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The effects of variation of contact rates between the environment, susceptible and infected population in a mathematical model of the transmission dynamics of Infectious Bursal Disease (IBD)

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Abstract: **We modeled a four-compartment model that explain the effects of variation of the contact rates between environment, susceptible and infected population in transmission dynamics of infectious bursal disease (IBD). We used Ordinary differential equations in formulating the model equations. Reproductive number has been derived using next generation Matrix. The disease-free equilibrium and endemic equilibrium stability are analyzed. MATLAB was used to carry out the numerical simulation. We found the higher that contact rate between the susceptible flock, infected population and the contaminated environment, the higher rate of transmission of the disease in the system.**

Keywords: **Modelling, Flock, Bursal, Infectious, environment, Contact rate, Gumboro.**

1. INTRODUCTION

Infectious bursal disease also known as Gumboro disease, is named after the place it was first discovered in 1957 in Gumboro district, Delaware in United States America (USA). It is a viral disease which is highly infectious and affects young chickens of 3-6 weeks old. A severe and acute outbreak of the disease in poultry farm causes 100% morbidity and mortality rate is also high, but the less acute or sub-clinical disease is common in 0-3-week-old birds [1].

The disease is caused by Infectious Bursal Disease Virus (IBDV), which is a member of birnavirus genus [3]. The virus consists of two segment of double-stranded ribonucleic acid (RNA), which has no envelop, which make it highly resistant to the outside environment [3]. IBD virus is highly resistant to environmental exposure and is transmitted laterally by direct or indirect contact between the environment, susceptible and infected flocks [4]. The virus is very di cult to eliminate since it is extremely hardy and can survive in a wide range of environmental conditions [3,5]. Due to the resistance nature of the virus, it is hard to clear it with most disinfectants and environmental factors, making poultry houses to be contaminated with IBD Virus that persist on the premises and tend to reappear in subsequent flock [2].

IBD is highly contagious and affects the immune system of young poultry. It destroys the lymphoid organs and particularly the bursa of fabricus of young birds, where B-lymphocyte is not mature [1,2]. According to Dey et al, IBDV infects the particularly the bursar of Fabricus actively dividing and differentiating lymphocytes of the B-cell lineage of chicks, resulting in morbidity, mortality and immunosuppression [2]. The bursa of fabricus is the assembly plant for immune system, once it is destroyed by IBD, it can no-longer produce enough B-cells which produces antibodies to help fight other secondary diseases. When chickens below 3 weeks old are infected by infectious bursal disease they show no detectable sign which is the most economically important as the disease can lead to severe long-lasting suppression of the

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immune system, while those of 3-6 weeks old are mostly susceptible to clinical symptoms of the disease. Birds infected by IBD virus shed the virus in their faeces thereby contaminating feeds, water and their house. The other birds in the house become infected by ingesting the virus [6]. IBDV remains infectious in the house for 122 days and 52 days in the feed and water respectively. Due to the hardy state of the virus, strict hygiene should be observed in poultry management and vaccination of the chickens should be done at attender age to curb the disease [7].

Infectious bursal disease virus (IBDV) has a short incubation period of two to three days, in acute cases, the birds are distressed and exhausted, prostrated, dehydrated, suffer from water diarrhoea, feathers are ruffled and soiled vent. Then followed by rapid mortality of the birds; surviving chickens recover a state of apparent health after five to seven days of infection [1.2]. According to Ganguly 2013, poultry industries suffers huge economic losses due to IBD. It leads to 100% morbidity and 90% mortality in susceptible flock [8]. If a very virulent infectious bursal disease virus (vvIBDV) strain is involved during the initial infection in the farm, the mortality rate can be 50 100% and if it is a classical strain, the virus can cause 10 50% of mortality rate in infected flocks [5,8]. If the IBD virus persist in the farm and is transmitted to successive flocks, the initial clinical sign will gradually be replaced by sub-clinical form of the disease. But still acute episode may occur in the farm [1,2]. IBD is of great concern to poultry industries, since its re-emergence in the farm in a severe virulence form can cause a highly contagious immunosuppressive disease can worsen infection caused by other viruses and bacteria.

