibrium Points

The existence of forward or backward bifurcation has an important implications on the epidemiological control measure of the infectious diseases. The bifurcation analysis of the equilibrium points tells the nature of the points and also tells whether the disease can be completely reduced or remain in the population. Therefore, in this part we use the centre manifold theorem as stated by Castillo-Chavez and Song (2004), Buonomo and Vargas-De-León (2013), Nyerere *et al.* (2014) and Mushayabasa *et al.* (2017) to investigate the changes of signs of the equilibrium points around $\mathcal{R}_0$ close to one.

Theorem 3.3

Castillo-Chavez and Song (2004), Consider the following general system of ordinary differential equations with a parameter ψ

$$\frac{dx}{dt} = g\left(x, \psi\right), \;\; g : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}^n \;\; and \;\; g \in \mathbb{C}^n(\mathbb{R}^n \times \mathbb{R}) \tag{58}$$

it is assumed that ψ is an equilibrium for system (58) for all values of the parameter ψ, $(that \;\; is \;\; g(0, \psi) \equiv 0)$. Now, suppose that:

(i) $M = D_x g\left(0, 0\right) = \frac{\partial g_i}{\partial x_j}\left(0, 0\right)$ is the linearized matrix of the system in (58) around the equilibrium 0 and ψ evaluated at 0. Zero is a simple eigenvalue of M and all other eigenvalues of M have negative real parts:

(ii) Matrix M has a non-negative right eigenvector ω and a left eigenvector v corresponding to the zero eigenvalue. Let g_k be the k^{th} component of g and

$$\begin{aligned}\mathcal{S} &= \sum_{k,i,j=1}^{n} v_k \omega_i \omega_j \frac{\partial^2 g_k}{\partial x_i \partial x_j}(0,0)\,,\\ \mathcal{T} &= \sum_{i,k=1}^{n} v_k \omega_i \frac{\partial^2 g_k}{\partial x_i \partial \psi}(0,0)\end{aligned} \tag{59}$$

the local dynamics of the system (58) around zero are totally determined by the signs of $\mathcal{S}$ and $\mathcal{T}$. If $\mathcal{S} > 0$ and $\mathcal{T} > 0$, then a backward bifurcation occurs at $\psi = 0$.

(i) $\mathcal{S} > 0$, $\mathcal{T} > 0$, when $\psi < 0$ with $|\psi| << 0$, is locally asymptotically stable and there exist a positive unstable equilibrium. When $0 < |\psi| << 1$, 0 is unstable and there exists a negative and a locally asymptotically stable equilibrium.

(ii) $\mathcal{S} < 0$, $\mathcal{T} < 0$, when $\psi < 0$ with $|\psi| << 0$, is unstable. When $0 < |\psi| << 1$, 0 is asymptotically stable and there exists a positive unstable equilibrium.

(iii) $\mathcal{S} > 0$, $\mathcal{T} < 0$, when $\psi < 0$ with $|\psi| << 0$, is unstable. When $|\psi| << 1$, 0 is unstable and there exists a locally asymptotically unstable equilibrium.

(iv) $\mathcal{S} < 0$, $\mathcal{T} > 0$, when $\psi < 0$ changes sign from negative to positive, 0 changes its stability from stable to unstable. The corresponding negative unstable equilibrium becomes positive and locally asymptotically stable.

Now, to apply the above theorem, the change of variables are made on the model system $(22a)$ to (24) by letting the variables as follows; $S_c = x_1$, $E_c = x_2$, $I_c = x_3$, $S_b = x_4$, $E_b = x_5$, $I_b = x_6$, $I_r = x_7$, $H = x_8$. These simplifications give $N_c = x_1 + x_2 + x_3$ and $N_b = x_4 + x_5 + x_6 + x_7$. With the vector notations $x = (x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8)^T$ and $\frac{dx}{dt} = (g_1, g_2, g_3, g_4, g_5, g_6, g_7, g_8)^T$ the system $(22a)$ to (24) is now re-written as;

$$
\begin{aligned}
\frac{dx_1(t)}{dt} &= g_1 = \mu\left(x_1(t)+x_2(t)+x_3(t)\right)-\left(\beta_1+\mu\right)x_1(t)\\
\frac{dx_2(t)}{dt} &= g_2 = \beta_1 x_1(t)-(\mu+\gamma)x_2(t)\\
\frac{dx_3(t)}{dt} &= g_3 = \gamma x_2(t)-(\delta_c+\mu)x_3(t)\\
\frac{dx_4(t)}{dt} &= g_4 = \mu\left(x_4(t)+x_5(t)+x_6(t)+x_7(t)\right)-(\mu+\beta_2)x_4(t)\\
\frac{dx_5(t)}{dt} &= g_5 = \beta_2 x_4(t)-(\mu+\rho\gamma)x_5(t)\\
\frac{dx_6(t)}{dt} &= g_6 = \rho\gamma x_5(t)-\left(\delta_b+\mu\right)x_6(t)\\
\frac{dx_7(t)}{dt} &= g_7 = (1-\rho)\gamma x_5(t)-\mu x_7(t)\\
\frac{dx_8(t)}{dt} &= g_8 = \alpha_c x_3(t)+\alpha_b\left(x_6(t)+x_7(t)\right)-\mu_v x_8(t)
\end{aligned}
\tag{60}
$$

$\beta_1 = \psi\frac{x_3(t)}{x_1(t)+x_2(t)+x_3(t)} + b\frac{x_7(t)}{x_4(t)+x_5(t)+x_6(t)+x_7(t)} + \frac{dx_8}{\kappa+x_8(t)}$, $\beta_2 = \frac{\varphi x_6(t)+a x_7(t)}{x_4(t)+x_5(t)+x_6(t)x_7(t)} + \frac{dx_8(t)}{\kappa+x_8(t)}$,
$x_1(t)+x_2(t)+x_3(t)=1,\ \ x_4(t)+x_5(t)+x_6(t)x_7(t)=1,\ x_i(t)\geq 0$ for $i=1,2,...,8$

Lets choose $\psi = \psi^*$ from the basic reproduction number as the bifurcation parameter. Using Maple for solving ψ^* from $\mathcal{R}_0 = 1$ we then have

$$
\psi = \psi^* = -\frac{1}{2}\left(\frac{\varpi}{\tau}\right) \tag{61}
$$

$$
\begin{aligned}
\varpi = & -\kappa\,\mu^3\phi\,\rho\gamma\,N_b\mu_v + \gamma^2\kappa\,\mu^2 N_b\delta_c\mu_v - \gamma\,a\kappa\,\mu^3 N_b\mu_v - \gamma^2\mu\,N_b\alpha_b dN_b\delta_b - \gamma\,\mu^2 N_b\alpha_b dN_b\delta_b \\
& - \gamma^2 a\kappa\,\mu^2 N_b\mu_v + \gamma^2 a\mu\,N_b\alpha_c dN_c + \gamma^2 a N_b\alpha_c dN_c\delta_b + \gamma^2\mu\,\rho\,\alpha_c bN_c dN_b - \gamma^2\mu\,N_b\alpha_b dN_b\delta_c \\
& + \gamma^2\rho\,\alpha_c bN_c dN_b\delta_b - \gamma^2 N_b\alpha_b dN_b\delta_b\delta_c - \gamma\,\mu^2 N_b\alpha_b dN_b\delta_c - \gamma\,\mu^2 N_b\alpha_c dN_c\delta_b + \kappa\,\mu^3 N_b\delta_b\delta_c\mu_v \\
& + 2\,\gamma\,\kappa\,\mu^3 N_b\delta_b\mu_v + 2\,\gamma\,\kappa\,\mu^3 N_b\delta_c\mu_v - \gamma^2\mu\,N_b\alpha_c dN_c\delta_b + \gamma^2\kappa\,\mu^2 N_b\delta_b\mu_v + \kappa\,\mu^5 N_b\mu_v \\
& + \gamma\,a\kappa\,\mu\,\rho\,N_b\delta_b\delta_c\mu_v - \kappa\,\mu^2\phi\,\rho\gamma\,N_b\delta_c\mu_v + \gamma^2\kappa\,\mu\,N_b\delta_b\delta_c\mu_v + 2\,\gamma\,\kappa\,\mu^2 N_b\delta_b\delta_c\mu_v \\
& - \gamma^2 a\kappa\,\mu\,N_b\delta_c\mu_v - \gamma^2 a\kappa\,N_b\delta_b\delta_c\mu_v - \gamma^2 a\mu\,\rho\,N_b\alpha_c dN_c - \gamma^2 a\rho\,N_b\alpha_c dN_c\delta_b + \gamma^2\rho\,N_b\alpha_b dN_b\delta_b\delta_c \\
& - \gamma\,a\kappa\,\mu^2 N_b\delta_c\mu_v + \gamma\,\mu\,\phi\,\rho\gamma\,N_b\alpha_c dN_c - \gamma\,\mu\,N_b\alpha_b dN_b\delta_b\delta_c + \gamma^2 a\kappa\,\mu^2\rho\,N_b\mu_v + \gamma\,a\kappa\,\mu^3\rho\,N_b\mu_v \\
& - \gamma^2 a\kappa\,\mu\,N_b\delta_b\mu_v + \gamma^2\mu\,\rho\,N_b\alpha_b dN_b\delta_b - \gamma\,a\kappa\,\mu^2 N_b\delta_b\mu_v - \gamma\,\kappa\,\mu^2\phi\,\rho\gamma\,N_b\mu_v + \gamma\,\mu^2\rho\,N_b\alpha_b dN_b\delta_b \\
& + \gamma^2 a\kappa\,\mu\,\rho\,N_b\delta_b\mu_v + \gamma\,a\kappa\,\mu^2\rho\,N_b\delta_b\mu_v + \gamma^2 a\kappa\,\mu\,\rho\,N_b\delta_c\mu_v + \gamma^2 a\kappa\,\rho\,N_b\delta_b\delta_c\mu_v + \gamma\,a\kappa\,\mu^2\rho\,N_b\delta_c\mu_v \\
& - \gamma\,a\kappa\,\mu\,N_b\delta_b\delta_c\mu_v - \gamma\,\kappa\,\mu\,\phi\,\rho\gamma\,N_b\delta_c\mu_v + \gamma\,\mu\,\rho\,N_b\alpha_b dN_b\delta_b\delta_c + \kappa\,\mu^4 N_b\delta_c\mu_v - \gamma\,\mu^3 N_b\alpha_c dN_c \\
& - \gamma^2\mu\,\alpha_c bN_c dN_b - \gamma^2\alpha_c bN_c dN_b\delta_b + \gamma^2\kappa\,\mu^3 N_b\mu_v + 2\,\gamma\,\kappa\,\mu^4 N_b\mu_v - \gamma^2\mu^2 N_b\alpha_c dN_c \\
& - \gamma^2\mu^2 N_b\alpha_b dN_b - \gamma\,\mu^3 N_b\alpha_b dN_b + \kappa\,\mu^4 N_b\delta_b\mu_v
\end{aligned}
\tag{62}
$$

$$
\tau = \gamma\,\left(\epsilon + \kappa\,\mu^3\mu_v + \kappa\,\mu^2\delta_b\mu_v - \kappa\,\mu\,\phi\,\rho\gamma\,\mu_v - \gamma\,\mu\,\alpha_b dN_b - \gamma\,\alpha_b dN_b\delta_b\right) N_b \tag{63}
$$

$$
\epsilon = a\gamma\,\kappa\,\mu\,\rho\,\mu_v + a\gamma\,\kappa\,\rho\,\delta_b\mu_v - a\gamma\,\kappa\,\mu\,\mu_v - a\gamma\,\kappa\,\delta_b\mu_v + \gamma\,\kappa\,\mu^2\mu_v + \gamma\,\kappa\,\mu\,\delta_b\mu_v + \gamma\,\rho\,\alpha_b dN_b\delta_b \tag{64}
$$

Then, the linearized system (60) is transformed with $\psi = \psi^*$ which has a simple zero eigenvalues and the centre manifold theory is used to analyze the dynamics of (60) near $\psi = \psi^*$. The Jacobian of the system (60) at $\psi = \psi^*$ has a right eigenvector associated with zero eigenvalues given by $\omega = (\omega_1, \omega_2, ..., \omega_8)^T$. The eigenvectors of the Jacobian matrix of the system (60) are obtained as follows:

$$
\begin{pmatrix}
-\mu & 0 & -\psi^* & 0 & 0 & 0 & -b & -\frac{d}{\kappa} \\
0 & -\gamma-\mu & \psi^* & 0 & 0 & 0 & b & \frac{d}{\kappa} \\
0 & \gamma & -\delta_c-\mu & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & -\mu & 0 & -\varphi & -a & \frac{-d}{\kappa} \\
0 & 0 & 0 & 0 & -\mu-\rho\gamma & \varphi & a & \frac{d}{\kappa} \\
0 & 0 & 0 & 0 & \gamma\rho & -\delta_b-\mu & 0 & 0 \\
0 & 0 & 0 & 0 & \gamma\,(1-\rho) & 0 & -\mu & 0 \\
0 & 0 & \alpha_c & 0 & 0 & \alpha_b & \alpha_b & -\mu_v
\end{pmatrix}
\begin{pmatrix}
\omega_1 \\ \omega_2 \\ \omega_3 \\ \omega_4 \\ \omega_5 \\ \omega_6 \\ \omega_7 \\ \omega_8
\end{pmatrix}
=
\begin{pmatrix}
0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0
\end{pmatrix}
\tag{65}
$$

This gives the following eigenvectors

$$\begin{aligned}
\omega_1 &= -\left(\frac{\gamma+\mu}{\mu}\right)\omega_2, \\
\omega_2 &= -\left(\frac{\mu}{\gamma+\mu}\right)\omega_1, \\
\omega_3 &= \frac{\left(b\kappa\omega_7 - d\omega_8\right)\gamma}{\left(\left(\mu+\gamma\right)\left(\delta_c+\mu\right)-\psi\gamma\right)}, \\
\omega_4 &= \left(\frac{\mu+\gamma\rho}{\mu}\right)\omega_5, \\
\omega_5 &> 0, \\
\omega_6 &= \left(\frac{\mu\kappa\rho\gamma\left(a\kappa\omega_7 - d\omega_8\right)}{\mu\kappa\left(\mu\kappa\left(\delta_b+\mu\right)-\rho\gamma\kappa\left(\varphi-\delta_b-\mu\right)\right)}\right) \\
\omega_7 &= \left(\frac{1-\rho\gamma}{\mu}\right)\omega_5, \\
\omega_8 &= \frac{\alpha_c\omega_3 + \alpha_b\left(\omega_6+\omega_7\right)}{\mu_v}
\end{aligned} \tag{66}$$

Also, the left eigenvector $v_i = \left(v_1, v_2, ..., v_8\right)^T$ associated with the zero Eigenvalues at $\varphi = \varphi^*$ gives following Jacobian matrix:

$$\begin{pmatrix}
-\mu & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & -\gamma-\mu & \gamma & 0 & 0 & 0 & 0 & 0 \\
-\psi^* & \psi^* & -\delta_c-\mu & 0 & 0 & 0 & 0 & \alpha_c \\
0 & 0 & 0 & -\mu & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & -\mu-\rho\gamma & \gamma\rho & (1-\rho)\gamma & 0 \\
0 & 0 & 0 & -\varphi & \varphi & -\delta_b-\mu & 0 & \alpha_b \\
-b & b & 0 & -a & a & 0 & -\mu & \alpha_b \\
-\frac{d}{\kappa} & \frac{d}{\kappa} & 0 & -\frac{d}{\kappa} & \frac{d}{\kappa} & 0 & 0 & -\mu_v
\end{pmatrix}
\begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \\ v_5 \\ v_6 \\ v_7 \\ v_8 \end{pmatrix}
=
\begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{67}$$

Then from the linear combinations it follows that

$$
\begin{aligned}
v_1 &= 0 \\
v_3 &= \frac{(\delta_c + \mu)\, v_2}{\gamma} \\
v_4 &= 0 \\
v_6 &= \frac{\mu v_5 - \gamma\rho\,(v_5 + v_7) - \gamma v_7}{\rho\gamma} \\
v_7 &= \frac{b v_2 + a v_5 + \alpha_b v_8}{\mu} \\
v_8 &= \frac{(\delta_c + \mu)\, v_3}{\alpha_c}
\end{aligned}
\tag{68}
$$

Computations of $\mathcal{S}$ and $\mathcal{T}$

From the system (60) the associated non-zero partial derivatives of the function g_k at disease free equilibrium are

$$
\frac{\partial^2 g_1}{\partial x_1 \partial x_3} = -\psi, \;\; \frac{\partial^2 g_1}{\partial x_1 \partial x_7} = -b, \;\; \frac{\partial^2 g_1}{\partial x_1 \partial x_8} = \frac{\partial^2 g_4}{\partial x_4 \partial x_8} = -\frac{d}{\kappa}
$$

$$
\frac{\partial^2 g_4}{\partial x_4 \partial x_6} = -\varphi, \;\; \frac{\partial^2 g_4}{\partial x_4 \partial x_7} = -a
$$

From equation (59) it then follows that

$$
\begin{aligned}
\mathcal{S} = {} & v_1\omega_1\omega_3 \frac{\partial^2 g_4}{\partial x_1 \partial x_3} + v_2\omega_1\omega_7 \frac{\partial^2 g_4}{\partial x_1 \partial x_7} + v_3\omega_1\omega_8 \frac{\partial^2 g_4}{\partial x_1 \partial x_8} + v_4\omega_4\omega_8 \frac{\partial^2 g_4}{\partial x_4 \partial x_8} \\
& + v_5\omega_4\omega_6 \frac{\partial^2 g_4}{\partial x_4 \partial x_6} + v_6\omega_4\omega_7 \frac{\partial^2 g_4}{\partial x_4 \partial x_7}
\end{aligned}
\tag{69}
$$

$$
\mathcal{S} = -\psi v_1\omega_1\omega_3 - b v_2\omega_1\omega_7 - \frac{d}{\kappa} v_3\omega_1\omega_8 - \frac{d}{\kappa} v_4\omega_4\omega_8 - \varphi v_5\omega_4\omega_6 - a v_5\omega_4\omega_7 \tag{70}
$$

$$
\mathcal{S} = -\omega_2 \left(\frac{\gamma + \mu}{\mu}\right) \left(b v_2\omega_7 + \frac{d}{\kappa} v_3\omega_8\right) - v_5\omega_5\,(\varphi\omega_6 + a\omega_7) \left(\frac{\mu + \gamma\rho}{\mu}\right) < 0 \tag{71}
$$

For the sign of $\mathcal{T}$, it can be shown that the associated non-zero partial derivatives of the function g_i at disease free equilibrium are:

$$
\frac{\partial^2 g_1}{\partial \psi \partial x_3} = -1, \;\; \frac{\partial^2 g_2}{\partial \psi \partial x_3} = 1 \tag{72}
$$

which gives

$$
\mathcal{T} = v_1\omega_1 \frac{\partial^2 g_1}{\partial x_3 \partial \psi} + v_2\omega_2 \frac{\partial^2 g_2}{\partial x_3 \partial \psi} \tag{73}
$$

and therefore

$$\mathcal{T} = -v_1\omega_1 + v_2\omega_2 \tag{74}$$

But $v_1 = 0$, then it follows from equation (74) that $\mathcal{T} = v_2\omega_2 > 0$. Since $\mathcal{S} < 0$ and $\mathcal{T} > 0$, regarding to Theorem $3.3\,(4)$, the equilibrium is positive, unique and locally asymptotically stable. Thus the system undergoes the forward bifurcation with $\mathcal{R}_0$ close to one.

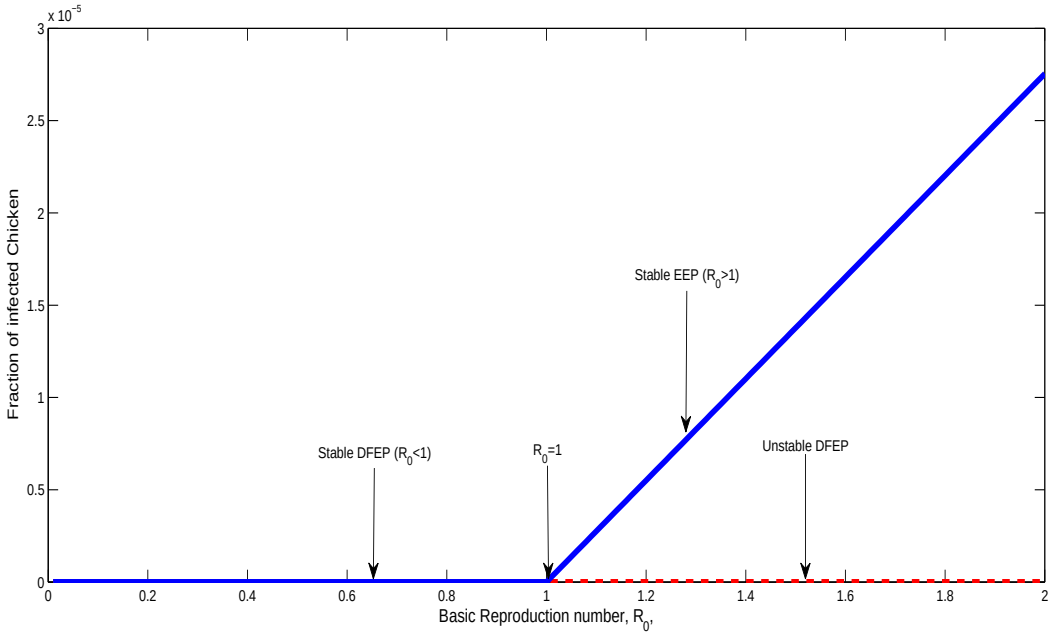


Figure 4: The Forward Bifurcation for a ND model in village chicken with environment and wild birds reservoirs.

The diagram shows the behavior of the disease near the point $\mathcal{R}_0 = 1$. For the disease to disappear from the village chicken population, making $\mathcal{R}_0 < 1$ is a necessary condition to reach the target. So $\mathcal{R}_0$ should be kept as low as possible to reduce the spread of ND in the village chicken population.

Theorem 3.4

The Equilibrium point of the ND model undergoes forward bifurcation and endemic equilibrium is locally asymptotically stable for $\mathcal{R}_0 > 1$ with $\mathcal{R}_0$ close to one.

3.2.7 The Basic Reproductive Number

The basic reproduction number $\mathcal{R}_0$, is defined as the average number of secondary cases caused by one infectious individual introduced in a population that consisting of entirely susceptibles (Foppa, 2005; Hartemink *et al.*, 2008). This number tells and quantifies the ability of an infectious disease to invade a purely susceptible population (Foppa, 2005; Hartemink *et al.*, 2008). The Epidemic persists when $\mathcal{R}_0 > 1$ and dies out when the $\mathcal{R}_0 < 1$ (Diekmann *et al.*, 2009; Hethcote, 2000; Van den Driessche and Watmough, 2002; Wang and Modnak, 2011). We compute the $\mathcal{R}_0$ by the next generation method as proposed by Van den Driessche and Watmough (2002). We firstly define our system for infections in compartments as

$$\frac{dX_i}{dt} = \mathcal{F}_i - \mathcal{V}_i \tag{75}$$

Where:

(i) X_i defines a set of infected classes

(ii) $\mathcal{F}_i$ defines the rate of new infections in compartment i

(iii) $\mathcal{V}_i = \mathcal{V}_i^- - \mathcal{V}_i^+$ the total transfer rate

$\mathcal{V}_i^-$ defines the rate of transfer of individuals out of compartment i and $\mathcal{V}_i^+$ is the rate of transfer of individuals into compartment i through interactions. Then it follows that;

$$\begin{pmatrix} \frac{dE_c}{dt} \\ \frac{dI_c}{dt} \\ \frac{dE_b}{dt} \\ \frac{dI_r}{dt} \\ \frac{dI_b}{dt} \\ \frac{dH}{dt} \end{pmatrix} = \mathcal{F}_i - \mathcal{V}_i = \begin{pmatrix} \left(\psi \frac{I_c}{N_c} + b\frac{I_r}{N_b} + d\frac{H}{\kappa+H}\right) S_c \\ 0 \\ \left(\frac{\varphi I_b + aI_r}{N_b} + d\frac{H}{\kappa+H}\right) S_b \\ 0 \\ 0 \\ 0 \end{pmatrix} - \begin{pmatrix} -(\gamma+\mu)E_c \\ \gamma E_c - (\delta_c+\mu)I_c \\ -(\gamma+\mu)E_b \\ \rho\gamma E_b - (\delta_b+\mu)I_b \\ (1-\rho)\gamma E_b - \mu I_r \\ \alpha_c I_c + \alpha_b\left(I_b + I_r\right) - \mu_v H \end{pmatrix}$$

The corresponding Jacobian matrices of F and V are the matrices of the derivatives of $\mathcal{F}_i$ and $\mathcal{V}_i$ with respect to $E_c(t)$, $I_c(t)$, $E_b(t)$, $I_r(t)$, $I_b(t)$ and $H(t)$ at the disease free equilibrium point, ϕ_0, which are given by

$F = \left(\frac{\partial \mathcal{F}_i(\phi_0)}{\partial X_j}\right)$ and $V = \left(\frac{\partial \mathcal{V}_i(\phi_0)}{\partial X_i}\right)$ respectively.

