Sensitivity and Numerical analysis on the spread and control of Pneumonia in the case of DebreBerhan town, Ethiopia

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Abstract: In this work, we have considered a nonlinear dynamical system. We divide the total population of Debre Berhan town into four compartments: Susceptible classS, Exposed class E, Infected classI and Recovered class R. We found two equilibrium points, the disease free equilibrium point and the endemic equilibrium point. We also found the basic reproduction number of the dynamical system $isR_0 = \frac{\beta(\alpha+p\mu)}{(\mu+\alpha)(\mu+d+\gamma)}$ which depends on six parameters. The numerical value of the reproduction number based on real data collected from Debre Berhan town isR_0 = 1.051448663 > 1. This shows that Pneumonia disease spread in the community of Debre Berhan town. We also proved that the disease free equilibrium point is unstable and the endemic equilibrium

point is stable. We also found the numerical simulation based on the real data collected from Debre Berhan town which supports the analytical findings. We used sensitive analysis to identify the most influential parameter to control the disease. The most influential control parameter that can help us to control the spread of the Pneumonia disease is the transmission coefficient β .

Key words:-Pneumonia, Sensitivity, reproduction number, stability

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I. Introduction

Infectious diseases can have a devastating impact on human life and welfare. One of the major contributors to morbidity and mortality is the bacterium Streptococcus Pneumonia (pneumococci). It is the main cause of respiratory tract infections such as otitis media and sinusitis, but it is also responsible for millions of deaths each year due to Pneumonia ^[8]. Infectious disease is caused by various microbes or pathogen. Most of them are usually Microorganisms. Few of them are visible by naked eyes. The most common pathogens are different types of viruses, bacteria, Fungi and Protozoa are known as pathogens and are responsible for various diseases. Diseases caused by these pathogens are termed as 'infectious' as these pathogens can be easily transmitted from one infected person to another non-infected person. Throughout history, infectious diseases have had a large impact on the human population. Although infectious diseases are present in human populations at all times to some degree, the effects of epidemics are the most noticeable and spectacular. There are two ways of infectious disease transmission namely direct or indirect transmissions ^[15].

Direct transmission is the transfer of an infectious agent from the infected individual directly to the host by touching or biting or sexual intercourse or indirect transmission is the transfer of an infectious agent by contaminated inanimate objects by aerosolized agents suspended in air for a long period of time or environment contamination or water or food contamination^[9].

Pneumonia is a severe form of acute lower respiratory tract infection that affects the lung ^[13, 16]. It is also a type of lung infection, caused by a virus or bacteria. The lungs are filled with thousands of tubes, called bronchi, which end in smaller sacs called alveoli. Each one has a fine mesh of capillaries. This is where oxygen is added to the blood and carbon dioxide removed. When a person has Pneumonia, the alveoli in one or both lungs fill with pus and fluids, which hinders the gas exchange. This is sometimes known as 'consolidation and collapse of the lung'^[16].

Pneumonia is usually caused due to an infection with a bacteria, virus, fungi or parasite. In adults it is mostly caused by bacteria whereas in children and infants it is commonly due to viruses. Physical or chemical injury to the lungs can also result in the condition. Individuals who smoke, who are hospitalized and have long-term illness such as asthma, heart disease, cancer, HIV/AIDS, lung diseases or diabetes are at a higher risk of developing Pneumonia. Hospital-acquired Pneumonia is also common^[3].

Pneumonia can be transmitted when airborne microbes from an infected individual are inhaled by an individual ^[12]. However, most instances of Pneumonia are attributable to self-infection with one or more types of microbes that originate in the nose and mouth. In healthy people, typical upper airway bacterial residents such

as Streptococcus pneumonia commonly referred to as "pneumococcal" and Homophiles' influenza are the most common bacteria causing community acquired pneumonia. Hospital-acquired pneumonia is usually caused by more resistant bacteria, such as Staphylococcus aureus, Klebsiella pneumonia,Pseudomonas aeruginosa, and escherichia coli. Individuals with a serious impairment of their immune system become susceptible to pneumonia caused by the so-called "opportunistic" microbes, such as certain fungi, viruses, and bacteria related to tuberculosis (mycobacterium), that would not ordinarily cause disease in normal individuals ^[2].

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 ^[17]. Socio-demographic factors such as the age, sex, parental income, and level of parental education had earlier been identified as risk factors of Pneumonia-related morbidity and mortality. Also, domestic crowding, maternal age, exposure to indoor air pollutants especially firewood burning, and parental smoking had each been recognized as important domestic/household risk factors. Other factors identified include attendance at day care facilities, breastfeeding practices, malnutrition, co-morbidities like diarrhea, HIV/AIDS, micronutrient deficiency (especially vitamin A and zinc), and inter-current infections such as measles and pertussis ^[11].

Epidemiology is a study of infectious diseases, the causes of their occurrence and their spread in space and time. Mathematical epidemiology is about obtaining and understanding of biological phenomena, translating assumptions regarding biological features to mathematical language, finding solutions mathematically then, translating the results back to biology.

The main use of mathematics in epidemiology is to gain insight on epidemics, too see how the dynamics of an infectious disease depends on the basic parameters that characterize it. Reality, however, is complex and even the most involves of the models are only sketches of it. To be able to describe the situation mathematically and to extrapolate on the basis simplifications but the model we must understand how the dynamics depends on the basic components of the model and how sensitive these parameters ^[4]. An epidemic model is a simplified means of describing the transmission of infectious diseases through individuals. The modeling of infectious of infectious diseases is a tool which has been used to study the mechanisms by which disease spread, to predict the future course of an outbreak and to evaluate strategies to control epidemics ^[11].

