Applied Mathematics & Information Sciences
An International Journal

http://dx.doi.org/10.18576/amis/170111

Mathematical Modeling of a Bimodal Pneumonia Epidemic with Non-breastfeeding Class

Fekadu Mosisa Legesse, Koya Purnachandra Rao and Temesgen Duressa Keno*

Department of Mathematics, Wollega University, P.O.Box 395 Nekemte, Ethiopia

Received: 2 Sep. 2022, Revised: 22 Nov. 2022, Accepted: 4 Dec. 2022
Published online: 1 Jan. 2023

Abstract: The world health organization (WHO) recommends breastfeeding is mandatory for two years. To support this recommendation several research work have been done to suggest that breastfeeding has clear advantages, specially in eradicating morbidity and mortality due to infectious diseases like pneumonia in childhood. Having these in consideration this work divides the susceptible individuals in to two different classes depending on the level of adequate nutrition particularly breastfeeding to study the transmission dynamics of Pneumonia. In addition the effect of hygiene care for children under five years is considered. The aim of this work is to examine the effect of breastfeeding and hygiene care in reducing the risks associated with pneumonia. This studies also compared breastfed with non-breastfed children that breastfed children have the highest protective immunity against infectious disease (Pneumonia). Finally, we performed a large scale of numerical simulations to verify the theoretical work and the result of the simulation reveals that hygiene care and infection reduction due to breastfeeding decreases the transmission dynamics of pneumonia disease.

Keywords: Breastfeeding, Pneumonia, Hygiene care, Un-breastfeeding, Sensitivity analysis, Basic reproduction number

1 Introduction

Pneumonia is one of an acute respiratory infection disease caused by pathogens (such as bacterial, viral, fungal) or other pathogens [1]. Among it bacterial pneumonia is the most common which is caused by Streptococcus Pneumonia, also known as pneumococcus and it affects the respiratory system specially lungs-particularly the alveoli [2,3]. The alveoli are small air sacs in the lungs that filled with pus or fluid in children with pneumonia, resulting in difficult to breathe, painful breathing, difficulty to intake oxygen, and ultimately a high risk of death [4].

Globally among lower acute respiratory infections (LARI) diseases, pneumonia and bronchiolitis, are a leading cause of death among infants and children of under five years of age which are parts of the population [5]. As a data conducted from United Nations Children's Fund, in 2015 within an hour 100 children died due to pneumonia and related causes, and in developing countries, 20/100 of death due to pneumonia occur compared to 4/100 in developed countries [3]. According to the WHO(World Health Organization, 2021), pneumonia killed 740180 children from under five years in 2019 which accounts 14/100 of all death of children under the age of 5 years old but 22/100 of all deaths in children aged from one to five [6]. The majority of deaths (99/100) due to pneumonia among children of less than five years old occur in developing countries [7].

The risk factors associated with pneumonia incidence and severity includes: parental smoking, malnutrition and conditions of poverty, living in crowded conditions, lack of breastfeeding or exclusive breastfeeding, pre-existing illnesses, indoor air pollution, alcoholism and drug abuse [1,3]. Since all the above increases a children susceptibility to pneumonia, among thus lack of breastfeeding is a vital one globally, especially in developing countries [1]. The risk of mortality and morbidity due to pneumonia and other infections can increase in the infants who are not breastfeed at all or partially breastfeed. For instance, according to WHO 45/100 of children deaths are associated with under-nutrition like un-breastfeed [8,9].

While healthy children most of the time fight against infection with their natural immunity, and children whose immune systems are compromised are at high risk to develop pneumonia. Since a children immune system may be weakened either by malnutrition or undernourishment, especially in infants who are not breastfed totally or

* Corresponding author e-mail: fekadumosisa22@gmail.com[1]*

exclusively breastfeed, then breastfeeding should be done to any children as possible [6]. Whey protein is the main element of breast milk that gives strengthens for infants' immune system. This whey protein contains lactoferrin, secretory IgA, lysozymes, and bifidus factor-which ultimately protect breastfeed infants better than their non-breastfeed counterparts [10]. Hence, breast milk is undoubtedly the ideal food for a human child. It also gives for children the best start in life. It is estimated that over one million children die each year due to diarrhea, respiratory and other infections because they are not effectively breastfed or not breastfeed totally. Most of children suffer from unnecessary illnesses (morbidity) that they would not have if they were breastfeed. Breastfeeding also helps to protect mothers' health [11]. Low rates of breastfeeding has its own contribution on infectious diseases (pneumonia) cases [12]. Effectively breastfeeding decreases pneumonia cases among children under-fives. Additionally feeding early infants with human breastfeed increases brain growth and intelligence quotients, and has an impact on mental development and reduces the prevalence of necrotizing enterocolitis [10].

In developing countries non-breastfed children experience a 14-fold increase in all-cause mortality compared to those who are exclusively breast-fed for 6 months [13]. There are a few reasons why someone should not or may not be able to breastfeed their baby. For example, some parents cannot produce a healthy breast milk supply, while others may take certain medications or need to undergo a medical treatment that is not breastfeeding safe. There are also a few medical conditions that are not compatible with breastfeeding. In addition, because of vertical transmission of infectious diseases like HIV, Ebola, [14] and the like a mother can pass the virus to his/her child through breastfeeding, in this case breastfeed is not allowed and also some children are not breastfeed due to death of mother during delivery or after delivery due to different cases.

Mathematical model plays an important role to understand easily the transmission dynamics of infectious diseases such as pneumonia, cholera, HIV aids and so on. The studies on these infectious diseases highlight how to reduce or control the spread and predict its future damage on the lives and economies of the country. Interestingly, there are so many mathematical models developed on the transmission dynamics and control mechanisms of pneumonia infection, but non of them consider susciptable variable as breastfeed and non-breastfeed to show the impact of breastfeed on the reduction of infectious diseases particularly pneumonia. Most of the studies considered above did not consider a compartmental approach which is used to develop the mathematical model for pneumonia transmission dynamics. The authors in [15,16], studied on the transmission dynamics of infectious diseases using mathematical model which gives a simple framework for our understanding about the dynamics of infectious [17].

Very few research works have been done in the last decade on the transmission dynamics of pneumonia which are abound and flourishing in the following literature [18,19], to cite a few and the references therein. Additionally, [20] studied the effect of hygiene care and breastfeeding by developing and analyzing MSEIR deterministic model for the transmission dynamics of pneumonia. But, none of the above scholars considered and incorporated breastfeeding and un-breastfeeding as two susceptible variables in their model with hygiene care efficiency simultaneously to the best of the researcher's knowledge. Hence, we are interested by the above-reviewed researches to undertake this study for fulfilling this entire gap.

Generally, the rest of this paper is organized as follows: In section two of this paper, mathematical model is formulated, parameters and state variables are also discribed therein. The third section of this study is devoted to how to compute equilibrium points and the basic reproductive number to deals with the local and global stability analysis of both of the equilibrium points followed by sensitivity analysis of the basic reproduction is conducted. In the fourth part numerical simulation is carried out. Lastly, discussions and conclusions are provided.

2 Dynamic Model Formulation and Description of Pneumonia Epidemic

Mathematical modeling is the best techniques which is used to analyzing the epidemiology of a disease. For the proposed model, the total population $N(t)$ is divided into five compartmental model according to their disease status. These, different classes include the number of susceptible individuals without breastfeeding (S_1), the number of susceptible individuals with breastfeeding (S_2), the number of carrier individuals (C), the number of infected individuals (I) and the number of recovered individuals (R).

Susceptible class is divided into two classes as breastfeeding and un-breastfeeding childes. Note that breastfeed susceptible children (S_2) have better immune to pneumonia comparing to the un-breastfeed susceptible children (S_1). Therefore, the un-breastfeed susceptible children (S_1) will be infected in contact with the infected populations (C and I) with higher rate in comparison with the breastfeed susceptible children (S_2). Thus, the transmission rate for un-breastfeed susceptible children (S_1) should be considered larger than this rate for breastfeed susceptible childes (S_2) and their force of infection are respectively given as, $\lambda_1 = \psi((I+\omega C)/N)$ and $\lambda_2 = k\lambda_1$ where $0 < k \leq 1$ and k represents the infection reduction of breastfeed susceptible children relative to un-breastfeed ones.