Then by differentiating the equation $\mathcal{F}_i$ and $\mathcal{V}_i$ $w.r.t$ the infected classes we get the following matrices

$$F = \begin{pmatrix} 0 & \psi & 0 & 0 & b\frac{N_c}{N_b} & d\frac{N_c}{\kappa} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \varphi & a & d\frac{N_b}{\kappa} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{76}$$

$$V = \begin{pmatrix} \gamma+\mu & 0 & 0 & 0 & 0 & 0 \\ -\gamma & \delta_c+\mu & 0 & 0 & 0 & 0 \\ 0 & 0 & (\gamma+\mu) & 0 & 0 & 0 \\ 0 & 0 & -\rho\gamma & (\delta_b+\mu) & 0 & 0 \\ 0 & 0 & -(1-\rho)\gamma & 0 & \mu & 0 \\ 0 & -\alpha_c & 0 & -\alpha_b & -\alpha_b & \mu_v \end{pmatrix} \tag{77}$$

The inverse of matrix V in equation (77) become

$$V^{-1} = \begin{pmatrix} (\gamma+\mu)^{-1} & 0 & 0 & 0 & 0 & 0 \\ \frac{\gamma}{(\gamma+\mu)(\delta_c+\mu)} & (\delta_c+\mu)^{-1} & 0 & 0 & 0 & 0 \\ 0 & 0 & (\gamma+\mu)^{-1} & 0 & 0 & 0 \\ 0 & 0 & \frac{\rho\gamma}{(\gamma+\mu)(\delta_b+\mu)} & (\delta_b+\mu)^{-1} & 0 & 0 \\ 0 & 0 & -\frac{(-1+\rho)\gamma}{(\gamma+\mu)\mu} & 0 & \mu^{-1} & 0 \\ \frac{\gamma\,\alpha_c}{(\gamma+\mu)(\delta_c+\mu)\mu_v} & \frac{\alpha_c}{(\delta_c+\mu)\mu_v} & -\frac{\alpha_b(-\gamma\,\delta_b-\gamma\,\mu+\gamma\,\rho\,\delta_b)}{(\gamma+\mu)(\delta_b+\mu)\mu\,\mu_v} & \frac{\alpha_b}{(\delta_b+\mu)\mu_v} & \frac{\alpha_b}{\mu\,\mu_v} & {\mu_v}^{-1} \end{pmatrix} \tag{78}$$

We then compute the next generation matrix FV^{-1} by multiplying the matrices of equation (76) and (78) which gives

$$FV^{-1} = \begin{pmatrix} R & S & T & \frac{dN_c\alpha_b}{\kappa\,(\delta_b+\mu)\mu_v} & \frac{bN_c}{N_b\mu} + \frac{dN_c\alpha_b}{\kappa\,\mu\,\mu_v} & \frac{dN_c}{\kappa\,\mu_v} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ U & V & W & \frac{\varphi}{\delta_b+\mu} + \frac{dN_b\alpha_b}{\kappa\,(\delta_b+\mu)\mu_v} & \frac{a}{\mu} + \frac{dN_b\alpha_b}{\kappa\,\mu\,\mu_v} & \frac{dN_b}{\kappa\,\mu_v} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{79}$$

where

$$R = \frac{\psi\,\gamma}{(\gamma+\mu)\,(\delta_c+\mu)} + \frac{dN_c\gamma\,\alpha_c}{\kappa\;(\gamma+\mu)\,(\delta_c+\mu)\,\mu_v}$$

$$S = \frac{\psi}{\delta_c+\mu} + \frac{dN_c\alpha_c}{\kappa\;(\delta_c+\mu)\,\mu_v}$$

$$T = \frac{bN_c\,(1-\rho)\,\gamma}{N_b\,(\gamma+\mu)\,\mu} + \frac{dN_c\alpha_b\,(\gamma\,\delta_b+\gamma\,\mu-\gamma\,\rho\,\delta_b)}{\kappa\;(\gamma+\mu)\,(\delta_b+\mu)\,\mu\,\mu_v}$$

$$U = \frac{dN_b\gamma\,\alpha_c}{\kappa\;(\gamma+\mu)\,(\delta_c+\mu)\,\mu_v},$$

$$V = \frac{dN_b\alpha_c}{\kappa\;(\delta_c+\mu)\,\mu_v}$$

$$W = \frac{\varphi\,\rho\gamma}{(\gamma+\mu)\,(\delta_b+\mu)} + \frac{a\,(1-\rho)\,\gamma}{(\gamma+\mu)\,\mu} + \frac{dN_b\alpha_b\,(\gamma\,\delta_b+\gamma\,\mu-\gamma\,\rho\,\delta_b)}{\kappa\;(\gamma+\mu)\,(\delta_b+\mu)\,\mu\,\mu_v}$$

The eigenvalues of the matrix (79) are

$$\begin{aligned}
&\lambda_1 = \lambda_2 = \lambda_3 = \lambda_4 = 0 \\
&\lambda_5 = -1/2\left(R + \frac{\varphi\,\rho\gamma}{(\gamma+\mu)\,(\delta_b+\mu)} + \frac{a\,(1-\rho)\,\gamma}{(\gamma+\mu)\,\mu} + \frac{dN_b\alpha_b\,(\gamma\,\delta_b+\gamma\,\mu-\gamma\,\rho\,\delta_b)}{\kappa\;(\gamma+\mu)\,(\delta_b+\mu)\,\mu\,\mu_v}\right) \\
&\quad + 1/2\sqrt{\left(R - \frac{\varphi\,\rho\gamma}{(\gamma+\mu)\,(\delta_b+\mu)} + \frac{a\,(1-\rho)\,\gamma}{(\gamma+\mu)\,\mu} + \frac{dN_b\alpha_b\,(-\gamma\,\delta_b-\gamma\,\mu+\gamma\,\rho\,\delta_b)}{\kappa\;(\gamma+\mu)\,(\delta_b+\mu)\,\mu\,\mu_v}\right)^2 + 4\omega}
\end{aligned} \tag{80}$$

$$\lambda_6 = \frac{1}{2}\left(R + \frac{\varphi\,\rho\gamma}{(\gamma+\mu)\,(\delta_b+\mu)} + \frac{a\,(1-\rho)\,\gamma}{(\gamma+\mu)\,\mu} + \frac{dN_b\alpha_b\,(\gamma\,\delta_b+\gamma\,\mu-\gamma\,\rho\,\delta_b)}{\kappa\;(\gamma+\mu)\,(\delta_b+\mu)\,\mu\,\mu_v}\right)$$
$$+\frac{1}{2}\sqrt{\left(R - \frac{\varphi\,\rho\gamma}{(\gamma+\mu)\,(\delta_b+\mu)} - \frac{a\,(1-\rho)\,\gamma}{(\gamma+\mu)\,\mu} - \frac{dN_b\alpha_b\,(\gamma\,\delta_b+\gamma\,\mu-\gamma\,\rho\,\delta_b)}{\kappa\;(\gamma+\mu)\,(\delta_b+\mu)\,\mu\,\mu_v}\right)^2 + 4\omega} \tag{81}$$

Basing on the eigenvalues of the matrix (79), the basic reproductive number $\mathcal{R}_0$ is the spectral radius $\rho(FV^{-1})$ of the next generation matrix (79). This gives the basic reproduction number as

$$\mathcal{R}_0 = \rho\left(FV^{-1}\right) = \frac{1}{2}\left((R+W) + \sqrt{(R-W)^2+4UT}\right) \tag{82}$$

$$\mathcal{R}_0 = \frac{1}{2}\left(R + \frac{\varphi\,\rho\gamma}{(\gamma+\mu)\,(\delta_b+\mu)} + \frac{a\,(1-\rho)\,\gamma}{(\gamma+\mu)\,\mu} + \frac{dN_b\alpha_b\,(\gamma\,\delta_b+\gamma\,\mu-\gamma\,\rho\,\delta_b)}{\kappa\;(\gamma+\mu)\,(\delta_b+\mu)\,\mu\,\mu_v}\right)$$

$$+\frac{1}{2}\sqrt{\left(R - \frac{\varphi\,\rho\gamma}{(\gamma+\mu)\,(\delta_b+\mu)} - \frac{a\,(1-\rho)\,\gamma}{(\gamma+\mu)\,\mu} - \frac{dN_b\alpha_b\,(\gamma\,\delta_b+\gamma\,\mu-\gamma\,\rho\,\delta_b)}{\kappa\;(\gamma+\mu)\,(\delta_b+\mu)\,\mu\,\mu_v}\right)^2 + 4\omega} \tag{83}$$

for

$$\omega = \frac{4\;dN_b\gamma\,\alpha_c}{\kappa\,(\gamma+\mu)\,(\delta_c+\mu)\,\mu_v}\left(\frac{bN_c\,(1-\rho)\,\gamma}{N_b\,(\gamma+\mu)\,\mu} + \frac{dN_c\alpha_b\,(\gamma\,\delta_b+\gamma\,\mu-\gamma\,\rho\,\delta_b)}{\kappa\;(\gamma+\mu)\,(\delta_b+\mu)\,\mu\,\mu_v}\right)$$

From the equation (83) the basic reproduction number $\mathcal{R}_0$ is influenced by parameters from all subpopulations of the model.

Term	**Description**
$\frac{\gamma}{(\mu+\delta_c)}$	Is the probabilities that village survives in the presence of ND
$\frac{\gamma}{(\mu+\delta_b)}$	Is the probabilities that wild birds survive in the presence of ND
$\frac{\psi}{(\mu+\delta_c)}$	is the probability of village chicken to acquire NDV when come into contact with the infectious village chicken
$\frac{\varphi}{(\mu+\delta_b)}$	is the probability of wild birds to acquire NDV when come into contact with

Continued on next page

Table 3 – *Continued from previous page*

Term	Description
$\frac{d}{\kappa(\gamma+\mu)}$	chronically affected wild bird The probabilities of village chicken and wild birds to acquire the NDV from the environment during the outbreak of disease.

3.2.8 Local Stability of the Disease Free Equilibrium Point

The stability analysis of the disease free equilibrium point (ϕ_0) of the model system $(22a)$ to (24) is examined by the Hurwitz Matrix criterion (Fallat and Johnson, 2011; Dyachenko, 2014). From the Jacobian matrix $J(\phi_0)$ is found by differentiating each equation of the model system with respect to its state variables at ϕ_0. Thus, the Jacobian matrix of the model system at ϕ_0 is then given by

$$J(\phi_0) = \begin{pmatrix} -\mu & 0 & -\psi & 0 & 0 & 0 & -b\frac{N_c}{N_b} & -d\frac{N_c}{N_b} \\ 0 & -\mu-\gamma & \psi & 0 & 0 & 0 & b\frac{N_c}{N_b} & d\frac{N_c}{N_b} \\ 0 & \gamma & -\delta_c-\mu & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\mu & 0 & -\varphi & -a & -d\frac{N_b}{\kappa} \\ 0 & 0 & 0 & 0 & -\mu-\gamma & \varphi & a & d\frac{N_b}{\kappa} \\ 0 & 0 & 0 & 0 & \rho\gamma & -\delta_b-\mu & 0 & 0 \\ 0 & 0 & 0 & 0 & (1-\rho)\gamma & 0 & -\mu & 0 \\ 0 & 0 & \alpha_c & 0 & 0 & \alpha_b & \alpha_b & -\mu_v \end{pmatrix} \tag{84}$$

From matrix (84) the first two roots of $J(\phi_0)$ are given by $(-u-\lambda)(-u-\lambda)=0$. Then the reduced 6×6 matrix become:

$$\xi=\begin{pmatrix} -\mu-\gamma & \psi & 0 & 0 & b\frac{N_c}{N_b} & d\frac{N_c}{N_b} \\ \gamma & -\delta_c-\mu & 0 & 0 & 0 & 0 \\ 0 & 0 & -\mu-\gamma & \varphi & a\frac{N_b}{\kappa} & d\frac{N_b}{\kappa} \\ 0 & 0 & \rho\gamma & -\delta_b-\mu & 0 & 0 \\ 0 & 0 & (1-\rho)\gamma & 0 & -\mu & 0 \\ 0 & \alpha_c & 0 & \alpha_b & \alpha_b & -\mu_v \end{pmatrix} \tag{85}$$

Then characteristic polynomial for the matrix ξ is

$$G(\lambda)=\lambda^6+a_1\lambda^5+a_2\lambda^4+a_3\lambda^3+a_4\lambda^2+a_5\lambda+a_6 \tag{86}$$

The corresponding Hurwitz matrix is

$$G_6=\begin{pmatrix} a_1 & a_3 & a_5 & 0 & 0 & 0 \\ 1 & a_2 & a_4 & a_6 & 0 & 0 \\ 0 & a_1 & a_3 & a_5 & 0 & 0 \\ 0 & 1 & a_2 & a_4 & a_6 & 0 \\ 0 & 0 & a_1 & a_3 & a_5 & 0 \\ 0 & 0 & 1 & a_2 & a_4 & a_6 \end{pmatrix} \tag{87}$$

where

$$a_1=\mu_v+3\,\mu+\varphi+\delta_c+\gamma$$

$$a_2=\gamma\,\rho\,t+\mu_v\,\gamma+3\,\mu_v\,\mu-\mu_v\,\varphi+\mu_v\,\delta_c+a\mu+a\delta_b+2\,\gamma\,\mu-\gamma\,\varphi-\gamma\,\psi-\gamma\,t+\gamma\,\delta_c+3\,\mu^2 - 3\,\varphi\,\mu+2\,\delta_c\,\mu-\varphi\,\delta_c$$

$$a_3=\mu_v\,\gamma\,\rho\,t+\gamma^2\rho\,t+2\,\gamma\,\mu\,\rho\,t+\gamma\,\rho\,t\delta_c+\mu_v\,a\mu+\mu_v\,a\delta_b+2\,\mu_v\,\gamma\,\mu-\mu_v\,\gamma\,\varphi-\mu_v\,\gamma\,\psi-\mu_v\,\gamma\,t + \mu_v\,\gamma\,\delta_c+3\,\mu_v\,\mu^2-3\,\mu_v\,\mu\,\varphi+2\,\mu_v\,\mu\,\delta_c-\mu_v\,\varphi\,\delta_c+\gamma\,a\mu+a\gamma\,\delta_b+3\,a\mu^2+3\,a\mu\,\delta_b + a\mu\,\delta_c+a\delta_b\,\delta_c-\gamma^2t+\gamma\,\mu^2-2\,\gamma\,\mu\,\varphi-\gamma\,\mu\,\psi-2\,\gamma\,\mu\,t+\gamma\,\mu\,\delta_c+\gamma\,\varphi\,\psi-\gamma\,\varphi\,\delta_c-\gamma\,s\alpha_c - \gamma\,t\delta_c+\mu^3-3\,\mu^2\varphi+\mu^2\delta_c-2\,\mu\,\varphi\,\delta_c$$

$$
\begin{aligned}
a_4 &= \mu_v\,\gamma^2\rho\,t + 2\,\mu_v\,\gamma\,\mu\,\rho\,t + \mu_v\,\gamma\,\rho\,t\delta_c - \gamma^3 r\rho - \gamma^2\mu\,r\rho + \gamma^2\mu\,\rho\,t - \gamma^2\psi\,\rho\,t - \gamma^2\rho\,s\alpha_b \\
&\quad + \gamma^2\rho\,t\delta_c + \gamma\,\mu^2\rho\,t + \gamma\,\mu\,\rho\,t\delta_c + \mu_v\,\gamma\,a\mu + \mu_v\,a\gamma\,\delta_b + 3\,\mu_v\,a\mu^2 + 3\,\mu_v\,a\mu\,\delta_b \\
&\quad + \mu_v\,a\mu\,\delta_c + \mu_v\,a\delta_b\,\delta_c - \mu_v\,\gamma^2 t + \mu_v\,\gamma\,\mu^2 - 2\,\mu_v\,\gamma\,\mu\,\varphi - \mu_v\,\gamma\,\mu\,\psi - 2\,\mu_v\,\gamma\,\mu\,t + \mu_v\,\gamma\,\mu\,\delta_c \\
&\quad + \mu_v\,\gamma\,\varphi\,\psi - \mu_v\,\gamma\,\varphi\,\delta_c - \mu_v\,\gamma\,t\delta_c + \mu_v\,\mu^3 - 3\,\mu_v\,\mu^2\varphi + \mu_v\,\mu^2\delta_c - 2\,\mu_v\,\mu\,\varphi\,\delta_c + 2\,\gamma\,a\mu^2 \\
&\quad - a\gamma\,\mu\,\psi + 2\,a\gamma\,\mu\,\delta_b + a\gamma\,\mu\,\delta_c - a\gamma\,\psi\,\delta_b + a\gamma\,\delta_b\,\delta_c + 3\,a\mu^3 + 3\,a\mu^2\delta_b + 2\,a\mu^2\delta_c \\
&\quad + 2\,a\mu\,\delta_b\,\delta_c + \gamma^3 r + \gamma^2\mu\,r - \gamma^2\mu\,t + \gamma^2\psi\,t - \gamma^2 t\delta_c - \gamma\,\mu^2\varphi - \gamma\,\mu^2 t + \gamma\,\mu\,\varphi\,\psi - \gamma\,\mu\,\phi\,\delta_c \\
&\quad - \gamma\,\mu\,s\alpha_c - \gamma\,\mu\,t\delta_c + \gamma\,\phi\,s\alpha_c - \mu^3\varphi - \mu^2\varphi\,\delta_c \\
a_5 &= 2\,\mu_v\,a\mu\,\delta_b\,\delta_c + \gamma^2\varphi\,\rho\,s\alpha_b + \gamma\,\mu\,\phi\,s\alpha_c - \mu_v\,\gamma^2\mu\,r\rho + \mu_v\,\gamma^2\mu\,\rho\,t - \mu_v\,\gamma^2\psi\,\rho\,t + \mu_v\,\gamma^2\rho\,t\delta_c \\
&\quad + \mu_v\,\gamma\,\mu^2\rho\,t - \mu_v\,\gamma\,a\mu\,\psi + 2\,\mu_v\,\gamma\,a\mu\,\delta_b + \mu_v\,\gamma\,a\mu\,\delta_c - \mu_v\,\gamma\,a\psi\,\delta_b + \mu_v\,\gamma\,a\delta_b\,\delta_c \\
&\quad + \mu_v\,\gamma\,\mu\,\varphi\,\psi - \mu_v\,\gamma\,\mu\,\varphi\,\delta_c - \mu_v\,\gamma\,\mu\,t\delta_c - \gamma^2\rho\,st\alpha_c - \gamma\,a\mu\,s\alpha_c - \gamma\,as\alpha_c\,\delta_b - 2\,\gamma^2\mu\,\rho\,s\alpha_b \\
&\quad - \gamma\,\mu\,s\alpha_b\,\delta_b - a\gamma\,\mu\,\psi\,\delta_b + a\gamma\,\mu\,\delta_b\,\delta_c + \mu_v\,\gamma\,\mu\,\rho\,t\delta_c + 3\,\mu_v\,a\mu^3 - \mu_v\,\mu^3\varphi + \mu_v\,\gamma^3 r + \gamma^3 s\alpha_b \\
&\quad + a\gamma\,\mu^3 + a\mu^3\delta_b + a\mu^3\delta_c + a\mu^4 - \gamma^2 s\alpha_b\,\delta_b - \gamma\,\mu^2 s\alpha_b + a\gamma^3 r\rho^2 - a\gamma^3 r\rho - a\gamma\,\mu^2\psi + a\gamma\,\mu^2\delta_b \\
&\quad + a\gamma\,\mu^2\delta_c + 3\,\mu_v\,a\mu^2\delta_b + 2\,\mu_v\,a\mu^2\delta_c - \mu_v\,\mu^2\varphi\,\delta_c - \gamma^3\rho\,s\alpha_b - \mu_v\,\gamma^3 r\rho + \mu_v\,\gamma^2\mu\,r \\
&\quad - \mu_v\,\gamma^2\mu\,t + \mu_v\,\gamma^2\psi\,t - \mu_v\,\gamma^2 t\delta_c + 2\,\mu_v\,\gamma\,a\mu^2 - \mu_v\,\gamma\,\mu^2\varphi - \mu_v\,\gamma\,\mu^2 t + \gamma^2 st\alpha_c + a\mu^2\delta_b\,\delta_c \\
a_6 &= \mu_v\,a\mu^2\delta_b\,\delta_c - \gamma^3\rho^2 st\alpha_b + \gamma^3\rho\,st\alpha_b - \gamma\,a\mu^2 s\alpha_c + \mu_v\,\gamma^3 ar\rho^2 - \mu_v\,\gamma^3 ar\rho - \mu_v\,\gamma\,a\mu^2\psi \\
&\quad + \mu_v\,\gamma\,a\mu^2\delta_b + \mu_v\,\gamma\,a\mu^2\delta_c - \gamma\,a\mu\,s\alpha_c\,\delta_b - \mu_v\,\gamma\,a\mu\,\psi\,\delta_b + \mu_v\,\gamma\,a\mu\,\delta_b\,\delta_c + \gamma^2\mu\,\varphi,\rho\,s\alpha_b + \mu_v\,a\mu^4 \\
&\quad + \mu_v\,a\mu^3\delta_b + \mu_v\,a\mu^3\delta_c + \mu_v\,\gamma\,a\mu^3 - \alpha_b\,\gamma^2\mu^2 s - \alpha_b\,\gamma\,\mu^3 s - \alpha_b\,\gamma^2\mu\,s\delta_b - \alpha_b\,\gamma\,\mu^2 s\delta_b \\
&\quad + \gamma^3 a\rho^2 s\alpha_b - \gamma^3 a\rho\,s\alpha_b
\end{aligned}
$$

The disease free equilibrium point is locally asymptotically stable iff the principal leading minors of G_n are all positive for $n = 1, 2, .., 6$. Thus

$$\Delta G_1 = a_1 = \mu_v + 3\,\mu + \phi + \delta_c + \gamma > 0$$

$$\Delta G_2 = \begin{vmatrix} a_1 & a_3 \\ 1 & a_2 \end{vmatrix} = a_1 a_2 - a_3$$

$$\Delta G_3 = \begin{vmatrix} a_1 & a_3 & a_5 \\ 1 & a_2 & a_4 \\ 0 & a_1 & a_3 \end{vmatrix} = a_1 a_2 a_3 - a_1^2 a_4 - a_3^2 + a_1 a_5$$

$$\Delta G_4 = \begin{vmatrix} a_1 & a_3 & a_5 & 0 \\ 1 & a_2 & a_4 & a_6 \\ 0 & a_1 & a_3 & a_5 \\ 0 & 1 & a_2 & a_4 \end{vmatrix} = a_1 a_2 \left(a_3 a_4 - a_2 a_4 + a_5\right) + a_3 \left(a_2^2 - a_3\right)$$
$$- a_3 \left(a_3 a_4 - a_2 a_5 + a_1 a_4\right) + a_5^2$$

$$\Delta G_5 = \begin{vmatrix} a_1 & a_3 & a_5 & 0 & 0 \\ 1 & a_2 & a_4 & a_6 & 0 \\ 0 & a_1 & a_3 & a_5 & 0 \\ 0 & 1 & a_2 & a_4 & a_6 \\ 0 & 0 & a_1 & a_3 & a_5 \end{vmatrix} = a_1 a_2 a_3 \left(a_4 a_5 - a_3 a_6\right) - a_1 a_2 a_5 \left(a_2 a_5 - a_1 a_6\right)$$
$$- a_1 a_4 \left(a_4 a_5 - a_3 a_6\right) + a_4 a_5^2 + a_1 a_6 \left(a_2 a_5 - a_1 a_6 - a_3 a_6\right) + a_5^2$$

$$\Delta G_6 = \begin{vmatrix} a_1 & a_3 & a_5 & 0 & 0 & 0 \\ 1 & a_2 & a_4 & a_6 & 0 & 0 \\ 0 & a_1 & a_3 & a_5 & 0 & 0 \\ 0 & 1 & a_2 & a_4 & a_6 & 0 \\ 0 & 0 & a_1 & a_3 & a_5 & 0 \\ 0 & 0 & 1 & a_2 & a_4 & a_6 \end{vmatrix} = a_1a_2a_3a_4a_5a_6 - a_1^3a_6^3 + a_6^2a_3^3 - a_6a_5^3 + 2a_6a_1a_4a_5^2$$

$$- a_4^2a_1^2a_6a_5 - 3a_6^2a_1a_5a_3 + 2a_6^2a_1^2a_5a_2 - a_4a_3^2a_5a_6 + a_4a_3a_6^2a_1^2 + a_3a_2a_6a_5^2 - a_6a_1a_2^2a_5^2$$

$$- a_2a_1a_6^2a_3^2$$

Therefore the disease free equilibrium point (ϕ_0) of a model system (84) is LAS only if $\Delta G_1, \Delta G_2, ..., \Delta G_6 > 0$. For $\Delta G_1 > 0$. We have $\mu_v + 3\,\mu + \delta_c + \gamma + \phi > 0$, $\Delta G_2 > 0$ if $a_1a_2 > a_3$, $\Delta G_3 > 0$ if $a_1a_2a_3 + a_1a_5 > a_1^2a_4 + a_3^2$. Also ΔG_4, ΔG_5 and ΔG_6 are grater than zero when $a_1a_2a_3a_4 + a_1a_2a_5 + a_3a_2^2 + a_1a_2a_4 + a_5^2 > a_1a_2^2a_4 + a_3^2 + a_3^2a_4 + a_1a_3a_4$; $a_1a_2a_3a_4a_5 + a_1^2a_2a_5a_6 + a_1a_3a_4a_6 + a_4a_5^2 + a_1a_2a_5a_6 + a_5^2 > a_1a_2^2a_3^2a_6 + a_1a_2^2a_5^2 + a_1a_4^2a_5 + a_1a_6^2 + a_1a_3a_6^2$ and $a_1a_2a_3a_4a_5a_6 + a_6^2a_3^3 + 2a_6a_1a_4a_5^2 + 2a_6^2a_1^2a_5a_2 + a_4a_3a_6^2a_1^2 + a_3a_2a_6a_5^2 + a_1^3a_6^3 > a_6a_5^3 + a_4^2a_1^2a_6a_5 + 3a_6^2a_1a_5a_3 + a_4a_3^2a_5a_6 - a_6a_1a_2^2a_5^2 + a_2a_1a_6^2a_3^2$ respectively. We therefore establish a theorem:

Theorem 3.5

$G(\lambda)$ is stable if and only if the leading principal minors of G_n $(for\ n \in \mathbb{R}^+)$ are all positive and thus the disease free equilibrium point is LAS.

3.2.9 Global Stability of the Disease Free Equilibrium Point

The global stability of the disease free equilibrium point of the Newcastle model is done by the theorem as described by Castillo-Chavez *et al.* (2002), Mafuta *et al.* (2013) and Mwanga *et al.* (2014). To apply the theorem, we write the model system $(22a)$ to (24) as:

$$\begin{aligned} \frac{dX\,(t)}{dt} &= F\,(X, \mathcal{I}) \\ \frac{d\mathcal{I}\,(t)}{dt} &= G\,(X, \mathcal{I})\,, G\,(X, 0) = 0 \end{aligned} \tag{88}$$

where X is the number of susceptible populations and $\mathcal{I}$ is the number of the infected populations whilst the disease free equilibrium point is given given by $\phi_0 = \{x^*, 0\}$. For the system (88) to be GAS, two conditions must be fulfilled:

(i) $\frac{dX(t)}{dt} = F(X, 0)$, X^* is globally asymptotically stable (GAS)

(ii) $G(X, \mathcal{I}) = B\mathcal{I} - \widehat{G}(X, \mathcal{I})$, $\hat{G}(X, \mathcal{I}) \geq 0$ for $(X, \mathcal{I}) \in \mathcal{D}$,

where $\mathcal{D}$ is the invariant region and $B = D_r G(X^*, 0)$ is an M -matrix with non-negative off diagonal elements. If the system (88) satisfies condition (i) and (ii) above then the theorem below holds:

Theorem 3.6

A disease free equilibrium point (ϕ_0) of a model is globally asymptotically stable if and only if $\mathcal{R}_0 < 1$ (LAS) and that condition (i) and (ii) holds.

Proof:

We need to show that condition I and II holds when $\mathcal{R}_0 < 1$. From the model system in equation $(22a)$ to (24); the set of non-infectious classes is given by $X = (S_c, S_b) \in \mathbb{R}^2$ and for the infectious classes is given by $\mathcal{I} = (E_c, I_c, E_b, I_b, I_r, H) \in \mathbb{R}^6$. The model system $(22a)$ to (24) is then transfered into the form of the system (88) as follows:

$$\frac{dX(t)}{dt} = F(X, 0) = \begin{pmatrix} \mu N_c(t) - \mu S_c(t) \\ \mu N_b(t) - \mu S_b(t) \\ 0 \end{pmatrix} \tag{89}$$

with $\phi_0 = \{N_c(t), 0, 0, N_b(t), 0, 0, 0, 0\}$. The system (89) is linear with the solutions $S_c(t) = N_c(t) + (S(0) - N_c(t))e^{-\mu t}$ and $S_b(t) = N_b(t) + (S_b(0) - N_b(t))e^{-\mu t}$. It is obvious that $S_c(t) \to N_c(t)$ and $S_b(t) \to N_b(t)$ as $t \to \infty$ depending on the value of initial conditions.

Thus, ϕ_0 is globally asymptotically stable and therefore condition I holds. At the meantime

$$\frac{d\mathcal{I}(t)}{dt} = G(X,\mathcal{I}) = \begin{pmatrix} \left(\psi\frac{I_c(t)}{N_c}(t) + b\frac{I_r(t)}{N_b}(t) + \frac{dH(t)}{\kappa+H(t)}\right)S_c(t) - (\mu+\gamma)E_c(t) \\ \gamma E_c(t) - (\delta_c+\mu)I_c(t) \\ \left(\frac{\varphi I_b(t)+aI_r(t)}{N_b} + \frac{dH(t)}{\kappa+H(t)}\right)S_b(t) - (\gamma+\mu)E_b(t) \\ \rho\gamma E_b(t) - (\delta_b+\mu)I_b(t) \\ (1-\rho)\gamma E_b(t) - \mu I_r(t) \\ \alpha_c I_c(t) + \alpha_b\left(I_b(t)+I_r(t)\right) - \mu_v H(t) \end{pmatrix} \tag{90}$$

We need to show that $G(X,\mathcal{I}) = B\mathcal{I} - \widehat{G}(X,\mathcal{I})$, $G(X,0) \geq 0$ for $(X,\mathcal{I}) \in \mathcal{D}$. The Jacobian matrix of equation (90) at ϕ_0 produce an M-matrix B as follows:

$$B = \begin{pmatrix} -(\mu+\gamma) & \psi & 0 & 0 & b\frac{N_c}{N_b} & d\frac{N_c}{\kappa} \\ \gamma & -(\delta_c+\mu) & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\gamma+\mu) & \varphi & a & d\frac{N_b}{\kappa} \\ 0 & 0 & \rho\gamma & -(\delta_b+\mu) & 0 & 0 \\ 0 & 0 & (1-\rho)\gamma & 0 & -\mu & 0 \\ 0 & \alpha_c & 0 & \alpha_b & \alpha_b & -\mu_v \end{pmatrix} \tag{91}$$

and

$$\begin{Bmatrix} \widehat{G}_1(X,\mathcal{I}) \\ \widehat{G}_2(X,\mathcal{I}) \\ \widehat{G}_3(X,\mathcal{I}) \\ \widehat{G}_4(X,\mathcal{I}) \\ \widehat{G}_5(X,\mathcal{I}) \\ \widehat{G}_6(X,\mathcal{I}) \end{Bmatrix} = \begin{Bmatrix} \psi I_c\left(1-\frac{S_c}{N_c}\right) \\ 0 \\ \varphi I_b\left(1-\frac{S_b}{N_b}\right) + aI_r\left(1-\frac{S_b}{N_b}\right) \\ 0 \\ 0 \\ 0 \end{Bmatrix} \tag{92}$$

From the equation (91), a matrix B comprises with all negative diagonal entries and all non-negative off-diagonal entries. Also, by examining equation (92) we find that $\widehat{G}_1(X,\mathcal{I}) > 0$ and $\widehat{G}_3(X,\mathcal{I}) > 0$ whilst $\widehat{G}_2(X,\mathcal{I})$=$\widehat{G}_4(X,\mathcal{I})$=$\widehat{G}_5(X,\mathcal{I})$=$\widehat{G}_6(X,\mathcal{I}) = 0$. At the disease free equilibrium point $\widehat{G}_i(X,0) = 0$ for $(i = 1,2,...6)$. Since $N_c(t) = S_c(t) + E_c(t) + I_c(t)$ and $N_b(t) = S_b(t) + E_b(t) + I_b(t) + I_r(t)$, it is almost surely that $S_c(0) \leq N_c(t)$ and $S_b(0) \leq N_b(t)$ for $\{(S_c(t), S_b(t)\} \in \mathcal{D}$. Therefore condition II holds which shows that the disease free equilibrium point ϕ_0 is GAS for $\mathcal{R}_0 < 1$ and hence the theorem (54) holds.

