Applied Mathematical Sciences, Vol. 8, 2014, no. 67, 3343 - 3358
HIKARI Ltd, www.m-hikari.com
http://dx.doi.org/10.12988/ams.2014.44263

Mathematical Model of Hepatitis B in the Bosomtwe District of Ashanti Region, Ghana

Isaac Kwasi Adu

Department of Mathematics
Valley View University, Techiman Campus
P. O. Box 183 B/A-Ghana

Anthony Yaw Aidoo

Department of Mathematics and Computer Science,
Eastern Connecticut State University,
Willimantic, USA

Isaac Owusu Darko

Department of Mathematics
Valley View University, Techiman Campus
P. O. Box 183 B/A-Ghana

Emmanuel Osei-Frimpong

Department of Mathematics
Kwame Nkrumah University of Science and Technology
Kumasi-Ghana

Abstract

We use SIR model to predict the prevalence and incidence of Hepatitis B in Bosomtwe District of Ghana. The study is made up of two sections. An SIR model without vaccination is used to explain the spread of the HBV in the Bosomtwe district followed by the modeling HB with vaccination in the district.

The model has two equilibrium states: the disease-free equilibrium and the endemic equilibrium states respectively. The stability condition of each equilibrium point is discussed. The basic reproductive number (R_0) of HB without vaccination is estimated to be 1.006 and the basic reproductive number (R_0) of HB with vaccination is estimated to be 0.9840. Our work shows that, the proportion of the population of Bosomtwe district that needs to be vaccinated in order to control HBV in the district is 871. According to the results of this study, whenever the transmission rate parameter value is increased, $R_0 > 1$, but when the transmission rate parameter value is reduced, $R_0 < 1$. A combination of increased vaccination of newborns and immunization of susceptible adults appears to reduce HB prevalence in Bosomtwe District to the minimum.

Keywords: HB, HBV, SIR Model, Epidemiology, Mathematical Model, Herd Immunity

.

1 Introduction

Hepatitis B (HB) is an infectious disease caused by hepatitis B virus (HBV) which infects the liver of hominoids, including humans and causes an inflammation called hepatitis. Originally known as "serum hepatitis", the disease has caused epidemics in parts of Asia and Africa, and it is endemic in China. About a third of the world's population, more than 2 billion people have been infected with the hepatitis B virus. This includes about 350 million chronic carriers of the virus [1].

The disease lasts for a few weeks and then gradually improves in most affected people. A few patients may have more severe liver disease (fulminant hepatic failure), and may die as a result of it. This infection may be entirely asymptotic and may go unrecognized. Chronic infection with hepatitis B may be either asymptotic or may be associated with a chronic inflammation of the liver (Chronic hepatitis), leading to cirrhosis over a period of several years. This type of infection dramatically increases the incidence of hepatocellular carcinoma (liver cancer). Chronic carriers are encouraged to avoid consuming alcohol as it increases their risk for cirrhosis and liver cancer [2].

In Ghana, the sero-prevalence rate was found to be about 15% in some studies [3]. Sero-prevalence surveys indicate that HBsAg carriage rates range from 4.8% in Greater Accra Region [4] to 21% in the Upper East Region. A small but significant number of infants get hepatitis in the neonatal period but childhood infection is very high. It has been estimated that 15% of the HBV carrier rate in Ghana is acquired through vertical transmission [5]. Prevalence of HBsAg among Ghanaian rural children aged 1-16 years was 21% (carriers of HBsAg indicate recent or chronic infection with HBV) and of any HBV marker (anti HBV, HBV Ag or HBeAg) was 75% [6]. In attempt to skirmish the HB in Ghana, various campaigns spearheaded by the Eagles Alliance clubs have been launched to create

awareness on the disease and for people to adopt responsible lifestyles to prevent its spread.

