IOSR Journal of Mathematics (IOSR-JM)
e-ISSN: 2278-5728, p-ISSN: 2319-765X. Volume 12, Issue 3 Ver. IV (May. - Jun. 2016), PP 110-120
www.iosrjournals.org

Population Dynamics of Dogs Subjected To Rabies Disease

Demsis Dejene[1], Purnachandra Rao Koya[2]
[1,2]*School of Mathematical and Statistical Sciences, Hawassa University, Hawassa, Ethiopia*

Abstract: *In this paper we have considered the population dynamics of dogs subjected to rabies disease. A new mathematical model SEI_PI_FR is presented which is designed and developed with some reasonable modifications to the corresponding epidemic $SEIR$ model. Disease spread controlling technique called vaccination is included in the present model and studied its impact. Vaccine can be given to both susceptible and exposed individuals so as to control the spread of epidemic. The basic reproduction number is derived using the next generation matrix method. Disease free equilibrium point is found and endemic equilibrium state is identified. It is shown that the disease free equilibrium point is locally and globally asymptotically stable if the reproduction number takes a value less than one unit and unstable if it is more than one unit. Numerical simulation study is conducted using ode45 of MATLAB. The results and interpretations are elaborated and included in the text.*

Keywords: *Rabies, SEI_PI_FR model, Vaccination, Simulation, Reproductive number.*

I. Introduction

Rabies is a fatal disease for both humans and all other mammals. Rabies is caused by a virus and is associated with animal bites. The rabies disease has been in existence for more than 3000 years and thus is the oldest infectious disease known to medical science [1]. The rabies disease is being occurred with more intensity in about 150 countries and territories around the world. The occurrence of rabies is very high in developing countries of African and Asian continents.

It is quite common that poor and rural people interact more with animals including dogs. Hence, these people have more chances to be bitten, wounded, and injured by animals and get rabies disease. About 55 thousands of people die with rabies worldwide every year. Children are the most affected by rabies. More than 40% of the people bitten by infected animals are children. Among children also those who play with infected animals have more probability to be effected by rabies. More than a hundred thousands of people are exposed to rabies disease every year worldwide but only about 15 million people receive post exposure vaccination [15, 16].

A large number of rabies cases have been reported to occur among the wild life animals such as raccoons, skunks, bats and foxes. The rabies virus also circulates among the domestic animals. In fact dogs stand as a main vector in transmitting rabies virus between animals and humans.

Most of the North American and European countries had fought against the rabies disease and were successful in eradicating the virus. Even than about 61 thousand deaths occurred in the year 2010 due to rabies disease world wide as reported by the WHO. Ofcourse about 95% of these deaths occurred in the African and Asian continents [3].

The rabies disease born by animals of dog family or canine born rabies has been found recently in large scale in India and China. Increasing number of these cases in these countries has drawn worldwide attention. The complexity of the problem was made known to the world by these findings. The rabies epidemic has been attributed to several social, economical and cultural factors, poor waste management systems etc. Dog – human interaction is the main problem underlying the rabies epidemic [5, 6].

In Ethiopia rabies is a main infectious disease that has been recognized many centuries ago. According to the Ethiopian Health and Nutrition Research Institute (EHNRI), rabies has been endemic in Ethiopia since early seventeenth century. The first major outbreak of rabies due to dogs occurred in many parts of Ethiopia in the year 1884. The first case of rabies epidemic was reported and had a high prevalence in Addis Ababa, the capital city of Ethiopia. According to EHNRI, rabies disease in Ethiopia is generally born from domestic animals specially dogs. However, the involvement of other domestic animals like cats, cattle, sheep, goats and equines also cannot be ruled out [14].

1.1 Signs and Symptoms of Rabies Disease

Rabid animals or the animals infected by rabies disease behave different during the period of disease (K. M. Addo, 2012). Mainly the disease period is divided in to three stages viz., Prodromal stage, Excitative stage, and Dumb or paralytic stage.

Prodromal Stage: It is the first phase of the disease. The prodromal period may last for one to three days. In this beginning stage there will be some changes in the behavior of animals. But, these changes are slight and thus they are neglected very often. The rabid animals of this stage do not feel comfortable in company. They do not mix easily with others, excite easily, feel uneasy, and also finds difficult in swallowing and salivation. There is a frequent irritation or stimulation in the body parts connecting the urinary and genital organs. These facts are evidenced by frequent urination, erection and sexual desire. The rabid animals may also stop eating and drinking.

Excitative Stage: This is the second or middle phase of the disease. This stage is also known as 'raging fury' or 'mad dog syndrome' stage. The behavior of animals of this stage is irrational, aggressive, restless, and excite. These animals also develop mental disorders and mania for battings and making sounds. These animals loose both caution and fear for natural enemies. They roam aimlessly in the streets and on the highways. They bite other animals and people without any reason and aim. They hunt and chase moving objects and swallow foreign bodies. During this stage the saliva is highly infectious. Also the muscles and legs of the rabid animal begin to tremble making the animal unable to walk steadily. Breathing too becomes very difficult.

Dumb or Paralytic Stage: This is the third or last stage of the disease. This stage happens shortly before the death. In this stage the animal develops paralysis in the muscles of the throat, face, trunk and the limbs. More salivation is produced in the mouth and swallowing anything becomes difficult. Dropping of the lower jaw, rarely attempt or the effected animals are able to bite. Animals with dumb rabies appear depressed, lethargic and uncoordinated. Gradually they become completely paralyzed. If paralysis is prominent, this stage is also called silent fury. Paralysis progresses to all parts of the body with coma and death in a few hours.

Treatment: the first step in treating a person bitten by any animal is to wash the wound with soap and water. Dangerous as it is, the rabies virus also happens to be one of the most delicate organisms known. It dies in dried saliva within a few hours. It is also killed by ordinary sunlight, heat, household detergent and disinfectants. Pure iodine and hydrogen peroxide however have no effect on the virus. The animal should either be caged and watched for signs of rabies or killed and its brain tissues watched for signs of rabies. Because there is no cure and death is almost certain when the symptoms begin to show up, treatment for rabies involves supportive care. However, if a dog or a person is bitten by a rabid animal and has not yet experienced symptoms, there is an extremely effective post-exposure treatment. Most of the time, stitches should not be used for animal bite wounds. There are vaccines that are derived from a variety of tissue culture or chicken embryo origins in live or inactivated forms which are used for treating rabies. Some of these require revaccination, others protect adequately for three years. Vaccination of Rabies: Rabies research scientists have developed an extremely effective rabies vaccine regimen that provides protection against rabies [12]. This vaccine works in two ways; either after an exposure or for before an exposure. A person, who becomes infected with rabies and does not obtain treatment before the symptoms occur, dies in a short period after experiencing convulsions and other violent nervous symptoms. Dogs continue to be the main carrier of rabies in Africa and Asia and are responsible for most of the human rabies deaths worldwide. Pre – exposure rabies vaccines are available for dogs, cats, ferrets, horses, sheep, and all other mammals. To be effective, these rabies vaccines must be injected before an animal is exposed to rabies. If exposed, the dog should get a booster shot. Post-exposure treatment for rabies should begin as soon as possible after an exposure. Administration of rabies vaccine is a medical urgency, not a medical emergency. Post – exposure rabies treatment consists of a regimen of one dose of rabies immune globulin and five doses of rabies vaccine given over a 28 day period.