3.2.10 Stability Analysis of Endemic Equilibrium Point

The global stability of the endemic equilibrium point (EEP) of the model is explored by using the Lyapunov method and the LaSalle's Invariant principle. To prove the global stability of point ϕ^*, lets consider a continuous and differentiable Lyapunov function defined as:

$$\mathcal{P}(t) = \sum_{n=1}^{8} \mathcal{T}_i(t)\left(y_i - y_i^* \ln y_i\right), \mathcal{T}_i > 0 \tag{93}$$

where $\mathcal{T}_i(t)$ is a Lyapunov factor, y_i a population variable at compartment i and y_i^* is the equilibrium point of the model at compartment i for $i = (1, 2, .., 8)$; where $y = y_1, y_2, ...y_8\}$ with $y_1 = S_c$, $y_2 = E_c$, $y_3 = I_c$, $y_4 = S_b$, $y_5 = E_b$, $y_6 = I_b$, $y_7 = I_r$, $y_8 = H$. From equation (93), the Lyapunov function $L(t)$ can be written as follows:

$$\begin{aligned}\mathcal{P}(S_c, E_c, I_c, S_b, E_b, I_b, I_r, H) = {} & \mathcal{T}_1\left(y_1 - y_1^* \ln y_1\right) + \mathcal{T}_2\left(y_2 - y_2^* \ln y_2\right) + \mathcal{T}_3\left(y_3 - y_3^* \ln y_3\right) \\ & + \mathcal{T}_4\left(y_4 - y_4^* \ln y_4\right) + \mathcal{T}_5\left(y_5 - y_5^* \ln y_5\right) + \mathcal{T}_6\left(y_6 - y_6^* \ln y_6\right) \\ & + \mathcal{T}_7\left(y_7 - y_7^* \ln y_7\right) + \mathcal{T}_8\left(y_8 - y_8^* \ln y_8\right)\end{aligned} \tag{94}$$

Since a function $\mathcal{P}$ is differentiable then from equation (93) the time derivative of $\mathcal{P}(t)$ along the solution of the model system in equation $(22a)$ to (24) is:

$$\begin{aligned}\frac{d\mathcal{P}(t)}{dt} = {} & \mathcal{T}_1(t)\left(1 - \frac{y_1^*}{y_1}\right)\frac{dy_1}{dt} + \mathcal{T}_2(t)\left(1 - \frac{y_2^*}{y_2}\right)\frac{dy_2}{dt} + \mathcal{T}_3(t)\left(1 - \frac{y_3^*}{y_3}\right)\frac{dy_3}{dt} \\ & + \mathcal{T}_4(t)\left(1 - \frac{y_4^*}{y_4}\right)\frac{dy_4}{dt} + \mathcal{T}_5(t)\left(1 - \frac{y_5^*}{y_5}\right)\frac{dy_5}{dt} + \mathcal{T}_6(t)\left(1 - \frac{y_6^*}{y_6}\right)\frac{dy_6}{dt} \\ & + \mathcal{T}_7(t)\left(1 - \frac{y_7^*}{y_7}\right)\frac{dy_7}{dt} + \mathcal{T}_8(t)\left(1 - \frac{y_8^*}{y_8}\right)\frac{dy_8}{dt}\end{aligned} \tag{95}$$

At the equilibrium point (y^*) we have

$$\begin{aligned}\mu N_c &= \lambda_1 y_1^*, \\ \lambda_1 &= \left(\psi\frac{I_c(t)}{N_c} + b\frac{I_r(t)}{N_b} + \frac{dH(t)}{\kappa + H(t)} + \mu\right), \\ \mu + \gamma &= \frac{\lambda_2 y_1^*}{y_2^*}, \\ \lambda_2 &= \left(\psi\frac{I_c(t)}{N_c} + b\frac{I_r(t)}{N_b} + \frac{dH(t)}{\kappa + H(t)}\right), \\ \mu + \delta_c &= \frac{\gamma y_2^*}{y_3^*},\end{aligned}$$

$$\mu N_b = \lambda_3 y_4^*,$$
$$\lambda_3 = \left(\frac{\varphi I_b(t) + aI_r(t)}{N_b} + \frac{dH(t)}{\kappa + H(t)} + \mu\right)$$
$$\gamma + \mu = \frac{\lambda_4 y_4^*}{y_5^*},$$
$$\lambda_4 = \left(\frac{\varphi I_b(t) + aI_r(t)}{N_b} + \frac{dH(t)}{\kappa + H(t)}\right)$$
$$\delta_b + \mu_b = \frac{\rho\gamma_b y_5^*}{y_6^*},$$
$$\mu = \frac{(1-\rho)\gamma_b y_5^*}{y_7^*},$$
$$\mu_v = \frac{\alpha_c y_3^* + \alpha_b\left(y_6^* + y_7^*\right)}{y_8^*}.$$

By substituting y^* into equation (95) and through simplifications, we then have

$$\begin{aligned}\frac{d\mathcal{P}(t)}{dt} &= -\mathcal{T}_1(t)\lambda_1 y_1\left(1-\frac{y_1^*}{y_1}\right)^2 + \mathcal{T}_2(t)\lambda_2 y_1\left(1-\frac{y_2^*}{y_2}\right)\left(1-\frac{y_1^*}{y_1 y_2^*}\right)\\ &+ \mathcal{T}_3(t)\gamma y_2\left(1-\frac{y_3^*}{y_3}\right)\left(1-\frac{y_2^*}{y_2 y_3^*}\right) - \phi_4(t)\lambda_3 y_4\left(1-\frac{y_4^*}{y_4}\right)^2\\ &+ \mathcal{T}_5(t)\lambda_4 y_4\left(1-\frac{y_5^*}{y_5}\right)\left(1-\frac{y_4^* y_5}{y_4 y_5^*}\right) + \mathcal{T}_6(t)\rho\gamma y_5\left(1-\frac{y_6^*}{y_6}\right)\left(1-\frac{y_5}{y_5 y_6^*}\right)\\ &+ \mathcal{T}_7(t)(1-\rho)\gamma y_5\left(1-\frac{y_7^*}{y_7}\right)\left(1-\frac{y_5^*}{y_7^*}\right) + \mathcal{T}_8(t)\left(1-\frac{y_8^*}{y_8}\right)\left(1-\frac{\alpha_c y_3^* + \alpha_b\left(y_6+y_7\right)}{\alpha_c y_3 + \alpha_b\left(y_6+y_7\right)y_8^*}\right)\end{aligned} \tag{96}$$

Thus from the equation (96)

$$\frac{d\mathcal{P}(t)}{dt} = r + s \tag{97}$$

where

$$\begin{aligned}r &= \mathcal{T}_2(t)\lambda_2 y_1\left(1-\frac{y_2^*}{y_2}\right)\left(1-\frac{y_1^*}{y_1 y_2^*}\right) + \mathcal{T}_3(t)\gamma y_2\left(1-\frac{y_3^*}{y_3}\right)\left(1-\frac{y_2^*}{y_2 y_3^*}\right)\\ &+ \mathcal{T}_5(t)\lambda_4 y_4\left(1-\frac{y_5^*}{y_5}\right)\left(1-\frac{y_4^* y_5}{y_4 y_5^*}\right) + \mathcal{T}_6(t)\rho\gamma y_5\left(1-\frac{y_6^*}{y_6}\right)\left(1-\frac{y_5}{y_5 y_6^*}\right)\\ &+ \mathcal{T}_7(t)(1-\rho)\gamma y_5\left(1-\frac{y_7^*}{y_7}\right)\left(1-\frac{y_5^7}{y_7^*}\right) + \mathcal{T}_8(t)\left(1-\frac{y_8^*}{y_8}\right)\left(1-\frac{\alpha_c y_3^* + \alpha_b\left(y_6+y_7\right)}{\alpha_c y_3 + \alpha_b\left(y_6+y_7\right)y_8^*}\right)\end{aligned}$$

and

$$s = -\mathcal{T}_1(t)\lambda_1 y_1\left(1-\frac{y_1^*}{y_1}\right)^2 - \mathcal{T}_4(t)\lambda_3 y_4\left(1-\frac{y_4^*}{y_4}\right)^2 \tag{98}$$

From the equation (97) and (98), the global stability holds only if $\frac{d\mathcal{P}(t)}{dt} \leq 0$. Now if $r < s$ then $\frac{d\mathcal{P}(t)}{dt}$ will be negative definite which implies that $\frac{d\mathcal{P}(t)}{dt} < 0$. But $\frac{d\mathcal{P}(t)}{dt} = 0$ if and only if $y_i = y_i^*$ for $(i = 1, 2, ...8)$. Hence the largest invariant set $\left\{y_1^*, y_2^*, ...y_8^* \in \mathcal{D} : \frac{d\mathcal{P}(t)}{dt} = 0\right\}$ is a singleton $\{y^*\}$. By the LaSalle's invariant principle(La Salle, 1976), it is then implies that y^* is globally asymptotically stable in $\mathcal{D}$ if $r < s$ and thus $\mathcal{R}_0 > 1$. We then establish the theorem below:

Theorem 3.7

The Endemic Equilibrium Point (EEP) of a ND $(22a)$ to (24) is globally asymptotically stable if and only if $R_0 > 1$.

3.3 Newcastle Disease model with Interventions

In this section, the Newcastle disease basic model as described in equation $(22a)$ to (24) is extended to include time dependent control terms $u_i(t)$ for $(i = 1, 2, 3)$ aiming at increasing the population of healthy chicken, reducing the number of the infected population of the village chicken as well as the Newcastle disease virus from the environment. The variable $u_1(t)$ represent the control efforts to reduce infections to the susceptible chicken through vaccination, $u_2(t)$ represents the control efforts to reduce the contacts of the infected chicken with the susceptible chicken by the culling strategy, and $u_3(t)$ represents the control of NDV from the environment through improving of the environmental hygiene. It is assumed that all control variables $u_i(t)$ are Lebesgue measurable such that $0 \leq u_1 \leq 1$, $0 \leq u_2 \leq 1$ and $0 \leq u_3 \leq 1$. In the new model, the terms $u_1(t) S_c$ and $u_2(t) I_c(t)$ represents the vaccination of the susceptible chicken and the culling of the infected chicken respectively.

The Village chicken population is now divided into four subpopulations: the susceptible chicken $S_c(t)$, the latently infected $E_c(t)$, the severely infected chicken $I_c(t)$ and the vaccinated village chicken population $V(t)$. Thus, the total village chicken population become $N_c(t) = S_c(t) + E_c(t) + I_c(t) + V(t)$. We assume that the susceptible village chicken are recruited by the density dependent birth rate μN_c and the chicken with low immunity that reverted back from the vaccinated population at the rate $\phi V(t)$. Chicken at the susceptible population acquires Newcastle disease virus when interacts with the mildly infected wild birds, $I_r(t)$, the severely infected chicken, $I_c(t)$ and the unhygienic environment, $H(t)$ and moves to the latently infected

class at the rate $\lambda_1 (I_c, I_r, H)$ defined by

$$\lambda_1 (I_c, I_r, H) = \left(\psi \frac{I_c S_c}{N_c} + b \frac{I_r S_c}{N_b} + \frac{dH}{k + H} \right) S_c \tag{99}$$

After some days, individuals in the latently infected population of the village chicken progress to the severely infected population at the rate γE_c. The severely infected population of village chicken is reduced at the rate $(\mu + \delta_c + u_2) I_c$. In the model we assume that the mortality of NDV in the environment is increased by the rate $(\mu_v + u_3) H(t)$. The wild birds population is also divided into four subpopulations: the susceptible population $S_b (t)$, the latent population $E_b (t)$, the severely infected wild bird population, $I_b (t)$ and the mildly infected wild bird population, $I_r (t)$, which gives its total population as $N_b (t) = S_b (t) + E_b (t) + I_b (t) + I_r (t)$. The susceptible wild bird population is recruited by the time dependent rate μN_b through birth. However, the susceptible village chicken acquires NDV when interacts with the severely infected wild birds, $I_b (t)$, the mildly infected wild birds, $I_r (t)$, and the unhygienic environment, $E (t)$ and moves to the latently infected class at the transmission rate $\lambda_2 (I_b, I_r, H)$ defined by

$$\lambda_2 (I_b, I_r, H) = \left(\frac{\varphi I_b(t) + a I_r(t)}{N_b} + \frac{dH(t)}{\kappa H(t)} \right) S_b(t) \tag{100}$$

After some days depending on the status of the wild birds in the latently infected population, a proportion ρ of the latently infected wild bird population progress to the severely infected population and the remained proportion, $1 - \rho$, progress to the mildly infected population of wild birds. The model assumes that village chicken and wild bird do not recover after getting sick from the Newcastle disease but dies due to disease induced death at the rate δ_c and δ_b respectively. The mildly infected wild bird population does have disease induced mortality, its assumed that they only die naturally. The rest of population dies at the same natural death μ. The environment has only one class denoted by a variable $H (t)$. Other parameters of the model are described in Table (2). All variables are assumed to be non-negative. Due to the complex nature of interactions between the village chicken population and the wild birds, we are not introducing the control variables in the wild bird population but the model assumes that, the environment is the factor which brings the two population together. The general interactions between village chicken, wild birds and NDV infested environment with control measures are presented by the schematic flow diagram in Fig.5 and the non-linear differential equations describing the model are given in model system (101) to (103).

3.3.1 Model Flow Diagram

Based on the assumptions made on the optimal control, the schematic diagram of the ND with controls is summarized in the flow diagram as follows:

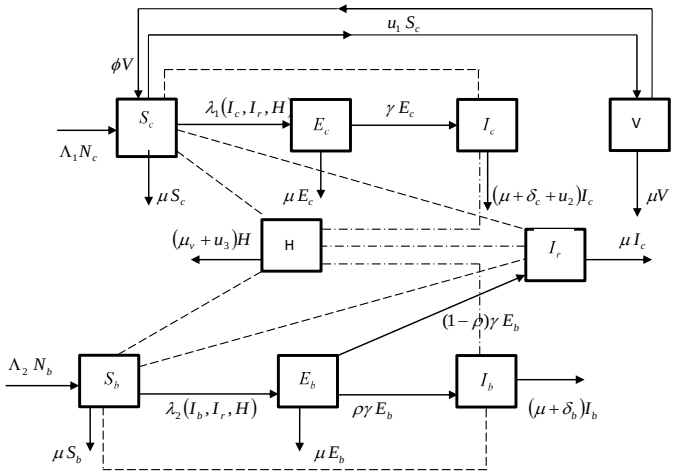


Figure 5: The flow chart showing the dynamics of ND with vaccination, culling and environmental hygiene and sanitation control measures.

From the above assumptions and the model flowchart, the following dynamical system with control measures u_1, u_2 and u_3 is formulated as follow:

Chicken

$$\frac{dS_c(t)}{dt} = \mu N_c + \phi V - \left(\psi \frac{I_c(t)}{N_c} + b\frac{I_r(t)}{N_b} + \frac{dH(t)}{\kappa + H(t)} + \mu\right) S_c(t) - u_1 S_c(t) \tag{101a}$$

$$\frac{dE_c(t)}{dt} = \left(\psi \frac{I_c(t)}{N_c} + b\frac{I_r(t)}{N_b} + \frac{dH(t)}{\kappa + H(t)}\right) S_c(t) - (\mu + \gamma) E_c(t) \tag{101b}$$

$$\frac{dI_c(t)}{dt} = \gamma E_c(t) - (\delta_c + \mu + u_2) I_c(t) \tag{101c}$$

$$\frac{dV(t)}{dt} = u_1(t) S_c(t) - (\mu + \phi) V(t) \tag{101d}$$

Wild birds

$$\frac{dS_b(t)}{dt} = \mu N_b - \left(\frac{\varphi I_b(t) + aI_r(t)}{N_b} + d\frac{H(t)}{\kappa + H(t)} + \mu\right) S_b(t) \tag{102a}$$

$$\frac{dE_b(t)}{dt} = \left(\frac{\varphi I_b(t) + aI_r(t)}{N_b} + d\frac{H(t)}{\kappa + H(t)}\right) S_b(t) - (\gamma + \mu)E_b(t) \tag{102b}$$

$$\frac{dI_b(t)}{dt} = \rho\gamma E_b(t) - (\delta_b + \mu)I_b(t) \tag{102c}$$

$$\frac{dI_r(t)}{dt} = (1-\rho)\gamma E_b(t) - \mu I_r(t) \tag{102d}$$

$$\frac{dH(t)}{dt} = \alpha_c I_c(t) + \alpha_b \left(I_b(t) + I_r(t)\right) - \left(\mu_v + u_3\right) H\left(t\right) \tag{102e}$$

Environment

$$\frac{dH(t)}{dt} = \alpha_c I_c(t) + \alpha_b \left(I_b(t) + I_r(t)\right) - \left(\mu_v + u_3\right) H\left(t\right) \tag{103}$$

With initial conditions,

$S_c(0) > 0$, $E_c(0) \geq 0, I_c(0) \geq 0$, $V(0) \geq 0$, $S_b(0) > 0$, $E_b(0) \geq 0$, $I_b(0) \geq 0$, $I_r(0) \geq 0$, $H(0) \geq 0$, $u_1(0) \geq 0$, $u_2(0) \geq 0$, $u_3(0) \geq 0$.

3.3.2 Formulation of the Cost Function

From the Newcastle model with controls in equation $(101) - (103)$, we use the variable $X\left(t\right)$ to represent the disease state variables and $u_i\left(t\right)$ to represent different control efforts used for reducing the spread of ND among the village chicken. Our state equation is now appear as:

$$\frac{dX}{dt} = \mathcal{M}\left(t, X(t), u_i\left(t\right)\right) \tag{104}$$

The time dependent control variable $u_i\left(t\right)$ $(i = 1, 2, 3)$ is considered on the time interval $\lceil t_0, t_f \rceil$ and allows the variable $X\left(t\right)$ to be minimized at any point in the interval. Our purpose is to minimize the number of the severely infected village chicken and the concentration of NDV in the surroundings while keeping the cost of control as low as possible. Therefore, to reach $\mathcal{J}\left(u^*\left(t\right)\right)$ at a minimum cost we formulate an optimal cost function of our problem over the optimal set of control $\mathcal{U} = \left\{u_1\left(t\right), u_2\left(t\right), u_3\left(t\right)\right\}$ as follows:

$$\mathcal{J} = \min_{u_i(t) \in \mathcal{U}} \int_{t_0}^{t_f} \left(A_1 u_1\left(t\right) S_c\left(t\right) + A_2 u_2\left(t\right) I_c\left(t\right) + A_3 u_3\left(t\right) H\left(t\right) + \frac{1}{2}\sum_{i=1}^{3} L_i u_i^2\left(t\right)\right) dt \tag{105}$$

subject to:

$$\frac{dX}{dt} = \mathcal{M}\left(t, X(t), u\left(t\right)\right), \quad t \in [t_0, t_f]$$

$$X\left(0\right) = X_0, \quad X\left(t_f\right)$$

$$u_i\left(t\right) \in \mathcal{U} : \left\{u_i\left(t\right); 0 \leq u_i\left(t\right) \leq 1\right\}$$

$$X\left(t\right) > 0, \quad u_i\left(t\right) \geq 0$$

where $\mathcal{M}\left(X\left(t\right)\right)$ denote the relative weight of the controls. In the equation (105), $A_1 S_c$, $A_2 I_c$ and $A_3 H$ are the costs associated with the control of the susceptible village chicken, severe infected wild birds and the unhygienic environment respectively, while the function $\frac{L_1}{2}u_1^2$, $\frac{L_2}{2}u_2^2$ and $\frac{L_3}{2}u_3^2$, are the additional costs associated with each control measure. We choose quadratic terms in the controls in the objective functional (105) with the assumption that the cost are in a nonlinear form and also to avoid the bang bang or singular optimal control cases (Joshi *et al.*, 2006; Kinene *et al.*, 2015; Asamoah *et al.*, 2017). Therefore, it is needed to find the optimal control $\left(u_1^*\left(t\right), u_2^*\left(t\right), u_3^*\left(t\right)\right)$ such that,

$$\mathcal{J}\left(u_i^*\left(t\right)\right) = \min_{u_i(t) \in \mathcal{U}} \left\{\mathcal{J}\left(u_i\left(t\right)\right)\right\} \tag{106}$$

Then, the Pontryagin's Maximum Principle (PMP) as described in (Lenhart and Workman, 2007; Anita *et al.*, 2011) is applied to find the optimal solution of the model (104). Firstly, the Hamiltonian function $\mathcal{H}\left(t, X\left(t\right), u\left(t\right), \lambda\left(t\right)\right)$ is formulated by introducing the adjoint function, $\lambda\left(t\right)$, which saves as the Lagrangian multiplier for our optimal control model and later the Pontryagin's Maximum Principle necessary conditions (adjoint, transvesality and the optimality conditions) are applied to find the optimal solution $\mathcal{J}\left(u^*\left(t\right)\right)$ of our model.

3.3.3 Analysis of an Optimal Control Model

Using the formulated optimal control problem (104) constrained with the control variables $\left\{u_i\left(t\right) \in \mathcal{U} |\ 0 \leq u_i\left(t\right) \leq 1\right\}, t \in [t_0, t_f]$, and the state variables $S_c\left(t\right)$, $E_c\left(t\right)$, $I_c\left(t\right)$, $V\left(t\right)$, $E_b\left(t\right)$, $I_b\left(t\right)$, $I_r\left(t\right)$, and $H\left(t\right)$ in (105), then we prove the following:

(i) Existence of the optimal controls

(ii) Characterization of the optimal control problem

(iii) Find the numerical solution of the optimal control model

(iv) And we investigate how the optimal control variables depends on various parameters of the system $(101) - (103)$ (Joshi *et al.*, 2006).

3.3.4 Existence of the Optimal Controls

An Optimal control problem exists if the five necessary conditions that defines the optimal solutions $\{u_i(t) \in \mathcal{U} |\ 0 \leq u_i(t) \leq 1\}, t \in [t_0, t_f]$ of the problem $(101) - (103)$ derived by Pontryagin's Maximum Principle are satisfied. Before proving for the existence of the optimal solution, we state the following theorem;

Theorem 3.8
Given an optimal problem $\mathcal{N}(t, X(t), u_i(t))$, subject to its initial boundary condition $t \in [t_0, t_f]$ with a state variable $X(t) \in \mathbb{R}^9$ and a control variable $u_i(t) \in \mathbb{R}^3$, then there exists an optimal solution $\mathcal{J}(u_i^*)$ such that $\mathcal{J}(u_i^*) = \min_{u_i \in \mathcal{U}} \{(\mathcal{J}(u_i)\}$ for $i = (1, 2, 3)$ if the following necessary conditions are satisfied;

(i) The set of controls and the corresponding state variables is non empty.

(ii) The control set $\mathcal{U}$ is convex and closed.

(iii) The right hand side of the state system is bounded by the linear function in the state and control variables

(iv) The integrand of the objective function is convex.

(v) There exists constants a_1, $a_2 > 0$ and $\omega > 1$ such that the integrand of the objective function is bounded below by $a_1 (|u_1| + |u_2| + |u_3|)^{\frac{\omega}{2}} - a_2$

Proof: the existence of an optimal control is verified by conditions stated in (Fleming and Rishel, 1975). From our optimal problem $\mathcal{M}((X)(t), u(t))$ in equation (104), the set of all state variables $X(t)$ and the control variables $\{u_i(t) \in \mathcal{U} |\ 0 \leq u_i(t) \leq 1\}, t \in [t_0, t_f]$ are non-negative, hence the first condition is satisfied (Kung'aro *et al.*, 2015). By definition, the optimal

solution $u_i^*(t) \in \mathbb{R}^n$ is convex and bounded in $\mathcal{U}$ and thus the second condition is also satisfied (Collins *et al.*, 2009; Mpeshe *et al.*, 2014*a*; Mlay *et al.*, 2015). sd The optimal system (104) is bounded which determines the compactness needed for the existence of the optimal control (Athithan and Ghosh, 2015) and hence the third condition holds. In addition, the integrand in the functional (105),

$$A_1 u_1(t) S_c(t) + A_2 u_2(t) I_c(t) + A_3 u_3(t) H(t) + \frac{L_1}{2} u_1^2(t) + \frac{L_2}{2} u_2^2(t) + \frac{L_3}{2} u_3^2(t)$$

is clearly convex on the control set $\mathcal{U}$ which proves the fourth condition. According to Mlay *et al.* (2015), since the state variables are bounded therefore the integrand is also bounded below as;

$$A_1 u_1(t) S_c(t) + A_2 u_2(t) I_c(t) + A_3 u_3(t) H(t) + \frac{1}{2} \sum_{i=1}^{3} L_i u_i^2(t) \geq a_1 \left(\sum_{j=1}^{5} |u_j| \right)^{\frac{\omega}{2}} - a_2 \quad (107)$$

that satisfies the last condition. With those five conditions satisfied, we therefore conclude that there exist control variables u_i^*such that, $\mathcal{J}(u_i^*(t)) = \min_{u_i(t) \in \mathcal{U}} \{(\mathcal{J}(u_i(t))\}$. And this completes the proof of the existence of the optimal control.

3.3.5 Characterization of the Optimal Control

Here the Pontryagin's Maximum Principle (PMP) is applied to derive the necessary conditions that an optimal control solutions must satisfy (Joshi *et al.*, 2006; Lenhart and Workman, 2007). The Principle is used to obtain the differential equations for the adjoint variables, corresponding boundary conditions as well as the characterization of an optimal solution $\mathcal{J}(u_i^*)$ for the optimal model (104). Characterization gives a representation of an optimal control in terms of state variables by minimizing the Hamiltonian, $\mathcal{H}(X, u, \lambda)$, with respect to the controls and the adjoint function (Namawejje *et al.*, 2015; Kung'aro *et al.*, 2015). To obtain the minimum Lagrangian of the optimal problem, we establish the Hamiltonian function $\mathcal{H}(X, u, \lambda)$, of the control problem with respect to its state variable $X(t)$, control variable $u(t)$ and the adjoint function $\lambda(t)$ as follows:

$$\mathcal{H}(X(t), u, \lambda) = A_1 u_1(t) S_c(t) + A_2 u_2(t) I_c(t) + A_3 u_3(t) H(t) + \frac{1}{2} \sum_{i=1}^{3} L_i u_i^2(t) + \sum_{j=1}^{9} \lambda_j F_j \quad (108)$$

where $\lambda_1, \lambda_2, .., \lambda_9$ stands for the adjoint functions and $F_1, F_2, ..., F_9$ stands for the coefficient of the state variable i in the i^{th} equation of the optimal control problem. Now, the expanded form of the Hamiltonian equation (108) becomes:

$$\begin{aligned}
\mathcal{H}\left(X\left(t\right), u, \lambda\right) = {} & A_1 u_1(t) S_c\left(t\right) + A_2 u_2(t) I_c\left(t\right) + A_3 u_3(t) H\left(t\right) + \frac{L_1}{2} u_1^2\left(t\right) + \frac{L_2}{2} u_2^2 + \frac{L_3}{2} u_3^2 \\
& + \lambda_1 \left(\mu N_c\left(t\right) + \varphi V\left(t\right) - \left(\psi \frac{I_c\left(t\right)}{N_c\left(t\right)} + b \frac{I_r\left(t\right)}{N_b\left(t\right)} + \frac{dH\left(t\right)}{\kappa + H\left(t\right)} + \mu + u_1\right) S_c\right) \\
& + \lambda_2 \left(\left(\psi \frac{I_c\left(t\right)}{N_c\left(t\right)} + b \frac{I_r\left(t\right)}{N_b\left(t\right)} + \frac{dH\left(t\right)}{\kappa + H\left(t\right)}\right) S_c\left(t\right) - (\mu + \gamma) E_c\left(t\right)\right) \\
& + \lambda_3 \left(\gamma E_c\left(t\right) - (\delta_c + \mu + u_2\left(t\right)) I_c\left(t\right)\right) \\
& + \lambda_4 \left(u_1(t) S_c\left(t\right) - (\mu + \varphi) V\left(t\right)\right) \\
& + \lambda_5 \left(\mu N_b\left(t\right) - \left(\frac{\phi I_b\left(t\right) + a I_r\left(t\right)}{N_b} + \frac{dH\left(t\right)}{\kappa + H\left(t\right)} + \mu\right) S_b\right) \\
& + \lambda_6 \left(\left(\frac{\varphi I_b\left(t\right) + a I_r\left(t\right)}{N_b\left(t\right)} + \frac{dH\left(t\right)}{\kappa + H\left(t\right)}\right) S_b\left(t\right) - (\gamma + \mu) E_b\right) \\
& + \lambda_7 \left(\rho \gamma E_b\left(t\right) - (\delta_b + \mu) I_b\left(t\right)\right) \\
& + \lambda_8 \left((1 - \rho) \gamma E_b\left(t\right) - \mu I_r\left(t\right)\right) \\
& + \lambda_9 \left(\alpha_c I_c(t) + \alpha_b \left(I_b\left(t\right) + I_r\left(t\right)\right) - \left(\mu_v + u_3\left(t\right)\right) H\left(t\right)\right)
\end{aligned} \tag{109}$$

Now, after having the the Hamiltonian function, $\mathcal{H}\left(X, u, \lambda\right)$, of the optimal problem, we need to find the minimum value of the Lagrangian equation.

