ISSN 2348 – 8034
Impact Factor- 3.155

GLOBAL JOURNAL OF ENGINEERING SCIENCE AND RESEARCHES

THE DYNAMICS OF A CHICKEN POX DISEASE IN A STAGE STRUCTURE POPULATION

Raid Kamel Naji[*1] and Hassan Fadhil Ridha[2]

[*1,2]Department of Mathematics, College of Science, University of Baghdad, Baghdad, IRAQ

ABSTRACT

In this paper, we have investigated the dynamical behavior of an SIR epidemic model represented by chicken pox disease that speared within a stage structure population in the absence of vaccine. Two types of transmitted modes are considered. The existence, uniqueness and boundedness of the solution of the model are discussed. The basic reproduction number , which represents an expected number of secondary cases produced, is computed. The local as well as global stability of the system in terms of basic reproduction number is investigated. The local bifurcation of the system is studied. The possibility of occurrence of Hobf bifurcation near the endemic equilibrium point is discussed. Numerical simulation is used to complete the analysis of the system.

Keywords- Chicken pox disease, Stability, stage structure, Local bifurcation, Hopf bifurcation.

I. INTRODUCTION

Chicken pox, is a highly contagious disease caused by the initial infection with varicella zoster virus (VZV). The disease results in a characteristic skin rash that forms small, itchy blisters, which eventually scab. It usually starts on the chest, back, and face, then spreads to the rest of the body. Other symptoms may include fever, feeling tired, and headaches, symptoms usually appear last five to ten days. Complications may occasionally include pneumonia, inflammation of the brain, or bacterial infections of the skin among others. The disease is more often, more severe in adults than children. Symptoms begin ten to twenty one days after exposure to the virus [1].

In the era of the absence vaccine, approximately 11,000 persons with varicella required hospitalization each year. Hospitalization rates were approximately 2 to 3 per 1,000 cases among healthy children and 8 per 1,000 cases among adults. Death occurred in approximately 1 in 60,000 cases. From 1990 to 1996, averages of 103 deaths from varicella disease were reported each year. Most deaths occur in immunocompetent children and adults. Since 1996, hospitalizations and deaths from varicella have declined more than 70% and 88% respectively [2]. The varicella vaccine has resulted in a decrease in the number of cases and complications from the disease. It protects about 70% to 90% of people from disease [3-6]. For this reason, epidemiological models have become important tools in analyzing the spread and control of infectious diseases after the simple model of Kermac–Mckendric on SIR system, in which the evolution of a disease which gets transmitted upon contact is described [7]. Afterward, several researchers work on the development of this model, where they studied the effect of vaccines, stage structure and treatments, for example, *Kribs-Zaleta* and *Velasco-Hernandez* in 2000 [8] have been proposed and studied the *SIS* epidemic model with vaccine for the diseases such as pertussis and tuberculosis Later on Arino et al. [9], generalized this model by allowing individuals recovering from the diseases to go into a temporarily immune class rather than directly back in to the susceptible class. Kribs-Zaleta and Martcheva [10] investigated the effects of a vaccination campaign upon the spread of non-fatal diseases such as Hepatitis A, B. *Alexander et al.* [11] and Shim [12] are discussing the transmission dynamics of influenza with vaccination through using *SVIR* models. d' Onofrio et al. [13] gave a family of models for information related vaccinating behavior. Aiello and Freedman [14] studied a stage-structured model of one species growth consisting of immature and mature members. Cui et al. [15] analyzed the effect of dispersal on the permanence of a stage-structured single-species population model without time delay. Cui and Song [16] proposed and analyzed a prey-predator model with stage structure for prey. Chen [17] studied the permanence of periodic predator–prey system with stage structure for prey. In this paper we proposed and studied a mathematical model for the dynamics of *SIR* epidemic, which represented by chicken pox disease, model within a stage structure population in case of absence of vaccine.

II. MATHEMATICAL FORMULA

In this section, an epidemiological model that describes the dynamics of *SIR* epidemic real world system represented by chicken pox disease, which speared within a stage structure population, is proposed to study. In order to formulate the dynamic equations for such a model the following assumptions are made.

ISSN 2348 – 8034
Impact Factor- 3.155

i. The population is divided into three compartments, namely susceptible, infected and removal due to the existence of SIR- type of disease.

ii. It is assumed that the susceptible population is a stage structure population and hence it divided into two classes, namely immature susceptible and mature susceptible.

iii. There is a constant number of the susceptible individuals entering to the system, which are representing the new born individuals, with recruitment rate $\Lambda > 0$.

iv. The disease can be transferred from the infected individual to the susceptible individual in both the susceptible classes, immature as well as mature, due to contact between the individuals in the infected compartment and those in the susceptible compartment with contact rates $\beta_1 > 0$ and $\beta_2 > 0$ for the immature susceptible and mature susceptible respectively. The disease can be transferred to the individuals in the susceptible classes through other external factors (such as insects, air, etc) with external incidence rates $\gamma_1 \geq 0$ and $\gamma_2 \geq 0$ for the immature susceptible and mature susceptible respectively.

v. The immature individuals in the susceptible compartment become mature with grown up rate $\alpha > 0$. However the individuals in all the compartments face natural death with mortality rate $d > 0$.

vi. Finally it is assumed that the individuals in the infected compartment may be recovered and gain permanent immunity with recover rate $\psi > 0$.

Let the densities at time t of immature susceptible, mature susceptible, infected and removal populations are represented by $S_1(t)$, $S_2(t)$, $I(t)$ and $R(t)$ respectively. Therefore the dynamics of the above described system can be represented mathematically with the following set of differential equations.

$$\begin{aligned}
\frac{dS_1}{dt} &= \Lambda - \alpha S_1 - \gamma_1 S_1 - \beta_1 S_1 I - dS_1 \\
\frac{dS_2}{dt} &= \alpha S_1 - \gamma_2 S_2 - \beta_2 S_2 I - dS_2 \\
\frac{dI}{dt} &= \gamma_1 S_1 + \beta_1 S_1 I + \gamma_2 S_2 + \beta_2 S_2 I - dI - \psi I \\
\frac{dR}{dt} &= \psi I - dR
\end{aligned} \tag{1}$$

with $S_1(0) > 0, S_2(0) > 0, I(0) \geq 0$ and $R(0) \geq 0$.

Clearly the interaction functions in the right hand side of system (1) are continuous and have continuous partial derivatives on $R_+^4 = \{(S_1, S_2, I, R) \in R^4 : S_1 \geq 0, S_2 \geq 0, I \geq 0, R \geq 0\}$ and hence they are Lipschtizaine. In addition all solutions of this model are uniformly bounded as shown in the following theorem. Therefore system (1) has a unique solution.

Theorem (1): All solutions of system (1) which initiate in R_+^4 are uniformly bounded.

Proof: Let $(S_1(t), S_2(t), I(t), R(t))$ be any solution of the system (1) with non-negative initial condition $(S_1(0), S_2(0), I(0), R(0))$, and let $N(t) = S_1(t) + S_2(t) + I(t) + R(t)$, then:

ISSN 2348 – 8034
Impact Factor- 3.155

$$\frac{dN}{dt}=\frac{dS_1}{dt}+\frac{dS_2}{dt}+\frac{dI}{dt}+\frac{dR}{dt}$$

which gives

$$\frac{dN}{dt}=\Lambda-dN$$

Now, by using Gronweall Lemma, it obtain that

$$N(t)\le\Lambda+\left(N_0-\frac{\Lambda}{d}\right)e^{-dt}$$

Therefore, $N(t)\le\frac{\Lambda}{d}$, as $t\to\infty$, that is independent of the initial conditions and hence the proof is complete.

