Academic Journal of Statistics and Mathematics (AJSM)
Vol.7, No.10; October - 2021;
ISSN (5730 – 7151);
p –ISSN 4052 – 392X
Impact factor: 4.93

SENSITIVITY ANALYSIS OF FOWL POX INFECTION

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Abstract: *In this study, a Susceptible, Exposed, Infected and Removed Mathematical model of fowl pox infection in poultry was formulated in order to capture possible effects of the disease. The Next-Generation matrix was used to obtain the reproduction number of the infection. We carried out sensitivity analysis on the model parameters in respect to the calculated reproduction number. The analysis showed that* α*, the transmission rate of the infection is the most sensitive parameter that increases the rate of transmission of the disease followed by the proportion of susceptible birds infected latently unnoticed,* θ*. The third most sensitive parameter was found to be the rate at which the exposed birds become infectious while the least sensitive parameter being* μ*, rate at which the treated infected birds join the susceptible class. This analysis is implemented graphically through maple 17.*

Keywords: susceptibility, latent, sensitivity analysis, infection and transmission

INTRODUCTION

Fowl pox, also known as avian pox, is a relatively slow spreading viral infection prevalent among chickens, turkey, pigeons and canaries worldwide. It is classified into two forms namely, the cutaneous (dry pox) and the diphtheritic (wet pox) forms. The infection leads to decline in egg production, weight loss in birds and can cause high mortality of 50-60% in unvaccinated birds (Udofia and Inyama, 2011a).

The primary method of transmission is mechanical transmission, which occur through injured skin or wounds. Mechanical vectors such as flies and other insects can carry pox virus and deposit it on susceptible birds. Infected mosquitoes' bite, results in rapid spread of pox virus throughout the farm. Infection may occur in the absence of injury because of the high susceptibility of the virus in the mucous membrane. In contaminated poultry houses, the infection can spread easily from bird to bird, cage to cage and by standing water in drinking cups

Office International Des Epizooties (OIE) (2008) stated that fowl pox is caused by a DNA virus classified in the set of closely related species called avipoxvirus. Avipoxviruses (or avian pox viruses) are large double stranded DNA viruses that multiply in the cytoplasm of the host cell. It is a subfamily of a family called poxviridae. Poxviridae are viruses that can as a family infect both vertebrate and invertebrate animals. About 232 species of birds are known to have been infected with avian pox virus

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(Pledger, 2005). Fowl pox is not curable but can be prevented by vaccination (Lee, 2012).

Mathematical models can help to reveal the progress of diseases and the likely outcome of epidemics. This helps in policy formulation aimed at controlling/eradicating its impact.

Julia (2009) investigated the relationship between vectors, pathogens and hosts in order to gain insight on the pathogen transmission. The study showed evidence suggesting that a mosquito has the ability to suppress the immune response of the host at the feeding site. This creates an opportunity for the pathogen to spread within the host without initial recognition by the immune system of the host. This is made possible because of the presence of molecules in the saliva of the vector or surface coats of the pathogen that suppresses the immune function of the host, through delay response or complete inhibition. However, the study suggested the possibility of an improved immune response by the host to the virus due to earlier exposure to mosquitoes. According to Silva *et al.* (2009), fowl pox is readily diagnosed by veterinarians based on flocks history and the presence of lesions. They further stated that in some cases, microscopic examination of the affected tissues may be necessary. Lee (2012) emphasized on baby chicks' vaccination. He suggested that broiler chicks hatched from August through November should be vaccinated for fowl pox by 14 days of age. He also stated that in areas with high mosquito populations, Turkey should be vaccinated initially between 3 to 4 weeks of age, followed by a second vaccination at 12 weeks of age to ensure lasting immunity. Butcher and Rossi (2012) stressed that the success of fowl pox vaccination depends on a vaccine that produces takes. A take consists of swelling of the skin or a scab at the site where the vaccine was applied. They stated that it is highly desirable to examine vaccinated birds for takes about 7-10 days after vaccination.

MODEL ASSUMPTIONS

(i) The chicks are vaccinated few days after being hatched,
(ii) Infected birds are treated,
(iii) Treated birds can join the exposed or treated population class,
(iv) Individualbirds die by infection and other causes,
(v) The exposed birds are not treated as such they do not join the susceptible population class,
(vi) The removed population is quarantined (they die off after some time),
(vii) A proportion of the susceptible birds that become exposed may be infectious with symptoms at latent state unnoticed,
(viii) The exposed birds that recover from the disease after treatment can join the susceptible population.