2. MODEL DESCRIPTION AND FORMULATION

This study considers a MSIR epidemic model. The total population (N) is divided into four classes: The passive immune class (M), Susceptible (S), Infected (I) and Recovery (R). The model starts with the infant chicks which enter the passive immune class M by birth or immigration of flock at a rate of the total population, N. This compartment (M) is decreased at a rate of due to loss of immunity and due to natural death. When flock loses passive immunity, they enter the susceptible class (S) at a rate of π. In this compartment, they can get infected if they come into enough contact with the infected birds at a rate of βIS or it diminishes due to contact with the contaminated environment at rate of σ and also due to natural death at the rate of μS. Once the birds in susceptible class are infected by IBDV, they move to infected compartment thereby increasing the population of the infected flock. This class is reduced by αI, ωI Finally, the infected flock enter into recovery class, if they recover, from the disease leading the class to increase at a rate of αI and it reduces by μI as a result of natural death and μI due to recovery from the disease, death caused by infection and dying naturally respectively.

The total population with respect to all the compartments is given by;

$$N(t) = M(t) + S(t) + I(t) + R(t).$$

The schematic diagram below shows how the disease spreads

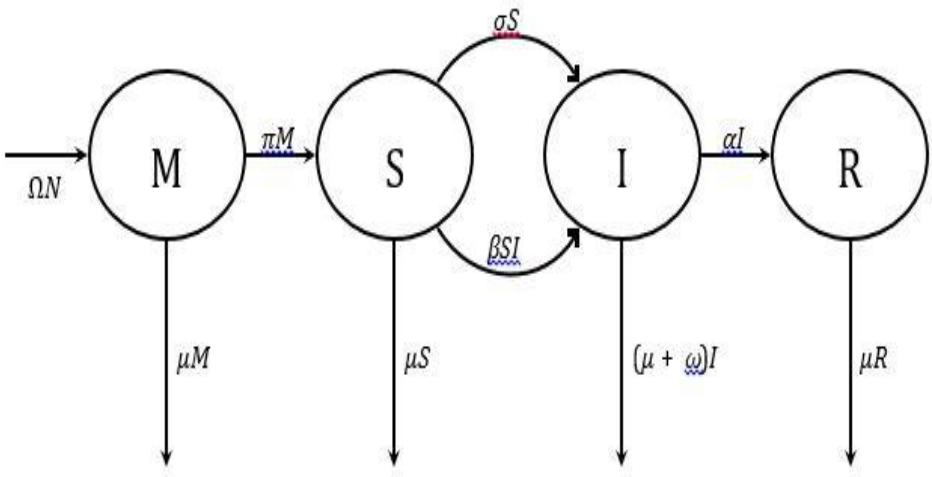

Figure 1: Schematic diagram showing the transmission dynamics of Gumboro disease

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Below are the description of the variables and parameters used.

Table 1: The variable used in the model

Variables	Description
M	Passive Immune
S	Susceptible
I	Infectious
R	Recovery

Table 2: The Parameters used in the model

Parameters	Description
Ω	Recruitment rate
μ	Natural mortality rate
β	Contact rate with infected birds
σ	Contact rate with contaminated environment
α	Recovery rate from infection
ω	Rate of mortality due IBD
π	Rate of passive immunity loss

The mathematical equations of the model can be described by a system of ordinary differential equations given below:

$$\begin{aligned}
\frac{dM}{dt} &= \Omega N - (\pi + \mu)M, \\
\frac{dS}{dt} &= \pi M - \beta IS - (\sigma + \mu)S, \qquad (1) \\
\frac{dI}{dt} &= \beta IS + \sigma S - (\omega + \alpha + \mu)I, \\
\frac{dR}{dt} &= \alpha I - \mu R.
\end{aligned}$$

And

$$\frac{dN}{dt} = (\Omega - \mu)N - \omega I$$

With initial conditions

$$M(0) = M_0 > 0, S(0) = S_0 \geq 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0$$

Assumptions

There are some assumptions made in developing the model:

1) The population is not fixed.
2) Birds get into the system by birth or immigration.
3) Chickens below two weeks old are protected by passive immunity from their mothers.
4) The population is mixed homogeneously (have the same interaction rate with one an-other).
5) Non-negative parameters are used.

