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Mathematical Modelling and Analysis of Treatment and Screening of Pneumonia

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Abstract

Pneumonia is one of the leading causes of serious illness and deaths among children under five years of age in Tanzania and around the world. In this paper, a mathematical model of the transmission dynamics of pneumonia with screening and treatment is formulated and analysed with the aim of understanding its transmission dynamics and the effects of these interventions. The conditions for the clearance or persistence of the pneumonia infection through the stability of the equilibria are derived. The model reproduction number, R_{ST}, is derived and the stability of equilibrium points is analysed. The results of the analysis shows that there exist a locally stable disease free equilibrium point, E_0 when $R_{ST} < 1$ and a unique endemic equilibrium, E_1 when $R_{ST} > 1$.

Key words: Pneumonia, Pneumococcus, Dynamics, Screening, Treatment.

1. Introduction

Pneumonia is one of the forgotten killer diseases (WHO, 2005). The disease is endemic and claims many lives. It is the second biggest killer of children in Tanzania, after malaria (Samarasekera, 2009), it is the leading cause of childrens' death in Africa (WHO,2006) and it kills more children per year than any other illness in the world. The death rates are around 2 million children worldwide, every year (WHO, 2005). Mathematical models of the dynamics of this disease with special emphasis on Tanzania are uncommon.

Pneumonia is an inflammatory condition of the lungs affecting the microscopic air sacs (alveoli) and is usually associated with fever, chest symptoms, and lack of air space (consolidation) on a chest (McLuckie and Leach, 2009). It is typically caused by infection. Infectious agents include; bacteria, viruses, fungi and parasites (Luckie, 2009). The disease has a wide range of aetiological factors (causative factors). Classic pneumonia is normally caused by *Streptococcus pneumoniae* (*pneumococcus*) (Dunn, 2005). *Streptococcus pyogenes* and *Staph aureus* may also cause pneumonia in debilitated adult patients. *Pneumocystosis* is commonly found in the lungs of healthy people, but being a source of opportunistic infection, it can cause a lung infection in people with a weak immune system.

Pneumococcus is spread through contact with people who are ill or who carry the bacteria in their throat. You

can get pneumococcal pneumonia from respiratory droplets from the nose or mouth of an infected person. It is common for people, especially children, to carry the bacteria in their throats without being sick. After a person is infected and diagnosed with pneumonia, he will be on medication for a particular period of time, the infection is contagious for 10 to 14 days after the infected person stops getting treatment (WHO/UNICEF, 2006).

The individuals that are at high risk of infection are children (under the age of 5years), the elderly (above 65years of age), and individuals with long-term health problem such as heart disease, sickle cell disease, alcoholism, lung disease (not including asthma), diabetes, or liver cirrhosis (WHO, 2005).

Prevention of pneumonia includes vaccination, environmental measures, screening and appropriately treating other diseases (Singh and Aneja, 2011).Methods of prevention of pneumonia infection among newborn infants include testing pregnant women for Group B *Streptococcus* and *Chlamydia trachomatis*, and providing antibiotic treatment (Dunn, 2005). Suctioning the mouth and throat of infants with meconium-stained amniotic acid decreases the rate of aspiration pneumonia (Singh and Aneja, 2011). Environmental prevention methods include reduction of indoor air pollution as well as smoking cessation and hand-washing when around a person with pneumonia, since the bacteria and viruses can also be spread through one's hands and then to the mouth. Treating other infections such as heart diseases, lung diseases, diabetes, sickle cell disease, alcoholism and AIDS also reduces the risk of pneumonia (Singh and Aneja, 2011).

Screening refers to the use of tests and examinations to find a disease, such as cancer, HIV/AIDS and pneumonia in people who do not have any symptoms. It reduces morbidity and mortality due to the diseases (Nygard, 2011; Ngiliule, 2014). Screening tests offer the best chance to identify and detect the disease at the early stages.

Research (Ruuskanen *et al*, 2011) has shown that prevention and proper treatment of pneumonia could avert one million deaths in children every year. Treatment alone can save at least 600 000 deaths annually. Treating underlying illnesses (such as AIDS) can decrease a person's risk of pneumonia (Ruuskanen *et al*, 2011; WHO/UNICEF, 2005). This paper investigates mathematically the dynamics of pneumonia disease with screening and treatment interventions.

The outline of the rest of the paper is as follows; section 2: model formulation, section 3: model analysis, section 4: sensitivity analysis, section 5: numerical simulations and discussion.

2. Model Formulation

In a compartmental model, the disease is assumed to have several progress stages. Individuals move between these stages with specific rates (i.e. each individual can only be in one compartment at a time). It is assumed that all individuals in each of the compartments have similar characteristics in that all are in the same stage of the disease progress, and as soon as an individual enters a compartment, there is no difference between him/her and other individuals in that compartment, this assumption is referred to as the "homogeneity assumption". This assumption originates from the fact that, the waiting time in each compartment is exponentially distributed and hence memoryless (Hamed *et al*, 2010).

We consider a model that describes the dynamics of pneumonia infection among four (4) sub–populations, namely the susceptibles (S), the asymptomatic Infectives (or simply carriers) (I_c), the symptomatic Infectives (I_i), the treated Infectives (T). We assume that the susceptible population is generated through birth at a constant rate

π as well as by recovery of infectives naturally or by treatment. The susceptible population is decreased through

infection with pneumonia at a rate λ, where the proportion q become symptomatic infectious immediately and join the symptomatic infectious population and the remaining proportion $1-q$ become asymptomatic infectious, it is also decreased by natural mortality at a constant rate μ. The asymptomatic infectious population is decreased through screening method at a rate α where they progress to symptomatic infectiousness and through recovery by gaining temporary immunity (Ong'ala *et al*, 2012) at the rate γ_c, it is also decreased by natural mortality at a constant rate μ. The symptomatic infectious population is increased through the change of status of the asymptomatic infectious class to symptomatic infectious through screening at a rate α and is decreased through natural recovery and natural death (at a constant rate γ_c and μ, respectively). The symptomatic infective population is increased by transfer of asymptomatic individuals who show the symptoms at the screening rate α, the infected proportion q of the susceptible individuals at the rate λ, the vaccinated individuals at the rate ρλ, it is also decreased by the treated individuals at the rate σ, the individuals who recovers naturally at the rate γi, and both disease induced death rate and natural mortality (at the rate δ and μ respectively). The treated infective population gains by administration of treatment at a constant rate σ. Treated infectives recover at a constant rate ωγ and join a class of susceptible. This class is also decreased both natural and disease induced deaths ($\mu+\tau\delta$). Since, a population dynamics model is considered; all the state variables and parameters are assumed to be non-negative.

