This model also reveals that there is a limit to the number of cell division a T cell clone can undergo, and that the progeny of clones that have expanded massively during a primary immune response are more prone. Thorley-Lawson (2001) in "Conceptual Model Of How Epstein-Barr Virus Establishes And Maintains Persistent Infection" developed a model that reveals that persistent infection by Epstein Barr virus appears to be steady-state equilibrium between host and virus with continuous shedding of infectious virus into the saliva, stable levels of infected cells in the blood and lymph nodes and a constitutively active antiviral immune response. The model also explains all the major features of Epstein Barr virus biology.

Giao and Fredrick (2012) in "A Mathematical Model Of Evolution and Coexistence Of Epstein Barr Virus Infections In Human". used these mathematical models to understand why Epstein Barr virus infects epithelial cells when B cells serve as a stable refuge for the virus and how switching between infecting each cell type affects virus persistence and shedding. They also proposed a mathematical model to describe the regulation of Epstein Barr virus infection within a host. This model was used to study the effects of parameter values on optimal viral strategies for transmission, persistence, and intrahost competition. They applied the results of the within-host model, and derived a model of Epstein-Barr virus dynamics in a homogeneous population of hosts that includes super infection. They used this model to study the conditions necessary for invasion and coexistence of various viral strategies at the population level. They concluded that the optimal strategy to maximize transmission is for viruses to infect epithelial cells, but the optimal strategy for maximizing intrahost competition is for viruses to mainly infect B cells.

Daniel Bernoulli (1976) and (Hethcote, 2000). Developed models to defend the practice of inoculation against smallpox. The calculations from this model showed that universal inoculation against smallpox would increase the life expectancy from 26 years 7 months to 29 years 9 months (Bernoulli & Blower, 2004). Daniel Bernoulli's work preceded our modern understanding of germ theory, and it was not until the research of Ronald Ross into the spread of malaria, that modern theoretical epidemiology began. Anderson (1991) in "A Mathematical Model of Transmission Dynamics And Control Of Infectious Disease Agents". revealed that the concept of an infection's basic reproductive rate, R_O, is central to an understanding of the population biology of infectious disease agents.

The parameter, R_O, measures the ability of an infection to give rise to secondary cases, and its value is determined by a variety of factors specific to the biology of the disease agent and that of its host.The model also revealed that the condition, $R_O=1$, defines a transmission threshold below which a disease is unable to maintain itself within the community. The value of R_O can be estimated from horizontal or longitudinal epidemiological studies of the prevalence and intensity of infection in various age classes of the population, measurement of this parameter provides a means of estimating the proportion of the community that must be immunized or receive treatment, either to eradicate an infection or to reduce its prevalence to a defined level.

Herbert et al (1986) in "Deterministic Models For Epidemics Which Occur Quickly And Long-term Endemic Diseases" considered births and deaths were considered. They formulated contact-rate matrices in terms of activity levels and subpopulation sizes by using a proportionate mixing assumption. They also presented the methods for estimating epidemic and endemic parameters in both homogeneous and heterogeneous populations.

3. ASSUMPTION

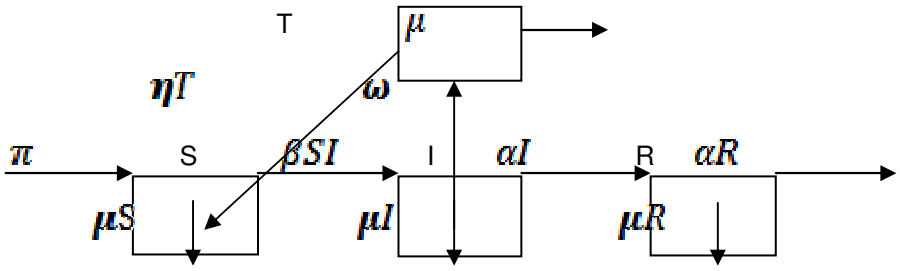
Due to the nature of the virus, and the SITR model we are using, we now make the following assumptions;

1. The only way a person can leave the susceptible class is to become infected.
2. The only way a person can leave the infected class is to be treated or removed (either by natural death or by the virus).
3. Age, sex, social status, and race do not affected the probability of being infected.
4. There is no inherited immunity
5. The member of the population mix homogeneously (have the same interactions with one another to the same degree).

