

Research Article

Global Stability of Pneumococcal Pneumonia with Awareness and Saturated Treatment

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Pneumococcal pneumonia, a secondary bacterial infection that follows influenza A infection, is responsible for morbidity and mortality in children, elderly, and immunocompromised groups. A mathematical model to study the global stability of pneumococcal pneumonia with awareness and saturated treatment is presented. The basic reproduction number, R_0 , is computed using the next generation matrix method. The results show that if $R_0 < 1$, the disease-free steady state is locally asymptotically stable; thus, pneumococcal pneumonia would be eradicated in the population. On the other hand, if $R_0 > 1$ the endemic steady state is globally attractive; thus, the disease would persist in the population. The quadratic-linear and Goh–Volterra Lyapunov functionals approach are used to prove the global stabilities of the disease-free and endemic steady states, respectively. The sensitivity analysis of R_0 on model parameters shows that, it is positively sensitive to the maximal effective rate before antibiotic resistance awareness, rate of relapse encountered in administering treatment, and loss of information by aware susceptible individuals. Contrarily, the sensitivity analysis of R_0 on model parameters is negatively sensitive to recovery rate due to treatment and the rate at which unaware susceptible individuals become aware. The numerical analysis of the model shows that awareness about antibiotic resistance and treatment plays a significant role in the control of pneumococcal pneumonia.

1. Introduction

Pneumonia is one of the severe forms of pneumococcal diseases caused by pneumococcus [1]. Pneumococcal pneumonia is preventable through vaccination, diagnostic testing, environmental control measures, and appropriate treatment [2]. However, treatment of pneumococcal pneumonia has become tricky due to antibiotic selection that increases resistance of *Streptococcus pneumoniae* to penicillin and the successive evolution of resistance to numerous classes of antibiotics [3]. Bacteria develop resistance against antibiotics, causing severe illness to an individual. Ultimately, it requires costly treatment to control and eradicate the disease [4]. The emergence of antimicrobial resistance threatens the successful treatment of pneumococcal infections [5]. Repeated and improper use of antibiotics is on an

increase and identified as the main cause of the emerging resistance [6]. In the event of acute side effects, patients tend to discard their treatment, only to return to the hospital with persistent infections of a more virulent and resistant strain of the bacteria [7]. Antibiotic resistance is a major worldwide threat to the provision of safe and effective health care. To control antibiotic resistance, vaccines have been proposed as an essential intervention, complementing improvements in antibiotic stewardship and drug pipelines [8].

In treatment of pneumonia, microorganisms occasionally persevere, emerge or reemerge despite the good clinical responses. Thus, recovered individuals may relapse and return to the infective class [9]. However, relapse may occur due to treatment failure with regard to elimination of an infecting causative agent. Recurrence of disease is a significant feature of some animal and human diseases; for

example, malaria, herpes, and tuberculosis [10]. Patients with HIV/AIDS commonly have a recurrence of pneumococcal bacteremia due to pre-existing drug resistance [11, 12]. Pneumococcal pneumonia patients infected with chronic diseases, such as HIV/AIDS, are more likely to relapse compared with individuals without chronic diseases [13].

Disease appropriate awareness in a population can control an infection effectively [14]. Enhanced levels of awareness, for instance, practice of better hygiene, voluntary quarantine, application of preventive medicine, or vaccination and avoidance of places containing large number of people, may reduce the spread and contraction of the disease [15]. Mass media awareness plays a vital role in changing behavior related to public health [16]. The spread of an infectious disease is reported by the media, such as television programs, newspapers, radio, or online social networks. Daily updates and reports about infections and mortality have a significant effect on the necessity of control of an epidemic [17]. Campaigns mainly focus on increasing an individual's knowledge about disease transmission and control measures that may reduce the likelihood of being infected [18].

Despite the application of all control methods that have been advocated for, individuals in developing countries lack information on the transmission and control of pneumococcal pneumonia. Individuals continue to practice self-medication [19, 20], which leads to delays in the administration of adequate antimicrobial treatment, increase resistance to antibiotics, and appear to increase the risk for hospital mortality [21].

Mathematical models that describe the influence of media coverage to inform individuals regarding disease and control in a population have been generalized in the forms of SIS, SIRS, SEI, and SEIR [22–25]. Media coverage, private individual information, and appropriate treatment have proven to be among control measures undertaken by health institutions and the public to reduce the spread of most infectious diseases. Models of infections that include awareness have been studied widely; for instance, Saha and Roy [26] performed a comparative study between two systems with and without awareness in controlling HIV/AIDS. The study revealed that aware populations were less vulnerable to HIV infection than the unaware population. Roy et al. [27] studied the effect of awareness programs in controlling the disease HIV/AIDS and found out that increased awareness campaign during high infection is likely to delay the inception of infection among aware compared with unaware population.

In this paper, we propose and analyze a mathematical model for pneumococcal pneumonia disease with awareness about antibiotic resistance and saturated treatment. The aim of this study is to investigate the impact of awareness about antibiotic resistance and treatment on the incidence and control of pneumococcal pneumonia in the human population. We consider a model developed by [2] and extend it by introducing new compartments of susceptible and infective individuals. That is, we subdivide the compartment of susceptible into aware individuals and unaware individuals

and the infectious into infected individuals receiving treatment and infected individuals resistant to first line treatment. A relapse of resistant individuals, a modified saturated treatment, and a reduced disease transmission rate with the effect of antibiotic resistance awareness through media are also added.

The remaining part of this paper is organized as follows. In Section 2, materials and methods of a mathematical model are presented. In Section 3, the basic reproduction number and local stability of the steady states of the model are presented. We present global stability of the steady states in Section 4 and give the numerical results, discussion, and conclusion in Section 5.

2. Materials and Methods

2.1. Model Description and Formulation. The total population under consideration is $N(t)$, comprising four classes with the susceptible and infectious individuals, each partitioned into two. The susceptible class consists of the aware susceptible individuals $S_a(t)$ who have had a chance to attend the available antibiotic resistance awareness programs and the unaware susceptible individuals $S_u(t)$ who have never heard of the prevailing programs or have heard of the existing programs but have not responded. The infectious class consists of infected individuals receiving treatment $I(t)$ and infected individuals but resistant to first line treatment $R(t)$. It is assumed that, in a population, antibiotic resistance awareness is disseminated by private individuals and the media at rates v and m , respectively. Furthermore, we assume that all classes are decreased by a natural mortality rate μ .

Assuming all new recruitments (through birth and immigrants) to be unaware, it is further assumed that aware susceptible individuals transfer to unaware susceptible class due to loss of memory or social factors. The unaware susceptible class is increased through a constant recruitment B , and fading of information by aware susceptible individuals is at a rate ξ . The infection is spread through the interaction of infected and susceptible individuals. We consider a reduced incidence rate of the form $g(I) = (\beta I - \beta_1(mI/m + I))$, where $m > 0$ is the effect of media coverage on the contact transmission [28], $\beta > 0$ is the maximal effective contact rate before awareness, and $\beta_1 > 0$ is the maximal reduced effective contact rate due to media alert in the presence of infective individuals. The transmission term has been considered because in real life, every individual will take precaution to protect themselves from infections as soon as infected individuals with antibiotic resistant bacteria have been identified/reported in a wholly susceptible population which will reduce the disease spread [22]. Due to the fact that the coverage report about antibiotic resistance against existing treatment does not prevent spread of disease completely, we assume $\beta \geq \beta_1 > 0$. The unaware susceptible individuals transfer to the aware class after receiving information through private individuals at a rate v .