Based on the model variables and parameters, the dynamics of the basic pneumonia model are described by the compartmental model shown in Figure 1.

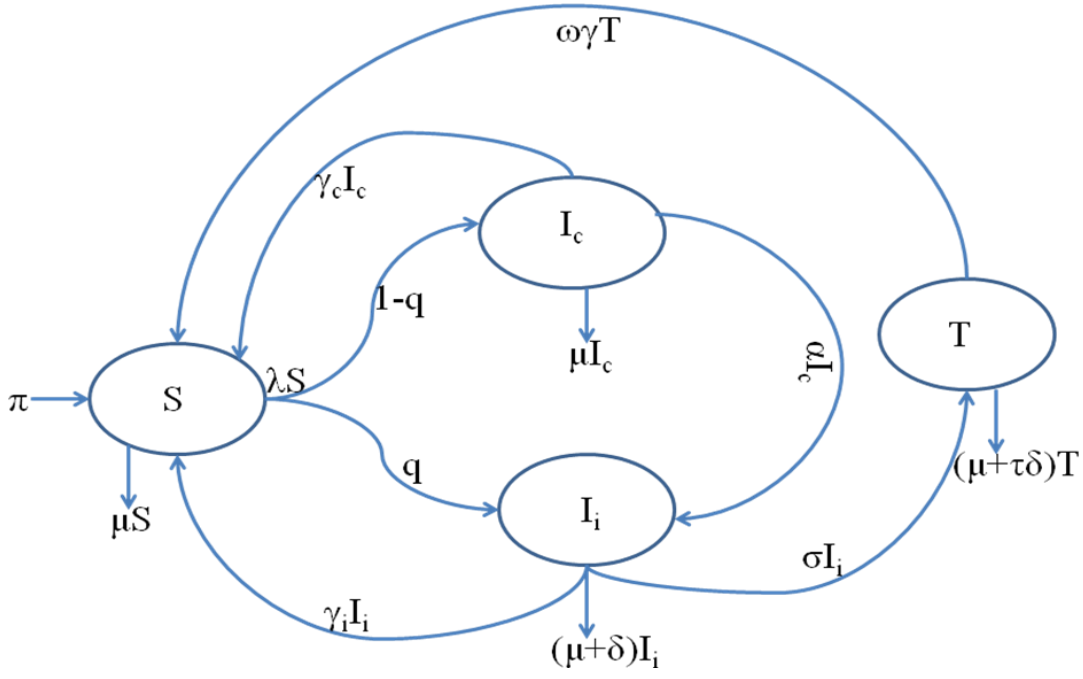

Figure 1: A compartmental diagram for a pneumonia model with screening and treatment interventions.

Applying the assumptions and the relationships that exist between the variables and parameters (Figure 1), the basic dynamics of pneumonia are described by the system of four ordinary non-linear differential equations given by;

$$\begin{aligned}
\frac{dS}{dt} &= \pi + \gamma_i I_i + \gamma_c I_c + \omega\gamma T - q\lambda S - (1-q)\lambda S - \mu S \\
\frac{dI_c}{dt} &= (1-q)\lambda S - (\mu + \gamma_c + \alpha) I_c \\
\frac{dI_i}{dt} &= q\lambda S + \alpha I_c - (\mu + \gamma_i + \sigma + \delta) I_i \\
\frac{dT}{dt} &= \sigma I_i - (\mu + \omega\gamma + \tau\delta) T,
\end{aligned} \tag{1}$$

with initial conditions $S(0) = S_0, I_c(0) = I_{c0}, I_i(0) = I_{i0}, T(0) = T_0$, and where the force of infections,

$$\lambda = c\beta\left(\frac{I_i + \nu I_c}{N}\right). \tag{2}$$

$N = S + I_c + I_i + T$ is the total population size which is changing at the rate;

$$\frac{dN}{dt} = \pi - \mu N - \delta(I_i + \tau T) \tag{3}$$

and the parameter β is the transmission rate. It is worth noting that $\beta = c\beta_0$, where c denotes the average number of effective contact and β_0 the probability of infection, ω and τ are the modification parameters, with $\omega \geq 1$ implying that, treatment increase recovery rate and $0 \leq \tau, q \leq 1$.

3 The Model Analysis

In this section, the basic properties of model system (1) useful for the study and proofs of stability of the system are outlined. The model properties are employed to establish criteria for positivity of solutions and well-posedness of the system.

3.1 Invariant Region

The initial value problem modeled by system (1) is well defined when supplemented with non-negative initial conditions. In the absence of the disease, the population size N approaches the carrying capacity. Since $\frac{dN}{dt} = \pi - \mu N - \delta(I_i + \tau T)$ can be written as $\frac{dN}{dt} \leq \pi - \mu N$, solution starting in the positive orthant $\mathbb{R}^4_+$ eventually enters the subset of $\mathbb{R}^4_+$ defined by

$$\Omega = \left\{ C = (S, I_c, I_i, T) \in \mathbb{R}_+^4 : C \geq 0, N \leq \frac{\pi}{\mu} \right\}. \quad (4)$$

Thus it suffices to consider solutions in the region Ω. Hence, the solution of our initial value problem starting in Ω exists and is unique on maximal interval [0, b) for some b > 0. Since solutions remain bounded in the positively invariant region Ω, the maximal interval is [0, ∞) (Mubayi *et al*, 2009). Thus, the initial value problem is well posed both mathematically and epidemiologically.

