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Impact of Self-Medication and Hospitalization on Pneumonia Transmission: A Mathematical Modeling Approach

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ABSTRACT
Pneumonia continues to pose a significant public health challenge, with treatment behaviors such as self-medication and hospitalization playing critical roles in disease control. This study developed a mathematical model to investigate the impact of these two treatment strategies on pneumonia transmission. The basic reproduction number (R_0) was computed using next generation matrix method, the disease-free equilibrium is locally and asymptotically when R_0 <1 and unstable when stable R_0 > 1 revealing that under effective hospitalization, while high reliance on self-medication promotes a stable endemic state. Sensitivity analysis showed that parameters representing the contact rate between susceptible and infected individuals and the progression from exposed to actively infected individuals increased disease spread, whereas hospitalization and vaccination rates reduced transmission. Numerical simulations confirmed that high rates of self-medication prolong infection and increase overall disease burden**,** while hospitalization and vaccination significantly reduce infections and accelerate recovery**.** Based on these findings, the study recommends improving access to hospitals, expanding vaccination coverage, and conducting public awareness campaigns to discourage self-medication.

Keywords:
Self-Medication;
Hospitalization;
Sensitivity Analysis;
Numerical Simulation;
Basic reproduction Number

INTRODUCTION

Pneumonia remains a major global public health concern and is widely recognized as one of the leading causes of morbidity and mortality across all age groups. It is an acute respiratory infection that affects the lung parenchyma, particularly the alveoli, leading to impaired gas exchange due to fluid or pus accumulation. Empirical evidence from recent studies indicates that pneumonia continues to account for a significant proportion of infectious disease-related deaths globally despite advancements in treatment and vaccination strategies (Anderson & Feldman, 2023; Kato, 2024). These findings underscore the persistent burden of the disease and highlight the need for sustained research and improved healthcare interventions. The etiology of pneumonia is complex and involves a wide range of pathogens, including bacteria, viruses, and fungi. Empirical studies have consistently identified *Streptococcus pneumoniae* as a leading bacterial cause, while atypical pathogens and viral agents also contribute significantly to disease incidence (Seid et al., 2024; Wang et al., 2023). A cross-sectional study conducted in Tanzania further demonstrated variations in bacterial pathogens and antibiotic resistance patterns,

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particularly during and after the COVID-19 pandemic, emphasizing the evolving nature of pneumonia etiology (Rukyaa et al., 2024). These empirical findings highlight the challenges in diagnosis and treatment, as pathogen diversity and resistance patterns continue to change. Recent epidemiological studies have also provided insights into the global and regional burden of pneumonia. For instance, a spatial epidemiological study conducted in Pakistan revealed significant variations in pneumonia incidence based on environmental and socio-demographic factors, indicating that disease distribution is influenced by contextual determinants (Ünsal et al., 2025). Similarly, empirical research in Ethiopia reported millions of annual pneumonia cases among children under five, identifying key risk factors such as malnutrition, poor healthcare access, and environmental exposures (Osman et al., 2025). These studies demonstrate that pneumonia disproportionately affects vulnerable populations, particularly in low- and middle-income countries. In addition to its high prevalence, pneumonia is associated with severe clinical outcomes and complications, particularly among high-risk groups such as children, the elderly, and immunocompromised individuals. Empirical evidence shows that pneumonia incidence is increasing among these populations, partly due to factors such as aging populations, comorbidities, and emerging infectious diseases (Kato, 2024; Tilahun et al., 2024). Furthermore, recent clinical studies have highlighted the role of co-infections and secondary bacterial pneumonia in worsening patient outcomes, particularly in the context of viral infections such as COVID-19 (Cheng et al., 2024). These findings reinforce the need for early diagnosis and effective management strategies. Efforts to reduce the burden of pneumonia have focused on prevention, improved diagnostics, and effective treatment strategies. Empirical research supports the use of vaccination, antimicrobial therapy, and improved healthcare access as key interventions in reducing morbidity and mortality (Anderson & Feldman, 2023; Kato, 2024). However, challenges such as antimicrobial resistance, limited healthcare infrastructure, and disparities in access to care continue to hinder progress. Consequently, a comprehensive and evidence-based approach that integrates clinical, epidemiological, and policy interventions is essential to effectively address pneumonia as a persistent global health threat.

Almutairi and El-Shahed (2025) developed a deterministic mathematical model to study the transmission dynamics of pneumonia and assess the effectiveness of control strategies. They analyzed both disease-free and endemic equilibrium states and demonstrated that the basic reproduction number (R_0) determined whether the disease persisted or was eliminated. The authors further incorporated optimal control measures such as awareness programs, screening, and treatment, and found that a combination of these interventions significantly reduced infection rates and improved disease management. Celestine, Ibrahim, and Yusuf (2026) proposed a fractional-order mathematical model to better describe pneumonia transmission dynamics. They incorporated memory effects, vaccination, and reinfection into the model and fitted it with real-world data. Their findings showed that the fractional-order approach provided more accurate predictions than classical models. The authors concluded that vaccination and waning immunity played significant roles in shaping the long-term behavior of pneumonia spread. Fatmawati and Khan (2025) examined the co-dynamics of pneumonia and malnutrition using a SEIRS modeling framework. They found that malnutrition significantly increased susceptibility to infection and worsened disease outcomes among children under five. Through sensitivity analysis, the authors identified transmission rate and nutritional status as key factors influencing disease spread. They concluded that integrating nutritional interventions with medical treatment was essential for effective pneumonia control. Alemneh and Kassa (2023) developed an age-structured mathematical model to analyze pneumonia transmission across different population groups. They incorporated treatment and nutritional interventions into the model and showed that these combined strategies significantly reduced the effective reproduction number and overall disease burden. Their stability analysis indicated that pneumonia could be controlled or eradicated under optimal intervention conditions, emphasizing the importance of targeted public health strategies.

The study sought to examine the transmission dynamics of pneumonia while assessing the relative effectiveness of hospital-based treatment compared to self-medication in reducing its burden within the human population. It was motivated by the need to develop a deeper and more comprehensive understanding of how pneumonia spreads and persists, as well as to identify effective intervention strategies for its control. By exploring these dynamics, the research aimed to generate insights that could support evidence-based public health decisions and inform policy development directed at minimizing the impact of the disease.

To achieve this aim, the study first developed a mathematical model that described the transmission dynamics of pneumonia within a population. It then determined the basic reproduction number (R_0), which served as a key threshold parameter for assessing whether the disease would spread or decline. The stability of the model was subsequently analyzed to understand the conditions under which the disease-free or endemic states would persist. In addition, sensitivity analysis was performed to evaluate how variations in key parameters influenced the reproduction number and overall disease

dynamics. Finally, numerical simulations were conducted to validate the analytical results and to provide further insight into the behavior of the model under different scenarios. The novelty of this study lies in its integration of self-medication and hospitalization as distinct control measures within a mathematical model of pneumonia transmission. Unlike previous models, it captures real-world treatment behaviors and compares their effectiveness in reducing disease burden. The study highlights the risks of self-medication, such as incomplete treatment and delayed recovery, versus the benefits of hospital-based care. Through sensitivity analysis and numerical simulations, it identifies key factors affecting disease dynamics, providing both theoretical insights and practical guidance for public health strategies aimed at optimizing pneumonia control.

