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Mathematical Analysis of the Global Dynamics of an SVEIR Epidemic Model with Herd Immunity

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Abstract- The SVEIR epidemiological model was presented to gain insight into the mathematical epidemiological model with herd immunity in the population. Positivity of solution was shown for the mathematical and epidemiological well posed of the model. The stability of the model was analyzed for the existence of disease free and endemic equilibrium points. The threshold quantity "Basic Reproduction Number" (R_0) with and without vaccine was derived using next generation matrix method (NGM), and it is shown that the disease free equilibrium point is locally asymptotically stable whenever the basic reproduction number is less than unity i.e. ($R_0 < 1$), otherwise endemic whenever it exceeds unity ($R_0 > 1$). Global stability of endemic equilibrium was analyzed using Lyapunov method and numerical simulation of the model was carried out using Runge-Kutta method of order four (4) with MAPLE 18.
Results showed that herd immunity can only be attained whenever everyone in the population is vaccinated against the infection, since ($R_{Vaccine} < R_0$).

Keywords- *Vaccine, Reproduction Number, Stability, Critical Point, Numerical Simulation*

I. INTRODUCTION

The term "herd immunity" appears first in a paper published in 1923 titled "The spread of bacterial infection: the problem of herd immunity" [20]. Herd Immunity is the direct result of vaccines that work and a vaccination rate that is high enough [4]. Herd immunity is also known as community immunity which is a population-seek name for a phenomenon in the context of disease that can be passed from person to person. Vaccine convey immunity to disease while immunized individuals have antibodies that will neutralize germs when they come in contact with them, making it much less likely to pass on to others [17].

Also, if nearly everyone is immune, then almost no one will spread the disease so, the people who have not been vaccinated, those whose vaccinations have become weakened and whose vaccine are not fully effective can be shielded by the herd immunity because vaccinated people around them would not get sick [8]. An important characteristic of most vaccines is that they provide both individual and community protection. Most of the diseases against which we vaccinate are transmitted from person to person. When a sufficiently large proportion of individuals in a community is immunized, those persons serve as a protective barrier against the likelihood of transmission of the disease in the community, thus indirectly protecting those who are not immunized and those who received vaccine but are not protected (vaccine failures) [13]. The vaccination rate that is critical for stopping the spread of disease depends on how infectious the disease is. Meanwhile the low vaccine efficacy and low vaccination rate result into no herd immunity while high in vaccine efficacy and its rate yields herd immunity, some diseases which vaccines can prevent or eradicate includes measles, pneumonia, pertussis, Rubella, Mumps, diphtheria Tetanus, Polio, Haemophillus influenza type B, Hepatitis B, Smallpox etc.[5]. The infectious diseases lead to endemic when it can be sustained in a population without the need for external inputs. This implies that on average, each infected person is infecting exactly one other person (anymore and the number of people infected will grow exponentially and there will be an epidemic, any less and the disease will out) [10].

Since 1962, the USA federal government has supported childhood vaccination programs through a grant program administered by the CDC. [19].

Mathematical epidemiology relating to vector-borne diseases has been repeatedly a source of important insights for the field of vaccination and herd immunity.

Interest in applying the "magic" of herd immunity in disease control has encouraged mathematical research exploring the theoretical implications of the subject [1, 7].

In 1796, Edward Jenner demonstrated that inoculation with material from a cowpox (vaccinia) lesion would protect against subsequent exposure to smallpox. This began the vaccine era, although it was nearly 100 years until the next vaccine (against rabies) was introduced. In the twentieth century, many new vaccines were developed and used, with spectacular impact on the occurrence of disease. The Centers for Disease Control and

Prevention (CDC) declared vaccinations to be one of the 10 great public health achievements of the twentieth century [3, 6]

Anderson RM and May RM (1982), used SIR epidemiology model to predict herd immunity, he concluded that the percentages of vaccinations required for herd immunity were based on the Ro and contact number and recommends increase in percentage of the population to be vaccinated in order to maintain herd immunity. He advised parents to realize that vaccinations are only effective if a large portion of the population receives the vaccination [1].

