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A Mathematical Model of Rabies Transmission Dynamics in Dogs Incorporating Public Health Education as a Control Strategy -A Case Study of Makueni County

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Authors' contributions

This work was carried out in collaboration between both authors. Author IC designed the study and wrote the first draft of the manuscript. All authors managed literature searches. Author JSM performed the Mathematical analysis of the study and the simulations. Both authors read and approved the final manuscript.

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Abstract

Rabies is a zoonotic viral disease that affects all mammals including human beings. Dogs are responsible for 99% of human rabies cases and the disease is always fatal once the symptoms appear. In Kenya the disease is still endemic despite the fact that there are efficient vaccines for controlling the disease. In this project, we developed SIRS mathematical model using a system of ordinary differential equations from the model to study the transmission dynamics of rabies virus in dogs using public health education as a control strategy. The reproduction number R_0 was calculated using the Next Generation Matrix. Both disease free and endemics equilibrium points were determined and their stability analysis performed. From the stability analysis results it was found out that the disease free equilibrium point is both locally and globally asymptotically stable when $R_0 < 1$ and the endemic equilibrium point is both locally and globally asymptotically stable when $R_0 > 1$. Numerical simulations done using Matlab indicated that education of the public on administration of both pre and post exposure vaccines to dogs and responsible dog ownership

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leads to a decrease in the numbers of rabies virus infected dogs which shows that public health education is an efficient means for controlling rabies.

Keywords: Rabies; reproduction number; stability; numerical simulation.

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1 Introduction

Rabies is an infectious zoonotic viral disease that pose a great threat to public health in the whole world [1]. It is spread through the scratch or bite of a rabid animal as reported by [2]. The main carrier of the virus remains to be dogs and are responsible for most human rabies death worldwide. Rabies virus infects the central nervous system leading to the inflammation of brain, paralysis and death. Once symptoms of the disease develop, rabies is always fatal.

According to [3], Rabies virus affects both wildlife and domestic mammals such as foxes ,skunks, raccoons, bats, cats ,dogs and human being. According to [1], the time between exposure to the virus and when symptoms start showing up varies depending on the location of the bite,intensity of wound and immunity of affected animal but in most cases its usually four to ten weeks. [3] points out that rabies virus symptoms resemble those of flu-like infection,when the disease progress the affected individual may experience discomfort at the sight of exposure,cerebral dysfunction, anxiety, confusion and even abnormal behaviors. [1] observed that rabies virus may cause the paralysis of the muscles from the point of bite or scratch which leads to coma and then death.

In Kenya,the first rabies case in a dog was reported in the outskirts of Nairobi in 1912,and a human case was reported in 1928, since then it has remained to be a public health problem [4]. Rabies is estimated to cause 2,000 human deaths per year and its placed among the top five animal diseases that affects people in the country [1]. Although there is effective vaccine for controlling rabies ,the disease is still endemic in Kenya as well as in other developing countries. The poor and vulnerable individuals are the most affected by the disease especially children below 15 years because of their free interactions with dogs [5].

Mathematical modelling serves as a very important tool in analysing and understanding epidemiological characteristics of infectious diseases and helps proving useful control methods for such diseases. Many scholars have used several models to help understand dynamics of rabies in wildlife like [6],[7], [8], [9] and [10]

Although there are several mathematical models, that describe various aspects of rabies in wildlife, domestic animals especially dogs which is main carrier of rabies virus in Kenya and even transmission dynamics between dogs and human beings incorporating mass vaccination, pre-exposure prophylaxis and post exposure prophylaxis as possible ways of eradicating rabies, the impact of public health education has not yet been considered which marks the motivation behind this study.

This study therefore aim at investigating the dynamics of rabies in which the impact of public health education is incorporated as a possible way of eradicating rabies among dog population. A case study of Makueni county.