III. THE EQUILIBRIUM POINTS AND BASIC REPRODUCTION NUMBER

It is easy to verify that the recovered population R is related with infected population only. Hence for fixed value of I, the value of R can be determined directly by solving the fourth equation in system (1). In fact, for $I=0$, then R approaches to zero asymptotically. However, for $I=I^*$, where $I^*>0$, then R approaches asymptotically to

$$R^*=\frac{\psi}{d}I^* \qquad (2)$$

Consequently, for simplifying, system (1) can be reduced to the following system, in which we can determine the value of I, by solving it, and then using Eq. (2).

$$\begin{aligned}\frac{dS_1}{dt}&=\Lambda-\alpha S_1-\gamma_1 S_1-\beta_1 S_1 I-dS_1\\ \frac{dS_2}{dt}&=\alpha S_1-\gamma_2 S_2-\beta_2 S_2 I-dS_2\\ \frac{dI}{dt}&=\gamma_1 S_1+\beta_1 S_1 I+\gamma_2 S_2+\beta_2 S_2 I-dI-\psi I\end{aligned} \qquad (3)$$

Now, straightforward computation shows that system (3) has at most two biologically feasible equilibrium points. These two points can be described as follows

In case of absence of disease ($I=0$), there is an equilibrium point represented by $E^o=\left(S_1^o,S_2^o,0\right)$ and it's called the disease free equilibrium point, where

$$S_1^o=\frac{\Lambda}{\alpha+d}\ \text{and}\ S_2^o=\frac{\alpha\Lambda}{d(\alpha+d)} \qquad (4a)$$

Clearly, the disease free equilibrium point exists uniquely in the interior of positive quadrant of S_1S_2- plane provided that the following condition holds

ISSN 2348 – 8034
Impact Factor- 3.155

$$\gamma_1 = \gamma_2 = 0 \tag{4b}$$

In case of existence of disease ($I > 0$), system (3) has an equilibrium point represented by $E^* = \left(S_1^*, S_2^*, I^*\right)$ and it's called endemic equilibrium point, where

$$S_1^* = \frac{\Lambda}{\alpha + \gamma_1 + \beta_1 I + d}, \quad S_2^* = \frac{\alpha\Lambda}{\left(\gamma_2 + \beta_2 I + d\right)\left(\alpha + \gamma_1 + \beta_1 I + d\right)} \tag{5a}$$

while I^* represents a positive root for the following equation

$$B_1 I^3 + B_2 I^2 + B_3 I + B_4 = 0 \tag{5b}$$

here $B_1 = -\left(d + \psi\right)\beta_1\beta_2 < 0$

$$B_2 = \Lambda\beta_1\beta_2 - \left(d + \psi\right)\left(\gamma_2\beta_1 + \alpha\beta_2 + \gamma_1\beta_2 + \beta_2 d + \beta_1 d\right)$$

$$B_3 = \left(\Lambda\gamma_1\beta_2 + \Lambda\gamma_2\beta_1 + \Lambda\beta_1 d + \alpha\Lambda\beta_2\right) - \left(d + \psi\right)\left(\alpha\gamma_2 + \gamma_1\gamma_2 + \gamma_2 d + \alpha d + \gamma_1 d + d^2\right)$$

$$B_4 = \Lambda\gamma_1\gamma_2 + \Lambda\gamma_1 d + \Lambda\alpha\gamma_2 > 0$$

Obviously, the endemic equilibrium point exists unique in the interior of positive octant of $S_1 S_2 I -$ space if and only if the following condition holds

$$B_2 < 0 \text{ or } B_3 > 0 \tag{5c}$$

It is well known that the basic reproduction number, which is denoted by R_0 , is an expected number of secondary cases produced, in a completely susceptible population, by a typical infective individual' [18]. Indeed, if $R_0 < 1$, then on average an infected individual produces less than one new infected individual over the course of its infectious period, and the infection cannot grow. Conversely, if $R_0 > 1$, then each infected individual produces, on average, more than one new infection, and the disease can invade the population.

It is clear that, in case of absence of disease, system (1) has a disease free equilibrium point $E^o = \left(S_1^o, S_2^o, 0\right)$ under the condition (4b). Therefore in order to compute R_0 , we first rearrange system (3) so that it becomes

$$\begin{aligned}
\frac{dI}{dt} &= \gamma_1 S_1 + \beta_1 S_1 I + \gamma_2 S_2 + \beta_2 S_2 I - dI - \psi I \\
\frac{dS_1}{dt} &= \Lambda - \alpha S_1 - \gamma_1 S_1 - \beta_1 S_1 I - dS_1 \\
\frac{dS_2}{dt} &= \alpha S_1 - \gamma_2 S_2 - \beta_2 S_2 I - dS_2
\end{aligned} \tag{6}$$

Let $X = \left(I, S_1, S_2\right)^T$, then system (6) can be written as

ISSN 2348 – 8034
Impact Factor- 3.155

$$\frac{dX}{dt} = f(X) - v(X)$$

where

$$f(X) = \begin{pmatrix} \gamma_1 S_1 + \beta_1 S_1 I + \gamma_2 S_2 + \beta_2 S_2 I \\ 0 \\ 0 \end{pmatrix}, \; v(X) = \begin{pmatrix} \psi I + dI \\ \alpha S_1 + \gamma_1 S_1 + \beta_1 S_1 I + dS_1 - \Lambda \\ \gamma_2 S_2 + \beta_2 S_2 I + dS_2 - \alpha S_1 \end{pmatrix}$$

Now straightforward computation shows that $Df(E^o) = \left(\frac{\partial f}{\partial X}\right)_{E^o}$ and $Dv(E^o) = \left(\frac{\partial v}{\partial X}\right)_{E^o}$ can be written as:

$$Df(E^o) = \begin{pmatrix} \beta_1 S_1^o + \beta_2 S_2^o & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} = \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix}$$

$$Dv(E^o) = \begin{pmatrix} \psi + d & 0 & 0 \\ \beta_1 S_1^o & \alpha + d & 0 \\ \beta_2 S_2^o & -\alpha & d \end{pmatrix} = \begin{pmatrix} V & 0 \\ J_1 & J_2 \end{pmatrix}$$

where $F = \left(\beta_1 S_1^o + \beta_2 S_2^o\right)_{1\times 1}$ and $V = (\psi + d)_{1\times 1}$, therefore the basic reproduction number represent is the spectral radius of the next generation matrix FV^{-1} [18], that means $\rho\left(FV^{-1}\right)$. Consequently, the basic reproduction number of system (3) can be written as

$$R_0 = \frac{\beta_1 S_1^o + \beta_2 S_2^o}{\psi + d} = \frac{\Lambda(d\beta_1 + \alpha\beta_2)}{d(\alpha + d)(\psi + d)} \tag{7}$$

