An Eco-epidemiological Model for Newcastle Disease in Central Zone Tanzania

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Abstract

Newcastle disease is contagious bird disease which affects main domestic and wild avian species. A deterministic compartmental model for Newcastle disease (ND) is developed and analysed using ordinary differential equation theory. The uncertainties of model parameters were therefore examined using Markov Chain Monte Carlo (MCMC) simulations for the data of chicken death cases due to Newcastle disease from five districts in two regions in Tanzania. The parameter distribution was tested using MCMC convergence diagnostics. The graphical diagnostic test for MCMC used include Trace plots or time series plot, two-dimensional parameter plots and autocorrelation function plots. Hence, model parameters were successfully estimated for numerical simulations and the results of simulations were presented.

Keywords: Eco-epidemiology; Newcastle Disease; Parameter estimation

1. Introduction

Newcastle disease (ND) is a contagious bird disease which affects many domestic and wild avian species (Muturi et al. 2013). Chickens are most susceptible species and other species which the disease affects include turkeys, ducks, geese, parrots, pigeons and wild cormorants. ND was first recognised in 1926 in Java, Indonesia and in Newcastle-Upon-Tyne, England (Arifin et al. 2011, Alexander et al. 2004). After the disease had been identified many outbreaks were reported in different places in the world (Yongolo et al. 2011). ND stains have been classified and discussed by Dortmans et al (2011) and it was listed by Office International des Epizooties (OIE) or World Organization for Animal Health due to its severe effect in poultry. It is found throughout the world moreover, it seems to have been controlled in Canada, the United States and some Western European countries through vaccination and biosecurity measures (OIE 2015). In African countries, the disease is still endemic with poor surveillance and the most affected countries include Lesotho, Togo, Ghana, Zambia, Tanzania and Guinea (Gardner 2014). However, Sudan, Ethiopia and Madagascar have provided useful and detailed information about the disease transmission in Africa (Musako and Abolnik 2012).

Many households in Tanzania keep chickens especially the indigenous for different purposes. Tanzania had about 43.8 million chickens of which 41.9 million (96%) were local, 1.3 million (2.7%) were layers and 0.6 million (1.3%) were broilers (Chuwa 2012). Tanzania has been affected by ND adversely, however, there are little plausible explanations regarding history, origin and spread (Yongolo and Machangu 2009). The highest prevalence occurs in free-range chickens from June to October and the factors that influence the spread of ND in Tanzania have identified by Knueppel et al (2009). The first isolation and pathotyping were reported by Loretu and Mkaria after a survey carried out in Morogoro, Tanzania where farmers reported that ND was the most killer disease of chickens in rural areas (Yongolo and Machangu 2009). Controlling ND is still a big challenge in rural areas in Tanzania, it became discourages in many households to keep chickens in large flocks (Ibrahim et al. 2012). However, Rural Livelihood

Development Company (RLDC) helps the farmers to get additional food through poultry and generating additional family or individual income. RLDC implemented many poultry projects in different districts such as Bariadi in Simiyu region, Iramba, Singida and Manyoni in Singida region, Bahi and Mpwapwa in Dodoma region (Ibrahim et al. 2012). While the government initiated vaccination campaign using I-2 vaccine against chickens with ND in Lindi and Mtwara regions (MLFD 2010). The mortality rate was reduced by 4% and recommends that more efforts are required to out-scale subsidised vaccination programmes.

Studies by Van Boven et al (2008), Yongolo et al (2011) and Nabila et al (2014) in Tanzania recommended that ND vaccination provides an excellent means to lessen clinical signs of infection while Yongolo and Maeda (2009) suggested that vaccination programmes should be initiated strategically to protect chickens before the active period of ND infections in June. Therefore, in this paper, we intend to apply a mathematical model for studying the dynamical behaviour of ND in Tanzania in order to understand the mechanism responsible for persistence or extinction of species (Glaser 2007). Modelling infectious diseases in species provides an important insight into disease behaviour and control measures which can be used to evaluate the relevance of intervention programmes through epidemiological data and economic cost of infection (Makinde and Okosun 2011).

2. Model Formulation

In this section, we formulate and analyse a mathematical model of ND in Tanzania. The modelled populations include chickens and human being. The epidemiological model comprises of five subclasses namely susceptible chicken $S_1(t)$, infected chicken $I_1(t)$, susceptible human $S_2(t)$, infected human $I_2(t)$ and human recovery class $R_2(t)$. The model presented under the following assumptions: The growth rate of the chicken population follows a logistic function with intrinsic growth rate r and carrying capacity k. The chicken population gets an infection when it comes into contact with other infected chicken and this contact process is assumed to follow the simple mass action kinetics with β_1 as the force of infection while human get an infection with the force of infection β_2. The natural death rate of chicken μ_1 and induced death rate due to disease m reduces the chicken population. The human population suffers loss due to the natural death rate μ_2 and increases due to recovery rate θ through treatment rate γ. The predation functional response of the human towards susceptible as well as infected chicken is assumed to follow Michaelis-Menten kinetics and is modelled using a Holling type -II functional response with predation coefficients b_1, c_1 b_2, c_2 and half saturation constant a_1, a_2, n_1 and n_2. Consumed susceptible and infected chicken is converted into a human with efficiency α_1, α_2 α_3 and α_4. These assumptions lead into the following schematic flow diagram

2.1 Mathematical Equations

Using the Fig. 1, we formulate the following model equations

$$\left.\begin{aligned}
\frac{dS_1}{dt} &= r\left(1-\frac{S_1}{k}\right)S_1 - \beta_1 S_1 I_1 - \frac{b_1 S_1 S_2}{a_1+S_1} - \frac{b_2 S_1 I_2}{a_2+S_1} \\
\frac{dI_1}{dt} &= \beta_1 S_1 I_1 - mI_1 - \mu_1 I_1 - \frac{c_1 I_1 S_2}{n_1+I_1} - \frac{c_2 I_1 I_2}{n_2+I_1} \\
\frac{dS_2}{dt} &= \frac{\alpha_1 b_1 S_1 S_2}{a_1+S_1} + \frac{\alpha_2 c_1 I_1 S_2}{n_1+I_1} - \beta_2 S_2 I_1 - \mu_2 S_2 + \theta R_2 \\
\frac{dI_2}{dt} &= \beta_2 S_2 I_1 - \gamma I_2 - \mu_2 I_2 + \frac{\alpha_3 b_2 S_1 I_2}{a_2+S_1} + \frac{\alpha_4 c_2 I_1 I_2}{n_2+I_1} \\
\frac{dR_2}{dt} &= \gamma I_2 - \theta R_2 - \mu_2 R_2
\end{aligned}\right\} \quad (1)$$

System (1) has to be analysed with the following initial conditions:

$S_1 > 0,\ \ I_1 > 0,\ \ S_2 > 0,\ \ I_2 > 0,\ \ R_2 > 0.$

2.1.1 Non-dimensionalization of the model

Sometimes reduction parameters in the model simplify mathematical analysis. The process is known as non-dimensionalization (rescaling the model dimension). Now, we rescale the model (1) by assuming

$s_1 = \frac{S_1}{k},\ l_1 = \frac{I_1}{k},\ s_2 = \frac{S_2}{k},\ l_2 = \frac{I_2}{k},\ r_2 = \frac{R_2}{k}$ and $\tau = k\beta_1 t$.

