Modifications to Susceptible, Infective and Recovery Model

Akeem Olanrewaju Adeoye, and Anthonia Idowu Alawaye

Abstract**—**The SIR model first proposed by Kermack and.McKendrick (1927) was modified in this research and the modified model was used to obtain the changes in number of susceptibles, infectives and removals with changes in the rates infective become removal, rates infective become removals and rates infective are removed. Rates chosen are: infective rates of 0.2, 0.4, 0.6, and 0.8, recovery rate were 0.2 ,0.4 and 0.6 for N=20. The results show that as recovery rate increases, the number of infectives reduces. In the original and modified model, infective rate is the dominant factor affecting the number of susceptible. The results show that higher rates of infection result in rapid reduction in number of susceptible. The implication of this is that when the rate of infection of a disease is high, immunization or vaccination must commence early. The main factor affecting the number of infective at the beginning of an epidemic is the rate of infection. As the number of infected however increases, it is reduced by the rate of recovery. At this stage, the higher the recovery rate the lower the number infected. There is no recovery at the initial stage until there is a number that is infected. Thereafter, the number of recovery becomes the dominant factor. At these rates, a minimum of 85% of the susceptible population needs to be immunized to control transmission of the disease in the both original and modified.

Keywords---Susceptible, infective, recovery, rates disease.

I. Introduction

THE outbreak and spread of disease has been studied for many years. The ability to make predictions about diseases could enable scientists to evaluate inoculation/vaccination or isolation plans and may have a significant effect on the mortality rate of a particular epidemic. The modeling of infectious diseases is a tool which has been used to study the mechanisms by which diseases spread, to predict the future course of an outbreak and to evaluate strategies to control an epidemic [6]. The distribution of an infectious disease over an animal population and its evolution through time are the results of the dynamic interactions of the host and pathogen systems. To design successful disease-control strategies, it is important to understand what the most important processes are, and how they combine to characterize the dynamics of the disease spread[1] . Modeling of infectious diseases has a long history in mathematical biology, starting with the works of Sir Ronald Ross at the beginning of the 20th century and William Ogilvy, Kermack and Anderson Gray McKendrick in the 1920's and 1930's. In recent years, it has even become part of epidemiology policy decision making in several countries, including the United Kingdom, Canada, and the United State. Modeling studies of diseases such as HIV/AIDS, foot and mouth disease and measles have had an impact on public health policy in these countries. Apart from these hot spots, a tremendous variety of mathematical models have been developed, analyzed, and applied to a tremendous variety of infectious diseases, such as malaria, rabies and Lyme disease [2],[3],[4],[5]. Majority of epidemiological models focus on human diseases, such as passive immunity, vaccination, gradual loss of vaccine and disease-acquired immunity, stages of infection, vertical transmission, disease vectors, age structure, social and sexual mixing groups, and spatial spread [3]. Analogous models have also been developed for animal diseases. An issue of increasing importance is global climate changes and other anthropogenic stressors which render natural populations of animals increasingly susceptible to diseases contracted by spillover from domestic animals, as well as render humans increasingly susceptible to diseases originally restricted to wildlife. Detailed models are often impossible to solve analytically and hence, their usefulness for theoretical purposes is limited, although their practical value may be high. In this study we deal with simple models in order to establish broad principles of mathematical epidemiology. Furthermore, these simple models have an additional value as they are the building blocks of models that include more detailed structure. As a matter of fact, we will never be able to predict the precise course of a disease, or which individuals will be infected. The best that we can hope for are models that provide confidence intervals on the disease behavior and determine the risk of infection for various groups of hosts [3]

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II. Methodology

The SIR Model

The SIR Model is used in epidemiology to compute the number that are susceptible, infected or recovered in a population. This model is use under the following assumptions.

1) The population is fixed.