The parameters $\lambda_1, \lambda_2 \geq 0$, are shows the rate (force of infection) at which a susceptible individual can be

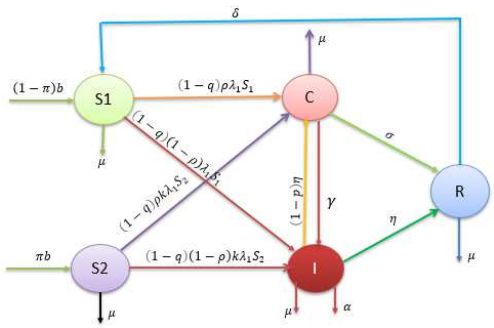


Fig. 1: Flow diagram of the S_1S_2CIR model

infected with pneumonia if he/she comes into effective contact with an infected individual. A composite parameter measuring the contact rate and the probability of transmission upon contact is given by $\psi = \kappa\beta \geq 0$ were β is a contact rate and κ is the probability of effective contact rate.The term $(I+\omega C)/N$ in λ_1 denotes the density or prevalence of infected and carrier individuals in the population where $\omega \in [0,1]$ is, the reduction in transmissible of carrier individuals. Note that the nonlinearity of the incidence rate is one of the key features of dynamic infectious disease models, because to model a population of individuals, the status of each individual is required. The transmission dynamics of pneumonia epidemic associated with the above five compartments are illustrated in Figure 1 and their sum gives as a total population size at time t which is denoted by N(t) and given by:

$$N(t) = S_1(t) + S_2(t) + C(t) + I(t) + R(t) \qquad (1)$$

Here we assumed that once the un-breastfeed or breastfeed children are infected, they cannot regain their previous innate immunity at the beginning of recovery period. Thus, the recovered individuals become re-infected and joins the un-breastfeed susceptible class due to lower immunity. Which mean that as an individuals once infected by any infectious diseases and recovered from it; during infection period they lose some amount of their immunity and they cannot regain their previous immunity at the beginning of their recovery duration. Hence the recovered individuals can re-infected again and joins S1 rather than S2 because of this conditions.
The non-linear mathematical model of pneumonia epidemic from the above flow chart is given as:

$$\begin{cases} \frac{dS_1}{dt} = (1-\pi)b + \delta R - (1-q)\lambda_1 S_1 - \mu S_1 \\ \frac{dS_2}{dt} = \pi b - (1-q)k\lambda_1 S_2 - \mu S_2 \\ \frac{dC}{dt} = (1-q)\rho\lambda_1 S_1 + (1-q)k\rho\lambda_1 S_2 - (1-p)\eta I \\ \qquad -(\sigma+\gamma+\mu)C \\ \frac{dI}{dt} = (1-q)(1-\rho)\lambda_1 S_1 + (1-q)(1-\rho)k\lambda_1 S_2 + \\ \qquad \gamma C - (\eta+\alpha+\mu)I \\ \frac{dR}{dt} = \sigma C + p\eta I - (\mu+\delta)R \end{cases} \qquad (2)$$

With the following initial conditions

$$S_1(0) \geq 0, S_2(0) \geq 0, C(0) \geq 0, I(0) \geq 0, R(0) \geq 0 \qquad (3)$$

In this model,b represents recruitment rate of individuals into the population where π is the proportion of recruited (migrated) childes into the population, with a fraction π breastfeed and the remaining $1-\pi$ un-breastfeed susceptible when entering the community. Newly infected individuals from both of susceptible class can be infected by force of infection and join carrier class C with the probability of ρ or transformed to the infected class I with probability of $1-\rho$. The total population in this model are decreased by two different rates μ and α, where μ denotes the rate of natural death that decreases populations of all compartments. Where as α is the rate of pneumonia induced death and which decreases only the population of infected class. Additionally if the carrier class shows disease symptom, then we can move it in to the infected class with a rate of γ and move to recovery class if they can gain natural immunity by a per capita rate σ. The individuals in infected compartment move to recovered class at η rate by treatment, with effectiveness of treatment p of proportion of individuals move to the recovered class or join the carrier compartment with (1-p) of proportion by adapting the treatment. Finally those who recover due to different treatment intervention or their natural immunity can possibly lose the immunity and be infected again, therefore they can rejoin the susceptible group at the rate of δ. And q is the percentage of effectiveness hygiene care with $0 \leq q < 1$.

3 Analysis of the model

3.1 Invariant region

Since the model given as a system on equation (2) shows human beings, it is assumed that all parameters and state variables in (2) are positive $\forall t \geq 0$. In this region we discuss the epidemiological sense of solution of (2) which means the solution of the system should be well posed and biologically meaning full in this region.

Theorem 1.*The region* $\Omega = \{(S_1,S_2,C,I,R) \in R_+^5 : N(t) \leq \frac{b}{\mu}\}$ *is positively invariant under the flow induced by (2) which means the corresponding should be well posed and biologically meaning full in this region.*

Proof: From the total population size at any time t we have:

$$\frac{dN}{dt} = b - \mu(S_1+S_2+C+I+R) - \alpha I \qquad (4)$$

From the (4) and equation (1) we have

$$\frac{dN}{dt} = b - \mu N - \alpha I \qquad (5)$$

If mortality due to pneumonia doesn't exist,then equation (5) becomes:

$$\frac{dN}{dt} = b - \mu N \tag{6}$$

Use separation of variable and integrate both sides of equation (6) with respect to t gives:

$$b - \mu N \geq Ae^{-\mu t} \tag{7}$$

use the initial condition $N(0) = N_0$ and apply some rearrangement on (7) gives

$$N \leq \frac{b}{\mu} - \frac{b - \mu N_0}{\mu} e^{-\mu t} \tag{8}$$

Therefore, the feasible steady states set of the model enters and remain in the region

$$\Omega = \left\{ (S_1, S_2, C, I, R) \in R_+^5 : N(t) \leq \frac{\mu}{b} \right\}$$

Therefore, the region Ω is a positively invariant set and on this set the basic model is well posed epidemiologically and mathematically. Hence, it is sufficient to study the dynamics of the model in Ω.

3.2 Positivity of the solutions

This section is devoted to the non-negativity of the steady state of the proposed model system (2)

Theorem 2.*Let* $\Omega = \{(S_1, S_2, C, I, R) \in R_+^5 : S_1(0) > 0, S_2(0) > 0, C(0) > 0, I(0) > 0, R(0) > 0\}$ *then the steady state set* S_1, S_2, C, I, R *are all positive for all* $t \geq 0$.

Proof: We can verify this theorem by choosing any one of equation from system (2) above. Let us take the first equation of system (2)

$$\frac{dS_1}{dt} = (1-\pi)b + \delta R - (1-q)\lambda_1 S_1 - \mu S_1$$

which implies

$$\frac{dS_1}{dt} \geq -[(1-q)\lambda_1 - \mu]S_1 \tag{9}$$

Integrating both sides (9) with respect to t will give as:

$$S_1(t) \geq Ae^{-((1-q)\lambda_1 - \mu)t} \tag{10}$$

Using initial condition $S_1(0) = S_{10}$ and (10) we obtain:

$S_1(t) \geq S_{10}e^{-((1-q)\lambda_1-\mu)t} \geq 0$ where $S_{10} = A$.
In a similar fashion, the remaining equations of system (2) can be proved and gives the following out puts:
$S_2(t) \geq S_{20}e^{-((1-q)k\lambda_1-\mu)t} \geq 0$ where $S_{20} = e^{c_2}$
$C(t) \geq C_0 e^{-(\sigma+\gamma+\mu)t} \geq 0$ where $C_0 = e^{c_3}$
$I(t) \geq I_0 e^{-(\eta+\alpha+\mu)t} \geq 0$ where $I_0 = e^{c_4}$
$R(t) \geq R_0 e^{-(\mu+\delta)t} \geq 0$ where $R_0 = e^{c_5}$
which is the complete proof of the theorem.

Hence all solutions of the model system (2) are positive $\forall t \geq 0$ in Ω.

