Analytical model for childhood pneumonia, a case study of Kenya

Abstract

Pneumonia is an infection of the lungs that is caused by bacteria, viruses, fungi, or parasites. For a long time to the best of our knowledge there have not been reliable mathematical model for childhood pneumonia in Kenya. This research study developed a deterministic model based on the Susceptible-Vaccinated-Infected-Treated-Recovered-Susceptible compartment classes. The study used the partial differentiation of control reproduction number(R_C) toinvestigate effects of; environment, efficacy of vaccination drug and treatment. Model analysis indicates the system lie in feasible region, it is bounded, has no backward bifurcation and there exists unique endemic equilibrium point when control reproduction number is greater than unity. Local and global stability of the equilibrium points indicated that control reproduction has to be maintained at less than unity to eradicate the disease. Sensitivity analysis of the control reproduction number indicates that improved vaccination drug's efficacy, attaining herd immunity, higher treatment rates and lower effects of environment are the best intervention strategies to lower impact of the pneumonia of the children under the age of five years in Kenya.

Keywords: Control reproduction number, herd immunity, sensitivity analysis, disease free equilibrium point (DFE), endemic equilibrium point (EEP), local and global stability**.**

1. Introduction.

Pneumonia is an infection of the lungs that is caused by bacteria, viruses, fungi, or parasites

Pneumonia is characterized primarily by inflammation of the alveoli in the lungs or by alveoli that are filled with fluid. When a person breathes pneumonia-causing pathogens into his lungs and body's immune system cannot prevent entry, the organisms settle in small air sacs called alveoli and continue multiplying. The host body sends white blood cells to attack the infection

causing the sacs to be filed with fluid and pus - causing pneumonia (World Health Organization(WHO), 2006).

Pneumonia is most dangerous for older adults, babies, and people with other diseases or impaired immune systems. Pneumonia symptoms include cough, crusty or green mucus coughed up from lungs, fever, fast breathing and shortness of breath, shaking chills, chest pain that usually worsens when taking a deep breath, fast heartbeat, fatigue and feeling very weak, nausea and vomiting, diarrhea, sweating, headache, muscle pain, confusion or delirium and dusky or purplish skin color (cyanosis) from poorly oxygenated blood (WHO, 2006).

Childhood pneumonia can be spread through inhaling viruses and bacteria that are commonly found in a child's nose or throat; they may also spread via air-borne droplets from a cough or sneeze, direct contact or through blood contact, especially during and shortly after birth (WHO, 2006).

Pneumonia can be prevented by immunization, adequate nutrition and by addressing environmental factors; it is also treatable. The following environmental factors also increase a child's susceptibility to pneumonia: indoor air pollution caused by cooking and heating with biomass fuels (such as wood or dung), living in crowded homes and parental smoking. Streptococcus pneumonia is the most common cause of bacterial pneumonia and remains a substantial source of morbidity and mortality in both developing and developed countries, despite a century of study and the development of antibiotics and vaccination. Viral pneumonias is caused by adenoviruses, rhinovirus, and influenza virus and parainfluenza virus. Viral pneumonia is treated with rest and plenty of fluids. Fungal pneumonia is not common, but it may occur in individuals with weakened immune systems due to AIDS immunosuppressive drugs or other medical problems. Fungal pneumonias are usually treated with antifungal medications. The most common parasites causing pneumonia are toxoplasma gondii, strongyloidesstercoralis and ascariasis .These parasites typically enter the body through the skin or the mouth and travel to the lungs, usually through the blood (WHO, 2006).

Bacteria and viruses are the primary causes of pneumonia while fungi and parasitic pneumonia can also occur to children under the age of five years with weakened immune system due to malnutrition, AIDS and other medical problems. Fungi and parasitic pneumonia can infect those children under the age of five years mildly or chronically and may lead to death (WHO, 2006).

In 2011, pneumonia was the leading killer of children under the age of five worldwide, responsible for nearly one in every five global child deaths annually. More than 99 percent of deaths from pneumonia occurred in the developing world, where access to healthcare facilities and treatment is often out of reach for many children (WHO, 2012).

Globally, reducing the mortality rate for the children under the age of five years by two thirds between 1990 and 2015 is one of the millennium development goals and requires a complex mix of interventions in health and other sectors. UNICEF indicated that Kenya is lagging behind in East Africa in attaining Millennium Development Goals (MDGs) on reducing child mortality rates (Mosoku, 2013).Globally, the mortality rate for children under the age of five years dropped by almost 50 per cent, from 90 deaths per 1,000 live births in 1990 to 48 in 2012, the under-five years of age deaths was mainly caused by preventable diseases; most of the 6.6 million deaths in children under the age of five years in 2012 were from leading infectious diseases such as pneumonia, diarrhea and malaria. Sub-Saharan Africa continues to confront a tremendous challenge of having the highest mortality rate in the world for children under the age of five years which is more than 16 times the average for developed regions (Programme, 2014).

The Kenya Vision 2030, recognized research as one of the pillars to accelerate development. Ministry of Health (MoH) was tasked to formulate policy which would focus on the preventive care as opposed to curative care (Kenya, 2007). Kenya is one of the countries in Africa which is not on target for the attainment of MDG because it requires to reduce the mortality rate under five years to 33 deaths per 1,000 live births by 2015 (Programme, 2014). According to UNICEF, many children in Kenya continue to die unnecessarily due to poor accessibility to recommended treatments. This is particularly the case for diarrhea and pneumonia, which still account for an estimated 20% and 16% of annual child deaths respectively. The Government of Kenya (GoK) has also initiated additional efforts to reduce child mortality, including the introducing of new vaccines to prevent diarrhea and pneumonia, but poor access to recommended treatments is still

a challenge (Ministry of public health, 2011). By 2009, pneumococcal vaccines (PCV 7, PCV 10, PCV 13 and PCV 6A) were introduced in Kenya (Mudhune et al 2009). After introduction of vaccine it was assumed effective treatment could avert the remainder of those deaths (Ministry of public health, 2011)

Surveillance data on Childhood Pneumonia in East African Region emphasized existence of different strains but it does not involve mathematical modeling (Mudhune, et.al, 2009).Existence of different types of pneumonia and vulnerability of the children under the age of five years immune system makes pneumonia a complex disease. Furthermore, many signs and symptoms of fungal infections are similar to those caused by bacteria and/or viruses. Most bacteria pneumonia would require culturing to isolate them which require a lot time (Ahmed, et al., 1998). In Kenya, to the best of our knowledge isolation of pneumonia is not carried out in the health facilities.

Laura L. Hammitt, et.al, 2012, conducted a case-control study of pneumonia etiology among children aged 1–59 months in rural Kenya and generally classified pneumonia into two broad categories that is severely infected and very severely infected. The Kenya Health information system (DHIS2), which is available online record age structured data of treated pneumonia in Kenya into two categories that is outpatient and inpatient classes. Vaccination of pneumococcal and haemophilus type b is already in place which is administered in three doses; 6 weeks, 10 weeks and 14 weeks after birth. The objective of this research study is to develop a deterministic model with Kenya attributes that is taking into account severely infected class (assumed to be mild infected in this study), very severely infected class (chronically infected in this study), inpatient class (assumed to be chronically treated in this study), outpatient (assumed to be mild treated in this study) and vaccinated class among others.

This research study involved mathematical modeling approach where the children under five years old will be subdivided into seven classes and the rate change from one class to another was formulated in nonlinear ordinary differential equations and analytical model analysis was carried

out. The study also determined partial derivative of the control reproduction number (R_C) to consider the effects of the environmental factors, critical treatment threshold and efficacy of vaccination drugs. The basic reproduction number (R_0) was determined from control reproduction number and will be used to determine the herd immunity. Model analysis was evaluated to ascertain qualitative behavior of the system.

2. Model development.

Joseph, et.al, 2012 developed a general pneumonia model for adults and children based on four compartmental classes (Vaccinated, Susceptible, Infected and Treated). Ngari, et.al, 2014, developed a general model based on three compartmental classes(Susceptible, Infected and Treated classes). Joseph, et.al, 2012 and Ngari, et.al, 2014 determined control reproduction number analyzed their model similarly like this research study. This study uses population based model where population was divided into seven compartments classes (Susceptible, Vaccinated, Mildly Infected, Chronically Infected, Mildly Treated, Chronically Treated and Recovered) based on infection characteristics and status of infection. The summary of the definition of terms, description of the variables and parameters are also available in appendix section.

2.1 Model description.

Let $N(t)$ be the total population of the under five years children which is divided into seven sub-classes: Susceptible to pneumonia class $S(t)$, vaccinated against pneumonia class V(t), mildly infected pneumonia class $I_M(t)$,chronically infected pneumonia class $I_C(t)$, chronically treated with pneumonia (in patient) class $T_C(t)$, mild treated with pneumonia (outpatient) class $T_m(t)$ and recovered from pneumonia R(t) after treatment.

The rates at which$I_M(t)$ and $I_C(t)$seek treatmentis given by $\tau_1 and$ τ_2 respectively, the recruitment rate (birth rate) is given by π, pneumonia induced death occur at a rate δ_1 and δ_2 in $I_C(t)$ and $T_C(t)$respectively, μ is the constant natural death rate in all the seven subclasses, β infection rate , ϕ is the proportion of born children vaccinated with available pneumonia vaccines (either streptococcus vaccines or haemophilus type b vaccine or both),ρis the rate of waning of treatment drugs after recovery, λ(t) is the force of infection, ϵ is the drug efficacy ($\epsilon = 1$ when the drug is 100% efficient $\epsilon = 0$ when the drug is useless),θ_1 is the rate at which the under five years $I_M(t)$ with pneumoniaprogresses to$I_C(t)$, θ_2 is the rate which under five years $T_C(t)$are discharged to $T_M(t)$ andthe recovery rates after treatment are $\gamma_1$andγ_2 for $T_M(t)$ and $T_C(t)$ respectively

2.2 Model assumptions.

The following assumptions were made when formulating the model;

- homogeneous mixing children population in Kenya,
- The recovery from natural immunity not significant.
- Through the literature search, to the best of our knowledge it is observed that no study has been carried out in Kenya to establish the type of childhood pneumonia in the population.
- It is not likely for less than five years of age to be infected with pneumonia immediately after treatment.
- Reinfection with pneumonia of different types and/or strain during treatment is assumed to be not significant in this study.
- The decreasing order of infectivity is; mildly infected, chronically infected, mildly treated and chronically treated.

