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A Mathematical Model of Avian Influenza for Poultry Farm and its Stability Analysis

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Abstract

This paper aims to estimate the basic reproduction number for Avian Influenza outbreak in local and global poultry industries. In this concern, we apply the SEIAVR compartmental model which is developed based on the well-known SEIR model. The SEIAVR model provides the mathematical formulations of the basic reproduction number, final size relationship and a relationship between these two phenomena. The developed model Equations are solved numerically with the help of Range-Kutta method and the values of initial parameters are taken from the several literatures and reports. The calculated result of basic reproduction number shows that it is locally and globally stable if it is less than and greater than one at disease free equilibrium and at endemic equilibrium, respectively. Furthermore, we have compared among the calculated susceptive, expose, infective, removal, virus and asymptotic compartments where infection rate and expose period are observed very sensitive compared to other parameters. In addition, the model result of infective is compared with the field data and other's model where the present model shows good performance against the field data.

Keywords: Avian influenza; Poultry farm; SEIAVR model; Basic reproduction number; Stability analysis

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1. Introduction

Avian influenza, is known informally as avian flu or bird flu, is the common disease for birds and animals caused by the virus and though avian flu is adapted to birds, it can also stably adapt and sustain animal-to-animal and person-to-person transmission. The outbreak of this flu occurs almost in every year all over the World and death can be observed at several countries. Bird flu occurs not only for death of the bird but also for human death. Iwami et al. (2007) observed the high death rate for highly pathogenic avian influenza and that was about 100% for birds and 70% for humans. Low pathogenic avian influenza also had breakouts in some countries and some area, such as in Germany, Sweden, America and Bangladesh. In past 2-3 years, bird flu was occurred in Bangladeshi poultry farm where the transmission was seen from bird to bird and developed into pandemic one (Biswas et al. 2008). Biswas et al. (2008) determined the attack rate of outbreaks epidemiology of avian influenza virus in chickens in Bangladesh and it's range was from 0.004-0.008. In 2014, avian influenza was critically infected in China and birds as well as Humans death were occurred there. Avian influenza is also common diseases which makes a catastrophic disaster in poultry farm in Bangladesh and causes vast loss in economic. However, research on bird flu in developing country, especially in Bangladesh is very limited yet. Because biosecurity is very poor due to economic conditions, graphical situations, and awareness about the diseases and take care of birds, etc.

Many researchers developed the mathematical formulae and investigated the avian influenza outbreak (Brauer et al. (2008); Wang et al. (2015); Tiwari et al. (2006); Brown et al. 2009; Bulaga (2003); Biswas et al. (2008); Rahman et al. (2012)). Brauer et al. (2008) discussed a model of seasonal influenza and determined the final size relation of the epidemic models, but environmental (virus compartment) class was not included. Wang et al. (2015), Tiwari et al. (2006) and Kung et al. (2003) showed that avian influenza virus was demonstrably presence in the environment, and this could be transmitted direct or indirect to the poultry farm. Wang et al. (2015) also observed a model in the presence of the avian influenza (H7N9) virus and showed that it oscillated seasonally with the point of peaks in spring and winter seasons. Brown et al. (2009) showed that the temperature, pH and salinity played a significant role in the transmission of avian influenza viruses in water and subsequently to the poultry farm. Rahman et al. (2012) observed that an avian influenza had the incubation period after attacks the bird and took some time to show the symptoms. They also added that the incubation periods for the disease were from 2 to 15 days. Seasonality also plays an important role for infectious diseases [Ratchagar et al. (2015)].

Further, Lin et al. (2016) fitted a simple epidemic model for avian influenza considering environmental compartment for the virus in the environment and this virus was proportional to individuals but avoided asymptomatic class. Longini et al. (2004) found that there was a significant portion of individuals those were infected but never exhibited any symptoms through the asymptomatic period. Kanamori et al. (2016) found that 5.2% to 35.5% birds were asymptomatic (absence of symptom) that is, they didn't show any symptoms of avian influenza virus. Mouaouine et al. (2018) discussed the stability of the SIR epidemic model based on the basic reproduction number at two equilibrium points (disease-free and endemic). Similar results were found by Dubey et al. (2015) for SIR model with the nonlinear incidence of transmitting diseases. Huang (2018) represented a global stability of an epidemic model on multiplex network and found the similar result of the stability of the model as Mouaouine et al. (2018). Kang et al. (2019) proposed a model on an avian influenza and its transmission to virus compartment and showed how to control and minimize the outbreak from birds to

human. Li et al. (2018) described a dynamical transmission of the bird flu between poultry and human and they investigated the local and global stability of the two equilibriums and found the similar results as shown by Mouaouine et al. (2018), Huang (2018), Kang et al. (2019) and Li et al. (2009).

From the above literatures and discussion, it is clear that the outbreak of the avian influenza and stability analysis of the model's results were investigated by several researchers. However, the model (SEIAVR) considering six compartments at a time is a new approach in this study and hope it will work well for the poultry farm. In addition, this study also will highlight the basic reproduction number and final size relation of the population which will provide the control system and awareness of the outbreak of an avian influenza.

2. Mathematical Formulation

2. 1. Model Description

The SEIAVR compartmental model is developed based on six individual compartments. The compartments are: Susceptible (S), Expose (E), Infective (I), Asymptomatic (A), Removal (R) and Virus compartment (V). According Lin et al. (2016), all these compartments are depended on infective population. A diagram of this model is given in Figure 1.

We assume that β is the transmitted rate to the susceptible due to Infection (I), Asymptomatic (A) and virus compartment (V). Further, we assume that p is a portion of expose members proceeds to the in the infective compartment at the rate σ, and the remainder portion of population goes to an asymptomatic compartment directly at the rate σ. Asymptomatic has infectivity reduced and goes to the removal compartment at the rate η. We also assume that the infected individuals go up, the number of viruses shaded in the virus compartment which is also go up and this virus is proportional to the infected individual. Furthermore, τ is the rate at which virus copies imported and a rate μ is the virus removed in the environmental class. Let infected members go to the removal compartment directly by a rate γ and at a rate λ, removal individual goes to susceptible.

These above assumptions lead to the mathematical model as

$$\dot{S} = \lambda R - \beta S(I + A + V), \tag{1}$$

$$\dot{E} = \beta S(I + A + V) - \sigma E, \tag{2}$$

$$\dot{I} = p\sigma E - (\tau + \gamma + \mu)I, \tag{3}$$

$$\dot{A} = (1 - p)\sigma E - \eta A, \tag{4}$$

$$\dot{V} = \tau I - \mu V, \tag{5}$$

$$\dot{R} = \gamma I + \eta A - \lambda R, \tag{6}$$

with the initial conditions:

$$S(0) = N, I(0) = I_0, E(0) = 0, A(0) = 0, V(0) = 0, R(0) = 0. \quad (7)$$

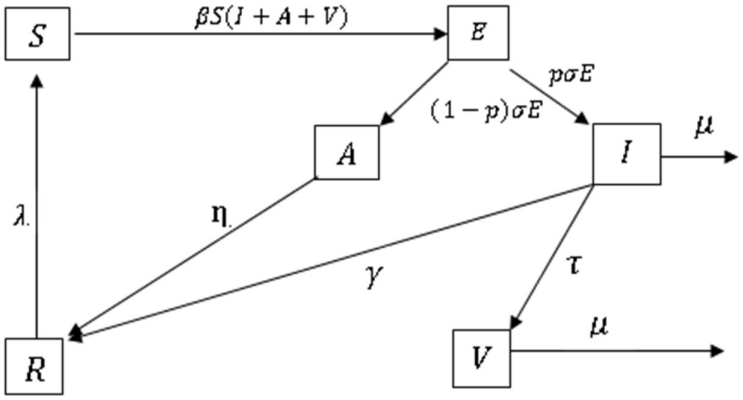


Figure1. Sketch of the compartmental model described by the Equations (1) – (6)

2.2. Disease-free equilibrium

For diseases-free equilibrium, an equilibrium solution of the systems of Equations (1) – (6)

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dA}{dt} = \frac{dV}{dt} = \frac{dR}{dt} = 0$$

can be found with aid of $E = I = 0$. After solving the above system and using the initial conditions, we have $A = T = R = 0$, where we set $S = S_0$. Thus, diseases-free equilibrium becomes $(S_0, 0, 0, 0, 0, 0)$.

