Analysis of SIR and SEIR models and their ODE's

Introduction to Computational Science

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ABSTRACT

This research presents a review of SIR and SEIR models respectively. The former model is examined in detail by numerical integration and space diagrams, analyzing the effect of available parameters. Alternatively, the model is fitted to an example dataset to provide some practical insight. The SIR model is thereafter expanded by the addition of birth and mortality rates, either natural of infection-induced. Ultimately, seasonality and the extra state of being exposed is added to the model, resulting in the SEIR dynamics that are analyzed similarly.

KEYWORDS

SIR, SEIR, Epidemics, Infectious disease, Vaccination, Mortality

1 INTRODUCTION

The year 2020 is one of the most epidemic-oriented years in recent history, the novel coronavirus has again shown global human society its weakness regarding the overcoming of infectious disease. Quantification of the behavior of such a disease is of major importance, for current developments or future prevention of spread of infectious disease. Various recent research investigates the spread of the novel coronavirus by a SIR (Susceptible-Infected-Recovered) model [2] [3]. This model was developed in the early twentieth century and follows from the Kermack-McKendrick model that originates from 1927 [4], it is a compartment model that provides a clear-cut insight into the spread of infectious disease [1]. It depends on Ordinary Differential Equations (ODE) and can be adapted to fit various disease specifics. However the models in this research might lack some of the complexity necessary to successfully describe spread of the novel coronavirus, they provide insight into underlying mechanisms and parameter influence. This research primarily focuses on the SIR model, comparing various demographic approaches, exploring small vaccination calculations and providing phase-space diagrams. Eventually, some Fourier analysis is performed and the expansion to a SEIR model is implemented and evaluated.

2 THEORETICAL BACKGROUND

The most approachable version of the SIR model, that disregards nativity, mortality and migration, assumes a small infectious proportion of the population to infect a significant amount of others within a short period of time. Regardless of this infection causing an actual epidemic or not, the infection spread is assumed to be fast and demographics are assumed to be disregardable. The model works with a closed population and also ignores contact patterns of infections, so it assumes homogeneous mixing [5].

This research uses a frequency dependent transmission term and the rate of transmission is therefore equal to βSI, the ODE's then look as follows:

$$\frac{dS}{dt} = -\beta SI \tag{1}$$

$$\frac{dI}{dt} = \beta SI - \gamma I \tag{2}$$

$$\frac{dR}{dt} = \gamma I \tag{3}$$

where γ represents the recovery rate. These equations can be numerically analyzed to calculate basic reproductive ratios (R_0) and asymptotic states, as will be done in the following sections. The R_0 can be defined as *the average number of secondary cases arising from an average primary case in an entirely susceptible population* [5]. This rate does not only show if an infectious disease will become an epidemic (when it is larger than 1.0), it can also help provide insight into the composition of vaccination plans as it is related to the proportion of susceptible individuals. In the SIR model, the reproductive ratio can be quantified as follows:

$$R_0 = \frac{\beta}{\gamma} \tag{4}$$

More specifically, Kermack & McKendrick [4] show that an infectious disease can only cause an epidemic if there is an initial susceptible population greater than $\frac{1}{R_0}$. This can in turn be used to determine the number of individuals that need vaccination to prevent an epidemic from happening.

2.1 Demography

The basic SIR model described above is first expanded by adding natural birth and death rates, as well as infection-induced mortality. The former two are implemented to be equal, so the population size is still constant and the demography solely introduces some oscillations in the population distributions. The latter, on the other hand, will remove people from the population and influence the fraction of recovered individuals.

$$\frac{dS}{dt} = \mu - \beta SI - \mu S \tag{5}$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I \tag{6}$$

$$\frac{dR}{dt} = (1-\rho)\gamma I - \mu R \tag{7}$$

where β and γ are defined as before, μ represents the natural nativity/mortality rates and ρ represents the infection-induced mortality. Again, the reproductive ratio can be calculated to provide

insight into the occurrence of an epidemic and vaccination decisions, it slightly changes and now looks as follows:

$$R_0 = \frac{\beta}{\gamma + \mu} \tag{8}$$

2.2 SEIR model

Various infectious diseases that have been historically observed show presence of additional stages in the structure of disease spread. One of these important phenomena is defined as the *incubation period*, representing the period of time where an individual has been infected with a disease and is not yet infectious to others. Introducing this phenomenon into the SIR model leads to an extra state of being *Exposed* before landing in the *Infected* state, resulting in the SEIR model. The incubation period is assumed to be a random variable that is exponentially distributed with parameter a (so average incubation period is $\frac{1}{a}$). The parameters from the SIR model remain intact and this results in the following ODE's:

