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Evaluating the Stability of Hepatitis B Virus with Infectious Latent

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ABSTRACT: *Mathematical models* of *infectious disease transmission dynamics have proven extremely valuable. They form an important theoretical epidemiology method, which has been used to the prevalence of hepatitis B and evaluate different immunization strategies. However, the mathematical process of HBV transmission as presented by various scholars varies, not only in the model structure, but also in the estimation of certain parameters. In this work, one of such models namely SEIR mathematical model was modified for the transmission of HBV within Latent Human with the aim to evaluate its stability. The model was analyzed in term of the population and the Latent individuals. The equilibrium point obtained were analyzed and found to be locally asymptotically stable.*

KEYWORDS*: HBV Modelling, Local Stability, Basic Reproduction Number R_0, Epidemiology and Infectious Latent.*

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I. INTRODUCTION

Hepatitis B Virus (HBV) is a DNA virus and was first identified in the 1960s. According to the ICTV classification, this virus belong to the genus orthohepadna virus of the Hepednaviridae family and, along with the spunvaretrovirinae sub-family of the retroviridae family, represents the only other animal virus with a DNA genome known to replicate by the reverse transcription of a viral RNA intermediate (Mehmood 2011). The Hepatitis B Virus is a blood-borne virus and roughly 75 – 200 times more infectious than HIV (Mukandavire et al. 2010).

Hepatitis B is a serious liver disease, caused by hepatitis B virus. The disease is transmitted through human body fluids such as blood and serum. It is an alarming public health problem worldwide. It's method of transmission include through mother to baby (perinating), sexual contact and the use of improper injection technique. More than two billion amongst the population alive today, would have been infected at some point or other in their lives by the hepatitis B Virus (HBV) and approximating 350 million of them are the carriers of the chronically infected disease (Thorley et al. 2008). Out of these, 25-30% would die as a result of the infection (WHO, 2003). These carriers are at high risk of this serious illness and death from cirrhosis of liver and/or primary liver carrier would kill more than one million of them per year. They also constitute a reservoir of infected individual, who perpetuate the infection from generation to generation.

According to the international organization for migration (IOM), there are more than 200 million migrants worldwide (World Bank, 2017). The united nation found that, in 2005 migrants around the world amount to three per cent of the world population and 115million lived in develop countries, with an increase of 26 million since 1990. Migration is one of the defining issues of our time. For Example, Nigeria is an important destination country for immigrants in the west Africa region, the number of immigrants residing in Nigeria has doubled in recent decades, from 477,135 in 1991 to 971,450 in 2005 (NPC, 1991). However, immigrants make up only 0.7 per cent of the total population. The majority of immigrants in Nigeria are from neighbouring economic community of West Africa states (ECOWAS), and more than 74% of immigrants arrive in Nigeria each year (ECOWAS, 2006). Countries of the world are increasingly connected through trade, war, travel and migration, and thus, migration has health implications since infectious diseases such as HBV do not remain isolated geographically. The United Kingdom (UK) hepatitis foundation estimated in 2007 that the number of hepatitis B cases in UK doubled in the previous six years chiefly due to immigration of infected people, many from the new member states of the European Union where the prevalence of viral hepatitis is higher. The U.S. preventive service task force (USPSTF) recommends screening for those born in countries with prevalence of HBV infection 20% or greater (Moneim et al. 2009). Similarly, the united Arab Emirates mandated hepatitis C testing to be done at the time of residence, visa renewals and has added hepatitis B to the list of disease such as

HIV and hepatitis C as disease warranting depuration, adding that immigrants for short visits and student should be subjected to test to reduce the number of immigrants with disease (Muhammad, 2013). Similarly, health service have also been accused of stigmatizing immigrants, with a tendency to focus more on protecting the resident population than benefiting the health of new arrivals (Abdulrahman et al. 2013), immigrants health in many areas of Britain has become the responsibility of communicable disease departments, giving the misleading impression that immigrants are vectors of infection, with the inevitable effects of increased stigmatization. Many studies reveal the infectiveness of screening and quarantine for HBV and suggested that detection of disease on screening must not be used as a reason to deny entry to the EU, not only for ethical reasons but also because public health measures to prevent further spread of the disease HBV cannot otherwise be enacted. Denying entry to EU because of detected infection will not prevent, but rather propagate the spread of the disease because infected migrants might become inaccessible to the public health service but still remain in contact with susceptible population within the EU. Migrants need to be offered the same access to health care services as the rest of the population. They should be screened, counselled and recommended for medical evaluation and management in resettlement communities, while adhering to migrant's health rights. These right entail limiting discrimination or stigmatization, removing impediment to migrant's access to preventive and curative interventions.