Theorem 2.2.1.

The basic reproduction number of the propose model can be expressed as

$$\Re_0 = \beta S_0 \left[\frac{p}{\gamma + \tau + \mu} + \frac{1-p}{\eta} + \frac{p\tau}{\mu(\gamma + \tau + \mu)}\right].$$

***Proof*:**

From the above proposed model, we can separate this into the disease and non-disease compartments which can be expressed as:

For disease compartment

$$\dot{E} = \beta S(I + A + V) - \sigma E,$$

$$\dot{I} = p\sigma E - (\tau + \gamma + \mu)I,$$

$$\dot{A} = (1-p)\sigma E - \eta A,$$

$$\dot{V} = \tau I - \mu V.$$

For non-disease compartment

$$\dot{S} = \lambda R - \beta S(I + A + V),$$

$$\dot{R} = \gamma I + \eta A - \lambda R.$$

In general, the above model can be written in the tensor form as

$$\frac{\partial x_i}{\partial t} = f_i(x_i, y_i) - v_i(x_i, y_i),$$

$$\frac{\partial y_i}{\partial t} = g_i(x_i, y_i),$$

where x and y be the sub-populations in disease and non-disease compartments. The functions f_i and v_i are rate of secondary infections only that increase the i^{th} disease compartment and the rate of other progression such as death, recovery that decrease the i^{th} disease compartment respectively, and are given by

$$f = \begin{bmatrix} \beta S(I + A + V) \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad \text{and} \quad v = \begin{bmatrix} \alpha E \\ -p\alpha E + \tau I + \gamma I + \mu I \\ -(1-p)\alpha E + \eta A \\ -\tau I + \mu V \end{bmatrix}.$$

The basic reproduction number can be determined using the next generation matrix $k = F / V_1$ and basic reproduction number is the positive eigenvalue of the matrix k at disease free equilibrium. To calculate the basic reproduction number, we have used the well-known method of Driessche et al. (2002) which is associated next-generation matrices. That is,

$$F = \frac{\partial f_i}{\partial x_j}(0, y_0) \text{ and } V_1 = \frac{\partial v_i}{\partial x_j}(0, y_0)$$

gives us

$$F = \begin{bmatrix} 0 & \beta S_0 & \beta S_0 & \beta S_0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \text{ and } V_1 = \begin{bmatrix} \sigma & 0 & 0 & 0 \\ -p\sigma & \tau + \gamma + \mu & 0 & 0 \\ -(1-p)\sigma & 0 & \eta & 0 \\ 0 & -\tau & 0 & \mu \end{bmatrix}.$$

According to Driessche et al. (2002), the basic reproduction number can be determined by the spectral radius of FV_1^{-1}. Thus, we obtain

$$\Re_0 = \beta S_0 \left[\frac{p}{\gamma + \tau + \mu} + \frac{1-p}{\eta} + \frac{p\tau}{\mu(\gamma + \tau + \mu)} \right].$$

Theorem 2. 2.

The disease-free equilibrium of the model is locally asymptotically stable if $\Re_0 < 1$, and unstable $\Re_0 > 1$.

***Proof*:**

The Jacobian matrix J of the systems of Equations (1) – (6) is given by

$$J = \begin{bmatrix} \frac{d\dot{E}}{dE} & \frac{d\dot{E}}{dI} & \frac{d\dot{E}}{dA} & \frac{d\dot{E}}{dV} & \frac{d\dot{E}}{dS} & \frac{d\dot{E}}{dR} \\ \frac{d\dot{I}}{dE} & \frac{d\dot{I}}{dI} & \frac{d\dot{I}}{dA} & \frac{d\dot{I}}{dV} & \frac{d\dot{I}}{dS} & \frac{d\dot{I}}{dR} \\ \frac{d\dot{A}}{dE} & \frac{d\dot{A}}{dI} & \frac{d\dot{A}}{dA} & \frac{d\dot{A}}{dV} & \frac{d\dot{A}}{dS} & \frac{d\dot{A}}{dR} \\ \frac{d\dot{V}}{dE} & \frac{d\dot{V}}{dI} & \frac{d\dot{V}}{dA} & \frac{d\dot{V}}{dV} & \frac{d\dot{V}}{dS} & \frac{d\dot{V}}{dR} \\ \frac{d\dot{S}}{dE} & \frac{d\dot{V}}{dI} & \frac{d\dot{V}}{dA} & \frac{d\dot{V}}{dV} & \frac{d\dot{V}}{dS} & \frac{d\dot{V}}{dR} \\ \frac{d\dot{R}}{dE} & \frac{d\dot{R}}{dI} & \frac{d\dot{R}}{dA} & \frac{d\dot{R}}{dV} & \frac{d\dot{R}}{dS} & \frac{d\dot{R}}{dR} \end{bmatrix}.$$

Now, the expression of differential coefficients can be found from the systems of Equations (1) – (6), which produces the following relation

$$J = \begin{bmatrix} -\alpha & \beta S & \beta S & \beta S & \beta(I+A+V) & 0 \\ p\sigma & -(\tau+\gamma+\mu) & 0 & 0 & 0 & 0 \\ (1-p)\sigma & 0 & -\eta & 0 & 0 & 0 \\ 0 & \tau & 0 & -\mu & 0 & 0 \\ 0 & -\beta S & -\beta S & -\beta S & -\beta(I+A+V) & \lambda \\ 0 & \gamma & \eta & 0 & 0 & -\lambda \end{bmatrix}. \quad (8)$$

At disease-free equilibrium, then the above Jacobian matrix can be written as

$$J = \begin{bmatrix} -\alpha & \beta N & \beta N & \beta N & 0 & 0 \\ p\sigma & -(\tau+\gamma+\mu) & 0 & 0 & 0 & 0 \\ (1-p)\sigma & 0 & -\eta & 0 & 0 & 0 \\ 0 & \tau & 0 & -\mu & 0 & 0 \\ 0 & -\beta N & -\beta N & -\beta N & 0 & \lambda \\ 0 & \gamma & \eta & 0 & 0 & -\lambda \end{bmatrix}.$$

We can write the above matrix shortly as

$$J = \begin{bmatrix} F - V_1 & 0 \\ U_1 & U_2 \end{bmatrix},$$

where

$$F - V_1 = \begin{bmatrix} -\sigma & \beta N & \beta N & \beta N \\ p\sigma & -\tau - \gamma - \mu & 0 & 0 \\ (1-p)\sigma & 0 & -\eta & 0 \\ 0 & \tau & 0 & -\mu \end{bmatrix},$$

$$U_1 = \begin{bmatrix} 0 & -\beta N & -\beta N & -\beta N \\ 0 & \gamma & \eta & 0 \end{bmatrix},$$

and

$$U_2 = \begin{bmatrix} 0 & \lambda \\ 0 & -\lambda \end{bmatrix}.$$

The disease-free equilibrium is locally asymptotically stable, if the eigenvalues of Jacobian matrix at disease free equilibrium have negative real part (Driessche et al. (2002)). Since the eigenvalues of J are those of $(F - V_1)$ and U_2 which are negative. Thus, the stability of J is depended on the eigenvalues of $(F - V_1)$. That is, disease free equilibrium is stable if all the eigenvalues of $(F - V_1)$ have negative real part. Here, F is non-negative and V_1 is non-singular.

