South East Asian J. of Mathematics and Mathematical Sciences Vol. 17, No. 2 (2021), pp. 385-398

ISSN (Online): 2582-0850

ISSN (Print): 0972-7752

A MATHEMATICAL MODEL OF TRANSMISSION DYNAMICS OF HEPATITIS B VIRUS

R. S. Jain, B. G. Urekar and B. S. Reddy

School of Mathematical Sciences, Swami Ramanand Teerth Marathwada University, Nanded - 431606, Maharashtra, INDIA

E-mail : rupalisjain@gmail.com

(Received: Sep. 22, 2020 Accepted: Aug. 10, 2021 Published: Aug. 30, 2021)

Abstract: In this paper, we have formulated a new mathematical model for transmission dynamics of Hepatitis B virus by using system of differential equations. Also we have obtained the conditions under which the model will be in disease free equilibrium state as well as we have found its basic reproduction number.

Keywords and Phrases: Hepatitis B virus (HBV), mathematical model, disease-free equilibrium, stability analysis, basic reproduction number.

2020 Mathematics Subject Classification: 92D30, 34C60.

1. Introduction

Hepatitis B is an infectious disease caused by the Hepatitis B virus (HBV) that affects the liver. It can cause both mild to severe infections which can result in death. According to WHO, 257 million people were living with severe Hepatitis B infection. Hepatitis B can be prevented by vaccines that are safe, available and effective and 80 - 90% of infants infected during the first year of life develop chronic infections and 20 - 30% of adults who are chronically infected will develop cirrhosis and liver cancer. There is still limited access to diagnosis and treatment of Hepatitis B in many resource contained settings. Since the observance of World Hepatitis Day 2019, WHO has been focusing on the eradication of Hepatitis B by the year 2030. For this, control strategies should be decided and the most effective control measure is vaccination. One of the primary reasons for studying Hepatitis B virus (HBV) infection is to improve control and finally to bring down the infection from the population [1]. Mathematical modelling can be a useful tool in this approach which helps us to optimize the use of finite resources and to take control measures more impressively. Many authors have studied transmission dynamics of Hepatitis B virus and other infectious diseases by proposing different mathematical models for example, see [2, 5, 6, 13, 14], [11], [3], [7, 8, 12, 15]. Recently, Blessing et. al. [4] proposed the mathematical model which is based on standard SEIR model.

Many mathematical models containing Hepatitis B vaccine usually considered that the vaccine is fully effective to avoid the infection of vaccinated individuals. In fact, the Hepatitis B vaccine should be taken in three doses at 0, 1 and 6 months. Generally 30-50% of individuals will gain anti-HBs antibody after the first dose, 80-90% will gain after the second dose and almost all the individuals will have high anti-HBs concentrations one month after the last dose that 99.80% of vaccines improve anti-HBs antibody. As soon as we give vaccination to susceptible individuals, they will be removed from susceptible population. But they should also differ from recovered individual s which have immunity beside the disease. It means that some of vaccinated individuals may still be susceptible to infection, but they will be infected at a lower rate than susceptible individuals whoever not vaccinated.

So, we extend the model proposed in [4], by adding three new compartments as V_1 , V_2 and V_3 of vaccinated individuals. We propose new mathematical model and study the transmission dynamics of Hepatitis B virus under the administration of vaccination. Further we establish necessary and sufficient conditions for the model to be stable. Also we obtain basic reproduction number for the newly proposed model.

The paper is organized as follows: Section 2 presents used assumptions, parameters and existing model. In section 3, we propose new mathematical model. In section 4, we discuss the equilibrium points, stability analysis and basic reproduction number and section 5, gives conclusion of the study.

2. Assumptions, parameters and existing model of HBV

We shall use the following assumptions and parameters :

2.1. Assumptions of the Model

- The population is homogeneous.
- The treated individuals recover.
- Influx in the population is by birth only.

- Exit out of the population is by natural and HBV related mortality only.
- The vaccinated individuals do not acquire permanent immunity.