$$\frac{dS}{dt} = \mu - \beta SI - \mu S \tag{9}$$

$$\frac{dE}{dt} = \beta SI - aE - \mu E \tag{10}$$

$$\frac{dI}{dt} = aE - \gamma I - \mu I \tag{11}$$

$$\frac{dR}{dt} = (1 - \rho)\gamma I - \mu R \tag{12}$$

However this research will not focus on infection-induced mortality in combination with the SEIR model, as the effect is similar in SIR model, the $(1 - \rho)$ is still in the final ODE for clarity. ρ can be assumed to be equal to 0 in the SEIR models in this research.

The ODE's of the SEIR model can again be analyzed and the quantification of the reproductive ratio R_0 now significantly changes to become:

$$R_0 = \frac{a\beta}{(a + \mu)(\gamma + \mu)} \tag{13}$$

2.3 Seasonal effects

As infectious disease can show a rate of transmission that varies over time, often varying in a periodically increasing and decreasing way, seasonal effects can improve reliability of results from epidemic modelling. Variation over time can be neatly implemented by a time varying sinusoidal rate of transmission [6]. ODE representations as provided before will all still hold, however an adjustment of β is necessary and usually looks as follows:

$$\beta_t = (1 + c\cos(dt))\beta_0 \tag{14}$$

where β_0 is a base or average rate of transmission, c influences the amplitude and d influences the period of time-variation.

2.4 Fitting data

Experimental or observed data can be fitted to the models mentioned above, according to the nature of the data, in various ways. The ODE's of an appropriate model can be numerically integrated and available data can be fitted by, for example, a least-squares method. This research provides some elementary analysis on a closed population dataset, fitting the data by minimizing the following least-squares equation:

$$\sum_{i}^{N} (y_{i,observed} - y_{i,predicted})^2 \tag{15}$$

where N represents the number of individuals in the dataset.

3 RESULTS

The following section will elaborate on the SIR and SEIR models discussed in section 2, analyzing their dynamics and presenting state phase plots. The non-demographic SIR model is additionally fitted to some historical data and the other models are examined for oscillatory behavior caused by nativity, mortality and seasonality. The time variable t in the presented models is always measured in days.

3.1 SIR without demography

The dynamics of compartment models can be analyzed by numerically integrating the ODE's of a model. The fraction of population per compartment can be presented over time and the dynamics depend on initialization and parameter settings. The initialization of population fractions per compartment is usually done by setting $S_0 = 1 - \epsilon$, $I_0 = \epsilon$ and $R_0 = 0$ and this research follows this approach, setting ϵ to 0.01. The important parameters in this model are the rate of transmission β and the recovery rate γ, a small grid of alternative values for these parameters and their dynamics is presented in Figure 1.

Four values for β (0.8, 1.5, 2.0 and 4.0) and three values for γ (0.5, 1.0 and 1.5) are represented in the columns and rows respectively. As the reproductive ratio R_0 in this model is obtained by division of the two parameters, the dynamics of the SIR model can be found for various R_0 values (denoted above each plot in Figure 1). The first plot in the second row and the first two plots in the third row present R_0 values lower than or equal to 1.0, these plots show no occurrence of an epidemic as expected. The other plots, with R_0 values larger than 1.0, all present epidemic states that have a infection peak size growing with the respective R_0 value. High R_0 values tend to infect the complete population and end with a population that is completely filled with recovered individuals.

Phase plots of the model with equal parameters as before can be found in Figure 5 in the Appendix. For the settings where $R_0 \leq 1.0$, initial conditions with I_0 close to 0 converge to a population with no infections very fast. Initial conditions with a larger fraction of infected individuals also leads to a quickly burning out disease, but takes longer as the fraction of initial infected individuals grows. R_0 values leading to an epidemic ($R_0 > 1.0$) show increasing curvature with the value of R_0, initial conditions with I_0 close to 0 also have increasing infections before burning out in this case.

3.2 Boys school epidemic

Historical data from an influenza outbreak at a boys school of 763 boys can be fitted to a SIR model without demography by least squares, as nativity and mortality is not expected to influence this population. The epidemic spreads and burns out in 15 days and is expected to have started with one infected individual at $t = 0$.