Here we now introduce some important terminology that is frequently used in this work. Compartmentalize a group of dogs with similar status or with respect to the same disease [20]. A dog is said to be susceptible if that has not yet infected by the disease but likely to get the disease in future. A dog is said to be exposed to a disease when the virus enters into the dog's body. At this stage the effects of the disease cannot be identified with the dog, because the effects are in sleeping state. A dog is said to be infected if it has the disease in its body and is able to transfer the disease to other susceptible dogs.

Incubation period is defined as the time duration between an individual gets exposed to an infection and it gets sickness or confirmation of the disease. The dog may be tested positive of the infection after this period. This is the time taken by a dog to shift from the compartment E to the compartment I for $SEIR$ and for SEI_PI_FR model. The incubation period is also known as latent period. A dog is said to be in the removed compartment if he will never again get infection or infect others. The dog of this compartment is dead. Contagious diseases are the diseases which spread by physical contact between susceptible and infected dogs.

Mathematical modeling has become an important tool in analyzing the epidemiological characteristics of infectious diseases. Several mathematical models have been proposed for modeling the spread of infectious diseases. The earliest account of the mathematical modeling of the spread of a disease was carried

out in 1766 by Daniel Bernoulli [Ref]. Trained as a physician, Bernoulli created a mathematical model to defend the practice of inoculating against smallpox. The calculations from this model showed that universal inoculation against smallpox would increase the life expectancy from 26 years 7 months to 29 years 9 months [10]. During all these years since, mathematicians, biologists, physicians, epidemiologists, and others have contributed to the maturing discipline of mathematical epidemiology. Several books have played a significant role in the development of theory [11].

Mathematical models associated with the study of rabies in various countries have existed over the years [12]. Early models of rabies dynamics followed the $SEIR$ framework where populations were subdivided into specific classes corresponding to susceptible S, exposed E, infectious I, and removed R individuals [13]. The dynamics were encapsulated through the construction of a system of ordinary differential equations $ODEs$ representing either single populations or linked Meta populations from which a variety of predictions can be drawn concerning temporal and spatial pattern. These early models made use of the basic $SEIR$ compartmental framework and these models were used to derive several critical features of disease emergence and spread. The models were used to calculate the critical threshold for epidemic emergence and the basic reproductive number R_0 for the virus. When R_0 is greater than 1, the infection will spread and an epidemic will result.

II. Model Formulation

Here we now formulate $SEIR$ and SEI_PI_FR models for describing the dynamics of dog rabies. We categorize the whole dog population into susceptible, exposed, infected and removed groups for $SEIR$ model and susceptible, exposed, infected but in prodromal stage), infected but furious stage and recover groups for SEI_PI_FR model. Susceptible groups have no disease, but they are likely to be infected in case of contact with rabid dogs, Exposed individuals are those who contracted the virus via bites or scratch, but still they have not shown symptoms. Infected individuals are those who develop clinical symptoms and they are unlikely to recover due to the nature of rabies [15]. The recovered classes are those who recovered through vaccination before they reach to infectious stage, whereas the rest get infected and die eventually.

2.1 SEIR Model And Transmission Of Rabies Without Vaccination

In a standard $SEIR$ model, the population is divided into four compartments. These are the susceptible compartment is neither exposed nor infected by rabies disease now. But they are very sensitive or easily influenced, likely to be affected by or having the quality of receiving the disease in future. Dogs that have been bitten by infected dogs but are not infectious make up the exposed class. Dogs that are infected with rabies virus and contagious make up the infective class. The removed class constitutes dogs which have died from the infection.

The proportions of individuals divided into the compartments S, E, I, R are denoted by the time dependent parameter notations: $S(t)$ denotes the number of dogs in the susceptible compartment where the dogs are capable of getting infected (ii) $E(t)$ denotes the number of dogs in the exposed compartment where the dogs are incubating the infection (iii) $I(t)$ denotes the number of dogs in the infected compartment where the dogs are infected with the virus and (v) $R(t)$ is the number of dogs in the removed compartment where the dogs are considered to be died [18].

Let N denotes the total population size. We assume that the duration of the epidemic is short compared to the life time of its hosts. So that we can neglect new births and non – disease related deaths during the epidemic

The population is therefore closed of constant size N and the situation is therefore illustrated as follows:

S □ **E** □ **I** □ **R**

Here, β denotes the transmission coefficient among dogs; λ denotes the latency or incubation rate in dogs; and γ denotes the death rate in dogs. The $SEIR$ model can be described by the following set of ordinary differential equations $ODEs$:

$$\mathrm{ds/dt} = -(\beta \mathrm{SI/N}) \quad (1)$$

$$\mathrm{dE/dt} = (\beta \mathrm{SI/N}) - \lambda \mathrm{E} \quad (2)$$

$$\mathrm{ds/dt} = \lambda \mathrm{E} - \gamma \mathrm{I} \quad (3)$$

$$\mathrm{dR/dt} = \gamma \mathrm{I} \quad (4)$$

Here we assumed that N is a constant and denotes the total number of population in the system. After transmission of the virus, susceptible individuals enter the exposed class E before they become infectious individuals and later either they recover or die. Also β is transmission rate of disease from susceptible to exposed, λ and γ are the average durations of incubation and death rate in dogs respectively.

We now consider the scaling of the SEIR model (1) to (4). Scaling is the technique that changes the dimensional equations in to into those of dimensionless. The dimension of a dimensionless quantity is considered as a unity. To scale an equation means to introduce dimensionless variables based on the scales of the variables in the equations. When the equations are scaled, it is easy to see which parts are more important and which are less important. Scaling removes unnecessary parameters and reduces the number of parameters. Now we scale $SEIR$ model $ODEs$ (1) to (4) by introducing a set of new variables as $u = SN,\ v = E,\ w = IN,$ $z = RN$ and also obtain the new dimensionless time coordinate as $\tau = \gamma t$. In terms of these scaled relations, the system (1) to (4) takes the form as below:

$du/d\tau = R_0 uw$ (5)

$dv/d\tau = R_0 uw - kv$ (6)

$dw/d\tau = kv - w$ (7)

Here in (5) to (7), we denote $z = 1 - u - v - w$, $R_0 = \beta/\gamma$ is the basic reproductive ratio and $k = \lambda/\gamma$ is the average infection period. The system (5) to (7) is the dimensionless equations of the $SEIR$ epidemic model (1) to (4). It is used to manage and understand the model in a simpler manner. Then simulation study can be done using the $MATLAB$ code.

