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A Model for Childhood Pneumonia Dynamics

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Abstract

This paper presents a deterministic model for pneumonia transmission and uses the model to assess the potential impact of therapy. The model is based on the Susceptible-Infected-Treatment-Susceptible compartmental structure with the possibility of infected individual recovering from natural immunity. Important epidemiological thresholds such as the basic and control reproduction numbers ($R_o$and R_c respectively) and a measure of treatment impact are derived. Infection free point was found to be locally stable but globally unstable. We found that if the control reproduction number is greater than unity, then there is a unique endemic equilibrium point and it is less than unity, the endemic equilibrium point is globally asymptotically stable, and pneumonia will be eliminated. Numerical simulations using Matlab software suggest that, besides the parameters that determine the basic reproduction number, natural immunity plays an important role in pneumonia transmissions and magnitude of the public health impact of therapy. Further, treatment regimens with better efficacy holds great promise for lowering the public health burden of pneumonia disease.

Keywords: Basic reproduction number, Control reproduction number, Infection free point, Endemic equilibrium point, Local and global stability of equilibrium points.

Contribution/ Originality

This study contributes in the existing literature by considering places where no studies have been done on types of pneumonia available in the population.This study uses new estimation methodology of determining initial conditions. This study originates new formula for determining dynamics of childhood pneumonia.This study is one of very few studies which have investigated possibility of infection during treatment with different strains especially where isolation of Pneumonia is not done before treatment.The paper contributes the first logical analysis of the model and validation through simulation. The paper's primary contribution is finding that boosting natural immunity and treatment are important intervention strategy of childhood Pneumonia.This study documents policy recommendation and recommendation for further research.

1. Introduction

Pneumonia is an infection of the lungs that is caused by bacteria, viruses, fungi, or parasites which is characterized primarily by inflammation of the alveoli in the lungs or by alveoli that are filled with fluid. Bacteria and viruses are the primary causes of pneumonia. When a person breath pneumonia-causing pathogens into his lungs and the body's immune system cannot prevent entry, the organisms settle in the small air sacs called alveoli and continue to multiply. The host body sends white blood cells to attack the infection causing the sacs to be filed with fluid and pus-causing pneumonia. The people most susceptible to Pneumonia are the old, infants, the sick and those with impaired immune systems [1].

According to WHO, childhood pneumonia can be spread through inhaling viruses and bacteria, air-borne droplets from a cough or sneeze, direct contact or through blood, especially during and shortly after birth. Pneumonia symptoms include cough, custy or green mucus coughed up from lungs, fever, fast breathing and shortness of breath, chills, chest pain that usually worsens when taking a deep breath , fast heartbeat, fatigue and feeling very weak, nausea and vomiting, diarrhea, sweating, headache, muscle pain, confusion or delirium and dusky or purplish skin color (cyanosis) from poorly oxygenated blood [2-4].

Childhood pneumonia is a major public health issue for Kenya. In 2008, joint report by Unicef and WHO pneumonia was described as 'forgotten killer of children' because it was the second cause of death among less than fives years, claiming equivalent to 16 % of child mortality in Kenya. According to WHO, Pneumonia can be

prevented by immunization, proper nutrition and by addressing environmental factors. Previous mathematical modeling studies on pneumonia use numerical simulation models rather than mathematical analysis. Moreover most of the models concentrate on bacterial pneumonia, antibiotic resistance and vaccination.

Huang, et al. [4] considered a pneumococcal transmission model which takes into account the risk of higher rates of transmission for children who attend child-care centres. Children are assumed to be able to carry only one serotype in a closed community. The results stress the importance of child-care centres in transmission.

Lipsitch, et al. [5] studied the issue of coexistence of pneumonia serotypes in a population and stressed the importance of correctly modeling the possibility of a host being able to become simultaneously invaded by more than one strain, taking into account difficulties in obtaining a second strain if already colonized and considering acquired immunity of new strains.

Otieno, et al. [6] considered bacterial pneumonia with possibility of carriers, temporal immunity and treatment. The results stressed importance of treatment and quarantine where possible.

This paper considers the weak nature of a child's immune system which is vulnerable to different types of pneumonia as long as they coexist in population. It also considers the effects of treatments and the likelihood of wrong treatment due to similar symptom. It stresses treatment above critical point and boosting of natural immunity to completely eradicate childhood pneumonia.

2. Model Development

The model is formulated as follows, P (*t*) be the total population density which is divided into three sub-classes: the susceptible class *S*(*t*), the infective class *I*(*t*) and class under treatment T(t). With pneumonia infective children can become susceptible again after treatment. γ is recovery rate, the recruitment rate of the Susceptible class is π, death due to disease occur at a rate α in infectious class, μ is the natural death rate, $\beta_1$and β_2 are infection rates in infectious class and treatment class respectively, Ψ is death rate due to disease during treatment, ϕ is the rate of treatment for children and τ is the rate of recovery from infectious state through natural immunity. Pneumonia infection occurs when susceptible individuals come into contact with infected individuals and/or those under treatment. $\lambda(t)$ is force of infection (number of individuals who become infected per unit of time)

$$\lambda(t) = \beta_1 I + \beta_2 T$$

$\beta_1 > \beta_2$ since treatment reduces significantly level of infectiousness of an individual after contact and health officers are likely to create awareness on how to handle patients after visiting Health centers hence reducing the level of infectivity.

$\gamma > \tau$ since the level of recovery after treatment is higher than natural immunity.

$\alpha > \Psi$ since treatment reduces likelihood of dying significantly.

We obtain the following systems of equations

$$\frac{dS}{dt} = \pi - (\lambda + \mu)S + \gamma T + \tau I, \quad (1)$$

$$\frac{dI}{dt} = \lambda S - \Omega_1 I, \quad (2)$$

$$\frac{dT}{dt} = \phi I - \Omega_2 T, \quad (3)$$

where,

$$\Omega_1 = \phi + \mu + \alpha + \tau,$$
$$\Omega_2 = \mu + \gamma + \Psi,$$
$$P(t) = S(t) + I(t) + T(t).$$

3. Model Analysis

3.1. Positivity and Boundness of the Solutions

Theorem 1.

The region R given by

$$R = \left\{[S(t), I(t), T(t)] \in R_+^3 \,|S(0) \geq 0, I(0) \geq 0, T(0) \geq 0, P(t) \leq \frac{\pi}{\mu}\right\}$$

is positively invariant and attracting with respect to the system of equations (1) - (3).