MODEL FORMULATION

Table 1; The model parameters and variables are as defined below;

S/No	Parameters and Variables	Description
1	S	Susceptible population of birds at time t
2	E	Exposed population of birds at time t
3	I	Infected population of birds at time t
4	R	Removed population of birds at time t,
5	β	Recruitment term (new birds born or brought into the susceptible class)

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6	α	Transmission rate of infection
7	μ	Rate at which the treated infected join the susceptible class
8	r_1	The removal rate of infected birds that don't respond to treatment
9	d_1	Death rate due to infection
10	d_2	Death rate of the removed population class
11	θ	Proportion of susceptible birds infected latently unnoticed
12	1- θ	Proportion of infected birds with disease at latent state $(\theta + 1 - \theta = 1)$
13	c_1	The rate at which the exposed birds become infectious
14	c_2	The rate at which the exposed birds recover
15	α_0	Death rate due to other causes such predation, harsh weather, poor housing and nutrition

Th

THE MODEL EQUATIONS

Based on the assumptions, parameters and variables, the modified SEIR model is given as follows:

$$\frac{dS}{dt} = \beta - \theta\alpha SI(1-\theta)\alpha SI + \mu I + c_2 E - \alpha_0 S \quad (1)$$

$$\frac{dE}{dt} = (1-\theta)\alpha SI - (c_1 + c_2 + \alpha_0)E \quad (2)$$

$$\frac{dI}{dt} = \theta\alpha SI + c_1 E - (d_1 + \mu + r_1 + \alpha_0)I \quad (3)$$

$$\frac{dR}{dt} = r_1 I - d_2 R \quad (4)$$

$$\beta = r + \delta \quad (5)$$

THE DISEASE FREE EQUILIBRIUM STATE OF THE MODEL

At the equilibrium state, $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$

Lemma 1.0

Given , $\alpha, \theta, (1-\theta), \mu, r_1, c_1, c_2, d_1, d_2, \alpha_0 > 0$,
it can be solved simultaneously from equations (1) to (5), that, there exist a disease free equilibrium state $(S, E, I, R) = (S^*, 0,0,0)$ where $S^* = \frac{\beta}{\alpha_0}$.

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THE BASIC REPRODUCTION NUMBER

Diekmann*et al.* (2010) defined the basic reproduction number, R_0 as the average number of new cases of an infection caused by one typical infected individual in a population consisting of susceptible only. They stated that the reproduction number is the most important and most urgently estimated quantity for emerging infectious diseases in outbreak situations. Its value provides insight when designing control strategies for established infections. The reproduction number is also defined as the dominant eigenvalue of the next generation matrix,G. $G$is comprised of two parts: $F$and V^{-1}.

$$F = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right] \quad (6)$$

$$V = \left[\frac{\partial V_i(x_0)}{\partial x_j}\right] \quad (7)$$

where the F_i are the new infections, while the V_i, transfer of infections from one compartment to another. $x_0$is the disease free-equilibrium state. $R_0$is the dominant eigenvalue of $G = FV^{-1}$. The ijth entry of G i.e. gij is the expected number of secondary infections of type i caused by a single infected individual of type j assuming that the population of type i is entirely susceptible. $G$is a non-negative square matrix which guarantees the presence of a single eigenvalue which is positive, real and strictly greater than all the others. The reproduction number is given by the spectral radius of G known as the dominant eigenvalue (James, 2007). Suppose the infection is divided into n disease stages.

Let $x(t)$ be the vector population in each stage.

$$x(t) = F_{ij}x(t) - V_{ij}x(t) \quad (8)$$

where$F$ is the transmission matrix whose (i,j) entry is the number of new infections at stage j caused by contact with diseased individual at stage i. $V$is the transition matrix whose (i,j) entry is the rate individuals in stage j progress to stage i (Udofia and Inyama, 2011a).