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3. INVARIANT REGION

Theorem 2.1 *The closed region is positively invariant attracting all solutions.*

Proof We obtain the invariant region, in which the solution is bounded. Considering the total population (N), where;

N=M+S+I+R: (2)

Differentiating N both sides with respect to (t) we get

$$\frac{dN}{dt} = \frac{dM}{dt} + \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \qquad (3)$$

$$\frac{dN}{dt} = \Omega - (\pi + \mu) + \pi M - \beta IS - (\sigma + \mu)S + \beta IS + \sigma S - (\omega + \alpha + \mu)I + \alpha I - \mu R$$

We obtain

$$\frac{\mathrm{dN}}{\mathrm{dt}} = \Omega - (\mathrm{M} + \mathrm{S} + \mathrm{I} + \mathrm{R})\mu - \omega \mathrm{I} \qquad (4)$$

Since N=(M+S+I+R) and in the absence of the disease, the mortality due to infectious bursal disease will be equal to zero ($\omega = 0$), therefore equation (4) reduces to

$$\frac{\mathrm{dN}}{\mathrm{dt}} =\leq \Omega - \mu \mathrm{N} \qquad (5)$$

Integration both sides of (5) and simplifying we get

$$\Omega - \mathrm{N}\mu \geq \mathrm{A}e^{-\mu t} \qquad (6)$$

By applying the initial conditions t=0, N (0) = N_0 in (6) we get

$$N \leq \frac{\Omega}{\mu} - \left(\frac{\Omega - \mu N_0}{\mu}\right) e^{-\mu t} \qquad (7)$$

As $t \to \infty$ in (7), the population size $\to \frac{\Omega}{\mu}$, which implies that$0 \leq N \leq \frac{\Omega}{\mu}$

Since the study represents living flock of birds' population, all state variables remain positive all the time. The solution set which is feasible invariant is given by

$$\Lambda = \{(M, S, I, R) \in R_+^4 : 0 \leq M + S + I + R \leq \frac{\Omega}{\mu} \qquad (8)$$

Hence Λ is a positive invariant region.

4. DISEASE FREE EQUILIBRIUM

To find the Disease-free equilibrium of IBD, we set the system of equation (1) to zero. At this state, there are no infection and recovery, that is I = R = 0.

$$\frac{dM}{dt} + \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$$

Thus

$$\begin{aligned} 0 &= \Omega N - (\pi + \mu)M, \\ 0 &= \pi M - \beta IS - (\sigma + \mu)S, \\ 0 &= \beta IS + \sigma S - (\omega + \alpha + \mu)I, \\ 0 &= \alpha I - \mu R. \end{aligned} \qquad (9)$$

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From $0 = \Omega N - (\pi + \mu)M$, we get

$$M^* = \frac{\Omega}{(\pi + \mu)}$$

Also, from $0 = \pi M - \beta IS - (\sigma + \mu)S$, we get

$$S^* = \frac{\Omega\pi}{(\pi + \mu)(\sigma + \mu)}$$

Thus $E_0^* = (M^*, S^*, I^*, R^*) = (\frac{\Omega}{(\pi+\mu)}, \frac{\Omega\pi}{(\pi+\mu)(\sigma+\mu)}, 0, 0)$

5. BASIC REPRODUCTION NUMBER

We use next generation matrix (NGM) approach to determine R_0. From NGM we have

$G=FV^{-1}$

Where F is the Jacobian of f_i and is the rate of new infections in compartment I.

V is the Jacobian matrix of v_i, where v_i is the rate of transfer of infections from one compartment to another

The model equation with new ineffective class is;

$$\frac{dI}{dt} = \beta IS + \sigma S - (\omega + \alpha + \mu)I,$$

The associated matrices from the model are

$$f_i = \beta IS + \sigma S$$

$$F = \frac{\partial f_i}{\partial I} = \beta S$$

Therefore,

$$F = \frac{\beta\Omega\pi}{(\pi + \mu)(\sigma + \mu)}$$

We have $v_i = (\mu + \omega + \alpha)I$

$$V = \frac{\partial v_i}{\partial I} = (\mu + \omega + \alpha)$$

$$V^{-1} = \frac{1}{(\mu + \omega + \alpha)}$$

Thus $FV^{-1} = \frac{\beta\Omega\pi}{(\pi+\mu)(\sigma+\mu)(\mu+\omega+\alpha)}$

The basic reproductive number R_0 is the spectral radius of the matrix FV^{-1} [10].

