MATHEMATICAL MODELLING OF PNEUMONIA DYNAMICS OF CHILDREN UNDER THE AGE OF FIVE

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Abstract

This paper work was designed to study the effect of treatment on the transmission of pneumonia infection. 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, basic reproduction number, effective reproduction number, existences and stability of the equilibrium point were established. Using Lyaponov function we discovered that the disease free equilibrium is unstable. The results are presented in graphs and it is discovered that the spread of the infection will be greatly affected by the rate of treatment and natural immunity.

Keywords: Positivity and Boundedness, Basic Reproduction Number, Control Reproduction Number, Infection Free Points, Endemic Equilibrium point, Stability, Global Stability.

Introduction

Stevenson (2010) stated that pneumonia is from the Greek word – pneúmōn meaning lung and the, according to Feigin (2004), the symptoms were described by Hippocrates (c. 460 BC-370 BC). Pneumonia was regarded by William Osler in the 19th century as "the captain of the men of death" (Osler, 1901). Pneumonia, stated McLuckie (2009) and Leach (2009), is an inflammatory condition of the lung affecting primarily the microscopic air sacs known as alveoli. The disease is usually caused by infection with viruses or bacteria and less commonly by other microorganisms (McLuckie, 2009; Jeffrey, 2010). 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 (Liu and Zhang, 2011). 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 (Liu and Zhang, 2011).

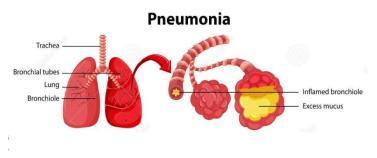


Figure 1: Pictorial representation of pneumonia infection

Pneumonia is a leading cause of morbidity and mortality in children. The infection continues to be a major contributor to childhood mortality and morbidity in developing countries including Nigeria (WHO, 2005). Pneumonia is responsible for a quarter of all deaths in under-five children. Many of the deaths occur in those less than 24 months especially in infants (WHO, 2005). A number of aetiologic agents, viruses and bacteria, have been associated with pneumonia, however it is the bacterial agents that are usually associated with severe pneumonia and result in complications or deaths. Streptococcus pneumonia and Haemophilus Influenzae remain the most important pathogens documented in previous studies (Rudan, Boschi-Pinto, Biloglav, Mulholland, and Campbell, 2008). Staphylococcus has also been found especially in patients with malnutrition. Pneumonia is the second cause of admission and deaths among children. Liu and Zhang (2011) reported that 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 (Liu and Zhang, 2011).

Mathematical models of infectious diseases have been used to successfully explain the transmission dynamics of many diseases and the use of such models has grown exponentially from mid-20th century (Hethcote, 2000). However, in this study, the transmission of pneumonia and the effect of treatment on children under age five are mathematically investigated

Model Formulation

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 infected class and class under treatment T(t). After treatment children with pneumonia can become susceptible again. Y is recovery rate, the recruitment rate of the susceptible class is π . Death due to disease occur at a rate of α in infection class, and μ is the natural death rate. M and N are infection rate in infection class and treatment class respectively. Φ Is the rate of treatment of children, ψ is the death rate due to disease during treatment and τ is the rate of recovery from infection state through natural immunity. Pneumonia infection occurs when susceptible individuals come into contact with infected individuals or those under treatment. If λ is force of infection at a given time, then;

 $\lambda = MI + NT$

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It is compulsory too note the following deduction

M > N, since we know that treatment reduces significantly level of infectiousness of an individual after contact.

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

 $A > \psi$ since treatment reduces likelihood of dying significantly...

Combining all the definitions and assumptions the model of transmission dynamics of pneumonia including the effect of treatment as proposed by *Otieno et al*, (2012). is given by the following

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

$$\frac{dI}{dt} = \lambda S - (\phi + \mu + \alpha + \tau)I _ (2)$$

$$\frac{dT}{dt} = \phi I - (\mu + \gamma + \psi)T _ (3)$$
Now,
$$P(t) = S(t) + I(t) + T(t) _ (4)$$

3.2 Model Analysis

We analyses the model for pneumonia transmission based on the following sub sections to determine all threshold parameter for pneumonia dynamics and effect of treatment.