The model (1) is then transformed into

$$\left.\begin{aligned}
\frac{ds_1}{d\tau} &= r_1(1-s_1)s_1 - s_1 l_1 - \frac{p_1 s_1 s_2}{a_3+s_1} - \frac{p_2 s_1 l_2}{a_4+s_1} \\
\frac{dl_1}{d\tau} &= s_1 l_1 - p_3 l_1 - \frac{p_4 l_1 s_2}{n_3+l_1} - \frac{p_5 l_1 l_2}{n_4+l_1} \\
\frac{ds_2}{d\tau} &= \frac{\alpha_1 p_1 s_1 s_2}{a_3+s_1} + \frac{\alpha_2 p_4 l_1 s_2}{n_3+l_1} - p_6 s_2 l_1 - p_7 s_2 + p_8 r_2 \\
\frac{dl_2}{d\tau} &= p_6 s_2 l_1 - p_9 l_2 + \frac{\alpha_3 p_2 s_1 l_2}{a_4+s_1} + \frac{\alpha_4 p_5 l_1 l_2}{n_4+l_1} \\
\frac{dr_2}{d\tau} &= p_{10} l_2 - p_{11} r_2
\end{aligned}\right\} \quad (2)$$

where

$$r_1 = \frac{r}{k\beta_1},\ \ p_2 = \frac{b_2}{k\beta_1},\ \ a_3 = \frac{a_1}{k},\ \ a_4 = \frac{a_2}{k},\ \ \ p_3 = \frac{m+\mu_1}{k\beta_1},\ \ p_4 = \frac{c_1}{k\beta_1},\ \ p_5 = \frac{c_2}{k\beta_1},\ \ p_6 = \frac{\beta_2}{\beta_1},$$

$$p_7 = \frac{\mu_2}{k\beta_1},\ \ p_8 = \frac{\theta}{k\beta_1},\ \ p_9 = \frac{\gamma+\mu_2}{k\beta_1},\ \ p_{10} = \frac{\gamma}{k\beta_1},\ \ p_{11} = \frac{\theta+\mu_2}{k\beta_1},\ \ n_3 = \frac{n_1}{k},\ \ n_4 = \frac{n_2}{k}.$$

System (2) has to be analysed with the following initial conditions:

$s_1 > 0,\ \ l_1 > 0,\ \ s_2 > 0,\ \ l_2 > 0,\ \ r_2 > 0.$

2.2 Model analysis

The system of equations (2) is quantitatively analysed to get insight into its dynamical features which will give a better understanding of the dynamical behaviour of ND infection.

2.2.1 Positivity and Boundedness of solutions

We denote by R_+^5 the set of the points $x_t = (x_1,\ x_2,\ x_3,\ x_4,\ x_5)$ in R^5 with the positive coordinates. We consider the system of the equation (2) with the initial values

$$x^0 = (x_1^0,\ x_2^0,\ x_3^0,\ x_4^0,\ x_5^0) \tag{3}$$

Using approach of Lungu et al (Lungu et al. 2013), we construct the lemma as follows

Lemma 1: *The system (2) can be transformed into the following differential inequalities*

$$\frac{dx_i}{dt} \geq A_i x_i + \sum_j^n B_{ij} x_j + \varepsilon\ , (i = 1.2,3,...,n), \tag{4}$$

where

$B_{ij} \geq 0, \qquad \varepsilon \geq 0$

If $x_i(0) \geq \varepsilon$ *then* $x_i(t) \geq 0$ *for all* $t > 0$ *and* $1 \leq i \leq n$.

We assume that $\varepsilon > 0$ *for the case,* $\varepsilon = 0$ *it approximates the system with the sequence* $\varepsilon = \varepsilon_k \downarrow 0$.

Proof by contradiction:

Suppose $x_i(0) \geq \varepsilon > 0$ for $1 \leq i \leq 5$ is not true. Then there exists the smallest number $t_0 > 0$ such that

$x_i(t) > 0$ for $1 \leq i \leq n$, $0 \leq t \leq t_0$,

$x_i(t_0) = 0$ for at least one i say $i = i_0$.

Then x_{i0} is a decreasing function at $t = t_0$, so that

$$\frac{dx_{i0}}{dt}(t_0) \leq 0 \tag{5}$$

At $x_{i0}(t)$, the differential inequality (4) becomes

$$\frac{dx_{i0}}{dt}(t_0) \geq \sum_j^n B_{ij} x_j + \varepsilon \geq \varepsilon > 0 \tag{6}$$

This contradicts our lemma (1).

For the state variables in our model, we always take

$s_1(0) \geq 0,\ \ l_1(0) \geq 0,\ \ s_2(0) \geq 0,\ \ l_2(0) \geq 0,\ \ r_2(0) \geq 0$.

Thus in the region, R_+^5 the model is epidemiologically and mathematically well posed and we can use it to study the dynamics of ND in Tanzania.

3. Steady states

The steady states of the system (2) are obtained by equating the equations of the system (2) equal to zero. The solution of $l_1^* = 0$ causes $l_2^* = r_2^* = 0$ and we solve for s_1 and s_2 as

$$s_1^* = \frac{p_7 a_3}{\alpha_1 p_1 - p_7}\ ,\ s_2^* = \frac{r_1 a_3 \alpha_1 (\alpha_1 p_1 - p_7 - p_7 a_3)}{(\alpha_1 p_1 - p_7)^2}.$$

3.1 The disease-free equilibrium point E_0

Therefore disease free equilibrium point is given as

$$E_0 = \left(s_1^*, 0, s_2^*, 0, 0\right) = \left(\frac{p_7 a_3}{\alpha_1 p_1 - p_7},\ 0,\ \frac{r_1 a_3 \alpha_1 \left(\alpha_1 p_1 - p_7 - p_7 a_3\right)}{\left(\alpha_1 p_1 - p_7\right)^2},\ 0,\ 0 \right) \tag{7}$$

The equilibrium point E_0 exists if and only if (i) $\alpha_1 p_1 \neq p_7$ and (ii) $\alpha_1 p_1 \neq \left(1 + a_3\right) p_7$ also E_0 will be positive if and only if (i) $\alpha_1 p_1 > p_7$ and (ii) $\alpha_1 p_1 > \left(1 + a_3\right) p_7$

where, $p_7 = \frac{\mu_2}{k \beta_1}$ and α_1 is the rate of conversion of the susceptible chicken (prey) into a human (predator), b_1 is the predation rate of the susceptible chicken, k is the carrying capacity of chickens population, a_1 is the half saturation of susceptible chicken and μ_2 as the natural mortality rate of human (predator).

3.2 Endemic equilibrium point E_1

Endemic equilibrium point E_1 is obtained by solving equations (7-11) and the results are summarised as follows

$$s_1^* = \frac{x_1 l_1^{*3} + x_2 l_1^{*2} + x_3 l_1^* + x_4}{p_8 p_{10} p_6 l_1^{*3} - x_5 l_1^{*2} - x_6 l_1^* + x_7} \ ,\ s_2^* = \frac{\left(x_8 {l_1}^4 + x_9 {l_1}^3 - x_{10} {l_1}^2 + x_{11} l_1 + x_{12}\right)\left(n_3 + l_1\right)}{\left(p_8 p_{10} p_6 {l_1}^3 - x_5 {l_1}^2 - x_6 l_1 + x_7\right)\left(n_4 + l_1\right) p_4}$$

$$l_2^* = \frac{p_6 s_2^* l_1^* \left(a_4 + s_1^*\right)\left(n_4 + l_1^*\right)}{p_9 a_4 n_4 + p_9 a_4 l_1^* + p_9 s_1^* n_4 + p_9 s_1^* l_1^* - \alpha_3 p_2 s_1^* n_4 - \alpha_3 p_2 s_1^* l_1^* - \alpha_4 p_5 l_1^* a_4 - \alpha_4 p_5 l_1^* s_1^*}$$

$$r_2 = \frac{p_{10} l_2^*}{p_{11}},$$

where $x_1 = p_8 p_{10} p_6 a_4$, $x_2 = p_6 p_{11} a_3 + p_8 p_{10} p_6 a_4 n_4 + p_8 p_{10} p_6 a_4 n_3$,

$x_3 = -\alpha_2 p_4 p_{11} a_3 + p_6 p_{11} a_3 n_3 + p_7 p_{11} a_3 + p_8 p_{10} p_6 a_4 n_4 n_3$,

$x_4 = p_7 p_{11} a_3 n_3$ $x_5 = p_6 p_{11} + p_8 p_{10} p_6 n_4 + p_8 p_{10} p_6 n_3$, $x_{12} = \left(p_3 n_4 + p_5 l_2\right) x_7 - p_7 p_{11} a_3 n_3 n_4$

$x_6 = \alpha_2 p_4 p_{11} - p_6 p_{11} n_3 + \alpha_1 p_1 p_{11} - p_7 p_{11} - p_8 p_{10} p_6 n_4 n_3$

