Mathematical Modeling of Epidemics: SI and SIS Models Analysis

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Introduction

Mathematical modeling of infectious diseases has been fundamental to epidemiology since Kermack and McKendrick's groundbreaking work in 1927, which introduced compartmental models to analyze disease spread. This approach gained more attention during the COVID-19 pandemic, where mathematical models became crucial tools for predicting disease trajectories and informing public health policies worldwide.

The compartmental models we analyze in this report—the SI (Susceptible-Infected) and SIS (Susceptible-Infected-Susceptible) models—represent the foundational building blocks of modern epidemiological modeling. The SI model captures the dynamics of permanent infections like HIV in the pre-treatment era, while the SIS model extends this framework to include recovery without immunity, making it relevant for diseases like the common cold and certain bacterial infections where previous exposure doesn't confer lasting protection.

These models have applications beyond human epidemiology, describing phenomena such as computer virus propagation, and information spread in social networks. Through computational implementation and mathematical analysis, we explore how these models capture different aspects of disease spread and examine the impact of various parameters on epidemic dynamics, providing insights into both the capabilities and limitations of simple compartmental models.

This report details the implementation and analysis of two epidemiological models: the SI (Susceptible-Infected) and SIS (Susceptible-Infected-Susceptible) models. The simulations were performed in Python using discrete-time difference equations. Key parameters include the transmission rate β, recovery rate γ, and initial conditions. Results are visualized to analyze infection dynamics and model realism.

1

The simplest model: no treatment... The SI model

1.1 Model the infection

The simplest SI model assumes the infection rate is proportional to the current number of infected individuals. The basic model is described by the difference equation:

$$I_t = I_{t-1} + \beta I_{t-1}$$

where β is the transmission rate, and I_t represents the number of infected individuals at time t. With initial parameters $N = 100$ (total population), $I_0 = 3$ (initial infected), and $\beta = 0.02$ (transmission rate), the simulation over one year revealed exponential growth in the infected population.

The entire population became infected by approximately 178 day. This reflects unchecked transmission due to the absence of recovery or immunity.

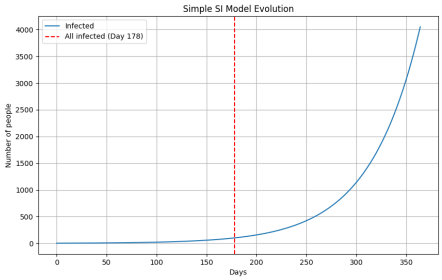

Figure 1: Exponential growth of infected individuals (SI model). Full infection occurs day 178.

1.2 Vary the parameters

Varying β and I_0 alters the epidemic trajectory:

- Higher β (e.g., $\beta = 0.03$) accelerates saturation.
- Lower β (e.g., $\beta = 0.01$) delays saturation.
- Larger I_0 (e.g., $I_0 = 10$) reduces time to saturation.

All simulations assume $I_0 \geq 1$ and $\beta \in (0, 1)$ for realism.

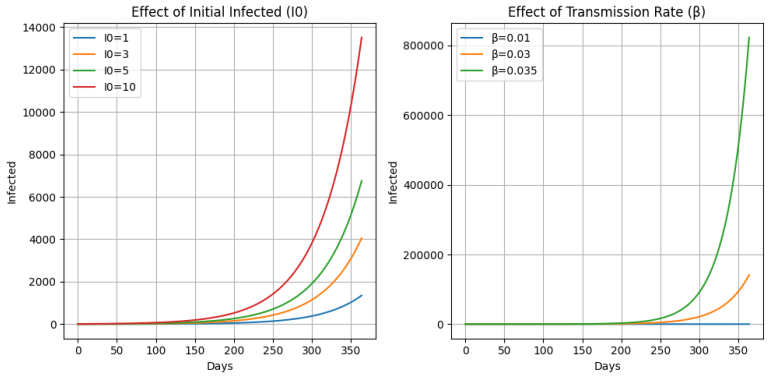

Figure 2:

1.3 Plot the S and I curve together

The susceptible population $S_t = N - I_t$ decreases symmetrically to I_t (Figure 3). The model is unrealistic as it ignores recovery, immunity, and demographic changes.

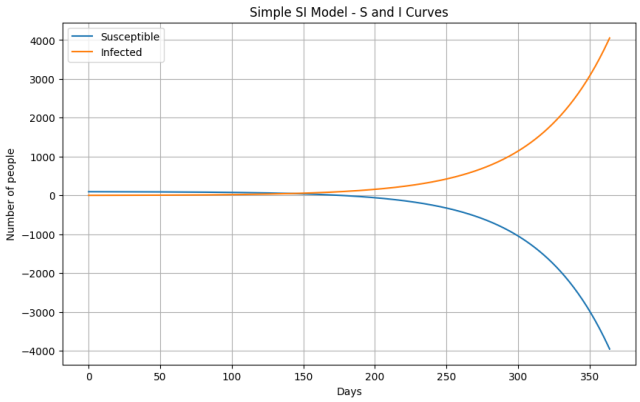

Figure 3: SI model: S_t and I_t over time. S_t depletes entirely as I_t saturates.

