# Mathematical Modelling on Epidemics with A Reference To Deterministic Approach

**M. L. Mallikarjuna[1], V.K Katiyar[2], K.S. Basavarajappa[3], T. K. Krishna Kumar[4]**

1. Department of Physics, Adichunchanagiri Institute of Technology, Chickmagalore
2. Department of Mathematics, Indian Institute of Technology, Roorkee
3. Department of Mathematics, Bapuji Institute of Engineering and Technology, Davangere
4. Department of Physics, Bapuji Institute of Engineering and Technology, Davangere

**ABSTRACT**

The study involves the investigation of the disease through human population in reference to spread of simple epidemics. Analysis consists of mathematical formulations to compute the population of Susceptible S(t) and Infectives I(t) using deterministic approach in the presence of Carriers C(t). The case of removals R(t) is introduced into human population to remove the number of infectives.Control mechanism in the case of spreading of the epidemic disease consists of subpopulation (infected) in the main population S(t). The epidemic model is constructed to indicate the change in the composition of a population in which initially the number of individuals having the disease will be decreasing.We formulate the deterministicapproach which gives quantitatively S(t) and I(t) with reference to various time intervals(01 to 12 days). A special reference to malaria caused by female mosquitoes bite is considered in the model with suitable assumptions. Malaria parasites are released into the blood stream so that the RBC cells from the infected individuals enter the normal individual. Further these parasites reproduce next generation of parasites and spreads to normal healthy individuals. The reference of rise in the human body temperature due to malaria causes a severe disorder in the digestion process. The major change in the health of human population due to spread of epidemics will mainly damage the digestive system. This ultimately leads to fatigue so that the effects of infected people will become dominant causes an Influenza which will spread to the other normal human population. Based on the assumptions proposed we construct the set of ordinary differential equations for change in susceptible population S(t) and the change in infectives I(t) with respect to time in relation to carriers and removals. Numerical scheme is introduced to quantify the values of S(t), I(t) in relation to C(t) and R(t) at various time intervals. Results give the evidence of the existence of the relations between S(t), I(t),C(t) and R(t) populations. Runge – Kutta – Fehlberg fourth order method is employed for numerical computations.

**Key words**: Epidemic, Pollution, Susceptible, Infectives

**Address for Correspondence:**
**Dr. K. S Basavarajappa**
Professor and Head, Department of Mathematics, Bapuji Institute of Engineering and Technology, Davangere-04
email:ksbraju@hotmail.com

**INTRODUCTION**

Pollution occurs in the nature in various forms such as: industrial pollution, air pollution, water pollution, soil pollution, noise pollution. The present study is aimed at the spread of epidemics due to industrial pollution by which female mosquito bite is primarily responsible for disease malaria.Human malaria is directly caused by four species of protozoa in the genus plasmodium, and they are transmitted to man only by the female mosquitoes belonging to certain species of the genus anopheles. Conversely, female mosquitoes can pick up the infection when they bite infected humans. Only female mosquitoes are involved since only they suck the blood: males take their liquid meals from fruit juices and elsewhere. In the case of malaria parasite it is, on the accepted definitions, the mosquito which is the final host, i.e., where the fertilized eggs are produced.Man is the intermediate host harbouring the asexual phase of development.Dueto the effects of industrial pollution on the surrounding vegetarianthe life can always be determined by estimating the extent of pollution in the given industrial domain or land area. The bursting of the RBC's and the release of malaria parasites into the blood stream occur with such great regularity that we can speak of generations of parasites. The zero$^{th}$ generation is the first set of parasites which enter the red blood cells. The first generation is the set of parasites which are produced by bursting these cells and which enter more RBC's. These in turn reproduce, giving the second generation of parasites, and so on.Therefore the pollution may be in-terms of chemical or biological waste, polluted land, particulate matter and water resources. However, in addition to above types of pollutants there are many indirect pollutants in industrial habitat areas which also cause health hazards. The affected population becomes prone to catch viral and bacterial infections and is susceptible for various diseases. It is also to be noted that the spread of infectious disease is not uniform in the entire industrial population. For a general consideration we consider workers of different categories with differences in the financial status. Due to unhygienic equipment, a sort of social pollution is prevalent in much industrial pollution. For this type of population with low paid unskilled workers, generally, transmitted diseases in the industrial area are very common. But the sections other than the above have better living conditions and medical care, the recovery rate is better.