3.3 Disease free-equilibrium of the Model

To obtain the disease-free equilibrium (DFE) point (E_0) of the model equate the right-hand side of system (2) to zero and make the disease state variables C=0 and I=0. After that solving the remaining equation will give us:

$$E_0 = \left(\frac{(1-\pi)b}{\mu}, \frac{\pi b}{\mu}, 0, 0, 0 \right) \tag{11}$$

3.4 The Basic Reproductive Number$(\Re_0)$

The basic reproduction number is defined as the average number of secondary infections produced by a single infected individual in a completely susceptible population by a typical infected individual through out his/her infectious entire lifetime. To calculate the basic reproduction number use the next generation matrix method [21] and which is calculated as follows. From equation (2) and using the notation X=(C,I) we have the following vector functions:

$$\mathscr{F} = \begin{bmatrix} (1-q)\rho\lambda_1 S_1 + (1-q)\rho k\lambda_1 S_2 \\ (1-q)(1-\rho)\lambda_1 S_1 + (1-q)(1-\rho)k\lambda_1 S_2 \end{bmatrix}$$

and

$$\mathscr{V} = \begin{bmatrix} (\sigma+\gamma+\mu)C - (1-p)\eta I \\ (\eta+\alpha+\mu)I - \gamma C \end{bmatrix}$$
$$= \begin{bmatrix} p_1 C - (1-p)\eta I \\ p_2 I - \gamma C \end{bmatrix}$$

where $p_1 = (\sigma+\gamma+\mu)$ and $p_2 = (\eta+\alpha+\mu)$. Shows the appearance of new infections, and the transfer of individuals in to and out of the infected compartments, respectively. The Jacobian matrices of F(X) and V(X) at DFE are, respectively given as:

$$\mathscr{F} = DF(E_0) = \begin{bmatrix} \frac{\partial F_1(E_0)}{\partial C} & \frac{\partial F_1(E_0)}{\partial I} \\ \frac{\partial F_2(E_0)}{\partial C} & \frac{\partial F_2(E_0)}{\partial I} \end{bmatrix}$$

where $F_1(t) = (1-q)\rho\lambda_1 S_1 + (1-q)\rho k\lambda_1 S_2$ and $F_2(t) = (1-q)(1-\rho)\lambda_1 S_1 + (1-q)(1-\rho)k\lambda_1 S_2$ and

$$\mathscr{V} = DF(E_0) = \begin{bmatrix} \frac{\partial V_1(E_0)}{\partial C} & \frac{\partial V_1(E_0)}{\partial I} \\ \frac{\partial V_2(E_0)}{\partial C} & \frac{\partial V_2(E_0)}{\partial I} \end{bmatrix}$$

where $V_1 = (\sigma+\gamma+\mu)C - (1-p)\eta I$ and $V_2 = (\eta+\alpha+\mu)I - \gamma C$. Now it is easy to calculate the inverse of V and given by:

$$\mathscr{V}^{-1} = \frac{1}{p_1 p_2 - (1-p)\eta\gamma} \begin{bmatrix} p_2 & (1-p)\eta \\ \gamma & p_1 \end{bmatrix}$$

Using the next-generation matrix

$$\mathscr{F}\mathscr{V}^{-1} = \frac{(1-q)(1+\pi(k-1))\psi}{p_1 p_2 - (1-p)\eta\gamma} \begin{bmatrix} \rho k_1 & \rho k_2 \\ (1-\rho)k_1 & (1-\rho)k_2 \end{bmatrix}$$

where $k_1 = (\omega p_2 + \gamma)$ and $k_2 = (\omega\eta(1-p) + p_1)$, and the corresponding eigenvalues are: $\lambda_1 = 0$ and

$$\lambda_1 = \frac{(1-q)(1+\pi(k-1))\psi}{p_1p_2 - (1-p)\eta\gamma}(\rho k_1 + (1-p)k_2)$$

The spectral radius (the governing eigenvalue) of the next generation matrix will give as the required basic reproduction number of the model.That is

$$\Re_0 = \frac{(1-q)(1+\pi(k-1))\psi}{p_1p_2 - (1-p)\eta\gamma}(\rho k_1 + (1-p)k_2) \quad (12)$$

General, if $\Re_0 > 1$, then pneumonia infections will persist in the community. If $\Re_0 < 1$, then pneumonia infections will eventually disappear from the populations. This threshold can be used to portray parameters which are most sensitive and important during the infection [21].

3.5 Local and global stability of disease free-equilibrium point

3.5.1 Local stability of disease-free equilibrium (DFE)

In this section, we investigate the local stability of the disease-free equilibrium of the model system (2). For local stability, the spread of infection depends on the initial sizes of the subpopulation. To prove the local stability of the disease-free equilibrium, the eigenvalues of the Jacobian matrix of the system computed at the DFE point are obtained. The Jacobian matrix is obtained from the linearization of the model system (2). The Jacobian matrix J evaluated at DFE point E_0 is given by [22].

Theorem 3.*The disease-free equilibrium point E_0 is locally asymptotically stable if $\Re_0$ is less than unity and unstable if greater than unity.*

Proof. The locally asymptotical stability of DFE (E_0) is analyzed using the sign of the eigenvalues of a Jacobian matrix evaluated at the disease-free equilibrium point E_0.

$$J(E_o) = \begin{bmatrix} -\mu & 0 & B_1 & B_2 & \delta \\ 0 & -\mu & B_3 & B_4 & 0 \\ 0 & 0 & B_5 & B_6 & 0 \\ 0 & 0 & B_7 & B_8 & 0 \\ 0 & 0 & \sigma & p\eta & -(\mu+\delta) \end{bmatrix}$$

where $B_1 = -(1-q)(1-\pi)\omega\psi$, $B_2 = -(1-q)(1-\pi)\psi$, $B_3 = -(1-q)\omega\psi k\pi$, $B_4 = -(1-q)\psi k\pi$, $B_5 = (1-q)\omega\psi k_1 - p_1$, $B_6 = (1-q)\psi k_1 + (1-p)\eta$, $B_7 = (1-q)\omega\psi k_2 - \gamma$ and $B_8 = (1-q)\psi k_2 + p_2$.
By expanding the characteristic equation $|\lambda I - J(E_o)| = 0$ with the first and second columns, we obtain two eigenvalues $\lambda_{1,2} = -\mu$. We calculate the remaining three eigenvalues from the reduced matrix as:

$$J(3) = \begin{bmatrix} B_5 & B_6 & 0 \\ B_7 & B_8 & 0 \\ \sigma & p\eta & -(\mu+\delta) \end{bmatrix}$$

The characteristic equation corresponding to J(3) is given by $|\lambda I - J(3)| = 0$. From this $\lambda_3 = -(\mu+\delta) < 0$ and the other values are obtained from:

$$\lambda_1^2 + D_1\lambda_1 + D_2 = 0$$

where

$$D_1 = -(1-q)\psi(\omega k_1 + k_2) + (\sigma+\gamma+\mu) + (\eta+\alpha+\mu)$$

$$D_2 = -(1-q)\psi(\omega k_1 p_2 + k_2 p_1 + \omega k_2\eta(1-p) + k_1\gamma) + p_1p_2 + (1-p)\eta\gamma$$

By Routh-Hurwitz criteria, $D_1 > 0$ which mean that:

$$-(1-q)\psi(\omega k_1 + k_2) < (\sigma+\gamma+\mu) + (\eta+\alpha+\mu)$$

Following the same criteria, $D_2 > 0$ mean that:

$$D_2 = -\frac{(1-q)\psi}{p_1p_2 + (1-p)\eta\gamma}\Phi + 1$$

where $\Phi = (k_1(\omega p_2 + \gamma) + k_2((1-p)\eta\gamma + p_1))$
From the fact $(S_1 + kS_2)/N = 1 + \pi(k-1) \leq 1$.
Hence

$$-\frac{(1-q)(1+\pi(k-1))\psi}{p_1p_2 + (1-p)\eta\gamma}\Phi + 1 \leq D_2$$

which implies

$$-\Re_0 + 1 \leq D_2$$

$$D_2 = 1 - \Re_0$$

Therefore, the sign of D_2 is positive if $\Re_0 < 1$. Using Routh-Hurwitz stability criterion the disease-free equilibrium point E_0 is stable if $\Re_0 < 1$ and unstable if $\Re_0 > 1$.

Hence, the disease-free equilibrium (E_0) is locally asymptotically stable if $\Re_0 < 1$ otherwise unstable.

