

Practical Compartmental Models for Infectious Disease Dynamics in Closed Populations

Avery Yu

Oak Park High School, Oak Park, CA

Abstract

Compartmental models are key tools for understanding infectious disease dynamics. This article introduces several classical and extended compartmental models, including the Susceptible–Infected–Recovered (SIR) model, the Susceptible–Infected–Deceased–Susceptible (SIDS) model where immunity wanes, the Susceptible–Infected–Deceased–Immune (SIDM) model where immunity does not wane, and the Susceptible–Infected–Recovered–Vaccinated (SIRV) model incorporating vaccination dynamics. For each model, we present the governing differential and difference equations, simulate typical epidemic trajectories, and demonstrate how to estimate model parameters for synthetic incidence data.

Keywords: compartmental model, susceptible, vaccinated, infected, removed, recovered, deceased, difference equation

1 Introduction

1.1 Background

The mathematical study of infectious diseases has a long and rich history, beginning well before the formalization of modern compartmental models. Early attempts to quantify the spread of disease can be traced back to John Graunt (1759) [1], who analyzed mortality data in *Bills of Mortality* to describe population-level patterns of death and disease—an early precursor to epidemiological statistics. Later, Edmund Halley (1693) [2] constructed

life tables that linked mortality rates to population dynamics, establishing a foundation for later demographic modeling.

A major step toward mathematical epidemiology came with Daniel Bernoulli's 1760 analysis of smallpox [3], [4]. Bernoulli developed one of the first compartmental frameworks to estimate the impact of smallpox inoculation on life expectancy in a population. His model divided the population into compartments representing susceptible and immune individuals and used differential equations to estimate mortality under various intervention strategies. This work remains one of the earliest examples of quantitative reasoning about infectious disease dynamics.

The next major advancement occurred in the early 20th century with the work of William Ogilvy Kermack and Anderson Gray McKendrick, who formalized the theory of epidemic processes in a series of seminal papers between 1927 and 1933 [5], [6], [7]. Compartmental models such as those of Kermack and McKendrick remain foundational in infectious disease modeling. They work by partitioning a population into classes (compartments) according to disease status—such as susceptible, infected, and recovered—and specifying the rates at which individuals transition between these states. Over the past century, numerous extensions and refinements have been proposed to accommodate features such as latency periods, demographic effects, spatial structure, stochasticity, and vaccination.

This paper focuses on several of these modifications and illustrates their application to modern epidemiological contexts.

1.2 Literature Review

Compartmental models have been used in the context of a wide range of cases. The classic SIR model was used by Abueldahab and Mutombo to showcase the effect of anti-retroviral treatment (ART) in the HIV/AIDS epidemic in Khartoum, Sudan [10]. To find a model that is both practical and accurate has been a chief goal of many papers. In one such paper, Adeyemo et al. model Co-Infection of Tuberculosis and HIV in South Africa using a model with nine compartments, predicting epidemiological trends 30 years in the future and gaining valuable insight [11]. The COVID-19 pandemic (2020-2023), as expected, has greatly increased renewed interest in mathematical epidemiology, expanding the compartmental models to incorporate a wide spectrum of possibilities ranging from social distancing to vaccines to migration. Cooper et al. predicted the spread of COVID-19 three months into the future from data gathered from January to June 2020. Notably, their model builds on the traditional SIR model by accommodating surges in the susceptible population in-

stead of assuming a constant N total population, allowing this model to explain the large increase in infections around the world at the beginning of the pandemic. The researchers proceeded by modeling the disease trends in different countries, namely China, South Korea, India, Australia, the US, and Italy, and hypothesized possible explanations for these trends, as well as disease control policies that could potentially be impactful. It was found that, as seen in the trend in South Korea, the number of infections can drop dramatically if the government acts quickly to control the virus—otherwise infection numbers spike [12]. During the pandemic, the steps governments have taken to limit the spread of coronavirus have come under heavy controversy and debate—many papers address this issue. Dong et al. use a SIR model with vaccination and time delay (to simulate the incubation period) to show that vaccination and social distancing are valid strategies to effectively control the infectious disease [13]. Another interesting factor to look at when simulating strategies to suppress the spread of the virus, is the basic reproduction rate of a virus. In “A mathematical model for COVID-19 considering waning immunity, vaccination and control measures,” researchers modeled the coronavirus epidemic in different countries and looked at the estimated basic reproduction rate at different time points. One of their major findings was that using a vaccine of low efficacy with a highly susceptible population does not suppress the spread of a disease due to a lack of herd immunity and re-infection. However, a high-efficacy vaccine dramatically reduced infection rates even when only given to a portion of the population. The data instead shows that long-term confinement and extensive testing show more promising results [14].