2.2. Variables and Parameters of the Model

- S(t) = proportion of the susceptible individuals at time t,
- L(t) = proportion of the latently infected/exposed individuals at time t,
- I(t) = proportion of the infectious individuals at time t,
- R(t) = proportion of the recovered/removed individuals at time t.

 $V_1(t)$ =proportion of vaccinated individuals who gain anti-HBs antibody after the first dose,

 $V_2(t)$ =proportion of vaccinated individuals who gain anti-HBs antibody after the second dose,

 $V_3(t)$ =proportion of vaccinated individuals who have high anti-HBs antibody after the last dose (third dose).

 ϕ_1 = rate of expiration of vaccine efficacy after the first dose

 ϕ_2 = rate of expiration of vaccine efficacy after the second dose

 ϕ_3 = rate of expiration of vaccine efficacy after the third dose

 ϕ_4 = rate of transfer from class V_1 to V_2

 ϕ_5 = rate of transfer from class V_2 to V_3

 $\mu = {\rm rate}$ of conversion from S to L

- $\mathbf{k} = \mathrm{rate} \ \mathrm{of} \ \mathrm{conversion} \ \mathrm{from} \ \mathrm{L} \ \mathrm{to} \ \mathrm{I}$
- ψ = rate of conversion from I to R
- π = rate of conversion from R to S
- $\beta = \text{HB-induced mortality}$
- η = natural mortality
- P = population of new births
- cP = immunized new births
- N = total population size

2.3. The Existing Model

We begin our model formulation by introducing the existing model given in [4].





Figure 1: Flow diagram of HBV transmission dynamics for the existing model

2.4. The Equations of the Existing Model

The model equations are as follows:

$$\frac{dM(t)}{dt} = cP - \phi M(t) - \eta M(t) \tag{1}$$

$$\frac{dS(t)}{dt} = (1-c)P + \phi M(t) + \pi R(t) - (\mu + \eta)S(t)$$
(2)

$$\frac{dL(t)}{dt} = \mu S(t) - qL(t) - kL(t) - \eta L(t)$$
(3)

$$\frac{dI(t)}{dt} = kL(t) - \psi I(t) - \eta I(t) - \beta I(t)$$
(4)

$$\frac{dR(t)}{dt} = qL(t) + \psi I(t) - \pi R(t) - \eta R(t)$$
(5)

3. The Extended Model

The population is conveniently partitioned into seven compartments namely S(t), L(t), I(t), R(t), $V_1(t)$, $V_2(t)$, $V_3(t)$ as described above in assumptions and parameter subsection. The flow diagram for the existing model is now amended to obtain the flow diagram for the extended model as follows:



Figure 2: Flow diagram of HBV transmission dynamics for the extended model

3.1. Equations of the Extended Model

Based on the above assumptions, parameters and flow diagram of the model equations of extended model we obtain model equations are as follows:

$$\frac{dV_1(t)}{dt} = cP - (\eta + \phi_1 + \phi_4)V_1(t)$$
(6)

$$\frac{dV_2(t)}{dt} = \phi_4 V_1(t) - (\eta + \phi_2 + \phi_5) V_2(t)$$
(7)

$$\frac{dV_3(t)}{dt} = \phi_5 V_2(t) - (\eta + \phi_3) V_3(t)$$
(8)

$$\frac{dS(t)}{dt} = (1-c)P + \phi_1 V_1(t) + \phi_2 V_2(t) + \phi_3 V_3(t) - (\mu + \eta)S(t) + \pi R(t)$$
(9)

$$\frac{dL(t)}{dt} = \mu S(t) - (q+k+\eta)L(t) \tag{10}$$

$$\frac{dI(t)}{dt} = kL(t) - (\psi + \eta + \beta)I(t)$$
(11)

$$\frac{dR(t)}{dt} = \psi I(t) + qL(t) - (\pi + \eta)R(t)$$
(12)

$$N(t) = V_1(t) + V_2(t) + V_3(t) + S(t) + L(t) + I(t) + R(t)$$
(13)