2.2 SEI_PI_FR Model Representing Rabies Transmission

2.2.1 Assumptions of the Model

The modified SEI_PI_FR model is the extension of the existing $SEIR$ model used to describe the dynamics of dog rabies and to compute the amount of susceptible, exposed, infected and recovered due to vaccination dogs in a population groups. Rabid animals exhibit three different behaviors during the disease [1, 2]. Upon infection, the dogs enter the first phase called prodromal phase associated with shyness and isolation. After that, they enter into the second phase called furious phase and exhibit high aggressiveness. Lastly, they enter the phase called paralytic stage and then die. Since their contact behavior is very different, the infected population is divided into two groups for prodromal and furious dogs. The paralytic stage is excluded since dogs are assumed not to bite any one as they suffer with paralysis. This model is an appropriate one to use.

The assumptions of the model are as follows: (i) the birth rate of dogs is assumed to be equal to their death rate so that the population under consideration is closed, (ii) the way a dog can leave the susceptible group is to become recovery class at the rate of θ due to pre expose vaccination and exposed class at the transmission rate of β, (iii) the lose rate of the recovered class go to the susceptible class directly at the rate of δ, (iv) the way a dog can leave the exposed group is to recover from the disease with the rate of θ due to post exposed vaccination and infected or prodromal I_P with the rate of λ, (v) the way the dog can leave from the infected prodromal I_P group to infected furious I_F class is with the rate of α, (vi) the rate γ of the infected furious I_F go to death due to rabies, (vii) there is no chance to recover from the disease for all infected dogs, (viii) age, sex, social status, and race do not affect the probability of being infected and (ix) all the parameters and state variables of the model are positive. The flow diagram of the SEI_PI_FR model is given in Figure 1.

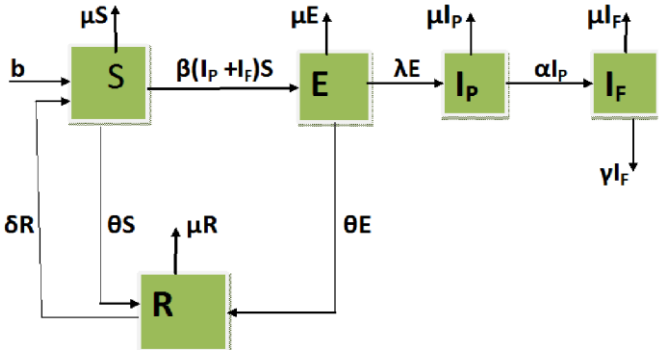

Figure 1: Flow diagram of the SEI_PI_FR model

The SEI_PI_FR model described in the Figure 1 can be expressed using a set of five ordinary differential equations as shown from (8) to (12).

$ds/dt = b - \beta(I_P + I_F)S - \theta S - \mu S + \delta R$ (8)

$dE/dt = \beta(I_P + I_F)S - \lambda E - \theta E - \mu E$ (9)

$dI_p/dt = \lambda E - \alpha I_P - \mu I_P$ (10)

$dI_F/dt = \alpha I_P - \gamma I_F - \mu I_F$ (11)

$dR/dt = \theta S + \theta E - \delta R - \mu R$ (12)

In this model (8) to (12), individuals in the susceptible compartment are subjected to an infected host with a contact rate of β. Once infected with the disease, they then enter the exposed phase. From the exposed compartment a portion λE of individuals enter the infected phase I_P while the remaining portion θE of the individuals enters the recovery phase. Recovery class from the susceptible phase is achieved with the rate of θ due to pre exposed vaccinations and again recovered from exposed phase is achieved with the rate of θ due to post exposed vaccination. Another possibility is for the individual to die from Furious compartment I_F infected at the rate of γ due to the rabies. In this model the total population size N is equal to the sum of the populations of these compartments and hence $N = S + E + I_P + I_F + R$.

2.2.2 Basic Reproductive Number

The basic reproduction number R_0 is a threshold parameter defined as the average number of secondary infection caused by an infectious individual by introducing in to a completely susceptible population. It is also called basic reproduction ratio or basic reproductive rate [7]. If more than one secondary infection is produced from one primary infection that is, $R_0 > 1$ then an epidemic occurs. When $R_0 < 1$ then there is no epidemic and it means that the disease dies out over a period of time. When Ro = 1 then the disease becomes endemic, meaning the disease remains in the population at a constant rate as one infected dog transmits the disease to one susceptible [Ref] (H. W. Hethcote, 2006). We compute the basic reproduction number using the next generation matrix approach by taking the infected compartments to be E, I_P and I_F from the equations (9) to (11). We construct the matrices f_i and $\mathrm{V_i}$ and the corresponding matrices of partial derivatives F and V. Also we find the inverse matrix V^{-1} of V. Finally we compute the reproduction number R_0 as the trace of the matrix FV^{-1}. All these matrices are computed and constructed and given below:

$$f_i = \begin{bmatrix} \beta(\mathrm{I_P} + \mathrm{I_F})\mathrm{S} \\ 0 \\ 0 \end{bmatrix},\ F = \frac{\partial f_i}{\partial x_j} = \begin{bmatrix} 0 & \beta\mathrm{S} & \beta\mathrm{S} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix},\ \mathrm{V_i} = \begin{bmatrix} (\lambda + \theta + \mu)\mathrm{E} \\ \alpha\mathrm{I_P} + \mu\mathrm{I_P} - \lambda\mathrm{E} \\ \gamma\mathrm{I_F} + \mu\mathrm{I_F} - \alpha\mathrm{I_P} \end{bmatrix}$$

$$\mathrm{V} = \frac{\partial \mathrm{V_i}}{\partial \mathrm{x_j}} = \begin{bmatrix} \lambda + \theta + \mu & 0 & 0 \\ -\lambda & \alpha + \mu & 0 \\ 0 & -\alpha & \gamma + \mu \end{bmatrix},\ \mathrm{V}^{-1} = \begin{pmatrix} \frac{1}{\lambda+\theta+\mu} & 0 & 0 \\ \frac{\lambda}{(\lambda+\theta+\mu)(\alpha+\mu)} & \frac{1}{(\alpha\mu)} & 0 \\ \frac{\lambda\alpha}{(\lambda+\theta+\mu)(\alpha+\mu)(\gamma+\mu)} & \frac{\alpha}{(\alpha+\mu)(\gamma+\mu)} & \frac{1}{\gamma+\mu} \end{pmatrix}$$