MATERIALS AND METHODS

Model Formulation.

The human population at time t, denoted by $N(t)$ is sub-divided into six(6) mutually exclusive compartments of Susceptible humans $S(t)$, Exposed humans $E(t)$, Infected humans $I(t)$, Self-medication humans $M(t)$, Hospitalized humans $H(t)$, and Recovered humans $R(t)$.

The recruitment rate of individuals into the susceptible population is at the rate Λ and there are vaccinated against pneumonia at the rate of g. The rate at which the vaccine fails or wanes is denoted as ω. The rate at which the vaccine proves to be effective and the vaccinated individuals progresses into the recovered class is denoted as k. We denote β as the effective contact rate of humans with the probability of been infected per contact with an infected pneumonia individual. Upon exposure to the pneumonia causing bacteria, an individual becomes infected with the virus at the rate of θ. The infected progress into the self-medication class at the rate of γ and can chose to progress into the Hospitalized class at the rate of τ for treatment. In a case when an infected under self-medication does not positively respond to the treatment through recovery, they may choose to progress into the hospitalized class at the rate of ψ. The individuals under self-medication recover at a reduced rate of $\rho\varepsilon$ where ρ is the modification parameter that accounts for reduced rate of recovery in the self-medication class. The individuals in the hospitalized class recover at the rate of ε. The natural death rate of humans is denoted as μ. The disease induced death of infected humans is denoted as δ_P. The disease induced death rate in the self-medication class is denoted as δ_M. The disease induced death rate in the hospitalized class is denoted as $\phi\delta_H$, where ϕ is the modification parameter that accounts for reduced death in the hospitalized class.

The total human population is denoted as: $N(t)=S(t)+V(t)+E(t)+M(t)+H(t)+R(t)$. In formulating the mathematical model for pneumonia transmission, several key assumptions were made to reflect realistic disease dynamics and treatment effects. First, it was assumed that the disease-induced death rate among hospitalized individuals is lower than that of individuals relying on self-medication, reflecting the improved medical care, monitoring, and supportive treatment available in hospitals. Second, the model accounted for the waning of vaccine-induced immunity over time, recognizing that protection against pneumonia may diminish, thereby affecting susceptibility within the population (Martínez *et al*, 2013). Third, it was assumed that infected individuals receiving hospital-based treatment recover more rapidly compared to those who self-medicate, capturing the higher effectiveness of professional treatment in reducing the duration and severity of infection. These assumptions allowed the model to differentiate between formal and informal treatment pathways and to realistically evaluate the impact of self-medication and hospitalization on pneumonia transmission and control.

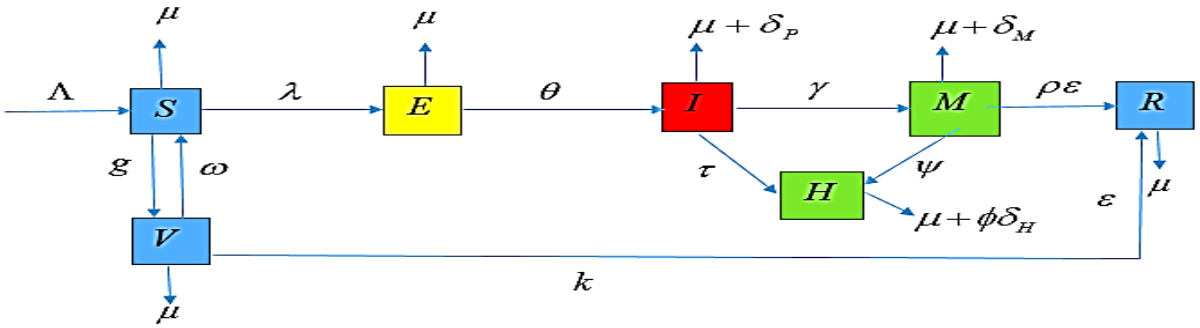

Fig. 1: Schematic diagram of the Pneumonia model

Table 1: Variable and Parameters description

Variable	Description
S	Susceptible Humans
V	Vaccinated Humans
E	Exposed Humans
I	Infected Humans
M	Self-Medication Humans
H	Hospitalized Humans
R	Recovered Humans
PARAMETER DESCRIPTION	
Λ	Recruitment rate of humans
β	Contact rate of susceptible and infected humans
g	Vaccination rate of susceptible humans
ω	Rate of vaccine failure
θ	Progression rate from exposed class to infected class
γ	Rate of self-medication.
τ	Progression rate from infected class to hospitalized class
ψ	Progression rate from Self-medication class into the Hospitalized class.
ρ	Modification parameter that accounts for reduced recovery in the self-medication class.
ε	Recovery rate of humans in the treatment classes
δ_P	Disease induced death rate of untreated infected humans
δ_M	Disease induced death rate of individuals under self-medication
δ_H	Disease induced death rate of individuals in the Hospitalized class
ϕ	Modification parameter that accounts for reduced death in the Hospitalized class
μ	Natural death rate of Humans

Model Equations

In the light of the description of the model above, the differential equations modeling the transmission dynamics of Pneumonia in the population is given as

$$\frac{dS}{dt} = \Lambda + \omega V - (\lambda + g + \mu)S,$$
$$\frac{dV}{dt} = gS - (\omega + k + \mu)V,$$

$$\frac{dE}{dt} = \lambda S - (\theta + \mu)E,$$
$$\frac{dI}{dt} = \theta E - (\gamma + \tau + \delta_P + \mu)I,$$
$$\frac{dM}{dt} = \gamma I - (\psi + \rho\varepsilon + \delta_M + \mu)M, \quad (1)$$
$$\frac{dH}{dt} = \tau I + \psi M - (\varepsilon + \phi\delta_H + \mu)H,$$
$$\frac{dR}{dt} = kV + \rho\varepsilon M + \varepsilon H - \mu R.$$

The force of infection of the Pneumonia model in (1) is given as:

$$\lambda = \frac{\beta(I + M + H)}{N},$$

$P_1 = (\omega + k + \mu)$,

$P_2 = (\theta + \mu)$,

Let $P_3 = (\gamma + \tau + \delta_P + \mu)$,

$P_4 = (\psi + \rho\varepsilon + \delta_M + \mu)$,

$P_5 = (\varepsilon + \phi\delta_H + \mu)$

RESULTS AND DISCUSSION

Invariant region of the Pneumonia model

The solutions of the proposed Pneumonia model are feasible for all t > 0, if they enter the invariant region D, which is given by:

$$D = \left\{ \begin{array}{l} (S,V,E,I,M,H,R): \\ S > 0, V > 0, E > 0, I > 0, \\ M > 0, R > 0, N < \frac{\Lambda}{\mu} \end{array} \right\}$$

