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Age-Infection Model and Control of Marek Disease

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Abstract: We formulated three compartmental model of Marek Disease model. 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 Marek disease and we drew five graphs to demonstrate this. The first figure shows the effect of both vaccination (v) and biosecurity measures (u) on the latently infected birds. The population of infected birds increases speedily and then remains stable without the application of any control measure, with the controls, the population increases to about 145 and then begins to reduce from day 8 till it drops to 50 on day 20 and then remains stable. With this strategy, only bird vaccination (*v)* is applied to control the system while the other control is set to zero. In the second figure, the effect of bird vaccination and its' positive impact is revealed, though there is an increase to about 160 before a decrease occurs. From the third figure, as the control (u) ranges from 0.2 to 0.9, we see that the bird population still has a high level of latently infected birds. This result from figure shows that the bird population is not free from the disease, hence, the biosecurity control strategy is not effective without vaccination of susceptible birds and hence it is not preferable as the only control measure for marek disease. The numerical result in the fourth figure shows that as the latently infected bird population increases without control, with vaccination it decreases as more susceptible birds are vaccinated. From the fifth figure we observe, that as the control parameter increases, the total deaths by infection reduces, also as the age of the infection increases to the maximum age of infection which is 6 months (thatis, T=24 weeks), the number of deaths increases to 30 in a day. Hence, control measures should be applied at the early ages of infection in order to avoid high mortality rate during the outbreak of the disease.

Keywords: Age-Infection Model, Marek Disease, Biosecurity Control Strategy, Vaccination, Compartmental Model of Marek Disease

1. Introduction

Marek's disease is a viral infection that only affects poultry caused by alphaherpesvirus known as 'Marek's disease virus' (MDV) or Gallid herpesvirus 2 (GaHV-2). It is a disease that affects mostly young chickens but older birds can also be affected, [4]. Birds infected with GaHV-2 can be carriers and shedders of the virus for life. Newborn chicks are protected by maternal antibodies for a few weeks. After infection, microscopic lesions are present after one to two weeks, and gross lesions are present after three to four weeks. The virus is spread in dander from feather follicles and transmitted by inhalation, [7]. It lives in the environment for long periods, and can spread between properties on people's clothes and on shared equipment. Birds are usually infected at a young age from two to five months old, but may not show signs of disease until some months later.

Marek's disease is a highly contagious viral neoplastic

disease in chickens. It is named after József Marek, a Hungarian veterinarian and often occasionally misdiagnosed as tissue pathology. It is caused by an alphaherpes virus known as Marek's disease virus' (MDV) or *Gallidherpesvirus2* (GaHV-2). The disease is characterized by the presence of Tcelllymphoma as well as infiltration of nerves and organs by lymphocytes, [7].

The vaccine was introduced in 1970 and the scientist credited with its development is Dr. Ben Roy Burmester. Before that, Marek's disease caused substantial revenue loss in the poultry industries of the United States and the United Kingdom. Diagnosis of lymphoid tumors in poultry is complicated due to multiple etiological agents capable of causing very similar tumors. It is not uncommon that more than one avian tumor virus can be present in a chicken, thus one must consider both the diagnosis of the disease/tumors (pathological diagnosis) and of the virus (etiological diagnosis). A step-wise process has been proposed for diagnosis of Marek's disease which includes (1) history, epidemiology, clinical observations and grossnecropsy, (2) characteristics of the tumor cell, and (3) virological characteristics, [31].

To understand the mechanisms of this virulence evolution and to evaluate the epidemiological consequences of putative control strategies, it is imperative to understand how virulence is defined and how this correlates with host mortality and infectiousness during MDV infection. It was considered in [15] a mathematical approach to quantify key epidemiological parameters. Host life span, virus latent periods and host viral sheddin grates were estimated for unvaccinated and vaccinated birds, infected with one of three MDV strains. The strains had previously been pathotyped to assign virulencescores according to pathogenicity of strains in hosts. The analyses show that strains of higher virulence have a higher viral shedding rate, and more rapidly kill hosts. Vaccination enhances host life expectancy but does not significantly reduce the shedding rate of the virus. While the primary latent period of the virus does not vary with challenge strainn or vaccine treatment of host, the time until the maximum viral shedding rate is increased with vaccination. In [2], it was formulated a mathematical model to predict the effectiveness of vaccines to reduce the outbreak probability and disease burden with in a barn. They found that the chance of an outbreak with in a barn increases with the virulence of an MDV strain, and is significantly reduced when the flock is vaccinated, especially when there is a the contaminant strainis of low virulence. With low quantities of contaminated dust, there is nearly a 100% effectiveness of vaccines to reduce MDV out breaks. However, the vaccine effectiveness drops to zero with an increased amount of contamination with a middle virulence MDV strain. It was predicted that the larger the barn, and the more virulent the MDV strain is, the more virus is produced by the time the flock is slaughtered. With the low-to-moderate virulence of the strains studied here, the number of deaths due to MDV was very low compared to all-cause mortality regardless of the vaccination status of the birds. However, the cumulative MDV incidence can reach 100% for unvaccinated cohorts and 35% for vaccinated cohorts. The results suggest that death due to MDV is an insufficient metric to assess the prevalence of MDV broiler barns regardless of vaccine status, such that actives urveillance is required to successfully assess the probability of MDV out breaks, and to limit transmission of MDV between successive cohorts of broiler chickens.