Kermack et al. [1] explained the mathematical theory of epidemics. Bailey [2] described the mathematical theory of infective disease. Hethcote [3] explained the qualitative analysis for communicable diseases models. G. F. Raggett [4] validate the standard epidemic model on real data to compare the vital parameter with reality. May et al.[5] discussed the epidemics of sexually transmitted infection in heterogeneous population.Saxena N P [6] analyzed the industrial pollution, population and spread of infectious diseases.Satsuma et al.[7] discussed the extension of the susceptible-infective-removal (SIR) epidemic model. Basavarajappa et al.[8] explained the mathematical model by coupled solutions differential equation for the spread of infectious disease when un-dissolved heavy metals enter the pancreas through the drinking water and the vegetables when untreated. Phenyo et al.[9] explained the statistical inference in a stochastic epidemic SEIR model with control intervention. Colizza et al.[10] explained the modeling of global epidemics with stochastic dynamics and predictability. C.Chiyaka et.al [12] studied the transmission model of human malaria in a partially immune population with three discrete delays is formulated for variable host and vector populations.J.D.Chapman et. al. [13] considers the implications of a structural identifiability analysis on a series of fundamental three-compartment epidemic model structures, derived around the general SIR (Susceptible-Infective-Removal) framework.Nikolaos P et. al. [17] studied the deterministic model for large populations, where random interactions can be averaged out, is used for the epidemic's rate of spread. Aresh Dadlani et al. [18] explained the population is divided into compartments based on the health status of each individual. Mick Roberts et. al. [21], discussed the nine challenges and need in the epidemic models.Abraham J et. al. [22] used the fractional derivatives by Non Standard Finite Difference (NSFD) schemes to obtain numerical solutions of the susceptible–infected (SI) and Susceptible–Infected–

Removal (SIR) fractional-order epidemic models.Yi Wang et. al [23] analyzed the SEIR dynamics with or without infectious force in latent period on random networks by using the probability generating functions, both of these models are governed with nonlinear systems of intrinsically three dimensional ordinary differential equations, which have the same dimension as the classical SEIR models. The simplest scenario for an epidemic is one in which the population is split into two parts, one is the infected and the other is part that is susceptible to being infected. We set the assumptions that: there are no people who are immune and all the infected people are infectious and vice versa. The infection is spread by contact between members of the community in which there is no removal from circulation by death recovery or isolation. Ultimately, therefore all susceptible become infected. We consider a population in which a small number of members become capable of spreading infectious diseases. To expose the details of the effect in the case of the carrier disease malaria, we construct system of linear differential equations usinginfectives I(t) and susceptible S(t) for various time intervals. The analytical solution of the constructed equations strongly give the evidence of the existence of the relation between the infections and infected numbers. Respectively S(t) and I(t) give the evidence that infective will become capable of spreading the disease at a faster rate for normal population. We consider in deterministic models one takes the actual number of new cases in short interval of time to be proportional to the numbers of both susceptibles and infectives cases, as well as to the length of interval. The population under consideration is divided into disjoint classes which will vary with time t. We have assumed that all the parasites released into the blood stream survive. This is not a reasonable assumption biologically. Suppose for simplicity that a fixed proportion of the released parasites survive and enter new RBC's. So from the initial parasitized RBC's, some parasites are released and a fraction of those survive. The susceptible S(t) class consists of those individuals who can catch the disease but not yet infective. We assume that the number offspring in each generation is fixed, but that the proportion of survived off-springs varies. Also this survival proportion depends only on the generation n. The infective I(t) class consists of those who are transmitting the diseases to others. The variation in the reproduction changes from generation to generation. The removable class R(t) consists of those who are removed from the susceptible – infective interaction by recovery with immunity, isolation or death. The fraction of the total population of these classes are denoted by S(t), I(t) and R(t) respectively as susceptible, infectives and removals.

A comparison of quantitative approach for establishing the infected population from I(t) to the quantitative approach for the susceptible population S(t) respectively taken as SIS and SIR epidemic models. In the deterministic approach we assume the following,

(i) S(t): That subpopulation composed of individuals who are uninfected and susceptible to the infection.
(ii) E(t): That subpopulation composed of individuals who are infected but who are not yet capable of spreading the infection.
(iii) I(t): That subpopulation composed of individuals who are infected and actively spreading the disease.
(iv) R(t): That subpopulation composed of individuals who are not susceptible or who have been infected and either subsequently cured or removed from possible contact with members of S(t).

