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A Mathematical Model and Analysis of an SVEIR Model for Streptococcus Pneumonia with Saturated Incidence Force of Infection

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Abstract: In this paper, the dynamics of SVEIR model with saturated incidence force of infection and saturated vaccination function for Streptococcus pneumonia (that is, model that monitors the temporal transmission dynamics of the disease in the presence of preventive vaccine) was formulated and analyzed. The basic reproduction number that determines disease extinction and disease survival was revealed. The existing threshold conditions of all kinds of the equilibrium points are obtained and proved to be locally asymptotic stable for disease-free equilibrium using linearization method and Lyapunov functional method for Endemic equilibrium. Qualitative Analysis of the model was obtained and the positive of solution obtained. It was revealed that the model is positively –invariant and attracting. Thus the region is positively invariant. Hence, it is sufficient to consider the dynamics of the model (1) in the given region. In this region, the model can be considered as been epidemiologically and mathematically well-posed. The governing model was normalized and also Adomian Decomposition method was used to compute an approximate solution of the non-linear system of differential equations governing the model. Maple was used in carrying out the simulations (numerical solutions) of the model. Graphical results were presented and discussed to illustrate the solution of the problem. The achieved results reveal that the disease will die out within the community if the vaccination coverage is above the critical vaccination proportion. The study indicates that we should improve the efficiency and enlarge the capacity of the treatment to control the spread of disease.

Keywords: Mathematical Model, SVEIR Model, Streptococcus Pneumonia, Saturated Incidence Force Ofinfection

1. Introduction

Streptococcus pneumonia is a facultative anaerobic bacterium, gram-positive which have the shape of a lancet. It exists in more than 90 serotypes. Most of these serotypes can originate from different diseases, with little of serotypes being the major factor of pneumococcal infections [6]. Research showed that this gram-positive bacterium "Pneumococcus "inhabits the respiratory tract and perhaps can be secluded from the nasal part of the pharynx, lying behind the nose of about 5–90% among individuals who are healthy, relatively on the population and setting. Adults forms about 5-10% carriers while 20–60% of school-aged children are probably carriers.

Investigations also revealed that about 50–60% of service personnel may be carriers. The time frame of carriage differs among different age group as it tends to be longer in children than adults. Furthermore, it is still unclear for researchers on how carriage and individuals' ability to develop natural immunity are related.

Most of the serious infections that are community-acquired (like; meningitis and bacteremia) has been traced to Streptococcus pneumonia being one of the primary cause among children who are below the age of 5 years, [20, 36]. Among patients who are HIV positive, S*p*has also be linked to be the major cause of sepsis and bacteremia, [22]. The infections instigated by S. pneumonia are one of the principal causes of death among

children in Nigeria today, [13]. This is recorded due to the poor access to adequate healthcare and can also be attributed to high endemicity of HIV infect, [4]. Studies have associated children who are colonized with S. pneumonia to have higher chance of being hospitalized compared those who are not colonized, [12].

A large number (especially young children) of people are still battling with infections caused by these bacteria, the elderly or patients with low immunity irrespective of the efforts to ensure that rate of mortality and morbidity among children in developing countries is reduced, [5]. Research have revealed that S. pneumonia is the most popular bacteria isolated from blood and sputum samples of children with severe S. pneumonia, [33]. Pneumococcal disease is generally preceded by asymptomatic colonization, which is mainly high in children, [5, 22]. Depending on the considered case, invasive pneumococcal diseases occurs immediately after colonization, and it has been revealed that the streptococcal nasopharyngeal carriage prevalence in unvaccinated children is similarly high in Africa ranging from 7–90%, [3, 4, 9, 21, 22].

The threat of *Sp* infection has been increasing despite interventions by widely available antibiotics, due to the increasing presence of multiple antibiotic-resistant *Sp* strains, [35, 31].

There is an urgent call for a better informed intervention targeted in mitigating the early occurrence of S*p*-mediated pathology through vaccination and treatment with little antibiotics [37, 38]. Formerly, proposed mathematical models considered *Sp* infections in the lung which is a normally sterile site of the airway epithelium. There have been numerous epidemic models designed and explored, also many vaccination campaigns to prevent eradicate or mitigate the speed transmission of the infectious childhood diseases (for example measles, tuberculosis, and flu). Bilinear incidence rate βSI has been frequently applied in many epidemic models according to [1, 2, 30, 39, 40, 41]. The saturated incidence rate βSI (1+αI) was introduced by [11]. This reveals that if βI (which estimates the infection force at time of disease total invasion in the susceptible population) is large together with 1/(1+αI) (which estimates the reacting effect out of the behavioral change of the susceptible population at the time we have a crowding effect of the infected population), then the model is certainly to be saturated. It comes up with the concept of continuous treatment in an SIR model as follows:

$$h(I) = \begin{cases} r, & I > 1 \\ 0, & I < 1 \end{cases} \qquad (1)$$

$$h(I) = \begin{cases} r, & I > 1 \\ 0, & I < 1 \end{cases} \qquad (2)$$

That takes care of some fraction of individuals who are successfully treated and the rest unsuccessful, [43]. Further, Wang considered the following piecewise linear treatment function

$$h(I) = \begin{cases} rI, & 0 \le I \le 1 \\ yI_0, & I > I_0 \end{cases} \qquad (3)$$

We agree this to be more reasonable than the usual linear function. This revealed that if treatment is delayed for infected individuals, the efficiency will be drastically affected. Furthermore, the continuous and differentiable saturated treatment function was introduced and used given as $h(I) = rI/(1 +kI)$, where r>0, k>0, *r* implies rate of cure, and k estimates the treatment delay level of the infected individuals. This reveals that if *I* is small, then the treatment function tends to *r*I, while it tends to *r/k* if *I* is large, [10, 45].

In the maximum amount because the dynamics of SIR or SIS epidemic models with the saturated incidence rate are recurrently utilized in several literatures. We have a tendency to still have very little researches regarding the saturated treatment operate even within the SEIR epidemic models. Other works which we looked at in this work are: [8, 15, 18, 28, 34, 25, 29, 45, 46, 47].

2. Model Formulation

2.1. Assumptions of the Model

The assumptions of the model are stated below:
1. We assume that the saturated incidence force of infection rate is $\frac{\beta I}{1+\alpha I}$
2. People in the Vaccinated compartment can be infected.
3. People can be infected only through contacts with infectious people except those who are immune.
4. Recovered individuals may go back to the susceptible class.
5. The population is homogeneously mixed (A population that interacts with one another to the same degree and fixed).

2.2. Symbols and Parameter of the Model

Below are the symbols and parameters of the proposed model.

Table 1. Symboles and parameters.

Symbols and Parameters	Description
S	Susceptible Individuals at time t
V	Vaccinated Individuals at time t
E	Exposed individuals at time t
I	Infectious individuals at time t
R	Recovered individuals at time t
μ (Mu)	Per capita natural mortality rate (Natural death rate)
β (Beta)	Contact rate

Symbols and Parameters	Description
$\Re_0$	Basic reproduction number
ξ (Xi)	Effectiveness of vaccine
$(1-\xi)$	Rate at which vaccines get waned
N	Total population
P	Fraction of recruited individuals into the vaccinated population
σ (Sigma)	infectious rate of exposed individuals
γ Gama	Recovery rate of infected individuals
ω (Omega)	Susceptible rate of vaccinated individuals
Λ (Capital Lambda)	Recruitment rate into the susceptible compartment
$\lambda = \dfrac{\beta I}{1+\alpha I}$	Saturated incidence force of infection
ρ (Rho)	Per capita rate of recovered individuals going back to the Susceptible class.
A	Death as a result of the infection
F_i	The rate of appearance of new infection in compartment I
v_i^+	The rate of transfer of individuals into the infected compartment
v_i^-	The transfer of individual out of the infected compartment

Now we have

$$N(t) = S(t) + V(t) + E(t) + I(t) + R(t)$$
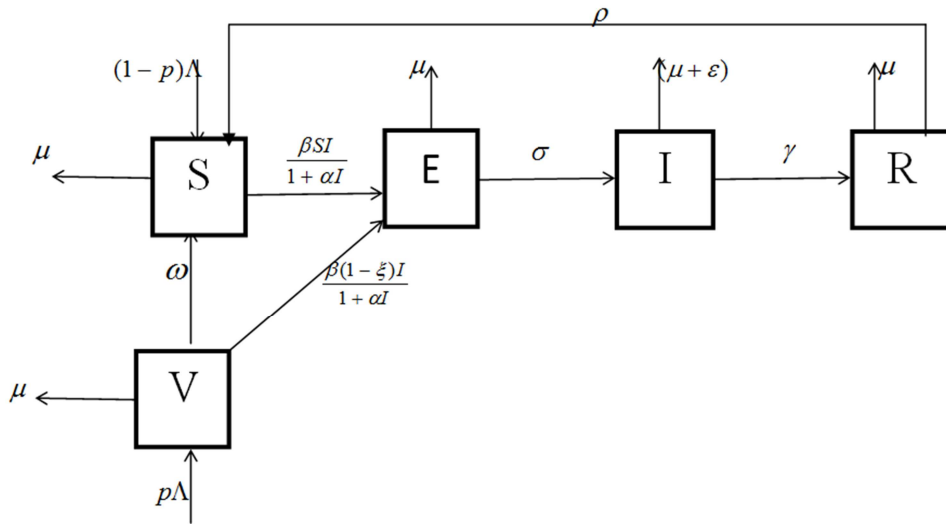
2.3. Flow Diagram of the Model

Figure 1. *Schematic Diagram of the Model.*

2.4. The Model Equation

Applying the symbols and parameters, assumptions and flow diagram, we now formulate the model equations as follows:

$$\left.\begin{aligned}
\frac{dS}{dt} &= (1-p)\Lambda + \omega\Lambda + \rho R - \frac{\beta I}{1+\alpha I}S - \mu S \\
\frac{dV}{dt} &= p\Lambda - \frac{\beta(1-\xi)VI}{1+\alpha I} - (\mu+\omega)V \\
\frac{dE}{dt} &= \frac{\beta I}{1+\alpha I}S + \frac{\beta(1-\xi)VI}{1+\alpha I} - (\mu+\sigma)E \\
\frac{dI}{dt} &= \sigma E - (\mu+\varepsilon+\gamma)I \\
\frac{dR}{dt} &= \gamma I - (\sigma+\rho)R
\end{aligned}\right\} \tag{4}$$

Where $\lambda = \dfrac{\beta I}{1+\alpha I}$ is the saturated incidence force of infection.