$x_7 = p_7 p_{11} n_3 - \alpha_1 p_1 p_{11} n_3$, $x_8 = p_8 p_{10} p_6 \left(p_3 - a_4\right)$,

$x_9 = \left(p_3 n_4 - a_4 n_3 + p_5 l_2 - 2 a_4 n_4\right) p_6 p_{10} p_8 - p_6 p_{11} a_3 - p_3 x_5$

$x_{10} = \left(p_3 n_4 + p_5 l_2^*\right) x_5 + \left(-\alpha_2 p_4 a_3 + \left(-a_3 n_4 + a_3 n_3\right) p_6 + p_7 a_3\right) p_{11} + p_3 x_6 +$

$\left(-\left(a_4 n_4 + a_4 n_3\right) n_4 + a_4 n_4 n_3\right) p_6 p_8 p_{10}$

$x_{11} = \left(x_7 - x_6 n_4\right) p_3 + \left(\alpha_2 p_4 - p_6 n_3 - p_7\right) a_3 p_{11} n_4 - p_{11} p_7 a_3 n_3 - p_8 p_{10} p_6 a_4 {n_4}^2 n_3 - p_5 l_2^* x_6$

l_1^* is the positive root of the equation

$$r_1 \left(1 - s_1^*\right) s_1^* - s_1^* l_1 - \frac{p_1 s_1^* s_2^*}{a_3 + s_1^*} - \frac{p_2 s_1^* l_2^*}{a_4 + s_1^*} = 0 \tag{8}$$

Therefore the endemic equilibrium point is $E_1 = \left(s_1^*,\ l_1^*,\ s_2^*,\ l_2^*,\ r_2^*\right)$.

Reproduction number and Stability analysis of the model (2)

4. 0 Reproduction number (R_0)

Dynamical system (2) has two infected classes that are infected chickens (l_1) and infected human (l_2), that we use to determine the reproduction number. Using next generation approach we obtain the epidemiological threshold for the system (2) which is the dominant eigenvalues of

$$R = \left[\frac{\partial F_i\left(E_0\right)}{\partial X_j}\right].\left[\frac{\partial V_i\left(E_0\right)}{\partial X_j}\right]^{-1}, \tag{9}$$

where $\mathbf{F}_i$ is the appearance of new infections and V_i is the transfer of individual into the compartment. At E_0 (disease free equilibrium point) of the model, we obtained the reproduction number R_0 as

$$R_0 = \frac{n_3 s_1^*}{p_3 n_3 + p_4 s_2^*}. \tag{10}$$

where $s_1^* = \dfrac{p_7 a_3}{\alpha_1 p_1 - p_7}$ in terms of the original parameter, we expressed R_0 as

$$R_0 = \frac{k\left(\alpha_1 b_1 - \mu_2\right) a_1 \mu_2 n_1 \beta_1}{\left\{\left(\alpha_1^{\,2} b_1^{\,2} + \mu_2^{\,2}\right)\left(\mu_1 + m\right) k n_1 + c_1 r a_1 \alpha_1^{\,2} k b_1\right\} - \left\{\left(m + \mu_1\right) 2\mu_2 n_1 \alpha_1 b_1 k + \left(k + a_1\right)\alpha_1 a_1 \mu_2 c_1 r\right\}} \tag{11}$$

Note: *The disease-free equilibrium point E is locally asymptotically stable if $R_0 < 1$ otherwise, it is unstable.*

4.1 Local stability of analysis

The local stability analysis of the model (2) is carried out using variation matrix of Jacobian to understand the dynamical behaviour and the nature of equilibrium points of the model.

4.1.1 Local stability of analysis of disease-free equilibrium point E_0

We study the local stability of E_0 through the following variation matrix

$$J = \begin{pmatrix} -2r_1 s_1 + r_1 - A_{11} & -s_1 & -A_{13} & -A_{14} & 0 \\ 0 & s_1 - p_3 - \dfrac{p_4 s_2}{n_3} & 0 & 0 & 0 \\ A_{31} & A_{32} - p_6 s_2 & A33 - p_7 & 0 & p_8 \\ 0 & p_6 s_2 & 0 & -p_9 + A_{34} & 0 \\ 0 & 0 & 0 & p_{10} & -p_{11} \end{pmatrix} \tag{12}$$

The variation matrix (12) at E_0 gives the following eigenvalues

$$\lambda_1 = -\frac{-s_1^* n_3 + p_3 n_3 + p_4 s_2^*}{n_3},\ \lambda_2 = -p_9 + A_{34},\ \lambda_3 = -p_{11}$$

$$\lambda_{4,5} = -r_1 s_1 + \frac{1}{2} r_1 - \frac{1}{2} A_{11} + \frac{1}{2} A_{33} - \frac{1}{2} p_7 \pm$$
$$\frac{1}{2}\sqrt{\left(\begin{array}{l} 4r_1{}^2 s_1{}^2 - 4r_1{}^2 s_1 + r_1{}^2 - 2r_1 A_{11} + A_{11}^2 + 4r_1 s_1 A_{11} + 4r_1 s_1 A_{33} - 4r_1 s_1 p_7 + A_{33}^2 - \\ 2A_{33} p_7 + p_7{}^2 - 2r_1 A_{33} + 2r_1 p_7 + 2A_{11} A_{33} - 2A_{11} p_7 - 4A_{31} A_{13} \end{array}\right)}$$

where

$$A_{11} = \frac{p_1 s_2 a_3}{\left(a_3 + s_1\right)^2}, \quad A_{13} = \frac{p_1 s_1}{a_3 + s_1}, \quad A_{14} = \frac{p_2 s_1}{a_4 + s_1}, \quad A_{31} = \frac{\alpha_1 p_1 s_2 a_3}{\left(a_3 + s_1\right)^2}, \quad A_{32} = \frac{\alpha_2 p_4 s_2}{n_3},$$

$$A_{33} = \frac{\alpha_1 p_1 s_1}{a_3 + s_1}, \; A_{34} = \frac{\alpha_3 p_2 s_1}{a_4 + s_1}$$

Therefore the E_0 is locally asymptotically stable whenever $\lambda_i < 0$ otherwise it is unstable.

4.1.2 Local stability of endemic equilibrium point E_1

The linearization of endemic equilibrium point E_1 of the model gives the following polynomial equation

$$\lambda^5 + A\lambda^4 + B\lambda^3 + C\lambda^2 + D\lambda + E = 0 \tag{13}$$

E_1 is locally asymptotically stable if equation (20) obeys the following Hurwitz criteria

$$A > 0$$
$$AB > C$$
$$A(BC + E) > A^2 D + C$$
$$(CD - BE)(AB - C) > (AD - E)^2$$
$$D[(CD - BE)(AB - C) - (AD - E)^2] > 0$$

where

$$A = p_{11} - B_{44} - B_{33} - B_{22} - B_{11}$$
$$B = -p_{11}B_{44} - p_{11}B_{33} - p_{11}B_{22} - p_{11}B_{11} + B_{41}B_{14} + B_{24}B_{42} + B_{44}B_{33} + B_{44}B_{22} + B_{44}B_{11} + B_{31}B_{13} + B_{23}B_{32} + B_{33}B_{22} + B_{33}B_{11} + l_1^* s_1^* + B_{22}B_{11}$$
$$C = -B_{33}l_1^* s_1^* + p_6 l_1^* B_{31}B_{14} + p_6 l_1^* B_{24}B_{32} - B_{44}l_1^* s_1^* + p_{11}l_1^* s_1^* - B_{31}s_1^* B_{23} + B_{32}B_{13}l_1^* - B_{31}B_{13}B_{22} - B_{23}B_{32}B_{11} - B_{33}B_{22}B_{11} + p_{11}B_{44}B_{11}$$
$$D = B_{44}B_{33}l_1^* s_1^* - B_{41}B_{13}B_{24}B_{32} - B_{42}B_{23}B_{31}B_{14} - B_{42}B_{14}l_1^* B_{33} + p_{10}p_8 B_{23}B_{42} + p_6 l_1^{*2} B_{32}B_{14} - p_6 l_1^* B_{31}s_1^* B_{24} - p_6 l_1^* B_{31}B_{14}B_{22} - p_6 l_1^* B_{24}B_{32}B_{11}$$
$$E = -p_{11}B_{41}B_{13}B_{24}B_{32} - p_{11}B_{42}B_{23}B_{31}B_{14} + p_{11}B_{41}s_1^* B_{24}B_{33} + p_{11}B_{41}B_{14}B_{33}B_{22} + p_{11}B_{24}B_{42}B_{31}B_{13} - p_{10}p_8 B_{41}B_{13}B_{22} - p_{10}p_6 l_1^{*2} p_8 s_1^* + p_{11}B_{44}B_{33}l_1^* s_1^*$$

$$B_{11} = -2r_1 s_1^* + r_1 - l_1^* - \frac{p_1 s_2^* a_3}{\left(a_3 + s_1^*\right)^2} - \frac{p_2 l_2^* a_4}{\left(a_4 + s_1^*\right)^2}, \; B_{13} = \frac{p_1 s_1^*}{a_3 + s_1^*}, \; B_{14} = \frac{p_2 s_1^*}{a_4 + s_1^*},$$

$$B_{32} = \frac{\alpha_2 p_4 s_2^* n_3}{\left(n_3 + l_1^*\right)^2} - p_6 s_2^*.$$