Transmission of the infection is assumed to be governed by the bilinear incidence term $\beta_2 S_a I$ with $\beta_2 > 0$

being the contact rate between aware individuals and infected individuals. The infected class is increased through the relapse of resistant individuals at a rate γ_1 . It is assumed that infected individuals upon receiving the first line of treatment tend not to complete the prescribed medication and develop resistant bacteria that may require costly treatment to be eliminated. A saturated treatment is considered because there exist delays in administering treatment by infected individuals, and the medical resources may be limited. Suppose that infected individuals are immediately treated with first line of treatment at a rate ϕ and are either cleared of the pneumococcus or acquires resistance. Then, we modify the saturated treatment proposed by Zhang and Liu [29] by incorporating $\tau = 1/1 + \Phi D$, which describes the effect of infected individuals delaying to seek treatment [30], where D is the number of days delayed in taking treatment. Thus, the saturated treatment we use in our model is of the form $T(I) = \Phi I/1 + \tau I$. We remark, thus, if $\tau = 0$, the saturated treatment function becomes linear, that is, $T(I) \approx \phi I$, if the number of infected individuals is minimal $T(I) \approx 0$, and if the number of infected individuals grows large, $T(I) \approx \Phi$. The probability of acquiring resistance to first line treatment is p . Finally, the rate of resistance acquisition upon treatment with the antibiotic drugs is $p\phi$, and a fraction of infected individuals respond to treatment and recover from the disease and thus transfer to the aware class at a clearance rate $(1 - p)\Phi$.

Individuals who are treated incorrectly or do not take the right dose at the prescribed time become resistant to first line treatment, and a correct treatment is administered after a delay period τ . We assume resistance is gained through mutations, due to exposure to antibiotics [31].

Descriptions of state variables and epidemiological parameters are summarized in Tables 1 and 2.

With the abovementioned assumptions, variables, parameters, and the transition diagram in Figure 1, the dynamics of pneumococcal pneumonia is governed by the system of nonlinear ordinary differential equations:

$$\begin{aligned} \dot{S}_u &= B + \xi S_a - (g(I) + vI + \mu)S_u, \\ \dot{S}_a &= vIS_u + (1 - p)\Phi T(I) - (\beta_2 I + \xi + \mu)S_a, \\ \dot{I} &= g(I)S_u + \beta_2 S_a I + \gamma_1 R - \Phi T(I) - (\mu + \delta_1)I, \\ \dot{R} &= p\Phi T(I) - (\gamma_1 + \mu)R, \end{aligned} \tag{1}$$

where $T(I) = I/1 + \tau I$ and $g(I) = (\beta I - \beta_1(mI/m + I))$, with initial conditions $S_u(0) = S_{u0} \geq 0$, $S_a(0) = S_{a0} \geq 0$, $I(0) = I_0 \geq 0$, $R(0) = R^0 \geq 0$. Since model (1), describes human populations, all state variables and parameters are assumed positive for all $t \geq 0$.

2.2. Positivity of Solution Trajectories in Model (1)

Lemma 1. *Solutions of model (1) with initial conditions $S_u(0) = S_{u0} \geq 0$, $S_a(0) = S_{a0} \geq 0$, $I(0) = I_0 \geq 0$ and $R(0) = R^0 \geq 0$ are always nonnegative for all $t \geq 0$.*

Proof. Suppose $S_u(t)$, $S_a(t)$, $I(t)$, $R(t)$ are solutions to model (1); then, from the first equation of model (1), we have

$$\frac{dS_u}{dt} = B + \xi S_a - g(I)S_u - vIS_u - \mu S_u \geq -(g(I) + vI + \mu)S_u. \tag{2}$$

By using the technique of separation of variables, we have

$$S_u(t) \geq S_{u0} \exp\left(-\left(\mu t + \int_0^t (g(I(\theta)) + vI(\theta))d\theta\right)\right) \geq 0. \tag{3}$$

Therefore, the solution of $S_u(t)$ in equation (3) is non-negative for all $t \geq 0$. Applying the same technique to the remaining variables of model (1), we get

$$S_a(t) \geq S_{a0} \exp\left(-(\xi + \mu)t + \int_0^t \beta_2 I(\theta)d\theta\right) \geq 0, \tag{4}$$

$$I(t) = I_0 \exp(-(\mu + \delta_1)t) \geq 0, \tag{5}$$

$$R(t) \geq R^0 \exp(-(\gamma_1 + \mu)t) \geq 0. \tag{6}$$

Therefore, all solutions of $S_a(t)$, $I(t)$ and $R(t)$ in equations (4)–(6) are nonnegative for all times $t \geq 0$. This completes the proof. \square

2.3. Invariant Region. Here, we obtain a region in which the solution of model (1) is bounded. For this model, the total population is $N = S_u + S_a + I + R$, such that the rate of change of the total population is

$$\frac{dN}{dt} = B - \mu N - \delta_1 I, \tag{7}$$

$$\frac{dN}{dt} \leq B - \mu N. \tag{8}$$

Equation (8) corresponds to instances of no pneumococcal pneumonia-related death. Therefore, we obtain

$$N \leq N_0 e^{-\mu t} + \frac{B}{\mu} (1 - e^{-\mu t}), \tag{9}$$

where $N_0 = S_{u0} + S_{a0} + I_0 + R^0$.

Thus, from equation (9), we have

$$N(t) \leq \max\left(N_0, \frac{B}{\mu}\right). \tag{10}$$

Then, $0 \leq S_u + S_a + I + R \leq B/\mu$. This implies that the region $D = \{(S_u, S_a, I, R) \in \mathbb{R}_+^4 : S_u + S_a + I + R = N \leq B/\mu\}$, which implies $N(t)$ is bounded and so are S_u, S_a, I , and R .

2.4. Existence and Uniqueness of the Steady States. From model (1), equating the right hand side to zero, Proposition 1 shall represent the existence and uniqueness of the endemic steady state.

TABLE 1: Description of parameters.

Parameter	Description	Value/day ⁻¹	Ref
A	Recruitment by birth/immigration	5	Assd
β	Maximal effective contact rate before antibiotic resistance awareness	0.0417	[32]
β_1	Maximal reduced effective contact rate due to media alerts	0.0046	Assd
β_2	Contact rate of aware susceptible with infective individuals	0.000007498	Assd
γ_1	Rate of relapse encountered in administering treatment	0.00145	Assd
μ	Natural mortality rate	2.0×10^{-4}	[2]
m	Efficiency of awareness through media coverage	0.5	Assd
δ	Excess death due to pneumococcal pneumonia	0.1	[33, 34]
ξ	Loss of information about disease by aware susceptible individuals	0.3	[15]
Φ	Recovery rate due to treatment	0.9	[35]
D	Number of days delayed in receiving appropriate treatment	1–14 days	[20]
τ	Rate of delay to receive appropriate treatment	0.2703	Assd
p	Probability of acquiring resistance during treatment	0 – 0.03	[30]
v	Rate at which unaware susceptible individuals become aware of antibiotic resistance	0.0029	Assd

TABLE 2: Description of state variables.

