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A Saturated Treatment Model for the Transmission Dynamics of Rabies

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ABSTRACT

Rabies is a viral disease that claims about 59 000 lives globally every year. The ignorance of the fact that man can be a carrier of the disease makes every practical and theoretical approach towards the study of the disease a good development. In this work, a mathematical model is designed to incorporate a saturated incidence rate such that the incidence rate is saturated around the infectious agents. The model is studied qualitatively via stability theory of nonlinear differential equations to assess the effects of general awareness, constant vaccination and the saturated treatment on the transmission dynamics of rabies disease. The effective reproduction number is derived and the numerical simulation is carried out to verify the analytical results. It is discovered that while general awareness plays pivotal roles in averting rabies death, multiple control measures have the tendency of driving rabies to extinction.

Keywords: *Rabies, stability theory, reproduction number, simulation*

1. Introduction

Rabies has a long history and was first recorded in China about 556 BC (Ruan, 2017). It is a viral infection that affects the brain of man and animals. Relevant data indicate that rabies occurs in over one hundred and fifty countries and territories worldwide (Asamoah *et al*., 2017). In the United States, the disease spread into human population through animals like foxes, bats, raccoons and skunks whereas, in Africa, Latin America and Asia, the disease gets into human population mainly through dogs. Rabies transmission is mostly attributed to bites or scratches from rabid animals. All mammals are susceptible to rabies infection but dogs are the major carriers of rabies and the cause of several human rabies deaths globally. The rabies virus affects the central nervous system, resulting in infection in the brain and eventually death. Once the rabies symptoms have developed, its mortality is almost sure. The mortality from rabies infection is nearly 100%. In many countries, rabies is neither epidemic nor endemic which makes so many people to be ignorant of it but rabies infection has a catastrophic effect to the degree that whoever is infected with it has less than one in hundred chances of survival.

Several mathematical models have been developed in order to gain deeper understanding of the transmission dynamics and management of rabies. Asamoah *et al*. (2017) developed a model to examine an optimal way of eliminating rabies propagation from dogs into human population by using pre-exposure prophylaxis (vaccination) and post-exposure prophylaxis (treatment) in the presence of public education. Their results indicated that global eradication of deaths from canine rabies by the year 2030 is achievable through continuous vaccination of susceptible dogs and continuous application of both pre and post exposure prophylaxis in man. Also, the impact of immigration, treatment and vaccination on the transmission dynamics of rabies was studied in (Tulu & Koya, 2017; Ibrahim et. al., 2018). It was discovered that rabies tended to disappear if there was effective control of infected immigration dogs and if the vaccination and treatment programmes are considerably improved. Otherwise, there would be a rapid

transmission of rabies in the dog population and the disease would become endemic. Studies on rabies also exist in (Njankou & Nyabadza, 2016; Sharomi & Malik, 2017).

Mathematical modeling is a powerful technique in the epidemiological studies because it offers opportunities to understand the basic mechanisms that affect the transmission of diseases and may suggest intervention strategies. Mathematical models examine the contributory factors to the emergence and dynamics of a disease, such as recovery rates and transmission rates, and predict how the infection will transmit over a period of time. Considerable attempts have been made by the researchers to design realistic mathematical models for investigating the transmission mechanisms of infectious diseases. The dynamics of disease transmission is built around the incidence rate which is the function that describes the mechanism of spread of the diseases. Fundamentally, the incidence function depends on both the susceptible and infectious categories of a population. The bilinear incidence rate is commonly applied to model epidemic diseases (Peter *et al.,* 2018; Akanni & Adediipo; 2018; Muthuri & Malonza, 2018; Rathi et. al., 2018). Bilinear incidence rate is centered on the law of mass action. For example, if the proportion of the susceptible and infectious individuals in a population is represented by S and I respectively, and if α is the effective contact rate, then we assume that the disease spreads with the rate αSI. The mass action law (contact law) is more suitable for infectious diseases such as Ebola, but not for sexually oriented diseases like gonorrhea.