The Encyclopedia of Public Health says "Survival of the (disease) agent is crucial if it cannot survive, it cannot invade and infect new hosts, and the epidemic ends". Any vaccination program for protection against Sexual Transmitted Infectious is different for two major reasons. First, there is extreme heterogeneity in the risk of acquiring and transmitting the infective and second, the diseases affect sexually active adults, and severe diseases often is restricted to a minority of cases, with the majority severe consequences at women rather than man. These factors will influence the effects of herd immunity and the target populations to be protected by vaccination programs. Preventive vaccines offer an ideal tool for the control of infectious disease while treatments which work in advance of any disease and associated morbidity which do not rely on the identification of any cases.

According to Meissner, (2015), if a sufficient number of people (herd) are immune, it will reduce the probability of susceptible person that will come in contact with an infectious person because the infection will no longer circulate. The benefits of herd immunity apply to various segments of our society among which are children, immune suppressed patients who cannot be vaccinated, elderly people who cannot mount an optimal immune response to vaccine, people in whom vaccine-induced has waned and the people who remain unvaccinated by choice Vaccines teach the immune system to fight disease by mimicking a natural infection [18].

Andrew wakefield (2016) explained herd immunity is the presence of adequate immunity within a population against a specific infection that operates to protect those at high risk of serious infection and consequently reduce morbidity and mortality from that infection [2]. The immune system is a network of cells, tissues and organs that work together to defend the body against attacks by foreign invaders. It is a form of immunity that occurs when the vaccination of a significant portion of the population provides a measure of protection for individual who are not vaccinated (John *et al*, 2000), [16]. The effect of vaccine on herd immunity shall be investigated in this research work.

II. Model Formulation

The total population size at time t denoted by N (t) is sub-divided into five (5) compartments of Susceptible individual S(t), Vaccinated individual V(t), Exposed individual E(t), Infected individual, I(t) and Recovered individual R(t) so that:

$$N(t) = S(t) + V(t) + E(t) + +I(t) + R(t) \qquad (1)$$

The susceptible population is increased by the recruitment of people (either by birth or immigration) into the population, all recruited individuals are assumed to be susceptible at a rate π, the population of Susceptible is further increased by the population of vaccinated individuals due to vaccine wanes at the rate (ϕ). Finally, the susceptible population decreases by infection which can be acquired following effective contact rate β, natural death, at the rate (μ) and by vaccinated individuals at the rate (ρ). Hence;

$$\frac{dS}{dt} = (1-\rho)\pi - \beta SI - \mu S + \varphi V \qquad (2)$$

The population of vaccinated individual is increased by the individuals that received vaccine at the rate (ρ). The population later decreased by the natural death rate (μ) and the rate at which the vaccine wanes (ϕ). Hence;

$$\frac{dV}{dt} = \rho\pi - (\mu + \varphi)V \qquad (3)$$

A proportion $(1-\omega)$ of newly infected individuals that produce active infection move to the exposed class E, while the remaining proportion ω move to the infected class I. The population of exposed class is reduced by the natural death rate (μ) and the progression rate (κ). Hence;

$$\frac{dE}{dt} = (1-\omega)\beta SI - (\mu + \gamma + \kappa)E \qquad (4)$$

The population of Infected individual is increased by the remaining proportion of individual that produce active infection at the rate (ω) and the progression of exposed individual at the rate (κ). The population is decreased by the treatment of infected individuals at the rate (σ), natural death and death due to infection at the rate (μ) and (δ) respectively. Hence;

$$\frac{dI}{dt} = \omega\beta SI + \kappa E - (\mu + \delta + \sigma)I \qquad (5)$$

The population of Recovered individual is increased by the number of infected individuals that are treated at the rate (σ). The population is decreased by the natural death rate of recovered individual at the rate (μ). Hence;

$$\frac{dR}{dt} = \sigma I - \mu R \qquad (6)$$

Thus in summary, the dynamics transmission model is given by the following system of non-linear differential equations.