The characteristic Equation of the Jacobian matrix is

$$|\lambda I - J| = 0,$$

which can be written into a biquadratic Equation in the form

$$a_4 S^4 + a_3 S^3 + a_2 S^2 + a_1 S + a_0 = 0, \tag{9}$$

where

$$a_4 = 1,$$

$$a_3 = 2\mu + \eta + \tau + \gamma + \sigma,$$

$$a_2 = (2\mu - (1-p)\beta N + \eta + \tau + \gamma)\sigma + \mu^2 + (2\eta + \tau + \gamma)\mu - p\sigma\beta N + \eta(\tau + \gamma),$$

$$a_1 = (\mu^2 + ((-2+2p)\beta N + 2\eta + \tau + \gamma)\mu + ((-1+p)\beta N + \eta)(\tau + \gamma))\sigma + \eta\mu^2$$

$$+\big(-p\sigma\beta N + \eta(\tau + \gamma)\big)\mu - p\sigma\beta N(\tau + \eta),$$

and

$$a_0 = (\tau + \gamma + \mu)((p-1)\beta N + \eta)\mu\sigma - \eta p\sigma\beta N(\mu + \tau).$$

According to Routh-Hurwitz condition, the system will be stabile if

$$a_0 > 0, a_1 > 0, a_2 > 0, a_3 > 0, a_3 a_2 > a_4 a_1 \text{ and } a_3 a_2 a_1 > a_4 {a_1}^2 + a_0 {a_3}^2.$$

.

By simple algebraically process with comparing terms and the conditions, we have

$$\Re_0 = \left[\frac{1-p}{\eta} + \frac{p}{\gamma+\tau+\mu} + \frac{p\tau}{\mu(\gamma+\tau+\mu)}\right] < 1.$$

Consequently, we can prove the system will be unstable if $\Re_0 > 1$. This is the complete proof.

Theorem 2.2.3.

The disease-free equilibrium of the model is globally asymptotically stable if $\Re_0 < 1$, and unstable $\Re_0 > 1$.

***Proof*:**

We can define Lyapunov function in the linear form as

$$V_2 = k_1\left(S - S_0 - S_0 \ln\frac{S}{S_0}\right) + k_2E + k_3I + k_4A + k_5V + k_6R, \tag{10}$$

where, k_1, k_2, k_3, k_4, k_5 and k_6 are constants and the derivative of Lyapunov function can be found using the value of $\dot{S}$, $\dot{E}$, $\dot{I}$, $\dot{A}$, $\dot{V}$, $\dot{R}$ from Equations (1) - (6) and $S_0 = N$ as

$$\dot{V}_2 = k_1(1-\frac{N}{S})(\lambda R - \beta S(I+A+V)) + k_2(\beta S(I+A+V) - \sigma E)$$

$$+k_3(p\sigma E - (\tau+\gamma+\mu)I) + k_4\big((1-p)\sigma E - \eta A\big) + k_5(\tau I - \mu V)$$

$$+(k_6+k_1)(\gamma I + \eta A - \lambda R). \tag{11}$$

Applies perturbation method in Equation (11) and which provides

$$k_1 = k_2 = p,$$

$$k_3 = 1 + \frac{k_4(p-1)}{p},$$

$$k_4 = \frac{p\beta N}{\eta} + \frac{(1-p)\beta N(\tau+\gamma+\mu)}{\eta\gamma},$$

$$k_5 = \frac{\beta N p}{\mu},$$

and

$$k_6 + p = \frac{(1-p)\beta N(\tau+\gamma+\mu)}{\eta\gamma}.$$

Now using the values of k_1, k_2 , k_3, k_4, k_5, and k_6 into Equation (11) and after simplifying, we get

$$\dot{V}_2 = -pN\tau\frac{\Re_0}{S} + (\tau+\gamma+\mu)\left[\beta N\left\{\frac{p}{(\tau+\gamma+\mu)} + \frac{p\tau}{\mu(\tau+\gamma+\mu)} + \frac{1-p}{\eta}\right\} - 1\right]I$$

$$\Rightarrow \dot{V}_2 = -pN\tau\frac{\Re_0}{S} + (\tau+\gamma+\mu)[\Re_0 - 1]I.$$

The disease-free equilibrium is globally asymptotically stable if $\dot{V}_2 \le 0$, which becomes $\Re_0 < 1$.

2.3. Endemic equilibrium

For endemic equilibrium, an equilibrium solution of the systems of Equations (1) – (6) can be found

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dA}{dt} = \frac{dV}{dt} = \frac{dR}{dt} = 0,$$

with the help of $E \neq I \neq 0$. After solving the above system in the case of endemic equilibrium, we have the following relations

$$S_e = \frac{N}{\Re_0},\ E_e = \frac{(\tau+\gamma+\mu)}{p\sigma}I_e,\ I_e = \frac{Np}{\left(\frac{1}{\sigma}+\frac{R_0}{\beta N}\right)(\tau+\gamma+\mu)}(1-\frac{1}{\Re_0}),$$

$$A_e = \frac{(1-p)(\tau+\gamma+\mu)}{\eta}I_e,\ V_e = \frac{\tau}{\mu}I_e,\ R_e = \frac{(\tau+\gamma+\mu)}{p\tau}I_e.$$

Thus, endemic equilibrium is $(S_e, E_e, I_e, A_e, V_e, R_e)$.

Theorem 2.3.1.

The endemic equilibrium of the model is locally asymptotically stable if $\Re_0 > 1$, and unstable if $\Re_0 < 1$.

Proof:

The Jacobian matrix J which is derived in Equation (8) can be written also for endemic equilibrium $(S_e, E_e, I_e, A_e, V_e, R_e)$ as

$$J=\begin{bmatrix} -\alpha & \beta S & \beta S & \beta S & \beta(I+A+V) & 0 \\ p\sigma & -(\tau+\gamma+\mu) & 0 & 0 & 0 & 0 \\ (1-p)\sigma & 0 & -\eta & 0 & 0 & 0 \\ 0 & \tau & 0 & -\mu & 0 & 0 \\ 0 & -\beta S & -\beta S & -\beta S & -\beta(I+A+V) & \lambda \\ 0 & \gamma & \eta & 0 & 0 & -\lambda \end{bmatrix}.$$

At disease-free equilibrium $\left(S_e, E_e, I_e, A_e, V_e, R_e\right)$, the above matrix becomes

$$J=\begin{bmatrix} -\sigma & \beta S_e & \beta S_e & \beta S_e & \beta(I_e+A_e+V_e) & 0 \\ p\sigma & -(\tau+\gamma+\mu) & 0 & 0 & 0 & 0 \\ (1-p)\sigma & 0 & -\eta & 0 & 0 & 0 \\ 0 & \tau & 0 & -\mu & 0 & 0 \\ 0 & -\beta S_e & -\beta S_e & -\beta S_e & -\beta(I_e+A_e+V_e) & \lambda \\ 0 & \gamma & \eta & 0 & 0 & -\lambda \end{bmatrix}.$$

For convenient, this can be written as $J=\begin{bmatrix} P_1 & P_2 \\ P_3 & P_4 \end{bmatrix}$,

where

$$P_1=\begin{bmatrix} -\sigma & \beta S_e & \beta S_e & \beta S_e \\ p\sigma & -\tau-\gamma-\mu & 0 & 0 \\ (1-p)\sigma & 0 & -\eta & 0 \\ 0 & \tau & 0 & -\mu \end{bmatrix}, P_2=\begin{bmatrix} \beta(I_e+A_e+V_e) & 0 \\ 0 & 0 \\ 0 & 0 \\ 0 & 0 \end{bmatrix},$$

$$P_3=\begin{bmatrix} 0 & -\beta S_e & -\beta S_e & -\beta S_e \\ 0 & \gamma & \eta & 0 \end{bmatrix}, \qquad P_4=\begin{bmatrix} -\beta(I_e+A_e+V_e) & \lambda \\ 0 & -\lambda \end{bmatrix}.$$

The endemic equilibrium is locally asymptotically stable, if the eigenvalues P_1 and P_2 of Jacobian matrix J at endemic equilibrium have negative real part (Driessche et al. (2002)). Moreover, the eigenvalues of P_4 will be negative if $\beta\left(I_e+A_e+V_e\right)$ is positive. That is,

$$\beta\left(1+\frac{\tau+\gamma+\mu}{p}+\frac{\tau}{\mu}\right)I_e>0.$$

Applying the value of I_e, the above equation becomes

$$\beta\left(1+\frac{\tau+\gamma+\mu}{p}+\frac{\tau}{\mu}\right)\frac{Np}{\left(\frac{1}{\sigma}+\frac{R_0}{\beta N}\right)(\tau+\gamma+\mu)}(1-\frac{1}{\Re_0})>0.$$

Furthermore, we have

$$(1-\frac{1}{\Re_0})>0\text{, Since }\beta\left(1+\frac{\tau+\gamma+\mu}{p}+\frac{\tau}{\mu}\right)\frac{Np}{\left(\frac{1}{\sigma}+\frac{R_0}{\beta N}\right)(\tau+\gamma+\mu)}>0.$$

Thus, $\Re_0>1$.

