

MODELING THE ROLE OF WILD BIRDS AND ENVIRONMENT IN THE DYNAMICS OF NEWCASTLE DISEASE IN VILLAGE CHICKEN

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ABSTRACT. Newcastle disease is common viral poultry disease which leads to a massive killing of chicken if preventive measures are not well taken. In this paper, we develop and analyze a deterministic model to investigate the role of wild birds and environment on the transmission dynamics of Newcastle disease in village chicken. We compute the basic reproduction number ($\mathcal{R}_0$), a threshold that tells the presence of the disease in a population. Finally, we performed the sensitivity analysis of parameters to see their relationship with the basic reproduction number ($\mathcal{R}_0$). The numerical results show that the basic reproduction number ($\mathcal{R}_0$) is more sensitive to the contact rate between the susceptible village chicken, wild birds and contaminated environment. This implies that, more contamination of the Newcastle virus into the environment increase the chance for the repeatedly occurrence of the disease. The results also shows that increasing the clearance rate of Newcastle disease virus in the environment reduces the rate of spread of the disease in chicken population. Therefore, contaminated environment plays a crucial role in the transmission of Newcastle diseases in the village chicken population.

1. INTRODUCTION

Newcastle disease (*ND*) is a viral disease that affects poultry and other wild birds [3, 10]. This disease is a major constraint to the development of village chicken industry particularly in Africa and Asia since it causes a high morbidity and mortality rate of chicken [3, 13, 24, 28]. The Newcastle disease is caused by Avian paramyxovirus-1 (APMV serotype-1) virus in paramoxyviridae family [13, 22, 27]. Depending on the severity of the disease during the

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outbreak, NDV is characterized as lentogenic, mesogenic or velogenic strains [4, 27]. Velogenic strains cause acute disease with high mortality of up to 100% for un-vaccinated village chicken compared to other pathotypes [27].

The symptoms of ND includes paralyzed legs and wing of the affected avian, coughing, head twisting, watery and greenish diarrhea and other nervous symptoms that follow in one or two weeks since infections [2, 25]. The seriousness of the disease depends on the virulence of the infecting viruses, susceptibility of the host, other diseases in the flock, environmental influences, and the vaccination history of the chicken [3]. Young birds in a flock are extremely susceptible to disease where death rate is about 100% [16, 24].

Although the Newcastle disease is endemic and sometime epizootic in village poultry, many aspects of its epidemiology are not clearly understood[13]. According to [19], other domestic birds, carrier chicken, wild birds and the physical environment are thought to be the contributing factors in the cycling occurrence of Newcastle disease. Initially, the disease is transmitted when an infected chicken is introduced in the flock. Further contacts of the infected chicken with other un-affected chicken leads to the spread of the virus in the entire flock [2, 27, 10]. Inhalation and ingestion of the virus through water and feed are the primary means of the spread of the virus [24, 27]. The infected and carrier birds may also shed 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 birds feathers [4]. The contact of the susceptible birds with the contaminated water, food, droppings or discharges of the infected birds and other farm utensils can also lead to the spread of the virus [8].

Over the years, different scholars have formulated and developed mathematical models to gain insight on the transmission dynamics of Newcastle disease in the village chicken[20, 14, 6].

[20] extended the model for foot and mouth disease in Thailand [29] to model the dynamics of ND among the village chicken. The model assumed that village poultry are the reservoir of Newcastle disease virus for other poultry sector. The model revealed that, in order for the vaccination of ND to be effective it must be conducted frequently in a large population of the village chicken. [14] developed a deterministic compartmental eco-epidemiological model with optimal control of Newcastle disease (ND) in Tanzania. The study revealed that ND can be controlled if farmers vaccinate chicken properly in time. [6] developed two mathematical models to study the influence of illegal harvest and effects of age structure of the of wild winged-Parakeets on the dynamics of Newcastle Disease. The relationship between of Newcastle Disease transmission and harvest were evaluated through the basic reproductive number. The findings showed that much harvest reduce the spread of the ND in the Parakeets population. Using their second model they concluded that age of the Parakeets can influence the spread of ND though not to a great extent.

To the best of our knowledge no any mathematical model which tried to address the role of wild birds and environment in the transmission of Newcastle disease. Therefore, based

on different clinical and epidemiological studies conducted on the transmission of Newcastle disease in village chicken, we develop a mathematical model to investigate the role of the environment and wild birds in the transmission dynamics of Newcastle disease in village chicken.

2. MATERIAL AND METHODS

2.1. Formulation of the Model. We develop a compartmental model where chicken and wild birds are categorized in different compartments depending on the status of their infection. The Chicken population are modeled as $S_c E_c I_c$ (Susceptible-Exposed-Infected) while wild bird population are modeled as $S_b E_b I_b I_r$ (Susceptible-Exposed-Infected-Carrier). The environment has only one class of Newcastle disease virus (NDV). The model is formulated based on the idea that wild birds and the environment are the primary reservoirs of Newcastle disease virus [2, 10, 24, 19, 17]. The village chicken population $N_c(t)$ is divided into three subpopulations namely: $S_c(t)$ denoting the number of susceptible village chicken, $E_c(t)$ denoting the number of the exposed village chicken, and $I_c(t)$ denoting the number of infected village chicken. The Wild birds population $N_b(t)$ is divided into four compartments namely; $S_b(t)$ denoting the number of the susceptible wild birds, $E_b(t)$ denoting number of wild birds exposed to NDV, $I_b(t)$ denoting the number of chronically infected wild birds and $I_r(t)$ denoting the number of reservoir wild birds. The environment contaminated with NDV is denoted by $H(t)$. In the model, we assume a density dependent recruitment rate μN_c for the village chicken population through birth. Initially, village chicken acquires Newcastle disease virus when a diseased village chicken is introduced in a flock and come into contact with other un-affected chicken. Village chicken can also acquire Newcastle disease virus when exposed to unhygienic environment as well as when interacting with other carrier wild birds [17, 6]. Thus chicken acquire infections at a rate $\varphi_1(t, I_c, I_r, H)$ that depends on per capita contact rate of the infected village chicken with its susceptible population ($\psi I_c(t)$), per capita contact rate between the carrier wild birds and the susceptible village chicken ($b I_r(t)$) and the density of Newcastle disease virus in the environment $\left(\frac{dH(t)}{k+H(t)}\right)$. The infected chicken becomes infectious after surviving within the incubation period $(1/\gamma)$ of two to six days since infections [2]. After surviving the incubation period, chicken starts to show aerosol signs and progress to chronic stage of the disease at a rate $\gamma E_c(t)$. We assume that village chicken cannot recover from the infections but they die naturally at a rate μ and by the disease induced death rate δ_c .