2 The Classical Susceptible–Infectious–Removed (SIR) Model

2.1 Theory

The classical Susceptible–Infectious–Removed (SIR) model is given by the following system of ordinary differential equations, where the total population size N is assumed constant

so that $S(t) + I(t) + R(t) = N$ for all $t \geq 0$:

$$\begin{cases} S'(t) = -\frac{\beta S(t)I(t)}{N}, \\ I'(t) = \frac{\beta S(t)I(t)}{N} - \gamma I(t), \\ R'(t) = \gamma I(t). \end{cases}$$

Here, $S(t)$, $I(t)$, and $R(t)$ denote, respectively, the number of susceptible individuals (those who have not yet contracted the disease), infectious individuals, and removed individuals (those who have either recovered with immunity or died, and thus are no longer capable of transmitting or acquiring the infection). The parameters $\beta > 0$ and $\gamma > 0$ represent the transmission rate and the removal rate, respectively. [15]

In this model, it is assumed that in an infinitesimally small interval $[t, t+dt]$, a susceptible individual has time to meet only one other individual in the population. The probability that this other individual is from the infectious group is $I(t)/N$, and the infection is transmitted with rate β ; thus, on average, $\frac{\beta S(t)I(t)}{N}$ susceptible individuals will become infected.

The model assumes a closed population of fixed size N , i.e., no births, deaths (apart from those due to the disease), immigration, or emigration occur. This assumption is one of the main limitations of the basic SIR model, as such closed populations rarely exist in real-world epidemiological contexts.

Since the above system generally lacks a closed-form analytical solution, it is often approximated numerically by discretizing time. Let $j = 0, 1, 2, \dots$ index discrete time steps with increment $\Delta t = 1$. Then the corresponding difference equations for the discrete-time SIR model are given by:

$$\begin{cases} S(j+1) = S(j) - \beta \frac{S(j)I(j)}{N}, \\ I(j+1) = I(j) + \beta \frac{S(j)I(j)}{N} - \gamma I(j), \\ R(j+1) = R(j) + \gamma I(j). \end{cases}$$

These recursive relations allow for straightforward numerical implementation and graphical visualization of epidemic dynamics over discrete time.

Next, we explain how parameter estimation is performed in this model. The model parameters β and γ can be estimated from discrete-time data using either the *method of moments* or the *regression approach*.

(i) *Estimation of β .* From the first difference equation,

$$S(j+1) - S(j) = -\beta \frac{S(j)I(j)}{N},$$

we can write

$$\frac{S(j+1) - S(j)}{S(j)I(j)} = \frac{\Delta S(j)}{S(j)I(j)} = -\frac{\beta}{N}.$$

Hence, an unbiased method-of-moments estimator of β is given by

$$\hat{\beta}_{\text{MM}} = -\frac{N}{n} \sum_{j=1}^n \frac{\Delta S(j)}{S(j)I(j)}.$$

Alternatively, by rearranging the second equation, we get

$$\frac{I(j+1) - I(j)}{I(j)} = \frac{\Delta I(j)}{I(j)} = \frac{\beta}{N} S(j) - \gamma.$$

One may estimate β using linear regression, where the slope of the regression line of $\frac{\Delta I(j)}{I(j)}$ on $S(j)$ corresponds to $\frac{\hat{\beta}_R}{N}$.

(ii) *Estimation of γ .* From the third equation,

$$R(j+1) - R(j) = \gamma I(j),$$

we obtain

$$\frac{R(j+1) - R(j)}{I(j)} = \frac{\Delta R(j)}{I(j)} = \gamma.$$

Thus, a method-of-moments estimator for γ is

$$\hat{\gamma}_{\text{MM}} = \frac{1}{n} \sum_{j=1}^n \frac{\Delta R(j)}{I(j)}.$$

Alternatively, γ can be estimated by the method of linear regression. When $\frac{\Delta I(j)}{I(j)}$ is regressed on $S(j)$, the intercept of the fitted line is equal to $-\hat{\gamma}_R$.