4.2 Global stability of DFE

In this section, we analyse the global behaviour of the disease-free equilibrium point for the system (1) by using the concept of Lyapunov function. We choose the Lyapunov function by following the approach of Massawe et al. (2015).

Theorem 5.2: *The DFE of the model (1) is globally asymptotically stable if* $\frac{dL}{dt} < 0$ *in the interior of* Γ.

Proof:

In order to prove that the DFE is globally asymptotically stable, we construct the Lyapunov function for our model as

$$L(t) = Al_1(t) + Bl_2(t) \tag{14}$$

Differentiating (14) and substituting the model equations (2), we get

$$\begin{aligned}\frac{dL}{dt} &= A\left\{(1-l_1) - p_3 - \frac{p_4(1-l_2)}{n_3+l_1} - \frac{p_5 l_2}{n_4+l_1}\right\} l_1 \\ &+ B\left\{p_6(1-l_2)l_1 - p_9 l_2 + \frac{\alpha_3 p_2 (1-l_1) l_2}{a_4+(1-l_1)} + \frac{\alpha_4 p_5 l_1 l_2}{n_4+l_1}\right\}\end{aligned} \tag{15}$$

where $s_1 = 1-l_1$ and $s_2 = 1-l_2$ (16)

Properly choosing $A, B > 0$, we have

$$\frac{dL}{dt} = P - Q \tag{17}$$

where

$$P = Al_1 + \frac{p_4}{n_3+l_1} + B\left\{p_6 l_1 + \frac{\alpha_3 p_2 l_2}{a_4+(1-l_1)} + \frac{\alpha_4 p_5 l_1 l_2}{n_4+l_1}\right\}$$

$$Q = A\left\{l_1 + p_3 + \frac{p_4 l_2}{n_3+l_1} + \frac{p_5 l_2}{n_4+l_1}\right\} l_1 + B\left\{p_6 l_2 l_1 + p_9 l_2 + \frac{\alpha_3 p_2 l_2 l_1}{a_4+(1-l_1)}\right\}$$

If $P < Q$ then $\frac{dL}{dt}$ is negative and $\frac{dL}{dt} = 0$ if and only if $l_1 = l_2 = 0$ at which will ultimately reduce the Newcastle disease in poultry. Hence, by LaSalle's invariance principle it implies that E is globally asymptotically stable in Γ.

5.1 Data and Parameter Estimation

The Central zone of Tanzania comprises of Singida and Dodoma regions and is categorised as a semi-arid zone with savannah climate features. The study was conducted in two regions, Dodoma and Singida in the central zone of Tanzania with five districts, namely Ikungi, Singida Rural, Mkalama districts in Singida region and Chamwino and Kongwa districts in Dodoma region as shown in Figure 2. The choice of these two regions was attributed to many factors, including a significant number of chicken keepers, the frequent of ND outbreak, husbandry skills and knowledge. The collected Data are summarised in Table 1.

5.2 Model Fitting and Parameter Estimation

Among the most important aspects in modelling includes model fitting and parameter estimation. We use the data for 2015 ND death cases to estimate parameters and fit our model since numerical simulations need to use parameter values that reflect ecological implications of each embedded parameter (Myung 2003). The idea of estimation starts with sum of squares of residual (SSR) and is given as

$$L(\theta) = \sum_{i=1}^{N} \left(y_i - y_i^{est} \right)^2 \tag{18}$$

where $\{y_i\}^N_{i=1}$ is the real data and $\{y_i^{est}\}^N_{i=1}$ is the solution of the model. The numerical results for parameter estimation are summarised in Table 2 and the fitting result is presented in Fig. 3.

Figure 3 shows the fitting of the model to the ND infected chicken death cases data. The trend moves close to the data but still not very sure to what extent these model parameters are correctly estimated (Tchuenche *et al.* 2011). Therefore, the Markov Chain Monte Carlo (MCMC) method is employed with 1000 as initial runs in maximum likelihood. The results are then used as prior distribution to re-run the model with 10000 numbers of simulations with convergence criteria of 10^{-8} in MCMC simulations. The numerical results are presented in Table 2.

5.3 Markov Chain Monte Carlo Results

Table 2 presents the numerical results of parameter estimation of the model (1) which was carried out using delayed rejection adaptive Markov Chain Monte Carlo (DRAM) method. The numerical value of each parameter that is generated in MCMC plot depicts the convergence of the chain. The correctness and goodness of the performance of MCMC results are analysed using scatter, autocorrelation, trace, and marginal distribution plots. Figure 4 shows the generated samples for the parameters which show good mixing compared to other parameters. It is therefore observed that the poor mixing of samples can be corrected and updated as depicted in Figure 5. The fitted model parameters oscillate within their confidence interval and show a positive and strong correlation as indicated in Figure 5. The convergence of MCMC sample parameters is therefore graphically visualised by plotting autocorrelation figures as shown in Figure 6. The parameter convergence oscillates within their confidence interval and finally stabilises at around zero with 220 lags as per Figure 6. The sample marginal distribution plots with mean and original parameters are depicted in Figure 7. The observations of marginal distribution show that most of the parameters skew to the right such as b_2, θ, m, α_2 and b_1 in respect to other parameters.

6. Numerical Sensitivity Analysis

The sensitivity analysis of ND model is carried out to determine the marginal changes in each parameter involved in reproduction number R_0. This helps to identify the most sensitive parameter that leads to greater impact in the prevalence of the infection.

Let R_0 be the function that depends on q_i, then

$$X_{R_0}^{q_i} = \frac{\partial R_0}{\partial q_i} \cdot \frac{q_i}{R_0} \tag{19}$$

where q_i is the model parameter that index is obtained. The numerical result summarised in Table 3.

From the Table 3, it is observed that the parameters μ_2, β_1, r and a_1 with positive index increase the endemicity of the disease as its values increases while keeping another parameter constant. The parameter with a negative index such as α_1, c_1, n_1, b_1, m, μ_1 and k reduces the endemicity as its value increases. Particularly, the convection rate of chicken to human α_1 and μ_2 death rate of human are the most sensitive parameters. This implies that an increase or decrease in either parameter will have an increasing or decreasing effect on the prevalence of the Newcastle disease in Tanzania. The force of infection or contact rate of susceptible and infected chickens β_1 also has great impact prevalence of the disease compared to others parameter as shown in Table 3.

7. Numerical simulations of the model

In order to visualise the interactions among chickens and human populations, we present numerical simulations of the model (1) using MATLAB R2013b version with a set of parameter values estimated using maximum likelihood estimation given in Table 2. The results of the computer simulation and plots are as follows:

The Fig. 8 shows a variation of the population with time. The susceptible and infected chicken oscillates with low amplitude and finally attains its equilibrium steady state about zero (see Figure 8 (a-b). Susceptible human population takes more time to converge compared to others as shown in Figure 8(c) while infected human Figure 8(d) increases at the beginning and exponentially decay toward steady states. Figure 9 shows the variation among the pair of populations, from the Figure 9(a) we observe positive correlation among susceptible human and susceptible chickens' populations. The interaction between susceptible and infected chickens shows negative correlation; the susceptible chicken decreases as an impact of a disease that ultimately increases infected chickens and finally decreases with low oscillations about zero.

