

# Dynamics of a modified SIR model

Zhen Gou, Linda Li  
Duke University

May 3, 2012

## 1 Introduction

In modeling the spread of infectious diseases, we can distinguish between three main classifications of behavior - epidemic, endemic, and outbreak. An epidemic disease is one in which intense outbreaks are followed by disappearance; examples include plague and cholera. An endemic disease is one in which there are relatively small fluctuations in the monthly case count, and only slow increases or decreases over the course of years. Examples include leprosy and tuberculosis. Finally, an outbreak is where the disease remains present in the population but flares up at frequent intervals, such as is the case with measles, malaria, and dysentery.

The SIR model is a basic model for monitoring the progress of an epidemic with immunity conferred by infection. In this model, S represents susceptibles, I, infectives, and R, recovered/removed. The original SIR model is defined below:

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

$$\frac{dI}{dt} = \beta SI - \gamma I \quad (1b)$$

$$\frac{dR}{dt} = \gamma I \quad (1c)$$

In this model,  $\beta$  represents the transmission rate from the interaction of susceptibles and infectives in the population.  $\gamma$  is the rate of recovery from the disease; therefore,  $1/\gamma$  is interpreted as the mean duration of infection.

There are some key assumptions present in the basic model. First, there is no birth or death from any category of the population, and second, infection always confers permanent immunity. These assumptions, however, are relatively unrealistic. Births and deaths could induce dramatic changes to the behavior of the system. In addition, for many diseases, infection only confers immunity for a certain period of time. Thus, we propose a new model as follows:

$$\frac{dS}{dt} = AS - kS^2 - \beta SI + \phi R \quad (2a)$$

$$\frac{dI}{dt} = \beta SI - C_1 I \quad (2b)$$

$$\frac{dR}{dt} = C_2 I - \delta R \quad (2c)$$

In this modified SIR model, we define several new parameters.  $A$  is the net population growth of susceptibles, absorbing births and deaths of susceptibles into one term.  $k$  is the logistic population growth factor.  $\phi$  is the rate of loss of immunity.  $C_1$  is the death rate plus the recovery rate of infectives.  $C_2$  is the recovery rate of infectives.  $\delta$  is the death rate plus the rate of loss of immunity of recovered. By definition, two conditions must hold.  $C_1 \geq C_2$ , and  $\delta \geq \phi$ . Throughout our analysis, we seek to develop an intuitive understanding of the parameters, particularly at bifurcations. The goal is to analyze the behavior of the modified SIR system and relate the model to its real-life implications.

## 2 Model analysis

To analyze the modified SIR model and simplify calculations, we rescale the system of equations from 7 to 4 parameters. There are several ways to rescale the system; however, we choose the following method to avoid undefined parameters where the denominator could equal zero. The scaling method and the rescaled system are defined below:

$$\bar{S} = \frac{\beta}{C_1} S, \bar{I} = \frac{\beta}{C_1} I, \bar{R} = \frac{\beta}{C_2} R, \bar{t} = C_1 t$$

\*From here on,  $S$ ,  $I$ , and  $R$  refer to  $\bar{S}$ ,  $\bar{I}$ , and  $\bar{R}$ .

Rescaled system:

$$\frac{dS}{dt} = gS - hS^2 - SI + eR \quad (3a)$$

$$\frac{dI}{dt} = SI - I \quad (3b)$$

$$\frac{dR}{dt} = I - fR \quad (3c)$$

where

$$g = A/C_1, h = k/\beta, e = \phi C_2/C_1^2, f = \delta/C_1$$

When there is positive net population growth ( $A > 0$ ), this system has three equilibria.

1. The first equilibrium is at the origin. This refers to the state where the population is extinct. Under realistic parameters, this point is always a saddle.

2. The second equilibrium occurs on the S axis at the point  $(\frac{g}{h}, 0, 0)$ . At this equilibrium, the disease dies out (epidemic). This point can be a stable node or a saddle depending on the values of the parameters (detailed later in 3.1).
3. The third equilibrium occurs with all three classes of the population present in the population. This point,  $(1, f\frac{h-g}{e-f}, \frac{h-g}{e-f})$ , is of primary interest in our analysis.

When there is negative or zero net population growth ( $A \leq 0$ ), the origin is the only relevant equilibrium. In the case where net population growth is zero ( $A=0$ ), the equilibrium with only susceptibles in the population coincides with the origin.

### 3 Case analysis

We analyze the modified SIR model in two different cases since they represent distinct real-world situations.