2) The only way a person can leave the susceptible group is to become infected. The only way a person can leave the infected group is to recover from the disease. Once a person has recovered, the person received immunity.
3) Age, sex, social status, and race do not affect the probability of being infected.
4) There is no inherited immunity.
5) The member of the population mix homogeneously (have the same Interactions with one another to the same degree).
In 1927, W. O. Kermack and A. G. McKendrick created a model in which they considered a fixed population with only three compartments, susceptible: S(t), infected, I(t), and recovered, R(t).The compartments used for this model consist of three classes:
• S(t) is used to represent the number of individuals not yet infected with the disease at time t, or those susceptible to the disease
• I(t) denotes the number of individuals who have been infected with the disease and are capable of spreading the disease to those in the susceptible category
• R(t) is the compartment used for those individuals who have been infected and then recovered from the disease. Those in this category are not able to be infected again or to transmit the infection to others.Using a fixed population, N = S(t) + I(t) + R(t), Kermack and McKendrick derived the following equations:

$$S_{(t+\Delta_t)} = S_{(t)} - \beta\Delta_t I_t S_{(t)}$$

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

$$I_{(t+\Delta_t)} = I_t + \beta\Delta_t S_t I_t - \gamma I\Delta_t$$

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

$$R_{(t+\Delta t)} = R_{(t)} + I\gamma\Delta_t$$

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

With the constant population S + I + R = N.The model above is the existing model and the modify model is giving below

$$S_{(t+\Delta_t)} = S_{(t)} - \beta\Delta_t S_{(t)} + \gamma I\Delta_t$$

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

$$I_{(t+\Delta_t)} = I_t + \beta\Delta_t S_t - \gamma I\Delta_t$$

$$\frac{dI}{dt} = \beta S - \gamma I \quad (5)$$

$$R_{(t+\Delta t)} = R_{(t)} + I\gamma\Delta_t$$

$$\frac{dR}{dt} = I\gamma \quad (6)$$

III. Results

Effect of Rates of Infection and Recovery on Number of Susceptible

From equation (1) and (4) the rate of decrease in the number of susceptible is similar when γ (recovery rate) is low (0.2) for levels of β (rate of infection) between 0.2 and 0.6 whereas an increase in β produces slower reduction in number of susceptible at high level of recovery rate γ for both original and modified model. The results show that the effect of rate of infection in reducing the number of susceptible is higher than the effect of recovery rate. In the original model ,By the 40th day, there does not remain any susceptible except at high recovery rate (0.6) and high infective rate (0.6) and low recovery rate (0.2) and low infective rate (0.2) when about 10% of the population still remains susceptible, while at the same time ,there does not remain any susceptible except at high recovery rate (0.6) and low infective rate (0.2) when about 25% of the population still remains susceptible in modified model . The number of susceptible decreases by 50% by 25days, 29 days, 25 days and 29 days when infective and recovery rates are (0.6, 0.2); (0.2, 0.2); (0.2, 0.6) and (0.6, 0.6) respectively in the original model while number of susceptible decreases by 50% by 23days, 25 days, 29 days and 33days when infective and recovery rates are (0.6, 0.2); (0.2, 0.2); (0.2, 0.6) and (0.6, 0.6) respectively in the modified model. For effective control, immunization should take place between 13 and 29 days, and between 15 and 33 days to avoid no susceptible in the population at low or medium rate of recovery and rate of infection in original and modified model respectively . High rate of recovery delays the reduction in number of susceptible in both original and modified model.

