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Stability Analysis And Control Of Coccidiosis Disease In Poultry

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Abstract
In this paper, we formulate a multi – group model of the Coccidiosis disease with eight (8) compartments. The susceptible birds are divided into two groups, latently infected birds are divided into three groups, infected birds are divided into two groups according to infectivity and the recovered birds. A deterministic model was formulated and analyzed using methods from dynamical systems theory. Conditions for the stability of the disease-free and endemic steady states were determined. The stability analysis shows that the disease-free equilibrium (DFE) is locally stable if $\mu > r_1 b$ *and* $\mu > r_2 b$*. The endemic equilibrium (EE) is locally stable if* $E_2 > \frac{\beta I_2}{r_1 b - \mu} - I_2$ *and* $E_2 > \frac{\beta I_2}{r_2 b - \mu} - I_2$*. The global stability of DFE and EE were proved using Lyapunov method. We then investigated the relative impact of each of the constant parameters on the basic reproduction number* (R_0)*, using the concept of sensitivity analysis. Three control measures were introduced. The control* $v_1(t)$ *represents the control variable based on bird vaccination,* $v_2(t)$ *the control variable based on good poultry management practices and* $v_3(t)$ *the control variable to measure the effectiveness of anticoccidial drugs. The Optimal Control approach is used to find the best strategy to fight the disease and minimize cost. The economic evaluation involved in the use of these control measures was also considered. Finally, a numerical solution (simulation) of the model was carried out to gain more insight on the dynamics of the disease.*
Keywords: *Coccidiosis Disease, Stability Analysis, Optimal Control, Sensitivity Index*

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I. Introduction

Avian Coccidiosis is a common protozoal gastrointestinal disease of birds caused by parasites of the genus *Eimeria called coccidia. It* develops withinthe intestine of most domestic birds. Seven species of *Eimeria* (*E. acervulina, E.brunetti, E. maxima, E. mitis, E. necatrix, E. praecox* and *E. tenella*) are recognized as infecting chickens. Although coccidiosis is a disease known for many years, it is still considered as the most economical important parasitic condition affecting poultry production worldwide (Gussem 2008).The coccidia hide in the intestine or caecum of the birds and passed out through the droppings. Droppings of infected fowls carry in them the coccidia in the oocyst, or egg, stagewhere they require favourable conditions (likein damp surroundings) outside the body for a short time (21 to 48 hours) to develop or ripen, and become infective. Consequently preventive measures based on management practices are effective in checking Coccidiosis. They become infective in damp surroundings and are spread from bird to bird when infected droppings are eaten in contaminated feed or water. Coccida may survive for long periods in litter.

The species of Eimeria that are reported as highly pathogenic are E. brunetti, E. maxima, E. necatrix, and E. tenella. Species reported as mildly pathogenic include E. acervulina, E. mitis, and E. mivati, whereas E. praecox and E. hagani are considered to be the least pathogenic, Jadhav *et al.* (2011).

The chronic intestinal form develops and spreads more slowly within the flock or unit than the cecal type, but as in the cecal form, the severity of the disease is determined largely by the number of Coccidia actually swallowed by the susceptible birds. Many birds may survive but remain unthrifty and unprofitable.

Coccidia causing the intestinal or chronic form of the disease *(E. necatrix, E. maxima)* usually do not produce so many oocysts as the cecal type *(E. tenella.* The rate of spread is less rapid, and the course of the disease tends to be less severe *(Graham &Brandly 2000)*. Symptoms of chronic intestinal coccidiosis-unthriftiness, poor appetite, loss of flesh, paleness, poor or rough feathering, weakness and dullness-are often poorly defined and a specific disease is not always suspected. Often the symptoms of chronic coccidiosis cannot be distinguished from those caused by various infectious diseases of poultry or by internal or external parasites or dietary deficiencies. Other clinical signs include mucus-like or bloody diarrhea, dehydration, anemia, restlessness, ruffled feathers, stunted growth, and death. Coccidiosis disease is also commonly associated with a drop in egg production.

The agents used for the control of coccidian infections are termed anticoccidial drugs. Sulfa drugs (like sulfamethazine administered at 0.1 percent for two days) are also effective, but shouldn't be used in layers .Administration of water dispersible vitamin A and K supplements may also enhance recovery. Specific prevention measures includes the use of vaccines and good poultry management practices like; Controlling moisture with the appropriate installation and management of watering systems and periodically moving the location of the chickens to allow the land fallow for several weeks so as to reduce the pathogen load in the environment.

Application of optimal control to mathematical modeling has become an important and useful tool in studying the spread and control of infectious diseases. Agusto (2013) applied optimal control theory to a system of ordinary differential equation describing a two-strain avian influenza transmission via the Pontryagin's Maximum Principle. To this end, a pair of control variables representing the isolation strategies for individuals with avian and mutant strains was incorporated into the transmission model. The infection averted ratio (IAR) and the incremental cost-effectiveness ratio (ICER) were calculated to investigate the cost-effectiveness of all possible combinations of the control strategies. The simulation results show that the implementation of the combination strategy during the epidemic is the most cost-effective strategy for avian influenza transmission.

A model to assess the impact of some control measures in the dynamics of Rift Valley Fever (RVF) was considered by Mpeshe *et al* (2014). They derived and analysed the conditions for optimal control of RVF with insecticides, vaccination, and personal protection using optimal control theory. It was shown that the control measures have a very desirable effect for reducing the number of infected individuals and that multiple controls are more effective than single control. Moreover, effective and optimal use of insecticides and personal protection without the use of vaccination was considered not beneficial if total elimination of the disease is desirable in the community.

