Mathematical Model for the Control of Infectious Disease

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ABSTRACT: We proposed a mathematical model of infectious disease dynamics. The model is a system of first order ordinary differential equations. The population is partitioned into three compartments of Susceptible $S(t)$, Infected $I(t)$ and Recovered $R(t)$. Two equilibria states exist: the disease-free equilibrium which is locally asymptotically stable if $R_o < 1$ and unstable if $R_o > 1$. Numerical simulation of the model shows that an increase in vaccination leads to low disease prevalence in a population.

Mathematical models are widely used to examine, explain and predict the dynamics of infectious disease transmission and models of specific diseases of global importance have played important role in developing public health strategies for control and prevention of infectious disease. (Anderson RM and May RM. 1991), (Grassly NC, Fraser C. 2008) and (Keeling M, Rohani P. 2007).

Infectious disease models provide a mathematical representation of the dynamic transmission cycle involving interactions between infected and susceptible individuals that are generally expressed as a set of Ordinary differential equations (ODEs). (Keeling M, Rohani P. 2007). Infectious diseases includes malaria, tuberculosis, cholera, AIDS, bird flu, lassa fever, ebola and could be transmitted through direct or indirect contact with contaminated body fluid or surface most especially through sex, blood transfusion, breast feeding, etc. (Shah and Gupta, 2013). Some examples on the use of mathematical model for the analyzes of treatment and control of infectious disease can be found in(Shah, N.H. and Gupta, J. 2013), (Seidu, B. and Makinde, O.D. (2014)., (Tripathi, A., et al 2007). The aim of this paper is to design and rigorously analyze a model that extends and complements the ones in the literatures by incorporating vaccination as control parameter

MATERIALS AND METHOD.

We formulate A non-linear deterministic model for the transmission dynamics of infectious diseases .The model subdivides the human population into three (3) compartments depending on the epidemiological status of individuals. The compartments are susceptible *S,* infected *I* and the recovered *R*. To indicate this mathematically, we have

$$\frac{dS}{dt} = \beta - \alpha SI - (\rho + \mu)S + \sigma R \qquad (1)$$

$$\frac{dI}{dt} = \alpha SI - (\gamma + \delta + \mu)I \qquad \ldots\ldots (2)$$

$$\frac{dR}{dt} = \gamma I - (\mu + \sigma)R + \rho S \qquad \ldots\ldots .(3)$$

The following assumptions were considered to construct the model

1   All susceptible individuals can be infected through a direct contact with infectious individuals

2.   Individuals are only recruited into the susceptible class.

3.   Birth rate is not equal to death rate.

4.   We assume that susceptible individuals get infected with typhoid at a rate proportional to the susceptible population.

5.   All parameters are non-negative.

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Equilibrium States of the Model: At equilibrium

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

Let

$$S = x, I = y, R = z \;\therefore$$

$$\beta - \alpha xy - (\rho + \mu)x + \sigma z = 0 \qquad (4)$$

$$\alpha xy - (\gamma + \delta + \mu)y = 0 \qquad (5)$$

$$\gamma y - (\mu + \sigma)z + \rho x = 0 \qquad (6)$$

Solving equation (4)-(6) gives the disease free equilibrium

Disease- Free Equilibrium State (DFE): The equilibrium state in the absence of infection is known as the disease- free equilibrium and is such that, y = 0,.

Therefore the Disease Free equilibrium is

$$(x, y, z) = \left(\frac{\beta}{\mu}, 0, 0 \right)$$

The Endemic Equilibrium State (EE): The equilibrium state with the presence of infection (i.e. $y \neq 0$) is known as endemic equilibrium. Therefor the EE is given as $x = \dfrac{\gamma + \delta + \mu}{\alpha}$

$$y = \frac{\mu\alpha\beta - \mu\rho\gamma - \mu\rho\delta - \rho\mu^2 - \mu^2\gamma - \mu^2\delta - \mu^3 + \alpha\sigma\beta - \mu\sigma\gamma - \mu\sigma\delta - \mu^2\sigma}{\alpha\left(\mu\gamma + \mu\delta + \mu^2 + \delta\sigma + \mu\sigma\right)}$$

$$z = \frac{\gamma\alpha\beta - \mu\gamma^2 - \gamma\mu\delta - \gamma\mu^2 + \rho\gamma\delta + \rho\gamma\mu + \rho\delta^2 + 2\rho\delta\mu + \rho\mu^2}{\alpha\left(\gamma\mu + \delta\mu + \delta\sigma + \mu^2 + \mu\sigma\right)}$$

Existence of the Model in a Feasible Region F: Here, we provide the following results which guarantee that the infectious disease model governed by equations (1) – (3) is epidemiologically well-posed in a feasible region F given by

Where F = $\left\{ (S, I, R) \in R_+^s : S \leq \frac{\beta}{\mu} \right\}$

Theorem 1: There exists a domain F in which the solution set $\{S, I, R\}$ is contained and bounded.

