



Modeling the Mathematical Transmission of a Pneumonia Epidemic Model with Awareness

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ABSTRACT: A new model for the transmission of a pneumonia epidemic, considering awareness and a mass action incidence function, is presented. Stability analyses for disease-free and endemic equilibrium points are conducted. The Basic Reproduction Number (R_0) for pneumonia with awareness is defined and analyzed, showing stability when ($R_0 < 1$) and transitioning to an endemic state when ($R_0 > 1$). Additionally, a special case is highlighted where the Basic Reproduction Number (R_0^*) (without awareness) is greater than (R_0) Basic Reproduction Number with awareness, i.e. ($R_0^* = 1.0965 > R_0 = 0.8772$). Furthermore, a numerical simulation is provided to depict how awareness influences the dynamic management of the disease. The results underscore the crucial role of awareness in educating the public about infection risks, ultimately contributing to a decrease in the health burden by mitigating the epidemic peak.

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Pneumonia is an infection of the lungs that is caused by bacteria, viruses, fungi or parasites. It is most dangerous for older adults, babies and people with other diseases or impaired immune systems (Fekadu *et al.*, 2023 and Getachew *et al.*, 2017). Pneumococcal is spread through contact with people who are ill or who carry the bacteria in their throat. One can get pneumococcal pneumonia from respiratory droplets from the nose or mouth of an infected person. It is common for people, especially children, to carry the

bacteria in their throats without being sick. After a person is infected and diagnosed with pneumonia, he should be on medication for a particular period of time; the infection is contagious for 10 to 14 days after the infected person stops getting treatment (Sayed *et al.*, 2022 and (WHO)a, 2021). When a person breathes pneumonia-causing germs into his lungs and his body's immune system cannot prevent its entry, the organisms settle in small air sacs called alveoli and continue multiplying. A lung infection caused by a

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compromised immune system can also lead to pneumonia which is also more prone to individuals with old age or respiratory problems than healthy people (Aleem *et al.*, 2021, Dipo *et al.*, 2023, Fekadu *et al.*, 2023 and Marcus *et al.*, 2021). As the body sends white blood cells to attack the infection, the sacs become filled with fluid and pus - causing pneumonia. Pneumonia has Bacterial, Viral, Fungal, and other primary causes. Other substances that caused pneumonia are smoke, abuse alcohol, those that have other medical conditions, such as chronic obstructive pulmonary disease (COPD), emphysema, asthma, or HIV/AIDS (Marcus *et al.*, 2021). This pneumonia is not common but may occur among those with weak immunity due to AIDS, immunosuppressive drugs and other medical problems (Dipo *et al.*, 2023 and (WHO) b, 2022). Pneumonia exists in two types: typical and atypical. Typical pneumonia is marked by symptoms such as cough, fever, dyspnea, sudden chills, pleuritic chest pain, with no constitutional symptoms. In contrast, atypical pneumonia lacks cough, presents milder symptoms like reduced fever and dyspnea, and includes constitutional symptoms like myalgia and rhinitis. Physically, typical pneumonia exhibits more respiratory distress, higher fever, and consolidation findings, unlike atypical pneumonia. (Das *et al.*, 2019, Etbaigha *et al.*, 2018 and Fekadu *et al.*, 2023). Many researchers have been done using mathematical models to understand the dynamical spread of disease (Goel *et al.*, 2020; 2019a; 2019b, Musibau *et al.*, 2022, Otunuga 2018, Otunuga 2017 and Qiuz 2008). To understand the dynamic spread of pneumonia, numerous researchers have dedicated efforts to developing mathematical models for this infectious disease. In the study conducted by Tilahun 2019, seven compartmental mathematical models were examined. The research asserts that to destabilize the endemic equilibrium and transition it to a disease-free equilibrium, the implementation of high efficacy treatment and vaccination programs as an optimal control strategy is imperative. The findings further demonstrate that a decrease in the contact rate related to either pneumonia or meningitis significantly contributes to the effective control of co-infection of pneumonia and meningitis at the population level. Marcus and Newton, investigated the dynamics of pneumonia disease using a deterministic SEIR model. They found that the pneumonia-free equilibrium is locally asymptotically stable when the $R_0 < 1$, and the pneumonia endemic equilibrium is globally asymptotically stable in the invariant region whenever $R_0 > 1$. The research also conducted sensitivity analysis, revealing that transmission rates and the rates at which exposed individuals become infectious are the most sensitive parameters. Center manifold theory