$$\left.\begin{aligned}
\frac{dS}{dt} &= (1-\rho)\pi N - \frac{\beta S(t)I(t)}{N} - \mu S(t) + \varphi V(t)\\
\frac{dV}{dt} &= \rho\pi - (\mu+\varphi)V(t)\\
\frac{dE}{dt} &= (1-\omega)\frac{\beta S(t)I(t)}{N} - (\mu+\kappa)E(t)\\
\frac{dI}{dt} &= \omega\beta S(t)I(t) + \kappa E(t) - (\mu+\delta+\sigma)I(t)\\
\frac{dR}{dt} &= \sigma I(t) - \mu R(t)
\end{aligned}\right\} \quad (7)$$

We rescale the state variables of the formulated model by normalizing as follows;

$$\bar{S} = \frac{S}{N}, \bar{V} = \frac{V}{N}, \bar{E} = \frac{E}{N}, \bar{I} = \frac{I}{N}, \bar{R} = \frac{R}{N}$$

So that $\bar{S} + \bar{V} + \bar{E} + \bar{I} + \bar{R} = 1$

Thus, after dropping of bars, model (7) leads to the following;

$$\left.\begin{aligned}
\frac{dS}{dt} &= (1-\rho)\pi - \beta S(t)I(t) - \mu S(t) + \varphi V(t)\\
\frac{dV}{dt} &= \rho\pi - (\mu+\varphi)V(t)\\
\frac{dE}{dt} &= (1-\omega)\beta S(t)I(t) - (\mu+\kappa)E(t)\\
\frac{dI}{dt} &= \omega\beta S(t)I(t) + \kappa E(t) - (\mu+\delta+\sigma)I(t)\\
\frac{dR}{dt} &= \sigma I(t) - \mu R(t)
\end{aligned}\right\} \quad (8)$$

We will consider the model system (8). Since R is independent of other variables.

$$\left.\begin{aligned}
\frac{dS}{dt} &= (1-\rho)\pi - \beta S(t)I(t) - \mu S(t) + \varphi V(t)\\
\frac{dV}{dt} &= \rho\pi - (\mu+\varphi)V(t)\\
\frac{dE}{dt} &= (1-\omega)\beta S(t)I(t) - (\mu+\kappa)E(t)\\
\frac{dI}{dt} &= \omega\beta S(t)I(t) + \kappa E(t) - (\mu+\delta+\sigma)I(t)
\end{aligned}\right\} \quad (9)$$

TABLE I. DESCRIPTION OF VARIABLES

Variables	Definitions
S	Susceptible individual
V	Vaccinated individual
E	Exposed individual
I	Infected individual
R	Recovered individual

TABLE II. DESCRIPTION OF PARAMETERS

Parameters	Definitions
π	Recruitment rate into the population
ρ	Vaccine rate
ϕ	Vaccine wanes.
ω	Proportion of new infection that produce active infection
μ	Natural death rate
κ	Progression rate
σ	Treatment of infected individuals.
δ	Death rate
β	Effective contact rate

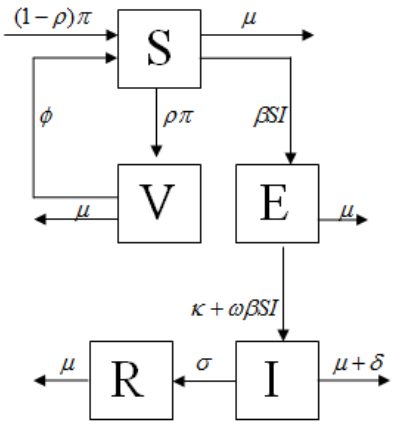

Figure 1. Schematic Diagram

III. POSITIVITY OF SOLUTION

For this SVEIR epidemic model to be epidemiological and mathematically well posed, it is necessary to prove that all state variables are non-negative for all $t > 0$.