2 Model Formulation

In this model, dog population of size N(t) is considered and it is divided into three classes. These are Susceptible class-S(t) which include health dogs but have probability of contracting the disease,

the Infected class- I(t) is made up of the dogs which have contracted the rabies virus and are infectious and Recovered class-R(t) includes dogs that have recovered from the virus exposure due to administration of pre-exposure vaccine which is as result of public education. Dogs are recruited to the susceptible class S(t) at the rate μ through births. We let $0 < \alpha < 1$ to be the rate at which public is educated on the administration of both pre-exposure vaccination and post-exposure vaccination of dogs, responsible dog ownership and creation of awareness on the impact of rabies virus on human health. Therefore susceptible dogs move to the infected class-I(t) at the reduced rate of infection $(1-\alpha)\beta SI$, where β is the probability of being with rabies virus due to interactions between susceptible dogs and infected dogs. Dogs in all classes experience natural death at the rate ρ and in addition, infected dogs die at the rate σ due to the rabies disease. When public is educated, a portion of susceptible dogs αS move directly to the recovered class-R(t) due to administration of pre-exposure vaccination because public education involves creation of awareness to the public, administration of both pre-exposure and post-exposure vaccines and responsible dog ownership. Recovered dogs become susceptible at the rate ϵ as the impact of public education dies off with time.

From the above description we have the following assumptions and flow diagram;

Assumptions:

i Dogs are the only carriers of rabies and so the susceptible compartment consist of dogs that are likely to be exposed to rabies virus.

ii Once a dog is infected by rabies it dies because immediately the symptoms appear the disease is always fatal.

iii Once the public is educated on the dangers associated with rabies,they take precaution measures such as provide pre and post exposure vaccines and also practice responsible dog ownership and as such the recovered compartment consists of dogs that recover immediately precaution measures are put in place.

iv A portion of recovered dogs is recruited to the susceptible compartment because of their reduced immunity over rabies.

v The precaution measure taken after the public is educated include administration of both pre and post exposure vaccine and responsible dog ownership.

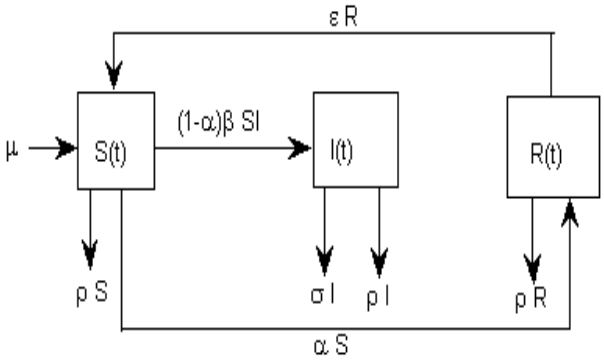

Fig. 1. Flow diagram

From figure 1 the following equations are developed:

$$\begin{aligned} \frac{dS}{dt} &= \mu + \epsilon R - (1-\alpha)\beta SI - \rho S - \alpha\ S \\ \frac{dI}{dt} &= (1-\alpha)\beta SI - \sigma I - \rho I \\ \frac{dR}{dt} &= \alpha S - \rho R - \epsilon R \end{aligned} \tag{2.1}$$

3 Model Analysis

Since the system (2.1) describes Dog population, all the solutions of state variable with non-negative initial conditions are non-negative $\forall\ t > 0$ and they are bounded in the feasible region $\Gamma = \{(S, I, R) \in \mathbb{R}^3_+; N \leq \frac{\mu}{\rho}\}$

3.1 Existence of Equilibrium points

In order to determine Disease free equilibrium point (DFE) E^0, we equate the right-hand sides of the equations of system 2.1 to zero and substitute $(S = S^0, I = 0, R = 0)$ for S,I,R, to get $E^0 = (\frac{\mu}{\rho+\alpha}, 0, 0)$

Now we obtain Endemic equilibrium point (EE) $E^*(S^*, I^*, R^*)$ by setting the right-hand sides of the equations of system 2.1 to zero and substituting for S,I and R with S^*, I^* and R^*. Thus

$$E^* = \begin{pmatrix} S^* \\ I^* \\ R^* \end{pmatrix} = \begin{pmatrix} \frac{\sigma+\rho}{(1-\alpha)\beta} \\ \frac{(\rho+\epsilon)(1-\alpha)\beta\mu+(\sigma+\rho)\alpha\epsilon-(\rho+\epsilon)(\rho+\alpha)(\sigma+\rho)}{(\sigma+\rho)(\rho+\epsilon)(1-\alpha)\beta} \\ \frac{(\sigma+\rho)\alpha}{(\rho+\epsilon)(1-\alpha)\beta} \end{pmatrix}$$

3.2 The basic reproduction number

Basic reproduction number is R_0 is a dimensionless number that is defined as the expected number of secondary cases produced by a single infected individual in a completely susceptible population.

