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Mathematical Modeling of Gonorrheal Disease a Case Study with Reference to Anantapur District-Andhrapradesh-India

By Dr. G.Prabhakararao
S.V.G.M. Government Degree College, India

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Keywords : *modeling, infectious diseases, epidemics, stratified populations, susceptible, chain-branched reactions.*

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4. W.O.Kermark and A.G.Mc.Kendrick,A contribution to the theory of epidemics Proc.Roy.Soc. (A) 115 (1923), 700-721,139(1932), 55

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Abstract - In this paper we have analyzed the Mathematical modeling of Gonorrheal disease, the spread of a contagious disease involves interactions of two populations: the susceptible and the infectives. In some diseases these two populations are from different species. For example, malaria is not passed directly between animals but by the anopheline mosquitoes, and schistosomiasis is passed from animal to animal only through contact with water in which live snails that can incubate the disease-causing helminthes. In other diseases, the infection can be passed direct from invectives to susceptible: Viral diseases like chicken-pox, measles, and influenza, and bacterial diseases like tuberculosis can pass through a population much like flame through fuel.

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I. Introduction

The spread of a contagious disease involves interactions of two populations: the susceptible and the infectives. In some diseases these two populations are from different species. For example, malaria is not passed directly between animals but by the anopheline mosquitoes, and schistosomiasis is passed from animal to animal only through contact with water in which live snails that can incubate the disease-causing helminthes. In other diseases, the infection can be passed direct from invectives to susceptible: Viral diseases like chicken-pox, measles, and influenza, and bacterial diseases like tuberculosis can pass through a population much like flame through fuel.

There are useful analogies between epidemics and chemical reactions. A theory of epidemics was derived by W.O.Kermack, a chemist, and A.G.Mc Kendrick, a physician, who worked at the Royal college of Surgeons in Edinburgh between 1900 and 1930.They introduced and used many novel mathematical ideas in studies of populations [4].One important result of theirs is that any infection determines a threshold size for the

Author : Department of Mathematics, S.V.G.M. Government Degree College, Kalyandurg, Anantapur-District, Andhra Pradesh, India. E-mail : nari.prabhu@gmail.com

susceptible population, above which an epidemic will propagate. Their theoretical epidemic threshold is observed in practice, and it measures to what extent a real population is vulnerable to spread of an epidemic. At roughly the same time V.I.Semenov [6] derived a theory of combustion that identified explosion meets beyond which combinations of pressure and temperature cause chemicals to begin explosive chain-branched reactions

These models can serve as building blocks to study other diseases, for example ones having intermediate hosts, and diseases in stratified populations, for example where there are mixing groups that have various contact probabilities, like families, preschools, and social groups. Some of these extensions are described in the exercises

II. Spread of an Epidemic and Deterministic Models

Consider the situation where a small group of people having an infectious disease mixes with a sizable population susceptible to carry the infection. The equations a use how many people will ultimately catch the disease? Will the infection spread rapidly or die out gradually? How does the epidemic evolve in time? In order to discuss this situation, we consider a fixed population and assume that three kinds of individuals in it exist viz, susceptibles, infectives and removals. Susceptibles can acquire the infection upon effective contact with an infective. Infectives have the disease and are capable of transmitting it. Removals are those who have passed through the disease process but are no longer susceptible or infective. We assume that a person who as permanent immunity and also the disease has a negligibly short incubation period. This implies that an individual who contracts the disease becomes infective immediately after words. The flow of the disease is describe as follows

$$S \rightarrow I \rightarrow R$$

Indicating that susceptible might become infectives, and invectives might be removed, but there is no supply of new susceptible to the processes.

We now begin with the Reed-Frost model which describes the spread of infection in population due to random sampling.

In a given population let S_n, I_n and R_n be the number of susceptible infected persons and removals at the n^{th} sampling time at the end of n^{th} sampling interval, the corresponding numbers are S_{n+1}, I_{n+1} and R_{n+1}.

The probability that a susceptible avoids contact with all of the infectives during the sampling interval is

$$q_n = (1-p)^{I_n}$$

Ref.

6. Martin Braun The threshold theorem of epidemiology, Differentail equations and their applications Springer-Verlag Publishers.

Therefore, the probability that

$$S_{n+1} = k \text{ is}$$

$$\binom{S_n}{k} q_n{}^k (1-p)^{S_n - k}$$

which is a simple binomial distribution. That is, the probability that k survive the sampling interval as susceptible is the number of ways k can be selected from among candidates times the probability that k avoid effective contact and the probability that have effective contact.

