

A Mathematical Modeling of Transmission Dynamics of Pneumonia Infection with intervention incorporating International Travellers Screening

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Abstract

Pneumonia is one of the global pandemics which have afflicted humanity in various aspects of life among the young children and elderly one. In some developed countries like Nigeria and Kenya, it is discovered to be a highly airborne transmitted disease which leads to a high mortality rate of human beings. It has been discovered by the World Health Organization (WHO) that almost 16% deaths of children are due to pneumonia infection in most regions like South Asia and Sub-Sahara Africa. There exist several mathematical models which aimed at mitigating the transmission of Pneumonia infection. This study takes into consideration the screening of international travellers with intervention seeking to minimize the rate of the circulation of pneumonia infection led by the movement of human beings. The originality of this research lies on International Travellers Screening as a preventive and control mechanism of pneumonia transmission. We create and examine the pneumonia disease dynamics from a mathematical view using SXEIT model. The model includes five non-linear compartments namely; "Susceptible (S), Screened (X), Exposed (E), Infected (I) and Treated (T)". Also, we develop the Basic Reproduction number (R_0) in the study and examine the existence of all the equilibrium points; the Endemic Equilibrium and Disease-Free Equilibrium, and analyze their stabilities. The model uses the preferred model system of differential equations which is subjected to numerical simulation using Matlab application. The research utilizes data collected from surveys of other writers and related works to parameterize and validate the model. The findings of this study are recommended to the Ministry of Health and World Health Organization (WHO) who may use them to strategize new ways of preventing the spread of Pneumonia infection.

Keywords: Pneumonia Transmission Dynamics, Mathematical Modeling, International Travellers Screening

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1. Introduction

Pneumonia being one of the lungs infection diseases is caused by bacteria and viruses. It is known to be characterized by alveoli's inflammation in the lungs. The mortality and morbidity of elderly one and children in developed countries like Kenya and Nigeria are mostly caused by pneumonia infection. The common symptoms associated with infected individuals with pneumonia are; "cough, fast breathing and shortness of breath, fever, muscle pain, chest pain, chills, sweating, nausea and vomiting, fatigue, fast heartbeat, headache, feeling very weak, diarrhoea and dusky or purplish skin colour (cyanosis) from poorly oxygenated blood" (Huang S. et al, 2005). The data on carriage among youths and adults is discovered to be minority (Regev-Yochay, et al., 2004) and most researches advocated that the source of pneumonia transmission to adults in families are children (Leino et al., 2001).

Compartment models are tools used to approximate many disease outbreaks and spread using minimum computational resources. They make use of differential equations in solving and showing how the different compartments in the system change with time and how those changes affect the dynamics of the disease. The population of each model is divided into different sub-groups depending on the model. For instance, the *SEIRD* model has five compartments which divided the total population. It is represented as "the Susceptible class (S), the Exposed class (E), the Infected class (I), the Recovered class (R), and the Dead class (D)". Each individual moves to the other compartment depending on the changes in their infection. According to Brauer et al. (2012), the future of pneumonia dynamics based on its derivative changes in the values of infection and recovery rate is forecast with the use of the *SIR* models. The mathematical models associated with infectious diseases like Hepatitis, Measles, coronavirus, pneumonia and so on, have been used successfully as dominant tools in providing essential insights into the transmission changes in a host population, the understanding of epidemiological procedures, the course of infection, and creation or execution of disease control programs (Moghadas, 2006).

In the research done by Eddy et al. (2005), they noted that the difficulty in breathing of individuals with pneumonia infection are caused by the excessive fluid in their lungs, and this extremely disturbs the youths, the elderly one as well as vulnerable individuals whose immune system have been compromised. In 2019, the World Health Organization (WHO) reported that many families in the world are affected by pneumonia infection, and it has become the single prevalent infectious disease which brings 741,180 under the age of 5 children death in 2019, being a cause of 15% of all under 5 years old children death but about 23% of all deaths. According to World Health Organization (WHO), vaccination, environmental factors and proper nutrition can be used as preventive measures of pneumonia infection. Vaccination is taken as

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"the common effective method to prevent certain bacterial and viral pneumonia in both children and adults. The two types of vaccines available against streptococcus pneumonia are the pneumococcal conjugate vaccine (PCV) and pneumococcal polysaccharide vaccine (PPV), whereby PCVs have been used in children only and PPV has been used for the at-risk adults and the elderly" (Feldman & Shaddock, 2019).

The decreases rate of aspiration pneumonia can be achieved through suctioning infant's throat and mouth with meconium-stained amniotic acid. Also, environmental prevention methods involve hand washing as well as indoor lessening of air pollution, and cessation of smoking in the midst of infected person since the circulation of the viruses and bacteria can occurs through one's hands to the mouth. Reduction risk of pneumonia can be achieved by treating effectively individuals with some diseases such as AIDS, lung diseases, heart diseases, and so on (Singh & Aneja, 2011).

International initiatives and prevention of pneumonia infection can't be wholly understood by biology, physics, chemistry and healthcare tools alone due to its complication which necessitates the development of medical services and medicine. Therefore, there is need for the infection counter measures to be estimated using mathematical model. With threat of the pneumonia infection spreading internationally everywhere, there is need to inculcate international travellers screening in order to mitigate its spreading. This research focuses on creating scientific awareness on the importance of international travellers screening of pneumonia infection.