Proof

Let (S, I, T) be any solution of the system with non –negative initial conditions From equation (1),

$$\frac{dS}{dt} + (\lambda + \mu)S = \pi + \gamma T + \tau I,$$

the right hand side of above equation is a first order linear equation in S, it follows that $\frac{d}{dt}[S(t)\, e^{\int_0^t \{\lambda(s) + \mu\} ds}] \geq 0$,since $S(t)\, e^{\int_0^t -\{\lambda(s) + \mu\}\, ds}$ is a non negative function of t, thus S(t) stays positive.

From equation (2),

$$\frac{dI}{dt} = \lambda S - \Omega_1 I,$$

This implies

$$\frac{dI}{dt} > -\Omega_1 I,$$

$$\frac{dI}{I} > -\Omega_1 dt,$$

$$I(t) > C\, e^{-\Omega_1 t},$$

where C is constant of integration, applying initial condition at t = 0,

$$C = I(0),$$

$\mathrm{I(t)} > I(0)\,\mathrm{e}^{-\Omega_1 \mathrm{t}}$,
it implies that
$\mathrm{I(t)} > I(0)\,\mathrm{e}^{-\Omega_1 \mathrm{t}} \geq 0.$
Also from equation
$\frac{\mathrm{dT}}{\mathrm{dt}} = \phi \mathrm{I} - \Omega_2 \mathrm{T}$
$> -\Omega_2 T,$
it follows that
$\mathrm{T\,(t)} > T\,(0)\mathrm{e}^{-\Omega_2 \mathrm{t}} \geq 0.$

For boundedness of solution we take the time derivative of our total population along its solution to obtain:
$\frac{\mathrm{dP}}{\mathrm{dt}} = \frac{\mathrm{dS}}{\mathrm{dt}} + \frac{\mathrm{dI}}{\mathrm{dt}} + \frac{\mathrm{dT}}{\mathrm{dt}} = \pi - \lambda \mathrm{S} - \mu \mathrm{S} + \gamma \mathrm{T} + \tau \mathrm{I} + \lambda \mathrm{S} - (\phi + \mu + \alpha + \tau)\mathrm{I} + \phi \mathrm{I} - \mu \mathrm{T} - \gamma \mathrm{T} - \Psi \mathrm{T},$
$= \pi - \mu(\mathrm{s} + \mathrm{I} + \mathrm{T}) - \alpha \mathrm{I} - \Psi \mathrm{T} = \pi - \mu \mathrm{P} - \alpha \mathrm{I} - \Psi \mathrm{T},$
now,

$$\frac{\mathrm{dP}}{\mathrm{dt}} + \mu \mathrm{P} = \pi - \alpha \mathrm{I} - \Psi \mathrm{T},$$
$$\leq \pi$$

so that
$\mathrm{P\,(t)} \leq \frac{\pi}{\mu}\,(1 + \mathrm{C}\mathrm{e}^{-\mu \mathrm{t}})$
where is a constant of integration

$$\lim_{\mathrm{t} \to \infty} \mathrm{P(t)} \leq \frac{\pi}{\mu}$$

This proves the boundedness of the solutions inside R. This implies that all solutions of the system (1) - (3) starting in R remain in R for all time $\mathrm{t} \geq 0$. Thus R is positively invariant and attracting and hence it is sufficient to consider the dynamics of our system in R [7, 8].

3.2. Equilibrium Points and Reproduction Number

3.2.1. Disease-Free Equilibrium Point (DFE)

The disease-free equilibrium point (DFE) of the system (1) - (3), is obtained by setting all the infectious classes and treatment classes to zero. We get

$$\pi - \mu S^0 = 0$$

which yields

$$S^0 = \frac{\pi}{\mu}$$

The DFE point for our system is given by,

$$E^0 = (S^0, I^0, T^0) = (\frac{\pi}{\mu}, 0, 0)$$

3.2.2. The Basic Reproduction Number $\mathbf{R_o}$ and Control Reproduction Number $\mathbf{R_C}$

We use the next-generation matrix method to determine the basic reproduction number, R_0, and control reproduction number R_c of the model [6-10]. Using the notation f for a matrix of new infections terms and ν for the matrix of the remaining transfer terms in our system, we get,
$f = \begin{pmatrix} \lambda S \\ 0 \end{pmatrix}, \ \nu = \begin{pmatrix} \Omega_1 I \\ -\phi I + \Omega_2 I \end{pmatrix}.$
Let $\mathrm{F}_1 = \lambda S$, $\mathrm{F}_2 = 0$, $\mathrm{F}_3 = \Omega_1 I$, $\mathrm{F}_4 = -\phi I + \Omega_2 I$.

We obtain the matrices F and V by finding the Jacobian matrices of f and ν evaluated at DFE point respectively to obtain.
$F = \begin{pmatrix} \frac{\partial F_1(E^0)}{\partial I} & \frac{\partial F_1(E^0)}{\partial T} \\ \frac{\partial F_2(E^0)}{\partial I} & \frac{\partial F_2(E^0)}{\partial T} \end{pmatrix}, \ V = \begin{pmatrix} \frac{\partial F_3(E^0)}{\partial I} & \frac{\partial F_3(E^0)}{\partial T} \\ \frac{\partial F_4(E^0)}{\partial I} & \frac{\partial F_4(E^0)}{\partial T} \end{pmatrix},$
This yield

$$F = \begin{pmatrix} \beta_1 S^0 & \beta_2 S^0 \\ 0 & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} \Omega_1 & 0 \\ -\phi & \Omega_2 \end{pmatrix}$$

$V^{-1} = \frac{1}{\Omega_1 \Omega_2} \begin{pmatrix} \Omega_2 & 0 \\ \phi & \Omega_1 \end{pmatrix}, FV^{-1} = \begin{pmatrix} \frac{\{\beta_1 \Omega_2 + \beta_2 \phi\} S^0}{\Omega_1 \Omega_2} & \frac{\beta_2 S^0}{\Omega_2} \\ 0 & 0 \end{pmatrix}$

The basic reproduction number is given by the spectral radius ζ (the dominant eigenvalue) of the matrix FV^{-1}, denoted by $\zeta(\mathrm{FV}^{-1})$. To obtain the eigenvalues η of FV^{-1} we solve the equation

$$|FV^{-1} - \eta H| = 0$$

where H is 2 by 2 identity matrix to obtain
$\begin{vmatrix} \frac{\{\beta_1 \Omega_2 + \beta_2 \phi\} S^0}{\Omega_1 \Omega_2} - \eta & \frac{\beta_2 S^0}{\Omega_2} \\ 0 & -\eta \end{vmatrix} = 0,$
$\eta^2 - \frac{\beta_1 \Omega_2 + \beta_2 \phi}{\Omega_1 \Omega_2} \eta = 0,$