Various mathematical investigations are used in medicine for the computations of populations for epidemic models and estimation of new probable infectives.

Formulations for S(t) and I(t) in relation to C(t) and R(t) indicate the details of effect of industrial pollution on the human population.

**FORMULATION AND ANALYSIS**

Following the assumptions on the populations of S(t), I(t), C(t) and R(t) at various time intervals we formulate the basic differential equations.An infectious disease introduced into a closed population. It is required to compute how many individuals will catch the disease.Any individual gets recovered from the disease will have the permanent immunity.

Introducing the population as I(t) - infectious, S(t) - susceptible, R(t) - removable and C(t) - carriers such that, population consists of I and S only, S(t) becomes infected and remain infected, I + S = C (constant), nobody is leaving the place, I and S are function of t (time), No control mechanism.S(t) and I(t) are time dependent representation of populations. The positive coefficients β, γ, δ are infection rate constant,susceptible rate constant and the removable rate constantrespectively. Mathematical study of the infectious diseases including epidemics due to industrial pollution is mostly confined to homogenous population consisting of single group. As for as population model is concerned, we have considered biological populations with two compartments in the same species. The same model is carried out to investigate on the effect on human population. The present analysis concerns the mathematical model for S(t) and I(t) using deterministic approach.

**For Susceptible S(t),**

$$\frac{dS}{dt} = -\beta S(t) C_0 e^{-\delta t} + \gamma (n - S) \quad (1)$$

Over a period of time 0 to t (i.e., number of days taken depending on the type of disease under consideration), introducing the initial conditions,$m_1$ is calculated then S(t) is given by

$$S(t) = \left[e^{\frac{\beta C_0 e^{-\delta t}}{\delta} - \gamma t} S_0 e^{\frac{-\beta C_0}{\delta}}\right] + \left[e^{\frac{\beta C_0 e^{-\delta t}}{\delta} - \gamma t} \int_0^t \gamma N \; e^{\frac{-\beta C_0 e^{-\delta t}}{\delta} + \gamma t} dt\right] \quad (2)$$

To estimate the varying population of S (t), we expressing in series form,

$$S(t) = S_0 e^{\frac{\beta C_0 e^{-\delta t}}{\delta} - \gamma t - \frac{\beta C_0}{\delta}} + \left((\gamma N e^{\frac{\beta C_0 e^{-\delta t}}{\delta} - \gamma t}\right)\left(\left[\frac{e^{(\gamma t)}}{\gamma}\right] + \left[\frac{-e^{(\gamma-\delta)t}}{(\gamma-\delta)}\right] + \left[\frac{e^{(\gamma-2\delta)t}}{2!(\gamma-2\delta)}\right] + \left[\frac{-e^{(\gamma-3\delta)t}}{3!(\gamma-3\delta)}\right] + -e(\gamma-4\delta)t4!(\gamma-4\delta) \quad (3)$$

For Non-homogenous population of size N, we can choose constants S(t) as,

$\delta = 1, \beta = 1, \gamma = 1, C_0 = 1$, N = 20, $S_0$ (t) = 5,

$$S(t) = [S_0 e^{(e^{-t} - t - 1)}] + \left((20e^{(e^{-t} - t)}\right)\left(\left[e^{(t)}\right] + \left[\frac{-e^{t}}{4}\right] + \left[\frac{e^{-2t}}{12}\right] + \left[\frac{-e^{3t}}{72}\right] - 0.8194\right) \quad (4)$$

For Homogenous population, S(t) becomes,

$$S(t) = S_0 e^{-\left(\beta C_0 e^{-\delta t} - \gamma t\right)} \quad (5)$$

Also by Runge – Kutta –Fehlberg method or embedded R-K method or Cash Karp R – K method, the solution as set of tabulated values by ignoring 6th value of K. Since the value lies beyond the range of reference on t – axis, we have modelled the governing equations of R-K-F 6th order method as,

$$S_{i+1} = S_i + K_j \quad (6)$$

Where ( i , j) : (1,1),(2,2),(3,3),(4,4) and (5,5)

$$K = h\left[\frac{2825}{27648} K_1 + \frac{18575}{48384} K_2 + \frac{13525}{55296} K_4 + \frac{277}{14336} K_5 + \frac{1}{4} K_6\right] \quad (7)$$

