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Full Length Research Article

MATHEMATICAL MODELING AND ANALYSIS OF PNEUMONIA INFECTION DYNAMICS

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ABSTRACT
Pneumonia is one of the leading causes of death worldwide, especially among children below 5 years, the elderly above 65 years and people with weaker immune system. It is usually referred to as the "captain of the men of death" because of the great toll it exacted on humanity. In this work, we examined the dynamics of the pneumonia disease from a mathematical perspective via a deterministic SEIR model. This consists of investigating the equilibrium, basic reproduction number, stability analysis, and bifurcation analysis. It is observed that the pneumonia free equilibrium is locally asymptotically stable if the basic reproduction number is less than one, and the pneumonia endemic equilibrium is globally asymptotically stable in the invariant region if the basic reproduction number is greater than one. The sensitivity analysis revealed that the rate of transmission and the rate at which exposed individuals become infectious are the most sensitive parameters, and the bifurcation analysis via the centre manifold theory revealed the presence of forward bifurcation.

Keywords: Pneumonia, SEIR, Model, Stability, Equilibrium, Bifurcation

INTRODUCTION

Pneumonia is a condition of the lungs that affects the alveoli; and dry cough, chest pain, fever, and trouble breathing are common symptoms of pneumonia. Viruses or bacteria usually cause Pneumonia (Angela, 2009). Eddy (2005) explained that the lungs of individuals with pneumonia are filled with fluid and this makes breathing difficult, and pneumonia disproportionately affects the young, the elderly, as well as vulnerable individuals whose immune system have been compromised. It preys on weakness and vulnerability. Tilahun (2017) revealed that pneumonia was described 2,500 years ago by Hippocrates, the father of medicine, and that Dr. William Osler, the founder of modern medicine, who studied pneumonia throughout his career, called pneumonia the "captain of the men of death" because of the great toll it exacted on humanity. Pneumonia is associated with the following risk factors: pulmonary disease, cystic fibrosis, asthma, diabetes, heart failure, poor ability to cough such as following a stroke, and a weak immune system. The disease may be classified by where it was acquired with community, hospital, or health care associated (Angela, 2009). For children under five years, the typical signs and symptoms of pneumonia include fever, cough, fast or difficult breathing, ongoing vomiting, unwillingness to drink, convulsions, extremes of temperature, and a decreased level of consciousness (Varinder & Satinder, 2011). George (2005) revealed that the introduction of vaccines and antibiotics in the 20th century improved the chance of survival of pneumonia patients, but among the very young, the very old, the chronically ill, and in developing countries, pneumonia remains a leading cause of death. Eddy (2005) explained that pneumonia often shortens suffering among those already close to death and has thus been called "the old man's friend"; while Angela (2009) opined that pneumonia, can be classified as one of the air-borne diseases, and It accounts for the death of millions of people through inhalation of pathogenic organism, mainly *streptococcus pneumonia.* Human beings of all ages can be affected by the pneumonia disease, from children to the elderly. This is even worsened when the immune system is lowered (WHO, 2008).

In order to understand the dynamics of infectious diseases, several scholars proposed different mathematical models to describe the dynamics of infectious diseases in the community and these models are used for making quantitative predictions of different intervention strategies and their effectiveness. Tilahun *et al* proposed a nonlinear deterministic mathematical model for the typhoid fever outbreak and the optimal control problem was also studied for a community with varying population. It was revealed that the model exhibits a forward transcritical bifurcation, and that treatment is the best cost effective strategy to eradicate the disease. Joseph (2012) studied the impact of treatment and vaccination in curtailing the spread of pneumonia disease, and it was revealed that the rate of transmission, vaccine protection, and the waning rate of vaccine are the main factors in fueling the spread of the disease, while the vaccination and treatment control parameters are inhibitors of the disease spread. He therefore posited that if the vaccination and treatment control programs targeted at both adults and children can reduce the effective reproduction number, R_{eff}, below unity, then a combination of both programs can effectively eliminate the pneumonia infection from the population. Several scholars also proposed a model on pneumonia dynamics. Kizito and Julius (2018) studied a model on the spread and control of bacterial pneumonia under treatment and vaccination and it was revealed that the disease-free equilibrium is stable if and only if the basic reproduction number, R_0, is less than unity, and the disease will be wiped out of the population, while for $R_0 > 1$, the endemic equilibrium is globally stable and the disease persists. Jacob *et al* (2013) developed a mathematical model for pneumonia among children under five years of age, and the analysis revealed that reducing the transfer rates between the carrier and the infected class reduces prevalence of the disease. The analysis also revealed a possibility of forward bifurcation. In order to investigate the dynamics of the co-infection of pneumonia and meningitis, Tilahun (2019) developed a deterministic mathematical model using ordinary differential equations which divides the population into seven compartments. He emphasized that in order to make the endemic equilibrium unstable so that it switches to disease-free equilibrium, intervention strategies like high efficacy treatment and vaccination programs are necessary. The results obtained revealed that decreasing the contact rate of

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either pneumonia or meningitis has a great influence on controlling co-infection of pneumonia and meningitis in the population. It was revealed that decreasing the contact rate of either pneumonia or meningitis has a great influence on controlling co-infection of pneumonia and meningitis in the population. All the above studies and so many others, have developed mathematical models on the pneumonia disease by considering different aspects. In this research work, we considered and analyzed a deterministic SEIR model for the pneumonia disease.

