MATHEMATICAL MODELING OF HEPATITIS B IN KADUNA METROPOLIS USING EULER METHOD

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Abstract
Hepatitis B is a serious global health threat, it is a liver infection disease caused by hepatitisBvirus (HBV), which weakens the immune system of the victim. Its mode of transmission is through sexual contact, mother to child at birth, contact with the virus fluid, infected blood. Mathematical Modeling has emerged as a vital tool for understanding the dynamics of the spread of many infectious diseases. In this work,the SIR model of HBVwas used to predict the outbreak of the diseases. Euler method, which is among the suitable techniques for solving initial value problem (IVP) for ordinary differential equations (ODE), was applied to solve the 3 basic equations of the SIR model. Stability condition of each equilibrium point was discussed, the basic reproductive number (R_0) of HBV without vaccination and the basic reproductive number(R_0) of HBV with vaccination were estimated. As the global dynamics were completely determined by the basic reproductive number (R_0) ratio, it was computed as 1.3213, which indicates that the population is in danger should there be an outbreak. Therefore, there is need to reduce the reproductive ratio to less than one; thus, vaccination of the more susceptible populace is imperative, which will guaranty immunity to the individual. The results obtained shows that the herd immunity is 0.2431, which implies that if 24.31% of the population could be immunized, the transmission rate of the disease would be greatly reduced, thereby enabling a control mechanism over the spread of the disease.

Keywords: Euler's method, Herd immunity, Reproductive number, Stability, SIR model, Vaccination

Introduction
Hepatitis B is a serious global health threat, a liver infection disease caused by Hepatitis B Virus (HBV), which weakens the immune system of the victim. Its mode of transmission is through sexual contact, mother to child at birth, contact with the virus fluid or infected blood. In the African Region, hepatitis B is endemic which affects over 100 million people, mainly in West and Central Africa.The disease is so devastating as a killer disease that more than 20 million (≈ 11.2%) of Nigerians are affected with about 5 million (≈ 3%) on death annually. Statistics had shown that it contributes to high percentage of death worldwide (WHO, 2016). Viral hepatitis is also becoming more and more a growing cause of mortality among people living with HIV. About 2.6 million people living with HIV are co-infected with the hepatitis B virus and about 1% of persons living with HBV infection (2.7 million people) are also infected with HIV. Conversely, the global prevalence of HBV infection in HIV-infected persons is 7.4%. Since 2015, WHO has recommended treatment for everyone diagnosed with HIV infection, regardless of the stage of disease. Tenofovir, which is included in the treatment combinations recommended in first intention against HIV infection, is also active against HBV(ibid). However, an effective vaccine is available for preventing viral hepatitis B, also an effective treatment is available for people with chronic hepatitis B infection, although for most people such treatment needs to be lifelong. In Nigeria there is a high burden of viral hepatitis B at a prevalence rate of 11.2% (ibid), the country has a high hepatitis B virus (HBV) vaccination coverage among children, although birth-dose coverage is sub-optimal. Screening and vaccination coverage among adults remain unsatisfactorily low

due to a lack of awareness among the general populace and health workers, low coverage of testing facilities, high cost of laboratory investigations and medications for those needing treatment. In May 2016, The World Health Assembly adopted the first "Global Health Sector Strategy on Viral Hepatitis, 2016-2020". The strategy highlights the critical role of Universal Health Coverage and the targets of the strategy are in line with those of the Sustainable Development Goals. The strategy has a vision of eliminating viral hepatitis as a public health problem and this is encapsulated in the global targets of reducing new viral hepatitis infections by 90% and reducing deaths due to viral hepatitis by 65% by 2030. WHO also organizes World Hepatitis Day on July 28 every year to increase awareness and understanding of hepatitis virus.

The main aim of this research work is to model and predict the spread and offer suggestions on the control of Hepatitis B infection in Kaduna Metropolis. This will be done by using Susceptible-Infected-Recovered (SIR) model with numerical solutions based on Euler's method. To show how the proportion of susceptible, infected, and recovered changes with time, determination of the nature of the Hepatitis B disease outbreak, determination of the effect of infected on the populationand an estimation of the proportion of the population that should be vaccinated. This paper only investigates the outbreaks of hepatitis B virus narrowing from several infectious diseases by applying the SIR mathematical model. The model does not consider differences in age, gender and non-constant population.

Related Work

Bratislava (2007) in the mathematics of infectious diseases aimed at providing an understanding of deterministic modeling applied to the population dynamics of infectious diseases. Zou *et al*. (2010) carried out modeling on the transmission dynamics and control of hepatitis B virus in China. Zou *et al* (2010), proposed a mathematical model to understand the transmission dynamics and prevalence of hepatitis B (HBV) infection in China. Based on the data reported by the Ministry of Health of China, the model provides an approximate estimate of the basic reproduction number Ro= 2.406 which indicates that hepatitis B is endemic in China and approaching its equilibrium with immunization program and control measures. The authors considered six epidemiological groups while Medley *et al.* (2001) considered only five which did not distinguish the recovered and vaccinated subgroups and those with two groups with latently infected (L), acute infections (I), carriers (C), and protective immunity (R). Letsa-Agbozo (2014) developed mathematical model of hepatitis Binfectious disease based on the Susceptible-Infected-Recovered (SIR) using the North Tongu district of the Volta region of Ghana as case study. The population size of the district was assumed to be constant. A system of non-linear differential equations was used to model the spread of the disease in the district. They solved the system numerically using the forth-order Runge-Kutta method. Simulation and sensitivity analyses were also performed on the model equations to determine the effect of different parameter values on the spread of the disease. It was shown that the global dynamics were completely determined by the basic reproductive number Ro. If Ro< 1, the disease-free equilibrium is globally stable and the disease always die out. On the other hand, if Ro> 1, an endemic equilibrium exists and globally stable in the interior of the feasible region, and the disease persists at an endemic equilibrium state if it initially exists. In the absence of vaccination, the susceptible population will reduce sharply when an Infective is introduced into the population. Rodriguez (2016) in a mathematical model of Hepatitis B virus dynamics during Antiviral therapy discussed antiviral therapy for patients infected with Hepatitis B virus is only partially efficient and also in understanding the connections between the virus, immune responses, short-term and long-term drug efficacy and the health of the liver. The viral pattern ranges from biphasic, triphasic, flat phase, to virus rebound. The research also analyzed the model of HBV to determine the biological markers that determines decay

patterns. An investigation on such markers affects length of therapy and the amount of liver damage. The stated vaccination for newborn children is to receive a dose within 24hours after birth and two boosters during their childhood. It is also recommended to vaccinate those who require transplantation/dialysis, healthcare workers, and travelers before visiting an endemic area, people in prison or person with multiple sexual partners. For chronic hepatitis B the approved drugs are interferon (IFN) and nucleos (t)ide. The model used was introduced by Perelson for the study of HIV infection and first used in HBV by Nowak et al. It considers three (3) compartments: target uninfected cells (T), infected cells or hepatocytes (I) and hepatitis B virus (V) in contrast to the Ciupe *et al.* (2007) five (5) compartments of: Targeted cells (T), Infected cells (I), free virus (V), immune effector cells (E) and refractory cells (R). The results provided a framework for the virological and immunological factors involved a successful drug therapy.

Materials and Method

The Kermack and Mckendrick (1927) classic epidemic theory was used to model the spread of Hepatitis B, basic reproductive number and herd immunity threshold were the key focus. The work equally used the Euler method for the numerical solutions.

Simple SIR Model of Hepatitis B

The population isdivided into three disjoint classes of individuals: the susceptible class (S), the infective class (I), the removed class (R). The susceptible class consists of individuals who are not infective, but are prone to getting the disease and becoming infective. The infective class consists of individuals who are capable of transmitting the Hepatitis B disease to others. The removed class consists of individuals who have had the disease and are dead, or have recovered and are permanently immune, or are isolated until recovery.