Besides, the bilinear incidence law may not produce appropriate results for a good number of reasons. For instance, the assumption of homogeneous mixing is not always realistic. In this sense, heterogeneity should be incorporated into the population structure such that a model is designed in terms of a basic form of nonlinear transmission because most real life phenomena are nonlinear and are better described by nonlinear equations. The mass incidence function can also be limited by the intervention policies adopted by the public authorities such as quarantine which will definitely affect the contact rate between the susceptible and infectious individuals. Therefore, researchers have argued for nonlinear incidence rates for modeling transmission dynamics of infectious diseases because the proportion of the effective contacts between the susceptible and infectious individuals can be saturated at high infection level as a result of the crowding of infectious individuals or due to the awareness or protective strategies by the susceptible individuals. It is on this note that we incorporated a saturated incidence rate into the model in Ayoade *et al*. (2017) in order to capture the essential dynamics of rabies and to provide a robust mathematical analysis of the disease.

2. Material and Methods

The impact of vaccination on measles epidemiology was studied in Ayoade *et al*. (2017) by using SIR compartmental model as a frame. It was discovered that measles outbreak is doomed to a rapid failure if the vaccination timing, coverage and efficacy were above certain critical threshold. However, a single intervention strategy was adopted in the analysis. Besides, the analysis was based on the bilinear incidence rate while saturated incidence rate was not considered. The present work adopts the model in Ayoade *et al*. (2017) but incorporates multiple interventions and saturated incidence rate to study the transmission dynamics of rabies disease. The transfer diagram for the model is as follows:

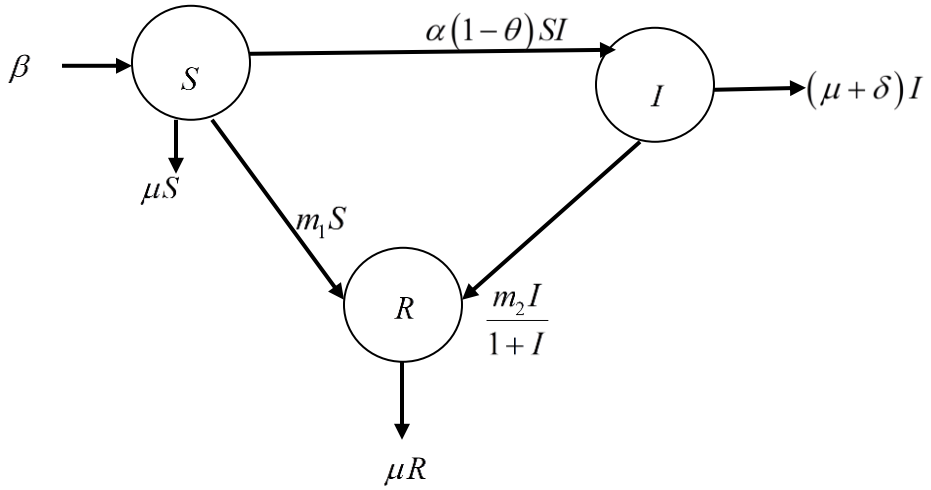

Figure. 1. Flow diagram of the model

The model is made up of compartments of susceptible individuals *S(t),* infectious individuals *I(t)* and recovered individuals *R(t).* Each of the compartments is a function of time which implies that the population in each of them can fluctuate with time. The susceptible population *S(t)* is generated through the coming of individuals as a result of birth at a rate β. The compartment however, reduces by the natural death rate μ, the infection rate α and by the acquired immunity from vaccination at a rate m_1. Certain proportion of the susceptible individuals who are successfully vaccinated would receive immunity against the infection and will move straight to the recovered class since they are immune to the disease. Also, the infection rate α is reduced by θ, which is the rate of awareness of rabies disease.

The population dynamics of the infected class *I(t)* is produced with the incidence rate $\alpha\ (1-\theta)SI$. The class however decreases by the natural death rate μ, by the disease-induced death rate δ and by the successful treatment of rabies patients at a rate m_2. Lastly, the recovered compartment *R(t)* is generated with the successful treatment of rabies patients at a rate m_2 and by the immunity conferred through the introduction of vaccination at a rate m_1. The class however decreases by the natural mortality rate μ. For convenience, we shall write *S(t), I(t)* and *R(t)* as *S*, *I* and *R* respectively. In view of the above transfer diagram and assumptions, we come about the following set of first order nonlinear ordinary differential equations:

$$\frac{dS}{dt} = \beta - \alpha(1-\theta)SI - \mu S - m_1 S \tag{1}$$

$$\frac{dI}{dt} = \alpha(1-\theta)SI - \frac{m_2 I}{1+I} - (\mu+\delta)\mathrm{I} \tag{2}$$

$$\frac{dR}{dt} = m_1 S + \frac{m_2 I}{1+I} - \mu R \tag{3}$$

The numerical values assigned to the parameters to conduct simulations are presented in table 1.