A paper by Okosun *et al* (2016), 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.

Models on Coccidiosis include that of Aytac *et al* (2011). They examined the risk factors of coccidiosis in poultry by using a logistic regression model. The model was analysed and used to assess variables that influenced the occurrence of Coccidiosis. Williams (1999) in his study formulated a compartmentalized model for the estimation of the monetary losses suffered by the world's poultry industry resulting from Coccidiosis of chickens and costs of its control. Equations are given for relationships among numbers of chickens, live weights, and weights of carcasses, feed consumptions, feed conversion ratio (FCR), prices of feeds, prices of anticoccidial therapeutic and prophylactic drugs, values of chickens, chicken rearing costs; and effects of Coccidiosis on mortality, weight gain and FCR. . Using these equations, each relevant loss element was calculated simultaneously. Addition of these elements gave an accurate global estimate of the losses due to chicken coccidiosis.

In this study, we present the stability and control of Coccidiosis disease in poultry birds. Our goal is to determine optimal strategy needed for the prevention and treatment of Coccidiosis in order to reduce incidence rate in poultry, hence improve poultry productivity.

II. Formulation Of Coccidiosis Disease Model

2.1 Assumptions of the model

The assumptions of the model are as follows:

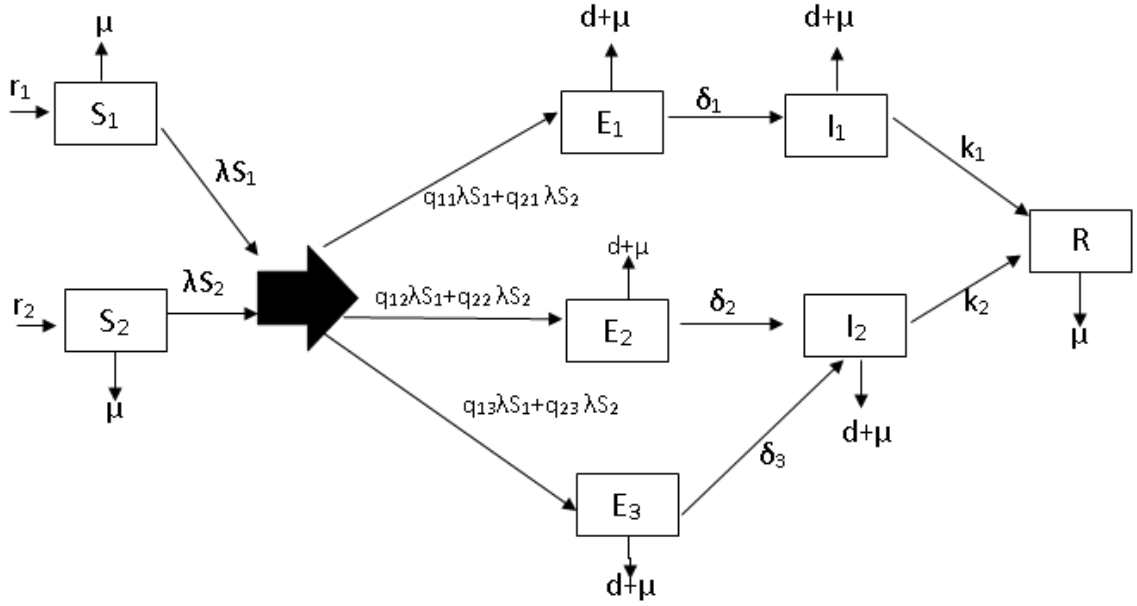
1. Recovered birds develop permanent immunity to the disease
2. Birds are recruited by birth
3. Infected birds can also die naturally

2.2 Model parameters for Coccidiosis disease

$S_1(t)$	-	Susceptible birds with no resistance
$S_2(t)$	-	Susceptible birds with partial resistance
$E_1(t)$	-	Latently infected birds with high – pathogenic occyst
$E_2(t)$	-	Latently infected birds with moderate pathogenic occyst
$E_3(t)$	-	Latently infected birds with low pathogenic occyst
$I_1(t)$	-	Infectious birds with high pathogenic occyst
$I_2(t)$	-	Infectious birds with moderate / low pathogenic occyst
$R(t)$	-	Recovered birds
b	-	Constant recruitment rate
r_1	-	Recruitment proportion of susceptible birds with no resistance
r_2	-	Recruitment proportion of susceptible birds with partial resistance

λ - Force of infection
q_{ij} - Proportion of susceptible birds that enter the infectious classes
δ_i - Progression rates from the exposed classes to the infectious classes
k_i - Recovery rate from the infectious classes to the recovered class
μ - Natural death rate
d - Disease – induced death rate
$v_1(t)$ - control variable based on bird vaccination.
$v_2(t)$ - control variable based on good management practices
$v_3(t)$ - control variable to measure the effectiveness of anticoccidial drugs

2.3 Model flow diagram



2.4 Equations of the model

We will make use of the level of natural resistance of birds to coccidiosis which incorporates the transmission heterogeneity in the epidemiological models. We classify the susceptible into two groups; Susceptible with no resistance (S_1) and susceptible with partial resistance (S_2). Based on the pathogenesis of the disease, latently infected birds are classified into three groups; latently infected birds with high – pathogenic occyst (E_1), moderate pathogenic occyst (E_2) and low pathogenic occyst (E_3). I_1 represent the infectious birds with high-pathogenic occyst while I_2 are birds infected with moderate and low occysts. Birds with moderate and low pathogenic occysts are most infectious than those with high pathogenic occysts given to the growth nature of the parasite, Graham & Brandly (2000).

