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Mathematical and Control Model of Bursal Disease (Ibd)

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Abstract: *We formulated a four compartmental model of Infectious Bursal Disease (IBD) for both the ordinary and control models. We first determined the basic Reproduction number and the existence of Steady (Equilibrium) states (disease-free and endemic). Conditions for the local stability of the disease-free and endemic steady states were determined. Further, the Global stability of the disease-free equilibrium (DFE) and endemic equilibrium were proved using Lyponav method. We went further to carry out the sensitivity analysis or parametric dependence on R_0 and later formulated the optimal control problem. We finally looked at numerical Results on poultry productivity in the presence of Infectious Bursal Disease (IBD) and we drew six graphs to demonstrate this. From Figure 4.1 we observe that the number of birds at the early stage of the infection increases rapidly and drops to a particular level. Applying the both control measures reduces the number to zero. This strategy shows a significant reduction in the number of birds at the early stage of the infection with control. It is observed from Figure 4.2 that the number of birds at the latter stage of the infection reduces with and without control. Without control the reduction is as a result of high mortality rate at this stage of the infection. With control the number reduces and drops to zero. Therefore we may conclude that applying both controls the infectious birds will drop to zero over time. We observe from Figure 4.3 that the number of infectious bird will keep reducing as the use of vaccination increases. At the optimal application of vaccines the number of infectious birds becomes less than 5 and remains at that level over time. From Figure 4.4, at the optimal application of only vaccines, the number of birds at the latter stage of the infection will drop to zero. This implies that strategy B (that is, when supportive measures to enhance recovery infectious birds are not applied) will be more effective on the infectious birds at the latter stage of the infection than on birds at the early stage of the infection.*

We observe that from Figure 4.5 that the number of birds at the early stage of the infection will also reduce when only supportive measures are applied but will increase the number of infectious birds gradually after about 10 days. When only supportive measures are applied in $I_{L,}$ we see from Figure 4.6 that the number drops to zero faster even at the early application of the strategy. Hence we can deduce that this strategy is more effective on birds at the latter stage of the infection and less effective on birds at the early stage of infection.

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I. Introduction

Infectious bursal disease also known as Gumboro disease is a highly contagious immunosuppressive viral infection of poultry birds. The causative agent of IBD is a bi-segmented, double-stranded RNA virus of the Birnaviridae family named IBD virus (IBDV). Although turkeys, ducks, guinea fowls and ostriches may be infected, clinical disease occurs solely in chickens, OIE (2008). However, serological evidence of the infection has been reported in free – living wild birds such as corden bleu and village weaver, Antartic penguins, wild water birds, cattle egrets and wild turkeys, Candelora et al (2010). Moreover infectious bursal disease virus antibodies were detectable in the sera of sedentary and migratory wild bird species in Japan, suggesting that they play a key role in the natural history of infectious bursal disease, Oluwayelu et al (2014).

It affects young chickens and is characterized by the destruction of the lymphoid organs. The infection when not fatal or in the early stage, causes an immune suppression, Janmaat (2010), in most cases temporary, the degree of which is often difficult to determine. IBD has been described throughout the world and socio – economic significance of the disease is considerable world – wide.

Infectious bursal disease is one of the major economically important diseases of poultry worldwide despite wide usage of vaccination programs. Most commercial chickens are exposed to IBDV early in life. The infectious bursal disease virus (IBDV) is the etiological agent of infectious bursal disease (IBD) also known as Gumboro disease. In unprotected flocks, the virus causes mortality and immunosuppression. Although mortality

can be quite significant, the major economic loss is the ability of IBDV to produce immunosuppression. Immunosuppressed flocks have poor performance which results in reduced economic return, Alfred et al (2016).

Infectious bursal disease (IBD) was first reported by Cosgrove in 1957in broilers of the Delmarva Peninsula of the United States. It was initially recognized as "avian nephrosis", and the syndrome became known as "Gumboro disease" because the first outbreaks occurred in the town of Gumboro, Delaware, USA. The causative agent of IBD is a bi-segmented, double-stranded RNA virus of the Birnaviridae family named IBD virus (IBDV). It had spread rapidly throughout the United States by 1965 but was effectively controlled by vaccinations in the mid-1970s (Lasher & Davis 1997). Initially avian nephrosis or Gumboro disease was thought to be caused by the Gray strain of infectious bronchitis virus (IBV) because of gross changes in the kidney. This misconception arose because the IBV and IBDV infections were concurrent in many cases and IBDV was difficult to isolate with the available methods at the time of discovery.

In later studies, (Winterfield et al in Mohamed 2006), succeeded in isolating the causative agent in embryonating eggs, and later Hitchner proposed the term "infectious bursal disease" for the disease. There is a significant antigenic, immunogenic, and pathogenic variation between IBDV strains which determines disease outcome. Some IBDV strains cause an immunosuppressive, subclinical form of disease with less than 5% mortality, while others can cause a clinical form with up to 100% mortality such as very virulent strains. The clinical signs and the degree of immunosuppression can also vary significantly. Vaccination is the primary means for control; and thus most efforts for protection against IBDV by the commercial poultry industry are focused on developing efficient vaccination programs. Successful immunization requires reliable IBDV field and vaccine strain characterization (Mohamed 2006).

There are two serotypes of IBDV: 1 and 2. All viruses capable of causing disease in chickens belong to serotype 1, whereas serotype 2 viruses are non-pathogenic for both chickens and turkeys (Muller et al 2012). Chickens are the only avian species known to be susceptible to clinical disease and lesions produced by IBDV. Turkeys, ducks and ostriches are susceptible to infection with IBDV but are resistant to its clinical manifestations (Alfred et al 2016). IBDV has also been isolated from African black-footed and Macaroni penguins and have been serologically identified as serotype 2 IBDV (Sudhir et al 2016), and further confirmed as serotype 2 by molecular identification.

In 1972, it was reported that IBDV infections at an early age were immunosuppressive (Boot et al 2005). The recognition of this immunosuppressive capability of IBDV greatly increased the interest in the control of this disease. The existence of serotype 2 IBDV was reported in 1980. The Delmarva Peninsula broiler growing area experienced a significant increase in mortality and higher percentage of condemnations in 1984 and 1985. The clinical syndrome had significant variability, but often was respiratory in nature. Lesions ranged from moderate to severe, with death usually being attributed to E. coli infection (38).

Rosenberger et al in Mohamed (2006) isolated four isolates designated as A, D, G, and E using vaccinated sentinel birds. These isolates differed from standard strains in that they produced a very rapid bursal atrophy associated with minimal inflammatory response. The available killed standard vaccines did not provide complete protection against these four new Delaware isolates. The Delaware isolates, A, D, G and E were designated as antigenic variants and killed vaccines were developed, tested and proven effective against them (Li et al 2015). Currently these and other similar variants are widely distributed in the United States. Snyder et al (1994), first described variant viruses as newly emergent viruses due to a major antigenic shift within serotype 1 (Stoute et al 2013). The terminology given to these newly emergent viruses was "IBDV variants" as they were the result of a major antigenic shift within serotype 1, while the older serotype 1 viruses discovered prior to these newly emergent viruses were called standard or classical strains of IBDV.

Acute IBDV outbreaks exhibiting 30% to 60% mortality in broiler and pullet flocks, respectively, have been commonly reported in Europe since 1987. The first reports were made by Chettle et al in Boot et al (2005), and van den Berg et al. (2000). Some of these acute outbreaks occurred in broiler flocks where appropriate hygienic and prophylactic measures had been taken. Although no antigenic drift was detected, these strains of increased virulence were identified as very virulent IBDV (vvIBDV) strains.

These very virulent strains have rapidly spread all over Asia and other parts of the world in an explosive manner, following their introduction into Japan in the early 1990s (30).In the America, acute IBD outbreaks due to vvIBDV strains have already been reported in Brazil, and the Dominican Republic (Mohamed 2006). To date the terminology given to serotype 1 IBDV is standard or classic, variant, and very virulent IBDV.