The eigenvalues are $\eta_1 = 0$ and $\eta_2 = \frac{\{\beta_1\Omega_2+\beta_2\phi\}S^0}{\Omega_1\Omega_2}$

$$\mathrm{Rc} = \zeta(\mathrm{FV}^{-1}) = \frac{\beta_1 S^0}{\Omega_1} + \frac{\beta_2\phi S^0}{\Omega_1\Omega_2}$$

R_c is control reproduction number with treatment and natural immunity.
In absence of control measures such as treatment the basic reproduction number R_o is obtained
By observing that
$F = (\beta_1 S^0)$, $V = (\mu + \alpha + \tau)$,
giving the spectral radius of FV^{-1},
$R_o = \frac{\beta_1 S^0}{\mu+\alpha+\tau}$

3.3. Stability Analysis of DFE Point for Pneumonia Model

3.3.1 Local Stability of the DFE Point

Theorem 2.

The DFE of the system (1) - (3) is locally asymptotically stable whenever $R_C < 1$.

Proof

Let

$$f_1(S, I, T) = \pi - (\lambda + \mu)S + \gamma T + \tau I,$$
$$f_2(S, I, T) = \lambda S - \Omega_1 I,$$
$$f_3(S, I, T) = \phi I - \Omega_2 T.$$

To establish the local stability of E^o, we use the Jacobian of the model evaluated at E^o. Stability of this steady state is then determined based on the eigenvalues of the corresponding Jacobian which are functions of the model parameters.

$$J(E^0) = \begin{pmatrix} \frac{\partial f_1(E^0)}{\partial S} & \frac{\partial f_1(E^0)}{\partial I} & \frac{\partial f_1(E^0)}{\partial T} \\ \frac{\partial f_2(E^0)}{dS} & \frac{\partial f_2(E^0)}{dI} & \frac{\partial f_2(E^0)}{dT} \\ \frac{\partial f_3(E^0)}{\partial S} & \frac{\partial f_3(E^0)}{\partial I} & \frac{\partial f_3(E^0)}{\partial T} \end{pmatrix}$$

The Jacobian matrix for system (1) - (3) is given by

$$J = \begin{pmatrix} -(\mu+\lambda) & -\beta_1 S + \tau & -\beta_2 S + \gamma \\ \lambda & \beta_1 S - \Omega_1 & \beta_2 S \\ 0 & \phi & -\Omega_2 \end{pmatrix}$$

The Jacobian of the model at E^o is:

$$J(E^0) = \begin{pmatrix} -\mu & -\beta_1 S^0 + \tau & -\beta_2 S^0 + \gamma \\ 0 & \beta_1 S^0 - \Omega_1 & \beta_2 S^0 \\ 0 & \phi & -\Omega_2 \end{pmatrix}$$

Solving the equation
$|J(E^0) - \eta H| = 0$,
Where H is an identity matrix we obtain

$$\begin{vmatrix} -\mu-\eta & -\beta_1 S^0 + \tau & -\beta_2 S^0 + \gamma \\ 0 & \beta_1 S^0 - \Omega_1 - \eta & \beta_2 S^0 \\ 0 & \phi & -\Omega_2 - \eta \end{vmatrix} = 0,$$

$$(-\mu-\eta)\begin{vmatrix} \beta_1 S^0 - \Omega_1 - \eta & \beta_2 S^0 \\ \phi & -\Omega_2 - \eta \end{vmatrix} = 0,$$

$\eta_1 = -\mu$,

$$\eta^2 + \eta(\Omega_2 - \beta_1 S^0 + \Omega_1) - \beta_2 S^0\Omega_2 + \Omega_1\Omega_2 - \phi\beta_2 S^0 = 0,$$

$$\eta^2 + \eta(\Omega_1[1 - \frac{\beta_1 S^0}{\Omega_1}] + \Omega_2) + \Omega_1\Omega_2\{1 - (\frac{\beta_1 S^0}{\Omega_1} + \frac{\beta_2\phi S^0}{\Omega_1\Omega_2})\} = 0,$$

$$\eta^2 + \eta(\Omega_1\{1 - R_C\} + \frac{\beta_2\phi S^0}{\Omega_1\Omega_2} + \Omega_2) + \Omega_1\Omega_2\{1 - R_C\} = 0,$$

A general quadratic of form
$\eta^2 + B\eta + C = 0$,
$\eta_i = -\frac{B}{2} \pm \sqrt{\{(\frac{B}{2})^2 - C\}}$.
For negative real part $B > 0$, $C \geq 0$,
$1 - R_C > 0$,
$R_C < 1$.
this proves theorem 2.

3.3.2. Global Stability of the DFE Point

Theorem 3

The DFE is globally stable if $\frac{\gamma\phi}{\Omega_1\Omega_2}+\frac{\tau}{\Omega_1}+\frac{S^0}{S}=0$ [11].

Proof

We propose the following Lyapunov function

$$\mathrm{L(S,I,T)}=S-S^0-S^0Ln\frac{\mathrm{S}}{S^0}+\mathrm{XI}+YT$$

it satisfies the conditions;

$\mathrm{L}(S^0,I^0,T^0)\ =\ 0,$ (i)

$\mathrm{L}(S,I,\mathrm{T})\ >\ 0,$ (ii)

therefore $\mathrm{L(S,I,T)}$ is positive definite.

For $\frac{\mathrm{dL(S,I,T)}}{\mathrm{dt}}$ to be negative definite, it must satisfies

$\frac{\mathrm{dL}(S^0,I^0,T^0)}{\mathrm{dt}}=0$ (iv)

$\frac{\mathrm{dL(S,I,T)}}{\mathrm{dt}}<0$ (v)

where X and Y are positive constants to be determined.