3.5.2 Global stability of E_0

In this section we analyze the global stability of disease free-equilibrium point E_0 by following [1]
Proof: To prove the global asymptotic stability of E_0 we start the proof by constructing the Lyapunov function define by:

$$L = k_1C + k_2I$$

Its derivative along the solutions of system (2) is

$$\frac{dL}{dt} = k_1\frac{dC}{dt} + k_2\frac{dI}{dt}$$

$$\frac{dL}{dt} = k_1((1-q)\rho\lambda_1S_1 + (1-q)\rho\lambda_1S_2 - (1-p)\eta I - (\sigma+\gamma+\mu)C) + k_2((1-q)(1-\rho)\lambda_1S_1 + (1-q)(1-\rho)k\lambda_1S_2 + \gamma C - (\eta+\alpha+\mu)I)$$

$$\frac{dL}{dt} = (1-q)\psi\frac{S_1+kS_2}{N}(\rho k_1+(1-\rho)k_2)(I+\omega C) +((1-q)\eta k_1 I - p_2k_2I + k_2\gamma C - p_1k_1C)$$

$$\frac{dL}{dt} \le (1-q)\psi(1+\pi(k-1))(\rho k_1+ (1-\rho)k_2))(I+\omega C)+((1-q)\eta(\omega p_2+\gamma)- p_2(\omega\eta(1-p)+p_1))I+((\omega\eta(1-p)+ p_1)\gamma - p_1(\omega p_2+\gamma))C$$

$$\frac{dL}{dt} \le \frac{(1-q)\psi(1+\pi(k-1))}{p_1p_1+(1-p)\eta\gamma}* (\rho k_1+(1-\rho)k_2)(I+\omega C)-(I+\omega C)$$

$$\frac{dL}{dt} \le (\Re_0-1)(I+\omega C) \le 0$$

Clearly $\frac{dL}{dt} \le 0$ if $\Re_0 \le 1$ because all parameter values are positive. Furthermore $\frac{dL}{dt} = 0$ if and only if $\Re_0 = 1$ or $I = C = 0$. Hence, L is a Lyapunov function on Ω and the largest compact invariant set in $\left\{(S_1,S_2,C,I,R)\in\Omega : \frac{dL}{dt}\le 0\right\}$ is the singleton E_0. Therefore, by LaSalle's invariance principle [1] every solution corresponding to system (2), with initial conditions in Ω, close to E_0 as $t\longrightarrow\infty$ if $\Re_0 < 1$.

Hence the E_0 is globally asymptotically stable on the set Ω if $\Re_0 \le 1$ completing the proof.

3.6 Endemic Equilibrium

Endemic equilibrium of the model system (2) is denoted by E_e and defined as a steady state (2). At this equilibrium point (E_e) disease persists in the community. Hence, the endemic equilibrium $E_e = (S_1^*, S_2^*, C^*, I^*, R^*)$ is determined by setting the right-hand side of system (2) equal to zero as follows:

$$\left.\begin{aligned}(1-\pi)b+\delta R-(1-q)\lambda_1S_1-\mu S_1=0\\ \pi b-(1-q)k\lambda_1S_2-\mu S_2=0\\ (1-q)\rho\lambda_1S_1+(1-q)k\rho\lambda_1S_2-(1-p)\eta I\\ -p_1C=0\\ (1-q)(1-\rho)\lambda_1S_1+(1-q)(1-\rho)k\lambda_1S_2\\ +\gamma C-p_2I=0\\ \sigma C+p\eta I-(\mu+\delta)R=0\end{aligned}\right\} \quad (13)$$

Then we get

$$S_1^* = \frac{(1-\pi)b+\delta R^*}{(1-q)\lambda_1^*+\mu}$$

$$S_2^* = \frac{\pi b}{(1-q)k\lambda_1^*+\mu}$$

$$C^* = \frac{(1-\rho)(1-p)\eta+\rho p_2}{(1-\rho)p_1+\rho\gamma}$$

$$I^* = \frac{p_5(kb\pi p_1A_3A_4+(1-\pi)bA_1)}{A_1A_2A_3A_4p_3-p_5A_1\delta A_5}$$

$$R^* = \frac{\sigma((1-\rho)(1-p)\eta+\rho p_2)+p\eta((1-\rho)p_1+\rho\gamma)}{(\mu+\delta)((1-\rho)p_1+\rho\gamma)}$$

where

$$A_1 = (1-q)k\lambda_1^*+\mu$$
$$A_2 = p_1p_2-(1-p)\eta\gamma$$
$$A_3 = (1-q)\lambda_1^*+\mu$$
$$A_4 = (1-\rho)p_1+\rho\gamma$$
$$A_5 = (\sigma(1-\rho)(1-p)\eta+\rho p_2+\delta p\eta A_4)$$

Therefore, $E_e = (S_1^*, S_2^*, C^*, I^*, R^*)$ is the required endemic equilibrium of system 2.

Lemma 3. A unique endemic equilibrium E_e exists for $\Re_0 > 1$, and no endemic equilibrium otherwise.

The proposed disease to be persist when $\frac{dC}{dt} > 0$ and $\frac{dI}{dt} > 0$ that is

$$(1-q)\rho\lambda_1S_1-(1-q)k\rho\lambda_1S_2-(1-p)\eta I-p_1C\ge 0 \quad (14)$$

$$(1-q)(1-\rho)\lambda_1S_1+(1-q)(1-\rho)k\lambda_1S_2+\gamma C-p_2I\ge 0 \quad (15)$$

From Equation (14),

$$p_1C \le (1-q)\rho\psi(I+\omega C)\frac{S_1+kS_2}{N}+(1-p)\eta I$$

From the fact that

$$\frac{S_1+kS_2}{N} = (1+\pi(k-1)) \le 1$$

Then

$$C(t) \le \frac{(1-q)\rho\psi+(1-p)\eta}{p_1-(1-q)\rho\psi\omega} \quad (16)$$

From equation (15) we have

$$p_2I \le (1-q)(1-\rho)k\psi(I+\omega C)\frac{S_1+kS_2}{N}+\gamma I$$
$$\Rightarrow p_2I \le (1-q)(1-\rho)k\psi(I+\omega C)+\gamma I$$

$$(p_2-(1-q)(1-\rho)k\psi)I < ((1-q)(1-\rho)k\psi\omega+\gamma)C \quad (17)$$

substituting (16) in to (17) will give as

$$(p_2-(1-q)(1-\rho)k\psi)I < ((1-q)(1-\rho)k\psi\omega+\gamma)\frac{(1-q)\rho\psi+(1-p)\eta}{p_1-(1-q)\rho\psi\omega}$$
$$\Rightarrow p_1p_2-(1-p)\eta\gamma < (1-q)\psi(\rho k_1+(1-p)k_2)$$
$$\Rightarrow 1 < \frac{(1-q)\psi}{p_1p_2-(1-p)\eta\gamma}(\rho k_1+(1-p)k_2)$$
$$\Rightarrow 1 < \frac{(1-q)(1+\pi(k-1))\psi}{p_1p_2-(1-p)\eta\gamma}(\rho k_1+(1-p)k_2) = \Re_0$$
$$\Rightarrow 1 < \Re_0$$

Thus a unique endemic equilibrium of system (2) exist provided that $\Re_0 > 1$.

3.6.1 Local and global stability of the endemic equilibrium

In this section we analyze local and global stability of the endemic equilibrium by applying the Routh-Hurwitz criterion and LaSalle's invariance principle [23] respectively.

3.6.2 Local stability of the endemic equilibrium

Theorem 4.*The endemic equilibrium (E_e) of system (2) is locally asymptotically stable if* $\Re_0 > 1$.