3.2.1 Positivity and Boundednesss of the Solutions

Theorem: The region R given by

$$R = \left\{ \left(S, I, T\right) \in R_{+}^{3} \middle| S(0) \ge 0, I(0) \ge 0, T(0) \ge 0, P(t) = \frac{\pi}{\mu} \right\} \text{ is positive for all } t \ge 0$$

Proof: consider the equation (1)

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

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

The integrating factor is $e^{\int_0^t (\lambda + \mu) ds}$

$$\int \frac{d \left[S(t) e^{\int_{0}^{t} (\lambda + \mu) ds} \right]}{dt} \ge 0$$

$$S(t) e^{\int_{0}^{t} (\lambda + \mu) ds} \ge C$$

$$S(t) e^{(\lambda + \mu)t} \ge C$$

$$S(t) \ge C e^{-(\lambda + \mu)t}$$

$$At t = 0$$

 $S(0) \ge C$ Thus, $S(t) \ge S(0)e^{-(\lambda+\mu)t} \ge 0$ Hence $S \ge 0$ Thus S(t) stays positive Next, we consider equation (2), Let $\Omega = \phi + \mu + \alpha + \tau$ $\frac{dI}{dt} = \lambda S - \Omega I$ Then $\frac{dI}{dt} \ge -\Omega I$ Dividing both side by I and integrate $\frac{1}{I}\frac{dI}{dt} \ge -\Omega$ $\int \frac{1}{I} \frac{dI}{dt} \ge \int -\Omega dt$ $InI > -\Omega t + C$ Taking the exponential of both sides $e^{InI} > e^{-\Omega t + C}$ $I(t) \ge Ae^{-\Omega t}$ where $A = e^{C}$ At t = 0 $I(0) \ge A$ Thus $I(t) \ge I(0)e^{-\Omega t}$ It implies that $I(t) \ge I(0)e^{-\Omega t} \ge 0$ Also we consider the third equation (3) $\frac{dT}{dt} = \phi I - (\mu + \gamma + \psi)t \text{ Let } \beta = \mu + \gamma + \psi$ $\frac{dT}{dt} = \phi I - \beta t$ Then $\frac{dT}{dt} \ge \beta t$ $\frac{dT}{dt} - \beta t \ge 0$ The integrating factor is $e^{-\int_0^t \beta dt} = e^{-\beta t}$ Multiplying both side by the integrating factor

$$\int \frac{d}{dt} \left[Te^{-\beta t} \right] \ge 0$$
$$Te^{-\beta t} \ge C$$
$$T(t) \ge Ce^{\beta t}$$

At t = 0 $T(0) \ge C$ Thus, $T(t) \ge T(0)e^{-\beta t} \ge 0$ Hence $T(t) \ge 0$ Thus T(t) stays positive. Recall that P(t) = S(t) + I(t) + T(t) Thus $P(0) \ge 0$ And this is sufficient to show that P(t) is bounded in the region R and it remains positive for all values of t > 0P(t) = S(t) + I(t) + T(t) $\frac{dP}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dT}{dt}$ $\frac{dP}{dt} = (\pi - \lambda S - \mu S + \gamma T + \tau I) + (\lambda S - \phi I - \mu I - \alpha I - \tau I) + (\phi I - \mu T - \gamma T - \psi T)$ $\frac{dP}{dt} = \pi - \mu S - \mu I - \alpha I - \mu T - \psi T$ Collecting like term $\frac{dP}{dt} = \pi - \mu(S + I + T) - \alpha I - \psi T$ Since S + I + T = P $\frac{dP}{dt} = \pi - \mu P - \alpha I - \psi T$ $\frac{dP}{dt} + \mu P = \pi - \alpha I - \psi T$ $\frac{dP}{dt} + \mu P \le \pi$ The integrating factor is $e^{-\int_0^t \mu dt} = e^{-\mu t}$ Then we have

$$\frac{d}{dt} \Big[P e^{\mu t} \Big] \le \pi e^{-\mu t}$$

Integrating both sides

$$\int \frac{d}{dt} \left[P e^{\mu t} \right] \leq \int \pi e^{-\mu t} dt$$
$$e^{\mu t} P \leq \frac{\pi}{\mu} (e^{\mu t} + C)$$