Theorem 2. 3. 2.

If $\Re_0>1$, then prove that the endemic equilibrium $(S_e,E_e,I_e,A_e,V_e,R_e)$ is globally asymptotically stable.

***Proof*:**

Consider the Lyapunov function in the following form

$$V_3=k_1(S-S_e-S_e\,ln\frac{S}{S_e})+k_2(E-E_e-E_e\,ln\frac{E}{E_e})+k_3(I-I_e-I_e\,ln\frac{I}{I_e})$$
$$+k_4(A-A_e-A_e\,ln\frac{A}{A_e})+k_5(V-V_e-V_e\,ln\frac{V}{V_e})+k_6(R-R_e-R_e\,ln\frac{R}{R_e}). \quad (12)$$

After differentiating, the Lyapunov function can be found as

$$\dot{V}_3=k_1\left(1-\frac{S_e}{S}\right)\dot{S}+k_2\left(1-\frac{E_e}{E}\right)\dot{E}+k_3\left(1-\frac{I_e}{I}\right)\dot{I}+k_4\left(1-\frac{A_e}{A}\right)\dot{A}+k_5\left(1-\frac{V_e}{V}\right)\dot{V}$$
$$+k_6(1-\frac{R_e}{R})\dot{R}$$

Substituting the expressions of $\dot{S},\dot{E},\dot{I},\dot{A},\dot{V}$ and $\dot{R}$ in the above Equation, we get

$$\dot{V}_3=k_1\left(1-\frac{S_e}{S}\right)\left(\lambda R-\beta S(I+A+V)\right)+k_2\left(1-\frac{E_e}{E}\right)(\beta S(I+A+V)-\sigma E)$$
$$k_3\left(1-\frac{I_e}{I}\right)(p\sigma E-(\tau+\gamma+\mu)I)+k_4\left(1-\frac{A_e}{A}\right)\left((1-p)\sigma E-\eta A\right)$$
$$+k_5\left(1-\frac{V_e}{V}\right)(\tau I-\mu V)+k_6\left(1-\frac{R_e}{R}\right)(\gamma I+\eta A-\lambda R) \quad (13)$$

After simplifying, Equation (13) can be written as

$$\dot{V}_3=(k_1\lambda R-k_1\beta S(I+A+V)-k_1\lambda\frac{S_eR}{S}+k_1\beta S_e(I+A+V))$$
$$+(k_2\beta S(I+A+V)-k_2\sigma E-k_2\beta\frac{SE_e}{E}(I+A+V)+k_2\sigma E_e)$$
$$+(k_3p\sigma E-k_3(\tau+\gamma+\mu)I-k_3p\sigma\frac{EI_e}{I}+k_3(\tau+\gamma+\mu)I_e)$$

$$+(k_4(1-p)\sigma E - k_4\eta A - k_4(1-p)\sigma\frac{EA_e}{A} + k_4\eta A_e)$$
$$+(k_5\tau I - k_5\mu V - k_5\tau\frac{IV_e}{V} + k_5\mu V_e)$$
$$+(k_6\gamma I + k_6\eta A - k_6\lambda R - k_6\gamma\frac{IR_e}{R} - k_6\eta\frac{AR_e}{R} + k_6\lambda R_e) \quad (14)$$

To eliminate the linear terms of E, I, A, V and R from Equation (14), we have used

$$-k_2\sigma + k_3\sigma p + k_4(1-p)\sigma = 0, \quad (15)$$

$$k_1\beta S_e - k_3(\tau+\gamma+\mu) + k_5\tau + k_6\gamma = 0, \quad (16)$$

$$k_1\beta S_e - k_4\eta + k_6\eta = 0, \quad (17)$$

$$k_1\beta S_e - k_5\mu = 0, \quad (18)$$

$$k_1\lambda - k_6\lambda = 0. \quad (19)$$

From Equations (15) to (19), we have

$$k_1 = k_2 = 1, k_3(\tau+\gamma+\mu) = \beta S_e + \beta S_e\frac{\tau}{\mu} + \gamma, k_4 = \frac{\beta S_e}{\eta} + 1,$$

$$k_5 = \frac{\beta S_e}{\mu}, \text{ and } \quad k_6 = 1. \quad (20)$$

Since $(S_e, E_e, I_e, A_e, V_e, R_e)$ is the solution set of a compartmental model, so we have the following:

$$\lambda R_e - \beta S_e(I_e + A_e + V_e) = 0, \quad (21)$$

$$\beta S_e(I_e + A_e + V_e) - \sigma E = 0, \quad (22)$$

$$p\sigma E_e - (\tau+\gamma+\mu)I_e = 0, \quad (23)$$

$$(1-p)\sigma E_e - \eta A_e = 0, \quad (24)$$

$$\tau I_e - \mu V_e = 0\,, \quad (25)$$

$$\gamma I_e + \eta A_e - \lambda R_e = 0. \quad (26)$$

From Equations (21) – (26), we can derive the following relations:

$$\frac{I_e E}{I}k_3\sigma p = \left(\beta S_e + \beta S_e\frac{V_e}{I_e} + \gamma\right)\frac{I_e^2 E}{E_e I}, \quad (27)$$

$$\frac{EA_e}{A}k_4(1-p)\sigma = (\beta S_e + \eta)\frac{EA_e^2}{E_eA}, \tag{28}$$

$$\sigma E = \left(\beta S_e + \beta S_e\frac{V_e}{I_e} + \gamma\right)\frac{I_eE}{E_e} + (\beta S_e + \eta)\frac{A_eE}{E_e}, \tag{29}$$

Now substituting the expressions of k_1, k_2 , k_3, k_4, k_5, and k_6 from Equation (20) into Equation (14) and using Equations (27) – (29), we get

$$\begin{aligned}\dot{V}_3 = &-\lambda\frac{S_eR}{S} - \beta\frac{E_e}{E}S(I+A+V) + \sigma E_e - \left(\beta S_e + \beta S_e\frac{V_e}{I_e} + \gamma\right)\frac{I_e^2E}{IE_e}\\ &+\left(\beta S_e + \beta S_e\frac{V_e}{I_e} + \gamma\right)I_e - (\beta S_e + \eta)\frac{EA_e^2}{E_eA} - (\beta S_e + \eta)A_e\\ &-\beta S_e\frac{V_e^2I}{I_eV} + \beta S_eV_e - \gamma\frac{R_eI}{R} - \eta\frac{R_eA}{R} + \lambda R_e\end{aligned} \tag{30}$$

Applying Equation (26) into Equation (30), we have

$$\begin{aligned}\dot{V}_3 = &-\lambda\frac{S_eR}{S} - \beta\frac{E_e}{E}S(I+A+V) - \left(\beta S_e + \beta S_e\frac{V_e}{I_e}\right)\frac{I_e^2E}{IE_e} + \left(\beta S_e + \beta S_e\frac{V_e}{I_e}\right)I_e - \gamma\frac{I_e^2E}{IE_e}\\ &-\beta S_e\frac{EA_e^2}{E_eA} - \eta\frac{EA_e^2}{E_eA} + \beta S_eA_e - \beta S_e\frac{V_e^2I}{I_eV} + \beta S_eV_e - \gamma\frac{R_eI}{R} - \eta\frac{R_eA}{R} + 3\lambda R_e\end{aligned} \tag{31}$$