4. Equilibrium Points

Let $E(V_1, V_2, V_3, S, L, I, R)$ be the equilibrium point of the system described by equations (6)-(13). At the equilibrium state, we must have

$$\frac{dV_1}{dt} = \frac{dV_2}{dt} = \frac{dV_3}{dt} = \frac{dS}{dt} = \frac{dL}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0.$$
 (14)

which gives,

$$cP - (\eta + \phi_1 + \phi_4)V_1 = 0 \tag{15}$$

$$\phi_4 V_1 - (\eta + \phi_2 + \phi_5) V_2 = 0 \tag{16}$$

$$\phi_5 V_2 - (\eta + \phi_3) V_3 = 0 \tag{17}$$

$$(1-c)P + \phi_1 V_1 + \phi_2 V_2 + \phi_3 V_3 - (\mu + \eta)S + \pi R = 0$$
(18)

$$\mu SI - (q + k + \eta)L = 0$$
 (19)

$$kL - (\psi + \eta + \beta)I = 0 \tag{20}$$

$$\psi I + qL - (\pi + \eta)R = 0 \tag{21}$$

In order to obtain the DFE state, we solve the equations (15)-(21) simultaneously.

4.1. Existence of a Trivial Equilibrium State (TES)

Let $E_0(V_{1o}, V_{2o}, V_{3o}, S_0, L_0, I_0, R_0)$ be the trivial equilibrium state (TES) of equations (6)-(13) of the model. There exists no TES since the population cannot be extinct, so long as new babies are born into the population.(i.e. $cP \neq 0$ and $(1-c)P \neq 0$).

i.e. $E_0(V_{1o}, V_{2o}, V_{3o}, S_0, L_0, I_0, R_0) \neq (0, 0, 0, 0, 0, 0, 0).$

4.2. DFE State

DFE state is the state of total elimination of disease. Let $E^0(V_1^0, V_2^0, V_3^0, S^0, L^0, I^0, R^0)$ be the DFE state. For DFE state we must have

$$I^0 = L^0 = 0 (22)$$

Putting the value of $I^0 = L^0 = 0$ into equations (15)-(21) and solving them simultaneously, we get

From equation (15), we obtain

$$V_1^o = \frac{cP}{\eta + \phi_1 + \phi_4} \tag{23}$$

From equation (16), we obtain

$$V_2^0 = \frac{\phi_4 V_1^0}{(\eta + \phi_2 + \phi_5)} \tag{24}$$

Putting the value of V_1^0 in equation (24), we get

$$V_2^0 = \frac{\phi_4 cP}{(\eta + \phi_1 + \phi_4)(\eta + \phi_2 + \phi_5)}$$
(25)

From equation (17), we obtain

$$V_3^0 = \frac{\phi_5 V_2^0}{(\eta + \phi_3)} \tag{26}$$

Putting the value of V_2^0 in equation (26), we get

$$V_3^0 = \frac{\phi_5 \phi_4 cP}{(\eta + \phi_1 + \phi_4)(\eta + \phi_2 + \phi_5)(\eta + \phi_3)}$$
(27)

From equation (18), we obtain

$$(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0 - (\mu + \eta)S^0 + \pi R^0 = 0$$
(28)

Putting the value of V_1^0 , V_2^0 and V_3^0 in equation (28), we get

$$(1-c)P + \frac{\phi_1 cP}{(\eta + \phi_1 + \phi_4)} + \frac{\phi_2 \phi_4 cP}{(\eta + \phi_1 + \phi_4)(\eta + \phi_2 + \phi_5)}$$
(29)

$$+\frac{\phi_3\phi_5\phi_4cP}{(\eta+\phi_1+\phi_4)(\eta+\phi_2+\phi_5)(\eta+\phi_3)} - (\mu+\eta)S^0 + \pi R^0 = 0$$

From equation (21), we have $qL^0 + \psi I^0 - (\pi + \eta)R^0 = 0$

$$\Rightarrow (\pi + \eta)R^{0} = 0 (since \ L^{0} = I^{0} = 0)$$
(30)

$$\Rightarrow Either(\pi + \eta) = 0 \quad or \quad R^0 = 0 \tag{31}$$

Since π and η are positive constants, $(\pi + \eta) \neq 0$ Therefore, $R^0 = 0$ Then equation (29) gives $(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0 - (\mu + \eta) S^0 = 0$ $S^0 = \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu + \eta)}$ (32)