The presentation of the rest part of this paper is in the following order: We first introduce the basic model via a compartmentalized deterministic system of non-linear ordinary differential equations. Some basic properties such as non-negativity of solution, invariant region and boundedness of solution, disease-free equilibrium as well as basic reproduction number of the model are then discussed. Some qualitative features of the model are also investigated. These include the local and global asymptotic stabilities of the model with respect to its basic reproduction number are also studied. Furthermore, the centre manifold theory is employed to study the bifurcation analysis of the model. Finally, the sensitivity analysis of the model parameters is then discussed.

Basic Model Formulation

In this section, we construct a deterministic SEIR mathematical model describing the transmission dynamics of pneumonia. According to the disease status of individuals, the total human population $\mathrm{N(t)}$ is subdivided into four mutually-exclusive time-dependent compartments comprising of the susceptible compartment $S(t)$, exposed compartment $\mathrm{E(t)}$, infectious compartment $\mathrm{I(t)}$ and recovered compartment $\mathrm{R(t)}$, in the sense that $N(t) = S(t) + E(t) + I(t) + R(t)$

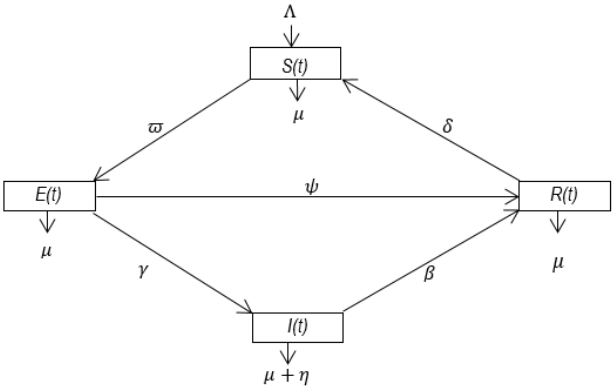

Figure 1: Flow diagram of the disease

The susceptible class consists of all individuals who can contract pneumonia, the exposed compartment consists of those individuals who have contracted the disease but are not infectious. The infectious compartment includes all individuals who have contracted the disease and can also infect others, whereas the recovered class includes all individuals who have recovered from pneumonia. We assume that individuals within the population have equal likelihood of catching pneumonia. Not all individuals who come in contact with infectious individuals immediately become infectious. Furthermore, recovered and exposed individuals do not play a part in transmitting pneumonia to the susceptible individuals. Disease induced death only occur in the infectious compartment. In view of the above assumptions as well as the schematic diagram in Fig. 1, the transmission dynamics of the pneumonia infection is governed by the following nonlinear system of ordinary differential equations:

$$\begin{cases} \dfrac{\mathrm{dS(t)}}{\mathrm{dt}} = \Lambda + \delta\mathrm{R} - (\mu + \varpi)\mathrm{S}, \\ \dfrac{\mathrm{dE(t)}}{\mathrm{dt}} = \varpi\mathrm{S} - (\mu + \gamma + \psi)\mathrm{E}, \\ \dfrac{\mathrm{dI(t)}}{\mathrm{dt}} = \gamma\mathrm{E} - (\mu + \beta + \eta)\mathrm{I}, \\ \dfrac{\mathrm{dR(t)}}{\mathrm{dt}} = \psi\mathrm{E} + \beta\mathrm{I} - (\mu + \delta)\mathrm{R}. \end{cases} \quad (1)$$

Associated with the system of equations (1) are the following initial conditions:

$$\mathrm{S(0)} = \mathrm{S_0}, \quad \mathrm{E(0)} = \mathrm{E_0}, \quad \mathrm{I(0)} = \mathrm{I_0}, \quad \mathrm{R(0)} = \mathrm{R_0}.$$

The parameter Λ denotes the recruitment rate of susceptible individuals. Susceptible individuals enter the exposed compartment with a force of infection $\varpi = \frac{\chi \mathrm{I}}{\mathrm{N}}$, where $\chi = \mathrm{k}\tau$, with k being the contact rate and τ the probability that a contact is effective enough to cause infection. As the disease progresses in the absence of treatment, exposed individuals join the infectious compartment at the rate γ, while recovery rate of the exposed individuals is ψ. Infectious individuals recover from pneumonia at rate β, while recovered individuals who have lost their immunity become susceptible at rate δ. The diseased induced death rate for the infectious compartment is η, while natural mortality for all individuals is at rate μ.