IV. LOCAL STABILITY ANALYSIS

In this section, the local stability analysis of each equilibrium points is studied in terms of basic reproduction number as shown in the following theorems. First the Jacobian matrix of system (3) at the point $E = (S_1, S_2, I)$, can be written as

$$J(E) = \begin{pmatrix} -\alpha - \gamma_1 - \beta_1 I - d & 0 & -\beta_1 S_1 \\ \alpha & -\gamma_2 - \beta_2 I - d & -\beta_2 S_2 \\ \gamma_1 + \beta_1 I & \gamma_2 + \beta_2 I & \beta_1 S_1 + \beta_2 S_2 - d - \psi \end{pmatrix} \tag{8}$$

Theorem (2): Assume that the disease free equilibrium point E^o exists, then it is locally asymptotically stable when $R_0 < 1$, while E^o unstable if $R_0 > 1$

Proof: Clearly the Jacobian matrix of system (3) at E^o which is denoted by $J\left(E^o\right)$ can be written

ISSN 2348 – 8034
Impact Factor- 3.155

$$J(E^o)=\begin{pmatrix} -(\alpha+d) & 0 & -\beta_1 S_1^o \\ \alpha & -d & -\beta_2 S_2^o \\ 0 & 0 & \beta_1 S_1^o+\beta_2 S_2^o-(d+\psi) \end{pmatrix} \quad (9)$$

Accordingly, the eigenvalues of $J(E^o)$ are given by

$$\lambda_{S_1}^o=-(\alpha+d)<0, \lambda_{S_2}^o=-d<0, \lambda_I^o=\beta_1 S_1^o+\beta_2 S_2^o-(d+\psi)$$

Therefore, all the eigenvalues will be negative and hence the disease free equilibrium point is locally asymptotically stable if and only if $R_0=\frac{\beta_1 S_1^o+\beta_2 S_2^o}{\psi+d}<1$ or equivalently $\lambda_I^o<0$. However it is unstable saddle point if and only if $R_0=\frac{\beta_1 S_1^o+\beta_2 S_2^o}{\psi+d}>1$ or equivalently $\lambda_I^o>0$. Hence the proof is complete.

Theorem (3): The endemic equilibrium point E^* of system (3) is locally asymptotically stable if the following sufficient conditions hold

$$\beta_1 S_1^*+\beta_2 S_2^*<d+\psi \quad (10a)$$

$$\beta_1 S_1^* R_3<A_1(a_{11}a_{22}+R_1)-(a_{22}+a_{33})R_2 \quad (10b)$$

where $R_i; i=1,2,3$ are given in the proof.

Proof: The Jacobian matrix of system (3) at E^*, say $J(E^*)$, can be written

$$\begin{pmatrix} -(\alpha+\gamma_1+d)-\beta_1 I^* & 0 & -\beta_1 S_1^* \\ \alpha & -(\gamma_2+d)-\beta_2 I^* & -\beta_2 S_2^* \\ \gamma_1+\beta_1 I^* & \gamma_2+\beta_2 I^* & \beta_1 S_1^*+\beta_2 S_2^*-(d+\psi) \end{pmatrix}=(a_{ij}) \quad (11a)$$

Hence, the characteristics equation of $J(E^*)$ is given by

$$\lambda^3+A_1\lambda^2+A_2\lambda+A_3=0 \quad (11b)$$

where $A_1=-(a_{11}+a_{22}+a_{33})$, $A_2=a_{11}a_{22}+R_1+R_2$ and $A_3=-[a_{11}R_2+a_{13}R_3]$; while $R_1=a_{11}a_{33}-a_{13}a_{31}$, $R_2=a_{22}a_{33}-a_{23}a_{32}$ and $R_3=a_{21}a_{32}-a_{22}a_{31}>0$. Furthermore we have that:

$$\Delta=A_1A_2-A_3=A_1(a_{11}a_{22}+R_1)-(a_{22}+a_{33})R_2-\beta_1 S_1^* R_3$$

ISSN 2348 – 8034
Impact Factor- 3.155

Now, according to Routh-Hurwitz criterion E^* will be locally asymptotically stable provided that $A_1 > 0, A_3 > 0$ and $\Delta > 0$. It is clear that, condition (10a) guarantees that $R_1 > 0$ and $R_2 > 0$. Hence we obtain that $A_1 > 0$ and $A_3 > 0$. However, $\Delta > 0$ if and only if conditions (10a)-(10b) are satisfied. Hence the proof is complete.

Recall that, since $S_1^* < S_1^o$ and $S_2^* < S_2^o$ then satisfying condition (10a) don't necessary leads to $R_0 = \dfrac{\beta_1 S_1^o + \beta_2 S_2^o}{\psi + d} < 1$.

V. GLOBAL STABILITY ANALYSIS

In this section, the region of global stability (basin of attraction) of each equilibrium point of system (3) is presented as shown in the following theorems.

Theorem (4): Assume that, the disease free equilibrium point E^o is locally asymptotically stable. Then it is a globally asymptotically stable in the sub region of R_+^3 that satisfies the following sufficient conditions:

$$(\gamma_1 + \beta_1 I) S_1 \left(1 + S_1^o\right) + (\gamma_2 + \beta_2 I) S_2 \left(1 + S_2^o\right) < (d + \psi) I \tag{12a}$$

$$\alpha^2 < 4d(\alpha + d) \tag{12b}$$

Proof: Consider the following positive definite function

$$w_1(S_1, S_2, I) = \frac{\left(S_1 - S_1^o\right)^2}{2} + \frac{\left(S_2 - S_2^o\right)^2}{2} + I$$

Clearly, $w_1 : R_+^3 \to R$ is a continuously differentiable function such that $w_1\left(S_1^o, S_2^o, 0\right) = 0$ and $w_1(S_1, S_2, I) > 0$, $\forall (S_1, S_2, I) \neq \left(S_1^o, S_2^o, 0\right)$. Further, we have

$$\begin{aligned}\frac{dw_1}{dt} &= \left(S_1 - S_1^o\right)\left[\Lambda - \alpha S_1 - \gamma_1 S_1 - \beta_1 S_1 I - d S_1\right] \\ &\quad + \left(S_2 - S_2^o\right)\left[\alpha S_1 - \gamma_2 S_2 - \beta_2 S_2 I - d S_2\right] \\ &\quad + \left[\gamma_1 S_1 + \beta_1 S_1 I + \gamma_2 S_2 + \beta_2 S_2 I - dI - \psi I\right]\end{aligned}$$

Now, by doing some algebraic manipulation and using the condition (12a), we get

$$\begin{aligned}\frac{dw_1}{dt} &\le -\left[\sqrt{(\alpha + d)}\left(S_1 - S_1^o\right) - \sqrt{d}\left(S_2 - S_2^o\right)\right]^2 \\ &\quad + (\gamma_1 + \beta_1 I) S_1 \left(1 + S_1^o\right) + (\gamma_2 + \beta_2 I) S_2 \left(1 + S_2^o\right) - (d + \psi) I\end{aligned}$$

ISSN 2348 – 8034
Impact Factor- 3.155

Consequently, due to condition (12b), $\frac{dw_1}{dt} < 0$ is negative definite and hence w_1 is a Lyapunov function with respect to E^o in the region that satisfies the given condition. Thus E^o is a globally asymptotically stable and the proof is complete. ■

Theorem (5): Assume that the equilibrium point E^* is locally asymptotically stable. Then it is a globally asymptotically stable in the sub region of R_+^3 provided that

$$\beta_1 S_1 + \beta_2 S_2 < d + \psi \tag{13a}$$

$$\alpha^2 < q_{11} q_{22} \tag{13b}$$

$$q_{13}^2 < q_{11} q_{33} \tag{13c}$$

$$q_{23}^2 < q_{22} q_{33} \tag{13d}$$

where $q_{11} = \alpha + \gamma_1 + d + \beta_1 I^*$, $q_{13} = \beta_1 S_1 - \gamma_1 - \beta_1 I^*$, $q_{22} = \gamma_2 + d + \beta_2 I^*$,

$q_{23} = \beta_2 S_2 - \gamma_2 - \beta_2 I^*$, and $q_{33} = d + \psi - \beta_1 S_1 - \beta_2 S_2$.

