

Prevalence of Infection in Seasonally Forced Compartmental Models

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Abstract

Seasonal Infection modeling is used to describe the behavior of infection during various seasonal cycles within a fixed population. We use compartmental differential equations to achieve an understanding of the behavior of each aspect of infection and transmission that is assigned to each equation. We carry out with graphical data based upon calculated sensitivity equations that are compared over time for each of the parameters involved in the model.

Introduction

Seasonal compartmental models are used to simulate and predict the changes in a population that is affected by an outbreak of disease. Forcing is used to simulate the effects of seasonal changes in transmission and infection. Here, the forcing serves to change the constant nature of the model's transmission rate /beta into a sinusoidal curve such that it resembles the natural change in the ebb and flow of susceptibility to infection (S), Infection (I), and recovery from infection (R).

Three models were studied, one that had a basic S,I,R compartment structure where beta was the transmission rate, gamma served as the infection recovery rate, and mu was the death rate from infection. The second model was similar to the first with the exception that mu was removed, due to the low incidence of death among many seasonal infections. The third model had two infected compartments, one for infected and untreated, and the second for infected and treated with vaccination.

The following equations were entered into a MATLAB program to solve the differential equations and to graph the results

SIR Model 1 with Death from Infection

$$\begin{aligned} S' &= \mu N - \frac{\beta S I}{N} - \mu S \\ I' &= \frac{\beta S I}{N} - \gamma I - \mu I \\ R' &= \gamma I - \mu R \end{aligned}$$

$$\beta(t) = \beta_0(1 + \beta_1 \cos(2t\pi))$$

SIR Model 2 with Single Outbreak

$$\begin{aligned} S' &= \frac{\beta S I}{N} \\ I' &= \frac{\beta S I}{N} - \gamma I \\ R' &= \gamma I \end{aligned}$$

$$\beta(t) = \beta_0(1 + \beta_1 \cos(2t\omega\pi))$$

SIR Model 3 with Vaccination

$$\begin{aligned} S' &= \beta(t)S \frac{I_u + \sigma I_{tr}}{N} \\ I'_u &= \beta(t)S [1 - f_0] \frac{I_u + \sigma I_{tr}}{N} - \gamma_u I_u \\ I'_{tr} &= \beta(t)S f_0 \frac{I_u + \sigma I_{tr}}{N} - \gamma_{tr} I_{tr} \\ R' &= \gamma_u I_u + \gamma_{tr} I_{tr} \end{aligned}$$