3. Qualitative Analysis

3.1. Positivity of Solutions

We prove the positivity of the solution by stating and proving the Theorem 3.1 below.

Theorem 3.1:

Suppose we have the initial solution of our model to be $\{S(0), V(0), E(0), I(0), R(0) \geq 0\} \in \varphi$. Then for all $t \geq 0$ the solution set $\{S(t), V(t), E(t), I(t), R(t)\}$ the system (3) remains positive

Proof:

From the first equation of (3) we have

$$\frac{dS}{dt} \geq -\mu S \Rightarrow \frac{dS}{S} \geq -\mu dt$$

$$\Rightarrow \ln S \geq -\mu t + c \Rightarrow S \geq e^{-\mu t + c} = S_0 e^{-\mu t} \geq 0 \quad where \quad S_0 = e^c$$

From the second equation of (3) we have

$$\frac{dV}{dt} \geq -(\mu + \omega)V$$

$$\Rightarrow \frac{dV}{V} \geq (\mu + \omega)dt \Rightarrow \ln V \geq (\mu + \omega)t + c \Rightarrow V \geq e^{(\mu + \omega)t + c} = V_0 e^{(\mu + \omega)t} \quad where \ V_0 = e^c$$

From the third equation of (3) we have

$$\frac{dE}{dt} \geq -(\mu + \sigma)E \Rightarrow \frac{dE}{E} \geq -(\mu + \sigma)dt$$

$$\Rightarrow \ln E \geq -(\mu + \sigma)t + c \Rightarrow E \geq e^{-(\mu + \sigma)t + c} = E_0 e^{-(\mu + \sigma)} \quad where \ E_0 = e^c$$

From the fourth equation of (3) we have

$$\frac{dI}{dt} \geq -(\mu + \varepsilon + \gamma)I \Rightarrow \frac{dI}{I} \geq -(\mu + \varepsilon + \gamma)dt$$

$$\Rightarrow \ln I \geq -(\mu + \varepsilon + \gamma)t + c \Rightarrow I \geq e^{-(\mu + \varepsilon + \gamma)t + c} = I_0 e^{-(\mu + \varepsilon + \gamma)t} \quad where \ I_0 = e^c$$

From the fifth equation of (3) we have

$$\frac{dR}{dt} \geq -(\mu + \rho)R \Rightarrow \frac{dR}{R} \geq -(\mu + \rho)dt$$

$$\ln R \geq -(\mu + \rho)t + c \Rightarrow R \geq e^{-(\mu + \rho)t + c} = R_0 e^{-(\mu + \rho)} \quad where \ R_0 = e^c$$

Hence the solution set $\{S(t), V(t), E(t), I(t), R(t)\}$ of the system (3) is positive for all $t \geq 0$.

Remark 1: $e^k > 0$ for all real values of k.

Theorem 3.2 (Invariant Region)

The following feasible region of the model (3)

$$\Omega = \left\{ (S, V, E, I, R) \in \mathbb{R}^5_+ : S + V + E + I + R \leq \frac{\Lambda}{\mu} \right\}$$

is a positive invariant region and attracting.

Proof: From model (3),

$$S + V + E + I + R = N \Rightarrow \frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$$

$$\frac{dN}{dt} = \Lambda - \mu S - \mu V - \mu E - \mu I - \mu R = \Lambda - \mu(S + V + E + I + R) = \Lambda - \mu N$$

$$\therefore \frac{dN}{dt} = \Lambda - \mu N$$

$$\Rightarrow \frac{dN}{dt} = \Lambda - \mu N \Rightarrow \frac{dN}{dt} + \mu N = \Lambda \Rightarrow \frac{d}{dt}(\mu N) = \Lambda + ce^{-\mu} \Rightarrow N = \frac{\Lambda}{\mu} + \frac{ce^{-\mu}}{\mu} \Rightarrow N \leq \frac{\Lambda}{\mu} \geq 0$$

$$\therefore \Omega = \left\{ (S, V, E, I, R) \in \Re_+^5 : S + V + E + I + R \leq \frac{\Lambda}{\mu} \right\}$$

is positive invariant and attracting.

Thus Ω is positive invariant. Therefore, it is very significant to study the behavior of our model (4) in region Ω, because in this region, the model is epidemiologically and mathematically meaningful, [14].

3.2. Existence of Steady States

The existence of steady state of the model occurs at

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

$$(1 - p)\Lambda + \omega\Lambda + \rho R - \frac{\beta I}{1 + \alpha I} S - \mu S = 0 \tag{5}$$

$$p\Lambda - \frac{\beta(1 - \xi)VI}{1 + \alpha I} - (\mu + \omega)V = 0 \tag{6}$$

$$\frac{\beta I}{1 + \alpha I} S + \frac{\beta(1 - \xi)VI}{1 + \alpha I} - (\mu + \sigma)E = 0 \tag{7}$$

$$\sigma E - (\mu + \varepsilon + \gamma)I = 0 \tag{8}$$

$$\gamma I - (\sigma + \rho)R = 0 \tag{9}$$

Solving (7)—(11) we have

From (7),

$$(1 - p)\Lambda + \omega\Lambda + \rho R - \frac{\beta I}{1 + \alpha I} S - \mu S \Rightarrow S^* = \frac{(1 - p)\Lambda + \omega V^* + \rho R^*}{(\lambda\beta I^* + \mu)} \tag{10}$$

From (8)

$$p\Lambda - \frac{\beta(1 - \xi)VI}{1 + \alpha I} - (\mu + \omega)V = 0 \Rightarrow p\Lambda(1 + \alpha I) = \beta(1 - \xi)VI - (\mu + \omega)(1 + \alpha I)V$$

$$\Rightarrow V^* = \frac{p\Lambda}{\lambda\beta(1 - \xi)I^* - (\mu + \omega)} \tag{11}$$

From (9)

$$\frac{\beta I}{1+\alpha I}S + \frac{\beta(1-\xi)VI}{1+\alpha I} - (\mu+\sigma)E = 0 \Rightarrow E^* = \frac{\lambda\beta I^*[S^* + (1-\xi)V^*]}{(\mu+\sigma)} \tag{12}$$

From (10)

$$\sigma E - (\mu+\varepsilon+\gamma)I = 0 \Rightarrow I^* = \frac{\sigma E^*}{(\mu+\varepsilon+\gamma)} \tag{13}$$

From (11)

$$\gamma I - (\sigma+\rho)R = 0 \Rightarrow R^* = \frac{\gamma I^*}{(\mu+\rho)} \tag{14}$$

Summarizing we have that the disease-free steady state is

$$\xi^0 = \left(S^*, 0, 0, 0, 0\right) \tag{15}$$

and the endemic steady states is

$$\xi_E = \left(S^*, V^*, E^*, I^*, R^*\right) \tag{16}$$

where S^*, V^*, E^*, I^*, R^* are as defined below.

$$\left. \begin{aligned} S^* &= \frac{(1-p)\Lambda + \omega V^* + \rho R^*}{(\lambda\beta I^* + \mu)} \\[2mm] V^* &= \frac{p\Lambda}{\lambda\beta(1-\xi)I^* - (\mu+\omega)} \\[2mm] E^* &= \frac{\lambda\beta I^*[S^* + (1-\xi)V^*]}{(\mu+\sigma)} \\[2mm] I^* &= \frac{\sigma E^*}{(\mu+\varepsilon+\gamma)} \\[2mm] R^* &= \frac{\gamma I^*}{(\mu+\rho)} \end{aligned} \right\} \tag{17}$$

3.3. Disease Free Equilibrium (DFE) of the Model

The model system (1) has a steady state at a given period where there is no S. Pneumonia in the population under consideration. That is; when $E^0 = I^0 = 0$. Hence, the disease-free equilibrium (DFE) denoted as ξ^0, of the model system (1) is obtained by Substituting $E^0 = I^0 = 0$ into (18)

Hence the disease free equilibrium of system (1) of the model equation is given by

$$\xi^0 = \left(S^0, V^0, E^0, I^0, R^0\right) = \left(\frac{\Lambda\omega + \Lambda\mu(1-p)}{\mu(\mu+\omega)}, \frac{\Lambda p}{\mu+\omega}, 0, 0, 0\right)$$

3.4. The Model Basic Reproduction Number

The local stability is established by using the next generation operator method on the system.

The basic reproduction number R_0 is defined as the effective number of secondary infections caused by an infected individual during his/her entire period of infectiousness, [42].

When $R_0 < 0$, it implies that each individual produces on average less than one new infected individual and hence the disease dies out with time. On the other hand, when $R_0 > 0$, it means each individual produces more than one new infected individual and hence the disease is able to invade the susceptible population. However, $R_0 = 0$ is the threshold below which the generation of secondary cases is insufficient to maintain the infection within human community? The basic reproduction number cannot be determined from the structure of the mathematical model alone, but depends on the definition of infected and uninfected compartments.