Using equation(8)

$$\begin{pmatrix}\dot{E}\\ \dot{I}\end{pmatrix} = \begin{pmatrix}0 & (1-\theta)\alpha S\\ 0 & \theta\alpha S\end{pmatrix}\begin{pmatrix}E\\ I\end{pmatrix} - \begin{pmatrix}c_1 + & c_2 + & \alpha_0 & 0\\ & c_1 & & d_1 + \mu + r_1 + \alpha_0\end{pmatrix}\begin{pmatrix}E\\ I\end{pmatrix} \quad (9)$$

$$F = \begin{pmatrix}0 & (1-\theta)\alpha S\\ 0 & \theta\alpha S\end{pmatrix} \quad (10)$$

$$V = \begin{pmatrix}c_1 + & c_2 + & \alpha_0 & 0\\ & c_1 & & d_1 + \mu + r_1 + \alpha_0\end{pmatrix} \quad (11)$$

Simplifying appropriately yields the following rsults;

$$R_0 = \frac{(C_1 + C_2\theta + \alpha_0\theta)}{(C_1 + C_2 + \alpha_0)(d_1 + \mu + r_1 + \alpha_0)} \quad (12)$$

Sensitivity analysis of R_0

According to Helana*et al.* (2013), sensitivity indices allow us to measure the relative changes in a variable when a parameter changes. They stated that the normalised forward sensitivity index of a variable with respect to a parameter is the ratio of

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the relative changes in the variable to the relative changes in the parameter. Therefore, given equation (12), the normalised forward sensitivity index of R_0, that depends differentially on the parameters $\alpha,\ \theta,\ c_1,\ \alpha_0,\ d_1,\ \mu,\ r_1$ and c_2 are presented on Table 2 below. Recall that;

$$R_0 = \frac{\alpha S(c_1+c_2\theta+\theta\alpha_0)}{(c_1+c_2+\alpha_0)(d_1+\mu+r_1+\alpha_0)}.$$

The following are assumed values as there were no available experimental data for the modified model at the time of this work.

$$S = 200,\ \alpha = 0.05,\ \theta = 0.3,\ c_1 = 0.6,\ \alpha_0 = 0.02,\ d_1 = 0.2,\ \mu = 0.5,$$
$$r_1 = 0.3, c_2 = 0.4$$

The results in Table 2 are generated using Maple 17 software. See Appendix A for details of results for Table 5 and for another set of sensitivity indices obtained by varying α (the most sensitive parameter) from 0.05 to 0.1 while other parameters values are the same as above.

Table 2: Sensitivity indices of R_0 with respect to the parameters

Parameters	Values	Relative changes	Sensitivity indices
α	0.05	$\frac{\partial R_0}{\partial \alpha} \times \frac{\alpha}{R_0}$	1
θ	0.3	$\frac{\partial R_0}{\partial \theta} \times \frac{\theta}{R_0}$	0.1735537190
c_1	0.6	$\frac{\partial R_0}{\partial c_1} \times \frac{c_1}{R_0}$	0.2382109869
d_1	0.2	$\frac{\partial R_0}{\partial d_1} \times \frac{d_1}{R_0}$	−0.1960784314
r_1	0.3	$\frac{\partial R_0}{\partial r_1} \times \frac{r_1}{R_0}$	−0.2941176471
α_0	0.02	$\frac{\partial R_0}{\partial \alpha_0} \times \frac{\alpha_0}{R_0}$	−0.03095122344
μ	0.5	$\frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0}$	−0.4901960784
c_2	0.4	$\frac{\partial R_0}{\partial c_2} \times \frac{c_2}{R_0}$	−0.2268676065

Below are graphs of R_0 against the parameters $\alpha,\ \theta,\ c_1,\ d_1,\ r_1,\ \alpha_0,\ \mu$ and c_2. The objective is to view changes in the basic reproduction number with respect to the parameters. From the sensitivity indices above, α, the transmission rate of the infection, has the greatest sensitivity index. For this reason, we shall vary the numerical value of α from $\alpha = 0.05$ to $\alpha = 0.1$ (see Appendix A (ii) for the sensitivity indices at $\alpha = 0.1$) in order to view how that may affect the disease state or

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changes in R_0. Therefore, Figures 1, 2, ..., 8 show the relationship of R_0 with each parameter when (a) $\alpha = 0.05$ (b) $\alpha = 0.1$.