Proof

The total population of the humans in the Pneumonia model is given as

$$N(t) = S(t) + V(t) + E(t) + I(t) + M(t) + H(t) + R(t)$$

The sum of the differential equations is

$$N'(t) = S'(t) + V'(t) + E'(t) + I'(t) + M'(t) + H'(t) + R'(t)$$

On evaluating the algebraic terms, we obtain

$$N'(t) = \Lambda - (S + V + E + I + M + H + R)\mu - \delta_P I - \delta_M M - \phi\delta_H H$$
$$N'(t) = \Lambda - \mu N - \delta_P I - \delta_M M - \phi\delta_H H$$
$$\frac{dN}{dt} \le \Lambda - \mu N$$

Solving the differential equation using the integrating factor method, we obtained

$$N(t) \le \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}$$

Applying Birkhoff and Rota's theorem on the inequality, we obtain

$$0 \le N \le \frac{\Lambda}{\mu} \text{ as } t \to \infty$$

Thus, D is a positively invariant set under the flow described by the model (1) so that no solution path leaves through the boundary of region D (Martínez *et al*, 2013). Thus, in this region, the Pneumonia only model can be considered as being epidemiologically and mathematically well posed.

Positivity of solution of the Pneumonia model

It is necessary to prove that all state variable of the Pneumonia model in are nonnegative for all time (t), for the model to be epidemiologically and mathematically well posed in a feasible region D given by:

$$D = \left\{ \begin{array}{l} (S,V,E,I,M,H,R) \in R_+^7 : \\ (S + V + E + I + M + H + R) \le N \end{array} \right\}$$

This is done by considering,

$$\{(S,V,E,I,M,H,R) \ge 0 \in R_+^7\}$$

Lemma 1:

Let the initial data for the model (1) be $(S,V,E,I,M,H,R) > 0$. Then the solutions (S,V,E,I,M,H,R) of the model (1) are positive for all time $t > 0$

Proof

Let

$t = \sup\{t > 0 : S > 0, V > 0, E > 0, I > 0, M > 0, R > 0 \in [0,t]\}$.

Thus $t > 0.$

We have from the first equation that

$$\frac{dS}{dt} = \Lambda + \omega V - (\lambda + g + \mu)S,$$
$$\frac{dS}{dt} \geq -(\lambda + g + \mu)S$$

This can also be written as

$$\int \frac{dS}{S} \geq -\int (\lambda + g + \mu)dt$$

We obtained:

$$\ln S \geq -(\lambda + g + \mu)t + C$$
$$S(t) \geq Ce^{-(\lambda + g + \mu)t}$$

Applying the initial condition; when $t = 0,\ S(0) = C$

Therefore, $S(t) \geq S(0)e^{-(\lambda+g+\mu)t} \geq 0$ since $(\lambda + g + \mu) > 0$

Similarly, it can be shown that $V, E, I, M, H, R > 0$

Asymptotic stability of the disease-free equilibrium of the Pneumonia model

The steady state where there is no infection (or absence of the disease), a point where $E = I = M = H = M = R = 0$ is called the disease-free equilibrium point (DFE) which is given

$$\eta_0 = \{S^*, V^*, E^*, I^*, M^*, H^*, R^*\} =$$
$$\left\{\frac{\Lambda(k+\mu)}{\mu^2 + (g+k)\mu + (k-\omega)g}, \frac{g\Lambda}{(k+\mu-\omega)g + \mu(k+\mu)}, 0, 0, 0, 0, 0\right\}$$

Thus, at the DFE point of the Pneumonia model, we obtain the total human population as

$$N = S + V = \frac{\Lambda(g+k+\mu)}{\mu^2 + (g+k)\mu + (k-\omega)g}$$

Basic Reproduction Number of the Pneumonia Model

The basic reproduction number of Pneumonia infected individuals denoted by R_0 is defined as the average number of secondary infections produced by a single Pneumonia infectious individual introduced in a wholly susceptible population during his or her entire infectious period (Martínez *et al*, 2013). We calculate the basic reproduction number by using the next generation operator method on the dynamical system (1).

Hence, it follows that

$R_0 = \rho(FV^{-1})$ where ρ is the dominant eigenvalue of FV^{-1}

$$F = \begin{bmatrix} 0 & \frac{\beta(k+\mu)}{g+k+\mu} & \frac{\beta(k+\mu)}{g+k+\mu} & \frac{\beta(k+\mu)}{g+k+\mu} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} P_2 & 0 & 0 & 0 \\ -\theta & P_3 & 0 & 0 \\ 0 & -\gamma & P_4 & 0 \\ 0 & -\tau & -\psi & P_5 \end{bmatrix},$$

$$V^{-1} = \begin{bmatrix} \frac{1}{P_2} & 0 & 0 & 0 \\ \frac{\theta}{P_2P_3} & \frac{1}{P_3} & 0 & 0 \\ \frac{\gamma\theta}{P_3P_2P_4} & \frac{\gamma}{P_3P_4} & \frac{1}{P_4} & 0 \\ \frac{\sigma(P_4\tau + \gamma\psi)}{P_4P_3P_2P_5} & \frac{P_4\tau + \gamma\psi}{P_4P_3P_5} & \frac{\psi}{P_4P_5} & \frac{1}{P_5} \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} \frac{((P_5+\tau)P_4+\gamma(P_5+\psi))(k+\mu)\theta\beta}{(g+k+\mu)P_4P_3P_2P_5} & \frac{\beta((P_5+\tau)P_4+\gamma(P_5+\psi))(k+\mu)}{(g+k+\mu)P_4P_3P_5} & \frac{\beta(k+\mu)(P_5+\psi)}{(g+k+\mu)P_4P_5} & \frac{\beta(k+\mu)}{(g+k+\mu)P_5} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

Therefore, the basic reproduction number of the Pneumonia only model is

$$R_0 = \frac{\beta\theta(k+\mu)((P_5+\tau)P_4 + \gamma(P_5+\psi))}{(g+k+\mu)P_2P_3P_4P_5}$$

Local Asymptotic Stability of the DFE of the Pneumonia Model

Theorem 1

The disease-free equilibrium point of the Pneumonia only is locally asymptotically stable (LAS) if $R_0 < 1$, *and unstable if* $R_0 > 1$.