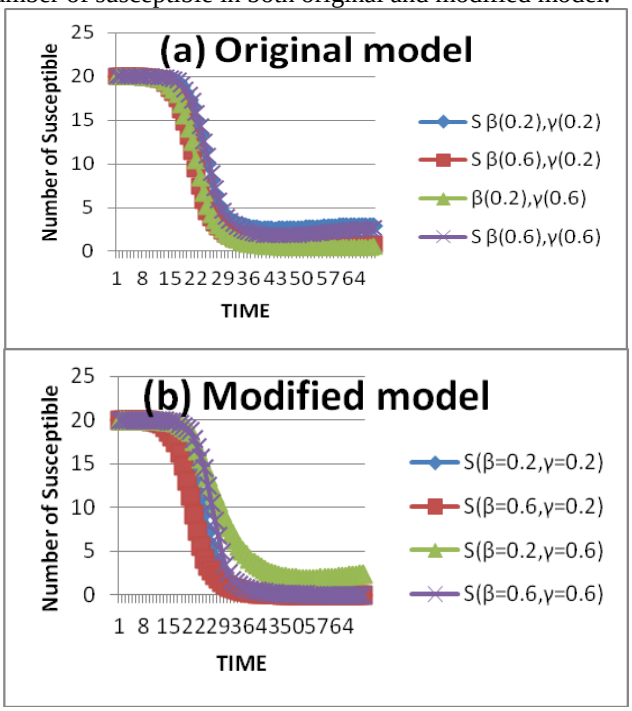

Fig. 1 Effect of infective and recovery rates on the numbers of susceptible

Effect of Rates of Infection and Recovery on Number of Infective

From equation (2) and (5) it was discovered that in the original model, the infective start from 2 while infective start from 0 in the modified model. The effect of increase in recovery rate on the number of infective is higher than the effect of increase in rate of infection in both original model. The rate of increase in number of infective is similar when infective rate (β) is 0.2 and 0.6 at low level of recovery rate. Increase in recovery rate however produces sharp decrease in the number of infective at both high and low levels of infective rate. The highest numbers of infective are obtained at about the 28th day for all combination of infective and recovery rates. By this time, about 100%, 85%, 60%, and 90% of the population will be infective for the combination of infective and recovery rates (β, γ) are (0.6, 0.2), (0.2, 0.2), (0.2, 0.6), and (0.6, 0.6) respectively in the original model, while in the modified model the highest numbers of infective are obtained at about the 28th day for all combination of infective and recovery rates and by this time, about 100%, 85%, 20%, and 40% of the population will be infective for the combination of infective and recovery rates (β, γ) are (0.6, 0.2), (0.2, 0.2), (0.2, 0.6), and (0.6, 0.6) respectively. This implies that high level of recovery rate and low level of infective rate give low infective. By 40 days 50%, 90%, 80%and 00% of the population would be infected when combinations of infective rates and recovery rates are (β=0.2,γ=0.6), (β=0.6,γ=0.2), (β=0.6,γ=0.6) and (β=0.2,γ=0.2) respectively in the original model, at this time 10%, 90%, 20%and 80% of the population would be infected when combinations of infective rates and recovery rates are (β=0.2,γ=0.6), (β=0.6,γ=0.2), (β=0.6,γ=0.6) and (β=0.2,γ=0.2) respectively in the modified model.