Epidemic modeling has three main aims. The first is to understand the spreading mechanism of the disease. For this, the essential part is a mathematical structure equations give us threshold values and other constants which we use to describe the behavior of the disease. The second aim is to predict the future course of the epidemic and the third is to understand how we may control the spread of the epidemic education, immunization, isolation ^[9]. In order to make a reliable model and predictions, to develop methods of control, we must be sure that our model describes the epidemic closely; it contains all its specific features. So it is important to validate models by checking whether they fit the observed data or not ^[9]. Mathematical models and computer simulations have become useful in analyzing the spread and control of infectious disease. They together build and test theories that involve with complex biological systems related disease, getting qualitative conjectures and determine parameter sensitive due to change and estimating parameters from data ^[4].

We used Deterministic model, also known as compartmental models it can attempt to describe and explain what happens on the average at the population scale. They fit well large populations. These models categorize individuals into different subgroups or compartments. This study was covers the dynamics of spreading and controlling of Pneumonia in all Kebeles of Debre Berhan Town, North Showa Amhara Region, Ethiopia, based on the application of mathematical models integrated with the concept of biology and epidemiology. In this thesis we construct a mathematical model to understand the spreading and controlling of Pneumonia and we model the disease through ordinary differential equations (ODEs). This model is used to determine which factors are most responsible for the spread of Pneumonia. The model divides the human population into four classes: susceptible class, exposed class, infected class and recovered class.

II. The Mathematical Model

In this study we consider the deterministic SEIR (Susceptible, Exposed, Infected and Recovered) model where the population partitioned in to components or classes based on the epidemiological state of individuals and it assume that the population size in a compartment is differentiable with respect to time and the epidemic process is deterministic. Therefore, the Pneumonia transmission dynamics between the compartments will be described by a system of ordinary differential equation. The work done by ^[10] is the initial model, where the population is divided into compartments containing susceptible, exposed, infectious and recovered individuals. Compartments with labels S, E, I, Rare used for epidemiological classes. The class S is the class of susceptible individuals; that is, those who can become infected. When there is an adequate contact of a susceptible with an infective so that transmission occurs, the susceptible enters the exposed class E of those in the latent period, who are infected but not yet infectious. At the end of the latent period, the individual enters the

class I of infective, who are capable of transmitting the infection (that is, infectious). At the end of the infectious period, the individual enters the recovered class R.

We extend the work ^[10] by considering the following assumptions. All new-born are susceptible i.e., no vertical transmission to the infection and are recruited at rate μN . Natural death rate and birth rate are equal that is μ . The death occurred due to natural case in each class. Total number of population is the sum of all compartments. The exposed become infected at rate (α) and the infectious individuals from the infection at rate(γ). The transmission coefficient β , the latency coefficient α , the recovery coefficient γ and the capital death rate μ are positive quantities i.e. all parameters are non-negative. Age structure is not considered. The infected compartment I(t) decrease by the disease induced death rate d. The impact of immunodeficiency as a factor of fast progression to pneumonia is taken to account. That is the proportion of fast progression rate (p) of the new infection directly move in to the infectious class as a result of co-infection of Pneumonia with other related disease like heart disease, diarrhea, HIV/AIDS and some substance abuse alcohol and tobacco, and the remaining proportion (1 - p) of new infection move to the latently infected but not infectious class due to slow progression rate to Pneumonia in the case when individuals highly immunized. Here, 0 .



Figure1:- The flow chart of the model

The corresponding dynamical system is

$$\frac{dS}{dt} = \mu N - \mu S - \frac{\beta SI}{N},$$
(1)
$$\frac{dE}{dt} = (1 - p) \frac{\beta SI}{N} - (\mu + \alpha) E$$
(2)
$$\frac{dI}{dt} = \alpha E + \frac{p\beta SI}{N} - (\mu + d + \gamma) I,$$
(3)
$$\frac{dR}{dt} = \gamma I - \mu R$$
(4)

Theorem-1:(positivity of the solution)

Suppose $S(0) \ge 0, E(0) \ge 0, I(0) \ge 0$ and $R(0) \ge 0$, then the solution region $\{S(t), E, v(t), I(t), R(t)\}$ of the system of equations (1) to (4) is always non negative for t > 0

Proof:-Let us assume that all parameters are positive.By considering the four ordinary differential equations and after taking some steps on finding their solution we do have:

i.
$$\frac{dS}{dt} = \mu N - \mu S - \frac{\beta SI}{N} \text{ whose solution is}$$

$$S(t) = S(0)e^{-(\mu t + \beta K(t)) + K(0)} + \mu e^{-(\mu t + \beta K(t)) + K(0)} \int_0^t (N(s)e^{(\mu s + \beta K(s)) - K(0)}) ds > 0, \text{ since } S(0) > 0$$
ii.
$$\frac{dE}{dt} = (1 - p)\frac{\beta SI}{N} - (\mu + \alpha)E \text{ whose solution is}$$

$$E(t) = E(0)e^{-(\mu + \alpha)t} + e^{-(\mu + \alpha)t} \int_0^t (1 - p)\frac{\beta SI}{N} e^{(\mu + \alpha)S} dS > 0 \text{ since } E(0) > 0.$$

iii.
$$\frac{dI}{dt} = \alpha E + \frac{p\beta SI}{N} - (\mu + d + \alpha)I \text{ whose solution is}$$

$$I(t) = \begin{cases} I(0)e^{-((\mu + d + \alpha)t - p\beta\varphi(t) + p\beta\varphi(0))} + \\ e^{-((\mu + d + \alpha)t - p\beta\varphi(t) + p\beta\varphi(0))} \int_{0}^{t} (\alpha E(s)e^{((\mu + d + \alpha)s - p\beta\varphi(s) + p\beta\varphi(0))}) ds \end{cases} > 0$$

$$I(t) = I(0)e^{-((\mu + d + \alpha)t - p\beta\varphi(t) + p\beta\varphi(0))} \text{since}I(0) > 0$$
iv.
$$\frac{dR}{dt} = \gamma I(t) - \mu R(t) \text{ whose solution is } R(t) = R(0)e^{\mu t} + e^{\mu t} \int_{0}^{t} (\gamma I(s)e^{\mu s}) ds > 0$$
Since R(0) > 0

Theorem-2:(Boundedness of the solution)

The solution of the dynamical system (1) - (4) in the feasible region

$$D = \{ (S(t), E(t), I(t), R(t)) \in R_{+}^{4}, 0 < N(t) \le C_{2}, S_{0} > 0, E_{0} \ge 0, I_{0} \ge 0, R_{0} \ge 0 \}$$

It is bounded.

