# MATHEMATICAL MODELLING AND PROPAGATION OF INFECTIOUS DISEASES IN CONFINED AREAS

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**Abstract:**

A mathematical model for the propagation of infectious diseases has been discussed in this paper. The model that has been examined is basically SIR model. The governing equations have been solved to the best possible solution. The influence of critical parameters over the system has been discussed with the graphical illustrations. It is observed that as the transmission (β) increases the sensitive population and, with the duration increasing, the vulnerable population diminishes and stays constant and is time-independent. Further, it is observed that as the rate of infection increases the susceptible population tens to zero. In addition to the above as R rate of recovered individual increases the susceptible population (S) decreases. The phenomena is an agreement with the real life situation. In addition to the above as R rate of recovered individual increases the susceptible population (S) decreases. The phenomena is an agreement with the real life situation**.**

## I. INTRODUCTION

An on-going hazard to humankind is infectious illnesses. A sickness can harm every human on the world. Infectious illnesses have developed and reoccurred into a major international concern. The development of preventive measures can assist an appropriate knowledge of the transmission processes of illnesses resulting from current and novel pathogins. Transmission prevention measures, including vaccinations and medicines, must be created at a rate equivalent to microorganisms. Another difficulty is to implement and properly utilize these advanced instruments for microorganisms. Different microorganisms or pathogenes produce an infectious illness. Usually the majority are micro-organisms. Various kinds of viruses and bacteria are the most prevalent pathogen. Pathogens are also known to be Fungi and Protozoa and are responsible for a number of conditions. These infections are called 'infectious' diseases, as these pathogens may readily be passed from an infected individual to someone else. Influenza or flu that is caused by certain viruses might be the common and recognized example of such disorders. The fact that millions have been infected by the diseases of HIV,

mumps and measles, rubella, little pox, malaria. Many of these illnesses continue to prevail at local or global level and are endangering public health.

## II. MODE OF TRANSMISSION:

Infectious illnesses may spread in numerous ways and diseases via diverse mechanisms of transmission cause infections. Some infections may be caused by direct touch, while others through indirect encounters. It is also possible to transmit through carriers or vectors. Malaria, Filaria, Western Nile, Dengue and Chikungunya, for instance, spread via mosquitos. The illness in the air transmitted by sneezing, tobacco, and laughing from an infected person to an uninfected individual. An infected individual may dislodge the germs on the dust particles or any other media.

## III. Mathematical Model (S I R )

An imagined microworld comprised of things who operate according to specific rules is a mathematical model. The language we use in mathematical matters is concisely and unambiguously to formulate these laws of behaviour, which compells and helps us to make our assumptions apparent.

Foy and Cooney [5] They were the first to attempt, by mathematical models, to examine the longitudinal study of type A and B infection among the students of Seattle. Subsequently, Anderson and R.M. May [6] extended the mathematical theory to studyPopulation Biology of Infectious Diseases. Thereafter, Hale and S.V. Lunel [7] introduced the concept of functional differential equations to study the concept of infected population dynamics.The mathematical model of Control of Communicable Diseases was intensively studied by Benenson [8] . Moreover, Daniel Bernoulli has also been credited with inventing early mathematical models since the 18th century, when he developed a model for smallpox to assess the efficacy of a healthy population with smallpox. Quick diagnostic tests, available clinical data and electronic monitoring can enable mathematical models to be applied to scientific hypotheses and to develop practical methods [9].

A model can determine if the related illness spreads across the population or died by calculating transmission, reproductive number and other factors and characteristics. It may also assess the impact of a control measure and give guidance for public health in subsequent attempts to eliminate illness. Since the mid 20th century, however, mathematical models have grown, after the publication of the work on epidemic models by Kermack and McKendrick [4] in 1927 that contained threshold conclusions that determined whether or not an epidemic could develop [10]. In the previous two decades, the number of modelling practises in the biological sciences has increased enormously. by Cohen [11].