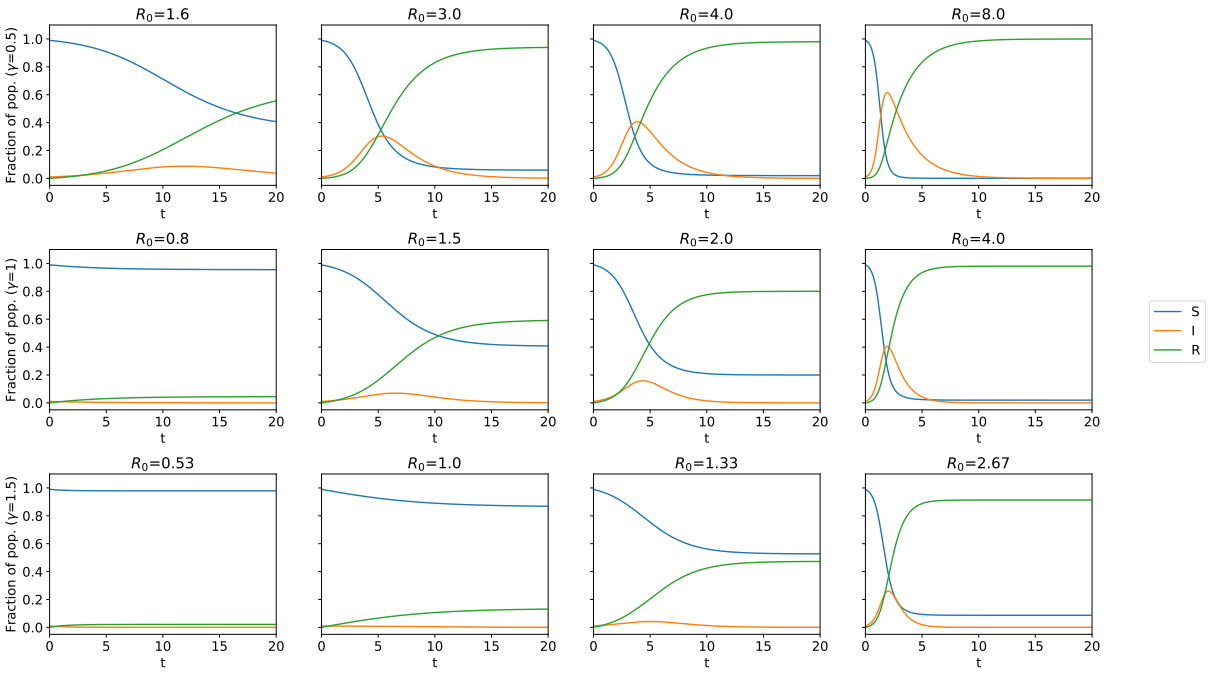

Figure 1: Dynamics of a non-demographic SIR model

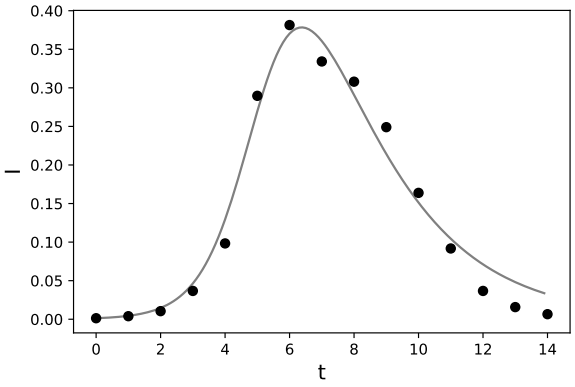

Figure 2: Infection spread at boys school

Figure 2 shows the observed data and the fitted values for $I(t)$ at the boys school, the model is fitted to the data accurately. The estimated rate of transmission for the model is 1.67 and the recovery rate is estimated at 0.45, R_0 can now be estimated using 4 and is equal to 3.72. The reproductive ratio is significantly higher than 1.0 and an epidemic is inevitable if a vaccination plan is not implemented. A specific fraction of the population will have to be vaccinated to lower the fraction of susceptibles in the population to below $\frac{1}{R_0}$, as described by Kermack & McKendrick [4]. In this case, S_0 should be smaller than $\frac{1}{3.72} = 0.27$ and at least 73 percent of the population should be vaccinated to prevent an epidemic from happening.

3.3 Including demography

A natural birth and death rate μ of 0.02 is added to the model, the equality of both rates assuring a constant population size. Dynamics of the model are again presented for the various β and γ values and some oscillatory behavior becomes visible for some of the parameter settings, the dynamics are plotted in Figure 6 in the Appendix.