At DFE point for our system $\mathrm{E}^0=(S^0,I^0,T^0)$,from equation (i) above

$$\mathrm{L}\ (S^0,I^0,T^0)=\ S^0-S^0-S^0ln\frac{S^0}{S^0}+\mathrm{X}\ I^0+\mathrm{Y}T^0$$
$$=0$$

The DFE point for our system $\mathrm{E}^0=\ (S^0,I^0,T^0)$ satisfies,

$$\pi=\beta_1\mathrm{I}^0\mathrm{S}^0+\beta_2T^0\mathrm{S}^0+\mu\mathrm{S}^0+\gamma T^0+\tau\mathrm{I}^0,$$
$$=\mu\mathrm{S}^0.$$

Determining the differential of equation (i)

$$\frac{\mathrm{dL(S,I,T)}}{\mathrm{dt}}=(1-\frac{\mathrm{S}^0}{\mathrm{S}})\frac{\mathrm{dS}}{\mathrm{dt}}+\mathrm{X}\frac{\mathrm{dI}}{\mathrm{dt}}+\mathrm{Y}\frac{\mathrm{dT}}{\mathrm{dt}}$$

$$=(1-\frac{\mathrm{S}^0}{\mathrm{S}}(\ \mu\mathrm{S}^0-\beta_1\mathrm{IS}-\beta_2\mathrm{TS}-\mu\mathrm{S}+\gamma\mathrm{T}+\tau\mathrm{I})+\mathrm{X}(\beta_1\mathrm{IS}+\beta_2\mathrm{TS}-\Omega_1\mathrm{I})+\mathrm{Y}(\phi\mathrm{I}\ -\Omega_2\mathrm{T}),$$

$$=-\mu\frac{(\mathrm{S}-\mathrm{S}^0)^2}{\mathrm{S}}-\beta_1\mathrm{IS}-\beta_2\mathrm{TS}+\gamma\mathrm{T}+\tau\mathrm{I}+\beta_1\mathrm{IS}^0+\beta_2\mathrm{TS}^0-\gamma\mathrm{T}\frac{\mathrm{S}^0}{\mathrm{S}}-\tau\mathrm{I}\frac{\mathrm{S}^0}{\mathrm{S}}+\mathrm{X}\beta_1\mathrm{IS}+\mathrm{X}\beta_2\mathrm{TS}-\mathrm{X}\Omega_1\mathrm{I}$$
$$+\mathrm{Y}\phi\mathrm{I}\ -\mathrm{Y}\Omega_2\mathrm{T}.$$

Now the positive constants X and Y are chosen such that the coefficients of I and T to zero we obtain the following equation,

$\mathrm{Y}\phi\ +\tau\ -\mathrm{X}\,\Omega1\ =0,$

$\gamma\ -\mathrm{Y}\Omega_2=0,$

Solving

$X=\frac{\gamma\phi}{\Omega_1\Omega_2}+\frac{\tau}{\Omega_1}, Y=\frac{\gamma}{\Omega_2}$

Substituting for X and Y in equation (i) we obtain

$$\mathrm{L(S,I,T)}=S-S^0-S^0ln\frac{\mathrm{S}}{S^0}+\left\{\frac{\gamma\phi}{\Omega_1\Omega_2}+\frac{\tau}{\Omega_1}\right\}\mathrm{I}+\frac{\gamma}{\Omega_2}T$$

$$\frac{\mathrm{dL(S,I,T)}}{\mathrm{dt}}=(1-\frac{\mathrm{S}^0}{\mathrm{S}})\frac{\mathrm{dS}}{\mathrm{dt}}+\left\{\frac{\gamma\phi}{\Omega_1\Omega_2}+\frac{\tau}{\Omega_1}\right\}\frac{\mathrm{dI}}{\mathrm{dt}}+\frac{\gamma}{\Omega_2}\frac{\mathrm{dT}}{\mathrm{dt}}$$

$$\frac{\mathrm{dL(S,I,T)}}{\mathrm{dt}}=\left(1-\frac{\mathrm{S}^0}{\mathrm{S}}\right)(\ \mu\mathrm{S}^0-\beta_1\mathrm{IS}-\beta_2\mathrm{TS}-\mu\mathrm{S}+\gamma\mathrm{T}+\tau\mathrm{I})+\left\{\frac{\gamma\phi}{\Omega_1\Omega_2}+\frac{\tau}{\Omega_1}\right\}(\beta_1\mathrm{IS}+\beta_2\mathrm{TS}-\Omega_1\mathrm{I}$$
$$+\frac{\gamma}{\Omega_2}(\phi\mathrm{I}\ -\Omega_2\mathrm{T})$$
$$=-\mu\frac{(\mathrm{S}-\mathrm{S}^0)^2}{\mathrm{S}}-\left(1-\frac{\mathrm{S}^0}{\mathrm{S}}\right)(\beta_1\mathrm{I}+\beta_2\mathrm{T})\mathrm{S}+\left\{\frac{\gamma\phi}{\Omega_1\Omega_2}+\frac{\tau}{\Omega_1}\right\}(\beta_1\mathrm{I}+\beta_2\mathrm{T})\mathrm{S}+\gamma\mathrm{T}+\tau\mathrm{I}-\gamma\mathrm{T}\frac{\mathrm{S}^0}{\mathrm{S}}$$
$$-\tau\mathrm{I}\frac{\mathrm{S}^0}{\mathrm{S}}\frac{\gamma\phi\Omega_1\mathrm{I}}{\Omega_1\Omega_2}-\frac{\tau\Omega_1\mathrm{I}}{\Omega_1}+\frac{\gamma\phi\mathrm{I}}{\Omega_2}-\gamma\mathrm{T}.$$
$$=-\mu\frac{(\mathrm{S}-\mathrm{S}^0)^2}{\mathrm{S}}+S(\beta_1\mathrm{I}+\beta_2\mathrm{T})\{\frac{\gamma\phi}{\Omega_1\Omega_2}+\frac{\tau}{\Omega_1}-\left(1-\frac{\mathrm{S}^0}{\mathrm{S}}\right)\}-\frac{\mathrm{S}^0}{\mathrm{S}}(\gamma\mathrm{T}+\tau\mathrm{I})$$
$$=-\mu\frac{(\mathrm{S}-\mathrm{S}^0)^2}{\mathrm{S}}+S(\beta_1\mathrm{I}+\beta_2\mathrm{T})\left\{\frac{\gamma\phi}{\Omega_1\Omega_2}+\frac{\tau}{\Omega_1}+\frac{\mathrm{S}^0}{\mathrm{S}}\right\}-S(\beta_1\mathrm{I}+\beta_2\mathrm{T})-\frac{\mathrm{S}^0}{\mathrm{S}}\gamma\mathrm{T}+\tau\mathrm{I})$$

$\frac{\mathrm{dL(S,I,T)}}{\mathrm{dt}}<0$ if $\frac{\gamma\phi}{\Omega_1\Omega_2}+\frac{\tau}{\Omega_1}+\frac{\mathrm{S}^0}{\mathrm{S}}\leq0$

Since,

$\gamma, \tau, \phi, \Omega_1, \Omega_2, S, S^0 \geq 0$ *[11]*.