If

$$S_{n+1} = k \text{ then } I_{n+1} = S_{n+1} - k \text{ and } R_{n+1} = R_n + I_n$$

This calculation is summarized by the formula

$$\Pr[S_{n+1} = \frac{k}{S_n} \text{ and } I_n] = \binom{S_n}{k} q_n{}^k (1-p)^{S_n - k}$$

and this called the Reed-Frost model [1].

The Kermack-Mc Kendrick model [2] is a nonrandom model that describes the proportion in large populations.

Let x_n denote the number of susceptible, y_n the number of infectives and the number of removals. It might be expected that the number of susceptible in the next sampling interval would be

$$x_n = (1-p)^{y_n} x_n$$

since the factor $(1-p)^{y_n}$ is the proportion of susceptible who avoid effective contact with all infectives.

Let $a = -\log(1-p)$ so that e^{-a} is the probability that a given susceptible will successfully avoid contact with each infective during the sampling interval. Assume that the sampling interval is the same as the interval of infectiousness. Supposing that those leaving the susceptible class enter directly into the infectious class, and that a proportion, say b, of the infectives remain infective at the end of each sampling interval. Then

$$x_{n+1} = e^{-ay_n} x_n \text{ and}$$

Ref

1. N.T.S.Bailey, The theory of infectives disease and its application, Charle Griffin, London, 1975.

$$y_{n+1}=(1-e^{-ay_n})x_n+by_n$$

determine their numbers. This model is the Kermack-Mc Kendrick model.

We now consider simple deterministic models without removal [3].In a given population at time *t*, let s be the number of susceptible *S (t), I (t)* be the number of infected persons. Let *n* be the initial number of susceptible in the population and let one person is infected initially. Assuming that the rate of decrease of *S (t)* or the rate of increase of *I (t)*, is proportional to the product of the number of susceptible and the number of infected. We obtain a model

$$\frac{dS}{dt}=-\beta SI\ ,\frac{dI}{dt}=-\beta SI$$

Since *S (0)* is *n* and *I (0)* is one and *S (t) +I (t) =N+1*.we obtain

$$\frac{dS}{dt}=-\beta S(n+1-S)$$

Integrating using the initial conditions

$$S(t)=\frac{n(n+1)}{n+e^{(n+1)\beta t}}\ ,\ I(t)=\frac{(n+1)e^{(n+1)\beta t}}{n+e^{(n+1)\beta t}}$$

So that

$$\underset{n\to\infty}{Lt}S(t)=0,\ \underset{n\to\infty}{Lt}I(t)=n+1$$

and, ultimately, all persons will be infected.

A modification of above model may be obtain by assuming that a susceptible person can become infected at a rate proportional to *SI* and an infected person can recover and become susceptible again at a rate γI so that we get the model

$$\frac{dS}{dt}=-\beta SI+\gamma I\quad \frac{dI}{dt}=\beta SI-\gamma I \qquad (2.1)$$

which gives

$$S(t)+I(t)=N=S(0)+I(0)=S_0+I_0\,(I_0\neq 0) \qquad (2.2)$$

from (2.1) and (2.2)

$$\frac{dI}{dt}=(\beta N-\gamma)I-\beta I^2=kI-\beta I^2$$

Ref.
3. J.N.Kapur Mathematical models in Biology and Medicine,East-west press private limited 2000.

Integrating, we obtain

$$I(t)=\begin{bmatrix} \dfrac{e^{kt}}{\dfrac{\beta[e^{kt}-1]}{k}+I_0^{-1}} & (k\neq 0) \\ \dfrac{1}{\beta t+I_0^{-1}} & (k=0) \end{bmatrix}$$

As $t\to\infty$,

$$I(t)=\begin{bmatrix} N-\rho \;\; if \;\; N\succ\rho=\dfrac{\gamma}{\beta} \\ 0 \qquad if \;\; N\leq\rho=\dfrac{\gamma}{\beta} \end{bmatrix}$$

We now discuss the deterministic model taking into account the number of persons removed from the population by recovery, immunization, death, hospitalization or by any other means. We make use of the following assumptions [5].

I. The population remains at affixed level N in the time interval under consideration. This means, of course, that we neglect births, deaths from causes unrelated to the disease under consideration, immigration and emigration.

II.The rate of change of the susceptible population is proportional to the product of the number of *(S)* and the number of members of *(I).*

III. Individuals are removed from the infectious class *(I)* at the proportional to the size of *(I).*

Let *S(t), I(t),* and *R(t)* denote the number of individuals in classes *(S),(I),*and *(R),*respectively, at time t. It follows immediately form rules (I-III) that *S (t), I (t),* and *R (t)* satisfies the system of differential equations.

$$\frac{dS}{dt}=-rSI$$

$$\frac{dI}{dt}=rSI-\gamma I \qquad (2.3)$$

$$\frac{dR}{dt}=\gamma I$$

Ref

5. Classic Papers in the theory of epidemics.See also A.G.Mc.Kendrick, Mathematics applied to medical problems, Roy.Soc.Edinb.,1926.