This definition is given for the models that represent

spread of infection in a population. It is obtained by taking the largest (dominant) eigenvalue or spectral radius of

$$\rho\left(F_i V^{-1}\right)$$

where

$$F_i = \left[\frac{\partial f_i(\xi^0)}{\partial X_j}\right] \text{ and } V_i = \left[\frac{\partial v_i(\xi^0)}{\partial X_j}\right]$$

f_i is the rate of appearance of new infection in compartment i and $v_i = v_i^- - v_i^+$

v_i^- is the transfer of individuals out of the compartment i ,

v_i^+ is the rate of transfer into compartment i by any other means,

ξ^0 is the disease free equilibrium

Then by linearizing f_i and v_i , and evaluating at the disease free equilibrium point we obtain the associated matrices F_i and V_i respectively.

The infected compartments are, E and I hence a

straightforward calculation gives

$$f(x) = \begin{pmatrix} \lambda(S + (1-\xi)V) \\ 0 \end{pmatrix}$$

and

$$v(x) = \begin{pmatrix} (\sigma + \mu)E \\ (\mu + \varepsilon + \gamma)I - \sigma E \end{pmatrix}$$

Then F and V which are the Jacobian of f and v evaluated at the DFE ξ^0 respectively becomes

$$F(\xi^0) = \begin{pmatrix} 0 & \beta(S^0 + (1-\xi)V^0) \\ 0 & 0 \end{pmatrix}$$

$$V(\xi^0) = \begin{pmatrix} \sigma + \mu & 0 \\ -\sigma & \mu + \varepsilon + \gamma \end{pmatrix}$$

Hence we compute V^{-1} and obtain

$$V^{-1}(\xi^0) = \frac{1}{(\sigma + \mu)(\mu + \varepsilon + \gamma)} \begin{pmatrix} \mu + \varepsilon + \gamma & 0 \\ \sigma & \sigma + \mu \end{pmatrix}$$

$$FV^{-1} = \frac{1}{(\sigma + \mu)(\mu + \varepsilon + \gamma)} \begin{pmatrix} 0 & \beta(S^0 + (1-\xi)V^0) \\ 0 & 0 \end{pmatrix} \begin{pmatrix} \mu + \varepsilon + \gamma & 0 \\ \sigma & \sigma + \mu \end{pmatrix} = \begin{pmatrix} \dfrac{\sigma\beta(S^0 + (1-\xi)V^0)}{(\sigma + \mu)(\mu + \varepsilon + \gamma)} & \dfrac{\beta(S^0 + (1-\xi)V^0)}{\mu + \varepsilon + \gamma} \\ 0 & 0 \end{pmatrix}$$

The basic reproduction number is given by $R_0 = \rho(FV^{-1})$, that is, the highest eigenvalue of FV^{-1} is
Hence

$$R_0 = \frac{\sigma\beta\Lambda[\mu(1 - p\xi) + \omega]}{\mu(\mu + \omega)(\sigma + \mu)(\mu + \varepsilon + \gamma)} \tag{18}$$

3.5. Local Stability of the Disease-free Steady State

Theorem 3.3: The disease free equilibrium (ξ^0) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$
Proof:
We prove the locally asymptotically stability of the disease free equilibrium (E^0) of model (1) using linearization approach.
We linearize the model equation to obtain the Jacobian matrix:

$$J = \begin{pmatrix} (\lambda\beta + \mu) & \omega & 0 & -\lambda\beta S & \rho \\ 0 & -\lambda\beta(1-\xi)I(\mu + \omega) & 0 & \lambda\beta(1-\xi)V & 0 \\ \lambda\beta I & \lambda\beta(1-\xi)I & -(\mu + \sigma) & \lambda\beta S + \lambda\beta(1-\xi)V & 0 \\ 0 & 0 & \sigma & -(\mu + \varepsilon + \gamma) & 0 \\ 0 & 0 & 0 & \gamma & -(\mu + \rho) \end{pmatrix}$$

At the DFE J becomes;

$$J = \begin{pmatrix} (1-\rho) & \omega & 0 & -\lambda\beta S & \rho \\ 0 & -\lambda\beta(1-\xi)I(\mu+\omega) & 0 & \lambda\beta(1-\xi)V & 0 \\ \lambda\beta I & \lambda\beta(1-\xi)I & -(\mu+\sigma) & \lambda\beta S+\lambda\beta(1-\xi)V & 0 \\ 0 & 0 & \sigma & -(\mu+\varepsilon+\gamma) & 0 \\ 0 & 0 & 0 & \gamma & -(\mu+\rho) \end{pmatrix}$$

Hence the characteristics equation of the above Jacobian is

$$|J-\lambda I| = \begin{bmatrix} \lambda+k_1 & \omega & 0 & \dfrac{-\beta}{\mu}k_1 & -\rho \\ 0 & -(\mu+\omega)-\lambda & 0 & \beta k_1 & 0 \\ 0 & 0 & -(\mu+\sigma)-\lambda & \dfrac{-\beta}{\mu}(k_1+\mu k_1) & 0 \\ 0 & 0 & \sigma & \lambda+(\mu+\varepsilon+\gamma) & 0 \\ 0 & 0 & 0 & \gamma & -(\mu+\rho)-\lambda \end{bmatrix} = 0$$

where I is 5×5 identity matrix

That implies that

$$k_1 = \frac{\Lambda\omega+\Lambda\mu(1-\rho)}{(\mu+\omega)}, \quad k_2 = \frac{(1-\xi)\Lambda p}{(\mu+\omega)}$$

where $(k_1-\lambda)[(\mu+\omega)-\lambda][(\mu+\rho)-\lambda][\lambda^2+(2\mu+\sigma+\xi+\gamma)\lambda+(\mu+\sigma)(\mu+\xi+\gamma)-\dfrac{\alpha\beta}{\mu}(k_1+\mu k_2)] = 0$

Obviously,

$$\lambda_1 = k_1, \ \lambda_2 = -(\mu+\omega), \ \lambda_3 = -(\mu+\rho) \ and \ \lambda^2+(2\mu+\sigma+\xi+\gamma)\lambda+[(\mu+\sigma)(\mu+\xi+\gamma)-\frac{\alpha\beta}{\mu}(k_1+\mu k_2) = 0]$$

Letting $A_2=1$, $A_1 = 2\mu+\sigma+\xi+\gamma$ and $A_0 = (\mu+\sigma)(\mu+\xi+\gamma)-\dfrac{\alpha\beta}{\mu}(k_1+\mu k_2)$, we obtain the equation

(19)

$$A_2\lambda^2 + A_1\lambda + A_0 = 0$$

Using the Routh-Hurwitz stability criterion according to [32] which state that all the roots of the characteristics equation (19) above have negative real parts if and only if the co-efficient A_i are positive and matrices $H_i > 0$ for $i = 0,1,2$

Hence from (18) we see that

$$A_2 = 1 > 0; A_1 = 2\mu+\sigma+\xi+\gamma > 0$$

And

$$A_0 = (\mu+\sigma)(\mu+\xi+\gamma) - \frac{\sigma\beta[\wedge\omega+\lambda\mu(1-\xi\rho)]}{\mu(\mu+\omega)}$$

$$\Rightarrow A_0 = \mu(\mu+\omega)(\mu+\sigma)(\mu+\xi+\gamma)-\sigma\beta\wedge\omega-\sigma\beta\wedge\mu+\sigma\beta\rho\xi$$

$$= \mu(\mu+\omega)(\mu+\sigma)(\mu+\xi+\gamma)+\sigma\beta\rho\xi-\sigma\beta\wedge(\omega+\mu)$$

$$= (\mu+\omega)[\mu(\mu+\sigma)(\mu+\xi+\gamma)]+\sigma\beta\rho\xi > 0$$

Again, the Hurwitz Matrix

$$H = \begin{pmatrix} A_1 & 0 \\ A_0 & A_2 \end{pmatrix}$$

With

$$H_1 = |A_1| = A_1 > 0$$

$$H_2 = \begin{pmatrix} A_1 & 0 \\ A_0 & A_2 \end{pmatrix} = A_1 A_2 > 0$$

This therefore shows that all the eigenvalues of the Jacobian of the system have negative real parts. Hence, the DFE is locally asymptotically stable.

3.6. Global Stability of Disease-free Steady State

Theorem 3.4: If $R_0 \leq 1$ for the model (4) then the infection-free equilibrium state (ξ^0) is the only non-negative equilibrium state of the model (4) and it is globally asymptotically stable.
Proof:
Let $L = c_1 E + c_2 I$ be the Lyapunov function, where c_1 and c_2 are constants

$$\frac{dL}{dt} = c_1 \frac{dE}{dt} + c_2 \frac{dI}{dt} (*)$$

$$\Rightarrow \frac{dL}{dt} = \frac{\sigma}{(\mu+\sigma)(\mu+\xi+\gamma)} \frac{dE}{dt} + \frac{1}{\mu+\xi+\gamma} \frac{dI}{dt}$$

$$= \frac{\sigma}{(\mu+\sigma)(\mu+\xi+\gamma)} \left[\lambda\beta SI + \lambda\beta(1-\xi)VI - (\mu+\sigma)E \right] + \frac{1}{(\mu+\xi+\gamma)} \left[\sigma E - (\mu+\xi+\gamma)I \right]$$

$$= \frac{\sigma}{(\mu+\sigma)(\mu+\xi+\gamma)} \left[\lambda\beta SI + \lambda\beta(1-\xi)VI \right] - \frac{\sigma E}{(\mu+\xi+\gamma)} + \frac{\sigma E}{(\mu+\xi+\gamma)} - I$$

At the D. F. E $\lambda = 1$

$$\Rightarrow \frac{dL}{dt} = \frac{\sigma\beta}{(\mu+\sigma)(\mu+\xi+\gamma)} \left[\frac{\wedge\omega + \wedge\mu(1-\rho)}{\mu(\mu+\omega)} + \frac{(1-\xi)\wedge\rho}{(\mu+\omega)} \right] I - I$$

$$\frac{dL}{dt} = \frac{\sigma\beta \wedge [\omega + \mu(1-\rho\xi)] I}{\mu(\mu+\sigma)(\mu+\omega)(\mu+\xi+\gamma)} - I$$

But

$$\frac{\sigma\beta \wedge [\omega + \mu(1-\rho\xi)]}{\mu(\mu+\sigma)(\mu+\omega)(\mu+\xi+\gamma)} = R_0$$

$$\Rightarrow \frac{dL}{dt} = R_0 I - I$$

$$\frac{dL}{dt} = I(R_0 - 1)$$

$$\frac{dL}{dt} \leq I(R_0 - 1)$$

Hence by Lyapunev-Lasalle asymptotic stability theorem in [24], when $R_0 \leq 1$ it implies that the disease-free equilibrium state (E^*) is globally asymptotically stable in R_+^5.