As before, R_0 values smaller than or equal to 1.0 do not show the presence of an epidemic. Other parameter settings provide insight into epidemic dynamics with multiple peaks of the infection population, the susceptible and recovered population also presents some up and down movement. The initial infection peak is comparably high to the non-demographic model, but as new individuals are introduced to the susceptible population by birth multiple additional peaks can be observed. An R_0 value of approximately 2.0 appears to result in an endemic state with equal fractions of susceptible and recovered individuals. Lover values of R_0 tend to have a higher susceptible population (less infected individuals) and R_0 above 2.0 have increasing recovered population, as infection is more likely.

More thorough analysis of the oscillatory behavior in the model can be performed by Fourier transform. The three models with $\gamma = 1$ and $R_0 > 1$ are analyzed and the Fourier transforms are plotted in Figure 3. Oscillations are mostly found for frequencies below 0.1, exceeding 0.1 for the higher R_0 values. The amplitude of the transform also increases for increasing R_0.

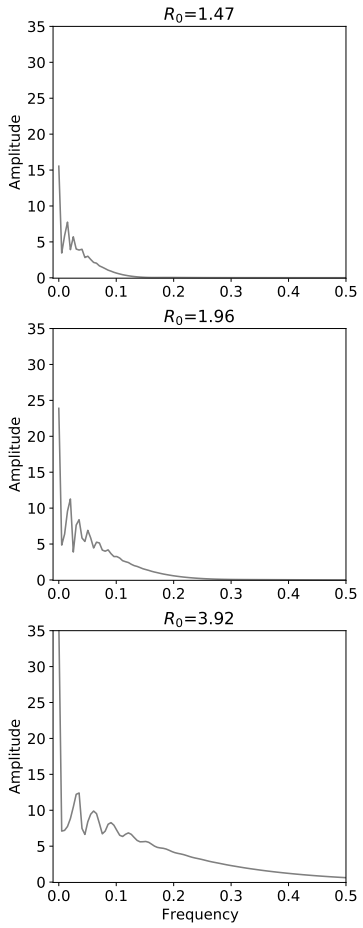

Figure 3: Fourier transform for $\beta = [1.5, 2.0, 4.0]$

In addition to natural mortality, infection-induced mortality is an often occurring phenomenon with infectious disease. The effect of the infection-induced mortality rate ρ is examined for three different values (0.5, 0.9, 1.0), note that this addition to the model will lead to a non-constant population size. For comparability and clarity, the model is evaluated for the same β values as before and γ is set to 1, model dynamics are plotted in Figure 7. The first striking change in the plots is the non-constant population size, the fractions of susceptible and recovered do not add up to 1 after burn out of the disease. The higher R_0, the lower the fraction of susceptibles in the endemic state and the higher the fraction of recovered individuals. Increasing the infection-induced mortality rate towards 1 obviously has an effect on the recovered state, as only $(1 - \rho)$ percent of the infected population moves to the recovered state. The endemic states for ρ close to 1 barely have any recovered individuals and if $\rho = 1$ there will obviously be no individuals that recover from the infection.

Phase plots of the SIR model with demography obviously show different behavior than the non-demographic model. Figure 8 presents plots with a more circular motion and can be categorized as stable spirals. Low R_0 values again have lines to the non-infected phase that are almost straight down, the higher R_0 show a more circular direction before stabilizing.

3.4 SEIR dynamics and seasonality

Section 2.2 elaborated on the addition of an extra state of being exposed to the model, the dynamics of this SEIR model can be analyzed in a parallel way to the SIR model. Apart from the addition of the exposed state, some seasonality in the form of a time varying rate of transmission is added simultaneously to investigate the effects. The natural nativity and mortality rate μ is again equal to 0.02. The infection-induced mortality rate is set to 0, as it will have similar effects to the ones presented in the previous section.

The time-varying sinusoidal rate of transmission can be clearly observed by the up and down movement of the four states in Figure 9. This model uses the definition of β_t from 14 with $c = 1$ and $d = 0.2$ so the transmission rate has a period of $10\pi \approx 31$ days. The endemic fraction of susceptible and recovered individuals behaves similar to those in the SIR model, respectively decreasing and increasing with R_0. The incubation period is set to 7 days and this is clearly visible in the delayed increase of the recovered fraction after the decrease of the susceptible population. The exposed and infected states move similarly, the exposed state obviously peaking a little earlier than the infected state.

Similar to the Fourier analysis for the SIR model, frequency plots are presented for 3 β values and $\gamma = 1$. A more periodic plot is found with a frequency peak at about $\frac{1}{30}$, which is obviously related to the period of the rate of transmission. Phase plots of the seasonal SEIR model are presented in Figure 10. These plots have similar behavior with relation to R_0 as in the SIR model, the circular motion is a little less smooth because of the time-varying rate of transmission.