$$\beta(t) = (\beta_0(1 + \beta_1 \cos(2t\omega\pi)))$$

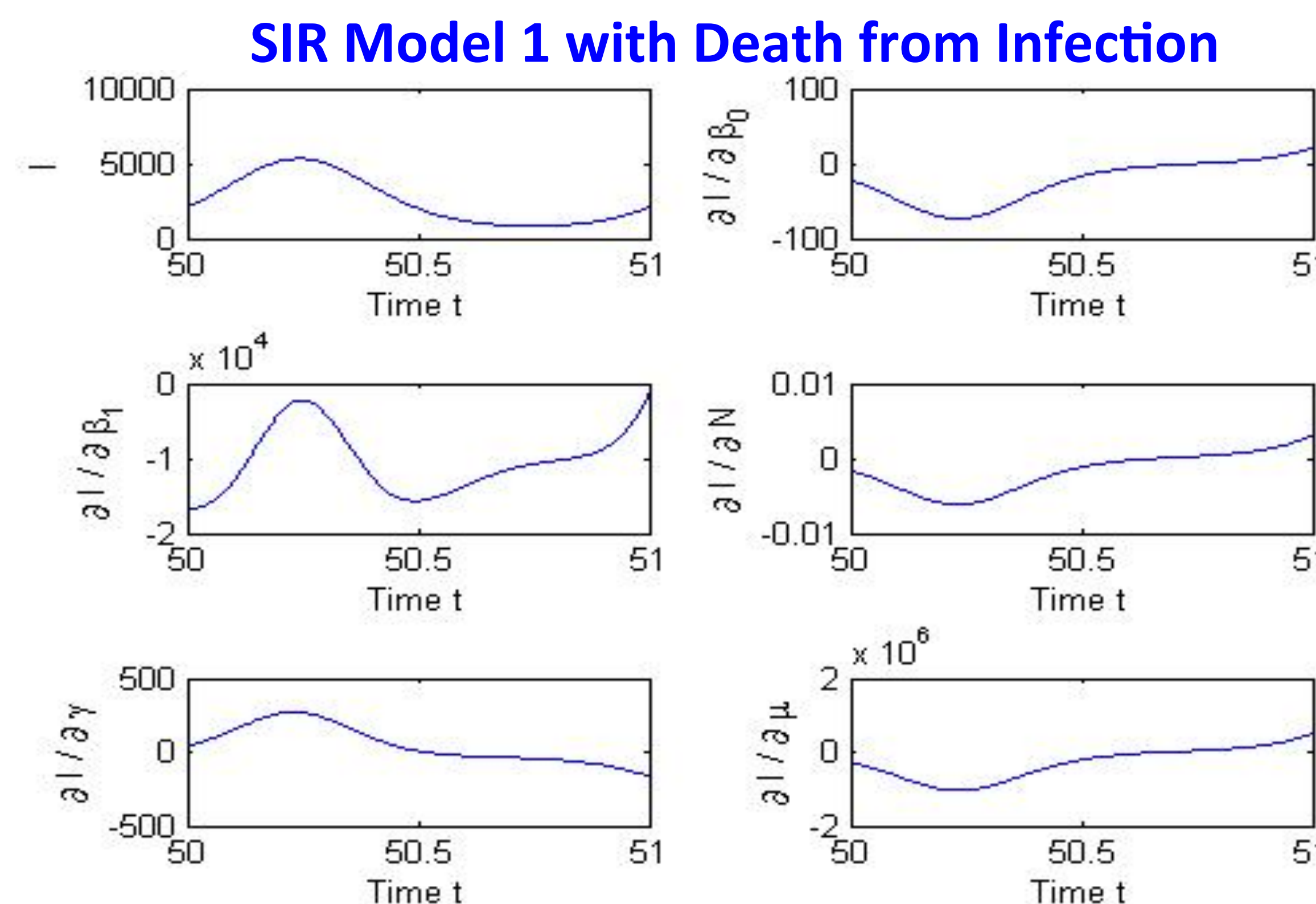


Figure 1: MATLAB generated graphs of Model 1 with Beta(1)=.1, N=10^7, Beta(0)=500, Gamma=50, and Mu=1/70;