- Constant natural death rate/progression rate to subsequent class
- Disease induced death is assumed to be higher in chronically infected than chronically treated class i.e.($\delta_1 > \delta_2$). Treatment reduces likelihood of dying significantly.
- Modification parameter k is such that $k \geq 1$, implying that the environmental factors increases force of infection.
- Once vaccinated child contract pneumonia, the vaccination drug is assumed to be useless.
- The effects of vertical transmission to pneumonia of the under five years from age brackets at least five years is assumed to be insignificant in this study.
- The pneumonia is assumed to be transmitted after effective contact between susceptible child under five years and infectious classes (mild and chronic) and/or treated classes (mild and chronic).
- The weak nature of child's immune system was assumed to be vulnerable to different types of pneumonia as long as they exist in population.

2.3 Model equations.

We obtain the following systems of equations representing the dynamics;

$$\frac{dS}{dt} = \phi\pi + \rho R - (k\lambda + \mu)S \qquad (2.3.1),$$

$$\frac{dV}{dt} = (1-\phi)\pi - (1-\epsilon)k\lambda V - \mu V \qquad (2.3.2),$$

$$\frac{dI_M}{dt} = Pk\lambda S + (1-\epsilon)k\lambda V - \omega_1 I_M \qquad (2.3.3),$$

$$\frac{dI_C}{dt} = (1-P)k\lambda S + \theta_1 I_M - \omega_2 I_C \qquad (2.3.4),$$

$$\frac{dT_M}{dt} = \tau_1 I_M + \theta_2 T_C - \omega_3 T_M \qquad (2.3.5),$$

$$\frac{dT_C}{dt} = \tau_2 I_C - \omega_4 T_C \qquad (2.3.6)$$

$$\frac{dR}{dt} = \gamma_1 T_m + \gamma_2 T_C - \omega_5 R \qquad (2.3.7).$$

where,

$N(t) = S(t) + V(t) + I_M(t) + I_C(t) + T_M(t) + T_C(t) + R(t),$

$\omega_1 = \theta_1 + \mu + \tau_1,$

$\omega_2 = \mu + \delta_1 + \tau_2,$

$\omega_3 = \gamma_1 + \mu,$

$\omega_4 = \gamma_2 + \mu + \theta_2 + \delta_2,$

$\omega_5 = (\mu + \rho)\ and\ 0 < P, k, \epsilon, \phi < 1.$

The initial conditions of the systems$[(2.3.1) - (2.3.7)]$ are represented by;

$S(0) = S_0, V(0) = V_0, I_M(0) = (I_M)_0, I_C(0) = (I_C)_0, T_M(0) = (T_M)_0, T_C(0) = (T_C)_0\ and\ R(0) = R_0.$

The force of infection denoted by $\lambda(t)$is given by:

$$\lambda(t) = \beta(I_M + \xi_1 I_C + \xi_2 T_M + \xi_3 T_C)$$

Where, $0 < \xi_3 < \xi_2 < \xi_1 < 1.$

Adding equations of system$[(2.3.1) - (2.3.7)]$, the rate of change of total population is given by,

$$\frac{dN}{dt} = \pi - \mu N - \delta_1 I_C - \delta_2 T_C.$$

3. Model analysis.

The model is analyzed by proving various theorems and algebraic computation dealing with different attributes.

3.1. Positivity and boundedness of the solutions.

We prove positivity and boundedness by stating and proving the theorem below.

Theorem1. *The region* Q is*given by*

$$Q = \left\{S(t), V(t), I_M, (t), I_C(t), T_M(t), T_C(t), R(t)(t) \epsilon R_+^7, N \leq \frac{\pi}{\mu}\right\}$$

is positively invariant and attracting with respect to model system$[(2.3.1) - (2.3.7)]$,

Proof.

Let$\{ S(t), V(t), I_M(t), I_C(t), T_M(t), T_C(t) and\ R(t)\}$ be any solutions of the system with non-negative initial conditions

$\{S(0) \geq\ 0, V(0) \geq 0, I_M(0) \geq 0, I_C \geq 0, T_M \geq 0, T_C \geq 0, R(0) \geq 0\}$.

Since,$\frac{dS}{dt} = \phi\pi + \rho R - (k\lambda + \mu)S$,it follows that $\frac{dS}{dt} \geq -(k\lambda + \mu)S$.On integration, we obtain $\frac{d}{dt}[S(t)e^{\int_0^t -(k\lambda+\mu)ds}] \geq 0$. Clearly, $S(t)e^{\int_0^t -(k\lambda+\mu)ds}$is a non-negative function of t, thus S (t) stays positive.

The positivity of $V(t), I_M(t), I_C, (t), T_M(t), T_C(t)\ and\ R(t)$ is proved along the same lines as follows:

$$\frac{dV}{dt} = (1 - \phi)\pi S - [(1 - \epsilon)k\lambda + \mu]V$$

$$\frac{dV}{dt} > -[(1 - \epsilon)k\lambda + \mu]V$$

$$\frac{dV}{V} > -[(1 - \epsilon)k\lambda + \mu]dt$$

$$V(t) = C_1 e^{-[(1-\epsilon)k\lambda+\mu]t}$$

Where C_1 is a constant of integration, applying initial condition at $t = 0$,

$$C_1 = V(0),$$

$$V(t) > V(0)e^{-[(1-\epsilon)k\lambda+\mu]t},$$

$$V(t) > V(0)e^{-[(1-\epsilon)k\lambda+\mu]t} \geq 0$$

Similarly,

$$\mathrm{I_M(t) > I_M(0)e^{-\omega_1 t} \geq 0,}$$

$$\mathrm{I_C(t) > I_C(0)e^{-\omega_2 t} \geq 0,}$$

$$\mathrm{T_M(t) > T_M(0)e^{-\omega_3 t} \geq 0,}$$

$$\mathrm{T_C(t) > T_C(0)e^{-\omega_4 t} \geq 0,}$$

$$\mathrm{R(t) > \mathit{R}(0)e^{-\omega_5 t} \geq 0.}$$

Taking the time derivative of our total population along its solution path gives:

$$\frac{\mathrm{dN}}{\mathrm{dt}} = \frac{\mathrm{dS}}{\mathrm{dt}} + \frac{\mathrm{dV}}{\mathrm{dt}} + \frac{\mathrm{dI_M}}{\mathrm{dt}} + \frac{\mathrm{dI_C}}{\mathrm{dt}} + \frac{\mathrm{dT_M}}{\mathrm{dt}} + \frac{\mathrm{dT_C}}{\mathrm{dt}} + \frac{\mathrm{dR}}{\mathrm{dt}}$$

$$\frac{dN}{dt} = \pi - \mu\mathrm{N} - \delta_1 I_C - \delta_2 T_C$$

Therefore,

$$\frac{dN}{dt} + \mu\mathrm{N} \leq \pi,$$

This implies that

$$\mathrm{N(t)} \leq \frac{\pi}{\mu}\{1 + \mathrm{c_2 e^{-\mu t}}\},$$

Where c_2 is the constant of integration.

Hence,

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

This proves the bounded of the solutions inside Q. This implies that all the solutions of our system$[(2.3.1) - (2.3.7)]$, starting in Q and remains in Q for all $t \geq 0$. Thus Q is positively invariant and attracting, and hence it is sufficient to consider the dynamics of our system in Q. This completes the proof.

3.2. Disease-free equilibrium point (DFE)

The disease-free equilibrium point (DFE) of the system$[(2.3.1) - (2.3.7)]$, is obtained by setting all the infectious classes, recovered class and treatment classes to zero. We get

$$\phi\pi - \mu S^0 = 0;\ (1-\phi)\pi - \mu V^0 = 0,$$

which yields,

$$S^0 = \frac{\phi\pi}{\mu};\ V^0 = \frac{(1-\phi)\pi}{\mu}$$

The DFE point for our system is given by,

$$E^0 = (S^0, V^0, I_C^0, I_M^0, T_M^0, T_C^0, R^0) = \left(\frac{\phi\pi}{\mu}, \frac{(1-\phi)\pi}{\mu}, 0,0,0,0,0\right).$$

The DFE point(E^0) is the infection free equilibrium point of the system$[(2.3.1) - (2.3.7)]$,which indicates that in absence of pneumonia the system will consist of two compartment classes (susceptible and vaccinated).

3.3. The basic reproduction number (R_o) and control reproduction number (R_C)

We use the next-generation matrix method to determine the control reproduction number $R_c$of the model (*Carlos Castillo-Chavez, 2001*) Using the notation f for a matrix of new infections terms andϖ for the matrix of the remaining transfer of infection terms in our system, we get,

$$\mathfrak{f} = \begin{pmatrix} Pk\lambda S + (1-\epsilon)\mathrm{k}\lambda \mathrm{V} \\ (1-P)\mathrm{k}\lambda S \\ 0 \\ 0 \end{pmatrix}, \varpi = \begin{pmatrix} \omega_1 I_M \\ -\theta_1 I_M + \omega_2 I_C \\ -\tau_1 I_M - \theta_2 T_C + \omega_3 T_M \\ -\tau_2 I_C + \omega_4 T_C \end{pmatrix}.$$

Let

$F_1 = Pk\lambda S + (1-\epsilon)\mathrm{k}\lambda \mathrm{V}$, $\quad F_2 = (1-P)\mathrm{k}\lambda S$, $\quad F_3 = 0,\ F_4 = 0,$

$\mathrm{F}_5 = \omega_1 \mathrm{I}_\mathrm{M}$, $\quad \mathrm{F}_6 = -\theta_1 \mathrm{I}_\mathrm{M} + \omega_2 \mathrm{I}_\mathrm{C}$, $\quad \mathrm{F}_7 = -\tau_1 \mathrm{I}_\mathrm{M} - \theta_2 \mathrm{T}_\mathrm{C} + \omega_3 \mathrm{T}_\mathrm{M},\ \mathrm{F}_4 = -\tau_2 \mathrm{I}_\mathrm{C} + \omega_4 \mathrm{T}_\mathrm{C}$

We obtain the matrices F and V by finding the Jacobian matrices of f and ϖevaluated at DFE respectively to obtain,

$$F = \beta\mathrm{k}\begin{pmatrix} R_1 & \xi_1 R_1 & \xi_2 R_1 & \xi_3 R_1 \\ R_2 & \xi_1 R_2 & \xi_2 R_2 & \xi_3 R_2 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

$where, R_1 = \mathrm{PS}^0 + (1-\epsilon)\mathrm{V}^0, R_2 = (1-\mathrm{P})\mathrm{S}^0 and,$

$$V = \begin{pmatrix} \omega_1 & 0 & 0 & 0 \\ -\theta_1 & \omega_2 & 0 & 0 \\ -\tau_1 & 0 & \omega_3 & -\theta_2 \\ 0 & -\tau_2 & 0 & \omega_4 \end{pmatrix}.$$