In her work [5] investigates how modern broiler farm practices can maked is ease eliminationd if ficultorim possible to achieve using an implusive differential equation. The study also investigated factors that may contribute to virulence evolution. The model suggests that by decreasing the cohort duration or by decreasing the flock density, Marek's disease can be eliminated from a barn with no increase in cleaning effort. The model also suggests that the sepractices will lead to disease evolution towards greater virulence. Additionally, it suggests that if intensive cleaning between cohorts does not rid the barn of disease, it may drive evolution and cause the disease to become more virulent.
It was developed in [19] a genetic–epidemiological model for Marek disease infection in poultry and assess the impact of genetic and vaccination strategies on over all MDV dynamics. A compartmental model considering susceptible, exposed, cytolyticphase1, latent cytoliticphase 2, proliferatives phases of MDV infection was developed and simulated stochastically in a population of 10,000 birds for 500 days. The

Result showed that the basic reproductive ratio and percent of the population infected in a MDV epidemic were 5.8 and 0% and 0.6 and 20% for unvaccinated and vaccinated cases respectively. The model outcome correctly identify that whilst the proportion of infected individuals in a genetically resistant population may be high, the incidence of disease wil lstill be rare, since infection may rare cause obvious clinical cases of disease. It is important to note that in the studies mentioned above, as well as in most other treatment scheduling studies, the age structure of the infection of the disease was not taken into consideration. In an attempt to give a more substantial control strategy of the disease, an age-structure model on the disease transmission is being considered in this study. A paper by [22] investigates the effectiveness and cost-effectiveness of leptospirosis control measures, preventive vaccination and treatment of infective humans that may curtail the disease transmission. For this, a mathematical model for the transmission dynamics of the disease that includes preventive, vaccination, treatment of infective vectors and humans control measures was considered. Firstly, the constant control parameters' case was analyzed, the basic reproduction number was calculated and the existence and stability of equilibria was investigated. The threshold condition for disease-free equilibrium was found to be locally asymptotically stable and can only be achieved when the basic reproduction number is less than unity. The model was found to exhibit the existence of multiple endemic equilibria. Other works looked at include the following: [6, 14, 16-18, 20, 21 and 23]. Other papers consolted and used include: [1, 3, 7-13, 24-30 and 31].

2. Method

2.1. Assumptions of the Model

1. Age–dependent birth rates does not dependon disease status
2. Allnewbornsareinthesusceptibleclass(ieallyoungbirdsare thesusceptibleclass)
3. There is intercohort mixing (i.e. infection can be transmitted between birds of different ages)

Table 1. *Parameters of the Model.*

S(a, t)	susceptible of age a at time t
I(a, t)	infectives of age a at time t
C(a, t)	carriers of age a at time t
λ(a, t)	Infection rate
δ(a)	removal rate in each class
d(a)	natural death rate in each class
μ(a)	Age-based, disease-induced death rate
Λ(a, t)	Recruitment rate of birds of age a at time t

2.2. Model Flow Chart

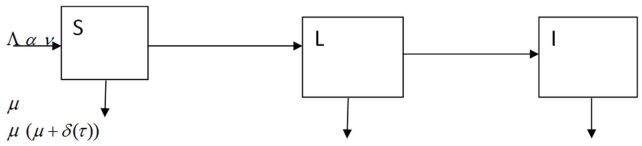

Figure 1. *Flow Diagram for Marek Disease.*

2.3. Equations of the Model

$$\left.\begin{aligned}
&\frac{\partial S}{\partial t}+\frac{\partial S}{\partial a}=\Lambda(a,t)-\lambda(a,t)S(a,t)-d(a)S(a,t)\\
&\frac{\partial I}{\partial t}+\frac{\partial I}{\partial a}=\lambda(a,t)S(a,t)-d(a)I(a,t)-\mu(a)I(a,t)-\delta(a)I(a,t)\\
&\frac{\partial C}{\partial t}+\frac{\partial C}{\partial a}=\delta(a)I(a,t)-d(a)C(a,t)-\mu(a)C(a,t)\\
&Initial\ condition\\
&S(a,0)=S_0(a)\ , I(a,0)=I_0(a),\ R(a,0)=R_0(a)\\
&N(a,t)=S(a,t)+I(a,t)+C(a,t)
\end{aligned}\right\} \quad (1)$$