For some positive constants *r* and γ.The proportionality constant *r* is called the infection rate, and the proportionality constant γ is called removal rate.

The first two equations of (2.1, 2.2) do not depend on R.Thus,we need only consider the system of equations.

$$\frac{dS}{dt} = -rSI, \quad \frac{dI}{dt} = rSI - \gamma I \tag{2.4}$$

For the two unknown functions *S (t)* and *I (t)*.Once *S (t)* and *I (t)* are known, we can solve for *R (t)* form the third equation of (2.3). Alternately, observe that $\frac{d(S+I+R)}{dt}$.

Thus,

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

so that *R(t)=N-S(t)-I(t),*

the orbits of (2.2) are the solution curves of the first-order equation

$$\frac{dI}{dt} = \frac{rSI - \gamma I}{-rSI} = -1 + \frac{\gamma}{rS} \tag{2.5}$$

Integrating this differential equation gives

$$I(S) = I_0 + S_0 - S + \rho \log \frac{S}{S_0} \tag{2.6}$$

where S_0 and I_0 are the number of susceptible and infectives at the initial time $t = t_0$ and $\rho = \frac{\gamma}{r}$. To analzethe behavior of the curves(2.6),we compute $I'(S) = -1 + \frac{\rho}{S}$. The quantity $-1 + \frac{\rho}{S}$ is negative for $S \succ \rho$,and positive for $S \prec \rho$ .Hence, *I(S)* is an increasing function of *S* for $S \prec \rho$ and a decreasing function of *S* for $S \succ \rho$ .Next, observe that $I(0) = -\infty$ and $I(S_0) = I_0 \succ 0$.Consequently, there exists a unique point S_∞ with $0 \prec S_\infty \prec S_0$, such that $I(S_\infty) = 0$, and $I(S) \succ 0$ for $S_\infty \prec S \leq S_0$. The point $(S_\infty, 0)$ is an equilibrium point of (2.2) since both $\frac{dS}{dt}$ and $\frac{dI}{dt}$ vanish when *I=0*.

Let us see what all this implies about the spread of the disease within the population. As *t* runs from t_0 to ∞, the point (*S(t) ,I(t)*)travels along the curve (2.4), and it moves along the curve in the direction of decreasing *S*, since *S(t)* decreases monotonically with time. Consequently, if S_0 is less than, then I(t) decreases

monotonically to zero, and *S(t)* decreases monotonically to S_∞.Thus, if a small group of infectives I_0 is inserted into a group of susceptible S_0, with $S_0 \prec \rho$,then the disease will die out rapidly. On the other hand, if S_0 is greater than, then *I(t)* increases as *S(t)* decreases to ρ, and it achieves a maximum value when $S = \rho$. It only starts decreasing when number of susceptible falls below the threshold value ρ. From these results we may draw the following conclusions.

Notes

Conclusions I. An epidemic will occur if the number of susceptible in a population exceeds the threshold value $\rho = \frac{\gamma}{r}$.

II. The spread of the disease does not stop for lack of a susceptible population; it stops only for lack of infectives. In particular, some individuals will escape the disease altogether.

a) Spread of Infection within a Family

What happens when an infectious diseases is introduced into a small family? What is the likelihood of spread within the family? We can answer these questions by carefully counting the possibilities.

Consider a fixed population and assume that three kinds of individuals in it are defined by a disease: susceptibles,infectives, and removals.Susceptibles can acquire the infection upon effective contact with an infective,infectives have the disease and are capable of transmitting it, and removals are those who have passed through the disease process but are no longer susceptible or infective.

$$S \rightarrow I \rightarrow R$$

indicating that susceptible might become infectives,and invectives might be removed, but there is no supply of new susceptible to the processes.

There are problems with taking such a simple view of a disease. For example, there are great variations in the level of susceptibility, infectiousness, and immunity among individuals in populations. Also, these definitions may depend on stratifying attributes such as age groups, genetic type or mixing groups, etc, there are many diseases whose transmission mechanisms are not known, nor how long are latency periods between becoming infective and the appearance of symptoms.