Wild birds are assumed to be recruited at a density dependent rate μN_b through birth. Like the village chicken, the susceptible wild birds gets NDV from the contaminated environment as well as when they interact with the clinically infected and reservoir wild bird population at the rate $\varphi_2(t, I_b, I_r, H)$ which is a function of the interaction between the susceptible wild

birds and the clinically infected wild birds ($\phi I_b(t)$), the reservoir population of wild birds ($aI_r(t)$) and $\left(\frac{dH(t)}{k+H(t)}\right)$, where the parameter d is the contact rate of the hosts with the environment. The latently infected population of wild birds is considered as a reservoir of the virus[4]. This makes the infected population of wild birds to be divided into two sub-populations; chronically infected, $I_b(t)$ and carrier, $I_r(t)$, at the proportions of ρ and $(1 - \rho)$ respectively. It is assumed that wild birds cannot recover from the disease once infected but they are reduced by the natural death at the rate μ and by the disease induced death at the rate δ_b . The Carrier wild bird population does have disease induced mortality, its assumed that they only die naturally.

Newcastle disease virus is introduced in the environment through shedding by the clinically infected village chicken, the clinically infected wild birds and the carrier wild birds at the rates α_c and α_b respectively[4, 24]. The Newcastle disease virus can survival for some months at a temperature between $20 - 30^\circ C$ and much longer at cooler physical environments [19]. Parameters and state variables used in the formulation of the Newcastle disease model are summarized in the tables below;

TABLE 1. Descriptions of parameters used in the formulation of the model system 2.1

Parameter	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 bird population
$E_b(t)$	Exposed wild bird population
$I_b(t)$	Chronically Infected wild birds population
$I_r(t)$	Carrier wild bird population
$N_c(t)$	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 system 2.1

Parameter	Description
a	Transmission rate between carrier wild birds and the susceptible population of wild birds
b	Transmission rate between carrier wild birds and the susceptible village chicken
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
μ	Recruitment rate and Natural mortality death of the host populations
ϕ	Transmission rate between chronically infected wild birds and the susceptible wild birds
ψ	Transmission rate between chronically infected village chicken and the susceptible chicken
κ	Half saturation constant of Newcastle disease virus in the environment
d	The contact rate between the susceptible population of village chicken and wild birds with contaminated environment
ρ	Proportion of the exposed wild birds which become chronically infected with NDV
μ_v	Clearance rate of the NDV from the environment
δ_b	Disease induced death rate in wild birds
δ_c	Disease induced death rate in the village chicken

2.2. Model Flow diagram. Based on the transmission dynamics of the Newcastle disease, model assumptions, definition of variables and parameters respectively, the dynamics of the Newcastle disease is summarized in the flow diagram as follows:

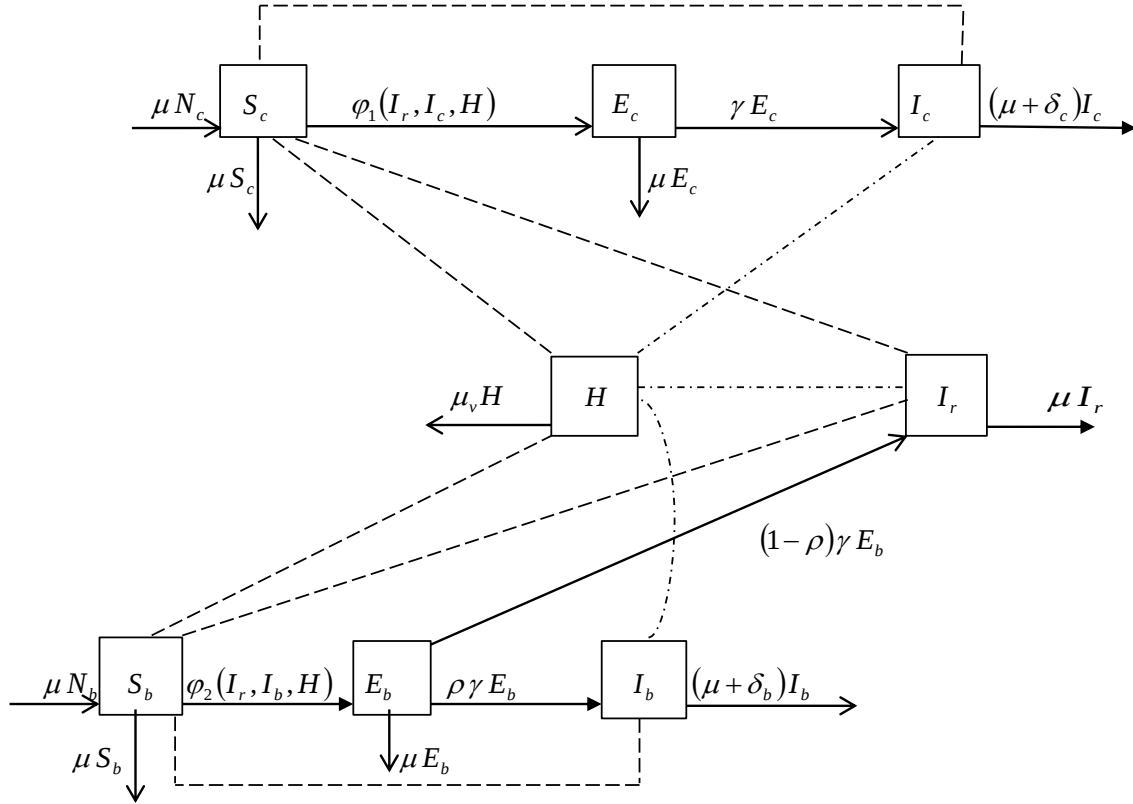


FIGURE 1. Compartment model diagram for the transmission dynamics of Newcastle disease in village chicken population. The solid lines shows the constant transmission from one compartment to another, the dotted lines shows the normal interactions between different compartments and a dash dot lines represents the shedding of NDV onto the environment