Theorem 3.9

Given $\{u_1^*\left(t\right), u_2^*\left(t\right), u_3^*\left(t\right)\}$ be the set of the optimal control $J\left(u_i^*\left(t\right)\right)$ and S_c^*, E_c^*, I_c^*, V, S_b^*, E_b^*, I_b^*, I_r^* and E^* be the corresponding solutions of the problem that minimizes $J\left(u_i^*\right)$ over $\mathcal{U}$, then there exists an adjoint $\lambda\left(t\right)$ such that together with $X\left(t\right)$, $u\left(t\right)$ and $\lambda\left(t\right)$ satisfying the following conditions:

$$\frac{d\lambda_1}{dt} = -\frac{\partial \mathcal{H}}{\partial S_c}, \frac{d\lambda_2}{dt} = -\frac{\partial \mathcal{H}}{\partial E_c} =, ..., = \frac{d\lambda_9}{dt} = -\frac{\partial \mathcal{H}}{\partial H} \;\; (adjoint\; condition) \tag{110}$$

$$\lambda_1\left(t_f\right) = \lambda_2\left(t_f\right) =, ..., \lambda_9\left(t_f\right) = 0 \;\; (transversality\; condition) \tag{111}$$

$$\frac{\partial \mathcal{H}}{\partial u_i} = 0 \; at \; u_i^* = 0, \; j = 1, 2, 3, \; (optimality\; condition) \tag{112}$$

Proof: to prove this, the function (109) is differentiated partially $w.r.t$ to its state variables which gives the adjoint system . With the Pontryagin's Maximum Principle, we get the following adjoint system evaluated at the optimal control pair corresponding to the state variables:

$$
\begin{aligned}
\frac{d\lambda_1}{dt} &= -\frac{\partial \mathcal{H}}{\partial S_c} = -A_1 u_1^*(t) + (\lambda_1 - \lambda_3)\Delta_1 + \lambda_1(\mu + u_1^*(t) - \Lambda_1) - \lambda_4 u_1^*(t) \\
\frac{d\lambda_{E_c}}{dt} &= -\frac{\partial \mathcal{H}}{\partial E_c} = -\lambda_1\Lambda_1 - \lambda_3\gamma + (\mu+\gamma)\lambda_2 + (\lambda_1 - \lambda_3)\frac{\psi S_c^*(t)(N_c^*(t) - I_c^*(t))}{N_c^2(t)} \\
\frac{d\lambda_3}{dt} &= -\frac{\partial \mathcal{H}}{\partial I_c} = -A_2 u_2^*(t) - \lambda_1\Lambda_1 + (\lambda_1 - \lambda_3)\frac{\psi S_c^*(t)(N_c^*(t) - I_c^*(t))}{N_c^2(t)} + \lambda_3(\delta_c + \mu + u_2^*) \\
&\quad - \lambda_9\alpha_c \\
\frac{d\lambda_V}{dt} &= -\frac{\partial \mathcal{H}}{\partial V} = -\lambda_1\Lambda_1 + \mu\lambda_4 - (\lambda_1 - \lambda_4)\phi - (\lambda_1 - \lambda_2)\frac{\psi I_c^*(t) S_c^*(t)}{N_c^2(t)} \\
\frac{d\lambda_5}{dt} &= -\frac{\partial \mathcal{L}}{\partial S_b} = -\lambda_{S_b}\Lambda_2 + \mu\lambda_{S_b} + \frac{(\lambda_1 - \lambda_2) b I_r^*(t) S_c^*(t)}{N_b^2(t)} + \Delta_2 \\
\frac{d\lambda_{E_b}}{dt} &= -\frac{\partial \mathcal{H}}{\partial E_b} = -\lambda_1\Lambda_2 + \lambda_6(\gamma+\mu) - \lambda_{I_r}\gamma - (\lambda_7 - \lambda_8)\gamma\rho - \frac{\left(\lambda_{S_c^*} - \lambda_{E_c}\right) b I_r^*(t) S_c^*(t)}{N_b^2(t)} \\
&\quad - \Delta_3 \\
\frac{d\lambda_7}{dt} &= -\frac{\partial \mathcal{H}}{\partial I_b} = -\lambda_5\Lambda_2 - \lambda_9\alpha_b + \lambda_7(\delta_b + \mu) - \frac{(\lambda_1 - \lambda_2) b I_r^*(t) S_c^*(t)}{N_b^2(t)} + \Delta_4 \\
\frac{d\lambda_8}{dt} &= -\frac{\partial \mathcal{H}}{\partial I_r} = -\lambda_5\Lambda_2 - \alpha_b\lambda_H + \lambda_8\mu + \left(\frac{b S_c^*(\lambda_1 - \lambda_2)}{N_b^2}\right) \\
&\quad + (\lambda_5 - \lambda_6) S_b(t)\left(\frac{a(N_b^*(t) - I_r^*(t)) - \varphi I_b^*(t)}{N_b^2(t)}\right) \\
\frac{d\lambda_9}{dt} &= -\frac{\partial \mathcal{H}}{\partial H} = (\lambda_1 - \lambda_2)\frac{d\kappa S_c^*(t)}{(\kappa + H^*(t))^2} + (\lambda_5 - \lambda_6)\frac{d\kappa S_b^*(t)}{(\kappa + H^*(t))^2} + (\mu_v + u_3^*)\lambda_9
\end{aligned}
\tag{113}
$$

where as;

$$\Delta_1 = \left(\psi\frac{I_c^*(t)(N_c^*(t) - S_c^*(t))}{N_c^2(t)} + \frac{bI_r^*(t)}{N_c^2(t)} + \frac{dH^*(t)}{\kappa + H^*(t)}\right), \quad \Delta_2 = (\lambda_5 - \lambda_6)\left(\frac{\left(N_b^*(t) - S_b(t)\right)(\varphi I_b(t) + aI_r(t))}{N_b^2(t)}\right)$$

$$\Delta_3 = \frac{(\lambda_5 - \lambda_6)\left(\varphi I_b^*(t) + aI_r^*(t)\right)S_b^*(t)}{N_b^2(t)}, \qquad \Delta_4 = (\lambda_5 - \lambda_6) S_b(t)\left(\frac{N_b(t)\varphi - (\varphi I_b(t) + aI_r(t))}{N_b^2(t)}\right)$$

With the Pontryagin's Maximum Principle, we prove the optimality condition and find the optimal solution of the optimal control model.

Theorem 3.10

An optimal control $u_i^*(t) \in \mathbb{R}^m$ that minimizes $\mathcal{J}(u_i^*(t))$ over the region $\mathcal{U}$ is given by $u_i^*(t) = \min_{u_i(t)\in\mathcal{U}} \{max(0, u^*(t)), 1\}$

Proof: To prove this we apply the optimality condition:

$$\frac{\partial \mathcal{H}}{\partial u_i} = 0 \ \ for \ \ \ i = 1, 2, 3 \tag{114}$$

Now, using equation (109) and (112), the Lagrangian function evaluated at $u_i^*(t)$ gives the following optimal control solutions:

$$u_1^*(t) = \frac{1}{L_1}((\lambda_1 - \lambda_4) - A_1) S_c(t) \tag{115}$$

$$u_2^*(t) = \frac{1}{L_2}(\lambda_3 - A_2) I_c(t) \tag{116}$$

and

$$u_3^*(t) = \frac{1}{L_3}(\lambda_9 H_c(t) - A_3) \tag{117}$$

By using the transvesality condition $\lambda_i(t_f) = 0 \ (for \ \ i = 1, 2, ..., 9)$ and the boundedness condition of our control variables, $\mathcal{U} = \{u_i^*(t) \mid 0 \leq u_i^*(t) \leq 1\}$ (with $i = 1, 2, 3$), the characterization of optimal control, $u_i^*(t)$, is bound below by zero and by one above. Now, lets consider the control bound , $0 \leq u_1^*(t) \leq 1$, this means that $u_1^*(t)$ is bound below by zero and by one above. By using the bounds for the control $u_1(t)$, we get the following solution:

$$u_1^*(t) = \begin{cases} 0 & \text{if} \ \ \frac{1}{L_1}((\lambda_1 - \lambda_4) - A_1) S_c^*(t) \leq 0 \\ \frac{1}{L_1}((\lambda_1 - \lambda_4) - A_1) S_c^*(t) & \text{if} \ \ 0 \leq u_1^*(t) \leq 1 \\ 1 & \text{if} \ \ \frac{1}{L_1}((\lambda_1 - \lambda_4) - A_1) S_c^*(t) \geq 1 \end{cases} \tag{118}$$

Hence, $u_1^*(t)$ is explicitly expressed as:

$$u_1^*(t) = \min\left\{\max\left(0, \ \frac{1}{L_1}((\lambda_1 - \lambda_4) - A_1) S_c^*(t)\right), 1\right\} \tag{119}$$

like the boundedness in $u_1^*(t)$, the rest of the control variable will be bound as follows:

$$u_2^*(t) = \begin{cases} 0 & \text{if} \ \ \frac{1}{L_2}(\lambda_3 - A_2) I_c(t) \leq 0 \\ \frac{1}{L_2}(\lambda_3 - A_2) I_c(t) & \text{if} \ \ 0 \leq u_1^*(t) \leq 1 \\ 1 & \text{if} \ \ \frac{1}{L_2}(\lambda_3 - A_2) I_c(t) \geq 1 \end{cases} \tag{120}$$

$$u_3^*(t) = \begin{cases} 0 & \text{if } \frac{1}{L_3}\left(\lambda_9 H^*(t) - A_3\right) \leq 0 \\ \frac{1}{L_3}\left(\lambda_9 H^*(t) - A_3\right) & \text{if } 0 \leq u_1^*(t) \leq 1 \\ 1 & \text{if } \frac{1}{L_3}\left(\lambda_9 H^*(t) - A_3\right) \geq 1 \end{cases} \tag{121}$$

and the controls $u_2^*(t)$ and $u_3^*(t)$ are therefore expressed explicitly as follows:

$$\begin{aligned} u_2^*(t) &= \min\left\{\max\left(0, \quad \frac{1}{L_2}\left(\lambda_3 - A_2\right) I_c(t), 1\right)\right\} \\ u_3^*(t) &= \min\left\{\max\left(0, \quad \frac{1}{L_3}\left(\lambda_9 H^*(t) - A_3\right), 1\right)\right\} \end{aligned} \tag{122}$$

From the equation (119) and (122), it is noted that, the optimality system consists of the state equations, the adjoint system together with the initial and transvesality conditions and the optimality conditions. The optimal control solutions will be shown numerically in Chapter four of the Thesis.

3.4 Cost-Effectiveness Analysis (CEA)

In this section, the cost-effectiveness analysis (CEA) for control measures of the ND in the village chicken is done. CEA is an economic evaluation that allows the comparison of the costs and the consequence of two or more strategies that competes for the limited available resources. The results of this analysis informs decision-makers who have to plan for the allocation of the limited health care resources. Cost effectiveness analysis can be done in various ways depending on the objectives and the nature of the problem. According to (Okosun *et al.*, 2013; Kinene *et al.*, 2015; Bornaa *et al.*, 2015), there are three types of cost-effectiveness ratios:

(i) The Average Cost-Effectiveness Ratio (ACER)

This ratio deals with a single intervention and evaluates the intervention against its baseline option.

(ii) The Marginal Cost-Effectiveness Ratio (MCER)

Is used to study the specific costs and effects when a programme is expanded or contracted.

(iii) The Incremental Cost-Effectiveness Ratio (ICER) is usually used for comparing between the costs and effects of two interventions which competes under the scarcity of resources.

In this thesis, the Incremental Cost-Effectiveness Analysis is employed to analyse a couple of strategies as in the Table 4:

Table 4: A list of Control Strategies and their combinations

Variable	**Description of the control strategy**
Strategy A	Vaccination of the susceptible village chicken
Strategy B	Culling of the infected village chicken from the flock
Strategy C	Environmental hygiene
Strategy D	Vaccination of the susceptible chicken and culling of the infected chicken from the flocks
Strategy E	Vaccination of the susceptible chicken and cleanliness of the flocks
Strategy F	Culling of the infected village chicken from the flock and cleanliness of the flocks
Strategy G	Combination of vaccination, culling and environmental hygiene strategies

3.4.1 Incremental Cost-Effectiveness Analysis (ICER)

When using the ICER method, two control strategies are compared while looking for the cheaper and most cost-effective intervention to users (Okosun *et al.*, 2013; Rodrigues *et al.*, 2014; Hove-Musekwa *et al.*, 2014). The ICER is represented as the ratio of the difference in cost between two interventions to the difference in outcomes between the two interventions,

thus,

$$\text{ICER} = \frac{\text{Net change in cost}}{\text{change in the total number of infections averted}} \tag{123}$$

In the ratio (123), the net change in the intervention costs is the difference between the costs of the two interventions and the change in the number of infections averted is the difference between the total number of infection cases without control and the number of cases of the two competing control strategies.

Table 5: The Cost and Infection averted for different controls Strategies

Strategy	Infection averted	Total cost in US Dollar
No control	0	0
Strategy A	2.3234×10^9	2.1457×10^3
Strategy B	1.6624×10^9	2.0286×10^6
Strategy D	2.3405×10^9	4.9651×10^5
Strategy E	2.3319×10^9	3.5456×10^4
Strategy F	1.9659×10^9	1.3938×10^6
Strategy G	2.3488×10^9	5.0648×10^5

Table 6: The arrangement of the Control strategies in ascending order of the total Infection averted

Strategy	Infection averted	Total Cost in US Dollar
No control	0	0
Strategy B	1.6624×10^9	2.0286×10^6
Strategy F	1.9659×10^9	1.3938×10^6
Strategy A	2.3234×10^9	2.1457×10^3
Strategy E	2.3319×10^9	3.5456×10^4
Strategy D	2.3405×10^9	4.9651×10^5
Strategy G	2.3488×10^9	5.0648×10^5

From Table 6, the ICER values are computed as follows:

$$\text{ICER(Strategy B)} = \frac{2.0286 \times 10^6}{1.6624 \times 10^9} = 0.00122$$

$$\text{ICER(Strategy F)} = \frac{1.3938 \times 10^5 - 2.0286 \times 10^6}{1.9659 \times 10^9 - 1.6624 \times 10^9} = -0.0062$$

$$\text{ICER(Strategy A)} = \frac{2.1457 \times 10^3 - 1.3938 \times 10^6}{2.3405 \times 10^9 - 2.3319 \times 10^9} = -0.009389$$

$$\text{ICER(Strategy D)} = \frac{5.0648 \times 10^5 - 4.9651 \times 10^5}{2.3488 \times 10^9 - 2.3405 \times 10^9} = 2.82 \times 10^{-7}$$

$$\text{ICER(Strategy G)} = \frac{1.3525 \times 10^6 - 1.3495 \times 10^6}{2.3488 \times 10^9 - 2.3234 \times 10^9} = 0.00012$$

$$\text{ICER(Strategy E)} = \frac{1.958 \times 10^4 - 1.3525 \times 10^6}{2.3319 \times 10^9 - 2.3488 \times 10^9} = 0.07887$$

Table 7: The Cost and Infection averted for Control B, F, A, D, G and E

Strategy	Infection averted	Total Cost in US Dollar	ICER
No control	0	0	-
Strategy B	1.6624×10^9	3.7193×10^6	0.00122
Strategy F	1.9659×10^9	3.484×10^6	-0.0062
Strategy A	2.3234×10^9	5.6662×10^3	-0.0094
Strategy D	2.3405×10^9	1.3495×10^6	2.82×10^{-7}
Strategy G	2.3488×10^9	1.3525×10^6	0.00012
Strategy E	2.3319×10^9	1.9580×10^4	0.07887

Comparing strategy B and strategy F, the ICER of strategy F is less than ICER of strategy B. Hence strategy B is more costly and less effective than strategy F. Therefore we omit strategy B and recalculate ICER again for the remaining strategies.

Table 8: The Cost and Infection averted for Control F, A, D, G and E

Strategy	Infection averted	Total Cost in US Dollar	ICER
Strategy F	1.9659×10^9	1.3938×10^6	0.00071
Strategy A	2.3234×10^9	2.1457×10^3	-0.00389
Strategy E	2.3319×10^9	3.5456×10^4	0.00392
Strategy D	2.3405×10^9	4.9651×10^5	0.05361
Strategy G	2.3488×10^9	5.0648×10^5	0.0012

In Table 8, the ICER values are computed as follows:

$$\text{ICER(Strategy F)} = \frac{1.3938 \times 10^6}{1.9659 \times 10^9} = 0.00071$$

$$\text{ICER(Strategy A)} = \frac{2.1457 \times 10^3 - 1.3938 \times 10^6}{2.3234 \times 10^9 - 1.9659 \times 10^9} = -0.00389$$

$$\text{ICER(Strategy E)} = \frac{3.5456 \times 10^4 - 2.1457 \times 10^3}{2.3319 \times 10^9 - 2.3234 \times 10^9} = 0.00392$$

$$\text{ICER(Strategy D)} = \frac{4.9651 \times 10^5 - 3.5456 \times 10^4}{2.3405 \times 10^9 - 2.3319 \times 10^9} = 0.05361$$

$$\text{ICER(Strategy G)} = \frac{5.0648 \times 10^5 - 4.9651 \times 10^5}{2.3488 \times 10^9 - 2.3405 \times 10^9} = 0.0012$$

Comparing strategy F and strategy A, the ICER of strategy A is less than ICER of strategy F. Hence strategy F is more costly and less effective than strategy A. Therefore we omit strategy F and recalculate ICER again for the remaining strategies.

Table 9: The Cost and Infection averted for Control A, E, D and G

Strategy	Infection averted	Total Cost in US Dollar	ICER
Strategy A	2.3234×10^9	2.1457×10^3	9.24×10^{-7}
Strategy E	2.3319×10^9	3.5456×10^4	0.00392
Strategy D	2.3405×10^9	4.9651×10^5	0.05361
Strategy G	2.3488×10^9	5.0648×10^5	0.0012

In Table 9, the ICER values are computed as follows:

$$\text{ICER (Strategy A)} = \frac{2.1457 \times 10^3}{2.3234 \times 10^9} = 9.224 \times 10^{-7}$$

$$\text{ICER (Strategy E)} = \frac{3.5456 \times 10^4 - 2.1457 \times 10^3}{2.3319 \times 10^9 - 2.3234 \times 10^9} = 0.00392$$

$$\text{ICER (Strategy D)} = \frac{44.9651 \times 10^5 - 3.5456 \times 10^4}{2.3405 \times 10^9 - 2.3319 \times 10^9} = 0.05361$$

$$\text{ICER (Strategy G)} = \frac{5.0648 \times 10^5 - 4.9651 \times 10^5}{2.3488 \times 10^9 - 2.3405 \times 10^9} = 0.0012$$

Comparing strategy A and strategy E, the ICER of strategy A is less than ICER of strategy E. Hence strategy E is more costly and less effective than strategy A. Therefore we omit strategy E and recalculate ICER again for the remaining strategies.

Table 10: The Cost and Infection averted for Control A, D and G

Strategy	Infection averted	Total Cost in US Dollar	ICER
Strategy A	2.3234×10^9	2.1457×10^3	9.24×10^{-7}
Strategy D	2.3405×10^9	4.9651×10^5	0.02891
Strategy G	2.3488×10^9	5.0648×10^5	0.0012

In Table 10, the ICER values are computed as follows;

$$\text{ICER (Strategy A)} = \frac{2.2826 \times 10^5 - 3.6979 \times 10^6}{2.3818 \times 10^9 - 2.3488 \times 10^9} = 2.44 \times 10^{-6}$$

$$\text{ICER (Strategy D)} = \frac{4.9651 \times 10^5 - 2.1457 \times 10^3}{2.3405 \times 10^9 - 2.3234 \times 10^9} = 0.02891$$

$$\text{ICER (Strategy G)} = \frac{5.0648 \times 10^5 - 4.9651 \times 10^5}{2.3488 \times 10^9 - 2.3405 \times 10^9} = 0.0012$$

Comparing strategy A and strategy D, the ICER of strategy A is less than ICER of strategy D. Hence strategy D is more costly and less effective than strategy A. Therefore we omit strategy D and recalculate ICER again for the remaining strategies.

Table 11: The Cost and Infection averted for Control A and E

Strategy	Infection averted	Total Cost in US Dollar	ICER
Strategy A	2.3234×10^9	2.1457×10^3	9.24×10^{-7}
Strategy G	2.3488×10^9	5.0648×10^5	0.0199

In Table 11, the ICER value of strategy E is computed as follows;

$$\text{ICER (Strategy G)} = \frac{5.0648 \times 10^5 - 2.1457 \times 10^3}{2.3488 \times 10^9 - 2.3234 \times 10^9} = 0.0199$$

In Table 11, it is noted that strategy A (vaccination only) is less costly and more effective than strategy G (combination of vaccination and environmental hygiene and sanitation) and the rest of other strategies.

3.5 Economic Burden of Newcastle Disease

An economic burden is a total loss in output that the investor can get compared to the expected turn-over (Asante and Asenso-Okyere, 2003; Rist *et al.*, 2015; Ding *et al.*, 2016). In health perspectives, the economic burden is considered as the total medical costs associated with the disease illness of the host as well as some measures of the income which are foregone as a result of disease morbidity and mortality (Sachs and Malaney, 2002; Asante and Asenso-Okyere, 2003; Bloom *et al.*, 2012; Hailu *et al.*, 2017). Depending on the nature of the disease, medical costs includes expenditures on prevention, diagnosis, treatment and care of the disease while

employing the prospective costing approach at micro and macro levels (Singh *et al.*, 2014; Rist *et al.*, 2015). The occurrence of diseases in a particular area normally affects the health of the hosts and may cause loss as well as the re-allocation of resources at household level to cover some of the medical expenses related to the disease.

In this case, the analysis address the economic consequences of the ND due to loss incurred through the re-allocation of resources at microeconomic level. In the analysis we consider both direct and indirect costs of production which are presented by the constraint equation $\mathcal{Q}$ defined by;

$$\mathcal{Q} = f\left(\mathcal{K}, \mathcal{L}, \vartheta, \mathcal{N}\right) \tag{124}$$

where variables in the function $\mathcal{Q}$ are defined as;

$$\mathcal{K} = \text{Capital stock or investment}$$

$$\mathcal{L} = \text{Labor force (Workers)}$$

$$\vartheta = \text{Indirect costs}$$

$$\mathcal{N} = \text{Newcastle disease prevalence}$$

Capital stock includes the costs for buying the feed staffs, buying vaccine, access to vaccine and administering the vaccines, identification (laboratory tests), veterinary experts, transportation and the disposing area as well as the official permit for killing the infected chicken, buying and administering vaccines, and the culling processes. It also includes the costs of buying the cleaning equipments. Indirect costs cover other emerged costs for prevention of other diseases in the flock

3.5.1 Data Collection and Operational Costs

Data collections were basing mainly in monthly death cases and the operational costs for prevention of ND and were collected from ten villages of Bagamoyo and Kibaha in a Pwani Region. The data collected are micro data that involving costs of preventions of ND at the household level. In Bagamoyo District, data were collected from Dunda, Kibinda, Lugoba and Zinga villages while in Kibaha District data were collected from: Gwata, Soga, Janga, Kilangalanga, Ruvu and Mlandizi wards. A total of 357 local chicken farmers were involved in

data collection where 308 which is 86.27% were women and the remaining population sample $(\text{that is}, 13.73\%)$ were men. The average chicken in the flocks per individual chicken grower was 80 chicken which made a total of 28560 chicken in the two areas of the study. Data collection only recorded the population of mature/older chicken, with a population of $3,256$ cocks and $25,304$ hens. Informations collected were basing on the number of eggs obtained per year, disease prevalences and the amount of money that a household spent to prevent the emergence of ND and other diseases in chicken flocks. The costs included the monthly capital for feeding chicken, laborers, and other indirect costs a chicken grower can incur for treatments of other diseases in the flocks.

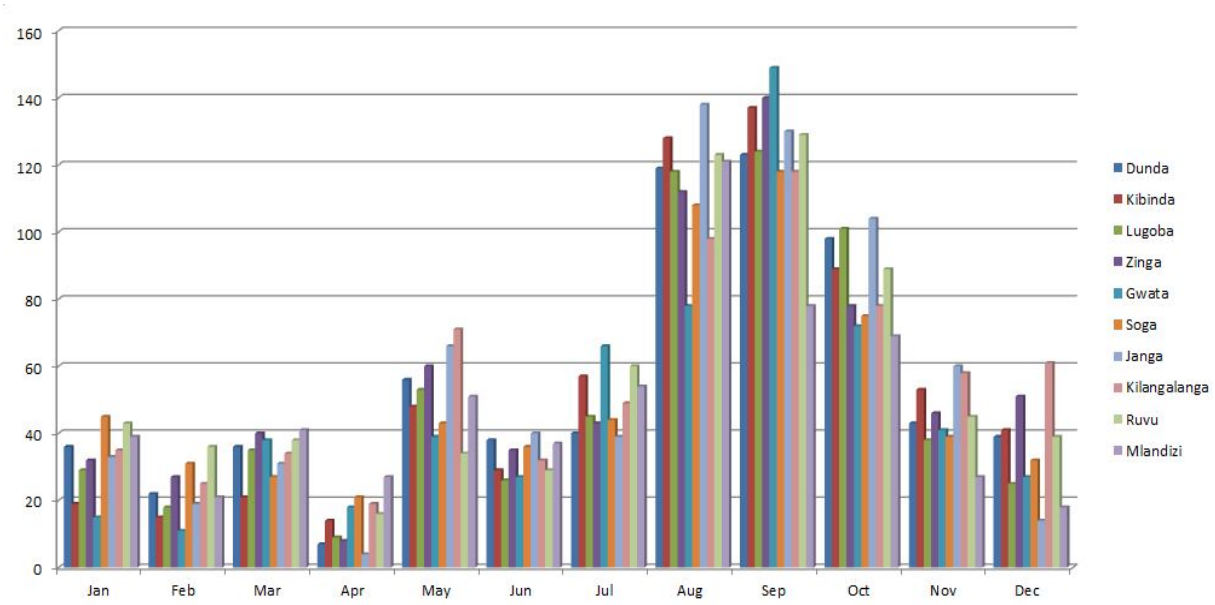


Figure 6: Incidence cases of ND as for data collected from Kibaha and Bagamoyo Districts in Pwani Region in 2017

Table 12: Distribution of ND incidence cases from Kibaha and Bagamoyo in, 2017

Month	Dunda	Kibinda	Lugoba	Zinga	Gwata	Soga	Janga	Kilanga	Ruvu	Mlandizi
January	36	19	29	32	15	45	33	35	43	39
February	22	15	18	27	11	31	19	25	36	21
March	36	21	35	40	38	27	31	34	38	41
April	7	14	9	8	18	21	4	19	16	27
May	56	48	53	60	39	43	66	71	34	51
June	38	29	26	35	27	36	40	32	29	37
July	40	57	45	43	66	44	39	49	60	54
August	119	128	118	112	78	108	138	98	123	121
September	123	137	124	140	149	118	130	118	129	78
October	98	89	101	78	72	75	104	78	89	69
November	43	53	38	46	41	39	60	58	45	27
December	39	41	25	51	27	32	14	61	39	18

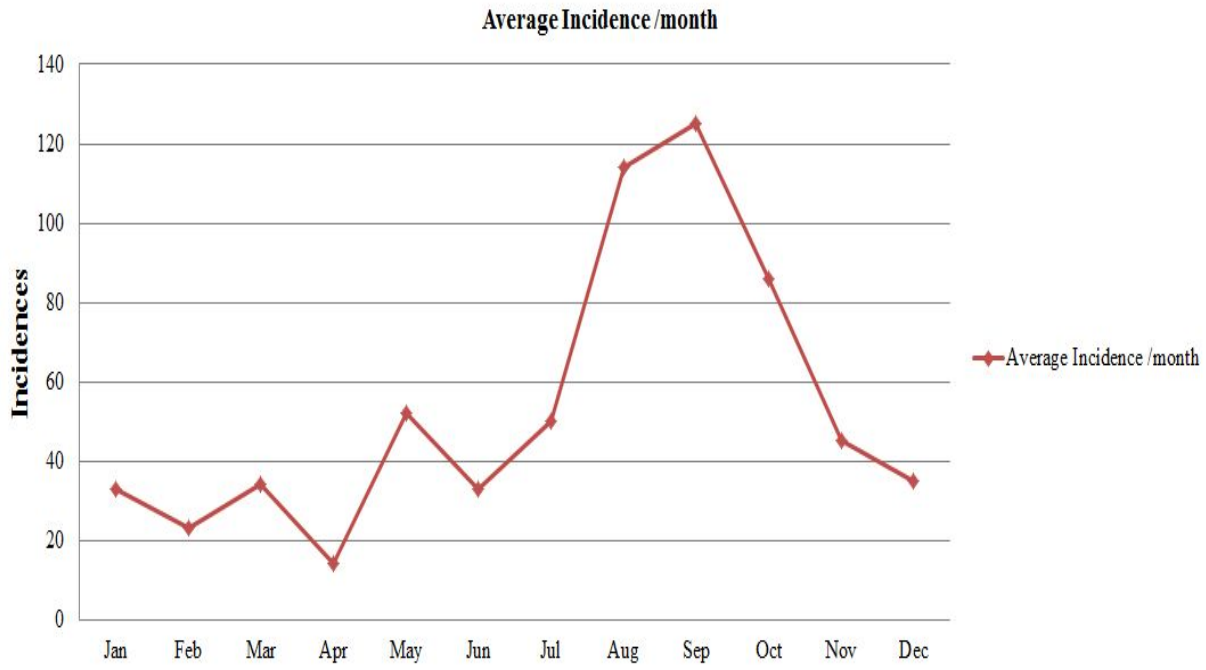


Figure 7: Average monthly distribution of Incidence of ND for Kibaha and Bagamoyo Districts between January and December, 2017

The average Operational costs for the prevention of the ND among the village chicken are estimated and converted from the local currency (T.Shillings) to US Dollar where (1 $=2279.20 T.shillings). The average cost of buying the Newcastle vaccine is 2.46$ applied for 100 chicken per a single dose and it is supposed to be applied up to four times a year, access to vaccine costs an average of 1.711$, administering the vaccine to chicken costs 0.154$ once per chicken and water cans have an average cost of 2.567$. Other estimates includes the costs for buying sprays, blooms and litters which are 2.194$, 0.65$ and 1.053$ respectively. Paying labors is on the average of 14.26$ per month and the average chicken price prior to the outbreak is 5.57$. Extra expenditures per month is 23.692$.