From the Figure 9(c) the infected chicken's increases at the beginning, since the infected chicken populations do not affect human then tends to increase it is population unlike in Figure 9 (d) where the infected chickens will all die out and infected human will all recover through treatment. Figure 10 describes the phase portrait of the model with alternating susceptible and infected human population. From Figure 10(a) we observe that susceptible classes increase as a result of a decrease of infected chicken while Figure 10(b) indicates that infected human and chicken population decreases with an increase of susceptible chicken. Note that there is no spread of ND from an infected human to susceptible chickens; this proves that the human being can not spread the ND to any other species as reported by Alexander et al (Alexander 2000).

Figure 11 (a-b) shows the relationship between susceptible and infected classes, the variation indicates that a large proportion of population fall in the phase of susceptible classes which indicates that all infected human will recover while infected chicken will die out.

8. Conclusions

ND eco-epidemiological model has been formulated and analysed both qualitatively and quantitatively to understanding the mechanism responsible for the spread of the disease in Tanzania. The maximum likelihood method for parameter estimation was successfully used to estimate the parameter values for the model (1) as shown in Table 2. The model was fitted to 2015 data as shown in Fig. 4 and the fitting shows a good trend. Numerical simulations were carried out using the estimated values generated by maximum likelihood method it results shows that the disease can be controlled through vaccination. The sensitivity analysis shows that prey convectional rate to a predator α_1 is the most sensitive parameter in our model (see Table 3). In the numerical simulation, it is observed that the increase of vaccination awareness among the farmers tends to increase chicken productivity in Tanzania (see Figure 10). A strategy like education campaign, particularly in remote and rural areas in Tanzania, plays a big role in eliminating the disease and basically, it helps to improve the life hood of farmers or families or individual that relies on poultry as shown in Figure 11. In order to eliminate the disease in Tanzania, every farmer must apply chicken vaccination in time and take care of all bio-security measures and hence, the human population can highly benefit from poultry in terms of collecting their eggs, or for meat and/or feathers (Solomon 2011).

9. References

Alexander, D. J., 2000. Newcastle Disease and Other Avian Paramyxoviruses. *Revue Scientifique et Technique (International Office of Epizootics)* 19 (2): 443–62.

Alexander, D. J., Bell, J. G., Alders, R. G., 2004. FAO Technology Review: Newcastle Disease with Special Emphasis on Its Effect on Village Chickens. *FAO Animal Production and Health* 4 (ISSN 0254-6019): 55.

Arifin, M. A ., Siti H. S., Maizirwan M., Mohamed I., Abdul K., Sharifah S. H., 2011. Optimisation of Newcastle Disease Virus Production in T-Flask. In *Internal Conference on Biotechnology Engineering*, 465–69. Kuala Lumpur, Malaysia.

Bornaa, C. S., Makinde, O. D., Ibrahim Y. S., 2015. Eco-Epidemiology Model and Optimal Control of Disease Transmission between Humans and Animals. *Commun. Math. Biol. Neurosci.*, no. ISSN:2052-2541: 1–28.

Chuwa, Albina. 2012. "United Republic of Tanzania National Sample Census of Agriculture." *National Bureau of Statistics*. Vol. III. Ministry of Livestock Development and Fisheries, Dar es Salaam. www.nbs.org.

Dortmans, J.C., Guus K ., Peter J. R ., Ben P. P., 2011. Virulence of Newcastle Disease Virus: What Is Known so Far? *Journal of Veterinary Research* 42 (1). BioMed Central Ltd: 122. doi:10.1186/1297-9716-42-122.

Gardner, E. G., 2014. Livestock Risks and Opportunities: Newcastle Disease and Avian Influenza in Africa. *Planet@ Risk* 2 (4): 208–11.

Glaser, A., 2007. Dynamics and Control of Infectious Diseases. *ReVision*, 1–29.

Hugo, A., Massawe E.S., Makinde O.D., 2012. An Eco-Epidemiological Mathematical Model with Treatment and Disease Infection in Both Prey and Predator Population. *Journal of Ecology and Natural Environment* 4: 266–79. doi:10.5897/JENE12.013.

Ibrahim, K., Silvio, G., Hussein, K., Athumani, Z., 2012. Transforming Traditional Poultry Keeping into a Commercial Venture The Experience of RLDC with the Comprehensive Poultry Rearing Model. *Rural Livelihood Development Company*.

Knueppel, D ., Peter C ., Ayubu O. M., Peter M., David M., Carol C., (2009). Improving Poultry Production for Sustainability in the Ruaha Landscape, Tanzania. *WildLife Conservation Society*, 24.

Lungu, E., Massaro, T. J., Ndelwa, E., Ainea, N., Chibaya, S., Malunguza , N J., (2013). Mathematical Modelling of the HIV/Kaposi's Sarcoma Coinfection Dynamics in Areas of High HIV Prevalence. *Computational and Mathematical Methods in Medicine* 2013: 1–19. doi:10.1155/2013/753424.

Yongolo, M.G., Maeda , A.M ., Minga, U.M., (2009). Newcastle Disease and Infectious Bursal Disease among Free Range Village Chickens in Tanzania. *The Onderstepoort Journal of Veterinary Research*, 332–35. doi:10.13748/j.cnki.issn1007-7693.2014.04.012.

Makinde, O.D., Okosun, K.O., (2011). The impact of Chemo-Therapy on Optimal Control of Malaria Disease with Infected Immigrants. *Biosystems* 104 (1). Elsevier Ireland Ltd: 32–41. doi:10.1016/j.biosystems.2010.12.010.

Massawe, L N., Massawe E. S., Makinde, O. D., (2015). Temporal Model for Dengue Disease with Treatment. *Advances in Infectious Diseases* 05 (March): 21–36. doi:10.4236/aid.2015.51003.

MLFD. (2010).Livestock Sector Development Strategy.*MLFD* http://www.tanzania.go.tz/egov_uploads/documents/development_strategy-_Livestock_sw.pdf.

Mukhopadhyay, B., Bhattacharyya, R.., (2009). The role of Predator Switching in an Eco-Epidemiological Model with Disease in the Prey. *Ecological Modelling* 220 (7): 931–39. doi:10.1016/j.ecolmodel.2009.01.016.

Musako, C., Celia A., (2012). Determination of the Seroprevalence of Newcastle Disease Virus (Avian Paramyxovirus Type 1) in Zambian Backyard Chicken Flocks. *The Onderstepoort Journal of Veterinary Research* 79 (1): E1–4. doi:10.4102/ojvr.v79i1.502.

Muturi, J. M ., Ricky G. I., Jane W., Elias, G., (2013). Protection Levels of New Castle Disease Vaccine in Indigenous Poultry in a Semi-Arid Region of Meru County of Kenya." *Journal Biological and Food Science Research* 2 (2): 17–21.

Myung, I. J, (2003). Tutorial on Maximum Likelihood Estimation." *Journal of Mathematical Psychology* 47 (1): 90–100. doi:10.1016/S0022-2496(02) 00028-7.

Nabila, O, Serageldeen S., Ahmed I A., Ragab S. I., Mahmoud S., (2014). Isolation and Pathotyping of Newcastle Disease Viruses from Field Outbreaks among Chickens in the Southern Part of Egypt 2011-2012. *Global Veterinaria* 12 (2): 237–43. doi:10.5829/idosi.gv.2014.12.02.82104.

OIE. (2015).Newcastle Disease: General Inf. Disease Sheets. *http://www.oie.int/fileadmin/Home/eng/Media_Center/docs/pdf/Disease_cards/NEWCAS-EN.pdf. 2015*, 1–6. www.0ie.int/international - standard-setting/terrestrial-code. Accessed on 20 January, 2016.

Sharma, S., Samanta, G. P., (2015). Stability Analysis and Optimal Control of an Epidemic Model with Vaccination. *International Journal of Biomathematics* 8 (3). doi:10.1142/S1793524515500308.

Solomon, P (2011). Molecular Characterization of Newcastle Disease Viruses from Live Bird Markets in Nigeria. *The University of Pretoria*.