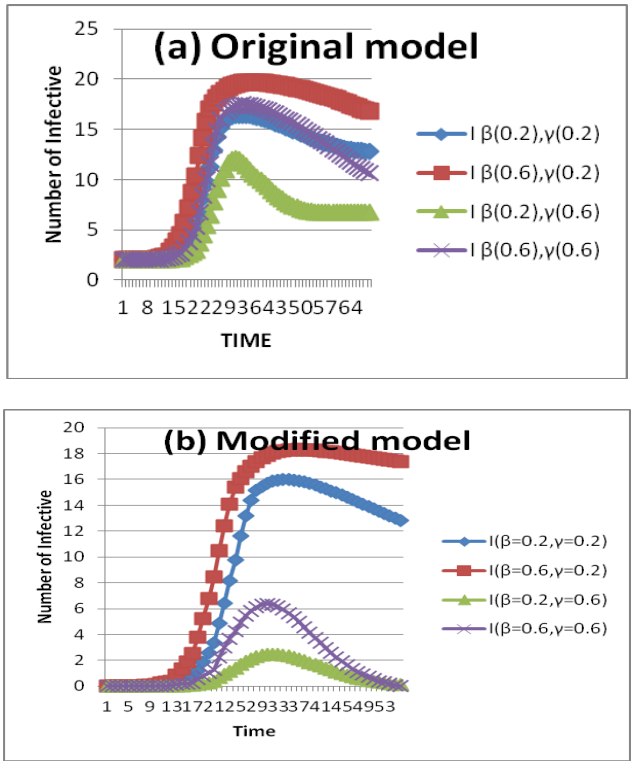

Fig. 2 Effect of infective and recovery rates on the numbers of infective

Effect of Rates of Infection and Recovery on Number of Recovery

Using the equation (3) and (6) it was discovered that there is no recovery in the first few days irrespective of infection and recovery rates in the original model but in contrast with original model in figure 2 where infective is 2a at the beginning. Also in the modified model it was discovered that there is no recovery in the first few days irrespective of infection and recovery rates this is because there are no infective during this period as observed in figure 2b. Thereafter, the numbers that recover depend mainly on the recovery rate and less on the rate of infection. The effect of high rate of recovery is more pronounced at high infective rate than at low infective rate. It was observed that by 40th day, 60%,10%,20% and 20% of the population would have recovered when infective and recovery rates are (β=0.2,γ=0.6), (β=0.6,γ=0.2), (β=0.6,γ=0.6) and (β=0.2,γ=0.2) respectively in both original and modified model. As would be expected, the numbers that recover depend mainly on recovery rate and less on infective rate. The effect of recovery rate is however higher since there are more infective on which the recovery rate can have effect.

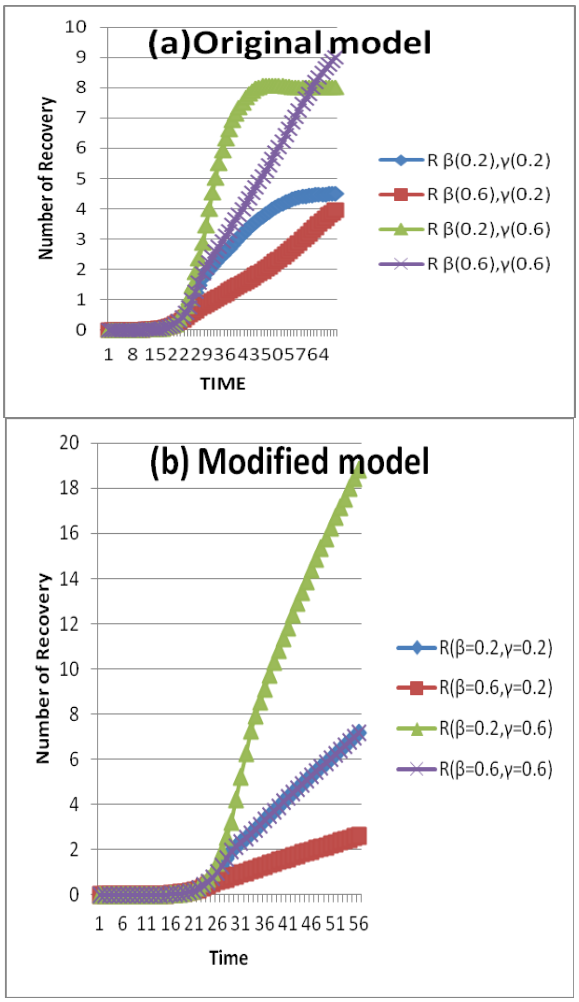

Fig. 3 Effect of infective and recovery rates on numbers that recover

IV. Conclusion

In this model, it is assumed that those who recover do not become susceptible and are not re-infectable . In the original and modified model, infective rate is the dominant factor affecting the number of susceptible. The results show that higher rates of infection result in rapid reduction in number of susceptible. The implication of this is that when the rate of infection of a disease is high, immunization or vaccination must commence early. This result is in consonant with equation (2). The main factor affecting the number of infective at the beginning of an epidemic is the rate of infection. The number of infective increases rapidly at the beginning with increase in infective rate but later moderated by recovery rate. At the beginning of an epidemic, the number of susceptible is high and therefore many are exposed to the disease with few infected and no recovery. The rate of infection is therefore the only factor that affects the number that will be infected. As the number of infected however increases, it is reduced by the rate of recovery. At this stage, the higher the recovery rate the lower the number infected. High level of recovery rate and low level of infective rate give minimum number of infective. There is no recovery at the initial stage until there is a number that is infected. Thereafter, the number of recovery becomes the dominant factor. The rate of infection (which determines the number infected) is also a moderating factor. High recovery rate is essential to reduce the number infected. Low infection rate and high recovery rate are required to delay infection, reduce the number infected and ensure maximum recovery. At these rates, a minimum of 85% of the susceptible population needs to be immunized to control transmission of the disease in the both original and modified.

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