Chung-Chai et al (2006) in their study investigated the phylogenetic origins of the genome segments of IBDV and estimated the time of emergence of its most recent common ancestors. With recently developed coalescence techniques, they reconstructed the past population dynamics of vvIBDV and timed the onset of its expansion to the late 1980s. Their analysis suggests that genome segment A of vvIBDV emerged at least 20 years before its expansion, which argues against the hypothesis that mutation of genome segment A is the major contributing factor in the emergence and expansion of vvIBDV. Alternatively, the phylogeny of genome

segment B suggests a possible reassortment event estimated to have taken place around the mid-1980s, which seems to coincide with its expansion within approximately 5 years. It was therefore hypothesized that the reassortment of genome segment B initiated vvIBDV expansion in the late 1980s, possibly by enhancing the virulence of the virus synergistically with its existing genome segment A. This report reveals the possible mechanisms leading to the emergence and expansion of vvIBDV, which would certainly provide insights into the scope of surveillance and prevention efforts regarding the disease.

Durairaj et al (2013) presented an in vivo experimental model developed to differentiate a new antigenic variant of IBDV. A hyper-immune serum to IBDV E/Del-type virus was generated in specific pathogen-free chickens and a standard volume of the hyper-immune serum was serially diluted and injected in specific pathogen-free birds via intravenous, subcutaneous, or intramuscular routes. The chickens were bled at different time points in order to evaluate the dynamics of virus neutralization titres. Based on the results, chickens were injected with different serum dilutions by the subcutaneous route. Twenty-four hours later, chickens were bled and then challenged with 100 median chicken infectious doses of the E/Del virus and a new IBDV variant. Chickens were euthanized at 7 days post infection and the bursa of Fabricius was removed for microscopic evaluation to determine the bursal lesion score. The determined virus neutralization titre along with the bursal lesion score was used to determine the breakthrough titre in the in vivo chicken model. Based on the data obtained, an antigenic subtype of IBDV was identified and determined to be different from E/Del. This model is a sensitive model for determination of IBDV antigenicity of non-tissue culture adapted IBDV.

In a recent study by Sufen et al (2016), they examined the bursa anatomical structure and pathological changes in specific-pathogen free (SPF) white leghorn chickens 0 to 8 weeks post hatch (w.p.h.) and IBDV BC6/85-infected SPF chickens 2 to 6 w.p.h. respectively, by histology, histopathology, immunohistochemistry, and transmission electron microscopy. This study showed that white leghorn chickens seem to be less susceptible to IBD.

Raja et al (2016) considered a novel infectious bursal disease virus.The findings from this study provide additional insight into the genetic exchange between attenuated and very virulent strains of IBDV circulating in the field. Furthermore, Alike &Rautenschein highlighted the pattern of virus evolution and new developments in prophylactic strategies, mainly the development of new generation vaccines, which will continue to be of interest for, research as well as field application in the future.

Abid (2016) formulated an optimal control problem for an SIR epidemic model with saturated incidence and saturated treatment. Two main efforts, namely treatment and vaccination are considered to limit the disease transmission. The impacts of vaccination and treatment on the disease transmission are discussed through the basic reproduction number. Then to achieve control of the disease, a control problem was formulated and the existence of the control is shown. Two control functions were used: one for vaccinating the susceptible and the other for treatment efforts for infectious individuals. Optimal control methods were used to characterize the optimal levels of the two controls. The effectiveness of the proposed control solution was shown by comparing the behaviour of controlled and uncontrolled systems. Numerical results show the impacts of two controls in decreasing both susceptible and infectious members of the population.

In view of the above, none of these studies have considered the optimal control strategies in preventing and controlling the outbreak of the disease. Therefore this study aims at providing a non – linear mathematical model which will study the impact of optimal control strategies for Infectious bursal disease in the presence of infective birds and non – linear incidence. Other works we have looked at in this paper were: Alfred et al (2016), OIE (2008), Chettle et al ( ), Mohamed (2006), Oluwayelu et al (2014), Rosenberger et al ( ), Winterfield et al

II. Formulation of Infectious Bursal Disease Model

2.1 Assumptions of the model

1. Infected birds first develop the early stage of infection
2. Birds at the early and latter stage of the infection are capable of transmitting the disease
3. Birds are immunosuppressed at the early stage of the infection
4. No natural death for infectious birds
5. Birds are recruited either by birth or immigration
6. Recovered birds will overtime become susceptible to the disease

2.2 **Parameters of the Model**

S(t) - Population of susceptible birds at time t
E(t) - Population of infectious birds at the early stage of the infection
L(t) - Population of infectious birds at the latter stage of the infection
R(t) - Population of recovered birds at time t
π - Recruitment rate of bids
β contact rate with infectious class

d - natural death rate of birds
λ - force of infection
μ - disease - induced death rate
α - rate of becoming acutely infected
p - probability of recovery from infection
τ - progression rate from early stage to latter stage of the infection
δ - rate of recovery of birds at the latter stage of the infection
ν - rate of losing immunity
u_1 - vaccination control measure
u_2 - supportive control measure
N(t) - Total population of birds at time t
N(t) = S(t) + E(t) + L(t) + R(t)

2.3 **Model Flow diagram**

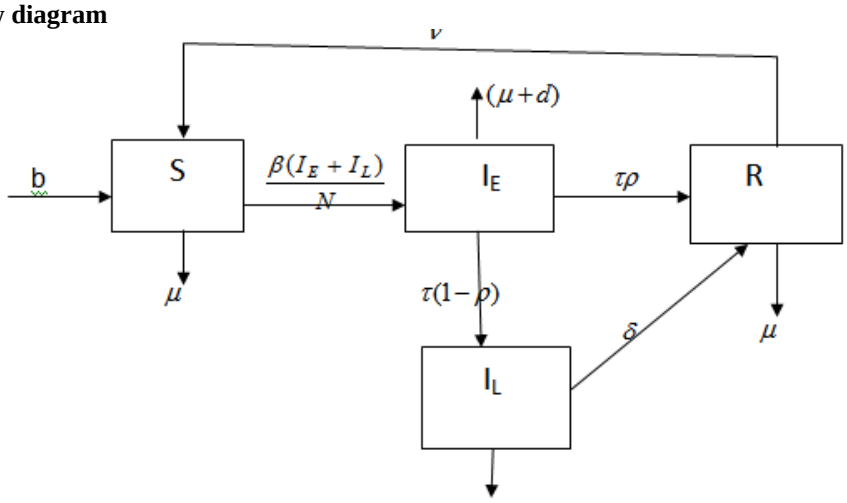

Fig. 2.1: Flow Diagram of Bursal Disease

2.4 **Equations of the Model**

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \frac{\beta(I_E + I_L)}{N}S - dS + \nu R \\
\frac{dI_E}{dt} &= \frac{\beta(I_E + I_L)}{N}S - \tau\rho I_E - \tau(1-\rho)I_E - (\mu + d)I_E \\
\frac{dI_L}{dt} &= \tau(1-\rho)I_E - \delta I_L - (\mu + d)I_L \\
\frac{dR}{dt} &= \tau\rho I_E + \delta I_L - (\nu + \mu)R
\end{aligned}\right\} \qquad (2.1)$$

2.5 Equation of Control Model

Now building in the controls $u_1(t)$ and $u_2(t)$ which are the vaccination control and the supportive control measures. These supportive measures are strategies such as proper ventilation, vitamin electrolyte therapy, water consumption etc used in enhancing the quick recovery of infected birds. The control model is given as

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \pi U_1 S - (1-U_1)\frac{\beta(I_E + I_L)}{N} S - dS + \nu R \\
\frac{dI_E}{dt} &= (1-U_2)\frac{\beta(I_E + I_L)}{N} S - \tau\rho I_E - \tau(1-\rho) I_E - (\mu + d) I_E \\
\frac{dI_L}{dt} &= \tau(1-\rho) I_E - U_2 I_L - (\mu + d) I_L \\
\frac{dR}{dt} &= \tau\rho I_E + U_2 I_L - (\nu + \mu) R
\end{aligned}\right\} \qquad (2.2)$$

III. Analysis of IBD model

3.1 Basic Reproduction number

The basic reproduction number (R_0) gives the expected number or average number of secondary infections produced when one single infected bird is introduced into the susceptible population. The basic reproduction number is calculated using the next generation operator approach. This approach calculates R_0 based on the definition of infected and uninfected compartments in the model.

Assuming that there are n compartments of which the first m compartments to infected individuals. We define,

$Vi\,(x) = V_i^-(x) - V_i^+(x)$

Where $V_i^+(x)$ is the rate of transfer of individuals into compartment i by all other means and $V_i^-(x)$is the rate of transfer of individual out of the ith compartment. It is assumed that each function is continuously differentiable at least twice in each variable. The disease transmission model consists of non-negative initial conditions together with the following system of equations:

$\dot{x_i} = h_i\,(x) = \mathcal{F}_i\,(x) - V_i\,(x)$, i=1,2,3,…n. where x is the rate of change of x.