3. Results

3.1. Existence of Steady State for the Marek Disease Model

In corporating the new births from each class, the model equation becomes

$$\left.\begin{aligned}
&\frac{dS(t)}{dt}=\Lambda[S(t)+L(t)+I(t)]-\alpha S(t)I(t)-\mu S(t)\\
&\frac{dL(t)}{dt}=\alpha S(t)I(t)-(\gamma+\alpha)L(t)\\
&\frac{\partial i(t,\tau)}{\partial t}+\frac{\partial i(t,\tau)}{\partial \tau}+(\mu+\delta(\tau))i(t,\tau)=0,\ with\ \ initial
\end{aligned}\right\}, \quad (2)$$

$$With\ \delta(\tau)=\delta e^{-k(T-\tau)} \quad (3)$$

$$I(t)=\int_0^T i(t,\tau)d\tau, \quad 0\le\tau\le T \quad (4)$$

With initial condition,

$$i(t,0)=B(t)=\gamma L(t)-\Omega I(t) \quad (5)$$

At the equilibrium states, let

$$S(t)=x, L(t)=y,\ I(t)=z,\ i(t,\tau)=\varphi(\tau) \quad (6)$$

So that,

$$z=\int_0^T \varphi(\tau)d\tau \ \ and \ \ \varphi(0)B(t)=\gamma y-\Omega z \quad (7)$$

Substituting (4) and (5) into the RHS of (1) at equilibrium states gives

$$\Lambda(x+y+z)-\alpha xz-\mu x=0 \quad (8)$$

$$\alpha xz-(\gamma+\mu)y=0 \quad (9)$$

$$\frac{\partial\varphi(\tau)}{\partial\tau}+h(\tau)\varphi(\tau)=0 \quad (10)$$

where

$$h(\tau)=\mu+\delta(\tau)=\mu+\delta e^{k(T-\tau)} \quad (11)$$

Solving (10) gives

$$\varphi(\tau)=\varphi(0)\exp\left\{-\int_0^\tau h(s)\right\} \quad (12)$$

$$\text{Let } \pi(\tau)=\exp\{-\textstyle\int_0^\tau h(s)ds\} \quad (13)$$

So that (12) becomes

$$\phi(\tau)=\phi(0)\pi(\tau) \quad (14)$$

Putting (14) in (8) gives

$$z=\textstyle\int_0^T \phi(0)\pi(\tau)d\tau=\phi(0)\{\int_0^\tau \pi(\tau)d\tau+\int_\tau^T \pi(\tau)d\tau\}=\phi(0)\int_\tau^T \pi(\tau)d\tau$$

$$z=\phi(0)\bar{\pi}, \text{ where } \bar{\pi}=\textstyle\int_\tau^T \pi(\tau)d\tau \quad (15)$$

Using (7) in (14) gives

$$z=\left(\gamma\, y-\Omega z\right)\pi \quad (16)$$

Solving (7), (8) and (16) simultaneously for (x, y, z) gives

the steady states for the Marek model,

That is,

$$\Lambda (x+y+z)- \alpha xz- \mu x=0 \quad (17)$$

$$\alpha xz-(\gamma +\mu) y=0 \quad (18)$$

$$z - (\gamma y - \Omega z)\bar{\pi}=0 \quad (19)$$

$$\text{From (19), } z = \frac{\gamma y \bar{\pi}}{1+\Omega \bar{\pi}} \quad (20)$$

Thus, substituting (18) in (17) gives

$$x = \frac{(\gamma + \mu)(1+\Omega \bar{\pi})}{\alpha \gamma_{\bar{\pi}}} \quad (21)$$

Substituting (20) and (21) in (17), gives,

$$y = \frac{(\Lambda+\mu)(\gamma+\mu)(1+\Omega\bar{\pi})^2}{\alpha\gamma\bar{\pi}[(1+\Omega\bar{\pi})(\gamma+\mu+\Lambda)+\Lambda\gamma\bar{\pi}]} \quad (22)$$

Substituting (22) in (20) gives

$$z = \frac{(\Lambda+\mu)(\gamma+\mu)(1+\Omega\bar{\pi})}{\alpha[(1+\Omega\bar{\pi})(\gamma+\mu+\Lambda)+\Lambda\gamma\bar{\pi}]} \quad (23)$$

Therefore the steady states are;

$$x = \frac{(\gamma + \mu)(1+\Omega \bar{\pi})}{\alpha \gamma_{\bar{\pi}}}, y = \frac{(\Lambda+\mu)(\gamma+\mu)(1+\Omega\bar{\pi})^2}{\alpha\gamma\bar{\pi}[(1+\Omega\bar{\pi})(\gamma+\mu+\Lambda)+\Lambda\gamma\bar{\pi}]}, z = \frac{(\Lambda+\mu)(\gamma+\mu)(1+\Omega\bar{\pi})}{\alpha[(1+\Omega\bar{\pi})(\gamma+\mu+\Lambda)+\Lambda\gamma\bar{\pi}]}$$

Setting y=z=0, we obtain the disease-free equilibrium (DFE)

$$\varepsilon_0 = (x, 0,0)$$

$$\varepsilon_0 = (\frac{(\gamma + \mu)(1+\Omega \bar{\pi})}{\alpha \gamma_{\bar{\pi}}}, 0,0)$$

3.2. Local Stability of the DFE

In this section we investigate the stability of the disease-free equilibrium state. First we obtain the characteristic equation. Let the equilibrium states be perturbed as follows;