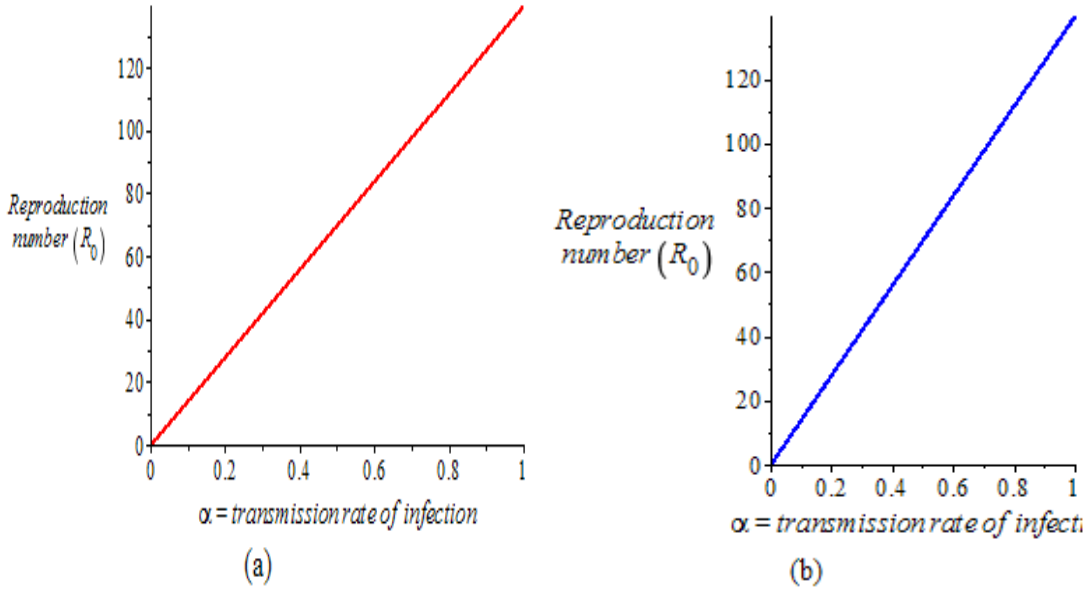

Figure 1:R_0 verses α when (a) $\alpha = 0.05$, (b) $\alpha = 0.1$

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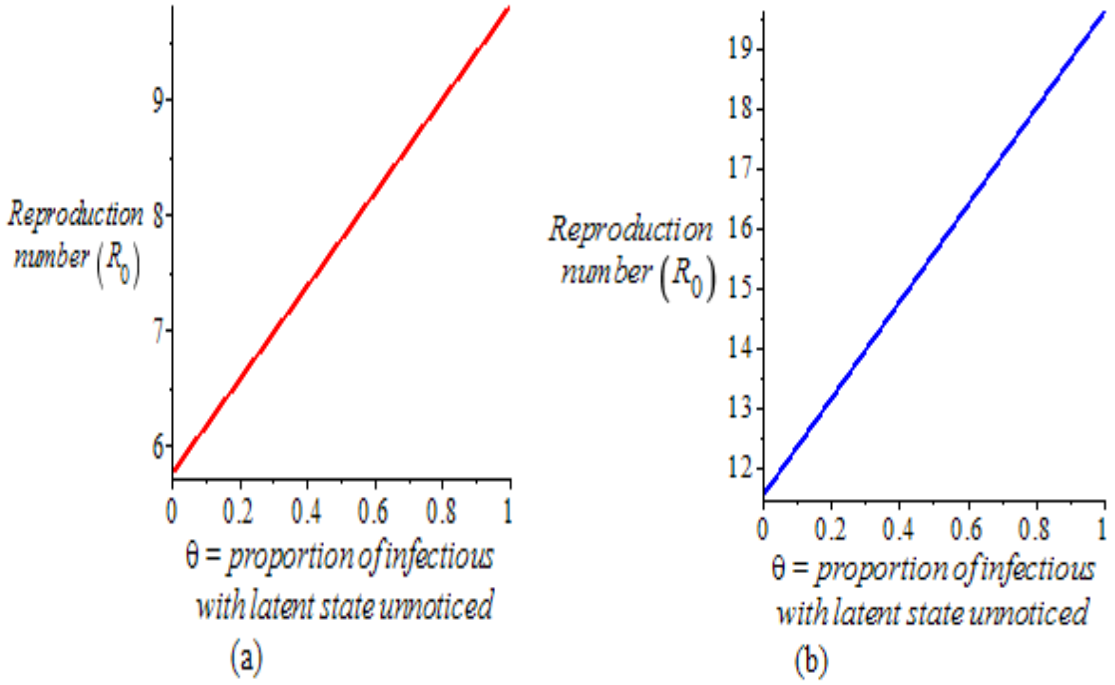