The next is the computation of the square matrices F and $V$of order $m \times m$, where m is the number of infected classes, defined by F= $\left[\frac{\partial x_i}{\partial x_j} x(0)\right]$and V= $\left[\frac{\partial v_i}{\partial x_j} x(0)\right]$ with $1 \le i,j \le m$, such that F is nonnegative , V is a non - singular matrix and x(0) is the disease – free equilibrium point (DFE). Since F is non-negative and V non-singular, then V^{-1} is non-negative and also FV^{-1} is non-negative. Hence the matrix of FV^{-1} is called the next generation matrix for the model. Finally the basic reproduction number R_0 is given by

$R_0 = \rho(FV^{-1})$

where $\rho(A)$ denotes the spectral radius of matrix A and the spectral radius is the biggest non-negative eigenvalue of the next generation matrix.

From the system (3.1a), $\mathcal{F}_i$ and V_i are defined as:

$$\mathcal{F}_i = \begin{bmatrix} \beta\left(\frac{I_E + I_L}{N}\right) S \\ 0 \end{bmatrix} \text{ and } V_i = \begin{bmatrix} (\tau + \mu + d) I_E \\ (\delta + \mu + d) I_L - \tau(1 - \rho) I_E \end{bmatrix}$$

The partial derivatives of $\mathcal{F}_i$ with respect to (I_E, I_L) and the Jacobian matrix of $\mathcal{F}_i$ at the disease – free equilibrium point is:

$$F = \begin{bmatrix} \beta & \beta \\ 0 & 0 \end{bmatrix}$$

Similarly, the partial derivatives of V_i with respect to (I_E, I_L) and the Jacobian matrix of V_i is:

$$V = \begin{bmatrix} (\tau\rho + \tau(1-\rho) + \mu + d) & 0 \\ -\tau(1-\rho)\delta + \mu + d & \end{bmatrix}$$

The inverse of the matrix V is given as:

$$V^{-1} = \begin{bmatrix} \frac{1}{(\tau\rho + \tau(1-\rho) + \mu + d)} & 0 \\ \frac{\tau(1-\rho)}{(\tau\rho + \tau(1-\rho) + \mu + d)(\delta + \mu + d)} & \frac{1}{\delta + \mu + d} \end{bmatrix}$$

Thus;

$$FV^{-1} = \begin{bmatrix} \frac{\beta}{\tau\rho + \tau(1-\rho) + \mu + d} + \frac{\beta\tau(1-\rho)}{\delta + \mu + d} \frac{\beta}{\delta + \mu + d} \\ 0 \qquad\qquad 0 \end{bmatrix}$$

The basic reproduction number (R_0) is the dominant eigenvalue or spectral radius $\rho(FV^{-1})$. Thus,

$$R_0 = \frac{\beta}{\tau\rho + \tau(1-\rho) + \mu + d} + \frac{\beta\tau(1-\rho)}{\delta + \mu + d}$$

$= \frac{\beta(\delta+\mu+d)+[\tau(1-\rho)(\tau+\mu+d)]}{(\tau+\mu+d)(\delta+\mu+d)}$

3.2 Existence of Steady (Equilibrium) states

Here we would like to know what would happen to the disease, IBD in a long run: will it die out or will it establish itself in the poultry population and become endemic? In order to answer these questions, we have to investigate the long – term behaviour of the solutions to the model. This behaviour largely depends on the equilibrium points that are time independent solutions of the system. Since these solutions do not depend on time, we set

$$\frac{ds}{dt} = \frac{dI_E}{dt} = \frac{dI_L}{dt} = \frac{dR}{dt} = 0$$

Hence we get the system of equations

$$bN - \beta\left(\frac{I_E+I_L}{N}\right)S - \mu S + \nu R = 0 \quad \text{(i)}$$

$$\beta\left(\frac{I_E+I_L}{N}\right)S - \tau\rho I_E - \tau(1-\rho)I_E - (\mu+d)I_E = 0 \quad \text{(ii)}$$

$$\tau(1-\rho)I_E - \delta I_L - (\mu+d)I_L = 0 \quad \text{(iii)} \qquad (4.1)$$

$$\tau\rho I_E + \delta I_L - \upsilon R - \mu R = 0 \quad \text{(iv)}$$

From (i)

$bN - \beta\left(\frac{I_E+I_L}{N}\right)S - \mu S + \nu R = 0 \Rightarrow S^* = \frac{b+\nu R^*}{\lambda^*+\mu}$ (v)

where $\lambda^* = \beta\left(\frac{I_E^*+I_L^*}{N}\right)$

From (ii)

$$\lambda S - (\tau\rho I_E + \tau(1-\rho)I_E + \mu + d)I_E = 0$$

$\Rightarrow S^* = \frac{(\tau\rho+\tau(1-\rho)+\mu+d)I_E^*}{\lambda^*} = \frac{kI_E^*}{\lambda^*}$ (vi)

with $k = \tau\rho + \tau(1-\rho) + \mu + d$

From (iii) $\tau(1-\rho)I_E - \delta I_L - (\mu+d)I_L = 0 \Rightarrow I_L^* = \frac{\tau(1-\rho)I_E^*}{(\delta+\mu+d)}$

From (v) and (vi)

$\frac{bN+\nu R^*}{\lambda+\mu} = \frac{(\tau\rho I_E^*+\tau(1-\rho)I_E^*+\mu+d)I_E^*}{\lambda} \Rightarrow R^* = \frac{(\lambda+\mu)kI_E^*-\lambda b}{\lambda\nu}$ (vii)

From (iv) $\tau\rho I_E + \delta I_L - \upsilon R - \mu R = 0 \Rightarrow R^* = \frac{\tau\rho k I_E^* + \delta I_E^*}{\upsilon+\mu}$ (viii)

From (vii) and (viii) $\frac{(\lambda+\mu)kI_E^*-\lambda b}{\lambda\nu} = \frac{\tau\rho I_E^* + \delta I_L^*}{\upsilon+\mu} \Rightarrow I_E^* = \frac{\lambda[\nu\delta I_L^*+(\nu+\mu)b}{(\nu+\mu)[(\lambda+\mu)k]-\lambda\nu\tau\rho}$

The steady states are:

$S^* = \frac{b+\nu R^*}{\lambda^*+\mu}$, $I_E^* = \frac{\lambda[\nu\delta I_L^*+(\nu+\mu)b]}{(\nu+\mu)[(\lambda+\mu)k]-\lambda\nu\tau\rho}$, $I_L^* = \frac{\tau(1-\rho)I_E^*}{(\delta+\mu+d)}$, $R^* = \frac{(\tau\rho k+\delta)I_E^*}{\upsilon+\mu}$

Setting $I_E^* = I_L^* = 0$, we obtain the disease - free equilibrium (DFE)

$\varepsilon_0 = (S^0, I_E^0, I_L^0, R^0)$

$\varepsilon_0 = (\frac{bN}{\mu}, 0, 0, 0)$

3.3 Local Stability of DFE

Here we look at the stability of the disease – free equilibrium (DFE) of the system for infectious Bursal disease model (3.1a).