Variable	Description
$S_u(t)$	Unaware individuals
$S_a(t)$	Aware individuals
$I(t)$	Infected individuals receiving treatment
$R(t)$	Infected individuals but resistant to first line of treatment
$N(t)$	Total population ($N(t) = S_u(t) + S_a(t) + I(t) + R(t)$)

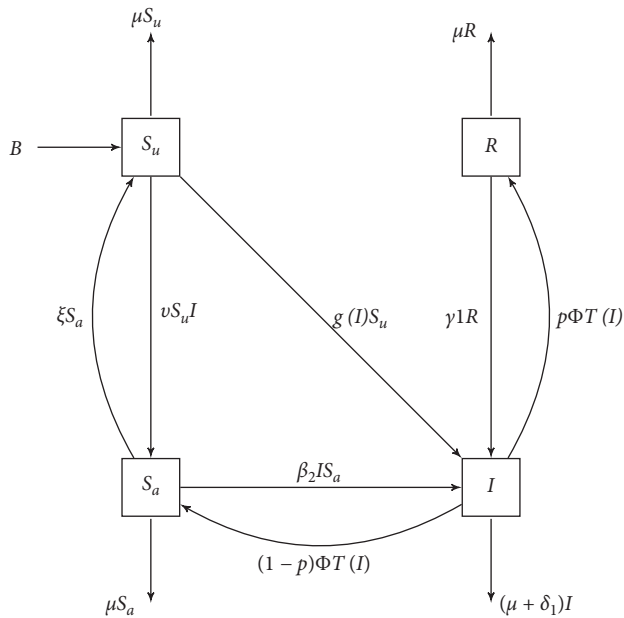


FIGURE 1: A transition diagram showing the dynamics of pneumococcal pneumonia.

Proposition 1. If conditions $(\gamma_1 + \mu)(\mu + \delta) > \Phi(\gamma_1(p-1) - \mu)$, $B > I^*d$, $(vI^* + \mu)(\beta_2 I^* + \xi + \mu) > vI^*(\beta_2 I^* + \xi)$, and $(\Phi + \mu + \delta_1)(\gamma_1 + \mu) > p\Phi\gamma_1$ hold, then model (1) will admit a unique endemic steady state, $E^* = (S_u^*, S_a^*, I^*, R^*)$ if $R_0 > 1$.

Proof. Suppose $E^* = (S_u^*, S_a^*, I^*, R^*)$ is a steady state of model (1) satisfying the following system.

$$\begin{aligned} B + \xi S_a^* - (g(I^*) + vI^* + \mu)S_u^* &= 0, \\ vI^*S_u^* + (1-p)\Phi T(I^*) - (\beta_2 I^* + \xi + \mu)S_a^* &= 0, \\ g(I^*)S_u^* + \beta_2 S_a^* I^* + \gamma_1 R^* - \Phi T(I^*) - (\mu + \delta_1)I^* &= 0, \\ p\Phi T(I^*) - (\gamma_1 + \mu)R^* &= 0. \end{aligned} \quad (11)$$

From equation (11) it follows that if we let $I^* = R^* = 0$, then we have the disease-free steady state $E_0^* = (S_u^0, S_a^0, I^0, R^0) = ((\xi + \mu)/v, (Bv - \mu\xi - \mu^2)/\mu v, 0, 0)$ that exists for all epidemiological parameter values.

On the other hand, if $I^* \neq 0, R^* \neq 0$, then we have the endemic steady state $E^* = (S_u^*, S_a^*, I^*, R^*)$, where

$$\begin{aligned} S_u^* &= \frac{(1 + \tau I^*)^2 (\beta_2 I^* + \xi + \mu) (B - I^*d) + (\beta_2 I^* + \xi) (1 - p)\Phi I^*}{K(1 + \tau I^*)}, \\ S_a^* &= \frac{vI^* (1 + \tau I^*)^2 (B - I^*d) + (1 - p)\Phi I^* (vI^* + \mu)}{K(1 + \tau I^*)}, \\ R^* &= \frac{p\Phi I^*}{(\gamma_1 + \mu)(1 + \tau I^*)}, \\ I^* = I^{**} &= \frac{-r_1 + \sqrt{(r_1^2 - 4r_2 r_0)}}{2r_2}, \end{aligned} \quad (12)$$

with

$$r_0 = m(\beta - \beta_1)S_u^* + m\left(\frac{\gamma_1 \Phi p}{\gamma_1 + \mu} + \Phi\right) - (\mu + \delta_1),$$

$$r_1 = (\beta + \tau m(\beta - \beta_1))S_u^* + \tau(\beta - \beta_1)m + \frac{\gamma_1 p \Phi}{\gamma_1 + \mu}$$

$$+ \phi - (\mu + \delta_1)(1 + \tau m),$$

$$r_2 = \tau(\beta S_u^* - (\mu + \delta_1)),$$

$$d = (\gamma_1 + \mu_1)((\gamma_1 + \mu)(\mu + \delta_1) - \Phi(\gamma_1(p-1) - \mu)),$$

$$K = (vI^* + \mu)(\beta_2 I^* + \xi + \mu) - vI^*(\beta_2 I^* + \xi).$$

(13)

Therefore, there exists a unique endemic steady state E^* , provided conditions $(\gamma_1 + \mu)(\mu + \delta_1) > \Phi(\gamma_1(p - 1) - \mu)$, $B > I^*d$, $(vI^* + \mu)(\beta_2I^* + \xi + \mu) > vI^*(\beta_2I^* + \xi)$, $r_1 < 0$, $r_2 > 0$, and $r_0 < 0$ hold. This completes the proof. \square

3. The Basic Reproductive Number and Stability of Steady States

The basic reproduction number, R_0 , is computed for model (1) by the method introduced by Van den Driessche and Watmough [36] according to which $R_0 = \rho(FV^{-1})$, where ρ is the spectral radius of a matrix (the maximum eigenvalue obtained from the matrix). Let \mathbb{F} and \mathbb{V} be vectors representing new infections and remaining transfer terms, respectively.

$$\mathbb{F} = \begin{pmatrix} g(S_u, I) + \beta_2 S_a I \\ 0 \end{pmatrix}, \tag{14}$$

$$\mathbb{V} = \begin{pmatrix} \Phi T(I) + (\mu + \delta_1)I - \gamma_1 R \\ (\gamma_1 + \mu)R - p\Phi T(I) \end{pmatrix}.$$

The infected compartments are I and R , and at the disease-free steady state, we obtain matrix Jacobian's F for \mathbb{F} and V for \mathbb{V} from (14) to obtain

$$F = \begin{pmatrix} (\beta - \beta_1)S_u^0 + \beta_2 S_a^0 & 0 \\ 0 & 0 \end{pmatrix}, \tag{15}$$

$$V = \begin{pmatrix} \Phi + \mu + \delta_1 & -\gamma_1 \\ -p\Phi & \gamma_1 + \mu \end{pmatrix}.$$