was applied to detect the presence of forward bifurcation in the model. While extensive research has focused on mathematical analysis, modeling, and optimal control of pneumonia disease in society (Dipo *et al.*, 2023), there remains a notable gap in understanding the impact of awareness on the dynamic spread of pneumonia. This paper addresses this gap by introducing an awareness parameter into the compartmental mathematical model presented. The significance of awareness in mitigating pneumonia's endemicity is emphasized, highlighting the crucial role of societal awareness in curbing the dynamic spread of the pneumonia infectious disease.

The study is organized as follows: Section 2 introduces a SEITR model for pneumonia with awareness. Section 3 presents the model analysis, while Section 4 covers the numerical analysis. The study concludes with the discussion and conclusions in Section 5.

Deterministic Model Formulation and Description:

The total population of human $N(t)$ is been subdivided into five compartments. That is, Susceptible persons $S(t)$, Exposed persons $E(t)$, Infected persons $I(t)$, Treated persons $T(t)$ and the Recovered persons $R(t)$, Therefore;

$$N(t) = S(t) + E(t) + I(t) + T(t) + R(t) \quad (1)$$

The susceptible population increases with new births or immigration at the rate π . Additionally, it grows when individuals from the recovered compartment transition back to the susceptible compartment due to treatment waning or loss of treatment immunity at the rate ν . Conversely, the population decreases with new infections at the rate λ and due to natural deaths at the rate μ . Hence;

$$\pi - (1 - \omega)\lambda S - \mu S + \nu R \quad (2)$$

The exposed compartment population rises when a susceptible individual, aware about pneumonia, contacts an infected person, with a force of infection denoted by λ . Although non-infectious at this stage, the population decreases as exposed individuals' transition to the infectious stage at a given rate κ . Moreover, the exposed population undergoes reduction due to natural death at a specified rate and natural immunity at a designated rate μ and τ_1 .

Hence;

$$(1 - \omega)\lambda S - (\kappa + \mu + \tau_1)E \quad (3)$$

The infected compartment population increases as individuals move from the Exposed class to the

Infected class at a given rate κ . This population is subsequently reduced by natural death and death due to the disease at rates μ and δ . Furthermore, it undergoes additional reduction through natural immunity and treatment of infected individuals at rates τ_2 and θ respectively.

$$\kappa E - (\mu - \delta + \tau_2 + \theta)I \tag{4}$$

The population of the treated individuals increased due to treatment of infected individuals at the rate θ . It decreases each time an individual recovers and is moved to the recovered class at the rate γ , the population is further reduced by the natural death at the rate μ . Thus,

$$\theta I - (\mu + \gamma)T \tag{5}$$

The population of the recover individuals are increased due to natural immunity of exposed and infected individuals at the rate τ_1 and τ_2 respectively. The recovered class also increased due to individuals who have been treated and fully recovered at the rate γ , it later reduced due individuals that loss treatment immunity and natural death at the rate ν and μ respectively. Thus,

$$\tau_1 E + \tau_2 I + \gamma T - (\mu - \nu)R \tag{6}$$

The force of infection that is associated to the disease is denoted by λ and is given by mass action function;

$$\lambda = \frac{\beta I}{N} \tag{7}$$

Putting equations (2) to (7) seven above together, we obtain the system of non linear differential equation with mass action function:

$$\begin{aligned} S' &= \pi - (1 - \omega)\lambda S - \mu S + \nu R \\ E' &= (1 - \omega)\lambda S - (\kappa + \mu + \tau_1)E \\ I' &= \kappa E - (\mu - \delta + \tau_2 + \theta)I \end{aligned} \tag{8}$$

$$\begin{aligned} T' &= \theta I - (\mu + \gamma)T \\ R' &= \tau_1 E + \tau_2 I + \gamma T - (\mu - \nu)R \\ \lambda &= \frac{\beta I}{N} \end{aligned} \tag{9}$$