Also the reproductive number is obtained as

$$\mathrm{R_0} = \rho(\mathrm{FV}^{-1}) = \frac{\beta\lambda(\gamma+\mu+\alpha)}{(\lambda+\theta+\mu)(\alpha+\mu)(\gamma+\mu)} \qquad (13)$$

2.2.3 Disease Free Equilibrium Point E_0 of SEI_pI_FR Model

Let $E_0 = (S^*, E^*, I_P^*, I_F^*, R^*)$ represents the disease free equilibrium point of the $\mathrm{SEI_pI_FR}$ model given by the system (8) to (12). Disease free equilibrium points are steady state solutions of a mathematical model indicating that there is no disease [19]. The compartmental classification of dog population reveals that the diseased dog population is distributed only in exposed and infected compartments. Hence, in the absence of infection we have $E^* = I_P^* = I_F^* = 0$ and the equilibrium points are obtained by setting the right hand sides of the model equations (9), (10) and (11) to zero, then the disease free equilibrium point $\mathrm{E_0}$ will be obtained as

$$E_0 = (S^*, 0, 0, 0, R^*) \qquad (14)$$

Here in (14) we used the notations $\mathrm{S^*} = \{[\mathrm{b}(\delta + \mu)]/[\mu(\theta + \delta + \mu)]\}$ and $\mathrm{R^*} = \{(\mathrm{b}\theta)/[\mu(\theta + \delta + \mu)]\}$. The disease free equilibrium point E_0 given in (14) satisfies two properties and those are stated as Theorem 1.

Theorem 1: If $R_0 < 1$ then the disease free equilibrium point E_0 of $\mathrm{SEI_pI_FR}$ system is (a) locally asymptotically stable and (b) globally asymptotically stable in the region Ω.

2.2.4 The Endemic Equilibrium Point E_1 of SEI_pI_FR Model

We shall now study the existence of the endemic equilibrium state of the modified model. Endemic equilibrium point E_1 is a steady state solution where the disease persists in the population. We now consider the existence and uniqueness of the endemic equilibrium point. Let E_1 be denoted by $\mathrm{E_1} = (\mathrm{S^1}, \mathrm{E^1}, \mathrm{I_P^1}, \mathrm{I_F^1}, \mathrm{R})$ and its coordinates should satisfy the conditions $(\mathrm{S^1}, \mathrm{E^1}, \mathrm{I_P^1}, \mathrm{I_F^1}, \mathrm{R}) > 0$. Upon imposing these conditions in the system of equations (8) to (12) we have respectively $[b - \beta I_P + I_F S - \theta S - \mu S + \delta R = 0$, $\beta I_P + I_F S - \lambda E - \theta E - \mu E = 0$, $\lambda E - \alpha + \mu I_P = 0$, $\alpha I_P - \gamma + \mu I_F = 0$ and $\theta S + \theta E - \delta R - \mu R = 0$. On solving the foregoing system of equations we get the following:

$$S^* = \frac{1}{R_0} = \left\{\frac{(\lambda+\theta+\mu)(\alpha+\mu)(\gamma+\mu)}{\beta\lambda(\gamma+\mu+\alpha)}\right\}$$

$$E^* = \left\{\frac{(\alpha+\mu)(\gamma+\mu)[(\mu/R_0)(\theta+\delta+\mu)-b(\delta+\mu)]}{[\delta\theta(\gamma+\mu+a)(\alpha+\mu)-\beta\lambda(\delta+\mu)(\gamma+\mu+a)]}\right\}$$

$$I_P^* = \left\{\frac{\lambda(\gamma+\mu)[(\mu/R_0)(\theta+\delta+\mu)-b(\delta+\mu)]}{\delta\theta(\gamma+\mu)(\alpha+\mu)-\beta\lambda(\delta+\mu)(\gamma+\mu+\alpha)}\right\}$$

$$I_F^* = \frac{\alpha\lambda\left[\frac{\mu}{R_0}(\theta+\delta+\mu)-b(\delta+\mu)\right]}{\delta\theta(\gamma+\mu)(\alpha+\mu)-\lambda\beta(\delta+\mu)(\gamma+\mu+\alpha)}$$

$$R^* = \frac{\theta\{(\alpha+\mu)(\gamma+\mu)[\mu(\theta+\delta+\mu)-R_0b(\delta+\mu)]+\theta\delta(\gamma+\mu)(\gamma+\mu)-\beta\lambda(\delta+\mu)(\gamma+\mu+\alpha)\}}{R_0(\delta+\mu)[\delta\theta(\gamma+\mu)(\alpha+\mu)-\beta\lambda(\delta+\mu)(\gamma+\mu+\alpha)}$$

III. Stability Analysis

3.1. Local Stability Of The Disease Free Equilibrium Point

Here we now investigate the local stability of the disease free equilibrium point.

Theorem 1: If $R_0 < 1$ then (i) the disease – free Equilibrium E_0 of system (3) is locally asymptotically stable (ii) The disease - free equilibrium E_0 of system (3) is globally asymptotically stable in the region Ω.

From (6) the disease free equilibrium point is given by $E_0 = (S^*, E^*, I^*, I_F^*, R^*)$ where $S^* = \{[b(\delta+\mu)]/[\mu(\theta+\delta+\mu)]\}$, $E^* = I^* = I_F^* = 0$ and $R^* = \{b\theta/\mu(\theta+\delta+\mu)\}$.

Theorem 2: (Routh - Hurwitz Criteria) given a characteristic polynomial $P(k) = k^n + a_1k^{n-1} + \cdots + a_{n-1}k + a_n$ where the coefficients a_i for all $i = 1 \ldots n$ are all real constants, we define the n dimensional Hurwitz matrices in terms of the coefficients a_i of the polynomial as follows:

$$H_1 = (a_1), H_2 = \begin{pmatrix} a_1 & 1 \\ a_3 & a_2 \end{pmatrix}, H_3 = \begin{pmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & 0 & a_3 \end{pmatrix} \text{ and } H_n = \begin{pmatrix} a_1 & 1 & 0 & 0 & \ldots & 0 \\ a_3 & a_2 & a_1 & 1 & \ldots & 0 \\ a_5 & a_4 & a_3 & a_2 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & 0 & 0 & \cdots & a_n \end{pmatrix}$$

Here in the matrices $a_j = 0$ whenever $j > n$. All the roots of the polynomial $p(k)$ are negative or have negative real part if and only if the determinants of all Hurwitz matrices are positive i.e. Det $H_j > 0$ for all $j = 0, 1, 2 \ldots, n$. Complete details on Routh – Hurwitz criteria are available in the literature [2, 15]. Next we derive the Jacobin matrices for the system (3). This is done by differentiating each of the equations of the system (3) in terms of state variables viz., S, E, I_P, I_F and R.