Figure 2:R_0 verses θ when (a) $\alpha = 0.05$, (b) $\alpha = 0.1$

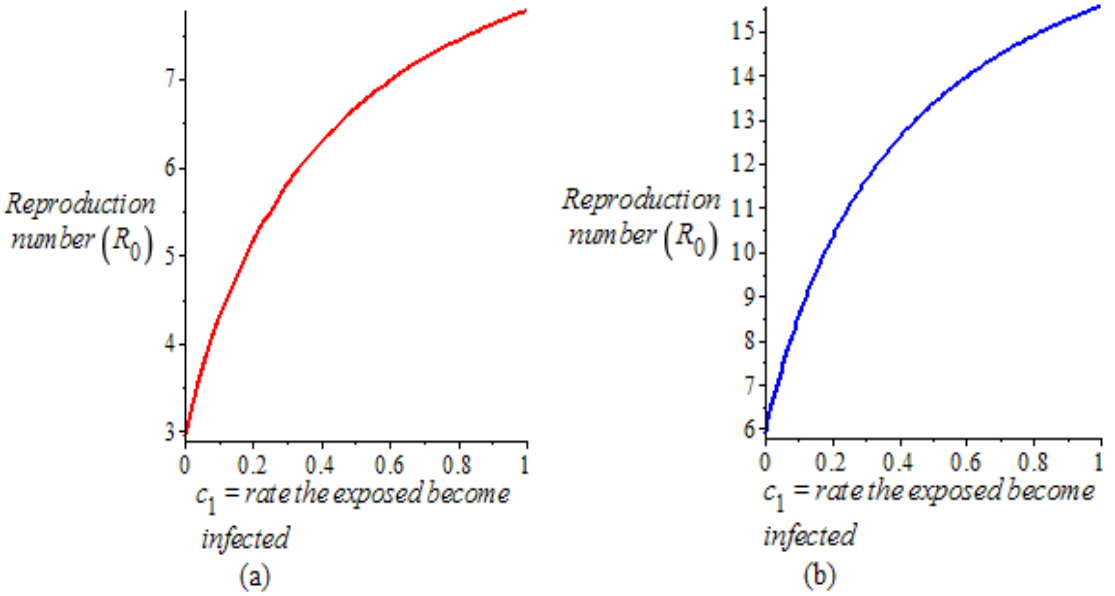

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Figure 3:R_0 verses c_1 when (a) $\alpha = 0.05$, (b) $\alpha = 0.1$