The Next generation Matrix method by [11] is used to find R_0.nThe Next generation matrix is given by $G = FV^{-1}$ where F and V are Jacobian matrices of f_j and v_j vectors respectively at E^0, where f_j is the rate of appearance of new infections in class j and v_j is the rate of transfer of dogs into and out of class j. The infected dog population is captured in the following equation.

$$\frac{dI}{dt} = (1-\alpha)\beta SI - (\sigma+\rho)I \tag{3.1}$$

Clearly equation (3.1) can be expressed as

$\frac{dI}{dt} = f_j - v_j$ where $f_j = (1-\alpha)\beta SI$ and $v_j = (\sigma+\rho)I$

By definition

$F = \frac{(1-\alpha)\beta\mu}{\rho+\alpha}$ and $V = (\sigma+\rho)$

since $S = S^0 = \frac{\mu}{\rho+\alpha}$

hence

$FV^{-1} = \frac{(1-\alpha)\beta\mu}{(\rho+\alpha)(\sigma+\rho)}$

Since FV^{-1} is a 1×1 matrix, in view of the definition of R_0, we have

$R_0 = \frac{(1-\alpha)\beta\mu}{(\rho+\alpha)(\sigma+\rho)}$

4 Stability Analysis

4.1 Local stability of the disease free equilibrium point

The DFE point of the model is the steady state solution in the absence of the infection. Therefore local stability analysis is presented in the theorem (1) below.

Theorem 1. *The DFE of the system (2.1) is Locally asymptotically stable whenever* $R_0 < 1$

Proof. The Jacobian of the system (2.1) is given as

$$J = \begin{bmatrix} -(1-\alpha)\beta I - (\rho+\alpha) & -(1-\alpha)\beta S & \epsilon \\ (1-\alpha)\beta I & (1-\alpha)\beta S - (\sigma+\rho) & 0 \\ \alpha & 0 & -(\rho+\epsilon) \end{bmatrix} \tag{4.1}$$

We evaluate the Jacobian matrix (4.1) at E^0 to get

$$J(E^0) = \begin{bmatrix} -(\rho+\alpha) & -\frac{(1-\alpha)\beta\mu}{\rho+\alpha} & \epsilon \\ 0 & \frac{(1-\alpha)\beta\mu}{\rho+\alpha} - (\sigma+\rho) & 0 \\ \alpha & 0 & -(\rho+\epsilon) \end{bmatrix}$$

The characteristic equation of $J(E^0)$ is given by

$$[-(\rho+\alpha)-\lambda][(\frac{(1-\alpha)\beta\mu}{\rho+\alpha} - (\sigma+\rho)) - \lambda][-(\rho+\epsilon)-\lambda] - [(\frac{(1-\alpha)\beta\mu}{\rho+\alpha} - (\sigma+\rho)) - \lambda]\alpha\epsilon = 0 \tag{4.2}$$

Upon expansion of equation (4.2), we obtain

$[(\frac{(1-\alpha)\beta\mu}{\rho+\alpha} - (\sigma+\rho)) - \lambda]\{\lambda^2 + [(\rho+\alpha)+(\rho+\epsilon)]\lambda + \rho^2 + \rho\epsilon + \rho\alpha\} = 0$
Implying that

$$\frac{(1-\alpha)\beta\mu}{\rho+\alpha} - (\sigma+\rho) - \lambda = 0 \tag{4.3}$$

or

$$\lambda^2 + [(\rho+\alpha)+(\rho+\epsilon)]\lambda + \rho^2 + \rho\epsilon + \rho\alpha = 0 \tag{4.4}$$

Clearly one of the eigenvalues is given by equation (4.3), that is

$\lambda = \left(\frac{(1-\alpha)\beta\mu}{\rho+\alpha} - (\sigma+\rho)\right) < 0$ for $R_0 < 1$

And other eigenvalues are obtained from equation (4.4). Using the Routh Hurwitz criterion for the second order polynomial, we investigate the signs of eigenvalues given by equation (4.4).