Therefore the DFE state of the model is given by $E^0(V_1^0,V_2^0,V_3^0,S^0,L^0,I^0,R^0)$

391

$$= \left(\frac{cP}{\eta + \phi_1 + \phi_4}, \frac{\phi_4 cP}{(\eta + \phi_1 + \phi_4)(\eta + \phi_2 + \phi_5)}, \frac{\phi_4 \phi_5 cP}{(\eta + \phi_1 + \phi_4)(\eta + \phi_2 + \phi_5)(\eta + \phi_3)}, \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu + \eta)}, 0, 0, 0\right).$$

4.3. Stability Analysis of the DFE State

To determine the stability of the DFE state E^0 , we consider the nature of the model population nearby this equilibrium solution. Here, we determine the conditions that must be met if the disease is to be totally eradicated. Remind that the system of equations in this model at equilibrium state is given by

$$cP - (\eta + \phi_1 + \phi_4)V_1 = 0$$

$$\phi_4 V_1 - (\eta + \phi_2 + \phi_5)V_2 = 0$$

$$\phi_5 V_2 - (\eta + \phi_3)V_3 = 0$$

$$(1 - c)P + \phi_1 V_1 + \phi_2 V_2 + \phi_3 V_3 - (\mu + \eta)S + \pi R = 0$$

$$\mu SI - (q + k + \eta)L = 0$$

$$kL - (\psi + \eta + \beta)I = 0$$

$$\psi I + qL - (\pi + \eta)R = 0$$

(33)

We now linearize the system of equations to obtain the Jacobian matrix J which is given by

$$J = \begin{bmatrix} \omega_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ \phi_4 & \omega_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi_5 & \omega_3 & 0 & 0 & 0 & 0 \\ \phi_1 & \phi_2 & \phi_3 & \omega_4 & 0 & 0 & \pi \\ 0 & 0 & 0 & \mu I^0 & \omega_5 & \mu S^0 & 0 \\ 0 & 0 & 0 & 0 & k & \omega_6 & 0 \\ 0 & 0 & 0 & 0 & q & \psi & \omega_7 \end{bmatrix}$$
(34)

where,
$$\begin{split} &\omega_1 = -(\eta + \phi_1 + \phi_4), \omega_2 = -(\eta + \phi_2 + \phi_5), \omega_3 = -(\eta + \phi_3), \\ &\omega_4 = -(\mu + \eta), \omega_5 = -(q + k + \eta), \omega_6 = -(\psi + \eta + \beta), \omega_7 = -(\pi + \eta) \\ &\text{At the disease-free equilibrium state, } E^0(V_1^0, V_2^0, V_3^0, S^0, L^0, I^0, R^0) \text{ , the Jacobian} \end{split}$$
 matrix J becomes

$$J = \begin{bmatrix} \omega_8 & 0 & 0 & 0 & 0 & 0 & 0 \\ \phi_4 & \omega_9 & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi_5 & \omega_{10} & 0 & 0 & 0 & 0 \\ \phi_1 & \phi_2 & \phi_3 & \omega_{11} & 0 & 0 & \pi \\ 0 & 0 & 0 & 0 & \omega_{12} & \omega_{13} & 0 \\ 0 & 0 & 0 & 0 & k & \omega_{14} & 0 \\ 0 & 0 & 0 & 0 & q & \psi & \omega_{15} \end{bmatrix}$$
(35)

where

$$\begin{aligned} \omega_8 &= -(\eta + \phi_1 + \phi_4), \\ \omega_9 &= -(\eta + \phi_2 + \phi_5), \\ \omega_{10} &= -(\eta + \phi_3), \\ \omega_{11} &= -(\mu + \eta), \\ \omega_{12} &= -(q + k + \eta), \\ \omega_{13} &= \mu \frac{(1 - c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu + \eta)}, \\ \omega_{14} &= -(\psi + \eta + \beta), \\ \omega_{15} &= -(\pi + \eta) \end{aligned}$$