Proof. To show the local stability of endemic equilibrium first determine the Jacobean matrix of system (2) at endemic equilibrium which is given on (18).

$$J(E_e) = \begin{bmatrix} B_1 & 0 & 0 & 0 & \delta \\ 0 & B_2 & 0 & 0 & 0 \\ B_3 & B_4 & -p_1 & (1-p)\eta & 0 \\ B_5 & B_6 & \gamma & -p_2 & 0 \\ 0 & 0 & \sigma & p\eta & -(\mu+\delta) \end{bmatrix} \tag{18}$$

where $B_1 = -(1-q)\bar{\lambda_1} - \mu$, $B_2 = -(1-q)\bar{\lambda_1}k - \mu$,
$B_3 = (1-q)\rho\bar{\lambda_1}S_1$, $B_4 = (1-q)\rho k\bar{\lambda_1}S_2$,
$B_5 = (1-q)(1-\rho)\bar{\lambda_1}S_1$, $B_6 = (1-q)(1-\rho)k\bar{\lambda_1}S_2$
The trace of the Jacobian matrix (18) is negative and where $\bar{\lambda_1}$ is defined as the force infection at the endemic equilibrium. We obtain the determinant of the Jacobian matrix at endemic equilibrium $(detJ(E_e))$

$$det(J(E_e)) = \begin{vmatrix} B_1 & 0 & 0 & 0 & \delta \\ 0 & B_2 & 0 & 0 & 0 \\ B_3 & B_4 & -p_1 & (1-p)\eta & 0 \\ B_5 & B_6 & \gamma & -p_2 & 0 \\ 0 & 0 & \sigma & p\eta & -(\mu+\delta) \end{vmatrix}$$

$$\begin{aligned} det(J(E_e)) = ((1-q)k+\mu)[-p_1p_2p_3((1-q)\bar{\lambda_1}+\mu) \\ +\delta((1-q)\rho)\bar{\lambda_1}(p\eta\gamma+\sigma p_2)+(1-q)(1-\rho)k\bar{\lambda_1}pp_1\eta \\ +(1-q)(1-\rho)(1-p)\sigma\eta\bar{\lambda_1}] \end{aligned}$$

which is positive if

$$\begin{aligned} ((1-q)k+\mu)p_1p_2p_3((1-q)\bar{\lambda_1}+\mu) < \\ ((1-q)k+\mu)[((1-q)\bar{\lambda_1}+\mu)(1-p)p_3\eta\gamma+ \\ \delta((1-q)\rho)\bar{\lambda_1}(p\eta\gamma+\sigma p_2)+(1-q)(1-\rho)k\bar{\lambda_1}pp_1\eta \\ +(1-q)(1-\rho)(1-p)\sigma\eta\bar{\lambda_1}] \end{aligned}$$

If the determinant of Jacobian matrix (18) above is positive and since there exist a unique endemic equilibrium of system (2) provided that $\Re_0 > 1$ as it stated in lemma 3 above,then by Routh-Hurwitz criteria, the endemic state $E_e = (S_1^*, S_2^*, C^*, I^*, R^*)$ is locally asymptotically stable [23]

3.6.3 Global stability of the endemic equilibrium point

We can verify the global stability of the endemic equilibrium E_e by constructing the Lyapunov function as in theorem 5 below.

Theorem 5.*If* $\Re_0 > 1$, *the endemic equilibrium* E_e *of system (2) is globally asymptotically stable.*

Proof. We apply [22] approach to prove global stability of E_e by defining Lyapunov function as

$$\sum_1^n \left(x_i - x_i^* - x_i^* ln\left(\frac{x_i}{x_i^*}\right)\right) \tag{19}$$

where x_i is a population in compartment i=1,...5 and x_i^* is the endemic equilibrium point. This is defined as

$$\begin{aligned} L(S_1^*, S_2^*, C^*, I^*, R^*) = \left(S_1 - S_1^* - S_1^* ln\left(\frac{S_1}{S_1^*}\right)\right) + \\ \left(S_2 - S_2^* - S_1^* ln\left(\frac{S_2}{S_2^*}\right)\right) + \left(C - C^* - C^* ln\left(\frac{C}{C^*}\right)\right) + \\ \left(I - I^* - I^* ln\left(\frac{I}{I^*}\right)\right) + \left(R - R^* - R^* ln\left(\frac{R}{R^*}\right)\right) \end{aligned}$$

The derivative of L is

$$\begin{aligned} \frac{dL}{dt} = \left(1 - \frac{S_1^*}{S_1}\right)\frac{dS_1}{dt} + \left(1 - \frac{S_2^*}{S_2}\right)\frac{dS_2}{dt} + \\ \left(1 - \frac{C^*}{C}\right)\frac{dC}{dt} + \left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} + \left(1 - \frac{R^*}{R}\right)\frac{dR}{dt} \end{aligned} \tag{20}$$

Next, we replace $\frac{dS_1}{dt}, \frac{dS_2}{dt}, \frac{dC}{dt}, \frac{dI}{dt}, \frac{dR}{dt}$ in (20) using system (2), to have

$$\begin{aligned} \frac{dL}{dt} = \left(1 - \frac{S_1^*}{S_1}\right)((1-\pi)b + \delta R - (1-q)\lambda_1 S_1 - \mu S_1) + \\ \left(1 - \frac{S_2^*}{S_2}\right)(\pi b - (1-q)k\lambda_1 S_2 - \mu S_2) + \\ \left(1 - \frac{C^*}{C}\right)((1-q)\rho\lambda_1 S_1 - (1-q)k\rho\lambda_1 S_2 - \\ (1-p)\eta I - p_1 C) + \left(1 - \frac{I^*}{I}\right)((1-q)(1-\rho)\lambda_1 S_1 + \\ (1-q)(1-\rho)k\lambda_1 S_2 + \gamma C - p_2 I) + \\ \left(1 - \frac{R^*}{R}\right)(\sigma C + p\eta I - (\mu+\delta)R) \end{aligned} \tag{21}$$

At endemic equilibrium EE from system (2), we have

$$\begin{aligned}(1-\pi)b &= -\delta R^* + (1-q)\lambda_1 S_1^* + \mu S_1^* \\ \pi b &= (1-q)k\lambda_1 S_2^* + \mu S_2^* \\ (\sigma+\gamma+\mu) &= \\ (1-q)\rho\lambda_1\frac{S_1^*}{C^*} + (1-q)k\rho\lambda_1\frac{S_2^*}{C^*} &+ (1-p)\eta\frac{I^*}{C^*} \\ (\alpha+\eta+\mu) &= \\ (1-q)(1-\rho)\lambda_1\frac{S_1^*}{I^*} + (1-q)(1-\rho)k\lambda_1\frac{S_2^*}{I^*} &+ \gamma\frac{C^*}{I^*} \\ (\mu+\delta) &= \sigma\frac{C^*}{R^*} + p\eta\frac{I^*}{R^*}\end{aligned} \tag{22}$$

Substituting (22) into (21), we get

$$\begin{aligned}\frac{dL}{dt} = \delta\left(R + R^*\frac{S_1^*}{S_1}\right) - \delta\left(R^* + R\frac{S_1^*}{S_1}\right) - \\ (1-q)\lambda_1\frac{(S_1-S_1^*)^2}{S_1} - \mu\frac{(S_1-S_1^*)^2}{S_1} - \\ (1-q)k\lambda_1\frac{(S_2-S_2^*)^2}{S_2} - \mu\frac{(S_2-S_2^*)^2}{S_2} + \\ (1-q)\rho\lambda_1(S_1+S_1^*) + (1-q)\rho\lambda_1 k(S_2+S_2^*) + \\ (1-p)\eta(I+I^*) - (1-q)\rho\lambda_1\left(C\frac{S_1^*}{C^*} + S_1\frac{C^*}{C}\right) \\ -(1-q)\rho\lambda_1 k\left(C\frac{S_2^*}{C^*} + S_2\frac{C^*}{C}\right) - \\ (1-q)\eta\left(C\frac{I*}{C^*} + I\frac{C^*}{C}\right) + \\ (1-q)(1-\rho)\lambda_1(S_1+S_1^*) + \\ (1-q)(1-\rho)k\lambda_1(S_1+S_1^*) + \gamma(C+C^*) - \\ (1-q)(1-\rho)\lambda_1\left(I\frac{S_1^*}{I^*} + S_1\frac{I^*}{I}\right) - \\ (1-q)(1-\rho)\lambda_1 k\left(I\frac{S_2^*}{I^*} + S_2\frac{I^*}{I}\right) - \\ \gamma\left(I\frac{C^*}{I^*} + C\frac{I^*}{I}\right) + \\ \sigma(C+C^*) + p\eta(I+I^*) - \\ \sigma\left(R\frac{C^*}{R^*} + C\frac{R^*}{R}\right) - p\eta\left(R\frac{I^*}{R^*} - I\frac{R^*}{R}\right)\end{aligned} \tag{23}$$