We now compute the inverse of V to obtain V^{-1} as below,

$$V^{-1} = \begin{pmatrix} \frac{1}{\omega_1} & 0 & 0 & 0 \\ \frac{\theta_1}{\omega_1\omega_2} & \frac{1}{\omega_2} & 0 & 0 \\ \frac{\theta_1\tau_2\theta_2 + \omega_2\omega_4\tau_1}{\omega_1\omega_2\omega_3\omega_4} & \frac{\theta_2\tau_2}{\omega_2\omega_3\omega_4} & \frac{1}{\omega_3} & \frac{\theta_2}{\omega_4\omega_3} \\ \frac{\theta_1\tau_2}{\omega_1\omega_2\omega_4} & \frac{\tau_2}{\omega_2\omega_4} & 0 & \frac{1}{\omega_4} \end{pmatrix}.$$

Multiplying the matrices F and V^{-1} to obtain,

$$FV^{-1} = \begin{pmatrix} T_1 & T_2 \\ T_3 & T_4 \end{pmatrix},$$

Where,

$$T_1 = \beta k \begin{bmatrix} \frac{R_1}{\omega_1} + \frac{R_1\theta_1\xi_1}{\omega_1\omega_2} + \frac{R_1\xi_2(\theta_1\theta_2\tau_2 + \tau_1\omega_2\omega_4)}{\omega_1\omega_2\omega_3\omega_4} + \frac{R_1\theta_1\xi_3\tau_2}{\omega_1\omega_2\omega_4} & \frac{R_1\xi_1}{\omega_2} + \frac{R_1\theta_2\xi_2\tau_2}{\omega_2\omega_3\omega_4} + \frac{R_1\xi_3\tau_2}{\omega_2\omega_4} \\ \frac{R_2}{\omega_1} + \frac{R_2\theta_1\xi_1}{\omega_1\omega_2} + \frac{R_2\xi_2(\theta_1\theta_2\tau_2 + \tau_1\omega_2\omega_4)}{\omega_1\omega_2\omega_3\omega_4} + \frac{R_2\theta_1\xi_3\tau_2}{\omega_1\omega_2\omega_4} & \frac{R_2\xi_1}{\omega_2} + \frac{R_2\theta_2\xi_2\tau_2}{\omega_2\omega_3\omega_4} + \frac{R_2\xi_3\tau_2}{\omega_2\omega_4} \end{bmatrix},$$

$$T_2 = \beta k \begin{bmatrix} \frac{R_1\xi_2}{\omega_3} & \frac{R_1\theta_2\xi_2}{\omega_3\omega_4} + \frac{R_1\xi_3}{\omega_4} \\ \frac{R_2\xi_2}{\omega_3} & \frac{R_2\theta_2\xi_2}{\omega_3\omega_4} + \frac{R_2\xi_3}{\omega_4} \end{bmatrix},$$

$$T_3 = T_4 = \begin{bmatrix} 0 & 0 \\ 0 & 0 \end{bmatrix}.$$

Using Mathematica software to obtain the eigenvalues $\{q(i), i = 1(1)4\}$of the matrix (FV^{-1}) we get,

$q(1) = q(2) = q(3) = 0,$

$$q(4) = \frac{\beta k}{\omega_1\omega_2\omega_3\omega_4}\{R_1\omega_2\omega_3\omega_4 + \xi_1(R_1\theta_1\omega_3\omega_4 + R_2\omega_1\omega_3\omega_4)$$
$$+ \xi_2(R_1\theta_1\theta_2\tau_2 + R_2\theta_2\tau_2\omega_1 + R_1\tau_1\omega_2\omega_4) + \xi_3(R_1\theta_1\tau_2\omega_3 + R_2\tau_2\omega_1\omega_3)\}.$$

The control reproduction number(R_C) is given by the spectral radius ζ (the dominant eigenvalue) of the matrix FV^{-1}, denoted by $\zeta(FV^{-1})$ is;

$$R_C = \frac{\beta k}{\omega_1\omega_2\omega_3\omega_4}\{R_1\omega_2\omega_3\omega_4 + \xi_1(R_1\theta_1\omega_3\omega_4 + R_2\omega_1\omega_3\omega_4)$$
$$+ \xi_2(R_1\theta_1\theta_2\tau_2 + R_2\theta_2\tau_2\omega_1 + R_1\tau_1\omega_2\omega_4) + \xi_3(R_1\theta_1\tau_2\omega_3 + R_2\tau_2\omega_1\omega_3)\}.$$

The control reproduction number(R_C) is the average number of susceptible children under the age of five years, one infectious child (mildly infected or chronically infected or mildly treated or chronically treated) can infect when combined interventions of vaccination and treatment are already in place.

The reproduction number($R_{\epsilon=1}$) when the vaccination drug efficacy is 100%, that is$\epsilon = 1$ is given by;

$$R_{\epsilon=1} = \frac{\beta k S^0}{\omega_1\omega_2\omega_3\omega_4}\{P\omega_2\omega_3\omega_4 + \xi_1(P\theta_1\omega_3\omega_4 + (1-P)\omega_1\omega_3\omega_4)$$

$$+ \xi_2(P\theta_1\theta_2\tau_2 + (1-P)\theta_2\tau_2\omega_1 + P\tau_1\omega_2\omega_4) + \xi_3(P\theta_1\tau_2\omega_3 + (1-P)\tau_2\omega_1\omega_3)\}.$$

The reproduction number($R_{\epsilon=1}$) is average number susceptible children under the age of five years, one infectious child (mildly infected or chronically infected or mildly treated or chronically treated) can infect when intervention of treatment are already in place and it is not possible for vaccinated children to contract pneumonia.

The reproduction number(R_T) when the rate at which mildly and chronically infected children ($\tau_1 and\ \tau_2$) are zero is given by;

$$R_T = \frac{\beta k}{(\omega_1 - \tau_1)(\omega_2 - \tau_1)}\{R_1(\omega_2 - \tau_1) + \xi_1\langle R_1\theta_1 + R_2(\omega_1 - \tau_1)\rangle\}.$$

The reproduction number(R_T) is average number susceptible children under the age of five years, one infectious child (mildly infected or chronically infected or mildly treated or chronically treated) can infect in absence of intervention of treatment.

The basic reproduction number(R_0) in absence of interventions is given by;

$$R_0 = \frac{\beta k S^0(P(\mu + \delta_1) + (\mu(1-P) + \theta_1)\xi_1)}{(\mu + \delta_1)(\mu + \theta_1)}.$$

The basic reproduction number(R_0) is average number susceptible children under the age of five years, one infectious child (mildly infected or chronically infected or mildly treated or

chronically treated) can infect in absence of interventions of treatment and vaccination.Thebasic reproduction number(R_0) will be used to determine herd immunity(q_c) as below,

$$q_c = 1 - \frac{1}{R_0}.$$

3.4. Existence of Endemic Equilibrium point for the model (EEP).

We state and prove the following theorem

Theorem 2

A positive endemic equilibrium exist whenever $R_C^* > 1$.

Proof

The dynamic systems of the equations$[(2.3.1) - (2.3.7)]$, is seven dimensional and analyzing it may not be tractable mathematically. After obtaining reproduction numbers of the full system, the goal of the model analysis is to understand qualitative behavior as opposed to exact solution of the system. The system$[(2.3.1) - (2.3.7)]$, is highly nonlinear and may be difficult to solve mathematically. The study proposed to reduce the system to four dimensions by combining all the infectious classes (mildly infected or chronically infected or mildly treated or chronically treated) into one compartment class (A). Let, $A(t) = I_M(t) + I_C(t) + T_M(t) + T_C(t)$,the time derivative of A is given by.$\frac{dA}{dt} = \frac{dI_M}{dt} + \frac{dI_C}{dt} + \frac{dT_M}{dt} + \frac{dT_C}{dt} = \frac{dN}{dt} - \frac{dS}{dt} - \frac{dV}{dt}$.

The system of equations$[(2.3.1) - (2.3.7)]$, reduces to,

$$\frac{dS}{dt} = \phi\pi + \rho R - (k\lambda + \mu + \phi\pi)S \qquad (3.4.1),$$

$$\frac{dV}{dt} = (1 - \phi)\pi - k(1 - \epsilon)\lambda V - \mu V \qquad (3.4.2),$$

$$\frac{dA}{dt} = -(\mu + \Omega)A - \delta_1 I_C - \delta_2 T_C + k\lambda S + k(1 - \epsilon)\lambda V - \gamma_1 T_m - \gamma_2 T_C \qquad (3.4.3),$$

$$\frac{dR}{dt} = \gamma_1 T_m + \gamma_2 T_C - \omega_5 R \qquad (3.4.4).$$

Since the following parameters$\{\gamma_1, \gamma_2, \delta_1, \delta_2, \mu, \xi_1, \xi_2$ and $\xi_3\}$ are less or equal to one or greater or equal to zero, it follows by closure property the members below can be expressed as subsets of A or A^*

- $\{I_M^*, \xi_1 I_C^*, \xi_2 T_M^*, \xi_3 T_C^*\} \subseteq A^*$,
- $\{\gamma_1 T_m, \gamma_2 T_C, \delta_1 I_C, \delta_2 T_C, \mu A, \xi_1 I_C, \xi_2 T_M, \xi_3 T_C\} \subseteq A$,
- $\{I_M^* + \xi_1 I_C^* + \xi_2 T_M^* + \xi_3 T_C^*\} \subseteq A^*$,
- $(\delta_1 I_C + \delta_2 T_C + \gamma_1 T_m + \gamma_2 T_C) \subseteq A$,
- $(\gamma_1 T_m + \gamma_2 T_C + \mu A) \subseteq A$,
- $(I_M + \xi_1 I_C + \xi_2 T_M + \xi_3 T_C) \subseteq A$.

Let us introduce these subsets as parameters of A or A^* as indicated below,

- $\gamma_1 T_m + \gamma_2 T_C + \mu A = \Omega_2 A$,
- $\delta_1 I_C + \delta_2 T_C + \gamma_1 T_m + \gamma_2 T_C = \Omega_1 A$,
- $I_M + \xi_1 I_C + \xi_2 T_M + \xi_3 T_C = \Omega_3 A$,
- $I_M^* + \xi_1 I_C^* + \xi_2 T_M^* + \xi_3 T_C^* = \Omega_3 A^*$,

Where,Ω_1, Ω_2 *and* $\Omega_3$is less or equal to one. It is clear from the system$[(3.4.1) - (3.4.4)], \Omega_3 \geq \Omega_1 \geq \Omega_2$. After the change of variables the system of equations $[(3.4.1) - (3.4.4)]$ becomes,

$$\frac{dS}{dt} = \phi\pi + \rho R - (k\lambda + \mu)S \qquad (3.4.5),$$

$$\frac{dV}{dt} = (1 - \phi)\pi - k(1 - \epsilon)\lambda V - \mu V \qquad (3.4.6),$$

$$\frac{dA}{dt} = k\lambda S + k(1-\epsilon)\lambda V \ - \Omega_1 A \qquad (3.4.7),$$

$$\frac{dR}{dt} = \Omega_2 A - \omega_5 R \qquad (3.4.8).$$

The control reproduction number (R_C^*) of the system $[(3.4.5) - (3.4.8)]$ using the second generation matrix is given by,

$$R_C^* = \frac{\beta k \Omega_3 H_2}{\Omega_1},$$

where, $H_2 = S^0 + (1-\epsilon)V^0$.

