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Mathematical Model for Pneumonia Dynamics with Carriers

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Abstract. There are major advances which have been made to understand the epidemiology of infectious diseases. However, more than 2 million children in the developing countries still die from pneumonia each year. The efforts to promptly detect, effectively treat and control the spread of pneumonia is possible if its dynamics is understood. In this paper, we develop a mathematical model for pneumonia among children under five years of age. The model is analyzed using the theory of ordinary differential equations and dynamical systems. We derive the basic reproduction number, R_0, analyze the stability of equilibrium points and bifurcation analysis. The results of the analysis shows that there exist a locally stable disease free equilibrium point, E^f when $R_0 < 1$ and a unique endemic equilibrium, E^e when $R_0 > 1$.The analysis also shows that there is a possibility of a forward bifurcation.

Keywords: Pneumonia Model, Basic reproduction number, forward bifurcation, Stability, Carriers

1 Introduction

Pneumonia is a high-incidence respiratory disease characterized by an inflammatory condition of the lungs and is caused by micro-organisms namely: bacteria, fungi, parasites and viruses. Among the four micro-organisms potential

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in causing pneumonia, bacteria is reported to be the leading cause[27],[18] especially *Streptococcus Pneumoniae* [9],[21],[23]. The bacteria enter the lungs, and settle in the alveoli and passages of the lung where they rapidly grow and multiply in number. The area of the lung that is invaded then becomes filled with fluid and pus as the body attempts to fight off the infection [24]. This makes breathing difficult, painful and limits the intake of oxygen.

Most cases of pneumonia are as a result of inhaling small droplets of coughs or sneezes containing the bacteria. These droplets gets into the air when an infected person coughs or sneezes [24],[27]. The bacteria can also be carried in the mouth or flora of nasopharynx of a healthy person without causing any harm [21],[24],[27]. Such people are referred to as carriers. For carriers, the bacteria can find its way to the lungs and invade to cause the infection [4],[24]. This is possible when the immunity of the individual is lowered.

There is limited information on the transmission patterns of the pneumococcal disease in the developing world [9], however, it is pointed out that the risk factors associated with the spread of the disease includes: malnutrition, lack of exclusive breastfeeding, indoor pollution, antecedent viral infection amongst others [9],[23].

Despite the increasing focus on the Millennium Development Goal 4 of United Nation-MDG [28] "to reduce child mortality", almost 1.9 million children still die from pneumonia each year in the developing countries, accounting for 20 % of deaths globally [1]. In Kenya, pneumonia contributes up to 16 % of child mortality [3]. It is evident that the management of the disease is challenging due to overlap of its symptoms with that of malaria hence a possibility of mistreatment with antimalarial drugs [12]. Deaths due to pneumonia can occur within three days of illness and any delays in proper treatment may not save life [17].

Therefore to realize the Millennium Development Goal 4 (MDG 4), research should be done to promptly diagnose, effectively treat and deduce other prevention strategies for pneumonia. For this to be achieved, accurate projections on possibility of epidemic or endemic and strategies to put up control measures is required. Mathematical models integrated in epidemiological research are powerful tools in studying the dynamics of diseases and to find threshold parameters necessary for controlling the disease. In this paper, we therefore develop and analyze a mathematical models for pneumonia dynamics in children.

2   Derivation of the Model

The transmission dynamics of pneumonia in the population under study is considered between four compartments based on the disease status, that is: Susceptible, Carriers, Infectious and Recovered. At time t, the total popula-

tion size (N) is divided into: susceptible (S), infected (I), carriers (C) and recovered (R) such that:

$$N = S + C + I + R \tag{1}$$

The per capita recruitment rate into the susceptible population is denoted ν. We assume that the infected immigrants are not included because they are not able to travel. New infection can be due to effective contact with either a carriers or a symptomatically infected individual, where the force of infection of susceptibles is denoted by λ. A newly infected individuals joins carrier class with a probability of ρ or symptomatically infected class with a probability of $(1 - \rho)$. Carriers can change their status to show symptoms (infected) [16] at the rate π. Infected individuals recovers at the rate η. A proportion q of the recovered individuals clear all the bacteria from the body and gain temporal immunity while $(1 - q)$ of them will still carry the bacteria[19],[25]. The carriers can also recover to gain temporal immunity at the rate β. In this model, the temporal immunity is a result of all possible ways that may lead to recovery from the disease. Studies in [22],[8],[7], show that there is a possibility of reinfection at the rate δ. There is a natural death rate of μ and a disease induced death rate of α. We define the force of infection as:

$$\lambda = \psi \left(\frac{I + \varepsilon C}{N} \right) : \psi = \kappa \mathcal{P} \tag{2}$$