Proof: Given the solution set $\{S, I, R\}$ with positive initial conditions

$S(0) = s_0$ I (0) = I₀, R(0) = R₀. We let

G = S + I + R $\Rightarrow$ G (t) =S (t) + I (t) + R (t)

Now from equations (1) – (3)

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Now from equations (1) – (3)

$$\frac{dG}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$$

$$= \beta - \alpha SI - (\rho + \mu)S + \sigma R + \alpha SI - \gamma I - \delta I - \mu I + \gamma I - \mu R - \sigma R + \rho S$$

$$= \beta - \mu(S + I + R) - \delta I = \beta - \mu G - \delta I$$

$$\therefore \frac{dG}{dt} = \beta - \mu G - \delta I \Rightarrow \frac{dG}{dt} \leq \beta - \mu G$$

$$\frac{dG}{dt} = \mu G \leq \beta - \mu G$$

(Where δI>0)

$$\Rightarrow \frac{dG}{dt} + \mu G \leq \beta$$

Where the integrating factor is $e^{\mu t}$
Now

$$Ge^{\mu t} \leq \int \beta e^{\mu t} dt$$

$$\Rightarrow Ge^{\mu t} \leq \frac{\beta e^{\mu t}}{\mu} + c$$

$$G \leq \frac{\beta}{\mu} + e^{-\mu t} \Rightarrow G(t) \leq \frac{\beta}{\mu} + \lim_{t \to \infty}(Ce^{-\mu t})$$

Hence

$$G(t) \leq \frac{\beta}{\mu}$$

Thus, the solution of the population are confined in the feasible region *F*. This shows that the feasible region for the model equation (1)-(3) exist.

Next is to show that the systems (1) – (3) are nonnegative in *F* for any time $t > 0$ since the model represents population of human being.

Theorem 2. The solutions *S, I, R* of the model equation (1) – (3) with positive initial values in the feasible domain *F* is nonnegative in F for all time, $t > 0$

Proof. This is going to be proved in respect to the methodology of [2] and [3].
From equation (1)

$\dot{S} = \beta - \alpha SI - (\rho + \mu)S + \sigma R \Rightarrow \dot{S} + (\rho + \mu)S = \sigma R + \beta - \alpha SI$

$$\dot{S} + (\alpha I + \rho + \mu)S = \sigma R + \beta$$

The integrating factor is given as $e^{(\alpha I + \rho + \mu)t}$
Now

$$Se^{(\alpha I + \rho + \mu)t} = \int e^{(\alpha I + \rho + \mu)t} \cdot (\sigma R + \beta)dt$$
$$= (\sigma R + \beta) \int e^{(\alpha I + \rho + \mu)t} dt$$
$$= \frac{(\sigma R + \beta)}{\alpha I + \rho + \mu} e^{(\alpha I + \rho + \mu)t} + C$$

$$S = \frac{\sigma R + \beta}{\alpha I + \rho + \mu} + Ce^{-(\alpha I + \rho + \mu)t}$$

Now

$$, \lim_{t \to 0} \left[\frac{(\sigma R + \beta)}{\alpha I + \rho + \mu} + Ce^{-(\alpha I + \rho + \mu)t} \right]$$

$$S = \frac{(\sigma R + \beta)}{\alpha I + \rho + \mu} + \epsilon C > 0$$

Since all the parameters are positive then S > 0. Following the same method, we can easily verify that $I(t) > 0$ and $R(t) > 0$ for all $t > 0$. Hence, the solution *S, I, R* of the model (1) – (3) is nonnegative for all time $t > 0$.