3.2 The Steady States

In this section the model system (1) is qualitatively analyzed by determining the model equilibria, carrying out their corresponding stability analysis and interpreting the results. Letting $E = (S^*; I_c^*; I_i^*; T^*)$ to be the equilibrium point of the system (1). Then, setting the right hand side of system (1) to zero, we obtain;

$$\begin{aligned} &\pi + \gamma_i I_i^* + \gamma_c I_c^* + \omega\gamma T^* - q\lambda^* S^* - (1-q)\lambda^* S^* - \mu S^* = 0, \\ &(1-q)\lambda^* S^* - (\mu + \gamma_c + \alpha) I_c^* = 0, \\ &q\lambda S^* + \alpha I_c^* - (\mu + \gamma_i + \sigma + \delta) I_i^* = 0, \\ &\sigma I_i^* - (\mu + \omega\gamma + \tau\delta) T^* = 0. \end{aligned} \quad (5)$$

From the second equation of (5), we have

$$I_c^* = \eta_1 \lambda^* S^*, \quad (6)$$

where

$$\eta_1 = \frac{1-q}{\mu + \gamma_c + \alpha}.$$

Substituting (6) in the third equation of (5) and solving for I_i^* leads to

$$I_i^* = \eta_2 \lambda^* S^*, \quad (7)$$

with,

$$\eta_2 = \frac{q(\mu + \gamma_c + \alpha) + \alpha(1-q)}{(\mu + \gamma_c + \alpha)(\mu + \gamma_i + \sigma + \delta)}.$$

From the fourth equation of (5), we have

$$T^* = \eta_3 \lambda^* S^*, \quad (8)$$

where,

$$\eta_3 = \frac{\sigma\eta_2}{\mu + \omega\gamma + \tau\delta}.$$

From equations (2), (6) and (7) we have,

$$\lambda^* = 0, \tag{9}$$

or

$$\eta S^* = N^*, \tag{10}$$

with

$$\eta = (\eta_1 + \eta_2 \nu) c\beta.$$

But,

$$N^* = S^* + I_c^* + I_i^* + T^*. \tag{11}$$

Substituting the equations (6), (7) and (8) into (11), leads to

$$N^* = \left(1 + \eta_1\lambda^* + \eta_2\lambda^* + \eta_3\lambda^*\right) S^*. \tag{12}$$

Substituting for N^*, in (10) we obtain

$$\lambda^* = \frac{\eta - 1}{\eta_1 + \eta_2 + \eta_3}. \tag{13}$$

Substituting for $\eta, \eta_1, \eta_2 \text{ and } \eta_3$ in (13) and after some lengthy algebraic manipulations, gives the value of λ^* as;

$$\lambda^* = \left(\frac{bed}{qb(d+\sigma) + (1-q)(ed + d + \sigma\alpha)}\right)(R_{ST} - 1) = Q_0 (R_{ST} - 1), \tag{14}$$

where,

$$b = \mu + \gamma_c + \alpha, \qquad d = \mu + \gamma_i + \sigma + \delta, \qquad e = \mu + \omega\gamma + \tau\delta \quad ,$$

$Q_0 = \left(\dfrac{bed}{qb(d+\sigma)+(1-q)(ed+d+\sigma\alpha)} \right)$ and R_{ST} is the model reproduction number.

3.3 The disease-free Equilibrium, E_0

Solution (9) leads to the DFE denoted by $E_0 = \left(S^0; I_c{}^0; I_i{}^0; T^0\right)$ given by;

$$E_0 = \left(\frac{\pi}{\mu}, 0, 0, 0 \right). \tag{15}$$

3.4 Model Reproduction Number, R_{ST}

In this section, the threshold parameter that governs the spread of a disease which is called the model reproduction number is determined. Mathematically, it is the spectral radius of the next generation matrix (van den Driessche and Watmough, 2002).

To decompose system (1), the equations are re-written starting with infective classes, to obtain,

$$\begin{aligned}
\frac{dI_i}{dt} &= q\lambda S + \alpha I_c - (\mu + \gamma_i + \sigma + \delta) I_i \\
\frac{dI_c}{dt} &= (1-q)\lambda S - (\mu + \gamma_c + \alpha) I_c \\
\frac{dT}{dt} &= \sigma I_i - (\mu + \omega\gamma + \tau\delta) T, \\
\frac{dS}{dt} &= \pi + \gamma_i I_i + \gamma_c I_c + \omega\gamma T - q\lambda S - (1-q)\lambda S - \mu S
\end{aligned} \tag{16}$$

From system (16), F_i and V_i are defined as

$$F_i = \begin{pmatrix} c\beta q \left(\dfrac{I_i + \nu I_c}{N} \right) S \\ c\beta a \left(\dfrac{I_i + \nu I_c}{N} \right) S \end{pmatrix}, \tag{17}$$

where $a = 1 - q$ and

$$V_i = \begin{pmatrix} -\alpha I_c + (\mu + \gamma_i + \sigma + \delta) I_i \\ (\mu + \gamma_c + \alpha) I_c \end{pmatrix}. \tag{18}$$

The partial derivatives of F_i and V_i (17) and (18) with respect to I_i and I_c are obtained. This gives

$$DF_i = \begin{pmatrix} c\beta q \frac{S}{N} & c\beta \nu q \frac{S}{N} \\ c\beta a \frac{S}{N} & c\beta \nu a \frac{S}{N} \end{pmatrix} \tag{19}$$

and

$$DV_i = \begin{pmatrix} \mu + \gamma_i + \sigma + \delta & -\alpha \\ 0 & \mu + \gamma_c + \alpha \end{pmatrix}. \tag{20}$$