$$J_{E_0} = \begin{bmatrix} -\beta(I_p^* + I_F^*) - (\theta+\mu) & 0 & -\beta S^* & -\beta S^* & \delta \\ \beta(I_p^* + I_F^*) & -(\mu+\lambda+\theta) & \beta S^* & \beta S^* & 0 \\ 0 & \lambda & -(\mu+\alpha) & 0 & 0 \\ 0 & 0 & \alpha & -(\mu+\gamma) & 0 \\ 0 & 0 & 0 & 0 & -(\delta+\mu) \end{bmatrix} \quad (7)$$

Up on evaluating the matrix given in (7) at the disease free equilibrium point $E_0 = (S^*, E^*, I^*, I_F^*, R^*)$ where $S^* = \{[b(\delta+\mu)]/[\mu(\theta+\delta+\mu)]\}$, $E^* = I^* = I_F^* = 0$ and $R^* = \{b\theta/\mu(\theta+\delta+\mu)\}$ we have

$$J_{E_0} = \begin{bmatrix} -(\theta+\mu) & 0 & -\beta S^* & -\beta S^* & \delta \\ 0 & -(\mu+\lambda+\theta) & \beta S^* & \beta S^* & 0 \\ 0 & \lambda & -(\mu+\alpha) & 0 & 0 \\ 0 & 0 & \alpha & -(\mu+\gamma) & 0 \\ 0 & 0 & 0 & 0 & -(\delta+\mu) \end{bmatrix}$$

$$J_{E_0} = \begin{bmatrix} -(\theta+\mu) & 0 & \frac{-\beta b(\delta+\mu)}{\mu(\theta\delta\mu)} & \frac{-\beta b(\delta+\mu)}{\mu(\theta\delta\mu)} & \delta \\ 0 & -(\mu+\lambda+\theta) & \frac{\beta b(\delta+\mu)}{\mu(\theta\delta\mu)} & \frac{\beta b(\delta+\mu)}{\mu(\theta\delta\mu)} & 0 \\ 0 & \lambda & -(\mu+\alpha) & 0 & 0 \\ 0 & 0 & \alpha & -(\mu+\gamma) & 0 \\ 0 & \theta & 0 & 0 & -(\delta+\mu) \end{bmatrix}$$

The characteristic equation of the matrix J_{E_0} is $|J_{Eo} - kI| = 0$. Here I is an identity matrix of class 5×5 and k is the eigenvalue. Using this characteristic equation we derive the characteristic polynomial as

$P(k) = k^5 + [x + z + w + y + r]k^4 + [xz + xw + xy + xr + zw + zy + zr + wy + wr + yr - \lambda c + \delta\theta]k^3 + xzw + xzy + xzr + xwy + xwr + xyr + zwy + zwr + zyr + wyr - x\lambda c + z\lambda c + \lambda cw + \lambda c\alpha + \theta\delta w + \theta\delta y + \theta\delta r]k^2 + [xzwy + xzwr + xzyr + xwyr + zwyr + \theta\delta\lambda c - (xz\lambda c + x\lambda cw + x\lambda c\alpha + z\lambda cw + z\lambda c\alpha + \theta\delta wy + \theta\delta wr + \theta\delta yr]k + xzwyr + \theta\delta\lambda cw + \theta\delta\lambda c\alpha - xz\lambda cw + xz\lambda c\alpha + \theta\delta wyr$

Here in what precedes we have used the notations $x = (\theta + \mu)$, $y = (\mu + \lambda + \theta)$, $c = [\beta b(\delta + \mu)/\mu(\theta + \delta + \mu)]$, $r = (\mu + \alpha)$, $w = (\mu + r)$ and $z = (\delta + \mu)$. The characteristic polynomial $P(k)$ can also be expressed as

$$P(k) = k^5 + A_1k^4 + A_2k^3 + A_3k^2 + A_4k + A_5 \quad (8)$$

Here in (8), the coefficients A_1, A_2, A_3, A_4 and A_5 are functions representing respectively $A_1 = [x + z + w + y + r]$, $A_2 = [xz + xw + xy + xr + zw + zy + zr + wy + wr + yr - (\lambda c + \delta\theta)]$, $A_3 = [xzw + xzy + xzr + xwy + xwr + xyr + zwy + zwr + zyr + wyr - (x\lambda c + z\lambda c + \lambda cw + \lambda c\alpha + \theta\delta w + \theta\delta y + \theta\delta r$, $A_4 = xzwy + xzwr + xzyr + xwyr + zwyr + \theta\delta\lambda c - xz\lambda c + x\lambda cw + x\lambda c\alpha + z\lambda cw + Z\lambda c\alpha + \theta\delta wy + \theta\delta wr + \theta\delta yr$ and $A_5 = xzwyr + \theta\delta\lambda cw + \theta\delta\lambda c\alpha - xz\lambda cw + xz\lambda c\alpha + \theta\delta wyr$.

Using the characteristic polynomial represented by (8) the Hurwitz matrices can be constructed and their determinants are computed as

(a) $H_1 = (A_1)$ and $\det(H_1) = A_1$

(b) $H_2 = \begin{pmatrix} A_1 & 1 \\ A_3 & A_2 \end{pmatrix}$ and $\det(H_2) = A_1A_2 - A_3 > 0$

(c) $H_3 = \begin{pmatrix} A_1 & 1 & 0 \\ A_3 & A_2 & A_1 \\ A_5 & A_4 & A_3 \end{pmatrix}$ and $\det(H_3) = A_1A_2A_3 - A_3^2$

(d) $H_4 = \begin{bmatrix} A_1 & 1 & 0 & 0 \\ A_3 & A_2 & A_1 & 0 \\ 0 & A_4 & A_3 & A_2 \\ 0 & 0 & 0 & A_4 \end{bmatrix}$ and $\det(H_4) = -A_4(A_1^2A_4 - A_1A_2A_3 + A_3^2) > 0$ or equivalently it can be observed that $A_1A_2A_3 > A_1^2A_4 + A_3^2$

(e) $H_5 = \begin{pmatrix} A_1 & 1 & 0 & 0 & 0 \\ A_3 & A_2 & A_1 & 1 & 0 \\ A_5 & A_4 & A_3 & A_2 & A_1 \\ 0 & 0 & A_5 & A_4 & A_3 \\ 0 & 0 & 0 & 0 & A_5 \end{pmatrix}$ **a**nd after some rearrangement the determinant of the Hurwitz matrix H_5 simplifies to $\det(H_5) = A_5A_1A_2(A_3A_4 - A_2A_5) - A_3(A_3A_4 - A_2A_5) + A_1A_4A_5(A_5 - A_1A_4) - A_5(A_5 - A_1A_4)$.