Proof

Using Jacobian matrix to prove the local stability of the disease free equilibrium point

$$J(\eta_0)=\begin{bmatrix} -(g+\mu) & \omega & 0 & -\frac{\beta(k+\mu)}{g+k+\mu} & -\frac{\beta(k+\mu)}{g+k+\mu} & -\frac{\beta(k+\mu)}{g+k+\mu} & 0 \\ g & -P_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -P_2 & \frac{\beta(k+\mu)}{g+k+\mu} & \frac{\beta(k+\mu)}{g+k+\mu} & \frac{\beta(k+\mu)}{g+k+\mu} & 0 \\ 0 & 0 & \theta & -P_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -P_4 & 0 & 0 \\ 0 & 0 & 0 & \tau & \psi & -P_5 & 0 \\ 0 & k & 0 & 0 & \rho\varepsilon & \varepsilon & -\mu \end{bmatrix}$$

Since the diagonal last column consist of only the diagonal element which forms the first eigenvalue, we thus can reduce $J(\eta_0)$ to

$$J_1(\eta_0)=\begin{bmatrix} -(g+\mu) & \omega & 0 & -\frac{\beta(k+\mu)}{g+k+\mu} & -\frac{\beta(k+\mu)}{g+k+\mu} & -\frac{\beta(k+\mu)}{g+k+\mu} \\ g & -P_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & -P_2 & \frac{\beta(k+\mu)}{g+k+\mu} & \frac{\beta(k+\mu)}{g+k+\mu} & \frac{\beta(k+\mu)}{g+k+\mu} \\ 0 & 0 & \theta & -P_3 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -P_4 & 0 \\ 0 & 0 & 0 & \tau & \psi & -P_5 \end{bmatrix}$$

The characteristics polynomial of $J_1(\eta_0)$ is given as

$$\lambda^6+A_1\lambda^5+A_2\lambda^4+A_3\lambda^3+A_4\lambda^2+A_5\lambda^3+A_6\lambda^2+A_7\lambda+A_8=0$$

Where,

$$A_1=P_5+P_4+P_3+P_2+P_1+g+\mu,$$

$$A_2=\frac{\begin{pmatrix}(-\omega+P_1+P_2+P_3+P_4+P_5)g^2\\+\left(\begin{pmatrix}(-\omega+2P_1+2P_2+2P_3+2P_4+2P_5)\mu+(-\omega+P_1+P_2+P_3+P_4+P_5)k\\+(P_5+P_4+P_3+P_2)P_1+(P_5+P_4+P_3)P_2+(P_5+P_4)P_3+P_4P_5\end{pmatrix}g\right.\\-(k+\mu)\begin{pmatrix}(-P_1-P_2-P_3-P_4-P_5)\mu+(-P_2-P_3-P_4-P_5)P_1\\+(-P_3-P_4-P_5)P_2+(-P_4-P_5)P_3+\theta\beta-P_4P_5\end{pmatrix}\end{pmatrix}}{g+k+\mu}$$

$$A_3=\frac{\begin{pmatrix}-(g+\mu+P_3+P_4+P_5)(g+k+\mu)P_2-(g+\mu+P_4+P_5)(g+k+\mu)P_3\\+(-P_4-P_5)\mu^2+((-2P_4-2P_5)g+(-k-P_5)P_4+\theta\beta-kP_5)\mu\\+(-P_4-P_5)g^2+((-k-P_5)P_4-kP_5)g+k(-P_4P_5+\theta\beta)\end{pmatrix}P_1}{-P_2\left((g+\mu+P_4+P_5)(g+k+\mu)P_3+P_4\left(P_5g+kP_5+P_5\mu+g^2\right)\right)}{g+k+\mu},$$

$$A_4=\frac{P_1\begin{pmatrix}\left(\begin{pmatrix}(g+\mu+P_3+P_5)(g+k+\mu)P_2+(g+\mu+P_5)(g+k+\mu)P_3\\+(g+\mu)(g+k+\mu)P_5-\theta\beta(k+\mu)\end{pmatrix}P_4\right.\\+(g+k+\mu)\left(\left((g+\mu+P_5)P_3+P_5(g+\mu)\right)P_2+P_3P_5(g+\mu)\right)\end{pmatrix}}{g+k+\mu},$$

$$A_5=\frac{P_1\left(\begin{pmatrix}\left(\begin{pmatrix}(g+\mu+P_3)(g+k+\mu)P_2\\+(g+\mu)(g+k+\mu)P_3-\theta\beta(k+\mu)\end{pmatrix}P_5\right.\\+(g+\mu)(g+k+\mu)P_3P_2-\beta\theta(k+\mu)(g+\mu+\tau)\end{pmatrix}P_4\right)}{g+k+\mu}+P_2P_3P_5(g+\mu)(g+k+\mu),$$

$$A_6=\frac{\begin{pmatrix}(g+\mu)\left(\left((\tau+P_5)P_4+\gamma(P_5+\psi)\right)\beta(k+\mu)\theta+P_5P_2P_3P_4(g+k+\mu)\right)P_1-\beta g\gamma\mu\omega\psi\theta\\+P_2P_3P_4P_5g\omega(g+k+\mu)(1-R_0)\end{pmatrix}}{g+k+\mu}$$

Applying Routh-Hurwitz criterion to the Characteristics polynomial, we have that

$$(1-R_0)>0$$

$$\Rightarrow R_0<1$$

Thus, the DFE point of the Pneumonia only model is locally asymptotically stable.

Global Asymptotic Stability of the Disease-free equilibrium point of the Pneumonia Model.

To investigate the global stability of the disease free equilibrium, we use the technique implemented by Castillo-Chavez and song (Abata et al, 2025).

To do this, we write the equation in the uninfected class as

$$\frac{dX}{dt} = F\left(X, Z\right)$$

And we re-write the equation in the infected class as

$$\frac{dz}{dt} = G(X, Z)$$

Where $X = \left(S, V, R\right) \in ¡^{2}_{+}$ denotes the uninfected population and

$Z = \left(E, I, M, H\right) \in ¡^{4}_{+}$ denotes the infected population

$\varepsilon_0 = (X^*, 0)$ represent the disease free equilibrium of the system, and it globally asymptotically stable if it satisfies the following conditions:

$H_1 : \frac{dX}{dt} = F\left(X^*, 0\right), X^*$ is globally asymptotically stable

$$H_2 : \frac{dZ}{dt} = D_Z G\left(X^*, 0\right) Z - \hat{G}\left(X, Z\right)$$

$\hat{G}\left(X, Z\right) \geq 0$ for all $\left(X, Z\right) \in D$ and where $D_Z G\left(X^*, 0\right)$ is an M- matrix (i.e the diagonal elements are no-negative and it is also the Jacobian of $\hat{G}\left(X, Z\right) \geq 0$ evaluated at $(X^*, 0)$.

If the system satisfies the above condition, then the theorem below holds.