It is given by;

$$\rho(FV^{-1}) = \frac{\beta\Omega\pi}{(\pi + \mu)(\sigma + \mu)(\mu + \omega + \alpha)}$$

Therefore, $R_0 = \frac{\beta\Omega\pi}{(\pi+\mu)(\sigma+\mu)(\mu+\omega+\alpha)}$

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6. STABILITY OF DISEASE-FREE EQUILIBRIUM

Theorem 2: *The disease-free equilibrium is globally asymptotically stable when all the eigenvalues have a negative real part for* $0 \leq t < \infty$ [9].

Proof: This theorem is proven by first obtaining the Jacobian matrix of the model system at DFE (E_0) as follows;

Let

$$X_1 = \Omega N - (\pi + \mu)M,$$
$$X_s = \pi M - \beta IS - (\sigma + \mu)S,$$
$$X_3 = \beta IS + \sigma S - (\omega + \alpha + \mu)I,$$
$$X_4 = \alpha I - \mu R.$$

$$\begin{pmatrix} \frac{\partial X_1}{\partial M} & \frac{\partial X_1}{\partial S} & \frac{\partial X_1}{\partial I} & \frac{\partial X_1}{\partial R} \\ \frac{\partial X_2}{\partial M} & \frac{\partial X_2}{\partial S} & \frac{\partial X_2}{\partial I} & \frac{\partial X_2}{\partial R} \\ \frac{\partial X_3}{\partial M} & \frac{\partial X_3}{\partial S} & \frac{\partial X_3}{\partial I} & \frac{\partial X_3}{\partial R} \\ \frac{\partial X_4}{\partial M} & \frac{\partial X_4}{\partial S} & \frac{\partial X_4}{\partial I} & \frac{\partial X_4}{\partial R} \end{pmatrix}$$

$$J(M, S, I, R) = \begin{pmatrix} -(\pi + \mu) & 0 & 0 & 0 \\ \pi & -(\mu + \sigma + \beta I) & -\beta S & 0 \\ 0 & \beta I + \sigma & \beta S - (\mu + \omega + \alpha) & 0 \\ 0 & 0 & \alpha & -\mu \end{pmatrix}$$

The Jacobian at DFE is given by the relation

$$J_0 = \begin{pmatrix} -(\pi + \mu) & 0 & 0 & 0 \\ \pi & -(\mu + \sigma) & \frac{\beta\Omega\pi}{(\pi + \mu)(\sigma + \mu)} & 0 \\ 0 & \sigma & \frac{\beta\Omega\pi}{(\pi + \mu)(\sigma + \mu)} - (\mu + \omega + \alpha) & 0 \\ 0 & 0 & \alpha & -\mu \end{pmatrix}$$

We let

$A = -(\pi + \mu)$

$B = -(\mu + \sigma)$

$C = \frac{\beta\Omega\pi}{(\pi+\mu)(\sigma+\mu)} - (\mu + \omega + \alpha)$

We then simplify and solve for λ which represent the eigen values, we obtain

$$(A - \lambda)(B - \lambda)\big((C - \lambda)(-\mu - \lambda)\big) = 0$$

We therefore obtain the eigen values as

$$\lambda_1 = -(\pi + \mu)$$
$$\lambda_2 = -(\mu + \sigma)$$
$$\lambda_3 = \frac{\beta\Omega\pi}{(\pi + \mu)(\sigma + \mu)} - (\mu + \omega + \alpha)$$
$$\lambda_4 = -\mu$$

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For DFE to be Asymptotically stable, we require $\lambda_3 < 0$

Substituting R_0 in λ_3 we obtain

$$R_0(\mu + \omega + \alpha) - (\mu + \omega + \alpha) < 0$$

This means that;

$$R_0(\mu + \omega + \alpha) < (\mu + \omega + \alpha)$$

$$R_0 < 1$$

Having shown that $R_0 < 1$, the DFE is both locally and globally asymptotically stabe.