Where κ is the rate of contact and $\mathcal{P}$ be the probability that a contact is efficient to cause infection. Combining all the definitions and assumptions, the model for the transmission dynamics of pneumonia is given by the following system of differential equations:

$$\left. \begin{array}{l} \frac{dS(t)}{dt} = \nu + \delta R(t) - (\lambda + \mu)S(t) \\ \frac{dI(t)}{dt} = (1 - \rho)\lambda S(t) + \pi C(t) - (\mu + \alpha + \eta)I(t) \\ \frac{dC(t)}{dt} = \rho(\lambda)S(t) + (1 - q)\eta I(t) - (\mu + \pi + \beta)C(t) \\ \frac{dR(t)}{dt} = q\eta I(t) + \beta C(t) - (\mu + \delta)R(t) \end{array} \right\} \tag{3}$$

3 Positivity and boundedness of solutions

We can show from Model (3) that the state variables are non-negative and the solutions remain positive for all time $t \geq 0$. Here the parameters in the model are assumed to be positive. We also show that the feasible solutions are bounded in a region: $\Phi = \{(S, I, C, R) \in \mathcal{R}_+^4 : N(t) \leq \frac{\nu}{\mu}\}$

Lemma 3.1 *Let the initial values of the parameters be $\{S(0) \geq 0, I(0) \geq 0, C(0) \geq 0, R(0) = 0 \text{ and } N(0) \geq 0\} \in \Phi$, then solution set $\{S(t), I(t), C(t), R(t), N(t)\}$ is positive for all $t \geq 0$*

Proof First, consider the first equation in (3)

$$\frac{dS}{dt} = \nu + \delta R - \lambda S - \mu S$$

We have that;

$$\frac{dS}{dt} \geq -(\lambda + \mu)S$$
$$\int \frac{1}{S}dS \geq \int -(\lambda + \mu)dt$$
$$S \geq S_0 e^{-(\lambda + \mu)t} \geq 0$$
$$\text{Hence, } S \geq 0$$

Next, we consider the second equation in (3)

$$\frac{dI}{dt} = (1 - \rho)\lambda S + \pi C - (\mu + \alpha + \eta)I$$
$$\frac{dI}{dt} \geq -(\mu + \alpha + \eta)I$$
$$\int \frac{1}{I}dI \geq \int -(\mu + \alpha + \eta)dt$$
$$I \geq I_0 e^{-(\mu + \alpha + \eta)t}$$
$$\text{Hence, } I \geq 0$$

We can proceed in a similar way to prove the positivity of C, R and N .

Lemma 3.2 *The solutions for the System 1 are contained and remain in the region Φ for all time $t \geq 0$*

Proof Consider Equation (1). Taking the derivatives with respect to time t of (1) and substituting onto it the set of equations in (3), we have,

$$\frac{dN(t)}{dt} = \nu - \alpha I - \mu N$$
$$\Rightarrow \frac{dN}{dt} \leq \nu - \mu N$$
$$\Rightarrow N \leq \frac{\nu}{\mu} + (N_0 - \frac{\nu}{\mu})e^{-\mu t})$$

Where $N_0 = $ is initial population size.
Thus,

$$\lim_{t \to \infty} N(t) \leq \frac{\nu}{\mu}$$

Using this result together with Lemma 3.1 and equation 1, we have that $0 \leq N(t) \leq \frac{\nu}{\mu}$ which implies that N and all other variable (S,I,C and R) is bounded and all the solutions starting in Φ approach, enter or stay in Φ.

4   Analysis of the Model

We analyze the model for pneumonia transmission based on the following sub sections to determine the basic reproduction number and other threshold parameters for pneumonia dynamics.

4.1   Stability analysis of the disease-free equilibrium (DFE).

The DFE of model (3) is obtained by equating the right-hand sides of the equations in the model to zero and it describes the model in absence of disease or infection. Here we define carrier and infected classes as diseased classes, DFE denoted by $(E^f = (S^f, I^f, C^f, R^f)$ is then given by; $E^f = \left(\frac{\nu}{\mu}, 0, 0, 0\right)$

Theorem 4.1 *There is a unique DFE (E^f) for the model represented by the system of equations in (3)*

Proof This lemma is proven by substituting the E^f into the system of Equations (3). The results shows all derivatives equal to zero, hence DFE is an equilibrium point.

To establish the linear stability of E^f, we use the next-generation operator approach [29] on the system (3) to compute the basic reproduction number (R_0). Using the notation of the matrices F and V as in [29], we have,

$$\mathbf{F} = \begin{pmatrix} (1-\rho)\psi & (1-\rho)\psi\varepsilon \\ \rho\psi & \rho\psi\varepsilon \end{pmatrix} \text{ and } \mathbf{V} = \begin{pmatrix} h_1 & -\pi \\ -(1-q)\eta & h_2 \end{pmatrix}$$

where, $h_1 = \mu + \alpha + \eta$ and $h_2 = \mu + \pi + \beta$ The eigenvalues for the matrix FV^{-1} are

$$0, \psi\left(\frac{\rho[\varepsilon h_1 + \pi] + (1-\rho)[h_2 + (1-q)\varepsilon\eta]}{h_1 h_2 - (1-q)\pi\eta}\right)$$