Ghana Medical Association Journal reports that, hepatic cirrhosis is the commonest liver disease causing death in Accra [7]. A nested case-control study was carried out to determine the phenomenon and the role of blood transfusion in transmission of the two viruses. A total of 70 patients with cirrhosis diagnosed on combined clinical and ultrasonographic evidence and 280 controls with non-hepatic diseases were recruited for the study. Further, anecdotal evidence from Bosomtwe District Hospital shows that Hepatitis B is endemic in the district. Several studies have attempted to model epidemiology of hepatitis B in various countries. However, mathematical models have not been used to study the spread of the disease in Ghana. Moreover, the role of hepatitis B (HBV) and C (HCV) virus infections in cirrhosis has not been well researched and documented generally in Ghana and Bosomtwe District in parts. The current study therefore aims at analyzing, modeling and predicting the spread of Hepatitis B disease in the Bosomtwe District of Ghana. The district was chosen for the study because it is one of the districts where the disease is considered to be endemic. The model captures salient aspects of epidemiology of Hepatitis B disease in the Bosomtwe district in Ghana, predicting the spread of the disease in the district. Two variations of the standard susceptible-infected-recovered (SIR) epidemiological model are utilized to study and analyze the spread of the disease. These are the basic SIR model to explain the spread of the HBV in Bosomtwe district, and by a model with vaccination component in the district.

2 Model Description

The model is a modification of the Kermack and Mckendrick model [8]. In the standard SIR model, population is divided into three compartments. These are the susceptible individuals (S) who are susceptible if they come into contact with the virus; the infected group (I), made up of all the individuals who are infected by the disease and can pass it on to the susceptible individuals; and the removed individuals R, who have recovered from the disease or are removed from HB infection. The proportions of individuals in the compartments S, I and R, at time t, is denoted as $S(t), I(t)$ and $R(t)$, respectively. Where β, is the transmission rate coefficient and γ is the removed rate coefficient. The flowchart in Figure 2.1 is the flowchart of SIR model.

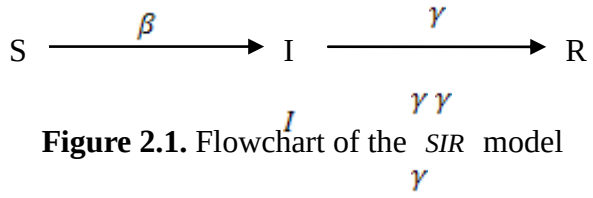

Figure 2.1. Flowchart of the SIR model

2.1 The Model Assumptions

The disease spreads in a closed environment; that is there is no emigration or immigration, and neither birth nor death in the population, so that the total population remains a constant K for all t, that is $S(t) + I(t) + R(t) = K$. If we let $s(t) = S(t)/K, i(t) = I(t)/K$ and $r(t) = R(t)/K$ then $r(t) + i(t) + r(t) = 1$, where $s(t)$, $i(t)$ and $r(t)$ are susceptible, infected and recovery fractions of the population respectively.

1. The number of susceptible who are infected by an infective individual per unit of time, at time t, is proportional to the total number of susceptible with the proportional coefficient (transmission coefficient) β, so that the total number of new infectives, at time t, is $\beta S(t) I(t)$.
2. The number removed (recovered) individuals from the infected compartment per unit time is $\gamma I(t)$ at time t, where γ is the recovery rate coefficient and the recovered individuals gain permanent immunity.
3. Age, sex, social status, and race do not affect the probability of being infected.
4. The members of the population mix homogeneously (have the same interactions with one another to the same degree.)

Here β is the infection rate of HBV and γ is the recovery rate of HBV respectively.
The model equations are as follows:

$$\frac{dS}{dt} = -\beta SI \qquad (1)$$

$$\frac{dI}{dt} = \beta SI - \gamma I \qquad (2)$$

$$\frac{dR}{dt} = \gamma I \qquad (3)$$

The equation for variable R is decoupled from the first two equations of the system (1) and (2), so we only need to consider the system:

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dS}{dt} = \beta SI - \gamma I$$

Equations (1) and (2) can be simplified as follows

$$\frac{dI}{dS} = (\beta SI - \gamma I)\left(\frac{1}{-\beta SI}\right)$$

$$\frac{dI}{dS} = -1 + \frac{\rho}{S} \qquad (4)$$

where $\rho = \frac{\gamma}{\beta}$ and ρ is the relative removal rate and its reciprocal $\frac{\beta}{\gamma}$ is the infections contact rate.