The spread of the HBV in Nigeria has posed a lot of threat to health and wellbeing citizens in Nigeria. It is evident that about a third of the world population, approximately 2 billion people gets infected with Hepatitis B Virus in their life time. About 360 million people remain chronically infected carrier of the disease, more of whom are unaware of their HBV status and about 20-30% of whom will eventually die from chronic sequel. The prevalence of HBV infection varies from country to country, depending upon the complex behavior, environmental and host factor. Chronic HBV can lead to hepatocellular carcinoma after 20years among persons with chronic HBV infection. The risk of premature death from cirrhosis or hepatocellular carcinomas is 15 – 25%.

Hepatitis B is a disease that is characterized by inflammation of the liver and is caused by infection by the hepatitis B Virus. According to (WHO, 2002) stated that hepatitis may be caused by drugs or viral agent, these viral agent include the hepatitis A, B, C, D, E, F, G, and H viruses. More than a billion people around the world have serological indicator of past or present infection with HBV.

According to White and Fenner (1994) and Platkov et al (2001) in their research over 300 million people are chronic carriers of the virus. The fast spread of the virus shows that it is very communicable.

It is evident according to (WHO, 2009) that HBV infection can be transmitted from mother to child (vertical), contact with an infected person (horizontal transmission), Sexual contact (homosexual and heterosexual transmission) with infected partners, exposure of blood or infected fluids and with HBV contaminated instruments.

According to (Anderson and May, 1992) transmission mechanism in a population in a mathematical frame-work is capable of predicting the behavior of the disease spread through the population.

In 2010, the World Health Assembly adopted resolution 63.18 to recognize viral hepatitis as a global health problem. In response, the WHO developed a four-prong strategy aimed at raising awareness/mobilizing resource, policy, preventing transmission, screening and treatment. 180 countries included Hepatitis B vaccination as part of their routine vaccination schedule and the worldwide coverage approached 80% in 2011.

Nigeria has made notable gains along the four-prong strategy, for example by registering hepatitis-related cancer cases, creating national guidelines for prevention of infection in health care workers, adopting universal vaccination and screening all donated blood. However, national policies aimed at prevention of mother to child infections and eliminating of HBV are lacking; this is perhaps reflected in our finding that approximately 14% of Nigeria were exposed to HBV from 2000 – 2013. This estimate would place Nigeria as one most affected country in Africa and indeed the world. The prevalence peaked at 53.9% in 2003, although this estimate was weighted by only two studies.

It is well documented that many disease, such as tuberculosis, HIV/AIDS and SARS, etc. have a contagious latent period, a latent individual can transmit the disease to the susceptible. This means that there are two forces of infection during their latent period.

This fact has been noticed by some researcher. HBV has a long incubation period which varies from six weeks to six months. The latent person can pass the HBV infection during their latent period. So, we add another sort of transmission of the HBV. The second way come from the contact between the latent person and the susceptible.

Li and Jin (2005) consider a SEI epidemic model with general contact rate that incorporates constant recruitment and has infectious forces in both the latent and infected periods. In their work Lyapunov function and Lassalle's invariant set theorem have been used to prove global asymptotical stable result of the disease-free equilibrium. They studied the stability of the epidemic equilibrium by using the Poincare Bendizson property. Also Li and fang (2009) studied the age structured SEIR epidemic model with infectivity in both the latent and infectious periods. By using the theory and method of differential and integral equations, they obtained the local

and global stability if $R_0<1$. In this case, the disease always dies out. When $R_0>1$ there exist a unique epidemic equilibrium which is a asymptotically stable under some certain condition.