Below are the assumptions of the SIR model and diagrammatic representation of the model as in Fig. 1:

1. Hepatitis B confers permanent immunity upon any individual who has completely recovered from it.
2. The members of the population mix homogeneously
3. It has a negligible short incubation period.
4. The population remains at a fixed level N in the time interval under consideration; so neglect births and deaths from causes unrelated to the disease under consideration, as well as immigration and emigration.

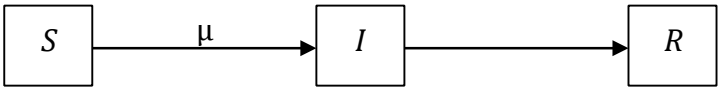

Figure 1: Flowchart of the SIR model of Hepatitis B without vital dynamics

Where the proportionality constants μ and α are the infection and removal rates respectively, based on the assumptions, the following model equations are obtained:

$$\frac{dS}{d} = \mu SI \quad (1)$$

$$\frac{dI}{d} = \mu SI \quad I \quad (2)$$

$$\frac{d}{d} = \quad I \quad (3)$$

With initial conditions S(0) = S_0, $I(0) = I_0 > 0$, $R(0) = 0$. Since $S(t) + I(t) + R(t) = 1$, calculate R from $R(t) = 1 - S(t) - I(t)$. The term $-\mu SI$ in equation (1) describes a transition

of infection due to the interaction between susceptible and infectives. The term – αI in equation (2) describes the recovery from the infection.

Observe from (2) that $\frac{dI}{dt} = 0$ when $I = 0$ or $S = \frac{\alpha}{\mu}$.When$S < \frac{\alpha}{\mu}$, and $\frac{dI}{dt} < 0$ thenI(t)decreases, and the disease dies out. On the other hand, when S>–and$\frac{dI}{dt} > 0$, then I(t) increases and an epidemic occurs i.e. an increase in infective individuals. These phenomena are illustrated in Fig.2 below.

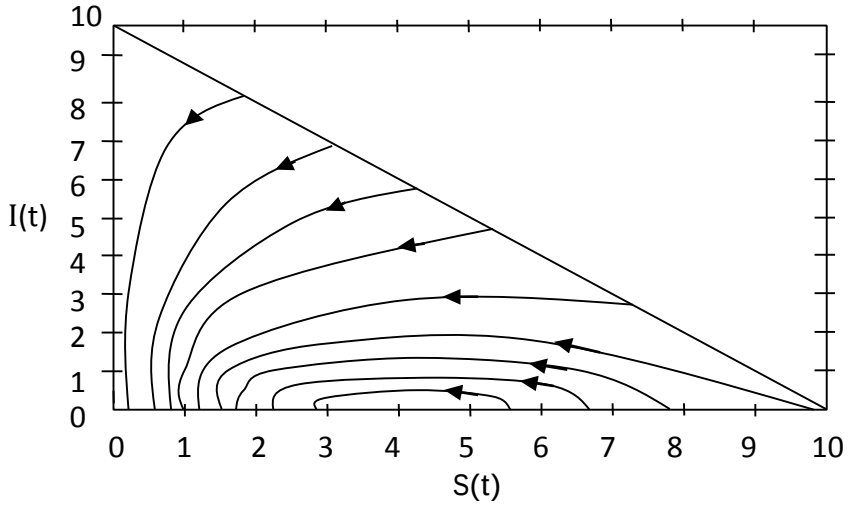

Figure 2: The phase portrait for the SI phase plane

Also (1) implies that if the term−μSI = 0, then either S = 0 or I = 0. If I = 0, then$\frac{dI}{d} =$ 0 which means an infection-free population will remain infection-free forever. On the contrary, if I ≠ 0, and S –then$\frac{dI}{d}$ 0 which is a threshold condition.

Basic Reproductive Number ( ) of Hepatitis B without Vital Dynamics: The basic reproduction number is one of the most important threshold quantities used in epidemiology, it is denoted byR_0 and it is defined as the average number of secondary infections produced when one infective is introduced into a host population where everyone is susceptible, (Heffernan *et al.*, 2005). This implies that, S 0 or equivalently$\frac{S\ 0}{}$. When $S(0) < \frac{\alpha}{\mu}$,it implies $\frac{\mu S(0)}{\alpha} < 1$ and this statement gives$\frac{\mu}{\alpha} < 1$. Again when S(0) >$\frac{\alpha}{\mu}$, it implies $\frac{\mu S(0)}{\alpha} > 1$ and this statement gives $\frac{\mu}{\alpha} > 1$.Refer to

$$R_0 = \frac{\mu}{} \tag{4}$$

When$R_0 = \frac{\mu}{\alpha} < 1$, then μ< , implying that the disease will die out. On the other hand, $R_0 = \frac{\mu}{\alpha} > 1$ implies that μ> , so an epidemic occurs.

Also, $R_0 = \frac{\mu}{\alpha} = \mu \times \frac{1}{\alpha}$, is the product of the contact rate μ per unit time and the average infection period $\frac{1}{\alpha}$. It can therefore be interpreted as the average number of adequate contacts, a typical infective, makes with both susceptible and infected persons within the period. To obtain an expression for the final size of an epidemic, dividing (2) by (1) gives

$\frac{dI}{dS} = \frac{\mu SI}{\mu SI} \frac{I}{} =$ $\frac{}{\mu S}$

$$I = \left(\quad \frac{}{\mu S} \quad S = \left(\quad \frac{1}{S} \quad S \right. \right. \tag{5}$$

Integrating equation (5) using the initial conditions gives

$$dI = -dS + \frac{1}{R_0 S} dS$$

$$\int_{I_0}^{I} dx = -\int_{S_0}^{S} dy + \frac{1}{R_0}\int_{S_0}^{S} \frac{1}{y} dy$$

$$I - I_0 = -(S - S_0) + \frac{1}{R_0} \ln \left| \frac{S}{S_0} \right|$$

Taking limits as $t \to \infty$ of S(t) and I(t) = 0, then

$$S_0 + I_0 = S_\infty - \frac{1}{R_0} \ln \left| \frac{S_\infty}{S_0} \right|$$

Let = $S_0 + I_0$, then $= S \quad \frac{1}{} l \quad \frac{S}{S}$

Making the reciprocal of R_0 the subject, gives

$$\frac{1}{} = \frac{S}{S \quad S} \tag{6}$$

Equation (6) is known as the final size equation.

SIR Model of Hepatitis B with Vital Dynamics

When a disease persists in a population for a long period of time, birth and death must be taken into consideration. Let S(t), I(t) and $R(t)$ be proportions of susceptible, infective and recovered individuals respectively, each with natural death rate of σ and birth rate of ε. This is shown in figure 3.

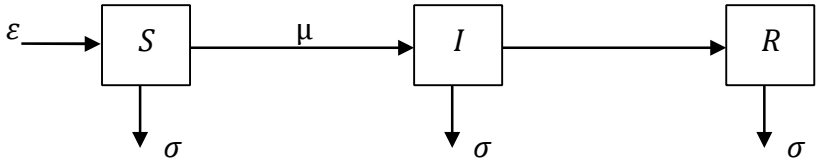

Figure 3: Flowchart of the SIR model of Hepatitis B with vital dynamics

With the notations given above, the SIR model with vital dynamics for Hepatitis B is obtained as

$$\frac{dS}{d} = \varepsilon \quad \mu SI \quad \sigma S \tag{7}$$

$$\frac{dI}{d} = \mu SI \quad \sigma I \quad I \tag{8}$$

$$\frac{d}{d} = \quad I \quad \sigma R \tag{9}$$

With initial conditions S(0) = S_0,$I(0) = I_0 > 0$, $R(0) = 0$, where assume $\varepsilon = \sigma$.
SinceS(t) + $I(t)$ + $R(t)$ = 1,R is calculated from $R(t) = 1 - S(t) - I(t)$, using the values of$S(t)$ and I(t) from the reduced systems (7) and (8) above.