1.4 What has changed?

Incorporating the term $\frac{S_{t-1}}{N}$ modifies the equation to:

$$I_t = I_{t-1} + \frac{\beta}{N} I_{t-1} S_{t-1}$$

This logistic growth model produces an S-shaped curve (Figure 4), peaking at $I_t = N$. The inflection point ($I_t = S_t = 50$) occurs at day 175, contrasting sharply with the simpler SI model.

1.5 Change the parameters

Higher β steepens the logistic curve, reducing the time to saturation. For $\beta < 0.01$, the infection fails to propagate (threshold effect). This contrasts with the original SI model, where even small β eventually saturates I_t. (Figure 5)

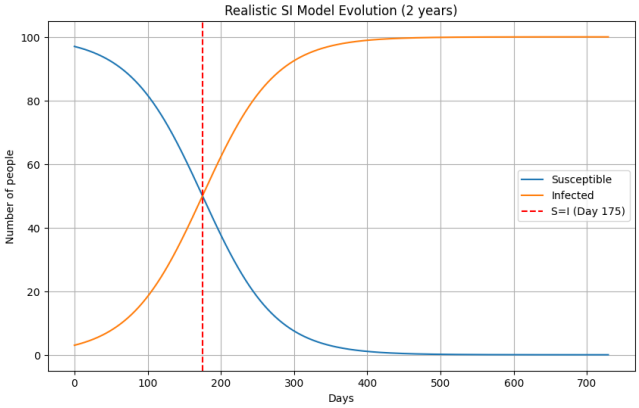

Figure 4: Logistic growth in the corrected SI model. Infection stabilizes at N after 2 years.

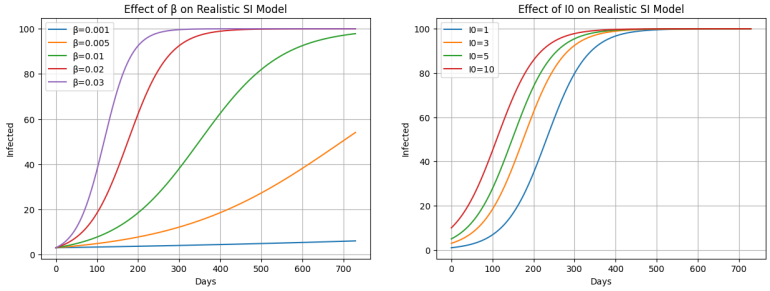

Figure 5: Left: varying β, Right: varying I_0

2 Adding a treatment: the SIS model

2.1 Simulate the curves S and I

The SIS model introduces recovery:

$$I_t = I_{t-1} + \frac{\beta}{N} I_{t-1} S_{t-1} - \gamma I_{t-1}$$

For $\gamma = 0.006$, the infection stabilizes at $I^* \approx 76$ (Figure 6). Increasing $\gamma = 0.009$ lowers I^* to 50, while $\gamma = 0.001$ allows near-saturation ($I^* \approx 97$).

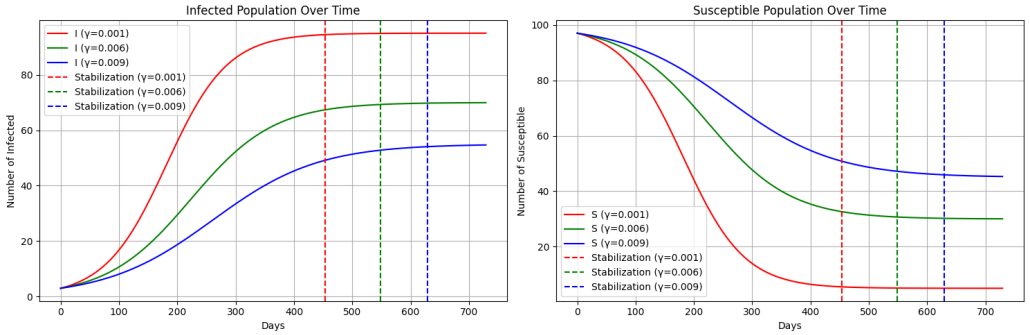

Figure 6: SIS model dynamics for varying γ. Higher recovery rates reduce equilibrium infections.