III. Calculation of the Severity of an Epidemic

Suppose that there are smooth functions $S(t), I(t), and\ R(t)$ and a small interval h such that $x_n = S(nh), y_n = I(nh),\ and\ z_n = R(nh)$. Since the sampling interval h is small, we must rescale a and b

Let $a = rh\ and\ b = 1 - h\sigma$. Then setting $t = nh$, we have

$$S(t+h) = e^{-rhI(t)}S(t)$$

$$I(t+h) = (1-h\sigma)I(t) + (1-e^{-rhI(t)})S(t)$$

$$R(t+h) = R(t) + (1-(1-h\sigma))I(t)$$

It follows that

$$S(t+h) - S(t) = (e^{-rhI(t)} - 1)S(t) \sim -rhI(t)S(t)$$

$$I(t+h) - I(t) = -h\sigma I(t) + (1-e^{-rhI(t)})S(t)$$

$$\sim -h\sigma I(t) + rhI(t)S(t)$$

$$R(t+h) - R(t) = h\sigma I(t)$$

Dividing these equations by h and passing to the limit $h = 0$ gives three differential equations for approximations to $S(t), I(t), and\ R(t)$; which we write as

$$\frac{ds}{dt} = -rIS$$

$$\frac{dI}{dt} = rIS - \sigma I$$

$$\frac{dR}{dt} = \sigma I$$

This system of equations is the continuous-time version of Kermark and Mc Kendrick's model.Incidentally,the calculation just completed gives a neat derivation of the law of mass action in chemistry in which the rate at which to chemical species ,say having concentrations S and , I interact is proportional to product SI. Thus, the Law of mass action follows from the binomial distribution of random interactions since the expected number of interactions occurring in a specified (short) time interval is

$$(1-q_n)S_n = (1-e^{-rhI_n})S_n \sim rhI_nS_n$$

Obviously, this law and the two model derived for epidemics depend on the assumption that the populations are thoroughly mixing as the process continues

We can solve the differential equations and so determine the severity of an epidemic.Taking the ratio of the first two equations gives

$$\frac{dS}{dI} = -\frac{rIS}{rIS - \sigma I} = \frac{-rS}{rS - \sigma}$$

Therefore

$$dI = (\frac{\sigma}{rS} - 1)\ dS$$

Integrating this equation gives

$$\mathrm{I} = (\frac{\sigma}{\mathrm{r}})\log \mathrm{S}\text{-}\mathrm{S} + C$$

where c is a constant of integration that is determined by the initial conditions:

$$C = I_0 - \log S_0 + S_0$$

Typical trajectories

In the infinitesimal sampling process, the threshold level of S* becomes

$$S^* = \frac{(1-b)}{(1-e^a)} \sim (\frac{\sigma}{r})$$

S^* is the value of S for which $\frac{DI}{Dt} = 0$ .We see that trajectories starting near but about this value describe epidemics that end at a comparable distance below this value.Trajectories that start well about S^* end up near $S = 0$ .However, in each case the final size of the susceptible population, S (infinity), is where the trajectory meets the $I = 0$ axis Therefore ,solving the equation

$$\mathrm{S}\text{-}(\frac{\sigma}{r})\log \mathrm{S} = \mathrm{C}$$

For its smaller of two roots gives the final size .this is not possible to do in a convenient form; however, it is easy to do using a computer. In this way ,we can estimate an epidemic's severity once we have estimated the infectiousness (a or r) and the removal rate (b or σ)

Recurrent diseases. Finally, there are diseases in which removals can eventually become susceptible again .This is the case for a variety of sexually transmitted diseases, for example gonorrhea. The flow of such a disease is depicted by the graph

$$S \to I \to S .$$

Without further discussion, we can write down a model of such a disease:

$$\frac{dS}{dt} = -rSI + \sigma I$$

$$\frac{dI}{dt} = rSI - \sigma I$$

Notes

Since $I + S$ is constant (its derivative is zero), we can reduce these equations to a single equation.

$$\frac{dS}{dt} = (\sigma - rS)(I_0 + S_0 - S) .$$

We see that if $S^* = \frac{\sigma}{r} \prec I_0 + S_0, then\, S \to S^*$ in this case! In the other case $S \to I_0 + S_0$, and the infection dies out of the population. When $S \to S^*$, the disease is epidemic. Stratification of the population, latency periods of the disease, hidden carriers, seasonal cycling of contact rates, and many other factors confound the study of epidemics, but the simple modals derived here provide useful and interesting methods.

IV. Spread of Gonorrhea in Anantapur District (a Case Study)

Gonorrhea ranks first today among reportable communicable diseases in the United States. There are more reported cases of gonorrhea every year than the combined totals for syphilis, measles, mumps, and infectious hepatitis. This painful and dangerous disease, which is caused by the gonococcus germ, is spread from person to person by sexual contact. A few days after the infection there is usually itching and burning of the genital area, particularly while urinating. About the same time a discharge develops which males will notice, but which females may not notice. Infected women may have no easily recognizable symptoms, even while the disease does substantial internal damage. Gonorrhea can only be cured by antibiotics (usually penicillin). However, treatment must be given early if the disease is to be stopped from doing serious damage to the body. If untreated, gonorrhea can result in blindness, sterility, arthritis, heart failure, and ultimately, death.