Theorem 3.1: The disease free steady (equilibrium) state of the IBD modelislocally asymptotically stable if and only if (i) $b < \mu$ and (ii)

$\beta < \tau + \mu + d$

Proof: The Jacobian matrix of the system (3.1a) is given as

$$J = \begin{pmatrix} b-\beta\left(\frac{I_E+I_L}{N}\right)-\mu & b-\beta\frac{S}{N} & b-\beta\frac{S}{N} & \nu \\ \beta\left(\frac{I_E+I_L}{N}\right) & \beta\frac{S}{N}-(\tau+\mu+d) & \beta\frac{S}{N} & 0 \\ 0 & \tau(1-\rho) & -(\delta+\mu+d) & 0 \\ 0 & \tau\rho & \delta & -(\nu+\mu) \end{pmatrix}$$

Evaluating the Jacobian at the DFE we have

$$J_{DFE} = \begin{pmatrix} b-\mu & b-\beta & b-\beta & 0 \\ 0 & \beta-(\tau+\mu+d) & \beta & 0 \\ 0 & \tau(1-\rho) & -(\delta+\mu+d) & 0 \\ 0 & \tau\rho & \delta & -(\nu+\mu) \end{pmatrix}$$

The eigenvalues of the matrix $J\varepsilon_0$ is obtained considering the determinant,

$$\left|J_{DFE}-\lambda\right| = \begin{vmatrix} b-\mu-\lambda & b-\beta & b-\beta & 0 \\ 0 & \beta-(\tau+\mu+d)-\lambda & \beta & 0 \\ 0 & \tau(1-\rho) & -(\delta+\mu+d)-\lambda & 0 \\ 0 & \tau\rho & \delta & -(\nu+\mu)-\lambda \end{vmatrix} = 0$$

The matrix has eigenvalues

$\lambda_1 = b-\mu$, $\lambda_2 = \beta-(\tau+\mu+d)$, $\lambda_3 = -(\delta+\mu+d)$, $\lambda_4 = -(\nu+\mu)$

For local asymptotic stability we require $\lambda_1, \lambda_2 < 0$, that is, $b-\mu < 0$ and

$\beta-(\tau+\mu+d) < 0$

Hence the disease free steady state is locally asymptotically stable only if

(i) $b-\mu < 0 \Rightarrow b < \mu$ and

(ii) $\beta-(\tau+\mu+d) < 0 \Rightarrow \beta < \tau+\mu+d$ ■

That is, for the disease-free steady state to be stable, $b < \mu$ and $\beta < \tau+\mu+d$. This means that for the disease to be under control and eradicated within a while from its outbreak, the per capita birth rate of bird (b) should be less than the death rate (μ) and the contact rate with infectious birds (β) less than the sum of the progression rate from early to latter stage of the infection and the death rates ($\tau+\mu+d$).

3.4 Local stability of endemic equilibrium (EE)

Here we consider a pure endemic case where the disease is prevalent in the bird population. This means re- infection will keep occurring which makes recovery impossible and the non – existence of the susceptible. That is $S^* = R^* = 0$.

Setting $S^* = R^* = 0$, we obtain the endemic equilibrium (EE)

$\varepsilon^* = (S^*, I_E^*, I_L^*, R^*)$

$\varepsilon^* = (0, \frac{\lambda[\nu\delta I_L^* + (\nu+\mu)b]}{(\nu+\mu)[(\lambda+\mu)k]-\lambda\nu\tau\rho}, \frac{\tau(1-\rho)I_E^*}{(\delta+\mu+d)}, 0)$

Theorem 3.2: The endemic Steady (Equilibrium) state of the IBD model is locally asymptotically stable if $\beta > b-\mu$.

Proof: Evaluating the Jacobian matrix of the model at the Endemic equilibrium (EE) we have

$$J_{EE} = \begin{pmatrix} b-\beta-\mu & b & b & \nu \\ \beta & -(\tau+\mu+d) & 0 & 0 \\ 0 & \tau(1-\rho) & -(\delta+\mu+d) & 0 \\ 0 & \tau\rho & \delta & -(\nu+\mu) \end{pmatrix}$$

Considering the determinant

$$\left|J_{EE}-\lambda\right| = \begin{vmatrix} b-\beta-\mu-\lambda & b & b & 0 \\ 0 & -(\tau+\mu+d)-\lambda & 0 & 0 \\ 0 & \tau(1-\rho) & -(\delta+\mu+d)-\lambda & 0 \\ 0 & \tau\rho & \delta & -(\nu+\mu)-\lambda \end{vmatrix} = 0$$

The matrix has eigenvalues

$\lambda_1 = b-\beta-\mu$, $\lambda_2 = -(\tau+\mu+d)$, $\lambda_3 = -(\delta+\mu+d)$ and $\lambda_4 = -(\nu+\mu)$

Hence the endemic steady state is stable if;

$b-\beta-\mu < 0 \Rightarrow b - \mu < \beta \Rightarrow \beta > b-\mu$ ■

This means that if the contact rate of infectious birds (β) with the susceptible birds is greater than or equal to the difference between the birth rate (b) and the natural death rate (μ) of the birds, the disease will be endemic.

3.5 Global stability of the DFE

Theorem 3.3: The DFE,ε_0, of the model (3.1a) is globally asymptotically stable if $R_0 \leq 1$.

Proof: We consider the Lyaponuv function

$$L = C_1 I_E + C_2 I_L$$

where $C_1 = \frac{k_2+\tau(1-\rho)}{k_1 k_2}$ and $C_2 = \frac{1}{k_2}$

Thus we have

$$L = \frac{k_2+\tau(1-\rho)}{k_1 k_2} I_E + \frac{1}{k_2} I_L$$

With Lyaponuv derivative given by

$$\dot{L} = \frac{k_2+\tau(1-\rho)}{k_1 k_2} \dot{I_E} + \frac{1}{k_2} \dot{I_L}$$

$$= \frac{k_2+\tau(1-\rho)}{k_1 k_2} [\beta \left(\frac{I_E+I_L}{N}\right) S] - k_1 I_E] + \frac{1}{k_2} [\tau(1-\rho) I_E - k_2 I_L]$$

$$= \frac{k_2+\tau(1-\rho)}{k_1 k_2} [\beta \left(\frac{I_E+I_L}{N}\right) S] - \frac{[k_2+\tau(1-\rho)]I_E}{k_2} + \frac{\tau(1-\rho)I_E}{k_2} - I_L$$

$$= \frac{k_2+\tau(1-\rho)}{k_1 k_2} [\beta \left(\frac{I_E+I_L}{N}\right) S] - I_E - I_L$$

$$= R_0 (I_E + I_L) \frac{S}{N} - (I_E + I_L)$$

Since $S \leq N = > \frac{S}{N} \leq 1$ in the domain that forms the invariant set, it then follows that,

$$\dot{L} \leq R_0 (I_E + I_L) - (I_E + I_L)$$

$$= (R_0 - 1)(I_E + I_L)$$

Therefore $\dot{L} \leq 0$ for $R_0 \leq 1$ and $\dot{L} = 0$ if and only if $I_E = I_L = 0$. Furthermore, $(S^*, I_E^*, I_L^*, R^*) \rightarrow (\frac{bN}{\mu}, 0,0,0)$ as $t \rightarrow \infty$, since $I_E \rightarrow 0$ as $t \rightarrow \infty$ and $I_L \rightarrow 0$ as $t \rightarrow \infty$. Consequently, the largest invariant set in $\{(S, I_E, I_L, R) \in D: \dot{L} = 0\}$ is the singleton $\{\varepsilon_0\}$ and by Lassalle's invariant principle ε_0 is globally asymptotically stable in D if $R_0 \leq 1$ ■

The epidemiological implication of the above result is that infectious Bursal disease elimination is possible irrespective of the initial sizes of the sub-populations of the model whenever the threshold parameter, R_0, is less than or equal to unity.

3.6 Global stability of the endemic equilibrium

We consider a special case where the disease – induced death rate (d) are assumed to be negligible and are set to zero. That is d = 0. Under this setting, the rate of change of the total population is given by

$$\frac{dN}{dt} = b - \mu N$$

Hence, $N \rightarrow (b/\mu)$ as $t \rightarrow \infty$. Thus we set $N(t) = b/\mu$.

Theorem 3.4: The endemic equilibrium of the model (3.1a) is globally asymptotically stable whenever $R_0 > 1$.