2.2 Applications

Using the SIR model described above, we simulate an epidemic in a closed population of 350 individuals, where initially only one person was infected. The simulation is run for 40 days with parameters $\beta = 0.7$ and $\gamma = 0.2$ (see Figure 1 below). We then estimate the parameters β and γ using the two previously described approaches: the method of moments and regression analysis.

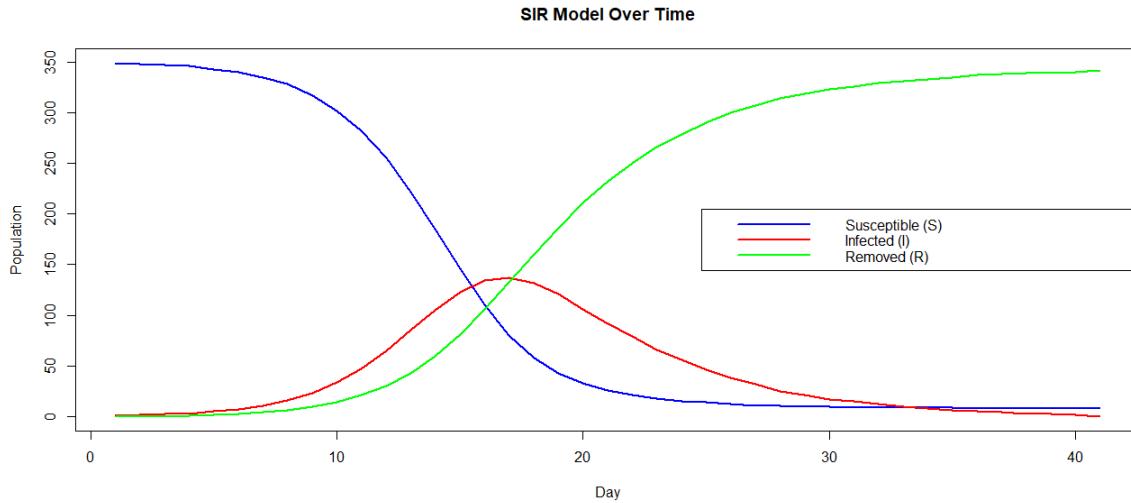


Figure 1. Simulated SIR model.

The parameters used for simulation are as follows:

$$\beta = 0.7, \text{ and } \gamma = 0.2.$$

The parameter estimates obtained using the method of moments are as follows:

$$\hat{\beta}_{MM} = 0.707701, \text{ and } \hat{\gamma}_{MM} = 0.218209.$$

Using the regression approach, the estimates are as follows:

$$\hat{\beta}_R = 0.766033, \text{ and } \hat{\gamma}_R = 0.241123.$$

In this simulation, the number of infected individuals peaks at day 18, reaching 137 cases. By the end of the 40-day period, nearly all individuals have recovered, and only a small fraction of the population remains uninfected. The estimated value of β around

0.7 indicates that when an infected and a susceptible individual come into contact, there is approximately a 70% probability that the susceptible person becomes infected. Consequently, it is not surprising that out of 350 individuals, 342 were ultimately infected by the end of the simulation.

3 Susceptible–Infectious–Deceased–Susceptible (SIDS) Model

3.1 Theory

Some adjustments can be made to allow more discretion in the "removed" category. After infection with a disease, a person may return to the susceptible category or proceed to the deceased category. In this model, those who return to the susceptible category have the same chance of becoming infected with the disease again as those who have never been infected before. Therefore, they are, for our purposes, indistinguishable. The following difference equations may be written:

$$\begin{cases} S(j+1) = S(j) - \beta \frac{S(j)I(j)}{N} + \alpha I(j), \\ I(j+1) = I(j) + \beta \frac{S(j)I(j)}{N} - \gamma I(j) - \alpha I(j), \\ D(j+1) = D(j) + \gamma I(j). \end{cases}$$

Here β is the rate of infection transmission, γ is the rate of removal due to death, and α is the rate at which infected individuals recover and return to the susceptible category.