At disease-free, i.e $E_0 = \left(\frac{\pi}{\mu}, 0, 0, 0\right)$ and $N \to \frac{\pi}{\mu}$, the matrices (19) and (2.20) become

$$F = \begin{pmatrix} c\beta q & c\beta \nu q \\ c\beta a & c\beta \nu a \end{pmatrix}$$

and

$$V = \begin{pmatrix} \mu + \gamma_i + \sigma + \delta & -\alpha \\ 0 & \mu + \gamma_c + \alpha \end{pmatrix}.$$

The matrices are of size 2×2 since there are two infectious classes. Taking the inverse of V leads to

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu + \gamma_i + \sigma + \delta} & \frac{\alpha}{(\mu + \gamma_c + \alpha)(\mu + \gamma_i + \sigma + \delta)} \\ 0 & \frac{1}{\mu + \gamma_c + \alpha} \end{pmatrix}.$$

We compute FV^{-1}, to get

$$FV^{-1}=\begin{pmatrix} \dfrac{c\beta q}{\mu+\gamma_i+\sigma+\delta} & \dfrac{c\beta\nu q}{\mu+\gamma_c+\alpha}+\dfrac{c\beta q\alpha}{(\mu+\gamma_c+\alpha)(\mu+\gamma_i+\sigma+\delta)} \\ \dfrac{c\beta a}{\mu+\gamma_i+\sigma+\delta} & \dfrac{c\beta\nu a}{\mu+\gamma_c+\alpha}+\dfrac{c\beta a\alpha}{(\mu+\gamma_c+\alpha)(\mu+\gamma_i+\sigma+\delta)} \end{pmatrix}.$$

The eigenvalues for the matrix FV^{-1} are

$\lambda_1=0$ and $\lambda_2=\dfrac{c\beta q}{\mu+\gamma_i+\sigma+\delta}+\dfrac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha}+\dfrac{c\beta\alpha(1-q)}{(\mu+\gamma_c+\alpha)(\mu+\gamma_i+\sigma+\delta)}$. Thus the spectral radius (dominant eigenvalue) of the matrix is λ_2. Hence the model reproduction number,

$$R_{ST}=\frac{c\beta q}{\mu+\gamma_i+\sigma+\delta}+\frac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha}+\frac{c\beta\alpha(1-q)}{(\mu+\gamma_c+\alpha)(\mu+\gamma_i+\sigma+\delta)}. \qquad (21)$$

$$R_{ST}=R_1+R_2+R_{12},$$

where,

(i) $R_1=\dfrac{c\beta q}{\mu+\gamma_i+\sigma+\delta}$, this represents the number of secondary infection individuals that is produced by symptomatic infective individuals who are at a time being treated.

(ii) $R_2=\dfrac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha}$, this represents the number of secondary infection individuals that is produced by asymptomatic infective individuals who have been screened.

(iii) $R_{12}=\dfrac{c\beta\alpha(1-q)}{(\mu+\gamma_c+\alpha)(\mu+\gamma_i+\sigma+\delta)}$, this represents the average number of secondary infection individuals that is produced by infective individuals who have been screened and are at a time being treated.

3.5 Analysis of R_{ST}

This section intends to evaluate the success of intervention programme comprising screening and treatment. The study aims to determine the necessary and sufficient conditions that may slow down the development of the disease through the reduction of the reproduction number below the threshold of one.

The following scenarios are studied under this section: (i) no intervention is used (in the absence of intervention), (ii) screening alone, (iii) treatment alone and (iv) a combination of both screening and treatment.

Below we discuss the reproduction numbers for the programmes mentioned above.

(i) **In the absence of interventions** (where there is no screening or treatment) that is;

($\sigma = 0, \alpha = 0$), R_{ST} is reduced to:

$$R_0 = \frac{c\beta q}{\mu + \gamma_i + \delta} + \frac{c\beta\nu(1-q)}{\mu + \gamma_c}. \tag{22}$$

The threshold quantity R_0 is the basic reproductive number of the model, commonly referred to as the average number of secondary infections generated by a single infective individual in a completely susceptible population (May and Anderson, (1988); Diekmann (2000)). The case $R_0 < 1$ the disease clears from the population and persists if $R_0 > 1$, prompting intervention of any kind.

(ii) **Screening only that is;** $(\sigma = 0)$ R_{ST} is reduced to:

$$R_S = \frac{c\beta q}{\mu + \gamma_i + \delta} + \frac{c\beta\nu(1-q)}{\mu + \gamma_c + \alpha} + \frac{c\beta\alpha(1-q)}{(\mu + \gamma_c + \alpha)(\mu + \gamma_i + \delta)} \tag{23}$$

Clearly, $R_S < R_0$ and if $R_S < 1 < R_0$ the disease clears, but if $1 < R_S < R_0$ the disease persists, thus, there is a need of introducing another intervention.

(iii) **With treatment only that is;** ($\alpha = 0$), the R_{ST} is reduced to:

$$R_T = \frac{c\beta q}{\mu + \gamma_i + \sigma + \delta} + \frac{c\beta\nu(1-q)}{\mu + \gamma_c}. \tag{24}$$

We now investigate to what extent and under what conditions treatment alone can slow down or eradicate the disease. From equations (22) and (24) observing the first terms of each equation it can be seen that,

$$\frac{c\beta q}{\mu + \gamma_i + \sigma + \delta} < \frac{c\beta q}{\mu + \gamma_i + \delta}.$$

From this observation, it is noted that, $R_T < R_0$, thus, treatment seems to reduce the R_0 ,

and it can clearly eradicate the disease if $R_T < 1 < R_0$, but if $1 < R_T < R_0$ the disease persists.

Special cases for treatment, that is, $\sigma = 0$ and $\sigma \to \infty$ are also considered.