Thus, the next-generation matrix for model (1) is evaluated as

$$FV^{-1} = \begin{pmatrix} \frac{((\beta - \beta_1)S_u^0 + \beta_2 S_a^0)(\gamma_1 + \mu)}{(\Phi + \mu + \delta_1)(\gamma_1 + \mu) - p\gamma_1\Phi} & \frac{((\beta - \beta_1)S_u^0 + \beta_2 S_a^0)\gamma_1}{(\Phi + \mu + \delta_1)(\gamma_1 + \mu) - p\gamma_1\Phi} \\ 0 & 0 \end{pmatrix}. \tag{16}$$

Therefore, making substitutions of S_u^0 and S_a^0 , the basic reproduction number for model (1) is

$$R_0 = \rho(FV^{-1}) = \frac{(\gamma_1 + \mu)((\beta - \beta_1)((\xi + \mu)/v) + (\beta_2(Bv - \mu\xi - \mu^2)/\mu v))}{(\Phi + \mu + \delta_1)(\gamma_1 + \mu) - p\Phi\gamma_1}. \tag{17}$$

3.1. Local Stability Behavior of the Disease-Free Steady States

Proposition 2. *If condition $\Phi + \mu + \delta_1 > p\Phi\gamma_1$ hold, then the disease-free steady state E_0^* is locally asymptotic if $R_0 < 1$ and unstable for $R_0 > 1$.*

Proof. The Jacobian matrix of model (1) at E_0^* is given as

$$J(E_0) = \begin{pmatrix} -\mu & \xi & J_{13} & 0 \\ 0 & -(\xi + \mu) & J_{23} & 0 \\ 0 & 0 & J_{33} & \gamma_1 \\ 0 & 0 & p\Phi & -(\gamma_1 + \mu) \end{pmatrix}, \tag{18}$$

with

$$J_{13} = -\frac{(\beta - \beta_1 + v)(\xi + \mu)}{v},$$

$$J_{23} = \frac{((1 - p)\Phi + v)(\xi + \mu)}{v} - \frac{\beta_2(Bv - \mu\xi - \mu^2)}{\mu v},$$

$$J_{33} = \frac{(\beta - \beta_1)(\xi + \mu)}{v} + \frac{\beta_2(Bv - \mu\xi - \mu^2)}{\mu v} - (\Phi + \mu + \delta_1). \tag{19}$$

The characteristic polynomial of the matrix in (18) is given by

$$|J(E_0) - \lambda I| = \lambda^4 + m_3\lambda^3 + m_2\lambda^2 + m_1\lambda + m_0 = 0, \tag{20}$$

where $m_3 = ((\beta_1 - \beta)(\xi + \mu)/v) + (4\mu + \varphi + \delta_1 + \gamma_1 + \xi) - (\beta_2(Bv - \mu\xi - \mu^2)/\mu v)$, $m_2 = (\xi + \mu)((\beta_1 - \beta)(\xi + \mu)/v) -$

$(\beta_2(Bv - \mu\xi - \mu^2)/\mu v) + (\Phi + \mu + \delta_1) + (\gamma_1 + \mu) + \mu((\xi + \mu) + \beta_1 - (\beta(\xi + \mu)/v) - (\beta_2(Bv - \mu\xi - \mu^2)/\mu v) + (\Phi + \mu + \delta_1) + (\gamma_1 + \mu)) + (\gamma_1 + \mu)((\beta_1 - \beta)(\xi + \mu)/v) - (\beta_2(Bv - \mu\xi - \mu^2)/\mu v) + (\Phi + \mu + \delta_1) - \gamma_1 p \Phi m_1 = \mu(((\xi + \mu)(\beta_1 - \beta)(\xi + \mu)/v) - (\beta_2(Bv - \mu\xi - \mu^2)/\mu v) + (\Phi + \mu + \delta_1) + (\gamma_1 + \mu) + (\gamma_1 + \mu)(\xi + \mu)((\beta - \beta_1)(\xi + \mu)/v) + (\beta_2(Bv - \mu\xi - \mu^2)/\mu v) - (\Phi + \mu + \delta_1)) - \gamma_1 p \Phi, m_0 = -(\xi + \mu)\mu((\gamma_1 p \Phi + \gamma_1 + \mu)((\beta - \beta_1)(\xi + \mu)/v) + (\beta_2(Bv - \mu\xi - \mu^2)/\mu v) - (\Phi + \mu + \delta_1))$. Therefore, the characteristic roots determined from polynomial equation (20) are

$$\lambda_1 = -\mu,$$

$$\lambda_2 = -(\xi + \mu),$$

$$\lambda_3 = -\left(\frac{f}{2} + \frac{(\gamma_1 + \mu)}{2} + \frac{\sqrt{(f^2 + 2f(\gamma_1 + \mu) + 4p\gamma_1\Phi)}}{2}\right),$$

$$\lambda_4 = -\left(\frac{f}{2} + \frac{(\gamma_1 + \mu)}{2} - \frac{\sqrt{(f^2 + 2f(\gamma_1 + \mu) + 4p\gamma_1\Phi)}}{2}\right), \tag{21}$$

with

$$f = \frac{(\beta - \beta_1)(\xi + \mu)}{v} + \frac{\beta_2(Bv - \mu\xi - \mu^2)}{\mu v} - (\Phi + \mu + \delta_1). \tag{22}$$

Since all eigenvalues computed from polynomial equation (20) are negative, then we have $f/2 + ((\gamma_1 + \mu)/2) > \sqrt{(f^2 + 2f(\gamma_1 + \mu) + 4p\gamma_1\Phi)}/2$, implying that $((\gamma_1 + \mu)((\beta - \beta_1)(\xi + \mu)/v + \beta_2(Bv - \mu\xi - \mu^2)/\mu v)/(\gamma_1 + \mu)(\Phi + \mu + \delta_1) - p\Phi\gamma_1) < 1$; hence, if condition $\Phi + \mu + \delta_1 > p\Phi\gamma_1$ holds, then $R_0 = (\gamma_1 + \mu)((\beta - \beta_1)(\xi + \mu)/v + \beta_2(Bv - \mu\xi - \mu^2)/\mu v)/(\gamma_1 + \mu)(\Phi + \mu + \delta_1) - p\Phi\gamma_1 < 1$, that is, $R_0 < 1$ implies that $\lambda_4 < 0$. Thus, E_0^* is locally asymptotically stable. Furthermore, if $R_0 > 1$, then $\lambda_4 > 0$, which implies E_0^* is unstable. This completes the proof. \square

3.2. Local Stability of Endemic Steady State

Proposition 3. Suppose condition $(vI^* + \mu)(\beta_2I^* + \xi + \mu) > vI^*(\beta_2I^* + \xi)$ holds; then, the endemic steady state E^* of model (1) is locally asymptotically stable in D for $R_0 < 1$.

Proof. The variational matrix at E^* is given by

$$J(E^*) = \begin{pmatrix} a & \xi & c & 0 \\ vI^* & e & f & 0 \\ g & \beta_2I^* & h & \gamma_1 \\ 0 & 0 & l & -(\gamma_1 + \mu) \end{pmatrix}, \tag{23}$$

where; $a = -(\beta I^* - (\beta_1 m I^*/m + I^*) + vI^* + \mu)$, $f = -(p - 1)\Phi - vS_u^* + \beta_2 S_a^*$, $e = -(\beta_2 I^* + \xi + \mu)$, $c = ((\beta_1 m^2/(m + I^*))^2 + v)S_u^*$, $g = (\beta I^* - (\beta_1 m I^*/m + I^*))S_u^*$, $l = p\Phi/(1 + \tau I^*)^2$, $h = \beta_2 S_a^* + (\beta - \beta_1 m^2/(m + I^*))S_u^* - \Phi/(1 + \tau I^*)^2 - (\Phi + \mu + \delta_1)$.