Proof: Consider the following positive definite function

$$w_2(S_1, S_2, I) = \frac{(S_1 - S_1^*)^2}{2} + \frac{(S_2 - S_2^*)^2}{2} + \frac{(I - I^*)^2}{2}$$

It is easy to verify that $w_2(S_1, S_2, I) \in C^1(R_+^3, R)$, in addition, $w_2(S_1^*, S_2^*, I^*) = 0$ while $w_2(S_1, S_2, I) > 0$ $\forall (S_1, S_2, I) \in R_+^3$ and $(S_1, S_2, I) \neq (S_1^*, S_2^*, I^*)$. Further by determine the derivative of w_2 with respect to t and simplifying the resulting terms, we get

$$\begin{aligned}\frac{dw_2}{dt} = &-\left[\frac{q_{11}}{2}(S_1 - S_1^*)^2 - \alpha(S_1 - S_1^*)(S_2 - S_2^*) + \frac{q_{22}}{2}(S_2 - S_2^*)^2\right] \\ &-\left[\frac{q_{11}}{2}(S_1 - S_1^*)^2 + q_{13}(I - I^*)(S_1 - S_1^*) + \frac{q_{33}}{2}(I - I^*)^2\right] \\ &-\left[\frac{q_{22}}{2}(S_2 - S_2^*) + q_{23}(I - I^*)(S_2 - S_2^*) + \frac{q_{33}}{2}(I - I^*)^2\right]\end{aligned}$$

Clearly q_{33} is positive under condition (13a), while conditions (13b)-(13d) give us that

ISSN 2348 – 8034
Impact Factor- 3.155

$$\frac{dw_2}{dt} < -\left[\sqrt{\frac{d_{11}}{2}}\left(S_1 - S_1^*\right) - \sqrt{\frac{d_{22}}{2}}\left(S_2 - S_2^*\right)\right]^2 - \left[\sqrt{\frac{d_{11}}{2}}\left(S_1 - S_1^*\right) + \sqrt{\frac{d_{33}}{2}}\left(I - I^*\right)\right]^2$$
$$-\left[\sqrt{\frac{d_{22}}{2}}\left(S_2 - S_2^*\right) + \sqrt{\frac{d_{33}}{2}}\left(I - I^*\right)\right]^2$$

Obviously, $\frac{dw_2}{dt}$ is negative definite and hence w_2 is Layapunov function with respect to E^*. So E^* is globally asymptotically stable in the sub region that satisfies the given conditions.

VI. LOCAL BIFURCATION ANALYSIS

In this section, the occurrence of local bifurcation (such as transcritical, pitchfork and saddle-node) around equilibrium points is studied. Consider system (3), which can be rewritten as $\frac{dY}{dt} = H(Y)$, where $Y = (S_1, S_2, I)^T$ and $H(Y) = (h_1, h_2, h_3)^T$. Recall that, the general Jacobian matrix of system (3), say $DH = \frac{\partial H}{\partial Y} = J(S_1, S_2, I)$, is given by Eq. (8). Then straightforward computation shows that for any non-zero vector $V = (v_1, v_2, v_3)^T$ we get

$$D^2H(V,V) = \begin{pmatrix} -2\beta_1 v_1 v_3 \\ -2\beta_2 v_2 v_3 \\ 2\beta_1 v_1 v_3 + 2\beta_2 v_2 v_3 \end{pmatrix} \tag{14}$$

However $D^3H(V,V,V) = \mathbf{0}$, therefore pitchfork bifurcation can't be occur for system (3).

Theorem (6): The system (3) undergoes a transcritical bifurcation but not saddle node bifurcation at disease free equilibrium point E^o if and only if $R_0 = 1$.

Proof: Since the Jacobian matrix of system (3) at E^o, that given by Eq.(9), has zero eigenvalue (say $\lambda_I = 0$) provided that $R_0 = \frac{\beta_1 S_1^o + \beta_2 S_2^o}{(d+\psi)} = 1$ or equivalently at $\psi = \beta_1 S_1^o + \beta_2 S_2^o - d = \tilde{\psi}$. So the Jacobian matrix $J\left(E^o, \tilde{\psi}\right)$ becomes:-

$$\tilde{J} = J\left(E^o, \tilde{\psi}\right) = \begin{pmatrix} -(\alpha + d) & 0 & -\beta_1 S_1^o \\ \alpha & -d & -\beta_2 S_2^o \\ 0 & 0 & 0 \end{pmatrix}$$

Now, let $K = (k_1, k_2, k_3)^T$ be the eigenvector corresponding to the eigenvalue $\lambda_I = 0$. Thus $\tilde{J}K = \mathbf{0}$, which gives:

ISSN 2348 – 8034
Impact Factor- 3.155

$$K=\left(-\frac{\beta_1 S_1^o}{\alpha+d}k_3,-\frac{\alpha(\beta_1 S_1^o+\beta_2 S_2^o)+d\beta_2 S_2^o}{d(\alpha+d)}k_3,k_3\right)^T ;\ k_3 \text{ any nonzero real number}$$

Let $\Psi=(\psi_1,\psi_2,\psi_3)^T$ be the eigenvector associated with the eigenvalue $\lambda_I=0$ of the matrix $\tilde{J}^T$. Then from $\tilde{J}\Psi=\mathbf{0}$ we obtain

$\Psi=(0,0,\psi_3)^T$; with ψ_3 any nonzero real number.

Now, consider

$$\frac{\partial H}{\partial \psi}=H_\psi(Y,\psi)=\left(\frac{\partial h_1}{\partial \psi},\frac{\partial h_2}{\partial \psi},\frac{\partial h_3}{\partial \psi}\right)^T=(0,0,-I)^T$$

So, $H_\psi\left(E^o,\tilde{\psi}\right)=(0,0,0)^T$ and then $\Psi^T H_\psi\left(E^o,\tilde{\psi}\right)=0$

Thus, according to Sotomayor's theorem [19] the saddle-node bifurcation cannot occur. While the first condition of transcritical bifurcation is satisfied. Now, since

$$DH_\psi(Y,\psi)=\begin{pmatrix}0&0&0\\0&0&0\\0&0&-1\end{pmatrix}$$

Hence we obtain that

$$\Psi^T\left[DH_\psi\left(E^o,\tilde{\psi}\right)K\right]=-k_3\psi_3\neq 0$$

Moreover, according to Eq. (14) and the eigenvector K above we have

$$D^2H\left(E^o,\tilde{\psi}\right)(K,K)=\begin{pmatrix}-2\dfrac{\beta_1^2 S_1^o}{d(\alpha+d)^2}\left(\alpha(\beta_1 S_1^o+\beta_2 S_2^o)+d\beta_2 S_2^o\right)k_3^2\\ 2\dfrac{\beta_2}{d(\alpha+d)}\left(\alpha(\beta_1 S_1^o+\beta_2 S_2^o)+d\beta_2 S_2^o\right)k_3^2\\ -\left[\dfrac{2\beta_1^2 S_1^o}{(\alpha+d)}+\dfrac{2\beta_2}{d(\alpha+d)}\left(\alpha(\beta_1 S_1^o+\beta_2 S_2^o)+d\beta_2 S_2^o\right)\right]k_3^2\end{pmatrix}$$

