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NUMERICAL SIMULATIONS OF A MATHEMATICAL MODEL FOR TRANSMISSION AND CONTROL OF MEASLES INCORPORATING VACCINATION AND TREATMENT.

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ABSTRACT

In this study, we carried out numerical simulations of a mathematical model for transmission and control of measles incorporating vaccination and treatment. We solved the model equations using Homotopy perturbation method. The results obtained were coded using Maple Mathematical Software and graphical profiles of each compartment generated in order to have a better understanding of the dynamic of the disease. Numerical simulations of the model show that, the combination of vaccination and treatment is the most effective way to combat the epidemic of measles in the community. The model strongly indicated that the spread of the disease largely depend on the contact rates with infected individuals within the population.

Keywords: *Mathematical Model, Vaccination, Treatment, Numerical Simulation and Treatment.*

Introduction:

Measles is an infection of the respiratory system caused by a virus of the genus Morbillivirus. The disease is spread through respiration following contact with fluids from an infected person's nose and mouth, either directly or indirectly. The disease is highly contagious with 90% chances of being transmitted to individuals without immunity. Measles infects about 30 to 40 million children every year. The disease has been, and remains, a major killer of children around the world. Despite the introduction of the measles vaccine in 1963, measles caused an estimated 2.6 million deaths in a single year as far back as 1980. According to WHO, in 2002 alone, the disease is estimated to have caused 614 000 death globally, with more than half of fatalities occur in sub-Saharan Africa. Survivor children subsequently suffer blindness, deafness or impaired vision, brain damage, and death (WHO, 2005).

The control of measles is largely based on the use of MMR (measles, mumps, and rubella) and MMRV (measles, mumps, rubella, and varicella) vaccines, which are believed to be about 95% effective. Worldwide, measles vaccination has been very effective, preventing an estimated 80 million cases and 4.5 million deaths annually (Simons et al, 2012).

Although global incidence has been significantly reduced through vaccination, measles remains an important public health problem. Since vaccination coverage is not uniformly high worldwide, measles stands as the leading vaccine-preventable killer of children worldwide; measles is estimated to have caused 614 000 global deaths annually in 2002, with more than half of measles deaths occur in sub-Saharan Africa (Ejima *et al,* 2012).

Kassem and Ndam (2010). in their work titled "A Stochastic modeling of recurrent measles epidemics", developed a simple stochastic mathematical model for the dynamics of measles with multidimensional diffusion process. In developing their model, they considered and partitioned the population into; susceptible, latent (exposed), infected and removed classes, they assumed, among other things that stochastic effects arise in the process of infection of susceptible individuals. The results of their simulation seemed to agree with the historical pattern of measles in Nigeria.

Momoh *et al*. (2013). developed a model that divides the total population (N) into four classes: Susceptible (S), Exposed (E), infected (I) and Recovered (R) classes, they incorporated testing and measles therapy into the dynamics at the latent (exposed) period to investigate the control of measles epidemiology at latent period. They assumed that both recovered individuals from exposed class as a result of testing and measles therapy and naturally recovered infected individuals becomes permanently immune, and developed a non-linear first order ordinary differential Equation. The result of their stability analysis showed that the system is asymptotically stable

(Bolarin, 2014). developed a mathematical model on the dynamical analysis of a new model for measles infection. His study used SEIR model modified by adding vaccinated compartment. His model determined the required vaccination coverage and dosage that will guarantee eradication of measles disease within a population

(Bakare *et al*., 2012). Studied modeling and simulation of the dynamics of the transmission of measles disease. They used SEIR model to discuss dynamics of measles infection and address the stability of disease free and endemic equilibrium states. The impact of vaccination in the control and elimination of measles was not considered in the work.

Ochoche and Gweryina (2014). developed a mathematical models of measles incorporating vaccination as a control strategy and capturing two phase of infectiousness ((i.e. asymptomatic infectives and symptomatic infectives). The basic reproduction Number R_0 was calculated using next generation matrix approach and proved that the system of Equations is locally asymptotically stable if R_0 is less than one. From their study they concluded that the disease will certainly be eliminated if all susceptible individual are vaccinated. But in their work they didn't incorporate screening and treatment of the measles disease.

Peter et-al (2018). Developed mathematical model of measles dynamics with vaccination by considering the total number of recovered individuals either from natural recovery or recovery due to vaccination. The population was divided into five compartments (Susceptible, Exposed, Infectious, Recover and Vaccination).