After the change of variables, the force of infection at endemic equilibrium point $(E^{**} = (S^{**}, V^{**}, A^{**}, R^{**})$ of the system $[(3.4.5) - (3.4.8)]$will be given by$\lambda^{**} = \beta\Omega_3 A^{**}$.The endemic equilibrium point,$E^{**} = (S^{**}, V^{**}, A^{**}, R^{**}$of the system $[(3.4.5) - (3.4.8)]]$, is obtained by equating the system to zero to obtain;

$$\phi\pi + \rho R^{**} - (k\lambda^{**} + \mu)S^{**} = 0 \qquad \text{(i)},$$

$$(1-\phi)\pi - (1-\epsilon)k\lambda^{**}V^{**} - \mu V^{**} = 0 \qquad \text{(ii)},$$

$$k\lambda^{**}S^{**} + k(1-\epsilon)\lambda^{**}V^{**} - \Omega_1 A^{**} = 0 \qquad \text{(iii)},$$

$$\Omega_2 A^{**} - \omega_5 R^{**} = 0 \qquad \text{(iv)}.$$

Solving the system of equation above [(i) – (iv)] in terms of λ^{**} using Mathematica software we obtain;

$$S^{**} = \frac{\phi\pi + \rho R^{**}}{k\lambda^{**} + \mu},$$

$$V^{**} = \frac{(1-\phi)\pi}{k\lambda^{**}(1-\epsilon) + \mu},$$

$$A^{**} = \frac{k\pi\lambda^{**}((-1+\epsilon)(k\lambda^{**}+\mu)-\epsilon\mu\phi)\omega_5}{(k(-1+\epsilon)-\mu)((k\lambda^{**}+\mu)\omega_5\Omega_1 - k\lambda^{**}\rho\Omega_2)},$$

$$R^{*} = \frac{\Omega_2 A^{**}}{\omega_5}.$$

Substituting A^{**} using Mathematica software to solve the equation below we obtain two cases

$$\lambda^{**} - \beta\Omega_3 A^{**} = 0,$$

Case 1; $\lambda^{**} = 0$, which correspond to the disease free equilibrium point(E^{00}) of the system $[(3.4.5) - (3.4.8)]$ given by ;

$$E^{00} = (S^{00}, V^{00}, A^{00}, R^{00}) = (\frac{\phi\pi}{\mu}, \frac{(1-\phi)\pi}{\mu}, 0,0)$$

Case 2; the value(s) of λ^{**} obtained by the quadratic equations below correspond to endemic equilibrium point.

$$a(\lambda^{**})^2 + b\lambda^{**} + c = 0,$$

Using Mathematica software a, b and c are obtained as follows,

$$a = k^2(-1+\epsilon)(\omega_5\Omega_1 - \rho\Omega_2) < 0,$$

$$b = k((-2+\epsilon)\mu - \left(\frac{\pi(-1+\epsilon)R_C^*}{H_2}\right)\omega_5\Omega_1 + \mu\rho\Omega_2,$$

$$c = \mu\left\{-\mu + \frac{\pi\big(1+\epsilon(-1+\phi)\big)R_C^*}{H_2}\right\}\omega_5\Omega_1.$$

For real positiveλ^{**}, $b^2 > 4ac$. $Since\ a < 0, \lambda^{**} > 0\ if\ and\ only\ if\ \ c > 0, i.e$

$$-\mu + \frac{\pi\big(1+\epsilon(-1+\phi)\big)R_C^*}{H_2} > 0,$$

After algebraic manipulation it follows that the conditions necessary and sufficient for$\lambda^{**} > 0$ are $R_C^* > 1$ which completes the proof.

3.5 Bifurcation analysis.

Mathematical models with vaccination often undergo bifurcation which makes the control of the infectious diseases difficult (Braurer, 2004). This bifurcation will be explored, using the Centre Manifold theory (Junli et,al, 2011). The change of variables are made first for simplicity. Let $S = y_1, V = y_2, A = y_3$ and $R = y_4$,So that$N = y_1 + y_2 + y_3 + y_4$.Further, by using vector notation$y = (y_1, y_2, y_3, y_4)^T$, the pneumonia model $[(3.4.5) - (3.4.8)]$can be written in the form $\frac{dy}{dt} = F(y),\ with\ F = (p_1, p_2, p_3, p_4)^T$, as follows:

$$\dot{y_1} = p_1 = \phi\pi - (k\beta\Omega_3 y_3 + \mu)y_1 + \rho y_7, \qquad (3.5.1),$$

$$\dot{y_2} = p_2 = (1-\phi)\pi - (1-\epsilon)k\beta\Omega_3 y_3 y_2 - \mu y_2 \qquad (3.5.2),$$

$$\dot{y_3} = p_3 = k\beta\Omega_3 y_3 y_1 + k(1-\epsilon)\beta\Omega_3 y_3 y_2 - \Omega_1 y_3 \qquad (3.5.3),$$

$$\dot{y_4} = p_4 = \Omega_2 y_3 - \omega_5 y_4 \qquad (3.5.4),$$

with, $\lambda^{***} = \beta\Omega_3 y_3$.

The method entails evaluating the Jacobian of the system $[(3.5.1) - (3.5.4)]$ at the disease free equilibrium point, $E_*^0 = (S_*^0, V_*^0, A_*^0, R_*^0) = (\frac{\phi\pi}{\mu}, \frac{(1-\phi)\pi}{\mu}, 0,0)$, denoted by $J(E_*^0)$. This gives:

$$J(E_*^0). = \begin{pmatrix} -\mu & 0 & -k\beta^*\Omega_3 S^0 & \rho \\ 0 & -\mu & -k(1-\epsilon)\beta^*\Omega_3 V^0 & 0 \\ 0 & 0 & k\beta^*\Omega_3 H_2 - \Omega_1 & 0 \\ 0 & 0 & \Omega_2 & -\omega_5 \end{pmatrix},$$

Where,$H_2 = S^0 + (1-\epsilon)V^0$.

Consider the case, where $R_C^* = 1$. Suppose, further, that $\beta = \beta^*$is chosen as a bifurcation parameter. Solving forβ^* from $R_C^* = 1$ gives$\beta^* = \frac{\Omega_1}{k\Omega_3 H_2}$Using Mathematica software the

Jacobian of $\frac{dy}{dt} = F(y)$ at the disease free equilibrium point, with $\beta = \beta^*$, denoted by $J(E_*^0)$, has eigenvalues $(-\mu, -\mu, -\omega_5 \; and \; 0)$. We obtain one zero eigenvalue and three negative eigenvalues hence, the Centre Manifold theory can be used to analyze the dynamics of the model(*Junli Liu, et.al 2011*). The theorem stated below will be used to analyze the dynamics of the model (*Okaka. et.al, 2013*).

Theorem 3.

Castillo-Chavez and Song .Consider the following general system of ordinary differential equations with a parameter β^*

$$\frac{dy}{dt} = \mathrm{f}(\mathrm{y}, \beta^*), \mathrm{f}: \; R^n \times \mathrm{R} \; \rightarrow R^n \text{ and } \mathrm{f} \; \in C^2(R^n \times \mathrm{R}),$$

where 0 is an equilibrium point of the system (that is, $\mathrm{f}(\mathrm{y}, \beta^*) \equiv 0$ for all β^*) and

1.$\mathrm{A} \; = \; D_y \mathrm{f}(0,0) \; = \left(\frac{\delta p_i}{\delta y_j}(0,0)\right)$,is the linearization matrix of the system around the equilibrium 0 with β^* evaluated at 0;

2. Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts;

3. Matrix A has a right eigenvector u and a left eigenvector v corresponding to the zero eigenvalue.

Let $p_k$be the kth component of p and

$$a = \sum_{k,ij=1}^{n} v_k u_i u_j \frac{\partial^2 p_k}{\partial y_i \partial y_j}(0,0),$$

$$b = \sum_{k,ij=1}^{n} v_k u_i \frac{\partial^2 p_k}{\partial y_i \partial \beta^*}(0,0),$$

then the local dynamics of the system around the equilibrium point (0,0) is totally determined by the signs of a and b.

Particularly when:

i. $a > 0$ *and* $b > 0$, when $\beta^* < 0$ with $|\beta^*| \ll 1, (0,0)$, is locally asymptotically stable and there exists a positive unstable equilibrium; when $0 < \beta^* \ll 1, (0,0)$ is unstable and there exists a negative and locally asymptotically stable equilibrium.

ii. $a < 0$ *and* $b < 0$, when $\beta^* < 0$ with $|\beta^*| \ll 1, (0,0)$ is unstable; when $0 < \beta^* \ll 1, (0,0)$ is asymptotically stable and there exists a positive unstable equilibrium.

iii. $a < 0$ *and* $b > 0$, when $\beta^* < 0$ with $|\beta^*| \ll 1, (0,0)$is unstable, and there exists a negative and locally asymptotically stable equilibrium; when $0 < \beta^* \ll 1, (0,0)$ is stable and there exists a positive unstable equilibrium.

iv. $a > 0$ *and* $b < 0$, when β^*changes from negative to positive, $(0,0)$ changes its stability from stable to unstable. Correspondingly a negative equilibrium becomes positive and locally asymptotically stable.

If a > 0 and b > 0, then a backward bifurcation occurs at $\beta^* = 0$ (Okaka. et. al, 2013).