Theorem 2

The equilibrium point $\eta_0 = (X^*, 0)$. *is globally asymptotically stable if* $R_0 \leq 1$

$$F(X, Z) = \begin{bmatrix} \Lambda + \omega V - \left(\lambda + g + \mu\right) S, \\ gS - \left(\omega + k + \mu\right) V \\ kV + \rho\varepsilon M + \varepsilon H - \mu R. \end{bmatrix},$$

$$G(X, Z) = \begin{bmatrix} \lambda S - \left(\theta + \mu\right) E \\ \theta E - \left(\gamma + \tau + \delta_P + \mu\right) I \\ \gamma I - \left(\psi + \rho\varepsilon + \delta_M + \mu\right) M \\ \tau I + \psi M - \left(\varepsilon + \phi\delta_H + \mu\right) H \end{bmatrix}$$

At disease free equilibrium,

$H_1 :$

$$\frac{dS}{dt} = \Lambda - \left(g + \mu\right) S$$

$$\frac{dR}{dt} = 0$$

$H_2 :$

$$D_z G\left(X^*, 0\right) Z = \begin{bmatrix} \beta\left(I + M + H\right) - \left(\theta + \mu\right) E \\ \theta E - \left(\gamma + \tau + \delta_P + \mu\right) I \\ \gamma I - \left(\psi + \rho\varepsilon + \delta_M + \mu\right) M \\ \tau I + \psi M - \left(\varepsilon + \phi\delta_H + \mu\right) H \end{bmatrix}$$

$$\hat{G}\left(X, Z\right) = D_Z G\left(X^*, 0\right) Z - G\left(X, Z\right)$$

$$\hat{G}\left(X, Z\right) = \begin{bmatrix} \beta\left(I + M + H\right)\left(1 - \frac{S}{N}\right) \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

Clearly, $1 \geq \frac{S}{N}$ this implies that $\hat{G}\left(X, Z\right) \geq 0$.

Therefore, the disease free equilibrium of the Pneumonia model is globally asymptotically stable.

Endemic Equilibrium Point of the Pneumonia Model

The endemic equilibrium point is the steady state where there is persistence or prevalence of a disease in the population.

Theorem 3

The endemic equilibrium point of the Pneumonia model in (1) is stable if $R_0 > 1$ *and unstable if* $R_0 < 1$

Proof

To obtain the endemic equilibrium we set the RHS of the differential equations in (1) to zero and solve for the state variables.

Thus, at the endemic equilibrium point, $\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dM}{dt} = \frac{dH}{dt} = \frac{dR}{dt} = 0$.

Let $\eta^{**} = \left(S^{**}, V^{**}, E^{**}, I^{**}, M^{**}, H^{**}, R^{**}\right)$ be the endemic equilibrium point.

We have that,

$$S^{**} = \frac{\Lambda P_1}{P_1\left(\lambda^{**} + g + \mu\right) - \omega g}$$

$$V^{**} = \frac{\Lambda g}{P_1\left(\lambda^{**} + g + \mu\right) - \omega g}$$

$$E^{**} = \frac{\Lambda P_1 \lambda^{**}}{\left(P_1 g + P_1 \lambda^{**} + P_1 \mu - \omega g\right) P_2}$$

$$I^{**} = \frac{\theta \Lambda P_1 \lambda^{**}}{\left(P_1 g + P_1 \lambda^{**} + P_1 \mu - \omega g\right) P_2 P_3}$$

$$M^{**} = \frac{\theta \Lambda P_1 \lambda^{**} \gamma}{P_4\left(P_1\left(\lambda^{**} + g + \mu\right) - \omega g\right) P_3 P_2}$$

$$H^{**} = -\frac{\theta \Lambda P_1 \lambda^{**}\left(P_4 \tau + \gamma \psi\right)}{P_2 P_5 P_4\left(\left(-g - \mu - \lambda^{**}\right) P_1 + \omega g\right) P_3}$$

$$R^{**} = -\frac{\left(\left(P_4 \tau + \gamma\left(P_5 \rho + \psi\right)\right)\theta \varepsilon \lambda^{**} P_1 + gkP_2 P_3 P_4 P_5\right)\Lambda}{\left(\left(-g - \mu - \lambda^{**}\right) P_1 + \omega g\right) P_3 \mu P_5 P_2 P_4}$$

Substituting them into the force of infection,

$\lambda^{**} = \dfrac{\beta\left(I^{**} + M^{**} + H^{**}\right)}{N^{**}}$, we obtained the following:

$f(\lambda)^{**} = \lambda^{**}\left(A\lambda^{**} + B\right) = 0$ and $\lambda^{**} = 0$ denotes the disease free equilibrium point of the Pneumonia Model.

Where,

$$A = \left(\mu\left(\theta + P_3\right) P_5 + \theta\tau\left(\epsilon + \mu\right)\right) P_4 + \theta\gamma\left(\left(\epsilon\rho + \mu\right) P_5 + \psi\left(\epsilon + \mu\right)\right)$$

,

$$B = \left(g + k + \mu\right) P_4 P_3 P_2 P_5 \left(1 - R_0\right)$$

$$\lambda^{**} = \frac{\left(g + k + \mu\right) P_4 P_3 P_2 P_5 \left(R_0 - 1\right)}{\left(\mu\left(\theta + P_3\right) P_5 + \theta\tau\left(\epsilon + \mu\right)\right) P_4 + \theta\gamma\left(\left(\varepsilon\rho + \mu\right) P_5 + \psi\left(\epsilon + \mu\right)\right)\left(\phi\delta_H + \varepsilon + \mu\right)}$$

Thus for λ^{**} to be positive at the endemic equilibrium point, $R_0 - 1 > 0$.

$\Rightarrow R_0 > 1$ and the endemic equilibrium point is stable.

Sensitivity Analysis of the Pneumonia Model

Sensitivity analysis is carried out to determine the parameters that enhances the spread as well as control of an infection in a population.

The sensitivity index of the reproduction number of the Pneumonia model with respect to any parameter say p is given by:

$$\Im_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}$$

Given that

$$R_0 = \frac{\beta\theta\left(k + \mu\right)\left(\left(P_5 + \tau\right) P_4 + \gamma\left(P_5 + \psi\right)\right)}{\left(g + k + \mu\right) P_2 P_3 P_4 P_5}$$

$$= \frac{\left(\left(\phi\delta_H + \varepsilon + \mu + \tau\right)\left(\rho\varepsilon + \mu + \psi + \delta_M\right) + \gamma\left(\phi\delta_H + \varepsilon + \mu + \psi\right)\right)\left(k + \mu\right)\theta\beta}{\left(g + k + \mu\right)\left(\rho\epsilon + \mu + \psi + \delta_M\right)\left(\gamma + \tau + \delta_p + \mu\right)\left(\theta + \mu\right)\left(\phi\delta_H + \varepsilon + \mu\right)}$$

$$\Im_\beta^{R_0} = 1.0000$$

$$\Im_\theta^{R_0} = \frac{\mu}{\theta + \mu} = 0.0344$$

$$\Im_\mu^{R_0} = -\frac{1}{\left(g + k + \mu\right)\left(\epsilon\rho + \mu + \psi + \delta_M\right)} = -0.1517$$
$$\left(\gamma + \tau + \delta_p + \mu\right)\left(\theta + \mu\right)\left(\phi\delta_H + \varepsilon + \mu\right)^2$$

$$\Im_\psi^{R_0} = \frac{\psi\left(\left(\rho - 1\right)\epsilon - \phi\delta_H + \delta_M\right)\gamma}{\left(\rho\epsilon + \mu + \psi + \delta_M\right)\left(\begin{matrix}\epsilon^2\rho + \left(\left(\rho+1\right)\mu + \delta_H\phi\rho + \rho\tau + \delta_M + \psi + \gamma\right)\epsilon + \mu^2 \\ + \left(\phi\delta_H + \gamma + \psi + \tau + \delta_M\right)\mu + \left(\phi\delta_H + \gamma + \tau\right)\psi + \phi\left(\delta_M + \gamma\right)\delta_H + \tau\delta_M\end{matrix}\right)}$$