2.3. Model Equations. The dynamics of NDV is described by the following system of nonlinear differential equations;

$$\begin{aligned}
 (2.1) \quad & \frac{dS_c(t)}{dt} = \mu N_c - \varphi_1(t, I_c, I_r, H) S_c(t) - \mu S_c(t), \\
 & \frac{dE_c(t)}{dt} = \varphi_1(t, I_c, I_r, H) S_c(t) - (\gamma + \mu) E_c(t), \\
 & \frac{dI_c(t)}{dt} = \gamma E_c(t) - (\delta_c + \mu) I_c(t), \\
 & \frac{dS_b(t)}{dt} = \mu N_b - \varphi_2(t, I_b, I_r, H) S_b(t) - \mu S_b(t), \\
 & \frac{dE_b(t)}{dt} = \varphi_2(t, I_b, I_r, H) S_b(t) - (\gamma + \mu) E_b(t), \\
 & \frac{dI_b(t)}{dt} = \rho \gamma E_b(t) - (\delta_b + \mu) I_b(t), \\
 & \frac{dI_r(t)}{dt} = (1 - \rho) \gamma E_b(t) - \mu I_r(t), \\
 & \frac{dH(t)}{dt} = \varphi_3(t, I_c, I_b, I_r) - \mu_v H(t)
 \end{aligned}$$

where;

$$\begin{aligned}
 \varphi_1(t, I_c, I_r, H) &= \left(b \frac{I_r(t)}{N_b} + \psi \frac{I_c(t)}{N_c} + d \frac{H(t)}{\kappa + H(t)} \right), \\
 \varphi_2(t, I_b, I_r, H) &= \left(\frac{\phi I_b(t) + a I_r(t)}{N_b} + d \frac{H(t)}{\kappa + H(t)} \right), \\
 \varphi_3(t, I_c, I_b, I_r) &= \alpha_c I_c(t) + \alpha_b (I_b(t) + I_r(t))
 \end{aligned}$$

With initial conditions;

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

2.4. Basic properties of the model.

2.4.1. Invariant region of the solution. The Newcastle disease model system (2.1) has three subpopulations where all parameters and variables are positive $\forall t \geq 0$.

Lemma 2.1. *Given the model system (2.1) 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*

$$\Omega = \{S_c(t), E_c(t), I_c(t) \in \mathbb{R}_+^3; S_b(t), E_b(t), I_b(t), I_r(t) \in \mathbb{R}_+^4; H(t) \in \mathbb{R}_+^1\}$$

Proof; To establish the feasible region of the Newcastle disease model solution, we apply the box invariant method as used by [1, 15]. For our dynamical system $\dot{q} = g(X, t)$, $X \in \mathbb{R}^n$, we

assume the continuity and the Lipschitz properties of its solution. The model system (2.1) is reduced to the form

$$(2.2) \quad \frac{dX}{dt} = Q(x)X + G$$

where $X = (S_c, E_c, I_c, S_b, E_b, I_b, I_r, H)^T$ and a column vector $G = (N_c, 0, 0, N_b, 0, 0, 0, 0)^T \geq 0$. G is a Lipschitz continuous [1, 21] and

$$(2.3) \quad Q(x) = \begin{pmatrix} Q_1(x) & 0 & 0 \\ 0 & Q_2(x) & 0 \\ 0 & 0 & Q_3(x) \end{pmatrix}$$

is a Metzler matrix for all $X \in \mathbb{R}_+^8$ with sub-matrix $Q_1(x)$, $Q_2(x)$ and $Q_3(x)$ from the village chicken, wild birds and environment respectively. We define the sub matrices from the system (2.3) as follows;

$$(2.4) \quad Q_1(x) = \begin{pmatrix} -(\varphi_1(t, I_c, I_r, H) + \mu) & 0 & \frac{\psi}{N_c} \\ \varphi_1(t, I_c, I_r, H) & -(\gamma + \mu) & 0 \\ 0 & \gamma & -(\delta_c + \mu) \end{pmatrix}$$

$$(2.5) \quad Q_2(x) = \begin{pmatrix} -(\varphi_2(t, I_b, I_r, H) + \mu) & 0 & 0 & \frac{b}{N_b} \\ \varphi_2(t, I_b, I_r, H) & -(\gamma + \mu) & 0 & \frac{a}{N_b} \\ 0 & \rho\gamma & -(\delta_b + \mu) & 0 \\ 0 & (1 - \rho)\gamma & 0 & -\mu \end{pmatrix}$$

$$(2.6) \quad Q_3(x) = \begin{pmatrix} 0 & 0 & \alpha_c & 0 & 0 & \alpha_b & \alpha_b & -\mu_v \end{pmatrix}$$

By combining the matrices in equation (2.4), (2.5) and (2.6) we get the matrix $Q(x)$ which is a Metzler matrix for all $X \in \mathbb{R}_+^8$ defined as;

$$(2.7) \quad Q(x) = \begin{pmatrix} -A_1 & 0 & \frac{\psi}{N_c} & 0 & 0 & 0 & 0 & 0 \\ A_2 & -(\gamma + \mu) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma & -(\delta_c + \mu) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_3 & 0 & 0 & \frac{b}{N_b} & 0 \\ 0 & 0 & 0 & A_4 & -(\gamma + \mu) & 0 & \frac{a}{N_b} & 0 \\ 0 & 0 & 0 & 0 & \rho\gamma & -(\delta_b + \mu) & 0 & 0 \\ 0 & 0 & 0 & 0 & (1 - \rho)\gamma & 0 & -\mu & 0 \\ 0 & 0 & \alpha & 0 & 0 & \alpha & \alpha & -\mu_v \kappa \end{pmatrix}$$

where

$$A_1 = \varphi_1(t, I_c, I_r, H) + \mu$$

$$A_2 = \varphi_1(t, I_c, I_r, H)$$

$$A_3 = \varphi_2(t, I_b, I_r, H) + \mu$$

$$A_4 = \varphi_2(t, I_b, I_r, H)$$

A reduced Metzler matrix $Q(x)$ in (2.7) has all negative values along its principle diagonal and the rest non-negative values in its off diagonal. Hence proves that all variables enters and remains in the invariant region Ω . This shows that the Newcastle disease model system (2.1) is epidemiologically meaningful and well posed in Ω .