Divide both sides by $e^{\mu t}$

$$P \le \frac{\frac{\pi}{\mu} (e^{\mu t} + C)}{e^{\mu t}}$$

$$P(t) \le \frac{\pi}{\mu} (1 + Ce^{-\mu t}) \text{ Where } C \text{ is the constant of integration}$$

$$\lim_{t \to 0} P(t) \le \frac{\pi}{\mu} (1 + Ce^{-\mu \infty})$$

 $\lim_{t \to 0} P(t) \le \frac{\pi}{\mu} (1) = \frac{\pi}{\mu}$ This proves the Boundedness of the solution inside R. This implies that

all solution of the system starting in R remain in R for all time $t \ge 0$. Thus R is positively invariant and attracting and hence it is sufficient to consider the dynamics of the system

Disease Free Equilibrum Points (DFE)

We can obtain the disease free equilibrium by setting all infectious classes and treatment classes to zero. It simply means a point where there is no existence of disease and nobody needed to be treated. So we have:

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

That is, $\mu S^0 = \pi$

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

The DFE point for the system is

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

Basic Reproduction Number R_{0} and Control Reproduction Number R_{C}

Using the next generation matrix method, we determine the basic reproduction number R_0 and control reproduction number R_c of the model. Let us represent the matrix of the new infections terms by f and matrix of the remaining transfer terms by v. fro our system, we get

$$f = \begin{pmatrix} \lambda S \\ 0 \end{pmatrix} \text{ and } v = \begin{pmatrix} \Omega I \\ -\phi I + \beta T \end{pmatrix}$$

Now let $F_1 = \lambda S, F_2 = 0, F_3 = \Omega I, F_4 = -\phi I + \beta T$

We can obtain the matrices F and V by finding the jacobian matrices of f and v evaluate at DFE point respectively, the jacobian matrice of F

$$F = \begin{pmatrix} \frac{\partial F_1(E^0)}{\partial I} & \frac{\partial F_1(E^0)}{\partial T} \\ \frac{\partial F_2(E^0)}{\partial I} & \frac{\partial F_2(E^0)}{\partial T} \end{pmatrix}$$

Recall that $\lambda = MI + NT$
So, $\frac{\partial F_1(E^0)}{\partial I} = \frac{\partial (MI + NT)S^0}{\partial I} = MS^0$
 $\frac{\partial F_1(E^0)}{\partial T} = \frac{\partial (MI + NT)S^0}{\partial T} = NS^0$
 $\frac{\partial F_1(E^0)}{\partial I} = 0$ since $F_2 = 0$
 $\frac{\partial F_2(E^0)}{\partial T} = 0$ since $F_2 = 0$ Thus
 $F = \begin{pmatrix} MS^0 & NS^0 \\ 0 & 0 \end{pmatrix}$

Also

$$V = \begin{pmatrix} \frac{\partial F_3(E^0)}{\partial I} & \frac{\partial F_3(E^0)}{\partial T} \\ \frac{\partial F_4(E^0)}{\partial I} & \frac{\partial F_4(E^0)}{\partial T} \end{pmatrix}$$

So,

$$\frac{\partial F_3(E^0)}{\partial I} = \frac{\partial \Omega I}{\partial I} = \Omega$$

$$\frac{\partial F_3(E^0)}{\partial T} = \frac{\partial \Omega I}{\partial T} = 0$$

$$\frac{\partial F_4(E^0)}{\partial I} = \frac{\partial (-\phi I + \beta T)}{\partial I} = -\phi$$

$$\frac{\partial F_4(E^0)}{\partial T} = \frac{\partial (-\phi I + \beta T)}{\partial T} = \beta$$
Thus,

$$V = \begin{pmatrix} \Omega & 0\\ -\phi & \beta \end{pmatrix}$$

The inverse matrix of V i.e V⁻¹ is calculated as follows

$$V = \begin{pmatrix} \Omega & 0 \\ -\phi & \beta \end{pmatrix}$$

The determinant
 $|V| = \Omega\beta + 0 = \Omega\beta$
Adjoint $V = \begin{pmatrix} \beta & 0 \\ \phi & \Omega \end{pmatrix}$ Thus,

$$V^{-1} = \frac{1}{|V|} Adj V = \frac{1}{\Omega\beta} \begin{pmatrix} \beta & 0 \\ 0 & \Omega \end{pmatrix} = \begin{pmatrix} \frac{1}{\Omega} & 0 \\ \frac{\phi}{\Omega\beta} & \frac{1}{\beta} \end{pmatrix}$$
Thus,