Pneumonia as one of the main airborne transmitted diseases has continued to cause local and global health challenges. This phenomenon has necessitated us to develop a mathematical model that incorporated the international travellers screening as an appropriate strategy against the transmission of this infectious disease. According to Ngiliule (2014), screening can be referred to the use of tests or examinations to discover diseases like Hepatitis, pneumonia, HIV/AIDS and cancer in individuals who have no symptoms of any disease. The reduction of mortality and morbidity rates as a result of any disease can be achieved through screening test. Screening test as a preventive healthcare presents the best means of detecting and identifying diseases at the initial stages. Thus, this research looks at International travellers screening as a preventative measure against the spreading of Pneumonia infection.

Several mathematical models have been generated in the past with the aim of mitigating the transmission of Pneumonia infection, but screening the international travellers has not been considered in these models. The study takes into consideration the screening of international travellers in order to minimize the circulation rate of pneumonia infection caused by the movement of human beings.

2. Description of Model and Formulation

Epidemic modelling can be termed as a mathematical tool for evaluating the existing infectivity status and outbreaks of the disease in a given community. The analysis of model formulation provides a structured approach to mitigate the spreading of pneumonia infection. In this study, we developed a *SXEIT* model which has five compartments at any given time (t). The

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Susceptible class is denoted with the symbol $S(t)$. They are those individuals who are at the risk of contracting pneumonia infection through human mobility. A group of international travellers who undergo screening of pneumonia infection before travelling are represented with $X(t)$. The Exposed class $E(t)$ are those already infected but cannot transmit the disease. This class has the disease but can't infect it to any person. The Infected class $I(t)$ represents those individuals who have been examined and approved to be infected with pneumonia infection and can infect it to anyone. The Treated class $T(t)$ represents those individuals who have received healing from the disease but they can still be re-infected with the disease. Pneumonia infection cannot be transmitted to susceptible individuals by exposed and treated individuals. However, not everyone who comes in contact with infectious person becomes infectious immediately. The arrows in the model symbolize the rates of incoming and exiting the compartments in order to create differential equations of the model. When the arrow enters the compartment, the rate becomes positive and when the arrow exits the compartment, the rate becomes negative.

The population size (N) at a given time (t) is denoted by:

$$N(t) = S(t) + X(t) + E(t) + I(t) + T(t)$$

2.1 Description of Model Parameters

In mathematical modelling, parameters are the numerical values that represent various aspects of the system being studied. These values are used in mathematical equations to describe and simulate the behaviour of the system. The descriptions of model parameters in the study include:

Π = Recruitment rate population to the susceptible population

μ = Natural mortality rate of individuals

θ = Rate at which Susceptible population is screened

α = Rate at which Susceptible population enters the Exposed compartment

ρ = Rate at which the Screened gets into Infectious population

δ = Rate at which the Screened gets into Exposed population

β = Rate at which Exposed population enter the infectious population

κ = Rate of Screened to Susceptible population

γ = Rate of the Infected population to Treated compartment

ω = Rate of the Treated population to susceptible compartment due to temporary acquired immunity

ψ = Rate of the dead ones due to the disease

S = Susceptible

X = Screened

E = Exposed

I = Infected

T = Treated

2.2 SXEIT Model Diagram

The diagram in Figure 1 below explains how the aforementioned model can be presented.

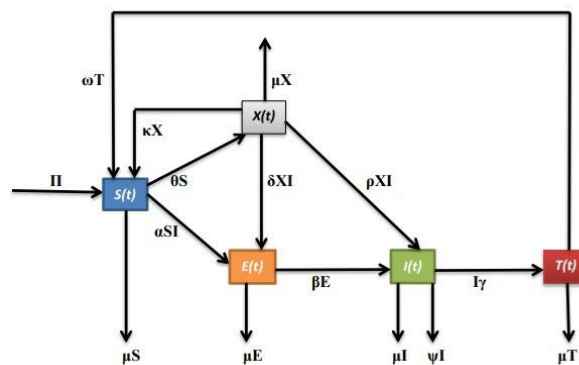


Figure 1: SXEIT Model Flow

Source: The Authors, 2024

$$\frac{dS}{dt} = \Pi + \omega T + \kappa X - (\theta + \alpha I + \mu)S \quad (1)$$

$$\frac{dX}{dt} = \theta S - (\kappa + \mu + \rho I + \delta I)X \quad (2)$$

$$\frac{dE}{dt} = \alpha SI + \delta XI - (\beta + \mu)E \quad (3)$$

$$\frac{dI}{dt} = \rho XI + \beta E - (\gamma + \psi + \mu)I \quad (4)$$

$$\frac{dT}{dt} = \gamma I - (\omega + \mu)T \quad (5)$$

When the compartments are subjected to the initial conditions, we have;

"S (0) ≥ 0, X (0) ≥ 0, E (0) ≥ 0, I (0) ≥ 0, T (0) ≥ 0".

Then the derivative of the population size N(t) with respect to t is written as;

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dX(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI(t)}{dt} + \frac{dT(t)}{dt}$$

2.3 Justification of the Model

Native and immigrant travellers are susceptible to Pneumonia transmission. The identification of those infected and those who are not infected is done through screening in the model. A treated person has no full immunity of pneumonia infection. The person can go back to the Susceptible class again even after being treated. However, an individual must be infected before he or she talks about being treated. There is no direct movement from the Screening to Treated compartment.

The goal of developing the *SXEIT* mathematical model is to demonstrate that pneumonia transmission disease, which has caused high rate of morbidity and mortality in children and elderly ones can be solved mathematically. Also, this study will assist other mathematicians greatly in identifying the areas that will need further investigations.

3 Results and Discussions

3.1 Solutions of the Model

3.1.1 Positivity of the Solution

The positivity of the solutions can be referred to the requirement that the solutions of the model equation should have non-negative values for all time (t) to be epidemiologically meaningful.