Now we begin the investigation by writing equation (4.4) as

$$\lambda^2 + a\lambda + b = 0 \tag{4.5}$$

where
$a = (\rho + \alpha) + (\rho + \epsilon)$
$b = \rho^2 + \rho\epsilon + \rho\alpha$

Clearly $a > 0$ $b > 0$ and $ab > 0$. Hence the Routh Hurwitz criterion for the second order polynomial is satisfied, implying that the roots of equation (4.5) lie in the left half of the complex plane. That is, the other eigenvalues are negative. Therefore the DFE is locally asymptotically stable whenever $R_0 < 1$ □

4.2 Global stability of the disease free equilibrium point

In this section the global stability of the disease free equilibrium point of the system (2.1) is investigated using Theorem (2).

Theorem 2. *E^0 is globally asymptotically stable provided that $R_0 < 1$*

Proof. Consider the following Lyapunov function

$$V(S, I) = I \tag{4.6}$$

Differentiating equation (4.6) with respect to t gives;

$$\frac{dV}{dt} = \frac{dI}{dt} \tag{4.7}$$

In view of the second equation of system (2.1), equation (4.7) assumes the form

$\frac{dV}{dt} = (1 - \alpha)\beta SI - (\sigma + \rho)I$

Since $R_0 = \frac{(1-\alpha)\beta\mu}{(\rho+\alpha)(\sigma+\rho)}$ we have $(1 - \alpha)\beta = \frac{R_0(\rho+\alpha)(\sigma+\rho)}{\mu}$

Therefore

$\frac{dV}{dt} = \frac{R_0(\rho+\alpha)(\sigma+\rho)SI}{\mu} - (\sigma + \rho)I$

or

$\frac{dV}{dt} \leq (\sigma + \rho)(R_0 - 1)I$

for

$S < S^0 = \frac{\mu}{(\rho+\alpha)}$

$\frac{dV}{dt} \leq 0$ for $R_0 < 1$ and $\frac{dV}{dt} = 0$ if and only if I=0. This implies that the only trajectory of the system (2.1) on which $\frac{dV}{dt} = 0$ is E^0. Therefore by Lasalle's invariance principle, E^0 is globally asymptotically stable in Γ. □

4.3 Local stability of endemic equilibrium point $E^*(S^*, I^*, R^*)$

The persistence of a disease in a population implies that the disease is endemic. The study of the stability analysis of the endemic equilibrium point is done using the theorem below.

Theorem 3. *The endemic equilibrium E^* of the system (2.1) locally asymptotically stable whenever $R_0 > 1$*

Proof. To prove this theorem, we express Jacobian matrix (4.1) at endemic equilibrium E^* to obtain

$$J(E^*) = \begin{bmatrix} -((1-\alpha)\beta I^* + (\rho+\alpha)) & -(1-\alpha)\beta S^* & \epsilon \\ (1-\alpha)\beta I^* & (1-\alpha)\beta S^* - (\sigma+\rho) & 0 \\ \alpha & 0 & -(\rho+\epsilon) \end{bmatrix}$$

or

$$J(E^*) = \begin{bmatrix} -\left(\frac{(\rho+\epsilon)(1-\alpha)\beta\mu+(\sigma+\rho)\alpha\epsilon-(\rho+\epsilon)(\rho+\alpha)(\sigma+\rho)}{(\sigma+\rho)(\rho+\epsilon)} + (\rho+\alpha)\right) & -(\sigma+\rho) & \epsilon \\ \frac{(\rho+\epsilon)(1-\alpha)\beta\mu+(\sigma+\rho)\alpha\epsilon-(\rho+\epsilon)(\rho+\alpha)(\sigma+\rho)}{(\sigma+\rho)(\rho+\epsilon)} & 0 & 0 \\ \alpha & 0 & -(\rho+\epsilon) \end{bmatrix}$$