The characteristic equation associated to the variational matrix (23) is given by

$$|J(E^*) - \lambda I| = \lambda^4 + b_3\lambda^3 + b_2\lambda^2 + b_1\lambda + b_0 = 0, \tag{24}$$

with $b_3 = -(a + e + h - (\gamma_1 + \mu))$, $b_2 = h(a + e) - (\gamma_1 + \mu)(h + a) + e(a - (\gamma_1 + \mu)) - (f\beta_2I^* + \xi vI^* + \gamma_1 l + cg)$, $b_1 = cg(e - (\gamma_1 + \mu)) + f\beta_2I^*(a - (\gamma_1 + \mu)) + (a + e)(\gamma_1 l + h(\gamma_1 + \mu)) + (h - (\gamma_1 + \mu))(\xi vI^* - ae) - (\xi f g + cv\beta_2I^{*2})$, $b_0 = -\beta_2I^*(\gamma_1 + \mu)(cvI^* - af) - g(\gamma_1 + \mu)(\xi f - ce) + \gamma_1 l(\xi vI^* - ae)(\gamma_1 l + h(\gamma_1 + \mu))$.

Evaluating the coefficients of polynomial equation (24) using parameter values in Table 1, we have

$$b_3 = 2531.7, \quad b_2 = 10981, \quad b_1 = 3545.26, \quad b_0 = 2215.745.$$

Thus, polynomial equation (24) becomes

$$\lambda^4 + 2531.7\lambda^3 + 10981\lambda^2 + 3545.26\lambda + 2215.745 = 0. \tag{25}$$

Using polynomial function in MATLAB, the eigenvalues obtained are

$$\lambda_1 = -0.7792,$$

$$\lambda_2 = -248.64,$$

$$\lambda_3 = -1.789 - 2.868i,$$

$$\lambda_4 = -1.789 + 2.86i. \tag{26}$$

Since $\text{Re}(\lambda_i) < 0$, the endemic steady state is locally asymptotically stable. This ends the proof. \square

4. Global Stability of Steady States

In this section, we deal with the global stability of steady states of model (1) using Lyapunov functionals with LaSalle's invariant principle. Since it is often hard to construct appropriate Lyapunov functions especially in epidemiological models with nonlinear and bilinear incidence rates, existing techniques for constructing Lyapunov functions have been improved, see [37]. We propose the combination of quadratic and linear Lyapunov forms in the construction of Lyapunov function to prove a theorem for global stability of disease-free steady state of the form:

$$U(x_1, x_2, x_3, x_4) = c \left(\sum_{i=1}^2 \frac{1}{2x_i^*} (x_i - x_i^*)^2 \right) + \sum_{i=3}^4 (x_i - x_i^*), \tag{27}$$

where c is a positive constant, $\sum_{i=1}^2 (1/2x_i^*)(x_i - x_i^*)^2$ represents a class containing susceptible population, and $\sum_{i=3}^4 (x_i - x_i^*)$ represents the remaining classes. The Goh-Lotka-Volterra Logarithmic of the form

$$V(x_1, x_2, x_3, x_4) = c \sum_{i=1}^n \left(x_i - x_i^* - x_i^* \ln \frac{x_i}{x_i^*} \right) \tag{28}$$

is used to prove the theorem on global stability of the endemic steady state.

4.1. Global Stability of the Disease-Free Steady State

Theorem 1. *By Proposition 2, if the disease-free steady state E_0^* of model (1) is asymptotically stable, then E_0^* is globally asymptotically stable in D .*

Proof. Consider $U: D \rightarrow \mathbb{R}$ that is defined by

$$U(S_u, S_a, I, R) = c \left(\frac{1}{2S_u^0} (S_u - S_u^0)^2 + \frac{1}{2S_a^0} (S_a - S_a^0)^2 + (I - I_0) \right) + c(R - R^0), \tag{29}$$

with $c = 1$. We find the derivative of the positive semidefinite function with respect to time along with the solution of model system (1) to get

$$\begin{aligned} \frac{dU}{dt} &= \frac{S_u - S_u^0}{S_u^0} \dot{S}_u + \frac{S_a - S_a^0}{S_a^0} \dot{S}_a + \dot{I} + \dot{R}, \\ \frac{dU}{dt} &= vS_u I \left(\frac{S_a}{S_a^0} - \frac{S_u}{S_u^0} \right) + \xi S_a \left(\frac{S_u}{S_u^0} - \frac{S_a}{S_a^0} \right) + \frac{\phi I}{1 + \tau I} (1 - p) \\ &\quad \cdot \left(2 + \frac{S_a}{S_a^0} \right) + \beta_2 S_a I \left(2 - \frac{S_a}{S_a^0} \right) + \left(\beta - \frac{\beta_1 m}{m + I} \right) \\ &\quad \cdot \left(2 - \frac{S_u}{S_u^0} \right) S_u I - \mu \left(\frac{S_u^2}{S_u^0} \left(1 - \frac{S_u^0}{S_u} \right) + \frac{S_a^2}{S_a^0} \left(1 - \frac{S_a^0}{S_a} \right) + I + R \right) \\ &\quad - (\delta_1 I + \gamma_1 R), \\ \frac{dU}{dt} &\leq \beta_2 S_a I \left(2 - \frac{S_a}{S_a^0} \right) + \frac{S_a^2}{S_a^0} \left(1 - \frac{S_a^0}{S_a} \right) + \left(\beta - \frac{\beta_1 m}{m + I} \right) \\ &\quad \cdot \left(2 - \frac{S_u}{S_u^0} \right) S_u I - \mu (I + R) - (\delta_1 I + \gamma_1 R). \end{aligned} \tag{30}$$

Lemma 2. *Let a_1, a_2, \dots, a_m be m positive numbers. The arithmetic mean $\bar{a} = (a_1 + a_2 + \dots + a_m)/m$ is greater or equal to the geometric mean $\bar{a}^* = (a_1 \times a_2 \times \dots \times a_m)^{1/m}$, that is, $\bar{a} \geq \bar{a}^*$. Applying Lemma 2 to equation (30), we have*

$$\frac{dU}{dt} \leq \beta_2 S_a I \left(2 - \frac{S_a}{S_a^0} \right) + \frac{S_a^2}{S_a^0} \left(1 - \frac{S_a^0}{S_a} \right) + \left(\beta - \frac{\beta_1 m}{m + I} \right) \left(2 - \frac{S_u}{S_u^0} \right) S_u I. \tag{31}$$

Thus, due to local stability of E_0 or E_1 , then $dU/dt \leq 0$ for all $(S_u, S_a, I, R) \in D$. However, the strict equality $dU/dt = 0$ is valid for $S_u = S_u^0, S_a = S_a^0, I = 0$ and $R = 0$. Then, the largest invariant set $\{(S_u, S_a, I, R) \in D: dU/dt = 0\}$ is reduced to the disease-free steady state E_0^* . Therefore, by LaSalle's Invariance Principle [38], E_0^* is an attractive point that is globally asymptotically stable in D . This ends the proof.