In the absence of treatment (i.e when $\sigma = 0$), $R_T = R_0$ and as $\sigma \to \infty$ we have,

$$R_T = \lim_{\sigma \to \infty} \left(\frac{\frac{c}{\sigma}\frac{\beta}{\sigma}\frac{q}{\sigma}}{1 + \frac{\mu}{\sigma} + \frac{\gamma_i}{\sigma} + \frac{\delta}{\sigma}} + \frac{c\beta\nu(1-q)}{\mu + \gamma_c} \right)$$
$$= \left(\frac{0.0.0}{1+0+0+0} + \frac{c\beta\nu(1-q)}{\mu + \gamma_c} \right)$$
$$= \frac{c\beta\nu(1-q)}{\mu + \gamma_c}.$$

Clearly, in the absence of treatment ($\sigma = 0$) and if $R_0 > 1$, the epidemic will develop, but if $R_0 < 1$ it will die out. As $\sigma \to \infty$ (all infected individuals having access to treatment), $R_T \to \Re_0 < R_0$, thus the epidemic will be fully controlled since this leads to no further infections. Thus treatment will succeed in clearing pneumonia.

Note that, $\Re_0 = \frac{c\beta\nu(1-q)}{\mu + \gamma_c}$.

(iv) **With screening and treatment**: This part is examined in two different scenarios; i.e, the reproduction number of the model with both interventions is compared to the reduced reproduction numbers when each of the interventions is separately used. It easy to show that $R_{ST} < R_0$, which implies that intervention strategies have a positive impact in reducing the spread of pneumonia, particularly if $R_{ST} < 1 < R_0$. The case $R_{ST} > 1$ has implications that treatment and screening are ineffective require other interventions that can reduce the disease burden.

- **Comparing (21) and (23), we have**

$$\begin{aligned} R_S &= \frac{c\beta q}{\mu+\gamma_i+\delta} + \frac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha} + \frac{c\beta\alpha(1-q)}{(\mu+\gamma_c+\alpha)(\mu+\gamma_i+\delta)} \\ &= \frac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha} + \frac{c\beta}{\mu+\gamma_i+\delta}\left(q + \frac{\alpha(1-q)}{\mu+\gamma_c+\alpha}\right) \\ &= \frac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha} + \frac{c\beta}{\mu+\gamma_i+\delta}\varphi \end{aligned} \tag{25}$$

$$\begin{aligned} R_{ST} &= \frac{c\beta q}{\mu+\gamma_i+\sigma+\delta} + \frac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha} + \frac{c\beta\alpha(1-q)}{(\mu+\gamma_c+\alpha)(\mu+\gamma_i+\sigma+\delta)} \\ &= \frac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha} + \frac{c\beta}{\mu+\gamma_i+\sigma+\delta}\left(q + \frac{\alpha(1-q)}{\mu+\gamma_c+\alpha}\right) \\ &= \frac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha} + \frac{c\beta}{\mu+\gamma_i+\sigma+\delta}\varphi \end{aligned} \tag{26}$$

with,

$$\varphi = \left(q + \frac{\alpha(1-q)}{\mu+\gamma_c+\alpha}\right).$$

Comparing the last terms of both (25) and (26), it is observed that

$$\frac{c\beta}{\mu+\gamma_i+\sigma+\delta} < \frac{c\beta}{\mu+\gamma_i+\delta},$$

the result which concludes that, $R_{ST} \le R_S$, thus a combination of both screening and treatment can clear the disease if $R_{ST} < R_S < R_0$, and it is even more efficient than when only screening intervention is in place. However, if $1 < R_{ST} < R_S < R_0$, then the disease persists.

- **Comparing (21) and (24), we have**

$$\begin{aligned} R_{ST} &= \frac{c\beta q}{\mu+\gamma_i+\sigma+\delta} + \frac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha} + g \\ &= \frac{c\beta q}{\mu+\gamma_i+\sigma+\delta} + \frac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha} - \frac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha} + g, \\ &= R_T - D \end{aligned}$$

where,

$$g = \frac{c\beta\alpha(1-q)}{(\mu+\gamma_c+\alpha)(\mu+\gamma_i+\sigma+\delta)}$$

and

$$D = \left(\frac{c\beta\nu(1-q)}{\mu+\gamma_c} - \frac{c\beta\nu(1-q)}{\mu+\gamma_c+\alpha} - \frac{c\beta\alpha(1-q)}{(\mu+\gamma_c+\alpha)(\mu+\gamma_i+\sigma+\delta)} \right)$$

$$D = c\beta(1-q)\left(\frac{\nu}{\mu+\gamma_c} - \frac{\nu}{\mu+\gamma_c+\alpha} - \frac{\alpha}{(\mu+\gamma_c+\alpha)(\mu+\gamma_i+\sigma+\delta)} \right)$$

$$= \frac{c\beta\alpha(1-q)}{\mu+\gamma_c+\alpha}\left(\frac{\nu}{\mu+\gamma_c} - \frac{1}{(\mu+\gamma_i+\sigma+\delta)} \right)$$

$$= \frac{c\beta\alpha(1-q)}{\mu+\gamma_c+\alpha}\left(\frac{\nu(\mu+\gamma_i+\sigma+\delta)-(\mu+\gamma_c)}{(\mu+\gamma_c)(\mu+\gamma_i+\sigma+\delta)} \right).$$

Thus,

$$R_{ST} = R_T - \frac{c\beta\alpha(1-q)}{\mu+\gamma_c+\alpha}\left(\frac{\nu(\mu+\gamma_i+\sigma+\delta)-(\mu+\gamma_c)}{(\mu+\gamma_c)(\mu+\gamma_i+\sigma+\delta)} \right). \qquad (27)$$

Result (27), shows that, $R_{ST} \le R_T$, if $\left(\nu(\mu+\gamma_i+\sigma+\delta)-(\mu+\gamma_c)\right) \ge 0$. This also shows that, a combination of both screening and treatment can clear the disease if $R_{ST} < R_T < R_0$, and it is once again more efficient than when only treatment intervention is in place. If $1 < R_{ST} < R_T < R_0$, then the disease persists.