The existence and uniqueness of solution for the model were tested using Lipchitz condition to ascertain the efficacy of the model and also the disease free equilibrium (DFE) and the endemic equilibrium (EE) for the Equation of the system were obtained and the basic reproduction Number R_0 were calculated which shows that is asymptotically stable.

The numerical simulation of the model shows that vaccination is capable of reducing the number of exposed and infectious population. But in their work they didn't include screening and treatment.

MATERIAL AND METHODS

Model description

We divided the population into six mutually-exclusive compartments namely; Susceptible Class (S), Vaccinated Class (V), Exposed Class (E), Exposed Receiving treatment Class (E_T), Infectious Class (I) and Recovered Class (R).

The population of Susceptible Class (S) increases through constant recruitment A and decreases by natural death at the rate μ and force of infection λ, it further decreases by rate at which susceptible individual receive vaccine γ. The Vaccinated Class (V) increases by rate at which susceptible individual receive vaccine γ and decreases by natural death at the rate μ. The Exposed Class (E) increases with force of infection λ of Susceptible Class (S) and decreases with natural death at the rate μ, rate at which exposed individuals becomes infectious α and rate at which exposed individual move to exposed treated class δ. The Exposed Receiving treatment Class (E_T) increases with rate at which exposed individual move to exposed treated class δ and decreases with natural death at the rate μ and recovery rate for the exposed treated individual ω. The Infectious Class (I) increases with the rate at which exposed individuals becomes infectious α and decreases with death due to measles infection μ_I, natural death at the rate μ and recovery rate for the infectious individuals β_2. Finally, the Recovered Class (R) increases with recovery rate for the infectious individuals β_2 and recovery rate for the exposed treated individual ω and decreases with natural death at the rate μ. The force of infection $\lambda = \frac{\beta c I}{N}$ where β is the Probability of infectious individual infecting others, c is per capita contact rate for measles and N is the total population.

This gives the following system of ordinary differential Equations;

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Model Equations

$$\frac{dS}{dt} = A - (\mu + \gamma + \lambda)S \qquad (3.1)$$

$$\frac{dV}{dt} = \gamma S - \mu V \qquad (3.2)$$

$$\frac{dE}{dt} = \lambda S - (\mu + \alpha + \delta)E \qquad (3.3)$$

$$\frac{dE_T}{dt} = \delta E - (\mu + \omega)E_T \qquad (3.4)$$

$$\frac{dI}{dt} = \alpha E - (\mu_1 + \mu + \beta_2)I \qquad (3.5)$$

$$\frac{dR}{dt} = \beta_2 I + \omega E_T - \mu R \qquad (3.6)$$

Where $\lambda = \frac{\beta c I}{N}$

Analytical solution of the model using homotopy perturbation method (HPM)

The fundamental of Homotopy Perturbation Method (HPM) was first proposed by Ji-Haun (2000). The Homotopy Perturbation Method (HPM), which provides analytical approximate solution, is applied to various linear and non-linear Equations. The homotopy perturbation method (HPM) is a series expansion method used in the solution of nonlinear partial differential Equations Jiya (2010).

To show the simple concepts of this method, he considered the following non-linear differential Equation:

$$A_3(U) - f(r) = 0, \qquad r \in \Omega \qquad (3.7)$$

Subject to the boundary condition

$$B_3\left(U, \frac{\partial U}{\partial n}\right) = 0, \qquad r \in \Gamma \qquad (3.8)$$

Where A_3 is a general differential operator, B_3 a boundary operator, $f(r)$ is a known analytical function and Γ is the boundary of the domain Ω. The

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operator A_3 can be divided into two parts L and N, where L is the linear part, and N is the nonlinear part. Equation (3.7) can be written as:

$$L(U)+N(U)-f(r)=0, \quad r \in \Omega \tag{3.9}$$

The Homotopy Perturbation structure is shown as follows

$$H(V,h)=(1-h)[L(V)-L(U_0)]+h[A(V)-f(r)]=0 \quad (3.10$$

Where $V(r,P):\Omega \in [0,1] \rightarrow R$

In Equation (3.9) $P \in [0,1]$ is an embedding parameter and U_0 is the approximation that satisfies the boundary condition. It can be assumed that the solution of the Equation (3.9) can be written as power series in h as follows:

$$V = V_0 + hV_1 + h^2V_2 + ... \tag{3.11}$$

And the best approximation for the solution is:

$$U = \lim_{h \rightarrow 1} v = v_0 + hv_1 + h^2v_2 + ... \tag{3.12}$$

The series (3.12) is convergent for most cases. However, the convergent rate depends on the nonlinear operator A (V).