4 FORMULATION OF THE MODEL

The Schematic Diagram

Schematic diagram is a representation of a system using abstract, graphic symbols rather than realistic pictures. Below is a schematic diagram for our mathematical model on Epstein Barr virus.



$$S(t) + I(t) + T(t) + R(t) = N$$

5. DESCRIPTION OF VARIABLES AND ASSOCIATED PARAMETERS

The table below depicts Variables and Associated Parameters

Table 1: **Description of Variables & Associated Parameters**

S(t)	susceptible population at a particular time
I(t)	infected population at a particular time
T(t)	treated population at a particular time
R(t)	removed population at a particular time
µI	natural death and infected population at a particular time
µR	natural death and removed class at a particular time
π	Human recruitment rate
B	rate at which susceptible class become infected
A	rate at which infected humans move to removed class
αR	rate at which untreated people are removed by the virus
N	the total population

6. THE DERIVATION OF ORDINARY DIFFERENTIAL EQUATIONS FOR THE MODEL

$$\frac{dS}{dt} = \pi - \beta SI - \mu S + \eta T \ldots\ldots\ldots\ldots\ldots\ldots (1)$$

$$\frac{dI}{dt} = \beta SI - \alpha I - \omega I - \mu I \ldots\ldots\ldots\ldots\ldots\ldots (2)$$

$$\frac{dT}{dt} = \omega I - \mu T - \eta T \ldots\ldots\ldots\ldots\ldots\ldots\ldots (3)$$

$$\frac{dR}{dt} = \alpha I - \mu R - \alpha R \ldots\ldots\ldots\ldots\ldots\ldots\ldots (4)$$

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = \frac{dR}{dt} = 0$$

Therefore;

$$S' = \pi - \beta SI - \mu S + \eta T = 0$$

$$I' = \beta SI - \alpha I - \omega I - \mu I = 0$$

$$T' = \omega I - \mu T - \eta T = 0$$

$$R' = \alpha I - \mu R - \alpha R = 0$$

$$The incidence rate is given by: \frac{rate of susceptible class becoming infected}{total population} = \frac{\beta SI}{N}$$

7. DISEASE FREE EQUILIBRIUM OF THE MODEL (DFE)

The disease free equilibrium is given as;

$$(S, I, T, R) = (x, v, y, z) = (x, 0, 0, 0)$$

This implies that at disease free equilibrium;

$$I = T = R = 0 \Longrightarrow v = y = z = 0$$

Substituting the values of I, T, and R in the equations above we have the disease free equilibrium as;

$$(S, I, T, R) = (x, v, y, z) = \left(\frac{\pi}{\mu}, 0, 0, 0\right)$$

8. THE DISEASE ENDEMIC EQUILIBRIUM OF THE MODEL (DEE)

Let;

$$(S, I, T, R) = (x, v, y, z)$$

At the disease endemic equilibrium, S,I,T,R exist

The equations are;

$$S' = \pi - \beta xv - \mu x + \eta y = 0$$

$$I' = \beta xv - \alpha v - \omega v - \mu v = 0$$

$$T' = \omega v - \mu y - \eta y = 0$$

$$R' = \alpha v - \mu z - \alpha z = 0$$

The disease endemic equilibrium of the model is given as;

$$(S, I, T, R) = (x, v, y, z) = \left[\frac{\alpha+\omega+\mu}{\beta}, \frac{\mu(\mu+\eta)(\alpha+\mu+\omega)-\pi\beta(\mu+\eta)}{\beta\eta\omega}, \frac{\mu(\alpha+\omega+\mu)-\pi\beta}{\beta\eta}, \frac{\alpha\mu(\mu+\eta)(\alpha+\mu+\omega)-\alpha\pi\beta(\mu+\eta)}{\beta\eta\,\omega(\mu+\alpha)}\right]$$

9. THE LOCAL STABILITY OF DISEASE FREE EQUILIBRIUM OF THE MODEL

To establish the local stability of the model, we first get the basic reproduction (R_O) using the jacobian matrix.