In this section we construct a mathematical model of the spread of gonorrhea. Our work is greatly simplified by the fact that the incubation period of gonorrhea is very short (3-7 days) compared to the often quite long period of active infectiousness. Thus, we will

assume in our model that an individual becomes infective immediately after contracting gonorrhea. In addition, gonorrhea does not confer even partial immunity to those individuals who have recovered from it. Immediately after recovery, an individual is again susceptible. Thus, we can split the sexually active and promiscuous portion of the population into two groups, susceptibles and infectives. Let $c_1(t)$ be the total number of promiscuous males, $c_2(t)$ be the total number of promiscuous females, $x(t)$ the total number of infective males, and $y(t)$ the total number of infective females, at time t. Then, the total numbers of susceptible males and susceptible females are $c_1(t)-x(t)$ and $c_2(t)-y(t)$ respectively. The spread of gonorrhea is presumed to be governed by the following rules:

I. Males infectives are cured at a rate a_1 proportional to their total number, and female infectives are cured at a rate a_2 proportional to their total number. The constant a_1 is larger than a_2 since infective males quickly develop painful symptoms and therefore seek prompt medical attention. Female infectives, on the other hand, are usually asymptomatic, and therefore are infectious for much longer periods.

II. New infectives are added to the male population at a rate b_1 proportional to the total number of male susceptibles and female infectives. Similarly, new infectives are added to the female population at a rate b_2 proportional to the total number of female susceptibles and male infectives.

III. The total numbers of promiscuous males and promiscuous females remain at constant levels c_1 and c_2, respectively.

It follows immediately from rules I-III that

$$\frac{dx}{dt} = -a_1 x + b_1(c_1 - x)y$$
$$\frac{dy}{dt} = -a_2 y + b_2(c_2 - y)x \qquad (4.1)$$

If $x(t_0)$ and $y(t_0)$ are positive, then $x(t)$ and $y(t)$ are positive for all $t \geq t_0$

If $x(t_0)$ is less than c_1 and $y(t_0)$ is less than c_2 then $x(t)$ is less than c_1 *and* $y(t)$ is less than c_2 for all $t \geq t_0$

We can show that equation (4.1)

(a) Suppose that $a_1 a_2$ *is less than* $b_1 b_2 c_1 c_2$ .Then, every solution $x(t)$, $y(t)$ of (4.1) with $0 \prec x(t_0) \prec c_1$ *and* $0 \prec y(t_0) \prec c_2$, approaches the equilibrium solution.

$$x = \frac{b_1 b_2 c_1 c_2 - a_1 a_2}{a_1 b_2 + b_1 b_2 c_2}, y = \frac{b_1 b_2 c_1 c_2 - a_1 a_2}{a_2 b_1 + b_1 b_2 c_1}$$

as t approaches infinity. In other words, the total number of infective males and infective females will ultimately level off.

Proof: The result can be established by splitting the rectangle $0 \prec x \prec c_1$ *and* $0 \prec y \prec c_2$ into regions in which both $\frac{dx}{dt}$ *and* $\frac{dy}{dt}$ have fixed signs. This is a accomplished in the following manner. Setting $\frac{dx}{dt}$=*0* in equation (4.1), and solving for *y* as a function of *x* gives.

$$-a_1 x + b_1 (c_1 - x) y = 0$$

$$b_1 y(c_1 - x) = a_1 x$$

$$y = \frac{a_1 x}{b_1 (c_1 - x)} = \phi_1 x$$

Similarly, setting $\frac{dy}{dt} = 0$ in (4.1)

$$-a_2 y + b_2 x (c_1 - y) = 0$$

$$b_2 x (c_2 - y) = a_2 y$$

$$x = \frac{a_2 y}{b_2 (c_2 - y)}$$

$$x b_2 (c_2 - y) = a_2 y$$

$$x b_2 c_2 - x b_2 y = a_2 y$$

$$x b_2 c_2 = a_2 y + x b_2 y$$

$$x b_2 c_2 = y (a_2 + x b_2)$$

$$\frac{x b_2 c_2}{a_2 + x b_2} = y$$

$$y = \frac{x b_2 c_2}{a_2 + x b_2} = \phi_2 x$$

Notes

Observe that $\phi_1 x$ and $\phi_2 x$ are monotonic increasing functions of *x;* $\phi_1 x$ approaches infinity as *x* approaches c_1 and $\phi_2 x$ approaches c_2 as *x* approaches infinity. Second, observe that the curves $y = \phi_1 x$, $y = \phi_2 x$ intersect at $(0,0)$ and at (x_0, y_0) where

$$x_0 = \frac{b_1 b_2 c_1 c_2 - a_1 a_2}{a_1 b_2 + b_1 b_2 c_2}, y_0 = \frac{b_1 b_2 c_1 c_2 - a_1 a_2}{a_2 b_1 + b_1 b_2 c_1}$$