Hence the combination of both screening and treatment can eradicate the pneumonia infection if R_{ST} can be reduced below unity.

Note that, $\frac{1}{(\mu+\gamma_i+\sigma+\delta)}$, is the mean infectious period.

The model reproduction number, R_{ST} obtained from the method of next generations by Van den Driessche and Watmough, 2002, determine the local stability of the disease free equilibrium point which is locally asymptotically stable for $R_{ST} < 1$ and unstable for $R_{ST} > 1$ so the following theorem holds:

Theorem 1 *The disease-free equilibrium (E_0) of the model system (1) is locally asymptotically stable whenever* $R_{ST} < 1$ *and unstable if* $R_{ST} > 1$.

3.6 The Endemic Equilibrium E_1

The endemic equilibrium point $E_1 = \left(S^*; I_c^*; I_i^*; T^*\right)$ is a steady state solution in which the disease persists in the population (i.e $I_c \neq 0 \; and \; I_i \neq 0$). In this section, the possible existence of the positive endemic equilibria of the model (1) is determined by solving the equations of the system (5) at steady state.

Solving system (5) in section 2.2 at steady state, gives the following solutions,

$$S^* = \frac{\pi}{\mu + \left(1 - \gamma_i \eta_2 - \gamma_c \eta_1 - \omega\gamma\eta_3\right) Q_0 \left(R_{ST} - 1\right)}, \qquad (28)$$

where, $\eta_1, \eta_2, \eta_3 \; and \; Q_0$ are as defined in (6), (7), (8) and (14) in section 2.2 above. It can easily be shown that, $\left(1 - \gamma_i \eta_2 - \gamma_c \eta_1 - \omega\gamma\eta_3\right) > 0$.

The endemic equilibrium is given by $E_1 = \left(S^*, I_c^*, I_i^* and \; T^*\right)$, where $S^*; I_c^*; I_i^* and \; T^*$ are the coordinates given by results (6), (7), (8), and (28) and they are always positive if and only if $R_{ST} > 1$. Therefore we state lemma 1 as;

Lemma 1 *A unique endemic equilibrium point E_1 exists and is positive if and only if* $R_{ST} > 1$.

Note that:- If $R_{ST} = 1$, the endemic equilibrium reduces to the DFE.

4 Sensitivity Analysis

In determining how best to reduce human mortality and morbidity due to pneumonia, we calculate the sensitivity indices of the model reproduction number, R_{ST} to the parameters in the model using the approach of Chitnis et al., (2008) and the approach by Blower and Dowlatabadi., (1994). These indices are crucial in determining the importance of each individual parameter in the transmission dynamics and prevalence of the disease. Sensitivity analysis determines parameters that have a high impact on R_{ST} and should be targeted by intervention strategies. Sensitivity indices allow us to measure the relative change in a state variable when a parameter changes (Chitnis et al., 2008). The normalized forward sensitivity index of a variable to a parameter is a ratio of the relative change in the variable to the relative change in the parameter. If a variable is a differentiable function

of the parameter, the sensitivity index may be alternatively defined using partial derivatives.

Definition 1 *The normalized forward sensitivity index of a variable, u, which depends differentiably on index of a parameter, p is defined as;* $\Upsilon_p^u = \frac{\partial u}{\partial p} \times \frac{p}{u}.$

From an explicit formula for R_{ST} in (21) we derive an analytical expression for the sensitivity of R_{ST} as $\Upsilon_p^{R_{ST}} = \frac{\partial R_{ST}}{\partial p} \times \frac{p}{R_{ST}}$ to each of the parameter involved in R_{ST}. For example the sensitivity index of R_{ST} with respect to β is $\Upsilon_\beta^{R_{ST}} = \frac{\partial R_{ST}}{\partial \beta} \times \frac{\beta}{R_{ST}} = 1,$ other indices $\Upsilon_\delta^{R_{ST}}, \Upsilon_{\gamma_i}^{R_{ST}}, \Upsilon_\mu^{R_{ST}}, \Upsilon_\alpha^{R_{ST}}, \Upsilon_{\gamma_c}^{R_{ST}}, \Upsilon_\nu^{R_{ST}}, \Upsilon_q^{R_{ST}}, \Upsilon_\sigma^{R_{ST}}$ *and* $\Upsilon_c^{R_{ST}},$ were obtained and evaluated at $\beta = 6, \mu = 0.02, \quad q = 0.13, c = 2, \gamma_i = 0.2, \gamma_c = 0.06, \nu = 0.051, \sigma = \ , \delta = 0.26,$ *and* $\alpha = 0.4$ to obtain the following results

Table 1: Numerical values of sensitivity indices of R_{ST}

Parameter symbol	Sensitivity index
c	1.5322
β	1
σ	-0.635941
ν	0.211433
γ_c	-0.110013
δ	-0.0826724
α	-0.06647494
γ_i	-0.0635941
μ	-0.0430303
q	-0.0116102

4.1 Interpretation of Sensitivity Indices

Table 1 shows the sensitivity indices of R_{ST} to the parameters for the pneumonia model with screening and treatment interventions, evaluated at the baseline parameter values indicated above. The parameters are ordered from most sensitive to least. The most sensitive parameter is the contact rate, c and the least sensitive parameter is the progression proportion of the disease, q. This result implies that, when the parameters c, β and ν are

increased keeping other parameters constant they increase the value of R_{ST} thus, they increase the endemicity of the disease as they have positive indices. While the parameters $\mu, q, \gamma_i, \gamma_c, \sigma, \delta$ and α decrease the value of R_{ST} when they are increased while keeping the other parameters constant, implying that they decrease the endemicity of the disease as they have negative indices.