For simplicity, equations (8) becomes

$$\begin{aligned} S' &= \pi - (1 - \omega)\lambda S - \mu S + \nu R \\ E' &= (1 - \omega)\lambda S - K_1 E \\ I' &= \kappa E - K_2 I \\ T' &= \theta I - K_3 T \\ R' &= \tau_1 E + \tau_2 I + \gamma T - K_4 R \end{aligned} \tag{10}$$

Where

$$\begin{aligned} K_1 &= \kappa + \mu + \tau_1, K_2 = \mu + \delta + \tau_2 + \theta, \\ K_3 &= \mu + \gamma, K_4 = \mu - \nu \end{aligned}$$

Table 1: Description of variables

Variables	Description
S	Susceptible
E	Exposed
I	Infected
T	Treated
R	Recovered

Table 2: Descriptions of Parameters and Values used for Simulations

Parameter	Description	Value
π	Recruitment Rate	50
ω	Awareness	0.2
θ	Treatment rate	0.3
λ	Force of Infection	
μ	Natural Death	0.03
ν	Immunity loss	0.01
κ	Progression to Infected	0.01
δ	Death due to Infection	0.02
τ_1	Natural recovery of Exposed	0.6
τ_2	Natural recovery of Infected	0.6
γ	Recovery of Treated	0.2
β	Effective contact rate	0.04

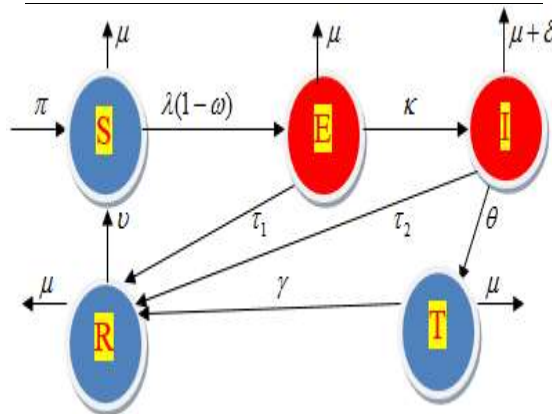


Fig. 1. Model Flowchart

Model Analysis: Since the model (9) monitors the human populations, all its associated parameters are

non-negative. Therefore, the following non-negativity result holds.

Theorem 3.1: For the model of systems of equation to be epidemiologically meaningful and mathematically well posed, we prove that all variables are non-negative $\forall t > 0$.

Let:

$$\{S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, T(0) \geq 0, R(0) \geq 0\}$$

Then, the solution:

$\{S(t), E(t), I(t), T(t), R(t)\}$ of the model system equation (8) are positive $\forall t \geq 0$.

Proof:

In order to prove the theorem (3.1), the equations of the system (8) were used. From the first equation of the model (8):

$$S' = \pi - (1 - \omega)\lambda S - \mu S + \nu R \quad (10)$$

It then follows that:

$$\frac{dS}{dt} \geq -\mu S$$

Thus,

$\frac{dS}{dt} + \mu S \geq 0$ is the first order homogeneous differential equation.

$$\text{I.F.} = e^{\int \mu dt} = e^{\mu t} \quad (11)$$

If we multiply both sides by IF, we get

$$e^{\mu t} \frac{dS}{dt} + \mu S e^{\mu t} \geq 0$$

It then follows that:

$$d(S e^{\mu t}) \geq 0 dt \quad (12)$$

Integrating on both sides gives:

$$S e^{\mu t} \geq C$$

Where C is a constant of the integration, it follows that:

$$S(t) \geq C e^{-\mu t} \quad (13)$$

Applying the initial condition that, when $t = 0, S(t) = S(0)$, we have:

$$S(0) \geq C$$

Hence:

$$S(t) \geq S(0) e^{-\mu t} \quad (14)$$

Since $\mu > 0$ and $S(0) \geq 0$, then:

$$S(t) \geq 0, \text{ if } t = 0 \text{ and } t \rightarrow \infty$$

Therefore:

$$S(t) \geq 0 \quad \forall t \geq 0.$$

Similarly, it can be shown that $E \geq 0, I \geq 0, T \geq 0$, and $R \geq 0 \quad \forall t \geq 0$.