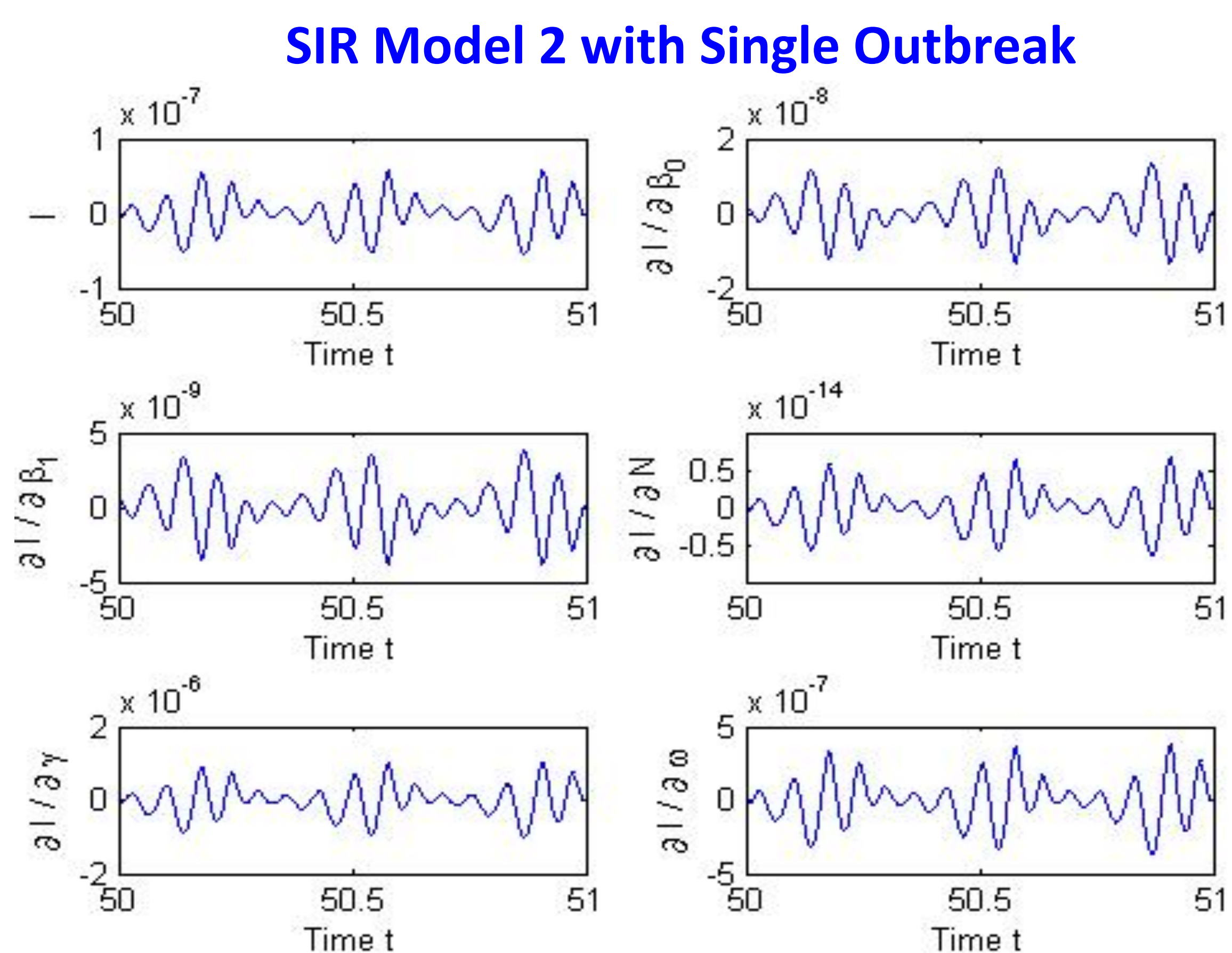


Figure 2: MATLAB generated graphs of Model 2 with Beta(1)=1/3, N=10000, Beta(0)=.5, Gamma=50, Omega=1/365, r0=gamma/Beta(0), sbar=n/r0, and ibar=(n/gamma)*r0