Theorem: Let $\{S(0) \geq 0,\ V(0) \geq 0,\ E(0) \geq 0,\ I(0) \geq 0\} \in \Gamma$

Then, the solution: $\{S(t), V(t), E(t), I(t)\}$ of the model system equation (2) are positive $\forall\ t \geq 0$.

Proof: $\frac{dS}{dt} = (1-\rho)\pi - \beta S(t)I(t) - \mu S(t) + \varphi V(t)$

From which it follows that:

$$\frac{dS}{dt} => -\mu S$$

Consequently:

$\frac{dS}{dt} + \mu S \geq 0$ is the first order homogeneous differential equation.

I.F. = $\ell^{\int \mu dt} = \ell^{\mu t}$

Multiplying by the Integrating factor on both sides will give:

$$\ell^{\mu t} \frac{dS}{dt} + \mu S \ell^{\mu t} \geq 0$$

It then follows that:

$$d(S\ell^{\mu t}) \geq 0dt$$

Integrating on both sides gives:

$S\ell^{\mu t} \geq C$ where C is a constant of the integration, it follows that:

$$S(t) \geq C\ell^{-\mu t}$$

Applying the initial condition that, when $t = 0,\ S(t) = S(0)$, we have:

$$S(0) \geq C$$

Hence:

$$S(t) \geq S(0)\ell^{-\mu t} \tag{10}$$

Since $\mu > 0 \, and \, S(0) \geq 0$, then:

$S(t) \geq 0,$ If $t = 0 \, and \, t \to \infty$

Therefore:

$$S(t) \geq 0 \quad \forall t \geq 0. \tag{11}$$

In a similar way, it can be shown that $V(t) \geq 0, E(t) \geq 0, I(t) \geq 0, \ \forall t \geq 0.$

Therefore, the model can be considered as been epidemiologically and mathematically well posed

A. Disease Free Equilibrium

For critical points, we set

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = 0$$

At disease free equilibrium, it is assumed that there is no infection; Hence (DFE) is given as

$$\varepsilon_0 = (S, V, E, I) = \left(\frac{\pi}{\mu} - \frac{\rho\pi}{(\mu + \varphi)}, \quad \frac{\rho\pi}{(\mu + \varphi)}, \quad 0, \right)$$

B. Endemic Equilibrium

The endemic equilibrium of the model (9) is given below as;

$$S^* = \frac{M}{A\mu K_1}$$

$$V^* = \frac{\rho\pi}{K_1}$$

$$E^* = \frac{K_3(\omega - 1)}{A}$$

$$I^* = \frac{\mu K_1 K_2 K_3}{M}$$

Where;

$$K_1 = \mu + \varphi, \ K_2 = \mu + \kappa, \ \ K_3 = \mu + \delta + \sigma$$

$$A = \kappa\omega - K_2\omega - \kappa$$

$$M = A\rho\pi\gamma - \rho\pi\gamma K_1(\kappa - K_2)$$
$$+\pi K_1\kappa(\rho + \omega - 1) - K_1 K_2(\pi\omega - K_3)$$

C. Basic Reproduction Number R_o

The basic reproduction number of the model (9) is calculated by using the next generation matrix [9]. Using the approach, we have,

$$\frac{d}{dt} = \begin{pmatrix} E \\ I \end{pmatrix} \left\{ \begin{pmatrix} (1-\omega)\beta SI \\ \omega\beta SI \end{pmatrix} - \begin{pmatrix} (\mu + \kappa)E \\ (\mu + \delta + \sigma)I - \kappa E \end{pmatrix} \right\} \tag{12}$$

After taking partial derivative (12) at the disease free equilibrium, we have:

$$F = \begin{pmatrix} 0 & (1-\omega)\beta S_0 \\ 0 & \omega\beta S_0 \end{pmatrix} \tag{13}$$

$$V = \begin{pmatrix} \mu + \kappa & 0 \\ -\kappa & \mu + \delta + \sigma \end{pmatrix} \tag{14}$$

Then,

$$R_0 = \frac{\beta S_0(\mu\omega + \kappa)}{\mu(\mu + \delta + \sigma) + \kappa(\mu + \delta + \sigma)} \tag{15}$$

Basic reproduction number is an important notion in epidemiological models and is the usually denoted by R_o. This number can be defined as the expected average number of secondary infection generated by infected infectious individual in his/her infectious period in the susceptible population.