To estimate the parameters of this model, we rewrite the above equations in a more convenient form:

$$\begin{cases} \frac{\Delta S(j)}{I(j)} = -\frac{\beta}{N} S(j) + \alpha, \\ \frac{\Delta I(j)}{I(j)} = \frac{\beta}{N} S(j) - (\gamma + \alpha), \\ \frac{\Delta D(j)}{I(j)} = \gamma. \end{cases}$$

From the first equation, by regressing $\frac{\Delta S(j)}{I(j)}$ on $S(j)$, we can estimate $-\beta/N$ as the fitted slope, and α is the fitted intercept. Thus,

$$\hat{\beta}_{R,1} = -N \cdot \widehat{slope}_1, \text{ and } \hat{\alpha}_{R,1} = \widehat{intercept}_1.$$

Similarly, from the second equation, regressing $\frac{\Delta I(j)}{I(j)}$ on $S(j)$ produces another set of regression estimates:

$$\hat{\beta}_{R,2} = N \cdot \widehat{slope}_2, \text{ and } (\widehat{\gamma} + \hat{\alpha})_{R,2} = -\widehat{intercept}_2.$$

The parameter γ can be estimated from the third equation using the method of moments,

$$\hat{\gamma}_{MM} = \frac{1}{n} \sum_{j=1}^n \frac{\Delta D(j)}{I(j)}.$$

Thus, the parameter α can be estimated by $\hat{\alpha}_{R,1} = \widehat{intercept}_1$, or by $\hat{\alpha}_{R,2} = -\widehat{intercept}_2 - \hat{\gamma}_{MM}$. The parameter γ , in turn, can be estimated by $\hat{\gamma}_{MM}$ or $\hat{\gamma}_{R,2} = -\widehat{intercept}_2 - \hat{\alpha}_{R,1}$.

3.2 Applications

Here we simulate the same situation as before, with 350 people (of which one is infected) over the course of 40 days, but with the SIRS model, where some people return to the susceptible category after becoming infected.

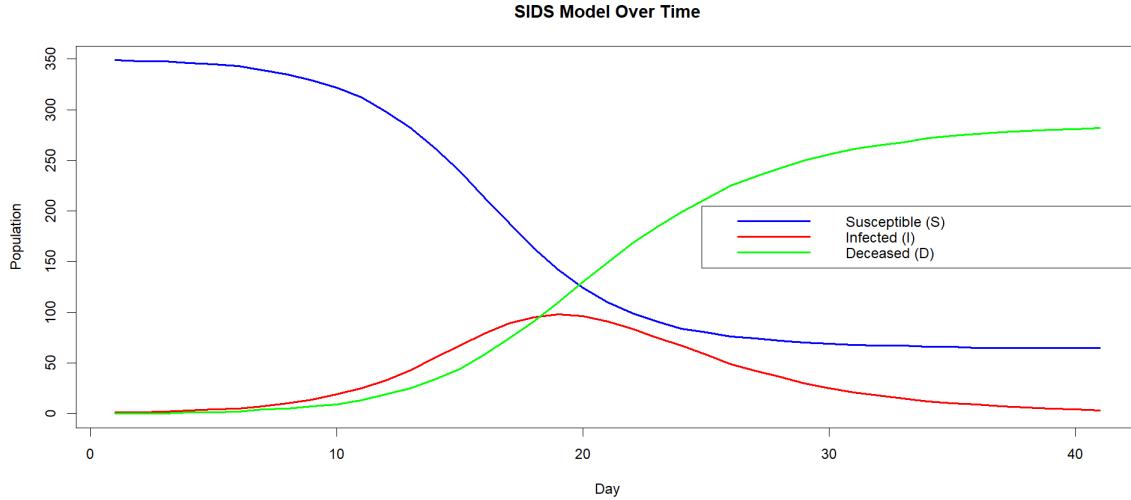


Figure 2. Simulated SIDS model.