$= -0.04215$

$$\Im_{\delta_p}^{R_0} = -\frac{\delta_p}{\gamma + \tau + \delta_p + \mu} = -0.1190$$

$$\Im_{\delta_M}^{R_0} = -\frac{\gamma\left(\phi\delta_H + \epsilon + \mu + \psi\right)\delta_M}{\left(\rho\epsilon + \mu + \psi + \delta_M\right)\left(\begin{matrix}\epsilon^2\rho + \left(\left(\rho+1\right)\mu + \delta_H\phi\rho + \rho\tau + \delta_M + \psi + \gamma\right)\varepsilon + \mu^2 \\ + \left(\phi\delta_H + \gamma + \psi + \tau + \delta_M\right)\mu + \left(\phi\delta_H + \gamma + \tau\right)\psi + \phi\left(\delta_M + \gamma\right)\delta_H + \tau\delta_M\end{matrix}\right)}$$

$= -0.0306$

$$\Im_{\delta_H}^{R_0} = -\frac{\left(\varepsilon\rho\tau + \mu\tau + \psi\gamma + \tau\left(\delta_M + \psi\right)\right)\phi\delta_H}{\left(\begin{matrix}\varepsilon^2\rho + \left(\begin{matrix}\left(\rho+1\right)\mu + \delta_H\phi\rho + \\ \rho\tau + \delta_M + \psi + \gamma\end{matrix}\right)\epsilon + \mu^2 \\ + \left(\phi\delta_H + \gamma + \psi + \tau + \delta_M\right)\mu + \\ \phi\left(\delta_M + \psi + \gamma\right)\delta_H + \psi\gamma + \tau\left(\delta_M + \psi\right)\end{matrix}\right)}$$

$= -0.0165$

$$\Im_\varepsilon^{R_0} = -\frac{\left(\begin{matrix}\rho\left(\rho\tau + \gamma\right)\epsilon^2 + 2\left(\left(\tau + \gamma\right)\mu + \left(\phi\delta_H + \psi\right)\gamma + \tau\left(\delta_M + \psi\right)\right)\rho\epsilon \\ + \left(\gamma\rho + \tau\right)\mu^2 + \left(\begin{matrix}2\left(\phi\delta_H + \psi/2\right)\gamma\rho + \psi\gamma \\ + 2\tau\left(\delta_M + \psi\right)\end{matrix}\right)\mu \\ + \delta_H\phi\gamma\left(\phi\delta_H + \psi\right)\rho + \left(\psi\gamma + \tau\left(\delta_M + \psi\right)\right)\left(\delta_M + \psi\right)\end{matrix}\right)\epsilon}{\left(\phi\delta_H + \varepsilon + \mu\right)\left(\rho\varepsilon\epsilon + \mu + \psi + \delta_M\right)\left(\begin{matrix}\varepsilon^2\rho + \left(\left(\rho+1\right)\mu + \left(\phi\delta_H + \tau\right)\rho + \delta_M + \psi + \gamma\right)\epsilon \\ + \mu^2 + \left(\phi\delta_H + \gamma + \psi + \tau + \delta_M\right)\mu + \\ \left(\phi\delta_H + \psi\right)\gamma + \left(\delta_M + \psi\right)\left(\phi\delta_H + \tau\right)\end{matrix}\right)}$$

$= -0.6585$

$$\Im_{\rho}^{R_0} = -\frac{\epsilon\gamma\left(\phi\delta_H+\epsilon+\mu+\psi\right)\rho}{\left(\rho\epsilon+\mu+\psi+\delta_M\right)\left(\begin{array}{l}\epsilon^2\rho+\left((\rho+1)\mu+\delta_H\phi\rho+\rho\tau+\delta_M+\psi+\gamma\right)\epsilon+\mu^2\\+\left(\phi\delta_H+\gamma+\psi+\tau+\delta_M\right)\mu+\\\left(\phi\delta_H+\gamma+\tau\right)\psi+\phi\left(\delta_M+\gamma\right)\delta_H+\tau\delta_M\end{array}\right)}$$

$= -0.0766$

$$\Im_{g}^{R_0} = -\frac{g}{g+k+\mu} = -0.4107$$

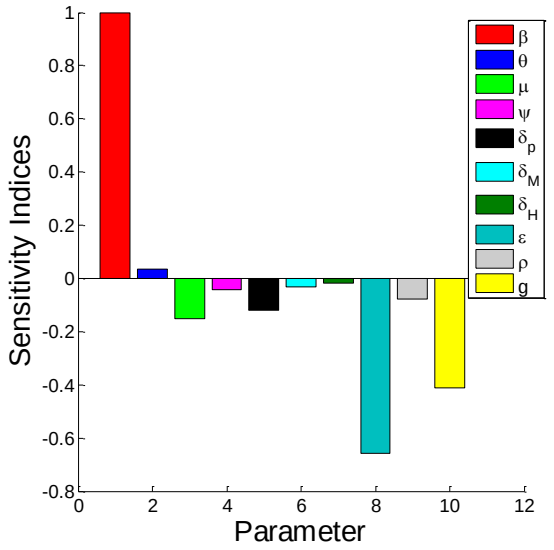

Figure 2 Bar chat of Pneumonia sensitivity Indices

The sensitivity analysis in this study revealed key factors affecting the spread of pneumonia. It showed that parameters such as the contact rate between susceptible and infected individuals, and the rate at which exposed individuals become infectious, have positive sensitivity indices. This means that increases in these factors raise the basic reproduction number(R_0) and promote disease transmission (Agbata et al., 2025). In practical terms, more frequent contact or faster progression to infection leads to more cases and a higher disease burden. On the other hand, some parameters exhibited negative sensitivity indices, indicating that their increase helps reduce pneumonia prevalence. These include vaccination rate, natural death rate, disease-induced death rate, and recovery rate. For example, increased vaccination lowers the number of susceptible individuals, while faster recovery shortens the infectious period. The results highlight the most important factors in controlling pneumonia and stress the importance of interventions like vaccination, early treatment, and reduced contact to limit disease spread.

Numerical Simulations

Numerical simulations confirmed the analytical findings of the pneumonia model. Increasing contact rates between susceptible and infected individuals raised infection prevalence, while higher vaccination coverage and hospitalization rates reduced disease spread and shortened the infectious period.