Recently, mathematical modeling have been used to study the transmission dynamics of HBV in various communities, regions and countries. Anderson and May (1991) used a simple deterministic, compartmental mathematical model to study the effect of carriers on transmission of HBV. Anderson et al (1992) and Wiah et al (2011) presented models of sexual transmission of HBV, which include heterogeneous mixing with respect to age and sexual activity. Edmunds et al (1996) investigated the relation between the age at infection with HBV and the development of carriers state. Medley et al (2001) proposed a model to show that the prevalence of infection is largely determined by a feedback mechanism that relates the rate of transmission, average age at infection and age related probability of developing carriage following infection. Thorley et al (2008) applied the model of Medley et al (2001) to predict chronic hepatitis chronic hepatitis B infection in New Zealand. The prevalence of HBV in developing countries is different from that in developed countries, since it appears that the rate of transmission in childhood is the major determinant of the level of HBV endermicity and little is known on the rate patterns of sexual contact in developing countries. Edmund et al, 1996a studied models of HBV transmission in developing countries and Wiah et al (2011) describe a model of HBV in United Kingdom, Zou et al (2009) proposed a mathematical model to investigate the transmission dynamics of prevalence of HBV in mainland china.

The model of Zou et al (2009) form the motivation of this study. In their work, a mathematical model was proposed to study the transmission dynamics and prevalence of HBV infection in mainland China.

They assumed that the newborns of carrier mothers infected at birth do not stay in the latent period, so they instantaneously become carriers. However, as pointed out by Anderson and May (1991) and White and Fenner (1994), an HBV carriers must have harbored the virus in the blood for at least six month. By this newborns of carrier mothers infected at birth are latently infected individual. Saher et al. (2012) supported the same view in his study and assumed that the proportion of the infected newborns to carrier mothers is latent. The role of treatment of HBV carriers as a measure of control was not considered in their model.

II. NOTATIONS

DNA	:	Deoxyribonucleic Acid.
RNA	:	Ribonucleic Acid
HIV	:	Human Immunodeficiency Virus
UNDP	:	United Nations Development Programme
WHO	:	World Health Organisation
NPC	:	National Population Commission

III. METHOD

The modified Hepatitis B realistic SEIR Model will be derived from the existing model of Li, G. and Ji, Z. (2005). The properties of the modified model and it's method necessary for analysis of the model are presented.

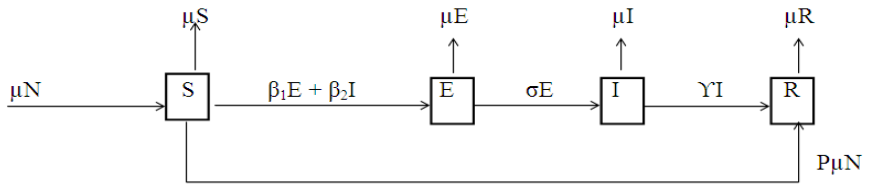
Li and Jin (2005) presented a SEIR model of Hepatitis B Virus which states the assumption, parameter and equation of the model. These models which take account of the infectiousness of the disease in the latency state here the effect of the infectivity of the latent population in addition to the transmission between the susceptive and infective population.

The model makes the following assumptions:

1. The total population size is a constant N, and the population is divided into four groups
a. The susceptive class, S, comprising those individual who are capable of catching the disease.
b. The expose (or latent) class, E, comprising those individual who are infected but not yet infectious.
c. The infective, I, comprising those who are infected and capable of transmitting the virus.
d. The recovered class R, comprising those individual who are immune.
2. The per capita birth rate is a constant N. as birth balance death we must have that the per capita death rate is also μ.
3. The population is uniform and mixed homogeneously
4. The infections rates β_1 and β_2 are defined as the total rate at which potentially infections contact occur between two individual (in other word contact which will result in the transmission of infection if one of the individual is susceptive and the other is infective for β_1 and latent for β_2).
5. The expose individual move from latent class to the infective class at a constant rate σ and so the average latent period, conditional on survival to the end of it, is $1/\sigma$
6. The infective moves from the infective class to the recovered class at a constant rate Υ, where $1/\Upsilon$ is the average infections period, conditional on survival to the end of it.

Parameters of the existing model
μ = as birth rate
σ = Rate of moving from latent class to infective class
ϒ = Rate of moving from infective class t recovered class
N = Size of the population
P = Proportion of vaccination
β_1 = Transmission of infection from susceptible to infective
β_2 = for latent rate

Flow diagram for the existing model is show below:



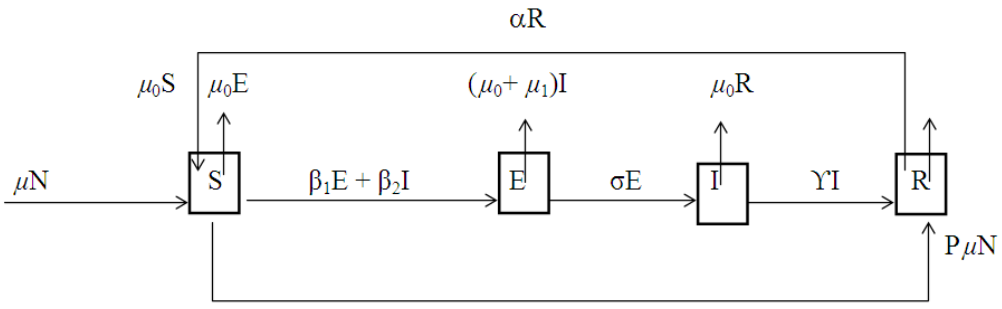
The Existing Model Equation
From the assumptions and parameters stated above, the model equations were derived.

$$\frac{ds}{dt} = \mu(I - P)N - \mu S - \beta_1 SE - B_2 SI \ldots\ldots 3.1$$
$$\frac{dE}{dt} = \beta_1 SE - \beta_2 SI - (N + \delta)E \ldots\ldots\ldots\ldots 3.2$$
$$\frac{dI}{dt} = \sigma E - (\sigma + \Upsilon)I \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 3.3$$
$$\frac{dR}{dt} = P\mu N + \Upsilon I - \mu R \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 3.4$$

The Modified Model
The model takes account of the infectiousness of the disease in the latency state, the effect of the infectivity of the latent population in addition to the transmission between the susceptible class and the recovered class.

The modified model state the following assumptions
1. The recovered individual confer temporally immunity
2. The recovered individual loss immunity and move to susceptible class at a constant rate α

The flow diagram for the existing model is now modified to obtain the flow diagram for the modified model.



Flow diagram for the modified model

Parameter for the modified model
α = As rate of loss of immunity
μ = As natural birth rate
μ_0 = As natural death rate
μ_I = As death rate due to infection

The Modified Model Equation

The modified equation were derived from the existing equation but some parameter were still added to obtain the equation stated below.

$\frac{dS}{dt} = (1-P)\mu N + \alpha R\,(\beta_1 E + \beta_2 I)\,S - \mu_0 S$ 3.11

$\frac{dE}{dt} = (\beta_1 E + B_2 I)S - (\mu_0 + \sigma)E$ 3.12

$\frac{dI}{dt} = \sigma E - (\mu_0 + \mu_1 + \Upsilon)I$ 3.13

$\frac{dR}{dt} = \Upsilon I + P\mu N - (\alpha + \mu_0)R$ 3.14

With S + E + I + R = N and R can always be found as R(t) = I – [s(t)+ E(t) + I(t)]. The non-negative initial condition for the modified model are S(0) > 0, E(0)≥0, I (0) ≥ 0, and all the parameter of the modified model are assumed to be non-negative as well.

DISEASE FREE EQUILIBRIUM STATE

To find the equilibrium state of the model equation, we set the left hand side of equation (3.11) – (3.14) to zero that is $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = 0$ which is

$0 = (1 - P)\,\mu N + \alpha R - (\beta_1 E + \beta_2 I)\,S - \mu_0 S - - - -4.1$

$0 = (\beta_1 E + \beta_2 I)\,S - (\mu_0 + \sigma)E - - - - - - - - - 4.2$

$0 = \sigma E - ((\mu_0 + \mu_1 + \Upsilon)\,I - - - - - - - - - - - - - 4.3$

$0 = \Upsilon I + P\mu N - (\alpha + \mu_0)\,R - - - - - - - - - - - - - -4.4$

Hence at the disease free equilibrium state we have absence of infection which implies there is no infection in the infected class E and I = 0

Furthermore equation (4.2) and (4.3) are zero from (4.4) we have

$R = \frac{\Upsilon I + P\mu N}{(\alpha + \mu_0)}$ that is, at disease free equilibrium state, there is no infection, impling

$R = \frac{P\mu N}{\alpha + \mu_0} - - - - - -4.5$

(4.2) and (4.3) are all zero, since I = E = 0.