3.4. Existence and Stability Analysis of the Endemic Equilibrium Point (EEP) for the Model

3.4.1. Existence of Endemic Equilibrium point.

Let $E^* = (S^*, I^*, T^*)$ represents an arbitrary endemic equilibrium of the model (1) - (3).
For which at least one of the infected components of the model (1) - (3) is non-zero).This implies
$\lambda^* = \beta_1 I^* + \beta_2 T^*$ which can be expressed as,

$$\lambda^* = R_C \Omega_1 \frac{I^*}{S^0} = \frac{\mu}{\pi} R_C \Omega_1 I^* \qquad \text{(i)}$$

be the forces of infection at steady-state.
Solving the equations of the model (1)-(3) at steady-state gives:

$$S^* = \frac{\pi + \gamma T^* + \tau I^*}{\mu + \lambda^*},\ I^* = \frac{\lambda^*}{\Omega_1}\left[\frac{\pi + \gamma T^* + \tau I^*}{\mu + \lambda^*}\right],\ T^* = \frac{\phi \lambda^*}{\Omega_2 \Omega_1}\left[\frac{\pi + \gamma T^* + \tau I^*}{\mu + \lambda^*}\right]$$

we obtain equations (ii) in term of λ*

$$I^* = \frac{\Omega_2 \lambda^* \pi}{\Omega_1 \Omega_2 (\lambda^* + \mu) - \lambda^* \gamma \phi - \Omega_2 \tau \lambda^*},$$

$$T^* = \frac{\phi \lambda^* \pi}{\Omega_1 \Omega_2 (\lambda^* + \mu) - \lambda^* \gamma \phi - \Omega_2 \tau \lambda^*}$$

$$S^* = \frac{1}{\lambda^* + \mu}\left\{\frac{[\pi \Omega_1 \Omega_2 (\lambda^* + \mu) - \lambda^* \gamma \phi - \Omega_2 \tau \lambda^*] + \pi \gamma \phi \lambda^* + \pi \tau \Omega_2 \lambda^*}{\Omega_1 \Omega_2 (\lambda^* + \mu) - \lambda^* \gamma \phi - \Omega_2 \tau \lambda^*}\right\}$$

Substituting I* in (i), and simplifying, gives,

$$\lambda^* = \frac{R_C \Omega_1 \Omega_2 \lambda^* \mu}{\Omega_1 \Omega_2 (\lambda^* + \mu) - \lambda^* \gamma \phi - \Omega_2 \tau \lambda^*}$$

$$\lambda^* = 0,\ \lambda^* = \frac{R_C \Omega_1 \Omega_2 \mu - \mu \Omega_1 \Omega_2}{\Omega_1 \Omega_2 - \gamma \phi - \Omega_2 \tau},$$

$$\lambda^* = \frac{\Omega_1 \Omega_2 \mu (R_C - 1)}{\Omega_1 \Omega_2 - \gamma \phi - \Omega_2 \tau}$$

$$\Omega_1 \Omega_2 - \gamma \phi - \Omega_2 \tau \geq 0$$

since

$$\Omega_1 \Omega_2 - \gamma \phi - \Omega_2 \tau = (\phi + \mu + \alpha)(\mu + \Psi) + \gamma(\mu + \alpha)$$

the positive endemic equilibrium of the model (1) - (3)

$$R_C > 1$$

Substituting for λ* we obtain equations (ii)

$$S^* = \frac{\pi}{\mu R_C},$$

$$I^* = \frac{\pi \Omega_2 (R_C - 1)}{R_C (\Omega_1 \Omega_2 - \gamma \phi - \tau \Omega_2)},$$

$$T^* = \frac{\phi \pi (R_C - 1)}{R_C (\Omega_1 \Omega_2 - \gamma \phi - \tau \Omega_2)}.$$

is the endemic equilibrium point E* [6].

3.4.2. Local Stability and Global Stability of the Endemic Equilibrium Point

For system (1) - (3), when $R_C > 1$, it has a unique positive EEP.

Theorem 5

The EEP is globally asymptotically stable if $R_C > 1$.

Proof

We propose the following Lyapunov function

$$K(S, I, T) = S - S^* - S^* ln \frac{S}{S^*} + C\left(I - I^* - I^* \mathrm{Ln} \frac{I}{I^*}\right) - D(T - T^* - T^* \mathrm{Ln} \frac{T}{T^*}).$$

where C and D are positive constants to be determined. This type of Lyapunov function has been mentioned in [1].
The positive equilibrium E* = (S*, L*,T^*) satisfies the following equations.

$$\pi = \beta_1 I^* S^* + \beta_2 T^* S^* + \mu S^* + \gamma T^* + \tau I^*,$$

$$(\phi + \mu + \alpha + \tau) I^* = \beta_1 I^* S^* + \beta_2 T^* S^*,$$

$$\phi I^* = (\mu + \gamma + \Psi) T^*.$$

We can now write the time derivative of L as

$$\frac{dK(S, I, T)}{dt} = (1 - \frac{S^*}{S})\frac{dS}{dt} + C\left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} + D\left(1 - \frac{T^*}{T}\right)\frac{dT}{dt}$$

$$\frac{dK(S, I, T)}{dt} = \left(1 - \frac{S^*}{S}\right)\beta_1 I^* S^* + \beta_2 T^* S^* + \mu S^* - \gamma T^* - \tau I^* - (\beta_1 I + \beta_2 T + \mu)S + \gamma T + \tau I]$$

$$+ C\left(1 - \frac{I^*}{I}\right)[(\beta_1 I + \beta_2 T)S - (\phi + \mu + \alpha + \tau)I] + D\left(1 - \frac{T^*}{T}\right)[\phi I - (\mu + \gamma + \Psi T]$$

$$= -\mu \frac{(S - S^*)^2}{S} + \beta_1 I^* S^* \left(1 - \frac{S^*}{S}\right) + \beta_2 S^* T^* \left(1 - \frac{S^*}{S}\right) - \gamma T^* - \tau I^* - \beta_1 IS - \beta_2 TS + \gamma T + \tau I +$$

$$\gamma T^* \frac{S^*}{S} + \tau I^* \frac{S^*}{S} + \beta_1 IS^* + \beta_2 TS^* - \gamma T \frac{S^*}{S} - \tau I \frac{S^*}{S} + C\left(1 - \frac{I^*}{I}\right)[(\beta_1 I + \beta_2 T)S - C(\phi + \mu + \alpha + \tau)I +$$

$$C(\beta_1 I^* S^* + \beta_2 T^* S^*) + D\phi I\left(1 - \frac{T^*}{T}\right) + D(\mu + \gamma + \Psi)T + D\phi I^* \qquad = -\mu \frac{(S - S^*)^2}{S} + \beta_1 I^* S^* \left(1 - \frac{S^*}{S}\right) +$$