7. ENDEMIC EQUILIBRIUM

This is a steady state solution that shows that the disease does not die in the population. Let $E^*_1 = (M^*, S^*, I^*, R^*)$ be the endemic point, where $M^*, S^*, I^*, R^* > 0$

Setting the system of equation (1) to zero and evaluating the state variables, the endemic equilibrium points would be as follows:

$$0 = \Omega - (\pi + \mu)M^*,$$

$$0 = \pi M^* - \beta I^* S^* - (\sigma + \mu), S^*$$

$$0 = \beta I^* S^* + \sigma S^* - (\omega + \alpha + \mu), I^*$$

$$0 = \alpha I^* - \mu . R^*$$

We obtain

$$M^* = \frac{\Omega}{(\pi + \mu)}$$

$$S^* = \frac{\Omega\pi}{(\pi + \mu)(\beta I^* + \sigma + \mu)}$$

$$I^* = \frac{-(1 - R_0) + \sqrt{(1 - R_0)^2 + 4\frac{\sigma}{\sigma + \mu}R_0}}{2(\frac{\beta}{\sigma + \mu})} \qquad (10)$$

$$R^* = \frac{\alpha I^*}{\mu}$$

7.1 Stability of Endemic Equilibrium

The endemic equilibrium point of the model $E_1^* = (M^*, S^*, I^*, R^*)$ as shown in equation (10)

We find that the stability of endemic equilibrium is globally asymptotically stable if by finding the eigen values they have negative real parts. Using Jacobian matrix obtained from equation (10), we obtain the eigen values as

$\lambda_1 = -(\pi + \mu), \lambda_4 = -\mu$ the characteristic equation

$$a_0\lambda^2 + a_1\lambda + a_2$$

Where

$$a_0 = 1$$

$$a_1 = \beta(I^* - S^*) + (2\mu + \omega + \alpha) > 0$$

$$a_2 = (\beta I^* + \sigma)(\omega + \alpha) > 0$$

Since all the coefficients of the characteristics equation are positive, it implies that the endemic equilibrium of the system is globally asymptotically stable.

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8. NUMERICAL SIMULATIONS

We solved the model equations in the previous chapter analytically and showed the model is locally and globally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. We used graphical presentations of numerical simulations resulting in following figures to support the analytical results.

We carried out simulations of model system (1) using MATLAB, to investigate the effect contaminated environment had on the transmission dynamics of IBD. This was achieved by use of the parameters on the tables 3 and 4 below which are assumed values.

Table 3: Variables used in the simulations.

Variables	Values
M (0)	800
S (0)	600
I (0)	150
R (0)	10

Table 4: Parameters used in the simulations.

Parameters	Values /Week
Ω	10
μ	0.001
β	0.001
σ	0.95
α	0.15
ω	0.225
π	0.565

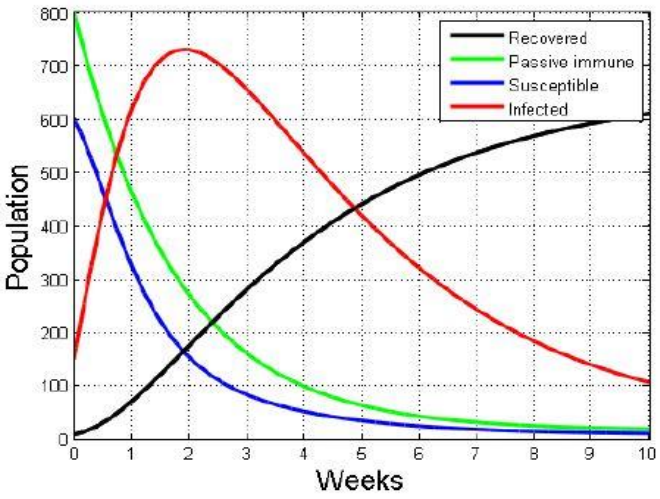

Figure 8.1: Low Contact rate of Susceptible and Infected

In Figure 8.1, we have used the following parameters in the simulation of the model system M,S,I,R of the total population when $R_0 < 1$ and the contact between environment and susceptible is high; $\Omega = 10$, $\mu = 0.001$, $\beta = 0.001$, $\sigma = 0.95$, $\alpha = 0.15$, $\omega = 0.225$, $\pi = 0.565$; $R_0 = 0.02792$.