Van Boven., M, Annemarie B., Teun H. F., Elly K., Leo H., Guus K., (2008). Herd Immunity to Newcastle Disease Virus in Poultry by Vaccination. *Avian Pathology : Journal of the W.V.P.A* 37 (1): 1–5. doi:10.1080/03079450701772391.

Yongolo, M. G., Henrik C., Kurt H., Uswege M., John E. O., (2011). On the Origin and Diversity of Newcastle Disease Virus in Tanzania. *The Onderstepoort Journal of Veterinary Research* 78 (1): 312. doi:10.4102/ojvr.v78i1.312.

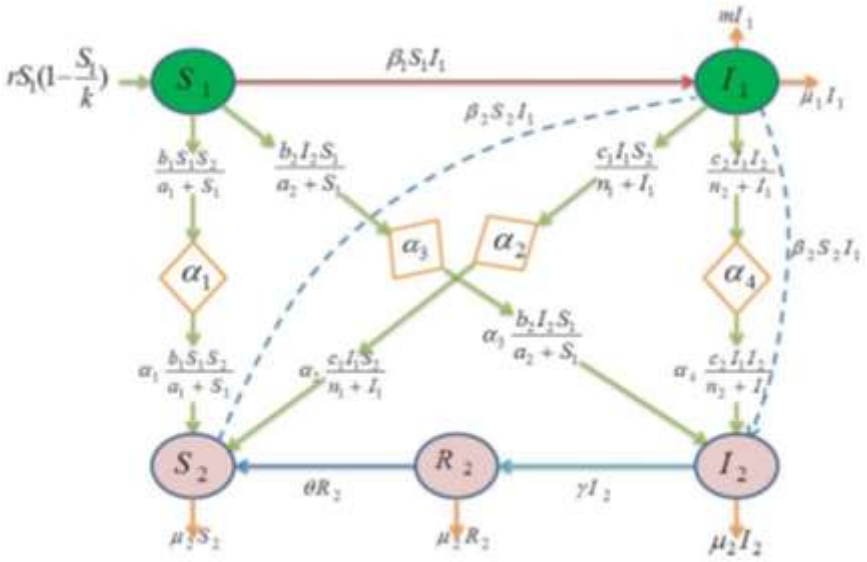


Figure 1: Model Flow diagram showing how the populations interact

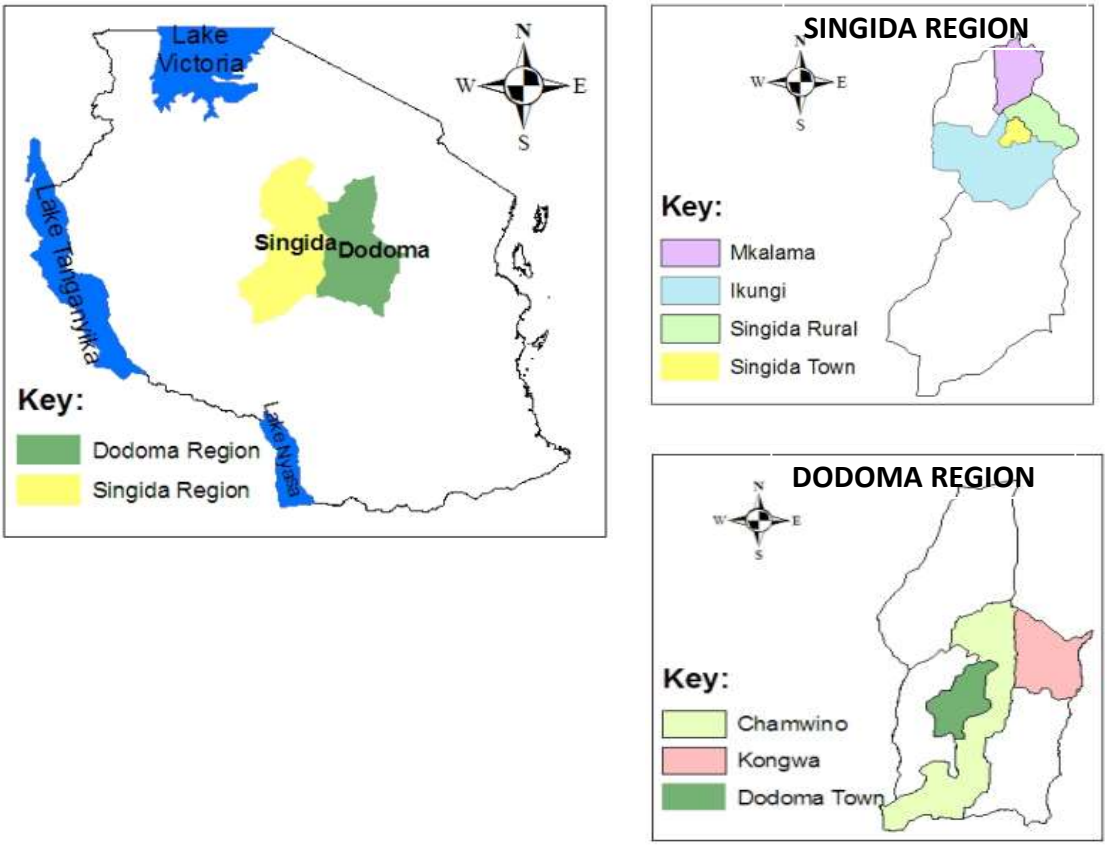


Figure 2: Tanzania map showing the location of districts and regions of study area

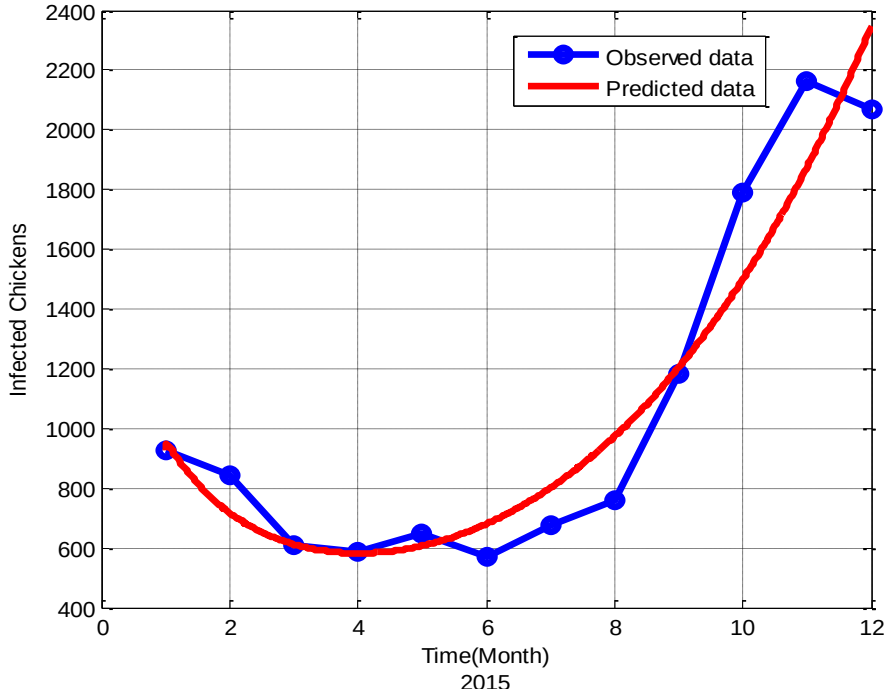


Figure 3: Mode fitting for five districts in Tanzania data of chicken death cases for 2015

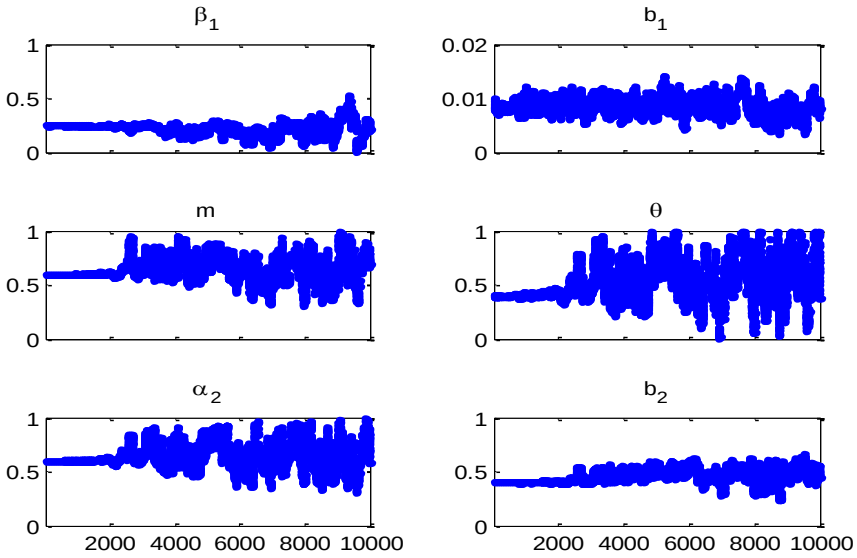


Figure 4: Markov Chain Monte Carlo (MCMC) Scatter Plots. The vertical axis represents samples and the horizontal axis represents a number of iterations.