4.2. Global Stability of the Endemic Steady State

Theorem 2. *If by Proposition 3, the unique endemic equilibrium of model (1) is asymptotically stable, then E^* is globally asymptotically stable in the interior of D .*

Proof. The Lyapunov function is defined as

$$W(S_u, S_a, I, R) = c_1 \left(S_u - S_u^* - S_u^* \ln \frac{S_u}{S_u^*} + S_a - S_a^* - S_a^* \ln \frac{S_a}{S_a^*} \right) + c_2 \left(I - I^* - I^* \ln \frac{I}{I^*} + R - R^* - R^* \ln \frac{R}{R^*} \right), \tag{32}$$

with $c_1 = 1, c_2 = R_0 = (\gamma_1 + \mu) / (\Phi + \mu + \delta_1)(\gamma_1 + \mu) - p\Phi\gamma_1$.

Function W in (32) is defined, continuous, and positive definite for all $S_u, S_a, I, R > 0$. Thus, the function $W(S_u, S_a, I, R)$ takes the value $W(S_u, S_a, I, R) = 0$ at the steady state E^* , and the minimum value of $W(S_u, S_a, I, R)$ occurs at the endemic steady state E^* . We compute the derivative of W along the solution trajectories of model (1) as

$$\begin{aligned} \dot{W} &= \dot{S}_u \left(1 - \frac{S_u^*}{S_u} \right) + \dot{S}_a \left(1 - \frac{S_a^*}{S_a} \right) + c_2 \left(\dot{I} \left(1 - \frac{I^*}{I} \right) + \dot{R} \left(1 - \frac{R^*}{R} \right) \right) \\ &= B \left(1 - \frac{S_u^*}{S_u} \right) + \xi S_a^* \left(1 - \frac{S_u^*}{S_u} \right) + g(I^*) S_u^* \left(\frac{S_u^*}{S_u} - 1 \right) \\ &\quad + vI^* S_u^* \left(\frac{S_u^*}{S_u} - 1 \right) + \mu S_u^* \left(\frac{S_u^*}{S_u} - 1 \right) + vI^* S_u^* \left(1 - \frac{S_a^*}{S_a} \right) \\ &\quad + (1 - p)\phi T(I^*) \left(1 - \frac{S_a^*}{S_a} \right) + \beta_2 I^* S_a^* \left(\frac{S_a^*}{S_a} - 1 \right) \\ &\quad + \xi S_a \left(\frac{S_a^*}{S_a} - 1 \right) + \mu S_a^* \left(\frac{S_a^*}{S_a} - 1 \right) + c_2 \left(g(I^*) S_u^* \left(1 - \frac{I^*}{I} \right) \right. \\ &\quad \left. + \beta_2 S_a^* I^* \left(1 - \frac{I^*}{I} \right) + \gamma_1 R^* \left(1 - \frac{I^*}{I} \right) + \phi T(I^*) \left(\frac{I^*}{I} - 1 \right) \right. \\ &\quad \left. + (\mu + \delta_1) I^* \left(\frac{I^*}{I} - 1 \right) + p\phi T(I^*) \left(1 - \frac{R^*}{R} \right) \right. \\ &\quad \left. + (\gamma_1 + \mu) R^* \left(\frac{R^*}{R} - 1 \right) \right). \end{aligned} \tag{33}$$

Since (S_u^*, S_a^*, I^*, R^*) is an endemic steady state of model (1), we have

$$B = g(I^*) S_u^* + vI S_u^* + \mu S_u^* - \xi S_a^*, \tag{34}$$

and making a substitution of B in equation (33) and collecting like terms, we get

$$\begin{aligned}
\dot{W} &= I^* S_u^* \left(v \left(1 - \frac{S_a^*}{S_a} \right) + c_2 \left(\beta - \frac{\beta_1 m}{m + I^*} \right) \right) - \mu \left(S_a \left(1 - \frac{S_a^*}{S_a} \right) \right. \\
&\quad \left. + c_2 I^* \left(\left(1 - \frac{I^*}{I} \right) + R^* \left(1 - \frac{R^*}{R} \right) \right) \right) + \Phi T(I^*) p \frac{S_a^*}{S_a} \\
&\quad \cdot \left(1 - c_2 \frac{R^*}{R} \frac{S_a^*}{S_a} \right) + \Phi T(I^*) \left(1 - \frac{S_a^*}{S_a} \right) + p c_2 \Phi T(I^*) \\
&\quad - \Phi T(I^*) c_2 \left(1 - \frac{I^*}{I} \right) - \delta_1 c_2 I^* \left(1 - \frac{I^*}{I} \right) + \frac{\beta_2 S_a^{*2}}{S_a} \\
&\quad \cdot \left(1 - \frac{S_a^*}{S_a} \frac{I^*}{I} \right), \\
\dot{W} &\leq v \left(1 - \frac{S_a^*}{S_a} \right) I^* S_u^* + R_0 I^* \left(1 - \frac{I^*}{I} \right) + R^* \left(1 - \frac{R^*}{R} \right) \\
&\quad + \frac{R_0 \gamma_1 R^{*2}}{R} \left(1 - \frac{R^*}{R} \frac{I^*}{I} \right) + \frac{\beta_2 S_a^{*2}}{S_a} \left(1 - \frac{S_a^*}{S_a} \frac{I^*}{I} \right) \\
&\quad + \Phi T(I^*) \left(1 - \frac{S_a^*}{S_a} \right). \tag{35}
\end{aligned}$$

Hence, since the arithmetic mean exceeds the geometric mean, we have

$$\begin{aligned}
\left(1 - \frac{S_a^*}{S_a} \right) &\leq 0, \\
\left(1 - \frac{R^*}{R} \right) &\leq 0, \\
\left(1 - \frac{I^*}{I} \right) &\leq 0. \tag{36}
\end{aligned}$$

We note that all model parameters are positive; therefore, $\dot{W} \leq 0$ for $R_0 > 1$, and the equality holds if and only if $S_a^* = S_a, I^* = I, R^* = R$. Hence, W is a Lyapunov function on the interior of D , with the largest compact invariant subset of the set where $\dot{W} = 0$ is a singleton $\{(S_u, S_a, I, R) = (S_u^*, S_a^*, I^*, R^*)\}$. By LaSalle's invariance principle [38], it follows that the endemic equilibrium E^* of model (1) is globally asymptotically stable in the feasible region D if it exists. \square

4.3. Sensitivity Analysis. In this section, model parameters are varied with respect to the basic reproduction number, R_0 , of model (1). Carrying out a sensitivity analysis of the model parameters will help us identify and verify model epidemiological parameters that most affect the basic reproduction number. Furthermore, values obtained for sensitivity indices indicate epidemiological parameters to be targeted for intervention purposes. We derive the sensitivity index using partial rank correlation coefficients (PRCC) of the

TABLE 3: Sensitivity index (S. I.) of R_0 w.r.t the parameters.