Thus from Theorem 2 of [29] we have

$$R_0 = \psi\left(\frac{\rho[\varepsilon h_1 + \pi] + (1-\rho)[h_2 + (1-q)\varepsilon\eta]}{h_1 h_2 - (1-q)\pi\eta}\right) \tag{4}$$

Lemma 4.2 *The the disease-free equilibrium (E^f) of (3) is locally asymptotically stable whenever $R_0 < 1$ and unstable when ($R_0 > 1$.*

Proof Consider the jacobian matrix for the Model (3)at E^f is given as

$$\mathcal{J}(E^f) = \begin{pmatrix} -\mu & -\psi & -\psi\varepsilon & \delta \\ 0 & -h_1 & \pi & 0 \\ 0 & (1-q)\eta & -h_2 & 0 \\ 0 & q\eta & \beta & -(\mu+\delta) \end{pmatrix} \text{ and}$$

$$\left.\begin{array}{l} Trace\left[\mathcal{J}(E^f)\right] = -(2\mu + \delta + h_1 + h_2) < 0 \\ Det\left[\mathcal{J}(E^f)\right] = \mu(\delta + \mu)[h_1 h_2 - (1-q)\pi\eta] > 0 \end{array}\right\} \tag{5}$$

Since the parameters μ, δ, h_1 and h_2 are all positive, then $-(2\mu+\delta+h_1+h_2) < 0$. Therefore $Trace\left[\mathcal{J}(E^f)\right] < 0$. On the other hand R_0 can never be negative and numerator $\{\rho[\varepsilon h_1+\pi]+(1-\rho)[h_2+(1-q)\varepsilon\eta]\}$ is positive, the denominator must also be positive i.e. $h_1 h_2 - (1-q)\pi\eta > 0$. Thin implies that $Det\left[\mathcal{J}(E^f)\right] > 0$, since $\mu(\delta + \mu) > 0$ and $[h_1 h_2 - (1-q)\pi\eta] > 0$.
Thus

$$R_0 = \psi \left(\frac{\rho[\varepsilon h_1 + \pi] + (1-\rho)[h_2 + (1-q)\varepsilon\eta]}{h_1 h_2 - (1-q)\pi\eta} \right) < 1$$

The solutions in (5) implies that E^0 is locally asymptotically stable whenever $R_0 < 1$

4.2   Stability of the Endemic equilibrium (EE) and Bifurcation analysis)

The endemic equilibrium is denoted by E^e and defined as a steady state solutions for the Model (3). This can occur when there is a persistence of the disease. Hence $E^e = \{S^e, I^e, C^e, R^e\}$ can be expressed as shown below.

$$S^e = \frac{N}{R_0}$$
$$C^e = \frac{(\mu + \delta)\left((1 - \rho)(1 - q)\eta + h_1\rho\right)(R_0 - 1)\nu}{R_0\left((\mu + \delta)(h_2 h_1 - (1 - q)\pi\eta) - \delta\left(\rho(\eta\pi q + h_1\beta) + (1 - \rho)(\eta q h_2 + (1 - q)\eta\beta)\right)\right)}$$
$$I^e = \frac{(\delta + \mu)(\pi\rho + (1 - \rho)h_2)(R_0 - 1)\nu}{R_0\left((\delta + \mu)(h_2 h_1 - (1 - q)\pi\eta) - \delta\left(\rho(\pi q\eta + h_1\beta) + (1 - \rho)(\eta q h_2 + (1 - q)\eta\beta)\right)\right)}$$
$$R^e = \frac{(\rho(\eta\pi q + h_1\beta) + (-\rho + 1)(h_2 q\eta + (1 - q)\eta\beta))(R_0 - 1)\nu}{R_0\left((\delta + \mu)(h_2 h_1 - (1 - q)\pi\eta) - \delta\left(\rho(\pi q\eta + h_1\beta) + (1 - \rho)(\eta q h_2 + (1 - q)\eta\beta)\right)\right)}$$

Lemma 4.3 *For $R_0 > 1$ a unique endemic equilibrium point E^e exist and no endemic equilibrium otherwise.*

Proof For the disease to be endemic, then $\frac{dI}{dt} > 0$ and $\frac{dC}{dt} > 0$, that is,

$$\begin{aligned}(1 - \rho)\psi\frac{S}{N}(I + \varepsilon C) + \pi C - h_1 I > 0 \\ \rho\psi\frac{S}{N}(I + \varepsilon C) + (1 - q)\eta I - h_2 C > 0\end{aligned} \tag{6}$$