D. Local Stability of Disease Free Equilibrium

Theorem 2: If $R_{Vaccine} < 1$, then, the disease free equilibrium is locally asymptotically stable and unstable in $R_{Vaccine} > 1$

Proof: The Jacobian matrix $J(P_0)$ of the model equation (9) evaluated at disease free equilibrium is given by;

$$J(P_o)=\begin{bmatrix} -\mu & \varphi & 0 & -\beta S_0 \\ 0 & -(\mu+\varphi) & 0 & 0 \\ 0 & 0 & -(\mu+\kappa) & (1-\omega)\beta S_0 \\ 0 & 0 & \kappa & -((\mu+\delta+\sigma)-\omega\beta S_0) \end{bmatrix} \quad (16)$$

The eigenvalues of $J(P_0)$ are $\lambda_1=-\mu, \lambda_2=-(\mu+\varphi)$ and the remaining sub-matrix is;

$$J_1(P_o)=\begin{pmatrix} -(\mu+\kappa) & (1-\omega)\beta S_0 \\ \kappa & -((\mu+\delta+\sigma)-\omega\beta S_0) \end{pmatrix}$$

The characteristics polynomial of equation (16) above is;

$$A_2\lambda^2+A_1\lambda+A_0=0 \quad (17)$$

Where; $A_2=1$

$$A_1=2\mu-\omega\beta S_0+\delta+\kappa+\sigma$$

$$A_0=\mu(\mu+\delta+\sigma)+\kappa(\mu+\delta+\sigma)-\beta S_0(\mu\omega+\kappa)$$

$$\mu(\mu+\delta+\sigma)+\kappa(\mu+\delta+\sigma)-\beta S_0(\mu\omega+\kappa)>0 \quad (18)$$

$$-\beta S_0(\mu\omega+\kappa)>-(\mu(\mu+\delta+\sigma)+\kappa(\mu+\delta+\sigma))$$

Divide both sides by the RHS of (18), gives;

$$\frac{\beta S_0(\mu\omega+\kappa)}{\mu(\mu+\delta+\sigma)+\kappa(\mu+\delta+\sigma)}<1$$

Hence $R_0<1$

It can be seen clearly from the above that $A_2>0$, $A_1>0$ and that $A_0>0$ if $R_0<1$,

From the above, all the eigen-values of the Jacobian matrix $J(P_o)$ are real and negative when $R_o<1$, therefore the disease free equilibrium is locally asymptotically stable

E. Global Stability of Endemic-Equilibrium

Lemma: For $R_0>1$, the equation (9) is globally asymptotically stable if

$s=s^*, v=v^*, e=e^*$, $i=i^*$ and $X<Y$ and unstable when $R_0\le 1$.

Proof: Using the constructed Lyapunov function, the global stability of the endemic equilibrium is proved by defining the Lyapunov function as follows:

$$V=s^*,\ v,\ e^*, q^* = \left(s-s^*-s^*\ln\frac{s^*}{s}\right)+\left(v-v^*-v^*\ln\frac{v^*}{v}\right)$$
$$+\left(e-e^*-e^*\ln\frac{e^*}{e}\right)+\left(i-i^*-i^*\ln\frac{i^*}{i}\right) \quad (19)$$