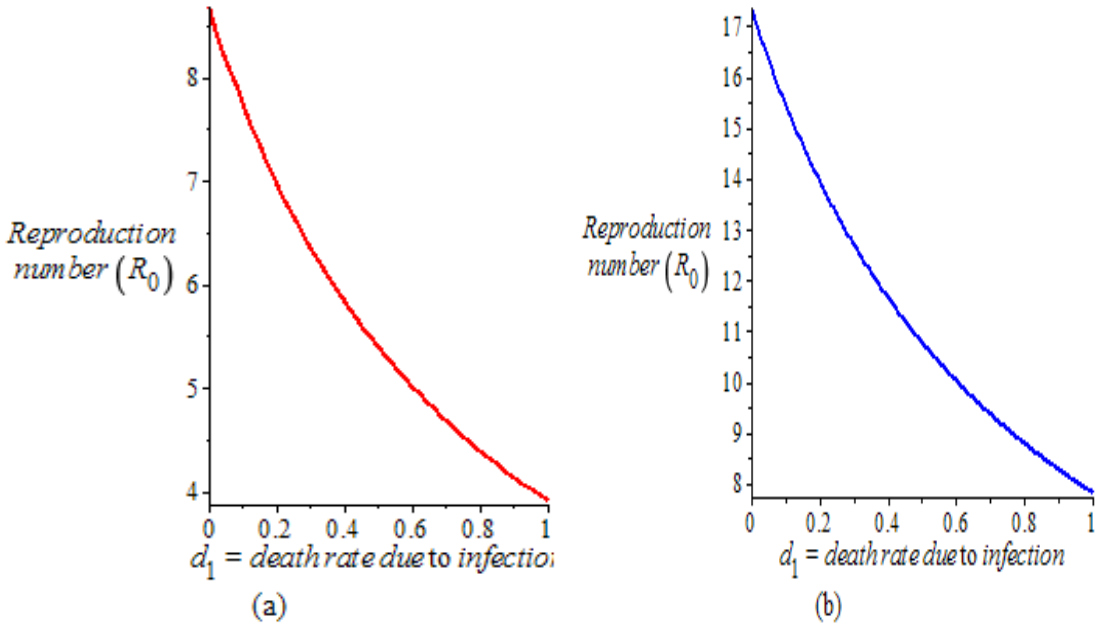

Figure 4:R_0 verses d_1 when (a) $\alpha = 0.05$, (b) $\alpha = 0.1$

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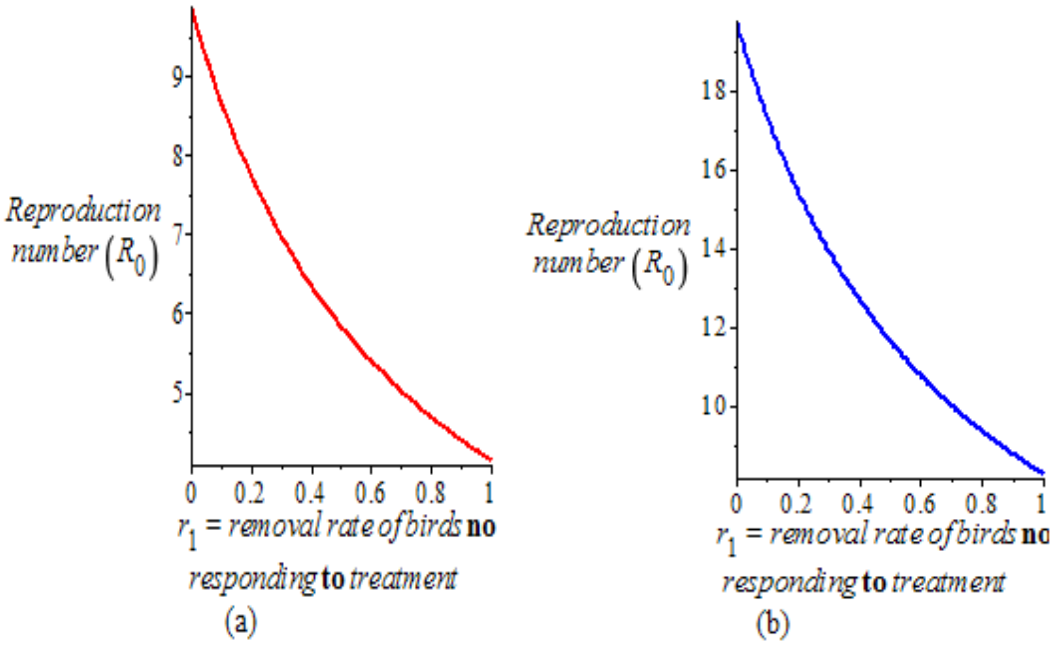