Proof: Considering the Goh-Volterra type lyaponuv function, we have

$$L = S - S^{**} - S^{**} In\left(\frac{S}{S^{**}}\right) + \sigma(\beta S - \beta S^{**}) \left[I_E - I_E^{**} - I_E^{**} In\left(\frac{I_E}{I_E^{**}}\right)\right]$$

$$+ \left(\frac{(\beta S^{**} - \beta S)(1-\sigma\beta S)}{\delta+\mu}\right) \left[I_L - I_L^{**} - I_L^{**} In\left(\frac{I_L}{I_L^{**}}\right)\right]$$

where $\sigma = \frac{1-\tau(1-\rho)}{(\delta+\mu)(\beta S - \tau\rho - \mu - \beta S\tau(1-\rho))}$, with Lyaponuv derivative,

$$\dot{L} = (\dot{S} - \frac{S^{**}}{S}\dot{S}) + \sigma(\beta S - \beta S^{**}) \left[\dot{I_E} - \frac{I_E^{**}}{I_E}\dot{I_E}\right] + \left(\frac{(\beta S^{**} - \beta S)(1-\sigma\beta S)}{\delta+\mu}\right) \left[\dot{I_L} - \frac{I_L^{**}}{I_L}\dot{I_L}\right] = b - \beta S \left(\frac{I_E+I_L}{N}\right) - \mu S -$$

$$\frac{S^{**}}{S}(b - \beta S \left(\frac{I_E+I_L}{N}\right) - \mu S) + \sigma(\beta S - \beta S^{**}) \left[\beta S \left(\frac{I_E+I_L}{N}\right) - (\tau\rho + \mu) I_E - \frac{I_E^{**}}{I_E}(\beta S \left(\frac{I_E+I_L}{N}\right) - (\tau\rho+\mu) I_E)\right] +$$

$$\left(\frac{(\beta S^{**} - \beta S)(1-\sigma\beta S)}{\delta+\mu}\right) \left[\tau(1-\rho) I_E - (\delta+\mu) I_L - \frac{I_L^{**}}{I_L}(\tau(1-\rho) I_E - (\delta+\mu) I_L)\right]$$

Simplifying we have

$$\dot{L} = b - \beta S \left(\frac{I_E+I_L}{N}\right) - \mu S - \frac{S^{**}}{S} b + \frac{S^{**}}{S} \beta S \left(\frac{I_E+I_L}{N}\right) + \frac{S^{**}}{S} \mu S + \sigma(\beta S - \beta S^{**}) \left[\beta S \left(\frac{I_E+I_L}{N}\right) - (\tau\rho + \mu) I_E -$$

$$IE{**}IE\beta SIE + ILN + IE{**}IE\tau\rho + \mu IE) + \beta S{**} - \beta S(1-\sigma\beta S)\delta + \mu\tau 1 - \rho IE - \delta + \mu IL - IL{**}IL\tau 1 -$$

$$\rho IE + IL{**}IL\delta + \mu IL)$$

At steady states

$$b = \beta S^{**} \left(\frac{I_E^{**} + I_L^{**}}{N}\right) + \mu S^{**}$$

$$(\tau\rho + \mu) = \beta S^{**} + \frac{\beta S^{**} I_L^{**}}{I_E^{**}}$$

$$\tau(1 - \rho) I_E = (\delta + \mu) I_L^{**}$$

Substituting the values for **b**, $(\boldsymbol{\tau\rho} + \boldsymbol{\mu})and\boldsymbol{\tau(1 - \rho) I_E}$ gives

$$\dot{L} = \beta S^{**}\left(\frac{I_E^{**}+I_L^{**}}{N}\right) + \mu S^{**} - \beta S\left(\frac{I_E+I_L}{N}\right) - \mu S\frac{S^{**}}{S}(\beta S^{**}\left(\frac{I_E^{**}+I_L^{**}}{N}\right) + \mu S^{**}) + \frac{S^{**}}{S}\beta S\left(\frac{I_E+I_L}{N}\right) + \frac{S^{**}}{S}\mu S + \sigma(\beta S - \beta S^{**}\beta S I E + I L N - \beta S^{**} + \beta S^{**} I L^{**} I E^{**} I E - I E^{**} I E \beta S I E + I L N + I E^{**} I E \beta S^{**} + \beta S^{**} I L^{**} I E^{**} I E) + \beta S^{**} - \beta S(1 - \sigma\beta S)\delta + \mu\delta + \mu I L^{**} - \delta + \mu I L - I L^{**} I L\delta + \mu I L^{**} + I L^{**} I L\delta + \mu I L)$$

Simplifying gives

$$\dot{L} = \beta(I_E^{**} + I_L^{**})S^{**} + 2\mu S^{**} - \mu S - \frac{\beta S^{**2}}{S}(I_E^{**} + I_L^{**}) - \frac{\mu S^{**2}}{S} - \sigma(\beta S - \beta S^{**})\frac{I_E^{**}}{I_E}\beta S(I_E + I_L) + \sigma(\beta S - \beta S^{**} I E^{**} \beta S^{**} + \beta S^{**} I L^{**} I E^{**} - \beta S^{**} - \beta S 1 - \sigma\beta S I L^{**} 2 I L + (\beta S^{**} - \beta S)(1 - \sigma\beta S) I L^{**}$$

$$= \mu S^{**}\left[2 - \frac{S}{S^{**}} - \frac{S^{**}}{S}\right] + \beta S^{**} I_E^{**}\left[1 - \frac{S^{**}}{S}\right] + \beta S^{**} I_L^{**}\left[2 - \frac{S^{**}}{S} - \frac{I_L^{**}}{I_L} - \frac{S}{S^{**}} + \frac{S I_L^{**}}{S^{**} I_L}\right] + \sigma\beta^2 S^{**} I_E^{**}\left[2S - \frac{S^2}{S^{**}} - S^{**} - \frac{S^2 I_L^{**}}{S^{**} I_E} + \frac{S I_L^{**}}{I_E}\right] + \sigma\beta^2 S^{**} I_L^{**}\left[\frac{S^2}{S^{**}} - S^{**} - \frac{S^2 I_L^{**}}{S^{**} I_L} + \frac{S I_L^{**}}{I_L}\right]$$

Since the arithmetic mean exceeds the geometric mean, it follows then that

$$\mu S^{**}\left[2 - \frac{S}{S^{**}} - \frac{S^{**}}{S}\right] \le 0, \quad \beta S^{**} I_E^{**}\left[1 - \frac{S^{**}}{S}\right] \le 0,$$

$$\beta S^{**} I_L^{**}\left[2 - \frac{S^{**}}{S} - \frac{I_L^{**}}{I_L} - \frac{S}{S^{**}} + \frac{S I_L^{**}}{S^{**} I_L}\right] \le 0$$

$$\sigma\beta^2 S^{**} I_E^{**}\left[2S - \frac{S^2}{S^{**}} - S^{**} - \frac{S^2 I_L^{**}}{S^{**} I_E} + \frac{S I_L^{**}}{I_E}\right] \le 0$$

And $\sigma\beta^2 S^{**} I_L^{**}\left[\frac{S^2}{S^{**}} - S^{**} - \frac{S^2 I_L^{**}}{S^{**} I_L} + \frac{S I_L^{**}}{I_L}\right] \le 0$

Furthermore, since all the model parameters are non – negative, it follows that $\dot{L} \le 0$ for $R_0 > 1$. Hence L is a Lyaponuv function in D. Also, $\dot{L} = 0$ if and only if $S = S^{**}$, $I_E = I_E^{**}$, $I_L = I_L^{**}$. Hence the largest invariance subset of the set where $\dot{L} = 0$ is the singleton $\{(S, I_E, I_L) = (S^*, I_E^*, I_L^*)\}$. Similarly $R \to \frac{(\tau\rho k + \delta) I_E^*}{\upsilon + \mu} = R^*$ as $t \to \infty$. By Lasalle's invariance principle, it follows that every solution in D approaches $\varepsilon^*$for $R_0 > 1$ as $t \to \infty$. ■

The result above implies that infectious bursal disease will be endemic in the poultry whenever the threshold quantity R_0 is greater than unity.

3.7 Sensitivity analysis or parametric dependence on R_0

Sensitivity analysis tells us how important each parameter is to disease transmission. The sensitivity indices allows the measurement of the relative change in the basic reproduction number (R_0), when a parameter changes. The normalized forward sensitivity index of R_0 with respect to a parameter is the ratio of the relative change in R_0 to the relative change in the parameter. Using the normalized forward sensitivity index we derive an analytical expression for the sensitivity of R_0 with respect to each parameter that comprise it.