The equations are given as;

$$S' = \pi - \beta xv - \mu x + \eta y = 0 \ldots\ldots\ldots\ldots\ldots\ldots\ldots (A)$$

$$I' = \beta xv - \alpha v - \omega v - \mu v = 0 \ldots\ldots\ldots\ldots\ldots\ldots\ldots (B)$$

$$T' = \omega v - \mu y - \eta y = 0 \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (C)$$

$$R' = \alpha v - \mu z - \alpha z = 0 \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (D)$$

The jacobian matrix of the above equations is given as;

$$J = \begin{bmatrix} \frac{\partial A}{\partial x} & \frac{\partial A}{\partial v} & \frac{\partial A}{\partial y} & \frac{\partial A}{\partial z} \\ \frac{\partial B}{\partial x} & \frac{\partial B}{\partial v} & \frac{\partial B}{\partial y} & \frac{\partial B}{\partial z} \\ \frac{\partial C}{\partial x} & \frac{\partial C}{\partial v} & \frac{\partial C}{\partial y} & \frac{\partial C}{\partial z} \\ \frac{\partial D}{\partial x} & \frac{\partial D}{\partial v} & \frac{\partial D}{\partial y} & \frac{\partial D}{\partial z} \end{bmatrix} = \begin{bmatrix} -(\beta v + \mu) & -\beta x & \eta & 0 \\ \beta v & \beta x - (\alpha + \mu + \omega) & 0 & 0 \\ 0 & \omega & -(\mu + \eta) & 0 \\ 0 & \alpha & 0 & -(\mu + \alpha) \end{bmatrix}$$

The disease free equilibrium is given as;

$$(S, I, T, R) = (x, v, y, z) = \left(\frac{\pi}{\mu}, 0, 0, 0\right)$$

Substituting the values of S, I, T and R into the jacobian matrix we have;

$$J = \begin{bmatrix} -\mu & -\beta\frac{\pi}{\mu} & \eta & 0 \\ 0 & \beta\frac{\pi}{\mu} - (\alpha+\mu+\omega) & 0 & 0 \\ 0 & \omega & -(\mu+\eta) & 0 \\ 0 & \alpha & 0 & -(\mu+\alpha) \end{bmatrix}$$

$m_1 = \frac{\pi}{\mu}, k_1 = (\alpha+\mu+\omega), m_2 = (\mu+\eta), m_3 = (\mu+\alpha)$ without loss of generality.

The characteristic equation is given as $|J - \lambda I| = 0$

Therefore;

$$|J - \lambda I| = \begin{bmatrix} -\mu & -\beta m_1 & \eta & 0 \\ 0 & \beta m_1 - k_1 & 0 & 0 \\ 0 & \omega & -m_2 & 0 \\ 0 & \alpha & 0 & -m_3 \end{bmatrix} - \lambda \begin{bmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{bmatrix} = 0$$

$\lambda_4 = -[(\mu+\alpha)] < 0,\ \lambda_3 = -[(\mu+\eta)] < 0,\ \lambda_1 = -\mu < 0$

$\lambda_2 = \beta m_1 - k_1$

If $\lambda_2 < 0 \Rightarrow \beta m_1 - k_1 < 0$

But $m_1 = \frac{\pi}{\mu}$ and $k_1 = (\alpha+\mu+\omega)$

Therefore;

$$R_{(0)} = \frac{\beta\pi}{\mu(\alpha+\mu+\omega)}$$

(The basic reproduction number at the disease free equilibrium)

If the basic reproduction number, $R_{(0)}$ is less than one (i.e $R_{(0)} < 1$), we therefore conclude that the disease free equilibrium of the model is locally asymptotically stable.

10. THE LOCAL STABILITY OF DISEASE ENDEMIC EQUILIBRIUM OF THE MODEL

The disease endemic equilibrium of the model is given as;

$$(S,I,T,R) = (x,v,y,z) = \left[\frac{\alpha+\omega+\mu}{\beta}, \frac{\mu(\mu+\eta)(\alpha+\mu+\omega)-\pi\beta(\mu+\eta)}{\beta\eta\omega}, \frac{\mu(\alpha+\omega+\mu)-\pi\beta}{\beta\eta}, \frac{\alpha\mu(\mu+\eta)(\alpha+\mu+\omega)-\alpha\pi\beta(\mu+\eta)}{\beta\eta\,\omega(\mu+\alpha)}\right]$$