Theorem: If " $S(t) > 0$, $X(t) > 0$, $E(t) > 0$, $I(t) > 0$, and $T(t) > 0$ ", then all the solution set ($S(t)$, $X(t)$, $E(t)$, $I(t)$, $T(t)$) are positive for $t \geq 0$.

Proof: Considering the system equation of the model (1), we get;

$$\frac{dS}{dt} = \Pi + \omega T + \kappa X - (\theta + \alpha I + \mu)S$$

$$\frac{dS}{dt} \geq -(\theta + \alpha I + \mu)S$$

$$\frac{dS}{S} \geq -(\theta + \alpha I + \mu)d(t)$$

$$\int \frac{dS}{S} \geq -\int (\theta + \alpha I + \mu)d(t)$$

$$S(t) \geq S_0 e^{-(\theta + \alpha I + \mu)t} \geq 0$$

So, we get " $S(t) \geq 0$, $X(t) \geq 0$, $E(t) \geq 0$, $I(t) \geq 0$ and $T(t) \geq 0$ " for all $t \geq 0$. Thus, the set of solutions given by ($S(t)$, $X(t)$, $E(t)$, $I(t)$, $T(t)$) is positive.

3.1.2 Invariant Region

The mathematical model of the study, *SXEIT* is a model with ODEs, Π and the variable and parameters of the positive which is assumed $\forall_t \geq 0$. In calculating the invariant region, the set Π attracts all solutions in \mathbb{R}^5_+ and is positively invariant. The population size at any time (t) is written as;

$$N(t) = S(t) + X(t) + E(t) + I(t) + T(t)$$

The derivatives of (N) are given as;

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} = \frac{dX(t)}{dt} = \frac{dE(t)}{dt} = \frac{dI(t)}{dt} = \frac{dT(t)}{dt}$$

Substituting the model equations of the system gives us;

$$\frac{dN(t)}{dt} = [\Pi + \omega T + \kappa X - (\theta + \alpha I + \mu)S] + [\theta S - (\kappa + \mu + \rho I + \delta I)X] + [\alpha SI + \delta XI - (\beta + \mu)E] + [\rho XI + \beta E - (\gamma + \psi + \mu)I] + [\gamma I - (\omega + \mu)T]$$

Collecting the like terms gives us;

$$\frac{dN(t)}{dt} = \Pi - \mu S - \mu X - \mu E - \psi I - \mu I - \mu T$$

In the absence of dead ones due to the disease ($\psi = 0$), equation gives us;

$$\frac{dN(t)}{dt} = \Pi - \mu(S + X + E + I + T)$$

$$\frac{N(T)}{dt} \leq \Pi - \mu N(t)$$

Divide both sides by;

$$\Pi - \mu N(t)$$

Integrating both sides, we get;

$$\int \frac{dN(t)}{\Pi - \mu N(t)} \leq \int dt$$

So, we let

$$\Pi - \mu N(t) = \eta$$

$$\frac{d\eta}{dN} = -\mu$$

We find dN as,

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$$dN = \frac{d\eta}{-\mu}$$

Therefore, we have;

$$\int \frac{d\eta / -\mu}{\eta} = \frac{1}{-\mu} \int \frac{d\eta}{\eta} = \frac{1}{-\mu} \ln(\eta)$$

$$\frac{1}{-\mu} \ln(\eta) \leq t + c$$

Calculating it we obtain;

$$\eta = e^{-\mu(t+c)} = e^{-\mu(t)} \cdot e^0$$

Taking the logs both sides, we get;

$$\ln(\eta) \geq -\mu(t + c)$$

So, we have

$$e^{\ln(\eta)} \geq e^{-\mu(t+c)}$$

Thus,

$$t = A \cdot e^{-\mu(t)}$$

But,

$$\eta = e^{-\mu(t+c)} = e^{-\mu(t)} \cdot e^c$$

This means that;

$$\eta \geq A \cdot e^{-\mu(t)}$$

Also,

$$\eta = \Pi - \mu N(t)$$

So, we can write it as;

$$\Pi - A \cdot e^{-\mu(t)} \geq \mu N(t)$$

$$\frac{\mu}{\mu} N(t) \leq \frac{\Pi}{\mu} - \frac{A \cdot e^{-\mu t}}{\mu}$$

$$N(t) \leq \frac{\Pi}{\mu} - \frac{A \cdot e^{-\mu t}}{\mu}$$

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At $t = 0$, we have;

$$N(0) \leq \frac{\Pi}{\mu} - \frac{A}{\mu} \cdot e^0$$

Multiply both sides by μ , we obtain;

$$\mu N(0) = \Pi - A$$

So,

$$A = \Pi - \mu N(0)$$

$$N(t) = \frac{\Pi}{\mu} - \frac{\mu N(0)}{\mu} \cdot e^{-\mu t}$$

Since $t \rightarrow \infty$, we have;

$$\left(\frac{\Pi}{\mu} - N(0)\right) \cdot e^{-\mu t} \rightarrow 0$$

Therefore,

$$N(t) \leq \frac{\Pi}{\mu}$$

$$N(0) \leq \frac{\Pi}{\mu}$$

The flow generated by the system model is positively invariant. Thus, the (S, X, E, I, T) model is epidemiologically and biologically meaningful.

3.1.3 Disease Free Equilibrium (DFE)

At a period when there are no infected persons in the population, it is termed as the Disease-Free Equilibrium. It means that there is no spreading of the disease. In the *SEIR* model, it occurs similarly when the number of Infectious individuals (I) and Exposed individuals (E) are zero. All differentials at the equilibrium are equal to zero. When there is no change in the system at any given time, the system is said to have equilibrium point (Ndairou et al., 2020).