$$FV^{-1} = \begin{pmatrix} MS^0 & NS^0 \\ 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{\Omega} & 0 \\ \frac{\phi}{\Omega\beta} & \frac{1}{\beta} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} \frac{M\beta S^0 + N\phi S^0}{\Omega\beta} & \frac{NS^0}{\beta} \\ 0 & 0 \end{pmatrix}$$
So,

$$FV^{-1} = \begin{pmatrix} (\frac{M\beta + N\phi)S^0}{\Omega\beta} & \frac{NS^0}{\beta} \\ 0 & 0 \end{pmatrix}$$

The basic reproduction number is given by the dominant eigen value of the matrix FV^{-1} and we denote it with $d(FV^{-1})$. Let the eigen values be denoted by η . To obtain the eigen value, we solve the equation.

$$\begin{vmatrix} FV^{-1} - \eta E \end{vmatrix} = 0 \text{ Where H is } 2 \times 2 \text{ identity matrix, then} \\ \begin{vmatrix} FV^{-1} - \eta E \end{vmatrix} = 0 \\ \begin{vmatrix} \left(\frac{(M\beta + N\phi)S^0}{\Omega\beta} & \frac{NS^0}{\beta} \\ 0 & 0 \end{vmatrix} - \eta \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{vmatrix} = 0 \\ \begin{vmatrix} \left(\frac{(M\beta + N\phi)S^0}{\Omega\beta} & \frac{NS^0}{\beta} \\ 0 & 0 \end{vmatrix} - \eta \begin{pmatrix} \eta & 0 \\ 0 & \eta \end{pmatrix} \end{vmatrix} = 0$$

$$\begin{pmatrix} \frac{(M\beta + N\phi)S^0}{\Omega\beta} - \eta & \frac{NS^0}{\beta} \\ 0 & -\eta \end{pmatrix} = 0$$

Find the determinant, we have

$$\eta^2 - \frac{(M\beta + N\phi)S^0\eta}{\Omega\beta} = 0$$

We have the eigen value at 2 points i.e

$$\eta = 0$$
 and $\eta = \frac{(M\beta + N\phi)S^0}{\Omega\beta}$ (the points where H is equal to zero)

Thus since we need the dominant eigen value,

$$R_{c} = d(FV^{-1}) = \frac{M\beta S^{0}}{\Omega\beta} + \frac{N\phi S^{0}}{\Omega\beta} = \frac{MS^{0}}{\Omega} + \frac{N\phi S^{0}}{\Omega\beta}$$
 Note that R_{c} is the control number with

treatment and natural immunity. But in the absent of control measures such as treatment and basic reproduction number, R_0 is obtained.

Observed that

$$F = (\beta S^0), V = (\mu + \alpha + \tau)$$
 Giving the $d(FV^{-1})$ as
 $R_0 = (FV^{-1}) = \frac{\beta S^0}{\mu + \alpha + \tau}$

Stability Analysis of Disease Free Equibrum (DFE) Point

Theorem2: The DFE point of the system is locally asymptotically stable whenever $R_C < 1$ Proof: let

$$\begin{split} f_1 &= (S, I, T) = \pi - (\lambda + \mu)S + \gamma T + \tau I \\ f_2 &= (S, I, T) = \lambda S - \Omega I \\ f_3 &= (S, I, T) = \phi I - \beta T \end{split}$$

We use the Jacobian of the model evaluated at E^0 to establish the local stability of E^0 . The stability is determined based on the Eigen values of the corresponding Jacobian which are function of the model parameters.

$$J(E) = \begin{pmatrix} \frac{\partial F_1(E^0)}{\partial S} & \frac{\partial F_1(E^0)}{\partial I} & \frac{\partial F_1(E^0)}{\partial T} \\ \frac{\partial F_2(E^0)}{\partial S} & \frac{\partial F_2(E^0)}{\partial I} & \frac{\partial F_2(E^0)}{\partial T} \\ \frac{\partial F_3(E^0)}{\partial S} & \frac{\partial F_3(E^0)}{\partial I} & \frac{\partial F_3(E^0)}{\partial T} \end{pmatrix} \text{ roots } \lambda = MI + NT$$

$$J(E) = \begin{pmatrix} -(\lambda + \mu) & -MS^{0} + \tau & -NS^{0} + \gamma \\ \lambda & MS^{0} - \Omega & NS^{0} \\ 0 & \phi & -\beta \end{pmatrix}$$