Equilibrium Points for SIR Model of Hepatitis B with Vital Dynamics

Linearization approximation is a standard phase plane technique used to analyze system dynamics. For any SIR system with a constant host population size, at points of equilibrium the derivatives in (7) and (8) equals zero, thus

$$\frac{dS}{d} = \varepsilon \quad \mu SI \quad \sigma S = 0 \tag{10}$$

$$\frac{dI}{d} = \quad \mu S \quad \sigma \quad I = 0 \tag{11}$$

Solving simultaneously, let I = 0 from (10), then $\varepsilon - \sigma S$ = 0and$S = \frac{\varepsilon}{\sigma}$. Since $\varepsilon = \sigma$, then it implies S = 1. Hence the equilibrium point is$E_0(S^*, I^*) = (1,0)$.This gives us a disease-free equilibrium of Hepatitis B.

From (11) $S = \frac{(\sigma + \alpha)}{\mu}$

Substituting the value of S into (10), gives

$$\varepsilon \quad \mu\left(\frac{}{\mu} \quad I \quad \sigma \frac{}{\mu}\right. \tag{12}$$

Making I the subject from equation (12)

$$I = \frac{\mu\varepsilon - \sigma(\sigma + \alpha)}{\mu} \times \frac{1}{(\sigma + \alpha)} = \frac{\mu\varepsilon - \sigma(\sigma + \alpha)}{\mu(\sigma + \alpha)}$$

thus the equilibrium point is

$$\left(\frac{}{\mu} \quad \frac{\mu\varepsilon}{\mu}\right. \tag{13}$$

This equilibrium point is called the endemic equilibrium point.

Hartman-Grobman Theorem states that in a continuous model, a steady state will be stable provided the eigenvalues of the characteristic equation are both negative (if real) or have a negative real part (complex). The stability can be determined by finding the Jacobian matrix from (10) and (11). This gives

$$\begin{matrix} \mu I & \sigma & \mu S \\ \mu I & \mu S & \sigma \end{matrix} \tag{14}$$

Stability of Disease-free Equilibruim for SIR Model of Hepatitis B with Vital Dynamics

From earlier calculations, the disease-free equilibrium is $E_0(S^*, I^*) = (1,0)$. In order to determine the stability of the model at this point, evaluate the Jacobian matrix at this equilibrium point and find the eigenvalues corresponding to this point. Evaluating the Jacobian at the disease-free equilibrium point, leads to

$$J \quad 0 \quad = \quad \begin{matrix} \mu & 0 & \sigma & \mu \\ \mu & 0 & \mu & \sigma \end{matrix} = \begin{pmatrix} \sigma & \mu \\ 0 & \mu \quad \sigma \end{pmatrix} \tag{15}$$

Next find the characteristic equation which is given by $\det A \quad \lambda I \quad = 0$ where λ is the eigenvalues of A and A an × matrix. Thus

$$\det(A - \lambda I) = det\left[\begin{pmatrix} -\sigma & -\mu \\ 0 & \mu - \sigma - \alpha \end{pmatrix} - \lambda \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix}\right]$$

$$= \det \begin{matrix} \sigma - \lambda & \mu \\ 0 & \mu - \sigma \quad - \lambda \end{matrix}$$

$$= (-\sigma - \lambda)\ (\mu - \sigma - \quad - \lambda) - (-\mu)\ (0)$$

Because det $(A - \lambda I) = 0$,

Implies $(-\sigma - \lambda)(\mu - \sigma - \quad - \lambda) - (-\mu)\ (0) = 0$

Therefore, $\lambda_1 = -\sigma < 0$ and $\lambda_2 = \mu - \sigma - \quad$. (16)

Thus, the stability of the disease-free equilibrium depends on the values of σ, and μ.

The Basic Reproductive Ratio ( ) of Hepatitis B with Vital Dynamics

From (7) and (8), it is concluded that the average time of an infection is $\frac{1}{\alpha+\sigma}$ and as infectious individuals infect others at rate μ, the basic reproductive number

$$R_0 = \frac{\mu}{} \tag{17}$$

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

Endemic Equilibrium of Hepatitis B with Vital Dynamics: The endemic equilibrium at the point in time where all the compartments of the population coexist is called the endemic period. Considering the situation whereby there is coexistence of the two main categories (i.e. the susceptible and the infective). This is seen in the endemic equilibrium point in the equation below

$$\left(\frac{}{\mu}\ \frac{\mu\varepsilon}{\mu}\right. \tag{18}$$

In order to determine the stability of this point, just resort to the same approach used in determining the stability of the disease-free equilibrium. The Jacobian matrix is evaluated at the endemic point by putting (18) into (14)

$$J(S^*, I^*) = \begin{pmatrix} -\mu\left(\frac{\mu\varepsilon - \sigma(\sigma+\alpha)}{\mu(\sigma+\alpha)}\right) - \sigma & -\mu\left(\frac{\sigma+\alpha}{\mu}\right) \\ \mu\left(\frac{\mu\varepsilon - \sigma(\sigma+\alpha)}{\mu(\sigma+\alpha)}\right) & \mu\left(\frac{\sigma+\alpha}{\mu}\right) - (\sigma+\alpha) \end{pmatrix}$$

$$= \begin{pmatrix} \left(\frac{-\mu\varepsilon + \sigma(\sigma+\alpha) - \sigma(\sigma+\alpha)}{(\sigma+\alpha)}\right) & -(\sigma+\alpha) \\ \left(\frac{\mu\varepsilon - \sigma(\sigma+\alpha)}{(\sigma+\alpha)}\right) & 0 \end{pmatrix}$$

$$= \begin{pmatrix} \frac{-\mu\varepsilon}{(\sigma+\alpha)} & -(\sigma+\alpha) \\ \frac{\mu\varepsilon - \sigma(\sigma+\alpha)}{(\sigma+\alpha)} & 0 \end{pmatrix}$$

The characteristic equation given by $\det(A - \lambda I) = 0$ is then solved, where λ is the eigenvalues and A is an matrix. Thus,

$$\det(A - \lambda I) = det\left[\begin{pmatrix} \left(\frac{-\mu\varepsilon}{(\sigma+\alpha)}\right) & -(\sigma+\alpha) \\ \frac{\mu\varepsilon - \sigma(\sigma+\alpha)}{(\sigma+\alpha)} & 0 \end{pmatrix} - \lambda\begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix}\right]$$

$$= det\begin{pmatrix} \left(\frac{-\mu\varepsilon}{(\sigma+\alpha)} - \lambda\right) & -(\sigma+\alpha) \\ \frac{\mu\varepsilon - \sigma(\sigma+\alpha)}{(\sigma+\alpha)} & -\lambda \end{pmatrix}$$

$$= \left(\frac{-\mu\varepsilon}{(\sigma+\alpha)} - \lambda\right)(-\lambda) + \left(\frac{\mu\varepsilon - \sigma(\sigma+\alpha)}{(\sigma+\alpha)}\right)(\sigma+\alpha)$$

$\det(A - \lambda I) = 0$, implies

$$\left(\frac{-\mu\varepsilon}{(\sigma+\alpha)} - \lambda\right)(-\lambda) + \mu\varepsilon - \sigma(\sigma+\alpha) = 0$$

$$\frac{\mu\varepsilon}{(\sigma+\alpha)}\lambda + \lambda^2 + \mu\varepsilon - \sigma^2 - \sigma\alpha = 0$$

$$(\sigma+\alpha)\lambda^2 + \mu\varepsilon\lambda + (\sigma+\alpha)(\mu\varepsilon - \sigma^2 - \sigma\alpha) = 0$$

$$\lambda_{1,2} = \frac{-\mu\varepsilon \pm \sqrt{(\mu\varepsilon)^2 - 4(\sigma+\alpha)(\sigma+\alpha)(\mu\varepsilon - \sigma^2 - \sigma\alpha)}}{2(\sigma+\alpha)}$$

$$\frac{-\mu \quad \left(\frac{\mu}{} \quad 4\ \mu\varepsilon}{} \tag{19}$$

Hence, the stability of the endemic equilibrium depends on the values of σ, , μ and ε. SIR Model with Vaccination: In general, use SIR model to describe the transmission dynamics of the disease if the vaccination leads to permanent immunity e.g. assume that a portion of susceptible, bS, go to the removal compartment R directly, due to permanent immunity obtained from vaccination, as depicted in figure 4.