Basic Reproduction Number: The basic reproduction number commonly denoted R_o. This quantity is known as the average number of secondary infections by the infected person during a specific period of time in a population of susceptible (Heffernan et al., 2005). Thus, whether a disease becomes persistent or dies out in a community depends on the value of the reproduction number, R_o. Furthermore, stability of equilibrium can be analyzed using R_o. If $R_o < 1$ it

means that every infectious individual will cause less than one secondary infection and hence the disease will die out and when $R_o > 1$ every infectious individual will cause more than one secondaryinfection and hence the disease will invade the population. It is Obtained by taking the largest (dominant) eigenvalue (spectral radius)

$$R_0 = \left[\frac{\partial F_i(x_o)}{\partial x_j} \right] \left[\frac{\partial V_i(x_o)}{\partial x_j} \right]^{-1}$$

Where F_i be the rate of appearance of new criminal in compartments, V_i is the transfer of individuals out of the compartment by another means, x_o is the disease free equilibrium. We compute the basic reproduction number using the next generation matrix approch. The basic reproduction number for the model in system (1)-(3) is given as
Hence,

$$R_0 = \frac{\alpha \beta}{\mu(\gamma + \delta + \mu)}$$

Stability Analysis of Disease Free Equilibrium: By employing the Basic Reproduction Number (R_0) obtained earlier, we analyze the stability of the DFE as follows

Theorem 3. The disease-free equilibrium, E_0 for model (1) – (3) is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$

Proof: Recall that the system of equations of the model at equilibrium state is;

$$\beta - \alpha xy - (\rho + \mu)x + \sigma z = 0$$

$$\alpha xy - (\gamma + \delta + \mu)y = 0$$

$$\gamma I - (\mu + \sigma)z + \rho x = 0$$

The Jacobian matrix of the system of equations is given by:

$$J = \begin{bmatrix} -(\alpha y + \rho + \mu) & -\alpha x & \sigma \\ \alpha y & \alpha x - (\gamma + \delta + \mu) & 0 \\ \rho & \gamma & -(\mu + \sigma) \end{bmatrix}$$

The Jacobian Matrix of the system (1) – (3) at the disease free equilibrium point E_0 is evaluated as follows:

$$J(E_0) = \begin{bmatrix} -(\rho + \mu + \lambda) & \dfrac{-\alpha\beta}{\mu} & \sigma \\ 0 & \dfrac{\alpha\beta}{\mu} - (\gamma + \delta + \mu) - \lambda & 0 \\ \rho & \gamma & -(\mu + \sigma + \lambda) \end{bmatrix}$$

The characteristics equation of the matrix above is obtained by

$$\det|J - \lambda I| = 0$$

The eigenvalues of $J(E_0)$ are $-(\rho + \mu)$, $-\left[-(\gamma + \delta + \mu) + \frac{\alpha\beta}{\mu}\right]$ and $-(\mu + \sigma)$

. The solution of the characteristic polynomial is $B(x) = x^3 + \vartheta_1 x^2 + \vartheta_2 x + \vartheta_3 = 0$ Where ϑ_3 is given by

$$\delta\mu^2 + \delta\mu\rho + \delta\mu\sigma + \gamma\mu^2 + \gamma\mu\rho + \gamma\mu\sigma + \mu^3 + \mu^2\rho + \mu^2\sigma - \mu\alpha\beta - \rho\alpha\beta - \sigma\alpha\beta > 0$$

Solving ϑ_3 directly, it can be factorised thus

$$\rho\mu(\delta + \gamma + \mu) + \sigma\mu(\delta + \gamma + \mu) + \mu^2(\delta + \gamma + \mu) - \alpha\beta(\mu + \rho + \sigma) > 0$$

$$(\delta + \gamma + \mu)(\rho\mu + \sigma\mu + \mu^2) - \alpha\beta(\mu + \rho + \sigma) > 0$$

$$(\delta + \gamma + \mu)(\mu + \rho + \sigma)(\mu) - \alpha\beta(\mu + \rho + \sigma) > 0$$

$$\mu(\delta + \gamma + \mu) > \alpha\beta \Rightarrow 1 > \frac{\alpha\beta}{\mu(\delta + \gamma + \mu)} \Rightarrow 1 > R_0$$

The solution of $B(x) = 0$ have negative real parts only if $R_0 < 1$. This shows that the Disease Free Equilibrium is Local Asymptotically Stable whenever $R_0 < 1$.
Proved.