 $\omega_{14} = -(\psi + \eta + \beta), \omega_{15} = -(\pi + \eta)$ The characteristic equation $|J - I\lambda| = 0$ is obtained from the Jacobian determinant with the eigen values λ_i (i = 1, 2, 3, 4, 5, 6, 7)

$$\begin{bmatrix} \lambda^{2} + (2\eta + \phi_{1} + \phi_{2} + \phi_{4} + \phi_{5})\lambda + (\eta^{2} + \eta\phi_{2} + \eta\phi_{5} + \phi_{1}\eta + \phi_{1}\phi_{2} + \phi_{1}\phi_{5} + \phi_{4}\eta + \phi_{4}\phi_{2} + \phi_{4}\phi_{5}) \end{bmatrix} \times \begin{bmatrix} \lambda^{2} + (\phi_{3} + 2\eta + \mu)\lambda + (\eta\mu + \eta^{2} + \phi_{3}\mu + \phi_{3}\eta) \end{bmatrix} \quad (36)$$
$$\times (-\pi - \eta - \lambda)[A] = 0$$

where

$$[A] = \begin{bmatrix} -(q+k+\eta) - \lambda & \mu \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu+\eta)} \\ k & -(\psi+\eta+\beta) - \lambda \end{bmatrix}$$

From equation (36), either

$$[\lambda^{2} + (2\eta + \phi_{1} + \phi_{2} + \phi_{4} + \phi_{5})\lambda + (\eta^{2} + \eta\phi_{2} + \eta\phi_{5} + \phi_{1}\eta + \phi_{1}\phi_{2} + \phi_{1}\phi_{5} + \phi_{4}\eta + \phi_{4}\phi_{2} + \phi_{4}\phi_{5})] \times [\lambda^{2} + (\phi_{3} + 2\eta + \mu)\lambda + (\eta\mu + \eta^{2} + \phi_{3}\mu + \phi_{3}\eta)] \times (-\pi - \eta - \lambda) = 0$$

$$(37)$$

or

$$-(q+k+\eta) - \lambda \quad \mu \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu+\eta)} \\ k \qquad -(\psi+\eta+\beta) - \lambda \end{vmatrix} = 0$$
(38)

From equation (37), we have $\lambda[\lambda + (\eta + \phi_1 + \phi_4)] + (\eta + \phi_2 + \phi_5)[\lambda + (\eta + \phi_1 + \phi_4)]$

$$\begin{aligned} & \times \lambda [\lambda + (\phi_3 + \eta)] + (\eta + \mu) [\lambda + (\phi_3 + \eta)] (-\pi - \eta - \lambda) = 0 \\ & [\lambda + (\eta + \phi_1 + \phi_4)] [\lambda + (\eta + \phi_2 + \phi_5)] [\lambda + (\phi_3 + \eta)] [\lambda + (\eta + \mu)] [-\pi - \eta - \lambda] = 0 \end{aligned}$$

$$\lambda_1 = -(\pi + \eta) \tag{39}$$

$$\lambda_2 = -(\eta + \phi_1 + \phi_4) \tag{40}$$

$$\lambda_3 = -(\eta + \phi_2 + \phi_5) \tag{41}$$

$$\lambda_4 = -(\phi_4 + \eta) \tag{42}$$

and
$$\lambda_5 = -(\eta + \mu)$$
 (43)

Let

$$[A] = \begin{bmatrix} -(q+k+\eta) & \mu \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu+\eta)} \\ k & -(\psi+\eta+\beta) \end{bmatrix}$$

For the DFE to be asymptotically stable we must have trace(A) < 0 and det(A) > 0.

$$det(A) = (q+k+\eta)(\psi+\eta+\beta) - \mu k \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu+\eta)} \text{ and } Trace(A) = -(q+k+\eta) - (\psi+\eta+\beta)$$

Obviously, trace(A) < 0, since all the parameters q, k, η , ψ and β are positive. For the determinant of A to be positive, we must have

$$(q+k+\eta)(\psi+\eta+\beta) - \mu k \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu+\eta)} > 0$$

or

$$(q+k+\eta)(\psi+\eta+\beta) > \mu k \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu+\eta)}$$
(44)

From equations (39)-(43), $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5$ of equation (35) are all have negative real parts.