By Routh – Hurtwiz criteria of Theorem 2 the determinant of Hurtwiz matrix becomes positive if the following conditions hold true $A_1 > 0$, $A_2 > 0$, $A_3 > 0$, $A_4 > 0$, $A_5 > 0$, $A_3A_4 > A_2A_5$, $A_5A_1A_2 > A_3$, and $A_5 > A_1A_4$. It required that all these requirements should hold true in our present model. Therefore, $A_1 = x + z + w + y + r$ is greater than zero. The parameter A_2 is positive if and only if $xz + xw + xy + xr + zw + zy + zr + wy + wr + yr > \lambda c + \delta\theta$. The parameter A_3 is positive if and only if $[xzw + xzy + xzr + xwy + xwr + xyr + zwy + zwr + zyr + wyr > x\lambda c + z\lambda c + \lambda cw + \lambda c\alpha + \theta\delta w + \theta\delta y + \theta\delta r$. The parameter A_4 is positive if and only if $[xzwy + xzwr + xzyr + xwyr + zwyr + \theta\delta\lambda c] > [x\lambda c(z + w + \alpha) + z\lambda cw + z\lambda c\alpha + \theta\delta wy + \theta\delta wr + \theta\delta yr$. Finally, the parameter A_5 is positive if and only if $xzwyr + \theta\delta\lambda cw + \theta\delta\lambda c\alpha > [xz\lambda cw + xz\lambda c\alpha + \theta\delta wyr]$. Hear all the requirements on the parameters A_1, A_2, A_3, A_4 and A_5 are found hold well.

Similarly, after exercising the same procedure it can be further observed that $A_3A_4 > A_2A_5$, $A_5A_1A_2 > A_3$ and $A_5 > A_1A_4$. That is, $[A_3A_4 + A_5A_1A_2 + A_5] > [A_2A_5 + A_3 + A_1A_4]$ holds. Hence all roots of the characteristic polynomial (8) are negative and this verifies that the system (3) is locally asymptotically stable.

Global Stability of the Disease Free Equilibrium Point

In this section, we study the global properties of the disease free equilibrium point. the global property of the disease free equilibrium point is provide in the form of a theorem as stated in the following:

Theorem 2 If the reproduction number satisfies the condition $R_0 < 1$ then the disease free equilibrium point $E_0 = \left(\frac{b(\delta+\mu)}{\mu(\theta+\delta+\mu)}, 0, 0, 0, \frac{b\theta}{\mu(\theta+\delta+\mu)}\right)$ is globally asymptotically stable in the region Ω. Further, if $R_0 > 1$ then E_0 is unstable.

Proof of Theorem 2: By the comparison theorem the rate of change of the variables representing the infected components of model given by the system (3) can be rewritten as

$$\begin{bmatrix} \mathrm{E}'(\mathrm{t}) \\ \mathrm{I}_{\mathrm{P}}'(\mathrm{t}) \\ \mathrm{I}_{\mathrm{F}}'(\mathrm{t}) \end{bmatrix} = [\mathrm{F}-\mathrm{V}] \begin{bmatrix} \mathrm{E} \\ \mathrm{I}_{\mathrm{P}} \\ \mathrm{I}_{\mathrm{F}} \end{bmatrix} = \begin{bmatrix} -(\lambda+\theta+\mu) & \beta s & \beta s \\ \lambda & -(\alpha+\mu) & 0 \\ 0 & \alpha & -(\gamma+\mu) \end{bmatrix} \begin{bmatrix} \mathrm{E} \\ \mathrm{I}_{\mathrm{P}} \\ \mathrm{I}_{\mathrm{F}} \end{bmatrix}. \quad (10)$$

Here in (10) the matrices F and V are defined by the expressions (4). But we also note that $s \leq \{[b(\delta+\mu)]/[\mu(\theta+\delta+\mu)]\}$ for all $t \geq 0$ in the region Ω and hence we obtain

$$\begin{bmatrix} \mathrm{E}'(\mathrm{t}) \\ \mathrm{I}_{\mathrm{P}}'(\mathrm{t}) \\ \mathrm{I}_{\mathrm{F}}'(\mathrm{t}) \end{bmatrix} \leq [\mathrm{F}-\mathrm{V}] \begin{bmatrix} \mathrm{E} \\ \mathrm{I}_{\mathrm{P}} \\ \mathrm{I}_{\mathrm{F}} \end{bmatrix} \quad (11)$$

We have seen that the eigenvalues of the matrix $[\mathrm{F}-\mathrm{V}]$ given in (10) are located on its main diagonal and are real and negative i.e. $-(\lambda+\theta+\mu)$, $-(\alpha+\mu)$, and $-(\gamma+\mu)$. The off diagonal elements of matrix $[\mathrm{F}-\mathrm{V}]$ are non – negative, since all the parameters are positive, and thus (10) is a Metzler matrix. It follows that the linear differential inequality system (11) is stable whenever the reproduction number satisfies the condition $R_0 < 1$.

Consequently, it also can be observed that $(\mathrm{E}, \mathrm{I}_{\mathrm{P}}, \mathrm{I}_{\mathrm{F}}) = (0,0,0)$ as $t \to \infty$. Further the evaluation of the system (3) at $E = I_P = I_F = 0$ and when $\mathrm{R}_0 < 1$ it results in obtaining $s = \{[b(\delta+\mu)]/[\mu(\theta+\delta+\mu)]\}$. Hence, the disease free equilibrium point E_0 is globally asymptotically stable in the region Ω.

Model variables and their descriptions are tabulated in the Table 1. Model parameters and their descriptions are tabulated in the Table 2.

Symbol	Description of the variables
$S(t)$	Susceptible dog population at time t
$E(t)$	Exposed dog population at time t
$I_P(t)$	Infectious or prodromal stage dog population at time t
$I_F(t)$	Infectious or furious stage dog population at time t
$R(t)$	Recovered dog population at time t

Table 1: Model variables and their description

Symbol	Description of the variables
β	Transmission coefficient of the disease among dogs
λ	Latency or incubation rate of the disease in dogs
γ	Death rate of dogs due to diseases
θ	Vaccination rate coefficient
δ	The loss rate of vaccination immunity for dogs.
μ	Natural death rate of dogs
α	Rate of propagation of furiousness among dogs
b	Birth rate of dogs

Table 2: Model parameters and their description

IV. Simulation Study of the Model

Here we consider simulation study of both $SEIR$ and SEI_PI_FR models with varying values of reproductive number R_0. The main focus of the simulation study is to investigate the response of model parameters up on the rabies epidemic. We have considered R_0 assigning different values few of which are less than and the other are greater than one unit and conduct simulation study.

3.1 Summation of $SEIR$ Model Describing Rabies Transmission without Vaccination

In this $SEIR$ model, standard values for the parameters of the ordinary differential equations are obtained from Ghana Veterinary Medical Association Report, 2010 and are used. The simulation studies and the analysis made are based on these standard values which are displayed below in Table 3.