$\beta_2 S^* T^* \left(1 - \frac{S^*}{S}\right) \gamma T^* - \tau I^* - \beta_1 IS - \beta_2 TS + \gamma T + \tau I + \beta_1 IS^* + \beta_2 TS^* - \gamma T \frac{S^*}{S} - \tau I \frac{S^*}{S} + \gamma T^* \frac{S^*}{S} + \tau I^* \frac{S^*}{S} - \tau I \frac{S^*}{S} + C\beta_1 IS + C\beta_2 TS - C\beta_1 SI^* - C\beta_2 T \frac{I^*}{I} - C(\phi + \mu + \alpha + \tau)I + C\beta_1 I^* S^* + C\beta_2 T^* S^* + D\phi I - D\phi I \frac{T^*}{T} - D(\mu + \gamma + \Psi)T + D\phi I^*$ Now the positive constants C and D are chosen such that the coefficients of SI, S T, I and T are equal to zero, that is,

$-\beta_1 + C\beta_1 = 0$ SI,

$-\beta_2 + C\beta_2 = 0$ ST,

$\tau - C(\phi + \mu + \alpha + \tau) + D\phi = 0$ I,

$\gamma - D(\mu + \gamma + \Psi) = 0$ T.

Solving the above equations yields

$C = 1, D = \frac{\gamma}{(\mu+\gamma+\Psi)} = \frac{\phi+\mu+\alpha}{\phi}$

The lyapunov function becomes

$$K(S, I, T) = S - S^* - S^* \mathrm{Ln} \frac{S}{S^*} + \left(I - I^* - I^* \mathrm{Ln} \frac{I}{I^*}\right) \frac{\gamma}{(\mu + \gamma + \Psi)} \left(T - T^* - T^* \mathrm{Ln} \frac{T}{T^*}\right) T$$

Or $K(S, I, T) = S - S^* - S^* \mathrm{Ln} \frac{S}{S^*} + (I - I^* - I^* \mathrm{Ln} \frac{I}{I^*} + \frac{\phi+\mu+\alpha}{\phi} (T - T^* - T^* \mathrm{Ln} \frac{T}{T^*})T$

And

$$\frac{dK(S, I, T)}{dt} = (1 - \frac{S^*}{S}) \frac{dS}{dt} + \left(1 - \frac{I^*}{I}\right) \frac{dI}{dt} + \frac{\gamma}{(\mu + \gamma + \Psi)} \left(1 - \frac{T^*}{T}\right) \frac{dT}{dt}$$

$$\frac{dK(S, I, T)}{dt} = (1 - \frac{S^*}{S})[(\lambda^* + \mu)S^* - \gamma T^* - \tau I^* - (\lambda + \mu)S + \gamma T + \tau I + \left(1 - \frac{I^*}{I}\right)[\lambda S - (\phi + \mu + \alpha + \tau)I] + \frac{\gamma}{(\mu + \gamma + \Psi)} \left(1 - \frac{T^*}{T}\right)[\phi I - (\mu + \gamma + \Psi T]$$

$$\frac{dK(S, I, T)}{dt} = -\mu \frac{(S - S^*)^2}{S} + \lambda^* S^* - \gamma T^* - \tau I^* - \lambda S + \gamma T + \tau I + \lambda S - (\phi + \mu + \alpha + \tau)I + (\phi + \mu + \alpha)I - \gamma T - \lambda^* S^* \frac{S^*}{S} + \gamma T^* \frac{S^*}{S} + \tau I^* \frac{S^*}{S} - \lambda S \frac{S^*}{S} - \gamma T \frac{S^*}{S} - \tau I \frac{S^*}{S} - \lambda S \frac{I^*}{I} + (\phi + \mu + \alpha + \tau)I^* - (\phi + \mu + \alpha)I \frac{T^*}{T} + \gamma T^*$$

$\frac{dK(S,I,T)}{dt} = -\mu \frac{(S-S^*)^2}{S} \lambda^* S^* - \lambda^* S^* \frac{S^*}{S} + \gamma T^* \frac{S^*}{S} + \tau I^* \frac{S^*}{S} + \lambda S \frac{S^*}{S} - \gamma T \frac{S^*}{S} - \tau I \frac{S^*}{S} - \lambda S \frac{I^*}{I} + (\phi + \mu + \alpha)I^* - (\phi + \mu + \alpha)I \frac{T^*}{T}$

$$\frac{dK(S, I, T)}{dt} = -\mu \frac{(S - S^*)^2}{S} + \lambda^* S \left(1 - \frac{S^*}{S}\right) + \gamma T^* \frac{S^*}{S} \left(1 - \frac{TS^*}{T^* S}\right) + \tau I^* \frac{S^*}{S} \left(1 - \frac{S^* I}{I^* S}\right) + \lambda S \left(\frac{S^*}{S} - \frac{I^*}{I}\right) + (\phi + \mu + \alpha)I^* \left(1 - \frac{IT^*}{I^* T}\right)$$

Consider $\lambda S(\frac{S^*}{S} - \frac{I^*}{I})$ since $\lambda(t) = \beta_1 I + \beta_2 T$

$$(\beta_1 I + \beta_2 T)S(\frac{S^*}{S} - \frac{I^*}{I}) = \beta_1 IS^* + \beta_2 TS^* - \beta_1 I^* S - \beta_2 TS \frac{I^*}{I},$$

$$= \beta_1 IS^* (1 - \frac{SI^*}{S^* I}) + \beta_2 TS^* (1 - \frac{SI^*}{IS^*}).$$

$$\lambda^* = R_C \Omega_1 \frac{I^*}{S^0}$$

Hence,

$$\frac{dK(S, I, T)}{dt} = -\mu \frac{(S - S^*)^2}{S} + R_C \Omega_1 I^* \frac{S^*}{S^0} \left(1 - \frac{S^*}{S}\right) + \gamma T^* \frac{S^*}{S} \left(1 - \frac{TS^*}{T^* S}\right) + \tau I^* \frac{S^*}{S} \left(1 - \frac{S^* I}{I^* S}\right) + \beta_1 IS^* \left(1 - \frac{SI^*}{S^* I}\right) + \beta_2 TS^* (1 - \frac{SI^*}{IS^*} + (\phi + \mu + \alpha)I^* (1 - \frac{IT^*}{I^* T})$$

since the arithmetic mean is greater than or equal to the geometric mean of the quantities

$\frac{S^*}{S} - 1 \geq 0$, $\frac{TS^*}{T^* S} - 1 \geq 0$, $\frac{S^* I}{I^* S} - 1 \geq 0$, $\frac{SI^*}{S^* I} - 1 \geq 0$, $\frac{SI^*}{IS^*} - 1 \geq 0$ and $\frac{IT^*}{I^* T} - 1 \geq 0$

Then $\frac{dK(S,I,T)}{dt} = 0$ holds only when S = S^*, I = I* and T = T^*: So the maximal compact invariant set in

{(S; E; I) ∈ ∏: $\frac{dK(S,I,T)}{dt} = 0$ } EEP is the only singleton using Lasalle's invariance principle [1, 12].