The Jacobian matrix is given as;

$$J = \begin{bmatrix} \frac{\partial A}{\partial x} & \frac{\partial A}{\partial v} & \frac{\partial A}{\partial y} & \frac{\partial A}{\partial z} \\ \frac{\partial B}{\partial x} & \frac{\partial B}{\partial v} & \frac{\partial B}{\partial y} & \frac{\partial B}{\partial z} \\ \frac{\partial C}{\partial x} & \frac{\partial C}{\partial v} & \frac{\partial C}{\partial y} & \frac{\partial C}{\partial z} \\ \frac{\partial D}{\partial x} & \frac{\partial D}{\partial v} & \frac{\partial D}{\partial y} & \frac{\partial D}{\partial z} \end{bmatrix} = \begin{bmatrix} -(\beta v+\mu) & -\beta x & \eta & 0 \\ \beta v & \beta x-(\alpha+\mu+\omega) & 0 & 0 \\ 0 & \omega & -(\mu+\eta) & 0 \\ 0 & \alpha & 0 & -(\mu+\alpha) \end{bmatrix}$$

Therefore imputing the values of $x, v, y and z$ into the Jacobian matrix, we have;

$$J = \begin{bmatrix} \beta\left[\frac{\mu(\mu+\eta)(\alpha+\mu+\omega)-\pi\beta(\mu+\eta)}{\beta\eta\omega}\right]-\mu & -\beta\frac{\alpha+\omega+\mu}{\beta} & \eta & 0 \\ \beta\left[\frac{\mu(\mu+\eta)(\alpha+\mu+\omega)-\pi\beta(\mu+\eta)}{\beta\eta\omega}\right] & \beta\frac{\alpha+\omega+\mu}{\beta}-(\alpha+\mu+\omega) & 0 & 0 \\ 0 & \omega & -(\mu+\eta) & 0 \\ 0 & \alpha & 0 & -(\mu+\alpha) \end{bmatrix}$$

For conveniences, let;

$$m_1 = \frac{\mu(\mu+\eta)(\alpha+\mu+\omega)-\pi\beta(\mu+\eta)}{\beta\eta\omega}, m_2 = (\alpha+\omega+\mu), m_3 = (\mu+\eta), m_4 = (\mu+\alpha)$$

The characteristic equation is given as; $|J-\lambda I| = 0$

thus;

$$|J-\lambda I| = \begin{bmatrix} \beta m_1-\mu & -m_2 & \eta & 0 \\ \beta m_1 & 0 & 0 & 0 \\ 0 & \omega & -m_3 & 0 \\ 0 & \alpha & 0 & -m_4 \end{bmatrix} - \lambda\begin{bmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{bmatrix} = 0$$

$$\lambda_4 = -[(\mu+\alpha)] < 0,\ \lambda_3 = -[(\mu+\eta)] < 0,\ \lambda_1 = \beta m_1 - \mu$$

Therefore if $\lambda_1 > 0$, It implies that $\beta m_1 - \mu > 0$

But $m_1 = \frac{\mu(\mu+\eta)(\alpha+\mu+\omega)-\pi(\mu+\eta)}{\beta\eta\omega}$

Therefore;

$$R_{(0)} = \frac{\beta[\mu(\mu+\eta)(\alpha+\mu+\omega)-\pi(\mu+\eta)]}{\beta\eta\omega\mu}$$

(The basic reproduction number at the disease endemic equilibrium)

If our basic reproduction number is greater than one (i.e. $R_{(0)} > 1$), therefore we conclude that the disease endemic equilibrium of the model is locally unstable.

This implies that the virus will spread within the population.

From the above equation, it is obvious that if infection rate, β, is equal to zero, there is no reproduction in the system.

ANALYSIS OF THE MODEL

Based on the nature of Epstein Barr virus we obtained the table of values below.