Substituting (16) into the model equation (1)

$$\frac{d}{dt}[x+\bar{p}e^{\lambda t}] = \Lambda(x+\bar{p}e^{\lambda t}) + \Lambda(y+\bar{q}e^{\lambda t}) + \Lambda(z+\bar{r}e^{\lambda t}) - \alpha(x+\bar{p}e^{\lambda t})(z+\bar{r}e^{\lambda t}) - \mu(x+\bar{p}e^{\lambda t})$$

$$= \Lambda x + \Lambda\bar{p}e^{\lambda t} + \Lambda y + \Lambda\bar{q}e^{\lambda t} + \Lambda z + \Lambda\bar{r}e^{\lambda t} - \alpha xz - \alpha x\bar{r}e^{\lambda t} - \alpha z\bar{p}e^{\lambda t} - \alpha\overline{pr}e^{\lambda t}e^{\lambda t} - \mu x - \mu\bar{p}e^{\lambda t}$$

$e^{\lambda t}$

But from (6), $\Lambda(x+y+z) - \alpha xz - \mu x = 0$, so we have,

$$\lambda\bar{p}e^{\lambda t} = \Lambda\bar{p}e^{\lambda t} + \Lambda\bar{q}e^{\lambda t} + \Lambda\bar{r}e^{\lambda t} - \alpha x\bar{r}e^{\lambda t} - \alpha z\bar{p}e^{\lambda t} - \mu\bar{p}e^{\lambda t}$$

$$\lambda\bar{p} = \Lambda\bar{p} + \Lambda\bar{q} + \Lambda\bar{r} - \alpha x\bar{r} - \alpha z\bar{p} - \mu\bar{p}$$

Collecting like terms

$$0=(\Lambda-\mu-\lambda)\bar{p} + \Lambda\bar{q} + (\Lambda - \alpha x)\bar{r} \quad (24)$$

$$\frac{d}{dt}[y+\bar{q}e^{\lambda t}] = \alpha(x+\bar{p}e^{\lambda t})(z+\bar{r}e^{\lambda t}) - (\gamma+\mu)(y+\bar{q}e^{\lambda t})$$

$$\lambda\bar{q}e^{\lambda t} = \alpha xz + \alpha x\bar{r}e^{\lambda t} + \alpha z\bar{p}e^{\lambda t} + \alpha\bar{p}e^{\lambda t}\bar{r}e^{\lambda t} - y(\gamma+\mu) - (\gamma+\mu)\bar{q}e^{\lambda t}$$

Taking cognizance of (24) and neglecting term of order 2, gives

$$\lambda\bar{q}e^{\lambda t} = \alpha x\bar{r}e^{\lambda t} + \alpha z\bar{p}e^{\lambda t} - (\gamma+\mu)\bar{q}e^{\lambda t}$$

$$\lambda\bar{q} = \alpha x\bar{r} + \alpha z\bar{p} - (\gamma+\mu)\bar{q}$$

Thus,

$$0=\alpha z\bar{p} - (\gamma+\mu+\lambda)\bar{q} + \alpha x\bar{r} \quad (25)$$

Substituting (15) & (16) in (1), gives

$$\frac{\partial}{\partial t}[\phi(\tau)+\eta(\tau)e^{\lambda t}] + \frac{\partial}{\partial\tau}[\phi(\tau)+\eta(\tau)e^{\lambda t}] + h(\tau)(\phi(\tau)+\eta(\tau)e^{\lambda t}) = 0$$

Taking cognisance of (16), we have,

$$\lambda\eta(\tau)e^{\lambda t} + \frac{\partial\eta(\tau)}{\partial\tau}e^{\lambda t} + h(\tau)\eta(\tau)e^{\lambda t} = 0$$

$$\lambda\eta(\tau) + \frac{\partial\eta(\tau)}{\partial\tau} + h(\tau)\eta(\tau) = 0$$

$$\frac{\partial\eta(\tau)}{\partial\tau} + \big(\lambda + h(\tau)\big)\eta(\tau) = 0 \tag{26}$$

Solving the ODE in (26), we have

$$\eta(\tau) = \eta(0)exp - \int_0^\tau \big(\lambda + h(s)\big)ds \tag{27}$$

Integrating (27) over [0, T], gives

$$\int_0^T \eta(\tau)d\tau = \eta(0)\int_0^T exp\{-\int_0^\tau \big(\lambda + h(s)\big)ds\}\,d\tau \tag{28}$$

Substituting (25) in (27) gives

$$\bar{r} = \int_0^T \eta(\tau)d\tau = \eta(0)\int_0^T exp\{-\int_0^\tau \big(\lambda + h(s)\big)ds\}\,d\tau$$

$$\bar{r} = \eta(0)c(\lambda) \tag{29}$$

With $c(\lambda) = \eta(0)\int_0^T exp\{-\int_0^\tau \big(\lambda + h(s)\big)ds\}\,d\tau$
We now find $\eta(0)$ as follows; from (5),

$$\phi(0) = B(t) = \gamma\,\mathrm{y} - \Omega\mathrm{z}$$

$$\Rightarrow \quad \phi(0) = B(t) = \gamma\big(y + \bar{q}e^{\lambda t}\big) - \Omega\big(z + \bar{r}e^{\lambda t}\big) \tag{30}$$