From the first inequality of 6 we have

$$h_1 I < (1 - \rho)\psi\frac{S}{N}(I + \varepsilon C) + \pi C$$

using the fact $\frac{S}{N} \leq 1$

$$I < \frac{(1 - \rho)\psi I + (1 - \rho)\psi\varepsilon C + \pi C}{h_1} \tag{7}$$

and from the first inequality of 6 we have

$$C < \frac{\rho\psi I + (1 - q)\eta I}{h_2 - \rho\psi\varepsilon} \tag{8}$$

substituting 8 into 7, we have

$$
\begin{aligned}
I &< \frac{(1-\rho)\psi I + [(1-\rho)\psi\varepsilon + \pi]\left[\dfrac{\rho\psi I + (1-q)\eta I}{h_2 - \rho\psi\varepsilon}\right]}{h_1} \\
1 &< \frac{(1-\rho)\psi I + [(1-\rho)\psi\varepsilon + \pi][\rho\psi I + (1-q)\eta I]}{h_1 h_2 - h_1 \rho\psi\varepsilon} \\
h_1 h_2 - h_1 \rho\psi\varepsilon &< (1-\rho)\psi h_2 + \rho\psi\pi + (1-\rho)\psi\varepsilon(1-q)\eta + (1-q)\eta\pi
\end{aligned}
$$

$$
1 < \frac{\psi[\rho(h_1\varepsilon + \pi) + (1-\rho)(h_2 + (1-q)\varepsilon\eta)]}{h_1 h_2 - (1-q)\eta\pi} = R_0
$$
(9)

Thus a unique endemic equilibrium exist when $R_0 > 1$.

4.2.1 Local Stability analysis of the Endemic Equilibrium

We study the local stability of the endemic equilibrium by applying the Routh-Hurwitz criterion [15].

Theorem 4.4 *If $R_0 > 1$ then the endemic equilibrium E^e of system 3 is locally asymptotically stable in $\mathcal{G}$*

Proof Consider the Jacobian matrix at endemic equilibrium denoted by J_{E^e}

$$
J_{E^e} = \begin{bmatrix}
-\overline{\lambda} - \mu & 0 & 0 & \delta \\
(1-\rho)\overline{\lambda} & -h_1 & \pi & 0 \\
\rho\overline{\lambda} & (1-q)\eta & -h_2 & 0 \\
0 & q\eta & \beta & -\mu - \delta
\end{bmatrix}
$$

Where $\overline{\lambda}$ is defined as the force of infection at endemic equilibrium. We obtain a characteristic equation $P(\lambda) = |\lambda I - J_{E^e}|$ where I is a 4×4 unit matrix. So that the characteristic equation becomes, $P(\lambda) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4$

Hence from Routh-Hurwitz criterion, we have the matrix

$$
\begin{bmatrix}
1 & a_2 & a_4 & \lambda^4 \\
a_1 & a_3 & 0 & \lambda^3 \\
a_2 - \dfrac{a_3}{a_1} & a_4 & 0 & \lambda^2 \\
a_3 - \dfrac{a_1 a_4}{\left(a_2 - \frac{a_3}{a_1}\right)} & 0 & 0 & \lambda \\
a_4 & 0 & 0 & 1
\end{bmatrix}
$$

Where,

$a_1 = 2\mu + \delta + h_2 + h_1 + \overline{\lambda}$

$a_2 = 2h_2\mu + 2h_1\mu + \mu\overline{\lambda} + \mu^2 + h_2\delta + h_1\delta + \delta\overline{\lambda} + \delta\mu - \eta\pi + \eta\pi q + h_2 h_1 + h_2\overline{\lambda} + h_1\overline{\lambda}$

$a_3 = (\mu + \delta)(h_1 + h_2)(\mu + \overline{\lambda}) + (h_2 h_1 - (1-q)\eta\pi)(\overline{\lambda} + \delta + 2\mu) - \overline{\lambda}\delta((1-\rho)q\eta + \beta\rho)$

$a_4 = (1-\rho)(q\pi - h_2 q - (1-q)\beta)\eta\overline{\lambda}\delta - (\mu + \overline{\lambda})(\mu + \delta)(\eta\pi - h_2 h_1)$
$\quad\quad - \beta\rho\overline{\lambda}\delta h_1 + \eta\pi\mu q(\mu + \overline{\lambda} + \delta)$

According the the Routh-Hurwitz criterion, For $R_0 > 0$, the endemic equilibrium (E^e) is locally asymptotically stable if $a_1 > 0, a_2 - \frac{a_3}{a_1} > 0, a_3 - \frac{a_1 a_4}{\left(a_2 - \frac{a_3}{a_1}\right)} > 0$

and $a_4 > 0$.

4.2.2 Bifurcation analysis

A bifurcation is a qualitative change in the nature of the solution trajectories due to a parameter change. The point at which this change take place is called a bifurcation point. At the bifurcation point, a number of equilibrium points, or their stability properties, or both, change. When $R_0 < 1$, the infectious disease will not invade the population unless otherwise. We prove using Center Manifold theorem the possibility of bifurcation at $R_0 = 1$.