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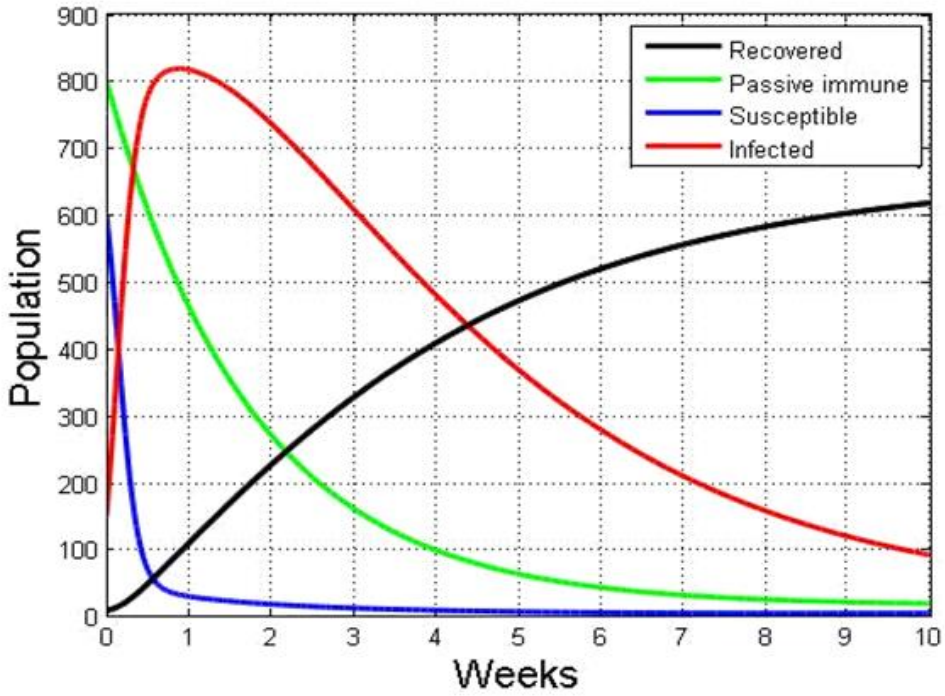

Figure 8.2: High Contact rates

In Figure 8.2, the following parameters are used in the simulation of the model system M,S,I,R of the total population when $R_0 < 1$ and the contact between environment and susceptible is high; $\Omega = 10$, $\mu = 0.001$, $\beta = 0.01$, $\sigma = 0.95$, $\alpha = 0.15$, $\omega = 0.225$, $\pi = 0.565$; $R_0 = 0.2792$.

