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Modeling the Effects of Detection and Treatment of Latent Hepatitis B Infection on Transmission Dynamics of Hepatitis B Disease

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Abstract

In this paper, we proposed an SVEIR model to understand the effect of detection and treatment of Hepatitis B at latent stage on the transmission dynamics of hepatitis B disease. Mathematical analysis was carried out that completely determines the global dynamics of the model. The impact of detection and treatment of Hepatitis B at latent stage on the transmission dynamics are discussed through the stability analysis of the disease free equilibrium.

Keywords: Hepatitis B Virus (HBV), Mathematical Model, Latent Hepatitis, Stability analysis

1. Introduction

Hepatitis B virus is the most common cause of cirrhosis and hepatocellular carcinoma in the world today. Of approximately 2 billion people who have been infected with HBV worldwide, more than 350 million, or about 5% of the world's population are chronic carriers, and with an annual incidence of more than 50 million (Khan *et al* 2007 & Patel *et al* 2004). Hepatitis B virus accounts for 500,000 to 1.2 million death per year.

Compartmental mathematical models have been widely used to gain insight into the spread and control of emerging and re-emerging human disease dating back to the pioneering work of Bernoulli in 1760 and likes of Ross, Kermack and McKendrick and others (Anderson *et al* 1982, 1991). The study of infectious diseases has been transformed by the use of mathematical models to gain insight into the dynamics of epidemics, to identify potential public health interventions, and to assess their impact (McCluskey *et al* 2003). Mathematical models were useful in informing policy during the foot and mouth disease outbreak in the United Kingdom in 2001, during the Severe Acute Respiratory Syndrome (SARS) outbreak in 2003 and in recent planning of responses to potential smallpox or pandemic influenza outbreak.

In this paper, we propose an SVEIR model to understand the effect of detection and treatment of hepatitis B at latent stage on the transmission dynamics of the disease. We solved the system of ordinary differential equation and obtain our disease free equilibrium. Stability analysis was carried out on the disease free equilibrium using Routh-Hurtwitz Theorem. The model derivation is given the next sections.

2. Model Formulation

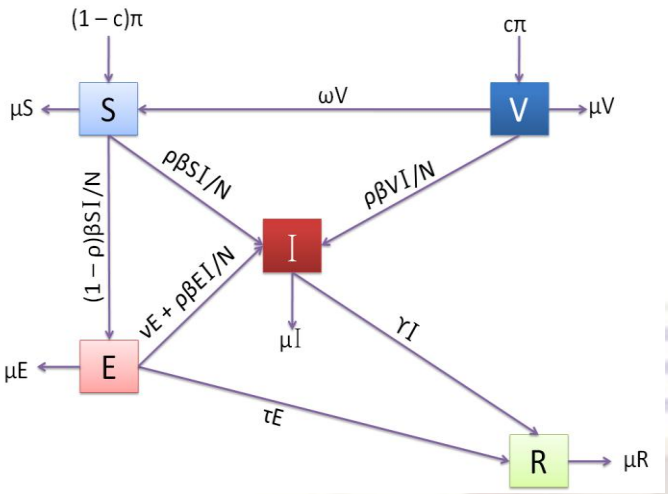
As with any modeling endeavor, various assumptions about the underlying biology must be made. At this stage, we wish to clearly state some assumptions.

- We assume that throughout the duration of the vaccines efficacy, latent Hepatitis B is completely undetectable.
- We also assume that the birth and mortality rate are equal.
- We assume that the efficacy of the vaccines wanes out at the rate ω.

Individuals enters the population with a recruitment rate of π. The natural mortality rate is denoted by μ. A proportion, c, of individuals entering the population are vaccinated as infants and hence enter the vaccinated class, V. the remaining proportion, $1-c$, enter the susceptible class, S. the efficacy of the vaccines used in the vaccines used in the vaccination wanes over time at the rate of ω and result in the movement of individuals from the vaccinated class to the susceptive class. We use the frequency-dependent description of disease transmission, with transmission coefficient β.