Proof

Let us consider $dI \leq dN$ and summing up all the four equations, we do have N(t) = S(t) + E(t) + I(t) + R(t)so that $\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$. Which implies that $\frac{dN}{dt} = \mu N - \mu S - \frac{\beta SI}{N} + (1 - p)\frac{\beta SI}{N} - (\mu + \alpha)E + \alpha E + \frac{p\beta SI}{N} - (\mu + d + \gamma)I + \gamma I - \mu R$. After some simplification we get $\frac{dN}{dt} = -dI$. That is $\frac{dN}{dt} \leq 0$. To find the upper bound we consider two cases. The first is $\frac{dN}{dt} = 0$ and integrating both sides gives $N(t) - N(0) = C_1$ or $N(t) = N(0) + C_1 = C_2$ (constant). The second case is $\frac{dN}{dt} < 0$ and integrating both sides gives $N(t) - N(0) = C_1$ or $N(0) < C_1$ or $N(t) < N(0) + C_1 = C_2$. From the two cases we do have $N(t) \leq C_2$. There fore $\lim_{t\to\infty} N(t) \leq \lim_{t\to\infty} C_2 = C_2$ which is the upper bound. Hence we have $0 < N(t) \leq C_2$. This shows all solutions (S(t), E(t), I(t), R(t)) of the dynamical system (1)- (4) is bounded in the interval $(0, C_2]$ where C_2 is positive constant.

III. Scaling the nonlinear dynamical system

Let us consider N(t) is the total maximum population size and assuming that N(t) is constant. Therefore, we set the proportion by introducing the new variables $s(t) = \frac{S(t)}{N(t)}, e(t) = \frac{E(t)}{N(t)}, i(t) = \frac{E(t)}{N(t)}, r(t) = \frac{R(t)}{N(t)}$ in the dynamical system (1) – (4) we get a new dynamical system which is topologically equivalent to the original system

$$\frac{ds}{dt} = \mu - \mu s - \beta si$$
(5)
$$\frac{de}{dt} = (1 - p)\beta si - (\mu + \alpha)e$$
(6)
$$\frac{di}{dt} = \alpha e + p\beta si - (\mu + d + \gamma)i$$
(7)
$$\frac{dr}{dt} = \gamma i - \mu r$$
(8)
With initial condition $S(0) > 0, E(0) > 0, I(0) > 0, R(0) > 0$

IV.

V. Existence of Equilibrium points of the dynamical system

4.1 Disease Free Equilibrium point

The disease free equilibrium point is obtained by assuming that i = 0 and by setting the right hand side of the system (5) - (8) equal to zero we obtained $E_0(s^0, e^0, i^0, r^0) = (1, 0, 0, 0)$.

4.2 Endemic equilibrium point

The endemic equilibrium point is obtained by assuming that $i \neq 0$ and by setting the right hand side of the system (5) - (8) equal to zero we obtained $E^*(s^*, e^*, i^*, r^*)$ where

$$s^{*} = \frac{(\mu + \alpha)(\mu + \alpha + \gamma)}{\beta(\alpha + p\mu)}$$

$$e^{*} = \frac{(1 - p)\mu(\mu + d + \gamma)}{\beta(\alpha + p\mu)} \left(\frac{\beta(\alpha + p\mu)}{(\mu + \alpha)(\mu + d + \gamma)} - 1\right)$$

$$i^{*} = \frac{\mu}{\beta} \left(\frac{\beta(\alpha + p\mu)}{(\mu + \alpha)(\mu + d + \gamma)} - 1\right)$$

$$r^{*} = \frac{\gamma}{\beta} \left(\frac{\beta(\alpha + p\mu)}{(\mu + \alpha)(\mu + d + \gamma)} - 1\right)$$

4.3 Basic reproduction number R_0

The basic reproduction number denoted by R_0 is defined as the average number of secondary cases produced by a typical infected individual during his or her entire life as infectious or infectious period when introduced or allowed to live in a population of susceptible which can be calculated using the next-generation method as follows: In the dynamical system (1)-(4) the rate of appearance of new infections \mathcal{F} and the transfer rate of individuals \mathcal{V} at the disease free steady state

$$F = \begin{pmatrix} 0 & (1-p)\beta \\ 0 & p\beta \end{pmatrix}, V = \begin{pmatrix} (\mu+\alpha) & 0 \\ -\alpha & (\mu+d+\gamma) \end{pmatrix} \text{ and } V^{-1} = \begin{pmatrix} \frac{1}{(\mu+\alpha)} & 0 \\ \frac{\alpha}{(\mu+\alpha)(\mu+d+\gamma)} & \frac{1}{(\mu+d+\gamma)} \end{pmatrix}.$$
 So that $FV^{-1} = \begin{pmatrix} \frac{\alpha\beta(1-p)}{(\mu+\alpha)(\mu+d+\gamma)} & \frac{1}{(\mu+d+\gamma)} \end{pmatrix}$.