Table 1: parameters description, symbol, values, units and sources

Parameter	Symbol	Value	Unit	Source
Human recruitment rate	β	0.0314	year $^{-1}$	Asamoah *et al.*, (2017)
Death rate due to rabies	δ	1	year $^{-1}$	Asamoah *et al.*, (2017)
Death rate unrelated to rabies	μ	0.0066	year $^{-1}$	Ruan, (2017)
Human vaccination rate	m_1	0.0001	year $^{-1}$	Assumed
Rate of rabies awareness	θ	0.001	year $^{-1}$	Assumed
Rate of rabies treatment	m_2	0.01	year $^{-1}$	Assumed
Contact rate (dog-man)	α	0.00000000229	year $^{-1}$	Assumed

The above parameters are drawn in accordance with the current happenings in most African countries especially Nigeria.

2.1 Basic Features of the Model

A disease model is suitable to conduct a study if it satisfies basic epidemiological conditions. We shall verify whether our model satisfies these conditions or not.

2.1.1 The Invariant Region

The invariant region establishes the domain in which the solutions of the model are both biologically and mathematically meaningful. Hence, we shall show that the region Ω where the model is sensible remains positively invariant and attracting for all $t \geq 0$. That is, all the solutions in Ω remains in Ω for all $t \geq 0$.

Proof 1.

The total human population N at any time t is obtained as

$N(t) = S(t) + I(t) + R(t)$

Since the human population can fluctuate with time then,

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

$$\therefore \frac{dN}{dt} = \beta - \mu N(t) - \delta I \tag{4}$$

The absence of rabies pathogen in eqn. (4) will account for the absence of rabies death i.e. $\delta = 0$. Thus, equation (4) reduces to

$$\frac{dN}{dt} \leq \beta - \mu N(t), \tag{5}$$

so that

$$\frac{dN}{\beta - \mu N(t)} \leq dt \tag{6}$$

Following Birkhoff and Rota's theorem as in Kadaleka (2011) then,

$$\ln(\beta - \mu N(t)) \geq t + c_1 \tag{7}$$

$$\therefore (\beta - \mu N(t)) \geq p_1 e^{-\mu t}, \tag{8}$$

where p_1 is a constant and $p_1 = e^{c_1}$

At initial time, $t = 0$ and $N(0) = N_0$. Then, put $t = 0$ and $N(0) = N_0$ in inequality (8) to obtain

$(\beta - \mu N_0) \geq p_1$.

Substituting for p_1 in inequality (8) to obtain

$$\beta - \mu N(t) \geq (\beta - \mu N_0) e^{-\mu t} \tag{9}$$

Rearranging inequality (9) in terms of $N(t)$ to obtain

$$N(t) \leq \frac{\beta}{\mu} - \left(\frac{\beta - \mu N_0}{\mu}\right) e^{-\mu t} \tag{10}$$

As $t \to \infty$ in inequality (10), then the total human population $N(t)$ reduces to

$$N(t) \leq \frac{\beta}{\mu} \tag{11}$$

In this regards, all the feasible solutions for the living population in the system (1) – (3) exist in the region

$$\Omega = \left\{(S, I, R) \in \Re_+^3, N(t) \leq \frac{\beta}{\mu}\right\} \tag{12}$$

The above is a positive invariant set of the model which shows that the model is both biologically and mathematically meaningful in the domain Ω. Hence, every analysis of the dynamics of the flow generated by the model can be considered in Ω.

2.2.2 The Positivity of Solution

The positivity of solution establishes the non-negativity of solutions of the model under study. Since the model monitors living population, it is assumed that all the variables and parameters of the model are positive for all $t > 0$ and we expect positive solutions for the model.

Theorem 1: Suppose the initial values for the state variables are all positive i.e. $S(0) > 0$, $I(0) > 0$ and $R(0) > 0$ then the solution $\{S(t), I(t), R(t)\}$ of the model equations (1) – (3) are all positive for all $t > 0$. i.e. the model has positive solutions for all the state variables as long as the initial values of the state variables are positive.