#### 3.1 Permanent immunity

When a disease confers permanent immunity upon infection, there is no flow from the recovered to the susceptible class. In the model, this translates to  $\phi=0$ , hence,  $e=0$ . Examples of diseases under this case include smallpox and chickenpox. The Jacobian of the system becomes:

$$\begin{pmatrix} g - 2hS - I & -S & 0 \\ I & S - 1 & 0 \\ 0 & 1 & -f \end{pmatrix} \quad (4)$$

We find the three equilibria identified above. The first equilibrium at the origin has eigenvalues -1, g, and -f. The second equilibrium at  $(\frac{g}{h}, 0, 0)$  has eigenvalues -g,  $\frac{g}{h} - 1$ , and f. The third equilibrium at  $(1, g-h, \frac{g-h}{f})$  has eigenvalues -f and  $\frac{-h \pm \sqrt{h^2 - 4(g-h)}}{2}$ . We continue with a behavior analysis of the three main disease classifications in the case with permanent immunity.

##### 3.1.1 Epidemic

Note that when  $g < h$ , the Jacobian at the S axis equilibrium has three negative eigenvalues; thus, it is a stable node with no spiral behavior. The origin remains a saddle and the third equilibrium goes to an irrelevant octant. This means that starting at any initial condition in the relevant octant with the disease present, the trajectory eventually reaches the equilibrium on the S-axis. In real-life, this refers to the situation in which a disease eventually dies out after it is introduced into the population (epidemic). See Figure 1.

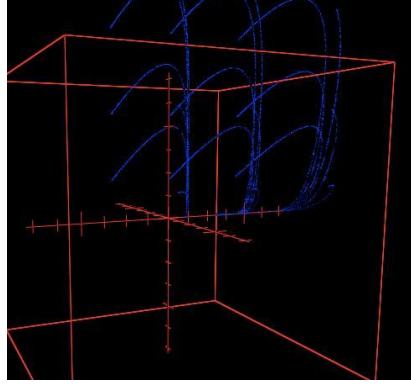


Figure 1: Epidemic behavior

This epidemic behavior occurs when  $g < h$ , therefore,  $\beta < kC_1/A$ . The disease transmission rate ( $\beta$ ) must be below some critical value that depends on the logistic growth factor, net population growth, and the rate of outflow of infectives (death rate plus recovery rate of infectives), and does not depend on the severity of the introduction of the disease. The epidemic behavior may disappear when the transmission rate increases, the logistic growth factor decreases, the recovery rate decreases, or the net population growth rate increases. Intuitively, it makes sense that fast population growth could prevent a population from eradicating a disease completely since there is always steady inflow of susceptibles. It also makes sense to conclude that a disease may not die out when transmission increases and recovery decreases.

### 3.1.2 Endemic

An endemic behavior emerges when  $g > h$ , or  $\beta > kC_1/A$ . Assuming that all parameters are positive, all three equilibria occur in the relevant octant. Both the origin and the equilibrium on the  $S$  axis are saddle points, while the third equilibrium becomes a stable spiral. This means that after the disease is introduced into the population, the number of incidences will fluctuate and gradually reach a stable level. In other words, the disease behaves like an outbreak before eventually becoming endemic. See Figure 2.

Note that the stable spiral node has two complex eigenvalues  $\frac{-h \pm \sqrt{h^2 - 4(g-h)}}{2}$ . Therefore, the value of  $h$  determines the attraction force of this node. Since  $h = k/\beta$ , the disease incidences fluctuate for a longer period of time before reaching a stable level when  $\beta$ , the disease transmission rate, is higher. See Figure 3.

In addition, a change in the value of  $g$  in the imaginary part of the two complex eigenvalues affects the duration of each cycle of the spiral. Since  $g = A/C_1$ , a higher net population growth rate or lower rate of outflow of infectives results in a shorter period for each fluctuation since the imaginary part of the complex