Third, observe that $\phi_2 x$ is increasing faster than $\phi_1 x$ at $x = 0$, since

$$\phi_2'(0) = \frac{b_2 c_2}{a_2} \succ \frac{a_1}{b_1 c_1}$$

Hence, $\phi_2 x$ lies above for $0 \prec x \prec x_0$ and lies below $\phi_1 x$ for $x_0 \prec x \prec c_1$. The point (x_0, y_0) is an equilibrium point of (4.1) since both $\frac{dx}{dt} and \frac{dy}{dt}$ are zero when $x = x_0$ and $y = y_0$.

Finally, observe that $\frac{dx}{dt}$ is positive at any point (x, y) above the curve $y = \phi_1 x$,and negative at any point (x, y) below this curve. Similarly, $\frac{dy}{dt}$ is positive at any point (x, y) below curve $y = \phi_2 x$, and negative point (x, y) above this curve. Thus, the curves $y = \phi_1 x$,and $y = \phi_2 x$ split the rectangle $0 \prec x \prec c_1 \; and \; 0 \prec y \prec c_2$ into four regions in which $\frac{dx}{dt} and \frac{dy}{dt}$ haved fixed sings.

It can be established that any solution *x (t), y (t)* of (4.1) which starts in region I at time $t = t_0$, will remain in this region for all future time $t \geq t_0$ that and approach the equilibrium solution $x = x_0, y = y_0$ as *t* approaches infinity.

Any solution *x (t), y (t)* of (4.1) which starts in region II at time $t = t_0$, will remain in this region II for all future time , must approach the equilibrium solution $x = x_0, y = y_0$ as *t* approaches infinity.

Any solution *x (t), y (t)* of (4.1) which starts in region IV at time $t = t_0$, will remain in this region IV for all future time , must approach the equilibrium solution $x = x_0, y = y_0$ as *t* approaches infinity.

We make use of the above mentioned deterministic model to study of severity of Gonorrhea diseased in Anantapur district during the period of 1995-2003 based on the data collection from the Head Quarters of Hospital Anantapur during this period.

Year wise male population in Anantapur district and case study of gonorrheal disease form the recorded data of Government Head Quarters Hospital, Anantapur, Andhra Pradesh

Table 1

Years	Total Male population	Total number of promiscuous Males	Total number of Infective Males	Total number of Males Cured
1995	1686038	16860.38	7250	6975
1996	1698463	16984.63	5178	4846
1997	1723455	17234.55	6147	5872
1998	1748654	17486.54	3065	2969
1999	1798563	17985.63	6598	5985
2000	1810943	18109.43	3657	3465
2001	1859588	18595.88	2713	2285
2002	1910464	19104.64	2577	2299
2003	1945084	19450.84	1954	1835

Year wise Female population in Anantapur district and case study of gonorrheal disease form the recorded data of Government Head Quarters Hospital, Anantapur, Andhra Pradesh

Table 2

Years	Total Female population	Total number of promiscuous Females	Total number of Infective Females	Total number of Females Cured
1995	1516549	15165.49	3549	3020
1996	1576910	15769.10	1988	1866
1997	1624704	16247.04	1769	1665
1998	1672291	16722.91	1935	1798
1999	1695168	16951.68	1701	1595
2000	1755574	17555.74	1278	1152
2001	1780890	17808.90	1356	1265
2002	1801626	18016.26	1093	943
2003	1839792	18397.92	899	844