Let $S = x_1, I = x_2, C = x_3$ and $R = x_4$, so that $N = x_1 + x_2 + x_3 + x_4$, then using (2) and (3) is re-written in the form:

$$\left.\begin{array}{l}
\frac{dx_1}{dt} = f_1 = \nu + \delta x_4 - \psi\frac{x_2}{x_1+x_2+x_3+x_4}x_1 - \psi\varepsilon\frac{x_3}{x_1+x_2+x_3+x_4}x_1 - \mu x_1 \\[2mm]
\frac{dx_2}{dt} = f_2 = (1-\rho)\psi\frac{x_2}{x_1+x_2+x_3+x_4}x_1 + (1-\rho)\psi\varepsilon\frac{x_3}{x_1+x_2+x_3+x_4}x_1 + \pi x_3 - h_1 x_2 \\[2mm]
\frac{dx_3}{dt} = f_3 = \rho\psi\frac{x_2}{x_1+x_2+x_3+x_4}x_1 + \rho\psi\varepsilon\frac{x_3}{x_1+x_2+x_3+x_4}x_1 + (1-q)\eta x_2 - h_2 x_3 \\[2mm]
\frac{dx_4}{dt} = f_4 = q\eta x_2 + \beta x_3 - (\mu + \delta)x_4
\end{array}\right\} \tag{10}$$

Suppose that we choose ψ_c as a bifurcation parameter. Then by using (4), we solve ψ_c at $R_0 = 1$ as:

$$\psi_c = \frac{\mu^2 + \mu\alpha + \mu\eta + \Pi\mu + \Pi\alpha + \beta\mu + \beta\alpha + \beta\eta + \eta q\Pi}{\rho\epsilon\mu + \rho\epsilon\alpha + \mu + \Pi + \beta + \epsilon\eta - \epsilon\eta q - \rho\mu - \rho\beta + \rho\epsilon\eta q} \tag{11}$$

The liberalization matrix of (10) at a disease free Equilibrium (E^f) corresponding to $\psi = \psi_c$ is given by:

$$\mathcal{J}(E^f)\mid_{\psi=\psi_c} = J_{\psi_c} = \begin{pmatrix} -\mu & -\psi_c & -\psi_c\varepsilon & \delta \\ 0 & -h_1 & \pi & 0 \\ 0 & (1-q)\eta & -h_2 & 0 \\ 0 & q\eta & \beta & -(\mu+\delta) \end{pmatrix}$$

Zero is a simple eigenvalue of J_{ψ_c} if $h_1 = \frac{\eta\pi(1-q)}{h_2}$. A right eigenvector (w) of J_{ψ_c} associated with the zero eigenvalues is given by $w = (w_1, w_2, w_3, w_4)^T$ where

$$w_1 = \frac{w_3(\eta\,\delta\,(qh_2+(1-q)\beta)-\psi\,(\mu+\delta)(h_2+(1-q)\epsilon\,\eta))}{\eta\,(1-q)(\mu+\delta)\mu}$$
$$w_2 = \frac{w_3 h_2}{\eta\,(1-q)}$$
$$w_3 = w_3$$
$$w_4 = \frac{w_3(qh_2+(1-q)\beta)}{(1-q)(\mu+\delta)}$$

and a left eigenvector (v) of J_{ψ_c} corresponding to the zero eigenvalues is given by $v = (v_1, v_2, v_3, v_4)^T$ where

$$v_1 = 0$$
$$v_2 = \frac{v_3 h_2}{\pi}$$
$$v_3 = v_3$$
$$v_4 = 0$$

We now reproduce the theorem stated by Castillo-Chavez and Song [2] .

Theorem 4.5 *[2]. Consider the following general system of ordinary differential equations with parameter ϕ*

$$\frac{dx}{dt} = f(x,\phi), f : R^n \times R \text{ and } f \in C^2(R^n \times R)$$

where 0 is an equilibrium point of the system (that is f(0, ϕ) $\equiv$ 0 for all ϕ) and assume:

1. *$A = D_x f(0,0) = (\frac{\partial f_i}{\partial x_j}(0;0))$ is the linearization matrix of the system around the equilibrium point 0 with ϕ evaluated at 0;*

2. *Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts*

3. *Matrix A has a right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue.*

Let f_k be the kth component of f and

$$a = \sum_{k,i,j=0}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) \tag{12}$$

$$b = \sum_{k,i=0}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0) \tag{13}$$

Then the local dynamics of system 1 around the x=0 are totally determined by a and b. Particularly,

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

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

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

4. $a < 0, b > 0$, when ϕ changes from negative to positive, x=0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes locally asymptotically stable*

The algebraic calculation from theorem 4.5 are shown in the working below.

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_2} = -2\frac{(1-\rho)\psi}{x_1}, \qquad \frac{\partial^2 f_3}{\partial x_2 \partial x_2} = -2\frac{\rho\psi}{x_1}$$

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_3} = (1-\rho)\psi x_1 \left(-\frac{1}{x_1^2} - \frac{\epsilon}{x_1^2}\right), \qquad \frac{\partial^2 f_3}{\partial x_2 \partial x_3} = \rho\psi x_1 \left(-\frac{1}{x_1^2} - \frac{\epsilon}{x_1^2}\right)$$