Solution of the model equations

$$\frac{dS}{dt}+(\mu+\gamma+\lambda)S-A=0 \tag{3.13}$$

$$\frac{dV}{dt}+\mu V-\gamma S=0 \tag{3.14}$$

$$\frac{dE}{dt}+(\mu+\alpha+\delta)E-\lambda S=0 \tag{3.15}$$

$$\frac{dE_T}{dt}+(\mu+\omega)E_T-\delta E=0 \tag{3.16}$$

$$\frac{dI}{dt}+(\mu_1+\mu+\beta_2)I-\alpha E=0 \tag{3.17}$$

$$\frac{dR}{dt}+\mu R-\beta_2 I-\omega E_T=0 \tag{3.18}$$

Solving equations (3.13) to (3.18) using Homotopy perturbation method with the following initial conditions $S(0)=S_{(0)}$, $V(0)=V_{(0)}$ $E(0)=E_{(0)}$, $E_T(0)=E_T o$, $I(0)=I_0$, $R(0)=R_0$, we obtained the general solutions of the model as

$$S(t)=S_0+(A-\frac{(\mu+\gamma+\beta cI_0)S_0}{N})t-\left[(\mu+\gamma+\frac{\beta cI_0}{N})(A-\frac{(\mu+\gamma+\beta cI_0)S_0}{N})\right]\frac{t^2}{2},$$

$$V(t)=V_0+(\gamma S_0-\mu V_0)t+\left[\gamma(A-\frac{(\mu+\gamma+\beta cI_0)S_0}{N})-\mu(\gamma S_0-\mu V_0)\right]\frac{t^2}{2}$$

$$E(t)=E_0+(\frac{\beta cI_0S_0}{N}-(\mu+\alpha+\delta)E_0)t+\left[\begin{array}{l}\dfrac{\beta cI_0(A-\frac{(\mu+\gamma+\beta cI_0)S_0}{N})-\beta c(\alpha E_0-(\mu+\mu_1+\beta_2)I_0)S_0}{N}\\ -(\mu+\alpha+\delta)(\frac{\beta cI_0S_0}{N}-(\mu+\alpha+\delta)E_0)\end{array}\right]\frac{t^2}{2}$$

,

$$E_T(t)=E_{T(0)}+(\delta E_0-(\mu+\omega)E_{T(0)})t+\left[\delta(\frac{\beta cI_0S_0}{N}-(\mu+\alpha+\delta)E_0)-(\mu+\omega)(\delta E_0-(\mu+\omega)E_{T(0)})\right]\frac{t^2}{2}$$

$$I(t)=I_0+(\alpha E_0-(\mu+\mu_1+\beta_2)I_0)t+\left[\alpha(\frac{\beta cI_0S_0}{N}-(\mu+\alpha+\delta)E_0)-(\mu_1+\mu+\beta_2)(\alpha E_0-(\mu+\mu_1+\beta_2)I_0)\right]\frac{t^2}{2}$$

and

$$R(t)=R_0+(wE_T+\beta_2I_0-\mu R_0)t+\left[\begin{array}{l}\beta_2(\alpha E_0-(\mu+\mu_1+\beta_2)I_0)+w(\delta E_0-(\mu+\omega)E_{T(0)})\\ -\mu(wE_{T(0)}+\beta_2I_0-\mu R_0)\end{array}\right]\frac{t^2}{2}$$

Numerical Simulation

In this section, we plot the graph of analytical solution of our model Equations using maple software.