Hence, it is obtain

$$\Psi^T\left[D^2H\left(E^o,\tilde{\psi}\right)(K,K)\right]=-\left[\frac{2\beta_1^2 S_1^o}{(\alpha+d)}+\frac{2\beta_2}{d(\alpha+d)}\left(\alpha(\beta_1 S_1^o+\beta_2 S_2^o)+d\beta_2 S_2^o\right)\right]k_3^2\psi_3\neq 0$$

ISSN 2348 – 8034
Impact Factor- 3.155

Thus, according to Sotomayor's theorem system (3) has transcritical bifurcation at E^o provided that $R_0 = 1$ or $\psi = \widetilde{\psi}$, which complete the proof.

Theorem (7): Assume that

$$\beta_1 S_1^* + \beta_2 S_2^* > d + \psi \tag{15a}$$

Then system (3) near the endemic equilibrium point E^* undergoes a saddle node bifurcation but not transcritical bifurcation, as the parameter ψ passes the following specific value

$$\psi^* = \beta_1 S_1^* + \beta_2 S_2^* - d - \mu \tag{15b}$$

here $\mu = \dfrac{a_{11}a_{23}a_{32} - a_{13}(a_{21}a_{32} - a_{22}a_{31})}{a_{11}a_{22}} > 0$

Proof: According to the Eq. (15b), it is easy to verify that the value of the determinant of $J(E^*)$, that given by A_3 in the characteristic equation Eq. (11b), will be vanish, that means $A_3 = 0$. Therefore, $J(E^*)$ has zero eigenvalue, say $\lambda^* = 0$, which makes E^* non hyperbolic point.

Consequently, the Jacobian matrix of system (3) at E^* with the parameter $\psi = \psi^*$ becomes

$J^* = J\left(E^*, \psi^*\right) = \left(a_{ij}\right)$; where a_{ij}, $i, j = 1,2,3$ are given in Eq. (11a) with $a_{33}^* = a_{33}(\psi^*) = \mu$.

Let $P = (p_1, p_2, p_3)^T$ be the eigenvector corresponding to the eigenvalue $\lambda^* = 0$. Thus $J^* P = \mathbf{0}$, which gives:

$P = (\xi_1 p_3, \xi_2 p_3, p_3)$; p_3 be any nonzero real number.

here, $\xi_1 = -\dfrac{a_{13}}{a_{11}} < 0, \xi_2 = \dfrac{a_{13}a_{21} - a_{11}a_{23}}{a_{11}a_{22}} < 0$

Let $W = (w_1, w_2, w_3)^T$ be the eigenvector associated with the eigenvalue $\lambda^* = 0$ of the matrix J^{*T}. Then we have $J^{*T} W = \mathbf{0}$,which gives

$W = (\zeta_1 w_3, \zeta_2 w_3, w_3)$; w_3 be any nonzero real number.

here $\zeta_1 = \dfrac{a_{21}a_{32} - a_{22}a_{31}}{a_{11}a_{22}} > 0; \zeta_2 = -\dfrac{a_{32}}{a_{22}} > 0$.

Now, consider

$$\frac{\partial H}{\partial \psi} = H_\psi(Y, \psi) = \left(\frac{\partial h_1}{\partial \psi}, \frac{\partial h_2}{\partial \psi}, \frac{\partial h_3}{\partial \psi}\right)^T = (0,0,-I)^T$$

ISSN 2348 – 8034
Impact Factor- 3.155

So, $H_{\psi}\left(E^*,\psi^*\right)=\left(0,0,-I^*\right)^T$, and hence $W^T H_{\psi}\left(E^*,\psi^*\right)=-I^* w_3 \neq 0$

Therefore, the transcritical bifurcation can't occur. While the first condition of the saddle-node bifurcation is satisfied.

Moreover, according to Eq. (14) and the eigenvector P above we have

$$D^2H\left(E^*,\psi^*\right)(P,P)=\begin{pmatrix} -2\beta_1\xi_1\xi_2 p_3^2 \\ -2\beta_2\xi_2 p_3^2 \\ 2\beta_1\xi_1 p_3^2+2\beta_2\xi_2 p_3^2 \end{pmatrix}$$

Hence, it is obtain

$$W^T\left[D^2H\left(E^*,\psi^*\right)(P,P)\right]=\left[2\beta_1\xi_1\left(1-\xi_2\zeta_1\right)+2\beta_2\xi_2\left(1-\zeta_2\right)\right]p_3^2 w_3$$

Now it is easy to verify that $W^T\left[D^2H\left(E^*,\psi^*\right)(P,P)\right]\neq 0$. Hence, the system (3) has a saddle-node bifurcation at E^* with parameter ψ^*.

In the following theorem, the possibility of occurrence of Hopf bifurcation near the endemic equilibrium point is investigated. Although, any parameter in system (3) can be a bifurcation parameter, the recover rate parameter (ψ) is choosing as a candidate bifurcation parameter which makes the endemic equilibrium point non hyperbolic point due to having two pure imaginary eigenvalues as this parameter passing through a specific value given in the following theorem.

Theorem (8): Assume that condition (10a) is satisfied and the recover rate parameter ψ passing through the following specific value

$$\hat{\psi}=\left(\beta_1 S_1^*+\beta_2 S_2^*\right)+\left(\frac{N_2}{2N_1}+\frac{1}{2N_1}\sqrt{N_2^2-4N_1N_3}\right)-d \qquad (16)$$

where $N_i; i=1,2,3$ are given in the proof. Then system (3) has two pure imaginary eigenvalues with the third real and negative. Further it still does not possess a Hopf bifurcation around the equilibrium point E^*.

Proof: According to the characteristic equation of the system (3) at E^* that is given by Eq.(11b). It is easy to verify that

$$\Delta=A_1A_2-A_3=0 \;\Leftrightarrow\; N_1a_{33}^2+N_2a_{33}+N_3=0 \qquad (17a)$$

where $N_1=-\left(a_{11}+a_{22}\right)>0$,

$$N_2=a_{13}a_{31}+a_{23}a_{32}-\left(a_{11}+a_{22}\right)^2$$

$$N_3=a_{13}\left(a_{11}a_{31}+a_{21}a_{32}\right)+a_{22}a_{23}a_{32}-a_{11}a_{22}\left(a_{11}+a_{22}\right)<0$$

ISSN 2348 – 8034
Impact Factor- 3.155

while $a_{ij}; i, j = 1,2,3$ are given in (11a). Consequently, the roots of Eq. (17a) are given by:

$$a_{33} = \frac{-N_2}{2N_1} \mp \frac{1}{2N_1}\sqrt{N_2^2 - 4N_1N_3}$$

Since, $a_{33} = \beta_1 S_1^* + \beta_2 S_2^* - (d + \psi) < 0$ by condition (10a), then we get

$$\beta_1 S_1^* + \beta_2 S_2^* - (d + \psi) = \frac{-N_2}{2N_1} - \frac{1}{2N_1}\sqrt{N_2^2 - 4N_1N_3}$$

Clearly as the parameter ψ passing through the value of $\hat{\psi}$ that given in Eq. (16), then Eq. (17a) is satisfied and hence we obtain $A_1 A_2 = A_3$ in the characteristic equation given by Eq. (11b). Therefore the characteristic equation can be rewritten as:-

$$P_3(\lambda) = (\lambda + A_1)(\lambda^2 + A_2) = 0 \qquad (17b)$$

which gives the following roots $\lambda_1 = -A_1 < 0$ under the sufficient condition (10a) and $\lambda_{2,3} = \mp i\sqrt{A_2} = \mp \rho_2(\hat{\psi})\, i$. Hence the first part of the theorem is proved.