Implies that

$$\begin{aligned}\dot{V}_3 = &\beta S_eI_e\left(3 - \frac{E_eSI}{ES_eI_e} - \frac{I_eE}{IE_e} - \frac{S_e}{S}\right) + \beta S_eA_e\left(3 - \frac{E_eSA}{ES_eA_e} - \frac{A_eE}{AE_e} - \frac{S_e}{S}\right)\\ &+\beta S_eV_e\left(4 - \frac{IV_e}{I_eV} - \frac{EI_e}{E_eI} - \frac{E_eSV}{ES_eV_e} - \frac{S_e}{S}\right) - \lambda\frac{S_eR}{S} + \beta S_eI_e\left(1 + \frac{S_e}{S}\right)\\ &+\beta S_eA_e\left(1 + \frac{S_e}{S}\right) + \beta S_eV_e\left(1 + \frac{S_e}{S}\right) - \gamma\frac{I_e^2E}{IE_e} - \eta\frac{A_e^2E}{AE_e} - (\gamma I + \eta A)\frac{R_e}{R}.\end{aligned}$$

After rearranging, we have

$$\begin{aligned}\dot{V}_3 = &\beta S_eI_e\left(3 - \frac{E_eSI}{ES_eI_e} - \frac{I_eE}{IE_e} - \frac{S_e}{S}\right) + \beta S_eA_e(3 - \frac{E_eSA}{ES_eA_e} - \frac{A_eE}{AE_e} - \frac{S_e}{S})\\ &+\beta S_eV_e(4 - \frac{IV_e}{I_eV} - \frac{EI_e}{E_eI} - \frac{E_eSV}{ES_eV_e} - \frac{S_e}{S})\\ &+\beta S_e(I_e + A_e + V_e)[1 + \frac{S_e}{S} - \frac{S_eR}{SR_e} - p\frac{I_eE}{IE_e} - (1-p)\frac{A_eE}{AE_e} - p\frac{R_eI}{RI_e} - (1-p)\frac{R_eA}{RA_e}]\end{aligned} \tag{32}$$

Since the arithmetic mean exceeds the geometric mean, so we can express the following inequalities:

$$3 - \frac{E_e SI}{ES_e I_e} - \frac{I_e E}{IE_e} - \frac{S_e}{S} \leq 0,$$

$$3 - \frac{E_e SA}{ES_e A_e} - \frac{A_e E}{AE_e} - \frac{S_e}{S} \leq 0,$$

$$4 - \frac{IV_e}{I_e V} - \frac{EI_e}{E_e I} - \frac{E_e SV}{ES_e V_e} - \frac{S_e}{S} \leq 0.$$

Note that

$$1 + \frac{S_e}{S} - \frac{S_e R}{SR_e} - p\frac{I_e E}{IE_e} - (1-p)\frac{A_e E}{AE_e} - p\frac{R_e I}{RI_e} - (1-p)\frac{R_e A}{RA_e} \leq 0$$

and from Equation (32), it follows that $\dot{V}_3 \leq 0$. Therefore, by the Lyapunov function and the LaSalle's principle, every solution of the proposed model approaches to the endemic equilibrium as $t \to \infty$ for $\Re_0 > 1$ and

$$1 + \frac{S_e}{S} - \frac{S_e R}{SR_e} - p\frac{I_e E}{IE_e} - (1-p)\frac{A_e E}{AE_e} - p\frac{R_e I}{RI_e} - (1-p)\frac{R_e A}{RA_e} \leq 0.$$

.

2.4. Final size relation

Final size relation is the relation between the basic reproduction number and the number of populations in the epidemic which remain at each disease-free compartment. Arino et al. (2007) discussed final size relation of the epidemic model and found the relation as

$$\ln\frac{S_0}{S_\infty} = \frac{\Re_0}{S_0}[S_0 - S_\infty] + \beta b {V_1}^{-1} x(0),$$

where $b = [0\ 1\ 1\ 1]$ and $x(0) = [0\ I_0\ 0\ 0]'$from initial conditions $E(0) = 0, I(0) = I_0$, $A(0) = 0$ and $V(0) = 0$.

The final size relation of the model becomes

$$\ln\frac{S_0}{S_\infty} = \frac{\Re_0}{S_0}[S_0 - S_\infty] + \beta\frac{I_0(\mu+\tau)}{\mu(\gamma+\tau+\mu)}. \quad (33)$$

Equation (33) shows that $S_\infty > 0$, since right side of Equation (33) is finite. That is, final size number population in the epidemic indicates that some susceptive will be uninfected.

3. Numerical Computations and Results

3. 1 Numerical method

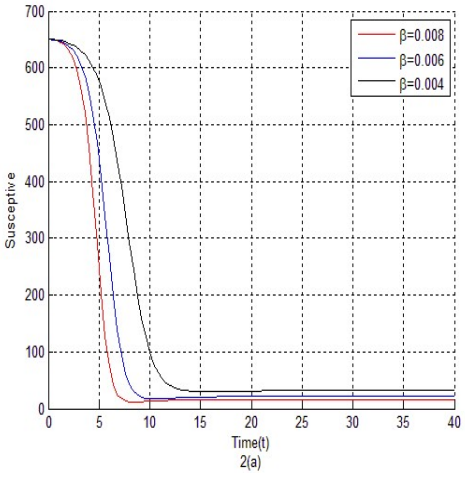
The systems of Equations (1) – (6) have been used as the governing Equations with the initial condition in Equation (7). These Equations are solved numerically with the aid of Range-Kutta method. We have solved the Equations numerically using various choices of parameters (Table 1) and time intervals and have found the solution for each compartment.

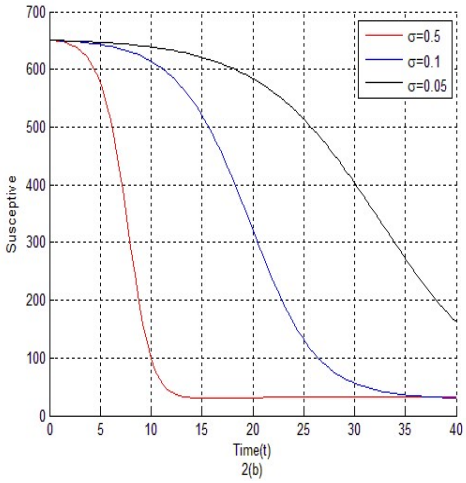
Table 1: Various choices of parameters with references that are used in the model

Name of the parameter	Value	Reference
Infection rate (β)	0.004-0.008	Biswas et al.(2008)
Expose rate (σ)	0.5-0.05	Rahman et al.(2012)
Asymptomatic rate ($1-p$)	5.2% - 35.5%	Kanamori et al.(2016)
Mortality rate (μ)	90%-100%	Payungporn et al.(2006)
Shaded virus rate (τ)	0.75-1.80	Lin et al.(2016)
Removal rate (γ) from infected	10%-0%	Payungporn et al.(2006)
Removal to Susceptive rate (λ)	90%-100%	---
Removal rate (η)from asymptomatic	50%-80%	Kanamori et.al. (2016)
Initials susceptive (S_0)	650	Alam et. al. (2010)
Initials infective (I_0)	6	Alam et. al. (2010)

3. 2. Computed results of successive, expose, infective and removal

Some typical results of susceptive, expose, infective, removal and asymptotic are estimated which are presented in Figures (2) – (7). In Figure 2, the variation of susceptive decreases rapidly at the initial stages and after that it has no variations with the increasing of time. Moreover, it can be seen that the susceptive rate is very sensitive to the expose rate (Figure 2(b)). Furthermore, the rest of the parameters have found the similar effects on successive compartment.