Hence the spectral radius of the matrix FV^{-1} is given by $max\{\lambda_1, \lambda_2\} = \lambda_2$. Therefore, using the next generation method the spectral radius of the matrix FV^{-1} is $\lambda_2 = \frac{\beta(\alpha + p\mu)}{(\mu + \alpha)(\mu + d + \gamma)}$ since $\lambda_1 = 0$. Therefore we found $R_0 = \frac{\beta(\alpha + p\mu)}{(\mu + \alpha)(\mu + d + \gamma)}$

Stability analysis of the equilibrium points 4.4. Theorem-3:

The disease free equilibrium point E^0 of the system of ordinary differential equation (1) - (4) is stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof

The Jacobian matrix of the dynamical system (1) – (4) at any equilibrium point (*s*, *e*, *i*, *r*) is

$$J(s, e, i, r) = \begin{bmatrix} -\mu - \beta i & 0 & -\beta s & 0\\ (1-p)\beta i & -(\mu+\alpha) & (1-p)\beta s & 0\\ p\beta i & \alpha & p\beta s - (\mu+d+\gamma) & 0\\ 0 & 0 & \gamma & \mu \end{bmatrix}$$

And then the Jacobian matrix of the dynamical system (1) - (4) at Disease free equilibrium point $E_0(s^0, e^0, i^0, r^0) = (1, 0, 0, 0)$ is

$$J(1,0,0,0) = \begin{bmatrix} -\mu & 0 & -\beta & 0\\ 0 & -(\mu+\alpha) & (1-p)\beta & 0\\ 0 & \alpha & p\beta - (\mu+d+\gamma) & 0\\ 0 & 0 & \gamma & -\mu \end{bmatrix}.$$
 The corresponding characteristic equation is
$$\begin{vmatrix} -\mu - \lambda & 0 & -\beta & 0\\ 0 & -(\mu+\alpha) - \lambda & (1-p)\beta & 0\\ 0 & \alpha & (p\beta - (\mu+d+\gamma)) - \lambda & 0\\ 0 & 0 & \gamma & -\mu - \lambda \end{vmatrix} = 0.$$

Or $(-\mu - \lambda)(-\mu - \lambda)[(-x - \lambda)(z - \lambda) - y\alpha] = 0$ whose eigenvalues are $\lambda_1 = -\mu$ and $\lambda_2 = -\mu$ and values of eigen values found by stability criterion of the equation $[(-x - \lambda)(z - \lambda) - y\alpha] = 0$. This can be rewritten $a_{2}a_{2}\lambda^{2} + a_{1}\lambda^{1} + a_{0}\lambda^{0} = 0$, where $a_{2} = 1$, $a_{1} = ((\mu + \alpha) + (\mu + d + \gamma)) - p\beta$ and $a_{0} = (\mu + \alpha)(\mu + d + d + \alpha)$ $\gamma - \beta \alpha + p \mu$.

Hence we found that If $a_1 > 0$ which implies $\frac{(\mu+d+\gamma)+(\mu+\alpha)}{p\beta} > 1$ and $b_1 > 0$ means $\frac{(\mu+d+\gamma)(\mu+\alpha)}{\beta(\alpha+p\mu)} = \frac{1}{R_0} > 1$, then the first column of the Routh array has no sign change this means all Eigenvalues have negative real part, in this case the disease free equilibrium point (1, 0, 0, 0) is stable. If either $a_1 < 0$ implies $\frac{(\mu+d+\gamma)+(\mu+\alpha)}{p\beta} < 1$ or $b_1 < 0$ $\operatorname{means}\frac{(\mu+d+\gamma)(\mu+\alpha)}{\beta(\alpha+p\mu)} = \frac{1}{R_0} < 1$, then the first column of the Routh array have sign change this means all Eigenvalues do not have negative real part, in this case the disease free equilibrium point (1, 0, 0, 0) is unstable.

4.5Stability analysis of the endemic equilibrium point

Theorem-4:

The endemic equilibrium point $E^*(s^*, e^*, i^*, r^*)$ of the system of ordinary differential equation (1) – (4) is stable if $R_0 > 1$.

Proof

The Jacobian matrix of the dynamical system (1) - (4) at the endemic equilibrium point (s^*, e^*, i^*, r^*) is $\Gamma - \mu - \beta i^*$ 0

$$J(s^{*}, e^{*}, i^{*}, r^{*}) = \begin{bmatrix} -\mu - \mu i & 0 & -\mu s & 0 \\ (1 - p)\beta i^{*} & -(\mu + \alpha) & (1 - p)\beta s^{*} & 0 \\ p\beta i^{*} & \alpha & p\beta s^{*} - (\mu + d + \gamma) & 0 \\ 0 & 0 & \gamma & \mu \end{bmatrix}$$
where
$$s^{*} = \frac{(\mu + \alpha)(\mu + d + \gamma)}{\beta(\alpha + p\mu)}, e^{*} = \frac{(1 - p)\mu(\mu + d + \gamma)}{\beta(\alpha + p\mu)} \Big(\frac{\beta(\alpha + p\mu)}{(\mu + \alpha)(\mu + d + \gamma)} - 1 \Big), i^{*} = \frac{\mu}{\beta} \Big(\frac{\beta(\alpha + p\mu)}{(\mu + \alpha)(\mu + d + \gamma)} - 1 \Big),$$

$$r^* = \frac{\gamma}{\beta} \left(\frac{\beta(\alpha + p\mu)}{(\mu + \alpha)(\mu + d + \gamma)} - 1 \right)$$

Note that the endemic equilibrium exists provided that $\frac{\beta(\alpha+p\mu)}{(\mu+\alpha)(\mu+d+\gamma)} - 1 > 0$ that is $R_0 - 1 > 0$.