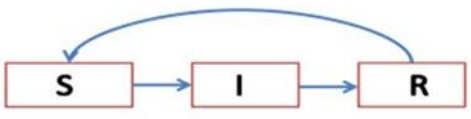
The illness has been shown to be significantly related to the overwhelming family and people in unclean settings. The death rate is closely associated with the number of persons in a house in the particular population region. The connection between the housing and workplace congestion and death from TB is considered to be substantial. The illness transmission is usually connected with lengthy intimate contact with the sick individual, and this is mathematically determined. by Guo and M. Li, [15].

The transmission method and the number of infected populations are described in an epidemic pattern. The number or fraction of the population remained uninfected can be

determined by such a model. Two common schematics for transmission of illness in Figure above. (a) infection with SIRS; The notion of population dividing is extensively employed in epidemic simulations.

These divisions are generally represented in mathematical convenience by letters S, E, I and R representing the population susceptible, exposed, infected and recovered, respectively. Persons sensitive to infection are classified as susceptible and belong to the compartment of S (susceptible). E (exposed) compartment includes an individual who already is sick, but does not display any symptoms or who is unable to transmit others. After an infected person infects others, they become like an infectious person and are part of the compartment. Finally, if someone is cured of the virus, they belong to the R (recovered) body. Either a recovered person will be there if he/she is permanently recovered or may again be vulnerable and go back to S.The intricacies of this unobserved factor underpin this study.

Population is supposed to be homogenously combined and people become infected or are constantly treated during these modelling sessions. Some schematics are displayed in the following Figure. The following system of normal differential equations might provide a rudimentary illustration of the SIR model.



(a)

An important process used by modellers for testing the robustness of mathematical model predictions is the comparison of multiple models[1–3]. Kermack and McKendrik's Simple SIR model [4].

In this model the populations are split into vulnerable, infecting and recovering individuals and their corresponding population fractions are shown in the functions S(t), I(t) and R(t) (measured, for example, in days). The changes in these quantities are represented using the differential equations

$$\frac{dS}{dt} = -\beta S I \quad (1)$$

$$\frac{dI}{dt} = \beta S I - \gamma I \quad (2)$$

$$\frac{dR}{dt} = \gamma I \quad (3)$$

Such that

$$S + I + R = k\ (\text{constant}) \text{ and } \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$$

Then

$$\frac{dS}{dt} = -\beta S I - \gamma S + \gamma \quad (4)$$

Where β represents the average number of individuals that are infected and ϒ is rate of recovery.

This basic model illustrates how sub-populations of sensitive, infectious and reclaimed classes develop, without taking into account the population demographics of the host population. The image above is altered, taking into account several elements that capture the major characteristics of the issues, however this change naturally increases the models complexity and makes analysis difficult and often impossible.

A crucial conclusion for the model of an epidemic is generally based on the basic reproductive number, which is commonly referred to as $R_0$, defined as the total number of secondary infections in a totally susceptible population of the infected person during the whole infectious period [17,18]. The basic reproductive number is a key driver of population-level disease infection patterns. If and only if that number is more than one, an epidemic breaks apart. This threshold characteristic gives vital information on the possible spread of the disease and its effects [18]. Recently, Srinivasarao veerla *et al* [19] obtained a simple mathematical model which explains the influence of several participating parameters on the new infections case. Such an example of SIS models would be bacterial infections. On the other hand, a SIR-like model would be suitable if the recovery were permanent and the recovered people were not vulnerable to this pathogen, as observed with viral infections.