Table 2: Table of Values

PARAMETER	DISCRIPTION	VALUES
π	Birth rate	0.3
μ	Natural death rate	0.4
β	Infection rate	0.3
ω	Treatment rate	0.2
α	Removal rate	0.3
η	Rate of susceptible after treatment	0.6

We will now use our parameter values to establish the local stability of the model using the basic reproduction gotten at the disease free equilibrium and disease endemic equilibrium.
At the disease free equilibrium, the basic reproduction number is given as;

$$R_{(0)} = \frac{\beta\pi}{\mu(\alpha+\mu+\omega)}$$

Therefore, substituting the parameter values into above relation we have;

$$R_{(0)} = \frac{(0.3)(0.3)}{0.4(0.3+0.4+0.2)} = 0.25 < 1$$

Since our basic reproduction number, $R_{(0)}$, at the disease free equilibrium is less than one ($i.e R_{(0)} < 1$), we therefore conclude that the disease free equilibrium of the model is asymptotically stable.

Also, the basic reproduction number at the disease endemic equilibrium is given as;

$$R_{(0)} = \frac{\beta[\mu(\mu+\eta)(\alpha+\mu+\omega)-\pi(\mu+\eta)]}{\beta\eta\omega\mu}$$

Therefore, by substituting the values of the parameter into the above relation, we have;

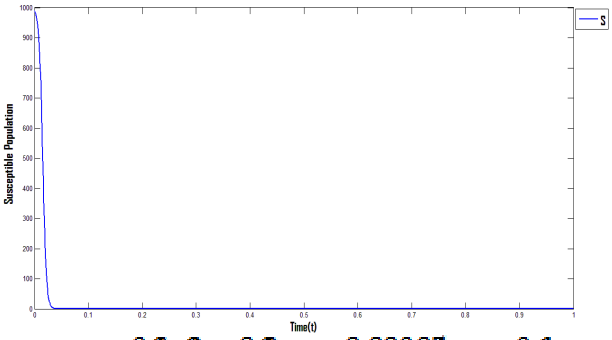
$$R_{(0)} = \frac{0.3[0.4(0.4+0.6)(0.3+0.4+0.2)-(0.3)(0.4+0.6)]}{(0.3)(0.6)(0.2)(0.4)} = 1.25 > 1$$

Since our basic reproduction number, $R_{(0)}$ at the disease endemic equilibrium is greater than one $(i.\, eR_{(0)} > 1)$, therefore we conclude that the disease endemic equilibrium of the model is unstable.

This shows that the virus will spread within the population.

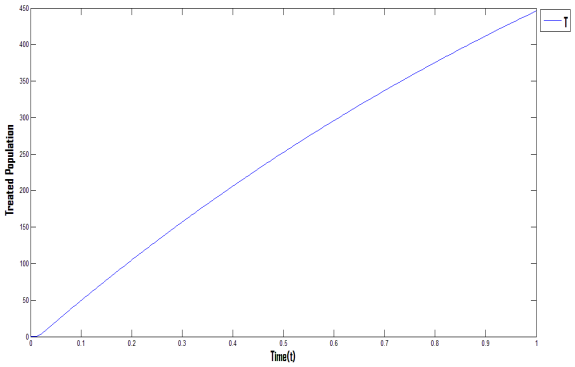
11. THE GRAPHICAL SOLUTION OF THE MODEL

Figure1:Graph Of Susceptible Population Against Time (T)



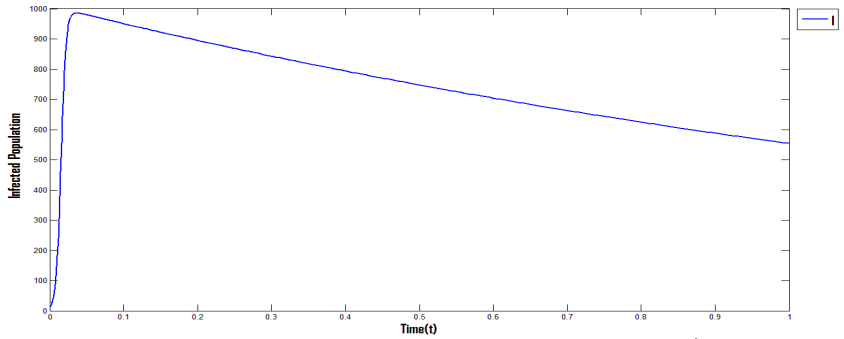
$\pi = 0.3, \beta = 0.3, \mu = 0.00005, \eta = 0.6$

Figure1:Graph Of Susceptible Population against Time (T)