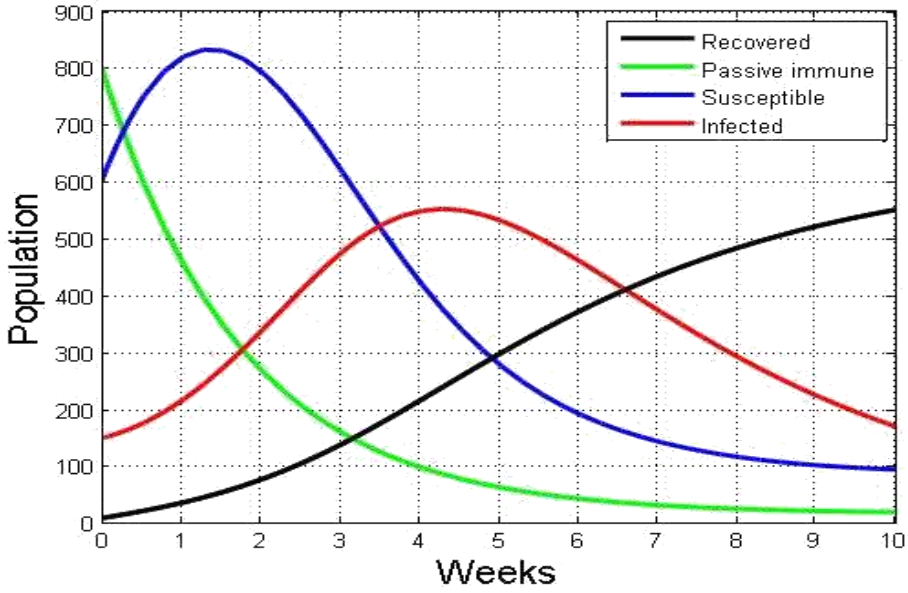

Figure 8.3: Low Contact rates

In Figure 8.3, The following parameters are used in the simulation of the model system M,S,I,R of the total population when $R_0 > 1$ and the contact between environment , infected and susceptible are low ; $\Omega = 10$, $\mu = 0.001$, $\beta = 0.001$, $\sigma = 0.001$, $\alpha = 0.15$, $\omega = 0.225$, $\pi = 0.565$; $R_0 = 13.274$.

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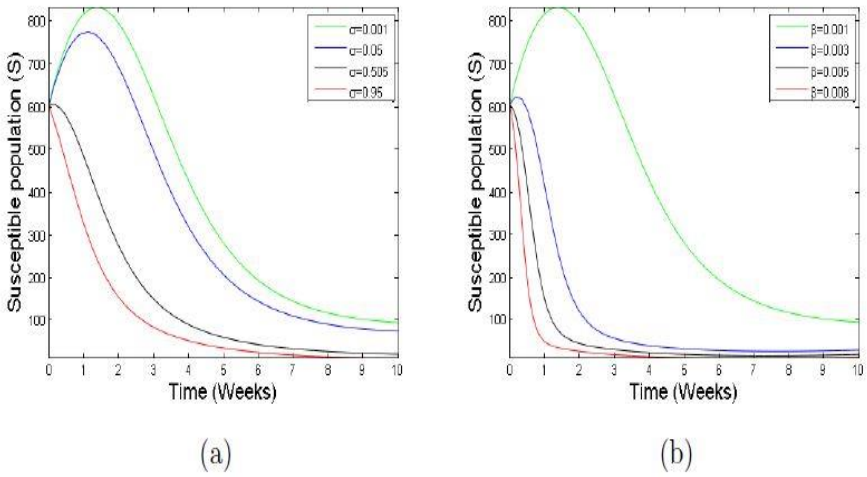

Figure 8.4: Comparing Environment and Infected

In Figure 8.4, the simulation of the susceptible population when $R_0 < 1$ and when $R_0 > 1$. In (a), the parameters used when varying σ the contact rate with the contaminated environment are: $\beta = 0.001$, $\mu = 0.001$, $\omega = 0.225$, $\pi = 0.565$, $\Omega = 10$, $\sigma =$ [0.001,0.05,0.0505,0.95]. In (b), the parameters used when varying β the contact rate between the susceptible and the infected population are: $\beta =$ [0.001,0.003,0.005,0.008], $\sigma = 0.001$, $\mu = 0.001$, $\omega = 0.225$, $\pi = 0.565$, $\Omega = 10$. In both cases when contact rates are high infection rate also increases.

9. CONCLUSION

In this paper, we developed a four compartmental model of transmission dynamics of infectious bursal disease (M, S, I, R) taking into consideration the effect of varying the contact rates between the susceptible population, infected population and the contaminated environment.

We derived the reproductive number R_0 using next generation matrix and found it to be locally and globally asymptotically stable if $R_0 < 1$. When the system is stable, it means that the spread of the disease can be controlled. For the endemic points in the model, I^* depends on the value of R_0, hence there exists unique points in the system.

From numerical simulations, we found that interaction between the susceptible, contaminated environment and the infected flock contributes in the transmission of the infectious bursal disease in the model system.

10. DATA AVAILABILITY

The data used in the analysis of the model were obtained from previously published articles and which have been cited accordingly. The parameter values are assumed. These articles are cited at relevant places within the text as references.

CONFLICT OF INTEREST:

The authors of this publication declare that there is no conflict of interest regarding the publication of this manuscript.

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