2.2 Basic Reproductive Ratio (R_0) Of HBV Transmission without Vaccination

The basic reproductive ratio is the average number of secondary infections produced when one infective is introduced into a host population where everyone is susceptible. The reproductive ratio can therefore be defined as

$$R_0 = \frac{\beta S_0}{\gamma} = \frac{S_0}{\rho} \qquad (5)$$

That is the infectious contact rate multiplied by the initial number of susceptible. If more than one secondary infection is produced from one primary infection, that is, $R_0 > 1$, then an epidemic occurs. When $R_0 < 1$, then there is no epidemic and the disease dies out. When $R_0 = 1$, the disease becomes endemic, meaning the disease remains in the population at a consistent rate, as one infected individual transmits the disease to one susceptible [9].

From equation (4):

$$\frac{dI}{ds} = -1 + \frac{\gamma}{\beta S}$$

Using separation of variables

$$dI = \left(-1 + \frac{\gamma}{\beta S}\right) ds$$

$$\int ds = -\int 1ds + \frac{\gamma}{\beta}\int \frac{1}{S} ds$$

$$I = -S + \frac{\gamma}{\beta} InS + C$$

Where C is an arbitrary constant. Thus the orbits of the solution are given multiplicity by the equation

$$I + S - \frac{\gamma}{\beta} InS = C$$

We assume the following as initial conditions: $S_0 = S(0)$ and $I_o = I(o)$.. We also

assume that $lim_t \to \infty I(t) = 0$ where $S\infty = Lim_t \to \infty S(t)$ gives the final number of susceptible, individuals after the epidemic is over. The above equality holds for both (S_0, I_0) and for $(S_\infty, 0)$.

Thus

$$Io + So - \frac{\gamma}{\beta} 1nSo = C$$

Consequently

$$Io + So - \frac{\gamma}{\beta} InSo = S_\infty - \frac{\gamma}{\beta} InS_\infty$$

Rearranging the terms, we have

$$Io + So - S_\infty = \frac{\gamma}{\beta} (InSo - InS_\infty)$$

$$\frac{\gamma}{\beta} = \frac{Io + So - S\infty}{InSo - InS\infty} \qquad (6)$$

If we let $K = Io + So$ and $\frac{\gamma}{\beta} = \rho$ then equation (3.6) Becomes

$$\rho = \frac{K - S\infty}{InSo - InS\infty} \qquad (7)$$

Also from equation (3.6)

$$\frac{\beta}{\gamma} = \frac{InSo - InS\infty}{Io + So - S\infty}$$

$$\frac{\beta}{\gamma} = \frac{In\frac{So}{S\infty}}{Io + So - S\infty} \qquad (8)$$

We note that since the population is constant $S\infty < So + Io.$
The implicit solution also allows us to compute the maximum number of infected individuals that is attained. This number occurs when $I = 0$, that is when

$$\rho = S = \frac{\gamma}{\beta}$$

From

$$I + S - \frac{\gamma}{\beta} InS = Io + So - \frac{\gamma}{\beta} InSo.$$

Substituting the expression for S and moving all the terms but I to the right – hand side leads to

$$I_{\max} = -\frac{\gamma}{\beta} + \frac{\gamma}{\beta} + In\frac{\gamma}{\beta} + S_0 + I_0 - \frac{\gamma}{\beta} InS_0 \qquad (9)$$

Finally if the expression for ρ is substituted into equation (8) we have

$$I_{\max} = -\rho + \rho In\rho + S_0 + I_0 - \rho InS_0$$

$$I_{\max} = K - \rho + \rho In\rho - \rho InS_0$$

Since

$$K = S_0 = I_0$$

$$I_{\max} = K - \rho + \rho(In\rho - InS_0) \qquad (10)$$

$$I_{\max} = K - \rho + \rho In\frac{\rho}{S_0}$$

$$I_{\max} = K - \rho(1 + InR_0) \qquad (11)$$

Equation (11) will be used to calculate for the maximum number of infectives during the epidemic.