Table I: Description of the model parameters

Parameters	Description
Λ	Recruitment rate of susceptible humans.
ϖ	Force of infection of the susceptible class.
χ	Rate of transmission.
k	Per capita contact rate of susceptible individuals with the infectious individuals.
τ	The probability that a contact is effective to cause infection.
β	Per capita recovery rate of the infectious.
ψ	Per capita recovery rate of the exposed.
η	Per capita disease induced mortality rate.
γ	Rate at which exposed becomes infectious.
μ	Per capita natural mortality rate of individuals.
δ	Rate at which treated individuals become susceptible.

MODEL ANALYSIS

Positivity of solutions: For model (1) to be epidemiologically meaningful and mathematically well posed, it is necessary to establish that all solutions of system with positive initial data will remain positive for all times $\mathrm{t} > 0$. This will be established in the following theorem.

Theorem 1: Let $\{(S, E, I, R) \in \mathbb{R}^4_+ : S(0) > 0,\ E(0) > 0,\ I(0) > 0,\ R(0) > 0\}$. *Then the solution set* $\big(S(t), E(t), I(t), R(t)\big)$ *of system (1) is positive for all* $t \geq 0$.

Proof: From the first equation of system (1), we have

$$\frac{dS(t)}{dt} \geq -(\mu + \varpi)S.$$

This implies $S(t) \geq S_0 e^{-(\mu+\varpi)t} \geq 0 \quad \text{for all} \quad t \geq 0.$

In the same manner, we also have $E(t) \geq 0,\ I(t) \geq 0,\ R(t) \geq 0 \quad \text{for all} \quad t \geq 0.$

Invariant Region: Apart from the positivity of the state variables, it is also important to determine the region in which the solution of model (1) is bounded. In this direction, we have that the dynamics of the total human population satisfies

$$\frac{dN(t)}{dt} \leq \Lambda - \mu N(t). \qquad (2)$$

This implies $N(t) \leq [1 - e^{-\mu t}]\frac{\Lambda}{\mu} + N(0)e^{-\mu t}$. As $t \longrightarrow \infty$, we get $N(t) \longrightarrow \frac{\Lambda}{\mu}$. Therefore, the feasible solution set of the system (1) given by

$$\Omega = \left\{(S, E, I, R) \in \mathbb{R}^4_+ : 0 \leq S + E + I + R = N \leq \frac{\Lambda}{\mu}\right\}, \quad (3)$$

is positively invariant, inside which the model is considered to be epidemiologically meaningful and mathematically well-posed.

Pneumonia Free Equilibrium Point: This is the point where there is no pneumonia within the population under consideration. Here $E = I = R = 0$ and the pneumonia free equilibrium $\mathbb{E}_0$ is obtained by setting the right hand side of each equation in system (1) to zero. Thus,

$$\mathbb{E}_0 = (S^0, E^0, I^0, R^0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right). \qquad (4)$$

Basic Reproduction Number: We compute the basic reproduction number R_0 for the model (1). This will prove in analyzing the stability of the equilibrium points. R_0 is a very important dimensionless epidemiological parameter which measures the average number of secondary cases generated by a primary case when the infected individual is introduced into a population of completely susceptible individuals. We will determine R_0 for system (1) by applying the next generation matrix approach as laid out by Van den and James (2002). The first step to calculating R_0 is to rewrite the infective classes of the model equations in the form:

$$X'(t) = \mathcal{F}(t, X(t)) - \mathcal{V}(t, X(t)),$$

where

$$\begin{cases} X(t) = \big(E(t), I(t)\big), \\ \mathcal{F}(t, X(t)) = \begin{bmatrix} \frac{\chi I}{N} S \\ 0 \end{bmatrix}, \\ \mathcal{V}(t, X(t)) = \begin{bmatrix} (\mu + \gamma + \psi)E \\ -\gamma E + (\mu + \beta + \eta)I \end{bmatrix}. \end{cases}$$

Here the matrices $\mathcal{F}$ and $\mathcal{V}$ denote the new infection terms and the remaining transfer terms, respectively. With the notations $k_1 = \mu + \gamma + \psi$ and $k_2 = \mu + \beta + \eta$, we determine the following next generation matrices

$$F = \begin{bmatrix} 0 & \chi \\ 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} k_1 & 0 \\ -\gamma & k_2 \end{bmatrix},$$

as the Jacobian matrices of $\mathcal{F}(t, X(t))$ and $\mathcal{V}(t, X(t))$, respectively, evaluated at the pneumonia free equilibrium. Next, we have

$$FV^{-1} = \begin{bmatrix} \frac{\chi\gamma}{k_1 k_2} & \frac{\chi}{k_2} \\ 0 & 0 \end{bmatrix}.$$

Thus, the basic reproduction number R_0 is obtained as the spectral radius FV^{-1}, that is,

$$R_0 = \sigma(FV^{-1}) = \frac{\chi\gamma}{k_1 k_2}. \qquad (5)$$

Local stability of the pneumonia free equilibrium: To examine the local stability of the pneumonia free equilibrium, we first obtain the following Jacobian matrix

$$J_{\mathbb{E}_0} = \begin{bmatrix} -\mu & 0 & -\chi & \delta \\ 0 & -k_1 & \chi & 0 \\ 0 & \gamma & -k_2 & 0 \\ 0 & \psi & \beta & -(\mu + \delta) \end{bmatrix},$$

of the system (1) at $\mathbb{E}_0$. Next, we then establish the following stability result.