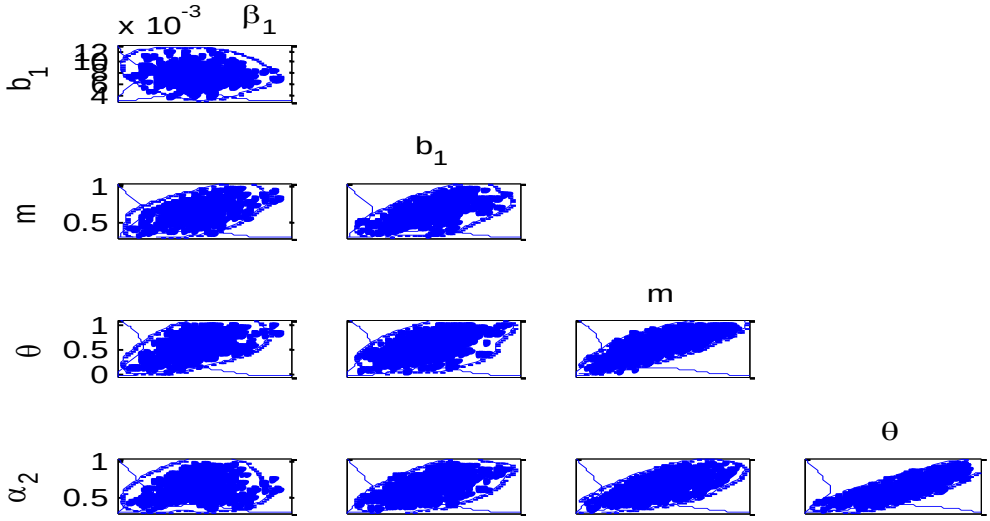


Figure 5: A plot showing pair correlation of parameters in confidence interval

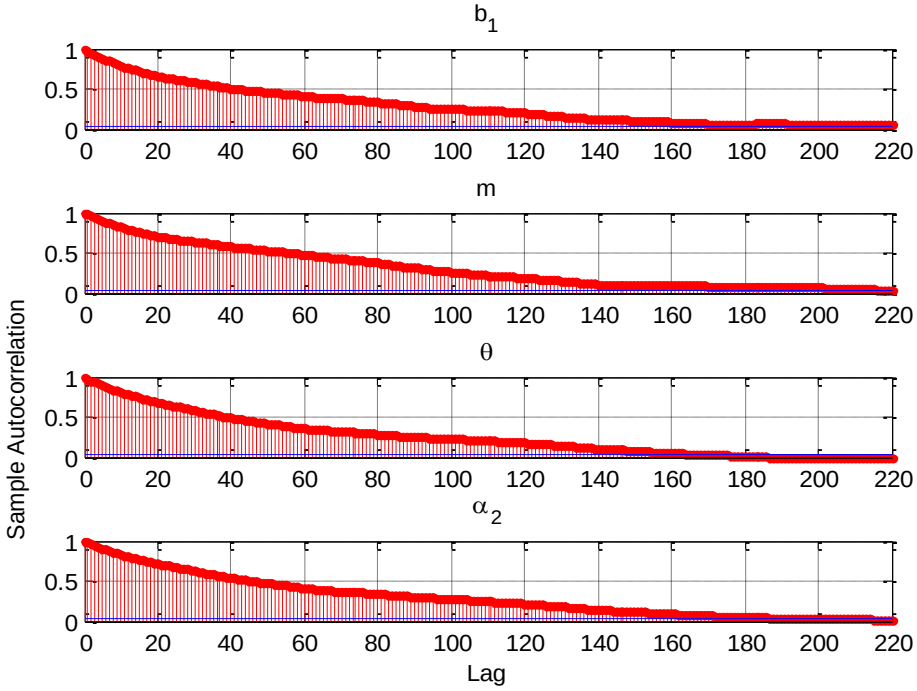


Figure 6: MCMC trace plots. The vertical axis represents samples while the horizontal axis represents a number of iterations.

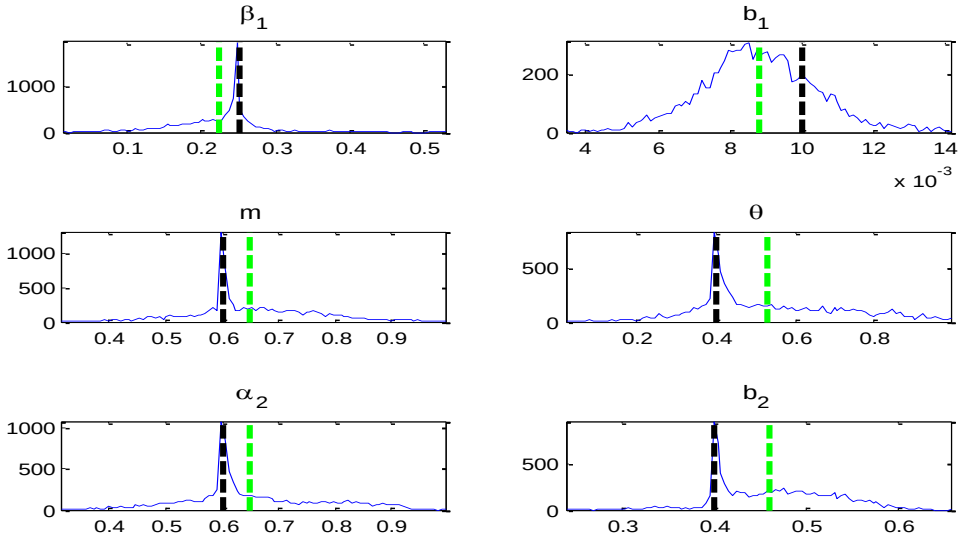


Figure 7: Marginal distribution plot and a mean of MCMC samples. The green colour represents sample meanwhile black represents the original parameter value.