Thus, the differential equations for the equilibrium can be calculated by zeroing the left sides of the differential equations of the study which gives the non-linear system.

$$0 = \Pi + \omega T + \kappa X - (\theta + \alpha I + \mu)S$$

$$0 = \theta S - (\kappa + \mu + \rho I + \delta I)X$$

$$0 = \alpha SI + \delta XI - (\beta + \mu)E$$

$$0 = \rho XI + \beta E - (\gamma + \psi + \mu)I$$

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$$0 = \gamma I - (\omega + \mu)T$$

We can easily obtain the Disease-Free Equilibrium by zeroing X^* , E^* , I^* , T^* that is $X^* = E^* = I^* = T^* = 0$ since no infected person exists in the society.

So, using equation $0 = \Pi + \omega T + \kappa X - (\theta + \alpha I + \mu)S$ where $T=X=I = 0$, we have;

$$0 = \Pi - (\theta + \mu)S$$

$$0 = \theta S$$

Making (S) the subject of the formulae in equation, $0 = \Pi - (\theta + \mu) S$ and we get;

$$S = \frac{\Pi}{(\theta + \mu)}$$

Since $0 = \theta S$, we have either $S = 0$ or $\theta = 0$. However; $S \neq 0$ and $\theta = 0$.

Therefore; $S = \frac{\Pi}{\mu}$

Thus, $(S^*, X^*, E^*, I^*, T^*) = (\frac{\Pi}{\mu}, 0, 0, 0, 0)$

3.1.4 Endemic Equilibrium (EE)

Endemic Equilibrium State can be defined as a state where the disease persists in the population. At the endemic equilibrium, all the variables in the system which include; "The Susceptible Class, the Screened Class, the Exposed Class, the Infectious Class, and Treated Class" are taken in consideration and must not be empty for the disease to exist among the individuals.

For instance, if $(S^*, X^*, E^*, I^*, T^*)$ is at the state of endemic equilibrium, then $(S^*, X^*, E^*, I^*, T^*) \neq (0, 0, 0, 0, 0)$.

To find the Endemic Equilibrium point of the equations, we equate the left sides of the differential equations to zero and calculate the variables $(S^*, X^*, E^*, I^*, T^*)$ from the equations;

$$0 = \Pi + \omega T + \kappa X - (\theta + \alpha I + \mu)S$$

$$0 = \theta S - (\kappa + \mu + \rho I + \delta I)X$$

$$0 = \alpha S I + \delta X I - (\beta + \mu)E$$

$$0 = \rho X I + \beta E - (\gamma + \psi + \mu)I$$

$$0 = \gamma I - (\omega + \mu)T$$

From the equation $0 = \gamma I - (\omega + \mu) T$, we make 'T' the subject of the formula from the equation and we obtain;

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$$I^* = \frac{(\omega + \mu)T}{\gamma}$$

From the equation $0 = \Pi + \omega T + \kappa X - (\theta + \alpha I + \mu)S$, we replace I and make X the subject of the formula and we obtain;

$$X^* = \frac{(-T\omega - \Pi + (\theta + \mu)S)\gamma + \alpha S(\omega + \mu)T}{\gamma\kappa}$$

From the equation $0 = \theta S - (\kappa + \mu + \rho I + \delta I)X$, we replace X and I , and make S the subject of the formula from the equation and we obtain;

$$S^* = \frac{((\delta + \rho)(\omega + \mu)T + \gamma(\kappa + \mu))(T\omega + \Pi)\gamma}{\alpha(\omega + \mu)^2(\delta + \rho)T^2 + (\omega + \mu)((\rho + \alpha + \delta)\mu + \alpha\kappa + \theta(\delta + \rho))\gamma T + \mu\gamma^2(\theta + \mu + \kappa)}$$

From the equation $0 = \alpha SI + \delta XI - (\beta + \mu)E$, we replace S , X and I , and make E the subject of the formula from the equation and we have;

$$E^* = \frac{T(\omega + \mu)(T\omega + \Pi)(\alpha(\delta + \rho)(\omega + \mu)T + \gamma(\alpha\kappa + \mu\alpha + \theta\delta))}{(\beta + \mu)(\alpha(\omega + \mu)^2(\delta + \rho)T^2 + (\omega + \mu)((\rho + \alpha + \delta)\mu + \alpha\kappa + \theta(\delta + \rho))\gamma T + \mu\gamma^2(\theta + \mu + \kappa)}$$

From the equation $0 = \rho XI + \beta E - (\gamma + \psi + \mu)I$, we substitute E , S , X and I , and make T the subject of the formula from the equation and we get;

$$T^* = 0$$

Theorem: The Endemic Equilibrium point (EE) is "asymptotically stable when $R_0 > 1$ and unstable when $R_0 < 1$ ".

Hence, there is endemic equilibrium for this model, $(S^*, X^*, E^*, I^*, T^*)$.