Theorem 2: The pneumonia free equilibrium $\mathbb{E}_0$ is locally asymptotically stable if $R_0 < 1$, otherwise it is unstable.

Proof: The characteristic polynomial of the matrix $J_{\mathbb{E}_0}$ is obtained as

$$\mathrm{Det}\big(\lambda I - J_{\mathbb{E}_0}\big) = (\lambda + \mu)(\lambda + \mu + \delta)(\lambda^2 + a\lambda + b) = 0,$$

where

$$a = k_1 + k_2 \quad \text{and} \quad b = k_1 k_2 - \chi\gamma = (1 - R_0)k_1 k_2.$$

The pneumonia free equilibrium $\mathbb{E}_0$ is locally asymptotically stable if and only if all roots of the above characteristic polynomial have negative real parts. Obviously, the first two eigenvalues $\lambda_1 = -\mu$ and $\lambda_2 = -(\mu + \delta)$ are negative while the remaining two eigenvalues can be obtained as roots of the characteristics equation $\lambda^2 + a\lambda + b = 0$. According to the Routh-Hurwitz criteria, the equation $\lambda^2 + a\lambda + b = 0$ have strictly negative real roots if $a > 0$ and $b > 0$ (Arthur & Theresa (2000), Van den & James (2002)). Clearly, we observe that $a > 0$ because it is the sum of positive parameters. Also and $b = (1 - R_0)k_1 k_2 > 0$ whenever $R_0 < 1$. Hence, the pneumonia free equilibrium is locally asymptotically stable if $R_0 < 1$.

Remark: Theorem 2 implies that pneumonia can be eradicated from the population (when $R_0 < 1$) if the initial sizes of the sub-

populations of the model (1) are in the basin of attraction of the pneumonia free equilibrium. To ensure that eradication of the pneumonia bacteria does not depend on the initial sizes of the sub-populations, it is important to establish that the pneumonia free equilibrium globally asymptotically stable

Global stability of the pneumonia free equilibrium: A very common approach to investigate the global asymptotic stability of the disease free equilibrium of epidemiological models is by constructing an appropriate Lyapunov function (Ana & James (1976), Michael *et al* (1999)), However, we will employ a simpler method introduced by Carlos and Song (2009). The method requires that we rewrite our proposed model (1) in the following form:

$$\begin{cases}\dfrac{dX}{dt} = L(X,Z), \\ \dfrac{dZ}{dt} = M(X,Z), \quad M(X,0) = 0, \end{cases} \tag{6}$$

where $X = (S,R) \in \mathbb{R}^2$ denotes (its components) the uninfected individuals and $Z = (E,I) \in \mathbb{R}^2$denotes (its components) the infected individuals. Here, the pneumonia free equilibrium is represented by $\mathbb{E}_0 = (X^*,0)$ where $X^* = \left(\frac{\Lambda}{\mu},0\right)$. $\mathbb{E}_0$ is a globally asymptotically stable equilibrium if the following two conditions are satisfied:

(C1): For$\frac{dX}{dt} = L(X,0)$, $X^*$is globally asymptotically stabile,

(C2): $\frac{dZ}{dt} = D_Z G(X^*,0)Z - \widehat{M}(X,Z)$ where , $\widehat{M}(X,Z) \geq 0$ for all $(X,Z) \in \Omega$.

In condition (C2) above, $D_Z G(X^*,0)$ is known as the Metlzer matrix with nonnegative off-diagonal elements and Ω is the region where the model makes biological sense. If the system (6) satisfies conditions (C1) and (C2), then the following theorem holds.

Theorem 3: The equilibrium point $\mathbb{E}_0 = (X^*,0)$of the system (6) is globally asymptotically stable if $R_0 < 1$ and conditions (C1) and (C2) are satisfied.

Proof: From the system (1), it is easy to see that

$$\frac{dX}{dt} = L(X,Z) = \begin{bmatrix} \Lambda + \delta R - \mu S \\ \psi E + \beta I - (\mu+\delta)R \end{bmatrix}, \tag{7}$$

and

$$\frac{dZ}{dt} = M(X,Z) = \begin{bmatrix} \frac{\chi I}{N}S - (\mu+\gamma+\psi)E \\ \gamma E - (\mu+\beta+\eta)I \end{bmatrix}. \tag{8}$$

Now, we consider the reduced system

$$\left.\frac{dX}{dt}\right|_{Z=0} = L(X,0) = \begin{bmatrix} \Lambda - \mu S \\ 0 \end{bmatrix}. \tag{9}$$