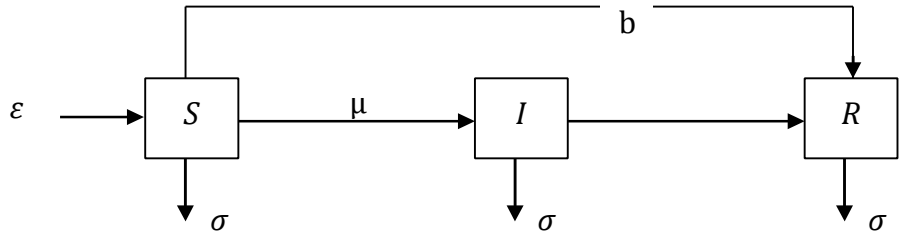

Figure 4: Flowchart of an SIR model with vaccination.

where$b$ is the vaccinating rate for the susceptible, from the diagram above (Fig. 4)

$$\frac{dS}{d} = \varepsilon \quad \mu SI \quad \sigma S \quad bS, \tag{20}$$

$$\frac{dI}{d} = \mu SI \quad \sigma \quad I, \tag{21}$$

$$\frac{d}{d} = \quad I \quad \sigma R \quad bS, \tag{22}$$

where it is assumed$\varepsilon = \sigma + b$.

Equilibrium Points for SIR Model with Vaccination:

The equilibrium points are solved using,

$$\varepsilon \quad \mu SI \quad \sigma S \quad bS = 0 \tag{23}$$

$$\mu S \quad \sigma - \quad I = 0 \tag{24}$$

Solving simultaneously and from (24) $\mu S \quad \sigma \quad = 0$

$$S = \frac{}{\mu} \tag{25}$$

Since $\varepsilon = \sigma + b$, then it impliesS = 1. Hence the equilibrium point is $_0(S^*, I^*)$ = (1,0). This gives us a disease-free equilibrium of Hepatitis B. Substituting the value of S into (23), leads to

$$\varepsilon \quad \mu SI \quad \sigma S \quad bS = 0$$

$$\mu SI = \varepsilon \quad \sigma S \quad bS$$

$$I = \frac{\varepsilon}{\mu S} \quad \frac{\sigma S}{\mu S} \quad \frac{bS}{\mu S}$$

$$I = \frac{\varepsilon}{\mu S} \quad \frac{\sigma}{\mu} \quad \frac{b}{\mu}$$

$$I = \frac{\varepsilon}{\mu}\left(\frac{\mu}{\sigma} \quad \frac{\sigma}{\mu} \quad \frac{b}{\mu}\right.$$

$$I = \frac{\varepsilon\mu \quad \sigma\ \sigma \quad b\ \sigma}{\mu\ \sigma}$$

$$I = \frac{\varepsilon\mu}{\mu} \tag{26}$$

Thus, the equilibrium point is

$$\left(\frac{}{\mu} \quad \frac{\varepsilon\mu}{\mu} \right. \tag{27}$$

This equilibrium point (27) is called the endemic equilibrium point.

The stability is determined by finding the Jacobian matrix using (23) and (24). This gives

$$J = \begin{matrix} \mu I & \sigma & b & \mu S \\ \mu I & & \mu S & \sigma \end{matrix} \tag{28}$$

Disease-Free Equilibrium for SIR Model with Vaccination

Evaluating the Jacobian at the disease-free equilibrium point, gives

$$J\ \ 0\ = \det \begin{matrix} \mu & 0 & \sigma & b & & \mu \\ & \mu & 0 & & \mu & \sigma & \alpha \end{matrix}$$

$$J\ \ 0\ = \det \begin{matrix} \sigma & b & & \mu \\ 0 & & \mu & \sigma & \alpha \end{matrix} \qquad (29)$$

Thus, $\det(A - \lambda I) = \det \begin{matrix} \sigma & b & & \mu & & \lambda\left(& 0 \\ 0 & & \mu & \sigma & \alpha & & \end{matrix}$

$$= \det \begin{matrix} \sigma & b & \lambda & & \mu \\ & 0 & & \mu & \sigma & \alpha & \lambda \end{matrix}$$

$$= \quad \sigma \quad b \quad \lambda \ \mu \quad \sigma \qquad \lambda \qquad \mu \ \ 0$$

And since det $(A - \lambda I) = 0$, this implies $(-\sigma - b - \lambda)(\mu - \sigma - \ - \lambda) - (-\mu)(0) = 0$

Therefore, $\lambda_1 = -\sigma - b$ or $\lambda_2 = \mu - \sigma -$, these are actually the eigenvalues corresponding to the disease-free equilibrium and so

$$_0\ S^*\ I^*\ =\ \sigma\ \ b \text{or} \mu\ \ \sigma \qquad (30)$$

Hence, the stability of the disease-free equilibrium with vaccination depends on the values of , , $ba\ \ \mu$.

Basic Reproductive Ratio ( ) of Hepatitis B with Vaccination

For $etA\ \ \lambda I$ to be asymptotically stable, both eigenvalues must be negative. From $etA\ \ \lambda I\ = 0$ it is clear that $\lambda_1 =\ \ \sigma\ \ b$ is negative and therefore if $\lambda\ = \mu\ \ \sigma$ 0 then both eigenvalues are negative and R_0 . Hence the disease-free equilibrium is asymptotically stable. On the other hand, if $\lambda_2 = \mu - \sigma -\ > 0$, then $\det(A - \lambda I)$ is unstable and the stability of the disease-free equilibrium with vaccination depends on the values of σ, , $b$and μ.

Endemic Equilibrium of Hepatitis B with Vaccination: The endemic equilibrium point is given by equation (31) below, where there is coexistence between the two main categories.

$$\left(\frac{}{\mu}\ \ \frac{\varepsilon\mu}{\mu} \qquad (31)$$

In order to determine the stability of this point, resort to the same approach used in determining the stability of the disease-free equilibrium. The Jacobian matrix at the endemic point is given by

$$J(S^*, I^*) = \begin{pmatrix} -\mu\left(\frac{\mu\varepsilon - (\sigma+b)(\sigma+\alpha)}{\mu(\sigma+\alpha)}\right) - \sigma & -\mu\left(\frac{\sigma+\alpha}{\mu}\right) \\ \mu\left(\frac{\mu\varepsilon - (\sigma+b)(\sigma+\alpha)}{\mu(\sigma+\alpha)}\right) & \mu\left(\frac{\sigma+\alpha}{\mu}\right) - (\sigma+\alpha) \end{pmatrix}$$

$$J(S^*, I^*) = \begin{pmatrix} -\left(\frac{\mu\varepsilon - (\sigma+b)(\sigma+\alpha)}{(\sigma+\alpha)}\right) - \sigma & -(\sigma+\alpha) \\ \left(\frac{\mu\varepsilon - (\sigma+b)(\sigma+\alpha)}{(\sigma+\alpha)}\right) & 0 \end{pmatrix}$$

$$J(S^*, I^*) = \begin{pmatrix} \frac{-\mu\varepsilon + (\sigma+b)(\sigma+\alpha) - \sigma(\sigma+\alpha)}{(\sigma+\alpha)} & -(\sigma+\alpha) \\ \frac{\mu\varepsilon - (\sigma+b)(\sigma+\alpha)}{(\sigma+\alpha)} & 0 \end{pmatrix}$$

$$J(S^*, I^*) = \begin{pmatrix} \frac{-\mu\varepsilon + (\sigma+\alpha)(\sigma+b-\sigma)}{(\sigma+\alpha)} & -(\sigma+\alpha) \\ \frac{\mu\varepsilon - (\sigma+b)(\sigma+\alpha)}{(\sigma+\alpha)} & 0 \end{pmatrix}$$

$$J(S^*, I^*) = \begin{pmatrix} \dfrac{-\mu\varepsilon + b(\sigma+\alpha)}{(\sigma+\alpha)} & -(\sigma+\alpha) \\ \dfrac{\mu\varepsilon - (\sigma+b)(\sigma+\alpha)}{(\sigma+\alpha)} & 0 \end{pmatrix}$$