4 CONCLUSION

In the year 2020, providing informative alternatives to epidemic modelling is of major importance because of the current novel coronavirus pandemic. This research presents alternative compartment models to analyze the dynamics of the spread of infectious disease. Initial SIR models without demography are subsequently expanded by nativity and mortality rates, eventually also introducing some infection-induced mortality which is a common phenomenon. The three-compartment SIR model is thereafter expanded to include a state of being exposed to the infection, this model also presents some effects of a seasonal changing rate of transmission of the disease.

The non-demographic SIR models allow for straightforward observation of the occurrence of an epidemic or not, the endemic state is reached reasonably fast. The effect of various R_0 values are observed and fitting some data to the model seems to work well. The epidemic of influenza at a boys school, of which historical infection data is used in this research, could have been prevented by vaccinating approximately 73 percent of the boys. The inclusion of nativity and mortality rates introduces oscillatory behavior in the dynamics, the endemic state is reached after a longer period of time. Infection-induced mortality causes the population to become non-constant and increasing the infection-induced mortality rate towards 1 will lead to an endemic state with no recovered individuals as expected. Seasonality in the SEIR model provides a more periodic process, the Fourier analysis of this model clearly shows the periodicity of the model. The incubation period of 7 days provides an obvious delay in the growth of the recovered fraction, but the other dynamics of the SEIR model are similar to the SIR dynamics.

Further research of interest would probably have to consist of a more thorough analysis of fitting a larger observed data to the presented models. Historical data should be fitted to the appropriate model, based on medical knowledge about the disease. The SIR and SEIR models in this research could also be extended to contain a state of passive immunity (MSEIR), return to the susceptible population (SEIRS) or a combination of both (MSEIRS).

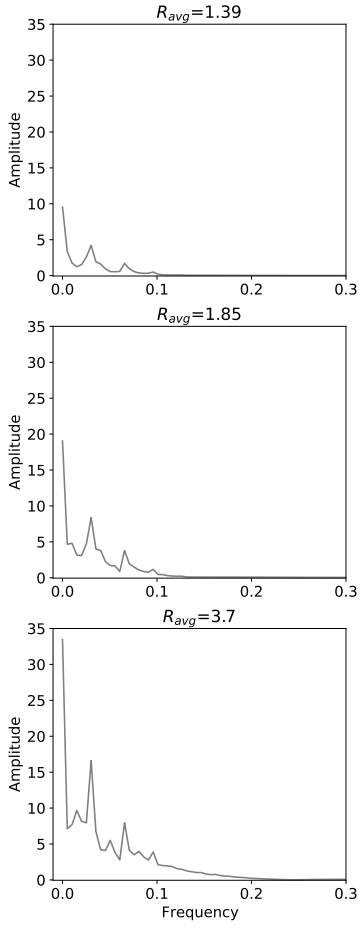

Figure 4: Fourier transform for $\beta = [1.5, 2.0, 4.0]$

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APPENDIX (NEXT PAGE)