Therefore, the model formulated is mathematically and epidemiologically well posed.

Lemma1.

The closed set

$$D = \left\{ (S, E, I, T, R) \in R_+^5 : S + E + I + T + R \leq \frac{\pi}{\mu} \right\}$$

is positively invariant.

Proof. By adding together all the equations of the model (8) gives,

$$dN/dt = \pi - \mu N - \delta I$$

At disease free, in view of the fact that $dN/dt \leq \pi - \mu N$, then, $dN/dt \leq 0$ if $N \geq \mu N$.

Thus, using a standard comparison theorem, $N \leq N(0) e^{-\mu t} + (\pi / \mu)(1 - e^{-\mu t})$

it is clear that $N(t) \leq \frac{\pi}{\mu}$ if $N(0) \leq \frac{\pi}{\mu}$ for all $t > 0$.

The solution with initial conditions in D remains in D for all $t > 0$ (i.e., the ω -limits sets of the system in (1) are contained in D). Hence, the model is epidemiologically and mathematically well-posed in D .

The Basic Reproduction Number, (R_0): The disease free Equilibrium (DFE) of the model (9) is given as;

$$E_0 = (S^*, E^*, I^*, T^*, R^*) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0 \right)$$

The Basic Reproduction Number, R_0 , in an epidemiological study is used to show how an infection is transmissible. It is the average number of

new infections which infected person can transmit in a transmittable period(Adesanya *et al*, 2016, Adewale *et al*, 2015a; 2015b; 2015c; 2016, Ajao *et al*, 2023, Akinola *et al*, 2021 and Akinwumi *et al*, 2021).

The Basic Reproduction Number is calculated using the method of next generation matrix and $R_0 = \rho(FV^{-1})$, where F is the new infection terms V is other remaining transfer terms. Given is the matrices F and V below,

$$F_1 = \begin{pmatrix} 0 & \frac{\beta\pi(1-\omega)}{\mu} \\ 0 & 0 \end{pmatrix} \quad (15)$$

$$V_1 = \begin{pmatrix} K_1 & 0 \\ -\kappa & K_2 \end{pmatrix} \quad (16)$$

Therefore,

$$R_0 = \frac{\beta\pi\kappa(1-\omega)}{\mu K_1 K_2}$$

It is easy to predict whether an infection will spread exponentially or die after some time or remain constant without spreading further, as measured by the value of the reproduction number, when $R_0 < 1$, the disease will dies off because every infected person will transmit the disease to less than one person in the transmittable period. When $R_0 = 1$, the disease will become endemic and will stay with each infected person transmitting to one new person. When $R_0 > 1$, a disease will spread and the infected people will grow exponentially which will lead to a pandemic as is seen in corona virus.

Lemma 2; Special case of basic of Basic Reproduction Number when Awareness $\omega = 0$

The Basic Reproduction Number (R_0^) Without Awareness:* The Basic Reproduction Number in the absence of awareness i.e. $\omega = 0$ is calculated using the method of next generation matrix and $R_0 = \rho(FV^{-1})$, where F is the new infection terms and V is other remaining transfer terms. Given is the matrices F and V in equations 17 and 18,

$$F_2 = \begin{pmatrix} 0 & \frac{\beta\pi}{\mu} \\ 0 & 0 \end{pmatrix} \quad (17)$$

$$V_2 = \begin{pmatrix} K_1 & 0 \\ -\kappa & K_2 \end{pmatrix} \quad (18)$$

Therefore,

$$R_0^* = \frac{\beta\pi\kappa}{\mu K_1 K_2}$$

It is very obvious here that, the Basic Reproduction Number R_0^* (without awareness) is greater than the Basic Reproduction Number R_0 (with awareness) i.e. $R_0^* > R_0$. This shows that awareness of the disease is an important tool that plays a major role in the dynamical curtailing or total eradication of a disease.