We assume that b is the constant recruitment rate to replenish the susceptible groups through birth. Then μ be the per capita death rate then $1/\mu$ is the average life span of the birds.

During the contact between an S_2 bird and an I_i bird, the transmission rate β of the infected bird is reduced to $x\beta$ with $0 < x < 1$, to account for the partial resistance to the disease.

Newly infected S_i birds (i = 1, 2) join the three latently infected classes with subscript j = 1, 2, 3 with respective proportions q_{ij} which satisfies $0 < q_{ij} < 1$ and $\sum_{j=1}^{2} q_{ij} = 1$

Therefore $\lambda = x\beta \frac{I_1}{N} + \beta \frac{I_2}{N}$. The model is given below.

$$\frac{dS_1}{dt} = r_1 bN - \lambda S_1 - \mu S_1$$
$$\frac{dS_2}{dt} = r_2 bN - \lambda S_2 - \mu S_2$$
$$\frac{dE_1}{dt} = (q_{11}\lambda S_1 + q_{21}\lambda S_2) - (\delta_1 + d + \mu)E_1$$
$$\frac{dE_2}{dt} = (q_{12}\lambda S_1 + q_{22}\lambda S_2) - (\delta_2 + d + \mu + \mu)E_2 \qquad (2.1)$$
$$\frac{dE_3}{dt} = (q_{13}\lambda S_1 + q_{23}\lambda S_2) - (\delta_3 + d + \mu)E_3$$
$$\frac{dI_1}{dt} = \delta_1 E_1 - (d + k_1 + \mu)I_1$$
$$\frac{dI_2}{dt} = \delta_2 E_2 + \delta_3 E_3 - (d + k_2 + \mu)I_2$$
$$\frac{dR}{dt} = k_1 I_1 + k_2 I_2 - \mu R$$
$$where\ N = S_1 + S_2 + E_1 + E_2 + E_3 + I_1 + I_2 + R$$

2.5 Control model

Three control measures are considered here. First is the control based on chicken vaccination (v_1), secondly is the control based on good management practices (v_2). This involves using appropriate watering systems like nipple drinkers to avoid spillage of water on litter and relocating birds periodically to leave the land fallow for a while. Thirdly, is the control that measures the effectiveness of anticoccidial drugs (v_3). These drugs can either be Coccidiostatic or Coccidiocidal agents. The control model is given as;

$$\frac{dS_1}{dt} = (1-v_1)r_1 bN - \lambda S_1(1-v_2) - \mu S_1$$
$$\frac{dS_2}{dt} = (1-v_1)r_2 bN - \lambda S_2(1-v_2) - \mu S_2$$
$$\frac{dE_1}{dt} = (q_{11}\lambda S_1 + q_{21}\lambda S_2)(1-v_2) - (\delta_1 + d + \mu)E_1$$
$$\frac{dE_2}{dt} = (q_{12}\lambda S_1 + q_{22}\lambda S_2)(1-v_2) - (\delta_2 + d + \mu + \mu)E_2 \qquad (2.2)$$
$$\frac{dE_3}{dt} = (q_{13}\lambda S_1 + q_{23}\lambda S_2)(1-v_2) - (\delta_3 + d + \mu)E_3$$
$$\frac{dI_1}{dt} = \delta_1 E_1 - (d + v_3 + \mu)I_1$$
$$\frac{dI_2}{dt} = \delta_2 E_2 + \delta_3 E_3 - (d + v_3 + \mu)I_2$$
$$\frac{dR}{dt} = v_3 I_1 + v_3 I_2 - \mu R$$
$$where\ N = S_1 + S_2 + E_1 + E_2 + E_3 + I_1 + I_2 + R$$

III. Analysis of the Model

3.1 Basic properties of the Model

For the Coccidiosis transmission model (2.1) to be epidemiologically meaningful, it is important to prove that all solutions with non-negative initial data will remain non –negative for all time.

Theorem 3.1: If $S_1(0)$, $S_2(0)$, $E_1(0)$, $E_2(0)$, $E_3(0)$, $I_1(0)$, $I_2(0)$, R(0), are non-negative then solutions of the system (2.4) are positive for all t > 0.

Proof:

It is clear from the first equation of (2.1) that

$$\frac{dS_1}{dt} \geq -(\lambda + \mu)S_1$$

So that by Gronwall's inequality,

$$S_1(t) \geq S_1(0)\exp[-\int_0^t (\lambda + \mu)\, du] > 0, \text{ for all } t > 0.$$

It can be shown, using similar approach, that $S_1(t) > 0$, $S_2(t) > 0$, $E_1(t) > 0$, $E_2(t) > 0$, $E_3(t > 0)$, $I_1(t) > 0$, $I_2(t) > 0$ and R(t) > 0 for all t > 0.

Theorem 3.2: The closed set

D = $\{(S_1, S_2, E_1, E_2, E_3, I_1, I_2, R) \in R_+^8 : N \leq \frac{b}{\mu}\}$ is positively – invariant.

Proof:

The rate of change of N is given by

$\frac{dN(t)}{dt} = r_1 b\,N + r_2 b\,N + r_3 b\,N - \mu N(t) - d[E_1(t)+E_2(t)+E_3(t)+I_1(t)+I_2(t)]$

Let $b = r_1 b\,N + r_2 b\,N + r_3 b\,N$

so that,

$$\frac{dN}{dt} \leq b - \mu N(t)$$

It follows that

$\frac{dN}{dt} \leq 0 \; if \; N \geq \frac{b}{\mu}$.