Table 4.1: shows initial conditions for each plot and parameters values

Parameters and State Variables	Value	Source
S	15,000	Assumed
V	1,500	Assumed
E	6000	Assumed
E_T	3000	Assumed
I	1000	Assumed
R	600	Assumed
β	0.1	Assumed
γ	Control parameter	Assumed
μ	0.0875	Agnes (2012)
δ	0.1	Assumed
α	0.125	Agnes (2012)
β_2	Control parameter	Assumed
μ_1	0.125	Fred (2012)
C	0.2	Assumed
N	27,000	Assumed
λ	0.0007	Calculated
A	0.2755	Agnes (2012)

Maple codes for simulations.

$$S(t) := S[0] + \left(A - \left(\gamma + \mu + \frac{\beta \cdot c \cdot F[0]}{N}\right) \cdot S[0]\right) \cdot t + \left(\left(\gamma + \mu + \frac{\beta \cdot c \cdot F[0]}{N}\right) \cdot \left(A - \left(\gamma + \mu + \frac{\beta \cdot c \cdot F[0]}{N}\right) \cdot S[0]\right)\right) \cdot \frac{t^2}{2};$$

$$S(t) := S_0 + \left(A - \left(\gamma + \mu + \frac{\beta\, c\, F_0}{N}\right) S_0\right) t + \frac{1}{2}\left(\gamma + \mu + \frac{\beta\, c\, F_0}{N}\right)\left(A - \left(\gamma + \mu + \frac{\beta\, c\, F_0}{N}\right) S_0\right) t^2$$

$$V(t) := V[0] + (\gamma \cdot S[0] - \mu \cdot V[0]) \cdot t + \left(\left(\gamma \cdot S[0] - \left(\gamma + \mu + \frac{\beta \cdot c \cdot F[0]}{N}\right) \cdot S[0] - \mu \cdot (\gamma \cdot S[0] - \mu \cdot V[0])\right)\right) \cdot \frac{t^2}{2};$$

$$V(t) := V_0 + (\gamma S_0 - \mu V_0)\, t + \frac{1}{2}\left(\gamma S_0 - \left(\gamma + \mu + \frac{\beta c F_0}{N}\right) S_0 - \mu(\gamma S_0 - \mu V_0)\right) t^2$$

$$E(t) := E[0] + \left(\frac{\beta \cdot c \cdot F[0] \cdot S[0]}{N} - (\delta + \alpha + \mu) \cdot E[0]\right) \cdot t + \left(\frac{\beta \cdot c \cdot F[0]}{N} \cdot \left(A - \left(\mu + \gamma + \frac{\beta \cdot c \cdot F[0]}{N}\right) \cdot S[0] - \frac{\beta \cdot c \cdot F[0] \cdot S[0]}{N} \cdot (\alpha \cdot E[0]) - (\mu + \mu[1]) + \beta[2]\right) - (\mu + \alpha + \delta) \cdot \left(\frac{\beta \cdot c \cdot F[0] \cdot S[0]}{N} - (\delta + \alpha + \mu) \cdot E[0]\right)\right) \cdot \frac{t^2}{2};$$

$$E(t) := E_0 + \left(\frac{\beta c F_0 S_0}{N} - (\delta + \alpha + \mu) E_0\right) t + \frac{1}{2}\left(\frac{\beta c F_0 \left(A - \left(\gamma + \mu + \frac{\beta c F_0}{N}\right) S_0 - \frac{\beta c F_0 S_0 \alpha E_0}{N} - \mu - \mu_1 + \beta_2\right)}{N} - (\delta + \alpha + \mu)\left(\frac{\beta c F_0 S_0}{N} - (\delta + \alpha + \mu) E_0\right)\right) t^2$$

$$E_{t1}(t) := E[1] + (\delta \cdot E[0] - (\omega + \mu) \cdot E[1]) \cdot t + \left(\delta \cdot \left(\frac{\beta \cdot c \cdot F[0] \cdot S[0]}{N} - (\delta + \alpha + \mu) \cdot E[0]\right) - (\omega + \mu) \cdot (\delta \cdot E[0] - (\omega + \mu) E[1])\right) \cdot \frac{t^2}{2};$$

$$E_{t1}(t) := E_1 + (\delta E_0 - (\omega + \mu) E_1)\, t + \frac{1}{2}\left(\delta\left(\frac{\beta c F_0 S_0}{N} - (\delta + \alpha + \mu) E_0\right) - (\omega + \mu)(\delta E_0 - (\omega + \mu) E_1)\right) t^2$$