The corresponding characteristic equation is

$$\begin{vmatrix} -(\mu + \beta i^{*}) - \lambda & 0 & -\beta s^{*} & 0 \\ (1 - p)\beta i^{*} & -(\mu + \alpha) - \lambda & (1 - p)\beta s^{*} & 0 \\ p\beta i^{*} & \alpha & (p\beta s^{*} - (\mu + d + \gamma)) - \lambda & 0 \\ 0 & 0 & \gamma & (-\mu - \lambda) \end{vmatrix} = 0$$

Or

$$\begin{aligned} -(\mu+\beta i^*) - \lambda [-(\mu+\alpha)(\mu+d+\gamma)\mu - (\mu+\alpha)(\mu+d+\gamma)\lambda + p\beta\mu(\mu+\alpha)s^* + p\beta(\mu+\alpha)s^*\lambda \\ &-\mu(\mu+\alpha)\lambda - (\mu+\alpha)\lambda^2 - \mu(\mu+d+\gamma)\lambda - (\mu+d+\gamma)\lambda^2 + p\beta\mu s^*\lambda + p\beta s^*\lambda^2 \\ &-\lambda^3 + \alpha\beta\mu(1-p)s^* + \alpha\beta(1-p)s^*\lambda] - p\beta^2 i^*s^*\lambda^2 \\ &+ [(\alpha\beta^2(1-p)i^*s^* + p\beta^2(\mu+\alpha)i^*s^* - p\beta^2\mu i^*s^*)]\lambda + (\mu+\alpha)p\beta^2\mu i^*s^* \\ &+ \alpha\beta^2\mu(1-p)i^*s^* = 0 \end{aligned}$$

Or this can be rewritten as $a_4\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda^1 + a_0\lambda^0 = 0$. Applying Routh-Hurwitz stability criterion we found that if all signs of the first column of the array have the same, all roots have negative real parts. In addition to this all polynomial coefficients must have the same sign, since a_4 is positive then the other coefficient of the polynomial must be positive. Then the endemic equilibrium point to be stable or unstable we restrict the sign of the remaining element in first column of the array, that is $b_1 = \frac{1}{a_3}(a_2a_3 - a_4a_1)$ and $c_1 = \frac{a_1a_2a_3 - a_3^2 - a_4a_1^2}{a_2a_3 - a_4a_1}$. Hence after some simplification we found that if $b_1 > 0$ and $c_1 > 0$ makes $R_0 > 1$ then the first column of the Routh-Hurwitz array have the same sign in this case the endemic equilibrium point (s^*, e^*, i^*, r^*) is stable. If either $b_1 < 0$ or $c_1 < 0$ then the first columns of the Routh-Hurwitz have sign change in this case the endemic equilibrium point (s^*, e^*, i^*, r^*) is unstable.

VI. Parameter Estimation based on Real data

The considered mathematical model can be used to represent the spread of Pneumonia disease in the case of Debre Berhan town. Our real data collected from our research site Debre Berhan town

Description	Total
Total number of male in Debre Berhan town	46,778
Total number of female in Debre Berhan town	56,672
Total number of population in Debre Berhan town	103,450
Number of new born individuals in Debre Berhan town	603
Number of individuals whose age between 0 and 2 years	2,578
Number of individuals whose age is greater than or equal to 65 years	4,815

Table 1: The gathered data about the total population and new born individual

Description	Total
Number of individuals who have taken pneumonia test	8,069
Number of individuals whose fast progress to the disease and directly go to the infected group	1,754
Number of individuals dies by pneumonia case	
Number of individuals dies by natural case	
Number of individuals who shows pneumonia positive	5,834

Table 2: The collected data about the pneumonia diseases and individuals who are died by the disease

Classes of initial population	Symbol	Total
Number of susceptible population initially	S_0	89,906
Number of exposed population initially	E_0	7,393
Number of infected population initially	I_0	5,834
Number of recovered population initially	R_0	317
Total initial population	N ₀	103,450

Table 3: The total number of initial populations in each of the compartments of the model

Most parameters are identified from the collected data from Debre Berhan town, but there exist few parameters which can be estimated based on different research findings. For instance, the latency coefficient and the recovery coefficient are estimated depending on different research findings. Incubation period is the time taken from being infected with the bacteria or virus to development of symptoms [6]. The incubation period for Pneumonia is seven to 10 days [5, 7]. Using this the mean incubation period is the mean value of seven to 10 days.That is; mean incubation period = $\frac{7+10}{2}$ = 8.5 days.The contagious period for Pneumonia is 24 to 48 hours [10, 8]. Using this the mean contagious period is the mean value of 1 to 2 days.That is; mean contagious period

 $=\frac{1+2}{2}=1.5$ days. The mean incubation period of the Pneumonia is the inverse of the latency coefficient. Using this information and the collected real date from Debre Berhan town we do have

$$s_{0} = \frac{Number of initial suceptible individuals}{total population}, e_{0} = \frac{Number of initial exposed individuals}{total population}, i_{0} = \frac{Number of initial recoverd individuals}{total population}, i_{0} = \frac{Number of initial recoverd individuals}{total population}, i_{0} = \frac{Number of initial recoverd individuals}{total population}, i_{0} = \frac{Number of population}{total population}, i_{0} = \frac{Number of population}{Thenumberof pneumonian fected individuals}, i_{0} = \frac{1}{meanincubation periodof pneumonia}, i_{1} = \frac{number of newbornindividuals}{total numberof newbornindividuals}, i_{1} = \frac{number of newbornindividuals}{total number of population}, i_{1} = \frac{number of newbornindividuals}{total number of population}, i_{1} = \frac{number of individuals}{total number of population}, i_{1} = \frac{number of individuals}{total number of population}}, i_{1} = \frac{1}{\frac{number of individuals}{total number of population}}}, i_{1} = \frac{1}{\frac{number of individuals}{total number of population}}}}, i_{1} = \frac{1}{\frac{number of individuals}{total number of population}}}}, i_{1} = \frac{1}{\frac{number of individuals}{total number of infected people}}}, i_{1} = \frac{1}{\frac{1}{2}}, i_{1} = \frac{1}{2}, i_{1}$$

$$= 1 - \frac{\text{Number of individuals whose fastly progress to the disease and directly go to the infected group}{Total number of infected individuals}$$

Descriptions	Symbols	Values
Fractions of susceptible individuals	<i>S</i> ₀	0.869076848
Fractions of exposed individuals	e_0	0.071464475
Fractions of infected individuals	i_0	0.056394393
Fractions of recovered individuals	r_0	0.003064282
The transmission coefficient	β	0.723014004
The latency coefficient	α	0.117647058
The recovery coefficient	γ	0.66666667
The natural birth and death rate	μ	0.003107781537
The pneumonia induced death rate	d	0.005485087819
Proportion of fast progression rate	p	0.300651354
Proportion of slow progression rate	(1-p)	0.699348645