Parameter	Description of the parameter	Standard values
β	Transmission coefficient	0.0030417 per month
λ	Rate of latency or incubation in dogs	$ 0.0000043973 per month
γ	Death rate	0.002293 per month
K	Average infectious period	0.0019177 per month

Table 3: standard values of the $SEIR$ model parameters

From the $SEIR$ model equations (3) we can obtain the basic reproductive ratio of rabies transmission as $R_0 = (\beta/\gamma) = (0.0030417/0.002293) = 1.3267$. Since $R_0 > 1$ here, the prevalence of

rabies is considered as an epidemic. This is because the transmission coefficient among dogs exceeds the death rate of dogs. If β is reduced and γ remains the same then the reproduction ration falls below one unit that is, for an example $R_0 = (\beta/\gamma) = (0.001908/0.002293) = 0.8321 < 1$. That is to conclude that keeping γ the same any value assigned for β that is less than 0.002293 will result in having $R_0 < 1$.

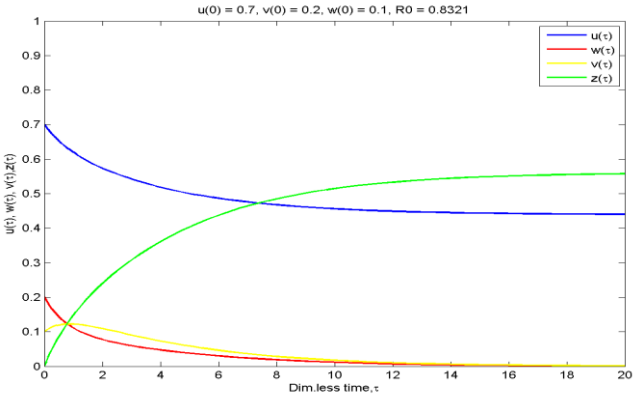

Figure2. The numerical simulation for rabies infected dogs model using the parametric values $\beta = 0.0030417$, $\lambda = 0.0000043973$ and $\gamma = 0.002293$ that is $R_0 < 1$

In Figure 2 it is illustrated the relationship among the model variables. It is represented that susceptible S with blue curve, exposed E with yellow curve, infected I with red curve, and removed R with green curve. For the purpose the standardized values are used. The analysis of numerical solution of $SEIR$ model representing dog rabies is as follows: Susceptible class decreases slowly in the time interval $(0, 10)$ and converges to a small constant for all the times greater than 10. Exposed and infected classes decreasing for time when the time is large then both compartment approaching to null or zero and the removed class increases as seen in figure. Thus, figure 2 shows that the disease dies out.

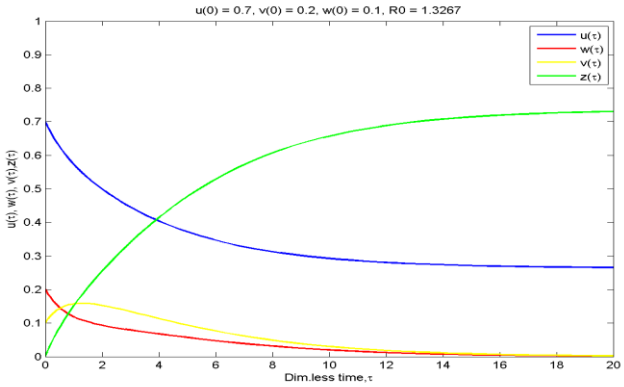

Figure 3: The numerical simulation of $SEIR$ model representing dog rabies with $\beta = 0.0030417$ and $\gamma = 0.002293$ that is $R_0 > 1$.

In figure 3 it is illustrated that the relationship among the model variables. Susceptible $\mathcal{S}$ is represented with blue curve, expose $\mathcal{E}$ is with yellow curve, infected $\mathcal{I}$ is with red curve, and removed $\mathcal{R}$ is with green curve. The removed dogs in figure 3 increase gradually throughout all the time while the susceptible dogs decrease. The numbers of exposed and infected dogs decrease to zero after the time is 16 units.

3.2 Simulation of $\mathcal{SE}_1\mathcal{I}_p\mathcal{I}_r\mathcal{R}$ Model Representing Rabies Transmission with Vaccination

Here we consider simulation study of the model (3) and draw some important observations. This simulation study is based on the parameter values mentioned in a tabular form in table 3.

Parameters	Values	Units	Source
β	0.733	Per year	assumption
λ	0.965	Per year	Assumption
γ	0.925	Per year	Assumption

θ	Variable	Per year	Assumption
δ	0.02	Per year	Assumption
μ	0.41	Per year	Assumption
α	0.975	Per year	Assumption
b	0.41	Per year	Assumption

Table 3 Description of parameters of the model (3)

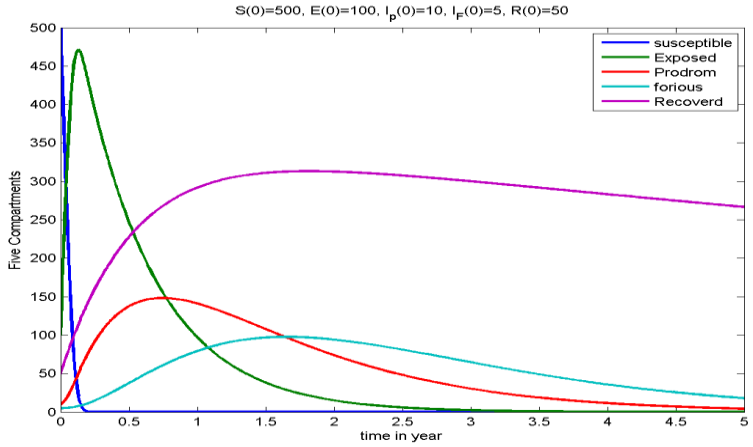

Figure 4: Numerical simulation of dog rabies model with vaccination rate $\boldsymbol{\theta = 0.9}$ and reproduction number $\boldsymbol{R_0 = 0.707}$

The population dynamics of SEI_PI_FR epidemic compartmental model with $R_0 = 0.7$ and $\theta = 0.9$ are considered. The susceptible decreases rapidly, whereas the number of exposed dogs is increasing rapidly to about 471 by the end sixth week and starting decreases to zero when the time approaches to 3 year. Also the number of infective with early symptom compartment dogs are increasing continuously to about 148 by the beginning of the ninth month and decreases and then approach to zero when the time approach to 5 year. The number of infected with later symptom compartment dogs are increasing continuously to about 98 and starts decreases to about 18 when the time approach to 5 year. Recover compartment increases steadily and approaches to 313 when the time approach to 1.8 years or 21.6 month and starting slowly decreasing to about 267 when the time approaches to five year. Finally the epidemic seems dies out.