The parameters used for simulation are as follows:

$$\beta = 0.7, \alpha = 0.1, \text{ and } \gamma = 0.2.$$

The estimated parameters using the regression method of the first and second equations (denoted accordingly) are as follows:

$$\hat{\beta}_{R,1} = 0.7130422, \quad \hat{\alpha}_{R,1} = 0.1080514$$

$$\hat{\beta}_{R,2} = 0.7265972, \quad \hat{\alpha}_{R,2} = 0.1148145, \quad \text{and} \quad \hat{\gamma}_{R,2} = 0.2030058.$$

The estimated gamma parameter using the method of moments is:

$$\hat{\gamma}_{MM} = 0.1962426.$$

The peak incidence (the time at which the most people are infected) here again occurs at day 19. However, there are only 98 cases at peak incidence. At 40 days, the infection has almost died out, and 65 people remain in the susceptible category. This is 57 more people than in the classic SIR model, which does not allow infected individuals to return to the susceptible category. Recall that in this model, individuals in the susceptible category do not necessarily have no history of the disease; they could have returned to the susceptible category after becoming infected.

4 Susceptible–Infectious–Deceased–Immune (SIDM) Model

4.1 Theory

Another adjustment can be made to allow a portion of the infected population to become immune after recovery. This requires the addition of a new compartment, the immune class, $M(t)$. Individuals in this compartment are permanently immune and cannot be infected again, nor can they transmit the disease to others. The resulting model, referred to as the Susceptible–Infectious–Deceased–Immune (SIDM) model, is governed by the following system of difference equations:

$$\begin{cases} S(j+1) = S(j) - \beta \frac{S(j)I(j)}{N} + \alpha I(j), \\ I(j+1) = I(j) + \beta \frac{S(j)I(j)}{N} - \gamma I(j) - \alpha I(j) - \omega I(j), \\ D(j+1) = D(j) + \gamma I(j), \\ M(j+1) = M(j) + \omega I(j), \end{cases}$$

where β is the rate of infection transmission, γ is the rate of removal due to death, α is the rate at which infected individuals recover and return to the susceptible category, and ω is the rate at which infected individuals become permanently immune.

To derive reasonable parameter estimators, we rewrite these equations as:

$$\begin{cases} \frac{\Delta S(j)}{I(j)} = -\frac{\beta}{N} S(j) + \alpha, \\ \frac{\Delta I(j)}{I(j)} = \frac{\beta}{N} S(j) - (\gamma + \alpha + \omega), \\ \frac{\Delta D(j)}{I(j)} = \gamma, \\ \frac{\Delta M(j)}{I(j)} = \omega. \end{cases}$$

From here, the following method-of-moments estimators for γ and ω are obtained:

$$\hat{\gamma}_{MM} = \frac{1}{n} \sum_{j=1}^n \frac{\Delta D(j)}{I(j)}, \quad \text{and} \quad \hat{\omega}_{MM} = \frac{1}{n} \sum_{j=1}^n \frac{\Delta M(j)}{I(j)}.$$

The parameter β can be estimated by regressing either $\frac{\Delta S(j)}{I(j)}$ on $S(j)$ or $\frac{\Delta I(j)}{I(j)}$ on $S(j)$.

The estimators are:

$$\hat{\beta}_{R,1} = -N \cdot \widehat{slope}_1, \quad \text{and} \quad \hat{\beta}_{R,2} = N \cdot \widehat{slope}_2.$$

The parameter α can be evaluated from the first regression as $\hat{\alpha}_{R,1} = \widehat{intercept}_1$, or from the second regression as

$$\hat{\alpha}_{R,2} = -\widehat{intercept}_2 - \hat{\gamma}_{MM} - \hat{\omega}_{MM}.$$

In addition, γ can be estimated by $\hat{\gamma}_{R,2} = -\widehat{intercept}_2 - \hat{\alpha}_{R,1} - \hat{\omega}_{MM}$. Likewise, ω can be estimated by $\hat{\omega}_{R,2} = -\widehat{intercept}_2 - \hat{\alpha}_{R,1} - \hat{\gamma}_{MM}$.