From (17)

$$i(t,\tau) = \phi(\tau) + \eta(\tau)e^{\lambda t}$$

$$=> i(t,0) = B(t) = \phi(0) + \eta(0)e^{\lambda t} \tag{31}$$

Substituting (5) in (25) gives,

$$\mathrm{B(t)} = \gamma\,\mathrm{y} - \Omega\mathrm{z} + \eta(0)e^{\lambda t} \tag{32}$$

Equating (28) and (32) gives

$$\gamma\big(y + \bar{q}e^{\lambda t}\big) - \Omega\big(z + \bar{r}e^{\lambda t}\big) = \gamma\,\mathrm{y} - \Omega\mathrm{z} + \eta(0)e^{\lambda t} \tag{33}$$

$$\gamma\mathrm{y} + \gamma\ \ \bar{q}e^{\lambda t} - \Omega\mathrm{z} - \Omega\bar{r}e^{\lambda t} - \gamma\,\mathrm{y} + \Omega\mathrm{z} = \eta(0)e^{\lambda t}$$

$$=> \eta(0) = \gamma\bar{q}\text{-}\Omega\bar{r} \tag{34}$$

Substituting (30) in (25), gives

$$\bar{r} = (\gamma\bar{q}\text{-}\Omega\bar{r})c(\lambda)$$

$$\Rightarrow \quad 0 = \gamma\bar{q}c(\lambda) - \big(1 + \Omega c(\lambda)\big)\bar{r} \tag{35}$$

Thus we have the linearized system of equations from (17), (18) and (19)

$$(\Lambda\text{-}\mu - \lambda)\bar{p} + \Lambda\bar{q} + (\Lambda - \alpha x)\bar{r} = 0$$

$$\alpha z\bar{p} - (\gamma + \mu + \lambda)\bar{q} + \alpha x\bar{r} = 0 \tag{36}$$

$$\gamma c(\lambda)\bar{q} - (1 + \Omega c(\lambda))\bar{r} = 0$$

The coefficients of $\bar{p}, \bar{q}$ & $\bar{r}$ in (36), give the Jacobian determinant

$$\begin{vmatrix} \Lambda-\mu-\alpha z-\lambda & \Lambda & \Lambda-\alpha x \\ \alpha z & -(\gamma+\mu+\lambda) & \alpha x \\ 0 & \gamma c(\lambda) & -(1+\Omega c(\lambda)) \end{vmatrix} = 0$$

Therefore the character is tice quation for the model is given by,

$$(\Lambda-\mu-\lambda)[(\gamma+\mu+\lambda)(1+\Omega c(\lambda))-\alpha x(\gamma c(\lambda))]+\Lambda\alpha z(1+\Omega c(\lambda))+(\alpha z\gamma c(\lambda))(\Lambda-\alpha x)=0 \quad (37)$$

At the disease-free or non-zero equilibrium,
$\varepsilon_0(x,y,z)$, let the characteristics equation (37) take the form

$$H(\lambda)=(\Lambda-\mu-\alpha z-\lambda)[(\gamma+\mu+\lambda)(1+\Omega c(\lambda)-ax(\gamma c(\lambda))]+\Lambda\alpha z(1+\Omega c(\lambda))+(\Lambda-\alpha x)(\alpha z\gamma c(\lambda))(\Lambda-\alpha x)=0 \quad (38)$$

If we set

$$\lambda = i\,\omega \quad (39)$$

In (39) we have ,

$$H(i\,\omega) = F(\omega) + i\,G(\omega) \quad (40)$$

From (28)

$$c(\lambda)=\int_0^T \exp\{-\int_0^\tau(\lambda+h(s))ds\}d\tau=\int_0^T e^{-\lambda\tau}\pi(\tau)d\tau$$

Therefore

$$c(iw)=\int_0^T e^{-iw\tau}\pi(\tau)d\tau=\int_0^T[cosw\tau - isinw\tau]\pi(\tau)d\tau$$

$$c(iw)=f(w)+ig(w)$$

and so

$$f(w)=\int_0^T \pi(\tau)coswτd\tau \text{ and } g(w)=-\int_0^T \pi(\tau)sinw\tau d\tau$$

$$f(0)=\int_0^T \pi(\tau)d\tau=\bar{\pi},$$

$$g(0)=0 \quad (41)$$

$$f'(0)=0$$

$$g'(0)=-\tau\int_0^T\pi(\tau)d\tau=-\tau\bar{\pi}$$

Hence,

$$H(i\omega)=(\Lambda-\mu-\alpha z-i\omega)[(\gamma+\mu+i\omega)(1+\Omega c(i\omega))-\alpha x(\gamma c(i\omega))]+\Lambda\alpha z(1+\Omega c(i\omega))+(\alpha z\gamma c(i\omega))(\Lambda-\alpha x)=0 \quad (42)$$

$$H(i\omega)=(\Lambda-\mu-\alpha z-i\omega)[(\gamma+\mu+i\omega)(1+\Omega(f(w)+ig(w)))-\alpha x(\gamma f(w)+ig(w))]+\Lambda\alpha z(1+\Omega(f(w)+ig(w)))+(\alpha z\gamma(f(w)+ig(w))(\Lambda-\alpha x)$$