**For Infectives I(t),** we derive the expression in the deterministic approach as,

$$\frac{dI}{dt} = \beta S(t) C(t) - \gamma I(t) \quad (8)$$

The solution is given by

$$I(t) = \left[e^{\frac{\beta C_0 e^{-\delta t}}{\delta} - \gamma t} I_0 e^{\frac{-\beta C_0}{\delta}}\right] + \left[e^{\frac{\beta C_0 e^{-\delta t}}{\delta} - \gamma t} \int_0^t \beta C_0 e^{-\delta t} N \;\; e^{\frac{-\beta C_0 e^{-\delta t}}{\delta} + \gamma t}\, dt\right] \quad (9)$$

To estimate the varying population of I(t), we expressing in series form,

$$I(t) = [I_0 e^{\frac{\beta C_0 e^{-\delta t}}{\delta} - \gamma t - \frac{\beta C_0}{\delta}}] + \left((\gamma N e^{\frac{\beta C_0 e^{-\delta t}}{\delta} - \gamma t}\right)\left(\left[\frac{-e^{(\gamma-\delta)t}}{(\gamma-\delta)}\right] + \left[\frac{e^{(\gamma-2\delta)t}}{(\gamma-2\delta)}\right] + \left[\frac{-e^{(\gamma-3\delta)t}}{2!(\gamma-3\delta)}\right] + \left[\frac{-e^{(\gamma-4\delta)t}}{3!(\gamma-4\delta)}\right]\right) \quad (10)$$

For Non - homogenous population of I(t) (as I(t) increases),

$\delta = 1, \beta = 1, \gamma = 1, C_0 = 1$, N = 20, $I_0(t) = 3$

$$I(t) = [I_0 e^{(e^{-t} - t - 1)}] + \left((20 e^{(e^{-t} - t}\right)\left(\left[e^{-(t)}\right] + \left[\frac{e^{-2t}}{8}\right] + \left[\frac{-e^{3t}}{18}\right] - 0.9201\right) \quad (11)$$

For Homogenous population, I(t) becomes,

$$I(t) = I_0 e^{-\left(\beta C_0 e^{-\delta t} - \gamma t\right)} \quad (12)$$

Set of numerical approximation for I(t) are obtained as,

$$I_{i+1} = I_i + K_j$$

Where ( i , j) : (1,1),(2,2),(3,3),(4,4) and (5,5)

$$K = h\left[\frac{2825}{27648} K_1 + \frac{18575}{48384} K_2 + \frac{13525}{55296} K_4 + \frac{277}{14336} K_5 + \frac{1}{4} K_6\right] \quad (13)$$

By defining R(t) as the removal population as the infected rate attains the population S(t) from the initial population, we can model for removal population as,

$$\frac{dR}{dt} = \gamma I(t) \quad (14)$$

Here $R(t = t_0 = 0) = R_0 = 0$ , initially at $S_0 = 0$ , then by using the initial condition,

S(t) + I(t) +R(t) = constant = n + 1

Choosing balancing conditions, $\gamma = 1$, N= 20 , $R_0 = 9$

$$R(t) = 16\, t - 9\, e^{-\gamma t} \quad (15)$$

As $t \to \infty \approx$ 12 days to 32 days then, the removal rate is not in control, the model behaves to indicate the increase of I(t). If removal is in control then I(t) decreases. Obviously S(t) increases which causes the decrease in infection.