Thus by Gronwall's inequality, it can be shown that,

$$N(t) \leq N(0)e^{-\mu t} + \frac{b}{\mu}[1 - e^{-\mu t}]$$

In particular, $N(t) \leq \frac{b}{\mu} \; if \; N(0) \leq \frac{b}{\mu}$. Thus the region D is positively invariant. Hence, it is sufficient to consider the dynamics of the model (2.1) in D.

Furthermore, if $N(0) > \frac{b}{\mu}$ then either the solution enters D in finite time, or N(t) approaches $\frac{b}{\mu}$asymptotically. Hence, the region D attracts all solutions in R_+^8 and our solutions remain bounded. In this region the model can be considered as been epidemiologically and mathematically well posed (Garba 2014).

3.2 Steady states and stability

3.2.1 Existence of steady (Equilibrium) state for the Coccidiosis disease model

The system is in a steady state if $\frac{dS_1}{dt} = \frac{dS_2}{dt} = \frac{dE_1}{dt} = \frac{dE_2}{dt} = \frac{dE_3}{dt} = \frac{dI_1}{dt} = \frac{dI_2}{dt} = \frac{dR}{dt} = 0$, that is,

$$r_1 bN - (\lambda + \mu)S_1 = 0 \quad \text{.....(i)}$$

$$r_2 bN - (\lambda + \mu)S_2 = 0 \quad \text{.....(ii)}$$

$$\lambda(q_{11}S_1 + q_{21}S_2) - (\delta_1 + d + \mu)E_1 = 0 \quad \text{.....(iii)}$$

$$\lambda(q_{12}S_1 + q_{22}S_2) - (\delta_2 + d + \mu)E_2 = 0 \quad \text{.....(iv)}$$

$$\lambda(q_{13}S_1 + q_{23}S_2) - (\delta_3 + d + \mu)E_3 = 0 \quad \text{.....(v)}$$

$$\delta_1 E_1 - (d + k_1 + \mu)I_1 = 0 \quad \text{.....(vi)}$$

$$\delta_2 E_2 + \delta_3 E_3 - (d + k_2 + \mu)I_2 = 0 \quad \text{.....(vii)}$$

$$k_1 I_1 + k_2 I_2 - \mu R = 0 \quad \text{.....(viii)}$$

Solving these eight equations for $S_1^0, S_2^0, E_1^0, E_2^0, E_3^0, I_1^0, I_2^0$, R we have the following:

From (i), we have $r_1bN = (\lambda + \mu)S_1^0 => S_1^0 = \frac{r_1bN}{\lambda + \mu}$

From (ii) $S_1^0 = \frac{r_2bN}{\lambda+\mu}$

From (iii) $\lambda(q_{11}S_1 + q_{21}S_2) - (\delta_1 + d + \mu)E_1 = 0 => E_1^0 = \frac{\lambda(q_{11}S_1^0 + q_{21}S_2^0)}{\delta_1 + d + \mu}$

Equating (v) and (vii) $E_3^0 = \frac{\lambda(q_{13}S_1^0 + q_{23}S_2^0)}{\delta_3 + d + \mu} = \frac{(d + k_2 + \mu)I_1^0 - \delta_2 E_2^0}{\delta_3}$

$=> E_2^0 = \frac{(\delta_3 + d + \mu)\,(d + k_2 + \mu)I_1^0 - \delta_2\,\lambda(q_{13}S_1^0 + q_{23}S_2^0)}{\delta_2\,(\delta_3 + d + \mu)}$

From (vi) $\delta_1 E_1 - (d + k_1 + \mu)I_1 = 0 => I_1^0 = \frac{\delta_1 E_1^0}{d + k_1 + \mu}$

From (vii) $I_2^0 = \frac{\delta_2 E_2^0 + \delta_3 E_3^0}{d + k_2 + \mu}$

From (viii) $k_1 I_1 + k_2 I_2 - \mu R = 0 => R^0 = \frac{k_1 I_1^0 + K_2 I_2^0}{\mu}$

Therefore the steady state is:

$$S_1^0 = \frac{r_1bN}{\lambda + \mu},\ S_2^0 = \frac{r_2bN}{\lambda + \mu},\qquad E_1^0 = \frac{\lambda(q_{11}S_1^0 + q_{21}S_2^0)}{\delta_1 + d + \mu},$$

$$E_2^0 = \frac{(\delta_3 + d + \mu)\,(d + k_2 + \mu)I_1^0 - \delta_2\,\lambda(q_{13}S_1^0 + q_{23}S_2^0)}{\delta_2\,(\delta_3 + d + \mu)} \qquad (3.1)$$

$$E_3^0 = \frac{\lambda(q_{13}S_1^0 + q_{23}S_2^0)}{\delta_3 + d + \mu},\ I_1^0 = \frac{\delta_1 E_1^0}{d + k_1 + \mu},\qquad I_2^0 = \frac{\delta_2 E_2^0 + \delta_3 E_3^0}{d + k_2 + \mu},\qquad R^0 = \frac{k_1 I_1 + k_2 I_2}{\mu}$$

The disease free steady state is $\xi_0 = \left(\frac{r_1bN}{\lambda + \mu}, \frac{r_1bN}{\lambda + \mu}, 0,0,0,0,0,0\right)$