Also from (4.1) with substitution for I = E = 0 and (4.5)

$0 = ((1 - P)\,\mu N + \alpha\,(\frac{P\mu N}{\alpha + \mu_0}) - \mu_0 S$

Collecting of likes terms of yield

$\mu_0 S = (1 - P)\,\mu N + \frac{\alpha P\mu N}{\alpha + \mu_0}$

$\mu_0 S = \frac{(\alpha + \mu_0)(1 - p)\mu N + \alpha P\mu N}{\alpha + \mu_0}$

Dividing through by μ_0 yield

$S_0 = \frac{(\alpha + \mu_0)(1 - p)\mu N + \alpha P\mu N}{\mu_0(\alpha + \mu_0)} - - - - - - - 4.6$

Therefore at the disease free equilibrium state of the model (3.1) – (3.13) is

$E_0 = (S_0, 0, 0, R_0) - - - - - - - -4.7$

Where

$S_0 = \frac{(\alpha + \mu_0)(1 - p)\mu N + \alpha P\mu N}{\mu_0(\alpha + \mu_0)}$

THE BASIC REPRODUCTION NUMBER, R_0

The basic reproduction number is defined as the average value of the expected number of the secondary cases produced by a single newly infected individual entering the population at the disease Free State. In this model the average value of the expected number of secondary cases produced by a single infected person. It has been shown that with constant rates β_1 and β_2 our model has one disease free equilibrium simulated result have been conducted for parameter values which insure that if the vaccination parameter P is not large enough to force that basic reproduction number R_0 to be less than one in value, the disease remain endemic in the population. The basic reproduction number is calculated as: firstly we bring out the number equations (3.12) to (3.13) in ascending order of the infected class.

$\frac{dE}{dt} = (\beta_1 E + \beta I)\, S - (\mu_0 + \sigma)E - - - - - - - - - - 4.8$

$\frac{dI}{dt} = \sigma E - (\mu_0 + \mu_1 + \Upsilon)\, I - - - - - - - - - - - - - 4.9$

$\frac{dI}{dt} = (1 - P)\,\mu N + \alpha R - (\beta_1 E + \beta_2 I)\, S - \mu_0 S - - - - 4.10$

From equation (4.8) to (4.9) we have the infective class which transfer from one class to another given as

$$W = \begin{bmatrix} (\beta_1 E + \beta_2 I)S \\ 0 \end{bmatrix} - - - - - - - - 4.11$$

The remaining transfer term in equation (4.8) and (4.9) gives

$$Q = \begin{bmatrix} (\sigma + \mu_0)E \\ (\mu_0 + \mu_1 + \Upsilon)I - \sigma E \end{bmatrix} - - - - - - - - 4.12$$

Hence we take the partial derivatives of (4.1) with respect to E and I at the disease free equilibrium

$$G = \Delta W = \begin{bmatrix} \beta_1 S_0 & \beta_2 S_0 \\ 0 & 0 \end{bmatrix} - - - - - - - - 4.13$$

The matrix of the partial derivative of (4.2) at the disease free equilibrium $E_0 = (S_0, 0, 0, R_0)$

$$U = \Delta Q_{(E_0)} = \begin{bmatrix} (\sigma + \mu_0) & 0 \\ -\sigma & (\mu_0 + \mu_1 + \Upsilon) \end{bmatrix} - - - - - - - - 4.14$$

We now find the inverse of U, but first we find the determinant and then the transpose of the adjoint det (U) = $(\sigma + \mu_0)(\mu_0 + \mu_1 + \Upsilon)$

Hence the transpose is given by

$$\begin{bmatrix} (\mu_0 + \mu_1 + \Upsilon) & 0 \\ -(\sigma) & \mu_0 + \sigma \end{bmatrix}$$

Therefore

$$U^{-1} = \frac{1}{\det(U)} \begin{bmatrix} (\mu_0 + \mu_1 + \Upsilon) & 0 \\ \sigma & \mu_0 + \sigma \end{bmatrix}$$

$$U^{-1} = \frac{1}{(\mu_0 + \sigma)(\mu_0 + \mu_1 + \Upsilon)} \begin{bmatrix} (\mu_0 + \mu_1 + \Upsilon) & 0 \\ \sigma & \mu_0 + \sigma \end{bmatrix}$$

$$U^{-1} = \begin{bmatrix} \frac{(\mu_0 + \mu_1 + \Upsilon)}{(\mu_0 + \sigma)(\mu_0 + \mu_1 + \Upsilon)} & 0 \\ \frac{\sigma}{(\mu_0 + \sigma)(\mu_0 + \mu_1 + \Upsilon)} & \frac{(\mu_0 + \sigma)}{(\mu_0 + \mu_1 + \Upsilon)} \end{bmatrix}$$