Local Stability of Disease Free Equilibrium

Theorem 3.2: The disease free equilibrium of the model equation (9) is locally stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: To determine the local stability of E_0 , the Jacobian matrix below is computed corresponding to Disease Free Equilibrium E_0 . Considering the stability of the disease free equilibrium at

$$\left(\frac{\pi}{\mu}, 0, 0, 0, 0 \right)$$

$$J(E_0) = \begin{pmatrix} -\mu & 0 & -(1-\omega)\frac{\pi}{\mu} & 0 & \nu \\ 0 & -K_1 & (1-\omega)\frac{\pi}{\mu} & 0 & 0 \\ 0 & \kappa & -K_2 & 0 & 0 \\ 0 & 0 & \theta & -K_3 & 0 \\ 0 & \tau_1 & \tau_2 & \gamma & K_4 \end{pmatrix} \quad (19)$$

The eigenvalues are $-\mu$, $-K_3$ and $-K_4$ and the remaining eigenvalues can be determined from the characteristics equation of the remaining sub-matrix given as;

$$\begin{vmatrix} -K_1 & (1-\omega)\frac{\pi}{\mu} \\ \kappa & -K_2 \end{vmatrix} = 0 \quad (20)$$

The characteristic equation from equation (20) is given below;

$$\lambda^2 + (K_1 + K_2)\lambda + \frac{\beta\pi\kappa\omega - \beta\pi\kappa + \mu K_1 K_2}{\mu} = 0$$

Let;

$$b_2 = 1, b_1 = (K_1 + K_2)$$

$$\text{and } b_0 = \frac{\beta\pi\kappa\omega - \beta\pi\kappa + \mu K_1 K_2}{\mu}$$

If $b_1 > 0$ and $b_0 > 0$, then, we have

$$\frac{\beta\pi\kappa\omega - \beta\pi\kappa}{\mu} > -\frac{\mu K_1 K_2}{\mu} \tag{21}$$

$$\text{Hence, } \frac{\beta\pi\kappa(1 - \omega)}{\mu K_1 K_2} < 1$$

Hence, the disease equilibrium point is locally asymptotically stable whenever $R_o < 1$. This theorem implies that if the initial sizes of the sub-populations of the model are in the basin of attraction of the disease-free equilibrium, the disease is controllable provided $R_o < 1$

Global stability of disease-free equilibrium:

We study the global stability of equilibrium without disease for a special case when $\nu = 0$ and we implement the approach of (Ajao *et al*, 2023) then the equations of the model may be rewritten in the form.

;

$$\frac{dM}{dt} = F(M, I)$$

$$\frac{dI}{dt} = G(M, I)$$

With $G(P, 0) = 0$, where $P \in R^3$ represents the uninfected classes (S, T, R) and $I \in R^2$ represents the infected classes (E, I) . Also, $E_o = (M^*, 0)$ denotes the disease-free equilibrium of the model. The two conditions (H1) and (H2) stated below must be satisfied for the model to be globally stable

(H1): For $\frac{dM}{dt} = F(M, 0)$, M^* is globally asymptotically stable

(H2):

$$G(M, I) = AI - \hat{G}(M, I), \quad \hat{G}(M, I) \geq 0 \quad \text{for } (M, I) \in D$$

Where $A = D_I G(M^*, 0)$ is an M-matrix (the off-diagonal elements of A are non-negative) and D is the

region is the feasible region where the model is biologically meaningful. If (H1) and (H2) are satisfied, then the following theorem holds;

Theorem 3.3: The disease-free equilibrium $E_o = (M^*, 0)$ is a globally asymptotically stable equilibrium of the model if $R_o < 1$ and that the conditions (H1) and (H2) are satisfied

Proof:

Now $M = (S, R)$ and $I = (I_s, I_p, L, T)$

$$F(M, 0) = \begin{pmatrix} \pi - \mu S \\ 0 \end{pmatrix} \tag{22}$$

And

$$A = \begin{pmatrix} K_1 & (1 - \omega)\beta \\ \kappa & K_2 \end{pmatrix} \tag{23}$$

$$\text{Then } \hat{G}(M, I) = \begin{pmatrix} (1 - \omega)\beta \left(1 - \frac{S}{N}\right) \\ 0 \end{pmatrix}$$