2.4.2. Positivity of the solution.

Theorem 2.1. *Let the initial value of variables of the model in the equation (2.1) be $S_c(0) > 0$, $E_c(0) > 0$, $I_c(0) > 0$, $S_b(0) > 0$, $E_b(0) > 0$, $I_r(0) > 0$, $I_b(0) > 0$ and $h(0) > 0$ 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 time t .*

Proof

Lets consider the first equation of the model (2.1)

$$(2.8) \quad \frac{dS_c(t)}{dt} = \mu N_c - \varphi_1(t, I_c, I_r, H)S_c(t) - \mu S_c(t)$$

$$(2.9) \quad \frac{dS_c(t)}{dt} \geq -(\varphi_1(t, I_c, I_r, H) + \mu) S_c(t)$$

$$(2.10) \quad \int_0^t \frac{dS_c(t)}{S_c(t)} \geq - \int_0^t (\varphi_1(t, I_c, I_r, H) + \mu) dt$$

$$(2.11) \quad S_c(t) \geq S_c(0)e^{-\mu t - \int_0^t (\varphi_1(t, I_c, I_r, H)dt)}$$

Thus as $t \rightarrow \infty$ then it follows that $S_c(t) \geq S_c(0)e^{-\mu t - \int_0^t (\varphi_1(t, I_c, I_r, H)dt)} > 0$. From the second equation of the model (2.1) we have

$$(2.12) \quad \frac{dE_c(t)}{dt} = \varphi_1(t, I_c, I_r, H)S_c(t) - (\gamma + \mu)E_c(t)$$

$$(2.13) \quad \frac{dE_c(t)}{dt} \geq -(\gamma + \mu)E_c(t)$$

$$(2.14) \quad \frac{dE_c(t)}{E_c(t)} \geq -(\gamma + \mu)dt$$

Integrating both sides of equation(2.14) *w.r.t* we have

$$(2.15) \quad \int_0^t \frac{dE_c(t)}{E_c(t)} \geq - \int_0^t (\gamma + \mu) dt$$

and finally we get

$$(2.16) \quad E_c(t) \geq E_c(0)e^{-(\gamma+\mu)t}$$

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

Also from the third equation of the model (2.1) we have

$$(2.17) \quad \frac{dI_c(t)}{dt} = \gamma E_c(t) - (\delta_c + \mu)I_c(t)$$

$$(2.18) \quad \int_0^t \frac{dI_c(t)}{I_c} \geq - \int_0^t (\delta_c + \mu) dt$$

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

2.5. Existence of the disease free Equilibrium point.

2.5.1. *Disease Free Equilibrium point (DFEP).* The model has a disease free equilibrium point (DFEP) which is obtained when all infectious states of the model are set to zero. Setting $I_c(t) = I_r(t) = I_b(t) = H(t) = 0$ in the model system (2.1) we then have the disease free equilibrium point $D_c^0 = \{N_c, 0, 0\}$, $D_b^0 = \{N_b, 0, 0, 0\}$ and $D_H^0 = 0$ 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 $D^0 = \{N_c, 0, 0, N_b, 0, 0, 0\}$.

2.6. **The basic Reproductive number, $\mathcal{R}_0$.** 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[9, 11, 23]. This number tells and quantifies the ability of an infectious disease to invade a purely susceptible population and persist[9, 11]. The Epidemic persists when $\mathcal{R}_0 > 1$ and dies out when the $\mathcal{R}_0 < 1$ [7, 12, 31, 32]. We compute the $\mathcal{R}_0$ by the next generation method as proposed by [31]. By [31], we define our system for infectious compartments as

$$(2.19) \quad \frac{dX_i}{dt} = \mathcal{F}_i - \mathcal{V}_i$$

From equation (2.19), X_i defines a set of infected classes, $\mathcal{F}_i$ defines the rate of new infections in compartment i and the total transfer rate is defined by $\mathcal{V}_i = \mathcal{V}_i^- - \mathcal{V}_i^+$, where $\mathcal{V}_i^-$ is 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;

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

The corresponding Jacobian matrices of F and V are the matrices of the derivatives of $\mathcal{F}_i$ and $\mathcal{V}_i$ w.r.t $E_c(t)$, $I_c(t)$, $E_b(t)$, $I_r(t)$, $I_b(t)$ and $H(t)$ at the disease free point, D^0 which are given by

$$F = \left(\frac{\partial F_i(D^0)}{\partial X_j} \right) \text{ and } V = \left(\frac{\partial V_i(D^0)}{\partial X_i} \right) \text{ respectively.}$$

Then we get the following matrices

$$(2.20) \quad F = \begin{pmatrix} 0 & \psi & 0 & 0 & b \frac{N_c}{N_b} & d \frac{N_c}{\kappa} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \phi & a & d \frac{N_b}{\kappa} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$(2.21) \quad V = \begin{pmatrix} \gamma + \mu & 0 & 0 & 0 & 0 & 0 \\ -\gamma & \delta_c + \mu & 0 & 0 & 0 & 0 \\ 0 & 0 & (\gamma + \mu) & 0 & 0 & 0 \\ 0 & 0 & -\rho \gamma & (\delta_b + \mu) & 0 & 0 \\ 0 & 0 & -(1 - \rho)\gamma & 0 & \mu & 0 \\ 0 & -\alpha_c & 0 & -\alpha_b & -\alpha_b & \mu_v \end{pmatrix}$$

The inverse of matrix V in equation (2.21) become

$$(2.22) \quad V^{-1} = \begin{pmatrix} (\gamma + \mu)^{-1} & 0 & 0 & 0 & 0 & 0 \\ \frac{\gamma}{(\gamma + \mu)(\delta_c + \mu)} & (\delta_c + \mu)^{-1} & 0 & 0 & 0 & 0 \\ 0 & 0 & (\gamma + \mu)^{-1} & 0 & 0 & 0 \\ 0 & 0 & \frac{\rho\gamma}{(\gamma + \mu)(\delta_b + \mu)} & (\delta_b + \mu)^{-1} & 0 & 0 \\ 0 & 0 & -\frac{(-1 + \rho)\gamma}{(\gamma + \mu)\mu} & 0 & \mu^{-1} & 0 \\ \frac{\gamma\alpha_c}{(\gamma + \mu)(\delta_c + \mu)\mu_v} & \frac{\alpha_c}{(\delta_c + \mu)\mu_v} & -\frac{\alpha_b(-\gamma\delta_b - \gamma\mu + \gamma\rho\delta_b)}{(\gamma + \mu)(\delta_b + \mu)\mu\mu_v} & \frac{\alpha_b}{(\delta_b + \mu)\mu_v} & \frac{\alpha_b}{\mu\mu_v} & \mu_v^{-1} \end{pmatrix}$$