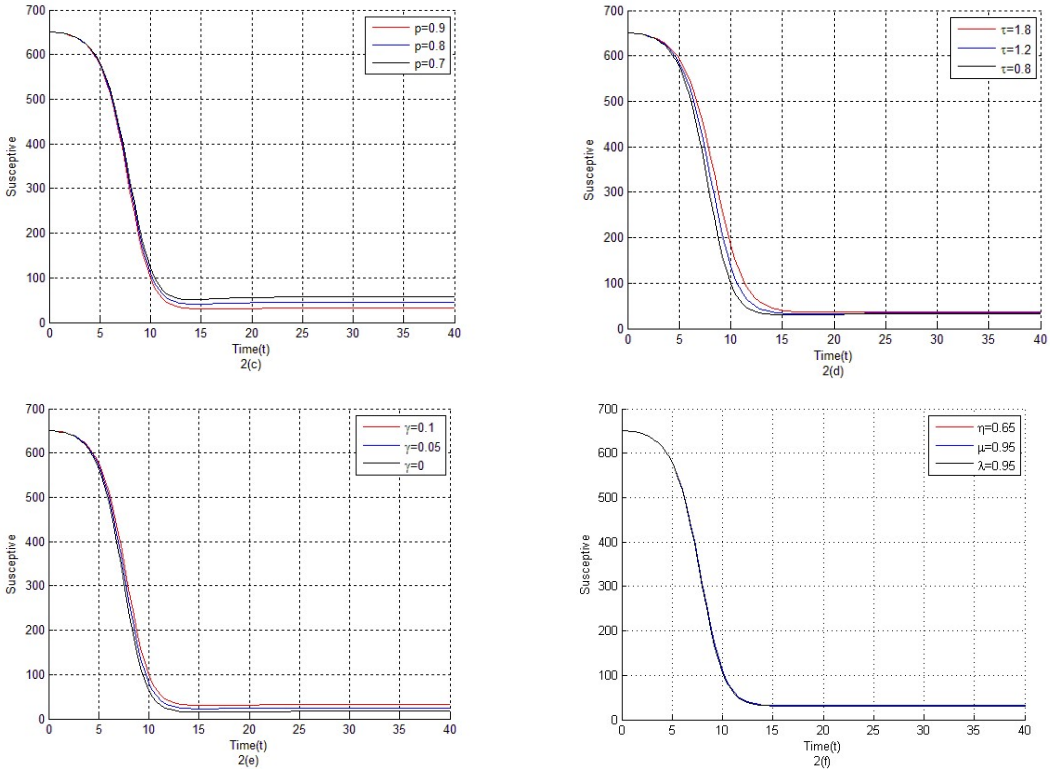
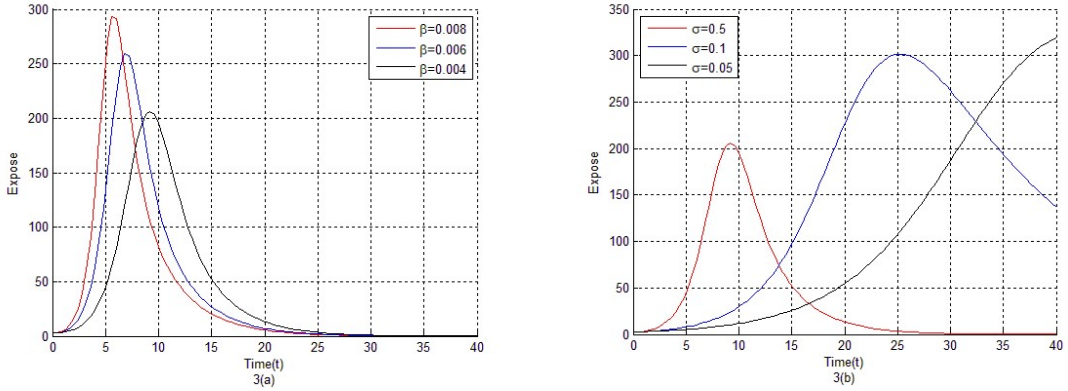


Figure 2. Profiles of the susceptive individuals for several parameters

In Figure 3, it is seen that the expose increases from initial position to a certain point and then decreases exponentially to dismiss. But in Figure 3(b), the expose shows the divergent nature as the decreasing of the value σ. Expose compartment is affected by the infection rate, symptomatic rate, expose period and shad virus and other parameters (mortality rate, removal rates) have the less effect those are shown in Figures 3(e) - 3(f).



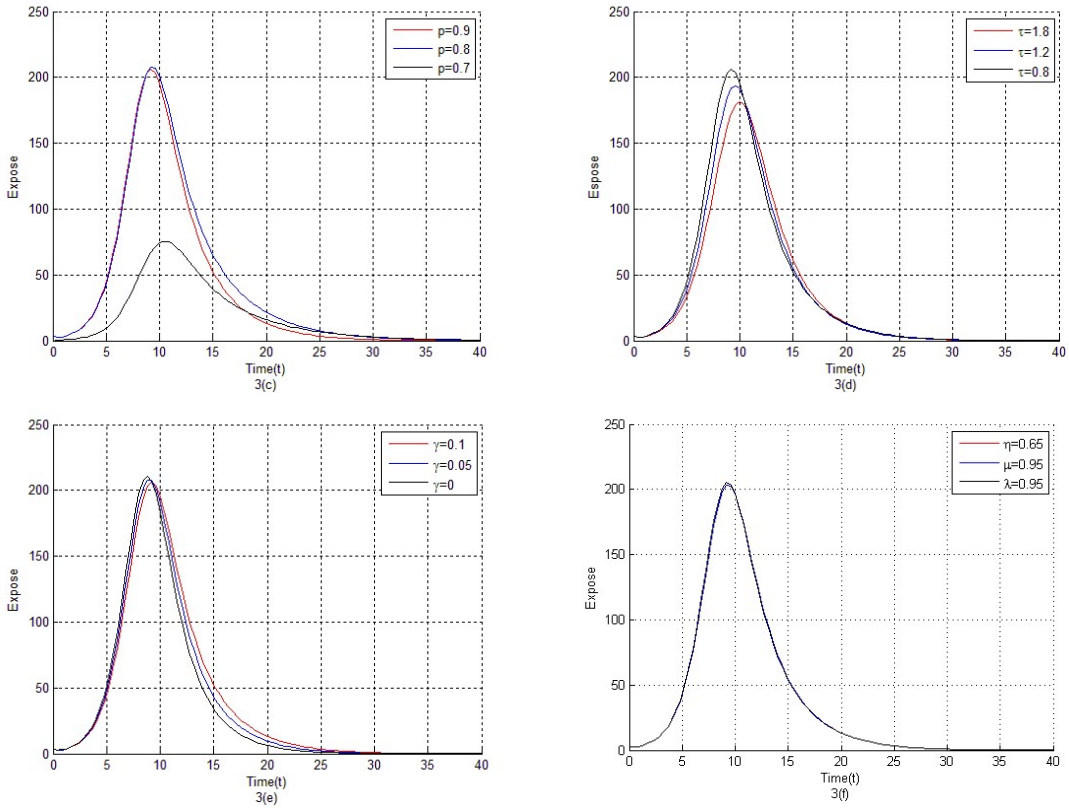
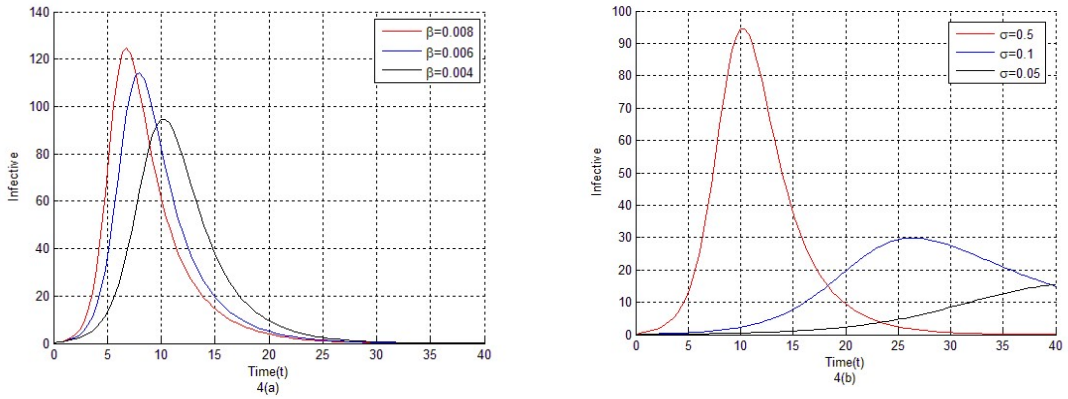


Figure 3. Variations of the expose individuals for several parameters

Furthermore, infective compartment (symptomatic compartment) is significantly affected by the infection rate, expose period, asymptomatic fraction, shaded virus rate and removal rate (Figures 4(a-e)) and other rate have less effect which is seen Figure (4f). Infective compartment is increased with the increasing of infection rate, expose rate, and symptomatic fraction, but decreases with the increasing of shades virus rate and removal rate, Figure 4.