We assume that assume that, upon infection with hepatitis B, an individual can either progress quickly to active hepatitis B or develop a latent infection and progress slowly to active hepatitis B. we denote the classes of latently infected and infected individuals by E and I respectively. The probability of a random individual exhibiting fast progression to active hepatitis B is denoted by ρ. latent infections progress slowly to active disease at the rate ν. We assume that individuals in the latent hepatitis class moves to removed class at the rate τ when treated. We also assume that active hepatitis B disease clears at the rate γ.

Model Diagram



Model Equations

$$\frac{dS}{dt} = (1-c)\pi - \frac{\beta SI}{N} + \omega V - \mu S \quad (1)$$

$$\frac{dV}{dt} = c\pi - \omega V - \frac{\rho\beta VI}{N} - \mu V \quad (2)$$

$$\frac{dE}{dt} = (1-\rho)\frac{\beta SI}{N} - \nu E + \rho\frac{\beta EI}{N} - \mu E - \tau E \quad (3)$$

$$\frac{dI}{dt} = \nu E + \rho\frac{\beta EI}{N} + \rho\frac{\beta SI}{N} + \rho\frac{\beta VI}{N} - \mu I - \gamma I \quad (4)$$

$$\frac{dR}{dt} = \gamma I - \mu R + \tau E \quad (5)$$

3. Mathematical Analysis

In this section, we wish to study the equilibrium state and analyze for stability of the system.

Disease Free Equilibrium

To establish analytic thresholds for when vaccination is a society's prudent choice, we consider the case of a population at the brink of eradicating hepatitis B. Mathematically; we must therefore consider the disease free equilibrium (DFE).

We recall that the full system is given by

$$\frac{dS}{dt} = (1-c)\pi - \frac{\beta SI}{N} + \omega V - \mu S \quad (1)$$

$$\frac{dV}{dt} = c\pi - \omega V - \frac{\rho\beta VI}{N} - \mu V \quad (2)$$

$$\frac{dE}{dt} = (1-\rho)\frac{\beta SI}{N} - \nu E + \rho\frac{\beta EI}{N} - \mu E - \tau E \quad (3)$$

$$\frac{dI}{dt} = \nu E + \rho\frac{\beta EI}{N} + \rho\frac{\beta SI}{N} + \rho\frac{\beta VI}{N} - \mu I - \gamma I \quad (4)$$

$$\frac{dR}{dt} = \gamma I - \mu R + \tau E \quad (5)$$

$\tau = \frac{r}{1-r}(\mu+\nu)$ where r is the population of latent infections which are detected and successfully treated before progression to active hepatitis B or death.

If a population is free of hepatitis B infection $(i.e.\ E = I = 0)$, the system reduces to

$$\frac{dS}{dt} = (1-c)\pi + \omega V - \mu S$$

$$\frac{dV}{dt} = c\pi - \omega V - \mu V$$

$$\frac{dR}{dt} = -\mu R$$

Recalling that $\frac{dS}{dt} = \frac{dV}{dt} = \frac{dR}{dt} = 0$ is the necessary for an equilibrium, we see that the derived as follows

$$(1-c)\pi + \omega V - \mu S = 0$$

$$c\pi - \omega V - \mu V = 0$$

$$-\mu R = 0$$

From (11), $R^* = 0$

Also, from (10), $c\pi - (\omega+\mu)V = 0$

$$V^* = \frac{c\pi}{(\omega+\mu)}$$

Substituting (12) into (9) we have,

$$(1-c)\pi + \omega V - \mu S = 0$$

$$(1-c)\pi + \omega\left(\frac{c\pi}{(\omega+\mu)}\right) - \mu S = 0$$

$$S^* = \frac{(1-c)\pi\mu + \omega\pi}{\mu(\omega+\mu)}$$

The disease free equilibrium of the model is given by

$$X^* = (S^*, V^*, E^*, I^*, R^*) = \left(\frac{(1-c)\mu\pi + \omega\pi}{\mu(\omega+\mu)}, \left(\frac{c\pi}{(\omega+\mu)}\right), 0, 0, 0\right)$$