Thus collecting positive terms and negative terms together from Equation (23) we obtain

$$\frac{dL}{dt} = P - N$$

where

$$\begin{aligned}P = \delta\left(R + R^*\frac{S_1^*}{S_1}\right) + (1-q)\rho\lambda_1(S_1+S_1^*) + \\ (1-q)\rho\lambda_1 k(S_2+S_2^*) + (1-p)\eta(I+I^*) + \\ (1-q)(1-\rho)\lambda_1(S_1+S_1^*) + (1-q)(1-\rho)k\lambda_1(S_1+S_1^*) \\ +\gamma(C+C^*) + \sigma(C+C^*) + p\eta(I+I^*)\end{aligned}$$

$$\begin{aligned}N = \delta\left(R^* + R\frac{S_1^*}{S_1}\right) + (1-q)\lambda_1\frac{(S_1-S_1^*)^2}{S_1} + \mu\frac{(S_1-S_1^*)^2}{S_1} \\ +(1-q)k\lambda_1\frac{(S_2-S_2^*)^2}{S_2} + \mu\frac{(S_2-S_2^*)^2}{S_2} + \\ (1-q)\rho\lambda_1\left(C\frac{S_1^*}{C^*} + S_1\frac{C^*}{C}\right) + \\ (1-q)\rho\lambda_1 k\left(C\frac{S_2^*}{C^*} + S_2\frac{C^*}{C}\right) + (1-p)\eta\left(C\frac{I^*}{C^*} + I\frac{C^*}{C}\right) \\ +(1-q)(1-\rho)\lambda_1\left(I\frac{S_1^*}{I^*} + S_1\frac{I^*}{I}\right) \\ +(1-q)(1-\rho)k\lambda_1\left(I\frac{S_2^*}{I^*} + S_2\frac{I^*}{I}\right) + \\ \gamma\left(I\frac{C^*}{I^*} + C\frac{I^*}{I}\right) + \sigma\left(R\frac{C^*}{R^*} + C\frac{R^*}{R}\right) \\ +p\eta\left(R\frac{I^*}{R^*} - I\frac{R^*}{R}\right)\end{aligned}$$

Thus if $P < N$, then $\frac{dL}{dt} \leq 0$.

It is sure that $\frac{dL}{dt} = 0$ if and only if $S_1 = S_1^*, S_2 = S_2^*, C = C^*, I = I^*, R = R^*$. Thus, system (2) has a unique endemic equilibrium point E_e which is globally asymptotically stable if $\Re_0 > 1$.

Therefore, the largest compact invariant set in $(S_1^*, S_2^*, C^*, I^*, R^*) \in \Omega : \frac{dL}{dt} = 0$ is the singleton E_e, where E_e is the endemic equilibrium of the system (2). By LaSalle's invariant principle [19,23], it implies that E_e is globally asymptotically stable in Ω if $P < N$.

3.7 Sensitivity analysis of the model parameters

Under this subsection we identify the most influential parameters for the expansion as well as for eradication of pneumonia in the community. In addition to this to reduce the mortality and morbidity due to pneumonia we should have to focus on the parameters that have a great influence on the tread shoot number which is denoted by $\Re_0$ as discussed earlier. Sensitivity analysis is used to determine the sensitivity index which is a measure of the relative change in a state variable when a parameter changes. We compute the sensitivity indices of $\Re_0$ corresponding to the model parameters with the approach used by [24]. These indices show the importance of each individual parameter in the disease transmission dynamics and prevalence.

The sensitivity index of $\Re_0$ with respect to a parametersay X is given by:

$$\Lambda_X^{\Re_0} = \frac{\partial\Re_0}{\partial X}\frac{X}{\Re_0} \tag{24}$$

Where X stands for all parameters in the $\Re_0$. That is

$$\Re_0 = \frac{(1-q)(1+\pi(k-1))\beta\kappa}{p_1p_2 - (1-p)\eta\gamma}(\rho k_1 + (1-p)k_2)$$

For $X = \pi$

$$\Lambda_{\pi}^{\Re_0} = \frac{\partial \Re_0}{\partial \pi}\frac{\pi}{\Re_0} = \frac{\pi(k-1)}{1+\pi(k-1)}$$

For $X = \rho$

$$\Lambda_{\rho}^{\Re_0} = \frac{\partial \Re_0}{\partial \rho}\frac{\rho}{\Re_0} = \frac{\rho(k_1-k_2)}{\rho k_1+(1-\rho)k_2}$$

For $X = \kappa$

$$\Lambda_{\kappa}^{\Re_0} = \frac{\partial \Re_0}{\partial \kappa}\frac{\kappa}{\Re_0} = \frac{\kappa}{\kappa} = 1 > 0$$

For $X = q$

$$\Lambda_{q}^{\Re_0} = \frac{\partial \Re_0}{\partial q}\frac{q}{\Re_0} = -\frac{q}{1-q} < 0$$

For $X = \beta$

$$\Lambda_{\beta}^{\Re_0} = \frac{\partial \Re_0}{\partial \beta}\frac{\beta}{\Re_0} = \frac{\beta}{\beta} = 1 > 0$$

For $X = k$

$$\Lambda_{k}^{\Re_0} = \frac{\partial \Re_0}{\partial k}\frac{k}{\Re_0} = \frac{k\pi}{1+\pi(k-1)}$$

For $X = \omega$

$$\Lambda_{\omega}^{\Re_0} = \frac{\partial \Re_0}{\partial \omega}\frac{\omega}{\Re_0} = \omega\frac{\rho p_2+(1-p)(1-\rho)}{(\rho k_1+(1-\rho)k_2)}$$

For $X = p$

$$\Lambda_{p}^{\Re_0} = \frac{\partial \Re_0}{\partial p}\frac{p}{\Re_0} = -p\eta\frac{\omega(1-\rho)\Phi_2+\Phi_1\gamma}{\Phi_1\Phi_2}$$

For $X = \sigma$

$$\Lambda_{\sigma}^{\Re_0} = \frac{\partial \Re_0}{\partial \sigma}\frac{\sigma}{\Re_0} = \sigma\frac{(\omega\rho+(1-\rho))\Phi_2-p_2\Phi_1}{\Phi_1\Phi_2}$$

For $X = \gamma$

$$\Lambda_{\gamma}^{\Re_0} = \frac{\partial \Re_0}{\partial \gamma}\frac{\gamma}{\Re_0} = \gamma\frac{\Phi_2-\Phi_1(p_2-(1-p)\eta)}{\Phi_1\Phi_2}$$

For $X = \alpha$

$$\Lambda_{\alpha}^{\Re_0} = \frac{\partial \Re_0}{\partial \alpha}\frac{\alpha}{\Re_0} = \alpha\frac{\omega\rho\Phi_2-p_1\Phi_1}{\Phi_1\Phi_2}$$

For $X = \eta$

$$\Lambda_{\eta}^{\Re_0} = \frac{\partial \Re_0}{\partial \eta}\frac{\eta}{\Re_0} = \eta\frac{\omega\Phi_2-\Phi_1(p_1-(1-p)\gamma)}{\Phi_1\Phi_2}$$

For $X = \mu$

$$\Lambda_{\mu}^{\Re_0} = \frac{\partial \Re_0}{\partial \mu}\frac{\mu}{\Re_0} = \mu\frac{(\omega\rho+(1-\rho))\Phi_2-\Phi_1(p_1+p_2)}{\Phi_1\Phi_2}$$

Table 1: Model parameter values with their source

Parameters	Value	References
α	0.33	[19]
π	0.867	[16]
η	0.0238	[19]
β	1-10	[8]
μ	0.002	[19]
b	$N_0\mu$	[25]
σ	0.0116	Assumed
ρ	0.338	[1]
k	$0 < k \leq 1$	Assumed
q	$0 \leq q < 1$	Assumed
δ	0.2	[25]
ω	0.001124	[1]
κ	0.89-0.99	[1]
p	0.5	[22]
γ	0.01096	[1]

where
$\Phi_1 = (\rho k_1+(1-\rho)k_2)$ and $\Phi_2 = (p_1p_2-(1-p)\eta\gamma)$

From Table 2, we observe that the sensitivity indices of some model parameters are positive and the others are negative. The parameters with positive sensitivity indices show that there is a direct relationship of the parameters and the basic reproduction number of the model. Similarly, parameters with negative sensitivity indices shows inverse relationship is there between the model parameters and the basic reproduction number. In this case, increasing or decreasing the value of the model parameters will decrease or increase the basic reproduction number. For instances, q, π, and ρ have negative sign, which implies that they have negative effect on the basic reproduction number. On the other hand, the parameters κ, β have positive sign, which implies that they have positive impact on the basic reproduction number. practically, this shows increasing (or decreasing) these parameter values automatically increases (or decreases) $\Re_0$. Figure 2 shows the diagrammatic representation of sensitivity indices of basic model parameters with their relative contribution on pneumonia management. From Figure 2, we easily see the impact of each basic parameter on the basic reproduction number for instance κ and β has great impact and p has almost zero contribution on $\Re_0$.