Linear zing the system (2.1) we have the Jacobian matrix as

$$J = \begin{pmatrix} r_1b - \lambda - \mu & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & r_2b - \lambda - \mu & 0 & 0 & 0 & 0 & 0 & 0 \\ q_{11}\lambda & q_{21}\lambda & -(\delta_1 + d + \mu) & 0 & 0 & 0 & 0 & 0 \\ q_{12}\lambda & q_{22}\lambda & 0 & -(\delta_2 + d + \mu) & 0 & 0 & 0 & 0 \\ q_{13}\lambda & q_{23}\lambda & 0 & 0 & -(\delta_3 + d + \mu) & 0 & 0 & 0 \\ 0 & 0 & \delta_1 & 0 & 0 & -(d + k_1 + \mu) & 0 & 0 \\ 0 & 0 & 0 & \delta_2 & \delta_3 & 0 & -(d + k_2 + \mu) & 0 \\ 0 & 0 & 0 & 0 & 0 & k_1 & k_2 & -\mu \end{pmatrix}$$

Theorem 3.3 The disease-free steady (equilibrium) state of the model (2.1) is asymptotically stable, if $r_1b - \mu <$ 0 and $r_1b - \mu < 0$.

Proof:

Evaluating J_{DF} at ξ_0 we have,

$$J_{DF_0}=\begin{pmatrix} r_1b-\mu & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & r_2b-\mu & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\delta_1+d+\mu) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\delta_2+d+\mu) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\delta_3+d+\mu) & 0 & 0 & 0 \\ 0 & 0 & \delta_1 & 0 & 0 & -(d+k_1+\mu) & 0 & 0 \\ 0 & 0 & 0 & \delta_2 & \delta_3 & 0 & -(d+k_2+\mu) & 0 \\ 0 & 0 & 0 & 0 & 0 & k_1 & k_2 & -\mu \end{pmatrix}$$

Finding the determinant $\left|J_{DF_0}-\lambda I\right|$, the eigen-values of the system are:

$r_1b-\mu$, $r_2b-\mu$, $-(\delta_1+d+\mu)$, $-(\delta_2+d+\mu)$, $-(\delta_3+d+\mu)$, $-(d+k_1+\mu)$, $-(d+k_2+\mu)$ and $-\mu$

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

(i) $r_1b-\mu<0 \Rightarrow \mu>r_1b$

(ii) $r_2b-\mu<0 \Rightarrow \mu>r_2b$ ■

That is, for the disease-free steady state to be stable, $\mu>r_1b$ and, $\mu>r_2b$, this means for the disease to be under control and eradicated within a while from its outbreak, the natural death rate of birds (μ) will be greater than the number of susceptible birds recruited (b)

3.2.2 Endemic Steady (equilibrium) State

In this section we investigate the stability of the endemic state E_E^*. Note that in a pure endemic state, $S_1^0=S_2^0=R=0$. From (3.1) $\xi_E^0=(,0,0,0,E_2^0,0,0,I_2^0,0)$ where E_2^0, I_2^0 are as defined in (3.2) below:

$$E_2^0=\left(\frac{(\delta_3+d+\mu)(d+k_2+\mu)I_2^0}{\delta_2(\delta_3+d+\mu)}\right),\quad I_2^0=\frac{\delta_2E_2^0}{d+k_2+\mu} \qquad (3.2)$$

This can be interpreted to mean that the latently infected birds with moderate pathogenic occyst (E_2) and Infectious birds with moderate / low pathogenic occyst (I_2) dominants when the disease is endemic. This is so because birds infected with moderate / low pathogenic occyst are considered most infectious, Graham & Brandly (2000).

Theorem 3.4: The endemic steady (equilibrium) state of the Coccidiosis disease model is locally asymptotically stable if $E_2>\frac{\beta I_2}{r_1b+\mu}-I_2$ or $E_2>\frac{\beta I_2}{r_2b+\mu}-I_2$

Proof: Evaluating J at ξ_E^0 gives

$$J(\xi_E^0)=\begin{pmatrix} r_1b-\Psi-\mu & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & r_2b-\Psi-\mu & 0 & 0 & 0 & 0 & 0 & 0 \\ q_{11}\Psi & q_{21\Psi} & -(\delta_1+d+\mu) & 0 & 0 & 0 & 0 & 0 \\ q_{12}\Psi & q_{22}\Psi & 0 & -(\delta_2+d+\mu) & & 0 & & 0 \\ q_{13}\Psi & q_{23}\Psi & 0 & 0 & -(\delta_3+d+\mu) & 0 & & 0 \\ 0 & 0 & \delta_1 & 0 & 0 & -(k_1+d+\mu) & & 0 \\ 0 & 0 & 0 & \delta_2 & \delta_3 & 0 & -(k_2+d+\mu) & 0 \\ 0 & 0 & 0 & 0 & 0 & k_1 & k_2 & -\mu \end{pmatrix}$$

where $\psi=\frac{\beta I_2}{E_2+I_2}$

The eigen values of the characteristic equation $\left|JE_E^*-I\lambda\right|=0$ are;

$r_1b-\psi-\mu, r_2b-\psi-\mu,\quad -(\delta_1+d+\mu),\quad -(\delta_2+d+\mu),\quad -(\delta_3+d+\mu),\quad -(d+k_1+\mu),$
$-(d+k_2+\mu)$ and $-\mu$

Following from Theorem (3.2), the endemic steady state will be asymptotically stable if;

(i) $r_1b-\dfrac{\beta I_2}{E_2+I_2}-\mu<0 \Rightarrow E_2>\frac{\beta I_2}{r_1b+\mu}-I_2$ and

(ii) $r_2b-\dfrac{\beta I_2}{E_2+I_2}-\mu<0 \Rightarrow E_2>\frac{\beta I_2}{r_2b+\mu}-I_2$

Thus the endemic steady state is stable if, $E_2>\frac{\beta I_2}{r_1b+\mu}-I_2$ and $E_2>\frac{\beta I_2}{r_2b+\mu}-I_2$. This means that the disease to persist, the latently infected birds with moderate pathogenic occyst at time t, (E_2) is greater than the difference between the ratio of the rate of transmission of infectious birds with moderate pathogenic occyst and the sum of the rates of recruitment and natural death rate of birds, and the number of infectious birds with moderately pathogenic occyst

3.2.3 Global stability of the DFE

Theorem 3.5: The disease free steady (equilibrium) state is globally asymptotical stable if $R_0<1$.