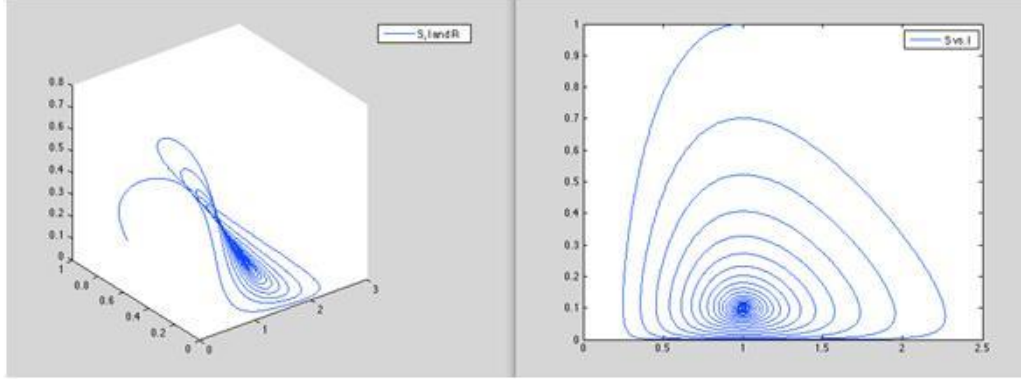


Figure 2: Endemic behavior

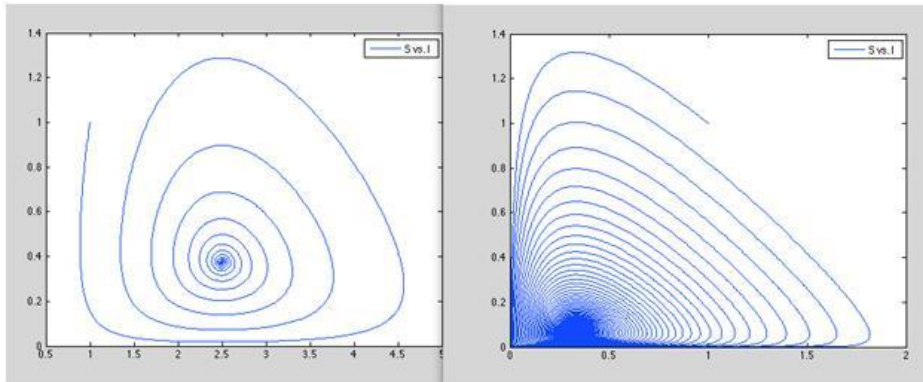


Figure 3: Epidemic behavior, low  $\beta$  vs. high  $\beta$

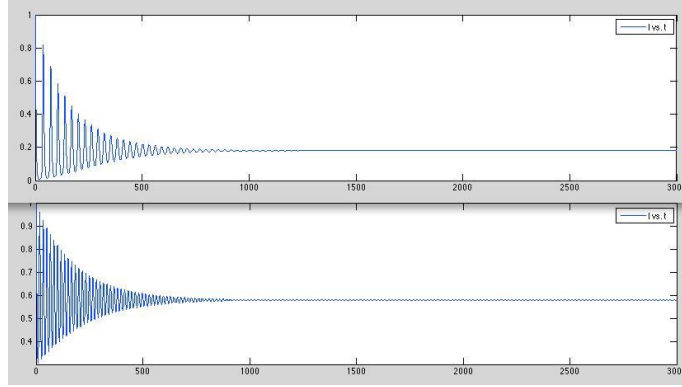


Figure 4: Epidemic behavior, low A vs. high A

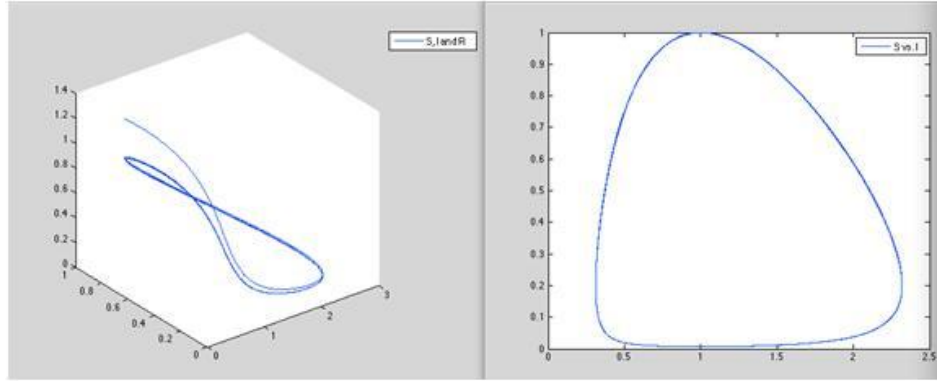


Figure 5: Outbreak behavior

eigenvalues increases in magnitude. The time it takes to reach the steady state, however, is not affected. See Figure 4 .