Table 4: The estimate values of the state variables and parameters

VII. Model Application

6.1 Stability of Disease free equilibrium point based on Real parameter estimation

After substituting the estimated parameter values in table 4 in to model(5) - (8), we do have the following system of non-linear differential equations

$\frac{ds}{dt} = 0.003107781537 - (0.003107781537 + 0.723014004i)s$,	(9)
$\frac{de}{dt} = 0.505638864 si - (0.003107781537 + 0.117647058)e$,	(10)
$rac{di}{dt} = 0.117647058e - 0.217375139si - 0.675259469i$,	(11)
$\frac{dr}{dt} = 0.6666666666i - 0.003107781537r$	(12)

Based on the above real data we found the basic reproduction number $R_0 = 1.051448663 > 1$. This shows that the disease free equilibrium point of our SEIR Pneumonia model system is unstable and the endemic equilibrium point is stable. This means Pneumonia can be spread through the community of Debre Berhan town. By making the right hand side of equations (9) - (12) equal to zero with assumption i = 0 we found that the disease free equilibrium point is $E_0 = (s^0, e^0, i^0, r^0) = (1, 0, 0, 0)$ and with the assumption $i \neq 0$ we found that the endemic equilibrium point is

 $E^*(s^*,e^*,i^*,r^*) = (0.951068688,0.0008806970415,\ 0.0002211458277,0.475463028\,).$

6.2 Stability analysis of disease free equilibrium point

The Jacobian matrix of the dynamical system (9) - (12) at the disease free equilibrium point $(s^0, e^0, i^0, r^0) = (1, 0, 0, 0)$ is

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J(1,0,0,0) =	-0.00310778 0 0 0	0 -0.120754839 0.117647058 0	-0.72301400 0.50563886 -0.45788812 0.66666666	04 0 4 0 19 0 -0.00310778	
The corresponding characte	eristic equation is				
-0.00310778 -	λ 0	-0.7	723014004	0	
0	-0.120754	$839) - \lambda = 0.5$	05638864	0	- 0
0	0.11764	7058 -0.45	7888119 – λ	0	- 0
0	0	0.	6666666	$-0.00310778 - \lambda$	

After some calculation we found that the eigen values are $\lambda_1 = -0.00310778$, $\lambda_2 = -0.00310778$, $\lambda_3 = -0.585803579$ and $\lambda_4 = 0.0071606215$. The first three eigenvalues of the Jacobian matrix are negative values but the fourth one is positive, therefore the disease free equilibrium point (1.0,0,0) is unstable. This implies that the disease exists in the community of Debre Berhan town.

6.3 Stability analysis of endemic equilibrium point

 $E^*(s^*, e^*, i^*, r^*) = (0.951068688, 0.0008806970415, 0.0002211458277, 0.475463028)$ The Jacobian matrix of the dynamical system (0) (12) at the orderic activities point is

The Jacobian matrix of the dynamical system $(9) - (12)$ at the endemic equilibrium point is						
	-0.0032676730	67 0	-0.694	413329	0	1
$I(s^* \ o^* \ i^* \ r^*) -$	0.00011181992	51 -0.1207	54839 0.480	388908	0	
$\mathbf{J}(\mathbf{S},\mathbf{e},\mathbf{t},\mathbf{r}) =$	0.000048071605	509 0.11764	7058 0.468	52078	0	
	L O	0	0.666	66666 -0	.0031077	78]
The corresponding charact	teristic equation is					
-0.0032676730	$67 - \lambda$	0	-0.694413329	0	I	
0.0001118199	0251 -0.1207	54839 – λ	0.480888908	0		- 0
0.0000480716	0509 0.117	647058 –	0.46852078 - 2	ι Ο		- 0
		0	0.66666666	-0.003107	$778 - \lambda$	
Or λ^4 + 0.090552962 λ^3 ·	+ 0.700864956 λ^2	+ 0.02660224	$8\lambda + 0.000002$	73612458 = 0	0	
Applying Routh Hurwitz s	tability criterion w	e found that the	Routh Hurwitz	array		
λ^4	1	0.7008649	56 0.0000)273612458	0	
λ^3 0	0.090552962	0.0266022	48	0	0	
λ^2 0	.407089378	0.00002736	12458	0	0	
λ^1 0	0.026601639	0		0	0	
0.00 ا ۵۷	000273612458	0		0	0	

By Routh-Hurwitz stability criteria from this Routh array we understand that the first columns have the same sign and thus all the roots of the characteristic polynomials are negative. Therefore, the endemic equilibrium point

 $E^*(s^*, e^*, i^*, r^*) = (0.951068688, 0.0008806970415, 0.0002211458277 0.475463028)$ It is stable. This means that Pneumonia is spread through the community of Debre Berhan town.

VIII. Numerical Simulation

In this section, we found the effect of control parameters on the basic reproduction number of our dynamical system $R_0 = \frac{\beta(\alpha+p\mu)}{(\mu+\alpha)(\mu+d+\gamma)}$. The parameters are the transmission coefficient β , the latency coefficient α , the recovery coefficient γ , the proportion of fast progression rate *p*, the disease induced death rate *d*, the birth rate μ . In this analysis we discuss the effect of each parameters change on the basic reproduction number graphically using win plot software, where the parameter values are taken from table 4.

7.1 Basic reproduction number R_0 versus transmission coefficient β

Let us take our control parameter to be the transmission coefficient β and the remaining parameters taken to be constant. Then $R_0(\beta) = 1.454257688\beta$. The graphical representation of the basic reproduction number R_0 versus the transmission coefficient β is



Figure 1: From this, graph we observe that when $\beta < 0.687636041$, then $R_0 < 1$. And when $\beta > 0.687636041$, and then $R_0 > 1$

7.2 Basic reproduction number R_0 versus recovery coefficient γ

Let us take the second control parameter γ with various values, which is recovery coefficient and the remaining parameters are assumed to be constant. Then $R_0(\gamma) = \frac{0.085736024}{0.001037630556 + 0.120754839 \gamma}$. The graphical representation of basic reproduction numberversus the recovery coefficient γ is given by



Figure 2: From this, graph we observe that as $\gamma \rightarrow \infty$, $R_0 \rightarrow 0$. If $\gamma < 0.624072037$, then $R_0 > 1$. And if $\gamma > 0.624072037$, and then $R_0 < 1$

The reproduction number R_0 is inversely proportional to γ .