We then compute the next generation matrix (FV^{-1}) which gives

$$(2.23) \quad FV^{-1} = \begin{pmatrix} R & S & T & \frac{dN_c\alpha_b}{\kappa(\delta_b + \mu)\mu_v} & \frac{bN_c}{N_b\mu} + \frac{dN_c\alpha_b}{\kappa\mu\mu_v} & \frac{dN_c}{\kappa\mu_v} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ U & V & W & \frac{\phi}{\delta_b + \mu} + \frac{dN_b\alpha_b}{\kappa(\delta_b + \mu)\mu_v} & \frac{a}{\mu} + \frac{dN_b\alpha_b}{\kappa\mu\mu_v} & \frac{dN_b}{\kappa\mu_v} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

where

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

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

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

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

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

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

Basing on the matrix (2.23), the basic reproductive number $\mathcal{R}_0$ is the spectral radius of the next generation matrix $(\rho(FV^{-1}))$. This gives the basic reproduction number as

$$(2.24) \quad \mathcal{R}_0 = \rho(FV^{-1}) = \frac{1}{2} \left((R + W) + \sqrt{(R - W)^2 + 4UT} \right)$$

$$(2.25) \quad \mathcal{R}_0 = 1/2 \left(R + \frac{\phi \rho \gamma}{(\gamma + \mu)(\delta_b + \mu)} - \frac{a(-1 + \rho)\gamma}{(\gamma + \mu)\mu} - \frac{dN_b \alpha_b (-\gamma \delta_b - \gamma \mu + \gamma \rho \delta_b)}{\kappa (\gamma + \mu)(\delta_b + \mu) \mu \mu_v} \right) \\ + 1/2 \sqrt{\left(R - \frac{\phi \rho \gamma}{(\gamma + \mu)(\delta_b + \mu)} + \frac{a(-1 + \rho)\gamma}{(\gamma + \mu)\mu} + \frac{dN_b \alpha_b (-\gamma \delta_b - \gamma \mu + \gamma \rho \delta_b)}{\kappa (\gamma + \mu)(\delta_b + \mu) \mu \mu_v} \right)^2 + 4\omega}$$

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 (2.25) the basic reproduction number $\mathcal{R}_0$ is influenced by parameters from all subpopulations of the model. In this equation, $\frac{\gamma}{(\mu + \delta_c)}$ and $\frac{\gamma}{(\mu + \delta_b)}$ respectively refers the probabilities that village chicken and wild birds survive throughout the whole incubation period in the presence of Newcastle disease and $\frac{\psi}{(\mu + \delta_c)}$ is the probability of village chicken to acquire NDV when come into contact with the infectious village chicken throughout its entire infectious period. The rate $\frac{\phi}{(\mu + \delta_b)}$ is the probability of wild birds to acquire NDV when come into contact with chronically affected wild bird through its entire infectious period. However $\frac{d}{\kappa(\gamma + \mu)}$ indicates the probabilities of village chicken and wild birds to acquire the Newcastle disease virus from the environment during the outbreak of the disease.

2.7. Sensitivity analysis of $\mathcal{R}_0$. 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 [26]. In order to get the sensitivity value of a parameter in the model system (2.1), a function $\mathcal{R}_0$ should be differentiable at a parameter q where q being any parameter in $\mathcal{R}_0$. Therefore, in this section we perform the sensitivity analysis of $\mathcal{R}_0$ by using the normalized forward sensitivity analysis index as applied in [5]. 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 derived 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 Newcastle disease induced death $\delta_c = 0.01984day^{-1}$. Wild birds are assumed to be five hundred thousand (500,000) and its disease induced death is assumed to be $\delta_b = 0.0025day^{-1}$. We estimate an average lifespan of village chicken to be two to ten years which yields a mortality rate $\mu = 1.37 \times 10^{-3}day^{-1}$ [18]. The parameter κ gives the density of Newcastle disease virus in the environment that yields 50% chance of infection which is assumed to be 10000 virus per cubic meter of the soil. Other parameter values and the sensitivity analysis values are summarized in Table 3 and table 4 respectively.

TABLE 3. Parameter values of the model 2.1

Parameter	Parameter value	Source
a	$0.01day^{-1}$	Assumed
b	$0.21day^{-1}$	[2, 8]
α_c	$0.01667virus^{-1}chicken^{-1}day^{-1}$	Assumed
α_b	$0.02virus^{-1}chicken^{-1}day^{-1}$	Assumed
ψ	$0.0083day^{-1}$	Assumed
μ	$1.37 - 5.48 \times 10^{-4}day^{-1}$	[18]
ϕ	$0.02day^{-1}$	Assumed
ρ	0.9	Assumed
d	$0.001day^{-1}$	Assumed
γ	$0.067day^{-1}$	[29, 30]
δ_b	$0.025day^{-1}$	[6]
δ_c	$0.01989day^{-1}$	[14]
μ_v	$0.00219day^{-1}$	Assumed
κ	10000 virus / m^3	Assumed

TABLE 4. Sensitivity Indices for Parameters of the model 2.1

Parameter	Index value	Parameter	Index value
a	+0.00345	ψ	+0.38706
b	+0.01530	d	+0.58770
α_b	+0.33654	α_c	+0.25116
γ	+0.00811	μ	-0.07953
δ_b	-0.30751	ϕ	+0.00647
δ_c	-0.62107	μ_v	-0.58770
κ	-0.58770		

The positive indices shows that the basic reproductive number increases as the value of the parameter increase. In Table 4, parameters a , b , α_b , α_c , ψ , γ , ϕ and d have positive indices. These parameters have a positive influence on the basic reproductive number, $\mathcal{R}_0$. The contact rate between the susceptible population of village chicken and wild birds with contaminated environment (d) is the most positive sensitive parameter of the model followed by the transmission rate between chronically infected village chicken and the susceptible chicken (ψ). The least positive sensitive index values is the transmission rate between chronically infected wild birds and the susceptible wild birds (ϕ). The negative index values implies the inverse relationship between the basic reproduction number and the value of the parameter. Parameters δ_c , δ_b , κ , μ , and μ_v have negative index values. According to these results, the Newcastle disease induced death rate (δ_c) in village chicken is the most negative sensitive parameter in the model. This tells us that more deaths of the village chicken during the outbreak of the disease reduce interactions between the infected population and the at risk population and hence lower the basic reproduction number. We observe also that the clearance rate (μ_v) have the inverse relations with the $\mathcal{R}_0$ and thus increasing clearance of the NDV from the environments lowers the value of the basic reproduction number and the vise versa. The natural birth rate (μ) is the least negative sensitive parameter in the model.