Since $0 \leq \varepsilon \leq 1$, clearly $\hat{G}(M, I) \geq 0$,

$$E_o = \left(\frac{\pi}{\mu}, 0\right) \text{ is a globally asymptotic stable}$$

equilibrium of the model equations. Hence, the two conditions above are satisfied. Therefore, the disease-free equilibrium is globally asymptotically stable. This implies biologically that the elimination of pneumonia is independent of the initial sizes of the sub-populations whenever the basic production number is less than one.

Sensitivity Analysis: Sensitivity analysis is used to investigate or determine how sensitive the threshold amount is to the basic reproduction number with respect to its parameters, through this investigation, we will know which of the parameters causes the highest decrease in the basic reproduction number and also which parameters have the greatest effect on basic reproduction number (Olopade *et al.*, 2016; 2017, 2021a; 2021b; and 2022). Intervention strategies must be targeted to find the most effective disease control. The analysis tells us how important each variable is in disease transmission. The normalized forward sensitivity index of the reproduction number with respect to its parameters will be computed below.

Definition: supposing a variable ‘P’ depends differentially on a parameter ‘w’, then, normalized

forward sensitivity index of ‘p’ with respect to ‘w’ is denoted by X_p , which is defined as

$$X_p = \frac{p}{w} \frac{\partial w}{\partial p}$$

As we have explicit for R_0 , we derive an analytical expression for the sensitivity of R_0 as

$$X_w^{R_0} = \frac{dR_0}{dw} \times \frac{w}{R_0} \quad (23)$$

For each parameter involved in R_0 , the Sensitivity Analysis is therefore calculated. To each parameter involved in Basic Reproduction Numbers (R_0), the sensitivity indices of R_0 with respect to each of the parameter are calculated as follows:

Table 3.Sensitivity Index of Parameters in (R_0)

Parameter	Sensitivity Expression	Sensitivity Value
π	1	1
τ_1	$-\frac{\tau_1}{K_1}$	-0.9376
τ_2	$-\frac{\tau_2}{K_2}$	-0.6316
μ	$-\frac{(3\mu^2 + 2(\delta + \kappa + \theta + \tau_1 + \tau_2)\mu + (\kappa + \tau_1)K_2)}{K_1K_2}$	-1.0785
κ	$\frac{\beta\pi(\omega - 1) - \mu\kappa K_2}{\mu K_1 K_2}$	0.9843
ω	$\frac{\omega}{\omega - 1}$	-0.2500
β	1	1
δ	$-\frac{\delta}{K_2}$	-0.0211
θ	$-\frac{\theta}{K_2}$	-0.3158

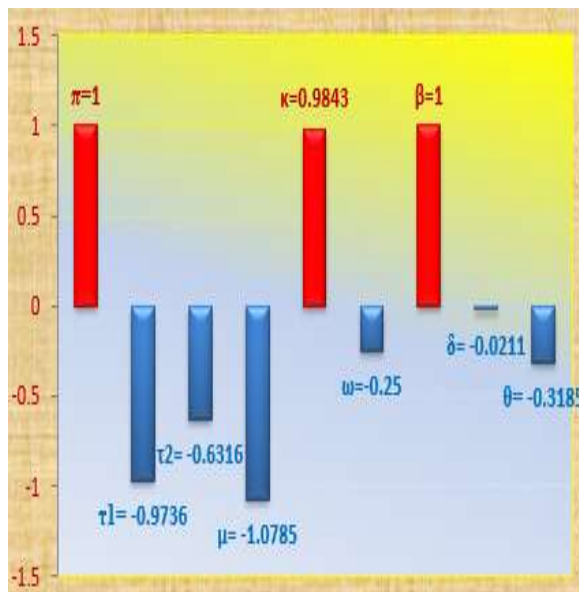


Fig. 2. Sensitivity Chart

Numerical Simulation: To authenticate the theoretical calculations of the model (9), the numerical simulations of the model are carried out by Elzaki Decomposition Method (Akinola *et al*, 2017) using a set of estimated limit standards specified as shown in Table (2) through preliminary assessment $S = 3000, E = 2500, I = 1000, T = 250, R = 250$