$$U^{-1} = \begin{bmatrix} \frac{1}{\mu_0 + \sigma} & 0 \\ \frac{\sigma}{(\mu_0 + \sigma)(\mu_0 + \mu_1 + \Upsilon)} & \frac{1}{(\mu_0 + \mu_1 + \Upsilon)} \end{bmatrix}$$

$$G.U^{-1} = \begin{bmatrix} \beta_1 S_0 & \beta_2 S_0 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\mu_0 + \sigma} & 0 \\ \frac{\sigma}{(\mu_0 + \sigma)(\mu_0 + \mu_1 + \Upsilon)} & \frac{1}{(\mu_0 + \mu_1 + \Upsilon)} \end{bmatrix}$$

$$\mathrm{G.U}^{-1}\begin{bmatrix}\dfrac{\beta_1 S_0}{\sigma+\mu_0}+\dfrac{\beta_2 S_0\sigma}{(\sigma+\mu_0)(\mu_0+\mu_1+\Upsilon)} & \dfrac{\beta_2 S_0}{(\mu_0+\mu_1+\Upsilon)}\\ 0 & 0\end{bmatrix}$$

$$[\mathrm{GU}^{-1}-\lambda I]=\begin{bmatrix}\dfrac{\beta_1 S_0(\mu_0+\mu_1+\Upsilon)+\beta_1\sigma S_0}{(\sigma+\mu_0)(\mu_0+\mu_1+\Upsilon)}-\lambda & \dfrac{\beta_2 S_0}{(\mu_0+\mu_1+\Upsilon)}\\ 0 & 0-\lambda\end{bmatrix}=0$$

$$[\mathrm{GU}^{-1}-\lambda I]=-\lambda\left[\frac{\beta_1 S_0(\mu_0+\mu_1+\Upsilon)+\beta_1\sigma S_0}{(\sigma+\mu_0)(\mu_0+\mu_1+\Upsilon)}-\lambda\right]=0$$

$$=\lambda^2-\left[\frac{\beta_1 S_0(\mu_0+\mu_1+\Upsilon)+\beta_1\sigma S_0}{(\sigma+\mu_0)(\mu_0+\mu_1+\Upsilon)}\right]\lambda=0$$

$$=\lambda\left[\lambda-\frac{\beta_1 S_0(\mu_0+\mu_1+\Upsilon)+\beta_1\sigma S_0}{(\sigma+\mu_0)(\mu_0+\mu_1+\Upsilon)}\right]=0$$

$$=\lambda_1=\frac{\beta_1 S_0(\mu_0+\mu_1+\Upsilon)+\beta_1\sigma S_0}{(\sigma+\mu_0)(\mu_0+\mu_1+\Upsilon)},\lambda_2=0$$

But R_0 is the leading eigenvalues
This implies $R_0=\lambda_1$, therefore

$$R_0=S_0\,\frac{\beta_1(\mu_0+\mu_1+\Upsilon)+\beta_2\sigma}{(\sigma+\mu_0)(\mu_0+\mu_1+\Upsilon)}---------4.15$$

Substituting equation (4.6) into (4.15)

$$R_0=\frac{\beta_1 S[\mu_0+\mu_1+\Upsilon][(\sigma+\mu_0)(1-P)\mu N+P\mu N]}{(\sigma+\mu_0)(\mu_0+\mu_1+\Upsilon)[(\mu_0(\alpha+\mu_1)]}+\frac{\beta_2\sigma S_0}{(\sigma+\mu_0)(\mu_0+\mu_1+\Upsilon)}$$

$$R_0=P(G-U^{-1})=\frac{\beta_1 S_0(\mu_0+\mu_1+\Upsilon)+\beta_2 S_0\sigma}{(\sigma+\mu_0)(\mu_0+\mu_1+\Upsilon)}$$

LOCAL STABILITY OF THE DISEASE FREE EQUILUBRUM

This section studies the stability of the disease free equilibrium state. In this case there is no disease in the population as E = T = 0. We will use the method linearization to obtain the Jacobian matrix of the system. Using equation (3.11) – (3.14)

$$f_1=(1-P)\mu N+\alpha R-(\beta_1 E+\beta_2 I)S-\mu_0 S$$
$$f_2=(\beta_1 E+\beta_2 I)S-(\mu_0+\sigma)E$$
$$f_3=\sigma E-(\mu_0+\mu_1+\Upsilon)I$$
$$f_4=\Upsilon I+P\mu N-(\alpha+\mu_0)R$$