Because (E^*) is the only point in R^5 where $\frac{dL}{dt} = \frac{dN}{dt} = 0$ that is the derivatives of both N and L are equal to zero, then is (E^*) unique.

This completes the proof.

Remarks: $R_0 \leq 1$ implies that $\frac{dL}{dt} < 0$ holds everywhere in

R_+^5 except the points where $\frac{dL}{dt} = 0$

By Lyapunov-Lasalle asymptotic stability theorem, this implies that the largest invariant set of the system is globally asymptotically stable.

The global stability of the D. F. E. state means that; any initial level of S. pneumonia infection, the infection will gradually die out from the population when $R_0 \leq 1$.

$R_0 > 1$ means that one infected individual living in an entirely Susceptible population will cause an average more than one infected individual in the next generation; in this case, S. pneumonia invades such a population and persists.

Basic reproduction rate R_0 is greater than 1 implies that the D. F. E. is unstable.

3.7. Global Stability of Endemic Steady State

Theorem 3.4: The unique endemic equilibrium of mode (1) given by E^{**} is globally asymptotically stable in Ω if $R_0 > 1$.

Proof:
Consider the following non-linear Lyaponuv function

$$L = c_1\left[S - S^{**} - S^{**}\ln\left(\frac{S}{S^{**}}\right) + E - E^{**} - E^{**}\ln\left(\frac{E}{E^{**}}\right)\right] + c_2\left[V - V^{**} - V^{**}\ln\left(\frac{V}{V^{**}}\right)\right] + c_3\left[I - I^{**} - I^{**}\ln\left(\frac{I}{I^{**}}\right)\right]$$

With the Lyaponuv

$$\frac{dL}{dt} = c_1\left[\left(1 - \frac{S^{**}}{S}\right)\overset{*}{S} + \left(1 - \frac{E^{**}}{E}\right)\overset{*}{E}\right] + c_2\left[\left(1 - \frac{V^{**}}{V}\right)\overset{*}{V}\right] + c_3\left[\left(1 - \frac{I^{**}}{I}\right)\overset{*}{I}\right]$$

$$\frac{dL}{dt} = c_1\left[\left(1 - \frac{S^{**}}{S}\right)\left((1-\rho)\wedge + \omega V + \rho R - \lambda\beta SI - \mu S\right) + \left(1 - \frac{E^{**}}{E}\right)\left(\lambda\beta SI + \lambda\beta(1-\xi)VI - (\mu+\omega)E\right)\right]$$

$$+ c_2\left[\left(1 - \frac{V^{**}}{V}\right)\left(\rho\wedge - \lambda\beta(1-\xi)VI - (\mu+\omega)V\right)\right]$$

$$+ c_3\left[\left(1 - \frac{I^{**}}{I}\right)\left(\sigma E - (\mu+\xi+\gamma)I\right)\right]$$

At the steady state;

$$(1-\rho)\wedge = \lambda\beta S^{**}I^{**} - \omega V^{**} - \rho R^{**} + \mu S^{**}$$

$$\rho\wedge = \lambda\beta(1-\xi)V^{**}I^{**} + (\mu+\omega)V^{**}$$

That implies that;

$$\frac{dL}{dt} = c_1\left[\left(1 - \frac{S^{**}}{S}\right)\left(\lambda\beta S^{**}I^{**} - \omega V^{**} - \rho R^{**} + \mu S^{**} + \omega V + \rho R - 2\beta SI - \mu S\right) + \left(1 - \frac{E^{**}}{E}\right)\left(\lambda\beta SI + \lambda\beta(1-\xi)VI - (\mu+\omega)E\right)\right]$$

$$+ c_2\left[\left(1 - \frac{V^{**}}{V}\right)\left(\lambda\beta(1-\xi)V^{**}I^{**} + (\mu+\omega)V^{**} - \lambda\beta(1-\xi)VI - (\mu+\omega)V\right)\right]$$

$$+ c_3\left[\left(1 - \frac{I^{**}}{I}\right)\left(\sigma E - (\mu+\xi+\gamma)I\right)\right]$$

Collecting all the terms without ** in the infected classes and equating them to zero (0)
Letting

$$-c_1\lambda\beta SI - c_1\lambda\beta S^{**}I + c_1\lambda\beta(1-\xi)VI - c_2\lambda\beta(1-\xi)VI + c_2\lambda\beta(1-\xi)V^{**}I + c_3\sigma E = 0$$

$$c_1\left[\left[\lambda\beta(1-\xi)V + \lambda\beta S^{**}\right] + c_2\left[\lambda\beta(1-\xi)V^{**} - \lambda\beta(1-\xi)V\right]I\right]$$

$$c_1 = \frac{\left[\lambda\beta(1-\xi)V - \lambda\beta(1-\xi)V^{**}\right]c_2}{\lambda\beta\left[(1-\xi)V + S^{**}\right]}$$

$$c_1 = \frac{(1-\xi)\left(V - V^{**}\right)c_2}{(1-\xi)V + S^{**}}$$

$$c_2 = (1-\xi)V + S^{**} \Rightarrow c_1 = (1-\xi)\left(V - V^{**}\right)$$

Also

$$c_3 \sigma E = 0; \sigma \neq 0 \Rightarrow c_3 = 0$$

$$L = (1-\xi)(V - V^{**})\left[S - S^{**} - S^{**}\ln\left(\frac{S}{S^{**}}\right) + E - E^{**} - E^{**}\ln\left(\frac{E}{E^{**}}\right)\right] + \left[(1-\xi)V + S^{**}\right]\left[V - V^{**} - V^{**}\ln\left(\frac{V}{V^{**}}\right)\right]$$

$$\frac{dL}{dt} = (1-\xi)(V - V^{**})\left[\overset{*}{S} - \frac{S^{**}}{S}\overset{*}{S} + \overset{*}{E} - \frac{E^{**}}{E}\overset{*}{E}\right] + (1-\xi)V\left[\overset{*}{V} - \frac{V^{**}}{V}\overset{*}{V}\right] + S^{**}\left[\overset{*}{V} - \frac{V^{**}}{V}\overset{*}{V}\right]$$

$$= (1-\xi)V\left[\overset{*}{S} - \frac{S^{**}}{S}\overset{*}{S} + \overset{*}{E} - \frac{E^{**}}{E}\overset{*}{E} + \overset{*}{V} - \frac{V^{**}}{V}\overset{*}{V}\right] - (1-\xi)V^{**}\left[\overset{*}{S} - \frac{S^{**}}{S}\overset{*}{S} + \overset{*}{E} - \frac{E^{**}}{E}\overset{*}{E}\right] + S^{**}\left[\overset{*}{V} - \frac{V^{**}}{V}\overset{*}{V}\right]$$

$$= (1-\xi)V\left[\begin{array}{l}(1-\rho)\wedge + \omega V + \rho R - \lambda\beta SI - \mu S - \frac{S^{**}}{S}\left((1-\rho)\wedge + \omega V + \rho R - \lambda\beta SI - \mu S\right) \\ + \lambda\beta SI + \lambda\beta(1-\xi)VI - (\mu + \sigma)E - \frac{E^{**}}{E}\left(\lambda\beta SI - \lambda\beta(1-\xi)VI - (\mu+\sigma)E\right) \\ + \rho\wedge - \lambda\beta(1-\xi)VI - (\mu+\omega)V - \frac{V^{**}}{V}\left(\rho\wedge - \lambda\beta(1-\xi)VI(\mu+\omega)V\right)\end{array}\right]$$

$$-(1-\xi)V^{**}\left[\begin{array}{l}(1-\rho)\wedge + \omega V + \rho R - \lambda\beta SI - \mu S - \frac{S^{**}}{S}\left((1-\rho)\wedge + \omega V + \rho R - \lambda\beta SI - \mu S\right) \\ + \lambda\beta SI + \lambda\beta(1-\xi)VI - (\mu + \sigma)E - \frac{E^{**}}{E}\left(\lambda\beta SI + \lambda\beta(1-\xi)VI - (\mu+\sigma)E\right)\end{array}\right]$$

$$+ S^{**}\left[\rho\wedge - \lambda\beta(1-\xi)VI - (\mu+\omega)V - \frac{V^{**}}{V}\left(\rho\wedge - \lambda\beta(1-\xi)VI - (\mu+\omega)V\right)\right]$$