- With $\gamma = 0.001$: The infection spreads rapidly and maintains a high level, as few people recover.
- With $\gamma = 0.006$: We see oscillatory behavior before reaching equilibrium.
- With $\gamma = 0.009$: the infection is better controlled, reaching a lower equilibrium value.

2.2 Vary $\sigma = \beta/\gamma$ and observe the S curve.

The ratio σ determines equilibrium behavior:

- $\sigma < 1$: The epidemic dies out (disease-free equilibrium) ($I^* = 0$).
- $\sigma = 1$: Critical threshold where the disease barely sustains itself
- $\sigma > 1$: The disease becomes endemic, reaching a non-zero equilibrium $I^* = N\left(1 - \frac{1}{\sigma}\right)$.

For $\sigma = 2$, $I^* = 50$; for $\sigma = 0.5$, $I^* = 0$ (Figure 7).

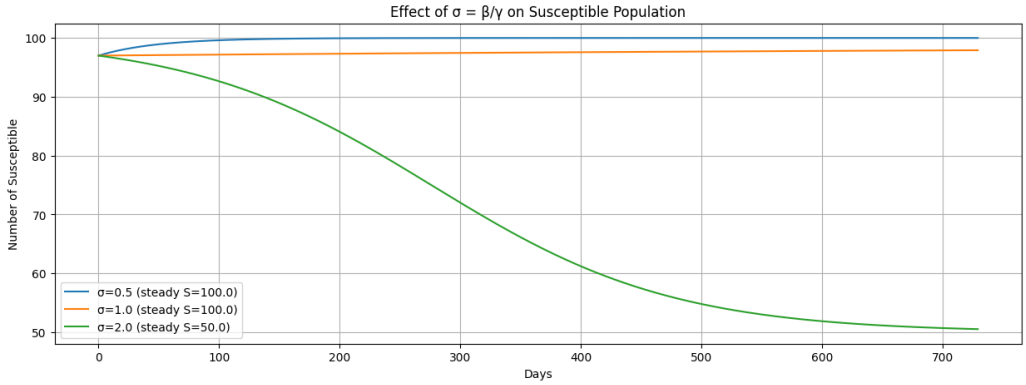

Figure 7: Stationary states depend on $\sigma = \beta/\gamma$.

2.3 Provide a mathematical explanation of the results

2.3.1 Differential Equation Derivation

The discrete-time SIS model can be approximated as a continuous-time differential equation. Starting from the recurrence relation:

$$I_t = I_{t-1} + \frac{\beta}{N} I_{t-1} S_{t-1} - \gamma I_{t-1},$$

where $S_{t-1} = N - I_{t-1}$. For small time steps, this becomes:

$$\frac{dI}{dt} = \beta I \left(1 - \frac{I}{N}\right) - \gamma I.$$

This is the **continuous-time SIS model**.

2.3.2 Equilibrium Solutions

Equilibrium occurs when $\frac{dI}{dt} = 0$:

$$\beta I \left(1 - \frac{I}{N}\right) - \gamma I = 0.$$

Factoring I:

$$I \left[\beta \left(1 - \frac{I}{N}\right) - \gamma\right] = 0.$$

This yields two equilibria:

- **Disease-Free Equilibrium (DFE):** $I^* = 0$.
- **Endemic Equilibrium:** Solve $\beta\left(1 - \frac{I^*}{N}\right) - \gamma = 0$:

$$I^* = N\left(1 - \frac{\gamma}{\beta}\right) = N\left(1 - \frac{1}{\sigma}\right), \quad where \sigma = \frac{\beta}{\gamma}.$$

2.3.3 Stability Analysis

The stability of equilibria depends on the σ:

- **DFE ($I^* = 0$):**
  - If $\sigma < 1$, the DFE is *stable* (infections die out).

- If $\sigma > 1$, the DFE is *unstable* (endemic equilibrium dominates).

- **Endemic Equilibrium ($I^* = N(1 - 1/\sigma)$):**
  - Exists only if $\sigma > 1$.
  - *Stable* when $\sigma > 1$, representing a persistent infection level.

2.3.4 Comparison with Simulations

- **Threshold Behavior:** Simulations (Section 2.2) confirm:
  - For $\sigma = 2$, $I^* = 50$.
  - For $\sigma = 0.5$, $I^* = 0$.

- **Jacobian Analysis:** Linearizing around equilibria:
  - At $I^* = 0$, Jacobian $J = \beta - \gamma$. Stability depends on $sign(J)$.
  - At $I^* = N(1 - 1/\sigma)$, Jacobian $J = -\beta + \gamma$, confirming stability for $\sigma > 1$.

3 Final Remarks

The SI and SIS models, while simplistic, highlight key factors in epidemic dynamics: transmission rates, recovery, and population interactions. Extensions like SIR or SEIR add realism by including immunity or latency. Future work could explore stochastic effects on populations.