Eigenvectors ofJ_{β^*}: For the case when $R_C^* = 1$, it can be shown that the Jacobian$[J(E_*^0)]$at $\beta = \beta^*$(denoted by J_{β^*}) has a right eigenvector given by $u = [u_1, u_2, u_3, u_4]^T$, where,

$$u_1 = \frac{-k\beta\Omega_3 S^0 u_3 + \rho u_4}{\mu} = \frac{-k\omega_5\beta\Omega_3 S^0 u_3 + \rho\Omega_2 u_3}{\mu\omega_5} < 0,$$

$$u_2 = \frac{-k(1-\epsilon)\beta\Omega_3 V^0 u_3}{\mu} < 0,$$

$$u_3 = u_3 > 0,$$

$$u_4 = \frac{\Omega_2 u_3}{\omega_5} > 0.$$

Further, J_{β^*} has a left eigenvectors$(v = [v_1, v_2, v_3, v_4]^T)$, where,

$$v_1 = 0,$$

$\mathrm{v}_2 = 0,$

$\mathrm{v}_3 = \mathrm{v}_3 > 0,$

$\mathrm{v}_4 = 0.$

Since$(v_1 = v_2 = \mathrm{v}_4 = 0)$, we only need to compute the partial derivatives of p_3 (at the disease free equilibrium point). For the system$[(4.5.1) - (4.5.4)]$the associated non-zero partial derivative of f_3 (at the disease free equilibrium) is given by

$$\frac{\partial^2 p_3}{\partial y_1 \partial y_3} = \frac{\partial^2 p_3}{\partial p_3 \partial y_1} = k\beta^* \Omega_3, \qquad \frac{\partial^2 p_3}{\partial y_2 \partial y_3} = \frac{\partial^2 p_3}{\partial y_3 \partial y_2} = (1-\varepsilon)k\beta^* \Omega_3.$$

It implies,

$$\mathrm{a} = \mathrm{v}_3 \sum_{\mathrm{i,j}=1}^{4} \mathrm{u_i u_j} \frac{\partial^2 \mathrm{p_k}}{\partial \mathrm{x_i}\, \partial \mathrm{x_j}},$$

$$a = 2\mathrm{v}_3 \left\{ \mathrm{u}_1 \mathrm{u}_3 \frac{\partial^2 p_3}{\partial y_1 \partial y_3} + \mathrm{u}_2 \mathrm{u}_3 \frac{\partial^2 p_3}{\partial y_2 \partial y_3} \right\}.$$

Since u_1 *and* u_2 are less than zero, it follows that,

$$a = 2\mathrm{v}_3\{\mathrm{u}_1\mathrm{u}_3 k\beta^* \Omega_3 + \mathrm{u}_2\mathrm{u}_3(1-\varepsilon)k\beta^* \Omega_3\} < 0.$$

Also,

$$\frac{\partial^2 p_3}{\partial y_3 \partial \beta^*} = \mathrm{k}\beta \Omega_3 S^0 + \mathrm{k}(1-\epsilon)\beta \Omega_3 V^0,$$

$$b = v_3 \sum_{i=1}^{76} u_i \frac{\partial^2 p_k}{\partial y_i \partial \beta^*} + v_4 \sum_{i=1}^{7} u_i \frac{\partial^2 p_k}{\partial y_i \partial \beta^*},$$

$$b = v_3 \left\{ u_3 \frac{\partial^2 p_3}{\partial y_3 \partial \beta^*} \right\}.$$

Since v_3 and u_3 are greater than zero it follows that,

$$b = v_3 u_3 \{\mathrm{k}\beta \Omega_3 S^0 + \mathrm{k}(1-\epsilon)\beta \Omega_3 V^0\} > 0.$$

Hence, it follows (from Theorem 3 above) that when$\beta^* < 0$ with $|\beta^*| \ll 1, (0,0)$is unstable, and there exists a negative and locally asymptotically stable equilibrium; when $0 < \beta^* \ll 1, (0,0)$ is stable and there exists a positive unstable equilibrium.

3.6. Local Stability of the disease free equilibrium point (DFE).

To determine the local stability of the disease free equilibrium point we state and prove the following theorem.

Theorem 4.

The DFE of the system $[(3.4.5) - (3.4.8)]$*is locally asymptotically stable* $\mathrm{R}_{\mathrm{C}}^* < 1$ *and unstable otherwise.*

Proof

To establish the local stability of the system$[(3.4.5) - (3.4.8)]$, we use the Jacobian of the model evaluated atE_*^0. Stability of this steady state is then determined based on the eigenvalues of the corresponding Jacobianwhich are functions of the model parameters. We let

$P_1 = \phi\pi - (\mathrm{k}\lambda + \mu)S \quad + \rho R,$

$P_2 = (1 - \phi)\pi - (1 - \epsilon)\mathrm{k}\lambda V - \mu \mathrm{V},$

$P_3 = \mathrm{k}\lambda \mathrm{S} + \mathrm{k}(1 - \epsilon)\lambda \mathrm{V} \quad - \Omega_1 \mathrm{A},$

$P_4 = \Omega_2 A - \omega_5 R.$

The Jacobian matrix evaluated at disease free equilibrium point E_*^0 is obtained as

$$J(E^0) = \begin{pmatrix} -\mu & 0 & -\mathrm{k}\beta\Omega_3 \mathrm{S}^0 & \rho \\ 0 & -\mu & -\mathrm{k}(1-\epsilon)\beta\Omega_3 \mathrm{V}^0 & 0 \\ 0 & 0 & \mathrm{k}\beta\Omega_3 H_2 \quad - \Omega_1 & 0 \\ 0 & 0 & \Omega_2 & -\omega_5 \end{pmatrix},$$

Where,

$$H_2 = \mathrm{S}^0 + (1-\epsilon)\mathrm{V}^0$$

Solving the equation

$$|J(E^0) - q(i)H| = 0,$$

where H is the identity matrix and , $i = 1(1)4$ are eigenvalues Using the Mathematica software we obtain the following eigenvalues

$q(1) = q(2) = -\mu,$

$q(3) = -\omega_5,$

$\mathrm{q}(4) = -\Omega_1 + \mathrm{k}\beta\Omega_3 H_2.$

Clearly three eigenvalues are negative but the conditions necessary and sufficient for $\mathrm{q}(4)$ is

$$-\Omega_1 + \mathrm{k}\beta\Omega_3 H_2 < 0,$$

$$\mathrm{k}\beta\Omega_3 H_2 < \Omega_1,$$

$$\frac{\mathrm{k}\beta\Omega_3 H_2}{\Omega_1} < 1,$$

$$R_C^* < 1.$$

This completes the proof.

3.7. Global stability of the disease free point.

To prove the global stability we state and prove the following theorem.

Theorem 5

The DFE is globally stable whenever $\mathrm{R}_\mathrm{C}^* < 1$ *unstable otherwise.*

Proof

We propose the following Lyapunov function for the system $[(3.4.5) - (3.4.8)]$

$$\mathrm{L}(\mathrm{S},\mathrm{V},\mathrm{A},R) = S - S^0 - S^0 Ln\frac{\mathrm{S}}{S^0} + X_1\left(V - V^0 - V^0 Ln\frac{\mathrm{V}}{V^0}\right) + X_2\mathrm{A} + X_3 R$$

$\mathrm{L}(\mathrm{S},\mathrm{V},\mathrm{A},R)$is positive definite satisfies the conditions;

$$L(S^0,V^0,A^0,R^0)=0\ and\ \mathrm{L}(\mathrm{S},\mathrm{V},\mathrm{A},R)>\ 0.$$

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

$$\frac{\mathrm{dL}(S^0,V^0,A^0,R^0)}{\mathrm{dt}}=0\text{ and }\frac{\mathrm{dL}(\mathrm{S},\mathrm{V},\mathrm{A},R)}{\mathrm{dt}}<0.$$

where$X_1,X_2 and X_3$ are positive constants to be determined.At DFE point$\mathrm{E}_*^0=$

$(S^0,V^0,A^0,R^0)$the system$[(3.4.5)-(3.4.8)]$ satisfies,

$$\phi\pi=\mu\mathrm{S}^0,$$

$$(1-\phi)\pi=\mu\mathrm{V}^0.$$

The time derivative of the lyapunov function is obtained as,

$$\frac{\mathrm{dL}(\mathrm{S},\mathrm{V},\mathrm{A},R)}{\mathrm{dt}}=\left(1-\frac{\mathrm{S}^0}{\mathrm{S}}\right)\frac{\mathrm{dS}}{\mathrm{dt}}+X_1\left(1-\frac{\mathrm{V}^0}{\mathrm{V}}\right)\frac{\mathrm{dV}}{\mathrm{dt}}+X_2\frac{\mathrm{dA}}{\mathrm{dt}}+X_3\frac{\mathrm{dR}}{\mathrm{dt}},$$

Substituting $\frac{\mathrm{dS}}{\mathrm{dt}},\frac{\mathrm{dV}}{\mathrm{dt}},\ \frac{\mathrm{dA}}{\mathrm{dt}}$ and $\frac{\mathrm{dR}}{\mathrm{dt}}$ to obtain;

$$\frac{\mathrm{dL}(\mathrm{S},\mathrm{V},\mathrm{A},R)}{\mathrm{dt}}=\left(1-\frac{\mathrm{S}^0}{\mathrm{S}}\right)\{\phi\pi-(\mathrm{k}\lambda+\mu)S\ \ +\rho R\ \}$$

$$+X_1\left(1-\frac{\mathrm{V}^0}{\mathrm{V}}\right)\{(1-\phi)\pi-k(1-\epsilon)\lambda V-\mu\mathrm{V}\}$$

$$+X_2\{\mathrm{k}\lambda\mathrm{S}+\mathrm{k}(1-\epsilon)\lambda\mathrm{V}\ \ -\Omega_1\mathrm{A}\}+X_3\{\Omega_2A-\omega_5R\}.$$

Substituting $\phi\pi$ and $(1-\phi)\pi$ to obtain;

$$\frac{dL(S,V,A,R)}{dt} = \left(1 - \frac{S^0}{S}\right)\{\mu S^0 - (k\beta\Omega_3 A + \mu)S \ + \rho R\ \}$$

$$+ X_1\left(1 - \frac{V^0}{V}\right)\{\mu V^0 - k(1-\epsilon)\beta\Omega_3 AV - \mu V\}$$

$$+ X_2\{k\Omega_3 AS + k(1-\epsilon)\Omega_3 AV \ - \Omega_1 A\} + X_3\{\Omega_2 A - \omega_5 R\},$$

$$\frac{dL(S,V,A,R)}{dt} = -\mu\frac{(S-S^0)^2}{S} - \mu\frac{(V-V^0)^2}{V}$$

$$+ \{-X_2\Omega_1 + X_3\Omega_2 + k\beta\Omega_3 S^0 + X_1 k\beta(1-\epsilon)\Omega_3 V^0\}A \ + \{X_2 - 1\}k\beta\Omega_3 AS$$

$$+ \{X_2 - X_1\}k\beta(1-\epsilon)\Omega_3 AV + \left\{\rho - X_3\omega_5 - \rho\frac{S^0}{S}\right\}R.$$

Setting AS, AV and $A$to zero we obtain the following equation,

$$X_2 - 1 = 0,$$

$$X_2 - X_1 = 0,$$

$$-X_2\Omega_1 + X_3\Omega_2 + k\beta\Omega_3 S^0 + X_1 k\beta(1-\epsilon)\Omega_3 V^0 = 0.$$

Solving the above equation to obtain;

$$X_1 = X_2 = 1,$$

$$X_3 = \frac{\Omega_1}{\Omega_2} - \frac{k\beta\Omega_3 H_2}{\Omega_2} = \frac{\Omega_1}{\Omega_2}(1 - R_C^*),$$

Where,

$$H_2 = S^0 + (1-\epsilon)V^0.$$

The derivative of lyapunov reduces to;

$$\frac{dL(S,V,A,R)}{dt} = -\mu\frac{(S-S^0)^2}{S} - \mu\frac{(V-V^0)^2}{V} + \rho\left(1 - \frac{S^0}{S}\right)R - \frac{\Omega_1}{\Omega_2}(1 - R_C^*)\omega_5 R$$

Since $\left(1-\frac{S^0}{S}\right) \leq 0$, the conditions necessary and sufficient for $\frac{dL(S,V,A)}{dt} < 0$ is $(1-R_C^*) > 0$.