Proof 2.

The above theorem shall be proved for eqn. (1) – (3). From eqn. (1),

$$\frac{dS}{dt} = \beta - \alpha(1-\theta)SI - \mu S - m_1 S$$

$$\frac{dS}{dt} \geq -(\mu + m_1)\mathrm{S} \tag{13}$$

Separating the variables in the above and integrate

$$\ln S \geq -(\mu + m_1)t + c_2 \tag{14}$$

$$S(t) \geq p_2\, e^{-(\mu+m_1)t}\ , \tag{15}$$

where $p_2 = e^{c_2}$. At initial time, t= 0 and, on substitution into inequality (15),

$S(0) \geq p_2$

Thus, inequality (15) becomes

$$S(t) \geq S(0)\, e^{-(\mu+m_1)t} \tag{16}$$

Repeating the same process for the second and third equations in the system (1) - (3) respectively, the following results are obtained:

$$I(t) \geq I(0)\, e^{-(\mu+\delta)t} \tag{17}$$

$$R(t) \geq R(0)e^{-\mu t} \tag{18}$$

Since $e^q > 0$ for all real values of q then it is sufficient to conclude that the solutions for each state variable $S(t), I(t)$ and $R(t)$of the model are positive for all $t > 0$. Having satisfied the basic features of the epidemiological models, the model equations (1) – (3) were suitable to study the dynamics of rabies disease.

3.0 Theory/Calculation

Since the model satisfies the basic biological conditions, it is suitable to conduct the study at hand. Hence, we shall perform equilibrium analysis, stability analysis and derive the effective reproduction number before conducting numerical simulations.

3.1 Equilibrium Analysis

Equilibrium is attained in disease models when the rate of change of the first derivative is zero. The solutions obtained at this point are called equilibrium solutions. Equilibrium is of two types in epidemiology: equilibrium attained when a society is free pathogen known as the disease-free equilibrium and the equilibrium attained when a society is under the threat of pathogen known as the endemic equilibrium.

3.1.1 Existence of Disease-Free Equilibrium, D_0

When the entire population is free from rabies pathogen then, nobody is infected with rabies and nobody recovers from rabies infections hence, $I = 0$ but $R \neq 0$ in this case unlike in many situations. The reason is that recovery is not only as result of successful treatment after infections but also as a result of immunity acquired through vaccination which has been put in place as a measure should rabies breaks out in the population. Hence, R is a function of m_1 and m_2 where m_1 has nothing to do with treatment after infection. Using the condition $I = 0$, $R \neq 0$ to solve eqns (1) – (3) then,

$$D_0 = \left(\frac{\beta}{(\mu+m_1)}, 0, \frac{m_1\beta}{\mu(\mu+m_1)}\right) \tag{19}$$

3.2 The Effective Reproductive Number, R_F

It is a non-dimensional quantity that measures the transmission potential of an infectious disease in a population where intervention strategies are on ground. How many people will an infectious individual infects should he get into the population of susceptible individuals where interventions are on ground? The answer to the question can be provided by the effective reproduction number. If the infectious individual is able to infect, say, three persons, it means $R_F = 3 > 1$ and the disease will spread in the population. On the other hand, if the infectious individual could not infect a single individual in the population, say, 0.3 person, it means $R_F = 0.3 < 1$ and the disease will not spread. Hence, while rabies will break out in the population if $R_F > 1$, it will not break out as long as $R_F < 1$. We shall obtain the effective reproduction number for our model by solving eqn. (2) and use the result to investigate whether the disease will spread or not.

From eqn. (2)

$\alpha(1-\theta)SI - \frac{m_2 I}{1+I} - (\mu+\delta)\mathrm{I} = 0$ and,

$I\left[\frac{\alpha\beta(1-\theta)}{(\mu+m_1)}I - (\mu+\delta)I + \frac{\alpha\beta(1-\theta)}{(\mu+m_1)} - (m_2+\mu+\delta)\right] = 0$

$I = 0$ corresponds to the disease-free equilibrium and, following the same approach as in (Xiao & Ruan, 2007; Bakare *et al.*, 2017), the effective reproduction number is

$$R_F = \left[\frac{\alpha\beta(1-\theta)}{(\mu+m_1)(m_2+\mu+\delta)}\right]. \tag{20}$$

If all the interventions are reduced to zero, the effective reproduction number reduces to the basic reproduction number given as

$$R_0 = \left[\frac{\alpha\beta}{\mu(\mu+\delta)}\right]. \tag{21}$$

3.3 Local Stability of the Disease-Free Equilibrium

The local stability analysis of the disease-free equilibrium shall be conducted via the linearization approach.