Clearly, $X^* = \left(\frac{\Lambda}{\mu},0\right)$is a globally asymptotically stable equilibrium point of the system$\frac{dX}{dt} = L(X,0)$. To verify this, it is easy to see from (9) that $S(t) \leq \frac{\Lambda}{\mu} + \left(S(0) - \frac{\Lambda}{\mu}\right)e^{-\mu t}$. This implies the global convergence of (9) in Ω since the solution $S(t)$ approaches $\frac{\Lambda}{\mu}$as $t \longrightarrow \infty$. Next, we obtain

$$D_Z G(X^*,0) = \begin{bmatrix} -(\mu+\gamma+\psi) & \chi \\ \gamma & -(\mu+\beta+\eta) \end{bmatrix}.$$

Hence, by (C2) we have

$$\widehat{M}(X,Z) = \begin{bmatrix} \chi\left(1 - \frac{S}{N}\right) \\ 0 \end{bmatrix}.$$

Since$\left(1 - \frac{S}{N}\right) > 0$, then $\widehat{M}(X,Z) = AZ - M(X,Z) \geq 0$for all $(X,Z) \in \Omega$. Thus, the conditions (C1) and (C2) are satisfied and the conclusion that the pneumonia free equilibrium of the model (1) is globally asymptotically stable follows immediately.

Pneumonia Endemic Equilibrium: In the presence of pneumonia within the population, that is when $E_e \geq 0, I_e \geq 0, R_e \geq 0$, the model (1) admits an equilibrium point known as the pneumonia endemic equilibrium and denoted by $\mathbb{E}_e = (S_e, E_e, I_e, R_e)$. Basically, this is obtained as a steady solution of (1) when pneumonia persists within the population. Noting that at the $\varpi_e = \frac{\chi I_e}{N_e}$ at $\mathbb{E}_e$where $N_e = S_e + E_e + I_e + R_e$, we equate the right hand side of each equation in (1) to zero and solve the resulting steady state system of equations to obtain

$$\begin{cases} S_e = \dfrac{((\mu+\psi+\delta)k_2+\gamma(\mu+\beta+\delta))\Lambda}{(\delta+\mu)((R_0-1)k_1k_2+\gamma\mu+k_2\mu)-((R_0-1)\delta-\mu)(\beta\gamma+k_2\psi)}, \\ E_e = \dfrac{k_2\Lambda(\delta+\mu)(R_0-1)}{(\delta+\mu)((R_0-1)k_1k_2+\gamma\mu+k_2\mu)-((R_0-1)\delta-\mu)(\beta\gamma+k_2\psi)}, \\ I_e = \dfrac{\Lambda\gamma(\delta+\mu)(R_0-1)}{(\delta+\mu)((R_0-1)k_1k_2+\gamma\mu+k_2\mu)-((R_0-1)\delta-\mu)(\beta\gamma+k_2\psi)}, \\ R_e = \dfrac{\Lambda(\beta\gamma+k_2\psi)(R_0-1)}{(\delta+\mu)((R_0-1)k_1k_2+\gamma\mu+k_2\mu)-((R_0-1)\delta-\mu)(\beta\gamma+k_2\psi)}. \end{cases} \tag{10}$$

Lemma 4: For $R_0 > 1$, a unique pneumonia endemic equilibrium $\mathbb{E}_e$ exists and there is no other endemic equilibrium otherwise.

Proof: For the disease to persist within the population, it must be that

$$\frac{dE}{dt} = \frac{\chi I}{N}S - (\mu+\gamma+\psi)E > 0, \tag{11}$$

and

$$\frac{dI}{dt} = \gamma E - (\mu+\beta+\eta)I > 0. \tag{12}$$

From eq. 11, we obtain

$$(\mu+\gamma+\psi)E < \frac{\chi I}{N}S \implies E < \frac{\chi SI}{N(\mu+\gamma+\psi)}. \qquad (13)$$

Also, from eq. 12, we have

$$I < \frac{\gamma E}{(\mu+\beta+\eta)}. \qquad (14)$$

Substituting eq. 13 into eq. 14, we have

$$I < \frac{\gamma\chi SI}{(\mu+\gamma+\psi)(\mu+\beta+\eta)N} \implies 1 < \left(\frac{\gamma\chi}{(\mu+\gamma+\psi)(\mu+\beta+\eta)}\right)\frac{S}{N}.$$

This implies

$$1 < \frac{\gamma\chi}{(\mu+\gamma+\psi)(\mu+\beta+\eta)} = R_0.$$

Therefore, a unique pneumonia endemic equilibrium exists when $R_0 > 1$.

Theorem 5: If $R_0 > 1$, the pneumonia endemic equilibrium $\mathbb{E}_e$ of the system (1) is globally asymptotically stable.