4 Numerical Simulation

To show the theoretical results of the model numerical simulations are carried out by using different set of parameter value obtained from published articles as presented in Table 1. When the parameter values are not available in the literature, we assume it depending on the realistic value according to our purpose of goal. The system corresponding to the model is simulated using MATLABr2018a and MAPL18 programming language

Table 2: Sensitivity analysis of model parameter

parameters	sensitivity index
κ	1.0000
β	1.0000
k	0.765
ω	0.2448
γ	0.1070
p	-0.01594
η	-0.0845
σ	-0.1012
ρ	-0.2230
μ	-0.2772
π	-0.765
α	-0.9567
q	-1.0000

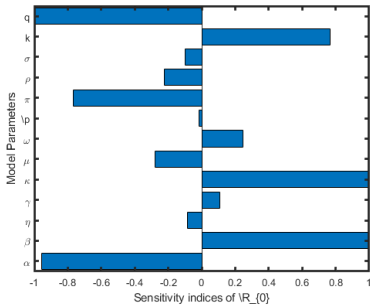
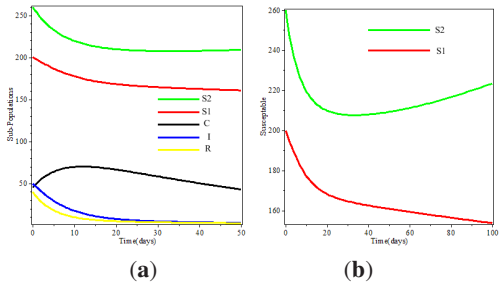


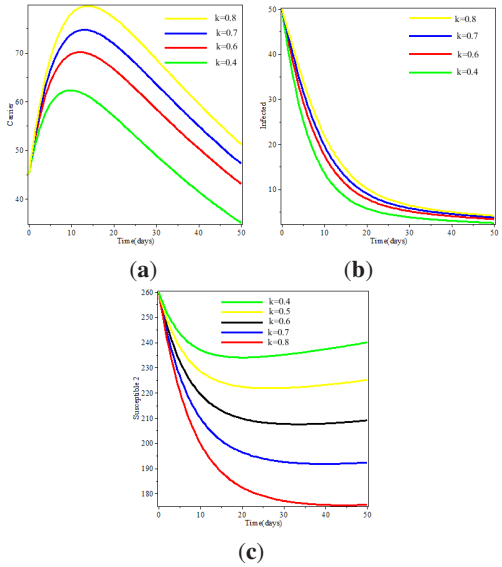
Fig. 2: Sensitivity indices of model parameter

and it identify which parameters influences the spread of pneumonia and how its influence can be managed.



(**a**) (**b**)

Fig. 3: (a) A population dynamics with time for DFE point and (b) Variation of the S_1 and S_2 under hygiene care and breastfeed interventions



(**a**) (**b**) (**c**)

Fig. 4: Impact of k on susceptible2,carrier and infected classes

5 Result and discussions

We investigate the impact of hygiene care rate,transmission rate and infection reduction of breastfeed susceptible on the spread or reduction of pneumonia diseases in the community.

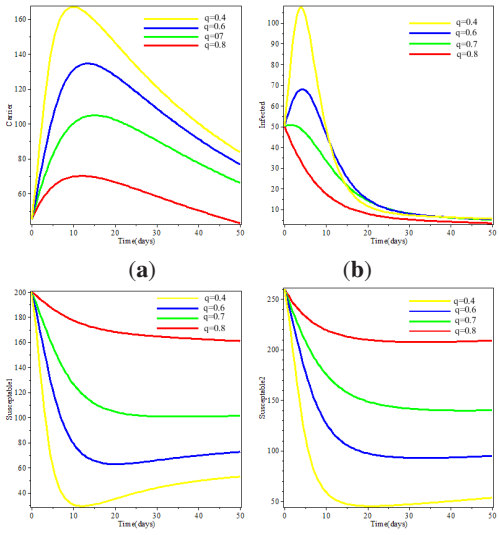
Figure 3(a) and (b) demonstrate respectively that the numerical solutions of the model found by MAPLE18 converge to disease free equilibrium (DFE) and S_1 is more susceptible than S_2. From Figure 3(b) we see that at the beginning S_2 decreases while after it increases due to the parameter value k.

To consider the effect of hygiene care and infection reduction due to breastfeed on the transmission dynamics of pneumonia, we plot the trajectory of carrier and infected class for different values of q and k see(Figure 4 and 5). Figure 4 (a) and (b) show that infection reduction of breastfeed susceptible children decrease while the number of infected and carrier population disease. This gurants that the locally asymptoticly stability of diseases free equilibrium. On the contrary, increasing the parameter k will decrease the number of breastfeed susceptible children see Figure 4(c).

As clearly seen from Figure 5 (a) and (b) when the hygiene care rate (q) increase then the two disease state variables will decrease. Similarly from Figure 5 (c) and (d) we conclude that q and the two susceptible classes have direct relation sheep. That is as hygiene care rate increases then S_1 and S_2 are also increases.

From Figure 6 we observe that the hygiene care and infection reduction rate due to breastfeed decreases the transmission rate of pneumonia. This implies that as the vale of β decreases then the diseases state variables will decrease.

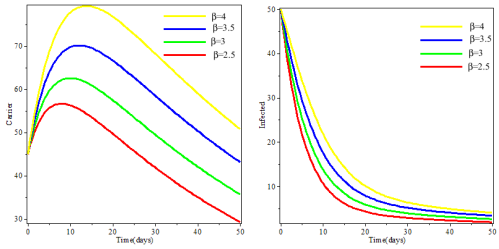
Both the Figures 7(a) and (b) evidently show that an increase in the susceptible1 population also increases the carrier or infected population and vies-verse. It can noticed from Figure 8(a) decreasing susceptible2 will increase carrier and the inverse is also true.



(**a**) (**b**)

(**c**) (**d**)

Fig. 5: Impact of q on the two susceptible,carriers and infected compartments

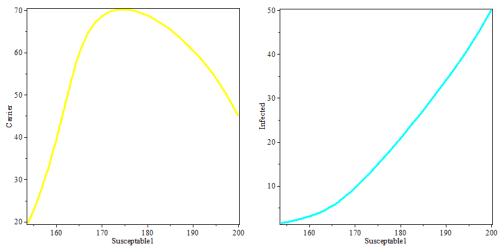


(**a**) (**b**)

Fig. 6: Impact of transmission rate on carriers and infections

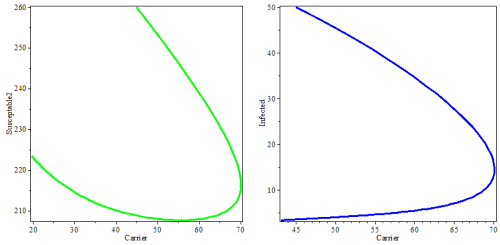
6 Conclusion

In this paper, we formulate a susceptible1, susceptible2, carrier, infected and recovered deterministic mathematical model with two kinds of controlling or eradicating the dynamics of pneumonia, that is the hygiene care efficacy and infection reduction due to effective breastfeed. The proposed model has two equilibrium points: diseases free and endemic equilibrium. The well-posedness of the model are verified by establishing existence and uniqueness, positivity and boundedness theorems. The basic reproduction number have been derived using next generation matrix and it shows that pneumonia will wiped out from the community if $\Re_0 < 1$ and persist in the society if $\Re_0 > 1$. The main finding of sensitivity analysis shows that the parameters q, β and κ have hight contribution in diseases control. A large scale of numerical simulations is performed using MATLABr2018a and MAPLE 18 to verify the theoretical work.