3 SIR Model of HBV Transmission with Vaccination

We assume that vaccination against HBV leads to permanent immunity. We also assume that a portion of susceptible, pS go to the removed compartment R directly, due to permanent immunity obtained from vaccination

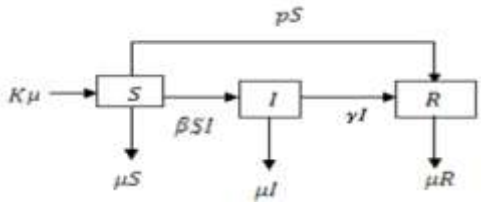

Figure 3.1 Flow chart for *SIR* model of Hepatitis B with vaccination

$$\frac{ds}{dt} = \mu K - \beta SI - \mu S - pS \qquad (12)$$

$$\frac{dI}{dt} = \beta SI - (\gamma + \mu) I \qquad (13)$$

$$\frac{dR}{st} = \gamma I - \mu R + pS \qquad (14)$$

where p is the vaccinating rate coefficient for susceptible.

Table 1. Model parameters

Parameter	Description	Value
μ	Natural death rate	0.0219
β	Infection rate	0.9842
γ	Recovery rate	2.5447
pS	Vaccinated susceptible	1.0×10^{-4}

3.1 Model Analysis

Equilibrium Points

In order to determine the stability of the model we first evaluate the equilibrium points or steady states of the system of the ordinary differential equations 11, 12, and 13. The points to be found are disease-free (where $I = 0$), and endemic (where $I \neq 0$). Setting the right-hand side of the equations in the system 3.12, 3.13, and 3.14 to zero and then solve for the values of $S, I,$ and R , we obtain

$$(S^*, I^*, R^*) = \left(\frac{\gamma+\mu}{\beta}, \frac{\beta\mu K - (\mu+p)(\gamma+\mu)}{\beta(\gamma+\mu)}, \frac{\beta K\gamma - (\mu+\gamma)(\gamma-p)}{\beta(\mu+\gamma)} \right) \quad (15)$$

3.2 The Disease-Free Equilibrium

At the disease free equilibrium, is the case where there is no infection. That is when $I = 0$. Putting $\frac{ds}{dt} = 0,$ the disease free equilibrium is $(S^*, I^*) = (1,0)$.

3.3 Stability Analysis of Disease-Free Equilibrium Point

To determine the stability of the system at the disease-free equilibrium $(S,I) = (1,0),$ we will consider the linearized system of the equations below about the equilibrium point by taking the Jacobian.

$$\frac{ds}{dt} = \mu K - \beta SI - \mu S - pS$$

$$\frac{dI}{dt} = \beta SI - (\gamma + \mu) I$$

The Jacobian is therefore given by

$$J(S,I) = \begin{bmatrix} -\beta I - \mu - p & -\beta S \\ \beta I & \beta S - \gamma - \mu \end{bmatrix}$$

The Jacobian matrix becomes

$$J(S,0) = \begin{bmatrix} -\mu - p & -\beta S \\ 0 & \beta S - \gamma - \mu \end{bmatrix},$$

since $S = 1$ and $I = 0$ at the disease free equilibrium.

$$\text{Det}_{(J-\lambda)} = \begin{vmatrix} -\mu - p - \lambda & -\beta S \\ 0 & \beta S - \gamma - \mu - \lambda \end{vmatrix} = 0$$

Det $(J - \lambda) = 0$

$(-\mu - p - \lambda)(\beta S - \gamma - \mu - \lambda = 0 \lambda_1 = -\mu - p$ and $\lambda_2 = \beta S - \gamma - \mu$

For Det$(J - \lambda)$ to be asymptotically stable, both eigenvalues must be negative. From Det $(J - \lambda)$=0, it is clear that $\lambda_1 = -\mu - p$ is negative and therefore if $\lambda_2 = \beta S - \gamma - \mu < 0$ then both eigenvalues are negative and $R_0 < 1$. Hence the disease free-equilibrium is asymptotically stable if $R_0 < 1$. On the other hand, if $\lambda_2 = \beta S - \gamma - \mu > 0$, then Det $(J - \lambda)$ is unstable.