The eigenvalues of $J(E^*)$ are given by $|J(E^*) - I\lambda| = 0$, that is

$$\begin{vmatrix} -\left(\frac{(\rho+\epsilon)(1-\alpha)\beta\mu+(\sigma+\rho)\alpha\epsilon-(\rho+\epsilon)(\rho+\alpha)(\sigma+\rho)}{(\sigma+\rho)(\rho+\epsilon)} + (\rho+\alpha)\right) - \lambda & -(\sigma+\rho) & \epsilon \\ \frac{(\rho+\epsilon)(1-\alpha)\beta\mu+(\sigma+\rho)\alpha\epsilon-(\rho+\epsilon)(\rho+\alpha)(\sigma+\rho)}{(\sigma+\rho)(\rho+\epsilon)} & -\lambda & 0 \\ \alpha & 0 & -(\rho+\epsilon) - \lambda \end{vmatrix} = 0$$

This gives the characteristic equation below

$$\begin{aligned} &-\left[\frac{(\rho+\epsilon)(1-\alpha)\beta\mu+(\sigma+\rho)\alpha\epsilon-(\rho+\epsilon)(\rho+\alpha)(\sigma+\rho)}{(\sigma+\rho)(\rho+\epsilon)} + (\rho+\alpha) + \lambda\right][\lambda(\rho+\epsilon) + \lambda^2] \\ &-[\sigma+\rho]\left[\frac{(\rho+\epsilon)(1-\alpha)\beta\mu+(\sigma+\rho)\alpha\epsilon-(\rho+\epsilon)(\rho+\alpha)(\sigma+\rho)}{(\sigma+\rho)(\rho+\epsilon)}\right][(\rho+\epsilon)+\lambda] + \lambda\alpha\epsilon = 0 \end{aligned} \tag{4.8}$$

Upon expansion of equation (4.8), we obtain

$$\begin{aligned} &\lambda^3 + \left[\frac{(\rho+\epsilon)(1-\alpha)\beta\mu+(\sigma+\rho)\alpha\epsilon}{(\sigma+\rho)(\rho+\epsilon)} + (\rho+\epsilon)\right]\lambda^2 \\ &+\left[\frac{(\rho+\epsilon)[(1-\alpha)\beta\mu-(\rho+\alpha)(\sigma+\rho)]}{(\sigma+\rho)} + \frac{(\rho+\epsilon)[(1-\alpha)\beta\mu-(\rho+\alpha)(\sigma+\rho)]+(\sigma+\rho)\alpha\epsilon}{(\rho+\epsilon)} + (\rho+\epsilon)(\rho+\alpha)\right]\lambda \\ &+(\rho+\epsilon)[(1-\alpha)\beta\mu-(\rho+\alpha)(\sigma+\rho)] + (\sigma+\rho)\alpha\epsilon = 0 \end{aligned} \tag{4.9}$$

Equation (4.9) can be written as

$$\lambda^3 + a\lambda^2 + b\lambda + c = 0 \tag{4.10}$$

where

$a = \frac{(\rho+\epsilon)(1-\alpha)\beta\mu+(\sigma+\rho)\alpha\epsilon}{(\sigma+\rho)(\rho+\epsilon)} + (\rho+\epsilon)$
$b = \frac{(\rho+\epsilon)[(1-\alpha)\beta\mu-(\rho+\alpha)(\sigma+\rho)]}{(\sigma+\rho)} + \frac{(\rho+\epsilon)[(1-\alpha)\beta\mu-(\rho+\alpha)(\sigma+\rho)]+(\sigma+\rho)\alpha\epsilon}{(\rho+\epsilon)} + (\rho+\epsilon)(\rho+\alpha)$
$c = (\rho+\epsilon)[(1-\alpha)\beta\mu-(\rho+\alpha)(\sigma+\rho)] + (\sigma+\rho)\alpha\epsilon$

Clearly $a > 0$, $b > 0$ and $c > 0$ for $R_0 > 1$ (i.e $(1-\alpha)\beta\mu > (\rho+\alpha)(\sigma+\rho)$)

And it follows that $ab - c > 0$

Hence by Routh-Hurwitz criterion for the cubic equation, the endemic equilibrium of the system (2.1) is locally asymptotically stable. □

4.4 Global stability of endemic equilibrium point $E^*(S^*, I^*, R^*)$

Now we investigate the global stability by use of Lyapunov function.