Recall that the necessary and sufficient conditions for having Hopf bifurcation in system (3), near the endemic equilibrium point and $\psi = \hat{\psi}$, are the existence of two complex conjugate eigenvalues say $\lambda_{2,3} = \rho_1(\psi) \mp i\rho_2(\psi)$ with the third eigenvalue is real and negative, so that $\rho_1(\hat{\psi}) = 0$. This is satisfied from the first part. The second condition of having Hopf bifurcation is that $\left.\frac{d\rho_1}{d\psi}\right|_{\hat{\psi}} = \rho_1'(\hat{\psi}) \neq 0$.

Now in order to check the occurrence of Hopf bifurcation in system (3) the second condition should be satisfied otherwise there is no such bifurcation.

From Eq. (17b), it's clear that there is a range around $\hat{\psi}$ for which, the complex eigenvalues of system (3) are written in general as $\lambda_{2,3} = \rho_1(\psi) \mp i\rho_2(\psi)$. Then by substituting $\lambda_2 = \rho_1(\psi) + i\rho_2(\psi)$ into the equation (17b), and calculating the derivative with respect to the bifurcation parameter ψ, that is $P_3'(\lambda) = 0$ and then comparing the two sides of this equation with equating their real and imaginary parts, we get

$$\begin{aligned} E(\psi)\rho_1'(\psi) - F(\psi)\rho_2'(\psi) &= -G(\psi) \\ F(\psi)\rho_1'(\psi) + E(\psi)\rho_2'(\psi) &= -H(\psi) \end{aligned} \qquad (17c)$$

here $E(\psi) = 3(\rho_1(\psi))^2 + 2A_1(\psi)\rho_1(\psi) + A_2(\psi) - 3(\rho_2(\psi))^2$

$$F(\psi) = 6\rho_1(\psi)\rho_2(\psi) + 2A_1(\psi)\rho_2(\psi)$$

$$G(\psi) = (\rho_1(\psi))^2 A_1'(\psi) + A_2'(\psi)\rho_1(\psi) + A_3'(\psi) - A_1'(\psi)(\rho_2(\psi))^2$$

$$H(\psi) = 2\rho_1(\psi)\rho_2(\psi)A_1'(\psi) + A_2'(\psi)\rho_2(\psi)$$

ISSN 2348 – 8034
Impact Factor- 3.155

Solving the resulting linear system (17c) for the unknown $\rho_1'(\psi)$ and $\rho_2'(\psi)$, it is observe that:-

$$\rho_1'(\psi) = \frac{-(EG + FH)}{E^2 + F^2} \tag{17d}$$

Hence the second condition of Hopf bifurcation will be reduced to verifying that

$$E(\hat{\psi})G(\hat{\psi}) + F(\hat{\psi})H(\hat{\psi}) \neq 0 \tag{17e}$$

Straightforward computation shows as:-

$$A_1{}' = 1,\ A_2{}' = -(a_{11} + a_{22}),\ A_3{}' = (A_1 A_2)' = A_1(a_{11} + a_{22}) + A_2$$

Thus for $\psi = \hat{\psi}$ we have

$$E = -2A_2,\ F = 2A_1\sqrt{A_2},\ G = -A_1(a_{11} + a_{22}),\ H = -(a_{11} + a_{22})\sqrt{A_2}$$

Therefore, substituting in equation (17e), we get

$$EG + FH = 0$$

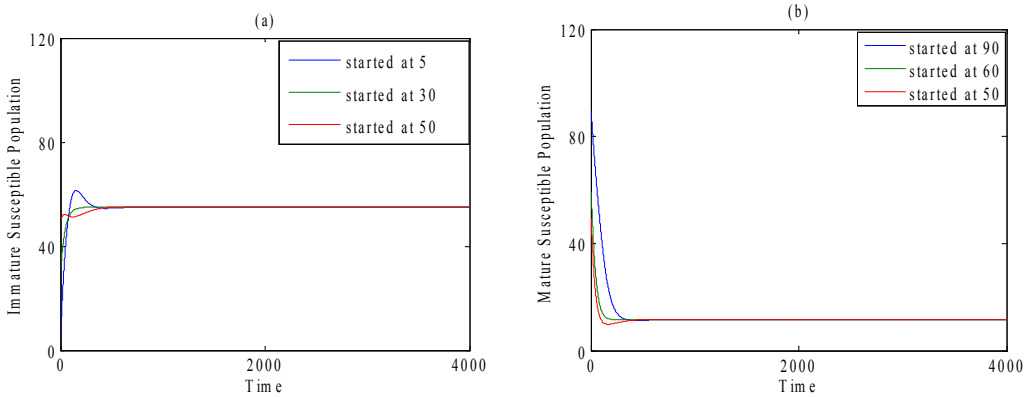
Hence, the Hopf bifurcation can not occur around the equilibrium point E^*. Thus the proof is complete.

VII. NUMERICAL SIMULATIONS

In this section, the global dynamics of system (1) is investigated numerically for different sets of initial values and different sets of parameters values. The objectives of such investigation are determine the effect of varying the parameters values and confirm our obtained results. It is observed that, for the following biologically feasible set of hypothetical parameters values:

$$\Lambda = 100, \alpha = 0.6, \gamma_1 = 0.001, \gamma_2 = 0.01, \beta_1 = 0.01,\ \beta_2 = 0.03,\ d = 0.4,\ \psi = 0.5 \tag{18}$$

The solution of system (1) approaches asymptotically to the endemic equilibrium point $E^* = (55.06,\ 11.57,\ 81.49,\ 101.86)$ as shown in Fig. (1), started from different sets of initial points.