Therefore,

$$F(w)=(\Lambda-\mu-\alpha z)(\gamma+\mu)+(\Lambda-\mu-\alpha z)(\gamma+\mu)\Omega f(w)+\Lambda\alpha z+\Lambda\alpha z\Omega(f(w)+\Lambda\alpha z\gamma(f(w)+w^{2-+}w^2\Omega f(w)-\Omega g(w)(\Lambda-\mu-\alpha z)w+\Omega g(w)\,w(\gamma+\mu)$$

$$G(w)=w(\Lambda-\mu-\alpha z)-(\gamma+\mu)w+(\Lambda-\mu-\alpha z)w\Omega f(w)-w\Omega f(w)(\gamma+\mu)-(\Lambda-\mu-\alpha z)(\gamma+\mu)\Omega g(w)+\Omega g(w)\,w^2$$

Hence,

$$F(0)=(\gamma+\mu)(\Lambda-\mu)(1+\Omega\bar{\pi})$$

$$F'(0)=0$$

$$G'(0)=(\Lambda-2\mu-\gamma)(1+\Omega\bar{\pi})-(\Lambda-\mu)(\gamma+\mu)\tau\bar{\pi}$$

The condition for $\mathrm{Re}\lambda < 0$ is given by the inequality

$$F(0)G'(0)-F'(0)G(0)>0 \quad (43)$$

We now need to obtain the condition for which the inequality holds. That is,

$$F(0)G'(0)>0, \text{since } F'=0$$

$$F(0)=(\gamma+\mu)(\Lambda-\mu)(1+\Omega\bar{\pi})=(\gamma+\mu)(\Lambda-\mu)\varphi(k)$$

$S_0(0)=Sgn\varphi(k)$ Hence, F(0) > 0 if $(1+\Omega\bar{\pi})>0$

Also $G(0)=(\Lambda-2\mu-\gamma)(1+\Omega\bar{\pi})-(\Lambda-\mu)(\gamma+\mu)\tau\bar{\pi}>0$

Hence, if $\varphi(k)>0$

The non-zero state will be stable, and if otherwise it will be unstable at least locally.■

When $\varphi(k)>0$ then the control parameter k must be correspondingly high, which leads to the instability of the zero state and the possible stability of the non-zero state, locally. A high level of k indicates longer life span for the infected birds.

3.3. Local Stability of the Endemic (Zero) Equilibrium

At the endemic equilibrium (zero)$\varepsilon_0=(0,0,0)$we assume that the disease causes high mortality rate that could wipe out the entire population of bird in the poultry, hence, the characteristic equation takes the form

$$(\Lambda-\mu-\lambda)[(\gamma+\mu+\lambda)(1+\Omega c(\lambda))]=0 \quad (44)$$

From (44)

$$(\Lambda-\mu-\lambda)(\gamma+\mu+\lambda)=0 \quad (45)$$

or

$$\left(1+\Omega c(\lambda)\right)=0 \quad (46)$$

Theorem1: The endemic equilibrium is stable when

$$\Lambda<\mu \text{ and } \varphi(k)<0$$

Proof: Equation (3.35) is a quadratic equation in λ with negative roots, when

$$\Lambda<\mu$$

In order to investigate the nature of the root of (38) for the stability of the system, let (40) take the form

$$h(\lambda)=\left(1+\Omega c(\lambda)\right)=0 \quad (47)$$

If we set $\lambda=iw$ in (36) we have that

$$h(iw)=h_1(w)+ih_2(w)$$

the condition for Re $\lambda<0$ will then be given by the inequality

$$h_1(0)\, h_2^{I}(0)-h_1^{I}(0)\, h_2(0)>0 \quad (48)$$

From (25),

$$c(iw)=f(w)+ig(w)$$

And so,

$$f(w)=\int_0^T \pi(\tau)\cos w\tau d\tau \text{ and } g(w)=-\int_0^T \pi(\tau)\sin w\tau d\tau$$

$$f(0)=\int_0^T \pi(\tau)d\tau=\bar{\pi}$$

$$g(0)=0$$

$$f^{I}(0)=0$$

$$g^{I}(0)=-\tau\int_0^T \pi(\tau)d\tau=-\tau\bar{\pi}$$

From (36)

$$h(iw)=(\gamma+\mu+iw)(1+\Omega(c(iw))-\alpha x\gamma[c(iw)]=(\gamma+\mu+iw)(1+\Omega(f(w)+ig(w))-\alpha x\gamma[f(w)+ig(w)]$$