## IV. SOLUTION METHODOLOGY

Since S+I+R=k (constant) then

$$\frac{dS}{dt}+\frac{dI}{dt}+\frac{dR}{dt}=0$$

Subject to initial conditions $I(0)=I_0, S(0)=S_0$ where $\beta \succ 0, I_0 \succ 0$ and $S_0 \succ 0$ Where the derivatives $\frac{dS}{dt}, \frac{dI}{dt} and \frac{dR}{dt}$ measure the rates of change of the quantities $S(t)$,$I(t)$, and $R(t)$. The transmission parameter $\beta$ is the average number of individuals that one infected individual will infect per time unit, assuming that all contacts that this individual makes are with susceptible individuals. Thus, a more highly infectious disease has a higher $\beta$. The number $\gamma$ is the rate of recovery, so that $\frac{1}{\gamma}$ is the average time period during which an infected individual remains infectious .The product $\beta$ $S(t)I(t)$ is the total infection rate, the fraction of the population that will be infected per unit time at time $t$. T o understand this, note that, if a fraction $I(t)$ of the population is currently infected, then they would infect a fraction $\beta$ $I(t)$ of the population per unit time if all of their contacts were with susceptible individuals, but as only a fraction $S(t)$ of the population is currently susceptible, they will only infect $\beta$ $I(t)S(t)$ per unit time.

The ratio $\frac{\beta}{\gamma}$ is also known as the basic reproductive number$R_0$, Which is a major index for the measurement of infections transmission. R0 is defined as the average number of persons infected with an infected person in the whole susceptible population throughout the infection period.

This simple model, which is the basis of numerous elaborations, offers some quite stunning forecasts. By inputting the differential equations mentioned above and selecting certain values for each numeric solution of differential

equations. $\beta$ and $\gamma$ Together with the initial S(0), I(0) and R(0) values, a matching epidemic curve may be generated for the population which will be infected by each day of the epidemic, a forecast for the group of the population. In addition, we may make broad conclusions about the model's solutions with the analytical instruments. The answers have been obtained accordingly.

Adding equations (2) and (4) we get

$$\frac{dS}{dt}+\frac{dI}{dt}=-\gamma S-\gamma I+\gamma$$

$$\frac{d(S+I)}{dt}=-\gamma(S+I)+\gamma$$

$$\frac{d(S+I)}{dt}+\gamma(S+I)=\gamma \qquad (5)$$

Which is a linear equation in S+I with respect to't', then the general solution is

$$(S+I)e^{\gamma t}=\int\gamma e^{\gamma t}dt+c$$

$$(S+I)=1+ce^{-\gamma t}$$

$$S=1+ce^{-\gamma t}-I \qquad (6)$$

Where c is the integration constant. Now using equation (6) in (2), we get

$$\frac{dI}{dt}=\beta I(1+ce^{-\gamma t}-I)-\gamma I$$

$$\frac{dI}{dt}=(\beta+c\beta e^{-\gamma t}-\gamma)I-\beta I^2$$

$y=\frac{1}{I}$, so that $\frac{dy}{dt}=\frac{-1}{I^2}\frac{dI}{dt}$ in equation (7) we get

$$\frac{dy}{dt}+(\beta+c\beta e^{-\gamma t}-\gamma)y=\beta \qquad (8)$$

Which is a linear equation in 'y' with respect to 't' then the general solution is

$$ye^{(\beta-\gamma)t-\frac{\beta c}{\gamma}e^{-\gamma t}}=\int\beta e^{(\beta-\gamma)t-\frac{\beta c}{\gamma}e^{-\gamma t}}dt+d \qquad (9)$$

Where 'd' is integration constant.

Considering the expansion of $e^{-\gamma t}$ and neglecting square and higher terms we get $e^{-\gamma t}=1-\gamma t$ .substituting back in (9), one gets

$$ye^{(\beta-\gamma)t-\frac{\beta c}{\gamma}(1-\gamma t)}=\int\beta e^{(\beta-\gamma)t-\frac{\beta c}{\gamma}(1-\gamma t)}dt+d$$

$$ye^{(\beta-\gamma+\beta c)t}e^{-\frac{\beta c}{\gamma}}=\int\beta e^{(\beta-\gamma+\beta c)t}e^{-\frac{\beta c}{\gamma}}dt+d$$

$$ye^{(\beta-\gamma+\beta c)t}e^{-\frac{\beta c}{\gamma}}=\int\beta e^{(\beta-\gamma+\beta c)t}e^{-\frac{\beta c}{\gamma}}dt+d$$