For the case of Mosquitoes population, infection rate is analyzed,

$$\frac{dM}{dt} = \delta (S - I) M + K M_0 \quad (16)$$

For solving this we obtain,

$$M = e^{\delta(S-I)t}\left(\int_0^t K M_0\, e^{\delta(S-I)t}\, dt\right) + M_0$$

For Non - homogenous population, M(t) becomes,

$$M = K M_0\, e^{\delta(S-I)t}\left(t - \frac{\delta(S-I)t^2}{2} + \frac{\delta(S-I)^3 t^3}{6} - \frac{\delta(S-I)^4 t^4}{24}\right) \quad (17)$$

M is Calculated for S – I = +1, S – I = -1

For Homogenous population, M(t) becomes,

$$M(t) = M_0 e^{\delta(S-I)t} = M_0[\, 1 + \frac{\delta(S-I)}{1!} + \frac{\delta(S-I)^2}{2!} + \frac{\delta(S-I)^3}{3!} + \cdots \quad (18)$$

Proposed mathematical model gives computation for M(t) for the case of S-I = -1, ( Non homogeneous :({S(t), I(t)} or{S(t), R(t)} or{R(t), I(t)} or{S(t), M(t)} or {M(t), I(t)}or{R(t), M(t)}or {S(t), I(t),M(t), R(t)}) as M(t) decreases Fig: S-I = -1, due to imposing of R(t). As a result S(t) increases.

The consequence of the decrease of M(t) also indicates the recovery rate appears upto8 days and M(t) decreases there onwards.Fig:3. S-I = 1. This shows the good agreement for the hypothesis of the present model that there exists a relation between S(t) and I(t) for zero$^{th}$ generation onwards to the present generation (n) and the carriers C(t) spread the disease to the normal individuals in (n+1)$^{th}$

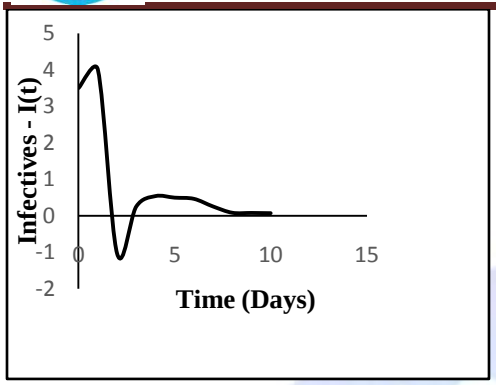
generation as parasites of female mosquitoes will enter the normal individuals for further spread of disease.

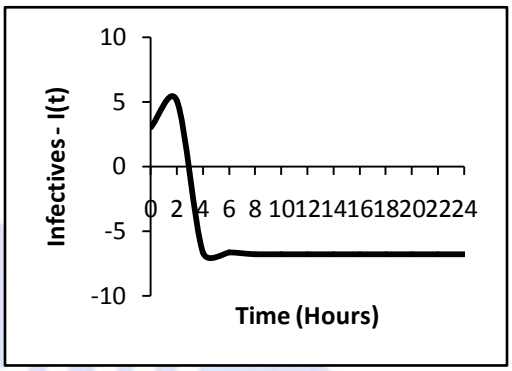
For homogeneous population :({S(t)} or { I(t)} or {R(t)} or M(t)}) , M(t) increases at S- I = 1 as the susceptibility decreases at S – I = -1. This also agrees with the above hypothesis.

**RESULTS AND DISCUSSION**

Mathematical model is studied to quantify the susceptible and infectives populations using deterministic approach. It predicts the number of Infectives I(t) for the corresponding Susceptible S(t) in the population where the disease is infectious but not sufficiently seriousFig (2) andFig (1).Using analytical method the computations for S(t) and I(t) show the continuous increase of I(t) in the absence of R(t), then infected individuals remain in the population to spread the disease.Numerical method R-K-F for $t_1$ to $t_5$ shows the smoothness of the graph approach which indicates the further validity of assumptions proposed. This is compared with [14]. There appears the sudden inflection downward in Fig. 3 (b) for the case of removals Fig. 3(a).

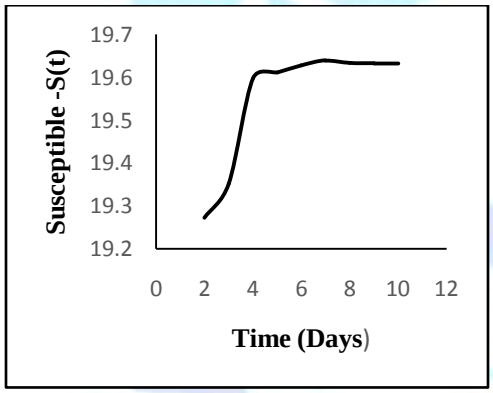
The computation for S(t) shows that when one in the total population is infected and other is the part that, few members are susceptible to become infected. For no removal case or the recovery rate is low, Fig. 4(a) andFig. 5, we can notice the long term effect of pollution in industrial towns on the human population in terms of spread of diseases. The population parameters I(t) and S(t) are expressed by introducing R(t), Fig. 4(b) give the control of epidemics. It is reasonable to assume that the individuals under the influence of pollutants remain infected forever.This means there is no recovery from the pollution while the recovery of diseases of infected members has been considered.