We now investigate the stability of the equilibrium by deriving the reproductive ratio, R_0.

3.4 The Basic Reproductive Ratio (R_0) Of HBV Transmission with Vaccination

In order to determine the stability of the systems (12) and (13) at the point $(S^*, 0)$, we substitute $I = 0$ and $S = S^*$ into the linearized Jacobian matrix to find the eigenvalues as follows

$$J(S,0) = \begin{bmatrix} -\mu - p & -\beta S \\ 0 & \beta S - \gamma - \mu \end{bmatrix}$$

$$\text{Det}(J - \lambda) = \begin{vmatrix} -\mu - p - \lambda & -\beta S \\ 0 & \beta S - \gamma - \mu - \lambda \end{vmatrix} = 0$$

$$(-\mu - p - \lambda)(\beta S - \gamma - \mu - \lambda) = 0$$

$$\lambda_1 = -\mu - p$$

Also $$(\beta S - \gamma - \mu) - \lambda] = 0$$

$$\lambda_2 = \beta S - \gamma - \mu$$

The parameter ranges are as follows, $0 < \mu < 1, 0 < p < 1, 0 < \gamma < 1$ and $0 < \beta < 1$.

λ_2 depends on β, S, γ and μ. Although, the first term βS is positive, the second and third terms are negatives. This implies that the sign of the λ_2 depends on βS, but $0 < \beta < 1$. If S becomes large, λ_2 will be positive and if S becomes small, β will make the value of the first term small, thereby making the λ_2 negative. Therefore $\beta S << \gamma$ and λ_2 is negative. Also $p << \gamma$ and that makes λ_2 the

most negative.
Therefore λ_2 is the dominant eigenvalue, since it is the most negative eigenvalue.

$$Ro = \lambda^* \times (\text{Infectious period}) + 1$$

We now consider the initial condition $S = S_0$

$$R_0 = \left(\frac{\beta So - \gamma - \mu}{1}\right) * \frac{1}{(\mu + \gamma)} + 1$$

$$R_{0.} = \frac{\beta So}{(\mu + \gamma)} \tag{16}$$

This is the average number of secondary infections caused by one infected individual during the mean course of infection in the case where the number of susceptible is counted from the disease-free equilibrium; that is the total susceptible of the population when disease is free.
When $Ro > 1$ there is an endemic and the solution is unstable but when $R_0 < 1$ a positive equilibrium occurs and the solution is stable.

3.5 The Endemic Equilibrium

We now consider the case where R_o > 1 when the system has an endemic infection. Then $(S^*, 0)$ is unstable.
From equations (3.12), (3.13) and (3.14)

$$S^* = \frac{\gamma + \mu}{\beta} \text{ and } I^* = \frac{\beta\mu K - (\mu + p)(\gamma + \mu)}{\beta(\gamma + \mu)}$$

Thus the endemic equilibrium is

$$(S^*, I^*) = \left(\left(\frac{\gamma + \mu}{\beta}\right), \frac{\beta\mu K - (\mu + p)(\gamma + \mu)}{\beta(\gamma + \mu)}\right)$$

To prove that (S^*, I^*) is asymptotically stable, substitute S = S^* and $I = I^*$ into the Jacobian matrix to find the eigenvalues λ.

$$(J - \lambda I) =$$
$$\begin{bmatrix} -\beta\frac{[\beta\mu K - (\mu + p)(\gamma + \mu)]}{\beta(\gamma + \mu)} - \mu - p - \lambda) & -\frac{\beta(\gamma + \mu)}{\beta} \\ \beta\left(\frac{\beta\mu K - (\mu + p)(\gamma + \mu)}{\beta(\gamma + \mu)}\right) & \beta\left(\frac{\gamma+\mu}{\beta}\right) - \gamma - \mu - \lambda \end{bmatrix}$$

$$\text{Det}\ (J - \lambda I) = \begin{vmatrix} \frac{-\beta\ \mu\ K}{(\gamma\ +\ \mu)} - \lambda & (-\gamma + \mu) \\ \frac{\beta\ \mu\ K}{(\gamma\ +\ \mu)} & -(\mu + p) - \lambda \end{vmatrix} = 0$$

$$\lambda^2 - \lambda\left(\frac{\beta\mu K}{\gamma\ +\ \mu}\right) - \beta\mu K + (\mu\ +\ p)(\gamma\ +\ \mu) = 0 \quad (17)$$