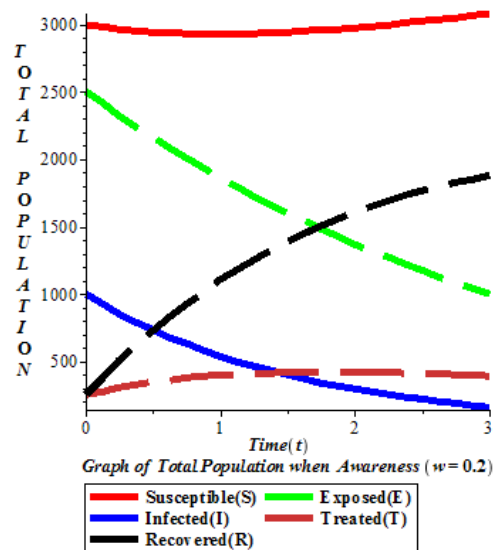


Fig. 3. Graph of Total population

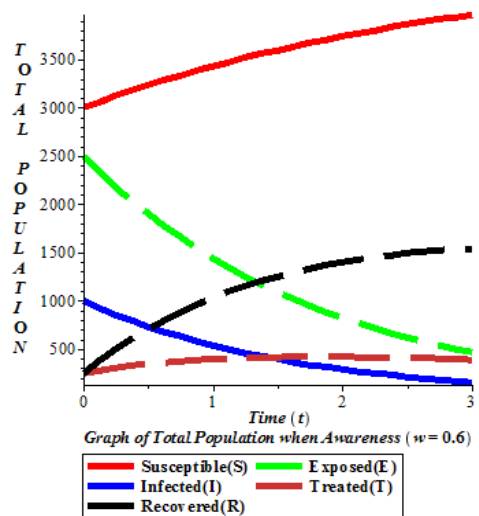


Fig. 4 Graph of Total population

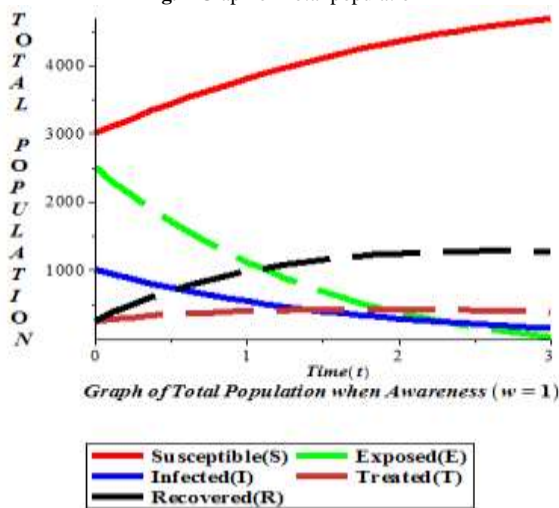


Fig. 5 Graph of Total population

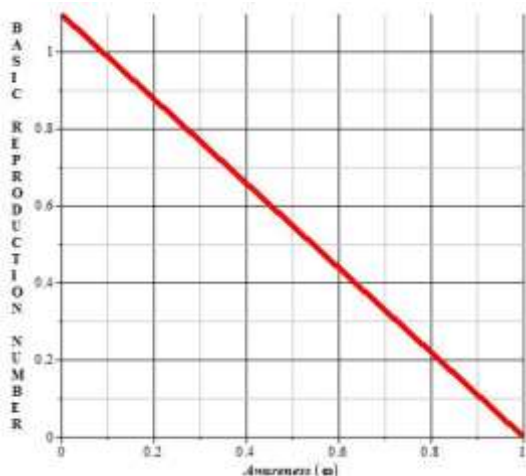


Fig. 6. Graph of Basic Reproduction Number Against the Awareness

This study has formulated five new nonlinear differential equations to investigate the impact of disease awareness on the dynamic spread of pneumonia. These equations aim to assess the dynamic patterns of illness propagation in the public, emphasizing the importance of awareness in controlling the Pneumonia epidemic model. The mathematical representation of this research was implemented and verified using the Maple program with the differential transformation method.