Proof: To establish the global stability of the endemic equilibrium $\mathbb{E}_e$,we define the following Lyapunov function:

$$V(S^*, E^*, I^*, R^*) = \left(S - S^* - S^* \ln\frac{S}{S^*}\right) + \left(E - E^* - E^* \ln\frac{E}{E^*}\right) + \left(I - I^* - I^* \ln\frac{I}{I^*}\right) + \left(R - R^* - R^* \ln\frac{R}{R^*}\right).$$

Calculating the derivative of *V* along the solution of (1), we obtain

$$\frac{dV}{dt} = \left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + \left(1 - \frac{E^*}{E}\right)\frac{dE}{dt} + \left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} + \left(1 - \frac{R^*}{R}\right)\frac{dR}{dt}.$$

This implies

$$\frac{dL}{dt} = A - B.$$

where

$$A = \Lambda + \delta R + (\mu+\varpi)S^* + (\mu+\gamma+\psi)E^* + (\mu+\beta+\eta)I^* + (\mu+\delta)R^* + \varpi S + (\gamma+\psi)E + \beta I,$$

and

$$B = \frac{\Lambda S^*}{S} + \frac{\delta R S^*}{S} + (\mu+\varpi)S + (\mu+\gamma+\psi)E + (\mu+\beta+\eta)I + (\mu+\delta)R + \frac{\varpi S E^*}{E} + \left(\frac{\gamma I^*}{I} + \frac{\psi R^*}{R}\right)E + \frac{\beta I R^*}{R}.$$

Observe that $\frac{dV}{dt} = 0$ if and only if $S = S^*, E = E^*, I = I^*, R = R^*$ and $\frac{dV}{dt} \leq 0$ if$A < B$. It follows that the largest compact invariant set in $\left\{(S_e, E_e, I_e, R_e) \in \Gamma: \frac{dV}{dt} = 0\right\}$ is the singleton set $\mathbb{E}_e$ which is the endemic equilibrium of the system (1). Therefore, by LaSalle's invariant principle [18], the pneumonia endemic equilibrium $\mathbb{E}_e$is globally asymptotically stable in Ω.

BIFURCATION ANALYSIS

A bifurcation is a qualitative change in the nature of the solution trajectories resulting from a parameter change. The point at which this change occurs is termed a bifurcation point. At the point of bifurcation, a number of equilibrium points, or their stability properties, or both, change. In the present work, we will investigate the nature of the bifurcation of the solution trajectories of the model (1) by using the method introduced by Carlos and Song (2009), and the method is based on the use of the center manifold theory.

Theorem 6 (Centre manifold theory): Let $f: \mathbb{R}^n \times \mathbb{R}$ *and* $f \in C^2(\mathbb{R}^n \times \mathbb{R})$. *Then consider the following general system of ODEs with a parameter* ϕ.

$$\frac{dx}{dt} = f(x, \phi), \qquad (*)$$

where $x = 0$ *is an equilibrium point of the system* $(*)$ *(that is,* $f(0, \phi) \equiv 0 \ \forall \ \phi$*). Assume that:*

A1: $A = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_j}(0,0)\right)$ is the linearization matrix of matrix of the system $(*)$ around the equilibrium point 0 with ϕ evaluated at 0. Zero is a simple eigenvalue of A and other eigenvalues of A have negative real parts;

A2: Matrix A has a (non-negative) right eigenvector w and a left vector v (each corresponding to the zero eigenvalue).

Let f_k be the k^{th} component of f and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \quad \text{and} \quad b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0). \qquad (**)$$

Then the local dynamics of the system $(*)$ *around 0 is totally determined by the signs* of a *and* b*:*

i. $a > 0, b > 0$. *When* $\phi < 0$ *with* $|\phi| \leq 1$, 0 *is locally asymptotically stable, and there exists a positive unstable equilibrium; when* $0 < \phi \ll 1$,0 *is unstable and there exists a negative and locally asymptotically stable equilibrium.*

ii. $a < 0, b < 0$. $When\ \phi < 0\ with\ |\phi| \ll 1$, 0 *is unstable; when* $0 < \phi \ll 1$,0 *is locally asymptotically stable, and there exists a positive unstable equilibrium.*

iii. $a > 0, b < 0$. $When\ \phi < 0\ with\ |\phi| \ll 1$, 0 *is unstable, and there exists a locally asymptotically stable negative equilibrium; when* $0 < \phi \ll 1$,0 *is stable, and a positive unstable equilibrium appears.*

iv. $a < 0, b > 0$. *When* ϕ *changes from negative to positive,* 0 *changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.*

Particularly, if $a > 0$ and $b > 0$, *then a backward bifurcation occurs at* $\phi = 0$.

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Theorem 7: The model in system (1) exhibits forward bifurcation at R_0.