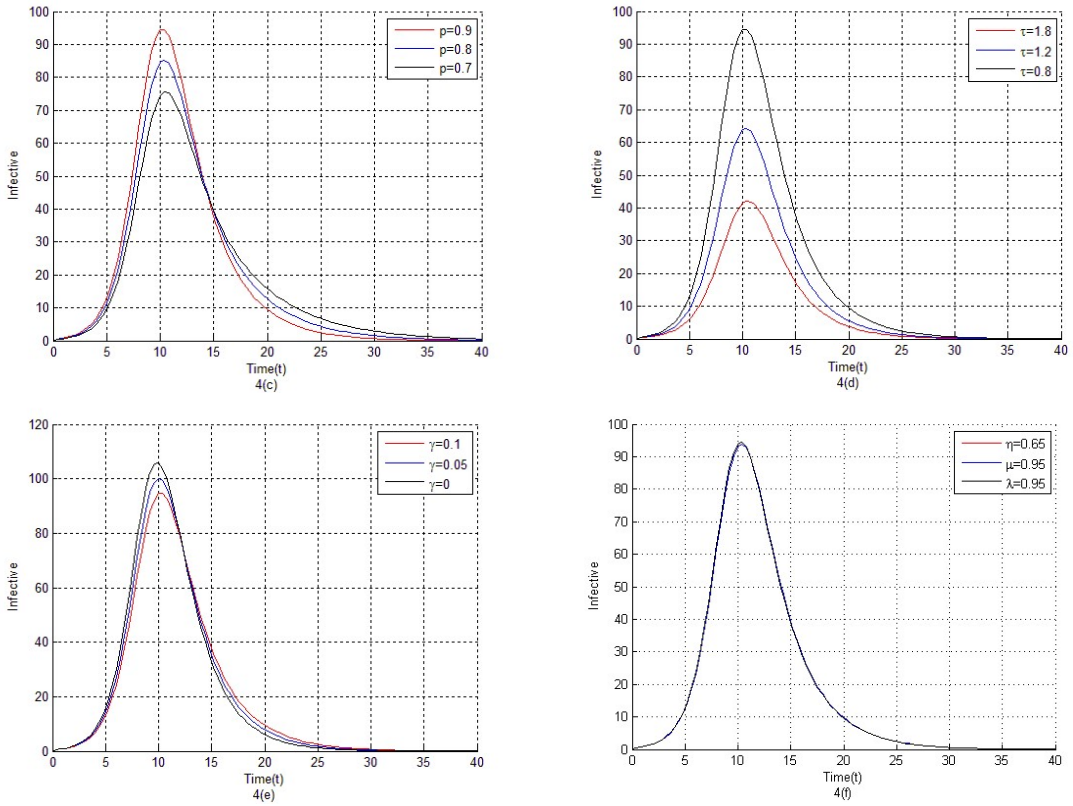
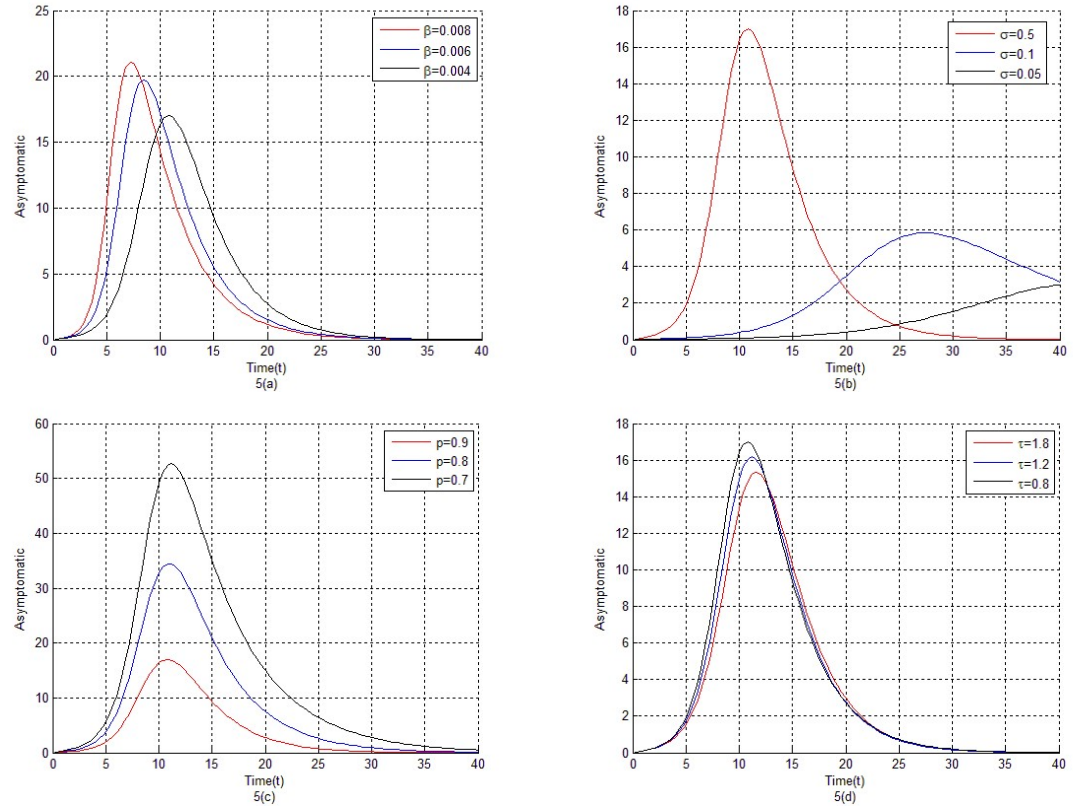


Figure 4. Variations of infective individuals with the variation of time for several parameters