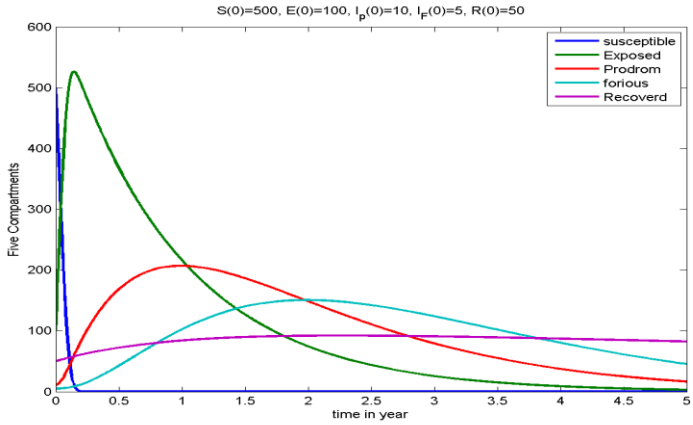

Figure 5: Numerical simulation of dog rabies model with vaccination rate $\boldsymbol{\theta = 0.1}$ and the reproduction number $\boldsymbol{R_0 = 1.22}$

We analyzed the numerical solution of dog rabies with vaccination rate $\theta = 0.1$ and the other parameters are the same as in figure 4**.** Then we get $R_0 = 1.22$**.** The susceptible compartment decreases rapidly whereas the number of exposed dogs is increasing rapidly to about 526 by the beginning of seventh week and starts to decrease to about 3 when the time approaches to 5 year. The number of infective with early symptom compartment dogs are increasing continuously to about 207 by the beginning of first year and starts decreases approach to 17 when the time approach to 5 years. The number of infected with later symptom compartment dogs are increasing continuously to about 151 by the beginning of second year and starts decreases to about 46 when the time approach to 5 years. Recover compartment slowly increases approaches to 92 when the time

approach to 2.28 years or 27.36 months and slowly decreasing to about 82 when the time approaches to five year.

V. Conclusions

In the present study we have formulated and analyzed a deterministic mathematical model for the dynamics of rabies transmission. Vaccination of dogs is the best controlling strategy for rabies disease. Increasing the vaccination coverage will decrease in the rate of transmission of rabies diseases. The basic reproduction number has been computed using next generation matrix method. We discussed the existence and stability of the disease free equilibrium points driven by using the Routh - Hurwitz criteria. The diseases free equilibrium points are shown locally asymptotically stable. Also disease endemic equilibrium point of the model has been derived. Simulation study and analysis of the model are performed by varying the vaccinated rate. It is observed that increasing of the vaccination rate of rabies has a significant impact on the rate of spread of rabies transmission. Further, on increasing the number of recovered in the model and reducing the vaccination rate of rabies had decreased the number of recovered in the model.

References

[1]. Wilkinson L. Introduction in Rabies. Edited by J. B. Campbell and K. M. Charlton. Boston: Kluwer Academic Publishers, 1988: 1–23.
[2]. Anderson R. M. May R. M. Infectious diseases of humans: Dynamics and Control. Oxford: University Press, 1991.
[3]. WHO expert consultation on rabies, Second report in WHO technical report series. Geneva: World Health Organization, 2013.
[4]. H. W. Heathcoat. The basic epidemiology models: Models expressions for R_0, parameter estimation and applications, 2006.
[5]. Harris G. Where streets are thronged with strays baring fangs. The New York Times, August 6, 2012.
[6]. McKenna M. Rethinking rabies: Experts urge mass dog vaccination to eradicate rabies. Scientific American, Jul 17, 2013.
[7]. N. Chitins, J. Hyman and J. Cushing. Determining important parameters in the spread of malaria through the sensitivity analysis of a malaria model. Bull. Math. Biology, 70, 2008, 1272 - 1296.
[8]. Hemachudha T, Laothamatas J, Rupprecht C E. Human rabies: a disease of complex neuropath genetic mechanisms and diagnostic challenges. The Lancet Neurology 2002, 1 (2): 101 – 109.
[9]. Kari Signor, Pankhurst, associate professor. Rabies: Its ecology, control, and treatment. Publication 420 – 036.
[10]. Bernoulli D., Blower S. An attempt at a new analysis of the mortality caused by smallpox and of the advantages of inoculation to prevent it. Reviews in Medical Virology, 2004, 14, 275 – 288.
[11]. Breuer, F. Castillo – Chavez. Mathematical models in population biology and epidemiology. New York, Springer, 2001.
[12]. M. Addo. An SEIR mathematical model for dog rabies, case study, Bongo district, Ghana. M.Sc. dissertation. Kwame Nkrumah University of Science and Technology, 2012.
[13]. Anderson R. M. and May R. M. Infectious diseases of humans: dynamics and control. Oxford University Press, 1991.
[14]. Ethiopian health and nutrition research institute (EHNRI). Rabies case report 1990 – 2010. EHNRI, Addis Ababa, Ethiopia, 2011.
[15]. Tesfaye Tadesse Ega, Livingstone S. Luboobi and Dmitry Kuznetsov. Modeling the dynamics of rabies transmission with vaccination and stability analysis. Applied and Computational Mathematics, 2015.
[16]. World Health Organization expert consultation on rabies, Second report. World Health Organization technical report series, p – 1, 2013.
[17]. Hemachudha T, Laothamatas J, Rupprecht C. E. Human rabies: a disease of complex neuropath genetic mechanisms and diagnostic challenges. The Lancet Neurology 2002, 1 (2), 101-109.
[18]. Purnachandra Rao Koya and Dejen Ketema Mamo. Ebola Epidemic Disease: Modelling, Stability Analysis, Spread Control Technique, Simulation Study and Data Fitting. Journal of Multidisciplinary Engineering Science and Technology (JMEST), Vol. 2, Issue 3, March 2015, pp 476 – 84. ISSN: 3159 – 0040. http://www.jmest.org/wp-content/uploads/JMESTN42350548.pdf
[19]. Dancho Desaleng, Purnachandra Rao Koya. The Role of Polluted Air and Population Density in the Spread of Mycobacterium Tuberculosis Disease. Journal of Multidisciplinary Engineering Science and Technology (JMEST). Vol. 2, Issue 5, May – 2015, Pp 1212 – 20. ISSN: 3159 – 0040. http://www.jmest.org/wp-content/uploads/JMESTN42350782.pdf
[20]. Tadele Degefa Bedada, Mihretu Nigatu Lemma and Purnachandra Rao Koya. Mathematical Modeling and simulation study of Influenza disease. Journal of Multidisciplinary Engineering Science and Technology (JMEST). Vol. 2, Issue 11, November 2015, Pp 3263 – 69. ISSN: 3159 – 0040. http://www.jmest.org/wp-content/uploads/JMESTN42351208.pdf
[21]. S. N. Sivanandam and S. N. Deepa. Linear system design using Routh column polynomials. Songklanakarin J. Sci. Technol. 2007, 29 (6), 1651 - 1659.