Theorem 4. *E^* is globally asymptotically stable provided that $R_0 > 1$*

Proof. Consider the Lyapunov function

$$V(S,I,R) = \left(S - S^* ln\frac{S}{S^*}\right) + M\left(I - I^* ln\frac{I}{I^*}\right) + N\left(R - R^* ln\frac{R}{R^*}\right) \tag{4.11}$$

Differentiating equation (4.11) with respect to t, gives;

$$\frac{dV}{dt} = \left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + M\left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} + N\left(1 - \frac{R^*}{R}\right)\frac{dR}{dt} \tag{4.12}$$

From system (2.1), we substitute for $\frac{dS}{dt}$, $\frac{dI}{dt}$ and $\frac{dR}{dt}$ in equation (4.12) to obtain

$$\begin{aligned}\tfrac{dV}{dt} = &\left(1 - \tfrac{S^*}{S}\right)[\mu + \epsilon R - (1-\alpha)\beta SI - (\rho+\alpha)\ S] \\ &+M\left(1 - \tfrac{I^*}{I}\right)[(1-\alpha)\beta SI - (\sigma+\rho)I] \\ &+N\left(1 - \tfrac{R^*}{R}\right)[\alpha S - (\rho+\epsilon)R]\end{aligned} \tag{4.13}$$

Upon rearrangement of equation (2.1) at endemic equilibrium , we obtain

$$\begin{aligned}\mu &= (1-\alpha)\beta S^* I^* + (\rho+\alpha)S^* - \epsilon R^* \\ (\sigma+\rho) &= (1-\alpha)\beta S^* \\ \frac{\alpha S^*}{R^*} &= (\rho+\epsilon)\end{aligned} \tag{4.14}$$

Now we substitute system (4.14) in (4.13) to get

$$\begin{aligned}\tfrac{dV}{dt} = &(1 - \tfrac{S^*}{S})[(1-\alpha)\beta S^* I^* + (\rho+\alpha)S^* - \epsilon R^* + \epsilon R - (1-\alpha)\beta SI - (\rho+\alpha)S] \\ &+M(1 - \tfrac{I^*}{I})[(1-\alpha)\beta SI - (1-\alpha)\beta S^* I] + N(1 - \tfrac{R^*}{R})[\alpha S - (\tfrac{\alpha S^*}{R^*})R]\end{aligned} \tag{4.15}$$

Upon substitution of $x = \frac{S}{S^*}$, $y = \frac{I}{I^*}$ and $z = \frac{R}{R^*}$ in (4.15) and simplification of the resulting equation, we obtain

$$\begin{aligned}\tfrac{dV}{dt} = &-\rho\tfrac{(S-S^*)^2}{S} + (1-\alpha)\beta S^* I^*(1 - xy - \tfrac{1}{x} + y) + \alpha S^*(1 - x - \tfrac{1}{x} + 1) + \epsilon R^*(z - 1 - \tfrac{z}{x} + \tfrac{1}{x}) \\ &+(1-\alpha)\beta S^* I^* M(xy - y - x + 1) + N\alpha S^*(x - z - \tfrac{x}{z} + 1)\end{aligned} \tag{4.16}$$

Hence by setting the coefficients of xy and x equal to zero and letting M =1 we get $N = \frac{(1-\alpha)\beta S^* I^*}{\alpha S^*}$ such that

$$\begin{aligned}\tfrac{dV}{dt} = &-\rho\tfrac{(S-S^*)^2}{S} + (1-\alpha)\beta S^* I^*(1 - xy - \tfrac{1}{x} + y) + \alpha S^*(1 - x - \tfrac{1}{x} + 1) + \epsilon R^*(z - 1 - \tfrac{z}{x} + \tfrac{1}{x}) \\ &+(1-\alpha)\beta S^* I^*(xy - y - x + 1) + (1-\alpha)\beta S^* I^*(x - z - \tfrac{x}{z} + 1)\end{aligned} \tag{4.17}$$