Theorem 2: The disease-free equilibrium of the system of eqns (1) – (3) is locally asymptotically stable if $R_F < 1$ and is unstable if otherwise i.e. if $R_F > 1$.

The local stability of the disease-free equilibrium implies that the disease will not break out in the population while the instability of the disease-free equilibrium implies that the outbreak of the disease is imminent and inevitable. The existence of stability or instability of the disease-free equilibrium of the model shall be established by solving the characteristic equation of the Jacobian matrix of the model at the disease-free equilibrium, D_0 i.e. eqn. (19). If all the eigenvalues of the characteristic equation are less than zero then the disease-free equilibrium of the model is locally asymptotically stable but if otherwise, the disease-free equilibrium of the model is unstable.

Proof 3.

The Jacobian matrix of the model system (1) – (3) evaluated at the disease-free equilibrium i.e. eqn. (19) is given as

$$J_{disease-free} = \begin{pmatrix} -(\mu + m_1) & -\alpha\frac{(1-\theta)\beta}{(\mu+m_1)} & 0 \\ 0 & \alpha\frac{(1-\theta)\beta}{(\mu+m_1)} - (m_2 + \delta + \mu) & 0 \\ m_1 & m_2 & -\mu \end{pmatrix} \quad (22)$$

The characteristic equation of the above matrix has the eigenvalues

$\lambda_1 = -\mu,\ \lambda_2 = -(\mu + m_1)$ and $\lambda_3 = \alpha\frac{(1-\theta)\beta}{(\mu+m_1)} - (m_2 + \delta + \mu)$

Obviously, all the eigenvalues are less than zero if $\alpha\frac{(1-\theta)\beta}{(\mu+m_1)} < (m_2 + \delta + \mu)$

Hence, the disease-free equilibrium of the model is locally asymptotically stable if $\alpha\frac{(1-\theta)\beta}{(\mu+m_1)} < (m_2 + \delta + \mu)$ otherwise, it is unstable. The inequality $\alpha\frac{(1-\theta)\beta}{(\mu+m_1)} < (m_2 + \delta + \mu)$ is true if $R_F < 1$

3.4 Global Stability of the Disease-Free Equilibrium

Local stability of a system investigates what happens to the equilibrium of the system on a small scale. The equilibrium of a system is restored only if some restrictions put the system around the equilibrium for local stability. Global stability, on the other hand, establishes what happens to the equilibrium of a system on a large scale when there is no restriction on the initial conditions of the model variables. The equilibrium is always restored and the solutions of the model approach the equilibrium for all initial conditions under global stability. While local stability analysis of a system restricts the analysis of the system to the region near the equilibrium point, global stability analysis of the system enables the analysis to be extended beyond only small region near the equilibrium. The global stability analysis of a model can be conducted by a number of methods which include among others the Lyapunov theorem and Castillo-Chavez global stability theorems. However, the former shall be used in this work.

Theorem 3: If $R_F < 1$, the disease-free equilibrium, D_0 of the system is globally asymptotically stable in Ω.

Proof 4:

β is dropped in eqn. (1) since it does not contain a variable which reduces eqns (1) – (3) to

$$\left.\begin{aligned} \frac{dS}{dt} &= -\alpha(1-\theta)IS - \mu S - m_1 S \\ \frac{dI}{dt} &= \alpha(1-\theta)IS - \frac{m_2 I}{1+I} - \delta I - \mu I \\ \frac{dR}{dt} &= m_1 S + \frac{m_2 I}{1+I} - \mu R \end{aligned}\right\} \tag{23}$$

Given the linear Lyapunov function L as

$$L(S,I,R) = b_1 S + b_2 I + b_3 R \tag{24}$$

where $b_1 > 0,\ b_2 > 0,\ b_3 > 0, b_1 - b_2 > 0$ and $b_2 - b_3 > 0$ (25)

The derivative of L w.r.t t is

$$\frac{dL}{dt} = b_1 \frac{dS}{dt} + b_2 \frac{dI}{dt} + b_3 \frac{dR}{dt} \tag{26}$$

Our aim is to show that $\frac{dL}{dt} < 0 \in \Omega$ to establish that $R_F < 1$

This is the necessary and sufficient condition for the disease-free equilibrium to be globally asymptotically stable.