Proof: Using comparison theorem. Let $S_B = S_T = S$. The equations for the infected components of the model (2.1) can be written as

$$\frac{dE_1}{dt}=\lambda(q_{11}S_1+q_{21}S_2)-(\delta_1+d+\mu)E_1$$
$$\frac{dE_2}{dt}=\lambda(q_{12}S_1+q_{22}S_2)-(\delta_2+d+\mu)E_2$$
$$\frac{dE_3}{dt}=\lambda(q_{13}S_1+q_{23}S_2)-(\delta_3+d+\mu)E_3$$
$$\frac{dI_1}{dt}=\delta_1E_1-(d+k_1+\mu)I_1$$
$$\frac{dI_2}{dt}=\delta_2E_2+\delta_3E_3-(d+k_2+\mu)I_2$$

These equations can be simplified as follows

$$\begin{pmatrix}\frac{dE_1}{dt}\\ \frac{dE_2}{dt}\\ \frac{dE_3}{dt}\\ \frac{dI_1}{dt}\\ \frac{dI_2}{dt}\end{pmatrix}=\left(\tfrac{S}{N}\right)F\begin{pmatrix}E_1\\E_2\\E_3\\I_1\\I_2\end{pmatrix}-V\begin{pmatrix}E_1\\E_2\\E_3\\I_1\\I_2\end{pmatrix}$$

$$=\left(\tfrac{S}{N}\right)F\begin{pmatrix}E_1\\E_2\\E_3\\I_1\\I_2\end{pmatrix}-F\begin{pmatrix}E_1\\E_2\\E_3\\I_1\\I_2\end{pmatrix}+F\begin{pmatrix}E_1\\E_2\\E_3\\I_1\\I_2\end{pmatrix}-V\begin{pmatrix}E_1\\E_2\\E_3\\I_1\\I_2\end{pmatrix}$$

$$=(\text{F-V})\begin{pmatrix}E_1\\E_2\\E_3\\I_1\\I_2\end{pmatrix}-(1-\tfrac{S}{N})F\begin{pmatrix}E_1\\E_2\\E_3\\I_1\\I_2\end{pmatrix}$$

$$\leq(\text{F}-\text{V})\begin{pmatrix}E_1\\E_2\\E_3\\I_1\\I_2\end{pmatrix}$$

The DFE is locally asymptotically stable when all the eigenvalues of the matrix F - V have negative real parts when $\rho(FV^{-1}) < 1$ or $R_0 < 1$. This is equivalent to the statement that all eigenvalues of F – V have negative real parts when $R_0 < 1$.
Consequently, $(E_1, E_2, E_3, I_1, I_2, R) \rightarrow (0, 0, 0, 0, 0, 0)$ as $t \rightarrow \infty$
Thus, by a comparison theorem, $(E_1, E_2, E_3, I_1, I_2, R) \rightarrow (0, 0, 0, 0, 0, 0)$ as $t \rightarrow \infty$
Substituting $E_B = I_B = E_T = I_T = 0$ in the model (3.4a) gives $S_1(t) \rightarrow S_1^*$ and $S_2 \rightarrow S_2^*$ as $t \rightarrow \infty$.
Thus $(S_1(t), S_2(t), E_1(t), E_2(t), E_3(t), I_1(t), I_2(t), R(t)) \rightarrow (S_1^*, S_2^*, 0, 0, 0, 0)$ as $t \rightarrow \infty$ for $R_0 < 1$. Thus DFE (E_0) is globally asymptotically stable if $R_0 < 1$.

3.2.4 Global stability of the EE

Theorem 3.6: The endemic steady (equilibrium) state is globally asymptotical stable if $R_0 > 1$.
Proof: Consider the following nonlinear Lyaponuv function using the Goh-voltera type Lyapunov function

$$L = S_1 - S_1^* - S_1^* \ln \frac{S_1}{S_1^*} + S_2 - S_2^* - S_2^* \ln \frac{S_2}{S_2^*} + E_1 - E_1^* - E_1^* \ln \frac{E_1}{E_1^*} + E_2 - E_2^* - E_2^* \ln \frac{E_2}{E_2^*} + E_3 - E_3^* - E_3^* \ln \frac{E_3}{E_3^*}$$

$$A(I_1 - I_1^* - I_1^* \ln \frac{I_1}{I_1^*}) + B(I_2 - I_2^* - I_2^* \ln \frac{I_2}{I_2^*})$$