This implies that disease free equilibrium point is globally stable if and only if $R_C^* < 1$ and unstable otherwise. This completes the proof.

3.8. Local Stability and global stability of the Endemic Equilibrium point (EEP).

At endemic equilibrium point (EEP), the global stability implies the local stability. To determine local and global stability we state and prove the following theorem.

Theorem 6

The DFE is globally whenever $R_C^{**} < \frac{H_2(V^{***}-V)^2}{S^{***}(V^{***2}+V^2)}$ *unstable otherwise.*

Proof

For the system $[(3.4.5)-(3.4.8)]$to be tractable mathematically consider a special case where the wanning due to drugs is zero i.e $\rho = 0$ to obtain (Junli Liu andTailei Zhang, 2011),

$$\frac{dS}{dt} = \phi\pi - (k\lambda + \mu)S \qquad (3.8.1),$$

$$\frac{dV}{dt} = (1-\phi)\pi - k(1-\epsilon)\lambda V - \mu V \qquad (3.8.2),$$

$$\frac{dA}{dt} = k\lambda S + k(1-\epsilon)\lambda V - \Omega_1 A \qquad (3.8.3),$$

$$\frac{dR}{dt} = \Omega_2 A - \mu R \qquad (3.8.4).$$

The controlled reproduction number(R_C^{**}), the force of infection(λ^{***}), disease free equilibrium point$E^{00} = (S^{00}, V^{00}, A^{00}, R^{00}) = (\frac{\phi\pi}{\mu}, \frac{(1-\phi)\pi}{\mu}, 0,0)$and endemic equilibrium point $E^{***} = (S^{***}, V^{***}, A^{***}, R^{***})$of the system $[(3.8.1)-(3.8.4)]$ is given by

$$R_C^{**} = \frac{\beta k \Omega_3 H_2}{\Omega_1}, \lambda^{***} = \beta \Omega_3 A^{***} \; and$$

$$S^{***} = \frac{\phi\pi}{k\lambda^{***} + \mu},$$

$$V^{***} = \frac{(1-\phi)\pi}{k\lambda^{***}(1-\epsilon) + \mu},$$

$$A^{***} = \frac{\frac{\pi(-1+\epsilon)\lambda^{***}(-1+\phi)}{k(-1+\epsilon)\lambda^{***}-\mu} - \frac{k\pi\lambda^{***}\phi}{k\lambda^{***}+\mu}}{-\Omega_1},$$

$$R^{***} = \frac{\Omega_2 A^{***}}{\mu}.$$

where, $H_{22} = S^{00} + (1-\epsilon)V^{00}$.

We propose the following Lyapunov function,

$$K(S,V,A,R) = S - S^{***} - S^{***} Ln\frac{S}{S^*} + Y_1\left(V - V^{***} - V^{***} Ln\frac{V}{V^{**}}\right) + Y_2\left(A - A^* - A^* Ln\frac{A}{A^*}\right)$$
$$+ Y_3\left(R - R^{***} - R^* Ln\frac{R}{R^{***}}\right),$$

where$Y_1, Y_2 \; and \; Y_3$ are positive constants to be determined. The lyapunov function $K(S,V,A,R)$ satisfies the conditions, $K(S^{***}, V^{***}, A^{***}, R^{***}) = 0 \; and \; K(S,V,A,R) > 0$, henceit is positive definite. For $\frac{dK(S,V,A,R)}{dt}$ to be negative definite, it must satisfies,

$$\frac{dK(S^*, V^*, A^*, R^*)}{dt} = 0 \quad \text{and} \quad \frac{dK(S,V,A,R)}{dt} < 0.$$

The endemic equilibrium point $E_{**}^* = (S^{***}, V^*, A^*, R^*)$ for the system satisfies,

$$\phi\pi = (k\beta\Omega_3 A^{***} + \mu)S^{***},$$

$(1-\phi)\pi = k(1-\epsilon)\beta\Omega_3 A^{***}V^{*} + \mu V^{***},$

$\Omega_2 A^{***} = \mu R^{***},$

$k\beta\Omega_3 A^{***}(S^{***} + (1-\epsilon)V^{***}) = \Omega_1 A^{***}.$

Determining the time derivative of the lyapunov equation we obtain,

$$\frac{dK(S,V,A,R)}{dt} = \left(1-\frac{S^{***}}{S}\right)\frac{dS}{dt} + X_1\left(1-\frac{V^{***}}{V}\right)\frac{dV}{dt} + X_2\left(1-\frac{A^{***}}{A}\right)\frac{dA}{dt} + X_3\left(1-\frac{R^{***}}{R}\right)\frac{dR}{dt},$$

Substituting for $\frac{dS}{dt}, \frac{dV}{dt}, \frac{dA}{dt}$ and $\frac{dR}{dt}$.

$$\frac{dK(S,V,A,R)}{dt} = \left(1-\frac{S^{***}}{S}\right)\{\phi\pi - (k\beta\Omega_3 A + \mu)S\}$$

$$+ X_1\left(1-\frac{V^{***}}{V}\right)\{(1-\phi)\pi - k(1-\epsilon)\beta\Omega_3 AV - \mu V\}$$

$$+ X_2\left(1-\frac{A^{***}}{A}\right)\{k\beta\Omega_3 AS + k(1-\epsilon)\beta\Omega_3 AV - \Omega_1 A\}$$

$$+ X_3\left(1-\frac{R^{***}}{R}\right)\{\Omega_2 A - \mu R\},$$

$$\frac{dK(S,V,A,R)}{dt} = \left(1-\frac{S^{*}}{S}\right)\{(k\beta\Omega_3 A^{***} + \mu)S^{***} - (k\beta\Omega_3 A + \mu)S\}$$

$$+ X_1\left(1-\frac{V^{***}}{V}\right)\{k(1-\epsilon)\beta\Omega_3 A^{***}V^{***} + \mu V^{***} - k(1-\epsilon)\beta\Omega_3 AV - \mu V\}$$

$$+ X_2\left(1-\frac{A^{***}}{A}\right)\{k\beta\Omega_3 AS + k(1-\epsilon)\beta\Omega_3 AV - \Omega_1 A\}$$

$$+ X_3\left(1-\frac{R^{***}}{R}\right)\{\Omega_2 A - \mu R\},$$

$$\frac{\mathrm{dK(S,V,A},R)}{\mathrm{dt}} = -\mu\frac{(\mathrm{S}-S^{***})^2}{\mathrm{S}} - \mu\frac{(\mathrm{V}-V^{***})^2}{\mathrm{V}}$$

$$+\{X_3\Omega_2 - X_1\Omega_1 + X_1k(1-\epsilon)\beta\Omega_3V^* + \mathrm{k}\beta\Omega_3S^{***}\}\mathrm{A} \quad + \{X_2-1\}\mathrm{k}\beta\Omega_3\mathrm{AS}$$

$$+\{X_2-X_1\}\mathrm{k}\beta(1-\epsilon)\Omega_3\mathrm{AV} + -X_3\mu R + \left(-\frac{S^{***}}{\mathrm{S}}\right)\{\mathrm{k}\beta\Omega_3A^{***}S^{***}\}$$

$$+X_1\left(-\frac{V^{***}}{\mathrm{V}}\right)\{k(1-\epsilon)\beta\Omega_3A^{***}V^{***}\}$$

$$+X_2\left(-\frac{A^{***}}{\mathrm{A}}\right)\{\mathrm{k}\beta\Omega_3A\mathrm{S} + \mathrm{k}(1-\epsilon)\beta\Omega_3A\mathrm{V} \quad -\Omega_1\mathrm{A}\}$$

$$+X_3\left(-\frac{R^{***}}{\mathrm{R}}\right)\{\Omega_2A - X_3\mu R\} + \mathrm{k}\beta\Omega_3A^*S^* \quad + X_1\{k(1-\epsilon)\beta\Omega_3A^{***}V^{***}\}.$$

Setting $\mathrm{AS}, \mathrm{A}V$ and $A$to zero we obtain the following equation,

$$X_2 - 1 = 0,$$

$$X_2 - X_1 = 0,$$

$$X_3\Omega_2 - X_1\Omega_1 + X_1k(1-\epsilon)\beta\Omega_3V^{***} + \mathrm{k}\beta\Omega_3S^{***} = 0.$$

Solving the above equations to obtain,

$$X_1 = X_2 = 1,$$

$$X_3 = 0.$$

Where,$H_2 = S^{00} + (1-\epsilon)V^{00}$.

$$\frac{\mathrm{dK(S,V,A},R)}{\mathrm{dt}} = -\mu\frac{(\mathrm{S}-\mathrm{S}^{***})^2}{\mathrm{S}} - \mu\frac{(\mathrm{V}-\mathrm{V}^{***})^2}{\mathrm{V}} + \mathrm{k}\beta\Omega_3\mathrm{A}^{***}\mathrm{S}^{***}\left(2 - \frac{S}{\mathrm{S}^{***}} - \frac{\mathrm{S}^{***}}{\mathrm{S}}\right)$$

$$+\mathrm{k}(1-\epsilon)\beta\Omega_3\mathrm{A}^{***}\mathrm{V}^{***}\left(2 - \frac{\mathrm{V}^{***}}{\mathrm{V}} - \frac{V}{\mathrm{V}^{***}}\right),$$

$$\frac{dK(S,V,A,R)}{dt} = -\mu\frac{(S-S^{***})^2}{S} - \mu\frac{(V-V^{***})^2}{V} + k\beta\Omega_3 A^{***}S^{***}\left(2-\frac{S}{S^{***}}-\frac{S^{***}}{S}\right)$$

$$+\Omega_1 A^{***}\left(2-\frac{V^{***}}{V}-\frac{V}{V^{***}}\right) - k\beta\Omega_3 A^{***}S^{***}\left(2-\frac{V^{***}}{V}-\frac{V}{V^{***}}\right),$$

$$\frac{dK(S,V,A,R)}{dt} = -\mu\frac{(S-S^{***})^2}{S} - \mu\frac{(V-V^{***})^2}{V} + k\beta\Omega_3 A^{***}S^{***}\left(-\frac{S}{S^{***}}-\frac{S^{***}}{S}+\frac{V^{***}}{V}+\frac{V}{V^{***}}\right)$$

$$+\Omega_1 A^{***}\left(2-\frac{V^{***}}{V}-\frac{V}{V^{***}}\right),$$

$$\frac{dK(S,V,A,R)}{dt} = -\mu\frac{(S-S^{***})^2}{S} - \mu\frac{(V-V^{***})^2}{V} - \Omega_1 A^{***}S^{***}\frac{R_C^{**}}{H_2}\left(\frac{S}{S^{***}}+\frac{S^{***}}{S}\right)$$

$$+\Omega_1 A^{***}\left\{S^{***}\frac{R_C^{**}}{H_2}\left(\frac{V^{***}}{V}+\frac{V}{V^{***}}\right)+\left(2-\frac{V^{***}}{V}-\frac{V}{V^{***}}\right)\right\}.$$