At these state $E_0(S_0,0,0,R_0)$e population, the disease die out completely from the population, then one partial derivatives for f_1, f_2, f_3, f_4 is given by.

$$\frac{\partial f_1}{\partial S}=-\mu_0,\quad \frac{\partial f_1}{\partial E}=-\beta_1 S_0,\quad \frac{\partial f_1}{\partial I}=-\beta_2 S_0,\ \frac{\partial f_1}{\partial R}=\alpha$$
$$\frac{\partial f_2}{\partial S}=0\ \frac{\partial f_2}{\partial E}=\beta_2 S_0-(\mu_0+\sigma)\quad \frac{\partial f_2}{\partial I}=\beta_2 S_0\quad \frac{\partial f_2}{\partial R}=0$$

$$\frac{\partial f_3}{\partial S}=0\ \frac{\partial f_3}{\partial E}=\sigma\quad \frac{\partial f_3}{\partial I}=-(\mu_0+\mu_1+\Upsilon)\ \frac{\partial f_3}{\partial R}=0$$
$$\frac{\partial 4}{\partial S}=0\quad \frac{\partial f_4}{\partial E}=0\quad \frac{\partial f_4}{\partial I}=\Upsilon\ \frac{\partial f_4}{\partial R}=-(\alpha+\mu_0)$$

Hence the Jacobian matrix JE for the disease Free State is given below

$$JE_0 = \begin{bmatrix} -\mu_0 & -\beta_1 S_0 & -\beta_2 S_0 & \alpha \\ 0 & \beta_1 S_0 - (\mu_0 + \sigma) & \beta_2 S_0 & 0 \\ 0 & \sigma(\mu_0 + \mu_1 + \Upsilon) & 0 & \\ 0 & 0 & \Upsilon & -(\alpha + \mu_0) \end{bmatrix} - - - - - - - - -4.16$$

Theorem: the disease Free State $E_0 = (S_0, 0,0, R_0)$ is locally asymptotically stable for the system of equation (3.11) – (3.14) if $R_0 < 1$ and unstable when $R_0 > 1$

PROOF

From the characteristic equation of the matrix we have two negative eigenvalues, if $R_0 < 1$, then $\beta_1 S_0 < \mu_0 + \sigma$ so we found that all the eigenvalues of the matrix have negative real part.

Therefore using Routh – Hurwitz condition for the stability of linear differential equation, the disease force state is locally asymptotically stable for $R_0 < 1$. If $R_0 < 1$ it implies that $R_0 - 1 > 1$. Thus the characteristic equation for this matrix must have a positive eigenvalues. Therefore the disease free state is unstable for the system of equation (3.11) – (3.14). Hence these complete the proof.

The eigenvalues are obtain from the corresponding characteristics equation $|JE - \lambda I| = 0$ in equation as follows

$$|JE - \lambda I| = \begin{bmatrix} -\mu_0 - \lambda & -\beta_1 S_0 & -\beta_2 S_0 & \alpha \\ 0 & \beta_1 S_0 - (\mu_0 + \sigma) - \lambda & \beta_2 S_0 & 0 \\ 0 & \sigma(\mu_0 + \mu_1 + \Upsilon) - \lambda & 0 & \\ 0 & 0 & \Upsilon & -(\alpha + \mu_0) - \lambda \end{bmatrix} = 0$$

$\lambda_1 = -\mu_0$ Or

$$-(\alpha + \mu_0) - \lambda \begin{bmatrix} \beta_1 S_0 - (\mu_0 + \sigma) - \lambda & \beta_2 S_0 \\ \sigma & -(\mu_0 + \mu_1 + \Upsilon) - \lambda \end{bmatrix} = 0$$

$\lambda_2 = -(\alpha + \mu_0)$ Or

Let $Q = \begin{bmatrix} \beta_1 S_0 - (\mu_0 + \sigma) - \lambda & \beta_2 S_0 \\ \sigma & -(\mu_0 + \mu_1 + \Upsilon) - \lambda \end{bmatrix} = 0$ $- - - - - - - - 4.17$

$$\lambda_2 + (2\mu_0 + \mu_1 + \Upsilon - \beta_1 S_0 + \sigma)\lambda - \sigma\beta_2 S_0 - \mu_0\beta_1 S_0 + \mu_0^2 + \mu_0\sigma - \mu_1\beta_1 S_0 + \mu_1\mu_0 + \mu_1\sigma - \Upsilon\beta_1 S_0 + \Upsilon\mu_0 + \Upsilon\sigma = 0$$

$$\lambda_2 + [(\mu_0 + \mu_1 + \Upsilon) + (\mu_0 + \sigma)\beta_1 S_0)]\lambda - (\mu_0 + \mu_1 + \Upsilon)\beta_1 S_0 + \mu_0(\mu_0 + \mu_1 + \Upsilon) + (\mu_0 + \mu_1 + \Upsilon)\sigma - \sigma\beta_2 S_0 = 0$$