ISSN 2348 – 8034
Impact Factor- 3.155

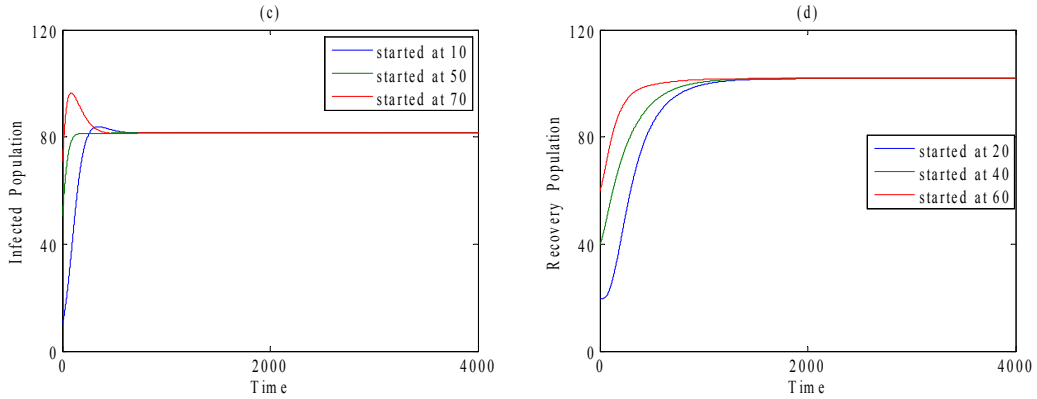
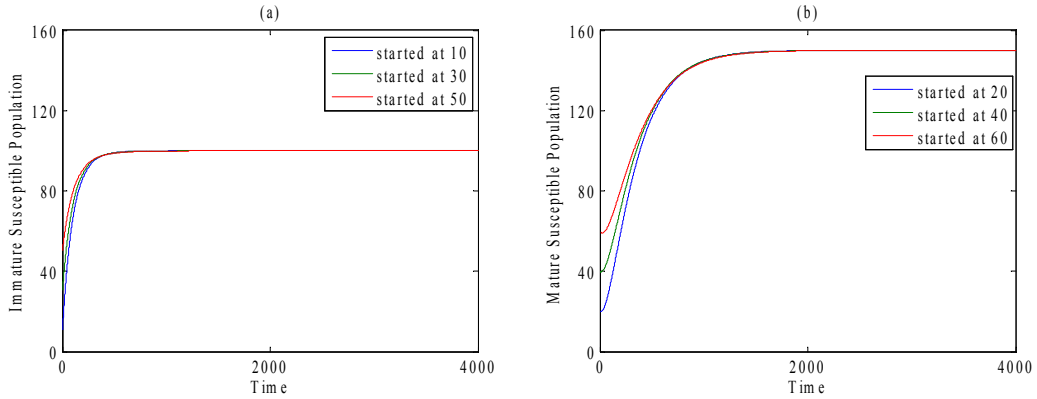


Fig. 1: Globally asymptotically stable positive equilibrium point of system (1) for the parameters set (18), started from different sets of initial point. (a) Trajectory of immature susceptible population. (b) Trajectory of mature susceptible population. (c) Trajectory of infected population. (d) Trajectory of removal population.

Clearly Fig. (1) confirms our obtained analytical results regarding to existence of a globally asymptotically stable positive equilibrium point when the parameters values are satisfying $R_o > 1$.

On the other hand, system (1) for the following set of hypothetical data approaches asymptotically to the DFE as shown in Figure 2:

$$\Lambda = 100,\ \alpha = 0.6,\ \gamma_1 = \gamma_2 = 0,\ \beta_1 = 0.001,\ \beta_2 = 0.003,\ d = 0.4,\ \psi = 0.5 \qquad (19)$$



ISSN 2348 – 8034
Impact Factor- 3.155

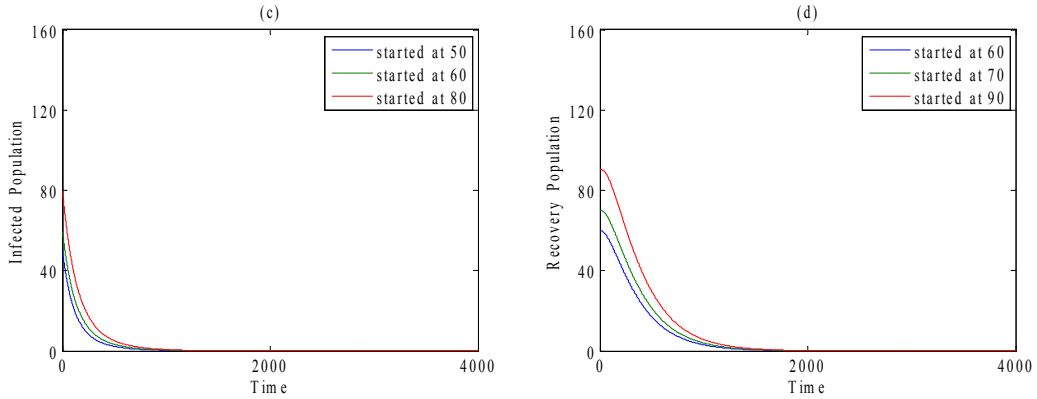


Fig. 2: Globally asymptotically stable DFE of system (1) for the parameters set (19), started from different sets of initial point. (a) Trajectory of immature susceptible population. (b) Trajectory of mature susceptible population. (c) Trajectory of infected population. (d) Trajectory of removal population.

It is easy to verify that for the data (19), we have $R_o = 0.66 < 1$, and the solution approaches to $E^o = (100, 150, 0, 0)$.

Now in order to investigate the effect of varying one parameter value at a time on the dynamical behavior of system (1), the following results are observed.

Varying of the parameters values $(\alpha, \gamma_1, \gamma_2, \beta_1, \beta_2, d)$ don't have qualitative effects on the dynamics of system (1) rather than that they have quantitative effects on the value of positive equilibrium point.

For the parameters values given in Eq.(18) with varying Λ in the range $\Lambda < 16$ and $\gamma_1 = \gamma_2 = 0$, the solution of the system (1) approaches asymptotically to disease free equilibrium point $E^o = (10, 15, 0, 0)$ as shown in the typical figure given by Fig. 3 below.

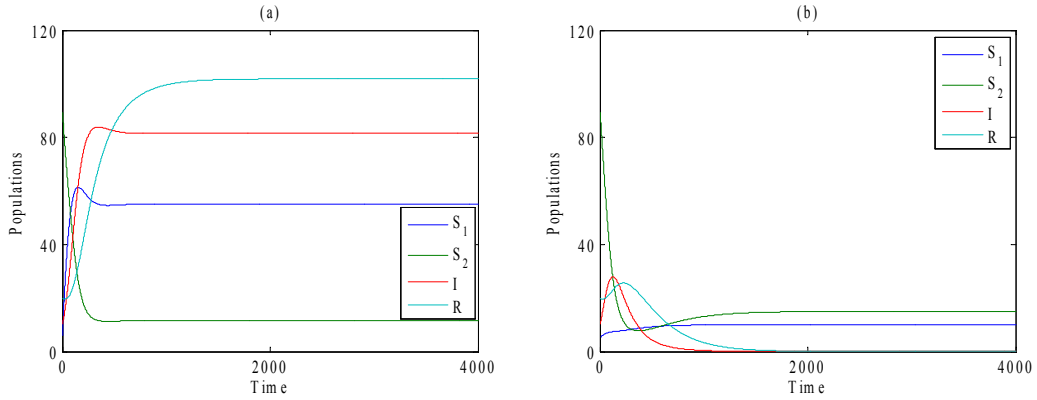


Fig. 3: Time series of the solution of system (1) for the data (18) with different values of Λ. (a) Globally asymptotically stable endemic equilibrium point for $\Lambda = 100$ (b) Globally asymptotically stable disease free equilibrium point E^o for $\Lambda = 10$.