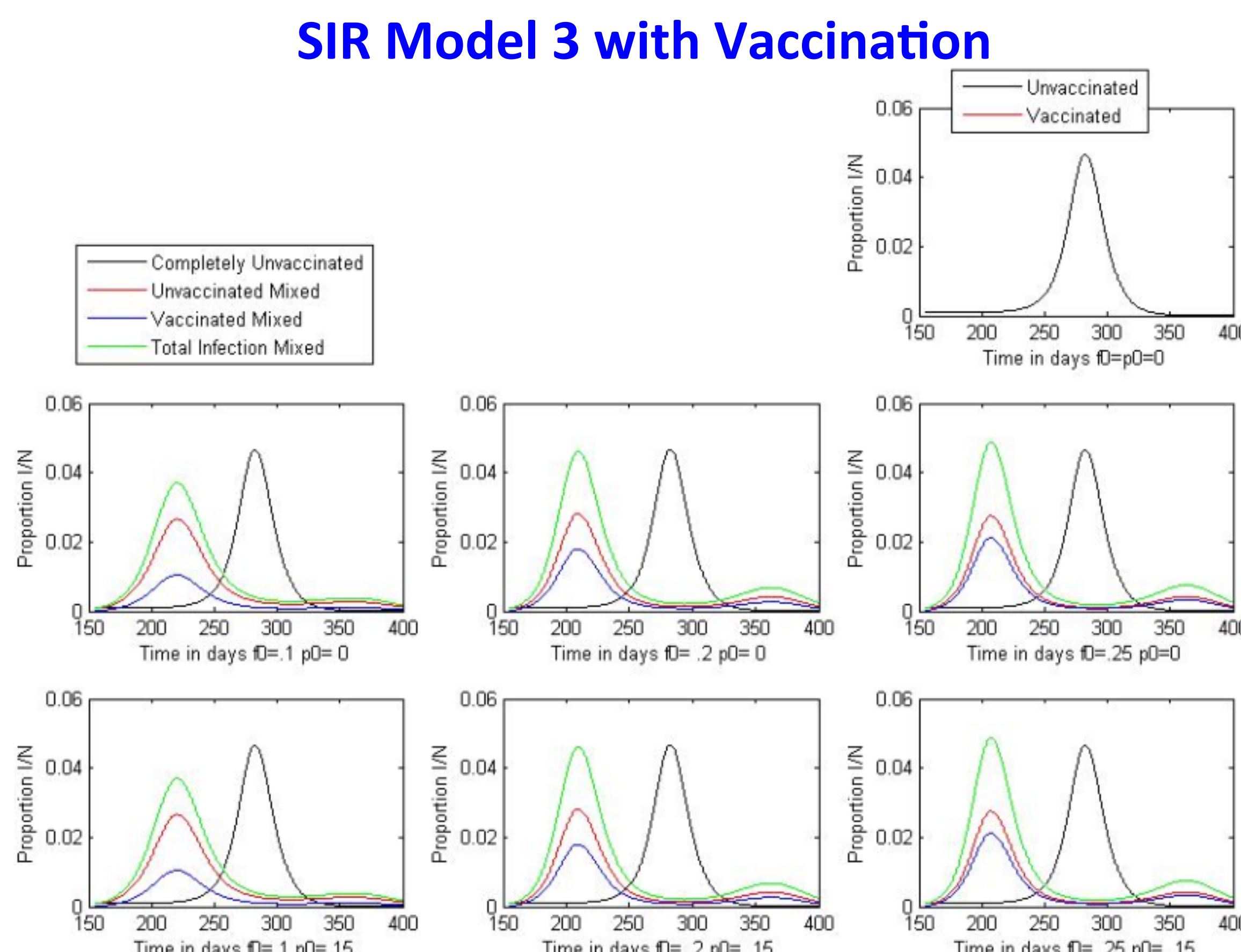


Figure 3: MATLAB generated graph of Model 3 with multiple types of vaccination and different parameters.