4.2 Applications

Next, we use the SIDM model in the same context:

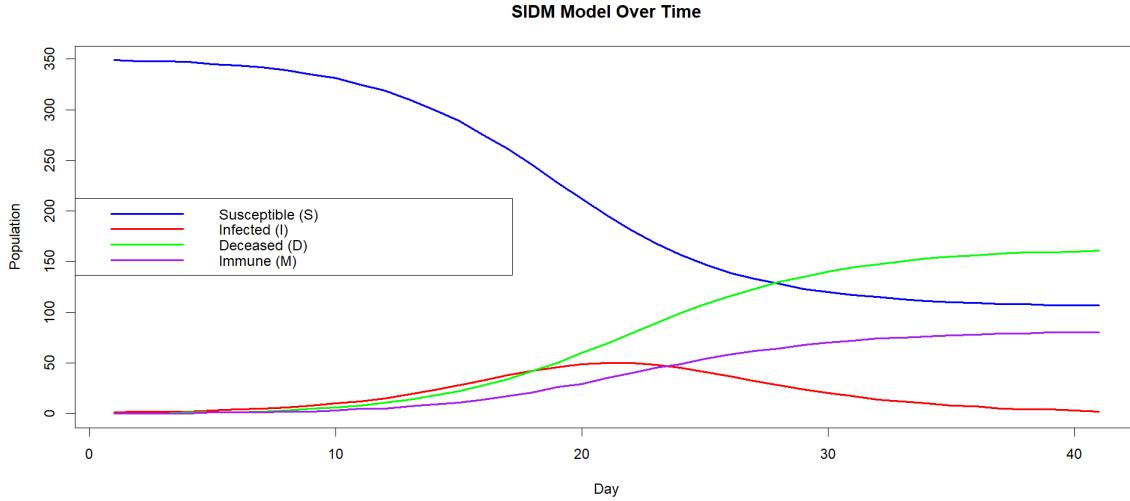


Figure 3. Simulated SIDM model.

The parameters used for simulation are as follows:

$$\beta = 0.7, \quad \alpha = 0.1, \quad \text{and} \quad \gamma = 0.2.$$

The estimated parameters using the method of moments are as follows:

$$\hat{\gamma}_{MM} = 0.1918512, \quad \text{and} \quad \hat{\omega}_{MM} = 0.09559131$$

The estimated parameters using the regression method on the first and second equations (denoted respectively) are as follows:

$$\hat{\beta}_{R1} = 0.7000437, \quad \hat{\alpha}_{R1} = 0.1081646,$$

$$\hat{\beta}_{R2} = 0.7394717, \quad \hat{\alpha}_{R2} = 0.1329169, \quad \hat{\gamma}_{R2} = 0.2166035, \quad \text{and} \quad \hat{\omega}_{R2} = 0.1207436.$$

This graph visually looks vastly different than the two prior graphs. Indeed, there are many significant differences in allowing infected people to join an "immune" category. We can think of this as the same effect as if in a previous model, γ was increased, except here we can visibly see the difference. The peak incidence here is during days 21 and 22. Only 50 people were infected at this peak incidence. At the end of the simulation, 107 people remain susceptible. However, like the previous model, these people could have been infected by the disease but returned to the susceptible category. After 40 days, 80 people are immune to the disease. In the "deceased" category there are 161 people.

5 Susceptible-Infectious-Removed-Vaccinated (SIRV) Model

5.1 Theory

If we assume that vaccinated individuals cannot become infected, the susceptible-infectious-removed-vaccinated (SIRV) model is governed by the following difference equations:

$$\begin{cases} S(j+1) = S(j) - \frac{\beta S(j)I(j)}{N} - \nu S(j), \\ I(j+1) = I(j) + \frac{\beta S(j)I(j)}{N} - \gamma I(j), \\ R(j+1) = R(j) + \gamma I(j), \\ V(j+1) = V(j) + \nu S(j). \end{cases}$$

Here ν is the constant rate of vaccination of the susceptible group, and $V(t)$ is the number of people who have become vaccinated and thus immune to infection.

To derive parameter estimators, we write the difference equations in a more convenient form:

$$\begin{cases} \frac{\Delta S(j)}{S(j)} = -\frac{\beta}{N} I(j) - \nu, \\ \frac{\Delta I(j)}{I(j)} = \frac{\beta}{N} S(j) - \gamma, \\ \frac{\Delta R(j)}{I(j)} = \gamma, \\ \frac{\Delta V(j)}{S(j)} = \nu. \end{cases}$$

From here, the parameter β yields two regression estimators, when $\frac{\Delta S(j)}{S(j)}$ is regressed on $I(j)$, and when $\frac{\Delta I(j)}{I(j)}$ is regressed on $S(j)$:

$$\hat{\beta}_{R,1} = -N \cdot \widehat{\text{slope}}_{R,1}, \quad \text{and} \quad \hat{\beta}_{R,2} = N \cdot \widehat{\text{slope}}_{R,2}.$$