$$ye^{(\beta-\gamma+\beta c)t}=\beta\frac{e^{(\beta-\gamma+\beta c)t}}{\beta-\gamma+\beta c}+\frac{d}{e^{-\frac{\beta c}{\gamma}}}$$

$$y=\frac{\beta}{\beta-\gamma+\beta c}+\frac{d}{e^{-\frac{\beta c}{\gamma}}e^{(\beta-\gamma+\beta c)t}}$$

$$y=\frac{\beta}{\beta-\gamma+\beta c}+de^{\frac{\beta c}{\gamma}}e^{-(\beta-\gamma+\beta c)t} \qquad (10)$$

Substituting $y=\frac{1}{I}$ and $\beta-\gamma+\beta c=k$ in equation (10), we get

$$\frac{1}{I}=\frac{\beta}{k}+de^{\frac{\beta c}{\gamma}}e^{-kt}$$

$$\frac{1}{I} = \frac{\beta + d\,k\,e^{\frac{\beta c}{\gamma}} e^{-kt}}{k}$$

$$I = \frac{k}{\beta + d\,k\,e^{\frac{\beta c}{\gamma}} e^{-kt}} \quad (11)$$

Applying initial conditions $I(0) = I_0$ and $S(0) = S_0$ in equation (6) and in (11), we get

$$S_0 = 1 + c - I_0$$

$$c = S_0 + I_0 - 1$$

and $I_0 = \dfrac{k}{\beta + d\,k\,e^{\frac{\beta(S_0 + I_0 - 1)}{\gamma}}}$

Consequently $d = \dfrac{k - I_0\beta}{k\,I_0\,e^{\frac{\beta(S_0 + I_0 - 1)}{\gamma}}}$

Substituting c, d values in equation (11), we get

$$I(t) = \frac{k}{\beta + k\left(\dfrac{k - I_0\beta}{k I_0 e^{\frac{\beta(S_0 + I_0 - 1)}{\gamma}}}\right) e^{\frac{\beta(S_0 + I_0 - 1)}{\gamma} - kt}}$$

(12)

Which represents infective population at any time't'.

From equations (6) and (12) with given initial conditions, we get susceptible population in terms of't' as

$$S(t) = 1 + (S_0 + I_0 - 1)(1 - \gamma t) - \frac{k}{\beta + k\left(\dfrac{k - I_0\beta}{k I_0 e^{\frac{\beta(S_0 + I_0 - 1)}{\gamma}}}\right) e^{\frac{\beta(S_0 + I_0 - 1)}{\gamma} - kt}} \quad (12)$$

Consequ ntly, from equations (12) and (13) we

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get recovered population expressed in terms of t' as

$R(t) = k - I(t) - S(t)$ implies that

$$R(t) = k - 1 - (S_0 + I_0 - 1)(1 - \mu t) \quad (14)$$

Solving the differential equations

$\dfrac{dS}{dt} = -\beta S I$ and $\dfrac{dR}{dt} = \gamma I$, we have

$$\frac{\frac{dS}{dt}}{\frac{dR}{dt}} = \frac{-\beta S I}{\gamma I}$$

$$\frac{dS}{dR} = \frac{-\beta S}{\gamma} \quad (15)$$

By using variable separable method from (15),we have

$$\log(S) = \frac{-\beta}{\gamma} R + \log(c)$$

$$\log\left(\frac{S}{c}\right) = \frac{-\beta}{\gamma} R$$

$$S = c e^{\frac{-\beta R}{\gamma}} \quad (16)$$

When R=0, S=1, from equation (16), we get c=1

Therefore $S = e^{\frac{-\beta R}{\gamma}}$ (17)

V. CONCLUSIONS:

The epidemic curve corresponding to a model under investigation may always be created by selecting acceptable values for β and ϒ , together with beginning conditions S(0), I(0) and R0.