$$= (1-\xi)\left[\begin{array}{l}\lambda\beta S^{**}I^{**} - \omega V^{**} - \rho R^{**} + \mu S^{**} + \omega V + \rho R - \lambda\beta SI - \mu S \\ -\frac{S^{**}}{S}\left(\lambda\beta S^{**}I^{**} - \omega V^{**} - \rho R^{**} + \mu S^{**} + \omega V + \rho R - \lambda\beta SI - \mu S\right) + \lambda\beta SI + \lambda\beta(1-\xi)VI - (\mu+\sigma)E \\ -\frac{E^{**}}{E}\left(\lambda\beta SI + \lambda\beta(1-\xi)VI - (\mu+\sigma)E\right) + \lambda\beta(1-\xi)V^{**}I^{**} + (\mu+\omega)V^{**} - \lambda\beta(1-\xi)VI - (\mu+\omega)V \\ -\frac{V^{**}}{V}\left(\lambda\beta(1-\xi)V^{**}I^{**} + (\mu+\omega)V^{**} - \lambda\beta(1-\xi)VI - (\mu+\omega)V\right)\end{array}\right]$$

$$-(1-\xi)V^{**}\left[\begin{array}{l}\lambda\beta S^{**}I^{**} - \omega V^{**} - \rho R^{**} + \mu S^{**} + \omega V + \rho R - \lambda\beta SI - \mu S \\ -\frac{S^{**}}{S}\left(\lambda\beta S^{**}I^{**} - \omega V^{**} - \rho R^{**} + \mu S^{**} + \omega V + \rho R - \lambda\beta SI - \mu S\right) \\ + \lambda\beta SI + \lambda\beta(1-\xi)VI - (\mu+\sigma)E - \frac{E^{**}}{E}\left(\lambda\beta SI + \lambda\beta(1-\xi)VI - (\mu+\sigma)E\right)\end{array}\right]$$

$$+ S^{**}\left[\begin{array}{l}\lambda\beta(1-\xi)V^{**}I^{**} + (\mu+\omega)V^{**} - \lambda\beta(1-\xi)VI - (\mu+\omega)V \\ -\frac{V^{**}}{V}\left(\lambda\beta(1-\xi)V^{**}I^{**} + (\mu+\omega)V^{**} - \lambda\beta(1-\xi)VI - (\mu+\omega)V\right)\end{array}\right]$$

Collecting all the terms with ** in the infected classes including all the $\mu S^{**}, (\mu+\sigma)E, (\mu+\sigma)E^{**}, (\mu+\omega)V$ and $(\mu+\omega)V^{**}$

$$
\begin{aligned}
= (1-\xi) & \left[\lambda\beta VS^{**}I^{**} - \lambda\beta\frac{VS^{**2}}{S}I^{**} + \lambda\beta\frac{V^{**}S^{**2}}{S}I^{**} - \lambda\beta\frac{V^{**2}S^{**}}{V}I^{**} \right. \\
& \left. + \lambda\beta(1-\xi)VV^{**}I^{**} - \lambda\beta(1-\xi)V^{**2}I^{**} \right]
\end{aligned}
$$

$$
+ (1-\xi)\left[\lambda\beta SV^{**}I\frac{E^{**}}{E} - \lambda\beta SIV\frac{E^{**}}{E} + \lambda\beta(1-\xi)VV^{**}I\frac{E^{**}}{E} - \lambda\beta(1-\xi)V^2I\frac{E^{**}}{E} \right]
$$

$$
+ (1-\xi)\left[\left(2V\mu S^{**} - V\mu S - \frac{V\mu S^{**2}}{S} \right) - \left(2\mu V^{**}S^{**} - \mu V^{**}S - \frac{\mu V^{**}S^{**2}}{S} \right) \right]
$$

$$
+ (1-\xi)\left[V(\mu+\sigma)\left(E^{**} - E \right) + V^{**}(\mu+\sigma)\left(E - E^{**} \right) \right]
$$

$$
+ (1-\xi)\left[(\mu+\omega)\left(2VV^{**} - V^2 - V^{**2} \right) \right] + (\mu+\omega)\left[2S^{**}V^{**} - VS^{**} - \frac{V^{**2}S^{**}}{V} \right]
$$

$$
= \lambda\beta(1-\xi)S^{**}V^{**}I^{**}\left[\frac{V}{V^{**}} - \frac{V}{V^{**}}\frac{S^{**}}{S} + \frac{S^{**}}{S} - \frac{V^{**}}{V} + (1-\xi)\frac{V}{S^{**}} - (1-\xi)\frac{V^{**}}{S^{**}} \right]
$$

$$
+ \lambda\beta(1-\xi)SVI\left[\frac{V^{**}}{V} - 1 + (1-\xi)\frac{V^{**}}{S} - (1-\xi)\frac{V}{S} \right]\frac{E^{**}}{E}
$$

$$
+ \mu(1-\xi)VS^{**}\left[2 - \frac{S}{S^{**}} \right] - \mu(1-\xi)V^{**}S^{**}\left[2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \right] + (1-\xi)(\mu+\sigma)VE^{**}\left[1 - \frac{E}{E^{**}} \right]
$$

$$
+ (1-\xi)(\mu+\sigma)V^{**}E^{**}\left[2 - \frac{V}{V^{**}} - \frac{V^{**}}{V} \right] + (\mu+\omega)V^{**}S^{**}\left[2 - \frac{V}{V^{**}} - \frac{V^{**}}{V} \right]
$$

It can be shown that

$$
(1-\xi)\frac{V}{S^{**}} - \frac{(1-\xi)V^{**}}{S^{**}} = 0
$$

And

$$
(1-\xi)\frac{V^{**}}{S} - (1-\xi)\frac{V}{S} = 0
$$

Hence we have

$$
\frac{dL}{dt} \leq \mu(1-\xi)VS^{**}\left[2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \right] - \mu(1-\xi)V^{**}S^{**}\left[2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \right]
$$

$$
+ (1-\xi)(\mu+\sigma)VE^{**}\left[1 - \frac{E}{E^{**}} \right] + (1-\xi)(\mu+\sigma)V^{**}E^{**}\left[\frac{E}{E^{**}} - 1 \right]
$$

$$
+ (1-\xi)(\mu+\omega)VV^{**}\left[2 - \frac{V}{V^{**}} - \frac{V^{**}}{V} \right] + (\mu+\omega)V^{**}S^{**}\left(2 - \frac{V}{V^{**}}\frac{V^{**}}{V} \right)
$$

Finally, since the arithmetic mean exceeds the geometric mean, then

$$
\left(2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \right) \leq 0
$$

$$
\left(2 - V - \frac{V}{V^{**}} \right) \leq 0
$$

Furthermore, since all the model parameters are non-negative, it follows that

$\dfrac{dL}{dt} \leq 0$ for $R_0 > 1$. Thus L is aLyapunov function of system (4) on Ω.

Therefore, it follows from Lasalle's Invariance Principle that

$$\lim_{t \to \infty} S(t) = S^{**}$$

$$\lim_{t \to \infty} V(t) = V^{**}$$

$$\lim_{t \to \infty} E(t) = E^{**}$$

$$\lim_{t \to \infty} I(t) = I^{**}$$

$$\lim_{t \to \infty} R(t) = R^{**}$$

3.8. Basic Concept of Variational Iteration Method

We consider the general nonlinear system

$$L[u(x)] + N[u(x)] = \psi(x)$$

Where L is linear operator, N is nonlinear operator and $\psi(x)$ is a given continuous function.

According to the variational iteration method He (1999) and He et al. (2006), we can construct a correction functional in the form;

$$u_{k+1}(x) = u_k(x) + \int_0^t \lambda(s)\left[L[u_k(s)] + N[\tilde{u}_k(s)] - \psi(x)\right]ds \tag{20}$$

Where $u_0(x)$ is an initial approximation with possible unknowns, λ is the Lagrange multiplier which can be determined optimally via variational theory, the subscript k denotes the kth approximation and $\tilde{u}_k$ is considered as a restricted variation such that $\delta\tilde{u}_k = 0$. It is shown that this method is very effective and easy for a linear problem, its exact solution can be obtained by only the first iteration because λ can be exactly identified.

Hence to solve the above equation, we proceed by considering the stationary condition of the correction functional, then the Lagrange multiplier λ becomes,

$$\lambda(s) = \frac{(-1)^m}{(m-1)!}(s-x)^{m-1} \tag{21}$$

where m is the highest order of the differential equation.
As a result, we have the iteration formula as;

$$u_{k+1}(x) = u_k(x) + \int_0^t \frac{(-1)^m}{(m-1)!}(s-x)^{m-1}\left[L[u_k(s)] + N[\tilde{u}_k(s)] - \psi(x)\right]ds \tag{22}$$

3.9. Implementation of Variational Iteration Method on SVEIR Model

First we consider the SVEIR model (4), we then apply Variational Iteration Method (VIM) by constructing the correction functional of each of the equations of the system, and we obtained the following;

$$S_{n+1} = S_n + \int_0^t \lambda_1(t)\left[\frac{dS_n}{dt} - (1-p)\Lambda - \omega V_n - \rho R_n + \frac{\beta \tilde{S}_n \tilde{I}_n}{1+\alpha I} + \mu S_n\right]dt \tag{23}$$

$$V_{n+1} = V_n + \int_0^t \lambda_2(t)\left[\frac{dV_n}{dt} - p\Lambda + \frac{\beta(1-\xi)I_n V_n}{1+\alpha I} + (\omega+\mu)V_n\right]dt \tag{24}$$

$$E_{n+1} = E_n + \int_0^t \lambda_3(t)\left[\frac{dE_n}{dt} - \frac{\beta I_n S_n}{1+\alpha I} - \frac{\beta(1-\xi)I_n V_n}{1+\alpha I} + (\sigma+\mu)E_n\right]dt \tag{25}$$

$$I_{n+1} = I_n + \int_0^t \lambda_4(t)\left[\frac{dI_n}{dt} - \sigma E_n + (\mu + \varepsilon + \gamma)I_n\right]dt \tag{26}$$

$$R_{n+1} = R_n + \int_0^t \lambda_5(t)\left[\frac{dR_n}{dt} - \gamma I_n + (\rho + \mu)R_n\right]dt \tag{27}$$