5 Numerical Simulations and Discussion

5.1 Numerical Simulation

All figures are plotted using the parameter values presented in Table 2.

Table 2: Parameter Values for pneumonia Model with interventions

Symbol	Value	Source
π	1000	Ndelwa (2012)
μ	0.01	Ndelwa (2012)
ω	$\omega \geq 1$	Brauer (2004)
γ_i	0.15-0.2	Estimate
γ_c	0.02-0.1	Estimate
γ	0.1	Ndelwa (2012)
β	0-10	Underdown (2011)
δ	0.0575-0.4605	Blower (2002)
α	0.001-0.1	Estimate
c	2	Estimate
σ	0.083-3	Underdown (2011)
q	0.05-0.2	Estimate
τ	$0 \leq \tau \leq 1$	Brauer (2011)
v	0.001-0.1	Estimate

The figures below show the outcomes from the analysis.

Figures (2) (a) and (b) display the effect of the screening rate. It is noted that α increases the symptomatic infected population and decreases the asymptomatic infected population. This shows that, due to screening we are able to recognize the carriers and hence provide them with the appropriate measures for clearance of the epidemic.

We use Figure (3) to investigate the effect of treatment on the dynamical behaviour of pneumonia infection we simulate the model over different values of the treatment rate σ (0.8, 1.2, 2). From the figure it is clearly observed that, these values give the corresponding values of the reproduction number R_{ST} (5.0571, 4.7830, 4.6690).