Figure 5:R_0 verses r_1 when (a) $\alpha = 0.05$, (b) $\alpha = 0.1$

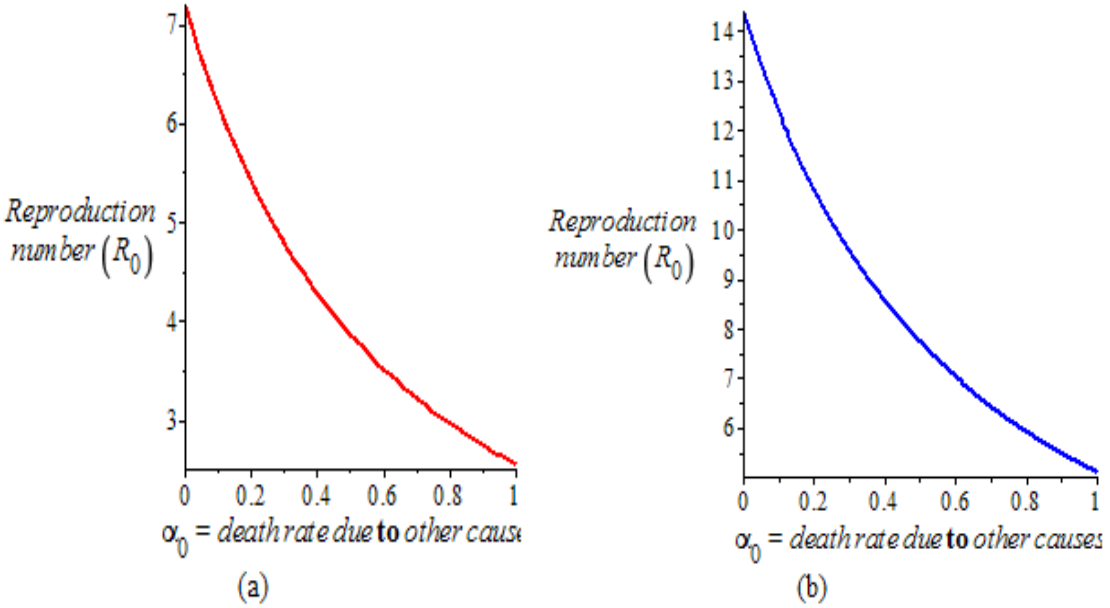

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Figure 6:R_0 verses α_0 when (a) $\alpha = 0.05$, (b) $\alpha = 0.1$