From the property that the geometric mean is less than or equal to the arithmetic mean, hence $\frac{dV}{dt} \leq 0$. The inequality $\frac{dV}{dt} = 0$ holds iff x=y=z=1 and $(S = S^*; I = I^*; R = R^*)$. By LaSalle's invariance principle in [12], every solution of the system (2.1) with initial conditions in $\Gamma = \{(S, I, R) \in \mathbb{R}^3_+; N \leq \frac{\mu}{\rho}\}$ approaches E^* , thus E^* is Globally Asymptotically stable. □

5 Numerical Simulation

A survey carried out by the ministry of health in 2014 [13] established that Makueni county has a higher rabies cases as a results of high dog to human ratio.

For example, the ratio of human population to dog population is 3:1 and since the human population is $1,002,979$ which is a projected population for the year 2018 based on 2009 census the the dog population is $334,326.3333$ which is approximately equal to $335,000$ as deduced from the ratio. Also, in every six dogs it is approximated that a single dog is infected with the rabies virus thus

from the total population of dogs the infected population is $55,833.3333$ which is approximated to $56,000$.

Using MATLAB ode45 solver and parameter values in Table 1, we carried out numerical simulation of the system (2.1) in order to understand the effect of educating the public on the control of rabies virus.

Table 1. Model's Parameter values

Parameter	Description	Value	Source
μ	Recruitment rate	$3.0 \times 10^{6} y^{-1}$	[14]
β	Transmission rate	$1.58 \times 10^{-7} y^{-1}$	[14]
ρ	Natural death rate	$0.056 y^{-1}$	[14]
σ	Disease induced mortality	$1 y^{-1}$	[14]
ϵ	Loss of immunity	$1 y^{-1}$	[14]
α	Education efficacy	$0 < \alpha < 1$	Assumed

Figure 2(a) shows trajectories of susceptible dogs at different levels of public education efficacy. When public education efficacy α is equal to 0.0(without public education), 0.3 and 0.6, the figure depicts a sharp increase of the numbers of susceptible dogs at the beginning followed by a sharp decrease then remain constant at certain numbers. This could be attributed to the ineffective public education. But for $\alpha = 0.9$, the number of susceptible dogs increase and remain constant as $t \to \infty$. This implies that the presence effective public education leads to reduction of disease prevalence whenever there is rabies outbreak, hence increase of susceptible dogs. Figure 2(b) shows trajectories of infected dogs at different levels of public education efficacy. When public education efficacy α is equal to 0.0, 0.3 and 0.6, the figure shows alternating decrease and increase of the number of infected dogs at the onset of an infection and lastly, the number of infected dogs remain constant as time $t \to \infty$. But for $\alpha = 0.9$, the number of infected dogs reduce to zero. This suggests that the presence of effective public education campaign eradicates the disease.

(a) (b)

Fig. 2. Impact of public education on (a) susceptible dogs and (b) infected dogs

6 Conclusion

A mathematical model of rabies transmission dynamics in dogs using public health education was developed in this study. Both disease free and endemic equilibrium point were studied which showed that, the disease free equilibrium point is both locally and globally asymptotically stable when $R_0 < 1$ and the endemic equilibrium point is both locally and globally asymptotically stable when $R_0 > 1$. Numerical Simulation results showed that as the public health education efficacy α increases from 0.0 to about 0.9 the number of susceptible dogs sharply increases and then stabilises. Also, when the education efficacy α is increased from 0.0 to about 0.9 the number of infected dogs

kept on decreasing until zero infections are obtained which implies, that educating the public on administration of both pre and post exposure vaccination and practicing responsible dog ownership would greatly reduce rabies cases in Kenya and therefore recommend that the government of Kenya foster public education on rabies control and elimination so as to attain its goal for having Kenya being rabies free country by 2030.

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Competing Interest

Authors have declared that no competing interests exist.

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