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_3} = -\frac{(1-\rho)\psi}{x_1}, \qquad \frac{\partial^2 f_3}{\partial x_2 \partial x_3} = -\frac{\rho\psi}{x_1}$$

$$\frac{\partial^2 f_2}{\partial x_3 \partial x_3} = -2\frac{(1-\rho)\psi \epsilon}{x_1}, \qquad \frac{\partial^2 f_3}{\partial x_3 \partial x_3} = -2\frac{\rho\psi \epsilon}{x_1}$$

$$\frac{\partial^2 f_2}{\partial x_3 \partial x_4} = -\frac{(1-\rho)\psi \epsilon}{x_1}, \qquad \frac{\partial^2 f_3}{\partial x_3 \partial x_4} = -\frac{\rho\psi \epsilon}{x_1}$$

$$\frac{\partial^2 f_2}{\partial x_2 \psi_c} = 1 - \rho, \qquad \frac{\partial^2 f_3}{\partial x_2 \psi_c} = \rho$$

$$\frac{\partial^2 f_2}{\partial x_3 \psi_c} = (1-\rho)\epsilon, \qquad \frac{\partial^2 f_3}{\partial x_3 \psi_c} = \rho\epsilon$$

Note: $\frac{\partial^2 f_k}{\partial x_i \partial x_j} = \frac{\partial^2 f_k}{\partial x_j \partial x_i}$

The rest of the second derivatives that are in (12) and (13) are all zero. Hence,

$$a = \left(-2\frac{v_3 w_3^2 \psi}{\pi x_1}\right)(\rho\pi + (1-p)h_2)\left(\frac{(h_2+(1-q)\epsilon\,\eta)(h_2\eta\,q+(1-q)\eta\,\beta+(\mu+\delta)(h_2+(1-q)\eta))}{\eta^2(-1+q)^2(\mu+\delta)}\right)$$

$$a < 0$$

(14)

$$b = \frac{v_3 h_2{}^2 w_3 (1-\rho)}{\pi\,\eta\,(1-q)} + \frac{v_3 h_2 w_3((1-\rho)\epsilon)}{\pi} + \frac{v_3 w_3 h_2 \rho}{(1-q)\eta} + v_3 w_3 (\rho\,\epsilon)$$

$$b > 0$$

(15)

Using the results in Theorem 4.5, the results in (14) and (15) indicates that there is a forward bifurcation at $\psi = \psi_c$ and there exist at least one stable endemic equilibrium when $R_0. > 1$

5   Numerical Simulation

To observe the dynamics of pneumonia model over time, numerical simulations are done using MAPLE 14.0. The parameters in table 1 that will be used in simulation is based on the data of children under five year of age. Some values assigned to the parameters have been derived from epidemiological literature and WHO database while other parameters have been allowed to vary within the possible intervals.

Using the parameter values, the numerical simulations show that a transcritical (forward) bifurcation is likely to occur at $\psi = \psi_c = 0.47, (R_0 = 1)$ where there is only one stable equilibrium point if $R_0 < 1$ (disease-free equilibrium) and a low endemicity when R_0 is slightly above one (See Figure 1 (a) and (b)). This is important to conclude that there can only be one stable endemic equilibrium when $R_0 > 1$. In models with multi-group infectious classes, forward bifurcation commonly exist [6]. This could be the reason for the existence of forwards bifurcation for pneumonia transmission dynamic.

The mathematical techniques involved in determining the global stability of the endemic equilibrium is quite complicated and therefore in this paper we determine the global stability of the endemic equilibrium using numerical simulation (See Figure 2). We observe from Figure 2 that starting with any number of infected individuals with the initial population $N_0 = 100$, the number of the Susceptible and the infected will always converge to a stable value ($S^e = 24.41243257, I^e = 4.549013989 \times 10^{-2}$). Assuming that we reduce the transfer rates between the Carriers and the Infected with the aim or reducing R_0, the infected population also reduce.

Using MALPLE 14, data for the infected population in both the cases (dotted line and continuous line in Figure 3) was generated and analyzed to check if their is any significant difference in the two populations. Table 2 shows the results of the statistical analysis. Since the P-value=2.1344×10^{-15} for the t-test for mean difference is less than 0.01, we conclude that there is a strong significant effect of reducing the rates of transfer between the carriers and the infected on reducing the infected populations. We also Simulate the effect of different proportion of carriers on transmission by considering different initial proportion of carriers and different rate of transfer leading to increase in carrier proportion in the population.

6   Interpretation of the model and Biological Implication

The results from the analysis of the model indicate that possible disease control strategy will be to reduce the number of new secondary infections (i.e. reducing

the value of the Basic reproduction number, R_0). Rewriting (4) into,

$$R_0 = \kappa \mathcal{P} \left(\frac{\rho[\varepsilon(\mu+\alpha+\eta)+\pi]+(1-\rho)[\mu+\beta+\pi+(1-q)\varepsilon\eta]}{(\mu+\alpha+\eta)(\mu+\beta+\pi)-(1-q)\pi\eta} \right)$$