RESULTS AND DISCUSSION
Numerical simulations were carried out to graphically illustrate the long term effect of protection on the dynamics of the infectious disease The Table below shows the set of parameter values and the state variables which were used. In order to support the analytical results, graphical representations showing the time graphs of different state variables are provided.

Table 1: Parameters Values for the Model

Description	Parameter	Initial Value	Source
rate of loss of immunity after recovery/vaccination	σ	0.44	Estimated
rate of recovery from infection	γ	0.01	Mushayaba (2011)
typhoid fever induced death rate	δ	0.013	Adetunde (2008)
natural death rate	μ	0.02	Assumed
contact rate	α	0.0011	Source
vaccination rate	ρ	0.33	Lauria et al (2009)
Recruitment rate	β	5	Assumed

Figure 1 is the diagram showing the dynamics of the susceptible population. The Susceptible population decreases as time increases. This decrease may be possibly because of the high rate of recovery due to vaccination, the contact rate also has large impact on the spread of a disease through a population. The higher the rates of contact, the more rapid the spread of the disease.

Fig. 2 shows the graph of infective against time. With high success of vaccination, there is low contact rate and low prevalence rate hence the infected population decreases over time. With low vaccination there is high contact rate and hence a high disease prevalence in the population. This rapid decline of the infected individuals may be due to early detection of the disease
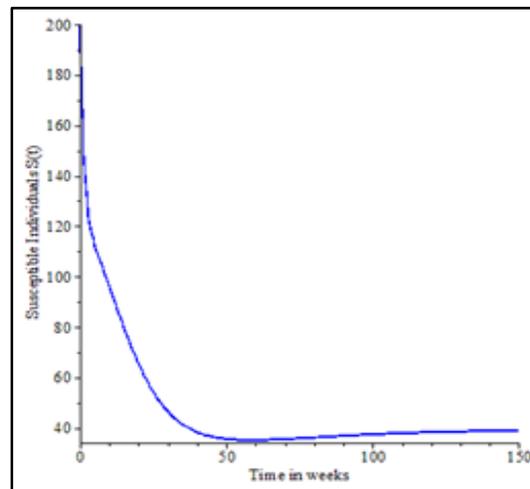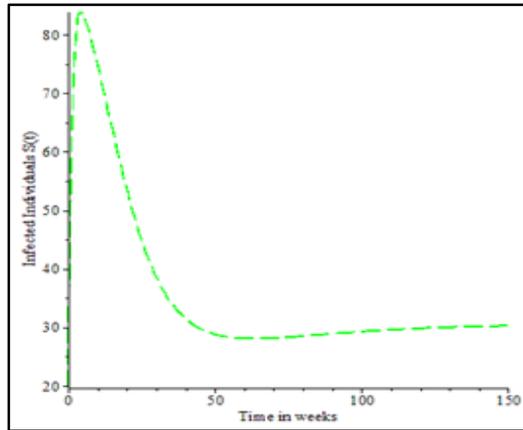

Fig 1: Simulation of susceptible population

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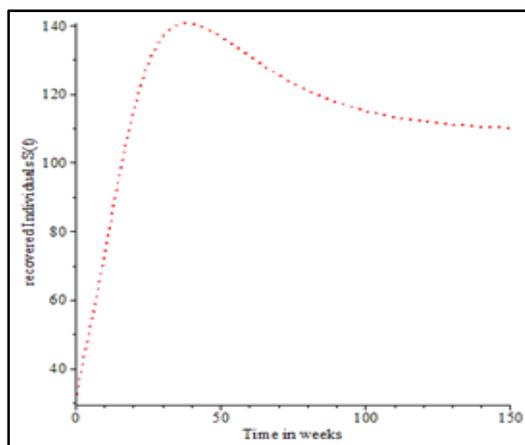
Fig 2: Simulation of infected population

Fig 3: Simulation of recovered population.

In Figure 3 it was observed that as the rate increases, the population of recovered individuals shows some rapid increase due to earlier treatment

*Conclusion***:** We conclude that effective control of infectious disease by vaccination will prevents rapid progression to infection especially in scarce resource setting where treatment is not readily available. Vaccination is effective to prevent disease induced death rate, receiving proper treatment for those already infected among others. Death due to infectious disease decreases with an increase in vaccination because there will be a decrease in the number of infected individuals.

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