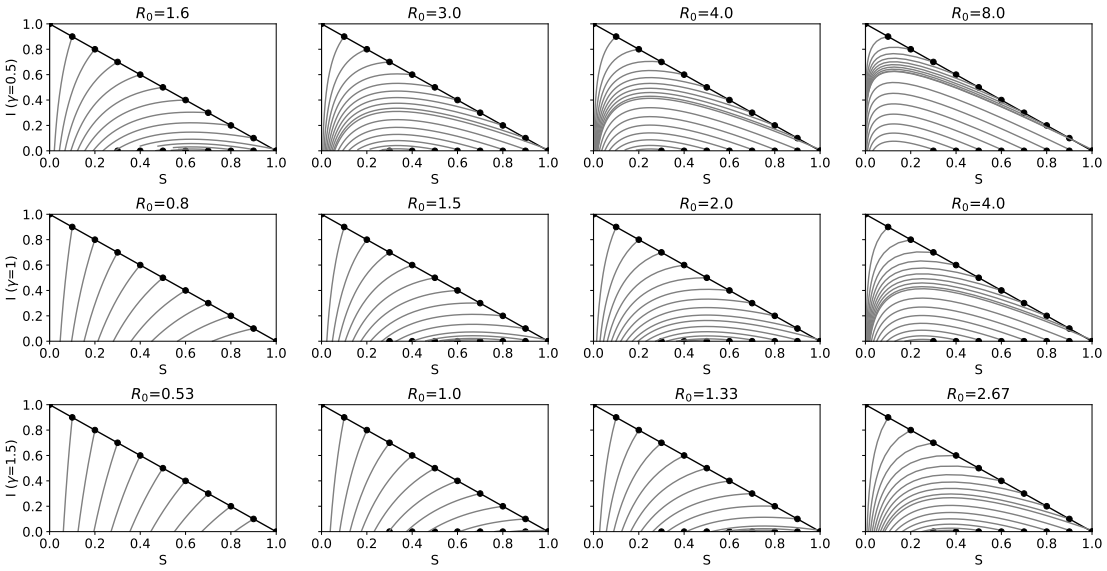

Figure 5: Phase plots of a non-demographic SIR model

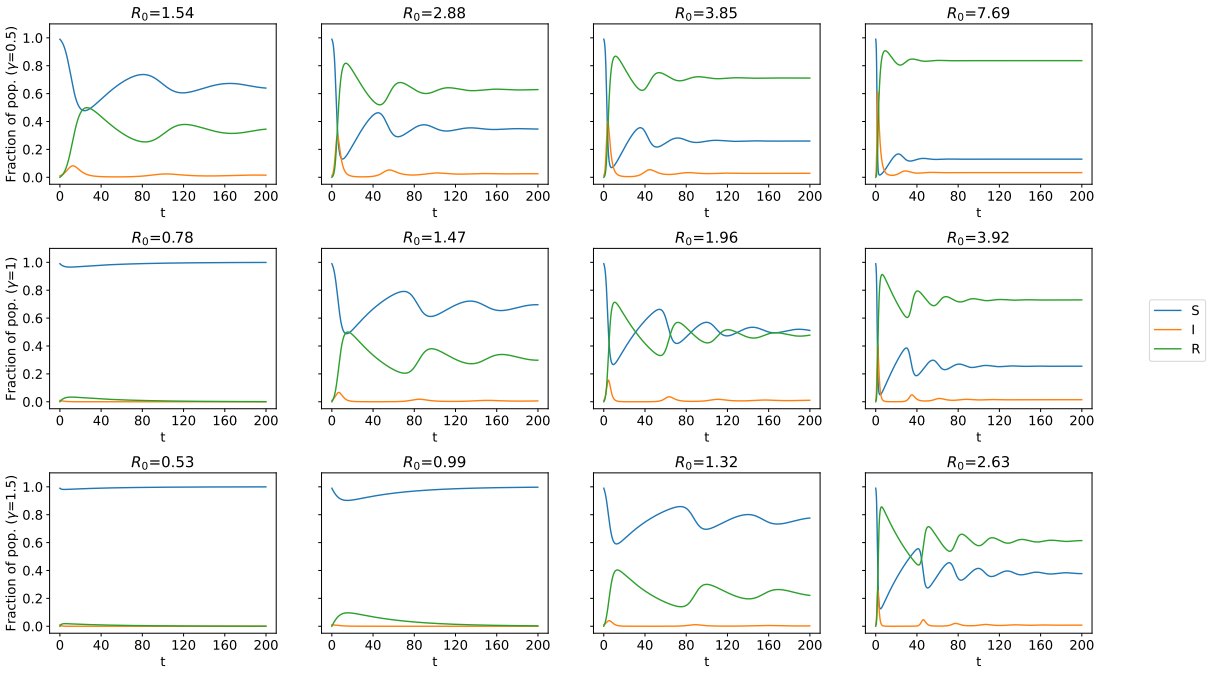