$$F(t) := F[0] + (\alpha \cdot E[0] - (\mu[1] + \mu + \beta[2]) \cdot F[0]) \cdot t + \left(\alpha \cdot \left(\frac{\beta \cdot c \cdot F[0] \cdot S[0]}{N} - (\delta + \alpha + \mu) \cdot E[0]\right) - (\mu[1] + \mu + \beta[2]) \cdot (\alpha \cdot E[0] - (\mu[1] + \mu + \beta[2]) \cdot F[0])\right) \cdot \frac{t^2}{2};$$

$$F(t) := F_0 + (\alpha E_0 - (\mu_1 + \mu + \beta_2) F_0)\, t + \frac{1}{2}\left(\alpha\left(\frac{\beta c F_0 S_0}{N} - (\delta + \alpha + \mu) E_0\right) - (\mu_1 + \mu + \beta_2)(\alpha E_0 - (\mu_1 + \mu + \beta_2) F_0)\right) t^2$$

$$R(t) := R[0] + (\omega \cdot E[1] + \beta[2] \cdot F[0] - \mu \cdot R[0]) \cdot t + (\beta[2] \cdot (\alpha \cdot E[0] - (\mu[1] + \mu + \beta[2]) \cdot F[0]) + \omega \cdot (\delta \cdot E[0] - (\omega + \mu) \cdot E[1]) - \mu \cdot (\omega \cdot E[1] + \beta[2] \cdot F[0] - \mu \cdot R[0])) \cdot \frac{t^2}{2};$$

$$R(t) := R_0 + (\omega E_1 + \beta_2 F_0 - \mu R_0)\, t + \frac{1}{2}\left(\beta_2 (\alpha E_0 - (\mu_1 + \mu + \beta_2) F_0) + \omega (\delta E_0 - (\omega + \mu) E_1) - \mu (\omega E_1 + \beta_2 F_0 - \mu R_0)\right) t^2$$

$$B1 := eval(S(t), \{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R = 600, \beta_2 = 0.25, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.25\});$$

$$B1 := 15000 - 5073.335610\, t - 858.0043975\, t^2$$

$$B2 := eval(V(t), \{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R = 600, \beta_2 = 0.25, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.25\});$$

$$B2 := 1500 + 3618.7500\, t - 820.1258675\, t^2$$

$$B3 := eval(E(t), \{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R_0 = 600, \beta_2 = 0.25, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.25\});$$

$$B3 := 6000 - 1863.888889\, t + 286.2672198\, t^2$$

$$B4 := eval(E_{t1}(t), \{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R = 600, \beta_2 = 0.25, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.25\});$$

$$B4 := 3000 + 37.5000\, t - 96.71006945\, t^2$$

$$B5 := eval(F(t), \{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R_0 = 600, \beta_2 = 0.25, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.25\});$$

$$B5 := 1000 + 287.5000\, t - 182.9774306\, t^2$$

$$B6 := eval(R(t), \{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R_0 = 600, \beta_2 = 0.25, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.25\});$$

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$B6 := 600 + 497.5000\,t + 16.04687500\,t^2$

$B21 := eval\left(V(t), \{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R = 600, \beta_2 = 0.25, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.25\}\right);$

$B21 := 1500 + 3618.7500\,t - 820.1258675\,t^2$

$B22 := eval\left(V(t), \{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R = 600, \beta_2 = 0.25, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.50\}\right);$

$B23 := eval\left(V(t), \{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R = 600, \beta_2 = 0.25, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.75\}\right);$

$B23 := 1500 + 11118.7500\,t - 1148.250868\,t^2$

plot([B21, B22, B23,], t = 0 ..2, thickness = [4, 5, 6,], color = [red, blue, black,], linestyle = [solid, dash, dot,]);

$B51 := eval\left(F(t), \{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R_0 = 600, \beta_2 = 0.25, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.25\}\right);$

$B51 := 1000 + 287.5000\,t - 182.9774306\,t^2$

$B52 := eval\left(F(t), \{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R_0 = 600, \beta_2 = 0.55, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.50\}\right);$

$B52 := 1000 - 12.5000\,t - 111.7274306\,t^2$

$B53 := eval\left(F(t), \{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R_0 = 600, \beta_2 = 0.70, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.75\}\right);$