Proof: Using the center manifold theory, we make the change of variables: $S = x_1$, $E = x_2$, $I = x_3$, $R = x_4$in our proposed model (1). Moreover, by using the vector notations $X = (x_1, x_2, x_3, x_4)^T$ and $F = (f_1, f_2, f_3, f_4)^T$so that $\frac{dX}{dt} = F(X)$ the proposed pneumonia model (1) can be re-written in the form

$$\begin{cases} \frac{dx_1}{dt} = \Lambda + \delta x_4 - \left(\mu + \frac{\chi I}{N}\right)x_1, \\ \frac{dx_2}{dt} = \frac{\chi I}{N}x_1 - (\mu + \gamma + \psi)x_2, \\ \frac{dx_3}{dt} = \gamma x_2 - (\mu + \beta + \eta)x_3, \\ \frac{dx_4}{dt} = \psi x_2 + \beta x_3 - (\mu + \delta)x_4. \end{cases} \quad (15)$$

We take the transmission rate χ as the bifurcation parameters so that at $R_0 = 1$, we have

$$\chi = \chi^* = \frac{(\beta + \eta + \mu)(\gamma + \mu + \psi)}{\gamma}. \quad (16)$$

The pneumonia free equilibrium is given as $\left(x_1 = \frac{\Lambda}{\mu}, x_2 = 0, x_3 = 0, x_4 = 0\right)$. Then the linearization matrix of the system of eq. 15 at the pneumonia free equilibrium is given by

$$J_{\mathbb{E}_0} = \begin{bmatrix} -\mu & 0 & -\chi^* & \delta \\ 0 & -k_1 & \chi^* & 0 \\ 0 & \gamma & -k_2 & 0 \\ 0 & \psi & \beta & -(\mu + \delta) \end{bmatrix}.$$

The right eigenvector $w = (g_1, g_2, g_3, g_4)^T$ of the matrix $J_{\mathbb{E}_0}$associated with the simple zero eigenvalue can be obtained from $J_{\mathbb{E}_0} w = 0$. These eigenvectors are obtained as

$$g_2 = g_2 > 0;$$
$$g_3 = \frac{\gamma g_2}{(\mu+\beta+\eta)};$$
$$g_4 = \frac{\psi g_2 + \beta g_3}{\mu+\delta} = \frac{(\psi(\mu+\beta+\eta) + \beta\gamma)g_2}{(\mu+\delta)(\mu+\beta+\eta)};$$
$$g_1 = \frac{\delta g_4 - \chi g_3}{\mu} = \frac{[(\mu+\beta+\eta)\psi\delta + \beta\gamma\delta - \chi\gamma(\mu+\delta)]g_2}{\mu(\mu+\delta)(\mu+\beta+\eta)}.$$

Next, the left eigenvector $v = (h_1, h_2, h_3, h_4)$associated with the simple zero eigenvalue can be obtained from $vJ_{\mathbb{E}_0} = 0$. These eigenvectors are obtained as:

$$h_1 = h_4 = 0, \quad h_2 = h_2 > 0, \quad h_3 = \frac{\chi h_2}{(\mu + \beta + \eta)}.$$

Next, we compute a and b using eq. $**$. Considering only the non-zero components $(h_2, h_3)$of the left eigenvectors, we have

$$a = -\frac{2\gamma\chi\mu}{\Lambda}\left(\frac{(\delta+\mu)(1+\beta+\eta+\mu)+\beta\gamma+(\beta+\eta+\mu)\psi}{(\delta+\mu)(\beta+\eta+\mu)^2}\right)g_2^2 h_2 < 0,$$
$$b = \frac{\gamma}{(\mu+\beta+\eta)}g_2 h_2 > 0.$$

Since the sign of the coefficient b is positive and the sign of the coefficient a is negative, the pneumonia model (1) exhibits a forward bifurcation and there exists at least one stable endemic equilibrium when $R_0 > 1$. Using expression for I_e in eq. 10 and the parameter values provided in Table 2, we plotted a forward bifurcation diagram as shown in Fig. 2.

Table II: Parameter values of the model

Parameter symbol	Value	Source
X	0.376	Estimated
γ	0.001 to 0.01095 per day	Assumed
μ	0.0012	Estimated
β	0.2	Tilahun, G. T. (2017)
ψ	0.02	Assumed
η	0.057	Tilahun, G. T. (2017)
Λ	0.001	Assumed
δ	0.1	Tilahun, G. T. (2017)

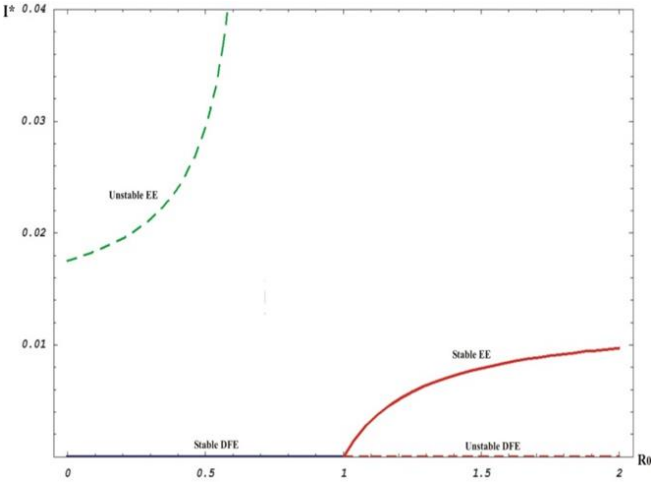

Figure 2: Forward bifurcation of the pneumonia model (1).