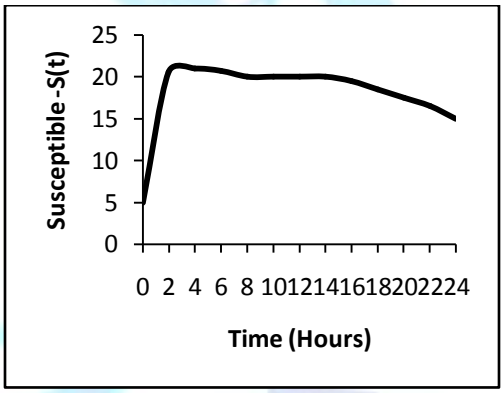



(a) R-K-F method (b) Non homogenous method

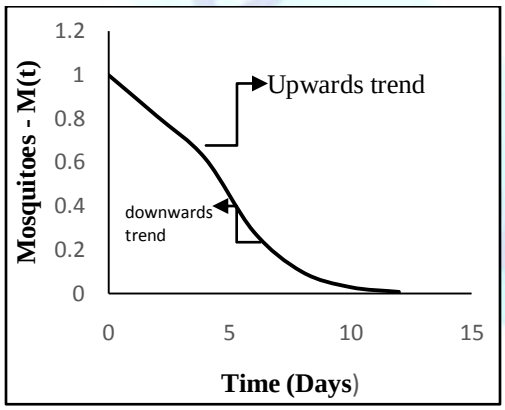
Fig: 1: Infectives - I(t) Vs Time (t)

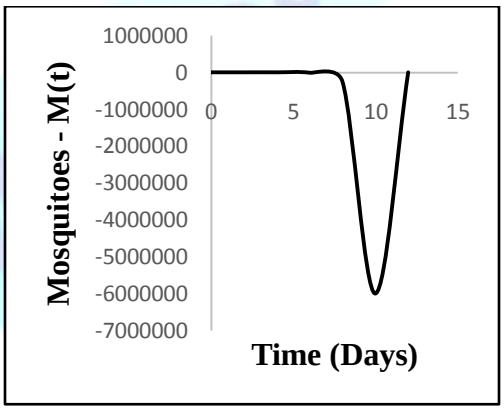



(a) R-K-F method (b) Non homogenous method

Fig.2: Susceptible – S(t) Vs Time (t)




(a) S – I = -1 (b) S – I = 1

Fig.3 (a). Fig.3 (b)