3.6 Stability Analysis of Endemic Equilibrium

The Jacobian matrix $(J - \lambda I)$ at the endemic equilibrium state,

$$(S^*, I^*) = \left(\left(\frac{\gamma\ +\ \mu}{\beta}\right), \frac{\beta\mu K - (\mu + p)(\gamma\ +\ \mu)}{\beta(\gamma\ +\ \mu)}\right) \quad (18)$$

is given by

$$(J - \lambda I) = \begin{bmatrix} -\beta\frac{[\beta\mu K - (\mu\ +\ p)(\gamma\ +\ \mu)]}{\beta(\gamma\ +\ \mu)} - \mu - p - \lambda & -\frac{\beta(\gamma\ +\ \mu)}{\beta} \\ \beta\left(\frac{\beta\mu K - (\mu\ +\ p)(\gamma\ +\ \mu)}{\beta(\gamma\ +\ \mu)}\right) & \beta\left(\frac{\gamma+\mu}{\beta}\right) - \gamma - \mu - \lambda \end{bmatrix}$$

The characteristics equation of $(J - \lambda I)$ is given by

$$\lambda^2 - \lambda\left(\frac{\beta\mu K}{\gamma\ +\ \mu}\right) - \beta\mu K + (\mu\ +\ p)(\gamma\ +\ \mu) = 0. \quad (19)$$

If the trace, $\lambda_1 + \lambda_2 < 0$ and its determinant,

$$\beta\left(\frac{\beta\mu K - (\mu\ +\ p)(\gamma\ +\ \mu)}{\beta(\gamma\ +\ \mu)}\right)\left(\frac{-\beta(\gamma+\mu)}{\beta}\right) > 0 \quad (20)$$

is positive, the endemic equilibrium is asymptotically stable. This conclusion is true since $R_0 > 1$ in the endemic equilibrium. On the other hand it becomes unstable when $R_0 < 1$.

We now compare equation (19) to the quadratic equation $\lambda^2 - p\lambda + q = 0$

$$\Rightarrow \text{p} = \frac{\beta\mu K}{\gamma\ +\ \mu} \quad \text{and} \quad q = -\beta\mu K + (\mu\ +\ p)(\gamma\ +\ \mu) = 0$$

If $\lambda_1 \; and \; \lambda_2$ are the eigenvalues of J then we have

$$p(\lambda) = (\lambda - \lambda_1)(\lambda - \lambda_2) = \lambda^2 - (\lambda_1 + \lambda_2)\lambda + \lambda_1\lambda_2$$

Thus we have the identities

$$\lambda_1 + \lambda_2 = tr(J) \text{ and } \lambda_1\lambda_2 = \det(J)$$

If p = $tr\,(J)$ and $q = \det(J)$ then

$$\lambda_1, \lambda_2 = \frac{p \pm \sqrt{p^2 - 4q}}{2},$$

The nature of the roots is determined by the discriminant

$$\Delta = p^2 - 4q$$

The parameters p, q and Δ allow us to determine the stability of the fixed points (origin) of the endemic equilibrium.

4 Sensitivity Analysis

We determine the nature of the model by conducting sensitivity analysis.