Table 13: Operational Costs (in T.Shillings) as corrected from Dunda, Kibinda, Lugoba, Zinga, Gwata, Soga, Janga, Kilanga, Ruvu and Mlandizi

	Villages									
Items	Dunda	Kibinda	Lugoba	Zinga	Gwata	Soga	Janga	Kilanga	Ruvu	Mlandizi
Vaccine	5,000	7,000	5,500	5,000	6,000	6,000	7,000	6,500	5,000	7,500
Access to vaccine	5,000	5,000	3,000	4,000	2,000	5,000	3,000	3,000	5,000	4,000
Administering vaccine	300	-	-	300	-	-	-	-	500	300
Chicken price	9,000	13,000	12,000	8,000	12,000	13,000	15,000	12,000	15,000	18,000
Spray	5,000	5,000	5,000	5,000	5,000	5,000	5,000	5,000	5,000	5,000
Bloom	2,000	1,000	1,000	2,000	271,000	1,000	2,000	2,000	2,000	1,000
Litters	2,000	1,500	2,000	1,000	2,000	1,500	3,000	4,000	4,000	3,000
Water cans	7,500	6,000	5,000	6,000	5,000	5,000	5,000	8,000	6,000	5,000
Laborers	30,000	15,000	40,000	60,000	25,000	45,000	10,000	20,000	30,000	50,000
Indirect costs	50,000	50,000	50,000	40,000	50,000	50,000	30,000	100,000	70,000	50,000

3.5.2 Burden of the ND at Family Level

At present the study focus on investigating the economic burden of the ND at the household level as the leading poultry disease that cause more deaths of chicken compared to other diseases of poultry. Economic losses due to the ND is then considered as the sum of the following loss factors;

(i) The number of chicken that die (D) or culled due to the outbreak of the ND

(ii) Loss of production due to the decline of the number of eggs laid (LP)

(iii) Loss due to prevention costs (PC)

Therefore, the total economic Loss/ Burden is expressed as:

$$\mathrm{T_{EL}} = \mathrm{MVL} + \mathrm{LP} + \mathrm{PC} \tag{125}$$

where MVL is the Monentary Value Loss due to number of chicken died Loss from mortality This refers to the loss obtained due to the death of the chicken (disease induced and culling) are a result of contracting a ND. It is considered as the product of the total number of incidence cases and the average market price of a chicken. From the Table 12, the total number of incidence cases (I.C) due to ND in Kibaha and Bagamoyo from January to December, 2017 was as follows; Dunda 661 cases, Kilangalanga 651 cases, Kibinda 621 cases, Zinga 672 cases, Gwata 581 cases, Soga 619 cases, Janga 678 cases, Kilangalanga 678 cases, Ruvu 681 cases and Mlandizi had 583 cases which all made a total of 6425 Newcastle disease incidence cases for a duration of one year. One healthy chicken can lay an average number of 60 eggs per year. Now, the Monentary value loss (MVL) due to mortality in all locations from January to December, 2017 is given by;

$$\mathrm{MVL} = \text{The total number of incidence cases} \times \text{Average price of a chicken}$$

$$\mathrm{MVL} = 6425 \times 5.57\$ = 35,787.25\$$$

3.5.3 Loss in Production due to the decline of eggs laid (LP)

Here loss in production is taken as the directly loss in eggs production due to chicken died from ND. The production is expressed in terms of Monentary sum as follows;

$$\mathrm{LP} = (\mathrm{T} - \mathrm{D})\,\mathrm{M}\,\mathrm{Z} \tag{126}$$

where as;

$$\begin{aligned} \mathrm{T} &= \text{Total number of infected chicken} \\ \mathrm{D} &= \text{Chicken died or culled} \\ \mathrm{M} &= \text{Average price of an egg} \\ \mathrm{Z} &= \text{Annual average eggs laid} \end{aligned} \tag{127}$$

$$\mathrm{LP} = (10000 - 6425) \times 0.132\$ \times 60 = 28,214\$ \tag{128}$$

The loss in production can also be considered as the proportional loss in productivity below the expected turn-over from the chicken to be sold before the outbreak of the Newcastle disease. By considering the Monentary value loss (MVL), we need first to calculate the expected turn-over (ET) revenue per year of the chicken to be sold. The expected turn- over (ET) is mathematically done by the following formula:

$$\mathrm{ET} = \text{Number of chicken} \times \text{Average price of chicken} \times \text{duration} \tag{129}$$

If all chicken can be sold once a year, then the duration is considered as one year. The Expected turn-over for an average price of chicken sold at $5.57\$ - 8.81\$$ become:

$$\text{Expected Turn-over} = 28560 \times 5.57\$ \times 1 = 159,079.2\$ \tag{130}$$

for an average price of $5.57\$$ and Now, the proportional loss in production (LP) become;

$$\text{Proportional Loss in Production} = \frac{\mathrm{MVL}}{\mathrm{EP}} \times 100\% \tag{131}$$

$$\text{Proportional Loss in Production} = \frac{35{,}787.25}{159{,}079.2} \times 100\% = 22.5\% \tag{132}$$

3.5.4 Loss due to Prevention Costs (PC)

The loss due to prevention costs is considered as the product of number of chicken died or culled (D) out from the population and the average prevention cost (G_i) of of the chicken before the outbreak of the Newcastle disease. Mathematically expressed as:

$$\mathrm{PC} = \mathrm{D}\sum_{i=1}^{n} \mathrm{U}_i \mathrm{G}_i; i = 1, 2, ...5 \tag{133}$$

where as:

$$\begin{aligned}
&\mathrm{U}_i = \text{The unit cost multiplier applied to each type of goods and services consumed}\\
&\mathrm{G}_1 = \text{Costs of buying the vaccine}\\
&\mathrm{G}_2 = \text{Costs for accessing vaccine}\\
&\mathrm{G}_3 = \text{Costs for administering the vaccine}\\
&\mathrm{G}_4 = \text{Costs for controlling other diseases in the flock}\\
&\mathrm{G}_5 = \text{Paying laborers}
\end{aligned} \tag{134}$$

Here it is assumed that vaccine is applied three times a year (by considering the age of chicken).

$$\mathrm{PC} = \mathrm{D}\left(\mathrm{U}_i\left(\mathrm{G}_1 + \mathrm{G}_2 + \mathrm{G}_3 + \mathrm{G}_4 + \mathrm{G}_5\right)\right) \tag{135}$$

$$\mathrm{PC} = 6425 \times (2.46{+}0.154{+}1.711{+}3.895) = 108,389.75\$ \tag{136}$$

From the equation 125, the total economic loss of Newcastle disease at family level become;

$$\mathrm{T}_{\mathrm{EL}} = \frac{35,787.25\$ + 28,214\$ + 108,389.75\$}{357 \text{ chicken growers}} = 482.89\$/\text{ chicken grower} \tag{137}$$

For the average chicken price of $8.81\$$ the total economic loss become $541.20\$$. This shows that the occurrence of the ND leads to an average of $482.89 - 541.20\$$ economic loss at family level. This amount is huge compared to social-economical status of the family. Therefore, more efforts should be inverted to assure the village chicken population are free from the Newcastle disease.

CHAPTER FOUR
RESULTS AND DISCUSSION

This chapter presents the numerical results and discussion of the study. The discussions ranges from the simulation of the basic ND Model followed with the simulation of the model with controls. Parameters values of the model were estimated depending on the ND scenarios, and some values were found from the literature. Numerical simulations and graphical representations of the analytical results are done using the MATLAB software.

Table 14: Parameter values of the Newcastle disease Model

Parameter	Parameter value	Source
a	$0.01day^{-1}$	Assumed
b	$0.21day^{-1}$	(Alexander *et al.*, 2004; Dortmans *et al.*, 2011)
α_c	$0.1667day^{-1}$	Assumed
α_b	$0.02virus^{-1}chiken^{-1}day^{-1}$	Assumed
ϕ	$0.02day^{-1}$	Assumed
ψ	$0.083 - 0.1day^{-1}$	Assumed
μ	$2.74 - 5.48 \times 10^{-4}day^{-1}$	(McDermott *et al.*, 2001; Lucchetti *et al.*, 2009)
φ	$0.02day^{-1}$	Assumed
ρ	0.998	Assumed
d	$0.001day^{-1}$	Estimate
γ	$0.067 - 0.2day^{-1}$	(Perry *et al.*, 1999; Sharif *et al.*, 2014)
δ_b	$0.025day^{-1}$	(Daut *et al.*, 2016)
δ_c	$1.99 \times 10^{-2}day^{-1}$	(Hugo *et al.*, 2017)
μ_v	$2.19 \times 10^{-2}day^{-1}$	(Chuma *et al.*, 2018)
κ	1.0×10^4 virus $/m^3$	(Chuma *et al.*, 2018)

4.1 Numerical Simulations of the Basic Model

The Fig.8 represents the dynamics of ND in the village chicken, wild birds populations and NDV in the environment in the endemic situation in the absence of any control.

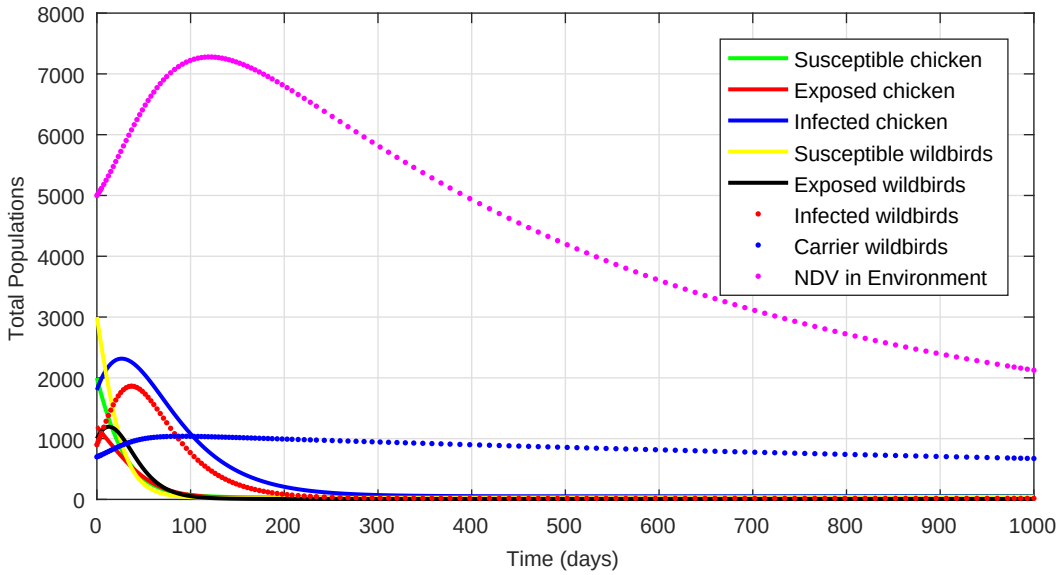


Figure 8: The Transmission Dynamics of ND in village Chicken Population, wild Birds Population and the NDV in the Environment during the outbreak in the absence of any control with initial state conditions

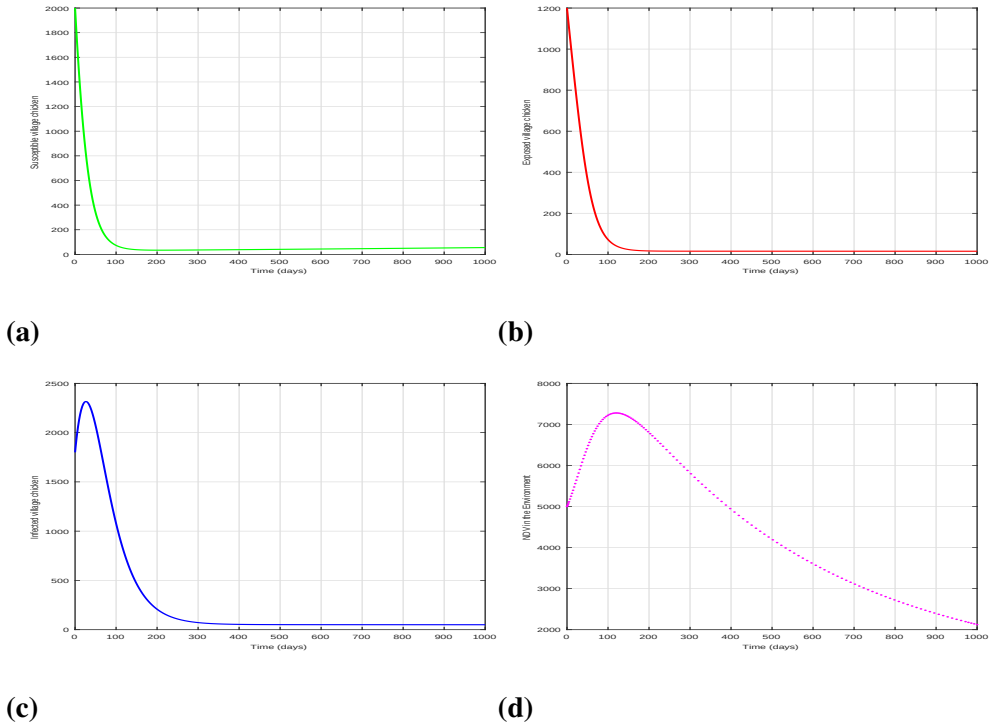
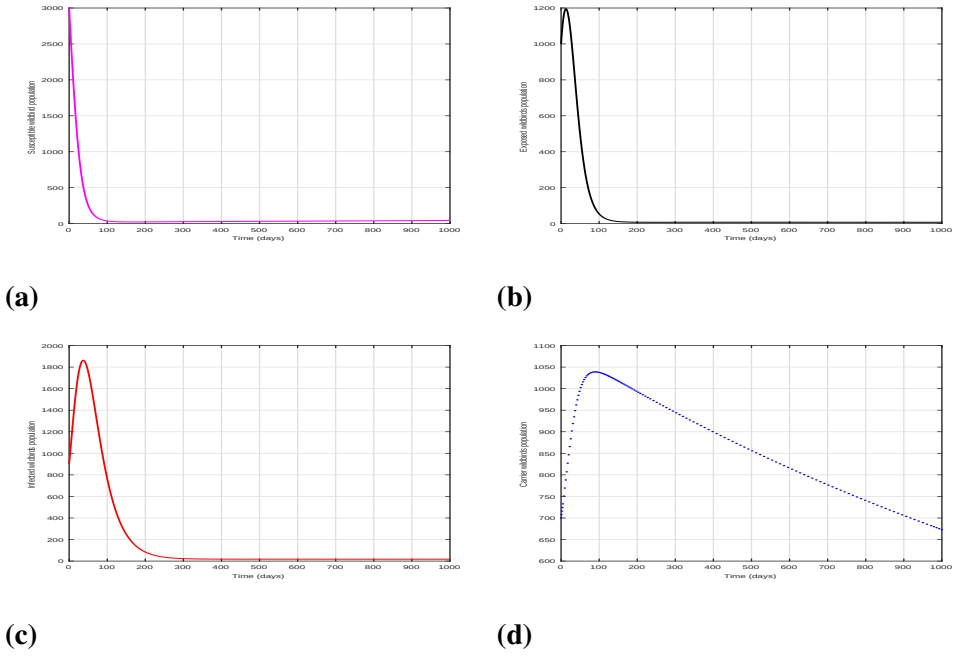


Figure 9: The Dynamics of ND in the wild birds. (a) Shows Susceptible Population, (b) Shows the Exposed Population, (c) Shows the Infected Population and (d) Shows the carrier population.

The Fig. $9\,(a-d)$ and Fig. $10\,(a-d)$ show simulations of the dynamics of ND in the village chicken and wild birds population in the endemic situation respectively. The susceptible population of the village chicken is exponentially decreasing due to both disease induced death and the natural mortality rate of the chicken. The population does not reach extinction due to a constant recruitment rate of chicken by birth. Fig. $9\,(b)$ represents the exposed population of the village chicken. For the first few days of the simulation, the exposed population of the village chicken is increasing due to ND infection, then the population decreases exponentially. Normally infections takes place between day one to day six before the the observable clinical signs of the disease on the individual chicken. Therefore as time goes on some chicken die naturally whilst others progress to the serious sickness class as shown in Fig. $9\,(c)$. The population of the infected chicken increase for a short period of time due to new chicken which progress from the exposed to the infected class. The population of this class suddenly decreases exponentially due to both disease induced and natural deaths. Fig. $9\,(d)$ represents the concentration of the NDV in the environment. The increase of the virus concentration in this compartment is a result of the high shedding rate of virus from both infected chicken and birds as well as carrier

birds. After reaching its maximum concentration, depending on the environmental conditions, the concentration of active virus decreases attributed by the decrease in the number of infected poultry. Fig. $9\,(a)$ represents the susceptible population of the village chicken which decreases due to the high infectious rate of the NDV. Fig. $10\,(b)$ represents the exposed population of the wild. The incubation of virus in this class takes few days before the poultry shows the signs of ND infection.



(a) **(b)**

(c) **(d)**

Figure 10: The Transmission Dynamics of ND in the Wild Birds. (a) Shows Susceptible Population, (b) Shows the Exposed Population , (c) Shows the Infected Population and (d) Shows the Carrier Population.

Then the population start to decrease exponentially due the natural death and other forces of infections that cause a proportion of that population to become infectious to the disease as in Fig. $10\,(c)$. However, the infected population of wild birds decreases exponentially by due to the disease induced death (δ_c) and the natural death (μ). Since the wild birds do not recover from the ND, then very few birds can survive (see the Fig.$10\,(c)$). Fig. $10\,(d)$ shows the population of the carrier wild birds which consists of the birds which has immunity against Newcastle disease.

4.1.1 Variations of Population Variables Over Time.

Different initial values are used to illustrate numerical stabilities of both disease free DFEP and the EEP of the model system $(22a)$ to (24). With different starting points, the initial values converges to a common point which assures the existence and stability of both disease free and endemic equilibrium points. The Fig. $11-18$ and Fig. $19-26$ shows the stability of the disease free and endemic equilibrium points respectively. In the Fig. $11-18$, different initial values of the model variables converges to constant value along the time axis which assures us that at this point $\mathcal{R}_0 < 1$. Fig. $19-26$ shows the stability of endemic equilibrium point which converges above zero which reveals that the point is at $\mathcal{R}_0 > 1$. Hence, all equilibria points are asymptotically stable.

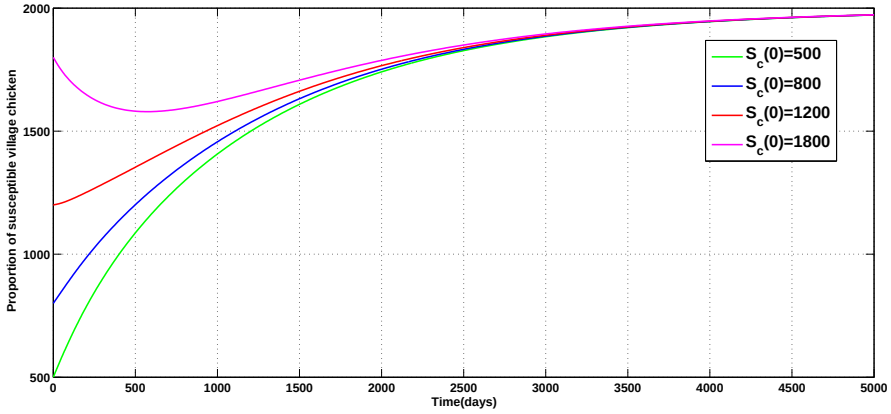


Figure 11: Variations of Susceptible Population of Village Chicken at Disease Free Equilibrium Point

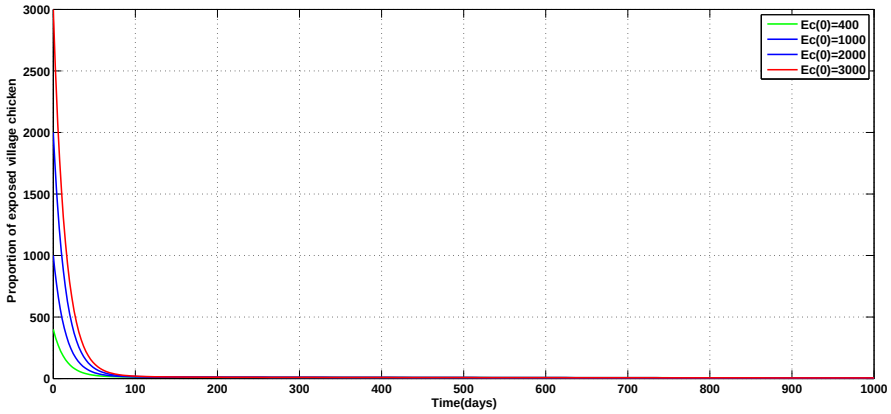


Figure 12: Variations of Exposed Village Chicken at Disease Free Equilibrium point

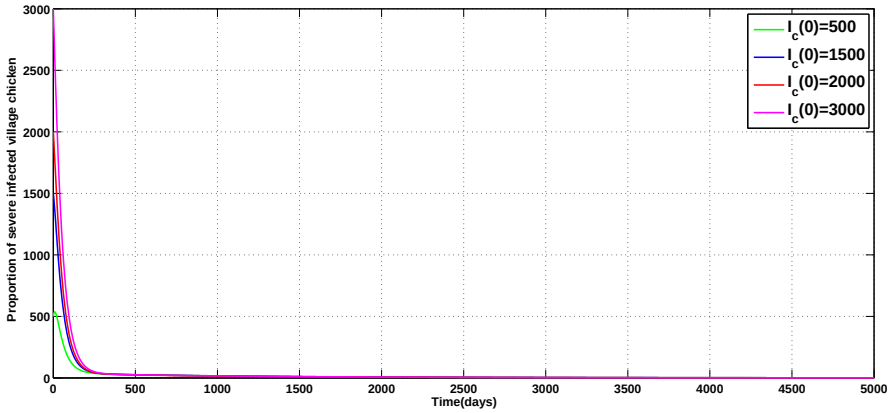


Figure 13: Variations of Severe Infected Village Chicken at Disease Free Equilibrium Point

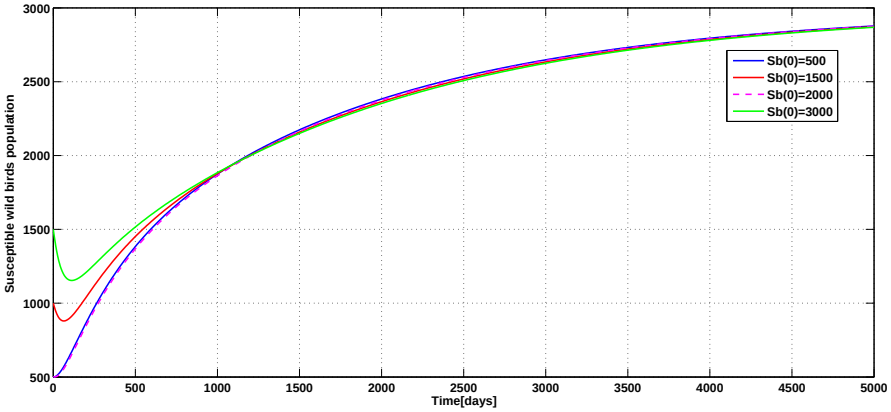


Figure 14: Variations of the Susceptible Wild Birds at Disease Free Equilibrium Point

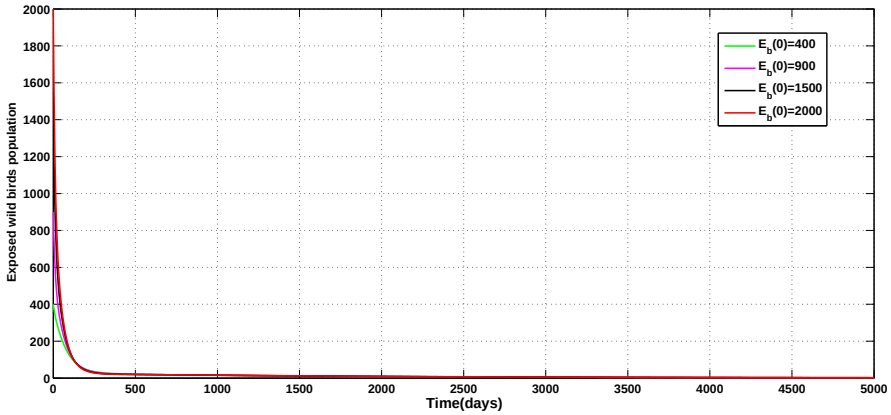


Figure 15: Variations of Exposed Wild Birds at Disease Free Equilibrium Point

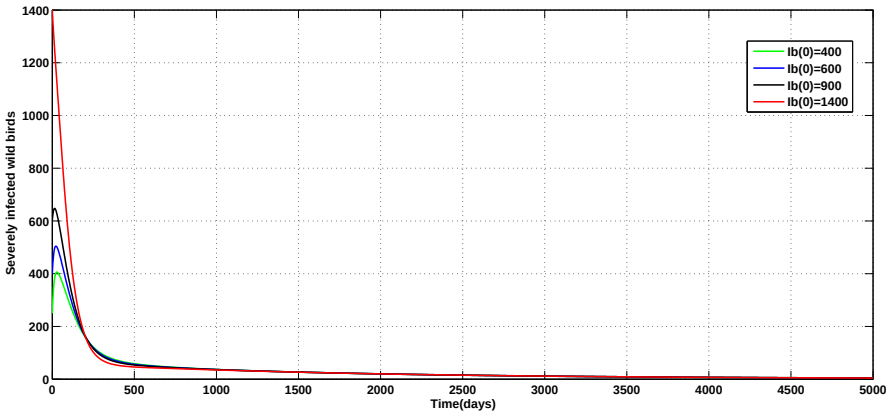


Figure 16: Variations of Severe Infected Wild Birds at Disease Free Equilibrium Point