Thus at DFE = E^o

$$J(E^{0}) = \begin{pmatrix} -\mu & -MS^{0} + \tau & -NS^{0} + \gamma \\ 0 & MS^{0} - \Omega & NS^{0} \\ 0 & \phi & -\beta \end{pmatrix}$$

Since we know that the origin value μ as stated under basic reproduction number, and our unit matrix μ £, than we solve the equation $|L(E^0) - mE| = 0$

$$\begin{aligned} \left| J(E^{0}) - \eta E \right| &= 0 \\ \left| \begin{pmatrix} -\mu & -MS^{0} + \tau & -NS^{0} + \gamma \\ 0 & MS^{0} - \Omega & NS^{0} \\ 0 & \phi & -\beta \end{pmatrix} \right| &= 0 \\ \left| \begin{pmatrix} -\mu - \eta & -MS^{0} + \tau & -NS^{0} + \gamma \\ 0 & MS^{0} - \Omega - \eta & NS^{0} \\ 0 & \phi & -\beta - \eta \end{pmatrix} \right| &= 0 \\ -\mu - \eta \begin{vmatrix} MS^{0} - \Omega - \eta & NS^{0} \\ \phi & -\beta - \eta \end{vmatrix} - (-MS^{0} + \tau) \begin{vmatrix} 0 & NS^{0} \\ 0 & -\beta - \eta \end{vmatrix} + (-NS^{0} + \gamma) \begin{vmatrix} 0 & MS^{0} - \Omega - \eta \\ 0 & \phi \end{vmatrix} = 0 \\ -\mu - \eta \begin{vmatrix} MS^{0} - \Omega - \eta & NS^{0} \\ \phi & -\beta - \eta \end{vmatrix} - (0 + 0) = 0 \\ \text{Let} - \mu - \eta_{1} = 0 \\ -\mu = \eta_{1} \end{aligned}$$

Thus we needed to solve the 2x2 matrix

$$\begin{array}{ccc} MS^{0}-\Omega-\eta & NS^{0}\\ \phi & -\beta-\eta \end{array}$$

$$(MS^{\circ}-\Omega-\eta)(-\beta-\eta-\phi NS^{\circ})$$

$$= (-M\beta S^{0} + \beta\Omega + \beta\eta - M\eta S^{0} + \Omega\eta + \eta^{2} - \phi NS^{0}) = 0$$

By re-arranging

$$(\eta^{2} + \eta(\beta - MS^{0} + \Omega) - M\beta S^{0} + \beta\Omega - \phi NS^{0}) = 0$$

$$(\eta^{2} + \eta \left(\Omega \left[1 - \frac{MS^{0}}{\Omega}\right] + \beta\right) + \beta \Omega \left(1 - \frac{MS^{0}}{\Omega} - \frac{\phi NS^{0}}{\Omega\beta}\right) = 0$$

Since $R_{c} = \frac{MS^{0}}{\Omega} + \frac{\phi NS^{0}}{\Omega\beta}$

Then we have

$$(\eta^{2} + \eta \left(\Omega \left[1 - R_{c} \right] + \frac{\phi N S^{0}}{\Omega \beta} + \beta \right) + \beta \Omega \left(1 - R_{c} \right) = 0$$

Comparing the to a quadratic equation of general from $\eta^2 + B\eta + C = 0$

$$\eta = \frac{-B \pm \sqrt{B^2 - 4AC}}{2A}$$
 Since A=1

For negative real part $B > 0, C \ge 0$ Then, we have $1 - R_C > 0$

 $R_C < 1$

Theorem proved

Global Stability of the Disease Free Equilibrium Point

The ΔFE is globally stable if $\frac{\gamma\phi}{\Omega\beta} + \frac{\tau}{\Omega} + \frac{S^0}{S} = 0$ (Chirove 2013)

Proof

Proposing the Lyaponov function below

$$L(S, I, T) = S - S^{0} - S^{0} \ln \frac{S}{S^{0}} + XI + YT$$
(i)
Which satisfy the condition