We now establish the necessary and sufficient conditions for the remaining two eigen values of equation (35) to have negative real part and this two eigen values of equation (35) will have negative real parts if and only if the determinant of A to be positive. i.e. det A > 0.

i.e.
$$(q + k + \eta)(\psi + \eta + \beta) > \mu k \frac{(1 - c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu + \eta)}$$
. The Routh-Hurwitz theorem say that the equilibrium state will be asymptotically stable if and only if all the eigen values of the characteristic equation $|J - I\lambda| = 0$ have negative real part. Using this theorem we examine that the disease-free equilibrium state of this model will be asymptotically stable if and only if

A Mathematical Model of Transmission Dynamics of Hepatitis B Virus

$$(q+k+\eta)(\psi+\eta+\beta) > \mu k \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu+\eta)}$$

$$\Rightarrow \quad \mu k \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu+\eta)} < (q+k+\eta)(\psi+\eta+\beta)$$
(45)

The above inequality gives the necessary and sufficient condition for the DFE state of the model to be asymptotically stable.

Alternatively, the inequality (44) can also be expressed as

$$(q+k+\eta) > \mu k \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu+\eta)(\psi+\eta+\beta)}$$

This shows that sum of the rate of recovery of latently infected people and the rate at which latently infected individuals improvement to active infection and the rate of natural death of individuals (in the population, i.e. total removal rate from the latent class) must obtain a lower bound given by $\mu k \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu + \eta)(\psi + \eta + \beta)}$

4.4. Reproduction Number (R_0)

The basic reproduction number (R_0) is the number of cases directly caused by an infected individual throughout his infectious period. R_0 is used to determine the ability of a disease to spread within a given population. The reproduction number (R_0) represents the transmissibility of a disease. The most important uses of R_0 are determining if an emerging infectious disease can spread in a population and determining what proportion of the population should be immunized through vaccination to eradicate a disease. If $(R_0) > 1$, then each person on average infects more than one other person so the disease will spread. If $(R_0) < 1$, then each person infects less than one person on average so the disease will die out. If $(R_0) = 1$, then each person will infect exactly one other person so the disease will become endemic, it will move throughout the population but not increase or decrease.

There are many simple methods for finding the reproduction number R_0 , namely, the survival function method, next generation method, constant term of the characteristic polynomial etc.

Here, we find R_0 , using the constant term of the characteristic polynomial. From equation (35), characteristic equation for the Jacobian matrix at the DFE state is given by

$$\begin{split} \lambda^2 &- [-(q+k+\eta) - (\psi+\eta+\beta)]\lambda + (q+k+\eta)(\psi+\eta+\beta) \\ &- \mu k \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu+\eta)(\psi+\eta+\beta)} = 0 \end{split}$$

Therefore the basic reproduction number (R_0) is given by

395

$$R_0 = \mu k \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu+\eta)(q+k+\eta)(\psi+\eta+\beta)}$$

The above equation has all roots with negative real parts if and only if each coefficient is positive. Hence $R_0 < 1$ and the disease will die out.

5. Conclusion

In this paper, we have proposed new mathematical model for transmission dynamics of HBV infection. The proportion dynamics of the classes is described using seven differential equations. We conclude that the trivial equilibrium state $E_0(V_{1o}, V_{2o}, V_{3o}, S_0, L_0, I_0, R_0)$ is unstable. The state there is no individual in the population. The disease-free equilibrium state, $E^0(V_1^0, V_2^0, V_3^0, S^0, L^0, I^0, R^0)$ of the model was determined and its stability analysed using the Routh-Hurwitz theorem. The basic reproduction number, R_0 for the model was computed using the constant term of the characteristic polynomial and shown that disease will die out if we carry out vaccination program so that $\mu k \frac{(1-c)P + \phi_1 V_1^0 + \phi_2 V_2^0 + \phi_3 V_3^0}{(\mu + \eta)(q + k + \eta)(\psi + \eta + \beta)} < 1$.