It is evident that R_0 is directly proportional to the contact rate κ and to the mean time spent in the diseased classes. $\frac{1}{(\mu+\alpha+\eta)(\mu+\beta+\pi)-(1-q)\pi\eta}$. The implication of reducing the contact rate $\kappa \to 0$ and mean time spent in the diseased classes ensures that $R_0 \to 0$. It is possible to reduce mean time spent in the diseased classes when the transfer rates between the Carrier and the Infected classes are reduced (i.e. $\pi \to 0$ and $q \to 1$) and when the transfer rate out from the diseased classes are increased (i.e. $\alpha, \beta \to \infty$). This indicates that quarantine (where possible), prompt and effective diagnosis and treatment of the carriers and the infected individuals may lead to possible reduction of the new infections to zero. A justification of controlling pneumonia by reducing the R_0 is indicated by the forward bifurcation results in the analysis. In the presence of a forward bifurcation implies that, a disease can be cleared from the population by just reducing the R_0.

7 Discussion

Mathematical models of infectious diseases have been used to successfully explain the transmission dynamics of many diseases and the use of such models has grown exponentially from mid 20th century [10]. Our main aim in this paper was to provide a mathematical explanation of pneumonia transmission dynamics, taking into consideration the role of carriers and recovery measures in the transmission. we only considered the bateremic pneumonia since it is the most common among children who are under five years of age.

The model that we have discussed here is based on the initial model that was studied by Doura et al.[5]. When studying the transmission dynamics of infectious diseases with an aim of suggesting control measures, it is natural to consider the stability of equilibrium points and possibility of bifurcation. In this paper we have established R_0, existence and stability of the equilibrium points and existence of bifurcation point. Our main results indicates that when $R_0 < 1$ then the disease free equilibrium is stable and become unstable when $R_0 > 1$. The Local stability of the endemic equilibrium point E^e changes its nature to unstable when ψ crosses the critical value ψ_c via a forward bifurcation. This is a clear indication that the effective control measure for pneumonia is achieved when R_0 is reduced.

Most of the results in this paper are in agreement with those of [5]. However, We find some interesting results in the numerical simulation that is; reducing the transfer rates between the carrier and the infected class reduces prevalence of the disease. This is a control strategy that can be employed for pneumonia dynamics.

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Appendix

Table 1: Parameter Value

Parameter	Value	Source
ν	μN_0	[5]
κ	1-10 per day	Estimated
$\mathcal{P}$	0.89 to 0.99	[5]
ψ	$\kappa\mathcal{P}$	Expressed as in (2)
ϵ	0.001124	[5]
ρ	0.338	[13, 14]
π	0.00274 to 0.01096 per day	[5]
η	0.0238 to 0.0476 per day	[26]
q	0.5 to 1	[5]
α	0.33	Estimated
δ	0.2	Estimated
μ	0.0002 per day	[20]
β	0.0115	[11]

Table 2: t-Test: Paired Two Sample for Means(testing for the significant different between infected populations when the values of π β and q are varied respectively)

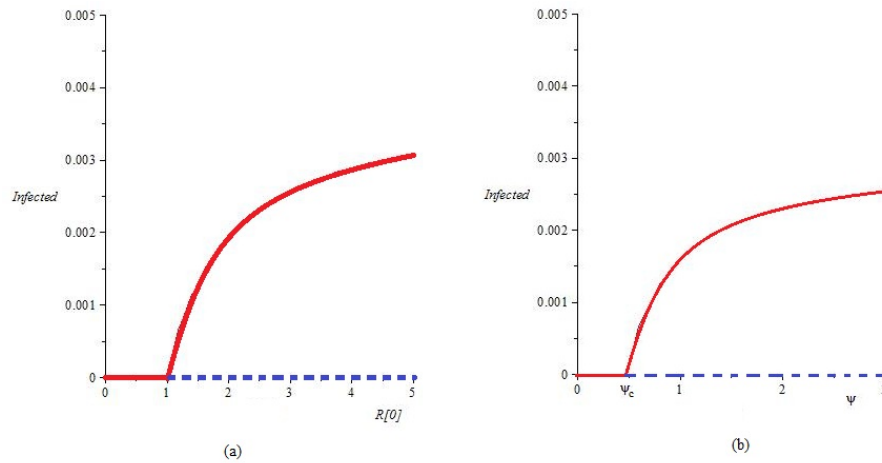| Statistics	Infected Population (π =0.005, q=0.75)	Infected population (π =0, q=0.999)
Mean	2.486300321	2.170599263
Variance	20.90683033	20.72202111
Observations	100	100
Hypothesis	Mean Difference=0	
df	99	
t Statistics	9.408577558	
P- Value	2.1344×10^{-15}	
t Critical	1.9842169	