4.1 Sensitivity Analysis of the R_0 Of HB Without Vaccination.

(a) If $\gamma \; and \; \beta$ values are increased and S_0 remains the same that is $\gamma = 0.9942, \beta = 3$ and S_o = 0.3891, $R_0 = 1.17 > 1$

(b) If γ and β values are reduced to $\gamma = 0.9742 \; and \; \beta = 2$ but S_0 maintained. $R_o = 0.80 < 1$

4.2 Sensitivity Analysis Of The R_0 Of HB With Vaccination

(a) If γ and β are increased and all the other parameters maintained,

thus, $\gamma = 0.9942, \; \beta = 4, \mu = 0.0219, S_0 = 0.3891, \; Ro = 1.5 > 1$

(b) If $\gamma$and βvalues are reduced to $\gamma = 0.9742, \beta = 2$ and the rest of the parameters are maintained $Ro = 0.781 < 1$

4.3 Sensitivity Analysis Of The Disease Free Equilibrium

(a) If γ and β values are increased to $\gamma = 0.9942 \; and \; \beta = 2.5525$ and μ

is maintained $\lambda_1 = 0.1972\ and\ \lambda_2 = -0.0219$. The eigenvalues are distinct. It is saddle and the origin is unstable.

(b) When γ and β values are reduced to $\gamma = 0.9742\ and\ \beta = 2.5325$,

$(S^*, I^*) = (0.3933, 0)$. $\lambda_1 = -6.7651 \times 10^{-5} and\ \lambda_2 = -0.0220$

Since λ_1 and λ_2 are both negative it is a nodal sink and the solution of the equilibrium point $(S^*, I^*) = (0.3933, 0)$ is asymptotically stable.

4.4 Sensitivity Analysis of the Endemic Equilibrium State

(a) When γ, and β values are increased to γ=0.9942 and β= 4, $\lambda_2 = 0.1292$ and $\lambda_2 = -0.1733$ is a saddle point and is therefore unstable.

(b) When the values γ and β are reduced to $\gamma = 0.9742$ and $\beta = 2.5325$ are reduced and other parameters maintained and substituting in Jacobian matrix, $\lambda_1 = -0.0672$ and $\lambda_2 = -0.3215$. This is a nodal sink and is therefore a symbolically stable.

5 The Herd Immunity Threshold

The Herd Immunity Threshold (H1) is the percentage of the population that needs to be immune to control transmission of the disease. The equation given by Diekmann and Heesterbeek [10] for the Herd Immunity threshold is

$$H1 = \frac{R_0 - 1}{R_0} = 1 - \frac{1}{R_0} = 1 - \frac{1}{\frac{\beta S_0}{\mu + \gamma}}$$

$$H1 = \frac{\beta So - (\gamma + \mu)}{\beta So} \tag{22}$$

As the amount of vaccinations increase, the herd immunity threshold also increases. By decreasing the amount of susceptible people, the herd immunity threshold decreases.

6 Discussion of Results

We used standard SIR model to predict the spread of Hepatitis B in the Bosomtwe District of Ghana. We discussed the existence and stability of the disease free and disease endemic equilibria of the model and performed sensitivity analysis of the parameters. We also considered Herd immunity as the sole immunization strategy in the research.

Based on the data obtained from Bosomtwe District, we estimated the basic reproductive number of the HB without vaccination to be $R_0 = 1.006$ in the Bosomtwe District. This indicates that Hepatitis B is endemic in Bosomtwe District. Basic reproductive number (R_0) of Hepatitis B with vaccination in Bosomtwe District was estimated to be $R_0 = 0.9840$. The decrease in the value of the R_0 may be due to the introduction of vaccination in the District.

In the stability analysis of the equilibrium points, the disease free equilibrium point was unstable. This is so because, the transmission rate of Hepatitis B in Bosomtwe district is greater than the recovery rate. Also the eigenvalues of the disease free equilibrium point in the model are $\lambda_1 = 7.4450 \times 10^{-5}$ and $\lambda_2 = -0.0220$ which is a saddle point and therefore unstable. The endemic equilibrium point of the model was also found to be unstable. This is because the eigenvalues produced by the endemic equilibrium point in the model are $\lambda_1 = 4.0864 \times 10^{-3}$ and $\lambda_2 = -0.0261$. This is saddle and therefore unstable.

In the sensitivity analysis of basic reproductive number of the Hepatitis B without vaccination in the district it was found that, whenever the value of the transmission rate (β) is increased $R_0 > 1$. But whenever value of β is reduced, $R_0 < 1$.This implies that if the disease is not combated in Bosomtwe District an epidemic may emerge. But if immunization and vaccination programmes are intensified in the district the disease could be eliminated.