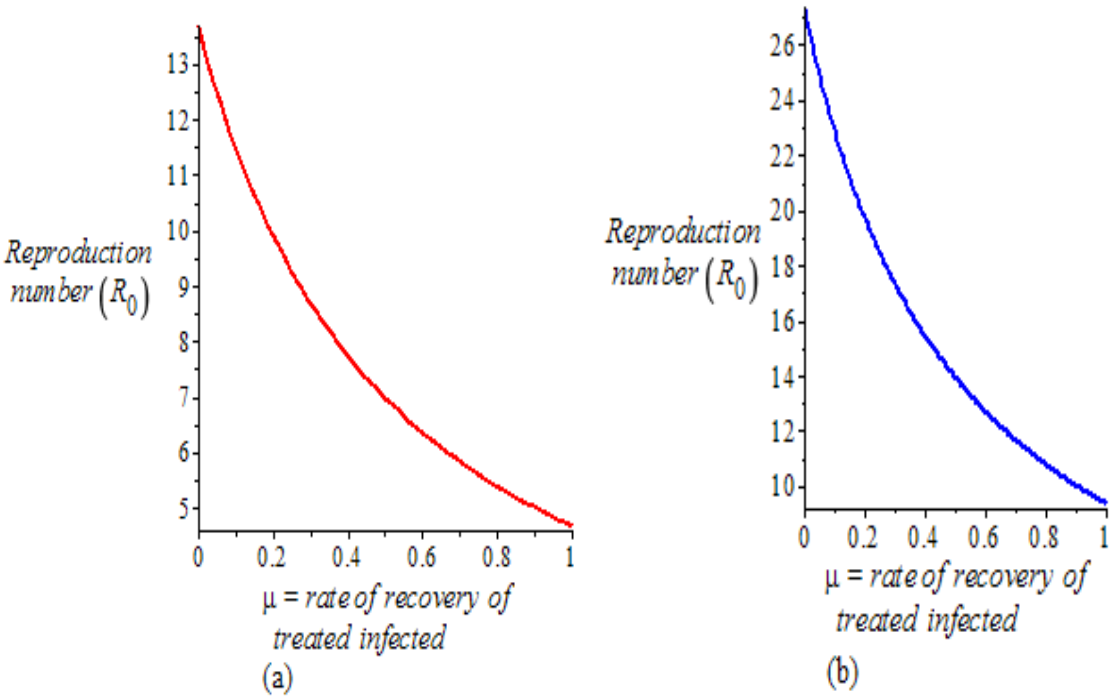

Figure 7:R_0 verses μ when (a) $\alpha = 0.05$, (b) $\alpha = 0.1$

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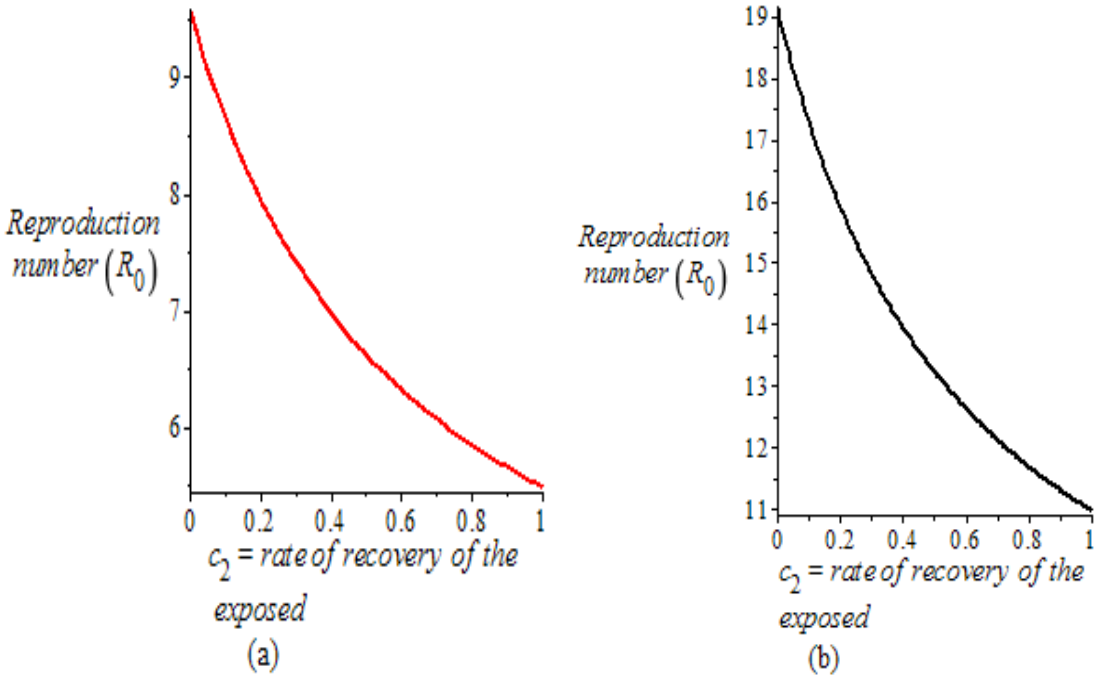

Figure 8:R_0 verses c_2 when (a) $\alpha = 0.05$, (b) $\alpha = 0.1$

DISCUSSION OF RESULTS

We hereby, discussed the result of the sensitivity analysis carried out during the study. The result is as displaced in table 2 and also simulated evident in figures one to eight. The discussion is as follows;

Figure1, shows that α, the transmission rate of the infection is the most sensitive parameter of the infection. Figure1 shows that R_0 is an increasing function of α. This agrees with reality in that, as more birds are infected, the more their chances of infecting others, thus increasing the basic reproduction number, R_0.

The result of figure 2 shows that R_0 is an increasing function of θ, the rate at which the susceptible become infectious with symptoms at the latent state unnoticed. It is also true to say that the more the susceptible birds become infected with latent state unnoticed the more their chances of infecting others.

Figure 3 also shows that R_0 is an increasing function of c_1, with decline in the increase as time proceed. This also reflects reality in that, the more the exposed birds, the more the number that become infectious. At the exposed state, the birds cannot infect others. Also, as they become infectious and are moved to the infective class, there is no or little chance of them infecting other birds. These reasons and the recovery of some exposed birds through natural immunity, suggest the reasons for decline in the increase of R_0 as time proceed.

The graphs in figure 4 reveals that R_0 is a decreasing function of d_1, the rate at which the infected birds die due to the disease.This reflects the fact that as infected birds die due to the infection, the chances of other birds becoming infected is reduced.

Here in figure 5, R_0 is a decreasing function of r_1, the removal rate of the birds that do not respond to treatment. $R_0$is a decreasing function because the removed class of

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birds have no or less chances of infecting other healthy birds.

Figure 6 explains the fact that R_0 is a decreasing function of α_0, the death rate of the susceptible, exposed and infected birds due to other causes. The decrease is as a result of the fact that these birds cannot infect others as it is assumed that they die due other causes not fowl pox.

In figure 7, the result shows that R_0 is a decreasing function of μ, the recovery rate of the treated infected. This result supports the assumption that treated infected birds can join the susceptible and the literature that recovered birds cannot be re-infected by the disease at the period of the epidemic. Thus, R_0 is decreasing with respect to μ because the treated recovered cannot infect other birds and cannot be re-infected at the period of the epidemic.

Figure 8 entails that R_0 is also a decreasing function of c_2, the recovery rate of the exposed birds. This reflects the reality that exposed birds are not infectious and once a bird recovers, it cannot be re-infected at the period of the epidemic and thus, decreasesR_0.

CONCLUSION

The analysis in the research work show that the parameters to tackle as built in the model that will increase the infectivity in the system are the transmission rate, Proportion of infectious with latent state unnoticed and the rate of those exposed becoming infected.

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