Fig.3: Mosquitoes – M (t) Vs Time (t), Non-Homogenous method

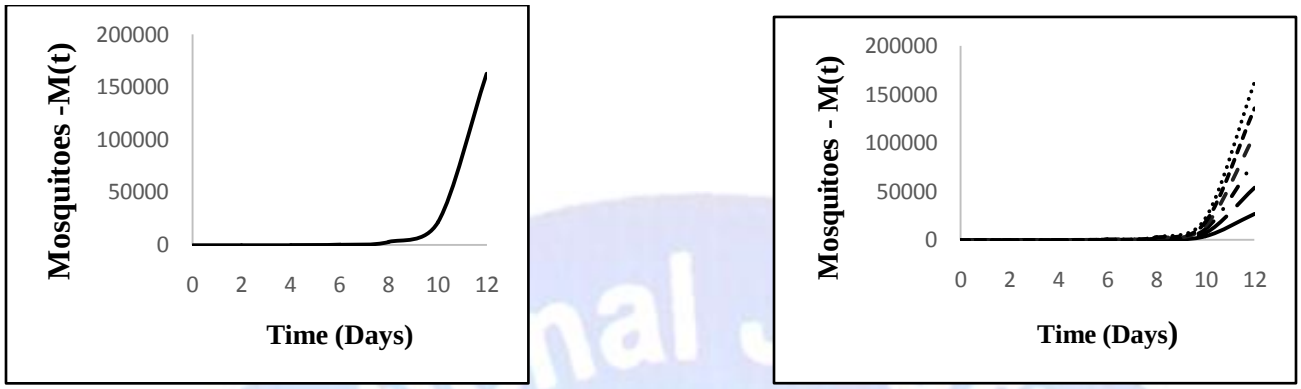



Fig.4 (a): S – I = 1, $M_0$ = 1/6, 2/6, 3/6, 4/6, 5/6, 1

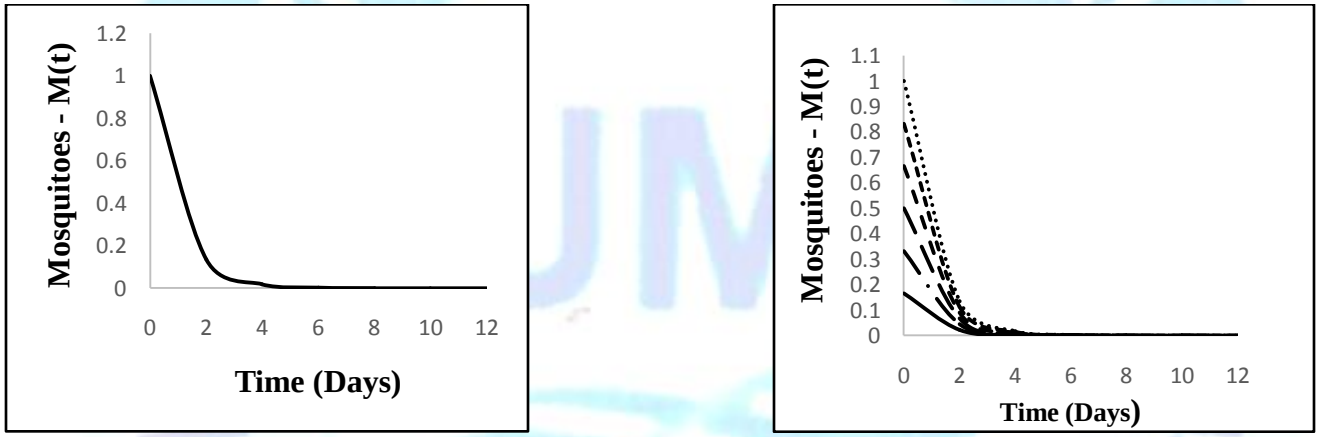



Fig.4 (b): S – I = - 1$M_0$ = 1/6, 2/6, 3/6, 4/6, 5/6, 1

Fig.4: Mosquitoes – M(t) Vs Time (t), Homogenous method

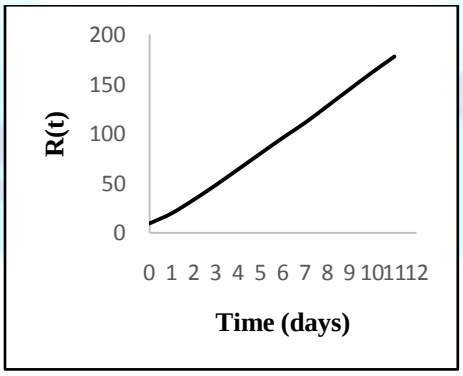



Fig: 5: Removal – R (t) Vs Time (t):Non homogenous method

**REFERENCES**

1. Kermack W O, MC - Kendrick A G [1927], 'Contributions to the mathematical theory of epidemics', Proc. Roy. Soc. A. 115, 138,100 - 721.
2. Bailey N T J [1975], 'The mathematical theory of infectious disease, (II edition), Harnam
3. Hethcote H W [1976], 'Qualitative analysis for communicable diseases models', Math. Biosci, Vol. 28, 335 - 356.
4. G.F. Raggett [1980], 'On the iterative solution of the limiting susceptible population in a model of epidemiology', Journal of Applied Mathematical Modelling, 4,479-482.
5. May R M, Jose M V, Anderson R M [1987], 'Epidemics of sexually transmitted infection in heterogeneous population', Journal of Theoretical Biology.
6. Saxena N P [1993], 'Industrial pollution, population and spread of infections diseases', SIAM, 319 – 326.
7. Satsuma J, Willox R, Ramani A, Grammaticos B and Carstea A S [2003], 'Extending the SIR epidemic model', J. Physica, Vol. 12, 20 - 23.
8. Basavarajappa K S [2005], 'Effect of fluoride ion concentration in water, surface soil and vegetables of three fluoride affected villages in Davangere district in southern part of India', International Conference on water and health, SJEC, Mysore.
9. Phenyo E. Lekone and Barbel F. Finkenstadt [2006], 'Statistical inference in a stochastic epidemic SEIR model with control intervention', Biometrics, Vol. 62, 1170 - 1177.
10. Colizza V, A Barrat, M Bartheleme, A Vestignani [2006], 'The modeling of global epidemics with stochastic dynamics and predictability', Bulletin of mathematical Biology, Vol. 68, 1893 - 1921.
11. Vittoria Colizza, Marc Barthelemy, Alain Barrat, Alessandro Vespignani [2007], 'Epidemic modeling in complex realities', C. R. Biologies, Vol. 330, 364-374.
12. C. Chiyaka, W. Garira, S.Dube, [2007], 'Transmission model of endemic human malaria in a partially immune population', Journal of Mathematical and Computer Modelling, 46,806-822.
13. J. D. Chapman, N. D. Evans, [2008], ' The structural identifiability of SIR type epidemic models with incomplete immunity and birth targeted vaccination',Proceedings of the 17th World Congress, The International Federation of Automatic Control Seoul, Korea, July 6-11, 9075-9080.