By direct calculating, the derivative of V along the solution of equation (19), we have;

$$\frac{dV}{dt} = \left(\frac{s-s^*}{s}\right)\frac{ds}{dt}+\left(\frac{v-v^*}{v}\right)\frac{dq}{dt}+\left(\frac{e-e^*}{e}\right)\frac{de}{dt}+\left(\frac{i-i^*}{i}\right)\frac{di}{dt} \quad (20)$$

$$\frac{dV}{dt} =\left(\frac{s-s^*}{s}\right)\left((1-\rho)\pi-\beta si-\mu s+\varphi v\right)+$$
$$\left(\frac{v-v^*}{v}\right)\left(\rho\pi-(\mu+\varphi)v\right)+$$
$$\left(\frac{e-e^*}{e}\right)\left((1-\omega)\beta si-(\mu+\kappa)e\right)+$$
$$\left(\frac{i-i^*}{i}\right)\left(\omega\beta si+\kappa e-(\mu+\delta+\sigma)i\right) \quad (21)$$

Substituting $s=s-s^*$, $v=v-v^*$, $e=e-e^*$, $i=i-i^*$ Into equation (21)

Collecting the like terms, we have:

$$\frac{dV}{dt}=\left(\frac{s-s^*}{s}\right)((i-\rho)+\varphi(v-v^*))-\left(\frac{(s-s^*)^2}{s}\right)$$
$$(\beta(i-i^*)+\mu)+\rho\pi\left(\frac{v-v^*}{v}\right)-(\mu+\varphi)\left(\frac{(v-v^*)^2}{v}\right)$$
$$+(1-\omega)\beta\left(\frac{(e-e^*)}{e}\right)(s-s^*)(i-i^*)$$
$$-(\mu+\kappa)\left(\frac{(e-e^*)^2}{e}\right)+\gamma\left(\frac{(i-i^*)^2}{i}\right)(\omega\beta(s-s^*)$$
$$-(\mu+\delta+\sigma))+\kappa\left(\frac{i-i^*}{i}\right)(e-e^*) \quad (22)$$

Open the brackets of (22)

$$\frac{dV}{dt} = (1-\rho)\pi - (1-\rho)\frac{s^*}{s} + \varphi v\left(\frac{s-s^*}{s}\right) - \varphi v^*\left(\frac{s-s^*}{s}\right) - \beta i\left(\frac{(s-s^*)^2}{s}\right) + \left(\frac{(s-s^*)^2}{s}\right)(\beta i^* + \mu) + \rho\pi - \frac{\rho\pi v^*}{v} - \left(\frac{(v-v^*)^2}{v}\right)(\mu+\varphi) + \beta si - \frac{\beta s^* i^* e^*}{e} - \omega\beta si + \frac{\omega\beta s^* i^* e^*}{e} - \left(\frac{(e-e^*)^2}{e}\right)(\mu+\kappa) + \left(\frac{(i-i^*)^2}{i}\right)(\omega\beta s - \omega\beta s^* - (\mu+\delta+\sigma)) + \kappa e + \frac{\kappa i^* e^*}{i} + \sigma i + \sigma i^* \quad (23)$$

Re-arranging the positive and negative terms

Where $\frac{dV}{dt} = X - Y$

$$X = (1-\rho)\pi + \varphi v\left(\frac{s-s^*}{s}\right) + \left(\frac{(s-s^*)^2}{s}\right)(\beta i^* + \mu) + \rho\pi + \beta si + \omega\beta\frac{e^*}{e}s^* i^* + \omega\beta s\left(\frac{(i-i^*)^2}{i}\right) + \kappa e + \frac{i^*}{i}\kappa e^*$$

$$Y = (1-\rho)\frac{s^*}{s} + \varphi v^*\left(\frac{s-s^*}{s}\right) + \beta i\left(\frac{(s-s^*)^2}{s}\right) + \rho\pi\frac{v^*}{v} + \left(\frac{(v-v^*)^2}{v}\right)(\mu+\varphi) + \beta\frac{e^*}{e}s^* i^* + \beta\omega si + \left(\frac{(e-e^*)^2}{e}\right)(\mu+\kappa) + \omega\beta s^*\left(\frac{(i-i^*)^2}{i}\right) + (\mu+\delta+\sigma)\left(\frac{(i-i^*)^2}{i}\right) + \sigma i^*$$

Hence, if $X < Y$, then we obtain $\frac{dV}{dt} \le 0$. Noting that $\frac{dV}{dt} = 0$ if and only if

$s = s^*$, $v = v^*$, $e = e^*$, $i = i^*$, therefore, the largest compact invariant set:

$\left\{(s^*, v^*, e^*, i^*) \in \Gamma : \frac{dV}{dt} = 0\right\}$ Is the singleton $\{E^*\}$ where E^* is the endemic equilibrium. Hence, by La Salle's principle, it implies that E^* is globally asymptotically stable in Γ if $X < Y$.