(**a**) (**b**)

Fig. 7: (a) The relation sheep between S_1 and C population and (b) The relation between S_1 and I population



(**a**) (**b**)

Fig. 8: (a) The relation sheep between S_2 and C population and (b) The graph of C verses I

Acknowledgement

I would like to thank all the references, editors and reviewers for their constructive comments, suggestions and questions, which have improved the manuscript.

References

[1] Kizito, Mohammed, and Julius Tumwiine. "A mathematical model of treatment and vaccination interventions of pneumococcal pneumonia infection dynamics." Journal of Applied Mathematics 2018 (2018).

[2] Sonego, Michela, Maria Chiara Pellegrin, Genevieve Becker, and Marzia Lazzerini. "Risk factors for mortality from acute lower respiratory infections (ALRI) in children under five years of age in low and middle-income countries: a systematic review and meta-analysis of observational studies." PloS one 10, no. 1 (2015): e0116380.

[3] Vivi Ninda Sutriana, Mei Neni Sitaresmi, and Abdul Wahab. Risk fac- tors for childhood pneumonia: a case-control study in a high prevalence area in indonesia. Clinical and experimental pediatrics, 64(11):588, (2021).

[4] Asad Ali, Sara Husain, Atif Riaz, and Huma Khawar. Status of in- troduction of pneumococcal conjugate vaccine in pakistan. Pediatric Infectious Disease, 8(2):6-66, (2016).

[5] Varinder Singh and Satinder Aneja. Pneumonia management in the developing world. Paediatric respiratory reviews, 12(1):52-59, (2011).

[6] Rory E Morty. World health day observances in november 2021: advo- cating for adult and pediatric pneumonia, preterm birth, and chronic obstructive pulmonary disease, L954-L957,(2021).

[7] Noha Fadl, Ayat Ashour, and Yasmine Yousry Muhammad. Pneumonia among under-
ve children in alexandria, egypt: a case-control study. Journal of the Egyptian Public Health Association, 95(1):1-7, (2020).
[8] Mansi Vijaybhai Dhami, Felix Akpojene Ogbo, Thierno MO Diallo, Bo- lajoko O Olusanya, Piwuna Christopher Goson, Kingsley Emwinyore Agho, Global Maternal, and Child Health Research Collaboration (Glo-MACH). Infant and young child feeding practices among adolescent mothers and associated factors in india. Nutrients, 13(7):2376, (2021).
[9] Laura M Lamberti, Irena Zakarija-Grković, Christa L Fischer Walker, Evropi Theodoratou, Harish Nair, Harry Campbell, and Robert E Black.Breastfeeding for reducing the risk of pneumonia morbidity and mortality in children under two: a systematic literature review and meta analysis. BMC public health, 13(3):1-8, (2013).
[10] Tatiana Mota Xavier de Meneses, Maria Inêes Couto de Oliveira, and Cristiano Siqueira Boccolini. Prevalence and factors associated with breast milk donation in banks that receive human milk in primary health care units. Jornal de pediatria, 93:382-388, (2017).
[11] Alison McFadden, Lindsay Siebelt, Joyce L Marshall, Anna Gavine, Lisa-Christine Girard, Andrew Symon, and Stephen MacGillivray. Counselling interventions to enable women to initiate and continue breastfeeding: a systematic review and meta-analysis. International breastfeeding journal, 14(1):1-19, (2019).
[12] Laura M Lamberti, Christa L Fischer Walker, Adi Noiman, Cesar Victora, and Robert E Black. Breastfeeding and the risk for diarrhea morbidity and mortality. BMC public health, 11(3):1-12, (2011).
[13] TKP Nguyen, TH Tran, CL Roberts, GJ Fox, SM Graham, and BJ Marais. Risk factors for child pneumonia-focus on the western pacific region. Paediatric respiratory reviews, 21:95-101, (2017).
[14] Yuanyuan Yu, Youjiang Li, Yingying Hu, Bin Li, and Jian Xu. Breastfed 13 month-old infant of a mother with covid-19 pneumonia: a case report. International breastfeeding journal, 15(1):1-6, (2020).
[15] Kermack, W.O. and McKendrick, A.G.. A contribution to the mathematical theory of epidemics. Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character, 115(772), pp.700-721,(1927)
[16] Dominic Otoo, Patrick Opoku, Sebil Charles, and Asekiya Prince Kings- ley. Deterministic epidemic model for (svcsycasyir) pneumonia dynamics, with vaccination and temporal immunity. Infectious Disease Model ling, 5:42-60, (2020).
[17] ML Diagne, H Rwezaura, SY Tchoumi, and JM Tchuenche. A mathematical model of covid-19 with vaccination and treatment. Computational and Mathematical Methods in Medicine, 2021, (2021).
[18] A Lochen and RM Anderson. Dynamic transmission models and economic evaluations of pneumococcal conjugate vaccines: a quality appraisal and limitations. Clinical Microbiology and Infection, 26(1):60-70,(2020).
[19] Getachew Teshome Tilahun, Oluwole Daniel Makinde, and David Malonza. Modelling and optimal control of pneumonia disease with cost-effective strategies. Journal of Biological Dynamics, 11(sup2):400-426,(2017).
[20] Viriyapong, R., and Kamyod, S.. The effect of children under five-year old with hygiene care and breastfeeding on dynamics of pneumonia model for Thailand. KKU Science Journal, 47(1), 154-166,(2019).
[21] Kassahun Getnet Mekonen, Tatek Getachew Habtemicheal, and Shiferaw Feyissa Balcha. Modeling the effect of contaminated objects for the transmission dynamics of covid-19 pandemic with self protection behavior changes. Results in Applied Mathematics, 9:100134, (2021).
[22] Mary C Swai, Nyimvua Shaban, and Theresia Marijani. Optimal control in two strain pneumonia transmission dynamics. Journal of Applied Mathematics, 2021, (2021).
[23] Halson O Nyaberi and Jane S Musaili. Mathematical modeling of the impact of treatment on the dynamics of typhoid. Journal of the Egyptian Mathematical Society, 29(1):1-11, (2021).
[24] Temesgen Duressa Keno, Oluwole Daniel Makinde, and Legesse Lemecha Obsu. Impact of temperature variability on sirs malaria model. Journal of Biological Systems, 29(03):773-798, (2021).
[25] Otieno, M. J. O. A. J., and Paul, O. . Mathematical model for pneumonia dynamics with carriers, (2013). http://dx.doi.org/10.1016/j.plrev.2012.09.002.

Fekadu Mosisa Legesse is one of a Ph.D. Scholar at Wollega University, Department of Mathematics, Nekemte, Ethiopia. He is a lecturer at Wollega University. Fekadu Mosisa received the B.Sc. degree in Applied Mathematics from University of Gonder and Masters of Science Degree in Mathematics(Differential Equations) from Haramaya University. Currently he is working his PhD research in Mathematical Modeling at Wollega University. His research interest are in the areas of Mathematical Model and Optimal control Theory and their application on Mathematical Epidemiology.

Purnachandra Rao Koya is Professor of Mathematics at Wollega University Department of Mathematics Nekemte, Ethiopia. So far he has published more than 80 research articles in reputable journals. Dr. Koya Purnachandra Rao has been working as a Professor at the Department of Mathematics, Wollega University, since August 01, 2018. Earlier, Dr. K.P. Rao had served various prestigious institutions in India including Saurashtra University, Bapatla Engineering College, Chirala Engineering College and Mother Teresa institute of Science Technology, and

also in Ethiopia he served Hawassa University. His research areas of interest include Cosmological Models, Epidemiological Models, Multi Specious Systems, Fluid Dynamics, Traffic Models, and Water Pollution Models.

Temesgen Duressa Keno is an Assistant Professor of Applied Mathematics at Wallaga University, Nekemte, Ethiopia. He received his PhD degree in "Optimization" at Adama Science and Technology University. Dr. Temesgen Duressa research work has contributed immensely to many scholars in the area of applied mathematics research and postgraduate training. He has taught, supervised and mentored several students at undergraduate and postgraduate levels. His research work covers: Optimization Theory, Optimal control Theory and applications, Dynamical Systems, Epidemiological Modeling and applications. He has published more than 7 research articles in reputed international journals.