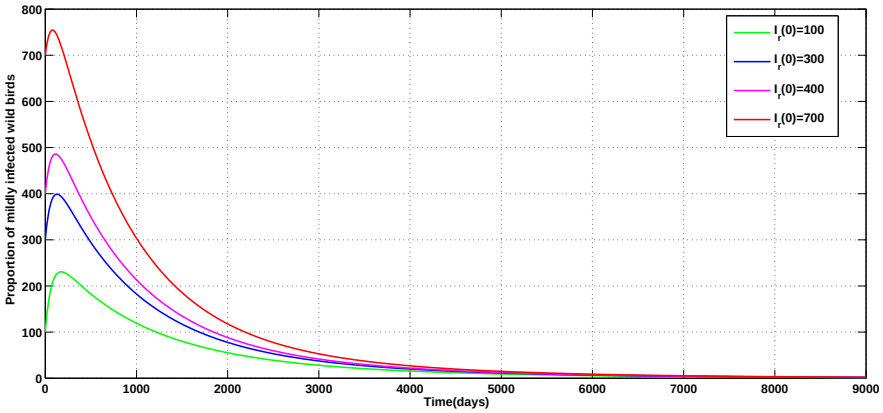


Figure 17: Variations of mild Infected Wild Birds at Disease Free Equilibrium Point

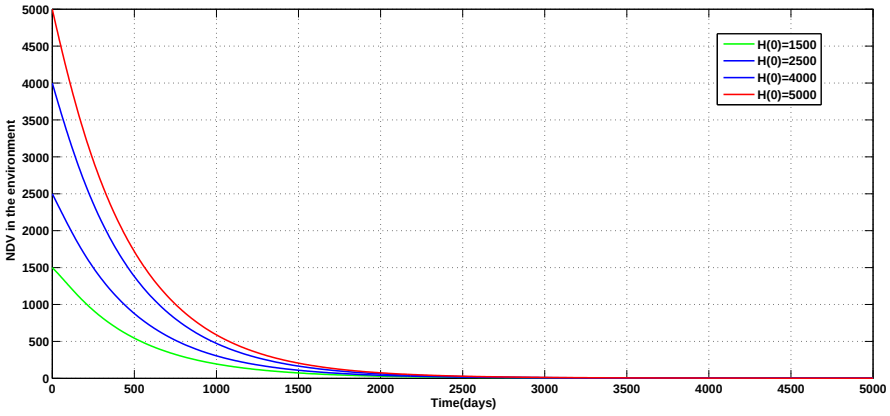


Figure 18: Variations of NDV in the environment at Disease Free Equilibrium Point

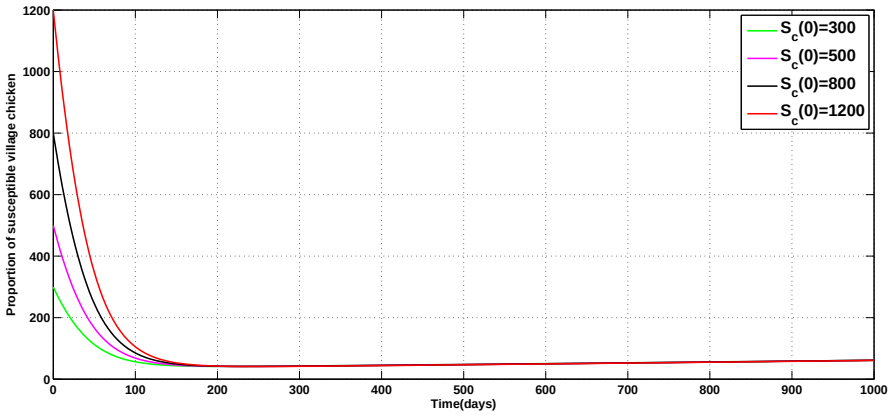


Figure 19: Variations of susceptible village chicken at EEP

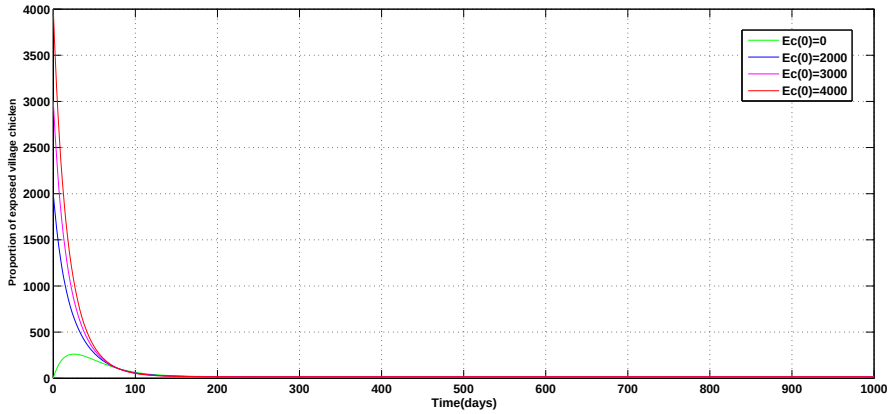


Figure 20: Variations of exposed village chicken at EEP

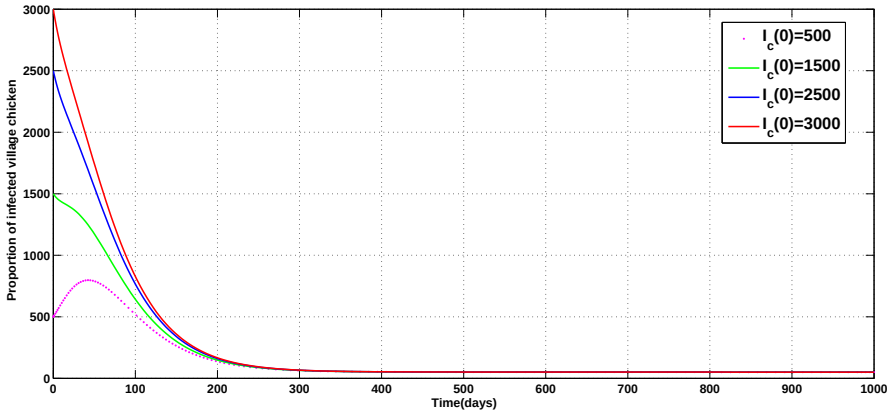


Figure 21: Variations of severe infected village chicken at EEP

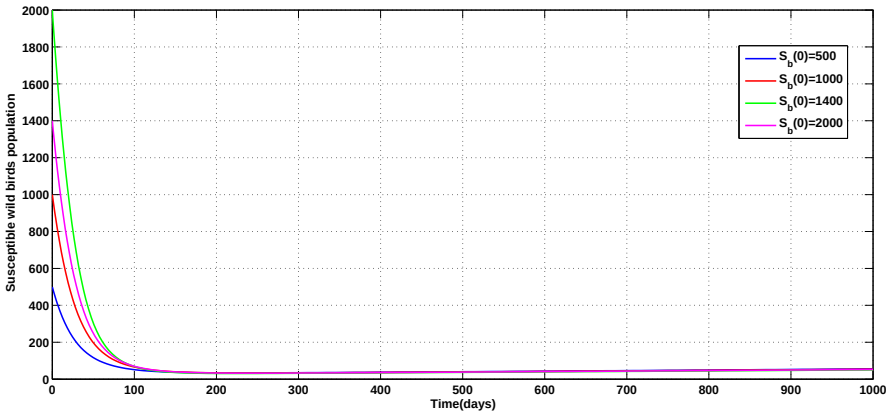


Figure 22: Variations of susceptible wild birds at EEP

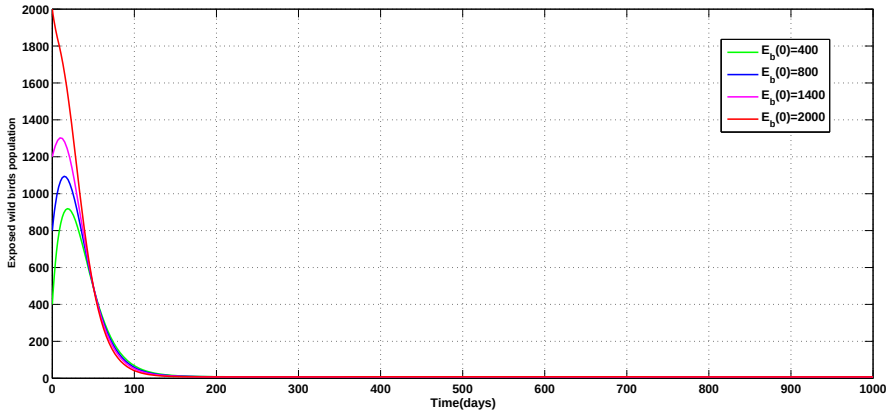


Figure 23: Variations of exposed wild birds at EEP

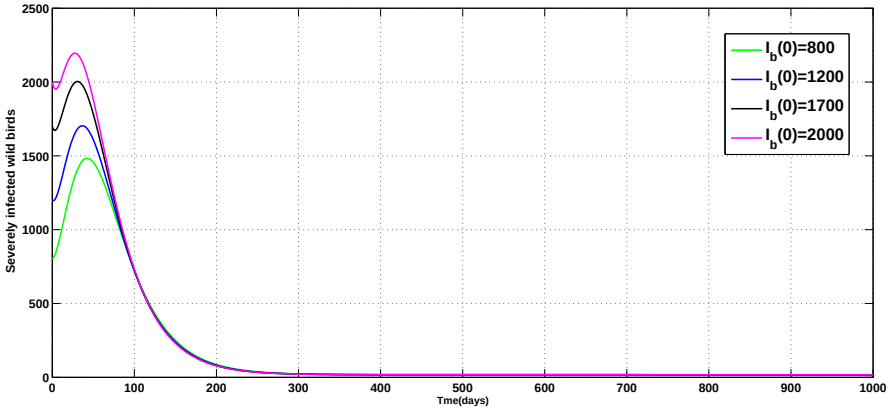


Figure 24: Variations of severe infected wild birds at EEP

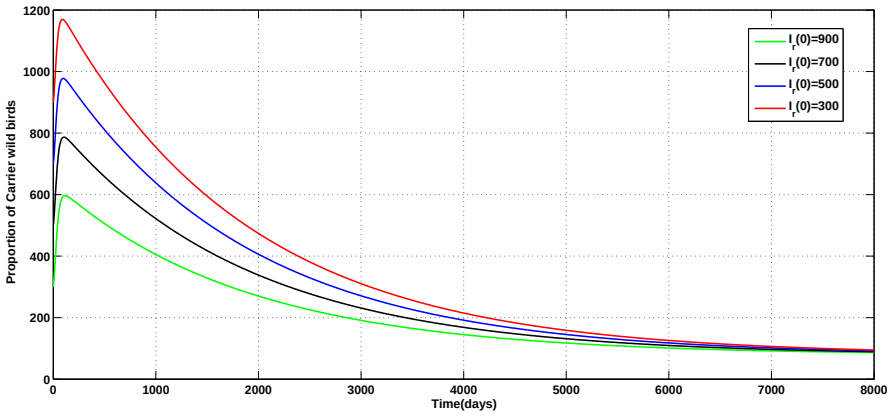


Figure 25: Variations of mild infected wild birds at EEP

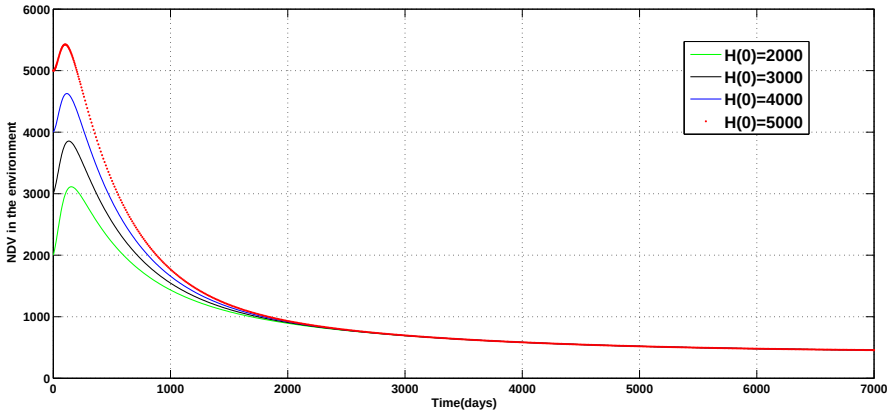


Figure 26: Variations of NDV in the environment at EEP

4.1.2 Sensitivity Analysis of the Basic Model

The sensitivity analysis of $\mathcal{R}_0$ helps to understand the behavior of parameters on the model output as well as their influence in the spread of the disease in the population (Ostermann, 2005). In order to get the sensitivity value of a parameter in the model system in equation $(22a)$ to (24), we apply the normalized forward sensitivity analysis index as discussed in Chitnis *et al.* (2008). In this method, a function $\mathcal{R}_0$ should be differentiable at a parameter q, where q being any parameter in $\mathcal{R}_0$. A normalized forward index of a variable q is defined by

$$\Upsilon_q^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial q} \times \frac{q}{\mathcal{R}_0}$$

Parameter values to be used are taken from the field study, the related literatures and others are assumed. The population of village chicken in Bagamoyo and Kibaha districts of a Coastal region (Tanzania) is estimated to be four hundred thirty one thousand seven hundred and fifty $(431, 750)$ with 90% death cases on absence of any control during the outbreak of Newcastle disease. This gives an estimate of the ND induced death $\delta_c = 7.5 \times 10^{-2} day^{-1}$.

Wild birds are assumed to be five hundred thousand (5.0×10^5) and its disease induced death is assumed to be $\delta_b = 2.5 \times 10^{-3} day^{-1}$. We estimate an average lifespan of village chicken to be five to ten years which yields a mortality rate $\mu = 1.678 \times 10^{-3} day^{-1}$ (Lucchetti *et al.*, 2009). The saturation constant (κ) of the NDV in the soil is not known hence is assumed in cubic meter as $1 \times 10^4\ cells m^{-3}$ which that yields 50% chance of infections. The assumed parameters have its base in the epidemiological phenomenon in the transmission dynamics of Newcastle disease among village chicken with wild birds and environment as primary reservoir of the NDV.

Using the basic reproduction number in 83 and the parameter values given in Table 14, computation gives

$$\mathcal{Y}_{\phi}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \phi} \times \frac{\phi}{\mathcal{R}_0} = +0.00647; \quad \mathcal{Y}_{\mu}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \mu} \times \frac{\mu}{\mathcal{R}_0} = -0.07953$$
$$\mathcal{Y}_{\gamma}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \gamma} \times \frac{\gamma}{\mathcal{R}_0} = +0.00811 : \quad \mathcal{Y}_{\psi}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\psi} \times \frac{\psi}{\mathcal{R}_0} = +0.38706$$

Now, through the same procedures we obtain the Sensitivity indices for parameter values of the model system in Table $22a$ to 24 summarized in the bar chart diagram here below;

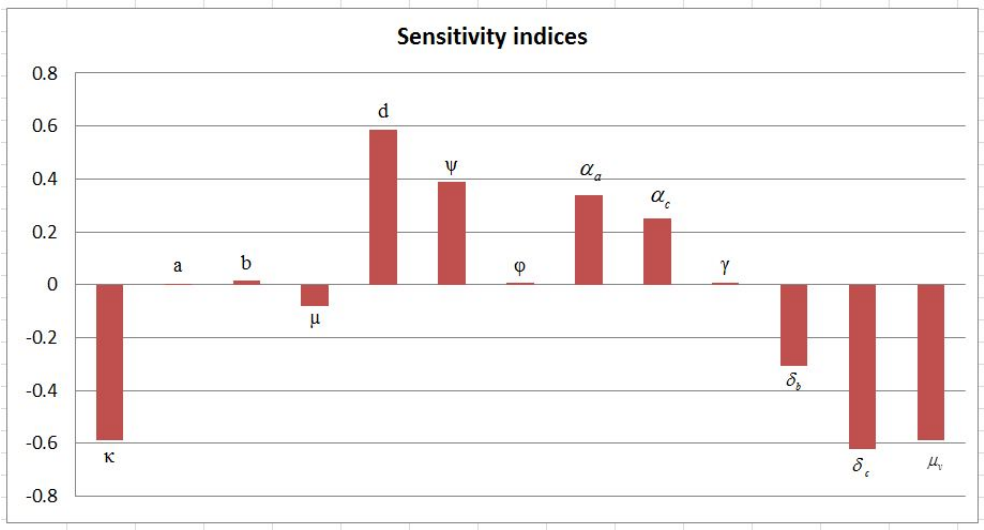


Figure 27: Sensitivity analysis of the basic Reproduction Number

In these results, the positive indices show that the basic reproductive number increases as the value of the parameter a, b, d, α_b, α_c, ψ, φ and γ increases (see Fig. 27). All of these parameters have a positive influence on the basic reproductive number, $\mathcal{R}_0$. The transmission coefficient of NDV between from the environment and the hosts (d) is the most positive sensitive parameter followed by transmission coefficient between the severely infected village chicken and their susceptible population (ψ). The least positive sensitive index values is the transmission coefficient between the mild infected population of wild birds and the susceptible population of wild birds (a). However the negative index values implies the inverse relationship between the basic reproduction number and the parameter value. The parameter δ_b, δ_c, μ, κ and μ_v have negative sensitivity index values. The ND induced death rate (δ_c) in village chicken is the most negative sensitive parameter in the model. This tells us that the more deaths of the village chicken during the outbreak of the disease reduce interactions and hence lower the basic reproduction number of the NDV model. The least negative sensitive index values is the natural death of the village chicken population (see Fig. 27).

4.1.3 Numerical Solution for the Optimal Control Problem

In this part we analyze numerically various combinations of the control efforts on the spread of ND among the village chicken population. The single control efforts and their combinations makes a total number of seven control efforts namely strategy A; strategy B; strategy C; strategy D as the combination of strategy A and B; strategy E as the combination of strategy A and C; strategy F as the combination of strategy B and C; and strategy G as the combination of strategy A, B and C as discussed in section 3.5.

We solve the optimal control problem consisting of the state variables, the adjoint or co-state system and the characterization system in equation (113), (119) and (122) by using Forward-Backward Sweep method which is the Runge-Kutta of order four as explained in (Lenhart and Workman, 2007; Kar and Ghosh, 2012; Sagamiko *et al.*, 2015; Kahuru *et al.*, 2017*b*). The state system is solved using a MATLAB software and the forward in time Runge-Kutta method of order four with initial conditions $N_c(0) = 2000$; $N_b(0) = 3000$; $S_c(0) = 2000$; $V(0) = 200$; $E_c(0) = 1200$; $I_c(0) = 1800$; $S_b(0) = 3000$; $E_b(0) = 1000$; $I_b(0) = 900$; $I_r(0) = 700$;

$E(0) = 5000$. The adjoint system is solved by the using the backward in time fourth order Runge-Kutta method with terminal conditions $\lambda_1(t_f) = \lambda_2(t_f) = ... = \lambda_9(t_f) = 0$ where $t_f = 365days$. The controls are bounded in the interval $0 \leq u_i \leq 1$ for $(i = 1,\ 2,\ 3)$ and the weights in the objective functional associated with disease status are chosen to be $A_1 = 200$, $A_2 = 300$ $A_3 = 500$ while those associated with each control are assumed to be $L_1 = 30$; $L_2 = 20$; $L_3 = 10$ as chosen from Hugo *et al.* (2017). These weights are theoretically chosen just to reveal the control strategies proposed in this study. Other parameter values used in the simulations are given in Table 14.

4.1.4 Numerical Algorithm

The numerical approximation of the optimal solution of the optimal state problem is performed using the Forward-Backward sweep method (FBSM) which is the Runge-Kutta method of order four (Kar and Ghosh, 2012; Mwanga *et al.*, 2014). The Forward-Backward Sweep method has the following successive steps;

(i) The total time is divided into r sub-intervals irrespectively to the state $X = (X_1, X_2, ..., X_{r+1})$ and the co-state variables as $\lambda = (\lambda_1, \lambda_2, ..., \lambda_{r+1})$

(ii) The controls are assumed to take zero intervals for starting an iteration such that $u = [0, 0, .., 0]$.

(iii) With the initial condition $X\,(0) = X_0$, the state solutions in the ODE with the controls are solved by using the forward in time Runge- Kutta method of order four.

(iv) With the transvesality condition $\lambda_{N+1} = \lambda\,(t_f)$ where t_f is a final time, the values for u and values for X, the co-state variables from co-state differential equation are solved by the backward in time Runge- Kutta method of order four.

(v) The Update of the control is done by entering the new X and λ through the rule

$$u^* = min\left\{u_{\max}, \max\left(u_{\text{sig}}, u_{\min}\right)\right\} \tag{138}$$

where the boundedness of controls is defined as;

$$u^* = \begin{cases} u_{\min} & \text{if} \quad \frac{\partial \mathcal{H}}{\partial u} < 0 \\ u_{\min} < u_{\text{sig}} < u_{\max} & \text{if} \quad \frac{\partial \mathcal{H}}{\partial u} = 0 \\ u_{\max} & \text{if} \quad \frac{\partial \mathcal{H}}{\partial u} > 0 \end{cases} \tag{139}$$

(vi) If last preceding iterations are negligible close, then the last iteration is the complete solution otherwise return to step (iii) above.

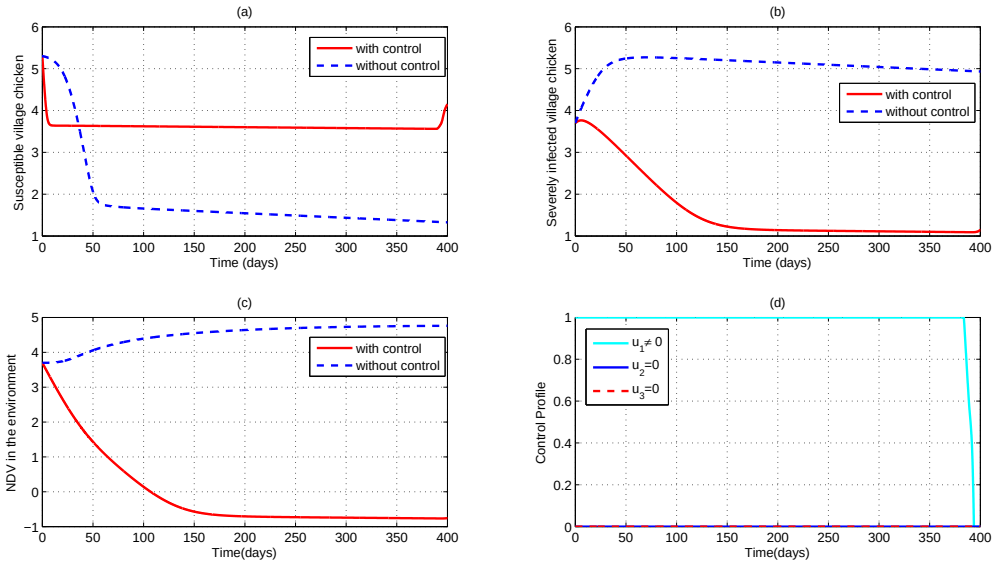


Figure 28: The Graph shows the effects of Vaccination in the Chicken Population

In this strategy, only vaccination (u_1) is used to optimize the objective functional $\mathcal{J}$ in (7) while u_2 and u_3 are set to zero. The control strategy have shown a significant difference in the dynamics of the susceptible chicken, infected chicken and the concentration of the NDV in the environment when compared with the case without any control measure (see Fig. $28\,(a)-(d)$). Using this control strategy, the number of severely infected chicken in Fig. $28\,(b)$ and the concentration of NDV in the environment (Fig. $28\,(c)$) have been significantly reduced. In Fig. $28\,(d)$ the control (u_1) is maintained at its maximum value for about 396 days and thereafter decrease to zero. This mean that using this control strategy the vaccination needs to be applied at 100% effort almost throughout the control period.

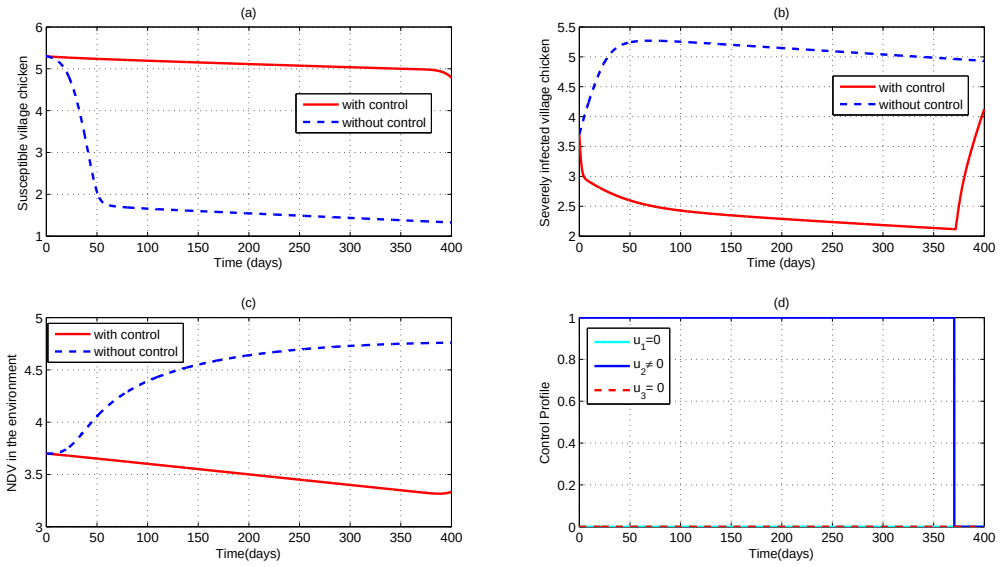


Figure 29: The Graph shows the effects of Culling in the Chicken Population

Here, the culling control (u_2) is used to optimize the objective functional $\mathcal{J}$ in (7) while u_1 and u_3 are set to zero. The strategy have shown a significant increase in the number of susceptible chicken (see Fig. $29\,(a)$) and a significant reduction in the number of infected chicken and the concentration of the NDV in the environment (Fig. $29\,(b)$ and $29\,(c)$) when compared with the case with no control measure. In the Fig. $29\,(d)$ the control u_2 is maintained at its upper bound for about 375 days and thereafter decreases sharply to zero. This shows that the control strategy can be used to reduce the rate of ND in the population but also needs to be applied at 100% effort almost throughout the control period which may be unachievable goal.

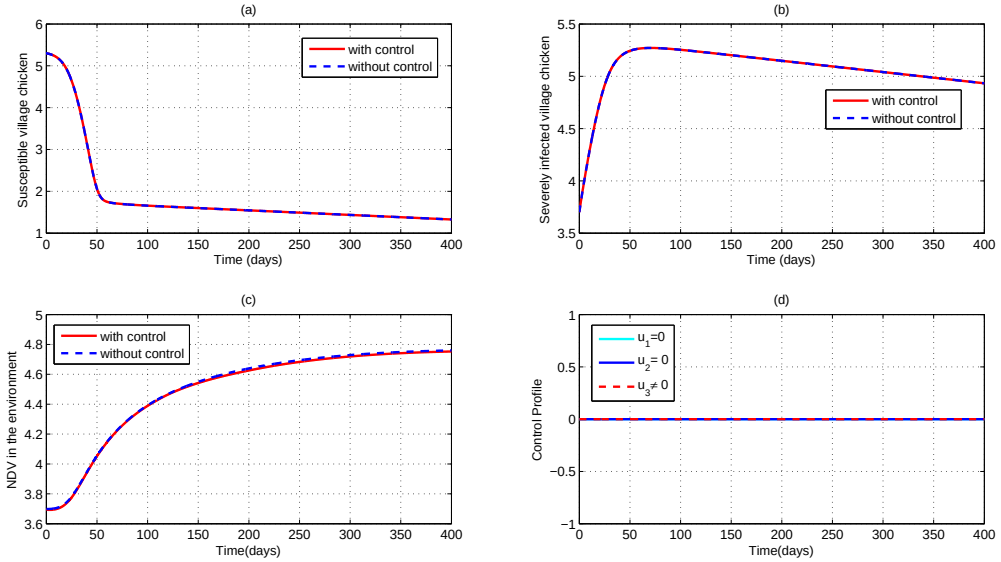


Figure 30: The Graph shows the effects of Environmental Hygiene and Sanitation in the Chicken Population

In this strategy, the environmental hygiene and sanitation (u_3) alone is used to optimize the objective functional $\mathcal{J}$ in (7) while u_1 and u_2 are set to zero. The results show that there is no any significant difference for this control and the case without any control measure in either of the populations as shown in Fig. $30(a\text{-}c)$. This results reveal that when the environmental hygiene and sanitation is applied singly, it is not a suitable control strategy for the control of Newcastle disease.