F. Numerical Simulation

Numerical simulation of the SVEIR epidemic model is carried out by MAPLE 18 software using Numerical Runge-Kutta method of order four (4). The table of the parameter values used as shown in table 3.

TABLE III. PARAMETERS AND VALUES

Parameters	Values
π	500
β	0.2
ω	0.2
κ	0.002
μ	0.5
δ	0.9
ρ	0.01
γ	0.2
υ	0.2
σ	0.2

IV. DISCUSSION OF RESULTS AND CONCLUSION

Five mathematical epidemiological compartmental model (SVEIR) was presented to gain insight into the effect of vaccine on herd immunity in the population. The stability of the model was analyzed for the existence of disease free and endemic equilibrium points. The threshold quantity "Basic Reproduction Number (R_0) with and without vaccine was derived using next generation matrix method (NGM), and it is shown that the disease free equilibrium point is locally asymptotically stable whenever the basic reproduction number is less than unity i.e ($R_0 < 1$), otherwise endemic whenever it exceeds unity ($R_0 > 1$). The result shows that Basic Reproduction Number (R_0) with vaccine is far less compared to when vaccine is absent i.e. ($R_{Vaccine} < R_0$).

Global stability of endemic equilibrium was analyzed using Lyapunov method and the result shows that endemic is stable whenever Basic Reproduction Number exceeds unity i.e. ($R_0 > 1$). Numerical simulation of the model in figure 2 shows that vaccination of susceptible individuals reduces the dynamical spread of the disease compared to when vaccine given is not implemented. Figure 3 shows that introduction of the vaccine plays a vital role in the dynamical control of the SVEIR diseases. Its influence over the basic reproduction number cannot be over emphasized. It reduces this threshold quantity that determines the spread of any contagious disease to the minimum point. Figures 4 and 5 show that; herd immunity can only be attained whenever everyone in the population is vaccinated against the infection. The result in figure 6 shows that herd immunity can be attained when the

vaccination of susceptible individuals and treatment of infected individuals is full.

Conclusively, full vaccination of susceptible individuals and complete treatment of infected individuals should be given adequate priority to forestall the dynamical spread of the disease in the population.

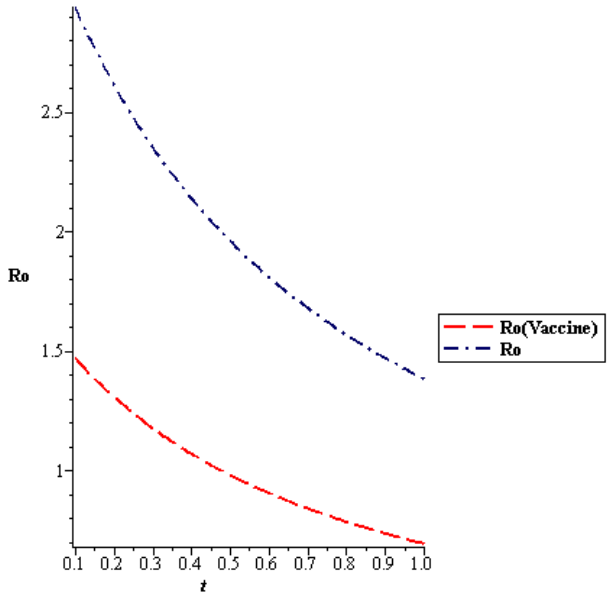

Figure 2. Graph of basic reproduction number against treatment (σ) rate at time (t)