Definition 3.1: The normalized forward-sensitivity index of a variable, v, which depends differentiable on a parameter, p, is defined as:

$$r_p^v = \frac{\partial v}{\partial p} \times \frac{p}{v}$$

In particular, sensitivity indices of the basic reproduction number, R_0, with respect to the model parameters are computed as follows:

$$r_\beta^{R_0} = \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} = 1$$

$$r_\delta^{R_0} = \frac{\partial R_0}{\partial \delta} \times \frac{\delta}{R_0} = -\frac{\delta k_1}{(k_2+k_1)k_2}$$

$$r_\tau^{R_0} = \frac{\partial R_0}{\partial \tau} \times \frac{\tau}{R_0} = \frac{\tau[k_3^2(1-\rho)-k_2]}{k_3[k_2+k_1]}$$

$$r_d^{R_0} = \frac{\partial R_0}{\partial d} \times \frac{d}{R_0} = -d\left[\frac{k_2^2+k_1k_3}{(k_3k_2)(k_2+k_1)}\right]$$

$$r_\mu^{R_0} = \frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0} = -\mu\left[\frac{k_2^2+k_2k_3+k_1k_3}{(k_3k_2)(k_2+k_1)}\right]$$

The positive sign of Sensitivity index of the basic reproduction number to the model parameters indicates that an increase (or decrease) in the value of each of the parameter in this category will lead to an increase (or decrease) in the basic reproduction number of the disease. For example, $r_\beta^{R_0} = 1$ suggests that increasing (or decreasing) the contact rate of infectious birds with susceptible birds by 10% increases (or decreases) the basic reproduction number, R_0, by 10%. On the other hand, the negative sign of Sensitivity Index

of the basic reproduction number to the model parameters implies that an increase (or decrease) in the value of each of the parameter in this category leads to a corresponding decrease (or increase) in the basic reproduction number of the disease. Thus, sensitivity analysis of the disease model provides a very good insight into the transmission dynamics of the disease.

3.8 Formulation of optimal control problem

We formulate the objective functional

$$J(u_1, u_2) = \int_0^T (nI_E + mI_L + c_1u_1^2 + c_2u_2^2)dt$$

where n, m, c_1, c_2 are positive weights. We seek an optimal control (u_1^*, u_2^*) such that

$J(u_1^*, u_2^*) = \min \{J(u_1, u_2) : (u_1, u_2) \in u \text{ and } U = \{ (u_1(t), u_2(t)) \mid 0 \leq u_1, u_2 \leq 1, t \in [0, T] \}$

Pontryagin's Maximum Principle

We convert the objective functional minimization problem coupled with the state variable into a problem of minimizing point – wise a Hamiltonian H,

$$H = nI_E + mI_L + c_1u_1^2 + c_2u_2^2 + \lambda_S[b(1-u_1)S - (1-u_2)\beta\left(\frac{I_E+I_L}{N}\right)S - dS + \nu R]$$
$$+ \lambda_E[(1-u_2)\beta\left(\frac{I_E+I_L}{N}\right)S - (\tau\rho + \tau(1-\rho) + \mu)I_E] + \lambda_L[\tau(1-\rho)I_E - (u_2+\mu)I_L]$$
$$+ \lambda_R[\tau\rho I_E + u_2I_L - (\upsilon + d)R]$$

Proposition: For the optimal control (u_1^*, u_2^*) that minimizes $J(u_1, u_2)$ over U, there exist adjoint variables λ_S, λ_E, λ_L and λ_R satisfying the following adjoint system

$$\frac{d\lambda_S}{dt} = -\frac{\partial H}{\partial s} = \lambda_S[bu_1 + (1-u_2)\beta\left(\frac{I_E+I_L}{N}\right) + d] - \lambda_E[(1-u_2)\beta\left(\frac{I_E+I_L}{N}\right)$$

$$\frac{d\lambda_E}{dt} = -\frac{\partial H}{\partial E} = -n + \lambda_S[(1-u_2)\beta\left(\frac{S}{N}\right)] - \lambda_E[(1-u_2)\beta\left(\frac{S}{N}\right) - (\tau\rho + \tau(1-\rho) + \mu)] - \lambda_L(\tau(1-\rho)) - \lambda_R(\tau\rho)$$

$$\frac{d\lambda_L}{dt} = -\frac{\partial H}{\partial L} = -m + \lambda_S[(1-u)\beta\left(\frac{S}{N}\right)] - \lambda_E[(1-u_2)\beta\left(\frac{S}{N}\right) + \lambda_L(u_2+\mu) - \lambda_R u_2$$

$$\frac{d\lambda_R}{dt} = -\frac{\partial H}{\partial R} = \lambda_S\nu + (\nu + d)\lambda_R$$

Transversality conditions

$$\lambda_S(T) = \lambda_E(T) = \lambda_L(T) = \lambda_R(T) = 0$$

The optimal control u_1^* and u_2^* can be solved from optimality conditions

$$\frac{\partial H}{\partial u_1} = 0, \frac{\partial H}{\partial u_2} = 0$$

That is,

$$\frac{\partial H}{\partial u_1} = 2c_1u_1 - \lambda_S bS = 0$$

$$\frac{\partial H}{\partial u_2} = 2c_1u_2 - \lambda_S\beta\left(\frac{I_E+I_L}{N}\right)S + \lambda_E\beta\left(\frac{I_E+I_L}{N}\right)S - \lambda_L I_L - \lambda_R I_L = 0$$

Moreover the optimal control is given by

$$u_1^* = min\left[1, max\left(0, \frac{\lambda_S bS}{2c_1}\right)\right]$$

$$u_2^* = min\left[1, max\left(0, \frac{(\lambda_S - \lambda_E)\left(\beta S\left(\frac{I_E+I_L}{N}\right)\right) + (\lambda_L - \lambda_R)I_L}{2c_2}\right)\right]$$

This implies that the optimal effort necessary to control the disease is

$$u_1^* = \frac{\lambda_S bS}{2c_1} \text{ and } u_2^* = \frac{(\lambda_S - \lambda_E)\left(\beta S\left(\frac{I_E+I_L}{N}\right)\right) + (\lambda_L - \lambda_R)I_L}{2c_2}$$

IV. Numerical Results on poultry productivity in the presence of Infectious Bursal Disease (IBD)

We have presented the results on the poultry productivity in the presence of IBD on the graphs represented in Figures 4.1-4.6. In the graphs so, presented we employed the optimal controls. The first control employed here is the use of vaccines (u_1), and the second control denoted by (u_2) represents the effort of supportive measures like increased heat, good ventilation, and adequate water consumption of poultrybirds, in enhancing the quick recovery of infected birds, since there is no specific treatment made available for the treatment of the disease (Morla et al 2016).

We have considered the following strategies in our simulation

A. The use of vaccine, i.e. the vaccination control measure $u_1(t) \neq 0$, when supportive measures $u_2(t) = 0$
B. Vaccination control measure $u_1(t) = 0$ when supportive measures $u_2(t) \neq 0$
C. Vaccination control , $u_1(t) \neq 0$ and supportive measures $u_2(t) \neq 0$

These strategies, A – C has been combined conveniently and presented for each of the affected compartments in the model as presented in Figures 4.1-4.4 below. The parameter values used in the simulation of the model is presented here on Table 4.1.

Table 4.1: Parameter values for Infectious bursal disease (IBD)

Parameter	Estimated value	Reference
b	40	Estimated
μ	0.02	Bornall et al (2015)
β	0.1	Sharma et al (2015)
d	0.5	Qin & Zheng (2016)
p	0.5	Elizabeth (2016)
τ	0.033	Elizabeth (2016)
ν	0.026	Elizabeth (2016)
δ	0.004	Elizabeth (2016)

The graphs from simulating the model, given in Fig. 1-Fig. 6, help to compare the population of the infected birds in the early (I_E) and latter (I_L) stage of the infection both with controls and without controls.

4.1 Graph of the Simulation

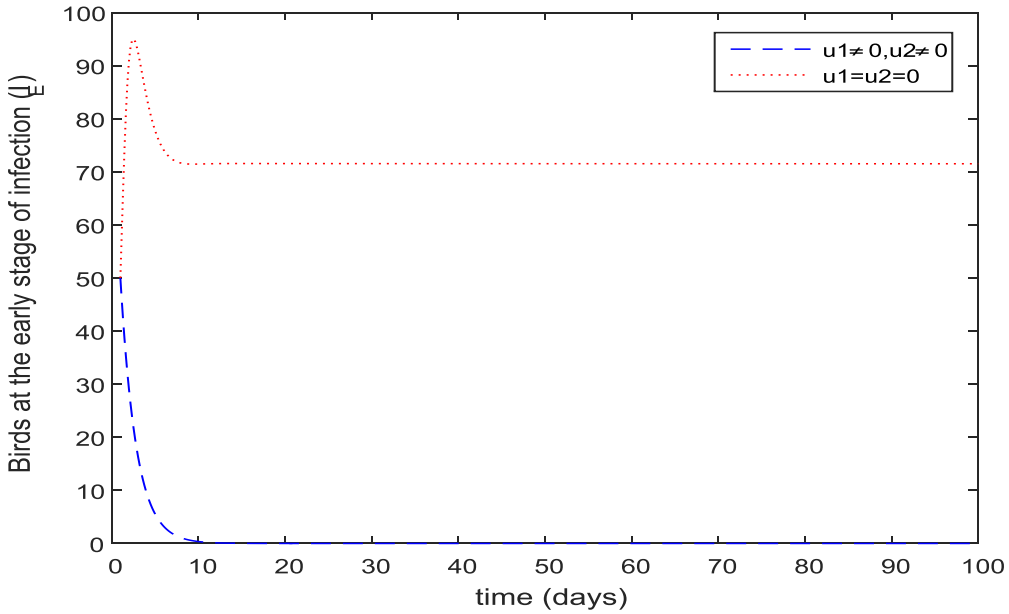

Figure 4.1: A graph showing birds at the early stage of the infection with and without control

From Figure 4.1 we observe that the number of birds at the early stage of the infection increases rapidly and drops to a particular level. Applying the both control measures reduces the number to zero. This strategy shows a significant reduction in the number of birds at the early stage of the infection with control.