### 3.1.3 Outbreak

Given that the two complex eigenvalues of the Jacobian at the third equilibrium are  $\frac{-h \pm \sqrt{h^2 - 4(g-h)}}{2}$ , a Hopf bifurcation occurs when  $h=0$ . When  $h>0$ , the real part is negative and the equilibrium is a stable spiral. The case when  $h<0$  is irrelevant because  $h=k/\beta$ , and both  $k$  and  $\beta$  are assumed to be positive. Our analysis is confirmed by numerical simulations in MATLAB showing periodic solutions at the bifurcation point ( $h=0$ , i.e.  $k=0$ , meaning no population growth logistic factor); however, the stable manifold of this equilibrium is a tilted plane. Within a certain boundary of initial conditions, periodic solutions exist everywhere, as in the basic predator-prey model. See Figure 5.

If an initial condition is chosen outside this manifold, however, the trajectory

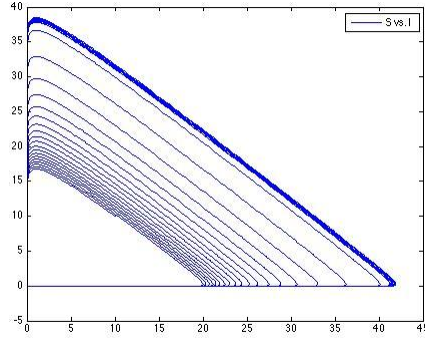


Figure 6: Outbreak behavior, initial condition outside manifold and inside larger orbit

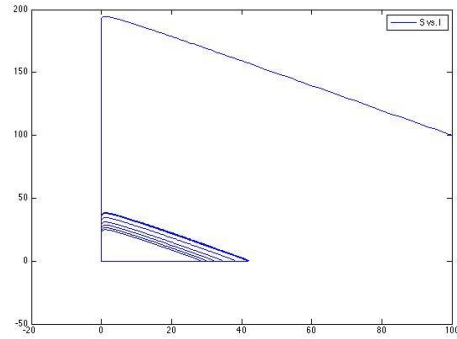


Figure 7: Epidemic behavior, initial condition outside larger orbit

spirals outwards towards another larger orbit, call it the global orbit, containing this equilibrium, and stays there. See Figure 6. Any initial condition outside the larger orbit will go to an orbit near the stable manifold and then go outwards, converging to the global orbit. See Figure 7. A sketch of the global behavior is shown in Figure 8.

Since the system has periodic solutions at  $k=0$  and  $\phi=0$ , we ask whether a very small perturbation of  $k$  or  $\phi$  would destroy the periodic solution. To do this, we test two cases: 1)  $k=0$ ,  $\phi=0.000001$  and 2)  $k=0.000001$ ,  $\phi=0$ . Both perturbations destroy the periodic solution. Although not immediately apparent due to the very small attraction force, a zoom in of the produced graphs reveals nonperiodic behavior. See Figure 9.