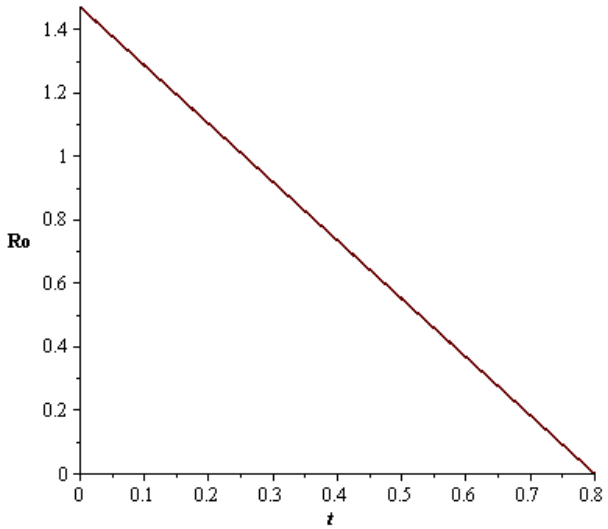

Figure 3. Graph of basic reproduction number against vaccine rate (ρ) at time (t)

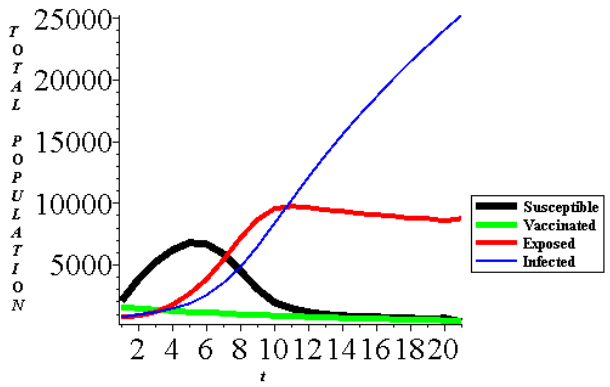

Figure 4. Graph of total population against time (t) when ρ = σ = 0

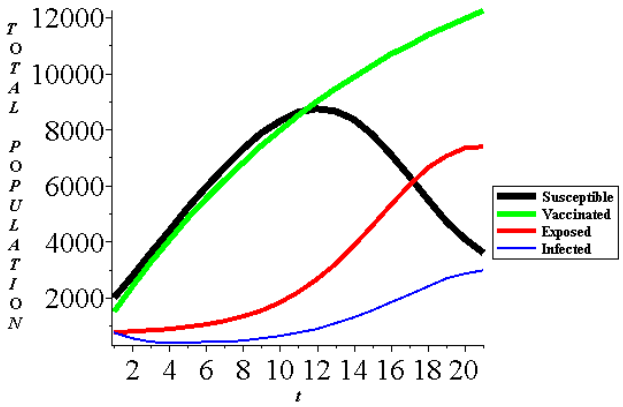

Figure 5. Graph of total population against time (t) when ρ = σ = 5

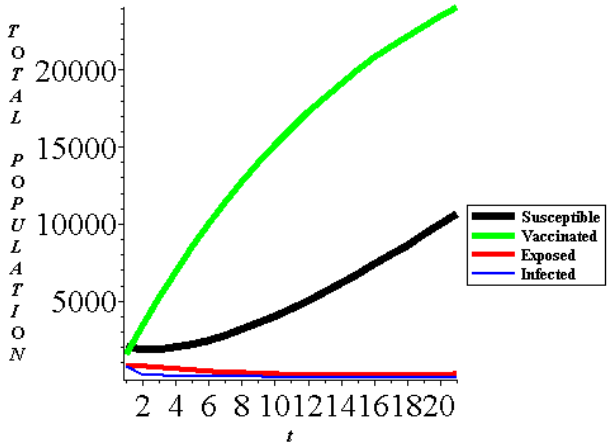

Figure 6. Graph of total population against time (t) when ρ = σ = 1

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