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

L(S,I,T)>0

Therefore L(S, I, T) is positive definite

Now, for the derivative of L(S, I, T) i.e. $\frac{dL(S, I, T)}{dt}$ to be negative definite, it must satisfies

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

Where X and Y are positive constants to be determined Now, at ΔFE point for our system

$$E^{0} = (S^{0}, I^{0}, T^{0})$$
From (1) above,

$$L(S, I, T) = S - S^{0} - S^{0} \ln \frac{S}{S^{0}} + XI + YT = 0$$
Since $I^{0} = 0$ and T^{0} and $\ln = 0$
Then at ΔFE point
 $T = \mu S^{0}$
Thus, the differential of equation (i)

$$\frac{L(S, I, T)}{dt} = \left(1 - \frac{S^{0}}{S}\right) \frac{dS}{dt} + X \frac{dI}{dt} + Y \frac{dT}{dt}$$
Substituting the values of $\frac{dS}{dt}, \frac{dI}{dt}, and \frac{dT}{dt}$ respectively
We have

$$\frac{L(S, I, T)}{dt} = \left(1 - \frac{S^{0}}{S}\right) (\mu S^{0} - MIS - NTS - \mu S + \gamma T + TI) + X (MIS - NTS - \Omega I) + Y (\phi I - \beta T)$$

$$= \frac{-\mu (S - S^{0})^{2}}{S} - MIS - NTS + YT + IT + MIS^{0} + NTS^{0} - \gamma T \frac{S^{0}}{S} - T \frac{S^{0}}{S} + XMIS + XNTS - X\Omega I + Y\phi I - \gamma\beta T$$
Extracting out the co-efficient of I and T and equating them to zero.
 $Y\phi I + \tau - X\Omega I = 0$ $\gamma - Y\beta T = 0$
 $(Y\phi + \tau - X\Omega)I = 0$ $(\gamma - Y\beta)T = 0$
 $Y\phi + \tau - X\Omega = 0$ * $\gamma - Y\beta = 0$
 $Y\beta = \gamma$
 $Y = \frac{\gamma}{\beta}$

Substituting
$$Y = \frac{\gamma}{\beta}$$
 in *
 $\frac{\gamma\phi}{\beta} + \tau - X\Omega = 0$
 $X\Omega = \frac{\gamma\phi}{\beta} + \tau$
 $X = \frac{\gamma\phi}{\beta\Omega} + \frac{\tau}{\Omega}$

Now substituting for X and Y in equation

$$\begin{split} L(S, I, T) &= S - S^{0} - S^{0} \ln \frac{S}{S^{0}} + \left(\frac{\gamma \phi}{\beta \Omega} + \frac{\tau}{\Omega}\right)I + \frac{\gamma}{\beta}T \\ \frac{L(S, I, T)}{dt} &= \left(1 - \frac{S^{0}}{S}\right)\frac{dS}{dt} + \left(\frac{\gamma \phi}{\beta \Omega} + \frac{\tau}{\Omega}\right)\frac{dI}{dt} + \frac{\gamma}{\beta}\frac{dT}{dt} \\ \frac{L(S, I, T)}{dt} &= \left(1 - \frac{S^{0}}{S}\right)(\mu S^{0} - MIS - NTS - \mu S + \gamma T + TI) + \left(\frac{\gamma \phi}{\beta \Omega} + \frac{\tau}{\Omega}\right)(MIS - NTS - \Omega I) + \frac{\gamma}{\beta}(\phi I - \beta T) \\ &= \frac{-\mu(S - S^{0})^{2}}{S} - \left(1 - \frac{S^{0}}{S}\right)(MIS - NTS) + \left(\frac{\gamma \phi}{\beta \Omega} + \frac{\tau}{\Omega}\right)(MIS + NTS) \\ &+ YT + IT - \gamma T\frac{S^{0}}{S} - T\frac{S^{0}}{S}\frac{\gamma \phi \Omega I}{\Omega \beta} - \frac{\tau \Omega I}{\Omega} + \frac{\gamma \phi I}{\beta} - \gamma T \\ &= \frac{-\mu(S - S^{0})^{2}}{S} - S(MI - NT) + \left(\frac{\gamma \phi}{\beta \Omega} + \frac{\tau}{\Omega} - \left(1 - \frac{S^{0}}{S}\right)\right) + \frac{S^{0}}{S}(YT + TI) \\ &= \frac{-\mu(S - S^{0})^{2}}{S} - S(MI - NT) + \left(\frac{\gamma \phi}{\beta \Omega} + \frac{\tau}{\Omega} + \frac{S^{0}}{S}\right) - S(MI + NT) + \frac{S^{0}}{S}(YT + TI) \\ &= \frac{-\mu(S - S^{0})^{2}}{S} - S(MI - NT) + \left(\frac{\gamma \phi}{\beta \Omega} + \frac{\tau}{\Omega} + \frac{S^{0}}{S}\right) - S(MI + NT) + \frac{S^{0}}{S}(YT + TI) \end{split}$$