ISSN 2348 – 8034
Impact Factor- 3.155

According to the Fig. (3), it's clear that the solution of system (1) approaches asymptotically to the disease free equilibrium point. Moreover, for the parameters values given in Eq.(18) with $\gamma_1 = \gamma_2 = 0$ and $\psi > 5.1$ the solution of system (1) approaches asymptotically to $E^o = (100,150,0,0)$ as shown in the typical figure that given by Fig. (4).

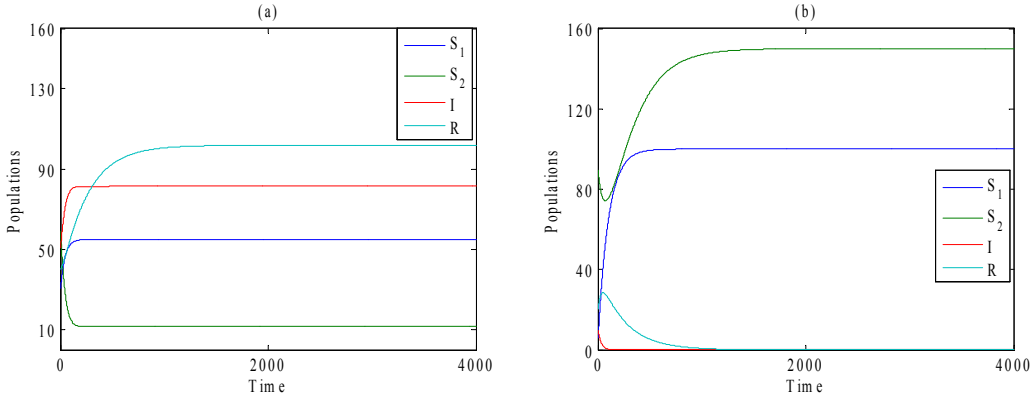


Fig. 4: Time series of the solution of system (1) for the data (18) with different values of ψ. (a) Globally asymptotically stable endemic equilibrium point for $\psi = 0.5$ (b) Globally asymptotically stable disease free equilibrium point E^o for $\psi = 5.5$.

According to the Fig. (4), it is clear that as the ψ increase the solution of system (1) approaches asymptotically to the $E^o = (100,150,0,0)$ equilibrium point.

VIII. CONCLUSIONS AND DISCUSSION

In this paper the effects of Chicken pox disease are formulated mathematically and studied analytically as well as numerically. The objective of this study is to understand the effects of all factors, which helping the spread of this type of disease and hence get the capability of control the disease.

The boundedness of the system has been discussed. The existence conditions of all possible equilibrium points of the system are established. All possible equilibrium points with their local and global stability are investigated. The qualitative dynamical behavior as a function of varying the parameters values is studied analytically as well as numerically. Finally, for the biologically feasible set of hypothetical data as given in Eq. (18), the system (1) is solved numerically and the obtained results are explained in some typical figures and we will summarize as follows.

1- System (1) does not have periodic dynamics; instead of that it approaches either to the disease free equilibrium point or else to endemic equilibrium point depending on the value of reproduction number.

2- For the set of hypothetical parameters values given by Eq.(18), the system (1) approached asymptotically to the global stable endemic equilibrium point E^*

3- Decreasing the natural birth rate of the susceptible Λ below the specific value causes destabilizing to the endemic equilibrium point and the trajectories of system (1) approached asymptotically to the disease free equilibrium point, which indicate to occurrence of bifurcation. Otherwise the system still has a globally asymptotically stable endemic equilibrium point.

ISSN 2348 – 8034
Impact Factor- 3.155

4- Increasing the recovery rate ψ above a specific value causes bifurcation in the system and the trajectory transferred from the endemic equilibrium point to the disease free equilibrium point asymptotically. Otherwise the system still approaches to the endemic point.

5- Finally all the other parameters have quantitative change but note qualitative change on the stability of the positive equilibrium point.

REFERENCES

[1] American Academy of Pediatrics (2003) Active immunization. In: Pickering LK (ed.)
[2] Red Book: 2003 Report of the Committee on Infectious Diseases, 26th edition. Elk Grove Village, IL: American Academy of Pediatrics.
[3] Seward JF, Watson BM, Peterson CL, et al. Varicella disease after introduction of varicella vaccine in the United States, 1995–2000. *JAMA* 2002;287:606–11.
[4] Annunziato PW and Gershon AA (2000) Primary vaccination against varicella. In: Arvin AM and Gershon AA (eds) Varicella-zoster virus Cambridge: Cambridge University Press.
[5] Balfour HH, Rotbart HA, Feldman S et al. (1992) Aciclovir treatment of varicella in otherwise healthy adolescents. The Collaborative Aciclovir Varicella Study Group. J Pediatr 120: 627–33.
[6] Leung J, Harpaz R, Molinari NA, et al. Herpes zoster incidence among insured persons in the United States, 1993-2006; evaluation of impact of varicella vaccination. *Clin Infect Dis* 2011;52:332-40.
[7] W.O. Kermack, A.G. McKendrick, Contributions to the mathematical theory of epidemics (part I), Proc. Roy. Soc. Ser. A 115 (1927) 700–721.
[8] Kribs-Zaleta, C.M., Velasco-Hern´andez, J.X.,(2000). A simple vaccination model with multiple endemic states. *Math. Biosci*. 164: 183-201.
[9] Arino, J., Mccluskey, C.C., van den Driessche, P.,(2003). Global results for an epidemic model with vaccination that exhibits backward bifurcation. *SIAM J. APPL*. MATH. 64: 260-276.
[10] Kribs-Zaleta, C.M., Martcheva, M.,(2002). Vaccination strategies and backward bifurcation in an age-since-infection structured model. *Math. Biosci*.177-178: 317-332.
[11] Alexander, M.E., Bowman, C., Moghadas, S.M., Summers, R., Gumel, A.B., Sahai, B.M.,(2004). A vaccination model for transmission dynamics of influenza. *SIAM J. Appl. Dyn. Syst*. 3:503-524.
[12] Shim, E.,(2004). An epidemic model with immigration of infectives and vaccination. M.sc. thesis. B.Sc. mathematics, The University of Britsh Columbia.
[13] d'Onofrio, A., Manfredi, P., Salinelli, E.,(2007). Vaccinating behaviour, information, and the dynamics of SIR vaccine preventable diseases. Theor. Popul. *Biol.* 71:301-317.
[14] W.G. Aiello, H.I. Freedman, A time-delay model of single-species growth with stage structure, Math. Biosci. 101 (1990) 139–153.
[15] J. Cui, L. Chen, W. Wang, The effect of dispersal on population growth with stage-structure, Comput. Math. Appl. 39 (2000) 91–102.
[16] Cui, J. and Song, X. 2004. Permanence of predator-prey system with stage structure, Discrete and Continuous Dynamical System. Series B, 4(3), pp:547-554.
[17] Chen, F. 2006. Permanence of periodic Holling type predator-prey system with stage structure for prey, Applied Mathematics and Computation, 182(2), pp:1849-1860.
[18] Diekmann, O., Heesterbeek, J. A. P. & Metz, J. A. J. 1990 On the definition and computation of the basic reproduction Ratio R0 in models for infectious diseases in heterogeneous populations. J. Math. Biol. 28, 365–382.
[19] L. Perko, Differential Equation and Dynamical Systems, third Edition, New York, Springer-Verlag Inc, 2001.