Thus, if we set $\alpha=0$ (that is the neighborhood of $\alpha=0$)

$$h_1(w)=(\gamma+\mu)+(\gamma+\mu)\Omega f(w)+w\Omega g(w)$$
$$h_2(w)=(\gamma+\mu)\Omega g(w)-w-w\Omega f(w)$$
$$h_1(0)=(\gamma+\mu)+(\gamma+\mu)\Omega\bar{\pi}$$
$$h_2(0)=0$$
$$h_1'(0)=-(\Omega\tau\bar{\pi}+w\Omega\tau\bar{\pi})$$
$$h_2'(0)=-(\Omega\bar{\pi}+\tau\bar{\pi}(\gamma+\mu))$$

Since $h_2(0)=0$,
Hence, the inequality gives

$$[(\gamma+\mu)(1+\Omega\bar{\pi})][\Omega\bar{\pi}+\tau\bar{\pi}(\gamma+\mu)]<0$$

Since $[\Omega\bar{\pi}+\tau\bar{\pi}(\gamma+\mu)]>0$, then the inequality will hold if

$$[(\gamma+\mu)(1+\Omega\bar{\pi})]<0$$

But Sgn $h_1(0)$ = Sgn$\varphi(k)$
Therefore

$$(\gamma+\mu)(1+\Omega\bar{\pi})<0 \text{ if } \varphi(k)<0$$

Therefore the system will be stable if $\Lambda<\mu$ and $\varphi(k)<0$.■

For $\varphi(k)<0$, k must be very low, this gives the condition for the stability of the origin A low level of kindicates high rate of death among the infected, the high prevalence of the disease in the poultry, hence, the stability of the origin leading to a wiping out of the population.

4. Discussion

We have presented here on the graphs, the results of the study based on this model. Two control measures were considered; bird vaccination (v) and biosecurity measures (u). The infection death rates is also simulated based on the ages of infection, starting from 4weeks to the maximum age of infection T = 24 weeks. The following parameter values are used for the simulation:

Table 2. *Parameters and Values.*

Parameter	Value	Source
α	0.1	Carly (2016)
μ	0.2	Carly (2016)
k	0.3	Carly (2016)
γ	0.03	Carly (2016)
Λ	40	Carly (2016)
Ω	0.02	Carly (2016)

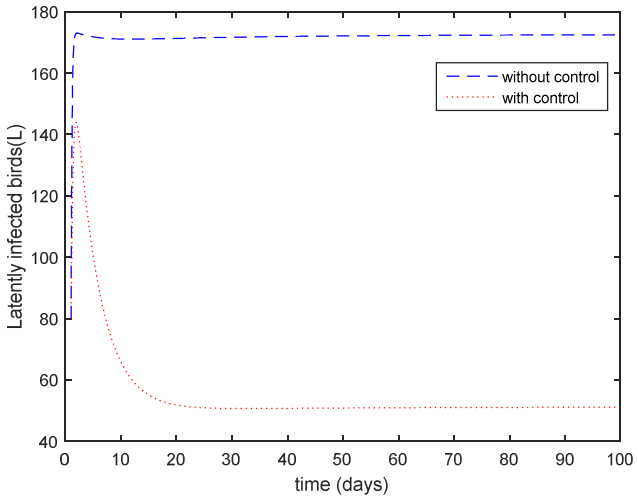

Figure 2. *Simulations howing the effect of vaccination and biosecurity measure son the latently infected Population..*

Figure 2 shows the effect of both vaccination (v) and biosecurity measures (u) on the latently infected birds. The population of infected birds increases speedily and then remains stable without the application of any control measure, with the controls, the population increases to about

145 and then begins to reduces from day 8 till it drops to 50 on day 20 and then remains stable.

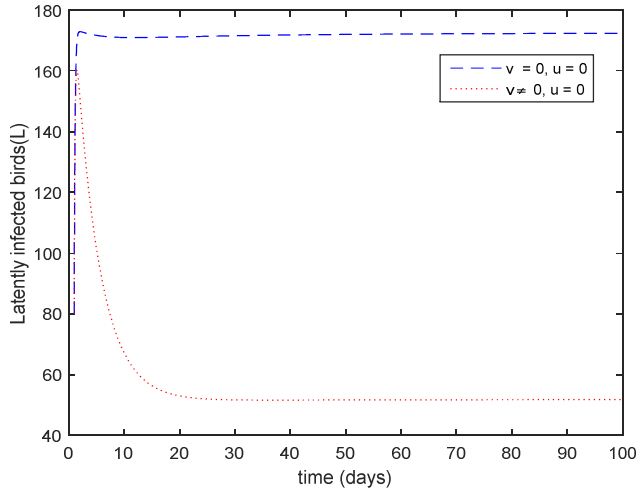

Figure 3. *Simulation showing the effect of vaccination on the latently infected population.*