Substituting eqn. (23) into eqn. (26) to obtain

$$\begin{aligned}\frac{dL}{dt} &= b_1(-\alpha(1-\theta)IS - \mu S - m_1 S) \\ &+b_2\left[\alpha(1-\theta)IS - \frac{m_2 I}{1+I} - \delta I - \mu I\right] \\ &+b_3\left[m_1 S + \frac{m_2 I}{1+I} - \mu R\right] \\ &= -\left[\alpha(1-\theta)(b_1 - b_2)\mathrm{I} + b_1(\mu + m_1) - b_3 m_1\right]S \\ &-\left[\alpha(1-\theta)(b_1 - b_2)\mathrm{S} + \frac{m_2}{1+I}(b_2 - b_3) + (\delta+\mu)\right]I \\ &-\left[b_3\mu\right]R\end{aligned} \tag{27}$$

Expressing $(1-\theta)$ in terms of R_F in eqn. (20) and put the result in eqn. (27) to have

$$\begin{aligned}\frac{dL}{dt} &= -\left\{\frac{1}{\beta}R_F(\mu+m_1)(m_2+\mu+\delta)(b_1-b_2)I + b_1(\mu+m_1) - b_3 m_1\right\}S \\ &-\left\{\frac{1}{\beta}R_F(\mu+m_1)(m_2+\mu+\delta)(b_1-b_2)S + \frac{m_2}{1+I}(b_2-b_3) + (\delta+\mu)\right\}I \\ &-\{b_3\mu\}R\end{aligned} \tag{28}$$

Since the system monitors living population then all the parameters as well as variables are non-negative. Also $R_F < 1$ does not imply negative value for R_F. Hence, from eqn. (28) and by following the conditions in inequality (25)

$$\left.\begin{aligned}&-\left\{\frac{1}{\beta}R_F(\mu+m_1)(m_2+\mu+\delta)(b_1-b_2)I + b_1(\mu+m_1) - b_3 m_1\right\} < 0 \\ &-\left\{\frac{1}{\beta}R_F(\mu+m_1)(m_2+\mu+\delta)(b_1-b_2)S + \frac{m_2}{1+I}(b_2-b_3) + (\delta+\mu)\right\} < 0 \\ &-\{b_3\mu\} < 0\end{aligned}\right\} \tag{29}$$

Therefore, inequality (29) establishes that $\frac{dL}{dt} < 0$ in Ω as required to be proved. Moreover, $\frac{dL}{dt} = 0$ if $S = 0, I = 0, R = 0$ in eqn. (28). Hence, the maximum invariant set in

$\left[(S,I,R):\frac{dL}{dt}=0\right]$ is the singleton D^0 . By LaSalle's invariance principle as in Bowong *et al.* (2011), D^0 is globally asymptotically stable in the invariant region Ω where D^0 is the disease-free equilibrium of the model

4.0 Results

We shall verify the stability condition of the disease-free equilibrium that was proved in subsection 3.3. It was stated that the disease-free equilibrium of the model is locally asymptotically stable if $\alpha\frac{(1-\theta)\beta}{(\mu+m_1)} < (m_2+\delta+\mu)$. By using the parameters values in table 1, the numerical value of $\alpha\frac{(1-\theta)\beta}{(\mu+m_1)}$ is 1.07×10^{-11} while the numerical value of $(m_2+\delta+\mu)$ is 1.02. Hence, the inequality $\alpha\frac{(1-\theta)\beta}{(\mu+m_1)} < (m_2+\delta+\mu)$ is true and the disease-free equilibrium of the model is locally asymptotically stable. In order to investigate the expected number of new cases of rabies in the population when the interventions are not on ground and when they are on ground, the values of the transmission and intervention parameters in table 1 shall be varied to examine the effect of changes in these parameters on the threshold quantities bearing in mind that the analytical results for R_F and R_0 are given by equations (20) and (21) respectively. The results of the analysis are presented in table 2.