The analytical techniques enable us to identify some unified, generalized models and research solutions which are essentially characterized as follows:

The epidemic thresh hold: Where inequality s(0), R0,>1, there is a declining tendency of the no of infected people which will indicate that after some time the epidemic will die.

If s(0)* $R_0$>1, The first no of the infected individual stays in the system will occur as an epidemic regardless of the amount.

The epidemic's magnitude will not depend on the first patients infected but rather depend on the first sensitive cases, such as s0 and R0. An implicit notion which happens on the last size of the epidemic is always narrowly smaller for the susceptible S0 than the starting population.

The conclusions in so far can be applied to the real life situation where in some critical implications occurs. more importantly , The epidemic threshold indicates that if we immunise, the population portion that spreads the illness might be controlled in some degree prior to the emergence of the sickness. This conclusion emphasises the idea of her immunity, which may be regulated more widely through avoidance of illness transmission. The other ways to achieve S(0)< ϒ * I*β which eliminates an epidemic through fractional quantities.

This model does not enable the fact that the individual cannot differ from individuals in the direction of transmission from illnesses. This model does not allow the individual to differ.

All predictions and conclusions made out of this model proves to be worthy of the contexts and a slight change in any of the parameters might alter the stability of the solution.this means that the predictions could prove to be unrealistic .in all such cases the problem needs to be reexamine with all moderate considerations in the model proposed.

Figure 1 depicts the influence of .ϒ for different values of time over the recovery population. It is seen that as ϒ increases the recovery population decreases.Further the relation is forecast to be linear.In addition to the above as time increasing for constant values of ϒ,the population recovery decreases,subsequently

Figure 2 shows that the influence of β for different values of time over the infected population.

In figure 2 it is observed that for constant β as time increases,the infected population increases and thereafter remains a constant.

Figure 3 shows that as β rises the sensitive population, and as the period increases, the vulnerable population declines and remains constant and time-independent.

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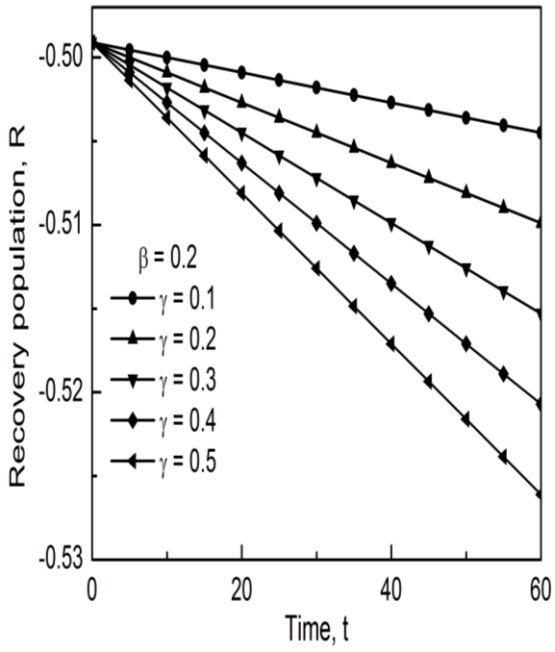