Acknowledgement

One of the authors (Dr. R. S. Jain) is thankful to the Swami Ramanand Teerth Marathwada University for providing financial assistance to carry out this research under Minor Research Project grant.

References

- Abu, O., Onalo, S. E., Numerical analysis of a mathematical model of hepatitis B virus transmission dynamics in the presence of vaccination and treatment, Journal of Scientific and Engineering Research, 4(9) (2017), 295-310.
- [2] Ali, V. K., Reza, A., Ali, A. H. and Aghileh, H., Mathematical modeling of transmission dynamics and optimal control of vaccination and treatment for hepatitis B virus, Computational and Mathematical Methods in Medicine., Vol. 2014, (2014). Article ID 475451, 15 pages.
- [3] Anderson, R. M. and May, R. M., Infectious diseases of human: dynamics and control, Oxford University Press, Oxford, UK., 28 (1992).
- [4] Blessing, O. E., Simeon, C. I., Mathematical model and analysis of hepatitis B virus transmission dynamics, (2018). doi: 10.12688/f1000research.15557.1.
- [5] Funk, S., Salathe, M. and Vincent, A. A., Modelling the influence of human behaviour on the spread of infectious diseases, J. R. Soc. Interface, (2010). doi:10.1098/rsif.2010.0142.pp.1247-1256.

- [6] Kimbir, A. R., Aboiyar, T, Abu, O. and Onah, E. S., A Simulation of a mathematical model of HBV transmission dynamics in the presence of vaccination and treatment, Mathematical Theory and Modeling, Vol. 4, No. 12 (2014), 44-59.
- [7] Kimbir, A. R., Aboiyar, T., Abu, O. and Onah, E. S., Modelling the HBV transmission dynamics in the presence of vaccination and treatment, Mathematical Theory and Modeling., Vol.4, No. 12 (2014), 29-43.
- [8] Medley, G. F., Lindop, N. A., Edmunds, W. J. and Nokes, D. J., Hepatitis-B virus endemicity: heterogeneity, catastrophic dynamics and control, Nature Medicine, vol. 7, no. 5 (2001), 619-624.
- [9] Mehmood, N., Modelling the transmission dynamics of hepatitis B and optimal control, J. Theor. Biol., Vol. 13 (2011), 1-17.
- [10] O'Leary, C., Hong, Z., Zhang, F., Dawood, M., Smart, G., Kaita, K. and Wu, J., A mathematical model to study the effect of hepatitis B virus vaccine and anti-virus treatment among the Canadian inuit population, CDC, (2008).
- [11] Stanca, M. C., Ruy, M. R., Patrick, W. N. and Alan, S.P., Modeling the mechanisms of acute hepatitis B virus infection, Journal of Theoretical Biology, 247 (2007), 23-35.
- [12] Thornley, S., Bullen, C. and Roberts, M., Hepatitis B in a high prevalence New Zealand population: a mathematical model applied to infection control policy, Journal of Theoretical Biology, vol. 254, no. 3 (2008), 599-603.
- [13] Wang, K., Zhang, T., Zhang, X., Modeling and analyzing the transmission dynamics of HBV epidemic in xinjiang, china., PLOS ONE, 10(9) (2015), e0138765. doi:10.1371/journal.pone.0138765.
- [14] Yan, C., Qiuhui, P. and Mingfeng, H., Stability analysis of hepatitis B virus model with incomplete immunization of hepatitis B vaccine, Abstract and Applied Analysis, Vol. 2014, (2014). Article ID 427639, 10 pages.
- [15] Zou, L., Zhang, W. and Ruan, S., Modeling the transmission dynamics and control of hepatitis B virus in China, Journal of Theoretical Biology, Vol. 262, no. 2 (2010), 330-338.