Table 2. Parameter values used for simulations

Parameter	Value	Source
β	0.007-0.6	Blyuss, (2016).
Λ	0.0413	Martínez *et al*, (2013)
μ	0.03	Yang, (2014).
θ	0.28	Estimated
ω	0.263	Martínez *et al*, (2013).
g	0.23	Yang (2014).
k	0.32	Estimated
γ	0.34	Martínez *et al*, (2013)
τ	0.39	Estimated
δ_p	0.1	Yang (2014).
ψ	0.32	Tilahun *et al*, (2017)
ρ	0.45	Estimated
ε	0.2	Tilahun *et al* , (2017)
δ_M	0.035	Martínez *et al*, (2013)
ϕ	0.1	Estimated
δ_H	0.057	Tilahun *et al*, (2017)

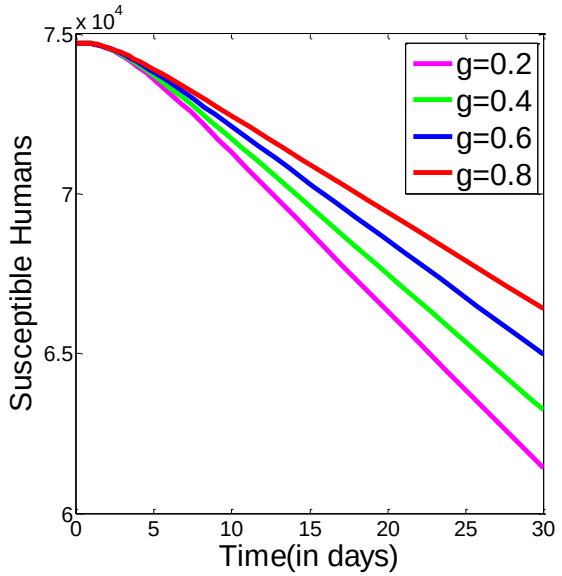

Figure 3a. Effect of varying g on the susceptible humans

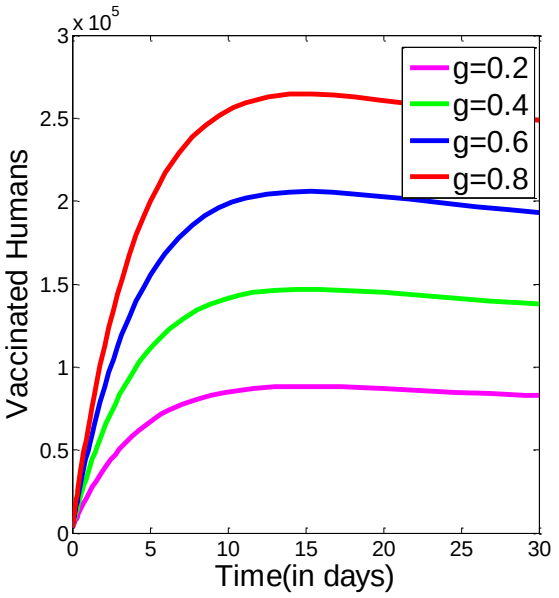

Figure 3b. Effect of varying g on the vaccinated class

From Figure 3a, it is evident that increasing the vaccination rate of susceptible individuals results in a gradual decline in the susceptible population. This suggests that effective vaccination strategies can significantly reduce pneumonia prevalence by lowering the number of people at risk of infection. Figure 3b further shows that as the vaccination rate increases, the population of vaccinated individuals grows over time, enhancing immunity within the community. This increase not only reduces the spread of the disease but also supports faster recovery and better overall health outcomes.

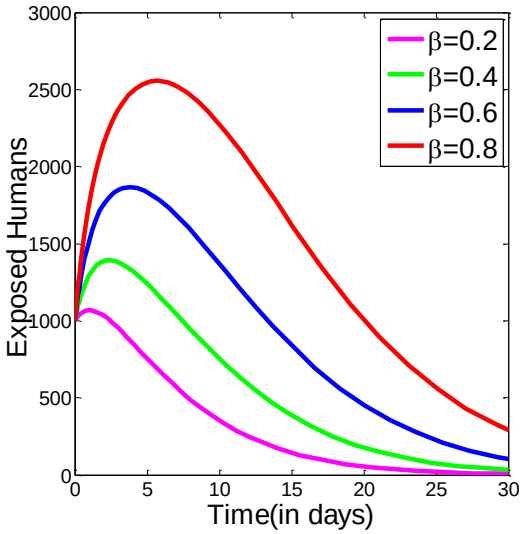

Figure 4a. Effect of varying β on the Exposed Humans

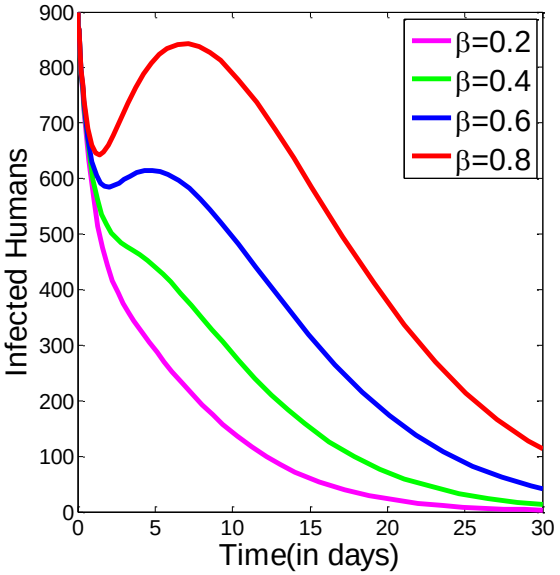

Figure 4b. Effect of varying β on the Infected Humans

rom Figure 4a, it is observed that an increase in the contact rate between susceptible and infected individuals initially causes a rise in the exposed population, as more people move from the susceptible class into exposure. However, this is followed by a decline in the exposed group, largely due to the rapid progression of pneumonia once individuals are exposed. Figure 4b shows that the number of infected individuals decreases over time as

they transition into the self-medication and hospitalized classes. This shift reflects the response to infection through treatment-seeking behavior. The reduction seen in the treatment classes is attributed to effective healthcare interventions, which promote recovery among infected individuals. As illustrated in Figures 5a and 5b, proper medical care and management significantly improve recovery rates and help reduce the overall disease burden.