Recall that

$\lambda^2 + A\lambda + B = 0$ ---------- 4.18

Where $A = \{(\mu_0 + \mu_1 + \Upsilon) + (\mu_0 + \delta) - \beta_1 S_0\}$

$B = \{-(\mu_0 + \mu_1 + \Upsilon)\beta_1 S_0 + \mu_0(\mu_0 + \mu_1 + \Upsilon) + (\mu_0 + \mu_1 + \Upsilon)\delta - \beta_1 S_0\sigma\}$

But our reproduction number R_o yield

$R_0^C = R_1 + R_2$ Which implies

$$R_0^C = \frac{\beta_1 S_0}{(\sigma + \mu_0)} + \frac{\beta_2 S_0 \sigma}{(\sigma + \mu_0)(\mu_0 + \mu_1 + \Upsilon)} - - - - - - - - - - -4.19$$

Note that $1 - \frac{\beta_1 S_0}{(\sigma + \mu_0)} > 0$ which implies that

$(1 - R1) > 0$ if and only if $R_1 < 0$ if suffice to show that A, B > 0

To show that A > 0. It therefore implies that

$$(\mu_0 + \mu_1 + \Upsilon) + (\mu_0 + \sigma)\beta_1 S_0 > 0$$

$$(\mu_0 + \mu_1 + \Upsilon) + (\mu_0 + \sigma)\left[1 - \frac{\beta_1 S_0}{\mu_0 + \sigma}\right] > 0$$

$(\mu_0 + \mu_1 + \Upsilon) + (\mu_0 + \sigma)(1 - R_1) > 0, iff\ R_1 < 0$

This implies that A is > o

To show that B > 0, implies that

$-(\mu_0 + \mu_1 + \Upsilon)\beta_1 S_0 + \mu_0(\mu_0 + \mu_1 + \Upsilon) + (\mu_0 + \mu_1 + \Upsilon)\sigma - \beta_2 S_0\sigma$

Rearranging the above equation we have

$$(\mu_0 + \mu_1 + \Upsilon)(\mu_0 + \sigma) - \sigma\beta_2 S_0 - \beta_1 S_0(\mu_0 + \mu_1 + \Upsilon)$$

$$(\mu_0 + \mu_1 + \Upsilon)(\mu_0 + \sigma)\,1 - \left[\left(\frac{\sigma\beta_2 S_0 + \beta_1 S_0(\mu_0 + \mu_1 + \Upsilon)}{(\mu_0 + \mu_1 + \Upsilon)(\mu_0 + \sigma)}\right)\right] > 0$$

$$(\mu_0 + \mu_1 + \Upsilon)(\mu_0 + \sigma)\left[1 - {R_0}^C\right] > 0 \; if \; {R_0}^C < 1$$

Hence the solution of the equation is asymptotically stable. It therefore implies that the disease will die out if ${R_0}^C < 1$ and endemic (unstable) if ${R_0}^C > 1$.

IV. DISCUSSION

The Li and Jin (2005) SEIR mathematical model was modified for the transmission of HBV within Latent Human. The model was analyzed in term of the population and the Latent individuals. The equilibrium point obtained were analyzed and found to be locally asymptotically stable. It was also seen that if the vaccination proportion P is not sufficient and R_0 above one in value the disease dies out. We also consider the local stability of the model where it was seen that R_0 is less than one, which implies that it is locally asymptotically stable. It was observed that gradually increasing vaccination rate to the population reduces the number of susceptible human against possible re-infection, thus in the long run, the number of infection human population decreases to its minimal level. With a proper vaccination and a concerted effort aimed at prevention, HBV can be eradicated or eliminated.

V. CONCLUSION

This study modified a model of HBV by Li and Jin (2005) by moving the recovered individual to join the partially immune class parameter. Analytically, study was carried out on the model using the method of linearized stability and the result show that the disease free-equilibrium point are locally asymptotically stable for the model.

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