The Lyapunov derivative given as

$$\dot{L} = (\dot{S}_1 - \frac{S_1^*}{S_1}\dot{S}_1) + (\dot{S}_2 - \frac{S_2^*}{S_2}\dot{S}_2) + (\dot{E}_1 - \frac{E_1^*}{E_1}\dot{E}_1) + (\dot{E}_2 - \frac{E_2^*}{E_2}\dot{E}_2) + (\dot{E}_3 - \frac{E_3^*}{E_3}\dot{E}_3) + A(\dot{I}_1 - \frac{I_1^*}{I_1}\dot{I}_1) + B(\dot{I}_2 - \frac{I_2^*}{I_2}\dot{I}_2)$$

$$where\ A = \frac{\beta x(S_1^* + S_2^*)}{k_1 + \mu}\ and\ \ B = \frac{\beta(S_1^* + S_2^*)}{k_2 + \mu}$$

Putting the appropriate equations from model (2.1) we have

$$\dot{L} = \mathrm{r}_1 b - \beta(xI_1 + I_2)S_1 - \mu S_1 - \frac{S_1^*}{S_1}(\mathrm{r}_1 b - \beta(xI_1 + I_2)S_1 - \mu S_1) + \mathrm{r}_2 b - \beta(xI_1 + I_2)S_2 - \mu S_2 - \frac{S_2^*}{S_2}(\mathrm{r}_2 b - \beta(xI_1 + I_2)S_2 - \mu S_2)$$

$$+[(q_{11}S_1 + q_{21}S_2)\beta(xI_1 + I_2) - (\delta_1 + \mu)E_1] - \frac{E_1^*}{E_1}[(q_{11}S_1 + q_{21}S_2)\beta(xI_1 + I_2) - (\delta_1 + \mu)E_1] + ([(q_{12}S_1 + q_{22}S_2)\beta(xI_1 + I_2) - (\delta_2 + \mu)E_2]$$

$$-\frac{E_2^*}{E_2}[(q_{12}S_1 + q_{22}S_2)\beta(xI_1 + I_2) - (\delta_2 + \mu)E_2]) + ([(q_{13}S_1 + q_{23}S_2)\beta(xI_1 + I_2) - (\delta_3 + \mu)E_3] - \frac{E_3^*}{E_3}[(q_{13}S_1 + q_{23}S_2)\beta(xI_1 + I_2) - (\delta_3 + \mu)E_3])$$

$$+\frac{\beta x(S_1^* + S_2^*)}{k_1 + \mu}(\delta_1 E_1 - (k_1 + \mu)I_1 - \frac{I_1^*}{I_1}[\delta_1 E_1 - (k_1 + \mu)I_1]) + \frac{\beta(S_1^* + S_2^*)}{k_2 + \mu}(\delta_2 E_2 + \delta_3 E_3 - (k_2 + \mu)I_2 - \frac{I_2^*}{I_2}[\delta_2 E_2 + \delta_3 E_3 - (k_2 + \mu)I_2])$$

At steady states from (3.1)

$\mathrm{r}_1 b = \beta(xI_1^* + I_2^*)S_1^* + \mu S_1^*$ and $\mathrm{r}_2 b = \beta(xI_1^* + I_2^*)S_2^* + \mu S_2^*$

Substituting the expressions for $\mathrm{r}_1 b$ and $\mathrm{r}_2 b$ above into the expression for $\dot{L}$

$$\dot{L} = \beta(xI_1^* + I_2^*)S_1^* + \mu S_1^* - \beta(xI_1 + I_2)S_1 - \mu S_1 - \frac{S_1^*}{S_1}((\beta(xI_1^* + I_2^*)S_1^* + \mu S_1^*) - \beta(xI_1 + I_2)S_1 - \mu S_1)$$

$$+\beta(xI_1^* + I_2^*)S_2^* + \mu S_2^* - \beta(xI_1 + I_2)S_2 - \mu S_2 - \frac{S_2^*}{S_2}((\beta(xI_1^* + I_2^*)S_2^* + \mu S_2^*) - \beta(xI_1 + I_2)S_2 - \mu S_2)$$

$$+[(q_{11}S_1 + q_{21}S_2)\beta(xI_1 + I_2) - (\delta_1 + \mu)E_1] - \frac{E_1^*}{E_1}[(q_{11}S_1 + q_{21}S_2)\beta(xI_1 + I_2) - (\delta_1 + \mu)E_1]$$

$$+([(q_{12}S_1 + q_{22}S_2)\beta(xI_1 + I_2) - (\delta_2 + \mu)E_2] - \frac{E_2^*}{E_2}[(q_{12}S_1 + q_{22}S_2)\beta(xI_1 + I_2) - (\delta_2 + \mu)E_2])$$

$$+([(q_{13}S_1 + q_{23}S_2)\beta(xI_1 + I_2) - (\delta_3 + \mu)E_3] - \frac{E_3^*}{E_3}[(q_{13}S_1 + q_{23}S_2)\beta(xI_1 + I_2) - (\delta_3 + \mu)E_3])$$

$$+\frac{\beta_x(S_1^* + S_2^*)}{k_1 + \mu}(\delta_1 E_1 - (k_1 + \mu)I_1 - \frac{I_1^*}{I_1}[\delta_1 E_1 - (k_1 + \mu)I_1])$$

$$+\frac{\beta(S_1^* + S_2^*)}{k_2 + \mu}(\delta_2 E_2 + \delta_3 E_3 - (k_2 + \mu)I_2 - \frac{I_2^*}{I_2}[\delta_2 E_2 + \delta_3 E_3 - (k_2 + \mu)I_2])$$

Also at steady states from (4.4a)

$\delta_1 E_1 = (k_1 + \mu)I_1^*$, $\delta_2 E_2 + \delta_3 E_3 = (k_2 + \mu)I_2^*$, $\delta_1 + \mu = \frac{(q_{11}S_1^* + q_{12}S_2^*)\beta(xI_1^* + I_2^*)}{E_1^*}$,