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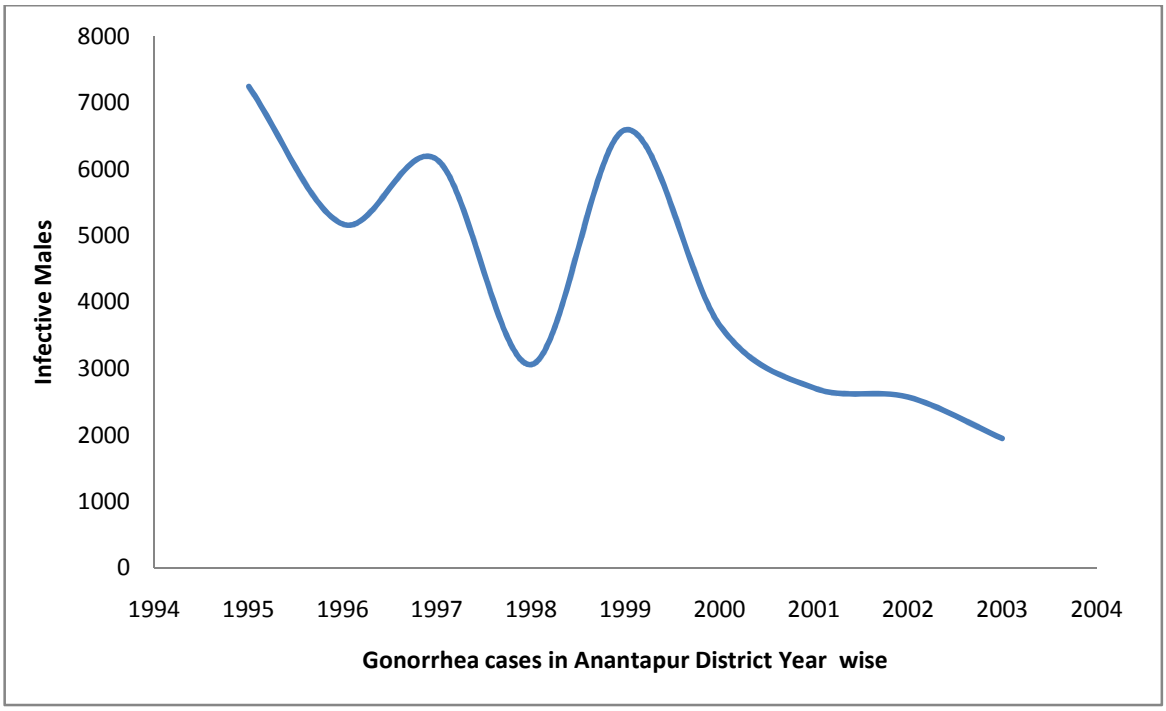

Figure 1 : **Profile of Infective Males with Gonorrheal Disease during the period of 1995-2003**

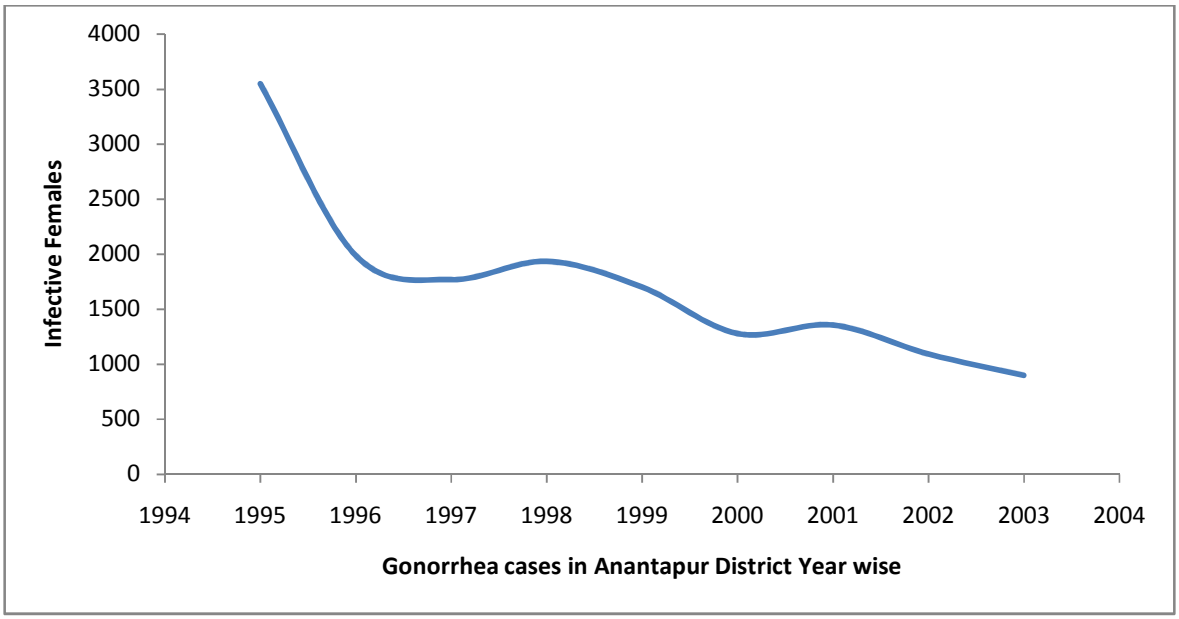

Figure 2 : **Profile of Infective Females with Gonorrheal Disease during the period of 1995-2003**