SIR Model 3 Sensitivity Equations

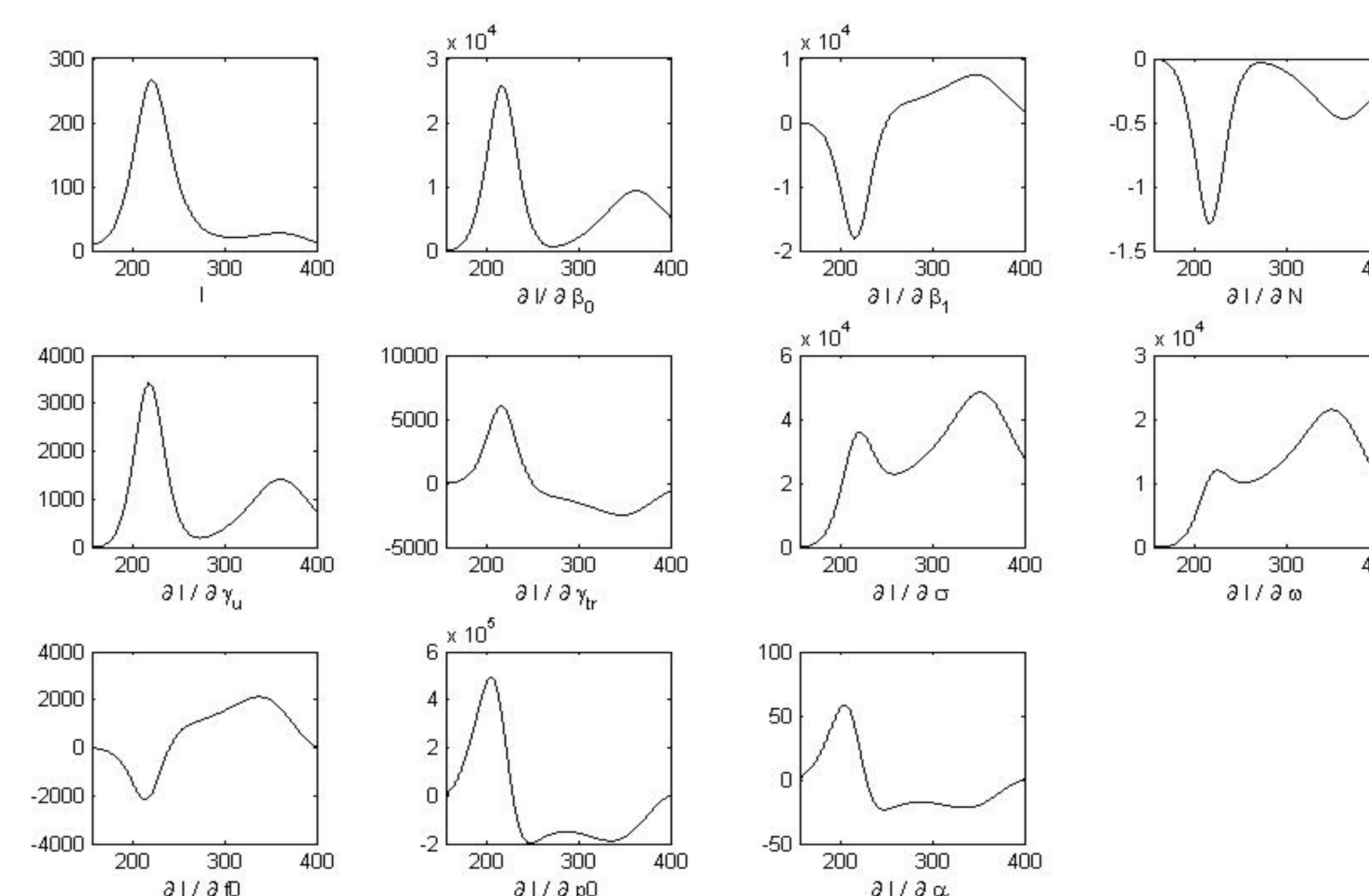


Figure 4: MATLAB generated sensitivity graphs of Model 3 with various parameters.

Discussion

The graphs in figure 1 are showing the partial derivatives of each parameter of the SIR Model 1 equations. These graphs are consistent with the graph of the infected population. Gamma is consistently negative, and is at its least when the transmission is at the highest. This makes logical sense, as more persons will be moving into the recovered compartment when the disease is at its peak.

The graphs in figure 2 are also showing the partial derivatives of the infected population with respect to each parameter. This model shows the effects of forcing upon the system, as the parameter omega is introduced to change the period of the system. This system oscillates in a manner that indicates that the transmission parameters are changing in opposition to the flow of the infected parameter. This indicates that the infection is likely passed when the person is not aware of their illness, as if the infection had a dormant phase.

The graphs in figure 3 model a completely unvaccinated system (top right) against a system that is adjusted to varying initial conditions f0 and p0. These initial conditions have the surprising effect of lengthening the time span of the infection, and resulted in the same peak proportion as the untreated population. The results shown here indicate that it is necessary to make the initial conditions time-dependent upon the outbreak of the infection in the untreated population, thus making the goal of treating the infection to cause the outbreak to occur later, and be of less magnitude than the untreated cases.

Discussion (cont.)

Working with the model that is adjusted to the initial conditions of f0=.1, and p0 = 0, the partial derivatives were graphed to look at their individual effects upon the system. Here, Beta0 peaks just days before the first peak of the outbreak indicating that the infection is likely spread a few days before symptoms are severe. The recovery rate of treated individuals is also higher than that of the untreated individuals, and the recovery rate if treated individuals are negative during the second peak. This indicates that the infection's second wave is reduced by the first round of treatment, and it may be wise to treat before the outbreak occurs, and adds to the conclusion that more research is needed on the time dependence of the treatment system. While f0 is almost inversely proportional to the infected population, p0 and alpha follow closely during the first peak and erratically during the second peak. These figures indicate that the purpose of f0 is reduce the number of infected initially, and that p0 and alpha reduce the number of subsequent infections.

Conclusion

It is clear that the type of infection modeled has a large impact on the type of model to use. When fitting data to a model set, it may help to understand the behavior and transmission of the infection. The transmission rate Beta is crucial to determining the magnitude and frequency of the model, and if the Beta parameters are not correct, then the model may not make accurate predictions. More research is needed to work on the third data set, and a further understanding of the role of f0, p0, sigma, and time dependence is needed.

References

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