Next is to find the characteristic equation which is given by det($A - \lambda I$) = 0. Where λ is the eigenvalue and A is an × matrix. Thus,

$$\det(A-\lambda I) = det\left[\begin{pmatrix} \dfrac{-\mu\varepsilon + b(\sigma+\alpha)}{(\sigma+\alpha)} & -(\sigma+\alpha) \\ \dfrac{\mu\varepsilon - (\sigma+b)(\sigma+\alpha)}{(\sigma+\alpha)} & 0 \end{pmatrix} - \lambda\begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix}\right]$$

$$= det\begin{pmatrix} \dfrac{-\mu\varepsilon + b(\sigma+\alpha)}{(\sigma+\alpha)} - \lambda & -(\sigma+\alpha) \\ \dfrac{\mu\varepsilon - (\sigma+b)(\sigma+\alpha)}{(\sigma+\alpha)} & -\lambda \end{pmatrix}$$

$$= \left(\frac{-\mu\varepsilon + b(\sigma+\alpha)}{(\sigma+\alpha)} - \lambda\right)(-\lambda) + \left(\frac{\mu\varepsilon - \sigma(\sigma+\alpha)}{(\sigma+\alpha)}\right)(\sigma+\alpha)$$

Because det($A - \lambda I$) = 0, implies

$$\frac{\mu\varepsilon \quad b\ \sigma}{\sigma} \quad \lambda \quad \lambda \quad \frac{\mu\varepsilon \quad \sigma\ \sigma}{\sigma} \quad \sigma \quad = 0$$

$$\lambda \quad \frac{\mu\varepsilon \quad b\ \sigma}{\sigma} \lambda \quad \mu\varepsilon \quad \sigma\ \sigma \quad = 0$$

$$\sigma \quad \lambda \quad \mu\varepsilon \quad \frac{b\ \sigma}{} \quad \lambda \quad \sigma \quad \mu\varepsilon \quad \sigma \quad \sigma \quad = 0$$

$$\lambda_1 = \frac{\mu\varepsilon \quad b\ \sigma \qquad \mu\varepsilon \quad b\ \sigma \qquad \sigma \quad \sigma \quad \mu\varepsilon \quad \sigma \quad \sigma}{\dfrac{}{2\ \sigma}}$$

$$\lambda_1 = \frac{\dfrac{\mu -}{} \quad \left(\dfrac{\mu -}{} \quad 4\ \mu\varepsilon\right.}{} \qquad (32)$$

Hence, the stability of the endemic equilibrium with vaccination depends on the values of σ, , μ, ε and b. Herd Immunity Threshold: In a large group of individuals where there exists a contagious disease, if a large enough of individuals is immune to the disease, the chances that a chain of disease transmission will be interrupted are very high, resulting in self-contained, small outbreaks that will die out quickly, (Diekman and Heesterbeek, 2000). Thus, the wall that is set up by the vaccinated ones will protect individuals that are not immune. The Herd Immunity Threshold (H_1) is percentage of the population that needs to be immune to control transmission of a disease. Diekman and Heesterbeek (2000) provided an equation for estimating the Herd Immunity Threshold. The equation is given as

$$H_1 = 1 - \frac{1}{R_0}$$

Substituting $R_o = \frac{\mu}{\sigma+\alpha}$, into the equation above, gives

$$H_1 = \qquad \left(\frac{\mu}{\sigma} \quad = \quad \frac{\sigma}{\mu}\right.$$

$$\therefore \quad H_1 = \frac{\mu}{\mu} \qquad (33)$$

Therefore, the level of vaccination is directly proportional to the herd immunity threshold; as the amount of vaccination increases, the herd immunity threshold also increases.

Numerical Solution Using Euler Method:This refers to the formulation of the SIR model using Euler method by first parametrizing the model and then transforming the SIR model into the Euler formula. Now, the SIR model is used to illustrate the transfer of the epidemic through the interaction of the following three different variables:

S = Number of people that are susceptible to Hepatitis B

I = Number of people infected with Hepatitis B

R = Number of people recovered from Hepatitis B with total immunity

It makes sense to assume that a fixed population of N people, whereby there are no births and deaths by natural cause i.e.

$$= S + I + R$$

This is because the population is fixed and therefore, there are only three compartments in which the population may fit into. Thus, the total of the number of people susceptible, infected and recovered in equivalent to the total population. The assumption that N is fixed, with no births or deaths, makes sense given in days, although it is a simplification (Hossain *et al.,* 2017).

These variables change over time, so define the variable t = time in days and set t = 0 at the initial time. The model uses two parameters β (the rate of infection) and (the rate of recovery), with β, > 0. Given these parameters, the model uses 3 differential equations. The rate of change of the number of people susceptible to the disease over time

$$\frac{dS}{d} = \beta IS \quad (34)$$

The rate of change of the number of people recovered over time

$$\frac{d}{d} = \quad I \quad (35)$$

The rate of change of the number of people infected.

$$\frac{dI}{d} = \beta IS \qquad I \quad (36)$$

Parameterization of the model: In order to calculate β(the rate of infection) and (the rate of recovery), it helps to define two more parameters.

= Duration of disease for those recovered

= Mortality rate for those who die per day

This leads to two further equations:

The rate at which the disease is spread

$$= \frac{1}{} \quad (37)$$

The infection rate of the disease

$$\beta = \frac{}{S} \quad (38)$$

Transformation of Euler Equations for SIR modeling: Consider a "slope formula," i.e., a way to calculate d / tat any point $(t,)$*, then generate a sequence of* y*-values,*

$$\quad _0 \quad _1 \quad \ldots$$

By starting from a given $_0$ and computing each *rise* as *slope* x *run*. That is,

$$_{+1} = \quad + \text{slope}_n \Delta t$$

whereΔt is a suitably small step size in the time domain.

It really does not matter in this calculation if the slope formula depends not just on t and y but on other variables. For instance, a case where x and z happen to be other dependent variables in a system of differential equations; obviously, for an SIR model, the dependent variable names are$S$, I and R;which leads to the three Euler formulas of the form:

$$S_{+1} = S + \text{slope}_n \Delta t \quad (39)$$

$$I_{+1} = I + \text{slope}_n \Delta t \quad (40)$$

$$R_{+1} = R + \text{slope}_n \Delta t \quad (41)$$

More specifically, given the SIR (34), (35), and (36),

The Euler formulas become

$$S_{+1} = S - \beta I S \Delta t \quad (42)$$

$$I_{+1} = I + (\beta I S - I)\Delta t \quad (43)$$

$$R_{+1} = R + I\Delta t \quad (44)$$

To calculate using these formulas, there must be explicit values *for*β, ,S(0), $I(0)$, $R(0)$ and Δt.