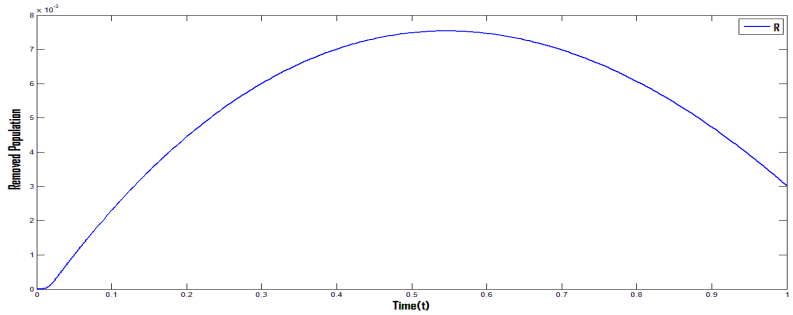
$\omega = 0.4, \mu = 0.00005, \eta = 0.6$

Fig 2: The Graph of Treated Population against Time (t)



$\beta = 0.3, \alpha = 0.00003, \omega = 0.4, \mu = 0.00005$

Figure 3: The Graph Infected Population against Time (t)



$\alpha = 0.00003, \mu = 0.00005$

Figure 4: The Graph of The Removed Population Against Time

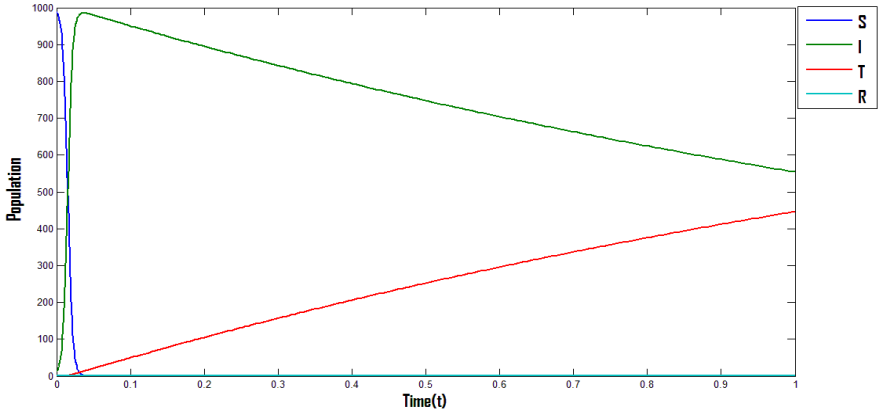


Figure 5: The Combined Graph against Time (t)

12. INTERPRETATION OF GRAPHICAL RESULTS

We discovered that Epstein Barr virus is a communicable disease that can transmit from one person to another, as the susceptible population dropped drastically; as we can see from figure one. This may be due to the fact that the virus has the ability to remain in the host for life without causing any disease; therefore the virus is not easily detected in a population. So, almost all the susceptible populations got infected. We also discovered that infected populations increased sporadically before decreasing as we can see from figure 2. This may be due to the fact that the virus is not detected early in a population and so there was delay in treatment. So the infected populations decreases when the treated populations increases. We discovered also that treatment rate has a little delay before taken effect as we can see from figure 3, this may result from the fact that the infected populations did not yield to treatment early due to the nature of the virus. Finally we discovered that the rate at which the virus remove people in the population hardly increase as we can see from figure 4. This may be due to the fact that the virus has the ability to remain in the host for life without causing the virus associated disease (infectious mononucleosis) and this shows that the virus hardly kills.

12.1 Possible Control

Due to the research we made and the result we obtained from the model; we therefore give the following possible control of Epstein Barr virus within human population.

i. People should avoid unnecessary kissing, since there is no vaccine to prevent against the virus.
ii. The world health organization (WHO) should improve on the treatment rate of the virus. Since the graphical solution revealed that increase in the treatment rate decreases the infection rate.

13. CONCLUTION

From the research we made and the result we got from the graphical solution of the model, it is obvious that Epstein Barr virus is a dangerous virus that is not easily discovered within a population. Also the virus has the ability to maintain long life persistent infection in the host, due to the fact that the removal rate is very low as we can from the graphical solution. We therefore conclude that Epstein Barr virus spread globally due to the fact that the virus is not easily discovered in a population. Also because there is no vaccine to prevent against the virus.

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