The parameters γ and ν may be estimated using either the method of moments or linear regression. The formulas are:

$$\hat{\gamma}_{MM} = -\frac{1}{n} \sum_{j=1}^n \frac{\Delta R(j)}{I(j)}, \quad \text{or} \quad \hat{\gamma}_R = -\widehat{\text{intercept}}_2,$$

and

$$\hat{\nu}_{MM} = \frac{1}{n} \sum_{j=1}^n \frac{\Delta V(j)}{S(j)}, \text{ or } \hat{\nu}_R = -\widehat{\text{intercept}}_1.$$

5.2 Applications

Using the same context as previous applications, we apply the SIRV model with the assumption that all vaccinated individuals become immune to the disease.

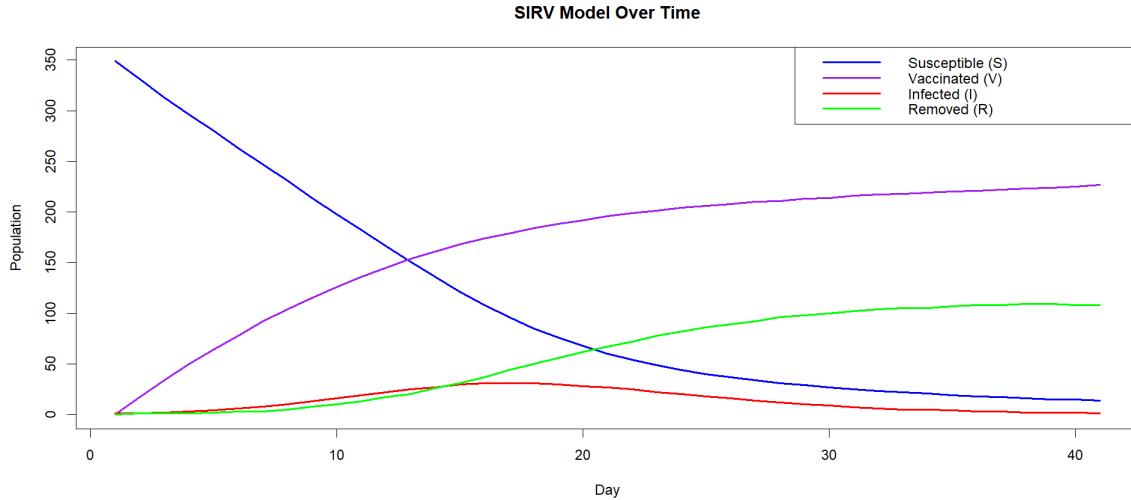


Figure 4. Simulated SIRV model.

The parameters used for simulation are as follows:

$$\beta = 0.7, \ v = 0.05, \ \text{and} \ \gamma = 0.2.$$

The estimated parameters using the method of moments are as follows:

$$\hat{\beta}_{MM} = 0.6641301, \ \hat{\gamma}_{MM} = 0.1852429, \ \text{and} \ \hat{v}_{MM} = 0.052853.$$

The estimated parameters using the regression method are as follows:

$$\hat{\beta}_{R1} = 0.7107359, \ \hat{\beta}_{R2} = 0.723759, \ \hat{\gamma}_R = 0.2036662, \ \text{and} \ \hat{v}_R = 0.04901355.$$

This model has a much more suppressed infection spread than any of the previous models. The peak incidence occurs on days 17 and 18 and consisted of only 31 cases. At the end of 40 days, 14 susceptible and 227 vaccinated people remain: a total of 241 people who have never been infected with the disease.

6 Conclusion and Discussion

6.1 Summary

Compartmental modeling is extremely useful as a tool in epidemiology. It allows epidemiologists to predict epidemic trajectories according to many different and easily alterable variables. The key advantage, as highlighted in this paper, of compartmental modeling is the simple manipulation of compartments and variables that allows coverage of almost all situations. From there, analysis on techniques to ameliorate epidemics can be performed. Through this paper, compartmental modeling is shown to be a tool in which adding on these additional details and variations is not a problem. Simply adding compartments, extra variables, and estimating accordingly is enough to cover almost all variations that exist with compartmental models. In addition, applying these models to real-world or simulated data is equally simple. Using the methods outlined in the paper, one is able to estimate the parameters of the model in two ways per model: the method of moments and the regression method.