3.1.5 Basic Reproduction Number R_0

The Basic Reproductive Number R_0 can be termed as "the average number of secondary infections caused by a single infectious individual during their entire infectious lifetime" (Diekmann, 1996). It helps us to ascertain whether the infection will circulate, fade out or remain constant. In the present work, the Next Generation Matrix has been used to establish the R_0 . The compartments with the infected people of the most vulnerable (E and I compartment) are considered. Here, we have these ordinary differential equations in the model;

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$$\frac{dS}{dt} = \Pi + \omega T + \kappa X - (\theta + \alpha I + \mu)S$$

$$\frac{dX}{dt} = \theta S - (\kappa + \mu + \rho I + \delta I)X$$

$$\frac{dE}{dt} = \alpha SI + \delta XI - (\beta + \mu)E$$

$$\frac{dI}{dt} = \rho XI + \beta E - (\gamma + \psi + \mu)I$$

$$\frac{dT}{dt} = \gamma I - (\omega + \mu)T$$

In this study, the denoted vector "X" and "Y" respectively is used in considering the infected and non - infected compartments. Hence, we get;

$$X = [E, I]$$
$$Y = [S, X, T]$$

In considering the infected compartments, we get;

$$\frac{dE}{dt} = \alpha SI + \delta XI - (\beta + \mu)E$$

$$\frac{dI}{dt} = \rho XI + \beta E - (\gamma + \psi + \mu)I$$

If we assume that $f(x)$ denotes the number of incoming new infections into the system while $v(x)$ denotes the number of incoming new infections exiting the system, then it is written as;

$$\frac{dx}{dt} = f(x) - v(x)$$

When the infected system in the study is linearized and writing the above equations in matrix, we get;

$$f(x) \Rightarrow \begin{pmatrix} f_1 \\ f_2 \end{pmatrix} = \begin{pmatrix} \alpha SI + \delta XI \\ \rho XI \end{pmatrix}$$

And

$$v(x) \Rightarrow \begin{pmatrix} v_1 \\ v_2 \end{pmatrix} = \begin{pmatrix} \beta E + \mu E \\ -\beta E + (\gamma + \psi + \mu)I \end{pmatrix}$$

By differentiating the above matrices of $v(x)$ and $f(x)$ with respect to I and E respectively, we get;

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$$F = \begin{pmatrix} \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} \\ \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} \end{pmatrix} = \begin{pmatrix} 0 & \alpha S + \delta X \\ 0 & \rho X \end{pmatrix}$$

And

$$V = \begin{pmatrix} \frac{\partial v_1}{\partial E} & \frac{\partial v_1}{\partial I} \\ \frac{\partial v_2}{\partial E} & \frac{\partial v_2}{\partial I} \end{pmatrix} = \begin{pmatrix} \beta + \mu & 0 \\ -\beta & (\gamma + \psi + \mu) \end{pmatrix}$$

At DFE, $(S^*, X^*, E^*, I^*, T^*) = (\frac{\Pi}{\mu}, 0, 0, 0, 0)$, we have;

$$F = \begin{pmatrix} 0 & (\frac{\Pi}{\mu})\alpha \\ 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} (\beta + \mu) & 0 \\ -\beta & (\gamma + \psi + \mu) \end{pmatrix}$$

The number of secondary infections are yielded by "the next generation matrix approach" FV^{-1} . We define V^{-1} ;

$$\text{Det } V = (\beta + \mu)(\gamma + \psi + \mu)$$

$$V^{-1} = \frac{1}{(\beta + \mu)(\gamma + \psi + \mu)} \begin{pmatrix} (\gamma + \psi + \mu) & 0 \\ \beta & (\beta + \mu) \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{(\gamma + \psi + \mu)}{(\beta + \mu)(\gamma + \psi + \mu)} & 0 \\ \frac{\beta}{(\beta + \mu)(\gamma + \psi + \mu)} & \frac{(\beta + \mu)}{(\beta + \mu)(\gamma + \psi + \mu)} \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{(\beta + \mu)} & 0 \\ \frac{\beta}{(\beta + \mu)(\gamma + \psi + \mu)} & \frac{1}{(\gamma + \psi + \mu)} \end{pmatrix}$$

Thus,

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$$FV^{-1} = \begin{pmatrix} 0 & \left(\frac{\pi}{\mu}\right)\alpha \\ 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{(\beta + \mu)} & 0 \\ \frac{\beta}{(\beta + \mu)(\gamma + \psi + \mu)} & \frac{1}{(\gamma + \psi + \mu)} \end{pmatrix}$$

Therefore,

$$FV^{-1} = \begin{pmatrix} \frac{\alpha\pi\beta}{\mu(\beta + \mu)(\gamma + \psi + \mu)} & \frac{\alpha\pi}{(\gamma + \psi + \mu)} \\ 0 & 0 \end{pmatrix}$$

The Basic Reproduction number is defined with "the spectral radius ρ of the next generation Matrix, FV^{-1} " (Michel et al., 2022). So, the value FV^{-1} is the Next Generation Matrix approach, $R_0 = \rho(FV^{-1})$.

To obtain the eigenvalue, we get;

$$FV^{-1} - \lambda I = \begin{pmatrix} \frac{\alpha\pi\beta}{\mu(\beta + \mu)(\gamma + \psi + \mu)} - \lambda & \frac{\alpha\pi}{(\gamma + \psi + \mu)} \\ 0 & 0 - \lambda \end{pmatrix}$$

$$\left(\frac{\alpha\pi\beta}{\mu(\beta + \mu)(\gamma + \psi + \mu)} - \lambda \right) (0 - \lambda) = 0$$

Then the eigenvalues calculated are;

$$\lambda_1 = \frac{\alpha\pi\beta}{\mu(\beta + \mu)(\gamma + \psi + \mu)}$$

$$\lambda_2 = 0$$

Thus, the Basic Reproductive Number R_0 is written as;

$$R_0 = \frac{\alpha\pi\beta}{\mu(\beta + \mu)(\gamma + \psi + \mu)}$$

3.2 Stability of the System

The stability of the system is noted when there is a controlled output. Stability analysis often involves mathematical techniques of determining the stability of a system. Any system is said to be stable "if the eigenvalues of the Jacobian Matrix lie within the stability region of the system" (Bolster & Hornberger, 2007).