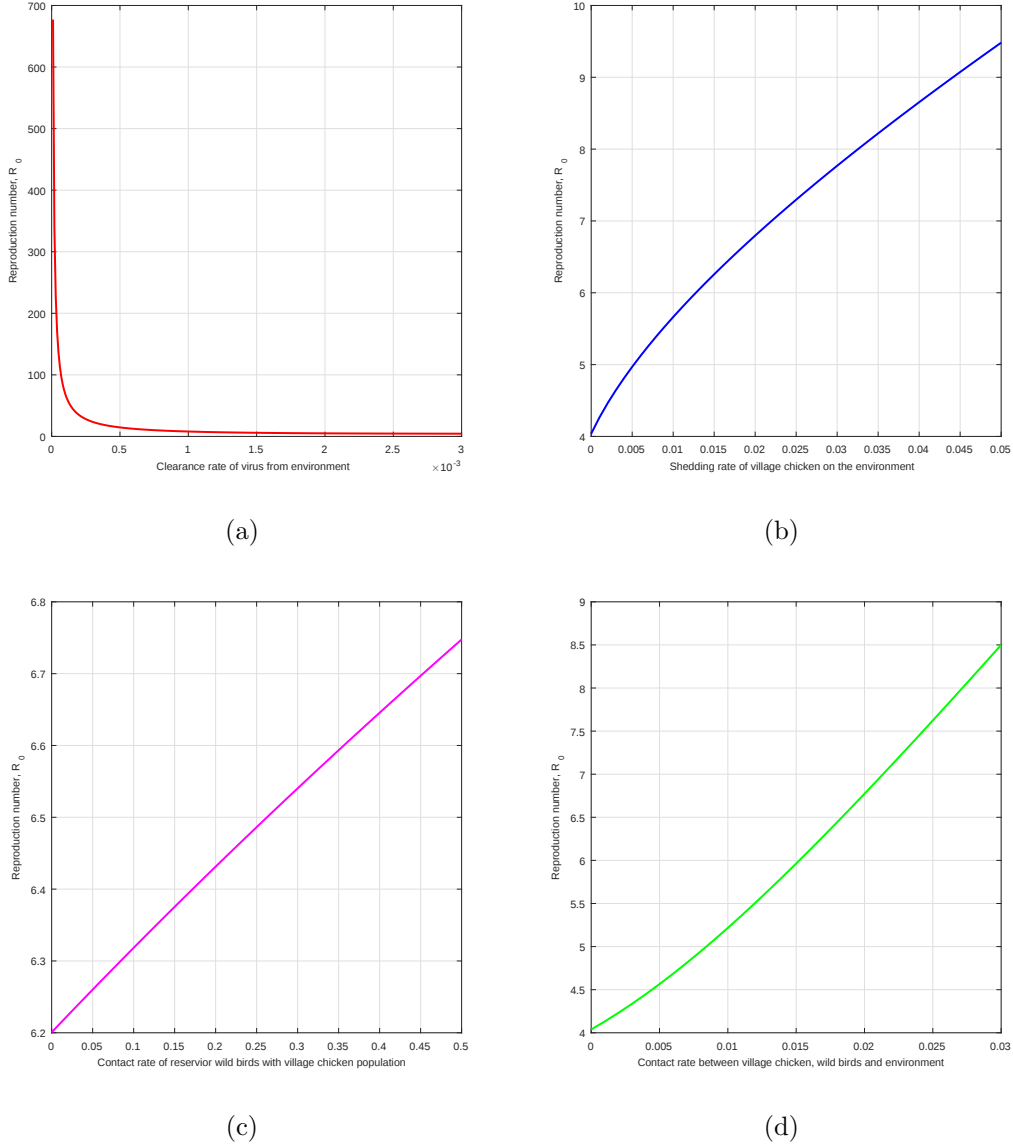
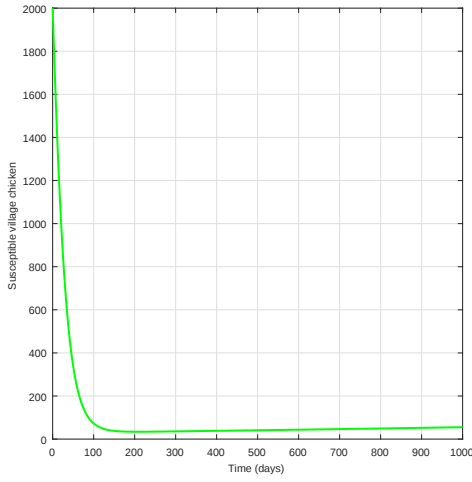


FIGURE 2. Shows (a) the effects of the clearance rate of NDV virus from the environment on $\mathcal{R}_0$, (b) the effects of the shedding rate of NDV into the environment on $\mathcal{R}_0$, (c) the effects of the contact rate of carrier wild birds and susceptible village chicken on $\mathcal{R}_0$ and (d) the effects of the contact rate between the susceptible population of village chicken and wild birds with contaminated environment on $\mathcal{R}_0$.

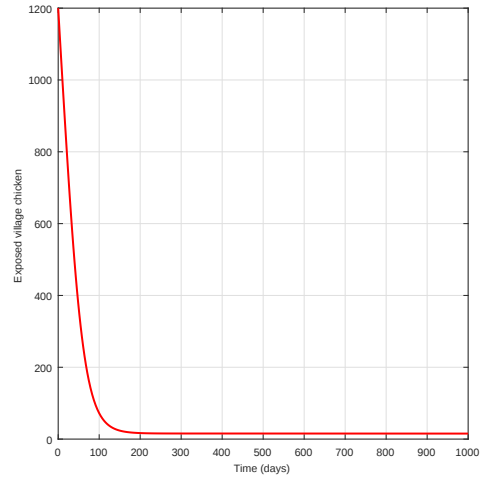
According to Figure 2 (a), increasing the clearance of the active Newcastle disease virus from the environment leads to the reduction of the value of $\mathcal{R}_0$ and hence reduces the rate of spread of the NDV in the avian population. Figure 2 (b) shows the direct relationship of the shedding rate of the NDV in the environment by the hosts on $\mathcal{R}_0$. The more the shedding

the higher the degree of the NDV transmission in the population. Figure 2(c) shows the direct effects of the contact rate of the village chicken and wild birds with the contaminated environment with the *NDV*. More contacts during the epidemic leads to the increase in the rate of spread of the Newcastle disease. Figure 2(d) shows the direct relationship of the contact rate between the susceptible population of village chicken and wild birds with contaminated environment on $\mathcal{R}_0$. Therefore in order to control the spread of Newcastle disease, the most positive sensitive parameters needs to be controlled.