$B53 := 1000 - 162.5000\,t - 42.35243055\,t^2$

plot([B51, B52, B53,], t = 0 ..2, thickness = [4, 5, 6,], color = [red, blue, black,], linestyle = [solid, dash, dot,]);

$$B31 := eval\left(E(t), \left\{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R_0 = 600, \beta_2 = 0.25, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.25\right\}\right);$$

$$B31 := 6000 - 1863.888889\, t + 286.2672198\, t^2$$

$$B32 := eval\left(E(t), \left\{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R_0 = 600, \beta_2 = 0.55, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.50\right\}\right);$$

$$B32 := 6000 - 1863.888889\, t + 284.8784420\, t^2$$

$$B33 := eval\left(E(t), \left\{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R = 600, \beta_2 = 0.70, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.75\right\}\right);$$

$$B33 := 6000 - 1863.888889\, t + 283.4896088\, t^2$$

plot([*B31*, *B32*, *B33*,], *t* = 0 ..2, *thickness* = [4, 5, 6,], *color* = [*red*, *blue*, *black*,], *linestyle* = [*solid*, *dash*, *dot*,]);

$$B61 := eval\left(R(t), \left\{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R_0 = 600, \beta_2 = 0.25, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.25\right\}\right);$$

$$B61 := 600 + 497.5000\, t + 16.04687500\, t^2$$

$$B62 := eval\left(R(t), \left\{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R_0 = 600, \beta_2 = 0.55, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.50\right\}\right);$$

$$B62 := 600 + 797.5000\, t - 36.45312500\, t^2$$

$$B63 := eval\left(R(t), \left\{S_0 = 15000, V_0 = 1500, E_0 = 6000, E_1 = 3000, F_0 = 1000, R_0 = 600, \beta_2 = 0.70, N = 27000, \mu = 0.0875, \delta = 0.1, \alpha = 0.125, \lambda = 0.0007, \mu_1 = 0.125, c = 0.2, A = 0.2755, \omega = 0.1, \beta = 0.1, \gamma = 0.75\right\}\right);$$

$$B63 := 600 + 947.5000\, t - 96.45312500\, t^2$$

plot([*B61*, *B62*, *B63*,], *t* = 0 ..2, *thickness* = [4, 5, 6,], *color* = [*red*, *blue*, *black*,], *linestyle* = [*solid*, *dash*, *dot*,]);

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Graphical Representation of Solutions of the Model Equation

The graphical representations are from the analytical solutions of the model equations. They are plotted using MAPLE software.

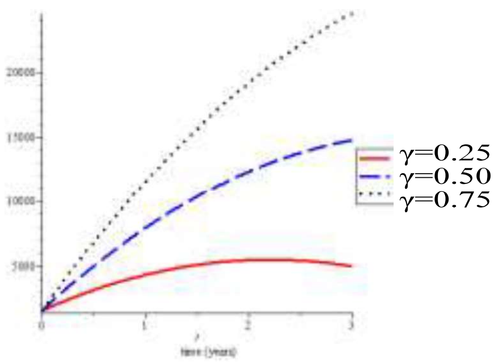


Figure 4.1: Graph of Vaccinated individual against time for different vaccination rate.

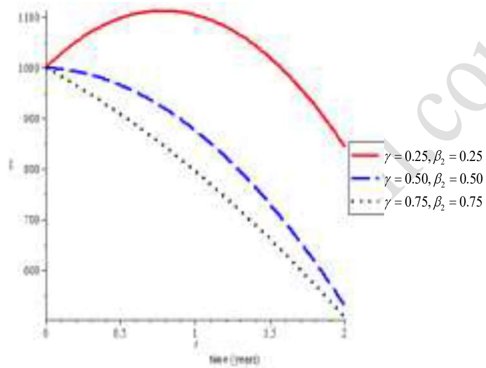


Figure 4.2: Graph of infectious individual against time for different vaccination and treatment rate.

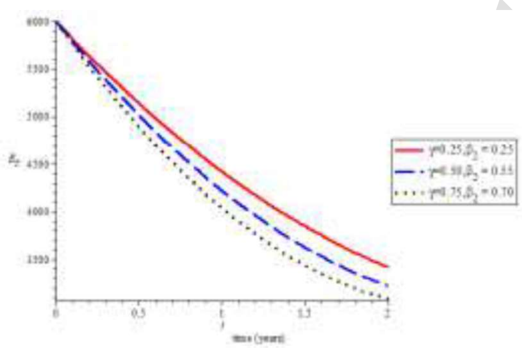


Figure 4.3: Graph of exposed receiving treatment against time for different vaccination and treatment.