Since all the other terms of $\frac{dL(S,V,A,R)}{dt}$ are less than zero, the condition necessary and sufficient for$\frac{dL(S,V,A,R)}{dt} < 0$ is given by,

$$S^{***}\frac{R_C^{**}}{H_2}\left(\frac{V^{***}}{V}+\frac{V}{V^{***}}\right)+\left(2-\frac{V^{***}}{V}-\frac{V}{V^{***}}\right) < 0,$$

$$S^{***}\frac{R_C^{**}}{H_2}\left(V^{***2}+V^2\right) < V^{***2}+V^2-2VV^{***},$$

$$R_C^{**} < \frac{H_2(V^{***}-V)^2}{S^{***}\left(V^{***2}+V^2\right)}.$$

Then $\frac{dV}{dt}$= 0 holds only when($S = S^{***}, V = V^{***}, A = A^{***}$ and $R = R^{***}$): So the maximal compact invariant set in $\{(S;\ E;\ I) \in \Pi: \frac{dV}{dt} = 0\}$ is the singleton $\{E_*^{**}\}$ using Lasalle's invariance principle $\frac{dL(S,I,A,R)}{dt} < 0$,Iff

$$R_C^{**} < \frac{H_2(V^{***}-V)^2}{S^{***}\left(V^{***2}+V^2\right)}.$$

4. Analytical results of the model.

We shall determine epidemiological thresholds and carryout sensitivity analysis of the control reproduction number(R_C) using partial derivatives.

4.1 Epidemiological thresholds.

We shall determine treatment thresholds, herd immunity and impact of treatment using control reproduction number and basic reproduction number.

4.1.1. Treatment thresholds.

We shall determine treatments for mildly infected($\mathrm{I_M}$) and chronically infected($\mathrm{I_C}$) using the control reproduction number.

4.1.1.1 Mildly treatment threshold($\tau_1{}^C$).

According to (Ngari, et.al, 2014), the mildly treatment threshold is determined when $\mathrm{R_C}$ is equated to one and solving for $\tau_1{}^C$(critical treatment for mildly infected children(I_M) to obtain,

$$\tau_1{}^C = \frac{\omega_1\omega_3}{\beta k\xi_2 R_1} - \{R_1\omega_2\omega_3\omega_4 + \xi_1(R_1\theta_1\omega_3\omega_4 + R_2\omega_1\omega_3\omega_4) + \xi_2(R_1\theta_1\theta_2\tau_2 + R_2\theta_2\tau_2\omega_1) + \xi_3(R_1\theta_1\tau_2\omega_3 + R_2\tau_2\omega_1\omega_3)\}.$$

4.1.1.2 Chronically treatment threshold($\tau_1{}^C$).

The chronically treatment threshold is determined when $\mathrm{R_C}$ is equated to one and solving for $\tau_2{}^C$(critical treatment for chronically infected children(I_C) to obtain,

$$\tau_2{}^C = \{\xi_2(R_1\theta_1\theta_2 + R_2\theta_2\omega_1) + \xi_3(R_1\theta_1\omega_3 + R_2\omega_1\omega_3)\}\langle\frac{\omega_1\omega_2\omega_3\omega_4}{\beta k} - \{R_1\omega_2\omega_3\omega_4 + \xi_1(R_1\theta_1\omega_3\omega_4 + R_2\omega_1\omega_3\omega_4) + \xi_2(R_1\tau_1\omega_2\omega_4)\}\rangle.$$

4.1.1.3 Measure of treatment impact(U).

(A. M. Clean and S. Blower, 1993), defined measure of treatment impact based on the reproduction numbers can be defined as,

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

$$(U) = 1 + ((\mu + \delta_1)(\mu + \theta_1)\Big((1 - P)S^0(\mu + \theta_1 + \tau_1)\left(\theta_1\xi_2\tau_2 + \omega_3(\xi_3\tau_2 + \xi_1\omega_4)\right)$$

$$+ (PS^0 + (1 - \epsilon)V^0)\Big((\mu + \delta_1 + \tau_2)(\xi_2\tau_1 + \omega_3)\omega_4$$

$$+ \theta_1\left(\theta_2\xi_2\tau_2 + \omega_3(\xi_3\tau_2 + \xi_1\omega_4)\right)\Big)\Big))$$

$$/(S^0(-P(\mu + \delta_1) + ((-1 + P)\mu - \theta_1)\xi_1)(\mu + \theta_1 + \tau_1)(\mu + \delta_1 + \tau_2)\omega_3\omega_4.$$

4.1.2 Herd immunity.

The herd immunity threshold is determined by,

$q_c = 1 - \frac{1}{R_0}$, where (q_c) is the critical vaccination threshold (Mary Holland and Chase E. Zachary, 2014).

Substituting for basic reproduction number(R_0), we obtain,

$$q_c = 1 - \frac{(\mu + \delta_1)(\mu + \theta_1)}{\beta k S^0(P(\mu + \delta_1) + (\mu(1 - P) + \theta_1)\xi_1)}.$$

4.2 Sensitivity analysis of the effective control number(R_C).

Impact of intervention strategies are vital in lowering burden of pneumonia It is important to investigate the sensitivity of R_C to: the rate at which mild and chronic infected children seek

treatment($\tau_1 and\ \tau_2$), with respect to vaccination drug efficacy ε and effects of environmental factors (k). Determining partial derivatives of R_C with respect to;

i. effect of environment (k) .

$$\frac{dR_C}{dk} = \frac{\beta}{\omega_1\omega_2\omega_3\omega_4}\{R_1\omega_2\omega_3\omega_4 + \xi_1(R_1\theta_1\omega_3\omega_4 + R_2\omega_1\omega_3\omega_4)$$

$$+ \xi_2(R_1\theta_1\theta_2\tau_2 + R_2\theta_2\tau_2\omega_1 + R_1\tau_1\omega_2\omega_4) + \xi_3(R_1\theta_1\tau_2\omega_3 + R_2\tau_2\omega_1\omega_3)\} > 0,$$

ii. rate at which mild infected children seek treatmentτ_1..

$$\frac{dR_C}{d\tau_1} = -\frac{\beta k}{\omega_1{}^2\omega_2\omega_3\omega_4}\{R_1\omega_2\omega_3\omega_4 + \xi_1(R_1\theta_1\omega_3\omega_4 + R_2\omega_1\omega_3\omega_4)$$

$$+ \xi_2(R_1\theta_1\theta_2\tau_2 + R_2\theta_2\tau_2\omega_1 + R_1\tau_1\omega_2\omega_4) + \xi_3(R_1\theta_1\tau_2\omega_3 + R_2\tau_2\omega_1\omega_3)\}$$

$$+ \frac{\beta k}{\omega_1\omega_2\omega_3\omega_4}\{\xi_1 R_2\omega_3\omega_4 + \xi_2(R_2\theta_2\tau_2 + R_1\omega_2\omega_4) + \xi_3 R_2\tau_2\omega_3\} < 0,$$

iii. rate at which mild infected children seek treatment τ_2.

$$\frac{dR_C}{d\tau_2} = -\frac{\beta k}{\omega_1\omega_2{}^2\omega_3\omega_4}\{R_1\omega_2\omega_3\omega_4 + \xi_1(R_1\theta_1\omega_3\omega_4 + R_2\omega_1\omega_3\omega_4)$$

$$+ \xi_2(R_1\theta_1\theta_2\tau_2 + R_2\theta_2\tau_2\omega_1 + R_1\tau_1\omega_2\omega_4) + \xi_3(R_1\theta_1\tau_2\omega_3 + R_2\tau_2\omega_1\omega_3)\}$$

$$+ \frac{\beta k}{\omega_1\omega_2\omega_3\omega_4}\{R_1\omega_3\omega_4 + \xi_2(R_1\theta_1\theta_2 + R_2\theta_2\omega_1 + R_1\tau_1\omega_4)$$

$$+ \xi_3(R_1\theta_1\omega_3 + R_2\omega_1\omega_3)\} < 0.$$

iv. With respect to vaccinated (V) drug efficacy(ε).

$$\frac{dR_C}{d\,\varepsilon} = -\frac{\beta k V^0}{\omega_1\omega_2\omega_3\omega_4}\{\omega_2\omega_3\omega_4 + \xi_1\theta_1\omega_3\omega_4 + \xi_2(\theta_1\theta_2\tau_2 + \tau_1\omega_2\omega_4) + \xi_3\theta_1\tau_2\omega_3\} < 0$$

5. Biological interpretation of the analytical results

We shall interpret the stabilities of equilibrium point, determine thresholds and determine partial derivate of control reproduction number(R_C).

5.1 Local and global stability of equilibrium points.

An equilibrium point is said to be locally asymptotically stable if all points in the neighborhood of the equilibrium point move towards it over time. An equilibrium point is globally asymptotically stable if all points move towards it over time. Disease free point of pneumonia model was locally and globally asymptotically stable when the control reproduction number(R_C^*) is maintained less than one; this means interventions should maintain control reproduction number less than one in order to avoid pneumonia persistence. The system did not exhibit backward bifurcation hence it is feasible to control pneumonia in Kenya. Interpretation of local and global stability of endemic equilibrium point will be part of future research.