Figure 6: Dynamics of a SIR model with demographics

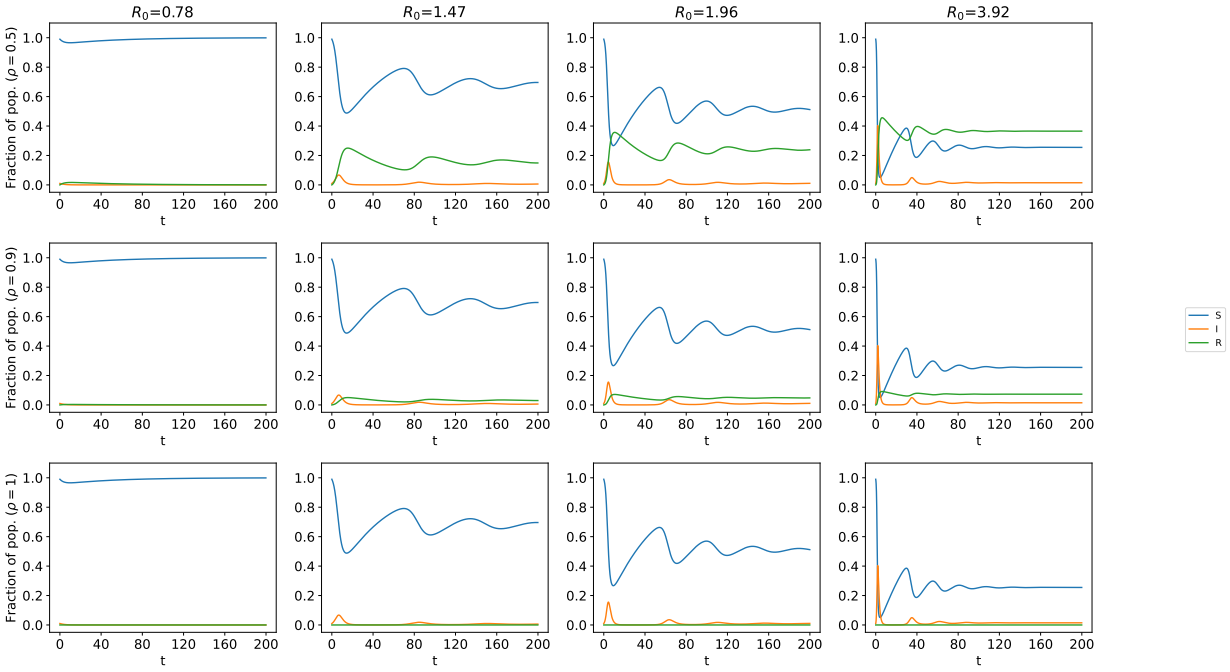

Figure 7: Dynamics of a SIR model with demographics and infection-induced mortality