Fig 1: Influence of time on recovery population ®

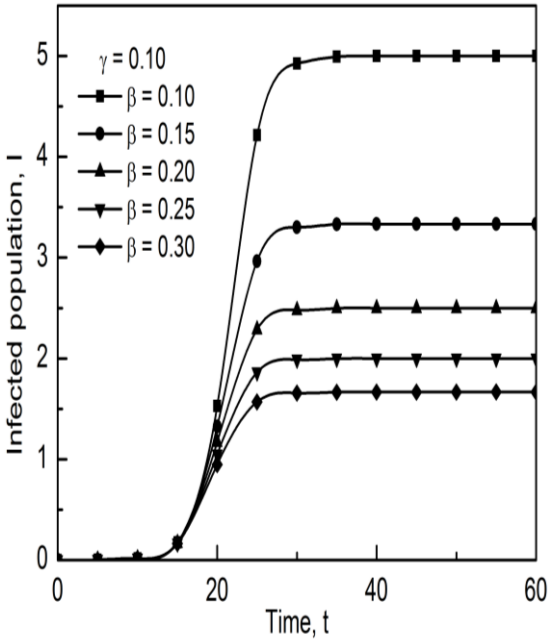


Fig 3: Influence of time over susceptible population

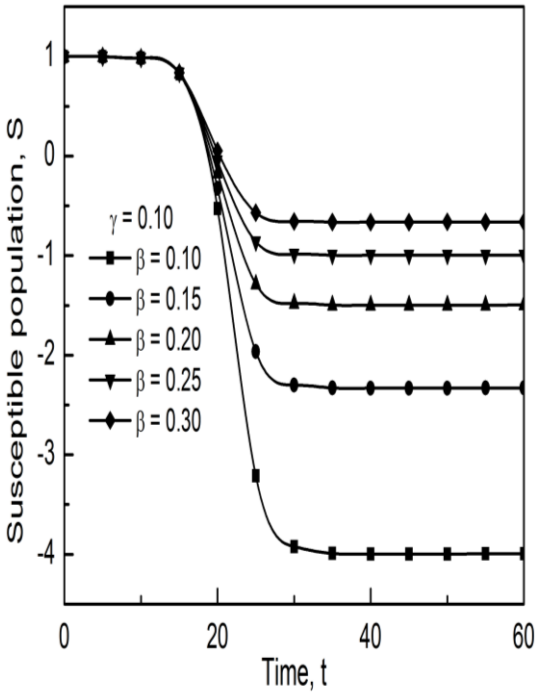


Fig 2: Effect of time over infected population

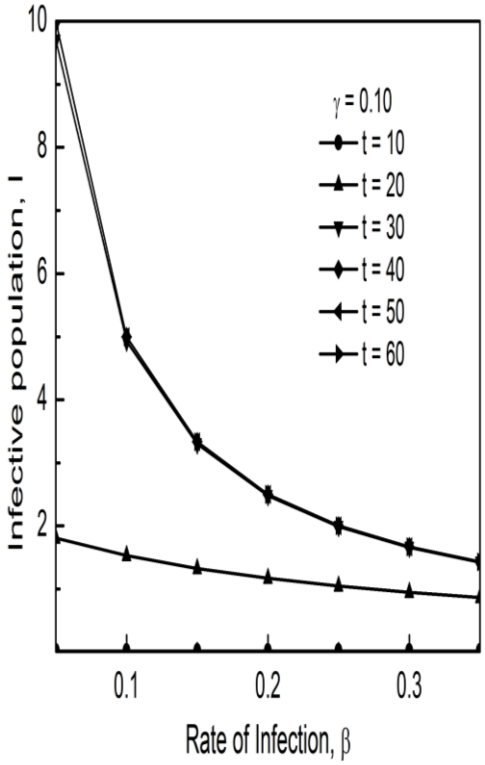


Fig 4: Rate of infection over infective population

Figure 5 depicts the relation between the rate of infection β and susceptible population (S) for

different recovered individuals when the recovery rates ϒ held constant. It is observed that as the rate of infection increases the susceptible population tens to zero. In addition to the above as R rate of recovered individual increases the susceptible population (S) decreases. The occurrences are in accordance with the situation in actual life.