R_0 with vaccination is 1.5 which is greater than 1 and this is due to the increase in the transmission rate value but when the transmission rate value is reduced R_0 is reduced to 0.781 which is less than 1. This is so because whenever vaccination is increased, the transmission rate is reduced and the recovery rate in the population is increased. Also since people obtain permanent immunity after recovering from Hepatitis B, vaccination will further reduce the transmission rate of Hepatitis B in the district.

In the sensitivity analysis of both the disease free equilibrium and the endemic equilibrium, whenever the transmission rate value (β) is increased, their equilibrium points become unstable but when their transmission rate value are reduced, they both become asymptotically stable. This shows that whenever the hepatitis B disease is unchecked in Bosomtwe district an epidemic will result. According to the estimated herd immunity threshold in this thesis, 871 persons need to be vaccinated in Bosomtwe district in order to control the spread of HB disease.

This therefore implies that the vaccination program in the district has not reached its maximum limit yet. Although, the vaccination programs in Bosomtwe district has reduced the spread of HBV based on the R_0 value of hepatitis B without vaccination and that of R_0 with vaccination estimated in the district. This shows that the vaccination programs in the district is not enough to completely check the spread of HBV among the people of Bosomtwe district.

7 Conclusion

Our work shows that, the increasing use of hepatitis B vaccine could have a significant impact on the rate of HBV transmission and future HBV transmission and related deaths in Bosomtwe district of Ghana. Increasing the HBV vaccination coverage rate in the Bosomtwe district will further decrease the prevalence of HB in the district. But decreasing the vaccination coverage could result in increase in the rate of transmission of HB in Bosomtwe district which will affect the development of human resources in Ghana.

Though this model did not consider mass vaccination as one of the methods to prevent the prevalence of HB in Bosomtwe district in Ghana but concentrated on herd immunity, its result can be used as a tool to facilitate the introduction of HB vaccination and improve HB vaccination in Ghana as a whole.

References

[1] S.T. Goldstein, F. Zhou, S.C. Hadler, B. P. Bell, E. E. Mastand , H. S. Margolis, Mathematical model to estimate global hepatitis B disease burden and vaccination impact, *International Journal of Epidemiology,*(2005).*; 34(6):1329.*

[2] K. N. Lai, L. Li, T. Au, Tong, Lai, Membranous nephropathy related to hepatitis B virus in adults. *The New England Journal of Medicine,(*1991). (324):1457-1463.35

[3] F. E. Martinson, K. A. Weigle, I. K. Mushahwar, D. J. Weber, R. Royce, & S. M. Lemon, Seroepidemiological survey of hepatitis B and C virus infections in Ghanaian children. *J Med Virol* 48, (1996) 278–283.

[4] J. K. Acquaye, A profile of Ghana's expanded programme on immunization in Ghana, (1991), Pages 10-12.

http//www.who.int/countries/gha/publications/EPI_Profile.pdf.

[5] J. K. Acquaye, and J. A. A. Mingle, Hepatitis B viral markers in Ghanaian pregnant women. *West Afr J Med 13*, (1994). 134–137.

[6] F. E. Martinson, K. A. Weigle, R. A. Royce, D. J. Weber, C. M. Suchindran, & S. M. Lemon, Risk factors for horizontal transmission of hepatitis B virus in a rural district in Ghana. *Am J Epidemiol* 147, (1998) 478–487.

[7] A. B. Akosah, and H. Armah, *Ghana Medical Association Journal*.Vol.39 ,No. 4, (2005).

[8] W. O. Kermack, and A. G McKendrick, *A Contribution to the mathematical Theory of Epidemics*. Proc. Roy. Soc. Ser. Lond. A 115, (1927) 700-721.

[9] H. Trottier, & P. Philippe,"Deterministic modeling of infectious diseases: theory and methods." *The Internet Journal of Infectious Diseases.* (2001).

[10] O. Diekmann, J.A.P. Heesterbeek, Mathematical Epidemiology of Infectious Diseases.Chichester: Wiley; 2000. p. 303.

Received: April 11, 2014