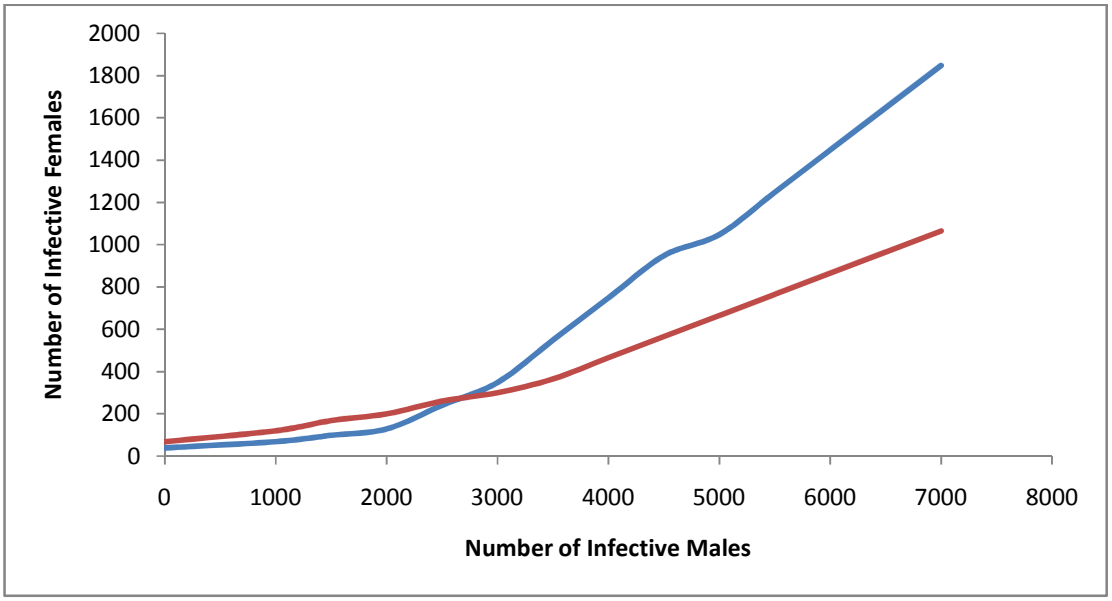

Figure 3 : **Profile of Number of Infective Males Vs Number of Infective Females during the period of 1995-2003**

V. Discussion

The Study of epidemic is quite interesting and important. When a small group of people having infected with an epidemic disease is inserted into a large population which is capable of catching the disease, the question arises what happens as time evolves. Will the disease die out rapidly or its spreads? How many people will catches is disease? To answer these questions, we have chosen a mathematical modeling consisting of a system of differential equations which govern the spread of infected disease within the population and analyse the behavior of its solution. This approach will lead to the famous threshold theorem of Epidemiology which states that an epidemic will occur only if the number of people who are susceptible to the disease exceeds a certain threshold value. In this paper, we discussed the spread of Gonorrheal disease among males and females in Anantapur District during the period 1995-2003, based on the recorded data available in Government Head Quarters Hospital Anantapur. As already stated, we assume in our model that an individual become infective immediately after contacting the Gonorrhea. The proportional rate a_1, a_2 and b_1 *and* b_2 are quite difficult to evaluate. However, we have made the crude estimate of these proportional constants based on available data. It is interesting note that condition $a_1 a_2 \prec b_1 b_2 c_1 c_2$ is satisfied by the said constants. This

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condition is equivalent to $1 \prec \left(\frac{b_1c_1}{a_2}\right)\left(\frac{b_2c_2}{a_1}\right)$. The expression $\left(\frac{b_1c_1}{a_2}\right)$ can be interpreted as the average number of males that one female infective contact during her infectious period, if every male is susceptible. Similarly the expression $\left(\frac{b_2c_2}{a_1}\right)$ can be interpreted as the average number of females that one male infective contact during his infectious period, if every female is susceptible. These quantity $\left(\frac{b_1c_1}{a_2}\right)$ and $\left(\frac{b_2c_2}{a_1}\right)$ are called the maximal female and male contacts rates. In view of the fact that this product of maximal male and female contact rates is greater than one, we may conclude that the solution of the mathematical model approaches the equilibrium solution and the Gonorrheal disease will approach a non zero steady state in course of time. This equilibrium solution also implies that the total number of infective males and infective females will ultimately level off, and from Fig (3) the point of the equilibrium approximately gives x_0 (Infective males) =260, y_0 (Infective females) =266. We may conclude that this Epidemic disease does not die out but ultimately approach a steady state with reference to its severity among the population of Anantapur District.

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