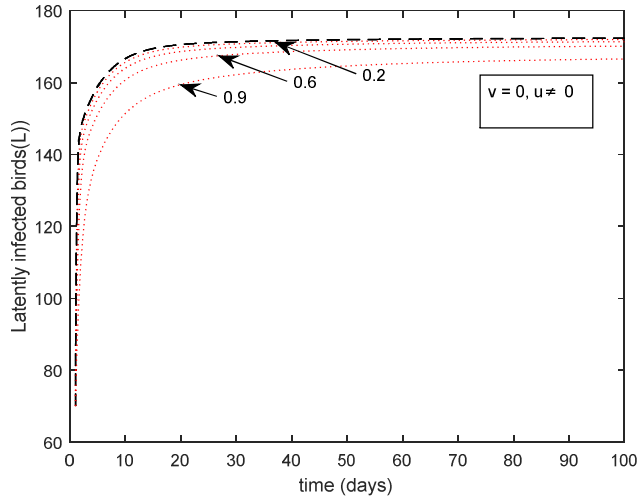

Figure 4. *Simulation showing the effect of biosecurity measure on the latently infected population.*

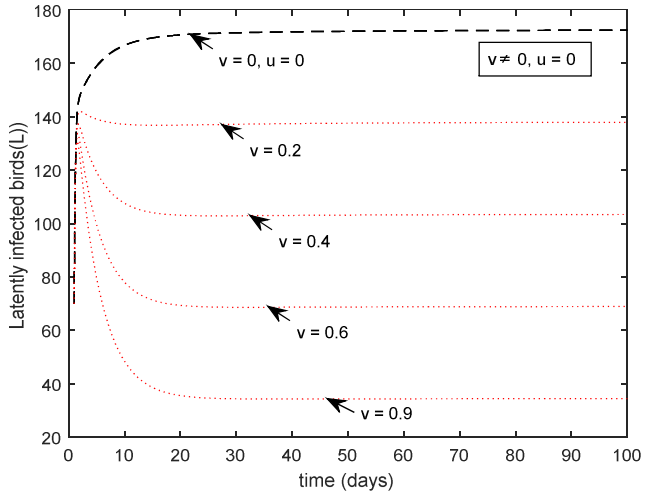

Figure 5. *Simulation showing the effect of vaccination on latently infected population.*

With this strategy, only bird vaccination (*v)* is applied to control the system while the other control is set to zero. In Figure 3, the effect of bird vaccination and its positive impact is revealed, though there is an increase to about 160 before a decrease occurs.

From Figure 4, as the control (u) ranges from 0.2 to 0.9, we see that the bird population still has a high level of latently infected birds. This result from figure shows that the bird population is not free from the disease, hence, the biosecurity control strategy is not effective without vaccination of susceptible birds and hence it is not preferable as the only control measure for Marek disease.

The numerical result in Figure 5, shows that as the latently infected bird population increases without control, with vaccination it decreases as more susceptible birds are vaccinated.

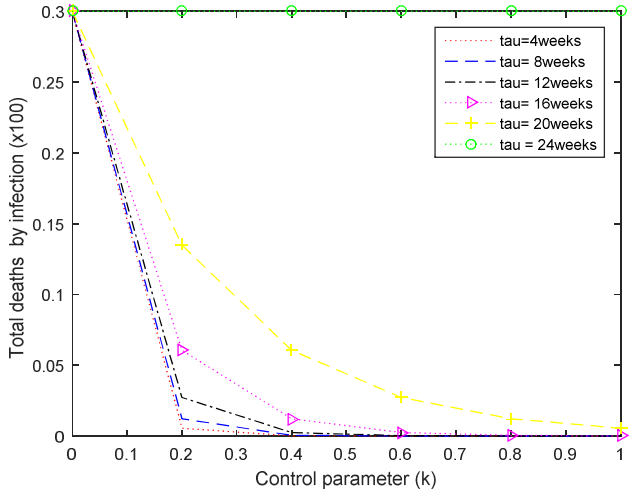

Figure 6. *A plot showing the total deaths by infection at different infection ages with* $0 \leq k \leq 1$..

From Figure 6, we observe, that as the control parameter increases, the total deaths by infection reduces, also as the age of th infection increases to the maximum age of infection which is 6 months (that is, T = 24 weeks), the number of deaths increases to30 in a day. Hence, control measures should be applied at the early ages of infection in order to avoid high mortality rate during the outbreak of the disease.

5. Conclusion

5.1. Summary

In this paper, we considered the theoretical analysis of compartmental Marek disease in a poultry. The study is briefly summarised below: Firstly, stability analysis were carried out using the Lyaponuv function theory and Lasslle's invariance principle for each of these disease models. 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.

Finally, the results of the study were presented using numerical simulations for each of the disease models, for which each intervention strategies were discussed and results established.

5.2. Conclusion

In the case of Marek disease, an infection–age structured model was formulated and analysed. The system was established to have a stable non–zero state (the state at which the disease will not wipe the entire bird population) when$\varphi(k) > 0$. This implies that at this stage, the control parameter will be high which indicates a longer life span for the infected birds.

Furthermore, the system will have a zero equilibrium (the state at which the disease wipes the entire bird in the poultry) if$\Lambda < \mu \; and \; \varphi(k) < 0 \; and$ for$\varphi(k)$< 0, k must be very low. A low level of k indicates high rate of death among the infected birds which implies the high prevalence of the disease in the poultry, hence, the stability of the origin leading to a wiping out of the population.

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, were commend 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.

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