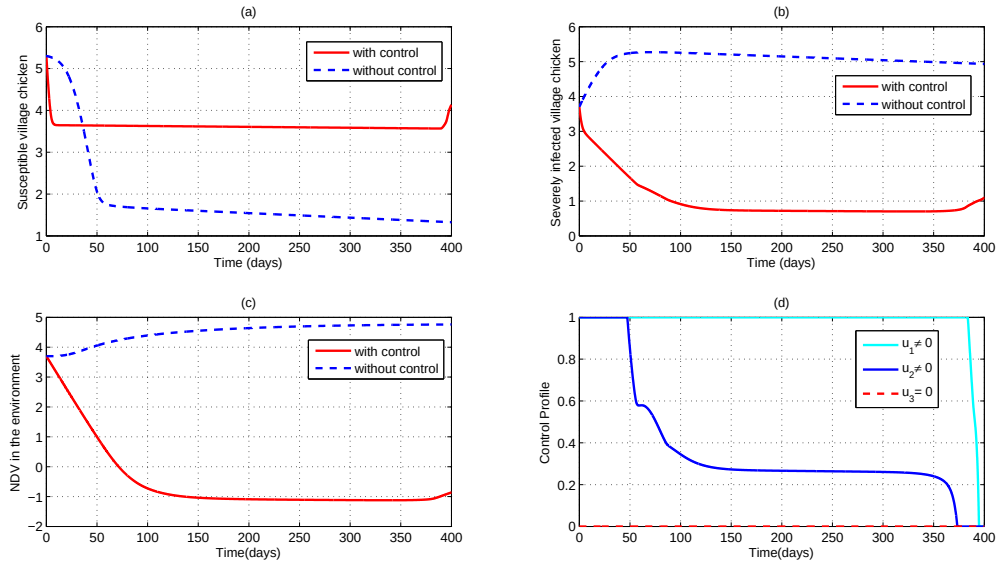


Figure 31: The Graph shows the effects of Vaccination and Culling in the Chicken Population

In this strategy, the vaccination (u_1) and culling (u_2) controls are applied together to optimize the objective functional $\mathcal{J}$ in (7) while u_3 is set to zero. We observed in Fig. $31(a\text{-}c)$ that due to the combination of these control strategies, the population of the susceptible chicken increases since the population is free from the disease as in Fig. $31(a)$, while in Fig. $31(b)$ the number of the infected chicken decrease. A Similar decrease is seen in the concentration of NDV in the environment as in Fig. $31(c)$. Its control profile in Fig. $31(d)$ shows that all controls works effectively when applied together and it is shown that vaccination (u_1) works for 396 days much longer and maintain its upper bound throughout a year than the culling control (u_2) which works effectively for the first 49 days of a year before start to decline gradually to its lower bound at 375 days. The result here shows that the vaccination control protects the chicken population for 100% and by culling out the infected chicken from the population it takes 49 days for the virus to be eliminated from the population.

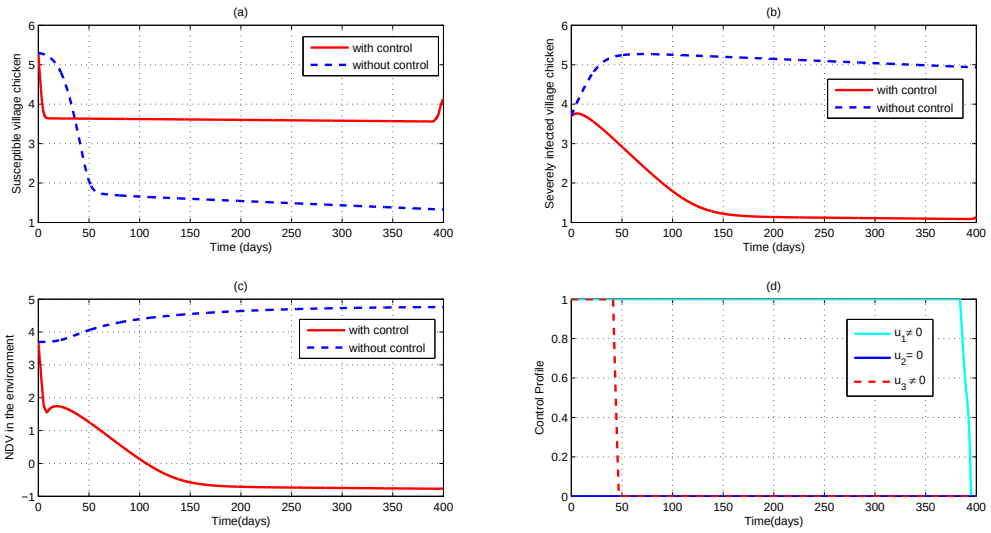


Figure 32: The Graph shows the effects of Vaccination and Environmental hygiene and sanitation in the chicken population

In this strategy, the vaccination (u_1) and environmental hygiene and sanitation (u_3) controls are applied together to optimize the objective functional $\mathcal{J}$ in (7) while u_2 is set to zero. The controls have shown a significant difference in the susceptible chicken, the infected chicken and the concentration of the NDV in the environment. The susceptible chicken in Fig. $32\,(a)$ have

increased since the population is well controlled. However, there is a decline in the infected population of chicken and the concentration of NDV from the environment as seen in the Fig. $32\,(b)$ and $32\,(c)$ respectively. The control profile in Fig. $32\,(d)$ shows that, the vaccination (u_1) remains at its upper bound throughout the year while the environmental hygiene and sanitation control (u_3) is maintained at its upper bound for the first 41 days and thereafter it steadly decline to its lower bound. From these results we can conclude that to control Newcastle disease it is important to emphasize on the provision of the vaccines than the sanitation and hygiene of the environment.

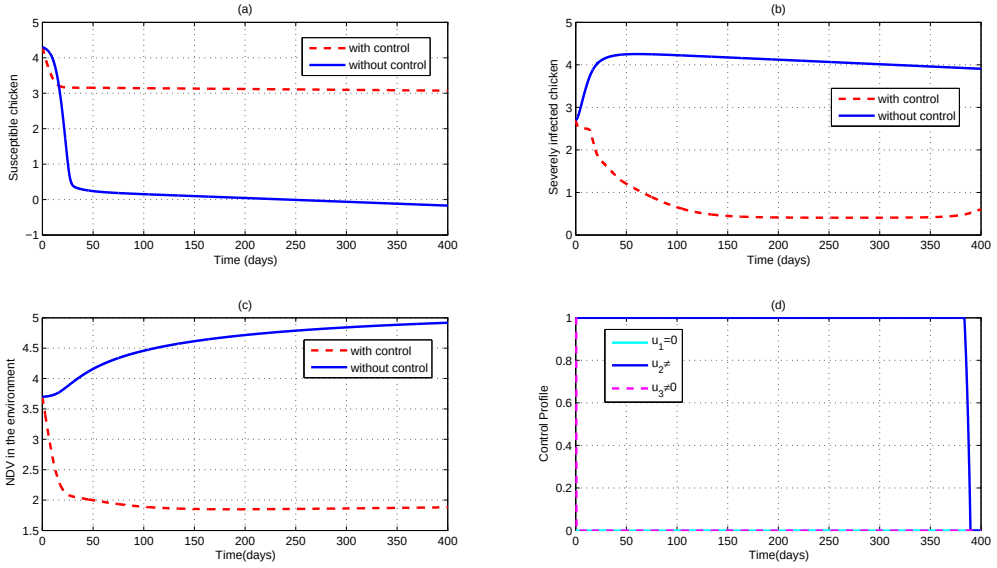


Figure 33: The Graph shows the effects of Culling and Environmental Hygiene and Sanitation in the Chicken Population

In this strategy, the culling (u_2) and Environmental hygiene and sanitation (u_3) controls are used together to optimize the objective functional $\mathcal{J}$ in (7) while u_1 is set to zero. In Fig. $33(a)$ we observe that, the number of susceptible chicken increases while in the Fig. $33(b)$ and $33(c)$ there is significant decrease of both infected chicken and the NDV in the surroundings respectively. Fig. $33(d)$ shows the control profile for u_3 is not stable as it drop to zero from the beginning of the control which make its practical implementation to be rather challenging.

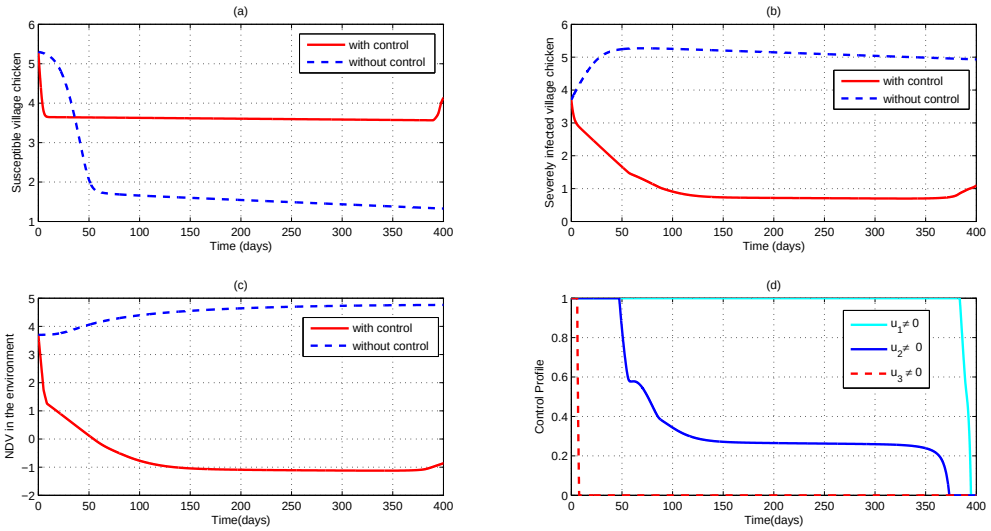


Figure 34: The Graph shows the effects of Vaccination, Culling and Environmental hygiene and sanitation

In this strategy, all controls (u_1), (u_2) and (u_3) are used together to optimize the objective functional $\mathcal{J}$ in (7). Under this control strategy, the combination shows a significant difference in comparison before and after the control. In the Fig. $34(a)$ it is shown that the susceptible population of chicken is increasing while the infected population of chicken and the concentration of NDV decline as in Fig. $34(b)$ and Fig. $34(c)$ respectively. In Fig. $34(d)$, the vaccination control (u_1) is maintained at its maximum value for the whole year while the culling control (u_2) maintains its maximum value for 49 days and then starts to decline slowly for 373 days and further decline to zero. The environmental and sanitation strategy (u_3) have very little contributions in this combination for controlling of the Newcastle disease as it reduces to zero after only 5 days. This strategy managed to reduce the rate of transmission of the disease to a very low level and maintain it at this same level for the entire period of the control program.

CHAPTER FIVE
CONCLUSION AND RECOMMENDATIONS

5.1 Conclusion

In this study, I have presented and analyzed the mathematical model for the transmission dynamics and the control of Newcastle Disease (ND) among the village chicken population. The aim was to get an insight of the transmission dynamics of ND by considering the unhygienic environment and wild birds as the vital sources of NDV to the village chicken population. The main tasks of the present work were: (1) to formulate and analyze a basic mathematical model for ND, (2) to formulate and analyze a mathematical model of ND with vaccination, culling and environmental hygiene and sanitation control strategies, (3) to evaluate the cost-effectiveness in the control of ND, and (4) to analyze the economic burden of ND at the family level.

Using the Next Generation Method approach of Van den Driessche and Watmough (2002), the basic reproduction number $\mathcal{R}_0$ which represents the number of secondary cases which one case would produce in a completely susceptible population is derived. Stability analysis of the model equilibria showed that the disease-free equilibrium exists and is globally asymptotically stable (GAS) when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$. Similarly, the model endemic equilibrium exists and is GAS stable if and only if $\mathcal{R}_0 > 1$. This basic model undergoes the phenomenon of forward bifurcation and the requirement $\mathcal{R}_0 < 1$ is a necessary and sufficient condition for ND disease elimination in village chicken population.

The bifurcation analysis of the ND model revealed that the equilibrium points of the basic model undergo the forward bifurcation and the endemic equilibrium point is locally asymptotically stable for $\mathcal{R}_0 < 1$ with $\mathcal{R}_0$ close to one. However, the sensitivity analysis of the basic model indicate that the transmission coefficient of NDV between the environment and the hosts is the most positive sensitive parameter in the transmission of the ND.

The basic model is then extended to include three time-dependent control variables: vaccination of the susceptible chicken u_1; culling of the infected chicken u_2; and environmental hygiene and sanitation u_3. The study established and proved the existence of an optimal control solution. The

necessary conditions for optimality were determined using Pontryagin's maximum principle (PMP). Based on the numerical results, The study reveal that the combination of the vaccination u_1 and environmental hygiene and sanitation u_3 controls reduce the number of the infected chicken and concentration of the NDV in the environment more than applying these control measures are applied singly. Thus, the most effective way to reduce the transmission of ND infection is to encourage the village chicken farmers to vaccinate their chicken regularly against ND infections and take preventive measures to improve environmental hygiene and sanitation around their poultry farms.

Further, the cost-effectiveness analysis shows that, the vaccination of the chicken population is less costly than any other control measure followed by the combination of vaccination and environmental hygiene and sanitation. The study reveals that, the economic loss at family level is due to the number of chicken which die due to the ND, the loss of production resulting from the reduction in the number of laid eggs, and the cost incurred to finance the control measures to prevent new infections and the spread of the ND and other chicken diseases in the chicken yards.

5.2 Recommendations

Population of the infected chickens can be reduced if $\mathcal{R}_0 < 1$, which is possible when vaccination is combined with maintaining a clean environment. We thus recommend the following to the chicken farmers, the government, and the policy makers:

(i) In order to avoid the economic losses due to the occurrence of the ND at family level, the village chicken farmers should be encouraged to timely vaccinate their chicken and make the area around the chicken yards as clean as possible through environmental hygiene and sanitation. These practices will help to increase the immunity to chicken but also prevent them from other bacterial and fungi disease that are caused by unhygienic environment.

(ii) The Government should initiate the education programmes to educate chicken farmers on how they can intensively manage their chicken as well as increasing their productivity.

(iii) Policy makers should enforce laws that restricts movements of chicken and their products from other countries especially during the outbreaks.

5.2.1 Limitations of the study

The studied model might not be perfect to some results due to the following limitations:

(i) The model under this study considered only three aspects: the horizontal transmission, environment and the environmental hygiene and sanitation for the transmission dynamics of Newcastle disease under the free range system.

(ii) The assumptions used to formulate the model limits some of the epidemiological factors for the dynamics of ND to the village chicken under the free range system.

(iii) The study investigated the economic consequence of the Newcastle disease only for two Districts of Bagamoyo and Kibaha, Pwani Region.

5.2.2 Future Work

Efforts were made to formulate and analyse a comprehensive mathematical model of the transmission dynamics of ND, however, this study is not exhaustive. Not all aspect of the transmission dynamics of ND among the village chicken population were included. Consequently, future research will potentially include some or all the following: age structure of the chicken population, seasonal variations (temperature and humidity), trans-ovarial transmission, human sub-model and other diseases in the chicken yards.

REFERENCES

Abate, A., Tiwari, A. and Sastry, S. (2009). Box invariance in biologically-inspired dynamical systems. *Automatica*. **45**(7): 1601–1610.

Alders, R. G., Spradbrow, P. and Young, M. (2009). Village chickens, poverty alleviation and the sustainable control of Newcastle disease. Australian Centre for International Agricultural Research.

Alexander, D. J. (2001). Newcastle disease. *British poultry science*. **42**(1): 5–22.

Alexander, D. J., Bell, J. G. and Alders, R. G. (2004). A Technology Review: Newcastle Disease, with Special Emphasis on its Effect on Village Chickens. number 161. Food & Agriculture Org.

Anishchenko, V. S., Vadivasova, T. E. and Strelkova, G. I. (2014). Stability of dynamical systems: Linear approach. *In:* Deterministic Nonlinear Systems. Springer. pp. 23–35.

Anita, S., Capasso, V. and Arnautu, V. (2011). An Introduction to Optimal Control Problems in Life Sciences and Economics: From Mathematical Models to Numerical Simulation with MATLAB. Springer.

Asamoah, J. K. K., Oduro, F. T., Bonyah, E. and Seidu, B. (2017). Modelling of rabies transmission dynamics using optimal control analysis. *Journal of Applied Mathematics*.

Asante, F. A. and Asenso-Okyere, K. (2003). Economic burden of malaria in Ghana. *World Health Organization (WHO)*. pp. 1–81.

Ashraf, A. and Shah, M. (2014). Newcastle disease: Present status and future Modeling the Dynamics, Control and economic loss of Newcastle Disease in village chicken: A case of Pwani Region in Tanzaniachallenges for developing countries. *African Journal of Microbiology Research*. **8**(5): 411–416.

Athithan, S. and Ghosh, M. (2015). Stability analysis and optimal control of a malaria model with larvivorous fish as biological control agent. *Applied Mathematics and Information Sciences*. **9**(4): 1893.

Awan, M. A., Otte, M. and James, A. (1994). The epidemiology of Newcastle disease in rural poultry: a review. *Avian Pathology*. **23**(3): 405–423.

Blayneh, K., Cao, Y. and Kwon, H.-D. (2009). Optimal control of vector-borne diseases: treatment and prevention. *Discrete and Continuous Dynamical Systems B*. **11**(3): 587–611.

Bloom, D. E., Cafiero, E., Jané-Llopis, E., Abrahams-Gessel, S., Bloom, L. R., Fathima, S., Feigl, A. B., Gaziano, T., Hamandi, A., Mowafi, M. *et al.* (2012). The global economic burden of noncommunicable diseases. Technical report. Program on the Global Demography of Aging.

Bornaa, C. S., Makinde, O. D. and Seini, I. Y. (2015). Eco-epidemiological model and optimal control of disease transmission between humans and animals. *Communications in Mathematical Biology and Neuroscience*. **2015**: Article–ID.

Brown, V. R. and Bevins, S. N. (2017). A review of virulent newcastle disease viruses in the united states and the role of wild birds in viral persistence and spread. *Veterinary Research*. **48**(1): 68.

Buonomo, B. and Vargas-De-León, C. (2013). Stability and bifurcation analysis of a vector-bias model of malaria transmission. *Mathematical Biosciences*. **242**(1): 59–67.

Castillo-Chavez, C., Feng, Z. and Huang, W. (2002). On the computation of ro and its role on global stability in mathematical approaches for emerging and re-emerging infectious diseases. *Mathematical Approaches for Emerging and Reemerging Infectious Diseases: an introduction*. **1**: 229.

Castillo-Chavez, C. and Song, B. (2004). Dynamical models of tuberculosis and their applications. *Mathematical Biosciences and Engineering*. **1**(2): 361–404.

Chitnis, N., Hyman, J. M. and Cushing, J. M. (2008). Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. *Bulletin of Mathematical Biology*. **70**(5): 1272.

Chuma, F., Mwanga, G. G. and Kajunguri, D. (2018). Modeling the role of wild birds and environment in the dynamics of newcastle disease in village chicken. *Asian Journal of Mathematics and Application*. **2018**(446): 23.

Collins, C., Fister, K. R., Key, B. and Williams, M. (2009). Blasting neuroblastoma using optimal control of chemotherapy. *Mathematical Biosciences and Engineering*. **6**: 451–467.

Daut, E. F., Lahodny Jr, G., Peterson, M. J. and Ivanek, R. (2016). Interacting effects of newcastle disease transmission and illegal trade on a wild population of white-winged parakeets in Peru: A modeling approach. *PloS One*. **11**(1).

Diekmann, O., Heesterbeek, J. and Roberts, M. (2009). The construction of next-generation matrices for compartmental epidemic models. *Journal of the Royal Society Interface*. p. rsif20090386.

Ding, D., Lawson, K. D., Kolbe-Alexander, T. L., Finkelstein, E. A., Katzmarzyk, P. T., Van Mechelen, W., Pratt, M., Committee, L. P. A. S. . E. *et al.* (2016). The economic burden of physical inactivity: a global analysis of major non-communicable diseases. *The Lancet*. **388**(10051): 1311–1324.

Dortmans, J. C., Koch, G., Rottier, P. J. and Peeters, B. P. (2011). Virulence of newcastle disease virus: what is known far?. *Veterinary Research*. **42**(1): 1.

Dyachenko, A. (2014). Total nonnegativity of infinite hurwitz matrices of entire and meromorphic functions. *Complex Analysis and Operator Theory*. **8**(5): 1097–1127.

Evans, L. C. (1983). An introduction to mathematical optimal control theory versio 2. *Lecture notes available at http://math. berkeley. edu.* .

Fallat, S. M. and Johnson, C. R. (2011). Totally nonnegative matrices. Vol. 35. Princeton University Press.

Fleming, W. and Rishel, R. (1975). Optimal deterministic and stochastic control. *Applications of Mathematics, Springer, Berlin. MATH.* .

Foppa, I. M. (2005). The basic reproductive number of tick–borne encephalitis virus. *Journal of Mathematical Biology*. **51**(6): 616–628.

Gilchrist, P. (2005). Involvement of free-flying wild birds in the spread of the viruses of avian influenza, newcastle disease and infectious bursal disease from poultry products to commercial poultry. *World's Poultry Science Journal*. **61**(02): 198–214.

Hailu, A., Lindtjørn, B., Deressa, W., Gari, T., Loha, E. and Robberstad, B. (2017). Economic burden of malaria and predictors of cost variability to rural households in South-Central Ethiopia. *PloS One*. **12**(10).

Hartemink, N., Randolph, S., Davis, S. and Heesterbeek, J. (2008). The basic reproduction number for complex disease systems: Defining r 0 for tick-borne infections. *The American Naturalist*. **171**(6): 743–754.

Hethcote, H. W. (2000). The mathematics of infectious diseases. *SIAM Review*. **42**(4): 599–653.

Hove-Musekwa, S. D., Nyabadza, F., Mambili-Mamboundou, H., Chiyaka, C. and Mukandavire, Z. (2014). Cost-effectiveness analysis of hospitalization and home-based care strategies for people living with hiv/aids: The case of Zimbabwe. *International Scholarly Research Notices*. **2014**.

Hugo, A., Makinde, O. D., Kumar, S. and Chibwana, F. F. (2017). Optimal control and cost effectiveness analysis for newcastle disease eco-epidemiological model in Tanzania. *Journal of Biological Dynamics*. **11**(1): 190–209.

Hunter, J. K. (2011). Introduction to dynamical systems. *UCDavis Mathematics MAT A*. **207**: 2011.

Ibitoye, E., Jimoh, A. and Mungadi, H. (2013). A retrospective (2007–2011) analysis of newcastle disease diagnosed at avian clinic of veterinary teaching hospital, Usmanu Danfodio University Sokoto, Nigeria. *Current Research in Poultry Science*. **3**(1): 12–17.

Joshi, H. R., Lenhart, S., Li, M. Y. and Wang, L. (2006). Optimal control methods applied to disease models. *Contemporary Mathematics*. **410**: 187–208.

Kahuru, J., Luboobi, L. and Nkansah-Gyekye, Y. (2017*a*). Stability analysis of the dynamics of tungiasis transmission in endemic areas. *Asian Journal of Mathematics and Applications*. **2017**.

Kahuru, J., Luboobi, L. S. and Nkansah-Gyekye, Y. (2017*b*). Optimal control techniques on a mathematical model for the dynamics of tungiasis in a community. *International Journal of Mathematics and Mathematical Sciences*. **2017**.

Kapczynski, D. R., Afonso, C. L. and Miller, P. J. (2013). Immune responses of poultry to newcastle disease virus. *Developmental & Comparative Immunology*. **41**(3): 447–453.

Kar, T. and Ghosh, B. (2012). Sustainability and optimal control of an exploited prey predator system through provision of alternative food to predator. *Biosystems*. **109**(2): 220–232.

Kim, B. N., Nah, K., Chu, C., Ryu, S. U., Kang, Y. H. and Kim, Y. (2012). Optimal control strategy of plasmodium vivax malaria transmission in Korea. *Osong Public Health and Research Perspectives*. **3**(3): 128–136.

Kinene, T., Luboobi, L. S., Nannyonga, B. and Mwanga, G. G. (2015). A mathematical model for the dynamics and cost effectiveness of the current controls of cassava brown streak disease in Uganda. *Journal of Mathematical and Computational Science*. **5**(4): 567.

Knueppel, D., Coppolillo, P., Msago, A., Msoffe, P., Mutekanga, D. and Cardona, C. (2009). Improving poultry production for sustainability in the Ruaha Landscape, Tanzania. *Wildlife Conservation Society TransLinks Program. Retrieved June*. **11**.

Kung'aro, M., Luboobi, L. and Shahada, F. (2015). Application of optimal control strategies for the dynamics of yellow fever. *Journal of Mathematical and Computational Science*. **5**(3): 430.

La Salle, J. P. (1976). The Stability of Dynamical Systems. SIAM.

Lawal, J., Jajere, S., Mustapha, M., Bello, A., Wakil, Y., Geidam, Y., Ibrahim, U. and Gulani, I. (2015). Prevalence of newcastle disease in gombe, northeastern nigeria: A ten-year retrospective study (2004-2013). *British Microbiology Research Journal*. **6**(6): 367.

Lenhart, S. and Workman, J. T. (2007). Optimal Control Applied to Biological Models. Crc Press.

Lucchetti, J., Roy, M. and Martcheva, M. (2009). An avian influenza model and its fit to human avian influenza cases. *Advances in Disease Epidemiology, Nova Science Publishers, New York*. pp. 1–30.

Macpherson (1956). Some observations on the epizootiology of newcastle disease. *Canadian Journal of Comparative Medicine and Veterinary Science*. **20**(5): 155.

Mafuta, P., Mushanyu, J., Mushayabasa, S. and Bhunu, C. P. (2013). Transmission dynamics of trichomoniasis in bisexuals without the E. *World Journal of Modelling and Simulation*. **9**(4): 302–320.

Martin, P. and row, P. (1992). The epidemiology of newcastle disease in village chickens. *Newcastle Disease in Village Chickens*. pp. 40–45.

McAsey, M., Mou, L. and Han, W. (2012). Convergence of the forward-backward sweep method in optimal control. *Computational Optimization and Applications*. **53**(1): 207–226.

McDermott, J., Coleman, P. and Randolph, T. (2001). Methods for assessing the impact of infectious diseases of livestock-their role in improving the control of newcastle disease in Southern Africa. In: *ACIAR proceedings*. ACIAR; 1998. pp. 118–128.

Mlay, G. M., Luboobi, L., Kuznetsov, D. and Shahada, F. (2015). Optimal treatment and vaccination control strategies for the dynamics of pulmonary tuberclosis. *Int. J. Adv. Appl. Math. and Mech*. **2**(3): 196–207.

Mpeshe, S. C., Luboobi, L. S. and Nkansah-gyekye, Y. (2014*a*). Optimal control strategies for the dynamics of rift valley fever. *Commun. Optimiz. Theory*. **3**: 1–18.

Mpeshe, S. C., Luboobi, L. S. and Nkansah-Gyekye, Y. (2014*b*). Stability analysis of the rift valley fever dynamical model. *Journal of Mathematical and Computational Science*. **4**(4): 740.

Munir, M., Chowdhury, M. and Ahmed, Z. (2016). Emergence of new sub-genotypes of newcastle disease virus in Pakistan. *J Avian Res*. **2**(1): 1–7.

Mushayabasa, S., Marijani, T. and Masocha, M. (2017). Dynamical analysis and control strategies in modeling anthrax. *Computational and Applied Mathematics*. **36**(3): 1333–1348.

Mwanga, G. G. *et al.* (2014). Mathematical modeling and optimal control of malaria. *A PhD Thesis, Acta Lappeenranta University*. .

Namawejje, H., Ghosh, S., Ferrari, M. and Luboobi, L. S. (2015). Modeling the impact of three dose vaccination and treatment strategies on optimal control of rotavirus disease. *Asian Journal of Mathematics and Applications*. **2015**.

Nwanta, J., Abdu, P. and Ezema, W. (2008). Epidemiology, challenges and prospects for control of newcastle disease in village poultry in Nigeria. *World's Poultry Science Journal*. **64**(01): 119–127.

Nyerere, N., Luboobi, L. and Nkansah-Gyekye, Y. (2014). Bifurcation and stability analysis of the dynamics of tuberculosis model incorporating, vaccination, screening and treatment. *Communications in Mathematical biology and Neuroscience*. **2014**: Article–ID.