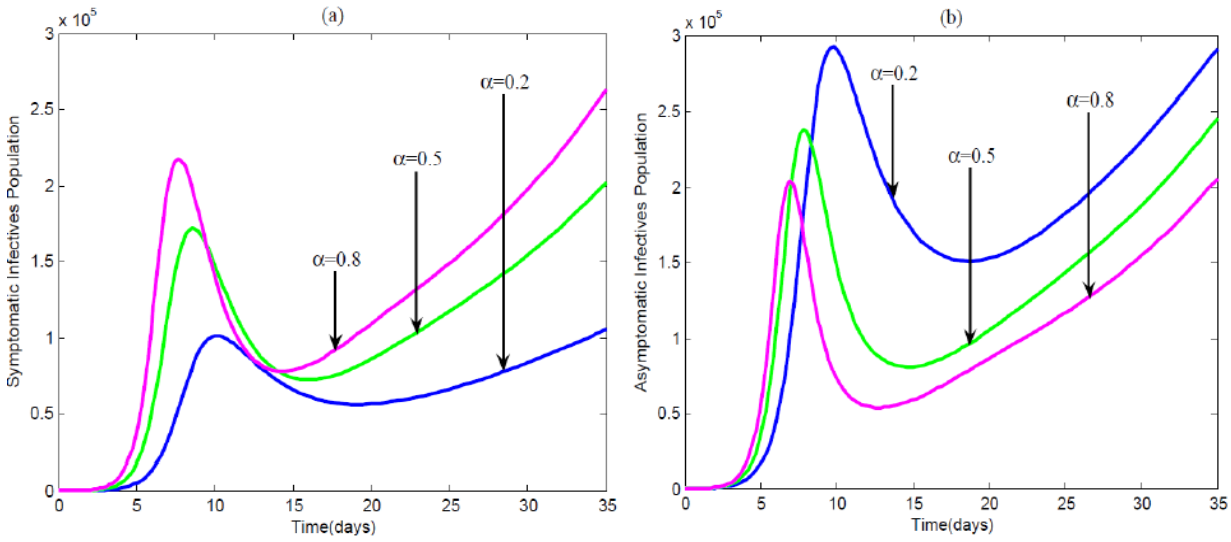

Figure 2: Variation of population under different values of screening rate α

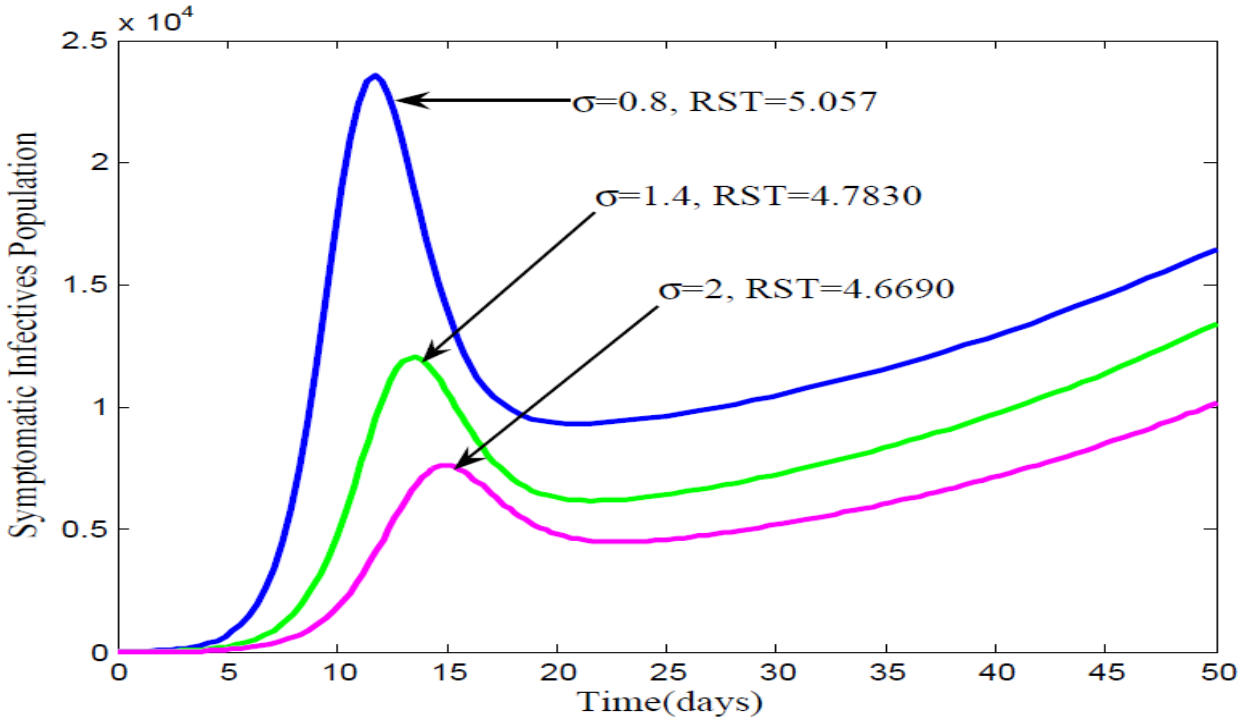

Figure 3: Variation of population under different values of treatment rate σ

Thus, the result in figure (3), show that, increasing treatment rate has the effect of reducing the number of secondary cases and subsequently reduce the pneumonia epidemic. Further increase in the treatment rate reduces the reproduction number below unity, this leads to the clearance of infection in the population. The result further show that, increasing the treatment rate decreases the severity of the epidemic as seen by gradual decrease in the peaks and time lags between peaks as σ increases.

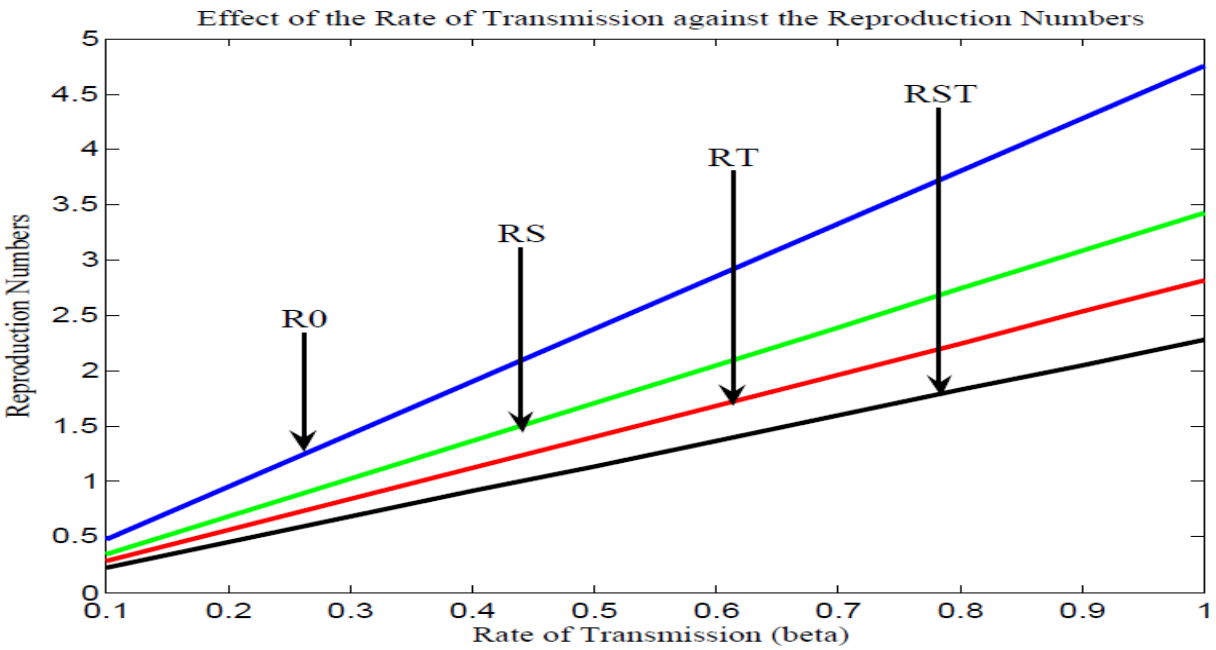

Figure 4: The relationship between reproduction numbers as β changes

Figure (4), shows the relationship between the reproduction numbers as β changes. It is observed that when $R_0 > 1$, β increases which implies that, when the number of secondary cases increases, β also increases. This result implies that, β has a direct impact on the dynamics of the disease by increasing the infection when there is no intervention. It is also observed that β decreases when $R_{ST} < 1$. Therefore we can conclude that, when interventions are introduced in the community, the campaigns should be conducted at high rate in order to reduce the spread of disease or to eradicate the disease totally.

Figure (4), also indicates that, $R_T < R_S < R_0$, that is, treatment alone is more effective than screening alone. However, it is also observed that, $R_{ST} < R_T < R_S < R_0$, this result depicts the result in section 3.5 (analysis of R_{ST}), which also showed that, the combination of treatment and screening is more effective than when only one intervention programme is at place.

5.2 Discussion

The model of Pneumonia with screening and treatment interventions was formulated and analysed. Using differential equations, the invariant set in which the solutions of the model are biologically meaningful was derived. Boundedness of solutions was also proved. Analysis of the model showed that, there exist two possible solutions, namely the disease-free point and the endemic equilibrium point. Further analysis showed that, the disease-free point is locally stable implying that, perturbations and fluctuations on the disease state will always

result in the clearance of the epidemic if $R_{ST} < 1$. Sensitivity analysis of the effects of screening and treatment was carried out to evaluate or assess the implications of the strategy on the behavior or in predicting the outcome of the epidemic. From the Figure 4 we understand that screening and treatment can eradicate the disease, as the figure clearly shows how the force of infection decreases when both screening and treatment are in place. Figures 2 and 3 also put it clear on how screening and treatment have an impact on the eradication of the disease. From both section 3.5 and 5, we conclude that, the combination of both screening and treatment provides better results than when only one of the intervention is on place. Therefore screening and treating at the same time can eradicate the pneumonia epidemic from the community.

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