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3.2.1 Local Stability of Disease Free Equilibrium

The Disease Free Equilibrium, $DFE = (\frac{\Pi}{\mu}, 0,0,0,0)$ is "locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$ ". We confirm the local stability of DFE by calculating the Jacobian matrix of the model equations, $J(S,X,E,I,T)$ and we get;

$$J = \begin{pmatrix} -(\theta + \alpha I + \mu) & \kappa & 0 & -(\alpha S) & \omega \\ \theta & -(\kappa + \mu + \rho I + \delta I) & 0 & -(\rho X + \delta X) & 0 \\ \alpha I & \delta I & -(\mu + \beta) & (\alpha S + \delta X) & 0 \\ 0 & \rho I & \beta & \rho X - (\gamma + \psi + \mu) & 0 \\ 0 & 0 & 0 & \gamma & -(\omega + \mu) \end{pmatrix}$$

To find the Jacobian Matrix at determinant system, we have;

$$J = \begin{pmatrix} -(\theta + \alpha I + \mu) - \lambda & \kappa & 0 & -(\alpha S) & \omega \\ \theta & -(\kappa + \mu + \rho I + \delta I) - \lambda & 0 & -(\rho X + \delta X) & 0 \\ \alpha I & \delta I & -(\mu + \beta) - \lambda & (\alpha S + \delta X) & 0 \\ 0 & \rho I & \beta & \rho X - (\gamma + \psi + \mu) - \lambda & 0 \\ 0 & 0 & 0 & \gamma & -(\omega + \mu) - \lambda \end{pmatrix}$$

Where λ is the eigenvalue.

Reducing the Jacobian Matrix at $DFE (\frac{\Pi}{\mu}, 0,0,0,0)$, we get;

$$J = \begin{pmatrix} -(\theta + \mu) - \lambda & \kappa & 0 & -\alpha(\frac{\Pi}{\mu}) & \omega \\ \theta & -(\kappa + \mu) - \lambda & 0 & 0 & 0 \\ 0 & 0 & -(\mu + \beta) - \lambda & \alpha(\frac{\Pi}{\mu}) & 0 \\ 0 & 0 & \beta & -(\gamma + \psi + \mu) - \lambda & 0 \\ 0 & 0 & 0 & \gamma & -(\omega + \mu) - \lambda \end{pmatrix}$$

Calculating the eigenvalues of the model system, we have;

$$J = \begin{pmatrix} -\mu \\ -\omega - \mu \\ -1/2 \frac{\gamma\mu + \beta\mu + 2\mu^2 + \psi\mu - \sqrt{4\beta\Pi\alpha\mu + \beta^2\mu^2 - 2\beta\gamma\mu^2 - 2\beta\mu^2\psi + \gamma^2\mu^2 + 2\gamma\mu^2\psi + \mu^2\psi^2}}{\mu} \\ -1/2 \frac{\gamma\mu + \beta\mu + 2\mu^2 + \psi\mu - \sqrt{4\beta\Pi\alpha\mu + \beta^2\mu^2 - 2\beta\gamma\mu^2 - 2\beta\mu^2\psi + \gamma^2\mu^2 + 2\gamma\mu^2\psi + \mu^2\psi^2}}{\mu} \\ -\mu - \theta \end{pmatrix}$$

Due to the fact that all the eigenvalues are negative, we finalize that "Disease Free Equilibrium is locally asymptotically stable".

3.2.2 Global Stability of Endemic Equilibrium

The behaviour of epidemic near the equilibrium points is done with the analysis of stability. The Disease Endemic Equilibrium is "globally asymptotically stable, if $R_0 > 1$ " (Bolster & Hornberger, 2007). In the study, we use Lyapunov function to prove the Global stability of Endemic Equilibrium as shown below;

$$L(S^*, X^*, E^*, I^*, T^*) = (S - S^* - S^* \ln \frac{S^*}{S}) + (X - X^* - X^* \ln \frac{X^*}{X}) + (E - E^* - E^* \ln \frac{E^*}{E}) + (I - I^* - I^* \ln \frac{I^*}{I}) + (T - T^* - T^* \ln \frac{T^*}{T})$$

By calculating the derivative of L, we get;

$$\frac{dL}{dt} = \left(\frac{S - S^*}{S}\right) \frac{dS}{dt} + \left(\frac{X - X^*}{X}\right) \frac{dX}{dt} + \left(\frac{E - E^*}{E}\right) \frac{dE}{dt} + \left(\frac{I - I^*}{I}\right) \frac{dI}{dt} + \left(\frac{T - T^*}{T}\right) \frac{dT}{dt}$$

By substituting the model equations in the above equation gives us;

$$\begin{aligned} \frac{dL}{dt} = & \left(\frac{S - S^*}{S}\right) [\Pi + \omega T + \kappa X - (\theta + \alpha I + \mu)S] + \left(\frac{X - X^*}{X}\right) [\theta S - (\kappa + \mu + \rho I + \delta I)X] \\ & + \left(\frac{E - E^*}{E}\right) [\alpha SI + \delta XI - (\beta + \mu)E] + \left(\frac{I - I^*}{I}\right) [\rho XI + \beta E - (\gamma + \psi + \mu)I] \\ & + \left(\frac{T - T^*}{T}\right) [\gamma I - (\omega + \mu)T] \end{aligned}$$

Simplifying the above equations, we have;

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$$\begin{aligned} \frac{dL}{dt} = & \left(1 - \frac{S^*}{S}\right) [\Pi + \omega T + \kappa X - (\theta + \alpha I + \mu)S] + \left(1 - \frac{X^*}{X}\right) [\theta S - (\kappa + \mu + \rho I + \delta I)X] \\ & + \left(1 - \frac{E^*}{E}\right) [\alpha SI + \delta XI - (\beta + \mu)E] + \left(1 - \frac{I^*}{I}\right) [\rho XI + \beta E - (\gamma + \psi + \mu)I] \\ & + \left(1 - \frac{T^*}{T}\right) [\gamma I - (\omega + \mu)T] \end{aligned}$$