7.3 Basic reproduction number R_0 versus disease induced death rate d

Let us take the third control parameter d with various values, which is disease induced death rate and the remaining parameters are assumed to be constant. we get $R_0(d) = \frac{0.085736024}{0.080878506 + 0.12754839 d}$. The following graph shows basic reproduction number in the vertical axes versus the disease induced death rate in the horizontal axes.



Figure 3: From this, graph we observe that as $d \to \infty$, $R_0 \to 0$. When d < 0.04022679 then $R_0 > 1$. And when d > 0.04022679, and then $R_0 < 1$. The basic reproduction number R_0 is inversely proportional to d.

7.4 Basic Reproduction number R_0 versus latency coefficient α

Let us take forth control parameter to be α which is the latency coefficient and the remaining parameters are taken to be constant we get $R_0(\alpha) = \frac{0.723014004 \,\alpha + 0.000675554}{(0.002098559128 + 0.675259539 \,\alpha)}$. The graphical representation of the basic reproduction number versus the latency coefficient in the is given by



Figure 4: From this, geaph we see that as $\alpha \rightarrow \infty$, $R_0 \rightarrow 1.070720163$. When $\alpha < 0.029798368$ then $R_0 < 1$. And when $\alpha > 0.029798368$ then $R_0 > 1$.

7.5 Basic reproduction number R_0 versus the birth rate μ

Let us take the fifth control parameter to be μ which is the birth rate and the remaining parameters are assumed to be constant get $R_0(\mu) = \frac{0.08506047 + 0.217375139 \,\mu}{(\mu^2 + 0.789798815 \,\mu + 0.079076676)}$. The graphical representation of the basic reproduction number versus the birth rate is given by



Figure 5: From this, graph we observe that analytically, $as\mu \rightarrow \infty$, $R_0 \rightarrow 0$. When $\mu < 0.010269206$ and then $R_0 > 1$. And when $\mu > 0.010269206$ then $R_0 < 1$

7.6 Basic reproduction number R_0 versus the proportion of fast progression rate p

Let we take the last control parameter to be p which is the proportion of fast progression rate and the remaining parameters are assumed to be constant. Then $R_0(p) = 1.04316381 + 0.027556364p$

The following graph shows basic reproduction number in the vertical axes versus is the proportion of fast progression rate in the horizontal axes.



Figure 6: From this graph, we observe that the basic reproduction number R_0 is directly proportional to p.

IX. Sensitivity Analysis

Sensitivity analysis investigates the relations between control parameters and basic reproduction number of the dynamical system and a property of the observable outcome, which represents some phenotypic features of the modeled system. In this work we use sensitivity analysis to determine the effect of those parameters and the most influential parameter that affects the basic reproduction number. The primary objective of Sensitivity analysis is to calculate $\frac{\partial R_0 x_i}{\partial x_i R_0}$ where x_i is represents those parameter β , α , p, μ , γ , d, which are exist in our mathematical model. Thus the normalized sensitivity indices of the reproduction number with respect to β , α , p, μ , γ and dare given by:

i) $SI(\beta) = 1$ ii) $SI(\alpha) = \frac{\alpha\mu(1-p)}{(\alpha+\mu)(\alpha+p\mu)}$ iii) $SI(p) = \frac{p\mu}{\alpha+p\mu}$

iv)
$$SI(\mu) = \frac{\mu[p(\mu+d+\gamma)-(2\mu+d+\gamma+\alpha)]}{(\alpha+p\mu)(\mu+d+\gamma)}$$

v)
$$SI(\gamma) = -\frac{\gamma}{\mu + d + \gamma}$$

vi) $SI(d) = -\frac{d}{\mu + d + \gamma}$

Based on the values of each parameter given in table 4 the values of normalized sensitivity indices are calculated as follows:

Parameter	Sensitivity index
β	-2.920911325808973
μ	+1
γ	-0.987274718
α	+0.0178568196314191
d	-0.008122932
р	+0.007881397404381

Table 5: Numerical sensitive indices of control parameters

From the sensitivity analysis we observe that the transmission coefficient β is more sensitive parameter than the other five parameters.

X. Results and Discussions

In this work we have been dealing with dynamics of Pneumonia disease based on the deterministic mathematical model. To understand the dynamics of the disease, we first discussed the initial mathematical model of Pneumonia disease, the authors divide the total population in four categories namely the susceptible group S(t), the exposed group E(t), the infected group I(t) and the recovered group R(t). In the initial mathematical model the authors assumed that all population group decreases by natural death rate μ , but in our extended model the infected peoples additionally decreases due to disease induced death rate d and also the authors of the initial mathematical model considers all susceptible peoples inters to the exposed group, but in this research the susceptible individuals directly inters to the infected group due the factor associated to fast progression rate p and the remaining susceptible people will inters to the exposed group due to strong immunity individuals by slow progression rate (1 - p).

A complete qualitative analysis of the model was done. It was first showed that the positivity of the solution, where the model is epidemiologically and mathematically meaningful and Boundedness of the solution region. The model has two equilibrium points, the disease free equilibrium point E^0 and the endemic equilibrium point E^* . The model basic reproduction number R_0 was calculated using the next generation matrix i.e. $R_0 = \frac{\beta(\alpha + p\mu)}{(\mu + \alpha)(\mu + d + \gamma)}$, which depends on six parameters. The stability analysis of the equilibrium points was investigated using Routh-Hurwitz stability criteria. Using real data collected from Debre Berhan town the model is numerically analyzed. Using these collected real data we determined the disease free equilibrium point is (1, 0, 0, 0)and the endemic equilibrium point is (0.951068688, 0.00088069704156, 0.0002211458277, 0.47546302) and the basic reproduction number is $R_0 = 1.051448663$ and thus we observe that the disease free equilibrium point is unstable and the endemic equilibrium point is stable since $R_0 > 1$.