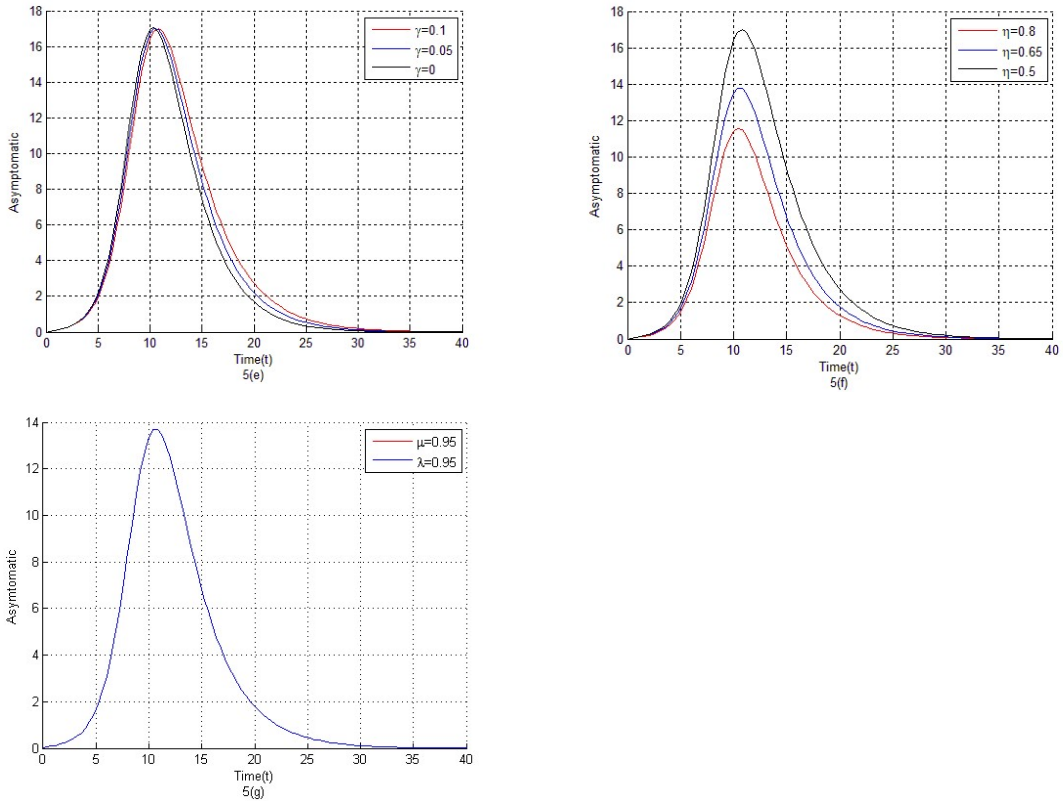
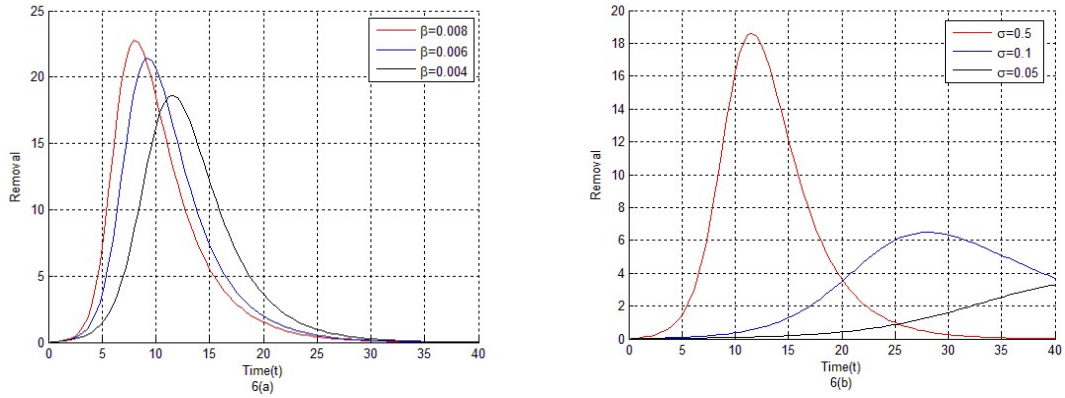


Figure 5. Variations of the asymptomatic individuals for several parameters

In Figures (5) and (6), we have presented the results for asymptomatic and removal individuals, respectively. Although the trends of the asymptomatic and removal curves are similar to the expose and infective curves in Figures 3 and 4, but removal curves in Figure 6 are significantly varies with γ. These types works were done by (Lin et al. 2016, Modnak el al. 2017, Putri et al. 2016). They derived the infected, exposed, asymptomatic, removal and found similar results.



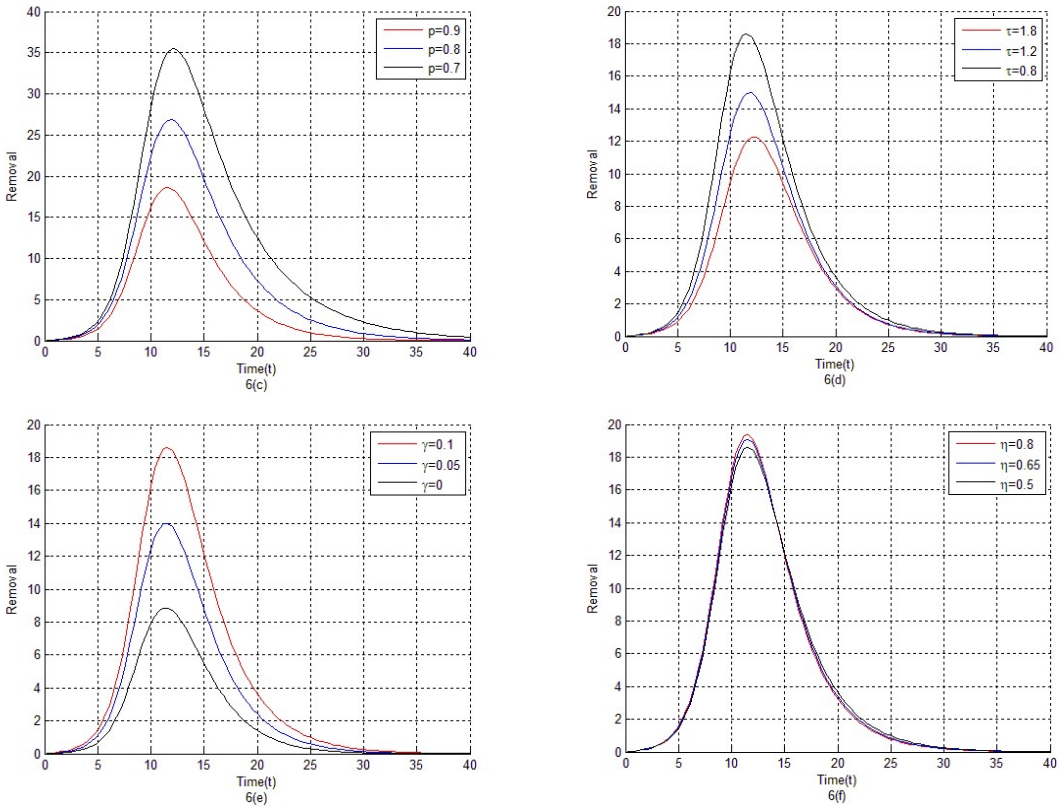


Figure 6. Profiles of removal compartment with the variation of time for several parameters

3.3. Comparison between computed results and data

The computed results of infected, exposed, asymptomatic, removal, shaded virus are plotted at the same time in Figure 7. It is observed that all the individual increases first and then decrease gradually to dismiss. Furthermore, it is also seen that the infection rate and expose period are very effective compared to other parameters.

Finally, the SEIAVR compartmental model's result, Lin et al. (2016) result and field data are compared in Figure 8. The result of our six-compartmental model shows good performance against the field data compared to Lin et al. (2016) result. The data were conducted by Alam et al. (2010) for avian influenza.

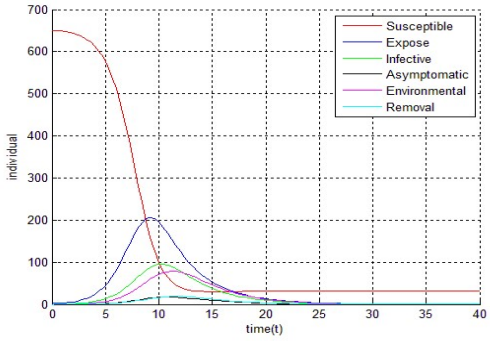


Figure 7. The graphical representation of individual model results

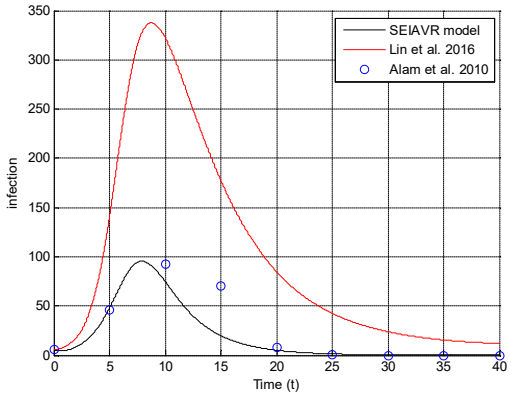


Figure 8. Comparison between the model results of infection and data

4. Conclusion

In this study, we presented a SEIAVR compartmental model based on avian influenza and discussed its local and global stability at the two equilibrium conditions. We also derived the basic reproduction number, final size relation and a relationship between these two phenomena. Final size relationship was pointed out that some susceptive population remained uninfected during epidemiology. The developed Equations were solved numerically with the help of Range-Kutta method and the values of initial parameters were taken from the several literatures and reports. The calculated results of susceptive expose, infective, removal, virus and asymptotic compartments were presented individually where all the parameters varied significantly for β, σ, p and τ. Furthermore, the above parameters also were compared at the same time and the infection rate and expose period observed very sensitive compared to other parameters. In addition, the model result of infection rate was compared with the field data and found satisfactory result.

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