4. Stability Analysis of Disease Free Equilibrium

We find out that the Jacobian of F about X^* is

$$DF(X^*) = \begin{bmatrix} -\mu-\lambda & \omega & 0 & -\beta S^* & 0 \\ 0 & -(\omega+\mu)-\lambda & 0 & -\rho\beta V^* & 0 \\ (1-\rho)\beta I^* & 0 & -(\nu+\mu+\tau)-\lambda & \rho\beta E^* & 0 \\ 0 & 0 & \nu+\rho\beta E^* & -(\mu+\gamma)-\lambda & 0 \\ 0 & 0 & \tau & \gamma & -\mu-\lambda \end{bmatrix}$$

At disease free equilibrium, $I^* = E^* = 0,$ the Jacobian reduce to

$$DF(X^*) = \begin{bmatrix} -\mu-\lambda & \omega & 0 & -\beta S^* & 0 \\ 0 & -(\omega+\mu)-\lambda & 0 & -\rho\beta V^* & 0 \\ 0 & 0 & -(\nu+\mu+\tau)-\lambda & 0 & 0 \\ 0 & 0 & \nu & -(\mu+\gamma)-\lambda & 0 \\ 0 & 0 & \tau & \gamma & -\mu-\lambda \end{bmatrix}$$

Noticing the form of the Jacobian, we immediately have that $-\mu-\omega$ is an eigen value and that $-\mu$ is a double eigen values. The remaining eigen values are those of the 2×2 sub-matrix.

$$\begin{bmatrix} -(\nu+\mu+\tau)-\lambda & 0 \\ \nu & -(\mu+\gamma)-\lambda \end{bmatrix} \quad (14)$$

To determine the nature of eigen values in (14), we present the Routh-Hurwitz necessary and sufficient condition for all roots of the characteristics polynomials to have negative real parts, thus implying asymptotic stability as applied by Ssematimba (2005) and Benyah (2008).

Routh-Hurwitz theorem

Let $J = \begin{pmatrix} F_x(x_*, y_*) & F_y(x_*, y_*) \\ g_x(x_*, y_*) & g_y(x_*, y_*) \end{pmatrix}$

be the Jacobian matrix of the non-linear system

$$\left.\begin{aligned} \frac{dx}{dt} &= f(x,y) \\ \frac{dy}{dt} &= g(x,y) \end{aligned}\right\}$$ evaluated at the critical point (x_*, y_*).

Then the critical point (x_*, y_*)

1. is asymptotically stable if trace $(J) < 0$ *and* $\det(J) > 0$
2. is stable but not asymptotically stable if trace $(J) = 0$ *and* $\det(J) > 0$
3. is unstable if either, trace $(J) < 0$ *and* $\det(J) > 0$

Let $A = \begin{pmatrix} -(\nu+\mu+\tau) & 0 \\ \nu & -(\mu+\gamma) \end{pmatrix}$

Then from matrix A we have,

$\det(A) = (\nu+\mu+\tau)(\mu+\gamma)$ and

$trace(A) = -(\nu+\mu+\tau)-(\mu+\gamma)$

Substituting $\tau = \frac{r}{1-r}(\mu+\nu)$ we have,

$$\frac{(\nu(1-r)+\mu(1-r)+r(\mu+\nu))(\mu+\gamma)}{1-r}$$

Thus, our model is asymptotically stable when $r < 0.$

Conclusion

From our analysis, we found out that the disease free equilibrium is asymptotically stable when the population of latent infections which are detected and successfully treated before progression to active hepatitis B or death is less than zero. Thus, if a country is able to detect and treat latent hepatitis B infection at the rate which exceed τ, the discontinuation of HBIB* vaccination will increase the stability of the disease free equilibrium. If the detection and treatment rate of latent hepatitis B is below τ, HBIB* vaccination will result in a more stable disease free equilibrium.

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