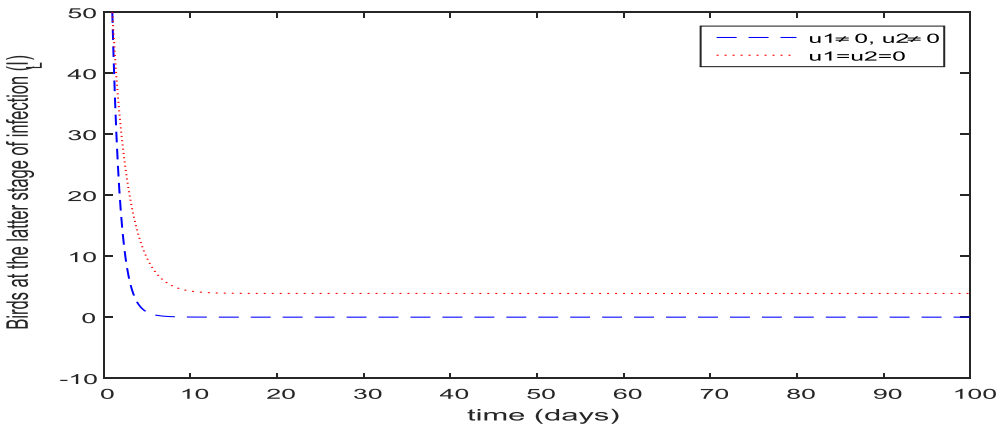

Figure 4.2: A graph showing birds at the latter stage of the infection with and without control

It is observed from figure 4.2 that the number of birds at the latter stage of the infection reduces with and without control. Without control the reduction is as a result of high mortality rate at this stage of the infection. With control the number reduces and drops to zero. Therefore we may conclude that applying both controls the infectious birds will drop to zero over time.

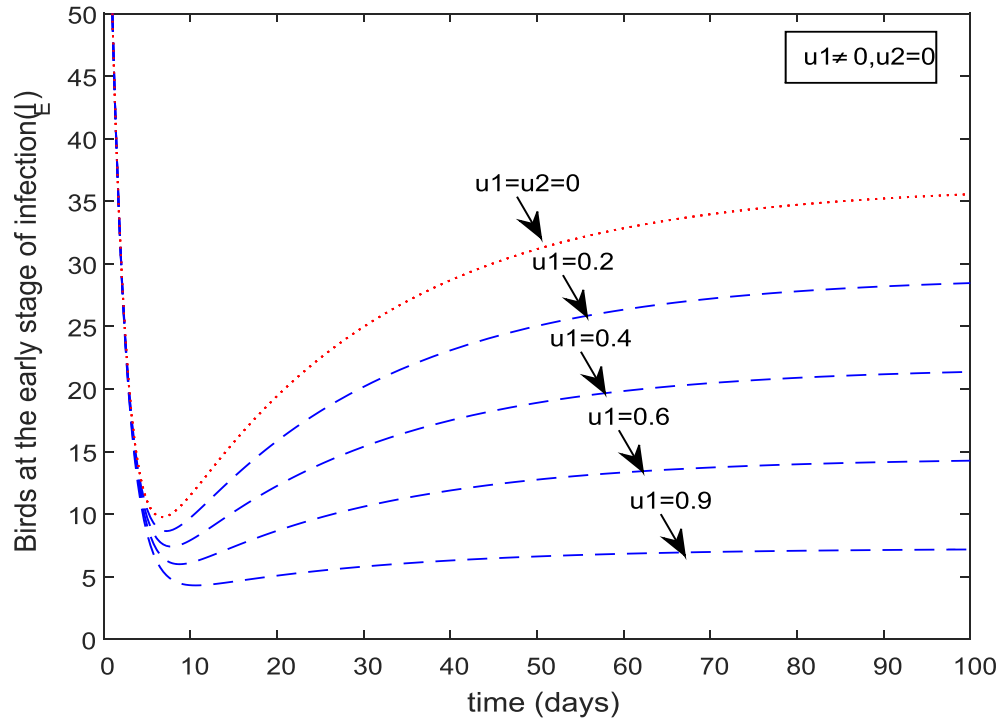

Figure 4.3: A graph showing the effect of only vaccination (ie $u_1 \neq 0$ with $u_2=0$) on birds at the early stage of the Infection.

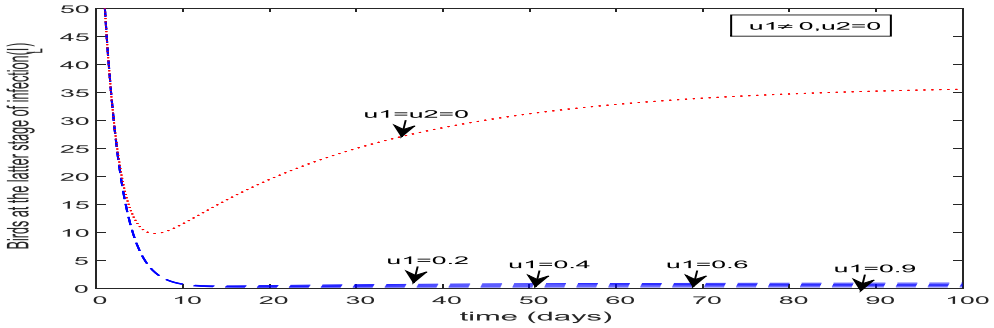

Figure 4.4: A graph showing the effect of only vaccination (ie $u_1 \neq 0$ with $u_2=0$) on birds at the latter stage of the Infection.

We observe from Figure 4.3 that the number of infectious bird will keep reducing as the use of vaccination increases. At the optimal application of vaccines the number of infectious birds becomes less than 5 and remains at that level over time. From Figure 4.4, at the optimal application of only vaccines, the number of birds at the latter stage of the infection will drop to zero. This implies that strategy B (that is, when supportive measures to enhance recovery infectious birds are not applied) will be more effective on the infectious birds at the latter stage of the infection than on birds at the early stage of the infection.

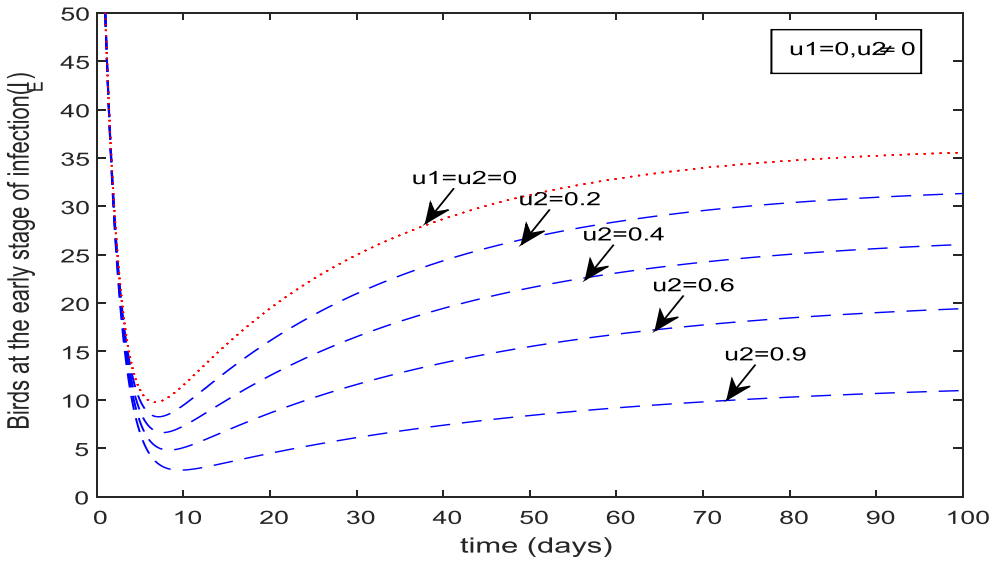

Figure 4.5: A graph showing the effect of only supportive measures (ie$u_1=0$ with $u_2 \neq 0$) on birds at the earlystage of the infection