Figure 1: Forward bifurcation diagram in (a) plane I,R_0 and in (b) plane I,ψ The continuous line represent a stable equilibrium. There are two stable equilibriums (disease free equilibrium for $R_0 < 1$ and an endemic equilibrium for $R_0 > 1$). The dotted line represent the unstable disease free equilibrium
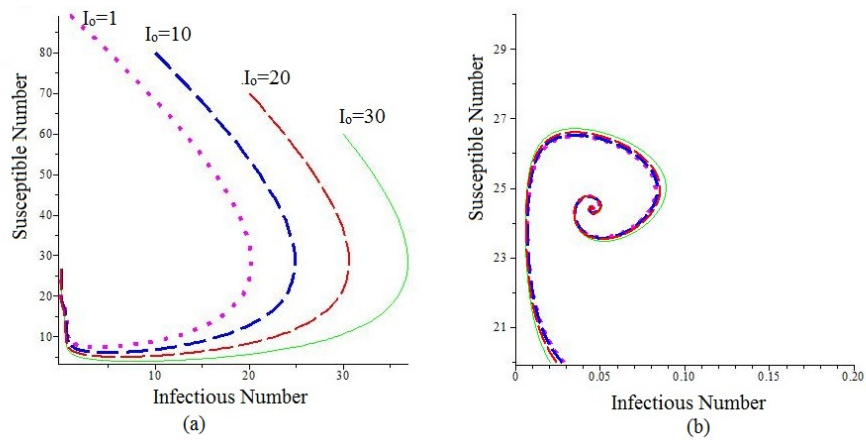

Figure 2: The phase plane portrait of S vs I for 3. The four curves correspond to the initial conditions $I_0 = 1$, 10, 20 and 30 respectively. They all converge at $I^e = 4.549013989$ and $S^e = 24.41243257$ as in (b) when the plot is magnified. showing global asymptotic stability of the endemic equilibrium
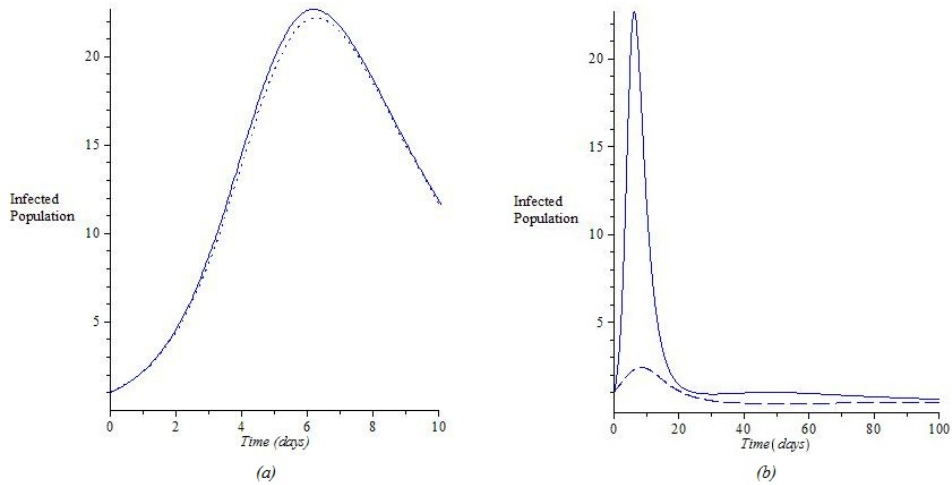
Figure 3: (a) Dynamics considering carrier-infected interaction rates. The continuous line is plotted when $\pi = 0.005$, and $q = 0.75$, while the dotted line is plotted when $\pi = 0$ and $q = 0.999$. (b) Dynamics considering recovery rate of the infected and carrier-infected transfer rates. The continuous line is plotted when $\eta = 0.03, \pi = 0.005$ and $q = 0.75$ while The dotted line is plotted when $\eta = 0.6$, $\pi = 0$ and $q = 0.999$
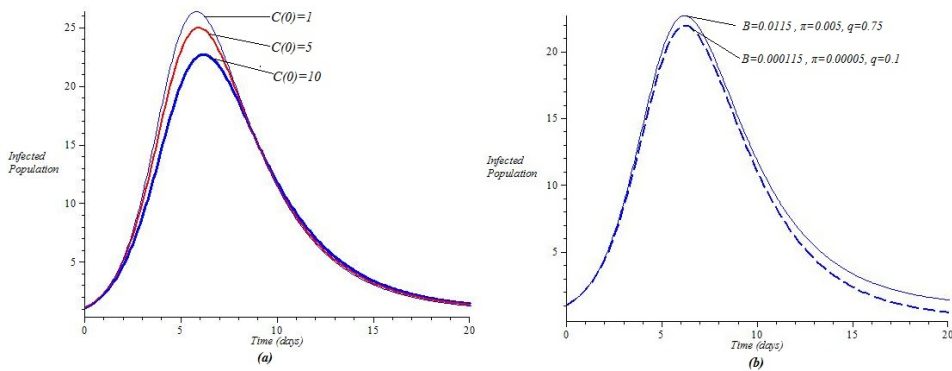

Figure 4: Simulation of model 3, (a) $C(0) = 1, C(0) = 5$ and $C(0) = 10$ and all other parameters are the same (b) with ($\beta = 0.01156$, $\pi = 0.005$ $q = 0.75$) and ($\beta = 0.0001156$, $\pi = 0.00005$ $q = 0.1$)