Okosun, K. O., Rachid, O. and Marcus, N. (2013). Optimal control strategies and cost-effectiveness analysis of a malaria model. *BioSystems*. **111**(2): 83–101.

Olaniyi, S., Lawal, M. A. and Obabiyi, O. S. (2016). Stability and sensitivity analysis of a deterministic epidemiological model with pseudo-recovery. *IAENG International Journal of Applied Mathematics*. **46**(2): 160–167.

Oluwayelu, D. O., Adebiyi, A. I., Olaniyan, I., Ezewele, P. and Aina, O. (2014). Occurrence of newcastle disease and infectious bursal disease virus antibodies in double-spurred francolins in Nigeria. *Journal of Veterinary Medicine*. **2014**.

Ostermann, A. (2005). Sensitivity analysis. *In:* Analyzing Uncertainty in Civil Engineering. Springer. pp. 101–114.

Otieno, G., Koske, J. K. and Mutiso, J. M. (2016). Cost effectiveness analysis of optimal malaria control strategies in Kenya. *Mathematics*. **4**(1): 14.

Otte, M., Nugent, R., McLeod, A. *et al.* (2004). Transboundary animal diseases: Assessment of socio-economic impacts and institutional responses. *Rome, Italy: Food and Agriculture Organization (FAO)*. .

Perry, B., Kalpravidh, W., Coleman, P., Horst, H., McDermott, J., Randolph, T. and Gleeson, L. (1999). The economic impact of foot and mouth disease and its control in South-East

Asia: a preliminary assessment with special reference to Thailand.. *Technical scientific Review, office of Epizootic diseases*. **18**(2): 478–497.

Poggiolini, L. and Spadini, M. (2011). Strong local optimality for a bang-bang trajectory in a mayer problem. *SIAM Journal on Control and Optimization*. **49**(1): 140–161.

Rist, C. L., Ngonghala, C. N., Garchitorena, A., Brook, C. E., Ramananjato, R., Miller, A. C., Randrianarivelojosia, M., Wright, P. C., Gillespie, T. R. and Bonds, M. H. (2015). Modeling the burden of poultry disease on the rural poor in Madagascar. *One Health*. **1**: 60–65.

Rodrigues, P., Silva, C. J. and Torres, D. F. (2014). Cost-effectiveness analysis of optimal control measures for tuberculosis. *Bulletin of Mathematical Biology*. **76**(10): 2627–2645.

Sachs, J. and Malaney, P. (2002). The economic and social burden of malaria. *Nature*. **415**(6872): 680.

Sadiq, S. F., Khan, M. A., Islam, S., Zaman, G., Jung, H. and Khan, S. A. (2014). Optimal control of an epidemic model of leptospirosis with nonlinear saturated incidences. *Annu. Res. Rev. Biol*. **4**(3): 560–576.

Sagamiko, T., Shaban, N., Nahonyo, C. and Makinde, O. (2015). Optimal control of a threatened wildebeest-lion prey-predator system in the Serengeti ecosystem. . .

Schńttler, H. and Ledzewicz, U. (2012). The pontryagin maximum principle: From necessary conditions to the construction of an optimal solution. *In:* Geometric Optimal Control. Springer. pp. 83–194.

Seidu, B. and Makinde, O. (2014). Optimal control of hiv/aids in the workplace in the presence of careless individuals. *Computational and Mathematical Methods in Medicine*. p. 19.

Selemani, M. A., Luboobi, L. S. and Nkansah-Gyekye, Y. (2016). On stability of the in-human host and in-mosquito dynamics of malaria parasite. *Asian Journal of Mathematics and Applications*. **2016**(353): 23.

Sharif, A., Ahmad, T., Umer, M., Rehman, A., Hussain, Z. *et al.* (2014). Prevention and control of newcastle disease. *International Journal of Agriculture Innovations and Research*. **3**(2): 454–460.

Singh, D., Kumar, S., Singh, B. and Bardhan, D. (2014). Economic losses due to important diseases of bovines in Central Indiasagamiko. *Veterinary World*. **7**(8).

Spradbrow, P. (2001). The epidemiology of newcastle disease in village chickens. In: *ACIAR Proceedings*. ACIAR; 1998. pp. 53–55.

Swai, E., Kessy, M., Sanka, P. and Mtui, P. (2011). A serological survey for infectious bursal disease virus antibodies in free-range village chickens in Northern Tanzania. *Journal of the South African Veterinary Association*. **82**(1): 32–35.

Tumwiine, J., Mugisha, J. and Luboobi, L. (2010). A host-vector model for malaria with infective immigrants. *Journal of Mathematical Analysis and Applications*. **361**(1): 139–149.

Van den Driessche, P. and Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences*. **180**(1): 29–48.

Wang, J. and Modnak, C. (2011). Modeling cholera dynamics with controls. *Canadian Applied Mathematics Quarterly*. **19**(3): 255–273.

Wiggins, S. (2003). Introduction to Applied Nonlinear Dynamical Systems and Chaos. Vol. 2. Springer Science & Business Media.

Yongolo, M. G., Christensen, H., Handberg, K., Minga, U. and Olsen, J. E. (2011). On the origin and diversity of newcastle disease virus in Tanzania. *Onderstepoort Journal of Veterinary Research*. **78**(1): 1–8.

Yongolo, M., Machangu, A. M. and Minga, U. (2002). Newcastle disease and infectious bursal disease among free-range village chickens in tanzania. *Characteristics and parameters of family poultry production in Africa. IAEA, Vienna*.

APPENDICES

INDEX 1: MATLAB code for the Bifurcation analysis diagram

Bifurcation diagram codes Recall the file served as 'mark2.m'file $R0_value = 0.01 : 0.01 : 2;$ $Root_array = zeros(length(R0_value), 2);$

Parameter values used

$t = 5;$

$psi = 0.0083 : 0.000001 : 1; gamma = 0.067; mu = 0.000548; delta_c = 0.01984; delta_b = 0.025; a = 0.01; d = 0.025; alpha_c = 0.01667; alpha_b = 0.002; mu_v = 0.00219; rho = 0.9; phi = 0.02; b = 0.021; kappa = 10000; N_b = 200000; N_c = 300000; holdonfori = 1 : 1 : length(R0_value); R0 = R0_value(i); varpi = -kappa*mu^3*phi*rho*gamma*N_b*(mu_v + gamma^2)*kappa*mu^2*N_b*delta_c*mu_v - gamma*a*kappa*mu^3*N_b*(mu_v - gamma^2)*mu*N_b*alpha_b*d*N_b*(delta_b - gamma)*mu^2*N_b - alpha_b*d*N_b*(delta_b - gamma^2)*a*kappa*mu^2*N_b*mu_v + gamma^2*a*mu*N_b*alpha_c*d*N_c + gamma^2*a*N_b*alpha_c*d*N_c*delta_b + gamma^2*mu*rho*alpha_c*b*N_c*d*N_b - gamma^2*mu*N_b*alpha_b*d*N_b*delta_c + gamma^2*rho*alpha_c*b*N_c*d*N_b*delta_b - gamma^2*N_b*alpha_b*d*N_b*delta_b*delta_c - gamma*mu^2*N_b*alpha_b*d*N_b*delta_c - gamma*mu^2*N_b*alpha_c*d*N_c*delta_b + kappa*mu^3*N_b*delta_b*delta_c*mu_v + 2*gamma*kappa*mu^3*N_b*delta_b*mu_v + 2*gamma*kappa*mu^3*N_b*delta_c*mu_v - gamma^2*mu*N_b*alpha_c*d*N_c*delta_b + gamma^2*kappa*mu^2*N_b*delta_b*mu_v + kappa*mu^5*N_b*mu_v + gamma*a*kappa*mu*rho*N_b*delta_b*delta_c*mu_v - kappa*mu^2*phi*rho*gamma*N_b*delta_c*mu_v + gamma^2*kappa*mu*N_b*delta_b*delta_c*mu_v + 2*gamma*kappa*mu^2*N_b*delta_b*delta_c*mu_v - gamma^2*a*kappa*mu*N_b*delta_c*mu_v - gamma^2*a*kappa*N_b*delta_b*delta_c*mu_v - gamma^2*a*mu*rho*N_b*alpha_c*d*N_c - gamma^2*a*rho*N_b*alpha_c*d*N_c*delta_b + gamma^2*rho*N_b*alpha_b*d*N_b*delta_b*delta_c - gamma*a*kappa*mu^2*N_b*delta_c*mu_v + gamma*mu*phi*rho*gamma*N_b*alpha_c*d*N_c - gamma*mu*N_b*alpha_b*d*N_b*delta_b*delta_c + gamma^2*a*kappa*mu^2*rho*N_b*mu_v + gamma*a*kappa*mu^3*rho*N_b*mu_v - gamma^2*a*kappa*mu*N_b*delta_b*mu_v + gamma^2*mu*rho*N_b*alpha_b*d*N_b*delta_b - gamma*a*kappa*mu^2*N_b*delta_b*mu_v - gamma*kappa*mu^2*phi*rho*gamma*N_b*mu_v + gamma*mu^2*rho*N_b*alpha_b*d*N_b*delta_b + gamma^2*a*kappa*mu*rho*N_b*delta_b*$

$mu_v + gamma * a * kappa * mu^2 * rho * N_b * delta_b * mu_v + gamma^2 * a * kappa * mu * rho * N_b * delta_c * mu_v + gamma^2 * a * kappa * rho * N_b * delta_b * delta_c * mu_v + gamma * a * kappa * mu^2 * rho * N_b * delta_c * mu_v - gamma * a * kappa * mu * N_b * delta_b * delta_c * mu_v - gamma * kappa * mu * phi * rho * gamma * N_b * delta_c * mu_v + gamma * mu * rho * N_b * alpha_b * d * N_b * delta_b * delta_c + kappa * mu^4 * N_b * delta_c * mu_v - gamma * mu^3 * N_b * alpha_c * d * N_c - gamma^2 * mu * alpha_c * b * N_c * d * N_b - gamma^2 * alpha_c * b * N_c * d * N_b * delta_b + gamma^2 * kappa * mu^3 * N_b * mu_v + 2 * gamma * kappa * mu^4 * N_b * mu_v - gamma^2 * mu^2 * N_b * alpha_c * d * N_c - gamma^2 * mu^2 * N_b * alpha_b * d * N_b - gamma * mu^3 * N_b * alpha_b * d * N_b + kappa * mu^4 * N_b * delta_b * mu_v;$

$tau = gamma * (a * gamma * kappa * mu * rho * mu_v + a * gamma * kappa * rho * delta_b * mu_v - a * gamma * kappa * mu * mu_v - a * gamma * kappa * delta_b * (mu_v + gamma * kappa) * mu^2 * mu_v + gamma * kappa * mu * delta_b * (mu_v + gamma) * rho * alpha_b * d * N_b * delta_b + kappa * mu^3 * mu_v + kappa * mu^2 * delta_b * mu_v - kappa * mu * phi * rho * gamma * mu_v - gamma * mu * alpha_b * d * N_b - gamma * alpha_b * d * N_b * delta_b) * N_b;$

$psi = 1/2 * (varpi./tau);$

$H = 0; I_r = 0; A = mu * N_c * (delta_c + mu) * (mu + gamma) * (kappa + H);$

$B = N_b * N_c * (kappa + H) + b * N_c * (delta_c + mu) * (mu + gamma) * (kappa + H) * I_r + mu * N_b * N_c * (kappa + H) * (delta_c + mu) * (mu + gamma);$

$C = b * mu * N_c * (kappa + H) * I_r + d * mu * gamma * N_b * (N_c).^2 * (1 - R0);$

$p = [A, B, C]; r = roots(p); len = length(r);$

$for\ t = 1 : 1 : len if(imag(r(t)) \ = 0 || real(r(t) < 0));$

$Root_array(i, t) = 0;$

$else$

$Root_array(i, t) = r(t);$

$end; end; end; f = 1;$

$f = f + 1; R0_value_Cr = f; for j = R0_value_Cr : 1 : length(R0_value) Root_array(j, :) = sort(Root_array(j, :)); end f1 = R0_value_Cr; while(Root_array(f1, 1) = 0) f1 = f1 + 1; end R0_value_Cr2 = f1; Zero_1st = R0_value(1, 1 : R0_value_Cr2 - 1);$

$y_zero = zeros(2, length(Zero_1st)); Unstable = R0_value(1, R0_value_Cr : length(R0_value));$

$figure(1)$

$plot(Unstable, Root_array(R0_value_Cr : length(R0_value), 2), 'r--', 'LineWidth', 4)$

$Xlabel('BasicReproductionnumber, R_0,' ,' FontSize', 12)$

$Ylabel('Fractionofinfectedlivestock',' FontSize', 12)$

$hold\ off$

$figure(2)$

$plot(R0_value, Root_array(:,1),' r--', R0_value, Root_array(:,2),' b',' LineWidth', 4)$

$Xlabel('BasicReproductionnumber, R_0,' ,' FontSize', 12)$

$Ylabel('Fractionofinfectedchicken',' FontSize', 12)$

INDEX 2. MATLAB Code used for the simulation of the ND basic model

clear all

close all

$tspan = [0\ 1000];$

$y0 = [2000\ \ 1200\ \ 1800\ \ 3000\ \ 1000\ \ 900\ \ 700\ \ 5000];$

$figure(1)$

$plot(t, y(:,1),' g',' LineWidth', 1.5)$

$Xlabel('Time(days)')$

$Ylabel('Susceptiblevillagechicken')$

$hold\ off$

$hold\ on$

$figure(2)$

$plot(t, y(:,2),' r',' LineWidth', 1.5)$

$Xlabel('Time(days)')$

$Ylabel('Exposedvillagechicken')$

$hold\ off$

$hold\ on$

$figure(3)plot(t, y(:,3),' b',' LineWidth', 1.5)$

$Xlabel('Time(days)')$

$Ylabel('Infectedvillagechicken')hold\ off$

$hold\ on$

$figure(4)$

$plot(t, y(:,4),' m',' LineWidth', 1.5)$

$Xlabel('Time(days)')$

$Ylabel('Susceptiblewildbirdpopulation')$

$hold\ off$

$hold\ on$

$figure(5)$

$plot(t, y(:, 5), 'k', 'LineWidth', 1.5)$

$Xlabel('Time(days)')$

$Ylabel('Exposedwildbirdspopulation')$

$hold\ off$

$hold\ on$

$figure(6)$

$plot(t, y(:, 6), 'r', 'LineWidth', 1.5)$

$Xlabel('Time(days)')$

$Ylabel('Infectedwildbirdspopulation')$

$hold\ off$

$hold\ on$

$figure(7)$

$plot(t, y(:, 7), '.b', 'LineWidth', 1.5)$

$Xlabel('Time(days)')$

$Ylabel('Carrierwildbirdspopulation')$

$hold\ off$

$hold\ on$

$figure(8)$

$plot(t, y(:, 8), '.m', 'LineWidth', 1.5)$

$Xlabel('Time(days)')$

$Ylabel('NDVintheEnvironment')$

$hold\ off$

$hold\ on$

$figure(9)$

$plot(t, y(:, 1), 'g', t, y(:, 2), 'r', t, y(:, 3), 'b', t, y(:, 4), 'm', t, y(:, 5), 'k', t, y(:, 6), '.r', t, y(:, 7),$
$'.b', t, y(:, 8), '.m', 'LineWidth', 1.5)$

$Xlabel('Time(days)')$

$Ylabel('TotalPopulations')$

$legend('Susceptiblechicken',' Exposedchicken',' Infectedchicken',' Susceptiblewildbirds',$

$'Exposedwildbirds',' Infectedwildbirds',' Carrierwildbirds',' NDVinEnvironment')$

$hold\,off$

INDEX 3. Main Function for plotting the equilibrium points

$functiondy = Trial1(, y)$

$dy = zeros(size(y));$

$Forthediseasefreeequilibriumpoint$

$psi = 0.0001; L = 0.0067; l = 0.2; delta_c = 0.02; delta_b = 0.075; a = 0.03; d = 0.001; alpha = 0.1; kappa = 10000; mu_v = 0.137; rho = 0.1; phi = 0.004; r = 0.28; sigma_1 = 100; sigma_2 = 200; Fortheendemicequilibriumpoint$

$psi = 0.0083; L = 0.067; l = 0.000548; delta_c = 0.01984; delta_b = 0.025; a = 0.01; d = 0.025; alpha_c = 0.01667; alpha_b = 0.002; mu_v = 0.00219; rho = 0.9; phi = 0.02; r = 0.021; kappa = 10000;$

$Sc = y(1); Ec = y(2); Ic = y(3); Sb = y(4); Eb = y(5); Ib = y(6); Ir = y(7); H = y(8);$

$N_b = 1000; N_c = 2000;$

$EquationsoftheNDbasicmodel$

$dy(1) = l * N_c - (r * Ir./N_b + psi * Ic./N_c + (d * H)./(kappa + H)) * Sc - l * Sc;$

$dy(2) = (r * Ir./N_b + psi * Ic./N_c + (d * H)./(kappa + H)) * Sc - (l + L) * Ec;$

$dy(3) = L * Ec - (delta_c + l) * Ic;$

$dy(4) = l * N_b - (phi * Ib./N_b + a * Ir./N_b + (d * H)./(kappa + H)) * Sb - l * Sb;$

$dy(5) = (phi * Ib./N_b + a * Ir./N_b + (d * H)./(kappa + H)) * Sb - (l + L) * Eb;$

$dy(6) = rho * L * Eb - (delta_b + l) * Ib;$

$dy(7) = (1 - rho) * L * Eb - l * Ir;$

$dy(8) = alpha_c * Ic + alpha_b * (Ib + Ir) - mu_v * H;$

INDEX 4. Endemic equilibrium point of the ND model

clear

close all

$tspan = [0\ \ 1000];$

$y0 = [1200\ \ 300\ \ 500\ \ 3000\ \ 400\ \ 900\ \ 700\ \ 5000];\ \ [t, y] = ode45(@Trial1,\ \ tspan, y0);$

$figure(5)$

$plot(t, y(:, 5), 'g', 'LineWidth', 1.5)$

$legend('I_c')$

$Xlabel('Time[days]')$

$Ylabel('Exposedwildbirdspopulation')$

$hold\ \ on$

$tspan = [0\ \ 1000]$

$y0 = [1200\ \ 300\ \ 500\ \ 3000\ \ 800\ \ 900\ \ 700\ \ 5000]; [t, y] = ode45(@Trial1, tspan, y0); plot(t, y(:, 5), 'b', 'LineWidth', 1.5)$

$xlabel('Time[days]')$

$ylabel('Exposedwildbirdspopulation')$

$tspan = [0\ \ 1000]; y0 = [1200\ \ 300\ \ 500\ \ 3000\ \ 1200\ \ 900\ \ 700\ \ 5000];\ \ [t, y] = ode45(@Trial1,\ \ tspan, y0);$

$plot(t, y(:, 5), 'm', 'LineWidth', 1.5)$

$xlabel('Time[days]')$

$ylabel('Exposedwildbirdspopulation')$

$hold\ \ on$

$tspan = [0\ \ 1000];$

$y0 = [1200\ \ 300\ \ 500\ \ 3000\ \ 2000\ \ 900\ \ 700\ \ 5000];$

$plot(t, y(:, 5), 'r', 'LineWidth', 1.5)$

$Xlabel('Time(days)')$

$Ylabel('Exposedwildbirdspopulation')$

$legend('E_b(0) = 400', 'E_b(0) = 800', 'E_b(0) = 1400', 'E_b(0) = 2000')$

$hold\ off$

INDEX 5. MATLAB Code used for Numerical Simulations of the ND Model with control measures

The main file for calling the state and the adjoint systems

clc

clear all

close all

$t0 = 0; tf = 400; N = 8000; time = linspace(t0, tf, N);$

$y0 = [200000\ 12000\ 5000\ 0\ 300000\ 10000\ 400\ 500\ 5000\ 0];$

$Constant = [0.4\ 0.067\ 0.00058\ 0.002\ 0.025\ 0.01\ 0.1\ 0.001667\ 0.000002\ 0.00219$

$0.998\ 0.02\ 0.21\ 1000000\ 0.0001\ 0.0001\ 0.01\ 202\ 20\ 10\ 10\ 0.0001];$

$psi = Constant(1); gamma = Constant(2); l = Constant(3); delta_c = Constant(4);$

$delta_b = Constant(5); a = Constant(6); d = Constant(7); alpha_c = Constant(8);$

$alpha_b = Constant(9); mu_v = Constant(10); rho = Constant(11); phi = Constant(12);$

$q = Constant(13); kappa = Constant(14); Lambda_1 = Constant(15);$

$Lambda_2 = Constant(16); A1 = Constant(17); A2 = Constant(18); A3 = Constant(19);$

$D1 = Constant(20); D2 = Constant(21); D3 = Constant(22); varphi = Constant(23);$

$lf = [0\ 0\ 0\ 0\ 0\ 0\ 0\ 0\ 0];$

TEST SECTION

$u1\ u2\ u3$

$U = [0\ 0\ 0];$

$time0 = linspace(t0, tf, N + 1);$

$init = y0; h = (tf - t0)./N;$

$uu = linspace(0, 0, N + 1);$

$u1 = uu'; u2 = uu'; u3 = uu';$

$U = [u1u2u3];$

$Uu = [u2'u2'u3'];$

break

FORWARD RUNGE KUTTA FOR STATES

$[Tx, X] = rk4foward(@Mark1_Sstate, t0, tf, N, init, U, Constant);$

$X = X';$

$Tx = Tx';$

$figure(1)$

$subplot(2, 3, 1)$

$plot(Tx, (X(:, 1)), '-r', 'Linewidth', 1.5);$

$hold\ on$

$plot(Tx,(Y(:,1)),'--b','Linewidth',1.5);$

$hold\ off$

$set(gca,'FontSize',20)$

$title('A','FontSize',30)$

$Xlabel('Time(indays)','FontSize',15);$

$Ylabel('Susceptiblechicken','FontSize',15);$

$subplot(2,3,2)$

$plot(Tx,(X(:,2)),'-r','Linewidth',1.5);holdon$

$plot(Tx,(Y(:,2)),'--b','Linewidth',1.5);holdoff$

$set(gca,'FontSize',10)$

$title('B','FontSize',10)$

$Xlabel('Time(indays)','FontSize',10);$

$Ylabel('Latentinfectedchicken','FontSize',10);$

$subplot(2,3,3)$

$plot(Tx,(X(:,3)),'-r','Linewidth',1.5);holdon$

$plot(Tx,(Y(:,3)),'--b','Linewidth',1.5);holdoff$

$set(gca,'FontSize',10)$

$title('C','FontSize',10)$

$Xlabel('Time(indays)','FontSize',10);$

$Ylabel('Severelyinfectedchicken','FontSize',10);$

$subplot(2,3,4)$

$plot(Tx,log10(X(:,4)),'-r','Linewidth',1.5);$

$hold\ on$

$plot(Tx,log10(Y(:,4)),'--b','Linewidth',1.5);$

$hold\ off$

$set(gca,'FontSize',10)$

$title('D','FontSize',10)$

$Xlabel('Time(indays)','FontSize',10);$

$Ylabel('Vaccinatedchicken','FontSize',10);$

$subplot(2,3,5)$

$plot(Tx,(X(:,9)),'-r','Linewidth',1.5);$

$hold\ on$

$plot(Tx,(Y(:,9)),'--b','Linewidth',1.5);$

$hold\ off$

$set(gca,'FontSize',10)$

$title('E','FontSize',10)$

$Xlabel('Time(indays)','FontSize',10);$

$Ylabel('Environmentalhygiene','FontSize',10);$

$subplot(2,3,6)$

$plot(Tx,Uu(:,1),'y-',Tx,Uu(:,2),'--b',Tx,Uu(:,3),'k','LineWidth',3);$

$set(gca,'FontSize',10)$

$title('F','FontSize',10)$

$Xlabel('Time(indays)','FontSize',10);$

$Ylabel('controlprofiles','FontSize',10);$

$init = y0;$

$init2 = lf;$

$h = (tf - t0)/N;\ \ u = linspace(0.5, 0.5, N+1);\ \ u1 = u'; u2 = u'; u3 = u';\ \ U = [u1u2u3];$

break

IMPLEMENTATION OF THE ALGORITHM

Test 1 topping condition 1 delta = 0.01;

X=init;

i=0; mm=size(X);

NumXX =10e10;

Xnew = rand(N+1,mm(2)).*(repmat(X,N+1,1));

DenXnew=norm(Xnew);

while NumXX/DenXnew¿delta

Xold = Xnew;

oldu = U;

FORWARD RUNGE KUTTA FOR STATES

$[Tx, X] = rk4foward(@Mark1_State, t0, tf, N, init, U, Constant);$

BACKWARD RUNGE-KUTTA FOR CO-STATES

$[Tp, P] = rk4back(@Mark2_Costate, t0, tf, N, init2, U, X, Constant);$

UPDATE THE CONTROLS

$S_c = X(1,:); E_c = X(2,:); I_c = X(3,:); V = X(4,:); S_b = X(5,:); E_b = X(6,:); I_b = X(7,:); I_r = X(8,:); H = X(9,:); L1 = P(1,:); L2 = P(2,:); L3 = P(3,:); L4 = P(4,:); L5 = P(5,:); L6_V = P(6,:); L7 = P(7,:); L8 = P(8,:); L9 = P(9,:); g1 = S_c + E_c + I_c + V; g2 = S_b + E_b + I_b + I_r;$

Control cases of Newcastle disease transmission

$case1 : u1 \neq 0, u2 = 0, u3 = 0,$

$u1 = min(1, max(0, Beta1));$

$u2 = zeros(1, N + 1);$

$u3 = zeros(1, N + 1);$

$Uu = [u1' u2' u3'];$

$U = 0.5 * Uu + 0.5 * oldu;$

$Xnew = X';$

$NumXX = abs(norm(Xnew - Xold));$

$DenXnew = norm(Xnew);$

$i = i + 1 end$

PLOTTING

X=Xnew;

Tx =Tx';

XX=X(:,1); YY=X(:,2); VV=X(:,3); ZZ=X(:,4); EE=X(:,5);MM=X(:,6);GG=X(:,7);

QQ=X(:,8);KK=X(:,9);

Up =[0 0 0]; $[T, Y] = ode45(@Mark1_State, time, y0, [], Up, Constant);$

save Category2

save('case1State','X');

save('case1Control','Uu');

clf figure(1)

subplot(2,2,1)

plot(Tx,log10(X(:,1)),'r',T,log10(Y(:,1)),'–b','LineWidth',2);

Xlabel('Time (days)');

Ylabel('Susceptible village chicken');

title('(a)','FontSize',10)

legend('with control','without control',2);

subplot(2,2,2)

plot(Tx,log10(X(:,3)),'r',T,log10(Y(:,3)),'–b','LineWidth',2);

Xlabel('Time (days)');

Ylabel('Severely infected village chicken');

title('(b)','FontSize',10)

legend('with control','without control',2);

subplot(2,2,3)

plot(Tx,log10(X(:,9)),'r',T,log10(Y(:,9)),'–b','LineWidth',2);

Xlabel('Time(days)');

Ylabel('NDV in the environment');

title('(c)','FontSize',10)

legend('with control','without control',2);

subplot(2,2,4) plot(Tx,Uu(:,1),'c',Tx,Uu(:,2),'b',Tx,Uu(:,3),'–r','LineWidth',2);

Ylabel('Control Profile');

Xlabel('Time(days)');

title('(d)','FontSize',10)

$legend('u_1 \neq 0','u_2 \neq 0','u_3 \neq 0', 3)$

collect all the incidence terms in the ODE U =[0 0 0];

$[Tx, Y] = ode45(@Mark1_Sstate, time, y0, [], U, Constant);$

Y=(Y);

Inew=sum(Y(:,10))-sum(X(:,10))

Solution of the objective functional $I_c = X(:, 3); I_r = X(:, 8); H = X(:, 9);$

u1=Uu(:,1); u2=Uu(:,2); u3=Uu(:,3); t=T; n=length(t);

for i = 1:n

g3=$A1 * u1(i) * S_c(i) + A2 * u2(i) * I_c(i) + A3 * u3(i) * H(i);$

h1=(D1/2)*u1(i)*u1(i)+(D2/2)*u2(i)*u2(i)+(D3/2)*u3(i)*u3(i);

Q(i)=g3+h1;

cost=trapz(r,Q)

RESEARCH OUTPUTS

Published Papers

1. Chuma, F., Mwanga, G. G. (2019) Stability analysis of equilibrium points of newcastle disease model of village chicken in the presence of wild birds reservoir. *International Journal of Mathematical Sciences and Computing*

2. Chuma F., Mwanga G.G and Masanja V.G (2019) Application of optimal control theory to Newcastle disease dynamics in village chicken by considering wild birds as reservoir of disease virus. *Journal of Applied Mathematics*