SENSITIVITY ANALYSIS OF MODEL PARAMETERS

By sensitivity analysis of the parameters of the basic reproduction number, it becomes obvious which parameter has high impact on the basic reproduction number. We compute the sensitivity indices of R_0 to the model parameters following the same approach used by Blower and Dowlatabadi (1994). The normalized forward sensitivity index of the basic reproduction number R_0 that depends differentiability index on a parameter υ, is defined as

$$\zeta_{\upsilon}^{R_0} = \frac{\partial R_0}{\partial \upsilon} \times \frac{\upsilon}{R_0} \quad (17)$$

Thus, we have

$$\begin{cases} \zeta_{\chi}^{R_0} = \frac{\gamma}{(\beta+\eta+\mu)(\gamma+\mu+\psi)} \times \frac{(\beta+\eta+\mu)(\gamma+\mu+\psi)}{\gamma} > 0; \\ \zeta_{\gamma}^{R_0} = \frac{\chi(\mu+\psi)}{(\beta+\eta+\mu)(\gamma+\mu+\psi)^2} \times \frac{(\beta+\eta+\mu)(\gamma+\mu+\psi)}{\chi} > 0; \\ \zeta_{\psi}^{R_0} = -\frac{\gamma\chi}{(\beta+\eta+\mu)(\gamma+\mu+\psi)^2} \times \frac{(\beta+\eta+\mu)\psi(\gamma+\mu+\psi)}{\gamma\chi} < 0; \\ \zeta_{\mu}^{R_0} = -\frac{\gamma\chi(\beta+\gamma+\eta+2\mu+\psi)}{(\beta+\eta+\mu)^2(\gamma+\mu+\psi)^2} \times \frac{\mu(\beta+\eta+\mu)(\gamma+\mu+\psi)}{\gamma\chi} < 0; \\ \zeta_{\eta}^{R_0} = -\frac{\gamma\chi}{(\beta+\eta+\mu)^2(\gamma+\mu+\psi)} \times \frac{\eta(\beta+\eta+\mu)(\gamma+\mu+\psi)}{\gamma\chi} < 0; \\ \zeta_{\beta}^{R_0} = -\frac{\gamma\chi}{(\beta+\eta+\mu)^2(\gamma+\mu+\psi)} \times \frac{\beta(\beta+\eta+\mu)(\gamma+\mu+\psi)}{\gamma\chi} < 0\,. \end{cases} \quad (18)$$

Using (18) together with the parameter values in Table 2 as obtained from literature, the sensitivity indices of the parameters are as shown in Table 2 with the list sorted in descending order of sensitivity index.

Table III: Sensitivity Indices of the Parameters of R_0

Parameter	*Value* (day^{-1})	*Source*	*Sensitivity Index*
χ	0.376	Estimated	+1.000000
γ	0.005975	Assumed	+0.780129
μ	0.0012	Estimated	-0.0488058
η	0.057	Tilahun, G. T. (2017)	-0.220759
ψ	0.02	Assumed	-0.735971
β	0.2	Tilahun, G. T. (2017)	-0.774593

Interpretation of Sensitivity Indices: It is obvious from the Table 3 that the rate of transmission, χ, and the rate, γ, at which the exposed individuals becomes infectious are the most sensitive parameters. The value of R_0 increases when the parameter values χ and γ increase while other parameters are kept fixed. Also keeping the values of χ and γ fixed while increasing the values of the other parameters of R_0, decreases the value of R_0.

DISCUSSION AND CONCLUSION

This study has considered a mathematical model and analysis of pneumonia infection dynamics. The model subdivided the population into four mutually-exclusive time-dependent compartments by using a system of non-linear ordinary differential equations. In analyzing the qualitative behavior of the model, we considered the non-negativity of solution, invariant region and boundedness of solution, disease-free equilibrium, endemic equilibrium, basic reproduction number, stability analysis and sensitivity analysis. It was established that at any instant, all compartments of the model assume positive values, and the total population is bounded. The basic reproduction, which was obtained via the next generation matrix, showed that when an infective individual is introduced into an entirely susceptible population, susceptible individuals become exposed and joins the exposed compartment at the rate χ, thereafter these exposed individuals become infectious and join the infectious compartment at the rate γ. The total time spent by these infected individuals both in the exposed compartment and the infectious compartment is $(\mu + \gamma + \psi)(\mu + \beta + \eta)$. It was also established that the pneumonia free equilibrium is locally asymptotically stable if $R_0 < 1$, and the pneumonia endemic equilibrium is globally asymptotically stable if $R_0 > 1$. The centre manifold theory was employed to study the bifurcation analysis, and we observed that the system exhibits forward bifurcation. The sensitivity analysis revealed that the rate of transmission, χ, and the rate, γ, at which the exposed individuals becomes infectious are the most sensitive parameters.

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