14. Lourdes Esteva, et.al [2009], 'Qualitative study transmission dynamics of drug resistant malaria', Mathematical and Computer modelling, 50, 611-630.
15. Michael J. Jeger et.al [2009], 'The effect of transmission route on plant virus epidemic development and disease control', Journal of Theoretical Biology, 258,198-207.
16. C. Okais et.al [2010],'Methodology of the sensitivity analysis used for modeling an infectious disease', Vaccine, 28, 8132-8140.
17. Marija Krsti, [2011], ' The effect of stochastic perturbation on a nonlinear delay malaria epidemic model', Mathematics and Computers in Simulation 82,558–569
18. Nikolaos P. Rachaniotis, Tom K. Dasaklis, Costas P. Pappis, [2012]. 'A deterministic resource scheduling model in epidemic control: A case study, European Journal of Operational Research, 216, 225–231
19. Aresh Dadlani et.al [2013] 'Deterministic Models in Epidemiology: from Modelling to Implementation', World Class University programme, GIST, South Korea, 1-30.

20. Fabio A.C.C. Chalub, Max O. Souza [2014], ´Discrete and continuous SIS epidemic models: A unifying approach' Ecological Complexity, Volume 18, 83–95.
21. L. Bolzoni, A. Pugliese, R. Rosa, [ 2015] 'Asymptotically constant; Demographic Allee effect; Geometric growth; Persistence,The role of heterogeneity on the invasion probability of mosquito-borne diseases in multi-host models', Journal of Theoretical Biology, Volume 377, 25–35.
22. Mick Roberts, Viggo Andreasen, Alun Lloyd, Lorenzo Pellise,[2015], Nine challenges for deterministic epidemic models, Journal of Epidemics,10, 49-53.
23. Abraham J. Arenasa, Gilberto Gonz´alez-Parrab, Benito M. Chen-Charpentierc [2016], 'Construction of nonstandard finite difference schemes for the SI and SIR epidemic models of fractional order', Journal of Mathematics and Computers in Simulation 121, 48–63.
24. Yi Wang, Jinde Cao, Ahmed Alsaedi, Bashir Ahmad, [2017] 'Edge-based SEIR dynamics with or without infectious force in latent period on random networks' Journal of Communication Nonlinear Sci. Numerical Simulation,45, 35-54.
25. 'Nomography and Empirical Equation',[1962],Dale S.Davis,New York, Reinhold Pub. Corp.
26. 'Mathematical Biology',[2004], J.D. Murray, Springer India Limited, 660-681.
27. 'Mathematical models in Biology and Medicine', [1992], J.N. Kapur, East west press Private Limited,257-285.

**Nomenclature:**

S(t) - Susceptible
I(t) - Infectives
R(t) - Removals
C(t) - Carriers
M(t) - Mosquitoes population
β - Infection rate constant
γ - Susceptible rate constant
δ - Removable rate constant
N - Number of Populations
T - Time