Code	Parameter	S. I.
1	β	+1.07189
2	β_1	-0.11824
3	β_2	+0.04635
4	p	+0.00079
5	ξ	+0.95282
6	v	-0.95345
7	B	-0.13570
8	μ	-0.04621
9	δ_1	-0.10006
10	Φ	-0.89974
11	γ_1	+0.79958

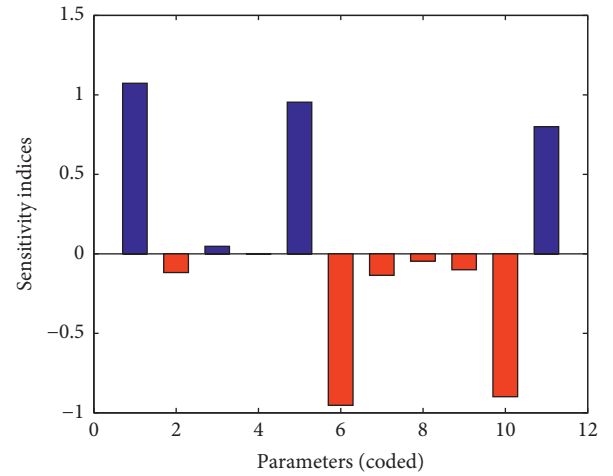


FIGURE 2: Sensitivity indices/partial rank correlation coefficients (PRCC) of R_0 , in relation to parameters (coded).

basic reproductive number with respect to the parameters [39]. The normalized forward sensitivity index technique is used to obtain the index of R_0 with respect to the parameters (Table 1). Hence, $\Delta_q^{R_0} = (\partial R_0 / \partial q) \times (q / R_0)$, where R_0 is a variable and q is a differentiable parameter.

Since the availability of the literature and data especially on antibiotic resistance awareness of pneumococcal pneumonia is lacking, the qualitative predictions of our model (1) is dependent on estimating some of the epidemiological parameter values in Table 1.

From Table 3, the positive sign of sensitivity index of the basic reproduction number with respect to the model parameters indicates that an increase (or decrease) in the value of each parameter in such a category will lead to an increase (or decrease) in the basic reproduction number of the disease. On the other hand, the negative sign of sensitivity index of the control reproduction number with respect to the model epidemiological parameters implies that, an increase (or decrease) in the value of the epidemiological parameter shall give rise to a corresponding decrease (or increase) in the control reproduction number. For instance, in Figure 2, the sensitivity ($\Delta_\xi^{R_0} \approx 1$) means that, when the loss of information about disease by aware susceptible individuals [ξ (5)] is increased (or decreased) by 10%, it leads to increases or decreases of R_0 by 10%.

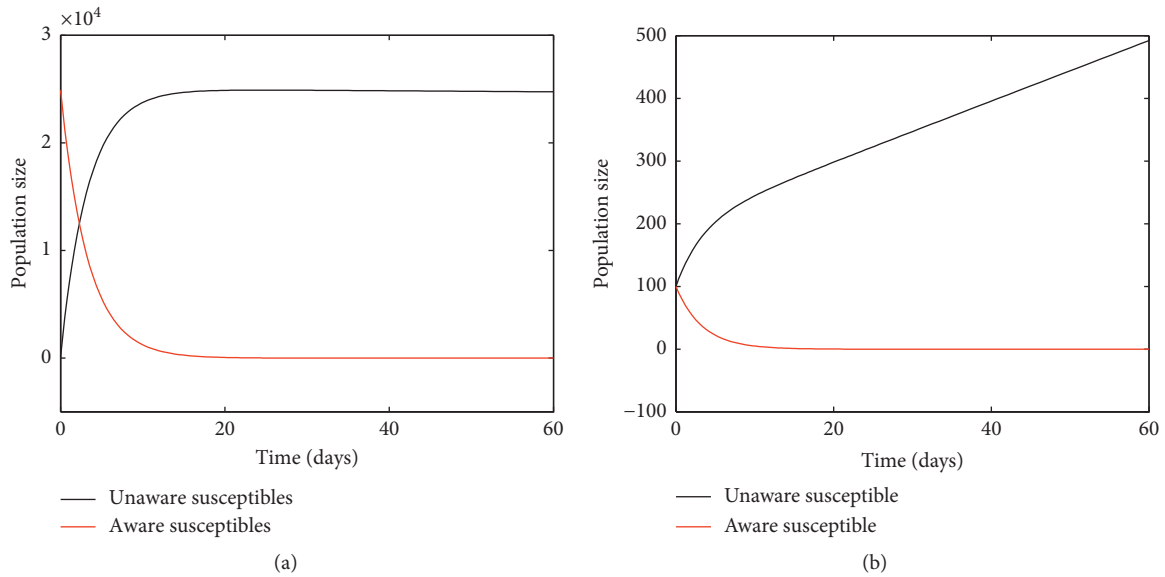


FIGURE 3: Stability of the disease-free steady state. (a) With parameter values $\beta = 0.0001865$ and $\beta_1 = 0.00046$. (b) With parameter values $\beta = 0.0417$, $\gamma_1 = 0.00145$, $\beta_1 = 0.046$, and $\beta_2 = 0.00007498$. Choosing initial values $S_u(0) = 103$, $S_a(0) = 24896$, $I(0) = 0$, $R(0) = 0$ and the remaining parameter values is as in Table 1.

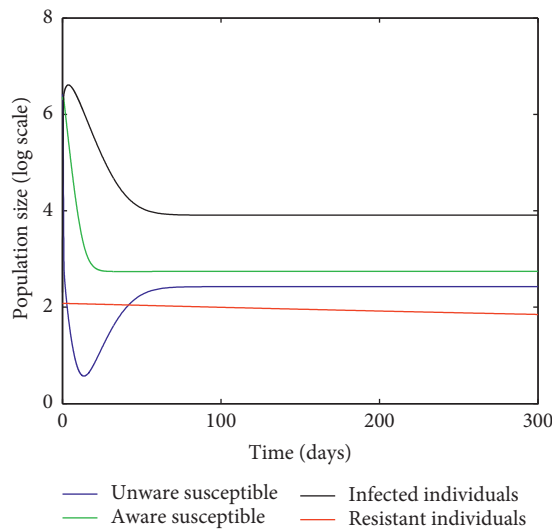


FIGURE 4: Global stability of the endemic steady state with initial variables $S_u^* = 606$, $S_a^* = 581$, $I^* = 10$, $R^* = 8$ and parameter value $\beta = 0.0147$; remaining parameter values are given in Table 1.

From Figure 2, it can be seen that the basic reproduction number is positively sensitive to the maximal effective contact rate before antibiotic resistance awareness ($\beta[1]$), loss of information about disease by aware susceptible individuals ($\xi[5]$), and rate of relapse encountered in administering treatment ($\gamma_1[11]$) and negatively sensitive to v , recovery rate due to treatment ($\Phi[10]$). Thus, with sensitivity analysis, one is able to get appropriate information on epidemiological parameters that can be targeted for intervention strategies that would help in preventing and controlling the transmission of pneumococcal pneumonia.

5. Numerical Simulation and Discussion

This section deals with the numerical simulation results of model (1) that are carried out in MATLAB’s standard solver for ODEs, the inbuilt function ode45. The epidemiological parameters chosen for the purpose of simulation are given in Table 1. The importance of R_0 is well demonstrated in all simulations.