5.2 Epidemiological thresholds.

The number of children under five years who can be infected with pneumonia by one infectious child when interventions such as vaccination, proper control of environmental factors and treatment are observed is referred to as control reproduction number($\mathrm{R_C}$) while the number of children under five years who can be infected with pneumonia by one infectious child without interventions referred to as control reproduction number($\mathrm{R_C}$).The control reproduction number ($\mathrm{R_C}$) and basic reproduction number ($\mathrm{R_0}$)of the system [(3.3.1) − (3.3.7)]are given by;

$$\mathrm{R_C} = \frac{\beta k}{\omega_1\omega_2\omega_3\omega_4}\{R_1\omega_2\omega_3\omega_4 + \xi_1(R_1\theta_1\omega_3\omega_4 + R_2\omega_1\omega_3\omega_4)$$
$$+ \xi_2(R_1\theta_1\theta_2\tau_2 + R_2\theta_2\tau_2\omega_1 + R_1\tau_1\omega_2\omega_4) + \xi_3(R_1\theta_1\tau_2\omega_3 + R_2\tau_2\omega_1\omega_3)\},$$

$$R_0 = \frac{\beta k S^0 (P(\mu + \delta_1) + (\mu(1 - P) + \theta_1)\xi_1)}{(\mu + \delta_1)(\mu + \theta_1)}.$$

5.2.1. Treatment thresholds.

When actual treatments ($\tau_1 and\ \tau_2$) are greater than critical treatment${\tau_1}^C\ and{\tau_2}^C$ respectively it can ensure total eradication of pneumonia. Also, treatment with sufficient coverage can succeed in eliminating infection when R_C is below unity. Because R_c measures the intensity of the epidemic, treatment, by lowering R_C, can have significant public health impact even if it fails to eliminate infection in a specific population. When,

$$\begin{aligned}(U) = 1 + ((\mu + \delta_1)(\mu + \theta_1)\Big((1 - P)S^0(\mu + \theta_1 + \tau_1)\left(\theta_1\xi_2\tau_2 + \omega_3\left(\xi_3\tau_2 + \xi_1\omega_4\right)\right) \\ + (PS^0 + (1 - \epsilon)V^0)\Big((\mu + \delta_1 + \tau_2)\left(\xi_2\tau_1 + \omega_3\right)\omega_4 \\ + \theta_1\left(\theta_2\xi_2\tau_2 + \omega_3\left(\xi_3\tau_2 + \xi_1\omega_4\right)\right)\Big)\Big)) \\ /(S^0\left(-P(\mu + \delta_1) + \left((-1 + P)\mu - \theta_1\right)\xi_1\right)(\mu + \theta_1 + \tau_1)(\mu + \delta_1 + \tau_2)\omega_3\omega_4) \\ > 0.\end{aligned}$$

Thus, population-level impact of treatment is always positive provided. This condition is likely to be satisfied for treatment with effective drugs.

***5.2.2* Herd immunity.**

Vaccination is a voluntary process and it is not possible to vaccinate all individuals in the population. When actual vaccination $(I - \Omega)\pi$ is greater than critical treatment (q_c) it can ensure total eradication of pneumonia.

5.3 Sensitivity analysis of the effective control number(R_C).

Clearly,R_C was directly proportional to k but inversely proportional to; ε, τ_1 and τ_2. Higher vaccination efficacy (ε) and higher rates rate of mildly and chronically infected children seeking treatment(τ_1 andτ_2) would decrease the control reproduction number and the intensity of the pneumonia endemic. Lower effect environmental factors (k) would decrease the control reproduction number (R_C)

6. Discussion and conclusion.

Most of the developed mathematical models concentrate on bacterial pneumonia, antibiotic resistance, and vaccinations. These models assume that pneumonia is isolated in population, during treatment and in death. Further, viral, fungi and parasitic pneumonia are mostly ignored.

Lipsitch, et.al, 2009, considered an in host model concentrating on bacteria pneumonia (streptococcus pneumonia). They considered issue of coexistence of pneumonia serotypes in a population. The study model assumes isolation is carried out in hospitals. The findings stressed correctly modeling the possibility of a host being able to become simultaneously invaded with more than one strain, taking into account difficulties in obtaining a second strain if already colonized and considering acquired immunity of new strains. Our research study developed general model pneumonia as is affect population of the under five years.

Huang et al, 2005, considered a pneumococcal transmission in host model which takes into account the risk of higher rates of transmission for children who attend child-care centers or who are often forced to spend time with children who attend these centers. The results stress the importance of child-care centers in transmission. The study model assumes isolation is carried out in hospitals and closed community. Our research study developed a general model of open community with births and deaths.

Otieno, et.al, 2012, formulated four compartmental classes in their model involving Susceptible, Carriers, Infected and Recovered. They assumed pneumonia is isolated in health facilities and studied bacterial pneumonia. The findings stressed importance of treatment and quarantine where possible. Our research study developed a general model and assumed that under five years cannot have carriers due to the weak nature of their immune systems.

Ngari, et.al, 2014, formulated three compartmental classes involving Susceptible, Infected and treated in their model. The study did not consider vaccinated class. Their findings stressed importance of natural immunity and treatment in lowering burden of pneumonia. Our research study included vaccinated class and subdivided infected and treated classes into mild and chronic. Furthermore recovered class was also introduced.

Joseph, et.al, 2012, formulated four compartmental classes involving Vaccinated, Susceptible, Infected and Treated in their model. They assumed treated class to be non-infective and the adults and children have same infection rates. The results stressed importance of drug efficacy in lowering burden of pneumonia. Our research study assumed treated classes to be infectious and also subdivided infected and treated classes into mild and chronic. Furthermore recovered class was also introduced.

G. O. Lawi, et.al , 2013, developed a deterministic co-infection model of malaria and pneumonia under five years of age. The study analyzed the reproduction number by partial derivatives.The result stressed the importance of increase in treatment rates to lower new disease incidences.

Laura L. Hammitt, et al, 2012, conducted a case-control study of pneumonia etiology among children aged 1–59 months in rural Kenya. They classified pneumonia in two categories (severe and very severe pneumonia).The result obtained indicated that very severe pneumonia cases

constituted twenty nine percent of the 810 case patients. Our research study used mathematic model approach to describe dynamics of pneumona.

The research study emphasized on the importance of treatment in lowering the burden of pneumonia, this was in agreement with research findings of (Joseph, et.al, 2012), (Ngari, et.al 2014), (Otieno, et.al, 2012) and (G. O. Lawi, et.al , 2013). To ensure eradication of pneumonia this research study detemined the minimum critical treatment thresholds which was not taken into account in previous pneumonia studies. In concurrent with (Laura L. Hammitt, et al, 2012) this research study also classified pneumonia in to broad categories but used mathematical approach.

Although (Joseph, et.al, 2012) also studied effect vaccination, their paper did not determine the herd immunity and also failed to consider the effects enviromental factors and the contribution of the treated classes to the dynamics of childhood pneumonia and, these have been taken into consideration in this paper.

7. Recommendations.

In order to reduce the burden of childhood pneumonia, it is suggested that the Government of Kenya should invest in;

- creating public awareness to parents on; symptoms of pneumonia,
- environmental factors which increase children susceptibility to pneumonia(like indoor air pollution, living in crowded homes and parental smoking),
- Creating awareness on the need to visit hospitals for treatment,
- Improving vaccination drug's efficacy,
- Achieving herd immunity.

The study analyzed a reduced system of equations $[(3.4.5) - (3.4.8)]$and$[(3.8.1) - (3.8.4)]$ instead of $[(2.4.1) - (2.4.7)]$Model analysis of full system $[(2.4.1) - (2.4.7)]$ can be part future research.

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Appendix

Table1: The model variables and parameters.

Variable	Description
N(t)	Total population of less or equal to five year's Children in Kenya
S(t)	Population of less or equal to five year's children susceptible to pneumonia in Kenya.
V(t)	Population of less or equal to five year's children vaccinated in Kenya.
$I_M(t)$	Population of less or equal to five year's children mildly infected with pneumonia in Kenya.
$I_C(t)$	Population of less or equal to five year's children chronically infected with pneumonia in Kenya.
$T_M(t)$	Population of less or equal to five year's children mildly treated with pneumonia in Kenya (outpatient).
$T_C(t)$	Population of less or equal to five year's children chronically treated with pneumonia children in Kenya (inpatient).
R(t)	Population of less or equal to five year's children recovered from pneumonia in Kenya.
Parameters	**Description**
β	Pneumonia infection rate of less or equal to five year's children in Kenya
π	Recruitment rate of less or equal to five year's children in Kenya (birth rate).
γ_1	Recovery rate of less or equal to five year's mildly treated children due to treatment in Kenya.
γ_2	Recovery rate of less or equal to five year's chronically treated children due to treatment in Kenya.
δ_1	Pneumonia induced death due to less or equal to five year's chronically infected children in Kenya.
δ_2	Pneumonia induced death due to less or equal to five year's chronically treated children in Kenya.
μ	Constant natural death rate in Kenya.
ε	Percentage of pneumonia vaccination drug's efficacy administered to less or equal to five year's children in Kenya.
ρ	Waning rate of treatment drug after recovery of less or equal to five year's children in Kenya.
θ_1	Rate at which less or equal to five year's mildly infected children progresses to chronic infected class in Kenya.
θ_2	Rate at which less or equal to five year's chronically treated children (inpatient) are discharged as mildly treated class (outpatient) in Kenya.

τ_1	Rate at which less or equal to five year's mildly infected children seek treatment in Kenya.
τ_2	Rate at which less or equal to five year's chronically infected children seek treatment in Kenya.
K	Coefficient at which force of infection is accelerated due to environmental factors.

Table 2: Definition of terms

`Endemic	It is long term infection which stays in the population at least 10 to 20 years.
Susceptible population	Proportion of the children population who are free of infection but at risk of contracting the infection
Vaccinated population	Proportion of the children populations who are free of infection and vaccinated with pneumonia but are at a lower risk of contracting the infection.
Mild Infected population	Proportion of the children population with the disease causing pathogen and capable of transmitting the infection to other children on contact but are non-severely infected.
Chronically Infected population	Proportion of the children population with the disease causing pathogen and capable of transmitting the infection to other children on contact but are severely infected.
Mild treated population	Proportion of the children population with the disease causing pathogen under treatment and capable of transmitting the infection to other children on contact. Mostly treated as outpatient in our health facilities
Chronic treated population	Proportion of the children population with the disease causing pathogen under treatment and capable of transmitting the infection to other children on contact. Mostly treated as inpatient in our health facilities
Recovered population	Proportion of the children population who are free of infection after treatment. The effect of treatment drugs is still in their body and they are highly unlikely to contract the infection.
Infectious	Diseases where individuals are infected by pathogen micro-organisms, for

Disease	instance viruses, bacteria, fungi or other micro parasites.
Alveoli	Microscopic sacs in the lungs that absorb oxygen.
Morbidity	Impairments as a result of a disease
Mortality	Susceptibility to death
Virulence	The degree of pathogenicity of a microorganism as indicated by the severity of disease produced and the ability to invade the tissues of the host.
Efficacy	A measure of how efficient is the drug. If the efficiency is 0% then it is useless but if it is 100% then it is perfect.
MATLAB	Mathematical software.
Etiology	The investigation of attribution of the cause or reason for something