As shown in Table 3 and Figure 2, Sensitivity analysis is a statistical technique used to assess how variations in the input parameters of a model affect its output. Sensitivity analysis with positive and negative values helps analysts understand the direction and strength of relationships within a model, providing insights into which factors are more influential and how changes in those factors impact the overall outcomes. Effective transmission rate β and recruitment π are the most sensitive parameters in this work. Medical professionals must focus on positive index parameters, particularly those that have a strong impact on the fundamental duplicate amount of (R_0) i.e. Effective diffusion rate β and recruitment π to maintain a disease-free environment. In Figure 3, the influence of awareness is depicted when the awareness parameter $\omega = 0.2$. The graphical representation illustrates a notable decline in the counts of both exposed and infected individuals within the population. The introduction of awareness to the model results in a substantial reduction in the exposed class, specifically decreasing from an initial count of 2500 to a new count of 1000. This observation underscores the significant impact that incorporating awareness can have on mitigating the number of individuals in the exposed category, indicative of the positive effect of awareness in curtailing the spread of the modeled epidemic. In Figure 4, the pronounced effect of disease awareness on exposed and infected individuals is evident. The graphical representation vividly illustrates that as the level of awareness increases, there is a significant reduction in the count of exposed and infected individuals within the population. Specifically, when the awareness parameter is set at a certain value, denoted as $\omega = 0.6$, it is observed that the population of exposed and infected individuals decreases notably from an initial count of 2500 to a substantially lower count of 400. This outcome underscores the substantial impact of heightened awareness in diminishing the number of individuals concurrently in the exposed and infected states, highlighting the effectiveness of awareness in mitigating the spread of the modeled disease.

In Figure 5, the visualization demonstrates that a substantial value of the awareness rate can result in the effective containment of pneumonia diseases. Specifically, when the awareness parameter reaches a sufficiently large value, denoted as $\omega = 1$, there is a remarkable decrease in the count of individuals within the exposed class. The numerical representation showcases a reduction from an initial count of 2500 to a complete elimination, reaching zero. This compelling observation underscores the pivotal role of awareness in effectively curtailing the progression of pneumonia epidemic diseases, highlighting its significant impact in achieving containment and prevention.

Figure 6 provides a comprehensive depiction of the dynamic interplay between awareness and the threshold reproduction number. When there is no awareness, indicated by $\omega = 0$ ($R_0^* = 1.0965 > 1$). However, as awareness increases, denoted by $\omega = 0.2$, there is a noticeable reduction in the threshold reproduction number, specifically decreasing from 1.0965 to 0.8772. This reduction in the threshold reproduction number signifies a consequential decrease in the endemic level within the community. The illustration effectively underscores the intricate relationship between awareness and the potential for disease endemicity, emphasizing the role of heightened awareness in mitigating and controlling the spread of the Pneumonia epidemic.

In conclusion, this research employs qualitative analysis to establish the well-posedness and uniqueness of solutions for the model. The computation of the basic reproduction number (R_0) using the next generation matrix, along with the examination of model equilibrium stability, indicates the existence of two equilibrium points for each model. The disease-free equilibrium is found to be locally asymptotically stable when ($R_0 < 1$) and unstable otherwise, paving the way for the presence of the endemic equilibrium whenever ($R_0 > 1$). Furthermore, sensitivity analysis of parameters within the basic reproduction number unveils that the effective contact rate and population recruitment (via birth or immigration) are the key factors contributing to an increase in the basic reproduction number, subsequently facilitating the further spread of the pneumonia disease in the environment. Moreover, the research underscores the pivotal role of disease awareness in controlling the pneumonia epidemic model.

Conclusion: It is noted that heightened awareness significantly diminishes the number of infected individuals and curtails the spread of the disease within the community. This observation highlights the crucial impact of awareness in mitigating and managing the dynamics of the modeled pneumonia epidemic.

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