Since $\gamma, \tau, \phi, \Omega, \beta, S, S^0 \ge 0$

4.1 Numerical Simulation

With the aim of observing the dynamics of pneumonia model over time, numerical simulations are done using Maple 13 software. We make use of the parameters in Table 1 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 susceptible children, the blue line represent infectious children and the black line represent treated children.

The results obtained are shown in Figures (1-3) after varying the rate critical treatment and recovery from natural immunity.

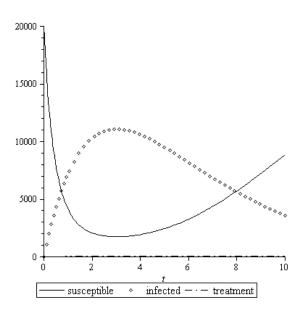


Figure 2: Graphical representation of susceptible, infected and treated population

Explanation

Figure 1; at the time when pneumonia is introduced to the population the number infected increases while the number susceptible children decreases gradually with time.

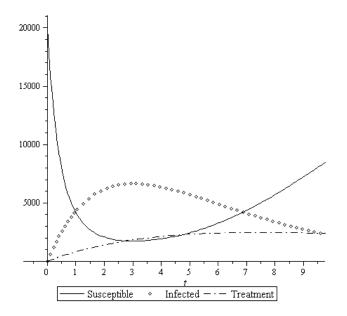


Figure 3: Graphical representation of susceptible, infected and treated population

In Figure 2, the number of infectious children decreases until it reaches an equilibrium (number of infectious children is equal to number of treated children) after introducing critical treatment and recovery from natural immunity.

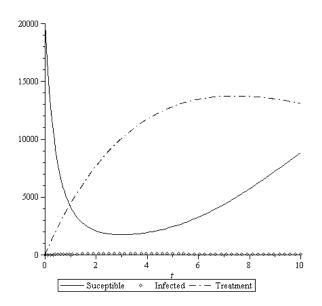


Figure 4: Graphical representation of susceptible, infected and treated population In Figure 3, Shows that the number of infectious individual was reduced to zero when treatment is at critical treatment and recovery from natural immunity increased.

| Variables parameters | and | Description | Estimation |
|-------------------------|-----|---|-------------------------|
| T(t) | | Population of treated children | 20000 |
| I(t) | | Population of Infected children | 10 |
| S(t) | | Population of susceptible children | 0 |
| М | | Infection rate with infected children | 0.22 |
| Ν | | Infection rate with children under treatment | 0.176 |
| π | | Recruitment rate | μ P (0) |
| γ | | Recovery rate due to treatment | 0.0476 to 0.0952 |
| α | | Rate of death due to disease in the infective class | 0.33 |
| μ | | Constant natural death rate | 0.0002 per day |
| Φ | | Treatment rate of infected children | 0.3545 |
| ψ | | Death rate due to disease in treatment class | 0.132 |
| τ | | Recovery rate due to natural immunity | 0.0238 to 0.0476 per |

Table 1: Model variables and parameter with description and their estimation

M is estimated as 80% of N since M > N, 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 , \emptyset is estimated at \emptyset^{C}

Discussion and Conclusion

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.

day

Our main results indicate that the disease free equilibrium is unstable. This means that the diseases can invade and persist in population if not intervened. 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. Therefore we propose a mathematical model which is based on the initial model that was studied by Otieno, *et al* (2012).

The analytical results from this paper are in agreement with those of Maple13 software.

Recommendation

This research suggests that government should increase the rate of treatment of pneumonia infection and find various means to boost child's immunity within population if the infection will be eradicate.

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