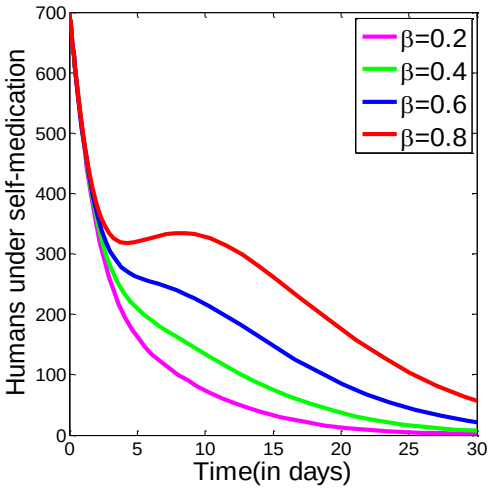

Figure 5a. Effect of varying β on humans

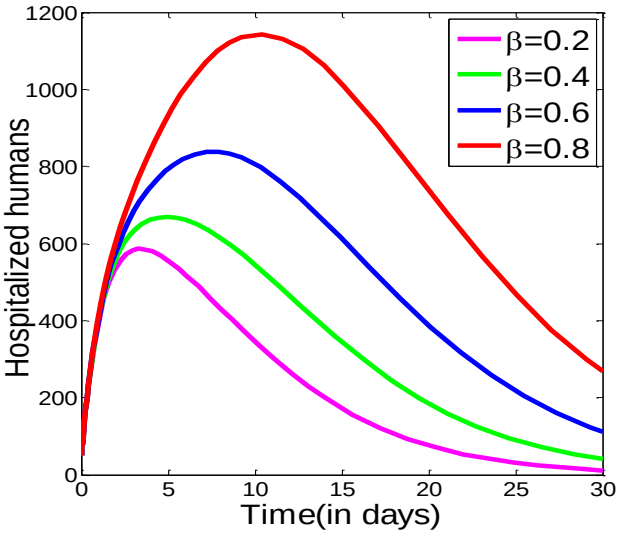

Figure 5b. Effect of varying β on the hospitalized humans under self-Medication

Figure 5a: This figure illustrates the effect of varying the parameter on humans under self-medication. As the parameter increases, the number of individuals practicing self-medication changes, indicating how access to or reliance on informal treatment influences disease progression. A higher value may initially increase this group, but over time it contributes to recovery or transition to proper medical care. **Figure 5b:** This figure shows the impact of varying the same parameter on hospitalized individuals. An increase in the parameter leads to changes in the hospitalized population, often reflecting improved access to healthcare services.

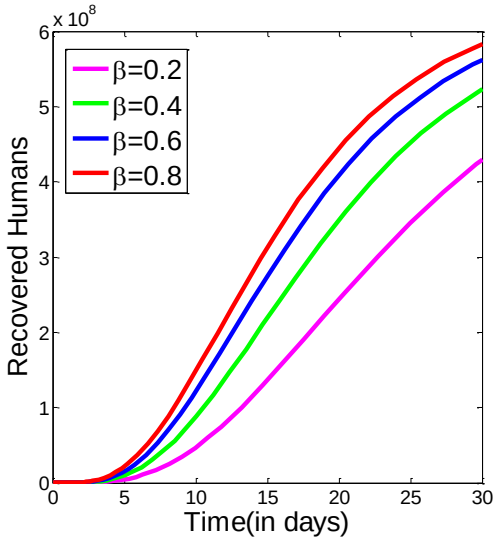

Figure 6a. Effect of varying β on the humans

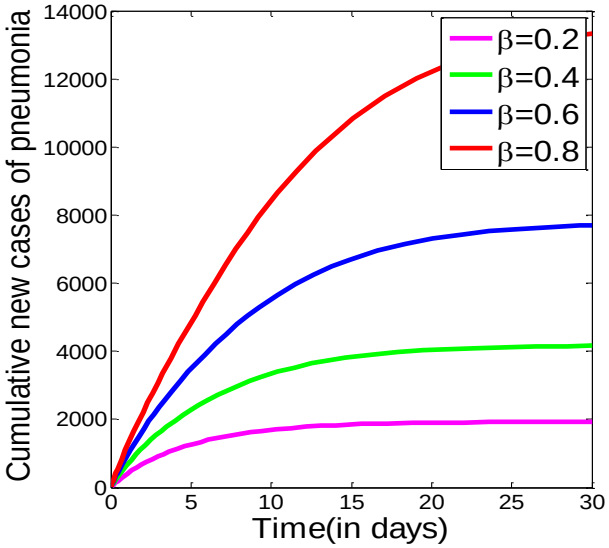

Figure 6b. Effect of varying β on the Cumulative recovered new cases of pneumonia

Figure 6a presents the dynamics of the recovery class in the human population. It is observed that the number of recovered individuals increases over time as a result of effective medical intervention. This improvement is mainly due to proper healthcare attention given to infected individuals in both the self-medication and hospitalized compartments. As treatment becomes more effective, more infected individuals successfully recover from pneumonia, leading to a continuous rise in the recovered population and a corresponding reduction in disease severity within the community. **Figure 6b** shows the effect of varying the contact rate between susceptible and infected individuals on the cumulative number of new pneumonia cases. The results indicate that an increase in the contact rate significantly raises the number of new infections over time. This occurs because higher interaction between susceptible and infected individuals enhances the probability of disease transmission, thereby accelerating the spread of pneumonia. Consequently, the cumulative case count increases steadily, highlighting the critical role of reducing contact rates in controlling the outbreak.

CONCLUSION

This study developed a comprehensive mathematical model to examine the transmission dynamics of pneumonia, explicitly incorporating vaccination and hospitalization as control measures. The computation of the basic reproduction number (R_0) provided critical insights into disease behavior, showing that the system achieves local asymptotic stability when $R_0 < 1$, global asymptotic stability when $R_0 \leq 1$, and maintains a stable endemic equilibrium when $R_0 > 1$.These findings indicate that pneumonia transmission can be effectively controlled if key intervention strategies are properly implemented, but may persist when control measures are insufficient or improperly applied. The sensitivity analysis identified key factors influencing pneumonia transmission. Higher contact rates between susceptible and infected individuals, as well as faster progression from exposure to active infection, were found to increase disease spread. Conversely, higher vaccination coverage, faster recovery, and increased hospitalization effectively reduced transmission and lowered the basic reproduction number. These findings highlight the critical role of preventive measures and timely treatment in controlling the spread of pneumonia. Numerical simulations complemented these analytical results, visually demonstrating the impact of varying key parameters. The simulations confirmed that reducing contact between susceptible and infected individuals, increasing vaccination coverage among susceptibles, and improving hospitalization of infected individuals substantially decrease infection prevalence and shorten the duration of disease within the population. The comparative analysis also emphasized the detrimental effects of self-medication, which prolongs infection and allows sustained transmission, whereas hospital-based treatment and vaccination consistently reduced disease burden.

The study showed that effective pneumonia control requires a multifaceted approach**,** combining vaccination programs, improved access to hospital care, and public awareness campaigns to discourage unsafe self-medication. Based on the findings of this study, several measures are recommended to reduce the burden of pneumonia in the population. The government should enhance its investment in healthcare infrastructure by establishing and equipping treatment centers to ensure timely and effective care for individuals affected by pneumonia. In addition, efforts should be intensified to provide accessible and potent vaccines, as widespread immunization is essential for lowering susceptibility and preventing disease spread. Public awareness campaigns are also crucial to educate communities about the risks associated with self-medication and to promote the benefits of seeking professional medical care. Together, these strategies can help reduce infection rates, improve recovery outcomes, and limit the overall impact of pneumonia on society. A key direction for future research is the incorporation of time-dependent optimal control strategies into the pneumonia model to evaluate dynamic interventions and more effectively reduce disease burden.

Conflict of Interest

The authors declare there is no conflict of interest regarding publication of this article

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