From the numerical simulations that has done above we observe that, when the transmission coefficient increases ($\beta > 0.687636041$), then the basic reproduction number also increases ($R_0 > 1$). This means the number of infected population increases in the community and the disease persist. If the recovery coefficient increases ($\gamma > 0.590832377$), then the basic reproduction number decrease ($R_0 < 1$) which indicates the number of infected people decrease in the community that is the spread of the disease reduces. When the disease induced death rate increases (d > 0.04022679), then the basic reproduction number decreases($R_0 < 1$), this implies that the number of infected population decreases and the spread of the disease reduce.

If the latency coefficient increase ($\alpha > 0.029798368$), then the basic reproduction number also increase ($R_0 > 1$). This means the number of infected population increases in the community and the disease persist. When the natural birth rate increase ($\mu > 0.010269206$), then the basic reproduction number decrease ($R_0 < 1$) which means that the number of infected population decreases and the spread of the disease reduce and if the proportion of fast progression rate increases, then the basic reproduction number R_0 increases. This means that the number of infected class increase.

In general, results from numerical analysis shows that when the transmission coefficient, latency coefficient and the proportion of fast progression rate increases, then the basic reproduction number increases, this implies that the infected population increases, this will result increasing on the transmission of Pneumonia, when it decrease the basic reproduction number decrease, this implies that the infected population decrease. This will result decreasing on the transmission of Pneumonia. In other way when the recovery coefficient, the natural birth rate and the disease induced death rate decrease, then the basic reproduction number increases, this implies that the infected population increases, this will result increasing on the transmission of Pneumonia. When it increases, then the basic reproduction number decrease, this implies that the infected population decrease. This will result decreasing on the transmission of Pneumonia. When it increases, then the basic reproduction number decrease, this implies that the infected population decrease. This will result decreasing on the transmission of Pneumonia.

XI. Conclusions and Recommendations

Conclusions

Based on our data collected from Debre Berhan town we have made the parameter estimation which gives the basic reproduction number is $R_0 = 1.051448663$. This shows that the Pneumonia generation number is greater than one and guarantees the spread of Pneumonia is high in the community of Debre Berhan town. From the sensitivity analysis we understand that the transmission coefficient β is more sensitive parameter than the other five parameters. This means that the transmission coefficient β is the most influential parameter on the increment of basic reproduction number than the other five parameters.

Recommendations

Based on the finding of the study the current researcher recommends the following. The basic control parameter that decreases the number of infected people is the transmission coefficient $\beta = 0.687636041$. Therefore, to be the basic reproduction number less than one the transmission coefficient β should be less than 0.687636041. The second basic control parameter that decreases the basic reproduction number is the recovery coefficient $\gamma = 0.590832377$. Therefore, to be the basic control parameter that decreases the basic reproduction number less than one the recovery coefficient γ should be greater than 0.590832377. The third basic control parameter that decreases the basic reproduction number is the latency coefficient $\alpha = 0.029798368$. Therefore to be the basic reproduction number less than one the latency coefficient should be less than 0.029798368.

References

- Abdulkarim, A. A., Ibraheem, R. M., Adegboye, A. O., Johnson, W. B. R., &Adeboye, M. A. N. (2013). Childhood pneumonia at the University of Ilorin Teaching Hospital, Ilorin Nigeria. *Nigerian Journal of Paediatrics*, 40(3), 284-289.
- [2]. Al-Sayouri, S. (2014). Predictive analytics of hospital readmissions using an integrated data mining framework. State University of New York at Binghamton.
- [3]. American Lung Association (2013), Pneumonia,
- [4]. Barbara Boldin, Deterministic structured population epidemic models, 2003. University of Ljubljana, Slovenia.
- [5]. Charles Patrick Davis, MD, PhD. (2017). Is Pneumonia Contagious?
- [6]. DrPoonamSachdev (2011), what is the incubation period of Pneumonia?
- [7]. Dr. Richard Foxx, MD, (2016). Is Pneumonia Contagious?
- [8]. Karlsson, D., Jansson, A., Normark, B. H., & Nilsson, P. (2008). An individual-based network model to evaluate interventions for controlling pneumococcal transmission. *BMC infectious diseases*, 8(1), 83.
- [9]. LenkaBubniakov'a, (2007). The mathematics of infectious disease, Comenius University. Bratislava, p.3,4
- [10]. MengistuKassa& Samba Narasimha Murthy (2016). Pneumonia Control Measures Under Five Year Children, Hawassa University, Volume 12, PP 64-70.
- [11]. Nita H.Shah, JyotiGupta.SEIR Model and Simulation for Vector Born Diseases, May 24, 2013.Department of Mathematics, Gujarat University, Ahmedabad, India.
- [12]. Nthiiri, J. K., Lawi, G. O., & Manyonge, A. (2015). Mathematical Model of Pneumonia and HIV/AIDS Co-Infection in the Presence of Protection. *International Journal of Mathematical Analysis*, 9(42), 2069-2085.
- [13]. Pneumonia Fact Sheet July 2014
- [14]. Pneumonia Fact sheet Updated September 2016.
- [15]. Roxana L opez-Cruz.Structured SI epidemic models with application to HIV epidemic. May 2006.Ariona state university.
- [16]. Wardlaw, T. M., Johansson, E. W., Hodge, M., World Health Organization, & UNICEF. (2016). Pneumonia: the forgotten killer of children. 2006. New York, NY.
- [17]. World Health Organization. (1991).Technical bases for the WHO recommendations on the management of pneumonia in children at first-level health facilities.