Results and Discussion

Computational Analysis of SIR and Euler Methods

Considering a Hepatitis B outbreak in Kaduna metropolis for 60 days, the recorded number (N) of people infected was 267, the number (I) of people who died as a result of the infection was 45 and the number (R) of people that recovered was 12. Note that the recovered includes both who died and the survivors with permanent immunity. Using this data and the following parameters with values:N = 267; I = 45; R = 12

Thus, the number of susceptible is $S = \quad I \quad R = 267 \quad 5 \quad 2 = 2 \quad 0$
The duration of the disease ranges from 2 to 21 days, therefore the estimated duration (days) of the disease at the midpoint is given by D = 11: Thus, the rate at recovery is

$$= — = 0.09$$

Using mortality rate of 0.105 and S =210, gives
Rate of infection$= \frac{0.10}{10} = 5 \quad 0^4$

In order to use the SIR model to predict the evolution of the disease, solve the system of differential equations using Euler method.

Euler Method

For each day, the values of S, $I$and$R$ using (42) (43) & (44) were calculated as

$$S_{+1} = S - \beta I S \Delta t$$

$$I_{+1} = I + (\beta I S - I)\Delta t$$

$$R_{+1} = R + I\Delta t$$

The following initial values were considered:
S_0 = 210; I_0 = 45; R_0 = 12; = 0.09; β =5 0^4;
Solving this explicitly for the transition from t = 0 to t = 1. The following values for S, I and R were computed (S, I, R over a two-month period); the results are shown in Table1.

Table 1: Computed Values of S, I & R Using Euler's Method

TIME (DAY)	S	I	R	TIME (DAY)	S	I	R
1	205	46	16	31	107	30	128
2	200	47	20	32	105	29	131
3	195	48	24	33	104	28	134
4	190	48	28	34	103	27	137
5	185	48	32	35	102	26	139
6	181	48	36	36	101	25	141
7	177	48	40	37	100	24	143
8	173	48	44	38	99	23	145
9	169	48	48	39	98	22	147
10	165	48	52	40	97	21	149
11	161	48	56	41	96	20	151

	12	157	48	60	42	95	19	153
	13	153	48	64	43	94	18	155
	14	149	47	68	44	93	17	157
	15	146	46	72	45	92	16	159
The	16	143	45	76	46	91	15	160
	17	140	44	80	47	90	14	161
	18	137	43	84	48	89	13	162
	19	134	42	88	49	88	12	163
	20	131	41	92	50	88	12	164
	21	128	40	96	51	88	12	165
	22	125	39	100	52	88	12	166
	23	123	38	104	53	88	12	167
	24	121	37	107	54	88	12	168
	25	119	36	110	55	88	12	169
	26	117	35	113	56	88	12	170
	27	115	34	116	57	88	12	171
	28	113	33	119	58	88	12	172
	29	111	32	122	59	88	12	173
	30	109	31	125	60	88	12	173

dynamics of the various compartments of the SIR model with the actual data, and Euler method during the outbreak are shown in Figures5&6

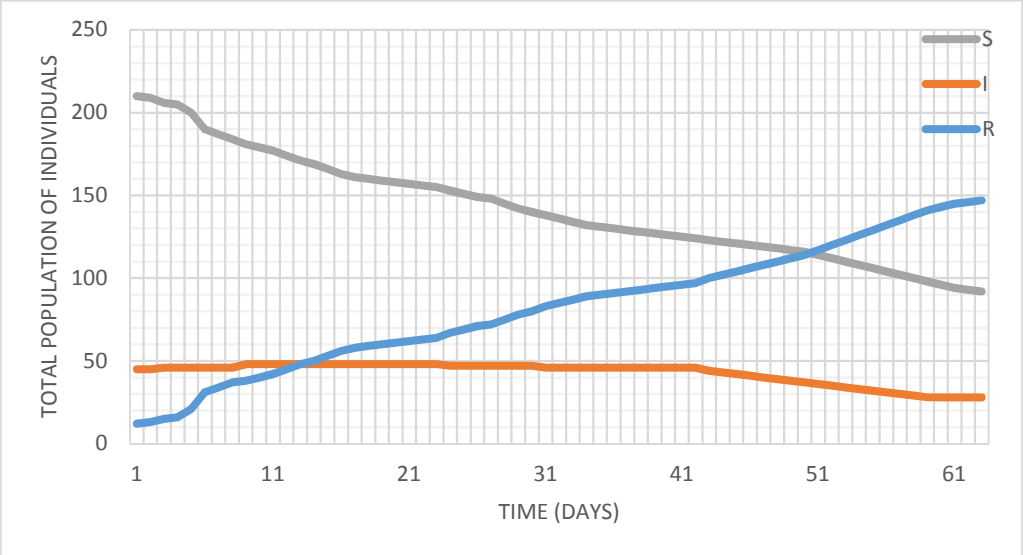

Figure 5: The Actual Number of Susceptible, Infectives & Recovered in Hepatitis B Outbreak

From figure 5, it was observed that the initial number of infectives was 45, while the proportion of the susceptibles sharply declined from an initial value of 210 to an approximate minimum value of 96 from day one to day 60. The proportion of the infectives declined asymptotically from first day reaching a minimum value of 28 on the 59th day, which was maintained onwards. Also, the proportion of the recovered population increased exponentially and reached a value of 143 at 60^{th} day.

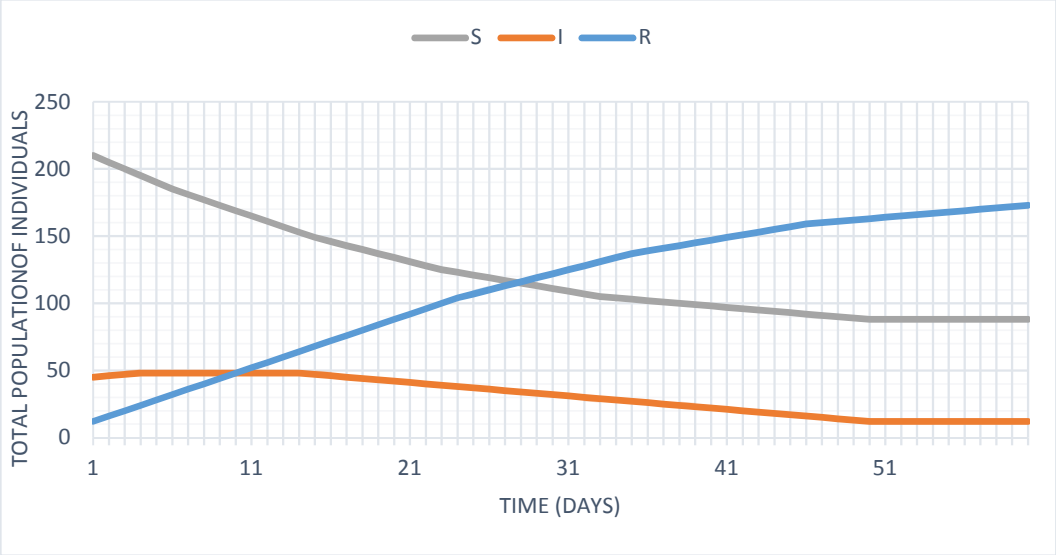

Figure 6: Chart of Susceptibles, Infectives & Recovered with Euler Method in Hepatitis BOutbreak

Figure 6 shows that while the initial proportion of infectives of the Euler method was 45, the proportion of the recovered rose exponentially from 12 at day one to a peak value of 173 at the 60th day. A steady decrease in the proportion of infectives from initial value of 46 to a value of 12 was also observed and it remained stable as days progresses. Similarly, the gradual reduction in the number of susceptibles from 210 to 89 was recorded, while from 49th day and onwards it stabilized at a value of 88.