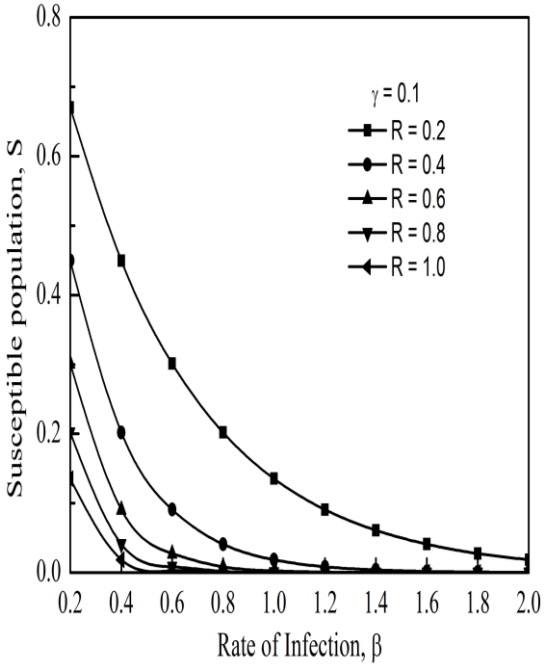


Fig 5: Influence of rate of infection Vs Susceptible population

Figure (5) illustrates the phenomena for the rate of recovery (ϒ) verses susceptible population S, from the illustration it is observed that as the rate of recovery increases the susceptible population also increases. However such an increase is not much significant as was seeing in the initial stages of the situation. further as R increases the susceptible population decreases at any point of instant for the rate of recovery.

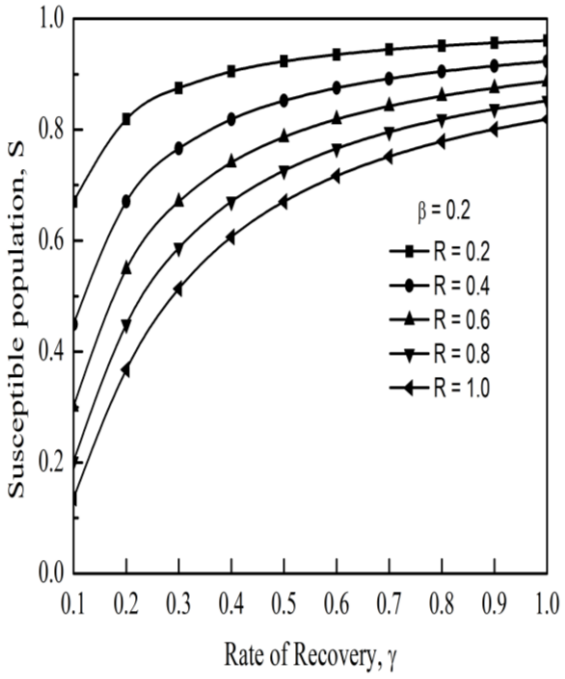


Fig 6: Influence of rate of recovery w.r.t susceptible population

Figure (6) demonstrates the impact of the recovery rate (ϒ) on the susceptible population (S).While all other parameters remaining constant. It is observed that, as (ϒ) increases the susceptible population in the system increases. In addition to the above it is noticed that as R increases the susceptible population (S) decreases. Such decrease is a parabolic in the initial stages and is

found to be linear thereafter.

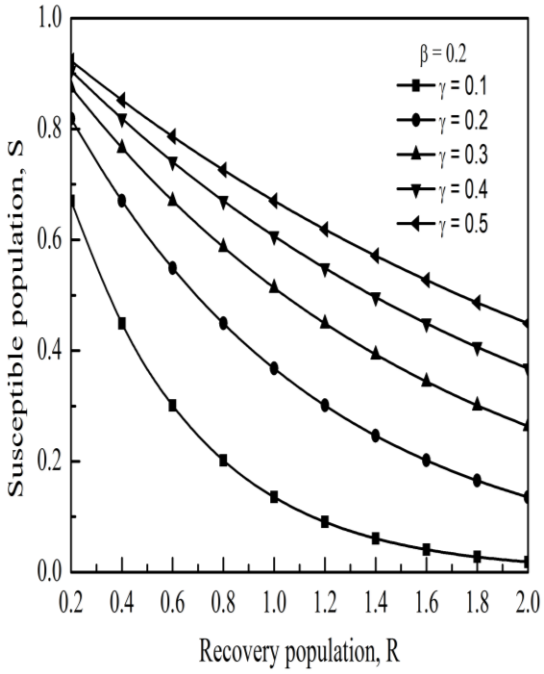


Figure 7:- Variation of susceptible population with respect to the recovery population

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