Where λ_i , $i = 1, 2, ... 5$ are the Lagrangemultiplier which can be identified optimally via variational Theory.

we now obtain the optimal values of λ_i

$$\lambda_i(t) = \frac{(-1)^m}{(m-1)!}(t-x)^{m-1}, \quad i = 1, 2, 3, 4, 5 \tag{28}$$

m is the highest order of the differential equations.

x is the compartments

Hence we have

$$\lambda_1(t) = \frac{(-1)^1}{(1-1)!}(t-S)^{1-1} = \frac{-1}{0!}(t-S)^0 = -1$$

$$\lambda_2(t) = \frac{(-1)^1}{(1-1)!}(t-V)^{1-1} = \frac{-1}{0!}(t-V)^0 = -1$$

$$\lambda_3(t) = \frac{(-1)^1}{(1-1)!}(t-E)^{1-1} = \frac{-1}{0!}(t-E)^0 = -1$$

$$\lambda_4(t) = \frac{(-1)^1}{(1-1)!}(t-I)^{1-1} = \frac{-1}{0!}(t-I)^0 = -1$$

$$\lambda_5(t) = \frac{(-1)^1}{(1-1)!}(t-R)^{1-1} = \frac{-1}{0!}(t-R)^0 = -1$$

Therefore, our iteration formulae become;

$$S_{n+1} = S_n - \int_0^t\left[\frac{dS_n}{dt} - (1-p)\Lambda - \omega V_n - \rho R_n + \frac{\beta \tilde{S}_n \tilde{I}_n}{1+\alpha I} + \mu S_n\right]dt \tag{29}$$

$$V_{n+1} = V_n - \int_0^t\left[\frac{dV_n}{dt} - p\Lambda + \frac{\beta(1-\xi)I_n V_n}{1+\alpha I} + (\omega + \mu)V_n\right]dt \tag{30}$$

$$E_{n+1} = E_n - \int_0^t\left[\frac{dE_n}{dt} - \frac{\beta I_n S_n}{1+\alpha I} - \frac{\beta(1-\xi)I_n V_n}{1+\alpha I} + (\sigma + \mu)E_n\right]dt \tag{31}$$

$$I_{n+1} = I_n - \int_0^t \lambda_4(t)\left[\frac{dI_n}{dt} - \sigma E_n + (\mu + \varepsilon + \gamma)I_n\right]dt \tag{32}$$

$$R_{n+1} = R_n - \int_0^t \lambda_5(t)\left[\frac{dR_n}{dt} - \gamma I_n + (\rho + \mu)R_n\right]dt \tag{33}$$

With the initial approximation $S_0(t) = 25$, $V_0(t) = 20$, $E_0(t) = 15$, $I_0(t) = 10$, $R_0(t) = 0$ which satisfy the initial conditions, to give;

$$S_1(t) = 25 + 7.57218t$$
$$V_1(t) = 20 + 1.8944104t$$
$$E_1(t) = 15 + 0.4470096\,t$$
$$I_1(t) = 10 - 3.8516\,t$$
$$R_1(t) = 0.714\,t$$

$$S_2(t) = 25 + 7.57218t + 3.428031601t^2 + 0.0133925719t^3$$
$$V_2(t) = 20 + 1.8944104t - 0.01980016004t^2 + 0.003283546738t^3$$
$$E_2(t) = 15 + 0.4470096t - 0.05587241364t^2 - 0.01667611864t^3$$
$$I_2(t) = 10 - 3.8516t + 0.7758508925t^2$$
$$R_2(t) = 0.714\,t - 3.89337352t^2$$

$$S_3(t) = 25 + 7.57218t + 3.428031601t^2 - 10.95779782t^3 + 0.02528127049t^4$$
$$- 0.0007185722909t^5 - 0.000002385690683t^6$$
$$V_3(t) = 20 + 1.8944104t - 0.01980016004t^2 - 0.003199072356t^3 - 0.00058403143211t^4$$
$$0.0000075626666608t^5 - 5.73214805\times10^{-7}t^6$$
$$E_3(t) = 15 + 0.4470096t - 0.05587241364t^2 + 0.01507381852t^3 - 0.001898403052t^4$$
$$+ 0.0007110096243t^5 + 0.000002958908163t^6$$
$$I_3(t) = 10 - 3.8516t + 0.7758508925t^2 - 0.104064693t^3 - 0.0004569256508t^4$$
$$R_3(t) = 0.714\,t - 3.89337352t^2 + 10.9640184t^3$$

3.10. Basic Concept and Implementation of ADM on SVEIR Model

In this section, we determine the solution of model (4) using Adomain Decomposition Method (ADM).
Let us consider system (4) in an operator form;

$$\left.\begin{aligned}
LS &= (1-\rho)\Lambda + \omega\Lambda + \rho\Lambda - \lambda\beta SI - \mu S \\[6pt]
LV &= \rho\Lambda - \lambda\beta(1-\xi)VI - (\mu+\omega)V \\[6pt]
LE &= \lambda\beta SI + \lambda\beta(1-\xi)VI - (\mu+\sigma)E \\[6pt]
LI &= \sigma E - (\mu+\varepsilon+\gamma)I \\[6pt]
LR &= \gamma I - (\mu+\rho)R \\[6pt]
&\text{where } L = \frac{d}{dt}
\end{aligned}\right\} \tag{34}$$

By applying the inverse operator $L^{-1} = \int_0^t (.)\,dt$ on both sides of (**), we have

$$
\begin{cases}
S(t) = S(0) + (1-p)\Lambda t + \omega \int_0^t V dt + \rho \int_0^t R dt - \lambda\beta \int_0^t SI dt - \mu \int_0^t S dt \\[2em]
V(t) = V(0) + pVt - \lambda\beta(1-\xi)\int_0^t VI dt - (\mu+\omega)\int_0^t V dt \\[2em]
E(t) = E(0) + \lambda\beta\int_0^t SI dt + \lambda\beta(1-\xi)\int_0^t VI dt - (\mu+\sigma)\int_0^t E dt \\[2em]
I(t) = I(0) + \sigma\int_0^t E dt - (\mu+\varepsilon+\gamma)\int_0^t I dt \\[2em]
R(t) = R(0) + \gamma\int_0^t I dt - (\mu+\rho)\int_0^t R dt
\end{cases}
\tag{35}
$$

Applying Adomain Decomposition Method, the solution of the equation (35) above becomes sum of the following series; The linear terms

$$
S = \sum_{n=0}^{\infty} S_n ; V = \sum_{n=0}^{\infty} V_n ; E = \sum_{n=0}^{\infty} E_n ; I = \sum_{n=0}^{\infty} I_n ; R = \sum_{n=0}^{\infty} E_n
$$

And we approximate the non-linear terms as follows

$$
SI = \sum_{n=0}^{\infty} A_n(S_0, \ldots, S_{n; I_0}, \ldots, I_n)
$$

$$
VI = \sum_{n=0}^{\infty} B_n(V_0, \ldots, V_n; I_0, \ldots, I_n)
$$

Where

$$
A_n = \frac{1}{n!}\left[\frac{d^n\left(\sum_{n=0}^{\infty} S_k \lambda^k\right)\left(\sum_{n=0}^{\infty} I_k \lambda^k\right)}{d\lambda^n}\right]_{\lambda=0}
\tag{36}
$$

and

$$
B_n = \frac{1}{n!}\left[\frac{d^n\left(\sum_{n=0}^{\infty} S_n \lambda^k\right)\left(\sum_{n=0}^{\infty} I_n \lambda^k\right)}{d\lambda^k}\right]_{\lambda=0}
\tag{37}
$$

Are the Adomain polynomials
Now, substituting A_n, B_n and the linear terms in (20), we have
Hence from the above equation (21) we define the following initial conditions and recursive formula

$$S_0 = S(0) + (1-p) \wedge t, V_0 = V(0) + p \wedge t, E_0 = E(0), I_0 = I(0), R_0 = R(0)$$

$$S_{n+1} = \omega \int_0^t V_n dt + \rho \int_0^t R_n dt - \lambda \beta \int_0^t A_n dt - \mu \int_0^t S_n dt$$

$$S_{n+1} = \lambda \beta (1-\xi) \int_0^t B_n dt - (\mu+\omega) \int_0^t V_n dt$$

$$E_{n+1} = \lambda \beta \int_0^t A_n dt + \lambda \beta (1-\xi) \int_0^t B_n dt - (\mu+\sigma) \int_0^t E_n dt \qquad (38)$$

$$I_{n+1} = \sigma \int_0^t E_n dt - (\mu+\xi+r) \int_0^t I_n dt$$

$$R_{n+1} = r \int_0^t I_n dt - (\mu+\rho) \int_0^t R_n dt$$

We compute the Adomain polynomial with help of Maple; and obtained

$$A_0 = S_0 I_0$$
$$A_1 = S_0 I_1 + S_1 I_0$$
$$A_2 = S_0 I_2 + S_1 I_1 + S_2 I_0$$
$$A_3 = S_0 I_3 + S_1 I_2 + S_2 I_1 + S_3 I_0$$
$$A_4 = S_0 I_4 + S_1 I_3 + S_2 I_2 + S_3 I_1 + S_4 I_0$$
$$A_5 = S_0 I_5 + S_1 I_4 + S_2 I_3 + S_3 I_2 + S_4 I_1 + S_5 I_0$$