The above equation points down to;

$$\begin{aligned} & \Pi + \omega T + \kappa X - (\theta + \alpha I + \mu)S - \Pi S^* - \omega T S^* - \kappa X S^* + (\theta + \alpha I + \mu)S^* + \theta S - (\kappa \\ & + \mu + \rho I + \delta I)X - \theta S X^* + (\kappa + \mu + \rho I + \delta I)X^* + \alpha SI + \delta XI - (\beta + \mu)E - \alpha S I E^* \\ & - \delta X I E^* + (\beta + \mu)E^* + \rho X I + \beta E - (\gamma + \psi + \mu)I + \rho I X I^* - \beta E I^* + (\gamma + \psi + \\ & \mu)I^* + \gamma I - (\omega + \mu)T - \gamma I T^* + (\omega + \mu)T^* \end{aligned}$$

From the above equation, X stands for positive terms while Y stands for negative terms and this gives us;

$$X = \Pi + \omega T + \kappa X + (\theta + \alpha I + \mu)S^* + \theta S + (\kappa + \mu + \rho I + \delta I)X^* + \alpha SI + \delta XI + (\beta + \mu)E^* + \rho X I + \beta E + \rho I X I^* + (\gamma + \psi + \mu)I^* + \gamma I + (\omega + \mu)T^*$$

$$Y = -[(\theta + \alpha I + \mu)S + \Pi S^* + \omega T S^* + \kappa X S^* + (\kappa + \mu + \rho I + \delta I)X + \theta S X^* + (\beta + \mu)E + \alpha S I E^* + \delta X I E^* + (\gamma + \psi + \mu)I + \beta E I^* + (\omega + \mu)T + \gamma I T^*]$$

From the above equation;

$$\text{Let } \frac{dL}{dt} = X - Y$$

Thus, if we have the condition $X < Y$, then $\frac{dL}{dt} \leq 0$,

So, $\frac{dL}{dt} = 0$, if and only if;

$S = S^*, X = X^*, E = E^*, I = I^*, T = T^*$. Hence, the largest invariant set in $(S^*, X^*, E^*, I^*, T^*)$:

$\frac{dL}{dt} = 0$ is E^* , where E^* , is the Endemic point.

This implies that “the endemic equilibrium (EE) is globally asymptotically stable” (Bolster & Hornberger, 2007).

4 Numerical Results

4.1 Pneumonia infection in relation to the human population dynamics

Figure 2 shows the pneumonia infection dynamics of the total population which comprises of "Susceptible (S), Screened (X), Exposed (E), Infected (I) and Treated (T)" are being analyzed. The findings have shown that the decrease in the number of the Exposed and Infectious populations of Pneumonia infection at any given time occur as a result of the increment of the Screened population of Pneumonia infection while the decrease in the number of the screened population of Pneumonia infection rises the number of the Exposed and Infectious populations of Pneumonia infection. By screening the International travellers, those who are infected with pneumonia infection are known and restrained from travelling thus, the rate of pneumonia transmission is minimized. When the Treated population increases with time per week, there are more recoveries than the Susceptible, Exposed and Infected populations. So, the death rate decreases due to the effect of Screening the international travellers of Pneumonia infection with time.

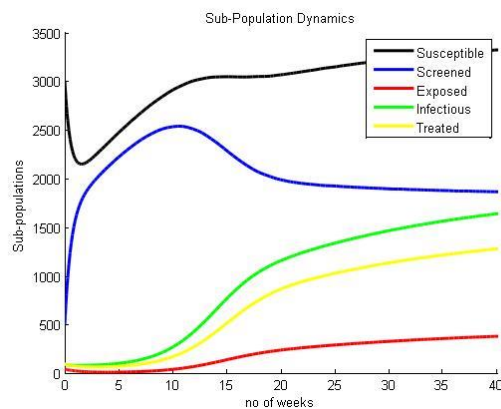


Figure 2: Pneumonia Dynamics Graph
Source: The Authors, 2024

4.2 Screened and Susceptible Population

Figure 3 demonstrates the graph of the Susceptible population and Screened population of Pneumonia transmission at any given time. The graph depicts that the decrease of the Screened population occurs as a result of the increase of the Susceptible population. Also, there will be at some time when the Susceptible and Screened populations will seem to be stabilized. This shows that screening procedure can serve as a control measure of pneumonia transmission.

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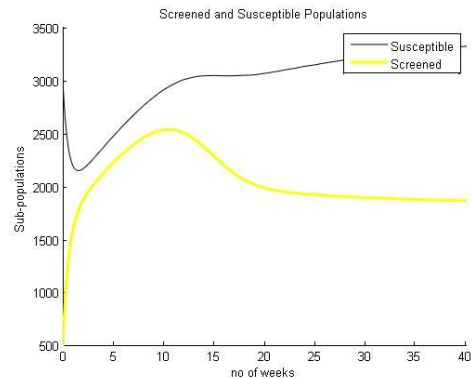


Figure 3: Screened and Susceptible Pnuemonia Population Graph
Source: The Authors, 2024

4.3 Exposed and Infected Population

Figure 4 shows the graph of the exposed and infected population in weeks. From the graph, it can be seen that the individuals who are exposed to pneumonia infection increase and decrease with the infectious population at any given time in weeks. This implies that the rise of screening procedure helps to reduce the number of exposed and infected population with time, hence it helps in the control measure of pneumonia infection.