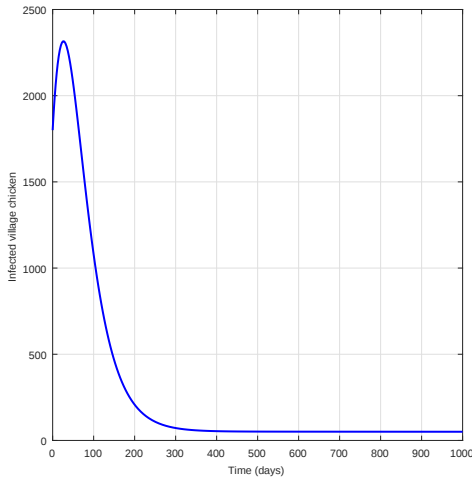
3. NUMERICAL SIMULATION OF THE BASIC MODEL



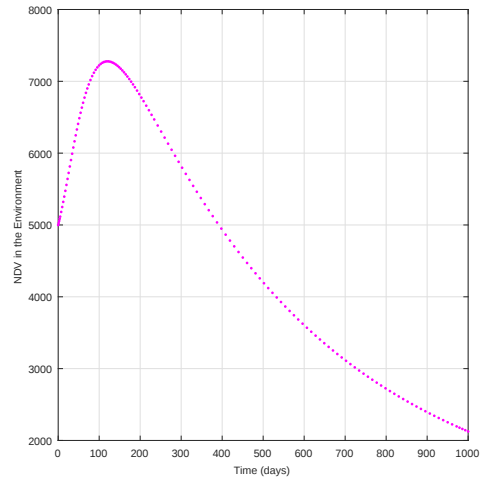
(a)



(b)



(c)



(d)

FIGURE 3. The dynamics of Newcastle disease in village chicken without any control. (a) shows susceptible population of the chicken (b) and (c) shows the exposed and infected population of the chicken and (d) shows the NDV in the environment

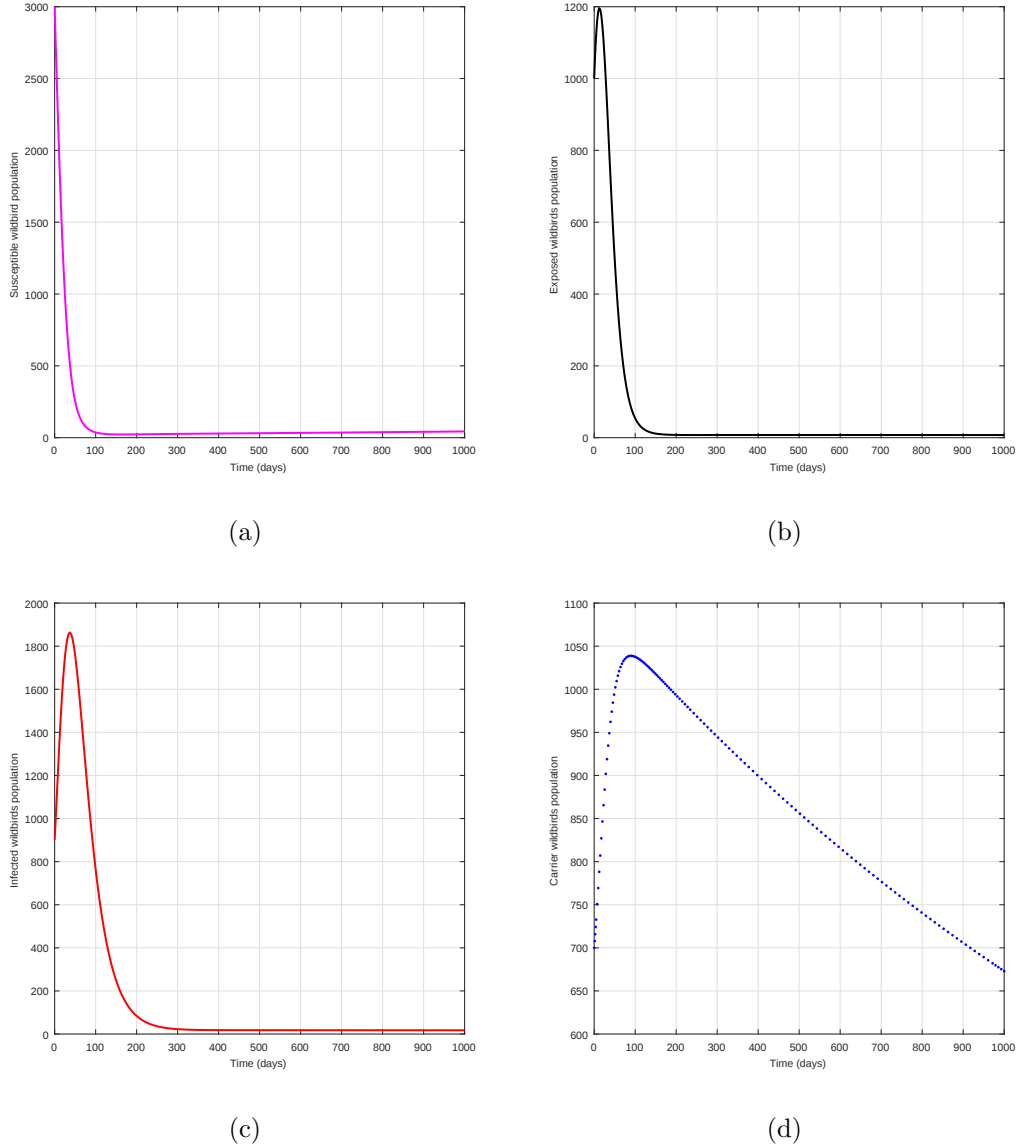


FIGURE 4. The dynamics of Newcastle disease in the wild birds. (a) shows susceptible population of the wild birds, (b) shows the exposed population of wild birds, (c) shows the infected population of the wild birds and (d) shows the carrier population of the wild birds