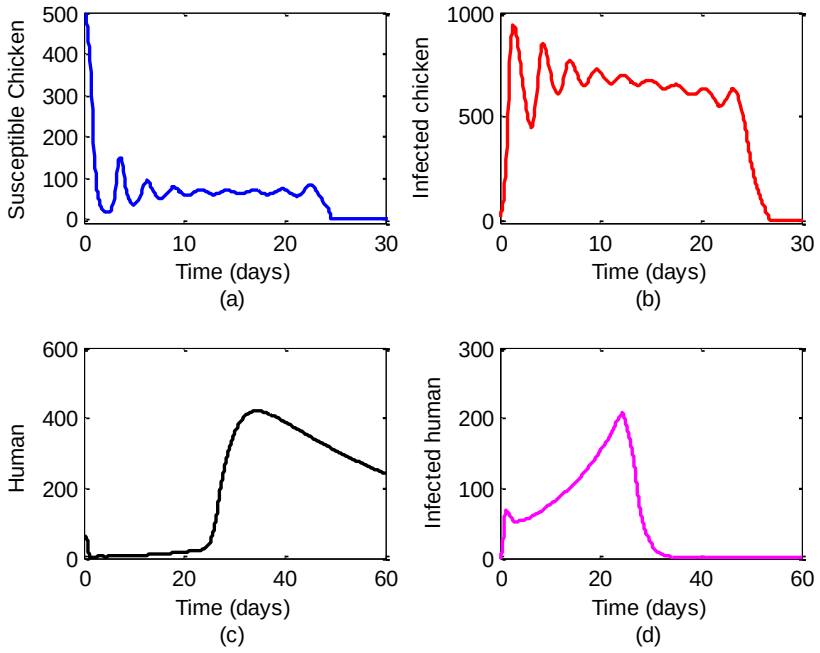


Fig. 8: Variation of the populations with time, parameter values in Table 2

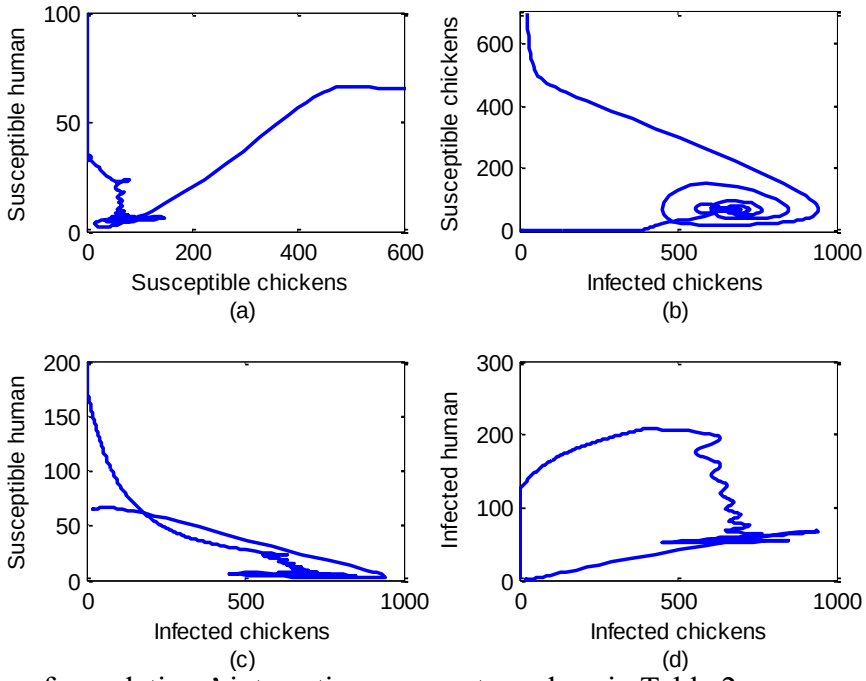


Fig. 9: Variation of populations' interaction, parameter values in Table 2

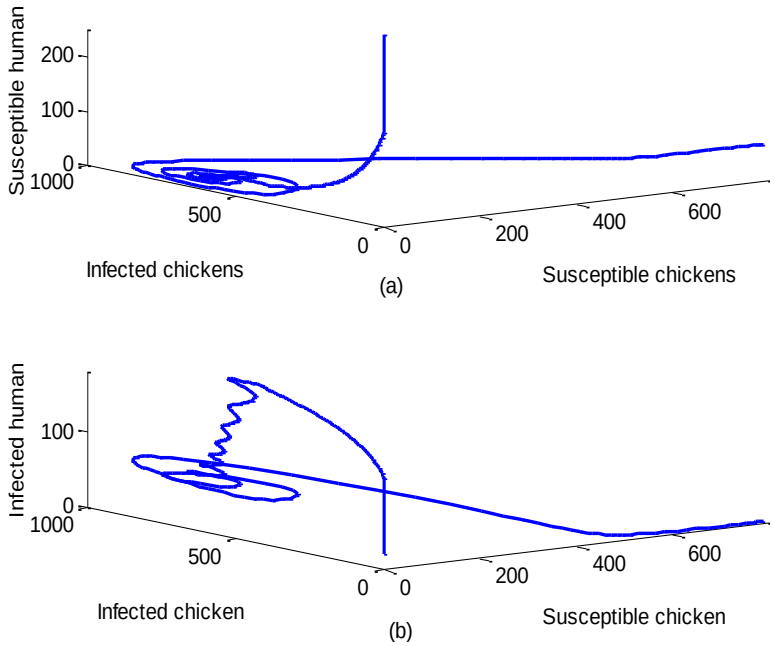


Figure 10: Interactions of the prey and predator population, parameter values in Table 2

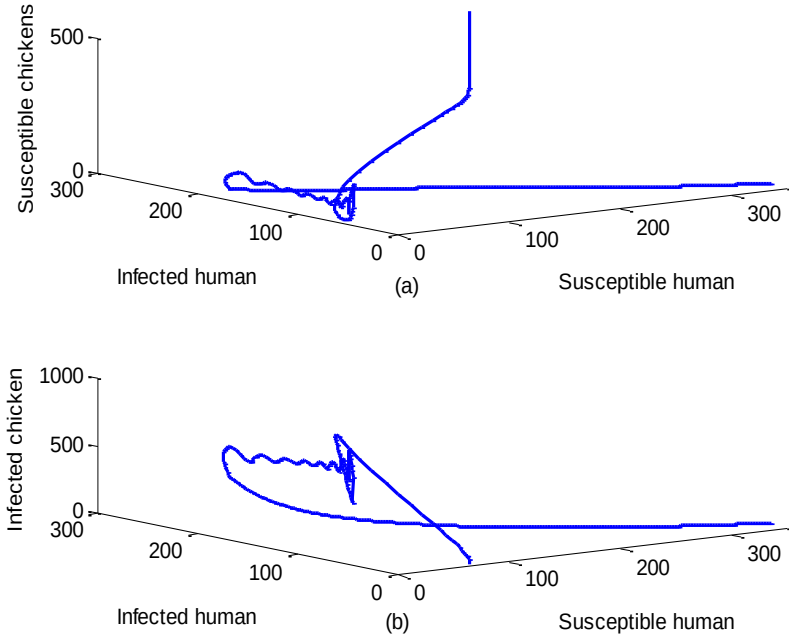


Figure 11: Interactions among human and infected chickens, parameter values in Table 2

Table 1: Chicken monthly death cases due to ND data per district for 2015

Year	Month	Kongwa	Chamwino	Mkalama	Singida	Ikungi	Total
	Jan	207	104	117	349	150	927
	Feb	197	195	165	138	148	843
	March	104	124	142	126	115	611
	April	102	135	125	126	101	589
	May	100	126	127	145	151	649
	June	62	137	129	78	165	571
	July	126	184	134	102	130	676
	Aug	241	126	165	104	124	760
	Sept	205	245	217	257	261	1185
	Oct	248	203	214	768	355	1788
	Nov	368	258	268	810	462	2166
2015	Dec	396	291	295	657	432	2071
		2356	2128	2098	3660	2594	12836

Table 2: Literature and Estimate Parameters values

Symbol	Literature value	Source	Estimate value
$\alpha_i \; i=1,..,4$	0.25, 0.6, 0.8, 0.6	(Hugo et al. 2012), Estimated	0.3093, 0.6078, 0.8939, 0.6008
β_1	0.1	(Sharma 2015)	0.1495
k	500	(Mukhopadhyay 2009)	500
μ_2	0.01	(Bornaa et al. 2015)	0.0244
c_1	0.02	Estimated	0.0202
r	10	(Bornaa et al 2015)	10
$a_i \; i=1,2$	0.25, 0.8	Estimated	0.2481, 0.6306
$n_i \; i=1,2$	0.03, 0.5	Estimated,(Mukhopadhya 2009)	0.0304, 0.503
b_1	0.4	(Mukhopadhyay 2009)	0.4085
b_2	0.6	(Hugo et al. 2012)	0.6019
m	0.6	Estimated	0.5968
μ_1	0.02	(Bornaa et al. 2015)	0.02488
θ	0.4	Estimated	0.4048
γ	0.6	Estimated	0.612
β_2	0.012	Estimated	0.0119
c_2	0.05	Estimated	0.4974

Table 3: Sensitivity indexes of R_0

Symbol	α_1	μ_2	β_1, r	c_1, n_1	b_1	a_1	m	μ_1	k
Sensitivity Index	-1.006	1.006	*1*	-0.979	-0.03	0.03	- 0.02	-0.001	-0.0003