$$B_0 = V_0 I_0,$$
$$B_1 = V_0 I_1 + V_1 I_0$$
$$B_2 = V_0 I_2 + V_1 I_1 + V_2 I_0$$
$$B_3 = V_0 I_3 + V_1 I_3 + V_2 I_2 + V_3 I_1 + V_4 I_0$$
$$B_4 = V_0 I_5 + V_1 I_4 + V_2 I_3 + V_3 I_2 + V_4 I_1 + V_5 I_0$$

$$S_1 = 0.8926t + 0.059216635t^2$$
$$V_1 = -1.5160096t + 0.1.1292557365t^2$$
$$E_1 = 0.4470098t + 0.06903010054t^2$$
$$I_1 = -3.85160t$$
$$R_1 = 0.7140t$$

$$S_2 = 3.368814965t^2 + 0.008862406378t^3$$
$$V_2 = 0.1094555764t^2 + 0.009177100611t^3$$
$$E_2 = -0.1249025142t^2 - 0.01829162969t^3$$
$$I_2 = 0.7758508926t^2 + 0.0002521899673t^3$$
$$R_2 = -3.493373520t^2$$

$$S_3 = -10.96666022t^3 - 0.001596915911t^4 - 4.641197940\times10^{-7}t^4$$

$$V_3 = -0.01237617297t^3 - 0.001236686070t^4 - 2.322281588\times10^{-7}t^5$$

$$E_3 = 0.03336544822t^3 + 0.002883733653t^4 + 6.963479520\times10^{-7}t^5$$

$$I_3 = -0.1043168833t^3 - 0.00007543893886t^4$$

$$R_3 = 11.04999716t^3 + 0.00002531987271t^4$$

4. Simulation (Numerical Solution) and Results

Here in this section, we present, the numerical simulation using some estimated parameters from the literature and others from noted secondary sources to showcase the analytical results. The simulation of the S. Pneumonia was done using MATLAB with the sole aim of investigating the effect or the contribution of the different parameters to the spread of the S. Pneumonia infection and their mitigation.

Table 2. *Parameter estimates for S. pneumonia model under interventions.*

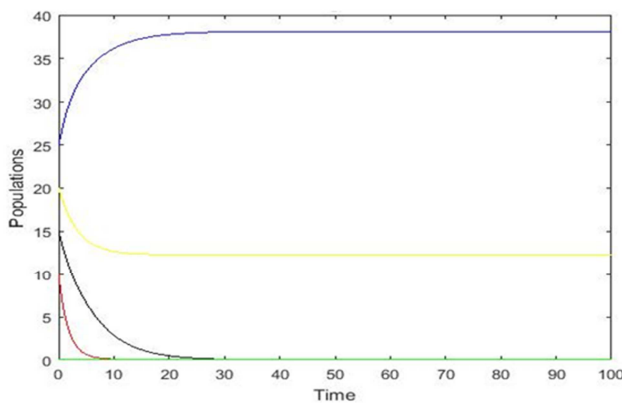| Symbols & Parameters	Description	Value	Source
S	Susceptible Individuals	25	Estimated
V	Vaccinated Individuals	20	Estimated
E	Exposed individuals	15	Estimated
I	Infectious individuals	10	Estimated
R	Recovered individuals	0	Estimated
μ	Per capita natural mortality rate	0.2	Estimated
β	Contact rate	0.0287	Mohammed and Tumwiine (2018)
ξ	Effectiveness of vaccine	0.3	Estimated
p	Fraction of recruited individuals	0.32	Mohammed and Tumwiine (2018)
σ	Infectious rate of exposed individuals	0.01096	Mohammed and Tumwiine (2018)
γ	recovery rate of infected individuals	0.0714	Mohammed and Tumwiine (2018)
ω	The rate at which vaccines wane	0.0621	Mohammed and Tumwiine (2018)
Λ	Recruitment rate into the susceptible compartment	10.09	Mohammed and Tumwiine (2018)
α	Proportion of antibody produced by individual in response to the incidence of infection	2	Liu and Yang (2012)
ρ	Per capita rate of recovered individuals going back to the Susceptible class	9.4	Mohammed and Tumwiine (2018)

Figure 2. *Plot of Population against time.*

The graph below shows the trend of all the population against time when the basic reproduction number is less than one. The result show that; as can be seen from that the infected class I (t) is drastically reduces to zero (0) in the long run when the basic reproduction number is less than unity (that is $\Re_0 = 0.1154 < 1$). While the vaccinated V (t) and susceptible S (t) remains in the population.
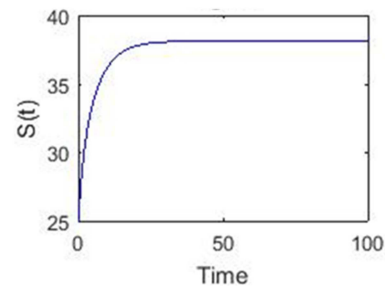

Figure 3. *Susceptible Population againsttme.*
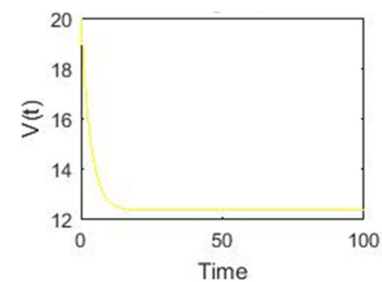

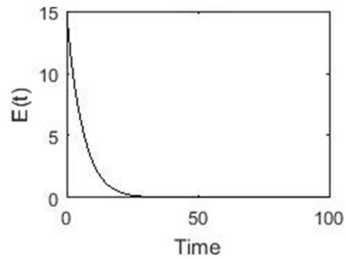**Figure 4.** *Vaccinated Pop. against time.*

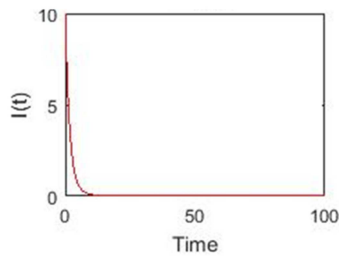*Figure 5. Plot of Exposed Pop against time.*

Figure 6. Plot of InfectedPop. against time.

The graphs above explicitly describe the trend of each of the classes against time when the basic reproduction number is less than unity. It is clear from the above trend that both the exposed population and the infected population are reduced to zero. This shows that our system is stable when the basic reproduction number is less than one (1).

While the vaccinated population decrease but remain constant at a particular time. The explanation to this is as a result of fear and lack of awareness of the vaccination campaign against S. pneumonia. But after sometimes people are aware of the vaccination which maintains the population at that static level.

Figure 7. Plot of Population against time.

Here in this graph we have the five classes and their trend over time when $\Re_0 = 1.1864 > 1$, it was revealed from the simulation that the infected population decreases but did not get to zero, which means that there are individuals who are still living with S. Pneumonia in the population. Our simulation further shows that within the period of 10days, the number of infected individual reduces drastically and then increases within 45 to 50 day slowly.
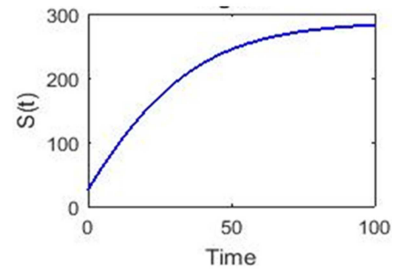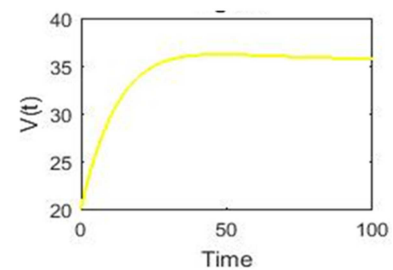

Figure 8. Susceptible Popu. Againsttime.

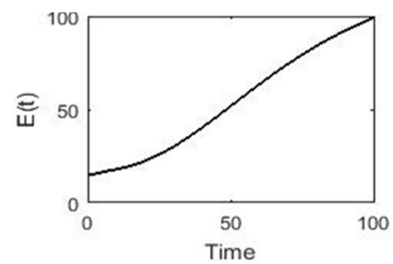
Figure 9. Vaccinated Pop. Againsttime.

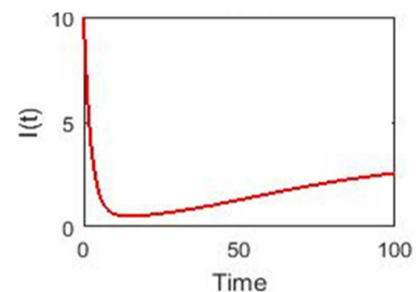*Figure 10. Exposed Pop. Against time.*

Figure 11. Infected Popu. Against time.

In the above simulation, we describe the trend of each of the classes to understand exactly what happened in Figure 9 and it reveals that, if the basic reproduction is greater than one, S. pneumonia becomes endemic in the population and persist since the exposed and infected classes were not reduced to zero, see Figure 10 *and* Figure 9.