Prediction of the Evolution

Naturally the prediction focuses on the infective individuals; results from all the scenarios have shown a steady rise (increase) of the number of infected individuals, which over an extended period of time decreases and give rise to the number of recovered individuals. This state can be attributed to two main reasons, sustained awareness of the disease and continuous medical support being given in order to assist in combating the transmission of the disease. Furthermore, an increased awareness opts up the consciousness of strategies of protection within the populace. The steep increases primarily recorded at the beginning is probably due to the minimal awareness, which in turn exacerbated the rate of transmission. The peak of each of the graph portrays the maximum number of infected individuals, after which transitional decrease occurred.

Comparative Analysis

However, in order for the model to have a validity and allow an informed government policy, it obviously needs to correspond fairly close to reality, thus compare the results obtained with the actual data, this shown in Table 2 and Figure 7.

Table 2: Real Life Data versus computed results using the SIR-Euler model

Day	Actual	SIR-Euler
1	45	46
2	46	47
5	46	48
9	48	48
10	48	48
15	48	46
21	48	40
24	47	37
26	47	35
27	47	34
30	46	31
31	46	30
32	46	29
35	46	26
39	46	22
40	46	21
45	41	16
51	35	12
54	32	12
56	30	12
57	29	12
60	28	12

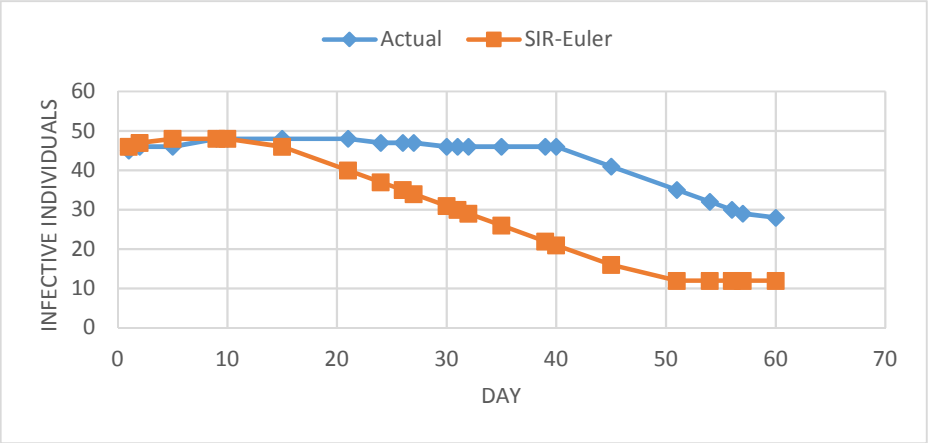

Figure 7: Graph of Infectives for Actual and SIR-Euler Method in Hepatitis B Outbreak

The graph in Figure 7, illustrates the outbreak of the infection of the SIR-Euler method with the actual number of infectives. Here the actual infectives exhibited similar behaviour to that of Euler's though with a slight increase. The two methods gave best approximations during the first 20 days and roughly estimated the values towards the last 10 days.

Results of SIR model with vital dynamics

For the SIR model with vital dynamics the estimated parameters in Table 3, which were computed from the sourced data, were used to generate the results.

Table 3: Parameter values for the SIR model with vital dynamics

Description	Parameter	Value
Birth rate	ε	0.083
Infectious rate	μ	0.169
Recovered rate	α	0.0449
Natural death rate	σ	0.083
Vaccination rate	b	0.042

From (17), the reproductive number is obtained as

$R_0 = \frac{\mu}{} = \frac{0.169}{0.0449 \quad 0.0} = \quad .32 \quad 3$

This means that on the average, one hepatitis B patient contacts 1.3213 susceptible people in the population during his/her infectious period. Since the reproductive number R_0 = 1.3213 > 1, an outbreak of hepatitis B will result in an epidemic.

Stability Analysis of the Model with Vital Dynamics

The linear stability of the infectious free equilibrium point $E_0(S^*, I^*) = (1,0)$, is analyzed by substituting the parameter values in Table3 into (16), the eigenvalues corresponding to the infectious free equilibrium are $\lambda_1 = -0.083$ and $\lambda_2 = 0.0411$; since the two eigenvalues are both real, one is positive and the other negative, it implies the disease free equilibrium is a saddle point, therefore unstable. The unstable equilibrium implies that the presence of a Hepatitis B positive patient will eventually result in an outbreak of the disease.

The endemic equilibrium point occurs at a time where all the compartments of the population coexists in the population. The introduction of an infected person will infect others, therefore changing the health condition of a lot of people. Substituting the parameter values in Table3into (19), to obtain the eigenvalues corresponding to the endemic equilibrium, which is given by

$$\lambda_{1,2} = \frac{-\left(\frac{\mu\varepsilon}{\sigma+\alpha}\right) \pm \sqrt{\left(\frac{\mu\varepsilon}{\sigma+\alpha}\right)^2 - 4\left(\mu\varepsilon - \sigma(\sigma+\alpha)\right)}}{2}$$

$$\lambda_{1,2} = \frac{\left(\frac{.1 \quad .}{. \quad . \quad 44}\right) \quad \left(\frac{.1 \quad .}{. \quad . \quad 44}\right) \quad 4 \; 0.169 \; 0.0 \quad 0.0 \quad 0.0 \quad 0.0449}{}$$

$$\lambda_{1,2} = \frac{0.109 \quad 0.109 \quad 0.01 \quad 6}{}$$

$\lambda_1 = -0.05484 + 0.02003$ and $\lambda_2 = -0.05484 + 0.02003$

Since the eigenvalues have real negative parts with complex conjugates, it implies the endemic equilibrium is asymptotically stable.

Sensitivity Analysis of the Model with Vital Dynamics

Vital dynamics that is introducing birth and death into a population when a disease persists for a long period of time, the results obtained are shown in Table4.

Table4: Classification of the disease-free equilibrium with Vital Dynamics

ε	μ	α	λ_1	λ_2	σ	R_0	Nature of the equilibrium
0.083	0.11	0.0449	-0.083	-0.0179	0.083	0.8600	Stable sink
0.083	0.169	0.0449	-0.083	0.0411	0.083	1.3213	Unstable saddle
0.083	0.130	0.0449	-0.083	0.002	0.083	1.0164	Neutrally stable
0.083	0.100	0.0449	-0.083	-0.0279	0.083	0.7819	Stable improper sink

From (16), it is observed that the eigenvalues, $\lambda_1 = -\sigma$ and since $\sigma > 0$, it implies that $\lambda_1 < 0$. Considering the second eigenvalue, $\lambda_2 = \mu - \sigma -$, stability can only be obtained if $\lambda_2 < 0$. Thus $\mu < \sigma + \alpha,$ and $\frac{\mu}{\sigma+\alpha} < 1$ implying $R_0 < 1$.

The disease-free equilibrium will be stable if the reproductive number is less than unity, i.e.$R_0 < 1$, whilst the disease-free equilibrium is unstable if the reproductive number is greater than unity, and the results in Table5 have shown this trend.

Table 5: Classification of equilibrium points of the endemic equilibrium with Vital Dynamics

ε	μ	α	λ_1	λ_2	σ	R_0	Nature of the equilibrium
0.083	0.11	0.0449	-0.01684	-0.08822	0.083	0.8600	Unstable saddle
0.083	0.169	0.0449	-0.05484 +0.80445i	-0.05484 -0.80445i	0.083	1.3213	Stable spiral sink
0.083	0.130	0.0449	-0.04218 +0.80450i	-0.04218 -0.80450i	0.083	1.0164	Stable spiral sink
0.083	0.100	0.0449	-0.03245 +0.80441i	-0.03245 -0.80441i	0.083	0.7819	Stable spiral sink

From Table 5, it is observed that the endemic equilibrium is stable when the reproductive number is greater than unity, i.e. $R_0 > 1$, and unstable when the reproductive number is less than unity, i.e. $R_0 < 1$.