6.2 Future Research Direction

While this paper explores four useful and practical compartmental models in depth, there are many other nuances that exist that remain unexplored. Of these, notable are the time latency after a vaccination (immunity or partial immunity can take a few weeks to develop after a vaccination), social distancing, and migrating populations.

Supplemental Materials

The simulated datasets and graphs, as well as the code used for the simulation, graphing, and variable estimation, are in the link below.

<https://github.com/yoyojiarun-rgb//Practical-Compartmental-Models-for-Infectious-Disease-Dynamics-in-Closed-Populations.git>

Acknowledgments

I would like to thank Dr. Olga Korosteleva, Professor of Statistics at California State University, Long Beach, for her invaluable guidance and support throughout this research. I would also like to thank Dr. Oleg Gleizer and the UCLA Olga Radko Endowed Math Circle for providing me a vibrant math education. In addition, I would like to thank my math teachers at Oak Park High School: Mrs. Lac, Mr. Weintraub, and Ms. Lory, as well as my middle school math teacher, Mr. Large, for teaching me so much and expanding my growth and interest in math! Last but not least, I would like to thank my family for supporting me and always encouraging me to challenge myself!

References

- [1] Postlethwayt, J., Graunt, J., Petty, W., Morris, C., and W. Heberden. (1759). *Collection of Yearly Bills of Mortality, from 1657 to 1758 Inclusive*. A. Miller Publisher.
- [2] Halley, E. (1693). An estimate of the degrees of the mortality of mankind, drawn from curious tables of the births and funerals at the city of Breslaw; with an attempt to ascertain the price of annuities upon lives. *Philosophical Transactions of the Royal Society of London*, 17: 596–610.
- [3] Bernoulli, D. (1760). Réflexions sur les avantages de l'inoculation. *Mercure de France*, 173–190.
- [4] Bernoulli D. (1766). Essai d'une nouvelle analyse de la mortalité causée par la petite vérole et des avantages de l'inoculation pour la prévenir. *Mémoires de l'Académie royale des sciences*, 1–45.
- [5] Kermack, W. O. and A. G. McKendrick. (1927). Contributions to the mathematical theory of epidemics, part I. *Proceedings of the Royal Society of Edinburgh. Series A*, 115: 700-721.
- [6] Kermack, W. O. and A. G. McKendrick. (1932). Contributions to the mathematical theory of epidemics. II - The problem of endemicity. *Proceedings of the Royal Society of Edinburgh. Series A*, 138: 55-83.

- [7] Kermack, W. O. and A. G. McKendrick. (1933). Contributions to the mathematical theory of epidemics. III - Further studies of the problem of endemicity. *Proceedings of the Royal Society of London. Series A*, 141 (843): 94-122.
- [8] Brauer, F. (2017). Mathematical epidemiology: Past, present, and future. *Infectious Disease Modelling*, 2(2), 113–127.
- [9] Macdonald, G. (1957). *The epidemiology and control of malaria*. Oxford University Press.
- [10] Abueldaab, S. M. E., and F. K. Mutombo. (2021). SIR model and HIV/AIDS in Khartoum. *Open Access Library Journal*, 8, e7334.
- [11] Adeyemo, S., Sangotola, A., and O. Korosteleva. (2023). Modeling transmission dynamics of tuberculosis-HIV co-infection in South Africa. *Epidemiologia*, 4(4), 408–419.
- [12] Cooper, I., Argha, M., and C. Antonopoulos. (2020). A SIR model assumption for the spread of COVID-19 in different communities. *Chaos, Solitons & Fractals*, 139, 110057.
- [13] Dong, S. et al. (2023). Application of a time-delay SIR model with vaccination in COVID-19 prediction and its optimal control strategy. *Nonlinear Dynamics*, 111, 10677–10692.
- [14] Ghosh, S.K., and S. Ghosh. (2023). A mathematical model for COVID-19 considering waning immunity, vaccination and control measures. *Scientific Reports*, 13, 3610.
- [15] Hen, N. et al. (2012). *Modeling Infectious Disease Parameters Based on Serological and Social Contact Data*, Spring.