Here we simulate the effect of proportion of antibody induced by the vaccine. Our simulation reveals that increase in α which is proportion of antibody produced by individual in response to the incidence of Infection, reduces the infected population.
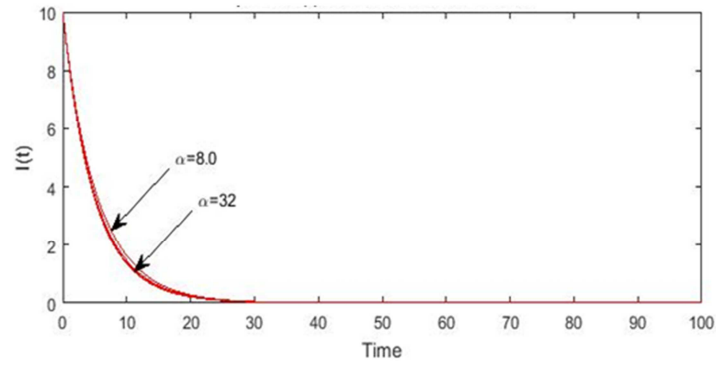
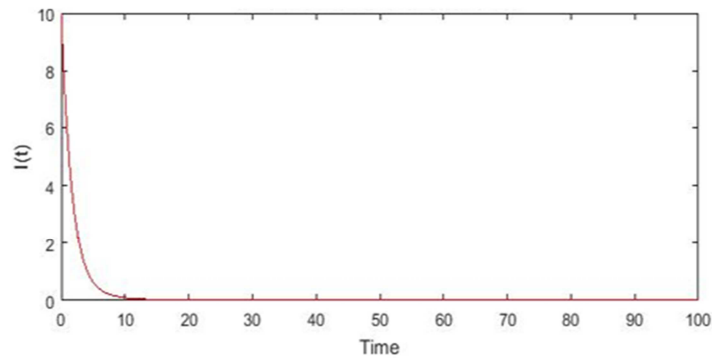**Figure 12.** *Plot of Infected population against time for different values α.*

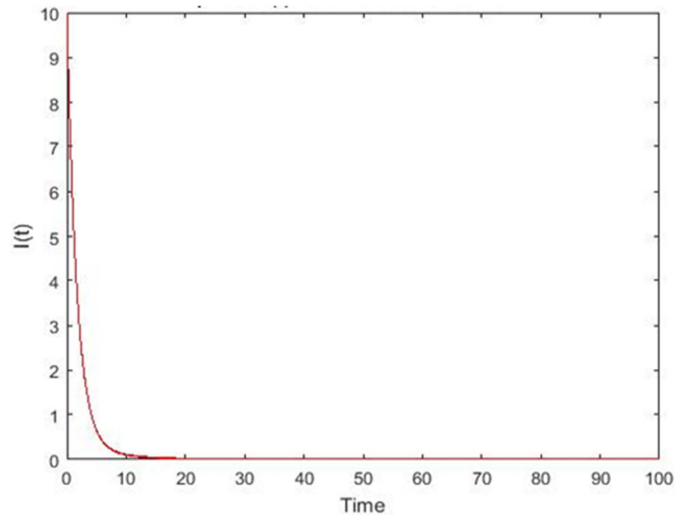**Figure 13.** *Plot of Infected population aginst time for different values of ω.*

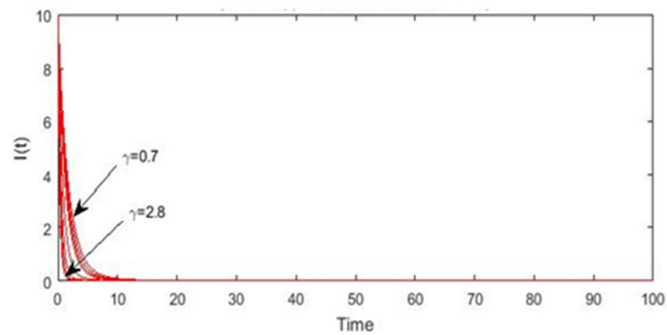**Figure 14.** *Plot of Infected population against time for different values of Λ.*

Figure 15. *Plot of Infected pop againsttime for different values of γ.*

Our simulation in the two graphs above, we observed that ω and Λ little or no variation in the infected population. The implication of this is that as more individuals with high proportion of antibody against S. pneumonia is recruited into the population, there is no need vaccinating them again, since their immune system is already in fighting against S. pneumonia. That is to say that an individual needs vaccination before he/she will be introduced into the population. Also, we see from the simulation above that increase in recovery rate γ reduces the number of infected individual over time.
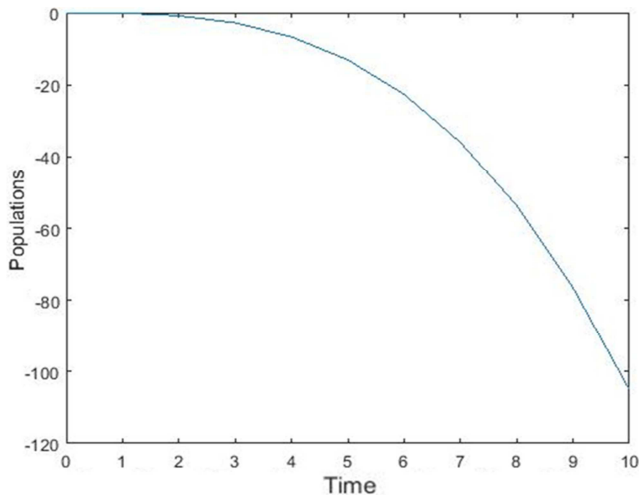

Figure 16. Plot of Population against time for VIM.
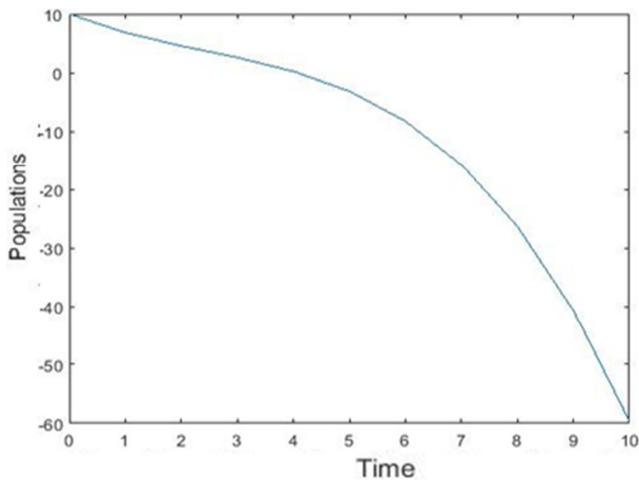

Figure 17. Plot of Population against time for ADM.

Figure 16 and Figure 17 shows the simulation for the Variational iteration Method (VIM) and Adomian Decomposition method (ADM) respectively. It was seen that in Figure 15, within the first 3 days the infection is still in the population until, the 4th day when the number of infected individual is reduced to zero. While Figure 17 shows that from the 1st day the infection is completely out of the population. Hence, it shows that the ADM converges faster than the VIM.

5. Summaries, Conclusion and Recommendation

5.1. Summary

In this paper, the dynamics of SVEIR model with saturated incidence force of infection and saturated vaccination function for Streptococcus pneumonia (that is, model that monitors the temporal transmission dynamics of the disease in the presence of preventive vaccine) was formulated and analyzed. The basic reproduction number that determines disease extinction and disease survival was revealed. The existing threshold conditions of all kinds of the equilibrium points are obtained and proved to be locally asymptotic stable for disease-free equilibrium using linearization method and Lypanov functional method for Endemic equilibrium. Qualitative Analysis of the model was obtained and the positive of solution obtained. It was revealed that the model is positively –invariant and attracting. Thus Ω is positively invariant. Hence, it is sufficient to consider the dynamics of the model (1) in Ω. In this region, the model can be considered as been epidemiologically and mathematically well-posed, Hethcote (2000). The governing model was normalized and also Adomain Decomposition method was used to compute an approximate solution of the non-linear system of differential equations governing the model. Maple was used in carrying out the simulations (numerical solutions) of the model. Graphical results were presented and discussed to illustrate the solution of the problem. The achieved results reveal that the disease will die out within the community if the vaccination coverage is above the critical vaccination proportion. The study indicates that we should improve the efficiency and enlarge the capacity of the treatment to control the spread of disease.

5.2. Conclusion

It is revealed that the DFE is globally asymptotically stable while the endemic equilibrium is not feasible which implies that the disease will be eradicated out of the population If $\Re_0 < 1$, . Furthermore, we can also see that if $\Re_0 > 1$ and $H > 0$, then endemic equilibrium is globally asymptotically stable. In order to regulator the disease, it will be strategical to decrease the BRN to barest minimum. From the manifestation of BRN $\Re_0$, it is obvious that the rate ξ representing vaccine efficiency, the rate/transfer of individual out of the infected compartment v_i^- and $(1-\xi)$ revealing the rate at which the vaccine get waned directly or indirectly impact the value of $\Re_0$. Obviously, if ξ, v_i^- or $(1-\xi)$ increase, implies $\Re_0$ decreases. Therefore, the need for public health interventions to control the epidemic by ensuring these parameters ξ, v_i^- or $(1-\xi)$ are increased to reduce $\Re_0$ can never be over emphasis.

5.3. Recommendation

Research has shown that Invasive disease attributable to *Sp* is a major public health problem for under aged children

irrespective of high use of the 7-valent pneumococcal conjugate vaccine (PCV7) in Nigeria and other African countries.

The epidemiology of human population especially those concerning under age children requires urgent and serious investigation so as to understand the diseases and proffer solutions that will completely eradicate it from our population. This could be achieved through

1. International partnership and research collaboration.
2. Improve funding of epidemiological research programs.
3. Advocacy and awareness creation among rural and urban communities.
4. Efficient and effective specialist vaccination centers in rural and urban communities.
5. Free and effective vaccination of under age children.

Thus the need for a greater understanding of Streptococcus Pneumonia and for more effective vaccination, treatment and control program is paramount to eradication of the infection. Therefore, we consent the effort of the US Food and Drug Administration (February 24, 2010) that licensed a new 13-valent pneumococcal polysaccharide-protein conjugate vaccine (PCV13) for under age children.

Hence we would also recommend from the above knowledge:

1. Healthy children and their counterparts (both those who have completed the previous vaccine PCV7) with other health challenging issues exposing them to high risk of IPD should be routinely immunize with PCV13.
2. "Catch-up" immunization should be conducted for children behind schedule; and,
3. There should be timely treatment and vaccination for infected individuals and those with compromised immunity including newborn babies respectively.

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