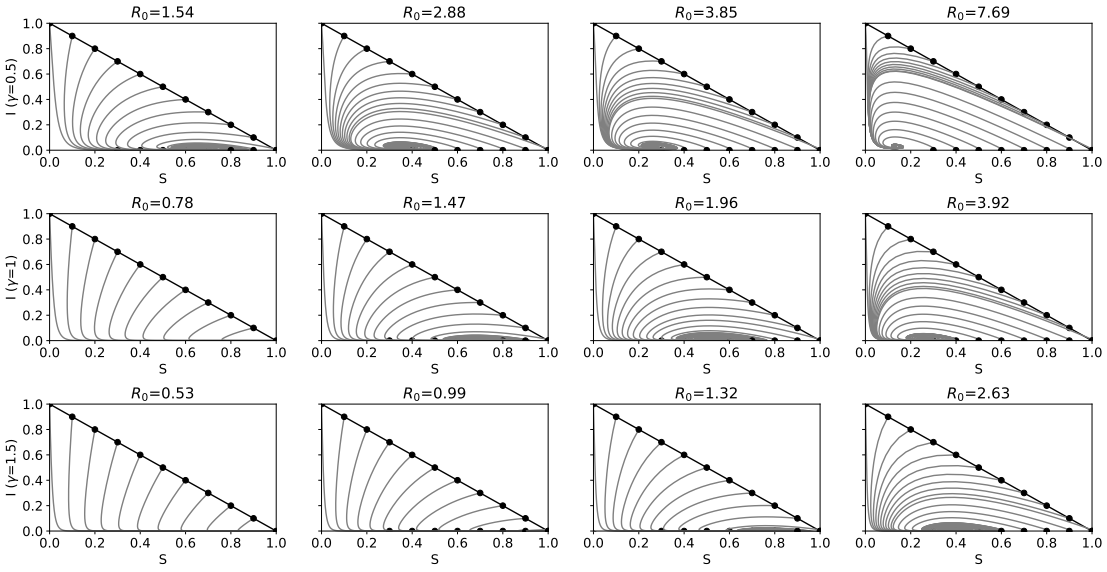

Figure 8: Phase plots of a SIR model with demographics

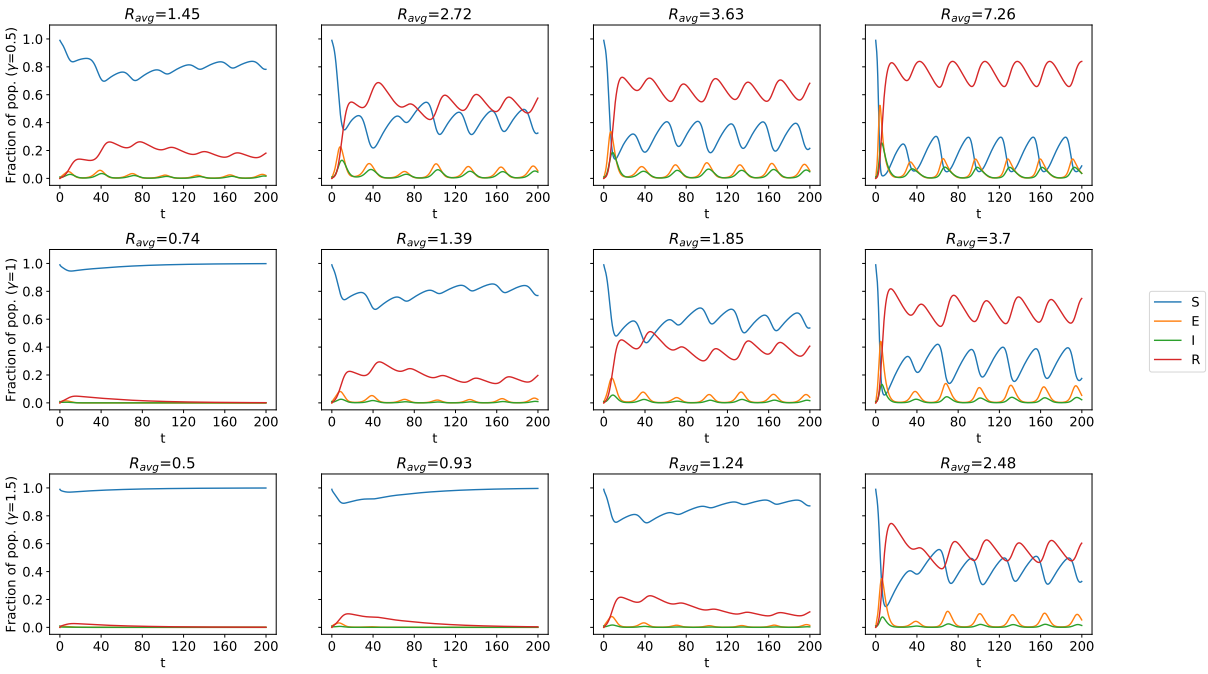

Figure 9: Dynamics of a SEIR model with demographics and seasonal effects

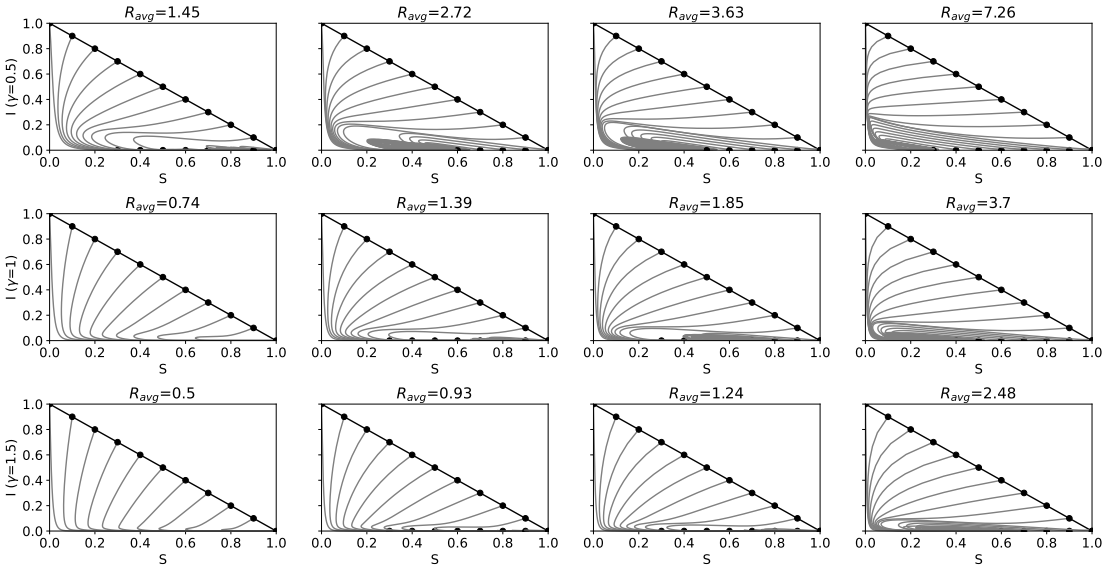

Figure 10: Phase plots of a SEIR model with demographics and seasonal effects