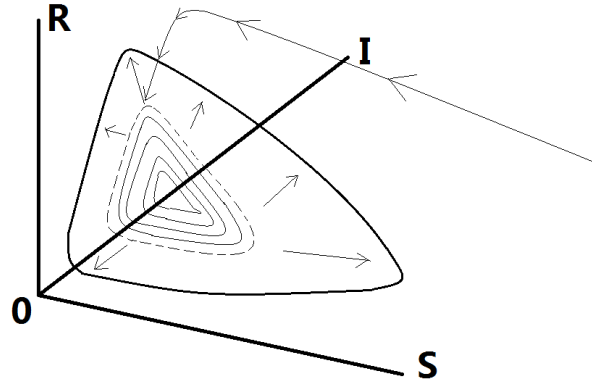


Figure 8: Outbreak behavior, global behavior

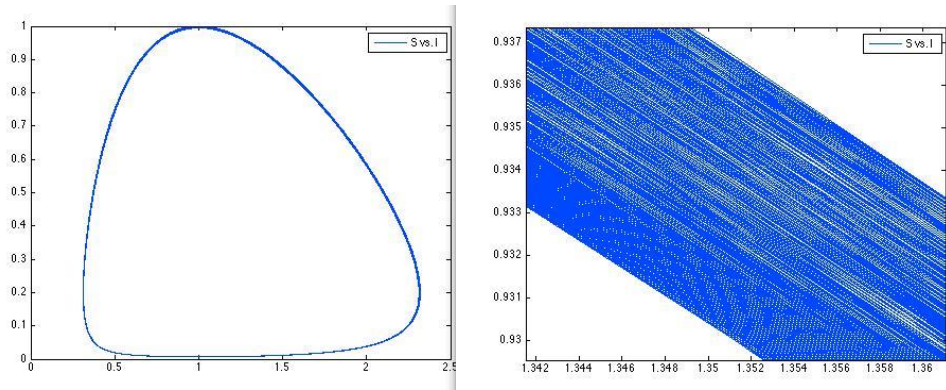


Figure 9: Outbreak behavior, perturbation of  $k$  or  $\phi$



### 3.2 Temporary immunity

When a disease confers non-permanent or temporary immunity upon infection, there is flow from the recovered to the susceptible class. In the model, this translates to  $\phi \neq 0$ , hence,  $e \neq 0$ . Examples of diseases under this case include influenza (different strains) and malaria. The Jacobian of the system becomes:

$$\begin{pmatrix} g - 2hS - I & -S & e \\ I & S - 1 & 0 \\ 0 & 1 & -f \end{pmatrix} \quad (5)$$

In this general case, we continue to observe three equilibria. The analysis for equilibria at the origin and the point on the S axis remain unchanged from 3.1. We are most interested in analyzing what happens to the third equilibrium at  $(1, f \frac{h-g}{e-f}, \frac{h-g}{e-f})$  with all three classes present in the population.

In order to avoid solving for the eigenvalues explicitly, we instead attempt to find bifurcations using the following test. Proposition 2.4.5 from Schaeffer and Cain holds that if A is a 3 x 3 matrix with real entries, then  $\text{Re}(\lambda_j) < 0$  if and only if the following three conditions are satisfied:

1.  $\text{tr}(A) < 0$
2.  $\text{tr}(A)^2 - \text{tr}(A^2) < 2\det(A)/\text{tr}(A)$
3.  $\det(A) < 0$

To look for a Hopf bifurcation, we look primarily at condition 2. We want to solve for when  $\text{tr}(A)^2 - \text{tr}(A^2) = 2\det(A)/\text{tr}(A)$ . Calculating the traces and the determinant, and plugging them into the equation, we obtain  $(g - 2h - V - f)^2 - [(g - 2h - V)^2 - 2V + f^2] = 2f \frac{h-g}{g-2h-V-f}$  where  $V = f \frac{h-g}{e-f}$ . Inputting parameters satisfying this condition, we do not observe a change in behavior from the original stable spiral. See Figure 10. We conclude that if bifurcations do occur, they occur outside of the first octant; therefore, they are numerically unobservable starting from feasible initial conditions.

When a disease does not confer permanent immunity upon infection, there are two possible behaviors. Under certain parameters and initial conditions, the disease can die out (epidemic), reaching the S axis equilibrium. In the other case where the disease remains in the population, our analysis suggests that the disease does not remain an outbreak (no periodic solutions found) but rather, reaches a stable equilibrium (endemic).

## 4 Further extension of model

As seen in 3.1.3, when there is permanent immunity upon infection ( $\phi=0$ ) and no logistic factor limiting population growth ( $k=0$ ), a periodic orbit appears. As stated before, this indicates that the disease is an outbreak, coming and going over time. Based on this observation, we seek to understand how the introduction of vaccination affects the behavior of the system. Vaccinations

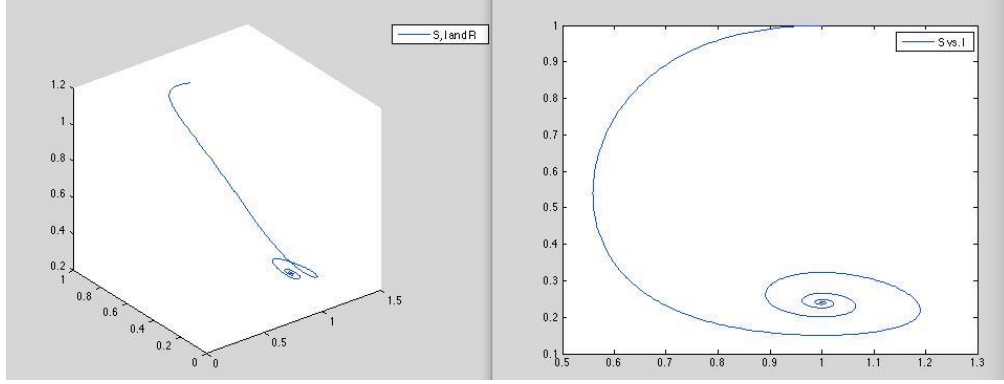


Figure 10: Typical behavior, stable spiral

can grant permanent immunity to some diseases, therefore adding flow from the susceptible class to the recovered class without passing through the infected class. Thus, we derive the following modified SIR system with vaccination, in which a  $\nu S$  term is added to S and a  $+\nu S$  term to R:

$$\frac{dS}{dt} = AS - \beta SI \quad (6a)$$

$$\frac{dI}{dt} = \beta SI - C_1 I \quad (6b)$$

$$\frac{dR}{dt} = C_2 I - \delta R + \nu S \quad (6c)$$

Here,  $\nu$  represents the proportion of susceptibles who receive vaccination. We redefine A, the net population growth rate, to absorb the new  $\nu S$  term in S. Rescaling using the same method as in the Model analysis, we obtain the following Jacobian matrix:

$$\begin{pmatrix} g - 2hS - I & -S & e \\ I & S - 1 & 0 \\ \frac{\nu}{C_2} & 1 & -f \end{pmatrix} \quad (7)$$

Comparing to the Jacobian of the modified SIR model without vaccinations (5), the matrix is almost identical. The only difference is to the entry in row 3, column 1, which was zero previously. In the case we are examining with permanent immunity ( $e=0$ ), the Jacobian entry 3,1 does not affect the characteristic equation. We obtain the same eigenvalues at each equilibrium as before; therefore, we expect the behavior of the trajectories near the equilibrium points to remain the same.

We confirm this conjecture numerically using MATLAB. Interestingly, the global behavior of the system also remains unchanged. This implies that vaccination does not affect the pattern of disease transmission. Nevertheless, recall that the parameter A is redefined from before to represent the net population

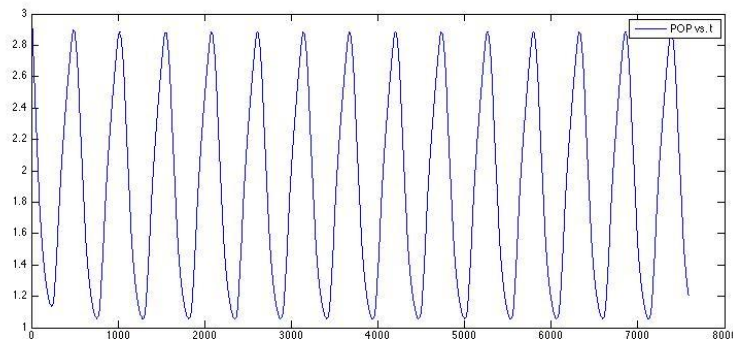


Figure 11: Outbreak behavior, total population over time

growth rate minus the vaccination rate of susceptibles, thus the definition of a positive, non-zero parameter  $A$  changes. If the rate of vaccination of susceptibles exceeds the net population growth rate, then the  $A$  is negative, removing all equilibria except for the origin from the octant of interest. Introducing vaccination of a disease into the system may prevent the population from reaching a stable equilibrium and eliminate the outbreak pattern of the disease (more discussion of the model's real-life relevance in this case in the conclusion).

## 5 Conclusion

By introducing births and deaths, and the possibility of temporary immunity into the model, we show that these factors can affect the behavior of the SIR model. Applying both analytical and numerical methods, we find situations under which epidemic, endemic, and outbreak behaviors occur.

Although the modified SIR system developed in this paper addresses some of the unrealistic assumptions of the original SIR model, there are a number of weaknesses to recognize in the new system. Firstly, our model can only be realistic with positive net population growth. When there is negative or zero net population growth, we only have one stable equilibrium at the origin. Thus, the population dies out regardless of the initial conditions chosen. In real-life, we expect a population to eventually reach a non-trivial stable equilibrium. Secondly, in the case where periodic solutions exist, our model predicts fluctuations that are too dramatic to be realistic. Figure 11 shows these extreme fluctuations in total population in which the population alternates between being almost extinct and flourishing.

In conclusion, our modified SIR model captures qualitative behavior of infectious diseases, but has less quantitative predictive power, which could be a focus for future research of the SIR model.

## 6 References

- Anderson, R. M. and May R. M. (1991) Infectious Diseases of Humans: Dynamics and Control. Oxford: Oxford University Press
- D.G. Schaeffer and J.W. Cain, ODEs: A Bridge Between Undergrad and Graduate Math. In preparation, 2011.
- Ellner, S. P. and Guckenheimer, J. (2006) Dynamic Models in Biology. Princeton: Princeton University Press