$\frac{dK(S,I,T)}{dt} < 0$

If $R_C > 1$

4. Numerical Simulation

To observe the dynamics of pneumonia model over time, numerical simulations are done using Matlab software. The parameters in table 1 are used in simulation based on the data of children under five years of age. Some values

assigned to the parameters have been derived from epidemiological literature while others are estimated. The red line represents Infectious children, the blue line represent susceptible children and the black line represent treated children.

The results obtained are shown in figures (1 - 3) after varying parameters τ and ϕ.

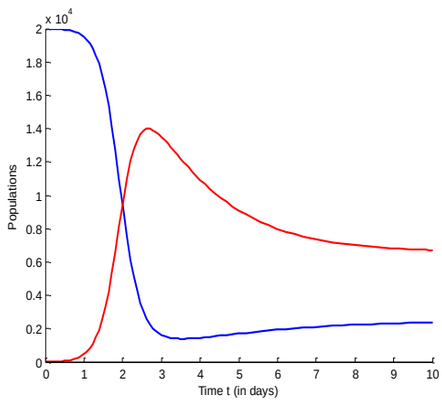

Figure-1. τ = 0, ϕ = 0

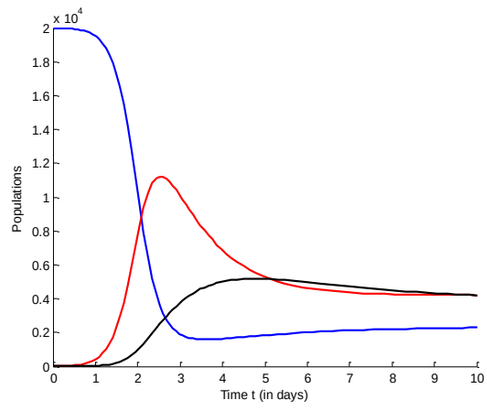

Figure-2. τ = 0.55, ϕ = 0.3545

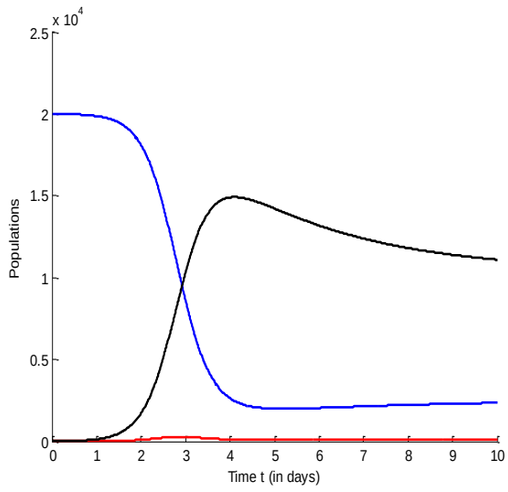

Figure-3. τ = 8, ϕ = 50

Explanation;

Figure i; when pneumonia invades the population the number infected increases with time while the number susceptible children decreases with time until they reach endemic equilibrium point.

Figure ii; After intervening with critical treatment and recovery from natural immunity, the the number infectious children decreases until it reaches an equilibrium whereby number of infectious children is equal to number of treated children.

Figure iii; Suggest that any treatment at critical treatment and increase in recovery from natural immunity can reduce number of infectious individual to zero.

5. Interpretation of the Model and Biological Implication

5.1. Local and Global Stability of Disease Free Equilibrium (DFE) Point and Endemic Equilibrium Point (EEP).

When equilibrium point is locally stable all the point near it tends to move towards it over time. Equilibrium point is globally stable if all initial starting conditions lead to it over time.

DFE point of Model was locally stable; this means that if initial conditions were to start near DFE they would move to it over time but they do not always start at neighborhood of DFE point. DFE point of Model was globally unstable; this means all initial starting conditions would not lead to it over time hence urgent need of intervention because the disease can establish itself by attaining endemic equilibrium point.

EEP of Model was globally stable if and only if $R_C < 1$ all the point near it tend to move towards it over time, this means intervention by policy makers would eradicate pneumonia over time. Our aim is to make EEP unstable so that it switches to DFE point; this requires intervention measures like treating disease with high efficacy drugs and adequate preventive measures.

5.2. Equilibrium Points and Thresholds

$$R_C = \frac{\beta_1 S^0}{\phi + \mu + \alpha + \tau} + \frac{\beta_2 \phi S^0}{(\Psi + \gamma + \mu)(\phi + \mu + \alpha + \tau)}$$

The treatment threshold is determined by equating R_C to one and solving for ϕ^C(critical treatment)

$$1 = \frac{\beta_1 S^0}{\phi^C + \mu + \alpha + \tau} + \frac{\beta_2 \phi^C S^0}{(\Psi + \gamma + \mu)(\phi^C + \mu + \alpha + \tau)},$$

$$(\Psi + \gamma + \mu)(\phi^C + \mu + \alpha + \tau) = \beta_1 S^0(\Psi + \gamma + \mu) + \beta_2 \phi^C S^0,$$

$$\phi^C(\Psi + \gamma + \mu) - \beta_2 S^0 \phi^C = \beta_1 S^0(\Psi + \gamma + \mu) - (\Psi + \gamma + \mu)(\mu + \alpha + \tau),$$

$$\phi^C = \frac{\beta_1 S^0(\Psi+\gamma+\mu)}{(\Psi+\gamma+\mu)-\beta_2 S^0} + \frac{(\Psi+\gamma+\mu)(\mu+\alpha+\tau)}{\beta_2 S^0-(\Psi+\gamma+\mu)}.$$

When actual treatment is greater than critical treatment it can ensure total eradication of pneumonia i.e

$$\phi > \phi^C.$$

Also, treatment with sufficient coverage can succeed in eliminating infection when R_c is below unity. Because R_c measures the intensity of the epidemic, treatment, by lowering R_c, can have significant public health impact even if it fails to eliminate infection in a specific population.