$\delta_2 + \mu = \frac{(q_{12}S_1^* + q_{22}S_2^*)\beta(xI_1^* + I_2^*)}{E_2^*}$, $\delta_3 + \mu = \frac{(q_{13}S_1^* + q_{23}S_2^*)\beta(xI_1^* + I_2^*)}{E_3^*}$

Substituting the expressions above we have,

$$\dot{L} = \beta(xI_1^* + I_2^*)S_1^* + \mu S_1^* - \mu S_1 - \frac{S_1^*}{S_1}(\beta(xI_1^* + I_2^*)S_1^* + \mu S_1^*) + \mu S_1^* + \beta(xI_1^* + I_2^*)S_2^* + \mu S_2^* - \mu S_2 - \frac{S_2^*}{S_2}(\beta(xI_1^* + I_2^*)S_2^* + \mu S_2^*) + \mu S_2^*$$

$$-\frac{E_1^*}{E_1}(q_{11}S_1 + q_{21}S_2)\beta(xI_1 + I_2) + (q_{11}S_1^* + q_{21}S_2^*)\beta(xI_1^* + I_2^*) - \frac{E_2^*}{E_2}(q_{12}S_1 + q_{22}S_2)\beta(xI_1 + I_2) + (q_{12}S_1^* + q_{22}S_2^*)\beta(xI_1^* + I_2^*)$$

$$-\frac{E_3^*}{E_3}(q_{13}S_1 + q_{23}S_2)\beta(xI_1 + I_2) + (q_{13}S_1^* + q_{23}S_2^*)\beta(xI_1^* + I_2^*) - \beta x(S_1^* + S_2^*)\frac{I_1^{*2}}{I_1} + \beta x(S_1^* + S_2^*)I_1^* - \beta(S_1^* + S_2^*)\frac{I_2^{*2}}{I_2} + \beta(S_1^* + S_2^*)I_2^*$$

Collecting terms with μS_1^*, μS_2^*, $\beta x I_1^* S_1^*, \beta x I_1^* S_2^*, \beta I_2^* S_1^*$ *and* $\beta I_2^* S_2^*$, we have,

$$\dot{L} = \mu S_1^*\left[2 - \frac{S_1^*}{S_1} - \frac{S_1}{S_1^*}\right] + \mu S_2^*\left[2 - \frac{S_2^*}{S_2} - \frac{S_2}{S_2^*}\right] + \beta x I_1^* S_1^*\left[3 - \frac{S_1^*}{S_1} - \frac{I_1^*}{I_1} - \frac{I_1 S_1}{I_1^* S_1^*}\sum_{i=1}^{3}\frac{E_i^*}{E_i}\right] + \beta x I_1^* S_2^*\left[3 - \frac{S_2^*}{S_2} - \frac{I_1^*}{I_1} - \frac{I_1 S_2}{I_1^* S_2^*}\sum_{i=1}^{3}\frac{E_i^*}{E_i}\right]$$

$$\beta I_2^* S_1^*\left[3 - \frac{S_1^*}{S_1} - \frac{I_2^*}{I_2} - \frac{I_2 S_1}{I_2^* S_1^*}\sum_{i=1}^{3}\frac{E_i^*}{E_i}\right] + \beta I_2^* S_2^*\left[3 - \frac{S_2^*}{S_2} - \frac{I_2^*}{I_2} - \frac{I_2 S_2}{I_2^* S_2^*}\sum_{i=1}^{3}\frac{E_i^*}{E_i}\right]$$

Since Arithmetic mean (A.M) ≥ Geometric mean (G.M), then the following inequalities hold

$$\left[2 - \frac{S_1^*}{S_1} - \frac{S_1}{S_1^*}\right] \le 0, \quad \left[2 - \frac{S_2^*}{S_2} - \frac{S_2}{S_2^*}\right] \le 0, \quad \left[3 - \frac{S_1^*}{S_1} - \frac{I_1^*}{I_1} - \frac{I_1 S_1}{I_1^* S_1^*}\sum_{i=1}^{3}\frac{E_i^*}{E_i}\right] \le 0,$$

$$\left[3 - \frac{S_1^*}{S_1} - \frac{I_1^*}{I_1} - \frac{I_1 S_1}{I_1^* S_1^*}\sum_{i=1}^{3}\frac{E_i^*}{E_i}\right] \le 0$$

$$\left[3 - \frac{S_2^*}{S_2} - \frac{I_1^*}{I_1} - \frac{I_1 S_2}{I_1^* S_2^*}\sum_{i=1}^{3}\frac{E_i^*}{E_i}\right] \le 0, \quad \left[3 - \frac{S_1^*}{S_1} - \frac{I_2^*}{I_2} - \frac{I_2 S_1}{I_2^* S_1^*}\sum_{i=1}^{3}\frac{E_i^*}{E_i}\right] \le 0 \quad \text{and}$$

$$\left[3 - \frac{S_2^*}{S_2} - \frac{I_2^*}{I_2} - \frac{I_2 S_2}{I_2^* S_2^*}\sum_{i=1}^{3}\frac{E_i^*}{E_i}\right] \le 0$$

Thus $\dot{L} \le 0$ for $R_0 > 1$

1Uwakwe, J. I" Stability Analysis And Control Of Coccidiosis Disease In Poultry." IOSR Journal of Mathematics (IOSR-JM) 14.6 (2018): 31-40.