Table 2: The Impact of variations in the values α, θ, m_1 and m_2 on R_0 and R_F

α	β	μ	δ	R_0	θ	m_1	m_2	R_F
0.00000000229	0.0314	0.0066	1	$1.08^*\ 10^{-8}$	0.001	0.0001	0.01	$1.01^*\ 10^{-11}$
0.000000229	0.0314	0.0066	1	$1.08^*\ 10^{-6}$	0.01	0.001	0.03	$9.13^*\ 10^{-9}$
0.0000229	0.0314	0.0066	1	$1.08^*\ 10^{-4}$	0.05	0.005	0.08	$2.85^*\ 10^{-6}$
0.00229	0.0314	0.0066	1	$1.08^*\ 10^{-2}$	0.1	0.01	0.1	$3.91^*\ 10^{-4}$
0.229	0.0314	0.0066	1	1.08	0.5	0.05	0.5	0.04
0.4	0.314	0.0066	1	1.89	0.8	0.1	0.8	0.05
0.5	0.314	0.0066	1	2.36	0.9	0.5	0.9	0.01

The disease-free equilibrium point and the endemic equilibrium point are shown to be locally and globally asymptotically stable. The interpretation of this is that globally, the outbreak of rabies could be averted under the conditions imposed by the model. As regards simulation results, from Table 2, it is observed that increase in the disease transmission parameter α has negative influence on both threshold quantities R_0 and R_F though the influence on R_F is minimised by the accompanying increase in the intervention parameters

θ, m_1 and m_2. The increase in the disease transmission rate up to certain level (Row 6 downward) results in outbreak of rabies when there is no control (i.e. R_0) whereas, the outbreak is well inhibited with the presence of interventions (i.e. R_F). The interpretation of the result is that outbreak of rabies is possible if there is negligence towards its prevention and control. The negligence has accounted for many rabies deaths in Africa where ignorance co-exists with poverty and poor healthcare delivery. The graphical illustrations are presented in Figures 2 and 3 to support Table 2 and to show the effects of the parameters considered on the overall transmission dynamics.