Following McLean and Blower [10], a measure of treatment impact based on the reproduction numbers can be defined as

$$(U) = 1 + \frac{R_C}{R_0},$$

$$= 1 + \frac{\beta_2\phi}{\beta_1\Omega_2},$$

$$= 1 + \frac{\beta_2\phi}{\beta_1(\Psi+\gamma+\mu)}.:$$

Thus, population-level impact of treatment is always positive provided that effective drugs are used for treatment.

5.3. Sensitivity of Effective Control Number R_C.

It can be shown that $R_C = R_0$ when $\phi = 0$. As expected, the control reproduction number becomes the basic reproduction number in the absence of treatment. It is important to investigate the sensitivity of R_C to changes in the treatment and natural immunity parameters ϕ and τ respectively. It can be shown that R_C is partly inversely related to ϕ and partly directly and inversely related to ϕ.

Since $\beta_1 > \beta_2$,

$$\frac{\beta_1 S^0}{\phi+\mu+\alpha+\tau} > \frac{\beta_2\phi S^0}{(\Psi+\gamma+\mu)(\phi+\mu+\alpha+\tau)}$$

Hence when ϕ increase R_C decreases. Thus, differentiating R_C with respect to ϕ yields

$$\frac{dR_c}{d\phi} = \frac{\beta_1 S^0}{(\phi+\mu+\alpha+\tau)^2} + \frac{\beta_2 S^0}{(\Psi+\gamma+\mu)(\phi+\mu+\alpha+\tau)} - \frac{\beta_2\phi S^0}{(\Psi+\gamma+\mu)(\phi+\mu+\alpha+\tau)^2},$$

$$= \frac{-\beta_1 S^0(\Psi+\gamma+\mu)+\beta_2 S^0(\mu+\alpha+\tau)}{(\Psi+\gamma+\mu)(\phi+\mu+\alpha+\tau)^2}.$$

R_C is negatively related to ϕ, therefore, higher treatment rates with drugs with higher and faster recovery rates and lower failure rates will decrease the control reproduction number and the intensity of the endemic.

Likewise, R_C is negatively related to τ. This can be shown by partially differentiating R_c with respect to τ yielding

$$\frac{dR_c}{d\tau} = -\frac{\beta_1 S^0}{(\phi+\mu+\alpha+\tau)^2} - \frac{\beta_2\phi S^0}{(\Psi+\gamma+\mu)(\phi+\mu+\alpha+\tau)^2},$$

$$= -\frac{\beta_1 S^0(\Psi+\gamma+\mu)+\beta_2\phi S^0}{(\Psi+\gamma+\mu)(\phi+\mu+\alpha+\tau)^2}.$$

As τ increases R_c decreases. We conclude that high immunity level will decrease the control reproduction number and the intensity of the endemic.

6. Discussion and Conclusion

Our main objective in this paper was to provide a general mathematical explanation of pneumonia transmission dynamics, taking into consideration the role of natural immunity and treatment in the transmission. We considered the possibility of a child contracting all types of pneumonia as long as they exist in a population since children who are under five years of age have a weak immune system. Poor nutrition and inadequate breastfeeding in developing compromises child's health status making the body vulnerable to pneumonia causing pathogens.

The model that we have discussed here is based on the initial model that was studied by Otieno, et al. [6]. When studying the transmission dynamics of infectious diseases with an objective of suggesting control measures, it is important to consider the stability of equilibrium points. In this paper we have established basic reproduction number, effective reproduction number, existence and stability of the equilibrium points. Our main results indicate that the disease free equilibrium is locally stable but globally unstable. That means the diseases can invade and persist in population if not intervened. The global stability of the endemic equilibrium point is achieved when $R_C < 1$. This is a clear indication that the control measure for pneumonia through treatment and boosting child's immune system can completely eradicate pneumonia, this would require all infected children to seek proper treatment which may not be completely achieved.

The analytical results from this paper are in agreement with those of Matlab numerical simulations. However, we need some interesting results in the numerical simulation to quantify recovery from natural immunity which still remains a challenge.

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Appendix

Table-1. Summary of model variables and parameters.

Variable	Description
T(t)	Population of treated children.
I(t)	Population of Infected children,
S(t)	Population of susceptible children.
Parameters	Description
β_1	Infection rate with infected children
β_2	Infection rate with children under treatment
π	Recruitment rate
γ	Recovery rate due to treatment.
α	Rate of death due to disease in the infective class
μ	Constant natural death rate
Φ	Treatment Rate of infected children
Ψ	Death rate due to disease in treatment class
τ	Recovery rate due to natural immunity

Table-2. Terminologies in the pneumonia model and their meanings

Endemic	It is long term infection which stays in the population at least 10 to 20 years.
Susceptible population	Proportion of the children population who are free of infection but at risk of contracting the infection
Infectious population	Proportion of the children population with the disease causing pathogen and capable of transmitting the infection to other children on contact.
Treated population	Proportion of the children population with the disease causing pathogen under treatment and capable of transmitting the infection to other children on contact.
Infectious Disease	Diseases where individuals are infected by pathogen micro-organisms, for instance viruses, bacteria, fungi or other micro parasites.
Alveoli	Microscopic sacs in the lungs that absorb oxygen.
Morbidity	Impairments as a result of a disease
Mortality	Susceptibility to death
Pathophysiology	Medical displine that converges pathology and physiology
Virulence	The degree of pathogenicity of a microorganism as indicated by the severity of disease produced and the ability to invade the tissues of the host.

Table-3. Summary of parameters estimation in the model

Parameters	Value	Source
β_1	0.22	[4]
β_2	0.176	Estimated
π	μP(0)	[4]
γ	0.0476 to 0.0952	Estimated
α	0.33	[4]
μ	0.0002 per day	[4]
Φ	0.3545	Estimated
Ψ	0.132	Estimated
τ	0.0238 to 0.0476 per day	[4]

β_2 is estimated as 80% of β_1 since $\beta_1 > \beta_2$,
γ is estimated as 200% range of τ,
P (0) is estimated as;
P (0) = S (0) +I(0) +T(0) =20010,
Where,
S(0) = 20000, I(0) = 10, T(0) = 0.
Ψ is estimated as 40% range of α,
Φ is estimated at ϕ^C.