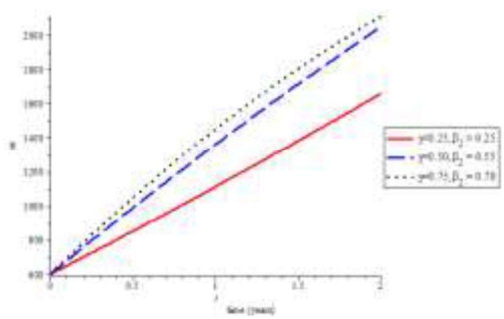
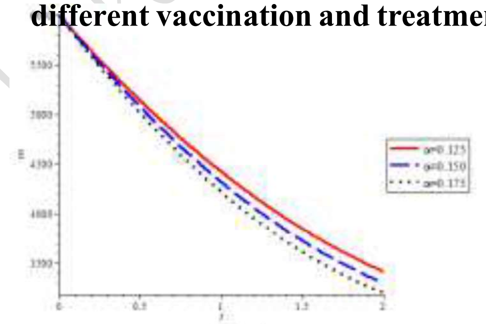


Figure 4.4: Graph of recovered individual against time for different vaccination and treatment rate.



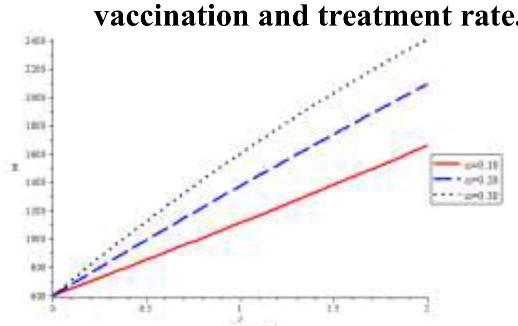


Figure 4.5: Graph of exposed individual against time for different rate at which exposed becomes infectious.

Figure 4.6: Graph of recovered individual against time for different recovery rate for the exposed treated individual.

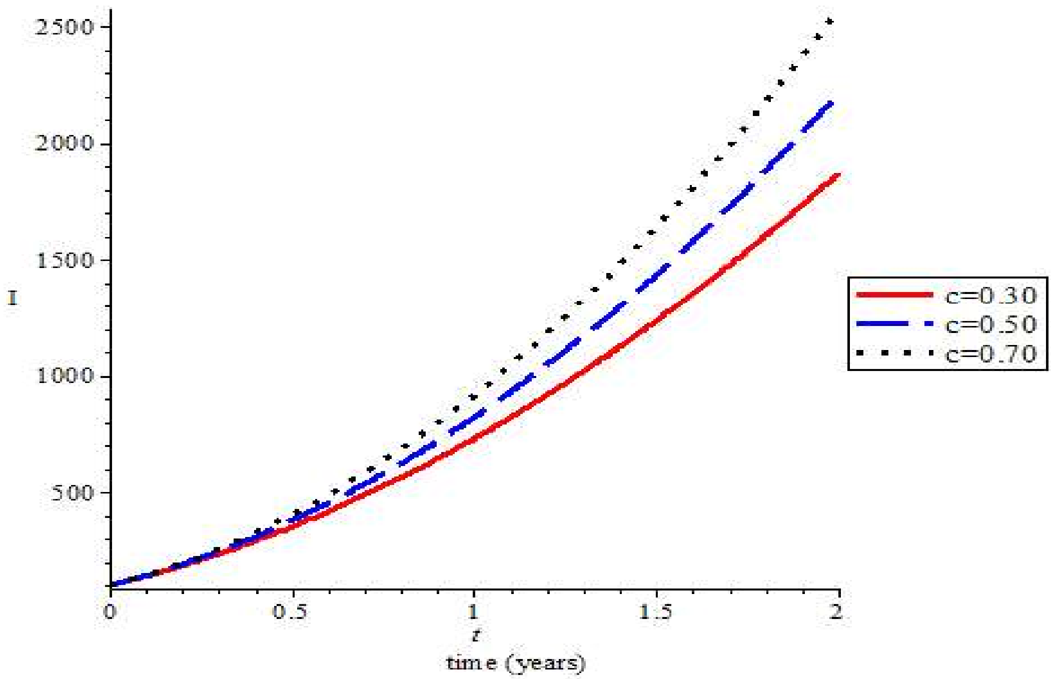


Figure 4.7: Graph of infected individual against time for different contacts rate

Discussion of Results

Figure 4.1 is a graph of vaccinated individuals against time for different vaccination rate. It shows that the population of the vaccinated individual increases to 20000 as the vaccination rate increases. It also shows that vaccination can reduce the peak of exposed and infected compartment drastically.