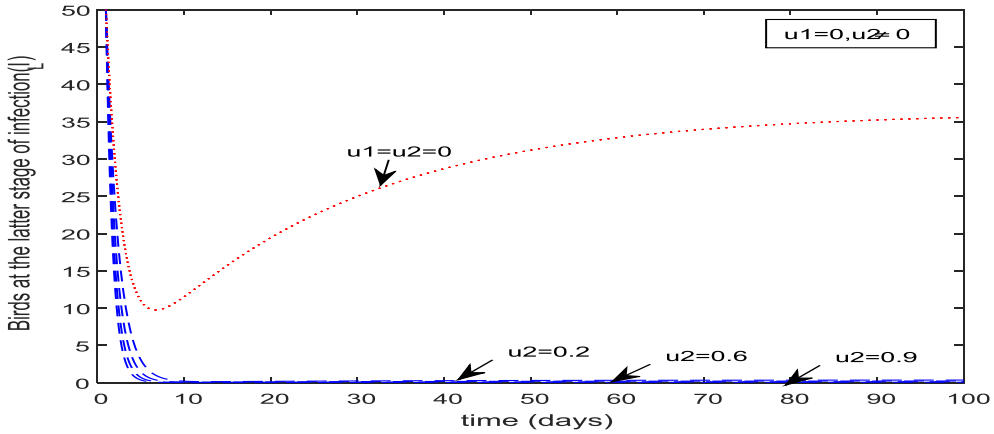

Figure 4.6: A graph showing the effect of only supportive measures (ie$u_1=0$ with $u_2 \neq 0$) on birds at the latterstage of the infection

We observe that from Figure 4.5, that the number of birds at the early stage of the infection will also reduce when only supportive measures are applied but will increase the number of infectious birds gradually after about 10 days. When only supportive measures are applied in $I_{L,}$ we see from Figure 4.6 that the number drops to zero faster even at the early application of the strategy. Hence we can deduce that this strategy is more effective on birds at the latter stage of the infection and less effective on birds at the early stage of infection.

5.1 Summary

In this thesis, we considered the theoretical analysis of compartmental Infectious bursal disease (IBD).The study is briefly summarised below;

Firstly, stability analysis was carried out using the Lyaponuv function theory and Lasslle's invariance principle for each of these disease model.. Subsequently optimal control problems were formulated for the control models and was analysed using the pontryagin's maximum principle. Sensitivity analysis was also carried out to find out how important each model parameters are to the disease transmissions. This was done using the normalized forward- sensitivity index.

5.2 Conclusion

In the case of Infectious bursal disease (IBD), we developed a relatively simple but reliable model which we analysed. From the stability analysis, the DFE and EE will be locally asymptotically stable if (i) $b < \mu$ and (ii) $\beta < \tau + \mu + d$ and if $\beta > b - \mu$ respectively. Using the lyaponuv function we established the global stability of the model. The DFE and EE were established to be globally asymptotically stable if the threshold quantity $R_0<1$, and $R_0>1$, respectively.

In the control model, we employed two strategies as control measures which are, the vaccination control (u_1) and supportive measures (u_2). The numerical results established that the combination of vaccines and supportive measures will reduce the incidence rate of the disease to zero over time, but where only supportive measures are carried out, without vaccination, the disease will still persist in the poultry.

5.3 Recommendations

The work was motivated by the possibility that mathematical modelling could improve the understanding of the dynamics of these diseases, particularly the impact of infection on poultry productivity. Based on the analysis of this study, we can conclude that poultry productivity can still be achieved even in the presence of perverse disease outbreak, if appropriate control measures are applied. Hence we recommend that control programs that follow the strategies stated for each of the diseases in this study, can be used effectively to prevent and reduce the spread of these diseases, in order to enjoy high poultry productivity in our poultry industries.

References

[1]. Abid, A. L. (2016). Optimal control of a SIR Epidemic Model with a saturated treatment; Journal of Applied Mathematics and Information Sciences, 10, No 11, 185 – 191.

[2]. Boot, H. J., Hoekwan A. J., Giekers A. I. (2005) The enhanced virulence of very virulent infectious Bursal Disease Virus partly determined by its B – Segment. Archive Virology, 150: 137 – 144.

[3]. Candelora, K. L., Spalding M. G., Sellers H. S. (2010). Survey for antibodies to Infectious BursalDisease Virus Sertyoe 2 in wild Turkeys and Sand hill cranes of Florida USA. Journal of Wild life Diseases 46(3) : 742-752. doi : 10.7589/0090-3558-46.3.742.

[4]. Chung-Chai, H., Tsan – Yuk L., Alexei D., Andrew R., Yiu – Fai L., Chai – Wai Y., Fanya Z., Pui Yi L., Patrick T., Frederick C. L. (2006). Phylogenetic Analysis reveals a correlation between the Expansion of very Virulent Infectious Bursal Disease Virus and reassortment of its Genome Segment. Journal of virology, Vol. 80, No. 17, pp 8503 – 8509.

[5]. Durairaj, V., Linnemann e., Icand A. H., Williams S. M., Sellers H. S., Mundt E. (2013). An in vivo experimental model to determine antigenic variations among Infectious Bursal Disease Virus. Journal of Avian Pathology 42(4) : 309 – 315. doi:10:1080/03079457-2013-793783

[6]. Lasher, H. N, Davis V. S. (1997) History of Infectious bursal disease in USA – the first two decades, Avian Disease, 41: 11 – 19

[7]. Li, Z., Qi, X., Ren L. C., Wang X., Zhu P. (2015). Molecular characteristics and evolutionary analysis of a very virulent infectious bursal disease virus. Science China Life Sci. 58, 731-738.

[8]. Oluwayelu, D. O., Adeiyi A. I., Olaniyian I., Ezewele P., Oluwasanmi A. (2014). Occurrence of Newcastle disease and infectious bursal disease virus antibodies in double – spurred Francolins of Nigeria. Journal of veterinary medicine106898, Doi: 10.1155/2014/106898.

[9]. Raja, P., Senthilkumar M. A., Parthiban M., Thangavelu A., Mangala A., Palanisammi A., Kumar K (2016). Complete Genome sequence Analysis of a naturally Reassorted Infectious Bursal Diasese Virus from India. American Society for Microbiology, Article in Genome Announcements, vol. 4, Issue 4, pp 709 – 716.

[10]. Sharomi, O., Malik T. (2015). Optimal control in epidemiology. Annals of operations Research, pp. 1-17. doi: 10.1007/s10479-015-1834-4.

[11]. Snyder, D. B., Vakharia V. N., Lutticken D., Mengelwherat S.A., Savage P.k., Edwards G (1994). Active and passive protection against variant and classic Infectious Bursal Disease Virus – strains induced by Baculovirus – Expressed structural proteins.Vaccine 12(5) : 452 – 456.

[12]. Stoute, S.T., Jackwood D. J., Summer – Wagner S. E., Crossley B. M., Woolcock P. R., Charlton B. R. (2013). Pathogenicity associated with coinfection with very virulent infectious bursal disease and infectious bursal disease virus strains endemic in the United States. Journal of Veterinary Diagnostic Investigation, 25, 352-358.

[13]. Sudhir, M., Deka P., Kumar S. (2016). Isolation of novel variants of infectious bursal disease virus from different outbreaks in Northeast India; Microbial pathogenesis, 93, 131-136.
[14]. Sufen, Z., Yuanyuan J., Deping H., Haiyan M., Syed Z., Ali S., Yunfei M., Kedao T. (2016) Influence of structural development of bursa on the susceptibility of chickens to Infectious Bursal Disease virus, Poultry Science, 0: 1-.
[15]. Van den Berg, T.(2000). Acute Infectious bursal disease in Poultry a review. Avian Pathology 29(3): 175 -190.

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