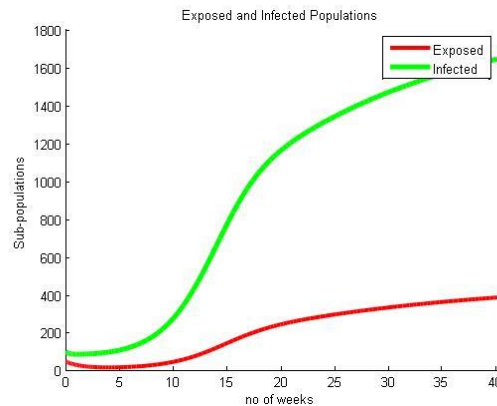


Figure 4: Exposed and infected Pneumonia Population Graph
Source: The Authors, 2024

4.4 Screened and Infected Population

Figure 5 depicts the graph of the Screened population and Infected population of Pneumonia transmission at any given time per week. From the graph, when the screening of susceptible population increases at the initial stage, there is the possibility of getting a large number of infectious population. Also, the decrement of the screened population leads to the increment of the infectious population at some stage. After sometime, the screened and infected populations are stabilized. This means that, optimized screening of international travellers minimizes the rate of pneumonia transmission in the society.

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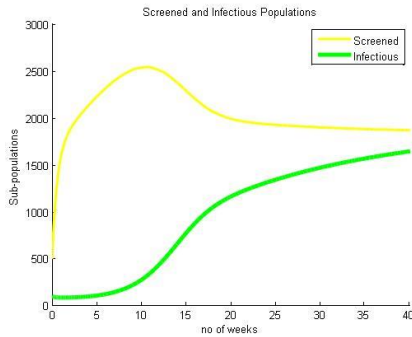


Figure 5: Screened and Infected Pneumonia Population Graph
Source: The Authors, 2024

4.5 Screened Population Dynamics

Figure 6 illustrates the graph for the pneumonia transmission dynamics of the screened population in weeks. The increment of the Screened population results to the decrement of infected population. So, screening of the Susceptible population mitigates the spreading of the pneumonia infection. Thus, we can minimize the rate of pneumonia infection by maximizing the rate at which the susceptible population is effectively screened at any given time per week.

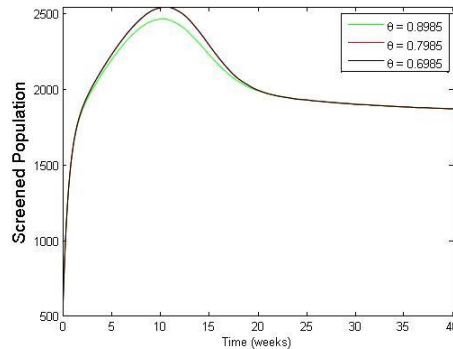


Figure 6: Contrasting the rate of Screened Population Graph
Source: The Authors, 2024

4.6 Infected Population Dynamics

Figure 7 demonstrates that the frequency of the infected population is directly proportional to the frequency of screened population. This means that the more international travellers are screened the more infectious population are known. From the graph, one can understand that screening procedure exposes the infectious international travellers and this phenomenon helps in reduction of the Pneumonia transmission with time.

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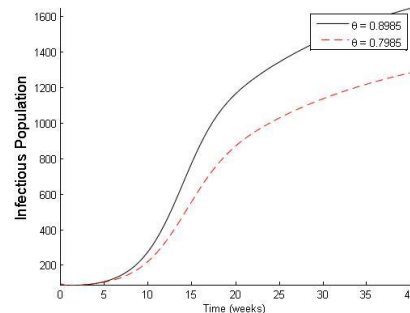


Figure 7: Contrasting the rate of infectious Population Graph
Source: The Authors, 2024

5 Conclusion and Recommendation

5.1 Conclusion

By taking into account of *SXEIT* model in the study, we examined the screening deterministic model for the dynamics of Pneumonia infection among the international travellers. This research has established the positivity of the solution and invariant region. The investigation of the stability of the model showed that both Disease Free Equilibrium and Endemic Equilibrium were well established together with their stabilities. The Next Generation Matrix approach was used to calculate the Basic Reproduction Number of the model R_0 which is "the average number of secondary infections caused by a single infectious individual during their entire infectious lifetime" (Diekmann, 1996). This meant that If $R_0 > 1$, then the infection will become endemic, while if $R_0 < 1$, the disease will wipe out.

The linearization of the differential equations in the system model is obtained with use of the Jacobian Matrix approach while Runge-kutta method is used in solving and approximating the solutions at different points of non-linear differential equations. The global stability of endemic equilibrium in the study was calculated using Lyapunov function and the local stability of Disease Free Equilibrium is obtained using the Jacobian matrix approach of the model system. The numerical simulations in the study showed that the best way of controlling the circulation of Pneumonia infection is to screen the international travellers. Thus, the numerical results have established that the increment of screened population result to the decrement in the number of those carrying Pneumonia infections.

Finally, conducting the screening of the international travellers' test considerably minimizes the rate of the circulation of Pneumonia infection in the society.

5.2 Recommendations

Based on the study findings, we recommend that governments should place severe rules and regulations to make sure that screening the international travellers of pneumonia infection with their certificates at the borders is compulsory and no infected individuals are permitted to travel without being cured. Other agencies like non - governmental organization and faith based institution can use the findings of this research to educate people across the country concerning the widespread nature of the pneumonia infection and its control measure. These recommendations can serve as control mechanisms in mitigating the spreading of the pneumonia

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infection. We suggest that further researches can be done on the study considering the effectiveness of screening the international travellers locally and globally by incorporating a vaccine therapy.

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