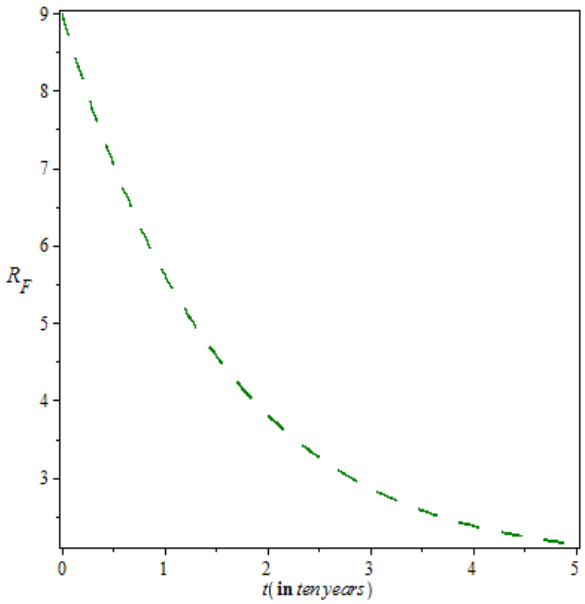

Fig 2. Rabies incidence with interventions

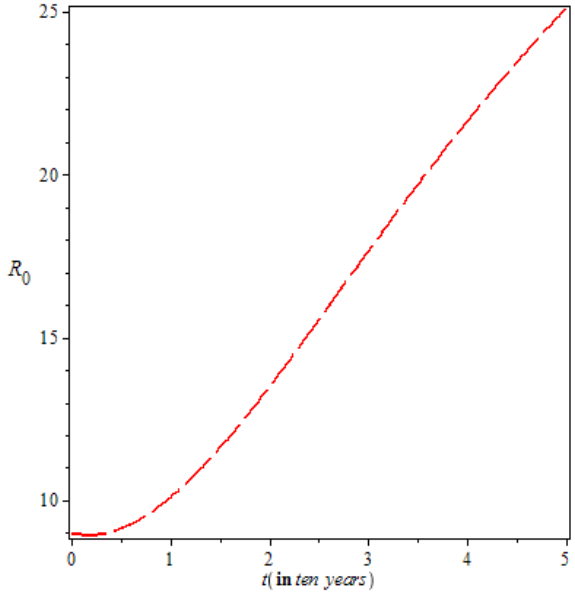

Fig 3. Rabies incidence without interventions

5.0 Conclusion

In the study, we formulate a simple deterministic compartmental mathematical model with saturated incidence rate to assess the possibility of reducing rabies death to zero. The basic properties of the epidemic models in terms of the boundedness and positivity of solutions are investigated for our model and we establish that the model is positive for all positive values of initial conditions. Besides, in the absence of rabies, the population tends to the carrying capacity. We conduct the equilibrium analysis, stability analysis and derive the reproduction numbers. We establish that both the disease-free equilibrium point and the endemic equilibrium point are locally and globally asymptotically stable. We carry out numerical simulations to verify the theoretical results and the results of the simulations are discussed. For a disease like rabies whose tendency of defying treatment is almost 100% (Ruan, 2017), we conclude that general awareness together with the availability of mechanism to detect infected individuals at the latent stage is the basis for global eradication of the disease.

References

Akanni, J. O., & Adediipo, A. D. (2018). Sensitivity analysis of the dynamical transmission of lassa fever. *Asian Research Journal of Mathematics*, *9*(3), 1-11.

Asamoah, J. K. K., Oduro, F. T., Bonyah, E. & Seidu, B. (2017). Modelling of rabbies transmission dynamics using optimal control analysis, Hindawi *Journal of Applied Mathematics*. http://doi.org/10.1155/2017/2451237

Ayoade, A. A., Odetunde, O., & Kazeem, A. B. (2017). Modelling the impact of vaccination on measles epidemiology. *Abacus. Journal of the Mathematical Association of Nigeria* (Mathematics Science Series), *44*(2), 46-53.

Bakare, E. A., Are, E. B., & Samuel, O. (2017). A SIR epidemic model with constant vaccination and saturated treatment. *Abacus. Journal of the Mathematical Association of Nigeria* (Mathematics Science Series), *44*(2), 259-270.

Bowon, S., Tewa, J. J., & Kamgang, J. C. (2011). Stability analysis of the transmission dynamics of tuberculosis model. *World Journal of Modelling and Simulation*, *7*(2), 83-100.

Ibrahim, M., Ayoade, A., Peter, O., & Oguntolu, F. (2018). On The Global Stability Of Cholera Model With Prevention And Control. Malaysian Journal Of Computing, 3(1), 28-36.

Kadaleka, S. (2011). *Assessing the effects of nutrition and treatment in cholera dynamics: The case of Malawi*. M. Sc. Dissertation, University of Der es Salaam

Muthuri, G. G., & Malonza, D. M. (2018). Mathematiccal modelling of TB-HIV co-infection, case study of Tigania West sub County, Kenya. *Journal of Advances in Mathematics and Computer Science*, *27*(5), 1-18.

Njankou, S. D. D., & Malik, T. (2017). An optimal control model for ebola virus disease. *Journal of Biological System*, *24*(1), 29-49.

Peter, O. J., Afolabi, O. A., Victor, A. A., Akpan, C. E., & Oguntolu, F. A. (2018). Mathematical model for control of measles. *Journal of Applied Science and Environmental Management*, *22*(4), 571-576.

Rathi, S., Cik Soh, S., & Akbarally, A. (2018). Coefficient Bounds For A Certain Subclass Of Close-To-Convex Functions Associated With The Koebe Function. Malaysian Journal Of Computing, 3(2), 172-177.

Ruan, S. (2017). Modelling the transmission dynamics and control of rabies in China. *Mathematical Biosciences*, *286*, 65-93.

Sharomi, O., & Malik, T. (2017). Optimal control in epidemiology. *Annals Operation Research*, *25*(1-2), 55-71.

Tulu, A. M., & Koya, P. R. (2017). The impact of infective immigrants on spread of dog rabies. *American Journal of Applied Mathematics*, *5*(3), 68-77.

Xiao, D., & Ruan, S. (2007). Global analysis of an epidemic model with nonmonotonic incidence rate. *Mathematical Biosciences*, *208*, 419-429.