From Figure 3(a), the unaware and aware populations approach disease-free steady state with unaware individuals maintaining a high number compared with the aware individuals. The basic reproduction number is less than

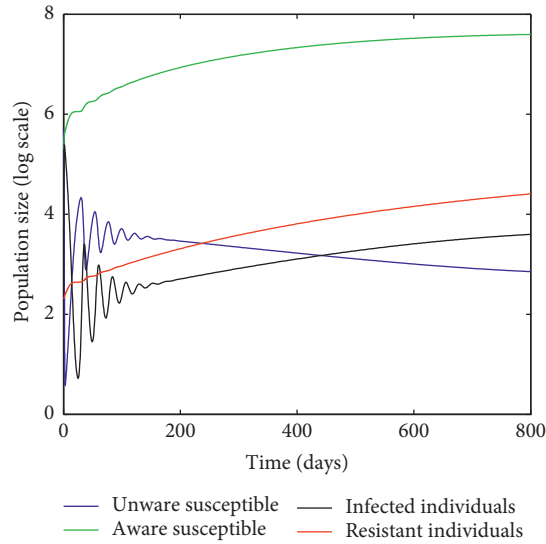


FIGURE 5: Time series global stability of the endemic steady state with initial state variables $S_u^* = 300$, $S_a^* = 200$, $I^* = 10$, $R^* = 10$. Parameter values $\xi = 0.003$, $m = 0.2$, $p = 0.02$, $\tau = 0.027$, and $\Phi = 0.6$; the remaining parameter values are specified in Table 1.

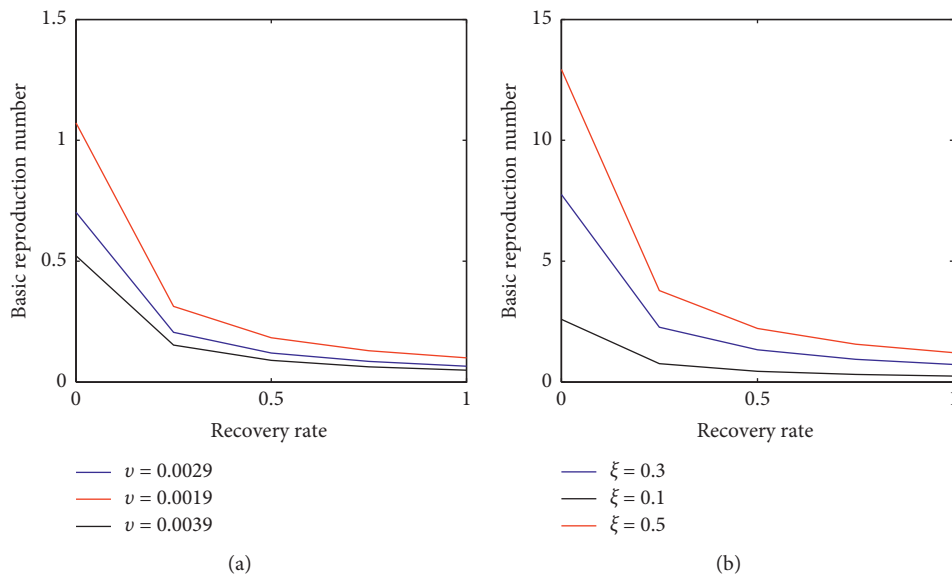


FIGURE 6: (a) Effect of varying the rate at which unaware susceptibles become aware of antibiotic resistance in treating the disease on the control reproduction number, with parameter value $\xi = 0.02$, and other parameter values remain as in Table 1. (b) The effect of varying loss of information about disease by aware susceptibles on the reproduction number and recovery rate, with the rest of parameter values unchanged as in Table 1.

unity $R_0 = 0.3323 < 1$; thus, the disease-free steady state is stable. The stability of the disease-free steady state means that pneumococcus bacteria can be completely eliminated from the population with no more cases of pneumococcal pneumonia reported. Figure 3(b), shows that, if $R_0 = 1.9258 > 1$, the disease-free steady state is unstable, implying that if one infected individual is introduced in a wholly susceptible population, infected cases would rise and more unaware individuals are at a risk of contracting pneumococcus bacteria. This would require control strategies to reduce the transmission of the disease at the earliest time possible. Considering the main results,

Proposition 4.2, and Theorem 2, the endemic steady state is locally asymptotically stable since $\text{Re}(\lambda_i) < 0$ and globally stable if $R_0 > 1$.

Figure 4, shows global stability of all solution trajectories to an endemic steady state E^* . This implies that pneumococcal pneumonia will continue to propagate in the population with more infected individuals compared with other states. Thus, the disease would persist in the population.

Figure 5, shows a unique endemic steady state attained in the long run with more aware individuals in the population. This implies that the pneumococcus bacteria could be controlled with appropriate techniques of awareness

programs, hence reducing the risk of acquiring the pneumococcal pneumonia infections in a population.

Figure 6(a), shows an exponential decay, that is, the family of curves switches due to the variation of private awareness from an increasing rate of growth to a decreasing rate, evidently suggesting the eradication of pneumococcus bacteria because of the presence of antibiotic resistance awareness and treatment.

Figure 6(b), shows effect of loss of information about antibiotic resistance awareness. An increase in the loss of information implies an increase in numbers of infected individuals and the basic reproduction number is high; however, due to the presence of treatment, the disease is always eradicated from the population. On the other hand, reducing the loss of information implies that more individuals are aware of the transmission and are able to take control measures of the disease and reduce its spread.

6. Conclusion

In this paper, the pneumococcal pneumonia model in the presence of awareness and saturated treatment is constructed. The threshold number (basic reproduction number, R_0) for pneumococcal pneumonia prevalence, the conditions for existence, and uniqueness of the equilibria are found.

The result of Proposition 2 indicates that if $R_0 \leq 1$, the disease-free steady state E_0^* is locally asymptotically stable. Biologically, this means that pneumococcus bacteria cannot successfully invade the susceptible population and, thus, can easily be wiped out as time increases. This suggests that pneumococcal pneumonia can be controlled by ensuring R_0 is below unity. If $R_0 > 1$, then the disease-free steady state E_0^* is unstable, implying that the disease could manifest in the population and more cases might arise causing an epidemic.

The quadratic-linear and Goh–volterra Lyapunov functional approaches are used to prove the global stabilities of the disease-free and endemic steady states, respectively. We found out that the endemic steady state is globally steady if $R_0 > 1$. Thus, pneumococcal disease would persist in a population regarding consciousness of people.

To control pneumococcal pneumonia, the results show that the family of decaying curves could help in providing mechanisms to design awareness strategies for containing pneumococcal pneumonia. The threshold parameter R_0 could be reduced to less than unity if antibiotic resistance awareness and treatment are implemented simultaneously to ensure eradication of pneumococcus bacteria; thus, spread of pneumococcus pneumonia in the population will die out.

Therefore, the model described, formulated, and analyzed and the findings presented in this work may help in controlling pneumococcal pneumonia through awareness. Awareness changes the trend of disease transmission and decreases the rate of infection within a human population.

6.1. Future Directions. To further understand the dynamics and control of the disease, it is paramount for researchers to extend the model by performing an optimization problem

that helps in minimizing the total number of infected persons, maximizes the number of susceptible individuals, and reduces costs related to treatment of the disease.

Data Availability

The data supporting this mathematical model are from earlier published articles, and they have been suitably cited as references in this paper. Parameter values taken from published articles are cited in Table 1 of this paper.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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