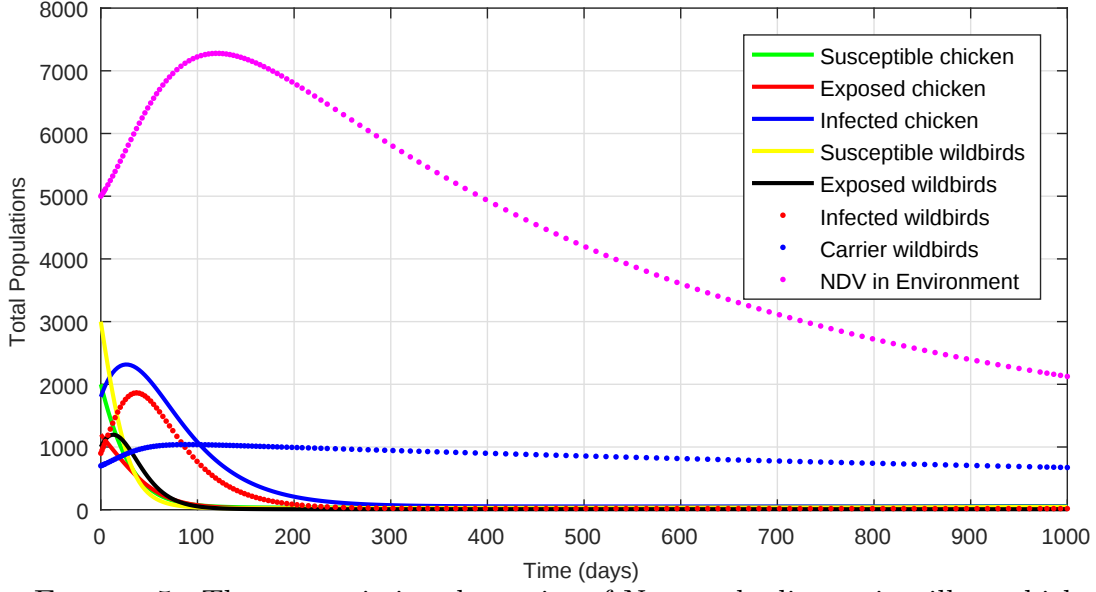


FIGURE 5. The transmission dynamics of Newcastle disease in village chicken population, wild birds population and the NDV in the Environment during the outbreak in the absence of any control with initial state conditions $S_c(0) = 2000$, $E_c(0) = 1200$, $I_c(0) = 1800$, $S_b(0) = 3000$, $E_b(0) = 1000$, $I_b(0) = 900$, $I_r(0) = 700$, $H(0) = 5000$ and the estimated parameter values $\psi = 0.0083$, $a = 0.01$, $\gamma = 0.067$, $\mu = 0.000548$, $\delta_c = 0.01984$, $\delta_b = 0.025$, $d = 0.025$, $\rho = 0.9$, $\alpha = 0.001667$, $\mu_v = 0.00219$, $\phi = 0.02$, $b = 0.21$ and $\mathcal{R}_0 \approx 6.867$.

The Figure 3(a-d) and figure 4(a-d) show simulations of the dynamics of Newcastle disease in the village chicken and wild birds population in the endemic situation respectively. In figure 3(a), the susceptible village chicken population is exponentially decreasing due to the presence of ND in the population. Figure 3(b) represents the exposed population of the village chicken. The population decreases exponentially right from the first day after the infections. Normally infections takes place between day one to day six before 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 severe sickness class as shown in Figure 3(c). The population of the infected chicken increase for a short period of time due to new chicken which progress from the exposed class. The population of this class suddenly decreases exponentially due to the higher mortality rate of the ND. Figure 3(d) represents the NDV in the environment. We observe that during the first 100 days since infection the concentration of NDV in the environment increases to the carrying capacity due to higher shading rate of the NDV by the hosts. At its maximal point depending on the environmental conditions, the concentration of active virus decreases and thus lowers the transmissions of the disease. Figure 4(a) represents the population of susceptible wild birds in which we observe a sharp

decrease due to higher rate of infection of the NDV. Due to short window period of the ND, we observe a marginal increase of the population of exposed wild birds during the first few days of the disease outbreak which is followed by an exponential decline to the lowest level (see Figure 4(b)). The similar pattern is observed in Figure 4(c), as the population of the infected wild birds increases during the first few days since the disease outbreak and thereafter followed by an exponential decline to the lowest level due to the fatality of the ND. Figure 4(d) is a population of the survivors of the disease. This includes wild birds with immunity against ND in such a way that they remain carrier of the virus throughout the outbreak. Figure 5 depicts the dynamics of the three population (village chicken, wild birds and NDV in the environment) at the endemic situation when plotted in the same axis.

4. DISCUSSION AND CONCLUSION

In this paper, we developed and analyzed a mathematical model to elucidate the transmission dynamics of Newcastle disease in village chicken which include vital components of the wild birds and the contaminated environment. The invariant region and positivity analysis of the model is presented which showed that the model is biologically meaningful and well posed in the feasible region. The basic reproduction number, $\mathcal{R}_0$ is computed and discussed. The sensitivity analysis results showed that, the shedding rate of *NDV* from the chronically infected village chicken, the infected wild birds and the carrier population of wild birds to the environment and the contact rate between susceptible chicken and wild birds with the environment (d), have direct impacts on the basic reproduction number, $\mathcal{R}_0$. The increase in values for these parameters leads to the increase of the basic reproductive number, $\mathcal{R}_0$ whilst their decrease lowers the value of $\mathcal{R}_0$. The clearance rate of the virus from the environment have also a great influence in the dynamics of the Newcastle disease among village chicken population. Increasing of the clearance rate, lowers the value of $\mathcal{R}_0$. Hence, to control the rate of spread of Newcastle disease in the Village chicken it is of a paramount importance to limit the contact between wild birds and chicken and to increase the clearance rate of NDV in the environment through sanitation. However, more improvements to include the age structure and seasonal variations on the model are needed for further understanding of the dynamics of Newcastle disease in the village chicken population. Our model therefore, provides a baseline for the investigation of the control strategies of Newcastle disease among village chicken under a free range management system.

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