Stability Analysis of the Model with Vaccination

Substituting the parameter values in Table3 into (30). The eigenvalues corresponding to the infectious free equilibrium are $\lambda_1 = -0.125$ and $\lambda_2 = 0.0411$. The eigenvalues are both real, one being positive and the other negative, implies the disease-free equilibrium is a saddle point, therefore unstable.

The endemic equilibrium point occurs when an infective is introduced into the population. Substituting the parameter values in Table3 into (32) to obtain the eigenvalues corresponding to the endemic equilibrium.

$$\lambda_{1,2} = \frac{\frac{-(\mu\varepsilon - b(\sigma+\alpha))}{\sigma+\alpha} \pm \sqrt{\left(\frac{\mu\varepsilon - b(\sigma+\alpha)}{\sigma+\alpha}\right)^2 - 4(\mu\varepsilon - \sigma(\sigma+\alpha))}}{2}$$

$$\lambda_{1,2} = \frac{\left(\frac{.1 \quad . \quad - . \quad 4 \quad . \quad . \quad 44}{. \quad . \quad 44}\right) \quad \overline{\left(\frac{.1 \quad . \quad - . \quad 4 \quad . \quad . \quad 44}{. \quad . \quad 44}\right) \quad 4 \; 0.169 \; 0.0 \quad 0.0 \quad 0.0 \quad 0.0449}}{}$$

$$\lambda_{1,2} = \frac{0.06 \quad \overline{0.06 \quad 0.01}}{}$$

$\lambda_1 = -0.03384 + 0.04761$ and $\lambda_1 = -0.03384 - 0.04761$

Since the eigenvalues have a complex conjugate with negative real parts, it implies the endemic equilibrium is asymptotically stable.

Sensitivity Analysis of the Model with Vaccination

Table6 displayed the sensitivity analysis of the model with Vaccination.

Table6: Classification of the disease-free equilibrium with Vaccination

ε	μ	α	λ_1	λ_2	σ	R_0	Nature of the equilibrium
0.083	0.11	0.0449	-0.125	-0.0179	0.083	0.8600	Stable sink
0.083	0.169	0.0449	-0.125	0.0411	0.083	1.3213	Unstable saddle
0.083	0.130	0.0449	-0.125	0.002	0.083	1.0164	Neutrally stable
0.083	0.100	0.0449	-0.125	-0.0279	0.083	0.7819	Stable improper sink

From (30), the eigenvalues $\lambda_1 = -\sigma - b$ and since σ and $b > 0$, it implies that $\lambda_1 < 0$.
The second eigenvalue is given as $\lambda_2 = \mu - \sigma -$. Stability can only be obtained if $\lambda_2 < 0$.
Thus $\mu < \sigma + \alpha,$ and $\frac{\mu}{\sigma+\alpha} < 1$.
The disease-free equilibrium will be stable if the reproductive number is less than unity, i.e. $R_0 < 1$, whilst the disease free equilibrium is unstable if the reproductive number is greater than unity.

Table7: Classification of equilibrium points of the disease endemic equilibrium with Vaccination

ε	μ	α	λ_1	λ_2	σ	R_0	Nature of the equilibrium
0.083	0.11	0.0449	0.02656	-0.05594	0.083	0.8600	Unstable saddle
0.083	0.169	0.0449	-0.03384 +0.80560	-0.03384 -0.80560	0.083	1.3213	Stable spiral sink
0.083	0.100	0.0449	-0.01145 +0.8050	-0.01145 -0.8050	0.083	1.0164	Stable spiral sink

From Table 7, it is observed that the endemic equilibrium is stable when the reproductive number is greater than unity, i.e. $R_0 > 1$, and unstable when the reproductive number is less than unity, i.e. $R_0 < 1$.

Results of Herd Immunity Threshold

From (33), the herd immunity ratio is given as

$$H_1 \quad \frac{0.169 \quad 0.0 \quad 0.0449}{0.169} \quad 0.2431$$

This implies that approximately 24.31 % of the susceptible population should be immunized in order to bring the spread of Hepatitis B under total control in the Kaduna metropolis.

Conclusion

The numerical solutions and sensitivity analysis gave us a clear picture of how sensitive and important each parameter is in the analysis. The infectious rate and the recovery rate play the dominant role in determining the outcome of Hepatitis B virus whenever there is an outbreak.

In the absence of vaccination, the susceptible population will reduce sharply when an infective is introduced into the population. The rate of decrease is directly proportional to the number of infective introduced into the population. With time, the infective population will reduce as more and more infective recover from the disease and become immune. The calculated reproductive ratio (R_0) was 1.1304 and this population is in danger should there be an outbreak. There is therefore the need to reduce the reproductive ration to less than one. To do this vaccination of more susceptible populace needs to be done, since it will give immunity to the individual. Also, awareness campaigns need to preached about the silent killer and by the campaign, horizontal transmission will be reduced since more and more people would be aware of the seriousness and consequence of sharing household items with someone whose HBV status is not known. The effect of vaccination was paramount from the solutions and sensitivity analysis. This shows that with increase in vaccination of the population, in addition to those who had already recovered from the disease will keep the population from an outbreak. The results obtained shows that the herd immunity is 0.2431, which implies that if 24.31% of the population could be immunized, the transmission rate of the disease would be greatly reduced, thereby enabling a control mechanism over the spread of the disease. Thus, the susceptible populace will be protected by the walls that are set up by the immune ones.

Future work

Further research work is needed for non-constant population since population cannot remain constant in reality and non-homogeneous population since the members of the population cannot always mix homogeneously.

References

Bratislava, L. B. (2007). The mathematics of infectious diseases. Hepatitis B. Wikipedia encyclopedia. Available From: https://en.wikipedia.org/wiki/Hepatitis_B [Accessed:28/07/2018]

Ciupe, S. M., Ribeiro, R. M., Nelson, P. W., Perelson A. S. (2007). Modeling the mechanisms of acute hepatitis B virus infection. *J Theory Biol., 247*(1), 23–35.

Diekmann, O., & Heesterbeek, J. A. P. (2000). *Mathematical epidemiology of infectious diseases.* Levin S, editor: John Wiley & Sons, Ltd.

Heffernan, J. M., Smith, R. J., & Wahl, L. M. (2005). Perspectives on the basic reproductive ratio. *J.R. Soc. Interface,* 2, 281-293.

Hossain, M. T., Miah, M. M., & Hossaon, M. B. (2017). Numerical study of kermack-mckendrik SIR model to predict the outbreak of Ebola virus diseases using Euler and fourth order Runge-Kutta Methods. *American Scientific Research Journal for Engineering, Technology, and Sciences (ASRJETS),* 37(1), 1 -21.

Letsa-Agbozo, J. K. (2014). Mathematical model of hepatitis B in the North Tongu district of the Volta region of Ghana. (Unpublished)

Kermack, W. O., McKendrick, A. G. (1927). Proceedings of the royal society of London. Series A. Containing Papers of a Mathematical and Physical Character. Volume 115, Issue 772, 700-721.

Medley, G. F., Lindop, N. A., Edmunds, W. J., & Nokes, D. J. (2001). Hepatitis-B virus endemicity: Heterogeneity, catastrophic dynamics and control. *Nat. Med.,* 7(5), 619-624.

Rodriguez, A. C. (2016). Mathematical models of Hepatitis B virus dynamics during antiviral therapy. March.

WHO (2016). Hepatitis B. WHO fact sheet. Available From: www.who.int/mediacentre/factsheets. [Accessed:27/07/2018]

WHO (2017). Global hepatitis report 2017. *World Health Organization.* Available from: https//www.who.int/hepatitis/publications/global-hepatitis-report2017/en/. [Accessed:29/07/2018]

Zou, L., Zhang, W., & Sndruan, S. (2010). Modeling the transmission dynamics and control Of hepatitis B virus in China. *Journal of Ttheoretical Biology*, 262, 330-338.