Figure 4.2 is a graph of Infectious individuals against time for different vaccination and treatment rates. The graph shows that the population of Infectious individuals decreases as vaccination and treatment rates increases. Finally the infected individuals of 1100 approaches zero which implies the disease seems to die out.

Figure 4.3 is a graph of Exposed individuals receiving treatment against time for different vaccination and treatment rates. The graph shows that the population of Exposed individuals receiving treatment decreases as vaccination and treatment rates increases. The decrement slightly varies for both low, moderate and high vaccination and treatment rates, this is because the infected individuals are partially infected so at any given vaccination and treatment rates, the disease can be eradicated fully.

Figure 4.4 is a graph of Recovered individuals against time for different vaccination and treatment rates. The graph shows that the population of Recovered individuals increases to 2000 as vaccination and treatment rates increases.

Figure 4.5 is a graph of Exposed individuals against time for different rate at which exposed individual become infectious. The graph shows that the population of Exposed individuals decreases as the rate at which exposed individuals become infectious increases. It reveals that the lower the rate of movement of individuals to treated compartment the higher individuals become infected.

Figure 4.6 is a graph of Recovered individuals against time for different recovery rate for the exposed treated individual. The graph shows that the population of recovered individual increases as the recovery rate for the exposed treated individual increases. Recovered individuals increase to 2400.

Figure 4.7 is a graph of infected individuals against time for different contact rates. It shows that the population of the infected individual increases as the contact rate increases. The infected individual increase to 2500 as contact rate increases, this implies that the disease largely depends on contact rate and person can transmit disease to more than one person and the spread of the measles disease will continue in a the society if unchecked.

REFERENCES

Bakare, E.A., Adekunle, Y.A., and Kadiri, K.O. (2012). Modelling and Simulation of the Dynamics of the transmission of Measles. *International Journal. Computer Trends and Technology.* 3,174-178.

Bolarin. G. (2014). On the Dynamical Analysis of a new Model for Measles Infection*. Anjorin Publishers, Osun State*. 32-38.

Ejima K.R., Omori, K.A, and Nishiura, H. (2012). Real-time Investigation of Measles Epidemics with Estimate of Vaccine Efficacy, *International Journal of Biological Sciences,* 8(5) 620-629.

Ji-Haun, H. (2000). A Coupling Method of a Homotopy and Perturbation Technique for non-linear problems, *International Journal*. *non-linear Mechanics*. 35:37-43.

Jiya, M. (2010). Application of Homotopy Perturbation Method (HPM) for the Solution of some Non-Linear .Differential Equations. *Pacific Journal of Science and Technology* 11(2), 268-272.

Kassem, T.G., and Ndam J.N. (2010). A Stochastic Modeling of Recurrent Measles Epidemics, *Science World Journal*, 3(4): 1597-634376.

Momoh A.A., Ibrahim M.O., Uwanta J.I., and Manga S.B. (2013). Mathematical Model for Control of measles Epidemiology. *International Journal of Pure and Applied Mathematics Volume* 87 No. 5 2013, 707-718.

Ochoche J.M., and Gweryina, R.I. (2014). A Mathematical Model of Measles with Vaccination and Two Phases of Infectiousness. *IOSR Journal of Mathematics*, *Volume* 10, 95-105.

Peter, O.J., afolabi, O.A., Victor, A.A., Akpan, C.E., and oguntolu, F.A. (2018). Mathematical Model of Measles Disease Dynamics with Vaccination by considering the total number of recovered individuals either from natural recovery or recovery due to vaccination. *J. Appl. Sci. Environ. Manage. Vol.* 22(4) 571 – 576.

Simons, E.F., Fricks, M.K., and Abhijeet, A.W. (2012). Assessment of the global measles mortality reduction. http://www.thelancet.com.

World Health Organization, Department of vaccines and biological, (2005). Measles Technical working Group: strategies for measles control and elimination. *Report of a meeting, Geneva*, 11-12 May .Geneva, Switzerland.